when the fever begins to decline. After that time, many require it, first in wine whey, half a wineglassful about every three hours; later, when weaker, brandy or whisky punch;—a tablespoonful of brandy, for instance, every four, three, or two hours, sometimes every hour, with the same or twice as much of milk. Beef-tea is indispensable in nearly all cases, from the second week. It may alternate with punch, hour by hour. As in typhus, a patient prostrated with severe typhoid fever should be waked from sleep to take the required nourishment, night and day; otherwise he may sink for want of it.

Quinine, I am satisfied, has no place as a *curative* of this fever. It is useful as a tonic after the critical period of the passing of the height of the fever; not more than eight or ten grains (in one or two grain doses) in twenty-four hours [F. 2].

In the first ten days, headache and heat of the head may call for the application of cold to it; sometimes for leeches to the temples or back of the neck. Dryness and heat of the surface of the body may be best allayed by sponging all over (one part only uncovered at a time) with tepid whisky and water. This operation, done in the evening, will promote sleep. Dr. Drasche,[1] of Vienna, extols the systematic use of cold water externally, to lower the temperature, which is morbidly increased in fever. Brand, Liebermeister, Ziemssen, Wilson Fox, and others have applied this remedy extensively, and with reported great success. Ziemssen's *gradual* method is the best: immersing the patient, when the heat is excessive, in a bath at 95° F., the temperature of which is gradually lowered ten or even twenty or more degrees, according to the effect produced. In this way, morbid heat may be lessened, without shock or undue depression.

Great tenderness of the abdomen may be treated by application of large poultices of hot mush, with which one-fourth or one-fifth part of mustard has been stirred. Diarrhœa being a symptom of the disease, it needs not to be checked unless the passages number more than three or four a day, or are uncommonly copious. Then, a pill of tannic acid and opium (3 grs. of the former to gr. ¼ of the latter), *pro re nata*—or *small* doses of paregoric or laudanum, will generally reduce it. Rarely is it necessary to use laudanum and starch enemata, or to add acetate of lead to opium in pill. Hemorrhage from the bowels is not apt to continue long, or to be dangerous. If it should, astringents, as lead and opium, by enema or by the mouth, must be used.

Shall we attempt to *medicate* the affection of the glands of Peyer? This also being symptomatic, its palliation only appears to be indicated. I am not satisfied that any special treatment for it is demanded in mild ordinary cases. But if, after the tenth or twelfth day, the *defervescence* does not take place, and restlessness is great, with abdominal tenderness, a dry tongue and considerable diarrhœa, oil of turpentine is recommended by authority (Wood) and experience. The dose should be not more than ten drops four times daily in mucilage, with a few drops of laudanum. Nitrate of silver is used instead by some. I have had no experience with it; but I

[1] British Medical Journal, Feb. 19, 1870.



have often seen the good effects of turpentine. It seems to act as a local alterative to the ulcerated surface of the bowel. Pécholier, of Montpellier, gave creasote, 3 drops daily, diluted with lime-water and orange-flower water, to sixty hospital patients, with typhoid fever, as an "anti-zymotic" remedy. He asserts that it lightened and shortened the attack.

Attention to the state of the bladder, day by day, to prevent or relieve retention of the urine, is important. Long-protracted cases may demand a great deal of care to avoid severe bed-sores. In anticipation of these, when threatened, frequent changes of position should be made, and the parts should be bathed with whisky, spirits of camphor mixed with olive or lard oil, or soap liniment. The bed-clothes must be kept smooth under the person. Adjustment of pillows, with the addition of small ones made for the purpose, may do much. When a part is unavoidably pressed upon, it may be protected by a piece of kid spread smoothly with soap plaster. Actual excoriations must be treated like ulcers—with simple cerate, lime-water, poultices, adhesive plaster, etc., according to their condition.

## PLAGUE.

Of this oriental disease, now fast being extinguished, little need be said here. It was probably plague of which Thucydides gives account at Athens during the 2d Peloponnesian war. Livy describes an epidemic, probably of plague, at Rome; Procopius, in Egypt and Palestine, in the 6th century; Boccacio, at Florence, in the 14th century; and Defoe, in London, near the end of the 17th. Its last outbreak at Marseilles, in 1720, destroyed nearly half of the population. Before that time, it had occasionally reached Paris also, and some of the German cities. Plague is a zymotic affection, allied to the fevers, of rapid course and great mortality. Its symptoms are debility, restlessness, fever, delirium, vomiting, hemorrhages, petechiæ, and glandular swellings, especially in the axilla, or carbuncles. Death often takes place in two or three days.

Plague was once thought to be the most contagious of diseases. Excellent reasons are given, however, for believing it not personally contagious at all; but locally infectious. Not quarantine, but sanitary police and hygienic improvements in the great cities (Cairo, in Egypt, for example) have almost put an end to it.

In **treatment** of plague, diaphoretics, opiates, and mineral acids are best reported of. Polli's treatment with the sulphites might be tried in it with propriety; and also that with carbolic acid in small doses.

## ERYSIPELAS.

**Synonyms.**—*St. Anthony's Fire; Rose.*

**Varieties.**—*Traumatic* and *idiopathic.*[1]

**Symptoms.**—These are both local and general. Sometimes the

[1] Billroth asserts that erysipelas *always* depends on morbid lymphatic absorption. This is doubtful; since the degree in which the lymphatic vessels are involved varies much in different cases.

former and sometimes the latter appear first. *Idiopathic* erysipelas generally begins with an ill-defined cold stage, followed by fever. The eruption most often commences on the face, with soreness to the touch, and redness; which spread like a slow conflagration, from part to part. This character of continuous diffusion or *spreading* is pathognomonic. Heat and moderate swelling attend the eruption. It may extend almost all over the body. It may also be superficial and transient, or the inflammation may involve the subcutaneous cellular tissue (especially on the limbs), causing suppuration and sloughing.

The fever of erysipelas has no special features, nor has the disease any definite period of duration. When the scalp is the seat of the eruption, delirium is common, and inflammation of the brain, or fatal coma, may follow. Otherwise, the danger of the disease seems to be from suppression of the function of the skin, and exhaustion. Traumatic or secondary erysipelas combines the danger of the disease itself with that of the injury, abscess, or other local affection from which it starts. *Sthenic* and *asthenic* forms or types of the disease may be discerned, according to constitution and circumstances.

Erysipelas is often destructive in surgical hospitals, as an endemic or infectious malady. Ventilation and cleanliness will do much towards its prevention. Absolute contagion is not proved of it; but the theory of "continuous molecular change" (Snow) applies very well to it. The immediate promotive cause of it would seem usually to be *accumulation of effete material* thrown off from the human body in connection with *inflammation*. It appears most probable that erysipelas is, pathologically, a *capillary lymphangitis*, *i. e.*, inflammation of the minutely distributed lymphatic vessels of the skin.

**Treatment.**—As above remarked, erysipelas may be more or less sthenic or asthenic. Thus we may account for the diverse views and results of its treatment. It is very common now to treat erysipelas with free stimulation. And yet I do not remember ever to have lost a case of erysipelas, in which life was not already in serious danger from a previous injury—either in private or hospital practice. Nor have I, in more than a very few out of a large number of cases, found occasion to give any alcoholic stimulant whatever.

I have commonly begun the treatment of an attack of the disease with a mild saline cathartic—as a *small* dose of Epsom salts, or one of Rochelle salt or citrate of magnesium. Then blue pill with ipecac, if the stomach be good (gr. $\frac{1}{2}$ of the former with gr. $\frac{1}{3}$ or gr. $\frac{1}{4}$ of the latter, every three hours) and neutral mixture or liquor ammonii acetatis. *Asthenic* cases appear to gain by the free use of iron—twenty drops of the tincture of the chloride every three hours. After C. Hamilton Bell, some practitioners use tincture of chloride of iron in *all* cases of erysipelas. I am not convinced of the propriety of this practice.

Locally, mild emollient applications are the best, except as *cordons sanitaires*, or lines of demarcation. At the very start, lard, tallow, or cold cream may almost "put out the fire" at once. Mucilage of slippery elm bark, or of flaxseed, and diluted lead-water, are all that my experience justifies for application to the eruption itself.[1] I would not try to *suppress* it. I think I have seen one death result in the practice of another physician from the attempt to do this with nitrate of silver over a large surface; cerebral congestion and coma took place.



To *head off* the eruption is perhaps only worth while when, from the face, it is spreading to the head. Tincture of iodine, or strong solution of nitrate of silver may, for this purpose, be painted in a line of half an inch in width; or a narrow strip of fly blister may be put on.

Lately (1874) a seemingly heroic practice (Hüter) is said to have been successful; viz., the hypodermic injection of a two per cent. solution of carbolic acid near the inflamed parts; avoiding, however, the most vascular tissues, for fear of carbolic acid poisoning.

When, in traumatic erysipelas, a limb is greatly swollen and inflamed, threatening destruction of the subcutaneous tissues, long incisions through the integument to relieve pressure and congestion may be justifiable.

A milk diet is usually suitable in this disorder.

## PUERPERAL FEVER.

In the time succeeding confinement, liability always exists (besides the transient "milk fever" about the third day) to *metritis*, and, more often, *peritonitis;* also, but with much less frequency, to that asthenic febrile affection, to which the designation *puerperal fever* is best given.

As this belongs rather to *obstetric* practice, I propose only a brief allusion to it. Beginning with a chill, its symptoms are, fever, with an extremely rapid pulse, pain in the abdomen, and tenderness on pressure, or on motion, as in drawing up the knees; tympanites, often; and a day or two later, vomiting, delirium, and tendency to collapse. Death may occur within a week; and more than half the cases are fatal. Sometimes the pain and tenderness of the abdomen are slight or temporary only; the general debility proceeding still to the fatal end.

**Autopsy** shows in much the larger number of cases the manifest lesions of peritonitis; serum, lymph, with extensive adhesions, or pus. In a few cases, however, these are absent entirely. Inflammation of the uterine veins has been met with.

In **causation**, it is observed that nearly all the cases of this disorder (distinct from simple *peritonitis* of the lying-in-room) take place in towns, or in hospitals, especially those which have surgical as well as obstetrical wards. Puerperal fever is many times *endemic* in such localities. Physicians have been beset with it, in some instances, in practice, as a "private pestilence;" every woman attended by one practitioner, for months together, being attacked by it; when others have none of it. Hence we infer two or three things.

One, that this fever has a material zymotic cause, which may be

[1] Solution of *bisulphite of sodium* has lately been used, internally and externally, with great asserted advantage. A solution of *camphor in ether* is said to have proved useful in some cases.

localized. Another, that the *materies morbi* seems to be conveyable by hand from person to person. Although disputed by eminent authorities, the evidence preponderates in favor of this opinion. Some such evidence has come immediately to my own knowledge. Further, several morbid poisons appear, in the peculiarly susceptible, *quasi-traumatic* state of the womb and abdomen after delivery, to promote the disease. *Erysipelas* does so, or at least the conditions productive of erysipelas; also, the typhus poison; perhaps that of smallpox and scarlatina, etc. As to erysipelas, it presents a close and striking analogy with puerperal fever. Thus:—

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|---|---|
| *Erysipelas* is an acute febrile disease, occurring most often in surgical hospitals, in which a peculiar diffusive inflammation is a prominent characteristic; the seat of this inflammation being the *skin*, and connective tissue. | *Puerperal fever* is an acute febrile disease, most common in lying-in hospitals, in which a peculiar diffusive inflammation is a prominent characteristic; the seat of the inflammation being the uterine veins and *peritoneum*. |

**Pathologically,** some questions are not yet entirely decided. Is puerperal fever a special disease, with one specific morbid material cause or virus? or is it a *cachœmia*, which *any* morbid poison has power to produce during the lying-in state? or, again, is it an *ichorhœmia*, from absorption of foul matter from the cavity of the uterus by its semi-patulous sinuses? or a *pyœmia* from inflammation and suppuration of the uterine veins?

I am not ready to answer these questions. Perhaps the *ichorhœmic* theory has the most of evidence at present in its favor; adding to that the hypothesis of "continuous molecular change," alluded to already in another place. Dr. Fordyce Barker believes the disease to be a specific disease, entirely peculiar to the puerperal state.

Practically, sanitary measures of precaution are clearly indicated to *prevent* puerperal fever. Lying-in hospitals must be great evils, rather than benefits, unless they have the best possible situation, construction, and administration. And no such hospital, or ward, should ever be under the same roof or in the same inclosure with a surgical ward or hospital. Moreover, in private practice, attendance on delivery by a physician who is visiting at the time a case of puerperal fever or of erysipelas, is at the risk of the patient; if the danger of conveyance of the disease be removable, it is only so by the most careful and thorough cleansing and disinfection. The clothes should be changed, and the hands washed in strong solution of chlorinated soda or carbolic acid, before making such a transit from the one patient to the other. Several physicians in this city always decline confinement cases under the circumstances named.

The **treatment** of puerperal fever has often proved unsatisfactory. I had intimate knowledge of the experimentation to which it was subjected in the wards of the Pennsylvania Hospital by Drs. Meigs and Hodge, between 1845 and 1849. Venesection, purging, and mercurials, etc., were tried amply, and failed most signally.



Quinine in tonic doses, with beef-tea, and, if collapse be threatened, alcoholic stimulation, has, though far from always successful, had at least better results. Leeching the abdomen freely, at the beginning of the attack, in the least asthenic cases, does important good. After the leeches, for a day, warm poultices may be applied, then a large blister. Sulphites and carbolic acid are worth trying in this disease. Béhier, Winckel, and Joulin[1] speak well of the application of ice, in a bladder, to the abdomen, continued until a reduction of the temperature has been obtained.

For the personal prophylaxis of puerperal fever, obstetricians of authority confirm from experience the reasonable view, that it is of great importance to empty the womb, and if possible the vagina, thoroughly, after child-birth. Good uterine contraction is indispensable as a safeguard. Washing out the vagina, within a few hours of delivery, with lime-water or solution of glycerin, may also be recommended for a similar end.

## CHOLERA.[2]

**Synonyms.**—*Epidemic, Spasmodic, Malignant, Asiatic, Indian Cholera; Cholera Algida; Cholera Asphyxia; Mordshi.*

**Symptoms and Course.**—Premonitory diarrhœa, mostly painless and watery, occurs in most, but not in all cases. Its duration varies from an hour or less to two or three days. The worst epidemics of cholera have been marked by some cases of fearful rapidity. In India, in a few instances, death has resulted, by collapse, in ten minutes.

Commonly, the diarrhœa increases in frequency and copiousness, and in a few hours vomiting commences. The discharges are colorless or "rice-water" like, and are spirted out with spasmodic force. The skin grows cold by degrees, and great debility comes on, with cramps in all the limbs usually. The temperature in the *rectum* and *vagina* has been several times found to be higher than natural. Dr. D. W. Parsons[3] asserts a constant difference of about 8° between the axilla and the tongue; the latter being the colder.

If not checked, *collapse* arrives; with intense thirst, oppression in breathing, loss of voice, disappearance of the pulse, suppression of urine, cold, *blue*, and shrunken skin, sometimes bathed in sweat, and, at last, cold breath; ending in death. This occurs, on the average, in about eighteen hours.

When reaction takes place, recovery may immediately become complete, or a low fever may supervene. The termination of this may be in death within a few days, or recovery in a week or two.

**Appearances after Death.**—*Rigidity* occurs *soon;* sometimes in less than an hour; generally within two hours. Startling *movements* of the corpse have been several times noticed; as of a patient, dead with cholera, slowly lifting both hands over the chest and

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[1] Am. Journal of Med. Sciences, April, 1871, p. 504.

[2] See, for the fuller statement of the author's views upon this disease, his essay, entitled "Cholera: Facts and Conclusions as to its Nature, Prevention, and Treatment." Philada., 1866.

[3] Liverpool Medical and Surgical Reports, Oct. 1871.

joining them; opening the eyes and rolling them downward, etc. *Increased heat* of the body, cold during the attack, has been sometimes observed after death. Internally, several of the great organs, the brain, spleen, and kidneys, at least, are commonly gorged with blood. So are the *right* cavities of the heart; but the left side of the heart is empty or with but little blood, and firmly contracted. The lungs are almost bloodless. The liver varies in appearance; but the gall-bladder is almost always *full of bile.* The urinary bladder is, constantly, greatly contracted. The stomach and intestinal canal are congested and swollen; the late Prof. Horner observed the frequent throwing off of the "epithelial" lining of the canal; Böhm, of Germany, confirmed this; Drs. Parkes, Gull, and Lindsay assert it to be a post-mortem occurrence.[1] The intestinal glands are found considerably enlarged. The *blood* has been carefully examined by Drs. Garrod, Schmidt of Dorpat, and others.[2] Its water and salts transude into the alimentary canal, with some of the albumen and fibrin; also *the contents of the blood-cells transude into the serum.* The blood drawn from a vein during life is (as I have seen it) dark, thick, and tarry, scarcely capable of flowing. Schmidt found the amount of oxygen in the blood-corpuscles less than half the normal proportion. The blood is *acid* sometimes in cholera; the reverse of its natural reaction.

The *ganglia* of the "sympathetic" system have been often examined, and are frequently changed in appearance; congested, softened, altered in color; but no *special* change has been *shown* to belong to them in cholera.

**Diagnosis.**—Common cholera morbus alone, when severe, resembles epidemic cholera so much as to be easily mistaken for it. The *collapsed* stage of the one, preceding death, is almost identical in appearance with the collapse of the other. But cholera morbus is *caused* by some irritant of the stomach and bowels, and is clearly an affection of *those organs*, not a *toxæmia* (*toxæmic neurosis*) or systemic disorder; it is sporadic, *not epidemic;* in it the discharges are always *bilious* at first, and mostly so to the last; collapse in any degree is *rare*, and death, under judicious treatment, very uncommon. In all these things, it differs greatly from Asiatic cholera.

**History.**—Putting aside some possible resemblance to this disease in descriptions of Aretæus and one or two other ancient authors, probably the epidemic in France of 1545, "*trousse-galant,*" came more near to it. The earliest distinct accounts of cholera were given by D'Orta, at Goa, 1563, and Bontius, a Dutch physician of Batavia, 1629. Willis (1684), Morton (1692), and others, described epidemic fluxes and "dysenteries" in England in such terms as strongly to remind us of cholera; and so did Degner, of Nymwegen, in the Netherlands (1736), and Morgagni in Italy, in 1733. Some British physicians (Greenhow, Aitken) now believe that cholera may have repeatedly visited England. It appears to me more probable, however, that this opinion is due to an over-estimate of the resemblance between the autumnal

[1] Edinburgh Med. and Surg. Journal, Jan. 1855.
[2] Brit. and For. Medico-Chirurgical Review, July, 1854.



cholera morbus of Great Britain (like our own) and the pestilential disease.

Certainly cholera must have existed in India for an indefinite time.[1] From 1781–2 dates its extended prevalence, in a most destructive form; at Calcutta, in Madras, on the Coromandel coast, and in Ceylon.

In August, 1817, Jessore was the birthplace of the first great migratory epidemic. Shortly after, in Calcutta, 36,000 were attacked in three months. At many military stations, it was very severe. Roads were covered with dead and dying, unable to reach their homes. In November, the grand army of the Marquis of Hastings was devastated by it. Of 90,000 men, in twelve days 9000 had died. On marching the army across a river to dry and elevated ground, the commander was relieved of this otherwise invincible enemy.

In 1818, the Birman empire was invaded by cholera; and there and elsewhere in Asia, its ravages were fearful. In 1819, 150,000 died of it in the Presidency of Bombay. It also reached Mauritius, 20° S. latitude, three thousand miles from any place before visited by it. The Island of Bourbon was visited in 1820; as well as the Philippine Islands. In 1821, Borneo and Java were affected; and a large Persian army was repulsed by it from before Bagdad, without a battle. In 1822 its limits were much narrowed, and its destructiveness abated.

India almost escaped in 1823, but China was ravaged by it; and it extended northwestward, in that year, to Orenbourg, on the Ural, near the borders of Europe and Asia. In 1826 it passed the great wall of China in its northwest progress; but almost left Western Asia. It reappeared in Persia in 1829.

Orenbourg was revisited in that year, and the epidemic there lasted from August till near the end of February. This city had a population at that time of 11,000, of whom 6000 were soldiers. Those first affected had no communication whatever with any infected place.

1831 saw the cholera in the north of Europe, as far as Archangel, near the Arctic Ocean, more than 64° N. latitude. It reached Warsaw in April, during an insurrection, and was very fatal. Hungary suffered from May to September; losing 100,000 of its population. In June, St. Petersburg, and in September, Moscow, were reached by the pestilence. Berlin had it also for three months and a half, beginning in August. Mecca was attacked during the visitation of throngs of pilgrims, in May; of 50,000, as many as 20,000 are said to have perished. In this year, while Hungary was infected, the Austrians surrounded Vienna by a double *cordon militaire;* but in vain. The disease began there in August and continued for three months. The southern provinces of Austria and the Rhineland were exempt. Constantinople was affected by it, but not with very great severity. The Turkish government, that year, maintained no quarantine. Cairo suffered dreadfully in 1830–31; and so did Smyrna.

[1] Professor Martin Haug has found some distinct references to it in ancient Sanscrit writings. Paisly (1774), Girdlestone (1782), Causis (1794), and Jameson (1820) were early English authors upon it.

Attacking Hamburg on the 11th of October, 1831, it was officially announced at Sunderland, England, October 26th. It had occurred in several cases in England months before. Three or four weeks later it appeared at Newcastle ; and, in December, at Haddington, a Scottish town on the Tyne.

Edinburgh and Glasgow first had cholera in January, 1832 ; London in February ; Dublin and Paris in March. London then suffered moderately ; Paris terribly—especially in April and May ; 20,000 deaths.

On the 8th of June, it first invaded our continent, at Quebec ; and within a week at Montreal. In the same month it was in New York and Albany. Philadelphia had its first cases in July. Between the 1st of July and the 18th of August, New York had reported 5337 cases, with 2068 deaths. That city lost 3513 in all.[1] From the 27th of July to August 18th, Philadelphia had 1610 cases, with 615 deaths. Boston and Baltimore were moderately affected in August.

Detroit, Buffalo, Elizabeth City in North Carolina, Wilmington and Newcastle, Delaware ; Norfolk and Portsmouth, Virginia ; and New Orleans, were the principal of more than fifty towns in the United States reached by cholera in 1832. It had entered twelve different States before September.

Havana and Mexico were attacked in the spring and summer of 1833. The *City* of Mexico, notwithstanding its great elevation above the sea, did not escape.

Portugal was also first visited in that year, Spain but slightly until 1834. Northern Italy was affected in the autumn of the same year. In 1835, Alexandria and Malta ; in 1836, Rome, Naples, Egypt, and Central America, especially suffered. North Germany, South France, Rome, Naples, Sicily, Malta, Egypt, and Syria, in 1837. After that, cholera disappeared from Europe and America for nearly ten years. It still existed, with variable violence and extent, in India.

In 1847, it ravaged a Russian army west of the Caucasus ; and in September it returned to Moscow. In 1841, Turkey, Russia, Austria, Prussia, Belgium, Holland, Great Britain, and France (though not Paris) were successively attacked. Then the cholera showed its power to traverse the sea without human aid or agency, by attacking two emigrant ships, a thousand miles apart, one sixteen and the other twenty-seven days out from Havre, when *no cholera was prevailing at that port.*[2] The *cholera-cloud* itself also reached New Orleans about the same time, and progressed up the valley of the Mississippi. New York was not affected by the visit of the infected ship ; the disease not occurring again there until May, 1849.

Paris was reached by it in February of that year, but suffered the worst in June. Lyons now had it for the first time. Tunis and Algiers were visited toward the end of the year.

In January, 1849, after Memphis, St. Louis, Missouri, was attacked. Chicago, Buffalo, and other towns on the lakes in May. New York and Philadelphia in the same month. Baltimore had this year only a local epidemic, in July, in the Almshouse ; the restriction of which to one side of the building was very remarkable. As in 1832, the mortality in Philadelphia was much less for the population than in New York : 1022 deaths occurred in our city ; New York had a mortality 450 per cent. greater.[1] Canada was reached this time from the westward.

[1] In 1834, New York lost 971 ; in 1849, 5071 ; in 1854, 2509. Dr. A. Clark, Lect. on Cholera.

[2] Report on Cholera in the United States, by Dr. James Wynne ; and Dr. Gavin Milroy, Brit. and For. Medico-Chirurg. Review, Oct. 1865, p. 444.



In 1848–49 the number of deaths from cholera in England and Wales was over fifty-four thousand (54,398) ; in 1832–3, nearly thirty-one thousand (30,924). In London,[2] probably owing to greater attention to sanitary means, the mortality was two-fifths less the second time than the first. Some parts of Southern Rhineland were visited in 1849 ; especially the filthy city of Cologne.

Cholera lingered in various places, almost sporadically, in Europe and America, from 1850 to 1854. Canada and the far West (Indiana also had cases every year) suffered the most in this way, on our continent. In the West, emigrants' camps and military stations seemed especially to furnish its required local conditions.

In 1853, Persia had it severely ; also some part of Northern, Central, and Southern Europe (Copenhagen, Hamburg, Berlin, Piedmont, Lyons, Paris, and Southern Portugal). Before the end of the year it was again in New York, New Orleans, and the West Indies. Mexico had been visited in the spring, and through the summer.

1854 was still more a cholera year in Europe and in this country. Scarcely any European state or kingdom was exempt. The French, English, and Russian troops suffered from it much in the Crimea. Greece, Italy, Germany, France, Spain, Portugal, in short, all Europe was traversed by it : 150,000 died of it in France alone ; in England and Wales about 20,000. Newfoundland, on our side of the ocean, was reached for the first time in 1854. This was the year of the epidemic at Columbia, Lancaster County, in this State; so remarkable for the absence of *some* of the usual promotive conditions of cholera. Our great cities, however, did not suffer nearly so much as in 1849.

In 1855, the disease was widely spread in Europe, though not very malignant except near the seat of war, before Sebastopol. Egypt and Palestine had it also. In Switzerland, which had been slightly touched before, Basle, Geneva, Zurich, and other places now suffered by it. The next year, 1856, still did not witness its withdrawal from Europe.

Since that period until 1865 I have no means at hand for tracing the movements of epidemic cholera. Dr. Gavin Milroy says that the countries hitherto exempted have been as follow : Australia, New Zealand, and other islands in the Pacific; the Cape of Good Hope and adjoining settlements ; the coast of Africa from the Cape as far northward as the Gambia, and including the islands of St. Helena and Ascension ; the Azores, Bermuda,

[1] Dr. J H. Griscom, Medical Record, March 15, 1866, p. 35.

[2] London had 13,098 deaths from cholera in 1849 ; in 1854, about 10,000.

Iceland, Faroe Islands, and also the Orkney and Shetland; the southern half of the eastern coast of South America, from the Rio Plata inclusive, Cape Horn, and the whole of the western coast of that continent, from the Cape and along the shores of Chili and Peru to Panama.

In 1865 every one was familiar with the accounts of cholera in Arabia and Egypt in the spring, at Constantinople in July,[1] and afterwards in several parts of Europe, extending, though with but moderate violence, as far as England. While its vast migrations seem to be as capricious or incalculable as the flight of birds or insects, two local causes contributed at least to its *severity* in Mecca and on the Nile. These were the crowds of religious pilgrims at the former place, in the spring, and, in Egypt, the insalubrious circumstances attending the operations at the new Suez Canal. In both, "crowd-poison" was intensified to the greatest degree; so that the pest-cause might well find there strength for the renewal of its flight onward to the northwest. In Paris, in 1865, 6383 deaths occurred.

In 1866, Europe was invaded by cholera from the south. In June it was in Egypt; then in Constantinople, Malta, Valentia, Ancona, Gibraltar, Barcelona, Madrid, Toulon, Paris, Southampton, Liverpool, and London; as well as, on this side of the Atlantic, in New York, Philadelphia, and other places. In 1867 also it existed, though less destructively, in Europe as well as in the West Indies, Central America, and South America. In 1868, in the early part of the year, it continued in South America, especially in Paraguay and the Argentine Confederation; in which it destroyed more than 40,000 people. It was in part of Russia and also in Cuba in the summer of 1870. In the summer of 1871, it is said to have been very destructive in Persia; and considerably so in some parts of the Russian territory in Europe. By August, it had reached Königsberg, in Germany, threatening a renewal of its westward course; by the beginning of September, being reported as present in Dantzic, Leipsic, and Vienna.[2]

In 1872, also, central Europe suffered by it; most of all in Hungary, Poland, and Prussia. In 1872 more than 80,000 deaths from cholera occurred in Prussia alone. A remarkably localized visitation of cholera occurred in the latter part of the summer of 1873, in a number of towns in several of the United States; especially in Kentucky, Tennessee, Illinois, Indiana, and Ohio.[3]

During the winter of 1873–4, cholera was present in some portions of South America; being especially severe at Buenos Ayres. Central Europe also was not free; 300 deaths at least occurred in Vienna alone, in 1873; in Hungary, more than 100,000 deaths.

I take from Dr. Brigham's treatise (published in 1832) the following table, of the deaths from cholera in 1832, and their proportion to population:—

[1] The first case occurred in that city on the 28th of June.

[2] August 15, 1871, Dr. Edmunds, in a note to the "Times," reported a "typical case of Asiatic cholera" in London; and 6 cases are said to have occurred in Paris in the week ending August 29.

[3] Although Dr. Peters constructed a sufficiently ingenious hypothesis for explaining this epidemic by "introduction," such a view was, in this instance, signally deficient in locally authenticated evidence.



| | Population. | Deaths. | Equal to. |
|---|---|---|---|
| Moscow | 350,000 | 4,690 | 1 in 74 |
| Petersburg | 360,000 | 4,757 | 1 " 74 |
| Vienna | 300,000 | 11,896 | 1 " 159 |
| Berlin | 340,000 | 1,401 | 1 " 242 |
| Hamburg | 100,000 | 446 | 1 " 224 |
| London | 1,500,000 | 1,223 | 1 " 1228 |
| Edinburgh | 150,000 | 72 | 1 " 2033 |
| Glasgow | 180,000 | 395 | 1 " 455 |
| Hungary | 8,750,000 | 188,000 | 1 " 46 |
| Paris | 800,000 | 20,000 | 1 " 40 |
| Montreal | 25,000 | 1,250 | 1 " 20 |
| Quebec | 22,000 | 1,790 | 1 " 12 |
| New York | 200,000 | 2,000 | 1 " 100 |
| Albany | 24,000 | 311 | 1 " 77 |

Supposing the population of Philadelphia to have been at that time 150,000, this with a little over 600 deaths, would give a proportion for our city of 1 in 250 of the inhabitants. In 1849 the ratio was considerably less.[1]

It is an important fact in the history of cholera, that before, during, and after the epidemic has visited a place, many cases, greatly exceeding in number those of typical cholera, occur, of diarrhœa, sometimes also with vomiting, not violent, yielding easily to treatment. To these the name of *cholerine* is often given.[2]

**Nature of Cholera.**—Without discussing opinions at length, it may be asserted that cholera is not at all, like our ordinary cholera morbus, a disorder simply of the stomach and bowels. Being clearly an acute *systemic* affection, changes in the blood are *proved* to occur in it, and may well be believed to be primary; that is, the morbid cause acts through the blood. But this is not all.

Cullen placed cholera, in his nosology, in the class *neuroses*, order *spasmi*. Many medical observers (Binaghi, Loder, Orton, Delpech, Lizars, Coste, Favell, C. W. Bell, Greenhow, G. Johnson, etc.) consider its principal effects to be referable to disturbed innervation, involving chiefly the ganglionic centres of organic life. Dr. Charles D. Meigs, years ago, graphically called the attack the "cholera squeeze." Velpeau, of Paris, lately repeated this, "le mal vous *tortille*." There, I think, is the pathology of cholera, in one word. As Dr. C. W. Bell says, it is not an adynamic, but a dynamic, or sthenic, collapse.

The heart, its left side at least, is, after death, contracted. The pulmonary artery and its branches are narrowed, making the lungs pale and anæmic. The gall-bladder is full of bile, but the duct is spasmodically closed and detains it there. The urinary bladder is shrunken to half its size or less. The bloodvessels of

[1] Moreau de Jonnes estimates the number *attacked* as, in France, 1 in 300 of the population; Russia, 1 in 20; Austria, 1 in 30; Prussia, 1 in 100; Poland, 1 in 32; Belgium, 1 in 120; Great Britain and Ireland, 1 in 131; Holland, 1 in 144; Germany, 1 in 700.

[2] The coincidence or anticipation of cholera by epidemic *influenza* and the potato blight has been several times noticed. But there is, clearly, no uniformity in any such association.

the whole alimentary canal press rigidly upon their contained fluid, and force its serum out into the stomach and bowels; whence it is, by spasmodic ejections, thrown out. The very skin is, by its involuntary muscular fibres, as well as by vascular constriction everywhere, drawn tightly and closely upon the body. The voluntary muscles suffer with cramps. All is cramp, cramp, within and without. The brain is almost in anæsthesia during the collapse—no delirium, but apathy—as from cerebral anæmia. The blood so compressed, grows thick as tar—it scarcely flows, is not aerated, and cyanosis follows; it is detained in the capillary and venous networks of the interior organs, in which congestion is found after death.

Cholera is, then, I say, a poison-spasm; a *ganglionic tetanus.*

**Causation.**—As to this, all cannot yet be known. But it is clear that cholera must have a specific, material, migratory cause. I agree with Dr. G. B. Wood, Dr. Austin Flint, Dr. Snow, of Providence, and some foreign authorities, for example the late Dr. Southwood Smith, "the father of modern sanitary reform," in believing that cholera is *not personally contagious.*

My theory is as follows: that the cause of cholera is a (yet undiscovered) protozoon, or microphyte, of extreme individual minuteness; which, on entering the human body, affects it as an organic poison.[1] That the varying quantity or number of these organisms may in different cases account (along with individual predispositions and exposures) for the unequal violence of different epidemics; as in the case of trichiniasis. Choleraic diarrhœa or cholerine, so frequent *before* as well as during and after the prevalence of cholera, may in some instances at least be explained by the action upon the alimentary canal only, of a minimum quantity of the cause. The dreadful fatality of some Indian seasons, is on the same view referred to an extreme *accumulation* of it.

A most important part of the theory is, further, that which concerns *promotive* causation. What conditions favor and maintain in life, multiplication, and migration, this *ens primalis?*

All the facts answer, I believe, that *animal matter in a state of rapid and foul decomposition*, putrefaction, along with moderately high (not the highest) temperature, and ordinary moisture, will afford those conditions; and that *nothing else* is required to explain the whole history of the propagation and extension of cholera.

[1] Hallier of Jena, Klob of Vienna, and Thomé of Cologne, have asserted the discovery of peculiar fungi in cholera dejections. Thomé gives the name of "cylindro-tænium" to one form. Hallier calls the fungus "urocystis," and ascribes it to the rice-plant. This view is opposed by Berkeley, a distinguished cryptogamic botanist. Prof. H. C. Wood, Jr., energetically disputes it. See Am. Journal of Med. Sciences, Oct. 1868, p. 333. Assistant Surgeon T. R. Lewis, of the British army in India, gives important facts against Hallier's theory. See "Report on Microscopic Objects found in Cholera Evacuations. Calcutta, 1870." E. Semmer (Virchow's Archiv, vol. i. part i. pp. 158–160) asserts partial confirmation of Hallier's views, by the production of the *milzbrand* or splenic cattle-disease, upon the injection of fungus-spores and micro-coccus-cells, cultivated from the blood of animals having that disease. Loesch, of St. Petersburg, reports the observation of *multitudes of cercomonads* in recent choleraic dejections. Not impossibly, such minute organisms, even though not peculiar to that disease, may exert a morbid influence by their *number.* (St. Petersburg Med. Journal, 1871.)



Nothing, I mean, but the admission of the existence of the protozoon, or protophyton, which in germs or in maturity, or both, may fly "on the wings of the wind;" or be conveyed to less distances by water; and, with these the above-named conditions of its vital maintenance, as its food and "habitation."

It is, in my mind, *obvious* that this theory will explain all the facts. I believe, also, that some well-known facts can be explained by it *alone.* Such are the facts which account, by the annual inundations, the crowding of the great fairs, the throwing of bodies into the river, and the inconceivable filth of the inhabitants, for the persistent residence of cholera in the Gangetic delta, while everywhere else it is only an occasional visitant.

In Europe and the United States, as well as in India, influences belonging to closely aggregated communities have always been observed to display a power to propagate cholera. It comes most often, stays longest, and is most destructive, in the densest and filthiest cities, and in the worst quarters of those cities.

Very important testimony exists as to the influence of the *drinking-water* of localities. Dr. Snow, of England, asserted the theory that this was the almost universal medium of its propagation. This has been shown to be quite insufficient. But all such testimony is still available in regard to the propagating and extending power of *animal contamination.* Thus, Bethlehem Hospital, supplied by an artesian well, had, in 1849, among 400 inmates, no case of cholera. It was the *only* large lunatic hospital in London which escaped; as it was the only one supplied with spring water. In the districts of London supplied from the Thames above the entrance of the sewers, the mortality ranged from 8 to 33 in 10,000 of the inhabitants; in those supplied from below the entrance of the sewers, from 28 to 205 of the same number.

In this country, Dr. James Wynne's report[1] affords, upon almost every page, matter of exactly the same purport as the above. In St. Louis, Louisville, Buffalo, New York, Philadelphia, Boston, etc., similar facts were recorded. It is unnecessary to extract them, they are now so familiar and so commonly accepted.

But the assertion that contamination of drinking-water is the *only* medium for the propagation of cholera, cannot be sustained. In India, for example, Dr. Sutherland, statistical officer to the Inspector-General, reports that the statements often made elsewhere concerning drinking-water "cannot be said to apply to the causation of the disease as it appeared in this country in 1867." Dr. Bryden, surgeon in the Bengal army, says that "I have anxiously sought for evidence of the highly poisonous character of cholera evacuations, with an unprejudiced mind. I do not go so far as to say that the evidence is against the presence of cholera germs in the evacuations. I think it highly probable that latrines are occasionally infected, especially hospital latrines."

Prof. Pettenkofer, of Munich, rejects absolutely the (now quite current) theory of the special extension of cholera by means of the stools of patients contaminating drinking-water. Dr. E. M.

[1] Presented to Parliament, and published in 1852.