

Cholera : its protean aspects and its management / by G. Archie Stockwell.

Contributors

Stockwell G. Archie
Royal College of Physicians of Edinburgh

Publication/Creation

Detroit, Mich. : G.S. Davis, 1893.

Persistent URL

<https://wellcomecollection.org/works/y47t5e3e>

Provider

Royal College of Physicians Edinburgh

License and attribution

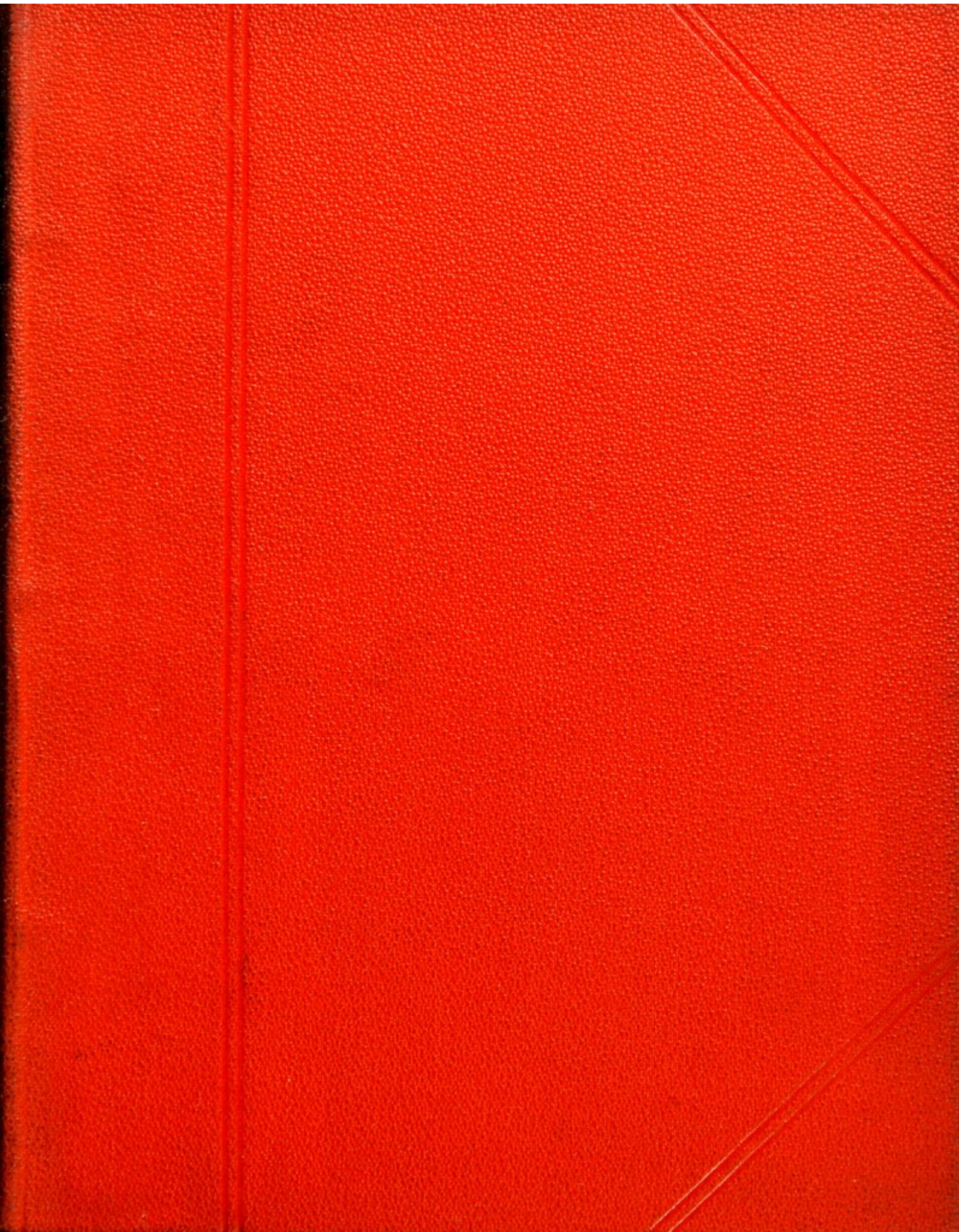
This material has been provided by This material has been provided by the Royal College of Physicians of Edinburgh. The original may be consulted at the Royal College of Physicians of Edinburgh. where the originals may be consulted.

This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

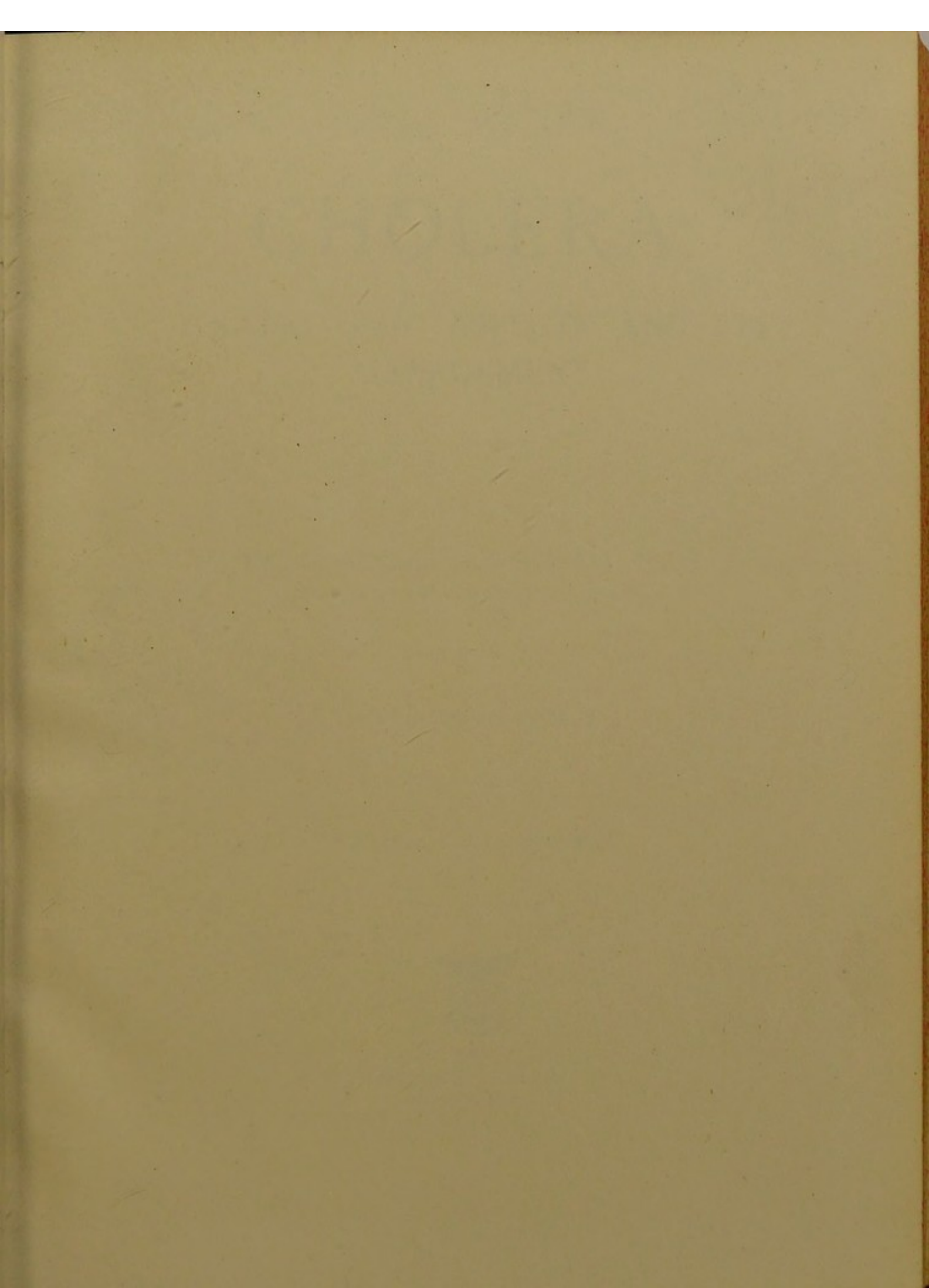
**wellcome
collection**

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>



²⁴Наз. 944

R36774



CHOLERA,

ITS PROTEAN ASPECTS AND ITS
MANAGEMENT.

BY

DR. G. ARCHIE STOCKWELL, F.Z.S.

(Member New Sydenham Society, London.)



IN TWO VOLUMES—VOL. I.

“Respice, aspice, prospice.”



1893.

GEORGE S. DAVIS,
DETROIT, MICH.

Copyrighted by
GEORGE S. DAVIS.
1893.

DEDICATION.

To Dr. Cyrus M. Stockwell, of Port Huron, Michigan—the kindest and best of fathers; the most thorough, exemplary, and painstaking of tutors; the companion of youth and manhood; who early inculcated the necessity of close habits of observation and ratiocination, including self-segregation in all matters scientific—this volume is dedicated in deepest sympathy and love by

THE AUTHOR.

December 1st, 1892

PREFACE.

When this volume was undertaken, on the spur of the moment and at the solicitation of the publisher, it was not expected to be more than an exponent of personal views; but in seeking collateral evidence, I was agreeably surprised to find myself by no means singular in an estimate of cholera—that many had recognized in greater or less degree the rôle played by the nervous system, and even admitted the possibility of profound toxicity of the cerebro-spinal centres. Hence was necessitated a work more extended in scope, and more generally particularized in outline.

Again, I am pleased with the opportunity of contributing my *mite* toward undoing the evil wrought by the greatest medical heresy of any age—a heresy that seeks to elevate to the acme of pathological knowledge, a vain, visionary, theatrical egoist, devoid of even the shadow of medical training. The exponents of bacillar pathology depend solely upon hypothetical assumptions, ignoring all forms of evidence not adduced by themselves. With them the microscope is no longer an accessory to skilled observation, but may supersede the latter altogether. With profound contempt for biológico-zoölogical laws and their applications, factitious maladies, artificially produced, are made to replace real maladies. Their pathology is merely an experimental experience admitting of neither negations or offsets; their therapeutics, a form of still hunt with untried weapons, in an unknown jungle, after a hypothetical prey. Indeed, it is a sad travesty upon medical science when authors and would-be teachers wantonly assert Rabies, Cholera, Yellow-fever, Dengue, Tetanus, Endo-carditis, Pneumonia, etc., are “dis-

eases whose microbic origin is positively known;" when two of these are supported only by manifest fraud, in two more the evidence has never been adduced in any form, and in the other three it is of the most flimsy, superficial character.—The list might be considerably increased as regards the latter. The tendency is to sacrifice truth to temporary self-aggrandizement; to assert individual preferences as established facts, regardless of results, forgetting Science is a stern mistress who permits neither preferences or personalities, and refuses to acknowledge evidence that is not freed from the errors of coincidence and ably supported by negatives.

In conclusion, I will here make special acknowledgment of indebtedness to Doctor Alexander Harkin of Belfast, Ireland, for copies of his researches into the ætiology, pathology and treatment of cholera, which lead anything hitherto published upon this subject; his views are so in accord with my own, that frequently the former have by preference been adopted *in toto*, consequently the chief merit of this work will remain with him—not me.

I have also availed myself freely of the labors of Felix von Niemeyer, Thomas Hawkes Tanner, and Hermann Lebert, whose cholera essays are models of diction and profound epitomes of information concisely expressed; also of the researches of Surgeon-Majors T. R. Lewis, D. D. Cunningham, J. M. Cunningham, E. A. Parkes, and J. C. Hall; Sir Wm. Aitken, Sir Thos. Watson, Geo. Johnson, M. J. von Pettenkofer, G. V. Black, A. M. Brown, Geo. Crookshanks, Rudolf von Jaksch, James Gagney, *et al.*

I will likewise acknowledge special courtesies at the hands of Dr. Thos. C. Minor, of Cincinnati, and the Secretary of the Tennessee State Board of Health, whereby was obtained a large portion of the material embodied in the Appendices.

Thus the volume makes little pretense to originality further than as a condensed rescript from others, supplemented by personal views as evolved by practical observation and experience.

GEO. ARCHIE STOCKWELL.

650 Congress Street E.,
Detroit, Mich.

CHAPTER I.

HISTORY.

In the ranks of the medical profession, no less than in lay circles, the subject of Cholera* is one of supreme and absorbing interest, especially now that, in epidemic form, it is knocking vigorously at our very gates, so to speak, and sedulously endeavoring to force an entrance.

For a number of years Americans have solaced themselves with the idea that modern science had effectually barricaded cholera from Europe, and consequently from the Western Hemisphere, except in the sporadic form that is always more or less prevalent in Mediterranean ports, and occasionally asserts itself in the United States as a concomitant of the heated term. But, while the general progress of sanitation in the civilized world has been considerable, and such as in great measure to remove the faulty conditions under which the disease flourishes, scientists are rudely awakened by the fact the danger line has by no means been eradicated, that the barriers of protection have been silently and unexpectedly forced, and that if immunity of the American continent is to be secured it is only at the price of "eternal vigilance" coupled with more perfect knowledge.

* From *χολὰς*, "the bowels," and *ῥέω*, "to flow"—not, as has been imagined, from *χολη*, "bile," and *ῥέω*.—S.

When are recalled the fearful and weird tales of various visitations, and the universal panic that in each instance has resulted, it is little wonder the public await breathlessly each successive cholera bulletin, fearing the dread malady has broken bounds and, consequently, may at any moment manifest itself in the very midst of the most remote community.

Evidently the disease has prevailed for untold centuries in portions of the Orient, and manifested itself as both an endemic and epidemic in the Indian Peninsulas, where—until quite recently at least—it has ever been regarded as a filth disease. It is only within the last three-fourths of a century, however, that it has manifested any predilection for Europeans, prior to this period being universally regarded as a plague peculiar exclusively to dirty, badly-housed, ill-fed "heathen." In Siam and Burma cholera is always present; in Hindustan it is wont to ravage certain districts with great regularity, being in the main an accessory to religious pilgrimages, shrine-worship, etc., especially when any considerable number of natives are assembled together. It remains a regular visitant at the annual festivals held at the great Conjeveram Pagoda, forty-five miles from Madras, whence the latter city is regularly infected, and at the great gatherings of Allahabad. It was a constant attendant upon "The Jagannath," as held at Puri, a town of 35,000 inhabitants, that was generally healthy in June and July, but invariably developed cholera the succeeding

month with the influx of some 150,000 pilgrims, who filled the lodging-houses almost to the point of suffocation, and crowded even the streets and fields, literally covering both with their urine and excrement the decaying odors of which terribly impregnated the atmosphere for miles about; here the disease claimed its victims, not by scores but by hundreds, yet speedily ceased its ravages on the dispersion of the multitude and subsequent thorough amalgamation of their filth with the soil.

It was only in the early part of the present century that the malady first appeared in Jessur, in Bengal (sixty-seven miles north-east of Calcutta), as an epidemic, infectious, pestilential disorder, and overstepping its natural boundaries began a deadly march northward and westward that did not cease for seventeen years, and is supposed to have claimed nearly 600,000 victims. Sir Archibald Allison thus tells the tale :*

“After the signature of the treaty of alliance with Scindia, November 5th, 1817, the cholera, then for the first time known in British history, broke out with the utmost violence in Lord Hastings’ army, and from the very outset committed the most dreadful ravages. The year had been one of great scarcity; the food crops were of inferior quality; the situation of the British cantonment low and unhealthy. Every-

* “History of Europe,” vol. vi. Edinburgh, 1865.

thing was thus prepared for the ravages of the epidemic, which soon set in with terrible severity. For ten days the camp was nothing but a hospital; in one week 764 soldiers and 8,000 camp-followers perished. At length the troops were removed to higher and more airy cantonments, and upon this the malady ceased—a memorable fact for the instruction of future times. As was afterwards experienced, the ravages of the pestilence were greatest among the lower classes of the people. Only 148 Europeans perished in November, but about 10,000 natives fell victims to the malady during the same period. When it spread to Calcutta it destroyed 200 a day for a long time, chiefly amongst the worst-fed and most destitute people.”

So far as can be gathered, the epidemic that now threatens this country, and is already afflicting portions of Western Europe, in March or April of the present year appeared in the northern portion of hither India, near the head waters of the Ganges. It first manifested itself in a region a hundred miles or so from Mirut, and two hundred and fifty north from Lucknow, among the pilgrims at the great Hurdwar Fair. Thence it moved northward by Lahore into Kashmir, reaching Peshawar, showing itself violently at Srinagar and in the Punjab in May, a month later ravaging Askabad after traversing the whole breadth of Afghanistan. Here, having reached the Trans-Caspian railway, it moved more rapidly, securing two

points of distribution—one at Baku via the Trans-Caucasian railway, whence it reached Tiflis, Persia, on June 26th; the other at Astrakhan, at the mouth of the Volga, where it broke out June 30th, and was carried by steamboat traffic up that river as far as Tzaritzyn, Saratov, Samara, Kazan, into the Province of Kostroma, even spreading to Perm and Nijni-Novgorod, and to Ekaterinburg in the Oural. Moskva (Moscow) became infected August 5th, St. Petersburg twelve days later, when the German ports quickly caught the epidemic, which probably entered both by crossing the Prussian frontier (rail route) and by sea (via the Baltic).*

Since its inception in the Northwest Province of India, not only has it traveled the course just indicated, but it likewise covered or overflowed many thousand square miles of territory, disseminating itself in very much the same way as did the terrible epidemic of sixty years ago; and all the epidemics that have afflicted Europe, both before and since, appear to have closely adhered to the same line of progression, showing that epidemic history tends to repeat itself.†

This is a matter worthy of more than passing at-

* It is to be remarked also, that Hamburg has ever appeared a dangerous point of distribution for Western Europe.—S.

† It is notable, both the present epidemic and that of 1867, began at the Hurdwar Fair, which is said to attract as high as three millions of pilgrims each season.—S.

tention, since the present plague, unless abruptly stayed in its course, may possibly (though it is not probable) in its ravages duplicate those of 1829-34. It may be added, moreover, that the malady follows precisely the same laws in Europe and America as in the Orient; that it adheres closely to trade routes, and advances from the various points where the seed is sown (for reasons hereinafter explained) with intervening intervals of uncertain duration, in proportion to the facilities of communication; that it is in no inconsiderable degree dependent upon, and influenced by, seasonal and atmospheric conditions, dying out when these latter are modified or removed. Further, since it is only too plainly apparent that land quarantines and sanitary cordons, which European nations are ever ready to enforce against their neighbors, have not been successful in keeping out the epidemic, the utterance of Prof. S. L. Pisani, who is one of the most practical as well as sanguine sanitarians, possesses double weight: "Experience has taught that it is not in our power to prevent the importation of the germs of cholera, and that on good sanitation we should exclusively rely; let the soil be sterilized; let the seed fall on barren rock, and it will not germinate whether the season be favorable or not."

According to Dr. Wm. Farr,† and also Dr. J. C. Morton, cholera has probably always existed in spor-

† "Report on the Mortality of Cholera in 1848-49." London, 1852.

adic form in England and Europe, and this fact only serves to add fuel to the flame of an epidemic. They cite several "plagues" as presenting choleraic characteristics, among others that so carefully described by Sydenham in the seventeenth century, but which, however, appears to have more nearly approached a dysenteric flux.*

*See Appendix A.

CHAPTER II.

EPIDEMIOLOGY.

The medical world is to-day as ignorant of the primary derivation of cholera as ever, though numerous and varied hypotheses are not wanting. It is possible, perhaps even probable, that in its natural home, in the great river deltas of lower Bengal and of Siam and Burma, where it is endemic, it results from *miasm*; but whether or not this miasm is developed on diseased rice, as has been suggested, it must be admitted the disease, in its more malignant form at least, was originally exotic to Europe and America, and even now depends for its vigor, more or less, upon the constitutional dyscrasiæ of individuals, and for its persistence upon certain telluric, meteorologic, atmospheric, and concomitant unsanitary conditions.

It is especially noticeable, all others things being equal, that epidemic cholera flourishes best—that is, attacks more people, is more fatal, and more rapidly extends itself geographically—under two special conditions:

First.—During a high temperature of air and earth:

Second.—At periods when the variations of ground water and temperature are capricious, abrupt, without warning.

Thus, in October, and early November perhaps, in the northern temperate zone, when the majority of people are day by day vacillating between light clothing and heavy wraps—when colds, influenza, and malaria, are especially rampant, and other diseases prevalent that are the outcome of carelessness, deficient body-temperature, or overheating,—the ravages of cholera are apt to be greater than in December, when heavy clothing and heated houses are the rule. So too, the epidemic is more active in July and August, when the days are insufferably hot and the nights cool with heavy dews, than in either September or June. Again, the history of the disease in Asia, Europe, and America, evidences that in high and dry situations, in cold climates, wherever there is a moderately uniform or gradually progressing or regressing temperature during the twenty-four hours, its spread in the main is limited and slow; while in low-lying moist places, especially in hot climates that present extremes of humidity between meridian and the succeeding sunrise, its ravages are apt to be most severe and oftentimes quite uncontrollable. In both farther and hither India, it has been observed the pestilence suddenly springs into activity with the arrival of the southeast monsoon which, charged with humidity from the Pacific Ocean, frequently causes a fall in temperature of as much as fifteen to thirty degrees within almost as many minutes.

Further evidence of the part played by telluric,

meteorologic—and likewise astronomic—conditions, is had in the fact that when cholera is endemic to volcanic regions, it becomes more active and virulent in those seasons when there is an accession of volcanic action, or when such is pending; the disease was exceedingly active and pernicious throughout the Indian Archipelago and the Malay Peninsula after the Sumbwa eruption, and all enteric maladies of choleraic nature were then greatly increased and obtained new impetus. In Middle and Eastern Europe, when the epidemic of 1830–34 was at its height, it was observed, birds, quadrupeds, fish, and even insects, suffered, both prior and during the epidemic, from unknown maladies that caused their death in great numbers; the same was true in India; also of the United States, in varying degree according to topographical features and surroundings. Dr. O. D. Norton, of Cincinnati, recalls that in that city, in 1849, when cholera was especially virulent, birds died in their nests, and even the house flies and mosquitoes were exterminated. The same phenomena have accrued to certain portions of Europe the present year. Again, each successive cholera epidemic that swept the civilized world, has been preceded or accompanied by profound disturbances within the solar system, this year it being the occultation of Mars; also by evidences of famine: Finally, the great epidemics of influenza of this century, though their appearance may be coincidences merely, are

certainly suggestive in that in each instance, viz., in 1805-34; 1847-49; 1851-53; 1864-65; 1890-91, they preceded an epidemic of cholera. Sir Thomas Watson especially notes this fact; and John McLean for twenty-five years a Hudson Bay factor, observed the same in the peninsula of Labrador, where the natives (Nascopies and Innuits) were first decimated by influenza, then ravaged with an epidemic of choleraic character; further, it is somewhat striking that both maladies, to use the words of Watson, "issuing from their cradle in the East, traversed the northern countries of Europe till, arriving at its western boundary, they divided into great two branches; the one proceeding onward across the Atlantic, the other turning in a retrograde direction toward the south and east." Between the two epidemics moreover, there is marked similitude or analogy, the main differences being that, whereas one spared but very few and was seldom fatal, the other smote very few, but with so deadly a stroke that the death rate was exceptionally high. Both are in a sense general epidemics, affecting the whole system but especially manifest in the nervous portion; in both the most prominent symptoms are referable, in the majority of cases, to the mucous membranes—to those of the air passages in influenza, to those of the alimentary canal in cholera.

Surgeon-Majors T. R. Lewis and D. D. Cunningham, who investigated cholera in India for eleven years consecutively (1869 to 1880) under orders from

Her Majesty's Secretaries of State for War and India, remark,* that in manifesting a marked partiality for a soil of the character of the Brahmaputric and Gangetic alluvium, "cholera is by no means singular, for it is a well established fact that malarious fevers and kindred disorders flourish with most vigor about the deltas of large rivers all over the world," . . . but they would not, however, "be understood to imply that the causes productive of malarial fevers and cholera are identical, or that the localities providing the conditions necessary for the development of the one must necessarily provide those for the other also."

Speaking of the Andamans, where this malady has never flourished and malaria is always rampant, Dr. Lewis says: "Notwithstanding the Islands are within three days of India, and twenty-four to thirty-six hours of Burma, and that during the last twenty years steamers have constantly passed between the two countries and the Settlement, . . . it is only on rare occasions that cases of cholera have been registered as occurring."

Of these rare cases Dr. Rean, principal medical officer of the Settlement, says "the patients were generally admitted from some feverish locality, or had been employed on works of an unhealthy character."

The importance of well authenticated cases of this nature can scarcely be overrated in connection

*"Physiological and Pathological Researches." London, 1888.

with the ætiology of cholera; they strongly evidence the correctness of the views promulgated by both Chas. Macnamara* and Max von Pettenkofer,† and assimilate, so to speak, the two. Questions of possible infection or of water-contamination by specific imported material, can hardly be seriously entertained here. With the restrictions surrounding this isolated convict settlement there can be no casual importation of cases, as the recent history of every person is accurately known. Similar seizures, moreover, occur habitually in every city of India, as well as every summer and autumn in all the large cities of Europe, and also in America, but excite no special comment unless an epidemic supervenes, or is already rampant, when these otherwise ignored cases are seized upon, collated, and described as foci of the pestilence. It is not the custom *then* to regard such cases as due to a localized generation of the disease, and the fact the *comma bacillus* may be detected is held conclusive evidence, ignoring the well established fact that this microbe is by no means pathognomonic, but present even in conditions of health.‡

* "A Treatise on Asiatic Cholera," London. 1870.

† "Die Verbreitungsart der Cholera in Indien, nebst Atlas." Braunschwig, 1871.

‡ At the moment of this writing it is announced cholera has found foot-hold in the city of New York, the evidence resting solely upon the presence of the *comma bacillus*. That this is, presumptively, an error is shown by the press reports,

That in the present stage of knowledge it is impossible to explain all the phenomena of cholera distribution by telluric or meteorologic conditions, may be allowed, yet neither can such influences be denied. It must be remembered the same difficulties obtain in regard to malaria and kindred diseases, and that one has as substantial claim to this theory of diffusion as the other. And that cholera in its ætiological relations does present marked parallelism to other diseases that are dependent chiefly upon topographical surroundings for propagation, is proved by the fact even malaria sometimes breaks loose from its endemic haunts and shows itself in places where it before was totally unknown. Thus, says M. von Hertz* it “sweeps over considerable regions of country as an epidemic, and over vast sections of the globe as a pandemic. . . . It does not seem probable that currents of air are capable of carrying the poison generated to a distance of any considerable number of miles; I believe rather it is in a majority of cases generated upon the spot. It is a still more difficult matter to account for those isolated areas of malarial

that declare two patients were ill with the malady *eight days*. In exotic cholera, fatality supervenes, or convalescence begins, usually, ere the fifth day succeeding seizure has opened. Presumably the cases were all indigenous in character.—S.

*“Cyclopædia of Practical Medicine;” von Ziemssen. New York. 1874.

poison that are often confined to single streets, to one side of a street, or even single houses."*

It is a matter of common experience that removal from a locality in which cholera exists is a remedy against the spread of the disease, and the East Indian Government has for many years acted on this knowledge, with regard to its troops and convicts, with gratifying success. It is equally a matter of experience the disease is most virulent in those years when, owing to telluric and meteorologic conditions, the food crops are more or less a failure and famine threatens; and that, while it manifests itself with unusual severity

* That the parallel between malaria and cholera is much more close than generally imagined is, however, evidenced in that both depend for their phenomena (as is shown of cholera in Chap. IV, p. 56, and Chap. VIII, pp. 105, 110) upon disturbances of the vaso-motor system.

The contracted vessels of the skin and the rigors associated with the cold stage in malaria, are evidences of hypertrophy and hyperæsthesia of vaso-motor nerves; while increased temperature, flushed surface, full pulse, and the dilated blood vessels accompanying pyrexia, exhibit tissue paralysis, both nervous and muscular. The splenic and hepatic engorgements, and the diarrhœas and dysenteries that are so frequent sequels of malarial poisoning, are derived from dilated and paralyzed arteries, and consequently excessive flow of blood to undilated, enfeebled tissue. In cholera, too, we have paralyzed blood vessels; but there is also another dangerous factor, in a measure specific and dependent upon the former, in that the blood itself is constantly and rapidly being deprived of its serum.—S.

in certain localities, in others closely contiguous its ravages are comparatively mild or wholly absent.— How often has it been observed in the case of an outbreak that shifting a ship a few hundred yards from its anchorage, or crossing to the other side of a river, has sufficed to end an epidemic!

An instance in fact may be cited in the visitation of America in 1853-54, when Sarnia, Ontario, and St. Clair, Michigan, suffered severely, while Port Huron, just across the river from the former and twelve miles above the latter, had but three cases, and these, there is every reason to believe, obtained the infection in Canada.

At this period neither of these towns was provided with sewers or any form of drainage other than afforded by natural topography, and the fermenting and decaying "sawdust pavements" of the streets of Port Huron, it might be supposed, would naturally tend to foster the epidemic. But the real reason for the immunity was, doubtless, the dwellings for the most part were confined to a sandy or loamy porous soil overlying a substratum of blue clay, the latter with a dip of from twenty to thirty feet to the mile, sloping toward the St. Clair River. Sarnia and St. Clair both rest on an outcropping of clay that was baked and seamed by the hot sun of a summer supervening upon a wet spring.

Again, on November 9th, 1817, cholera attacked the camp of the East India Company troops stationed

on the borders of Scindia; this cantonment was on the *right* bank of the Sindh or Betwa river; but the ravages were stayed as by magic when the forces were moved over to the *left* bank, a distance of not more than three-eighths of a mile.

It is also interesting, in this connection, to know that in India, in 1819, the citadel of Jaragurth, situated in a slight depression 1,000 feet above the level of the plain, lost many of its inhabitants, while a city near the foot of the mountain, with good natural drainage, entirely escaped attack!

Another peculiarity more or less positive in its evidence is, that while certain districts are exempt during any one epidemic, or any series of epidemics, the same may on a subsequent occasion be attacked, though there is always a decided predilection for some localities at all times. It is not uncommon to find the epidemic passing over large tracts of country, with the wind perhaps in its very teeth, and it seldom spreads itself on any systematic or geographical plan, since it may appear simultaneously in regions a thousand miles apart.

Indeed, nothing can be more capricious than the variation in the intensity of the malady in different places and at different times, or at different times in the same places. An imported case may end in a local attack confined to a single room or single house; even a simultaneous importation of a number of cases at different points may exhaust itself in a number of

local (circumscribed) epidemics; while at other times a single case suffices simply to produce a general epidemic or even a raging pestilence. The history of different epidemics in large cities shows the greatest variety of effect, according as the cholera poison found the conditions for development more or less suitable.

And when the disease is fairly established as an epidemic, its spread in a severely infected place is by no means general, or in anyway uniform. A row of houses, a series of streets or blocks, or perhaps a ward or other section, becomes an epidemic centre. Then, again, there are individual room (or several room), epidemics, sometimes with a certain preference for damp cellar lodgings; or individual groups of houses are attacked in one street; often only one side of a thoroughfare is ravaged, or out of a series of blocks perhaps only one complete square and one or two streets will be visited, while all about in the vicinity there will only be, here and there an isolated case, or none at all. Here is illustrated the combined effect of importation and of local fixation of cholera germs in the ground or drinking water, in the moisture of the walls, in the damp, heavy, musty air of unventilated rooms, and in the emanations of sewers; while the dissemination is effected by adhesion of the germs to the washing, bedding, vessels, etc.

What the primary factor may be, then, is unknown, but the fact remains that the production and

course of the malady are so greatly under the control of sanitation that neglect of measures essential to the latter, on the part of individuals, and municipal, county, and state authorities, as well as the general government, is little (if any), less than criminal.

Even without knowledge of essential cause, we are perfectly familiar with results and effects, and these afford the text upon which to work. We do know, aside from telluric or meteorologic conditions,* that

*The arguments for and against the contagious nature of cholera are many and varied, and some of the positive are the result of misunderstanding and misapprehension. Thus certain German writers are frequently quoted as contagionists, when, in fact, more careful perusal of their writings show they are only infectionists; this error arises from the fact there is but one term (*Ansteckung*) in the German language to express both conditions.

Surgeon-Lieut. Colonel J. M. Cunningham, Health Commissioner of India, who for years has studied cholera where it is endemic, emphatically declares it is not contagious. Dr. Edward Goodeve, who likewise has had extended experience in various portions of the Orient, insists the malady "does not spread from the sick to the well by any rapidly acting emanation. Surgeon-General Chas. Hunter, in his report upon the epidemic in Egypt in 1884, is equally assured of its non-contagious nature. Many others have noted that patients ill with the disease may be attended, washed, lifted, etc., with very little risk, and that the discharges from stomach and bowels are the chief, if not the only, sources of danger. Hermann Lebert pointedly remarks (Ziemssen's "Cyclopædia, of the Practice of Medicine," vol. i):

the disease, though not in strict sense contagious, during epidemic times is, in considerable degree, at least, infectious, the poison apparently being constant in the dejections of cholera patients; that this poison may be disseminated at points remote from the ravages of the malady by being carried thither in the intestines of individuals, who perhaps present no evidences of cholera other than a slight intestinal flux, hence have no idea they are victims, or being made involuntary means of communication; a transient traveler may thus, through a single privy or water-closet, infect a whole community.

Niemeyer tells us that in 1848 a detachment

“Cholera can be spread only by contagion, that is by germs which are carried from a diseased to a healthy person; but these germs infect only comparatively rarely by intercourse or contact with cholera patients, since they possess relatively but little vitality in the air of the sick-room, and are present mostly in inconsiderable quantity. On the other hand, a certain number of the germs and a given vitality are necessary for the propagation of the disease, and these conditions are better met in fluids than in the air; hence contagion is more frequent when the germs are communicated through a fluid than when transmitted through the air.”

Thus it seems, while Lebert apparently pronounces for *contagion*, he really means what in English would be *infection*. Felix von Niemeyer (“Text Book of Practical Medicine,” vol. ii.) expresses himself in like manner, and subsequently adds he is a non-contagionist, later explaining his position, making him in fact an *infectionist*.—S.

of recruits from Stettin, where cholera was raging, came to Magdeburg, two of whom on the night of their arrival fell ill of the malady, and were immediately sent to the military hospital without coming in contact with the inhabitants. Nevertheless, a few days later cholera asserted itself first, in the house where they had sojourned a few brief hours, and later along the street on which the dwelling was situated—all from the use of a privy by one of the unfortunates. Again he remarks:* “A small epidemic in Greifswald gave me excellent opportunity for observing the spread of cholera, and in almost every case I could find that the patients had used the privy of affected houses, or that they had used a privy in common with persons from these houses who had diarrhœa.”

F. D. Alexandré tells of a soldier attacked with diarrhœa, who arrived at the village of Haime, from Paris where cholera was raging, April 4th, 1849, and remained three days at his father's house, when he went to hospital—there was no supposition of cholera in his case, so light was the attack: In the course of ten days seven persons in the household contracted the malady, four of whom died. Fred'k Wm. Goëring corroborates with an account of a vagabond, suffering in like manner, who was committed to the workhouse at Dieburg, with the result the epidemic

*“Text-Book of Practical Medicine,” vol. ii. New York, 1884.

swept through that institution, but nowhere else manifested itself in the town, save in a single instance; the exception was the woman who acted as laundress to the prison.

Also, Prof. von Pettenkofer relates the case of a man committed to the prison of Ebrach, from Munich, during the existence of cholera at the Bavarian capital, and who suffered from intestinal flux. His diarrhœa persisted, though its nature was not recognized, and he was sent to prison hospital. Immediately the epidemic developed within the institution, the first victim being a female prisoner who had washed the clothing soiled with the diarrhœal discharges of the Munich convict.

Of the pernicious effects of general or common latrines may be cited the fact that, during epidemics in America, the soldiers of the U. S. Army and of the Marine Corps suffered more severely than any other relative number of people; the closets or privies in connection with barracks are usually in a foul state, and in most instances are merely open trenches with but a rude shelter to protect from the weather.

Since it has been shown that the malady is only transferred to healthy persons—persons possessing no special or abnormal receptivity—through the dejections of those afflicted with the disease, the previously enigmatical and apparently contradictory observations concerning the spread of an epidemic are satisfactorily explained. Further, that it spreads more rapidly than

formerly is accounted for by the fact the facilities for travel and communication are greatly increased; and it is little wonder the generally traveled routes exhibit the greatest ravages, or that extension is against wind, or by long leaps with occasional retrocessions, while places intervening escape—traveling cholera-victims infect only those places where they leave their dejections. Again, if the cholera germs were contained only in the dejections of those who suffer from the severest form of the disease, as they cannot travel, long springs of cholera epidemics could only occur through spontaneous generation, or when persons infected with the poison traveled during the period of incubation, and the disease in them did not assert itself *en route*. But besides such, numerous examples prove that persons suffering from simple choleraic diarrhœa (as in the Magdeburg cases cited by Niemeyer) and who at no time are very ill, carry with them the fatal germs, and by infecting a single water-closet or out-house may start an epidemic *de novo*, as it were.

Lebert believes even a healthy person may carry the germ from locality to locality, and yet suffer no inconvenience—*i.e.*, he may escape the malady altogether. He calls attention to the fact one seizure also renders individuals in some degree personally immune, though their aptitude for carrying the infection is in no way lost providing they have been exposed to its influence. While he declares his doubt whether

the cause is an organic poison or living organism, he is inclined to accept the mycetic theory which, as he remarks, "explains without strained effort why it is that fluids, and especially stagnant fluids, containing more or less organic nutritious matter, are chief vehicles of the cholera germs, as they are of all proto-mycetic forms. It is on this account that the water of the soil, the drinking water, and every fluid, play so highly important a rôle in the diffusion of the disease; and yet neither the ground water or the drinking water theories can ever prevail in sole sovereignty as causes of the disease, since such are not necessary for the development of the germs, but only become so when they can furnish these germs with proper nutritious matter, when other favorable conditions of growth are presented, and when more especially the way of communication with the human organism is open. The germs of cholera may be spread without ground or drinking water just as easily as with them, through the air, by becoming attached to solid bodies, etc. . . . True, cholera finds in drinking water also a very frequent and most potent medium of dissemination, as it may be impregnated with the poison (from water of the soil by filtration from privies and sewers) which may then flourish in further development; still drinking water alone cannot be considered as the exclusive or necessary means of dissemination. Over-flowing or badly cemented drainage or sewer pipes, for instance, conveying infectious matter, may

carry their foul contents directly into the ground walls of cellars, and dwellings, and swiftly develop destruction among the inhabitants.”

It is probable, however, the dejections do not when first expelled contain the cholera germs in the stage of development necessary to infection, but that they become strictly aggressive only after having undergone fermentation, which result is furthered by admixture with decomposing animal substances—and this is why the midden privy is always a greater source of danger than the modern closet. This theory is supported by numerous facts.

Dr. O. D. Norton, a veteran practitioner of Cincinnati, who had extended experience with cholera in the epidemic of 1849, remarks, regarding experiments with *fresh* alvine excretions of “rice-water” character, that he and a confrère fed such by *buckets-full* to numerous chickens and pigs, but induced in them no evidences of the disease; on the contrary, these creatures seemed to “grow fatter” thereon.*

The observations of C. von Thiersch show that while recent dejections are not dangerous to animals, feeding the same with *old* excreta of the same sort invariably induces the malady.†

Further, experience shows that physicians passing

* Cincinnati Lancet-Clinic, vol. xxiv; Sept. 24th, 1892.

†“Meine Cholera-Infektionsversuche vom Jahre, 1854, und die des Herrn Dr. B. J. Stokvis vom Jahre, 1866.” Munich, 1867.

from bedside to bedside are comparatively immune.— Niemeyer says his experience in cholera epidemics, wherein he wrapped patients in blankets and often held them in his arms for some time, made him a “decided anti-contagionist;” that those who wash the body and bed linen after they have lain some time, are more apt to be infected than those who directly care for the patient, even to removing the dejecta.

Again, Lebert says: “I have noticed in all epidemics, and have seen it mentioned in the writings of many authors, that practicing physicians, even hospital physicians, are seldom attacked with cholera.”

In Cairo, Egypt, in 1831, of one hundred servants employed as rubbers or *masseurs* of cholera patients, not one was ever attacked; of eighty rubbers at the hospital at Mansurah, and sixty at Damietta, all escaped save one. None of the physicians or nurses of the cholera service at Constantinople in 1855–56, and Oran in 1861, ever suffered from any form of the disease.

Washerwomen, whenever they wash linen soiled with cholera dejections, without any precautions, are attacked in all places in no small numbers.* In Branson,

* The frequency with which washerwomen fall ill with the disease from contact with infected linen has often been mentioned, but there are also examples where cholera has been spread by rags and other objects. The same is true in still higher degree of unclean bedding. C. von Zehnder ascribes the origin of two cholera centres in the Zürich epi-

in the Canton of Valais (Switzerland), in 1867, one of the Sisters of Charity nursed, with the greatest self-sacrifice, all the cholera patients in very filthy chambers, and yet remained healthy, but at the close of the epidemic "her sympathy prompted her to assist in washing up the soiled linen, when she was attacked with the disease and died. It was from a washer-woman, who died after washing the clothes of a cholera fugitive, that the epidemic developed later in Zürich, 1867." (Lebert.)

Very numerous facts might be cited to demonstrate that cholera may be communicated, and carried from place to place, by clothing or other material soiled by cholera dejections; the observations of Etienne Moulin, Gaston Pellissier, Jas. Simpson, Jules Bucquoy, J. M. and D. D. Cunningham, Max von Pettenkofer, Antoine Fauvel, Augusto Guastella, and others, are most definite. Guastella remarks:† "There were persons living in places sheltered from the epidemic who, after washing linen soiled with the dejections of cholera, carried the disease afar." Fauvel adds other facts‡ showing that camping places where an

demic of 1867 to an accumulation of bedding, mattresses, pillows, etc., that had been used on the beds of cholera patients, and afterwards piled up, before being carried off for disinfection, in the neighborhood of the houses affected.

—S.

† "D'igiene e medicina navale ad uso della marina mercantile." Trieste, 1861.

‡ "Le Choléra; etiologie et prophylaxie." Paris, 1868.

epidemic has occurred, hospital wards, sick chambers, ships and cars carrying cholera patients, etc., may preserve for some time, under certain circumstances, the power of transmitting the disease; nevertheless, such examples are comparatively rare. To transmit cholera by clothing, he considers, demands certain conditions, viz.: "To transport it a short distance requires certain contact with objects in connection with the patients, especially those soiled by vomit and rectal discharges; to transport long distances, the objects previously exposed to contact must be confined to close quarters where the fresh air is not renewed, and where sunlight does not enter. There are few examples of objects freely ventilated carrying the disease for any long time, or long distance, while there are many cases to prove that the transmission may easily occur where soiled effects have been closely packed for several months."

As to the influence of dead bodies in disseminating infection directly—*i. e.*, by handling,—Lebert expresses himself as doubting it very much. "We occupied ourselves almost daily in Paris, in 1849—my friends and myself—with investigations into the pathological anatomy of cholera. In Zürich, in 1855, I made all the post mortem examinations, with my assistant, Dr. Wegelin, and neither of us, and no one of our dead-room attendants, were attacked with the disease. I consider it, therefore, merely an accident when a body-carrier falls sick. I believe, indeed,

that animal putrefaction rather diminishes the capacity for infection, and that the bacteria of decomposition destroy the germs of cholera.”

Hugo Wilhelm von Ziemssen* lays especial stress upon the fact “it is more dangerous for the persons in a house if the evacuations are emptied into a privy filled with excrement, into a cess-pool, or thrown on a dunghill, as in such places the germs seem to find circumstances most favorable to their development and increase.”

A. von Hirsch† insists marshy and malarial regions are especially favorable to the dissemination of cholera, in that they furnish nourishment for the germs, favoring their multiplication; also because the soil in such localities is eminently fitted to transmit, by soaking and slow filtering, cess-pool fluids and sewage waste, carrying into cellars and basements; that thus the privy of a neighbor may be more dangerous than one’s own, especially if in close proximity to the residence of the latter.

From observations made during the last three epidemics in France, Dr. Hippolyte Mireur concludes cholera is not transmitted directly from the ill to the well by contact or through the respiratory passages; that the products emanating from cholera patients—

* “Die Choleraepidemie, vom Jahre 1867.” Greifswald, 1870.

† “Rückblick auf die neuere Choleralitteratur.” Schmidt’s Jahrbücher. Bd. lxxxviii.

the dejections and vomited matters—alone contain the germs, which are not immediately transmitted by themselves, but when placed under favoring conditions give rise to an infectious principle; that clothing and merchandise,—such as skins, hides, rags, etc.—much more than individuals, are the agents for the transportation of this principle. *

If then there is any justice in the belief of Lebert, Hirsch, *et al.*, that the cholera germ lies within a spore, † it is more than probable the ripening of such in the fæces after evacuation is the real source of infection; or that the product of the ripened spore, on being returned to an economy, further develops producing perhaps certain alkaloids that, in turn, taken up by the absorbents, induce violent toxic symptoms—symptoms that, made manifest through the nervous system, constitute the phenomena of the disease. Dr. Thos. King Chambers remarks: ‡ “There is every reason to believe the chief exciting cause of the disease is a poison generated by decomposing

*“ Etude historique et pratique sur la prophylaxie et le traitement du choléra,” etc. Paris, 1884.

† The question of a spore is an interesting one from a certain standpoint since the majority of observers of and believers in, deny such to the comma bacillus; yet Huppe, of Prague, declares this germ does possess “a fructification propensity by virtue of an arthrosporulation,” which he personally observed.—S.

‡ “The Renewal of Life; Lectures Chiefly Clinical.” London, 1864.

organic matter and received into the body from without. To judge by its effects, it seems widely diffused through the air, especially in the neighborhood of its origin—in the air of privies, cess-pools, sewers, putrid marshes, and crowded human habitations. One is perhaps tempted to ask how it is, if the poison is spread so broadcast, that everybody does not get poisoned; but it must be remembered two things are necessary to poisoning, viz.: Not only a poison, but a person in condition to be poisoned; and in point of fact *the latter* is the more important element in the transaction.”

Thus the weight of evidence goes to show cholera epidemics, for existence and dissemination, demand three prime factors, viz.:

First. Conditions of soil, atmosphere, etc. (general surroundings), favorable to the nourishment of the germ or germs, which would otherwise speedily lose the power of infecting:

Second. Conditions in each human subject, individually, favoring receptivity:

Third. Direct infection *per se*:

And regarding the last, it is believed the principal, if not the only way of insuring infection, is through the medium of the intestinal canal and its absorbents:

That the germs are ordinarily carried but a short distance through or by the air:

That the great danger lies with the alvine evacu-

tions and vomited matters, but only after fermentation has been set up therein:

That contamination of the water supply and of food, by cholera discharges, is ever a grave factor:

And, finally, that linen, cotton, or woollen fabrics, soiled by cholera discharges, if excluded from air and sunlight, serve to keep alive the germs of the disease for an indefinite period.—The history of the barque *Swanton*, on which cholera did not appear until she had been at sea for twenty-seven days, when clothing was unpacked by the passengers, also of the ship *New York*, on which the disease did not manifest itself until she was sixteen days out from her port of departure, and then under the same circumstances as on board the *Swanton*, both evidence the truth of this statement.

CHAPTER III.

TRANSMISSION DANGERS.

It is to Prof. von Pettenkofer we are indebted for the discovery that porosity of the soil, by enabling the contents of privies and cess-pools containing the cholera germs to freely permeate and soak the ground for some distance around, and poison wells and sewers, favors the rapid extension of the disease, while the opposite quality to some extent inhibits dissemination; and the same author was the first to demonstrate that the "manifest fitness of any locality for the disease depends on excrement, containing the germs, permeating the soil and exposed to circumstances favorable to decomposition."

Next to soaking of the soil is the danger from gutters and drains, which may carry the infection from house to house; and it is well known that a soil-pipe, or untrapped rain-gutter, has carried the disease into an uninfected dwelling through a window of the latter being contiguous to, and at higher elevation than, the upper end of the latter.

There can be no doubt foul drinking water plays no inconsiderable rôle in dissemination. Mr. J. Snow* established the connection of the fearful local epi-

*"Cholera and the Water Supply in the South Districts of London in 1854." London, 1856.

demic in Broad street, St. James' Parish, London, in 1854 with an infected well; its ravages ceased when this supply of water was shut off from the public. J. Simon* declares in the portion of London supplied with river water drawn from the stream after it had received the contents of a large number of sewers, so that it had forty-six grains of solid constituents to the gallon, the number who succumbed to the malady was thirteen out of every thousand, while in other situations, under precisely parallel circumstances and surroundings, save the water supply contained but thirteen grains of solids to the gallon, the death rate was only 3.7 per 1,000. Edward Frankland,† speaking of the same city and relation of water supply to cholera, says:

“On the 18th of August, 1866, a family removed from London to Margate; on the 26th there was a storm with heavy fall of rain, and the water had an unusual odor and taste. On the 27th four persons were attacked with cholera, and on the following day still more, the most of whom died. The water in the well at the end of the garden furnished, in 100,000 parts, 93.4 of solid matters, of which 7.36 parts were of organic or volatile nature. The cess-pool adjoining the garden had clearly poured its contents into

*“Report on the Two Last Cholera Epidemics as Affected by the Consumption of Impure Water.” London, 1856.

†“The Water Supply of London and the Cholera.” Quarterly Journal of Science, 1867.

the well after the overflow caused by the rain, and this had caused the fatal contamination, for an analysis made Sept. 18th showed 82.75 solids (in 100,000), of which but 1.13 parts were of organic or volatile nature. It was proven that all who were attacked had drunk from the well. A similar occurrence was established by Dr. Lancaster, of Epping Forest."

The same author declares the inhabitants of London who used Thames water from Kew, above the city, showed a mortality from cholera of but 8 in 10,000; those who used the water from Hammer-smith, 17 in 10,000; from Battersea to Waterloo Bridge—that is water contaminated by the sewage of the city—163 in 10,000. In 1854 only the half of a district was supplied from Teddington Loch, and the mortality therein was 87 in 10,000, but in 1866, all the water in the Loch having been drawn off, the mortality was less than one-tenth of that in 1854. Again, in 1866 the cholera was very severe in the East End, which was supplied by the East London Water Company, from Oldford, the reservoir, on the river Lee, being little better than an open excrement and sewer receptacle, even filtration being neglected. The result was the mortality in this portion of the city was from 63 to 112 per 10,000, while the balance of the London, with a pure water supply, exhibited a death rate of only 2 to 12 per 10,000.

Manchester suffered terribly from cholera in 1832 and 1849, when the water supply was very impure,

but in 1854 and 1866, the water being derived from the interior of Derbyshire through an aqueduct, there were very few cases, and these only of a sporadic character.

Dr. W. Schiefferdecker, too, mentions a fact worthy of note concerning the six great cholera epidemics that ravaged Königsberg, Prussia, from 1831 to 1866, in which more than 2500 people succumbed out of nearly six thousand attacked.* The inhabitants of those portions of the city supplied with drinking water from the river Pregel, and from wells, were those that suffered most, while those supplied by a system of water-works from the so-called "upper tank," in which the water was exceedingly pure, suffered much less severely; the Pregel and wells were fed with ground water and sewage.

Dr. J. Grätzer also describes an instance occurring in Breslau during the epidemic of 1867,† in which the walls of a badly constructed privy attached to a newly built and well arranged house rendered the water in an adjacent well impure; besides, the privy vault was not regularly emptied, and its contents overflowed into an unwalled excavation in the neighborhood of a large accumulation of ground-water. The consequence of this contamination, which affected the drinking water was, that in the beginning of the

*"Die Choleraepidemie vom Jahre 1871 in Königsberg." Königsberg 1873.

† "Die Breslauer Cholera-Epidemic." Breslau 1873.

epidemic no less than twelve of the inhabitants of the house were attacked, *eleven* of whom died; also other persons in the vicinity, who obtained water from the same well, were seized. In this instance, it was proved the cholera poison first entered the privy, thence passed into the ground water, then into the drinking water, and so on into the digestive organs of the unfortunates.

Again, when cholera is once introduced it sometimes happens only those are attacked who are in the house where the first infection is received, or who visit the same closet; and in some instances the malady has been restricted solely to house epidemics—further illustration of the care that should be exercised in the way of sanitation.

Niemeyer believes the poison is rarely taken into the system in the drinking water, but in the main enters the economy during the act of respiration and, lodging in nose, mouth, or throat, is swallowed with the saliva. “Using infected privies is so dangerous, because they are the favorite lurking-places of cholera germs, and the gases arising always contain dust-like particles.” The poison passes from the closet to the dwelling, and A. Biermer insists the latter are “more liable than individuals to infect.”

So far I have said nothing, relatively, regarding the claims of two schools the members of one of which, like sheep, blindly follow their bell-wether over any obstruction regardless of cause or reason. First

of these stand the followers of Louis Pasteur—a visionary whose utterances have never been worthy of dependence, who is utterly devoid of physiological or other medical knowledge, and who, moreover, is not even a reputable chemist—the bacillary pathologists. Second, those walking in the footsteps of Francesco Selmi and Armand Gautier, the latter of whom stands first in the discovery and investigation of vital alkaloids, and has had courage to approach some problems of physiology and pathology most abstruse and complex in nature, opening up a wide territory that has hitherto escaped investigation. It might be added there is a third coterie, who, between the “devil and the deep sea,” have appeared anxious to reconcile the foregoing, and by blending the two evolve a new pathology, Emanuel Klein, perhaps, being the most able representative of the class.

Practical medicine has suffered much from the invasion of new theories as well as experimental methods; traditional pathology has given way to the experimental, and spontaneous maladies to those forms that may be artificially provoked in inferior creatures. Further, the upholders of bacteriological pathology have followed too closely in the steps of their masters, in that they sedulously ignore all negative evidence while magnifying the positive. For instance, the monumental report on cholera just issued from the Government press at Washington, was com-

piled by an ardent bacteriologist, who, while gathering everything favorable to his view, carefully excluded all evidence calculated to invalidate; he quotes the early reports of Surgeon-Major D. D. Cunningham, which are indefinite, and sedulously ignores the later investigations of the same author as well as those of Surgeon-General Wm. R. Cornish, Surgeon-Major Timothy R. Lewis, Professor Ray Linkster, Sir Wm. Aitken, Doctor A. N. Brown, *et al.* —an act which renders the “sin of omission” even greater than that of commission. *Ætiology*, theoretically, has been very much simplified by the discovery of microbes, but certainly scientific medicine has very little profited thereby, since attention has been diverted from clinical observation and research. The fallacious charms of the germ theory have caused medical men to forget their mission, have prevented the relief of the ill, and produced misconceptions of disease. Dr. B. W. Richardson* only recently protested against the idea it was necessary “to subject a patient to a kind of modified snake-bite in order to settle a question of diagnosis,” and adds: “Twenty years ago the profession was steering well and steadily towards great principles on the preventive, as well as curative side of medicine; then crept in the wild enthusiasm of bacteriological research — research good enough in its way as a piece of natural history,

*The Asclepiad, 1891.

and as disclosing some curious tribal phenomena developed under morbid states of the organic structures and the blood, but a positive insanity when accepted as the one absorbing pursuit, restoring humoral pathology, ignoring nerve function, leading to Babel with its utter confusion of tongues, and separating for a time the modern art of cure from the accumulated treasures of knowledge, wisdom, and light of over two thousand years."

It may here be remarked, likewise, that the majority of maladies to which popular pathology ascribes bacterial origin, are very severe in character, and most generally fatal. Therefore, in cases where dissolution is rapidly impending, it is not without the bounds of probability or possibility—indeed has been triumphantly demonstrated in many instances,—that partial death may affect the fluids and tissues to greater or less extent preceding the general death of the organism; that is to say the sum of the deaths of the constituent elements. Again, in certain cases, changes usually observed *post-mortem* may and do take place in the fluids and tissues, or a portion thereof, *ante-mortem*—sloughing, gangrene, phthisis, malignant diseases, etc., are evidences of this. Consequently, bacteria, which under natural surroundings and habits of life are ever the products of decomposition, may be found in the tissues, blood, and other fluids during life, and be the results merely of the advanced degree of the diseased condition—not the

cause of its development. And to force inductions as the result of artificial cultivation of microbes, and the artificial diseases induced thereby, instead of through natural development of the same and the pathological factors under which such development occurs, is not alone contrary to all scientific precedent, but must always remain a source of error. Just as tame fruits differ from the wild, and domestic creatures from those that are *feræ natura*, so must bacilli differ according to their mode of cultivation and development, and in their results; a parallel under such circumstances cannot but be fallacious, as every zoölogist or botanist well understands, and a more thorough knowledge of these branches of science, on the part of the medical profession, would lead to fewer errors in pathological investigation.

Again, Dr. Burdon-Sanderson's investigations* prove the development of infective inflammatory products as the result, purely, of chemical irritants, while Lewis and Cunningham alike observed bacteria in the blood of creatures dying from such experiments. These important facts have a most pertinent bearing on the ætiology of cholera, as will be observed in a subsequent chapter.

In the report of the *German Cholera Commission* dated Calcutta, February 2d, 1884, Koch announced the comma bacillus as the specific cause of

* The Lancet (London), vol. i, 1873.

cholera; and since his claimed discovery was based on microscopic slides from four specimens of cholera in natives, said slides being furnished by Surgeon-Major T. R. Lewis, the latter, in conjunction with Surgeon-Major D. D. Cunningham, deemed the matter worthy of further investigation. In their report it is remarked:*

“So far, therefore, the selection of the comma-shaped bacillus as the *materies morbi* of cholera appears to be entirely arbitrary. Dr. Koch and his colleagues have adduced no evidence to show that it is more pernicious than any other microbe; indeed, as a matter of fact, the sole argument of any weight which has been brought forward . . . is the circumstance it is more or less prevalent in every case of the disease, and that the *German Cholera Commission* had not succeeded in finding it in any other.”

With regard to the suggestion that the cholera process may in some way favor the growth of these bacilli, and that they are not necessarily a cause of the disease, Dr. Koch, in the report from Calcutta, declares such a view is “untenable,” inasmuch as it would have to be assumed “that the alimentary canal of a person stricken with cholera must have already contained these bacteria; and, seeing that they have been invariably found in the comparatively large number of cases of the disease in Egypt and India, it

*“Physiological and Pathological Researches.” London, 1888.

would be necessary to assume further, that every individual must harbor them in his system. This, however, cannot be the case, because, as already stated, the comma-like bacilli are never found except in cases of cholera.”

To this Drs. Lewis and D. D. Cunningham reply,* if Koch and his colleagues had submitted the secretions of the mouth and fauces—the very commencement of the alimentary canal—to careful microscopic examination of the same kind as that to which they have submitted the alvine discharges, “we feel persuaded that such a sentence would not have been written, seeing that comma-shaped bacilli, identical in size, in form, and in reaction to anilin dyes, with those found in cholera dejecta, are ordinarily present in the mouth of perfectly healthy persons.”

Koch subsequently admits he had examined the mouth of healthy persons but found no comma bacilli; on another occasion, as claimed by C. S. Dalley† (though I can nowhere find such utterance in Koch’s own publications), he declared his familiarity with the comma bacillus of the mouth, and that it differs from his cholera bacillus in “being longer, more slender, and not so blunt at the ends,” etc.; but T. R. Lewis, Douglas Cunningham, Ray Linkster, Arthur E. Brown, and Sir Wm. Aitken subsequently proved the identity

* *Ibid.*

† “Technology of Bacteriological Investigation.” Boston, 1885.

of the organisms by accurate tests and measurements applied respectively to colonies taken:

(a) From the mouth of healthy human beings ranging from four to fifty years of age:

(b) From the alvine discharges of cholera-affected persons:

(c) From the intestines of persons who had died of cholera:

(d) From cultivations of all three in agar-agar jelly, in weakly alkaline peptone gelatin, etc., and:

(e) By the reaction of all to staining fluids—fuchsin, gentian violet, methylen blue, etc.

Further, during a subsequent and independent investigation, D. D. Cunningham found ten different kinds of comma bacilli in the dejections of sixteen consecutive cholera patients, that of Koch "being very far from the most numerous of the lot." Lewis, too, found the Koch bacillus most abundantly in the drinking waters of India in the season when the people using such waters were absolutely free from cholera. And M. Neller* observed the bacillus of the present cholera epidemic in Europe to differ markedly from that claimed by Koch to have been discovered in India, in that it is thicker, shorter, and larger than the latter, causes turbidity of bouillon, and in peptonized gelatin grows more rapidly; it was found in twenty-nine cases of cholera, also in the

* Le Progrès Médicale. 1892.

sputum of a case of broncho-pneumonia; and in thirty additional cases of cholera, *no comma bacillus whatever* could be found.

The result of these investigations conclusively prove the so-called cholera-bacillus to be only an "old friend under a new name"—an everyday *spirillum*.

Again, the investigations of Pettenkofer,* Biermer, and others, are founded on clinical data too

* Prof. von Pettenkofer who, together with Robert Koch, has been actively engaged in studying and observing cholera in the present Hamburg epidemic, declares that the latter's theory of the origin of the malady "has not stood the test of experience." It has not been proved that the pestilence was brought to the city in the way indicated by Koch, or that the comma bacillus is the cause of the epidemic. Commenting editorially upon this, *The Lancet* (London) remarks: "One of the difficulties attending the acceptance of the cholera bacillus theory of the causation of cholera is, to account for the occurrence of isolated and sporadic cases of that disease, which in the aggregate amount to a large number—in India, for example. One man is attacked and dies of cholera in the barrack room occupied by a number of others, the air, food, water supply and all other conditions being the same for all alike. There is perhaps no other case of cholera in the station at the time, nor is there any history of any having occurred before; and this is not at all an uncommon but a frequent occurrence in India at certain seasons, and outside and beyond the so-called endemic area. The relation of these cases to larger outbreaks and epidemics has not hitherto been exactly defined, for when the epidemic occurs later on, it does not at all follow that it should be at the

absolute for disapproval, while Koch declares:† “In these experiments, as to the influence of development-inhibiting materials, the surprising fact was established that comma bacilli extraordinarily *easily die when they are dried;*” and again—“For the spread of the infective material the main condition is, that the dejections should remain in a moist state, for *as soon as they dry up they lose their activity.*”‡ And laboratory experiments and experiences have demonstrated these bacilli are among the most sensitive and non-resistant of organisms of their class, since a temperature of either 15° or 50° C. (59° or 122° Fahr.) is sure death thereto within a *very brief* period of time; moreover their resistance to chemical agents is almost *nil*, especially in relation to acids, hence their destruction would be assured in the stomach by means of the gastric juice if the latter is of normal reaction; only in an alkaline or neutral medium is it possible to secure development. Koch tacitly admits this, and Klein and Herman Bigg, and the whole host of followers are compelled to corroborate. Further, the

same place or time or soon after the occurrence to which we are referred; and parallel with this inquiry, how did the River Seine become infested with the comma bacillus, and how was the cause of the disease introduced into Hamburg, or, to take the latest example, into the well at Portel, the fishing village near Boulogne?—S.

† Report of July, 1884—“Cholera in Europe and India,” by E. O. Shakespeare, Washington, 1892.

‡ Italics mine.—S.

fact may be recalled that while cold in the main seems to inhibit certain epidemics of cholera—as those of 1849 and 1866—in certain other instances it appeared to have no effect; in 1830 and 1831, in the height of a severe Russian winter, its ravages were of a most virulent nature, in Moscow with a temperature of *minus* 4° Farh., and even in Orenburg with a temperature of *minus* 22° Farh.

If, then, the comma bacillus is the cause, infection should be much more active at the bedside than in the closet—among physicians and nurses than laundresses, scavengers, and those who handle and cleanse the dried and soiled bed-clothing and body-linen,—whereas the precise contrary has been repeatedly proven by the best of all tests, practical experience. (Thiersch, Niemeyer, Tanner, Flint, Watson, Lebert, *et al.*) Again, it is a well known fact cholera dejections may be boiled to the utter destruction of the last bacillus, yet be not deprived of their virulence; (Virchow, Aitken, Lewis.)

Arnaldo Cantani and Klebs repeatedly obtained cholera poisoning from sterilized cholera fluids, and Lewis and D. D. Cunningham as far back as 1874 made a like observation. Finally, Jno. Simon, A. Delpech, Wm. Sedgwick, Dutrieux Bey, Alex. Harkin, J. M. Cunningham, Lionel Beal, Thos. J. Mays, B. W. Richardson, John Chaine, Mariano Semmola—the latter recognized as one of the most profound pathologists of our century—and others, attach no

importance to microbes, which they believe at most to be only modified anatomical elements, and sequels of the pathology instead of a cause thereof; that they are accidental accompaniments of disease, of which nothing can be predicated of the action of any particular form; they are scavengers only.

Thus is confirmed the trite utterance of Trichum, *à propos* of Koch's bacillus, when he adjured his listeners not to believe the purported discovery settled the question of cholera any more than knowledge of the tubercular bacillus would eradicate pulmonary phthisis. Semmola adds:* "This doctrine at best is based only on a hypothetical basis;" and, "I cannot comprehend how true clinicians can accept as of practical value results that are established solely in the laboratory."

Again, the Koch theory of infection embodies the belief that when a patient fails to succumb under an attack of cholera, it is owing to his organism having not only withstood the onslaught of the bacilli or the virulence of their morbid products, but that the invading hordes must have perished within the body. But that these hordes are a sequel rather than a cause, has just been shown by Dr. Cornet, of Berlin and Reichenhall, who discovered that persons apparently convalescent from the malady may carry about with them in their intestines, active living Koch bacilli.†

* Berliner Klinische Wochenschrift. 1891.

† "The case in which this important discovery was

Regarding the teachings of the Gautier school, it may be said they have little in common with pan-germic and bacterial theorizings, but rest upon data positive, precise, easy of verification; and if the indications submitted meet with the attention they seem to deserve, a sweeping reformation will result.

Both Gautier and M. Peter, following the lead of the great Selmi, believe there are elements resident within the economy that may induce certain specific diseases; that these elements remain inoffensive whilst elimination and oxydation of detritus is normally operative, but give rise to disease if from some cause or other this elimination and oxydation are interfered with whereby the detritus accumulates and exerts a toxic influence upon the nerve centres; in fact, that not only after death, but even during life, the animal organism—in accordance with physiological and chemical processes readily determined—has the power of elaborating a numerous class of alkaloids essen-

noticed was that of a man whose mother, wife, and son died of cholera. He himself had a slight attack, and was put under the care of Dr. Carl Lauenstein, of the Seemann Hospital. He was nine days in the hospital, . . . recovered perfectly, and was on full diet. Last Friday he was up and anxious to go home, but was induced to stay. On Saturday he was still better, and no motion at all, and it was with difficulty he was induced to remain. On Sunday Dr. Cornet discovered that in the stools passed there were large quantities of comma bacilli."—*British Medical Journal*, Oct. 8th, 1892.

tially toxic in properties, those evolved from dead tissue being termed *ptomaines*, those from living tissue *leucomaines*.* But this is not all. Gautier has also ascertained that in the living animal economy there are elaborated azotized uncrystallizable salts, substances the precise character of which is still undetermined, and which are the extractive matters (toxalbumins?); and while the ptomaines and leucomaines are both highly poisonous, the extractives are far more toxic than either! This discovery of ptomaines, leucomaines, etc., though perhaps of comparatively little value from a therapeutic standpoint, is nevertheless of the greatest importance to pathology.

The importance of such authoritative teachings has not escaped the germ theorists, who are forced to admit the symptoms that supervene upon a cholera attack obviously indicate a form of poison. Even Koch, along with others, has been compelled to hesi-

* Ptomaine, from *Πτώμα* a "carcase" or dead body, and *ίνο*, "material—or *in* from Latin, *inus* "belonging to." Leucomaine from *Λεύχωμα*, anything whitened as albumen or white of egg.

This nomenclature is far from satisfactory. The selection of the term *ptomaine*, indicative merely of the conditions under which animal alkaloids were first discovered, as a root whence to derive a name for these bodies, is too restrictive, since it is only appropriate for alkaloids of post-mortem origin. A title is still needed for alkaloids formed by morbid processes during life—the *ptomaines of disease*.—S.

tate, and to inquire if some ptomaine is not a specific cause, even while claiming a microbic origin for the disease; and Klein strongly supports this view. Dr. A. M. Brown,* who is vouched for by Gautier as an English exponent of his ideas, remarks:

“Cholera, . . . monopolizing as it does so much scientific interest, supplies the finest field for airing the respective claims of the two pathological theories, . . . the one bacillar, organic, and strictly biological; the other toxic, inorganic, and strictly bio-chemical. The first, so high in favor, and with the entire field to itself, has failed egregiously in its assumed solution. The various expeditions undertaken—Indian, African, and European—in bacterio-bacillar interests, have proved as practically hopeless and unprofitable as the discovery of a north-east passage to Cathay. By such missions Koch has only added to his hypothetical perplexities, while Klein and Bouchard, with modified appreciation, preserve their germ proclivities, and hope by vested but truly humoralistic concessions to solve the cholera problem.”

Koch thought he had found the pathogermic entity, but, confronted and constrained by cold *facts*, has later felt himself obliged to modify his positive utterances, and consequently now admits the comma bacillus does not directly engender cholera, and that it

* “Animal Alkaloids—The Ptomaines and Leucomaines.” London, 1889.

can only do so indirectly by the intervention of a ptomaine which he supposes it secretes. Thus he seeks to ally himself with the third class, who endeavor to reconcile theory with fact by preaching the "Good Lord, good Devil" doctrine! And this must imply two suppositions—first, a specific bacillus which Koch *has not* discovered; second, the secretion of a ptomaine by that bacillus which the Berlin savant and his following are equally as far from discovering.—Says Sir Wm. Aitken,* *à propos* of the foregoing:

"A. G. Pouchet obtained an oily base belonging to the pyridin series from cholera stools, and Brieger got from pure cultivations of the comma bacillus in beef broth, in addition to the common ptomaine of putrefaction, two poisons which he regarded as specific products of this bacillus." But none of the poisons which have been thus isolated produce exactly the symptoms of cholera: In Cantani's experiments, tremor, prostration, spasms, and repeated vomiting were observed; Klebs noticed muscular contractions and alterations of the kidney; the poison obtained by Pouchet irritated the stomach and slowed the heart; one of L. Brieger's produced muscular tremor and cramps, while the other induced lethargy and feebleness of circulation with occasional bloody diarrhœa. These facts, remarks Aitken, evidence "the symptoms of cholera are not caused by a poison formed by the

*"Animal Alkaloids." London, 1889.

action of the comma bacillus, and it is evident that much more extended investigation is required before the pathology of the disease is accurately understood." C. H. Fagge* suggests that in all such investigations one must bear in mind the possibility of the poison being formed, *not* in the intestines merely, but in the blood, nerves, and general tissues.

—Thus Koch assumes, Gautier demonstrates, and Klein and Chas. Bouchard† vacillate while sedulously essaying to promote fusion.

* "Principles and Practice of Medicine." London.

† Since the above was written, attention has been called to an editorial in the Boston Medical and Surgical Journal for July 28th, 1892, from the pen of my friend Dr. E. P. Hurd, of Newburyport, Mass., as follows:

"The toxic theory of Bouchard, as set forth in his book 'On the Auto-Intoxications,' fairly well explains the symptomatology of cholera. He demonstrated, as early as 1884, by experiments made with toxic substances found in the stools and urine of cholera patients, that the pathogeny of cholera may be referred to multiple intoxications.

"Professor Bouchard has some doubts as to the fact of the comma bacillus being the pathogenic agent of cholera. 'The only serious argument,' he says, 'in favor of Koch's claim, is the presence in the intestines of cholera patients of special micro-organisms, which are not supposed to be found in the intestines of healthy persons or of persons affected with other diseases. These micro-organisms exist often in considerable abundance, from the very first, and often to the exclusion of every other microbe in the digestive tube. Apart from this empirical ascertainment, which warrants only a presumption, all the other arguments which have been alleged are illusory.'

"The toxic alkaloids which Bouchard has extracted from the intestines and urine of cholera patients, greatly ex-

ceed those ordinarily contained in fæcal matters. One of these, which forms acicular crystals, seems to have a special virulence, and to be identical with the 'cholera poison' which Koch and Brieger have isolated from the intestinal contents of cholera patients, and which they believe to be generated by the comma bacillus. There is, however, no agreement as to what really are the soluble toxic substances secreted by the microbe of cholera. Bouchard affirms that the real virus is eliminated in the urine in appreciable quantities. In injecting into the veins of animals cholera urine, he has caused pronounced cyanosis, collapse, albuminuria, anuria, cramps, and pale, yellowish or bloody diarrhœic evacuations, like those which characterize true cholera. In injecting the alcoholic extract of the urine of cholera patients, he has determined somnolence, albuminuria, diarrhœa, and death in two days.

“‘There is,’ says Bouchard, ‘in cholera-urine a poison which I call the true cholera poison. I cannot chemically define it; I only know by its physiological properties. I know not if it is fabricated by the sick organism or by microbes.’

“Bouchard’s view then is, that besides the primary infection there exists in the pathogeny of cholera a secondary intoxication, consequent on the infection. He thinks that the symptoms considered as characteristic of cholera are the result of this intoxication. To this we may attribute the cyanosis, the chilliness, the respiratory troubles, the hic-cough, the special diarrhœa, the intestinal desquamation, the cramps, the de-hydration of the blood and tissues, the albuminuria, the anuria. But very soon ‘there supervenes a new source of systemic intoxication superadded to the first, and this clinically expresses itself by intellectual torpor — by somnolence, apathy, and coma. The respiratory rhythm changes, sometimes rising, sometimes falling; it is the rhythm of uræmia. The pupils are contracted, and become punctiform.’

“This is evidently a different symptom-aggregate from that of the initial period, and is due to another kind of poisoning; in other words, we have the clinical tableau of uræmia from excess of disassimilation and blocking of the kidneys.

“‘In short, cholera furnishes us an example of a double auto-intoxication; one by an abnormal product,—this the

choleric intoxication properly so called; the other by normal products,—constituting a variety of uræmic poisoning.’”

From the foregoing it would seem Bouchard is gradually “undergoing a change of heart.”—S.

CHAPTER IV.

PATHOLOGICAL DISCUSSION.

Setting theories aside, I may now deal with real facts. With all the wrangling of pan-germists, biochemical physiologists, and fusionists, one thing is most evident, viz.: The neurotic character of cholera!

As far back as the time of Wm. Cullen, who was upheld by Sir Thomas Watson, the disease found place in nosological nomenclature under the "*order* Neuroses, *class* Spasms." Jules Marey always considered the nervous system as primarily affected by cholera poison, and as determining the principal phenomena of the attack, even the gastro-intestinal symptoms. The poison, he declares, first excites the sympathetic system, whence ensues the contraction of the muscles under the dependence of that system. The spasm of the arteries of the greater and lesser circulation, as well as of the bronchial radicles, explains the phenomena of the cold period. In the period of reaction, the arterioles and capillaries relax, and there is stasis of the circulation and excessive watery exudation. And everything manifestly points to profound toxication of nerve centres inducing changes that are chiefly revealed through the great sympathetic, particularly in its abdominal and thoracic area.

Sir Henry MacCormac* and Chas. Lever,† both of whom had extended experience in Ireland in the epidemic of 1834, alike regarded the malady as provoked by a lesion of the sympathetic—a view supported by Wm. Sedgwick, Jas. Johnson, Claude Bernard, D'Arsonval, the elder Chermak, Fillipo Picani, D. Cannataci, Foster, Guérin, and Alex. Harkin.

But it is evident we must look even further. Manifestly the gastric and enteric pathology is not primary, but secondary, since in “dry cholera” (*cholera sicca; cholera asphyxia; cholera siderans*) death supervenes ere there is any evidence of intestinal or stomachal disorder or distress, and through failure of the respiratory and cardiac centres. Indeed, the latter factors are so prominent in every attack that Drs. E. A. Parkes, Jonathan Hutchinson, Geo. Johnson, J. Snow, W. Grissinger, and Surgeon-Major J. C. Hall, were inclined to believe the real morbid factor exists in the blood whereby is induced spasm of arterioles inhibiting pulmonary circulation and preventing oxydation, laying especial stress on the fact that there is likewise evidence of spasmodic contraction of the circular organic muscular fibres of the bronchi. Says Dr. Parkes:‡ “That there is some

*“Observations on Spasmodic Cholera; Its Origin, Nature and Treatment,” etc. London, 1834,

†“Cholera in the South and West of Ireland.” Dublin, 1834.

‡“Researches into the Pathology and Treatment of Asiatic or Algid Cholera.” London, 1847.

impediment or arrest of the circulation in the capillary system generally, and in the pulmonary capillaries in particular, appears almost certain; and it is by no means improbable, from the whole bearing of the facts, that this is due to chemical change in the fibrin and in the mode of its combination consequent on the direct action of the active cause."

Evidently the latter "builded better than he knew," and had a partial insight into the truth, as is seen in the terminal portion of the above sentence.

Dr. George Johnson, as cited by Thos. Hawkes Tanner, remarks: "During the state of collapse the passage of the blood through the lungs from the right to the left side of the heart is in greater or less degree impeded."* But he differs from Dr. Parkes as to the cause of this impeded circulation, his hypothesis being that the poisoned blood causes contraction of the muscular walls (instead of spasm) of the minute pulmonary arteries, the effect of which is necessarily to diminish or arrest the flow of blood through the pulmonary capillaries. Alburtus Eulenberg, Chas. Francois, J. M. French, and others, attribute cholera-algidity to cardiac adynamia provoked by nervous irritation proceeding from the intestine, a theory that derives support from the experiments of Tarchonoff and Franck who showed that irritation of the digestive tube and

* "Notes on Cholera, its Nature and Treatment," 1866
—"The Practice of Medicine." London, 1874.

mesenteric nerves may determine more or less prolonged arrest of the heart.

Though these views in a measure clash, they may all be considered as containing great germs of fact, being based upon observation in different individuals under variable conditions; moreover they are, in a measure, reconcilable when later pathological knowledge is brought to bear thereupon, and due consideration is given to the influence of the nervous system.

Though the theory of primary intestinal lesion has many advocates, who lay especial stress on two manifest symptoms, viz., dehydration of blood and tissues, and blood poisoning—that under the influence of the profuse watery discharges, provoked by such intestinal lesion, the blood and tissues became unfit for nutrition and functional work,—it must be remembered that in many cases no changes whatever are to be observed after death, either in the stomach, intestines, or elsewhere, save perhaps congestion of the pulmonary and cutaneous systems; but the left heart (as shown by Simon and others) is generally empty, while the right is distended and filled with blood. Sicluna and J. M. Bruce,* performing autopsies on the victims of the epidemic that ravaged Malta in 1887, always observed the cavities of the left heart empty and of the right filled with blood!

*“Treatment of Cholera.” Dublin Jour. Med. Science. March, 1890.

George Budd* reports concentric hypertrophy in cases of sudden death from cholera: and Jas. Jackson† especially noticed that at post-mortems of victims the hearts usually exhibited hypertrophy of the left ventricle.

Again, the elder Flint‡ lays stress upon the fact “epidemic cholera has no constant, appreciable, anatomical characters—none which appear to be commensurate with the gravity of the malady; the morbid appearances after death do not afford an adequate explanation of the symptomatic phenomena, nor do they elucidate the pathology of the disease.”

Tanner declares§ “post-mortem examinations have thrown little light . . .”; that “we naturally look first to the gastro-intestinal mucous membrane, but beyond distension of the follicles with serum, an œdematous condition of the mucous lining, patches of venous congestion, and here and there rupture of the vessels producing ecchymoses, we find nothing. The blood is altered more or less, is usually of tarry appearance and consistence, the proportion of water being much diminished, the fibrin being either reduced in quantity or affected in character, and the corpuscles increased, while the serum is rich in albumen, contains a slight excess of urea, and its salts,

* Medical Chronicle, vol. xxi.

† “Report on Cholera in France.” London, 1872.

‡ “Practice of Medicine.” Philadelphia, 1873.

§ “The Practice of Medicine.” London, 1874.

collectively, perhaps, diminished . . . the heart is often flaccid, its right side dilated, the left side contracted.”

Niemeyer, barring those cases where death occurred during the stage of reaction, emphasizes the fact* “the characteristic changes consist chiefly in extensive catarrh of the intestines accompanied by detachment of the epithelium and copious transudation, and in decided thickening of the blood and excessive venous hyperæmia of the kidney” (the latter mentioned also by Tanner as an occasional but by no means constant feature).

Lebert remarks:† “The anatomical lesions of cholera are of peculiar character, but clearly more the *consequences* than the cause of the disease, hence possess no pathognomonic character whatever.” And again: “The anatomical changes, the hyperæmia of the mucous membrane, the distension of the mesenteric veins with thick blood, the ecchymoses and hæmorrhagic suffusions of the mucous membrane, the swelling and great softening of the lymphatic apparatus of the small intestines, are, I am convinced, *not* the cause of ‘rice-water’ stools.”

Rudolf von Jaksch, Jas. Cagney, Hoppe-Seyler, C. Schmid, C. Zehnder, Jas. Sterling, T. R. Lewis, F.

* Text Book of Practical Medicine,” vol. ii. New York, 1882.

† “Ziemssen’s Cyclopædia of the Practice of Medicine,” vol. i. New York, 1874.

Delafield, *et al.*, corroborate these authors, admitting an inadequate pathology as evinced by the disease in any stage; and when is further recalled the fact there is no malady in the whole nosology that is more efficiently assisted in gaining a foothold in the economy, or the fatal tendency of which is more vigorously promoted and hastened by mental causes, we have most convincing evidence the ultimate source or cause of cholera lies deeper than in the organs that permit of general review and inspection.

Chas. Bouchard insists "from the study of the various attempts of pathologists to explain the symptomatology of cholera, it results that we must admit multiple causes. If the most powerful come under the head of intoxications, we must still make due account of the dehydration of the blood and tissues, and of the *reflexes* which take their start in the digestive tube and *affect the vaso-motors*. In favor of this latter influence, may we not refer to the alidity and collapses which sometimes follow the gastric crises of tabes, and which bear so striking a resemblance to cholera?"

CHAPTER V.

CHOLERA CHARACTERISTICS.

The characteristics of cholera, those most manifest in its so-called epidemic or malignant form, and upon which differential diagnosis chiefly rests, are:

First Stage.—A feeling of stupidity, general weakness, chilliness—more rarely a regular chill,—followed by uncontrollable watery diarrhœa devoid of color or nearly so, lacking also in odor. The first dejections are apt to be dark and pappy, but once the contents proper of the intestines are cleared out, they become of whey-like character, sometimes of pale reddish hue owing to admixture with blood, with not the slightest traces of bile pigment, and on standing usually deposit a fine granular, whitish-gray substance which contains triple phosphates, bacteria, fine shreds of algæ and blood-corpuscles, sometimes also phosphate and crystalloid salts of lime; this fluid is alkaline, being disproportionately rich in sodium chloride, and may contain some albumen, though not in great quantity. The investigations of Surgeon-Major Lewis evidence the flakes and corpuscles of “rice-water” stools do *not* consist of epithelium, nor of its *debris*, but that their formation “appears to depend upon the effusion of blood plasma;” that “the bodies found by Surgeon E. A. Parkes, moreover, correspond very closely in their microscopic and chemical characters, as well as in manifestations of vitality, to the corpus-

cles which are known to form in such fluid and are generally to greater or less degree associated with blood-cells, even when the presence of such is not suspected, especially when the disease tends toward fatal termination, when the latter have been frequently seen to replace the former altogether.”* This diarrhœa, which varies in frequency in different epidemics, may be regarded as the warning of an attack; and where it is absent, patients before the outbreak usually feel depressed, tired, and uncomfortable. These first manifestations, which are however some times totally absent, may be regarded as *stadium prodromorum*.

The duration of prodromic diarrhœa in cases of absolute cholera, as ascertained by Lebert, does not as a rule exceed three days, “but may continue five or even eight days.” He gives a table of thirty-five cases, closely observed during the Zürich epidemic of 1855, which is here reproduced:

DURATION OF PRODROMIC DIARRHŒA.	DIED.	RECOVERED.	TOTAL.
One day.....	1	7	8
One to two days.....	3	6	9
Three days.....	4	5	9
Five days.....	1	1	2
Six days.....	1	1	2
Eight days.....	1	3	4
Three <i>weeks</i>	1	..	1
	12	23	35

* “Pathological and Pathological Researches.” London, 1888.

Second Stage.—This stage, with which cholera not infrequently precipitately commences, constitutes the attack proper, and has been defined as algid or asphyctic—terms not altogether pertinent. There is a feeling of stupidity, general weakness, chilliness—occasionally a marked rigor,—followed by increased intestinal flux, the passages being expelled with great and sudden force, without warning; there appears to be complete loss of power over the sphincters. In addition to the “rice-water” evacuations, there is usually vomiting—which in many cases is a prominent and persistent symptom,—the expelled matters, like those from the intestine, being devoid of color and odor; this emesis may appear as a much more terrible symptom than the diarrhœa. A notable fact is, absence of pain, whereby the sufferer is enabled to endure the attack with comparative indifference up to the actual occurrence of cramps.

The cramps—in the lower extremities and abdomen, perhaps across loins, rendering the muscles as “hard as wood” or “drawing into knots,” as it were, the frequency of which varies in different epidemics, — constitute one of the most remarkable symptoms of the malady. Lebert usually observed in the second half of the attack proper, rarely earlier, and that they always assumed a tonic character in adults, while in children the tetanic form commonly obtained; occasionally, though rarely, the muscles of the face are involved. Each attack lasts but a few minutes, but

the frequent recurrence and excruciating character mark as the most distressing manifestation of the disease. Sometimes cramps persist to the very end in rapidly fatal cases, but usually they cease with the progress of the asphyxia, and in more protracted cases, in the cold period just preceding reaction. In a few instances both cramps and vomiting are observed together at the very outset of the malady, in conjunction with dizziness, headache, very great disquiet and anxiety—though, as a matter of fact, most patients exhibit a certain indifference. At the height of a very intense epidemic are sometimes seen patients who rapidly collapse with symptoms of great distress, becoming cold, cyanotic, dying after one, two three, five, six or more hours; but in such there is usually an abundance of colorless transudation into the intestine.

In the most intense or malignant development of the malady, persons may die pulseless, cold, cyanosed, etc., with no evidences of vomiting or diarrhoea, and with positively no characteristic changes to be found in the intestines or elsewhere, and no tangible cause for fatality; but these cases are so rare of late years that a large number of medical men are inclined to deny the existence of “dry cholera,” which was generally considered as proved in the earlier epidemics.

Thirst is invariably a most assertive symptom, and usually very urgent, though its degree is apt

to be in inverse proportion to the severity of the seizure; patients clutch at the attendants' hands as the glass or cup is held to them, in terror lest it should be taken away too soon.*

The circulation is greatly diminished, the pulse frequent and proportionately weakened, ranging from 120 to 140 per minute, though under some circumstances it becomes more and more feeble without acceleration, and in more pronounced or asphyctic conditions may fall below normal. When the state of collapse is fully developed the pulse is extinct at the wrist; next the pulsation in the carotids disappears; finally there is feebleness or absence of the apex beat of the heart, and of all cardiac sounds, evidencing greatly diminished power of the central organ of circulation. There is also general venous stasis giving rise to remarkable lividity or blueness at the roots of

* Says a correspondent of the British Medical Journal, a volunteer nurse of the Eppendorf hospital, writing from Hamburg recently:

“There are two pretty yellow-haired sisters who lie in a cot, with whom the characteristic ravenous thirst is the most pronounced symptom. They have no vomiting and but little diarrhœa. They sleep about twenty-three hours out of the twenty-four, and in their lethargy thirst seems to be the only consciousness. One lies grasping a cup with both her small hands, and if an attempt be made to take it from her, she automatically and drowsily opens her mouth for a draught, not knowing when the milk is given her, but swallowing mechanically.”

the nails, in the lips, face, and on the surface of the body generally; icy coldness of the skin everywhere— of the nose, tongue, and even the breath; frequently noises in the ears or head, dizziness, dimness of vision, deafness. The skin becomes shrivelled, and if picked up in a fold remains puckered for a time, retracting but slowly. Finally the entire surface of the body is bedewed with death-like dampness.

The number of respirations is usually increased, often to twenty-four, thirty, or even forty per minute, at the same time short, confined, and imperfect, frequently of sighing or irregular rhythm; the expired atmosphere, when collapse is complete, besides being of low temperature, contains more oxygen and less carbonic acid than in health, evidencing notable deficiency in the changes incident to the function. There is also marked alteration of the voice (*vox cholericæ*), which becomes whispering and unnatural owing to diminished volume of respiratory gases; oppression and pain at the præcordia are manifest, often of such excruciating nature as not to be accounted for solely by the dyspnœa present.—In many instances this dyspnœa is more particularly marked during the period of violent discharges from stomach and intestines and at the beginning of the absolute algid stage, only to disappear again with the conclusion of this period; pressure over the stomach usually aggravates. Cough is scarcely ever observed; and stertor is but exceptionally noticed and only in fatal

cases. In many instances complete aphonia supervenes, the motion of the lips being seen during efforts at articulation alone; and though this condition may at times yield for a brief moment, it is usually only when the intensity of the spasmodic muscular contractions causes the patient to cry out.

Nothing is more constant in this stage than the participation of the kidneys, and the manifold effects resulting therefrom. Albuminuria may sometimes supervene, the cloudiness varying from light opalescence to abundant deposit on ebullition, followed by partial or complete suppression of the renal secretion. Usually the microscope reveals a large number of wavy casts, most manifest when the urine has not been clouded by heating; also uric acid salts, as well as some blood corpuscles; and test with muriatic acid is apt to exhibit a large amount of indigo pigment (indican) which is certainly suggestive of profound disturbance of nerve centres, and also of the close relationship of the disease to malarial disorders. Lebert shows, in his report on the Zürich epidemic of 1855, that discoloration and commencing fatty degeneration may be recognized in the cortical substance of the kidneys of individuals who have died early in the asphyctic stage, and that this degenerative parenchymatous nephritis, which is more distinctly anatomical the longer it lasts, is not sufficient to explain the anuria that supervenes; further, it must be noted that the nephritis of cholera depends for its origin upon

disturbance in the nerve-centres, since it never by any accident becomes chronic, but disappears with the final vestiges of the disease. Lebert adds: "In all the four years after the epidemic in Zürich, I never was called upon to treat one of my patients for chronic nephritis, and among a great number of nephritic patients in Basel, I never found one in whom the nephritis could in any way be referred to a past attack of cholera."

It should be remarked that all the foregoing symptoms are apt to follow each other much more rapidly in children, especially under three years of age, than in adults, and death usually terminates with convulsions; all, however, may exhibit different combinations in different degrees of intensity, and so establish from the start the distinction between lighter and graver forms as well as between the numerous transitional grades. As a rule patients really suffer but a very few hours, and then, as already noted, only in consequence of cramps, since the intestinal and stomachal discharges are painless, between which profound rest occurs that closely borders on apathy. The expression of the face, in the beginning may manifest exhaustion and discomfort, but such is speedily followed by apparent indifference; later the sunken eyes, which are peculiar to grave cases, become remarkably dull and dry, and are only imperfectly covered by the lids.

The diarrhœal discharges may vary from three to twenty, but seldom exceed ten or twelve, each

amounting to perhaps four or six ounces, so that on the average the material transuded from the intestines does not exceed three or five pints; the quantity lost by emesis is perhaps much less; further, in either case the amount of fluid lost is not more copious in fatal seizures than in those which recover. The ease with which the contents of the stomach are expelled is most remarkable, since the act partakes of the character of simple regurgitation, occurring in series of efforts, repeated three, four, eight, ten, or more times. The whole period of all discharges varies between eight and twenty-four hours; they then become more and more infrequent, finally wholly cease for several hours, perhaps for a day or two, only to return, perhaps, at irregular periods; vomiting is especially apt to return after the ingestion of fluids. The absence of bile pigment in the stools seldom lasts more than twenty-four hours, when the period of reaction (third stage) sets in and they become yellowish-green; but before they wholly return to normal, there is an irregular exhibition of fæcal material varying between moderate diarrhœa and constipation, unless dysentery complicates.

There is entire absence of fever or febrile condition. The temperature is depressed, the thermometer falling to 93° and to 90° Farh.—rarely below the latter figure, though it has been known to reach 75° Farh.,—notwithstanding which the sufferer complains of oppression and prefers to lie uncovered;

generally during both collapse and reaction, the temperature in vagina or rectum (or both) is three or four degrees higher than in the axilla, which in turn is at least one or two degrees lower than in the mouth.

Malignant (asphyctic) cholera, runs a very acute course, and patients may die at any time in from two to twelve, eighteen, or twenty hours; death, however, is comparatively rare in the first twelve hours, occurring usually in the succeeding ten or sixteen, and when occurring on the second day is usually in consequence of imperfect reaction. The algid stage rarely lasts longer than two days, and the evacuations often cease some time before dissolution, though this is very far from being a favorable sign, since it is due, not to cessation of transudation, but to complete paralysis of intestinal muscles; *per contra*, patients in whom the evacuations continue for a long time, recover more frequently than those in whom they cease suddenly; consequently the occurrence of intestinal paralysis must be regarded as a most unfavorable manifestation, while, on the other hand, persistence of evacuations evidences such paralysis has not supervened, hence justifies more favorable prognosis.

Niemeyer early advanced the opinion that temperature diminishes only at the periphery of the body remaining elevated within, an assumption now generally accepted by the medical world. Through numerous careful observations of the temperature of cholera pa-

tients in the algid stage, Jüterborgk arrived at the following conclusions:

“The head, extremities, etc., are colder than in almost any other disease:

“The temperature of the cavities of the body, such as the vagina and rectum, is the highest (that can be measured) in the body, and should always be taken for measurements:

“Whether the case be favorable or fatal, the temperature within the body is usually increased, rarely normal, and more rarely diminished, although no cause for this has ever been found in the pathological symptoms during life or on autopsy—[Another evidence of the neurotic character of the disease.—S.]:

“The temperature of the whole body usually rises with the approach of death; but there are cases where this rise takes place without one being able to find any reason for this deviation:”

Again: “The commencement of reaction is not accompanied by any elevation of temperature, but the interior of the body usually cools off, while the outer parts warm up:

“In cases of protracted reaction, the temperature of the whole body generally sinks below the normal:

“The inflammatory sequelæ usually, if not always, excite decided elevation of temperature:

“During perfect convalescence, an abnormal elevation of temperature is often seen without any pathological cause therefor being discoverable.”

An abnormal and notable condition of the nervous system, is manifest from the fact that, while the intellect remains clear to the last*—and though the sufferer is sometimes quite hopeful,—there is in general an apathetic condition frequently amounting to complete callousness; there are no apprehensions, and little care as to what the ultimate result may be, though perhaps prior to the seizure there may have been intense dread of the disease.—The terrible clearness of mind and recognition of the end which is said to have characterized the earlier epidemics, has not been witnessed during the present ravages of the disease in Europe. In some cases there is great restlessness and tossing—unconscious movements to-and-fro,—though often the sufferer is quiet, and appears to experience no inconvenience save when disturbed by the evacuations, vomitings, or cramps. Delirium is generally absent, but occurs

*“ It is remarkable, notwithstanding the great debility which makes every motion difficult, and the profound prostration that is expressed in every feature,” says Lebert, “ that the patients not infrequently possess entire consciousness. This, to me, was one of the most disagreeable impressions of the Paris epidemic—to hear sufferers in whom the pulse was no longer perceptible, in whom the face was cyanosed and cold, still speaking with the most perfect possession of all the faculties of the mind.” According to Reinhardt and Leubuscher, some insane patients entirely recover sanity for the time being, though the sanity vanishes with convalescence; others remain insane to the end.—S.

more frequently among alcoholics, and later in the typhoid state (third stage) during which it alternates with sopor; in cases of pronounced uræmia, it is sometimes attended with convulsions. Strange to say, muscular strength, real or apparent, appears to persist in most extraordinary degree to the very last, and patients if not prevented will frequently get up and walk about a few moments before dissolution. Again, "walking cholera" is by no means uncommon, the sufferers keeping their feet until fairly in the throes of death, and in such cases locomotion appears to hasten fatality.

Death is usually peaceful, by asthenia and, as already noted, may take place at any time from two to twenty-four hours after the attack—it is a gradual "going out," the "rattling of the throat" which pertains to most diseases, being conspicuous only by its absence; or if surviving beyond twenty-four hours, there is usually manifest evidence of amendment. Commonly patients become lethargic, the lethargy culminating in sleep; sleep in turn is merged into coma, and coma into dissolution. As in many other severe diseases, there is usually observed an elevation of temperature as the fatal end approaches, while the exhalation of carbonic acid gas is very much diminished; the temperature continues to rise, in many instances even after death, as Davey observed as long ago as 1839—an observation that has since been confirmed in a series of other satisfactory tests. The bodies cool off very slowly.

Regarding the mortality of cholera, it may be remarked no one has ever been able to complete statistics of the graver cases, because the lighter ones for the most part escape accurate observation, while as to the more serious the mortality varies (according to the most conscientious statements), between two-fifths and three-fifths (the average may be put at one-half), though in some local epidemics under unfavorable circumstances, especially in asylums for the aged and for incurables, it reaches as high as two-thirds, or even three-fourths. Nearly one-third of the deaths occur within twenty-four hours, and about one-half of all the deaths occur in the first two days. In the neighborhood of one-sixth die on the third day in consequence of imperfect reaction, and about one-third during protracted convalescence and in the typhoid state—after from four to twelve days. In favorable cases of confirmed cholera, and in half of *all* cases that recover, convalescence occurs in three or four days; in the other half which recover, it occurs irregularly up to the second half of the first week. From the beginning of convalescence to perfect recovery, a period of from three to eight days usually intervenes, varying according to the character of the attack, and the characteristics of the individual patient and his surroundings.

CHAPTER VI.

REACTION AND CONVALESCENCE.

Third Stage.—Although in the preceding stage all symptoms may reach such intensity that a large number of those seized cannot survive, yet in other numbers a third or so-called “stage of reaction” supervenes, which exhibits most remarkable tendency to restoration of physiological function, though certainly not always with equal results; it is possible (though rarely witnessed), for recovery to be most rapid.

The first improvement is manifested by some repression of the discharges; even though emesis and diarrhœa are still persistent, the quantity is diminished and the whey-like character lost; in some instances, thus early, repression may be complete, even to a degree constituting absolute constipation. Another evidence of improvement is when the stomach fails to reject the fluids ingested, whereby assurance is had that the function of absorption is no longer in abeyance, and restoration of the fluids of the circulation, lost by transudation, possible. The capillary circulation is first renewed. Next that controlled by the carotids. Then the radial pulse (which before could not be felt or was scarcely perceptible) quickly regains its strength, and in a few hours is often stronger and fuller than in the normal condi-

tion; usually, also it is rapid, though seldom increased above ninety or one hundred, perhaps with distinct dicrotic beat. The double heart tones also soon become normal and regular, the blowing sound synchronous with the diastole, disappearing. Should venesection now be attempted, the blood will be found to flow almost as freely as in health, though of course, the proportion of serum is greatly diminished. As soon, too, as circulation is restored, the cyanosis disappears, though many patients preserve for some time a marble or cadaverous appearance. Heat gradually diffuses itself into the peripheric parts of the body, in fact often transcends a normal medium temperature; and possibly profuse perspiration may be induced, either of spontaneous character, or as the result of hot drinks; when temperature exceeds the normal, and the cheeks become suffused with dark red, the eyes also injected, lachrymose, and painful, and general evidences of fluxionary cerebral and other organic hyperæmia, a clinical picture is presented that oftentimes is most difficult to interpret; it sometimes disappears spontaneously, and again is evidence of imperfect reaction and threatening sequelæ. Such congestions are more frequent, violent, and dangerous in children; indeed, the stage of reaction is, in the main, more intense in the little folk, though of shorter duration, and demands watchful care. Even most adults complain of a feeling of cold and heaviness in and about the head, more manifest

about the occiput or sinciput; roaring of the ears or tintinabulations are common; and notwithstanding a certain tendency to somnolence, those persons most enfeebled are usually sleepless.

Cramps cease, as a rule, with the beginning of reaction; but the urine remains scanty, or altogether suppressed, for twenty-four or more hours, and always exhibits traces of albumen as soon as passed, which traces persist for from two to seven days.* Respiration is normal; dyspnœa absent or nearly so, having been markedly lessened toward the conclusion of the second stage. When the convalescence is rapid, the tongue clears off, the bad taste is lost, appetite returns, sometimes to a degree that causes error in diet and consequent relapse. The discharges from the intestines may persist, but soon assume a more

*In observations at Zürich, Lebert found the first urine after total suppression, was not passed until forty-eight hours after the beginning of the disease. As a rule it was regarded the secretion would be restored in the course of the third or in the beginning of the fourth day. "The first urine passed was, usually, small in quantity, in two cases bloody, and once attended with violent pains about the kidneys. Several hours, from eight to twelve, usually elapsed between this first and the second discharge. Specific gravity varied between 1.007 and 1.010. At first there was considerable brown coloring matter present, and on boiling with nitric acid it often showed a light bluish tint (indigo coloring matter). Only once was the first urine somewhat cloudy, without albumen; in all other cases albumen was present, and

natural color and solid consistence; the casts and albumen disappear, and progress toward health is rapid, so that the latter, barring accidents and slight after-pains, may be regarded as established in from ten to fourteen days subsequent to the primary seizure. Says Lebert, "If nearly half the patients die in the algid stage, in more than half of the rest, the stage of reaction goes on to favorable termination." In women, metrorrhagias, during or in the intervals of menstruation, are not infrequent during reaction and convalescence.

At all periods of life, especially in advanced age, reaction may be imperfect, may even be followed by a relapse to the second stage; yet many cases recover in spite of numerous vicissitudes and fluctuations: Or the diarrhoea and vomiting may recur from time to time with critical symptoms; or a dysentery may alter-

remained for three or four days and sometimes longer; the quantity of albumen varied, and when it disappeared the quantity of urine became much more copious."

According to investigations of Lehmann and Volk, confirmed by Prof Buhl, of München, the first urine voided is only quantitatively small and albuminous, but contains traces of sugar, a little sodium chloride, and relatively very little urea; but in the two succeeding days the quantity of urine, as well as its relative proportions of urea and salt, greatly increases, even far exceeds the normal, and then, after some variation, again returns to the natural condition, when the albumen, casts, and abnormal pigment disappear, and the specific gravity becomes normal.—S.

nate with obstinate constipation, the former green or greenish yellow and gelatinous; the tongue does not clear up; anorexia, bad taste in mouth, and thirst, continue to torture; the little nourishment taken is either speedily rejected or induces profound distress.

Catarrhal inflammation of the genito-urinary tract is especially apt to delay convalescence, and often in conjunction with, or succeeded by, a diphtheritic condition of the *prima viæ* induced by irritation of the denuded intestines or their contents. Most patients who fall into this state die of exhaustion.

Pneumonia, or so-called typhoid pneumonia, is especially apt to supervene; and Niemeyer remarks that "in old, decrepit persons, if physical examination be neglected, the outward resemblance and the subjective symptoms often cause pneumonia to be diagnosed as catarrhal fever, nervous influenza, typhus, etc." According to his experience, acute croupous nephritis, with the retention of urine it causes by plugging of the uriniferous tubules, is a common sequel of *cholera asphyxia*, but by no means the constant cause of cholera typhoid, as has often been asserted.

"If the secretion of urine remains suppressed after the disappearance of the symptoms of collapse, or if the scanty urine contains quantities of albumen and fibrinous casts for days; if vomiting recommences and the patients complain of severe headache and become comatose, or have epileptiform convulsions;

it is safe to make a diagnosis of acute croupous nephritis with so-called uræmic intoxication; in such cases the skin has occasionally been found encrusted with crystallized urea." (Niemeyer.)

If the first or second day after the cessation of the asphyctic symptoms the patients do not pass a normal or at least large amount of urine, or the albumen, at first very constant, does not disappear after a few days, they are apt to fall into a state of exceeding apathy and stupor, or muttering delirium, when the tongue becomes dry and crusted, the pulse frequent and often double, the temperature elevated, and they slip down toward the foot of the bed; indeed the condition so exactly resembles severe enteric fever as to fully warrant the title of cholera typhoid. Besides, there is usually a fœtid diarrhœa in which are discovered shreds of epithelium; and while the patients can scarcely be aroused from the comatose state, they twitch the face, or recover consciousness and complain of pain, if strong pressure is made upon the abdomen.*

If there is catarrhal or diphtheritic inflammation of the intestine, or of the genito-urinary tract, a pneumonia, a pleurisy, or other of the inflammatory sequelæ of cholera, the appearance of the patient does not materially differ from the above description. The

* So called *cholera typhoid* is one of the most common forms of protracted convalescence, and considered by Frerichs as a uræmic condition purely.—S.

typhoid peculiarities prevail in completeness, while the symptoms of the original or local disease become subjective, falling into the background or disappearing entirely. Finally, in many cases, neither during life nor on autopsy, is it possible to discover any local lesions to which can consistently be referred the exhausting fever, of which so many die after the cholera proper has run its course—further evidence of nerve-toxæmia.

Particular importance has been attached by some to the fact that during the so-called cholera typhoid a maculated, papular, erythematous exanthema has been observed, that may appear of decided urticarial character, or show a roseola-like appearance; it differs from the eruption of typhus in that it begins at the toes and spreads up to the trunk, where it is most manifest, becoming very imperfect on the face and head; the spots and papules may also run together and form a diffuse redness in different places—very much as is seen in certain forms of malaria. This eruption seldom manifests itself before the end of the first, and often not until during the second week, and notably most of the patients thus affected recover; it is not, however, so constant a symptom of cholera typhoid as to be pathognomonic, and is more apt to occur, perhaps, where sinapisms have been repeatedly or continuously applied to the extremities during the algid stage, or massage has been energetically used.

Sleeplessness, a condition of sullenness, etc., sometimes supervenes during apparent convalescence, when

the patients may either fall back into the condition of asphyxia, or continue to improve; but the latter is usually at the expense of numerous suppurations, manifested as a crop of boils, perhaps as abscesses of the parotid or of the larynx, or by general pyæmia.

It is needless to remark that this third stage is one of danger accordingly as it manifests in greater or less degree the pneumonic or typhic condition, though in any event it is apt to induce general impairment of the system that persists for a long period.

Of the anatomical changes that take place during or supervene upon a cholera attack, it is impossible to speak in complete detail. However, the great withdrawal of serum from the blood enables the bodies of those deceased to resist decomposition to a remarkable degree, hence the changes usually encountered post-mortem are lacking. It has before been remarked that the corpses are greatly shrunken, of dusky or livid color; that rigor mortis is rapidly developed and persists for an unusual length of time; and that very remarkable and violent contractions of the muscles are by no means uncommon, so much so as to give rise to weird tales of unfortunates buried alive, etc.*

Bodies of those who succumb six, twelve or eighteen hours after the attack, exhibit the same

*See pages 112, 113.

cyanotic appearance, and collapse of features, as in the last hours of life.

The circulatory organs and the blood exhibit the following conditions: When death is early, absence of the pericardial fluid is noted, or it is scant and sticky; later it is normal or slightly increased. The portion of the pericardium, which lies open and is attached to the outer side of the heart, *i.e.*, the visceral layer, is almost constantly the seat of ecchymoses, most numerous towards the base and posteriorly; it is rare to find on the parietal layer. Much more blood appears in the right heart than in the left—indeed the left heart is almost always empty,—which is apt to be of a pappy appearance, or exhibit soft coagulable and fibrinous clots, the latter gelatinous, or firm and colorless, either of which conditions may be present in the typhoid stage.

Lebert remarks he once found a fine fibrinous clot separated in the form of a membrane spread over the whole inner surface of the right ventricle; and that the perfectly soft dissolving clots which are often seen, correspond to no particular stage or condition. He also made a chemical examination of the blood of a patient dying in the typhoid stage, that revealed no increase either of urea or carbonate of ammonia as a constant condition, yet adds he would not “like to draw conclusions from these individual examinations.” Virchow admirably described the increase in the number of the white corpuscles in the heart clots.

As to the respiratory organs, they are seldom affected in their principal portions, though occasionally secondary diphtheritic and pseudo-membranous processes are encountered. The mucous membranes of trachea and bronchi are very much engorged with blood in cases of early death, and when there is moderate hyperæmia often covered with mucus in which are discovered more or less leucocytes; in exceptional cases the glands of the trachea are considerably swollen. Ecchymoses are not uncommon, but appear more frequently on the surface of the lungs, which latter are deeply engorged with blood, especially in their inferior and posterior portions, and often œdematous. Purulent mucus in the smallest bronchi, and the anatomical lesions of broncho-pneumonia and of typhoid-pneumonia, are conditions sometimes seen in cases of death—after three, five, or eight days; and pleuritis with sero-purulent effusion also belongs to these rarer complications; hæmorrhagic pulmonary infarctions are not infrequent.

The isolated œsophageal glands are often excessively swollen, the tube itself being cyanotic in the algid stage and ecchymosed at a later period. In his Zürich autopsies, Lebert often “found the epithelium detached, and once the lower part of the œsophagus covered with fibrinous diphtheritic membranes.” The stomach is distended and filled with colorless fluid in cases of early fatality, but later is empty and collapsed; when death occurs after the third or fourth

day it is apt to be filled with yellowish-green, sticky, gelatinous or mucous fluid; and the mucous membrane, at first hyperæmic, shows later numerous ecchymoses, and occasionally spots of bloody infiltration; when death occurs late, it is covered with abundant, tough, thick mucus, and perhaps spots of softening that probably are, in part at least, the effects of commencing decomposition.

The most marked changes, however, are found in the small intestine. Where the malady runs a rapidly fatal course, the intestinal peritoneum is dry, of a rosy color, or covered with a light layer of sticky fluid. In more prolonged cases the lesser bowel contains a greenish pultaceous mass, while the colon cœcum, etc., may harbor half-solid fæces; in the early periods the contents are of "rice-water" character.

During the attack proper, and immediately following, the glands of the small intestine are chiefly affected: Brunner's first, a condition that is constant; then the isolated and agminated glands, the former standing out in relief, their size varying from that of a millet-seed to a pea; Peyer's patches are granulated on the surface; swelling and engorgement most pronounced toward the ileo-cœcal valve. Aside from the hyperæmia and ecchymoses, the prominent glands give the surface a pale, milky, or yellowish appearance, and if the follicles are pierced they exude a whitish-gray fluid with fine granules and cell-nuclei, without leucocytes; the surface is smooth, for

the most part deprived of epithelium and villi, and the engorged glands admit of perfect artificial injection.

These typical changes are generally found in the first forty-eight hours; Lebert often observed the engorgement began to diminish at the end of from thirty-six to forty hours, though in some cases the glands continued infiltrated for four or five days, particularly if this condition persisted in the tissues immediately adjacent; as a rule, however, infiltration rapidly diminishes at the end of the second or in the course of the third day, when the glands present a flattened and somewhat wrinkled appearance, later becoming almost granular; they are still prominent, however, though shrunken in circumference, and of yellowish-gray (later almost slaty) color; occasionally a blackish-gray, brown, or brownish-red pigment is noticed, especially if ecchymoses have previously existed. In the second week, with rare exceptions, all infiltration and congestion disappears, though the glands may still continue thickened and abnormally colored. In the first period Peyer's patches are often found converted into a net-work, the follicles fissured as if ruptured; and as this condition is present in bodies twelve to eighteen hours after death, the supposition it is a post-mortem phenomenon is manifestly incorrect. In cases of early death, in some epidemics, the patches are ulcerated as in typhoid fever; the glands of the large intestine, also are found swollen,

prominent, and they likewise collapse at a later period, showing the same retrograde changes as those of the small intestine.

The mucous membrane between glands may share in the infiltration, and in the first stage is very deeply congested (almost cyanosed), generally ecchymosed or with extensive extravasations, so that great patches of mucosa are deeply ensanguined, a condition more frequently observed in the colon than in the small intestine; at an early stage also it quite often is softened, swollen, even œdematous, a condition that later is confined chiefly to spots; extensive softening of either the small or large intestine, however, is rare. The same changes in color, as noticed in the retrograde metamorphoses of the glands, occur throughout the mucous membrane, though less pronounced.*

The anatomical characters of secondary colitis of a diphtheritic or dysenteric nature, are seen comparatively often in some epidemics, in others are almost entirely absent. The mesenteric glands are quite often moderately swollen, but usually without much infiltration.†

“The spleen is in general small, rather wrinkled and shrunken, of good consistence, and moderately

*Lebert faithfully pictures all these details in his “Atlas of Pathological Anatomy.”—S.

†For the succeeding anatomical descriptions I must acknowledge my indebtedness to Herman Lebert’s monograph on the Zürich epidemics.—S.

supplied with blood, though sometimes seen enlarged in consequence of apoplectic effusion; in cases where cholera complicates typhoid fever, it is usually enlarged.

“The liver, in speedily fatal cases is often hyperæmic, and shows also numerous sub-peritoneal ecchymoses; at a later period is pale, marble-yellow or red, with isolated islands of fatty degeneration; the gall-bladder distended, in the first period with dark-brown bile, later of a bright-green color, semi-fluid, resembling mucus. Catarrh of the biliary passages, even of purulent nature, occasionally develops as a secondary affection. During the attack the bile is retained, but later, when again discharged, during a protracted convalescence or in the typhoid state, it seems to be abnormally constituted—a fact which makes chemical examinations at this period much to be desired.

“The bladder usually shows nothing abnormal; if death occurs in the first two days, it is contracted and empty. Still I have found in it, in exceptional cases, an ounce or more of cloudy albuminous urine, even after the disease has lasted from thirty-six to forty hours. Usually a little urine is found in the bladder in case of death on the third day, though often it may be empty even when death occurs on the fourth or fifth day. The mucous membrane of this viscus, in the beginning takes part also in the general cyanosis, but it is comparatively little marked and of little consequence.

“ The kidneys may early take part in the disease processes, and even when death occurs in from sixteen to twenty-four hours, there is always observed an increase in the volume, and at the same time they are generally filled in both the cortical and medullary substances with blood in the form of stripes and punctated injections, and on the surface in star-shaped and marbled spots, with numerous and thick anastomoses; the superficial inter-canalicular vessels and capillaries of the Malpighian glomeruli also share in this congested condition, and ecchymoses are likewise not infrequent. Even in cases of death in the second half of the first day, the cortical substance of the kidneys is often found in an unmistakable condition of commencing decoloration, extending even from the surface deep down into the pyramids; also the capsules are frequently abnormally adherent. The microscope reveals at this early stage a remarkable epithelial proliferation in the urinary canaliculi, with cloudy swelling of the cell, the contents of which (consisting of numerous albuminoid granules) may be dissolved by acetic acid. Now and then may be discovered, as early as at the end of the first day, distinct transparent cylinders in the interior of the urinary canals. The kidneys, therefore, are decidedly affected on the very first day of a pronounced attack of cholera.

“ In the course of the second day I have noticed either the same commencing decoloration, or more

marked changes. The hyperæmia is now either confined to spots, or general with simultaneous decoloration of the canals and cortex; casts are present in great quantity—pressure empties from the papillæ a cloudy albuminous urine containing them, and not infrequently crystals of uric acid. The mucous membrane of the calyces and pelves is usually hyperæmic, with injection of the fine vessels, and the microscope reveals progressive degeneration of the cells. In the course of the third day the decoloration is so far increased as to involve the whole cortex, and granulations are present; the blood seems to be very unequally distributed; the surface, uneven, rough, and closely adherent to the capsule. The cells continue to be detached, the development of casts proceeds, the fatty elements increase and now show themselves as granules and oil drops in constantly increasing quantity in the epithelium and outside of it, in the interior of the canaliculi, and in the casts.

“These alterations increase in the typhoid stage as well as during imperfect convalescence. The kidneys, according to many accurate measurements, are from one-sixth to one-third larger than normal, the granulations more abundant, and the decoloration advanced to the pyramids and even between them. The substance of the kidneys is now softer, more easily torn, and infiltrated with a dirty yellow, fatty, and albuminous fluid. The mucous membrane often

seems thickened. In cases where death occurs late, all the signs of resolution are present; in favorable cases, all these seemingly grave lesions quickly disappear, and the kidneys regained their normal condition. Strange to say, chronic nephritis caused by or incidental to cholera, is rare" (Lebert).

The chemical examinations of the various organs undertaken by Staëdeler, in Zürich, in 1855, yielded no special results. Leucin was found in the liver, and small quantities of uric acid in different organs. In the spleen, leucin was once detected; in other cases inosite, uric acid, and much pigment. The kidneys contained comparatively a great amount urea, some leucin, bile-pigment, and uric acid, but no inosite.

Glancing now at the whole duration of cholera: For the period of incubation may be allowed from five to seven days, often much less, sometimes longer. Where prodromata exist, their average duration is from one to three days. Next comes the stage of attack, which is the second, or (when prodromata are lacking) at times the first stage; this may prove fatal, in from six to eight hours, or even less, but varies in fairly severe cases from twelve to twenty-four hours.

With the end of the attack proper (the second stage) comes the period of reaction, usually at the end of from eighteen to twenty-four hours, and now the patient either dies from cyanotic asphyxia, or the reaction is perfectly established and the real cholera

is happily and definitely over. The stage of reaction may pass into speedy convalescence, which may terminate in the second half of the first week; or the convalescence is protracted, either without further critical symptoms or with transition into the typhoid stage. The typhic stage, in turn, may lead to fatal or fortunate termination in the last days of the first, or in the first days of the second week of the whole duration of the disease. It is a rare exception for patients to die of cholera after ten or twelve days, or to be affected with long-continued, bad sequelæ, though a weak invalid condition may persist for a long time as the result of defective nerve action.

Finally, it must be remarked that it is impossible, no matter what the scope of the work, to describe all the sequelæ and complications of cholera; where the circulation is so profoundly disturbed, and the nerve toxæmia so virulent, the most multiform local congestions and inflammations are possible.

CHAPTER VII.

CHOLERA DIARRHŒA AND CHOLERINE.

The mildest form of cholera is that simulating a simple diarrhœa, in which the evacuations follow each other more or less closely, are very copious and watery, but *not* altogether devoid of consistence and color, and retain in some degree the characteristic fœcal odor; they perhaps are not accompanied by colicky pains or tenesmus, and cause no constitutional or other disturbance except a moderate degree of depression and relaxation. Such frequently do not appear in official lists, but, as Niemeyer pertinently observes, "though not considered by the police as cholera, they should be so recognized by science. This is shown:

(1) "By the larger number of cases of diarrhœa occurring during cholera times, although almost all sensible people carefully avoid errors of diet, catching cold, and other sources of injury:

(2) "The great obstinacy of these cases:

(3) "The well known transportation of the disease by persons suffering therefrom:

(4) "Especially by the numerous transformations of simple cholera diarrhœa into the most severe form of the malady.—Many patients, especially of the poorer classes, worried by a diarrhœa which will not give way to domestic remedies, go to the doctor for a prescrip-

tion at noon, and in the evening lie cold, pulseless, and cyanotic, almost in a hopeless state. . . . I deem it much more important to determine the frequent occurrence of gradual transformation from simple cholera diarrhœa to so-called cholérine, and to malignant cholera, and to prove the identity of these three forms, than to seek for pathognomonic signs of epidemic cholera.”*

Cholera diarrhœa and cholérine, which constitute the milder forms of the disease, during the prevalence of epidemics also present varied phases, and more than all varied characteristics in different epidemics. Lebert, Zehnder, Karl Liebermeister, and others, have noted how greatly personal predisposition favors or inhibits, as the case may be, extension or repression; that an imported case in any one locality may be confined to a single individual, to a single house, even to single room in a dwelling, while at other times one case may be the focus of a raging pestilence. Thus the history of different epidemics, particularly in large cities, shows the greatest variety

* “Text Book of Practical Medicine,” Vol. ii. New York, 1884.

* How probable it is that a swiftly fatal attack is developed from profound toxicity of the central nervous system through the medium of the absorbents, especially those of the portal area, by an excess of the cholera poison, is shown by phenomena developed in acute arsenical poisoning, which has several times been mistaken for true cholera (see p. 107).

of effect, accordingly as the cholera poison finds conditions for development more or less suitable. Again, it has been observed, as a rule, that prodromic diarrhœas are more frequent and more widely diffused in malignant and extensive epidemics than in those of less extent. Yet Lebert declares,† in the great epidemic in Paris of 1849, which is computed to have claimed something like ten thousand victims, premonitory intestinal flux was wholly absent in from five to ten per cent. of the pronounced cases; in Zürich it was absent in 33.3 per cent. of pronounced cases; and that absence or presence of prodromic diarrhœa had no apparent influence in determining the ultimate result of the attack, as the recoveries and deaths were about equally proportioned in regard to this symptom. Also, he adds, "I found that the prodromic diarrhœa was absent in seven-eighths of the cases of true cholera (with colored stools). In Paris as well as in Zürich and Breslau, in 1866 and 1867, I saw a number of cases of diarrhœa which were due to the influence of cholera, recover without treatment and without subsequent cholera. On the other hand, in July and the beginning of August, 1866, I witnessed such obstinate and violent cases of cholera diarrhœa in the Breslau garrison of cuirassiers, where I had charge of a large ward of wounded patients, that it required the utmost effort to prevent an outbreak of cholera."

† Ziemssen's "Cyclopædia of Practical Medicine." New York, 1874.

There is no marked distinction between common intestinal catarrh and cholera diarrhœa, yet the latter presents certain definite peculiarities which are entitled to consideration in seeking a definite diagnosis.

Cholera diarrhœa is usually sudden and unexpected: As already remarked, is apt to be excited, or receive its ultimate development, through "taking cold" or errors of diet; and to *intemperance* in the habitual use of alcoholic beverages more than any other single cause may the proclivity to this form of flux be ascribed.

Loss of appetite, thirst, indigestion, are either present or absent in only slight degree; patients generally, however, complain of an excessive feeling of lassitude—are extraordinarily weak, feeble and uncomfortable,—and not unfrequently exhibit despondency, out of all proportion to the frequency or quantity of the flux.

There is nothing specially peculiar about the stools other than they are simply fluid, as in any diarrhœa; often there are but two or three discharges during the twenty-four hours, more rarely from six to eight, and are more apt to supervene during the night, and to be accompanied by distressing rumblings and gurglings.

In some cases the diarrhœa persists only a few hours or days; in others, one or two weeks, with considerable intermissions, during which the individual feels perfectly well. Sometimes it regularly intermits or

remits during the whole course of the epidemic in that particular locality, and ceases only as the latter disappears. Even in the milder cases of cholera diarrhœa may be sometimes observed individual evidences of cholera proper, such as suppression of urine, light cramps in calves, colorless and odorless "rice-water" stools, that are merely of temporary duration, and are more generally characteristic of cholera. Neither is it a rare occurrence for a cholera diarrhœa, which remains in other respects without danger, to suddenly increase in virulence, with violent, quickly repeated stools, as in true cholera, and yet cease quickly and be followed by prompt recovery.

Cholera, as the term indicates, is a lesser cholera; in other words, it is a condensed picture of the mildest form of the more grave attack, and may, indeed often does, present some of the serious symptoms peculiar to the latter. It is usually ushered in by such prodromal symptoms as malaise, headache, diminution of appetite, muscular or general physical weakness, of at least twelve and often twenty-four hours' duration; there is also general restlessness, insomnia—or at least the hours of sleep are productive of discomfort rather than rest; and the attack culminates in the middle of the night or early morning by sudden awakening due to demand for stool. Now is observed a copious yellowish-brown, almost watery discharge, with characteristic diarrhœic odor, but succeeded by a second still more fluid, and others

again at brief intervals to the number of three, six, eight, twelve or more, until—when they become very numerous—there is gradually less and less evidence of odor, color, or form, and they assume a decided “rice-water” character.

With the first evidence of diarrhœa, nausea is apt to supervene which, after repeated stools, is merged into emesis, the ejected matter being first of a yellowish green hue with intensely sour bitter taste, very fluid in character, finally, perhaps, colorless, whey-like, showing a deposit very like grains of bruised rice; after a few times, the quantity is lessened, the act itself becomes more infrequent, and finally it ceases altogether after a few hours; this vomiting is not at all painful, retching being a comparatively rare concomitant, but the fluid pours forth in a manner resembling an easy act of regurgitation, though its profuse quantity is very suggestive of its choleraic nature. The sufferer in the meantime becomes very much reduced; but with the cessation of vomiting and diarrhœa, either recovers very rapidly, or temporary typhoid symptoms manifest themselves. While complete convalescence may be established in a few days, it is often a matter of two or three weeks. In the favorable cases of cholera, convalescence occurs in two, three or four days.

Sometimes the inclination to diarrhœa lingers for some days after the convalescing stage has set in, with all the concomitants of anorexia, borborygmus,

occasionally twinges of colic, etc.; not infrequently also there is continued nausea, especially after the ingestion of food; and a distressing tendency toward cramping of muscles of legs (calves) is more or less apparent according to the severity of the attack. Lebert remarks he has also seen moderate cooling of extremities; that he personally experienced almost complete aphonia for twenty-four hours after a seizure, and did not fully recover his voice until "at the end of several days;" also noticed considerable reduction in the quantity of urea, as well as the temporary occurrence of albumen and casts in very dark scanty urine. It is perhaps needless to say that gastric catarrh not infrequently interferes with convalescence; that errors in diet may lead to fatal relapse; and that the temporary typhoid state, when it supervenes, is manifested by such symptoms as headache, vertigo, roaring in ears, cloudiness of vision, great debility, sopor, etc.

It is also of interest to remember that where this "little cholera" is seen in houses and families, frequently in the course of a few days a malignant asphyctic cholera will develop, and that it therefore may lead to genuine and fatal cholera when the individual (or individuals) is sufficiently receptive toward the poison; that cholérine may itself prove fatal in weak and aged patients; and when cholera prevails in a large city with great severity, the number of persons attacked with real cholera is always relatively

smaller than those attacked with cholera diarrhœa and cholérine. Moreover, cholera diarrhœa and cholérine occur much more frequently in places some distance from the centres of the disease, while true cholera prevails at these centres. In all these cases it is probably the diminished capacity of the surroundings for infection accounts for the relatively greater dissemination of the lighter forms of the disease; but, as just remarked, these lighter forms by rapid multiplication, or increased virulence of the poison of the disease, under favorable conditions of the organism, may lead to dangerous attacks of cholera.

In enumerating these phases of the epidemic more specifically, it must be understood that in differentiating the three forms there is no recession from the position I shall, in a later chapter, endeavor to establish that all are *de facto* cholera. And, further, it may be added, it is a matter of great personal doubt whether *any* cholera is derived from a special germ or poison, other than that resultant upon any common decomposition; or possibly that the Asiatic form is due to the common poison supplemented by an imported one, that after all is but the same intensified in its original habitat and by transmission; or, again, that all cholera is due to a poison of common decomposition that was foreign originally, but since importation has become in a sense acclimated. There are few medical men who have not seen cholera nostras—or as it is termed in

this country, sporadic cholera, or cholera morbus—with positive “rice-water” discharges, colorless and odorless, outside of the period of any epidemic; I have repeatedly encountered such in my own experience, and in the service of others; and Lebert, J. M. and D. D. Cunningham, Bouchard, Leiter, Zehnder, Fayrer, Niemeyer, Trousseau, Semmola, Tanner, Sir Wm. Aitken, and a list who are fairly “Legion,”* corroborate, and have also expressed the opinion cholera infantum is only cholera sporadica, *modified by age*.—*A’ propos* of this it may be mentioned that Chas. Talamon† has recently adduced considerable evidence tending to demonstrate the unity of these maladies :

He points out that ravages of cholera diarrhœa, of epidemic nature, occur from time to time without any evidence of a foreign or imported factor, in localities in which true cholera has, sometime or another, manifested itself; and likewise reference is made to a recent epidemic, the exact prototype of another occurring in 1866, in the neighborhood of Paris, clinically diagnosed as cholera nostras, but which on investigation in no way differed pathologically from the so-called Asiatic malady. The cholera bacillus was demonstrated in the dejections also.

*See “Nature and Treatment of Sporadic Cholera” by Alex. Harkin. W. Renshaw, London, 1885.—S.

† La Médecine Moderne, 1892.

CHAPTER VIII.

SPECIFIC PATHOLOGY.

I have been thus particular in enumerating the general outlines and peculiarities of this disease, which presumably are familiar—or, at least, they should be,—to every medical man, in order to more forcibly call attention to their physiologico-therapeutical relations. It has already been remarked, the neurotic character of the disease is most apparent, and that there is no relation between the quantity of fluid lost by the bowel and stomach and the malignancy of the onslaught.

The simple diarrhœa which ushers in an attack, or that may constitute almost the only manifestation of the malady, is especially remarked upon by all observers for its stubborn character; it yields to none of the customary remedies, and is influenced scarcely at all by opiates or astringents. This presupposes a lesion deeper than, and behind, the visceral and thoracic organs that, apparently, are most involved, and one has no option but to refer the same to the *central nervous system*.

In 1870 M. F. Moreau, and also S. G. Vasquez, demonstrated by a series of observations,—corroborating those of Edward Pflüger and Otto Nasse, and since repeated by many others,—the influence of the sympathetic nerves upon the intestinal canal,

which influence is most pertinent as evidencing the source and character of the watery diarrhœa in cholera, and likewise its sudden occurrence without any form of premonition or warning.

Both found when these nerves were divided, the portion of the intestines segregated by the operation rapidly filled with clear alkaline fluid, colorless and slightly opaline—except as, at the outset, they might become mixed with material still remaining within the gut,—which precipitated flocculi of organic matter on boiling.* Transudation of watery fluid into the intestines, due to capillary diffusion, takes place as the result of paralysis of the sympathetic; the occurrence of sudden hyperæmias, transudations, and ecchymoses, in some thoracic or abdominal organ, may have a neurotic basis.†

Paralysis or hyperæsthesia of the sympathetic—conditions that differ physiologically *only in degree*,‡—induce vomiting, retching, etc. When the nerves are divided below the solar plexus, which is situated in most intimate relation with the splanchnic area, the

* *Vide* Flint's "Physiology of Man." New York, 1874.

† *Vide* Carpenter, Dalton, Draper, Kirk, Paget, Landois and Sterling, Foster.

‡ There are several circumstances that support this view, chief of which are, the association of pain with both hyperæsthesia and numbness, especially in highly sensitive parts, and the difficulty of differentiating the condition producing the one from the other. "It is clear," says Dr. C.

secretion of urine and urea sinks quickly to a minimum, and may perhaps cease entirely, as shown by Coni Peyrani, who is corroborated by Brown-Séguard and Claude Bernard. These latter authors, moreover, further observed that partial segregation of the sympathetic below the inferior cervical ganglion induced marked depression of temperature, made more manifest toward the surface and in the cutaneous area, but succeeded by rapid *increase* of temperature after death. "Paralysis of vaso-motor nerves decreases temperature," says James Kirk; and the sympathetic pertains to the vaso-motoric system.

Lebert declares that while the most important and constant anatomical clinical localization of the disease occurs in the small intestines, there "is a possibility the often so violent discharges of serum are incited by the central nervous system, the excitement originating in the *vaso-motor centres*." Again: "The cerebro-spinal fluid is entirely absent in cases where death occurs at an early period, or is present only in slight quantity and of almost pasty consistence. But I have seen it more copious when death has occurred

Handifield Jones, "there is no opposition between them: all are present together. Now paralysis, numbness, anæsthesia, hyperæsthesia are evidently failure of functional power; and of the same import is the occurrence of various degrees of paralysis, or of paresis, paræsthesia, analgesia, which may be, and are, associated as analogous affections of the motor and sensory nerves."—S.

at the end of thirty-six hours; and, where death occurs still later, it may even exceed the normal amount. The pia mater loses its marked hyperæmia in a more protracted course of the disease, and becomes dry, perhaps icteric. The fluid of the ventricles remains scanty even when death occurs at later periods. * * * Ecchymoses of the external surface of the brain (pia), or on the internal surface (ependyma) are not rare. There may be capillary effusion into the pons, and once I saw a fresh effusion of blood between the dura and arachnoid."—All this is certainly suggestive.

Again, it must be borne in mind that while "rice-water" stools accrue to all choleraic attacks, except the most pronounced and violent form known as *cholera asphyxia*, *cholera siderans* or *cholera sicca*, they are, *per se*, by no means pathognomonic, but result always from influences of the most powerful character brought to bear upon the central nervous system. They are a concomitant of terror and mental perturbation; of heat apoplexy; of arsenical poisoning;* of autogenetic

* How probable it is that a swiftly fatal attack is developed through nerve irritation manifested in a portion of the small intestine, is shown by the action of arsenic, which may induce fatal poisoning in a few hours, with symptoms perfectly resembling those of cholera. Late in the autumn of 1854, a woman who had been suddenly taken sick during a criminal trial, entered Lebert's Clinic in Zürich. She died in hospital after a number of violent, perfectly colorless,

toxication by specific ptomaines; of toxication by certain alkaloids, etc.; and, moreover, the dejections, as in cholera, hold a profusion of material commonly supposed to be intestinal epithelium (but which is really the result of transformation taking place in the effusion of blood plasma), and likewise contain serum-albumen, mucin, and a large amount of salts, chloride of sodium preponderating to such an extent as often to exceed in amount all organic matters.* But the blood in cholera, as an almost invariable rule, is free from bacteria, either actual or potential; this is the case as well shortly after death as during life, and holds in regard to every stage of the disease.

Further, choleraic, arsenical, and alkaloidal poisonings, of a particular class, likewise thermic apoplexy, alike induce a high specific gravity of the blood—an

very copious discharges. The small intestine showed an immense accumulation of colorless fluid, and clinical examination of the contents of the stomach revealed unmistakable evidences of arsenical poisoning. Lebert further adds: "In the summer of 1847 I was told by Louis, in the Hotel Dieu, that the Duke of Choiseul, who had been arrested the day before for the murder of his wife, died suddenly of cholera, and he wondered at it greatly, because cholera was nowhere prevalent. Louis was the Duke's physician and, as is well known, one of the greatest diagnosticians of our time; nevertheless it soon turned out the Duke had poisoned himself with arsenic."—S.

* The proportion of solid constituents in all, varies from 1.20 to 2.40.—S.

average of 1.0701, against 1.0503 in health,—which is remarkably tough and viscid, the corpuscles increased in number but abnormally impoverished as to salts; the amount of fibrin is unaffected, but the serum is very dense, extremely rich in albumen, and contains more phosphates and potash salts (though less collectively) than normal; moreover, contains some urea, together with extractive matters that seem to possess the quality of rapidly converting the former into carbonate of ammonia (Day, Hoppe-Seyler, C. Schmid). Draper also remark: “In cholera the constitution of the blood is so changed that the cells can no longer carry oxygen into the system; the heat-making processes are put a stop to, and the temperature declining, the body becomes of marble coldness characteristic of this terrible disease,”—phenomena that accrue to *muscarine* and certain other alkaloidal poisonings.

Armand Trousseau says the varied and peculiar symptoms exhibited by asphyctic cholera, can be explained only by referring to a poison having specific effect upon the nervous system; and Wilhelm Erb adds,* referring to poisons as a class: “They cause lasting paralysis, as a rule, only when their action is slow and repeated, more rarely when they are acute; they cause the most varied forms of paralysis and paraplegia, from simple weakness and paresis to com-

* Ziemssen's “Cyclopædia of the Practice of Medicine.”
New York, 1874.

plete paralysis, . . . with or without disturbance of sensibility.

One of the functions of the sympathetic, if the deductions of Jno. W. Draper maybe accepted, is “the equalization or balancing of nerve force, storing up all transient excesses, and providing for all transient deficiencies.”

This sympathetic nerve system, too, transmits sensations so tardily that the economy may be violently, even fatally poisoned through the central nerve organs, long before the customary effects are manifested by the usual symptomatology. Says Jno. C. Dalton :* “Evidences of sensibility are much less acute than in other nerves, and show themselves *only after prolonged application of the exciting cause*”!

Damp, cold, malaria, chorea, and certain poisons, all act in the same precise way by deranging molecular nutritive actions of the nerve structure, and so unfitting the latter to fulfil its function : and anæsthesia and hyperæsthesia are alike failures of functional power, varying only in degree. Again hyperæsthesia and irritation may be the result of dual action, viz : Of poisoning of cerebral centers, and of local irritation.

The foregoing most certainly sheds new and most welcome light on the pathology of cholera, especially the frequent steady march to fatality during moments

* “ Human Physiology.” New York, 1890.

that promise so much in the way of amendment as to mislead the most astute observer.

And herein perhaps lies the arrow that fatally pierces the joints of our therapeutic armor, when is sought to oppose the disease by treating that which is most apparent—the local manifestation; the evil has been wrought long before its faintest symptomatology is made appreciable to human understanding, and consequently is apt to be beyond the power of any method or theory of antagonism or revulsion, however perfect. In cholera, then, often the patient may be *fatally poisoned* before the presence of the disease can be detected or even surmised.

In *facies cholericæ* is observed a condition that may be duplicated by segregation of the superior cervical ganglion of the sympathetic, or by its extirpation; the eyeball is drawn back into the orbit, causing partial closure of the upper and lower lids and flattening of the corneæ; the countenance becomes withered and ghastly; in short, is brought about a cadaverous aspect that sometimes precedes death in long-standing disease, but here supervenes in an hour or two.

When death occurs during the invasion of the disease or in the stage of collapse, in the more marked cases the appearances, as previously shown, are: The bodies remain warm for some time—the temperature may rise after death, perhaps even to 103° Farh., and so continue for several hours (note the observations

of Brown-Séquard and Claude Bernard, before cited); the rigor mortis* soon appears and is extended over a preternaturally long period of time; the muscles (particularly of hands, arms, and legs) sometimes exhibit a peculiar spasmodic twitching before rigor mortis sets in, so much so that the Cholera Gazette for 1832

* Professor A. Paltauf read a paper before the Association of German Physicians, at Prague, on some experiments made to show the causal connection between rigor mortis and deaths from poison. For the purpose of these experiments such poisons were used as were known to exert a certain influence on the muscular system, either by acting directly on the muscle substance, or indirectly by affecting the nervous system. Amongst the poisons belonging to the first series, curare always considerably *delays* the occurrence of rigor mortis. Amongst those acting on the central nervous system, strychnine, picrotoxin, camphor, and the salts of ammonium and arsenic, *accelerate* the occurrence of rigor mortis. The acceleration is still more increased by artificially prolonging the stimulation of the muscular system, but is again arrested on the occurrence of paralysis. Veratrine and physostigmine cause only a slight acceleration of the rigor mortis, but with caffeine and its chemical derivatives—the rhodan salts—this acceleration becomes considerable. To study the influence of the nervous system at the time of occurrence of the rigor mortis, Paltauf divided the nerves and the spinal cord, with the result that the more a muscle had been stimulated by the poison the sooner was the rigor mortis observed, independently of its connection with the spine, if such connection existed. The reaction of the rigid muscles was in the case of many poisons, as has been generally believed, acid. Other poisons, however (such as camphor, ethyl-theo-

declares the soldiers were accustomed to bind the limbs of their dead comrades to the bed-frames in order to prevent shocking the more timid of the living.* All these manifestations point indubitably

bromine and the rhodan salts), gave, contrary to the general assumption, an alkaline reaction. This alkaline reaction affected, however, only the anterior portion of an animal in which, after the poisoning, the cord had been divided. The posterior part of the animal, in which the rigor mortis was delayed, showed the usual acid reaction until the alkaline reaction of putrescence took place. Where the reaction of the anterior portion of the animal was alkaline it often became, after the reduction of the rigidity had passed off, neutral or slightly acid before putrescence once more made it alkaline. Division of a single nerve had the same result, and it was possible to cause either alkaline or acid reaction of the various muscles of one extremity by respectively leaving the nerve entire or by dividing it. Paltauf also approached the solution of the question of the existence of a cataleptic rigor mortis. He found that the convulsive muscular contractions of an animal poisoned by camphor and suddenly killed by strangling led to immediate rigor mortis, and he therefore believes in a cataleptic rigor mortis.—*Weiner Medicinische Presse.*

Some time previous to this paper, Prof. Paltauf announced he had observed "the greater the hyperæsthesia of the sympathetic, the greater the rigor mortis; but if the sympathetic had only attained a condition of paralysis, the rigor mortis was very greatly delayed."—S.

* Bodies of those who died from cholera, on resurrection after burial were sometimes found turned and twisted in their coffins; and this accrues often to those that have suc-

to poisoned nerve centres, being phenomena especially apt to follow upon specific toxic fatality. And long since T. Lauder Brunton recognized the symptoms of cholera were precisely paralleled by those of muscarine poisoning. Muscarine, though an alkaloidal product of *Agaricus muscarius* (fly agaric), is also a ptomaine, and consequently of animal as well as vegetable origin, and one of the most powerful nerve-poisons known. Its specific action is upon the sympathetic, inducing either paralysis or hyperæsthesia according to the degree of toxicity; it induces, as just remarked, all the phenomena of cholera, even to stoppage of the heart in diastole, and reflex derangement of kidneys with suppression of urine; moreover, its toxic manifestations are exceedingly dilatatory for the same reason.

Another point of moment to be recalled is that the functions of the sympathetic, which belong to the vaso-motor group, are adjuvant to the cerebro-spinal system.

Carpenter appears also to have had a partial insight into the workings of the sympathetic, for he says:† The nerves of this system—"in which tubercumbed to other specific poisons, as the result of decomposition and stimulus of nerve centres by the formation of cadaveric alkaloids that seem to have the power of inducing in dead tissue the same phenomena that result upon galvanic stimulus.—S.

† "Principles of Comparative Physiology." London, 1854.

lar fibres derived from the cerebro-spinal system are combined in various proportions with those gray or organic fibres which have their centres in the proper sympathetic ganglia,—possess a certain degree of power of exciting muscular contraction in the various parts to which they are distributed. Thus by irritating them, contraction may be excited in any part of the alimentary canal from the pharynx to the rectum, according to the trunks that are irritated; in the heart after the ordinary movements have ceased; in the aorta, vena cava, and thoracic duct; in the ductus choledochus, uterus, Fallopian tubes, vas deferens, and vesiculæ seminales; and the very same contractions may be excited by irritating the roots of the spinal nerves from which the sympathetic trunks receive their white fibres; and there is strong reason to believe that the *motor* power of the latter is entirely dependent upon the cerebro-spinal system. That even the *sensory* endowments the sympathetic trunks possess, are probably to be referred to the same connection. The parts exclusively supplied by the sympathetic trunks do not appear to be in the least degree sensible; and no sign of pain is given when the sympathetic trunks themselves are irritated. But under certain diseased conditions of these organs, violent pains, are felt in them; and these pains can only be produced through the medium of fibres communicating with the sensorium through the spinal nerves. . . . There is much reason to believe, however, that it (the

sympathetic system) constitutes the channel through which the passions and emotions of the mind affect the organic functions; and this especially through the power of regulating the calibre of arteries. We have examples of the influence of these states upon the circulation in the palpitation of the heart which is produced by an agitated state of feeling; in the syncope or suspension of the heart's action which sometimes comes from sudden shock; in the act of blushing or turning pale, which consists in the dilatation or contraction of the small arteries (arterioles); in the sudden increase of the salivary, lachrymal, mammary, gastric and intestinal secretions under the influence of particular states of the mind, which increase is probably due to the temporary dilatation of the arteries that supply these organs.* It is probable that the sympathetic system not only brings the organic functions into relation with the animal, but also that it tends to harmonize the former with the latter, so as to bring the various acts of secretion, nutrition, etc., into mutual conformity. The distinctive functions of the gray or organic fibres, and of their ganglionic centres, constituting the proper visceral system . . . not improbably have some direct influence upon the chemical processes which are involved in such changes, and may thus affect the *quantity* of such secretions; whilst the office of the tubular fibres may be rather to

*Also in the enuresis and diarrhoea that supervenes upon fright.—S.

regulate the diameter of the blood-vessels supplying the organ, and thus to determine the *quality* of their products.”

At the risk of prolixity, since the point to be made is of the utmost importance, I may repeat it is essential to the better understanding of the pathology of cholera to note that the phenomena peculiar to the malady, and the symptoms and manifestations relied upon for both ante-mortem and post-mortem diagnosis, are conclusive evidences of its neurotic origin, and of profound toxicity by some agent closely allied to certain alkaloids and acting upon the central nervous system, fatality being hastened by predisposing causes that induce depression of such nerve centres.*

* Toxic alkaloids as the cause of many cases of accidental poisoning have come into prominence of late years, and every summer instances of ptomaine poisoning are reported. An interesting article on this subject appears in the “Boston Medical and Surgical Journal” of August 4th, 1892. Much remains to be discovered relative to the kinds of ptomaines that may develop in both animal and vegetable substances out of the body, as well as of the toxins that may form in food after its ingestion. Doubtless the possibilities of ptomaine formation are very great, and under unusual conditions of insalubrity—hot, damp weather, sewage emanations, etc.,—the work of decomposition may go on with extraordinary rapidity, and, under such influence, tox-albumens of great power may form in food that to the eye and taste is still wholesome. There is accumulative evidence to show that this is so. Cases

—That we do not know the poison specifically, is of little matter; neither do we know whether this poison is developed external to the economy, or induced within the body through miasmatic, bacillar, telluric, meteorologic or physiologic metabolism:

1. Watery diarrhœa and vomiting, both fluids being odorless and colorless:

2. Tetanic convulsions or cramps, followed by muscular flaccidity and lack of cutaneous sensibility and elasticity:

3. Diminished respiration; spasmodic contrac-

of poisoning by food of a relatively innocuous character are not uncommon. Some portion of the ingesta becomes an irritant poison, a fit of indigestion ensues, and often the offending substance is speedily expelled by vomiting. The matter of idiosyncrasy need not detain us here; it is known that certain articles of diet, such as cheese and shell-fish, are toxic to some persons. Generally the state of the alimentary canal at the time is responsible. The most common and best known of the various forms of indigestion is that in which, from the absence or the deficiency of gastric juice and other digestive fluids, the alimentary bolus becomes a gastro-intestinal irritant, provoking vomiting, purging, and a catarrhal condition of the digestive tract. A second stage in the process of acute indigestion arises from the presence of abnormal fermentations and decompositions in the alimentary canal. The food substances break up into organic acids and alkaloidal products of a lower order, which are in part absorbed and produce constitutional disturbances. This stage borders very closely on that of ptomaine formation. There is good reason to regard cholera

tion of circular fibres of bronchi, proximate and ultimate; flagging circulation, with more rapid heart-beat:

4. Depression of temperature below normal—perhaps from five to twenty and more degrees:

5. Suppressed urinary secretion without sufficient pathological manifestation—manifestly due to powerful reflex causes:

nostras and the gastro-intestinal catarrh of infants as kinds of ptomaine poisoning due to multiple causes, of which weakening of the alimentary canal and consequent poverty of digestive fluids, the ingestion of food of an indigestible character, the putrefaction of the latter and the formation of toxins, are the principal factors. That a considerable number of persons may simultaneously be attacked with illness after a large meal, owing to the influence of causes such as have been above mentioned, cannot be a matter of serious doubt. Similar conditions of faulty hygiene produce, in individuals with similar organic susceptibility, results essentially the same. A remarkable case of this kind occurred in America on July 23d of this year. Out of a total of seventy persons who dined together, about fifteen became more or less violently ill within a few hours, exhibiting the symptoms of cholera. The general opinion, as advanced by physicians who had opportunities of studying the facts is, that the sufferers were persons who had eaten rather immoderately of indigestible dishes; that the hot weather, the bad air, and the bad water of the place, co-operated in bringing about the necessary predisposition in the guests, and that therefore the "Salisbury sickness" was a local and limited epidemic of cholera nostras.—*The Lancet* (London), Sept. 17th, 1892.

6. Intense thirst—an evidence of functional disturbance and vicarious nourishment:

7. Immediate disorganization of blood corpuscles, similar in character but slower in degree to that supervening upon serpent poisoning:

8. After death an abnormal persistent increase of temperature, reaching 103° Farh. perhaps:

9. Speedy post-mortem desquamation of epithelium:

10. Post-mortem spasmodic muscular contractions such as follow upon galvanic stimulus:

11. Speedy rigor mortis persisting for an abnormally long period, and in proportion to the hyperæsthesia of the sympathetic:

12. The most virulent symptoms may usually be mitigated by antagonizing the sympathetic:

13. One attack of cholera seems to protect the individual against a second, in greater or less degree, since recurrences of the malady are not frequent:

14. Everything that induces shock (a purely nervous condition), or reduces muscle and nerve vitality—the two being in a measure physiologically synonymous and inter-dependent—increases predisposition:

15. Other epidemics, as influenza, that have a depressing effect upon the nervous system, promote cholera.—All other diseases during cholera epidemics become more aggravated and more fatal :*

*See observations on pages 10, 11 and 127.

16. Severe attacks of cholera are especially apt to follow wine suppers and debauches of all kinds— from depressed or deficient nerve tone:

17. Fear is often a prime factor in promoting the spread of the malady.—Though Lebert thinks this has obtained undue evidence, because “the greater the fear, the more minutely are all preliminary measures carried out.” Manifestly Lebert’s proposition is more true in abstract than in reality.

CHAPTER IX.

PROPHYLAXIS.

The cause of cholera has been shown, then, to be some morbid agent exercising a toxic effect through the central nervous system. The precise nature of the agent is unknown, but its effects are only too apparent; and experience has taught that it attacks the poor in a much larger proportion than the rich, the unclean rather than those practicing sanitation and the laws of hygiene.

It is also well known that in proportion to the prevention of distribution of water fouled with sewage, and to the removal of destitution, filth, foul air, and other great factors of disease, so, generally, is destroyed the agency through which the cholera poison operates.—Thos. Hawkes Tanner, especially remarks the lesson taught by the epidemics of 1853-54 and 1865-66 in England, viz., that even poverty-stricken denizens of an unhealthy neighborhood, supplied with *pure* water, are more certain to escape than the wealthy residents of fashionable parks and squares when the latter consume bad water. Still, that overcrowding is an important factor in disseminating the malady—probably through re-consumption of exhaled gases of respiration, tending to poisoning of the circulation and central nervous system with carbonic oxide—is not to be denied. Within the walls of an

establishment for pauper orphans at Tooting, England, in 1853-54 there were assembled 1,395 children, little more than one hundred cubic feet of breathing space being available for each child, against a requirement of 1,500 (and 500 is the very smallest compatible with health). Here, in a single night, the epidemic seized sixty-four of the inmates, 300 being laid low within a week, during which time 180 died. Again, in the workhouse at Taunton, with 276 occupants, and with breathing space in many of the rooms not exceeding sixty-eight cubic feet for each person, cholera swept away nearly twenty-four per cent. in the brief period of six days; while in the gaol of the same town, where each prisoner had a breathing space of nearly 900 cubic feet (and in some instances more), not a single case of cholera or diarrhœa occurred!

When an epidemic is prevalent, there are also certain conditions other than filthy surroundings, bad water, and foul air, that render individuals liable to the disease, such as unwholesome, indigestible food—stale meat and fish, game that is “high,” butcher-made sausages (since these latter are largely worked up from scraps and unsold remnants at the spoiling turn), withered vegetables, over- or under-ripe fruit, etc. Other predisposing causes are vitiated *damp* air; exposure to all forms of miasm; intemperance; insufficient protection from cold and inclement weather; excessive fatigue; long abstinence from food; diar-

rhœa; in fact all irregular habits, including loss of sleep, excessive venery, *et al.*

Hence the old maxim of an ounce of prevention *vs.* a pound of cure, in this malady receives apt illustration. To secure immunity it is essential to form regular habits of life—to live by rule, in a measure, for sudden radical changes, even if for the better in a general way, are sometimes prone to work an effect quite the reverse of that sought. It is important the residence should be in a clean, dry, airy locality, in a house that has no defects in sanitation, whether from plumbing, from decaying wood, or from water beneath the floors; to avoid the use of purgatives, especially of an acrid or drastic nature—the patent liver-pills, “pleasant” pellets and other nostrums, are extremely pernicious, the larger containing aloes, the smaller aloin, croton oil, or elaterium; to check any manifest laxity of the *prima viæ* by rest in the recumbent posture and employment of plain farinaceous foods; to use for drinking purposes only water that has been boiled, and when cold filtered through a mixture of sand and charcoal—I may state a personal preference for rain water filtered through the Kedzie filter, which I employed for many years.

If there is matter within the intestines tending to irritation, this should be gotten rid of by means of a simple aperient—Hunyadi Janos water, the effervescent draughts, castor oil, or the like; or if there are loose, watery evacuations, modification of the

secretions may be obtained by small and repeated doses of leptandrin, bismuth or cerium oxalate, ipecac, and camphor monobromide, combined; or by chloroform water or spirit; by coto; by chloranodyne; these may be employed either with or without aromatics. The tendency to intestinal flux, even if not choleraic, is necessarily a source of danger in that it must needs be more or less debilitating, thus inducing a condition that predisposes the individual to receive the cholera virus. It is obvious that conservation of the vital powers is of the utmost importance; but that an ordinary diarrhœa, the result of indiscretion in eating, of "taking cold," etc., can, *per se*, produce the specific germ of cholera, as some seem to imagine, is ridiculous to the extreme of absurdity.

But right here let it again be impressed that during a cholera epidemic the precise character of any diarrhœa must necessarily be difficult of determination; it may be truly choleraic, yet never manifest its real nature, and thus become a source of infection. For this reason, on such occasions all stools should be carefully gathered and cremated—no other measure is positively safe,—not emptied into the closet or privy; and these latter receptacles should be carefully disinfected. Again, no traveler or stranger should be permitted to use such closets—not even a neighbor; and no person should ever venture to enter a strange closet, privy, or latrine, since, as has already

been shown, the emanations therefrom may communicate the infection. No device of sanitary expert, or care of plumber, has yet sufficed to render a closet perfectly safe. The practice of wearing a broad, thick web of flannel as a belt, snugly embracing the abdomen, as a prophylactic against intestinal disturbances, far from being reprobated should be encouraged, especially among those of advanced life or sedentary habits.

Clothing soiled by diarrhœaic evacuations, whether the linen of the person or of the bed, should at least be subjected to the prolonged action of intensely hot water—be boiled, in fact,—and also of strong antiseptics, as measures of safety;* and if there is any suspicion of choleraic tendency, it is greatly to be preferred they too go to the fire. With all our boasted knowledge of antiseptics and antiseptics, we are as greatly in the dark as to what is available in destroying the poison of cholera, the element of fire excepted, as we are regarding the precise nature of the virus.

Finally, it may not be generally known that there is no disease in the whole nosological record that is more aided in the onset, and the fatal tendencies of

*I have already shown that Zehnder ascribed the origin of two cholera centres in Zürich in 1867, to an accumulation of bedding, mattresses, pillows, etc., which had been used on the beds of cholera patients, and which before disinfection were piled up in the neighborhood of the houses affected.—S.

which are more vigorously promoted and hastened by mental depression and fear, than cholera—less fear of cholera itself, however, than developed through other sources. The fact is, this malady in the main claims far fewer victims in proportion to the general population of any one country than many others that are regarded with comparative complaisance and often receive scarce passing attention save from medical men and those whose homes are directly invaded. Yearly whole districts are ravaged by typhoid fever, diphtheria, scarlatina, etc., to a greater degree than cholera is apt to do; further, I believe there are no valid reasons, other than the rapidity of the attack and the brief period that may elapse between inception and fatality, for regarding this disease, in fairly sanitary localities, as ultimately more dangerous, taking into consideration all results, than epidemic influenza. Again, it is notable that on every occasion when it assumes its most malignant and epidemic form, and spreads beyond the boundaries of the regions where it is endemic, there have been meteorologic or telluric conditions, or both, favorable to its dissemination; that generally throughout the temperate regions of the globe, as well as in the tropics, a marked tendency to enteric disease prevails*. In the present year (1892), as in 1891, this

* As early as the beginning of 1827 cholera appeared with renewed intensity at Calcutta, and here it is mentioned for the first time that many animals also showed the influ-

latter fact has been most manifest, and never for twenty-five years has the diarrhœaic mortality of London, Paris, Berlin, and other European cities reached greater height than during the past summer.

The present epidemic, as has been shown, readily found its way into Russia, where, by reason of the

ence of the disease (p. 365). . . . It was observed in North Germany at that time (1831) that chickens and pigeons, and in many instances fishes, perished in great numbers. (p. 359).—Ziemssen's "Cyclopædia of Practical Medicine," vol. i.—See Appendix A.

Even in India the development of cholera demands a medium degree of humidity of the soil and air. Great and protracted dryness, as well as excessive long-continued moisture of the soil, are alike unfavorable; therefore it is that in the hot regions of the East Indies, where dryness predominates and rainfalls are scanty, the cholera breaks out, as a rule, in the rainy season; while in the hot regions of lower Bengal, where wet weather predominates and rainfalls are abundant, the malady prevails in spring seasons which lack their usual rain. Great weight is attached to the monsoon season by Indian physicians, who for the most part in past years have been upholders of the miasmatic theory. Von Pettenkofer undoubtedly states the truth when he explains the influence of the monsoon by the saturation of the soil. Again, in Paris in 1849, after a wet spring, cholera reached an unusual degree of fatality in the first eight days of June, which were very warm; on the ninth, however, occurred a violent storm, when the number of new attacks diminished one-third as compared with the days of the preceding week. The same was true in the same year at Vienna, in Austria, and Christiania, in Denmark.—S.

famine and the consequent train of circumstances which make an entire population susceptible to additional scourge, it obtained firm foothold, and thence, in spite of sanitary barriers, reached the more civilized portions of Europe; the same circumstances, precisely, existed in Southern Ireland prior to and during the epidemic of 1834, in which locality the malady was exceptionally fatal until the famine was in great measure relieved, and wholesome food became the rule rather than the exception—thanks to the bounties of the world.

This with reference to the epidemic form of cholera; for it must be remembered that sporadic cholera like the poor, is “always with us” to greater or less extent, and constantly exists in the Levant, in Southern Europe along the Mediterranean, and occasionally appears as an endemic even on the borders of the German Ocean, and in the far interiors of Canada and the United States: The fact these sporadic cases are generally classed as aggravated cholera morbus, cholera nostras, English cholera, cholera infantum, etc., matters little, since they are pathologically one and the same, differing only in degree—an opinion that is upheld by the very highest authorities in India and Europe.* And I here insist, taking every feature

* See “Pathological Researches” of T. R. Lewis; London, 1888. “Nature and Treatment of Sporadic and Epidemic Cholera,” by Alex. Harkin, London, 1885. “On the Origin, Habits, and Diffusion of Cholera,” by Sir J. Fayrer, K. C. S. I., M. D., F. R. S.

into consideration, and giving all negative evidence due weight, that cholera is *cholera* wherever it occurs, and its epidemic prevalence and intensity are phases or accidents in its history. Surgeon-Majors D. D. Cunningham, and J. M. Cunningham, the latter the Health Commissioner of India, and many others who are very properly considered as expert authorities on Asiatic cholera, believe that cholera nostras is the same precise malady, "exhibited under conditions unfavorable for its perfect expression—that at times the conditions may and do become favorable, and then an epidemic results." The disease occurring in the deltas of the Ganges and Irawadi, in Moscow, Hamburg, London, New York or Winnipeg, is practically the same; and within a quarter of a century I have seen numerous cases in the Great Lake region of North America as serious as any that ever bore the name Asiatic, two that would have been pronounced *asphyctic* had the malady only been raging as an epidemic!

[Since the above was written, it has been my lot to see three other cases of cholera nostras (*morbus*) in Detroit, either of which was so virulent there would have been not the least hesitancy on the part of anyone viewing in pronouncing true Asiatic cholera, had there been the slightest chance of infectious origin. I may further add, the bacillus of Koch was specifically identified in the dejections of all three; also, that all were speedily relieved by inhibition of the sympathetic.]

Owing, presumably, to the utter failure of remedies, external or internal, to check the march of this dire disorder, the efforts of medical men for the most part—almost exclusively, I should say—have been directed to measures of prevention; and it is perhaps for this as well as for other reasons, that the therapeutics of cholera has not kept pace with that of other diseases. Sanitary measures have almost wholly superseded sanatory considerations, and to such an extent that in most dissertations of the leading advocates of State Medicine, the former are gravely heralded as panaceas for all epidemic disorders, and *sure*, in course of time, to eliminate from the nosological record the whole train of such accidents. Says Dr. Alex. Harkin:*

“These enthusiasts seem to have adopted for their motto, and emblazoned on their ‘banner with the strange device,’ *Sanitas Sanitatum, et omnia Sanitas*; and yet I fear the saying of the Wise Man, *Vanitas Vanitatum, et omnia Vanitas*, is not altogether obsolete in their regard, but fairly applicable to many of their most confident vaticinations. I am not one unfairly to decry the value of sanitation or scientific hygiene—I should rather prefer that sanitary and therapeutic measures should go hand-in-hand; but in the recent experience of a fatal form of typhoid fever, which numbered many victims in some

* Dublin Journal of Medical Sciences March, 1890.

of the finest cities of America, France, and Ireland, and in the actual presence of epidemic influenza which has prostrated thousands in the British Isles and on the continents of Europe and America, the impotence of sanitary arrangements to alone repel an attack of infectious disorder must be hopelessly apparent.”*

Looking at the history of cholera in Malta antecedent to 1887, we find after the epidemic of 1865, considered the most fatal up to that date on record, every measure that sanitary science, or engineering skill could effect was carried out; yet, notwithstanding, the cholera unheeding swept down upon the Island in 1887 in a more malignant form than ever, for out of 626 cases only 164 recovered—a death rate of 73.5 per cent. In 1865, after which Dr. Sutherland and the eminent engineer Osbert Chadwick visited the Island, the mortality stood at 60 per cent.; while in 1867, an intermediate visitation, there was a mortality of only 64 per cent., and this before the sanitary improvements were complete. In the presence of such adverse statistics it is difficult to agree wholly with sanitary scientists.

* This conviction appears to be entertained by some of the most enlightened organs of public opinion, as I find in an able article on Influenza in the Standard (London) this sentence: “And those who hoped that in the case of this, as of more serious scourges, the comparative excellence of sanitation would secure an absolute immunity, have to confess they have carried their faith in drainage a little too far!”

CHOLERA,

ITS PROTEAN ASPECTS AND ITS
MANAGEMENT.

BY

DR. G. ARCHIE STOCKWELL, F.Z.S.

(Member New Sydenham Society, London.)

IN TWO VOLUMES—VOL. II.

“Respice, aspice, prospice.”



1893.

GEORGE S. DAVIS,
DETROIT, MICH.

Copyrighted by
GEORGE S. DAVIS.
1893.

CHAPTER X.

PAST MANAGEMENT OF CHOLERA.

It certainly is a sad travesty upon therapeutic progress when the members of the profession at large, as voiced by Thos. Hawkes Tanner, and by The Lancet, of London,* is forced to confess "it is folly to talk of curing cholera" since the "very principles which should guide are undetermined;" that "we may be comforted by the better knowledge of the diffusion of the disease, and the better powers of sanitary authorities for resisting it; and the more we study allied diseases, and learn effectually to cope with them, the greater confidence we may feel in contemplating the invasion of a malady *which admits of so little satisfactory treatment,*" etc.; that it is "unknown whether ultimate convalescence depends upon a persistence of the intestinal evacuations, or upon their suppression!"

To be sure, patients recover under all forms and methods of treatment, as is universally allowed; but it must be remembered the period, as well as the character and form of the disease, greatly influences the apparent action of medicines; that he who treats disease by *name* only, is utterly unworthy of rank in the medical profession; that in all epidemics a remedy that seems to be of the utmost benefit in one is of no

* August 20th, 1892.

value in another, and in the same epidemic varies according to continuance; and in cholera, as in all diseases of its class, during its onset and *fastigium* as an epidemic the course is marked by the great percentage of fatalities, while with its wane recoveries are the general rule! So, too, the attacks of the disease tend, in the majority of instances, independent of all remedial measures, to improve at certain seasons, or under certain meteorologic, telluric, or atmospheric conditions, and to retrograde at others.

It is a notorious fact that in more than ninety per cent. of cases the drugs which are ingested, or employed in enema, are quickly returned in the vomit or stool, or, failing this, are mingled with the fluids of the *prima viæ* without absorption until the crisis is over, when they are perhaps more apt to prove mischievous than beneficial. The very condition of stomach and bowels evidences their inability to absorb remedies, and the sole chance is, that some, by localized influence upon the nerve fibrillæ terminating in the gastric viscus, may induce through the nervous system a revulsive action.

Prior to the appearance of the present (1892) epidemic, in England and the United States there were few practitioners who did not believe it a duty to attempt to check the so-called premonitory diarrhœa with astringents and opiates; and reports of thousands of cases might be collected where medical men have believed that thereby they prevented the devel-

opment of the stage of collapse, though it is apparent the theory upon which such practice was based is far from infallible. Further, the whole subject has been so complicated by the publication, both in medical journals and in the general press, of immature hypotheses, extravagant conceits, and infallible receipts, and the views held by different individuals (the majority of whom recognize only the effects and ignore the initial lesion) seeing the same class of cases, even in the same institutions, are so opposite and conflicting, it appears difficult to form a trustworthy opinion. These publications not only frequently serve to show the ignorance, weakness, and credulity of the writers, but likewise tend to bring discredit on the medical profession generally. Physicians, only in each succeeding epidemic learn that the lessons they so carefully taught, and the principles that they so sedulously inculcated during the preceding, are possessed of no real value — in reality have a mischievous tendency.*

*But when the regular symptoms peculiar to the severe form of cholera had set in, medicine, I repeat, had very little influence upon it; and accordingly, as might have been expected, a hundred different cures of the disease were announced, most of them all but impossible. Some persons held that timely bleeding would save the patient; others relied confidently upon mustard emetics. Hot air baths were manufactured, and sold to a great extent, to meet the apprehended attack in that manner without delay. Certain practitioners maintained that the disease was to be remedied by

Nearly every article in the materia medica has been tried and received an ephemeral glory of praise: Large doses of calomel, reaching as high as two drachms, while one individual found a perfect panacea in *two ounces*; lupulin in doses of *six pounds (!)* opium, and opiates, still considered the "sheet anchor" by many, though Dr. Norton, of Cincinnati, expresses the opinion, based upon experience, that by the free use of these cholera is apt to take on a typhoid con-

introducing into the system a large quantity of neutral salts, which were to liquefy and redden the blood, and to restore the functions of the circulation; but of this practice it was said, in a sorry but true jest, that however it might be with pigs or herrings, *salting* a patient in cholera was not always the same thing as *curing* him. In a great number of the sick the blood was mechanically diluted by pouring warm water, or salt and water, into the veins. Some physicians put their trust in brandy, some in opium, some in cajeput oil, which rose to I know not what price in the market; some, again, in calomel alone.

Now I would not willingly mislead or deceive upon this point, by speaking with a confidence which I really have no warrant for, of the success or propriety of any of these expedients. I believe that each in some cases did good, or *seemed* to do so; but I cannot doubt some of them did sometimes also harm. I had not more than six severe cases under my own charge; and I congratulate myself that the mortality among them was not greater than the average mortality. Three died, and three (I will not say they were cured, but) recovered. The three that died I was called in to see when the disorder was at its height; and in each case it went on with frightful rapidity, in spite of all the means adopted, and

dition in its third stage, and Sir Thomas Watson frankly admits many cholera patients have been ushered out of the world thereby; brandy, which while in moderation may act in some measure as a preventive by stimulation of the cardiac and respiratory centres, by prolonged use entails depression and lack of vitality in the same; acetic, muriatic, nitric, sulphuric, fluoric, tannic, gallic, salicylic, carbolic, and lactic acids; oils of cajeput, castor, croton, wintergreen, etc.; creasote; chloroform; sugar; sul-

proved fatal a few hours afterwards. The three that recovered I saw somewhat earlier, but still not till the specific symptoms were present; one was a girl in the hospital. They all recovered under large and repeated doses of calomel. Yet (as I said before) I do not venture to affirm that the calomel cured them. In the first case that was treated in that way, I merely followed up the plan that had been begun by Dr. Latham, who had visited the patients for me when I was accidentally absent. I found that he had felt better, less sick, and less faint, after taking half a drachm of calomel at a dose; and I repeated the same dose for many times, for after every dose his pulse rose somewhat and he appeared to rally. This was the same man whom I mentioned before as having made no urine from the Sunday to the Wednesday; all that time he kept discharging "rice-water" stools. At last, on the fourth day, he passed a *little* water, and his alvine evacuations became rather more consistent, and began to look *green*; and from that time he gradually got well. Afterwards I treated my hospital patient in the same way, and with the same event. Yet I will not pretend to say that these persons might not have done quite as well if they had been left entirely to themselves.

phur; tobacco; lead salts; asafœtida; logwood; musk and musk-root; bryonia; pulsatilla; turpentine; belladonna; atropine; pilocarpine; digitalis; caffeine; cocaine; camphor; coto; cannabis Indica; cantharides; capsicum; castoreum; piperine; creolin; chloral-hydrate; acetanilid; alum; antipyrin; phenacetin; cresol; cinnamon; picrate of ammonia; carbonate of ammonia; strychnine; Ignatia; copper salts; iron sub-sulphate, perchloride, and muriate tincture; salol; cor-

Some of the expedients recommended had certainly a very marked and immediate effect upon the condition of the patients, especially the injection of warm water into the veins. Many instances of this were related at the time. One I myself saw. The patient was a young man, who was nearly moribund, apparently. His pulse had almost if not quite disappeared from the wrist; he was very blue, and his visage was ghastly and cadaverous; in one word, he was in an extreme case of collapse. Out of this he was brought in a few minutes by injecting warm water into one of the veins in the arm. The pulse again became distinct and full; and he sat up, and looked once more like one alive, and spoke in a strong voice. But he soon relapsed; and a repetition of the injection again rallied him, but not so thoroughly; and in the end he sunk irretrievably. Dr. Babington told me of a patient whom he saw speechless and all but dead, and whose veins were injected. He then recovered so as to sit up, and talk, and even joke, with the bystanders; but this amendment did not last either. Yet even this temporary recovery might sometimes be of great importance: might allow a dying man to execute a will, for example.

It was remarked of those who recovered that some got

rosive sublimate; ipecac; naragamine; naphthalin; potassium iodide, bromide, permanganate, sulphate, sulphite, chlorate, bichromate, nitrate; sodium salts; podophyllin; physostigmine; phosphorus and the phosphates; quinine; sulpho-carbolates; arsenic, and arsenical preparations; lime-water; pepsin; silver salts; rhubarb; catechu; kino; matico; bismuth salts; zinc salts; milk, both by stomach and by intravenous injection; coarse salt; ice-bags to spine; hot

well rapidly and at once; while others fell into a state of continued fever, which frequently proved fatal some time after the violent and peculiar symptoms had ceased. Some, after the vomiting and purging and cramps had departed, died comatose; *over drugged* sometimes, it is to be feared, by opium. And the process of artificially replenishing the veins was certainly attended with much danger. The injection of *air* with the water—inflammation of the vein from violence done to it—an over-repletion and distension of the vessels by the liquid—*might*, any one of them, and sometimes I suppose *did*, occasion the death of the patient. Never, certainly, was the artillery of medicine more vigorously plied—never were her troops more meritoriously active. To many patients, no doubt, this busy interference made all the difference between life and death. But if the balance could be fairly struck, and the exact truth ascertained, I question whether we should find that the aggregate mortality from cholera was any way disturbed by our craft. Excepting always the cases in which preliminary diarrhœa was checked, just as many, though not, perhaps, the very same individuals, would, probably, have survived had no medication whatever been practiced.—Sir Thomas Watson, in "*Practice of Physic.*"

salt-bags to spine; oxygen gas; hot baths; cold baths; wet packs, hot and cold; hot air baths; bleeding; cupping; leeches; hot saline enemata; hot-water and saline injections into veins and bowels (hypodermoclysis and enteroclysis); transfusion, etc.

The evidence on which the majority of these remedies is based is irrelevant and in some cases absurd. Directly a case recovers—as the majority do toward the last end of the epidemic,—sometimes even before convalescence is fairly established, the medical attendant imagines the beneficial result (real or assumed) is due to his individual efforts, and therefore immediately sets pen to paper to record his success, at the same time belittling the efforts of others.

Some of these remedies deserve more than passing notice. Mercury, for instance, has been highly lauded, especially by Dr. Josephus Ayre, who showed that of 725 unequivocal cases treated with it 360 recovered. He fails to state the stage of the epidemic when the success was attained, and also to remember that the revulsive effect of the drug is very difficult to secure under the pathological conditions existing in the malady. Moreover, Sir Wm. Gull and Dr. Wm. Baly, in their "Report on Cholera" to the College of Physicians and Surgeons,* show that under opposite methods of treatment, even in the most severe cases, the average percentage of recoveries ranges from 45 to 55 per

*"The Practice of Medicine," by Tanner. London, 1873.

cent., according to the period of the epidemic—consequently the observations of Dr. Ayre are without force.

Dr. O. D. Norton, of Cincinnati, however, corroborates the evidence of practitioners of the last generation, who held that of all the remedies generally recommended, calomel appears to be the most serviceable. He states he was called one evening to see several men, in the same house, attacked with cholera—all “very ill indeed,”—and ordered mustard sinapisms, at the same time leaving a number of powders of mercurous chloride. On visiting them the succeeding morning, it was found all were very much better, when the attendant declared he had mixed together the entire quantity left for each patient and administered as a single dose. In another very severe case attended by Dr. Norton, and where vomiting persisted in spite of the mercurial, the amount was doubled with each administration until eight scruples constituted the final dose. This last, he remarks, “relieved him; he was never salivated, and never suffered any ill effects from it.* He received no more

* Dr. Norton does not seem to be aware that the dangers of salivation are diminished in proportion to the size of the dose; that while five grains may procure ptyalism, such in the same patient would be impossible in a dose of two scruples, without the intervention of a powerful acid. Nevertheless, the good effect attributed to the calomel, is more likely, considering the history of the drug, to have been a mere coincidence.—S.

medicine, but suitable nourishment, and in three days was up and around."

Castor oil in frequent doses was strongly advised by Dr. George Johnson,* and has recently been advocated by a number of practitioners in the United States as "almost infallible;" but the Medical Council of the Board of Health of Great Britain, after investigating several cases so treated, observed: "The details have been carefully investigated by the Committee, and it appears that in eighty-nine cases of cholera treated by fourteen different practitioners with castor oil, on the plan recommended by Dr. Johnson, sixty-eight were fatal—recovery occurred in only fifteen instances, while six cases still remain under treatment."

Dr. George Steel* details a case in which he first bled the patient in the arm until six ounces were taken; next twenty grains of tobacco were infused in five pints of hot water and employed as an enema, followed by the application of caustic potash over the spine from one extremity to the other. The patient became progressively worse, and when moribund was subjected to the influence of galvanism. "An incision was made over the glosso-pharyngeal nerve, where one wire was inserted, while the other was applied to the epigastrium; he was kept under the galvanic stimulus for three hours. A powerful effect

* Medical Times and Gazette, Sept., 1854.

† The Lancet (of London), vol. ii, 1866.

was produced on the respiratory function. The air expired grew warmer, and his lips and whole countenance, which had been previously livid, became of their natural color. He died about an hour after the galvanism was discontinued."

Mr. John Gason says:* "The abdomen should be tightly swathed with a broad flannel binder sprinkled with chloroform and the patient strictly confined to the horizontal posture [a by no means bad idea.—S.]. As soon as the 'rice-water' evacuations have commenced, and true fæcal evacuations ceased, a tightly rolled-up towel, in length about eight or nine inches, and in circumference about three inches, and moistened with an antiseptic, should be placed length-wise between the buttocks so that the orifice of the rectum may be about midway of the roller. No evacuations of the bowels should be permitted, which will be completely obstructed by the towel."

Dr. George Rogers of Bristol advises "Calomel twenty grains; ginger, five grains; opium, one-eighth grain;" taken in powder every fifteen minutes "until warm perspiration is produced;" and in addition "would suggest—as a diffusible stimulant, diuretic, and astringent—spirits of turpentine in doses of from one to three drachms frequently administered. Having found turpentine *most useful in arresting uterine*

* The Lancet (of London), vol. ii, 1866.

hæmorrhage, and diffusing an instantaneous warmth through the system, I cannot believe but its action would be most beneficial in cholera."*

The formula of Dr. Stone of New Orleans, which was very popular in the South and Southwest, from 1845 to 1856, is much the same as that of Dr. Rogers. It is calomel ten grains, camphor four grains, capsicum one grain, opium one-half grain, with charcoal, chalk, and sugar-of-milk.

Commenting on the foregoing and other equally absurd procedures, Dr. Tanner caustically remarks: "How would the reader desire to be treated should he unfortunately suffer from an attack? Is it probable that he, as a medical man, will wish to take from one to one hundred and twenty grains of calomel every fifteen minutes, or large doses of strychnine repeatedly, or half an ounce of castor oil every half-hour or so until between ten and eighteen ounces have been swallowed; or will he be bled, or allow leeches to be applied around his anus, or have blood transfused into his veins? Will he think it well to be narcotized, or stimulated, or cauterized, or galvanized, or corked up by Mr. Gason, or left to the delicate medication of Mr. George Rogers; or will he, as many have wisely done before, become restive and decline to be tormented and to have his stomach converted into a filthy drug shop?"

* *Ibid.*

“Should he choose the latter course, it may comfort him to remember that, according to Mr. [Jos. Scott] Bowerbank, when the patients in the prisons and hospitals of Jamaica refused to take the medicine prescribed, they were placed upon a mattress on the floor, with a bucket of iced water and a mug by their side. For the most part, we are told, they received little if any further care; while certainly they were not covered with blankets and rubbed, as the more tractable sufferers were. Nevertheless, the majority of those left to their own resources got well. So, also, Dr. E. A. Parkes tells us that in India the Asiatics were seldom admitted into hospitals until the disease was well marked, as they were either incredulous of the power of medicine, or resigned to the decrees of an inexorable fate; and that he never saw one of them bled. ‘Yet the mortality certainly was not greater than among Europeans’ [*i. e.*, in hospital—S.]. And finally, it is well known that during the epidemics of 1849 and 1864 very many of those attacked passed safely through the stage of collapse and secondary fever without any treatment; while in a number of other instances the same success attended the use of ice and beef tea only.”

A plan of treatment thought by Dr. Tanner deserving of notice, “is that by salines as suggested by Dr. Stevens, in spite of the admission that ‘on extended

* Edinburgh Medical Journal, September, 1866.

trial the failures have been numerous, though by no means so great as with astringents and various preposterous practices.'” The following is the outline as employed in the prison of Coldbath Fields:

“Patients presenting the premonitory symptoms of diarrhoea and vomiting were removed into an observation ward, where an even temperature was maintained. A Seidlitz powder was immediately administered; if sinking was felt without purging, three or four teaspoonsful of Epsom salts were added to the powder. On these agents acting, plenty of thin beef-tea, well seasoned with salt, was given; if there was any pain, a sinapism was applied to the gastric region; and thirst was relieved with seltzer, soda, or pure water, *ad libitum*.”

“If, however, cramps, coldness, or sinking of the pulse came on, the patients were considered as cholera cases in the second degree. The following was then administered, about every half-hour, dissolved in water:

℞	Sodic chloride.....	gr. xx.
	Sodic carbonate.....	gr. xxx.
	Potassic chlorate.....	gr. vij.

“If there was much irritability of stomach, a large sinapism was applied; if much heat or burning pain, an additional quantity of carbonate of soda added to the mixture.

“In cases in state of collapse, a strong solution of the same salts, dissolved in hot water (100°

Farh.), was thrown into the bowels, and the enema repeated every two or three hours. Sinapisms were also applied to the stomach, between the shoulders, etc.; and in the cold stage, frictions with warm towels were used. A pure air for the patient to breathe was considered of the greatest importance.”*

With regard to the foregoing, now being generally lauded as entirely new and novel, it may be remarked it did not originate with Stevens, but is almost as old as cholera itself. It was practiced by the native physicians of the Orient long before the European gained foothold in that part of the world; it was advocated by one Dr. Wm. Marsden, of London, as far back as 1832, and the same gentleman published a treatise thereon which, manifestly, is the basis of later claims.† The fluid recommended by him was practically the same as that of William Stevens, consisting of a “solution of sodium chloride, sodium carbonate, and potassium oxymuriate (chlorate), forty-eight ounces of which, at a temperature of 100° Farh. were slowly injected into the veins of the arms,” while every fifteen minutes the same was given by both mouth and rectum. The late Dr. B. B. Brown, of Sacramento, California, employed

* “On Asiatic Cholera,” by William Stevens, London, 1863.

† “Symptoms and Treatment of Malignant Diarrhœa, better known by the name of Asiatic or Malignant Cholera.” London, 1834.

Marsden's fluid during the epidemic of 1849, with some success, but was honest enough to acknowledge the source of his remedy. Dr. Rumpf, too, the head of the Eppendorf Hospital, during the early part of the present epidemic in Hamburg, employed for a time saline injections of the strength of one-half of one per cent., but ultimately gave them over in spite of the fact they appeared of temporary benefit; it was observed by him, the same as by Sir Thomas Watson, that the majority of those receiving such treatment, *ultimately died!*

Indeed, in Hamburg, medicine is rarely given, Dr. Reider, assistant at the Allgemeine Krankenhaus, states intra-venous injections of salines appeared "merely of temporary value, and then only in *very few* instances." Tannin enemata, so highly praised "proved wholly ineffective and useless: as did also *camphor, ether, and salol*, [recently greatly lauded on supposed theoretical grounds.—S.] by the mouth, and the two former subcutaneously." The patients, however, were allowed as much tea and coffee *sans* sugar, and seltzer water, as they desired, some taking as much as six pints of the latter daily; a little pounded ice was occasionally given; hot bottles also employed." Again, he remarks, "if the condition became progressively worse, and the circulation drained of fluid, one or two litres of salt and water were injected into the veins of the arm, and if necessary, later on, into the other arm or the leg.

Sometimes the patients winced at the incision, as at the prick of the hypodermatic syringe, but generally they were so lethargic they appeared not to feel it. "This salt and water injection was given only in extreme cases, and with the exception of one girl, *we did not see any patient live long after it.*"*

Dr. Lauenstein, of Berlin, endeavored to ascertain what treatment would give the best results by devoting one barrack at a time to some one of the many plans advocated; his report is summarized as follows: †

1. *Expectant and Symptomatic.*—This included the administration of wine, analeptics, anodynes, warm stupes, hot baths, friction, hot coffee and tea, with enema of tannin (one to two quarts given, so as to pass up high into the intestine). On the whole he is disposed to think the best results were had from this method.

2. *Hypodermatic Injections of Morphine.*—He gave fair trial to this method, loudly advocated by an American doctor from Memphis. "It was tried in thirty cases, and *did no good whatever.* This consisted of warm subcutaneous injections of: ℞ Acid sulph. dil., 1.00 gramme; morph. sulph., 0.01 gramme; aq., 45.00 grammes; the whole injected at once at blood temperature. In five cases there occurred deep-

* Italics mine.—S.

† British Medical Journal, Oct. 8th, 1892.

seated abscess with gangrenous destruction of the subcutaneous tissues.”

3. *Creolin*.—He also gave trial to the creolin treatment, so strongly recommended by Ferd. Hueppe, of Greifswald. “It was administered by the mouth—a two and a half per cent. solution in water, a couple of dessert-spoonful every two hours,—and also *per rectum* as a high injection; but only one per cent. strength was used for the latter purpose. This also was found of no value. It was most objectionable to the patients, and constantly caused intolerable itching and burning.” Dr. Lauenstein briefly characterized as a “horse cure” (*Pferde-Kur*).

4. *Tannin and Lactic Acid*.—Lastly he used very largely the method introduced and strongly recommended by Professor Cantani, of Naples, during the epidemic of 1884, consisting of copious high intestinal injections of warm one per cent. solution of tannin, and lactic acid by the mouth against vomiting; the lactic acid administered in a solution of thirty parts to five hundred of water. “No good whatever was obtained by the use of the lactic acid, and it was greatly objected to by the patients. The tannin injections, sometimes, seemed to do good, and the patients generally felt better after them, when the colicky pains which often ensued had passed off. That the tannin in no way acts as a disinfectant, is clear from the existence of active bacilli in the bowels of a convalescent patient for whom they had been freely used.”

5. *Intravenous and Subcutaneous Injections.*—For both purposes, a solution of six-tenths per cent. of common salt, rendered slightly alkaline by bicarbonate of sodium, in water sterilized by boiling, was used. “The quantities given subcutaneously varied from one, to one and a half litres (about two to three pints), and intravenously even as much as two litres (somewhat more than two quarts); the transfusion, in both cases, made by gravitation only. The effect was sometimes astounding; the cyanotic, algid, pulseless, breathless creature, with shrunken wrinkled features, sunken lack-lustre eyes, and the whole aspect of a moribund, would, as if from a deep sleep, arouse himself, and tell the delighted doctor he felt vastly better; but *unfortunatety the results were not always lasting.*”

It may not be amiss in this connection to reproduce the utterances of The Lancet, of London, on factitious death in cholera, and the use of salines for resuscitation:

“There are special causes for the uneasiness, not to say positive panic, which the slightest hint of the advance of cholera arouses in the populations of Southern Europe, particularly among the Mediterranean shores. Their custom of allowing but a few hours' interval, sometimes hardly a day, to elapse between decease and burial, has on the occasion of increased mortality from epidemics induced certificates of death to be prematurely given, with the hor-

rible result that apparent corpses have come to life on their way to the tomb, or the crematorium, and with the necessary inference that not a few must have been buried or cremated when the vital spark, so far from being extinct, might still have been kept alive. The great Tuscan pathologist, Filippo Pacini, some thirty years ago, published a memorable pamphlet on 'La Morte Apparente della Colèra' and having instanced a number of cases in which the seeming corpse had been snatched from the very brink of the grave, he proceeded to give rules by which even in collapse the apparent victim to cholera might be resuscitated.

“Among his prescriptions, that of the intravenous injection of bay salt, as suggested and practiced in 1832 by Dr. James Mac Intosh of Edinburgh, held a prominent place, and by this means, particularly in the cholera visitations of 1877 and 1884, the restoration to life of many duly certified as dead was just in the nick of time effected. In the latter year, however, a remarkable and extremely painful instance of the all-too-tardy resort to the practice occurred in the person of a distinguished Genoese physician, who having overworked himself in the public cause during the epidemic, was himself stricken down just when it was in full retreat. Every care was bestowed upon him, but he rapidly sank into collapse, and within forty-eight hours was thought to be, and certified as, dead. Burial arrangements were in progress, and the family, sitting disconsolate in a room adjoining that in which

he was laid in his shroud for burial, were expecting the arrival of the undertaker every moment, when, to their mingled horror and delight, the door was feebly opened, the apparent corpse presented himself and, in a voice scarcely audible from weakness, remonstrated with them for having left him so long unattended. Instantly they conveyed him back to bed, and employed, under the best professional advice available, every means that could be imagined for his rescue; but in vain. He died a few hours after of cardiac failure.

“ This case was much commented on at the time as a typical example of what might occur on the strength of death certificates prematurely given, and so Pacini’s method was reapplied with enhanced vigilance on every cholera patient who had reached the stage of collapse. The epidemic ran its course; the panic and the vigilance born of it disappeared, till now, when cholera is again within measurable distance of the Mediterranean seaboard, Pacini’s name and practice are once more trotted out. The Southern populations, however, must surely have learned by this time that prevention is a better safeguard than cure, be it as ingenious as it may, and that to leave their ports in a mediæval state of filth and neglect, invites those cholera explosions that never occur on so sudden and so vast a scale in the more civilized harbor towns of the British Isles or of the Dutch and Danish coasts. With the experience of Naples and

Spezzia still recent, sanitary rehabilitation must have made some way toward the protection of regions to which quarantine and hygienic cordons have ceased to give the security so long laid to their credit."

Lieutenant Colonel and Surgeon R. Lewins, commenting on the above, in a letter to *The Lancet*, says:

"The Dr. James Mac Intosh alluded to is doubtless Dr. John Mackintosh, for many years extra-academical lecturer to the Argyle-square Medical School. On consulting his work on the 'Practice of Physic,' it will be seen that he gives the credit of originating this heroic treatment of a disease that 'begins with death' (as he was accustomed in his lectures to term cholera) to Drs. Latta and Lewins, of Leith, in which seaport the epidemic of 1832 was particularly malignant. The latter named physician was my father, and I have often heard him speak of the improvement effected by the warm salt-water injection as *only temporary, and in no single case resulting in recovery*, as apparently claimed for Dr. Pacini, practically, in *The Lancet*."

CHAPTER XI.

GENERAL MANAGEMENT DURING EPIDEMICS.

There is, perhaps, no malady so fatal to the overweening self-confidence and egoism of the medical man as cholera, especially in its well-marked asphyctic stage. During an epidemic even the most careful prophylactic treatment may fail, and it may be impossible to fulfill the indications from the cause or from the disease. The immunity of individuals is generally in proportion to vital resistance and the hygienic surroundings that tend towards good health, and it is an error to imagine vital resistance is in proportion to muscular energy; hence it is not astonishing that very vigorous persons are frequently stricken by the malady, while others possessed of less physical powers escape. Besides the more or less complete immunity inherent to individuals, there is likewise a temporary immunity which is left after the epidemic has in a great measure exhausted itself. In almost every plague of this character, especially after its fastigium has been reached and passed, and the number of fatalities are in greatly less proportion than the recoveries, certain specifics are wont to be recommended by medical men of all degrees—by the reputable practitioner no less than the charlatan; yet the reputation of such specifics has always been so ephemeral as to never persist beyond the first week of the next succeeding epidemic. Those last heralded as *new* and based upon the assumption the intestines are the seat

of the lesion, such as salol, copper arsenite, tannin, muriatic and sulphuric acids, hot water and saline injections (enteroclysis and hypodermoclysis) with the exception of the two first, do not even possess the merit of novelty; they have been repeatedly tried and found wanting; in the meantime the older panaceas, such as opium, camphor, calomel, ether, cajeput oil, etc., are returned to (for lack of better knowledge, and in the vain hope they *may* prove of some little benefit), only to be thrown aside in despair.

It is practically useless to look for any form of therapeusis that demands the administration of drugs by either mouth or rectum, since the mucous membrane of the *prima viæ* is unable to absorb in the slightest degree if the attack is in any way serious, and even in the milder ravages of the disease is incompetent to perform its function save in a relative and most imperfect manner. From the earliest appearance of cholera in Europe to the present time, a large proportion of practitioners have depended solely upon some mixture containing opium or camphor, and teachers sedulously recommend the same for lack of better knowledge, typical remedies of this class being Squibb's cholera mixture, the so-called Russian cholera drops, etc.*

Again, aside from the fact the mucous membrane

*℞ Tinct. valerian æth., 2 drachms; wine of ipecac, 1 drachm; laudanum, 1 scruple; oil of peppermint, 5 minims.
—S.

is incapable of absorbing, it could hardly be expected that any remedy could develop its action in the presence of a large quantity of "rice-water" fluid. Moreover, it has repeatedly been proven that active interference is more injurious than a simple mode of treatment, and it may be added this is almost even more true of the third stage, or period of reaction, than of the attack itself.

From the evidence just presented, and that moreover embodied in the preceding chapter, the practitioner will, I am sure, realize the inutility of extravagant methods of therapeutics, and consequently scout all measures that tend to worry the sufferer with nauseous remedies.

That there is no such thing as an antidote—in the full acceptation of the word—to cholera, remains incontestable; yet this is no reason why the practitioner should idly fold his hands under the supposition he can be of no service, or that the chances are equally as great without as with his intervention. This is not necessarily true, for it must be remembered there are a large number of cases of severe diarrhœa, and likewise cholérine, that if deprived of proper care will be merged into cholera; such fact merits strict attention. Neither is cholera proper, the form that asserts itself without premonition or warning, always beyond reach if physiological phenomena are considered, and met strictly in accordance with their manifestations by logical means of therapeusis.

During a cholera epidemic, the supervention of any form of malaise, especially if accompanied with looseness of the bowels, should be regarded with suspicion, particularly where the habits of life of the patient are known to be irregular, "loose," or "fast." It may be only a passing evidence of indigestion due to indiscretions in appetite, yet it is best always to be on the safe side; even an indiscretion *may* become the focus through which the cholera poison finds opportunity to assert itself; for there is, after all, more truth than speculation in the common saying that in the time of a cholera epidemic "the disease is in the air." I have already shown that telluric and meteorologic influences have a decided bearing on predisposition, dissemination, and recovery; it is also well known that in cholera times even a minor surgical operation may induce or favor the malady through shock, no matter how limited, to the nervous system. Lebert remarks when cholera seizes women in the puerperal bed, abortion takes place, and the disease is usually fatal. "I had in my own practice," he adds, "a sad case of this kind in which, notwithstanding the most energetic treatment of the prodromal diarrhœa, the disease continued on uninterruptedly to the fatal end." He likewise observed in Velpeau's Clinic, in Paris, in 1849, that the most trivial operations, such as lancing an abscess, extirpation of a sebaceous cyst, puncture of a hydrocele, etc., led to cholera.

First of all, when an attack of cholera, cholericine,

or even diarrhœa supervenes, as a measure of safety the patient should be isolated so far as possible, and in a way to secure the greatest amount of pure fresh air; at the same time an even temperature must be maintained in the apartment.

Second, great care is to be taken that his drinking water is pure, particularly that it has not stood in a receptacle for any length of time, or been drawn from a well near any sewer, or any river receiving filth and drainage; it may in any event, with advantage be boiled, cooled, and iced. Or he may drink freely of aërated waters, iced coffee or tea, etc.—warm drinks are objectionable, especially hot tea, as it tends to favor nausea. If no carbonated water of good quality is available, effervescent powders may be employed, made from four parts of bicarbonate of soda and three parts tartaric or citric acid, administered from time to time in a few teaspoonsful of water; another agreeable and refreshing beverage is made by adding to a moderately sweetened solution of potassium bicarbonate and sodium bicarbonate, a sufficient measure of newly expressed lemon or lime juice.—These mixtures all, of course, to be taken at the moment of effervescence, else their value is in considerable measure inhibited. The carbonic acid gas introduced into the gastric viscus seems to serve the purpose, not alone of relieving thirst which is very intense in real cholera (especially after diarrhœa has persisted some time, evidencing that

this symptom is a manifestation of the economy at large protesting against the extensive withdrawal of fluids), but of stimulating the sympathetic system through the endings of the nerve fibrills in the stomach, whereby nausea is allayed, and transudation of serum through the intestinal walls in some measure inhibited. It is often advisable to permit pieces of ice, varying in size from a pea to a filbert, to be swallowed at intervals of every three or five minutes, and between the ingestions of carbonated water.

If the attack presents no manifestation other than diarrhœa, as a measure of safety the patient should remain supine in bed, partaking of cooling drinks, and be carefully nursed; everything should be done to ensure composure of mind. Subcutaneous injections of small doses of morphine muriate may be given to relieve pain; but the manifestations are usually best met by some sedative mixture, such as chlor-anodyne, administered perhaps upon loaf sugar. Chlor-anodyne contains, beside morphine muriate, chloroform and hydrocyanic acid—the latter the most effective remedy, by the stomach, that has yet been found in combating true cholera,—capsicum and cannabis Indica; hypodermatic injections of morphine at most are but mere palliatives, and in cholera are especially prone to induce abscesses, deep-seated suppurations, and sometimes *gangrene!* * If deemed

* The muriate salt is less likely, in this disease, to provoke accidents.—S.

essential, sinapisms or poultices may be applied to the abdomen and held in position by a broad flannel bandage snugly applied, and reaching from the illia to the xiphoid cartilage; better, by far, than these is half a dozen thickness of flannel, saturated with a mixture of chloroform and alcohol, one part of the former to twelve of the latter, topped by rubber cloth or oiled silk to prevent too rapid evaporation, confined to the body by the bandage.

The diet should be light, nutritious, properly cooled, and largely of farinaceous character. The stools should invariably be cremated, since, as has already been shown, a diarrhœa of very moderate character may carry the poison, that by fermentation in suitable media, and reduplication, becomes a serious menace to the health of others. Says Prof. Lebert:

“When the cholera poison has once reached the small intestine through the air or through fluids, the development of its action within the economy depends partly upon the quantity in which it has been introduced, and partly upon the favorable and unfavorable conditions that may be encountered. In some individuals, it passes through without leaving a trace. In others the effect is exhausted in an intestinal irritation [reflex of the central nervous system.—S.] of one or several days duration, manifested in a diarrhœa. In not a few the poison rapidly increases, when occurs either a grave attack of cholera, without prodromic

diarrhœa, or the diarrhœa sets in and develops into regular cholera in a few hours, or one or two days. In still other cases again, there is neither a cholera diarrhœa, nor a cholera attack with its colorless stools, but only a moderately violent attack of vomiting and diarrhœa, or cholérine, which scarcely differs from a cholera nostras. But all these forms belong together; each may pass into the others, and each may show besides the most favorable symptoms, individual manifestations of the most pronounced attack.”*

* “Vortage über die Cholera,” Erlangen, 1864.

CHAPTER XII.

MANAGEMENT OF PRONOUNCED CHOLERA.

So far, latterly, I have dealt more particularly with threatened cholera, or the more simple and uncertain manifestations of the malady. I will now consider the more serious form premising, as heretofore stated, that the term Cholera as generally understood, is applicable only in a generic (instead of specific) sense; that properly it covers the diseases ordinarily differentiated as Asiatic or epidemic cholera, sporadic cholera, cholera morbus (cholera nostras, English cholera), cholérine, cholera infantum, cholera diarrhœa, and, likewise, certain forms of "summer" and "winter" diarrhœa; that these affections are one in character, and although presenting different degrees, bear the same ætiological relations and are dominated by the same pathological principle, consequently, to a greater or less degree, must be amenable to like methods of management. Moreover, a large number of eminent authorities, Indian and European, have given expression to similar opinions, notably Orton, who (cited by Aitkin) maintained as early as 1832 that the disease presented "as many diversities of aspect and symptoms as scarlet fever, for between serious cases and those of only ordinary intensity a disparity fully as considerable is to be remarked." Also, Dr. Scriven, in a communication to

the Epidemiological Society of London, admits sporadic and epidemic cholera are of precisely the same nature, "just as sporadic variola resembles its epidemic form." Likewise, Dr. Guérin, before the Academy of Medicine of France, recently asserted the "distinction made between the different forms is purely arbitrary," and that "this affection exhibits special variation in intensity,"—thus corroborating Dr. Dutrieux Bey (of Alexandria) and Surgeons J. M. and D. D. Cunningham, T. R. Lewis, Sir J. J. Frayer, Max von Pettenkofer, and others. Indeed, Pettenkofer insists that the dejections of persons suffering from so-called cholera morbus or cholera nostras, or from cholerae or its lesser form cholera diarrhoea, under suitable surroundings, are as capable as those of epidemic cholera of provoking the most extreme form of the infliction.

In the management of these diseases, then, it is necessary to bear in mind their relationship as well as their individual and collective factors, and at the same time to establish some pathological data as a point of departure. This latter, in a measure, I have attempted to delineate elsewhere, pointing out the unmistakable evidence of poisoning of the central nervous system and the influence thereby manifested upon and through the great sympathetic. It is likewise essential to remember (as elsewhere stated), that this toxicity may already be complete to the point of fatality ere its first manifestations are revealed externally, or

brought within the range of diagnostic perceptivity; and for these reasons cholera, in its most extreme form, must ever remain a fatal scourge, baffling to those who practice the medical art.

At the risk of prolixity it may be well to refer again to the evidences of the pathology. Note then :

The intimate relation of the seat of all local pathology to the great sympathetic:

The close resemblance of cholera in certain forms to certain manifestations of tabes; likewise to the toxicity induced by poisons acting directly upon the central or general nervous system,* and that death supervenes through nervous exhaustion, the usual manifestations of this form of toxication as encountered in the intestine being exhibited in greater or less degree (even to absence) in proportion to the degree of poisoning:

The entire lack oftentimes of local pathology, particularly in the more aggravated and malignant forms of the malady:

The evidence of paralysis induced in those portions of the body which are governed by the great sympathetic, as by desquamation of intestinal epithelium, etc.; also the transudation of serum indicat-

* It must be remembered Virchow long since proved that arsenic introduced directly into the circulation produces all its peculiar phenomena, and much more readily than when introduced by the stomach.—S.

ing paralysis or hyperæsthesia of certain portions of the sympathetic *per se*:

The reflex suppression of urine, and changes that frequently take place in the kidneys, yet never result in chronicity:

The eyes sunken in their orbits, and insensibility of conjunctiva and cornea, indicating sympathetic inhibition in the cervical area:

Manifest evidences of nerve degeneration in all the different tissues and organs; the (according to Doyère) diminished exhalation of carbonic acid gas—for reasons given in a previous chapter; the rise of temperature after death coupled with the fact dead bodies cool off but slowly—Lebert unhesitatingly declares the sensorium is early affected:

The peculiar sopor; the manifest anæsthesia, hyperæsthesia and paralysis, affecting not only the surface of the body but the vital organs themselves; the manifestations afforded by the tongue; the peculiar bitter, nasty, pasty taste within the mouth:

Susceptibility to shock—the most trivial surgical operations, performed upon those seized even in the slightest degree with the malady, induce most serious and usually fatal consequences:

Death with all the manifestations of uræmic toxication:

The indican found in the urine, which is ever an evidence of profound nerve exhaustion; the lack of tonicity of certain nerve-plexuses such as the cœliac,

the solar, the uterine; the readiness with which abscesses, furuncles, etc., supervene; the total inhibition of secretion in the third stage—this, of course, as the result of transudation, since all secretion is blood-depuration:

The depression of temperature below normal:

The watery diarrhœa and vomiting, both fluids being colorless and odorless—“Uncontrollable watery diarrhœa is an outcome of terror and nerve perturbation” (Fothergill):

Flagging circulation with more rapid heart-beat; death with the heart in diastole—a peculiarity which obtains to toxicity from serpent-venom; spasm of arterioles and contractile circular muscular fibres (even of bronchi, proximate and ultimate)—this in connection with the transudation of serum, fully accounts for the terrible cramps:

Finally, the well known fact that the pathology of the disease as exhibited, is in no way commensurate to the character of its manifestations either in its minimum or maximum; the readiness with which the malady is superinduced upon any act or phenomenon that provokes or tends towards depression of the nervous system (wine suppers, excessive venery, etc.); the mental relations; the slow recovery, often dependent upon an intermediate typhoid stage.—And there is every evidence of extremely low nervous vitality which cannot be relegated to the local lesion, neither is the latter capable in any degree of account-

ing for the transudation of serous matters from the circulation!

Thus the influence of the nervous system is made most manifest in subjective and objective symptoms. The vomitings and numerous stools result from either a paralytic or a hyperæsthetic condition of the sympathetic—conditions that, apparently antagonistic, as before shown, are physiologically the same, differing only in degree. The crises, cramps, vertigo, anxiety, aphonia, spasms, tremors, all betray their neural origin; the evidences of collapse, and the algidity, are dependent upon the hyperæsthetic (or irritative) and hypertrophic condition of the sympathetic system; while to the vaso-motor nerves may be attributed that depression of the function of respiration and circulation which constitutes the most dangerous symptom of the malady. Again, the entire series of symptoms are precisely paralleled in the toxic manifestations of certain cadaveric alkaloids, one of which, (muscarine), as before remarked, is had both as an animal and vegetable alkaloid, and is likewise one of the most powerful nerve poisons known.

The practical application of admitted physiological and pathological principles, and the discovery of constant relations of cause and effect, suggest the idea of a well-defined law in this affection. And, moreover, since it is to the sympathetic system that must be referred the depression of the functions of respiration and circulation, therefore in antagonism

of this system is afforded the key to rational and physiological treatment.

The fact may be recalled that the sympathetic pertains to the vaso-motor system, and that the inhibitory nerve in most complete relation thereto is the pneumogastric, which unites with the sympathetic to form the cardiac, solar and hypogastric plexuses; that the pneumogastric (vagus) possesses an action antagonistic to that of the sympathetic on the heart. The vagus and sympathetic are also in intimate connection in certain secondary plexuses, as the aortic, spermatic, renal etc.

Again, the parts chiefly supplied with sympathetic nerves are usually capable of none but involuntary movements, and when the cerebrum acts upon them at all, it is only through strong excitement, the depressing influence of some passion, or through some voluntary movement with which the actions of the involuntary part are commonly associated. Also, it should be remembered the solar plexus lies directly behind the stomach, and is the one to which must particularly be referred the peculiar derangements that occur in the splanchnic area as the result of the cholera poison.

Thanks then to modern physiological research and clinical observation, we know that the pneumogastric or vagus, which is a cerebro-spinal nerve, is an antagonistic and controlling agent. As already shown, to the vaso-motors must be referred the de-

pression of the functions of respiration and circulation, which constitute the gravest symptoms of cholera, proof of which is found in the rapid deaths supervening upon *cholera sicca*, or "dry cholera," which have been observed during certain epidemics, and on many occasions overtakes individuals apparently in full tide of health. Again, the fatal result in a large proportion of cases of asphyctic cholera, is usually preventable by the use of hydrocyanic acid or the dilute virus of *antiaris toxifera*. The prompt relief which may be afforded in such cases if seen at the very onset of the asphyctic manifestations—cases apparently the most desperate,—conflicts with the opinion that cholera is a serious organic affection.

By stimulating the sympathetic part of the heart its contractions are augmented; but by acting thus on the vagus it is possible to arrest the heart in full diastole. The stimulation of the vagus then is an important indication; that is, the re-establishment of the cardio-inhibitory functions of this nerve, which are evidently absent in cholera. By so doing the violent contractions and palpitations cease, and the energies of the heart cavities (especially those of the left side), are restored; the congestions of the pulmonary and cutaneous system likewise disappear.

In all attacks of cholera, then, regardless of the stage, the first indications are to stimulate the vaso-inhibitory apparatus and antagonize the sympathetic

(especially in its cardiac and solar plexuses) by sedation, by reflex, or by both.

The success which is sometimes obtained in the lesser degrees of the malady from internal administration of camphor, chloroform, ether, etc., is due entirely to their action upon the sympathetic system; but such are available only when absorption is in greater or less degree possible. The remedies most valuable for sedation of sympathetic are in order: Hydrocyanic acid, antiaris toxifera, chloroform, cocaine, cannabis Indica. As before noted we have in chlor-anodyne a preparation which contains certain of these remedies in combination (notably, the hydrocyanic acid, chloroform and cannabis Indica) in connection with morphine and capsicum. This is a preparation that is not alone an improvement upon the old proprietary chlorodyne, but by the dropping of certain inert and nauseous ingredients, is far less antagonistic to sensitive or irritated stomachs. The action of this fluid is in a measure dual, in that it allays the irritation of the sympathetic induced by the cholera poison, and at the same time stimulates the vaso-inhibitory apparatus, thereby relieving the spasmodic congestion of the arterioles that leads to oppression and depression of both cardiac and pulmonary functions. It is still further valuable in that it is intensified by the powerful revulsive effect of the contained capsicum.—Coca cordial too, for like reasons, often has a most satisfactory effect.

In more severe cases, or in cases where chloroanodyne is not sufficient, the sympathetic may be further and more powerfully antagonized by stimulation of the pneumogastric, as advocated by Dr. Alexander Harkin (and later by Groneman), and so successfully employed in Malta during the epidemic of 1887. Three preparations are available for this purpose, presenting varying degrees of intensity:

(1) Equal parts of saturated tinctures of ginger and capsicum:

(2) Either the oil of mustard or oil of horseradish: Croton oil has been suggested, but is altogether too slow and too uncertain in action:

(3) Finally, the epispastic liquor of the British Pharmacopœia, made by percolating five parts of cantharides with twenty parts of acetic ether.

Whichever is deemed necessary to employ, it should be applied freely over the branches of the pneumogastric in the neck, beneath, in front of and behind the ear, covering three inches of surface, preferably on the right side—Coleman having demonstrated the right vagus commands the smaller intestine. But if the case be one of extreme urgency, the liquid may also be applied beneath the eye; or, if desired, both right and left vagi may be excited.

The effect is almost instantaneous—in some instances, quite so,—and all morbid phenomena are annihilated long before vesication takes place. The purging, the vomiting, and the cramps cease; the

patient falls asleep to find, on awakening again, he is entirely relieved from misery.

With the first two preparations, if mitigated at all, vesication may perhaps be avoided; but in cases of marked collapse, the epispastic liquid should receive entire preference; if desired, it may be used on all occasions, to the exclusion of the others, and certainly is safer.

Bear in mind carefully the fact, that absorption, so far as the *prima viæ* is concerned, is in the main inhibited; yet hydrocyanic acid, chloroform, coca cordial, or the chlor-anodyne compound may, if desired, be given by the mouth in full doses, as their volatile character insures the maximum of absorption when absorption is at all possible.

It is needless to dwell on the importance of rapidity of relief in a malady where every movement is fraught with danger, or call attention to the calmative effects of this treatment upon the nervous system, as evidenced by its sleep-inducing powers in the presence of intense suffering; to its efficacy in restoring the balance of circulation and respiration; of at once aborting or jugulating the disease, and of preventing the possibility of lapsing into a typhoid condition or secondary fever so fatal in prolonged cases. That the same topical remedy is of equal efficacy in every phase of the disease, if desired to employ it, from "summer" diarrhœa up through cholera diarrhœa, cholericine, cholera nostras, to so called Asiatic cholera,

strengthens the presumption that they are pathologically one and the same malady; and likewise affords another illustration of the soundness of Dr. Peter Latham's apothegm,* viz., "that the treatment of diseases, rightly considered, is part of their pathology. What they need and what they can bear, the kind and strength of the remedy, and the changes which follow its application, are among the surest tests of nature and tendency."

* "Latham on Diseases of the Heart."

CHAPTER XIII.

EVIDENCES OF VALUE OF VAGUS TREATMENT.

Herewith are embodied certain reports afforded by the kindness of Dr. Alexander Harkin of Belfast, Ireland. Dr. Harkin says:

“In 1885 cholera was very prevalent on the Continent, but happily did not get footing on our shores (Great Britain). Two years later, however, it made an incursion on Malta, and I then took advantage of its presence to have my theory thoroughly tested by competent and independent authority, and for this purpose, at the instance of my friend Lord Knutsford (then Sir Henry Holland, Secretary of State for the Colonies), forwarded a copy of my brochure on the nature and treatment of sporadic and epidemic cholera to Sir Lintorn Simmons, Lieutenant-Governor of Malta, with the request he invite the attention of the medical faculty of the Island to the work. This was done. The medical men loyally responded to the call, with what result the official reports will show.

“I desire now to present independent testimony as to the value of the effects of the treatment in English cholera in all its phases, from an English practitioner of standing and position, personally unknown to me, who communicated the same to the profession through the medium of *The Lancet*. I refer to Dr.

Harry Pool Berry, who, in a letter headed 'Stimulation of the Vagus,' writes:

"In an annotation you state the success attending the treatment recommended by Dr. Harkin is 'almost too good to be true.' This may be so. Fortunately I have had no opportunity of trying the remedy in Asiatic cholera; but as to its being remarkably effective in English cholera, or the summer diarrhœas and vomitings which we so frequently meet with, I feel convinced after repeated trials. The letter of Dr. Harkin appearing in *The Lancet* of August 16th, 1884, was pointed out to me by Mr. Thurston, of Ashford, and since I have tried the external application in the manner described,—viz. blistering behind the angle of the jaw—in at least twenty cases which were more or less severe, and varying from infancy to old age. In all the cases the treatment was attended with marked and immediate success, the vomiting and diarrhœa being controlled almost at once; in some of these cases I had vainly tried acids and opium, catechu, chalk, logwood, etc., in the usual doses. It is, at any rate, a method of treatment which is attended by no risk, and in no way interferes with any other which the practitioner may see fit to carry on."*

The Lancet of October 3d, 1885, remarks:

"So much success has apparently attended the mode of treatment recommended by Dr. Alexander Harkin that no apology is needed for reminding our

* Dr. Berry, on November 18th, 1889, wrote Dr. Harkin, confirming his previously expressed opinion after increased experience.—S.

readers of it. The treatment is in itself remarkably simple—viz., stimulation of the vagus nerve so as to inhibit the action of the sympathetic on the abdomen—for from a consideration of the phenomena of cholera, Dr. Harkin arrived at the conclusion foreshadowed half a century ago by Dr. MacCormac, that in the inordinate action of the sympathetic we have an explanation of the violent purging, cramps, and other characteristic symptoms; and he argues from known physiological effects of the relations between the vagus and the sympathetic to the trial of the remedial measure above stated. Certainly the cases cited by him are striking instances of the rapidity and efficiency of his plan in severe choleraic diarrhœa, and it is incumbent upon those who have to deal with the graver malady to prove the value of Dr. Harkin's recommendations. If by so simple a means he has really discovered a remedy which will diminish the high rate of mortality in cholera, he will have rendered a great service to medicine and to humanity at large. Formerly, in the diarrhœal stage Dr. Harkin employed the routine plan of absolute rest, warmth, counter-irritation to the abdomen, liquid diet, and the administration of sulphuric acid and opium. Now, when at liberty to do so, he discards all internal remedies, and merely applies some epispastic fluid with a camel's-hair pencil, commencing behind the ear and extending on the course of the pneumogastric nerve as far as the angle of the lower jaw. The result is that the purging at once ceases; the patient quickly falls asleep, and awakes cured long before vesication takes full effect. So, also, in the second and algid stages the same good result is experienced. It is almost too good to be true."

Prof. Pisani, in his comprehensive "Report upon

Cholera in Malta in 1887," declares the disease was first manifested in the Island on July 25th, was officially notified on the third of the succeeding month, clean bills of health being delivered on the 11th of November. It does not appear, however, from various causes, that there is any record of Dr. Harkin's treatment being adopted until the 31st of August. In this report, which is largely historical and topographical, Dr. Pisani, referring to this treatment, states: "In many cases the improvement was very rapid after the protracted sleep which followed the counter-irritation of the vagus nerve of one (the right) or of both sides."

Again, chiefly at Zabbar, Zeitun and Manoel Hospitals, strong counter-irritation was applied to the pneumogastric on the right side, or on both sides, on that portion lying between the mastoid process and the angle of the lower jaw. It acted "frequently like magic, the patient sleeping after its use and awaking well."

I now offer a transcript of the evidence obtained in Zabbar and Zeitun Hospitals by Drs. Inglott and Cannataci, the opportunity of reproducing being afforded me through the courtesy of Dr. Harkin. He remarks, "unfortunately, there were no records preserved at the Manoel Hospital, which was merely improvised for the occasion."

Dr. Inglott's Report:—"Dr. Harkin found a direct means of acting on the sympathetic nerves independ-

ently of the rest of the nervous system. This means consisted in counter-irritation over the vagus which I have successfully applied not only in epidemic cholera, but also in whooping-cough. The treatment in my hands proved to be very beneficial, and my personal experience during the late epidemic is quite in accord with Dr. Harkin's opinions. Often cases of very severe type were arrested by means of this treatment when all other resources had been employed in vain. The treatment was also used by my friend, Dr. Cannataci, while in charge of Zeitun Hospital, and we worked together hand in hand, helping each other, communicating daily our observations. Dr. Harkin's treatment acted in our hands in most cases like magic. I am glad to be able to state my conviction that we saved several patients from death by such means. I remember well, in the Zeitun Hospital, the case of a poor boy, eight years of age who was in so advanced a stage of algidity there was very little hope of saving his life; all internal remedies had failed. I was quite astonished on seeing him, after apparently dying in the morning, quite convalescent in the afternoon as the result of strong vesication over the vagus. In conclusion, I have no hesitation in saying that Dr. Harkin's treatment is a remedy both reliable and speedy in its action in all severe cases of cholera."

Dr. Cannataci commences his report by confessing the vagus treatment failed in several instances, but in

many more "acted like magic." Herewith is presented a summary of cases as outlined by Drs. Inglott and Cannataci:

CASE I.—William Quintal, 7 years of age, was removed to the Zabbar Hospital, on August 31st, 1887, at 10 P.M.

Actual Condition.—Eyes very hollow; lips violet; hands and forearms cold; neck and upper part of the chest of bluish color; intense diarrhœa and vomiting; voice feeble; cramps very severe; thirst intense; suppression of urine; pulse imperceptible; extreme weakness; the patient refused to take internal medicine. Dr. Harkin's treatment applied at once. After sixty minutes the patient slept for nearly three hours, and soon after took lemonade with ice and cognac.

Sept. 1st, 3 A.M.—Counter-irritation was applied again on the left side of the neck.

At 7 A.M. the patient took coffee and milk, and continued to improve. After two days he left the Hospital, weak, but perfectly cured.

CASE II.—Carmela Briffa, 49 years of age, was admitted into Zabbar Hospital on Sept. 4th, 1887, at 7 A.M. During the night she had slight diarrhœa, preceded by chill and perspiration, which were neglected; soon after she had vomiting. At 6:30 A.M. I was called to assist her; at 7 she was removed to Hospital.

Actual Condition.—Face livid and violet; cramps very violent in the upper and lower extremities—the pain produced contortions of the face; eyes sunken in their orbits, encircled by a dark blue line; aphonia; dejections resembling "rice-water;" expression of great anxiety; tongue dry; hands violet; whole body cold; suppression of urine.

Treatment.—7 A.M., injections of ether and stimulants by the mouth; 8 A.M., same state; 9 A.M., thirst intense, pulse very weak, cramps severe, vomiting and diarrhœa; 2 P.M., no change up to this hour. Counter-irritation on both sides of the neck. At 3 P.M., slightly disposed to sleep; pulse active, no cramps, no vomiting, one stool; 5 P.M., slept one hour; soon after took some coffee with brandy; 11 P.M., improving.

Sept. 5th.—4 P.M., passed a very good night; declared convalescent; beef-tea, Marsala wine.

Sept 7th.—At 10 A.M., left the Hospital perfectly recovered.

CASE III.—Antonio Abela, married, laborer, 27 years of age, was admitted into Zabbar Hospital on Sept. 11th, 1887. On the 10th he had been taken ill, nearly at midnight; after that he committed dietetical errors, eating a quantity of fish commonly called lamperchi. At 8 A.M., September 11th, I was called to visit him.

Actual Condition.—Universal cramps; expression of intense suffering; voice extremely feeble; face violet and livid, eyes sunken in their orbits; vomiting very intense; no diarrhœa; whole body cold; pulse imperceptible; respiration very difficult; suppression of urine. Removed patient at once to Hospital.

Treatment.—Injection of ether; stimulant mixtures.

9 A.M.—Vesication over the vagus on the left side of neck.

11 A.M.—Found patient had slept nearly one hour; cramps stopped, pulse active, vomiting suppressed, urine passed freely. The patient, in my presence, took some broth and two spoonful of Marsala wine. He continued to improve daily, and after a few days left the Hospital able to work.

CASE IV.—Guiseppe Galt, 42 years of age, shop-keeper, residing at Vicolo, San Francesci, Zabbar. This patient watched his wife when attacked with cholera with great anxiety, but she refused to undergo the vagus treatment, and died thirty hours after seizure. Four hours after her death he was seized with violent diarrhœa, and refused to be removed to Hospital. The following were the symptoms observed:—Diarrhœa very intense (“rice-water”); vomiting; eyes very hollow; lips violet; body cold; voice feeble; pulse weak; respiration disturbed; sense of oppression; thirst intense; cramps very severe in the lower extremities; general debility; suppression of urine.

Treatment.—Injections of ether, vesication very strong over the vagus on both sides of the neck. After two hours I visited the patient again, and found he had slept nearly one hour; cramps stopped; pulse active; diarrhœa suppressed; urine passed freely. I called again to visit the patient in the afternoon, and found him weak but recovered. The vagus treatment acted like magic!

CASE V.—Michele Buchagica, 52 years of age, was seized with violent vomiting and diarrhœa on Sept. 12th, 1887, at 11 P. M. I was called to visit him at his residence, Vicolo, San Francesci, Zabbar.

Symptoms.—Eyes very hollow; hands, forearms, neck and upper part of chest cold; thirst intense; diarrhœa (“rice-water”); vomiting; sense of great oppression; voice very weak; pulse almost imperceptible; cramps confined exclusively to the lower extremities.

Treatment.—Used at the very moment the epispastic fluid on both sides of the neck, and prescribed a stimulant mixture. After four hours I visited again

and found him convalescent. Being very weak, I advised to continue the stimulant mixture, and likewise to take milk with cognac. After four days he was able to leave his bed.

CASE VI.—Vincenzo Barbara, 26 years of age, of very good constitution, married, admitted into Zeitun Hospital on Sept. 18th, 1887.

History.—Well-nourished man and good laborer; had committed for several days dietetic indiscretions; stated he was in perfect health on Sept. 17th; worked as usual, and slept well. Seized with cholera 4 A.M., Sept. 18th; in only three hours had ten discharges; the diarrhœa was not attended or preceded by colic or other pains in the abdomen; cramps occurred soon after the attack and continued without intermission. At 9 A.M., Sept. 18th, he was admitted into Hospital.

Actual Condition.—Face livid, violet; eyes hollow; pulse small and feeble; somnolent, but sleep checked by cramps; thirst intense; diarrhœa profuse, like boiled rice; vomiting intense and frequent. The patient seemed feeble to an extreme degree.

Treatment.—Injections with sulphuric ether; enteroclysm with tannic acid, quinine and carbolic acid; excitant mixture with liquor ammonia; cognac with ice.

11 A.M.—Blue color very marked; pulse scarcely sensible; painful cramps of the lower extremities very frequent; body cold; diarrhœa profuse; vomiting frequent; respiration disturbed; voice hoarse; no urine. Counter-irritation applied on the left side of the neck.

3 P.M.—Respiration less frequent; cramps less frequent and less severe; diarrhœa continued. Counter-irritation applied again.

6 P.M.—The patient slept one hour; same symptoms, but less severe; same treatment.

Sept. 19th, 6 A.M.—The patient slept four hours during the night; body cold but not like ice; diarrhœa moderate; no urine; same treatment.

20th.—The patient passed a good night; good broth and two eggs; a dose of Marsala wine.

21st.—The patient convalescent.

22d.—Gradually improving. On Oct. 5th left Hospital cured.

CASE VII.—On the same day was admitted into Zeitun Hospital, Cornate Jarionolite, 13 years of age, of scrofulous constitution, with the following symptoms: Face, hands, and forearms bluish; body cold; eyes very hollow; diarrhœa ("rice water"), with vomiting; cramps severe, confined exclusively to the lower extremities; anæmia; pulse very weak; respiration disturbed.

Treatment.—Applied at once the vagus treatment on left side of neck; excitant mixture internally. After two hours the patient slept well, and the symptoms were less severe. She continued to improve gradually, and on Sept. 28th left the Hospital perfectly cured.

CASE VIII.—On Sept. 26th called at 3 A.M. to visit A. L——, 32 years of age, of very weak constitution; she was seized at 1 A.M. with severe diarrhœa and vomiting; I removed her at 3:30 A.M. to the Zeitun Hospital while suffering very severe symptoms of cholera. I applied at once the vagus treatment on both sides of the neck, and after four hours found the patient improving; she was discharged cured on Oct. 2d.

CASE IX.—T. C., 8 years of age, came with her mother to the Hospital on Sept. 27th with the follow-

ing symptoms:—Face livid, violet; eyes hollow; pulse small and feeble; somnolent; thirst intense; cramps; profuse diarrhœa, like boiled rice; vomiting intense; all symptoms of a severe attack of cholera in fact. I applied the vagus treatment at 7 A.M., and soon after she slept well and began to improve. On Oct. 29th she left the Hospital cured.

CASE X.—On same day L. A., 22 years of age, was also admitted into Zeitun Hospital in a very weak state, and with most severe symptoms of cholera. The vagus treatment was applied at once, and the patient improved immediately.

In his "Report" Prof. Pisani gives the clinical history of three cases treated by Dr. Inglott on the ordinary system, two of whom died and one recovered, before the adoption of the vagus treatment, the fatal cases not more virulent than those saved by the topical remedy; the favorable one proved successful only after nine days of continuous medication, and *not until tentative counter-irritation* by liniment of iodine was applied behind the ears. This instructive case is as follows :

Madalena Briffa, 23 years of age, living in No. 3 Vicoli i Strada Bazadey, came to Zabbar Hospital on Sept. 2d, 1887, at 11 P.M. A spinster, very poor, of good character and good constitution, but slightly scrofulous. Had committed no excesses; for nearly five days continually assisted her mother, who suffered from attacks of heart disease. Being poor, she had been eating only rice boiled in water and some bread. On Sept. 2d, at 6 P.M., she had a mod-

erate diarrhœa. The dejections suddenly became frequent; at 10:30 assumed a serious form, and became like boiled rice; cramps began simultaneously with the vomiting; the voice became feeble and the body cold. The patient requested urgently to be removed to the Hospital. I was called at 10:40, and gave her temporary assistance; she was removed to the Hospital at about 11 P.M.

Actual Condition.—Surface of the whole body icy cold; color of face so deep as to be almost black; the skin of the extremities singularly wrinkled and livid; voice nearly lost; the expression of countenance one of great anxiety; eyes much sunken; the upper and lower extremities affected with cramps in violent degree; cold perspiration; suppression of urine; vomiting, the expelled fluid being thrown with great violence and to a great distance; pulse imperceptible; thirst intense; great prostration; respiration difficult.

Treatment.—Stimulation by spirituous liquors; subcutaneous injection of sulphuric ether; hot bottles; frictions with spirit of camphor, and soap and ammonia liniment; enema of spirit of camphor and hot infusion of coffee; large sinapism on the spinal column; large poultice of cumin seed and chamomile to abdomen; stimulant mixture of liquid acetate of ammonia and spirit of chamomile; cognac in an effervescent mixture of bicarbonate and chloride of soda.

Sept. 3d, 1887, 12:30 A.M.—Features immovable; great prostration; eyes dull, sunken; face, including lips, livid and cold; arms and feet cold; tongue pale and cold; great thirst; complete aphonia; suppression of urine; expression of great anxiety; diarrhœa persistent (“rice water”); no vomiting; same treatment.

At 6 A.M., same condition; same treatment.

2 P.M.—Dull of intellect; face always cold; nose icy; expression of great suffering; arms icy cold; complete aphonia; no urine; hearing and sight greatly deranged. Patient suffered less from cramps after being rubbed and warmed by hot bottles; extremities warmer; same treatment.

4:30 P.M.—Same state; same treatment.

8:30 P.M.—Patient very bad; respiration disturbed; diarrhœa, very profuse; folds of skin when raised between two fingers disappeared very slowly, as in the case of corpses (constant symptom in all bad cases of cholera which ended fatally).

9:30 P.M.—Symptoms unchanged; five hypodermatic injections of spirit of camphor, two at each arm and one at the left leg; enteroclysm of cognac, ether, and spirit of camphor, in decoction of chamomile; drops of ether and liquid acetate of ammonia in hot water; frictions of ether with hot vinegar; large poultice of cumin seeds, chamomile, and mustard meal, from the upper part of chest to lower part of the abdomen; strong liniment of iodine on the spinal column.

12 A.M.—Patient a little better, but symptoms not improved; two injections of ether on the spinal column, one at the upper, the other at the lower part; enema of strong infusion of cumin seed with two drachms of spirit of chamomile.

Sept. 4th, 12:30 A.M.—Slightly disposed to sleep; purging less frequent; cramps less violent; vomited once; extremities cold; chest and abdomen warmer; respiration difficult; intelligence heavy; pulse perceptible, but very weak; speaking caused great fatigue. Ordered perfect rest, medicines to be stopped, perfect silence.

3 A.M.—Slept one hour; one stool, with slight

efforts to vomit; pulse little excited; enema and gum Arabic mucilage; a dose of bismuth internally.

8 A.M.—Slept two hours; no vomiting, no stools; pulse feeble; surface warm; comatose; two spoonful of broth and a good teaspoonful of Marsala wine every two hours.

10 A.M.—One stool; very little urine; infusion of chamomile and large poultice to abdomen; pulse weak; face pale; no cramps; no vomiting; expression of great weakness; eyes brighter; intelligence clear; a little thirsty; same treatment with lemonade, and Marsala (iced), every three hours.

12:30 P.M.—Vomited greenish matter; one stool; one ounce of urine since 10 o'clock; thirst less; tongue red at border and top with mucous coating; great debility.

5 P.M.—Patient complained of sounds similar to ringing of bells in the ear, which prevented her from hearing well; liniment of iodine behind the ears; respiration normal; pain at the extremity of the last rib on the right side; friction of laudanum; broth and Marsala wine.

Sept. 5th, 8 A.M.—Voice natural; urine copious; diarrhœa ceased; very weak; a few spoonful of beef-tea every two hours; a mixture of tinct. quassia and cinchona every four hours.

5 P.M.—Felt better; same treatment.

Sept. 6th, 8 A.M.—Passed a good night, and slept very well for four hours; relished coffee; still weak; same treatment.

Sept. 7th.—The menses, which stopped at the beginning of the disease, returned during the night. Patient very weak and unable to move her arms; beef-tea, Marsala wine, two eggs.

8th.—Appetite tolerably good; voice natural;

face pale; appearance of small abscesses; syrup of hypophosphite of iron, a tablespoonful morning and evening.

9th.—Slept well; no dejections; urine very abundant; skin warm; face pale; weak; same treatment.

10th.—Improving; same treatment.

11th.—Patient out of bed and declared convalescent. Continued to improve gradually. On Sept. 19th discharged, cured, but being very poor was kept on the Hospital diet-book.

This was one of the most severe cases I ever saw followed by recovery.

The case of Madalena Briffa, was especially remarkable for its virulence, and the narration thereof exhibits in strong light the devotion of Dr. Inglott, and the fertility of resources at his command. But contrast her case with that of Carmela Briffa, 49 years of age (Case II), admitted two days later into Zabbar Hospital, whose medical history is comprised in the few words: "Admitted on Sept. 4th, at A.M. Hypodermatic injections and stimulants failing to relieve, at 2 P.M. counter irritation on both sides neck; soon after slept. Next day, Sept. 5th, 4 A.M., declared convalescent. Sept. 7th, 10 A.M., left Hospital, perfectly recovered!"

These clinical reports certainly do not call for any lengthened comment; they tell their own tale.

And now believing enough has been said to carry conviction that stimulation of the vagus is the

key to the treatment of cholera in any and all of its diversified forms, or at least that it is worthy of most careful consideration, I will say

“Vale.”

APPENDIX A.

THE HISTORY OF EUROPEAN CHOLERA AND ASIATIC CHOLERA.*

This morbid affection was known in times of greatest antiquity. The Bible, in the book of Sirach, says men given to crapulation are threatened with cholera. In the *Cohélet* or Ecclesiastes, caput vi, it is written: "*Cholira est aliud malum sub sole frequens apud homines.*" And Deuteronomy, the Vulgate, chap. xxviii, says: "*Augebit Dominus plagas tuas infirmitates pessimas et perpetuas cholaim-raim.*"

[Perhaps it is needless to remark that these purported quotations are fraudulent, in that they are garbled, and further do not in any way refer to cholera; the word *Cholira* in the one instance, and *Cholaim-raim* in the other, are interpolations of the author.

The sentence in Ecclesiastes (chapter vi, verse 1), in the Vulgate is: *Est et aliud malum quod vidi sub sole, et quidem frequens apud homines*, or "There is a certain evil which I observe daily, that is frequent among mankind"—the Preacher in this instance

* Translated from Ozanam's "Histoire Médecine Générale et Particulière des Maladies Épidémiques," by Thomas C. Minor, M.D., Cincinnati—Embodied by permission of the Translator.

referring definitely to *vanity*, without even a thought of gastric griefs or peristaltic woes.

Again, Deuteronomy (chap. xxviii, verse 59), winding up a threat of many evils, the Vulgate has it: *Augebit Dominus plagas tuas, et plagas seminis tui, plagas magnas, et perseverantes, infirmitates pessimas et perpetuas*: "God will multiply plagues and the plagues of thy seed, great plagues of long duration, and severe illnesses of great continuance."

The King James version gives these respectively as: "There is an evil which I have seen under the sun, and that is common among men," and: "Thus the Lord will make thy plagues wonderful, and the plagues of thy seed, great plagues of long continuance, and sore sickness, and of long continuance." —S.]

Hippocrates ("De Morbo,") cites the observation of Silenus, who died the eleventh day of an attack of cholera, coming on after hard work and excessive drinking; and, in his sixth book on "Epidemics," he speaks of an Athenian who was cured the third day of the disease.

Cornelius Celsus and Cœlius Aurelianus also speak of cholera. The latter mentions certain symptoms that are manifest in Indian cholera, such as the vomiting of white watery fluid.

But it is in Cappadox Aræteus, that true painter of diseases, that we find a very concise and clear description. "It is," says he, "an inverse movement of morbid matter which reflects on the stomach and intestines; it is a very acute affection. The materials collected in the stomach are rejected by vomiting; those carried

into the intestine are evacuated by the stools. The first vomitings are watery; the first stools are liquid, stercoraceous and infected, and at times mucous or bilious; at times the malady begins in a benign manner, without pain, but afterwards tension of the epigastrium comes on, with constriction of the throat and violent intestinal colic. As the disease progresses there is increase of the colic, mental depression, muscular contractions, and a lowered vitality. If the patient drinks there is nausea, accompanied by internal noises; bilious vomiting, with stools of the same nature; distension of nerves; muscular contractions of limbs; curved-in fingers; vertigo; hiccough; blue nails; cold extremities; rigidity of the body. If the patient grows worse, there is profuse sweating, throwing off of black bile by both orifices, spasm of the bladder, stoppage of urine, which may have been profuse before, noting the same derivation that acts on the intestinal tube; aphonia now becomes manifest; pulsation of the arteries is scarcely apparent and very rapid; there is continual nausea, and tenesmus without dejections; and death arrives in the midst of atrocious pains, convulsions, and a feeling of strangulation. This malady occurs principally in summer, rarely in winter. It attacks the young and robust individual, and the infant, rather than the old."

Diogenes, the Cynic, died at Corinth of cholera, after having eaten rare beef's foot.

Ancient physicians appear never to have observed cholera under its epidemic form; they only mention it as a grave sporadic affection of short duration, not always fatal.

It was in the sixteenth century that cholera was noticed to reign epidemically. Mezeray, the historian, reports that a colic called *Trousse-Galant* appeared in

France in 1528 and prevailed until 1531, the epoch when the horrible plague of which it was the precursor ravaged Europe.

Alcmarinus Forestus ("Observationum et Curationum Medicinalium," liber 18) describes epidemic cholera as it prevailed at Alkmaert in 1548. This outbreak was characterized by vomiting and dejections of an aqueous, limpid material, followed by general prostration, cold sweats, syncope and death. This epidemic ravaged Delft at the same time.

Lazare Riviere ("Observationes Medicæ et Curationes Insignes," etc., liber 26, Lyons, 1680) remarks: "In the year, if I be not mistaken, 1645, before the plague attacked Nimes, there came a disease called cholera, killing many patients in four days; nevertheless, those who sought medical advice at the commencement of the malady almost all escaped by this method: Patients drank little; they were given quince jelly and their limbs rubbed with aromatics; embrocations of oil of chamomile, heated, and topical applications of aromatics on the epigastrium, were used; cordials were administered, and astringent opiates with rhubarb and clysters."

The most celebrated epidemic of ancient cholera was that which prevailed in England from 1669 to 1672. Thos. Sydenham has left an excellent description of this outbreak; he was himself attacked while suffering from gout.* The following is his sketch of the malady:

"It was at the commencement of August, 1669, that the plague started in London. This malady was easily recognized by the continual nausea, enormous

* See page 7.

vomiting, black and fœtid stools difficult of emission; atrocious pains in the intestines, tympanic distension of the abdomen, cardialgia, irregular and accelerated (sometimes feeble) pulse; heat and dryness of the skin, colliquative sweats, intense thirst, contraction of the limbs, sadness, cold extremities, and other symptoms that were much more serious and that were often the fore-runners of death, in the space of twenty-four hours. It also showed itself under the dry form, with colic, without vomiting or stools. Hippocrates ("De Ratione Victus in Acutis," liber 11) and several other authors of ancient times, have observed a similar complaint. It is characterized by abdominal tympanites and flatulence at the upper and lower orifices."

It was at the commencement of August that Sydenham observed the malady at London, and, as before stated, it was easily diagnosed by the great vomiting, continual nausea, etc. Sydenham observed that purgatives aggravated the disease, while narcotics and astringents prevented the exit of excrementitious matter, and hence were dangerous. He sought a new method of treatment by aiding the evacuations by diluents, such as chicken broth and skimmed milk; he gave clysters of decoctions of lettuce and water lily, and also used syrups in drinks. After using these for two or three hours he terminated the treatment by a calmative potion, with a little laudanum. When the physician was not called in until after ten or twelve hours, at the time when the patient, owing to repeated vomiting and purging, was exhausted, he prescribed laudanum immediately; this was continued morning and night, notwithstanding the cessation of the evacuations, up to such a time as the patient had recuperated his strength.

This epidemic only lasted during the month of August in that year.*

During the following years, 1670, 1671, and 1672, the same epidemic prevailed in London; it tended towards dysentery in character, and of which it was often a degeneration. It attacked by preference the young of hot or bilious temperament. The intestinal pains were atrocious; the patient appeared constricted as by a strong girdle, or pierced by some sharp instrument. These pains diminished little by little from time to time, but only to recommence with renewed violence. During the paroxysm the patients' countenances were distorted, and they uttered lamentable cries. The vomitings were not very frequent, and constipation readily yielded to cathartics; but, pains, from the first erratic, fixed upon one point. During the progress of the affection the vomitings increased, the belly was contracted, and the peristaltic action of the bowel became totally inverted; from them iliac passion declared itself, clysters and excrements were emptied by the mouth, admixed with greenish, yellow, and other curious colors.

The treatment consisted in general bleeding, three or four times after anodynes; the next day a mild cathartic, which was repeated, after a day's interval, three times, according to the abundance of the humors, that were diluted by drinks such as milk and beer. In the iliac passion cathartics were useless, and were only employed among subjects whom one knew to be easily relaxed; in such cases some mild laxative, such as tamarinds, senna, rheu-

* I fail to see a picture of cholera in either this or the subsequent description.—S.

barb, or syrup of roses, were used. If patients could not stand liquid medication, resort was had to pills. But if the stomach still refused, treatment was commenced by prescribing an anodyne potion, and a cathartic a few times after; then repeated the first remedy morning and evening, up to such a time as the pains disappeared. Carminative lavements (injections) carried the trouble into the intestinal system, and made the disease more rebellious. The diet was light, composed of barley cream, and panada; later a little chicken and fish were allowed. The drink was milk or light beer diluted with water. For patients of wealth, horseback exercise was advised to recuperate the strength.

The following observation by Dr. I. Frank, of Ulm, made in the years 1695 and 1696, is curious and valuable:

“In the year 1695 the winter was very cold; a dry freezing spell lasted until almost spring-time. Then suddenly came rains and unhealthy fogs, and almost all children had violent coughs. In the month of May measles appeared; this disease prevailed until July, when it became complicated with diarrhœa. August came in cold and rainy. The prevalent diseases disappeared towards the equinox of autumn, but at the commencement of October a new epidemic, *i.e.*, bilious colic, became manifest; this malady was accompanied by fever, constipation, terrible pains in the abdominal region, with cruel spasms that started at the loins and extended to the umbilicus. These spasms only ceased to renew themselves. The belly was retracted, becoming concave like that of certain hysterical women. Sometimes the pains were felt deeply seated in the right hypochondrium, at the spot where the pancreatic duct and ductus choledochus

are inserted into the duodenum. Vomiting occurred from time to time, caused by the spasmodic contractions of the colon, or convulsions attacked the limbs, degenerating into contractions and paresis. This disease prevailed especially among men given to drink, and women were not spared. One knew not whether to attribute the malady to new wine made from unripened grapes, to the variations of the atmosphere, or to some terrestrial conditions."

The following is an example of the affection:

"A literary man, aged about forty years, of delicate constitution and habitually constipated, experienced pain in the loins, with frightful colic. He was put to bed and took heated remedies in order to provoke sweating, but received no relief; on the contrary, the colic was increased, to his great prostration, and he had a pain in the dorsal region corresponding to that in the gastric and umbilical quarters; this pain extended to the hypochondrium, descending to the perineum, going back into the belly again and retracting the navel; sometimes it was all over the abdomen, including the scrotum. The sixth day two injections were given him, with a decoction of veronica (*toad flax*), with a laurel electuary and Hannes confection — that which quenched the pains; the patient also took peppermint water. He recovered in a few days. Veal soup flavored with anise seed was employed with success, but purgatives aggravated the malady."

In the year 1695 Jean Jacques Schaller, of Basle, gave the history of a similar malady that prevailed epidemically in Switzerland, and was attributed to the bad quality of the wine. An obstinate constipation was manifest, with vomiting and loss of appetite, constant nausea and eructation, prostration and lassi-

tude, accompanied by a little fever; thirst, wakefulness, convulsions, and painful ischuria were the principle symptoms of the disease. The affection was treated with oily injections of sweet almonds and Spanish wine, with tincture of castor and syrup of peppermint, two spoonsful of the mixture every three hours. Infusion of chamomile and mint, with syrup of poppy and orange peel, were also employed. The treatment was terminated with a light solution of manna or cream of tartar in chamomile water; stronger purgatives were injurious.

Towards the close of 1717 the inhabitants of Pegau, in Lower Saxony, were attacked by epidemic colic, with the following symptoms: Light chill, followed by intense fever and great thirst, bilious vomiting, acute pains in the hypochondrium, tension in the præcordial region, violent cough, hiccough, the latter principally among pregnant women; difficult respiration, feeling of weight at the diaphragm, face becoming sub-icteric; the urine, clear at the commencement of the malady, grew red and sedimentary towards the decline; the pulse was rapid; a painful constipation, or frequent alvine dejections, persisted during the whole course of the disease. Bleedings and febrifuges were so injurious that suffocation, delirium, and death, invariably followed their use; refrigerants and light laxatives always cured the patient on the fifth or seventh day.

Antoine Augustini, of Venice, speaking of the epidemics of the year 1741, states:

“Through the Venitian States that summer a violent epidemic of colic prevailed. The attacks commenced with great præcordial anxiety, feverish pulse, dyspnœa, flatulence, acute pains in the bowels, and constipation. The malady passed promptly into

a tympanitic condition, or degenerated into dropsy or dysentery, and, if not promptly treated, old persons when attacked died. Light bleedings, the application of leeches to the hæmorrhoidal veins, clysters, and emollient drinks, were the most appropriate remedies to be used in the disease."

The "Memoires de l'Academie des Sciences de Paris" are full of observations on epidemics of in Paris. The learned Dr. Malouin writes:

"Cholera morbus suddenly appeared in Paris in the month of July, 1750, and soon became epidemic. It had at first the aspect of hepatic colic, owing to the pain the patients experienced in the neighborhood of the liver; but the patients were not jaundiced, however, and their excrement was not white. Many patients succumbed the third day, especially if the colic was accompanied by indigestion. In general, the face was drawn and the patient hollowed-eyed, especially when vomiting was present. The pulse was rapid, but compressible; the belly was distended; constipation was obstinate, with pains in the hypochondrium and lumbar region. Bleeding seemed to aid some of the sick by diminishing the convulsive tension of the abdomen; afterwards emollient drinks were employed, and tepid water, with chicken broth, followed by light purgatives; and the cure was terminated by the use of Vichy water."

Dr. Lentin has left on record the history of an epidemic of so-called European cholera that occurred at Dunaburg in 1765:

"For several years we saw prevail a sporadic form of epidemic cholera that attacked a large number of persons. There was a right lateral pain, with cough, in this affection, with sanguinolent expectoration, accompanied by febrile heat; but this morbid affection

soon changed its character, for a chill came on, followed by severe pain in the left side, with præcordial anxiety, nausea, vomiting, and headache. The fourth day there was high fever, with thirst and very dry mouth and pain in the throat; the disease now invaded the abdomen; there was obstinate constipation, frequent belching, which provoked regurginous vomiting, and then degenerated into a putrid diarrhœa with tension of the abdomen; the pulse became feeble and compressible; the urine was sour; there was often great pain present, and even tumors at the articulations; these latter were almost a sure indication of near death. As to treatment, the patient was bled, and powders of nitre and camphor, with decoctions of oats, emollient clysters, and laxatives, given. Bladders full of hot milk, cataplasms of mallow, and sometimes blisters, were applied over the seat of pain. When the diarrhœa became fœtid, camphorated quinquina was given."

In the month of July, 1766, an epidemic of bilious colic, complicated with cholera, prevailed in London, and is described as follows by Dr. Sims, in his "Observations on Diseases."

"Women were more frequently attacked than men. Sometimes the invasion of the disease was sudden, at other times it was preceded for two or three days with depression, and if at the commencement of the malady we used lemonade as a drink, freely given, or something that gently acted on the bowels, the malady was frequently cut short. If there was a bilious congestion of the alimentary canal, the pulse was small and intermittent."

In May, 1779, cholera morbus broke out in Fougères, in Brittany, and attacked principally peasants and English prisoners of war. This epi-

demic prevailed until October, but reappeared the following spring. The symptoms were violent pains in the entrails, heavy weight in the epigastrium, nausea, vomiting of bile, acute pain in umbilicus and sometimes in kidneys. There was ordinarily constipation and red urine, with bitterness in the mouth and retraction of the umbilicus. The alvine dejections were bilious, usually preceded by hard lumps of matter resembling sheep's dung; there were large quantities of bile in the vomit.

The so-called European cholera often declares itself on vessels in tropical regions; this disease almost destroyed the Australian expedition that sailed from Trieste in 1821 to make the tour of the world, and was commanded by Baron Schimmellpening, who was one of the victims, as well as the captain, and the famous botanist, Bohun.

The Asiatic cholera is an epidemic malady in India and other equatorial regions, and from thence spreads to Europe and other portions of the world.

Its synonyms are: *Sitanga* or *sinanga* (Sanskrit); *Ho Louang* (Chinese); *Morxi* or *mordechi* (Indian); *Ouebb* (Persian); *Hachaiza* (Arabic); *Haoucha* (Armenian); the *gripping disease* (Indo-English); *Braal Loop* (Dutch); *Cornaja Coleza* (Russian); *Cholera spasmodique* or *Trisplanchnite* (French).

Cholera, since times of antiquity, has prevailed in Asiatic countries under its sporadic form, seldom becoming epidemic; but suddenly, in 1817, it assumed this latter shape and invaded the banks of the Ganges, advancing from the southeast towards the south and northwest, covering two continents with the veil of death. In its immense flight it covered one hemisphere and then the other; it hovered over each country in turn, and, like a vulture, seemed to fly in cir-

cles, marking in advance the scene of its devastations; then suddenly, with the rapidity of lightning, it immolated its victims by the thousands. English physicians in India, like Dr. Tytler, have observed that the cholera always exercises its fury upon the frontiers of a province before going to the interior. Happy would it be for humanity if, like the plague, cholera were contagious (it has been proved that it is not); it would be easy then to preserve ourselves from its ravages; but its genesis, purely epidemic, renders it a thousand times more disastrous and unmanageable on this account.

The march of cholera has given rise to the following reflections, that we submit before tracing the further history of the Asiatic plague:

Great movements of the physical phenomena of the universe are made from east to west, such as the moon, stars, the rise and fall of the ocean tides, while the Earth makes its revolutions in a contrary sense. The human race had its birth in the Orient; it propagated from east to west, from the borders of the Euphrates to the western rivers. Thus the first children of Earth peopled India, Africa, and South-eastern Europe. In the ages following, the Scythians and Tartars crossed the Oural and Caucasian Mountains to populate Russia, Turkey, and the borders of the Black Sea, and other of the larger water-courses of Europe. From thence came the Huns, Lombards, Vandals, Swedes, Germans, Gauls, Goths, and Vis-Goths, while the Bourguignons settled themselves along the Danube, Rhine, and Northern Ocean, overflowing this territory like a flood. Later the Moors, coming also from the Orient, established themselves in Spain, while the Normans invaded Western Gaul, and the Saxons Great Britain. In more modern

times we saw the inhabitants of Europe going west to found colonies in America. Finally, all outbreaks of barbarians have been from the Orient to the Occident.

All religions have followed the same march, such as Judaism, Christianity, Islamism, and even the schisms of these churches.

The same march is held by epidemics: the plague, small-pox, measles, and leprosy, were brought from Turkey and Syria by the Moors, Jews and Crusaders who were chased out by the Caliphs. The Black Plague of the fourteenth century started at Katai, in China, and only ceased on the borders of the ocean. The catarrhal epidemics of 1239, 1311, 1323, 1400, 1427, 1557, 1580 and other occasions too numerous to mention, called "*The Russian*," "*The Muscovite*," "*The Influenza*," "*The Dando*," "*The Coquette*," etc., all came from Northeastern Europe, and only ceased on reaching the borders of the Atlantic. Typhus came from Hungary in the sixteenth century, and was called the "*Hungarian Fever*." Finally, cholera came to us from the eastern borders of China and India. We might cite numerous epidemics, notably that of 1814, which followed the same course. No epidemic ever originated in the West or New World; and though it is claimed that syphilis and yellow fever came from there, the proof of this statement is wanting; these two diseases are not epidemic, nor are they the product of atmospheric influences, but are due rather to a contagious virus as regards the first, and to infection in matter of the latter.

Cholera is a purely epidemic malady, carried by the atmosphere, following currents and oscillations of the air, just as it goes up against the courses of rivers. We do not know why this plague was called cholera

or cholera morbus, as it does not correspond with the disease we have described, for the bile plays no part in the affection; the name probably arose from a few symptoms these diseases had in common.

Let us now enter into a brief history of what is known by moderns as

ASIATIC CHOLERA.

We shall only trace the history of this pestilence from 1817 until a more modern date. We shall note the frightful rapidity with which it spreads and the painful disasters it has inflicted on the Old World, as well as America. We have before spoken of cholera morbus, as it was called in Europe; we are now dealing with the disease that is endemic in India. Sanscrit works describe this disease as existing from time immemorial. It was known to the Arabs, and prevailed in Europe as an epidemic as early as the sixteenth century.

Alexander of Tralles ("De Arte Medica," liber vii, cap. 14) speaks of cholera with vomiting and white liquid dejections; while Cœlius Aurelianus (lib. iii, cap. 20) also remarks: "*Crescente passione aquati ac tenuis liquoris fit egestio et aliquando similis loturæ carnis. Feruntur etiam cum his humoribus plerumque sub albida desputa,*" with other symptoms of cholera.

But Jacob Bontius was the first European physician who mentions the disease, over two hundred years ago, in his work entitled "Medicina Indorum" (cap. vi), in the following terms:

"*Fit itaque cholera cum materia biliosa et retorrída ventriculum et intestina infectans per gulam simul ac per anum continno ferme cumque magna copia reficitur. Morbus est acutissimus Ideoque præsentis eget remedio,*" etc.

They employ, continues Bontius, astringent drinks, and a certain fruit juice called billigbing, or the syrup of lemon.

About the same period Zactus, of Lisbon, gave the following simple notice of the epidemic that prevailed in Europe in 1600:

“Anno 1600, quando hæc pestifera lues Europam fere totam oppreerat, obsevavi plures qui hoc diro dolore affecti venenosis symptomatibus excruciat, occubiere omnes; nullus quartum diem pertransivit,” etc.

[It was described by Garcia d'Acorta, a physician of Goa, in the first book ever printed in India.—S.]

Dr. Englishman (“Bibl. Britan.,” April, 1830) reports that the Chinese have observed this disease in the Celestial Empire for ages, and term it *Ho Louang*. It was a Chinese medical writer, Vang-chou Ko, who described the malady long before it assumed its epidemic character in India.

This disease ravages the coast of Coromandel and the Maldivh Isles, and all the borders of India, especially during the hot summer months, succeeding the season of monsoons, when the winds of the southwest, charged with the humidity of the Pacific Ocean, suddenly lower the temperature twenty-five or thirty degrees in a few hours.

It was only towards the end of the last century that physicians and European naturalists collected observations of Indian cholera outbreaks. Paisley described that of Trincomali in 1773; Somerset that of Coromandel in 1774 and 1780; while other observers have written of that in the Isle of Mauritius in 1775 and that of Calcutta in 1781.

[Sonnerat as long ago as 1768-71 described an

epidemic in the neighborhood of Pondicherry—Presidency of Madras—that destroyed 60,000 lives and was undoubtedly cholera. The malady prevailed also in what was then French India in 1780–81, and there are reports of it in Madras from the year 1774, '81 and '82, as well as earlier, and later accounts from other parts of the East Indies, all of which describe its exceeding fatality.—S.]

Dr. Levington, who was in Bengal at the time of the famous outbreak of cholera, asked a Chinese physician for information as to the disease, and the latter showed him a Chinese medical work entitled "Teching-Tchu Tching-Ching, printed in 1790, which describes the malady as follows:

"The *Ho Louang* comes on with a sudden pain in the heart and belly, accompanied by vomiting and 'rice-water' discharges; the patient is cold and lacks animal heat and there is headache and vertigo. When the disease attacks the heart the vomiting is the first symptom manifested; when it commences in the abdomen there are 'rice-water' discharges, coming on with great frequency; when the heart and intestines are both attacked at the same time, then vomiting and frequent stools are simultaneous. When the disease is intense the patient has spasms that, extending over the abdomen, promptly induce death."

Levington translated this in the month of June, 1817, when epidemic cholera prevailed throughout all the valley of the Ganges. The type of the disease then prevalent in India, as described by him, was as follows:

"The attack is sudden and obstinate. A man laid down at night feeling perfectly well; soon he felt a painful sensation that he could not attribute to any

general visceral lesion. I saw similar cases at Macao, in China; entire families would retire perfectly well at night, were suddenly attacked by the disease before morning, and, perhaps, all dead before the next day at noon. The first symptoms are soon succeeded at irregular intervals by ardent heat in the region of the stomach, vomiting, and frequent stools similar in character to a decoction of rice. Cramps in the fingers and limbs gradually invade the body. Finally, the muscles of the chest and abdomen complete the circle of spasmodic movements, and continue until vitality is entirely abolished by death. In the last period of the disease to be seen, the vomiting and spasms often cease, owing to the complete exhaustion of the physical forces. The observing physician may often predict an imminent attack of the cholera, by the stretched out and anxious depressed air of an individual supposed to be in a state of health. The changes experienced by the pulse and skin at different periods of the disease are very remarkable. The pulse at the start is rapid, small, feeble; during the paroxysms it becomes imperceptible in the limbs, or disappears some time before death. The circulation seems extinguished in the superficial tissues; the blood accumulates in the internal organs and congests them; these viscera are found gorged with thick black blood, which explains the alteration of the organs of respiration and the secretions. At the commencement of the disease the skin is pale, cold, and covered by a viscid sweat, resembling the feeling of a frog just out of the water. At later periods, in fatal cases, it becomes altogether cadaveric."

It was at Jessora, a town situated at forty leagues [Jessur: sixty-seven miles instead.—S.] north-west of

Calcutta, that cholera for the first time in its epidemic form appeared—on August 9th, 1817.

[Though the first real, world-famous pestilence dates from Jessur, in 1817, Von Hirsch has proven the existence of other cholera epidemics in India as early as 1816, and in the first months of 1817, so that it is probable that it found its way to the region northeast of Calcutta from the Northwestern Province. It was at Jessur, however, that it first began to excite the attention of the authorities on account of its general and wide-spread extent.—S.]

Dr. Tytler,* an Englishman, first observed it; he thought the first patient he saw was dead from poisoning. The disease was attributed then to unseasonable weather and bad rice. The cholera had already appeared, in the month of May, at Nudday, and spread over the country between Sillhet and Monghir, and from the mouth of the Ganges to its confluence with the Jumna. The natives were astonished at the manner in which it was propagated; it described a perfect circle around one province without entering its interior; then it subsided in such a way as led people to think it had exhausted its force. Suddenly it returned, several weeks, and even several months afterwards, and ravaged the whole interior of the country. It was noticed to go up and then descend again one of the branches of the Ganges, then suddenly to stop, cross the river and devastate the opposite bank.

Cholera was epidemic in Calcutta for the first

* "On Morbus Oryzeus," etc., Calcutta, 1820.—S.

time in September, 1817, but did not reach its acme until the following year. Two thousand persons died weekly in Calcutta out of a million of population.

On November 9th, 1817, cholera attacked the camp of the East India Company upon the right bank of the Betwa, [Sindh.—S.] traveling from the east to the west. It made such terrible ravages among the ten thousand English and eight thousand native troops that the vast majority only survived a few moments after being attacked; those who lived on vegetables died first. Strange to say, women and children escaped. The disease suddenly ceased when the army crossed the Betwa. Nine thousand soldiers died in the camp in ten days.*

The cholera then extended largely over almost all of India, ravaging successively the villages of Nagpur, Aurungabad, and Ponany, following the movements of troops.

Cholera struck Bombay on August 11th, 1818, and in six months 1,133 persons died of the disease. In the month of September, 1820, in the same city, following a heated term, 235 persons died in five days from the disease.

In March, 1818, it traveled—always from east to west—to Allahabad, at the confluence of the Jumna and Ganges; it was carried to Delhi, Jajpur, and into a camp of fifteen thousand troops; it carried off by preference the poorly nourished, and such domestic animals as camels, goats and dogs. The epidemic went back to the sources of the rivers entering into

* This is liable to misinterpretation. As a matter of fact some 8,000 Sepoys *and camp followers* died, but only 764 of the English soldiery.—S.

the Ganges; it thus spread to the western or Malabar coast and to the coast of Coromandel, and marched without stopping both north and south.

It was seen at Nellur in October, 1818; at Madras in January; at Pondicherry, Carwar and Bellary in June following. In January, 1819, it was noticed at Manaar island [off the coast of Ceylon, which latter island had been attacked the previous year.—S.] Its invasion was sudden and its irregular propagation seemed to have no connection with the variations of temperature. At the same time cholera appeared at Ceylon it struck towards the east, to Aracan, Malacca, Singapur, to the Islands of Penang and Java, and returned to these places in 1821, proving very murderous.

On October 18th, 1820, cholera attacked Canton and Manilla with great fury; it came on following a terrible storm; it afterwards visited [Cochin-China.—S.] the Celebes group, and extended to Amboyna and Macassar, where even cattle, monkeys and dogs perished from the malady.

In the month of February 1821, the effects were felt at Surat, then upon both banks of the Indus; then in Arabia, at Muscat, Mosul, Bender-Abbas, and Basra, [in which latter city nearly one-fourth the entire population succumbed to the malady.—S.]; from thence up the Euphrates, and at the end of August it had reached Ispahan, Shiraz and Baghdad, and slain ten thousand persons. Here it was given the name of *Haouwa*, which means "the tempest." The Persian army, camped between Baghdad and Kurdistan, lost two thousand men. Vessels at sea were devastated by the affliction.

At Colapur [Umrawutti District, India.—S.], sixty persons embarked to cross the stream and were

attacked on the trip; only three had the strength to reach the other shore; the rest died.

The disease was in such a violent form at Muscat that those attacked *often died in ten minutes*.

[It was especially destructive in Borneo and Java; the island of Java is said to have lost 100,000 inhabitants by its ravages, and Batavia alone over 17,000. In this year (1821) cholera covered a space of forty three degrees in latitude by seventy in longitude.—S.]

In 1822 the cholera reappeared in Java; at the same period it gained Mosul in August; Mardin in September; in October Karum; in November Beru, Aintab, and Aleppo, extending into Syria and up to the frontiers of Egypt.

[This same year India was again attacked, but the ravages were less severe in the islands. In Mesopotamia it appeared with renewed intensity, and Syria was soon seized upon. It spread also in Persia, and from Ispahan reached Kazan in July, Tauris in September, and soon after Erzerum.—S.]

On June 10th, 1823, it declared itself at Latakia, and on the 20th at Antioch; here, for the time being, it terminated its cruel excursion. [It also attacked Tripoli, in Syria, and in Palestine penetrated to the foot of Lebanon. In this year also, Orenburg, on the extreme boundary of Europe, was attacked.—S.] In the countries thus invaded death usually occurred in two hours. All human aid was useless; it was only towards the end of the epidemic that a certain number of patients were saved by copious bleedings, hot foot-baths and medicinal decoctions.

Towards the end of August, 1821, when the epidemic had devastated Baghdad, it grew murderous at Shiraz; persons walking along the roads would fall and expire as though struck by lightning, without having time to even complain of illness. Workmen died with their tools in their grasp, farmers fell at their plows, and priests died while at their prayers.

From Shiraz the disease went north, passing to Zenjan and Magen; from thence eastward to Yezd. It ceased to prevail with cold weather, but reappeared the following spring, ravaging Ainad, Kashan, Kum, Kurum, Susa, Khoi [all cities in Persia.—S.]

In summer it came to Tebris, where it prevailed until winter; but in the following spring struck the frontiers of Russia. In May it was at Serachs on June 17th at Lenkoran, on the Caspian Sea; it then returned to Kur and reached Baku, a town of thirteen thousand inhabitants, where there was a festival, with much dissipation; here thirteen persons died on the public square. Three dropped while conversing on the street, falling in convulsions, taken with nausea, cephalalgia and vomiting; these latter symptoms were more formidable than spasmodic accidents. Those individuals who fasted had most chance of recovery. Treatment was necessary at the very commencement of the attack; patients were stripped in the streets and submitted to massage and cold affusions; the limbs were subjected to strong frictions; the body and chest were rubbed as well as the cramped limbs. This massage was kept up for two or three hours, a dozen persons sometimes relieving each other over one patient. Fresh water was poured on the body; then the sick man was put to bed and made to take hot tea until sweating was induced, when the patient

was considered out of danger. For several days after the attack the convalescing sick were dieted.

In the month of September the cholera reached Astrakhan and Krasnoi-Yar, killing seven-tenths of the dogs; then it passed to Calcutta and Madras again, to the Island of Java, to Pekin and Han-Kow. The preceding year it had entered China as far as the Siberian frontiers. It reappeared in Orenburg in 1828.

[In 1824, '25, and '26 the malady made little progress. Says Lebert: "The two threatened divisions of the earth, Europe and Africa, had yet (for the most part) been spared, and toward the close of the year 1826 the hope was cherished the epidemic was near its end; but as early as the beginning of 1827 it appeared with renewed intensity in Calcutta, and here it is mentioned many animals showed the influence of the disease." In India it was a popular belief that the prevention of human sacrifices had excited the anger of the gods, and it is a fact worthy of record that the wife of a Hindu in Palcala resorted to every device to obtain permission to violate the law by being burned upon the funeral pyre of her husband, who had died of cholera. She maintained she had done the same thing four times already in previous states of existence, and that, if she might repeat it for the fifth time, the epidemic would cease within a fortnight. The consent of the rajah was finally obtained, but the ashes of the poor creature failed to stay the plague. The Coromandel coast suffered again, severely; Lahore, Kashgar, and Kabul

were ravaged, the disease spreading to the high mountains that constitute the foot-hills of the Himalayas, and thence to the regions about the Aral Sea. —S.]

In 1829 it passed over Persia again, and prevailed along the Caspian Sea, to Teheran, thence to the Provinces of Mazanderan and Shirwan; it appeared, too, at Tarsüs and in the Caucasus, penetrating to Tiflis. On August 8th, 1829, many religious ceremonies occurred at this place, which favored the propagation of the epidemic; out of a population of forty thousand, five-eighths perished. From Tiflis it went to the shores of the Caspian Sea, and reached Astrakhan again on July 31st, 1830, seven years after its first appearance there; the deaths in this Province were twenty-one thousand.

It traveled up to Volga and Don and the Emba and arrived at Moscow on September 28th, crossing from Nijni-Novgorod, having traveled over three hundred and fifty leagues in two months, and ravaging the Cossack country, the borders of the Black Sea and sea of Azov, as far as Taganrog, Sevastopol, Nikolaiiev, Kerson and Odessa. The following winter it traveled slowly along the banks of the Danube. Cholera devastated Moscow from September 28th to October 30th; there were 5,960 persons attacked, of whom 2,549 died.

In 1831 the cholera broke over the Government quarantines, traversing Transylvania and Hungary, from thence entering Poland and Galicia; it declared itself at Broda, a city of thirty thousand inhabitants, of whom twenty-four thousand were Jews; here seventeen hundred cases occurred and eight hundred patients died. In Galicia, from June 1st to August

8th, 86,687 persons fell sick with cholera, and of this number 24,600 died. In Hungary there were 19,175 cases and 8,266 deaths.

During the summer of this year the disease continued to travel north, arriving at St. Petersburg and Archangel, then descending on the coast of Finland.

It appeared in Prussia on August 18th, and Berlin and Stettin; from thence it went to Vienna, where it ravaged the suburbs before penetrating the city proper.

Finally, in 1832, the cholera crossed the Rhine, was carried to England, then threw itself with fury on Belgium and France. In Paris it lasted from March 22d, until the commencement of August, and killed 26,300 victims; thirty physicians contracted the disease, of whom eighteen died. In France, during this epidemic, 229,534 persons fell ill with the disease, of whom 94,626 died.

[Here may be left the history of M. Ozinam, and the tale completed by Lebert, who is much more thorough and accurate, and further, much more familiar with the ravages that overtook Europe.—S.]

The year 1830 witnessed a new phase in the dissemination of the epidemic which, from this time on, possessed the greatest interest for all Europe. It was now, relatively, not very severe in the English Indies and Eastern Asia, but its advance towards the north-east was more positive than ever before. It soon again extended to the coast of the Caspian Sea, and Orenburg, which had already been reached in the last months of 1829, was again attacked. Almost a tenth of the population was now seized with cholera, though the number of fatal cases—scarcely a fifth of these

attacked—was usually small. From this place it spread all over the neighborhood, and now the high places and mountain regions were very severely visited. In spite of isolation and quarantine the epidemic appeared in Nijni-Novgorod in the month of August. It had already been creating havoc in Persia since May, and now Teheran was seized upon, then Tauris again, where it was exceedingly severe. From Tauris it soon reached Tiflis, where, although the majority of the population had fled, the fourth part of those that remained fell victims; I may say however, that I do not attach much importance to any of the numbers given.

The Caucasian mountains now no longer formed a wall against the progress of the disease, as it attacked the higher regions, overleaping the mountain chains in several places, and as a general thing followed the course of streams. Towards the middle of July it again traveled from the Caspian Sea to Astrakhan, where it was not only much more destructive than before, but showed again a definite tendency to spread in the direction of Europe. It now followed the regions along the Volga, over a wide extent of territory, and in a few months reached 130 leagues northwest of Astrakhan. Kazan was now soon, but lightly, attacked. The epidemic spread also along the tributaries of the Volga. Besides this northern excursion, a western one soon showed itself. The territory of the Don was seized upon. The pest also reached Azov and Saratov, where many chickens perished also, in their yards. Somewhat later the disease visited the coasts of the Azov and Black Seas; among other cities here, Sevastopol and Odessa were attacked. The cholera now followed up the Dneiper, reaching Nijni-Novgorod and Kiev in October, and

extending in November to Podolia and Volhynia. From still another side it penetrated into the heart of Russia. From Saratov it advanced, after again over-leaping the sanitary regulation lines, to Moscow, where it raged with great intensity from October to April of the next year. The destruction was exceedingly great in this city, but it was considerably lessened by the presence of the Czar, who came over from St. Petersburg and visited the patients in person, providing proper arrangements for their care and comfort.

In 1831 this fearful pestilence excited still further the apprehensions of Europe. It first appeared again, however, in the spring in the Orient, especially in Mecca and Medina, and raged with great violence. Over 100,000 pilgrims were assembled in the two cities when the disease broke out, and here again we observe that all crowds, under these circumstances, are particularly dangerous. Soon afterwards the cholera showed itself in Syria, and Egypt; it appeared in Alexandria, but it was especially in Cairo that it created unheard of destruction; 30,000 people are said to have perished here in the first months. Though Ibrahim Pasha believed himself safe by having surrounded his court and harem with a triple *cordon*, many of the inmates were attacked. In Egypt the disease penetrated up the Nile to the higher regions of the country. Constantinople was also attacked this year, though but lightly, while Smyrna was seized upon most savagely.

In Russia, the pestilence still raged, not only in the south, but also in the north, as far as Archangel. Finland, Esthonia, and Livonia were visited, and St. Petersburg attacked with especial severity. Notwithstanding the presence of the Czar in the capital, a

popular revolt was excited, caused really by senseless restrictive regulations, but was soon quieted. The central region of the Russian Empire was also sharply seized upon, and from the south the pestilence spread out westward in two lines, south-westwardly towards Moldavia and north-westwardly towards Poland. In the first mentioned land Jassy in particular was severely attacked during the summer. But the north-western extension of the disease was far more dangerous. It stretched out southwardly from the borders of Poland towards Galicia, where Broda and Lemberg suffered severely. To the north it reached Brzesc and Grodno. Warsaw was now soon visited, and the Prussian border was over-stepped for the first time at Kalisch. At Cracow there were many victims. Prussia was now attacked from the other side. From Riga, where no less than one-twenty-sixth of the population perished, it passed on to Mitau; soon it showed itself in Dantzig. From this place it entered Elbing, Marienburg, Tilsit, and Königsberg. From Poland it passed to Silesia and to the neighboring countries, Bohemia and Moravia. Following the course of the Oder it reached Küstrin and Frankfort, and now spreading itself out over the regions of the Havel and Spree, on August 30th entered Berlin. I was myself present during the entire four months of the stay in this city. The mortality was not very severe, in all some 2,500 among 200,000 inhabitants. Fear of the disease was not great in Berlin, on account of the cheerful disposition of the people. Caricatures were even published with incredible frivolity in ridicule of the hypochondriacs who could not collect together sufficient protectives against the disease. It was at that time that there appeared the well known cartoon upon Rust, the contagionist, who had at-

tempted to secure the isolation of the sick by a great sanitary *cordon*.

The cholera disappeared from Berlin at the end of the year 1831. It was observed in North Germany, at that time, that chickens and pigeons, and in many rivers fishes, perished in great numbers. It was an interesting fact that the greatest number of persons were attacked on Tuesday, the least on Saturday, which is clearly to be attributed to the Monday excesses of the laboring classes. In October of the same year the disease appeared in Breslau, and soon spread all over Silesia. It raged exceedingly severely in Hungary, and Vienna suffered also in this year from a widespread though not very fatal epidemic. The course of the disease along the territory of the Elbe attracted attention. Among the cities here attacked were, successively, Magdeburg and Hamburg. The more western extension took its departure chiefly from Vienna. In North Germany, although the disease was very widespread, it shared the territory along the course of the river. The Scandinavian countries were perfectly exempt from attack, excepting a light epidemic in Stockholm, which reached the city through Finland in the westward march of the disease from St. Petersburg. From Hamburg it overleaped the sea, and on Oct. 26th, 1831, reached Sunderland, on the east coast of England. Soon now it spread itself over the islands of Great Britain. London was attacked in January, Edinburgh in February, and Dublin in March, 1832. From England it was carried over to France, just as later, in 1849 and 1853. In March, 1832, it showed itself in Calais and Paris. It excited the greatest terror in Paris, where it raged with extraordinary intensity, destroying about one-fourty-third of the whole population. Here, too, it

excited a dangerous popular revolt. The people complained here, as in many other places, that the wells had been poisoned, and this caused the greatest mortality. From Paris the disease spread out northwardly. On the northwest it passed into Brittany, some 120 hours' journey distant from Paris, while its southern radiation scarcely exceeded 30. A new phase in the epidemic presented itself towards the middle of the year 1832, when the cholera crossed the Atlantic Ocean and appeared in North America, with especial violence in Canada. It was very severe in Quebec and Montreal. New York was attacked as early as July, and shortly thereafter Philadelphia and Baltimore in succession. The epidemic ran through the country as far as New Orleans, which was reached in November, 1832, and in the summer of 1833 it invaded the Mexican States. Here it was most severe in the cities of Vera Cruz and Mexico. This epidemic, many particulars of which I have received from several practitioners, was not only very destructive, but was most remarkable from the fact it reached a city higher above the level of the sea than the highest Alpine pass in Europe. The epidemic had almost ceased in Europe at this time, when it again broke out in June, 1833, on the coast of Portugal, where Lisbon was visited with especial severity. In North America, the northwestern march of the disease passed over 3,000 or 4,000 miles. The leap from Mexico to Portugal was somewhat after the manner as if it were about to return to its first point of departure, in an opposite direction to the first ellipse about the equator. In the winter of 1833, and in the year 1834, cholera prevailed in Portugal and Spain, in which countries the interior was first attacked, and afterward the coasts. In Madrid, Seville, and Bar-

celona it was especially severe. Spreading now eastwardly from the coasts of Spain, it attacked Cette, Marseilles, Toulon, Nice, and Genoa. From this place it entered Lombardy and Piedmont, attacking such places as Coni and Turin, finally reaching Leghorn, Florence, Trieste, etc. In 1835 it passed over from Spain into North Africa. A large part of the Mediterranean coast was attacked. None of the islands suffered but Malta, where one-fortieth of the population perished.

In the years 1836 and 1837 the disease prevailed again in Italy, especially in Naples and Rome. In 1836 Munich was also attacked, and South Bavaria and Tyrol severely. In the summer of 1837 it again entered Berlin, Breslau, and many places in East Germany. In October, 1837, it prevailed in Algiers over a pretty wide extent. With this year ended this great, almost world-wide epidemic, which in 1830 had manifested a renewed intensity. But in all these twenty years we observed, on the one hand, that it mostly extended in a north-westerly direction, and, on the other, that it was continually breaking out with renewed intensity at its centre and point of departure in the East Indies, when its extension would become still greater.

But this interruption in the prevalence of the disease did not last long, and perhaps even there was in reality no interruption. As early as the beginning of the fourth decade, we see the cholera again severe in India, especially along the courses of the Ganges and Sindh. From here it again reaches Kabul and Bokhara, and appeared as early as September, 1845, in Samarkand. In 1845 it prevailed severely in all Persia, spreading especially from the east to the west. Thus it traveled from Meshed to Teheran and Bagh-

dad. In Teheran, a city of 130,000 inhabitants, 7,000 are said to have perished. From Baghdad it traveled northwardly along the courses of the Tigris and Euphrates; we see it again passing southward to reach Basra. A great part of Arabia was next attacked. In Mecca and Medina it appeared again in November, 1846, and again caused extraordinary destruction among the assembled pilgrims. Up to this time the cholera had travelled from Meshed to Mecca, about 625 leagues, in ten months, or something like 55 leagues in a month. In the two last months of 1846 it traveled over a distance of 120 leagues, again overleaping the Caucasus to appear in Astrakhan. From this time on, it followed the course of the Volga, until it reached the north of Russia. In another line of extension it went out from the east towards the west, particularly along the Kur, penetrated Georgia, reached Trebizond, and on October 24th, 1847, broke out in Constantinople, which now became a chief centre of radiation. From Constantinople, all Syria and a large part of the countries about the Mediterranean Sea, were attacked. Roumania, also Wallachia and Bulgaria, were severely visited. To the north it again entered Russia, raging as a most violent epidemic, most especially in Moscow. Poland was now seized again, and the bordering provinces of Prussia, as well as Galicia, Bohemia, and Hungary, from another direction; then a large part of North Germany, especially Berlin, where it prevailed in 1848 with extraordinary intensity. Following the course of the Elbe, it again passed through Hamburg and from there over the sea to England, particularly to London, and thence to France. It broke out in Paris on March 11th, 1849, after it had previously appeared in a poor-house in St. Denis. I

happened to be at the Hotel-Dieu when the first patient was brought in, presenting already a perfect picture of cholera asphyxia. The disease lasted here up to the end of the year, a period of about nine months. At first it appeared slowly, attacking rather the poorer classes and debilitated persons; then it gradually seized upon all classes of people until it reached a hitherto unheard-of intensity, particularly during the first eight days of June. The number of dead daily numbered between 700 and 900. General terror was excited over the whole city. Hearses no longer sufficed to carry corpses to the cemeteries, and transport vehicles of all kinds, even artillery munition wagons, had to be impressed for service. At the same time the heat was oppressive, while the weather was clear and beautiful. The greatest contrast was exhibited between the profound desolation on the one hand and the extreme frivolity on the other; often while returning home at night or early morning, deeply depressed by scenes among the sick and dying, I would meet parties on the way, pale and reeling from the excess of their nightly orgies. On June 9th, a severe storm occurred, and from this day on the number of sick and dead markedly diminished, so that the fearful epidemic was almost forgotten during the political excitement which prevailed in the riot of June 13th. Still, lighter forms of the epidemic continued to recur quite frequently. Some 10,000 inhabitants in all fell victims to the disease. Many of the patients operated on in the hospitals died of cholera. In several localities the intensity of the disease was most strongly marked; that experienced in the Salpêtrière, the hospital for aged females, was almost unknown in the past history of the epidemic. Of the 5,000 inhabitants of this great institution,

about 1,200 perished with cholera, and during the first three months this locality furnished almost one-fourth of the cases attacked, and one-third of the whole mortality. This local epidemic left far behind it that of Wall St., in Berlin, in 1848, which furnished only one-twentieth of all the fatal cases.

A large part of France was now attacked, and the disease prevailed in almost all Middle Europe in this year, during which it again reached America (towards the close of the year 1848), entering, not at the North as before, but at New Orleans, whence it spread in various directions.

This second great epidemic presented a somewhat similar course to the first, but with manifold differences in the details and modes of its dissemination. The ellipse of its whole course covered, in the same direction, a wider space. Yet there escaped, in their central parts, Savoy, a part of the mountains of Tyrol, and, to a considerable extent, the course of the Rhine, the Rhone, and the Isere, especially in their upper regions. Immunity from the disease was limited, however, to a much smaller number of places, as compared with previous epidemics; this was particularly the case with that of 1854.

This second epidemic passed almost without interruption into the next. Before the end of the year 1850 it showed itself again in Persia, but we do not know whether or not it reached that country again from India. In 1851 and 1852 it produced the greatest destruction in this part of Asia, extending, as had both preceding epidemics, to the southern range of the Caucasus. The regions of the Black and Caspian Seas, and the cities of Moscow and St. Petersburg, were now again attacked in succession. The territories of the Volga and Dnieper suffered

severely. Towards the end of the year 1852 and the beginning of 1853, Poland, the neighboring Prussian provinces, and later Berlin and Hamburg, were again attacked. To the north, the disease spread as far as Archangel. The Scandinavian countries were seized with great intensity. England and, later, France, were attacked in 1853, the latter especially in 1854 and 1855, over a wide extent. The coasts of the Mediterranean Sea, Marseilles, Genoa, and many other points, the Greek coast especially, were all seats of the disease. In South Germany, it prevailed in those countries in which it had manifested itself in 1836. In Munich and Augsburg it was especially severe.

The cholera now entered England again in 1853: but it was not until 1854 that it prevailed to a wide extent in England, Scotland, and Ireland. In 1853 and 1854 it extended over a considerable portion of the United States of America and the Antilles. In 1854 and 1855 it showed itself in Switzerland. Its spread and desolation during the Crimean war are well remembered. South America also, spared up to this time, was now severely visited in 1855, in Brazil.

It is most astonishing that the opinion should be generally accepted that Switzerland had escaped attack up to the year 1854; the fact is, the Canton of Tessin was long ago the seat of the disease. Cholera first appeared in Lugano and Mendrisio and their vicinities in July, 1836, having crept in from the province of Como; but it did not pass over Mont Cenis. Quite the same conditions prevailed in 1849, while in 1854 and 1855 Magadino, on Lake Maggiore, and Cadenazzo, places on the other side of the mountain were attacked. It is remarkable that while the disease was transported from Genoa to Tessin in

1854, nothing similar happened to the cholera fugitives who came into the valley of the Rhone over the Simplon pass, and the light Geneva epidemic, mentioned later, was carried over Mont Cenis. The Tessin epidemics lasted, on the average, about three months, but were never very marked. The first really great epidemic in Switzerland was that of 1854 in Aarau, where it first broke out in the poor-house and then rapidly spread over the city. It is probable that it was imported from Munich, where it was raging, as well as in Augsburg, in 1854, with great intensity. I observed a light local epidemic in Zürich in 1854; but the great epidemic did not occur until the following year, when the disease, having been imported, probably from Alsace, appeared in epidemic form during the spring in Basle, and also in the canton of Baselland. That portion of the city of Basle situated below the general level was especially attacked, while in Zürich, later, it was the higher regions of the city which were the chief seats of the disease. In both cities the epidemic lasted about ten weeks and in the country regions six weeks. A light local epidemic showed itself also in Geneva in August and September, 1855; it was probably imported from France and attacked in all but ninety-two persons. Among the greatest of the Swiss epidemics is that of 1867, which has been excellently described by Zehnder, and concerning which we are indebted to Biermer for some very valuable communications. Strange to say, a light, inconsequential epidemic appeared also in the village of Branson, opposite Martigny, in the Canton of Valais.

In later years cholera has broken out in many places in Middle and Northern Europe, and has become even epidemic in some cities, as Warsaw and

Königsberg, but never assumed the pandemic character which we observed in the first twenty-five years of the disease in Europe, and which we noticed for the last time in 1866, during the German-Austrian war, when almost more Prussian soldiers were killed by cholera than by battles. Breslau, a city so often visited by cholera, never had so murderous an epidemic as that of 1866, in which—aside from all the lighter cases, and those of cholérine—something like the twenty-fifth part of the whole population was attacked, and that with a mortality of over fifty per cent.

A retrospective view of the course of the disease up to the present time, teaches us that most the different parts of the earth's surface have been reached by the disease in its pandemic form, and that the islands, lands and countries hitherto exempt, such as for instance a part of the coast of West Africa, the Polynesian Archipelago, a part of North America, some of the northern countries of Europe—Lapland, Iceland, etc.—owe their immunity, partly to their comparatively isolation, and partly to accident. On the other hand, it is quite possible that favorable conditions of soil and drinking-water may oppose great obstacles to the development of cholera germs in different places. The constant escape of certain cities and regions during the last four decades, even in the midst of great destructive epidemics, is a strong argument in favor of this view.

[Thus Polish Lissa,, a great railroad junction, has always remained free from cholera; its water, which is of very good quality, is brought from outside the city through sound pipes; even imported cases have never extended the disease at this place.

Laubau is also supplied with pure spring water through iron pipes from without the city, and although a number of epidemics have occurred in the vicinity, it has always enjoyed immunity from cholera. The same is true of Pless, notwithstanding its marshy surroundings. Neumarkt, Groëenberg and Glogaü owe their constant escape to the same cause. In the latter, the parts of the town supplied with pipes remained free from the epidemic even after cholera had been imported, while the part of the city on the right bank of the Oder, which is supplied with water from wells, lost one and one-half per cent of its population in the epidemic of 1866. The same is true also of Jauer, whose upper eastern portion, supplied with excellent wells sunk deep into the rock, escaped attack, while the western, low-lying parts, supplied by bad shallow wells, suffered much from the disease. Zobten, a town very near Breslau, likewise escaped; it is but poorly supplied with water, as many of the wells, only 20 or 30 feet deep, are bored out of the solid rock and dried up during the summer months. Tarnowitz, also, even after the importation of cases, has always remained free from cholera. — S.]

APPENDIX B.

Attention has been called to a publication of the Tennessee State Board of Health—a concise chronology of the various epidemics of cholera and their course,—which seems worthy of reproduction:

ASIATIC CHOLERA IN EUROPE AND AMERICA.

1629.—Bontius, a Dutch physician at Batavia, described the disease and first made it known to the medical profession in Europe.

1817.—It raged with great violence at Jessur (Bengal), India, whence it spread, not very swiftly, but with great certainty, in all directions.

1818.—By August it had reached Bombay*. Thence it traveled through Arabia, Persia, Mesopotamia, Syria, etc., on its westward course; and, continuing to extend itself eastwardly from its place of origin, invaded Burma, Siam, Java, Borneo, the Phillipines, China, and other populous countries of that portion of the globe.

1823.—It appeared at Orenburg and Astrakhan, Central Asia, and on the eastern frontier of Russia.

1828.—Remained here until this year, when it increased in violence, attacking a tenth of the inhabitants of the Province of Orenburg, proving fatal to a fourth of those affected.

1830.—Reappeared at Astrakhan. In less than a month 4,000 persons died of it in that city, and over 21,000 in the Province.

* And the Northwest Province.—S.

1831, June 26th.—Appeared at St. Petersburg, having ascended the Volga and destroyed thousands in Moscow. From Astrakhan it also diverged along the northern coast of the Black Sea, and thence spread into Austria, Poland, Prussia and North Germany.

1831.—In August it was conveyed to Cairo by a caravan from Mecca. Over 15,000 died of it.

1831, October 26th.—It appeared for the first time in Sunderland, England, whence it spread slowly through the northern part of the island into Scotland.

1832, February 14th.—It broke out in London.

1832, June 8th.—The cholera broke out at Quebec, its first appearance in America. Two days afterward it was in Montreal.

1832, June 24th.—New York was attacked; thence it spread to Albany, Philadelphia, Cincinnati, New Orleans, etc. In New York it reached its height on July 21st.

1836.—It lingered in the United States for four years, and then entirely ceased. This first epidemic of cholera cost Great Britain and Ireland 40,000 lives out of 116,000 persons attacked. In the cities of Quebec, Montreal, New York, and Philadelphia, embracing then about 450,000 inhabitants, there were over 18,000 cases and 8,000 deaths. In India it remained endemic. Other Asiatic countries also suffered severely.

1846.—It appeared at Karatschi early this year, near the mouth of the Indus, with terrific violence. Thence to Teheran, capital of Persia, where its severity was such that 300 perished daily, for several weeks, in a population of not more than 60,000.

1847 and 1848.—Cholera ravaged parts of Russia and Turkey, having entered Europe by almost the

identical route as before. It traveled, however, with much greater rapidity.

1848.—In the autumn it appeared in France and Great Britain, revisiting during the next eight months, with almost unerring certainty, every place in which it had appeared in 1832-33, and seeking out the same filthy lanes and undrained sections of the cities where it had then committed its greatest ravages. It was even more malignant than in its previous visit. In England and Wales it carried off 53,293 persons.

1848, December 4th.—The barque *New York* from Havre, arrived at Staten Island, N. Y., with cholera among her passengers.

1849.—It occurred in New York. The whole number of cases reported outside the hospitals, in fifty-two days, was 2,631, of which 915 died. Also in New Orleans, and spread over the greater part of the Eastern and Western States.

1850.—In New Orleans, deaths from cholera, May to December, inclusive, 824. Cases occurred as late as February 15th, 1851.

1850.—At Cincinnati, from June 1st to August 15th, 1,400 deaths from cholera. At Columbus, Ohio, from July 24th to August 15th, 195 deaths from cholera—a great mortality for the population.

1851.—A second visitation at Cincinnati. Some 200 deaths, mainly in July.

1851.—From April to August, inclusive, 766 deaths from cholera in St. Louis. Total for the year, 847.

1852, May, June, and July.—Numerous cases in Cincinnati.

1852.—Total deaths in St. Louis for the year 789, of which 508 occurred in June and July.

1854.—Cholera as virulent in St. Louis as it was

in 1849. Total deaths 1,543, mainly in May, June, July, and August.

1855.—Disappeared from the United States.

1853, 1854.—Prevailed in Great Britain.

1855, 1856.—The allied armies in the Crimea suffered intensely.

1865–1874.—Cholera persisted in Europe about ten years.

1865.—In the beginning of May it broke out with terrible fury among the pilgrims at Mecca. On the tenth or eleventh of May the first death occurred at Alexandria. In June it had reached Cairo. On July 3rd, at Constantinople, where it produced a terrible panic. From Alexandria a steamer conveyed it to Marseilles. Thence travelers carried it to Paris.

1865, September.—Several cases in Southampton, England. Did not spread.

1865, November 3d.—Steamship *Atalanta* came into the lower bay of New York with 400 German immigrants, and cholera. Precautions taken; no spread.

1866, July 7th.—At Ancona in Italy, from Alexandria.

1865.—Great epidemic at Valencia, in Spain. Thirty-one out of forty-nine provinces in Spain were ravaged from July till the close of the year. It extended also into Portugal.

1866.—Cholera was early reproduced in almost all the localities it had visited in 1865. It extended northward as far as St. Petersburg. It appeared in several localities in Bavaria, Saxony, and Prussia, also in Belgium and Holland. It still existed in Paris and extended to the northwest of France.

1866.—An epidemic in Liverpool from July 22d, to the end of November carried off 1,792 victims. In

London for the three weeks ending August 4th, the deaths were 3,481, 1,097, 1,178. More or less diffused over England during the summer.

1866.—It broke out in New York about the beginning of May, and gradually spread over the country, following the lines of travel. Prevailed extensively in the United States Army, causing over 1,200 deaths among officers and men. During summer and fall prevailed extensively at New Orleans, and at St. Louis also.

1867.—A general abatement in Europe. Prevalent in South America. Buenos Ayres suffered greatly.

1867.—At New Orleans, reappeared in June; 571 deaths the following six months. Again at St. Louis during summer and fall.

1868.—Completely died out in Europe.

1869.—By its old route it reached Nijni-Novgorod, and broke out in September.

1870.—A vast outburst of cholera. In Russia, 9,386 deaths.

1871.—In Russia, 124,834 deaths.

1872.—In Russia, 113,196 deaths.

1873.—In Russia, 4,395 deaths.

1872.—Very widely diffused over Europe. Imported into England on several occasions. Its spread stopped by the local sanitary authorities.

1873.—Began to subside in Europe.

1872, December, and 1873, January. — There arrived at New Orleans a total of nearly two thousand immigrants from cholera-infected districts of Europe.

1873, February 9th.—First death at New Orleans. Two hundred and fifty-nine fatal cases occurred during the epidemic.

1873, April 8th.—First case, fatal, at Vicksburg.

1873, June 30th.—First case, fatal, at Little Rock. Four importations; no spread, owing to the energy and efficiency of the medical men in whose care the initial cases occurred.

1873, April 15th.—First case, fatal, at Memphis.

1873, May 24th.—First case, fatal, at Chicago. Total number of deaths from cholera and cholera morbus, May and September, 116. Many towns and villages suffered greatly.

1873.—First case at St. Louis, died May 11th. A mild epidemic followed. Other localities visited.

1873.—First case at Paducah, died May 21st. Very widely diffused throughout Kentucky.

1873, June 15th.—First death reported at Cincinnati. Two hundred and seven deaths during the summer. Other cities and towns in Ohio visited.

1873, June 6th.—First death at Evansville. Other localities in Indiana visited.

1873.—During June and July 62 deaths at Huntsville, Ala. Birmingham, with about three thousand inhabitants, was terribly scourged during June and July.

1873, June 15th.—First case, fatal, at Wheeling.

1873.—But two authenticated cases of cholera occurred in the State of Georgia. Both were residents of and refugees from Chattanooga. One died at Atlanta, population 22,000, on July 2d; the other, at Dalton, population 5,000, on July 3d. Both instances terminated fatally in communities in which the auxiliaries to the rapid development of a cholera epidemic were present, the specific causes once having been imported; yet in both instances, by the prompt and energetic action of the medical men having the cases in charge, the power of the disease was confined to the infected individual, and the health of

the residents of the respective houses and of each community was efficiently guarded.

1873.—During this year some two hundred cities and towns in the Mississippi valley were more or less afflicted.*

1882.—Made its appearance in Egypt, where, in three or four months it occasioned a mortality of 30,000 to 50,000 of the inhabitants.

1884.—On June 13th or 14th it invaded the French military post, Toulon. Then the cities of Toulon and Marseilles, and spread through the south and south-east of France, and partly in central and western France.

1885.—At Marseilles and Bretagne.

1884.—About August, in Spain.

1885.—Invaded almost the whole of Spain.

1884.—Brought into Italy.

1885.—Great ravages at Palermo, Sicily.

1885-6.—At Venice.

1886.—From April to the end of the year it ravaged the peninsula of Italy.

1886.—At Trieste, and also the Austro-Hungarian shores of the Adriatic.

1887.—Again in Sicily and in Italy.

1884-1887.—The epidemic of cholera in Europe cost France 15,000 inhabitants in 1884, 1885, and 1886; Spain, 180,000 inhabitants in 1884 and 1885; Austria-Hungary, 4,000 inhabitants in 1886; Italy, about 50,000 inhabitants in 1884, 1885, 1886, and 1887; Malta, 500 inhabitants in 1887—a sum approximately of 250,000 inhabitants of Europe. In other words, the epidemic has removed from France about one inhabitant for every 3,000, from Italy one inhabi-

* See "Public Health," vol. i, pp. 234-252.

tant for 550 or 600, from Spain one inhabitant for every 100, from Austria-Hungary one inhabitant for every 9,000. An approximate calculation of these losses, estimated from the purely material point of view, shows a sum total of about \$80,000,000 of value destroyed. A still greater loss resulting from the damages caused by the disease through idleness, interference with commerce and navigation, interruption of business, etc., would increase the sum total of the losses occasioned by the cholera to about \$200,000,000 in three or four years.*

1886.—Cholera introduced into the city of Buenos Ayres, and to the Argentine Republic, in November, by the ship *Perseo*, plying between that city and Genoa. A conspicuous instance of official pride and stupidity. An extensive epidemic developed, and the disease spread through the inland provinces. The city was cut off entirely from the commercial world; Banda Oriental, Brazil, Paraguay, and most of the European ports, quarantined against it.

1887, January 19th.—Cholera officially declared at Montevideo, Banda Oriental, after many denials of its existence.

1887, January 2d.—Cholera at San Felipe, a town situated at the base of the Andes, 40 miles north of Santiago. The latter city severely scourged. Commerce of Chili interrupted, with heavy losses.

1887, September 23d.—The steamship *Alesia* arrived at New York from Marseilles with cholera on board. At Naples some 600 emigrants from the cholera districts of Italy and Sicily were taken aboard. Proper precautions used by the quarantine

* See Report of E. O. Shakespeare, M.D., United States Commissioner.

officials, and the disease not allowed to spread. Much credit claimed, and justly, considering their limited means.

However, the case of the Italian steamship *Indépendenté*, which arrived at New York, in October, with a large number of immigrants, and, not showing cholera on board, was allowed to discharge her passengers after a few hours of detention necessary for a thorough inspection, shows the insufficiency of this New York safeguard. The next day numerous squads of these immigrants, with their baggage, departed for at least twelve great cities in widely distant parts of the country.

CHOLERA IN EUROPE IN 1892.

1892, June 27th.—At Baku, the Russian port on the Caspian, 48 new cases and 38 deaths. The town in a deplorably filthy condition and without the least pretense to sanitary arrangement.

June 30th.—Rome. Five cases have occurred in Italy.

July 1st.—The administration of the towns in the Asiatic provinces of Russia taking energetic measures to prevent its spread.

July 1st.—Many cases reported in the outskirts of Paris.

July 6th.—Saratov on the Volga scourged.

July 8th.—Panic in Astrakhan.

July 11th.—In Paris 14 deaths.

July 14th.—Terrible in Astrakhan.

July 17th to 21st.—Russian official returns announce 4,839 cases and 2,590 deaths for this period.

July 23d.—Advance toward the Russian frontier. Absorbs public attention in Berlin.

July 23d.—At Nijni-Novgorod and Moscow. Expected at St. Petersburg.

August 5th, 6th.—Cholera returns for all Russia these two days show a total of 6,741 new cases and 3,496 deaths. Prior to August 1st, total deaths, 23,919 (official).

August 1st to 12th.—In St. Petersburg 154 cases and 31 deaths between these dates officially admitted.

August 11th.—In Northern and Central Russia increasing. In Moscow many factories closed.

August 13th.—Returns for Russia this day show a large increase in new cases and mortality.

August 15th.—Total number of new cases reported in Russia, 7,600; deaths, 3,900. Two-thirds of the towns attacked can make no reports for lack of telegraphic connections.

APPENDIX C.

THE RECENT EPIDEMIC AT HAMBURG.

BY PRIVY COUNCILOR MAX VON PETTENKOFER.*

The outbreak of cholera in Hamburg in August, 1892, naturally excited all Germany. The alarm—which the explosive occurrence of the disease in the chief commercial city of Germany, hitherto regarded as the type of a rational drainage system—spread throughout Europe and beyond it, and was not less than that of sixty years ago when cholera first advanced into Russia from Asia. It is excusable that in 1892, as in 1831, physicians and governing bodies thought first of all how to prevent the further progress of the destroying angel—how to localize it at Hamburg.

The rigid regulations in force throughout Germany to insure this limitation, may be compared with the military cordons and others measures of detention and isolation in vogue sixty years ago. They are based on the belief that cholera is simply an infectious or contagious disease, passing from the sick and their excreta to the healthy; and that the virus can only be taken with the food, and especially in water. This is now by many deemed to be absolutely proved since Koch claimed the discovery of the comma bacillus. The strife against the bacillus is held to be the only real prophylaxis, to the ignoring of the great mass of

* Abstract of a paper contributed by Prof. Von Pettenkofer to the *Munchener Medicinische Wochenschrift*, Nov. 15th, 1892. Reproduced from the *Scientific American Supplement*.

epidemiological facts which are entirely opposed to the mere contagionist view of cholera. Many confine themselves to the behavior of comma bacilli in test-tube or on plate, and do not trouble themselves at all about the behavior of cholera in its epidemic extension. Many years ago I declared the ætiology of cholera appeared to me as an equation with three unknown quantities— X , Y , and Z . Let X be a specific germ disseminated by human intercourse; Y something which depends on place or time, the “local disposition;” and Z the individual disposition met with in all infectious diseases, both the directly infectious, as syphilis and small-pox, and others, as typhoid fever and malaria. The contagionists have eliminated the Y , finding a sufficient explanation in Koch’s discovery of the X , and seeing in individual tendency or absence of immunity the factor Z ; so that if Z be granted, cholera must occur should people introduce the bacilli into the mouth by unwashed hands, or take them into the stomach with water and food. The view is simple and easy, sufficient for him who only concerns himself with individual cases, but it does not satisfy the epidemiologist; for the latter knows that there are not only cholera-immune people, but also cholera-immune places, and that even in places where cholera has prevailed there are seasons when it will not spread, although introduced. This is what I mean by the Y . It is not easy to define as is the X , and so far one can only say that it is related to the quality and dampness of soil.

Man alone of all living creatures is markedly susceptible to the cholera virus, and therefore experiments on animals with comma bacilli can determine nothing. The effects on the guinea-pigs—previously prepared by the administration of soda solution—of

an injection of a culture of bacilli, followed by one of laudanum, or the results of the intra-peritoneal injection of fresh cultures, are of no manifest importance as against the fact, confirmed a thousand times, that epidemics of cholera are never accompanied by epizootics. Now and then it has been noted that cholera has coincided with undue fatality among cats or poultry, but the association in the main has been purely accidental. Guinea-pigs did not suffer during the late outbreak at Hamburg. Similar experiments on animals with non-pathogenic fungi—*e.g.*, the bacterium coli commune—are fatal, and the bacteria multiply in the body just like the comma bacilli. So that the only indisputable experiments on infection with comma bacilli are those made on man.

Now as Munich, in the year of grace 1892, in spite of much travel of persons from Hamburg and Paris, and in spite of its October fair, remained free from cholera, I did not scruple to experiment on myself with the comma bacillus. Of it I had received from Hamburg, through my colleague, Dr. Gaffky, a pure agar culture, and from this my junior colleagues, Drs. Pfeiffer and Eisenlohr, prepared a sufficient quantity of broth culture to be taken by the mouth. As Gruber found that fresh cultures acted on guinea-pigs more powerfully than cultures several days old, I employed a broth culture which had been in the incubator barely twenty-four hours. Fifteen minims of this was found to contain innumerable bacilli after being diluted a thousandfold, so that I could take at one dose many billions of bacilli, very many more than one could possibly introduce by unwashed hands. Since Koch states that comma bacilli are destroyed by the acid of the gastric juice, I was careful to take them on an empty stomach—*viz.*, two hours and a

quarter after my "fruhstuck"—when, according to my friend, the physiologist Carl von Voit, there would not be more than three and a half ounces of gastric juice with 0.2 per cent. of hydrochloric acid in the stomach. To neutralize this free acid the broth culture of bacilli (fifteen minims) were taken in three and a half ounces of water, containing fifteen grains of bicarbonate of soda. The vessel was afterward rinsed with two ounces of water so as to insure my taking all the bacilli. I drank this cholera mixture in the presence of witnesses on Oct. 7th, it tasting like very pure water. Some were anxious about me, and begged I would allow them to sacrifice themselves for their old teacher, but I wished to act on the old medical principle, *fiat experimentum in corpore vili*. I was right in regarding myself as a *corpus vile*. I am seventy-four years old, have had glycosuria for years, have not a single tooth in my head, and only use my artificial teeth when I have to make a speech, not needing them for mastication; and I also feel other burdens of advancing age. Even if I had deceived myself, and the experiment had endangered my life, I should face death calmly, for it would not be as a thoughtless or cowardly suicide. I should die in the cause of science, like a soldier on the field of honor. Health and life, as I have often said, are very great earthly gifts, but not the highest. He who wishes to rank higher than the brute must be ready to sacrifice even life and health for great ideals. However, to me the matter did not seem quite so tragic, for I was firmly convinced my *X* could not kill without my *Y*.

[Professor Von Pettenkofer here gives a detailed account of his condition, *de die in diem*, as to tempera-

ture, pulse, sleep, food, intestinal symptoms, etc. On the 9th he began to have diarrhœa, and did not feel very well, having some abdominal discomfort; the diarrhœa increased on the 10th and continued up to the 14th. He took no medicine to control the diarrhœa, although advised to do so lest it should become chronic.]

The motions were examined bacteriologically by Drs. Pfeiffer and Eisenlohr to trace the fate of the comma bacilli. The first loose motion contained a large quantity, and the subsequent watery stools contained pure cultures of the bacilli. On Oct. 14th, there were only a few isolated bacilli, and by the 16th these had disappeared. Bacteriologists generally admit that comma bacilli do not excite cholera by invading the body from the bowel, but that, remaining in the intestine, they give rise to the virus, which is absorbed, and then causes choleraic symptoms. Virchow more than twenty years ago pointed out the resemblance of cholera to acute arsenical poisoning. How great must have been the amount of poison formed by the many billions of comma bacilli during their eight days' sojourn in my intestine! But I did not suffer at all from poisoning, was quite well, retained my appetite, had no trace of nausea, no fall of temperature, no albumen in the urine, etc., and went about my daily avocations, so that I could only conclude that, although comma bacilli may cause diarrhœa, they can not cause cholera, either European or Asiatic. Possibly in Hamburg my experiment might have ended fatally, because there, on Oct. 7th, in addition to the Asiatic *X* there was plenty of the Hamburg *Y* present, and it might have been that a much smaller dose would have excited severe cholera.

[When this experiment had terminated, another was made in the person of Professor Emmerich, conducted on the same lines, except that he took a more restricted diet. A similar record is given of the daily condition in this case, the experiment commencing on October 17th. Early next morning there was one fluid motion, and in the course of the day diarrhœa set in, so that on the 19th to 20th there were fifteen to twenty colorless, watery evacuations; an enema containing tincture of thebaica was administered, and on the 20th one of tannic acid and opium. The motions became natural in the course of the 21st. Comma bacilli were found in the stools from October 18th to 28th, the motions on the 19th being almost pure cultures. On the 24th Professor Emmerich returned to his usual diet. Throughout his general condition was undisturbed, appetite retained, no pain in abdomen, and only some weakness from the diarrhœa. Apart from the diarrhœa he had some hoarseness of voice and dryness of pharynx.]

These two experiments on man show that the comma bacillus does not generate the virus of Asiatic cholera, thus confirming Bouchard's results of the different effects of injections into animals of pure cultures and of the excreta (stools and urine) from cholera patients. Choleraic symptoms were not induced by the former, but only by the latter. Bouchard's experiments also show that the special cholera poison is only formed in the human organism. Perhaps one should concede that Emmerich and I did have a mild attack of cholera, as Koch and his numerous sup-

porters would say, but I cannot admit the correctness of their view of the sufficiency of *X* and *Z* to cause an epidemic, to the exclusion of *Y*, no more than I can agree to the regulations enforced in Germany, Austro-Hungary, and Italy, based on the discovery of the comma bacillus; and Dr. Baur and Dr. von Ziemssen, who have had large experience with cholera, affirm the symptoms we exhibited were not those of which they had experience in cholera epidemics. Yet according to the contagionist practice, on the discovery of comma bacilli in my evacuations I ought to have been ruthlessly confined in the isolation barracks of Munich and my dwellings thoroughly disinfected. It seems a pity that this did not take place, for if it had the contagionists would have been able to loudly proclaim they had saved Munich from cholera, since Emmerich and I, by our stools, which were discharged without disinfection into the closets and drains, might otherwise have certainly infected the town. Joking apart, I too would become a contagionist, so comforting and so sparing of all further trouble is the view, if it could only be explained to me why so many places into which cholera has repeatedly entered have never had an epidemic. Lyons, which stands on the direct line of traffic between Paris and Marseilles, two infected foci, is a striking instance. Koch's explanation of this from the practice of washing linen on boats in the fast flowing Rhone and Saone can hardly apply, since a like practice obtains at Zürich and Stuttgart, which do not enjoy such an immunity. The contagionists neither attack nor dispute the epidemiological facts which I have published on this head; they only ignore them, for they harmonize so little with contagionist theory.

Since 1831 Hamburg has been visited by cholera

fifteen times and Berlin twelve times (Table I). The traffic by land and water between the two cities is extremely intimate, giving every chance for the transference of comma bacilli.

TABLE I.—CHOLERA IN BERLIN.

Year.	Commenced.	Terminated.	Population.	Deaths.	Per 1000.
1831	Aug. 30	Jan. 26, 1832	229,843	1423	6.2
1832	June 17	Mar. 14, 1833	234,171	412	1.8
1837	Aug. 11	Dec. 6	265,394	2338	8.8
1848	July 27	" 9	400,557	1595	3.9
1849	May 30	" 11	401,802	3552	8.8
1850	Aug. 6	Nov. 24	405,707	711	1.8
1852	Sept. 4	Dec. 31	413,517	165	0.4
1853	Aug. 7	Nov. 30	415,425	940	2.3
1854	—	—	—	—	—
1855	July 26	Nov. 26	419,241	1385	3.3
1856	—	—	—	—	—
1857	—	—	—	—	—
1859	—	—	—	—	—
1866	June 14	Nov. 17	658,251	5457	8.3
1871	Aug. 14	" 3	826,341	55	0.07
1873	July 21	" 7	918,841	740	0.8

CHOLERA IN HAMBURG.

Year.	Commenced.	Terminated.	Population.	Deaths.	Per 1000.
1831	Oct. 31	Jan. 10, 1832	145,363	476	3.2
1832	Feb. 2	Dec. 17	146,365	1459	10.2
1837	—	—	—	—	—
1848	Sept. 1	Dec. 31	167,291	1674	10.0
1849	June 14	Nov. 22	168,061	563	3.3
1850	July 26	Jan. 11, 1851	171,013	400	2.3
1852	—	—	—	—	—
1853	June 23	Oct. 29	182,534	244	1.3
1854	" 14	Nov. 14	284,274	281	1.5
1855	" 30	Oct. 22	185,641	175	0.9
1856	" 13	Nov. 14	187,896	67	0.3
1857	" 9	" 27	191,910	463	2.4
1859	" 9	Oct. 5	196,747	1109	5.6
1866	" 30	" 22	214,174	1093	5.1
1871	Aug. 1	Sept. 24	325,332	141	0.4
1873	June 14	Nov. 8	348,127	1001	2.9

The lack of correspondence as to time and severity of the outbreaks in the two places, is notable, especially in the years 1831, 1837, 1848, and 1849. Again, since 1831 neither of these towns has had a winter epidemic, whereas in Munich two of the three outbreaks during the same period have been in the winter—viz., 1836-37, 1873-74. The seasonal variations in cholera are well shown in the statistics compiled by Brauser from all cases occurring in the kingdom of Prussia from 1848 to 1859, the numbers being grouped in semi-monthly periods. He found that the minimum of cases and deaths fell in the first half of April, the totals being for these twelve years, 71 and 50 respectively; while in the first half of September they amounted to 57,395 cases and 31,048 deaths. Taking the half monthly minimum of deaths as unity, the rise and fall in the incidence of the disease, as gathered from the statistics, may be thus given (Table II):

Date.	Relative mortality.	Date.	Relative mortality.
April 1-15	1.0	October 1-15	389.2
" 16-30	1.2	" 16-31	316.2
May 1-15	2.2	November 1-15	227.2
" 16-31	6.7	" 16-30	125.3
June 1-15	39.2	December 1-15	84.9
" 16-30	48.9	" 16-31	60.1
July 1-15	61.0	January 1-15	28.5
" 16-31	108.6	" 16-31	17.8
August 1-15	233.4	February 1-15	10.2
" 16-31	439.2	" 16-28	6.6
September 1-15	620.9	March 1-15	3.3
" 16-30	510.2	" 16-31	1.1

How is it possible to account for this enormous rise from 1 to 620, according to season, by the properties of the comma bacillus? Why does the bacillus act so powerfully in Hamburg and Berlin at one epidemic and so feebly at another? There must be a seasonal influence: What is it?

Some think it is due to *temperature*, but although

in Hamburg and Berlin epidemics generally begin in June and end in November, less often in October or December, yet there have been severe winter epidemics in Moscow, St. Petersburg, Munich, and elsewhere. In Calcutta, where it is endemic, the minimum of cholera is in August or September, the maximum from January to April, mostly April, and the mean temperature in Calcutta in April is 86° Farh., of August 82.4° Farh.—*i.e.*, is nearly equal. But if the comma bacilli simply pass from man to man, season should have no influence, for the temperature of the intestine is constant at 98.5° Farh.—a tropical climate. There is, however, at Calcutta, another climatic factor—*viz.*, the *rainfall*—which in April has a mean of 2.4 inches and in August of 14.6 inches, the annual average being 64 inches unequally distributed throughout the year. The rains begin in May and cease at the end of September or October, the remaining months passing, perhaps, without a drop. Now the monthly mortality of cholera forms a curve exactly inverse to that formed by the rainfall. Rain can hardly affect bacteria, especially comma bacilli which flourish in moisture and are destroyed by dryness. In Prussia the same correspondence between rainfall and cholera holds, and perhaps the cause of the winter outbreaks at Munich may similarly be explained. The 1873 epidemic in Munich bears this out. It began suddenly in August, and as rapidly declined, so that very few cases occurred during October, although at this time there was much movement of the population (changing houses, opening schools, etc.). At the commencement of November it broke out again, and by December 4th it had reached a height of 56 cases daily, whereas in the whole of October there were only 21 cases reported.

Inexplicable from the bacillary contagionist view, it can be readily explained from the localist standpoint. The germ *X* was present in Munich long before the first case occurred. It is an epidemiological fact that cholera in certain places in India and beyond India may remain quiescent for months, and then break out again, while even longer periods of quiescence must be admitted for the outbreaks in Hamburg, Berlin, and Munich.

In the summer of 1868, at Bellinghausen, in Essen and elsewhere in the Prussian Rhine Provinces, and in Westphalia, there were cholera epidemics, but nowhere else in Europe; and unless its autochthonous origin be accepted, these outbreaks must be referred to the residues of the epidemic of 1866, which had been dormant in Essen. The great epidemic in Egypt in 1883 did not affect the Mediterranean ports that year. France saw in this the efficacy of quarantine, nevertheless in 1884 it broke out in that country. So in July and August, 1873, the local outbreak in Munich occurred after a very dry July, and was checked by an exceptionally wet August, but reappeared in the winter during another abnormally dry season.

It is remarkable that in Augsburg, which suffered so severely in 1854, and into which, in 1873, cases were introduced from Munich, no epidemic occurred in that year. This was not due to disinfection—it was before the discovery of the comma bacillus—but, more likely, to the excessive rains with which Augsburg was visited in 1873.

There is no doubt that like atmospheric conditions played a part in the epidemic at Hamburg in 1892. The summer had been exceptionally dry and hot; the heat in August was almost unbearable, being about 9° Fahr. above the average, while the rainfall in

July, August, and September was considerably below the mean:

	1892.	Mean rainfall.
July.....	24 m.m.	99 m.m.
August.....	53 "	77 "
September.....	46 "	64 "
	<hr/> 120 m.m.	<hr/> 238 m.m.

Naturally also the level of the subsoil water was lowered. The temperature of the Elbe, which is taken in mid-stream every morning, rose in August to 71.6° Farh. Yet similar conditions of weather prevailed in other parts of North Germany, which were also susceptible to cholera, but where it has not yet become epidemic, and where possibly epidemics may break out later. As regards the rainfall, only those places can be compared where observations have been continuous, for, as at Munich and Augsburg, neighboring districts, where the average rainfall is the same, may differ very widely in particular years and seasons. The cholera germ from Russia has been more widely disseminated this year (1892) in France than in Germany, and that it only found a favorable soil in Hamburg must be due to a special reason.

Hamburg for years has been making a special hygienic experiment on the largest scale, for it thinks it can be cleansed by using an extremely impure water. The Hamburg water works distribute unfiltered Elbe water throughout the town and suburbs. In the mains are found dense layers of fungi of vegetable and animal origin; here and there a tap is plugged by the head of an eel. For drinking purposes the water may be filtered at home, or if this does not suffice, thirst may be quenched by wine, beer, seltzer, etc. For all domestic uses, for cleans-

ing rooms and dwellings, courts and streets, the unfiltered Elbe water is alone used, and then, owing to the excellent drainage into the Elbe, part of the refuse of the city must actually flow back into the water mains, thus rendering nugatory the sanitary aim of the drainage system. Above the reservoir the Elbe furnishes a purified water; but it becomes so contaminated on passing through Hamburg that within this area it cannot undergo self-purification. To be serviceable for consumption or domestic use its water should be further purified, as by filtration through sand, as is done at Altona, a few kilometers below Hamburg. At Cuxhaven the Elbe again becomes free from Hamburg impurities. It is plain also that the same degree of contamination may act differently on different soils, and all epidemics of cholera in Hamburg have shown essential differences between quarters situated upon marshy land and upon high ground.

Reincke has shown that since the introduction of a drainage system into Hamburg the frequency of typhoid fever has diminished, except for some variations in abnormally dry seasons. Epidemics of typhoid fever have this in common with cholera; but that Hamburg, in spite of its excellent drainage, may still be a fruitful soil for typhoid is shown by the epidemics of that disease during 1885 to 1887, while in 1892, associated with cholera, there was a notable increase in typhoid fever.

We are more fortunate in Munich, for formerly, with very good drinking water but high level of subsoil water, we often had more than twenty deaths annually from typhoid fever among 10,000 inhabitants. However, since 1881, with subsoil water at a very low level, the deaths have not exceeded one. The once

notorious typhoid soil of Munich has been gradually purified by sanitation, and we regard with some confidence any fresh visitation of cholera. In Hamburg itself a system of sand filtration was commenced until the cholera intervened, but the new water works are expected to be completed in the autumn of 1893. These works, on a gigantic scale, are under the direction of Engineer-in-Chief A. Meyer, and will cost many millions of marks, but not so much as Hamburg has lost by the cholera of 1892.

The part played by water in this epidemic has been variously explained. The "drinking water theorists" think that comma bacilli from Russian Jews found their way from the Elbe to the reservoirs, and were thus distributed throughout the town—a most plausible and comfortable explanation for the laity and the profession. Still experience does not always fit in with this theory, for outbreaks quite as "explosive" have occurred without the drinking water being at fault. I leave it undetermined whether in 1892 the Hamburg water operated directly as drinking water or indirectly as foul usable water. It is strange that in spite of the most careful search they should have had the misfortune not to find any comma bacilli in the Elbe water or in the water from the Hamburg mains. This is said to prove nothing, since other bacteria may be present which fluidify nutrient gelatin. Yet Koch found his bacilli in a Calcutta tank, and Fraenkel in the water of the Rhine where lay a boat containing a case of cholera. The water of the Indian ponds is not only drunk, but used for bathing and washing. The comma bacilli escaping from the human intestine into river or pool must be overcome by other bacteria and disappear very rapidly. I am not asserting that the comma bacillus

has no ætiological importance, but I believe it impossible for it to be the *X* which can excite and develop cholera epidemics apart from the *Y*. And if we have found a specific micro-organism in an infectious disease, we ought to hope that some means may be found thereby to combat the disease. Tuberculosis is a striking example. The discovery of the tubercle bacillus in the sputa of phthisical patients was scientifically as interesting and important as that of the comma bacillus in choleraic evacuations. But since the discovery of the tubercle bacillus, which is older than that of the comma bacillus, neither more or less people have died from phthisis than formerly.

The present protective measures against cholera rest entirely on purely theoretical contagionist bases. We have become very one-sided, thinking that cholera must be met by prevailing theories and not that theory should follow after cholera. It is deemed most important to seize the comma bacillus from the first case occurring in a place, and when its presence is proved to isolate the patient, and disinfect his excreta and his dwelling; and then the village or town is supposed to be protected from cholera. If in Hamburg the first case had been isolated and disinfected, the epidemic, so people think here, could not have broken out. Until a case comes to official knowledge it is in contact with others and the evacuations are not disinfected; and when reported, it must be determined bacteriologically whether it is a case of Asiatic cholera or one of cholera nostras. Then the source of infection must be traced, and often the disease breaks out in many parts simultaneously, as it did in Hamburg, adding to the difficulty of the task. Nothing but absolute stoppage of all traffic could avail, and that would be a greater misfortune than the

cholera. The spread of the cholera germ is not to be prevented, either in India or elsewhere, by isolation, disinfection, cordons, quarantines, etc. Just as, in spite of customs houses, goods are still smuggled over the frontier, so bacteria and viruses will be smuggled through all barriers. Still, improved sanitation may do much to prevent the smuggled germs gaining a foothold. In the sixties, when Prussia, Belgium, Holland, and France had most severe epidemics, Great Britain was only moderately invaded, and since 1866, in spite of its colossal traffic with the motherland of cholera, and the introduction of numerous cases from other lands, England has not had a single local epidemic, neither during the cholera time on the Continent from 1871 to 1874, nor during 1884 to 1887. Even this year (1892) cholera has not invaded Great Britain from Hamburg, Russia, and France, although England (London) was the only land which fearlessly trafficked with ships coming from infected ports.

If one cannot act against the introduction of the germ X , one must seek to act in the direction of Y and Z , and strive to make places or people immune. Every epidemic shows that many do not possess the Z , the individual disposition, and that they are immune. Some are protected by their own serum, perhaps, and it may be possible to ward off cholera, like variola, by protective inoculation. The contagium of variola was not mastered by isolation and disinfection until vaccination proved successful. Cholera is not contagious like variola, but depends on local conditions, and susceptible places may be rendered immune, like Fort William in Calcutta. There have been in other places as severe epidemics as this in Hamburg, but where all these measures of isolation, etc., were not in

vogue, and where the disease arose and subsided as quickly as it did at Hamburg.

[Professor von Pettenkofer here contrasts the epidemic at Munich of 1854 with the Hamburg outbreak. He goes on to contend that neither military cordons as in Russia, quarantines, or the forbidding of fairs, etc., have any influence; and he expresses the fear that in 1893, if there is a lack of rain, cholera may overrun Germany.]

The contagionists say that when cholera breaks out in a place, the rules demanded by their theory should be enforced. I am convinced that this is a mistake. I know of cases where nothing of this sort has been done, and the epidemic has been remarkably mild, much milder than where the contagionist practice has been carried into effect.

[In proof of this Professor von Pettenkofer cites at some detail the outbreak of cholera in Bavaria in 1836, and shows that the authorities acted on the anti-contagion plan, only transferring those cases to hospital which could not be cared for at home, and not interfering with public meetings and feasts. At that time Munich was in an unsanitary state, yet the epidemic was the mildest of the three it has experienced. There was no dread of cholera in those days. Nothing was known of bacteria; and cholera was attributed not to comma bacilli, but to the genus epidemicus. Traffic in and out of Munich was not deranged.

He concludes by expressing the hope that the

recent appearance of cholera in Europe will not lead to restrictive regulations based on merely theoretical grounds, so greatly hampering free intercourse, and even contrary to humanity, without any practical effect, and that the money expended on them will be devoted to attainable sanitary aims. Reflecting how many millions must have been thus sacrificed to theory in a single town like Hamburg, it is inconceivable, he remarks, "how opposition can be made to the new military bill before the Reichstag, which is based on much sounder grounds than the contagionist rules against cholera. I live and die in the conviction that our army will subdue enemies who would invade us from the east or the west, but *not* that our capture of bacilli, our cholera barracks, isolations, disinfections, prohibitions of entry and transit, or our quarantines, can prevent the invasion and dissemination of cholera."]

INDEX.

A.	Page
Aarau.....	227
Abdomen, retraction of.....	197, 202
Abdominal binder as a prophylactic.....	143, 161
Abscesses—see Suppuration.	
Absorption, capability of <i>prima viæ</i> for.....	134, 156, 167, 173
Acid hydrocyanic, value of.....	160, 170, 173
lactic, inutility of.....	150
Afghanistan.....	4
Africa.....	214, 222, 228, 233
<i>Agaricus muscarius</i>	114
Aged, cholera in the.....	81
Ainad.....	213
Aintab.....	212
Air baths, hot.....	135
, conditions and relations of—see Meteorology.	
, humidity and dryness of.....	123, 128
—effect of on nerves.....	110
, pure—necessity for.....	28, 32, 147
space, amount demanded for health.....	123
AITKEN, Sir WM.....	VI, 39, 43, 47, 52, 103, 163
Albany, N. Y.....	231
Albumen in urine.....	69, 79
Alcohol and chloroform mixture.....	161
, value of.....	137
Alcoholic excesses—see Intemperance.	
“Alesia,” steamship.....	237
ALEXANDER of Tralles.....	205
ALEXANDRÉ, F. S.....	21
Alexandria.....	233

	Page
Algidity—see Cholera, second stage of.	
, cause of.....	168
Algiers.....	222
Alkaloids, animal and cadaveric.....	38, 50, 52, 168
—auto-intoxication by...	
49, 50, 53, 83, 96, 109, 164,	168
Alkmaërt.....	194
Allahabad.....	2, 210
ALLISON, Sir ARCHIBALD.....	3
Aloes.....	124
Aloin.....	124
Alsace.....	227
Altitude and cholera.....	9, 221
Alvine secretions—see Dejections.	
Amboyna.....	211
America, North—see U. S. and Canada.	
, South.....	226, 234, 237
Anæsthesia of nerves.....	110, 160, 166
, unity of with hyperæsthesia and paralysis....	
110, 160, 166	
Anatomical characteristics.....	59, 60, 83, 84, 106
lesions, cause of.....	61, 83
, no appreciable.....	60, 61, 62, 83, 165, 167
Ancona.....	233
Andaman Islands.....	12
Animals and cholera.....	10, 25, 127, 210, 211, 214, 217, 220
, effect of cholera dejections on.....	25, 47
, experiments on with comma bacillus—inutility of	
241, 242	
<i>Antiaris toxifera</i>	170, 171
Antilles, the.....	226
Antioch.....	212
Anxiety, cause of appearance of.....	168
Aphonia, cause of.....	168

	Page
Apoplexy, heat.....	107, 108
Appetite.....	79, 98
Arabia.....	211, 223, 230
Aracca.....	211
ARÆTEUS, CAPPODAX.....	192
Aral Sea.....	215
Archangel.....	216, 218, 226
Archipelago, Indian.....	10
Army, U. S.....	22, 234
Arsenic poisoning—see Poisoning.	
Arteries, spasm of circular fibres of.....	56, 167
Arterioles.....	167
Arthrosporulation.....	30
Artificial diseases.....	41
Askabad.....	4
Asthenia.....	75
Astrakhan.....	5, 214, 215, 217, 223, 230, 231, 238
Astringents.....	134, 135
Astronomical influences and relations.....	6, 10, 12
“Atalanta,” steamship.....	233
Atlanta, Ga.....	235
Atmospheric influences and relations.....	8-11, 15, 19, 32, 127, 128, 204
Attendants, immunity of.....	19, 25, 26, 28
Augsburg.....	226, 227, 250, 251
AUGUSTINA, ANTOINE.....	199
AURELIANUS, CÆLIUS.....	192, 205
Aurangabad.....	210
Australian Expedition and cholera nostras.....	202
Austria.....	231, 236, 237
Autopsies—see Post-mortem.	
AYRE, JOSEPHUS.....	140
Azov Sea.....	215, 217

	Page
B.	
BABINGTON, Mr.....	138
Bacillary pathology—see Pathology.	
, sins of.....	38, 39, 246
Bacilli, cause of.....	40
, pathology of.....	40
, significance of.....	40
, tubercular.....	254
Bacillus of Koch—see Comma bacillus.	
Baghdad.....	211, 213, 222, 223
Baku.....	213, 238
Baltimore.....	221
BALY, WM.....	140
Bandage, flannel abdominal.....	126, 161
Barcelona.....	221
Baselland.....	227
Basle.....	70, 198
Basra.....	211, 227
Batavia.....	212, 223
Baths, hot-air.....	135
, foot.....	212
BAUR, Dr.....	246
Bavaria.....	222, 233, 256
BEALE, LIONEL.....	47
Bedding and infection.....	18, 22, 27, 28, 30, 126
, care of.....	126
Beef tea.....	145
Belgium.....	216, 233, 255
Bellary.....	211
Bellinghausen.....	250
Bender-Abbas.....	211
Bengal.....	8, 128, 230
Berlin.....	128, 216, 219, 220, 222, 223, 225, 226, 238, 247, 249
Bern.....	212

	Page
BERNARD, CLAUDE.....	57, 105, 112
BERRY, HARRY POOLE.....	176, 177
Betwa or Betowa (Sindh) river.....	17, 210
Bible, the—misquoted.....	191
BIERMER, A.....	37, 45, 227
BIGG, HERMAN.....	46
Bile, the.....	90
Bio-chemical pathology.....	38
Birds and cholera.....	10, 25, 127, 128, 220
Birmingham, Ala.....	235
Bismuth.....	125
BLACK, G. V.....	VI
Black plague, origin of.....	204
Black Sea.....	215, 217, 225, 231
Bladder, the.....	90
Blood, the—see Circulation also.....	56-60, 67, 85
, analysis and quality of.....	60, 85
, changes in.....	61
, chemical examination of.....	60, 85
corpuscles, disorganization of.....	120
, white—increase of.....	85
, dehydration of.....	62
, high specific gravity of.....	108, 109
-letting—see Venesection.	
Board of Health, Great Britain.....	142
, Tennessee.....	236
Bohemia.....	219, 223
BOHUN the Botanist.....	202
Bokhara.....	222
Bombay.....	210, 230
, fatality of cholera in.....	210
BONTIUS, JACOB.....	205, 230
Borneo.....	212
BOUCHARD, CHAS.....	53, 55, 62, 103, 245

	Page
Bowel, "corking" of.....	143
Bowels, condition of in reaction.....	79-81
BOWERBANK, JOS. SCOTT.....	145
<i>Braal-Loop</i>	202
Brain, ventricles of.....	107
Brandy, use and abuse of.....	137
Branson.....	26, 225
BRAUSER, Dr.....	248
Brazil.....	226, 237
Breslau.....	36, 97, 220, 222, 228, 229
Bretagne.....	236
BRIEGER, L.....	52
British Pharmacopœia, epispastic liquid of.....	172
Brittany.....	221
Broda.....	215, 219
Broncho-pneumonia, comma bacilli in.....	45
BROWN, ARTHUR E.....	43
, A. M.....	VI, 39, 51
, B. B.....	147
-SEQUARD, CHAS. E.....	106, 112
BRUCE, J. M.....	59
BRUNTON, T. LAUDER.....	114
Brzesc.....	219
BUCQUOY, JULES.....	27
BUDD, GEORGE.....	60
Buenos Ayres.....	234
BUHL, Dr.....	80
Bulgaria.....	223
Burials, premature—supposed.....	113, 150-152
Burma.....	2, 8, 230
C.	
Cadenazzo.....	226
CAGNEY, JAS.....	VI, 61
Cairo.....	26, 218, 231, 233

	Page
Cajeput oil treatment.....	136
Calais.....	220
Calcutta.....	3, 127, 206, 209, 210, 214, 249, 253, 255
Calomel.....	136, 137, 143, 144
, large doses of.....	141
salivation, relative dangers of.....	141
Camels and cholera.....	210
Camphor.....	171
Canada—see U. S. and Canada.	
Cannabis Indica.....	160, 171
CANNATACI, Dr.....	57, 178, 179
CANTANI, ARNALDO.....	47, 52
—mode of treatment by.....	149, 150
Canton.....	211
Capsicum.....	160, 172
Carbon-dioxide, exhalation of.....	75, 166
CARPENTER, WM. B.....	114
Carwar.....	211
Cashgar.....	214
Caspian Sea.....	213, 215, 216, 217, 225
Castor oil treatment.....	124, 142, 144
Catarrh, intestinal.....	61, 81, 82, 90
Catarrhal inflammation.....	61, 81, 82, 90
Cats and cholera.....	242
Cattle and cholera.....	211
Caucasus.....	215, 217, 223, 225
Celebes.....	211
CELSUS, CORNELIUS.....	192
Cerebral phenomena.....	78, 168
changes.....	106
Cerebro-spinal fluid, the.....	106
Cerium oxalate.....	125
Cette.....	222
Ceylon.....	211

	Page
CHADWICK, OSBERT	132
CHAINED, JNO.....	47
CHAMBERS, THOS. KING.....	30
Changes, anatomical—see Anatomical.	
, post-mortem.....	84, 126
Chattanooga, Tenn.....	235
CHERMAK, the elder.....	57
Chicago.....	235
Chickens, effect of cholera on.....	10, 25, 127, 217, 220
of fresh dejections on.....	25
Child-bed and cholera.....	158
Children, cholera in	70, 78
China.....	214, 230
Chlor-anodyne.....	125, 160, 171
Chloroform.....	143, 160, 171, 173
and alcohol mixture	161
spirit	124
water.....	125
Cholera, absorption of <i>prima viæ</i> in.....	134, 156, 167, 174
, action of drugs in.....	134, 137-140
, advantages of non-treatment in	145, 148, 149
ætiology.....	241
, and altitude.....	221
animals, birds, etc.....	
10, 25, 128, 210, 211, 214, 217, 220, 241, 242	
arsenic poisoning—see Poisoning.	
atmospheric variations.....	
9, 11, 15, 19, 28, 32, 127, 128, 222, 247-250	
bilious colic, similarity of.....	200
child-bed	158
cold	47, 197, 249
Crimean war.....	226, 233
crowding.....	122, 197
drains, gutters, and sewers.....	24, 33, 252

	Page
Cholera, and drinking water.....	
18, 24, 37, 124, 159, 223, 228, 229, 251-253	
drought.....	250
epizootics.....	10, 11, 81, 120, 127, 132, 241
famines.....	3, 10, 15, 129
filth.....	232
German-Austrian war.....	228
ground-water....	18, 24, 36, 37, 128, 226, 229, 252
human sacrifices.....	214
influenza.....	10, 11, 81, 120, 127, 132
malaria.....	12-15, 29, 83
moisture.....	248, 250
pilgrimages.....	2
poisoning.....	107, 108, 109, 114, 165, 168, 218, 221, 244
rainfall.....	128, 247-249, 250, 256
sanitation.....	18, 19
sewers, gutters, and drains.....	24, 33
shrine worship.....	2
soil—see Telluric.	
surgical operations.....	158, 166, 224
temperature.....	80, 197, 224
, antiquity of.....	205
, asphyctic.....	72, 168, 170
—cause of.....	168
, attacks, peculiarity of.....	220
bacillus—see Comma bacillus.	
, capricious character of.....	17, 18, 163, 219, 250
, causes of...2, 8-19, 24, 28, 31, 32, 39, 62, 83, 122-128	
, chronology.....	230
Commission, the German.....	41
contagion.....	241, 255
, convalescence from.....	73-81, 90, 94, 100
, dangers of.....	127

	Page
Cholera, death rate of	4, 61, 66, 75, 76, 80, 83, 91, 110,
III, 165, 166, 167, 219, 224, 225, 228, 230, 232-239	
—graduation of in epidemics	134
development, in India	128
, effect of sunlight on	28, 32
diarrhœa	13, 97, 98, 100, 102, 103, 117,
127, 128, 130, 156, 162, 163, 164	
, characteristics of	98
, mortality of	127, 128
, repression of	96, 134
domain of	69, 72
drops, Russian	156
duration of attacks of	59
ellipses of	221
epidemics, factors of	4-18, 31, 96, 102, 197, 235
, eruption of	83
, fatality of	216, 218
—Asiatics <i>vs.</i> Anglo-Saxons	145, 210
—extraordinary	213, 214, 215, 216
, febrile condition in	71
Gazette, The Indian	112
germ, the—see Comma bacillus, also	30
, effect of sunlight on	28, 32
, Government Report on (U.S.)	38
, grades of—see Cholera unity.	
, heart in	59-61, 85, 114
, history of	1-7, 191-205
, home of	2, 8
hypotheses	135
immunity	17, 120, 155, 225, 241
, artificial	255
, inadequacy of comma bacillus to produce	244, 246
infantum—see Cholera unity.	
infection	18-23, 33, 126

	Page
Cholera, infection by bedding, clothing, utensils, etc.....	18, 22, 126
drinking water.....	18, 24, 37, 124
ground water.....	18, 24, 36, 37
privies, water-closets, etc....	20-23, 34, 125
, influences of.....	9
, involuntary.....	20, 23
, intestinal changes in.....	59, 60, 84, 106, 166
laws.....	6, 168
, lessons taught by.....	135
literature, character of.....	135
malignant—see Cholera asphyctic.	
—as an epidemic.....	127
management of—past.....	133
—points to remember in.....	164
, meaning of term.....	I, 204-205
meteorologic relations of—see Meteorology.	
, monetary losses by.....	237
morbus—see Cholera nostras.	
mortality—see Cholera death rate.	
, graduation of in epidemics.....	134
, movements of.....	17, 203
, mycetic theory of.....	24
, neurotic origin of.....	v, 15, 30, 56, 62, 104, 110, 114
nostras.....	
13, 102, 103, 117, 129, 130, 161-164, 197, 200, 202	
, ancient treatment of.....	200, 202
, and drinking water.....	44
, comma bacillus in.....	103, 130
, epidemic.....	200
pathology—see Pathology and Neurotic..	110, 165, 169
, uncertainty of,.....	61, 62, 133, 165, 167
poison.....	17-20, 54, 109, 110
, action of.....	56, 109

	Page
Cholera, preferences of	17
, primary factor of	17
prodromes	63, 64, 93, 98, 99, 164
prophylaxis	93, 122, 124, 143, 161
, futility of	254
, pulse in	67
, quarantine	237, 238, 240
—futility of	254
ravages, relative danger of	127, 224
, relations of to other diseases	158
surgery	158, 166, 224
, rigor mortis of	84-88, 112
riots	218, 221
, Russian official returns of	238, 239
sanitation	37, 131
, sequels of	73
, severity of	15
sicca	72, 168
, cause of death in	170
<i>spasmodique</i>	202
specifics	155-157
, sporadic—see Cholera nostras.	
—in Canada and U. S.	129, 130, 221, 225, 226, 228, 231, 233
—in England and Europe ...	6, 194, 195, 198-200, 215, 220, 221, 223, 225, 226, 231, 234, 236, 255, 256
, spread of	15, 17, 19, 22, 24, 25, 29, 222, 255
stage, the first	63
—character of dejections in	63
, the second or asphyctic	65, 66, 67, 72, 93
—cause of algidity in	168
, the third	71, 75, 77, 94
, synonyms of	202

	Page
Cholera, taste in.....	79, 166
teachings, value of.....	135
, telluric relations of.....	
8-10, 33, 123, 128, 197, 226, 227, 228, 229, 247, 248	
, temperature in—see Temperature.	
therapeutics—see Therapeutics.	
, thirst in.....	66, 67, 98, 120
, tongue in.....	79, 166
, transmission of—see Cholera infection.	
, treatment (management of).....	156
typhoid.....	81-83, 92, 127, 167, 173, 252
, variations of.....	134
, vaso-motor disturbances in—see Nerve....	15, 62, 106
, vertigo of.....	168
, unity of.....	13, 70, 96,
102, 117, 129, 130, 157, 162, 163, 164, 173	
, urine in—see Urine.	
, walking.....	75
Cholérine.....	13, 96, 99, 102, 117, 129, 130, 157, 161-164
, characteristics of.....	99
Chorea, effects of on nervous system.....	110
Christiania.....	128
Cincinnati.....	10, 231, 232, 235
Circulation—see Blood.	
, changes in the.....	61
, failure of function of—cause.....	56, 167, 169, 170
, renewal of in reaction.....	77, 78
Clothing, soiled—disposal of.....	126
—infection from.....	18, 22, 126
Coca cordial.....	171, 173
Cocaine.....	171
Cochin China.....	211
Colapur.....	211
Cold, action of on nerves.....	110

	Page
Cold, effects of.....	123, 197, 249
, taking	98, 125
Coldbath Fields Infirmary.....	146
COLEMAN, Dr	172
Colic, billious—epidemic.....	201
—similarity of cholera to.....	200
, hepatic.....	200
Colitis, diphtheritic.....	89
Collapse—see also Stage, second.....	67
Colon, diphtheritic inflammation of.....	89
, spasmodic contraction of.....	198
Columbus, O.....	232
Coma	75
Comma bacillus, the.....	
13, 14, 40, 42, 43, 44, 48, 51, 103, 240, 241,	249
, absence of.....	45
, action of.....	244
, ætiological importance of.....	244, 253
and gastric juice—destroyed by.....	242
moisture.....	249
, arthrosporulation of.....	30
, easily destroyed.....	46, 253
, effect of drouth on.....	249
on guinea pigs.....	241
temperature on... ..	46, 47
, experiments with useless on animals.....	
.....	241, 242, 246
inadequate to produce cholera.....	244, 246
in drinking water.....	25, 44, 253
fluids, behavior of.....	46
mouth and throat.....	43
, presence of in broncho-pneumonia.....	45
cholera nostras.....	44, 130
, reactions and measurements of.....	43

	Page
Comma bacillus, von Pettenkofer on.....	45, 240-255
Como.....	226
Congestion.....	78
Conjeveram Pagoda.....	2
Conjunctiva, insensibility of.....	166
Constantinople.....	26, 218, 223, 233
Contagion and infection—see Cholera infection.	
Convalescence—see Cholera convalescence.	
and digestion.....	72
, protracted.....	90
Convulsions, spasmodic.....	113, 120
, tetanic.....	118
Corinth.....	193
<i>Cornaja Coleza</i>	202
Cornea, the.....	11, 70, 166
CORNET, Dr.....	49
CORNISH, Surg. Gen'l. WM. R.....	39
Coromandel.....	206, 211, 214
Coto.....	125
Cough.....	68
, whooping.....	179
Countenance and expression.....	70, 84, 85, 111, 168
Cracow.....	219
Cramps.....	65, 66, 79, 113, 118, 120, 167
, cause of.....	168
Creolin.....	150
Crises, cause of.....	168
CROOKSHANKS, GEO.....	vi
Croton oil.....	124, 172
Crowding, effect of.....	122
CULLEN, WM.....	56
CUNNINGHAM, Surg. Maj. D. DOUGLASS.....	
vi, 27, 39, 41, 42, 43, 47, 103, 130, 164	

	Page
CUNNINGHAM, Surg. Lieut. Col. J. M.....	
vi, II, 19, 27, 47, 130,	164
Cuxhaven.....	252
D.	
D'ACORTA, GARCIA.....	206
DALLEY, C. S.....	45
Dalton, Ga.....	235
DALTON, JNO. C.....	110
Damietta.....	26
Damp, effect of on nerves.....	110, 128
Dantzig.....	219
Danube river.....	215
D'ARSONVAL.....	57
DAVEY.....	75
DAY, GEO. E.....	109
Death, ante mortem.....	40
, cause of.....	75, 93, 166
during reaction.....	61
rate—see Cholera death rate or Mortality.	
-rattle, absence of.....	75
, rise of temperature following.....	111
with heart in diastole.....	167
Dejections.....	25, 30, 61, 71, 98
, absence of bile in.....	71
, changes in.....	25, 30
, character of.....	63, 65, 98
, constitution of.....	63, 108
, disposal of.....	125, 161
, duration of.....	71
, number of.....	70, 71
—relation to recovery.....	72
, repression of.....	96, 134
, “rice-water”—cause of.....	61
—dangers of.....	25

	Page
DELAFIELD, FRANCIS.....	62
Delft.....	194
Delhi.....	210
Delirium.....	74
DELPECH, A.....	47
Desquamation, epithelial intestinal.....	120, 165
Detroit, Mich.....	130
Deuteronomy misquoted.....	191, 192
Diarrhœa..25, 63, 71, 79, 82, 95, 96, 98, 100, 102, 103, 104,	167
, amount of.....	70, 71
, cause of.....	105, 168
discharges, number and frequency of.....	70, 71
, persistence of.....	98, 99, 102, 103
, prodromal.....	63, 64, 97, 104, 158, 160
—checking of.....	134
, repression of.....	96, 134
, simple—relations of to cholera.....	95-98, 125
, source of.....	105, 167
, “summer ”—see Cholera unity.....	163
, watery—source of.....	105, 167
Diarrhœas, differential diagnosis of.....	125-127
Diebourg.....	21
Diet.....	98, 123, 161
, influence of errors in.....	72, 98
Digestion and convalescence.....	72
DIODENES, death of.....	193
Diphtheria.....	127
Diphtheritic colitis.....	89
inflammation.....	81, 82, 86
Diseases, artificial in pathology.....	39, 41
, character of those of supposed bacterial origin..	40
, special virulence of in cholera times.....	158
, treatment a part of their pathology.....	174
Districts, exempt and immune.....	17

	Page
Dneiper river.....	217, 225
Dogs and cholera.....	210, 214
Don river.....	215, 217
DOYÉRE, Dr.....	166
Drainage, influence of.....	15, 17, 122, 252
Drains, gutters, and sewers.....	24, 33
DRAPER, JNO. W.....	109, 110
Draughts, effervescent.....	124, 159
Drinking water, influence of.....	
18, 24, 36, 37, 124, 139, 223, 228, 229, 251-253	
on comma bacilli.....	25, 44, 253
Drugs employed, list of useless.....	137-140
, inutility of.....	134
, non-absorption of.....	134
Dry situations, influence of.....	9
Dublin.....	220
Dunaberg.....	200
DUTRIEUX BEY.....	47, 164
Dyspnœa.....	68
E.	
Ebrach prison.....	22
Ecchymoses, intestinal.....	87, 89, 105
, pulmonary.....	86
Ecclesiastes misquoted.....	191
Edinburgh.....	220
Effervescing draughts.....	124, 159
Egypt.....	19, 42, 212, 218, 231, 233, 236, 250
EISENLOHR, Dr.....	242, 244
Ekaterinburg.....	5
Elaterium.....	124
Elbe river.....	219, 220, 223, 251-253
Emba river.....	215
EMMERICH, Prof.....	245, 246
Enemas, (enteroclysis).....	134, 149, 150, 197

	Page
England—see London, Manchester, etc.....	
194, 216, 220, 223, 226, 231-234,	255
ENGLISHMAN, Dr.....	206
Enteroclysm.....	134, 149, 150, 197
Epidemic laws.....	5, 6
phases.....	96, 102
rises.....	4, 31
Epidemics, circumscribed character of.....	17, 18
, ellipses of.....	221
, localized.....	250
, migrations of.....	204
, phases of.....	221
, the Hamburg.....	221
, winter—see Winter.....	249
Epidemiology.....	8
Epispastic liquid of B. P.....	172, 173
, no vesication by.....	173
Epithelium, intestinal.....	86
—desquamation of.....	120, 165
Epping Forest.....	35
ERB, WILHELM.....	109
Eruption, character of in cholera.....	83
Erzerum.....	212
Esthonia.....	218
Essen.....	250
Ether.....	148, 171
EULENBERG, ALBERTUS.....	58
Euphrates river.....	211, 223
Europe.....	195, 198-200, 215-221, 225-236, 256
Evansville, Ind.....	235
Exanthem, cholera.....	83
Exemption districts.....	17
Extension—see Cholera, spread of.....	
Eyes, the.....	11, 70, 166

	Page
F.	
<i>Facies cholericæ</i>	70, 84, 85, III, 168
FAGGE, C. H.....	53
Famine, influences of.....	3, 10, 15, 129
FARR, WM.....	6
Fatality—see Cholera death rate.	
of epidemics.....	120
Fauces, comma bacilli in.....	43
FAUVEL, ANTOINE.....	27
Fear, influence of.....	121, 127
Febrile condition and cholera.....	71
Fever, Hungarian.....	204
, malarial—see Malaria.	
, thermic.....	107, 108
, typhoid—see Typhoid.	
, yellow.....	204
Fibres, circular—spasm of.....	56, 167
Filter the Kedzie.....	124
Filth and cholera.....	3, 232
Finland.....	216, 218, 220
Fire as an antiseptic.....	146
Fish and cholera.....	10, 128
Flannel bandage—see Abdominal.	
FLINT, AUSTIN, Sr.....	47, 60
Florence.....	222
Fluid, cerebro-spinal—absence of.....	106
Fluids and comma bacillus—see Comma bacillus.	
Fly agaric.....	114
Food, abstinence from.....	123
, dangers of high game.....	123
indigestible.....	98, 123, 161
old vegetable.....	123
spoiling.....	123
Foot baths.....	212

	Page
FORESTUS, ALCMARINUS	194
FOSTER, M.....	57
FOTHERGILL, J. MILNER	167
Fougeres.....	201
FRAENKEL, Prof.....	253
France	193, 216, 220, 223-227, 232-236, 250, 251, 255
, death rate in.....	216
FRANCK, Prof.....	58
FRANCOIS, CHAS.....	58
FRANK, I.....	197
Frankfort.....	219
FRANKLAND, EDWARD.....	34
FRAYER, Sir JOSEPH.....	103, 164
French India.....	207, 211
FRENCH, J. M.....	58
Fruit, dangers of.. ..	123

G.

GAFFY, Dr.....	242
Galicia.....	215, 219, 223
Galvanism	142
Ganges river.....	202, 207, 209, 210, 211, 222
Gangrene.....	160
GASON, JOHN	143
GAUTIER, ARMAND.....	38, 49, 50, 51, 53
Genoa.....	222, 226, 227
Georgia (Asiatic).....	223, 235
, U. S.....	233, 235
Germ, cholera.....	28, 30, 32
theory, the—see Pathology.....	24, 39
German-Austrian war, cholera mortality in.....	228
Germany.....	220, 222-226, 231, 233, 248, 249, 251, 253
Gezd.....	213
Ginger, concentrated tincture.....	172

	Page
Glands, intestinal (Brunner's, Peyer's, etc.).....	87-89
, mesenteric.....	89
, œsophageal.....	86
, tracheal.....	86
Goats and cholera.....	210, 211, 214
GOERING, FED'K. WM.....	21
GOODEVE, EDWARD.....	19
GRATZER, J.....	36
Great Britain—see England, Scotland, Ireland and Wales.	
Greece.....	226
Griefswald.....	21
"Griping Disease," the.....	202
GRISSINGER, W.....	57
Grodno.....	219
Groënberg.....	229
GRONEMAN, Dr.....	172
Ground water—see Water, ground-	
GRUBER, Prof.....	242
GUASTELLA, AUGUST.....	27
GUÉRIN, Dr.....	57, 164
Guinea pigs, effect of comma bacilli on.....	241
GULL, Sir WM.....	140
Gutters, drains, and sewers.....	24, 33

H.

<i>Hachaiza</i>	202
Haime.....	21
HALL, Surg. Maj. J. C.....	vi, 67
Hamburg.....	5, 45, 46, 67, 220, 223, 226, 242, 246, 248-254
, epidemics.....	240
, therapeutics in.....	148-150
Hammersmith.....	35
Han-Kow.....	214
<i>Haoucha</i>	202

	Page
<i>Haouwa</i>	211
HARKIN, ALEXANDER.....	vi, 47, 57, 131, 172, 175, 178
HASTINGS, Lord—army of.....	3, 210
Havel river.....	219
Health Board of Great Britain.....	142
, Tennessee.....	vi, 236
Heart, action of cholera poison on.....	114, 167
muscarine on.....	114
pneumogastric on.....	170
, fibrinous clot of.. ..	85
, stimulation of.....	170
, the—in cholera.....	59, 60, 61, 85, 114
—death in diastole.....	167
Heat apoplexy.....	107, 108
HERTZ, M. von.....	14
High situations, influence of.....	9, 221
Himalayas.....	215
Hindustan.....	2
HIPPOCRATES.....	192, 195
HIRSCH, A. von.....	29, 30, 209
History of cholera.....	1-7, 191-229
Holland.....	223
HOLLAND, Sir HENRY.....	175
<i>Ho-Louang</i>	202, 206, 207
HOPPE-SEYLER.....	61, 109
Horseradish oil.....	172
House sanitation.....	124
HUEPPE, FERD von.....	150
Humidity, effect of.....	9, 110, 128, 203
Hungary.....	215, 220, 223
HUNTER, CHAS.....	19
Huntsville, Ala.....	235
Hunyadi-Janos water.....	124
HURD, E. P.....	53

	Page
Hurdwar Fair, the.....	4, 5
HUTCHINSON, JONATHAN.....	57
Hydrocyanic acid.....	160, 170, 171, 173
Hyperæsthesia, anæsthesia, and paralysis—unity of.....	166
Hypodermatics and hypodermoclysis... ..	139, 145, 149, 151, 160
I.	
IBRAHIM PASHA.....	218
Ice and ice water.....	145, 159, 160
Iceland.....	228
Immunity, cholera.....	17, 120, 155
—artificial.....	255
“Independenté,” steamshlp.....	237
India.....	2, 12, 16, 42, 45, 128, 207, 212, 222, 225, 230, 250
, French.....	207, 211
Indian archipelago, the.....	10
physicians, views of.....	128
sacrifices and cholera.....	214
Indican in urine.....	69, 166, 202
Indies, West.....	226
Indigestion—see Food.	
Indus river.....	211, 231
Infection.....	18–22, 33, 126
and contagion.....	19, 20
crowding.....	122
soil.....	8–10, 33, 123, 128, 197, 226, 227, 228, 229, 247, 248
sunlight.....	28, 32
ventilation.....	22, 28
by camping grounds.....	28
clothing, bedding, utensils, etc.,	18, 22, 27, 30, 126
dead bodies.....	28
drains, gutters, and sewers.....	33
drinking water.....	18, 24, 37, 124, 139, 252

	Page
Infection by ground water.....	18, 24, 36, 37, 128, 251, 252
hospital wards	28
ships, cars, etc.....	28
water-closets, latrines, privies, etc.....	20, 23, 34, 36, 37, 125
, capacity for.....	102
, predisposition to.....	102
, through respiratory tract.....	29
Inflammation, catarrhal	61, 81, 82, 90
, diphtheritic	81, 82, 89
Influenza.....	10, 11, 81, 120, 127, 132, 204
and cholera.....	10, 11
in Labrador	10, 11
, synonyms of.....	204
INGLOTT, Dr.....	178
Injections—see Enema and Hypodermatic.	
Innuits.....	11
Insects and cholera.....	10
Insomnia	83
Intemperance.....	75, 98, 121, 123, 167, 198
Intestinal absorption—see Absorption.	
catarrh.....	61, 81, 82, 90
changes.....	59, 61, 87, 89
ecchymoses	86, 89, 105
inflammation, diphtheritic.....	81, 82, 89
irritation, management of simple.....	124
lesions—see Intestinal changes.	
Intestines, influence of pneumogastric on	169, 172
sympathetic on.....	104, 105, 167
Ipecac.....	125
Ireland.....	127, 226, 231
Isero river.....	225
Ispahan.....	211, 212
Italy.....	222, 233, 236, 237

J.

Jagannath, (Juggernaut) the.....	2
Jajpur.....	210
JAKSCH, RUDOLF von.....	VI, 61
Jamaica.....	145
Jaragurth.....	17
Jassy.....	219
Jauer.....	229
Java.....	211, 212, 214, 230
Jessur (Jessora).....	3, 230
JOHNSON, GEO.....	VI, 57, 58, 142, 208, 209
, JAS.....	57
JONES, C. HANDFIELD.....	105, 106
Jumna river.....	209, 210
JUTERBORGK, on temperature.....	73

K.

Kabul.....	214, 222
Kalisch.....	219
Karatschi.....	231
Karum.....	212
Kashan.....	213
Kashmir.....	4
Katai.....	204
Kazan.....	5, 212, 217
Kerson.....	215
Kew.....	35, 130
Khoi.....	213
Kidneys, the—in cholera.....	69, 70, 82, 91, 165
—action of muscarine on.....	114
Kiev.....	217
KIRK, JAS.....	106
KLEBS, Prof.....	47, 52
KLEIN, EMANUEL.....	38, 46, 51
KNUTSFORD, Lord.....	172

	Page
KOCH, ROBERT.....	42, 43, 44, 45, 46, 48, 51, 52, 53, 54, 240, 241, 242, 245, 246, 253
Koch's bacillus—see Comma bacillus.	
Königsberg.....	36, 219, 227
Kostroma, Province of.....	5
Krasnoi-Yar.....	214
Kum.....	213
Kunstrin.....	219
Kur.....	213, 223
Kurdistan.....	211
Kurum.....	213

L.

Labrador.....	10, 11
Lactic acid.....	150
Lahore.....	4, 214
LANCASTER, Dr.	35
Lapland.....	228
Latakia.....	212
LATHAM, PETER.....	137, 174
Lauban.....	229
LAUENSTEIN, CARL.....	49, 149, 150
Laundresses and infection.....	26, 27
Law, well defined of cholera.....	168
Laws, epidemic.....	5, 6
LEBERT, HERMANN ...	VI, 19, 23, 26, 27, 28, 30, 47, 61, 65, 69, 70, 74, 80, 85, 88, 93, 96, 97, 101, 103, 106, 107, 108, 121, 158, 166, 216
Leeches.....	200
Lee river.....	35
Leghorn.....	222
Lemberg.....	219
LENKIN, Dr.....	200
Lenkoran.....	213

	Page
Leprosy.....	204
Leptandrin.....	125
Lesions—see Anatomical characteristics.	
Lessons of cholera.....	135
Lethargy.....	75
LEUBUSCHER, Dr.....	74
Leucocytes.....	86
Leucomaines.....	50
Levant, the.....	129
LEVER, CHAS. JAS.....	57
LEVINGTON, Dr.....	207
LEWINS, Surg.-Maj. R.....	154
LEWIS, Surg.-Maj. TIMOTHY RICHARDS.....	VI, II, 12, 39, 41, 42, 43, 47, 61, 63, 164
LIEBERMEISTER, KARL.....	96
LINKSTER, RAY.....	39, 43
Lisbon.....	221
Lissa.....	228
Little Rock, Ark.....	235
Liver, the.....	90
Liverpool.....	233
Lividity, cause of.....	67
Living, burial of—supposed.....	113, 150, 151, 152
Livonia.....	218
Lombardy.....	222
London.....	33, 34, 35, 128, 194, 195, 196, 201, 220, 223, 231, 234, 255
, plague of 1669.....	193, 194
Lucknow.....	4
Lugano.....	226
Lungs—see Respiration and Respiratory passages.	
Lupulin, pretended enormous doses of.....	136
Lyons.....	194, 246

	Page
M.	
Macao.....	208
Macassar.....	211
MACCORMAC, Sir HENRY.....	57, 177
MAC INTOSH (or MACKINTOSH), JOHN.....	152, 154
MAC LEAN (or MCLEAN), JOHN.....	11
MACNAMARA, CHAS.....	13
Madras.....	2, 207, 211, 214
Madrid.....	221
Magadino.....	226
Magdeburg.....	21, 23, 220
Magen.....	213
Maggiore Lake.....	226
Malabar.....	211
Malacca.....	211
Malaria.....	12-15, 29, 83
as an epidemic.....	14
Malarial fevers—see Malaria.	
Malay Peninsula.....	10
Maldivh Isles.....	206
MALOUIN, Dr.....	200
Malpighian glomeruli, the.....	91
Malta.....	59, 132, 172, 175-190, 222, 236
Manaar Island.....	211
Manchester.....	35
Manilla.....	211
Mansurah.....	26
Mardin.....	212
MAREY, JULES.....	56
Margate.....	34
Marienburg.....	219
Marine Corps, U. S.....	22
Mars, occultation of.....	10
MARSDEN, WM.....	147

	Page
Marsden's fluid	148
Marseilles.....	222, 226, 233, 236, 246
Massage.....	214
Mauritius.....	206
MAYS, THOS. J.....	47
Mazanderan.....	215
Measles.....	204
Mecca.....	218, 223, 231, 233
Medical men, immunity of.....	26-28
Medina.....	218, 223, 224
Mediterranean coasts.....	222, 250
Memphis, Tenn.....	233
Mendrisio.....	226
Mental phenomena.....	62, 74
Mercurials—see Calomel also.....	140
Meshed.....	222, 223
Mesopotamia.....	212, 230
Meteorological relations...8-15, 19, 25, 127, 128, 224, 247-250	
Metrorrhagia	80
Mexico.....	221
City.....	221
MEZERAY.....	193
Miasm, rice.....	8
Miasmatic theory.....	8, 24, 123
Migrations, epidemic.....	204
, racial.....	203
, religious.....	204
MINOR, THOS. C.....	VI, 191
MIRÉUR, HIPPOLYTE.....	29
Mirut (or Meroot).....	4
Mississippi Valley....	235, 236
Mittau.....	219
Moldavia.....	219
Monghir.....	209

	Page
Monkeys and cholera.....	211
Monsoons, influence of.....	9, 128, 206
Mont. Cenis.....	226, 227
Montevideo.....	237
Montreal.....	221, 231
Moravia.....	219
<i>Mordechi</i>	202
MOREAU, F.....	104
Morphine—see Opium also.	
, hypodermatic use of.....	149, 160
muriate, superiority of.....	160
Mortality.....	4, 61, 66, 75, 76, 80, 83, 91, 96, 110, 111, 165,
166, 167, 219, 224, 225-228, 230, 232-239	
among Asiatics.....	145, 210
, under no treatment.....	145, 148, 149
MORTON, J. C.....	6
<i>Morxi</i>	202
Moscow.....	5, 47, 80, 215, 218, 223, 225, 231, 239, 249
Mosul.....	211, 212
MOULINE, ETIENNE.....	27
Mountain heights, relation of.....	17
Mouth, comma bacilli in.....	43
Movements, involuntary from sympathetic.....	169
Mucous membranes, the.....	86, 89
Multiple causes of cholera.....	62
Munich..	22, 80, 222, 226, 227, 242, 246, 248, 249, 250, 251, 256
Murawutti District.....	211
Muscarine, action of.....	114, 168
Muscat.....	211, 212
Muscles, influence of poisons on.....	112
Muscular contractions, spasmodic post-mortem.....	113, 126
Mustard oil.....	172
Mycetic theory, the.....	24

	Page
N.	
Nagpur.....	210
Naples.....	222, 237
Nascopies.....	11
NASSE, OTTO.....	104
Nausea.....	100
NELLER, M.....	44
Nellur.....	211
Nephritis—see Kidneys, also.....	82
, chronic—rarity of.....	93
, croupous.....	81
, parenchymatous.....	69, 70
Nerve centres, poisoning of.....	113, 114
degeneration, evidences of.....	69, 166
exhaustion.....	69, 166
hyperæsthesia, anæsthesia, and paralysis..	110, 160, 166
perturbation.....	167
, pneumogastric—rôle of.....	106, 169, 172
reflexes.....	128
, sympathetic—functions of.....	106, 110, 114, 169
tone and predisposition to cholera....	121
toxæmia.....	49, 50, 53, 83, 96, 109, 113, 114, 164, 165
, vagus—see Pneumogastric.	
Nerves, effect of chorea on.....	110
cold and damp on.....	110, 128
malaria on..	110
, sympathetic, functions of.....	106, 110, 114, 169
, vaso-motor—rôle of.....	15, 62, 106, 169
Nervous system, action of drugs on.....	112
, central—rôle of.....	104
, depression of.....	167
, in cholera.....	74
, poisoning of.....	83, 96, 109, 113, 114, 165
—evidences of.....	164

	Page
Nervous system, sympathetic—influences of.....	
	56, 104, 106, 110, 114, 169, 170, 171
Neumarkt.....	229
Neurotic origin of cholera.....	v, 15, 30, 56, 62, 104, 110, 114
New Orleans.....	221, 225, 231, 232, 234
New York City.....	13, 221, 231, 234, 237
“New York,” the ship.....	32, 232
Nice.....	222
NIEMEYER, FELIX von.....	vi, 20, 37, 47, 61, 72, 82, 95, 103
Nijni-Novgorod.....	5, 215, 217, 234, 239
Nikolaiev.....	215
Nile river.....	218
Nîmes.....	194
Northwest Province of India.....	5, 230
NORTON, O. D.....	10, 25, 136, 141
Nudday.....	209
Nurses, immunity of.....	25, 26, 28

O.

Oder river.....	219, 229
Odessa.....	215, 217
Œsophagus.....	86
Ohio.....	235
Oil, cajeput.....	136
, castor.....	124, 142, 144
, croton.....	172
, horseradish.....	172
, mustard.....	172
, turpentine.....	143
Oldford.....	35
Opium and Opiates.....	134, 135, 136, 137, 139, 149, 160
Oran.....	26
Orenburg.....	87, 212, 214, 216, 230
ORTON, Dr.....	163

	Page
<i>Ouebb</i>	202
OZINAM'S "History of Cholera".....	191-216

P.

PACINI, FILIPPO.....	57, 152-154
Paducah, Ky.....	235
Pain—see Cramps.....	197
, præcordial.....	68
PAISLEY, Dr.....	206
Palcali.....	214
Palermo.....	236
Palestine.....	212
PALTAUF, H.....	112
Paraguay.....	237
Paralysis, anæsthesia, hyperæsthesia, etc....	110, 160, 166
, evidences of sympathetic.....	106, 165
Paris.....	128, 200, 216, 220, 221, 223, 224, 233, 236, 246
, death rate in.....	97, 224
PARKES, Surg.-Maj. E. A.....	57, 63
Pasteur, Louis.....	38
Patent nostrums, dangers of.....	124, 155-157
Pathology and artificial diseases.....	39, 40
, bacillar.....	38-50, 240, 241, 248, 249
—fallacies of.....	38, 39, 40, 41, 47, 50, 244, 246, 254
—Lionel Beale on.....	47
—A. M. Brown on.....	39, 50
—B. W. Richardson on.....	39
—M. Semmola on.....	47
—sins of.....	38, 39, 246
, bio-chemical.....	38
, inadequacy of.....	61, 84, 92, 126, 133, 165, 167
, lack of local.....	165
, treatment a part of.....	174

	Page
Pegau.....	199
Pekin.....	214
PELLISSIER, GASTON.....	27
Penang, Island of.....	211
Perm.....	5
“Perseo,” ship.....	237
Persia.....211, 212, 213, 215, 217, 222, 225, 230,	231
Pertussis.....	179
Peshawr.....	4
PETER, M.....	48
PETTENKOFER, MAX J. von.vi, 13, 22, 27, 33, 45, 128, 164,	240
PEYRANI, CONI.....	106
PFEIFFER, Dr.....	242, 244
PFLUGER, Prof.....	104
Philadelphia, Pa.....	221, 231
Phthisis.....	254
Physical phenomena, great movements of.....	203
Physicians, immunity of.....	25, 26
Piedmont.....	222
Pigeons and cholera.....	220
Pigs, effect of cholera dejections on.....	25
PISANI, S. L.....	6, 177, 178
Plague.....	204
, black—origin of.....	204
, of 1669.....	193, 194
Pless.....	229
Plexus, the aortic.....	169
, cardiac.....	169, 171
, cœliac.....	166
, hypogastric.....	169
, renal.....	169
, solar.....	167, 169, 171
—relations of.....	105
, spermatic.....	169

	Page
Plexus, uterine.....	167
Plexuses, lack of tonicity in.....	166, 170
Pneumogastric, functions of.....	106, 169, 172
—cardio-inhibitory.....	170
, stimulation of.....	171
Pneumonia.....	81
as a cholera sequel.....	169, 172
Podolia.....	218
Poison, the cholera—specific effect of....	56, 107, 109, 112, 114
and rigor mortis.....	84, 88, 112, 113
Poisoning, alkaloidal.....	38, 50, 52, 83, 96, 108, 109, 164, 168
, arsenical.....	107, 108, 165, 168, 244
, autogenetic.....	107, 108, 117
, cholera—parallels of.....	107, 108, 165, 168, 244
, muscarine.....	109, 114
of nerve centres.....	83, 96, 109, 113, 114, 164, 165
, fatal.....	164
, profound.....	96
, ptomaine.....	38, 50, 52, 83, 96, 107, 108, 117, 120, 160, 168
, serpent.....	127, 160
, uræmic.....	166
Poisons, action of as a class.....	109
, influence of on muscles.....	112
rigor mortis.....	84, 88, 112, 113
Poland.....	215, 219, 223, 226, 231
Polynesia.....	228
Ponany.....	210
Pondicherri.....	207, 211
Portel river.....	46
Port Huron, Mich.....	16
Portugal.....	221, 233
Post-mortem evidences.....	61, 62, 84, 126, 133, 165, 167
examination, chemical results of.....	93, 126

	Page
Post-mortem phenomena.....	84-88, 113, 150
Potassium bicarbonate.....	159
POUCHET, A. G.....	52
Præcordia, pain in.....	63
Pregel river.....	36
Prima viæ, absorption by.....	134, 156, 167, 174
Privies, latrines, and water closets.....	20, 22, 29, 31, 34, 36, 37, 125
	—use of by strangers.. 125
Prodromata.....	63, 64, 98, 99, 104
Prophylaxis.....	122, 124, 143, 161
Prussia—see Germany also.....	216, 219
Ptomaines.....	50
, poisoning by.....	38, 50, 52 83, 96, 107, 108, 117, 120, 160, 168
Puerperium and cholera.....	158
Pulmonary—see Respiration, ecchymoses.....	86
Pulse, the.....	67
Punjab, the.....	4
Purgatives—see Calomel and Castor Oil also.....	198
Puri (or Pooree).	2
Pyæmia—see Suppuration.	

Q.

Quarantines.....	256
, inadequacy of.....	237, 238, 240
, regulation of in Europe	246
Quebec.....	221, 231
Quotations, false from Bible.....	191

R.

Rainfall, relations and influence of—see Water, ground- also	128, 247-249, 250, 251, 256
Reaction—see Cholera convalescence.	

	Page
REAN, Dr.....	12
Reflexes.....	62
REIDER, Dr.....	148
REINCKE, Dr.....	252
REINHARDT, Dr.....	74
Religious migrations.....	204
Repression of cholera discharges.....	96, 134
Respiration.....	68, 79, 118, 120
, causes of failure of function of.....	169, 170
, cubic space demanded for successful.....	123
Respirations, number of.....	68
Respiratory passages and infection.....	29
, changes in.....	37, 86
, infarctions in (lungs).....	86
Revolt caused by cholera.....	218, 219
Revulsive preparations.....	172
Rhine river.....	216, 225, 253
Rhone river.....	225, 246
Rice, diseased—and cholera.....	8
“Rice water” discharges—see Dejections also.....	28
, fresh—effect of on animals... ..	25
, stale—effect of on animals.....	47
RICHARDSON, BENJ. W.....	39, 47
Riga.....	219
Rigor mortis.....	84, 88, 112
and the sympathetic.....	113
poisons.....	84, 88, 112, 113
Riots, cholera.....	218, 221
River Betwa (Sindh).....	17, 210, 222
Danube.....	215
Dneiper.....	217, 225
Don.....	215, 217
Elbe.....	219, 220, 223, 251-253
Emba.....	215

	Page
River Euphrates.....	211, 223
Ganges.....	202, 207, 209, 210, 211, 222
Havel.....	219
Indus.....	211, 231
Isere.....	225
Jumna.....	209, 210
Lee.....	35
Nile.....	218
Oder.....	219, 229
Portel.....	46
Pregel.....	36
Rhine.....	216, 225, 253
Rhone.....	225, 246
St. Clair.....	16
Saone.....	246
Spree.....	219
Thames.....	35
Tigris.....	223
Volga.....	215, 217, 223, 225, 231
RIVIERE, LAZARE.....	194
ROGERS, GEORGE.....	143
Rome.....	222, 238
Roumania.....	223
RUMPF, Dr.....	148
Russia.....	223, 230-234, 238, 239, 255
, death rate in.....	234, 238, 239
RUST, Dr.....	219
S.	
Safety measures.....	158-161
St. Clair, Mich.....	16
river.....	16
St. James' Parish, London.....	34
St. Louis, Mo.....	232, 234, 235
St. Petersburg.....	216, 218, 220, 225, 230, 239, 249

	Page
Saline treatment	145, 147, 148
Salines, intravenous use of (hypodermoclysis).....	136, 146-152
—fatality of	148, 149
—non-utility of.....	148, 149, 154
Salisbury sickness, the.....	117
Salivation, relative dangers of calomel	141
Salol, inutility of	148
Salpêtrière hospital, death rate in.....	224
Samara.....	5
Samarkand.....	222
SANDERSON, BORDON.....	41
San Felipe.....	237
Sanitation	37
, weakness of.....	131
Santiago, Chili.....	237
Saone river.....	246
Saratov (or Saratof).....	5, 217, 218, 238
Sarnia, Ontario.....	16
Savoy.....	225
Saxony.....	233
Scandinavia.....	220, 226
Scarlatina.....	127
SCHALLER, JEAN JACQUES.....	198
SCHIEFFENDECKER, W.....	36
SCHIMMELPENING, Baron.....	202
SCHMID, C.....	61, 109
Scientific American Supplement.....	240
Scindia.....	17
Scotland.....	226, 231
SCRIVEN, Dr.....	163
Secretion blood depuration.....	15, 84, 167
, inhibition of.....	134, 156, 167
SEDGWICK, WM.....	47, 57
SELMI, FRANCESCO.....	38

	Page
SEMMOLA, MARIANO	47, 48, 103
Sensorium, effect of cholera on.....	166
Serachs.....	213
Serum, diarrhœal—composition and transudation of,.....	109, 165, 167
Sevastopol.....	215, 217
Seville.....	221
Sewage water, relation of to cholera distribution.....	15, 17, 122, 252
Sewers, gutters, and drains.....	24, 33
SHAKSPEARE, E. O	38
Shiraz	211, 213
Shirwan.....	215
Shock, susceptibility to.....	120, 166
Siam.....	2, 8, 230
Siberia.....	214, 219
Sicily.....	236
SICLUNA, Prof.....	59
SILENUS.....	192
Silesia.....	220
Sillhet.....	209
SIMMONS, Sir LINTORN.....	175
SIMON, JNO.....	34, 47
SIMPSON, JAS.....	27
SIMS, Dr.....	201
<i>Sinanga</i>	202
Sinapisms	161
Sindh river.....	17
Singapur.....	211
<i>Sitanga</i>	202
Skin, characteristics of in cholera.....	68
Sleeplessness	83
Small-pox, the.....	204
Smyrna.....	218

	Page
SNOW, J.....	33, 57
Soil, effects of damp and dry—see Infection also.....	
	123, 128, 228, 229, 247, 248
SOMERSET, Dr.....	206
SONNERAT.....	216
Sopor.....	166
Southampton.....	233
Spain.....	221, 222, 233, 236, 237
Spasm—see Cramps.	
, intestinal.....	198
of circular fibres.....	167
Spasmodic twitching, post-mortem.....	112, 113
Spleen, the.....	89
Spore, a cholera.....	30
Spree river.....	219
STAEDLER, Dr.....	93
Stages, the—see Cholera stages.	
STEEL, GEORGE.....	142
STERLING, JAS.....	61
Stettin.....	21, 216
STEVENS, WM.....	145
Stevens' fluid.....	146
Stockholm.....	220
Stomach.....	86, 87
, inhibition of absorption by.....	134, 156, 167
STONE, Dr.....	144
Stools—see Dejections and Diarrhœa.	
Strength, factitious.....	75
Strychnine.....	144
Stuttgart.....	246
“Summer” diarrhœa—see Diarrhœa.....	163
Sunlight and cholera germs.....	28, 32
Sun-stroke.....	107, 108
Suppuration sequels.....	84

	Page
Surat.....	211
Surgical operations and cholera.....	158, 166, 224
Susa.....	213
SUTHERLAND, Dr.....	132
“Swanton,” the barque.....	32
Switzerland.....	226, 227
SYDENHAM, THOS.....	7, 194, 195
Sympathetic nerve, division of intestinal portion of.....	105
, functions of.....	106, 110, 114, 169
, involuntary movements from.....	169
system.....	56, 169, 170, 171
, antagonism of.....	110, 170, 171
, functions of.....	110, 114-120
, identity of anæsthesia, hyperæsthesia and paralysis of.....	105
, relation of rigor mortis to.....	113
, sensitiveness of.....	110
, stimulation of.....	170, 171
Syphilis.....	204
Syria.....	212, 218, 223, 230

T.

Tabes.....	62, 165
TALAMON, CHAS.....	103
Tanganrog.....	215
TANNER, THOS. HAWKES..v, 47, 58, 60 61, 103, 122, 133, 145	
, criticism of cholera management by..	144
Tannin enemæ (enteroclysis), futility of.....	148-150
TARCHONOFF, Dr.....	58
Tarnowitz.....	229
Tarsus, the.....	215
Taste, character and sensation of.....	79, 166
Taunton Workhouse.....	123

	Page
Tauris.....	212, 217
Tebris	213
Teching-Tchu Tching-Ching.....	207
Teddington Loch.....	35
Teheran.....	215, 217, 222, 223, 231
Telluric relations.....	10, 14, 15, 19, 127, 158, 206, 247, 248
Temperature.....	9, 71-73, 78, 197, 224, 250
and the comma bacillus.....	46-47
, depression of.....	109, 119, 166
—causes of	106
, high.....	250
, post-mortem—rise of.....	111, 120, 166
Terror, effect of on intestines.....	107, 166
Tessin, Canton of.....	226
Thames water	35
Therapeutic nihilism, advantages of.....	145, 148, 149
Therapeutics, early.....	194, 195, 196
, general inutility of.....	111, 133-136, 140, 157
, weakness of	111, 157
THIERSCH, Prof.....	47
Thirst, character and significance of.....	67, 98, 120
THURSTON, Mr.....	176
Tiflis	5, 215, 217
Tigris river.....	223
Tilsit.....	219
Tissues, ante-mortem death of.....	40
, dehydration of.....	62
Toad flax.....	198
Tobacco.....	142
Tongue, condition of.....	79, 166
Tooting Pauper Asylum.....	123
Toulon.....	222, 236
Tox-albumins.....	50
Toxicity—see Poisoning.	

	Page
Transfusion—see Salines also	144
Transmission—see Infection.	
Transylvania.....	215
Treatment, advantages of non-.....	145, 148, 149
by cajeput oil.....	136
calomel....	136, 137, 141, 143, 144
castor oil	124, 142, 144
“corking”.....	143
creolin.....	150
ether.....	148
galvanism.....	151
intravenous injections—see Saline.	
morphine hypodermatically.....	149
opiates	134-139, 149, 160
tannin enemata.....	148-150
venesection.....	135, 142, 144
, expectant	145, 148, 149
, futility of popular and specific.....	155, 156
, nihilistic.....	145, 148, 149
, recoveries under all forms of.....	133
, revulsive.....	172, 196
, saline—inutility of.....	144, 145, 148, 149
, symptomatic.....	149
, vagus.....	172, 179, 190
—clinical reports on.....	180, 189
—comparative evidence of.....	189
—prompt action of.....	179
TRICHUM, Prof.....	48
Trieste.....	222, 236
Trincomali.....	206
Tripoli, Syria.....	212
<i>Trisplanchnite</i>	202
TROUSSEAU, ARMAND.....	109
<i>Trousse-galant</i>	193

	Page
Tubercular bacilli.....	254
Tuberculosis.....	254
Turin.....	222
Turkey.....	231
Turpentine.....	143
Typhoid condition.....	81-83, 92, 127, 167, 173, 252
, causes of.....	136, 137, 252
, induced by opiates.....	136, 137
Typhus.....	81, 204
, synonyms of.....	204
Tyrol.....	222, 225
TYTLER, Dr.....	203, 209
Tzaritzyn.....	5

U.

Ulm.....	197
United States and Canada.....	221, 225, 226, 228, 231, 233
Army and Marine Corps.....	22, 234
Uræmic poisoning.....	166
Urine, the.....	79, 81, 82
, albumen in.....	69, 79
, indican in.....	69, 166, 202
, microscopy of.....	69
, reaction of.....	119
, red—see Indican.	
, specific gravity of.....	79
, suppression of.....	69, 166

V.

Vagus—see Nerves and Pneumogastric.	
Valais, Canton of.....	27, 227
Valencia.....	233
VANG-CHOU KO, Dr.....	206
Vaso-motors—see Nerves.	
VASQUEZ, S. G.....	104

	Page
Venery, effect of.....	124, 127
Venesection.....	135, 142, 144, 200, 212
Venice.....	199, 236
Ventilation.....	28, 32, 147
Vera Cruz.....	221
Veronica.....	198
Vertigo, causes of.....	168
Vicksburg, Miss.....	234
Vienna.....	128, 216, 220
VIRCHOW, RUDOLF.....	47, 85, 165, 244
Voice, the.....	68, 69
, loss of—see Aphonia.	
VOIT, CARL von.....	243
Volga river.....	215, 217, 223, 225, 231
Volhynia.....	218
VOLK, Dr.....	80
Vomiting.....	30, 71, 198
, amount of fluid.....	71
, cause of.....	105, 168
, character of fluid.....	30, 71, 100
<i>Vox cholericæ</i> —see Voice.	
Vulgate, the.....	191, 192

W.

Wales.....	232
“Walking cholera”.....	75
Wallacia.....	223
Warsaw.....	219, 227
Washerwomen—see Laundresses.	
Water-closets.....	20, 22, 23, 29, 31, 34, 36, 37, 125
, drinking.....	18, 24, 36, 37, 124, 139, 252
—comma bacillus in.....	44, 253
—filtering of.....	124
, Elbe river.....	253

	Page
Water, ground-.....	18, 24, 36, 37, 128, 251, 252
, hot—intravenous injection of	138
, Hunyadi-Janos	124
, iced.....	145, 159, 160
, Portel River.....	46
, Pregel River	36
, Rhine river.....	253
, Rhone river.....	246
, saline—see Salines.	
, Saone river.....	246
, sewer—relation to cholera distribution. 15, 19, 122,	252
, Teddington Loch	35
, Thames River.....	35
, warm—revulsive effects of.....	138
Waters, aërated.....	159
WATSON, Sir THOS.	VI, 11, 47, 56, 135, 137
WEGELEIN, Dr.	28
West Indies.....	226
Westphalia.....	250
Whooping-cough	179
Winnipeg.....	130
“ Winter ” diarrhœa.....	163
epidemics of cholera.....	80, 197, 218, 249
Z.	
ZACTUS.....	206
ZEHNDER, C.....	61, 96, 103, 126, 227
Zengan.....	213
ZIEMSEN, HUGO WILHELM von.....	29, 286
Zobten.....	229
Zürich..	27, 28, 64, 69, 70, 79, 86, 89, 93, 97, 107, 126, 226, 246

