CHAPTER XIII.

PERICARDITIS.

BETWEEN the pericardium of the anatomist, and that of the pathologist, there is no difference. Rheumatic pericarditis consists in rheumatic inflammation of the investing membrane of the heart. As already explained, such inflammation is most common in acute cases, and in young subjects.

Of all the serous membranes of the body, the pericardium is the only one which invests an organ having free and active movements. So far as functional activity is concerned, the pericardium is as much above other serous membranes, as the valves of the left side of the heart are above those of the right, and the fibrous tissues and serous linings of the large joints above those of the small. In its liability to rheumatic inflammation the pericardium is in the same position as the serous lining of a large joint.

Pericarditis occurs in different degrees of severity. The whole membrane may be the seat of acute inflammation: or only a small part of it may be affected. According to its extent and severity are the symptoms to which it gives rise. As a rule, subjective symptoms are more marked than in endocarditis.

In acute and severe cases, pain in the region of the heart is generally present at the outset, and is often the first thing complained of. Usually it is increased by pressure over the heart, or in the epigastrium. The patient is restless and distressed; his countenance has an anxious expression; the breathing is quickened; there is generally a short frequent cough; the heart's action is vigorous and rapid—maybe tumultuous, violent, and irregular. On auscultation there is heard the friction sound produced by the rubbing against each other of the roughened pericardial surfaces.

But the heart symptoms are not always so distinct in these acute cases. Occasionally their place is taken by nervous symptoms so marked that the case is apt to be mistaken for one of cerebral rheuma-

tism. The onset of the pericardial inflammation may be ushered in by delirium; and delirium, stupor, and coma, may be its characteristic symptoms throughout. From beginning to end there may not be a single subjective symptom of cardiac disturbance—nothing, in short, but the evidence of cerebral disturbance. The occurrence of head symptoms in acute rheumatism is always a reason for suspecting and carefully examining the heart.

The following case, recorded by Andral,[1] is a good illustration of the manner in which acute pericarditis may simulate inflammatory mischief in the nervous centres:—

"Symptoms of Meningitis. Acute inflammation of the pericardium."

CASE I.—"A woman, aged 26, the mother of two children, and who had recently had a miscarriage, was admitted into la Charité early in the year 1820, in such a state of delirium that no information could be got regarding her antecedents. The delirium was remarkable for the obstinate taciturnity by which it was accompanied. When asked a question, the patient looked fixedly at one without answering; the face was pale; the lips, separated from each other, and agitated from time to time as by a convulsive trembling, allowed the tongue to be seen, moist and white. The pulse was frequent and small, but regular; the skin rather cold. During the next two days there was frequent bending backwards of the head, sudden raising of the trunk at intervals, and *subsultus tendinum*. The patient spoke, and appeared to understand what was said to her, but was quite incoherent in what she said. The face was very pale; the pulse very frequent, and intermittent. On the fourth day after admission the delirium ceased; the patient complained only of great weakness; the muscles of the face were agitated by almost continual convulsive movements; and the upper extremities were affected from time to time with an almost tetanic rigidity. On the fifth day the delirium returned, the features were distorted: during the course of the day the patient became comatose, and died in the evening."

On *post-mortem* examination it was found that "there was no appreciable change in the color or consistence of the brain, spinal cord, or their membranes. The digestive canal, opened throughout its whole

[1] Andral, Clinique Médicale, Tome I., p. 34.

extent, presented only a slight injection here and there. The other abdominal viscera were free from lesion. The lungs were slightly congested posteriorly. The substance of the heart presented no trace of morbid change: the vessels entering and issuing from it, were also healthy. But the pericardium was covered with a lymphy deposit (par des concrétions albumineuses), which stretched at many points like a soft bridle from the visceral to the parietal surface. Effused into its cavity, too, were several ounces of a greenish and flocculent serum."

In subacute pericarditis the subjective symptoms are less marked. There may be no more than a sense of uneasiness about the heart, with some increased rapidity of its action. It is on the auscultatory signs that we depend for a diagnosis.

In some mild cases there are no subjective symptoms whatever—nothing but the objective pericardial rub to point out the existence of the disease.

Some cases of pericarditis there are, too, in which the inflammation is limited to that portion of the membrane which surrounds the great vessels at the base of the heart; and in which there is during life no evidence, either subjective or objective, of its existence. In the *post-mortem* room it is that the occurrence of this very limited form of the malady is occasionally demonstrated in the persons of those who have died of some other complication.

The earliest stage of inflammation of the pericardium consists in hyperæmia of the membrane. It cannot be said that this stage has any specially characteristic symptoms or physical signs. But in acute cases it is accompanied by increased vigor and energy of the heart's action. Without producing any *bruit,* this increased vigor imparts to the first sound of the heart an exaggerated ring or tone, which may be sufficiently marked to enable us to suspect, if not actually diagnose, commencing inflammation of the pericardium. This "tension sound," as it has been called, may be regarded as the earliest objective evidence of the onset of pericarditis. It is only in acute cases that it is observed; and in them the first stage of the disease, during which alone it is heard, lasts for so short a time that this sign may readily escape detection. Even when observed, it is soon thrown into the background by the more striking and important indications of the following stage.

The second stage comes in quick succession to the first. It is characterized by the effusion of lymph, and the formation of a fibrinous layer of new material, on the surface of the inflamed membrane. It is at this period that the signs and symptoms of the disease come to the front, and that its existence is generally diagnosed. The new material which is formed on the surface of the pericardium produces a marked alteration there. Instead of a smooth, glistening surface, allowing the visceral to glide gently and easily over the parietal portion of the membrane, there is a coating of lymph which is so soft that its surface gets roughened by the friction to which it is subjected, and becomes ragged and shaggy in appearance. The rubbing against each other of the thus roughened surfaces of the pericardium produces the "to and fro" friction sound characteristic of the disease. This sound is generally double, but may be single, and then is usually short and with difficulty distinguished from an endocardial murmur. It is generally heard first near the base of the heart; but it may be distinct over the whole organ. It is superficial in character, like a pleuritic rub; but easily distinguished from that by its situation, and by its being independent of the respiratory movements. There is no increase of cardiac dulness. This is the stage at which any subjective symptoms which may exist are usually felt.

The morbid process may go no further than this. The inflammation may decline; the lymph may be reabsorbed; and the pericardium be restored to its natural state. Or the two roughened surfaces may adhere together to a greater or less extent.

In acute cases there is generally a third stage, characterized by the effusion of serum into the sac of the pericardium.

A certain amount of fluid is thrown out during the second stage, at the same time as the lymph. The quantity may be so small that it gives no physical evidence of its existence, and is quickly absorbed when the inflammation subsides.

The presence of a larger quantity gives very decided evidence of its existence. It separates the visceral from the parietal layer of the pericardium. Rubbing of these surfaces against each other thus becomes impossible; friction ceases to be heard; and any pain which there may have been, disappears. The area of cardiac dulness is increased. If the sac of the pericardium be quite full, the region of dulness has the triangular shape of that sac—with the apex above and the base below.

The dulness extends to the left of, and beyond, the apex point. The heart's impulse is not felt as in hypertrophy of the organ. The sounds are distant and indistinct. The pulse is quick and feeble—maybe irregular. There is increased frequency of respiration; and the patient may be in considerable distress.

Under proper treatment, and in the absence of other complications, the fluid is generally absorbed. As it diminishes in quantity, the region of cardiac dulness also decreases. The two layers of the still roughened pericardium once more come into contact; and friction is again heard for a day or two. Here, as in the case in which recovery takes place at the end of the second stage, the friction gradually disappears, and ultimately everything seems to return to its natural state—recovery being apparently perfect. It is doubtful, however, if the pericardium ever quite regains its natural condition. It may possibly do so in some cases; but in most instances more or less extensive adhesions are formed between its two layers. Where the inflammation has been severe, such adhesions are formed over the whole surface of the heart, and the sac of the pericardium is obliterated.

Such obliteration has been regarded by some as a source of much embarrassment to the heart's action, and a cause of hypertrophy and dilatation of that organ. By others it has been said to produce some degree of atrophy of the heart. While a third set of observers maintain that an adherent pericardium gives rise to no symptoms during life, and to no morbid change in the muscular substance of the heart.

It is probable that the age and mode of life of the patient have much to do with the variety of the results noted by different observers; for if the function of the pericardium be to facilitate free movement of the heart, obliteration of its sac will cause most disturbance in those in whom the movements of that organ are most free and active—in young and excitable persons, and in those whose habits or tastes lead them to take much exercise, and so increase the vigor and frequency of the heart's action.

The function of the pericardium is to provide for the free and active movements of special occasions, and unusual efforts. If such occasions do not arise, and such efforts are not made, the obliteration of its sac causes no embarrassment, and no change in the heart. The ordinary work of that organ can be quite well done without the pericardium;

and so long as this work is not unduly increased, and extraordinary efforts are not required, no harm results from the obliteration of its sac. It is for the free movements of special occasions that it is required. "The free motion of the heart within the pericardium is required in health, not so much to meet the necessities of the circulation in its tranquil and ordinary condition, as to provide for the contingency of excited action, and to give abundant scope for the smooth and painless motion of the heart under those circumstances in which the habitual equilibrium of the circulation is disturbed."[1]

It is doubtful if a severe attack of pericarditis, resulting in obliteration of the sac of the pericardium, ever occurs without the muscular structure of the heart participating more or less in the morbid process. The evidence of its being affected would be lost in the more prominent signs of the pericarditis. Such myocarditis might lead to subsequent change in the heart—a change which would not unnaturally be attributed in the *post-mortem* room to the obliteration of the pericardium.

Occasionally the fluid effused into the pericardial sac becomes purulent; but such an occurrence is much more rare than in inflammation of the pleura.

There is one fact in the history of rheumatic pericarditis which has a more important bearing on the pathology of the disease than any other circumstance noted during its course. This fact is that *the disease almost always commences in the visceral layer of the pericardium, and at the base of the heart.*

The evidence of this is as follows:—

1. In slight and circumscribed attacks, the inflammatory change is, as a rule, confined to this portion. Affecting only the visceral layer, and only a small part of it, there may be no physical signs or symptoms by which its existence may be diagnosed. But we know that such slight attacks do occur; for in the *post-mortem* room it is not uncommon to find some thickening and opacity, the result of inflammation of this part of the pericardium, without any evidence of the disease having existed elsewhere.

2. In cases in which the disease spreads over the body of the heart,

---

[1] On the Favorable Terminations of Pericarditis, by W. T. Gairluer, M.D., Edinburgh Monthly Journal of Medical Science, 1851.

and affects both layers of the pericardium, the friction sound is generally heard first near the base.

3. In cases in which death takes place in the early stage of the disease, it is found that while that part of the membrane which is situate over the body and apex of the heart is merely hyperæmic—is still in the first stage of inflammation,—that which surrounds the origin of the great vessels at the base has reached the second stage, and is covered with shreds of lymph. "In ordinary acute pericarditis the earliest stage is seen as a minute injection of its vessels, causing a blush of redness, which close observation resolves into a beautiful red network. This injection is almost a certain proof of pericarditis, but when you see it you should look at the base of the heart, about the great vessels, where you will always find some shreds of inflammatory lymph."[1]

Is there any possible explanation of these facts—any reason why the rheumatic poison should act primarily and chiefly on this part of the pericardium? There is no structural peculiarity to account for it. It cannot be due to greater functional activity; for movement is freer at the apex than at the base. *The only peculiarity of that portion of the pericardium which seems specially liable to rheumatic inflammation, is that it is situate over, and in near contact with, the fibrous textures of the heart*—in near contact, that is, with that particular portion of the cardiac structures, which is specially liable to suffer from the action of the rheumatic poison. The inference is inevitable, that inflammation of the pericardium may be due, not to the direct action of the rheumatic poison on that membrane, but to the extension to it of an inflammatory process originating in the subjacent fibrous textures.

We have already seen that inflammation of the inner lining membrane of the heart is limited to that part of it which is in direct relation with the fibrous rings and valves, and is secondary to inflammatory change in these structures.

There is not a little evidence to show that in many cases, if not in all, inflammation of its outer investing membrane, has primarily a like limitation and a similar pathology.

The fact cannot be too strongly insisted on, that the membranes which line the interior and exterior of the heart, are essentially different

---

[1] Wilks and Moxon, op. cit., p. 98.

both in structure and function: they have indeed nothing in common but their membranous nature and cardiac situation. The endocardium has no vessels, and is therefore not liable to inflame. The pericardium is very vascular. It is the analogue in the heart of the lining membrane of the joints. Its function is to facilitate the free and active movements of a solid structure. Like the lining membrane of the joints, it is very susceptible to inflammation, and the process is liable to spread from one point over the whole surface.

We have already seen reason to believe that inflammation may extend from the fibrous ligaments to the synovial membrane of a joint. There is also reason to believe that it may extend to the pericardium from the fibrous textures of the heart. Indeed, what was said regarding the extension of the inflammatory process to the synovial membrane from the fibrous structures of the joint, is equally applicable to the case of the pericardium. It is in more or less acute cases that inflammation of the synovial membrane, and effusion into the joint, take place: it is in such cases also that inflammation of, and effusion into, the pericardial sac, occur. The more acute the case the more marked the synovitis and joint swelling. The more acute the case the more likely is there to be inflammation of, and effusion into, the sac of the pericardium.

In chronic and in mild subacute cases the fibrous structures of the joints are inflamed, and there may be some thickening of their texture; but, as a rule, there is no inflammation of the synovial membrane, and no effusion into the joint. In similar cases there may be some inflammation of the fibrous structures of the heart; but, as a rule, there is no extension of the inflammation to the pericardium, or effusion into its sac.

CHAPTER XIV.

MYOCARDITIS.

INFLAMMATION of the muscular walls of the heart, is an ailment which owes its recognition to *post-mortem* observation.

Numerous cases have been recorded in which these walls have been found, after death, to be the seat of inflammatory softening, induration, or even suppuration. With this evidence of myocarditis there has usually been associated the usual *post-mortem* signs of endocarditis, of pericarditis, or of both. The existence of this association, and the rarity of *post-mortem* evidence of myocarditis except in combination with inflammation of the membranes, has led to the not unnatural belief that inflammation of the muscular substance is secondary to that of the membranes, and results from the direct extension to it of an already existing inflammation of one or both of these.

Myocarditis occurring independently of endo-pericarditis is described by Walshe [1] as "an affection, to say the least, of extreme rarity."

Peacock [2] says that myocarditis "is rather interesting in a pathological point of view than practically important. It probably always occurs in connection with one or both the other forms of disease," *i.e.*, endocarditis or pericarditis.

"Inflammation of the heart substance is frequently set up in the layers contiguous to an inflamed endocardium or pericardium." [3]

"Inflammation of the muscular substance of the heart rarely occurs except in connection with peri- or endo-carditis. In pericarditis a greater or less thickness of the muscular walls in contact with the inflamed serous membrane is often distinctly implicated; and there is no doubt that their inner aspect may be similarly involved during the course of an attack of endocarditis." [4]

---

[1] Diseases of the Heart, 4th ed., p. 263. 1873.

[2] St. Thomas's Hospital Reports, 1875, p. 12.

[3] The Theory and Practice of Medicine, p. 39, by F. F. Roberts, M. D., F.R.C.P., 3rd ed. 1877.

[4] The Theory and Practice of Medicine, p. 516, by J. S. Bristowe, M.D., F.R.C.P. 1876.

Such are some of the most recently expressed opinions. They accurately represent the view generally entertained by pathologists.

But when we come to examine the grounds on which this opinion is based, we find that they are scarcely adequate to its support, and that the view that myocarditis is secondary to, and dependent on, prior inflammation of the endocardium is one which cannot be maintained.

When considering the subject of endocarditis, we saw that the lining membrane of the heart is a non-vascular structure, in which inflammation cannot, and, as a matter of fact, does not, spread. If the endocardium cannot itself be the seat of inflammation, it is impossible for that process to extend from it to other structures.

The morbid change which takes place in it during the course of acute rheumatism, is secondary to change in the subjacent fibrous tissue, and is limited entirely to that part of the membrane which is reflected over the fibrous valves and rings. It never extends to the part which lines, and is in contact with, the muscular substance, and from which alone it could extend to that substance.

Limited as the morbid process thus is, and showing no tendency to spread, it is in the highest degree improbable, if not actually impossible, that inflammation should ever extend from the endocardium to the muscular substance of the heart.

In cases in which myocarditis coexists with inflammatory or ulcerative change in the muscular portion of the endocardium, the course of events is the reverse of what is usually supposed—the myocarditis is primary, the endocardial mischief secondary.

The only part of the endocardium which suffers in the course of acute rheumatism, is that which is reflected over the valves and rings; and even here, mechanical friction of the segments, consequent on inflammatory thickening of the subjacent fibrous textures, seems necessary to the production of the change. What is chiefly to be noted is that the change in the endocardium is consequent on a prior change in the fibrous tissues. The inflammatory process spreads from fibrous tissue to membrane, and not from membrane to fibrous tissue.

When the endocardium and the muscular substance are involved, we have to deal with a similar course of events: the extension is from muscle to membrane, and not from membrane to muscle.

A consideration of the pathology and mode of production of rheu-

matic endocarditis, lends no support to the view that inflammation ever extends to the muscular substance of the heart from its lining membrane.

It is different with the pericardium. Here we have to deal with a membrane which is richly supplied with blood-vessels, which readily takes on the inflammatory process, and in which that process very readily spreads. That inflammation may, and sometimes does, extend to the pericardium from the muscular substance, there can be no doubt (it was so, for instance, in Mr. Stanley's case, to be presently related). But there is also good reason for the belief that the morbid change sometimes takes the reverse course, and extends from the pericardium to the heart; for in fatal cases of pericarditis, it is sometimes noted that the layer of muscular tissue in immediate contact with the inflamed membrane, is the seat of more decided inflammatory softening than that which lies nearer the endocardium. Sometimes it is the only part which is distinctly softened.

But myocarditis occurs independently of pericarditis; and may involve the whole thickness of the cardiac walls. It is then situated chiefly at the base of the heart, and is usually regarded as due to extension of inflammation from the endocardial lining. But that we have seen to be not only inconsistent with the facts of the case, but to be all but practically impossible. How, then, is it produced? There are two possible explanations. One is to regard it, as we regard all cases of inflammation of the endocardium, and many cases of inflammation of the pericardium, as an extension of the inflammatory process from the fibrous textures. The other is to regard it as primary, as due to the direct action of the rheumatic poison on the muscular substance.

1. The muscular fibres of both auricle and ventricle are attached to the fibrous ring which surrounds, and forms, the auriculo-ventricular opening. This ring is also the tendinous attachment of the cardiac muscles. If inflammation spread to these muscles from any structure, it is most likely to be from that with which they are most intimately connected, provided only that it is the seat of inflammation. The muscular fibres of the heart are most intimately connected with the fibrous rings—so intimately, indeed, that it is difficult to draw a pathological line between them. These rings are a common seat of rheu-

matic inflammation. It is, therefore, in the highest degree probable that, if inflammation spreads from them at all, it will be to the muscular substance. Inflammation may extend even more readily from the fibrous structure of the rings to the very vascular muscular walls, than from the fibrous structure of the valves to their non-vascular endocardial covering. In connection with this point it is to be noted, that localized myocarditis is most common in the neighborhood of the fibrous rings.

But myocarditis is not always so localized; and the question arises whether the disease may not sometimes be primary—due to the direct action of the rheumatic poison on the cardiac muscles.

2. That muscular tissue is sometimes the seat of rheumatism is generally admitted. Most authors describe a form of the disease which, from its situation, is called "muscular rheumatism." Such rheumatism may coexist with the more regular and distinctive arthritic form of the malady.

When considering the action of the rheumatic poison, we saw that the structures specially liable to be affected by it are those which are habitually subject to stretching and strain. There are not very many muscles of which it can be said that they are habitually liable to be stretched. But there are some. The chief of these are the posterior muscles of the spine—those situate between the occiput and the pelvis. These muscles are more or less stretched every time that we stoop or bend. The points at which the stretching is greatest are those at which the spine moves most freely—the neck, and the lumbar region. These are the very points at which rheumatism of the muscles is most apt to be developed. The muscles of the neck and of the loins are at once more liable to be strained, and more subject to rheumatism, than any other voluntary muscles.

But of all the muscles of the body, those which are most subject to strain are the muscles of the heart. If we recognize the existence of a rheumatic affection of voluntary muscle, and of a connection between liability to strain and the tendency to rheumatism, we must also regard rheumatism of the cardiac muscles as a likely occurrence. For much more than voluntary muscles are they apt to be strained; and much more necessary is it that they, like the fibrous textures of the

joints, should be prepared to resist strain. Undue stretching of a voluntary muscle may cause pain, but does no material damage. Undue stretching of the walls of the heart may lead to most serious consequences.

Here, again, we see the difficulty of drawing a pathological line between the fibrous and muscular structures of the heart; for a force which strains and stretches the muscular walls, must also strain the fibrous rings.

There is no *a priori* reason why the rheumatic poison may not act directly on the cardiac muscles, and it is probable that it not unfrequently does so. It is equally probable that inflammation may extend to these muscles from the fibrous textures. In the former case, the inflammation is more likely to be general and involve the whole ventricle. In the latter, it is likely to be local and limited to the neighborhood of the fibrous rings.

Though a point of some pathological interest, it is fortunately a matter of no practical importance which view we take. The important point is that we should recognize the occurrence of myocarditis *independently of inflammation of the membranes;* and should look upon it, as we look upon endocarditis and pericarditis, as a not uncommon complication of acute rheumatism, and as one which may exist, either alone or in combination with inflammation of the other textures of the heart.

It would be at once more accurate for pathological, and more satisfactory for clinical, purposes, if the cardiac lesions of acute rheumatism, instead of being described under the heads of pericarditis, myocarditis, and endocarditis, were classified as pericarditis, myocarditis, and valvulitis,—by pericarditis being meant inflammation of the pericardium; by myocarditis, inflammation of the muscles and fibrous rings of the heart; and by valvulitis, what is usually described as endocarditis—inflammation of the textures of the valves.

But whatever classification we adopt, and whatever names we use, the important points for us to bear in mind are: (1) that the fibrous structures of the heart are the chief seat of the action of the rheumatic poison; and (2) that the muscular substance is frequently involved in the morbid process.

The symptoms of myocarditis are very obscure—so obscure that the

diagnosis of the disease during life is generally regarded as, at the best, a matter of inference rather than of certainty.

"I am not aware that the existence of carditis has ever been diagnosed."[1]

"There are no means by which inflammation of the muscular structure of the heart could be diagnosed during life."[2]

"It is not to be diagnosed during life except as a matter of probability."[3]

"It will be almost impossible to make a diagnosis."[4]

"Myocarditis is but seldom diagnosed during life."[5]

Such is the general opinion. I believe it to be an exaggerated expression of the difficulties of the case. That the diagnosis of myocarditis is always difficult, and sometimes impossible, there can be doubt. But it is equally certain that there are cases in which its existence can be determined with tolerable certainty.

The difficulties are great in consequence of the absence of such distinctive physical signs as are noted in inflammation of the membranes; but they are not insurmountable. They have been much increased by faulty pathological views regarding the causation of the disease. The existence of inflammation of the muscles of the heart, independently of inflammation of the membranes, has not been recognized. It, therefore, has not been looked for; and the indications of its existence are not sufficiently marked to force themselves on our notice.

We are so accustomed to trust to a physical examination of the heart for the diagnosis of morbid change in that organ, that a cardiac malady which cannot thus be readily diagnosed, is likely to escape detection. It has been so with myocarditis. The difficulty has been still further increased by the fact that in many cases subjective symptoms either do not exist, or are not such as are calculated to direct attention to the heart.

Rheumatic myocarditis, like rheumatic endocarditis, is almost en-

---

[1] Walshe, op. cit., p. 264.
[2] Peacock, St. Thomas's Hospital Reports, 1875.
[3] Aitken, Science and Practice of Medicine.
[4] Schroetter: Ziemssen's Cyclopædia of Medicine.
[5] Niemeyer, Text-book of Practical Medicine.

tirely confined to the left ventricle. It may be partial, or general—involving a part, or implicating the whole thickness and extent of the ventricular walls.

1. The former is the more common. Its usual situation is at the base of the ventricle. It probably results in most cases from an extension of the inflammatory process from the fibrous structures. As there is usually a similar extension of that process to the endocardial lining, any symptoms to which the myocarditis may give rise, are lost in the more obvious indications of the valvulitis. It cannot in such a case be diagnosed during life. This is not a matter of much importance; for such limited myocarditis is not a source of danger, and the treatment applicable to the accompanying valvulitis, is equally applicable to it. The evidence of its occurrence is found in the *post-mortem* room, in the form of circumscribed patches of induration of the muscular wall of the ventricle, chiefly at the base, and generally in company with thickening and induration of the fibrous rings and valves.

2. General rheumatic inflammation of the walls of the left ventricle, like other local inflammations, occurs in varying degrees of severity. When very acute, it may give rise to such destructive change in the ventricular walls that recovery is impossible. In a mild and subacute form, it causes simply softening of the ventricular walls—a condition which may be perfectly recovered from, or may result in more or less induration of the muscular substance.

The symptoms and results of these two forms of myocarditis are so different that it will be well to consider them separately. But, though for clinical convenience considered separately, they are merely varieties of the same disease.

Acute myocarditis is a formidable disease, apt to be fatal, and apt to be overlooked. It is apt to be fatal, because of the importance of the tissue inflamed; it is apt to be overlooked, because of the obscure and even misleading character of some of its most common and prominent symptoms.

Corvisart[1] divided cases of myocarditis into two classes, the *distinct* and the *latent*,—those in which the symptoms clearly indicate the

[1] Essai sur les Maladies et les Lesions Organiques du Cœur et des Gros Vaisseaux. Paris, 1818. 3me ed.

nature of the disease, and those in which symptoms directly referable to the heart scarcely exist.

Acute pain in the epigastrium, or præcordial anguish, a sense of oppression and anxiety, embarrassed respiration, the evidence of defective aeration of the blood, without any pulmonary lesion to account for it,—such are the symptoms which may present themselves in distinct cases of acute myocarditis. Seldom, if ever, do they all exist at the same time. Now one, now another predominates. The most common are præcordial uneasiness, and evidence of defective aeration of the blood. With these there are often associated symptoms of disturbance of the sensorium.

Acute myocarditis is generally accompanied by inflammation of the endocardium or pericardium, or both. It is then part of a general carditis. The physical signs of the membranous inflammation are so obvious, that all the symptoms are apt to be ascribed to it. It is probable that the less obvious myocarditis plays a not unimportant part in the production of many of them; but how much of the patient's disturbance is due to the membranous, and how much to the muscular, inflammation, it is impossible to say.

It is a matter of course that when both the walls of the ventricle, and the membranes are inflamed, there is likely to be greater disturbance and irregularity of the heart's action, and a greater tendency to death, than when the membranes only are involved. But it is impossible to diagnose the extent of the myocarditis as we do that of the endopericarditis. All that we can say is that if, in the course of a case of rheumatic inflammation of the membranes, we find either a marked degree of cerebral disturbance, without high temperature; or evidence of defective blood purification, without any pulmonary lesion, or any serious amount of pericardial effusion, we may feel sure that the muscular substance of the heart is seriously involved in the mischief, and that death is threatened by asthenia.

The physical signs of the membranous inflammation, so predominate over any change in the cardiac sounds to which the myocarditis might give rise, that we can better diagnose the existence, and gauge the extent of this latter, by careful observation of the general symptoms, than by a physical examination of the heart.

There are cases of acute myocarditis, as of acute pericarditis, which

run their whole course to a fatal termination without any symptom directly referable to the heart; the only symptoms being those of cerebral disturbance. An admirable illustration of this is found in a case recorded by Mr. Stanley in the seventh volume of the "Medico-Chirurgical Transactions," 1816. So far as I know, it is the earliest recorded case of the kind.

CASE II.—"A boy, aged twelve years, although of a delicate frame, had enjoyed generally a good state of health. On Saturday the 20th of April he was apparently quite well, having been on that day on a visit to one of his relations by whom this remark was made. On the next morning he was brought to the Infirmary, discovering at that time the usual symptoms of fever, namely, great bodily heat, a quick pulse, the tongue white and much furred. On the next day (Monday), his fever was much increased, but the only pain of which he complained was in the left thigh and knee, which ceased before night; in the afternoon he became delirious with much watchfulness. On Tuesday the delirium was very considerable, but without any comatose tendency; the pupil of the eye much dilated, but not insensible to light. He complained but little of pain, but when closely pressed upon the subject, he pointed to his forehead. Early in the afternoon of that day he had a convulsive fit which soon went off. In the evening all his symptoms became aggravated, and he passed the night almost without sleep. On the following morning he appeared much sunk; his breathing for the first time became difficult. He was then sufficiently sensible to answer any question put to him, but soon afterwards he became insensible, and gradually declined till about two in the afternoon, when he expired."

Those who saw this boy thought that his symptoms were of cerebral origin, and "that there was effusion within the head."

The idea of there being anything the matter with the heart did not suggest itself to his attendants; and "at no period of his illness did he complain of pain in any part of the thorax, nor was there any irregularity, either in the action of the heart or pulsation of the arteries."

"It having been considered from the general character of the symptoms that the cause of death was to be sought in the head, this was the part first inspected; but after an attentive examination of the brain,

nothing further could be remarked than that the vessels were generally turgid; not more so, however, than is frequently seen when death has taken place under circumstances that lead to no suspicion of affection of the head." The abdomen was healthy. So were the lungs. "On opening the pericardium it was found to contain between four and five ounces of turbid serous fluid, with flakes of coagulable lymph floating in it. The internal surface of the membrane, both where it constituted the exterior bag, and the reflected layer upon the heart, was covered in various situations with a thin layer of lymph exhibiting a reticulated appearance. The size of the heart was natural in relation to the age of the patient. Upon cutting through its parietes the fibres were exceedingly dark-colored, almost of a black appearance. This evidently depended on the nutrient vessels being loaded with venous blood. The fibres were also very soft and loose in their texture, being easily separable, and with facility compressed between the fingers. Upon looking closely to the cut surface exposed in the section of either ventricle, numerous small collections of dark-colored pus were visible in distinct situations among the muscular fasciculi. Some of these depositions were situated deeply, near to the cavity of the ventricle, while others were more superficial, and had elevated the reflected pericardium from the heart. The muscular fibres of the auricles were also softened in their texture, and loaded with blood, but without any collections of pus between them. All the cavities of the heart were loaded with coagulated blood. The internal lining, valves, and every other part of the organ exhibited nothing worthy of remark, except the state of general turgescence in the capillary vessels, which had also extended to the lower part of the trachea, bronchi, etc."

This case serves well to show that we may have inflammation of the muscles of the heart, sufficiently severe to give rise to suppuration, and to prove fatal in a few days, without a single symptom to call special attention to that organ.

The mistake which is most likely to be made in cases of myocarditis characterized by marked head symptoms, is that which actually was made in Mr. Stanley's case. The symptoms are apt to be attributed to cerebral, rather than to cardiac, mischief.

Such a mistake might prove a serious one to the sufferer, for the treatment appropriate to cerebral rheumatism might prove injurious in

a case of myocarditis. It is, therefore, of importance that they should not be mistaken for each other.

The points of distinction are as follows:—

In cerebral rheumatism.	*In acute myocarditis with prominent head symptoms.*
1. The temperature generally rises to 105° or more.	1. The temperature is seldom above 104°.
2. The pulse is very quick, but of fair strength, and regular.	2. The pulse is quick, but feeble; and may be irregular or intermitting.
3. The breathing is quickened, but not, as a rule, embarrassed.	3. The breathing is quickened, and is generally more or less embarrassed. There may even be orthopnœa towards the end.
4. The face is flushed; the eyes are redly injected; and the lips are generally natural in color.	4. The face is pale or dusky; the eyes are not injected; and the lips are paler than natural, or bluish in color.
5. The heart's sounds are distinct, and the impulse fair.	5. The heart's sounds are indistinct, and the pulse less than natural.
6. Death is threatened by coma.	6. Death is threatened by asthenia.

Acute myocarditis may terminate in the formation of numerous small abscesses, of one larger abscess, or in general softening of the muscular substance, without suppuration.

As a result of suppurative change, ulcers may form on the endocardial, and it is said also on the pericardial, surface. Softening, if it do not prove fatal, may result in weakening, and subsequent dilatation, of a portion of the ventricular wall; leading to the formation of cardiac aneurism, and maybe ultimately to death by rupture of the weakened portion. In very acute cases recovery is scarcely to be looked for.

Subacute myocarditis occurs in the course of acute and subacute rheumatism. Its existence is not generally recognized. I believe it to be of not uncommon occurrence. But so slight are the symptoms and physical signs to which it gives rise, that it may very readily be overlooked. It may exist either in connection with, or independently of, inflammation of the membranes.

When there is a coexistent endo- or peri-carditis, the symptoms and physical signs of this attract undivided attention, and the existence of myocarditis may not even be suspected. Its non-recognition is then not of so much practical importance; for attention is already directed to

the heart by the membranous inflammation, and any signs of feeble or failing action to which the myocarditis may give rise, though not attributed to that agency, are met by appropriate treatment.

It is when there are no signs of membranous inflammation that its recognition becomes a matter of consequence. For though by no means so formidable an ailment as the acute form of the disease, it is one which cannot with safety be ignored.

The signs of its existence are so indistinct and obscure, and so apt to escape observation, that the disease, though recognized in the *post-mortem* room, has hitherto been looked upon as unrecognizable during life. It is not always so. There are cases in which it can be diagnosed. How is the diagnosis made?

Of subjective symptoms there are practically none. Sometimes, indeed, in the more marked forms of the malady, the patient may feel less well, or have a sense of weakness without any apparent reason to account for it, but, as a rule, his sole complaint is of the pain in the joints; and there is nothing, either in his appearance or symptoms, to draw attention to the heart. On examining that organ, as one always does in the course of a rheumatic attack, there is heard no abnormal *bruit*—nothing but the systolic and diastolic sounds, succeeding each other with normal rhythm.

It is in the character of the sounds that lies the diagnostic sign of myocarditis. On careful examination they are found to lack their natural pitch. They are quite audible; but they want the clear tone of health—they are muffled and indistinct.

The character of the sounds here referred to, is not the mere feebleness which is noted in simple debility of the cardiac walls. It more resembles the alteration which is found in connection with hypertrophy. In that condition the ventricular walls are thickened, and their muscular substance increased. But, notwithstanding the greater force of the systole, the first sound at the apex is less clear than normal—it is "dull, muffled, prolonged."[1]

It is a somewhat similar muffling which characterizes myocarditis.

In this condition there is also increased thickness of the ventricular walls, the result of inflammatory swelling, effusion into, and congestion

[1] Walshe, op. cit.

of, their substance. But that which produces the thickening in myocarditis, gives rise, not to increased, but diminished, force of the systole; and this it is which makes the peculiarity of the muffling that accompanies it. The muffling of hypertrophy is accompanied by a sense of force, which is wanting in that of myocarditis.

There can be little doubt that in the thickened condition of the ventricular walls we have the explanation of this peculiar character of the heart's sounds. For the only morbid changes in connection with which it is noted, are also the only ones in which the walls are thickened. In further support of this view it is to be observed that the opposite condition of the ventricular walls, thinning and dilatation, is characterized by an opposite condition of the heart's sounds—unusual clearness.

Muffling of the heart's sounds is not peculiar to myocarditis. But muffling of the heart's sounds *arising in the course of acute or subacute rheumatism*, is diagnostic of inflammatory thickening of the ventricular walls.

When there is coincident inflammation of the membranes, this character of the sounds may be lost, if the membranous affection is at all marked.

The change is one which may very readily be overlooked, and, as a matter of fact, is constantly overlooked. The heart is daily and carefully examined in every case of acute and subacute rheumatism. But what we are intent on finding out, when we make this examination, is whether or not there is any evidence of endocarditis or pericarditis. These are the only forms of rheumatic inflammation of the heart which we recognize, or look for, at the bed-side. We acknowledge the existence of myocarditis in the *post-mortem* room; but we do not acknowledge, either its occurrence independently of inflammation of the membranes, or the possibility of diagnosing it during life. We, therefore, do not look for it. If the membranes are not inflamed, we conclude that the heart is all right.

It is a common thing after an examination of the heart to hear the remark made:—"The sounds are rather indistinct, but there is no *bruit*, no sign of any inflammatory mischief." The very indistinctness to which reference is made, is probably the result of such mischief affecting the walls of the ventricle. There must be a cause for it. If it is

of recent origin, and if there is nothing else to account for it, it is probably due to inflammatory change in the muscular walls.

In certain enfeebled conditions of system there may be a natural indistinctness of the heart's sounds. But apart from this, and apart from pericardial effusion, such indistinctness does not arise in the early part of an attack of acute or subacute rheumatism, except as a result of inflammatory thickening of the ventricular walls. If the sounds are indistinct on the first occasion on which the heart is examined, we may be unable perfectly to satisfy ourselves whether this condition is due to natural debility, or to inflammatory change. But if such indistinctness arise in the course of the case, especially if it be developed early in its course, and quickly; and if the sounds be muffled rather than feeble —a distinction which the practised ear will at once recognize and appreciate,—then we may be quite sure that the walls of the ventricle are inflamed, and that we have to do with a case of myocarditis.

How important it is that we should recognize the existence of rheumatic myocarditis occurring both in connection with, and independently of, inflammation of the membranes, will be seen when the question of treatment is discussed.

By various observers attention has been directed to the occasional occurrence of sudden death in acute rheumatism. It is probable that the explanation of this result is to be found in inflammatory softening of the ventricular walls. It certainly was so in the following case.

Case III.—A girl, aged 20, who had previously enjoyed good health, suffered from an attack of acute rheumatism in March, 1872. The attack was an ordinary one of medium severity, presenting no unusual feature, but complicated by a slight attack of endocarditis which gave rise to no subjective symptom; and the only sign of whose existence was a soft and short systolic blow at the apex. She was treated with acetate of potass, ten grains every three hours.

The endocardial blow persisted, but the rheumatic symptoms had diminished in severity by the end of the second week. On the 17th day of her illness she got up of her own accord. Her sister, who was in the room, stated that her feet had scarcely touched the floor, when she fell back on her bed, and lay there motionless. She never moved, or gave any sign of life. I saw her twenty minutes later, quite dead.

A few minutes before making the effort, she expressed herself as feeling very well, and free from pain.

On *post-mortem* examination, the mitral valve was found to be somewhat thickened, and on its auricular surface was a line of lymphy deposit. The heart's substance, especially that of the left ventricular walls, was rather dark in color, and softer than natural. On firm pressure, it broke down under the finger. On microscopic examination, the normal striæ were wanting in distinctness; and here and there the fibres had a granular aspect. There was a little effusion into the pericardium. There was no ulceration of the endocardial surface; no appearance of embolism; nothing to explain the fatal result, except the altered condition of the ventricular walls. The cranial and abdominal contents were normal.

In this case, endocarditis was diagnosed during life, and with that diagnosis I was satisfied. It did not occur to me that the muscular substance of the heart might be involved; for up to that time I had not regarded myocarditis as of more than pathological interest. The case of this girl, however, made a great impression on me. Since it occurred, I have examined into the condition of the muscular substance of the heart in every case of acute and subacute rheumatism, with as much care and attention as I have devoted to ascertaining that of the endocardium and pericardium; and have satisfied myself that the recognition of myocarditis during life is not the impossible thing that it is usually supposed to be.

The two following cases illustrate the change in the heart's sounds which is believed to result from, and be indicative of, inflammatory thickening and softening of the walls of the left ventricle, in its subacute or latent form.

Case IV.—A man, aged 44, had an attack of rheumatic fever in November, 1874. He had a similar illness in 1866. Was then confined to bed for eight weeks; but made a perfect recovery. His second attack commenced with shivering, *malaise*, and pain in the joints. He was first seen on the third day of it, the 24th of November.

November 24th. Has anxious, pained expression. Lies on his back unable to move, the least effort to do so causing intense pain. Skin covered with acid perspiration; tongue moist and furred; bowels moved

by medicine; urine scanty and high-colored; pulse 120, small and regular, temperature 101.8°; *heart's sounds normal.* To have twenty grains of acetate of potass every four hours, and ten grains of Dover's powder at bedtime. Food to consist of milk, beef tea, and light puddings.

25*th.* Passed an almost sleepless night; general state unchanged. Has great pain in the joints of knees, ankles, wrists, and fingers, which are all swollen; cannot move. Pulse 120, feeble; temperature 103°; *heart's sounds muffled in character and wanting in clearness; no bruit.* Continue treatment.

26*th.* Had an hour's troubled sleep after the Dover's powder. General condition unchanged; lies on his back quite unable to move; profuse acid perspiration; pulse 120, feeble, regular; temperature 102.8°; heart's sounds indistinct and muffled; no *bruit:* cardiac dulness normal. Omit potass and Dover's powder. To have twelve grains of salicin every three hours.

27*th.* Had a better night, sleeping in snatches; looks better, and says he feels so; joints less swollen and tender; skin covered with acid perspiration; pulse 100, of better volume, soft and compressible; temperature 99.6°; heart's sounds still somewhat muffled. Continue salicin.

28*th.* Had a pretty good night; pain nearly gone, but still felt on movement; large joints almost natural in size; those of fingers still swollen; pulse 84, of good volume and character; temperature 98.5°; heart's sounds have lost their muffled character, and are now distinct and normal, though not loud.

Patient made a good convalescence and remained well.

Case V.—A girl, aged 19, who had previously enjoyed perfect health, was laid up with an attack of acute rheumatism in May, 1878. The symptoms of the disease were all well marked.

May 10*th.* Two days ago felt out of sorts; was cold; and had aching in limbs. Yesterday the knees and ankles became very painful. Now she is in bed, the knees and ankles are slightly swollen and very tender; pulse 116°; temperature 101.2°; tongue furred; skin moist, perspiration acid; urine scanty, and high-colored. There is a slight short and soft systolic blow at the apex: cardiac dulness and impulse normal. Has no pain or uneasiness in chest. To have twenty grains

of salicin every hour for six hours, and then twenty grains every two hours.

11*th*. Has had twelve powders, equal to 240 grains of salicin. Feels better; pain and swelling of joints is less; pulse 84; temperature 99°; skin covered with acid perspiration. There is a distinct soft systolic blow at the apex; and the first sound is muffled in character; cardiac dulness normal. Continue salicin, twenty grains every two hours.

12*th*. Had a good night; feels quite well; no pain; perspiration still acid. Systolic blow unchanged; first sound still indistinct and muffled. Continue salicin, twenty grains every four hours.

14*th*. Was not seen on 13th. Feels quite well, and wishes to get up. The *bruit* at apex remains, but the first sound has lost its muffled character, and has now more the tone of health.

To take twenty grains of salicin three times a day; to have good food, and to remain in bed for a week.

In these two cases we have instances of muffling of the first sound of the heart, arising in the course of acute rheumatism. It will be seen that in each, this character of the systole was developed after the patient came under notice; and disappeared soon after the cessation of the joint symptoms and febrile disturbance.

That this change in the heart's sounds resulted from the action of the rheumatic poison, there can be no reasonable doubt; it appeared soon after its action on the system had declared itself, and disappeared soon after the usual evidence of that action had ceased.

The only recognized cause of muffling of the cardiac systole, is thickening of the ventricular walls. Such transient muffling as existed in these cases, must have been caused by a quickly produced and temporary increase in the thickness of the walls of the ventricle. It must have been quickly produced, because the muffling was quickly developed; it must have been temporary, because the muffling lasted only a few days. The only possible cause of such a temporary change in the muscular walls of the heart, is inflammatory thickening and softening, such as was noted after death in Case III.

The circumstances under which the change in the heart's sounds occurred in Cases IV. and V., *i.e.*, during an attack of acute rheumatism, are also those under which such a change in the walls, if it ever occur, is most likely to be developed.

From whichever point we approach the subject, the conclusion is forced upon us, that the change in the cardiac sounds noted in these two cases, resulted from rheumatic inflammation of the walls of the left ventricle.

With such an instance as Case III. before us, it cannot be said that the diagnosis of this condition may not be a matter of supreme importance to the patient.

CHAPTER XV.

THE TREATMENT OF LOCO-MOTOR RHEUMATISM.

If the pathology of rheumatism has been unsatisfactory, its treatment has been not less so. There is probably no disease in which so many different modes of treatment have been had recourse to. There is none in which medicinal treatment has, until very recently, more completely failed to shorten the duration of the malady.

The special mode of treatment in vogue at a given time, has generally depended on the views held regarding the nature and mode of production of rheumatism.

During the last century, and the first half of this, rheumatism was regarded as a "phlegmasia"—as an inflammation dependent, like other inflammatory affections, on exposure to cold; and differing from them only in the nature of the textures involved.

The treatment of inflammation was, at that time, essentially antiphlogistic, and consisted in the adoption of various means of depletion. The chief of these was bleeding.

Sydenham, the father of English medicine, wrote in 1666 that "the cure of rheumatism is to be sought by blood-letting." His rule was to take ten ounces of blood as soon as he saw the patient, to repeat the operation the following day, to do it again in a day or two, and, for the fourth and generally the last time, three or four days later.

But he was not satisfied with the results of this practice; for in 1679, ten years before his death, he says in a letter to Dr. Brady, "I, like yourself, have lamented that rheumatism cannot be cured without great and repeated losses of blood. This weakens the patient at the time; and if he have been previously weak, makes him more liable to other diseases for some years. . . . Reflecting upon this, I judged it likely that diet, simple, cool and nutritious, might do the work of repeated bleedings, and saving the discomforts arising therefrom. Hence I gave my patients whey instead of bleeding them." He gives the particulars of a case treated dietetically, in which the patient "recovered his full

strength, escaping all such discomforts as ten years before a similar attack, which I treated by bleeding, had entailed upon him."

Cullen, though he regarded blood-letting as "the chief remedy of acute rheumatism," and taught that "large and repeated bleedings during the first few days of the disease seem to be necessary," was careful to add that "to this some bounds are to be set; for very profuse bleedings occasion a slow recovery, and if not absolutely effectual, are ready to produce a chronic rheumatism."

Though the indiscriminate use of the lancet was condemned by other able observers, such as Heberden, Fowler, Latham, etc., bleeding continued, till well on in this century, to be the sheet-anchor in the treatment of acute rheumatism.

"In undertaking the treatment of acute or subacute rheumatism, whether we view the inflammatory state of the aponeurotic membranes as primary and idiopathic, or secondary and symptomatic, it is necessary in the first instance to adopt the antiphlogistic method of treatment, and to carry it on with some degree of energy, and to a considerable extent.

"The different branches of the antiphlogistic regimen requisite in the treatment of rheumatism are blood-letting, general and local, the occasional employment of cathartics, the occasional employment of emetics, especially tartar emetic, the use of diaphoretics, and the use of revellents. . . .

"*First.* General blood-letting, in order to be beneficial, ought to be performed early in the disease, and carried to a considerable extent. . . . It should be carried at first to twenty or twenty-five or thirty ounces at once if possible; and within twenty-four hours to as much more.

"*Secondly.* The influence of general blood-letting must be aided by the conjoined operation of various adjuvants. Full vomiting produced by ipecacuanha and antimony is in the majority of cases requisite; and complete evacuation of the bowels by eccoprotics and even cathartics is quite indispensable.

"*Thirdly.* It is of the utmost importance, in attempting the thorough removal of rheumatic pains, to conjoin with blood-letting, or after its use, the administration of full doses of tartrate of antimony.

"*Fourthly.* It is of great moment, if the bowels have been previ-

ously well opened, to exhibit, after the first blood-letting, an opiate of forty or fifty minims of the solution of muriate of morphia; or if the bowels have not been freely moved, to effect this indication, and take a second blood-letting, and after this to administer the opiate, which may be either given alone or conjoined with antimony."[1]

So wrote Dr. Craigie in 1840.

In that year appeared also Bouillaud's "Traité Clinique du Rhumatisme Articulaire," in which the treatment by bleeding *coup sur coup* was advocated with characteristic ability and energy.

To Bouillaud, indeed, belongs the credit of having systematized this mode of treatment. The full extent of his credit in this respect is not generally recognized. Previous to his time there was little or no method in the practice of phlebotomy. To take so many ounces of blood, and to repeat the operation in one, two, or more days, was all the recommendation. Bouillaud insisted that there should not be too long an interval between the different bleedings—that the second should be had recourse to before the effects of the first had fully passed off, and the third before the benefit of the second was lost. That is what he meant by his recommendation to bleed *coup sur coup*. It was the frequent repetition of the operation, rather than the quantity of blood taken, which formed the characteristic feature of his mode of treatment.

If the pathological views which then prevailed were correct, and if bleeding were the important therapeutic agent which it was believed to be, there can be no doubt that Bouillaud's idea was therapeutically sound. No single dose of any remedy could stop a disease like acute rheumatism. It would have to be repeated from time to time; and to get its full beneficial effects the second dose should be given before the first had quite ceased to act. Bouillaud's merit consists in having applied this sound therapeutic rule to the practice of phlebotomy.

The use of the lancet implied also the adoption of other antiphlogistic measures, low diet, purgatives, diaphoretics, etc.

About the middle of this century the practice of phlebotomy, and the pathological views on which it was founded, were vigorously assailed. Facts tended to show that patients recovered more quickly and satisfac-

---

[1] Elements of the Practice of Physic, by David Craigie, M.D., F.R.S.E., Vol. II. 1840.

torily when they were not bled, than when they were. This was noted in acute rheumatism, as in other acute diseases. The rapid accumulation of such facts produced a marked reaction against the old mode of treatment; and within twenty years of the time that Bouillaud's book appeared, the practice of bleeding in acute rheumatism was all but abandoned.

Other remedies besides bleeding were used to allay the inflammation.

Purgatives were at one time a good deal used. Those most in vogue were the saline, chiefly the sulphates of magnesia and soda. Calomel was also thus employed, especially by Latham, and with results which gave satisfaction.

Diaphoretics, especially ipecacuanha and antimony in combination with opium, have been at all times much used. Dover's powder has enjoyed a specially high reputation. Referring to it, Cullen says, "Notwithstanding what I said in favor of venesection, I must own that I never saw a cure very quickly expedited by venesection alone, in the cure of any violent case of the disease; for the disease is liable to linger, and continue for a long time, and to pass into a chronic state. The Dover's powder gives us an opportunity of more effectually and more safely curing the disease than by bleeding alone."

Opium alone, except as a diaphoretic, was condemned by Cullen; but has had much said in its favor in more recent times by Corrigan, Trousseau, and others.

Cinchona, and its alkaloid quinine, have at different times had their claims to favorable consideration pressed. Morton was the first to use cinchona in acute rheumatism. Cullen gave the great weight of his authority against it. He regarded its employment as "absolutely improper and manifestly hurtful," except in cases in which the acute stage had been subdued by bleeding and other measures, and in which the ailment threatened to become periodic. Haygarth,[1] who first used it on the recommendation of Dr. Fothergill, brought forward much testimony in its favor. George Fordyce[2] used it early and freely.

Its alkaloid quinine was at one time freely used, especially in France. A suspicion that it gave rise to cerebral symptoms and dangers pre-

---

[1] Clinical History of Diseases, Part I. 1805.
[2] Elements of the Practice of Physic. 1791.

vented many from trying it, notwithstanding the strong recommendations of Briquet, Monneret, and others.

Garrod [1] has tried to revive this treatment in a modified form. He gives the quinine along with carbonate of potass—five grains of the former and thirty grains of the latter, every four hours "until the joint affection and febrile disturbance have completely abated." The benefits which he claims for this plan are its greater efficacy, a diminished tendency to relapse, and a more satisfactory convalescence.

Colchicum has been much used in the treatment of rheumatism; but there is no valid evidence of its exercising any beneficial action. Garrod, who regards its power to subdue gouty inflammation as beyond doubt, says that "it possesses no influence in checking the progress of rheumatic fever." To give relief to the pain of rheumatism it requires to be given in quantity large enough to cause depression of the heart's action; and that is a condition which cannot safely be induced in the course of an ailment which tends specially to affect the heart.

Guaiacum has long enjoyed considerable reputation as a remedy in rheumatism. Originally introduced by Dr. Dawson, it found its chief advocate in Fuller, who regarded it as useful in all stages of the disease. It is nowadays prescribed chiefly in the chronic form. Any good effects which it produces are probably due to its stimulant action on the skin.

Nitrate of potass was at one time a good deal used as a diuretic and refrigerant in febrile ailments. Brocklesby recommended it in acute rheumatism. He gave as much as two drachms dissolved in some diluent three, four, or five times a day. Given thus, there was got both a diaphoretic and diuretic action.

This treatment was revived by Dr. Basham, who not only gave the nitre internally, but also applied it locally to the inflamed joints. The results of this treatment seem to have been as good as those of any other.

Other remedies, aconite, veratrine, digitalis, actæa racemosa, etc., have enjoyed a passing reputation, and had their claims advocated by different observers. But not one of them has stood the tests of time and investigation.

About the time that bleeding went out of fashion, new views began to be entertained regarding the pathology of rheumatism. This was all

[1] Reynolds' System of Medicine, article "Rheumatism," Vol. I. 1866.

but inevitable. If bleeding was wrong, the pathological view on which that treatment was founded was also likely to be erroneous. If to take blood not only did no good, but even did positive harm, then were there grave reasons for questioning the soundness of what was generally believed regarding the nature of rheumatism. Doubts were started, investigation was stimulated, the symptoms of the malady were subjected to fresh scrutiny, the opinions of the older writers were regarded with a healthy scepticism, and by-and-by new views began to be ventilated.

The question began to be discussed whether rheumatic inflammation was not altogether peculiar, and due to some special poison circulating in the blood, rather than to the operation of any external agency.

The acid condition of the secretions attracted early and prominent attention, and was regarded as a possible cause of the rheumatism. Prout made the definite suggestion that lactic acid was the rheumatic poison. Ably advocated by Todd, Fuller, and others, this view was soon generally accepted, at least in this country.

Altered views of causation led to altered treatment. If lactic acid were the rheumatic poison, it was plain that the proper treatment was to promote its elimination, and to counteract its effects.

It is probable that the *materies morbi* in rheumatic fever is lactic acid or some analogous agent. We know that the natural emunctory of this is the skin. Many chemists maintain that it will also escape by the kidneys; and if it ever does so, perhaps this is more likely during rheumatic fever than at any other time.

Again, since vitiated digestion is apt to produce it in undue quantity, and it, therefore, is formed abundantly in the stomach, there is every reason to think a certain proportion of it may be carried off through the alimentary canal. The indications are, then, to promote the action of the skin, the kidneys, and the bowels; to use antacid remedies; and to give large quantities of fluid for the free dilution of the *materies morbi*, and to supply the waste caused by the drainage from diaphoresis and diuresis."[1]

The acid theory naturally led to alkaline treatment, and that has continued till very recently to be *the* treatment for rheumatism. "If the *materies morbi* be indeed an acid or an acidulous compound—if it be

[1] Todd's Clinical Lectures, p. 69.

lactic acid, for instance, as there are cogent reasons for believing it to be,—then will its neutralization be effected, its irritative property probably diminished, and its elimination promoted, by a free exhibition of alkalies and neutral salts." [1]

The alkaline salts chiefly used have been the bicarbonate and acetate of potass, given in the dose of fifteen to thirty grains every three or four hours. The evidence adduced by Fuller, Garrod, Basham, and others in support of this treatment, is sufficient to demonstrate its superiority over any which preceded it. But the sanguine anticipations of its earlier advocates have not been realized; for it has been found in practice that alkalies may be given so as to render the urine alkaline, without diminishing the joint pain or allaying the fever.

As time advanced, and facts accumulated, it became evident that the alkaline treatment did not materially shorten the natural duration of acute rheumatism, or decidedly diminish the tendency to heart complications—the two advantages which have been specially claimed for it.

We have already seen that the theory on which that treatment is based is untenable; and that the rheumatic poison is not lactic acid, but in all probability a poison introduced from without.

The theory on which the alkaline treatment was founded, being erroneous, we are not surprised to find that treatment fail to produce the good results which its early advocates anticipated. But though it has failed to do all that was hoped from it, there can be no doubt that in some cases the administration of alkalies is undoubtedly beneficial, as will be explained hereafter.

Impressed with a sense of the failure of this mode of treatment, physicians looked about for something better. Owen Rees used lemon-juice, and got from it results which were at least as good as those which followed the administration of alkalies.

Some gave up all medicinal treatment, and simply kept the patient warm in bed, gave him light simple diet, and administered some *placebo*.

Dr. Flint [2] published in 1863 an account of thirteen cases treated on this plan with good results.

Two years later an equally good report was given by Dr. Sutton,[3] of

---

[1] Fuller On Rheumatism, p. 77.

[2] American Journal of the Medical Sciences, July, 1863.

[3] Guy's Hospital Reports, 1865.

forty-one cases treated in Guy's Hospital, and which got medicinally only mint water.

This expectant plan of treatment was adopted by many, with results as satisfactory as those got from more active measures.

"I am quite certain," says Dr. Garrod, "that many cases, even of severe rheumatic fever, get rapidly well without the administration of drugs; and on simple colored or camphor water the improvement is often so quick and satisfactory that, had not the nature of the treatment been known, great virtue would surely have been ascribed to it."

This expectant plan of treatment is really that which was recommended and practised by Sydenham in the later part of his career. To treat a patient by mint water, is practically the same as treating him by whey, which Sydenham did two hundred years ago.

It is curious to find the leading physicians of the nineteenth century going back to the same plan of treatment which was recommended by the father of English medicine in the seventeenth.

Dr. Russell Reynolds, dissatisfied with the alkaline treatment, tried in acute rheumatism a remedy which had proved serviceable in some forms of spreading inflammation, the tincture of muriate of iron. His results were as good as those got by any other treatment; though his cases are too few to be of statistical value.

Dr. Herbert Davies, reviving an old practice, had recourse to free blistering of the inflamed joints, with very satisfactory results.

Such are the chief remedial measures which have been adopted in the treatment of acute rheumatism. Antiphlogistic treatment, the alkaline treatment, and the expectant treatment, are the only ones which have met with anything like general approval.

Antiphlogistic treatment was practised, not because of the proved excellence of its results—for two hundred years ago these were regarded as unsatisfactory by Sydenham, and have frequently since then been called in question by others,—but because such treatment was the legitimate outcome of the views held regarding the nature and mode of production of rheumatism.

The alkaline treatment was adopted, not because it had been proved to be specially beneficial, but because such treatment was a therapeutic sequence of the generally accepted acid theory.

The expectant treatment was the practical expression of the opinion which had gradually been gaining ground, that the results of the alkaline treatment were not satisfactory. It succeeded the failure of the alkaline treatment in the nineteenth century, just as in the hands of Sydenham it succeeded the acknowledged failure of the antiphlogistic treatment in the seventeenth.

Adopting the pathological views advocated in these pages; regarding the rheumatic poison as an organism which is propagated in the system, and the extent of this propagation as dependent on the quantity of the second factor which naturally exists there, we have no difficulty in explaining both the occasional successes, and ultimate general failure, of every plan of treatment to which reference has been made.

If this theory be correct, it is evident that, no matter what the treatment adopted, cases must occur in which the attack will be short, and quickly got over, because the system of the sufferer contains little of the second factor requisite to the propagation and action of the rheumatic poison. Such cases would get quickly well under any treatment, or no treatment.

It is equally evident, on this theory, that no plan of treatment hitherto adopted could materially curtail the duration of the disease in one in whom the rheumatic constitution was very marked, and in whom the second factor existed in large quantity, and was speedily reproduced. In such a one the disease would be prolonged and tedious under any of the old forms of treatment; for not one of them could prevent, or even check, the action of such a substance as, on this theory, the rheumatic poison is believed to be.

Quinine is the only remedy hitherto used which seemed to hold out a prospect of success; and it had already been well tried without any marked result.

But the fact that it exercises a specific action on the disease to which we have seen that rheumatism is most nearly allied, was to be accepted as a hopeful indication that there might be found a remedy capable of counteracting the effect of the rheumatic poison, in the same way that quinine does that of ague.

In connection with the action of quinine on the various forms of intermittent and remittent fever, and indeed in connection with the

action of the Cinchonaceæ generally on the diseases of tropical climates (ipecacuanha in dysentery, for instance) one fact had always strongly impressed me—that the maladies on whose course they exercise the most beneficial action are most prevalent in those countries in which the Cinchonaceæ grow most readily,—nature seeming to produce the remedy under climatic conditions similar to those which give rise to the disease.

Impressed with this fact, and believing in the miasmatic origin of rheumatism, it seemed to me that a remedy for that disease was most hopefully to be looked for among those plants and trees whose favorite habitat presented conditions analogous to those under which the rheumatic miasm seemed most to prevail.

A low-lying damp locality, with a cold rather than warm climate, are the conditions under which rheumatism is most likely to arise.

On reflection, it seemed to me that the plants whose haunts best corresponded to this description, were those belonging to the natural order Salicaceæ—the various forms of willow. Among the Salicaceæ, therefore, I determined to search for a remedy for rheumatism.

The bark of many species of willow contains a bitter principle called salicin. This principle was exactly what I wanted. To it, therefore, I determined to have recourse.

I began to use salicin in the treatment of rheumatism in November, 1874. A short experience of it sufficed to show that my expectations were likely to be more than realized.

In March, 1876, I published[1] some account of my experience up to that date. What I then said was as follows:—

"From so small an experience of salicin as I have had, I would not assert in anything like a dogmatic manner the full extent of its usefulness. I would simply indicate the following conclusions as those to which I have been led, and which, I hope, a more extended experience of its use may confirm.

"1. We have in salicin a valuable remedy in the treatment of acute rheumatism.

"2. The more acute the case, the more marked the benefit produced.

"3. In acute cases its beneficial action is generally apparent within

---

[1] Lancet, March 4 and 11, 1876.

twenty-four, always within forty-eight, hours of its administration in sufficient dose.

"4. Given thus at the commencement of the attack, it seems sometimes to arrest the course of the malady as effectively as quinine cures an ague, or ipecacuanha a dysentery.

"5. Relief of pain is always one of the earliest effects produced.

"6. In acute cases, relief of pain and a fall of temperature generally occur simultaneously.

"7. In subacute cases the pain is sometimes decidedly relieved before the temperature begins to fall; this is especially the case when, as is frequently observed in those of nervous temperament, the pain is proportionally greater than the abnormal rise of temperature.

"8. In chronic rheumatism, salicin sometimes does good where other remedies fail; but it also sometimes fails where others do good."

Subsequent experience, and more extended observation, have necessarily led to a fuller knowledge of the therapeutic uses of the drug; but they have not led me to recall, or even to modify, any of these conclusions. And not only have they been confirmed by my own subsequent experience—they have also been very generally endorsed by the profession.

While these observations on salicin were being made, Kolbe, having discovered a method of manufacturing salicylic acid (originally prepared from salicin) from carbolic acid, was bent on finding some use for it. First tried by surgeons as an antiseptic, it was also freely experimented with by physicians in all sorts of diseases, but chiefly in those attended by fever. Its febrifuge properties were soon recognized, and much that was favorable hoped from, and reported of, its action in typhoid fever, diphtheria, erysipelas, pyæmia, etc. But it was soon seen that the disease in which it did most good was acute rheumatism.

Early in 1876, Stricker[1] and Riess[2] published a most favorable account of their experience of its employment in that disease. Their results were quite in accordance with those which I had got from salicin.

The conclusions at which he had arrived are thus formulated by Stricker:[3]—

---

[1] Berliner Klinische Wochenschrift, Nos. 1 and 2, 1876.

[2] Ibid., No. 7, 1876.

[3] Ibid., February 21, 1876; and London Medical Record, June 15, 1876.

1. Salicylic acid appears to be a rapid and radical remedy in recent cases of genuine acute rheumatism of the joints.

2. It is not injurious to the human organism when administered every hour in doses varying from 7½ to 15 grains.

3. It can be given in these doses for a longer time to young and strong individuals than to the old and feeble.

4. In the latter, it produces toxic symptoms more readily than in the former.

5. The toxic symptoms vary in degree.

6. Those most commonly met with are noises in the ears, difficulty of hearing, and diaphoresis; when these occur the administration of the medicine should be discontinued.

7. If salicylic acid be found to fully answer the expectations entertained regarding it, the internal administration of a certain quantity may be expected to prevent the occurrence of fresh attacks in hitherto unaffected joints, and also secondary inflammation of serous membranes, especially the endocardium.

8. To prevent relapse, the medicine must be continued in smaller doses for some days after the termination of the main treatment.

9. Salicylic acid is of doubtful utility in chronic articular rheumatism.

10. It is not likely to be of use in gonorrhœal or diarrhœal rheumatism, or in the polyarthritis attending septicæmia.

Previous to the publication of the German reports, I had myself, while making my observations on salicin, tried salicylic acid in a case of subacute rheumatism. It did good to the rheumatism, but caused so much irritation of the throat and stomach that it was omitted, and salicin given instead. I did not again have recourse to it, till its use had become general.

Results so striking as those got by the German observers from salicylic acid, and by myself from salicin, could not fail to attract general attention. Observations were made on all hands. The journals in England, France, Germany, and America contained numerous reports of cases of rheumatism treated by these drugs; and it soon became evident that the statements of the original observers were, in the main, correct.

The favorable experience of Stricker and Riess is endorsed by German physicians generally.

In France it has been equally successful. M. Sée tells us that by this treatment he cures acute rheumatism in from two to four days.

In America, Dr. Brown[1] found that from the commencement of treatment to the cessation of pain, the average time was 2.85 days. Dessau[2] found that the majority of his patients could return to their avocations in a week; and thinks that the new treatment is destined to supersede all others.

In this country, the results have been equally good; and from all parts of it have come the warmest commendations of the new treatment. "There are few practitioners who have reported themselves as disappointed in the use of this drug; or, to put it at once strongly and carefully, more disappointed than in the use of quinine for ague. There has not been, in fact, such a consensus of medical opinion on any therapeutic question for many years, as on the power of this drug in one form or other to cure rheumatic fever."[3]

Though the recommendations of the original observers have been, in the main, borne out by the experience of others, there have been reported cases which proved fatal by hyperpyrexia, or by cardiac complications, notwithstanding the free administration of salicin or salicylic acid.

But no one ever claimed for these remedies the power to rob acute rheumatism of all its dangers. In my original paper the action of salicin in acute rheumatism was compared to that of quinine in intermittent fevers, and of ipecacuanha in dysentery. We do not deny the specific effects of quinine in ague, and of ipecacuanha in dysentery, or call in question their power to arrest the course of these maladies, because they fail to cure every case.

In studying this question, it is essential that we should distinguish between the immediate and constant effects of the rheumatic poison, and the more or less accidental morbid changes which may take place in the course of the disease to which it gives rise. An anti-rheumatic remedy may exercise a powerful influence in arresting the former, without having any direct action on the latter.

It would be as unreasonable to expect the salicyl compounds to put

---

[1] Boston Medical and Surgical Journal, February 8, 1877.
[2] New York Medical Record, April 7, 1877. [3] Lancet, July 8, 1877.

a stop to the cerebral and cardiac complications of acute rheumatism, as it would be to expect the cinchona compounds to prevent splenic enlargement in aguish subjects; or ipecacuanha to abolish the hepatic complications of dysentery.

The full extent of, and the exact limits to, the usefulness of the salicyl compounds in acute rheumatism, can be understood only when we have a definite idea, on the one hand, of the true nature of rheumatism, and of its characteristic morbid changes; and on the other, of the mode of action of the salicyl compounds.

The former point we have already considered. We have seen reason to regard rheumatism as a miasmatic disease, resulting from the propagation in the system of the minute organisms which constitute its poison. Its characteristic morbid lesion consists in inflammation originating in the white fibrous tissues, and frequently spreading from them to contiguous textures. The results of such a process, obviously depend on the situation and surroundings of the special portion of fibrous tissue which happens to be the seat of inflammation.

Rheumatic inflammation of the dura mater very soon gives rise to morbid changes which inevitably cause death, in spite of all treatment.

Rheumatic inflammation of the valves of the heart can scarcely exist without producing serious damage to the affected tissue—a damage, too, which is generally permanent. But rheumatic inflammation of the fibrous textures of a joint may persist for weeks without producing more than temporary inconvenience. The cause of the disturbance is the same in each. *It is no peculiarity in the morbid process, but the peculiarity of its seat,* which makes the marked difference in the results—as will be more fully explained further on.

The time has not yet, and cannot for some years come, at which a comprehensive and satisfactory statistical statement of the results of the new treatment can be compiled, and that for several reasons:—

First, the statistics are not sufficiently numerous.

Second, the proper mode of applying the treatment has not been sufficiently known and practised to make even those statistics which we have of like value; and selected statistics lack the impartiality necessary to truthful demonstration.

Third, our knowledge of the pathology of rheumatism is not sufficiently accurate to enable us to distinguish between those cases to which

the salicyl treatment is specially applicable, and those to which it is less suited.

And fourth, the difficulty is still further increased by our ignorance of the mode of action of salicin and salicylic acid.

Were I to utilize any of the statistics and cases which have been published, I should have to utilize all: otherwise I should be open to the charge of partiality, and of selecting only those which seemed favorable. Were I to utilize all, I should present for the reader's consideration a mass of cases differing so much in nature, and in details of treatment, that useful inference would be impossible.

The objects in view are to indicate (1) the cases to which the salicyl treatment is, and those to which it is not, suitable; and (2) the manner in which that treatment should be applied. These objects will be better attained by a judicious selection of cases, successful and unsuccessful, than by giving statistics in which all cases, no matter how different, are slumped together under the one common denomination—Rheumatism; and in which all who took salicin or salicylic acid are regarded as having been treated alike, no matter how different the dose, or how varied the mode of administration of the remedy.

The forms of rheumatism with which we have to deal are the acute, subacute, and chronic. It is to the acute and subacute forms, *i.e.*, to rheumatic fever, that the salicyl treatment is most applicable. To get its full beneficial effects the remedy must be given in full and frequently repeated dose. The patient, of course, is confined to bed; the bowels are relieved by medicine, if necessary; and the diet is light and simple, consisting mainly of milk and farinaceæ.

The salicyl treatment should be begun as soon as possible; for, in the interest of the heart, every hour is of importance. Twenty to forty grains should be given every hour till there is decided evidence of its action. It will generally be found that before an ounce has been consumed, often before half that quantity has been taken, there is a marked improvement. Relief of pain is the first indication of this. With the continuance of the remedy, improvement progresses; and, often within twenty-four, generally within forty-eight, hours from the commencement of treatment, the pain is gone, and the temperature is at or near the normal standard.

As the symptoms decline, the dose may be diminished: but it is

well not to do this too quickly, or too early; for if the remedy be omitted too soon, or given in inadequate dose, the symptoms are apt to recur. The object in view is to keep the system persistently under the salicyl influence.

The salicyl compounds are so rapidly eliminated, that their full beneficial effects can be got only by giving them frequently, as well as in full dose. Moreover, one can never tell the time at which their curative effects have been completely produced. For this reason the salicyl treatment should be gone on with for some time after convalescence seems to be established. There is no definite time at which one can say to the patient, "Now you are all right, and may leave off your medicine without any fear of a return of your ailment." On the contrary, he should be given to understand that there is a tendency to a recurrence of the symptoms, and that the medicine must be taken for some time after all pain has ceased.

It takes about an ounce of salicin or salicylic acid to remove the acute symptoms. That quantity should be taken within the first sixteen or twenty-four hours, in doses of twenty to forty grains, at first every hour, and then every two hours, as the acute symptoms begin to decline. A second ounce should be consumed in the next forty-eight hours. After that, twenty to thirty grains may be taken every four hours, for two or three days; and for a week or ten days more, that quantity should be taken three times a day. By that time the patient will most likely be safe. Any threatening of a return of the rheumatic symptoms must be at once met by a return to large, and frequently repeated, doses of the remedy.

If salicin be the form in which it is given, it may be gone on with for some time without hesitation; for it is a good bitter tonic as well as an anti-rheumatic, and those treated by it convalesce more quickly, and pick up more rapidly, than those who take salicylic acid.

The patient should be in bed for a week. No matter how speedily the pain is relieved and the fever abolished, the affected fibrous textures cannot at once resume their normal condition: and until they have had time to do so, they should have no work to do. It is of the utmost importance that a rheumatic attack should be perfectly recovered from, for the chances are that there will be more of them. Any injury done during a first attack, is almost sure to be increased by subsequent ones.

Treated thus, the course of uncomplicated acute rheumatism is arrested, the pain is abolished, and permanent convalescence begins frequently within twenty-four, and generally within forty-eight, hours of the time that treatment commences. In first attacks, and in young subjects, such is almost invariably the course of events, when there is no cardiac complication.

How different from the duration of the disease under other methods of treatment! Under all of them it was common for the acute symptoms to last for three or four weeks; and twice that time frequently elapsed before the patient was free from pain or able to leave his bed. Now it is often difficult to keep him in bed for more than two or three days.

The following cases illustrate the good effects of the salicyl compounds:

CASE VI.—Margaret T——, aged 16, nursery-maid; never had rheumatism before. Was quite well on April 12, 1876. On the 13th felt out of sorts, and had a general feeling of cold, with some pain in the limbs. On the 14th the pains increased, and towards evening got very bad. Being anxious to continue at work, she got up on the 15th and went about as usual, saying nothing to any one about her pains, which were severe in both arms and legs. On the 16th she got up, but was obliged to go back to bed. In the evening I received a note from her mistress, requesting me to see her as soon as possible, as she was afraid that the girl was dying. On arriving at the house, I was told that the girl was in bed, screaming with agony. I heard her cries before entering the room. She was not hysterical, and the screams were clearly caused by pain.

April 16th, vesp. Lies in bed unable to move, and every now and then screaming with pain. The back, shoulders, elbows, wrists, knees, and ankles are all the seat of severe pain, but the knees and ankles are most complained of. All these joints are slightly swollen, and so exquisitely tender that the least touch or movement causes her to scream. Has no pain in chest. Skin hot, not perspiring; tongue moist and furred; urine scanty, high-colored, and loaded with pink urates. Has a soft blowing murmur with first sound, loudest at apex, but audible over whole heart. Pulse 112; respiration 20; temperature 103.8°. To have fifteen grains of salicin every hour till three powders are taken, and then one every two hours.

17*th, mane.* Wandered at times during the night, but had occasional short snatches of sleep. Pain, especially in ankles and knees, is still severe, but not nearly so bad as yesterday. Can move the right leg a little, and does not complain when the joints are touched. Indeed she allows them to be pretty firmly grasped without complaining; yesterday the least touch made her scream. Tongue furred; skin moist, and perspiration acid. Urine scanty and high-colored; bowels confined. Cardiac blow is softer in character, and precedes as well as accompanies the first sound at the apex. Still heard over whole heart, but not so distinctly as yesterday. Pulse 96; respirations 26; temperature 102.8°. Has had eight powders, equal to 120 grains of salicin. To continue to take fifteen grains every two hours.

18*th, mane.* Had a good night, free from pain. To-day feels quite well. Has no pain in any of the joints, and can move the limbs without more discomfort than what is caused by a slight feeling of stiffness in the knees. Tongue cleaning; skin natural. Pulse 72, barely perceptible; respirations 20; temperature 99.6°. Heart's action irregular; murmur still soft, has lost its systolic character, and is now purely præ-systolic; distinct at apex, but scarcely audible an inch from that point. Has had eighteen powders, equal to 270 grains of salicin.

19*th.* Slept well; is free from pain, and feels quite well. Wishes to get up. Tongue clean; skin natural; bowels moved; urine abundant, of pale amber color. Pulse 70, feeble, irregular; respirations 20; temperature 98.2°. Has had in all 405 grains of salicin.

The cardiac blow remained, but gave rise to no subjective phenomena. There was no return of the rheumatism. She went on with the salicin for a fortnight.

Rapid as was the cure in this case—the pain having been practically abolished within twenty-four, and the temperature brought to the normal within, at the most, sixty hours of the time that treatment commenced—it would probably have been still more rapid had I given the salicin in the full dose in which I now administer it. At the time that Margaret T——'s case occurred (April, 1876) the remedy was still on its trial, and the present certainty regarding the safety as well as the desirability of large doses had not been attained. To such a case occurring now I would give double the dose which Margaret T—— got;

and probably with a more rapid result—as in the following case, which bore a close resemblance to hers.

CASE VII.—Jane S——, aged 17; had always enjoyed good health. With the exception of measles and scarlatina in childhood, never had any ailment.

On May 26, 1878, she felt out of sorts, and had such aching in the limbs that she did not leave the house. On the following day she was worse, and towards night got very bad.

May 28th. Lies in bed, unable to move, the least effort to do so causing intense pain, and making her scream. Has anxious pained expression. The knees and wrists are most painful, but the ankles, right shoulder and neck are also complained of. The affected joints are a little swollen, and exquisitely tender; except over the wrists, there is no redness of the surface. Tongue moist and furred. Skin hot, not perspiring; bowels moved by medicine; urine scanty, and loaded with urates. Heart's sounds normal. Pulse 112; respirations 22; temperature 103°. To have thirty grains of salicin every hour till decidedly relieved.

She began to take the medicine at 6 p. m. Was then in great pain. She felt easier after the third powder; and after the fifth (taken at 10 p.m.), was so decidedly relieved that she fell asleep. Her mother, who remained beside her all night, stated that she would probably have slept on, but that she woke her up to give the salicin at eleven o'clock, and at midnight. After that, she had a powder only every second hour.

29th. Has a pleased, smiling expression. Is quite free from pain, except when joints are pressed; allows one to grasp them, and can move them without more than a feeling of stiffness. Thinks she would have slept all night if her mother had not wakened her to give the medicine. Perspired a good deal during night. Skin is now covered with acid perspiration; saliva acid; tongue cleaner; pulse 88; respirations 20; temperature 99.8°. Heart's sounds normal. Up to this time (9.30 a.m.) has taken eleven powders, equal to 330 grains of salicin. To have thirty grains every two hours.

7 p.m. Has had no pain, and says she would like to get up. Complains only of a slight degree of deafness. This her mother noticed before the patient did. Has perspired a good deal; reaction acid. The

joints feel stiff when she tries to move them, but can be firmly pressed without pain. Pulse 76; respirations 20; temperature 98°. Heart's sounds normal. Has had in all exactly one ounce of salicin, 16 thirty-grain doses.

She continued to take the salicin in gradually diminishing dose for four days, during which she was kept in bed. At the end of that time she was allowed to get up, and the salicin was given in twenty-grain doses four times a day for a week longer. She made a perfect recovery, and had no return of pain.

In this case the acute pain was abolished within six, and all joint tenderness within twenty-four, hours of the time that treatment commenced. Within that time, too, the temperature had fallen from 103° to the normal standard, a fall of five degrees.

CASE VIII.—James S——, æt. 30, a robust well-built man; had rheumatic fever for the first time four years ago. Was then two months in bed, and three off work.

December 31, 1878. Two days ago was seized with pain in right knee. Yesterday the right was a little better, but the left became very painful. To-day the right shoulder and left ankle are also affected. Of the affected joints the right knee is the only one that is swollen, and it is only slightly so; the other joints are very tender, but none of them red. Had little or no sleep last night from severity of pain. Tongue furred; bowels moved by medicine; urine high-colored, and depositing pink urates; skin moist, perspiration acid; pulse 100; respirations 22; temperature 101°. Heart normal. To have thirty grains of salicin every hour for six hours; then every two.

January 1, 1879. Felt better after four powders, and by night was so relieved that he slept well, waking up only twice—on each occasion taking a powder. Has taken altogether ten of them, equal to 300 grains of salicin. Is quite free from pain. Felt so well this morning that he got up and dressed, and at time of visit was walking about the house. Was ordered to go to bed. In bed is quite free from pain; but when walking about felt his left knee and ankle a little. Skin covered with acid perspiration; urine less scanty, of amber color, with slight deposit of urates. Heart normal. Just after getting into bed pulse was 88; respirations 20; temperature 98.8°. A powder every two hours.

He perspired very freely during the day, and in the evening felt so well that he got up again for a couple of hours.

2nd. Slept well all night. Took no powder between ten last night and seven this morning. Has had in all eighteen powders, equal to 540 grains. Perspiring freely—secretion acid; has no pain; swelling and tenderness quite gone; pulse 72; respirations 18; temperature 98.3°. He was ordered to take the salicin three times a day for ten days. He remained well, and resumed work on the 8th of January. In this case the attack was abolished within twenty-four hours.

CASE IX.—John W——, æt. 34; has twice had rheumatic fever, once at 18, and again at 24.

On each occasion was laid up for six weeks. Present illness began three days ago with *malaise* and aching in limbs. Pains have steadily got worse, and have become localized in joints.

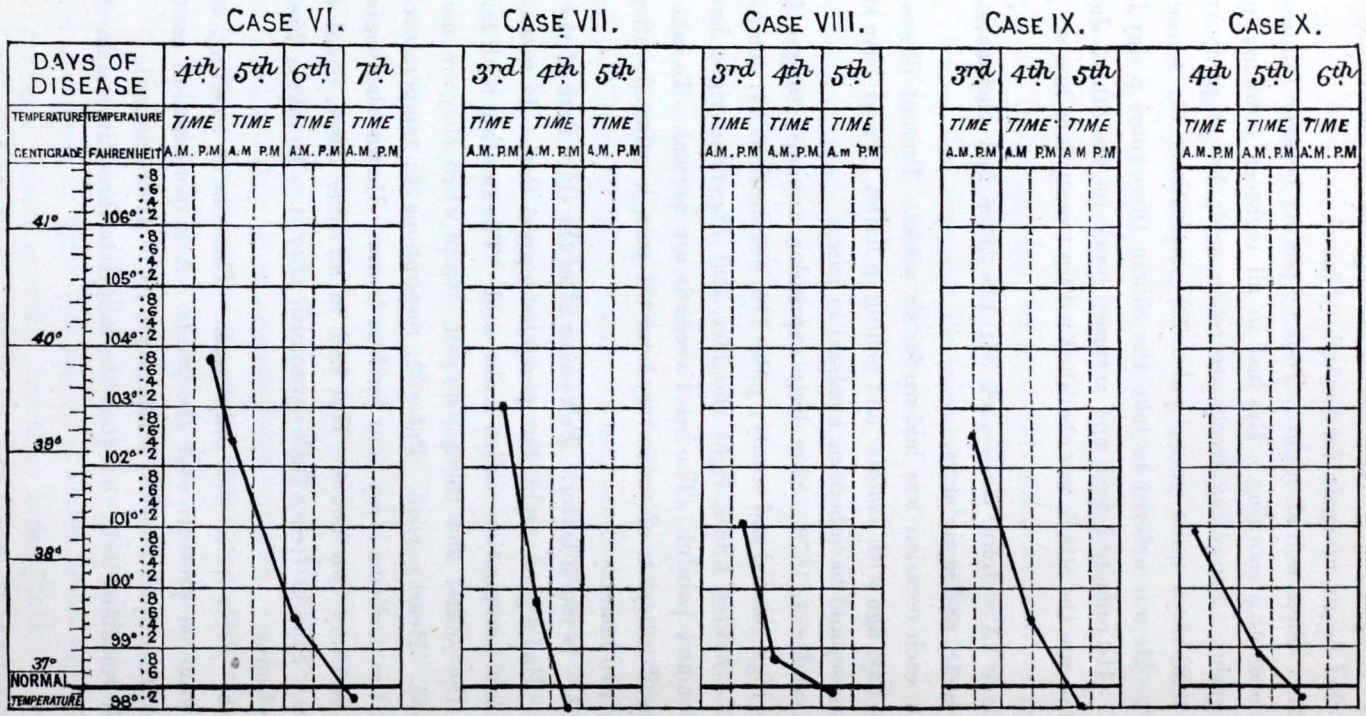
November 4, 1878. Skin hot; expression anxious; tongue furred; urine high-colored and scanty; pulse 116; respirations 24; temperature 102.5°; ankles, knees, right shoulder, and fingers of right hand are swollen and painful. The heart's sounds are normal. To take thirty grains of salicylate of soda every hour till pain is relieved; after that, every two hours.

5th. Is much better. Felt easier after the third dose of the medicine. Had a good night, but perspired a great deal. Is now covered with acid perspiration; saliva is also acid. The swelling is all but gone from the joints; and there is no pain except when they are moved or pressed. Heart normal. Pulse 92; respirations 20; temperature 99.6°. Took the medicine every hour for four hours. He was then easier, and took it every two hours. Has had in all nine doses, equal to 270 grains. Says he feels a little squeamish after it. To take a dose every three hours.

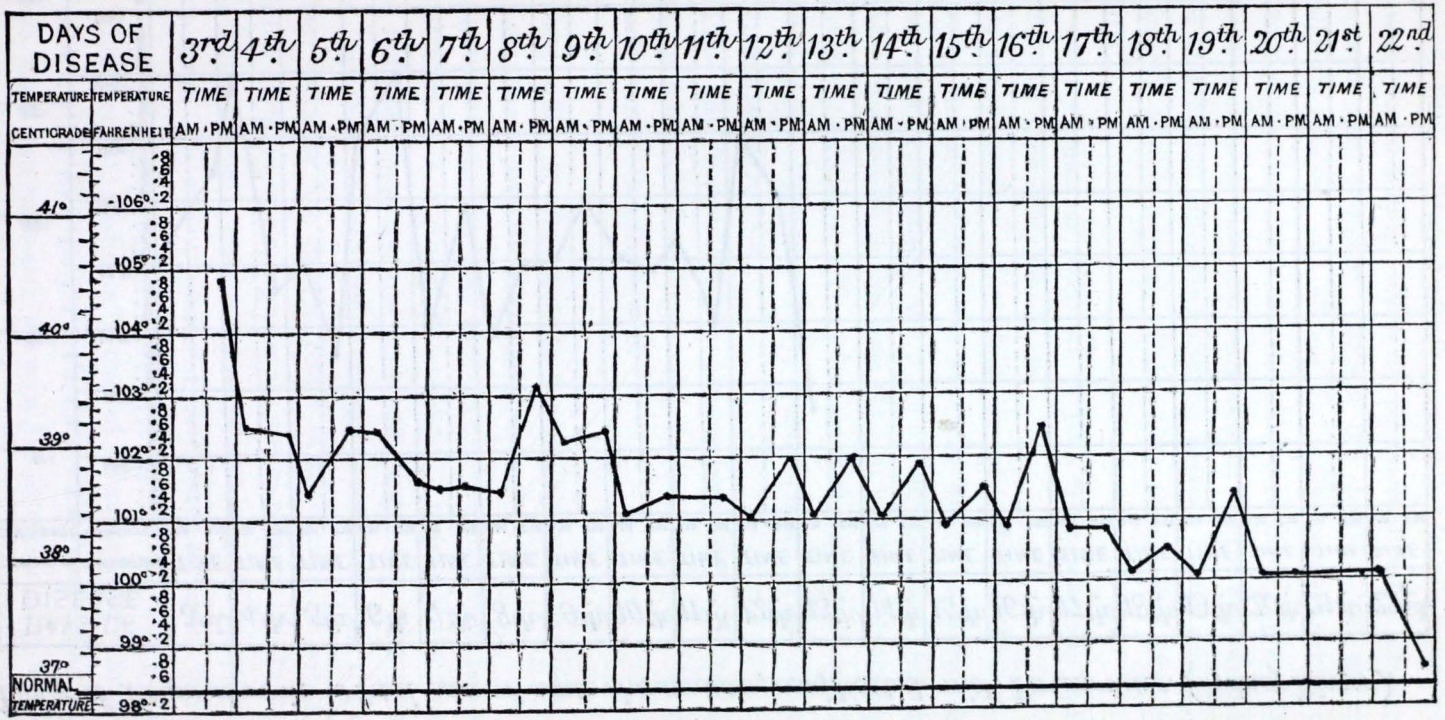
6th. Feels quite well only weak. There is no swelling, and no pain even on pressure over the joints. Acid perspiration continues. Pulse 70; respirations 18; temperature 98°. Heart normal.

He remained well; but took the salicylate three times a day for ten days.

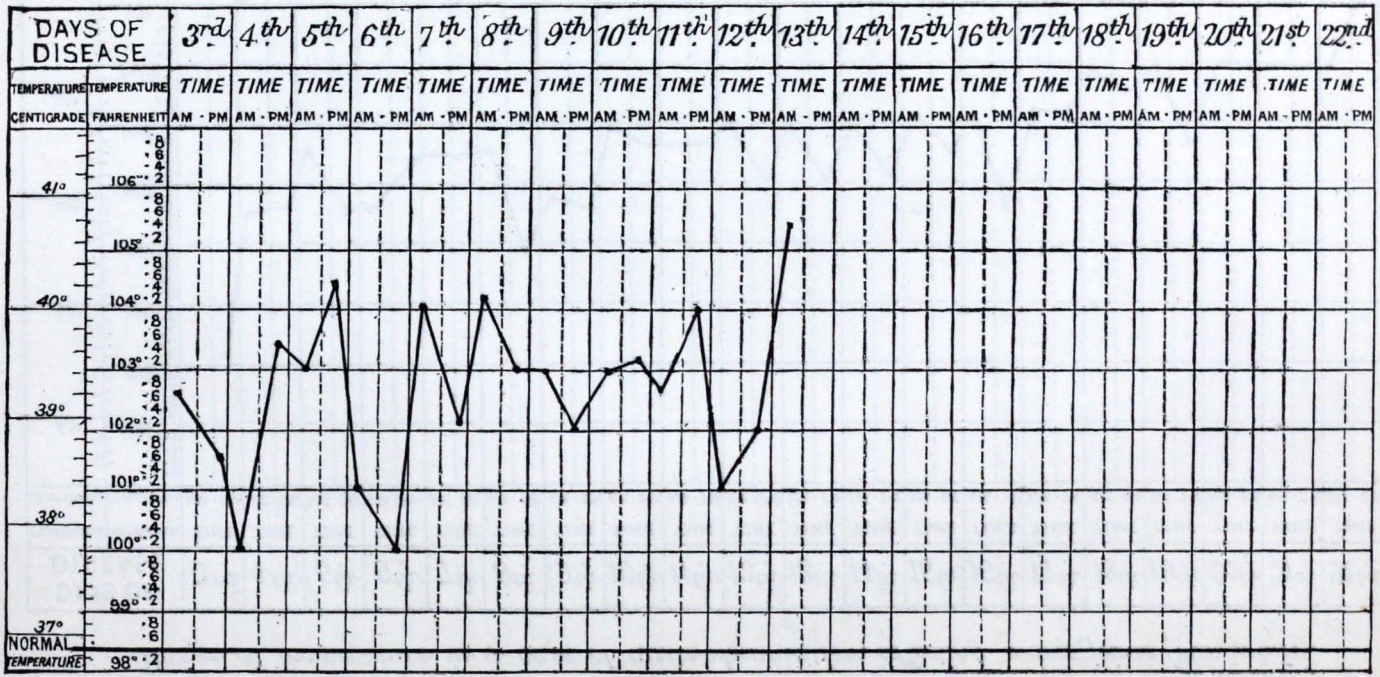
CASE X.—Frederick G——, æt. 22, never had rheumatism before.


CASE VI.
CASE VII.
CASE VIII.
CASE IX.
CASE X.
DAYS OF DISEASE
4th
5th
6th
7th
3rd
4th
5th
3rd
4th
5th
3rd
4th
5th
4th
5th
6th
TEMPERATURE CENTIGRADE
TEMPERATURE FAHRENHEIT
TIME A.M. P.M
41°
106°
105°
40°
104°
103°
39°
102°
101°
38°
100°
99°
37°
NORMAL TEMPERATURE
98°

Range of Temperature in a case of acute rheumatism affecting many joints (Wunderlich).



Range of Temperature in a fatal case of acute rheumatism, complicated with Pericarditis. (Sydney Ringer).



December 12, 1877. Present attack commenced four days ago with sore throat and general aching. Now the pain is localized in left knee, both ankles, and right shoulder. Except the shoulder, the affected joints are slightly swollen, and all are distinctly tender, though the pain is not very acute when he is at rest. Any movement aggravates it much. The skin covered with acid perspiration; tongue furred; urine high-colored and loaded with fawn-colored urates; bowels moved by medicine; heart's sounds normal. Pulse 104; respirations 20; temperature 100.8°.

To have twenty grains of salicylate of soda every hour till pain is decidedly relieved; then every two hours.

13*th*. Is much better. Was decidedly relieved after five or six doses of the medicine, but went on with the hourly dose for eight hours. After that he fell asleep, and woke up only now and then during the night. Has had up to this time (11 a.m.) thirteen doses, equal to 260 grains of the salicylate. The joints are stiff and slightly swollen, but the pain is gone. Perspires freely; secretion acid. Pulse 84; respirations 20; temperature 98.8°.

14*th*. Pulse 68; respirations 18; temperature 98.2°. Heart normal. Took the salicylate four times a day for four days, and thrice a day for a week more. Remained well.

These cases suffice to illustrate the controlling power exercised by the salicyl compounds over the rheumatic process.

In young subjects, and in those who have not suffered from repeated rheumatic attacks, such is generally the course of events, if the remedy is given in sufficient quantity, and for a sufficient time. But it is of importance that these two conditions should be observed; for if given in insufficient quantity, the desired result is got slowly, or not at all; and if omitted too soon, the symptoms are apt to recur. On the opposite page is a diagrammatic representation of the range of temperature in these five cases. By way of contrast I have placed alongside of them a similar representation of the temperature range of two cases given in Aitken's "Practice of Medicine"—one of Wunderlich's which recovered, and one of Sydney Ringer's which proved fatal. The difference between these two cases and my own five is too striking for comment. These five are representative of many others.

The tendency to a recurrence of the disease in those treated by the salicyl compounds has been noted by many. In most cases it is attributable to the too early omission of the remedy, or to its administration in insufficient quantity.

The following two cases illustrate this point.

CASE XI.—Jane M——, aged 25; had rheumatic fever five years ago. Was then laid up for two months. Present attack began five days ago with *malaise* and pains in limbs.

March 2, 1878. Face flushed and anxious; right knee, ankles, and wrists, swollen and very painful; acid perspiration; tongue furred; urine scanty and loaded with urates; pulse 104; temperature 102.4°. Heart normal. To have light diet, and thirty grains of salicin every hour till pain is relieved; then thirty grains every two hours.

3*rd*. Took a powder every hour for five hours; after that felt easier, and took one every two hours while awake; has had in all thirteen powders, equal to 390 grains. Is now free from pain; the joints are stiff and slightly swollen, but not tender; pulse 80; temperature 99.8; skin covered with acid perspiration; bowels moved: heart's sounds normal. To have a powder every two hours till a dozen are taken; and then one every four hours for a week, and to remain in bed for that time.

The patient felt so well that she neglected these precautions; got up on the 5th, and took only an occasional powder after that time.

On the 8th the joint pains returned; and on the 9th I saw her with her old symptoms back again, knees, ankles, and wrists inflamed; acid sweats; pulse 100; temperature 101.9°. She had thirty grains of salicin every hour for six hours; and then every two hours.

10*th*. Is free from pain; pulse 76; temperature 98.5°. Has had 360 grains of salicin. To continue it every three hours for four days, and after that, four times a day for ten days. This time she did as directed, and remained well.

In this case there can be no doubt that the relapse would have been prevented had the patient gone on with the salicin as she was told to do.

What happened was as follows:—

1. The salicin was taken, during the first attack, long enough and in sufficient quantity to destroy nearly, but not quite, the whole of the rheumatic poison.

2. What remained was reproduced, and gave rise to a renewal of the rheumatic symptoms.

3. On the second seizure, the drug was taken for a sufficient length of time, and in sufficient quantity, to destroy the whole of the poison.

4. Convalescence was, therefore, permanent.

CASE XII.—John W——, æt. 44, had rheumatic fever when he was 28 years of age, again when he was 30, a third time when he was 33, and a fourth when he was 37. On the first occasion he was confined to the house for three months, and was unfit for work for other six, nine in all. The second attack was equally long. During the third and fourth attacks he was in bed for six weeks, and off work for three months. Was always treated by potass.

After each attack, chronic shifting pains continued to trouble him for some time after the more acute symptoms had disappeared. When he first returned to work, it took him three quarters of an hour to go a quarter of a mile, so stiff were his joints.

A fortnight ago he began to suffer from twinges of pain in the back, neck, and right leg. Two days ago got much worse, and was obliged to take to bed. For two nights the pains have been so severe that he has had no sleep.

December 1, 1878, 1 *p.m.* Has anxious expression; tongue furred; bowels confined; skin perspiring; perspiration and saliva acid; pulse 116; respirations 30; temperature 102.1°. Has great pain in left heel, all along right leg, and in knee and hip joints of the same side. The neck, and the right wrist and hand, are also painful and tender to touch. The affected parts are tender, but not red or distinctly swollen. The breathing seems a little oppressed. The heart's sounds are free from *bruit*, but are muffled in character; and there is a slight click with the systole. To have an aperient, and thirty grains of salicin every hour.

7 *p.m.* Bowels moved; has had five powders, equal to 150 grains of salicin. Feels altogether better; says he felt easier after second powder; expression improved; can now move his right leg, which he could not do in the morning; moves the arms more freely, and his head with less pain. Has slept more this afternoon than he has done for two nights. Breathing and heart's sounds unchanged; pulse 108; respirations 28; temperature 102.2°. Is perspiring more freely; perspiration acid. Continue powders, one every hour while awake.

2nd, 9 *a.m.* Took a powder every hour till 4 a.m. At that hour he fell asleep, and slept on till eight. Has had up to this time sixteen powders, equal to one ounce of salicin. States that he has no pain, only stiffness of joints and limbs; can bear firm pressure everywhere. Indeed, he felt so well that he got up and at time of visit was sitting in front of the fire, free from pain, but stiff and weak. Skin acting freely; perspiration and saliva acid; pulse 100; respirations 28; temperature 98.4°. States that he can scarcely believe it possible that he is so well. To keep bed, and take thirty grains of salicin every two hours.

8 *p.m.* Has been quite free from pain; so much so that he neglected his medicine, and went from his room into a cold water-closet. Has no pain; but the pulse is 108, the respirations 30, and the temperature 101°. There is a slight click with the first sound of the heart, but the muffled character of the sounds has disappeared.

To remain in bed, and take a powder every hour.

3rd, 10 *a.m.* Had a good night; slept from ten till three without waking; felt quite well then; got up and went to another room to see what o'clock it was; felt weakened and chilled by doing so; slept again from four till seven. Has no pain, but feels weak; perspiration abundant and acid; pulse 92; respirations 28; temperature 98.1°. To have salicin (30 grains) every two hours.

4th. Passed a very good night, awake only once for a short time; feels quite well; heart's sounds normal; acid perspiration continues; pulse 76; respirations 20; temperature 98.2°

At this time I ceased to visit him, but gave strict injunctions that the salicin was to be taken in thirty-grain doses every four hours for a week. Five days afterwards I was sent for to see him again.

Stated that he felt so well that he did not take the medicine regularly, as instructed, but took only half a powder occasionally. Practically the salicin was omitted on the 5th; and on the 7th he put on his clothes and went about the house. The weather at the time was bitterly cold.

On the 8th the pains began to trouble him again.

9th, 5 *p.m.* Has pain in both ankles and right wrist, all of which are swollen and very tender, but not red. Skin hot, not perspiring; pulse 100; temperature 103.3°. Heart's sounds normal. To have thirty grains of salicin every hour

10*th*. After five powders (150 grains) fell asleep, and passed a good night, waking only once. Pain is gone, only stiffness and slight tenderness on pressure remaining; heart normal; pulse 100; temperature 102.7°. Continue salicin every hour. Has had 210 grains in all.

11*th*. Has scarcely any pain, but feels wretched and out of sorts; the joints are not swollen, but are more or less tender on pressure; perspiration acid, not very abundant; pulse 100; temperature 101.5°. Heart normal. Has had 540 grains of salicin. To have thirty grains of salicin and fifteen of bicarbonate of potass every two hours.

12*th*. Is much the same, no special pain; but the tenderness on pressure remains; perspiration slight, but distinctly acid; pulse 100; temperature 101.3°. Continue powders. Has had 750 grains of salicin.

13*th*. Feels much better; pain and tenderness gone; pulse 90; temperature 98°. Has taken 930 grains of salicin.

14*th*. Feels quite well, only weak; pulse 76; temperature 98°. Has good appetite.

He was more careful on this occasion, remained in bed for a week, took salicin for a fortnight, and made a good recovery without any drawback.

In this case the pain was decidedly relieved within six, and was gone within twenty, hours of the time that he came under notice. The temperature, too, had fallen to the normal standard.

It will be noted that during the first attack, neglect of the salicin, on December 2nd, led to a rise of the temperature from 98.4° in the morning to 101° in the evening. Its resumption in hourly doses, led to its speedy fall to the normal.

The second attack was fairly attributable to its early omission.

But the beneficial effects of the salicyl compounds are not always so marked as in the cases which have been given. In those especially who have suffered from repeated and long-continued attacks of acute or subacute rheumatism, these remedies often fail to give the speedy and complete relief which follows their administration in earlier attacks.

The two following cases illustrate this.

Case XIII.—William H——, aged 36; had been subject to rheumatic attacks for sixteen years. The first occurred when he was 20.

Since then has seldom gone more than a year without being laid up with it for some weeks. During the earlier attacks, he was generally laid up for two or three months at a time, but made a recovery which, though slow, was perfect. About six or eight years ago he began to find that the attacks, though really less severe, did not go off as they used to; recovery being less complete, and the joints remaining more or less stiff. As time wore on, and rheumatic attacks repeated themselves, this stiffness became more marked, the pains more constant, and his condition more and more helpless. For two years he has been quite unfit for work; and during the winter and spring unable to go out for fear of catching cold, and making himself worse. Warm dry weather suits him best. The wrists have always suffered much, and are now so stiff that his hands are practically useless. The case, in short, has become one of what is commonly termed chronic rheumatism, varied every now and then by a subacute seizure. These latter occur at irregular periods and intervals. I saw him during one of them in January, 1878. He had been better than usual up to within a week of the time that he came under observation. He had then been confined to bed for four days. His face had an expression of pain and anxiety. Skin moist; perspiration scanty and acid; tongue slightly furred; pulse 96; temperature 100.3°. Urine high-colored, sp. gr. 1.025; no albumen; no sugar; depositing urates on standing. The heart's sounds were normal. The elbows, wrists, fingers, knees, and ankles were stiff, swollen, and painful on pressure and on movement. The wrists, knees, and ankles were most complained of. There was no deformity or alteration in the shape of the joints, except such as was attributable to a general thickening of their fibrous textures. In the wrists this thickening was specially marked.

He was ordered thirty grains of salicin every two hours. On the following day, after 300 grains had been taken, he felt decidedly better, and stated that he had experienced more relief from the powders than from any medicine he ever had before. There was no change in the appearance of the joints, but movement and pressure caused less pain; the pulse was 84, and the temperature 99°. On the next day the pulse was 84, the temperature 98.8°; and he himself feeling better, and comparatively free from pain, so long as he kept quiet. For a day or two more he seemed to improve a little; but it soon became evident that

the salicin was doing no further good—the stiffness and swelling of the joints being undiminished. At the end of seven days, after 1600 grains had been taken, the drug was omitted. During the last three days of its administration its physiological effects were distinct.

Salicylate of soda was then given; twenty grains every two hours for a couple of days; and every three hours for four days more. It produced a feeling of nausea and depression, but did not relieve the pain.

He then got iodide of potass, an opiate at night, and had blisters applied to the affected joints. Under this treatment he improved; the subacute exacerbation was got over; but the state of chronic thickening and pain on movement remained. The patient's circumstances were such that other treatment than that which could be applied at home was inadmissible.

The facts to be noted in this case are, first, the long continuance of the malady; second, the frequency of the attacks; third, the permanent nature of the damage to the fibrous textures; fourth, the good results got from the salicyl treatment during the first day or two of its administration; and fifth, the failure of that treatment to thoroughly remove the symptoms, and rectify the morbid changes.

What had to be dealt with in this case when it came under notice, was an attack of subacute rheumatic inflammation of these altered fibrous textures. There were thus two distinct morbid conditions to be treated: (1) the subacute inflammation of the fibrous textures, due to the direct and present action of the rheumatic poison; and (2) a state of chronic thickening and irritation of these textures, the result of past rheumatic attacks. The salicyl compounds, in virtue of their anti-rheumatic action, allayed the former, but had no action on the latter. Hence their beneficial effects were confined to the first few days of their administration.

The following case exemplifies a less marked, but somewhat similar state of matters.

Case XIV.—George G——, æt. 45; has suffered from rheumatic fever five times, at the ages of 20, 24, 28, 31, and 37. The first three attacks were very prolonged; on each occasion was confined to bed for two months, and unfit for work for four. The last two were shorter,

but his recovery was less perfect, as there seemed to remain some degree of stiffness of the knees, ankles, and wrists.

His present attack (the sixth) began a week ago with pain in the back and limbs. Has been confined to bed for two days. Has had no sleep for two nights, so severe have the pains been.

January 20th. Has pained, anxious expression; tongue furred, and moist; skin covered with acid perspiration; urine scanty, loaded with urates; pulse 112; respirations 26; temperature 102°. Has great pain in right knee and hip, ankles, and wrists; the last-named joints are swollen, and all are tender. Heart's sounds normal. To have an aperient, and thirty grains of salicylate of soda every hour for six hours; after that, every two hours.

21*st.* Has had 450 grains of salicylate (fifteen doses). Says he began to improve after the fourth, and was so free from pain during the night that he slept for three hours on end. The joints are now free from pain except when they are moved or touched; skin covered with acid perspiration; pulse 90; temperature 99.2°. Heart's sounds normal. To have thirty grains of salicylate every three hours.

22*nd.* Slept well; has no pain so long as he remains quiet, but movement brings it back; joints are tender and still slightly swollen, especially the wrists and ankles; pulse 84; temperature 99°; scanty acid perspiration; says the medicine makes him feel sick. To have thirty grains of salicin, instead of the salicylate, every three hours.

23*rd.* Sickness gone; likes the salicin; pulse 80; temperature 98.8°; joints as before.

He went on with the salicin till the 27th. His condition remained unchanged; the joints were slightly swollen, and painful on movement or pressure; the temperature remained from half a degree to a degree above the normal; the secretions continued to be hyper-acid. On the 27th the salicyl treatment was omitted, and he was ordered five grains of iodide and fifteen of bicarbonate of potass in an ounce of water every four hours. Under this treatment he quickly improved; the joints became less tender; the acid state of the secretions gradually passed away; and on the 4th of February he was able to get up. The wrists and ankles remained stiff for some time; they had not quite recovered when he passed from notice. He was last seen on the 19th of February.

The facts to be noted in this case are (1) that the salicyl treatment

speedily allayed the acute symptoms, but failed thoroughly to cure the disease; (?) that after a time it ceased to have any further beneficial influence; and that the potass treatment then did what the salicyl failed to accomplish.

Why was this?

It was, of course, in virtue of its anti-rheumatic action that the salicylate so speedily allayed the acute symptoms. What we have to explain is why it did not thoroughly remove the disease, and make the patient quite well.

The reason is, that in this patient there were two separate morbid conditions to be dealt with. It was his sixth attack of rheumatic fever. The five previous ones had left their mark on him in the form of some thickening and abnormal susceptibility of the textures involved. The two morbid conditions with which we had to deal in the sixth, were: first, the recent inflammation of the fibrous textures, consequent on the present action of the rheumatic poison; and second, the chronic irritation of these textures, produced by the previous rheumatic attacks. The former condition was speedily allayed by the salicyl treatment; the latter was one over which such treatment could have no control.

The lactic acid formed during the early days of the sixth seizure no doubt kept up this irritable state of the fibrous textures; and it was probably by counteracting this agency, and aiding in its elimination, that the alkaline treatment did so much good; it relieved the altered fibrous textures from the irritating action of that acid, and permitted them to return to the condition in which they were prior to the sixth seizure.

These two cases serve to illustrate a morbid condition which it is of much importance that we should recognize. It is a sequence of rheumatism, rather than a distinct form of the disease. It essentially consists in chronic thickening and irritability of fibrous textures which have been the seat of repeated rheumatic attacks.

The history of acute rheumatism abundantly shows, what a very slight knowledge of pathology would indicate, that the textures which have been the seat of inflammation, do not recover their natural tone as soon as the inflammation ceases. There always remains for a time, which varies with the length and severity of the seizure, some thickening of the fibrous textures of the affected joints, causing the stiffness

which is felt after the acute symptoms have disappeared. If the attacks are frequent and obstinate, this morbid change in the fibrous textures is less and less perfectly recovered from: by each succeeding seizure a little more damage is done; and ultimately there is induced a condition of chronic thickening of these textures, which is permanent.

That such a change should take place as a result of frequent and long-continued rheumatic attacks, consists with what we know of the mode of production of similar pathological changes in other organs. Sir William Jenner has shown—and all pathologists admit the accuracy of the observation—that the continued presence in an organ of an increased quantity of blood, gives rise to induration of its substance—that is, to increase of its fibrous tissue. If such a change occurs in organs like the liver and kidney, of whose structure fibrous tissue forms a comparatively small part, it is much more likely to occur in ligaments and capsules which are composed entirely of it.

Clinical experience likewise teaches that, when an organ has been the seat of repeated attacks of inflammation, the local symptoms to which such inflammation gives rise, are apt to recur in a minor degree from the operation of causes which would not have sufficed to induce the original attack. What more common than for chronic bronchitis to be developed as a sequence of one or more acute attacks; and for the course of the chronic malady to be interrupted by subacute seizures, brought on by causes which would not have sufficed to induce the original malady?

A similar instance we have in the readiness with which dysenteric symptoms may be developed in those who have once suffered from the acute form of the disease. Exposure to cold, overfatigue, mental disturbance—causes which could never have originated the disease—will often bring back some of the local symptoms of the original attack in a milder, but still quite characteristic, form.

So it is with fibrous textures which have been weakened and altered by repeated rheumatic attacks. They are rendered irritable and weak by the changes which have taken place in them, and are apt to be disturbed by agencies which have no effect on healthy fibrous textures. Irritation of fibrous textures, no matter how induced, causes pain in the affected part. Hence such disturbance as arises in these altered textures from exposure to cold and damp, gives rise to the same symp-

toms as would result from the action of the rheumatic poison. Originating in true rheumatic attacks, occurring in those who have given decided evidence of being of rheumatic constitution, and characterized by symptoms which are associated with true rheumatism, it is not unnatural that the symptoms should be regarded as due to the action of the rheumatic poison. But such a view is pathologically inaccurate, and pregnant with therapeutic errors. The condition which has to be dealt with is one which, though originally induced by repeated rheumatic attacks, exists, after it has been developed, independently of the cause which gave rise to it. It bears a resemblance to that which frequently follows mechanical injury to the fibrous textures of a joint. When a joint is severely strained, there is more or less injury to the capsule, ligaments, and tendons. This may be so great as to cause permanent change in, and thickening of, these textures. Such altered tissues are frequently the seat of pain which is indistinguishable from that of rheumatism; and such pain is frequently induced by cold, damp, or other disturbing cause.

It is the same with the altered state of the fibrous textures, which results from repeated rheumatic attacks; cold, damp, east wind, change of weather, suffice to induce the same troublesome pain which was originally caused only by the rheumatic poison.

This morbid condition exists in various degrees. The early stage of its development is instanced in Case XIV.: a more fully developed stage in Case XIII.

These are the cases in which the salicyl treatment fails to effect a cure.

When the fibrous textures are in this altered and susceptible state, irritation may be set up in them by other causes than the rheumatic poison. No matter how induced, the symptoms of such irritation are always the same. Pain, swelling, slight rise of temperature, even increased formation of lactic acid, may thus result from inflammation of these altered fibrous textures induced by cold, just as like symptoms would result from similar disturbance set agoing by the rheumatic poison. We may thus have all the symptoms of subacute rheumatism, without any action of the rheumatic poison. Over such an attack, the salicyl compounds can exercise no control.

If the subacute exacerbation be due to the action of the rheumatic

poison, the salicyl treatment will do good for a time, but will fail to cure, because the irritable textures will take some time to regain their normal condition, after the rheumatic poison has ceased to act; and because the lactic acid, formed as a result of their inflammation, tends to keep up disturbance in them. Such a case treated by salicin or salicylic acid would be, and with justice, instanced as one in which these compounds gave only partial and temporary relief.

If the subacute exacerbation were due to the action of cold or exposure, the salicyl compounds would have no remedial effect; and the case would, with justice, be given as an instance in which these compounds failed to give any relief, though given in full and frequent dose.

It is in these cases in which the fibrous textures have been the seat of prior attacks of rheumatic inflammation, that the alkaline treatment often does much good. The change resulting from the former attacks, renders these textures more irritable, and more liable to disturbance from the presence of lactic acid. As a consequence of this, the local symptoms are apt to persist for a time after the action of the rheumatic poison has ceased. They are kept up by the lactic acid; and anything which hastens the elimination of this from the system, tends to shorten the duration of the attack. Hence in such cases the alkaline treatment should be combined with the salicyl. The latter puts a stop to the rheumatic process; the former aids in the elimination from the system of the lactic acid formed during it.

When it is a first or second attack with which we have to deal, the salicyl treatment suffices to effect a rapid cure. But when the patient has suffered from repeated attacks, and when, as a consequence of these, more or less change has been induced in the fibrous textures of the joints, there can be no doubt that alkalies tend to shorten the duration of the malady. They do so by neutralizing the lactic acid, and aiding its elimination by other organs than the skin. They do not cure the rheumatism properly so called; but they hasten the elimination of morbid products formed during its course.

As compared with the frequency of the occurrence of the acute and subacute forms of rheumatism in which it originates, this chronic thickening of the fibrous textures is not common, at least in its fully developed form. For this there are two reasons: first, it is only in a minority of cases that the rheumatic constitution is so marked as to lead

to attacks sufficiently frequent and long-continued for its production; and second, in a large number of those who possess this markedly rheumatic constitution, the heart suffers as well as the joints, and death ensues from the cardiac trouble before there has been time for the development of permanent thickening of the fibrous textures.

Nowadays, treatment is so successful in shortening the duration of acute and subacute rheumatism, that it may reasonably be hoped that this condition will year by year become less common. Meantime, there are many such cases. The existence of these makes it necessary that the true nature of the ailment should be recognized.

Chronic thickening of the fibrous textures is a condition over which drugs exercise little or no control.

One never can be quite sure that the pain at a given time may not be due to the present action of the rheumatic poison. The salicyl compounds should, therefore, always be given for a time, not with the idea of removing the chronic thickening, but with the object of relieving any purely rheumatic symptoms.

Iodide of potass, arsenic, guaiacum, sulphur baths, and other remedies, may be tried. For those who can afford it, the treatment of some of the baths of France and Germany, holds out the best prospect of relief. For those who cannot go there, that treatment should be, as nearly as may be, imitated at home. Blisters and rubefacients often afford considerable relief.

Chronic rheumatism is the ailment for which this condition of chronic thickening of the fibrous textures is most apt to be mistaken. Occurring, as the latter does, in those who have suffered from repeated attacks of acute or subacute rheumatism, and presenting, as it does, many of the symptoms of rheumatism, it could scarcely fail to be mistaken for the chronic form. It is of great importance that the two ailments should not be confounded, for their prognosis and treatment are essentially different.

Wherein chronic thickening of the fibrous textures consists, its name implies. Its pathology we have just considered.

Chronic rheumatism, properly so called, is a very different condition. It is due to the presence and direct action of the rheumatic poison, and is not necessarily, or even usually, accompanied by any perceptible change in the textures involved. It consists simply in rheu-

matic disturbance of the affected tissue. It differs from the acute and subacute forms—not in nature, but in degree, and sometimes in the special textures involved. It is a true rheumatic attack, in which the morbid process and local disturbance are not sufficiently marked to raise the temperature, or to lay the patient up.

The textures involved are the same as those which suffer in the acute and subacute forms, with this difference, that the fibrous aponeuroses and muscles are more apt to be affected.

Indeed, for clinical purposes, cases of chronic rheumatism might usefully be divided into two classes—chronic articular rheumatism, and chronic aponeurotic or muscular rheumatism.

Chronic articular rheumatism usually affects the same joints as suffer in the acute and subacute forms.

Seldom more than one or two joints are affected at the same time; and in none of them is the pain bad enough to lay the patient up. It shifts from joint to joint, and may last, off and on, for months or even for years, the patient during the whole time being never really ill, and yet never quite well for more than a few weeks at a time.

The following two cases serve to illustrate this form of the disease.

Case XV.—Mrs. S——, æt. 34, the mother of seven children; had rheumatic fever when she was 15. Was in bed for three months at that time. At 27 had a second, but much milder attack, which lasted only for eight or ten days. States that she had been subject to joint pains almost ever since her first attack. Is frequently quite well, and free from pain; but for last six months has had it very constantly, but never bad enough to prevent her going about.

May 12, 1877. Has been ailing all the winter. For last six weeks has not been out of the house, as going out always made her worse. Has now pain and stiffness of knees, ankles, and wrists; the last are a little fuller than natural. Heart normal; pulse 70; temperature 98.5°. To have thirty grains of salicin every three hours.

14*th*. Feels better than she has done for months. Pain nearly gone. Has taken thirteen powders, equal to 390 grains of salicin.

16*th* With the exception of a little stiffness of the joints, feels quite well. Is free from pain.

A year and a half afterwards she consulted me about one of her children. Told me that the salicin did her so much good that she had taken it, off and on, almost ever since. Has never enjoyed such good health, or been so free from pain as during the last year.

CASE XVI.—James K——, æt. 35. Had an attack of rheumatic fever when 18 years of age. Was laid up for two months. Has suffered from occasional pains in the joints almost ever since. Thinks he has never been more than three months on end quite free from pain. Its usual seats are the knees, ankles, and wrists. Generally only one joint is affected at a time. For the last few months the pain has been more troublesome, especially in right knee.

December 8, 1878. Has pain and stiffness of right knee and ankle. Neither joint is swollen, but each is tender on firm pressure. Heart's sounds normal; pulse 74; temperature 98.6°. To have thirty grains of salicin every two hours.

9*th*. Feels decidedly better.

11*th*. Is free from pain, and more comfortable than he has been for many months. Has had in all 360 grains of salicin.

He continued it three times a day, in twenty-grain doses, for several weeks; and by my advice took it for some months twice a day.

In December, 1879, he wrote to me as follows:—"It is just a year since you prescribed the salicin for me. I thought it might interest you to know that I have continued to take it ever since, off and on. The dose I take is twenty grains once or twice a day. I have never passed more than a week without it. I was never better in my life than I have been during the past year, and never so free from pain."

The heart is less apt to suffer in the chronic than in the acute and subacute forms; but it does sometimes become involved, as in the following case.

CASE XVII.—John M——, æt. 38, a medical man, consulted me in November, 1878, regarding some cardiac symptoms from which he was suffering. The heart was much enlarged, there was marked regurgitation at both aortic and mitral orifices.

He stated that he began to suffer from occasional rheumatic pains

in 1867, and that he has done so more or less ever since. Was never so bad as to keep the house, except for about a fortnight in 1869. Pain has generally been in knees or ankles, occasionally in the elbows, but seldom in more than one joint at a time. The left knee was the chief seat, but it shifted about very frequently. Has never felt unfit for work except on the occasion referred to, in 1869. Has been short of breath for some time, but always regarded it as asthmatic in nature. The left knee is now occasionally the seat of pain.

The cardiac ailment increased; and he died of it two months after I first saw him.

Rheumatism of the muscles and aponeuroses, is nearly always chronic. Occasionally in the course of an acute or subacute attack, the aponeuroses, especially that of the thigh, may be affected. But such rheumatism, occurring by itself, independently of the articular form, is rarely other than chronic.

It is more common in women than in men. When it occurs in man, it is generally at a more advanced age than the acute articular form.

Its chief seats are the loins, thighs, and shoulders.

Whether or not the fibrous aponeuroses ever suffer without the muscles being involved, and whether or not the muscles ever suffer without the aponeuroses being affected, are points which it is impossible accurately to determine—so intimately are the two structures blended together.

The chief symptom of this form of rheumatism is pain, dull and aching in character, increased by movement and by pressure, varying in degree, but not so apt to shift about as that of the articular form of the malady.

The ailments for which it is most apt to be mistaken, are chronic gouty pain, and neuralgia.

From the former it is to be distinguished by the age, sex, and history of the case. The gouty state is most common in men; and there is generally evidence of its existence, either in prior attacks, or in the present state of the joints, or general health.

From neuralgia it is distinguished chiefly by the situation, and dull aching character, of the pain. Neuralgia of the loins and thighs is generally due to uterine disease.

The following cases illustrate this form of rheumatism.

CASE XVIII.—Mrs. F——, æt. 25; had for several months suffered, off and on, from pains in shoulders, loins, and right thigh. During the last few weeks they had got worse. Pain is increased by motion. Never had rheumatic fever, or any affection of the joints. She has no other local ailment, but feels weak and out of health. This she attributes to pain and want of sleep. Pulse and temperature normal. To have twenty grains of salicin every two hours till the pain is relieved; and then three times a day for a fortnight.

Three weeks later she again consulted me. Stated that the powders quite cured her in three days, and that she then omitted them. A week after she omitted the salicin, the pains returned in the old localities. It was again given in twenty-grain doses every two hours for three days; after which time she was ordered to take a powder three times a day for three weeks. This she did; and with the result that the pain disappeared, her general health improved, and she remained well.

CASE XIX.—Mrs. P——, æt. 28; never had articular rheumatism; had suffered for six weeks from pain in both thighs, hips, and lumbar region. Affected parts are somewhat tender to pressure made with the point of the finger, but not to similar pressure with palm of hand. The tenderness is most marked over upper and outer parts of thighs. Has been rubbing on various anodyne and stimulant applications, without relief. Pulse and temperature normal. To have twenty grains of salicin every two hours.

On the following day she was decidedly relieved; and on the next, after having taken in all 360 grains, was all but free from pain.

She took twenty grains of salicin three times a day for a fortnight, and remained well.

As will be seen from these cases, the treatment of this form of rheumatism is not different from that of the articular form of the disease.

The cases which have been given, serve to illustrate the controlling power exercised by the salicyl compounds over the rheumatic process.

It remains for us to consider how this power is exercised—how the salicyl compounds produce their anti-rheumatic effect.