The second reason which should render wounds of the type ordinarily received during surgical operations and post-mortem examinations specially prone to septic involvement, is the fact that they are likely to present a maximum of virulence, in conjunction with the important condition of a minimum of mechanical or chemical trauma. Trauma elicits inflammation, which is substantially the sum of those actions by which the animal organism brings into play its defensive apparatus against microbial or other harmful invasion. Diapedesis, serous effusion, fever, and pain (inducing immobilization of the affected part) are defensive means spontaneously brought into play in the presence of any extensive trauma; all these are far less active in the case of minute wounds, than in that of injuries of greater magnitude. The relation between the extent of the local reaction at the point of inoculation on the one hand, and the duration of life and number of organisms present in the blood on the other, have long been recognized in the experimental septicæmias of the lower animals.[10] Just as we find that many of the defensive proteids, the so-called "phylaxins" (the mycophylaxins and the toxophylaxins) are not manufactured by the animal organism except in the presence of bacterial onslaughts, so it is not unreasonable to suppose that certain of the "offensive proteids" of the invading microbes may not be manufactured by them, except in the face of opposition to their growth exhibited by the defensive activity of the animal cells, and that when these "offensive proteids" are not thrown out, a defensive chemotaxis is less actively evoked and the virulent bacteria, insidiously implanted, may continue to multiply unopposed, until they have acquired a reserve power for attack which they might never have attained had their inoculation been attended with more extensive traumatism.

(*b*) *The Condition of the Constitution which Favors Spreading of the Infection.*—The question of the condition of the constitution which favors the spreading of the infection in dissection and operation wounds is most intimately connected with the doctrine of mixed infections. Aside from the well-known fact that a generally debilitated condition of the body, such as is produced by insufficiency in food and rest, protracted anxiety and nervous strain, and prolonged exposure to cold and other influences more or less powerfully depressing to the resisting forces, and the coexistence of such disease as diabetes, syphilis, alcoholism, anæmia, obesity, typhoid and other fevers, Bright's disease, etc., some of which have been shown, upon most conclusive clinical evidence, to be predisposing causes of infection with the pyogenic cocci, we must give proper weight to the conditions of enhanced virulence that may develop as the result of the concurrent development in the tissues of two distinct forms of bacteria. Whereas this association may be without special influence as regards infection, yet more frequently the concurrent inoculation of two species of bacteria increases the danger from one or both. "A combination of two species, each in itself harmless, may prove fatal" (Roger). A bacterium of attenuated virulence may become augmented in virulence by inoculation in combination with another species, which need not necessarily be itself pathogenic, or sometimes simply in combination with the *chemical products* of another species.[11] Nor is it essential to the appearance of enhanced evil effects that the inoculation of such independent growths be simultaneous. There is no lack of clinical cases to illustrate the occurrence of severe pyogenic mixed infections, in which, in one set of cases, the pyogenic process was latent or in abeyance, and was roused to extreme or even fatal activity by the admixture of a fresh, non-pyogenic infection; and in another set of cases, a previously latent non-pyogenic infection, when later mingled with an infection of pyogenic type, gave rise to the gravest manifestations of sepsis. The influence, in this regard, of epidemic influenza has been recorded by Bennett, in four cases, which we can but briefly glance at:[12]

Case I. Simple synovitis of the knee, almost well, influenza, suppuration, pyæmia, amputation.

Case II. Cystitis, improved, urine suddenly excessively fœtid, after forty-eight hours of influenza, obstinate cystitis, pyæmia.

Case III. Patient apparently convalescent from influenza noticed swelling and redness about a small crack in lower lip. Erysipelatous inflammation followed, with abscess over right side of face and throat. A few days later, pneumonia and secondary pyæmic deposits over right knee and left thigh, followed by sudden filling of left knee joint.

Case IV. Urethral stricture, facial herpes followed by influenza, erysipelatous inflammation, pyæmia.

The influence, on the other hand, of a pre-existing stercoræmia, a "bad cold," or other mild, non-pyogenic infection, as a cause predisposing to pyogenic infection, is so generally recognized as to make any more specific citation of cases superfluous.

Now how frequently is the surgeon or the pathologist obliged to continue his work when himself suffering from many such mild ailments, yet how considerable a bearing they may have on the outcome of an accidental inoculation with pyogenic germs during operation or autopsy is scarcely appreciated until the unexpected happens, and a trifling injury is seen to take on all the aspects of a virulent local, or even general, infection.

(*c*) *The Various Forms of Internal Resistance Offered to the Advancement of the Microbes.*—It is not within the scope of this article to elucidate the whole pathogenesis of pyogenic infection, yet a proper understanding of the dire results which may follow upon a slight dissection or operation wound demands that we should at least briefly follow the history of the microbes which have been inoculated with the receipt of the wound.

The "cytomachia" which follows the introduction of pathogenic bacteria among the constituent cells of the animal or human economy, is but a single, limited, yet intensely interesting phase of the general biologic phenomenon of the struggle for existence. In one sense, indeed, it is the struggle of the individual animal, as a whole, against an opposing entity, the invading disease; yet in a sense equally true, but only appreciable to us since the recent advances of scientific investigation, it is the struggle of a countless number of individuals of an invading host, against countless numbers of individual cells acting on the defensive. Looked at from this latter point of view, it is a struggle of cell against cell and a crossing of cell weapon with cell weapon. The invading hosts are the pathogenic bacteria, and their weapons are, what I have ventured to call, the "offensive proteids." These "offensive proteids" have been ascertained to be of two kinds, the toxins, and certain proteolytic substances (ferments?), capable of causing the death and disintegration of the body cells, but yielded up only at the death of the individual bacteria. The defending hosts are the militant cells of the animal body, chiefly the leucocytes and certain comparatively undifferentiated connective-tissue cells, the so-called "Schlummerzellen," and the defensive proteids are of two kinds, the antitoxic proteids, directed against the "offensive proteids," *i.e.*, against the toxins (antitoxins); and the proteolytic (antimycotic) proteids (bacteriolysins), directed against the opposing bacteria, and capable, under favorable circumstances, of causing their death and disintegration. Besides this "long-distance" combat, the analogy with human warfare is still further borne out by the actual grappling of individual cell with individual bacterium. In some cases this represents the attack of the bacterium on the leucocyte (*e.g.*, the gonococcus); more often it represents the attack of the leucocyte on the bacterium, and the phenomenon is then known as phagocytosis.

In the conflict between animal cells and bacteria all these factors are simultaneously or successively brought into play, and certain forces prevail and predominate over other forces, until victory, at least temporarily, rests with one party or with the other.

According to the relative predominance of one or more of the embattled forces we have one or another pathological picture before us. One of the earliest and the

commonest pathological phenomena which are observed to follow the invasion of the animal body by pathogenic germs, is active or inflammatory œdema. This is the result of what is known as chemotaxis, the marshalling of the forces of the defence: the leucocytes in vast numbers assemble on the field of battle to engage the invading bacteria.

Another common phenomenon is localized suppuration and abscess formation. Here the leucocytes have not only assembled but have actually engaged the bacteria, and with the death of their foemen, or of members of their own forces, there have been liberated the proteolytic substances which have caused the liquefaction of the surrounding tissues.

Though the pus within an abscess cavity may swarm with bacteria, yet not infrequently bacteria in such pus are few, or may be altogether absent, *i.e.*, the pus is "sterile" (suppurating buboes, tuberculous abscesses, gonorrhœal salpingitis, empyemic exudates). In the first case the abscess has been more or less immediately caused by the bacteria, and they have "slain their thousands" among the leucocytes, the bodies of the defunct leucocytes have become dissolved through the action of the proteolytic ferments, and only sparse, disembodied nuclei, but faintly dyed by staining reagents, are to be found in the fluid; such pus is thin and watery, it is spoken of as "ichorous" pus, and has long been recognized as "acrid," "erosive," and "virulent," *i.e.*, it contains many victorious bacteria. In the second case, on the other hand, the suppuration has been evoked more or less immediately by the leucocytes, which by an exaggerated chemotaxis have crowded about the invading bacteria and have overcome them, some by phagocytosis, some doubtless by means of the antimycotic proteids (mycosozins, mycophylaxins, bacteriolysins); such pus is thick and creamy, contains plump, distinctly nucleated "pus cells" (polynuclear leucocytes) and is known as "laudable pus," and recognized as far less virulent and acrid, *i.e.*, it contains few (sometimes none at all) debilitated and attenuated bacteria.

Another phenomenon occasioned by the invasion of the tissues with pathogenic bacteria is more or less extensive necrobiosis and sloughing. Here is a case in which the toxic influences of the bacteria have been locally exercised to a more extensive degree than the leucocytes immediately engaged have been able to cope with, and larger or smaller masses of the less energetically vitalized tissues, in the neighborhood of the focus of invasion, have been literally poisoned to death by the concentrated toxins. This is generally, but not always, accompanied by more or less suppuration of either the first or the second type; we deem it to be of the first type when the sloughing is progressive (necrosis progrediens); of the second more benign type when the sloughing is not progressive, but is rather the benign effort of nature to expel useless tissue by a process, as it were, of spontaneous amputation.

Again, in another case, the best efforts of the defending leucocytes are barely equal to the task of holding in check the local development of the bacteria, but the latter nevertheless produce their toxins in such quantity that the antitoxins of the animal cells (the toxosozins and toxophylaxins) are no longer sufficient completely to neutralize them, the unneutralized excess of bacterial toxins circulates in the blood serum, and the profound intoxication, which we saw involve the death of vulnerable tissues in the immediate neighborhood of the focus of invasion, exerts a similar, but happily generally a less intense, effect upon all the tissues of the organism; toxæmia is the result, with headache, vomiting, general malaise, and the system generally reacts against the wholesale poisoning of its tissues, with fever; yet in the more malignant intoxications such widespread and profound intoxication of the tissues may be produced that the individual succumbs at once, without any reactionary fever, without suppuration, sometimes even before marshalling of the forces of the defence by chemotaxis, "septicémie foudroyante"; the whole drama is ended in a few days or even a few hours, with a normal or even a subnormal temperature, and without reaction to the overwhelming dose of the toxins.

A still higher triumph of the invading bacteria over the defences of the body is manifested when the former not only send their unneutralized toxins coursing through the veins, but themselves escape bodily through the second barrier of defence constituted by the lymph channels and lymph glands, and, mingling with the blood corpuscles in the blood-stream, continue to grow there, and in the very bosom of the life fluid itself multiply their numbers and evolve their noxious products. This condition, that of septicæmia *strictiori sensu*, is most typically developed in the specific experimental septicæmias of the lower animals, in which, after the inoculation of the specific virulent germs, these are presently found swarming in countless numbers in the blood of the victims. In this aggravated form this invasion of the blood with pyogenic bacteria is rarely, though indeed occasionally, seen in man (*vide infra*, Case IV.), but in the human subject analogous symptoms are developed when even a few pyogenic cocci are discernible in the blood after careful examination. It would seem that bacteria having sufficient vitality to establish themselves and multiply within the blood are nearly beyond the reach of effective attack by the animal defences, and cases in which pyogenic cocci, especially streptococci, have been demonstrated in the circulating blood must be regarded as having a very grave prognosis, unless extraordinary aid can be afforded to the defensive apparatus of the patient. Nevertheless, battle is sometimes offered the pyogenic bacteria even within the blood-vessels, and the severe chills and enormous excursus of the temperature curve indicate the desperate character of the conflict waged by the defensive cells. The cells die in multitudes and in expiring bring into play their most powerful weapon, the antimycotic proteids (the mycosozins and the mycophylaxins and bacteriolysins). Now along with the leucocytes (the militant cells of the blood) numbers of the red blood corpuscles also undoubtedly succumb in conditions of severe sepsis, and a fibrinogenous substance, metaglobulin,[13] is developed, a product of the disintegration of cell protoplasm, which has the attribute of inducing coagulation of the blood and thrombus formation. In these thrombi the battle is renewed much as it was first fought in the connective tissues, and each separate thrombus becomes immediately invaded with septic germs, and becomes converted into a suppurating focus, a pyæmic abscess. As the invading bacteria successively invade each new thrombus, and cast a fresh load of toxins upon the staggering forces of the patient, the event is heralded with a chill, the characteristic phenomenon of a pronounced pyæmia. The struggle is now continued for a time, at an almost hopeless disadvantage, until death, either from exhaustion or in profound septic intoxication, closes the scene.

Leaving for other articles in this work to give the further details of the process of septic infection which we have thus broadly sketched, it must suffice us, in considering the development of dissection and operation wounds, to familiarize ourselves with the following general conclusions of Marmorek in regard to them, as a limited sub-class of general or local septic infections:

"Not only is it to be considered that all those diseases which are classed as septic are etiologically one and the same, and that one and the same bacterium can produce all of its forms, but we must also recognize that even the clinical pictures which they afford are capable of full and complete interpretation from the same etiological standpoint. All the manifold disease forms are simply the consequence of that variability of the different factors involved which the constantly changing relations between the invading forces of disease, on the one hand, and the resisting forces of the organism, on the other hand, occasion. Those conditions, which writers speak of as 'septic intoxication,' 'infection,' 'mycosis,' and pyæmia, are naught else but the results of infection, principally by one particular microbe, the Streptococcus pyogenes,

and only occasionally by the Staphylococcus pyogenes aureus."

"Erysipelas, phlegmons, and carbuncles are forms of septic infection where the conflict is fought within the first system of defence of the invaded organism; in lymphangitis and lymphadenitis the conflict lies within the second system of defence; and septicæmia and pyæmia are evidences of conflict within the third and last means of animal defence, the blood itself."[14]

"When bacteria, possessed in a high degree of the faculty of manufacturing toxins, gain access to the organism, or when bacteria find within the tissues of their host conditions favorable to the abundant production of toxins, then the result of their invasion will be a toxæmia or a septicæmia; when these conditions favoring abundant production of toxins are wanting, the bacteria, provided they are nevertheless capable of maintaining and multiplying themselves, can work their noxious effects upon the organism only through the means of those proteïds which are set free by the death of individuals among them, *i.e.*, the proteolytic ferments; the result is a pyæmia. Inasmuch, however, as in most cases toxin production and proteolytic-ferment production proceed side by side, a sharp line can never be drawn between toxæmia, septicæmia, and pyæmia." (Sitzmann.[15])

(d) The Means of Eliminating the Microbes and their Toxins from the System.—The active struggle between the invading bacteria and the defending body cells may have any one of three terminations: The active struggle may degenerate into a quasi "guerilla warfare," finding its expression in a tedious succession of smaller disease processes, the sequelæ of the disease inaugurated by the primary infection; or, the conflict may terminate in a truce, as it were, of more or less prolonged duration; or, the victory of one side or of the other may be complete, and either the infected individual succumb in death, in the one case, or the bacteria be completely routed and driven forth, in the other. We will now briefly consider these three possibilities.

The condition which we have considered as analogous to a "guerilla warfare" is one in which the septic process does not actually subside entirely, but either is continued, in a more mild type, at the original site of infection, or manifests itself in constantly recurring furuncles, abscesses, or other septic processes in the same general neighborhood, or in some cases indeed at more remote points, yet in a manner to be distinguished from pyæmia, both by the mildness of the processes and by their local and regionary distribution; the type of the struggle differs chiefly in degree from that of the severer forms of sepsis described in the preceding section.

The condition which we have described as analogous to a truce is one which has received careful study at the hands of many distinguished bacteriologists, notably by Halban[16] and by Perez.[17] The conclusions of Perez are quite in consonance with the careful experiments of Wauters,[18] who believes, contrary to the commonly received impression, that the bactericidal properties of the lymph glands are relatively small, as compared with those possessed by some other tissues of the body, notably by the red bone marrow. The observations of Perez are likewise in accord with those of the investigators who, with Ehrlich,[19] maintain an essential distinction between the different classes of leucocytes, distinguishing between the "myeloblasts," possessed of marked phagocytic abilities, and the "lymphoblasts," possessing this quality but in very slight degree, if at all. The observations and experiments of Perez lead him to the belief that it is the function of the lymph glands to detain the pathogenic bacteria in their meshes, and there, not annihilate them as do some of the more actively bactericidal tissues, but rather expose them to the peculiar influence of the lymphoblasts, which consists rather in robbing them of their pathogenic qualities than in compassing their entire destruction. An intermission in the conflict between invading bacteria and resisting body cells occurs when the previously active bacteria are temporarily eliminated from the tissues "at large," to be gathered into the emasculating embrace of the lymph glands, from whence, possibly, they may from time to time emerge as unruly prisoners—somewhat shorn, indeed, of their most virulent characteristics—to induce a relapse of an apparently terminated warfare, and manifest themselves often in ways totally different clinically from the first onslaught of the infection; *e.g.*, what first manifested itself as a pyogenic process may, under the influence of the same bacteria, modified by their sojourn in the lymph glands, take on any of the types of non-purulent disturbance which we saw that the pyogenic germs were capable of inciting (*vide supra*), and appear as a neuralgia, as a catarrhal affection of a mucous membrane, or as a serous or sero-fibrinous serositis, of acute or chronic type. Thus, according to the results of the latest experimental research, do the lymph glands afford another form, quite *sui generis*, of protecting agency to the body in its conflict with infecting germs, and if unable to annihilate the invaders completely, conclude with the foe an advantageous armistice, at the close of which the body may resume the conflict under other and presumably more favorable terms.

The third and most decisive outcome of the struggle against infection is the complete destruction of the bacteria and their elimination from the system. This has likewise been long the object of closest observation and most ingenious experiment. Here again the earlier ideas of the excretion of the bacteria themselves by the emunctory organs of the body have had to give place before more recent and more exact studies, and the same is largely true of the elimination of the bacterial toxins. In regard to the elimination of the bacteria themselves, we have the authority of von Klecki,[20] corroborated by that of Cotton,[21] for the statement that though many kinds of bacteria can, indeed, be excreted by the normal bile passages in very small numbers, and probably but not certainly by the normal kidneys also,—this, however, only when they are present in large numbers in the blood,—yet their elimination from the body in this manner, in any considerable number, argues a lesion of the excretory organ in question. "In no wise," says Cotton most positively, "is the commonly received opinion to be accepted that the excretion of bacteria by the emunctories is to be regarded as part of the normal defence system (*Schützvorrichtung*) of the body." Goldberg likewise was unable to demonstrate the excretion of tetanotoxin by the kidneys, and his experiments serve to throw doubt upon the excretion of bacterial toxins, as such, from the body by any of the excretory organs.[22] We must look them for some other way which the body possesses of ridding itself first of the microbes, and secondly of their toxins.

Thanks to the studies of Nuttall,[23] Gamaleïa,[24] Denys, Sawtschenko,[25] and Pfeiffer, we are happily in a position to offer a reasonable opinion as to the method by which the system rids itself of bacteria when it has succeeded in extinguishing their vitality. Says Nuttall in regard to the anthrax bacilli: "In the bodies of bedbugs (and of other blood-sucking insects) they become less and less capable of becoming stained with methylene blue and finally altogether so"; "they are simply digested," "*werden einfach verdaut.*" According to Gamaleïa, it may now be regarded as settled that the bactericidal substances in the body (*vide supra*, the proteolytic ferments) dissolve the bacteria. To our eyes the process becomes first appreciable as a disappearance of the staining capacity (*i.e.*, a chromatolysis), in a word, a bacteriolysis. A variety of alkaloids, *e.g.*, caffeine, and a variety of ptomaïns, such as methyl- and ethylamin, have chromatolytic properties, as have a number of other bodies—among them, more particularly casein. On further investigation to determine which derivative of casein it is that occasions chromatolysis, it was found that the ammonia salt of glutaminic acid possessed this quality in marked degree. When a fluid in which chromatolysis has taken place is treated with acetic acid and filtered, and the residue on the filter paper dissolved with ammonia, we obtain bacteriolysin, which not only occasions chromatolysis, but totally disintegrates the bacteria, separates the rods (of anthrax bacilli), and causes these to fall to pieces, to

form but an amorphous mass of detritus. The thick, opaque emulsion of bacteria changes, under the action of this ferment, in the course of from six to twelve hours into a transparent fluid with scarcely apparent turbidity. From this last solution of bacteria the ferment can again be precipitated by means of acetic acid. The ferment which is obtained by treating anthrax bacilli with glutaminic acid has a bacteriolytic action only upon anthrax bacilli and upon no others. By the action of the casein derivative, or of an amido-acid, upon the cholera vibrio and upon the diphtheria bacillus, corresponding bacteriolysins are developed.

With these data before us, it is possible to understand fairly well how the pathogenic microbes are eliminated from the system. They are, as we have seen, actually dissolved and digested by the tissues, and it is probable that by this peptonizing action of the tissues their proteïds are split up, in a manner more or less remotely analogous to the splitting up of proteïd molecules in the process of gastro-intestinal digestion, until they are reduced to a chemical condition which permits them to be excreted by the ordinary emunctory channels, in a form more or less indistinguishable from the products of the disintegration of proteïd molecules derived from any other indifferent source. It is probable that the liver is largely concerned in rendering the dissolved bacteria fit for excretion, as we know it is influential in rendering innocuous other poisonous products.

As it is only under exceptional conditions that the bacteria themselves are eliminated as such by the kidneys and the other emunctories, so likewise it is only under exceptional conditions that the bacterial toxins are eliminated as such, and in recognizable form, by the emunctory organs. On the contrary, it is recognized, in the first place, that they are in great measure neutralized by the body antitoxins before their elimination (which would in itself account for the non-appearance of specific toxins in the urine, the fæces, and the sweat); and in the second place, it is more than likely that the neutralized toxins are further broken up by the action of the liver, and prepared thereby for elimination eventually by the ordinary emunctory channels in an undifferentiable form, along with the ordinary waste products of metabolism.

Having now considered in some detail first the etiology and then the pathogenesis of dissection and operation wounds, we have dealt as far as is necessary to our purpose with those characteristics of these affections which may be deemed to distinguish them from other septic processes; and it now remains for us to deal, only much less exhaustively, with the questions of their clinical course, their diagnosis, their prognosis, and their treatment, which are to all intents and purposes those of sepsis, from whatever cause arising, and are more fully treated under other headings in this work.

C. Clinical Course.—The clinical course of dissection and operation wounds can best be made plain by the citation of four characteristic and illustrative cases.

Case I. That of Dr. K. Zmigrodzki, of St. Petersburg.[26] The infection in this case was received from a patient who suffered primarily from facial erysipelas. The patient was dismissed from the hospital apparently well on July 7th, 1888, but was received into the hospital again (August 30th?) on account of an extensive phlegmon of the right scapular region. On September 5th, 1888, Dr. Z. made several incisions to void the pus, and packed and drained the wound. The patient recovered quickly, and left the hospital on September 22d.

In performing this operation on September 5th, Dr. Z. acquired, he does not know how, a purulent infection, perhaps through some slight abrasion. Already during the night following the operation he felt severe pain in the left thumb, near the nail, on the ulnar side. On the morning of the 6th, there was a developing panaritium subunguale; on the 7th, he himself injected cocaine and opened the beginning abscess and applied an antiseptic dressing. He did not feel the slightest doubt of a prompt recovery, which, however, did not follow; the wound did not heal but became dry and very sensitive, and occasioned some fever. Under cocaine, a colleague removed part of the nail and made two deeper cuts, but found no pus; an antiseptic dressing was applied.

Healing was now confidently expected, but instead, on September 20th, the patient suddenly had a severe chill, with pain in the left side of the chest along the margin of the ribs. Thinking this to be a colitis, the patient took a dose of castor oil and calomel, but experienced no relief. A careful examination by a colleague on the 23d revealed an affection of the left lung; in the upper lobe, near the linea axillaris anterior, there were dulness on percussion, increased vocal fremitus, pleuritic friction sounds, and a circumscribed patch of subcrepitant râles. The coughing was at first very slight, but on the third day the sputum raised was somewhat bloody, brownish in color, and consisted of lumps of inspissated mucus. After two or three more days, all the other signs of infiltration about this spot in the left lung had disappeared, with the exception of the râles, and the pains in the side ceased: the temperature fell quickly, and the sputa, which had become copious, came to consist of mucus only. From these clinical phenomena there was made a diagnosis of hemorrhagic infarct, of embolic origin, which was deemed to be closely related, etiologically, to the panaritium on the left hand, as this showed not the slightest indication of healing.

Says Dr. Z., who reports the case himself, "I lay especial emphasis on the fact that there was absolutely no appearance of the advance of the inflammation beyond the distal phalanx of the thumb, neither at this time nor during the whole course of my disease; absolutely no lymphangitis, erysipelas, or lymphadenitis, the customary accompaniments of septico-pyæmic infections, was at any time present."

After a few days, a second infarct made its appearance, under strictly analogous circumstances, followed by a third, a fourth, a fifth, and a sixth in the course of the next three or four weeks, and all in the same left lung. The consultants called at this time confirmed the diagnosis of pyæmic infarcts and offered an unfavorable prognosis, *quoad vitam*. The treatment was symptomatic, with special attention to the preservation of the general strength and the maintenance of the heart function. The radial pulse throughout the whole sickness remained strong, though the patient refused alcoholic stimulation.

At this time there developed a circumscribed phlebitis above the inner malleoli of both feet (phlebitis venarum saphenarum interiorarum). This manifested itself as a livid, excessively tender swelling of the skin, which imparted to the examining finger a "doughy" feeling, if not an actual sensation of fluctuation. Under bichloride dressings this quickly subsided, leaving only some pigmentation behind.

"On October 17th, the temperature, which had all along been very high, dropped from 100.4° F. to normal, and, in spite of the dubious prognosis, I began slowly to improve, my appetite returned, and on October 31st, that is, on the forty-second day of my illness (counting from September 20th, the day of the chill), I left my bed. After about a week and a half I went out for the first time, and my strength slowly returned. I still felt constantly dull pains in the spinous processes of the lumbar vertebræ, specially after long-continued sitting; to this, however, I paid but little attention.

"One day, on getting out of a carriage, I felt suddenly a severe pain, like a sprain, in the outer malleolus of the left foot; from that moment on pain, of a rheumatic type, persisted for a long time, extending along the whole of the left lower leg. After a couple of days there developed an evident periosteal swelling on the anterior aspect of the upper third of the left tibia, below the tuberosity, and another over the lower end of the left fibula, on the posterior aspect of the leg. I did not wish to resume my bed, and so kept about, applying hot applications from time to time.

"As I had no attacks of feverishness, I made no observations of the temperature from October 31st till November 10th. On November 11th I was again obliged to