Subacute bacterial endocarditis

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SENIOR THESIS

SUBACUTE BACTERIAL ENDOCARDITIS

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1935
SUBACUTE BACTERIAL ENDOCARDITIS

Subacute bacterial endocarditis may be said to be a disease, bacterial in origin, which takes advantage of previously damaged heart structures, principally the valves, and there causes pathological proliferative changes which later complicate the disease with a multiplicity of symptoms and signs, these in turn making the problem of diagnosis appalling.

In reviewing the literature the writer found that most authors were of the opinion that the disease was first described by Sir William Osler in his Glustonian Lectures of the year 1885.(1) This in a sense is no doubt true, since only such a great observer as he could properly describe a new clinical entity of disease. However, Kerlin, (2) in his reading has reported the finding of an article "On Some of the Principle Effects Resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart and their Mixture with the Circulating Blood" written by one W. Senhouse Kirkes in 1852.(3) In this paper Kerlin says is described a very comprehensive account of the disease now known as Subacute bacterial endocarditis.

As stated above, Sir William Osler (1) in 1885 first called attention to the disease in its various forms and described certain peculiar skin lesions, of which mention is made below, which have gained the name of Osler's sign, or Osler's
painful nodules. These skin lesions were noted and reported by various authors and observers between the years 1873 and 1900. In 1899, a woman observer, Mabel Austin, reported and described the first case due to the Bacillus influenzae. (2)

During the present century there have been many important contributions by Koeniger (20), Lohlein(21), Libman(6)(7), Coombs(22) and Clawson(23)(38) on the pathological side; Schottmeuller(8), Rosenau(24), Horder(25), Major(26), Kinsella (27), Fox(28), Lynch(29) and Clawson(23)(38) on the bacterial and serological aspects; Osler(1)(40), Libman(6)(7), Billings (16), Vacquez(30) and Kostner(31) on clinical viewpoints.

Waddell(4) states the Libman has put in the most extensive study of any one man and believes him to be the most authoritative on the disease, although it is hard to estimate just who is the leader in this field, since it is composed of so many competent observers and highly conscientious workers.

As to the disease itself, Cecil(5) believes it to be of more importance than the acute type because of its comparative frequency. It is very common for an average 200 bed hospital to have several cases every year and in localities where rheumatic disease is common there are naturally more cases. In addition, the disease is becoming more frequently recognized. Libman, in his 1918 paper (6) stated that those who considered
this disease of less importance because of its infrequency only asked those of that category to consider his own practice in which he had seen over three hundred cases personally in those first years of this century, and that then they should judge its importance.

ETIOLOGY
In 1910, Libman and Celler(7) and their co-workers reported the results of their investigations on 43 cases over a period of eight years. Of these 43, 29 were seen in the four years just previous to their report (1906-1910). In this series of cases the duration of the disease was from four months to one or one and one-half years. Cases which progressed longer than this period he grouped as the chronic type. None of these cases recovered.

In all there were nineteen autopsies and in fifteen of these there had been antemortem blood cultures made. One of these was consistently negative, which according to later reports by this same and other authors was no doubt due to the fact that cultures often must be allowed eight to ten days to grow before they are declared negative, and then such cultures should be repeated regularly.

In these cases the pathological process was always engrafted upon a previously diseased or damaged heart valve and the history nearly always pointed to a previous rheumatic infection. The lesion was always vegetative and not ulcerative. When the aortic valve was involved, there was a marked tendency
for the process to extend down to the ventricle, to the ventricular surface of the aortic flap of the mitral valve and to the chordae tendinae. If the mitral valve was involved there was a marked tendency for the process to spread over the wall of the left auricle and chordae tendinae of that side. The latter structures were often found to be torn. In certain cases the auricular and ventricular involvement was often more marked and extensive than that on the valves. This, Libman says, accounts for the fact that auscultatory findings change so little during the disease.

Interesting is the manner of distribution of the lesions. In ten cases the mitral valve alone was involved, although accompanied by marked auricular vegetations. In two cases the aortic valve alone was the site of the process with some ventricular involvement. In seven instances both valves were the site of the disease and in six of these cases there was marked auricular endocarditis. It is thus apparent that lesions of the auricle practically always occur when the mitral valve is involved.

Changes found in one case indicated that the lesions of subacute bacterial endocarditis may heal to a certain extent, some were undergoing organization, while others were taking on a fibrous or calcareous character. In this case there were no bacteria in the blood nor in the vegetations. As a
rule the vegetations contain innumerable organisms.

In one case of this series the Bacillus influenzae was found. Before death this case had given the clinical picture and findings which were essentially the same as that of the group which demonstrated cocci. This case had even the painful erythematous nodules.

All cultures were made on blood plates. On these plates wherever a colony grew, the hemoglobin seemed to be attracted. The colonies then would become deep red and the remaining portion of the plate would become pale. In some cases the cultures would show large numbers of organisms, while in others cultures would have to be made several times before a positive result could be obtained. In one instance after repeated blood cultures had failed, five different media were used with no avail. At this early date Libman believed that six days were necessary for culture before it was declared negative. However, in a later paper, 1913 (6), he stated that from eight to ten days should be the time factor. Kerlin (2) also stated this. Usually the cultures are found to be positive in 24 to 48 hours. In order to properly identify this organism which was found in over 95 percent of the cases, it was first necessary to rule our the Pneumococcus and the Streptococcus mucosus, which closely resemble the cocci found in these cases.
The results of their experimental investigations led to the following conclusions:-

1) The type of capsule of the Pneumococcus and Streptococcus mucosus, described by Buerger(34), are diagnostic of all organisms present and such capsules are dissolved by bile.

2) The solution of the capsule by bile is diagnostic of the Pneumococcus and the Streptococcus mucosus. The latter is distinguished by its capsule and other features.

3) The absence of a precipitate speaks against the organisms being streptococci.

The cocci found in cases of subacute bacterial endocarditis were carefully studied along planned lines. These organisms are all gram positive. They have never been found to have a capsule and they are either round, ovoid, lancet-shaped, or bacillary in form, and are found in groups or in chains. Every organism was found sooner or later to grow in long, convoluted chains. They are usually smaller than the ordinary Pneumococci or Streptococci and grow more poorly, especially when first isolated.

Schottmeuller (3) described this disease about the same time as Libman and gave it the name of Endocarditis lenta.
He cited five cases and isolated these organisms which he called Streptococcus mitior or Streptococcus viridans since they were poorly growing and produced a greenish coloration on the culture plate.

Libman and his co-workers called their organisms the Endocarditis cocci par excellence or the attenuated Streptococcus of endocarditis, and thought it to be undoubtedly the same as that described by Schottmeuller, although the latter observer believed it specific. On this point the Americans did not agree. At the conclusion of his article Libman summarizes, "As we have already indicated, we have thus far found no help in diagnosticating this disease by microscopic examination of the blood, by opsonic agglutination, or by complement fixation tests. The absolute diagnosis must, for the present, rest upon the cultural study of the blood."(7)

Thayer in 1925 reported his observations on cases for a period of 35 years. Of these he found that 25 cases were of purely rheumatic origin without a complicating bacterial septicemia. He found that the age incidence ranged from six to 47 years, that 76 percent of all cases occurred in the first two decades of life and 88 percent occurred in the first three decades. He adds that in rheumatic disease death is usually later and that in subacute bacterial endocarditis, death pre-
In Cecil's Textbook of Medicine(5), we find the author of the opinion that at least two factors are apparently essential in order that the disease become manifest in the human being. One is the presence of a pre-existing valve injury which is almost always due to an earlier rheumatic infection. Libman believes this factor to be almost constant(7). The other factor is a superimposed blood stream infection. In most cases one can usually elicit a history of infected tonsils, abcessed teeth or otitis media, etc. It is under these conditions that bacteria, prevalent around the focal lesion, can and do invade the blood stream and so, also, the damaged valve. It may be necessary to assume that fresh erosions of the valve have occurred. In older individuals, atheromatous changes in the aorta and aortic valve, as well as non-rheumatic forms of endocarditis(chronic) may constitute the preceding damage. Congenital defects of the heart are also easily infected.

Waddell(4) modifies the factor of rheumatic history in that he states that only about fifty percent give a history of rheumatic infection. Of these that do, most of them show a period of good health in between the attack of rheumatic disease and the onset of subacute bacterial endocarditis. At the time he published his article(1925) he stated, by repiti-
tion of Libman(7), that the only positive method of diagnosis is by blood culture. He also adds that the accountable reason for the slowness of the disease in progressing is due to the fact that the streptococcus viridans is one of low pathogenicity.

Kerlin(2) emphasises that the disease is one of young adults and adolescence. That is, it occurs during the period of greatest incidence of valvular heart disease from all causes except syphilis and arteriosclerosis. As to the bacteriology, he considers only the Streptococcus viridans in 95 percent of the cases and the Bacillus influenzae in the remaining five percent. He further observed as to sex incidence and found that about sixty percent of the cases were found in males and about forty percent in females.

TERMINOLOGY

No definite agreement has been reached in regard to the exact meaning of subacute bacterial endocarditis. Libman(6) includes the so-called chronic ulcerative endocarditis, chronic malignant endocarditis, and endocarditis lenta. He does not consider the valvular defects of rheumatic or syphilitic infections as of the subacute type. Blumer(10) considers all protracted cases as subacute. Even the terminology is not constant, although in most localities it is known as subacute bacterial endocarditis. Some of the various terms which have
been used are:

1) Chronic ulcerative endocarditis - Reisman (41).
2) Subacute infectious endocarditis - Libman(6)(7).
3) Endocarditis lenta - Schottmeuller(8).
4) Chronic septic endocarditis - Heubner(42)
5) Chronic streptococcic endocarditis - Litten(43)
6) Chronic malignant endocarditis - Weber(44).

Kerlin(2) makes objections to the terms in part in that certain of them give an erroneous idea to the pathology, etiology or character. He even states that one of them is entirely false and is in fact an exact opposite of the type of growth found within the heart. He objects to malignant since it throws no light upon the etiology or pathology; to septic, since in the disease there is no pus formation; to ulcerative, because the pathological lesion is not ulcerative but proliferative; to infectious, because in that terminology would be included rheumatic disease and the endocarditis of syphilis.

SYMPTOMATOLOGY

In nearly all cases the disease begins insidiously and it is often difficult to determine with any accuracy the exact date of onset. The disease as a whole is very characteristic one, although each case, of course, shows its own variations. According to Libman's first report of cases(7), there is fever present in all cases. This is at times very low, while
at other periods it is quite high and intermittent. According to Kerlin(2) the temperature is usually not high, the average daily being about 102 degrees Fahrenheit. Waddell(4) believes that an unexplained fever of a low grade type is one of the most common initial symptoms. Along with the fever there is usually digestive disturbance, cardiac symptoms, and sweats. Sometimes the fever is accompanied by chills, at others by sweats. In still other cases there occur fever, chills and sweats in a regular sequence.

There is a progressive weakness and usually emaciation. The latter in most cases is a prominent feature, although in some cases the patient is well nourished up to the time of death. The spleen is usually enlarged and palpable, and generalized peripheral pains are quite constant. The latter in some cases are localized in bones, joints, muscles, tendons and ligaments. Others are purely neural according to Libman(7). He further adds that these pains at times may be due to embolic aneurysms. The painful erythematous nodules of Osler(1)(40), are, according to Libman almost pathognomonic(7). Of special interest is the symptom to which little attention was paid up until the time it was appreciated by Libman(7), is that of tenderness of the lower portion of the sternum. In his series of cases reported in that year(1910), nearly all of those patients were afflicted. He did not believe that this symptom was due to any anemic cause since it was present long before
the anemia was great enough to account for it.

Respiratory symptoms and signs occur in about 75 percent of all cases reported according to Hurxthal (11). These he said are explainable in some instances by splenic infarct, with or without a perisplenitis accompanying. Such splenic involvement gives rise to low chest pain, especially upon breathing. In other cases the pain may be due to a mitral stenosis or a weakened myocardium, while in a few cases pulmonary infarction of embolism may be the cause, since in certain cases which complained of this symptom before death, such pathology was found at autopsy. Long before death, however, these symptoms develop which cannot be explained by these factors alone. Since the majority of lesions are of the left heart, there may be still another source of explanation. Miller (12) in 1925 pointed out the relation of the bronchial arteries to the bronchial tree, and indicated the possibility of infarction of these vessels. Hurxthal (11) proposes the theory that there may be numerous emboli, too small to produce gross infarction, but large enough to plug up these smaller bronchial vessels and thereby set up sufficient local disturbance to give rise in part to the frequency of pulmonary signs and symptoms not explainable on any other basis.

Pulmonary tuberculosis, which is sometimes an early diagnosis of this disease, can be ruled out by the use of the X-ray
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studies of the chest, although the clinical presence of a rheumatic heart lesion should make one skeptical of the diagnosis of tuberculosis. (11)

Chronic lung infections of an unknown etiology such as empyema, bronchiectasis, and subphrenic abscess have occasionally been the preliminary diagnosis, when the real lesion was an endocarditis. Since clubbing of the fingers may occur in any of these conditions, the case may be unusually misleading. In Hurxthal's series, clubbing of the fingers did not occur without a palpable spleen, except in one case. Clubbing of the fingers seems to be a later development than splenomegaly. (11) Dyspnea is a common complaint. (2)

Cardiac symptoms and signs. The patient usually shows some evidence of organic or congenital valvular disease, but that this may not always be the case was pointed out by Blumer (10). Since the diagnosis depends essentially upon the evidence of embolic manifestations, all diagnoses that are to be antemortem can be so made without examination of the heart. In other words, although heart findings are suggestive, the absence of evidence of valvular disease of the heart should not rule out the possibility of subacute bacterial endocarditis being present.

Pain in the shoulder and precordium occur at times. Libman (6) pointed out that infarction of the spleen with
diaphragmatic irritation was possibly responsible for this. Hurxthal (11) was profoundly impressed by the presence of aortic lesions found at autopsy on patients who had complained of precordial pain before death. This was true in nine out of ten cases. Both of these observers are of the opinion that an acute pericarditis is the result of infarction of the heart, or a nearby pneumonic lesion, or in association with a terminal nephritis. Hurxthal found either an acute or chronic pericarditis present in about 25 percent of cases. In a few instances the latter observer made electrocardiographic studies of patients. From these he found that a normal rhythm was always present and the only occasional abnormalities which he found were right axis deviation, low T-2 waves, and a lengthening of the P-R interval. The pulse is usually thought to be accelerated. Hurxthal's cases showed periods of several days duration in which the pulse rate was entirely normal, even in the presence of fever. Such occurrences may lead the observer to doubt the diagnosis, or even worse, give the patient false hopes of recovery. Kerlin observes that the pulse rate is usually entirely normal except in the very late stages of the disease.(2)

Abdominal pain and splenomegaly. Abdominal pain occurs in about seventeen percent of all cases according to one observer(11), and is usually confined to the upper quadrants. The possibility of an erroneous diagnosis in this phase is
clear, especially when the pain is accompanied by fever, tenderness, muscles spasm and leucocytosis. The pathologic-al syndromes of appendicitis, gall stone colic, or renal calculus may be simulated. As the pain is usually the result of an embolus, the characteristic feature is the abruptness of onset. Thus, renal calculus is apt to be the preliminary diagnosis, since it often produces a similar sudden, severe pain.

The lesion of the kidney were first described by Baehr (13), and according to his investigations he found that there was an associated glomerulo-nephritis in about one-third, and renal infarction in about one-half of cases. The renal function is usually little impaired and the phenolsulphonphthalein test shows a decrease in only a very small percentage, and it is only in these cases with a lowered kidney function that there is found an increase in the blood non-protein nitrogen. The specific gravity of the urine varies normally in this disease. In about one-third of the cases edema appears, but it is not known, or at least not stated, whether or not it is of the dependent type.

Hypertension of any magnitude like that found in chronic glomerulo-nephritis is not found in subacute bacterial endocarditis. Usually if patients have a blood pressure of over 150 millimeters of mercury systolic, there is also present an
aortic regurgitation with a low diastolic pressure.

To quote Hurxthal(11), he states that, "It seems that in most cases, whether bacteria free cultures are found or not, there is enough clinical evidence of the disease ante-mortem so that differentiation of embolic nephritis of sub-acute bacterial endocarditis form chronic glomerulo-nephritis should not be difficult, even though in the latter disease, valvular lesions are not uncommon."

Libman(7) observed that hematuria was quite common in his series, and Waddell(4) noted that in all cases which came to autopsy there was found what he stated as "some form of glomerulo-nephritis."

Symptoms and signs due to involvement of the Central Nervous System. One of the most interesting phase of this disease is the complexity of neurological pictures that may be present. The insidious onset in most cases explains why cerebral manifestations may be the first thing noted by the patient, and thus mask the real cause of the trouble. Such clinical pictures as meningitis, brain abscess of tumor, with choked discs and hemiplegia or other paralyses may present themselves. Most characteristic is the presence of a high white blood cell count in the spinal fluid with no organisms or growth on culture. In all probability this is the reaction to infarction, a form of aseptic meningitis, of a kind not
infrequently associated with sterile brain abscess.

A curious cerebral symptom is vertigo. (6) It usually occurs when the patient arises or after exertion. In addition there is headache, irritability and insomnia which are followed later by stupor, delerium and coma. There may be hemorrhages into the brain, ventricles, or subarachnoid spaces which are the result of rupture of embolic aneurysm. At times the patient may be seen to have severe head pain, neck rigidity, hyperesthesia and, in some cases, a positive Kernig's sign. There may be paralyses of the intrinsic or extrinsic muscles of the eye. In the case of cerebral hemorrhage, it is not necessarily assumed that the patient will die from this, since at times the patient recovers from one hemorrhage, only for it to recur again at some later date, or the patient may suffer only one such attack and finally die from some other cause. The lumbar puncture in such cases reveals a bloody spinal fluid and an increase in the number of lymphocytes. Of course the most serious complication involving the central nervous system is the lodgement in the cerebral vessels of disrupted portions of the valvular vegetations which act as emboli. The latter condition is often the cause of the termination of the disease, which naturally assumes the death of the patient.

Blood findings. An odd feature of the disease is the fact that the white blood cells are apt to be extremely
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variable. This point, according to Hurxthal (11), is apt to be in some degree diagnostic. The majority of cases show a leucocytosis at one time or another. Leukopenia occurs at times but it is rarely continuous. Libman (7) stated that a very high leucocytosis was found only in advanced cases or in those with secondary lesions. Kerlin (2) varied this point in that his observations disclosed the fact that throughout the disease there is a progressive increase or decrease, or both, of the number of white blood cells present. According to one author (11) there may be a marked variation in the number of leucocytes from day to day with the sudden appearance of macrophages. In some of this authors cases marked differences were noted in the number and character of white blood cells found in samples of blood taken simultaneously from different sites. According to Cecil (5), the average white blood cell count is from twelve to eighteen thousand and that of the red blood cells about three million.

Macrophages were first reported in this disease by Van Nuys in 1907 (14). At the time of the discovery of these cellular elements of the blood their significance was not appreciated. The origin of these cells is not exactly clear, although there are theories advanced as to their site of formation. Some investigators believe them to be of the same origin as the endothelial leucocytes, that is, in the endothelial surfaces. The macrophage is from two to ten times larger than
the large mononuclear cell of normal blood. The presence of these cells may be observed at times in the blood during other infections. When found they are usually present in large numbers in subacute bacterial endocarditis, and they present histologically a somewhat similar appearance to the large mononuclear, except when pseudopodia, vacuolization, or ingestion of polymorphonuclear leucocytes or red blood cells are present. On the other hand, there are increases in the number of large mononuclear cells which are indistinguishable from those of normal blood. This condition may be observed in the blood during other infections. These latter cells also may may at times show ingested red blood cells or vacuolization. Under these circumstances these cells are then known as endothelial phagocytes as a distinction from macrophages. It is of the opinion of Van Nuys (14) and Hurxthall (11) that both forms are somewhat important evidences of diagnosis. They believe that the macrophage bears more diagnostic weight than the endothelial phagocyte. Hurxthall in addition states that he believes the macrophage when found in the blood is pathognomonic of bacterial endocarditis, and that the finding of endothelial phagocytes is highly suggestive of the disease.

In addition to the above changes in the blood picture of this disease, there is marked secondary anemia usually, with an abundance of platelets, but with seldom any alteration in
the bleeding or coagulation times. The anemia in some cases is so marked that the disease at times is mistaken for pernicious anemia (2). Libman injects the thought that a progressive anemia accompanied by a waxy, dirty facies is a noteworthy feature of the condition.

Acute arthritis occurs in about thirty percent of cases. Whether or not this condition is of rheumatic origin can be differentiated by the use of salicylates (3). If the latter produce no effect in a suspected case of rheumatic disease, one should begin to think of subacute bacterial endocarditis. Libman (6) noted that joint swelling was not an infrequent symptom and that patients often complained of arthritis and periartritic pain. The cause of these pains is not well known but Libman believes them due mostly to the accompanying anemia, although at times they may be explained upon the basis of embolic aneurysm. (7)

Edema may be found at times early, but in most instances it appears later in the course of the disease. It is usually due to cardiac weakness and partly to the anemia, according to Libman. Baehr (13) reported the presence of edema in about one-third of his cases but offered no definite explanation of it, nor did he declare whether or not he considered it dependent. According to Libman, the edema is usually of a soft type, but if thromboses are present it is apt to be firmer.
A generalized edema is more often due to the anemia than to a nephritis. Libman found that in the influenzal type of the disease, the edema is more apt to be of a nephritic origin. At times the picture may be accompanied by gangrene, undoubtedly due to blocking of peripheral vessels with emboli.

Clubbing of the fingers is quite frequent and its recognition early is often quite puzzling. Opinions as to the exact presence of clubbing are often apt to disagree. Hurxthal (11) suggests that a good point in determining the presence or absence of this condition is the formation of a rim of fine, new, pinkish skin around the margin of the nail, along with tenderness of the tips of the fingers.

Skin manifestations. Petechiae occur in about fifty percent of cases as reported by one author.(11) According to him the diagnostic importance of these should be held in doubt until the definite etiological factors have been proven. The exact nature of these lesions has yet to be established pathologically. Lewis and Hamer have shown that in subacute bacterial endocarditis there is injury to the walls of the small blood vessels and that petechiae may be produced easily by an increase of venous pressure.(45) This would seem to indicate that a good many lesions are purely purpuric and therefore are not especially indicative of endocarditis. Of these various locations of petechiae, Hurxthal believes that the
conjunctival form is the most reliable sign. These are known to be embolic in origin, not petechial, as is shown by the fact that pigment has not been observed after the erythema has subsided. The next most reliable and consistent finding are the small pinkish or reddish cutaneous macules from two to four millimeters in diameter. These lesions blanch on pressure at first, but later may or may not leave a small area of fawn-colored pigment. They are found most commonly on the palms, fingers, soles and toes and are probably embolic in origin. Osler's nodes are not so frequent as is most commonly believed. However, Libman almost contradicts the latter statement with the belief that these above mentioned nodules are almost pathognomonic. Embolic plugs of the retina are not uncommon and purpura, tender erythematous nodules and various types of skin eruptions have been reported. To quote Hurxthal, "Unless conjunctival lesions or Osler's nodes are found, little emphasis should be laid upon the skin lesions of subacute bacterial endocarditis." 

Ocular symptoms. Most commonly the ocular changes in this disease are due to the presence of petechiae, or occasionally, hemorrhages into the fundus. In certain instances one may observe white or yellowish-white spots in the fundus. These, however, occur at times in any general infectious diseases and are known as Roth's spots. They are not often found in this disease. Amblyopia or central scotomata may occur in
the event of embolism and furthermore there may be a strabismus if the eye muscles are involved. If optic neuritis occurs, it is usually bilateral and is perhaps toxic in origin.

One of the most consistent findings occurs in the form of gastrointestinal disorders. There is usually loss of appetite, accompanied by flatulency and constipation. (2) Diarrhea is not infrequent and vomiting is troublesome at times. All these are not definitely explainable, but may be assumed to be on the basis of irritation from embolic processes along the gastro-intestinal tract.

Generally speaking the sequence of events of the disease are about as follows. The organisms enter the blood stream at points of infected foci such as teeth, tonsils, otitis media, etc. These foci, since this organism is one of the most common found in the mouth and is of low virulence, are probably the primary source of this type of endocarditis. After entering the general circulation, the organisms lodge on a previously disease or damaged valve, which is usually the aortic or mitral. Here, then, are formed grayish masses composed of fibrin, red blood cells and white blood cells or platelets, and some epithelial cells. The organisms then grow and multiply in the vegetations, which in turn become larger. As a result of the enlargement, small portions of them are brushed off by the rush of blood through the heart. These small particles, when brushed off, circulate freely in
the blood stream, and there they then become potential sources of trouble in that they are emboli. This phenomenon occurs at regular intervals throughout the disease.

The course of the disease, as a rule, is not progressively downward as there are remissions frequently. However, it is only with but few exceptions that the disease ends fatally. Of 281 cases reviewed by Kerlin, death occurred between the end of the third and eighth weeks. Out of 212, or 75 percent, only nine lived over sixteen months and only six of these lived over two years. (2)

DIAGNOSIS

According to Cecil(5)," During any obscure fever which is accompanied by heart murmurs, one should obtain blood cultures repeatedly." This, in subacute bacterial endocarditis, is, above all, the most effective diagnostic procedure. When signs of petechiae or infarcts appear, the diagnosis clinically is relatively easy. This disease must be sharply differentiated from acute rheumatic endocarditis, which comes on more suddenly and with more marked heart symptoms and in conjunction with a bacteria-free blood culture; from an ulcerative endocarditis, which is part of an acute pyemia, to which the heart lesion is secondary. There is usually a primary focus which is easily distinguishable and the blood culture is positive for the Staphylococcus, Streptococcus pyogenes, or the pneumococcus. The duration is only a few days or weeks, and is almost always fatal. The lesions of the heart are
found to be ulcerative and not vegetative. The disease must also be differentiated from the endocarditis which results as a terminal event in chronic diseases. The last should not give trouble in a differential diagnosis.4 According to Cecil, certain cases of acute rheumatic endocarditis show a blood culture positive for Streptococcus viridans. However, if the culture is repeated, it is usually found to be negative. In addition, the course of the disease with the usual recovery of the patient confirms the distinction. Furthermore, the signs of myocardial affection are not as pronounced in subacute bacterial endocarditis as in acute rheumatic endocarditis.

PROGNOSIS

All observers give a very bad prognosis to the disease. The multiplicity of symptoms at times so mask the disease that the physician may be misled early in the disease and, mistaking the diagnosis, may give the patient and relatives false hopes of recovery. To exemplify the statistics compiled by a few observers as to the matter of prognosis, one might mention a few reports. Osler(40), in 1909 reported ten cases with a total of ten deaths. Lenhartz(15), in 1904, saw sixteen cases with sixteen deaths. Billings(16), between the years 1909 and 1910 occasioned fourteen cases with one recovery and thirteen deaths. Harber(17), in 1920, reported 150 cases with one recovery and 149 deaths. And Libman(6), between 1912 and 1913 encountered 150 cases with four cases recovering and
with 146 fatalities. These are only the results obtained by a few observers, but it can be readily seen from them that the outlook on the disease is very poor.

PROPHYLAXIS

Obviously, in order to avoid the possibility of affliction by the disease, all patients with known valvular heart lesions should take the utmost care and precautions to prevent infections of the teeth, tonsils, female generative organs, etc. By all means, potential foci of infection should be removed as early as possible.

CONCLUSIONS

Death occurring from cardiac failure before age forty is practically always due to, or a result of, valvular injury.

Endocarditis associated immediately with acute rheumatic disease seldom causes immediate death. If so, it is usually the result of percardial infection or cardiac failure.

Embolic processes are common in all forms of bacterial endocarditis. These processes are most frequent in the subacute form and probably can be accounted for by the longer duration of the disease. Embolic processes do not seem to occur in acute rheumatic endocarditis.

Embollic glomerulo-nephritis is rarely found in any form of endocarditis except the subacute bacterial type, in which it occurs in a fairly high percentage of cases. Its occurrence seems to bear no relationship to other forms of embolic processes.
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The chief factor in determining the presence of the disease seems to lie in the isolation and culture of organisms of low virulence in the bloodstream and from the vegetations on the heart valves. *Streptococcus viridans* is the usual organism, but the *Bacillus influenzae* may be the cause. In some cases it may be due to the Hemolytic *streptococcus*.(38)

The presence of embolic processes in cases with healed defective valves suggests that bacterial forms of endocarditis not infrequently heal, since in rheumatic endocarditis embolic processes are not found.

Splenic enlargement which is palpable, associated with cardiac trouble, generally indicates an active inflammatory condition of the valves, rather than an enlargement from passive congestion.

Active involvement of auricular and ventricular endocardium rarely occurs except in subacute bacterial endocarditis. Valvular ulceration is practically never found in this disease and the term, Ulcerative endocarditis should not be used to indicate the presence of embolic processes, for the presence of the latter bears no relationship to ulcerated valves.

Previously injured valves are apparently more susceptible to infection that normal valves. In most cases they seem to have resulted from rheumatic infection, but they may be produced by the healing of bacterially infected valves.

Myocarditis and pericarditis are seldom associated with
bacterial endocarditis.

Rheumatic infection commonly precedes subacute bacterial endocarditis, but usually not until some free interval has elapsed.

The finding of the streptococcus viridans in the blood in a case of endocarditis indicates a subacute bacterial form. This is also true of the Bacillus influenzae.

Apparently the two main factors to keep in mind in interpreting variations in the findings in subacute bacterial endocarditis are the duration of the process and the virulence of the causative organism. The former seems to be dependent upon the latter.
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