A MANUAL

OF THE

DISEASES OF THE HEART

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DISEASES OF THE HEART,

THEIR PATHOLOGY, DIAGNOSIS, PROGNOSIS, AND TREATMENT

BY

ROBERT HUNTER SEMPLE, M.D.

OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON, PHYSICIAN TO THE STANDARD LIFE ASSURANCE COMPANY; PHYSICIAN TO THE BLOOMSBURY DISPENSARY AND TO THE EASTERN DISPENSARY, LONDON; FELLOW OF THE MEDICAL SOCIETY AND OF THE PATHOLOGICAL SOCIETY OF LONDON.

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PREFACE

NOTWITHSTANDING the numerous excellent works which have appeared in various parts of the civilised world on the Diseases of the Heart, it seems to me that the time is not inopportune for the production of a small English manual on the same subject. As with all other topics connected with our profession, old views on cardiac maladies are continually undergoing revision and new ones are introduced to notice, though perhaps eventually destined to share the fate of their predecessors; and my object is to exhibit the present phase of what cannot but be considered a very interesting field of inquiry. Besides these considerations, I have myself made some practical clinical investigations on matters connected with cardiac disease, and as I conceive it to be the duty of every one to contribute his stock of information, however small, I venture to lay my own humble experience, with some of the lessons it conveys, before my professional brethren.

R. H. SEMPLE.

8, TORRINGTON SQUARE,
LONDON.

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DISEASES OF THE HEART

CHAPTER I

INTRODUCTION

ANDRAL, in editing the fourth edition of Laennec's 'Traité de l'Auscultation Médiate et des Maladies des Poumons et du Cœur,' in the year 1837, writes in a note: "In spite of many laborious researches the history of the diseases of the heart is far from being yet finished; it still presents a great many doubts to be removed and many gaps to be filled up, and the time is not yet arrived when the diagnosis of these diseases can be regarded as being so easy and certain as that of the diseases of the lungs. Still, the continual progress which has been made from the time of Lancisi to that of Laennec, and from the latter to that of M. Bouillaud, must inspire us with hope for the future."

It is quite true that the whole history of the diseases of the heart is even now not yet completed, and that some of them are still enveloped in a certain amount of obscurity; that the relations existing between the physical and rational symptoms during life on the one hand, and the appearances found after death on the other, are not yet absolutely determined, and that very many problems of treatment yet remain to be solved. Still, much more has been done in all these directions than Andral, in the preface referred to, appears willing to admit; and it may be asserted, with tolerable truth, that the diagnosis of cardiac diseases is at least as easy and certain as that of the diseases of the lungs; while, in reference to therapeutics, there can be no doubt that a very great advance has been manifest,

not subsided. But as I could detect no evidence of cardiac disease, and indeed found no signs of congestion anywhere, I attributed the persistence of the symptoms to the treatment and not to disease, and pursued a totally different plan, ordering wine, iron and quinine, and with the very best results, for the lady completely recovered, and subsequently married; and on a recent visit to this country she called upon me to express her gratitude for the change of treatment which had been so opportunely adopted, and which perhaps saved her from an infinity of suffering, if not from death itself. In the present day, probably, such a case would not be thought of much interest, but it illustrates well enough the point to which I am referring, namely, the injurious influence of the doctrines taught by some of the most distinguished writers on cardiac disease in the earlier half of the present century.

It is now well known that bleedings and low diet increase the palpitations of the heart, and the very symptoms which led Laennec and Bouillaud, and their followers, to persevere in the depletory system, afford the strongest indications to the practitioners of the present day in Great Britain to discontinue it, if it has been commenced, or to abandon it altogether. In many cases treated by excessive depletion the patients have no doubt survived, but who shall say how many have sunk under it?

Dr Hope, whose classical 'Treatise on Diseases of the Heart' has been of invaluable service to the progress of cardiac pathology and diagnosis, very properly condemns the treatment recommended by Laennec, although he admits that he himself has invariably found the greatest benefit to be derived, in the early stages of hypertrophy of the heart, from sparing abstractions of blood at intervals of two or three weeks or more. "I have constantly noticed," he writes, "that when from the severity of the dyspnœa and palpitation, in the advanced stages of the complaint, the practitioner was induced, or thought himself compelled, to resort to frequent bleedings at short intervals, the patient, though perhaps temporarily relieved, progressively declined from that moment, dropsy increasing, and the paroxysms recurring more frequently and with greater violence, until they eventually terminated in his destruction."[1] Dr Hope's own plan of treating hypertrophy of the heart consisted in the abstraction of blood to the amount of four, six, or eight ounces, every two, three, four, or six weeks, according to the age and

[1] 'Treatise on Diseases of the Heart,' Fourth Edition, p. 264, 1869.

strength of the patient, so as merely to keep down palpitation, dyspnœa, and strong impulse of the heart. But although this plan was recommended, and no doubt adopted, only about thirty years ago, there are very few practitioners of the present day who would consider it either safe or judicious.

Although Dr Hope recommends only the cautious abstraction of blood in hypertrophy of the heart, he is by no means sparing of this mode of depletion in the acute affections of that organ. In the treatment of acute pericarditis and endocarditis he advises that the antiphlogistic treatment, in as energetic a form as circumstances will allow, should be employed with the utmost promptitude. If the attack is recent, and the patient's strength will admit, he recommends that blood should, in the first place, be drawn freely from a large incision in the arm of the patient in the erect position, so as to bring him to the verge of syncope. From five and twenty to forty leeches, according to strength, should then be applied to the præcordial region as soon as the faintness from the venesection disappears and reaction commences. "Unless," he writes, "the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be repeated two, three, or more times, according to the strength, at intervals of from eight to twelve hours; or, what is a better rule, as soon as the pulse and the action of the heart denote a commencement of reaction. It is not, however, in every case," he continues, "that so active a treatment is required. I have seen a single, prompt, and abundant application of leeches, or a cupping, at once subdue every formidable symptom."[1] The last sentence probably contains the true rule of treatment in such cases, for it is quite conceivable, although not perhaps capable of demonstrative proof, that at the very outset of an acute inflammation, the removal of a certain quantity of blood may so affect the general circulation, and, through it, the nervous system, as to nip the incipient disease, as it were, in the bud.

Bouillaud, writing in 1835, announces as a happy discovery his method of bleeding copiously and frequently in pericarditis; and he declares that this kind of treatment has been so successful in his hands that recovery has become the rule in such cases and death the exception, while, according to his predecessor and fellow-countryman, Corvisart, death was the rule and recovery the exception. "As a general rule," he writes in his chapter on the Treat-

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

ment of Pericarditis, "in a subject in the prime of life, three or four bleedings from the arm, in the first three or four days, assisted by the application of twenty-five to thirty leeches, or of cupping-glasses, repeated two or three times, will be sufficient to cure the disease."[1]

There is no physician of our age and country whose opinions have justly commanded more respect and deference, in relation to diseases of the heart, than the late Dr Latham, and in his deeply thoughtful and excellent lectures on subjects connected with Clinical Medicine he thus discourses on the treatment of the acute diseases of the heart. "Bloodletting," he writes, "and mercury, and opium, are your remedies for these diseases of the heart; and so they are for acute rheumatism, irrespective of inflammation of the heart. . . . . Bleeding, mercury, opium, the very remedies you used in acute rheumatism, are, I say, still your main reliance, when inflammation attacks the heart. . . . . With respect to venesection, if in acute rheumatism any of those symptoms referable to the heart are present which have been already mentioned, auscultatory or non-auscultatory, and especially if they have arisen under your own observation, or though not under your own observation, if they be now present, and you have reason to believe that they have *recently* arisen, then, should the pulse be found to have even a notable degree of that hardness which is deemed inflammatory, blood must be taken from the arm. Should there be any doubt about venesection, any misgiving whether the inflammatory hardness of the pulse is quite enough to require it, let it be employed nevertheless. There is greater hazard in omitting it wrongfully than in practising it wrongfully."[2]

This advice was offered to the profession in 1845, and it would be easy to quote opinions almost exactly similar in their nature, from most, if not all, of the clinical teachers of that and preceding periods; and many, like myself, can very well recollect that these doctrines were taught and acted upon in the medical schools of London and elsewhere within the last thirty years. For my own part I can perfectly well remember, as clinical clerk in a London hospital, having, under the directions of my teachers, drawn a large quantity of blood from patients presenting symptoms of acute cardiac disease; and when afterwards, practising in a large parochial district and in

[1] 'Traité Clinique des Maladies du Cœur,' Paris 1835, p. 480.

[2] 'Lectures on Clinical Medicine,' by P. M. Latham, M.D. 1845, vol. i, p. 248.

private cases, I carried out the same kind of treatment on my own responsibility. But I must honestly admit that although I then believed, and do still believe, in the efficacy of bloodletting occasionally employed in certain acute inflammatory and congestive diseases, I never found any very beneficial effects to ensue from this measure in either the acute or chronic diseases of the heart, although, as I have previously remarked, when commenting on a passage from Dr Hope, I think it probable that if some blood could be drawn at the very outset of an acute cardiac inflammation, it might cut it short altogether. When acting on the principles formerly prevailing as to the effect of bleeding in diminishing the fibrine of the blood, I bled patients over and over again in acute rheumatism, and was astonished to find, in opposition to the then existing doctrines, that the relative amount of fibrine was in no way diminished, but in many cases was actually increased by this treatment, while the rheumatic affection was scarcely ever improved. As to the venesection obviating the supervention or arresting the progress of pericarditis or endocarditis, I can truly state that, as far as I could observe, it had no perceptible effect in that direction at all, for cardiac disease often supervened in the cases where bleeding was freely practised, and the inflammation of the membranes of the heart pursued its course although the bleeding was continued.

I am not prepared, however, to state that the treatment by venesection did positive harm in any of the cases in which I practised it, or saw it practised, in this country. The doctrines of Bouillaud, and his merciless evacuations of blood *coup sur coup*, were never in much favour in Great Britain, and, in the cases I witnessed, the abstraction of blood was practised with a certain amount of moderation and with a definite object. I do not recollect a single fatal case of cardiac inflammation where the result could be considered due to excessive depletion. In many of the cases, however, the persistence of abnormal sounds in the cardiac region after recovery proved that the valves had been permanently affected or that the heart was otherwise damaged.

Most of the patients I have lost sight of, as generally happens with hospital and parochial cases, but several others, whom I attended in private practice, are still living and enjoying good health and engaged in the active pursuits of life, although in some I have since convinced myself that the morbid indications remained and in all they probably exist. Whether such patients would have escaped

cardiac complications if they had not been bled is by no means clear, for multitudes of cases of cardiac complications occur where no bleeding has been practised; but I think it may be stated generally that if any one should repeatedly employ venesection in acute rheumatism or in acute disease of the heart, in the hope of preventing or putting an end to fibrinous effusion in the pericardium or on the valves, he will not only fail in his object, but will probably do much more harm than good, and will exhaust the patient instead of curing the disease.

The change of opinion as to the treatment of the acute diseases of the heart may be very well traced in examining the writings of the late Dr Todd, whose clinical teachings, opposed, as they often were, to the views generally entertained by his contemporaries, were always marked by boldness, independence, and originality, although it must be admitted that some of his theories were pushed too far, and that the practice founded upon them has, in many instances, been carried to a dangerous extreme. It was no doubt useless, if not injurious, to draw away large quantities of blood in the hope of curing acute rheumatism or of obviating inflammation of the cardiac structures, but it by no means follows, therefore, that all inflammatory and congestive diseases and all fevers should be treated by the administration of enormous doses of alcohol, and yet it may be stated, without much exaggeration, that such appeared to be the tendency of Dr Todd's practice, especially in the later years of his life.

In Dr Todd's Gulstonian lectures delivered in 1843, this physician recommends that the utmost vigilance should be exercised in detecting the first indications of acute cardiac disease in cases of acute rheumatism; and if any abnormal sounds should be discovered, then the practitioner should have immediate recourse to local bleeding over the cardiac region. A large number of leeches should be at once applied, or a good quantity of blood should be taken by cupping. "I have no doubt," he writes, "that by prompt practice of this kind inflammation of the heart may be checked at its commencement, and as this is the time when the interference of art may be the most efficacious, it is plain how much the future comfort of the patient depends on the vigilance and sagacity of his medical attendant at this juncture."[1]

[1] 'Practical Remarks on Gout, Rheumatic Fever, and Chronic Rheumatism of the Joints,' by R. B. Todd, M.D., F.R.S., p. 197.

Dr Todd immediately adds, in opposition to the views of Bouillaud, Hope, Latham, and others, that his experience leads him to value very lightly the efficacy of general bleeding in inflammation of the heart. He says, "*I have never seen an instance in which it unequivocally did good,*" and he proceeds to show that many of Bouillaud's cases, as recorded by that physician himself, by no means bear out the views which he inculcates, and that, under the large bleedings practised, the patients suffered repeated relapses or their convalescence was very much protracted.

In a subsequent course of clinical lectures on certain acute diseases, published in 1860, Dr Todd goes a step further, and condemns the abstraction of blood altogether. In this work, which contains a great amount of sound reasoning, he remarks that two objects are to be kept in view in the treatment of the cardiac affections accompanying rheumatism, namely, to check the morbid process completely, or to restrain it from producing such changes as may prove destructive to the heart; and secondly, to obviate liquid effusions which may distend the pericardium and compress the heart, and so become seriously detrimental to life. With regard to the first point, Dr Todd thinks it clear that bleeding has no power to stop or prevent the formation of the fibrinous concretions which are apt to collect on the valves. He not unaptly regards these concretions as being formed, in a great measure, mechanically, and thinks that they would form around or upon any opposing material, just as fibrine will coagulate round a bunch of twigs by which blood is beaten as it flows from a vein. If such is the mode, therefore, in which plastic concretions are developed on and in the heart in inflammations of that organ, the object of the practitioner ought to be to prevent the development of the altered state of nutrition which *precedes* the fibrinous formation, and bleeding will not effect this object any more than it will do the same thing in rheumatism. Again, Dr Todd asks whether bleeding will alter the state of the blood which is favorable to the formation of plastic deposits, and he answers the question in the negative. Bleeding, in fact, as is proved by experiments, considerably diminishes the red particles, augments very much the proportion of water, and affects the fibrine very little. He quotes Dr (now Sir Robert) Christison, who has shown that the amount of fibrine increased under bleeding, and Dr Beale, who showed the same fact in the blood of a dog bled on four successive days; and I have before observed (p. 7) that I have often noticed the same fact myself.

"If, then," Dr Todd concludes, "bleeding will not stop the inflammatory state which creates the undue determination of the blood to the pericardial and endocardial surfaces, and if it will not prevent the plastic formations, but rather favours them, surely it is not the remedy for pericarditis and endocarditis. And if the effects of venesection be—as beyond all doubt they are—to diminish all the solids of the blood but the fibrine, and to augment the water, surely the employment of this treatment is fraught with the greatest danger of creating liquid effusions into the serous and synovial sacs, which are so exposed to the action of the rheumatic matter. These are the theoretical grounds on which I object to the practice of bleeding, whether local or general, for the cardiac affections of rheumatic fever, and my experience confirms me in the belief that the practice of bleeding is altogether unsatisfactory in its remedial results and prejudicial in its consequences. I have likewise learned by experience that the practice of abstaining from this mode of treatment is perfectly safe, and tends to the best results." [1]

I consider these remarks exceedingly valuable, and I regard the arguments as conclusive against the practice of *large* bleedings, at least, in rheumatism and acute diseases of the heart, and I think that the general experience of medical practitioners in the present day will confirm the views thus lucidly expressed.

The foregoing remarks are only historical, and the whole question of the treatment of cardiac disease will be discussed in its proper place.

[1] 'Clinical Lectures on Certain Acute Diseases,' by R. B. Todd, M.D., F.R.S., pp. 68, 71.

CHAPTER II

THE ANATOMY AND PHYSIOLOGY OF THE HEART

Before any person can be competent to pronounce an opinion as to the diseased condition of any organ he ought to be fully acquainted with its healthy state and with the varying phenomena which it may present consistently with the performance of its healthy functions. Thus, in the case of the heart, it is not only necessary to be accurately informed as to the usual dimensions of the organ, and as to its natural impulse and natural rhythm, but also to be aware that certain deviations and disturbances in these respects are not inconsistent with healthy structure and action, for the size of the heart differs very much in different individuals, though they may all be in perfect health; and its impulse and the rate of its movements may vary considerably from peculiarities of constitution, or may be increased by exercise, by eating, or by the use of stimulants although in moderation. Its functions may also vary even according to the standing or recumbent position of the patient. It is necessary to bear in mind that mental impressions may also act powerfully upon the rhythm of the heart, either increasing or diminishing its movements or its impulse, or otherwise disturbing the regularity of its action.

These remarks, although they may appear of a common-place nature, are of very great importance in practice, and will be found of special interest to those whose duty may call upon them to examine into, and report upon, the *healthy* condition of the heart, as for instance, in examinations of persons for Life Assurance. In such cases it is often found that the mere fact of being examined by auscultation and percussion, and indeed the fact of undergoing any medical examination at all, will disturb or accelerate the heart's action even in a strong and perfectly healthy person; and the more especially if, as is frequently the case, the success of the application for life assurance is a matter of anxiety to the proposer. The circumstance of having walked rather quickly, or of having just eaten a meal, or of

having ascended a flight of steps, will often make a remarkable difference in the rhythm of the heart, and it need scarcely be remarked that the action of the organ may be still further disturbed by any quickness of manner, however unintentional, on the part of the examiner. Hence, inquiries on the above-mentioned points should always be made before any examination is instituted, and if the heart's action should be accelerated or otherwise disturbed, it will be expedient to make the person rest for some time, or to engage him in cheerful or indifferent conversation, so that the attention may be drawn off from the immediate subject of the medical interview, and the heart be allowed to return to a perfectly normal state. I have myself been often induced to delay the examination of proposers for life assurance owing to transient anomalies in the heart's action, and I have on many occasions postponed the inquiry for a few days in order to give an opportunity for a thorough reconsideration of the case. In many instances, after taking all the precautions just indicated, it was eventually found that the rhythm of the heart was perfectly normal, but in others, notwithstanding the removal of every external disturbing cause, the irregularity remained, and in such cases an unfavorable opinion has necessarily been formed.

An illustrative instance occurred while these pages were being written. A gentleman who wished to insure his life had been examined by the medical referee in the country, in the usual manner, and the latter reported that there was a slight mitral regurgitant murmur, and I, of course, forming my judgment only from the written report, considered that the life was below the perfectly healthy standard, and expressed my opinion accordingly. Subsequently, however, the gentleman applied to another assurance office, and was examined by another medical referee, who pronounced the heart to be perfectly healthy, and the life was accordingly accepted in that office. As there was thus a discrepancy of opinion on the subject, and as the case was again brought before the office with which I am connected, it was proposed that the gentleman should come to London to be examined by me, and he accordingly came for that purpose. I engaged him in quiet conversation for some little time, and could detect nothing abnormal in the cardiac functions except a little irregularity and intermission of the pulse, although I thought I perceived a faint murmur about the apex of the heart. The gentleman having retired, I was in some doubt on the diagnosis, and a medical friend being with me at the time, it was suggested that the proposer

should again be examined, it being necessary that he should ascend a considerable number of stairs after having just gone down them. After this little excitement the murmur over the apex of the heart became quite audible, and thus the opinion of the first medical referee was at length confirmed. I may mention, *en parenthèse*, that in this case there were no rational signs of disease of the heart, there had been no history of rheumatism, and the proposer was apparently in perfect health.

It is not consistent with the size or object of this work to describe at length the anatomy of the heart, or even to enter fully into the consideration of the functions which it performs, but it is necessary to indicate briefly some points in its structure which have a practical bearing upon its diseases, and to refer to its physiology so far as to distinguish its healthy from its morbid actions.

The HEART lies immediately behind the sternum and a little to the left of that bone. It is placed obliquely in the chest, the apex pointing downwards and forwards between the fifth and sixth ribs on the left side, and the base corresponding to the space between the cartilages of the second and third ribs. The base is comparatively the fixed point, being attached to the great vessels, while the apex is free. The lower surface of the heart lies upon the diaphragm, and being separated only by that moveable partition from the stomach, liver, spleen, and other abdominal organs, the heart is necessarily embarrassed mechanically in its actions when the last-named organs are enlarged, distended, or otherwise altered in their volume. But the heart instead of being injuriously affected only by disease, or other unusual conditions of the abdominal structures, would be liable to constant disturbance of its functions by the action of the diaphragm itself, were it not for the admirable provision of nature which has placed the heart upon the *cordiform tendon*, or motionless portion of the diaphragm, instead of placing it on the muscular part which is in constant motion in the processes of respiration.

The heart is completely enclosed in a bag or sac called the *pericardium*, formed of fibrous structure externally, and lined internally by a serous membrane which is common to the internal surface of the pericardium and the external surface of the heart; and the cavities of the heart are lined by another membrane called the *endocardium*, which is reflected over the valves and is continuous with the lining membrane of the arteries.

The heart, although it appears to be a single organ, is in reality a double one, not being divisible, however, into two equal halves, but into two portions, unequal in size and distinct in their functions. It consists in fact of two hearts, the *pulmonic* and the *systemic*, the former being connected with the circulation in the lungs, the latter with that in the system in general. This separation actually exists in some of the lower animals, as in the fishes for instance, and partially in the amphibia and the reptiles; and in some of the mammalia, as in the dugongs, the heart is cleft at its apex, thus indicating its division into two parts. Even in man himself the twofold character of the heart is indicated by a furrow on its external surface, between the pulmonic and systemic portions. In the mechanism of the fœtal heart, the structure and the functions in a great measure correspond to those conditions which are permanent in some of the lower orders of the animal kingdom, and it is only at the first gasp of extra-uterine life that the consolidation of the two hearts is completed, while the functions of each become distinct, and the harmonious co-operation of both is secured. The closure of the foramen ovale, and the obliteration of the ductus arteriosus, shut off the communications between the right and left hearts, and thenceforth they act with independent, though mutually necessary forces.

In the fully formed human heart there are four cavities, two for each side, surrounded by fleshy walls, and the terms *auricles* and *ventricles* are employed to denote both the cavities and their walls. The words, however, though thus conventionally used, are inappropriate and incorrect, for the word *auricle* is really only applicable to the *ear-like* appendage at each side of the base of the heart, while the word *ventricle* (or little stomach) may indicate the cavity, but it very imperfectly represents the muscular walls by which each cavity is bounded and its dimensions are controlled. The four cavities are pretty equal in capacity, and each contains, as far as can be estimated, about three ounces of blood.

The left side, which propels the blood into the system, is much thicker and stronger than the right. The walls of the left ventricle have an average thickness of four-and-a-half lines, those of the right one-and-a-half line. The two sides are completely separated from each other in the fully formed heart by partitions of muscle, or *septa*. But the auricle on each side opens into its corresponding ventricle, the line of transition being marked by the *auriculo-ventricular ring*, and the attachment of the *auriculo-ventricular valves*.

The muscular columns, or *columnæ carneæ*, which are very prominent objects in both ventricles, are much longer and stronger in the left than in the right, for the reason above indicated. It may here also be noticed that disease of the left side of the heart is far more common than that of the right.

It has been already mentioned that although the heart is double, it is not divisible into two equal halves, and in fact, unlike many other organs in the body, it is not symmetrical. The terms *right* and *left*, therefore, as applied to the heart, are calculated to mislead, for the right side is really in front, and the left side is behind, although inclining to the left side. On removing the sternum in the dead subject and then pushing aside the lungs on each side, the right or pulmonic heart is exposed to view, while only a small portion of the left, or systemic, can be discerned.

The Arteries of the heart are the *coronary*, two in number, given off at the origin of the aorta, immediately above and behind the semilunar valves. They together encircle the heart, running along the groove marking the division between the auricles and ventricles, and they each send down a branch running along the furrow which marks the division between the right and left ventricle, and they anastomose with one another at the apex of the organ. The veins of the heart pursue the same course as the arteries, but, of course, in an opposite direction, and they enter the right auricle by a single trunk. The coronary arteries, being the nutritious vessels of the heart, are essential to its well-being, and some serious and even fatal diseases have been attributed to disease of these vessels; and although the progress of modern pathology serves rather to invalidate than to confirm this proposition, yet certain recent physiological researches appear to indicate that the coronary arteries play a very important part in the cardiac functions.

The Nerves of the heart are chiefly derived from the *Sympathetic System*, which consists of a long series of ganglia and connecting threads abundantly distributed to the internal organs of the body and spread over the coats of all the vessels. These nerves and ganglia, not being supplied to the external parts of the body, and, apparently not being endowed with ordinary sensibility, are removed from the cognisance of the senses and the control of the will, and, by this merciful provision of nature, the performance of many functions essential to life, such as circulation and digestion, is secured during health from the disturbances which would necessarily ensue if any

other arrangement existed. The fact that the heart is supplied from this system of nerves and ganglia is perfectly well known, and the nerves themselves can be easily traced as far as the heart, but the exact mode of their distribution and termination in the substance of the heart itself has long been a subject of active and sometimes acrimonious controversy, owing to the fact that the minute nerve-filaments and nerve-centres can with difficulty be distinguished from the surrounding tissues, and even the microscope and chemical analysis throw but little light upon this rather obscure question. Among the more recent researches, however, upon the subject, it may be stated that the German anatomists, Bidder and Rosenberger, have described certain nerve-centres, or ganglia, in the substance of the heart, clustered together chiefly in the lines of juncture between the auricles and ventricles; and which are supposed by Mr (now Sir James) Paget, whose opinion on such a point is entitled to very great weight, to regulate the rhythmical movements of the organ.[1] These views have been confirmed by subsequent researches, and there can be little doubt that the motions of the heart and the course of the blood through the system are in great measure regulated by this peculiar chain of nerve-centres and filaments. Although the functions in question are liable to disturbance from internal and external causes, and are influenced both by corporeal and psychical impressions, yet they are beyond the domain of the will, and a man can no more accelerate or retard or otherwise disturb his own circulation by any mere exercise of volition than he can command the ebb and flow of the tides.

But the heart also receives a nerve-supply from a different source, namely, *the Pneumogastric Nerve* or *Par Vagum*, which rises from the medulla oblongata in the brain, and, passing down the neck and thence into the chest and abdomen, gives a number of branches to important organs in its course. Its functions are complicated, and even yet are rather imperfectly understood, but that it plays a most prominent part in the phenomena of life is indubitable. A slight reference to its distribution will at once indicate its extensive connexion with many vital organs, for it will be found to give branches to supply the larynx and its muscles, motor and sensitive functions being thereby exercised in that organ; other branches to supply the heart, as has just been mentioned; others to the lungs; and others to the stomach. Besides these numerous and important branches, it

[1] "On the Cause of the Rhythmic Motion of the Heart." 'Transactions of the Royal Society,' May 28, 1857.

receives communicating threads from the sympathetic system of ganglia and nerves, from the spinal nerves in the neck, from the glosso-pharyngeal nerve, and from the spinal accessory. Thus the pneumogastric nerve not only gives nervous supplies to the larynx, the heart, the lungs, and the stomach, but it is more or less associated with all the other nerves and ganglia of the body and with the brain.

The heart is essentially composed of muscular fibres, which are arranged in a peculiar manner, as may be seen in a heart which has been boiled for six or eight hours, so as to soften the connecting areolar tissue, and to facilitate the separation of the muscular layers from one another. It is then seen that the muscular fibres of the heart are disposed in a partly spiral and partly circular direction. The superficial fibres are spiral and the deeply seated ones are circular. The former appear to start from the base of the ventricles, then they pass obliquely downwards towards their apex, round which they curl in a spiral manner, and then they change their direction, become deep-seated, and run upwards along the septum of the ventricles and the internal surface of these cavities, and they terminate in the fleshy columns (*carneæ columnæ*) and the inner border of the auriculo-ventricular rings, which mark the separation of the auricles from the ventricles. The deep-seated fibres are wrapped round the ventricles in a nearly circular direction, their points of origin and attachment being the auriculo-ventricular rings and the points of the fleshy columns. From this arrangement of the muscular fibres it follows that when the ventricles contract, the heart is somewhat elongated and its apex is tilted forwards. The protrusion of the point of the heart at the time of contraction was seen and described by the illustrious William Harvey, and the elongation of the organ under the same circumstances, although denied by some observers, has been proved by applying a shoemaker's rule to the heart while in active motion. This experiment has been performed on calves, sheep, and horses, which were stunned, and in which artificial respiration was subsequently kept up, and the result has been verified by Dalton in the case of dogs, rabbits, and frogs.[1]

[1] 'A Treatise on Human Physiology,' by John C. Dalton, jun., M.D. Third Edition. Philadelphia, 1864, p. 275.

The heart contains four cavities, two of which belong to the pulmonic and two to the systemic portion, or, in other words, each heart contains two cavities, and into one of these the blood is received and from the other the blood is propelled. The auricles are as it were the ante-chambers of the heart, and the ventricles receive the blood from the auricles and then send it out respectively to the lungs and to the general system. Both hearts, that is to say, both sides of the heart, act simultaneously, so that while the blood is being received from the lungs it is also received from the system, and while it is being propelled into the lungs, it is at the same time, although from another cavity, propelled into the system. But in order to prevent the regurgitation, or flowing back, of the blood after it has passed from the auricles into the ventricles, or from the latter into the great vessels which convey it to the lungs (I. *pulmonary arteries*), and to the general system (II. *the aorta*), nature has provided two sets of valves, which open to the advancing current of the blood, but immediately close against its reflux.

The VALVES of the heart, the morbid conditions of which will hereafter be specially and fully described, are the flood-gates through which the on-going current of blood is allowed to pass and by which its reflux is effectually prevented. In other words, while the blood is being propelled forwards, the valves recede to allow its passage, but as soon as it has passed, they close in order to prevent its return. The beauty of the mechanism of the cardiac valves and the exactness with which they perform their functions in health, afford some of the most striking examples of creative wisdom to be found in living bodies, and the adaptation of means to the end to be accomplished is no less strikingly manifested in the alternate but harmonious action of these structures. The happy idea of Watt in inventing the valves of the steam-engine, without which contrivances the full development of the powers of steam could never have been effected, is obviously founded upon the mechanism of the cardiac valves, two of which are always open while the other two are shut, and thus the alternate progression and retardation of the current are effectually secured.

The valve on the right side of the heart, guarding the entrance from the right auricle to the right ventricle, is called the *tricuspid*, because it has three points, which are of a somewhat triangular shape, attached at their base to one another and to the right auriculo-ventricular ring, and having their apices and sides continuous with a

number of cords called *chordæ tendineæ*, by which they are attached to the muscular columns or *carneæ columnæ*, which, together with the rest of the muscular structure, make up the walls of the ventricle. The valves are all formed by the duplicature of the lining membrane of the heart, or *endocardium*, enclosing some tendinous fibres. The other valves on the right side of the heart are placed at the entrance of the pulmonary artery, and consist of three semicircular folds, also composed of a doubling of the endocardium over some tendinous fibres. By one end they form together a kind of ring attached to the circumference of the artery, but their other ends are free. When at rest, as in the dead subject, they lie against the walls of the pulmonary artery, but in the living body they lie in that position only while the blood is flowing from the ventricle into the artery, and when it has done so, then they spread across the artery, meet together in the centre, and thus effectually prevent the reflux, or *regurgitation*, as it is called, of the blood.

The mechanism and arrangement of the valves on the left side of the heart are exactly analogous and very similar to those observed on the right. The auriculo-ventricular opening is guarded also by a valve called here the *mitral*, from its fancied resemblance to a bishop's mitre, but this valve is much thicker and stronger than the tricuspid, and consists of only two divisions instead of three, and hence it is sometimes called *bicuspid*. In other respects the valve is similar to its fellow on the other side, and it is attached to the auriculo-ventricular ring by its base, while its apices and sides are attached to tendinous cords (*chordæ tendineæ*) by means of which it is connected with the muscular columns (*carneæ columnæ*), which again are much thicker and stronger than those of the right ventricle, and though individually smaller, are more numerous and more densely aggregated together. The other valves on the left side, corresponding to the semilunar valves on the right side, are, like their fellows, called *semilunar*, from the similarity of their structure and appearance; although it is now far more common to call them *aortic valves* for the sake of distinction, while the others are called *pulmonary*. But the aortic valves are thicker and stronger than the pulmonary valves. The aortic valves lie against the sides of the aorta while the blood is passing into the system, and they spread across the area of the vessel, in order to prevent its return into the ventricle. Behind the aortic valves the wall of the aorta forms three somewhat hemispherical pouches, called the *sinuses of Valsalva*,

and from two of these arise the *coronary arteries*, which furnish the supply of blood to the heart itself. (See p. 15.)

The leading facts connected with the anatomy of the nerves which supply the heart have already been briefly indicated. There are three ganglia in front of the vertebral column in the neck of the human subject, and these are named in their order the superior, the middle, and the inferior cervical ganglion, and each of these ganglia sends down a cardiac branch to supply the heart, each branch being called respectively the superior, the middle, and the inferior cardiac nerve. These cardiac nerves are continued either singly or together, to form a network of nerves and ganglia, called the *cardiac plexus*, situated behind the arch of the aorta, and giving off filaments to the heart. But the cardiac nerves are not only joined by other branches of the sympathetic system, but they are also joined by some branches of the pneumogastric nerve (*par vagum*), which likewise contribute to form the cardiac plexus. Some of these branches of the pneumogastric are derived from the *recurrent laryngeal* branch of that nerve. This recurrent laryngeal nerve on the right side winds rounds the subclavian artery, and on the left side round the arch of the aorta, and as it makes this turn on each side it gives off the nervous branches to the deep cardiac plexus. But some cardiac branches are derived from the trunk of the pneumogastric nerve as it lies in the chest by the side of the trachea, and others are given off both in the upper and lower part of the neck. It should also be mentioned that the cardiac nerves derived from the sympathetic system are connected by means of communicating branches with the spinal nerves as they issue from the vertebral column, and the close connection thus established between the different systems of nerves supplying the heart explains many phenomena connected not only with the physiology of the organ, but also with its morbid conditions.

The exact course, distribution, connections, and relations of the nervous filaments distributed to the heart are liable to very great varieties, but the above summary may convey a fair general idea of the recognised anatomical facts as they may be traced by ordinary dissection, although the precise mode in which the nerves are supplied to the substance of the heart itself, or the way in which they terminate, are subjects still involved in doubt. The fact is that, notwithstanding the accuracy of research, which is

characteristic of the anatomical and physiological investigations of the present day, it is extremely difficult to distinguish the more delicate nerve-fibrils from the threads of ordinary areolar tissue, or to mark off clearly the nerve-centres, or *ganglia*, from some of the tissues in which they lie embedded. Hence it must be admitted that our knowledge of the exact nerve-supply of the substance of the heart is rather to be gained by comparative investigations and experiments on the lower animals than by actual demonstration on the human subject, while the part played by the respective cardiac nerves and ganglia is estimated chiefly by the results of pathological observations, and by those experimental researches on the lower animals which have distinguished the physiologists of the present day. Nevertheless, it is right to mention that the ganglia of the heart have been described by Robert Lee, Remak, and others, as has been intimated in a previous page.

Dr William Rutherford, the distinguished Professor of the Institutes of Medicine in the University of Edinburgh, who is one of the most recent investigators of the nervous apparatus of the heart, admits that with regard to the nature of the nervous mechanism existing within the organ, we know nothing definite, although we may be safe in supposing that the muscular fibres are presided over by motor nerves connected with cells of the intra-cardiac ganglia.[1]

As I have just observed, our knowledge of the mode in which the functions of the heart are performed, and of the precise arrangements of its nerve-supply, is mainly drawn from experimental researches on the lower animals and from reasoning on the phenomena obtained, and Dr Rutherford's recent lectures offer most valuable information of this character.

He exhibited to his class a heart removed from a frog which had been narcotised by opium. The heart went on pulsating rhythmically, showing that in its structure there existed the conditions necessary for its movements independently of any central nervous system. But he further showed that these conditions did not exist in every part of the heart, for on dividing the organ transversely through the ventricle (the frog, it must be recollected, has only one ventricle), the apex lay motionless, while the base went on beating rhythmically as before. Nor was this immobility due to the want of contractility of the muscular fibres, for, on sending a faradic

[1] 'Lancet,' Dec. 16, 1871. "Lectures on Experimental Physiology," by Wm. Rutherford, M.D., p. 841.

shock through the apex, it contracted, and then again became motionless. Hence it is assumed that the conditions necessary for the rhythmical motion exist at the base and not at the apex of the frog's heart.[1] Ganglia are found about the base, but not in the apex of the heart, and hence it is generally believed that Remak was right in maintaining that the intra-cardiac ganglia are essential to the heart's rhythmical actions.

It being admitted that the nerve-supply of the heart is obtained from the sympathetic system and from the pneumogastric nerve (see above, p. 20), it becomes a very interesting question to determine what functions these nerves respectively perform, and here very great information has been gained by experimental investigation on the lower animals. Dr Rutherford divides the vagus (pneumogastric) nerve in the middle of the neck of a narcotised frog, and then irritates the lower or cardiac end, at first gently and then severely. During the gentle stimulation the heart beats more slowly, and during the powerful stimulation it comes to a standstill, in a state of relaxation. This is not due to paralysis of the muscular fibres of the heart, for when they are irritated they will contract. From a series of reasonings deduced from the facts observed it is inferred that this retardation of the heart's motion is due to an *inhibitory* action on the part of the pneumogastric nerve, or, in other words, that the function of this nerve is to restrain or diminish the pulsations of the organ.

But it would also appear, from the results of experiments and reasoning, that the different cardiac branches of the vagus nerve exercise different functions. The *superior* cardiac branch seems to be the sensory nerve of the heart, for, when it is divided and its upper end stimulated, signs of pain are produced, and it is also an *excito-cardio-inhibitory* nerve, for it conveys influences to the spinal cord or the medulla oblongata which excite the cardio-inhibitory fibres of the vagus. But the most remarkable result which follows stimulation of the central end of this nerve is a fall in the arterial blood-pressure, the effect being due chiefly to dilatation of blood-vessels, and chiefly those of the abdominal viscera. The *inferior* cardiac branch of the vagus inhibits the heart, bringing it to rest, and dilating it by rendering it passive; the *superior* cardiac branch inhibits blood-vessels, bringing them into a state of dilatation,

[1] 'Lancet,' Dec. 16, 1871. "Lectures on Experimental Physiology," by Wm. Rutherford, M.D., p. 841.

that is, a state of rest. The former is *cardio-inhibitory*, the latter *vaso-inhibitory*.

The function of the cardiac branches of the sympathetic system of nerves appears to be, in some respects, the reverse of that of the branches of the vagus, for the former excite the cardiac action, and may be called *excito-cardio-motor*. They antagonise the influence of the cardio-inhibitory nerves derived from the vagus. The inferior cardiac branch of the vagus inhibits, the sympathetic branches accelerate the heart's actions, but the precise nature of the influences which bring them into play is not accurately known. There can be no doubt, Dr Rutherford thinks, that the pressure of the blood upon the lining membrane of the heart can influence the motions of that organ, for it has been shown by Ludwig and Von Bezold that, when the vagi and sympathetic nerves of the heart are divided, increased pressure within the heart accelerates cardiac action. On the other hand, diminution of the pressure is followed by diminished speed of the heart.[1]

It would thus appear easy to draw the conclusion that the heart's action is influenced chiefly by two series of nerves, being accelerated by the one and inhibited or restrained by the other, the first series being the branches of the sympathetic, and the second the inferior cardiac branches of the pneumogastric. If the former set were in an uncontrolled state of activity the heart would beat too fast, and if the latter were unduly active the heart would not beat at all. But it must not be supposed that these views are universally or generally admitted, for some physiologists have drawn different deductions, and Dr Rutherford himself admits that the whole subject requires further investigation before definite conclusions can be drawn. The problem is altogether a difficult one, and the anatomical and physiological details are necessarily complicated, for the sympathetic system receives through its ganglia filaments both from the motor and the sensitive nerves, and it is almost impossible, perhaps quite so, to disentangle and to distinguish these nervous threads from one another. Even the primary fact that the sympathetic system is destitute of ordinary sensation must be a statement received with many reservations, for it is undoubted that although in a state of health this system appears to suffer no pain, and indeed does not manifest any sensibility to any common stimuli, yet in many morbid conditions, as is well known, it is the seat of acute pain. This somewhat paradoxical fact has been

[1] 'Lancet,' Dec. 16, 1871. "Lectures on Experimental Physiology," by Wm. Rutherford, M.D., p. 844.

explained on the supposition that under ordinary circumstances the sensory nerve-filaments in passing through the sympathetic ganglia are deadened in their function by the influence of the latter; while, in morbid conditions, it may be conceived that the sympathetic ganglia are paralysed, and the sensation of the ordinary nerves is thereby allowed to develope itself.

In connection with the physiology of the heart, enough has now been adduced to show why its healthy functions are performed without the control or influence of the will, and why the organ itself is destitute of ordinary sensibility. The painful affections of the heart may receive some explanation from the suggestions just thrown out, and the effect of medicines may perhaps be explained by reference to its nerve-supply. Such explanations will be subsequently offered in the course of this work.

As it is not my intention to give an historical account of the various theories entertained as to the movements of the heart, and the respective periods of their occurrence, I pass over the views expressed on this subject by Haller, Harvey, and Laennec, only observing that the first physiologist failed to notice any period of repose between the contractions of the auricles and ventricles, or at any rate considered such repose only as an occasional occurrence;[1] that the second showed that the contraction of the heart constituted only one movement, in which both auricles and ventricles take part, and that during the interval between each contraction and its successor the heart is absolutely at rest;[2] and that the third, Laennec, agreed with Harvey in thinking that the heart, far from being in a constant state of movement, presents alternate periods of action and repose, like some other muscles of the animal economy, as the diaphragm and the intercostal muscles.[3] Laennec, in fact, roughly calculated, (on the

[1] Post auricularum constrictionem, celerrimé in calido et sano animale, aliquanto lentius in frigido et languente, et nonnunquam satis magno etiam in calidis tempusculo interposito sequitur ventriculorum contractio (Haller, 'Elem. Physiologiæ,' tom. I, lib. iv, sect. 4). The word *nonnunquam* seems to imply that the interval of rest is not a constant event.

[2] Primum sese contrahit auricula et in illa contractione sanguinem contentum in ventriculum cordis conjicit; quo repleto, cor sese erigit, continuo omnes nervos tendit, contrahit ventriculos, et pulsum facit. . . . Isti duo motus, auricularum unus, alter ventriculorum, ita per consecutionem fiunt, servata quasi harmonia et rhythmo, ut ambo simul fiant et unicus tantum motus apparet. Harvey, 'Opera omnia,' 1766, p. 33.

[3] Laennec, vol iii, p. 53.

supposition that of the whole time occupied by a rhythmic movement of the heart one fourth is occupied by absolute repose, one half by the contraction of the ventricles, and one fourth by that of the auricles), that in twenty-four hours the ventricles have twelve hours of repose and the auricles eighteen. Such calculation, however, is somewhat incorrect, for the filling and subsequent contraction of the auricles is a period of activity, although not accompanied with any sound, and the amount of absolute rest, therefore, must be less than that supposed by Laennec.

The succession of phenomena in a cardiac revolution has been well described by Dr Hope, and his description may be regarded, even in the present day, as expressing pretty accurately the actual facts. He states that the first motion of the heart, in a rhythmical period of its action, is the systole of the auricles, which interrupts the period of repose; then the systole of the ventricles commences suddenly, the auriculo-ventricular valves close, the apex of the heart is tilted up and is drawn towards the base, or rather it may be said to execute a spiral movement from right to left, and from behind forwards; then comes the diastole and the retrocession of the apex of the heart from the walls of the chest; and next succeeds the interval of repose, during which the ventricle remains at rest, in a state of fulness though not distension, through the whole period intervening between the second and first sounds. The auricle, however, remains at rest only during the first portion of that period, the remainder being occupied by its next contraction, which interrupts the period of repose and recommences a cardiac revolution.[1]

Dr Hope agrees in the main with Laennec in supposing that the ventricular systole occupies about half the time of a whole beat, that the ventricular diastole occupies a fourth, or at most a third, and that the interval of ventricular repose occupies a fourth, or rather less, during the latter half of which the auricular systole takes place. It will be observed, however, that Laennec was incorrect in assuming that the heart was *in absolute repose* during a fourth of the period, for towards the end of that period the auricles are contracting.

But the opinion entertained by Hope, and previously held by Haller and Laennec, that the duration of the contraction of the heart represented something like half of the whole period of a cardiac revolution, is strongly combated by Dr Burdon Sanderson, who

[1] Hope on 'Diseases of the Heart,' p. 50.

considers that the time occupied by the heart in contraction is very much shorter.[1] This distinguished physiologist quotes some experiments by Chauveau, showing that the exact time occupied by the contraction of the ventricles in a horse was about a fifth of a second; and Dr Sanderson's own observations with the sphygmograph confirm the views of Chauveau. In the last edition of 'Kirkes's Physiology' (edited by Morrant Baker) the periods of ventricular contraction and dilatation are considered to be in the proportion of 4 to 7; and Dr Herbert Davies's researches (presently to be noticed) on the comparative areas of the four orifices of the heart further confirm the truth of Dr Sanderson's opinion.

It is a clearly established fact that the contraction of both ventricles is simultaneous, and thus while the blood is being propelled through the aorta to the system, the current is also being propelled through the pulmonary arteries into the lungs. The contraction of both auricles is also simultaneous, occurring just before the contraction of the ventricles, and the blood is transmitted through the mitral valves into the left ventricle at the same point of time at which the blood is passed through the tricuspid valves into the right ventricle. What is also to be noted is that *the same quantity* of blood is sent into the system through the aorta as that which is sent into the lungs through the pulmonary artery, and *the same quantity* of blood is also sent into the left ventricle as that which is sent into the right. But as the blood sent into the system has a much greater distance to travel than that sent into the lungs, it follows that the force by which it is propelled is much greater, and its velocity is likewise greater, and this force and this velocity are impressed upon it by the muscular walls of the left ventricle, which are much thicker and stronger than those of the right. On the other hand, as the force and the velocity of the blood travelling through the pulmonary artery are less than its force and velocity when travelling through the aorta, the area of the pulmonary orifice is greater than that of the aortic orifice, so as to oppose a less degree of resistance to the current. Exactly the same truth holds good, although the reasons are not at first quite so apparent, as to the passage of blood through the respective auriculo-ventricular openings, for the current passing through the tricuspid valves has a less degree of force and a less degree of velocity than that which passes through the mitral valves, and consequently the area of the tricuspid orifice is greater than that of the mitral.

[1] 'Handbook of the Sphygmograph,' by J. B. Sanderson, p. 53.

From careful measurements of the four orifices of the heart it is found that the circumference, and consequently the area, of the tricuspid orifice are greater than those of the mitral, and that the circumference and area of the pulmonic orifice are greater than those of the aortic.

The actual measurements, as given by Dr Peacock in his Croonian Lectures for 1865, give the following as the mean circumference of the four orifices, expressed in English inches:

	Males.	*Females.*
Tricuspid	4·74	4·562
Pulmonic	3·552	3·493
Mitral	4	3·996
Aortic	3·14	3·019

From these data of the *circumference* of the respective orifices, Dr Herbert Davies deduces the measurement of the radius and then of the *area* in each case, and he finds the following to be the mean areas in the male (substituting vulgar fractions for the more accurate decimals, for facility of recollection):

	Square inch.
Tricuspid	1¾
Pulmonic	1
Mitral	1¼
Aortic	¾

Thus the apertures differ very considerably in area from each other, the tricuspid having the largest area of all, its orifice being more than double the size of the aortic opening.

But Dr Herbert Davies further shows that however irregular these areas appear to be in magnitude as compared with each other, there is a distinct and constant law presiding over them, as may be discovered by comparing the *ratios* of the areas of corresponding orifices.[1]

Thus, by mathematical calculations which he gives in his paper, he shows that the area of the tricuspid orifice bears nearly the same relation to the area of the mitral which the area of the pulmonic orifice does to that of the aortic, and in fact his figures show that these two ratios differ from each other only by one tenth, the ratio in the case of the tricuspid and mitral orifices being nearly 1·4 and that of the pulmonic and aortic orifices being nearly 1·3 or 1·4 —1·3 = 0·1.

[1] 'On the Law which regulates the Relative Magnitude of the areas of the Four Orifices of the Heart,' by Herbert Davies, M.D. (From the Proceedings of the Royal Society, No. 118, 1870.)

This calculation is made upon the measurements of the orifices as given by Dr Peacock, but on taking the measurements as given by Dr Reid (which differ somewhat from those of Dr Peacock) the *ratios* are still found to be very nearly the same, the difference being still less than when taking Dr Peacock's measurements. The ratio of the area of the tricuspid and mitral orifices (according to Dr Reid's measurements in males) is found by Dr Herbert Davies to be 1·31, and that of the pulmonic and aortic orifices 1·26, so that the difference is only ·05, namely, 1·31—1·26= ·05, and taking Dr Reid's measurements for females the difference is still less, for it is only ·04.

Dr Herbert Davies remarks that the actual measurements of the orifices of the heart are necessarily liable to error, but that the close identity of the respective ratios may justify the conclusion that they are really identical, and that the small differences in the calculable results depend entirely on the impossibility of obtaining absolutely correct measurements of the boundaries of the orifices. It is clear, therefore, that in whatever proportion the tricuspid orifice is larger than the mitral, in exactly the same proportion is the pulmonic orifice larger than the aortic. Dr Davies records the results of a series of observations made by himself on the measurements of the cardiac orifices in the lower animals, as the horse, the donkey, the ox, the calf, the sheep, the pig, and the dog, and he finds in all a confirmation of his views, and he therefore concludes that in the healthy human heart, and most probably in the hearts of most animals, the areas of the four apertures bear an exact mathematical relationship to each other, and consequently that if the areas of any three of the openings be known, the area of the fourth orifice can be correctly calculated. In confirmation of this view, Dr Davies gives the measurements and calculations which he made in the case of a strong healthy man who died from the effects of a fractured spine. The heart being perfectly healthy, Dr Davies measured the pulmonic, mitral, and aortic orifices, then calculated the area of the tricuspid, and then measured its circumference. Without here giving the details of the mathematical calculations founded on the theoretical law and on the actual measurements, it is sufficient to state that Dr Davies shows that the area of the tricuspid orifice by measurement was 2·070 square inches, and by calculation it was 1·972, the difference thus being only ·098 and offering a strong confirmation of the truth of Dr Davies's law.[1]

[1] Op. citato, p. 272

By examining the measurements it is also found that the *ratio* of the areas of any two corresponding orifices is almost constant in the same animal and probably in all animals, including man. Thus, Dr Davies goes on to show, the area of the tricuspid is nearly 1·3 times the area of the mitral orifice, and the area of the pulmonic of course bears the same proportions to that of the aortic opening. "By measuring, therefore, the two orifices of the right (supposed healthy) we are enabled by this law to deduce approximately the magnitude of the areas of those of the left heart and *vice versâ*. A healthy orifice being known, the area of the corresponding opening on the other side of the heart can be approximately calculated; and should the latter be diseased, its deviation from the normal area can be determined, and the amount of abnormal contraction or dilatation fairly estimated."[1]

In illustration of this approximative law Dr Davies exemplifies, by a case of mitral constriction described by Dr Walshe, the mode in which the amount of constriction may be calculated. In the case alluded to, the area of the tricuspid was of the natural dimensions or 1·9 square inches, but the area of the mitral (owing to the disease) was only ·28, and therefore the area of the former was seven times larger than that of the latter instead of being only 1·3 times larger, and the amount of contraction of the mitral orifice is obtained by subtracting its actual size from its normal size, or (the normal size) 1·45—·28=1·17 square inches, which is the amount of contraction of the mitral orifice.

The blood is propelled with very great force, by the strong muscular walls of the left ventricle, into the aorta, and thence through the system. From comparative experiments on other animals it has been estimated that the vigorous action of the human heart would sustain a column of blood in the aorta about 7½ feet high, or, in other words, that the force with which the heart ordinarily propels the blood through the aorta is equal to that which would be generated by the weight of a column of blood of the same size and 7½ feet high, which weight would be about 4½ lbs. But the force is really as much greater than this as the area of a plane passing through the base and apex of the left ventricle is greater than the area of the transverse section of the aorta; and as the proportion of the former to the latter is about 3 : 1, so the real force of the heart may be stated at about 13 lbs.[2]

[1] Op. citato, p. 272.

[2] 'Carpenter's Manual of Physiology.' Fourth Edition, p. 420.

The blood being sent into the arteries, those vessels, having elastic walls, increase both in length and breadth at each jet of the fluid, and the alternate rising and falling of the arterial coats constitute the *pulse*. From this property of elasticity possessed by the arteries the flow of the blood is equalised in the system; for if they were rigid tubes, the stream would be intermittent throughout, whereas, in consequence of their anatomical structure and their supply of muscular and elastic fibres, the pulsation, or, in other words, the intermittence of the current, is developed most at the commencement of the arterial system, it becomes less and less in proportion to the distance of the arteries from the heart and their subdivision, and it is not perceived at all in the minute vessels. The chief force which transmits the blood through the arteries is the propulsive power of the left ventricle, the *vis à tergo*, as it is termed, being thus propagated through the whole of these vessels, and indeed throughout the whole of the circulating system, but the muscular walls of the arteries undoubtedly contribute to produce the same effect. It is now sufficiently proved, indeed, that the muscular contractility of the arteries may be exerted independently of the heart altogether, as for instance by the local application of such stimuli as act upon muscular fibre in general, such as electricity.

It is also inferred, from the results of experiments, that the contractility of the arterial walls is very materially influenced by the nervous supply from the sympathetic system which is distributed upon them, and which constitutes what is called the *vaso-motor* system of nerves. The division of the sympathetic trunk on either side of the neck produces an enlargement of the small arteries on that side of the face, and from this and other experiments as well as from some more familiar phenomena (such as blushing for instance), it is conjectured that the vaso-motor system of nerves exerts, in the healthy state, a kind of *inhibitory* power over the small vessels, controlling the current of blood and restraining and regulating its flow. But when this system is disturbed, or deranged, or impaired in its functions by accidental circumstances, or by disease, then the arteries dilate, the blood flows into them in undue quantities, heat, redness, and swelling are perceptibly induced, and a number of phenomena, partly natural and partly morbid, are accounted for. The influence of mental impressions and of external agencies on certain parts of the arterial system may thus in some measure be explained, and probably the whole range of pathology and therapeutics

may receive more or less light from a further study of the functions of this part of the nervous apparatus.

It is a very curious fact that the arterial system possesses nearly the same capacity in every part; or, in other words, if all the arteries proceeding from the aorta could be cut through, the united areas would be equal to the area of the aorta, and those of the pulmonary arteries would be equal to that of their trunk.[1] But the diameters of the great trunks far exceed those of the lesser arteries, and nevertheless the areas are proved to be the same, from the mathematical law that the areas of circles are as the squares of their diameters. Thus a trunk may have a diameter of 7 and may subdivide into two branches, each having a diameter of nearly 5, but the square of 7 is 49, and double the square of 5 (or 25) is 50, and therefore 49 is the area of the trunk and 50 (only 1 more) is the area of the branches.

From the arteries the blood enters into the series of vessels called the *capillaries*, which include all the minute vessels passing between the arteries and the veins. This *capillary system* of vessels is the seat of some of the most important functions of the human body, such as nutrition and secretion, and also of some of the most remarkable morbid phenomena, such as inflammation and congestion. The blood passes naturally through these minute vessels in an even and continuous stream, this effect being secured by the elasticity of the walls of the arteries as before explained. The chief force which drives the blood onward through the capillaries is still the *vis à tergo* from the left ventricle, but the action is assisted by the muscular walls of the arteries, and also by the performance of the functions of the capillaries themselves. Thus the capillaries of the system give out oxygen to the tissues and receive carbonic acid, and the capillaries of the lungs give out carbonic acid to the atmosphere and take in oxygen, and if any of these changes be prevented from taking place a retardation of blood in the capillaries will ensue.

The capillaries unite together to form the commencements of the veins, and these latter are the vessels which convey back to the heart the blood which has traversed the system and has performed the functions of nutrition, secretion, excretion, &c., and which has lost its oxygen, has taken in exchange carbonic acid, and has altered its colour from scarlet to black. The veins have nearly the same

[1] 'Carpenter's Manual of Physiology.' Fourth Edition, p. 426.

structure as the arteries, but they have less elastic and muscular tissue in their coats, and consequently have less elastic and propulsive power.

The motion of the blood through the veins is still effected chiefly by the *vis à tergo* from the left ventricle of the heart, aided by the muscularity of the arteries and the forces exercised in the capillaries, but there are additional forces to assist the passage of the blood through the venous system, and which compensate for the deficiency of propulsive power in the venous walls. Thus the compression exercised by the muscles on these vessels facilitates the passage of the blood through their cavities, and the fluid is prevented from retrogression by the *valves* with which the veins (as contra-distinguished in this respect from the arteries) are abundantly supplied. A familiar instance of this fact was supplied in the times when bleeding at the arm was a common operation, for, when the stream of blood was languid, it was accelerated by requesting the patient to clench his fist or grasp some neighbouring object, so as to put the muscles into action, and thus promote the flow of the venous current towards the bleeding orifice. In addition to these circumstances which favour the passage of the blood towards the heart, the movement of inspiration contributes a certain amount of influence, for in this process the cavity of the chest is increased in capacity and a partial vacuum is caused which the blood is drawn in to fill. But notwithstanding all these conditions which tend to transmit the blood through the veins, these vessels are so much inferior to the arteries in propulsive power that even the force of gravity occasionally interrupts the progress of the returning stream, and thus it is found that venous congestion is not uncommon in the depending parts of the body, as in the legs of persons who stand much, or in the back, when the subject, especially after long illness, has been lying in the recumbent position. Hence, too, dropsical effusions often commence in the ankles from mere weakness of the veins, especially in anæmic subjects, and from congestion and distension of these vessels, in cases of disease of the heart, as will hereafter be described.

The blood having now traversed all parts of the body, and having performed all its functions,—starting from the heart in a vigorous but intermittent stream, which becomes less vigorous but continuous as the arteries become smaller; then coursing through the capillaries in millions of minute channels, and giving off nutritious

or excrementitious matters as it runs along; and then moving onwards, though with much less force and velocity, in the veins—the dark and sluggish current at length reaches the right side of the heart, to which it is carried by the superior and inferior venæ cavæ, and in which it is joined by the small quantity of blood coming from the heart itself through the single opening of the coronary vein.

The comparative force with which the blood passes through the arterial and the venous tubes has been ascertained by experiment. Dr Stephen Hales, as long ago as 1732, estimated the pressure of the blood in the jugular vein as only one ninth to one fourteenth of the pressure on the arterial side; and Valentin, by more recent and accurate experiments, determined the pressure in the jugular vein to be one tenth to one twelfth of the pressure in the carotid artery. Thus it is quite evident that the force and velocity of the blood entering the right auricle, and thence passing to the right ventricle, must be much smaller than the force and velocity of the fluid passing out of the left ventricle by the aorta, and thus, as Dr Herbert Davies justly remarks, we can understand why the area of the tricuspid opening is much greater than that of the aortic, namely, in order that its much larger orifice may compensate for the comparatively sluggish stream which it has to transmit. Dr Davies, in his papers referred to, indicates the circumstances just adduced, which progressively diminish the velocity and momentum of the blood as it passes through the arteries, capillaries and veins, and cause the stream eventually to enter the right auricle of the heart slowly and with little force, but still with sufficient velocity and momentum to expand the right ventricle. Now the right ventricle is much thinner and consequently less powerful than the left, although it is quite sufficient to propel the blood into the lungs through the pulmonary artery, and from this comparative weakness of its walls it offers but a small resistance to the blood entering by the tricuspid orifice. But the slowness of the current on the right side is compensated by the larger area of the opening through which it passes, and the comparatively long period during which the ventricle is open to receive the blood, so that a stream of small velocity and force is sufficient to expand it. Dr Herbert Davies thinks that the force exerted by the contraction of the auricle is so small, and is in operation for so short a period of time, that its chief use must be to complete the closure of the tricuspid valve.

Dr Davies also draws attention to the fact that the particles of

the blood-stream which have entered the tricuspid orifice in a direction nearly at right angles to the axis of the pulmonary artery must, when the ventricle has become filled, change their direction of motion when they are driven out of the ventricle as it contracts. He therefore suggests the idea that at the end of the diastole the whole of the contents of the ventricle are momentarily at rest and ready to take up a new movement in a course nearly at right angles to their line of entrance from the auricle. "If this view," he goes on to say, "which has escaped the attention of physiologists, be correct, we observe an additional reason for the blood which enters the ventricle possessing an amount of velocity and energy just sufficient, and no more, to complete the dilatation of the chamber, and, having performed its task, to assume for a moment an attitude of repose before the contraction of the ventricle sends it forth in a different direction."[1] The remarks thus applicable to the right ventricle are equally so in the case of the left, the current on the left side losing, under the same circumstances, its motion for a moment.

The passage of the blood through the tricuspid and mitral orifices takes place at the same time, and the volume of the blood in each case is the same, but the current passing through the latter dilates the ventricle more powerfully than that passing through the former. The two ventricles being of unequal thickness, and therefore of unequal strength, "require currents of blood of different force to overcome their respective *inertia*, fill their chambers, and complete their dilatation in exactly equal and the same times." In mathematical language, the momentum of the mitral is greater than that of the tricuspid current, or in other words, the volume of the mitral column multiplied by its velocity is *greater than* the volume of the tricuspid column multiplied by its velocity; but the volume of each current is *the same:* and hence, eliminating *volume* from each side of the above formula, it is proved that the velocity of the mitral is greater than that of the tricuspid current.[2] The mitral orifice has a less area than the tricuspid, as has been shown above (see p. 27), but the greater velocity of the current through the mitral effects the same end as the less velocity through the tricuspid, because in the latter the area is greater and the resistance to the current is consequently less.

[1] Dr. Davies. Op. citato, p. 282.

[2] Op. citato, p. 283.

CHAPTER III

THE SOUNDS OF THE HEART

When the ear or the stethoscope is applied over the præcordial region of a healthy person, a peculiar double sound is heard consisting of two successive sounds, of which one is dull and somewhat prolonged and the second shorter and clearer. It is very difficult to represent these sounds to the eye by means of written characters, but the accented syllables | lŭbdŭp | lŭbdup | &c., give, perhaps, as good an idea as any that have been devised. The first sound coincides with the beat of the point of the heart against the walls of the chest, it is almost, if not quite, synchronous with the radial pulse, and its maximum of intensity is between the fourth and fifth rib below and a little outside the nipple, or rather about an inch above the point where the apex of the heart strikes against the thorax. The second sound, clearer, shorter, and more superficial, is produced after the pulsation of the arteries, and its maximum of intensity is pretty nearly on the level of the third rib, a little above and on the right of the nipple, towards the left border of the sternum.

The sounds of the heart are repeated in pairs, and in the following order: first, the dull sound coinciding with the stroke of the heart against the chest-wall; then a very short interval of silence; then the clear sound, and then a longer interval of silence. The relative periods of time occupied by these events have been variously estimated by different observers, some dividing the whole rhythmic period into four parts (Volkmann), of which the two sounds together occupy two, and the interval the remainder; while others divide it into three parts (Barth and Roger), of which the first sound occupies rather less than a third, the short interval of silence and the second sound each a sixth, and the long period of silence rather more than a third.

It cannot be too strongly impressed upon the young auscultator, and also especially on those whose duty it is to examine and report upon healthy lives, that these sounds may vary much in their intensity or pitch, and in the extent to which they may be heard, within the limits of health. It has very often fallen to my own lot, as the superintending physician of a large Life Assurance Office, to examine and report upon the condition of the hearts of individuals which were supposed to present some abnormal peculiarity, whereas, in many cases, the organ was really quite healthy, the error having arisen from mistaking a well-accented sound for a *murmur* or morbid one, or by neglecting to make allowance for the extent to which the sounds may be propagated by the physical conformation of the chest, or the peculiar constitution of the individual. Thus the sounds are louder in nervous subjects, with a narrow chest, than in those who present the opposite physical conditions; and they are also increased in intensity when any accidental cause temporarily accelerates the circulation, and, generally speaking, their loudness is in direct proportion to the energy and rapidity of the heart's contractions. The *extent* to which the sounds are heard also varies within the limits of health in different individuals; thus in an adult, moderately stout, they are heard distinctly over the region of the heart, then they grow gradually weaker as the auscultator removes his ear from this part, but in fat subjects they are confined within narrower limits, and in lean ones, on the contrary, they are heard more extensively, and may even be heard pretty clearly in the back towards the right side. The sounds, too, are rather clearer in young subjects than in old ones, and even a distended stomach will render them clearer than when the stomach is empty. Disease in other organs will also affect the intensity of the sounds of the heart, which may itself be healthy, as, for instance, where a tuberculous lung may render the heart-sounds more clear, or an emphysematous one may make them more feeble and indistinct; and all these matters must be carefully considered in forming an opinion as to the nature of the sounds, and as to the healthy or morbid conditions they may respectively indicate.

The *causes* of the sounds of the heart have been sought for in some of the actions which occur during its rhythmical movements. A great variety of theories has been proposed, and many have been rejected as being inconsistent with the ascertained sequence of the cardiac functions. Thus Laennec attributed the first sound to the

contraction of the ventricles, and the second to the contraction of the auricles; at least, such is the inference to be drawn from his own words, although Andral endeavours to show that he has been misrepresented in this respect. Laennec states, indeed,[1] that the first sound *corresponds* to the contraction of the ventricles, and the second *corresponds* to the contraction of the auricles, and therefore, in this sentence, he does not actually state that the two sounds are *caused* by those actions, but in the succeeding parts of the same chapter he clearly states that the sound and the contraction are to be considered as identical, or at least synchronous. For instance, he states that the sound of the contractions (*le bruit des contractions*) is similar on the two sides; that in certain specified morbid conditions the flapping (*claquement*) of the auricle is much less loud than in the natural state; that in other conditions, the sound of the auricle becomes more feeble; that in other conditions, again, the sound produced by the ventricles is clear and rather sonorous, and resembles *that* of the auricles (*il se rapproche de la nature de celui des oreillettes*); that in healthy subjects, having hearts with rather thin walls, the contraction of the auricles *is heard* (*s'entend*) more *loudly*, &c.; that sometimes the contraction of the auricle produces only a dull *sound*, &c.

It cannot therefore be doubted that Laennec did really attribute the second sound to the contraction of the auricles, as he attributed the first to the contraction of the ventricles. That his theory of the production of the first sound is partially if not wholly true, will be presently shown, but it is expedient to state at once that the theory of the second sound cannot be true in the face of the accurate observations which have been made since the time of the illustrious author. In point of fact, the second sound does *not* correspond with the contraction of the auricles, and therefore cannot be caused by it. The second sound follows almost immediately after the contraction of the ventricles, and *before* the auricles have begun to contract, and the latter event occurs during the period of silence intervening between the second sound and the recommencement of the first sound.

Some of the circumstances causing the sounds are attributable to the action of the heart itself, some to the friction excited by the passage of the blood, some to the mutual action exerted between the heart and surrounding parts. A detailed account of all the explana-

[1] 'Traité de l'Auscultation Médiate.' Vol. iii, chap. iii.

tions offered on these points would be, in the present day, an unprofitable task, but the following analysis will exhibit, in a brief and compendious manner, some of the principal opinions which have been entertained and expressed on the subject.

Dr, now Sir Dominic Corrigan,[1] attributed the first sound to the entrance of the blood into the ventricles in consequence of the contraction of the auricles, and the second to the collision of the internal surfaces of the ventricle, at the end of the ventricular contraction. But there are several objections to this view, and among others, that the first sound is perfect while the auricles are motionless, and that the second sound is not coincident with the ventricular contraction. Dr Hope, in 1831, attributed the first sound to the collision of the molecules of the blood during the systole, and the second to the same kind of collision during the diastole,[2] but in 1839[3] he came much nearer to the true explanation, by referring the first sound to the tension of the valves, the sound of muscular extension, and the rotatory sound during the systole; and the second sound to the closure of the aortic valves during the diastole. Majendie, in a memoir read at the Académie des Sciences, in 1831, explained the first sound, with some show of reason, by the impulse of the apex of the heart against the chest at the moment of ventricular contraction; and, less successfully, attributed the second to the impulse of the anterior surface of the heart during the dilatation. Bouillaud[4] was not very far wrong in attributing the first sound to the sudden elevation of the auriculo-ventricular valves and their collision against one another, and the sudden depression of the aortic and pulmonary valves during the systole; and in explaining the second sound by the elevation of the aortic and pulmonary valves and the collision of their opposite surfaces, and the sudden depression of the auriculo-ventricular valves during the diastole. Skoda, of Vienna, whose notions on this and other auscultatory phenomena are eccentric, though often highly ingenious, supposes that the first and second sounds of the heart are produced simultaneously in both ventricles and in the aorta and pulmonary artery, both of them in each of these four points at the same time. He attributes the sounds to various causes, such as the impulse of the blood against

[1] Transactions of the College of Physicians of Ireland.
[2] Hope on 'Diseases of the Heart'
[3] Ibid., third edition, 1839.
[4] Bouillaud, 'Traité Clinique des Maladies du Cœur,' second edition.

the valves, the impulse of the apex of the heart against the walls of the chest, the impulse of the blood against the walls of the aorta and pulmonary artery, the retrograde impulse of the arterial blood against the aortic and pulmonary valves, all these sounds acting together, although the sounds are heard consecutively.[1] Dr C. J. B. Williams, again, not unsuccessfully, attributes the first sound to the muscular contraction of the ventricles, and the second (although here he is, perhaps, not so successful) to the retrograde impulse of the arterial currents against the semilunar valves during the diastole.[2] The Dublin Committee, which met for the determination of this question in 1835, confirmed the views of Dr Williams as to the cause of the first sound, and referred it to muscular contraction and the friction of the blood against the walls of the ventricles during their systole; and this Committee referred the second sound to the tension of the semilunar valves of the aorta and pulmonary artery during the diastole. As the Dublin Committee did not consider that all the questions proposed for investigation had been definitely settled, a Committee was subsequently formed in London, in 1836, which confirmed the results previously obtained. This Committee found that the first sound was due to several causes, and among others to the sudden muscular tension of the ventricles during their contraction, and the impulse of the heart against the chest; and the second to the sudden closure of the semilunar valves (aortic and pulmonary) by the arterial columns.

All of these explanations as to the causes of the sounds are ingenious, and several of them are true, and the discrepancy existing between them is reconciled by the general admission which now prevails that the first sound, at least, is compound in its nature, being produced by a number of causes. The circumstances which accompany this sound are the muscular contraction of the ventricles, the impulse of the heart against the walls of the chest, the sudden elevation of the semilunar valves against the walls of the arteries, the sudden tension of the mitral and tricuspid valves, the collision of the blood against the valves, the reciprocal influence of the corresponding surfaces of the valves, the molecular collision of the blood compressed and propelled towards the orifices, and the friction of the blood against the walls of the ventricles. The second sound, as will be immediately noticed, is far more simple in its nature.

[1] Skoda on 'Auscultation.'

[2] 'The Pathology and Diagnosis of Diseases of the Chest,' 1840, p. 295.

The efficient causes of the first sound seem to be—1. The muscular contraction of the ventricles; for the sound may be perceived, though less distinctly, when the heart has been removed from the body, and emptied of its blood, as was proved by the London Committee. 2. The impulse of the apex of the heart against the chest; for when the walls of the chest, against which the apex usually beats, were removed, the sound was very much diminished in intensity, though not extinguished. 3. The tension of the auriculo-ventricular valves: inferred to be a cause of the sound from the analogy in function of these valves with the semilunar valves, although their closure is not nearly so sudden as that of the latter. 4. The impulse given by the blood to the lower surface of the semilunar valves, and to the base of the arterial columns of the aorta and pulmonary artery: a view supported by experiments made upon tubes out of the body, and by comparing the intensity of the sound at the base of the heart where it was loudest, and over the region of the auriculo-ventricular valves where it was inaudible.

The principal phenomena which accompany the second sound are the dilatation of the ventricles and the collision of the blood which flows into their cavities, the sudden depression of the auriculo-ventricular valves, the sudden tension of the semilunar valves, and the retrograde impulse on their upper surfaces of the columns of blood thrown into the aorta and the pulmonary artery. It is now generally admitted that the second sound of the heart is produced exclusively by the closure of the semilunar valves, for in the experiments made on the lower animals it was found that when the valves of the aorta and of the pulmonary artery were simultaneously hooked up so as to prevent their closure, the sound was invariably absent, but returned when the valves were again liberated.

I should mention here that Andry, Dalton, and some other physiologists believe that the first sound has a similar origin with the second, and that it is dependent altogether on the closure of the auriculo-ventricular valves,[1] just as the second sound is caused by the closure of the semilunar valves (pulmonary and aortic). The first sound, according to this view, is longer, because the auriculo-ventricular valves being connected by the tendinous cords (*chordæ tendineæ*) to the muscular walls of the heart require some time to be raised and approximated by the action of the blood passing between

[1] 'A Treatise on Human Physiology,' by John C. Dalton, jun., M.D., Third edition. Philadelphia, 1864, p. 271.

the muscular walls and the valves; whereas the semilunar valves have free borders, and consequently stretch suddenly across the arterial cavities. The opinion that the first sound, as well as the second, is a valvular one, appears now to be very generally entertained, and Dr Carpenter, in the fourth edition of his 'Manual of Physiology' thinks that, although the tension of the auriculo-ventricular valves may not be the sole cause of the sound in question, it is probably the principal one.[1]

In reference to the causes of the sounds of the heart, I think it is right to allude to some observations made on this subject by Dr Leared,[2] which, although they are not altogether free from objection, are characterised by great ingenuity, and certainly explain some circumstances which are otherwise difficult of interpretation. Dr Leared adduces several experiments to show that the sounds in question may be caused by the blood alone, without any necessary co-operation of the solid structures. He passed an intermittent stream of water from an india-rubber bottle outside into a reservoir full of water, and on placing a stethoscope in the fluid near the point where the intermittent stream was propelled through the larger body of fluid, he found that a blowing sound was produced. But on filling the reservoir with glycerine instead of water, and performing the same experiments, the sound was produced with great difficulty, and hence he argues that the viscidity of the fluid has a tendency to deaden or annihilate the sound. In applying this view to disease, he refers to the fact that in anæmia, where the blood is attenuated, there is frequently a murmur heard over the great vessels, and this murmur disappears as the blood regains its consistency. In some other experiments he shows that when fluids are subjected to pressure while passing through tubes, no sound is produced, but when this pressure is diminished then sound becomes audible. For instance, let a tube be inserted in a perpendicular manner at the bottom of a large cistern, and its extremity be immersed in water; if the cistern be filled with water, and considerable pressure exercised on it as it flows through the tube, little if any sound is produced; as, however, the water in the cistern diminishes, a low continuous sound is developed.[3] Hence he argues that when a full stream of blood is passing through the

[1] Op. citato, p. 417.

[2] 'On the Sounds caused by the Circulation of the Blood,' by Arthur Leared, B.A., M.D. Dublin, 1861.

[3] Op. cit., p. 7.

arteries and veins, no sound is heard over those vessels because their walls are acting vigorously on their contents, but when the blood itself is attenuated, and the vascular tension is feeble, as in anæmia (more properly *spanæmia*) then a sound is heard both in arteries and veins. Whatever may be thought of Dr Leared's theory in other respects, it is certainly applicable to the phenomena of spanæmia. In a state of health, Dr Leared believes that four conditions are necessary for the production of the perfect first sound, namely, a due degree of consistence of the blood, sufficient force in the circulation, sufficient pressure upon the blood, and the absence of obstruction at the outlets of the heart; and that the mechanism of the second sound closely resembles that of the first. According to his view, the first sound is caused by the blood which is driven from the ventricles coming into forcible contact with the blood in the aorta and pulmonary artery, the impact between the two masses of fluid giving rise to the sound; and the second sound has a similar origin, but is shorter and sharper because the separation between the opposing columns of blood is more complete.

CHAPTER IV

THE PHYSICAL EXAMINATION OF THE HEART IN HEALTH

THE volume of the heart or the space which it occupies, the thickness or thinness of its walls, and the manner in which it performs its functions, are determined by various kinds of physical exploration, as by the sight, by palpation, by percussion, and by auscultation.

1. INSPECTION

On *looking* at the chest of a healthy person, it will be observed that the two sides of the chest are symmetrical, and that the apex of the heart beats on the left of the sternum, between the fifth and sixth ribs. It must be noticed, however, that the pulsations of the heart are *seen* better in proportion as the subject is thinner, and in robust or fat persons the pulsations in health may not be seen at all. On the other hand, when the heart is enlarged and thickened, and its action excessive, its beats may be easily seen, and thus the visible pulsation of the heart often becomes an important indication of disease.

2. PALPATION

Palpation, or feeling by the hand, is a very important measure for ascertaining the condition of the heart. When the organ is healthy, and the subject is not very fat, it will readily be felt to beat with a soft and moderate impulse between the fifth and sixth ribs, immediately to the left of the sternum. If the subject is muscular and robust, the impulse will be felt less plainly than when he is thinner, and when he is very fat it may be difficult to perceive the pulsation at all. The mind is quite unconscious of the beatings of the heart when the organ is healthy, and it is mercifully arranged that it should be so, for if each person were conscious of every movement of the chief organ of the circulation, continual discomfort and

apprehension would ensue. It is chiefly in cases of exalted sensibility, and in some forms of hypochondriacism, not amounting to actual disease, that the movements of the healthy heart become perceptible to the individual, and what is worse, when the pulsations are intermittent, as they often are under such circumstances, each intermission is painfully self-evident.

3. Percussion

The percussion of the præcordial region in the healthy state gives, of course, a dull sound, but not commensurate in extent with the size of the heart, and not uniform in all cases. The heart is, in fact, overlapped by a portion of the left lung, and hence the dulness is more or less modified by the amount of spongy and air-containing tissue by which it is covered. As the result of numerous observations it is generally admitted that the natural dulness of the præcordial region extends only about two inches in every direction, beginning above at about the fourth rib and extending downwards to the sixth, having its maximum at the centre of this region and gradually becoming less and less marked, till it disappears on both sides, where it is lost in the clear lung-sound. Below, and on the left side, the dulness is replaced by the hollow sound of the stomach, and on the right side, inferiorly, it is sometimes continuous with the dulness of a portion of the liver.

4. Auscultation

By auscultation, either mediate or immediate, the *sounds* of the heart are heard. This mode of investigation also gives information as to the impulse of the organ, for when the latter action is increased, the stethoscope is moved, and the head of the auscultator is moved likewise; but as the hand or any other part of the body of the observer would be equally affected by this cause, the application of the ear is not specially valuable in measuring the impulse of the heart. But auscultation is essential in estimating the nature of the sounds elicited by the cardiac movements. In a healthy state of the heart, these sounds are two in number, and, although very easily recognised, they are very difficult to describe in words. Perhaps the combination of syllables *lubb dup* (see p. 35), the first syllable being longer than the second, a formula devised by Dr C. J. B. Williams, is the nearest approach to a written explanation of the sounds. These sounds are consecutive and are called respectively the *first* and *second* sounds of

the heart, the first being more dull than the second, and both are repeated several times in the minute, the variations in the number being of course identical with those of the pulse at the wrist. The first sound is longer than the second, and coincides with the impulse of the apex of the heart against the thoracic walls, immediately preceding the radial pulse; the second sound is clearer, shorter, and sharper than the first, and is heard after the pulsation of the arteries. As to the rhythm of the sounds, they take place in pairs, the first dull sound coinciding with the impulse of the heart, and being succeeded by the clear and short sound. To be strictly accurate, the first sound is succeeded by a very short interval of rest, during which the arteries pulsate; then comes the clear and short second sound, and lastly a longer interval of rest precedes the recommencement of the rhythmic movements. The relative periods of time occupied by this succession of actions and of repose, supposing the whole to be divisible into three parts, would be expressed by representing rather less than one third to be occupied by the first sound, one sixth to be occupied respectively by the short interval and the second sound, and rather more than a third by the long interval of rest. Some authorities, however, divide the whole period in a somewhat rougher manner, into four parts, nearly two being occupied by the first sound, one by the second sound, and one by the final interval of repose; and it should be mentioned that the very short interval of repose succeeding the first sound is inappreciable when the pulse exceeds the rate of sixty in a minute. Other and more minute subdivisions of the heart's rhythm have been proposed, but they are practically unnecessary.

It is important to recollect that the first sound is generally called also the *systolic*, being synchronous with, and in some measure caused by, the contraction of the ventricles (συστολη, contraction) and is heard best over the apex of the heart; and that the second sound is called also the *diastolic*, being synchronous with, although not caused by, the dilatation of the ventricles (διαστολη, dilatation) and is best heard in the middle of the sternum.

It should be very distinctly understood that the extent to which the impulse of the heart is felt, or its sounds are heard, and the greater or less distinctness of the sounds, may vary most considerably, even within the limits of health, in different individuals; and moreover it may happen that the extension of the sounds and of the impulse of the heart may be due in reality to disease of some

other organs, as the lungs, the heart itself being perfectly healthy. Thus it often happens that a tuberculous lung, acting as a conductor of sound and of impulse, may give rise to suspicions of enlargement of the heart, which however may be really of normal structure; and it is well known that an increased degree of clearness of the heart's sounds over a considerable space of the thorax is one of the most valuable stethoscopic signs of pulmonary tubercle. On the other hand, an emphysematous lung, by the clearness of the sound it elicits on the percussion of the chest, and by its non-conducting power, may so mask the impulse and the sounds of a diseased heart as to lead to the erroneous conclusion that it is healthy. Again, within the limits of health, in fat subjects the sounds will be restricted within a narrower space than in persons of normal proportions; and, on the other hand, both the impulse and the sounds will be perceptible over a very wide extent in thin persons with a contracted thorax, in whom the sounds may often be heard at the back of the chest. The intensity of the sounds is also somewhat greater in young persons, and somewhat less in the aged.

It is impossible to insist too strongly upon the necessity of a careful consideration of all these circumstances in endeavouring to come to a correct conclusion respecting the healthy condition of the heart.

CHAPTER V

THE PHYSICAL EXAMINATION OF THE HEART IN DISEASE

1. Inspection

On looking at the chest of a person with disease of the heart, no difference in appearance will be observed unless the heart is thickened and its impulse much stronger than in the natural state; and, it should be added, unless, as happens in some very rare and exceptional cases, the sternum is deficient, and thus allows the pulsations even of the healthy heart to be visible. Supposing the bony structure of the chest to be normal, the subject to be of moderately robust conformation, and to be free from any accidental cause of excitement, the appearance of the heart beating against the walls of the chest and elevating them more or less by this cause, will indicate, in all probability, the existence of what is called hypertrophy of the heart. But it is obvious that this sign is not of great importance in itself, although it may afford collateral evidence when taken in connection with other and more trustworthy indications.

2. Palpation

Palpation affords very valuable evidence in estimating the existence of certain diseased conditions of the heart. It has already been mentioned that the natural impulse of the organ will be perceived by applying the hand or the fingers between the fifth and sixth rib on the left of the sternum, and this natural impulse may be greater or less in different individuals even in the healthy state. But in certain morbid conditions of the organ, as when the heart is increased in bulk and its walls are thickened, the impulse is proportionately augmented. When there is, moreover, a diseased condition of one or more of its apertures, the beat of the heart communicates to the hand a powerful thrilling sensation very readily to be recognised by the practised observer, although very difficult to describe in words. The name given by Laennec to this thrill or vibration was *frémissement cataire*,

literally, *cat-tremor*, and it is compared by him, very justly, to the sensation conveyed to the hand when applied to the back of a cat when it is purring. It is therefore often designated in English as the *purring tremor*. The symptom itself was first observed by Corvisart, who regarded it as a symptom of ossification of the valves, and especially of the mitral valve; but Laennec asserts that he has observed it frequently in cases where there was no disease of the heart whatever. This view, however, is strongly contested by Andral, and by all subsequent authorities on disease of the heart, and indeed there can be no doubt that Laennec's statement on this point is erroneous, and that the existence of the purring tremor really indicates a morbid condition of the heart. Laennec attributed the sensation, the exact nature of which he admitted he was unable to explain, to "a particular modification of the innervation."[1]

The opinion of Corvisart that the *frémissement cataire* is a symptom of ossification of the valves of the heart, is true to a certain extent, but it does not express the whole truth, for many cases of disease of the valves, including ossification, do not present this symptom, and, moreover, the existence of the purring tremor is by no means a special indication of disease of the mitral valves. Andral, in a note appended to the observations made by Laennec on this subject, declares that he has always found, in the examinations made after death of those in whom he has observed, for some considerable period, the thrill in question, some lesion, either in the orifices of the heart or in the pericardium, which has accounted for the symptom. This lesion was either ossification of the valves rendering their surface unequal, or some considerable thickening of these membranous folds, or it consisted in an inflammatory disease of the pericardium, giving rise to the production of false membranes, which by their attrition against the folds of the serous membrane gave rise to the thrill.[2] The latter lesion being curable, the *frémissement cataire* may become indistinct, and finally cease altogether, and Laennec may have probably had such cases in his mind when he attributed the thrill to merely nervous causes. Dr Hope, who investigated the nature and the causes of the purring tremor with great care, more especially in connexion with the existence of morbid cardiac sounds or murmurs, arrives at the conclusion that the phenomenon is due to modifications in the motion of the blood

[1] Laennec, 'Traite de l'Auscultation Mediate.' Vol. iii, p. 130, 1837.
[2] Op. citato, p. 123.

occasioning increased friction and vibration. It will be observed that Dr Hope by no means attributes the *frémissement cataire* exclusively to valvular disease, but he considers that it may be due to altered conditions of the blood itself independent of structural lesion. In order to determine the physical symptoms caused by loss of blood, Dr Hope made, in connexion with the late Dr Marshall Hall, a series of experiments on dogs, and, among other results, he found that after the animals had been bled several times, a thrill and throbbing became perceptible over the whole body, and upon a repetition of the bleeding the *frémissement cataire* became excessive. There can be little doubt that the explanation of this peculiar thrill or vibration, although it is not yet perfectly satisfactory, is that it depends upon an unfilled state of the vessels or of the cavities of the heart, and that it may therefore be caused by regurgitation through the cardiac valves, or by any condition of the blood itself (independently of cardiac disease) which prevents it from completely filling the cavities of the heart or the interior of the vessels. Thus the *frémissement cataire* is felt in cases of anæmia, or it may even be produced artificially in a large artery in a healthy subject by pressing upon it, the thrill not being felt at the point at which pressure is made, nor between it and the heart, but at the opposite point, where of course the artery is *unfilled*.

But independently of anæmic conditions of the system, or of artificial methods of developing the thrill in question, it may be concluded that, in relation to the morbid conditions of the heart, the presence of the *frémissement cataire* is due, as has already been observed, to increase in the bulk and thickness of the heart, together with such a diseased state of one or more of its apertures as to permit of regurgitation. It should be observed that Dr Bellingham, whose Treatise on Diseases of the Heart is a most valuable contribution to Practical Medicine, differs from the opinion of Andral by rejecting pericardial inflammation as a cause of the purring tremor. The friction caused by the deposition of lymph between the opposed layers of the pericardium, says Dr. Bellingham, causes indeed a peculiar thrill or vibration, but it has a different character from that of the *frémissement cataire;* it is felt at a different part of the præcordial region, and a somewhat similar feel may be experienced over the region of the lung in cases of pleurisy with false membranes.[1]

For myself, I consider the *frémissement cataire* a very valuable

[1] 'A Treatise on Diseases of the Heart,' by Dr O'B. Bellingham, p. 117

auxiliary indication of disease of the Heart, and, when I have felt it, I have seldom or never failed to discover the other indications proving the existence of the morbid conditions of which the thrill in question is a prominent exponent.

3. Percussion

The percussion of the præcordial region does not afford any very valuable or trustworthy information in disease, except when the heart is considerably increased in bulk, or when the cavity of the pericardium is distended with fluid. As the natural dulness over the heart does not extend more than two inches in any direction, owing to the proximity of the lungs, it is not to be expected that a diminution in the volume of the heart will occasion much difference in the percussion-sound, and an increased resonance over the præcordial region would, in all probability, rather indicate an emphysematous condition of the lungs than any alteration in the dimensions of the heart. But when the dulness of the præcordial region is much increased in extent, the indication becomes valuable, and the principal point to determine will be, whether the sign is due to an enlargement of the heart itself, or to effusion of fluid in the pericardial sac. In practice, there can be no great difficulty in drawing the distinction, because enlargement of the heart takes place slowly and gradually, and is accompanied by symptoms more important than those yielded by percussion; while effusion of fluid, or dropsy of the pericardium, comes on rapidly, and is also accompanied with other symptoms which leave very little room for doubt. Still, it may be noticed that, in the case of effusion, the dulness will occupy a triangular space, the base of which lies on the diaphragm, and the level of the fluid varies sometimes according to the position of the patient.

In a recent monograph, Dr Burresi,[1] an Italian physician, proposes an accurate method of exploring the præcordial region. It is founded upon a plan first devised by De Giovanni, and it consists in taking three pleximetrical points over the præcordial region, drawing lines between them so as to form a triangle, the angles of which are larger in proportion as there is enlargement of one part or another of the heart.

4. Auscultation

Auscultation conveys to the ear the sounds produced by the motions of the heart; and having already explained the nature and

[1] Percussione del Cuore. By Professor Burresi, of Siena, 1872.

the causes of the healthy sounds, I now proceed to describe the modifications or alterations which these undergo under the influence of disease. Auscultation also affords information as to the increased or diminished impulse of the organ, for when the impulse is increased the stethoscope itself is moved by the action, and the head of the observer is moved also; and when the impulse is so much diminished that it cannot be felt by the hand, it may be discovered by the application of the stethoscope. Still the word *auscultation* strictly means the act of *listening*, and the chief object of auscultation, therefore, is to ascertain the nature of *the sounds* conveyed to the ear. But the sounds and the impulse are so closely associated together that it is quite impossible, when using the stethoscope, to separate the one from the other, though it is not difficult to determine when one of them unduly preponderates.

In the normal condition, the maximum of intensity of the first sound is immediately below and a little to the left of the nipple on the left side, and that of the second sound is a little higher, above and to the right of the nipple, and these sounds diminish in intensity in proportion as they are distant from the præcordial region till they are absent altogether on the right side and the back of the chest. Remembering these data, the auscultator will be able to appreciate the abnormal conditions which the sounds present when disease exists. Even when there is an absence of morbid sounds, the healthy ones may be altered in their situation, their extent, their intensity, their pitch, or their order of succession, and it is very essential to observe that these alterations which sometimes accompany or help to characterise serious diseases of the heart, may only result from abnormal conditions of the blood, or may be due to disturbed states of the nervous system, and are occasionally caused even by morbid affections of different organs unconnected with the heart except in a physiological sense.

Thus, for instance, the sounds of the heart and the impulse which accompanies the first sound, will be altered in their seat, and will be perceived above the points just indicated, in cases where the diaphragm is pressed upwards by flatulent distension of the abdomen or by ascites; and they are perceived to the right instead of to the left of the sternum in the well-known cases where the heart is dislocated, or thrust out of its place, by a pleuritic effusion on the left side.

The extent to which the sounds will be heard will also vary very considerably, being sometimes confined to an unusually limited space

over the præcordial region, and sometimes spread over the greater part of the front and even the back of the chest. The diminution in the extent to which the sounds are heard may depend upon the diminished size of the heart, or on the feebleness of its action, or on general weakness of the system, and the same conditions are almost always accompanied by a diminution in the impulse of the organ. On the other hand, the extent to which the sounds are heard and the impulse felt, will often be very much increased by a number of circumstances, some of great pathological importance, and many of no great significance: and the physician must carefully examine not only the sounds themselves, but all the concomitant circumstances of each case, in order to arrive at a correct conclusion. Thus an increase in the extent of the sounds of the heart may denote a dilatation of the organ, which, however, is often accompanied by hypertrophy, and will therefore present other and more characteristic signs. Hence, except in rare instances, a mere increase in extent of the sounds without a corresponding augmentation of impulse, does not denote organic disease of the heart, and is usually indicative only of some peculiar abnormal condition of the nervous system, or is dependent on some disease in adjacent or even remote structures. Nothing, in fact, is more common than to find, especially in thin persons, children, and women, the sounds of the heart audible over a very considerable portion of the front of the chest, and yet there may be no organic or other disease, either of the heart or of the lungs, or of any other structure. It is still more important, however, to recollect that in tuberculous disease of the lungs, or in any other affection which causes consolidation of those organs, the sounds of the heart will be heard over a very extensive space, because the condensed tissue will then act as a conductor of the sound. This is often one of the earliest signs of pulmonary tuberculosis, and in not a few cases the heart has been pronounced to be diseased when the actual malady was in the lungs. There is really some difficulty in the diagnosis in many instances, and although the dulness on percussion, and the presence of bronchial breathing, and bronchophony, and other symptoms, will generally point out the true nature of the affection, yet in the onset of the case even acute stethoscopists may be mistaken.

The intensity of the sounds of the heart also varies very considerably, being sometimes so loud as to be heard by the patient himself, or by persons at a distance, and sometimes so feeble that the ear, when applied to the walls of the chest, can scarcely hear

them at all. In hypertrophy of the heart the first sound is diminished and the impulse is increased; while in dilatation, the first sound is increased and the impulse is diminished. But as the usual morbid condition of enlargement of the heart is a combination of hypertrophy with dilatation, the sounds and the impulse and the extent to which they are perceived, are all increased in the same proportion. Again, the intensity of the sounds is diminished and their extent limited, and the impulse of the heart is enfeebled, in those conditions of the organ in which it is diminished in size and weakened in its action, or, in other words, in those conditions which are the opposite to hypertrophy with dilatation.

It was Laennec who first established the fact that the pulsations of the heart might sometimes be heard at a certain distance from the patients. In 1823 he observed this curious phenomenon in a young girl, and in 1824 he had two patients who exhibited it in a more remarkable degree than in his first case. He never heard the pulsations at a greater distance than from a foot and a half to two feet, and he convinced himself that the sound was that which accompanied the contraction of the ventricles, by its isochronism with the pulse at the wrist. Laennec states that, out of more than twenty patients in whom he had heard the pulsations at distances varying from ten inches to two feet, only about three or four were suffering from organic disease of the heart, all the rest presenting only nervous palpitation, and in all of them the phenomenon was transient. He therefore attaches to it no great pathological importance, but attempts to account for it by a theory which now seems absurd, and was never supported by any facts. He considered it to be due to the effusion of gas in the pericardium.

In a note appended to the last edition of his "Traité de l'Auscultation Médiate," Laennec states that he observed this phenomenon in his own person, the sounds of the heart being audible to himself, and also to others who were in the same room with him. He was ill at the time, and he suddenly felt and heard the contractions. He caused an attendant to place her ear about six inches from the walls of the chest, and she also heard them very distinctly. The reason in his own case was evidently the pressure exercised by a distended stomach, for he felt his own epigastric region inflated by gas, and after an eructation the sounds were no longer perceptible at a distance.[1]

[1] Laennec, 'Traité de l'Auscultation Médiate,' vol. iii, p. 130.

This observation is the more interesting, as it was written by Laennec on his death-bed, and was almost the last that he wrote. In a note appended to Laennec's work by Andral, the latter physician confirms the truth of Laennec's observations, and relates an interesting case in which he found the same phenomenon to exist. The patient was highly nervous and hysterical, and Andral heard the pulsations of the heart at a distance of several feet from the chest. The sounds were heard, however, only during certain hysterical or epileptic attacks, to which the patient was occasionally subject.

Since the time of Laennec the sounds of the heart have been heard by numerous observers at a distance from the chest, but generally in cases where there has been a morbid condition of the heart or of the blood. Dr Elliotson, who was one of the earliest investigators in England of the auscultatory phenomena of the heart, once heard a cooing murmur so loud that it was audible at the distance of nearly a foot from the patient. Dr Hope states that he once heard a sound of this character at the distance of two feet from the patient's chest.

Another curious circumstance occasionally observed in relation to the sounds of the heart is that, instead of the ordinary double sound, there are three sounds, each systolic sound being repeated, the diastolic sound remaining single; or the diastolic sound being repeated, the systolic remaining single. In other cases there are four sounds, the systolic and the diastolic being both doubled; and, on the other hand, it is sometimes observed that only one sound is audible, which is always the first. When a triple sound exists it is most frequently owing to a reduplication of the systolic sound. Dr Bellingham[1] states that in two cases under his care, where the first sound was doubled, he had the opportunity of examining the bodies after death, and he found the right ventricle much hypertrophied and dilated in both cases, and forming the apex of the heart; the left ventricle was normal in one, and slightly dilated in the other, but the valves on the left side of the heart were sound in both cases, and no murmurs had been audible during life.

[1] 'A Treatise on Diseases of the Heart,' by Dr O'B. Bellingham, p. 132.

CHAPTER VI

MORBID SOUNDS OR MURMURS

THE presence of morbid sounds, or *murmurs*, as they are generally called, in the region of the heart, or over the course of the vessels, has always been regarded, since the discovery of auscultation, with the greatest interest, because these phenomena often denote important structural disease, and what is still more important, they are often the only indications which exist, the rational signs being, in very many cases, entirely absent. But nevertheless it would be a serious error to suppose that these sounds are always caused by appreciable alterations of structure, or that they are always heard when more anatomical reasoning would lead us to expect their presence. Although they very often accompany or denote morbid changes in the structure of the heart or in that of the vessels, yet it is constantly found that no appreciable lesion can be detected after death in cases where these sounds have been heard; and, on the other hand, although by no means so frequently, morbid conditions of the heart have been discovered after death when no abnormal sounds were detected during life. Anomalies like these evidently puzzled Laennec, to whom is unquestionably due the discovery of the sounds designated by him *bruit de soufflet* (bellows-sound), *bruit de scie, bruit de râpe* (sawing and rasp sounds), *bruit musical ou sibilant* (musical or whistling sound), *bruit de cuir* (new leather sound), *bruit de diable* (humming-top sound); but who was so little impressed with their pathological signification as to declare that, "among all the phenomena which have been discovered by mediate auscultation, these alone (the bellows, rasp, sawing sounds, &c.) are not associated with any organic lesion in which it is possible to discover their cause."[1] He goes on to state that he has traced

[1] 'Traité de l'Auscultation Médiate,' vol. iii, p. 70.

many cases, both of acute and chronic disease, which presented the bellows murmur during the latter periods of life, and sometimes for several months, in a very obvious degree, both in the heart and arteries, but in which, after death, he failed to discover any organic lesion which constantly coincided with the sounds, or which were not frequently met with in cases where the sounds were never heard. Laennec at first considered the bellows sound of the heart to be a sign of contraction of the orifices, in which conjecture he was partially correct, but he abandoned this idea when he observed that the sound frequently existed in cases where no such contraction was subsequently discovered; and, on the other hand, when he occasionally found cases of ossification of the valves, the existence of which had never been announced by this anomalous sound. It was reserved for the followers of Laennec, and more especially for British physicians, to trace out the real value and significance of the sounds, the existence and the modifications of which he was the first to discover and to announce to the medical world.

The fact is, that the abnormal sounds of the heart, to which the term *murmur* is collectively applied, have a very deep significance in most cases, and their presence always points to some morbid condition, either of the heart itself or of the vessels, or of the blood, or of adjacent tissues or organs. Thus they may be due to obstructions existing at the orifices of the heart, or to imperfect closure of its valves, or to active inflammation inside or outside the heart, or to degeneration of the coats of arteries, or to an impoverished condition of the blood, or to the effects of mechanical pressure on vessels by adjacent structures.

Speaking generally, it may perhaps be said that all murmurs are due to *friction*, which, indeed, in a philosophical sense, occurs in all the actions and motions of the animal body, as well as in all mechanical movements whatever, but which, in the healthy state of the body, as in the well-oiled piston of a steam-engine, is diminished by the presence of a lubricating fluid. In the case of the animal body this fluid is supplied by the natural secretions poured out upon the internal membranes. In morbid conditions of the system this natural friction becomes abnormally exaggerated, either by the presence of tumours or excrescences, or exudations developed from the solid parts, or by the passage of unhealthy fluids over healthy solid structures. Thus, however loud or well-marked may be the morbid sounds developed by inflammation of the pericardium, or by

disease of the aortic valves, they may be equally loud, or perhaps louder, when an attenuated or impoverished blood passes over healthy valves, or through healthy vessels, as happens in most cases of chlorosis and anæmia (*spanœmia*), or even in cases where large quantities of blood have been lost or abstracted. The sounds may also be artificially produced by pressure upon a large blood-vessel.

It is by no means difficult to explain the reason why irregularities on the valves or on the lining membranes of the vessels should give rise to abnormal sounds when these parts are exposed to the friction caused by the passage of the blood. It is a well-known principle in physics that the passage of fluids through pipes is retarded by friction, and the retardation is increased by any irregularities or projections in the interior of the tubes, or even by a curve in the tube. A common illustration of this truth may be roughly offered by the noise caused by the projection of flood-gates against a running stream, or by the noise caused by a river where it makes a sudden bend. But it is not so easy to explain how a morbid condition of the blood, when this fluid pssses over healthy structures, can produce the same kind of sound as healthy blood does when passing over diseased structures. Dr Hope, however, from careful experiments and reflection, offers a complete explanation of this apparent anomaly. Having adduced the physical fact that the friction of fluids in tubes increases with the increase in velocity of the motion of the fluid, he shows that the bellows-murmur was artificially produced in dogs by repeated bleeding, by which, of course, the blood was diminished and attenuated, and he therefore concludes that diminution and attenuation of the blood are circumstances eminently calculated to produce murmurs. For as the weight of the blood is diminished, its velocity, or the facility of its transmission, is increased, and the diminished tension of the arteries allows more latitude for sonorous vibration both of their walls and of the blood; and, moreover, the attenuation of the particles of the blood renders them better calculated for rapid motion, and consequently for the production of murmurs and vibration by collision against each other, and against the walls of the containing vessel.[1]

The morbid sounds of the heart, then, are called *murmurs*, whatever may be their cause or their seat, and the names by which the murmurs are respectively designated are derived from some well-known sounds which the murmurs more or less resemble, as bellows

[1] 'Treatise on the Diseases of the Heart,' by Dr Hope, p. 95.

murmur, sawing, filing, rasping murmur, new leather sound, &c. With regard to their situation they are sometimes *exocardial*, or situated outside the heart; sometimes *endocardial*, being due to various conditions of the valves or of the blood; sometimes they are seated in the arteries, sometimes in the veins. It may be thought, at first sight, that it will be sufficiently easy to distinguish between a friction murmur outside the heart, due to an inflammatory affection of the pericardium, and one situated in the interior of the organ and connected with the v[illegible], or caused by attenuation of the blood. But the distinction is by no means so easy as it appears, a circumstance that may be accounted for by the remark made above—viz., that all these morbid sounds are really due to friction of some kind, either of one surface against another, or of a healthy fluid against an unhealthy surface, or an unhealthy fluid against a healthy surface. Skoda, in fact, whose views on auscultation, as on all other subjects, are marked by strong originality and singular independence, says: "I know of no sign by which the friction-sounds of the pericardium can be distinguished from the internal murmurs of the heart excepting this, that the internal murmurs correspond pretty exactly to the rhythm and to the natural sounds of the heart, while the pericardial friction-sounds seem to follow upon the movements of the heart;" and as the sounds and movements of the heart are coincident, he goes on to observe that "this distinctive sign is only available when the murmur is somewhat prolonged: if it be of short duration, we cannot determine whether it is endocardial or exocardial."[1]

The following diagnostic distinctions, however, between pericardial friction-sounds and endocardial murmurs are laid down by Barth and Roger in their 'Traité Pratique d'Auscultation,' and they deserve a careful consideration in all cases likely to be attended with doubt. The bellows murmur (endocardial) is often heard at the origin of the great vessels, while the friction sound (pericardial), when it is limited, is generally seated lower down towards the apex of the heart. The bellows murmur is generally heard over a rather considerable extent of the præcordial region, while the friction-sound is sometimes very much circumscribed; and it may happen that immediately in the vicinity of its greatest intensity the normal sounds are heard. Then the bellows murmur appears more deeply seated, and does not change its position; while the friction-sound is more superficial and peripheric, and may

[1] 'A Treatise on Auscultation and Percussion,' by Skoda. Translated by Dr Markham, p. 219.

change its place, being one day more marked on the right side, and another day more distinct on the left. The bellows-murmur is most commonly simple, and always associated with the same period of the heart's action; while the friction-sound is most frequently double, and when single it is less exactly synchronous with that one of the two sounds to which it belongs. The bellows murmur is often prolonged into the carotid arteries, but this is never the case with the friction-sound. Finally, the permanent bellows murmurs are scarcely ever subject to any but slight variations of intensity, and, if they change their character, it is only after long intervals; while the transformations of the friction-sounds are generally much more rapid. MM. Barth and Roger add the practical remark, that the bellows murmur and the friction-sound may be, as indeed they often are, combined, and then the coincidence is manifested by the simultaneous existence of the characters peculiar to each of these abnormal sounds.[1]

The above distinctions between the bellows murmurs and the friction-sound are very ably drawn, and some of them are exceedingly valuable in a practical point of view, while others are of so refined a character that it would perhaps be difficult to realise them at the bed-side of the patient. But it is hardly necessary to insist upon their absolute truth, because, in the first place, as MM. Barth and Roger admit, the two are sometimes combined together; and, in the second place, the diagnosis in cases of difficulty will be greatly facilitated by a careful examination of all the collateral circumstances. No great practical evils can result from the difficulty of distinguishing in all cases between an exocardial and an endocardial sound, because the former can exist only in acute cases, while the latter may be common to acute and chronic cases alike; and while the bellows murmur and the friction-sound may for a time exist simultaneously in cases of acute disease, a *persistent* murmur can be caused only by chronic disease within the heart.

It must be repeated that all the morbid sounds or murmurs are to be attributed to *friction*, either of one solid part against another, or of the blood against a contracted orifice, or against a tumour or excrescence upon a valve; or it may be due to the vibration of the blood against the walls of an unfilled cavity, or to that of attenuated blood against a weak or relaxed vessel. Thus,

[1] Barth and Roger, 'Traité Pratique d'Auscultation.' Quatrième édition, p. 466.

then, a murmur may indicate, according to circumstances, an acute attack of pericarditis, an obstruction of the cardiac valves, regurgitation of the blood through valves imperfectly closed, friction of the blood against projecting growths or tumours, or the passage of impoverished blood (as in anæmia and chlorosis, or after excessive blood-discharges) through relaxed valves and vessels. It is also stated by some writers that the murmurs may sometimes be due merely to certain abnormal conditions of the nervous system, as hypochrondriasis and hysteria, without appreciable disease of the heart or of the blood; but I am unable to confirm this statement by my own experience, and the phenomenon would be certainly opposed to the results of ordinary medical observation.

But although in a general sense it may be affirmed that all the murmurs are due to friction, and are therefore acoustically the same, yet it is indispensable to examine them individually in order to determine their relative importance in the diagnosis of disease. It should also be repeated that the practitioner, in forming his opinion, ought to take into consideration all the concomitant circumstances of each case, and not to trust too implicitly to the evidence afforded by auscultation; for if he were to judge exclusively by the character of the murmur, he would be liable to fall into as many mistakes as another who should discard the significance of the murmurs altogether and rely entirely on the rational signs.

1. *Friction-Sound—Exocardial Murmurs.*—In a healthy state, the adjacent surfaces of serous membranes glide over one another without any noise, in consequence of the presence of serous fluid thrown out on their surfaces for the purpose of diminishing friction, on the same principle that grease is applied to the axle of a wheel, or oil to the piston-rod and other movable working parts of a steam-engine. But when the natural serous secretion of the animal membranes is dried up, or when lymph is poured out from the contiguous surfaces, then the two sides of the membrane grate against each other, and produce a creaking noise, exactly analogous in its nature to that produced by an ill-greased axle. The sound heard under such circumstances in the animal body, on the application of the stethoscope, is known by the name of *friction*, or *rubbing* sound. This sound presents various degrees of intensity, being sometimes *soft*, like the rustling of silk; sometimes *rough*, like the creaking of new leather, to which it is indeed compared

(*bruit de cuir neuf*) and sometimes *scraping*, like that produced by a scraper or rasp. The first sound indicates an effusion of lymph of recent occurrence, soft in consistence and small in quantity; the second indicates that the effusion is older, thicker, more unequal, and more resisting; and the third, or the scraping sound, is caused by the attrition of more hardened morbid products, such as cartilaginous or osseous deposit in the false membranes resulting from inflammation.

The pericardial friction-sound is generally double, accompanying the two movements of the heart, but it is usually more distiuct in the systole than in the diastole, though the reverse is sometimes the case. Its duration, although never very long, is variable, being sometimes only for two or three days, and sometimes from one to two weeks, and sometimes it disappears for a few days and then returns, and finally ceases altogether. The pericardial friction-sound is the same in character with the friction-sound of the inflamed pleura, and the conditions are the same in both cases, but the situation of the sounds is of course different, the pericardial friction-sound being confined to the præcordial region; and while the one is synchronous with the movements of the heart, the other coincides with the acts of respiration. Hence, therefore, arises the diagnostic distinction, and it is further to be noticed that in the greater number of cases of pleurisy, the movements of respiration are made with very great pain, and are accompanied by cough, or efforts to suppress cough; whereas it frequently happens that there is no pain complained of by the patient in pericarditis. It is therefore the more necessary to observe the physical signs of the latter disease, which are often the only trustworthy indications, and the only guides to a correct diagnosis.

The pericardial friction-sound, heard under some well-defined conditions, is a certain indication of acute inflammation of the pericardium, and is necessarily only temporary in its duration, and will be modified in proportion to the progress of the disease. It is produced at an early period of the inflammation, when an effusion of albumino-fibrine has taken place, but, when the quantity of the effusion is still small, the sound may be only that of a slight rustling. As the effusion increases, the sound becomes more rough, but when the effusion is still more considerable, the sound may diminish instead of increasing, because the effusion separates the opposed surfaces and prevents them from rubbing together; and it

ceases entirely if fluid effusion exists in such quantity as to distend the pericardial sac. It may reappear when absorption begins and the fluid effusion diminishes in quantity, and when consequently the opposed surfaces of the serous membrane are again brought into juxtaposition with one another and with the false membranes which the inflammation has developed; and, when the false membranes have become hard and of a cartilaginous consistence, the sound may become loud and scraping. The friction-sound will cease quickly if extensive adhesions are rapidly formed, because in that case the surfaces are glued together and cannot move on one another, or the sound will last longer if the adhesions are formed more slowly. The friction-sound will quickly disappear when a large effusion of serous fluid takes place rapidly in the pericardium, constituting dropsy of that membrane, and its disappearance will therefore be, in that case, the reverse of a favourable indication; it will be, in fact, one of the worst signs that can present itself. The pericardial friction-sound, therefore, although a most valuable diagnostic sign when it is present, is necessarily temporary, and can exist only where there is a comparatively small quantity of effused lymph on the anterior surface of the heart in the cavity of the pericardium. When there is adhesion of the adjacent surfaces of the membrane, or when there is dropsy of the pericardium, the friction murmur will alike disappear, but its disappearance is a far more dangerous indication in the latter case than in the former.

It should be observed, in justice to the merits of British medical science, that the friction-sounds of pericarditis were first distinctly described, and their true significance in diagnosis was pointed out, by Dr Stokes, of Dublin, who also originally classified them in three divisions—namely, the slight friction-sound, the rasping sound, and the new leather sound. It is to be regretted that in this, as in many other respects, the claims of British stethoscopists have been unduly disregarded by some continental writers on Auscultation, for although M. Collin had previously described the *bruit de cuir neuf*, or new leather sound, it was reserved for the British physician to indicate with exactness the pathological significance of this and other allied sounds in cardiac disease.

2. *Endocardial Murmurs—Bellows, Rasp, Sawing Sounds, &c.*—These sounds are of various kinds, being sometimes soft and blowing, constituting the true bellows sound; sometimes rough, like that

of a file; and sometimes sharp, like that of a saw. I do not myself know that these varieties of sound are respectively allied to any definite and distinct pathological conditions of the heart, or of the vessels, or of the blood, and I believe that they are not; and moreover, the intensity or loudness of the sound is by no means necessarily proportionate to the gravity of the disease. Indeed, the contrary is often the case, a very slight obstruction sometimes causing a loud murmur, while a very serious deviation from the healthy state may produce only a very slight one. It is almost unnecessary to insist that the presence of a murmur, however slight, is a pathological indication of great diagnostic importance, although it does not necessarily indicate the presence of a serious disease. Generally speaking, it is a far more significant indication of diseased structure in the male than in the female subject, for in the latter it is very often caused by abnormal conditions of the blood, without any appreciable lesion of the heart, and may disappear altogether as the general health improves. It should also be observed that although a murmur in the male subject may be due to serious disease of the heart or the arteries, yet it may also be caused solely by the pressure of some gland or tumour, or solidified lung, over the course of an artery, as not unfrequently happens in the case of the subclavian artery.

I am aware that some French writers, as Laennec and Bouillaud, and also Barth and Roger, consider that the rough murmurs, such as the rasp, file, and sawing sounds, indicate a more serious disease than the bellows sound, and usually denote a more hardened or osseous state of the valves over which they are heard. But in practice these distinctions cannot be maintained, for the rough sounds are often heard when the valves have undergone no calcareous degeneration, and, on the other hand, a very advanced stage of this degeneration may produce only a bellows-sound.

As an instance of the comparatively slight disease which may produce a very loud murmur, I here quote the following case which occurred to Dr C. J. B. Williams, and is recorded by him in the second volume of the 'London Journal of Medicine,' 1850. A man, aged about thirty, was admitted into University College Hospital for a slight ailment, attended with palpitation. The remarkable feature of the case was a very loud musical murmur, of a cooing character, following the second sound; it was most distinct in the mid-sternum, but it was also heard in every part of the chest, in the arteries of the neck, and, although faintly, in the radial artery at the wrist. The

patient was kept in the hospital, not because his health was suffering, but as a kind of stethoscopic curiosity. But it unfortunately happened that he caught typhus fever, which was prevailing at the time, and he died in the hospital. On examining the body the ventricles were found moderately enlarged and thickened, but the valves were all healthy, except the aortic, and the only morbid appearance on them was that one of them had its free margin neatly retroverted, so as to leave a fine smooth chink for regurgitation.[1]

It has already been shown that morbid cardiac sounds in general are all due to *friction*, and in describing the exocardial sounds, or those developed in acute pericarditis, the explanation was given on ordinary mechanical principles, namely, that the sounds were the same both in nature and causation as those produced by an ungreased axle, or by the rubbing together of two pieces of new leather, or by the rustling of silk, or by many other equally familiar operations. But the explanation of the endocardial or bellows murmurs, although equally based upon philosophical principles, lies somewhat deeper than that of the exocardial sounds, and must be sought in a combination of arguments and reasonings deduced both from the laws of hydraulics and from the results of observation and experiment on the living body. It is found, accordingly, that the passage of fluids through pipes is attended with a certain amount of friction, depending in degree upon the greater or less smoothness of the pipe, the friction being increased in proportion as any impediment exists to the passage of the fluid, as by projections or irregularities, or by sudden bends, and by this increased friction sound is produced. Now, in the living human body it has been ascertained, by clinical investigation, that when murmurs have existed in the heart and the arteries, the causes of these murmurs have been found after death, in most cases, to be exactly such irregularities or projections presented against the flow of the blood, as would have caused friction and consequently *sound* in ordinary water-pipes. So far, again, the explanation is tolerably easy, for when a valve of the heart is thickened, or projections exist on its edges, or the inner coat of an artery is converted into a cartilaginous or bony crust, it may be expected that the blood in its passage over those structures will originate a morbid sound, or *murmur*, and it is found that it does so. But it also happens, as in many of the cases which puzzled Laennec, that murmurs exist during

[1] Op. citato, p. 314.

life, but are quite unexplained by post-mortem examination, there being, in such cases, no lesion or impediment discoverable in the heart or arteries. The cause, therefore, is to be sought not in the visible structure of the heart or of the vessels, but in the constitution of the blood itself, and it has been accordingly found that, by subjecting the lower animals to large and repeated bleedings, artificial bellows murmurs were produced, together with that peculiar thrill or vibration, called by Laennec the *frémissement cataire*. The same truth is proved by clinical observation, for it is found that persons who have accidentally lost large quantities of blood, or whose blood has been impoverished by constitutional causes, develope in their heart, and arteries, and veins, those very same morbid sounds, or murmurs, which in other cases denote organic disease of the heart or arteries. The sounds may accordingly be caused by a diminished quantity or an attenuated condition of the circulating fluid, the velocity of which is increased as its viscidity diminishes, and its vibrations increasing with its velocity; and the vessels themselves, moreover, being ill-supplied with their sources of nutrition, become weak and relaxed, and are therefore thrown into vibrations corresponding to those of the blood circulating within them. Dr, now Sir Dominic Corrigan, by whom and by the Irish school of physicians generally the phenomena of cardiac disease have been most ably elucidated, has very aptly summarised the facts developed in connexion with endocardial murmurs by drawing the following conclusions as to the causes of the sounds in question: '1. A current-like motion of the blood (instead of its natural equable movement) tending to produce corresponding vibrations in the sides of the cavities or arteries through which it is moving," and "2. A diminished tension of the parietes of the arteries or cavities themselves, in consequence of which their parietes are easily thrown into vibrations by the irregular current of the contained fluid; which vibrations cause, on the sense of touch, *frémissement*, and on the sense of hearing, *bruit de soufflet*." [1]

It is therefore clear that the bellows murmur, or the allied sawing or rasping murmur, heard over the region of the heart or arteries, indicates the existence of some morbid condition within the heart or the vessels, and is due either to diseased valves, or alteration of the coats of arteries; or, on the other hand, to the passage of diseased, or diminished, or attenuated blood over weakened cavities, valves, or

[1] 'Dublin Journal of Medicine,' vols. x and xiv.

vessels. But although these two conditions are very different and almost opposite in their character and consequently in their pathological signification, the diagnosis between them is by no means easy in all cases, and it is necessary in every given instance to study the collateral circumstances in the patient's history. It is a very common occurrence for persons to present themselves to the medical practitioner complaining of palpitation of the heart, difficulty of breathing, and perhaps cough, with rapid and, weak pulse, and, on examining the chest, a murmur is perceptible over the cardiac region; and yet very many of such patients have no organic disease of the heart whatever, and soon lose their unfavourable symptoms under the use of tonic and chalybeate remedies, and appropriate hygienic and dietetic treatment. These persons are most usually females, of delicate constitution and appearance, and until their cases have been fully considered, and the physical and rational symptoms have been duly examined, weighed, and compared, and the results of treatment observed, it is occasionally very difficult to form a correct opinion. The absence of any previous rheumatic attack, and the presence of chlorosis or of derangement of the uterine functions, will throw some light upon the diagnosis, which may still, however, remain rather obscure, and the more so because it is not impossible that functional and organic disease may co-exist in many instances. Again, even in men, in whom the existence of a functional murmur is far less common than in women, it must not be concluded at once that the presence of the morbid sound indicates necessarily endocardial or valvular disease, because any circumstance which tends to impoverish the blood, or to diminish its quantity, or even some accidental pressure upon a large artery, may give rise to the murmur, and might thus lead to erroneous deductions unless all the concomitant features of the case and the previous history were taken into account.

The morbid sounds or murmurs may accordingly be divided into two classes, namely, those caused by actual endocardial disease or by disease of arteries, and those caused by pressure on healthy vessels or by alterations in the quality of the blood. Among the former class may be placed contraction of the orifices of the heart caused by inflammatory exudations upon, or thickening, or hardening, or ossification of, the valves; growths, tumours, or vegetations, offering obstacles to the flow of the blood; abnormal adhesions of the valves to the walls of the ventricles or of the arteries, and laceration or perforation of the valves; or any other causes which interfere with

their perfect closure, and thus permit regurgitation into the ventricles. Among the latter class, or functional murmurs, are included those cases in which a murmur is caused by altered or depraved conditions of the blood, such as may be induced by accidental or constitutional losses of that fluid, by certain diathetic states of the system, as chlorosis or anæmia, or by wasting diseases which attenuate the blood.

MM. Barth and Roger, in their Treatise on Auscultation, lay it down as a law, that the cardiac murmurs, which are independent of organic disease, are always connected with the first sound, and never with the second, and this observation is very valuable as far as it goes; but it must be remembered that in many cases of organic disease of the heart, the cardiac murmurs are heard also only in connexion with the first sound. Hence, therefore, the presence of a diastolic murmur will be a pretty certain indication of the existence of organic disease, though the converse is by no means true, namely, that its absence indicates the affection to be due merely to morbid conditions of the blood. But the existence of a double murmur, accompanying or replacing the natural sounds of the heart, may be considered as almost an infallible sign of organic disease.

A careful examination of the concomitant circumstances in each case is of very great importance in distinguishing between the organic and the functional diseases of the heart. The former are frequently accompanied by other characteristic symptoms denoting, more or less clearly, an enlargement of the dimensions of the heart or an embarrassment of its action. These symptoms may be præcordial dulness, *frémissement cataire*, the co-existence of pneumonia, bronchitis, or pulmonary apoplexy, alterations in the characters of the pulse and of the breathing, the presence of œdema in the extremities, and other signs of serious disturbance of the circulation. But still it must be remembered that there are thousands of cases of diseased heart where any or all of these concomitant symptoms may be wanting, and the most trustworthy information is to be sought for in the auscultatory signs; and, on the other hand, many of the above-mentioned circumstances may be present when there is no organic disease at all. Difficulty of breathing and dropsy, for instance, may be present in chlorosis and anæmia and exhausting diseases, and in these conditions the action of the heart may be accelerated or excited, and its first sound accompanied or replaced by murmurs.

It is mentioned by Barth and Roger as another diagnostic sign between the organic and functional affections of the heart, that the cardiac murmur, which is a sign of organic disease, may be very well marked without the co-existence of the murmur along the course of the carotid arteries, while in alterations of the blood there is never a well-marked murmur over the heart without the co-existence of murmurs in the neck. These functional murmurs may be arterial or venous, and are often exceedingly loud: the former being intermittent, and synchronous with the cardiac systole, the latter being continuous, and easily made to cease by pressure on the jugular vein *above* the point where the murmur is heard.

This observation, however, is true only to a certain extent, for in organic disease of the heart, attended with degeneration of, or vegetations upon, the cardiac valves, it very often happens that there is also degeneration of the arterial system, which gives rise to a murmur over a great number of the arteries, the carotids in particular. I have repeatedly heard murmurs over the subclavian and carotid arteries in cases of valvular disease of the heart, and after death I have found fatty, or atheromatous or calcareous degeneration over the whole course of the aorta, and over many of the principal arterial trunks. As the friction of the blood against rough or rigid vessels will produce the same kind of sound as the friction of impoverished blood against relaxed and weakened, though organically healthy, vessels, it is quite evident that the information derived from auscultation must be received with due caution in forming a diagnosis. It is true that the well-practised eye and ear and touch will, perhaps, be able to distinguish, with considerable facility, between the sallow aspect, the loud humming and musical murmur, and the nervous irritability of the heart's motions in functional diseases on the one hand, and the congested countenance, the blowing or rasping murmur, and the increased impulse or the vibratory thrill in organic disease on the other. But still it is essential to insist upon the difficulties which often attend the diagnosis, and it is only by assiduous cultivation of the senses, and by frequent practice, that the distinctions can be clearly drawn, and even with the aid of the most skilful and practised eyes, ears, and hands, it cannot be said that all the sources of error are necessarily excluded.

The above remarks have a very important bearing upon the selection of lives for Assurance; for it is obvious that a degenerated or ossified condition of the valves of the heart or of the arteries,

is a far more important feature in reference to the eligibility of a given life than an attenuated or impoverished condition of the blood, which may disappear spontaneously or be removed by remedial treatment. Yet the stethoscopic symptoms of both conditions are essentially the same, and as persons proposing themselves for life assurance are seldom or never in a state of *visible* ill-health, it is very difficult to obtain much assistance from the collateral features and symptoms of the case. In many instances in the male subject, when no disease of the heart could be detected, I have met with a harsh, grating, or blowing murmur over the arch of the aorta, the innominata, or the carotid arteries. Now, in the female subject such a murmur might probably be attributable to anæmia or chlorosis, or loss of blood; but in the male cases just alluded to I have generally regarded it as indicating a degenerated condition of the large arteries proceeding from the heart. Still I have been by no means certain as to the exact significance of this murmur in all cases, more especially as I have frequently heard it in the case of young men, otherwise apparently in good health, and who continue in good health, and it is just probable that it may be due to accidental pressure on one of the arteries, or to comparatively unimportant and remediable conditions of the blood. The best plan to pursue in such cases is to defer the proposal for assurance for a few months or years, and, if the murmur should disappear, its nature will then be pretty obvious.

Drawing up the diagnostic distinctions between organic and functional murmurs of the heart in a tabular form, the result would be somewhat as follows:—

Organic Murmurs.	*Functional Murmurs.*
1. Although often single, are frequently double.	1. Are always single.
2. Are sometimes prolonged into the large arteries proceeding from the heart, but are never heard in the jugular veins.	2. Are almost always prolonged into the large arteries proceeding from the heart, and are often heard in the jugular veins.
3. Are often associated with hypertrophy of the heart, and consequently with increased force of the heart's action, and with a strong thrill felt by the hand.	3. Are often associated with undue excitement and irritability of the heart, but not with increased impulse or strong thrill.

4. Are often associated with œdema of the legs, cough, and secondary bronchitis, pneumonia, or pulmonary apoplexy.	4. Are often associated with œdema of the legs, and cough, but not with bronchitis, pneumonia, or pulmonary apoplexy.
5. Are often connected with a history of rheumatism.	5. Are unconnected with any history of rheumatism.

Pathological Signification of Organic Murmurs.—Having now pointed out the principal distinctions existing between murmurs arising from functional causes and those produced by organic disease, I next proceed to describe, as far as possible, the exact nature of the organic lesions which give rise to cardiac murmurs, and the special seat of disease which is indicated by those abnormal sounds, as heard on different external parts of the præcordial region, and at different periods of the heart's rhythmical action. For a murmur heard over the apex of the heart has a different signification from one heard at the base, and a murmur replacing or obscuring the first sound denotes a different condition from that indicated by one which is synchronous with, or follows the second sound.

In this department of practical medicine a remarkable degree of accuracy has been attained, and, thanks to the repeated physical examinations made and recorded during life, together with the careful observations of the appearances found after death, the seat of disease within the heart may be determined almost as exactly as if the parts were exposed to view. But while the seat of disease may thus be very accurately ascertained by stethoscopic examination, its extent or severity can unfortunately by no means be predicated with equal certainty, nor, as has previously been remarked and illustrated, can the loudness or distinctness of a murmur be regarded as a necessary exponent of its pathological importance. All we can assert with confidence is that the presence of amurmur heard at a given time or over a certain spot, indicates very accurately both the seat of endocardial disease, and the nature of the embarrassments which the blood meets with in its onward passage through the cavities of the heart.

It may be stated generally that organic cardiac murmurs arise from two distinct causes—namely, from obstruction offered to the passage of the blood, or from its regurgitation through apertures which it has already passed, and though which it ought not to return. The seat of these murmurs is now almost universally admitted to be in the cardiac valves, although it was once supposed

that the abnormal sounds might be caused by disease of the heart independent of valvular lesion. The valvular affections giving rise to the murmurs may therefore be classed under the two heads of *contraction* and *insufficiency*, the former condition offering a greater or less impediment to the flow of the blood, and the latter allowing the blood to flow back through the barriers which ought to have arrested its progress.

By a reference to the normal action of the heart and the synchronism of its natural sounds, the significance of the morbid sounds will be readily appreciated. The murmur may be heard coincidently with, or before the first sound, or with the second, or with both; it may be heard over the region of the aortic valves or over that of the mitral valves. These are the valves most commonly affected with disease, but it should be recollected that the pulmonary semilunar valves and the tricuspid valves may be affected also, and that the former lie on the same plane with the aortic valves, and the latter on the same plane with the mitral valves. The tricuspid valves, however, lie more on the right side than the mitral.

Systolic Murmurs.—A murmur heard with the first sound of the heart is synchronous with the contraction of the ventricles, and is consequently called a systolic murmur. At this moment the blood, in the healthy heart, is being propelled by the muscular walls of the ventricles through the orifices of the aorta and the pulmonary artery, the valves at the commencement of those vessels falling back to allow its free passage; and on the other hand, at the same moment, the blood is shut off from the cavities of the auricles by the closure of the mitral and tricuspid valves. In the normal state, therefore, the aortic and pulmonary semilunar valves are freely open at the same moment when the mitral and tricuspid valves are closely shut. But if the former set of valves are contracted when they ought to be open, or the latter are open when they ought to be completely closed, then the blood will be obstructed in its passage through the first set of valves, and will flow back, or *regurgitate*, through the second. Therefore, a murmur heard with the first sound of the heart will indicate either a contraction or other obstruction of the aortic or pulmonary semilunar valves, or an insufficient closure of the mitral or tricuspid valves.

Diastolic Murmurs.—But if the murmur accompanies or replaces the second sound of the heart, it is synchronous with the dilatation of the ventricles, and is called a diastolic murmur. During the

diastole, the blood is passing from the auricles into the ventricles through the mitral and tricuspid valves, which are of course patent, while at the same moment the aortic and pulmonary semilunar valves are suddenly closed to prevent the reflux of the blood which has been propelled by the ventricular contractions into the aorta and the pulmonary artery. Hence, if the auriculo-ventricular orifices are contracted or obstructed, the blood will be impeded in its progress, and if the valves of the aorta or the pulmonary artery are insufficiently closed, the blood will regurgitate into the ventricles. Practically it is found that in by far the great majority of cases a diastolic murmur indicates an insufficiency in the arterial valves, the aortic being almost always affected.

The reason why an obstructive murmur was heard comparatively seldom or with difficulty over the auriculo-ventricular orifices, is perhaps because the current of the blood does not pass through these openings with sufficient velocity and force, although in other respects the conditions of the murmur are the same as in the arterial valves. The existence of auriculo-ventricular obstructive murmur was accordingly almost ignored by the general body of practitioners, owing to the difficulty of detecting it; but Dr Peacock and many other writers have lately called attention to the fact that these murmurs, although supposed to be rare, are much more frequent than is generally supposed.

Præsystolic Murmur.—As the blood is passing from the auricles into the ventricles at the period of the dilatation of the heart, it ought to follow that in cases of obstruction of the auriculo-ventricular valves, the murmur should be heard either during the diastole or immediately before the succeeding systole of the ventricles, and such indeed is the case when the murmur is heard at all. But, as a matter of fact, this obstructive murmur was, until very recently, seldom heard, or at any rate recognised, although the conditions of its existence might be present. Dr Hope attributed the feebleness of the obstructive murmur to the weakness of the diastolic current out of the auricle, and he stated that this murmur was absent unless the contraction of the valve was considerable; but he also failed to discover it when the contraction was great, and he was compelled to rely, in most cases, on the regurgitant murmur as the evidence alike of obstruction and regurgitation. "On the whole," says Dr Hope, "this murmur is exceedingly rare, though Laennec and authors in general have supposed quite the contrary, from mistaking it for the

murmur of aortic regurgitation."[1] Dr Walshe while describing the obstructive murmur of the mitral valve, in the third edition of his 'Treatise on Diseases of the Heart,' admits that it is frequently absent, and that thus the positive diagnosis of mitral obstruction is far from easy. Even where constriction was discovered after death, Dr Walshe often found the murmur wanting, and he attributes the deficiency to the weakness of the auricular systole, and to the smoothness of the constricted orifice. In confirmation of the latter view, he adduces, in a note, the case of a patient in whom, during life, there had been no constrictive murmur, but in whose heart there was found a mass of ossiform substance, smooth on the auricular surface at the union of the two tongues of the mitral valve. Dr Walshe anticipated the present views on the subject, for he considers this murmur, when it is heard, to be rather post-diastolic or præsystolic, than as actually diastolic in rhythm.[2] The same author, writing in 1873, prefers the term præsystolic, but admits that the precise synchronism of the murmur is often very difficult of establishment.[3]

The difficulty of distinguishing the obstructive mitral murmur was generally admitted by most other writers and observers, but Dr Gairdner, of Glasgow, on the contrary, has long regarded such a murmur as being of rather common occurrence, and thinks that it is among the most easily detected of all cardiac murmurs. Dr Thomas Hayden described it in two Clinical Lectures delivered by him at the Mater Misericordiæ Hospital, Dublin, in 1866. Dr Peacock examined this subject afresh, in consequence of a careful perusal of Dr Gairdner's remarks, and in a communication made to the 'British and Foreign Medico-Chirurgical Review,' in 1867, he states that he has considerably modified the views he previously entertained, that he now believes the direct mitral murmurs to be less uncommon than he formerly supposed, and he conceives that they frequently furnish very valuable aid in the diagnosis of obstructive disease.[4] Still, however, Dr Peacock is far from agreeing with Dr Gairdner, either as to the frequency of these murmurs or the facility of their detection, and out of his very extensive field of observation he adduces, in the paper now referred to, only two cases where a præsystolic as well as a systolic murmur of mitral origin, was distinctly

[1] Hope on 'Diseases of the Heart,' p. 363.
[2] Walshe on 'Diseases of the Heart,' pp. 104 and 373.
[3] Idem. Fourth edition, p. 100.
[4] 'Brit. and Foreign Med.-Chir. Review,' October, 1867.

heard by him, and where the post-mortem examinations confirmed the diagnosis. Dr Peacock goes on to observe that when there is only one murmur heard, it is often very difficult to decide whether it precedes or accompanies the systole, but that the direction in which the murmur is propagated, will often afford considerable assistance in the diagnosis. Thus, he says, an obstructive murmur is heard at the apex of the heart, and extends thence towards the lower end of the sternum, while a regurgitant one extends from the apex towards the left axilla, and is usually heard in the left dorsal region. The distinction between obstructive and regurgitant mitral murmurs is not merely a point of minute diagnosis, but it explains some secondary pathological changes affecting the heart and lungs, and may perhaps hereafter lead to some important practical results in reference to prognosis and treatment. In mitral obstruction the consequences of the disease fall mainly upon the left auricle and the right side of the heart, while in mitral regurgitation the most marked changes occur in the left ventricle and auricle, those cavities becoming greatly increased in capacity. Dr Peacock also thinks that the prognosis is less serious in obstructive than in regurgitant mitral disease, because the former is more slowly induced, and the system has time to accommodate itself to the altered circumstances, while the latter is often rapidly and even suddenly brought about.

In the Fourth Volume of the Clinical Lectures and Reports of the London Hospital, 1867, Dr Sutton confirms the opinion of Dr Gairdner, that præsystolic bruits are by no means very rare, and he adduces a case attended by himself at the Victoria Park Hospital, in which the existence of the murmur in question was distinctly proved during life, and in which, after death, the walls of the mitral orifice were found indurated with calcareous matter, and the orifice itself admitted the tip of one finger with difficulty. In this case the præsystolic murmur was heard most distinctly, close under the left nipple, and it was separated from the second sound by an appreciable period of silence, so that it could not be regarded as a diastolic bruit, and, moreover, it preceded the pulse and also the impulse of the heart against the chest wall. Dr Sutton also refers to another case seen by him, although under the care of Dr (now Sir) William Gull, at Guy's Hospital, in which a præsystolic bruit was heard of the same nature as in his own patient, and he moreover states that twelve cases came under his notice during two years, in which there were præsystolic murmurs.[1]

[1] 'Clinical Lectures and Reports of the London Hospital,' vol. iv, 1867-8, p. 297.

Dr Flint, writing in 1870,[1] considers the præsystolic murmur, or, as he calls it, *the mitral direct or the mitral diastolic murmur*, less frequent than either of the other three murmurs, viz., the mitral regurgitant, the aortic direct, and the aortic regurgitant. But he confirms the fact of its occurrence, and he agrees with those who disbelieve in its rarity. In his own experience, out of 123 cases of mitral lesion, the mitral direct murmur was noted by him as present in forty-seven. He thinks the quality of the murmur is distinctive, and he describes it as being rough, and as resembling the sound produced by throwing the lips or tongue into vibration with the expired breath. As regards the *time* when the murmur occurs, he agrees with most other writers, and thinks that the term *præsystolic* is quite applicable. Dr Flint, however, considers that the mitral regurgitant or systolic murmur is that which is most frequently met with in organic disease of the heart. But he argues, from experience, that although a mitral regurgitant current almost invariably produces a murmur, the converse is not equally true, namely, that a mitral systolic murmur uniformly denotes insufficiency or regurgitation. He shows that a murmur at or near the mitral orifice may be due to roughness or calcareous deposit, without the valves being insufficient. This important statement appears to foreshadow the present impression in reference to the nature and the import of mitral murmurs and the rhythmic period at which they are heard.

The ideas now current among physicians who are studying the auscultatory history and nature of cardiac diseases are rather to be gathered from the discussions often held at our medical societies, from the living cases exhibited at these meetings, and from the scattered monographs in our medical journals, than from any books specially devoted to the subject. It would appear, from some of these sources of information, that so far from the præsystolic murmur being of rare occurrence, it is one of the most common of the auscultatory phenomena of the diseased heart, and that, so far from being difficult of diagnosis, its recognition is exceedingly easy. The fact just alluded to, as stated by Dr Flint, viz., that causes of obstruction, such as roughness or calcareous deposit on the mitral valve, may give rise to what was formerly considered as a systolic murmur, is quite established, but then the murmur is not systolic at all, but præsystolic. In fact, our views on this subject are now being changed, and the loud and distinct murmur so often heard over the mitral region is

[1] 'Treatise on Diseases of the Heart,' by Austin Flint, M.D. Second edit., p. 205.

perhaps often the præsystolic one, and denotes the presence of obstruction, while the systolic murmur, if it exist, is posterior in point of time. The true diagnostic character is to be drawn from the pulse, either at the wrist or at the carotids, which *succeeds* the præsystolic murmur, but is synchronous with the systolic.

Now, as in the case of the aortic valves an obstructive murmur may be heard alone, or be accompanied by a regurgitant murmur, so the same conditions may be found in the mitral valve, where a regurgitant murmur may follow the obstructive one, or either murmur may exist alone. But the view which has hitherto been held as to the nature of the mitral murmur appears to some to be no longer tenable, and instead of the regurgitant murmur being almost the only recognisable one, they believe the obstructive murmur to be the more frequent. The latter, then, is really the præsystolic murmur, the very existence of which was almost denied by Hope, and pronounced to be of very rare occurrence by Walshe, while the sound which succeeds the præsystolic murmur is, if of natural tone, the normal first sound of the heart, and, if rough or blowing, it is the systolic murmur of regurgitation. The combination of aortic obstruction with mitral regurgitation, hitherto considered as the most common of the morbid conditions of the heart, is no longer to be regarded as representing the true connexion of phenomena, but rather aortic obstruction with mitral obstruction. There may, of course, be mitral obstructive (præsystolic) *and* mitral regurgitant murmur, but when only one exists it is, in all probability, the præsystolic, but the relative frequency of the occurrence of these murmurs, or of their coexistence, must now be studied anew by the light of recent experience and observation.

The above remarks on the præsystolic murmur are chiefly historical and critical, and the subject will be again referred to in treating of the diseases of the mitral valve, but in the meantime I may state that I have myself investigated the subject as well as circumstances would permit, and that I have clearly detected the murmur in several instances. But while I fully admit its existence, I am not yet convinced either that it is so common as some writers allege it to be, or that it is very easy of discrimination. In the first case of the kind which fell under my notice—that of a little girl—since the publication of Dr Gairdner's, Dr Peacock's, and Dr Sutton's papers, I distinctly heard the præsystolic murmur, and I verified the observation not only by frequent examinations of the patient, but by appealing, for confirmation, to the evidence of professional friends

whom I knew to be well skilled in auscultatory diagnosis. At the present time I have two cases under frequent observation, and I have presented them, without any previous intimation as to the seat of disease, to advanced medical students, and their appreciation of the murmur in several instances has been a convincing proof of the correctness of my own diagnosis. But I have often heard, when cases have been exhibited at the medical societies, considerable doubt and hesitation expressed as to the existence or the period of the murmur, so that I am convinced of the difficulty of its detection at least by many persons.

Locality of the Organic Murmurs.—Thus it has been shown that the murmurs indicate different conditions, according to the *time* of the heart's rhythmic action at which they are heard; but it remains to be shown that the particular valves affected are indicated by the *place* where the murmurs are detected. The aortic and pulmonary semilunar valves are placed on the same level with one another, and lie of course towards the base of the heart, while the mitral and tricuspid valves also lie on the same level with one another, but considerably lower than the arterial valve. Now, although the level of the two sets of valves respectively is not so distant that we should conclude from *à priori* reasoning that their morbid indications ought to be discovered at a great interval from one another, yet it is found in practice that a murmur heard over or towards the base of the heart—namely, over the upper part of the sternum—indicates disease of the aortic or pulmonary semilunar valves, whereas a murmur heard over or towards the apex of the heart—namely, between the fifth and sixth ribs on the left side—indicates some morbid conditions of the mitral or tricuspid valve. This rule does not imply that the sounds are necessarily limited to some one definite spot, but merely that the sound which is most intense over the upper half of the heart indicates disease of the arterial valves, while that which is most intense over the lower half of the heart indicates disease of the auriculo-ventricular valves. It should also be further remarked that when the aortic valves are the seat of disease, the murmur is often prolonged into the carotid arteries, while in disease of the mitral and tricuspid valves the murmur is more confined in its limits.

Recapitulation.—Thus, supposing that the diagnosis of organic disease has been established, a murmur with the *first* sound of the heart, heard over the upper part of the sternum and prolonged into

the arteries, will indicate contraction of the arterial orifices, fibrinous concretion upon or degeneration of the semilunar pulmonary or aortic valves, or some other cause impeding, interrupting, or opposing the passage of the blood through the aorta or pulmonary artery. A murmur with the same sound heard over the apex of the heart will indicate insufficiency of the mitral or tricuspid valves, thus allowing regurgitation of the blood into the auricles. A murmur with the *second* sound, heard over the base of the heart, will indicate an insufficiency of the aortic or pulmonary semilunar valves, allowing regurgitation of the blood into the ventricles; and a murmur just before the systole heard over the apex of the heart would indicate contraction of the auriculo-ventricular valves, although in practice this sound very often escapes notice.

"In the majority of cases," says Dr Bellingham, "the *bruit de soufflet* is heard at the period of the ventricular systole, and replaces or accompanies the first sound of the heart; the force with which the blood is propelled by the left ventricle being much greater than that with which it enters it, the friction between the blood and the parts along or through which it passes must be much more considerable during the former action than the latter. In the former it is generally sufficient to generate a murmur, in the latter it only occasionally does so, and when a murmur is developed it has a different character. For instance, when the mitral valve or orifice is diseased, so as to permit regurgitation, a *bruit de soufflet* will be heard at the period of the ventricular systole, which quite obscures the normal first sound of the heart; when the aortic valves permit regurgitation, a *bruit de soufflet* is audible at the period of the ventricular diastole, which likewise obscures the normal second sound of the heart. But the two murmurs have a different character—that which accompanies mitral regurgitation is usually loud, strong, and blowing; that which accompanies aortic regurgitation is usually soft and whispering, because the force with which the blood is propelled from the ventricle is much greater than that with which it enters it."[1]

The murmurs have been regarded generally in the preceding pages as indicating the diseases or defects of the arterial orifices on the one hand, and of the auriculo-ventricular orifices on the other; and as the aortic and the pulmonary semilunar valves, which belong to the left and the right side of the heart respectively, lie on the same plane with one another, and as the mitral and tricuspid valves, which be-

[1] 'A Treatise on Disease of the Heart,' by Dr O'B. Bellingham, p. 143.

long also to the left and right side of the heart respectively, also lie on the same plane with one another, it is obvious that there might be very great difficulty in determining, by auscultatory evidence, which side of the heart is affected in any given case. The difficulty, instead of being hypothetical, would be a real one, if disease of the right side of the heart were as common as it is on the left, but the fact is that disease of the right side of the heart is comparatively rare. Amidst a number of post-mortem examinations the right side of the organ is seldom found to exhibit any traces of disease, the proportion of such cases being estimated as about one to twenty; nor do the symptoms observed during life point very often to disease affecting the right side, either exclusively or in conjunction with disease of the left side. In the brief description, given in another page, of the anatomy and physiology of the heart, it was observed that the chief organ of the circulation is really double, one part belonging to the lungs and the other to the system in general; and these two hearts, although united closely together and acting in harmonious rhythm, are shut off from one another as soon as the fœtus has emerged from the womb, and has drawn atmospheric air into its lungs. Ever after this period, the right and the left hearts are distinct though mutually dependent organs, and although closely bound together by the same muscular structure, their functions are of a dissimilar character, and the force required for their respective performance is disproportionate. The left ventricle of the heart being destined to propel the blood to the whole system, is provided with much more robust muscular walls than the right ventricle, which sends the blood only to the lungs, and, when disease is set up in the heart, it usually attacks the side which has the most work to perform.

Still it is rather difficult to account for the general immunity of the right side of the heart from the morbid alterations which so often affect the left, and after every explanation that may be offered, it is necessary to accept the ultimate fact that such a difference exists, and hence in cases attended with obscurity in diagnosis, the knowledge that the left side is most usually affected, will afford considerable assistance to the auscultator. Nevertheless, the right side of the heart is often affected, especially in fœtal life, and although the diagnosis by auscultation is sometimes difficult, yet there are collateral circumstances, especially those connected with the pulse and the venous circulation, which tend to assist the diagnosis, and throw light upon the real nature of the case.

But, leaving the collateral symptoms and the question of the pathology of the right side of the heart for future consideration, it may be observed that certain special auscultatory signs accompany the diseases of the right auricle and ventricle, and as the semilunar pulmonary valves lie a little to the right of the aortic valves, and the tricuspid valves lie a little to the right of the mitral valves, the diagnosis will mainly depend upon the exact situation at which the morbid sound is heard. When, therefore, the tricuspid valves are affected, a murmur will be heard above the epigastric region; while in case of mitral disease the murmur is heard over or towards the apex of the heart, and extends backwards to the lower angle of the scapula. If a murmur should be heard at both these points it may be assumed that both the auriculo-ventricular valves are diseased. It is right to mention that tricuspid regurgitant murmurs sometimes occur, and that their seat, when they are heard, is immediately over the ensiform cartilage of the sternum.

Again, when the aortic valves are diseased, the murmurs are heard over the base of the heart, and if the pulmonary valves are diseased, the murmur will be heard over the same plane, but a little to the right side. Dr Hope never saw the latter diseased, though he once found them incapable of closing, owing to dilatation of the pulmonary artery;[1] and indeed pulmonary valvular disease, except when originating in fœtal life, is very rare.

It must be repeated, therefore, that the left side of the heart is far more frequently diseased than the right. The most usual murmur is that which is heard synchronously with the contraction of the ventricles, and if it is heard simultaneously over the base and apex of the heart, it indicates both obstructive disease of the aortic valves and insufficiency of the mitral valves, and this is a very common, and indeed it has hitherto been considered the most common complication. When a double murmur is heard over the base of the heart, the sounds indicate obstruction of the passage of the blood to the aorta, together with regurgitation through the aortic valves, and when there is a double murmur over the apex of the heart, the præsystolic murmur (which, however, is not always audible) indicates obstruction of the mitral valve, while the systolic sound indicates regurgitation.

[1] Hope on 'Diseases of the Heart,' p. 343.

CHAPTER VII

THE GENERAL OR RATIONAL SYMPTOMS OF DISEASE OF THE HEART

THE physical signs of disease of the heart, or those obtained by the aid of percussion and auscultation, having now been considered, it becomes necessary to describe, though much more briefly, the symptoms of a more general character which may be observed without the aid of the plexımeter and the stethoscope. These rational signs, as they are technically called, are sometimes sufficiently striking, but in very many cases they are partially, or even wholly, wanting; and, whether present or not, the diagnosis of heart disease can be made without their aid. But their existence affords valuable assistance to the investigation, although they are in themselves deficient in precision and of comparatively inferior value and importance. At a time when the practice of auscultation was unknown, it cannot be a matter of wonder that the whole subject of heart-disease was involved in doubt and uncertainty. The state of the pulse and the breathing, the feelings of the patient, the presence of local pain, and other similar symptoms, though useful as collateral evidence, are, taken by themselves, unworthy of reliance, and become important only when taken in connexion with the physical signs. Even the pulse, to which, however, special reference will presently be made, is often a fallacious guide as to the nature or existence of disease of the heart, although its beats of course derive their character from the force imparted by the chief organ of the circulation; and even the presence of palpitation of the heart affords no evidence of the presence of organic disease, especially when it is felt and complained of by the patient himself.

It is necessary, however, to remark that while the heart may be free from any organic disease, or, in other words, from any altera-

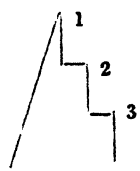
tion, either acute or chronic, of its structure, it may be painfully affected by functional disorder, which, although not of a nature to threaten life, may very seriously impair the comfort of the patient. Here, however, the stethoscope affords important evidence, although of a negative character, and the confidence with which the statement may be made, in many such cases, of the absence of appreciable lesion of the heart's structure, will often materially contribute to the alleviation or cure of the disease. I should also remark here, although I am anticipating what will be the subject of observation hereafter, that even with the revelations made by the stethoscope, the relief of heart disease is not so difficult, nor its prognosis nearly so gloomy, as was once supposed.

The Pulse.

Speaking of heart-disease in general, the indications given by the pulse, taken by themselves, are anomalous and unsatisfactory, for a full and rapid pulse may co-exist with a weak and irritable heart, while a feeble pulse may accompany an hypertrophied and violently acting organ. However, putting out of the question those cases (and they are very numerous) where the pulse in disease of the heart does not deviate from the healthy or usual standard, it may be said that it is far more commonly increased than diminished in rapidity in such disease; a slow pulse in disease of the heart, even in great obstruction of the cardiac orifices, having seldom been observed, although in softening of the organ this kind of pulse has been noted.

Within the last few years an attempt has been made, and with very great success, to measure the force of the pulse, and to render its strokes visible to the eye, by means of the instrument called the *sphygmograph*, invented by Marey, and which is so constructed that by being placed upon the arteries, and brought into connexion, by means of a hand or indicator, with tracing paper, the force of the beats is exhibited in visible lines upon the paper. As the artery rises under the impulse of the blood passing through it, the indicator marks a vertical line of greater or less height upon the paper, and as the artery sinks preparatory to the next pulsation, the indicator marks a corresponding downward line. This explanation, however, does not embrace the whole of the phenomena of a beat of the pulse, which is found to be divisible into three periods in the normal state,

being, as it is called, *tricrotic* (τρις thrice, κροτος a stroke). The following will roughly represent the tracing of the sphygmograph in a natural pulse:



Here it will be observed there are three waves—one, the highest or primary wave, and two secondary ones; the first being caused by the distension of the artery from the contraction of the left ventricle. But as soon as this stroke is recorded by a sudden and upward jerk of the lever (1) the line begins to descend, when it is interrupted by a notch (2), and a second notch (3), the whole forming a series of three waves or strokes. It would appear that the notch (3) is caused by the reflux of blood towards the heart, and the obstruction naturally offered to its passage by the sudden closing of the aortic valves. But when these valves are open in consequence of disease, the blood no longer experiences the same obstruction as it does in the natural state, and the pulse-wave therefore represents the following lines: in which, it will be observed, there are only two waves, or in other words, the pulse is *dicrotic* (δις twice, and κροτος a stroke), and dicrotism thus becomes an indication of aortic regurgitation. Although the remark has no bearing upon the diagnosis of heart disease, I may observe parenthetically that in the highest paroxysm of fever, the pulse becomes *monocrotic*, or exhibits only a single wave or stroke.



Hence it would appear that the sphygmographic tracings afford a very valuable diagnostic sign of aortic regurgitation, and there is no doubt that, in a scientific point of view, the indication is a most valuable and interesting one; but it unfortunately happens that other conditions of the system, quite independent of aortic disease, afford the same tracings on the sphygmograph, and moreover there are indications of the ordinary kind, presently to be noticed, and which may be observed on the pulse by the usual means, quite as trustworthy as those of Marey's ingenious instrument. Mere functional disorders will often afford the same sphygmographic evidence as that given by regurgitant disease of the aortic valves, and as Dr Sanderson himself, who has greatly distinguished himself by his researches on the application of the sphygmograph, observes, "all such cases (*i. e.*, of heart-disease) can be just as satisfactorily made out by the ordinary methods of examination." . . "With the one excep-

tion of aneurisms, the sphygmograph is not an instrument of diagnosis; for affections the most diverse communicate to the pulse the same graphical characters. Its use is to enable the physician to investigate the state of the circulation and circulatory organs in diseases of which the general nature is already recognised, &c."[1]

But it is right to mention that the late Dr F. E. Anstie, who also investigated with great care and success the indications afforded by the sphygmograph, entertained a somewhat higher opinion of the value of the instrument as a means of diagnosis. "The diseases," he says, "in which it is now evident that the sphygmograph will prove of the greatest diagnostic value are these: In aortic regurgitation, by estimating the amount of valvular imperfection; in discovering unsuspected commencing cardiac hypertrophy, senile diseases of arteries, or capillary disease dependent on degenerative processes in the intimate tissues; and, above all, in discovering the existence of intrathoracic aneurisms, and in deciding the locality of an aneurism. A striking example of the correctness of the instrument's indications in this respect is recorded in the 'Lancet,' where I was able confidently to say, from a comparison of the two radial pulses, that an aneurism bulging the anterior triangle of the neck was not subclavian or axillary, but arose from the aorta or innominata. The post-mortem revealed the truth of this diagnosis."[2]

The normal rate of the pulse being from seventy to eighty in the minute, any rate which either falls below or exceeds that average may be considered as unusual, but not always necessarily indicative of disease. Speaking generally (for precision in this instance is impossible), it may be considered that a pulse which exceeds ninety implies the existence of some functional or organic disorder, while one under sixty may be compatible with perfect health. Undue slowness of the pulse, taken by itself, may be dependent only on constitutional peculiarity, but undue rapidity is a more or less serious indication. Cases of unusually rapid action of the pulse have been lately recorded by Sir Thomas Watson, Dr Cotton, and several others, in the medical journals, and such cases have been sometimes found associated with dangerous disease of the heart; and one curious instance is recorded by Dr Broadbent, where the pulsations of the heart were 168 in the

[1] "Lecture on Characters of the Arterial Pulse," by J. Burdon Sanderson, M.D., F.R.S. 'British Medical Journal,' July 13, and July 27, 1867.

[2] 'Biennial Retrospect of Medicine, Surgery, and other Allied Sciences for 1865-6.' Printed for the New Sydenham Society, p. 171.

minute, and regular, though very strong, while the wrist-pulse was feeble, irregular, and intermittent.[1]

When, therefore, the pulse is very rapid, and there exist at the same time symptoms of derangement of the heart's action, the case must be considered as one of a suspicious nature, even although the stethoscope may afford no diagnostic information. But an habitually undue rapidity of the pulse, by which I mean a pulse of from 90 to 120 in a minute, when it is the only rational symptom and is unaccompanied by any auscultatory evidence of cardiac disease, is a symptom with the pathological significance of which I am not yet fully acquainted, and of the gravity of which I am not yet entirely convinced.

Among the many hundred persons—the great majority, of course, being quite healthy—whom I have examined for Life Assurance, I have on a few occasions found the pulse preternaturally accelerated, without any evidence whatever, either by auscultation or otherwise, of cardiac disease. I need scarcely observe that in all such cases allowance was made for nervousness, for hurried walking or running, for eating or drinking, or other obvious causes for the acceleration. I did not conclude, in the cases to which I refer, that the rate of 112 or 120 in the minute (for such were the numbers of the pulse) was the normal one until I had felt it under a variety of circumstances and at different times. Guided by the general opinion of the profession, and by the recorded doctrines of the most distinguished writers on Practical Medicine, I have hitherto regarded such an acceleration of the pulse as a morbid condition, and one which was unfavourable to the prospects of longevity, and therefore an obstacle in reference to a safe life assurance. But I am not quite certain that the symptom, taken by itself, is one of such grave importance as is generally supposed, and I am inclined to believe that in some cases it may be due only to some want of the inhibitive power of the vaso-motor nerves over the coats of the arteries, and to be really unconnected with any disease of the heart, or any serious disorder even of the nervous system.

In one of the cases to which I allude, the subject was a gentleman with whom I was acquainted, and whose health was quite unexceptionable, and yet on careful examination of the pulse at various times, and under all necessary conditions of perfect repose of mind and body, I found its rate to be 120. This gentleman I have

[1] 'British Medical Journal,' Aug. 3, 1867, p. 86.

known for more than twenty-five years, and he always has been, and is now to the best of my belief, in excellent health. I must therefore express my conviction that rapidity of the pulse, even to 120 in the minute, *may* be compatible with health, although the instances in support of this suggestion are certainly rare, and in forming an opinion on a given case great care must be taken to avoid all sources of fallacy.

Intermission of the pulse must, I think, be altogether disregarded as a pathognomonic sign of cardiac disease, although it is a symptom which often painfully excites the apprehensions of the patient. Intermission of the heart's action may and undoubtedly does exist in many diseases of that organ, but it far more frequently results from mere nervousness, or hypochondriasis, or indigestion. In some cases it cannot be explained even by reference to these very common though comparatively unimportant affections. Dr B. W. Richardson, in the course of some interesting lectures which he published, on "Intermittent Pulse and Palpitation," mentions the case of an aged man, whose heart he examined after death, and who for many years had presented the phenomenon of intermittency in a most remarkable manner, the pulse never failing to intermit less often than once in eight beats. His death was from senile decay, and his heart was found perfectly healthy; there was no trace of valvular disease, no departure from the natural size and condition of the heart's cavities, or the thickness of their walls, the coronary arteries were normal, and the muscular structure was quite free from fatty and granular degeneration.[1] My own opinion, and that of the profession in general, is, I think, in accordance with that advanced by Dr B. W. Richardson, in the lectures referred to—namely, that there is no known morbid condition of the heart itself, structurally considered, that produces the phenomenon of intermittent action, which must rather be referred to some remote and perhaps independent cause, seated, in all probability, in the nervous system.

Nevertheless, although the pulse, even in heart disease, may justly be denominated, as Celsus called it, *fallacissima res*, yet there are certain modifications of its force, its frequency, and its rhythm, which are often observed in cardiac disease and ought by no means to be overlooked, especially when taken in connexion with the physical signs.

[1] "Lectures on Experimental and Practical Medicine," by B. W. Richardson, M.D., F.R.S. 'Medical Times and Gazette,' Jan. 4, 1868.

Thus, in the onset of *pericarditis*, the pulse is always frequent, and generally full and hard, although regular, and the characters of fulness and frequency are observed in *endocarditis;* while in *hydro-pericardium*, where the heart is embarrassed by circumjacent fluid effusion, the pulse is small, frequent, and irregular.

In the early condition of *hypertrophy* of the heart, where the muscular structure is augmented in volume, and when the force which impels the blood is thereby increased, the pulse is strong and full, although its velocity may not be greater than in the normal state. As the hypertrophy increases and is accompanied, as it usually is, with dilatation of the cavities, the strength and fulness of the pulse continue, but when the enlargement of the whole organ has passed beyond a certain point, the contractile power of the muscular fibres is diminished, although their bulk is very great, and the blood is no longer propelled with the same force through the system; and then arises the apparently anomalous and paradoxical condition of a heart beating with enormous force against the parietes of the chest, and a small and weak pulse felt in the radial arteries. This disproportion between the action of the heart and the force of the pulse may be observed even where there is no accompanying valvular disease, but if this exists, and an obstruction is thus offered to the passage of the blood, the weakness of the pulse will receive an additional explanation.

In *dilatation* of the heart, where the muscular walls are attenuated and the capacity of the cavity is increased, the power of the heart is necessarily diminished, and the pulse is soft and weak. Practically speaking, simple hypertrophy and simple dilatation of the heart are each respectively exceedingly rare, the most usual condition which is met with being the combination of hypertrophy with dilatation, and the pulse is full and strong as long as the hypertrophy is predominant; but in proportion as dilatation advances, and the muscular force is thereby diminished, the pulse becomes soft and compressible.

In *obstructive disease of the aortic valves,* unless the contraction is extreme, the pulse is full, strong, and regular; for although the stream of blood thrown into the aorta is somewhat diminished in its volume, it is propelled with greater force, owing to the increased force of the ventricular walls, and thus the pulse is increased, rather than otherwise, in its strength and fulness. To Dr Hope is due the credit of the observation that the pulse, in contraction of the aortic valves, is regular, for Corvisart, Louis, and Bouillaud all

described the pulse in aortic contraction as irregular; but it must be observed that these authors were formerly but imperfectly acquainted with the diagnosis of valvular disease, on which so much light has been thrown in recent years by British writers on cardiac maladies.

In *aortic regurgitation* the pulse is so peculiar that its characters are sometimes almost pathognomonic of this affection, for it is jerking, and receding, but regular, and the pulsation is visible and locomotive. This visible pulsation, which has been observed not only in the radial but also in the temporal and other arteries and their branches, was first pointed out as a symptom of patency of the aortic valves by Sir Dominic Corrigan. The arteries in such cases appear tortuous in their character, like small worms beneath the skin, and their movement is seen to be synchronous with the contractions of the left ventricle. But this visible pulsation must by no means be regarded as necessarily indicative of regurgitant disease of the aortic valves, or of any other serious affection of the heart, for the rigidity of the arterial tubes in old age will give rise to this phenomenon when the heart and even the arterial system appear to be healthy. The best practical rule to be observed is probably this, that visible pulsation of the radial artery at the wrist, or of the temporal artery *in a person not advanced in years,* is a valuable symptom of aortic disease. Thus, I have now two patients, one aged forty-five and the other forty-six, in whom the pulsations at the wrist can readily be seen, as well as in the right carotid and facial arteries, and in both of these cases there is well-marked disease of the aortic valves; and I do not recollect to have seen cases at that age exhibiting the same phenomenon where the heart was healthy. I am not quite sure that visible pulsation of the arteries in aortic disease necessarily depends on aortic *regurgitation;* at all events, I have sometimes failed to recognise more than the murmur of obstruction.

But the jerking and receding pulse, or, as it has been called, the *pulse of unfilled arteries,* is very characteristic of aortic regurgitation, although the same kind of pulse, and from somewhat analogous causes, may be felt in some anæmic conditions of the system. "The diastole (dilatation) or beat of the artery is short and quick," writes Dr Hope, "as if the blood were smartly jerked or shot under the finger, the vessel during the intervals feeling unusually empty. This is the most remarkable, appreciable, and constant pulse produced by disease of the heart.

In the immense majority of cases, the practitioner may conjecture the disease by this sign alone. It differs from the jerking pulse of anæmia in being more marked, and in not necessarily being frequent, as the anæmic pulse is, when its jerk is distinct."[1]

The jerking pulse of unfilled arteries derives its peculiarity from the fact that the blood imperfectly distends the arterial tubes, and this imperfection may depend either upon scanty quantity or diminished viscidity of blood on the one hand, as in anæmia (spanæmia), or, on the other hand, on a partial emptying of the arteries after the blood has been propelled into them by the heart. Now, the jerking character of the pulse in aortic regurgitation is readily explained by reference to the condition of the aortic valves, which in this affection are open, when they ought to be closed. The contraction of the left ventricle, in the healthy subject, having propelled the blood into the arteries, these tubes dilate from the column of blood which is injected into them, and their elastic coats pulsate from the resistance they offer to the distending fluid, which is prevented from flowing back by the closure of the aortic valves, and from flowing forwards by the capillaries, which, by their minuteness and the slowness of the circulation within them, retard the velocity of the blood as sent from the ventricle. But when the aortic valves are insufficiently closed, then the blood, instead of being prevented from flowing backwards, is assisted in doing so, and, in fact, a part of it does re-enter, or regurgitate into the left ventricle, and the beat of the arteries is consequently incomplete, because the arterial column of blood is no longer pressed forwards by the column behind it, or at least is incompletely pressed forwards. The arteries are therefore incompletely filled, and hence the column of blood appears to the finger held over the pulse to be interrupted, and to exhibit a jerking and receding character. The same effect is produced, as I have just observed, by diminution in quantity or impoverished quality of the blood itself, independent of disease of the valves, but a consideration of the diagnostic marks, as pointed out by Dr Hope, and of the history of the case, will sufficiently determine the real nature of the affection.

It will be recollected that in this peculiar pulse the sphygmograph exhibits very characteristic tracings, and if other and better signs of aortic regurgitation did not exist, the aid of this instrument would be very valuable; as it is, the sphygmograph affords corro-

[1] Hope on 'Diseases of the Heart.' Fourth edition, p. 355.

borative evidence, but nothing more, and the signs it gives in aortic regurgitation are exactly the same as those developed by other affections, even when the heart is not suffering from any organic lesion.

In *regurgitant disease of the mitral valves*, in which case the blood is thrown back into the left auricle, the pulse is but little affected at first, but as the disease advances it becomes invariably weaker than in the natural state, and it is also usually described as being irregular, intermitting, or unequal. But in many cases of mitral disease which I have watched for considerable periods, the pulse has never been irregular or intermittent, and indeed these characters are not now considered as essentially connected with this or indeed any other form of cardiac lesion, at least until the disease is very far advanced, and has become associated with some secondary affections, as of the digestive, the respiratory, or the nervious system.

In *obstructive disease of the mitral valves*, which very frequently exists, although the auscultatory indications are at present sometimes obscure, the pulse is also weak, and indeed both obstructive and regurgitant disease of the mitral orifice are attended by the same pathological conditions, for in the former the cavity of the left ventricle is imperfectly filled, and in the latter a portion of the blood which has entered it passes back into the auricle, and thus in either case the ventricular walls are insufficiently stimulated to contract upon their contents, and the pulse is necessarily reduced in force.

In that hitherto mysterious disease, *fatty degeneration of the heart*, it cannot be said that the nature of the pulse throws, in general, much direct light upon the diagnosis; but in the advanced stages of this affection, in consequence of the softening of the muscular walls, the force of the circulation is diminished and the pulse becomes small, weak, irregular, and unequal. But all these characters may exist in cases of merely weak heart, or exhausted condition of the system from numerous causes, and they cannot be received as pathognomonic signs. Sometimes in the fatty degeneration the pulse has been found much more slow than in the natural state, and Dr Stokes and Dr Quain have recorded cases of this affection where the pulse was only thirty-two, twenty-eight, or twenty-four. It would be erroneous, however, to make a diagnosis of fatty degeneration of the heart from slowness of the pulse, even when accompanied by feebleness and irregularity; and indeed there must be many concomitant circumstances during life to render the diagnosis of this disease certain or even probable.

To sum up the characters of the pulse of cardiac disease in a few words, it may be stated generally that in hypertrophy the pulse is strong; in dilatation it is weak; in aortic obstruction it is strong, full, and regular; in aortic regurgitation it is jerking, visible, and locomotive; in mitral obstruction or regurgitation it is weak and sometimes intermittent; in fatty degeneration it may be small, weak, irregular, unequal and sometimes slow. The most important pathognomonic indication of the pulse is afforded in regurgitant disease of the aortic valves. It cannot be too carefully impressed upon the mind that cardiac diseases usually present themselves in a combined form, and that the characters of the pulse will vary as one condition or the other predominates.

Palpitation

Palpitation is a very common, though by no means necessary symptom of disease of the heart, and it may indicate, according to circumstances, either structural disease or mere functional derangement. In the latter case it is usually a very painful symptom, and causes great distress to the patient: in the former it is a very valuable aid to diagnosis, but is not necessarily or even generally felt by the patient at all. It has already been mentioned, that in a state of health, the pulsations of the heart are not felt by the individual, and when they are perceptible, it is owing to the fact that there is some deviation, although perhaps very slight, from perfect health. But palpitation, or undue action of the heart, is itself a disease, or it may be a symptom of disease, in which category it is described in this place.

When palpitation is not merely functional but is caused by disease of the heart, it is usually due to the presence of acute inflammation or of hypertrophy, and in either case it comes on gradually. In cases of pericarditis and endocarditis, it lasts as long as the inflammation is in the acute stage; in hypertrophy it may last for an indefinite time, and indeed in such cases it is scarcely ever absent, especially when, as is most usually the case, the hypertrophy is combined with dilatation, and the whole bulk of the organ is increased. The palpitation is then readily felt by the hand placed over the chest, and the space over which it is felt is greater than in the healthy condition of the heart; the impulse is strong and heaving, and so powerful as to elevate the hand, or the stethoscope, or the head of the observer. But it by no means

follows as a rule that the patient himself is aware of this palpitation; and although, under such circumstances, palpitation may be complained of, it is far more likely to be a troublesome and painful symptom in cases where there is no actual disease of the heart at all, but merely an exalted sensitiveness of the cardiac nerves. In valvular disease uncomplicated with hypertrophy, and in simple dilatation of the heart (which is rare), palpitation may exist, although it is not of much value as a diagnostic sign, and it has not the same heaving character as in hypertrophy with dilatation. When palpitation is accompanied, as it sometimes is, with the purring tremor, or *frémissement cataire*, an hypertrophied condition of the heart is indicated, together with regurgitation through some of the cardiac valves.

Palpitation, taken by itself, however, is no more to be relied upon as a symptom of disease of the heart than is the pulse, nor is it always perfectly easy in any given case to determine whether the palpitation is due to organic disease or to mere functional derangement. It may be stated generally that palpitation from the latter cause is less frequent in males than in females, but that in males it is usually more painful and difficult to treat; that palpitation from organic disease comes on slowly while in nervous palpitation the attacks are generally sudden; that palpitation from organic disease is constant, while in cases independent of organic disease the palpitation may intermit or remit, or cease altogether; that in organic disease the impulse is strong, heaving, and prolonged, while in functional disease it is not heaving or prolonged, and has rather a slapping or tapping character; and that in organic disease, namely, hypertrophy with dilatation, the præcordial dulness is increased, while such is not the case with nervous palpitation. The collateral circumstances must of course be taken into account in making a diagnosis, and the stethoscopic indications will here prove of inestimable value; and the presence of lividity of the countenance will corroborate the evidences of heart disease, while this symptom will be absent in mere functional disorder. Again, while exercise, as riding, walking, or running, will aggravate the palpitations in organic disease, those caused by nervous affections will rather be increased by want of exercise; and stimulants will often relieve palpitation from nervous causes, while the same agents would be inappropriate and perhaps injurious in palpitations of organic origin.

Other Rational Symptoms of Cardiac Disease

The other rational symptoms of disease of the heart will be explained more appropriately in connexion with the special diseases themselves; but it may be stated generally, that as any obstruction to the onward progress of the blood will cause congestion or stoppage in the parts behind the obstruction, so any impediment to the circulation in the heart itself will cause inflammation or congestion, or hæmoptysis or dropsy, in organs and structures related to the heart either by proximity of situation or by physiological connexions. Thus when the blood is prevented from passing through the mitral valves, or rushes back when it ought to have passed those valves, it will be thrown upon the lungs, and will cause inflammation or gangrene, or extravasation of blood in those organs, or hæmoptysis. The same obstruction will also act upon more remote structures, and by blocking up one part of the circulation will cause congestion of the capillaries in the skin, the liver, the kidneys, or the intestines; and the congestion of the veins will retard absorption, and thus give rise to dropsy of the extremities or of the abdomen. Again, when the blood is prevented from flowing freely through the aortic valves, or when it falls back into the left ventricle, the aorta carries an insufficient supply of blood for the nourishment of the different organs and tissues, and emaciation consequently ensues.

As disease of the heart has been often found associated with congestion of the brain or effusion of blood into the latter organ, an opinion has been entertained that there is some necessary connection, in the sense of cause and effect, between the diseases of these two important organs; and Dr (now Sir George) Burrows in particular, has maintained, with considerable show of reason, that congestion of the brain is often the result of hypertrophy of the left ventricle of the heart.[1] This distinguished physician has brought forward a great number of facts, from actual experience and from experiments on some of the lower animals, to prove the truth of the facts themselves and of the theory which he proposes to account for them. He has satisfactorily shown, by his experiments, that the cranial cavity is capable of containing, at certain times and under certain circumstances, more blood than it contains at other times and

[1] 'On Disorders of the Cerebral Circulation, &c.,' by George Burrows, M.D., 1846.

under other circumstances, and he attributes this capability of receiving a varying amount of blood on the part of the brain to the existence of the cerebro-spinal fluid, which, by its alternate presence in the ventricles of the brain and its subsidence into the spinal canal, permits a greater or less quantity of blood to circulate through the brain. But Sir George Burrows has not, I think, so satisfactorily proved that congestion or extravasation of blood in the brain is caused by hypertrophy of the left ventricle, although such a proposition carries with it a *primâ facie* evidence of probability. There is no doubt that in a large Hospital or among any large number of cases selected for clinical observation, there are numerous instances where congestive or apoplectic disease of the brain is combined with hypertrophy of the left ventricle of the heart, but it is by no means clear that these conditions are associated together as cause and effect. For it must be recollected that pure uncomplicated hypertrophy is comparatively rare, and even when it does exist, the mechanism by which the blood enters into and passes out of the cranial cavity, together with the oscillation of the cerebro-spinal fluid just alluded to, prevents any undue or dangerous pressure upon the brain, even if the blood should be propelled into it with more than the ordinary force. But, in fact, the hypertrophy of the left ventricle is usually associated with, and even caused by, disease of the valves, whereby the blood is prevented from passing through the aorta with undue force, and thus the entrance of blood into the brain is rather retarded than promoted.

I must state, as far as my own experience goes, that I have not observed any necessary or even constant connexion between enlargement of the heart and apoplectic or congestive diseases of the brain. I have noticed and kept under view a great number of cases of hypertrophy of the heart without any brain complication whatever, and *vice versâ* I have attended during life, and examined after death, a great number of cases of apoplexy in which there was no disease of the heart. I do not by any means deny that such a combination is often observed, but I regard it as a coincidence rather than in any other light. It is far more probable, and indeed it is almost certain, that extravasation of blood into the substance of the brain occurs only when the coats of the cerebral vessels are in such a state of disease that they are liable to rupture, and that so long as these vessels are healthy, no rupture takes place, even although the left ventricle of the heart may be hypertrophied. On the other hand,

where there is no disease of the heart at all, but the cerebral vessels are in a brittle condition, rupture of the coats may take place from comparatively slight causes.

But although it is by no means clear that apoplexy or extravasation of blood in the brain is due to the existence of cardiac hypertrophy, it is extremely probable that an opposite condition of the heart, namely, a weakened condition of the left ventricle, may so diminish the supply of blood to the brain that softening or some other depraved condition of the latter organ may ensue. Dr Law, an Irish physician, who was the first to call attention to the injurious effects of a deficient supply of blood to the brain, has recorded cases showing that cerebral softening may occur in connexion with disease of the heart, when the effect of such disease is either directly or indirectly to diminish the supply of blood to the brain, and that this cerebral lesion may be connected with disease of either the mitral or aortic orifices, but more usually with obstructive or regurgitant disease of the mitral. The following case, which fell under my own notice, illustrates very clearly the connection between *ramollissement* of the brain and contraction of and regurgitation through the mitral valve.

Case. Softening of the Brain—Pneumonia—Disease of the Mitral Valve. I was sent for to attend Sarah T., æt. 52, a tall, pale, sallow-looking woman. When I arrived, I was informed that she had had a fit, and had been insensible about a quarter of an hour. When I saw her, however, she was in full possession of her mental faculties; but she spoke very indistinctly, in consequence of an accident she had met with several years before, and by which she lost, by exfoliation, large portions of the left upper jaw-bone and of the nasal bones. Her mouth, in consequence of the same accident, was habitually distorted. She had not been inclined to sleep previously to the attack, but, on the contrary, had suffered from the want of sleep. *She complained of total inability to move the left arm,* and when it was lifted by another person, as soon as the hold was relaxed, it fell down like an inanimate mass; but when the skin of the hand or arm of this side was pinched, *she immediately complained of the pain.* She was able to move the left leg as well as the right, and both legs seemed endowed with the usual amount of motory and sensitive power. The pupils presented no unnatural appearance, but were of the usual size and shape; she complained of great pain in the abdomen, with flatus and a disagreeable taste

in the mouth; tongue moist; not much thirst; pulse rapid and irregular; bowels rather confined. The action of the heart was very irregular; *a loud blowing murmur, heard at the apex,* accompanied the first sound; there was no murmur in the neck.

Next day, Nov. 17, I examined her again very carefully, in order to form, if possible, a correct diagnosis; but although the paralysis of the left arm gave me reason to suppose that some disease of the brain existed, the other symptoms were too vague and unsatisfactory to point out the precise nature of the cerebral lesion. The pupils were of their natural appearance; she had passed a sleepless night; she was in full possession of her mental powers. The left arm was still perfectly sensible, though it had entirely lost the power of motion; no paralysis existed in any other part. The patient complained of great pain in the abdomen and back, increased on pressure; but as this external sensibility was general over the whole body, it was considered to be neuralgic.

Nov. 19. The symptoms are much the same, but she seems to be sinking; pulse very feeble, but the action of the heart remains strong, the murmur loud, and its movements irregular; tongue dry and furred; great thirst; headache, and occasional nocturnal delirium.

Nov. 20. On a physical examination of the chest, I detected a crepitant rhonchus at the lower and posterior part of the right lung. There was no cough, dyspnœa, or expectoration; but the presence of pneumonia was clearly indicated by the physical signs, and by them alone.

On the two following days, the symptoms remained essentially the same; the crepitant rhonchus continued, but, as at first, without any cough, expectoration, pain, or any other rational symptom of pneumonia. She gradually sank until November 23, when she expired, having been in the full possession of her mental faculties for many hours before her death, complaining of excessive tenderness of the abdomen and back, but never referring her complaint either to the head or the chest, which were the real seats of disease, and which were so considered by me during life.

Post-mortem Examination. *Head.* The skull-cap, skull, and dura mater presented no remarkable appearance. The vessels of the arachnoid membrane were very minutely injected; the membrane itself presented an opalescent appearance, owing to a quantity of thin, transparent serous fluid effused beneath it, and which escaped in great abundance from the surface of the brain and spinal cord during

the dissection. The substance of the *left* hemisphere was healthy, and of its natural consistence; the ventricle on the same side contained an unusual quantity of thin serous fluid. On examining the *right* hemisphere, a patch of softening was discovered in the anterior lobe, above the roof of the ventricle, and not extending to the surface of the brain; the softened part was two inches in length and one in breadth, and of a curved form; it had a dirty brown colour, and a ragged, ulcerated appearance. On inspecting the right ventricle, it was found that the corpus striatum of this side was softened, its surface presenting a ragged appearance, and being uncovered by the layer of membrane which lined the rest of the cavity. On tracing the diseased mass, it was found that the softening involved the whole of the right corpus striatum, and extended as far as the inferior surface of the brain. The whole of the softened part was of a pulpy consistence, and of a dirty ash-grey colour, the grey and white substances being apparently mixed up together in a confused mass.

Chest. The heart, on a superficial view, appeared larger than usual. The right auricle and ventricle, the pulmonary artery, and the venæ cavæ, contained a large quantity of half-coagulated blood, together with large coagula of fibrine of a straw colour. The parietes of both ventricles were rather thick, and the columnæ carneæ were increased in size. The tricuspid valve and the semilunar valves of the aorta and pulmonary artery were healthy, but the mitral valve was diseased. The opening was very much contracted, so as scarcely to admit one finger, and the curtains were thickened and indurated so as to form a strong, narrow tube.[1] The right lung was generally congested, and the posterior part of the inferior lobe was highly inflamed. The cut surface presented a dark-red colour, and a large quantity of frothy fluid flowed from the incision. The substance, however, was not hepatized, but crepitated as usual, showing that pneumonia had not proceeded beyond the first stage.

In connexion with this subject, namely, the occurrence of softening of the brain as the result of disease of the cardiac valves, Dr Kirkes, whose early loss the medical profession had reason to deplore, pointed out in a paper published in the year 1852, in the 'Transactions of the Medico-Chirurgical Society,'[2] that

[1] I ought to have discovered a præsystolic murmur in this case; but when it occurred, that murmur was neither described nor known to exist.

[2] 'Medico-Chirurgical Transactions,' vol. xxxv, p. 281.

softening of the brain may result from the obstruction of one of the cerebral arteries by the lodgment of a portion of fibrine in its canal. This plug of fibrine, by intercepting the passage of the blood, would partially cut off the supply from the brain, and thus lead to the *quasi* death of a portion of the cerebral mass. Dr Kirkes, as the result of his observations, was led to conclude that cerebral softening might result from the formation of such a plug of fibrine and its arrest in one of the cerebral arteries, and he moreover showed in his cases that the fibrine was detached from an inflammatory vegetation on the valves of the left side of the heart, and was thence carried into the circulation.

But the interesting observations and suggestions of Dr Kirkes were not confined to the explanation of cerebral softening alone in connexion with valvular disease of the heart, for he carried his generalisations much further, and stated that excrescences formed upon the valves, and capable of removal from their surfaces, are full of peril to the patient by being carried into the current of the blood, and afterwards arrested within some arterial canal, thus cutting off the supply of blood to some important organ. If the fibrinous masses are of large size and only loosely adherent, they may at any time be detached from the valves, and carried away until they plug up some important artery, or the deposits on the valves may be detached in smaller masses, and pass on into arteries of much less size, or even into the capillaries, where, being arrested, they may cause congestion or inflammation. The parts of the vascular system within which these transmitted masses of fibrine may be found will of course depend upon the side of the heart where they are formed. Thus, if they have been detached from the aortic or mitral valves, they will pass into the aorta and its subdivisions, and may be arrested in any of the arteries or their ramifications, or in the capillaries of the brain, liver, spleen, or kidneys. But if they have been detached from the pulmonary or tricuspid valves, then the pulmonary artery and its subdivisions will be the seat of the depositions. Dr Kirkes also suggests the probability that many cases of partial and temporary paralysis suddenly ensuing in one or more limbs, may, especially if accompanied with signs of cardiac disease, be due to interruption of the arterial supply to the brain from the temporary plugging up of a cerebral artery, the symptoms ceasing when the plug is dissolved or comminuted. But he adds that probably in the majority of cases the coagulum, when once firmly

impacted in the vessel, will tend to form an organised adhesion to the walls, and so produce permanent obliteration of the canal.[1]

Thus the arteries of the brain are by no means the only vessels in which these fibrinous masses may be arrested, and they have accordingly been found in other arteries, as the iliac, the femoral, and the renal arteries, in Dr Kirkes' cases, and in many other arteries, in cases subsequently recorded. The instances are now so numerous in which masses of fibrinous matter detached from the valves of the heart have been carried away with the current of the blood, until they have been arrested in some arterial branch and have caused the death or disorganisation of the structures beyond it, that almost a new department of pathology has been instituted, to which the term *Embolism* has been applied, from εμβολον, a plug. A very intelligent and laborious German physician, named Cohn, published, in the year 1860, a volume containing the whole history of the facts and theories which had then been presented to the medical profession on the subject of Embolism, and illustrated his text by many clinical observations of his own; and in reference to heart-disease he fully confirms the original views propounded by Dr Kirkes, to whom, however, he scarcely does sufficient justice. The German physician, perhaps from a feeling of patriotism, attaches far more importance to the researches and the theories of his countryman Virchow, who has indeed, in this as in many other departments of pathology, done eminent services to the science of medicine.[2]

The connexion between inflammatory disease leading to the formation of fibrinous coagula on the valves of the heart, and the establishment of secondary diseases in other and perhaps remote structures or organs, has not yet been fully investigated, and much information may still be expected in this new and interesting field of inquiry. It may, perhaps, eventually be found that many forms of parenchymatous diseases, as those of the liver, spleen, kidneys, lungs, and other organs, are not caused solely, as is generally supposed, by the physiological congestion due to deficient or perverted action of the heart, but by the mechanical obstruction of arteries or capillaries by plugs of fibrine arrested in the canal of nutrient arteries, or by disintegrated particles of fibrine carried into the parenchymatous structure of vital organs.

[1] Op. citato, p. 302.

[2] 'Klinik der embolischen Gefässkrankheiten,' von Dr. B. Cohn, Berlin, 1860.

CHAPTER VIII

THE PROGNOSIS OF HEART DISEASE

An opinion long prevailed among the public, and even in the profession, that diseases of the heart are especially dangerous, and that they are for the most part incurable; and almost every case of death, which is otherwise unaccounted for, is even now generally attributed, in the popular view, to disease of the heart. Now it is quite true, strictly speaking, that the life of every individual depends upon the healthy condition of this organ, for if it cease to beat, life must cease; but, in a medical and scientific sense, it is by no means true that diseases of the heart are more especially dangerous than those of other important organs of the body, or that they are less amenable to treatment. At a period when the results of auscultation were unknown, the most erroneous views prevailed as to the nature, the progress, and the significance of heart disease; mere guessing supplied the place of knowledge, and prognosis was nothing more than vague speculation upon an unsubstantial basis.

But the knowledge of heart disease has been most materially extended of late years, and this improvement in medical science is altogether due to the employment of the stethoscope, or rather of auscultation, coupled with the more frequent and systematic performance of post-mortem examinations, and the careful comparison of the symptoms observed during life with the lesions presented after death. At the commencement of the present century, the knowledge of the existence of heart disease was generally acquired only after the death of the patient, that is to say, if post-mortem examinations were made, which was not usually the case even in the great hospitals; and the phenomena observed during life, even if they were noted, were not referred to their true causes. Laennec himself, who first described with accuracy the morbid sounds of the

heart, failed, as has already been stated, to explain their true pathological significance, and even at present, with our accumulated knowledge and experience since the time of Laennec, something still remains to be learned in this direction. Making due allowance for difficulties in diagnosis, there can be no doubt that some serious lesions of the heart may occasionally escape detection during life, although the examination may be made by the most competent observers; and, on the other hand, the existence of the most marked murmurs does not invariably possess the same pathological importance which was even recently attached to them.

The labours and researches of Dr Hope, Dr C. J. B. Williams, and Bouillaud, perhaps contributed more than any others to the diffusion of correct diagnostic views in explanation of the morbid sounds which had originally been described, but not satisfactorily explained, by Laennec; and the first result of this newly acquired knowledge on the part of the medical profession was the detection of many cases of heart-disease which had previously been either mistaken or unsuspected. But as such disease in any form was then generally regarded as a most dangerous affection, and almost necessarily fatal, a great amount of alarm was excited in the public mind, and the injury thus occasioned almost counterbalanced the benefits which the revelations of the stethoscope, and the successful researches of physicians, had conferred upon mankind. In fact, in some cases, the knowledge acquired by the stethoscope led to the very consequences which medical science is intended to avert, and a needless amount of nervous suffering was inflicted on susceptible patients, who were unaware of being the subjects of any serious ailment, and whose lives were thus embittered by apprehensions although they might have perhaps suffered nothing at all from the actual disease. In one case, which occurred some years since, an amiable and well-known member of the medical profession, on being informed by a medical friend that he had a slight murmur over the cardiac region, was so depressed by the unwelcome and unexpected intelligence that he destroyed himself a day or two afterwards. Being a nervous man, he seems to have preferred actual death to the continual apprehension of that event, which might have occurred in the course of nature, and which would have been probably neither accelerated nor retarded by the knowledge which the stethoscope conveyed to his well-meaning but rather injudicious friend.

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The instance just related opens the question how far it is advis-

able for the medical attendant, who has discovered some abnormal sound or impulse in the heart of a patient, to inform the latter of the fact. In my opinion it is better, in most cases, for the medical man to keep the discovery to himself, or at any rate to communicate the intelligence only to the relations or intimate friends, and then leave them to take what course they may think proper. For, in the first place, it is probable that the information, directly conveyed, would produce an unfavourable effect upon the patient, since there are very few persons, even among those who possess the strongest mental powers, on whom the constant thoughts of death, which such information might inspire, would not exert a depressing influence, and perhaps cause the supervention of needless and long-continued suffering. It has been truly and beautifully observed that the "veil which conceals the future is woven by the hand of mercy," and although it is the duty of all to be prepared for death, which must come to every one sooner or later, it is not necessary that the idea of dissolution, especially of sudden death, which is usually attributed to heart disease, should be always present to the mind, for such an idea, instead of familiarising the patient with death, may rather render him unfit for life and incapable of fulfilling its duties.

In the case of a gentleman with whom I was intimately acquainted, I detected the presence of regurgitant disease of the mitral valve several years before his death, but as he was a man of nervous and somewhat desponding temperament, although of a highly intelligent, philosophic, and religious mind, I did not acquaint him with the fact, although I mentioned it to his family. The consequence was that unless when he was suffering from dyspepsia or bronchitis, which occasionally happened, he did not trouble himself about his health, and never directed his own attention to the state of his heart, and indeed experienced no inconvenience, as far as he knew, from that organ. With the exception of some attacks of chronic bronchitis, usually occurring in the winter, and of ordinary dyspepsia of which he now and then complained, he continued to enjoy very fair health till nearly seventy years of age, when he died of the cardiac disease the existence of which I had discovered during life. In his case it was quite unnecessary to remind him of the duties of religion, because, as I have stated, he was a very religious man, and it was equally unnecessary to caution him against imprudence in diet or otherwise, because he was in all respects careful, prudent, and abstemious. What is also rather remarkable is that, being desirous of

making a provision for his family, he managed to get his life insured at some assurance offices, although he was declined at others; and as he paid large premiums, in consequence of his comparatively advanced age, and extra premiums for bronchitis, and after all lived to about the usual period, perhaps neither he nor his family had any reason to complain, nor did the Assurance Offices probably suffer any loss. If I had been consulted in this matter, I could not have recommended the life in a strictly medical and scientific point of view, but he took the step himself. Reflections on this case, however, and on others to which I am about to refer, have led me to doubt whether even confirmed organic disease of the heart is always so soon or so inevitably fatal as to shorten materially the average duration of human life.

This leads me to the second point to which I wish to refer in connexion with the question of communicating the discovery of cardiac disease to the patient himself. I consider it unnecessary to do so, as well as inexpedient, because the most recent experience has shown that diseases of the heart, both acute and chronic, are far less fatal than is generally supposed; and while persons afflicted with such maladies may be unconscious of their presence, they may and sometimes do reach the ordinary term of human existence. But this point will be now fully discussed.

The forms of heart-disease which are usually and perhaps correctly described as the most common causes of sudden or early death, are hypertrophy and dilatation of the heart (particularly the latter), diseases of the valves, and fatty degeneration. These affections, and more especially the last, are often so insidious in their invasion and progress, and sometimes cause so little inconvenience, that they very frequently escape detection altogether during life, and their existence is revealed only by post-mortem examiuation. One of the most extraordinary instances, as it is supposed, of this kind was afforded by the case of an eminent Judge, who died suddenly while being lifted from his couch to his bed, and in whom, on post-mortem examination, there was found fatty disease of the heart, but no disease of the valves. The case is related in the 'Lancet,' of August 1, 1863, but it is imperfect, because the microscopical examination of the muscular fibres of the heart was either omitted or was not recorded, and in the absence of such information there is no positive proof of fatty degeneration. All authorities on this affection draw a very clear and marked distinction between fatty

accumulation on the heart and the interstitial fatty deposition within its muscular fibres, and mere softness and paleness of the muscular walls do not necessarily indicate fatty degeneration. Still the fact remains, that the Judge in question did actually die of a weak heart, the condition of which was known to no one, and was not suspected by himself, during life; and any reflections against the trustworthiness of medical science, so far as this case is concerned, must be silenced by the facts that no medical man was ever asked to examine the heart, that the deceased always believed himself to be in perfect and robust health, that he regularly performed his arduous and responsible duties for a long series of years, and that his death took place only indirectly, though suddenly, from the shock of an accident at a somewhat advanced period of life.

But not only is there this comparative immunity from suffering in those cases of heart-disease which steal on, as it were, in this gradual and imperceptible manner, but even the more severe and well-marked forms of valvular and other cardiac disease, either arising spontaneously or supervening on the progress of rheumatism, are often quite compatible with the enjoyment of life and the performance of its ordinary duties.

I think I may take credit to myself for having been one of the first to point out that disease of the heart, even of a decidedly marked character, and involving the structure of the valves, is not so dangerous to life as was generally supposed, and that persons may attain even to a very advanced age when labouring under such an infirmity. Several years ago I was attending the practice of a large infirmary which contained a great number of old people, and I made a point of conducting post-mortem examinations in every case, so far as it lay in my power, in order to compare the history of each during life with the appearances observed after death. I was rather surprised to find a considerable number of persons suffering from heart diseases who, nevertheless, attained to an advanced age, and many of whom were quite unconscious of having any affection of the heart at all.

In 1850 I presented a paper on this subject to the Medical Society of London, and it was read and discussed at one of the meetings. It was illustrated by cases which had fallen under my own observation, and I argued that the most serious diseases of the cardiac valves might exist without being attended with any remarkable symptoms, and might have no effect in shortening life or even

in materially deranging the health. I did not assert then, nor do I now, that such diseases are unattended with danger and risk to human life, but I maintained that the fatality and importance of such cases was exaggerated by the then existing race of medical practitioners. I even ventured to suggest that, in cases of Life Assurance, the mere existence of some abnormal sound in the heart should not be regarded in all cases as absolutely fatal to the eligibility of the life, although, of course, this opinion was given, and is now repeated, with many reservations.

The following are the remarks I made at the period to which I allude:—

"If it can be proved that actual organic disease of the valves of the heart may exist for a considerable period, and without shortening the average duration of life, such a conclusion may tend to divest this class of maladies of some part of the terror which their existence now so commonly inspires; and a patient, who is unfortunately labouring under a disease of this description, may nevertheless be encouraged by the reflection, that a disease of the heart, although of a decidedly serious character, may not prematurely put a stop to his career. And if such be the consolation which may, I think, fairly be afforded to those suffering under confirmed and extensive disease, how much more applicable is it to those who are affected only by some of those minor affections of the heart and its valves, which occur so frequently among the sequelæ of rheumatism. The subject is one of great importance in its relation to the average duration of human life, and the practice of life insurance. The existence of any disease of the heart is, I believe, an insurmountable barrier to effecting a policy of insurance; but I think it probable that, in proportion to the accumulation of facts bearing upon the subject, the deductions drawn only from the physical exploration of the heart, will not be so exclusively regarded as they have been of late years. But upon this point I wish to be understood as offering a very guarded opinion; for I have no wish whatever to undervalue the importance of the information derived from auscultation; nor do I pretend to assert that the existence of cardiac disease is a matter of slight moment. All I presume to argue is, that cardiac disease, even of a very decided and extensive character, *may* be compatible with long life and with moderate health.'[1]

[1] "Observations on Valvular Disease of the Heart," by R. H. Semple, M.D 'London Journal of Medicine,' November, 1850.

At the time when I first brought forward this view, it met with very few supporters and with much opposition, but at present I believe it meets with general favour and acceptance, and it has certainly been confirmed by subsequent experience. Putting out of consideration the numerous cases in which an erroneous diagnosis of disease of the heart has been given, there are many well-ascertained instances of patients with such affections who have lived to the usual period and been even unconscious of having any disease. Some of the individuals I had in view when the above-mentioned paper was written are alive now, and many, whom I have attended since, prove the truth of the same proposition.

Dr T. K. Chambers, in his valuable 'Lectures, chiefly Clinical,' arrives at somewhat the same conclusions as myself on this subject, and he considers, probably with justice, that the more tranquil the life of a patient with disease of the heart, the greater is his chance of comparative comfort and longevity. "With persons in easy circumstances," he says, "valvular lesions exist for years and years, perhaps through the greater part of a long life, and not only do not prove fatal, but may fail even to cause symptoms bad enough to make them consult a medical practitioner."[1] Dr Chambers then quotes, from his private notes, several cases of persons in easy circumstances in whom the stethoscope or the history of the case, or both, gave every indication of injury to the valves of long standing, but in whom no inconvenience sufficient to be called illness by themselves had followed. Dr Chambers proceeds, however, to observe that the evil import of valvular disease lies in its probable consequence, namely, enlargement of the muscular walls, in which view I do not at all agree with him, because the enlargement of the walls is probably to be regarded rather as a preservative power to ensure the transmission of the blood through the system than as an additional element of danger to the patient. This question, however, will be more fully treated hereafter.

Attempts have been made, but hitherto I think with little success, to determine the comparative danger to life and health caused by disease of the respective valves, and the difficulty and uncertainty which hang over the question are sufficiently indicated by the discrepancies in the statements of different writers who are each entitled to confidence. Thus Dr Walshe considers that the chief

[1] 'Lectures chiefly Clinical,' by Thomas King Chambers, M.D., p. 302, London, 1864.

valvular derangements may be placed in the following descending series on the basis of their relative gravity, namely, 1, mitral regurgitation and constriction; 2, aortic regurgitation; 3, pulmonary constriction; 4, aortic coustriction; while Dr T. K. Chambers maintains that the worst cases are those in which both the aortic and mitral valves are simultaneously imperfect; the next worst, those in which the aortic have suffered; and the most favourable, those where the mitral alone are inefficient.

In reference to the question of the relationship existing between the occurrence of sudden death and disease of the valves, Dr Walshe particularly draws attention to one valvular lesion, namely, aortic regurgitation, in which he believes that the tendency to kill the patient is particurlarly marked, and he urges that this fact should always be borne in mind in estimating the prognosis in valvular disease.[1] He relates a very striking instance in corroboration of this view, which occurred under his observation in 1852, and he states that since that time he has collected at least eight cases of a similar character.

I have had several cases under my own notice of aortic regurgitation, but I am not at present in a position either to confirm or to deny Dr Walshe's opinion on this point. I had a well-marked case of the kind under my care in the person of a man whose business was one of the very worst for a person labouring under such a disease, for he was obliged to navigate one of the large coal barges on the Thames, guiding the vessel with two huge oars and using necessarily a great amount of muscular exertion. He had had three attacks of acute rheumatism; there was visible pulse at both wrists; there was strong purring tremor (*frémissement cataire*) over the præcordial region, and there was a loud diastolic murmur over the base of the heart. He was under my notice for several months, and I earnestly warned him to try to obtain some lighter employment, but I eventually lost sight of him.

In another case, now under my treatment, there is both a systolic and a diastolic murmur heard over the aortic valves, and there is both a systolic and præsystolic murmur heard over the mitral valves, and yet the patient is carrying on the work of a porter, drawing a truck for a considerable time every day, and exposed to every kind of weather. I have had him under observation now for about

[1] 'Practical Treatise on the Diseases of the Heart and Great Vessels.' Third edition, p. 390, 1862.

two years, and he has been seen and examined by many of my medical friends, and I need hardly say that he has been warned by me as to the risk he runs in pursuing his avocation.

I have mentioned the above two instances only to show that most serious valvular disease is not incompatible with the performance even of laborious work. In several other cases which have been or are still under my observation, I have recommended, and succeeded in securing, light and easy occupation for the patients.

I am inclined to believe that a certain degree of valvular disease does not interfere materially with the circulation, and that even a very advanced state of such disease may still be compatible with the maintenance of life and tolerable health. The human organism possesses the power of adapting itself to varying circumstances, and if the blood be unable to pass with a full stream through the cardiac orifices, or if it meet with some impediments to its passage, it diminishes its volume in proportion to the obstacle it encounters, and the system gradually accommodates itself to the diminished supply. The languid stream is somewhat accelerated by the increase of the muscular walls which often attends or follows valvular disease, and hence I believe, in opposition to Dr T. K. Chambers, that the hypertrophy is advantageous rather than prejudicial in such cases.

It is almost impossible to explain how the body was nourished in some of the cases I am about to relate, and in which it will be seen that the aortic orifice was almost completely blocked up by osseous deposition.

CASE. DISEASE OF AORTIC AND MITRAL VALVES, APPARENTLY OF LONG STANDING. DEATH AT SEVENTY YEARS OF AGE. J. H., æt. 70, formerly a seafaring man, had been ill for some months before I saw him. I was asked to see him at a house where he lodged; he was standing up and dressed, but he complained of great debility, with cough and some expectoration. He made no particular complaint concerning his chest.

Physical Signs. Percussion gave no peculiar sound. Auscultation of the lungs gave no morbid indications. Auscultation of the heart gave a distinct but not very loud bellows murmur, accompanying the first sound of the heart, and prolonged so as in great measure to conceal the second. This murmur was heard at the apex of the heart as well as at the base, and there was no appreciable difference in the quality of the sound in either situation. The murmur was

not to be traced far above the base of the heart, and it was not heard at all over the carotid arteries. The impulse of the heart was not increased.

Diagnosis. Disease of mitral and aortic valves. I find it recorded in my notes made at the time, that the general appearance of this man was not indicative of the serious disease under which he laboured, and he merely looked like a person suffering from age, poverty, and privation. He was ordered to have a light and nutritious diet, and to take a mixture containing some tincture of henbane and a little ipecacuan wine; but he died rather suddenly and unexpectedly, after passing a restless night.

Post-mortem Examination twenty-four hours after Death. The body was that of a somewhat spare man, but was not emaciated. *Chest.* There were numerous adhesions between the costal and pulmonary portions of the pleura. The heart was somewhat enlarged, the apex being situated below the sixth rib. The right auricle and ventricle, with their valves, were quite healthy. The left ventricle was slightly thickened. The mitral valves were thickened with a cartilaginous deposit, but the auriculo-ventricular opening was patulous. The aortic valves were excessively diseased. Viewed from above, they appeared to be stretched entirely across the aorta, meeting in the centre, and allowing only a very small chink for the passage of the blood. Each segment was encrusted by a thick osseous deposit, of a nodulated character, which rendered it perfectly rigid. Viewed from below, the convex surfaces of the valves were seen to be also thickened with osseous deposit. There was likewise a deposit of bony laminæ in the aorta itself, a little above the valves.

In this case, there was obstructive disease of the aorta of the most formidable character, together with regurgitant disease of the mitral valves. It is, I think, impossible to doubt that the disease must have lasted for a considerable time, and yet the man attained an average old age, and did not, so far as I could ascertain, suffer any very severe or distressing symptoms.

In the next case, I was personally acquainted with the patient, and I know that she suffered no remarkable symptoms whatever.

CASE. DISEASE OF THE MITRAL AND AORTIC VALVES, APPARENTLY OF LONG STANDING. SUDDEN DEATH WITHOUT ANY PREVIOUS ILLNESS AT SIXTY-NINE YEARS OF AGE. Mrs. C., æt. 69, was a lady of very active habits, of mild disposition, temperate and abstemious, of rather tall stature and spare figure. Although acquainted with her

and her family for ten years previous to her death, and although I was the medical attendant of the family, I had never known her to complain of any illness. From personal knowledge I can state that she was by no means a person of sedentary habits, as she was continually attending to the duties of a large house, and I met her walking in the streets almost every day. One day, about four in the afternoon, I was requested to see her immediately, as she had fallen down in a fit, although she had been previously in perfect health. At the time of the attack she was superintending the affairs of the family, when she suddenly fell down. As I lived at that time next door to her, I arrived immediately upon being summoned, and found her lying on the floor with a pale countenance and very feeble pulse, and quite insensible. I was wholly at a loss how to account for her condition, as I had never known her to be ill during my whole acquaintance with her. I succeeded, with assistance, in placing her upon a sofa. Mustard poultices were immediately applied to the feet, and spirits of hartshorn held to the nose, but we had scarcely laid her down, when she expired.

Post-mortem Examination forty hours after Death. There was lividity on many parts of the body, apparently from gravitation, the lividity being chiefly observable on the under surface. Very little fat beneath the skin. *Head.* The vessels of the scalp were somewhat congested. The skull presented no remarkable appearance. The dura mater was slightly adherent to the inner table of the skull on the left side. Some fluid blood was found lying between the skull and the dura mater. The arachnoid membrane was somewhat opaque, and there was a considerable quantity of thin serous fluid effused beneath it; the veins of the pia mater were congested, especially in the posterior part of the brain. The brain itself, with the exception of being a little softer than is usual in persons of the age of the deceased, was quite healthy. *Chest.* The lungs were quite healthy, with the exception of slight congestion. The heart appeared externally to be larger than usual, but not remarkably so. There was very little fluid in the pericardium. The right auricle and ventricle, with their valves, were perfectly healthy. The left auricle was also healthy. The mitral valves were thickened by a deposition of semi-cartilaginous substance. The parietes of the left ventricle were very much thickened, but the cavity was of the usual size. The aortic valves were found very much diseased, and on looking at them from above downwards from the aorta, they ap-

peared completely to close the passage. They were found to be converted into masses of bone, which nearly filled up the valvular orifice, leaving only a small chink for the passage of the blood. These bony masses were rough and nodulated, and were deposited apparently within the sinuses of the valves, and one portion of great size extended from one of the valves down into the cavity of the ventricle. The lining membrane of the aorta also presented a great number of bony plates.

In this case of remarkable disease of the valves, the patient had enjoyed almost uninterrupted good health, and was in much better health, indeed, than most persons at her age. Nor can it be said that her immunity from unpleasant symptoms, and the delay of the fatal termination, was owing to unusual care on the part of her friends, or to the indulgence of rest; for she was a person of great activity, both of mind and body.

In the next case, the patient, who suffered from valvular disease of the heart to an enormous extent, actually lived to the age of *eighty-three* and died, eventually, of another complaint. With the history of this case I was well acquainted. I made the diagnosis of serious valvular disease nearly ten years before her death; and I had the gratification of confirming the correctness of my diagnosis, as well as of strengthening the opinion which I have been induced to entertain, that valvular disease, to a very great extent, *may* exist without shortening human life, or much impairing its comfort. I brought the heart, with a great portion of the aorta, and of the arteries of the head, to the Westminster Medical Society; and those who were present saw the accumulation of osseous matter in the aortic valves, along the course of the aorta, and in the carotid and cerebral arteries.

Case. Extensive Disease of Aortic and Mitral Valves. Death at the age of Eighty-three. G. D. died on the 4th April, 1850, æt. 83. The history of the case was as follows:—The patient was a thin, emaciated woman, of a pale complexion, who had long been an inmate of the infirmary. I began to attend her in 1840, and had seen her from time to time since that period. She stated that she had had several attacks of rheumatism previously to my attending her, and that she had been bled three or four times for those attacks. She had not suffered from any of the symptoms which are usually considered among the prominent rational indications of heart disease; such as swelled legs, palpitation, shortness

of breath, &c.; and although she came into the infirmary at various times, for several ailments, it was never on account of any cardiac symptoms; and it was only by auscultation that I became aware that she had any affection of the heart. In fact, I was disbelieved as to the existence of any serious disease in her heart; and although I frequently recommended that she should be kept constantly under medical superintendence, my recommendations were disregarded

January, 1842. She was seized with catarrh, but an attack of chronic bronchitis supervened. During this attack the state of the heart and lungs was particularly examined, when it was found that, besides mucous, sibilant, and sonorous rattles over the lungs, there was a loud rasp sound over the region of the heart. This sound was very loud, and much prolonged, taking the place of the first natural sound, and almost obscuring the second. This sound was heard most loudly over the base of the heart, and extended over the region of the arch of the aorta, and the right side of the neck The impulse of the heart was not stronger than natural: pulse 80, regular. I should here notice that the pulse was always regular, and feeble, whenever I examined it. The treatment was directed, in the first place, to the relief of the bronchitic symptoms; and on the 4th of Feb. 1842, the expectoration had diminished, and the rales had disappeared. The rasp sound, however, still remained as loud as ever, and I was convinced that the woman had confirmed and serious disease of the aortic valves. I thenceforward, therefore, always adopted a somewhat tonic method of treatment; recommending vegetable tonics, with light nutritious diet, and avoidance of exertion.

Oct. 5, 1846. She now complained of faintness, nausea, want of appetite, and a disagreeable taste in her mouth. She said that she had lately suffered from palpitation of the heart, and difficulty of breathing, when she exerted herself. She passed her water freely; never spat any blood; had no swelling of the legs: bowels open, tongue clean, no thirst, pulse 58, quite regular and weak. *Physical Signs.* The impulse of the heart was not increased, but the loud, sawing murmur was heard over the base, completely superseding the first sound, and was much prolonged; but a second, though much shorter murmur, was heard after it. Towards the apex of the heart this murmur was also heard, but not so distinctly; it was distinctly heard over the region of the arch of the aorta, and over the right carotid artery. The diagnosis was still,

of course, that the chief disease lay in the aortic valves. She was directed to take a moderate diet, consisting of mutton-broth, bread, milk, and tea; and to take some tonic medicine.

Oct. 7. Much the same; she complains of great faintness. She was ordered to have porter daily.

From that time until her death I had not many opportunities of seeing this woman; and I heard that she died (without manifesting any particular symptoms beyond those resulting from her great age) on the 4th of April, 1850, aged eighty-three.

Post-mortem Examination thirty-six hours after Death.—The body was emaciated; the legs were slightly œdematous; right side dull on percussion. *Head.* Scalp natural; cranium very thick, especially at its posterior part; sutures all obliterated; dura mater thick, very firm, and dense; arachnoid membrane of a pearly, transparent appearance, due to the presence of serum below its layers, which escaped on making an incision. The brain itself was remarkably healthy; no softening, nor extravasation, in any part; arteries of the brain rigid and patent, owing to osseous and atheromatous deposition,—this was especially the case in the ophthalmic, vertebral, and internal carotid arteries; very little fluid in the ventricles. *Chest.* Left lung appeared of its natural colour and consistence. The right pleural cavity contained a large quantity of fluid. About ℥xx of serous fluid were removed from this part; and in the fluid there was a large mass of coagulable lymph, very soft, and of the consistence of thin jelly. Right lung healthy, and crepitant at the apex; but below, the texture was of a very dark colour, friable, and heavy, so that it sank in water. On cutting into this portion of the lung, it was found very much congested, did not crepitate, and, on squeezing it, a bloody serosity flowed out. On this side, therefore, there was acute pleuro-pneumonia, which was probably the immediate cause of the woman's death. The heart, seen *in situ*, occupied its natural position, and the apex was placed between the fifth and sixth rib. After removing the lung on each side, the heart, with the arch of the aorta, the innominata, the left subclavian and carotid, and the whole of the thoracic aorta, were all carefully dissected out, and removed together. The right auricle was natural; the tricuspid valves were patent, and admitted three fingers; but the attached margins were rather hard, and thickened. The semilunar pulmonary valves were natural, but the pulmonary orifice was somewhat contracted. The left auricle was natural; the mitral

valves admitted two fingers, but they were thickened and hardened, and contained a considerable quantity of osseous matter; *the walls* of the *left* ventricle were *rather thickened, but not very considerably.* The aortic valves were completely rigid, and converted into bone; and they met in the centre, leaving only a small chink at their juncture in the middle line. Looked at from above, the valves were seen stretched across the aorta, and each of the segments contained, in its sinus, a large mass of bony deposit. Both the ascending and transverse aorta were very much dilated, being, at least, twice their natural diameter; the innominata was also very much dilated. The lining membrane of all these parts was most extensively diseased, part being converted into atheromatous matter, part exhibiting numerous plates of bone, and part being ulcerated. These appearances were found in many other arteries,—not only in the cerebral arteries, already described, but also in the subclavians and carotids.

This, then, was a case of extensive valvular disease of the heart, in a patient who suffered hardly any of the rational cardiac symptoms, but who was attended, at different periods, for various ailments, and finally died at the advanced age of eighty-three, of an acute attack of pleuro-pneumonia.

In the next case which I am about to relate, the patient is still living.

Case. Five Attacks of Acute Rheumatism. Pericarditis. Endocarditis. Disease of Mitral and Aortic Valves,—Hypertrophy of Heart.

The patient is a young lady, and I have her history from the earliest period: she is now twenty-nine years of age. She first had an attack of acute rheumatism in Belgium when she was ten years old. She was laid up for six months, and was said to have then suffered from disease of the heart. The second attack occurred under my care in 1854, when she had again acute rheumatism attended with the usual symptoms of that disease, and had also unquestionably pericarditis and endocarditis. Acting upon the principles of treatment which then prevailed, I bled her, and found that the blood was buffed and cupped. The bleeding appeared to do neither good nor harm. The other part of the treatment consisted in the free administration of lemon-juice, and after about six weeks she recovered completely. She subsequently took a situation as governess in a family. She had a third attack of rheumatism in

1860, when she was admitted as an in-patient at Guy's Hospital. The symptoms of endocarditis and pericarditis were again well marked, together with those of rheumatism. The principal treatment consisted in the administration of repeated doses of hydrochlorate of morphia and of bicarborate of potash. After remaining about two months in the hospital, she left it, but had a relapse soon afterwards. She eventually recovered and returned to her duties as a governess. She had a fourth attack in 1861, at Chislehurst, when the heart was again inflamed, and blisters were applied to the chest. Again, however, she recovered.

In May, 1863, she had a fifth attack of acute rheumatism, with inflammation of the heart, and this time the symptoms were more severe than ever. She was suffering from pains all over the body, but more especially in the joints, which were red, hot, swollen, and very tender. She had also great pain in the chest, but I attributed it rather to rheumatism of the diaphragm and the thoracic muscles than to the cardiac inflammation. The heart, however, was again unquestionably affected; the pulse was rapid, 130; there was distinct purring tremor over the præcordial region, the impulse of the heart was very strong, there was bellows-murmur over the base of the heart and also over the apex, synchronous with the systole of the ventricles. As I had now long been convinced that bleeding, if not absolutely injurious, was useless in such affections, I did not adopt that measure, but I applied blisters over the præcordial region, gave repeated doses of tincture of opium, and a large quantity of bicarbonate of potash, which was administered together with lemon-juice, the two being given in the state of effervescence. I found that the quantity of bicarbonate administered during the whole attack amounted to one pound and a quarter. The diet was almost entirely beef tea and milk. She was frequently delirious at night, but I attributed this symptom to the inflammatory affection of the heart, and I persevered in the use of the tincture of opium. The symptoms continued without much change for about five weeks, but she at last became convalescent about June 15, the attack having commenced at the beginning of May. For some time after convalescence she suffered so severely from neuralgic pains that it was rather difficult to determine whether the rheumatic affection was actually removed, but on June 22 she was decidedly better, the pulse had fallen to 84, there was no pain anywhere, and she slept well. Nevertheless,

the heart was still extensively diseased and the valves were seriously injured. There was increased impulse with purring tremor, and a loud systolic murmur over both the base and apex of the heart. She went home to the North of England, on the 6th of July, to resume her occupation as a governess.

The sequel of this case was curious, but satisfactory. She continued quite well, as far as she herself believed, and performed her duties without interruption, and in the spring of 1867 I was informed that she had accepted an offer of marriage, and was to sail with her husband, by the long sea-voyage, to New Zealand. I saw her on several occasions after her marriage, and before her departure, but as I did not see her in a medical capacity and was not consulted, I refrained from any physical examination of the heart. I have no doubt, however, that the abnormal conditions were the same as those I have described, and if my opinion had been sought as to the expediency of her taking a long sea-voyage, with its attendant discomforts and sea-sickness, I think that I could not have conscientiously recommended such a step; but fortunately I was not asked, and things took their course. I have been subsequently informed that she arrived safely at New Zealand, after an unusually long and very tempestuous passage, and has been confined of a child, and both thechild and herself, according to her own account, written in excellent spirits, are quite well.

This is a case of undoubted valvular disease of the heart, following or accompanying attacks of acute rheumatism, and yet the lady is quite able to perform the ordinary duties of life, and, moreover, encounters and accomplishes a long sea-voyage, and is now in apparently perfect health.

The prognosis as to the results of disease of the heart, even when such is unquestionably proved to exist, ought therefore always to be formed with great caution, and with the full assurance of the fact that even extensive disease of the valves is not incompatible with the maintenance of life, the enjoyment of fair health, and the attainment of average longevity. I am inclined to believe that some of the worst and most suddenly fatal cases of heart disease are those which we have at present no means of accurately distinguishing during life, and which are only revealed by the microscope after post-mortem examination; but perhaps in course of time the diagnosis of such cases may be as precise as of those which are now ascertained during life by means of auscultation.

Still, while arguing that valvular and other diseases of the heart are not so dangerous or so rapidly fatal as was once supposed, I by no means maintain that they are unattended with great peril, or that they do not often involve serious complications. The comparative immunity from suffering enjoyed by many persons who are labouring under heart-diseases is indeed in great measure due to the absence of those derangements in other organs which heart-disease so often entails. As long as the blood is propelled sufficiently, though perhaps not fully, through the chambers of the heart, and thence into the system or into the lungs, there may be no material interference with the functions of life, and even although the blood may be thrown back upon the lungs, as happens in mitral regurgitation, these organs may, by habit, escape any material damage. But when, as often occurs, the blood is insufficiently supplied to the system in consequence of obstruction of the valves, the vital organs fail to receive their due amount of nutrition, the blood is not impelled with sufficient force, and capillary congestion, or, as has before been mentioned, even softening of the brain, may ensue; or the patient may die at once from syncope or exhaustion. When, also, the blood is sent back into the cavities from which it ought to have been completely expelled, a whole train of secondary disorders is often induced, for the blood in the cavities of the heart then reacts on the column behind it, and congestion ensues in the lungs, or the liver, or the kidneys, or the capillaries of the body, or in the general system of the veins. Hence arise bronchitis and pneumonia, and pulmonary apoplexy, and hæmoptysis, as far as the lungs are concerned; and congestion of the liver, and the spleen, and the kidneys; and infiltration of fluid in the general areolar subcutaneous tissue, constituting anasarca, owing to exudation from the congested capillaries, and to the inability of the overloaded veins and absorbents to take up the effused water. Weakness of the heart, also, from dilatation of its walls or degeneration of its muscular structure, may lead to congestion, in consequence of the inability of the left ventricle to propel the blood with sufficient force, for the capillaries thus become overloaded, and fail to transmit the blood to the veins.

Thus, therefore, a variety of symptoms supervene which might be mistaken for those of other diseases, if the stethoscope did not point out the real source of the pathological changes in the system. Among the most common symptoms is indigestion, resulting from the congested condition of the liver and the imperfect secretion of

bile, and sometimes this is the chief or only symptom of which the patient complains. Cough and spitting of blood are by no means constant or characteristic symptoms of heart-disease, but the latter symptom often attends regurgitation through the mitral orifice, which condition most frequently also induces bronchitis, pneumonia, and pulmonary apoplexy. The congestion of the kidneys may cause the urine to be scanty, high-coloured, or albuminous; and the congestion of the mucous membrane of the intestines may induce constipation. Dropsy is one of the worst symptoms in heart disease, as it indicates a general failure of the vital powers, and the blood itself often loses its due proportion of red corpuscles, and by the exudation of its watery constituents through the capillaries contributes to swell the amount of the dropsical effusions.

The absence or the appearance, then, of these secondary diseases or symptoms will assist very materially in the formation of a prognosis of heart diseases, when it becomes necessary to estimate the danger of such affections, or to calculate the chances of longevity in a given case. The habits and mode of life must also be taken into account in forming an opinion, for there can be no doubt that tranquillity of mind and body, and a moderate enjoyment of the comforts of life, will materially assist in mitigating the symptoms of heart disease, and in warding off secondary and dangerous affections; while, on the contrary, a life of toil and privation, and exposure to the weather, and the depression of mind so generally associated with poverty, will aggravate the dangers of the cardiac affection, and accelerate the supervention of dangerous complications. With regard to diet and habits of living, the rule "in medio tutissimus ibis" will be found most applicable. All excess in either eating or drinking must of course be avoided, but on the other hand, a very low diet is decidedly objectionable. The orifices of the heart being contracted, or their valves being insufficiently closed, a greater effort is required on the part of the organ to propel the stream of blood through the system, and a moderately generous diet is required in order to accomplish this object. For the same reason, a carefully regulated allowance of alcoholic stimulants is not only unobjectionable, but to a certain extent necessary; and sherry, or even port, with gin, whisky, or brandy, may be beneficially administered. In confirmed cases of heart-disease the lighter wines, having very little alcoholic strength, are not of much service, and porter and ale are perhaps objectionable, as they may aggravate the dyspeptic symptoms of

which cardiac sufferers very frequently complain, and which are sometimes the only subjective signs of the existing disease.

In reference to the subject of Life Assurance and the eligibility of a person in whom some degree of heart-disease may be found to exist, my opinion is that such a life ought not always to be absolutely declined. In some life offices, as in that of which I am the physician, it is a rule that any deviation, however slight, from the standard of perfect health is an obstacle to eligibility; but in other offices it is often considered safe to guard against extra risk by the imposition of a larger premium. The remarks I have made in reference to prognosis in heart-disease sufficiently indicate my opinion that an extra premium might probably counterbalance the risk incurred in some cases of heart-disease, when the affection is uncomplicated and the patient is in easy circumstances and of regular habits. But each case should be most carefully considered in all its bearings if such a question should arise.

CHAPTER IX

A GENERAL SKETCH OF CARDIAC PATHOLOGY

Diseases of the heart are of the most varied and even opposite character. Sometimes the muscular structure of the organ is increased; sometimes it is diminished; sometimes it is degenerated. Sometimes the organ itself is enormously enlarged; sometimes it is shrivelled into very small proportions. Sometimes the disease begins on the external membrane, sometimes on the internal. sometimes it attacks both simultaneously. In some cases the morbid condition results from some constitutional poison circulating in the system, as that of rheumatism; in others it is due to the natural degeneration of the tissues in advanced life; in others, to the excessive use of alcoholic liquors. Sometimes premature degeneration developes itself in early or middle life. In very many cases where the heart has been the seat of great suffering to the patient, no material or perceptible lesion can be detected by the scalpel or microscope after death, and yet such functional derangements must be included among the varieties of cardiac pathology. For pathology, in its enlarged but true sense, includes not only those visible changes which the chemist, the anatomist, or the microscopist can demonstrate, but also all those perversions or aberrations of functions which, although they cannot be presented as objective realities, are equally the causes or the essences of disease. A few preliminary remarks on cardiac pathology in general will form an introduction to the special sections on the diseases of the heart.

1. *Disease of the heart may commence on its external membrane.*—Disease does not commonly originate in the heart itself, except in the case of fatty degeneration of the muscular fibre, and in the rather rare one of cancer of the organ. Inflammation of the heart itself, or Carditis, as an idiopathic affection, can hardly be said to

exist, or at any rate there is but little evidence of its existence, and hypertrophy and atrophy of the heart are changes usually consecutive upon other diseases or upon affections of the pericardium or endocardium. Inflammation of the pericardium, involving, of course, the serous membrane which immediately invests the heart, causes great disturbance of the cardiac functions, and indeed is marked by symptoms quite as important and dangerous as if the heart itself were attacked. There are, besides the physical signs peculiar to pericarditis, violent disturbance of the circulatory and respiratory apparatus, rapid, tumultuous, and irregular pulse, palpitation of the heart, shortness of breath, and occasionally faintness. When pericarditis terminates in the effusion of fluid, constituting dropsy of the pericardium, the action of the heart is embarrassed by the pressure of the fluid, and unless the malady is relieved, death must result. But when it terminates in the effusion only of coagulable lymph and the consequent agglutination of the adjacent surfaces of the serous membrane of the pericardium, the results are not so immediately dangerous as in dropsy, and in fact are not incompatible with life and restoration to a moderate degree of health. Still, from the obstacles opposed to the movements of the heart within the pericardium, or from some other cause not easily explained, it often follows that the muscular structure of the heart in such cases becomes excessively developed, and hypertrophy is induced, leading by gradual steps to a greater and greater embarrassment of the circulation. Atrophy, or wasting of the heart, has also occasionally been seen to arise from the pressure of effused lymph resulting from pericarditis.

2. *Disease of the heart may commence on its internal membrane.*—As the pericardium, or membrane surrounding the heart, may be inflamed, and its inflammation may lead to consecutive changes in the structure of that organ, so the inflammation of the endocardium, or membrane lining the cavities, may lead to the same results. It will be shown hereafter that the symptoms of endocarditis during life, if we exclude the physical signs, are very obscure, and even after death the appearances are by no means characteristic, except those observed on the valves. Nevertheless, the existence of endocarditis is fully established, and many if not most of the forms of valvular disease are due to its agency. It should be remarked that although the pericardium and the endocardium are separated by the muscular walls of the heart, yet a close sympathy of struc-

ture or function, or both, seems to exist between these two membranes, and when one is inflamed the other is similarly affected in a great number of cases, though perhaps not invariably. Hence while a layer of albumino-fibrine covers the surface of the heart as the result of pericarditis, it is most probable that a similar exudation from endocarditis is at the same time taking place in or upon the valves; and, when the acute symptoms have passed away, the adhesion of the adjacent surfaces of the pericardium is contemporaneous with a thickening or hardening of the valves. From the embarrassment caused by both these conditions the action of the heart, its muscular walls are stimulated to increased efforts to propel the blood, and hence often arises hypertrophy, a condition demanding special and particular examination, and not to be confounded with mere enlargement of the heart.

3. *The muscular structure of the heart is increased.*—The increase of muscular structure in any part of the body may ensue from increased action in that part, as in the arm of the blacksmith or in the calf of the leg in the ballet-dancer; a d so the muscular structure of the heart may be increased by any cause which excites it to undue action. Again, in some persons, without any special reason, there is a tendency to the formation of a large proportionate amount of muscular fibre in all parts, and in the heart among the rest. Thus it often happens that without the existence of any known or appreciable disease, the impulse of the heart in a person otherwise healthy is considerably increased, showing a somewhat undue thickness of the muscular structure; but if this physical sign is unaccompanied by any murmur, and if the dulness of the præcordial region is not too extensive, and there are no general signs of cardiac derangement, the phenomenon is perhaps not of much importance. The muscular structure and the size of the heart vary also very materially in different individuals within the limits of health, and therefore a mere increase of impulse is not to be necessarily regarded as a pathological indication, although it may prove that the heart's walls are somewhat thicker than usual. As the greater or less amount of impulse of the heart within the limits of health cannot be accurately described in words, the practitioner should constantly exercise his hand and his ear, both with and without the stethoscope, to determine for himself what is the average healthy impulse of the heart, and what, on the other hand, is the amount of force which may be considered excessive.

4. *The muscular structure of the heart is diminished.*—An opposite condition to that just described is where the muscular structure of the heart is diminished from want of healthy nutrition. As the increase described above may be due to a general tendency to an excessive development of muscular fibres throughout the body, so the diminution may be referred to the opposite cause. Thus it may happen, from general defect of nutrition, that the muscular structures of the body all suffer diminution, and that of the heart among the number; and although this condition may be due to mere constitutional peculiarities, yet it is most strikingly marked in certain wasting diseases, such as phthisis and cancer. Very frequently, however, a diminution of the muscular substance of the heart is found to exist, while the rest of the body is apparently healthy and well nourished. As in the case of increase of the muscular structure, a moderate degree of increased impulse is not a pathological condition of much importance, so, on the other hand, a somewhat diminished impulse need not be regarded in a serious light; and indeed there are many persons whose heart is naturally small and weak, but who nevertheless enjoy good health. Still, as in the case of the increased impulse the practitioner ought to be able to recognise any deviation from the average standard of health, the same observation is to be made as to the opposite condition, which is far more likely to occur in the present day. For a number of depressing causes, as the increasing cares and anxieties of life, the speculative character of the age, with its attendant reverses, the excessive cultivation of the intellectual as compared with the bodily powers, the constitution of the atmosphere, and other recognised or unrecognised causes, are now leading to the prevalence of the asthenic over the sthenic forms of disease; and the diminished size and power of the heart, either caused by or causing other depressing conditions of the system, constitute a very general ailment, and one which, if neglected or misunderstood, or erroneously treated, may lead to very serious consequences. No definite rules can be laid down in writing as to what is a morbidly diminished impulse of the heart, but the knowledge must be gained by experience, and remedial measures must be adopted if such a condition should present itself. It is only by practice, and by comparison of a great number of cases with each other, that morbid increase of the heart's structure and action on the one hand, and morbid diminution of its structure and action on

the other, can be fairly determined, and the collateral symptoms and other circumstances will very much assist in forming the diagnosis.

5. *The heart is hypertrophied.*—As the heart is enlarged in this condition, it may at first sight appear that hypertrophy ought to have been included in the third section, treating of the increase in the muscular structure of the heart; but the causes which produce hypertrophy and those which merely lead to increased muscular development are really very different, and what in the latter case is a circumstance of no very great importance is, in the former, a pathological change of the highest significance. It has already been mentioned that the muscular structure may be increased merely as a coincidence with the general muscular development of the body, and it may be slightly in excess in the heart itself from some undefined and inexplicable disturbance of the balance of the local nutrition of the organ. But true hypertrophy or excessive development of the muscular structure of the heart, accompanied by excessive action, is induced by morbid changes in the constitution of the blood, by inflammation of the external and internal membranes of the heart, or, as is very commonly the case, by diseases of its valves; or it is caused by morbid states of organs with which the heart is intimately and functionally related. Thus, as already mentioned, hypertrophy may be, and perhaps often is, induced by pericarditis; and the existence of disease of the valves, obstructing the passage of blood along the arteries and through the chambers of the heart, calls for increased efforts on the part of the muscular fibres, which are consequently augmented and thus hypertrophy is set up. The same effect is produced by some chronic diseases of the lungs, which, by obstructing the passage of the blood and interfering with its due aëration, cause its accumulation in the cavities of the heart, and require a corresponding amount of force to remove the obstruction. Unfortunately, too, the hypertrophied heart too often fails in effecting its object of propelling the blood sufficiently through the lungs and through the system, for it impetuously drives the fluid through contracted orifices, or the blood is sent back through imperfectly closed valves, and thus the arteries are insufficiently filled, the blood is imperfectly oxygenated, and the structures at a distance from the heart are inadequately nourished or they are passively congested, while the heart itself is increased in bulk and beats with immoderate force.

6. *The heart is dilated.*—In hypertrophy, the muscular walls of the heart are increased in thickness, and the impulse of the heart is also unduly increased; but when the cavities, as often happens, are augmented in capacity while the muscular walls are diminished in thickness, the condition called dilatation is produced. As in hypertrophy the impulse is unduly augmented, so in dilatation it is unduly diminished, because in the latter case the muscular walls are thin and weak, and their propulsive power is thereby enfeebled. But although the conditions of hypertrophy and dilatation are, when taken separately, opposite to one another, yet it is found in practice that they are usually combined, for the increase in the thickness of the muscular walls is most commonly accompanied by an increased capacity of the cavities of the heart, and thus the whole organ is greatly increased in bulk, and its cavities are at the same time distended with an unusually large quantity of blood. As may be expected, therefore, the symptoms of this combined condition will be a combination of those respectively denoting hypertrophy and dilatation.

7. *The heart is shrivelled into very small dimensions, or is atrophied.*—This condition, as contra-distinguished from enlargement or hypertrophy with dilatation, is by no means common, and in fact it is exceedingly rare as an idiopathic affection. Dr Walshe, in his 'Treatise on Diseases of the Heart,' states that he has only in a single instance imagined that he met with an example of this kind, and then he did not verify his opinion by post-mortem proof. He has, however, seen an atrophous condition of the left ventricle, produced by the close embrace of pericardial induration matter,[1] and Laennec observed the same fact. Andral has also found the heart atrophied in some cases of chronic pericarditis, in consequence of which disease some very thick and dense false membranes were formed round the heart, and enveloped it like a kind of nutshell.[2]

8. *The muscular structure of the heart is degenerated.*—Thus it has been shown that the muscular structure of the heart may be excessive or defective, and the impulse will be accordingly greater or less in proportion. In all these cases the muscular fibre undergoes no appreciable pathological change, but preserves its usual anatomical characters when examined under the microscope. In certain cases,

[1] Walshe on 'Diseases of the Heart,' p. 274.

[2] 'Traité de l'Auscultation Médiate,' par Laennec. Andral's edition, vol. iii, p. 227.

however, the heart undergoes degeneration, which is usually of the fatty kind, and may or may not coexist with the deposition of an undue amount of fat throughout the body. In cases of fatty degeneration of the heart its bulk may be neither increased nor diminished, and no alteration of structure may be visible to the naked eye. Unfortunately, too, the symptoms of this disease during life are obscure, and notwithstanding the light that has been thrown in later days on its pathology, its diagnosis and treatment are still the subjects of anxious thought among medical practitioners. The deposition of fat about the heart is common enough, more especially in persons who have passed the meridian of life, and is hardly a circumstance deserving much special notice; but the substitution of minute oil-globules for the muscular elements constitutes a very serious but insidious disease, the causes and the results of which are even now but imperfectly known.

9. *Diseases of the heart may result from morbid matter or poison circulating in the system.*—Inflammation of the pericardium and of the endocardium, and, as a consequence, disease of the heart itself, are often associated with or caused by certain morbid principles or poisons circulating in the system, these morbific agents being either matters in themselves natural to the human body, but existing in excessive quantity, or excretory products which are retained instead of being eliminated, or they are substances introduced into the organism from without. Thus a very common cause of heart disease is the existence of the poison of rheumatism, whatever that poison may be, which is generated from the natural constituents of the body. Another cause of heart disease is the presence of an undue amount of urea in the blood, urea being an excretory product which ought to pass away from the kidneys; and pericarditis and endocarditis, with their consequent evils, may also be produced by the excessive absorption of alcohol, in consequence of the abuse of fermented liquors, which may act as poisons to the whole system and to the heart among the other organs. The remarks just made involve some theoretical considerations which may be discussed hereafter.

10. *Diseases of the heart may result from the natural degeneration of the tissues in advanced life.*—This is a very common source of disease of the heart, and one which has not been hitherto sufficiently described. It has been too often the practice to attribute almost all diseases of the heart and its valves to an

attack of rheumatism or to uræmia, or to some analogous cause, but it is scarcely sufficiently understood that cardiac diseases are very often nothing more than the natural and necessary results of the decay and degeneration which the body undergoes in its onward progress to age and death. It is true that in some favoured organisms the seeds of dissolution are developed late, and persons arrive at a great age in the apparent integrity of their bodily and intellectual faculties; nay, more, it must be admitted that in some very rare instances, the vital spark is at last extinguished by the mere feebleness of the system, and post-mortem inspection reveals no appreciable organic change. But, in by far the greater number of cases, some insidious, if not declared malady, is gradually sapping the foundations of life as years advance, and the supervention of serious changes in the brain, the heart, the lungs, the liver, the kidneys, or other important organs, is the evidence that the elements of disease have triumphed over the resistance offered by the living and healthy organism. As Mr (now Sir James) Paget has very justly and eloquently remarked, "All the expressions usually employed about these changes imply that they are not regarded as the result of disease, nor should they be; and were it not that the forces which are efficient in degeneration are probably very different from those which actuate the formative processes, we might justly call the degeneration of advanced age another normal method of nutrition. For, to degenerate and die is as normal as to be developed and live; the expansion of growth, and the full strength of manhood, are not more natural than the decay and feebleness of a timely old age; not more natural, because not more in accordance with constant laws, as observed in ordinary conditions. As the development of the whole being, and of every element of its tissues, is according to certain laws, so is the whole process regulated, by which all that has life will, as of its own working, cease to live. The definition of life that Bichat gave is, in this view, as untrue as it is illogical. Life is so far from being the sum of the functions that resist death, that it is a constant part of the history of life that its exercise leads naturally to decay, and through decay to death."[1]

The fact is that, as years advance, the tissues undergo some change involving decay or degeneration, and they either simply wither away or they become loaded with fat. The latter substance may develope itself on the surface of the body or around

[1] 'Lectures on Surgical Pathology,' by James Paget, F.R.S., vol. i, p. 94.

the viscera, giving the ordinary characters and appearances of obesity, or it may insinuate itself into the substance of the vital organs and structures, replacing their natural and healthy constituents. Hence the internal and middle coats of the arteries often present, when seen under the microscope, the appearance of being scattered over with a multitude of minute oil-globules, which at length coalesce, giving, even when seen by the naked eye, a peculiar pultaceous or pappy and patchy appearance on the lining membrane of arteries, resembling atheromatous degeneration. Of the morbid and fatal results of this deposition in the arteries of many of the vital organs, and in the arteries themselves, it is not my purpose at present to treat; but the lining membrane of the heart, being analogous to that of the arteries, undergoes very frequently the same morbid change. Hence the valves, which are reflexions of the lining membrane, become incapable of duly performing their functions of regularly opening and closing; the circulation is embarrassed, the respiration suffers, and chronic disease of the heart and lungs is established. As a further step in the progress of degeneration, the atheromatous or fatty masses become converted into osseous matter, and brittleness of the arteries and ossification of the cardiac valves are the necessary consequences, giving rise, as the case may be, to sudden rupture of the vessels, to obstruction of the circulation in the extremities ending in gangrene, or to fatal impediment to the passage of blood to or from the heart. These changes, thus considered, can therefore be scarcely regarded as diseases, in the strict sense of the term, but rather as the natural consequences of the alterations which the human body, in the greater number of cases, must gradually undergo.

11. *Diseases of the heart may arise from premature degeneration in early or mature years.*—The changes to which allusion has just been made are those usually attendant on old age; but it often happens, whether from faulty habits in the individual, or from obscure and inexplicable constitutional predisposition, that the same process of degeneration commences in early or mature life, and cuts off the individual at an untimely age. Thus the disease known as fatty degeneration of the heart is by no means confined to the aged, and some of its victims are in the prime of life. It often happens also, especially to those who examine persons for the purpose of life assurance, to find in subjects apparently healthy, both young and middle-aged, some abnormal

murmurs about the aortic valves, or along the large vessels arising from the heart; and making due allowance for the existence of anæmic and venous murmurs, there can be little doubt that in such cases there often exists degenerative disease of the middle and internal coats of the arteries, and incipient disease of the valves, and it is probable that such disease, though often unsuspected by the patient, may suddenly or gradually develope itself into some dangerous or fatal malady. But I think that very great caution should be exercised in pronouncing an opinion in such cases, and I will relate one or two instances in reference to this suggestion.

Case.—A Colonel in the army, a tall, robust man, about fifty years of age, who had lived freely, was brought to me for examination for the purpose of Life Assurance, and on listening to the region of the heart, I distinctly heard a blowing sound, synchronous with the systole of the heart, over the arch of the aorta and along the tracks of the carotid and subclavian arteries. I discovered no morbid sound over the heart itself. I reported that the case was not eligible for life assurance, on the ground that degenerative disease was in all probability advancing in the great vessels, and would also probably reach the vital organs and the heart. Attempts were made to insure the life in other offices, but the physicians took the same view as I did, and the life was declined. In about a year afterwards, this gentleman died from what is called a breaking-up of the constitution.

Case.—A gentleman, about fifty-four years of age, of robust configuration and healthy appearance, but who had indulged very largely in alcoholic drinks, consulted me for symptoms of dyspepsia, characterised by loss of appetite, sickness, and other similar inconveniences, but not by any signs indicating cardiac disease. On examination I found a distinct blowing murmur over the apex of the heart, synchronous with the systole. Without mentioning the exact nature of the case to the patient himself, I warned him to be very cautious in his habits, and I gave an unfavourable prognosis to his relatives. He died in a few months afterwards of a general break-up of the constitution, the prominent symptoms being general dropsy, and the immediate cause of death being, as I was informed, dropsy of the pericardium.

The cases which follow are of a different character.

Case.—A young gentleman, aged about twenty-four, whom I had

known ever since he was a boy, who had never suffered from any illness, and whose habits were in every respect regular and moderate, desired to insure his life, but was declined for reasons which, of course, were not explained to him. Although he had not consulted me as to the insurance, he came to tell me that he had been declined, and asked me to examine his chest. I did so, and discovered that there was a blowing murmur over the aorta and the great vessels springing from it, and this abnormal sound was, no doubt, the cause of his rejection. I could not myself recommend the life for acceptance, but the gentleman went to another office, where either the morbid sound was not detected, or the physicians did not attach much importance to its presence, or the Directors were not very particular, and the life was accepted. Since this occurrence, about ten years have elapsed and the gentleman in question is quite well, and I have very strong doubts whether the auscultatory sounds really indicated any important lesion in the great vessels.

Case.—A gentleman, aged about twenty-five, of perfectly regular habits, wished to effect an insurance on his life. He was, as far as he knew, in perfect health, and had never suffered from any serious illness, but on being examined by a medical gentleman in a town in Hampshire, the latter reported to the assurance office that, although all the organs were healthy, there was an anomalous sound which he could not explain, beneath the left clavicle, and he therefore wished that the proposer should be examined by a physician in London. He accordingly came to me, and I found that he was in all respects healthy and robust, his weight, age, and stature being all in good proportion, and his heart and lungs quite sound. But the medical gentleman in the country was perfectly right as to the abnormal sound beneath the left clavicle, for there was a distinct, though not loud murmur over a limited portion of the subclavian artery, synchronous with the pulse at the wrist. Taking all the circumstances into consideration, and with my present experience of murmurs such as those described, I did not attach much importance to the abnormal sound, which I believe may be due in such a case to accidental pressure from surrounding parts, even if it be not caused by some unusual condition of the blood. But the life was declined.

The above cases are related and contrasted only for the purpose of showing that while the presence of murmurs over the heart or great vessels is often of great importance as indicative of organic disease,

its significance is in other cases, to say the least, dubious. The collateral circumstances should always be taken into account, and ought to form an important element in the prognosis. I offer no dogmatic opinions upon the subject, but I think that the whole question of the significance of arterial murmurs as affecting Life Assurance deserves the most careful revision.

CHAPTER X

THE SPECIAL DISEASES OF THE HEART

I. PERICARDITIS

Pericarditis is an inflammation of the membrane covering the heart, and is one of the most important diseases to which that organ is liable, because it is not only a dangerous affection in itself, but it gives rise to consequences of a very serious nature. It is characterised by the ordinary conditions of inflammation, namely, swelling, redness, heat, and pain; but these symptoms are either undiscoverable during life in consequence of the position of the heart, or they are unfelt by the patient, owing to collateral circumstances which mask their existence. Pericarditis is recognisable to the eye only by its results, discoverable on a post-mortem examination, but its presence is determined in the living subject by a careful examination and comparison of the general and physical signs.

Pathology and Morbid Anatomy

Pericarditis being an inflammation of the pericardium, and inflammation consisting essentially, at least in the first instance, of a dilated condition of the capillaries, which are distended with blood, it should follow that *redness* would be one of the most prominent appearances seen on the inflamed membrane. But although this coloration does exist, and its degree and its varieties have been described by authors, it is by no means essential that its presence should be detected after death, because, in the first place, inflammation being a process set up in the living body, it is possible that none of its primary appearances may remain after life has ceased; and in the second place, even in the living body, after effusion or adhesion has taken place, no traces of active inflammation may be discoverable.

Redness again, in the dead subject, is by no means to be regarded as necessarily the result of pericarditis, or indeed of any inflammation at all, because the natural tendency of the arteries, just before death, is to discharge the blood they contained during life into the capillaries, and thus the latter very frequently assume a dilated condition, which, although often attributed to inflammation, may have no connexion with that pathological state. After death, therefore, the only trustworthy proofs of inflammation are to be drawn from the presence of the results, which consist of effusions of various kinds, as of serum, lymph, or pus, or of thickenings caused by interstitial depositions in the substance of membranes or organs.

The most characteristic appearances after death from pericarditis are effusions of albumino-fibrine (or lymph), or of serum into the pericardial sac, or the presence of adhesions between its adjacent surfaces. It was once asserted as an axiom in pathology that the inflammatory processes in the case of serous and mucous membranes respectively were different in their nature, and that while inflammation of serous membranes was followed by the exudation of serum and coagulable lymph, that of mucous membranes was attended by the exudation of pus. Under the influence of these views the inflammation of the pericardium would necessarily determine the effusion of serum and coagulable lymph. But Sir James Paget has very ably shown that the distinctions just alluded to, and which were laid down by that very eminent pathologist, Bichat, are not really founded on fact, and that the nature of the exudation depends rather upon the vital conditions of the system at the time of the exudation than upon the anatomical formation of a particular structure. The characteristic primary product of the inflammatory process, according to Sir James Paget, is the liquid known under the name of "lymph" or "coagulable lymph," but which has more recently been named "inflammatory exudation." It is probably always at its first exudation a pellucid liquid, transuding through the capillaries of the inflamed part, and its most characteristic properties are its capability of spontaneously organising itself, and its power, after its organisation, of developing tissues like the natural structures of the body. The effusion of serum, according to the same authority, is probably a rare event except as the result of the lowest degree of inflammation or when the serum is a diluent of other products. What is usually regarded as a serous effusion in inflammation is in many cases a fluid that contains fibrine. Again, when the inflammatory lymph,

instead of developing itself into the natural tissues of the body, undergoes degeneration, it may be converted into pus, and this, indeed, is the most frequent form of such degeneration.[1]

These doctrines, though very much amplified and extended, are in great measure those which are now accepted by modern pathologists as explaining the results of inflammation, but the process has been lately most carefully watched by competent observers. Some of them, especially Cohnheim, have actually seen the migration of the white corpuscles of the blood through the walls of the blood-vessels, and there seems now to be very little doubt that these white corpuscles are identical with the pus-cells, while the fluid in which the pus is suspended closely resembles the fluid portion of the blood or liquor sanguinis. It would appear that in inflammation the walls of the minute vessels undergo some molecular change, which allows of the transudation of the white corpuscles, while the latter assume a vital activity and undergo various changes of form (hence they are called amœboid leucocytes), and are thus enabled to escape from the containing vessels by a vital process (*Germ in*, Bewegliche Korperchen, Wanderzelle). Such being the case, it is fair to consider the process of suppuration as essentially one of degeneration; for inasmuch as the white corpuscles of the blood are the *first* formed, and are developed into the red ones which undergo no further change, so, in inflammation followed by suppuration, the elements of the blood seem to go *back* to their original condition of *leucocytes*, or white corpuscles. But inflammation is not always followed by suppuration, and indeed what is called (though perhaps erroneously) *healthy inflammation* has no such result, but is chiefly characterised by the effusion of a large number of cell-structures which multiply themselves by the formation of new cells, and eventually constitute new formations.[2]

Regarded in this view, then, the exudation in pericarditis consists primarily and essentially of the formation of coagulable lymph, although there may exude at the same time that thin fluid which looks like serum, but which really contains fibrine. Still, serous effusion

[1] 'Lectures on Surgical Pathology,' by James Paget, Lecture XIV.

[2] For a masterly description of the modern views of the inflammatory process, the reader is referred to the article by Dr Burdon Sanderson on the "Changes which have their Seat in the Blood-vessels," in the fifth volume of Holmes's 'System of Surgery.' In this article the researches of Dr W. Addison in 1842, of Dr Augustus Waller in 1846, and the more recent investigations of Recklinghausen and Cohnheim, are fully explained.

does also occur in pericarditis, but only, as Sir James Paget would say, as the result of the lowest degree of inflammation. If the pericarditis should terminate in resolution, namely, an entire obliteration of the inflammatory process, which I suspect is seldom the case, there will of course be no appreciable alteration of the normal structures; but in the great majority of cases there is an exudation of lymph and of fibrinous serosity, giving rise to adhesions between the adjacent surfaces of the pericardium, and in other cases, probably in those where the vital powers are very low, there is an abundant effusion of serum, giving rise to the disease called *hydro-pericardium* or *hydrops pericardii*.

The presence of pus in large quantity in the pericardium as a result of pericarditis is, I apprehend, a very rare circumstance, and indeed the existence of this fluid either in the heart itself or its membranes has not often been detected. The reason probably is that the great amount of vital force residing in this central organ of the circulation prevents the inflammation from assuming any form of degenerative action in which, according to modern views, suppuration must be supposed to consist. Cardiac inflammation is more likely to be attended by the generation of plastic matter capable of organisation, and such indeed is generally the case.

The appearances observed after death in a recent case of pericarditis are very characteristic, and cannot possibly be mistaken. The surface of the membrane is covered with a partial or continuous layer of lymph, of a pale straw colour, and of a soft consistence, and of greater or less tenacity and firmness, while at the same time there is usually present more or less fluid of a thicker or thinner consistence, and showing its fibrinous character by the flakes and filaments of solid matter floating within it. The adherent surface of the coagulable lymph, the thickness of which may vary from a line to half an inch, is smooth, while the opposite surface is rough, and presents a shaggy appearance like the wool upon the back of a sheep. The free surface is sometimes pitted with small depressions at almost regular intervals, resembling the appearance of a honeycomb, or that of the second stomach of the calf. Such is the aspect when the layer is thin and uniform, but it is often thick and irregular, and then the depressions are larger and the partitions thicker and more coarse.

The tendency of this effusion, as I have just observed, is to organise itself and to become developed into a substance resembling the natural structures, and this organisation of lymph, says Dr Hope,

"sometimes takes place with astonishing rapidity, as within the space of twenty-four hours—a fact which has been ascertained by experiments on living animals, and by pathological observations on the human species. The exceptions I have generally found to occur," he continues, "either in very intense inflammations, where the violence of Nature's operations would seem to counteract their sanitary tendencies, or in atonic and cachectic subjects of bad constitution, in whom the lymph effused is of an unhealthy character, and ill-suited for organization, just as we find in the case of external wounds affecting the same subject."[1]

Now, although pericarditis is a serious disease, and the consequent effusion is a morbid process, yet the presence of coagulable lymph is really a beneficial operation of Nature to obviate the irritation which would otherwise ensue from the friction of the inflamed surfaces upon one another. Mr Hilton, in his very thoughtful and suggestive lectures on the "Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases," shows how Nature in very many instances provide in serious diseases those very appliances for the repose of injured parts which medical science and surgical art do their best to imitate. Thus, in the case of a fractured limb, when the fractured pieces are not nicely adjusted or are subjected to frequent local disturbance, Nature throws out a quantity of soft material, technically called *callus*, which keeps the fractured ends of the bone at rest and promotes their union, or in other words she provides a soft splint placed on the exterior of the bone, including the fracture. So in the case of inflamed serous membranes, if they are allowed to rub constantly and freely on one another, the irritation must necessarily be increased, but by the coagulation of the lymph upon the free surfaces, the latter are protected against friction, as in the case of the inflamed pericardium. As soon as the lymph is poured out, the serous membrane is put, as far as possible, in a state of rest, or freedom from friction. In discussing the question of treatment, it will be seen how important it is to bear in mind the necessity of *rest* as a therapeutical agent in diseases of the heart, and to imitate and to assist the operations of Nature in the cure or alleviation of such affections.

But besides providing a temporary couch or pillow on which the

[1] Hope on 'Diseases of the Heart,' p. 139.

inflamed heart may repose while the inflammatory action is proceeding, the development of the coagulable lymph into an organised structure is in itself a beneficial operation, or at least it is the preferable of two evils. If the surfaces remained separate and the inflammatory process continued, the heart would be subjected to continual irritation, and the sac of the pericardium would be distended with inflammatory products so as to threaten constant danger. But by the adhesion of the opposite surfaces, by which result all further effusion is prevented, life may be prolonged for years, and, as I believe, without any material inconvenience in a great number of cases. I am quite aware that in thus somewhat underrating the gravity of adhesion of the pericardial surfaces I place myself in opposition to the opinions of some eminent authorities; but I think that modern experience will confirm the views I adopt, namely, that such adhesion is the most favourable termination which can generally be hoped for in pericarditis, and that its occurrence is not necessarily incompatible with average longevity or even immunity from suffering.

In this view I am confirmed by the high authority of Dr Stokes, who, without denying that a general adhesion of the pericardial surfaces may induce hypertrophy and dilatation of the heart, yet doubts whether such an effect necessarily or even commonly follows that process. He has often found the heart itself in a perfectly natural condition, although the pericardial sac was obliterated, and he observed the patients exhibiting no symptoms of heart-disease for many years before death. In one of his cases, seven years had elapsed between the attack of pericarditis and the death of the patient from hepatic disorder, and during this interval no symptoms of disease of the heart were manifested. Again, he remarks that when pericarditis is followed by recovery (as I may observe it very generally is under modern treatment), we cannot doubt that adhesion more or less complete has occurred, and yet any increased liability of such patients to enlargement of the heart has not fallen under his observation. He goes on to suggest that it is in the cases where pericarditis is accompanied or followed by valvular disease that hypertrophy and dilatation supervene, but that in cases where there is no evidence of valvular lesion there need be little apprehension of the after-occurrence of organic cardiac disease.[1] Dr

[1] 'The Diseases of the Heart and Aorta,' by William Stokes, M.D., 1854, p. 11.

Stokes also adduces physiological arguments to show that although hypertrophy may be supposed to result from the efforts of the heart to propel the blood through a contracted orifice, as in obstructive valvular disease, yet that the effect of obliterated pericardium would be to diminish the contractions of the heart, and thus to induce atrophy rather than hypertrophy. He concludes, from his experience and from physiological reasoning, that obliteration of the pericardium does not necessarily induce any manifest change in the condition of the heart; that where any alteration of its muscular structure is found in connexion with this obliteration, it is not necessarily a state of hypertrophy, but may be of an opposite nature; that valvular obstructive disease is more likely to cause hypertrophy than is adhesion of the pericardium; and that analogy shows that obliteration of other serous membranes is more often followed by atrophy than hypertrophy.[1]

The process of adhesion, although it is difficult to fix the period of time which it occupies, is easy of explanation on well-known physiological principles. The effusion of lymph, which at first is of the consistence of jelly, gradually becomes thicker and more tenacious from the absorption of its fluid portions, and the opposite sides of the serous membrane become, as it were, glued together. The solidified lymph then becomes connected with the membranes from which it was originally poured forth, and vessels are formed in the newly organised structure which convey the blood from one surface of the membrane to the other through the new tissue, until at last there is no further trace of lymph at all, but the pericardial sac is obliterated. What once was lymph is now fully organised areolar tissue, and the heart and the pericardium form one undistinguishable structure, only to be separated after death by dissection. But no great embarrassment appears to be offered to the movements of the heart from this obliteration of the pericardium, because, as the heart, in the natural state, fills almost, if not entirely, the pericardial sac, it cannot be much impeded in its motions when the latter has its surfaces combined instead of being separate. As the adhesion of the surfaces of the pleura offers no embarrassment to the action of the lungs, and is indeed a very common and by no means dangerous condition, so it is easy to conceive that adhesion of the pericardial surfaces may offer no very great obstacle to the movements of the heart, though at the same time

[1] Op. citato, p. 12.

it must be admitted that in the latter instance other pathological changes co-exist or follow which may entail serious consequences. All I intend to maintain is that these consequences are not proved to be wholly due to inflammation of the pericardium and adhesion of its opposite surfaces.

Among the appearances once supposed to denote the previous existence of partial pericarditis are the white or milky spots often observed on the surface of the heart, but there is certainly no evidence that they are due to such a cause. These spots, which vary in size from that of a fourpenny piece to a half-crown, are usually found on the surface of the right ventricle, are about as thick as the nail, and may sometimes be detached without injury to the pericardium beneath, although in other cases they appear to be situated below the membrane. They are more commonly observed in adult than in early life, and are found more abundantly in old than in middle-aged persons. Of late it is generally believed that they are due to friction rather than inflammation, and this is probably the true explanation of their existence; but an attempt recently made to show that these spots are more common in soldiers than in other persons, and that they are caused by the loaded packs and the cross-belts worn in military service, does not appear to me to be altogether justified by the facts. The occurrence of these spots on the hearts of persons in all conditions of life is so common that there is no reason to suppose that soldiers are especially liable to them. I have often observed them myself in post-mortem examinations.

Besides coagulable lymph, which is the primary and normal product of pericarditis, and which, as has just been mentioned, tends to become organised and to develope into adhesions, serous fluid is also sometimes poured out in great abundance into the pericardial sac, although only perhaps as the result of the lowest form of inflammation; and pus, which may be regarded as degenerated lymph, is occasionally, though rarely, found in the same locality. In treating of serous effusion into the pericardium (or pericardial dropsy), a distinction must be drawn between the instances which are due to the existence of inflammation, even of a low type, and those which are found as concomitants of general dropsy, or which result from a passive exudation of fluid from the serous surfaces of the pericardium. Nothing is more common than to find in post-mortem examinations a greater or less amount of fluid in the peri-

cardial sac, and this effusion may scarcely deserve to be considered morbid: and again, the pericardium may present a serous effusion only in common with the other cavities in the body, as is the case in general dropsy. But still, after excluding these cases, a certain number of instances remain, where, instead of the effusion of coagulable lymph, the pericardial inflammation gives rise to the effusion of a large quantity of serum, sometimes amounting to more than a pint, which is rapidly poured out in the pericardial cavity, thus seriously embarrassing the action of the heart, and threatening death by its mechanical pressure upon that organ.

Signs and Diagnosis.

The symptoms of pericarditis may be divided into the *general*, or, as they are often called, *rational*, and the *physical*, the latter being those deducible from percussion and auscultation. It may be stated at once that the general signs are often absent, are sometimes fallacious, and not rarely are positively misleading; while the physical, although far more trustworthy, are by no means free from difficulty in their detection and appreciation.

The general symptoms are those which denote inflammation of the heart, and it might be supposed that the most prominent would be those of inflammatory fever and of disordered circulation and respiration. Hence it might be expected that besides the ordinary characters of fever, there would be rapidity and irregularity of the action of the heart, pain over the præcordial region, and difficulty of breathing, and these symptoms really present themselves in many cases. But pain is far from being a constant indication of pericarditis, and M. Bouillaud has found it so often absent that he attributes it, when present, to the co-existence of pleurisy, and Dr Hope, although he does not altogether agree with M. Bouillaud in this opinion, yet states as the result of his extensive experience that in the great majority of cases of pericarditis the pain was either wholly absent or was of a mild and endurable kind. When pain does exist, however, it is an important sign, more especially when it is increased on pressure in the interspaces between the ribs, or upwards under the left hypochondrium; and when the patient makes no complaint of pain, it may sometimes be elicited by pressure in the region just indicated. If this symptom, which is very characteristic, should be accompanied by fever and increased action of the heart, then, as Dr Hope says, there can be little doubt of

the existence of pericarditis. The pain, when present, is increased by full inspirations; the patient is unable to lie on the left side, and sometimes he can lie only on the back; there is acceleration of the breathing, palpitation of the heart, and rapidity, though not always irregularity, of the pulse. Still, few of these symptoms can be relied upon as diagnostic of pericarditis, for many of them may be caused by other inflammations of the chest, as for instance pleurisy, with which indeed pericarditis is often associated. As an element of difficulty in the diagnosis of pericarditis, it must never be forgotten that this disease very frequently, and indeed generally, accompanies or follows acute rheumatism, which is itself a very painful affection, involving not only the limbs and joints, but the muscles or at least their sheaths. Hence there may be very acute pain over the chest, increased by pressure, by movement, or by the act of respiration; and yet the pericardium may be free from disease. This circumstance should never be forgotten in estimating the value of pain as a diagnostic symptom of pericarditis. For my own part, for the reasons indicated, I place but little reliance on this sign. If pericarditis should terminate in extensive serous effusion, or in other words, dropsy of the pericardium, the heart's action will become weak, and the pulse will become small, feeble, intermittent, irregular and unequal, and there will also be faintness, paleness, cold perspiration, and eventually the other usual symptoms denoting the approach of death.

But among the most common, although one of the most anomalous, of the general symptoms of pericarditis, is delirium, a condition which, when present, not only masks all the other general symptoms of the disease, but may actually lead to the belief that the brain, and not the heart, is the organ affected. Dr Stokes, indeed, mentions that in Irish practice delirium tremens not unfrequently accompanies pericarditis, and that typhus fever is also a not unfrequent concomitant of this inflammation; but, setting aside such cases, there can be no doubt that delirium is very often a symptom of uncomplicated pericarditis, and this circumstance should be carefully borne in mind by the medical practitioner. It did not escape the notice of Andral, Latham, and Hope, who describe "a fictitious inflammation of the brain" as likely to divert the attention from the organ really affected, and Sir Thomas Watson particularly mentions this source of fallacy in his Lectures. Some little time ago I was attending a case of pericarditis, in which

delirium was a very marked symptom, and I had some difficulty in persuading one of the other medical attendants that the patient was not labouring under inflammation of the brain, but the result proved that my diagnosis was correct. This case is recorded in a subsequent page.

Such being the uncertainties of the rational or general symptoms of pericarditis, it cannot be a subject of wonder that the presence of this disease is often overlooked, or that pericarditis is sometimes mistaken for or confounded with other affections. But as it is very frequently, although not invariably, found to co-exist with or to supervene upon rheumatic fever, it is always usual, during the course of the latter disease, to make frequent examinations of the præcordial region with the view of detecting, at the earliest possible period, the physical signs which will now be indicated.

In the section devoted to the consideration of the "Morbid Sounds or Murmurs of the Heart" (page 61), the nature and the value of the auscultatory signs furnished in pericarditis are pretty fully explained, and it will be unnecessary to repeat at length the observations which are there given. It is only necessary to state that the exudation of lymph on the pericardial membrane gives rise, from physical causes which are easily explicable, to a grating or rubbing sound, which is in fact generally known as a *friction-sound*, and as this particular sound is developed on the outside of the heart, it is called an *exocardial* murmur. But the sound in question is said by some writers to be due not only to the exudation of lymph on the serous surface of the pericardium, but also to that early condition of the disease which exists before any exudation has taken place, and when the serous surfaces are merely in a dry state in consequence of the absorption, or the absence, of the natural serous secretion. In this case the grating sound would be due, not to the presence of adventitious matter, but to the deficiency of the fluid by which the surfaces ought to be lubricated, and the sound would be acoustically analogous to that which is heard in the axle of an ungreased wheel, or in a portion of a steam-engine where the moving rods and pistons are not duly supplied with oil. But it is very difficult, if not impossible, to *prove* that the friction-sound is so produced, although argument and analogy appear to support such a view; for the stage of dryness of the serous surfaces is soon succeeded by that of exudation, and it could only be in the very rare instances, where death occurs before the inflammation is fairly set in, that

post-mortem evidence could be adduced to show that a friction-murmur might be developed without exudation.

Dr Stokes, however, seems to disbelieve the assertion that a friction-murmur is heard under such circumstances, and although he is one of the most eminent authorities upon the value of the friction-sound in pericarditis, he considers that the first stage of the disease, before exudation, is not discoverable by physical signs. This period before exudation lasts only a few hours, perhaps for a day or two, and during this time the nature of the disease can only be a matter of plausible conjecture. But when the friction-murmur, in the progress of an acute disease, and especially of acute rheumatism, is suddenly developed in the præcordial region, there can be very little doubt of the real character of the case. It has already been mentioned that although the *bruit de cuir neuf*, or new-leather sound, was previously described by M. Collin, a French physician, yet the true nature and significance of the exocardial sounds were first clearly pointed out by Dr Stokes, who in 1833 and 1834 published valuable memoirs on the subject, and indicated the distinction existing between these and other murmurs.[1] The pericardial friction-sound is double, corresponding to the systole and diastole of the heart, and has been aptly though familiarly designated by Sir Thomas Watson, from its essentially double character, as a *to and fro* sound. Supposing it to be clearly proved that the serous inflammation is not that of the pleura (and the situation of the pleura and its relation to the function of respiration will furnish the diagnosis), then a friction-murmur of a double character, over the præcordial region, will in all probability indicate the existence of pericarditis. But I would here repeat what I observed in a former page, that it is by no means very easy to distinguish between the exocardial friction-murmur denoting pericarditis and the endocardial murmur arising from disease of the valves; and it is pretty certain that before the first memoir of Dr Stokes appeared, physicians were in the habit of confusing the sounds together, and attributing them all to an endocardial cause. For it must be remembered that the *new-leather sound* is by no means the only friction-sound heard in pericarditis, and, as Dr Stokes pointed out in 1833, the sounds may, even in a single case, undergo a series of

[1] "Contributions to Thoracic Pathology," by Wm. Stokes, M.D.; 'Dublin Journal of Medical and Chemical Science,' vol. iii, 1833; and "Researches on the Diagnosis of Pericarditis," in the same Journal, vol. iv, 1834.

modifications, passing from the loudest sound of friction of two very rough surfaces to a sound similar to the most faint *bruit de soufflet;* and, on the other hand, I have now under my care, a case where the disease is distinctly chronic and endocardial, but in which the murmur is of such a loud grating or scraping character that it might be supposed to be due only to the rubbing together of two almost cartilaginous or horny structures. It is therefore by no means exclusively by the sounds themselves, all of which I have already shown, whether exocardial or endocardial, to be due to friction, that the diagnosis can be made, and the Profession is indebted to Dr Stokes for having been the first to describe the real grounds of distinction.

These grounds have already been stated to be the double nature of the sound in pericarditis, while the sound in valvular disease is usually (but not always) single; and the localisation of the sound in pericarditis in the region of the heart itself, while in disease of the aortic valves the sound is generally propagated to the aorta and to the great arterial trunks arising from its arch. Dr S okes adds, moreover, that by pressure with the hand, or by increasing the pressure of the head on the ear-piece of the stethoscope, a notable increase will be produced in the loudness and distinctness of the exocardial friction-sounds, but not of the endocardial murmurs. It must also be recollected that the exocardial murmur, from its very nature, must be transient, and that it can only exist so long as there is a layer of lymph of a certain degree of thickness between the serous surfaces of the pericardium; and when these surfaces have either been widely separated by serous effusion or have been consolidated together by the process of adhesion, the friction-sound must necessarily disappear, and in the latter case it can never return. When, therefore, a murmur continues for a very long time, it cannot be exocardial.

But it follows from the same explanation that during the existence of acute pericarditis the friction-sound may appear and disappear, or present varying degrees of loudness or of quality, in proportion as the effused lymph is greater or less in quantity, or is perhaps of thicker or thinner consistence. In proportion as the effusion increases, the distinctness of the friction-sound diminishes, but if the effusion should subside the friction-sound may probably reappear. In such cases the sound diminishes gradually from below upwards, and when it re-appears it gradually increases from above

downwards. It should also be noticed that the friction sound may be absent in pericarditis, for where the fluid effusion is in large quantity the sound will necessarily be lost. Thus it will be seen that while the presence of the friction murmur, under the circumstances and with the limitations just specified, denotes the presence of pericarditis, the absence of the murmur by no means proves the reverse, and a very serious inflammation may exist although the friction sound has passed away or perhaps may never have been heard.

Such are the auscultatory signs of pericarditis; but the sounds derived from *percussion* are very little different from those observed in health, unless the serous effusion is very considerable. As the bulk of the heart is not increased in simple pericarditis, the extent of dulness over the præcordial region is not altered, but when the serous sac is distended with fluid, the resonance of the præcordial region is diminished in extent in proportion to the amount of the effusion. By the pressure of the effused fluid the apex of the heart is pushed out of its place between the fifth and sixth rib and thrust up to the second, third, or fourth intercostal space, and the area of dulness on percussion mounts up the sternum in the direction of the great vessels. (See HYDROPS PERICARDII.) In short, when a large serous effusion exists in the pericardial sac, the action of the heart is necessarily embarrassed, its sounds become indistinct, and its impulse is feeble and irregular, and therefore, if it should be found that in a case of pericarditis the friction-sound disappears and the natural sounds and impulse of the heart are diminished in intensity or become lost, it is evident that effusion to a very great and dangerous extent has ensued.

Perhaps in the great majority of cases pericarditis terminates in adhesion, and this condition appears to constitute in the minds of some writers the disease which they call *chronic pericarditis;* but I consider that the name is rather erroneously applied, as the adhesion is a mere consequence of the pre-existing inflammation. Attempts have been made to ascertain, by physical exploration, the existence of adhesion, and in cases where pericardial friction-sound has been distinctly heard and has afterwards entirely ceased, it may generally be assumed that adhesion has taken place. Dr Hope, however, describes as a character of adherent pericardium a strong jogging and sometimes double jogging action of the heart, caused by the organ being bound to the spine by the adhesion; but I am inclined to agree with Dr Walshe that the positive diagnosis of adhesion of

the pericardium is an impossible problem in most cases, and I can quite concur in what he says as to his having known adhesions found after death, when, notwithstanding frequent physical examinations of the heart, their existence had previously not even been suspected, and on the other hand the pericardium being found after death in a perfectly normal condition, where adhesions had been affirmed during life with overweening confidence.[1]

Still, it will most probably happen that, in consequence of the heart being bound to the pericardium, and thus prevented from exerting its natural freedom of action, the ensiform cartilage will be drawn in towards the cavity of the chest, and this sign, in connexion with other evidence of embarrassment in the cardiac functions, will afford a fair indication of the existence of adherent pericardium.

[1] Walshe on 'Diseases of the Heart,' p. 241.

2. ENDOCARDITIS

ENDOCARDITIS is an inflammation of the lining membrane of the heart, and very generally, though not invariably, it accompanies pericarditis. It is so common a complication that, before the researches of Dr Stokes and the publication of his memoirs in 1833 and 1834 above alluded to, no distinction was recognised in the respective symptoms. But that distinguished physician showed, by post-mortem evidence compared with physical exploration during life, that pericarditis and its auscultatory signs might exist without any endocardial disease; and, *vice versâ*, it is conceivable that endocarditis may exist without any accompanying pericarditis. But I repeat that the two diseases are usually found in combination or in close relationship with one another, are amenable to the same treatment, and arise apparently out of the same pathological conditions; although the local appearances, the physical signs, and the ultimate consequences of each are different. Our knowledge of endocarditis as a distinct disease is justly due to Bouillaud, the veteran physician of the Hôpital de la Charité in Paris, but it cannot be affirmed that the malady is so well marked as pericarditis, its local characters being far less distinct, its pathology more obscure, its physical characters not by any means free from fallacy, and its general symptoms being even more uncertain than those of pericarditis.

PATHOLOGY AND MORBID ANATOMY

What has been said on the subject of post-mortem appearances in pericarditis is equally applicable in the description of endocarditis, namely, that the existence of inflammation during life can only be detected by the presence of the *results* of that process, and not by any persistent traces of the inflammation itself. Hence it is in vain to expect that the redness, or in other words the excessive

vascularity of the lining membrane during life, will be continued after death; and the redness, which is often actually found in post-mortem examination of the heart, is due only to a passive congestion of the capillaries, generally independent of any inflammatory action whatever. Unlike the pericardial membrane, too, the endocardium does not become coated, by the process of inflammation, with a layer of adherent lymph, and its surface may for the greater part of its extent be found apparently in a normal state after death, although it may have been the seat of inflammation during life. The endocardium being constantly bathed in a current of blood, any serous fluid which may be exuded and even some of the lymph or the coagula which may possibly be developed by the inflammatory process, are washed away as soon as they are formed, and it is almost only by the coagulated or semi-coagulated masses of lymph deposited upon the surfaces of the valves that the existence of acute endocarditis can be demonstrated after death.

The true inflammatory character of endocarditis is therefore known by the appearance of lymph on the free surfaces of the valves, and the valves themselves are sometimes found thickened, puffy, and swollen. The valvular apparatus of the left side is much more frequently inflamed than that of the right, although it is not easy to give an explanation of this circumstance upon any pathological reasoning; but it is stated that the inflammation may be limited to the right side in certain cases, or may affect both sides. It is a curious fact that in fœtal life inflammation of the valves of the right side of the heart is not uncommon, and I had lately a well-marked case in which the disease had led to chronic disease of the right valves and right ventricle, the patient eventually dying at the age of 19. The specimen, taken from this patient, and exhibiting right valvular disease, is now in the Museum of the Royal College of Surgeons of England.

There can be no doubt that inflammation of the valves is attended with very serious consequences, perhaps not so much immediate as remote, for these structures, either by being swollen and thickened offer an obstruction to the passage of the blood, or, by the inflammatory process, they are made incapable of closing completely, and thus they permit of regurgitation. This disease of the valves often continues for the rest of life, and although, as has been previously remarked, its existence is not inconsistent with longevity or with comparative comfort, it is nevertheless a serious complication.

It will hereafter be shown how the valves, under the influence of chronic disease, gradually lose their elasticity, or become thickened by the interstitial deposition of lymph or other matters, or coated by vegetations; and how, from analogous causes, or from actual laceration, they become incapable of closing and thus permit the regurgitation of the blood. From the obstruction offered by the thickened valves on the one hand, or from the reflux of the blood on the other, the muscular structure of the heart is stimulated to increased action in order to meet and overcome these morbid conditions, and hence arise hypertrophy and dilatation, or in other words increased bulk and capacity of the heart. I have already expressed an opinion that this increase is far less due to pericarditis than to endocarditis, and indeed it is not very intelligible, arguing on general pathological grounds, how pericarditis can induce hypertrophy.

But there is another danger arising from the existence of plastic exudations or excrescences on the surface of the valves which has been recently pointed out, and to which I have already referred (see p. 98), namely, the liability of fragments of the organised lymph to be carried away, in the current of the circulation, from the valves where they were originally deposited, and to be conveyed to other and sometimes distant parts or organs, where they may form solid plugs blocking up important arteries, and inducing, according to the locality and the structures involved, mortification of an extremity, or inflammation or congestion of a parenchymatous tissue.

Endocarditis may be *acute* or *chronic*, and the latter is very frequently the consequence of the former. It is very difficult to determine in cases of long-standing disease, and where there has been no acute attack, whether the affection of the valves is really due to inflammation, or to those changes in the tendinous and membranous structures to which the term *degeneration* is more strictly applicable. In acute endocarditis, however, the process is undoubtedly inflammatory, and the morbid changes take place in the deeper layers of the endocardium, which are infiltrated with young cells, giving rise to new tissue causing the epithelium to project, and forming vegetations on the surface of the valve. In chronic endocarditis, the morbid process is the same in its nature, but more slow and gradual in its progress. As a matter of practical experience, acute endocarditis usually passes into the chronic form, the exceptions being the instances where resolution takes place, and all traces of the acute inflammation are removed; but I apprehend that this fortunate

result rarely follows, although it is difficult to prove absolutely either the positive or negative side of the problem.

Signs and Diagnosis.

The symptoms of endocarditis are still obscure, and although Bouillaud insists very strongly upon the possibility of detecting the disease during life, he admits that it may be confounded with pericarditis, and I am rather inclined to think with Dr Stokes that its diagnosis will never be established with the same precision as that of the latter affection. Dr Stokes, indeed, states that he has never met with a case of simple idiopathic endocarditis fit to be considered as a type of the signs and symptoms of the disease; and I suspect that of the many cases recorded by Bouillaud in his well-known *Traité Clinique des Maladies du Cœur*, several are not instances of pericarditis at all, but rather of phlebitis and pyæmia.

For instance, in the first three cases recorded by Bouillaud as illustrative of acute endocarditis, there was evidence in all of disease of the veins, two being due to traumatic causes, and the third to very severe chilblains. In the first case, in which phlebitis was set up by bleeding in the arm, the appearances after death are described as consisting in the presence of clots of blood in the right cavities of the heart, *redness* of the internal surface of those cavities, and *redness* of the pulmonary valves, and *redness* of the aorta. Many of the other cases adduced by him are instances of long-standing disease of the heart, such as hypertrophy and disease of the valves, and although in many cases induration and thickening of the valves are described, it is often difficult to ascertain, from the descriptions, whether the appearances were due to recent inflammation or to chronic lesion.[1] In some, however, the author describes very clearly the vegetations found on the cardiac valves, and in one case he represents them as being "exactly similar to certain pseudo-membranous granulations which are developed on serous membranes when they are chronically inflamed:"[2] but the identity of this case with one of endocarditis, as we now know that disease, is very much weakened by the facts that in this instance, also, there was phlebitis from vene-

[1] 'Traité Clinique des Maladies du Cœur.' Par J. Bouillaud. Tome Second.

[2] Op. cit., p. 78.

section, and the patient had been previously suffering from pleuro-pneumonia.

The presence of redness on the endocardium and on the surface of the valves, although much insisted upon by Bouillaud, is really not characteristic of acute inflammation, as I have already stated; and the presence of clots, of various kinds and dimensions, which he also very carefully describes as existing in the cavities of the heart, can hardly be allowed to proceed from inflammation.

These clots, which are sometimes composed of coagulated blood of ordinary character and sometimes consist of masses of fibrine, are evidently regarded by Bouillaud as of great diagnostic importance, as being the results of endocarditis, but they have in my opinion no such significance. They were described in some of the older treatises on Pathology as polypi of the heart, and were regarded as being of some importance by the physicians of a past generation, but they lost their interest in this respect when they were found to occur very frequently in post-mortem examinations where no particular symptoms of their existence had been previously detected. For my own part, having made a great number of post-mortem examinations, and under circumstances very favorable for comparing the appearances with the symptoms observed during life, I found these fibrinous clots of such common occurrence that at last I regarded them as of very little pathological moment and almost ceased to describe them in my published reports. When therefore Dr B. W. Richardson represented these fibrinous coagula as the *causes* of death in many cases, I was at first somewhat sceptical as to the correctness of this view of their nature. But Dr Richardson's cases and observations have led me to believe that, under certain circumstances, these coagula may probably be formed before death and become the cause of urgent and distressing symptoms, and this view is adopted, though with the necessary limitations, by some authors of eminence on Diseases of the Heart. In alleging the existence of these fibrinous clots, however, as *causes* of death, and not as concomitants or consequences of that event, it should in all cases be clearly proved that no other known lesion of a fatal character is present, for if there be valvular disease, or hypertrophy, or dilatation, or indeed, any other fatal malady, it would be idle to maintain that a very common post-mortem appearance can explain the symptoms and the result which are fully accounted for on other grounds.

The leading symptoms attending the separation of fibrine in the

heart, according to Dr Richardson, are falling temperature, pallid or livid surface, feeble, irregular or fluttering pulse, muscular prostration, and gasping respiration. He himself advises that all the usual causes of these symptoms should be excluded, in a given case, before the diagnosis of fibrinous clots in the heart is arrived at. With regard to the physical signs of this occurrence, Dr Richardson admits that he was at one time of opinion that few or none existed of a trustworthy character, but he has more recently corrected that impression, and he now thinks that not only can fibrine be detected within the heart, but that its actual position in regard to the cavities can be ascertained with great precision. The rules he lays down for forming a diagnosis are founded on his own actual experience, together with a reference to the place and the time of the heart's sounds. It being established that the tricuspid and mitral valves act together, and that the pulmonary and aortic valves also act together (see p. 26 et seq.), it is evident that if, in any given case, the action of the valves on one side of the heart is impeded, then the sounds produced on that sound will be reduced or even lost altogether. Thus if the fibrine be on the right side of the heart and interfere with the valvular movements, there will be deficient or feeble sounds over the tricuspid or the pulmonary valves, that is to say, on the right side of the sternum, and on the other hand, where the separation of fibrine is on the left side, these physical signs will be reversed.[1]

Thus it is only necessary to discover, in a given case, whether the sounds caused in the tricuspid and pulmonary valves, or in the mitral and aortic valves respectively, are deficient or absent, in order to define the exact position of a fibrinous clot within the heart.

Dr Richardson has since carried out, with his well-known energy and perseverance, the views on this subject first proposed by him; but, unless I am mistaken, some of his latest cases have been instances where the production of fibrinous clots within the heart have been due to wounds or injuries, which, from various causes, have given rise to phlebitis and pyæmia. If so, the production of these clots probably belongs to the same category as many of those related by Bouillaud (see p. 150), or perhaps they are due to the transference of *emboli* or plugs detached from inflamed surfaces, and then, although the diagnosis laid down by Dr Richardson may be sufficiently accurate, the fatal result which usually follows in such cases would rather be due

[1] Richardson on the "Separation of Fibrine from the Blood within the Circulation."—*Medical Times and Gazette*, 1868, vol. ii, p. 581.

to the circulation of poisonous matter in the vessels than to the production of fibrinous clots regarded as an idiopathic phenomenon. But the whole question is still under investigation.

The general signs of endocarditis are those of inflammation of the heart, but pain, which is by no means a constant or trustworthy symptom in pericarditis, is usually entirely absent in endocarditis, although Dr Hope states that he has sometimes noticed that a slight pain existed. The other general symptoms are not at all characteristic, and, if the circulation continue free, there is no embarrassment of the pulse or the respiration. The former is full, strong, and regular, and the other symptoms are those of ordinary inflammatory fever, and, as Dr Walshe says, rheumatic endocarditis may run a perfectly latent course in regard to local and general subjective symptoms.

The existence of endocarditis during life can be detected only by auscultation, which discovers a murmur or murmurs over the situation of the valves. But Niemeyer states that a blowing sound over the heart during an attack of acute rheumatism is not by any means a sufficient evidence of the existence of endocarditis. Such a sound, he says, may be heard in at least one half of all rheumatic attacks, and it depends in a great degree on the irregular tension of the valves to which the excited and uneven action of the heart gives rise.[1] While, according to Niemeyer, this blowing sound over the heart is so common in acute rheumatism, the frequency of the complication of acute articular rheumatism with endocarditis may, according to Bamberger, be estimated at about twenty per cent. Under the head of "Pathological Signification of Organic Murmurs" (p. 70), the relations existing between the murmurs and the special valves affected are fully pointed out, and it is therefore unnecessary to indicate in the present place the means of distinguishing one valvular disease from another. It is not easy to explain why certain valves are involved more than others in endocarditis, but the facts show that, except in fœtal life, those of the right side are seldom affected in this disease. Dr Walshe arranges the murmurs of purely acute endocarditis in the following order of frequency, the left side, it will be observed, being alone affected: aortic obstructive; mitral regurgitant; aortic obstructive and mitral regurgitant together; aortic obstructive and regurgitant together. Considering the conditions of inflammation on the cardiac valves, and the fact that any recent exudation of lymph

[1] Niemeyer's 'Text-Book of Practical Medicine,' vol. i, p. 330.

on their surfaces must be continually exposed to the current of the blood, I confess that it is not very easy to understand how such recent exudation can be localised by stethoscopic examination, and I suspect that the murmurs can really only be heard after the disease has lasted for some little time, and the exudation has become adherent on the valves, or the latter have become thickened or otherwise altered in their structure. Nor is it easy to explain how it happens that, while the murmur indicating obstruction of the aortic valves in endocarditis is very common, that indicating obstruction of the mitral valves is but rarely observed in this disease. The absence of this sound, or at least the difficulty of hearing it probably depends, as formerly stated (p. 73), upon some acoustic principle connected with the morbid sounds of the heart, which has hardly yet been fully developed; for it cannot be doubted that in endocarditis the mitral valves may be obstructed just as much as the aortic are. (This subject is more fully considered in treating of præsystolic murmurs.) It is also very probable that the situation of endocarditic murmurs may be changed in consequence of the exuded lymph being washed away from one valve to another, but there are no trustworthy observations to prove that such is the case, or that the locality of the murmur is altered from any such cause. When the valves have become thickened and hardened it is easy to understand how a murmur may then be produced by blood passing over them, and it is equally easy to explain how a stiffened condition of the valves or a defect in their construction may prevent their closure and thus permit of regurgitation; but it is not so easy to apply these facts and this reasoning to the case of endocarditis, which is an acute disease, and in which the products of inflammation are as yet unorganised. Dr Walshe, however, is probably correct in assuming that surface-roughness and lymph deposits are the main causes of the obstructive class of murmurs, while those of regurgitant character may be traced to intertwined lymph interfering with the play of the tendinous cords and the muscles of the heart, or to actual destruction of the substance of the valves, or even to a disordered dynamic condition of the apparatus concerned in closure.

I have before expressed a suspicion that the endocardial murmurs, notwithstanding the above ingenious explanation, may really only indicate the *results* of inflammation and not the inflammation itself, and it is therefore quite conceivable that, although endocarditis exists, the murmurs may be absent. This opinion is, indeed, entertained

by Dr Walshe, who justly observes that the conditions producing murmur are roughness of the valves, lymph among the tendinous cords, insufficiency of valves from puckering, and notable roughness, from the presence of lymph, of the ventricular surface; and as endocarditis may exist without any one of these pathological effects, the inference is unavoidable that the disease may be unattended with murmur. The same author goes on to state that he has seen distinct patchy redness with thin films of lymph on the ventricular endocardium where there had been recent excitement of the heart without newly developed murmur.[1]

3. CARDITIS OR MYOCARDITIS.

It might be supposed that as the pericardium covering the outside, and the endocardium lining the inside, of the heart are liable to inflammation, so the muscular substance of the heart itself would be liable to the same affection, and, accordingly, authors have usually described the inflammation of the heart itself under the head of carditis, or, from the muscular structure being involved, myocarditis. But on examining the accounts given of this disease, even by the best authorities, I confess that I am not quite convinced of the reality of its existence, and I have certainly never seen any case during life, nor examined any after death, in which I could convince myself that, independently of pericarditis and endocarditis, there was any evidence that the muscular substance of the heart was inflamed. Still, it is probable enough that when inflammation attacks both the outside and inside of the heart, its own muscular tissue does not escape. But inflammation can be demonstrated to the sight and the other senses only by its results, and as these results, such as effusion of lymph or serum, are observed only on the surface of the membranes, it is intelligible enough that the muscular organ itself, even although it has been diseased, may exhibit no visible traces of morbid action. The evidence from symptoms is still less to be depended upon, because, as it has been shown above, even pericarditis and endocarditis may run their course without giving rise to any subjective symptoms, and there are no objective indications observable, either rational or physical, which can determine the presence of carditis. I am aware

[1] Walshe on 'Diseases of the Heart,' 3rd edition, p. 254.

that pus has occasionally, though very rarely, been found in the substance of the heart, and that ulceration has sometimes, though still more rarely, been seen on its wall.

The above remarks on the subject of myocarditis really contain the essence of what is known of this affection. Even from the works of continental authors, especially German and French, who take an exhaustive view of such conditions as myocarditis, very little information of importance is to be gained. In Virchow's[1] "Pathologie und Therapie," for instance, a rather long chapter is devoted, in the volume on "Krankheiten des Herzens," by N. Friedreich, to diseases of the myocardium, but very little light is thrown upon the subject. The very able writer divides the disease into acute and chronic, but he admits that the first is a rare disease (*sie gehört zu der selteneren Krankheiten*), and that the second, although more common, cannot be detected during life by any known symptoms. In treating of acute myocarditis, he refers to the rare instances where abscesses have formed in the substance of the heart (*eiterige Myocarditis*) and to others, where the infiltration of albumen or fatty matters so alters the structure of the organ that perforations occur either into the pericardial sac or the interior of the ventricles, or they constitute cardiac aneurisms.

Other authors have attributed the supervention of hypertrophy of the heart to an inflammation of its muscular structure, and have considered that fatty degeneration is also due to the same cause; but it is quite impossible to admit the justice of these statements unless *all* changes whatever of a morbid character are to be regarded as inflammatory. It is, therefore, unnecessary in a practical treatise like the present to describe at greater length a disease which is admitted to be very rare, even if it exists at all; and of the symptoms and treatment of which we know little or nothing.

[1] Virchow's 'Handbuch der Pathologie und Therapie,' Vter Band, 2te Abtheilung, "Krankheiten des Herzens," bearbeitet, von N. Friedreich, p. 272.

CHAPTER XI.

ON THE CAUSES AND COMPLICATIONS OF CARDIAC INFLAMMATION

THE diseases just described are seldom or never idiopathic or primary, that is to say, they scarcely ever spring up spontaneously in a previously healthy constitution. It was once supposed, indeed, that inflammations of internal organs might burst forth suddenly in a healthy body, just as a fire may break out in a building, and it was also supposed, and in fact asserted, that as the fire-engines, with their supply of water, are able to extinguish a conflagration if they are employed sufficiently early, so the physician with his armamentarium of bleeding and purging is, or ought to be, able to arrest the progress and avert the consequences of inflammation. It really would appear that there was, in former periods at least, some justification for this kind of reasoning, and I need only appeal to those who have in former days seen an inflammation of the eye, for instance, visibly subside under the effect of bloodletting, to confirm the truth of the analogy.

But whatever may have been the case formerly, it is certain that no such simple explanation will now suffice to account for the origin of internal inflammations, or to suggest the means of counteracting or removing them. I am rather inclined to agree with Dr Wilks in his belief that, according to present experience, diseases are all, or almost all (with the exception of fevers and other epidemics) constitutional, and that when a local inflammation manifests itself, it does so only as a development, in a particular spot, of a general cachectic condition. The study of constitutional diseases has made rapid strides in the present day, and in proportion as those affections have been better understood and their symptoms and characters have become better defined, the real significance of local diseases has been more accurately appreciated, and their scientific treatment has more steadily advanced.

It is therefore, perhaps, incorrect to allege that one disease is *caused* by another, the more probable view being that when two or more diseases either accompany or follow one another, they are to be regarded not as linked together in the way of cause and effect, but rather as being perceptible, simultaneous, or consecutive manifestations of some constitutional, though perhaps hitherto latent, morbid condition.

If this reasoning is correct, as I believe it to be, in relation to the general pathogeny of internal local disease, it is especially applicable to the inflammatory diseases of the heart, which are all so obviously related to or connected with certain well-defined constitutional affections as to leave no room for doubt upon the subject. Putting out of consideration the very few cases in which inflammation of the pericardium may probably be caused by the infliction of some external injury, as by mechanical violence or by the accidents of warfare, the inflammations of the heart are almost universally due to the presence of morbid matter circulating in the system, which in some cases leaves the heart untouched, but in others fixes upon that organ as one of the *foci* of its manifestations. Thus the poison of rheumatism and the deranged condition of the fluids in the malady called "Bright's disease," are both of them, especially the former, fertile sources of cardiac inflammation, and there may be other similar constitutional affections accompanying, or followed by, the same local outbreak.

Bright's disease, an affection comprehending a great number of pathological conditions, is usually described as having its seat in the kidney, and there is no doubt that this organ exhibits, during the course of the affection, many structural changes which are very characteristic and are capable of being arranged in some kind of nosological classification. But I am inclined to believe that (setting aside acute nephritis) the granular, or fatty, or waxy degeneration of the kidney is not the primary link in the chain of pathological causation, and that these conditions of the organ are rather the consequences of some pre-existing disturbance of the system involving important changes in the constitution of the blood and the nutrition of the principal organs of the body. Just as Tubercular Consumption, which fixes upon the lung, has generally its origin in an hereditary or constitutional taint, and as a cancer, which may develope itself in the breast, is really due to some morbid matter circulating in the whole body, so I believe that the degenerated state of the kidney in

Bright's disease is due to that condition, not unaptly called "a breaking up of the system," which consists, in fact, of a decomposition of the blood.[1]

In what is called Bright's disease the symptoms are by no means confined to the urinary apparatus, but involve the whole system, and the malady is preceded by anomalous indications not pointing to the local affection of one particular organ or set of organs, but to general derangement of the constitution; and after death it is rare indeed to find that the kidneys are the organs exclusively implicated. In this disease, too, not only is albumen found in the urine, but urea, which ought to be excreted from the body, is retained in the blood, and the latter fluid loses its red corpuscles, which, in health, form its chief and most characteristic constituents, and are the great agents in the nutrition of the body. For these and many other reasons which it would be here somewhat out of place to adduce at length, I think that Bright's disease, or albuminuria, or the disease or diseases passing under these names, are in their nature really constitutional and not local diseases, and are developed by some morbid taint which, although difficult to detect, and impossible, with our present chemical and microscopical resources, to isolate or to analyse, is really the source of the organic derangements eventually recognised by the scalpel of the anatomist. In relation to the pathogeny of cardiac inflammation, it is certain that pericarditis and endocarditis frequently accompany or follow the morbid conditions grouped together under the name of Bright's disease.

But the disease which above all others precedes or accompanies cardiac inflammation is, without doubt, acute rheumatism, and this connexion has long been observed as an established pathological fact. It was first noticed by Dr Pitcairn in 1788, and the discovery was published by Baillie in his *Morbid Anatomy* in 1794, and was confirmed by Sir David Dundas in 1808. Since those periods the intimate relationship between acute rheumatism and inflammation of the heart has been recognised by all medical writers and teachers. It should also be mentioned that acute rheumatism itself sometimes follows or accompanies Bright's disease, but this is by no means a necessary or even frequent occurrence, for among the multitudinous cases of acute rheumatism which occur comparatively very few exhibit

[1] I am fully aware that the whole of this subject has been lately, and still is, under consideration by the most eminent pathologists of this and other countries.

the symptoms of Bright's disease; and *vice versâ*, among the numerous cases of Bright's disease, there are not very many which present the characters of acute rheumatism. Still the possible connexion of the two diseases should always be borne in mind, more especially in reference to prognosis; for a case in which Bright's disease is proved to exist must always be considered as a very serious one, while a case of rheumatism, however severe, and even when complicated with cardiac inflammation, but where Bright's disease is proved to be absent, may generally be regarded in a hopeful light.

Systematic writers on diseases of the heart sometimes enumerate, among the causes or coincidences of the inflammation of its membranes, many other affections besides the two first alluded to, and scurvy, cancer, tuberculosis, and other constitutional affections have been named as the efficient causes of pericarditis. But such statements are usually made only to be refuted by the writer himself, and do not demand any detailed notice. Phlebitis, or inflammation of the veins, especially when arising after wounds or surgical operations, has also been mentioned as a cause of cardiac inflammation, and many of Bouillaud's cases of endocarditis are described as being due to this cause; but I have already expressed my doubts whether, notwithstanding that physician's high authority, they were instances of endocarditis at all, at least, in the sense in which endocarditis is now understood. Again, pericarditis may and does sometimes arise from the proximity of some other disease, as pleurisy or pneumonia, but I think it far more likely that inflammation of the pericardium should extend to the pleura or the lungs than that inflammation of the latter structures should give rise to pericarditis.

But the connexion existing between acute rheumatism and cardiac inflammation admits of no doubt or question whatever, and the only difficulty lies in explaining the exact nature or cause of the relationship. It is so common, in the course of acute rheumatism, to observe the pain, swelling, heat, and redness suddenly remove from one joint to another, that the actual transference of the disease from its original seat to another spot has sometimes been imagined, and the malady is said, in vulgar language, *to fly* from one part to another. So again, in certain cases, where the pains in the joints disappear, and symptoms of internal disease are developed rather suddenly, it seemed to a bygone race of pathologists that a *metastasis* or change of seat had been effected, but this doctrine has been proved to be erroneous. For pericarditis and endocarditis often

coexist with the acute inflammation of the joints, and, on the other hand, rheumatic pericarditis may occur without any previous affection of the joints at all. The disease, in fact, *extends* from one fibro-serous tissue, such as that of the joints, to another fibro-serous tissue, such as that of the pericardium, and the difference of locality indicates no change in the essential characters or nature of the morbid action, which affects in common all the structures having the same anatomical relationship to one another.

Still, it is by no means unimportant to bear in mind, as a valuable axiom in pathology and as a guide in therapeutics, that the sudden disappearance of any external, eruptive, or inflammatory disease, and the supervention of internal complications, are very serious events and bear a manifest relationship to one another. Whether the doctrine of metastasis be true or false, it is far better that a gouty inflammation of the toe or finger should run its course than that it should suddenly cease, and be succeeded by cramps of the stomach or by congestion of the brain; and *vice versâ*, very serious internal complaints have disappeared, or at least have seemed to do so, on the supervention of a fit of the gout or the opening of a superficial ulcer. But in the case of acute rheumatism, and the attacks of pericarditis and endocarditis with which it is often associated, the diseases, as has just been mentioned, frequently proceed simultaneously, and they generally, and indeed usually, require almost identically the same treatment, making allowance, however, for the pre-eminent importance of the chief organ of the circulation, and the consequent necessity of preserving the integrity of its powers. But this subject will be more fully considered in the next section, which refers to the difficult and much-vexed subject of treatment, and in which that of acute rheumatism and that of cardiac inflammation will necessarily be included together.

CHAPTER XII

THE TREATMENT OF CARDIAC INFLAMMATION

THE treatment of pericarditis, endocarditis, and myocarditis is precisely the same, whatever may be their pathological distinctions, and as they are all connected with, if not most intimately allied to, acute rheumatism, it is necessary to offer some remarks upon the nature and treatment of the last-named affection before proceeding to consider the management of cardiac inflammation. If acute rheumatism can be effectually and safely arrested in its progress, the supervention of cardiac inflammation is no longer an object of apprehension, and even if the rheumatism should run its course, a judicious method of treatment may perhaps avert any cardiac complication and thus obviate a great source of danger. Rheumatism, in its various forms, although a most painful and distressing affection, can scarcely be described as one that by itself threatens life, and it is only when it is attended with complications, such as the inflammations of the heart, that its presence excites any serious alarm. But the supervention of heart-disease upon acute rheumatism is so common that it must always be watched for, and, if possible, averted, and one of the most important therapeutical indications in rheumatism is to accomplish that object.

There needs little argument to prove that acute rheumatism is not a local but a constitutional disease. Although sometimes excited into immediate activity by cold and wet, it owes its existence to neither of those agencies, and it may be developed in their absence or be absent when they are repeatedly present. It attacks both sexes and is very common in young children, and indeed appears to be rather a disease of youth than of age, the liability to its attacks decreasing as age advances. It is distinct from gout both in its nature and in the mode of its manifestations, but it is allied to gout, for it assumes the well-known and characteristic form known as

rheumatic gout, which blends together the features of both diseases. Rheumatism is properly denominated *a blood disease*, inasmuch as its essence seems to lie in some matter circulating in the blood, and it is also called a *diathetic* disease, from the Greek word διαθεσις, constitution.

Rheumatism is supposed, and with great probability, to derive its origin from some morbid material or poison in the system, but the exact nature of this poison is still a matter of question. As the secretions in this disease are all acid, the morbid material is presumed to be of an acid nature and, upon theoretical grounds, *lactic acid* is supposed to be the morbific agent. It would appear that this acid is a kind of intermediate product between the starchy matters of the food and their ultimate conversion in the system into carbonic acid and water, which are eliminated from the lungs by respiration; and hence the accumulation of lactic acid in the blood would constitute a kind of arrest of oxidation, and rheumatism might therefore be regarded as a disease of *sub-oxidation*; the remedies for rheumatism ought consequently to be such as are capable (if any such exist) of facilitating the oxidation, and thus destroying the poison. But this explanation, however ingenious, is not quite satisfactory, and the necessary connexion between lactic acid and rheumatism is by no means so clearly proved as that existing between uric acid and gout, for uric acid has actually been detected in the blood in gout, and moreover the oxidation of uric acid, and its conversion into carbonic acid and water, may be regarded as the best mode of curing or warding off that disease. Lactic acid, however, has not actually been detected in the blood in rheumatism, and its oxidation has never been shown to be a curative process, whatever may be the theoretical grounds for believing that it might be so.

Although, therefore, it is more than probable that acute rheumatism owes its origin to some morbid material in the system, it cannot be affirmed with certainty that lactic acid is the morbid material in question, for even admitting that this acid is present in patients suffering under acute rheumatism, it may be only an effect of the operation of the poison and not the poison itself. It is no more necessary to identify lactic acid with the rheumatic poison than it is to assume that albumen is the poisonous principle in Bright's disease. Albumen is eliminated by the urine in the last-named malady, and albumen is discharged in the rice-water evacuations of Asiatic cholera, and uric acid is discharged with the urine in gout, and

lithate and purpurate of ammonia are eliminated with the urine in many feverish and other affections, but it does not therefore follow that albumen, or uric acid, or lithate, or purpurate of ammonia is the very *materies morborum* of the respective maladies, of which those chemical compounds are in some measure the accompaniments or the exponents. The morbific poison in most diseases is probably of a very subtle nature, and incapable of being made manifest to the senses,[1] and I suspect that the poison of acute rheumatism forms no exception to the general rule.

The presence of *acid*, however, is a very conspicuous feature in acute rheumatism, abundant perspiration of an acid reaction and a disagreeable sour smell being a constant and characteristic symptom. This perspiration probably effects some beneficial purpose in the disease by carrying off the *materies morbi*, whatever the latter may be. The urine is scanty and of high specific gravity, and contains a large amount of urea and urates, but according to the careful observations of Dr Parkes, it is not particularly acid, the amount of uncombined uric acid being only slightly increased. That physician found that the sulphuric acid in the urine was increased during the height of acute rheumatism, but other analysts have failed to find any difference.

Rheumatism, in its acute form, attacks certain structures of the body, but seems to leave the others untouched. Its chief seats are the white fibrous tissues, such as those surrounding the joints; the serous membranes, such as the pericardium; and other lining membranes, as the endocardium and the inner coats of the arteries. It may attack most or all of the joints of the body, either successively, alternately, or simultaneously, and when confined to the limbs it is attended in a highly marked degree with the well-known symptoms of inflammation, namely, swelling, redness, heat, and pain. But although the inflammation of the joints is exceedingly severe, it very seldom happens, in a simple case of acute rheumatism, that any effusion takes place into the cavity of the articulations or that any thickening is left after the disease itself has disappeared. Where effusions do take place, there has been some serious complication, such as scarlatina, scrofula, or pyæmia. In fact, acute rheumatism, although entailing on the patient for a time the most intense suffering, may be considered, when we put aside the cardiac complications, as one of the most

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harmless of diseases, so far as its effects are concerned. It would seem as if the poison, which, whatever it may be, is developed within the system, assumes for a period the most extraordinary potency and deranges every function of the body, but as soon as it has run its course, it departs from the system with almost as much rapidity as that exhibited in its first development, and leaves behind it nothing but weakness. After an attack of acute rheumatism (always assuming that there has been no cardiac complication and no combination of other diseases), the patient becomes as well as he was before the attack, and there is absolutely no trace left of the existence of his most painful malady. But unfortunately an attack of acute rheumatism, unlike smallpox, measles, hooping-cough, and the like, does not secure to the sufferer any immunity from subsequent seizures, and, indeed, when acute rheumatism has once occurred, it is very likely to return.

Under most circumstances, acute rheumatism lasts for a considerable time, whatever may be the treatment employed. In this respect it would appear to resemble typhus and typhoid fevers, measles, scarlatina, and other maladies which have a definite duration and cannot be abbreviated in their course by any of the known resources of art. The average duration of acute rheumatism is from four to five weeks, but many writers on the disease, and especially those who wish to advocate some particular plan of treatment, affirm that this period may be very considerably reduced. Thus, Dr Hope states that by one or two full bleedings, and the nightly administration of pretty large doses of calomel and opium, followed in the morning by senna and salts, and accompanied during the day by moderate doses of colchicum, the patient is made well in a week[1]. I confess that I myself was under the impression, at one time, that acute rheumatism might thus be cut short, and in a paper which I published many years ago, the following case appeared in confirmation of the views I then entertained:

J. F—, æt. 19, a brickmaker, was seen by me on the 12th of April, 1842. He had been attacked with lassitude and heaviness, together with pains in various parts of his body. The joints especially affected were the right knee, which was excessively tender on pressure; the left hand and wrist, which were swollen and painful, the pain being increased upon the slightest touch; both elbow-joints, and both shoulders. The pulse was full and strong, 120 in the minute, considerable thirst, no appetite, bowels costive. I

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immediately bled him to sixteen ounces, which did not make him faint, and I ordered him to take immediately five grains of calomel with fifteen of jalap, and a draught every four hours consisting of half a drachm of colchicum wine, with sulphate and carbonate of magnesia and infusion of senna. The next day he was a little better; the pain had quitted the left hand and had fixed itself very severely in the right. The bowels had been opened, there was great thirst, the pulse was still full and strong. The blood drawn the day before was buffed and cupped, the buffy coat being very thick. I bled him again to *twenty-four ounces*, after which he felt faint, but did not actually faint. I ordered the draughts to be continued, and *ten* grains of calomel and one of opium to be taken immediately. The next day, April 14th, only two days after I first saw the patient, he was much better, was quite free from pain, and now felt only a little stiffness in the limbs, and he could bear pressure without inconvenience; the gums were not affected, and he had not slept well at night. The blood drawn the day before, and preserved purposely in two vessels, was buffed nd cupped in both. The draughts were continued. On the 16th of April, only four days after I first saw him, he was so much better as to be able to walk up and down stairs and in the street, without difficulty, and he entirely recovered soon afterwards.[1]

Now here was a striking case, honestly recorded, in which the rapidity of the cure was apparently attributable to the vigour of the treatment, and I have no doubt that some of the cases recorded by Dr Hope and others were equally successful, and in an equally short space of time, and under analogous modes of treatment. But when I published the case I fairly stated that I had attended many other cases of the same disease in which I had failed to obtain the same success, and I then attributed the failure to the fact that I had not bled sufficiently early or to a sufficient extent. But I may state now, with equal candour, that although I did bleed early and largely in many other cases I was disappointed in the results, and I suspect therefore that the cure recorded was an exceptional one. In the other instances I found that the symptoms continued in spite of the bleeding, but I cannot say they continued in consequence of it, for I have since seen numerous cases run on their usual tedious course where not a drop of blood was drawn, and where all the more modern precautions and appliances of medicine were carefully adopted.

[1] *Lancet*, 1842, p. 222.

As I have seen or read of nearly all the plans proposed for the treatment of acute rheumatism, and known them adopted and carried out with more or less success, I shall now glance over some of those plans *seriatim*, commenting upon each of them, and terminating the review with a statement of what I believe to be the safest and best method of managing the disease.

The plan of treatment most generally adopted during my earlier days of pupilage and practice, and that which I myself carried out, was pretty nearly that recommended by Dr Hope, and to which I have already alluded. It consisted in bleeding the patient in the first instance, and repeating that operation at intervals; in administering calomel and opium, in pretty full doses and at stated periods, together with saline purgatives; in giving colchicum in repeated doses, together with opium and salines; and in ordering low diet. Some of the practitioners of that period carried the bleeding to a greater extent than others, some pushed the calomel in larger or smaller doses, some trusted more than others did to copious evacuations by the bowels, and some relied on the specific powers of colchicum. Each part of this somewhat complex plan of treatment requires a little notice in detail, as each was founded upon certain definite therapeutical views, however fallacious these may now appear.

Bleeding was recommended and adopted with a view not only to diminish febrile action and excitement, but to reduce the amount of fibrine in the blood, the buffy coat being an invariable appearance when that fluid was drawn in acute rheumatism; and it was supposed that the withdrawal of the excess of the fibrine would necessarily be attended by a diminution of the symptoms. This view, however, was erroneous, and I was myself often struck by the circumstance that, under the influence of repeated bleedings, the fibrinous coat on the blood, instead of being diminished, was actually increased, and in most cases the bleeding did not in any way arrest the progress of the case or mitigate the symptoms. But even if the bleeding had diminished the fibrine in the blood, it does not follow that the disease would have been necessarily relieved, for fibrine is certainly not the *materies morbi* of rheumatism, although its excess is undoubtedly a feature of acute rheumatic inflammation. It would be equally absurd to expect to accomplish the cure of acute rheumatism by checking the perspiration or diminishing the amount of lithates excreted by the urine, as to think of achieving such an

end by diminishing the fibrine of the blood. Still, I cannot doubt that, putting all theoretical questions aside, a copious bleeding at the very onset of the affection in plethoric persons might and often did cut short the disease, and not only my own case related above, but those recorded in many most trustworthy treatises, and many others related to me by practitioners of known repute and veracity all corroborate this view, and seem to justify in some measure the practice of bloodletting. I cannot believe that a man with such a reputation as Dr Hope possessed, and with the large experience he enjoyed, would positively state as he does in his book on 'Diseases of the Heart,' that his patients often became well in a week under the use of bleeding and other depleting measures, if such had not been the truth.

The great mistake made, as I conceive it, was to suppose that a measure, because it succeeded in some cases, was applicable to all; and a still greater mistake was made in repeating the bleedings, after the first had failed, in the vain and fruitless hope of diminishing the inflammatory character of the blood. To a single bleeding at the onset of acute rheumatism I see no great objection even now, although I doubt its utility except in a very limited number of cases; but a repetition of bleeding is utterly useless, inasmuch as blood-letting does not in any way attack the essence of the disease, and it becomes positively injurious when repeated, because it then impoverishes the blood, increases the proportionate amount of fibrine, and by diminishing the vital powers retards convalescence. In addition to these reasons against reiterated bleedings, this course of treatment, by increasing the relative amount of fibrine in the blood, increases the liability to cardiac inflammation.

Purging by strong cathartics and salines was another measure supposed likely to be beneficial, not only by lowering the vital powers and thus promoting the subsidence of the inflammation, but also by diminishing the consistence of the blood and by carrying off from the system the *materies morbi* in the evacuations. But in practice, the only result of violent purging was to lower the vital powers without diminishing the force of the inflammation; and the same objection may be made to excessive purging as was made to repeated bleedings, namely, that the feebleness caused by such treatment very naturally retarded the convalescence, without making any impression upon the disease. According to present pathological views also, inflammation is very properly regarded as being in itself

a state of *depression* rather than one of exaltation of the vital powers, and strong purgatives must therefore only increase instead of alleviating the mischief. The purging no doubt drew away the watery parts of the blood, and by its weakening effects it induced a watery consistence in what was left, but it did not thereby relieve the circulating fluid of the poison it contained, nor was there ever any evidence to show that the cure of acute rheumatism was in any way accelerated in proportion to the profuseness of the evacuations. There is still another objection against purging in this disease, namely, that it necessitates constant movement on the part of the patient, thereby aggravating the pain and suffering under which he is already labouring.

Calomel has been very strongly recommended as a drug having almost specific powers in what is called sthenic inflammation, and especially in those forms of it, which, like acute rheumatism, are attended with the development of excess of fibrine in the blood. Calomel does in fact seem to possess the power of diminishing the consistency of fibrine, and there is considerable evidence to show, as was first pointed out by Mr Travers, that its administration is often attended by the absorption of lymph (or albumino-fibrine) which has been effused on the surface, or in the substance, of internal organs. Hence it was natural to conclude that, in the case of acute rheumatism, in which excess of fibrine in the blood is a most remarkable and characteristic feature, the use of calomel would dissolve the fibrine and thus cause its disappearance. It was also thought, as a consequence of the same train of reasoning, that the danger of effusion of fibrine on the surface of the heart, as in pericarditis, and on its lining membrane, as in endocarditis, would be averted by the timely use of the mercurial. But the answer to be made to any such reasonings and to the practice itself is, that, in point of fact, calomel produces no such effect in acute rheumatism as that which is expected. I myself never saw an instance in which the fibrine of the blood was diminished under the use of calomel given in acute rheumatism; and even if it did produce that effect, it would not cure the disease, inasmuch as fibrine is not itself the morbid material, but only exists in excess as a result of the action of the poison. It is also a curious circumstance, that notwithstanding the large amount of fibrine in the blood in acute rheumatism, there is but little tendency in this disease to cause fibrinous effusions in the joints, and therefore calomel can be of no service, for the result apprehended is

not likely to ensue. But it may be argued that the administration of calomel may prevent the development of pericarditis or endocarditis, or even of inflammation of the brain, during an attack of acute rheumatism, and the only answer again to be made to this suggestion is, that, whatever may be the theoretical arguments adduced, calomel seems to have no such power, inasmuch as those cardiac, or, it may be, cerebral complications very frequently supervene when calomel has been assiduously given, and are still more frequently absent when no mercurial at all has been administered.

It has been recommended to continue the use of calomel in acute rheumatism until the gums are affected, this result being the evidence that the mercury has thoroughly entered the system and has set up an action antagonistic to the disease. But in the first place, I may remark from my own observation, that it is very difficult to procure salivation in acute rheumatism; and in the second place, that if it were possible to attain this object by long-continued mercurial treatment, the convalescence would be retarded by the mercurial disease established in the system. Still, many eminent authorities believe that a few doses of calomel at the commencement of acute rheumatism may prove beneficial. If it be so, they act in the same way as the abstraction of blood, namely, by lowering the febrile action, and perhaps in some instances, by arresting the specific inflammation. But I am convinced that the continuance of the mercurial treatment is quite useless, if not injurious, and that it has no effect in neutralising the poison of acute rheumatism.

Opium, as an element of the above-mentioned complex treatment, was generally given in combination with calomel, with a view of preventing that drug from passing out of the system, and thus of securing its supposed specific action. It was also given in combination with colchicum and purgatives, to prevent griping, and it was often given alone, probably with the mere view of allaying pain. So far as its action of retaining mercury in the system is concerned, its administration is unnecessary, because mercury is now proved to have no effect in destroying the rheumatic poison; but, in other respects, opium, as I shall hereafter show, whether as tincture, or pill, or Dover's powder, or the salts of morphia, or in other forms, is a most valuable medicine in acute rheumatism, although I do not believe that it possesses any specific control over the disease.

Colchicum is a drug exercising a very well-marked action upon the system, being a sedative, a diuretic and a cathartic, and, in large

doses, an irritant poison. It is decidedly useful in gout, and has been supposed, on theoretical grounds, to have the power of eliminating from the system uric acid, which, whether it be the *materies morbi* of gout or not, certainly exists in the system in that disease. I have no hesitation whatever in bearing testimony to the efficacy of colchicum in the treatment of gout, although its indiscriminate employment in this disease is to be condemned. I believe that it is also very useful in some of the milder forms of rheumatism, though I do not profess to explain its *modus operandi*. But while I fully believe in the remedial powers of colchicum, cautiously employed and in combination with other medicinal and dietetic treatment, in cases of gout, I am equally convinced that it exercises very little, if any, curative influence over acute rheumatism. I have repeatedly seen gout subside under the use of colchicum, and in such a marked manner as to leave no reasonable doubt as to the beneficial influence of the remedy; but I have very seldom observed any beneficial effect from the use of the same medicine in acute rheumatism, although I have given it in repeated doses and with all the precautions recommended by the best authorities. Whether it eliminate uric acid or not, it is certain that it relieves a paroxysm of gout; but neither from theory nor from practice is there any evidence that it relieves acute rheumatism or shortens its duration. All that can be said of its use in the latter disease is that it does no harm, although of course, if it produced excessive purging, it might be mischievous, like other purgatives, by lowering the vital powers and thus preventing or retarding convalescence.

On the subject of *low diet*, which also formed a part of the treatment, there is not much to be said, and against it there are not many objections to urge. It must be generally conceded that there can be no good object gained by plying with food a patient who is suffering from high fever and loss of appetite, and who is moreover generating in his system, by perverted nutrition, a continual supply of poisonous material to feed his disease. Common sense, as well as the feelings of the patient, will therefore suggest that the diet should be generally spare, until the manufacture, so to speak, of the poison has ceased, and the function of nutrition begins to be carried on in a normal manner.

As it was found, then, that the system of polypharmacy was ineffectual, if not injurious, in the treatment of acute rheumatism, attempts were subsequently made to discover some simpler, but at the

same time efficient and, if possible, specific mode of treatment. The complex system, consisting of bleeding, starving, purging, mercurialisation, and the administration of colchicum, was gradually abandoned and was replaced by direct modes of treatment, having for their object the elimination of the rheumatic poison, and the consequent abbreviation of the term of the disease. It cannot, however, I think, be asserted that any specific has yet been discovered, although it must be admitted that the modern methods of treatment are far better than the old ones, and at any rate they have the merit of simplicity. Some drugs have been given empirically, while the administration of others has been justified on chemical or physiological grounds; some methods of treatment, although apparently resting on no scientific basis, seem to have been justified by the results obtained; and even the absence of treatment altogether, at least so far as drug-medication is concerned, has not been followed by dangerous results.

Nitrate of Potash in very large doses was recommended some years ago in the treatment of acute rheumatism by Dr Basham, who published several successful cases in confirmatio ' of the therapeutical value of this salt, but it does not appear that the favorable results he obtained have been confirmed by the observation of other physicians.

Lemon-Juice was introduced as a remedy for acute rheumatism by Dr Owen Rees, and the treatment by this very simple and rather agreeable fluid has been successfully adopted by many other practitioners. At the time of its introduction, I myself was so much dissatisfied with the then existing modes of treating acute rheumatism that I resolved to try the effects of so simple a remedy, and the results were certainly satisfactory. I had abandoned the idea of cutting short the disease by any heroic treatment, but by keeping the patients in bed, on low diet, giving them daily the juice of six lemons in the twenty-four hours, and without administering any other medicine, except an occasional dose of opium, I was surprised to find that the cases entirely recovered in a much shorter time than was occupied in the former methods of treatment. This plan I adopted not only in private practice but in a large infirmary, where a strict watch was kept that no fallacy should be introduced into the results, and that the treatment should be faithfully carried out. The patients recovered without any bad consequences, and suffered very little even from weakness, and the convalescence was always very rapid. The use of lemon-juice is not altogether empirical, for attempts have been

made to explain its *modus operandi* in acute rheumatism on theoretical grounds, though I confess that I am not nearly so much convinced of the soundness of the theory as I am of the success of the practice. It is assumed, as I have before stated, that excess of lactic acid is the essential cause of acute rheumatism, that this acid is, chemically speaking, an intermediate step between the starchy matters of the food and their ultimate conversion by oxidation into carbonic acid and water, and therefore that the conversion, as quickly as possible, of lactic acid into the two last-named compounds is the *desideratum* in the treatment of acute rheumatism. Now, *citric acid*, which is the chief chemical constituent of lemon juice, contains a considerable quantity of oxygen, and it is believed that it supplies this element to the lactic acid and thus converts it into carbonic acid and water. Perhaps it may be necessary here to remind readers who are not very deeply versed in Organic Chemistry, that both lactic acid and uric acid contain but a small proportionate amount of oxygen, and that the addition of oxygen to either of them converts it into carbonic acid, which although a weak acid, contains a large proportionate amount of oxygen, and is eliminated together with water from the lungs. Citric acid therefore, by containing a large proportion of oxygen, may become the medium of effecting the conversion of lactic acid into carbonic acid and water. The chemical formula for lactic acid is $C_3 H_5 O_3$; that for uric acid is $C_5 H_4 N_4 O_3$; and that for citric acid is $C_6 H_5 O_7$; showing that the last acid contains a much larger quantity of oxygen than the two others. But the fatal objection to this theoretical view of the action of citric acid is, that it is *lemon juice*, and not citric acid alone, which is the efficient drug in rheumatism, as it is in scurvy. Lemon-juice contains a small quantity of potash, but far too little to act in any way by neutralising the lactic acid. The lemon-juice treatment, however, has not maintained the reputation which it once acquired.

The application of *blisters* to the inflamed joints in acute rheumatism has been recommended and adopted by Dr Herbert Davies, and he has published several memoirs and papers showing the success of the practice. In one case in the London Hospital the patient was cured by this plan in seven days. Dr Davies claims for the blistering treatment the power of saving the heart from inflammatory mischief, of relieving the pains in the affected parts, of diminishing the rapidity of the pulse, of altering the reaction of the urine, and promoting rapid convalescence. He even states that the

pain is not nearly so severe as might be expected, and that the patients do not dislike the plan. The temperature of the body falls rapidly, with a free discharge from the close proximity of the inflamed joints, the pulse diminishes in frequency and force, and Dr Davies believes that the blood is relieved of the *materies morbi*. The effect of the blistering on the urine was remarkable in many of Dr Davies' cases, for although that fluid remained acid in some instances, in many others it became neutral, and in a considerable proportion it even became alkaline. Although this last result was obtained without the simultaneous employment of alkaline remedies, Dr Davies does not doubt that those agents might be advantageously combined with the blistering treatment.[1] So highly does Dr Davies think of this plan of treatment that he says, "Were it to fall to my lot to be attacked with acute rheumatism I would, without reference to any possible or probable amount of pain to be feared from the blisters, undergo any extent of the application to save my heart from becoming involved in the affection and rendered crippled for life."[2]

The application of *cold* to the affected joints has been frequently practised in acute rheumatism and has been attended with relief. The fears entertained by many practitioners that such a mode of treatment would have the effect of driving the inflammation to some other region, and thereby of increasing the danger of the case, do not seem to have been realised by the results of experience. But of late years, the bold expedient has been proposed and adopted of plunging the whole body in cold water in acute rheumatism, as in the case of other febrile diseases where the temperature is very high. This plan appears to be conceived on the supposition that the elevation of temperature belongs to the essence of the disease, and that its reduction is therefore a valuable means of cure. Be this as it may, the treatment has frequently been crowned with success. In this country, the bath in which the patient is immersed is gradually cooled, but a recent French writer, Dr Raynaud, of the Hôpital Lariboisière, in Paris, plunges the patient at once into water cooled with ice. This physician, while admitting the extreme simplicity of the principle on which the treatment is founded and the success which has frequently followed its adoption, has nevertheless seen cases where it has been attended with unfavorable results, and he

[1] 'Clinical Reports of the London Hospital,' 1864, vol. i, p. 293, and 1865, vol. ii, p. 138.

[2] Ib., 1865, p. 140.

therefore advises extreme care in resorting to this kind of medication. But he gives a remarkable case from his own experience, where a patient treated by the cold immersion was not only cured of acute rheumatism, but also of cerebral meningitis which supervened during the attack.[1] If such success be hereafter confirmed by the history of other cases, it would seem as if the cold immersion, so far from driving the malady to other parts, arrested its course altogether.

Many other remedies of a special character have been recommended and have been highly eulogised by those who have introduced them. The substances called *propylamine* and *trimethylamine*, two isomeric bodies, the latter of which is obtained from herring-brine, have been employed in Paris, and are said to have been very successful in the hands of Dr Dujardin-Beaumetz, of Paris.[2] These substances are limpid, colourless liquids, very volatile, and having some of the chemical characters of an ammonia (to which class they in fact belong) having a very disagreeable smell like that of decaying fish, and with a specific gravity somewhat less than water. Dr Dujardin-Beaumetz gives propylamine in the dose of a gramme (about fifteen grains), and he stated that its action is sometimes very promptly beneficial, sometimes in so short a space of time as twelve hours. In other cases, however, the cure was by no means so rapid.[3]

The *cyanides*, especially those of *zinc* and *potassium*, have been strongly recommended in acute rheumatism by Dr Luton, of Rheims, who prefers them to colchicum and to propylamine and trimethylamine.[4] These latter substances are very nauseous, and indeed this circumstance is one of the chief objections to their use, whereas the cyanides are not disagreeable, and the cyanide of zinc has neither taste nor smell, and may be given without the patient's knowledge. These salts are dissolved in the gastric juice, and their efficacy may probably be owing to the hydrocyanic acid which is developed when they enter the stomach. It is almost needless to observe that the cyanides are poisonous, especially the cyanide of potassium, and therefore must be used with caution. The cyanide

[1] "On the Employment of Cold Baths in Cerebral Rheumatism," by Dr Maurice Raynaud. 'Journal de Thérapeutique.' Paris, Nov. 25th, 1874.

[2] 'L'Union Médicale,' May, 1872.

[3] As these sheets are going through the press, Dr. Spencer, of Bristol, confirms the statement as to the efficacy of trimethylamine in rheumatism.

[4] 'Bulletin Général de Thérapeutique.' Paris, Jan. 15th, 1875.

of zinc is easily taken in pills or dissolved in mucilage, and the doses employed by Dr Luton varied from five, fifteen, or twenty centigrammes (about three quarters of a grain to three grains, English weight), the form of pill being preferred in the case of cyanide of potassium. The action of the heart and of the pulse is lowered by the action of the cyanides, and it is to this sedative action that their efficacy is to be attributed. It appears that Professor Brera, of Padua, had previously recommended the use of prussic acid in acute rheumatism and in several other inflammatory affections.

The *alkaline treatment*, especially that by potash and its carbonates, is unquestionably recommended to notice in the strongest manner on chemical and physiological grounds. If it be once admitted that the *materies morbi* in acute rheumatism is lactic acid, it ought to follow, almost as a matter of course, that the antidote and therefore the cure is to be found in potash or its carbonates, and it should be as easy to cure a case of acute rheumatism by the administration of potash as it is to neutralise a poisonous dose of oxalic acid by means of the administration of magnesia or lime. But, in the first place, it is by no means proved, as I have before remarked, that lactic acid *is* the *materies morbi*, although it is undoubtedly present in large quantity in acute rheumatism; and in the second place, experience does not demonstrate that the administration of alkalies invariably cuts short the disease. In the case I have related at p. 114 the disease lasted five weeks, accompanied with endocarditis, although the quantity of bicarbonate of potash administered amounted to *one pound and a quarter*, and that case is by no means an isolated one. Still, although it cannot, I think, be alleged that the potash treatment has the power of aborting or cutting short the disease, the evidence in its favour is very great, and this circumstance, together with its simplicity, its innocuity, and its chemical action, has made a great impression upon my own mind, and this feeling appears to be shared by many if not most of the practitioners who have written or thought much upon the subject. One of the most remarkable contributions to the statistics of the alkaline treatment in acute rheumatism has been made by Dr Dickinson in the forty-fifth volume of the 'Medico-Chirurgical Transactions,'[1] wherein it is shown that out of forty-eight patients, subjected to the full alkaline treatment only one had pericarditis, commencing after the treatment was begun.

[1] 'Medico-Chirurgical Transactions for 1862,' p. 343.

It has been shown on very satisfactory evidence that acute rheumatism will disappear and often leave no bad consequences under the administration of *mint-water*, or in other words, without any drug-medication at all, and without doing anything beyond keeping the patients in bed and watching the symptoms. This plan has been followed out pretty largely at Guy's Hospital, and the results are recorded in the Reports of the medical and surgical practice of that establishment.[1] But without at present offering any dogmatic opinion on the merits or demerits of this plan, or comparing it with the alkaline treatment, I must express my opinion that even on the evidence afforded by the results of the cases themselves, heart-disease seems to have supervened and to have proved fatal in a considerable proportion. It is alleged, in the comparison of large numbers of patients treated on the alkaline system at St. George's Hospital, with others treated on the mint-water or do-nothing system at Guy's, that the former exhibit a greater proportionate immunity from heart-disease than the latter, but as the comparative experiments are still being pursued, the question cannot even yet be pronounced as decided.

I now proceed briefly to describe the treatment which I adopt myself and which I believe to be the safest and the best. It aims at relieving as far as we can the sufferings of the patient, preventing, if possible, the supervention of heart-disease, checking, as far as is practicable, the progress of the specific affection, and preserving the vital powers so as to obviate exhaustion when convalescence begins. Excessive depletion, or, indeed, depletion at all, forms no part of the plan; calomel does not enter into the category of drugs employed; colchicum I regard as entirely useless, and purging I believe to be injurious.

Bicarbonate of potash in pretty large and often-repeated doses, as a drachm every four hours, is the remedy in which I think most reliance ought to be placed, and I accordingly always recommend it. Even allowing that it does not act as a specific, it at any rate neutralises the extreme acidity of the secretions and very probably corrects the acid properties of the blood. It must also be said that if it does no positive good, it certainly does no harm, and I have never seen or heard of any bad consequence which could in any way have been attributed to its employment. But as I have also a favorable opinion of the use of lemon juice, although I am not

[1] 'Guy's Hospital Reports,' 1865 and 1866

quite sure as to its *modus operandi*, I administer that fluid also, and often order it in combination with the bicarbonate. The effervescence is agreeable to the patient's feelings, and the resulting compound (the citrate of potash) has neither the nauseous taste of the bicarbonate, nor the sour taste of the lemon juice, while on the other hand it preserves the chemical character of both, so far as the therapeutical effects are concerned. The citrate of potash thus formed is again converted in the system into the carbonate of potash, and thus all the beneficial influence of this salt is preserved and its taste only is disguised. If the lemon juice has really any specific influence, that advantage may be secured by giving it at intervals without the bicarbonate, and, to render it agreeable, it may be given with a little sugar. I should give the juice of at least six lemons a day, but the patient should have more if he can take it, and he generally rather likes the juice than otherwise. No harm can result even from supersaturating the system with the potash, and there is no fear of giving an overdose of lemon juice.

Opium in some of its forms, namely, the tincture, or Battley's sedative solution, or Dover's powder, or the acetate or hydrochlorate of morphia, is, I think, a very useful drug in the treatment of acute rheumatism. I do not attribute to it any specific power over the disease but, as acute rheumatism is always attended with pain, sometimes of a most agonising character, the administration of opium procures a certain measure of relief and is always well borne by the patient. There may be danger in giving too much of this valuable drug in the treatment of any disease, but it is to be observed that in acute rheumatism pretty large doses are required. One to two grains every day or twice a day, or even oftener, or equivalent doses of the tincture or of the morphia salts, are attended only by relief of the pain, and are never followed, so far as I am aware, by any bad consequences. But if there be any valid objection to the use of opium, then *henbane*, in the form of the extract or the tincture, may be given at frequent intervals in the dose of five grains of the former, or half a drachm or a drachm of the latter; but, on the whole, henbane is far inferior in efficacy to opium.

The question of *diet* is a very important one. When I first began to treat the disease, and indeed ever since, I have regarded low diet as an essential feature in its management. Considering that one principal feature in acute rheumatism is the mal-assimilation of the elements of the food, and that, whatever may be the true

chemical theory, these elements are converted into an excess of acid, I have always thought that the withdrawal of food was one very effectual method of diminishing the amount of acid in the system. It appeared to me that food administered in acute rheumatism only served the purpose of literally "feeding the disease," for, during the continuance of the morbid condition, the nutrition is so perverted that the formation of healthy and normal tissues is interrupted and an enormous secretion of acid is the only, or at least the chief work of the organs which elaborate the fluids of the body. I have therefore always recommended low diet, for instance, milk and water or weak beef-tea, and have never observed any bad consequences from the adoption of this plan, for as soon as convalescence begins, then the secretion of excessive acid is discontinued and the food passes into the proper channels for the nutrition of the system.

I do not know that the moderate allowance of certain alcoholic drinks is particularly objectionable in acute rheumatism, and if it be true that alcohol is not properly a food, but that it arrests the waste of the tissues, then I think there is an additional argument for its employment in acute rheumatism. In this disease food is not wanted, and in my opinion its administration does more harm than good, but an agent which will preserve the body from wasting while the rheumatic poison is being eliminated from the system appears to me to be a great desideratum, and this is furnished, I think, by alcohol. But of course great care must be taken in the selection of the drink, for it need scarcely be said that such fluids as port and sherry wines, from their containing a large amount of sugar and other nutritious matters, or beer, ale, and porter, from their being also rich in this respect, are quite objectionable. The administration of a little gin, which may be given with the lemon juice, seems to me not only harmless, but likely to be beneficial, for not only may the alcohol it contains prevent the waste of the tissues, but the juniper, which is one of its principal constituents, will act upon the kidneys and help to carry off the *materies morbi*. Pure brandy or pure whisky are also unobjectionable, although they want the diuretic properties of gin, and I see no objection to the use of some light claret or hock if the patient likes these drinks. He should of course be allowed as much water or toast-and-water, or weak tea, as he pleases to take; and cream of tartar water, and Seltzer water, but especially potash water and lithia water, are very suitable to the case from the amount of alkali they contain, and they may be

given either alone or in combination with some claret or hock, or with a little gin, brandy, or whisky.

The above plan I believe to be the best adapted to relieve the sufferings of the patient, to guard against excessive weakness in convalescence, to prevent the waste of the tissues, and to obviate the supervention of cardiac inflammation; but if the last-named complication should unfortunately make its appearance, then the treatment must in many respects be modified, for although the inflammation is essentially of the same nature, its results are very different, and the organ attacked is one of the most important, if not the most important, in the body. In acute rheumatism, notwithstanding the violence of the inflammatory symptoms and the abundance of fibrine formed in the blood, the danger of plastic exudation into the inflamed parts is very small, and in most cases of acute rheumatism the normal condition of the joints, including of course their complete mobility, is restored. But the case is far different in pericarditis and endocarditis, in both of which plastic or other exudations are the necessary consequences of the inflammation, and lead to permanent injury of the structures concerned.

Hence it becomes a matter of primary importance in the treatment of pericarditis and endocarditis, not only to allay the urgent symptoms and to arrest the progress of the inflammation, but to obviate the supervention of those exudations of lymph or other matters which embarrass the action of the heart or injure the efficiency of the valves. But although it is the aim of all treatment to prevent the adhesion of the pericardium or to obviate dropsy of that sac, in pericarditis, and to prevent permanent valvular disease in endocarditis, I fear that these objects are very seldom completely accomplished, and that there always remains some mischief, to a greater or less extent, either within or without the heart as a consequence of the attack. The least dangerous of these forms of mischief is the adhesion of the pericardium to the heart, and I believe that this condition very often escapes detection during life, in consequence of the small comparative inconvenience to which it gives rise. It is of course conceivable that inflammation of the pericardium or endocardium may terminate in resolution, or in other words, in an entire cure, leaving the affected parts in as healthy a state as before, but it is exceedingly difficult to prove such a result, because, for obvious reasons, the fatal cases alone admit of demonstrative evidence.

Although, therefore, it may be generally admitted that uncompli-

cated acute rheumatism may disappear in time under any rational mode of treatment, and that in all probability it will leave no unfavorable consequences, the case is far different when the structures of the heart are involved, for then there is undoubted danger to life both from the attack itself and from the after consequences which it entails. Hence practitioners have always regarded cardiac inflammation as a very serious complication, and many eminent authorities have considered the prognosis as necessarily unfavorable. From the time of Corvisart, who seems to have thought all serious diseases of the heart to be incurable, and who affixed to his book the motto from Virgil, *Hæret lateri lethalis arundo,* to that of Sir Thomas Watson, who while admitting that in a certain proportion of cases of pericarditis the patients seem to recover, fears that very few, if any, really do so, inflammation of the membranes of the heart has always been regarded with apprehension.

Nevertheless, I think I am borne out by general experience in affirming that, under the present methods of treatment, the mortality from this disease has very much diminished, and in fact the *post-mortem* examinations in acute inflammation of the heart, both at the hospitals and in private practice, have become the exceptions instead of the rule. Not only is acute rheumatism less frequently attended than formerly by cardiac inflammation, in consequence, as I believe, of the more judicious treatment of the former disease, but the inflammations of the heart themselves have been more successfully treated, and their danger, both immediate and remote, has been very greatly diminished. When I see, as I have often occasion to do, persons whom I have attended for the most serious cardiac inflammation, transacting, at the present hour, all the usual business of life and suffering no inconvenience, I am unable to endorse the gloomy motto of Corvisart, and cannot avoid taking a far more hopeful view of such cases in general than many practitioners entertain, or than I myself entertained in former years. The errors long committed in the treatment of cardiac inflammation consisted in attempting to do too much, in leaving too little to the reparative powers of nature, in trusting too implicitly to the efficacy of depletion and the specific action of drugs, and in neglecting to support the heart itself in its struggle with disease.

Even those who had ceased to bleed in acute rheumatism were afraid to omit that measure in some form or other when the membranes of the heart became inflamed; and in the present day Oppolzer, who may be supposed to represent the most recent views

of the German school of practical medicine, advocates, in his lectures on the *Krankheiten des Herzens*, the propriety of venesection in certain cases, in pericarditis, but recommends that it should be employed with caution. He advises its adoption in combination with the use of quinine, more especially when, together with weakness of the heart's contractions, there are indications of congestion in the brain and lungs. He thinks, too, that bleeding is especially indicated in pericarditis, when the fever is high, and the patient is strong and young, and otherwise healthy; and when he complains of violent pains in the breast and head, or of tightness of the chest, or of difficulty of breathing; and bleeding, he considers, is also advisable when there is hyperæmia of the brain or lungs, but not when there is irregularity of the respiration, or alternate diminution and irregularity of the pulse.

While Oppolzer may be considered an authority on this point in Germany, Dr Austin Flint gives the results of his treatment, and those of his countrymen, in the United States. The latter writer, while alluding to the practice of Bouillaud and Hope in the employment of blood-letting, and to that of Stokes and Todd, in abstaining from it, adopts a middle course in reference to this powerful agent. He thinks that its indiscriminate use in cardiac, as in other inflammations, cannot but be productive of great harm, whereas, when judiciously employed, it may in certain cases do good. A person in fair health and vigour attacked with acute pericarditis may be a proper subject for bloodletting at the onset of the disease. Resorted to under these circumstances, Dr Flint considers that it will not cut short the disease, nor perhaps abridge its duration, but it may contribute to diminish the intensity of the inflammation, and thus besides affording immediate relief may lessen the prospective dangers of the disease. These views were propounded by Dr Flint, in the first edition of his work on 'Diseases of the Heart,' and they are essentially the same as those he has advanced in the second, which has recently appeared; but, he adds, that during the ten years which have elapsed he has had no experience as regards either the good or the bad results of bleeding in pericarditis, for in none of the cases he has observed during this period has this measure been employed.

Now the question is, what good can be expected from general bleeding in this disease? If bloodletting be adopted in order to diminish the amount of fibrine in the blood, and thus to obviate

the danger of plastic exudation, it is quite certain that it produces no such effect, and, under the influence of repeated bleedings, the relative amount of fibrine rather increases than diminishes. Again, as the great danger to be feared in pericarditis is the supervention of paralysis of the heart, it is obvious that general bleeding is rather calculated to facilitate than to avert such a result, and therefore the measure is likely to be mischievous as well as useless. The only manner in which I believe that bloodletting may be admissible in cardiac inflammation is in the application of a few leeches over the præcordial region at the onset of the disease. Such a step can do no harm, and as it removes a little blood from the immediate vicinity of the inflammation, it may relieve the general inflammatory condition of the parts and do some positive good to the local affection. In spite of the arguments adduced by high authority against the practice of bloodletting altogether, I still believe that measure beneficial in certain cases, and more especially in local inflammations, in which a cautious amount of depletion by means of leeches or cupping is followed in numberless instances by appreciable relief. I think the indiscriminate rejection of bloodletting is almost as much to be deprecated as its injudicious employment, and I am convinced that a moderate amount of bleeding at the onset of many inflammatory and congestive diseases has often been attended with marked benefit. While therefore I altogether condemn general bloodletting in the treatment of cardiac inflammation, I consider the application of a few leeches over the upper part of the præcordial region at the onset of the disease a very justifiable and beneficial proceeding.

Mercury, especially in the form of calomel, formerly regarded as a valuable remedial agent in acute rheumatism, and perhaps still holding its ground in that respect in the opinions of some practitioners, was still more highly esteemed than bleeding for its supposed specific power of subduing the inflammatory affections of the pericardium and endocardium. Even those, therefore, who placed no faith in calomel in cutting short acute rheumatism, thought it imperative to give that drug when cardiac inflammation had commenced, and the theoretical grounds on which it was administered were satisfactory enough. Mercury exerts a solvent action on the blood, and appears to have the power of diminishing the relative amount of fibrine in that fluid, and it was therefore believed it would prevent the effusion of lymph, or albumino-fibrine, in or upon the cardiac mem-

branes. So, too, from the visible effect of mercury in the cure of iritis, it was believed that the mineral had the power of causing the absorption of fibrine after it had been effused, and therefore that it would cause the absorption of the inflammatory products thrown out on the pericardium and endocardium. Unfortunately, however, practice does not confirm these theoretical views, and it is found not only that it is very difficult in pericardial or endocardial inflammation of rheumatic orgin to bring the system under the influence of mercury, but also that under all or any circumstances of mercurial treatment the effusion may take place. Calomel, therefore, must be considered as having no specific influence in controlling the progress of cardiac inflammation, and its use is now far less recommended than formerly in that affection, more especially since it has been shown that pericarditis may come on during salivation.

Colchicum I believe to be practically useless in acute rheumatism, and therefore regard it as equally unavailing in cardiac inflammations of rheumatic origin. *Digitalis*, which is a most useful medicine in certain affections of the heart, I consider to be of very little service in pericarditis and endocarditis, and in large doses it would seem even to be contra-indicated, for its tendency is said to be to produce syncope, which is one of the dangers to be apprehended in cardiac inflammation.

In discussing the general question of treatment in pericarditis and endocarditis, it should be borne in mind that the therapeutical management must be regulated according to the causes or complications of the cardiac inflammation, and that although in most cases acute rheumatism is the original disease, yet that it is not invariably so. Bright's disease, as I have already shown, is sometimes the precursor or the attendant both of rheumatism and of cardiac inflammation, and as it is a constitutional affection of a very serious nature, its detection will greatly complicate the leading features of the case and materially affect the chances of recovery. Although, however, Bright's disease, or the assemblage of morbid conditions which often passes under that name, is a not uncommon cause of cardiac inflammation, I do not discuss at length the treatment to be adopted in that affection, because there is by no means such a direct relation between it and cardiac disease as there is between cardiac disease and rheumatism. Bright's disease, too, especially in its chronic form, is a far more serious malady than rheumatism, and, unlike the latter, it kills the patient in the great majority of cases. When cardiac inflam-

mation or pericardial effusion follows Bright's disease it is usually only one result out of a series of morbid consecutive changes induced by the blood-poisoning of the system. Difficult as it is to demonstrate the *materies morbi* of acute rheumatism, that of Bright's disease is still more mysterious in its nature; and while the former, whatever it may be, is often eliminated from the system, the latter is seldom or ever so removed.

Idiopathic pericarditis or endocarditis I believe to be exceedingly rare; and traumatic pericarditis, if it should occur, must of course be treated without reference to any constitutional taint.

In the treatment of pericarditis and endocarditis I think the principal objects to be held in view are to give rest to the inflamed organ, to facilitate, as much as possible, the absorption of the inflammatory products, to tranquillise the general excitement of the system, and to support the heart in its struggles to throw off the disease.

In the first place, then, it is absolutely necessary that the patient should be kept perfectly at rest, and the maintenance of this state of repose is really an essential part of the treatment. Although inflammation of the heart is a dangerous disease, it nevertheless exhausts itself within a certain period, and it is well if the physician does not, by over-anxiety in the application of his therapeutical measures, increase the mischief he wishes to overcome. I am much struck with the force of Mr. Hilton's observation in his work on 'Rest and Pain,' that even the effusion in the pericardium, in pericarditis, may serve a beneficial purpose in providing a soft cushion for the repose of the inflamed heart, for experience shows that even adhesion of the pericardium is the least dangerous of the consequences of pericarditis.[1]

As the administration of alkalies appears on the whole to be the best therapeutical measure in the treatment of acute rheumatism, these substances, especially potash and its carbonates, should be administered in cardiac inflammation of rheumatic origin. They may be given in various ways, either dissolved in water, or barley-water, or contained in various mineral or artificial drinks, as Seltzer-water, Seidlitz powders, Rochelle salt; or as contained in cream-of-tartar water, or tamarind-water, or the juice of grapes, currants, gooseberries, &c. In the latter category the potash is combined with various organic acids, as the tartaric, the citric, the malic, &c., but all these acids are converted into carbonates in the system, and they are agreeable to the patient's feelings, they neutralise the acid in the

[1] 'Lectures on Rest and Pain,' by John Hilton, F.R.S., p. 355.

blood, and, what is also a point of great importance, all the potash salts are more or less diuretic, and assist in carrying off the *materies morbi* by the kidneys. But where Bright's disease exists, the use of diuretics must be as far as possible avoided, because these agents unduly irritate the kidneys which are already in a morbid state, and they thereby increase the disease.

The best method, in my opinion, to be adopted for the purpose of facilitating the absorption of the inflammatory products is the application of blisters to the præcordial region. These agents can never do any harm, and they often are productive of very beneficial results. I think that there can be no question as to the remedial effect of what was called "revulsion" by old medical writers, who signified by that term the plucking or turning a disease away from some internal organ to an external and less important part. Without entering into any hypothetical explanations as to the nature or essence of disease in general, it is a matter of common experience that a superficial irritation or inflammation will often cause the disappearance of an internal affection of a similar character, and, *vice versâ*, the sudden disappearance of a cutaneous affection is very often, and indeed generally, followed by more or less serious internal mischief. There is no principle in pathology better understood or more generally admitted, and, in therapeutical ministrations, practitioners have gladly availed themselves of its application. Hence the common and often successful practice of applying mustard poultices and other rubefacients in cases where there is internal pain or uneasiness, whatever may be its nature, and hence the use of blisters in internal inflammations, congestions, and effusions. In the case of pericarditis and endocarditis, the application of blisters over the præcordial region sets up an inflammatory action on the skin which is antagonistic to the inflammatory action going on within; and it is far better that the skin should be temporarily inflamed and some serum discharged externally than that the inflammation and effusion should occur in a vital organ.

When the blisters have been removed, warm fomentations of poppy-heads, or poultices of bran or linseed meal may be applied over the chest, and these applications are always agreeable to the patient's feelings, and contribute to the repose of the inflamed internal structures. I purposely make but little allusion to the relief of pain afforded by such applications, because in cardiac inflammation pain is by no means a prominent symptom, and indeed is very

frequently absent, and if pain is excited by the blisters, the indication is favorable rather than otherwise, for it shows a reaction on the part of the external surface and usually precedes a general improvement in the internal disease. In the treatment of serious internal affections I have far more frequently had occasion to regret the absence of cutaneous pain and irritation than to feel anxious about their presence.

The use of *opium* I believe to be of the greatest value in the treatment of cardiac inflammation. I attribute no specific virtue to this drug, but inasmuch as it tranquillises the whole system, it gives repose to the inflamed heart, and co-operates with the restorative powers of nature in moderating the effects of the inflammation, or, at least, in conducting the process to a safe termination. I am at a loss to understand why some writers disparage or condemn the use of this valuable drug in pericarditis and endocarditis, for it is unquestionably the most important internal agent we possess for relieving the restlessness which is a characteristic feature of these affections, and it is, moreover, extremely well borne by the patient. It is a very curious and noteworthy circumstance, that in this kind of inflammation opium and its different preparations appear to lose the properties they possess when given to persons in health. Instead of causing stupor, as large doses would ordinarily do, their only effect is to procure sleep; instead of causing delirium, they mitigate that symptom; and they even lose the power, which is usually so well-marked a sign of their operation, of contracting the pupil. It would seem, in fact, that opiates given in cardiac inflammation expend their powers upon the disease, and it is not until this has abated that they begin to exercise their ordinary physiological or toxic effects. Hence, very considerable and indeed extraordinary doses of opiates may be safely and usefully given in cardiac inflammation, and, as far as I know, they never cause any subsequent inconvenience.

Opium may be given in the form of the tincture, or, what is better, in that of Battley's sedative solution, in the dose of twenty minims every four hours, or the acetate or hydrochlorate of morphia may be given at the same intervals in doses of a third to half a grain in the form of the officinal solutions. I may here remark parenthetically that the solution of morphia of the present *British Pharmacopœia* (1867) contains only half as much morphia as the corresponding solutions of the former London Pharmacopœia, and

they must accordingly be given in larger doses. One fluid drachm of the solution, either of hydrochlorate or acetate of morphia of the present Pharmacopœia, corresponds to half a grain of the salt, and this is about the dose which should be administered in cardiac inflammation.

In discussing the treatment of acute rheumatism I stated that the allowance of a moderate amount of alcoholic fluid was by no means objectionable, especially in the form of gin, whisky, or brandy. These fluids, while they do not afford any nutrition to the system, in all probability prevent the waste of the tissues, and support the constitution till the disease has passed away. But while the use of alcoholic fluid is allowable in acute rheumatism, I think that it is imperatively required in cardiac inflammation. I believe that to Dr Stokes belongs the merit of having pointed out the propriety of administering stimulants in such a case, and he did so at a time when such a suggestion was at variance with the precepts and the practice of the medical profession in general. The great danger to be apprehended in this disease, as I have already pointed out, is paralysis of the heart, probably from the extension of the inflammation from the membranes to the muscular substance, and it is therefore absolutely necessary that the organ should be supported, as far as possible, against so fatal a catastrophe. Now there are no agents so effectual as alcoholic fluids in arousing the energies of the nervous system, and consequently in sustaining and stimulating the flagging powers of the heart, when its muscular fibres are embarrassed in their action by the presence of inflammation. All the arguments formerly employed against the use of alcohol in such affections have been proved to be worthless. The rapid pulse, instead of being further accelerated by the employment of this agent, is actually reduced in velocity under its use, and instead of alcohol causing delirium, there is no better method known of diminishing or dissipating delirium in cardiac disease than by its administration. Like opium, alcohol acts in a totally different manner in disease and in health; in the former its beneficial effects are alone apparent, and its intoxicating properties disappear. Besides the special action of alcohol in sustaining the flagging energies of the heart, this agent accomplishes the further object of preventing the waste of the tissues generally, and thus of sustaining the system until the inflammation has run its course. For the reasons already mentioned while treating of the therapeutics of acute rheumatism, I

should not recommend either beer or any sweet or full-bodied wine in cardiac inflammation, but brandy, whisky, or gin may be given in such quantities as the nature of the disease may seem to require, the rapidity of the pulse and the existence or persistence of delirium being circumstances rather indicating the necessity of increasing the dose than warning the practitioner to diminish it.

It only remains to add a few words on the general subject of diet. In the case of acute rheumatism I consider that the diet should be low, because a great part of the nutritious matter taken into the system in that disease is changed by the perverted action of nutrition, into a poisonous agent, and not into the healthy structures of the body. But in the case of cardiac inflammation there is a double danger to guard against, namely, the undue accumulation of lactic acid in the system (when the disease is of rheumatic origin) and the depression of the heart's action, and the latter danger is perhaps the greater of the two. It is therefore necessary to support the powers of the system and of the heart at all hazards, and there is no fear that the amount of fibrinous or serous effusion in or upon the heart will be increased by a supply of nutritious diet. The products of cardiac inflammation are not lactic acid or uric acid, but lymph and serum, and perhaps, as a secondary product, pus, and these may be regarded as results of deficient vital action, and therefore as indicating the necessity of supporting the system. Milk, therefore, strong beef-tea, soups, broths, jellies, should all be administered, and the more the patient takes the more probable is it that he will pass safely through the crisis of the malady.

The following case is an interesting instance of a very severe form of pericarditis, treated successfully, having its origin, no doubt, in a rheumatic diathesis, but not preceded, or accompanied, or followed by the ordinary characters of rheumatic fever. It would appear that the rheumatic poison circulating in the system concentrated itself almost in the first instance upon the heart, thus rendering the premonitory symptoms very obscure and the disease itself doubly dangerous.

Mr. ——, a gentleman, aged 32, tall, and of robust appearance, of an active and energetic disposition, engaged in an extensive mercantile business and accustomed to generous living, but not amounting to excess, became affected about the end of June with

several anomalous symptoms. He was occasionally feverish, restless at night, disinclined to food, but rather thirsty, and his mind was somewhat affected at intervals, especially at night. These symptoms, however, assumed a kind of intermittent character, for on some days he could go to town and transact his ordinary business, while at others he would remain at home and lie in bed the greater part of the day. He was perfectly rational when spoken to by his medical attendants, and the wandering showed itself chiefly to some members of his own family.

June 24.—I was attending a member of his family at a little distance in the country, and I came to town with him; he was then quite rational and apparently in good health, and I left him in the west-end of London. The next day when I went to visit the other patient, he was in bed, and I examined him carefully in conjunction with his ordinary medical attendant. We could find nothing definite in his symptoms, which somewhat resembled those of incipient typhoid fever, a disease then prevailing, not only in the neighbourhood, but in the house itself; but I felt sure that he was not labouring under this malady, because I had attended him some years before in a well-marked attack of this very fever, and I thought it highly improbable that he would take it a second time. There were no symptoms of typhus, the pulse was rather rapid, the head was warm but not hot, the skin was moist, there was no eruption, there was some thirst, and he was quite rational. We, therefore, recommended rest, and a little saline medicine, and a moderate amount of supporting diet. The next day I saw him incidentally in the evening, as I was seeing the other patient. He was then in much the same condition; and when I saw him again he was in bed, but quite tranquil, and apparently getting better; and I left him under the care of his usual medical attendant. I did not see him again until July 5th, when I received a telegraphic message from his wife to go down and visit him at Broadstairs. This message was sent to me without his knowledge, and was caused by his restless and excited manner, want of sleep, and frequent incoherence of mind, but there were not any other remarkable symptoms. On visiting him I found him quite tranquil, but rather low-spirited; he was sitting up, and was quite able to walk about. As he did not admit that he was ill, I had some little difficulty in making a careful medical examination, but I found that his head was not hot, the skin was cool and moist, and the pupils acted normally; the pulse was rather quick, but soft.

All I could ascertain was that he had had very restless nights, with a total want of sleep and great incoherence. It should be mentioned that, since I saw him at home, he had been actively engaged in various pursuits, and had indeed been making rather violent efforts to "shake off" the complaint, and had come down to Broadstairs by himself, not only without any advice, but rather in opposition to it. As his condition was unsatisfactory and the symptoms were anomalous, I determined not to leave him for the night, and as he was labouring under evident excitement and irritability of the nervous system, I ordered him to take half a drachm of Battley's sedative solution at bedtime, and the dose to be repeated if necessary, requesting that I might be called during the night in case of his being restless. I was called accordingly, and found that the opiate had produced no effect at all, and I therefore repeated the dose, and recommended more to be given afterwards. After taking about two drachms of it no effect was produced, and the night was passed, I believe, absolutely without sleep. I now changed the Battley for the hydrochlorate of morphia, of which I gave, in solution, a quantity amounting to half a grain, but still no sedative effect was produced. I remained with the patient the greater part of the day of July 6th, and gave him additional doses of the hydrochlorate, one third of a grain at a time, but still no benefit ensued. I was obliged to return to London, but directed that the morphia should be continued, and a local practitioner called in. The next day I received a note from the patient, perfectly well written, but of a somewhat peculiar character, and seeming to express some annoyance at my having been sent for to visit him, and he made no allusion to his being ill. For the next few days I heard nothing of him, except that I was informed, in conversation with some of his relatives, that a local practitioner had been called in, and that "gout" had developed itself in one of his feet. I was rejoiced to hear this statement, as I now thought that a clue was given to the nature of the affection. It should be mentioned that the patient had never suffered from rheumatic fever.

July 10.—I was summoned by telegraphic message to go down immediately to Broadstairs, as the patient was much worse. I accordingly went down and met Mr Walter of that town, who showed extreme care and ceaseless attention throughout the progress of the case. My first inquiry on seeing the patient was, whether there was any pain or swelling of the feet, and I found a slight

redness on the left great toe and some tenderness on pressure; but these symptoms were quite transient, and an hour or two afterwards I could not detect them. But in other respects the patient was very dangerously ill. He was lying prostrate in bed, breathing rapidly and laboriously, with a quick, rather small and intermittent pulse, head moist, pupils acting normally. On examining the region of the heart, I found that the præcordial dulness was increased, particularly upwards, while the impulse and sounds of the heart were quite imperceptible. It was now evident that there was pericardial inflammation with effusion into the sac of the pericardium. I, therefore, in consultation with Mr Walter, directed a large blister to be laid over the præcordial region, and at the same time strong blistering fluid was applied to the feet and toes. The solution of morphia (which had been continued at frequent intervals ever since I first ordered it) was directed to be still given, and brandy was administered in the dose of a tablespoonful every two or three hours, together with strong beef-tea and milk. The objects aimed at in this treatment were—first, to support the strength of the system and to stimulate the flagging action of the heart; secondly, to divert the effusion of serum or lymph from the surface of the heart; thirdly, to produce a kind of artificial rheumatism in the extremities; and, fourthly, to tranquillise the nervous system while the process of repair was going on in the pericardium. I considered that depletory measures were unadvisable, and that no benefit could be expected from mercury in any form.

The above treatment did not produce any immediately beneficial effects; but the blisters answered the purpose of causing rather extensive vesication over the chest and over the feet and toes, though without much pain. Nothing like rheumatism or gout was developed, either then or afterwards; but after a day or two the action of the heart became perceptible, both to the touch and the stethoscope, although its impulse was still weak, the sounds were feeble, and there was no murmur. The patient was quite delirious, but not violently so, his thoughts wandering chiefly about matters of business; his head was always cool and moist to the touch; the pupils were quite regular, and acted on the stimulus of light; the bowels were moderately open; the urine was passed freely; there was abundance of acid perspiration. It should be mentioned that the urine was frequently examined, but it contained no sugar or albumen, nor did it appear thick or high coloured.

Thus matters went on for some days, the breathing being rapid and laborious, 40 to 50 in a minute, the pulse rapid and intermittent, so that it could hardly be counted though it appeared to be about 160, and the unconsciousness and delirium continued. There was, indeed, very little favorable about the case, except that the patient continued to live and to take support and stimulants, which were given very freely, and the acetate of morphia was regularly administered in the dose of a third of a grain to half a grain every three hours. Notwithstanding this enormous quantity of morphia, the pupils were unaffected, and very little genuine sleep was procured.

It was now thought advisable that another London opinion should be requested in consultation, owing to the extreme gravity of the symptoms, and accordingly I went down in company with a distinguished physician on the 19th of July. On relating to him while on the journey the features of the case, he informed me that he had seen two cases, and only two, as far as I understood him, of a similar kind. We found the patient nearly in the same state as that I have just described, but the inflammation had now spread to both lungs, and there was pleuro-pneumonia on both sides; the action of the heart was still weak, but it was perceptible, and there was a distinct but not very loud friction-sound. The opinion of this gentleman both as to the nature of the disease and the treatment to be pursued coincided entirely with my own, and no change whatever was recommended. Brandy was to be regularly given in the dose of a tablespoonful every two hours, or more if necessary; milk and strong beef tea, or jelly, or turtle-soup, were to be taken as freely as possible; and the morphia was to be continued in the same doses as before until the pupils were affected.

Still no great change was observed, and it became obvious that the case would run on for the same length of time as one of ordinary rheumatic fever, if the patient survived the heart-affection, and this turned out to be the fact, for when I saw him on the 5th of August, which was about five weeks from the commencement of the attack, he was beginning to get a little better, and was becoming rational. Still the pulse was very rapid, weak, and intermitting, the breath very quick and laborious, and there was extensive dulness all over the posterior part of the lungs, with friction-sound and tubular breathing; the action of the heart was now distinctly perceptible, with occasional friction-sound, but I could never detect any endocardial murmur. There was profuse perspiration and a miliary eruption over

the skin. A rather alarming symptom presented itself about this time —namely, swelling, tenderness, and redness along the course of the right basilic vein, and I was under some apprehension that thrombosis or some other obstruction might develope itself; but by assiduous fomentation of the inflamed vein, the pain and tenderness gradually subsided, and gave no further trouble. As the general symptoms were now relieved, the quantity of brandy was reduced, but the use of the morphia was still continued, though in somewhat smaller doses, and at rather more distant intervals. I last saw the patient at Broadstairs on the 14th of August, when he was very much better, was quite rational, and was able to eat his meals as usual. He was still suffering, however, from shortness of breath, the pulse was very rapid, and he was very weak but not much reduced in size.

I saw nothing more of him until the 6th of September, when I was asked to visit him in the vicinity of London. He was now able to walk, although he was still weak, and his breath was short on making any exertion. The pulse was weak, rapid, and intermittent, and the breathing rapid; there was some dulness on the back of the right lung, but I could detect no murmur or friction-sound over the heart. After that time the patient went to Scotland, where he began sailing, riding, and walking, and then he came to London, and pursued his business as usual.

Remarks.—The above case was one of rheumatic pericarditis, in which the affection was altogether, or almost altogether, confined to the investing membrane of the heart; for I cannot recognise the slight and transient inflammation of one toe as more than an indication, though an important one, of the true nature of the disease. The early symptoms were rather those of a cerebral than of a cardiac nature, but it is well known that in pericarditis the intellect is very often impaired, and this latter circumstance is, indeed, sometimes almost diagnostic of pericardial inflammation. (See p. 141.) The treatment throughout, it will be observed, was of a soothing and sustaining kind, no depletion having at any time been recommended. The amount of stimulants administered was very large, twelve ounces of brandy having been given daily in the twenty-four hours regularly, for at least three weeks, and a smaller quantity before and after that period. The amount of morphia given was also very great, one third of a grain to half a grain having been administered at frequent intervals during the whole of the illness. What may perhaps appear astonishing to those who are unacquainted with the

results of such treatment is, that no injurious effect whatever was produced, either by the alcoholic liquids or by the morphia; the delirium diminished in proportion as the alcohol was increased, and the morphia produced no constipation of the bowels, and did not even contract the pupils.

CHAPTER XIII

CHRONIC PERICARDITIS

THE most favorable result of acute pericarditis is, of course, resolution, or the return of all the affected structures to their normal condition. But this, I believe, seldom occurs, and the acute disease most generally merges into a chronic state. "Post hæc indicia," says Celsus, after describing the leading and dangerous symptoms of acute disease in general, "votum est" (or *notum est*, according to another reading) "longum morbum fieri; sic enim necesse est, nisi occidit. Neque vitæ alia spes in magnis malis est, quam ut impetum morbi trahendo aliquis effugiat, porrigaturque in id tempus, quod curationi locum præstet."[1] This doctrine, containing a great amount of truth in itself, is very applicable to pericarditis, for if the acute malady remained, the heart would be continually liable to derangement in its action; and the chronic stage, or that in which the disease is protracted (*trahendo morbum*) affords repose to the organ, and in fact protects it from a fresh acute attack. The great desideratum is that the result of the chronic stage should be such as to procure this repose for the heart. Now there are three modes of termination of acute pericarditis, namely, resolution, which I believe is very rare, if it ever take place; adhesion of the pericardium; and effusion of fluid, or dropsy; and the last two only are to be considered in a practical point of view.

Adhesion of the Pericardium very frequently and indeed most commonly results from an attack of acute pericarditis, but the importance of this result has been exaggerated, and it is at least doubtful whether it entails much suffering on the patient or is productive of much consecutive morbid action. The heart being pretty

[1] 'Celsus de Medicinâ,' Book II, Chap. 5.

closely enveloped by the pericardium, the movements of both are nearly equal, that is to say, they both move together, and consequently there is not much difference when they are agglutinated to one another. Still, it must be recollected that there is a certain though small amount of movement of the heart *within* the pericardium and independent of it, and there is always a small but appreciable amount of fluid present in the sac. The pericardium is fixed to the diaphragm below, to the lungs on each side, and to the sternum in front, and is therefore in itself immoveable, while the heart is continually in motion. Hence, when the heart is bound to the pericardium by strong adhesions, it follows that the former must drag the latter with it in all its pulsations, and the pericardium must also draw with it the diaphragm, the lungs, and the sternum. Such, in fact, are the consequences of adherent pericardium, and the visible signs of that condition are to be sought in the retraction of the anterior part of the chest-walls, especially at the lower part of the sternum. There are no auscultatory signs of any importance to denote the adhesion, and, with the most careful inspection and examination, it must not be forgotten that, on the one hand, the adhesion may be overlooked, and, on the other, that that condition may be supposed to exist when in fact it is absent.

Hydropericardium or Hydrops Pericardii are the names given to denote the presence of an abnormal amount of fluid within the pericardial sac, but the condition so designated can scarcely ever, perhaps never, be considered as an idiopathic form of dropsy. It occurs, however, under three circumstances; first, in the form of effusion of serous fluid mixed with flakes of lymph in acute pericarditis, as in all probability existed in the case detailed at p. 188; secondly, as a condition concomitant with the presence of dropsical effusions in other parts and cavities of the body; and, thirdly, as a passive exudation occurring from mere weakness of the walls of the vessels a short time before death. The last condition requires no special notice, and the first is included under the symptoms and results of acute pericarditis.

Hydropericardium, however, sometimes acquires a special significance when it accompanies general dropsy, for while the latter condition, by itself, may be attended with little suffering, yet when fluid accumulates in the pericardial sac, the heart is embarrassed in its action, and difficulty of breathing, tendency to fainting, feebleness and irregularity of the pulse, and death will supervene; and

thus pericardial dropsy requires energetic special treatment in addition to the adoption of general therapeutical measures. I am inclined to believe that Bright's disease is the most usual cause of pericardial dropsy, and that rheumatism rather leads to the acute disease, or pericarditis, characterised by the effusion of lymph and followed by adhesion. The abuse of spirituous liquors is also a fertile cause of pericardial dropsy, though only in connexion with dropsical effusions in other parts. Bright's disease itself, indeed, is often caused by the vice alluded to, but this is by no means invariably or usually the case; and alcoholic poisoning claims an independent place for itself as a pathological condition.

It would be out of place here to discuss the physiological question as to the effects of alcohol on the human organism, but it may be observed that the prevailing opinion is probably correct, namely, that its consumption in moderate quantity not only serves as a useful, if not necessary stimulant, but that it moreover prevents the waste of the tissues. If, however, moderation be exceeded, it acts as a poison and induces a series of important and dangerous changes in the vital organs and tissues. By being continually carried from the stomach through the portal vessels to the liver, it unduly irritates the latter organ, causing a kind of hypertrophy of its fibrous tissue which eventually compresses the nutrient vessels and the excretory ducts, thus embarrassing the biliary secretion and throwing the bile back upon the general system. On the kidneys an analogous change is effected, and by the same kind of irritation a species of hypertrophy of their fibrous elements (called both in the liver and the kidneys *cirrhosis*) is set up, the result being to choke up the uriniferous tubules, to impede the excretion of urea, and thus to poison the whole system. It would also appear that, from the chemical composition of alcohol (which is mainly composed of carbon and hydrogen) the undue indulgence in alcoholic liquors tends to the development of oil or fat (also a hydrocarbon) in the tissues of the arteries, and thus extensive disease of their coats, to which reference has been made as a result of old age at p. 128, is prematurely developed. Hence the whole arterial system, and indeed the whole of the capillary system as well, are enfeebled and embarrassed in their action; they are unable to take up the water in the general system and discharge it through the kidneys, and, from the diseased condition of the vascular coats, the water oozes through into the surrounding tissues. Such I believe to be the mechanism of

alcoholic dropsy, and such are the conditions, which give rise, *inter alia*, to hydro-pericardium.

Dropsy of the pericardium, then, although occurring in connexion with general dropsy, and probably never as an idiopathic affection, may exist to such an extent as to become a prominent morbid indication, and most seriously to threaten life. In such cases the fluid pushes downwards the central tendon of the diaphragm, and there is increased dulness on percussion, but the pyriform outline traced in acute pericarditis is absent in the chronic affection. Dr Sibson, who gives some beautiful representations of both the healthy and diseased conditions of the pericardium in his 'Medical Anatomy' (published in one volume in 1869), states that more than three pints of fluid have been found in the sac.

The remedy for this condition consists in repeated blistering, and when this measure fails, paracentesis of the pericardium has been recommended, and practised with a certain amount of success. Dr Sibson strongly recommends it in all those cases where the affection is so great as to cause alarming distress, orthopnœa, obstructions to the venous circulation, and interference with the heart's action. In such cases he would employ the fine exploring trocar and cannula, plunging the instrument in below the heart, either to the left of the xiphoid cartilage or through the fifth intercostal space, close to its anterior extremity. The fluid can be drawn off through the finest cannula by means of a syringe. Dr Bellingham,[1] however, who gives a brief but lucid history of the operation, is not aware of any instance in which paracentesis of the pericardium has been practised in Ireland, and he states that few cases of its successful issue are on record.[2] Trousseau has recorded a successful case of the kind, the operation having been performed by Jobert, and M. Aran, of Paris, has not only successfully performed paracentesis of the pericardium, but has also injected a solution of iodine into the sac.

[1] 'British and Foreign Medico-Chirurgical Review,' July, 1854. Review of Bellingham and Stokes on Diseases of the Heart, by Dr Sibson.

[2] Bellingham on Diseases of the Heart, p. 330.

CHAPTER XIV

HYPERTROPHY AND DILATATION OF THE HEART

Disease of the walls of the heart is very often attributable to morbid affections of the valves, and there is a growing disposition on the part of pathologists to regard hypertrophy and dilatation, whether separate or combined, as being caused almost exclusively by valvular lesions either obstructive or regurgitant. Indeed hypertrophy is now generally considered as a compensatory condition in valvular disease, and, on this view, is not a disease at all; while dilatation, on the contrary, being caused by the overflow of blood into the cavities of a heart with weakened walls, incapable of resisting the strain put upon them, is a condition full of danger. Dilatation itself, however, is compensated by hypertrophy, and hence the very common combination of dilatation with hypertrophy may be regarded without too much apprehension, as it is a compensatory effort of nature to overcome the difficulties of the circulation caused by valvular stenosis or by insufficiency. Nevertheless, the diseases of the heart-walls ought not to be too exclusively regarded as dependent on valvular disease, inasmuch as they may arise without antecedent valvular affections, and may, in fact, be the causes and not the effects of valvular lesions. The dilatation of the right ventricle, for instance, may, by the mere stretching and expansion of the walls, give rise to tricuspid regurgitation, the curtains of the valve being separated from each other by the widening of the aperture.

Dr Quain has very recently called particular attention to the diseases of the muscular walls of the heart in his Lumleian Lectures, delivered at the College of Physicians, and has specified the exact changes which the muscular walls undergo in hypertrophy. He has shown that enlargement of the heart may depend on an increase of its muscular fibres, or of its connective tissue, or of its fat,

which last condition is not, however, to be confounded with fatty degeneration. He has also called or rather recalled attention to the fact that hypertrophy may arise independently of valvular disease, as for instance, from athletic sports carried to excess, or even from emotions of the mind, or from the wear and tear caused by the excitement of business. From reference to the statistics of the Registrar-General, Dr Quain shows that heart-disease, among men, has greatly increased, while in women it has remained almost stationary.

Hypertrophy of the heart consists in a thickening of the walls of the organ, and dilatation in an enlargement of its cavities. These conditions do not appear theoretically to be necessarily connected with one another, and the mind can easily conceive them to be perfectly distinct; and yet in practice they are so constantly found combined, in a greater or less degree, that it is convenient, even if it be not absolutely necessary, to treat of them together. When systematic writers describe these conditions separately, they are necessarily betrayed either into repetition or indistinctness, because, when treating of hypertrophy in its various degrees, they are compelled to include at least some forms of dilatation, and *vice versâ*, or when they attempt to delineate each condition separately they are often induced to draw a picture of an affection which has little real existence. Simple hypertrophy, for instance, would represent a disease where the walls of the heart are thickened, and the cavities retain their natural dimensions, and in practice this condition sometimes exists; and in simple dilatation the walls of the heart would be of their natural thickness, while the cavities would be enlarged in capacity, a combination of conditions equally conceivable in theory but practically and really amounting to hypertrophy with dilatation. Although, therefore, it is very easy to give what may be called a book-definition of hypertrophy and dilatation respectively, according to the etymological signification of the words, yet it is a matter of some difficulty to convey to the mind an accurate idea of the changes in the dimensions or structure of the heart which are implied by pathologists when they use the terms in question. "By hypertrophy of the heart," says Oppolzer, one of the latest practical writers on cardiac disease, "is meant an increase of the substance of the heart, when its walls are abnormally thick, or when, by the enlargement of its cavities, they exhibit only their normal thickness; and this state is distinguished from passive dilatation in which the heart, or

a portion of it, exhibits an increased circumference with a diminished thickness of its walls."[1] But in this definition it is plain that in one of the instances of hypertrophy the writer includes hypertrophy with dilatation, and in the case of dilatation there is the additional morbid change of thinness of the cardiac walls.

It is difficult, therefore, to consider either hypertrophy or dilatation of the heart as simple conditions of disease respectively, and what is known as enlargement of the heart usually includes the double morbid state of hypertrophy with dilatation. Still it is important to recollect that either condition may predominate in a given case, and hence the necessity of noting the characters peculiar to each, in order that the signification of the symptoms may be accurately understood.

HYPERTROPHY

It has been already stated (p. 122) in reference to hypertrophy, that there is so much variation in the size and thi kness of the heart within the limits of health that it is not always easy to determine whether a heart is hypertrophied or not, and certainly not in the slighter forms of this affection. Oppolzer divides hypertrophy into *true* and *false*, the latter consisting of an increase in the bulk of the heart from the introduction of foreign elements into its structure, such as fat, lardaceous matter, and the products of inflammation. Practically, however, this false hypertrophy may be disregarded, and then hypertrophy will represent that condition of the heart in which the increase is caused by an excessive development of the muscular fibres. Conflicting opinions have long been entertained as to the question whether hypertrophy is due to an increase in number of the muscular fibres, or only to their enlargement, and the aid of the microscope has been invoked on both sides of the question. "The microscopical characters of the individual muscular fibre in hypertrophy," writes Dr Walshe, "do not deviate from those of health; and as the primitive fasciculi do not increase in thickness new ones are obviously formed."[2] But Oppolzer, on the contrary, who writes more recently, declares that the increase in bulk is now proved to be due to the enlargement of the fibres, and he claims for

[1] 'Oppolzer's Vorlesungen,' "Die Krankheiten des Herzens," &c., Erlangen, 1866, p. 111.

[2] Walshe on 'Diseases of the Heart,' p. 279, 1862.

one of his countrymen, Hopp, the merit of having established the fact, and of having actually demonstrated the hypertrophied fibres.[1]

A distinction has been made, but I think most erroneously, between what was called *concentric* and *eccentric* hypertrophy. In the former case, although the heart appears of its natural size, or even perhaps smaller, the ventricular cavities, especially the left, are diminished in capacity, while the muscular walls are apparently increased in thickness. But I have seen this condition so frequently in post-mortem examinations of persons who never suffered from any disease of the heart, that I have long regarded it as of little or no pathological importance, and as being due only to that emptying of the left ventricle which occurs shortly before death, and to the gradual but powerful contraction of the muscular walls, from the *rigor mortis*, which gives the appearance of undue thickness, though no such condition has existed during life.

The term *eccentric hypertrophy*, however, is still retained. In this form of disease the cavities are enlarged, while the walls are either of their normal size or are increased in thickness. In the first case, the eccentric hypertrophy would correspond to the condition known as *simple dilatation*, and in the second, namely, where the walls are thickened and the cavities are enlarged, the case is one of *active dilatation*, or, in other words, of *hypertrophy with dilatation*. This last-named combination, which thus corresponds to active dilatation and to eccentric hypertrophy, occurs very frequently, and it constitutes the most ordinary form of enlargement of the heart, and is usually designated, for the sake of brevity, as hypertrophy.

Hypertrophy of the heart is generally an accompaniment or a consequence of some other pathological process either in the heart itself or in some physiologically allied organs. Thus it is a very general complication of valvular disease of the heart or of some chronic affection of the lungs. Still, I agree with Dr Quain in thinking there are very many cases where no such complication exists, and where the hypertrophy is due to causes originating in the system at large, such as excessive exercise of the muscular system, as in rowing, and other athletic employments; and sometimes I have found hypertrophy in cases where not even such causes could be assigned for its presence. Men are much more liable than women to hypertrophy, the proportion being

[1] Oppolzer, op. cit.

about two to one, and this circumstance is in all probability owing to the fact that men are more subject than women to the influence of the causes which excite the disease.

Hypertrophy may be *general,* when it affects the whole of the heart, or it may be limited to one portion of the organ. The left ventricle is by far the most frequently affected, and according to Dr Walshe the left auricle comes next in order of frequency, closely followed by the right ventricle, while the right auricle is seldom affected.[1]

The thickness of the wall of the left ventricle is increased in this disease from half an inch, which is its normal measurement, to an inch and a half or two inches, and the septum of the ventricles is likewise thickened so as to encroach on the cavity of the right ventricle. The hypertrophied heart assumes the form of an obtuse-angled triangle, and its weight is so much increased that it may weigh one to two pounds, while in the natural state it weighs only from eight to ten ounces. Its volume may be doubled or trebled, and its increase takes place in the long diameter, the heart lying more horizontally on the diaphragm, the base being considerably to the right of the sternum, while the apex lies several inches to the left beyond its normal position. In such cases the left ventricle is chiefly affected, but if the right ventricle should be hypertrophied, which is a rare occurrence, the increase takes place in the transverse diameter. In both cases the hypertrophy is usually associated with dilatation, and as the latter condition predominates, the outline of the heart becomes more and more altered from a triangular to a rounded form, one of the distinguishing characters in a dilated heart being the rounding off of its apex.

The Symptoms of Hypertrophy.—Hypertrophy of the heart, as has just been stated, is very often associated with, and indeed caused by, diseases of the cardiac valves, and it is somewhat difficult to point out the symptoms which exclusively denote each of these two morbid conditions. Still, I am convinced that one of these conditions may exist independently of the other, and I have lately carefully and frequently examined cases in which, on the one hand, there was marked evidence of hypertrophy without valvular disease, and on the other hand, though perhaps not so frequently, there was valvular disease without hypertrophy. In many cases, where acute rheumatism had

[1] Walshe on Diseases of the Heart, p. 277.

undoubtedly occurred, I have found evidence of hypertrophy, but no valvular mischief, and it appears to me probable that in such cases the pericardium or the subjacent structure of the heart may have been affected, and the endocardium may have escaped; or it is probable that the rheumatic affection may have attacked the muscular structure alone, though this last view is incapable of demonstration. On the other hand, where the valves only are affected, it may perhaps be assumed that the endocardium alone has been the subject of the action of the rheumatic poison in the cases where the rheumatic diathesis has been the cause of disease. When the hypertrophy is very considerable, the præcordial region is sometimes unduly prominent, especially on the left side, and even the whole of the left side of the chest may be enlarged. The *impulse* of the heart is increased, and the extent of surface over which it is felt is increased likewise, and this latter character is one of the most important signs in distinguishing hypertrophy from mere excitement of the heart. The impulse is so much increased in hypertrophy that the fingers laid flat over the part corresponding to the apex of the heart are sensibly raised, the stethoscope laid over the same part is also raised, and the head of the observer, if placed upon the ear-piece of the stethoscope, may be raised likewise. The impulse may also be *seen*, the apex beating between the sixth and seventh, or even between the seventh and eighth rib. Percussion indicates a greater or less increase of præcordial dulness in proportion to the extent of the hypertrophy, although this sign may sometimes be masked by the overlapping of the heart by an emphysematous lung. The dulness may reach from the second to the eighth rib longitudinally, and transversely from an inch and a half or two inches on the right of the sternum to three inches or more outside the vertical line of the nipple. It will be remembered that the natural dulness of the præcordial region does not extend more than two inches in any direction.

The *sounds* of the heart in hypertrophy are usually dull and muffled so as to be sometimes nearly inaudible, but as hypertrophy is almost always accompanied with dilatation, it is found practically that in most cases the sounds undergo no very great change. In a case of hypertrophy with dilatation, the impulse is increased while the sounds may be somewhat diminished in intensity, but in proportion as dilatation keeps pace with, or preponderates over, hypertrophy, the impulse somewhat diminishes and the intensity of the sounds increases. I think it may be generally asserted that no

murmurs are heard in a case of hypertrophy unless there exist at the same time some disease of the valves, but as hypertrophy is most frequently caused by disease of the valves, the murmurs are very generally heard, and might be, and indeed have been attributed to hypertrophy alone. It was at one time supposed that murmurs might be caused by hypertrophy or by spasmodic contraction of the great vessels of the heart, and even Dr Walshe states that a systolic blowing murmur is sometimes heard in cases of pure hypertrophy even where there is no coexistent spanæmia, but, notwithstanding such great authority, I am inclined to adopt the more generally received view, that murmurs must be referred only to the existence of valvular disease or imperfection, except in cases where the blood itself is altered in its constitution by disease.

The general symptoms of hypertrophy are sometimes very characteristic, but at other times they are not well marked, and may even be fallacious. Patients often complain of palpitation, but this is by no means a constant symptom, and where it does exist, its presence constitutes no special evidence of hypertrophy. Indeed, it is most generally complained of in nervous affections where the heart is not organically diseased at all. The pulse is neither irregular nor too frequent, but it is full and strong, as it might be expected to be from the increased muscular volume of the heart. When dilatation, however, exists in a very marked degree, the pulse is less strong, and when, as is very frequently the case, valvular disease exists as well as hypertrophy, the character of the pulse is modified according to the circumstances already explained in the section treating of the general symptoms of heart-disease, p. 77.

But the general symptoms of hypertrophy are sometimes so well marked that they cannot be mistaken, for the pulsation of the heart may often be seen as well as felt by ordinary bystanders or attendants, as well as by the patient himself; and there is often pain in the region of the heart, with some difficulty of breathing, increased on exercise, or on going upstairs, or up a hill. Other complications, such as pain in the head, loss of appetite, indigestion, paroxysms of asthma, and other ailments of greater or less severity, are not peculiar to hypertrophy, and may be due to other affections by which the hypertrophy is accompanied.

A general opinion is gaining ground that although hypertrophy is, strictly speaking, a morbid condition, it is a process usually set up by nature to compensate for some more serious disease elsewhere.

Thus when the valves are unable efficiently to perform their office and they either offer an obstruction to the onward progress of the blood, or they allow that fluid to return to the cavities from which it has been propelled, the muscular structure of the heart is called into increased activity, and consequently into increased nutrition, in order to compensate for the defect of the valves, and to force the blood with sufficient velocity through the impeded or inefficient orifices. Hence, hypertrophy in such cases can scarcely be considered a disease, but rather as a salutary and beneficial condition. These remarks, however, would only apply to those cases where valvular disease is proved to have been previously in existence. It is said that hypertrophy also compensates in some way for the obstruction offered to the heart's action by the adhesions of pericarditis, and it is not difficult to see the force of such a statement or of the reasoning on which it is founded. But admitting the very general coincidence or sequence of pericarditis and hypertrophy, I regard the latter as a disease developed by the same constitutional conditions as the former, though not necessarily caused by it, for neither upon mechanical nor physiological principles can it be contended that pericardial adhesions necessarily give rise to hypertrophy.

The treatment of hypertrophy will be considered in connexion with that of dilatation.

DILATATION

Dilatation of the heart, which is the *aneurism* of the heart described by Corvisart, Lancisi, and most of the earlier writers on cardiac pathology, may, like hypertrophy, affect either the whole heart or only a part of it. As its name implies, it signifies a widening of the cavities of the heart, and the name is, in fact, represented by the word *aneurism*, which is of Greek extraction, from *ανευρυνω*, to *widen* or *dilate;* but the term *aneurism* is now almost entirely restricted to certain well-defined diseases of arteries. Dilatation of the heart is most commonly associated with hypertrophy, and indeed it can scarcely ever be said to occur alone, or uncomplicated with some other altered condition. It is usually divided into simple, active, and passive dilatation.

In the *simple* form, the cavity is increased in capacity and its walls exhibit only their natural thickness, but even in this form it is evident that some hypertrophy must exist, for the cavity is

dilated, and consequently the extent of the walls, if not their thickness, must be increased. In *active dilatation*, the cavity is increased and the walls are considerably thickened, and this condition is therefore the same as that described as eccentric hypertrophy, and the two names are synonymous. But in *passive dilatation* there is enlargement of the cavity with a diminished thickness of the walls.

When the dilatation is of the *simple* or of the *active* form, the heart presents more or less of the same appearances and characters as in hypertrophy, because the two affections are combined together. But when the dilatation is *passive*, the heart is flabby and soft in texture and pale in colour, its apex is rounded, its transverse diameter is increased, its muscular structure is wasted, the chordæ tendineæ, which unite the valves to the muscular columns, are thinner and longer than in the natural state, and the valves are unable to close completely. Like hypertrophy, dilatation may involve the whole heart or only one of its divisions, but it usually affects both ventricles. The right ventricle, however, is sometimes exclusively dilated, and the auricles are sometimes, though rarely, more affected than the ventricles. The causes of dilatation are pretty much the same as those of hypertrophy, valvular disease being the most general.

The Symptoms of Dilatation.—When dilatation is associated with hypertrophy, the symptoms of both are the same, as has already been mentioned. But sometimes, as in the form called passive dilatation, the dilatation predominates over the hypertrophy and the symptoms are very different. Instead of the violent and visible impulse of the apex of the heart, the action of the dilated organ is imperceptible to the eye, and can scarcely be felt by the hand. The dulness on percussion is not increased as it is in cases of considerable hypertrophy. With respect to the sounds of the heart in passive dilatation very much difference of opinion has prevailed, and very conflicting statements have been made, even by writers of great and acknowledged reputation for accuracy of observation. Thus Laennec states, in opposition to Corvisart, who founded the diagnosis of dilatation chiefly upon the characters of the radial pulse and of the impulse of the heart, that "the only certain sign of dilatation of the left ventricle is that afforded by the stethoscope, namely, the clear and distinct sound of the contractions of the heart heard between the cartilages of the fifth and sixth ribs. The degree of the clearness of this sound and

its extent," he goes on to state, "are the measures of the dilatation; thus when the sound of the ventricular contraction is as clear as that of the auricular, if at the same time the heart's sound is easily heard in the right part of the back, the dilatation is extreme." [1]

But Andral, in a note appended to this passage of Laennec's work, raises some doubt as to the value of this sign; and subsequent writers, among the most recent of whom is Oppolzer, state that the sounds of the heart, so far from being increased in dilatation, are actually very much diminished in intensity, owing to the feeble condition of the organ. It is probable that the truth lies between the two opinions. Andral, who refers the sounds in question to the action of the valves, plausibly argues that the sounds may be heard most distinctly when the walls of the heart are attenuated; and Dr Walshe states that the systolic sound varies in its character in dilatation according as the cardiac tissue is firm or soft, and this probably is the real explanation of the different views entertained by eminent authorities upon a point of practical interest. When, therefore, the muscular fibres of a dilated heart are firm and fairly nourished, the systolic sound, although short and abrupt, is louder and more distinct than in health, but if the dilated ventricle is soft and flabby, the systolic sound is very faint and weak. The diastolic sound which, in the case of the firm muscular fibre, is not much affected, may, in the case of the soft and flabby condition of the muscles, be almost or quite inaudible at the apex of the heart. I think it may be stated generally that in proportion as hypertrophy is combined with dilatation, the impulse is stronger, and the sounds, though perhaps not louder, are more diffused; while in proportion as the dilatation preponderates over the hypertrophy, and the walls of the heart are thinner, and the muscular structure more soft and flabby, the sounds are diminished in intensity, and more limited in extent. Although, as Andral suggests, the valvular sounds may be intensified by the thinness of the ventricular walls, yet in extreme cases of dilatation the valves themselves are enfeebled in their action by the laxity of the tendinous cords and the flabbiness of the muscles, and the sounds are accordingly reduced to their minimum. There is no murmur heard in dilatation, unless it be in those cases where the valves are unable to close completely in consequence of their want of tension and the general flabby condition of the surrounding

[1] 'Traité de l'Auscultation Médiate,' par Laennec, vol. iii, p. 191.

structures, and if any murmur should then exist it will of course be regurgitant.

It is unnecessary to describe at any length the general symptoms of dilatation of the heart as an isolated affection, because upon none of them can reliance be placed. When dilatation is combined, as it usually is, with hypertrophy, the characters will be those already described under hypertrophy, and when both are combined with, or are developed as a consequence of valvular disease, the characters peculiar to the latter affection will be present. In the advanced stages of dilatation the general symptoms are usually those which belong to some other affection with which the dilatation is complicated, and in the early stages there may be no general symptoms at all, and the patient is probably unconscious that his heart is otherwise than healthy. In the early stages of dilatation, too, even the physical signs are very obscure, and in the advanced states they are not very characteristic, and may perhaps be mistaken for those of other diseases of the heart, such as softening and fatty degeneration.

Treatment of Hypertrophy and Dilatation

When the revelations of the stethoscope, or rather of auscultation, first began to call special attention to the morbid conditions of the heart, attempts were made to remedy these faulty states of the organ by therapeutical appliances, and in the case of hypertrophy and dilatation it was hoped and expected that the enlargement which had been detected might be reduced by measures often successfully employed in cases of external growths, tumours, and thickenings. Thus, I have known, when a student at the hospitals, leeches applied to the region of the heart, and blood drawn from the same region by cupping, in order to reduce the enlargement in cases of hypertrophied heart, and I have seen absorbents, especially iodide of potassium, prescribed internally and in large doses with the same object. But these attempts were never attended with success, and physicians have now ceased to rely upon any such plans in the treatment of hypertrophy. When the impulse of the heart is very strong and the pulse is full and bounding, and the patient is generally plethoric, I see no objection to the application of a few leeches over the præcordial region, and although I do not believe that bleeding has any power to reduce the bulk of the heart, yet I think that it may sometimes relieve congestion and tranquillise the system. But

to repeat the bleedings frequently, either with the view of reducing palpitation or of removing the morbid condition of the heart, I consider to be a vain and useless, and even mischievous proceeding, for palpitation would be more likely to increase than diminish under the practice of bloodletting, and the strength of the system would be reduced, the bulk of the heart remaining the same.

The very best therapeutical agent, if the reverse of motion can be strictly called an agent, is Rest. The hypertrophied heart is in a state of over-action and of over-nutrition, and the best method of reducing its nutrition is to diminish its activity, on the same principle that an ordinary muscle increases in size the more it is used, and diminishes under the reverse condition. Putting valvular lesion and pulmonary disease for the moment out of the question, hypertrophy is intensified by excessive emotions of the mind or over-action of the general muscular structures of the body, and the remedial measures must be sought in those plans or those therapeutical agents which secure most rest to the excited part. All excitement of the mind must therefore be avoided, all athletic exercises forbidden, and even all ordinary exercises must be limited as much as possible. To give three instances of hypertrophied heart taken from my own practice, and which strike me at the moment, I may illustrate my remarks by the case, first, of a soldier, accustomed to march and to drill; second, a bargeman, rowing a barge on the Thames; and, third, a blacksmith. In all these cases I, of course, recommend a discontinuance of the usual exertions and the selection of lighter employments, and by such a course, although the bulk of the muscular structure of the heart may not be diminished, yet the activity of the organ is lessened, the distressing effects of over-excitement are avoided, and serious or even fatal consequences may often be averted.

The remedies to be used must have the same tendency, namely, to procure and to maintain Rest, and they must be selected from the list of sedatives. Opium, however, is by no means to be recommended for its specific effect in tranquillising the heart, and it has the disadvantage of increasing general congestion, of causing fulness of the head, and inducing constipation of the bowels. Digitalis is, I believe, the true opiate of the heart when hypertrophy preponderates, and its effect in tranquillising over-action, and reducing the impulse to normal bounds, is truly marvellous, while the administration of the drug I regard as perfectly safe. I shall treat more at length on

the use of this valuable medicine under the head of the "Treatment of Valvular Disease," but I may state in the meantime that it may be given in doses of ten to twenty minims of the tincture three or four times a day. Next to digitalis, in my opinion, as a useful and innocuous sedative in hypertrophy of the heart, and indeed in heart-disease generally, is hyoscyamus, which possesses most of the beneficial properties, without producing the injurious effects, of opium. Henbane may be given either in the form of extract or in that of tincture, the former in doses of five grains, the latter in doses of half a drachm, three or four times a day, and the extract may be given in combination with some aloetic or other aperient, if the bowels are costive, and the tincture may be given in combination with digitalis. Dr Walshe speaks very highly in favour of the use of aconite in relieving the painful sensations often experienced in cases of hypertrophy. I myself consider aconite rather an uncertain remedy, but Dr Walshe states that it may be given with perfect safety in the cases now under consideration, in doses of one eighth of a grain of the alcoholic extract of the root. It may be observed that this particular extract is not contained in the British Pharmacopœia of 1867, which, however, orders a watery extract, the dose being from one to two grains; and there is also a tincture of aconite, the dose of which is from five to fifteen minims, this preparation being much weaker than either of the tinctures of aconite contained respectively in the former Dublin and the London Pharmacopœias. Another most useful medicine as a sedative in hypertrophy is belladonna, more especially, and perhaps I ought to say exclusively, used as an external application in the form of plaister over the præcordial region; the effects of this application are generally most satisfactory, and the plaister may be worn until it comes off and then its place should be supplied with a new one. Other sedatives have been recommended, as prussic acid and acetate of lead, but I have no experience of their effects, and should not be inclined to put much faith in them in the treatment of hypertrophy. I have as little faith in the internal administration of belladonna, however much I advocate its use as an external application.

Although all violent purging should be avoided in treating hypertrophy, yet it will be useful and advantageous to administer occasionally a gentle aperient, in combination with some light tonic medicine. As the liver is often congested, a little blue pill may now and then be administered, the dose being about two to three grains,

and the mercurial may be combined with the compound rhubarb pill or the compound colocynth pill. The light bitter tonics, such as infusion of quassia, of calumba, or of gentian, may be combined with small doses of the mineral acids, and administered two or three times a day. The diet should be light and nutritious, and while all excess in alcoholic drinks should be carefully avoided, I see no objection to allowing the patients suffering from hypertrophy to take a little bitter beer or some dry sherry, or a little gin-and-water, the diuretic effect of the last being sometimes very advantageous.

The treatment of dilatation as distinguished from that of hypertrophy requires no lengthened remarks. The earlier stages of this affection are very difficult, if not impossible, to recognise, and perhaps the patient is unconscious of suffering any inconvenience; and in the more advanced and developed cases there is almost always some complication either of hypertrophy or valvular disease, or pulmonary affection, which of course requires special treatment. But it must never be forgotten that dilatation of the heart is a very dangerous condition, and when it predominates over hypertrophy the worst consequences are to be apprehended. Digitalis, although most valuable in the latter, is quite contra-indicated in the former, and all purgatives and any other kind of depletion are alike objectionable and perilous.

CHAPTER XV

VALVULAR DISEASE.

In many of the preceding sections the subject of valvular disease has been noticed, and much of what relates to the symptomatology, diagnosis, and prognosis of valvular affections has been anticipated. Thus it has been shown that the only trustworthy symptoms indicating disease of the cardiac valves are derived from the sounds heard on auscultation; that the diagnosis is made by remarking the situation where the morbid sounds or murmurs are heard; and that the prognosis, although always to be made with caution, is not so unfavorable as was once supposed. It remains now, however, to describe *seriatim* the lesions of the different valves, the relative frequency of their occurrence, the comparative importance of each lesion, the complications it induces, and the treatment it requires. But by way of preface it is necessary to allude to the mechanism and the structure of the valves in general, although with special reference to the diseases or imperfections to which these parts are liable.

The valves are formed by thin layers of fibrous tissue enclosed between folds of the lining membrane of the heart. They form a series of movable flaps, or folding doors, or flood-gates, which open to allow the onward passage of the blood, but close immediately afterwards to prevent its reflux.

In the case of the *semilunar valves* which are placed at the base respectively of the aorta and the pulmonary artery, the fibrous tissue of which the valves consist is continuous with a tendinous ring lying between the muscular fibres of the ventricle and the elastic fibres of the walls of the artery. While the blood is moving onward, the valves lie flat against the arterial walls, but when the blood has passed, the walls of the artery are dilated so that the free margins of the valves which previously lay in contact with the inner surface

of the artery are drawn inwards towards the centre, and are pressed together so that they come in contact and close the arterial tube. If the blood above the valves should press down upon them, the only effect of this backward movement would be to approximate the valves more closely together, and thus more securely to close the passage, and it is only in cases where the valves are torn, or are deficient, or are rigid, or are in some other abnormal condition, that their closure is prevented, and the reflux or regurgitation of the blood is permitted.

The *auriculo-ventricular valves*, placed respectively between the right auricle and ventricle, and between the left auricle and ventricle, perform the same kind of function as the semilunar valves of the aorta and the pulmonary artery, and they equally secure the closure of the auriculo-ventricular orifices by the contact of parts of their surfaces, but the margins and points of the semilunar valves meet together in a different manner to that displayed by the auriculo-ventricular valves. The latter, called respectively the mitral and the tricuspid valve, are different in structure from one another, one having two pointed portions and the other three, although, strictly speaking, there is between each two principal points a smaller one, so that the tricuspid valve consists of six portions and the mitral of four. Each of these portions is more or less triangular, the base of each triangle being attached, as in the case of the semilunar valves, to a tendinous ring, but the apex and sides of each auriculo-ventricular valve are attached to the muscular walls and columns of the ventricles by delicate tendinous chords, which are called, in fact, *chordæ tendineæ*. Now, while the blood is passing from the auricles into the ventricles, or, in other words, when the latter cavities are being dilated, the sides of the valves lie flat against the ventricular walls; but when the muscular substance begins to contract, the valves are drawn away from the sides by the tendinous chords, their free edges and parts of their borders come in contact, and the blood, which is now going onward into the great arteries, is prevented from going backwards through the auriculo-ventricular valves, because the latter are the more firmly closed by the blood which now presses them together through being placed between the muscular substance and the valves themselves. Hence the backward current of the blood, so far from forcing a passage through the auriculo-ventricular valves, only presses their sides and borders together more closely, and, as in the case of the semilunar valves,

it is only in certain abnormal conditions that regurgitation can take place.

The valves, then, are composed of fibrous structure enclosed in folds of a lining membrane, which is the same as that which covers the inside of the heart and lines the vessels, and is analogous, though not identical, in its nature with that of serous membranes. Now, it would appear that fibrous structures are especially liable to become diseased in attacks of acute rheumatism, and I am probably correct in stating that it is the fibrous and not the serous tissue of the valves which is primarily affected in the so-called rheumatic endocarditis. Notwithstanding the eminent authority of Bouillaud, who would describe the whole of the endocardium as being inflamed in endocarditis, I am inclined to think that it is only the valves, and especially their fibrous tissue, which are thus affected, and indeed there are no post-mortem appearances of a trustworthy character in endocarditis which imply disease of any other part. The redness and congestion of the lining membrane sometimes described as indications of inflammation are really of no pathological importance, and are probably only post-mortem changes caused by passive congestion of the capillaries, and it is only by the discovery of thickening of the fibrous tissue of the valves, or by the presence of lymph on their surfaces or borders, that the inflammatory nature of the attack can be determined. It is a property of inflammatory lymph, also, to assume characters belonging to the part in which it is produced, and hence when lymph has been effused in the fibrous texture of the valves, it will be developed into tough fibrous matter, and may cause the thickenings which give rise to obstructive disease on the one hand, or to rigidity of the valves, and their consequent insufficiency, on the other.

It is universally admitted that one of the most fertile causes of valvular disease is acute rheumatism, which peculiarly affects the fibrous tissues of the body in general, and consequently often invades the valves of the heart. The great desideratum in the treatment of acute rheumatism is to prevent the implication of the cardiac structures, because, as long as the disease is limited to the joints, it is a comparatively harmless affection, however painful it may be to the patient. It is stated, on statistical authority, that the modern treatment of acute rheumatism does really obviate cardiac complications in a very great measure, and I have already expressed my belief in the truth of this statement; but it nevertheless happens in

a large number of cases that the valves are involved. When this is the consequence or the concomitant of acute rheumatism, lymph is effused into the fibrous texture of the valves, or is deposited, while it is recent, on their surfaces, and hence the valves become diseased. The lesions thus broadly sketched constitute the acute diseases of the valves, but they often and indeed generally lead to more or less permanent impairment of these organs, and then the lesions become chronic, and it is in this condition that they usually present themselves to notice.

The chronic diseases of the valves are in very many instances due to the pre-existence of acute disease in connexion with articular rheumatism, but this is by no means invariably the case, for many instances of valvular disease present themselves in which there is no history of rheumatism. In old age, as I have previously remarked, there is a frequent tendency to the deposition of fatty, atheromatous, and even osseous matter in the fibrous structure of the valves or in the coats of the vessels, and these deposits lead to the same morbid conditions of the valves as are caused by the inflammatory process attending acute rheumatism. But not only does this degenerative process occur as age advances, but it is sometimes developed in early or mature life, and hence it is not uncommon to find valvular disease in middle age, or even in children. There are also other diathetic diseases besides acute rheumatism which may induce valvular lesions, as for instance Bright's disease, to which I have before alluded; and excessive indulgence in alcoholic drinks may also induce premature degeneration in general, and valvular disease in particular. But whatever may be the cause of valvular disease, the lesions hitherto described all consist in the development, in the substance or on the surface of the fibrous tissue, of the products of inflammation, or of that peculiar matter to which the term *atheroma* has been given, and to which attention must now be directed.

It is found that a deposit very frequently takes place beneath the lining membrane of the arteries or within the fibrous tissue of the valves, and it has a whitish colour and a soft feel, something like that of pap, from which the name *atheroma* is derived, the word being of Greek origin and signifying *pap*. This deposit, which is very commonly seen in the post-mortem examination of the vessels, especially in the aged, appears to be of a partially fatty nature, and to be one of the products of the degenerative process in the human body. The first stage of this degeneration, so far as the valves and

the arteries are concerned, appears to consist in the deposition and multiplication, within the fibrous tissue, of minute cells mixed with globules of oil, and afterwards of masses of fat and cholesterine, which eventually coalesce into patches and plates of greater or less size, and constitute atheroma properly so called. But this atheromatous condition gradually passes on to a further stage of degeneration, in which the soft and pappy consistence of the deposition is changed for a hard and tough one, the deposit now resembling cartilage, and finally this cartilaginous substance is replaced by inorganic masses resembling bone, although not constituting osseous tissue in the strict sense of the term.

Thus the steps of degeneration are, first, the proliferation of cells and the deposition of minute oil-globules in the substance of the tissue; then the aggregation of disintegrated fibres and cells, fatty matters, and cholesterine, and their coalescence into atheromatous patches; then the conversion of atheroma into a substance resembling cartilage; and finally the deposition of inorganic matter, especially the phosphate of lime, in the cartilaginous masses. Hence, eventually, the valves appear to be completely converted into bone, although the osseous matter does not present to the microscope the same details of structure as are found in the true bones. The term ossification, therefore, although very frequently employed to denote the condition just described in the valves and arteries, is not strictly correct; and as the appearances depend mainly, if not entirely, on the deposition of calcareous salts in the degenerated masses, the words calcification and petrifaction would perhaps be more appropriate.

But besides these causes, which may be called constitutional, of valvular diseases, there are others which may be termed accidental, as when a portion of a valve is torn or ruptured, or otherwise undergoes some mechanical injury; and sometimes the disease is due to malformation. The valves are sometimes indirectly injured by external accidents, and sometimes by violent muscular exercise.

Dr Peacock, who has collected a number of instances of valvular diseases in his lectures delivered before the Royal College of Physicians of London in 1865,[1] states that the first instances in which the valves were actually shown to have been seriously injured during muscular exertion are those reported by Corvisart, one of them having occurred

[1] 'On Some of the Causes and Effects of Valvular Diseases of the Heart,' by T. B. Peacock, M.D., London, 1865.

in the practice of that physician in the early period of the first French Revolution. Since that time many other cases have been reported of the same kind, and several specimens exhibiting the lesions in question have been exhibited before the Pathological Society of London, and have been figured and described in the Transactions of that Society. Dr Peacock himself relates four cases, in all of which the aortic valves were injured during violent muscular exertion, the patients being respectively a sailor, a dock-labourer, a flyman, and a ship's carpenter, each of whom was engaged in some rather violent exertion when the injury to the valve took place, which was subsequently discovered on post-mortem examination. The four cases were all instances of injury of the aortic valves, and an analysis of other cases on record shows that these valves, being exposed to the pressure of the blood in the arteries, are the parts most frequently injured. But the other valves are occasionally ruptured, and it appears from an analysis made by Dr Peacock, that out of seventeen cases the aortic valves were injured in ten, the columns of the mitral valve in four, and the columns of the tricuspid valve were torn in three cases. As illustrating the kinds of muscular exertion which are reported to have caused the valvular injuries, it is stated that in one case the patient had made a long and rapid journey on horseback, one was rowing, two were pulling or rolling heavy casks, two were running, others were climbing, and, in some, violent coughing appeared to be the cause of the rupture. In the above cases the valves were previously healthy when the accidental rupture occurred, but in other instances the valves, being previously diseased, give way under some slight exertion or shock.

Valvular disease is also sometimes caused by malformation of these structures, either congenital or acquired. Thus the segments of the semilunar, aortic, and pulmonary valves may be either too many or too few in number. Dr Peacock thinks that the segments of the valves are originally double, but become blended together in the progress of development, and that when there is an excess in the number of the segments, it is owing to an arrest of development rather than to an excess of growth. When the number of the segments is defective, there may be only two, or the valvular apparatus may consist of only a single imperfectly-formed curtain. This malformation may also be accounted for by supposing that, during fœtal life, the angles and contiguous sides of two or more of the

valves become adherent. When the segments of the valves are too numerous, no particular inconvenience is caused, but when they are deficient in number, the malformation causes more or less obstruction, and may lay the foundation for serious disease. Besides the congenital cases of malformation of the valves, there are many others which appear to be developed from disease, as when the aortic valves form a septum stretched across the aortic canal, and there is a triangular opening in the centre of the septum, or as when the segments are perforated by apertures; and there are many other anomalous malformations, described in recent works, and which are exhibited and recorded in Pathological Societies.

Valvular disease is far more frequent on the left than on the right side of the heart. Dr Hope states it as his general impression that out of 400 cases of valvular disease only 20 were instances of disease of the right valves, or, in other words, the proportion was only five per cent., and he suspects that the proportion is even lower. It is very difficult to offer a satisfactory explanation of this disproportion, but Dr Hope agrees in the view advanced by Corvisart that the left valves have a more decidedly fibrous organisation than the right, and are therefore more liable to suffer from the effects of rheumatic inflammation, and also to undergo transformation into cartilaginous and bony matter. It is also certain, although the explanation of the fact is difficult, that endocarditis is far more frequent on the left than on the right side of the heart; and it may further be suggested that the left valves are more strained by the greater power of the left ventricle and the stronger retrograde pressure of the blood in the aorta.

Dr Peacock, however, does not admit the force of the views expressed by Corvisart and Hope as to the more fibrous structure of the left valves rendering those parts more liable than the right to disease. He thinks that the disproportion may be due to the larger amount of fibrine in arterial as compared with venous blood, and also to the more active functions and greater exposure to variations of pressure and to injuries of the left side of the heart, from its connexion with the systemic circulation.

It appears from the evidence of statistics that men are more liable to valvular disease than women, the former being more liable to aortic valvular affections. The cause of the difference is to be found in the greater liability of the aortic valves in men to injury from accident and over-exertion, and to the fact that men are more liable than

women to diseases arising from cold and wet, and from intemperate habits.

The Consequences of Valvular Disease

It has been seen that valvular diseases may be distinguished into those which allow regurgitation in consequence of insufficiency of the valves, and those which cause an obstruction to the current of the blood by some narrowing (*stenosis*, from στενος, *narrow*) of the orifices. *Insufficiency* and *stenosis* are therefore the terms sometimes employed to denote these two conditions. The immediate consequences of insufficiency of the valves are regurgitation of the blood into that cavity of the heart which is placed behind the affected valves, a consequent overflow of blood into that cavity, and a passive dilatation as the result of the overflow. With this dilatation hypertrophy is usually associated, and is of the kind called excentric. This latter affection, namely, excentric hypertrophy, is, under the circumstances now described, a result of the highest importance in securing the continuance of life, because, if it did not supervene, the passive dilatation of the cavity lying behind the affected valve would soon be so considerable, in consequence of the repeated regurgitation, that the walls of the heart would be unable to exert sufficient power to cause contraction, and thus the circulation would be arrested and the heart would cease to beat. This hypertrophy must therefore be regarded as a compensating power to counteract the insufficiency of the valves, and as a condition calculated to rectify the disturbance which would otherwise be occasioned in the circulation of the blood.

Thus, then, the immediate physical result of insufficiency of the valves is always a dilatation of that cavity of the heart into which the regurgitation of the blood takes place. But in stenosis or narrowing of the apertures the case is different, for then the portion of the heart behind the constriction is called upon for greater efforts to propel the blood through the contracted orifice, and consequently this portion of the heart becomes gradually hypertrophied. If this hypertrophy did not supervene, too little blood would be propelled through the constricted orifice, and thus there would be on the one hand a deficiency of blood above the constriction and an excess of blood below it. The hypertrophy, therefore, of the part below the constriction compensates in some measure for the disturbance caused by the valvular obstruction. But this hypertrophy does not continue for a long time without causing dilatation, because, notwithstanding

the more powerful contraction of the muscular walls, the continual overflow of blood into the cavity causes dilatation sooner or later, and thus excentric hypertrophy is established. The results, therefore, of insufficiency and stenosis are practically the same, although the order of sequence in which the changes occur is reversed, and in both cases the subsequent morbid condition of the heart must be regarded in the light of a compensating power to rectify the valvular disease.

The remote consequences of insufficiency and obstruction of the valves are of great practical importance, and constitute some of the most remarkable rational symptoms of those affections. Both insufficiency and stenosis cause an overflow and consequent stoppage of the blood in that part of the heart which is below the insufficient valve or the contracted orifice. This stoppage or retardation of the blood is propagated backwards, and in the case of the tricuspid and pulmonary valves is observed in the right auricle, or in the right auricle and ventricle, and thence in the two venæ cavæ; and in the case of insufficiency or stenosis of the mitral valve or of the aorta, the stoppage is traced from the left auricle or the left auricle and ventricle into the lungs, and so onwards to the right side of the heart, and thence equally into the venæ cavæ.

Thus it will be perceived that whether insufficiency or stenosis be situated on the right or the left side of the heart, the general results are the same, namely, that retardation of the blood-current is caused somewhere *behind* the diseased orifice; but in cases of disease of the right side, the retardation exists only in the right auricle and right ventricle and in the venæ cavæ, while in disease of the left side the retardation and its consequent lesions exist *also* in the lungs. In connexion with heart-disease, therefore, it might be supposed that pulmonary congestions and inflammations were almost wholly dependent on disease of the left valves, but the very important fact must not be forgotten that not only may insufficiency and stenosis of the left valves cause diseases of the lungs, but that the same diseases may be caused by the disintegration and detachment of fragments of lymph, from the pulmonary semilunar valves or the tricuspid valve. This cause of pulmonary disease, constituting one of the morbid phenomena to which the name of *embolism* has been applied, and to which reference has already been made, is daily attracting more and more the attention of pathologists and practical physicians, and many obscure affections of the lungs will probably receive a satisfactory expla-

nation when the conditions under which embolism occurs are more thoroughly investigated and understood.

As congestion and obstruction of the blood are caused by insufficiency and stenosis of the cardiac valves, the venæ cavæ, which bring the blood from all parts of the system, become over-distended, and this distension acts upon all the parts, which are, so to speak, situated *behind* the place of obstruction. Thus, the liver, which transmits its blood to the inferior vena cava by the hepatic vein, becomes congested when the former vein is obstructed, and for a similar reason the kidneys, which discharge their blood by the renal veins into the inferior vena cava, are also congested in valvular disease. The same observation applies to the spleen and indeed to all the organs situated in the abdomen, nor do the head and neck escape, for these parts send their blood into the superior vena cava, and are alike affected when obstruction of this vessel ensues from valvular disease. But not only do the large organs situated in the great cavities of the body suffer from congestion of their structure and consequent impairment of their functions, but the capillaries of the skin suffer from over-distension, and hence the cutaneous surface presents a more or less livid appearance from the inability of the capillaries to transmit the blood sent into them by the arteries. As a consequence, too, of the over-distension of the capillaries, the serous portion of the blood exudes through the walls of those vessels into the circumjacent areolar tissue, and thus constitutes dropsy, usually commencing at the lowest parts of the body, as the ankles and feet, and gradually ascending up the legs, thighs, and abdomen.

The effects produced upon the arterial system by insufficiency and stenosis of the cardiac valves are different from those caused in the venous system, for the arteries receive an insufficient supply of blood, and what they do receive they are unable to propel through the overloaded capillaries. Hence the arterial blood is deficient in quantity, while the venous is in excess, and the general result upon the circulation is that the arterial current is retarded, and, as a rule, the pulse is slower than usual in valvular disease. It is also sometimes intermittent, although this is not by any means so frequent a result as might be anticipated. From the venous congestion in the head there is often headache in valvular disease, and as the liver and the stomach are congested, indigestion is a very common symptom, and indeed is sometimes the only one experienced by the

patient. In the case of a gentleman whom I attended for many years, who had mitral disease, I have already stated that the only inconvenience of which he was conscious was indigestion. From the congestion of the kidneys, the function of these organs in eliminating urea is impaired, and Bright's disease may be established, though this affection is as often the cause as the consequence of the imperfection or obstruction of the cardiac valves.

Having thus described generally the symptoms, causes, and consequences of valvular disease, I proceed to notice separately, but briefly, the symptoms of each of the diseases of the valves. In this department of Practical Medicine an extraordinary degree of exactness has been attained in diagnosis, and I think that the great merit of having first accurately determined the significance of the morbid sounds of the heart in reference to valvular affections is justly due to Dr C. J. B. Williams and Dr Hope. The latter physician points out that Laennec, while he unquestionably discovered the existence of the cardiac murmurs, was unacquainted with their real significance, and that in the explanation of some of the sounds of the heart the great French auscultator was mistaken altogether. Thus Laennec attributed the murmurs to spasm of the heart only, while Dr Hope has very clearly shown that these sounds depend either upon valvular disease or on an altered condition of the blood. Laennec was unacquainted with the whole class of regurgitant murmurs which were subsequently discovered by Dr Hope; and the former further laboured under the disadvantage of attributing the second sound of the heart to the contraction of the auricles, while Dr C. J. B. Williams, and after him many others, proved that the auricles yield no sound, and that the second sound is due to the closure of the semilunar valves.

If the special diseases of the valves were described in the order these organs occupy in the course of the circulation, the lesions of the tricuspid valve would be first considered, then those of the pulmonary semilunar valves, then those of the mitral, and lastly those of the aortic valves. But the diseases of the tricuspid valve and of the semilunar pulmonary valves are comparatively rare, and it is therefore convenient to commence with those which are most frequent.

Diseases of the Mitral Valve.—The mitral valve may present an obstruction to the current of the blood, or it may allow regur-

gitation in consequence of the insufficient closure of its curtains. There can be no doubt that both these conditions very frequently exist, and yet it is an extraordinary fact that only the physical sound of mitral regurgitation was or is generally heard in the great majority of cases.

It has already been mentioned, however, that the obstructive murmur, now called *præsystolic*, has been brought into notice of late years (see p. 76), and that it may be usually detected if due precaution be taken in watching the period of the heart's rhythmic action at which it occurs. In former times there is no doubt that the obstructive and regurgitant murmurs were mistaken for one another or confounded together, and even in the present day the mistake is by no means unfrequent.

The merit of first describing this præsystolic murmur is usually attributed to Dr Gairdner, of Glasgow, who developed his views in a lecture published in 1861, but Dr Hilton Fagge, in a paper on the subject in the 'Guy's Hospital Reports' for 1871,[1] shows that M. Fauvel, of the Hôtel Dieu in Paris, promulgated precisely the same opinions in the 'Archives Générales,' 1842. Dr Fagge, in his historical *résumé*, does justice to those who have described this murmur, but he shows that, on the whole, it has not been recognised or described, until lately, by either British, German, or French physicians. Dr Sutton, in 1867, in a paper already referred to (p. 74), while concluding, from his experience, that præsystolic murmurs are not rare, yet admits that they are difficult of diagnosis.[2]

Dr Hayden, of Dublin, certainly deserves the credit of having been the first to describe the præsystolic murmur in Ireland. In two clinical lectures delivered in 1866 [3] at the Mater Misericordiæ Hospital, Dr Hayden gives the history of six cases of mitral obstruction, in five of which the diagnosis was confidently made during life, and in three the post-mortem examination confirmed it. In one of the cases, which Dr Hayden justly regards as a typical example of this valvular disease, the post-mortem examination showed that the mitral orifice consisted of a funnel-shaped passage formed by the agglutination of the segments of the mitral valve, and projecting into the left ventricle. The passage was an inch and a

[1] "On the Murmurs attendant upon Mitral Contraction." By C. Hilton Fagge, 'Guy's Hospital Reports,' 1871, p. 247.

[2] 'Clinical Lectures and Reports of the London Hospital,' 1867-8, p. 297.

[3] 'Clinical Lectures on Diseases of the Heart: Mitral Obstruction.' By Thomas Hayden, Dublin, 1866.

quarter long, and so narrow at the end as barely to admit the point of the little finger, and the segments of the valve were much thickened.

The præsystolic murmur, therefore, when it is heard, denotes an obstruction of the orifice of the mitral valves. On post-mortem examination this condition is easily recognised, and has often been described, and indeed I have described it myself in one of my own cases (see p. 97); but, as I have before observed, the physical sign during life was supposed to be the systolic mitral murmur. The characters of the disease are the narrowing of the mitral orifice and the induration of its walls, which occasionally contain calcareous matter; and the aperture sometimes resembles a tube, sometimes it is funnel-shaped, and sometimes it has a *button-hole* appearance.

In Dr Hilton Fagge's very able paper, in the practical researches connected with which he was favoured by the ample field of observation offered in Guy's Hospital, he describes several cases which came under his own observation. That the appreciation of a præsystolic murmur, however, as indubitably connected with mitral obstruction, is not, or was not, very common, may be estimated by the fact that during some years in the great practice of the establishment referred to, Dr Fagge adduces only six or seven cases where a direct mitral or præsystolic murmur was heard during life, and in which the mitral orifice was found after death to be contracted. In forty other cases which he relates, the mitral orifice was found after death to be narrowed, but no præsystolic murmur had been heard during life; and in nineteen others a præsystolic murmur was recognised by auscultation, but no opportunity was afforded of verifying the diagnosis by post-mortem examination. I may perhaps mention here in a parenthesis, that it would hitherto appear that the mitral obstructive disease is far from being the most dangerous of the valvular lesions of the heart, and, although experience is as yet wanting to warrant any decided assertion, it might almost be advanced that it was much less so than the others.

I think, then, it may be assumed that the præsystolic murmur sometimes does not exist, even in cases where the mitral stenosis might lead us to expect it, and I am sure that even when it does exist its presence is often overlooked. The difficulty of always finding it, when present, is partly due to the fact that its exact relationship in point of time to the rhythmic movements of the heart is often disregarded or mistaken. It is obvious, from all that has been advanced, that this murmur immediately *precedes* the systole

of the ventricles, and therefore precedes the arterial pulse. Hence it is always necessary, in examining the heart, to count the pulse at the wrist while listening with the stethoscope, and this precaution will generally indicate the true position, in point of time, of the mitral murmur. But it is preferable to place the finger on the carotid artery in order to be certain of the diagnosis, and it is better still to place the finger lightly on the apex of the heart itself at the time of making the stethoscopic examination.

Another collateral indication of the true nature and cause of the præsystolic murmur has been derived from the cardiograph, which is a modification of the sphygmograph. By this instrument it has been shown that, in cases of mitral stenosis, there is often a prolonged contraction of the hypertrophied left auricle during the ventricular diastole, and hence the præsystolic murmur is plausibly considered to be due to the sound caused by the propulsion of the blood through a contracted orifice by an hypertrophied auricle. Not only, therefore, may the nature of the præsystolic murmur be fairly inferred by the use of this instrument, but its occurrence in point of time may be accurately ascertained.[1]

In some cases, where this hypertrophied condition of the auricle does not exist, it is possible that, although there may be mitral stenosis, the blood is not propelled with sufficient force to cause a murmur, and hence it is not appreciable.

The only other question which suggests itself on this subject is, whether the præsystolic murmur is so rare as was once supposed, or is so common as some authorities now believe it to be. I am inclined, on the whole, to conclude that, for the reasons above advanced, it is comparatively rare. Dr Fagge, as has just been seen, had forty cases of mitral stenosis, in which no præsystolic murmur was heard during life, and Dr Austin Flint, who seems to have been long well acquainted with this murmur, found only 47 cases, out of 123 of mitral disease, in which it was noted.[2]

In the following case, which is now, among several others, still under my observation, there can be no doubt that there exists stenosis of the mitral valve, together with other lesions indicated. In fact the patient has *four* morbid sounds and four corresponding morbid conditions of the heart.

[1] "The Physiology and Clinical Use of the Sphygmograph." By F. A. Mahomed, 'Medical Times and Gazette,' May 18, 1872, p. 569.

[2] Flint on 'Diseases of the Heart,' 1870, p. 205.

Case.—W. P., æt 17, a thin and emaciated boy. He had acute rheumatism when he was 15 years old, and he was then ill about six or eight weeks. *Present state.*—There is visible pulsation of the heart, and also of the right common carotid and external carotid arteries, but not of the radial. The pulse is 84, and jerking; it is the *pulse of unfilled arteries.* There is *frémissement cataire* over the præcordial region, and increased impulse of the heart. There is a distinct double murmur over the apex of the heart, the first before the radial pulse, the second synchronous with it. There is double murmur also over the base of the heart.

This boy follows a light employment, and enjoys what appears to be tolerable health. He has no swelling of the legs, no cough, the appetite is good, and he sleeps well.

A *regurgitant mitral murmur* is one of the most common physical signs of valvular disease of the heart. Dr Flint considers it to be the most common of all the organic murmurs.[1] It is heard during the systole, often, and indeed, generally, obscuring the natural first sound, and it indicates the reflux of blood through the incompetent valves. The mode of distinguishing this incompetence after death is to put a ligature round the aorta, and then, having cut off the apex of the heart, to invert the organ and to allow a stream of water to fall from the ventricle towards the inferior surface of the valve. If the water is completely arrested in its progress, the valve is of course healthy and competent, but if, on the contrary, the water partially or wholly passes through, then the valve is in all probability diseased, some allowance, however, being made for the absence of the vital contractions which, in the living state, contribute to its closure. The regurgitant murmur may be soft and blowing, constituting the bellows murmur, or it may be harsh and grating, constituting the *file* sound, or it may be loud and scraping; but although these varieties are supposed by some authorities to indicate different characters or qualities of the physical cause of the imperfection of the valve, I am not aware that there is any constant relation between the loudness or the pitch of the sound and the gravity or otherwise of the physical lesion. The quality of the sound, in fact, probably depends more on the physical condition of the muscular structure of the heart and the force of the circulation than on the specific lesion causing the imperfection.

The *causes* of mitral regurgitation and of obstruction may be iden-

[1] Flint on 'Diseases of the Heart,' p. 208.

tical, for the same fibrous or cartilaginous or ossific deposit, which offers an obstruction to the current of the blood, will also so stiffen or otherwise impair the mechanism of the valve as to prevent its adequate closure. It must therefore be generally understood that although, as has been observed, the detection of a mitral obstructive murmur is a problem of some difficulty, the obstruction nevertheless may exist in many cases where a regurgitant murmur alone is heard by the auscultator. Still there are some special causes which may give rise to regurgitation through the mitral valve, such as shortening of the chordæ tendineæ, which prevents the closure of the tongues of the valve; and a mitral regurgitation may be caused without any actual disease of the valve, by a maladjustment of its segments, or even by such a dilatation of the muscular walls of the heart as prevents the complete closure of the auriculo-ventricular orifice. Regurgitation through the mitral orifice, then, is indicated by a murmur which may be of various character, heard over or towards the apex of the heart during the systole, and synchronous, or almost so, with the carotid and radial pulse. This character distinguishes it from the præsystolic murmur which occurs, as the name implies, *before* the systole. The greatest intensity of the systolic mitral murmur is over the apex of the heart on the left of the sternum, and the sound is propagated laterally towards the lower angle of the left scapula, where it may often be distinctly heard. This character generally distinguishes it very clearly from the tricuspid systolic murmur, which is loudest over the ensiform cartilage and is *not* propagated to the angle of the scapula.

The pulse in mitral disease, as has already been mentioned, is usually small, and sometimes it is intermittent or irregular. As a consequence of mitral regurgitation, the muscular walls of the ventricle very often become hypertrophied, the enlargement being a kind of compensatory operation set up by Nature to counteract the injurious effects of regurgitation. Disease of the lungs is another very common sequence of mitral regurgitation, and exhibits itself in the form of bronchitis, pneumonia, or pulmonary apoplexy. Hæmoptysis is for the same reason, namely, the reflux of blood towards the lungs, a somewhat common symptom of mitral disease; and without any physical lesion of the pulmonary structures, but in consequence of the obstacles experienced by the blood in passing through the chambers of the heart, difficulty of breathing, often culminating in attacks of spasmodic asthma, is a very common symptom. But, as I have

before proved, mitral disease may exist for many years without producing much constitutional disturbance, the system apparently accommodating itself gradually to the defective condition of the circulation.

Diseases of the Tricuspid Valve.—It has already been mentioned that appreciable disease of the valves on the right side of the heart is comparatively rare, although the explanation of this disproportion is not easy; and even where such disease does exist the physical signs are sometimes not well marked. It may, however, be stated that regurgitant disease of the tricuspid valve is indicated by a systolic murmur towards the right of the apex of the heart, and that this murmur is usually heard loudest over the ensiform cartilage, and is almost inaudible at the left apex. By this distinction the diagnosis between mitral and tricuspid regurgitation may be determined. One of the best indications of disease of the tricuspid valve is seen in the distension and pulsation of the cervical veins—a circumstance not difficult of explanation when it is recollected that the reflux of blood through the right auriculo-ventricular opening, or its obstructed passage through that aperture, will send back the venous blood to the venæ cavæ, and thence to the other veins, among which the external jugular, being visible, will exhibit the swollen and pulsatile condition alluded to. Obstruction of the tricuspid valve ought to give a præsystolic murmur, heard loudest at the ensiform cartilage, but inasmuch as this murmur is heard with some difficulty, even in cases of mitral obstruction, which is a common affection, it is still more difficult to hear such a murmur in tricuspid obstruction, which is comparatively a very rare one. Dr Flint observes that a tricuspid direct murmur (by which he indicates the præsystolic murmur) is "one of the rare curiosities of medical experience."[1] It would appear also that when tricuspid disease does exist, it is usually in connexion with mitral disease, although this combination, according to some results recorded by Dr Peacock, only occurs in about the proportion of three per cent. in valvular diseases of the heart in general.[2]

Dr Sutton, in his paper published in the 'Clinical Lectures and Reports of the London Hospital,' believes that regurgitant disease of the tricuspid valve is a common affection, and that it is perfectly

[1] On 'Diseases of the Heart,' p. 240.

[2] Peacock on 'Valvular Disease of the Heart,' p. 75.

practicable to distinguish it during life. The murmur is usually heard with the greatest intensity over the ensiform cartilage, but this is not so in every instance, for in some, he says, it is heard loudest a little to the left of the sternum. The tricuspid murmur also differs from the mitral in not being heard at the angle of the left scapula. Dr Sutton relates several cases in which the murmur in question was heard during life, and the tricuspid valve was found patent on post-mortem examination.[1]

When viewed in the light and according to the explanations proposed by Dr Sutton, it will, I think, be admitted that tricuspid regurgitation is by no means rare, although the visible signs of disease of this valve are seldom observed after death, for in Dr Sutton's cases the regurgitation through the tricuspid orifice was brought about by the great distension of the right ventricle, the latter being so dilated that the valves could not close the orifice. The cause of this distention was, in six of his cases, chronic disease of the lungs, and in two, contraction of the mitral orifice, and these conditions will, as is well known, very frequently induce enlargement of the whole heart, including the right ventricle, and the patency of the tricuspid orifice is easily explained by the consequent separation or widening of its walls, which are thereby unable to contract completely, and thus they prevent the due transmission of the blood-current. Dr Sutton, however, admits that the murmur, which he regards as characteristic of tricuspid regurgitation, is often absent even in the cases when, according to his view, it ought to be heard, and, on the whole, the question to be determined is not whether regurgitation through the tricuspid orifice may not occur under the very common conditions just described, but whether this regurgitation can be accurately determined during life by stethoscopic examination. Dr Sutton does not allege that obstructive disease of the tricuspid valve is at all common, and in the post-mortem examinations of the cases of tricuspid regurgitation which he records there was scarcely any other appearance presented by the valve than the fact of its patency brought about by the general enlargement of the right ventricle.

As far as my own experience goes, I fully admit the existence of tricuspid regurgitation and the reasons for its occurrence. The diagnosis of such a condition is made without much difficulty during life, and doubts can only arise when there is a complication with

[1] 'Clinical Lectures and Reports of the London Hospital,' vol. iv, 1867-8, p. 288.

other valvular diseases. After death the tricuspid valve itself exhibits nothing abnormal except its great patency, allowing four or five fingers to pass through with facility, and a certain flabby condition of the fibrous element of the valvular structure. In a case of tricuspid regurgitation without complication with other valvular disease there is dilatation of the heart, and indeed the latter is the necessary condition causing the former. The walls of the heart are weak, and the cavity of the right ventricle is dilated, and consequently the circumference of the valvular orifice is not brought together so as to close the passage, and regurgitation consequently ensues. A case of the kind occurred to me while I was writing these pages. The patient was seen by me on March 15th, and died suddenly on the next day; and on the post-mortem examination which I made on the 18th, the condition of the tricuspid orifice above indicated was clearly shown.

But even the organic diseases of the tricuspid valve are perhaps not so rare as is generally supposed, and I am surprised that they have received so little notice in systematic books on the diseases of the heart. They appear, in fact, to be not uncommon as congenital maladies, and most probably they derive their origin from some cardiac disease existing before birth. Dr Hope mentions only two cases of disease of the tricuspid valve, but in both the malady was recent and was complicated with corresponding disease of the mitral valve;[1] and Dr Austin Flint states that the instances of tricuspid regurgitation occurring in consequence of organic changes analogous to those which affect the mitral valve are exceedingly few.[2]

I have myself met with several cases of organic disease of the tricuspid valve, but principally in children, and one of those cases is now under my care. They were instances of congenital disease. In the following case, which was also probably congenital in its origin, the patient had arrived at the age of nineteen. It is a very interesting case of disease chiefly affecting the right side of the heart. The heart was exhibited by me at one of the meetings of the Medical Society of London, and it is now in the pathological department of the Museum of the Royal College of Surgeons of England.

Case.—A. C., a feeble and emaciated girl, æt. 19, had long been under my care as a patient at the Bloomsbury Dispensary. She had

[1] Hope on 'Diseases of the Heart,' p. 74.
[2] Flint on 'Diseases of the Heart,' p. 239.

never menstruated; there was no history of rheumatism. She had had a bad attack of scarlatina when a child, and had always been of a very sickly constitution. She was cyanosed, and breathed with great difficulty, but nevertheless she used to attend as an out-patient and continued able to go out until within a comparatively short period before her death. The symptoms noted were as follows. She complained of palpitation of the heart, difficulty of breathing, and cough, and she also suffered from copious hæmoptysis. These conditions were combated with more or less success by the usual remedies, including digitalis. The examination of the chest gave the following results.

The heart was distinctly seen beating through the emaciated chest walls, and it was felt beating much more strongly than naturally, and communicated to the hand the *frémissement cataire*. The area of præcordial dulness, however, was not unusually great. Over the apex of the heart there was a well-marked blowing systolic murmur, heard also distinctly over the angle of the left scapula. There was in addition a loud systolic sawing murmur over the base of the heart, and a diastolic one. Over the lungs, and especially the left, there was a well-marked and very extensive muco-crepitant rhonchus. The diagnosis given was hypertrophy of the heart, regurgitant disease of the mitral valves, obstructive and regurgitant disease of the aortic valves, and broncho-pneumonia. In the early part of the month of April she became worse, the difficulty of breathing increased, anasarca supervened, and she died on the 17th of April.

Post-mortem Examination.—The body was emaciated. On removing the sternum the heart was seen to be not very much enlarged; there were strong and extensive adhesions on both sides of the chest, and abundant false membranes were found in both pleuræ. The pericardium was not at all adherent, and it contained a moderate quantity (perhaps half a teacupful) of thin, straw-coloured serous fluid. The heart was hypertrophied, all parts being thickened, and there were clots, partly fibrinous, in all the cavities. The heart weighed 8½ oz. after all the blood and clots had been removed.

The right auricle was thickened, as was also the right ventricle, the thickness of the latter being ¾ of an inch. The tricuspid valve was extensively diseased, containing large masses of cartilaginous-like matter, and the carneæ columnæ of this ventricle were very thick. The pulmonary semilunar valves were also much diseased and contained an abundant deposit of the same cartilaginous-like matter as

the tricuspid valve. The left ventricle was also thickened, but not so much so as the right, its thickness being $\frac{5}{8}$ of an inch. The mitral valve was diseased, presenting the same irregular cartilaginous-like nodular masses as the tricuspid valve, but not so extensively. The aortic valves were examined as to their sufficiency by the water-test, and they were found insufficient, and on further examination they were found to be partially hardened, the corpora arantii being thickened, and the attached bases of the valves being stiff; but these valves were certainly not nearly so much diseased as the pulmonary semilunar valves.

The lungs were extensively diseased, the left lung being the most so. This was throughout dense and hard, the hardness in many parts being of a fibroid character. There were numerous *vomicæ* throughout the organ, but more abundant about the middle than at the apex. The right lung was similarly diseased, but not quite so extensively. It might be said that the lungs presented a mixed appearance of pneumonia (in the second stage) with tuberculosis, fibroid degeneration, and cavities containing pus. The immediate cause of death was evidently the extensive disease of the lungs, for the left one was quite useless as an organ of respiration, being uniformly condensed by pneumonia and tuberculosis, and the right was nearly so.

Remarks.—This case presents many curious features and the pathology, or rather the pathogeny, is obscure. The diagnosis during life was partially incorrect, or rather it should be said, it was imperfect, but still it is not easy to show how it could have been otherwise. The disease of the heart was not rheumatic in its origin, and certainly there had been no pericarditis. The most extensive disease was on the *right side* of the heart, and whereas the relative normal thickness of the right to the left ventricle in an adult is as $\frac{1}{3}$ to 1, in this case the relative thickness was three fourths of an inch to five eighths of an inch, both ventricles, however, being thickened, for it must be remembered that the patient was a feeble, emaciated, undergrown, and undeveloped girl. The murmur heard over the apex of the heart (and heard also distinctly and repeatedly over the angle of the left scapula) was of course attributed to insufficiency of the mitral valve, and the disease of the tricuspid valves was masked. The loud double sawing murmur over the base of the heart was also attributed to disease of the aortic valves, the morbid condition of the pulmonary valves (from the great infrequency of

its occurrence) being overlooked. I regarded the diseased conditions as comprising mitral and aortic disease, and, as a consequence of these lesions, broncho-pneumonia and pulmonary apoplexy; but the chief disease was in the right side, and was probably congenital.

Diseases of the Aortic Valves.—The aortic valves are very frequently diseased, and the physical signs of obstruction and regurgitation are alike easily appreciable by the auscultator's ear. The obstructive and the regurgitant murmurs may both exist, or one may exist without the other, but it is more common for the obstructive murmur to exist without the regurgitant than *vice versâ*.

Obstruction or *Constriction* or *Stenosis* of the aortic orifice is indicated by a systolic murmur heard over the situation of the base of the heart, namely, the upper part of the sternum corresponding to the second rib, and extending to the middle of the sternum, but disappearing, and at length becoming inaudible in proceeding towards the apex of the heart. The absence of murmur at the apex indicates that the mitral valve is free from disease; but while the murmur of aortic obstruction is not prolonged downwards towards the ensiform cartilage, it is prolonged upwards along the course of the ascending and transverse portions of the aorta and into the subclavian and carotid arteries, and this prolongation of the murmur affords, in fact, a valuable corroborative proof that the disease really exists in the aortic valves. In the murmur attending pericarditis the sound is not prolonged into the arteries, and thus a diagnostic character is established between acute inflammation of the pericardium and acute inflammation of the aortic valves. An aortic systolic murmur, then, indicates obstruction of the aortic orifice, the obstruction being either in the form of deposits of lymph upon the surface of the valves, or of fibrous matter in their interior, or of calcareous or ossiform metamorphosis of their whole structure. It is unnecessary to describe all the various conditions which may constitute stenosis of these valves, but the enormous amount of calcareous or ossiform deposit which they may contain, and without causing any very material symptoms, or indeed any at all, during life, has been exemplified in several cases recorded in this work. (See pp. 104, 105, and 107.)

The pulse in aortic obstruction is usually regular, and, unless the constriction is considerable, is not materially affected in other

respects. But when the orifice is much narrowed and there is at the same time hypertrophy of the left ventricle, the pulse is hard and wiry; and again, when the obstruction is so great as to close almost completely the channel for the passage of the blood, the pulse becomes weak and small. The lungs are not specially affected in constrictive disease of the aortic orifice, and the capillary system does not exhibit indications of congestion, nor is dropsy a common complication even when the orifice is very much contracted.

Regurgitation through the aortic valves, or *insufficiency* of these valves, is indicated by a diastolic murmur heard over the base of the heart. The causes of this regurgitation are analogous to, though by no means identical with, those described as causing regurgitation through the auriculo-ventricular orifices. The lesions of the aortic valves may result either from disease, as from acute rheumatism and endocarditis; or from fibrous, atheromatous, or osseous degeneration; or they may consist in some accidental injury of the valves, as laceration, in consequence of some strong muscular effort. According to Dr Walshe, aortic regurgitation is the most dangerous form of valvular disease, and the most likely to cause sudden death, and that physician adduces a remarkable fact from his own practice, in connexion with Life Assurance, in corroboration of this view.[1] I cannot either confirm or contradict this opinion, but I lately had a remarkable case presented to me for examination for Life Assurance, where the subject appeared in perfectly good health, and had never suffered from any illness, but in whom there was a distinct diastolic murmur, unaccompanied with any systolic murmur, over the base of the heart. I lately also examined a female child, who appeared in perfectly good health, and who had not suffered from any rheumatic attack, and who presented the same peculiarity. In both these cases the lesion was probably of traumatic origin, or in the child it might perhaps have been congenital. I fully concur with Dr Walshe in regarding this murmur as an indication full of the gravest significance.

The pulse in aortic regurgitation has been previously described as being peculiar and characteristic. It is a jerking pulse, or *the pulse of unfilled arteries*, the arterial stream being interrupted in its onward passage, and part of the blood being thrown backwards, the pulsation being thus made double, or *dicrotous*, and the throbbing of the radial and other superficial arteries being generally

[1] Walshe on 'Diseases of the Heart,' third edition, p. 391.

visible to the eye as well as palpable to the touch. The other effects of aortic regurgitation are not very remarkable, nor is the capillary system much affected in this disease.

Diseases of the Pulmonary Valves.—It has already been mentioned that diseases of the right side of the heart are much more rare than those of the left, and diseases of the pulmonary semilunar valves are still more rare than those of the tricuspid valve. Moreover, even when disease of the pulmonary valves does exist, its diagnosis is, even according to the most experienced stethoscopists, a matter of great difficulty. Dr Hope states that a pulmonary may be distinguished from an aortic constrictive murmur by the former appearing to be closer to the ear than the latter, the anatomical position of the two arteries being the reason of the difference.

But, besides this distinction, the pulmonary murmur is heard most clearly in the second intercostal space on the left side of the sternum. Like the other murmur it may be obstructive or regurgitant, according to the condition of stenosis or insufficiency of the pulmonary valves, and it may be single or double, as one or other condition occurs together or separately. The obstructive murmur is not transmitted to the carotid arteries, and is thus distinguished from the corresponding aortic murmur; but there is no certain method of distinguishing a pulmonic regurgitant murmur from an aortic one, unless there be both obstructive and regurgitant pulmonic murmur. Dr Austin Flint relates a curious case in which, during life, there was a murmur between the third and fifth rib on the left side of the sternum, but none at the apex of the heart, or at the base on the right side of the sternum, and no murmur in the carotids. But on post-mortem examination all the valves were found healthy, and the explanation of the murmur was found in the existence of a cavity between the pulmonary artery and the aorta, with an opening of the size of a crow-quill into the right ventricle just below the pulmonary valves.[1]

Disease of the pulmonary valves is generally congenital, and in a case previously described by me it was in all probability of that nature. It is usually accompanied by hypertrophy of the right ventricle, as in the case to which I allude.

[1] Flint on 'Disease of the Heart,' p. 242.

CHAPTER XVI

TREATMENT OF VALVULAR DISEASE

THE treatment of valvular disease is a very difficult problem for the physician in consequence of the insidious manner in which the lesion very often becomes developed, and also from the impossibility of reaching directly the seat of the malady by any known curative agents. When valvular disease has once become established it is by no means certain that art has the power to remove it, and yet cases are on record which encourage us to believe that a cure may and does take place in many instances where the valves are very seriously affected. It ought always to be the aim of the physician to assist the restorative powers of nature, which, in the disease in question, may possibly lead to a complete cure; or at least to favour those conditions which contribute to bring about such compensatory changes in the condition of the heart as may render the original lesion comparatively innocuous to life. As valvular disease, in its acute form, is most frequently associated with or preceded by acute rheumatism, it is obvious that the remedial measures tending to mitigate or remove the rheumatic affection will also tend to alleviate or remove the valvular lesions. Moreover, as prevention is better than cure, it ought to be a main object of treatment in acute rheumatism to prevent the supervention of valvular disease of the heart, and it would really appear that that object is now at least partially attained by the improved methods at present employed in treating the former malady. Judging by the statistics furnished on a large scale at many public hospitals, it may be stated that not only are the valvular diseases of the heart more successfully treated than they formerly were, but that they are very often prevented from being developed at all. Not only is the mortality from heart-disease in connexion with acute rheumatism very considerably diminished in the present day, but the number of

heart diseases in comparison with the number of cases of acute rheumatism is greatly reduced. The discontinuance of large bleedings in the treatment of acute rheumatism has in all probability tended to diminish the relative proportion of the fibrine in the blood, and thus to obviate the deposition of this principle in and about the cardiac valves; while the general employment of the alkalies, especially potash and its salts, has served not only to neutralise the acid present in the rheumatic diathesis, but to dissolve and disintegrate the fibrine, the excessive production of which is promoted by the same diathesis.

When, however, the valvular disease is established, whether it be in consequence of the operation of the rheumatic poison, or from some other constitutional peculiarity, the symptoms often become so urgent as to demand the utmost care in their management. It has already been shown that valvular diseases are usually accompanied by hypertrophy and dilatation, and that this complication is by no means to be regarded as necessarily an unfavorable feature in any given case, but rather as a compensation provided by nature to counteract the disturbances in the circulation caused by the stenosis or the insufficiency of the valves. Still, when such a complication exists, the patient may and does often suffer very considerable inconvenience, and the resources of medical art are available for his relief. It is not only in the administration of medicines that the therapeutics of valvular disease consist, but also in the due management of the regimen and the diet, which contributes most materially to control the course of the disease, and to alleviate the sufferings of the patient.

All powerful exercises should be avoided, such as running, rowing, riding, dancing, &c., all of which are likely to induce difficulty of breathing; and in cases where dilatation predominates over hypertrophy, and where, in consequence, the compensatory power is wanting to counteract the valvular defect, even slight movements of the body may cause great inconvenience such as palpitation and giddiness. The diet should be light and nutritious, consisting of soup, fish, eggs, poultry, and game; and alcoholic drinks, if taken in moderation, are rather beneficial than otherwise. Light wines, such as those of France and Germany, or dry sherry, may be allowed; a little bitter ale may also be recommended, and spirituous liquors are occasionally permissible in consequence of the tendency to faintness which sometimes exists.

The principal medicine employed in the treatment of valvular disease of the heart is Digitalis. But as this drug is extensively employed not only in this but in many other cardiac affections, and as its exact *modus operandi* is, even now, not fully understood, it appears necessary in this place to enter into some detail with regard to its peculiar action as a remedy in heart-disease. Its beneficial effect in many cases is indisputable, and its injurious operation, although no doubt occasionally observed, has been enormously exaggerated: but it is still an important question for the practitioner to determine what are the cases most suitable for its administration, and under what circumstances it is contraindicated.

The opinions of scientific writers on therapeutics have long been divided as to the mode by which digitalis affects the heart, and as to the causes to which its remedial powers in heart-disease are to be attributed. The view formerly entertained as to its action was that the drug was wholly sedative; that it reduced the pulsations of the heart when they were too strong; and that, on the other hand, in enfeebled conditions of the organ its use ought to be altogether avoided. That digitalis is a poison in large doses is unquestionable, but such enormous quantities have lately been given in certain cases without any injurious results, that one might be tempted to ask whether the goodness of the drug was sufficiently tested in all the experiments made; or whether certain conditions of disease might not cause resistance to its action; or whether the recorded effects were not perhaps due to some inexplicable idiopathic peculiarity of the patients. However this may be, there is no doubt that digitalis is now prescribed much more boldly than it was in former times, and that the consequences, on the whole, have been beneficial rather than injurious. I recollect some years ago attending a private patient in consultation with a physician now deceased, the case being that of a lady with feeble and irregular action of the heart, for which we prescribed digitalis and squills, in doses of one grain of each, both in powder, and the patient, who had, however, long been declining, died rather suddenly. In thinking over the case on many occasions afterwards, I could not help fearing that we had been wrong in administering digitalis in such a case, and that the fatal result might be in some way due to its employment. But subsequent reflection has induced me to believe that the drug had no effect in causing the death of the patient, and I am inclined to endorse the opinion expressed by some distinguished physicians, who

wrote in the 'Medical Times and Gazette,' July, 1865, that persons with fatty or otherwise degenerate hearts "are liable to die suddenly, and will do so now and then, whether digitalis be given or not." Still I think it right to mention my case, not as a warning against the use of digitalis, but as a caution that the disease for which the medicine is prescribed is often a fatal one, that it often kills suddenly, and that the result should be attributed to the malady and not to the drug.

The following remarks on the physiological action of digitalis must be received, as they are offered, with the necessary drawback that all speculations on the exact effect of drugs upon the vital organs must always be unsatisfactory to a certain extent. For although it may be asserted that such and such a drug acts upon the heart, and another upon the brain, or another upon the kidney, and so on, and although experiment may seem to bear out the assertion, yet we are very far from knowing, after all, *how* a particular foreign substance taken into the system acts upon a particular living organ, and by what media it effects its operations; and even if the medium or media be guessed at or discovered, what is the relation set up between the agent and the part acted upon, so as to produce the resulting effect.

Dr Headland, whose opinions on the action of medicines is entitled to great weight, conceives that digitalis depresses the action of the heart in consequence of its action on the pneumo-gastric nerve, and he quotes some experiments performed on the lower animals by Weber, who found that when the function of this nerve was disturbed by mechanical irritation, the rate of the heart's action was first accelerated, then retarded and rendered irregular, and finally the action was arrested.[1] The effects thus produced are thought by Dr Headland to resemble those caused by the administration of digitalis in certain doses, and hence he forms his opinion that the drug acts through the medium of the pneumo-gastric nerve. But the same opinion has been expressed by Traube, although his reasoning is rather different. The latter seems to regard the nerve in question as being essential to the regular action of the heart, for when both vagi were cut, tumultuous and irregular action of the organ was the result. Digitalis acts on the heart through the medium of the nerve in question, and, according to some recent experiments, it appeared

[1] 'On the Action of Medicines in the System.' By Dr F. W. Headland, 1867, p. 291.

that digitalis did not act so rapidly in animals where the vagi were divided.

Traube's reasoning seems to be that the pneumo-gastric nerve regulates or controls the action of the heart, and that digitalis, when administered medicinally, assists it in so doing; that when the nerve is divided, the action of digitalis is impaired; and that, therefore, digitalis acts on the heart through the medium of the pneumo-gastric nerve.

It would appear, from the present state of physiological and therapeutical science, that digitalis acts upon the sympathetic and vaso-motor system of nerves, and through them on the heart and the vascular system generally, but that it also acts upon the pneumo-gastric. The exact way in which it acts is, however, at present problematical, and the experiments made by various observers are so contradictory, that it is quite impossible to lay down at present any general results which are worthy of acceptance. In an able essay on 'Digitalis,' by Dr J. M. Fothergill, which received the Hastings prize of the British Medical Association in 1870, the author relates the results of a number of experiments made by Dybkowsky, Pellikan, and himself, as to the effect of digitalis on the heart and the vessels. It was found that when the heart of a living frog was laid bare by removing the walls of the chest and opening the pericardium, the contractions of the organ became accelerated, and more and more complete, until at last the ventricle came to a standstill in firm contraction. But in order to show that this effect was produced upon the nerves of the heart themselves, and not upon the trunks from which they are derived, Eulenberg and Ehrenhaus took out the heart of a frog and dipped the apex into a solution of digitalin, when the rhythmical action which normally goes on after death in a cold-blooded animal's heart under the influence of the cardiac ganglia, was rendered slow and irregular or was stopped altogether. But it was also observed by M. Malan and by Dr Fothergill that the direct application of infusion of digitalis to a frog's foot produced contraction of the vessels of the web, and as these vessels are under the control of the vaso-motor system of nerves it follows that the drug must act through the medium of these nerves. From these and other reasons which it would be inopportune to adduce in the present place, it may, I think, be inferred that digitalis really acts on the vaso-motor or sympathetic system of nerves.

But it also appears from the experiments just alluded to that the effect of digitalis is to cause *contraction* of the muscular fibres of the heart and the vessels, and this result is a most important one in a practical point of view, and it is the more necessary to allude to it particularly, because it is in opposition to, or at least not in accordance with, the views formerly entertained in reference to the action of digitalis, and its special uses in cardiac disease. In experiments related by Dr Fothergill, it was shown that in fishes and birds subjected to the action of digitalis, the heart after death was found firmly contracted, and it may be mentioned incidentally that aconite produces a precisely opposite effect, for when the tincture of this plant was administered to a sparrow, the heart after death was found paralysed and distended.

Assuming, then, as a fact that digitalis, whether applied directly to the heart and the vessels, or administered by the stomach, causes contraction of the muscular fibres of the heart and of the vascular system, it is not very difficult to explain the action of the drug in various and often opposite morbid conditions. In a heart which is free from functional or organic disease, the beats of the organ are performed at regular intervals, with a certain and definite amount of force, and, what is a point to be carefully noted, without the particular cognisance of the subject. But when the heart becomes diseased, either functionally or structurally, then the beats are often irregular, the force of the heart's action is insufficient, although perhaps it may be exaggerated, and the patient becomes conscious of the overtaxation to which the organ is subjected. In other words, palpitation and irregularity are characteristic symptoms of cardiac disorder. Palpitation, instead of being due to increased power of the heart's action, denotes failure of power; and irregularity in rhythm is also an evidence of debility. Intermittency, although sometimes a symptom of heart-disease, is not necessarily so, as has been previously explained (see p. 82), and it may be due only to some nervous condition which in the present state of our knowledge it is often difficult to explain.

Palpitation indicates laborious action of the heart, but not a stronger action; as the case is put very aptly by Dr Fothergill: "It is the evidence of effort, not of capacity. It is barren in result—a laborious stroke in place of a normal one, but not of more effect, not always of so much."[1] When digitalis is adminis-

[1] 'Digitalis, its Mode of Action and its Use,' p. 13.

tered in such a condition, the contractions are rendered more complete and more regular, the heart therefore appears to be tranquillised, although its impulse is really stronger, and hence digitalis was, and is still, supposed to act as a sedative upon it and to lower its action. But palpitation may occur in very different or opposite pathological states of the heart, namely, when its walls are attenuated, or even when they are normal, or when they are hypertrophied. Now, hypertrophy was and is believed to produce increased force, which digitalis was once supposed to diminish, and to this effect was attributed the beneficial influence of the drug in hypertrophy; but the explanation now given is that hypertrophy, although exercising increased force, does not exercise *enough* of such force in the morbid states in which hypertrophy is set up, and palpitation indicates not *excess* of power, but that the power is still insufficient. Digitalis, in such cases, co-operates with the hypertrophy, and renders the latter efficient. For it must be remembered that hypertrophy is usually a compensatory condition set up by nature to overcome some obstacle opposed to the circulation of the blood, and when the hypertrophy is insufficient for this purpose, then palpitation is the index of the insufficiency, and digitalis assists to render the hypertrophy adequate to its object, palpitation is allayed, and the heart's action becomes regular.

But whatever may be the theoretical opinions as to the physiological action of digitalis upon the heart, there can be no doubt whatever, arguing even upon empirical grounds, that the drug possesses a wonderful power of *regulating* the action of the organ, apparently strengthening it when its force is insufficient, or diminishing its activity when this appears excessive. These apparently contradictory, though equally beneficial, results have repeatedly been observed, and have been verified by daily clinical observation. Hence the drug is equally useful in nervous palpitation, when the organ is weak, and in the palpitation accompanying hypertrophy, when the same organ is (at least in appearance) very strong. These apparently paradoxical results certainly receive a happy explanation on the supposition that digitalis causes contraction of the muscular fibres, and thus equalises the movements of the heart and other parts of the circulatory apparatus.

Hypertrophy and dilatation of the heart are usually the consequences of valvular disease, and hence the treatment of all these morbid conditions is to be considered collectively. The obstruction

offered to the current of the blood through the thickened or rigid valves must be met by increased power in the walls of the heart to drive the blood through the narrowed or obstructed orifices, and hence arises hypertrophy, just as the presence of a stricture in the urethra causing obstruction to the flow of the urine is met by hypertrophy of the muscular walls of the bladder. Again, in cases when the valves are insufficiently closed, the blood regurgitates into the cavity from which it ought to be wholly expelled, and there meeting with the other portion of blood which the cavity ought normally to contain, the two portions together are now in excess in proportion to the capacity of the cavity, and hence the latter is enlarged in order to accommodate the increased amount of blood, or in other words dilatation occurs. But as simple dilatation must necessarily be attended with attenuation of the walls of the heart, the latter must be thickened in order to propel the increased amount of blood, and hence the combined conditions of hypertrophy and dilatation, the former acting as the corrective of the latter. Now, digitalis co-operates with hypertrophy in assisting the driving power of the heart, and in dilatation it acts in a similar manner, though not so successfully, because the walls in simple dilatation are attenuated. But a comparatively small dose of digitalis is required in hypertrophy, because the drug is *assisting* nature in restoring the balance of the circulation; while a much larger dose is tolerated and required in dilatation, because here the medicine is *counteracting* the feebleness of action due to the attenuated walls. When the dilatation is accompanied by hypertrophy, nature is keeping up the balance, and a medium dose only of digitalis is required. According to Dr Fothergill's views, in which I do not altogether coincide, in dilatation not counteracted by compensatory hypertrophy, and where the muscular walls are therefore unable to induce a regular tonic contraction of the heart, the use of digitalis may be continued, not only without danger but with decided benefit, for an indefinite period; and indeed, without its use, or in the absence of compensatory hypertrophy, the dilatation would continue to become more and more extensive till the heart is unable to propel the blood at all.

For the converse reason the long-continued use of digitalis in hypertrophy must be injurious, because, as Dr Fothergill suggests, the danger now is that the ventricular contractions may become excessive from the combined effect of the increased nutrition of the

muscular walls and the physiological action of the drug on the muscular fibres. In pure uncomplicated hypertrophy, therefore, the use of digitalis is unnecessary, as Niemeyer (quoted by Dr Fothergill) has stated, and it is required only in those cases where the hypertrophy, although distinctly present, is unequal to the increased duty, and where the over-taxation of the heart is shown by palpitation and other morbid indications.

The use of digitalis, therefore, in valvular disease of the heart, is explained by its effect in aiding the compensatory condition of hypertrophy or in counteracting the weakening tendency of dilatation, but it is necessary to allude more particularly to the special uses of digitalis in the diseases of each valve respectively.

In *stenosis*, or obstruction of the mitral valve, the blood is unable to enter the left ventricle, and is thereby driven back upon the lungs and the right ventricle, the walls of which have thus an additional strain imposed upon them and require compensatory hypertrophy to supply the demand. In such a case digitalis acts beneficially in assisting the driving power of the heart and especially the right ventricle, which, acting *à tergo*, propels the blood into the lungs, and thence to the left side of the heart.

In *mitral insufficiency*, causing regurgitation into the left auricle, the lungs are also congested by the reflux of the blood, and this reflux propagated from the lungs to the right ventricle distends the latter cavity, and makes a correspondingly greater demand upon the driving power of its walls. The compensatory power for mitral regurgitation is hypertrophy of the right ventricle, and it is by assisting this compensatory power that digitalis becomes useful.

In *aortic stenosis* the compensatory power is obtained by the thickening of the walls of the left ventricle, and in the absence of any disease of the mitral valve, the hypertrophy is capable of counteracting the obstructive tendency of the stenosis. No interference by medicines is therefore necessary in a great number of cases, because nature herself is doing what art would endeavour to accomplish, and it is only when the hypertrophy is inadequate to its task that digitalis may be administered as an adjuvant. Small doses of digitalis will therefore suffice for this purpose, as large ones might cause over-contraction. But, according to Dr Fothergill, whose ingenious views on this subject I do not altogether endorse, the effect of digitalis would depend largely on the condition of the muscular fibre. "If there were only a passing disturbance, and the

muscular fibre were fairly structurally sound, small doses alone would be required; if the hypertrophy were passing into degeneration, much larger doses would be called for."[1] Dr Fothergill conceives that the efficacy of digitalis in degenerated conditions of the muscular fibres depends on the fact that the drug acts more powerfully upon those fibres which remain healthy, and thus compensates for the deficiency of those which have become degenerated.

In aortic regurgitation, the efficiency of digitalis, or indeed of any remedial measure, is doubtful. I am not aware of any clinical experience bearing on this point, but Dr. Fothergill endeavours to show, on theoretical grounds, that in this condition the administration of digitalis is hazardous. He argues that in this form of valvular disease the left ventricle necessarily becomes both enlarged and thickened; that by the increased quantity of blood thrown by each ventricular contraction into the aorta endo-arteritis and disease of the arterial walls are set up; and that by the diminution of the flow of blood into the coronary arteries, the nutrition of the heart itself is arrested, the hypertrophy of the walls passes into degeneration, and thus their compensatory usefulness is lost or impaired. The administration of digitalis is dangerous, because it may drive the ventricle into a state of contraction which may never be relaxed, and thus bring the heart to standstill in systole. These views, however, must be tested by experience before they can be accepted.

Tricuspid regurgitation is very often associated with, if not caused by, dilatation of the right ventricle, and as there is no compensatory change behind to counteract the insufficiency, it is impossible that digitalis can be of any service in such a condition. Dr Fothergill states that in one case where it was tried, it was of no apparent use, although given unsparingly.

In *tricuspid obstruction* there is no experience to justify or contra-indicate the use of digitalis, as the affection itself is very rare.

A very important therapeutical application of digitalis in valvular disease of the heart is derived from the well-known *diuretic* action of the drug, which renders it extremely useful in relieving the dropsy very often existing as a consequence of the valvular lesion. The exact operation of digitalis in increasing the flow of water from the kidneys is not yet fully explained, and some of the results of experiment and clinical observation are somewhat paradoxical. In

[1] Op. citato, p. 50.

opposition to what might be regarded as the physiological action of the drug, it appears that when administered to persons in health it does not increase the flow of urine, and when given in poisonous doses the amount of urine is actually diminished, as has been shown by Dr Brunton in experiments made on himself, and even total suppression has been observed in cases of poisoning by the drug. These results are entirely in opposition to the views generally entertained, but they must be received with respect as being recorded by trustworthy observers.

It is not at all unusual, however, for a drug which possesses some peculiar property when acting upon a healthy body, to exercise some very different or contrary action on the body in a state of disease, and it is a perfectly well-known fact that the secretion of urine is increased by the use of digitalis in cases of dropsy. The explanation of its *modus operandi* is, however, by no means easy, for on theoretical grounds it ought rather to diminish the urinary excretion than to increase it. The flow of urine in health is undoubtedly increased by the operation of all those causes which, by paralysing the vaso-motor system of nerves, and thereby impairing the inhibitory action on the capillaries of the kidney, allow of the passive transudation of the watery parts of the blood into the urinary tubules. Now, digitalis is proved, by direct experiment, to contract the small vessels, and thereby, as one would suppose, to increase the inhibitory power, and we find that, according to the experiments of Brunton and others, digitalis actually causes suppression or diminution of the urinary secretion in certain cases. But then comes the question, why the same drug acts as a diuretic in dropsy? I think the answer probably is that it does not really act as a diuretic at all, but that, especially in the case of cardiac dropsy, it so regulates the contractile powers of the muscular fibres of the heart as to propel the blood uniformly through the different cavities of the organ, and thereby to relieve the venous congestion *à tergo* which, if unrelieved, eventuates in dropsy. The explanation given by Dr Fothergill as to the effect of digitalis on the urinary secretion, by supposing that the drug in some way acts on the Malpighian tufts of the kidney, I regard as quite unsatisfactory, for there seems to me to be no proof whatever that it does act on these structures, or, if it does, how it affects the urinary secretion.

In concluding these remarks on a very important drug, I should

observe that digitalis may be uncertain in its operation from a variety of causes which are independent of the powers it naturally possesses. The *Digitalis purpurea* is an indigenous plant, the leaves of which are gathered and dried and reduced to a powder, and afterwards converted into a tincture and an infusion; and it may happen that from want of care in the selection, or in the gathering and drying of the leaves, or from too long keeping, or even from exposure to the light, their efficacy may be more or less impaired. Hence it is necessary that great care should be taken in procuring pure and good specimens of the plant in the first instance, and that the preparations of the drug should be entrusted only to capable and responsible persons. Many of the anomalous effects of digitalis, and many of the disappointments attending its therapeutical employment, are in all probability due to the operation of such accidental causes as those just alluded to.

The dose of the tincture of digitalis is from ten to fifteen minims, given in some aromatic water, and repeated two or three times a day. Much larger doses have been given, and without any bad effects, but I have found the doses just mentioned quite sufficient to reduce and equalise the pulse in the cases of heart-disease for which the drug is appropriate. As digitalis possesses the peculiar property of accumulating silently in the system when given in moderate doses for a long period, and then at length producing toxical effects, it is necessary to watch rather carefully over its administration; but I confess that although I have very often prescribed digitalis, and have continued its use for considerable periods, I have never observed the poisonous effects described in works on toxicology, but which, no doubt, do occur in certain instances. Still, I am so fully impressed with the danger which might arise from the incautious continuance of the use of the drug that both in private and public practice I always give strict injunctions that the medicine should always be given under medical superintendence, and that it should by no means be employed by patients at their own discretion, or given at hospitals or dispensaries as a mere matter of routine. The dose of the decoction of digitalis is half an ounce. Digitalis may be combined with expectorants when the lungs are inflamed or congested; and with diuretics, especially the salts of potash, when dropsy supervenes upon heart-disease; and when the powers of the system are reduced, digitalis may be most usefully combined with iron.

It has repeatedly been stated in these pages that valvular disease of the heart very often causes little or no suffering to the patient, at least so far as its subjective symptoms are concerned, and therefore no remedial measures are sought for in many instances. Sometimes, however, there is no such immunity, and a variety of distressing sensations are present. These are usually connected with the respiratory functions, and consist of difficulty of breathing, tightness of the chest, and cough, sometimes attended with spitting of blood. Dyspeptic symptoms are also common, and flatulence, want of appetite, costiveness, and irregularity of the bowels, are very common concomitants. Many persons who complain only of dyspepsia are found, by accidental stethoscopic examination, to be really suffering from valvular disease of the heart. Sometimes, however, although rarely, the symptoms are really referred to the heart, and then they constitute the affection known as Angina Pectoris, which will be treated of in a subsequent chapter, and its treatment explained.

The valvular diseases of the heart are also often accompanied by appreciable disease of other organs, especially the lungs, and bronchitis, pneumonia, and apoplexy of the lungs, are very common complications of the cardiac malady, and must receive appropriate treatment.

But one of the most common of the concomitants or consequences of valvular disease of the heart is dropsy, the cause of which, as dependent on cardiac obstruction, has already been explained. This kind of dropsy, known as cardiac dropsy, to distinguish it from that which results from obstructions in the liver and the kidneys, is a very serious complication of heart-disease, and adds most materially to the gravity of the prognosis. Whatever I have advanced in preceding pages as to the rather favorable view which may sometimes be taken as to the chances of longevity or of immunity from serious danger, in cases of valvular or other organic disease of the heart, is wholly inapplicable if dropsy has once made its appearance.

Still, the condition of the patient may be alleviated, even under such circumstances, but the dropsy requires a suitable and special mode of treatment. The fluid accumulated in the areolar subcutaneous tissue, especially in the lower extremities, must be eliminated, if possible, and this object is to be accomplished by discharging it through some of the great outlets of the body. Supposing the kidneys

to remain healthy, the water may be voided through those organs by the aid of diuretics, and this, on the whole, is the best channel to be selected in cardiac dropsy. The use of digitalis as a diuretic has already been alluded to as promoting this object, and this drug indeed is eminently useful, especially in combination with well-known diuretics, as the salts of potash—the nitrate, the bicarbonate, the acetate; or with scoparium and juniper; or with the various preparations of squills—the tincture, the oxymel, the syrup, the pilula scillæ composita. The last-named drugs are especially useful when there exists, as there generally does, some pulmonary complication, and then hyoscyamus or conium (but not opium) may be advantageously combined with them.

Purgatives, to a certain extent, are admissible in cardiac dropsy, but the gentle ones are the best, because there is such a tendency to syncope in this affection that any violent cathartics might produce a fatal effect. Castor oil, therefore, or the pulvis jalapæ compositus, in moderate doses, or the saline purgatives, as Rochelle salt, or Epsom salts, are to be preferred to such drastic cathartics as gamboge, elaterium, scammony, colocynth, &c., which are available in some other forms of dropsy.

CHAPTER XVII

FATTY AND OTHER DEGENERATIONS OF THE HEART

THE first subject now introduced is one of the most important, but at the same time one of the most obscure, in the whole range of cardiac pathology and therapeutics. The presence of fat in or around the heart is a very common occurrence, but its significance or its danger depends precisely on the position which it occupies, and hence it is necessary to divide this pathological condition into two categories, namely (*a*), the excessive accumulation of normal fat in the subpericardial areolar tissue, and (*b*), the fatty degeneration of the muscular substance of the heart, to which latter, indeed, the term "fatty degeneration of the heart" ought exclusively to be applied.

1. *Fatty Accumulation around the Muscular Fibres.*—An excessive growth of the normal fat around the muscular structure of the heart is a condition generally unattended with any serious consequences, and it therefore gives rise to no remarkable symptoms, although it may be supposed that the pressure of the fat on the surface of the organ might give rise to an atrophy or disappearance of the muscular structure. But it also happens that when there is such a preternatural development of fat, the inter-muscular areolar tissue is infiltrated with the same material, and the energy of the heart's movements is more or less materially diminished. This fatty accumulation on the heart often occurs in persons who exhibit an abnormal tendency to the development of fat in all parts of the body, and also sometimes in individuals attacked with tuberculosis and carcinoma, and in drunkards, the parts chiefly affected being the apex and base of the organ, the longitudinal furrow, and the course of the coronary arteries.

It is unnecessary to describe the symptoms of this affection,

because the diagnosis is very obscure, and even if it were made, the knowledge is not very important, for, unless the accumulation of fat be very great indeed, no serious sufferings seem to result from its presence. Dr Walshe states that he has watched the symptoms of such a condition in only one case which he attended during life and examined after death, and in which the heart was loaded with sub-pericardial fat, and positively free from serious softening or notable amount of intra-sarcolemmous oil.[1] The patient was a male aged sixty-four, and the symptoms generally indicated weakness of the system, such as tendency to fainting, pain about the præcordial region, inclination to coldness of the extremities, occasional giddiness, feeble cardiac impulse, and inability to walk quickly: but the pulse was regular, and of medium frequency.

2. *Fatty Degeneration of the Substance of the Heart.*—This fatty degeneration or fatty metamorphosis of the heart is a far more serious affection than any that has hitherto been considered. In this disease the muscular fibrils are replaced by fat granules and globules, which, being at first few and solitary, at last become so numerous that they fill the sarcolemma entirely to the exclusion of the sarcous elements. Hence the colour of the muscular fibres is changed to a pale yellow, and their structure can be easily torn. This fatty degeneration occurs either in small circumscribed spots, or in larger portions of the muscular walls, generally in the left side of the heart, but sometimes invading the whole of the organ.

The most complete and earliest monograph on this subject appeared in the *Medico-Chirurgical Transactions* for the year 1850, in the shape of a communication from Dr Richard Quain, whose attention had been drawn to the investigation of the subject, some five years previously, by the sudden death of a gentleman in whose body no sufficient explanation of the fatal event could be found, except that a peculiar fatty condition of the heart was observed and recorded during the examination. After that event two similar cases presented themselves, and led Dr Quain to the conclusion that the presence of this fatty matter in the texture of the organ bore some important relations to its structure and functions.

As in the case of many other important discoveries and observations in pathology, it would appear that occasional references were made by some earlier writers, especially of the French school, to the

[1] Walshe on 'Diseases of the Heart,' fourth edition, p. 331.

probable existence of the morbid condition now under consideration, although the descriptions are generally deficient in clearness and circumstantiality. Corvisart, for instance, states that he had heard of fatty degeneration of the heart, although he had seen no cases himself, but Laennec not only saw but described the disease, and devoted a chapter in his 'Maladies de la Poitrine' to its description. He clearly described the distinction between mere fatty infiltration and fatty degeneration.

"La dégénération graisseuse du cœur est l'infiltration de la substance musculaire par une matière qui présente toutes les propriétés physiques et chimiques de la graisse; c'est une altération tout-à-fait semblable à la dégénération graisseuse que Haller et Vicq-d'Agyr ont observée dans les muscles."—Laennec, 'Traité de l'Auscultation, &c.,' vol. iii, p. 256. Laennec goes on to state that he has never met with this alteration except in a very small portion of the heart, and only towards the point. He expressly states that by the term fatty degeneration of the heart, he does not mean the same condition as that described by Corvisart, who evidently referred to ordinary deposition of fat in excess around the heart. Laennec, moreover, refers to cases in which it was thought that serious symptoms, or even sudden death, were caused by fatty deposition in or around the heart; but he gives no instances from his own experience, and he expressly states that he has never seen a rupture of the heart caused by this change, and he knows no symptoms which can be attributed to it.

Dr C. J. B. Williams, in 1840, stated that the fatty matter is deposited in, and not on, the muscle of the organ, which thereby becomes changed in nature and impaired in function;[1] and, in the same year, Dr Peacock communicated to Dr Quain the particulars of some cases of true fatty degeneration. In those cases the changes were most evident in the interior of the organ, the surface being destitute of fat, and it is almost unnecessary to state that this peculiar form of fatty degeneration has no relation or connexion with the ordinary deposition of fat in or about the general tissues of the body. In Dr Peacock's cases, microscopical examination showed the fibres of the heart almost or entirely deprived of their transverse striæ and covered with small oil-globules.

The late Dr Ormerod, of Brighton, whose name is also creditably associated with the development of the modern views as to the nature

[1] 'The Pathology and Diagnosis of the Diseases of the Chest,' 1840, p. 279.

and history of fatty disease of the heart, published a series of cases in 1849, which elucidated the circumstances under which the disease may occur, and Dr Quain, in the paper to which I have referred,[1] gives the abbreviated histories of thirty-three cases of the disease, in which there was fatty degeneration of the muscular tissue of the heart. He gives the histories, similarly abbreviated, of many other cases, in which there was excessive accumulation of fat on the surface of the heart, but these latter are excluded from the category.

Anatomical and Microscopical Characters.—The disease exists essentially in the muscular structure of the heart, and it is not necessarily connected with any enlargement of the organ, any valvular disease, or any disease of the coronary arteries. I do not assert that these are never coincident conditions, and indeed it might be supposed, arguing from analogy, that any or all of them might be the causes of fatty degeneration, but I strongly maintain that, practically speaking, they are not so.

The heart, then, may be natural in size, but its colour and consistence are changed, the colour being pale, but more generally of a yellowish-brown colour, something like a faded leaf, a resemblance first pointed out by Laennec; and the consistence is soft and flabby, so that the finger may be pushed easily through the texture, or the muscular structure may be torn asunder with a very slight effort. Dr Walshe[2] states that both ventricles are most commonly affected, the left being more frequently alone affected than the right alone; that the layers of substance immediately beneath the endocardium and pericardium generally, though not always, suffer earlier than the intervening strata; and that the auricles seem but little prone to this morbid change, although he has certainly observed it in the right auricle.

In advanced periods of the disease, the quantity of fat present is so considerable that the greasy appearance is visible to the naked eye, the scalpel used in dissection is greased, and so is blotting-paper when applied to the cut surface; but no reliance should be placed on any of these characters when determining the presence or absence of fatty degeneration, the characters of which are clearly distinguishable only by the microscope.

[1] 'Medico-Chirurgical Transactions,' vol. xv, 1850; Dr. Quain on "Fatty Diseases of the Heart."

[2] Walshe on 'Diseases of the Heart,' p. 334.

The ultimate fibres constituting the muscular structure of the healthy heart are enclosed in a delicate sheath, called the sarcolemma, and each fibre is marked by transverse bands which divide it into a row of somewhat square-looking portions called sarcous elements or *sarcode*.

Now, in fatty degeneration of the heart the transverse bands or *striæ* become obscure and ill-defined, and the *sarcode* is replaced by fat-granules and oil-globules, which are distinguished from ordinary oil-globules by being much smaller. These fat-granules and oil-globules are at first disposed in longitudinal rows, but they eventually lose all regularity of arrangement, and take the place of the sarcous elements by a gradual process of molecular transformation. Dr Walshe states that the addition of ether, which, of course, dissolves the oil, will exhibit portions of fibre still remaining intact, in spots where heaped-up granules had rendered them invisible. The diseased fibres are not notably, if at all, says the same authority, less in diameter than healthy ones. Oil is found also outside the sarcolemma, and, as a rule, this sheath continues discernible, and hence the outline of the fibre is preserved, but in extreme cases the sarcolemma itself disappears, although the exact cause of its disappearance is not clearly established.

In reference to the primary causes and pathological nature of fatty degeneration of the heart little certain is known, but perhaps it may be said that the same uncertainty exists as to the intimate essence of any morbid process whatever. We know disease only by its effects and visible changes, and its nature and origin are too often among the secrets we vainly attempt to penetrate. Dr Quain, however, intimates that the process which the muscular fibres of the heart undergo in fatty degeneration is identical with, or at least analogous to, the changes that muscular and other tissue sometimes presents out of the body. In other words, he thinks that when the protein compounds, albumen and fibrine, are effused in a form not readily susceptible of organisation, when even they enter into the composition of textures the organisation of which is imperfect from inherent depravity, from natural decay, and from other deteriorating causes, then those substances degenerate and pass into fatty matter. Albumen and fibrine, in fact, then yield to ordinary chemical influences, and undergo a species of degradation.[1]

[1] Quain on 'Fatty Diseases of the Heart,' op. cit., p. 145.

In partial confirmation of the view that the nutrition of the heart is impaired in fatty degeneration, and that the organ is unable to resist the influences tending to the disintegration of its tissue, Dr Quain reviews the circumstances, so far as they are known, under which the degeneration occurs. He shows, among other illustrations, that, out of twenty-five cases recorded by Dr Ormerod, three had suffered from hæmorrhage, three from phthisis, one from delirium tremens, one from cancer, and two from fever; and among his own cases, he shows that one was in a child with cancrum oris, another in a woman who had suffered from long-continued gastric irritation and chronic phthisis, and a third in a patient suffering from hæmaturia. Dr Quain also refers to ossification and obstruction of the coronary arteries as leading to localised or partial fatty degeneration, and he states that he has himself found more or less obstruction of these vessels in cases he has examined; and endo- and pericarditis are also supposed by him to contribute to the disease. In thirteen out of thirty-three cases, of which he gives the history, he found more or less ossification or obstruction of the coronary arteries; and he justly alludes to the similarity thus exhibited between softening of the brain and fatty degeneration of the heart, the arterial disease being apparently the cause of both.

If this view of the disease by Dr Quain had been confirmed by subsequent researches, the pathology of fatty degeneration of the heart would be clearly explained; but I doubt very much whether such confirmation has been afforded by recent post-mortem investigations.

Dr Quain suggests the similarity of fatty degeneration of the heart to the conversion of muscular fibre into adipocere, a change well known to result occasionally when dead animal tissues are exposed to moisture and excluded from the air; and he adduces an instance in which he observed a healthy heart, in his possession, to become changed into fat by immersion for a few weeks in weak spirit and water. On careful examination the specimen was found to exhibit the most advanced stage of fatty degeneration, differing only in one circumstance from what occurs during life, namely, that the change was universal. In degeneration occurring during life, many of the muscular fibres retain their healthy organisation.[1]

Symptoms and Physical Signs.—The symptoms and physical

[1] Op. citato, p. 143.

signs of this affection are alike obscure. The pulse, as might be expected, is weak, but the varieties of the pulse are so great, even in healthy individuals, that no diagnostic importance can be attached to this circumstance. Dr Flint states that remarkable slowness of the pulse has been observed in this disease, the beats having sometimes been so low as eight and nine in the minute, and he has himself observed a notable degree of capillary congestion of the extremities.[1] A tendency to syncope, difficulty of breathing, palpitation and oppression in the præcordial region, are all symptoms which might be expected, and do sometimes occur; but they are by no means constant in their appearance, or trustworthy as diagnostic signs. Dr Flint, after referring to other authors in proof that pseudo-apoplectic seizures sometimes denote fatty degeneration of the heart, alludes to two cases of his own which apparently confirmed this view.

Mr Canton, in some papers in the 'Lancet,' and afterwards in a monograph on the subject of the "Arcus Senilis, or Fatty Degeneration of the Cornea,"[2] regards this appearance as denoting the existence of fatty degeneration of internal organs, and he brings forward a number of instances from his own practice and that of others in confirmation of this view. But the results of subsequent experience have not proved that the arcus senilis invariably denotes fatty degeneration, although it may fairly form an element in a case where the diagnosis is doubtful.

The physical signs of fatty degeneration of the heart are not by any means well marked, and are all rather negative than positive. As the heart is not necessarily enlarged in this affection, there is no increased dulness on percussion, and, if there be no concomitant valvular disease, there are no murmurs audible on auscultation. The impulse of the heart is, of course, feeble, but mere feebleness of action, unaccompanied by any other positive sign, has very little diagnostic significance.

Treatment.—While the symptoms of fatty degeneration are so uncertain, the treatment must necessarily be rather unsatisfactory. If it could be asserted with confidence that the fatty deposition in the cardiac sarcolemma was an accompaniment or a consequence of fatty deposition in the general tissues of the body, it might be reasonably expected that a course of regimen calculated to diminish the amount

[1] Flint on 'Diseases of the Heart,' p. 100.

[2] 'Arcus Senilis,' by Edwin Canton, F.R.C.S., London, 1863.

of fat would be successful; but no such relation can be proved to exist, and lean persons as well as fat ones are equally subject to this mysterious malady. Still, if the disease be indicated by any of those symptoms or events which denote a failing action of the heart, such as occasional syncope, or dyspnœa after exertion, and all other organic disease of the heart be excluded by the absence of the appropriate physical signs, then such treatment should be adopted as is calculated to strengthen the weakened organ, and to prevent, if possible, the further deposition of oil-cells within the cardiac tissue.

All violent exercises should be forbidden, but at the same time passive locomotion, and living much in the open air, are to be recommended. Substances containing fatty matter or which are likely to develope it, such as all saccharine and starchy bodies, are to be avoided, and meat, eggs, milk, and other nitrogenous or flesh-forming foods, are to be preferred. If there should be pain or difficulty of breathing, it may be necessary to administer some sedative, such as opium, but I cannot agree with Dr Bellingham or Dr Quain in believing, as they do, that the abstraction of blood (although only locally) can be beneficial. When there is faintness, or syncope, stimulants should be administered either by the mouth or the nostrils, as, for instance, brandy by the former, and ammonia by the latter. A course of tonic medicine will be very beneficial, especially of iron, in combination with quinine, and Dr Law has recommended strychnia in small doses, and Dr Bellingham has used the extract of nux vomica, with the object of strengthening the weakened heart. Alcoholic liquids are used with advantage, and, indeed, they form an essential part of the treatment; and port wine, sherry, stout, bitter ale, are all serviceable. Chloroform is to be avoided, and it is a well-known axiom in operative surgery that this agent should not be administered as an anæsthetic in cases where fatty degeneration of the heart is known or even strongly suspected to exist.

Softening of the Heart.—This condition, although present in fatty degeneration of the organ, may exist independently of it. The heart may be so soft as to be readily perforated or torn by the finger, and yet no fatty degeneration may be discoverable. Softening may exist in connexion with dilatation of the heart, and it may also occur as an accompaniment or consequence of fever, as has been observed by Louis, in Paris, and Dr Stokes, in Dublin. The former physician appears to have first recognised softening of the heart as a

consequence of fever, and he published his observations on this point in his *Recherches sur la Gastro-entérite.* It is perhaps unnecessary to observe that the type of fever usually prevailing in Paris is what is now called *typhoid* or *enteric,* corresponding to the *gastro-entérite* of Louis; while that generally known in Ireland is the *typhus* or *maculated fever.* It may further be mentioned incidentally that the division of fevers into typhus and typhoid is not even yet generally admitted in Ireland. Nevertheless, it would seem that the softening of the heart now under consideration is the same as was observed by Louis, in Paris, and Dr Stokes, in Dublin, the latter fully admitting the truth of Louis' description, and moreover clearly pointing out the pathological characters of the heart in this affection.[1]

Both these physicians agree in considering the disease to be unconnected with any inflammatory process, and they do not regard it as necessarily associated even with fever, for it occurs in other weakened states of the system. The amount of softening of the heart in one instance is graphically depicted by Dr Stokes, who states that when the organ, removed after death, was grasped by the great vessels, and held with its apex pointing upwards, it fell down over the hand, covering it like the cap of a mushroom.[2]

The Dublin physician was not only the first to describe the physical signs denoting softening of the heart in fever, but also to indicate the therapeutical measures rendered necessary in such cases. The impulse of the heart is weakened or lost, especially at the apex, and the first sound is impaired or weakened, or lost altogether, the second sound remaining; and sometimes, though very rarely, the second sound is also lost, and then very extreme weakness of the heart is denoted. Dr Stokes relates a number of cases in which the diagnosis was made during life, and was confirmed by some of the post-mortem examinations; but he had also the satisfaction to observe that recovery took place in many instances where his practice of administering stimulants in such cases had been adopted.

Although the efficacy of Dr Stokes' plan is now generally admitted and acted upon, the time is not very far distant when such a mode of treatment would have been considered rash and unjustifiable. It is reasonable to suppose that, under the influence

[1] 'The Diseases of the Heart and the Aorta,' Dublin, 1854, p. 366.

[2] Op. citato, p. 373.

of nourishment and the use of stimulants, the weakened and softened heart may often regain its tone and repair its structure.

Of other forms of degeneration it is unnecessary to say much. Cartilaginous deposits have been occasionally, though very rarely, found in the muscular structure; and cancer of the heart is also very rare, though Dr C. J. B. Williams states that he saw the septum of the heart in one case converted into a substance which seemed to him to be of a carcinomatous nature.[1]

[1] 'The Pathology and Diagnosis of Diseases of the Chest,' by Dr. C. J. B. Williams, 1840, p. 279.

CHAPTER XVIII

FUNCTIONAL DISEASES OF THE HEART

THE functional diseases of the heart, as distinguished from the organic, although they convey no information to the morbid anatomist, are nevertheless full of interest to the pathologist and the practical physician. To the first, they are interesting because, in a broad view of pathology, impaired function is of equal importance with impaired structure; to the second, because they are met with in every-day practice, and among all classes of society. The female sex is more liable to them than the male because the organisation of the former is more delicate and excitable, and because the uterine functions play an important part in cardiac as in all other bodily derangements. The functional diseases of the heart are, moreover, far more common than the organic, and, what is a strange and somewhat unaccountable circumstance, they are usually attended with much more suffering to the patient. It may almost be said that if a patient complains very much of suffering about the heart, such an one is *not* the subject of organic disease; but still it must not be forgotten that functional disease may be added to the organic.

The distress and anxiety experienced by patients who are the subjects of functional disorders of the heart, are often of the most painful kind; the fear of death haunts them day and night, and it is always difficult, sometimes impossible, to persuade them that their malady is not really of a serious nature. In those who are suffering from organic disease, on the other hand, the subjective symptoms are often of a very insignificant character, or may sometimes be absent altogether, and even if the patients are informed that organic disease exists, the knowledge of the fact does not usually produce much impression. In many cases which are the subjects of organic disease, there are no rational heart-symptoms at all, and the complaints made are usually of dyspepsia; in one instance, that of a

young member of my own profession, a knowledge of heart-disease existed, but without producing any alarm or interfering with the usual pursuits of life. He himself informed me that he was the subject of mitral disease, and at his own request I examined his heart and found the statement to be too correct. I often saw him, not without a feeling of anxiety on my part, engaging in such active exercises as dancing in crowded rooms without inconvenience, and he married; but the nature of the complaint under which he was all the time labouring was at length fatally manifested. The examples of an opposite character, where patients only imagine themselves the subjects of heart-disease, and who request to be examined by the stethoscope, as if for the purpose of confirming their own painful convictions, are innumerable, and a record of such cases would be tedious and unprofitable.

A reference to the anatomy and physiology of the heart, particularly in connexion with its nervous supply, will in great measure, though by no means altogether, explain some of the phenomena of its functional disturbances. (See pp. 22 *et seq.*) It is found that the heart derives its nerves from the *sympathetic system*, so called, indeed, from its very general distribution, and the *sympathies* which it thus seems to establish between all parts and structures of our corporeal frame. The ganglia and the threads of this great nervous network, spreading from the brain to the heart and the lungs, to the stomach, the liver, and the spleen, to the intestines and to the bladder, to the arteries and the veins, and besides, in females (a most important fact) to the uterus and the ovaries, establish, as it were, a telegraphic communication between the most distant regions and structures, and cause the central organ of the circulation to sympathise in the remote sufferings or derangements of any part of the complicated apparatus. When, in addition to this general connexion with all parts of the body by the sympathetic system, it is recollected that the heart is also supplied by the pneumo-gastric nerve with filaments which bring it into special relation with the brain, the larynx, the lungs, and the stomach, it will be readily understood how the heart may *sympathetically* be deranged in its functions when any of those other important organs are disturbed in their operations either temporarily or otherwise.

Neuralgia.—It has already been mentioned that although the heart is abundantly supplied with nerves, its functions are performed in health without any pain, and indeed without any consciousness;

and it may be added that even severe injuries of the organ may be unattended with much sensibility. This fact has been proved by experiments on the lower animals, and by observation of the effects of wounds accidentally inflicted on the human subject. But although the sympathetic system of nerves is certainly not endowed with ordinary sensibility, and hence the natural functions of the organs it supplies are performed unconsciously, yet, as is well known, these very organs in disease are the seats of the most acute suffering. This paradox has been explained on the plausible hypothesis that the sensory nerves in passing through the sympathetic ganglia are paralysed or deadened, as it were, by the influence of the latter in a state of health; while in disease, the anæsthetic power (if it may be so called) of the ganglia is suspended, and the natural sensitiveness of the sensory nerves is developed. Be this as it may (*causa latet vis est notissima*), inflammation of the stomach, or the bowels, or the kidney is usually accompanied by the most acute suffering, and the pain is often as great or even greater when there is no inflammation at all, but where there is som muscular spasm, or some internal obstruction, as in the passage of gall-stones or of a renal calculus. Still, as if anomalies and paradoxes in physiology and pathology were illimitable, even inflammation is often unattended with pain, and mortal disease in vital organs may run a latent and insidious but fatal course. It has already been stated that inflammation affecting the membranes of the heart or the heart itself are no exception to the rule, or rather that they confirm it, and I have therefore strongly insisted upon the fallacious nature of the evidence to be expected from pain in the diagnosis of inflammatory affections of that organ.

But when there is no inflammation at all, and no organic disease, the pain experienced in the region of the heart is often excessive, and in fact constitutes the disease for which the patients apply for relief. This pain is to be distinguished from rheumatism of the intercostal muscles by its more deep-seated character, and by its not being aggravated by movements of the body; and from ordinary neuralgia, by the fact that the pain is rather relieved than aggravated on pressure.

Independently, however, of this painful condition, probably due to some affection of the sensory nerves which pass through the cardiac plexus, complaint is very often made, especially by females, of pain about the region of the heart, which to them appears to be the

chief seat of disease. But it will be found, in most cases, that the affection is only a sympathetic one, and is caused by some derangement of the stomach, or the bowels, and, in women, of the uterus, the derangements of which, and even the performance of its natural functions, are often the fertile sources of much cardiac suffering.

Palpitation.—Of all the disturbances of the heart's action, unassociated with organic disease, undoubtedly the most common is palpitation, which, however, it must not be forgotten, may also accompany structural mischief. But, putting the latter category of cases aside as having been already sufficiently described, palpitation may arise from very different or even opposite causes, or it may be impossible to connect it with any known cause at all. Thus it may be associated with a plethoric condition of the system, or with the opposite state, which latter is by far the more common; it may be due to dyspepsia in either sex, or to disordered menstrual function in the female, or to the presence of the gouty diathesis, which more usually exists in the male; and it is a prominent symptom in exophthalmic goitre, as will be hereafter more particularly explained.

Palpitation usually occurs in paroxysms, the patient being conscious of the violent and tumultuous beating of the heart, and this consciousness is one of the most distressing features of the affection, for the mind is constantly directed to the symptom, and the most gloomy forebodings are inspired or even cherished. It is in vain for the medical attendant, in many such cases, to attempt to dissipate the patient's fears, for the present suffering and uneasiness are so great that worse results are almost invariably anticipated, and as the palpitation very often comes on at night, the sufferer is worn out with want of rest in addition to his other causes of uneasiness. The malady, however, being paroxysmal, the suffering is relieved after a period of longer or shorter duration, but only to be renewed after an interval equally uncertain.

Many causes have been assigned for the production of this painful affection, amongst which may be mentioned mental emotion and excitement, a very common cause, especially in this active and enterprising age; the influence of the passions, especially that of love; the pursuit of study to an excessive degree, as in the candidates for University honours or for competitive examinations; the indulgence to excess in alcoholic drinks—a vice unfortunately too common throughout the civilised world; the use of tobacco, which induces palpitation of the heart in some constitutions; the consumption of

tea, especially green tea, a fact well ascertained, however difficult may be the explanation,—all these and many other such causes are fertile sources of the nervous disturbance which manifests itself in palpitation of the heart.

But in addition to all these causes, some of a physical and some of a psychical character, there are altered conditions of the blood which, in a remarkable manner, give rise to the affection now under consideration. Some of these conditions of the blood are difficult of recognition by chemical analysis, and are rather inferred to exist than actually proved; but others are denoted by well-known and appreciable tests. Among the most common in males are the cases where the blood is overloaded with lithic acid, constituting the gouty diathesis, in which palpitation of the heart is a common symptom, disappearing, however, when the paroxysm of gout has been fully developed. A still more common cause, in females especially, is the condition of the blood known as anæmia (or as it ought to be termed spanæmia), where the red corpuscles are deficient and the water is in excess. In this affection, as in the cognate case of chlorosis (for chlorosis and anæmia or spanæmia are by no means interchangeable terms), palpitation is a very common and a very prominent symptom.

Diagnosis.—The diagnosis between the affections just referred to, and the organic diseases of the heart, mainly depends upon the physical signs developed by auscultation and percussion. In nervous palpitation there is no enlargement of the heart, and consequently the area of cardiac dulness is not extended, and valuable evidence, though of a negative character, is thus obtained. Inspection of the thorax gives no very trustworthy indication, because the pulsations of the heart in a thin subject may be visible and well marked, whether the disease be functional or organic; but palpation affords much more valuable characters. It may be stated, as a general rule, that the *frémissement cataire,* so often previously alluded to, and which usually denotes a certain amount of hypertrophy of the heart combined with an unfilled condition of the cavities, is always absent in mere functional disturbance, except perhaps in those very rare cases where this peculiar thrill may have been caused by repeated bleedings.

The difference beeween the palpitation caused by organic disease and that due to nervous disturbance has already been fully pointed out, and it is unnecessary to recapitulate the distinctive characters. But the auscultatory phenomena deserve the closest attention,

because, even with our advanced knowledge of the indications afforded by the stethescope, it is not by any means easy in some cases to distinguish between the sounds caused by structural mischief and those produced by altered conditions of the blood. It has been already pointed out (p. 59) that the *murmurs* or morbid sounds of the heart are all due to *friction* of some kind, either of one solid part against another, or of healthy blood against unhealthy valves, or of unhealthy blood against healthy valves, or perhaps (though this is not so certain) by the mere collision of the particles of attenuated blood against one another. Nor is it to be forgotten that *both* of the usual causes of friction-murmurs, namely, diseased structures and diseased blood, may be co-existent. For instance, when there is a history of rheumatic fever, which has lasted for a long time and has been perhaps treated by depletory measures, it is often very difficult for the practitioner who has never seen the case before, to determine whether a murmur over the aortic valves be due to disease of those valves or to an anæmic (spanæmic) condition, or to both those causes, and probably the result of treatment will alone clear up the difficulty.

Again, the question has been proposed even by the most distinguished auscultators, as, for instance, by Dr Walshe[1] and Dr Flint,[2] whether simple palpitation may not produce a murmur, although the heart be healthy and the blood in a natural condition. Both these authorities admit the possibility of such an occurrence, although they consider it to be rare; and Dr Walshe seems to agree with Dr Stokes in believing that even a systolic mitral murmur may be produced at the apex of the heart without organic disease.[3] Still, in the great majority of cases, the two facts of the inorganic murmur being systolic in time and basic in seat, will afford valuable diagnostic information, and more especially if this murmur, heard over the base of the heart and also in the carotid arteries, be associated with a continuous murmur in the veins, to which attention will now be directed.

Chlorosis and Spanæmia.—Many cases, more especially in females, present themselves with apparent symptoms of disease of the heart, in which, however, that organ, although weak, is sound in structure. The disease, in fact, is in the blood, which is deficient in red cor-

[1] 'On Diseases of the Heart,' p. 172.
[2] Flint on 'Diseases of the Heart,' p. 468.
[3] Loco citato—note at bottom of the page.

puscles, and contains an abnormal proportion of water. This condition of the blood exists in the disease called chlorosis, which is usually associated with some irregularity of the menstrual function, but it may also be caused by any circumstance which impoverishes the circulating fluid, such as repeated hæmorrhages, insufficient food, or wasting diseases. Violent palpitations are experienced in such cases, and the pale or yellow colour of the skin, the blue appearance of the eye-ball, and the feeble pulse, will all materially assist the diagnosis.

But the stethoscopic signs are very remarkable, and there is not only a blowing murmur in the arteries, but also a loud continuous murmur in some of the veins. Unlike the murmurs caused by organic disease, these murmurs are transient in their duration, disappearing when the condition of the blood is improved. The physical cause of these morbid sounds is the reaction of attenuated blood against the vessels, for, as Dr C. J. B. Williams remarks, a thin watery fluid is more readily thrown into sonorous vibrations than one of a richer, more viscid character.[1] The position of the arterial murmurs is over the base of the heart and is always systolic, and the sound may also be very often heard in the carotid arteries. But a sound of a very loud character may also be often heard in the jugular veins, as was first pointed out by Dr Ogier Ward in 1837. This murmur is continuous and of a humming character, and from its resemblance in sound to that produced by a humming-top, called in French *diable*, it was named by Bouillaud *bruit de diable*. It may be at once arrested by pressure *above* the stethoscope. The nature of this murmur was examined and reported upon by a Committee of which Dr C. J. B. Williams was the Reporter, in 1837, and Dr Ogier Ward's observations were confirmed; and the Committee proved further that precisely similar sounds could be produced by the passage of water through thin india-rubber tubes.

Treatment of Functional Diseases of the Heart

The object to be held in view in the treatment of the functional diseases of the heart is to procure rest to the disturbed organ; and this object may sometimes be attained by removing the causes on which

[1] 'On the Pathology and Diagnosis of Diseases of the Chest,' 1840, p. 910

the disturbance depends, and sometimes by the use of agents which act directly on the organ itself. Thus, if mental excitement have conduced to the malady, all circumstances leading to such excitement must be avoided, but, at the same time, change of scene, moderate exercise in the open air, or passive locomotion, as, for instance, riding or sailing, are to be recommended; and bathing, whether by plunging or swimming in the sea or in lakes or rivers, or by the use of the shower or douche baths, is a valuable therapeutic measure. The diet is also most carefully to be regulated, and indigestion, which is a fertile source of cardiac disturbance, is to be obviated. A moderate allowance of alcoholic liquids is rather beneficial than otherwise, but, of course, all excess is to be strongly deprecated, as being likely to cause or to aggravate the very conditions which these stimulants are designed to alleviate.

The medicines or the drugs to be employed will vary according to the nature of each case. Generally speaking, all depletion is contra-indicated, and I can by no means agree with those writers who recommend bleeding in any form. Purging is also to be avoided, although it is necessary to open the bowels by gentle aperients, if there be any necessity for such a step; and in females, in whom menstrual irregularity is very often associated with functional disorder of the heart, it will be in many cases absolutely necessary to prescribe aloes or colocynth, or other drugs of a similar character. In the very common affection called chlorosis, the administration of iron is eminently beneficial, and a course of this mineral, given in such form and in such combination with other drugs as the circumstances demand, will generally effect a cure; and, for obvious reasons, a course of chalybeate waters at such springs as Tunbridge Wells in this country, and Spa, in Belgium, or Langen-Schwalbach, in Germany, will be alike curative, especially as in such places the effect of the waters is enhanced by the change of scene, the amusements of the place, the excursions into the adjoining country, and the regularity of the diet. It must be observed, however, that (when chlorosis and anæmia are excluded) the iron preparations and chalybeates in general are not very useful in functional diseases of the heart, and indeed often do more harm than good. Opium, also, though a most valuable remedy as a sedative in most diseases, and especially useful in acute inflammations of the heart, is not to be recommended in functional maladies of that organ, except in special cases to be noticed in their respective places. Henbane and conium are very much to be preferred, as

they do not cause headache or constipate the bowels; and hydrocyanic acid is a very powerful and excellent sedative in the cardiac maladies now under consideration.

There is a class of remedies generally called antispasmodics, the operation of which it is very difficult, if not impossible, to explain on scientific principles, but which are decidedly beneficial in many nervous diseases, including the functional disorders of the heart. Among the foremost of these is valerian, which has an extraordinary power over the disordered innervation in many functional disorders; and to the same category of drugs belong many others, some of an odoriferous and some of even a nauseous character, such as castoreum, assafœtida, galbanum, musk, sumbul. Chloroform, given internally, and the chloral hydrate are very useful as nervine sedatives, and the aromatic spirit of ammonia forms an excellent remedy, both as removing acidity and exercising a gently stimulating action on the depressed though excitable nervous system. When indigestion is present, as it very often is, the alkalies, potash and soda, or the alkaline earths, lime and magnesia, are to be recommended; and if there be a gouty diathesis present, potash and lithia (as being special solvents of uric acid) must be regularly and fully administered. In other cases, not characterised by the presence of acidity or by the gouty diathesis, the mineral acids, as the sulphuric, the nitric, the phosphoric, and especially the nitro-hydrochloric, may be advantageously prescribed in combination with light bitter tonics, as gentian, calumba, quassia, or cascarilla. In the neuralgic class of cases, especially when the pain returns at certain intervals, quinine is, of course, to be recommended, but, except when neuralgia is distinctly present, this powerful tonic and antiperiodic is not especially useful in the functional diseases of the heart. In some mixed cases, however, the combination of antispasmodics with tonics, as is exemplified in the valerianates of zinc and iron, may prove useful. Belladonna, employed in the form of a plaister over the præcordial region, is exceedingly serviceable in relieving the pain so often experienced in palpitation of the heart, and in reducing the palpitation itself.

Digitalis, which is so often useful in the organic diseases of the heart, is scarcely less efficacious in those of a functional or nervous character. Whatever may be the precise *modus operandi* of this powerful drug, it is quite clear that it exercises a *regulating* power over the heart's action, lowering it when it is too strong, and raising it when it is too weak, and sensibly diminishing the velocity of the

pulse. Digitalis may therefore be used in very different forms and conditions of functional disease of the heart, and it may be combined, as occasion requires, with aperients, such as the neutral salts; or with sedatives, such as hyoscyamus and conium; or with tonics, especially iron, in those cases where there is over-action of the circulatory system together with a watery condition of the blood.

It only remains to be added that the functional diseases of the heart often require the closest attention and sometimes the exercise of the greatest patience on the part of the medical practitioner, and much disappointment will often result even after the most careful selection of remedies, and the most judicious advice as to diet, exercise, and habits. Nevertheless, the prognosis in such cases is always good, and treatment is generally successful in the end.

CHAPTER XIX

ANGINA PECTORIS

THE etymology of *Angina Pectoris* conveys no information as to the nature of the malady which the term is intended to designate. The word *angina*, which appears to be derived from the Greek, αγχω, *to strangle*, has a very indefinite signification, and is employed in the vaguest manner by some of the older medical writers, but generally in connexion with some complaint of the throat. Thus Bretonneau at first called the malady afterwards known as tracheal diphtheria, *Malignant Angina*. The term *Angina Pectoris* was first employed by our own countryman Heberden to denote the disease now under consideration, and it literally means a "choking or strangling of the breast," but it can scarcely be said that this expression represents the peculiar symptoms of the disease. It is very commonly employed in popular language to explain any anomalous painful feeling connected with the heart, and is also sometimes vaguely stated as being the cause of sudden death in cases where no post-mortem examination has been made. For reasons presently to be stated, I doubt whether angina pectoris is so fatal a disease as is generally supposed, but this observation applies only to those cases where the malady is uncomplicated with organic disease of the heart.

The question, however, which arises in the first instance is whether such a disease as angina pectoris really exists as a substantial entity, or whether the name only represents a group of symptoms dependent on some definite cardiac lesion? I think the balance of evidence must lead us to believe that there is such a disease existing *per se*, but that, like asthma, it is comparatively rare, while its manifestations, as symptomatic of other and more serious maladies, are not uncommon. If angina pectoris were more frequently observed in the female sex than in the male, it might be suspected to belong to the hysterical group of diseases, but it is rather more common in males,

and the symptoms, on the whole, are very different from those of hysteria. Again, considering the immense importance of the heart as the centre of the circulatory system, and the disturbances to which its derangements are likely to lead, it might be supposed that angina pectoris, as symptomatic of heart-disease, would be a very common malady, but such is not the case, and, as has often been observed in previous chapters, the most serious lesions of the heart are often unattended with any special sufferings referred to that organ by the patient.

Dr Heberden first described angina pectoris in a short chapter of his 'Commentaries,' and he states that he had seen nearly a hundred cases of the disease,[1] three having been women, one a boy, and the rest men near or past fifty years old. He regards it as a spasmodic complaint, the attacks being sudden, and sometimes fatal, but often exhibiting long intervals of perfect health. "Those who are afflicted with it," he says, "are seized while they are walking (more especially if it be up hill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or continue; but the moment they stand still, all this uneasiness vanishes."[2] But he states that others have been seized while they were standing still or sitting, or upon first waking from sleep, and the pain sometimes reaches to the right arm as well as to the left, or even down to the hands.

There can be no doubt that, on the whole, Heberden has given a good though very brief description of this curious affection, but, since he wrote, the advance in cardiac pathology has been so great as to exclude a large number of cases which would otherwise be described as angina pectoris, and to relegate them to the category of organic heart-disease. It is evident that if there be valvular lesion or degeneration of the heart-substance, all the suffering experienced by the patient (if there be any) must be referred to the primary morbid condition, and only those cases in which there is *no* appreciable or demonstrable alteration in structure ought now properly to be regarded as instances of true angina pectoris.

When these observations are borne in mind, the number of cases of angina pectoris will be very much reduced, and the conflicting accounts of the disease given by many authors will be more

[1] 'Commentaries on the History and Cure of Diseases.' By William Heberden, M.D. 1806, p. 366.

[2] Op. citato, p. 364.

easily comprehended and reconciled. If, for instance, in a given case, there be paroxysms of dyspnœa, and feelings of suffocation and faintness, and after death there be found valvular disease of the heart or alteration of its muscular structure, it is clear that the symptoms were all due to such organic changes; but, on the other hand, when we find persons suffering under what are considered symptoms of angina pectoris, in whom no cardiac or other lesion can be discovered during life, and who perhaps eventually die of other diseases, it is fair to assume that the group of objective and subjective phenomena then denote only one of those derangements which, for want of a better name, are denominated "nervous."

For my own part, I believe that angina pectoris, under the limitations above specified, is a nervous disease, and I therefore include it among the functional affections of the heart, and I further believe that it is not so fatal as is usually supposed. In offering this opinion, I distinctly exclude all those cases where there is organic disease, whether it be discovered before death or afterwards, for then the malady is no longer angina pectoris, but aneurism of the aorta, or valvular lesion, or fatty metamorphosis, or some other appreciable morbid condition. Of course I do not deny the possibility of death ensuing from a sudden spasm of the heart or from a sudden stoppage of its action, without the existence of any lesion perceptible to the stethoscope, or the microscope, or the scalpel; all I advance (and I do so as the result of considerable experience) is that such events are rare. Some years ago, I made a number of post-mortem examinations in cases of sudden death, and I have made many since; one series of such cases was published in the 'Lancet' for 1842.[1] The causes of death in all these instances, although they were very various, were perfectly demonstrable, some being from hypertrophy and dilatation of the heart, some from cerebral congestion, some from internal hæmorrhage; and I may mention that I have hardly met with a single case in which (excluding cases of natural and senile decay) life has been prematurely cut short without some appreciable cause. When, therefore, I find it mentioned that such and such a person has died of angina pectoris, my first question is whether there was any post-mortem examination, and if not, I conclude it to be just as likely that death ensued from the rupture of an

[1] "On the Pathology of Sudden Death." By R. H. Semple, 'Lancet,' 1842, p. 76.

aneurism, or from valvular disease of the heart, or from fatty degeneration, or from congestion of the brain, or from some other appreciable lesion, which would have been found, *if it had been looked for*.

But angina pectoris, as a specific disease, presents several well-marked characters, and indeed cannot well be mistaken. As Heberden observes, it comes on suddenly, sometimes in the daytime when walking, but sometimes in the night, on awaking from sleep. It is characterised by pain in the region of the heart, extending generally to the left side or towards the shoulder or down the left arm, or even to the fingers. But a feeling still more characteristic and more distressing than pain is that of choking or suffocation, with an apprehension of impending death, and this sensation is so distinct, and is so fully impressed upon the mind of the patient, that a repetition of the attack is regarded as almost a certain prelude to dissolution. There is not much difficulty of breathing, but the action of the heart is often irregular and intermitting, the pulse feeble, the countenance pale, and the body covered with a cold sweat. The paroxysmal nature of the affection is another of its remarkable features, for the patient may and probably does entirely recover from one attack, and may experience another at longer or shorter intervals, or may be free from any of them for the rest of life, or may be subject to them after the lapse of months or years.

Angina pectoris, thus characterised, is not a common disease, and although, as I have stated, it is essentially a nervous affection, yet it *may* accompany organic lesions of the heart. But I entirely agree with Dr Flint that such connexion is rare. That physician found that out of more than a hundred and fifty cases of organic disease of the heart observed by him before the year 1859, angina pectoris existed only in seven; and in a hundred and eighty-eight cases of a collection analysed by him in preparing the second edition of his work in 1869, the disease in question existed in only eight,[1] the proportion, therefore, being 15 in 388. Again, the same authority states that during ten years he noted only four cases of true angina pectoris, that is to say, where the disease was unconnected with cardiac or aortic lesions.

Pathology.—The description given by Heberden clearly comprehends two classes of cases, one in which there is some organic disease of the heart or the aorta, and the other in which there is no

[1] Flint on 'Diseases of the Heart,' p. 297.

organic disease. The advance of cardiac pathology has enabled us to ascertain, in the great majority of cases, whether the heart or aorta is structurally altered, and, if so, the alterations will be referred to their respective categories as diseases of those organs, and the assemblage of phenomena constituting angina pectoris will be reduced to the rank of symptoms. When we endeavour to ascertain the pathological cause or causes of angina pectoris, *per se*, namely, of the affection as an idiopathic malady, the inquiry is a difficult one. It was once currently stated that the cause was to be found in ossification of the coronary arteries, and it is probable enough, arguing only from abstract pathological principles, that such a condition might give rise to the symptoms of angina pectoris. But, in the first place, it may be asked whether ossification of the coronary arteries is ever found as an isolated phenomenon apart from ossification, or rather calcification, of the aortic valves or the aorta, and I think that the question must be answered in the negative. I have never seen such a condition myself, and I have never heard or read of its having occurred in the practice of others. All the cases mentioned, as far as I can learn, are instances where the calcification of the coronary arteries is merely subsidiary to or connected with ossific degeneration of other arteries or structures, and, so far from the disease of the coronary arteries causing the painful paroxysms characteristic of angina pectoris, I have already shown that even enormous obstruction by ossific or calcareous deposit on the aortic valves may be unattended by any serious symptoms at all. (See p. 104, *et seq.*) It must therefore be evident that *if* angina pectoris be ever connected with disease of the coronary arteries, it must be only in those rather rare cases where the symptoms are due to calcified valves, or ossific degeneration in other arteries besides the coronary. My own impression is that angina pectoris, as an idiopathic malady, is caused by some affection of the pneumogastric nerve, the phrenic being also in all probability likewise involved. It is difficult, perhaps impossible, in the present state of our knowledge to determine exactly in what this nervous affection consists; but its place in nosology must be found among other conditions, called, for want of a better term, *neuroses*, and including such maladies as hysteria, neuralgia, &c.

Prognosis.—If the truth of the above observations be admitted, the prognosis of angina pectoris (always regarding it as idiopathic) will not be difficult. Like neuralgia and hysteria, the disease

is a very painful one, but not likely to be fatal. In the fatal cases recorded by authors, there has been some organic lesion of the heart or aorta, sometimes an aneurism of the latter. If, therefore, no such organic lesion can be detected during life, in all probability the case will terminate favorably, or at all events will not be attended with fatal consequences. But it must not be forgotten that, even with all the refinements of modern diagnosis, organic disease of the heart or great vessels *may* exist without being discovered during life, and a consciousness of this fact should always be present to the mind when offering a prognosis, and should suggest caution in expressing our opinions to patients. I do not encumber this book with the details of many cases, but I adduce the following, out of many others I have attended, as being typical of the disease now under notice.

CASE.—Mr W. S—, a gentleman of high education and somewhat nervous temperament, called me up in the middle of the night some fifteen years ago, and I learned that he had long been a sufferer from angina pectoris. I had not attended him before, but he had been a patient of the late Dr Hodgkin. The symptoms were well marked, and need not be detailed. I ordered him some opiate, and brandy and ammonia. He soon after fell asleep, and the next day he was quite well and pursued his professional avocations as usual. I became very intimate with this gentleman from that period, and was frequently called to see him professionally for the same malady and for others, chiefly of a nervous or dyspeptic nature. But I wish particularly to remark that, although I examined his chest repeatedly, I never could detect any lesion whatever in the heart, aorta, or lungs, or, indeed, in any other organs, although when I first attended him he was nearly seventy years of age. At the age of eighty-four, after taking a little cold, he evidently manifested symptoms of failing vital power, and I regularly attended him, and saw him frequently in consultation with a distinguished physician and stethoscopist, but although we both examined him most carefully we could detect no mischief whatever in the heart or great vessels, or in any other organ. He sank gradually, and ultimately died from mere exhaustion and decay of nature. I think that this was a model case of angina pectoris.

TREATMENT.—After what has been stated on the subject of treatment of functional diseases of the heart in general, little need be added on that of angina pectoris. The object to be held in view

is to relieve the urgent symptoms, and, if possible, to obviate their recurrence. The first indication is fulfilled by the administration of sedatives, among which opium holds a prominent place, although others may be given. Stimulants and anti-spasmodics are also eminently useful. In the case of a female patient now under my care, who has very frequent attacks of angina pectoris, and who accurately describes her symptoms, among which the darting pain, starting from the heart and proceeding towards the side, and also the terrible dread of death, are the most prominent, I have found that the administration of sedatives and stimulants has been very serviceable, and, hitherto at least, my constant assurance to her that she was in no real danger of sudden death has been verified by the present results. In this case, as in many others, the application of a belladonna plaister over the præcordial region has afforded great relief. I have repeatedly examined this patient's heart, and can find no evidence whatever of organic disease.

CHAPTER XX

EXOPHTHALMIC GOITRE

Among the anomalous affections of the heart, with which disturbance of other organs is mysteriously associated, is that peculiar disease known under the name of Exophthalmic Goitre, or, as it is also called, after the names of the physicians who first accurately described it, Graves's disease and Basedow's disease. In this malady there are three prominent and constant features, namely, palpitation of the heart, hypertrophy of the thyroid gland, and prominence of the eye-balls. The last-named feature is so remarkable that the disease can be at once recognised by its appearance; and it is so obviously due to physical change in the structure of the parts that the patient experiences a difficulty in closing the eyelids completely, and the eye-balls are often partially uncovered during sleep. A case is quoted by Trousseau, in which the eye-balls were protruded so much that one of them actually came out of the orbit, necessitating its replacement by the fingers. Nevertheless, as Trousseau observes, the prominence of the eye-ball sometimes escapes notice, and then the character of the disease must be developed by other means;[1] and indeed the exophthalmos appears to vary in extent under the influence of mental emotions, and in women during the performance of the menstrual function.

The next important feature, then, in this disease is the enlargement of the thyroid gland, and this character is a very striking one. In a case still under my care, and which will be hereafter described, the prominence of the eye-balls is so remarkable as to excite notice when the patient is walking abroad, and the swelling of the thyroid gland is no less obvious a feature. Yet, in this particular, the appearance is not always constant, for, in the patient to whom I now refer, the swelling of the thyroid body is less than it was, and Trousseau relates

[1] Trousseau's 'Lectures on Clinical Medicine,' 1867, vol. i, p. 542.

a case where a striking hypertrophy of this organ was succeeded by atrophy. In some instances, too, the hypertrophy, on the same authority, is said to have developed itself almost suddenly.

Most usually, however, the hypertrophy is gradual in its development, beginning insidiously, and sometimes being temporarily arrested in its progress, and afterwards increasing so much as to modify the voice and breathing. It has been found, by pathological investigation, that there is glandular hypertrophy in these cases, but there is also great development of the blood-vessels of the organ, and to such an extent that the swelling is said to have been sometimes mistaken for an aneurism.

The state of the heart in this disease is especially deserving of notice, the patients always complaining of palpitation. There is violent beating of the heart, and the valvular sounds are increased, and they are said by Trousseau to be generally accompanied by a soft bellows-murmur, audible also in the large arteries. It is not yet quite certain whether in exophthalmic goitre the heart is only functionally affected, as is supposed by Dr Stokes (who regards the affection as a cardiac neurosis), or whether it is organically diseased, as the increased impulse and bellows-murmur at the base of the heart, described by Trousseau, would seem to indicate. Trousseau's own opinion is that, as a rule, Graves's disease is not necessarily attended with disease of the heart.

Cases which I have seen myself might be adduced on either side of the question. In the remarkable case to which I shall presently refer, I have examined the præcordial region and that of the neck on repeated occasions, and I have found distinct *frémissement cataire*, with increased impulse of the heart, and a full and very rapid pulse, and on applying the stethoscope I have found invariably a loud bellows-murmur over the situation of the aortic valves, propagated into the carotids. I cannot help regarding this case as one of organic disease, more especially as I have often tried the effect of iron, which has not in any way changed the character of the murmur. In other cases, however, of exophthalmic goitre, I have found, indeed, palpitation of the heart with rapid pulse, but there has been no *frémissement cataire*, nor any bellows-murmur, although the natural sounds have been intensified. The pulse, too, in these instances, although rapid, was weak.

With my present experience, combined with reading, I think that Trousseau was correct in coming to the conclusion that the disease in

question is not necessarily attended with organic disease of the heart.

Exophthalmic goïtre is pretty common in women, but rare in men, although Trousseau gives an instance in which it occurred in a man, aged thirty-five. The case, however, was not a typical one, as appears from the clinical history. Out of fifty cases collected by Withuisen (quoted by Trousseau) only eight occurred in males.

It would appear that the disease is a rare one in America. Dr Flint states that when the first edition of his work on 'Diseases of the Heart' was published he had met with only a single case, and at the date of his second edition (1870) he observes that he has recorded only five cases in the previous ten years in a wide field of clinical observation, and he further remarks, as an evidence of its infrequent occurrence, that in a service at Bellevue Hospital during eight months of the year for eight years he had seen only two cases.[1]

Pathology.—The pathology of this curious malady is almost as mysterious as the symptoms are anomalous. The three prominent features by which it is characterised, namely, rapid action of the heart, protrusion of the eyeballs, and swelling of the thyroid gland, are so remarkably manifest in the typical cases that they would appear due to some demonstrable physical causes, and yet this is by no means the fact. The heart, although its pulsations are greatly accelerated, is not necessarily affected with organic disease; the protrusion of the eyeballs is not demonstrably due to any hypertrophy of the eye itself, or dropsy of its humours, or disease of its muscles; and even the swelling of the thyroid gland, though a visible and tangible reality, is by no means constant, but is subject to alternations of increase and subsidence, and in some cases may disappear altogether.

I have already expressed my concurrence in the opinion of Trousseau, that exophthalmic goitre is not necessarily attended with organic lesions of the heart, and the experience of many other observers seems to confirm this view. The evidence, however, is but scanty, and for the best of all reasons, namely, that the cases of the affection are rare, the deaths from the disease are not numerous, and the post-mortem examinations consequently few in number. Trousseau gives the particulars of an interesting case, which was watched during life, and examined after death, but it is by no means a typical example of the disease, for the patient had suffered

[1] Flint on 'Diseases of the Heart' (1870), p. 307.

from various other maladies, as intermittent fever, angina pectoris, ascites, neuralgia, and epistaxis, and died at 60 of apoplexy. The only prominent feature of the disease, when it came under Trousseau's care, was the exophthalmos, for the heart's action was not very rapid, the pulse being only 96, and the thyroid gland was of small size. The case therefore loses very much of its value as an instance of exophthalmic goïtre, and the immediate cause of death was found to be a large hæmorrhagic effusion in the left hemisphere of the brain, and the liver, the spleen, and the kidneys were all more or less diseased. What relates to the exophthalmos was interesting, for it was found that the cause of the protrusion of the eyeballs was a large amount of areolar and adipose tissue in the sockets, though the eyeballs themselves and the ophthalmic arteries were not larger than in health. The thyroid body, although very small in size, was very hard and almost scirrhous in consistence, and its lobes "had a lobulated quasi-cirrhotic aspect, owing to the retraction of their fibrous elements."[1] The heart was hypertrophied, and there was some slight disease of the aortic and mitral valves. Among the structures examined (and they all seem to have been examined with the greatest care) the cervical ganglia of the sympathetic were carefully dissected by the clinical assistants of the Hôtel Dieu, and microscopically investigated; the result being, as Trousseau informs us, that there was a predominance of the connective tissue and diminution of the nervous elements. There was a further peculiarity in the onset of this case which occurred seven years before her admission; for it appears that in the course *of a single night*, when the patient was suffering from strong mental emotion, the three symptoms of Graves's disease showed themselves, namely, palpitation, swelling and throbbing of the thyroid body, and exophthalmos. The two former characters, however, had disappeared when the patient came under Trousseau's notice at the Hôtel Dieu.

The above case, then, offers no great confirmation of the necessary connexion of organic lesion of the heart with exophthalmic goïtre, for there was so much disease in other organs that the cardiac mischief might have been only a concomitant circumstance. But a Danish physician, named Withuisen, has collected the particulars of seven fatal cases in which post-mortem examinations were made, and in all of them some organic disease of the heart was detected, but it seems to be admitted that they had been ill for several years, and in

[1] 'Trousseau's Lectures,' New Sydenham Society, 1868, p. 581.

one of them there was atheromatous disease of most parts of the arterial system and fatty matter in the heart itself.

As to the existence of hypertrophy of the heart in this disease, the fact has been asserted by some, and denied by others; but it seems to be now admitted that while this form of enlargement may co-exist with the other symptoms, it is not essentially and necessarily present. When there is violent action of the heart, it is often assumed that the organ is enlarged, but it by no means follows that such is the case, unless the area of cardiac dulness is increased and the pulse is full and vibrating. In order to account for and to reconcile conflicting opinions on this point, Trousseau has recourse to an ingenious and plausible hypothesis, namely, that although the complaint does not necessarily bring on hypertrophy of the heart, yet it may give rise to a lesion analogous to the cardiac hypertrophy which occurs during pregnancy, a condition which is often only transient. When we reflect on the manner in which the uterine muscular fibres increase during pregnancy and diminish after delivery, it is certainly no very great stretch of imagination to conceive that a similar alteration may exist in Graves's disease.

This leads to the consideration of the other important features of the disease, namely, the protrusion of the eyeballs and the swelling of the thyroid gland, neither of which are necessarily hypertrophied any more than the heart. In reference to the thyroid gland, it is perhaps almost unnecessary to remark that although the disease is called exophthalmic goître, it is totally and essentially different from goître, properly so called, and requires a wholly different treatment. The goître of Switzerland and Derbyshire, and elsewhere, is an enlargement of the gland, gradually commencing and increasing, due to some tolerably well-known climatic, local, and meteorological conditions, and often admitting of a cure by known topical appliances and surgical operations, but the exophthalmic goître arises from no such conditions and admits of no such cure. In the latter there is indeed enlargement, and there is often hypertrophy, and the blood-vessels are increased, but, as has been shown, this condition may be developed suddenly, and it may pass away entirely, or it may end in atrophy, or it may be absent altogether. Paradoxical as the expression may seem, Trousseau regards the affection as being really conducive to *atrophy* of the thyroid gland, or at least of its proper substance; for he shows that the original hyperæmia of the organ may give rise to the excessive development

of fibrous tissue constituting a kind of cirrhosis which constricts the parenchyma, cuts off its supply of nutrition, and thus ends in atrophy and degeneration; the change, in fact, being analogous to what occurs in the cirrhotic liver.

With respect to the protrusion of the eyeballs, it was indeed seen in one of Trousseau's cases that the appearance was caused by the presence of a large amount of areolar and adipose tissue in the orbit; but when it is known that this symptom sometimes rapidly appears in a paroxysm and then disappears, it is impossible to suppose it to be due to any constant pathological or physical condition, and it must rather be attributed to some transient congestion. It may, perhaps, be caused by the pressure exerted on the jugular veins by the enlarged or obstructed thyroid gland, which, by arresting the passage of blood from the ophthalmic veins, may cause venous congestion in the orbit, and the disappearance simultaneously of the goïtre and the exophthalmos would explain the phenomenon. I was lately strengthened in this opinion by observing the case of an infant five months old, in whom there is an enormous swelling of the lymphatic glands of the neck, and in whom there is protrusion of the eyeballs, due apparently to the pressure on the jugular veins. At any rate, all efforts to discover any constant pathological change in the eye itself, or the ophthalmic artery, or the ophthalmic vein, have been only partially successful, and the results announced by some observers are often contradicted, or at least are not confirmed, by the researches of others.

What, then, is the pathology of exophthalmic goïtre? As it is impossible to connect it with any known and constantly occurring pathological change in the heart, or the thyroid gland, or the eye, the seat of the disease must be sought in the nervous system, and in all probability in the sympathetic. Without entering into all the interesting facts and theories lately put forward to explain the influence of this system upon the internal vital organs, it may be stated generally, as an admitted truth, that it exercises a kind of inhibitive or restraining influence upon the vascular system, and that when this influence is withdrawn the vessels become distended with blood, and the circulation is accelerated. I purposely abstain from arguing in the present place the question as to the relative functions of the sympathetic system and of the pneumo-gastric nerve in regulating, accelerating, or controlling the action of the heart; and I admit that the exact effect produced by the sympathetic in exophthalmic

goïtre cannot be closely and logically demonstrated. Nevertheless, both by the existence of some evidence, and by the way of exclusion, it may be asserted that the disease is intimately allied with or caused by some derangement of the vaso-motor system, and the very facts of the occasional sudden appearance and the mysterious disappearance of the symptoms are explicable upon hardly any other hypothesis. Trousseau, therefore, is probably right when he compares the rapid action of the heart in this disease and the swelling of the thyroid gland and of the eyeballs to the temporary congestions occurring in the erectile tissue of man and the lower animals under several well-known circumstances; and he may be equally right when he suggests "that the congestion of the thyroid gland and the eyeballs may be regarded as a kind of pathological erection of those organs,"[1] analogous to those congestions which Claude Bernard produced at will in different regions of the body by irritating or by cutting certain branches of the sympathetic nerves.

Of one thing I am pretty well convinced in reference to exophthalmic goïtre, namely, that it is not necessarily a fatal disease, and although not very curable, that it cannot be regarded as particularly dangerous.

The *treatment* of exophthalmic goïtre is rather unsatisfactory because its nature is so imperfectly understood that it is difficult to determine the remedial measures which are indicated. Any surgical interference with the thyroid gland or the protruding eyeballs would be either ineffectual or mischievous, and the only rational indication in the disease appears to be to act in some way on the nervous centres, from which the morbid influence, giving rise to the symptoms, appears to originate. Trousseau, whose therapeutics were singularly bold and original, and by no means always in accordance with those of the French school, recommends three remedial measures, namely, bleeding, digitalis, and hydropathy. His advocacy of the first is certainly paradoxical, because no man has more energetically than himself denounced blood-letting in many diseases in which such a step really appears to be admissible; and, for my own part, I consider that bleeding is entirely contra-indicated in exophthalmic goïtre. If the malady depends—as it probably does—on some want of inhibitive power on the part of the sympathetic system, then bleeding, by further weakening that power, must tend to aggravate rather than relieve the symptoms, and it is well known that, when the heart's

[1] 'Trousseau's Lectures,' p. 578.

action is accelerated by nervous influence, the loss of blood accelerates it still more. Hydropathy, by which is meant the water-treatment (though *water suffering* is the etymological meaning of the word), appears to me to be equally unsuitable, although Trousseau gives an instance where it was successful; but when he points out to his class, as an argument for its adoption, that they "know what good effects may be obtained from it in anæmia, chlorosis, and hysteria," the only comment which strikes the mind is that if those diseases are cured in France by hot and cold water, internally and externally, they must present a very different type to that which they assume in England and other countries.

Digitalis, however, is undoubtedly useful. That drug, as has been fully mentioned already, has an extraordinary power of retarding and regulating the action of the heart, exerting its therapeutic properties in all probability through the influence of the nerves, and hence it is peculiarly adapted to exophthalmic goïtre, in which violent and rapid action of the heart is a prominent and very characteristic symptom. Trousseau recommends it to be given boldly and in large doses, and to be continued until symptoms of incipient poisoning are produced, such as vertigo, cephalalgia, and nausea, but that when the pulse falls to 60 or 70, the digitalis should be stopped, or the dose diminished. In the case I am about to relate, it will be observed that digitalis was taken for a very long period and with beneficial effects, and that the pulse never fell very considerably during its administration. As debility and anæmia are frequently associated with exophthalmic goïtre, I should certainly recommend the use of iron in combination with digitalis; and, as in females the disease is often accompanied by derangement or stoppage of the catamenia, the remedies appropriate to such a condition should be administered. Various sedatives—such as hyoscyamus, conium, and bromide of potassium—are also likely to prove useful, but I think opium is objectionable on many grounds. While Trousseau recommends blood-letting, and water-bathing and water-drinking, in this disease, he seems to me equally paradoxical in omitting belladonna from the category of remedies, for this is a favourite medicine of his in diseases dependent on irregular conditions of the sympathetic system, and it has been employed by some practitioners in exophthalmic goïtre with a certain amount of success.

I subjoin a very remarkable and typical case of this disease occurring in my own practice.

S. J. S— was first seen by me on July 19th, 1872, and she said that she had been ill for three years. She is a servant, single, and 29 years old. She is tall and thin, and the nature of her complaint is at once apparent, for the eyeballs are very large and prominent, appearing to start out of their sockets, and very imperfectly covered by the eyelids; and she has a very large goïtre extending on both sides of the neck. No cause can be assigned for her illness, and she has led a very quiet life, having been from her childhood a servant to the same lady with whom she is now living, and who treats her more like a relation than a subordinate.

She is rather nervous, and speaks in a subdued voice, but she is not hoarse, and she has no difficulty of breathing. In addition to the appearance of the eyes just described, there is great photophobia, so that she cannot bear the light of the laryngoscope, which it was proposed to use to examine the larynx, but she does not shrink from the ordinary light of day. (Subsequently, however, whether from increasing photophobia or from a wish to conceal the very prominent and ghastly-looking eyeballs, she wore goggles.) The face and skin generally are pale, the tongue is natural, the appetite is fair, neither very good nor very bad, and there is no thirst; the menses have stopped. There is restlessness at night; the temperature is good, and the demeanour quiet and gentle. The pulse is very rapid, full, and strong—160 in the minute; there is violent palpitation of the heart, with visible impulse, and on palpation there is *frémissement cataire*, and the impulse is felt over an extended space. On a careful examination of the heart with the stethoscope, made over and over again, it is found that the impulse is increased, lifting up the instrument at every beat, and while the sounds at the apex are only natural, though exaggerated in tone, there is a most distinct, harsh, and loud murmur (systolic) over the base, but the second sound is natural. The murmur is distinctly prolonged up the great arteries on both sides of the neck, especially the right; and there is also a loud humming, continuous murmur (*bruit de diable*) over the right jugular vein, stopped by pressure *above* the stethoscope; there is also violent throbbing of the arteries in the goïtre.

As a great variety of treatment was adopted by me in this case, which I have had under continuous observation since that time, and have still under my care, it would be tedious to enumerate the dates of attendance, and the particulars of all the medicines ordered.

It should be observed, moreover, that the treatment was rendered the more difficult and embarrassing from the whims of the patient as to some drugs, and her inability, or supposed inability, to take pills, so that I was unable (as I wished to do) to give aloes and colocynth and some similar remedies with a view of acting upon the menstrual secretion. However, on the date referred to, I began by giving twice a day ten minims of tincture of digitalis and ten of tincture of henbane in an ounce of peppermint water, and this medicine somewhat reduced the rate and strength of the pulse, but not very much; for the rate did not fall, and indeed never has fallen, below 140, notwithstanding the repeated and continuous employment of digitalis, and the pulse has always maintained its strength as well as its rapidity. After about a fortnight after this medicine had been taken, namely, August 2, 1872, I tried the effect of bromide of potassium in doses of ten grains twice a day together with the first mixture, but there was no beneficial effect, and the patient complained of the medicine, though without any definite grounds. But as I thought that anæmia might be one of the factors of the disease, I, a fortnight afterwards, namely August 16, gave her ten minims of the tincture of perchloride of iron and ten of tincture of digitalis twice a day, and this she continued for nearly a month, when she complained of this medicine also, and I therefore gave her only a simple bitter tonic, but substituted for this on the 20th of September a quinine mixture, though without any good effect.

On the 4th of October, thinking that many of her symptoms were probably nervous, I ordered her a combination of twenty minims of compound tincture of valerian and twenty of spirits of chloroform, with peppermint water, twice a day; but as no improvement resulted, I fell back, on the 18th of October, on the combination of digitalis and henbane first prescribed, and this medicine, with the occasional addition of spirits of chloroform, was continued for nearly five months, with considerable relief, the pulse being somewhat reduced; but as I have before remarked, not very considerably so, and the violent action of the heart, the blowing murmur over the base, the *frémissement cataire*, and the murmurs in the neck, all remained pretty much as before. About this time, iodide of potassium was also prescribed, and taken for about a month, without any marked result, and the digitalis was omitted for a short time, but resumed, and now in larger doses, twenty minims of the tincture

being given twice a day, and continued in this dose for nearly three months.

On the 26th of June, 1873, I was suddenly called in the evening by a telegram to see this patient, who lived at some distance from my house, she having always previously come to see me. I thought that some dangerous complication had arisen, either from the prolonged use of the digitalis, or from the heart-disease, and feared some serious catastrophe; but when I arrived, I found her indeed apparently very ill, but with symptoms in no way connected with either of the circumstances from which my apprehensions arose. She was, in fact, lying on the floor, occasionally convulsed, with repeated vomiting, all the symptoms being probably hysterical. I should mention, in a parenthesis, that this person was of perfectly sober habits, and, as I have before stated, her mode of life was very tranquil and monotonous. I ordered her some antispasmodic medicine and some hydrocyanic acid to check the vomiting, and was in some little anxiety to know the result of the case; but I was not called again to visit her, and, to my surprise, she appeared before me as usual a few days afterwards, and was better, and was again put upon the combination of digitalis and henbane (mxx of the tincture of each), which was continued for more than three months, when, although she was neither then nor at any subsequent time suffering from the effects of the digitalis, I tried merely carminatives in order to allay some dyspeptic symptoms which manifested themselves together with occasional diarrhœa.

At the end of March, 1874, as no other medicine seemed to suit her so well, I again fell back upon the mixture of digitalis, henbane, and spirits of chloroform (mxx of each), and this was continued, with the temporary use of bromide of potassium, and the temporary discontinuance of the chloroform (in accordance with the patient's whims), for many months longer. Iron was again tried, without any good result, but digitalis and henbane (always in doses of twenty minims each) seemed to be uniformly beneficial.

At the date of the present report (February 26, 1875), the patient is certainly no worse, and indeed is rather better than when she came under my treatment nearly three years ago. The prominence of the eyes remains the same as at first, and she now wears goggles, but the goître is considerably diminished in size, the pulse is 140, full and strong; the strong impulse of the heart remains, and also the loud, harsh blowing systolic murmur over the base,

and the murmurs in the neck. She expresses herself as being better, and the menses have returned.

She continues to perform her domestic duties, and often walks out, sometimes by herself, sometimes with her mistress, but, as before mentioned, she wears goggles to conceal the exophthalmos.

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