The superiority claimed for it over other antipyretics was based not only on its greater solubility and more rapid action, but also on its perfect harmlessness. The result of experiments by Dr. Isaac Ott, however, shows that in very large quantities it produces the same effects as phenacetin and similar compounds. He reports: (1) that upon frogs it produces a general paralysis, due to an action upon the cerebrospinal axis; (2) upon rabbits it produces a cyanotic condition of the ears, and reduces the force and frequency of the heart; (3) it kills through an action upon the centre of respiration.

The investigations and clinical reports upon the action of this new remedy have been chiefly made in Germany and Italy, but Drs. Cerna and Carter, of Philadelphia, have done some very thorough experimental work, to determine the comparative action of antipyrin, phenacetin, and phenocoll. The following are their conclusions regarding its action on the heart and circulation:

1. Phenocoll, in ordinary amounts, has practically no effect upon the circulation.

2. Large doses diminish the blood pressure by influencing the heart.

3. Phenocoll reduces the pulse rate by stimulating the cardio-inhibitory centres. It then increases the rapidity of the pulse by paralyzing said centres. The final diminution is of cardiac origin.

4. Upon the blood itself phenocoll has no action.

As to the relative action of the three antipyretics that were experimented with, they sum all as follows:

1. Antipyrin, phenacetin, and phenocoll all fail to produce any effect on the heat functions of the normal animal.

2. Antipyrin produces a decided fall of temperature in the first hour after its administration in the fevered animal. This reduction is due to a great increase in heat dissipation, together with a fall in the heat production.

3. Phenacetin, both in septic and in albumose fevers, produces a very slight fall of temperature during the first and second hours after its ingestion by the stomach, but the greatest reduction occurs during the third hour after its ingestion. The fall of temperature results chiefly from a decrease in heat production, with a slight increase in the heat dissipation. The increase in dissipation is not as great as with antipyrin. Probably the delayed action of the drug depends on its insolubility.

4. Phenocoll causes in fever a very decided fall in temperature, which occurs during the first hour after the administration of the drug by the stomach. This reduction is the result of an enormous diminution of heat production, without any alteration of heat dissipation.

Phenocoll has been recommended as an antipyretic, analgesic, antirheumatic, and antiperiodic, but has not proved itself of particular value. Its action resembles that of phenacetin, but phenocoll requires to be given in larger doses. In some instances ill effects have followed its employment. Excessive sweating, dyspnœa, marked depression, rashes, darkened urine, and many other unfavorable symptoms have been reported.

Beaumont Small.

PHENOL-BISMUTH — $C_6H_5O.Bi(OH)_2$ — is a practically odorless and tasteless, white, non-irritant, and non-toxic powder, containing nineteen per cent. of phenol. Like other bismuth preparations, it is used as a mechanical sedative and antiseptic to the gastro-intestinal tract, but in addition, as shown by the urine, sets free some of its phenol. No poisonous effects have been noticed (Jasenski) from taking 5 gm. (gr. lxxv.) a day for three weeks. R. W. Wilcox says that it is superior to all other forms of bismuth in fermentative dyspepsias and in chronic gastritis marked by pyrosis, or boulimia. The dose is 1–5 gm. (gr. xv.–lxxv.) daily. *W. A. Bastedo.*

PHENOLPHTHALEIN — $C_6H_4.CO.C(C_6H_4OH)_2.O$ — is prepared by digesting ten parts of phenol, five parts phthalic anhydride, and four parts concentrated sulphuric acid for several hours, boiling the residue with water to remove soluble matter, and then boiling the remaining resinous substance in benzol. Phenolphthalein is a yellowish-brown powder, which, in 1 to 30 alcoholic solution, serves as an acid-alkali indicator in volumetric analysis. Colorless in acid solutions, it turns a brilliant pink on neutralization with an alkali. It is not, however, a safe indicator for the carbonated alkalies.

At the British Medical Association, 1902, Tunnicliffe reported over one thousand cases of its use as a cathartic. It may safely be employed in renal disease as it is excreted by the intestines and not by the kidneys. The dose is 0.15–1 gm. (gr. iiss.–xv.) in tablets, 0.3 gm. (gr. v.) being usually sufficient to purge an adult.

W. A. Bastedo.

PHENOL, POISONING BY.—This substance, now a very familiar antiseptic, is known more generally as carbolic or phenic acid, also as coal-tar creosote. True creosote, the characteristic ingredient of wood-tar, especially that from beech-wood, is not identical with phenol.

Phenol, as the common name indicates, has some acid properties, but is, more strictly speaking, an alcohol. Its formula is C_6H_5HO. When pure it is a colorless, crystalline, deliquescent mass, soluble in water, alcohol, and glycerin, with a well-marked odor, and a burning taste. The *crude carbolic acid* of commerce is variable in composition, and often consists of little else than neutral tar oils, which are destitute of any antiseptic qualities.

Applied to the skin, phenol produces a white superficial eschar; on the mucous surface the effect is more severe. A number of cases are recorded in which death has resulted from external application, even to a limited surface. The introduction, at a comparatively recent date, of phenol in antiseptic surgery has been responsible for several fatal cases.

When phenol is swallowed in moderate concentration an intense burning sensation is immediately experienced in the throat, œsophagus, and stomach, the mucous membrane becoming white and hardened. Vomiting of a frothy mucus occurs. The skin becomes cold, the lips and ears livid, pupils contracted and insensitive, and breathing difficult; the pulse may be 120 and irregular. The urine becomes dark-colored, and may be suppressed. These symptoms are soon followed by insensibility, with stertorous breathing. The appearances after death are largely those of local action of the poison, but the train of symptoms shows that, as in the case of nearly all other poisons, there is a distinct action on the nervous system to which the fatal result is largely due.

The fatal quantity is somewhat difficult to fix, owing to the great variation in strength of the commercial solutions, in which form the acid is generally encountered in cases of poisoning. In one case noted by Taylor a woman died in about half an hour after swallowing a wineglassful of, probably, a weak aqueous solution of phenol. The minimum fatal dose is given by some authorities as one drachm, but recovery from such an amount is possible. Half an ounce is almost invariably fatal. Fatal results have several times occurred rather rapidly, that is, in less than an hour.

The best antidote is alcohol—the strong commercial spirit for external application, common whiskey, or the commercial spirit diluted considerably, for internal use. The alcohol not only stops the action of the poison, but if the damage be not very great, it restores the condition of the tissues. Other chemical antidotes that have been advised are magnesium sulphate, sodium sulphate (these are supposed to form less active sulphonates), syrup of lime, and even vinegar. The manner in which the last-named acts is not explained, but it has been strongly recommended by some persons. After the severe symptoms have abated, the stomach should be washed out with tepid water. It is not advisable to attempt to produce vomiting either by emetics or by hypodermic use of apomorphine.

Henry Leffmann.

PHENOL-SODIUM SULFORICINATE is a yellowish liquid soluble in water and alcohol, and recommended by Von Tovolgyi for tuberculous laryngitis. Used like lactic acid without preparatory anæsthetization of the throat, it reduces the tuberculous infiltration and favorably influences the dysphagia. It has also been used for diphtheritic throats and in skin diseases. *W. A. Bastedo.*

PHENOLURIA. See *Urine, etc.*

PHENOSAL—$C_6H_4.OC_2H_5.NH.CO.CH_2.O.C_6H_4COOH$—is the aceto-salicylate of phenetidin, and occurs in sparingly soluble needles or plates of acidulous taste. In the alimentary tract it breaks up, yielding fifty-seven per cent. of phenetidin and thirty-four per cent. of salicylic acid. It is antipyretic, and is especially recommended in rheumatism. The dose is 0.3–0.7 gm. (gr. v.–x.) three or four times a day. *W. A. Bastedo.*

PHENOSALYL.—This compound antiseptic is the result of a series of experiments upon various antiseptics by Dr. de Christmas, in the Pasteur Institute, Paris. He has shown that when certain antiseptics are associated together in one and the same solution, the microbicide power is greater than that of the sum of the solutions of each acting separately. The preparation to which he has given the name phenosalyl is considered by him to be a most efficient antiseptic, its action on the various bacteria being exceeded only by sublimate. It has the following composition: Carbolic acid, 90 parts; salicylic acid, 10 parts; lactic acid, 20 parts; menthol, 1 part.

The three acids are heated up to the point of liquefaction, when the menthol is added. It is very soluble in glycerin, and in water to the extent of four per cent.

Beaumont Small.

PHENOSUCCIN—pyrantin, para-ethoxy-phenyl succinimide, $C_6H_4.OC_2H_5.N(COCH_2)_2$—obtained by the action of succinic acid on para-amido-phenol, occurs in colorless needles which are insoluble in water and ether, but soluble in alcohol and acetic acid. It is antipyretic and antineuralgic in dose of 1–3 gm. (gr. xv.–xlv.) daily, clinical experience showing that it has no depressing effect except in large quantities. The sodium salt forms a sweetish solution with water. *W. A. Bastedo.*

PHENYL-ACETIC ACID—alpha-toluic acid, $C_6H_5.CH_2.COOH$—is obtained by boiling benzyl cyanide with potassium hydroxide solution. It occurs in white glassy scales of burning aromatic taste and soluble in hot water and alcohol. It is given in dose of 0.06–0.15 gm. (gr. i.–iiss.) with cod-liver oil for tuberculosis of the lungs.

W. A. Bastedo.

PHENYLHYDRAZINE — $C_6H_5.NH.NH_2$. — a colorless oily liquid which solidifies into tabular crystals. It is slightly soluble in water. An hydrochloride forms in colorless scales which are readily soluble in water. Phenylhydrazine is an intermediary product in the preparation of many antipyretics, notably antipyrin and hydracetine, but its toxic action is too marked to allow of its employment as a therapeutic agent. *Phenylhydrazine levulinic acid*, under the registered title of *antithermin*, was employed as an antipyretic in doses of five grains. It is now but little used, as its action is uncertain, and is not so safe as that of antipyrin and other similar preparations.

Phenylhydrazine is best known as a test for the presence of sugar in urine, and is known as Fischer's test. It was discovered by Prof. Emil Fischer, and depends upon the property of the sugar forming, in the presence of phenylhydrazine, crystals of phenylglucosazone.

Beaumont Small.

PHENYL-SALICYLIC ACID—ortho-oxy-diphenyl-carboxylic acid, $C_6H_5.OH.C_6H_3COOH$—is a white powder, slightly soluble in water and more so in alcohol and glycerin, and is employed as an antiseptic dusting powder.

W. A. Bastedo.

PHILADELPHIA, PA.—Philadelphia, founded by William Penn, was the first capital of the United States. The population was estimated January 1st, 1903, at 1,347,712. The city, situated at the confluence of the Delaware and Schuylkill Rivers, in latitude 30° 57′ north, is nearly on a line with Madrid and Lisbon, and is about sixty miles from the sea in a direct line. The intervening portion of the State of New Jersey is almost a level plain, abounding in pine, oak, and other timber of second growth. To the westward the land rises gradually and the nearest mountain ridges are from fifty to one hundred miles distant. The highest elevation within the limits of Philadelphia is 450 feet. The mean annual temperature is 53° F., with extremes of —6° F. (1889) to 103° F. (1901). The extremes in 1902 were 12° F., February 5th, and 95° F. July 9th. Precipitation, 49.76 inches. Days with precipitation of 0.01 inch or more, 128. Snowfall, 32.2 inches, distributed as follows: January, 7.7 inches; February, 14.3 inches; March, 4.2 inches; December, 6 inches.

Clear days, 131; partly cloudy, 103; cloudy, 131. Thunderstorms, February, 1; March, 3; April, 1; May, 3; June, 8; July, 13; August, 9; November, 1; total, 39.

The prevailing direction of the wind was northwest and the maximum velocity was 52 miles an hour, from the north, on December 5th. While the mean temperature for the year is about 5.7° F. higher than at London, the mean for January is 3° F. lower, and for July 15° F. higher. The relative humidity at 8 A.M. and 8 P.M. is 75 per cent. and 68 per cent. The rainfall averages 43 inches, considerably greater than that of London (24.84 inches). London, however, exceeds Philadelphia, as well as New York, Boston, Chicago, and all the principal cities of the United States in the number of rainy days. There are about 129 clear days each year in Philadelphia, which is less than at Baltimore (141), at Denver (150), or at Phœnix, Arizona (259). The spring opens in Philadelphia two or three weeks earlier than at Boston, and autumn lasts longer. Roses may bloom through November. The winters are not generally severe. Comparatively little snow falls, yet there may be days or weeks of temperature below the freezing point. Philadelphia has 36 parks, the largest, Fairmount Park, containing over 3,300 acres, through which flows the Schuylkill River, spanned by four bridges. Within the limits of the park the river reaches a width of about one-fourth of a mile. At the northernmost boundary of the East Park the romantic Wissahickon stream empties into the Schuylkill, and the beautiful paths along its borders are favorite resorts for driving, riding, cycling, and walking, while the well-wooded hills that rise just beyond are attractive places for picnics. There is excellent boating on the Schuylkill and on the Wissahickon. Throughout the park at convenient places are houses of rest, restaurants, dairies, and playgrounds. The natural beauties of the grounds are preserved as far as possible. At the southwestern border of the park is the extensive Zoological Garden.

Fairmount Park is of inestimable value to the citizens of Philadelphia, and doubtless exerts a controlling influence on the death rate, particularly among children.

The general death rate of Philadelphia in 1902 was 17.67 per 1,000 population. It was lower than that of New York City (18.74), and reflects credit on the energetic measures adopted by the Bureau of Health. Careful and minute attention is paid to disinfection after contagious disease has been reported. Vaccination has been vigorously carried out. During the past year (1902) the deaths from smallpox numbered 231; from scarlet fever, 143; from diphtheria, 435; from diseases of the heart, 1681; from pulmonary tuberculosis, 2845, and from pneumonia, 2976. The deaths from consumption have recently fallen to second place, owing to a wider knowledge of the principles governing the spread of the disease and to the distribution of pamphlets showing how the disease may be prevented. The Pennsylvania Society for the Prevention of Tuberculosis has assisted in this way to limit the spread of the disease.

Great good will accrue from the recent gift of $1,000,000 by Mr. Henry Phipps for a systematic effort in Philadelphia to eradicate tuberculosis by the establishment of

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.