

**Let your Computer Read this document for you:**

**Click [View](#)**

**Click [Read Out Loud](#)**

**Click [Activate Read Out Loud](#)**

**Then click [View](#) again and select**

**[Read Out Loud](#)**

**[Read this Page Only](#)**

**or**

**[Read to the End of Document](#)**



## LECTURE VI.

E.M. HALE, M.D.

### ENDOCARDITIS.

**Definition — Anatomical Characters — Causes and Pathology —  
Symptoms— Physical Signs — Diagnosis — Prognosis — Treatment.**

GENTLEMEN : I shall, in this lecture, take up one of the most important of the inflammatory affections of the heart; more important than pericarditis, because of the serious valvular lesions which it usually leaves.

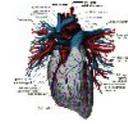
An inflammation of the membrane which lines the cavities of the heart, and is duplicated to cover the valves, is called endocarditis. It is not more than fifty years since this disease has been clearly described and recognized, but clinical experience has demonstrated that it is by no means infrequent. In the majority of cases it occurs as a complication of acute rheumatism. The inflammation may be acute, subacute, and chronic, but these distinctions are not of practical value.

#### **ANATOMICAL CHARACTERS.**

This inflammation is said to be confined, in the vast majority of cases, to the membrane lining the cavities of the left side of the heart. The lining membrane of the right auricle and ventricle is rarely inflamed, and when inflamed it is also present on the left side.

All portions of the endocardial membrane of the left side are not equally subject to inflammation. You must bear in mind that it generally attacks the membrane covering the valves and lining the orifices, and that it is in these situations that it leaves the most troublesome results. There are two reasons for the tendency to attack these localities. 1st. The membrane here is most exposed to the blood-currents; the valvular portion is in constant motion, and is almost constantly in a tense or strained condition. 2nd. The membrane is here under laid by fibrous tissue, and not, as in other situations, in close proximity to the muscular walls of the heart!

Flint believes there are grounds for believing that the fetus *in utero* is subject to endocarditis, and that the inflammation is then limited to the *right* side. He believes the malformations found in infants — cyanosis, etc.—may thus be accounted for.



In the occasional instances in which death has occurred during the inflammation, there has been found (a) redness from vascular injection, (b) alterations in the membrane itself, and (c) the presence of inflammatory products.

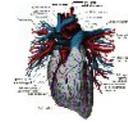
(a) *Redness*, due to endocarditis, is caused by injection of the vessels which ramify in the areolar tissue beneath the membrane, but it is not always found, and may disappear as a *post-mortem* change. But the *redness*, when found, is not always a proof of inflammation, for it may be the effect of the imbibition of haematin dissolved out of the red globules of the blood which the cavity contained after death. This redness from imbibition, however, is distinguished from that due to inflammation by these differences: (1) It is not an arborescent, but a uniform redness, and when examined with a lens, injected vessels are not visible; (2) It has a deeper and darker color than inflammatory redness; (3) It is *not* more likely to be limited to the left than the right side, nor is it usually limited to the valves and orifices, and is more conspicuous in the arteries than in the cavities of the heart. Moreover, in redness from inhibition, the membrane preserves its normally firm, polished appearance.

(b) *Alterations in the membrane itself* are much more indicative of inflammation than redness. These changes are: "loss of the smooth, polished appearance which the membrane has in a healthy state; instead of which it becomes opaque, rough, velvety, and felt-like ; more or less swelling and softening; and brittleness of the subjacent areolar tissue." (Flint.)

(c) *The presence of inflammatory products* will prove to you conclusively, if present, that endocarditis has existed. You remember that the endocardium, unlike other serous membranes, is *not* a shut sac, wherein inflammatory products

may be collected and retained. Such products may be washed away by the currents of blood and carried along with the circulation. Another difference is that the free surface of the endocardium is in contact with the blood itself, and that while the fluid detaches and removes-morbid products, it may also *furnish* deposits by yielding a portion of its fibrin, which undergoes coagulation.

These products of inflammation, then, may be derived from two sources, namely: the exudation of lymph, and the coagulation of fibrin from the blood. The exuded lymph occurs on the free surface of, as well as beneath, the endocardium. That on the free surface, if not washed away, forms layers, as in pericarditis. The roughness of the exuded lymph attracts—as it were—fibrin from the blood, as the threads did when



passed through the arteries in Dr. Simon's well known experiments. When we consider that in acute rheumatism the fibrin of the blood is in excess, we can see how the tendency to fibrinous depositions is greatly increased in endocarditis from that cause.

Various *morbid growths* are to be enumerated as the result of endocarditis. They are commonly called *vegetations*, and are found either at the base or the free extremities of the valves. They occur in the form of small granular masses, or beads, from the size of a pin's head to a millet seed, studding the margins of the curtains of the mitral valve, and fringing the crescentic extremity of the fibrous portion of the segments of the valves of the aorta.

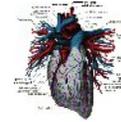
Virchow denies the occurrence of the exudation of lymph in endocarditis. He charges the formation of excrescences and membraniform layers wholly to proliferation or morbid growth, and the coagulation of coagulated fibrin from the blood within the cavities of the heart. Flint, however, believes that morbid growths may have, as a nucleus, the true vegetations or exuded fibrin, to which may be added the coagulated fibrin from the blood.

Other morbid changes may occur, namely: loss of substance by ulceration and erosion; perforation of the valves; lacerations; and even gangrene. Among the rarer occurrences are adhesions of the valves to each other, or to the walls of the heart.

The *remote* effects of endocarditis will be considered in future lectures, when we come to consider the Organic Diseases of the Heart. I will only say here, that in a very large proportion of cases, valvular lesions, involving either obstruction or regurgitation, or both, owe their origin to the anatomical changes which occur as a result of endocarditis. It may be months or years before serious structural changes obtain; but you should carefully watch your patients who have had this disease, and be on the look-out for the •first symptom of organic affection, for it is rare that they are escaped altogether.

#### CAUSES AND PATHOLOGICAL RELATIONS.

Endocarditis is rarely an idiopathic affection. Like pericarditis, it is usually associated with acute articular rheumatism. It differs, however, from the former, in occurring independently of that affection, and is a comparatively rare affection as occurring in other pathological connections. When the two diseases occur together, the combined affections are designated *endo-pericarditis*.



Of 474 cases of rheumatism, collected and analyzed by Fuller, endocarditis existed in 214, the ratio being 1 to every 2.25 cases. Of 204 cases, endocarditis existed in 138, pericarditis in 19, and endopericarditis in 38. Bamberger says endocarditis occurs in 20 per cent. of all cases of rheumatism.

The connection of endocarditis with acute rheumatism is the same as that of pericarditis. It is not developed as a metastasis, but depends upon the same morbid condition that causes the rheumatic affection.

Endocarditis may become developed in connection with renal disease, owing to the analogy of structure between the endocardium and the serous membranes. (See Pericarditis.)

It is said that in non-rheumatic endocarditis the aortic valves are more likely to be the seat of inflammation than the mitral; the reverse being true, as has been seen, of rheumatic endocarditis.

Endocarditis and pericarditis are frequently associated. Fuller says that in 204 cases, analyzed by him, this combination existed in 38. Either affection may take precedence in point of time.

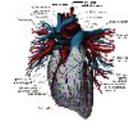
Endocarditis is sometimes associated with pleurisy or pneumonia. It is less frequently associated with these affections than pericarditis. Endocarditis is occasionally developed in connection with the eruptive and continued fevers, and with that morbid condition known as pyaemia, but its occurrence in these connections is rare. It may also be produced by injuries of the chest; but cases of traumatic endocarditis are rare.

The experiments of Richardson, made by injecting into the peritoneal cavity of a dog a solution of lactic acid, containing ten per cent. of the acid, seems to show that *lactic acid*, when absorbed into the blood, will cause endocarditis.

In about twelve hours after the operation, the symptoms and physical signs of endocarditis appeared. Richardson regarded his experiment as proving synthetically that rheumatic endocarditis is produced *by* a similar agent. In rheumatism the morbid conditions are supposed to be caused by the presence of *lactic acid* in the blood.

#### PATHOLOGICAL RESULTS OF ENDOCARDITIS.

Endocarditis may give rise to immediate pathological results which are important, namely: emboli, or plugs, consisting of detached vegetations, or excrescences, which, propelled with the current of blood into the



arteries, are at length arrested in their course in trunks too small to permit their further progress, giving rise to arterial obstruction and diminished supply of blood to certain parts. For a further consideration of this subject, see *Valvular Lesions*.

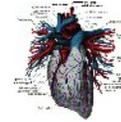
"The solidified products in cases of endocarditis, namely: fibrin and lymph, are, to a greater or less extent, disintegrated by the blood-currents, and carried into\* the circulation, either in solution or suspended in the form of minute particles. It is supposed that the comminuted solid deposits, transported to different organs, and becoming arrested in the capillary vessels, may give rise to vascular obstruction and secondary inflammation in these organs. The kidneys and spleen are most likely to be the seat of disease thus induced. These effects are primarily mechanical; but it is highly probable that morbid changes in the blood itself are sometimes induced by the admixture of the liquid products of endocardial inflammation. It can hardly be otherwise if, as is not improbable, purulent matter is occasionally formed on excoriated or ulcerated surfaces, which are in some instances observed after death in cases of endocarditis." (Flint.)

The formation of large masses of fibrinous coagula in the cavities of the heart belongs to the immediate pathological effects of endocarditis. It is supposed that these ante mortem clots are the formations called by the older writers, polyps of the heart.

#### **SYMPTOMS OF ENDOCARDITIS.**

The symptoms of endocarditis are less distinctive even than those of pericarditis. Occurring generally in connection with acute rheumatism, its symptomatic indications are merged in those of the latter affection. In a large proportion of cases, there are no symptoms which attract attention to the heart as the seat of any disease. Examination, however, with a view to determine the presence, or otherwise, of phenomena which point to endocarditis, may elicit symptoms which are of importance in the diagnosis. These symptoms consist of pain referable to the heart, symptomatic fever, and excited action of the organ, or palpitation. Symptoms arising from obstruction to the passage of blood through the orifices of the heart, do not belong properly to the symptomatology of endocarditis, but are due either to lesions resulting from endocardial inflammation, or to accidental events, such as the formation of coagula. (Flint.)

*Pain* is very rarely a prominent symptom, and, as in other serous inflammations, is sometimes altogether absent. Even when it is present it is not easy to refer it to endocarditis, except by taking into account other



symptoms, and especially the physical signs. The pain is generally dull and obtuse, rarely sharp or lancinating. A feeling of uneasiness hardly amounting to pain, is sometimes referred to the praecordia. The suffering which patients endure from the pain in the joints is so much more severe, that they will not be likely to mention the uneasiness in the heart unless you question them closely. If the pain in the region of the heart is so severe and acute as to cause complaint, the probabilities are that pleurisy or pericarditis is present, rather than endocarditis.

The *fever* is not of any importance, for it is not distinctive of this disease.

*Palpitation* in endocarditis may arise indirectly from excitation of the muscular structure of the heart. The action of the heart may be irregular, as well as unnaturally excited.

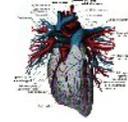
The *pulse* may not correspond with the action of the heart, for while the latter may be acting with increased *force*, the pulse may be weak.

If you observe these symptoms occurring during the course of acute rheumatism, you may safely suspect the presence of endocarditis, and you should resort to physical examination without delay.

#### PHYSICAL SIGNS OF ENDOCARDITIS.

Increased extent and degree of dullness on *percussion*, due to tumefaction of the heart, and accumulation of blood within its cavities, is considered by Bouillard and others as a physical sign of endocarditis. Flint, however, doubts whether the cardiac enlargement often, if ever, exceeds the limit of healthy variations. He thinks if the heart is found enlarged, there must have been a previous hypertrophy. He says, •" how far the size of the heart undergoes alterations during the progress of endocarditis, I am unable to say from my own observations, but it is evident that percussion cannot afford very important information with reference to this disease, except in a negative point of view, that is, by aiding in the exclusion of other cardiac affections, more especially pericarditis."

*Palpation* and *inspection* will furnish evidence of excited action of the heart. The impulse is seen and felt to be more violent than in health, or out of proportion to the amount of febrile movement which exists. But the signs furnished you by these methods of exploration will be of little value to you except as associated with other evidences of endocardial inflammation.



*Auscultation* will furnish you with the only positive proof of the existence of endocarditis, and this proof is the development of the endocardial murmur.

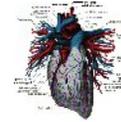
This assertion has been substantiated by clinical experience, and you should give particular attention to its study. The murmur is usually soft, having the character of a bellows sound. It is systolic, for it accompanies the first, or systolic sound of the heart, but you will not find it always at the commencement of the disease. In fact, its existence is considered rare in that stage. I cannot give you any certain data as to the period of inflammation in which this murmur occurs. There are conflicting opinions on this point. The time of its appearance is, however, in the opinion of all authorities,, quite *variable*. The endocardial murmur is not, *of itself*,. absolute proof of existing endocarditis. Previous valvular lesions may be a cause of the murmur. It occurs in consequence of blood-changes, independently of inflammatory or organic disease of the heart.

Under what circumstances then, you will ask,, is the presence of the murmur a diagnostic symptom of endocarditis? If you find an endocardial murmur in connection with symptoms denoting cardiac inflammation, and if. acute rheumatism co-exists; and, further, if you have made previous careful explorations, and failed to discover any murmur, you may then conclude that the murmur you have detected is a sign of endocarditis. If, however, the murmur is discovered on *first* examination the symptom is of doubtful value.

A murmur developed by endocarditis generally continues not only throughout the duration of the disease, but even afterward. There seem to be some exceptions to this rule, for Flint says he has repeatedly known a *mitral* murmur to disappear entirely after recovery from rheumatism, when it was very marked during the disease. This could only occur in those rare cases in which the swelling of the valves diminishes, and the vegetations are detached and- washed away, leaving the endocardial surface smooth.

I need not inform you of the *cause* of the murmurs alluded to. It is doubtless due to a roughness of the endocardial membrane covering the valves, produced by the lymph, fibrin, and vegetations; although some authors have conjectured that the murmurs were due to a spasmodic action of the papillary muscles, preventing the mitral valve from fulfilling its function—allowing regurgitation to take place.

The *heart-sounds* may be abnormally modified during endocarditis. Reduplications have been observed. The first sound, and sometimes the



second, may be less distinct than in health, or the first sound may be wanting.

### DIAGNOSIS.

You will find that the diagnosis of endocarditis does not depend upon symptoms, but almost entirely on evidence developed by auscultation. Those of you who attempt to rely upon symptoms alone will necessarily overlook this important disease, and allow your patients to lose the aid which might be afforded in the prevention of organic disease. Never neglect this in cases of acute rheumatism. Examine your patient every day, and be on the watch for the *endocardial murmur*, which is the only sure evidence of this disease.

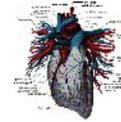
But, as I stated a few moments ago, you\* cannot give a positive opinion unless you have been watching for this murmur at a time before it existed, for if you heard it at the first examination, it may have previously existed from some valvular lesion of long standing. You will readily deduce from this caution, that it will be very difficult, if not impossible, for you to detect endocarditis in a patient who has any organic disease of the heart.

As idiopathic endocarditis is the rarest of all diseases of the heart, I will not enter into discussion of its diagnosis. I must inform you, however, that pericarditis is so often associated with endocarditis, that you will rarely find the latter free from the former. In the diagnosis of endopericarditis, you will have to compare the signs of both; but, happily, the treatment of these affections is not materially different.

### PROGNOSIS.

The prognosis of endocarditis is generally favorable, so far as any immediate danger to life is concerned. The symptoms may continue, and the condition become chronic, and cause great inconvenience, and exist for a long period. Chronic endocarditis may be suspected if the patient continues to complain of uneasiness in the heart, and that organ continues unnaturally excited. But this disease, when chronic, so nearly simulates valvular disease, that the prognosis depends on the extent of the structural lesion.

Certain accidental events may occur during endocarditis which may seriously endanger life, namely: the formation of fibrinous coagula; the detachment of vegetations or of masses of fibrin or lymph, constituting emboli; the admixture of disintegrated solid deposits; and purulent infection of the blood.



If we can judge of the prognosis from recorded clinical experiences, we can believe that, in a large majority of cases of endocarditis, recovery takes place without serious accidents. The cardiac symptoms gradually disappear. The patient, however, is generally left exposed to the evils arising from valvular disease, which may become developed at a period more or less remote, unless your treatment has prevented such a result.

### **TREATMENT OF ENDOCARDITIS.**

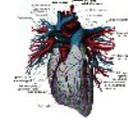
The treatment of endocarditis cannot be disposed of so summarily as some authors have done, by asserting that there is "no essential difference" in the treatment of endocarditis and pericarditis. Not only is the treatment not the same, but the objects of treatment are dissimilar. Flint very tersely observes, that in pericarditis the compression of the heart by the accumulation of liquid within the pericardial sac is a source of distress and danger; and to prevent this accumulation, and promote its removal, are important therapeutical ends. In endocarditis, however, the action of the heart is free from all mechanical restraint. In pericarditis the inflammation is more generally diffused, and a greater effect is produced upon the muscular walls, first by excitation, and afterwards by paralysis. In endocarditis the inflammation is seated especially in the membrane connected with the valves and orifices, when it is not in contact with the muscular walls, and the latter are consequently affected in a less degree. In pericarditis the aim of the practitioner is often to avert impending death. In endocarditis there is little fear of a fatal result.

But although the two affections are so dissimilar in many respects, the general principles of management are in a great measure alike applicable to both.

The therapeutical indications in the treatment of endocarditis relate mainly to the alterations to which the membrane is exposed, and to the products of inflammation.

Your objects must be to diminish as much as possible the local effects of the inflammation; then to aid in restoration from these effects, and thus protect the organ from the remote consequences arising from incurable and progressive unsoundness. In other words, you must accomplish, if possible, the following results:

1. Abate the intensity of the inflammation.
2. Abridge its duration.



3. Limit the exudation of lymph.
4. Diminish the precipitation of fibrin.
5. Effect the removal of solid deposits.

The remedies to which you must resort to gain these ends are, mainly: *aconite*, *veratrum viride*, *digitalis*, *arsenicum*, *spigelia*, *phosphorus*, *colchicum*, *bryonia*, *asclepias*, *lachesis*, *naja*, *sulphur*, *belladonna*, *rhus*.

*Aconite* is useful in the first stage of the disease, when the pulse is hard, small, and quick, and there is pain of a sharp, pricking description in the cardiac region, with anxiety, fear of death, faintness, oppression, and tumultuous action of the heart, (see also "*Aconite* " in Pericarditis.)

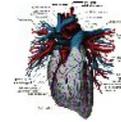
*Veratrum viride* you will find useful in those cases which are ushered in by a more violent congestion, pain, and intense force of the circulation. The bounding pulse—full, hard, and quick; the intense throbbing headache, *without delirium*, will indicate this remedy, in the lowest dilutions. With these two agents, aided by a low diet and absolute rest, you will be able to abate the inflammation and shorten its duration. But the disease is often so rapid in its course, that you will be obliged to anticipate the second stage, and alternate with the above:

*Bryonia*, if the pains are of the severe *stitching* character peculiar to the medicine. This remedy is powerful against the exudation of lymph, and is equally useful in endocarditis as in pericarditis.

*Asclepias tuberosa*, as I remarked when speaking of pericarditis, has nearly the same sphere of action as *bryonia*.

*Colchicum* is probably more suitable to pericarditis than endocarditis. I am of opinion, however, that it is not so much the local condition that indicates this remedy, as the condition of the blood. *Colchicum* causes an extraordinary increase in the secretion of uric acid, and it is this which makes it such an invaluable remedy in cardiac affections occurring during attacks of gout. It is, however, equally useful in rheumatic endocarditis.

*Belladonna* is an excellent remedy in endocarditis, especially when there is secondary irritation and congestion of the brain, and also where there occurs congestion of the chest. It is best indicated when the first is passing into the second stage, and we have the following symptoms: aching in the cardiac region, taking one<sup>^</sup> breath away and causing anxiety; occasional intermittence of the pulse; irregular, unequal



contractions of the heart; throbbing pain beneath the sternum, near the epigastrium; violent heart-throbbing, with jarring of the head and neck.

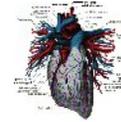
*Digitalis*, from the very nature of its action, cannot prove of much value in endocarditis, unless it be associated with pericarditis. As Baehr very properly remarks, "In endocarditis it seems almost impossible to indicate special heart symptoms requiring the use of *digitalis* ; in such cases, the constitutional symptoms will have to determine our choice." However, as endocarditis rarely occurs unattended by some pericardial inflammation, *digitalis* should always be thought of, and selected according to the indications given under pericarditis.

*Arsenicum* will not control acute endocarditis, but will be found useful in those severe paroxysms of palpitation, or the attacks of cardiac syncope, which sometimes occur. It will be most indicated if Bright's disease or pyaemia be the cause of the endocardial inflammation.

In direct contradiction to Baehr, Dr. C. Muller asserts that " *Arsenic* has been found serviceable in palpitations, carditis, endocarditis, rheumatic, and organic diseases, especially of the left side of the heart," and he adds that " *Arsenic* possesses the most perfect specific and homoeopathic relation to endocarditis. It is indicated not only in the commencement of the disease, but also when exudations and vegetations have formed on the endocardium and valves, especially of the left ventricle. It is hence the main remedy in Bouilland's so-called chronic endocarditis. It is indicated when the following physical signs are present: dullness over a greater extent than usual in the cardiac region, especially in a vertical direction; violent and irregular action of the heart, with febleness or almost complete extinction of pulse; indistinctness, or roughness, of both sounds of the heart, or a bellows-murmur with the first sound, heard on the left ventricle and along the aorta, but loudest over the aortic valves, viz., at the edge of the third rib, near the left edge of the sternum."

*Spigelia*, according to Baehr, is only useful in incipient endocarditis, or to endocarditis generally before marked valvular changes have taken place. For the special indications for *spigelia*, I refer you to your notes on pericarditis, and the remarks I shall make in the future consideration of valvular diseases.

*Phosphorus* will undoubtedly be of use to you in some cases of endocarditis. I can do no better than to give you the indications pointed out by Baehr, which I consider reliable. He says "*Phosphorus* is clearly suitable for inflammation of the endocardium and the muscular tissue, never for pericarditis. The constitutional symptoms would lead us to

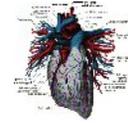


recommend *phosphorus* in endocarditis associated with pneumonia, against the cardiac inflammation, not against the pneumonia, for we have already stated that when pneumonia is complicated with cardiac inflammation, the presence of the latter constitutes an absolute obstacle to the absorption of the pneumonic exudation. In the next place, *phosphorus* takes the precedence over every other medicine in cardiac inflammation, when occurring as complications of such processes as lead us to infer a dissolution of the blood, like scurvy, puerperal fever, malignant exanthematous diseases, typhus, etc. Finally, we possess few remedies that embody in their pathogeneses as plain a picture of nephritic and cardiac inflammation as *phosphorus*."

Baehr is of opinion that we have often cured cardiac inflammations with *phosphorus* without knowing it. In the conclusion of his observations he says, "Finally we have to point out a symptom which most decidedly indicates phosphorus, we mean the dilatation which develops itself during endocardial inflammation with, such surprising rapidity."

I will add that *phosphorus* appears to be pathologically indicated in this disease. The *post-mortem* appearances observed after poisoning by *phosphorus* are: the muscular tissue is flabby and easily torn; opaque appearance and interstitial distension of the endocardium; bloody infiltration, which in certain circumscribed spots penetrates the whole thickness of the muscular tissue. If, in addition to these appearances, we consider the very feeble, small, and exceedingly frequent pulse; the frequent observation that the sounds of the heart either often disappear or are replaced by murmurs, we cannot deny that this remedy ought to be tried in severe cases, especially those of a secondary character.

*Lachesis*, as well as all the serpent-poisons, exerts a profound and specific action on the heart, and more upon the endocardium than the pericardium. In all cases we find an unusual increase of the frequency of the pulse, which becomes feeble and small, or intermittent; there is pain in the region of the heart, the pain being sometimes intense; palpitation of the heart; fearful anguish; bloody expectoration, with constant hacking cough; marked symptoms of cyanosis; icy coldness of the extremities, with cold perspiration; paralytic sensation in the whole left side of the body, or a violent pain in the left shoulder and left arm. In my opinion, *lachesis* (also *crotalus* and *naja*) is indicated in the second and third stages of very acute and severe cases of endocarditis, when arising from pyaemia, typhus, or during malignant eruptive fevers. It is homoeopathic to the vegetations and structural changes which occur, for *post-mortem* examinations after death from serpent-poisoning have always shown the heart more or less affected. The endocardium in the region of the valves is infiltrated and easily torn; ecchymoses cover its



internal and external surfaces; and exudations occur in the muscular tissues of the heart. Take these indications, together with the many symptoms recorded in our *materia medica*, and you will have no difficulty in selecting *lachesis*. *Dose*: the dilutions from 6th to 30th.

*Iodine* and its combinations should not be neglected by you in endocarditis. They are indicated in sub-acute cases, and not so much for the inflammation as for the structural changes occurring on the valves.

*Cuprum aceticum* has been reported by a Dr. Kissell<sup>1</sup> as having cured three cases of rheumatic endocarditis, but the testimony is based on the observation that "Auscultation revealed a bellows-sound at every other beat of the heart; feeble pulsation of the heart." The symptoms of the heart were: "pressure under the lower part of the sternum, and oppression of the chest; pulse, 85; quick pulsation of the heart, and the tone feeble and dull, as if it came from great depth."

*Dose*: the tincture, or 3rd to 6th triturations.

*Apocynum androsemfolium*. In endocarditis-rheumatica, when the rheumatic affection has been confined to the hands and feet, and there is present considerable gastric and hepatic irritation.<sup>2</sup>

*Dose*: the first dilution has been found most useful.

*Asclepias syriaca*, as you will observe from the provings, will likely prove of service in endocarditis from renal affections, or from a retention of scarlatinal poison.

*Dose*: the tincture, a few drops, in water.

*Baptisia* will prove in your hands an unrivalled remedy in endocarditis during or following typhoid or other low fevers, especially when you have present its well known characteristic symptoms.

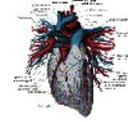
*Dose*: the first decimal dilution, in drop doses.

*Cactus grandiflorus* will never fail you, in the severest cases of the acute form, if that peculiar symptom, "Sensation of constriction of the heart, as if from a band around it, preventing movement," is present. No other remedy equals it in controlling the severe nervous palpitations common to the disease. I get the best effects from the 1st dil.

---

<sup>1</sup> Marcy and Hunt's Practice.

<sup>2</sup> New Remedies, 2nd Ed.



*Cimicifuga* must not be forgotten in the treatment of endocarditis rheumatica. It may be better suited to pericarditis, but if the former affection have a rheumatic origin, I see no reason why this remedy should be neglected. If the endocardial inflammation occur in a woman from suppression of the menses, and there is also intense headache, delirium, stitching pain in the region of the heart, in the infra mammary region, and down the left arm, you can give the *cimicifuga* with every prospect of success.

*Dose:* the dilutions below the 4th.

*Hamamelis.* I have reason to believe that in certain cases of phlebitis, the inflammation may be transferred or extend to the heart. If such a condition does occur, this remedy will be found useful. In the provings we find it to cause pricking pain in the region of the heart, felt also in the superficial veins of both arms, continuing for ten days. By referring to the provings you will find other important symptoms of cardiac inflammation. This medicine acts best in the lowest dilutions.

*Lachnanthes* will have to be used if cerebral symptoms occur, which indicate its use. The cardiac symptoms of this remedy are also notable.

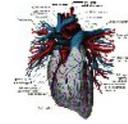
*Lobelia inflata* has been used successfully in some cases of endocarditis. The characteristic symptoms calling for its administration are very striking: excessive vomiting; an intense sensation of sinking at the stomach; sighing; great dyspnoea; deep seated pain in the region of the heart; and small, quick, and feeble pulse.

*Dose:* the dilutions from 1st to 6th.

*Phytolacca* may prove useful in sub-acute or chronic endocarditis if the patient has rheumatic symptoms, and the cardiac pain extends to the *right* shoulder and arm instead of the *left*.

*Sanguinaria.* The sphere of action of this medicine resembles that of *phosphorus*, in so far as the complication of endocarditis with pneumonia may occur. If you meet this complication in practice, do not forget to study the provings of *sanguinaria*, for you may find it indicated when *phosphorus* or *sulphur* have been used without the expected curative results. If the *right* lung is the seat of the inflammation, it is to be selected instead of *phosphorus*.

You will remember that in my lecture on pericarditis I mentioned the efficacy of the alkaline treatment, as a means of combating the existing pathologico-chemical cause of rheumatism. The same observations will



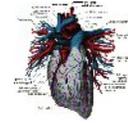
apply to that treatment in endocarditis. It may also prevent the formation of the solid products incident to the latter, by depriving the blood, to a certain extent, of its excess of fibrin. The *carbonate of ammonia* has been highly recommended for this purpose, as being more powerful than other alkalies, and it is affirmed that its use will prevent the formation of large fibrinous coagula.

The indications during convalescence and subsequently are: to advise against undue exercise, the abuse of stimulants, the avoidance of agitating emotions, and excesses of any kind, which might bring back the inflammation, or hasten the development of structural lesions. But you must not go so far as to alarm your patient to exceeding caution, for an extreme sedentary life should be equally avoided. An amount of physical activity necessary to vigorous health is not unfavorable as regards the liability to organic disease. You must not excite the fears of your patient, for the moral effect of looking forward to probable organic disease is not favorable to mental or bodily health.

#### MYOCARDITIS.

A brief allusion to myocarditis will close this lecture. This is an inflammation of the muscular structure of the heart. The muscular substance of the heart is the seat of inflammation much less frequently than the investing and lining membranes of the organ. It rarely, if ever, occurs independently of endo- and peri-carditis. Either the investing or lining membranes, or both, are implicated. The inflammation, probably, in most instances extends from these membranes to the muscular substance. Theoretically, it ought to occur more frequently as a complication of muscular than synovial rheumatism. Practically, however, we do not know that this is the case. The inflammation is usually limited to certain portions of the heart, and it occurs much oftener in the left than in the right ventricle. It may be confined to the outer or inner layers of muscular fibres; or it may extend throughout the walls and affect the *columnae carnae*. The septum is less likely to be affected than the ventricular walls. The results of myocarditis are *suppuration, induration, and aneurismal dilatation*. Rupture of the heart may also occur.

Where abscesses exist, there is purulent infiltration of the surrounding parts. The formation of abscesses destroys the muscular structure to a great extent. They may discharge their contents into the pericardial sac, giving rise to acute pericarditis; or they may evacuate into the ventricular cavity, and give rise to purulent infection of the blood. In either case a fatal result is inevitable.



The *diagnosis* of myocarditis is nearly impossible. There are no symptoms or signs which can warrant a positive opinion.

The *treatment* does not differ materially from that advised in the former diseases. I will suggest, however, that where there is reason to suspect myocarditis, *aconite*, *bryonia*, *belladonna*, *cimicifuga*, and other medicines having a special affinity for muscular structure, would be chiefly indicated.