On the pathology and treatment of valvular disease of the heart and its secondary affections : being the Gulstonian lectures, delivered at the Royal College of Physicians in February 1851 / by Edward Latham Ormerod.

ON THE

PATHOLOGY AND TREATMENT

OF

VALVULAR DISEASE OF THE HEART

AND ITS

SECONDARY AFFECTIONS:

BEING THE

GULSTONIAN LECTURES,

DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS IN FEBRUARY 1851.

BY

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LECTURE I.—Part I.

Diseases of the Heart—difficulties of the subject—scope of the present Lectures—Andral's survey of our knowledge of diseases of the Heart twenty-five years ago—deficiencies noted then—how far supplied now—division of the subject.

It would be hard, Sir, to find an entirely new subject fitted for the present occasion. For that could scarcely be of sufficient importance which had wholly escaped the attention of the many talented and industrious observers who are now engaged in the study of pathology in all its branches. I may seem to have run into the contrary error by choosing a subject so trite as Diseases of the Heart. But you cannot have forgotten what fresh light and interest Dr. Budd threw on abscess of the liver, and Dr. Baly on dysentery, from this place, on similar occasions, in their masterly reviews of these subjects as they had come respectively under their observation. May I shelter myself under their example, in trying to follow, at a humble distance, in the same manner, on a different track?

Apart from its own difficulties I cannot but feel that I have undertaken a very delicate task in addressing you from this place on diseases of the heart. Our accurate knowledge of the matter, indeed, is of no great age; yet its literature is very extensive, and still we are not arrived at that period that we may adopt the conclusions which have been deduced, and forget that they have ever been the subject of controversy, or that our gratitude is due to more than one independent observer for substantiating single points. I cannot think that I have not erred, on occasion, when referring to such points, by defective or inaccurate acknowledgments of the sources of our information concerning them; but I have erred in ignorance, unintentionally.

Besides the systematic works to which one naturally looks for information, there is another class of writings, towards which, from their very numbers, such errors of omission of reference are more excusable. There are masses of valuable cases and essays buried in the volumes of our own and the foreign periodical literature, which it is almost impossible, out of London, to consult and reduce within the limits which the present purpose assigns. The pleasure of having accidentally met with such, of no inconsiderable merit, now and then, has been marred by the thought of how many more there may be to which no such lucky accident has led.

But I should be defeating the object which I had proposed to myself were I to attempt to condense here all the literature and statistics of a subject whose every axiom, perhaps, has been as warmly asserted as denied. And it would be unnecessary; for Dr. Clendinning (Croonian Lectures, Med. Gaz., 1838, p. 442) has supplied the statistics, and Dr. Bellingham (Lectures on Diseases of the Heart, Med. Gaz., 1850) has laid me under great obligations for references to the original papers in which the discoveries on this subject were made known. I would refer to their more comprehensive lectures for this information.

At the risk of incurring the charge of affected humility, I must add one remark. The observation of nature is open to all: "Nature denies her instructions to none who desire to become her pupils;" and with such opportunities as the wards and dead-house of St. Bartholomew's Hospital supplied for testing the accuracy of the descriptions or explanations given by authors, and for comparing their discrepant conclusions with the result of fresh observation, it was scarcely possible not to form an opinion of one's own on the disputed points: the more so, as often occurring in the form of questions from intelligent students: and this with every assistance, which I take this opportunity of acknowledging, from friends and students in collecting obscure cases, and from Dr. Latham and Dr. Burrows in clearing up these obscurities. I could only wish that the results had borne a larger proportion to the opportunities. With the most sincere desire to elicit the truth, it has been my chief pleasure to draw from fresh observations results agreeing with those which have satisfied the minds of the more esteemed inquirers into those particular points. I could desire no more, in seeking the truth, independently as far as might be, than to have their testimony that I had attained it. But, in communicating these results, I feel on how much surer grounds they have known, and how much better they have expressed, what I have laboured to ascertain. It may be thus with all who, devoting themselves to any subject, acquire a feeling of veneration for the very words of the great masters of that particular sci-

ence. I feel it so towards all those, —the highest names in the past and present generation of physicians in our own country, or on the continent,—who have placed the knowledge of thoracic diseases, and especially diseases of the heart, on its present foundation.

It is now a quarter of a century, since Andral, writing on diseases of the heart,* pronounced our knowledge of them,—such as his immortal countrymen, Senac, Corvisart, and Laennec had described them,—to be almost complete. Since that time this subject has engaged the attention of many of the ablest minds in our profession, especially in this country; and additions to our information have continually accrued from their inquiries. Yet will any one say, even now, that our knowledge of the subject is nearly complete? Indeed, information is wanting on many of its most important points, and the solution of one question only opens another inquiry. After all our labour, still, the higher the structure grows, the wider does the district yet to be explored appear from its rising summit.

The points which at that time appeared to Andral to need further investigation were generally these four:—First, the whole subject of pericarditis was a complete mystery; next, the causes of valvular disease were very obscure; thirdly, the effects of valvular disease, either direct, as witnessed in the pulse, or indirect, as in the lesions of different organs, seemed to require a thorough investigation, in order to explain away many seeming anomalies; and lastly, the results of auscultation and percussion appeared, even in the best hands, to lead occasionally to such erroneous conclusions, that it was evident that this most elementary part of the whole subject was imperfectly understood.

I. On each of these points much has been done since Andral first wrote. The whole subject of pericarditis, of which Louis' elaborate researches† served but to show the extent of our ignorance, has been mastered, and chiefly by the skill of Dr. Watson and Dr. Stokes.‡ Indeed, difficulties greater than Andral had conceived have been surmounted as, with our increasing knowledge, they have come into view; for we have learned to look in the now familiarly understood diseases of the pericardium for the explanation of symptoms which had been thought to depend on disease of the brain. And such is the accuracy with which we can now investigate pericardial affections, that it is scarcely too much to say that the amount of fluid in the pericardium can often be measured by the experienced ear with an exactness little short of that of the practised eye. And if the diagnosis of adhesion of the pericardium still remain unsettled, and an obloquy to auscultators, is it not because, in truth, adhesion of the pericardium is of itself unimportant, and has really no symptoms?

II. In the knowledge of the causes of valvular disease, also, great advance has been made. Dr. Latham* and M. Bouillaud have satisfactorily traced valvular lesions, in many cases, to acute rheumatism. A disease which had been, with few exceptions, considered all but harmless, save in its present effects, would appear to be, in its remote consequences, almost as great a scourge to mankind as typhus fever. Perhaps this discovery is the single most important contribution to clinical medicine that has been made for many years. More recently, Dr. Barclay† has analyzed this subject in an essay, to which there will be frequent occasion hereafter to refer.

III. Again, something has been done towards supplying the third class of Andral's desiderata. Dr. Blakiston‡ has called attention to the modes in which some of the secondary affections are produced, tracing them, with whatever measure of success, to lesions of particular parts of the heart. And Dr. Burrows§ has done for the diseases of the brain which arise from the heart what, to display valvular disease in all its terrors, it needs some one to do for the corresponding class of pulmonary affections which crowd our hospitals on each return of winter. This field of observation is still open in all directions. The inquiry may be long and difficult, but, under the hands of numerous and able observers, it is daily bearing fruit.

IV. But it is in the fourth particular that the greatest advance has been made; and it is in the improvements in the physical diagnosis that much of the progress in the other particulars turns. Dr. Hope and Dr. Williams have explained the mode of production of the first sound of the heart‖, and Dr. Carswell has cleared up

* Clinique Médicale, tome iii. 1826.
† Recherches Anat. Path., p. 284.
‡ Watson's Lectures, lect. lxi. 1st edit.

* On Diseases of the Heart, vol. i. p. 101.
† Med.-Chir. Trans., xxxi. p. 185.
‡ On Diseases of the Chest, 1848.
§ On the Cerebral Circulation.
‖ Objections have been made to the theory of the cause of the first sound of the heart, as now generally received, as being too complex. It is a sufficient answer to these objections, that any explanation which did not include all the elements would be too limited. It does not fall within the scope of these remarks to discuss the question physiologically, even had I anything new to add; but it is worth while to point to two pathological phenomena bearing upon it. What share the friction of the blood, or other possible causes, may have in producing the first sound, I have had no means of ascertaining, and cannot say; but my experience would lead me to regard

many of the difficulties which beset the earliest auscultators, by his correct interpretation of the second sound. Dr. Hope has applied all this knowledge to practice in his doctrine of regurgitation, hereby explaining symptoms which occur in a large proportion of all cases of heart disease, and which must, before his discovery, have been misinterpreted. Meanwhile, by a close scrutiny of the character of particular murmurs, and the circumstances under which they occur, the category of valvular murmurs has been weeded of some that did not properly belong to it. Dr. Ogier Ward has brought an entirely new element into the field by his discovery of what we now call the venous murmur,* and thus supplied us with a means whereby to investigate the nature and causes of coincident arterial murmurs: in the same way, as already noticed, pericardial have been distinguisded from endocardial murmurs.

To our knowledge of the physical conditions for the production of murmurs generally, no entirely new fact has been added, as might, indeed, have been expected; for the theories which still divide opinion were all foreshadowed by Laennec,† though he does not appear always to have chosen the best of them.

Much as auscultation is indebted for the rapidity of its progress to the talent of these original observers, it is no less indebted for the steadiness of its advance to the zeal and industry with which less gifted individuals have followed in their track, to confirm or correct their conclusions by a continual recurrence to the observation of nature. Scarce a stone is laid in the structure but it is marked by the name of him who placed it there, and by the names of those who have tried its stability and soundness in all possible ways. For any statement to be generally received with regard to the diagnosis of disease of the heart, indeed, it needs to be quite indubitable.

Still, with all this, the subject is far from complete, even to the extent that the best auscultators and most accomplished physicians may pronounce disease to be present when it is not—even to the extent that the most confirmed valvular disease may sometimes escape detection.‡ The interpretation of the common valvular murmurs is not yet perfect; and there are some less common signs, such as purring tremor and metallic cliquetis, of which something has yet to be learned. There is reason to think that we know little more than half of the causes of valvular disease, and the progress of that disease on the valves needs to be more fully traced. And the whole sad subject of death by disease of the heart is so continually receiving light from new observations, that its deficiencies, as we have it laid down now, must obviously be very many.

The subject of diseases of the heart is too large to be included in the compass of three lectures: nor, did even time allow me fully to enter upon it, could I ask your attention to my version of what Hope, Latham, Watson, and Williams, have already put forward with more ability and more authority. For the present occasion, the consideration of one part—namely, valvular disease—will suffice; and here, too, rather limiting ourselves to the examination of a few points which still remain open to discussion, than glancing cursorily over the whole subject. I fear that this mode of proceeding may give an unconnected character to all that I have to say, and that less important points may obtain undue prominence, while some of the most essential and most practical points are passed over almost without notice: but the circumstances of the case, and the nature of the materials from which these lectures have been composed, render this mode of treating the subject in some sort necessary. And I would offer this series of essays rather as a commentary on or as supplementary to the more complete treatises with which I may assume you to be familiar, than as constituting in themselves a

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the dull sound of hypertrophy, and the sharp click of dilatation, as illustrating the muscular and valvular elements of the first sound, respectively, in their extreme degrees. I cannot reconcile with pathological observation any theory which would exclude either of these from the formation of the normal first sound of the heart.

* Medical Gazette, vol. xx. p. 9.

† Hope on the Heart, pp. 80-96, et seq.

‡ The Dublin School of Pathology, to which we owe so much in the knowledge of diseases of the heart as in other subjects, published in 1838 (Dublin Journal, vol. xiv. p. 178), in the names of Dr. Graves and Dr. Stokes, a formal expression of the recognised difficulties of the physical diagnosis of valvular disease:—

1. That the physical signs of valvular disease are not yet fully established

2. That, taken alone, they are in no case sufficient for diagnosis.

3 That, even in organic diseases, *the nature and situation* of murmurs may vary in the course of a few days.

4. That all varieties of valvular murmurs may occur *without* organic disease.

5. That organic disease of the valves may exist to a very great degree without any murmur whatsoever.

It is a small matter to express my own present assent to these propositions; but, as it is always harder to profess incompetency than practical superiority, we owe much to Dr. Graves and Dr. Stokes for lending the protection of their names to those who would dare to express a doubt and a difficulty where all may be made to seem so plausible and so easy.

compendium even of that single part of this subject to which I have limited myself.

The subjects for investigation may be conveniently referred to four heads :—

I. The causes of valvular disease.

II. The physical diagnosis.

III. The organic changes on which the physical signs depend.

IV. The general symptoms, and their treatment.

I. CAUSES OF VALVULAR DISEASE OF THE HEART.

The inquiry must rest on morbid anatomy —Influence of age on the results— Various causes—Rheumatism, value of an endocardial murmur during—Difficulties of the inquiry—Congenital malformation involving elementary imperfection—Renal disease distinguished from rheumatism in the nature and situation of the effects—Inflammation of neighbouring parts—Other causes—Inconclusive nature of the evidence.

We have already seen how much has been done in this matter since Andral wrote his classical survey of the existing state of practical medicine. A few years ago we should have deemed this subject better understood than we do now; for Dr. Taylor has added a good deal to our knowledge, and thrown doubt on some of the received doctrines of the causes of pericarditis.* Dr. Barclay has carried the same investigation into the subject of endocardial affections,† to which our attention at present must be limited.

For the purposes of the present inquiry, 181 cases of valvular disease of the heart, disclosed by dissection, have been tabulated. They are divided into two series :— 1. Containing 39 cases where the valvular lesion was the cause of death ; 2. Containing 142 cases where it was of less pathological importance. In some, indeed, of the latter series, the valvular lesion might be looked upon as a mere morbid appearance, which had produced no symptoms during life, and appeared quite incapable of so doing. My friend Dr. Barclay's most elaborate paper, based on 79 cases, with a larger basis on particular points, supplies a means wherewith to compare the results.

The cases selected for the present inquiry have been restricted to those where the existence of valvular lesion has been determined by dissection. A much larger basis of observation, it is true, might have been obtained by tabulating the results of auscultation. It does not, however, appear safe to conclude from auscultations and symptoms alone without dissection. It is needless to enlarge on this point : a single instance will suffice. The results of auscultation and dissection in early life are directly opposed to each other—that is to say, murmurs are most commonly heard at the sigmoid orifice, while disease is most commonly found on the auriculo-ventricular valves. This contradiction depends on the frequent occurrence of functional murmurs at this period of life. They occur, indeed, so frequently as to conceal and even invert the proportion of lesions of the sigmoid and auriculo-ventricular valves which dissection establishes at this period. Unless we can eliminate this fallacy, we must be content to forego all assistance from such a source, as only calculated to mislead, and limit our inquiries to the results of dissection.

The reputed causes of valvular disease are not many. Rheumatism ; the exanthemata (of which I have no experience as such) ; renal disease ; congenital malformation ; want and intemperance ; accident ; inflammation of neighbouring organs : these include all the causes of most common occurrence.

Generally speaking, it may be said that these causes have each a tendency to affect particular valves. Considering the different ages at which these causes are most

Age.	1st Series.					2d Series.					Total.				
	No.	Mit.	Aor.	Tric.	Pul.	No.	Mit.	Aor.	Tric.	Pul.	No.	Mit.	Aor.	Tric.	Pul.
— to 20	9	9	5	1		21	18	12	1		30	27	17	2	
20 „ 30	10	8	4	2	2	21	18	10	1	1	31	26	14	3	3
30 „ 40	9	5	3	3		36	19	24	2	2	45	24	27	5	2
40 „ 50	5	5	3	2		27	20	19	1	1	32	25	22	3	1
50 „ 60	2	1	2	1		22	14	20	3		24	15	22	4	
60 „ —	4	4	3			15	12	11			19	16	15		

* Med.-Chir. Trans. vol. xxviii. p. 453.

† Ibid. vol. xxxi. p. 185.

active, especially (to anticipate thus much) rheumatism and the effects of want or intemperance, it were to be expected that the simple difference of age would show a great difference in the results of disease, according to the predominant action of any one cause during the particular period. These results are set out in the preceding table.

This table shows clearly the preponderance of affections of the mitral valve during the earlier periods. The same appears from Dr. Barclay's table. He takes 34-35 as the point of division, and finds—

	Cases.	Lesions. Mitral.	Aortic.
Before 34	18	17	15
After 34	61	25	37

agreeing sufficiently with the above results.*

What the proportion of diseased to healthy valves at each of these ages may be, I have no means of determining. Of about five hundred dissections, valvular disease of some kind and degree or other was found in one hundred and eighty one cases. But this is probably above the average. For some of these patients were watched for a very long time to obtain an inspection of the heart after their death, and the pains which I myself took, and the assistance I have derived from friends in extending my experience, has been greater in this than in any other form of disease. The results accordingly are disproportionate, and unfitted for exact numerical comparison.

Of the conditions above enumerated there are four which have been found to precede, or coexist with, valvular disease, sufficiently often to allow one to think that the occurrence was more than a mere coincidence, and to give some means for tracing the nature of the connection, namely, rheumatism, congenital malformation, want and intemperance, and renal disease. In the annexed table, the two series of cases fatal by valvular disease (α), and of cases presenting evidence of valvular disease after death (β), have been kept distinct under their several conditions.†

Cause.		No. of cases	Seat of the Lesion. Mitral.	Aortic.	Tricuspid.	Pulmonary
Rheumatism	α	21	18	11	5	1
	β old.	9	8	5		
	β recent.	12	12	10	1	
	Total.	42	38	26	6	1
Congenital malformation.	α	5	3	3	1	2
	β	2	1	2		
	Total.	7	4	5	1	2
Want and intemperance.	α	6	4	3	2	
	β	35	22	26	1	2
	Total.	41	26	29	3	2
Renal disease.	α	12	11	8	2	1
	β	30	25	17	2	
	Total.	42	36	25	4	1

The inference is plain that rheumatism and renal disease coincide with affection of the auriculo-ventricular rather than of the sigmoid valves of either side; but of both valves of the left side rather than of either of those of the right. That congenital

* It is scarcely necessary to do more than indicate one circumstance which limits the application of these tables, namely, that the death of the patient does not commonly occur within exactly the same period as the causation of the disease. Many valves, for instance, injured by rheumatism may not come under the hands of the anatomist till extreme old age; years after the infliction of the injury.

† Two cases, namely, one of mitral and aortic and one of pulmonary valvular lesion, have been entered under both rheumatism and congenital malformation. Some cases entered under renal disease have been also set down under rheumatism, and others under want and intemperance.

My results differ a little from those of Dr. Barclay. He finds rheumatism (op. cit. p. 190) a cause of valvular disease in 15 out of 79 cases, or 18·9 per cent. In the cases analysed above, it was found in at least 42 out of 181, or in 23·2 per cent.

The lesions in my 42 cases (O,) and in 50 (B,) which Dr. Barclay has analysed, were thus distributed—

B.	O.	
In —	4	the mitral, aortic, and tricuspid valves were affected.
—	2	mitral and tricuspid.
41	19	mitral and aortic.
5	13	mitral only
4	3	aortic only
—	1	pulmonary only.
50	42	

From which it would appear that there is not always in acute rheumatism so strong a tendency to affection of both sets of valves on the left side of the heart as occurred in Dr. Barclay's cases. Speaking now of the results of rheumatism as disclosed by dissection at all ages.

malformation, on the contrary, induces sigmoid rather than auriculo-ventricular disease; observing this preference on both sides of the heart, though still affecting the left rather than the right side. The various causes which are included under the name of want and intemperance appear to act almost exclusively on the left side of the heart, and, by a slight preference, on the sigmoid valves. On the effects of three of these causes, namely, rheumatism, congenital malformation, and renal disease, there are a few remarks to be made which will find their most appropriate place here.

Rheumatism.—It is often asked, what becomes of the patients who are dismissed from our large hospitals after an attack of acute rheumatism with an endocardial murmur? In other words, what is the pathological value of an endocardial murmur under such circumstances? The question is very natural and simple, and part of the answer is certain and satisfactory. Some of these patients have no valvular disease at all, the murmur is anæmic or functional, and as they get strong the murmur ceases. Some of them, again, die, at a more or less distant period, of valvular disease of the heart. But the answer, as it relates to the remainder, is, with all its importance, so very obscure, that it will not be superfluous to point to a few of the causes which have continued to keep it in obscurity, in spite of the industry and intelligence which have been for some years bestowed upon the solution of the question. And the same facts may serve to show the grounds on which we form our prognosis under these circumstances.

Practically, the question is little more than one of morbid anatomy. All other sources from which information might be expected are almost entirely closed against inquiry. For comparatively few of the patients who have been the subjects of observation at the beginning of the disease fall again under notice, and still more rarely has the same observer the opportunity of watching the disease through, in one individual. The same remark, of course, applies more or less to all chronic diseases; but it applies pre-eminently to the results of acute rheumatism, as pre-eminently the disease of the poor patients of our hospitals. And difficult as it is to follow up the cases of all hospital patients, yet here another difficulty is added, from the fact that a large proportion of rheumatic patients are young persons of no settled habitations, many of them servant-girls, so that they can very rarely indeed be observed, for any length of time, after they have left the hospital.

We may conveniently divide the history of rheumatic valvular disease into three periods. Our knowledge of the first period, namely, that when the valvular lesion is inflicted, is pretty complete in all its details, for the application of the cause is readily appreciable; and the effects are easily recognized, very characteristic, and of not uncommon occurrence.

Again, another tolerably well-defined period, is that when life fails under the effect, direct or secondary, of obstruction to the circulation by valvular disease; and here, again, our knowledge is pretty complete, for the various lesions may be recognized and interpreted with greater facility than in most other organs; and the symptoms and physical signs are well understood. But between these two periods there is much of the utmost interest and importance almost entirely unknown, and this is the void to be filled up.

As far as my knowledge of what has been expressed on this subject goes, I think that physicians in general have filled up this void in rather gloomy colours; and naturally enough, we come to take an unfavourable view of the question. For we can speak only of what we see and know, and of nearly all the cases of which we know anything, we know that the disease has gone on from bad to worse, till it has destroyed life. And this, too, happens naturally enough, because, so long as the patients are well, they do not offer themselves to observation, and we see nothing, except by the merest chance, of those who have completely recovered. It is only those who have not recovered who fall under notice.

So we come insensibly to look upon an endocardial murmur arising in the course of acute rheumatism as indicative of the existence of fibrinous growths on the edges of the valves, and upon these fibrinous growths as the first stage of the most serious valvular lesions: for several reasons. First: because patients with an endocardial murmur dying during, or shortly after, an attack of acute rheumatism, mostly present such fibrinous growths; then, because many patients coming under observation with confirmed valvular disease of the heart date their symptoms from an attack of acute rheumatism:* and lastly, because the facts to justify a contrary opinion are so few and so liable to error.

All that pathology has to tell us, then, serves but to illustrate the unfavourable half of the answer; it tells us only what is too obvious to remain long concealed. Where there is any thing that can attract a

* Bouillaud (Rhumatisme Articulaire, p. xiij. Paris, 1840,) says that of 300 patients with disease of the heart, more than half dated their symptoms from an attack of acute articular rheumatism.

patient's notice,—the recollection that he could not run up stairs, or that he used to find his shoes tight in an evening, for instance,—there something may possibly be learned. But where health has been restablished; where there are, consequently, no present symptoms for the patient to complain of, nor for the physician to enquire after, relative to the heart, nothing is learned. And this is happening every day, —a great medical truth, the solution of a most important problem, is actually passing through our hands, which we, from our ignorance of the fact, are unable to grasp. From the majority of patients, the subjects of previous rheumatism, who come under notice, nothing is learned. Yet it is not only our want of information as to the fact of the previous occurrence of rheumatism which stands in our way. For even where our attention has been fully turned to this point, the information which can be obtained is small, and the evidence conflicting to a degree of which those only who have been at much pains to collect and estimate it can have any conception. Practically, the question is, indeed, little more than one of morbid anatomy, to which head I would defer its further consideration.

Congenital malformation. — That congenital malformation is a very important cause of valvular disease, especially of that form which there will be occasion hereafter to call the second form, may fairly be deduced from the foregoing table. It will, however, be of interest to dwell for a moment on the mode of connection of these two conditions, as involving a principle of very wide application.

It is a general law, that parts imperfectly developed are, as such, especially liable to disease or spontaneous degeneration. This law applies even to the products of disease, which are themselves obnoxious to further degeneration, in some sort according to the rapidity of their development. Moreover, it may be inferred that the degeneration which such parts spontaneously undergo, or the disease which they take on under the influence of causes insufficient to affect the same parts when normally constituted (call it disease or degeneration, as we will), is that which the same parts, when normally constituted, have a specific tendency to. If this inference be correct, the form of valvular disease to be called the second, which is so well illustrated in these cases of congenital malformation of the sigmoid valves, must be regarded as exemplifying that to which the valves are by nature liable.

In other words, the results of the ordinary processes of decay of normally constituted valves, and the disease which destroys congenitally malformed valves in early life, claim to be considered as identical. Such allowance only must be made for the difference of results as the comparatively greater activity of the process, and the presence of an exciting cause in the latter case, might reasonably demand.

But before assuming this identity on such grounds, it is necessary to meet one very obvious, though, apparently, only superficial objection—namely, that this so-called natural disease is not as common as that whose cause we seem to find in rheumatism or renal disease. The supposition involved in this objection is, I believe, contrary to the real state of things, if the lesser manifestations of each form and at all ages be taken into the account. Yet, were this not so,—were rheumatism the cause of the greatest number of valvular lesions, and those of the first (as they are), and not of the second form,—still the objection is unreal. For no one will deny that rheumatic affection of the valves of the heart is an accident, in the medical sense of the word. However far the tendency to rheumatism, even to rheumatic endocarditis, may be innate in the individual, none can deny that the attack itself, and its results, are purely accidental.* They are the effects of some contingent circumstance, for all that they may be of more common occurrence than the results of natural decay; just as death by old age, even among old people, is a much less common event than is death by the contingencies of bronchitis or paralysis. But for all that, it is the natural end of life.

Renal disease.—The results of renal disease, too, appear to demand a few remarks, not precisely on the same grounds as the effects of the two causes just specified, but to disentangle them from the results of other causes with which, in the tabular analysis, they have become combined. The results of renal disease, as there numerically estimated, scarcely differ from those of rheumatism. But this fact requires, and will repay, a little closer examination as to—first, the frequency of affection of particular valves; and second, the nature of the valvular lesions.

1. First, as to the proportionate frequency of the affection of particular valves.

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* "The laws by which innumerable things happen of the greatest influence upon the affairs and state of the world,—these laws are so wholly unknown to us, that we call the events which come to pass by them accidental; though all reasonable men know certainly that there cannot, in reality, be any such thing as chance, and conclude that the things which have this appearance are the result of general laws, and may be reduced into them."—Butler's Analogy, Part II. Chap. iv. § iii.

On this question the cases of renal disease from Series I. are of no use; for almost all of them had also suffered acute rheumatism. Indeed, they quite contradict some of the inferences from Series II., where these two pathological conditions were as uniformly distinct. To facilitate inquiry, in the following table the numbers have all been adjusted proportionably to one hundred cases; the mean of the patient's age has been taken, and the cases of a corresponding period of life, from a previous table (p. 6), have been reduced to a similar scale. The proportion of valves affected and sound (allowing four valves to the heart), is given in the last column.

Series II.	Age.	Mitral.	Aortic.	Tricus.	Pulm.	Valves per cent. Affected.	Sound.
Renal disease . . .	45·4	83·3	56·6	6·6		36·6	63·4
All other causes . .	40 to 50	74·0	70·3	3·7	3·7	37·9	62·1
Rheumatism . . .	25·6	95	70	5		42·5	57·5
All other causes . .	20 to 30	85·7	47·6	4·7	4·7	35·7	64.3

The distinctions between the effects of renal disease and those of rheumatism on the endocardium, thus numerically estimated, are tolerably clear. Renal disease does not induce quite so large a per centage of valvular lesions as do all the diseases whose combined results in this particular were examined at the same period of life. Singularly, no rheumatic case falls within these limits of age—forty to fifty—in my cases, and only one in Dr. Barclay's cases. Comparing, therefore, for want of any other data, the total effects of the two causes, regardless of the difference of the age at which they each most distinctly show themselves, the per centage of valvular disease induced by renal disease appears very much lower than that induced by rheumatism. On the same grounds it appears that renal disease affects the mitral rather than the aortic valves in a higher proportion than even rheumatism does.

But while thus numerically estimating the effects of rheumatism and renal disease severally, we must recollect how common a complication of renal disease sub-acute rheumatism is. The history, indeed, of the cases tabulated from Series II. represents the two affections as occurring independently in the particular instances. And the numerical conclusion from such representation cannot be gainsaid. I owe it to those who have expressed their opinion on this subject to show how far the results of my observations agree with theirs. But I should be expressing an assurance which I do not feel were I not to add, that the facts on which the comparison is based do not bring conviction to my own mind; for in many cases the occurrence of rheumatism may have been overlooked.

The same remark applies to the effects of want and intemperance, with which, as might be expected, several cases of renal disease in Series II. coincide. The positive statement of their having been in want or dissipated is, unhappily, only too trustworthy. But this very fact much detracts from the value of the negative assertion concerning the non-occurrence of rheumatism. It places them in a class habitually exposed to disease of all kinds, and notoriously unobservant of their symptoms.

2. Secondly, as to the nature of the lesions, as far as we may go without anticipating what will form the subject of inquiry elsewhere. In Series I. the renal and rheumatic cases are nearly identical; and the morbid products must be considered in so far as the common result of the two causes. And so far, almost uniformly, what there will be occasion to describe as the second form of valvular disease prevails, and in its most extreme degree. In the renal cases of Series II. the results are much less striking, as might naturally be expected. But the changes have a character of their own. In their description the terms of opacity and general thickening of the valves are of the most common occurrence; while in that of the rheumatic cases the square edgings of the valves, and the concentration of the lesions on the edges of contact, are more particularly noticed.

To deny that renal disease has any influence in inducing valvular disease would be contrary to the fair inference from these observations. But I cannot look upon its independent influence as of any very great importance in this point of view. It is as a modifying rather than as an original cause of valvular disease that it seems to me to possess any high pathological interest. Dr. Barclay, however, in his valuable paper already quoted, would give renal disease a higher place among the causes of valvular disease than my observations allow me to assign to it. He has also met with it more frequently—namely, in forty-one out of seventy-nine cases, or about 52 per cent.—it occurred in not more than 23·2 per cent. of my cases.

There are still a few conditions whose claim to the consideration of causes of valvular disease requires to be investigated.

The occurrence of inflammation in a neighbouring part—chiefly the lungs or pleuræ—is another reputed cause of inflammatory affections and consequent organic disease of the investing or lining membrane of the heart. The grounds of this opinion, as far as concerns the endocardium, do not appear to me to be satisfactory.

There are necessarily but two sources whence the evidence can be drawn,—namely, cases of recent inflammation of these parts, or, cases displaying traces of its former presence. Neither of these appear to me to furnish the required data.

I. As to previous inflammatory affections of neighbouring parts. Practically, we are almost limited to the observation of the traces of such previous action in the pleuræ; for the traces of previous pneumonia are confessedly of too equivocal a character to be made the basis of any further inferences. Now, in a very large majority of cases of pleural adhesions, of whatever extent, tubercles are found in the lungs. But the admitted antagonism of the tubercular diathesis* and valvular imperfection, and the equally admitted fact of the close connection of pulmonary tubercles with pleural adhesions, must tend very much to diminish the evidences of the connection between pleural inflammation and valvular disease.

Notwithstanding this large deduction, the proportion in which valvular disease of the heart, and the traces of old pleurisy, are found to coincide, is certainly higher than that which is found to exist in patients dying of all disease, including pulmonary phthisis. In the examination of the bodies of 486 patients, dead of all diseases, of which I have more or less complete records available for the present purpose, pleural adhesions were found in 225; less than one half. In 182 of these 486 patients, who presented evidences of valvular disease of the heart, of whatever kind or degree, pleural adhesions were found in 94; rather more than one half. But before accepting the conclusion from these numbers, we must allow much for the greater attention bestowed on the examination and description of the thoracic organs, in cases of fatal thoracic disease. A fact this, which would obviously raise the proportion in which pleural adhesions have been found in connection with valvular disease of the heart.

Granting, on the grounds of their frequent coincidence, a more than accidental connection between the traces of old pleural adhesions and old valvular disease, we are yet very far from being entitled to assume that in the pleurisy we see the cause of the endocarditis. They may have a common cause; as they have, for instance, in cases of rheumatic pleurisy. We cannot hope to arrive at any certain conclusions, without a certain knowledge of the cause of the pleurisy, or at least of the date of its occurrence. I cannot, therefore, put any confidence in the deductions from cases of old pleural adhesions only discovered after death, in our present, and what must be, I believe, our perpetual state of uncertainty concerning the occasion of their origin, in the great majority of cases.

II. The examination of recent cases of pulmonary inflammation makes very much against the existence of any close connection between inflammation of the endocardium and that of the lungs or pleuræ. I have more or less complete notes of the dissection of eighty-eight cases dying with recent pleurisy, and of ninety-five with recent pneumonia. Several of the cases of these two affections are identical; but for the present purpose it is needless to enter into any elaborate analysis, and to set out this distinction with accuracy, for, out of the whole number, only six had coincident endocarditis. Five of these six cases had at the time acute rheumatism. The sixth died of meningitis. In her the existence of pneumonia was questionable, and the inflammation of the endocardium was limited to

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* Rokitansky, who has brought a larger mass of facts to bear on this subject than have hitherto been employed in its examination, speaks very decidedly of the antagonism between tuberculosis and valvular imperfection (Path. Anat. Bd. i. S. 22). But he finds more reason to connect his "pale yellow granular [croupös] tubercle with disease of the valves of the heart, and of the great vessels (S. 524).

Dr. Walshe, in his able report on phthisis (Brit. and For. Med. and Chir. Rev., Jan. 1849, p. 252) says, "I have never once seen cardiac disease, of such kind as to cause hæmoptysis, coexistent with *phthisis*, using the term in its practical sense; but in a fair number of instances I have seen advanced cardiac disease, in persons whose lungs contained *crude tubercles and grey granulations*. It may be, therefore, that the conditions of the system existing in heart disease are unfavourable to the development of tubercle; but the unfrequency with which the two kinds of disease are found together, doubtless depends, in the main, on the difference in the periods of life at which each is especially prone to occur."

In the notes of my own dissections the number of cases of valvular disease is, from various causes, disproportionately large. But, where this peculiarity does not interfere, the numerical conclusions agree pretty closely with the above.

Tubercles or their remains were found in the lungs of

2 in 39 = 5·1 per cent. who died of valvular disease.

31 in 145 = 21·3 per cent. who had, but did not die of, valvular disease.

142 in 495 = 28·6 per cent. who died from all causes with all diseases.

Valvular disease, of whatever kind or degree, was found in about 30 per cent. of patients having tubercles in their lungs; 37 per cent. of all cases, with or without tubercles.

vascular injection of two patches of the mitral valve. It appears, therefore, that inflammation of the lungs and pleuræ, apart from the cause of that inflammation, have little to do with producing endocarditis.

My experience of acute non-rheumatic pericarditis is too limited to allow me to speak decidedly on the subject; but, such as it is, I believe inflammation of the investing has little tendency to spread to the lining membrane of the heart, apart, as before, from the original cause of the pericarditis.

So, too, with regard to purulent inflammations of, or depositions in, the areolar tissue in the neighbourhood of the heart. My own more limited experience in this matter would show that such processes have no more tendency to induce endocardial affections, than have inflammations of the same nature occurring anywhere throughout the body.

The question, how far the effects of a previous attack of endocarditis may be looked upon as the cause of a second or other attacks, is more complex. Indeed, it requires for its complete solution that we should know the exact history of endocarditis as a morbid process; to the discussion of which part of the present subject its consideration must therefore be deferred.

On the connection, in the last place, between atheromatous disease of the arteries, and valvular disease of the heart, the records of my dissections do not allow me to offer any remarks, for atheromatous disease is not noted in my dissections, unless when of considerable amount. And being of almost universal occurrence in some degree, it would be necessary to have a very carefully graduated table of its amount, if we would trace its connection with any particular condition. Such I have not at command. Only, of the cases where great disease of the coats of the arteries was noticed, valvular disease was found in more than half. And the form of valvular disease noticed under these circumstances, was, almost without exception, of that kind which has already been alluded to as the second; which is coincident also with renal disease, and with want and intemperance.

Before leaving this subject, I would ask your attention to one remark on what has been said. It is needless to explain the difficulties which beset any inquiry resting on the statements of poor hospital patients, as this does. And I should be claiming great merit to myself if I said that. I had surmounted them. I do not apologise for my numbers being so small, but beg you to accept with caution what, with the utmost care, may conceal important errors. And this the more with regard to those conclusions which are at variance with those which Dr. Barclay has made in his valuable essay on this subject, already so often referred to. It is a matter in which we do well to be cautious, and hesitate before we accept any inference as true, and admitting of further application.

Though there were no reason to doubt the conclusions above drawn, yet it would be almost impossible for one observer, in one life, to prove their correctness. For the greater number of diseased valves are found on dissection, not of patients where the one assigned cause, and that only, has operated for a known period, but of patients dying of chronic bronchitis or anasarca many years after the infliction of the original injury. These, too, having been exposed intermediately to all the causes usually considered as capable of inducing valvular disease of the heart, such as habitual intemperance, violent muscular exertions, and all the distresses and privations of a poor man's life. Besides, the assertion that these patients have or have not ever suffered from acute rheumatism is rarely sufficiently trustworthy to be taken as a basis for any calculation. Much less so is their statement as to whether symptoms of affection of the heart were noticed during such an attack of rheumatism. Sometimes even auscultation fails to supply us with the exact date of the commencement of valvular disease; as, for instance, when the organic lesion has happened not to interfere, at its commencement, with the action of the affected valves.

LECTURE I.—Part II.

II. Physical Diagnosis of Valvular Disease of the Heart.

Valvular Lesions distinguished by the position of Murmurs—Secondary importance of the intensity o tone of Murmurs, or of the measurements of the exact position of the Heart.

1. *Organic Endocardial Murmurs—Distinctions between Tricuspid and Mitral Murmurs obscure—between Pulmonary and Aortic, practical utility of.—Direct Mitral—double Mitral—Bruit de rappel—Murmurs audible at a distance—Mechanism of Valvular Murmurs—of Murmurs not Valvular—Muscular Sound—Pericardial—Metallic Cliquetis—Purring Tremor, its Nature and Pathological Import.*
2. *Functional Endocardial Murmurs—Fallacies of Cardiac Auscultation—corrected by Pathology—General and Local Causes of Murmurs to be distinguished—Abstract meaning of Murmurs always the same—the observation more often correct than its interpretation.*

If the physical diagnosis of valvular murmurs had no higher object than the reference of each murmur to its proper seat, we might dismiss the subject in a few words. For the rules of diagnosis are clearly laid down, and when those can be applied, the diagnosis follows directly, as a plain inference. But in a higher point of view,—namely, as embracing the interpretation of such an inference, and forming the grounds of our prognosis,—the interest of the subject is as great as its importance and its difficulty. For it involves the *vexata quæstio* of the diagnosis of functional and organic affection of the heart; and, besides all the niceties of particular valvular diagnosis, the recognition of the effects of such valvular disease on the heart itself.

The fundamental law on which all diagnosis of the lesion of particular valves rests, is, that murmurs are heard loudest in certain parts, and propagated loudest in certain directions, according as they depend on lesions of different valves. When the time and position of a murmur can be exactly determined, its interpretation may be correctly made, according to established rules. Even if there be two or more murmurs coexisting in the heart of the same individual, so long as their time be determinable, and there be an appreciable distance between their positions of greatest intensity, this most complex case may be unravelled. This is not merely a statement handed on from one physician to another, but a fact of the truth of which any one may assure himself by careful observation in the wards and dead-house of any large hospital school. And it is most important that he should thus assure himself of it, for all the practice of cardiac auscultation hinges upon it.

While claiming the highest importance, in a diagnostic point of view, for the position, and next, for the time of murmurs, the information derived from the intensity or tone of murmurs must be entirely rejected, or received with great caution. For between these and the amount or character of the lesions on which the murmurs depend, there would appear to be no direct relation; in the former particular, indeed, the ratio is rather inverse.

As concerns the intensity of the abnormal sound, the loudest murmurs may be heard where there is no organic disease, and contrariwise, the amount of organic disease may be such as entirely to preclude the development of a murmur, either at the seat of such extreme disease, or lower down in the course of the circulation. And although a murmur ordinarily becomes feebler before it disappears, yet it does not certainly follow because it becomes feebler that therefore it is about to disappear, and that the valve is recovering itself.

So, again, with regard to the character of this sound, the loudest, shrillest, murmur may arise from anæmia; the softest blowing whisper may ensue from the passage of the blood through a rugged chink. And the same musical note may be heard, with all conceivable states of the valves, from an apparently healthy condition, up to rigid ossification of the parts. There are, then, no certain grounds for connecting the harsher murmurs with the more serious lesions, or for inferring from the diminution of the harshness, as before from that of the intensity of a murmur, that the valve is recovering itself.

It would be very desirable to have a code of signals, so to say, by which we might recognise particular forms of disease, or watch their daily progress. But certainly, neither the intensity nor the tone of the endocardial murmur can be safely trusted as furnishing any such. And, indeed, there

is nothing more in our confessed inability to draw such practical inferences from these grounds, than what an abstract consideration of the premises would have led us to anticipate. Doubtless there is a cause for each modification of each sound; but analogy would lead us to look for this cause anywhere rather than in the nature of the walls of the aperture of sound. At least within certain limits.

For by varying the form of the aperture, or altering the mode of propulsion of the current of air, we can elicit a wide range of sounds from an instrument composed of almost anything capable of being thrown into sonorous vibrations at all. Or conversely, an equal variety may be elicited by varying the mode of propulsion of the current, or the length of the tube leading to or from the aperture, while the aperture itself remains unaltered. And the range we can obtain by either of these methods, on any instrument, as far as I know, exceeds, both in tone and pitch, the limits within which the variation of endocardial murmurs is found to occur.

The case, then, stands thus. Briefly, in Dr. Latham's* words, "Experience does not countenance the belief that the *kind* of endocardial murmur follows the *kind* of endocardial disease." Nor, abstractedly considered, and by analogy, have we any reason to think that it should. But, apart from all such general considerations, the daily, nay the hourly variations of the character of endocardial murmurs, in some cases, show that these differences of tone or pitch depend on some cause capable of rapid change, rather than on fixed organic disease. Probably, differences in the mode of the heart's contraction, or in that of the coaptation of the valves, are sufficient to explain all these varieties. Probably, from the fact that these temporary variations occur during the systole, and at the auriculo-ventricular valves. For where the form and mode of action of the parts is not so susceptible of physical modifications, as, for instance, during the diastole, and at the sigmoid valves, though we have the same range and variety of sounds, taking one case with another, yet these variations do not all occur in the same person. Under the circumstances supposed, the limits of the scale are much narrower.

The truth is so very obvious here, on a little reflection,† that it may seem almost trifling to have dwelt so long on exposing this source of fallacy. And it may seem to be throwing needless difficulties in the way, by thus parading all our weaknesses and shortcomings in the foreground. But we are still inclined to interpret before we understand, to take up with a plausible error rather than to rest in doubt, and so far in truth. As Thucydides* told us long ago, "*Ουτως ἀταλαίπωρος τοῖς πολλοῖς ἡ ζήτησις τῆς ἀληθείας, καὶ ἐπὶ τὰ ἑτοῖμα μᾶλλον τρέπονται.*" And our minds need, no less than our eyes, to be warned at each moment not to trust to what is intrinsically unsound, however specious.

The determination of the exact position of the heart in the thorax has lately occupied a good deal of attention; more, apparently, than the intrinsic importance of the subject could command. For the position of the heart is not so uniform in all cases, as that we need to inquire into the cause of each lesser deviation. The hand and the ear are the best guides to the relative or actual position of the heart, in the individual case; for they tell us not only the present situation of the heart, but the nature of the cause, if so it be, of its displacement. If we can neither feel the apex of the heart, nor ascertain the point of greatest intensity of the first or second sound, all accuracy of diagnosis is at an end. But if these can be made out, we want no further information, than what an ordinary knowledge of anatomy supplies, for our educated senses to explain the condition of the parts in their natural or altered relations.

But let me not be misunderstood as seeming to underrate the value of these researches, in confessing my own inability to apply their results to cardiac pathology; or, as judging of their applicability in other hands, and to other diseases, from what they have appeared to me in diseases of the heart. Whatever value may be assigned them, as contributions to practical medicine, it is impossible not to appreciate most highly the talent and industry with which the investigation has been conducted by Dr. Sibson,† who has placed our knowledge in these particulars on so much more sure a foundation.

Such is, I believe, a just estimate of the comparative value of the indications afforded by the time, situation, tone, and intensity, of murmurs presumed to be endocardial. Having thus generally glanced at these preliminaries, it remains to apply them to the more particular examination of some of the physical signs of valvular disease.

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* Op. cit. p. 51.

† Bouillaud has most happily expressed, in words which lose their force in a translation, the difference between the musical and the ordinary blowing murmurs. "Il y a la même différence qu'entre l'action de souffler et celle de siffler."—Mal. du Cœur, tom. i. p. 196, 2d. edit.

* De Bello Pelop. A. 20.

† Medical Gazette, 1848; Prov. Med. and Surg. Trans. vol. xii.; Med. Chir. Trans. vol. xxxi.

Endocardial murmurs, distinguished according to their cause, are of two kinds,—organic and functional. On each of these there is something to be said. It is difficult to separate what are in nature so inextricably involved as these two subjects. It appears best, therefore, to speak of murmurs, in the first instance, in their probable relation to organic disease, and then to subjoin such considerations as diminish or destroy their pathological importance in particular cases, reducing them to the category of functional murmurs. And first of—

I. Organic Murmurs.

It requires no particular skill, in an ordinary case, to determine whether a murmur be heard loudest at the base or the apex of the heart, and with the first or the second sound. But it is much more difficult to get beyond this, and to discriminate between the affections of the right and left valves respectively at these points.

There is no accurate unfailing rule by which we can always distinguish mitral from tricuspid murmurs, so short is the distance between the two points where these valves respectively give evidence of their imperfections. A forcible pulsation, with extended dulness to the right of the lower end of the sternum, combined with the general signs of venous obstruction, has sometimes, to my experience, correctly indicated the existence of functional imperfection of the right auriculo-ventricular valve. Andral* notices such an occurrence. But tricuspid regurgitation is said by those who have paid most attention to this subject to be rarely accompanied by a murmur.† The inference would be, that a murmur heard loudest at the apex of the heart is pretty surely referable to imperfection of the mitral valve. Assuming the correctness of this inference, still the determination of the exact situation of greatest intensity of murmurs heard loudest about the apex is important; for by this means we may usefully subdivide the mitral murmur itself. For, judging from the results of morbid anatomy and auscultation alike, there appears an important difference between auriculo-ventricular murmurs, according as they are heard at the very apex, or a little above it. Thus, a murmur heard loudest about an inch to the right of, and rather above the situation where the apex of the heart pulsates, may be a mitral or a tricuspid murmur; may depend on a fixed organic, or on a functional cause: it is, in fact, subject to all the fallacies of cardiac diagnosis. But a murmur heard loudest at the apex itself, and thence transmitted up the side of the chest to the left of the mamma, admits of much less doubt. With only one exception, I have never found this to depend on anything but organic disease of the mitral valve.*

At first sight the diagnosis of pulmonary from aortic murmurs might seem much more difficult than that of mitral from tricuspid murmurs. But it is not so. The best proof of its practicability is the fact that in the only three cases of disease of the pulmonary valves which have fallen under my knowledge, where the heart has been examined during life, a correct diagnosis has been made by three different observers. The valves themselves, it is true, are so very near to each other, that, by the examination of their region only, no accurate information could be obtained. But as the vessels diverge from each other, the signs referable to each may be examined separately at the distance of half the width of the chest, or even, in some cases, the vessels of the neck, and the ramifications† of the pulmonary artery in the lungs, may give their independent testimony to the easy practicability of the diagnosis. Most commonly, however, all that is attainable is the appreciation of the more superficial character of the sound over the valves themselves, and of its distinct propagation along a line from the sternal end of the third left intercostal space for two or three inches towards the middle of the left clavicle.

* Clin. Méd. iii. p. 154.

† Dr. Blakiston on Diseases of the Chest, p. 226.

* I feel so sure of the fact, that I am averse to mixing it up with what may prove an erroneous explanation; but the following seems the most probable:—The chordæ tendineæ of the lesser flaps of the mitral valve are inserted, by means of their carneæ columnæ, more to the left, though, indeed, further from the apex than those which retain the larger flap on Lieutaud's valve. By the law that the signs of valvular lesion are transmitted loudest in the direction of the attachments of those valves, we might expect to find a difference according as one or other of the flaps of the mitral valve were affected: and hence it is fair to suppose that sound caused by regurgitation through the mitral orifice, being transmitted equally along all the chordæ tendineæ, will be equally audible about the attachments of them all; while sound produced only on the larger flap will pass down only its own chordæ tendineæ, and therefore be traced less distinctly to the left than in the former case. The murmurs, then, heard more to the left than usual, would, in this view, most likely be due to regurgitation. Those heard in the ordinary situation—that is, rather to the right of the apex—might arise either from this cause, or from the blood being thrown into vibration as it runs up into the aorta against the chordæ tendineæ, traversing the ventricle in that direction. In this last case there would be many chances against their being organic; in the former there would be everything in favour of such an explanation of their origin. But all this may or may not be the true explanation. The fact is as above stated.

† See Med.-Chir. Trans. vol. xxiii. p. 352; and on the whole of this subject, Ed. Med. and Surg. Journal, vol. lxv.

This, however, is quite sufficient for the diagnosis in those cases where the entire absence of the murmur from the course of the ascending aorta, or from a corresponding situation on the right side of the chest, clearly shows it not to have its existence in that vessel.*

The determination of the existence of a murmur in the pulmonary artery is, in fact, easy enough. The distinction, however, is not urged here merely as a refinement in the particular diagnosis of valvular lesions, but rather in relation to the most interesting, yet most obscure part of cardiac pathology—the distinction between functional and organic murmurs of the heart, to much of which I believe that this is the key. For, under these circumstances, a comparison of different observations—that is, of auscultation—tells us something more than that the murmur is here or there; practising the ear like intricate scales in music, but leading to nothing beyond. It tells us something of the cause of the murmur: for disease of the pulmonary valves is so exceedingly rare, that in an enormous majority of cases we may certainly infer that pulmonary murmurs are independent of organic changes. The recognition of a murmur in this situation inclines to a favourable interpretation of all other murmurs in the particular case; for it renders probable the present active existence of a cause, other than organic disease, adequate to their explanation. At the same time, it supplies additional means for ascertaining the correctness of such an interpretation: for it increases the field of observation of the variation of murmurs. For instance: if a murmur be heard always at the base of the heart, the presumption is, that it depends on an abiding—to wit, an organic cause. But if, though always audible at the base, thus generally expressed, it be sometimes audible in the aorta only, and sometimes only in the pulmonary artery, the case is quite altered. All the doubt and anxiety which hang over the long continuance of an endocardial murmur are dispelled, when that murmur, as under such circumstances, is clearly shown to be independent of organic changes of the valves.

Without exploring the subject of probabilities to determine what is the exact diagnostic value of the fact of the recognition of a murmur in the pulmonary artery in relation to other co-existing murmurs in particular cases, it may be said, for all purposes of practice, to be very considerable. But it would be better for each one for himself to determine its just value, than to rely on any general inferences, where the consequences of an error may be so serious. It may appear very irrational to think one way and to act another; but however justly we may be assured that the frequency of affection of the heart in rheumatism is much overrated, still we are not justified in withholding the appropriate treatment from particular cases on the strength of such a belief. And this, too, though we may seem to hold the right clue in our hands: for the same process — namely, the rigorous investigation of the subject with the stethoscope and the scalpel, which assures us that the right valves are generally healthy—proves how very often the left are diseased.

The safe rule for practice, as applying to acute cases where the lesion appears to be progressive, is this:—A pulmonary murmur, if it be the only sign of endocardial lesion, may, under almost all circumstances, be disregarded: but when co-existing with an aortic or mitral murmur, its co-existence should not blind us to the possible importance of those other murmurs. It is true that the signs from the left valves *may* mean as little as those from the right, but they *may* mean much more; and in neglecting them we *may* be losing the favourable moment for treating organic disease.

Sinking, however, the use of the recognition of a pulmonary murmur to its lowest degree—that, namely, of giving us a hint often, and an assurance now and then, still, recollecting how obscure these cases often are, we may often turn it to the best account. Keeping within every limit of even the most superlative caution, still I believe the value of the recognition of murmurs in the pulmonary artery to be very great; and having for some years paid much attention to the subject, have found no reason to deviate from this opinion formerly expressed.*

It is scarcely possible to cultivate any part of a subject like practical medicine for any length of time without obtaining information, either in itself new, or as a new form of expression or confirmation of a recognized truth; and the practice of St. Bartholomew's Hospital seems to have supplied two or three instances of the latter in connection with the present inquiry. It appears better, on the present occasion, to regard our knowledge of the signs of valvu-

* Dr. Latham notices the frequent occurrence of a murmur with such negative evidence of its being in the pulmonary artery in cases of phthisis. Op. cit. vol. i. p. 66. See, also, MED. GAZ. vol. xx. p. 9. Dr. Ogier Ward.

* Ed. Med. and Surg. Journal, vol. lxv. Two rheumatic cases there mentioned in the second table have since come under notice. In one the murmurs had all disappeared; in the other the apex murmur and the murmur in the lungs had disappeared, and the existence of a murmur at the base was very questionable. She was then, however, suffering from another attack of rheumatic fever.

lar disease through this point of view, than to recapitulate what I will not merely say you already know, but what you have had a large share in originally discovering or establishing.

One of the rarest of all morbid cardiac sounds is the direct mitral murmur; and its origin is attributed, as its name implies, to obstruction to the onward passage of the blood through the mitral orifice. Here is a case in point, showing the correctness of the general opinion:—

A woman, past middle age, was admitted with anasarca, and albuminuria, and bronchitis. Her heart for some time presented no abnormal sound, but about three or four days before her death a murmur was audible with the second sound at the apex. On examination of the body after death, the left ventricle was found dilated; and at a short distance beneath the ring of the mitral valve, with its edge projecting into the onward stream of blood, there was a large coagulum adherent. It was evidently some days old; it had the form of an obliquely-truncated cone, and the upper edge presented to the stream of blood an angle of about forty degrees. In connection with a severe attack of neuralgia which she had suffered from three years before in the right leg, it is of interest to notice, in passing, the existence of a few cartilaginous flakes on the posterior roots of the nerves on the same side of the lower end of the spinal cord.

This single diastolic murmur at the apex is exceedingly rare. I never met with more than one other instance of it. But a double murmur has been recognised at the apex in eighteen cases. Out of these eighteen cases only four are known to have died,—one during the period of observation, and three at subsequent periods. Eleven of them left the hospital much relieved. If, in the absence of more certain data, and of the observation of single cases watched through the whole course of their disease, it be allowable to infer from the greater frequency of murmurs at one period of a disease than at another, a very curious conclusion follows with regard to the murmur under consideration. For, on these grounds, it appears to be a more common sign at some distance from the fatal period than during the disturbance of the heart's action which more immediately precedes death: since, out of many scores of observations, in only this one has the double murmur been heard about the period of dissolution.

The lesion on which this combination depends would seem, however, to be very serious, judging from the symptoms. And in the only dissection which has been obtained, two years subsequent to the last auscultation, the amount of the changes was certainly very striking. This patient had annular contraction and thickening of the mitral and tricuspid valves in the most extreme degree.

The singularity of any sign is scarcely a sufficient cause for detaining us on the present occasion. So the reduplication of the first sound at the apex,* which Bouillaud has denominated the *bruit de rappel*, claims here only a passing mention, and this only lest it should be confounded with the double murmur at the apex, just noticed, to which it bears so close a resemblance. The distinction, however, is important rather in theory than in practice; for my own more limited experience agrees with that of Bouillaud in connecting it, like the murmur last noticed, with extensive disorganization of the heart. Still, as depending rather on the hypertrophy which that disorganization has induced, than on the cause of the hypertrophy, it is in so far a sign of less importance than is the double valvular murmur at the apex above described.†

We need some better term for it than that in common use, namely, the reduplicated first sound.‡ The French name is much more expressive.

Sometimes an endocardial murmur may be heard by the patient himself or by other persons at a considerable distance from his body. The most probable explanation is that suggested by Laennec,§ who connects this phenomenon with the accidental circumstance of the vessel where the murmur originates lying in the neighbourhood of a cavity full of air. This is the generally received opinion; and it was well borne out by the most striking case of this kind which has come under my own observation. In connection with a murmur audible at a considerable distance from the patient, a large aneurism was found immediately over

* Bouillaud, Des Maladies du Cœur, tom. i. p. 213.

† Dr. Williams "has heard it sometimes where there was no reason to suspect permanent lesion, but where the action of the heart was languid."—Diseases of the Chest, p. 211, 4th ed.

‡ Sometimes the second sound is reduplicated over the sigmoid valve: apparently from the time of closure of the aortic and pulmonary valves not exactly coinciding.

§ Aus. Med. tome iii. p. 133. Dr. Abercrombie, Ed. Med. Chir. Trans. vol. i. p. 48, mentions a curious circumstance, which was noticed in one of his patients, of interest in relation to this subject. "He attempts to relieve his uneasiness by frequent and very deep inspirations..... While the lungs are inflated in the state of full inspiration, a sound is heard by himself, and by a person sitting near him, exactly resembling the loud tick of a watch: it corresponds in frequency with the frequency of the pulse, and is only heard while the lungs are fully inflated; but it continues to be heard as long as he keeps them inflated, by resting upon the deep inspiration."

the bifurcation of the trachea. Cases, however, are continually occurring where no such disposition of neighbouring parts is found to explain this peculiarity of the murmur.

It would seem that the most definite meaning which we can attach to this peculiarity of being audible at a distance from the chest, relates more to the situation than to the cause of the murmur. In this way, though it gives no fixed grounds for modifying the prognosis of the cases in which it occurs, yet, as regards diagnosis, its recognition might be useful. For, recollecting that a stomach distended with air may conduce, just as well as the trachea or bronchi, by its proximity, to the manifestation of this peculiar sign, we may look here for an explanation of the cardiac symptoms which sometimes accompany an attack of dyspepsia. For, the same cause which, by distending the stomach, disturbs the heart's action and causes the murmur, may also make the murmur audible at a distance from the patient. But in illustration of this I have not at command a case sufficiently in point to offer to your notice.

The study of the exact mechanism of abnormal murmurs is one of exceeding beauty and interest, though it must be confessed that it is wholly inapplicable to treatment. Why these murmurs should come and go, what are the causes of their variety, why in this the stress should rest on the beginning, in that on the end, are all questions which arise so often, that, out of sheer curiosity, to rank the feeling no higher, one is very anxious to have a satisfactory solution of them. A means of imitating, after death, the exact mode of play of the mitral valve during life is here the great desideratum. For there is reason to think that many of the varieties of particular cases may arise from peculiarities in the form of the lesion, rendering this valve (in relation to which the varieties most often occur) inefficient at particular stages of its action. Some of these differences, however, admit of explanation.

The commonest, and probably the most important defect in the mitral valve, is where the edges do not fit after the valve is shut to; and where, consequently, regurgitation takes place through the aperture during the contraction of the ventricle till the complete obliteration of its cavity. A murmur indicating this condition, however arising, whether from thickening or perforation of the valve itself or from shortening of its chordæ tendineæ, begins suddenly with the first sound, and fades gradually until the occurrence of the second sound, which coincides with the cessation of the contraction of the ventricle. But sometimes the murmur at the apex, instead of suddenly rising to its maximum of intensity and gradually declining, rises less abruptly, but suddenly falls with a smart click and jerk sensible to the hand all over the cardiac region. From careful examination, it appears that this click and jerk are really the termination of the first sound, which the murmur accompanies, but does not follow. It may hence be inferred that regurgitation takes place with a murmur through the mitral aperture until the walls of the ventricle have approximated so that the edges of the valve can meet. The valve, being in itself sound and efficient, now precludes all regurgitation, and checks the stream suddenly, thus sending back an impulse nearly equal to that of the contraction of the left ventricle, only more sudden; similar to what we may produce by suddenly checking the flow of a stream of water or air through a tube.

It may here be objected, by those who only care to establish the fact that there is disease of the heart in a particular case, that this is a useless refinement. And the force of the objection could not be denied, were this refinement the only object attainable. But, with time to observe, and an unlimited field of observation, the interest of solving one of these problems is very great. And the ear, taught in this difficult and complicated inquiry, finds itself more perfectly at ease among the ordinary incidents of daily practice, where more important results than the solution of curious problems hang on its accurate perceptions: "For it breeds great perfection if the practice be harder than the use." I cannot meet this objection in more forcible words than those of Dr. Elliotson on the same subject.* "To condemn accurate diagnosis is to condemn accurate knowledge,—to rest satisfied with imperfect information, when industry would give us more, is to admire ignorance when knowledge is within our reach."

By thus accurately determining when and where murmurs are loudest, and the character and history, so to say (though the history be that of less than half a second of time), of each murmur, we learn to separate several sounds from the category of indications of valvular disease. It appears worth while to notice a few of those of most common occurrence in practice. Such are the muscular murmur, if it be correctly thus designated, the pericardial murmur, and metallic cliquetis. And to these have been subjoined a few remarks on purring tremor in its relation to organic

* Lumleian Lectures, p. 5.

disease, anticipating as little as might be the observations which this sign seems to require in relation to functional murmurs.

1. *Muscular sound.*—Sometimes a systolic murmur may be heard with equal intensity over the region of the ventricles, which is not transmitted at all along the great vessels, as its loudness, even up to the base, would have led us to expect. A closer examination of this murmur confirms the inference which would follow from the above particulars. It has no abrupt beginning or ending, its time coincides exactly with that of the contraction of the heart; and the sound is so like that of muscular contraction, that it is impossible, on all these grounds, not to believe it to have its seat really in the muscular structure of the heart. Why it should be audible only in some hearts, and only for a limited time, is not clear. But as occurring in patients who are the subjects of acute rheumatism, it is important to separate it from their valvular murmurs, which are of longer continuance and more serious importance. Be it muscular or not, it is worth while to distinguish it from the admitted endocardial murmurs, if only for the reason that it goes away and leaves the heart, in all its functional, and, for what I have been able to learn, in all its organic integrity.

2. *Pericardial murmur.*—There is another murmur which, by careful observation, may be shown not to be valvular but pericardial. The anatomical history of the white spots so often seen on the pericardium has been satisfactorily made out. Mr. Paget has adduced proof of the correctness of the common opinion, that they are generally the result of previous inflammation.* But their clinical history is still incomplete.† Of the white spots themselves I have nothing to say; but, with regard to the pericardial adhesions about the base of the heart, on which their correct interpretation depends, a few cases worthy of notice have come under my observation. In some patients a simple systolic murmur, of the character of a friction sound, has been audible over the first part of the pulmonary artery for several days in succession. These were generally the subjects of acute rheumatism,—women, and anæmic. Was this seeming friction sound really the sign of the existence of this limited pericarditis? I think it was; but death so rarely occurs during the progress of acute rheumatism, that I have been unable to obtain actual demonstration of the fact. The following case, however, seems in point:—

Sarah Emmet, aged 13, a large, overgrown girl, came into St. Bartholomew's Hospital, in April 1845, with acute rheumatism and endocarditis. On the second day after admission she is described as having a harsh systolic murmur all over the region of the heart, most audible at the base, and about the third left costal cartilage, of a rustling character, but nowhere double. The systolic murmur at the apex, first heard two days before, continued unchanged. There was extended dulness on percussion over the region of the heart.

During the course of the next month, while she remained under observation, frequent examinations of her chest were made. The harsh dry sound moved a little; being for three weeks most audible at the junction of the fourth left cartilage with the sternum, from which point it disappeared at the end of that period. The notes preserved always carefully distinguish it from a soft blowing murmur which during this period manifested itself in the aorta and pulmonary artery. She was discharged five weeks after this, having escaped with difficulty from a severe attack of intercurrent pleuropneumonia; and this sound remained then unexplained.

In three months more she returned, the symptoms of anæmia being now exchanged for those of valvular disease. She gradually sank. Her friends, fearing she would die, and her body be examined, removed her from the hospital. Just then pericarditis came on, and a loud double friction-sound was audible all over the region of the heart, save in that part only where, on the previous occasion, we had so long heard the dry systolic murmur.

Probably the two surfaces of the pericardium were thus far either adherent, or, if not adherent, prevented by thickening, consequent on the former attack of inflammation, from so readily taking on inflammatory action; but the matter still needs to be elucidated by dissection.* The

* Med.-Chir. Trans. xxiii. p. 29.

† Three of Dr. Blakiston's cases contribute to supply this deficiency: cases 52, 43, and 50.

* My friend Dr. Kirkes has been led to infer, from the rarity of adhesion of the pericardium in his dissections, contrasting with the frequency of the occurrence of the signs of pericarditis during life, that general adhesion is not the ordinary termination of general pericarditis. I cannot but think that some contingent circumstance has reduced the number of cases of adherent pericardium below the average during the time to which this inference alludes; for, during some years' observation in the same field, the number of adherent pericardiums bore as high a ratio to the number of cases of pericarditis as did any other organic lesion discovered after death to its symptoms noticed during life. But while advocating the opinion commonly received, that the ordinary termination of general pericarditis is by adhesion and obliteration of the cavity, we must admit the occasional occurrence of very striking exceptions to this rule. Dr. Kirkes mentions one such in his paper, MEDICAL GAZETTE, vol. xlv. p. 581.

question is one, if not of importance, at least of frequent occurrence; and it is of especial interest with regard to a subject previously under discussion—namely, the diagnosis of pulmonary murmurs. The real difficulty lies rather in their discrimination from such circumscribed pericardial than from aortic murmurs.

3. *Metallic cliquetis.*—Occasionally there may be heard over the region of the heart a very peculiar sound, to which the name of metallic cliquetis, or tinnitus, or costal percussion, has been applied. In the interpretation expressed by the use of the last appellation both Hope* and Bouillaud† agree, and Hope gives some details explanatory of the mode in which the sound appears to him to be produced. Laennec‡ explains the occurrence of this sound in the same way by costal percussion—"When the heart, beating in a sharp and rapid manner, though without any really forcible impulse, the apex only comes to strike the walls of the chest." Now, obviously, this explanation is somewhat shaken by the fact that this peculiar sound—which there can be no question about, for any one may produce it quite pure for himself in the way specified by each of these observers§—is not always audible at the apex in cases where it may be heard at the base of the heart. Dr. Hope correctly remarks that thin nervous persons are the most common subjects of it, but they are not exclusively so; and this fact again impairs the general applicability of the details of his explanation. The sound may be, and I believe is, trivial, and almost wholly unimportant; but, for all that, we need not accept a wrong or an unduly limited explanation.

I have met with it under four different conditions:—First, in nervous anæmic subjects, when the action of the heart was sharp; secondly, with more violent action of the heart, under circumstances which have led me erroneously to conclude, from the third condition with which I have less frequently found it connected, that it was indicative of the commencement of pericarditis. The fourth condition with which it has been found counected is a rough, almost scaly, state of the pericardium about the base of the heart; and perhaps a fifth might be subjoined, but for the fact that it s not always constant under these circumstances—namely, adhesion of the pericardium, with which we may class the occurrence of this sound over an aneurism. Dr. Latham tells me that he frequently meets with it in children, in whom he has noticed that it may be entirely suspended by a little pressure on the walls of the chest.

What should be the conclusion from these facts? The explanation by costal percussion is insufficient: it does not take in all the cases. The explanation by affection of the pericardium again is insufficient; certain as far as it goes:* yet there is positive proof, in the continuance of the sound after the two surfaces of the pericardium have become adherent, that the explanation does not go all the way. There are, then, three explanations of the mode of production of this sound—namely, costal percussion, friction of free surfaces, and movement of connecting areolar tissue. Each probably applies to a limited number of cases. There does not appear to be any one general explanation; nor, considering the nature of the sound, ought we perhaps to look for one.

4. *Purring tremor* is a sign which naturally attracts much attention. Really, it means little more than any valvular murmur heard under simular circumstances.† It is important, therefore, to dwell on all the facts which detract from its value as a certain evidence of irreparable organic disease.

Purring tremor may be produced either by the action of causes wholly external to the blood-vessels, or by abnormal conditions, organic or functional, of the blood-vessels themselves; or, lastly, by changes in the composition of the blood. Pericarditis occasionally furnishes an instance of

* Op. cit. pp. 41, 602.
† Œuv. cit. tome i. p. 218.
‡ Tome iii. p. 105.
§ By laying the palm of the hand over the ear, and lightly tapping the back of the hand with a finger.

* This interpretation is favoured by three of Bouillaud's cases (28, 30, 73), where metallic cliquetis was noticed in connection with traces of inflammation of the pericardium discovered after death.

† Opinions are at variance on this matter. Dr. Hope (Diseases of the Heart, p. 125) says that "he had never known tremor to exist in the heart independent of organic causes." Bouillaud (Mal. du Cœur, tome i. p. 182) takes the opposite view. Laennec (Aus. Méd. tome iii. pp. 123, 130) says that he has frequently noticed its occurrence without there being any organic affection of the heart, and quotes a case where the tremor was produced by a simple change of position. And Andral (Œuv. cit. note, pp. 123-5), while he cautions us about accepting Laennec's statement, and maintains an opposite opinion, quotes a case where this sign depended on at least a removable cause. Dr. Graves (Clin. Med. p. 926, 1st edit.) gives the particulars of a very striking case, where the tremor apeared to be independent of organic change. My own experience is to the effect that this sign is usually manifested in the most severe cases of cardiac disease, but that it does not infallibly indicate the existence of such disease. The three most striking cases of this kind that I can recollect presented in two cases dilatation of the right auricle, while in the third the auscultatory signs of cardiac lesion were removed, and I believe that no organic disease of the heart or vessels ever existed in this patient.

the first kind. With this, however, beyond the mention, we have here nothing to do; the altered conditions of the blood, and its containing vessels, alone falling within the scope of the present remarks.

It appears that purring tremor may be produced immediately in healthy animals by large depletion.* It might be referred, under such circumstances, to imperfect distension of the vessels. This tremor, however, thus produced, not only continues, but increases in intensity in the course of a short time: but we know, from Dr. G. O. Rees' observations,† that the quantity of blood abstracted by venesection is rapidly replaced by absorption of water. We must therefore consider extreme attenuation of the blood as at least as powerful a predisposing cause of this sign as an imperfect distension of the blood-vessels can be. Certainly it is a cause in more durable, and probably in more frequent operation.

Now, obviously, no fluid could communicate this sensation to our ears or hands when perfectly at rest. We can admit the attenuation of the blood only as a predisposing cause of purring tremor. We look for the exciting or proximate cause in the movements of the circulation. In the experiments just alluded to the tremor was produced in healthy animals. Applying this to practice, even if all clinical experience were opposed to the statement (which it is not), we must admit that purring tremor does not necessarily imply the existence of organic disease of the blood-vessels, but that the movements of, or induced by, healthy circulating organs are capable of producing it in a subject so predisposed. We must admit that blood attenuated to a certain degree is capable of being thrown into vibration—to use this more general expression—by causes insufficient to affect healthy blood after such a manner—namely, by the ordinary movements of the circulation: but it requires unnatural agitation to throw normally-constituted blood into such a state of vibration; and, more than this, it is required that the fluid pressure shall be less at the point where the vibration manifests itself than elsewhere.‡

Both these requisite conditions of the circulating fluid are found under the circumstances where purring tremor is usually produced. The contracted aortic opening, for instance, does not allow of the immediate equalisation of pressure of the blood in the ventricle and in the aorta; and the same physical impediment which causes this, causes also unnatural disturbance of the current of the circulation. So it is also in many cases of mitral, or more especially tricuspid regurgitation, where the blood runs under pressure from the ventricle into the dilated and nearly fully distended auricle. So, again, these conditions are present in an aneurismal varix; the opening from the artery into the vein supplying the means whereby the current of the circulation is disturbed, and the fluid pressure is rendered unequal.

On such principles the occurrence of purring tremor under certain circumstances may be explained. But why is it not always found under these circumstances? Why, for instance, does it not occur always with sigmoid regurgitation, or, indeed, during the filling of the ventricles of the healthy heart? A very sufficient answer lies in the fact that, under ordinary circumstances, the blood is not in a condition to be thrown into such a state of vibration by those movements. But, apart from this, I think that these apparent exceptions illustrate a very important point in the explanation of the mode of production of purring tremor, on which it will be of interest briefly to dwell.

In both these instances one condition of the containing vessels is wanting. Regarding the purring tremor simply as a vibration, whether of the blood, or vessels, or both, obviously some degree of tension of the parts concerned is requisite for its

* Hope on Diseases of the Heart, p. 100, 3d edition.

† Gulstonian Lectures, MEDICAL GAZETTE, 1845, p. 851.

‡ We are indebted to Dr. Corrigan (Dublin Journal, vols. x. and xiv.) for a very clear explanation of the mode of production of this curious phenomenon. Of the correctness of the principle advocated any one may easily satisfy himself, by repeating the very simple experiments by which Dr. Corrigan supports his theory. A tube of metal, or animal membrane, through which a stream of water is running, is to be constricted at one point, so that the egress of the fluid from the lower portion of the tube may be freer than its ingress. In the previously silent stream of fluid, now, below this point, a loud murmur with purring tremor will become manifest. Constrict the tube in a second place, at a lower point, and the murmur and the tremor will both cease over the interspace, as soon as the equilibrium of pressure shall have been restored on each side of the point first constricted.

In the same able essays Dr. Corrigan enters very particularly into the conditions requisite for the production of a murmur. But in all diffidence I must here venture to withhold such complete assent to his conclusions. The theory does not appear to me so beautifully and closely applicable to murmurs, as to purring tremor. At least the conviction of its truth is not so forcibly impressed by the examination of each case of endocardial murmur, as it is by that of each case of purring tremor. Probably, as Dr. Williams (Dis. Chest, p. 217) remarks, the flaccid tube only receives the sound or tremor from the point of stricture, as the tube of a trumpet does that from the lips. It is an instrument necessary for the manifestation of the grosser sign, the tremor, but not for that of the more delicate sign, the murmur.

manifestation. Nothing moving in one direction only can be, in so far, in a state of vibration; and the force being, in the case supposed, exerted in one direction only, a certain degree of tension or elasticity of the parts is required to ensure alternate motion and vibration. This condition is wanting in the cases under examination; for such regurgitant or direct stream, as the case may be, flows not into a cavity whose walls are at this degree of tension, but into a loose yielding bag, as it were; and, under ordinary circumstances, as soon as the walls of this cavity have acquired that degree of tension, the currents are interrupted or reversed by the regular movements of the heart.

An instance occurs now and then clearly showing that the rule which has been proved for simple tubes does apply to the complex arrangements and action of the heart; and enough is generally found on dissection to explain why the purring tremor should have been perceptible at some one given point in particular cases: but it is commonly very difficult to ascertain why it should have been perceptible only at one, and why particularly at that one point. Perhaps the above considerations may tend in some measure to remove that difficulty. Probably, too, the direction in which sounds connected with lesion of particular parts have a tendency to be propagated, is an important element in the explanation of each individual case; for there is no reason to doubt that purring tremor, in this respect, obeys the same laws as valvular murmurs.

There is one case seemingly so very apposite, that it may at first sight appear strange that the whole of this question has not been discussed on that ground—namely, the case of aneurisms. The evidence, however, from this source is almost entirely negative; for a purring tremor is far from being a constant, or, indeed, a common, sign of the existence of an aneurism: and the so-called aneurismal thrill, said to be perceptible in the pulse, has its origin, to my experience, where not in the state of the heart of the patient, in the imagination of the observer.

The structural conditions requisite for the manifestation of purring tremor are, indeed, seldom present in aneurisms. For the common form of aneurism—the false one, nosologically speaking—is not an elastic bag, to be emptied by pressure, but a hard mass, with thick unyielding walls. Nothing could be better calculated to stop the transmission of any vibration than these walls, with their irregularly laminated structure and unequal consistency. And their firm unyielding nature would render the blood in their cavity very unlikely to originate any vibrations. For, having no power of emptying itself, the cavity must always be nearly full: so the amount of blood injected at each contraction of the heart would be very small, and commonly under scarcely any greater degree of pressure than the blood already in the aneurism. So that aneurisms have, on both these accounts, as originating or transmitting vibrations, but little to do with the matter under discussion, generally speaking.

Generally speaking: but when there is a cavity with thin elastic walls, traversed by a stream of blood, and capable of considerable changes of dimension, possessing, in fact, those properties which a common aneurism has not, then this tremor is manifested in the greatest perfection. Thus we find it in aneurismal varices,—thus in the true nosological aneurisms; and thus it may be produced in the large superficial vessels of almost any person, but especially of the anæmic, by artificially applied pressure.

II. Functional Murmurs.

In what has been said above of the physical diagnosis of valvular disease, each murmur has been referred, as far as might be, to its specific local organic cause. But there has been frequent occasion to remark on the necessity of distinguishing each organic from its corresponding functional murmur. Much, indeed, of what might have properly found its place here, has already so been anticipated; and, but that the importance of the subject of functional murmurs claims a separate consideration, nearly all that remains to be said here might have been with equal propriety inserted in the preceding remarks on organic murmurs, so closely are the two subjects interwoven in practice.

To the practical rules by which the distinction between functional and organic murmurs is to be made, there is nothing here to add to what has been already said; for, lay down what rules we may, the diagnosis is at times very difficult, whether the physical or the constitutional signs, or both, be consulted. It cannot be facilitated by multiplying rules of uncertain application, but rather by making these rules more practically intelligible.

No very long experience is wanted to show that there is scarcely any one of the ordinary endocardial murmurs from which we are in the habit of inferring the existence of valvular disease, which may not co-exist with a healthy condition of the valves. Even the diastolic murmur at the base is not free from this fallacy in connection with the neighbouring veins. Were our knowledge of heart diseases during life,

then, limited to what auscultation tells us, it would be very uncertain.

But pathology, guided by a little observation, enables us to group the exceptional cases, where we are exposed to this fallacy, in one class, as the anæmic or functional. This position is supported by the direct results of numerous observations on the human subject,* showing that those whose blood is attenuated are particularly liable to have endocardial murmurs; just as it has been shown experimentally of fluids in tubes, that a murmur may be induced in a stream or suspended, simply by diluting the moving fluid or adding to its density, as the case requires.† We recognise, therefore, in the anæmic—to use this as a general expression—a condition of great susceptibility to the production of murmurs. They constitute a class in whom the ordinary movements of the circulation are sufficient to produce that, for whose manifestation, in most persons, a great disturbance of those ordinary movements is requisite. This, in fact, is precisely the same as has just been shown with regard to some of the subjects of purring tremor.

This principle of a predisposition to the production of a murmur must never be lost sight of in any inquiry into the nature of a murmur. It explains how many causes, insufficient thereto in a healthy person, may produce a murmur in a person so predisposed; and so far it detracts from the absolute value of the observation of the existence of a murmur at any particular point. It has been already shown that the point of greatest intensity of murmurs is very important to be ascertained, as determining the seat of the cause in which they originate. The limitation of a murmur, however, to a particular part does not show that it has its origin in an organic lesion of that part; for, in a person so predisposed, the natural conformation of the vessels at this point, without the existence of any abnormal obstruction, may so disturb the circulating fluid as to give rise to a murmur audible there, and there only. Instances of this are familiar, though the exact explanation of each in particular is not always obvious.

For instance, a venous murmur may be heard loud in one jugular,‡ and not at all in the other, or of different tones or intensities on the two sides; or it may be heard in the innominatæ, and nowhere else, or in the ascending cava, and nowhere else. Or a blowing murmur in the jugulars may become a cooing or a humming sound in the innominatæ veins; or a murmur in the pulmonary artery may be transmitted as a blowing sound through one lung, and as a musical note through the other.

In many such cases the circumstances at once render it clear that the local murmur is independent of any deviation from the natural conformation of the parts concerned. We may have such assurance from the murmur being referable to parts which we know to be almost invariably free from disease; and from other circumstances. But in some cases we must be content to remain in doubt while we watch for something to clear up the difficulty; for the existence of a general anæmic condition obviously does not negative the possibility of the existence of a local organic cause: it only increases the effect. Now suppose, in such a case, uncertain to the last, on dissection we ascertain the existence of organic disease, obstructing or disturbing the current of blood about the point where the murmur was most audible, we are content to look upon the organic disease as the physical cause of the murmur. Or suppose, on the other hand, that we find the same part—say the aortic valves—healthy, we are content to believe that the murmur—say a systolic murmur—was functional. Is it in the contentedness of ignorance that we accept these two opposite results as equally satisfying the conditions of the problem which we failed to solve during life? Certainly not: that a murmur was audible, is one fact; that there is or is not organic disease, is another. The question is not, are these seemingly contradictory results possible? for they plainly are so; but on what theory are the admitted facts explicable and consistent? The explanation need not occupy us long.

When we speak of a murmur as referable to an organic lesion, we must recollect that the same effect may be produced under three conditions of the organs of the circulation—namely, obstruction may be offered, or regurgitation may be allowed, or there may be a seemingly healthy condition of the parts in question. And the explanation which applies only to one case,—obstruction,—although the most common, is insufficient, and contradictory to the explanation of the rest. We owe Dr. Corrigan much for carrying our ideas beyond what is expressed by the term obstruction, to the effects which that obstruction may cause.

As to organic murmurs: of the applicability of Dr. Corrigan's theory to practice in many cases there can be no doubt; and,

* Andral, Hematologie, p. 53; Bouillaud, Œuv. cit. tome i. p. 256.

† Archives Générales de Méd., Août 1838.

‡ Laennec (Aus. Méd. tome iii. p. 80) says that he has heard a murmur loudest most commonly on the right side of the neck. Bouillaud (Des Mal. du Cœur, tome i. p. 239) has heard it more often on the left. My own observations make me think that it is heard loudest, *cæteris paribus*, on the side farthest from the observer,—possibly from the fact of the integuments being generally made more tense on that side during auscultation.

strictly speaking, it must be applicable to nearly all cases of organic valvular disease; for, necessarily, when a fluid is moving under intermittent pressure in a certain direction, at the moment of the application of the moving power the pressure is less on the further than on the nearer side of any obstruction to the stream. But my own experience would not enable me to say that the degree of intensity of the murmur bears any very close relation to the amount of the mechanical effects of the organic lesion in this respect. I should feel, therefore, as a general rule, more disposed to refer the murmurs arising from organic disease simply to disturbance of the circulation than to admit (though not rejecting) Dr. Corrigan's more exact explanation.

So, too, with regard to functional murmurs. Abstractedly, Dr. Corrigan's theory holds good; for as the blood advances in the arteries, it naturally, yielding in the direction of least pressure, at each moment is less closely embraced by the blood-vessels. Here, then, is a cause sufficient, in a person so predisposed, to produce a murmur: but here, too, though the abstract correctness of the principle is unquestionable, I would rest content with the less definite expression of disturbance of the circulating fluid; for the effects are not manifested with any very close relation to the time and place where the presumed cause should have most influence.

Traced, then, into their elementary processes, functional and organic murmurs may each be explained clearly and consistently. The effect—namely, the murmur—is the same in both, and the proximate cause is the same.

With the more remote causes auscultation, as such, has nothing to do; for auscultation tells us only the ultimate acoustic effects: it has nothing to say of obstruction, or of the condition of the blood, or of the state of the valves. Between a functional and an organic murmur there is to auscultation, abstractedly speaking, no essential difference. They both equally indicate disturbance of the circulation.

But they indicate no more. All further information is to be obtained from pathology. It is pathology which, by laying the information from other sources to that which auscultation gives, tells us that in one case the blood is in a condition very susceptible of the influence of any ordinary disturbing cause, and that in the other there is some extraordinary disturbing cause in action. And now we know that the murmur in one case indicates disease of the blood; in another, diseased action or structure of the blood-vessels.

Yet one word more, which I should take shame to myself for saying, did I not feel that what we all now know so many of you have laboured hard to establish, and what we smile at now, you had once difficulty to controvert.

We err more often as pathologists than as auscultators. To detect the time and place of a murmur, and to measure the force of the heart's action, is not so very difficult; but it is often very hard to interpret correctly what all these signs mean. It is not very hard to see which way the compass points, but it requires a philosopher to explain its variations. Yet the compass is not an idle toy, nor auscultation a vain refinement, because their indications are not infallible.

When Andral published his classical survey of the condition of practical medicine at the time, our knowledge of diseases of the heart was, as we have seen, very different from what it is now. I have pointed, in the preceding pages, to several of the advancements which have been made in this department of practical medicine. But I cannot forbear adverting to some other particulars in which the subject of diseases of the heart now stands in quite a different place from that which it held when Andral wrote. There were then few auscultators, and many opponents;* but this has been all reversed. And if the weaker vessels then sheltered themselves under the name and example of those whose position gave weight to their rejection of the new-fangled doctrines, and to their incredulity, this, too, has been reversed.

It is not that the intimations of the ear are of more universal application in medicine than those of the other senses, but that they only just now take the place with the other senses which they should have held long ago; and their admitted importance results from the extent to which they are found to be applicable.

It is not that the intimations of the ear are more trustworthy or infallible: indeed, they are often very fallible and ambiguous. But those who use it most should know its use the best,—what it cannot explain, as well as what it can. And with regard to the profession of disbelief in auscultation,—thanks to those who have so clearly laid down its principles for us,—I think most would now be content to meet it in the same words which have been so well applied to the rejection of evidence on another subject: "There is cause to think that scepticism itself is often no more than a form of very unreasonable enthusiasm, demanding conviction without the pains of inquiry."†

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* Hope on Diseases of the Heart, Dedication, 3d edit.

† Davison on Prophecy, p. 100, 4th edit.

LECTURE II.

III. Morbid Anatomy of Valvular Disease of the Heart.

Physicians and morbid anatomists not conversant with the same classes of cases. Morbid changes are of two forms: causes of each form: general characters how far distinctive of each: microscopic distinctions negative; pathological more important.

First form.—Effects of simple or rheumatic inflammation.—Are they really inflammatory? — Warty growths, their seat, early history, minute anatomy.—Ulterior changes, to reparation, to disorganisation. —Allied changes.—Primary and secondary effects on the valves compared.

Second form. — Specific inflammatory or other affections; typified in the filamentous and globular growths; early history. — Filaments; successive changes; minute anatomy; compared with growths of the first form; size; softening, earthy degeneration.—Globular vegetations; history; compared with the filaments.—Allied changes.—Seat of the changes in the two forms contrasted.

In the preceding lecture we reviewed the chief causes of valvular disease, and the physical signs from which we conclude that those causes have taken effect. We turn next to the consideration of the nature of the lesions giving rise to those physical signs; that is to say, to the morbid anatomy of valvular disease.

It now becomes necessary to bear in mind that the classes of cases of valvular disease with which we have to deal as physicians and morbid anatomists respectively, are not the same. Even supposing the number of cases which we met with in these two separate capacities were the same, (making the proper allowances), still the two classes could in nowise be considered as identical.

As physicians, we have to do with cases of acute inflammation where the first foundation of those structural lesions is being laid; and with cases where structural lesions, arising from whatever cause, and dating from whatever period, are producing present disturbance of the circulation. We miss, as physicians, the end of those cases which get entirely well after acute inflammation, and the beginning of those cases where structural lesions have arisen from chronic insidious processes.

As morbid anatomists, of course, we meet with structural changes of all kinds and all degrees, and as such we meet with the beginnings of chronic processes, of which, as physicians, we only know the ends. But, as such, on the other hand, we see nothing, or rather know nothing when we do see them, of those cases where perfect reparation has taken place; cases of which we have seen the beginning as physicians, of which (may we say?) we have, as such, effected the cure.

It is needless, on the present occasion, to enumerate all the characters of a healthy valve. They must be often seen to be apprehended. The standard of health for each particular subject must be ascertained, as nearly as may be, by a comparison of the left valves with those on the right side, which so rarely suffer from the same kind of disease. It will be sufficient merely to indicate the points to be kept in view.

The opaque nodules so commonly found in the auriculo-ventricular, especially in the tricuspid valves, must be practically known to be distinguished from the morbid growths, which they in some sort resemble, found especially in the mitral valve. Speaking generally, the thinness, elasticity, and transparency, with the fan-like expansion of their chordæ tendineæ, just stopping short of their delicate edges, are the characters most important to notice in the auriculo-ventricular valves. In the sigmoid valves, the double crescentic line commencing from the corpus Arantii of each, and marking their edges of contact,—their thin free edges, — their exact symmetry, and the accuracy with which they can fall back against the sides of the artery during the onward passage of the blood, should be familiarly known. For it is in these particulars that disease is apt to induce the most important structural changes.

Abstractly speaking, the principalchanges are not many. The valve may be simply thickened and more or less rigid, or its substance may be increased by external accretion, or removed by erosion. And with these changes of form and texture more or less important changes in composition may be allied.

A more particular survey of the various morbid changes, waiving the minor differences which the variety of situation and extent of these changes and their mutual

connections give rise to, enables us to group them into two classes. There seem to be sufficient grounds for subdividing diseases of the valves of the heart into two distinct forms, each characterized by external differences of figure of the morbid products, each owning different causes, allied to different changes of other parts, and passing through different phases from their first beginning to the fatal termination*.

The external figure of the accretions which characterise one of these forms is that of small warty granules studding the edges of contact of the valves. To the other belong those long leathery tails or filaments, and the round pedicled masses, known as Laennec's globular vegetations, which sometimes hang from the edges of the valves†. The one we recognise most commonly as the effect of acute rheumatism, judging from the circumstances under which it occurs; the other seems more closely connected with congenital malformation or predisposition, or with renal disease. We may group with the one the membranous flakes which sometimes overlie the interior of the left ventricle where it runs up into the aorta, and most of the changes of figure of the orifices of the heart or of the valves independent of present accretion or erosion. The other claims to itself those patches of papillary growths which sometimes stud the interior of the left auricle; and here nearly all the changes of form by ulceration‡ or erosion belong. In one the cause acts but for a while, and the subsequent changes which the organic results of the temporary action of this cause undergo are due to contraction, or consolidation, or obsolescence of the morbid products. But, in the other, the cause which has induced the growths acts permanently, and the growth increases at one part while it decays at another; softening and earthy degeneration may be found at the root, or in the centre of the yet growing mass. Such, in general terms, are the distinctions which it is proposed to display more in detail in the following remarks*.

We have already inquired into some of those conditions whose relation to disease of the valves of the heart, speaking generally, appeared to be that of cause to effect. It remains here briefly to show how far we may ascribe to any of these conditions a specific power of determining the existence of one or other of the two forms of valvular disease. Rheumatism, renal disease, and congenital malformation, seem more particularly to have such specific influence, and to require a separate consideration in this point of view.

1. *Rheumatism.*—Of the ordinary form of acute endocarditis, scarcely any other cause than articular rheumatism is familiarly known. Indeed, seeing with how slight an attack of articular rheumatism endocarditis may coexist, and how often the articular affection may be discovered by patient watching, in cases where we had believed it to be absent, one feels more inclined to doubt one's own accuracy than to record a case as an instance of non-rheumatic endocarditis. Though there is no question that such cases are occasionally observed, especially, (though I have no experience on this point) after the exanthemata, and of these after scarlatina. And what is true of the results of recent endocarditis applies more or less strictly to older morbid products, as they are more or less certainly referable to such a cause. Rheumatism would appear to be a most active cause of the first form of valvular disease.

* Dr. Williams has classed valvular lesions under three general heads, referring them:—1, to the thickening with or without induration; 2, to thickening with softening and ulceration; 3, to atrophy. The valvular lesions which have come under my own notice have appeared more readily to fall into two classes. But in venturing to differ with Dr. Williams as to the claims of his third class to a distinct consideration, I must acknowledge the correctness with which he has, in so few words, delineated the chief characters of the form of disease which he has described there, as here, in the second class. (Path. and Diag. of Dis. of the Chest, 4th edit. p. 260.)

Dr. Kingston has considered atrophy of the valves at some length. (Med.-Chir. Trans. Vol. xx. p. 90.) The importance of the principle inculcated I would not question. But I have not yet met with a case where the consequences of simple atrophy have been a cause of valvular imperfection, the only cases falling strictly under that denomination having been to my experience sigmoid valves with reticular edgings (if, indeed, this be not congenital) the apertures being beyond the edges of contact, and therefore unimportant, and valves whose diminishing proportions have apparently kept pace with the general atrophy of the heart. Contraction of the chordæ tendineæ, or of the valves, or perforation with traces of some organic process, should rather be referred to such previous processes than to atrophy. And they have accordingly been distributed under these several heads in the ensuing pages.

† Their more common situation is at the tips of the auricles or ventricles, where only were they known to Laennec. (Aus. Méd. iii. p. 345.)

‡ The ulcerous patches of which the upper surface of the mitral valve is the common seat admit of no very certain explanation. Considering them as evidences of something which has been (for my experience of them extends no farther) and has ceased to exist, leaving only irregular serpiginous markings on the valves, the investigation of a small number of cases would scarcely justify any inferences as to their essential nature.

* With regard to specific, and particularly malignant diseases of the valves of the heart, more facts are requisite before any attempt can be usefully made to arrange what is already known on the subject. The subject of tubercle has already been noticed in its negative bearings. (Walshe on Cancer, ad locum Med.-Chir. Trans. vol. xxx. pp. 1 and 39.)

In the other form it holds a lower place. Out of seventeen extreme cases, selected as the most unequivocal examples of this form of lesion, rheumatism appears as a cause in no more than seven. And this is the more striking, when, taking the converse view of the remark just made, we recollect how easily any previous attack of pain may be interpreted as rheumatism.

2. Of *renal disease* there is little here to observe more than has already been said in the previous lecture. Only it may be added to those remarks, in their present application, that renal disease existed in ten of the seventeen cases selected as the most characteristic examples of the second form of valvular disease.

3. *Congenital malformation*, as displayed most unequivocally in the development of only two sigmoid* valves, aortic, or pulmonary, or both, as the case might be, has been observed in the dissection of twelve patients, affecting fourteen out of their twenty-four sets of sigmoid valves. Six of these patients and six sets of valves were affected with what has been above designated as the second form of valvular disease, in an extreme degree, presenting long leathery growths hanging down into the vessels from the edges of the valves. Five other of these patients had valvular disease, not indeed of a nature so decided, or in so marked a degree as the six just quoted, or as to be considered with them as types of the affection, yet quite of a different nature to the results of ordinary rheumatic endocarditis†. Only one patient out of twelve, and three sets of valves out of fourteen, thus ill-developed, namely, one aortic and two pulmonary (this in the two double cases) had escaped disease.‡ We may contrast with this the curious fact that sets of four sigmoid valves, where there is no imperfect but rather redundant development, are generally found healthy. In my small experience (for this is a much less common appearance) they have been invariably so*.

Within certain limits, the distinctions between the effects of these several causes may be clearly traced. But these limits are not very wide. Disease of the valves of the heart, indeed, would be very unlike disease of any other part, if the different forms did not insensibly shade into each other; if simple inflammation, for instance, did not sometimes arise from the irritation of specific disease, or if this last did not sometimes extend itself in consequence of, and mingle with the products of, simple inflammation. So that the line cannot always be drawn where we may say this results from the one, and here end the products of the other form of disease. And there are two cases in particular where the products of the two forms are inextricably involved.

1. Extreme cases of simple rheumatic endocarditis may, there is no reason to doubt, shade into the second form. The warty granules may, when of rapid growth, acquire the form and properties of the long leathery accretions.†

2. Simple induration and thickening of contiguous parts may ensue upon the existence of either form; and the results of chronic inflammation and of the original disease, of whatever kind, may be so intermingled as to exceed all the powers of discrimination of the anatomist to disentangle them. It could not well be otherwise.

So far, however, as the delineation of the characters of the two forms is concerned, this need cause no difficulty. The deficiencies and indefinite results of one observation may be readily made good from others; and the succession of the several lesions, as traced in a large number of dissections, is complete and continuous. So that there is no reason to doubt that the two forms of disease which are denoted respectively by simple thickening of the edges of contact of the valves, and by minute filaments growing from their surface, are as distinct in these their lesser manifestations as in their more highly characteristic development, namely, in the warty granules and the long leathery growths which they each display at another period of their existence. But this by the way, only observing, as will appear further on, that the morbid appearances here placed in contrast do not indicate exactly the same period in the

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* Dr. R. Quain (Edin. Monthly Journ. Dec. 1846) has published some cases where the sigmoid valves had been apparently reduced to the number of two by the rupture of one of their attachments. I do not think, however, that this explanation applies to any of the cases referred to above.

† Since writing the above another case has come under my notice, at too late a period, however, to allow me to add it to the tabular analysis. The three aortic valves were affected with recent endocarditis, the two pulmonary with old thickening and earthy degeneration. The man had renal disease. The muscles of his neck were infested with *trichina spiralis*.

‡ I owe my first knowledge of this fact (the liability of valves thus constituted to disease) as of many others, to Mr. Paget, who has remarked upon it in the Med.-Chir. Trans. vol. xxvii. p.187. Dr. Barclay says, "I am not aware that, during the two years from which the cases are selected, any instances of this abnormal conformation (the existence of only two aortic valves) occurred unconnected with endocardial disease." (Med.-Chir. Trans. vol. xxxi. p. 200.)

* Dr. T. Thompson mentions the occurrence of four healthy pulmonary valves in a case of congenital malformation of the heart. He refers to other cases where the valves were found to differ in size, but are not said to have been diseased. (Med.-Chir. Trans. vol. xxv. pp. 249—251.)

† See Rokitansky, Path. Anat., Bd. ii. S. 478.

existence of each of the two forms respectively.

The general tendency of microscopic observations of the intimate structure of the growths by which the different forms of valvular disease are characterized, is rather to reduce them all to one type than to establish any essential differences. For many of the anatomical distinctions which appear so plain to the naked eye fade away under the more rigid, yet more partial scrutiny of the microscope.

Microscopic examination has not appeared to me to justify any exact essential distinction of the two forms, even in their most extreme and most characteristic development. The differences it indicates are but differences in degree. The structure of the morbid products in both forms alike is that of an imperfectly fibrous, more or less distinctly laminated mass. Through this mass are scattered numerous small granular bodies and granules, with a variable quantity of oil globules; the whole being cleared, but not entirely removed from sight, by acetic acid. I do not think that anything could be predicated more particularly of the intimate nature of the growths characterizing either of these forms; and I believe that this description is equally applicable to them both.

At first sight it might appear that this negative evidence of the microscope on so important a point is sufficient to set aside all other distinctions. Not so. For the microscope also tells us generally that the fibrinous traces of old inflammation, after a certain period, cease to present any characters distinctive of the known circumstances under which the inflammation took place. We have no particular reason, therefore, to expect to find any such distinctive characters in the particular case under consideration. The distinction, however, between the two forms rests on a safer foundation, in their observed pathological tendencies, than in these ambiguous differences of their microscopic structure.

Pathologically these two forms of valvular disease are distinguished, as already noticed, by the characters which the principle predominating in their development has impressed upon them. In the one form there is an original tendency to grow rapidly,* and still, while growing, to degenerate and decay. In the other there is a definite period of growth, on the determination of which the morbid products contract and consolidate themselves. In the one the morbid product inclines to be (for it is only a question of degree) soft, brittle, and granular; in the other it is rather firm, elastic, and of a fibrous structure. Such is the most general expression of the results of ordinary observation in this matter.

In speaking of an original disposition, and ascribing to morbid products living powers in so large a measure independent of the surrounding parts, I am presuming your acceptance of the doctrines which these terms involve. I need scarcely occupy your time with any detailed expositions of these doctrines. They have been made familiar to you by Mr. Paget.* And in their application to the matter under consideration I gladly acknowledge the extent of our obligations to Dr. C. J. B. Williams† for his labours in this department of general, as before in that of cardiac pathology. With these in your hands details from me would be superfluous.‡

It is necessary here only briefly to recapitulate, before applying them, the two general principles relating to the present subject, on which our attention must be fixed. These are:—First, that there are from the first essential differences in the products of the same disease according to the constitution of the patient. And, secondly, that such constitutional peculiarities imprint on the morbid products certain tendencies, which take effect after the morbid products have entered upon a condition of comparatively independent existence.

Valvular Lesions resulting from Simple or Rheumatic Inflammation.

The process which gives rise to the ordinary form of valvular disease has received the name of endocarditis. It is known better in connection with acute rheumatism, than as of apparently spontaneous origin. Yet, as the identity of the disease under both circumstances has been established, in the following remarks, as already by anticipation, the valvular lesions with which we are familiar as the effects of acute rheumatism will be considered as resulting from simple or rheumatic inflammation indifferently.

It has been urged that the changes which acute rheumatism induces in the valves are not due to inflammation at all, but merely

* See on this point "Dalrymple on the Rapid Organization of Lymph in Cachexia," Med.-Chir. Trans., vol. xxiii. p. 205. Dr. Bennett, "On Cancerous and Cancroid Growths," p. 164, says "[fibrin] is more connected with the decaying than with the formative stage of life."

* Lectures, MED. GAZ., 1847-49-50. Also "Simon's Lectures on General Pathology," Lect. VI., Lancet; and Dr. A. Clarke, MED. GAZ., xlii., p. 416.

† "Principles of Medicine," 2d edit. chaps. 3 and 4.

‡ See also a very practical exposition of the surgical application of these principles, "Humphry's Lectures," Prov. Med. and Surg. Journ. Lecture V.

to deposition of fibrin from the blood. Now, this is not a matter to be definitely settled in the present state of our knowledge; for there is no certain anatomical character by which we may distinguish the effects of inflammation from those of deposition in these morbid products. Neither do I think that on this point the results of different modes of treatment can be trusted as diagnostic of the elementary constitution of the organic mass. Each one must form his own conclusion from the different weight he attaches to certain general principles, from which alone have we any means of judging of the question at issue.

It may safely be admitted that, after a certain period, namely, when the morbid product has acquired a certain size, deposition does take place, and that the total results are most probably, in any case, of mixed origin. For experiments shew the tendency of any substance projecting into the arterial current to become the nucleus of fibrinous accretion.

But, obviously, such contingent deposition has nothing to do with the first beginning of the valvular lesion. With regard to this, the real question at issue, we must not overlook the pathological condition of other parts with which this change of the valves often coincides, namely, inflammation in its most aggravated form in the pleuræ, lungs, or pericardium. And the fact, to which we shall return hereafter, that the subsequent valvular changes are essentially not superficial, nor limited to the point of the original lesion, makes very much against any theory which would explain these changes as the result of simple deposition in the first instance.

The absence of *vasa vasorum* from the lining membrane of an artery is the strongest reason that has been urged why these growths cannot possibly result from inflammation in the ordinary sense of the term. But this objection is more specious than real. For inflammation may be carried on by the same organic means as nutrition. And if, in any part, nutrition by unusual means effect an ordinary end, surely inflammation in the same part may do so likewise.

This is a point on which the most trustworthy information might be expected from accurate observations of the other changes of the valves which coincide with or precede the earliest appearance of these equivocal growths. But from the fact that all the attempts to settle this much disputed question habitually refer rather to abstract principles and general inferences than to direct observation, we may conclude that opportunities of noticing the very earliest changes resulting from so called endocarditis are exceedingly rare in this country. For my own part I have almost entirely to rely on the observation of others* for the accuracy of the statement that redness, with subsequent swelling, and apparently softening and loosening of the tissues of the valves, precede the appearance of the warty excrescences at their edges; for the disease had advanced to this last stage in all the unequivocal cases which have been the subjects of my own observation.

Staining and softening must be received in any case with great caution as evidences of the presence of inflammation of the lining of the heart and its valves. But, while on our guard lest we should misinterpret the results of decomposition in particular cases, we must not allow excess of caution to carry us into an opposite error. We must not at once dismiss these appearances as useless, though we cease to regard them as infallible guides. We must allow appearances which elsewhere would be considered as characteristic of inflammation,† to bear that signification here also,—at least in the absence of facts justifying any other interpretation; or there would be an end to the question, which, though myself inclined to believe that the growths, under these circumstances, are the effects of inflammation, I hold to need further examination.

The first of the changes more familiarly known as the result of endocarditis is recognised as the accretion of little warty granules running like a row of beads along the edges of contact of the valves, most commonly the mitral and aortic. Their appearance is very striking, for the edges (which it is important to distinguish from the edges which come in contact, on closure, with the other flaps of the valves) retain, at this early period, their normal configuration, and the disease is limited to this little bank, which seems piled up, as it were, on a healthy valve. The beading is often uniform and continuous, running, at the distance of about a line from the edge of the valve, round a considerable part of the orifice affected; or, if the beading be not continuous and uniform, generally all the angular projections are tipped, or the re-entering angles filled with a little wart of this nature.

Why the edges of contact rather than the free edges,‡ and why especially the angular projections should be thus affected, is readily explained by the greater degree

* Rokitansky, Path Anat. Bd. ii. S. 428. Dr. Kirkes has recently supplied me with the notes of a case where in connection with rheumatism the valves were distinctly swollen and vascular. In this case there were no fibrinous growths.

† See Gendrin. Hist. des Infl. § 931, and § 1447.

‡ Watson's Lectures, Vol. ii. p. 67, 1st edit.

of attrition, tension, and pressure, to which these parts are exposed during the action of the valves. For whatever be the cause of the formation of these granules, whether they arise by deposition from the blood or exudation from the inflamed valves, it is easy to see how such a process might be determined at this particular part by any cause calculated, like the above, to lacerate, or roughen, or even press the endocardium there.

These double crescentic lines on the sigmoid valves, and the irregular beading with its angular projections along the auriculo-ventricular edges of contact, furnish commonly the earliest and certainly the most unequivocal evidence of the presence of what is generally known and is considered in the following remarks as endocarditis.

There is nothing else with which they can be confused. At the same time, however, their size must not be taken as an exact measure of the intensity of the local inflammatory action; for deposition from the blood is a process which may and very often does take place; and as any roughness on the surface of a valve would be likely to attract fibrin from the passing blood, even in a healthy individual, such an event might yet more readily ensue in a case where the blood has already a strong tendency to deposit fibrin. Such is the case in acute rheumatism, as appears not only from the analysis of the blood, but from the large inflammatory exudation into the pericardium or pleura which occurs coincidently in some such cases. Here, as in so many other cases, where two causes act conjointly, the energy of the one may in greater or less part supersede the operation of the other in the production of their joint effects. The exact amount of the results of each process we have no means of estimating.

However produced, there can be no doubt that these granules are, from the first, a serious lesion. The singular tendency which they display to grow most freely on all the angles of the valves, explains how, from their earliest formation, they obstruct the onward flow of blood through the aortic aperture, and allow of regurgitation through the mitral valve, which they wedge open. The amount of blood hindered, and the amount regurgitated at each beat of the heart, may not, it is true, be much, but a very small fractional part, when multiplied by more than seconds, and minutes, and hours, amounts to a good deal in a few years: and when, after the vicissitudes of a laborious life, the patients who have left the hospital after an attack of acute rheumatism, with an endocardial murmur, apparently depending on the existence of granules of this nature, come again under observation, the serious importance of the lesion, which was then only indicated by a faint abnormal sound, is now too obvious in the state of the heart and the symptoms generally. All thought of effecting a cure is now at an end; the utmost that we can hope to effect by treatment is to ward off present death or palliate suffering by dividing the burden among the different organs. Many of these patients sink at once, others after two or three more of such melancholy visits; and it is with no little interest that we look to dissection to show what has been going on intermediately.

The first fact which presents itself is a negative one: the warty growths which characterise endocarditis are, after an uncertain period, no longer to be seen.* What the exact term of their existence may be it would be hard to say, and, indeed, dissection favours the belief that they last very different periods in different subjects; but the fact is certain, that, with few exceptions, this peculiar morbid appearance is not met with in patients dying years, or even months, after the particular attack of acute rheumatism to which the cardiac symptoms appear referable. A fresh attack of rheumatism may have induced the growth of a fresh crop of these granules: but this only proves the rule; for this second crop is found planted on the solid basis which the changes in the first crop have prepared for its reception.

The comparatively rare and exceptional cases, where the granules remain as such, need not detain us long. Under these circumstances the granules shrink up and become hard, obtaining, like all other masses of indurated contracted fibrin, the name, however incorrectly applied, of cartilaginous.†

More commonly, as already stated, this granular appearance is superseded by other changes. Of these, the first seem to have no regard to the ultimate end of the process: that is to say, whether the whole process shall end in perfect functional and organic reparation of the valvular lesion, or whether it have the contrary tendency, the first stage in either case is alike,—the granules are effaced. In the place where there is reason to suppose that the beaded line ran along the edges of contact of the valves, we now find a round bank of fibrin. This little bank assimilates itself to the disposition of the original beading by its

* See Rokitansky, Path. Anat., Bd. ii. S. 436; and Watson's Lectures, ii. p. 286.

† Bouillaud, Rhumatisme Articulaire, p. 181, mentions one such case. I have seen also very rarely these granules having undergone earthy degeneration without quite losing their original form.

greater distinctness at all the angular projections of the valves.

This morbid appearance may be taken as displaying the completion of the second series of changes in this form of valvular disease, as the formation of the granules constituted the first. We are not led to speculate abstractly on the connection of these two stages, for dissection supplies numerous cases to illustrate the gradual transition from the detached warty granules to the smooth continuous wall of fibrin. The original fringe of little beads, closely set, touching each other at the sides, gradually assumes the form of a continuous wall or crest, the serratures in which correspond to the divisions of the original granules; and this as gradually takes the form of the round bank above described, the serratures becoming obliterated, and the surface polished, though still remaining uneven.

The recent soft growths may easily be stripped off from the heart, leaving the part of the endocardium on which they have rested perfectly sound. And in the same way these granules may be easily separated from their connections with the subjacent tissues, in cases where they have been implanted on the edges of valves thickened by old disease. Zehetmayer, who has paid some attention to this matter, says that the epithelium is not continued beneath even the recent granules*. I have not been able to satisfy myself of the fact.

But when the granules have existed for a longer time, they become agglutinated more firmly to the valve, as well as to each other. In one case, as the laminated structure of the fibrinous crest might have led one to anticipate, a layer could be torn off, consisting of the crest and the endocardium together, leaving the valve, where the experiment had best succeeded, to all appearance perfect, only wanting the endocardium; but the growths were not separable from the valve without at least this amount of injury to its structure. In another case, speaking rather of single, the most satisfactory experiments, than generally by way of deduction from them all, the growths and endocardium and all the tissues of the valves were firmly matted together. The endocardium, when raised in strips from the neighbouring healthy surface, tore away with it a little of the covering of the thickened edge or crest; and, in the same way, on raising a strip from the superficial part of the crest, the tear ran into the endocardium of the neighbouring surface. But the separation could not be carried far; the result of any attempt to separate the whole mass was the destruction of the edge of the valve. The diseased mass, here, as in the former case, was of a fibrous texture.

In one case, the left ventricle, where it leads up into the aorta, was overlaid with a fibrinous patch slightly corrugated in parts. This was composed of long parallel wavy fibres wanting the distinct outline of the fibres of the endocardium, looking indeed quite woolly, apart from all other differences, as compared with these. On raising this, the endocardium was found beneath it smooth and polished, only a little thickened. The separation of this flake from the endocardium was made with increasing difficulty on approaching the sigmoid valves, and from the surface of these, which it overlaid, it was found quite inseparable, the substance of the valve tearing down, and the subjacent layers coming away with the firmly adherent adventitious membrane.

The results of these and other examinations of the minute anatomy of these growths in their several stages, greatly favour the opinion that they have their origin in inflammatory exudation. It is easy to see how, under any circumstances, whether deposited or exuded, the new product might become firmly agglutinated to the endocardium. But without some antecedent process affecting the deeper seated tissues of the valve, such as inflammation, it is not so easy to see why the whole substance of the valve should be implicated, and valve and growth be matted together into one mass; as is commonly found to be the case, when the external form has undergone those changes which mark the completion of the second period.

Thus far the processes have appeared to be identical, whatever the end in view might be, whether perfect repair of the lesion or disorganization of the heart. It will be most convenient to take this stage, characterised by the coalition of the granules, as the point of divergence from which repair or disorganization ensues. Perhaps a more natural distinction might be drawn from an earlier period, according as absorption or other processes predominate in the secondary changes. But, at least, this arbitrary period seems to be that when one or other of these processes begins legibly to stamp its own characters on the valvular lesion*.

* Herz-krankheiten, S. 327.

* It is not intended to assert that from a certain period the changes in any one case are all for repair, or altogether tend to disorganization. Obviously, if analogy has any weight, the disorganizing changes might ensue at any period short of repair in any one case, and reparation might commence in the eleventh hour in any other. It may seem almost superfluous to advert to this, or to take any precaution against such an inference being deduced from the statements in the text.

I. Tracing, first, the process of repair:— The form of the fibrinous ridge changes; on section it appears square and angular rather than round. Next, all traces of the original mode of formation are lost; the fibrinous ridge has become smooth like the rest of the endocardium, with which it is inseparably united. It is now, to all appearance, an integral part of the valve, and, in its altered form, possibly itself the seat of new granulations arising from a recurrence of the disease in which itself originated.

The period occupied by these changes is apparently of different duration in different cases. Such a near approach to complete reparation was found in the mitral valve of a woman dying of syncope three months after the beginning of an attack of acute rheumatism, with endo- and pericarditis which had never been completely subdued. In her, as in another dying of syncope in a third attack, twenty and thirty years respectively after the two former attacks of rheumatism, the polished ridge had become the seat of new granulations. Another patient dying of bronchitis seven and ten years after two attacks of rheumatism presented the same ridges without any recent deposit. But reparation was quite as far advanced in a case dying ten months after the attack of rheumatism, as in those where life had been protracted to a longer period. Perhaps the original lesion had not been equally severe in all these cases.

I feel too sincere a desire to reach the truth in this matter to attempt to strengthen the above by extracting from an analytical table of valvular lesions, cases which might possibly conceal an error, and would only remark further, that this square edging in all its gradations is a very common appearance. And though, by the nature of the case, it is only discovered at a time when many of the opportunities for inquiring into its history are lost, the facts of such cases, as they stand, in no degree contravene, to rate their evidence at the lowest, the inferences which seem fairly to arise from the few cases where the particulars could be accurately ascertained.

There is no reason to suppose that organic reparation, having gone thus far, does not advance beyond this point. But I have not a case at command, to prove that reparation can be complete. All that I have attained to stops here. Indeed, it is hard to see what positive proof could be given of the occurrence of complete organic reparation in any one case: for with the removal of the disease the proof of its previous existence for all purposes of argument is gone.

In some such cases I have assured myself that the functional reparation was complete, though the organic disease had not been entirely removed. But here, too, a link is wanting in the chain of evidence; for I have not yet met with such a case where I have had satisfactory proof that there ever had been functional imperfection. However, this is not an abstract difficulty, as in the case last supposed, viz. the determination of the pre-existence of organic disease, but one which only requires more extended observation for its removal.

Summarily, and subject to all the fallacies already enumerated, the first organic results of acute endocarditis are not permanent, but are replaced after an uncertain period by other morbid appearances denoting the progress of more serious lesions, or of reparation, as the case may be. Perfect functional integrity (presumably functional reparation) is compatible with that degree of organic reparation of whose existence we have good evidence. And the occurrence of perfect organic reparation also is most highly probable. This, however, is a point not to be settled directly by single observation, but by way of inference from accumulated facts, where, therefore, the basis of observation should at least exceed the limits of error, which at present it does not.

II. In tracing the further changes in the process of disorganization of the valves, we are continually reminded of the remark already made that the distinction between simply inflammatory and other changes of the valves cannot always be accurately observed. The distinction is often quite lost in these extreme results of disease. Absorption has apparently the chief share in bringing about reparation of the valves, but henceforth, in tracing the progress of advancing disease, we have to deal with other processes. It is from thickening, contraction, and adhesion, separately or combined, that the most serious valvular lesions in this form arise. And this either directly, or more remotely, in consequence of earthy degeneration or softening; or more remotely still in consequence of changes basing on these last, such as laceration of the valves, or further obstruction by means of adventitious growths.

Thickening, in its most harmless form, occurs in the substance of the valves, far away from their edges, and, but that experience shows that the most delicate valves are strong enough for all the purposes of life, might be considered here as a salutary process. But when accompanied by rigidity, and affecting the edge of the valve, it is, on more than one account, a serious lesion, for it hinders the valves from closing: and judging from the rough edges which such thickened valves commonly present,

it would seem that their surface is liable to be injured by the irregular pressure and tension which their office subjects them to, but which their unyielding nature resents. And as if a certain local irritation was thus constantly kept up, it rarely happens that thickening goes on to any great degree without inducing general contraction.

The effect of simple *contraction** is best seen in the shortening of the chordæ tendineæ of the mitral valve. The mere fact of these cords being shortened might easily escape notice, but for the thickening which would seem to precede, and determine the occurrence of, this action. But none can doubt its importance, as measured, not by lines after death, but by the stethoscope and the observation of the general symptoms during life. In its extreme and most obvious degree, however, and as affecting the valves, this process cannot well be considered apart from *adhesion*, which completes the annihilation of the functions of the valves.

With the conviction that nothing has been made in vain, it is impossible sufficiently to admire the beautiful mechanism of the heart, faintly as we can appreciate its perfection. Each of the interlacing bundles of fibres has a definite duty, oftener than each moment, to perform, and each valve is suited to its purpose in a way that no human art has yet imitated: for the regular action of the best valves in our mechanical contrivances compared with these is so imperfect, that such a state in the heart constitutes disease Sufficient when called into action, when they are not wanted the valves of the heart fall back so as to take up the least possible room, and to leave the passage free. Small in substance, differing little in specific gravity from the medium in which they play, great in strength, and accurate in their adaptation in health, how are they changed in disease! Thick, brittle, rigid, bony, and uneven, projecting into, and obstructing the blood as it goes on, and gaping when they should prevent its reflux,—such might be thought to be the extreme degree of organic change consistent with life. But no; these shrunk and puckered valves can do some duty still, and can still become more diseased. Creeping round the edges of the valves from their re-entering angles, another process now begins, which finally annihilates all their functions. If life is not previously interrupted by some contingent affection, the fissures between the flaps which compose the valves become gradually obliterated by adhesion,* and a little split in a thick, white, rigid membrane, or less commonly, an irregular hole in a kind of diaphragm, is all that remains of the mitral or sigmoid valves.

It will be of interest here briefly to notice two other conditions belonging to this form of valvular disease, affecting chiefly the aortic valves; for they are of a nature not to be comprehended in any merely general description of the successive effects of simple endocarditis, to which, however, the history of the cases renders it probable that they are to be referred.

The first, as consisting simply of the rolling back of the free edges of these valves, analogous in some respects to the rolling up of the great omentum in chronic peritonitis, may fall well under the head of adhesion and contraction, just considered. It is a change that has often been described, and could not well escape observation, the less as the pulmonary valves are so generally healthy as to afford a good standard of comparison for all deviations from the normal configuration of the aortic sigmoid valves.

The other form is, if I may judge by my own experience, very liable to escape observation, unless the attention is particularly turned to it, consisting as it does simply of a lengthening of one or more of the attachments of these valves. In such a case, the finger, when slid down the aorta, does not hitch in the pouch of the affected valve, as it does in the others, but slips on into the ventricle, from the doubling over of the edge of the valve, which is no longer retained by its proper attachment. And that the same yielding occurred during life, when the column of blood pressed against the valve, is shown by the loud diastolic murmur audible in such cases over the situation of the aortic valves. Of the actual cause of this lengthening it would be difficult to speak with certainty. The nature of the changes, however, with which it

* Dr. Corrigan has made a paper on cirrhosis of the Lungs the means for communicating some very good remarks on the effect of contraction on the mitral and aortic sigmoid orifices respectively (Dublin Journ. Vol. xiii. p. 280).

* Dr. Elliotson (Lumleian Lectures), and after him Bouillaud (Des Maladies du Cœur, Tom. 2, pp. 95, et seq.), mention adhesion of the flaps of the auriculo-ventricular valves to the walls of the ventricle as a result of inflammation of the endocardium. I have never met with such a form of adhesion, though the sudden supervention of a loud aortic diastolic murmur at an early period of acute rheumatism in one case made me suspect its occurrence in the sigmoid valves. It is not, however, uncommon to find in cases of advanced disease of the heart that the auriculo-ventricular valves in the neighbourhood of their attached edges are drawn down as it were close to the walls of the ventricle. It seems as if the edge of attachment had extended itself; in other words, that adhesion had taken place. But, from careful examination of such cases, I am more inclined to refer this appearance to the results of contraction of the valve itself and of its chordæ tendineæ.

ordinarily co-exists, and the history of the cases, point to endocarditis.

Doubtless, reviewing this subject, we are right in attaching the value that we do to the little warty granules which furnish the earliest unequivocal evidences of endocarditis. In themselves they are a serious lesion: but their natural tendency is to disappear, to become obsolete, as we say; and their pathological value, as far as they themselves are concerned, declines from the moment that they cease to grow. Though it be the rule that they interfere with the functions of the valves, they do so only contingently. They add temporarily to the mass of the valves, and so far temporarily may interfere with its action. But the injury caused by the slight material addition is trifling compared to the injury done in other ways by the cause of whose action these are the earliest and most obvious results, and which action their existence may possibly tend to renew.

Look, for instance, at an old thickened valve along whose edges a new crop of warty granules has grown up in consequence of recent endocarditis. Will the amount of valvular imperfection which they occasion bear a moment's comparison with that which the immobility of the whole valve, or the contraction of its orifice, gives rise to? Look at the most extreme effects of recent endocarditis on a previously healthy valve; the amount of organic disease there displayed is incomparably smaller than what almost any old thickened valve exhibits, taking into account the extent over which the lesion is spread in each case respectively. There can be no question that the subsequent or consequent changes are of much greater pathological importance than the results of the first outbreak of endocarditis.

It seems that the material importance of these warty growths—that, in other words, which they derive simply from their size—has a tendency to continually diminish. For two or three successive crops of them may leave no more serious organic result than a smooth laminated ridge to denote their previous existence. But regarded in a more comprehensive point of view, pathologically, they are of much greater importance: the inflammatory changes with which they are allied extending back into the substance of the valves, induce graver and more permanent results than the growths themselves. There is, indeed, no sufficient reason to deny that these secondary changes can ever be repaired. On the contrary, the examination of the results of previous inflammation of other fibrous tissues quite justifies the abstract conclusion that they are capable of repair. But the same examination practically forbids us to hope for much in parts circumstanced as the valves of the heart are,—never at rest—quite out of the reach of all mechanical remedies; two conditions these which in affections of fibrous structures are all-important.

The serious nature of any process which induces rigidity and contraction of the valves, diminishing the extent of their efficient surface and impeding their action, is obvious. But we may find other equally serious, though less obvious effects, ensuing upon inflammation of the valves, even in cases where the primary result of inflammation might seem at first sight to have been unimportant, perhaps even salutary.

Of this kind is the lengthening of the attachments of the aortic valves. Looking at the ordinary effect of inflammation in inducing contraction and rigidity, we are not at once reconciled to the idea of fibrous parts being relaxed and lengthened from such a cause. But structures which, in consequence of disease, have, in howsoever small a degree, exchanged their vital powers of adaptation to circumstances for a fixed addition to their mere mechanical powers of resistance, are rarely stronger for the purposes of life. The power of resisting a greater strain by increased mechanical strength is a very poor exchange for the power of repairing the injury which that strain may have occasioned in a weaker part. When this is lost, it fares with the heart as with the hardest substances in nature which have no power of repairing their injuries. It is not a strain now and then, but the constant wear and tear which does the mischief:—"Gutta cavat lapidem non vi sed sæpe cadendo." And the attachments of the aortic valves which have been the seat of inflammation yield to the continued pressure of the blood in the large vessels. If they ever had any increased mechanical strength, they lose it: they lengthen, and allow the edges of the valves to fall over, and the blood to regurgitate into the ventricle.

Valvular Lesions resulting from specific inflammatory or other affections.

The form of valvular disease remaining to be considered differs generally from that just described, in the same manner as inflammation in an unhealthy individual, or in a diseased part, differs from the same process in an individual, or in a part of a healthy constitution. The elementary process may indeed be the same, for of the nature of this we are in almost entire ignorance, and necessarily so; but the ultimate results are different, for the organic elements, though similarly combined, are differently constituted.

Probably, in its lesser manifestations, this

form should not be considered as inflammation at all, except in that vague sense in which we habitually attribute all slow organic changes to chronic inflammation. It is the form of disease to which the lining of the heart and great vessels is specifically liable, under the influence of no more immediately exciting cause than what the advance of years and the circumstances of civilized life supply.*

But to produce any thing beyond these lesser manifestations, or the monstrosities (I know of no term short of this sufficiently expressive), which we occasionally meet with, something more active is required. Such might be acute inflammation, such the condition of the system which accompanies that renal disease with which this form of cardiac lesion has appeared to be connected.

The growths which characterise this form of valvular disease display themselves at the earliest period, as already noticed, as little filaments or buttons attached to the edges of the valves. At this period they are so minute, and their specific characters are so little developed, that it may seem at first sight almost unreasonable to attempt to connect them with the larger masses, which there will be occasion to describe, as their complete manifestation. The argument for their connection rests, however, pretty surely on the occasional coexistence of these two extreme degrees in the same case; on their habitual occurrence, when separate, in cases of a strictly analogous nature, and on the observation of many intermediate gradations by which the change from one to the other may be most satisfactorily traced.

From the observation of a single filament or two, the next step is to that of more growths of the same form, but of larger size. At this point, with the increase of size, the difference between the little pedicled growths and the simple filaments becomes more distinct. Leaving the former, however, for the present, to return to them under the name of Laennec's globular vegetations, we may here trace the successive changes of these filaments from their first indistinct rudiments till they become the broad leathery excrescences which give their name to this form of disease.

The appearances observed in the case which supplies the next observation, in order of time, of the progress of these filamentous growths, are very characteristic. They are described in the notes of the case as long growths of fibrin hanging from the free edges of the valves; two of these long, soft, gelatinous tails floating loose in the stream, while the third, which was of a deep red colour in parts, and sprung more immediately from the corpus Arantii, sat closely, so as to form a double tuberosity on the valves.

From this stage the progress of growth is distinctly to be traced as the threads become flattened, so as no longer to merit the names of filaments, into long, strap-shaped, or triangular excrescences. Smaller processes, however, are generally found in company with the larger masses, whose forms, while they display the stages through

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* I am indebted to Dr. Bond, Regius Professor of Medicine at Cambridge, as on many other occasions, so here also, for the notes of the following very interesting case. Its connection with the present part of the subject lies in a small compass, namely, in the description of the extremely characteristic morbid changes. But the symptoms dependent on these morbid changes are too striking to be passed over. I have given, therefore, the case at length, almost in the words which Dr. Bond has kindly communicated to me from his note-books.

A gentleman of more than sixty years of age presented the ordinary symptoms of hectic fever. He had two paroxysms recurring pretty regularly every day, viz. at 4 A.M. and at 4 P.M. He lost his appetite, and wasted away; and he had all the appearance of senile phthisis, from which he was popularly supposed to be suffering.

But a more particular examination disclosed the existence of a diastolic murmur at the base of his heart, and of an intermitting pulse, and his breathing was uneasy during his sleep.

Such were the symptoms for about four months. During the last few days of his life the dyspnœa became more urgent, compelling him to the upright posture, and expiration being accompanied by a peculiar involuntary sound, like that which paviours make. But occasionally the dyspnœa subsided of itself, coincidently with a large discharge of urine.

On the evening of April 17 he was in great distress; the dyspnœa was intense, but his position was prostrate; he had constant jactitation, and intolerance of the least confinement by the bed-clothes; his mind wandered, but with lucid intervals; there was fair power in the pulse. Towards morning all these symptoms were relieved upon a free action of the bowels and kidneys. He passed that day altogether calmly, and died, in a moment, on April 19, as he was rising from his bed.

On examination of the body:—The lungs were found œdematous, but free from tubercle. The heart was enlarged in all its dimensions, its muscular structure rather flabby; the left side and the entire thoracic aorta were tense with recent coagulum, which entirely filled them; the mitral, and more especially the aortic valves, were fringed, and their surfaces covered with caruncular vegetations,—some forming conical projections, others pendulous—one remarkably long,—of different consistence, but all reducible by pressure with the finger.

Dr. Bond tells me that he has since met with a somewhat similar case, where with a more chronic cardiac affection, declared by a similar diastolic murmur, there was a distinctly marked daily hectic paroxysm. In this case, the constitutional symptoms subsided upon some blood being taken from the scapular region, as indicated by the other symptoms. The same hectic had also been seen in one other case by Dr. Watson, who was in consultation with Dr. Bond on the case above narrated.

My friend, Mr. Scott, of Brighton, had also recently under his care a case very similar to these two less marked ones. Dr. Bond's narrative seems to illustrate the fuller development, and to supply the explanation of the anomalous symptoms in all three of them.

which the masses have attained their present irregular development, point to their possible origin in some cases by rupture of chordæ tendineæ.

It is to these growths that the names of combs, wattles, cauliflowers, and so on, have been applied, as descriptive of their external form, and sufficiently expressive of the difference which subsists in that respect between them and the beading so characteristic of the results of rheumatic endocarditis.

But other processes than the mere addition of new matter on the exterior are generally required to produce these appearances. The effect of simple growth is merely to form long processes or filaments. It is to the other processes, which advance simultaneously with growth, namely, earthy or atheromatous degeneration, laceration, and perforation, of these, and of the parts in their immediate neighbourhood, that the singularity of their form, and the grave extent of the lesion, are mainly owing.*

A more minute inquiry into the nature of these morbid changes gave the following results. As before, the most satisfactory single observations, rather than the general inferences, are detailed.

In one case the growth is described as soft and leathery. It sprang rather from the interior than from the surface of the valve, and the endocardium stopped abruptly at its root. It was possible to distinguish in the mass numerous small granules and some fat. This growth had given origin to a similar one on the corresponding edge of the other flap of the mitral valve.

Another case of the same kind displayed soft growths, over which the endocardium was not continued, but was thickened in their immediate neighbourhood. The growths had a granular, not a fibrous texture; they were insoluble in ether and acetic acid, but soluble in liq. potassæ. Throughout the soft mass were scattered cubic crystals, with striated facets, soluble, with effervescence, in hydrochloric acid. These crystals were accumulated chiefly on the surface of the diseased parts. The same granular texture was observed in the ends of some ruptured chordæ tendineæ which were found in this case.

The above may be taken as fair instances o this second form of disease in its most characteristic development. But I think that, whatever general rules might be laid down, it would be impossible to tell, from the microscopic examination only, except at particular periods and under very favourable circumstances, what was originally the cause of the disease of the valves. At least, after some pains spent in the inquiry, I am sure that I cannot certainly do so.

There is one very important distinction between growths of this kind, and those which result from rheumatic endocarditis; namely, the minute distance which separates the points where we may find severally, the growth advancing, the traces of its degeneration, and a healthy state of the valve from which it has sprung. All these may be seen within the space of three or four lines. This fact quite falls in with the result of general observation elsewhere noticed; namely, that the products of disease bear the stamp of their origin upon them in their tendency to undergo certain ulterior changes, and to decay nearly in proportion to the rapidity of their growth. But it illustrates another very important point; for it has been shown, when on the subject of the first form of disease, of how serious a nature were the changes implicating the structure of the valve, and creeping back into its substance. Such changes are in this form almost entirely wanting. The substance of the valve is affected to no further extent than the existence of the growths would seem sufficient to explain. Even to this extent the different tissues are not matted together: though they are thickened, they appear even looser than natural. On the one side they shade rapidly off into the healthy structure of the valve; on the other, the change into the morbid growth is still more abrupt. The affection of the endocardium especially would seem to be quite secondary, for it commonly terminates abruptly at the edge of the growth, just as the skin does sometimes at the edge of a malignant tumor springing from a different subjacent tissue; and for the reason that this form is not primarily and essentially a disease of the endocardium.

The large size to which such masses may attain in patients scarcely arrived at manhood, or even at puberty, is very characteristic of this form of growth. In one patient, aged 21 years, one of these growths from the aortic valves measured 1½ inches in length.

With these large masses obstructing the onward flow of blood, and with such extreme disease of the valves, it is at first sight almost inconceivable how the circulation can be maintained. But their form and consistence in some degree explain this; at least, as far as concerns the sigmoid orifices, where it is most commonly that they attain to such a size. For the elongated masses or tails floating loose in the onward stream would not obstruct the circulation so much as, from their size, might be imagined; and, during the diastole, there can be little doubt that valves, growths,

* See Watson's Lectures, vol. ii., p. 286, for a description of two cases of this kind.

and all, are jammed down by the backward pressure so as to plug the orifice and prevent regurgitation. Auscultation, at least, tells us that a diastolic murmur is repeatedly absent in such cases.

But this contingent advantage of the soft yielding nature of these growths is more than compensated for by the liability to a most serious accident which not uncommonly befals valves thus affected. Evidence of such an occurrence may be found in their frequent coincidence with torn or perforated* sigmoid valves, and in the ruptured chordæ tendineæ, which cannot always be distinguished from the original filamentous growths. It is important to notice such a secondary lesion, as tending to show that the integral structure of the valve may be altered more than its external appearance might lead one to expect. For such lesions sometimes take place at a point not connected, by visibly continuous changes of the endocardium, with the seat of the principal organic lesion.

The term *ossification*, as implying the formation of anything possessing more of the physical properties of bone than hardness, is scarcely applicable to the changes which ensue in this direction in these cases: that of earthy or calcareous degeneration seems more appropriate. Their importance can scarcely be over-rated; for the existence of the rugged earthy masses thus produced is quite inconsistent with the pliability and the maintenance of that elongated form which alone render the effects of the original lesion less serious. And it is a change which these growths are singularly liable to undergo.

The above description needs but little alteration to be applied to the allied form known as Laennec's globular vegetations. But let the following narrative in the same way supply the place of any more abstract description of these growths, reserving the points of difference for subsequent consideration. The lesions were extreme, and, apart from the immediate object with which the case is cited, it displays well the connection of congenital malformation and albuminuria with disease so extreme and of such a kind.

* Two cases of this nature have been detailed by Mr. Thurnam (Med.-Chir. Trans., vol. xxi., p. 256); one from the notes of Dr. Watson, the other from the Museum of Guy's Hospital. They had each only two aortic valves, both of which were thickened and one perforated. Dr. Watson's case had suffered acute rheumatism, and had apparently diseased kidneys. I do not quote these as being rare, but in order to collate my own experience with that of others. Also Bouillaud (Rhumatisme Articulaire, Paris, 1840, p. 146) describes a case where, in connection with a growth of this nature, one of the aortic valves was perforated.

Thomas Blakey, aged 22,* a hawker, married, temperate, habitually healthy, but always incapable of any great exertion, owing to shortness of breath and palpitation. His mother died of disease of the heart. Once, when a child, he was in imminent danger from a sudden attack of dyspnœa, with lividity; but he has not been usually livid. Eight weeks before admission, after exposure to cold and wet, his feet began to swell, and his symptoms continually increased, till he was admitted with anasarca, ascites, palpitation, dyspnœa, and pain in the right hypochondrium.

On *auscultation* there was a purring tremor over the cardiac region, with forcible impulse and extended transverse dulness. There was a loud, harsh, double murmur over the whole cardiac region, most intense at the junction of the fourth rib with the sternum, but heard also in the ascending aorta. A loud systolic murmur was audible in the epigastrium, and thence was communicated upwards over the region of the right ventricle.

He died in the course of about a month after his admission, the fatal termination being accompanied by pericarditis.

The heart was found, on dissection, generally much enlarged. The tricuspid valve presented, along its free edge, a number of little globular excrescences of a yellow colour, some of which had little irregularly excavated depressions at their free extremities. There were only two pulmonary valves, both of which were thickened and opaque, and had large masses of fibrin, mixed with earthy matter, attached to their free border. One of these masses, three-quarters of an inch in length, which now hung into the cavity of the ventricle, seemed to have drawn out the narrow portion of the valve to which it was attached, by the resistance which it had offered to the stream of the blood. The lower surface of one portion of the mitral valve was crusted with vegetations like those on the right side of the heart; otherwise, like the aortic valves, it was healthy and apparently efficient. There was a free communication, apparently congenital, between the two ventricles, opening, from behind the large carnea columna of the right ventricle. The sinus of the pulmonary artery was considerably dilated; there was much atheromatous disease of the coats of the aorta.

These are changes not to be misunderstood; they interpret themselves. And the symptoms in some of these cases are equally characteristic. They may have been quite absent, or, as more commonly

* Ed. Med. Surg. Journal, vol. lxv.: "On a Sysmolic Murmur in the Pulmonary Artery," this case is more briefly detailed.

happens, present all life in an extremely low degree. But when they are at length fully aroused, they run on to death with great rapidity. And such are the morbid changes on which they depend.

The comparative rarity of these particular growths necessarily renders the description of their successive changes in so far incomplete. But the two extremes are well marked, and we do not need many intermediate links to connect them. One of these extremes has already been described as displaying little yellow globular growths hollowed out with small cup-shaped cavities, bearing some resemblance to the common cup-moss. The other extreme, illustrating the earliest stage, displays them as round granules (studding the edge of the tricuspid valve in the case to which this description particularly refers), rough and apparently partially abraded at their summits, but not as yet softened within; and co-existing with perforation of the mitral valve. The morbid appearances tell their own story of the intermediate processes. The mass softens from the centre, till the shell is no longer able to retain its fluid contents; then the globule ruptures, and the little stem and shell are left behind like a cup, while the rest is hurried along the current of the circulation. The mechanical influence to which these vegetations are exposed, owing to their position on the valves, does not allow the internal softening to advance far before the growth ruptures. And an examination of these masses, as they are found on the valves only, would favour the belief that they are abraded from their summits downwards. The examination, however, of the larger growths of the same kind, as they are found in the tips of the auricles or ventricles, may suffice to correct this impression.

It is with these globular vegetations just as with the long leathery growths already described; they all alike grow rapidly, and as they grow they soften. And though the more variable shape of the filamentous growths does not always allow us to perceive that they have ruptured or been torn as readily as in the case of the globular vegetations, yet attentive observation will often find the proofs of such an occurrence.*

The analogy between these two morb appearances is indeed very close. T first stage in both of them is nearly t same, consisting in the growth of a sm tubercle, filamentous or globular, as t case may be, of a moderately firm cons tency, with a smooth white surface, sprin ing, as far as concerns us on the prese occasion, from the edge of the valves. He the two diverge. The filaments go on become long leathery tails; while the gl bular tubercles assume the form of litt buttons with contracted bases. There a certain obvious mechanical consideratio which go some way to explain the muc larger size to which the long filaments ma attain, as compared with the ordinary d mensions of the globular vegetations; bu

* Laennec quotes a case from Crüwel, where one of these globular vegetations had apparently been detached from its situation, and carried down the stream, as far as the pulmonary valves, where it had caught. The growth in this instance had undergone partial earthy degeneration. (Aus. Méd. iii. p. 265.)

The more ordinary course of events is, as above stated, for the growth to be broken down and its component particles to be mixed with the circulating fluid. The whole subject of capillary phlebitis—the changes to which these particles are thought to give rise—has been so ably considere by Rokitansky, that any remarks of mine on th anatomy and pathology of these morbid appear ances would be superfluous. Referring to hi work (Path. Anat. Band i. S. 242; Band ii. 680, &c.) for such information, I would here onl subjoin an analysis of twenty-seven observation of this disease which have occurred to me illus trating its connection with valvular disease o the heart.

Although these growths are found to exis where the valves of the heart are quite healthy yet there is certainly a very close relation o coincidence between them and valvular disease especially what has been described as of the second form. For of twenty-seven cases where these growths were observed, nine had disease of the valves of this form, and two had Laennec's globular vegetations either on the valves or on the muscular walls of the cavities of the heart. Four more had simple extreme atheroma of the valves without lesion of the surface. In one case the heart could not be examined after death. In the remaining eleven the valves were either quite healthy, or the amount of disease was very slight. But one of these had little organized tufts, growing from the interior of the ascending aorta, and six of them had ulceration, simple or specific, of internal organs. One was a young healthy man killed by a fall.

Allowing largely for errors of observations, there are two facts which come out most prominently from this analysis:—First, the connection of this peculiar morbid appearance with the second form of valvular disease; and, secondly, the want of any connection between capillary phlebitis and tubercular deposition,—though this last result appears rather in arranging the cases for analysis, than in the analysis itself. The almost uniform absence of tubercles from all the other organs in each case shows that, whatever ground for error there may be in other particulars, there is very little real danger of confounding capillary phlebitis with tubercle. This fact quite agrees with the observations of the extreme rarity of the coincidence of phthisis with valvular disease; though the form of valvular disease which coincides with capillary phlebitis is of the two the more compatible with the tubercular diathesis.

In the twenty-seven cases at present under examination, this morbid appearance was found in the spleen eighteen, kidneys eleven, lungs five, and brain three times. It is worth consideration how many of the cases of softening of the brain which accompany extreme valvular disease of the heart may be referable to this cause. In the muscular substance of the heart I have only met with one, and that a very equivocal instance of capillary phlebitis.

e cause which originally determined the fference of form has doubtless the largest are in influencing the mode of succession the further changes.

For though their elongated form may deed protect the growths which affect is peculiar shape, yet they have an addi- onal protection in their firmer internal xture, in which, from whatever cause, the ftening appears not to advance as rapidly s in the rounder masses. We may occasion- ly find quite long filamentous growths dis- laying no evidence of laceration, while the sults of mechanical injury constitute one f the most striking features of the globular getations on the valves. On the valves: r as we have seen, growths of the same ature, when occupying in comparative uiet the apex of a ventricle or auricle, ttain a large size. We need all this to how that these two kinds of growths are ot identical: of their close analogy there s no room to doubt.

The fibrinous patches which may some- imes be seen overlaying the left ventricle, s it leads up into the aorta, have already een noticed, and their origin connected with the first form of valvular disease. They have their analogues in the form un- er consideration, in the papillary growths which are found clustering in patches in the left auricle. These have been compared to the papillæ inside a sheep's mouth, to which, indeed, their resemblance is tolera- bly exact. Of five cases where these papillary growths were observed, four had leathery accretions on the valves. The same coin- cidence was noticed in one of Laennec's cases.* Further, four of these cases had granular disease of the kidney; in the fifth there was no opportunity of ascertaining this point.

It has already been shown that the imme- diate consequences of endocarditis—those, namely, from which we infer its recent ex- istence, are less to be dreaded than those which may more remotely arise from it. The same may be said generally of the morbid changes belonging to the second form of valvular disease. But, there the changes were to be traced in the valves apart from the growth; here the subsequent changes have a closer connection with the morbid product originally charac- terizing the disease. Perhaps the state- ment that the valves in the one case, and the growths themselves in the other, are the seat respectively of the secondary conse- quences most to be dreaded, would not be made clearer by being qualified so as to meet every case. But, speaking generally, and more particularly with regard to the extreme cases, we may safely adopt this expression of the difference of the seat of the changes which lead to disorganization of the heart in the two forms respectively.

* Aus. Méd. iii. p. 337.

LECTURE III.—Part I.

IV. Symptoms and Treatment of Valvular Disease of the Heart.

Earliest evidences of valvular disease. Endocarditis, its obscurity, encouragement to its active treatment—its consequences, chronic valvular disease.

General considerations. Physiognomy of valvular disease, affected by age. Hypertrophy and dilatation, their causes, purposes, and contingent dangers—their relation to treatment. Affections of other organs—their importance—the mode of their succession—their relation to particular cardiac lesions. General principles of treatment.

Cerebral complications:—Apoplexy and softening, their relation to valvular disease—other affections—principles of their treatment.

Pulmonary:—Emphysema—pleural effusion—pulmonary obstructions as abiding or temporary condition—mode of relief. Pathology of cerebral and pulmonary affections contrasted.

Abdominal:—Structural affections of the solid viscera—their pathological interest. Vomiting and diarrhœa, their treatment and pathology.

We have now reviewed the causes of valvular disease, the physical signs by which that disease makes its presence known, and the structural changes of the valves on which those signs depend. It remains to consider the symptoms which these valvular lesions give rise to, and, little though it be, what can be done for their relief.

Our knowledge of the symptoms of the first beginnings of valvular disease is almost confined to cases of rheumatic endocarditis. Even here, where we have learned to look for them, they are often obscure enough. And, that they rarely force themselves upon our notice unsought for, we are assured from the long time that the connection between rheumatism and endocarditis remained unknown.

The only sign of valvular disease at its commencement, in many of these cases, is an endocardial murmur. In some, however, there may be uneasiness, and even pain in the region of the heart, with increased hardness and frequency of the pulse. In others, percussion may show, besides, that the heart occupies more than its natural space, a condition which, in its occasional connection with other symptoms, and from the good effects of depletion in relieving it, may be considered as an effect of inflammation. Pain then, and swelling, or distension (if we may venture to call by one of these names the cause of this increased dulness), with the general signs of inflammatory fever, and the local auscultatory signs are the means, by some or all of which the presence of valvular disease, or, more correctly speaking, of endocarditis, may be manifested in the first instance. Endocarditis, it is true, like pericarditis, may sometimes display itself through the nervous system, in the symptoms of the most violent, even of fatal chorea.* It is necessary to bear in mind the possibility of the occurrence of such extreme cases. But practically, it is much less important to know how anomalous or violent the symptoms of endocarditis may sometimes be, than to be assured how obscure they usually are—how often they are entirely wanting; and this at the time when their effects are to be obviated, or if ever, to be removed.

It scarcely falls within the scope of these remarks to enter at length upon the treatment of endocarditis, or other causes of valvular disease, did either space allow, or had I anything to add to what others, and most recently, Dr. Latham, have told us. The sum of my experience may be very briefly expressed:—Of cases of rheumatic endocarditis treated in all ways, the immediate and ultimate results were better in

* Clinically, cases of chorea with rheumatism should be divided into two classes. First, those coinciding with capsular rheumatism, where the articular affection is commonly slight and chronic. Second, those coinciding with fibrous rheumatism, where the articular affection is commonly very intense, and there is pericarditis or endocarditis also.

What share the rheumatism itself may have in inducing the nervous symptoms in either of these classes I could not say. I would accept the explanation, which is sometimes offered, that it acts as a blood poison, inducing organic disease of the heart, and functional disturbance of the brain, only as an expression of our ignorance, not as a solution of the question. For I have seen fatal chorea with pericarditis consequent on disease of the kidneys, independent of rheumatism. I am inclined to connect it with the cardiac affection.

Whatever share, however, carditis (using this as a general term) may have in causing the chorea, we must not hastily dismiss the consideration of its connection, in treating the disease. For the prompt recognition and treatment of the cardiac affection appears to be a very important element in ensuring a successful termination of the case. Dr. Burrows, Cerebral Circ. p. 215. See also Dr. Kirkes, Med. Gaz. vol. xlvi. pp. 1004-1049.

those treated actively by mercury and depletion, than in those treated in any other way.* I cannot express in less vague and general terms the opinion which these cases have seemed to justify on this point of practice; and it is needless to enumerate the possible sources of fallacy which this expression may conceal, especially with regard to the ultimate effects, with which we have here chiefly to do. Only I would not withhold this testimony, however inconclusive, to the correctness of the practice inculcated by Dr. Latham, as having had large opportunities, during my connection with St. Bartholomew's, of observing its results, in the field from whence his information was derived.

The attack of acute rheumatism passes away: the acute period of endocarditis has gone by, perhaps never to return. And with it, too often, all prospect of a cure passes away. We have now no longer to deal with acute inflammation, but with its organic results, and with the chronic changes which these morbid products undergo. The treatment has no longer in view the cure of the disease, but merely the palliation of its symptoms.

The question what becomes of these patients has already been discussed on a previous occasion, on other grounds. We have here to do only with those in whom the disease goes on, from bad to worse, to destroy life at a more or less remote period.† And here all the distinctions of the causes of valvular disease cease in the consideration of the symptoms common to the secondary effects of them all.

The ways in which suffering and death may ensue in the subjects of valvular disease of the heart are various. The difference chiefly arises from the prominence which diseases of particular organs assume in the several cases. The subject naturally divides itself in correspondence with these secondary affections. But, before entering upon their examination, there are some more general conditions, of much interest and practical importance, which require a separate consideration.

I. There are few diseases but have their peculiar physiognomy.* Among the many advantages resulting from the collection of vast masses of disease at our large hospitals, must be reckoned the opportunity thus afforded of learning rapidly to recognise the external characters, literally the features, of disease. Disease of the heart, too, stamps its peculiar marks on the face. It will not be superfluous to trace what Corvisart† did not deem beneath his notice.

Many diseases communicate to the face an expression of their own, in which all distinctions of age are merged, and the child puts on the face of the old man. But it is not so with valvular disease of the heart. On the contrary, we may readily, and without any forced refinement, distinguish between the features which childhood and more advanced age respectively assume under these circumstances‡.

In children suffering from valvular disease of the heart, the face and conjunctiva are pale and bloodless. Only, through the unusually transparent skin, small vessels may be seen ramifying over the malar eminences and the alæ of the nose, and the face becomes dusky on any exertion or emotion. The features are full, not pinched, as in most of the chronic diseases of childhood. There is no expression of anxiety. With the eyelids partly closed, and the eyes turned down, they sit, motionless, save for the constant heaving of their chests, and even of their whole bodies, with the beating of their hearts. Sad experience has taught them which posture is the freest from suffering, and this they steadily maintain, either upright, with the head leaning a little forwards on the chest, or, as the disease advances, reclining back so as to extend the trachea to the utmost: doubtless, on account of the relief which they thus afford to some labouring organ. They are sensible, and intelligent often to a remarkable degree, like other children who have suffered much from sickness, and seem to

* I am led to this conclusion, as far as concerns the immediate effects, by comparing cases submitted to all forms of treatment during their stay in the different wards of St. Bartholomew's Hospital, or wholly neglected for an indefinite period before their admission. Of the remote effects I have less certain means of judging; none, indeed, but what chance cases, which have come under observation at a subsequent period, supply.

† From on analysis of my own cases, in about eight years. Probably an analysis of other than Hospital cases would give a much higher average.

* Dr. Corfe's Remarks on the Physiognomy of Disease, p. 71, and Dr. Hope, op. cit. p. 401, may be consulted on this subject.

† Le hazard fait tomber les yeux de Corvisart sur un portrait: à l'instant, il s'écrie: "Si le peintre a été exact l'original de ce portrait est mort d'une maladie de cœur." Corvisart avait diviné juste.—Pariset, Eloges, tome i. p. 111.

‡ The differences of the expression of patients suffering from valvular disease of the heart have appeared to most who have written on this subject capable of being reduced to two classes. Dr. Bellingham, Med. Gaz. vol. xlvi. p. 562, would connect them with differences in the nature of the valvular imperfections. Hope and Corvisart connect them rather with the temperament of the individual. My own observations generally agree with their conclusion, for the divisions in which youth and the lymphatic temperament respectively predominate usually coincide. But I think that, where the two divisions do not exactly coincide, the age overrules the temperament.

take pleasure in being auscultated and made objects of attention. But as soon as they are left to themselves, their eyes fix again upon some object, and they lie looking at it without apparently noticing it, like figures in wax.

The expression is quite different in persons of more advanced age; it is no longer one of placid quiet, but of anxiety and pain. There are still the same constrained unnatural postures, and the same fixed yet vacant gaze, but the face is drawn and livid, the lips are blue and pinched, the cheeks are of a dusky purple, and wherever the skin is put upon the stretch by subjacent parts, vessels may be seen ramifying just beneath the surface. Commonly the skin hangs in a fold from the lower eyelid, so as to expose the dull dingy conjunctiva covering the sclerotic; bloodless, indeed, but wanting that pearly white which we see in anæmia. Much as bronchitis has to do with the sufferings of these patients, and with these particular appearances, yet attentive observation will recognise here quite a different expression to what we see in connection with that disease. There is neither the intense restless agony of the present attack, nor the passive endurance when the acute stage of bronchitis is past. But to an expression which might suit with the most urgent distress and despair, there is joined a quietude of demeanour which might betoken perfect resignation. In all the patient's distress and uneasy movements, the great object of his efforts is to maintain the most complete rest. As if it were a question whether to bear the present posture, or the wearying search after one more tolerable to his feelings, were the greater misery. "He cannot bear to be at rest, for rest brings him no relief; he seeks but in one to be freed from the feeling of another pain."*

II. The effect of valvular disease on the heart itself in producing hypertrophy or dilatation of its walls or cavities respectively, enters into the pathology of the secondary complications of valvular disease to such an extent as to require preliminary consideration. Hypertrophy and dilatation, like most scientific words in very common use, are not always applied with strict precision to the things intended to be so designated. In their ordinary acceptation, however, these terms are quite sufficient for most, as for the present purposes; the one or the other, or both, being employed as the condition thereby expressed predominates. Hypertrophy, indeed, almost always implies the co-existence of some degree of dilatation, and the converse.

Dilatation would seem to be most commonly predominant on the right side of the heart and in the auricles, essentially the feeblest parts of the organ. Hypertrophy is found most often, and manifests itself most strikingly, in the left ventricle, essentially the strongest chamber of the heart. But as disease of the pulmonary valves is as surely followed by hypertrophy of the right, as is disease of the aortic valves by that of the left ventricle, the explanation why sometimes dilatation ensues, and sometimes hypertrophy, must be sought rather in the difference of their causes than in that of the constitution of the two sides of the heart respectively. Dr. Hope says:*—

"The exciting causes of hypertrophy are equally those of dilatation; and supposing no unknown agencies to interfere, it depends on the proportion which the cause bears to the reacting energy of the cavity exposed to its influence, whether that cavity become affected with hypertrophy, with dilatation, or with a combination of the two. It may be said generally, that when congestion is *constant* in a cavity, dilatation is more commonly the result; and that when there is only resistance to the expulsion of the blood, without constant engorgement of the cavity, it is more common for hypertrophy to be produced. Contraction, for instance, of the aortic orifice, causes hypertrophy of the left ventricle in a greater degree than dilatation; whereas, patescence of that orifice, attended with regurgitation, and constant engorgement of the cavity, causes dilatation in a greater degree than hypertrophy."

Perhaps this is as near the truth as we are likely to attain, for the principle is not of very generally precise application, the causes being commonly mixed: but when an opportunity does occur of watching the effects of these causes separately, the result is agreeable to Dr. Hope's observation. Thus, emphysema and chronic bronchitis, which always cause more or less obstruction to the circulation, but on occasion for a while completely interrupt it, and cause the blood to accumulate on the right side of the heart, induce dilatation of these cavities. Granular disease of the kidney, on the contrary, where the obstruction to the circulation is tolerably uniform, and habitually surmounted, induces hypertrophy of the corresponding ventricle. It would seem, too, that hypertrophy ensues rather in the robust, dilatation in the cachectic and weakly. And, as a corollary to this, dilatation more commonly affects both sides of the heart than hypertrophy is found to do.

* Aretæus de Tonsillarum ulceribus.

* Diseases of the Heart, 3d ed. p. 250.

In one point of view—namely, in relation to the maintenance of the circulation—hypertrophy and dilatation may be considered as conservative processes:* for, unless the heart be made strong enough to overcome the obstruction, or large enough, so that a sufficient quantity of blood shall, after allowing for regurgitation, be distributed to the different organs, the man must die, or at least be incapable of exertion. But by such means, in some cases, and supposing that the valvular lesion remains unaltered, health may be *pro tanto* restored; and thus life may be prolonged for many years, and the patient remain ignorant alike of the existence of his disease, and of the beautiful adjustment by which it is rendered imperceptible or tolerable to his feelings.

But, in other points of view, hypertrophy and dilatation must be regarded as in themselves most serious diseases. The same circumstance which gives rise to these changes of the heart's structure necessitates the continuance, and commonly the increase of them. For even supposing that the valvular lesion were always to remain the same, it is easy to see how the adjustment can only in very few cases be absolutely and permanently perfect. Apparently, the effects of sigmoid obstruction would be most remediable by these means, then those of sigmoid regurgitation, and least of all those of auriculo-ventricular regurgitation. But the fact that the valvular lesion does very rarely remain unchanged renders it quite superfluous on the present occasion to discuss that hypothetical case. Whether or no hypertrophy and dilatation must still go on increasing in any particular instance, even though the valvular lesion remain unchanged, at least they certainly do so in the vast majority of cases where the valvular lesion does not remain the same.

Mechanically, indeed, the remedy is temporarily perfect. An hypertrophied heart can sustain the contraction long enough, or impel the blood with force enough, to maintain the normal pressure beyond the stricture. Again, a dilated heart can contain blood enough to allow for regurgitation to any amount. But the cure of a diseased heart, unhappily, is not a question of mechanics; for this mechanical remedy acts more or less at the expense of the capillaries, pulmonary or systemic, against which is now made the pressure which the walls of the heart were by nature intended to bear. Hence hæmorrhage, or œdema, visceral obstructions or inflammations, under one or all of which the patient sinks.

More than this: hypertrophy and dilatation are temporary expedients to prolong life, requiring continual adaptation to the progressive alterations of the valvular lesion whose effects they are calculated to obviate. While we admire, to the fullest extent, the manner in which the heart can thus make good its own defects, and adapt itself to the altered circumstances of disease, we cannot fail to see that the resulting condition is in itself one of serious disease. They are necessary evils—necessary to support life under the circumstances, but not the less evils; for it is not in the body, as in a coarse machine, that there is merely so much work to be done: it is of the first importance how that work is done. The circulation must not merely be maintained any how, but it must be smoothly and uniformly maintained. And this is very rarely the case in valvular disease of the heart, however perfect the compensation may seem to the patient himself, or to ordinary observation. A careful examination of a patient with valvular disease of the heart, for the relief of whatever ailment he may have applied, rarely fails to disclose to the physician the existence of the cardiac lesion: and it is most commonly a morbid state of the circulation, as indicated by the pulse, the immediate consequence of hypertrophy or dilatation, which leads thus directly to the discovery of the disease.

It would be impossible to determine absolutely the limits within which these changes of the muscular substance of the heart might be considered as safe or salutary, for probably they vary much in different cases; but the limits apparently are generally very narrow, and soon passed. Obviously the condition of the heart is neither safe nor wholly salutary when it is a source of distress to the patient; and scarcely so when, as just instanced, a full, or hard, or jerking pulse, denotes to the physician the abnormal pressure which is made on different organs, or the irregularity of the supply of blood which they receive.

But even supposing that no functional derangement detectible either by the physician or the patient arises, still the patient whose circulation has to be maintained by a dilated or an hypertrophied heart, however accurate the adjustment may be, is not in a state of health. Obviously it is a very serious state of things when the pressure which should be resisted by the

* "In speaking of what *nature intends*, it is difficult not to use language which may seem to favour some foolish hypothesis; but, in point of fact, we always must form a judgment of what nature is aiming at whenever we interfere with what nature is doing. And, as our practice is sometimes right, it is presumed that a right judgment is both attainable and sometimes attained."—Latham, Diseases of the Heart, vol. ii. p. 344.

mitral valve—to take the commonest case—has to be supported by the delicate pulmonary capillaries, with only that amount of relief which the increase of capacity of the auricle supplies. It is life bought by disease, by sheer force—a make-shift, not a remedy. Nor yet, when the heart itself sustains the increased pressure, as in the case of sigmoid obstruction, does hypertrophy consist with perfect health; for the power of adaptation to different circumstances, wherein animal mechanism surpasses artificial contrivances, is here most sorely impaired. The power of the hypertrophied heart is indeed most accurately calculated to the mean daily requirements of the body; yet, though the adjustment be correct, and the heart's action safe for all ordinary purposes, the impulse of an hypertrophied heart under excitement cannot be regarded without apprehension. Take, for instance, such a case of aortic obstruction where the impediment is exactly counterbalanced by the increased power of the heart. It is obvious that the more the arteries are distended on the further side of the obstruction, the more forcibly will the shock of the heart's contraction be transmitted past the obstruction to distant parts. The most trivial cause, impeding for a moment the flow of blood through the jugular veins, may readily induce such a condition of the arteries leading from the heart to the brain: the more readily, the slighter the obstruction—that is to say, generally, the earlier in the disease. And when the effect of the organic obstruction at the sigmoid orifice has been thus neutralised, a few full, forcible contractions of the left ventricle may place such a patient in the most imminent danger of extravasation of blood into the substance of the brain.

It is our duty as physicians to prevent, as far as may be, what we cannot cure. This rule must never be absent from our minds, but must enter into all the minutiæ of the treatment of valvular disease of the heart. By enjoining rest and prudence we may delay the advance of dilatation and hypertrophy; but we cannot reasonably attempt their cure, except by removing their cause, which to a great extent is impracticable. It is worse than idle to cup and leech, and use antimony and mercury, to try to cure them, or the disease of the valves on which they depend, except so far as their existence is connected with a condition of parts or a general condition indicating the employment of such remedies. Such treatment cannot remove them; and, if it could, we must needs bring them back again, or the patient would die.

The rational indications of treatment in many such cases are satisfied rather by the use of quinine and iron* than by antiphlogistic means. For serious as are the secondary effects of increase in the strength and capacity of the ventricles on other organs, the direct effects of weakness of the heart are much more to be apprehended. It is a very imperfect view of the effects of valvular disease on the heart itself which sees only a progressive increase of its strength or capacity, and does not look forward to that period when its powers of adaptation fail, and, instead of rising to meet, the heart sinks beneath its burden.

The data are as yet wanting from which to investigate this most important practical question,—under what circumstances, namely, this state of things occurs. On the more obvious explanations, which the effects of the original size of the coronary trunks in limiting the supply of blood, or which the deteriorated quality of the imperfectly aerated blood suggest, there is as yet room for little beyond conjecture, however probable. Only let us keep clear of one fallacy. Fatty degeneration of the heart is apparently the natural termination of hypertrophy of that organ where the hypertrophy has depended on a temporary cause. But such is essentially not the case in valvular disease. The cause of the hypertrophy is rather progressively increasing than temporary. These two forms of disease of the heart—fatty degeneration and valvular disease—have little in common: their coincidence is but rare, and the mode of their connection in these rare cases quite inexplicable.

But there will be occasion to refer to this hereafter. The failure of the heart's action marks a distinct period; and, whether it be the rhythm or the power that fails, the influence that should direct, or the substance that should sustain the action—whether it supervene in the same gradual way as the hypertrophy, or in a moment fatally, it is a matter of the deepest interest. It is the last alternative awaiting those who have avoided or struggled through those complications of other organs which first demand our attention.

III. However great be the value of observations of the effect of valvular disease in inducing hypertrophy and dilatation of the heart, as a measure of the virtual amount of that disease, yet, as a rule, the duration of life under such circumstances depends more on the degree to which the various organs can assist or tolerate the impeded circulation, or the heart can accommodate itself to its altered circum-

* See on this subject Dr. S. Alison on the Use of Iron in Organic Affections of the Heart; and a most excellent practical paper by Dr. Law, Dublin Medical Journal, vol. xvii. p. 192.

stances, than on the amount of valvular imperfection. Of this degree, obviously, these changes of the muscular substance of the heart afford very inadequate indications. The grounds for prognosis are to be sought rather in the age and condition of life and constitution of the patients. Children, in whom there is scarcely any limit to the tolerance of their various organs, and in whom the principle of growth is so active, and those whom Providence has placed above the necessity of labouring for their existence, may bear almost without distress an amount of valvular disease which would sink a poor, or an old, or a weakly patient at once.*

And, as man is born to toil, and liable to disease and to old age, so these conditions interfere largely to prevent the most natural being the most common termination of valvular disease. The secondary symptoms succeed each other very regularly. Commonly the failure of the action of one valve, most frequently on the left side, impedes the regular course of the circulation behind it; and hence ensue dyspnœa,† and all the symptoms which betoken the obstruction of the passage of blood through the lungs. At a later period, when the faulty action transmits its influence yet further back, through the right side of the heart, anasarca follows, with congestion of other organs besides the lungs: and it is from one or all of these that the patient sinks, under local disease or general exhaustion.

To all these patients the condition of each of these organs, and of the general powers of the constitution, are of as great importance as is the condition of the heart itself; for though there are very few diseases of the different vital organs in whose secondary affections we should not have to include diseases of most of the other vital organs, it is so, perhaps, in the highest degree as concerns diseases of the heart. And here the secondary affections have yet a graver interest, in the fact that it is by their means, in the majority of cases, that the primary disease of the heart proves fatal. Headache,—to take an instance from the brain,—epistaxis, even mental anxiety, which might be safely overlooked when secondary to disease of some other organs, require continual watching in cases of disease of the heart; for they indicate both the organ through which the fatal termination may be reached at last, and the approach of that event.

It does not appear that any of these secondary affections of particular organs are definitely and exclusively connected with any particular valvular lesion. Their local origin cannot be stated with more precision than to connect them simply with disorganization of the right or left side of the heart, according to the situation which the suffering organ holds in relation to either of these chambers; nor does the intimate mode of their production admit of any more elaborate subdivision. They depend simply on increased or diminished arterial pressure, or on venous obstruction.

The effects which such changes in the degree of arterial pressure produce are best displayed among the cerebral complications of valvular disease, for there only do these changes assume any pathological importance; but the effects of venous obstruction, which is the more usual form of derangement of the circulation, seem to require some general notice in this place.

As a rule, such secondary effects of valvular disease are displayed behind—in the order of the circulation—the particular chamber of the heart affected. Affection of the left side of the heart, for instance, is most often attended by pulmonary obstruction or œdema; of the right, by general venous congestion or anasarca. In practice, one of the earliest indications of imperfection of the heart is usually found in dyspnœa on exertion; while the accession of general dropsy, or its rapid increase, commonly denotes the entire failure of the system, and the approach of death. Abstractedly, however, we are not justified in disregarding failure of the left side because its symptoms occur in slight cases, often many years before death; nor are we correct in attaching that absolute value to functional imperfection of the right side of the heart which the circumstances under which its symptoms are usually met with at first sight seem to demand.

Whether disease of the valves of the

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* It is a matter of interesting consideration what share other peculiarities of children, besides the greater tolerance of their various organs, may have in sustaining life with such disorganisation of the mechanical apparatus of the circulation. The greater extent of the respiratory movements, and the comparative freedom of the lungs from disease—phthisis, perhaps, excepted, which, however, has little to do with the present question—naturally suggest themselves; and others will readily occur: but, besides these obvious differences between childhood and more advanced age, I think that we must not overlook the *vis a fronte*, the force which draws the arterial blood to the capillaries, as distinguished from the *vis a tergo*, the impulsive action of the heart. Perhaps, indeed, this is only the legitimate physiological expression of what we are content to call clinically the tolerance of the organs in childhood—that the organs are not really passively tolerant of congestion, but actively engaged, by the aid of the organic affinities which are so much stronger in children than in adults, in thus maintaining the burthen for which the heart is insufficient. See on this subject Reid's Physiological Researches, p. 49.

† Andral (Clin. Méd., iii. p. 100) thinks that dyspnœa does not supervene so early in proportion in old as in young subjects of valvular disease.

right side of the heart is more rapidly fatal than disease of the left valves, I am not able to determine; for the ordinary results of practice give no means for settling this question, which it must be allowed that the more limited observation of less equivocal cases leaves doubtful. We cannot, in the majority of cases, judge of the absolute pathological value of disease of the right side of the heart from its apparent effects; for these are usually mixed with, and, indeed, based on, the effects of disease of the left valves—namely, on a condition of the pulmonary similar to that of the systemic capillaries. And by the time that the effects of the obstruction have reached the systemic capillaries, they have made themselves felt pretty well all through the circulation. For by this time the disease has probably assumed a very serious aspect, from the secondary affections of the left side, independent of this new source of danger and distress from those of the right.

Whatever be the importance of functional imperfections of the right side of the heart,—and great, confessedly, it is,—yet they should hold a lower place, pathologically, than diseases of the left—the effect, to wit, being lower than the cause. For dilatation and hypertrophy—the indices of the virtual amount of imperfection—are, with two exceptions,* rarely met with independent of organic valvular disease, that attribute of the left side. All experience agrees in placing, on such grounds, disorganization of the left side of the heart first in the series of changes, and assigning to it the highest importance. Functional imperfection of the tricuspid valve, and consequent disorganization of the right side of the heart, however frequent and serious its occurrence may be (and I may, with many others, have often erred in overlooking its existence), is commonly but a secondary effect of disorganization of the left side.†

IV. In the nature of the secondary affections themselves, abstractly considered, there is little peculiar; but in the mode of their treatment and the choice of remedies there is much to be remarked. The bronchitis, for instance, which ensues upon disease of the heart, does not differ in such a way from that which arises in connection with disease of the kidneys or emphysema that it could be certainly recognised as such at first sight; but the difference may be well made to appear in the treatment. From this point of view the secondary affections have been chiefly regarded in the following remarks. The cure, indeed, of the original disease is a matter to be left out of consideration; but its existence and continual agency must never be lost sight of. Our treatment must relate entirely to the secondary symptoms; bnt it must be modified by the knowledge that, as the cause of the disease will still remain behind, the same thing may still have to be done over and over again, and each time under less favourable circumstances than before.

Yet, indeed, these are not cases to leave alone, nor routine cases, to all of which one uniform plan of treatment is applicable. Each case is a study of itself; and, provided only that the "*nimia medici diligentia*" do not place the patient in a worse condition to resist any future attacks, there is no cause to fear doing too much to relieve these patients. And the close attention to their symptoms which such a plan of treatment would involve, is now and then rewarded by the discovery of some happy idiosyncrasy with regard to the effects of particular remedies, or of the action of particular organs on the system at large.

Our knowledge of diseases of the heart has advanced much since the time of Senac; but could the most accomplished physi-

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* Viz. dilatation of the right side of the heart, consequent on pulmonary, and hypertrophy of the left, on renal obstruction.

† The subject of functional imperfection of the right side of the heart engaged the attention of the late Mr. Wilkinson King (Guy's Hospital Reports, vol. ii. p. 104) and others. More recently, Dr. Blakiston (Diseases of the Chest) has pursued the subject in its pathological bearings.

Of the fact that the tricuspid does not act as perfectly as the mitral valve at all times, there is scarcely room to doubt. Of the cause of this imperfection there is some difference of opinion. Physically, indeed, I think we must admit that the tricuspid valve is less efficient than the mitral,—as far, at least, as the very simple, but very equivocal, experiment of injecting water backwards against the valves of the dead heart goes; but that regurgitation always takes place at each contraction of the right ventricle is scarcely admissible. The hypothesis that tricuspid regurgitation takes place on occasion, when required, by dilatation of the right ventricle, the segments of the valves being by this means drawn asunder, in consequence of the divergence of their bases, is more satisfactory; and Dr. Blakiston's pathological observations of cases where dilatation has become the abiding condition greatly favour this view.

But I think that all purely physical explanations fall far short of the truth. The more complex structure of the right ventricle, and its comparative immunity from disease, seem to show that its functions are of a less purely mechanical nature than are those of the left. Apart from these very important considerations, to which Dr. Williams (Diseases of the Chest, p. 266) has called attention, it would be difficult to believe that so important a function as the regulation of the pulmonary circulation had been left to any mere mechanical contrivance, however beautiful, and that no remedy had been provided, under such circumstances, against congestion of the lungs, short of that degree which should thus relieve itself by forcible distension of the right ventricle.

cian of the present day express more clearly the rules that should guide their treatment than Senac* has done? His remarks apply, it must be observed, to what we commonly know as old disease of the heart. Senac's knowledge, however, stretched beyond that of the past epoch of *morbus cordis* to that of the present day.

"The further we explore the subject of diseases of the heart, the more barren in resources does medicine appear. The remedial agents which are required are but few, and those who are so prodigal in the application of remedies, are ignorant alike of the causes with which they have to contend, and of the real use of the means which they are employing. The resources of our art rest rather in the patients themselves than in any drugs. What can we hope for from medicines, for instance, in dilatation of the heart? Will they restore to its normal form an organ whose action is always abnormal? If the substance of the heart become bony, will medicine soften it? Will medicine melt down tumors or earthy concretions? Can it enlarge constricted passages? Will it dissolve polypi that resist all known solvents? Ignorance and credulity alone could hope for such unheard-of success.

"Must we, then, leave these diseases to themselves? — No; but our powers are limited to arresting their progress, to alleviating their symptoms, to preventing or removing their consequences. The fundamental cause is abiding, and on this, as its basis, all the treatment must hinge. If we adopt any other view, we shall expose our patients to risk without any chance of benefiting them.

"I say that the fundamental cause will persist in spite of our efforts. This is true for the most part; still, if we could take these diseases at the beginning, probably many of them would be amenable to treatment. How many observations have proved that internal aneurisms are not quite beyond our resources! May not aneurisms of the heart and ascending aorta, and other cardiac diseases, be in the same case?

"But what are the remedies from which we could hope to derive any advantage? They must be suggested by the causes from which the patient has reason to apprehend danger. Such causes, summarily, are those which may either disturb anew the action of the heart, may overload its cavities, or may check the circulation there. * * * * * *

"After having provided for the safety of the heart, as far as regards the blood, the functions of the stomach and of the bowels, the condition of the nervous system must not be neglected. * * * *

"It is not less necessary to preserve the tranquillity of the mind. The passions disturb the action of the heart, agitating it even when it is free from disease. We must needs, therefore, avoid them, or moderate them, and forbid all employments of the mind requiring long application or over-exertion.

"The treatment of the symptoms must be guided by the same principles, our remedies being employed strictly according to the indications and urgency of the case. I will not enter here into further details, but give only general rules. It seems hard that, after speaking of particular diseases, one should be able to point out no more particular mode of treatment: but this is not the fault of our art; we can expect of it no more than what is possible, and it cannot possibly repair what in its nature is irreparable."

I. *Cerebral complications.*—To the description of the cerebral complications of disease of the heart which Dr. Burrows* has given us, there is little to add. I would only, as having drawn my experience from the same field—namely, the wards of St. Bartholomew's Hospital, express my humble confirmation of the correctness of his remarks.

Of the frequency of the connection between apoplexy and valvular disease of the heart there can be no doubt: it has been made matter of arithmetical demonstration. But it is surprising how what is now so clear should have so long remained unknown, or have been even denied; and the probable reason of this circumstance, as involving a great point of pathology, is worth investigation.

Lallemand† says that in no cases of apoplexy which he has read or observed has he ever found contraction of the aortic orifice, which yet he considers to be the most common cause of hypertrophy of the heart. For that, under these circumstances, the increased force of the heart is lost in overcoming the resistance occasioned by the contraction, and does not affect the brain. Hence he infers that it is only in cases where the obstruction causing the hypertrophy does not lie between the left ventricle and the carotids that sanguineous apoplexy can ensue; and he adds that it is not usually in the apoplectic cases of heart disease that we observe the livid lips and cheeks, or the œdema, which point to obstruction of the circulation in the veins.

* Traité de la Structure du Cœur, &c., tome ii. p. 328-331.

* On the Cerebral Circulation, &c.

† Recherches Anat.-path. sur l'Encéphale, tome i. p. 44, note.

As far as the above statement goes, I believe it is literally correct. Real sanguineous apoplexy is very rare under the circumstances; but the symptoms of apoplexy—sudden coma and hemiplegia, for instance—are not quite so rare in connection with advanced valvular disease of the heart as might be supposed from a less literal interpretation of Lallemand's statement? What, then, is the nature of the changes on which the symptoms depend?

There appears, from all that I have been able to observe or to read of the observations of others, no reason to question the accuracy of the conclusion which Dr. Burrows has expressed, that "hypertrophy of the left ventricle must be admitted as a powerful predisposing, or even exciting, cause to apoplexy and sudden hemiplegia."* But where the hypertrophy is not more than sufficient, from whatever cause, to make good the valvular imperfection, we should be wrong in expecting *commonly* to find the results of increased arterial pressure. And sanguineous apoplexy, as already observed, is rare under such circumstances: from whence we may also infer that venous congestion is not one of its common causes.

The evidence of the older writers, as far as I have been able to consult them or the conclusions from them, is negative on this point: but at least it may be inferred from their silence that they did not connect sanguineous apoplexy with advanced and obvious disease of the heart. An analysis, however, of the cases detailed by Andral† and Bouillaud,‡ the most available, for the present purpose, of those invaluable masses of detailed observations in which the French medical literature is so much richer than our own, give a very striking result, which goes far to explain the cause of the discrepancy between former and more recent observation as to the connection between the symptoms of apoplexy and those of valvular disease of the heart.

From these two writers we may collect twenty-eight observations of cerebral disease of limited extent accompanying disease of the heart. Fourteen of these cases had softening of, and fourteen had sanguineous effusion into, the substance of the brain. This different nature of the changes may be observed to present a close correspondence with the varying amount of the symptoms of the valvular lesion. Of the exact nature of the valvular lesion itself, however, the details do not always allow me to speak. The cases may be thus arranged:—

Sanguineous Apoplexy.

Andral 11 cases, age 57·5.	10* had no general symptoms of valvular disease, the heart being more or less hypertrophied.
	1 had anasarca.
Bouillaud 3 cases, age 54·6	1 no general symptoms, 1 anasarca. [&c.
	1 pulmonary complication.

Cerebral Softening.

Andral 8 cases, age 59·2.	4 no general symptoms.
	4 anasarca, dyspnœa, &c.
Bouillaud 6 cases, age 32·3.	All had more or less general symptoms of valvular disease.

The general symptoms of valvular disease were therefore

	Apoplexy.	Softening.
Present in . . .	3	10
Absent	11	4
	14	14

I fear to weaken the force of the conclusions of this table by any comments, or by any verbal expression of what the figures so clearly convey.†

My own experience on this subject,

* Op. cit. p. 121.

† Clin. Méd. tome v. 4me éd., illustrating sanguineous apoplexy and softening of the brain generally.

‡ Des Maladies du Cœur, tome ii. 2me éd. All the cases of valvular disease in which cerebral disease referable to either of these two heads occurred.

* One of these ten cases had œdema at the time of death, which followed two years after the attack of apoplexy.

† Dr. Law (Dublin Quarterly Journal, vol. xvii. p. 181) has called attention to the importance of the distinction, in cases of paralysis accompanying disease of the heart, between the effects of increased and those of diminished arterial pressure on the brain. He refers sanguineous apoplexy to one, and softening to the other cause. He attributes the first of these to the action of an hypertrophied, the other to that of a debilitated or inefficient heart.

Dr. Bright (Med Reports, vol. ii. p. 195), again, regarding the subject from another point of view, has taken a case of extreme disease of the heart as furnishing an illustration of that form of softening of the brain where, "from obstructed circulation, the part undergoes a change analogous to gangrene—the more genuine form of the disease" (Case lxxxi). There appears good reason, however, for admitting the existence of another cause of this particular change. It would seem, in some cases, to be more properly referable to that form of disease known as capillary phlebitis in other organs, than to gangrene.

But in speaking of the conditions of the heart which co-exist, by preference, with one or other of these forms of disease, there is some danger of overlooking a most important change of the vessels which is connected equally with both apoplexy and softening—namely, an atheromatous state of the arteries of the brain. There is no doubt of the close connection of this condition alike with disease of the valves of the heart, with sanguineous apoplexy, and with softening of the brain. See Watson, Lectures, i. p. 516; Abercrombie, Dis. Brain, p. 241, referring to Scarpa; Paget, Med. Gaz. 1850, on Fatty Degeneration of the Small Vessels of the Brain.

though very limited, is quite in accordance with the above. Of four cases of dissection after death from sanguineous apoplexy, with disease of the valves of the heart, the valvular affection was small in degree and simple in kind, and the heart had met the imperfection by hypertrophy of its muscular walls.

Of four dissections after death from extreme disease of the heart, with cerebral symptoms—namely, hemiplegia—in two there was softening of the brain, and in two no explanation at all was found of the paralysis, which, however, it should be noticed, happened in one of these eight months before death, and had been recovered from.

I believe that, without analysing other series of cases, I might confidently appeal to each one's experience for a confirmation of the statement, that it is in comparatively early cases, where the general symptoms of heart disease are scarcely developed, that sanguineous apoplexy most commonly occurs; not in those patients whom a cold winter sends into our hospitals, loaded with dropsical accumulations, and with venous blood,—cold, livid, and struggling for breath. These are rather the subjects of softening of the brain, or of serous effusion, than of sanguineous apoplexy.

If this be correct, it is easy to see how the connection between valvular disease of the heart and apoplexy may have been overlooked at a time when neither auscultation nor morbid anatomy lent such aid to pathology as at present in the recognition of the physical signs and morbid changes, respectively, of valvular disease. And it is, moreover, easy to see how, when this connection was asserted, it should have been denied, on the unjustifiable grounds, that, if the two diseases were really connected, the greater the disease of the heart the more frequent should be the occurrence of apoplexy. But, indeed, the question was not capable of a true solution on such grounds; and a more thorough examination of the correct premises has returned a different answer.

There are other affections of the brain depending on valvular disease besides those which leave organic traces in the forms of cerebral softening and apoplexy. In some of these venous congestion plays a considerable part, while others seem referable to increased arterial pressure. Dr. Latham* has described two of these. He speaks of a state of things where "the heart, by the simple vehemence of its action, has the power to kill, and to kill through the medium of the brain." There is intense headache, sleeplessness, delirium, and death by exhaustion. This is the effect of increased arterial pressure, which we must carefully discriminate by the history of the case, and the character of the pulse, may be, (for auscultation will help us but little) from the symptoms of simple exhaustion: for the alternative of life or death may depend on the correctness of our diagnosis.

Another form of disease presents the symptoms of apoplectic coma, suddenly supervening, and when it passes away leaving no paralysis behind. Here "neither serum nor blood has been let loose upon the brain. The whole mischief is effected by the blood still within its proper vessels, by its congestion, retardation, or remora." In this case "the disease of the heart consists of passive dilatation."

Then there are all the symptoms, if not actually attaining to either of these conditions, at least of sufficient importance to demand especial notice,—such as headache, vertigo, drowsiness, mental anxiety, and the spontaneous relief of these—epistaxis. There is epilepsy, always terrible, and not least so when connected with such a hopeless cause as valvular disease of the heart. And there is syncope—a symptom closely connected with advanced disease of this organ, and not uncommonly the mode of its fatal termination. To this, under the head of failure of the heart's action, there will be occasion to recur.

There is little to be added to what Dr. Burrows* and others† have told us of the pathology and treatment of these affections. As far as a few words can express a general rule of practice in these cases, it is this:—In all cases of disease of the heart we can scarcely pay too much attention to cerebral symptoms which might seem trivial when viewed in connection with disease of any other organ. Under these particular circumstances, drowsiness, headache, and even mental anxiety, claim a consideration which they do not ordinarily possess; and the rule of letting secondary symptoms alone, unless they are dangerous to life, does not apply, for the contingencies which these point to really are dangerous to life in the highest degree.

But when, from whatever cause, the symptoms to be dreaded—namely, those of apoplexy—have supervened, the rule must in some sort be reversed. Now the care must be, not to over-treat the organic disease, as it was before not to under-treat the threatening symptoms. For, not to do more than mention the danger arising from the excessive reaction of an hypertrophied heart on the injured brain, it must be remem-

* On Diseases of the Heart, vol. i. p. 326; ii. p. 336.

* Op. cit.

† See especially Corvisart, Des Maladies du Cœur, p. 141, 3me éd.

bered that the lesion may be either softening or sanguineous effusion. And without expressing any definite opinion as to the essential nature of softening, it approaches too near to that of gangrene* for us to venture rashly on reducing the already weakened constitutional powers by over-active treatment.

II. *Pulmonary Complications.*—The pulmonary affections dependent on valvular disease of the heart may be either primarily referable to venous obstruction, or they may be contingent thereupon. Of this latter class are pneumonia and bronchitis, pleural effusion, or pulmonary œdema; and to these must be added, though its practical importance could scarcely demand even this passing notice, pulmonary apoplexy.

It is not so easy to assign an exact place to pulmonary emphysema† among this class of complications of valvular disease. Probably, in the majority of these cases, it is only secondary to the pulmonary obstruction, chronically and generally resulting from causes similar to those which induce its development in an acute form in the uninflamed parts of the lungs of children suffering from pneumonia.‡ We habitually connect the idea of common asthma with pulmonary emphysema, with which so often, and so often with which alone, it coincides. But in the more complex case of cardiac asthma, I think we need to be reminded of the separate value of emphysema as a possible element of the mixed results which we are called upon to treat under this name. I would not anticipate on this subject what falls under the head of the treatment of abiding pulmonary obstruction. I would here only point to the existence of pulmonary emphysema, as explaining, in some cases, much of the dyspnœa attendant on valvular disease, and in that exact proportion, it must be added, limiting our expectations of the curative effects of our remedies.

With regard to the other complications separately; and first to pleural effusion. Of this there is little to be said here, for the knowledge of its existence in nowise affects the treatment or the prognosis of the cases in which it occurs. Doubtless, it is a very serious complication, but my observation has not led me to think worse of the cases in which it is found* than of others, where large effusions exist in the areolar tissue or serous cavities. Nor has observation shown me any more particularly successful method of removing it.

Pneumonia arising under these circumstances has more claims to distinct notice. When occurring at an early period of valvular disease, it is, as far as I have seen, very amenable to the ordinary medical treatment, but singularly liable to recur after its removal. More commonly, however, it occurs at a later period, under conditions which forbid any treatment especially directed for its removal. The very unfavourable nature of its prognosis under such circumstances is explicable, partly on general grounds, and partly by the tendency which it then displays to run into the third stage. More than all the other pulmonary complications of valvular disease, pneumonia is to be considered in the light of an accident,—but as an accident of the most serious nature, always more and more liable to recur, and always more and more dangerous on each recurrence.

As to the other forms of pulmonary secondary complication—namely, bronchitis and pulmonary œdema—their consideration cannot well be separated from that of their immediate cause—their primary form, pulmonary obstruction: for, practically, they are little more than aggravations of this habitual condition, whether we regard their symptoms or their several characteristic morbid changes.

Without underrating all the means and appliances for arriving at, and availing ourselves of, a more accurate diagnosis, I believe that I should be vaunting a refinement which can find no place in practice if I spoke of anything more definite on this subject in connection with valvular disease of the heart than pulmonary obstruction. Nosological distinctions almost entirely fail us here, as after death by fever. They cannot be accurately maintained in the gorged, œdematous, and obstructed lung, by dissection; and they certainly do not afford us the safest grounds for treatment. The general principles of treatment, and what each of these affections has in common, claim attention rather than the specific differences which secondary causes may have developed in the individual case.

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* See Abercrombie, Dis. Brain, p. 22, 3d ed.

† I am indebted to Dr. Hawkins for recalling my attention to the frequency of the coincidence of valvular disease with this condition, which I had otherwise omitted to notice in this place.

‡ See Dr. West's lectures on the Diseases of Children, p. 178. I need scarcely guard myself against being supposed to adopt generally this mechanical (so to call it) explanation of emphysema. It must be regarded, indeed, from such a point of view in the present case: but I would refer to Dr. Budd's paper on this subject (Med. Chir. Trans. vol. xxiii. p. 37), and to the authorities there quoted, for a more comprehensive survey of this interesting pathological question than the present purpose requires, or my limits permit.

* These remarks apply to the chronic cases. Probably each one, from his own experience, could supply instances where patients suffering from acute inflammatory disease of the heart have been carried off in a day, or less, by sudden pleural effusion.

Pulmonary obstruction, then, to adopt this most general term, may be considered in two points of view,—as a temporary or as an abiding condition. Its symptoms as a temporary condition, induced suddenly by violent exertion, or other passing cause, are familiar to us all. It is an accident to which all are more or less liable; and art can do little more, whatever the cause, than aid in maintaining that perfect rest which nature does her best to enforce. But when pulmonary obstruction, though it be temporary only, is induced by the ordinary exertions of life, itself becomes a matter of serious consideration, and the detection of its cause of the highest importance.

On the present occasion we have only one cause to deal with,—namely, valvular disease of the heart. Under these circumstances, it is but a short stage from these temporary attacks to that affection known as winter cough, when the obstruction is present for a considerable portion of the year. And another still shorter step brings us to where this has become the abiding state of things, on which attacks of bronchitis or pneumonia are engrafted from time to time, and under one or other of which life at last terminates.

Still, however short these stages may be, in the treatment of such a case, apart from all other considerations, the question of the temporary or abiding nature of the pulmonary obstruction is of importance. For, if it be only temporary we may venture on more active measures, trusting to the reparatory powers of the constitution during the interval before the next attack, than we could do if the diseased condition were permanent.

The mode of relief which nature adopts in cases of pulmonary obstruction, that, namely, by local extravasation of blood inducing hæmoptysis, is the most direct. But there are many objections to allowing the congestion thus to relieve itself. For the existence of blood and serum in the bronchi tends largely to aggravate the obstruction to respiration. And its removal necessitates frequent violent exertion in coughing. For the serous effusion, to bring relief, must be very abundant, and the expectoration frequent and copious. And though serous excreation may be easy enough, yet blood so effused does not usually come up quietly, being loose, as we say; but it is tenacious, being bound up with the glairy mucus which results as a secretion from that conditionof parts which induces the hæmorrhage. The relief obtained by abstracting blood, less directly than from the congested membrane itself, may not be so great, but it is incomparably safer, and cupping or leeches to the chest will generally effect the object in view of relieving present pulmonary obstruction. This, however valuable, is yet a mode of relief which we cannot indiscriminately adopt; its fittest application is to the cases where at present the affection is only temporary.

With regard to abiding pulmonary obstruction in connection with valvular disease. Such a case is hopeless from the beginning; all that can be done is to palliate present suffering, or to remove present danger. Obviously, therefore, nothing but the most absolute necessity should induce us to do anything which might at all tax the constitutional powers of the patient. If the vessels are overloaded they may be relieved by cautious abstraction of blood. If the bronchi are obstructed with glairy mucus, or with abundant secretion, they, too, may be indirectly relieved by expectorants and diuretics, either to facilitate the removal of the viscid mucus, or to draw the watery discharge from the blood to another quarter. Or, if present danger to life threatens, we must of course, without regard to more remote dangers, relieve the suffering organ by whatever means, and at whatever cost to the constitution. But if rest and warmth alone will enable the lungs to bear the burden of the circulation, these simple means should be trusted to; for a diarrhœa or a diuresis, the next available means, though powerful instruments for good, cannot be maintained without great exhaustion. They are instruments, moreover, which act with most effect on their first application. It is of the utmost importance to know that such a patient is most readily acted on through his bowels, and such a one through his kidneys, and by such particular remedies: but this knowledge need not be always put in practice. It is only for what warmth and rest will not, or do not appear likely to do, that abstraction, of blood, diuretics, purgatives or expectorants, should be employed under such circumstances.

But though we are sorely straightened in our use of means of relief, by the knowledge that the pulmonary obstruction is an abiding condition, this very circumstance, the habitual presence of the malady, enables us to employ a remedy which under other circumstances would not be available. In such cases we may safely employ opium in the face of symptoms which would otherwise contra-indicate its use, and by its means procure a night's rest with present safety and ulterior benefit. And it is no little thing to say that opium is not quite a forbidden remedy in cases of abiding pulmonary obstruction dependent on valvular disease.

How different is the pathology and treatment of the pulmonary and cerebral

complications respectively of valvular disease! The brain, as we have seen, may suffer from increased or diminished arterial pressure, or from venous obstruction, and of these the first two are of infinitely the greatest importance. The lungs, on the contrary, suffer, in a large majority of cases, under the like circumstances, from venous obstruction. The cerebral complication is in truth rightly so called, for its occurrence tends only to augment the amount of disease. But in the pulmonary affection we may often recognise a spontaneous attempt at the relief of the labouring heart. Could it well be otherwise than that the treatment of the two should present an equally striking contrast with their pathology? It is so. In the one we do all, even by excessively active treatment, to prevent; for we can do but little to cure, even if the immediate danger be escaped. In the other, too, we may try to prevent; but so far from leaving the actual lesion entirely to nature, observation shows that the best mode of reaching the heart is to direct our treatment to it through the lungs, irrespective of the degree of prominence of the pulmonary symptoms.

It might almost seem, from what has been said thus far of the cerebral and pulmonary complications of valvular disease, that the treatment resolved itself into a simple question, of how much depletion the patient could bear? Not so. But at least depletion is the most important and most available means in such cases, and my limits scarcely allow me to touch on any besides these. For the other complications our remedies are more numerous, and, as usual, under such circumstances, of less certain application.

Abdominal complications.—It is only from their position in the same cavity of the body, not from any thing else which they have in common, that the secondary affections of the abdominal viscera are here classed together. For valvular disease of the heart acts injuriously in a very different way on the solid, to what it does on the hollow viscera of this cavity.

Of the importance of the structural diseasesof the liver and kidneys, which coincide with valvular disease of the heart, there can be no doubt, were it only for the share they have in inducing dropsy under such circumstances. But in many cases these changes should be placed side by side with the cardiac disease, rather as parallel effects of some common cause,* than as resulting, themselves, from the obstruction to the circulation. Such, I believe, is their true pathology in most, and these the most important, instances. But it is scarcely the place to discuss the principles of their pathology here. Its explanation is a part of that great problem of the effects of chronic inflammation, and degeneration of the products of previous disease, on organic tissues, which is now being worked out by so many independent observers. We scarcely appreciate the importance of the results already obtained, from their having so insensibly grown upon us, and incorporated themselves with all our previous pathological knowledge, which they at once illustrate and advance. The pathology of these structural changes forms part of a great subject, of which valvular disease of the heart is itself but a branch. But it would exceed alike my limits and my purpose to discuss them here. I would restrict myself to those few remarks which the secondary affections of the stomach and intestines, and the functional lesions of the solid viscera, seem to require.

Constant vomiting and pain in the epigastrium are not unfrequently met with.* But for the uncertainty which hangs over the interpretation of the morbid appearances of the gastric mucous membrane,† one would feel inclined to connect their symptoms with the intense congestion which this membrane often displays in fatal cases of valvular disease.‡ The remedies which have appeared to me most de-

* It is satisfactory on such a point to be able to cite Dr. Latham's authority (Diseases of the Heart, vol. ii. p. 307). "If I except those cases, in which the damage done to the heart could be clearly traced back to some distinct attack of accidental disease, such as rheumatic inflammation, my records of dissection do not supply me with a single instance of a person reputed to die of disorganized heart and its consequences, in whom after death other parts also were not found disorganized, such as the liver, the kidneys, serous and mucous membranes, and above all, and more frequently than all the rest, the whole arterial system. And the kind of disease in other parts has been such as could have in no wise been derived from the heart; but it must have grown out of special morbid processes within themselves, whether prior or subsequent to, or simultaneous with, the disease of the heart."

* Testa (Dei Mal. del Cuore, ii. p. 197, 2nda ed.) speaks of an uncomfortable sensation in the stomack only allayed by taking food, an habitual intolerance of an empty condition of the stomach, as an occasional symptom of cardiac disease. In some cases, indeed, this symptom was so prominent, as to mislead the medical attendants into the belief that the primary disease was in the stomach, and not in the heart. The opposite form of gastric affection, where everything is rejected from the stomach, is, however, much more commonly met with.

† This very interesting subject may be pursued at greater length, in two papers by Dr. Yelloly, Med. Chir. Trans. vol. iv. and vol. xx.

‡ Dr. Todd mentions a case of hæmatemesis, in connection with rupture of the chordæ tendineæ of the tricuspid valve, where he was disposed to connect the hæmorrhage with the patent condition of the tricuspid orifice (Dublin Journal, vol. xxxiii. p. 1).

serving of confidence in the treatment of the epigastric pain are dry cupping, and especially blisters. In attempting to allay the vomiting dependent on valvular disease, I have not observed any particular medicaments to possess specific properties in this respect, apart from the general indications on which they have been prescribed. Only once I saw the alkalies, empirically administered for the relief of obstinate vomiting dependent on such a cause, produce much more good than any peculiarity of the case could have led me to anticipate.

Diarrhœa is not a common accompaniment of heart disease; on the contrary, constipation is much more frequently met with. But it has much interest in connection with advanced valvular disease, as being at times the cause of sudden death, under these circumstances, through exhaustion and syncope.* This is particularly the case in children,—as far, at least, as induction from a limited number of cases, and a general impression from a more extended observation, would allow me to infer. The fact, however, whether observed in children or adults, is explicable on the principle previously adduced, that life maintained under difficulties is destroyed at last by the most trivial causes. We find parallel illustrations in the trivial causes which may suddenly destroy those whose respiration has been long impeded by some tumor or laryngeal affection. And a more painful illustration is found in the rapidly fatal effects on the aged inmates of workhouses, of a slight fall of the external temperature.†

With regard to the treatment of the diarrhœa there is nothing to say, for there is nothing peculiar in it, except this one possible contingency of syncope. Forewarned, fore-armed. We cannot always foresee and prevent diarrhœa, but we can generally check it. If we know when to expect syncope, on the contrary, we can commonly obviate it; but when it has supervened, in cases of advanced valvular disease of the heart, all treatment often comes too late to save life.

It would seem that venous congestion of organs, as involving imperfect arterialization of the blood supplied to them, is in so far an unfavourable condition for the growth of fibrinous tissues. Valvular obstruction, therefore, as far as its effects in inducing venous congestion can be separated from any general pathological conditions (as already indicated) on which itself may depend, tends to produce but slight changes in the structure of organs. But on their secretions its effects are more positive, albeit seemingly at times inconsistent, or at least inexplicable. For it does not appear why, in connection with valvular imperfection, sometimes the watery and sometimes the solid constituents of the secretions should be in excess.

But, by the side of affections causing distress and danger to life, all minor derangements sink into nothing. We recognise, indeed, in valvular imperfection of the heart, a sufficient cause for jaundice, in all its degrees, from mere biliary congestion, to the well-marked form of the disease. Again, the explanation of albuminuria under the same circumstances is obvious. But these are scarcely in themselves, under these circumstances, objects of treatment. The great interest, on the present occasion, of the subject of venous congestion of these organs, turns on its connection with cardiac dropsy, rather than with these minor functional derangements.

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* Contrast with the well-known disproportionately exhausting effects of diarrhœa, the equally well-known fact that the system will bear with safety a much larger amount of hæmorrhage from the hollow abdominal viscera than from any other source, except, perhaps, the recently emptied uterus. Yet the contradiction is only apparent. A large secretion may be, temporarily, as exhausting as a large hæmorrhage.

† Liebig, Chemistry of Pathology, &c., p. 255. "We see, in hospitals and charitable institutions (in Brussels, for example) in which old people spend the last years of life, when the temperature of the dormitory, in winter, sinks two or three degrees below the usual point, that by this slight degree of cooling the death of the oldest and weakest, males as well as females, is brought about. They are found lying tranquilly in bed, without the slightest symptoms of disease, or of the usual recognizable causes of death."

LECTURE III.—Part II.

IV. Symptoms and treatment of valvular disease of the heart (continued).

Cardiac dropsy: effusion from inflammation, from venous obstruction—Principles of treatment—Influence of rest on the heart's action—Use of purgatives, diuretics. Complications of dropsy, mortification, erysipelas; may occur without dropsy.

Effects of valvular disease on the action of the heart—Palpitation—Failure of the heart—Effects of a shock on the healthy, on the diseased organ.

Modes of death from valvular disease.

IV. *Cardiac dropsy.*—Under this name I would class only the effusions of fluid which occur in the serous cavities, or the general areolar tissue, in connection with valvular disease; though the distinction which would exclude œdema of organs, and notably serous apoplexy of the brain, from this category, is rather convenient than pathologically accurate. On the same plea of convenience, however, we may group with general dropsy the various gangrenous or erysipelatous inflammations of the integuments, which commonly arise from it, and last, though not least, complete the sad catalogue of the secondary consequences of valvular disease of the heart. On what causes does this class of secondary affections depend, and what can we do for their relief?

The history of patients suffering from dropsical effusions in connection with valvular disease commonly speaks of exposure to cold, or distress, or of unusual exertion. Instead of laying any great stress on the statements of patients for the most part very incompetent to observe their own symptoms, we may simply refer these, in the present instance, to the two heads of inflammation and venous obstruction respectively.

The effects of inflammation under these circumstances are shown, perhaps, most commonly by effusion into the general areolar tissue, rather than into the serous cavities. On neither of these, however, is it necessary to dwell here; for the facts and appearances, in the first class of cases, will of themselves explain the relation which the inflammation bears to the exudation—namely, whether it be cause or effect. And as to the second, the comparative rarity of the occurrence, under these circumstances, of inflammatory effusion into the serous cavities, to any large amount, forbids any lengthened notice of this form of disease. But the possibility of the last occurrence should at least be borne in mind; for this may sometimes throw a more favourable light on particular cases, allowing us to interpret their more distressing symptoms of pain and dyspnœa, not as the physical effect of an irremediable organic disease, but as signs of a present action which it is in our power to control by careful antiphlogistic treatment. Such cases, however, are exceptions to the general rule; effusion, in the majority of these cases, depending on obstruction rather than on inflammation.*

It might seem, at first sight, that with the statement that the remaining cases of cardiac dropsy were dependent on mechanical causes, and these, so far as concerns us at present, essentially irremediable, the subject was exhausted. Not so. It does not, indeed, admit of a systematic examination of the details within the present limits: and as to the general principles, we have already considered the influence of one or other side of the heart in the production of cardiac dropsy. But, taking up the subject a little further on, and speaking of the removal of the dropsy, rather than of its accession, as being the rather a matter of clinical inquiry, here is a field in which each observation of less absolute importance may find its proper place. The general principles of treatment of this affection,—nor of this affection only, but applicable alike to all the other forms of secondary complications of valvular disease,—will well repay attention. For, indeed, they are general principles which are required here. There are fewer lucky hits made by guess-work in cardiac than in other dropsy.

The first thing to be done is to set the patient, and so his heart, at rest,—the most simple, and yet the most effectual means in our power, for his relief. The influence of

* With regard to the chemistry of this subject, it is hardly too much to say that it is almost wholly unknown. I am not aware of any other source of exact information than Andral's sixteen cases analysed in his Essai d'Hématologie pathologique, Bruxelles, p. 136-8. Yet dropsical effusion is a subject in which we have a right to look for information from organic chemistry.

Vogel (Path. Anat. by Dr. Day, p. 37-43) gives references to different authors who have treated on this subject. I have not been able to refer to these authors, but, judging from his own expressions, the chemical part—the relation, namely, of the effused fluid to the serum of the blood—remains as obscure as ever.

simple rest may be beneficial in several ways. First, as the strength of the heart is accurately adjusted to the mean requirements of a day's work, by rest so much is directly saved; of exertion to the heart, if it be sound; of suffering, if the valves be imperfect, to the organs against which, under such circumstances, the pressure is made: and this, whatever be the mode of the heart's action. But the functions of the heart are more perfectly executed when the heart's action is quiet, than when it is morbidly excited: and the action of a diseased heart is, as a rule, more liable to be so excited, and from a less cause, than is that of a healthy heart. It follows that the effects of rest will be in such case, in that exact proportion, the more salutary.

The closure of the sigmoid valves is a simple matter enough; but for the perfect coaptation of the auriculo-ventricular valves during the whole period of the systole, much precision in the action of all the parts concerned is required. There can be no doubt what the effect of an imperfect coaptation would be; and I believe as little that such a condition would be induced by a very slight change in the relative degree and time of contraction of the different fibres of the heart's muscular walls.* That is to say, a very slight alteration of the rhythm of the heart's action in detail is quite a sufficient, and most probably the actual cause, of functional imperfection of the valves in many cases. The most effectual means in our power for correcting this alteration, and the consequent regurgitation, is rest.

We cannot, obviously, look to auscultation for absolute demonstration of the correctness of this explanation in the cases with which we have here to do. Neither can auscultation alone determine the degree, even, in which the heart's imperfections have been remedied, if so it should happen, by such means: for probably the murmur dependent on the organic lesion would become only the more audible when the rhythm of the heart was restored, and the superadded effects of this functional lesion were removed. We are to look for it in the quiet, steady action which has replaced the tumultuous palpitations, and in the patient's altered feelings. If we cannot hear it from his heart, we may often hear from his lips how much present ease he owes to the calming the action of his heart, to simple rest. Perfect rest is almost a necessary preliminary to our successful treatment of cardiac dropsy. It is at times all that is necessary, and supersedes all other treatment.

It might provoke a smile to refer to a period, not so many years past, when the expression *morbus cordis* shed a seeming of knowledge over a class of symptoms presumed to depend on disease of the heart, —to be, as such, essentially incurable, and therefore not to be likely to repay the trouble of a minute examination. And it would be superfluous to repeat here how these, the most unattractive cases, have become the most interesting, and how much has been learned from their study by which to discriminate the disease at an earlier period, and to prevent many of its consequences. There still remains much for us to do in palliating the effects of what we have not prevented, and which we cannot cure.

Speaking generally, as far as my own observation on this subject goes, greater and more permanent relief has been obtained from the use of purgatives than from any other means in cases of cardiac dropsy; and of these, the saline purgatives with senna, given every morning, or every other morning, as the patient could bear them, have seemed more useful than the occasional employment of more active means, such as elaterium. But this mode of treatment is rather adapted to those cases where the powers of the constitution are as yet little affected. It does not succeed so well, and is accompanied by considerable risk, in the more advanced cases. Here diuretics are a safer, though a less certain means of relief:* and of these, my

* Dr. Hope (op. cit. p. 392), quotes instances where the functional integrity of the heart was restored by simply loosening the tight dress which had impeded its action; though he adopts a different explanation of the production of the murmur to that in the text, and which the circumstances seem more naturally to suggest. Excluding the fallacies from coincident endocarditis, the mitral murmurs so often heard in chorea seem to me to fall under the same category of the results of an abnormal mode of contraction of the heart. And further illustrations will readily suggest themselves.

The disordered rhythm of the heart's contraction here spoken of must be distinguished from that irregular action which we may often find in patients who have, perhaps, no other symptoms of disease of the heart; and particularly in the subjects of pulmonary emphysema, irrespective, however, of the amount of this change. Perplexing as such a tumbling irregular heart is to the auscultator, the rhythm of its contraction, judged by the symptoms, has been disturbed in a much less serious manner than in the form of disease under consideration.

* Dr. Hope's experience led him to a somewhat different conclusion on this matter (Diseases of the Heart, p. 409). He puts diuretics in the first place, as being the remedies deserving the first trial; and speaks of purgatives only as the second alternative. Of these, too, he mentions elaterium with greater commendation than my own observations, in St. Bartholomew's Hospital or elsewhere, would seem to me to justify. Not that, as Dr. Hope remarks, the effects of elaterium are not at times truly asto-

own observation would induce me to prefer the nitrate of potash, assisted in the lesser details by what the circumstances of the case might suggest. Such are warm stimulant diuretics or purgatives exactly apportioned to the powers, and, what is equally important, to the habits of the patient. These supplementary matters are not beneath consideration; nothing which alleviates suffering can properly be so. A knowledge of, and a due regard to, the habits which have had a large share in inducing disease, may sometimes assist us in our attempts at its removal. Dropsy, with diseased heart and obstructed liver and kidneys, which has defied all the regular appliances of pharmacy, may yield to —gin.

Under such a very cautious plan of treatment, sometimes the present symptoms have been relieved, the effusion removed, and the patient, to all but the physician, has seemed to be quite well. Sometimes our rational attempts may be aided by a happy idiosyncrasy on the part of the patient. Perhaps one particular diuretic, such as the acetate of potash, may act again and again surprisingly well; and sometimes digitalis will succeed beyond all expectation; and this, I think, wholly irrespective of the particular preparation employed.* Iron, again, is a remedy which no one would neglect to employ in all cases where debility of the system at large was known to exist. But whatever may be the good obtained from iron in these cases, it is not in cardiac, as in renal dropsy, that iron does wonders.†

With whatsoever success this complication be treated, yet, after all, it is but a complication, and, considered abstractly and in itself, one of but secondary importance. For it is not ordinarily by effusion into the general areolar tissue or the serous cavities, by sheer exhaustion, or by obstruction to the functions of different organs, that cardiac dropsy destroys life. Much as the serous effusion may have to do with inducing the fatal termination, it is not commonly the immediate cause of that event, especially when occurring in the form of anasarca. Extensive œdema cannot long exist under these circumstances without giving rise to excoriations, whose results tend more directly, by pain and loss of sleep, and by local action, to destroy life.* It remains to consider these, the associate complications of cardiac dropsy.

Mortification,† as a consequence of valvular disease of the heart, most commonly ensues upon the erysipelatous inflammation to which anasarca gives rise, and is limited to the integuments and the parts immediately subjacent. The ordinary course of events, under such circumstances, is but too familiar to daily experience. I would therefore pass over these, and dwell rather on some forms of disease which fall less frequently under observation.

In some cases of valvular disease of the heart, the mortification, which usually begins in, or is limited to, the integuments, implicates the deeper structures. My own observation furnishes a single instance of this very unusual occurrence, where the right leg mortified in its entire extent, though there had been neither anasarca nor evident inflammation precedently. Unfortunately, no examination of the body after death was permitted. The details of the case, therefore, possess no such particular interest as to need to be recalled here at length. Briefly, it was a case of dry gangrene, occurring in a woman forty-three years of age, who had been suffering for some time from a first (?) attack of subacute rheumatism, and had, on her admission, pericarditis and considerable aortic sigmoid obstruction. The gangrene was accompanied by intense pain in the limb, with insensibility to external impressions. Death happened at the end of five weeks,

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nishing; but that the employment of this remedy in cases where there is room for such effects to be produced, is commonly not without danger.

Probably the correct principle of practice is that inculcated by Dr. Williams (London Journal, 1850, p. 467)—that, namely, of restoring *all* the secretions to their normal condition before we determine the plan of our further proceedings, or select the secretion by which, in particular, we should endeavour to remove the effused fluid.

* But see on this subject Dr. Munk's paper, Guy's Hospital Reports, vol. xi. p. 295.

† Of cases of recovery against hope—of instances of the good effects of steady persistence in a fixed plan of treatment—the most striking which have come under my own observation have been renal dropsies treated by preparations of iron.

* There is another respect in which cardiac differs from renal dropsy. In both alike, excoriations and sloughing of the integuments are of most unfavourable augury, and in both alike it is the exception when they are not followed by fatal consequences. Still, in cardiac dropsy they are sometimes recovered from, and the relief afforded by the free outlet thus offered to the dropsical fluid sometimes tends to prolong life. But in renal dropsy, sometimes, from these consequences, the patient seems to date a new period of his existence, and as the slough parts, and the wound suppurates and slowly heals, the fluid is absorbed, the albumen disappears from the urine, the heart ceases to strive, and, judged by the rules by which we determined him to be ill before, the patient seems to be again well. Morbid anatomy, indeed, tells us here that the recovery is only seeming; clinical experience, however, has not such a pleasing semblance even to tell us of as concerns cardiac dropsy.

† See Cyclop. Pract. Med. vol. iii. p. 131; Hope, op. cit. p. 224; and a striking case, Med. Gaz. xv. p. 748; also Dublin Journal, vol. x. N. S. p. 301.

resulting apparently from the exhaustion of diarrhœa.

Mortification must be regarded rather as indicating the amount of incurable mischief done, and as displaying the action of injurious influences, than as itself exerting such influences, in any marked degree, beyond the limits of the organic lesion. The case just narrated might serve to point this conclusion; for there, mortification of half an extremity was only after five weeks, and then only contingently, a cause of death.

Anasarca, inflammation, mortification—this is the regular order of succession. In the last case the two first processes seemed to be absent; in other cases the integuments inflame and slough, not as an effect of the anasarca, but, as it might seem, primarily, from venous obstruction. Under these circumstances the inflammation assumes a much more formidable aspect than when it is consequent on anasarca. The few cases on record which I have met with agree with what my own observation has supplied, in representing this form of disease as most rapidly fatal.

We may express, as a general conclusion, what these consequences of venous obstruction illustrate in but one,—albeit, the most important form. Inflammation of the integuments is more to be dreaded, when, in connection with valvular disease of the heart, it arises from some widely diffused cause, instead of originating in some limited point, as more commonly happens; for so the cause which has presided over its first manifestation prevails generally, and the system, under these circumstances, is ill calculated to withstand its attacks. There would seem to be less cause for present fear in the inflammation which extends from a wound, where, for instance, the serum has made its way through the cuticle, or has been evacuated by acu-puncture. Extreme is the patient's misery from such a cause; yet, as a question of time merely, inflammation commencing in this way, and running on to sloughing, certainly does not bring so much present danger to life as when it occurs under the condition above noticed.

There is no reason to question the correctness of the explanation generally received, which, connecting the diffused gangrene secondarily with the condition of the heart, would refer it more immediately to the ossified state of the arteries, and to their obstruction by coagula: but I have nothing to add to the data on which this opinion is founded.

In all these cases, where active treatment is out of the question, a good deal may be done to relieve distress by fomentations and poultices, and the very cautious application of leeches. The necessity of maintaining particular positions, on account of the dyspnœa, interferes with the use of the water-bed: but the want of this most useful appliance is not so much felt here as might seem at first to be the case; for the sloughs are usually on the sides of the legs, and in the folds of the skin, rather than on the hips and sacrum, and parts exposed to pressure, as in fever. As an encouragement to treat these cases with that care and assiduity which only the hope of success can inspire, a case is recorded by Andral,* where the use of quinine and stimulants, and the external application of bark, was followed, as far as concerned the gangrene, with the most complete success. And that these slight hopes may not rest on routine treatment, but on the fulfilment of the rational indications in each individual, an instance is supplied by Andral of the successful issue of a case of the same disease treated on the most opposite plan. The complication was diffuse inflammation of the integuments; and, under the use of very free depletion, the patient so far did well. Such cases, however, form the exceptions: they are very rare.

Effects of valvular disease on the action of the heart.

We have considered, in a former part of this lecture, the organic effects of valvular disease on the muscular structure of the heart, as lying at the root of almost all the secondary complications above described; and perhaps the changes which the functions of the heart undergo from this cause might, on the same grounds, have claimed a preliminary notice. But these seemed to find their fittest place here, at the close of the sad catalogue of symptoms which the other suffering organs display. For let all these complications of other organs have been avoided, whether by accident or by the patient's happy constitution in these particular respects, whether the disease have been averted or fairly met by the physician, or have never threatened at all, still there is the natural termination of the disease to be apprehended—death through failure of the heart's action.†

And there has already been occasion to notice one or two conditions of disease into which a deranged action of the heart, as distinct from valvular imperfection,

* Clin. Méd. tome iii. p. 84-86.

† Not unfrequently we find advanced valvular disease of the heart in the bodies of patients who have sunk unaccountably from diseases or accidents which may have seemed scarcely severe enough to destroy life. This, however, is not exactly the death from failure of the heart intended in the text. Such an event, moreover, is more frequently explained by coincident renal than by cardiac disease.

seemed to enter. There is still another condition, which, though it has scarcely been named, yet much of what might be said of it under its own name has been already forestalled in the consideration of the diseases with which it coincides. Palpitation, as a disease in itself, apart from hypertrophy and dilatation—the instruments by which it becomes a source of so much distress and danger, and apart from that functional imperfection of the valves by which the symptoms may temporarily be so aggravated, is a subject of great practical importance: yet it has no claims for any lengthened notice here; for it coincides, perhaps, as often with a healthy as with a diseased state of the valves; and it is, *per se*, a sign of an irritable rather than of a labouring heart.

Of its treatment and pathology, where depending on other causes than valvular disease, it is beside the present purpose to speak at length. We must keep in mind that palpitation coinciding with, need not depend on valvular disease, though it may be much aggravated by this means. So far as it is connected with valvular disease and its complications, the treatment, of course, will bear a due regard to the cause; and, under these circumstances, nitrate of potash, or digitalis, with perfect rest, have always appeared to be of most avail. But the distress and alarm which this symptom of palpitation gives rise to are so great, that we must make its relief a special point of treatment, and act on its own indications when we fail in its removal by a due attention to those which the valvular disease suggests.

Does the mind, in very terror at the much dreaded name of disease of the heart, produce this symptom of palpitation? The same upright candour which forbad us to hold out deceitful hopes of cure to the patient will now give him confidence in our assurance of the absence of present danger, and so aid to fulfil our promise of speedy relief. What iron, or tonics, or opium, or belladonna plasters, with a more liberal diet, will do for anæmic palpitation where the heart is sound, they will do here, though the heart be diseased; or, if the irritability be inflammatory rather than anæmic, a few leeches,* or the cautious use of digitalis, or the insertion of a seton, may calm, almost, as it were, by magic, the throbbing heart.†

But this by the way.* We have here to do, not so much with these lesser perversions or derangements of the heart's action, as with its failure; and of this, palpitation gives no certain indications. It is a sign of active life, however feeble,—of resistance to, or irritability under, disease. We are dealing now rather with the signs of coming death.

The failure of the heart is a condition which enters largely into the termination of nearly every protracted case of heart disease, in the ordinary course of events: and Dr. Williams justly calls attention† to its earliest indications, as constituting an important element in the prognosis of the duration of life in any particular case; though, among the various complications of valvular disease, they are very likely to be overlooked.

Again, the gradual failure of the heart's action may be, under very favourable circumstances, the sole indication of coming death, or, as we say, of the breaking up of the constitution: and as it gradually makes itself felt, the patient may sink to death by almost imperceptible decay. But on neither of these forms of the disease have I here anything to remark: for I have nothing to add to what, in one or other shape, is so familiar to daily experience. Rather, in pursuance of the plan which has been followed, as closely as might be, through these lectures, I would limit my remarks to a less common form, where the action of the heart is suddenly deranged, or the patient is struck down in a moment by fatal syncope.

It needs no lengthened exposition here to show that the heart is something more than a hollow muscle,—that it is endowed with something higher than mere mechanical functions. Perhaps, even the attempt to prove it weakens the consciousness which each one has in his own breast of the fact. We have seen how the proper contraction of the heart may be impeded, and how mechanical disease may be secondarily induced, by mental emotions, just as well as by more tangible physical causes. But

* Latham, Diseases of the Heart, vol. ii. p. 255.

† The coincidence between enlargement of the thyroid body and palpitation of the heart has been noticed by numerous writers. Extreme habitual anæmia, nervous excitability, and staring prominent eyeballs, mark very characteristically the subjects of this coincidence. Since Dr. Parry's observations called my attention to the subject (Collections. vol. ii. p. 111.), I have often met with notices of the same coincidence. Neither reading nor observation, however, have yet shown me any particularly successful method of treating this peculiarly violent and intractable form of palpitation.

* It is scarcely necessary to dwell here on what have been so very fully described by systematic writers,—namely, the effects of valvular disease on the pulse. In the present point of view, setting out, as we do, from the knowledge of valvular disease of the heart, the feel of the pulse but repeats the intimations of the stethoscope. The great practical value of the information derived from this source is rather to suggest inquiry than to confirm what we know more surely on other grounds.

† London Journal, vol. ii. p. 312.

thus the cause is not mechanical nor necessarily irremediable. When it has ceased to act, there are no incurable organic effects left behind, at least in the first instance,—no obstacle to the immediate restoration of health,—nor, save a liability to the recurrence of the same functional disturbance, to its permanency. Yet this perverted action may go on to induce organic disease, or even to destroy life.

Even as such, freed from all this obscurity which coexistent symptoms may throw around it, in the mixed or opposing results of different causes, derangement of the heart's action now comes before us as the most prominent, and, indeed, the fatal symptom. Such an event is to be apprehended rather in children than in adults. This arises partly from the greater extent of tolerance of the different organs in children, which allows life, as already observed, to be continued in them under greater difficulties than could be so surmounted in an adult, and partly from the fact that they are much less able to withstand a sudden shock, when it does come, than an adult is. So, in them, valvular disease runs on more often to its natural termination, death through the heart itself. Life continues till the heart comes actually to a standstill. The various organs can do much to relieve the effects of stagnation of the blood, but they alone cannot move it onwards, and death comes at last suddenly, by syncope, when some, perhaps trivial, cause has given to the heart's action a momentary check, from which it cannot recover itself.

Dr. Latham* mentions two striking cases, where, after a sudden shock, caused by fear and anger respectively, all the symptoms of valvular disease set in, and ultimately destroyed life: yet, after death, no lesion of the mechanical apparatus of the heart, sufficient to explain these symptoms, was discovered.

It would seem that, in the moment of passion, the rhythmical action of the fibres of the heart had been suddenly deranged, and had never been restored. Thence ensued valvular imperfection, and thence disease and death, just as if the valvular imperfection had depended entirely on a physical cause. Only, as the patients' hearts were healthy, they could bear the first shock which, where the heart is already diseased, is capable of destroying life.

There is little or nothing to add here to what Dr. Latham has so graphically described under the name of the effects of a shock to the heart. The subject may be pursued at greater length in his Lectures, from which I have, throughout, so largely drawn, and, I feel, so insufficiently acknowledged. I believe that he has expressed and duly arranged all that is known on this matter, throwing the light of modern pathology on the quaint narratives in which the older observers have so well recorded what had moved their curiosity, yet passed their understanding.

It remains to trace upon a heart diseased, and already failing under its burden, the effects of what we see to be so serious an injury to the heart. No general description could display these effects better than the brief details of the following case. For the particulars of the early history I am indebted to Mr. Paget.

A gentleman, twenty years of age, had always enjoyed good health, and had always passed for a singularly healthy and robust man, almost to the moment of his sudden death. He was fond of all athletic excercises—riding, and playing at cricket; only he had a great aversion to bathing, as he said that "the cold water made him feel so tight across the chest." One day, having got up in his usual robust health, he was suddenly taken ill on Holborn Hill,—so suddenly, that he turned into a shop, and begged for a glass of water, and leave to sit down for a while. He rose and said he would walk on, but he looked so pale that the people went to call a cab:—before it could come, he was dead.

On examination of the body, his heart was found to be enormously enlarged; the mitral valve was much contracted, and there was very extensive earthy deposit round its base. The aortic valves were only two in number,—ragged, earthy, and hollowed out like carious teeth.*

Why the heart should have stopped at this particular period is perhaps less a matter for surprise than why it should have gone on for so long without a symptom. For his was not a life of quiet and repose,—he mixed in and enjoyed all the most athletic exercises. The increased obstruction which the valvular disease caused was overcome by the most exactly commensu-

* Op. cit. vol. ii. p. 205-206.

* I am indebted to Dr. Taylor, of Guildford Street, for some particulars of great interest in relation to the congenital origin of the disease in this case.

The son died, æt. 20, as above described.

The mother died three years ago, after forty-eight hours' illness. She had been considered to have dilatation with thinning of the walls of the heart: but no examination of the body was made after death.

The father died, since these Lectures were delivered, æt. 66, of rupture of the heart, after ten hours' illness. His heart was soft and rotten. The aortic valves were thick and opake, but efficient, the coronary arteries pervious. There was no opportunity of examining the heart microscopically.

rate increase of muscular power. Suddenly the heart failed: the huge mass which, by the most accurate adjustment of its various parts, had been doing its work as smoothly, to his own feelings at least, and imperceptibly as in health, and made up for the imperfections of the instrument by its strength, received a sudden shock; and death resulted almost as rapidly as when the pressure of the blood is suddenly removed on the rupture of an aneurism. It is of interest to trace, in the only symptom of his disease which this man had ever displayed, indications of the heart's impatience of any sudden shock. The sudden shock of cold water on the breast, though momentary, was felt much more keenly than the severe sustained exertion of the sports in which he delighted and excelled.

If the occurrence of death, under these circumstances, owned any fixed unvarying laws, it would be better to occupy our time in endeavouring to establish these laws, with however small a measure of success, than in illustrating particular points. But it is not so: we have attained no further on this matter than to know that this or that may happen. We have a succession of pictures rather than one continued view. I would, then, ask your patience, while I bring forward but one more picture of human suffering from this gloomy series, selecting, as before, a case where the usual earlier symptoms had been absent, or at least unnoticed. Not to say that the history of disease of the heart might not have been elicited, by inquiry, either in this or the preceding case, had circumstances allowed; at least, to them the symptoms appeared to date from the stated period. But for the accident, the subject of the following might have lived on till death overtook him as suddenly as it did that of the preceding narrative. For the opportunity of observing this patient, I have much pleasure in acknowledging my obligations to Dr. Jenks, of Brighton.

Thomas Elliott, aged 25, was admitted into the Sussex County Hospital, under the care of Dr. Jenks, September 1849, pale and dusky-looking, suffering intense dyspnœa. He said that he had suffered acute rheumatism on two occasions, thirteen and five years ago, in St. George's Hospital. Before the attacks of rheumatism he had been strong, but since then he had been at times liable to pain in the region of the heart, and to palpitation. However, he thought himself well till ten days before his death, when, walking home in the dark, he tripped over a stone and stumbled, but did not fall. From that moment his symptoms set in: as he said, he stumbled a healthy man, and he rose up such as he came in, after eight days, to die.

On auscultation, the heart was found to beat with very great rapidity and irregularity, but there was no morbid sound. The impulse of the heart was not forcible, and its situation was ill defined. He had abundant small crepitation, with dulness on percussion, over the upper lobe of the left lung.

He was supported by wine, while an attempt was made to relieve his circulation by taking a little blood from over the lungs by cupping: but he died in a few hours from the time that I saw him.

The pericardium was found to be closely adherent by tough old areolar tissue. The right side of the heart was scarcely larger than natural. The left was enormously enlarged, the mitral valve thick and stiff, evidently not efficient, the chordæ tendineæ elongated and much thickened. The aortic valves were rigid, their attachments lengthened, and their edges rolled back.

Dissection fully explained this very obscure case, though it did not favour the interpretation which was put upon the symptoms and history before death. A previous attack of rheumatism, subsequent enjoyment of health suddenly interrupted by a fall, then all the symptoms of extreme valvular disease, with no evidence of enlargement of the heart,—how were these to be explained? Nothing seemed more probable than that an extensive rupture of a valve had taken place: nothing was further from the truth. The physical examination of the heart tended only to confuse the diagnosis, for in his condition no thorough examination could be made, and the evidence obtained was altogether inconclusive or negative. There had been no mechanical injury inflicted on the heart by the slight shock which it had sustained; but this slight shock had inflicted an irreparable injury on the nice adjustment of the action of its several parts. The regularity of its action had been for a moment checked, and could not be restored again; and this irregularity had induced sudden pulmonary congestion, with rapid and extensive effusion into the vesicular structure of the lungs.

It needs but little more to complete the subject of valvular disease of the heart,—but a few words on the last end of our ineffectual pains, and of the sufferings of the patient, on the period when the evident approach of death suspends every other indication, save that of soothing the last moments of the dying man.

It were a sad story to tell in how many various ways death has carried off patients

suffering from disease of the heart, though, in many cases, the history is singularly uniform.

Thus: they have had a slight diarrhœa, they are observed to look pale on returning to bed; with all haste assistance is sent for, but death has already done its work, and neither brandy, nor ammonia, nor ether, can relieve the heart of the burthen which has weighed it down. Perhaps the patient has been sitting up, as being a little more cheerful than usual; perhaps the unusual heat has exhausted him; or perhaps we turn to the cold as the explanation of the sudden fatal syncope. But in truth we need very little more explanation than dissection gives us, for it is to be wondered at how the heart went on so long, and how smoothly everything else must have gone on not to suspend its action long before. It would be yet more to be wondered at, if, after any interruption, it could have righted itself.

For dissection shows us in these cases the most extreme disorganization that might seem to be compatible with any continuance of the functions of the heart and of life. The orifices may be contracted to almost complete closure, the organ hypertrophied and dilated, forcing the lungs out of its way; its walls rigid and fibrous, creaking under the knife; or the pericardium may be agglutinated with the walls into a leathery elastic mass, which we wonder to think how it could have contracted to expel its contents at all.

But the approach to death has often greater terrors than death itself,—so calm, although so sudden, as just described. Indeed, it were a sad story to tell how patients with disease of the heart have died,—the tragedies, so to say, of the medical wards of our large hospitals. How some, wrung with pain, have struggled in the week-long agony of death. How some have, for days together, fixed themselves in the most fantastic postures, the only way in which they could find relief; some leaning forwards,* resting their heads on a stool to catch a few minutes' sleep; some on their hands and knees, till the approach of death, blunting their sensations, allowed them to lie down,—a sure sign of coming dissolution. How some, not one only, in their gloomy despondency,—for

. . . . "we are not ourselves
When nature being oppressed commands the mind
To suffer with the body,"—

have laid violent hands on themselves, and others have shrieked in terror when the agony of death at last came upon the dreams which had cheered them in the thought that their sufferings were but for a while, and in the hope of renewed health and strength. He had done a good service to humanity, who had taught us how to prevent disease of the heart; and he, as society is constituted, yet more, who had taught us how to cure it. But his were no little diminution of the cup of human misery who should teach us but its enthanasia.

* Especially in connection with dilatation of the ascending aorta.

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