public clinics, sanatoria, and the dissemination of information as to the means of prevention. This will greatly aid the work of the Free Hospital for Poor Consumptives. The recent gift of Mr. Andrew Carnegie of $1,500,000 for thirty branches of the Free Library of Philadelphia, and the recent opening of the Boys' High School, a building which cost with its equipment $1,500,000, and which is probably the finest school building in the world, are notable steps toward the city's progress in education.

Philadelphia is in a transition state with reference to great municipal improvements. Chief of these is the construction of enormous filtration beds at Torresdale, on the Delaware front, in the northeastern portion of the city, at Roxborough in the northern portion, and at Belmont in the northwestern portion. These are partially completed and will cost over $17,000,000, and they will insure a satisfactory water supply.

The Philadelphia Hospital, with its insane department and almshouse, is situated on the lower Schuylkill, and comprises within its walls a total population of about five thousand. Steps have recently been taken to remove the hospital for the insane and the almshouse to a new location below Torresdale, and on the property known as Blockley a new Philadelphia General Hospital will be erected. The original buildings constructed in 1834 will be torn down and new buildings erected in accordance with modern plans.

The Municipal Hospital for Contagious diseases will shortly be removed to a new site in the northeastern portion of the city. There are over fifty hospitals in Philadelphia. Chief of these is the Pennsylvania Hospital, the oldest institution of the kind in the United States.

The Medical Department of the University of Pennsylvania was established in 1765 by Dr. John Morgan, Dr. William Shippen, Dr. Adam Kuhn, and Dr. Benjamin Rush, who constituted the first medical faculty in America. The number of its graduates is 12,361, and with the sister schools of Jefferson College, the Medico-Chirurgical, and the Woman's Medical College, this school has had a strong influence in maintaining the high standard of medical education in the United States.

Philadelphia has long been famous for its teachers of medicine and surgery, and their contributions to medical literature, issued by the well-known medical publishers of the city, have carried the fame of American medicine throughout the world. The names of Benjamin Rush, Shippen, Physick, Wistar and Horner, Barton, Chapman, Pancoast, Gross, Stillé, Hodge, Pepper, Wood, Da Costa, Agnew, and Mitchell are household names in the medical history of our country. *Guy Hinsdale.*

PHILIPPINES, THE. See *Manila.*

PHIMOSIS. See *Sexual Organs, Male, Diseases of.*

PHLEGMON.—Definition.—To set exact limits to the term phlegmon is far from easy. Etymologically the word signifies no more than inflammation (the idea of "heat" or "burning" being equally present in both terms —φλέγειν—inflammare). Naturally, therefore, it has ever been loosely used. The concept has been merged, on the one side, into that of the so-called cellulo-cutaneous erysipelas; on the other, into that of the localized abscess.

French and German surgeons use the word in its widest sense. By phlegmon they mean any pyogenic inflammation beginning in the subcutaneous cellular tissue; even furuncle and carbuncle are by some described as varieties of the class phlegmon. In this idea they usually classify phlegmons as (*a*) simple or circumscribed, a localized inflammation resulting in a localized abscess; and (*b*) diffuse or spreading.

English and American surgeons, however, have come rather generally to attach to the word almost exclusively the latter signification, that of a diffuse process.

Inclining to the latter view the writer would adopt Ziegler's definition and restrict the term phlegmon to that pathological process in which there occurs a more or less extensive inflammatory exudate of sero-purulent or sero-fibrino-purulent nature (often called purulent œdema), spreading rapidly in the subcutaneous or in any of the submucous tissues over a somewhat large area. The causative agent is, so far as we know, always bacterial. The process may spread deeply and involve muscles, fascia, and even periosteum, and may lead to pyæmia or septicæmia.

In this sense the term must include those cases which are usually called "phlegmonous erysipelas," for the pathological and even the clinical pictures are practically identical, and the etiological factor is the same. Phlegmonous erysipelas, however, will be found discussed under *Erysipelas* in this Handbook.

On the other hand, we have those comparatively mild cases of "cellulitis" which, after showing some tendency to spread, subside easily under hot applications, or spontaneously, without causing material anxiety. These, too, must be considered to be phlegmonous, though of a mild type, because their nature pathologically and bacteriologically is the same as that of the more severe destructive process with which we usually associate the idea of phlegmon. The essential—that of a spreading inflammatory exudate caused by pyogenic organisms—is the same; and the difference, one only of degree.

While the process usually has its main seat in the subepithelial structure, it may at times involve principally deep areas of areolar tissue, such as the mediastinum, the prevertebral, or the deep perineal region. In such cases the atrium of infection may not be evident.

Etiology.—Phlegmons are in all cases due to the invasion of micro-organisms in a soil unable to resist their growth. It is with reference to both our bacteriological and our clinical knowledge that the writer would suggest the following classification*:

I. Those caused by streptococci, staphylococci, or both; also those ascribed to rarer organisms, *e.g.*, pneumococcus, gonococcus, etc.

II. Those in which the entrance of gas-forming bacteria, with or without (but most often with) the abovementioned organisms, leads to the development of a subcutaneous emphysema and gangrene in addition to the inflammatory signs of the ordinary phlegmon. This class is called "progressive gangrenous emphysema," "gangrène foudroyante," or "gas phlegmon."

III. Those caused by the extravasation of urine.

Such a classification is naturally far from arbitrary. The classes frequently overlap. For instance, a urinary extravasation is no doubt often, in part, a streptococcus phlegmon, and may be also in part a B. aerogenes capsulatus infection. The last-named is mostly combined with an infection of Class I. Class I. affords by far the greatest number of phlegmons.

It is evident that the discussion of the etiology of phlegmons must be almost entirely bacteriological. It will be in place, however, to say first a few words in regard to the *mode of entrance* of the organisms concerned. In general the *atrium* is a wound of some sort—from the most insignificant abrasion to the most complicated injury. As a matter of fact we find that phlegmons develop most frequently in connection with the more severe injuries. The contusion of the tissues in such cases renders them less resistant, while the recesses of large wounds offer greater opportunities, both for the entrance of infective matter and for the development of anaërobic bacteria, and also render cleansing less easy.

The classical descriptions of severe phlegmon, especially of the gaseous form, are those furnished us by military surgeons of the two preceding generations. Gunshot wounds and open fractures are clinically the injuries most frequently complicated by phlegmon, punctured wounds less frequently, clean cuts least often of all. Two or three cases are on record of the development of gas phlegmon following a hypodermic injection, or the subcutaneous injection of saline solution. In some cases the wound of entrance may not be in the skin but in a mucous membrane. Finally, in some cases, no point of entrance can be found.

Going on to discuss more in detail the bacteriology of phlegmon, we shall have to treat particularly of: (*a*) the relative rôles of the pyogenic cocci; (*b*) the bacillus aerogenes capsulatus and other gas-forming bacteria; and (*c*) the nature of the phlegmon of urinary extravasation.

(*a*) Since the work of Ogston and Rosenbach in the early period of bacteriology (1880–85), it has been taught, and is still generally believed, that, while the staphylococcus is nearly always the cause of circumscribed abscesses, phlegmonous inflammation is due to the streptococcus pyogenes. More extended knowledge, however, has shown us that such a proposition, while possibly true in many cases, must suffer numerous exceptions. It would carry us beyond the proper limits of this article to go fully into the question of the streptococcus as a disease-producer. Nevertheless, inasmuch as the phlegmon has hitherto been considered a streptococcus inflammation *par excellence*, it may not be amiss to consider here whether such a conclusion is quite justified or not.

What is the relative etiological importance of these organisms in the causation of phlegmon? When we come to examine the literature of the subject, we find really a very small number of phlegmons, comparatively speaking, in which the streptococcus pyogenes has been found as the causative agent. Janowski[1] in his monograph upon suppuration, says: "Numerous experiments have shown that the streptococcus is not necessarily more virulent than the staphylococcus; on the contrary, that it produces decidedly less often than the staphylococcus its effect upon the organism of the host. It has been found also that whereas the streptococcus alone, or more frequently combined with the staphylococcus, occurs in many cases of small abscesses, it is discovered comparatively seldom in phlegmons, for which in particular it was claimed (Ogston and Rosenbach) to be characteristic. Thus Steinhaus,[2] examining 10 cases of phlegmon, found the streptococcus only once, and in that case combined with staphylococci. In the other 9 cases the latter alone were present. Janowski,[3] in 8 phlegmons found the streptococcus only once alone, in 4 cases staphylococci alone, and in 3 the two combined. Szczegolew[4] examined 21 cases and found the streptococcus alone only in 7."

In 19 cases occurring during late years in the Royal Victoria Hospital, Montreal, the bacteriological examinations revealed the staphylococcus (mainly aureus, or albus, or both) in 8; streptococcus pyogenes in 8; and in 3 a mixed growth.* It may thus be inferred that the staphylococcus (aureus or albus) plays at least as great a rôle in the causation of phlegmon as does the streptococcus pyogenes. I have been unable to find in the literature of the last few years any special discussion upon this point.

(*b*) *Gas Phlegmons.*—That variety of acute phlegmon which produces gas in the subcutaneous tissues has ever been greatly dreaded by the surgeon. The term *foudroyante* (gangrène foudroyante), given to it by Maisonneuve, expressed its terrible character. It was the "progressive gangrenous emphysema" of the older surgeons, and indeed still goes by that name. The Germans call it "Gasphlegmon."

With the modern method of treating wounds it is becoming a much rarer disease than formerly. The chapter of its etiology is still far from being closed; nevertheless, the researches of Welch and Nuttall, Flexner, and several others in this country, and of Fraenkel in Germany, have thrown a flood of light upon the question.

In 1891 Welch and Nuttall,[5] of Baltimore, discovered the organism to which they gave the name "bacillus aërogenes capsulatus." In 1893 Fraenkel,[6] ignorant of Welch's work, discovered the same organism independently, and gave to it the name of "bacillus phlegmonis emphysematosæ." It is by Welch's name that it has come to be most widely known. This organism is a strict anaërobe; and it is possible that the comparative paucity of thorough anaërobic work, both before and since 1892, may account for the fact of its not having been earlier discovered, and also for the fact that there exists still, after ten years, but a comparatively small literature upon the question. Up to a late period the bacillus of malignant œdema was held to be accountable for practically all cases of "gangrène foudroyante." In the last few years, owing to the publications of Welch and Fraenkel, the pendulum has swung to the opposite extreme, and the bacillus of malignant œdema is allowed but slight if any part in the causation of the gaseous phlegmon. Welch,[7] in a thorough discussion of the subject, in which he reviews forty-six cases of bacillus aërogenes infection, remarks on the need of a more accurate knowledge concerning the malignant œdema bacillus. Neither he nor Fraenkel could find it in their comparatively numerous cases of emphysematous gangrene; and he believes that older investigators worked with insufficient methods.

Fraenkel[8] regards the disease caused by his bacillus (which is identical with Welch's) as one *sui generis*, and that due to the malignant œdema bacillus as quite a different clinical entity, because in animal experiments the latter produced no gas.

Hitschmann and Lindenthal,[9] on the contrary, believe that gangrenous emphysema is an anatomico-clinical entity, but due to different infections. Of these the bacillus of malignant œdema would be the one most frequently found; Welch's bacillus next; while finally the bacillus coli communis and the proteus might be responsible for a few cases.

The most recent work upon this question is that of Silberschmidt.[10] His conclusions, based on extremely thorough and straightforward work, certainly carry weight. In three cases of phlegmon accompanied by the development of gas, he found in one the bacillus œdematis maligni; in another, an organism belonging to the "group of malignant œdema bacilli"; and in the third an undetermined non-pathogenic anaërobe. In all cases there was mixed infection; in the first with B. coli communis, in the second with streptococcus pyogenes, and in the third with staphylococci and streptococci. He concludes that the B. œdematis maligni may certainly cause the formation of gas in "gangrène foudroyante."

In a fourth case of infection and death, following the opening of a cold abscess of the femur, in which there occurred a gradual formation of gas in the course of the six days subsequent to the operation, Silberschmidt found, in addition to the ordinary staphylococci and streptococci, a strictly anaërobic streptococcus which produced a foul odor. He comes to the conclusion that gangrène foudroyante may be caused by a number of different organisms. He is inclined to ascribe typical gas gangrene to anaërobes alone. He contests the strict classification of Welch and Fraenkel, and agrees with Lindenthal and Hitschmann that the same clinical picture as is recognized to be due to Welch's B. aërogenes capsulatus may be produced by other anaërobes and in especial by the bacillus of malignant œdema.

There is some evidence in late literature to show that other anaërobic bacteria besides Welch's bacillus and the bacillus of malignant œdema may produce gas in the tis-

* In the following discussion I have avoided the term "malignant œdema" because of its lack of exactness. It has evidently hitherto been used in a loose sense, to designate cases both of gas-bacillus infection (*i.e.*, gangrène foudroyante, progressive emphysematous gangrene, etc.) and of severe strepto- or staphylococcus phlegmon, as well as of phlegmon due to the bacillus of malignant œdema (Koch). Further, the term in itself suggests that the bacillus of malignant œdema is the causative factor, whereas late investigation has shown that this bacillus is but rarely at fault.

* The possibility of some degree of error in figures such as those quoted must be admitted. The difficulty of growing the streptococcus pyogenes on artificial media; the fact that, as Marmorek has shown, they soon exhaust the medium and refuse to grow further; their liability to be outgrown by the hardier staphylococcus; the frequent failure on the part of bacteriologists to examine stained slides of the original pus; and finally the fact that some streptococci are strict anaërobes, whilst anaërobic cultures are rarely set up as a routine practice,—all these points render an unqualified acceptance of statistics upon the point in question impossible, save in cases in which we know that very careful work has been done. Nevertheless the figures are extremely suggestive.

sues *intra vitam*. Lindenthal[11] found an anaërobic bacillus (in conjunction with the B. coli communis) belonging to the group of the œdema bacilli, which both *in vitro* and in the tissues produced gas. He isolated it from cases of colpohyperplasia cystica, characterized by the formation of gas cysts in the vaginal wall with surrounding necrosis and cell infiltration.

It has been claimed by various observers that the B. coli communis may cause gas formation in the tissues, especially in cases of diabetes. Such statements, in the light of our general knowledge concerning B. coli infections and concerning the rôle of anaërobes in gas production, must be viewed with considerable scepticism. The observations thus far have not been based upon sufficiently thorough work. The same may be said of the proteus Hauseri, for which similar claims have been made.

In this question of etiology we have been discussing the seed only. But the soil has also a great importance. These gas-producing anaërobic organisms are ubiquitous in their nature; they are found in practically any specimen of earth, or dung, or dust. J. C. Friedman[12] has found seven different anaërobes in the cæcum and appendix of man, of which the B. aërogenes capsulatus was the most frequent. Why then are gas phlegmons comparatively so rare? The reason must be sought in the unsuitability of the soil. The seed is constantly being sown; but the soil nearly always destroys the seed, or at least refuses nourishment. Its resistance must be diminished before the seed can grow. And thus as a matter of fact we find that these infections nearly always complicate severe injuries; in the majority of cases recorded it is an open fracture. There is nearly always some chemical or mechanical lesion of the tissues. This was well proved experimentally by Berson.[13]

Symbiosis is also, without doubt, an extremely important factor. An old observation shows that the virulence of the streptococcus may be enormously increased by growing it with the B. prodigiosus. The greater number of the more severe septic processes are found bacteriologically to show mixed infections. In all six cases of severe infection reported by Silberschmidt there was mixed aërobic and anaërobic infection; and it is especially in the case of these anaërobic organisms that this question of symbiosis becomes of importance. For it has been demonstrated in the test tube that even a strict anaërobe will grow well enough in the presence of some oxygen, provided a hardy aërobic organism is inoculated with it. The latter probably uses up what oxygen is present, and thus creates a partially anaërobic atmosphere.

Practically the commonest germs found with the gas-producing bacilli have been the pus cocci, B. coli communis, B. typhosus, and putrefactive bacilli. For the B. aërogenes capsulatus, or the B. œdematis maligni to be found as the sole organism in severe or fatal cases of gas phlegmon is of rare occurrence.

It goes without saying that, apart from local injury and the symbiosis of bacteria, all such general diseases as reduce the vitality of the patient predispose to the development of a soil favorable to the invasion of disease germs. Thus, for instance, a phlegmonous erysipelas may be fatal in the advanced stages of tuberculosis or cancer; and the urinary extravasation of the strictured alcoholic is notoriously dangerous to life.

(*c*) In *urinary extravasation* the urine infiltrates the perineum, scrotum, penis, and frequently also spreads to the upper part of the thigh, the groin, and the lower abdominal region. Aseptic urine in the subcutaneous tissue in small quantities has been shown experimentally to produce but slight inflammatory reaction, or none at all. Nevertheless, clinically, the infiltration of urine seems to produce almost in every case a most decided inflammation. In many cases no doubt the urine is already infected. Even if not infected at the moment, as in cases of wounds of the healthy urinary tract, it is usually given every chance to become so by the necessary catheterization. Moreover, it is driven into the tissues not only with great force, but also in great quantity, the bladder muscle acting as a *vis a tergo*. Some of the organisms which most frequently infect the bladder decompose the urine with the formation of ammonium carbonate and often also of free ammonia. The B. coli communis, B. lactis aërogenes, or other bacteria belonging to the same group; the diplococcus ureæ liquefaciens, the proteus Hauseri are those most often found. The staphylococcus and streptococcus are also not seldom present, and would render still more acute the inflammatory process set up. When we consider what a powerful combination of factors for ill we have in these cases,—the mechanical distending action of the urine, the chemical action exerted by the products of its decomposition, the special inflammatory action of the bacteria present, and finally the run-down condition of most of the subjects,—we can understand how virulent the process often is, and how the patient so rapidly succumbs to the sepsis engendered.

Clinically we usually find the extravasation taking on a phlegmonous character very rapidly. A painful, vividly red swelling appears, and it needs no long time for the decomposed, frequently ammoniacal urine to cause breaking down and putrefaction of the tissues, with the formation of foul-smelling pus and very frequently gas. High fever soon sets in, and the general condition becomes rapidly bad. Frequently operation, even early operation, comes too late, and death follows with septic symptoms.

One point, that of the frequent presence of gas in these phlegmons, remains rather unsettled. Whether the colon bacillus can be accused is very doubtful; at the most it might produce gas in diabetics. It is possible again that the gas represents in part at least free ammonia from the decomposition of the urea.

Anaërobic bacteria, such as those concerned in gas phlegmon elsewhere, may be at the botton of it, but I have been unable to find literature upon that point. Welch, in his exhaustive article[7] upon cases of gas-bacillus infection, shows that the B. aërogenes capsulatus may not only gain entrance to the body by way of the urinary tract, but also set up its own infection in the tract itself. The gas produced "may be either free in the cavity of the bladder, ureters, or renal pelvis, or contained within submucous blebs, or in both situations." But though the presence of this bacillus in the urine is well attested *inside* the urinary tract, I can find in a somewhat careful search of the literature no record of its having been demonstrated in urine *outside* the tract—I mean in extravasations. This is a point for future investigation.

Morbid Anatomy.—In discussing the morbid changes which occur in this disease, we take as our type the acute phegmon of a limb which occasionally complicates a severe injury, and which is due to the ordinary pyogenic cocci.

The gross and microscopic changes in the tissues are practically the result of a very intense inflammatory process, and involve principally the subcutaneous cellular tissue, but also the true skin and in some cases the deeper structures. The skin becomes deeply red, and there often occurs a decided lymphangitis, so that clinically we see the well-known red lines running toward the neighboring glands. In the cellular tissue the organisms advance rapidly along the lymphatics and connective-tissue spaces, while the host reacts with a copious outpouring of inflammatory lymph. The parts become so infiltrated that there is imparted to the examining finger the hard sensation of brawn—a brawny œdema. The skin later assumes a dusky red tint, and exudation proceeds to the point of threatening gangrene. Occasionally gangrene actually occurs, the skin becoming marbled with purplish-red areas and being thrown off finally, either in patches or over larger areas.

If incisions are made in the early stage, say within the first day or two, there exudes nothing but a thin serum which may or may not be slightly turbid from admixture of leucocytes and flakes of fibrin. If the process has gone on to the "brawny" stage the cut surface shows a pork-like aspect, or sometimes more like orange pulp, especially at the area of greatest intensity. A little later,

vesicles filled with a turbid fluid may form in the skin, owing to localized exudation in the rete.

Meanwhile in the cellular tissues, if the case be severe, the inflammatory process goes on from mere infiltration to a coagulation necrosis. The necrosis is due not only to the mechanical distention of the exudate, with the venous thrombosis and general circulatory stasis which it produces, but also to the accumulation of bacterial toxins. It seems to be a frequent property of the organisms usually concerned (virulent streptococci or staphylococci) to cause intense and rapid necrosis of the parts infiltrated, while their peptonizing power remains in the background. Nevertheless, after the necrosing process has continued a variable time, there succeeds—if the patient have not succumbed to a fulminating septicæmia—a certain amount of liquefaction or peptonization, so that in the course of a few days we have in the subcutaneous tissues what might be called a lake of pus, in which float shreds and masses of sloughy tissue. After incision these shreds often have to be pulled or cut off, and resemble strands of "wet tow" or "wet chamois leather." If the liquefying process has been less active, we get a series of small ponds, or irregular bayous of pus, separated by isthmuses or peninsulæ of wholly or partly dead tissue.

If incision does not give vent to the pus, it requires no long time for the latter to find its way out through the skin. In such cases sloughy ulcers persist, through which shreds of necrotic tissue and pus are discharged, and healing takes place slowly by granulation. In rare instances it may burrow deeply and invade joints, destroy muscles and tendons, or corrode arteries. If deeply situated and covered by strong fasciæ—such as in the pelvirectal space, the retroperitoneal tissue, the mediastinum or under the fronto-occipital fascia—it may not seldom break through into hollow viscera, or attack the serous membranes.

The *microscopical phenomena* are largely those of ordinary inflammation—outpouring of inflammatory serum, diapedesis of leucocytes, phagocytosis, the accumulation of round cells in groups in the corium and in the septa between the fatty masses of the panniculus; the reactionary proliferation on the part of the lymphoid and fixed tissue cells; and the dilatation of the lymphatics, which are filled with organisms.

The microscopical appearances in detail will vary according to the severity of the infection. In mild cases, subsiding rapidly under incision, there is evidently comparatively slight reaction of the body cells; pus may not be found; and the exudate is reabsorbed. In the typical severe case the early extreme serous exudation becomes in the course of a few days invaded by a large number of leucocytes; the proteolytic ferments of the pyogenic bacteria come into play; necrosed tissue is liquefied, and pus is formed. This pus is not localized, but is distributed over considerable areas as an infiltration. Finally, in the fulminating cases we find again but slight evidence of any cellular reaction. The bacterial toxins kill before the individual defenders of the body can marshal to resist. Welch[7] has found this last-mentioned state of affairs to be especially true of pure infections with the B. aërogenes capsulatus. This bacillus, if pure, leads mainly to necrosis, the nuclei disappearing by karyolysis, while leucocytes and cellular reaction are remarkable by their absence.

The characteristic points, however, in ordinary phlegmon are the excessive primary exudate of serum, the comparatively late proliferation and advance of the leucocytes and other body cells, and the marked necrosing power of the microbic toxins.

While the above would represent the changes in the average severe case of phlegmon, many other less frequent types might be set up, dependent upon the situation of the process and its degree of virulence. Thus we have the deep phlegmon, situated underneath the deep fascia, in which the pus accumulates in the intermuscular septa, which it may infiltrate in long strands. The superficial structures meanwhile may show for many hours or for many days no sign of the underlying infection; yet sooner or later œdema develops. In mild cases the exudate may never become purulent and may be finally reabsorbed if incisions have not been made. At the other extreme, we meet the very malignant type in which the whole limb becomes, within twenty-four to forty-eight hours, intensely œdematous, while the patient succumbs, or the limb is amputated, before pus has time to form. Such cases are due probably to the extremest degree of virulence of the pus cocci.

In cases of emphysematous gangrene the above picture becomes more or less typically modified. The wound secretion, hitherto comparatively healthy, becomes increased and smells horrible; thin, fairly clear serum flows from the drainage openings; the parts around the wound become swollen; the neighboring skin begins to show, in spots or in patches, a purplish or blue-black coloration; air collects in the interstices of the subcutaneous tissue, giving to the examining finger a sensation of fine crackling. This emphysema, accompanied by extreme œdema, spreads rapidly up the limb; the skin takes on a special coloration, which was particularly noticed by the first observers and was compared to that of a dead leaf, or to that of Florentine bronze. Sometimes it looks like a week-old ecchymosis. The distended veins stand out in blue against this dark-brown background; here and there show up patches of a darker color, beginning gangrene, which, in this form of phlegmon, tends greatly to become extensive. The emphysema may extend so rapidly that its advance has been followed for inches during a few minutes' observation.

Clinical Course.—This will vary to some extent according to the nature and degree of virulence of the causative factor. If we take, as before, an ordinary case of rather severe wound infection, the signs are somewhat as follows. They may be considered as (*a*) local, and (*b*) constitutional.

(*a*) *Local.*—At a variable period following the infliction of the wound, but usually within three or four days, inflammatory signs appear in its neighborhood. The edges grow red and œdematous; the same condition spreads rapidly up the limb, invading the subcutaneous cellular tissue *per continuum*; at the same time pain of variable intensity is usually felt in the parts. The lymphatics may show as red lines in the skin leading up to the nearest lymph glands. The œdema may involve a whole extremity within thirty-six to forty-eight hours. Vesicles frequently form and are filled with turbid serum.

If numerous incisions are made at an early stage, the process may frequently be arrested and subside with or without the formation of pus. The converse of this picture is seen in those cases in which, in the absence or failure of operation, the inflammation extends rapidly beyond the limits of the extremity involved and attacks the trunk. *Exitus letalis*, within a very few days, is then the most frequent ending.

(*b*) *Constitutional.*—The symptoms are usually grave. Even the cases of slight or moderate severity are ushered in with chilliness, fever, general malaise, etc. In the very severe cases the chill is marked, the fever high, the pulse rapid; the patient may become somewhat delirious, and after a variable lapse of time sink into a typhoid condition and die of acute general sepsis. In other cases, especially in those whose resistance has been weakened greatly, the course may be of an asthenic type from the beginning, and the patient shows neither locally nor constitutionally any appreciable effort at throwing off the *noxa*. Cases of this nature are to be set down, in the present state of our knowledge, to infection with the streptococcus pyogenes, the staphylococcus aureus or albus, or to a combination of both; with the additional presence, in some cases, of still other bacteria, such as those of the colon group and proteus.

The ordinary severe case, left to itself or operated late, is characterized in its later course by the results of the extensive cellular necrosis. Through spontaneous or operative perforations pus and shreds of slough are discharged for many days or many weeks. If finally the wounds granulate up, the structures involved—skin,

muscles, tendons, fasciæ, nerves, and arteries—are all involved in the reparative scar; and the patient may be left with an impotent limb. In some cases healing does not occur; suppurative fever continues; pyæmia or septico-pyæmia develops and ultimately leads to the death of the patient.

When Welch's B. aërogenes capsulatus or, less often, the bacillus of malignant œdema enters the field, either alone or combined with the pyogenic cocci, the clinical picture is usually more grave. Our classical clinical descriptions are given by Maisonneuve and Pirogoff. In their day, with the abundance of military surgery, cases were much more frequent than now. Pirogoff divides the cases into two clinical groups. As I am unable to get access to the original literature, I paraphrase from Welch.[7]

"(*a*) In the very virulent there is but slight local reaction while the part goes on into crepitating gangrene. The emphysema and necrosis spread rapidly and the patient usually dies in a few days with symptoms extremely toxic and asthenic. (*b*) In the other group there is reaction. The emphysema is preceded and accompanied by local œdema or purulent infiltration, as well as by febrile reaction; it appears later after the injury, and spreads less rapidly. All gradations are, however, observed."

The cases in which phlegmon has been ascribed to the invasion of bacteria other than those mentioned above are very rare. Almkvist[14] describes a case of extensive infiltrating abscess of the foot in which gonococci alone were found. He refers to four similar cases in the literature.

A cellulitis of the orbit has been described as due to the influenza bacillus.[15] Netter[16] reports a case of phlegmon due to Fraenkel's pneumococcus.

Other than these I have been unable to find in the literature.

In addition to the above general consideration of phlegmon the writer has thought it advisable to make a few remarks upon the characteristics of phlegmon, in its various localizations, before going on to the questions of prognosis, diagnosis, and treatment.

Phlegmons of the Scalp.—The anatomical peculiarities of this region give an especial interest to the question of phlegmon. The parts are so unyielding that inflammatory processes tend to spread widely and to infiltrate. This is true of inflammations both above and beneath the fronto-occipital aponeurosis, but especially of the latter. The subaponeurotic connective tissue being continuous with the cranial periosteum, phlegmon, when it attacks the former, is extremely apt to destroy the latter and so lay bare the bone. The dangers of the subjacent, more or less inevitable, bone inflammation, or of vein thrombosis, are evident. The internal periosteum (*i.e.*, the dura mater) may easily become involved. Von Bergmann refers to cases of deep-seated brain abscesses arising solely by infection transmitted by contiguity along thrombosed veins. As compared with the superficial soft parts, the aponeurosis, together with the connective tissue binding it to the periosteum, necroses very easily. This is due largely to the manner of its blood supply. Whereas the main vessels of the superficial parts run horizontally to the surface and thus frequently escape injury in lacerated wounds, those supplying the aponeurosis and periosteum run mainly vertically to the parts nourished, and are apt to be torn across in wounds of the scalp. Moreover, the tension which the tight aponeurosis exercises upon any large exudate leads mechanically toward death of the tissue.

Diffuse phlegmon, therefore, of the scalp, especially if deeply situated, is one of the most formidable complications of contused and lacerated wounds. Fortunately, aseptic surgery has made it rare. The accompanying fever is high, and the resulting abscesses are numerous, while the liability to intracranial suppuration is not small.

The erysipelas which attacks deep scalp wounds is especially to be feared. The subaponeurotic connective tissue is in such wounds the part most injured; and the *locus minoris resistentiæ* thus created attracts, so to speak, a superficial erysipelas into the deep parts, and thus gives rise to a phlegmonous erysipelas.

Phlegmon occurs usually as a complication of wounds of the head, but also follows osteomyelitis of the cranium; or, again, it may be an extension from face phlegmons. Pain, swelling, high fever, and especially the swelling of glands behind the ear are the early *symptoms*. Early *diagnosis* is important, because here, if anywhere, is early and deep incision—down to the bone if the phlegmon is deep—necessary, if both extensive necrosis and also the danger of intracranial mischief are to be avoided. The incisions must be kept well open with gauze or drainage tubes.

Phlegmons of the Neck.—Inflammatory processes in the neck are usually circumscribed and end in abscess. The diffuse phlegmon is comparatively infrequent; it may arise primarily as such, or may be secondary to a localized inflammation.

It is caused by infection of wounds by extension from neighboring inflammations, either by contiguity or by the lymphatics through an adenitis. Rarely can it be ascribed to a hæmatogenic infection, as in pyæmia.

The clinical picture varies somewhat according to the anatomical region involved. Of these the most important is the *submaxillary phlegmon*, for which the ordinary term is *Ludwig's angina*. It is certain that Ludwig's angina is a name which has been too loosely used. Most frequently it has been confounded with other inflammatory processes which have as their most striking symptom an œdema of the glottis, such as phlegmonous erysipelas of the larynx, or acute perilaryngeal infection dependent on other causes. As a matter of fact the disease described by Ludwig, in 1838, was an acute infection of the connective tissue of the submaxillary spaces secondary to an adenitis of this region, the original lesion being usually a carious tooth, a mucosal ulcer, or a tonsillitis. The depth of the inflammation, the extreme pressure exerted on the exudate by the unyielding deep fascia covering the space, and the liability therefore toward involvement of neighboring organs (in especial the larynx) gave the disease a characteristic picture—that of a severe and frequently fatal infection. The term Ludwig's angina should be reserved for cases showing the above pathological condition.

Bacteriologically the streptococcus is found most frequently. In four cases reported by Leterrier the streptococcus was found twice, the staphylococcus aureus once, and in the fourth an undetermined bacillus. In four examined by Gasser, the streptococcus, combined with a very virulent B. coli communis, was found in each case (quoted by Jordan in the "Handbuch der praktischen Chirurgie").

Pathologically there are found a purulent infiltration of the connective tissue of the space, and an extensive inflammatory œdema of the floor of the mouth, the larynx, and the pharynx.

The *symptoms* are in general those of the acute phlegmon anywhere. But the local conditions add the characteristic signs of difficult or impossible deglutition, great dyspnœa, impossibility of opening the mouth, salivation, and *fetor ex ore*. Death frequently follows in a few days with symptoms of general sepsis, or from laryngeal œdema if tracheotomy be not quickly done. The infection may kill in from two to three days. Such fulminating cases are due to an extremely virulent streptococcus toxæmia, and the earliest surgical interference may be insufficient to prevent death. In some cases of a milder degree of infection a localized abscess results, and breaks into the mouth or through the skin.

The *prognosis* has certainly brightened of late years. Delorme in 1893 was able to report a series of seven recoveries in cases operated early.

Treatment.—Incision should be practised at the earliest possible moment, even before pus can be diagnosed. It is best made a finger's breadth below and parallel to the alveolar border of the jaw. After superficial incision it is best to proceed deeply with a blunt-pointed instrument. With great dyspnœa tracheotomy should be done without hesitation.

Phlegmons may develop at the angle of the jaw and in the submental region; but in these situations they tend to localization and heal easily upon incision. Phlegmons may arise in the loose areolar tissue surrounding the large vessels of the neck, the vessel sheath. They come in frequency next to those of the submaxillary region, and originate most often from the deep cervical glands following anginal affections, carious teeth, otitis, etc. While infection in this region is frequently of the comparatively mild, circumscribed type, yet when the severe spreading type does occur, it is one of the most dangerous conditions possible. The swelling is deep and hard and may extend rapidly from the ear down to the clavicle, and from the spine to the trachea. The skin becomes reddened rather late, and wry-neck and trismus come on. Frequently death occurs from sepsis before pus forms. When abscess forms, the pus may sink into the mediastinum or axilla, and may cause compression of the trachea, œsophagus, large vessels and nerves, and these complications may be the immediate cause of death.

The *prognosis* is bad. *Treatment* consists in very early multiple incisions.

Complications.—Besides those already mentioned there may occur erosion and perforation of vessels. In the case of a vein, bleeding may be absent, if the vein has been occluded by a thrombophlebitis; in the case of an artery, however, there follows not infrequently fatal hemorrhage, although at times the bleeding is moderate and repeats itself at intervals. The carotids have been ligated in continuity in such cases with success.

Reclus[24] has described under the name of "phlegmon ligneux du cou," a rather chronic form of phlegmon, developing very slowly, of very hard consistence, and involving a large area on one side of the neck. It may simulate closely a new growth.

Phlegmons of the Axilla.—These are either subcutaneous or subfascial. They have their origin usually in a lymphangitis running from a primary infection in the hand, arm, or breast. Frequently the atrium of infection is scarcely to be found, and the first symptom is pain and swelling in the axillary glands.

The subcutaneous phlegmon is easy of diagnosis; more difficult is it when the phlegmon begins deeply under the pectoralis and spreads toward the axilla. With the ordinary constitutional disturbance we get a dull pain and a restriction of shoulder movements, especially of abduction. It is only after several days usually that a dilatation of superficial veins and a slight swelling over the pectoral region are seen.

Incision should be made, as soon as the affection is diagnosed, at the posterior edge of the great pectoral through the deep fascia, and then blunt dissection should be continued with the finger under the muscle, until the pus is reached.

Phlegmons of the Upper Arm.—In this region phlegmons of a comparatively mild nature and insidious onset are not infrequent on the inner side of the arm, and originate in inflammatory conditions of the lymphatics or lymph glands in the bicipital sulcus or in a bursitis olecrani.

Apart from the ordinary subcutaneous phlegmons Jaboulay[25] distinguishes cases in which the process is situated under the deep fascia running in the sheath of the vessels, thus forming a band along the inner side of the arm to the axilla, which upon abduction of the arm is painful. Naturally the fascia must always be split in order to lay open the focus.

Phlegmon of the Fingers and Hands.—Phlegmonous processes in the hand require especially early diagnosis and thorough treatment in view of the too frequent loss of function, sometimes of "earning power" (as the Germans call it), following upon failure or neglect in this particular.

The panaritia, confined to the terminal phalanx, are hardly to be considered here. Nor do the infections beginning in the periosteum and confined to one of the phalanges properly come into our classification of phlegmons. It is true that either of them may lead to phlegmonous processes, but to discuss them would lead beyond the limits of this article.

The important phlegmons of this region are those which involve the tendon sheaths, either primarily or secondarily. It is necessary to remember with regard to phlegmonous processes, whether in finger or hand, the anatomical peculiarities of the subcutaneous connective tissue. On the dorsal surface the connective-tissue bands, uniting skin to fascia, run at an acute angle, in fact nearly parallel to the long axis of the limb, thus allowing of a great deal of elasticity in the subcutaneous tissue. On the palmar surface the reverse is true; the connective-tissue bands run mainly vertical to the long axis of the limb, and are characterized by thickness and density. The result is that any inflammatory exudate occurring on the palmar aspect meets with great resistance in its spreading toward the surface, but finds an easy path toward the dorsum. Thus it happens that a marked cellulitis, with great redness and œdema, frequently appears on the dorsum of the hand, while the focus is really situated on the palmar surface and may, if beneath the palmar aponeurosis, have occasioned on that aspect of the hand no sign at all suggestive of inflammation. No doubt the cyanotic œdema of the dorsum in such cases is caused partly also by pressure on the deep dorsal veins as they pass forward between the metacarpals to join the palmar arch.

In such cases an exact examination is required, lest dorsal incisions be counted sufficient, and the one really necessary palmar incision down to the focus be overlooked. The one criterion available is an exact localization of the point of greatest tenderness. The whole hand should be examined for this area with a blunt-pointed probe. The point of greatest tenderness will always correspond with the primary spot of infection; and as a matter of clinical experience that focus is in the great majority of cases on the palmar side.

With regard to infections of the tendon sheaths we must remember their anatomical relationships to each other. The thumb sheath runs up to a point under the annular ligament, while that of the little finger extends under the annular ligament, and in some cases a short distance up the arm. Both communicate with the large palmar bursa. Infection of either of these two may lead by continuity, even in the absence of direct communication, to involvement of any or all the others.

The course is frequently very acute. In a very few days the sheath may be filled with pus; and, being normally but poorly nourished, it necroses very readily. In such cases the necessity for the promptest surgical interference is exceptionally evident; expectant treatment is apt to leave a practically useless hand. If not opened early the infection may spread to the deep connective tissue and cause an extensive phlegmon of the palm; it may extend from here up beneath the annular ligament and cause a deep phlegmon between the muscles and tendons of the forearm. The possible further danger of septic thrombophlebitis and pyæmia is well known. Even nowadays such progressive phlegmons, which might have been arrested in the hand by proper interference, lead occasionally to amputation of the arm. Failing this, convalescence extends over months, and at the end the patient is left with an arm whose usefulness is largely destroyed. The importance of early interference in, for instance, the case of a workingman is enormous. Rather too long and too deep a cut than too small or too late a one. It is scarce possible for it to be too early. It is wise in incising the palm for a deep infection, to push forward bluntly with a pair of forceps after getting through the skin. A counter opening on the dorsum with through-and-through drainage is often advisable. General anæsthesia is probably the best for these extremely painful phlegmons of the hand.

With regard to after-results a certain amount of fixity of the tendon involved is scarcely to be avoided. Yet if massage, hot bathing, passive movement, and electrization of the corresponding muscles be instituted as soon