EPIDEMIC CEREBROSPINAL MENINGITIS

BY W. J. CLASS, M.D.
MEDICAL INSPECTOR CHICAGO HEALTH DEPARTMENT.
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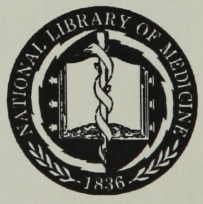

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INTRODUCTION.

The objects which led to the investigation of which this report is the outcome were: 1, to ascertain whether true epidemic cerebrospinal meningitis existed in the city of Chicago, and 2, to trace if possible its origin and the avenues by which it spread. In regard to the first of these objects, it was soon discovered that there was a greater prevalence of the disease than was generally supposed, and enough data were collected, which we hope are of sufficient interest to the general practitioner, to excuse the writer for encroaching on those particulars of the disease which, strictly speaking, do not perhaps belong to the territory of the sanitarian. Chicago has for a number of years fortunately escaped the ravages of a disease which has been in a constantly increasing number of instances prevalent throughout a large part of the country, particularly in its eastern portion. There is hardly room for doubt that sporadic cases of epidemic cerebrospinal meningitis have almost constantly occurred in our midst; often perhaps escaping recognition from the fact that it is a disease the practitioner has not generally been accustomed to encounter. It was not, however, until the early part of 1898 that these sporadic cases, occurring with increasing frequency, attained the dignity of an epidemic. The greatest number of cases were recorded in April and May; the latter part of August and the early part of September found the city practically free from the disease. The total number of cases which occurred in this epidemic can not be positively stated, as there was no system of notification, but, approximately, the number probably approached close to two

hundred during the first seven months of 1898. In presenting this report it is not the intention of the writer to dwell to any considerable extent on the history, classification, symptomatology and treatment of the disease, as these subjects have been exhaustively treated by other writers, more particularly in the excellent monograph by Councilman, Mallory and Wright, recently published by the Massachusetts State Board of Health, but a brief synopsis of the aforementioned subjects will, we hope, be deemed admissible.

The writer wishes to express his indebtedness to Drs. Christopher and Hook of this city for histories of cases furnished by them; to Dr. D. H. Ghon, assistant pathologist to the Pathological Institute of Vienna, Austria, for valuable data, especially in regard to findings made by him in his investigation of the epidemic of cerebrospinal meningitis which raged in Trifail, Steiermark; also for the many courtesies shown him by the resident and attending staffs of the Alexian Brothers, Mercy, Michael Reese, Cook County, West Side, St. Elizabeth, Augustana, St. Joseph and Maurice Porter Memorial hospitals, Chicago, and the Daily News Sanitarium for sick babies.

HISTORY.

The first authentic description of this disease is generally accredited to Vieusseaux,[1] a physician of Geneva, who described an epidemic in that city which presented a symptom-complex that previous to his time had not been recognized as an independent disease. In America the first cases of this disease were described by Danielson and Mann[2] in 1806, in which year a number of cases had occurred in Medfield, Mass. About this time also an epidemic occurred in Southern France. In Germany the disease first made its appearance in 1822.

That these were really the initial epidemics of a disorder which had previously not existed is extremely doubtful, especially when the confusion existing in

regard to the different fevers during the eighteenth and early part of the nineteenth centuries is taken into consideration. The almost identic phenomena to which at the present time the name of cerebrospinal meningitis is given, appear in descriptions published in the eighteenth century, under the name of cerebral typhus, jail fever, spotted fever, etc. Even as late as 1846 there was considerable doubt among physicians whether they really had to deal with an independent disease in cerebrospinal meningitis, as is shown by a paper by Rampold.[3] Arnell[4] described an epidemic of spotted fever, the symptoms of which were closely analogous to cerebrospinal meningitis, which existed in Orange County, N. Y., in 1808-1809. From this time until about 1825 the disease was very prevalent in the eastern part of the United States, more particularly in New England. Its spread was, however, not uniform, several towns widely separated being attacked simultaneously, while in the intervening places the disease would perhaps put in an appearance after a lapse of a few years. Very few of the larger towns in Massachusetts and Connecticut entirely escaped the disease during this period, and it was, as a rule, of a more violent type than has been described in recent epidemics, death often occurring within twenty-four to forty-eight hours after its onset; the skin lesions were also much more prominent, hence the name of spotted fever generally given to it. During the period mentioned, Europe remained comparatively immune, although the neighborhood of Geneva, where the disease was first recognized, seemed to remain infected, and small epidemics occurred from time to time.

During the thirties, the disease became more prevalent in Europe, prevailing principally in the western and southern portions, the central States enjoying comparative immunity. No epidemics were reported in the United States from 1830 to 1842. In 1848 Chipley[5] described some cases of epidemic cerebrospinal meningitis occurring in Cincinnati, and in

1846, Mayne[6] some cases in Dublin. Von Ritter[7] of Prague describes this disease in 1851, and Roger[8] in 1846, reported some of the early cases noted in southwestern Germany. In Ireland the disease again made its appearance in 1855, in which year there appeared a report on it by Banks.[9] From 1850 to 1860 the number of cases reported was small, there being no very extensive epidemics noted. During the sixth decade and the first half of the seventh a great increase was noted, epidemics being reported as occurring in almost all the countries of Europe and the United States, and a large addition to the literature on this subject was made. Among the publications of this period are those by Andrew,[10] Baennler,[11] Barton,[12] Day,[20] Gibbon[22] and Martin[30] of Great Britain; Bessey[13] of Canada, Bleything,[14] Borland,[15] Chandler,[18] Conklin,[19] Gifford,[23] Jones,[26] Upham[33] and Webber[34] of the United States; Buttersack,[16] Cannstatt,[18] Hampeln,[24] Heller,[25] Maier,[29] Maier,[31] and Wiebecke[35] of Germany; Spörer[32] of Russia and Drasche,[21] Kratschmer[27] and Loudon[28] of Austria. From 1875 to 1885 there was a partial remission of the progress of the disease, only a few scattered epidemics and sporadic cases being reported. The year 1887 was distinguished by the most important discovery in connection with the study of this disease, viz, that of the micro-organism now generally conceded to be the causative factor—the diplococcus intracellularis. This organism was first described by Weichselbaum,[36] who had discovered it in the exudate of a number of cases of cerebrospinal meningitis, and it was by him considered as the probable causative factor of the disease. From 1885 to 1895 the disease appeared more generally in Germany than it had previously.

The most notable accounts of cerebrospinal meningitis during this time are those by Blümm,[37] who described several small epidemics occurring in Bavaria; Leichtenstein,[38] who published an excellent account of the disease as it occurred in the Rhine Province; Bonome,[39] whose articles on the bacteriology of cere-

brospinal meningitis are very good; Lor,[40] who described localized epidemics in the eastern counties of England; Flexner and Barber's description of an outbreak of cerebrospinal meningitis at Lonaconing; Petersen's[42] article on the epidemiology of cerebrospinal meningitis, and Lemoine's[43] report of an epidemic in France in 1891. Since 1895 numerous epidemics have occurred, both at home and abroad; among the more important is the epidemic in Boston in 1897, which has been ably described by Councilman, Mallory and Wright[44]; in the same year there occurred an epidemic in Wuertemberg, which was reported by Benedict.[45] In the winter of 1897–98 a great number of deaths were reported as occurring among the modern Argonauts of the Klondike. During the early part of 1898 a severe epidemic raged in Trifail in Austria, while epidemics and sporadic cases were reported as occurring in different places in the United States, among others Los Angeles,[46] St. Paul[47] and among the soldiers at Chickamauga Park, Ga. The epidemic in Chicago began in January, 1898.

ETIOLOGY.

In treating on the etiology of this disease each factor will be considered under a separate heading, since by doing so it will be easier for the reader to get a view of the entire subject.

Epidemic nature.—As a rule, this disease occurs in well-marked epidemics, as is seen from the preceding chapter; sporadic cases, however, are of frequent occurrence, especially in the larger cities, very closely resembling diphtheria in this respect. The writer is of the opinion that these sporadic cases are of more frequent occurrence than is generally conceded to be the case. The infrequency with which postmortem examinations, particularly those of the head, are made, probably accounts for their apparent rarity.

Age.—The disease attacks children and young adults chiefly, although cases may also occur among persons more advanced in life. In Chicago quite a large per-

centage of cases occurred between the ages of 30 and 40 years, as is shown by the following figures: Under 1 year, 3; between 1 and 5 years, 6; between 5 and 10 years, 3; between 10 and 15 years, 1; between 15 and 20 years, 7; between 20 and 25 years, 6; between 25 and 30 years, 2; between 30 and 40 years, 7; over 40 years, 3. Total, 38. These figures coincide with those found in the majority of recent epidemics. During those occurring in the early part of the present century, adults were less often affected, the disease occurring almost exclusively in young children.

Sex.—Sex does not seem to be a factor of much importance in the etiology of this disease, although males appear to be rather more liable to contract it than females. In our series of cases there were twenty-four cases in males and fourteen in females.

Season.—The majority of epidemics have occurred during the latter part of winter and during spring, the greatest number of cases in Chicago being during April and May. Berg[76] gives some interesting statistics on the number of deaths from cerebrospinal meningitis in New York City during the years 1892 and 1893; they are as follows:

	1892	1893
January	15	22
February	8	19
March	30	53
April	27	54
May	30	107
June	23	58
July	27	52
August	20	25
September	15	19
October	12	24
November	14	19
December	9	17
Totals	230	469

Meteorologic factors.—It has been frequently noticed that epidemics of cerebrospinal meningitis occur after a cold winter followed by a cold, wet spring. Such was the case, among others, in the epidemic

described by Ford[77] and Brooks[78] in central New York. The meteorologic data for Chicago were as follows:

Month.	Temperature. 1897–98.	Mean for twenty-five years.	Monthly precipitation, 1897–98.	Mean for twenty-five years.	Prevailing direction of wind, 1897–98.
September	70	64	0.84	2.93	N.E.
October	58	52	0.18	2.93	S.E.
November	38.7	38	3.06	2.82	S.
December	25	30	1.62	2.34	W.
January	28	23	3.54	2.11	W.
February	28	27	2.59	2.31	S.
March	40	34	4.60	2.40	S.
April	44	46	0.76	3.22	N.
May	56	56	2.23	3.72	N.E.
June	69	67	5.30	3.80	N.E.
July	73	72	1.94	3.49	N.E.
August	72	73	3.03	2.98	S.W.

From the above tables it will be seen that although the winter of 1897–98 was not very cold, it was a very wet one; the months of January, February and March presented very changeable weather, with a great deal of slush and rain.

Association with other diseases.—There is no distinct connection between the occurrence of cerebrospinal meningitis and the prevalence of other diseases occurring simultaneously in a community. Typhoid fever has in some instances, among others in Litchfield and Norwich, followed an epidemic of cerebrospinal meningitis. Epidemics of pneumonia have also frequently occurred simultaneously with epidemics of cerebrospinal meningitis, but these coincidences are considered by modern writers to be purely accidental.

Relative prevalence in city and country.—It has been generally noted that this disease is comparatively more prevalent in farming districts and smaller towns than in our large cities. Osler[29] states that in 1873 the disease was very prevalent in the rural districts of the Ottawa River valley, while the cities of Ottawa and Montreal remained comparatively immune. There is no reason known that will explain this fact satisfactorily.

Nasopharyngeal disease as a predisposing factor.—Weichselbaum, Yaeger and others have expressed it as their opinion that in the majority of instances, the germ which causes the meningitis gains access to the meninges through the nasal cavities, and the meningococcus has frequently been found in the nasal secretions. Considering this fact, it appears plausible that when the mucous membrane of these areas is in a diseased condition, it will afford a better nidus for the growth of the germ and its conveyance to adjacent parts. A large number of observers, among them Richter, Strümpell, Councilman, Mallory and Wright, have found nasal catarrh in a large percentage of the cases observed by them.

Overcrowding, etc.—The disease has been often noted in overcrowded transport vessels, jails, etc. It is, however, hardly probable that the overcrowding in itself should have been the predisposing cause, except in so far as to render the observation of sanitary rules more difficult. The bacteriologic causes, and the influences which unsanitary conditions exert over the disease will be considered elsewhere.

PATHOLOGY AND BACTERIOLOGY.

The pathology of epidemic cerebrospinal meningitis is essentially that of a purulent inflammation of the inner coverings of the brain and cord. The exudate is often localized in the basal region of the brain, more particularly in the neighborhood of the cerebellum, frequently, however, the purulent exudate covers the entire convexity in a thick layer, The greatest accumulations of pus are usually found along the course of the larger blood-vessels, and in the fissures and sulci of the convexity of the brain. But rarely—and then only in cases of a very virulent character—has an entire absence of exudate been found; in these cases there was only a serous effusion, and in some few cases, only an intense congestion of the meninges. The spinal cord is usually affected in a similar degree, generally being covered with an

extensive layer of purulent exudate. The more recent the process, the more purely purulent is the exudate, often bearing close resemblance to gonorrheal pus. In cases of longer duration the pus is frequently caseous in character and has undergone more or less fatty degeneration. Very frequently the ventricles contain an exudate, purely purulent in more recent cases, while in those of longer standing the ependyma is coated with crumbly, cheesy masses. As a rule the brain substance itself is not implicated in the process, Ghon, however, in his series of postmortems in Trifail, found in the brain substance of some of the cases, purulent encephalitic areas, either localized in the cortical area or studding the entire brain substance.

In a postmortem made by Thompson,[55] the cord and brain were so extensively degenerated as to be unfit for microscopic examination. In the spinal cord the posterior surface is chiefly affected, the exudate being greatest in the lumbar region. The substance of the cord is frequently the seat of minute hemorrhages. Councilman[44] states that in the more chronic cases the most marked condition is the edema and general thickening of the meninges. The change is most marked in those areas where the acute process is most evident. Along the vessels the meninges are thickened and whitish, with little exudation. At the base the meninges are opaque, enormously thickened, and there are bands of organized tissue extending from point to point. Microscopically the main change is a purulent infiltration of the meninges and the superficial layers of the cortex of the brain. The pus-cells may be found either closely packed together or scattered here and there; sometimes they are enclosed in a network of fibrin. The fixed tissue-cells, as a rule, show little change. The blood-vessels are injected and, usually, those of the meninges are packed with lencocytes. Thrombi are also occasionally met with.

Among the chief pathologic changes in the other organs are inflammations of the bronchi and lobular

pneumonia; these pneumonias were formerly considered by various authors to be the starting-point of the infection; they are now generally looked upon as accidental complications, due to the diplococcus pneumoniæ. The heart muscle is generally found to present areas of parenchymatous degeneration; sometimes fatty degeneration is also found. The liver is usually similarly degenerated, with considerable difference in the degree of the degeneration in the different cases. In uncomplicated cases the spleen is seldom much enlarged. The stomach and intestinal tract present no constant lesions; the kidneys are generally found to be more or less degenerated. In the epidemic at Trifail, Ghon found in a number of cases a very intense desquamative catarrh of the mucous membrane of the pelvis and pyramids; frequently this condition was associated with numerous hemorrhagic spots in the areas mentioned.

Biggs[65] found in one case innumerable hemorrhages in the wall of the bladder, associated with some superficial erosions and deposits of fibrin, which process had extended up the ureter and involved the pelvis of the kidney. The pathology of the accidental complications presents nothing characteristic of the disease.

The bacteriology of this disease has been a question which has given rise to much discussion among writers on this subject; at this date, however, the evidence seems to be in favor of the diplococcus intracellularis meningitidis being the causative factor in the majority of cases. This organism was first described by Weichselbaum, in 1887, who found it in the meninges of six cases of acute cerebrospinal meningitis. As described by Mallory,[44] it is a micrococcus of about the same size as the ordinary pus micrococci, and appears in diplococcus form of two hemispheres separated by an unstained interval. It stains with any of the ordinary stains for bacteria, and is decolorized by the Gram method of staining. There is considerable irregularity in staining, some organisms being more brightly stained than the other. There may also be con-

siderable variation in size, and the larger organisms stain imperfectly. In the smaller organisms there is often a brightly stained point in the center, while the remainder of the cell is scarcely colored. These variations in size and in staining appear to be due to degeneration, and are more common in old than in fresh cultures. The two organisms are usually sharply separated, but in some there seems to be a small amount of material uniting them. Division commonly takes place in one plane, giving rise to diplococci; tetrads are occasionally seen. There is little or no tendency to growth in the streptococcus form, although short chains of four to six organisms may be found. In cultures the organism does not grow profusely; blood serum is the best culture-medium, while on agar the growth is scanty; in bouillon the growth is also feeble, the medium becoming slightly cloudy.

Ghon of Vienna considers the meningococcus as closely allied to the gonococcus, especially as its method of division is identic and also since it, as well as the gonococcus, is found within the cells. According to Ghon, the meningococcus grows poorly on glycerin-agar, fairly well on plain agar, and best of all on blood-serum-agar, on which culture-medium it forms colonies identic with those of the gonococcus. On bouillon it forms a thin scum on the surface, after a few days' growth, in this respect also resembling the gonococcus. At ordinary room temperature there is no growth, this being the reason that it does not grow on gelatin. On potato there is a feeble growth. In order to have a continuous growth of the meningococcus, it is necessary, according to Ghon: 1, to protect the cultures from drying out; 2, to keep the cultures in a thermostat, at a constant temperature, not below 25 C. By observing these rules it has been possible for him to keep cultures of the meningococcus viable for seventy days; to the gonococcus the same rules apply, with like results when observed. In regard to the number of meningococci found in the cerebral exudate, the same author states that as a rule

in cases of only a few days' standing the number is very great, cell upon cell being filled with the organisms, presenting a picture identic with that found in acute gonorrhea. After a few days the number of cocci diminishes greatly, and it is just this absence of bacteria which is, according to him, characteristic of epidemic cerebrospinal meningitis. The disease may relapse, and then the cocci will again be present for a few days. In the nasal secretion, even in the absence of a nasal catarrh, both Jaeger and Ghon have found large numbers of meningococci. Fronz,[66] in a case of arthritis complicating cerebrospinal meningitis, obtained a pure culture from the joint, no other bacteria being present. Bonome[67] thinks a variety of organisms can cause epidemic cerebrospinal meningitis, which he classifies as follows:

1. The diplococcus intracellularis meningitides.

2. Streptococcus pyogenes, found present in one case by Neuman, and four times by Netter in isolated cases.

3. A short thick bacillus found by Neuman and Schaefer; it resembles the typhoid bacillus, but its growth on potato is different.

4. An encapsulated bacterium which was found in one case due to otitis media; it resembles the pneumobacillus of Friedlaender.

5. The diplococcus pneumoniæ of Fraenkel, which is the most frequent causative factor of cerebrospinal meningitis. Holt and Prudden,[68] in their description of a case of cerebrospinal meningitis, in 1891, considered the pneumococcus as the offending micro-organism.

Biggs,[69] in a series of cases from which cultures were made, found no one constant organism; the majority of the cases showed the diplococcus pneumoniæ, which he considers as the chief etiologic factor; other organisms found were the pneumococcus of Friedlaender, bacillus of influenza and the streptococcus pyogenes; in one case the bacillus coli communis was found in the exudate from the base of the brain. In

cases complicated with dysentery, occurring during the epidemic at Lonaconing, the stools, according to Flexner and Barker,[41] contained an organism identic with the micrococcus lanceolatus. Klemperer[70] found pneumococci in the watery contents of herpes, occurring in cases of cerebrospinal meningitis. Onadu[71] found the diplococcus of pneumonia in the blood of a meningitis patient. Spitzer[72] also considers the pneumococcus as the cause. As the meningococcus in a superficial examination may be confused with the pneumococcus, it is possible that some of the cases reported as being due to the pneumococcus were really caused by the meningococcus. To differentiate the two, Heubner[73] states:

1. The meningococci are found chiefly in the pus-cells, less so in the fluid; pneumococci found in fluid.

2. They always occur in pairs, with the broad sides against each other, while the pneumococci join with their narrow sides.

Jaeger[73] also calls attention to these differential features and states that they are constant. To differentiate between the meningococcus and the gonococcus, Kiefer[74] states that the gonococci are more uniform in size, that in the exudate from a case due to the meningococus about 90 per cent. of the leucocytes are filled with the cocci, while in gonorrheal pus only a small number of leucocytes contain the organism. This author, however, does not agree with Jaegar in regard to two differential points, viz., that the gonococci do not occur in the nuclei of cells and that the meningococci found within the nuclei possess a capsule. These two statements Kiefer opposes, and states that the gonococci are also not infrequently found within the nuclei; and that the picture then presented by them is identic with that shown by the meningococci. The writer is of the opinion that if the meningococcus and the gonococcus are not identic organisms, they are at any rate closely related, as is shown by the comparative frequency with which young men affected with gonorrhea are attacked by meningitis during the

presence of an epidemic of this disease; we refer in this respect to a report by Fuerbringer[75] and to cases occurring during the Chicago epidemic.

SYMPTOMS.

Among the different classifications of this disease, that of Hisch[49] is probably the best, although like every subject whose different phases approach each other, this disease can not be divided into distinct forms without there being some cases which belong partly in one form and partly in another. Hisch's classification is: 1, meningitis cerebrospinalis epidemica siderans; 2, meningitis cerebrospinalis epidemica abortiva; 3, meningitis cerebrospinalis epidemica intermittans; 4, meningitis cerebrospinalis epidemica typhoides.

Under the first form come those cases which commence with violent initial symptoms, agonizing headache, backache, chills, high temperature, uncontrollable vomiting, coma and death within a period varying from twenty-four hours to two or three days. Cases of this nature were of frequent occurrence in the older epidemics, but have of late years become rare.

That type called the abortive[50] has also been of unfrequent occurrence in the later epidemics. In it the initial symptoms are also of a marrow-freezing nature, resembling in severity those of the first form described, but instead of ending fatally, the patient after two or three days makes a remarkably quick and permanent recovery. Cases resembling the description of this form were not unfrequently found in young children during the Chicago epidemic, but the consensus of opinion was against their being true cases of cerebrospinal meningitis; the vast majority being found in cases where dietary errors had been committed, recovery generally following a cleaning of the gastro-intestinal tract.

The cases which might be classified as intermittent are usually those which run a protracted course, lasting six to eight weeks or longer. In these lengthy

cases it will sometimes occur that all alarming symptoms disappear, sometimes with surprising rapidity, and the patient be apparently on the road to recovery, when a relapse occurs, the case taking possibly a fatal ending.

Cases of medium duration are by far the most frequent, lasting two to four weeks, and frequently passing into a sort of typhoid condition. The disease begins as a rule quite suddenly, with severe headache, chiefly in the occipital region; pain in the cervical region and stiffness of the neck, and a general feeling of debility. Quite frequently there will be vomiting, which, especially in children, may be uncontrollable. With these initial symptoms there is as a rule some rise in temperature. In the majority of cases disturbances of the sensorium soon follow in the wake of the other symptoms; they vary in severity from simple dizziness and drowsy feeling to a distinct delirium. An initial chill is sometimes met with.

The headache in cerebrospinal meningitis is usually very intense; as a general rule it is most marked in the occipital region, extending down the back and in some cases radiating into the shoulders. The pain may be of a throbbing character which keeps up constantly, even when under the influence of opiates. In some of the severer cases, the patient will frequently moan and roll about, denoting a pain which, in a large number of instances, death alone can conquer. In a smaller number of cases the pain is neuralgic in character, there being periods of comparative ease between the paroxysms. With the extension of the inflammatory process to the spinal meninges, there is tenderness of the entire vertebral column, which through contracture of the erector muscles of the spine is stiff and straight, sometimes even decidedly opisthotonic, the head being drawn backward. The cranial and spinal nerves in this disease present an interesting variety of disturbances, most frequent among them being those affecting the motor nerves of the eye, such as inco-ordinate movements of the eyeballs, nys-

tagmus, inequality in size of pupils, and tardy reaction to light and accommodation.

Lagophthalmus was noticed by Blümm.[51] Ptosis was an early prominent symptom in the Viennese cases described by Schroetter.[52] In the territory supplied by the facial nerve, muscle contractures are of frequent occurrence, giving the face a peculiar drawn expression. The nerves of special sense are also often implicated, so for instance Stovell[53] describes a case in a child where there was temporary blindness extending over a period of five months, the optic disc being bluish and indistinct; a similar case was also reported by Presser.[54] Thompson[55] and Randolph[56] report cases of trophoneurotic ulceration of the cornea; Knapp[52] saw ten cases of cerebrospinal meningitis with intraocular disease, in all but one, blindness being the result; this complication came on during the second or third week of the disease, and, after a course of three to four days, ended in blindness. Knapp considered it as a hyperplastic choroiditis with consecutive retinal detachment. Flexner and Barker,[58] in a description of an epidemic in Lonaconing, state that out of thirty-five cases the fundus oculi was normal in only seven. Difficulty in hearing, and other disturbances of the ear, have been of frequent occurrence in some epidemics, among others in the one described by Blümm.[51] The auditory nerve itself may be influenced and pus may be found in the labyrinth. Disturbances of the spinal nerves are less frequent, among the most frequently noted being hyperesthesias and twitchings of the muscles of the extremities. Contractures of the limbs have also been met with.

Belor,[59] in his description of an epidemic in Aranzo, states that hemiplegia was of frequent occurrence. Bernhardt[60] mentions a case in which there was paraplegia, followed by paralysis of both the upper and lower extremities, while the bladder and rectum remained intact. Among other forms of paralyses may be mentioned a case by Gahlberg,[61] in which there was paresis of the tongue, and one by ——,[62] which

was followed by a loss of the power of articulation. The mucous membranes were affected in a number of epidemics described. Berg[63] states that in the epidemic of 1893, in New York City, conjunctivitis and photophobia were present in almost every case. Strümpel says a considerable number of his cases were preceded by nasal catarrh. The reflexes are not uniform in their behavior in this disease; the skin reflexes as a rule are somewhat exaggerated, while the tendon reflexes are sometimes so affected; in other cases they are retarded. In a large number of cases there is no deviation from the normal. Skin symptoms were very prominent in the earlier epidemics; in the later ones, a herpetic eruption on the lips and face was the only cutaneous lesion to which importance could be attached. Other eruptions, such as roseola, urticaria, petechiæ, etc., have also been observed. Disturbances of the gastro-intestinal tract other than the initial vomiting are rare. Loss of appetite and more or less irregularity of the fecal movements occurred in the severer cases, but presented nothing characteristic. The spleen is sometimes enlarged, but rarely to the extent of the typhoid spleen. Multiple joint lesions have been observed in some epidemics. Kotsonopulos,[64] in an epidemic described by him, found acute inflammation of the joints in a large number of cases, as did also Berg[63] in the epidemic in New York in 1893.

The genito-urinary apparatus is not as a rule affected, although albuminuria has been of frequent occurrence in some epidemics; polyuria and glycosuria have also been observed. The lungs are frequently affected in the severer forms of the disease; hypostatic and "schluck" pneumonia occur occasionally in the later stages. Anatomic disturbances of the heart are rare, endocarditis being the one most often found. The pulse-rate is usually slightly increased, but not in proportion to the height of the temperature. Leucocytosis is almost constantly present. The temperature shows no regularity, and its height is not proportionate to the severity of the disease; fatal cases

sometimes run their course with little or no rise in temperature. The majority of cases show an irregularly remittent type of fever. There is no distinct evening rise in temperature; in some cases, the morning temperature will even exceed that of the evening. Increasing severity of the cerebral symptoms is not as a rule attended by a corresponding rise in temperature. In fatal cases there is often hyperpyrexia shortly before death, temperatures of 109 and 110 F. having been observed. After the disease has run its course favorably, the patient is, as a rule, left in a greatly exhausted and debilitated condition. Temporary or permanent deafness is a frequent sequence of the disease. Disturbances of vision often remain, giving evidence of intraocular lesions. Permanent paralyses of the motor muscles have also been observed after recovery, but are comparatively rare. Where the disease has occurred in young children, the mental faculties frequently remain permanently impaired.

DIAGNOSIS.

During the presence of a well-marked epidemic, the diagnosis of cerebrospinal meningitis as a rule presents little difficulty. More difficulty is encountered in the diagnosis of sporadic cases, especially when they come under observation for the first time when already in a delirious or unconscious condition, as is frequently the case in those sent to the hospitals, especially if no history is available. In these cases it becomes necessary to exclude typhoid fever, tubercular meningitis, miliary tuberculosis and some of the severer septic disturbances. From a diagnostic standpoint, the most important symptoms are the characteristic headache,the rigidity and tenderness of the nape of the neck, the herpetic eruption and the well-marked leucocytosis. If it can be made, a lumbar puncture will usually show conclusive evidence as to the nature of the disease. During the Chicago epidemic, typhoid fever was sometimes confused with this disease. Below are given some of the differential features between the two conditions:

CEREBROSPINAL MENINGITIS.	TYPHOID FEVER.
1. Onset, as a rule, sudden with nausea, vomiting and frequently a chill or chilly sensation.	1. Onset, as a rule, gradual, with malaise and bronchial catarrh.
2. Temperature during early stage very irregular, may be high or may be normal. No step-like increase. (See charts.)	2. Temperature during early stage usually regular between 100 and 108 degrees F. Step-like increase in the evening, temperature of each day being slightly higher than that of the preceding day.
3. Headache very agonizing.	3. Headache of a less severe type.
4. Tenderness of cervical column and retraction of head.	4. Tenderness of abdomen, cervical column normal.
5. Abdomen retracted, boat-shaped.	5. Abdomen prominent, often tympanitic.
6. Herpetic eruption on lips, sometimes petechial spots.	6. Rose spots on abdomen.
7. Spleen rarely enlarged.	7. Spleen enlarged and palpable.
8. Conjunctivitis and photophobia of frequent occurrence.	8. Very rare.
9. Fever irregular, no constant evening rise.	9. Temperature during fastigium uniformly high, with evening exacerbation.
10. Pulse-rate not as a rule very frequent, does not increase with temperature, except in some instances.	10. Pulse rapid, its increase is generally proportionate to the increase in temperature, often dicrotic.
11. Urine as a rule normal, diazo reaction negative.	11. Urine often slightly albuminous. Diazo reaction positive.
12. Blood shows large and constant increase in leucocytes. Widal test negative.	12. Leucocytosis exceedingly rare. Widal test positive.
13. Joints frequently affected.	13. Joints rarely affected.
14. Bowels as a rule regular.	14. Obstipation during early part of the disease; after second week, slight diarrhea with "pea-soup" stools.
15. Tongue has no characteristic coating, frequently not coated at all.	15. Tongue coated thickly in center, edges and tip are clean.
16. Convulsions in children and twitching of muscles in adults frequent.	16. Convulsions and twitchings of muscles rare.
17. Paralysis of frequent occurrence.	17. Paralysis very rare, and then only as sequelæ.
18. Disturbances of sensation (hyperesthesia, etc.).	18. Not present.
19. Lumbar puncture shows increased pressure in spinal canal, and a superabundance of cerebrospinal fluid.	19. Lumbar puncture negative.
20. Duration of disease very variable.	20. Duration of disease usually three to four weeks.

Tubercular meningitis.—The diagnosis of this disease from the epidemic form sometimes offers considerable difficulty, especially if there is no clear family history and an absence of tubercular lesions elsewhere. The chief differential points are as follows:

EPIDEMIC CEREBOSPINAL MENINGITIS.	TUBERCULAR MENINGITIS.
1. Family history as a rule negative.	1. Generally tubercular family history, and tubercular lesions in other parts of the body, most often in lungs.
2. Occurs at all ages, most frequent between the ages of 10 and 20.	2. Cases generally occur in children under 10 years of age.
3. Onset sudden.	3. Onset gradual.
4. Delirium early in disease.	4. Delirium late in disease.
5. Temperature as a rule comparatively high.	5. Rise in temperature as a rule very slight.
6. Occurs as a rule in epidemics.	6. Is never epidemic.
7. Affects chiefly the convexity of the cerebrum.	7. Affects chiefly the base of the brain.
8. Cerebrospinal fluid during acute stage contains meningococci, later sterile.	8. Cerebrospinal fluid contains tubercle bacilli.
9. Herpes frequently present.	9. Herpes never occurs.

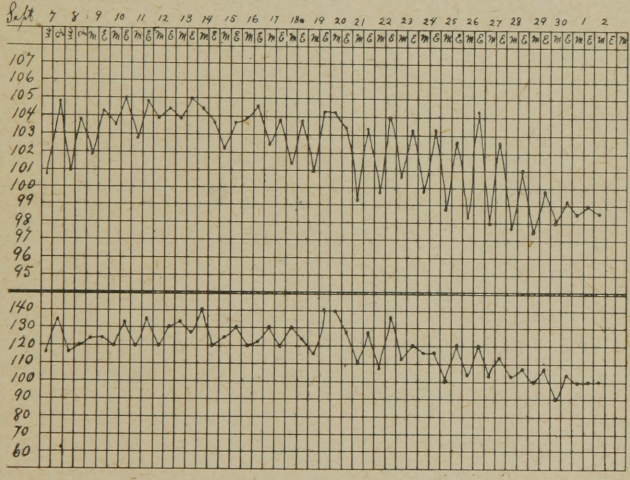
The meningitic symptoms in cases of *pneumonia* are sometimes of such a nature as to resemble true cerebrospinal meningitis. There are, however, in these cases, rarely retraction of the muscles of the neck or opisthotonos. The eye and joint symptoms are also as a rule absent. Undoubtedly, however, pneumonia and cerebrospinal meningitis caused by the meningococcus may exceptionally occur together. Certain *septic conditions* may sometimes simulate cerebrospinal meningitis. A careful search for the causative factor and a consideration of all the symptoms will, as a rule, soon clear the diagnosis. In some epidemics of *influenza* cases have occurred which resembled cerebrospinal meningitis; when any doubt exists in this respect, however, a bacteriologic examination of the mucus from the nasopharynx will as a rule speedily clear the diagnosis, as that from an influenza patient generally teems with Pfeiffer's bacilli.

PROGNOSIS.

The physician, in expressing an opinion as to the probable outcome of a case of cerebrospinal meningitis, should avoid all positive assertions. Although, as a rule, in the epidemics of later years the majority of patients have recovered, the mortality seldom exceeding 30 per cent., the outcome of any individual case is always doubtful; and even should the disease not end fatally, its ravages often leave the patient a

mental and physical wreck. In estimating the patient's chances as to recovery, the condition of the pulse and temperature offer no index; more to be relied upon are the cerebral symptoms. A delirium of a noisy character, the patient tossing about and grasping at imaginary objects, augurs ill; a prolonged state of unconsciousness, especially when setting in early, is also of grave significance.

Severe vomiting, which is difficult to control, means a fatal ending, or at any rate slow convalescence on



Temperature and pulse chart. Typhoid fever (Osler).

account of the exhausted condition in which it leaves the patient. Involuntary evacuations of urine and feces denote extensive destruction of nerve tissue. Dysphagia is an almost certain sign of approaching dissolution. Ulceration of the cornea or intraocular lesions usually mean an extensive and virulent type of inflammation. On the other hand, if the vomiting is easily checked and a fair amount of food is assimilated, the mind being only slightly clouded, and

the second or third week of the disease being reached without any of the unfavorable symptoms previously mentioned occurring, the prognosis may be set down as favorable, even though the temperature remains high. Unforeseen complications which may occur and frequently do occur in apparently the most favorable cases, must be taken into consideration when expressing an opinion.

TREATMENT.

Little can be said on this subject, as the treatment of cerebrospinal meningitis is at present still purely symptomatic. The therapeutic value of lumbar puncture is still doubtful. Although some cases where it has been performed with apparent beneficial results have been reported, the amelioration of the symptoms in these cases was probably due to the reduction of intracranial pressure exerted by the fluid withdrawn. Ice-bags to the head and spine are almost universally used and have a tendency to diminish the pain. Hot baths have recently been advocated by German writers, but the philosophy of this mode of treatment is not quite clear. Narcotics are indicated if the pain is very severe. Considering the probable origin of the disease, frequent nasal douches with mild antiseptics are considered advisable as a prophylactic measure.

The cases here cited occurred under the observation of a number of skilled diagnosticians. All uncertain and doubtful cases were eliminated, thus curtailing the number, but we hope its value is thereby increased. One regrettable feature is that a large number of post-mortem records were not available. The records are as exhaustive as was considered necessary, although varying somewhat in detail by being collected from a number of hospitals.

Case 1.—Male, aged 24, admitted July 1, 1898; has been sick six days; last Saturday had a chill, followed on Tuesday by another, with vomiting, headache, backache, diarrhea, fever, delirium, rigidity and tenderness of neck and back; eyes congested, dull; tongue thickly coated; gurgling in iliac fossæ;

slight tympanites; rigidity and retraction of abdomen; temperature 103 degrees F.; pulse 74; lumbar puncture made, no fluid obtained; discharged recovered, Aug. 3, 1898.

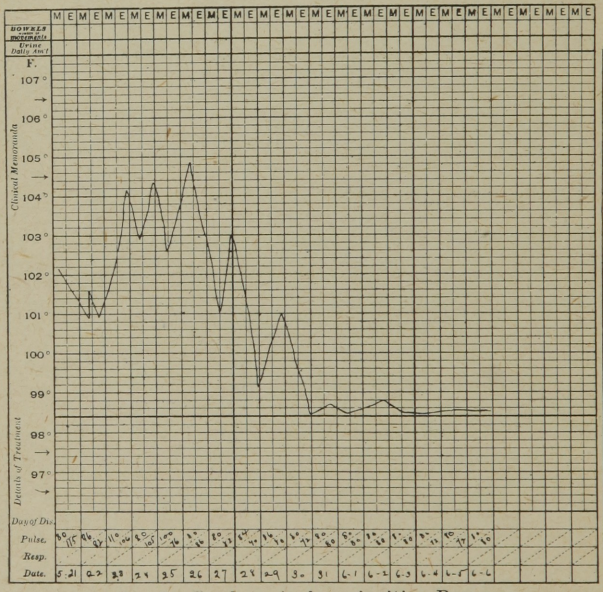
Case 2.—Male, aged 45, admitted June 17, 1898; five days before taken suddenly with vomiting and diarrhea, followed by severe headache, backache and pains in the extremities; when first seen complained of severe headache; mind very dull; patient constantly stroked his head and neck with his hands. and groaned; great tenderness on pressure; pupils contracted and equal, reacted normally; no ocular nor other paralysis; pulse 48, regular, full and soft; temperature 98; respiration 28; no eruption; the night following was in continuous pain and very restless; morphin. The pulse remained slowed and the temperature on the day following ranged between 100 and 101 degrees. During the following day the pulse remained slow, while the temperature on the 22d reached 103 degrees, with a pulse of 68. Headache and mild delirium continued. No new symptoms were observed except a slight right sided facial paresis. An opthalmoscopic examination on the 22d showed no change in the fundus; there was a marked cupping of the right disc. The delirium became more marked and continuous; headache and tenderness with rigidity of the neck persisted; but under the influence of bromids or morphin the patient slept four to six hours during the night. The pulse increased in frequency; on the 25th it ranged from 100 to 120; temperature 100–102.4 degrees. Lumbar puncture was made on the 26th, 20 c.c. of fluid being removed, from which cultures were made showing the diplococcus of Weichselbaum. The puncture was followed by apparently beneficial results; patient discharged recovered. (Case reported by Bassoe, JOURNAL AMERICAN MEDICAL ASSOCIATION, July 23, 1898.)

Case 3.—Male, aged 7, admitted April 3, 1898; taken sick three days previous with chill, vomiting, headache, rise in temperature. On admission is delirious, head retracted, eruption on lips, tenderness along spine; temperature 100 degrees; pulse 78. April 6, abdomen retracted, pupils dilated, most so on left, delirious. April 10, patient somewhat better, delirium abated. April 12, complains of pains in wrists, tender on pressure. April 18, temperature normal, patient takes more interest in surroundings. May 5, discharged recovered. No lumbar puncture was made.

Case 4.—Female, aged 12, admitted April 7, 1898; history of sudden onset with vomiting and severe headache; on admission, delirious, head retracted, spine tender, slight strabismus, abdomen retracted; temperature 103 degrees; pulse 122: temperature very irregular during the course, as high as 105.5 one day, the next day not over 100 degrees. Discharged in about two months; no lumbar puncture.

Case 5.—Male, aged 40; taken sick June 10, 1898, with severe head and backache, also vomiting. On admission, June

14, there was severe headache, causing sleeplessness; the neck rigid and painful, with but slight retraction; no paralysis; over abdomen and thighs were many petechial spots; mind clear, but the patient constantly complaining of his head. He lived until 10:45 P.M., June 19, dying on the tenth day from the onset. During the last thirty six hours the pulse became more rapid. He never vomited after admission. He gradually became wildly delirious. The petechial spots faded somewhat before death, no new ones appearing. There was no herpes. No paralysis was at any time made out. Post mortem: Entire pia infiltrated with a thick purulent fluid, especially marked

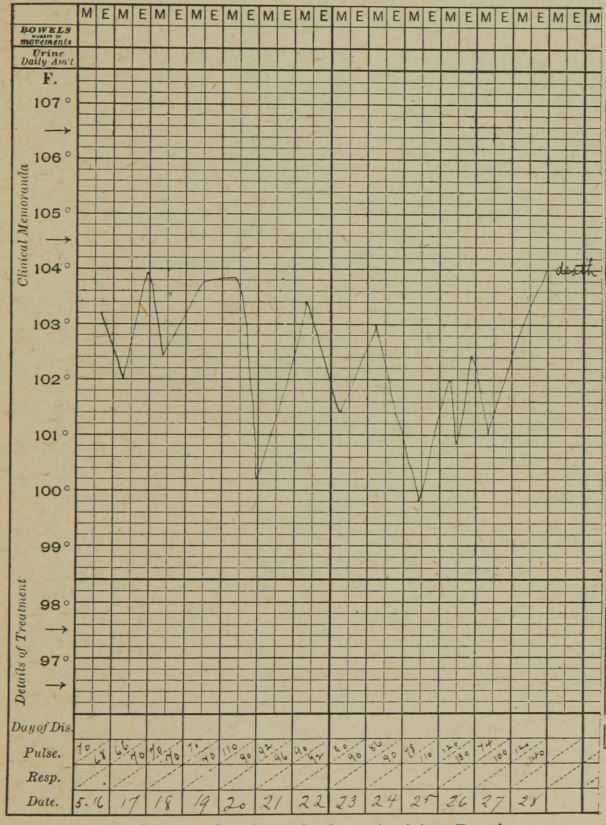


Case 7.—Cerebrospinal meningitis. Recovery.

about posterior margins of the cerebellum. The amount of cerebrospinal fluid is small, but the lateral ventricles contain a quantity of purulent fluid, which in the horns becomes thick pus; walls of the ventricles soft. There is exudate around the upper end of the spinal cord, but the spinal canal was not opened. Cultures from the meningeal exudate showed a coccus, with all the characteristics of the diplococcus intracellularis meningitidis. (Case reported by Herrick, JOURNAL AMERICAN MEDICAL ASSOCIATION, July 2, 1898.)

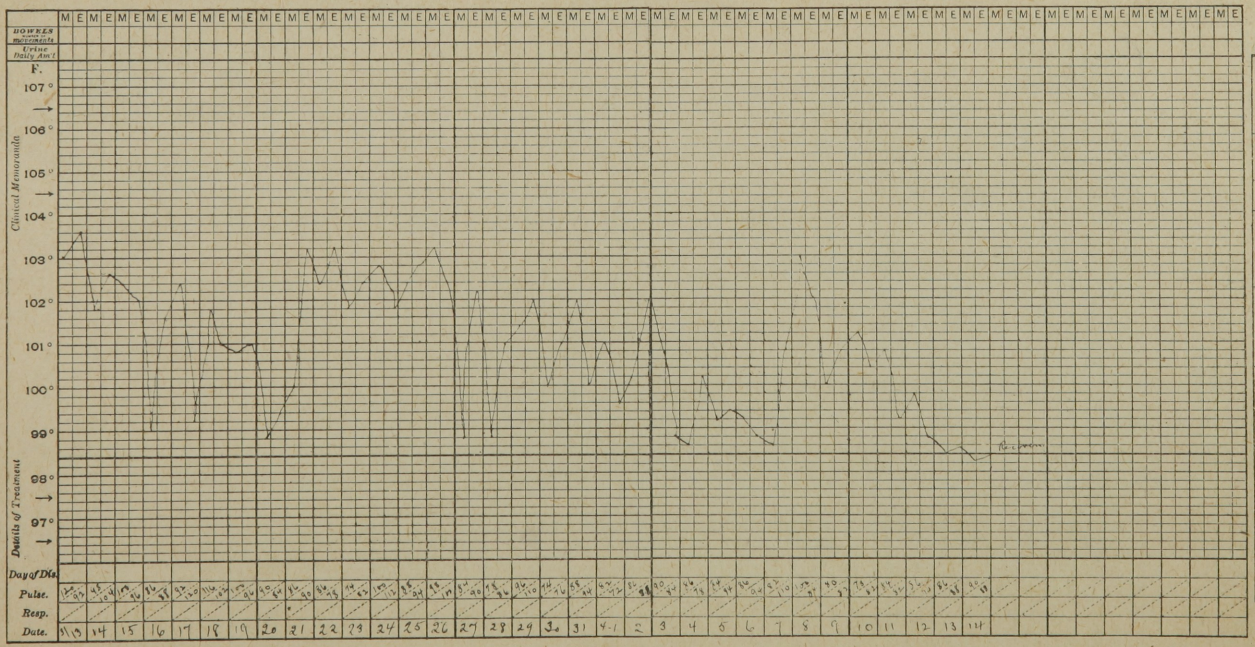
Case 6.—Male, aged 45, admitted June 24, 1898; very deliri-

ous; temperature 101.2 degrees; taken sick two or three days before; had severe pains in back; headache, soon followed by delirium; eyes congested, boat-shaped abdomen, coma, casts; lumbar puncture made, and 30 c.c. of clear fluid removed; pressure in spinal canal, 205 mm.; sp. g. of fluid, 1006; bacteriologic examination of fluid negative; patient lived twenty-four hours after admission; no post-mortem.



Case 8.—Cerebrospinal meningitis. Death.

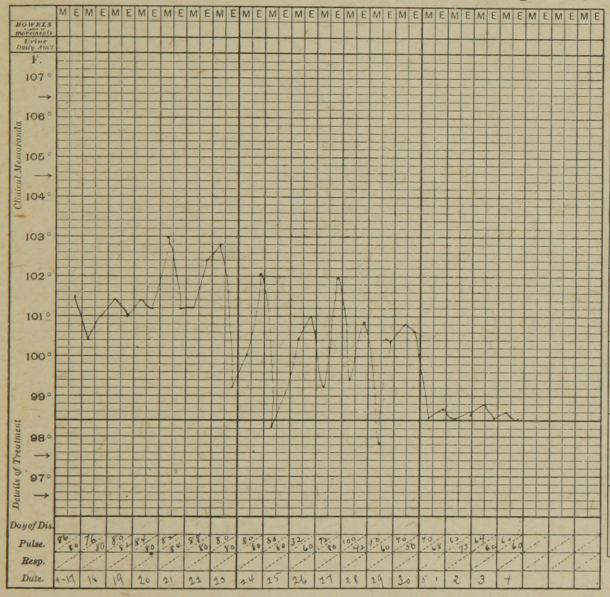
Case 7.—Male, aged 24, admitted May 21, 1898; about six to eight days before, began to complain of head and backache; about three days afterward had a chill, followed by vomiting,



Case 9.—Cerebrospinal meningitis. Recovery.

severe headache, pain in abdomen, fever and delirium; tongue coated in center; gurgling and tenderness in iliac fossa; spleen enlarged and palpable; herpes; head retracted, tenderness over cervical vertebræ; blood examination for typhoid, negative. This case showed only a slight decrease in temperature after a bath was given. Lumbar puncture was not made. Delirium and other symptoms gradually abated; patient discharged June 9.

Case 8.—Male, aged 15, admitted May 16, 1898; about eight days before, complained of pains in arms, eyes congested, bone

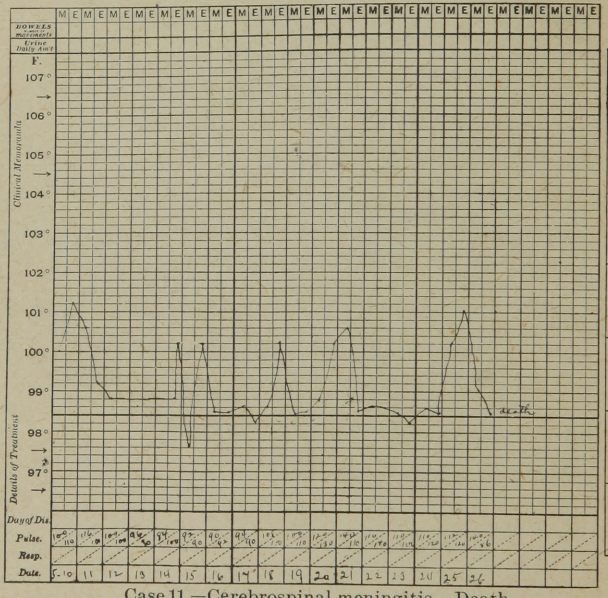


Case 10.—Cerebrospinal meningitis.

and backache; then he lay on the floor in a draught a couple of hours, asleep; next day had a severe headache and vomiting, fever and delirium; pain in back of neck, with retraction of head; delirious and restless; tongue coated; constipated; May 20, trophoneurotic ulceration of left cornea; catarrhal conjunctivitis of left eye; May 26, facial paralysis on left, May 28, general paralytic condition; puncture between third and fourth lumbar vertebræ, 65 c.c. of clear fluid removed; pressure in spinal canal, 750 mm.; sp. gr. of fluid, 1007, slightly albuminous; bacteriologic examination negative; died May 28; no post-mortem.

Case 9.—Male, aged 22, admitted March 13, 1898; pain in back of neck, back and legs, came on very suddenly, with chill, nausea and vomiting four days previous to admission; has persisted since: most severe in back of neck; retraction of head; cries out with pain when head is flexed; pulse 120; temperature 103 degrees F.; tongue coated; abdomen boat-shaped; reflexes increased; muscles tender; no albumin in urine; no puncture; patient discharged April 22.

Case 10.—Male, aged 21, admitted April 19, 1898. Four days previous to admission he was taken with a very severe headache, the next day had a chill, and then ached all over; last

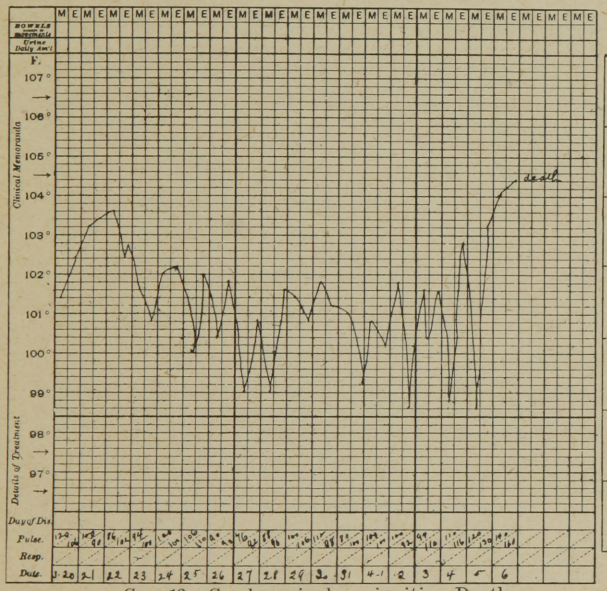


Case 11.—Cerebrospinal meningitis. Death.

three days has vomited everything that he has taken; two days ago began to have stiffness in the neck, was constipated, and suffered from insomnia; well nourished, pale, nystagmus, increased patellar reflex; on standing is very dizzy; tongue slightly coated, spleen enlarged and palpable. Patient made a gradual recovery, and was discharged May 15; no puncture.

Case 11.—Male, aged 18, admitted April 29, 1898. The patient had been complaining for two weeks, of headache, pain in limbs, chilliness and weakness, also had a cough; April 20, had cough, pain in abdomen and tympanites; April 28, very

restless at night, could not sleep, very severe headache, anodynes did not produce sleep; April 29, delirious, rigidity of neck first noticed yesterday morning, left hand hyperemic and puffy; April 30, severe chill lasting thirty minutes; May 2, involuntary evacuations of urine and feces; muttering delirium; patient has double vision; May 4, patient complains of severe pain in pit of stomach; May 6, some improvement, slept well; May 7, not much change; May 10, slightly delirious, subsultus tendinum very marked; May 12, very restless and delirious, involuntary evacuations; May 20, less delirious; May 23, pupils very much dilated; May 25, pulse very irregu-

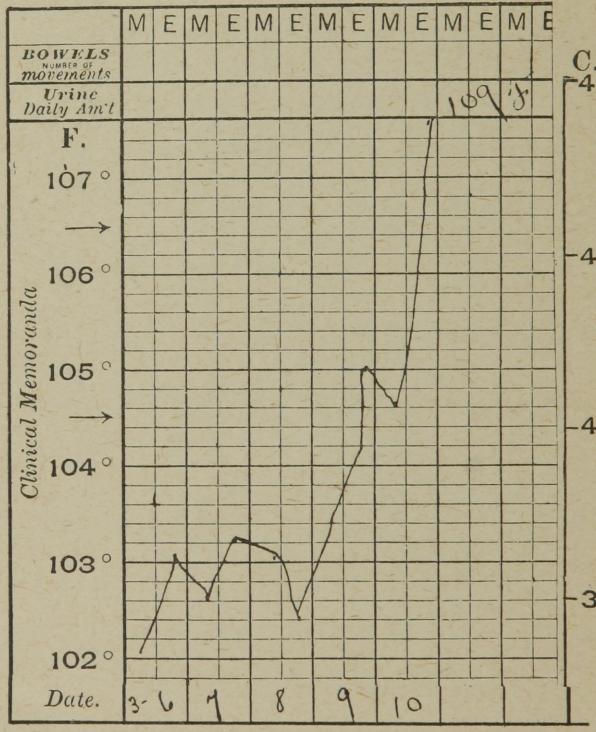


Case 12.—Cerebrospinal meningitis. Death.

lar; May 26, 9 A.M., became cyanosed and could not be aroused from stupor for about twenty minutes, then seemed to sleep quietly, respiration regular, pulse 70; died suddenly at 3 P.M.; no post mortem.

Case 12.—Male, aged 40, admitted March 20, 1898; at five previous evening had severe pain in epigastrium; was able to work during the day; at 2 A.M. began to vomit; has vomited three or four times since; vomitus slightly tinged with blood; entered hospital intoxicated, but previous to last night had drunk no liquor for eight months; mind clear but apathetic;

well nourished; skin dry and hot; pupils react; tongue coated at sides; spleen not palpable; extremities negative; reflexes diminished. Temperature 101 degrees F.; pulse 120 on admission; March 23, epistaxis, pain in head and neck; March 25, for last twenty-four hours patient has complained of pain in left ankle and shoulder. Examination shows slight redness, swelling and considerable tenderness; March 30, symptoms

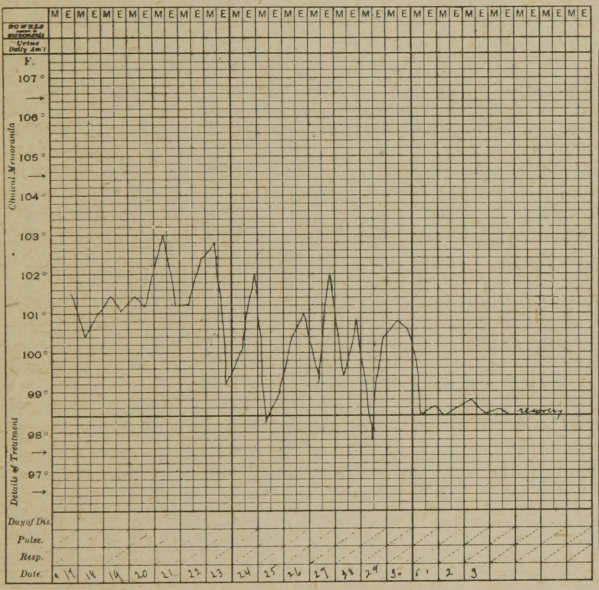


Case 13.—Cerebrospinal meningitis. Showing ante-mortem rise in temperature.

continue; April 2, ankle-joint symptoms almost subsided; April 5, patient semiconscious, pupils dilated, the right larger, react to light; moves hands aimlessly, fingers of right clutched; involuntary urination; face drawn to left; right hand apparently paralyzed; died 5 A.M., April 6.

Case 13.—Female, aged 25, admitted March 4, 1898; three days ago was taken with pain in left wrist, headache, vomiting

and fever; was very nervous and had one or two convulsions; not unconscious during the attacks; complained of pain in precordial region; wrist was very sore, red and swollen; had been sore before but began to be worse at this time; could not sleep at night; four years ago had an attack of rheumatism which lasted three weeks. She says she does not know of having had any heart trouble at that time; face flushed; expression dull; tongue moist, not much coated; eyes react to light; pulse 118, regular; temperature 102.5 degrees F.; loud systolic murmur heard plainest at apex; no friction sound; spleen not palpable; no rose spots, no tenderness; urine albuminous;



Epidemic cerebrospinal meningitis.

very slight edema of legs; March 5, had been delirious during night; March 14, severe headache; March 16, delirious during night; March 18, patient apparently in stupor; has convergent strabismus, pupils small and sluggish; patellar reflex on right marked, absent on left; sensation and motion good in both arms and legs; strabismus not constant, can move eyes through whole of arc; no facial paralysis; March 21, involuntary evacuations of urine and feces; March 22, profuse perspiration over whole body; has pain in stomach; vomited colorless liquid; March 24, more conscious; complains of constant

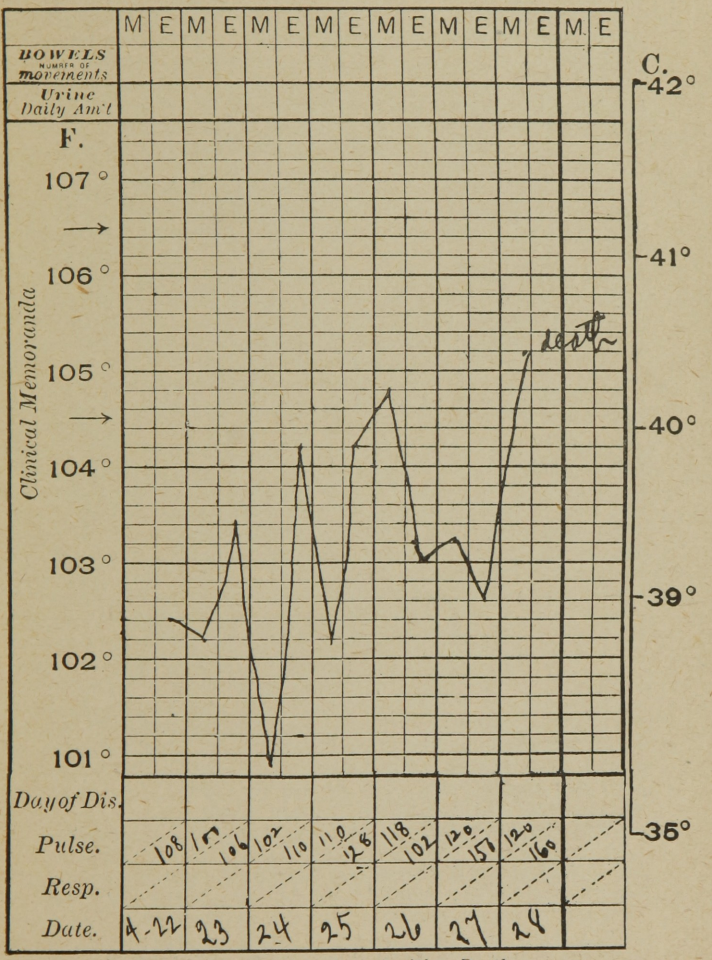
headache; March 26, appears somewhat improved; March 31, patient complains of pain in back and lower part of body; April 1, tremor in right hand and arms; conjunctiva injected; no pain in head or neck though rigid; April 7, not much change; sweats profusely; April 9, has difficulty in swallowing; April 10, gradually growing weaker during night; died at 8:40 P.M.

Case 14.—Female, aged 28, admitted May 10, 1898; patient delirious; no history obtained; general nutrition good; pupils react; tongue has a brownish coating; abdomen somewhat prominent and tender, spleen not palpable; temperature 103 degrees F.; pulse 108; May 12, delirious, very restless; May 13, muscles twitch from head to foot; tries to get out of bed; involuntary evacuations; May 15, quieter; May 16, back of neck very painful; May 18, difficulty in swallowing; May 20, lay in stupor all night; died at 11:30 A.M. Temperature at no time very high, there being little difference between morning and evening temperature. Shortly before death it became normal.

Case 15.—Male, aged 20, admitted April 22, 1898, was taken sick about a week ago with prodromal symptoms of typhoid; face flushed, tongue broad and dry; abdomen rather distended, tenderness in iliac and splenic areas; no rose spots; skin dry; patient chilly; headache; tenderness over cervical vertebræ; vomited medicines; April 24, slept but very little, was restless, but did not vomit after 2 A.M. April 25, vomited greenish fluid, could not retain nourishment. April 26, face flushed, eyes dull, pupils dilated, tongue very dry and glazed, abdomen distended, tender; patient delirious. Delirium gradually gave way to coma, and patient died April 28, 12:10 P.M.

Case 16.—Male aged 40, admitted Jan. 9, 1898, lies on his back motionless, oblivious to surroundings; eyes open; nystagmus; face flushed; skin moist; pupils react; abdomen depressed; spleen not palpable; reflexes somewhat exagerrated; January 10, seems to understand some things; involuntary evacuations; January 12, conjunctiva congested; died at 9:30 P.M. Post mortem examination of brain shows dura mater adherent to pia, especally along superior longitudinal fissure; meningeal vessels congested.

Case 17.—Male, aged 24, admitted April 2, 1898, had been on a spree, and four days ago, when he awoke after the spree, felt very sick, complaining of a severe headache, vomiting and pain in the bones; says that he is very nervous and can not sleep; is often nauseated; nutrition excellent; pupils react; spleen not palpable; abdomen slightly tympanitic; temperature 103.2 F.; pulse 108; April 4, very delirious, April 5, patient complains of very severe headache and pains in back of neck; during afternoon became semicomatose; is rigid; tenderness and hyperesthesia along spine; marked tremor in hands and same in muscles of face; herpes on lips. April 6, head drawn toward



Case 15.—Cerebrospinal meningitis. Death.

the left; conjunctiva congested; breathing rapid; subsultus tendinum; swallowing difficult; died April 8. Post-mortem: Veins of dura distended; vessels of pia injected; over convexity of hemisphere, especially along course of vessels, is a tenacious yellowish exudate, apparently purulent; all the vessels of the membrane are markedly distended: great deal of turbid fluid escapes when the dura is cut; lateral ventricles not distended; one or two small areas of exudate around vessels at base; about upper extremity of spinal cord is seen seropurulent fluid; spinal canal not opened.

Case 18.—Male, aged 33, admitted April 6, 1898; present illness began Tuesday morning with headache and backache; no vomiting; bowels constipated since Tuesday; herpes since Wednesday; patient walked to the hospital; has been drinking some; stupid, slightly restless; eyes normal, except dull; pupil normal; lips covered with vesicles; heart shows no abnormality; abdomen not distended; area of splenic dulness slightly increased; reflexes slightly diminished; April 4, in evening, patient almost comatose; April 5, patient has difficulty in swallowing; rigidity of neck. April 6, patient comatose, died same day. Post-mortem: Convexity and base of entire brain covered with fibrinopurulent exudate; substance soft; cerebrospinal fluid poured from spinal canal is slightly turbid; cocci and diplococci found in exudate, not enclosed in cells.

Case 19.—Male, aged 19, admitted April 28, 1898; illness began about a week previous, with headache and vomiting; somewhat emaciated; very irritable; mind lethargic; is, however, readily aroused; cries aloud; temperature 99.2 F.; pulse 120 and very weak; pupils react, right more readily than left; nearly deaf in left ear; tongue seems swollen; neck retracted; spinal muscles rigid; shoulders permanently shrugged; marked *taché cerebrale;* abdomen retracted; patellar reflexes not demonstrable; April 29, patient delirious; April 30, slight nystagmus and paresis of left side of face; eyes do not move together; apparent paresis of right external rectus; died April 30, 10:30 P.M. Post mortem: On pia, especially about commissures, is a thick purulent exudate; dura negative; overconvexity of lobes; along vessels is some purulent exudate; pia everywhere injected; vessels involved in a mass of exudate; lateral ventricles are unchanged; brain substance is soft.

Case 20.—Male, aged 4, admitted April 18, 1898; present illness began suddenly about noon, April 16, with vomiting and headache; vomiting continued almost incessantly all through the day and night; about 2 P.M., April 17, became unconscious and had twitching movements of the extremities about every twenty minutes; when conscious could not see; eyes, mouth and throat negative; head drawn back to slight extent, and slight rigidity of neck muscles; also tenderness along nape; reflexes normal; semicomatose; died May 19, 1898; no post-mortem made.

Case 21.—Male aged 5, admitted April 25, 1898; was taken suddenly ill two days ago; had repeated convulsions on day that he was admitted; temperature normal; pulseless at wrist; respirations 36; rigidity of neck muscles and tenderness of neck and spinal column; eyes negative; reflexes present; very restless, talking and crying incessantly, convulsions about every thirty minutes, twitching of extremities; died at 2 P.M., April 26, 1898. Post mortem showed a small amount of pus under pia mater along course of larger vessels.

Case 22.—Male, aged 24, admitted May 24, 1898, in a comatose condition, head retracted, *taché cerebrale*, pupils unequal; previous history, as far as it could be obtained, that of cerebrospinal meningitis of the epidemic form; died about one-half hour after admission.

Case 23.—Male aged 3 years, 7 months; seen March, 1898; taken sick with earache and fever; throat red; examination of secretion showed presence of diplococci; about four days later showed symptoms of meningitis, vomiting, headache, retraction of head, unequal pupils; about same time developed a croupous pneumonia accompanied by a serous exudate in pleural cavity, which showed pneumococci; purulent discharge from ear; death on nineteenth day of disease.

Case 24.—Female, aged 1 year, 6 months; seen April, 1898; taken sick suddenly with vomiting, retraction of head; *taché cerebrale;* tenderness along spine; abdomen boat-shaped; high irregular temperature, delirium, coma and death.

Case 25.—Female, aged 5, sister of above; taken sick about same time, with symptoms of typhoid fever which were soon followed by the characteristic symptoms of cerebrospinal fever; during its course pus was discharged from ear, containing diplococci; died within an hour of her sister.

Case 26.—Female, aged six months, admitted April 3, 1898; badly developed; rickety; taken sick suddenly, April 2, with convulsions, followed by vomiting; retraction of the head; tenderness along spinal column; *taché cerebrale*; pupils contracted; gradually became comatose, convulsions brought on by the slightest touch; death April 13, 1898.

Case 27.—Male, aged 17, admitted June 7, 1898, had been sleeping in a room in which the sunlight never penetrated; was taken sick suddenly with nausea, vomiting, chill, severe headache, rise in temperature, pains in back and limbs, delirium; head retracted, tenderness along spine, herpes on lips; no paralysis; made a gradual recovery but his mental faculties have been disturbed, as he is dull and forgetful.

Case 28.—Female, aged 1 year, 6 months, admitted July 5, 1898; had an attack of pneumonia previous to admission to hospital, followed by cerebrospinal meningitis, the acute symptoms of which had abated at time of admission; extremely emaciated; temperature 95.4 F.; pulse 120; rigidity of spinal muscles; July 6 temperature rose to 100.8 F. early in morning;

pulse weak and rapid; another slight rise in temperature on 7th; from this date to death, July 13, temperature not above normal; death apparently due to exhaustion and inanition.

Case 29.—Male, aged 3, admitted July 1, 1898; brother of Case 28. After the acute symptoms in his sister's case had subsided the patient was taken sick with typic symptoms of cerebrospinal fever; sudden onset, vomiting, headache, herpes, retraction of head, tenderness of spine, *taché cerebrale*; temperature 102 F.; pulse 96; gradually grew worse and died July 6; no paralytic symptoms until near death; no post-mortem.

Case 30.—Male, aged 38, admitted July 6, 1898. Two days before admission he was taken sick with headache and vomiting, no chill; headache became very severe and soon patient passed into a semicomatose state; unconscious on admission, pupils somewhat contracted, reflexes increased, tenderness over greater part of body; retraction of head; involuntary evacuations; no rash; pulse 62, temperature 99.2 F. on admission; died suddenly July 7; no post mortem.

Case 31.—Male, aged 38, admitted June 30, 1898; present illness began June 28, following a spree; prodromal symptoms resembling those of delirium tremens, followed by a chill; dull pain in face and forehead, vomiting, gradual loss of consciousness, with trembling and twitching of limbs; on admission, unconscious, temperature 103 F., pulse 90; touching body or head seemed to cause pain; tongue coated; pupils contracted, very restless; spleen not enlarged; no rash; June 3, very restless, temperature 103.2 F.; June 12, very delirious, lumbar puncture made, fluid removed showed presence of meningococci; died July 16; respirations 54 and pulse 152 shortly before death.

Case 32.—Female, aged 17, admitted June 28; taken sick about two weeks previous with a chill, vomiting; severe headache, backache, herpes; brought to hospital delirious; retraction of head and neck; delirium continued for two weeks; temperature very irregular; Aug. 5, 1898, patient convalescent, but very weak; no lumbar puncture. This case ran a rather insidious course, patient at times being apparently much improved when graver symptoms would again become noticeable.

Case 33.—Female, aged 18, admitted June 29, 1898; illness began a week previous with headache, vomiting, diarrhea and general weakness; brought to hospital delirious; head markedly retracted; tenderness along spine; herpes; boat shaped abdomen; slight strabismus of left eye, conjunctivitis; temperature 101 F.; pulse 114; reflexes slightly exaggerated; delirium lasted about three weeks; after a four-weeks course the symptoms gradually abated, leaving patient in a greatly emaciated condition; patient discharged recovered August 5.

Case 34.—Female, aged 46, admitted July 5, 1898; sick for some time before admission, symptoms resembling typhoid fever, with very severe headache; comatose; marked retraction of the head, neck rigid and tender; no eye symptoms; no

eruption; contractures of muscles; temperature 101 F.; pulse 100; July 8, great difficulty in swallowing; died July 9.

Case 35.—Female, aged 31, admitted May 14, 1898; taken ill suddenly with severe headache, nausea and vomiting; on admission is suffering from severe headache and pains in chest, moans continuously, vomited, very restless and nervous; spleen enlarged; Widal's test shows partial reaction; hyperesthesia over greater part of body, greatest over spine; marked rigidity of neck; tongue coated and scaly; May 17, patient very restless, severe pain in head and neck; May 26, severe vomiting spell; May 27, patient delirious, moans continuously; May 30, better, not delirious; May 31, again delirious; June 4, great difficulty in swallowing, pupils dilate and contract from time to time; June 5, pulse very weak, reflexes normal; June 11, trembles constantly; June 13, great difficulty in swallowing, which persisted up to time of death; June 30, right pupil dilated, left contracted; died July 1; ante mortem rise in temperature to 109 F.

Case 36.—Female, aged eight months, admitted June 6, 1898; has been sick about two months; feverish and cried a good deal at first; semicomatose, at times rigid; head retracted, nystagmus; vomits frequently, abdomen tender: not improving, opisthotonos so that child could be lifted by the heels and head; June 15, same condition: June 16, had several convulsions last night, restless; June 24, condition about the same, no more convulsions; June 27, looks somewhat better, retention of urine since 4 P.M. yesterday; July 5, slight convulsions; July 8, marked contracture of muscles; boat-shaped abdomen; died July 12. In this case temporary strabismus had been noticed at times.

Case 37.—Female, aged 4, seen June 20, 1898; ill three weeks; taken sick suddenly with headache, vomiting, stiffness of neck and fever; semi-comatose, pupils dilated, muscles of neck rigid, spine tender, opisthotonos, great emaciation, no eruption; abdomen and chest negative; joints not swollen; temperature 101 F.; swallowing difficult; remained in very much the same condition, with the exception of occasional convulsions, until one week before death, when paralysis of the muscles of the eyeball set in; died July 10, 1898.

Case 38.—Female, age seven months, sister to Case 37, was taken sick with similar symptoms about three days after onset of the disease in her sister; ran a more chronic course, with only a slight rise in temperature; had frequent vomiting spells, scaphoid abdomen and marked retraction of head; circumference of the head considerably increased as shown by measurements; marked bulging of the fontanelles; died August 3.

SYMPTOMATOLOGY.

Although this subject has been exhaustively treated in descriptions of epidemics occurring elsewhere,

yet as almost every epidemic offers some distinctive features, a closer consideration of the symptoms met with in these cases may be useful.

Pain.—This is predominant and, as a rule, the earliest symptom of epidemic cerebrospinal meningitis. It varies in severity from a moderately severe headache to an agonizing pain radiating apparently through the entire body. Headache was found present in all of the cases where a complete history could be obtained. As a rule it affected the entire head, being most severe in the occipital region. In one case the patient, at the onset of the disease, complained chiefly of severe pain in the limbs, while the head was not much affected, and in four cases severe backache was the symptom causing the greatest distress to the patient. In three instances abdominal pain was noted and in two there was hyperesthesia of the greater part of the body, so intense in one case that the slightest touch would throw the patient into convulsions. In one case the pain was chiefly referred to the chest, without there being any objective signs of a diseased condition in the thoracic viscera; another patient complained of boring pain in the bones, resembling that of osteomyelitis. Tenderness in the iliac and splenic areas was noticed in one case. The spine was generally sensitive, although in a varying degree, and in some cases the slightest touch would elicit pain, while in others deep pressure was necessary. Attempted flexion of the head usually caused pain, which in some cases was of an excruciating nature, causing the patient to cry out aloud. In some of the cases the headache and tenderness of the spine persisted after the patient had made an apparent recovery.

Chills.—As an initial symptom, chills occurred in seven out of the thirty-eight cases in the series. In two cases the patients had complained of a chilly sensation several days before the onset of the acute symptoms. In one case a chill lasting thirty minutes occurred on the third day of the disease. The majority of the cases beginning with a chill ended fatally.

Gastro-intestinal symptoms.—Vomiting is one of the most constant initial symptoms of cerebrospinal meningitis, having occurred in twenty-six cases at the onset, while in four it occurred during the first days of the disease. In the cases occurring in adults, true projectile vomiting was seldom met with at the onset, although it was later on noticed in several. In some, vomiting was almost continuous, and proved to be a very annoying symptom, medication being of little avail. It is generally regarded as being due to irritation of the vomiting center in the medulla. As a rule it was independent of the taking of food. The tongue was coated in the majority of cases, but showed nothing pathognomonic; in some of the cases it was coated thickly in the center, with clean edges; in others the edges were thickly coated and swollen, while the center was clean; of greater diagnostic value was the trembling of the tongue often noted on its being protruded. The condition of the bowels also varied greatly. In six cases diarrhea occurred, in two instances being of a rather intractable nature. Constipation was more frequent, occurring in nine of the cases.

There is one symptom to which the writer wishes to call especial attention, as it has to his knowledge not been dwelt upon to any extent in previous reports. This is difficulty in swallowing. It was noticed in six cases, each of which terminated fatally; it must therefore be considered as a grave omen in the prognosis. As a rule it was found late in the disease, when the patient had become semicomatose, but in one instance it occurred several weeks before death, while the patient was still perfectly conscious. Its causation is not clear, but it seems plausible that it is due to an accumulation of fluid in the fourth ventricle exerting pressure on the nerve-centers which control deglutition in its immediate vicinity. As lumbar puncture was not made in any of the cases where this symptom was present, the effect of the withdrawing of the fluid could not be ascertained.

Skin.—Herpes labialis was the most prominent skin lesion and was present in twelve cases, which is about the same percentage as found in the Boston statistics of Councilman, Mallory and Wright. One case showed a papular eruption; another a reddish-brown macular eruption. Petechial patches were noted once and *taché cerebralis* was found in a number of the cases.

Retraction of the head.—In thirty cases there was retraction of the head, varying in degree; in some so slight as only to be noticeable on close examination, or on attempting flexion, while in others marked opisthotonos was present. In one case the rigidity was so great that the child could be lifted by the head and heels, without any noticeable change in the degree of the opisthotonos.

Eyes.—The eyes were more or less affected in a large proportion of cases and form an item of interest in connection with diagnosis. The ocular manifestations varied from a simple congestion of the conjunctiva, which occurred in six cases, to trophoneurotic ulceration of the cornea and destruction of the eye in one. In two there was purulent conjunctivitis and one showed, on ophthalmoscopic examination, a cupped disc. The pupilary symptoms were not constant; in five cases the pupils were dilated, and in two of these the dilation was more marked in one eye than in the other; they were contracted in four, and dilated in one eye and contracted in the other in one case. In one they were seen to dilate and contract from time to time, without apparent cause. Nystagmus was observed five times, and paralysis of the muscles of the eyeball once, the eye remaining fixed. Strabismus occurred in four cases, in one instance being transitory, and paresis of the right external rectus was noted once. In one case there was absolute blindness extending over a considerable period of time, without any apparent lesion to account for it. Puffiness of the eyelids occurred twice, and diplopia once. Photophobia, which is generally described as one of the

symptoms, was observed comparatively few times. Lesions of the fundus also did not form a prominent feature, although this may be partly due to the fact that an ophthalmoscopic examination was frequently omitted.

Sensorium.—In twenty cases the patients became delirious in the course of the disease, and the delirium in some cases, especially where the disease occurred in robust young men, was of a violent type. In some instances hallucinations were present resembling those of delirium tremens. The delirium in some of the cases appeared early, the patient being markedly delirious on the second or third day of the disease; in others it did not appear until the second or third week. The delirium in these cases did not seem to have any connection with the height of the temperature. In five of the cases the patients, though conscious, seemed to be perfectly apathetic in regard to their surroundings and conditions, while in others, again there was noted great irritability of temper, the slightest cause producing anger. Coma was observed five times and was as a rule absolute, it being impossible to arouse the patients, although in one or two cases the patient suddenly regained consciousness, but relapsed into his former state after a brief interval. In two cases the mental faculties were seriously impaired after recovery from the acute symptoms had taken place.

Motor disturbances, reflexes, etc.—The motor disturbances of the eye have already been considered under another heading, so we now take up those of the body in general. Paralysis of all the voluntary muscles of the body was observed in two cases shortly before death; paralysis of the facial muscles on the left side was also twice noted, and paralysis of the right hand once In two cases contractures of the muscles occurred, in three there was muscular twitching, chiefly in the extremities, and tremor of the hands was observed in two instances. Involuntary evacuations of urine and feces during the course of the dis-

ease were noted six times. This is not necessarily a fatal omen, as several of these cases recovered. Subsultus tendinum was found twice, and, as in typhoid, is a grave sign. Convulsions were rare in adults, but frequent at the onset in children. In several children convulsions occurred from time to time during the course of the disease; in one the slightest touch would bring on an attack. The state of the reflexes was not constant, but as a rule they were not perceptibly altered. In five instances the knee-jerk was exaggerated, and diminished in three, while in one it was entirely absent, and in another was absent on one side and increased on the other. The cremasteric reflex was absent in one case and increased tricipital reflex was noted in one. The causation of this variation in the reflex action has never been satisfactorily explained.

Abdomen.—In twelve cases there was retraction of the abdominal walls producing the condition described as "boat-shaped" abdomen. Tympanites was present in three cases, and in one of these the abdomen was markedly tender. Splenic enlargement was observed in four cases, but in none was the enlargement as great as it usually is in typhoid fever. The liver was found enlarged in two instances. Gurgling in the iliac fossæ was elicited in two examined for this, rather unreliable symptom.

Joints.– Joint affections were encountered five times, varying in severity from simple tenderness and pain on motion to a condition closely resembling acute articular rheumatism; in no instance, however, were these symptoms of long duration, and suppuration was not noticed. The joints chiefly affected were the wrists, and next the ankle-joints.

Blood.—A careful blood analysis was made only in a limited number of cases; in all instances leucocytosis was found to exist. In one pigmented leucocytes were found, together with a few extracorpuscular bodies containing pigment. Widal's test was generally negative, although in one or two instances a partial reaction was noted.

Urine.—In seven of the cases the urine was albuminous; in one sugar as well was present. An excess of earthy phosphates was noted once. In none of the cases where albumin was discovered were casts found. Cylindroids were found in one case; the urine being otherwise normal.

Thorax.—True lobar pneumonia as a coincident lesion was noted in two instances, in both, however, the symptoms of meningitis predominated. Other symptoms were found in five cases, chiefly diminished resonance, which could not be properly explained. This condition was found three times. A blowing systolic murmur at the apex-beat occurred twice, and Cheyne-Stokes respiration once during the course of the disease, and in several instances shortly before dissolution took place.

Pulse and temperature.—In regard to the pulse and temperature curves, which have been charted, the same variations and irregularities as those described by Wunderlich were found. As a rule the initial symptoms were accompanied by a rise in temperature which rarely exceeded 102 during the first forty-eight hours. In some of the cases this initial rise was followed by a period of three or four days, during which the temperature would remain normal, after which there would be another rise. The height of the initial fever afforded no criterion whatever as to the severity of the disease, as in a number of the cases which began with violent symptoms and a considerable increase in temperature the patient made a rapid recovery, while in others which began insidiously and in which the rise was very slight at the onset, the symptoms gradually increased in severity, the disease ending fatally. Often after a continued rise extending over two or three weeks there would be a sudden drop to normal, the temperature remaining low from three days to a week, followed by another rise which in some instances exceeded the primary one. One curious feature was that as a rule the drop was not attended by a corresponding amelioration of the other

symptoms; while in other cases there would be considerable improvement for a certain period, notwithstanding the temperature remained high. The time of day seemed to exert little influence on the temperature; some cases showed a distinct morning remission, but this was not constant, as it frequently occurred that on the day following the one on which there had been a morning remission, the morning temperature would exceed that of the previous evening; the next day would perhaps again reverse the order. In some cases the disease ran almost an afebrile course; in fact, it may almost be said of this disease that the only regular feature of its temperature curve is its irregularity. An ante-mortem rise in temperature was noted in three cases; in one of these the axillary temperature was 109 F. There was no relation between the temperature and the pulse. It may be given as a rule that the characteristic feature of the pulse in epidemic cerebrospinal meningitis is its slowness, although this rule is not constant, as there are cases met with in which the heart's action is accelerated considerably beyond normal. In one instance in our series, where the patient had a temperature of 105 F., the pulse at the same time was only 80. Within a few hours previous to death there was, as a rule, a great increase in the frequency of the pulse, even in those cases where it had been slow during the course of the disease. In volume the pulse is usually full; a dicrotic pulse was seldom met with.

Special symptoms.—Among special symptoms, ear troubles, on which special stress has been laid by previous observers, were only noticed in a small number of cases. Some deafness was met with comparatively often, and in one or two instances suppurative middle ear disease, but aside from these there was an almost complete absence of aural complications. Lesions of the nasal and pharyngeal mucous membrane were also unfrequent in their severer forms, although redness or a slight catarrhal condition occurred in a number of the cases. Epistaxis was found once. Bulg-

ing of the fontanelles occurred as a rule in younger children, in one case the intracerebral accumulation of fluid causing a considerable increase in the circumference of the skull. In protracted cases convalescence usually found the patients greatly emaciated and exhausted, from which condition they recovered very slowly, some of the young children, in fact, succumbing from inanition after the acute symptoms had subsided.

General course of the disease.—As to the general course of the disease, the average duration of the Chicago cases with recovery was four weeks. Some ran a much shorter course, recovery taking place within two weeks, while in others it was delayed from six to eight weeks. Relapses were noted in several instances. It can not be said, however, that this is a disease prone to relapses. In the fatal cases death usually took place within two weeks. Cases such as have been described under the name of "meningitis siderans," in which death takes place within a few hours or days, were very rare during the epidemic. Of the thirty-eight cases described, twenty-five ended fatally, a mortality of 65 per cent.; this figure is far from correct when applied to the entire number of cases in the city, as cerebrospinal meningitis is not properly a hospital disease, comparatively few cases being sent to the hospitals, and these only where the disease was severe. This explains the apparently excessive mortality, as our cases were almost exclusively taken from the hospital records. The writer has the testimony of a number of physicians to the effect that mild cases not requiring hospital care were very frequent, and that if these were averaged up with the more severe cases, the mortality would probably not exceed 20 per cent.

Sanitary Aspect.—In considering those features of the disease which are of interest to the sanitarian, it becomes necessary to take up each feature separately, and afterward arrive at our conclusions from a study of the salient points of the whole. There are

few diseases excepting leprosy about which so many different opinions have been expressed as to the mode of propagation as in epidemic cerebrospinal meningitis. As it is a disease which, the world over, is rather on the increase than on the decrease, and is, therefore, a constant menace to an unprotected populace, the study of the factors which enable it to spread and the means by which it can be prevented becomes one of considerable interest to the sanitarian.

Propagation by contagion.—A question such as this can only be decided by a careful weighing of the testimony. Those factors which speak against the disease being spread by direct contact, are: 1. Scarcely any of the epidemics have shown continuous extension, the disease often appearing simultaneously in widely separated communities, while the intervening country remained immune. In this respect it thus differs from most diseases classed as contagious, as smallpox or yellow fever. 2. The source of infection, as a rule, can not be discovered—in this respect also differing from the diseases mentioned. 3. During a number of epidemics it has almost been the rule that inmates of the same home where the disease occurred escape contagion, only isolated cases occurring. In a large number of instances isolated cases occurred in various sections of a city, the disease not being confined to any particular locality. 5. Persons in no way coming in contact with those having the disease are afflicted. 6. The nurses and physicians in attendance on cases of epidemic cerebrospinal meningitis rarely take the disease.

In favor of its contagious nature the following ing facts are quoted: 1. The disease quite frequently attacks members of the same family, as is shown by Brooks,[78] Kohlman,[80] Trevelyan[82] and others. 2. Persons nursing cases of cerebrospinal meningitis have taken the disease, as is stated by Erdman[81] and others. 3. Epidemics in hospitals and jails, nearly every inmate taking the disease; the disease sometimes being confined to one ward. Instances of this kind

were reported by Lancereaux and Besancon[93]. 4. Cases occurring grouped in little foci, as in the epidemic at Lonacoming. 5. Evidence that in some instances the disease is carried from one person to another by a carrier who remains immune, as was shown by Peterson,[84] in a child 5 years of age; the brother-in-law, who was tax collector, and had to enter a house where the disease was prevailing, lived in the same rooms with the patient—the child mentioned taking the disease after a few days, while the brother-in law escaped. In this same epidemic, described by Peterson, the contagion could be traced in many instances. 6. In the French epidemics during the early '40s the disease extended with the movements of the troops.

In the Chicago epidemics there were a number of instances recorded in favor of the disease being contagious. In one instance there were three cases in one house, two being in the same family. These cases developed within about a week of each other; in four other instances which came to the writer's notice, two cases occurred in the same family. About one-half the cases mentioned in this report occurred within a territory about a square mile in extent, while the remainder were scattered over almost the entire city, the extreme south and west sides remaining immune.

It will be seen by studying the pros and cons mentioned above, that they are almost exactly contradictory, and therefore would have a tendency to neutralize each other, making the testimony worthless, either way; this is, however, not the case when the disease is considered in the light of recent research. The writer firmly believes that epidemic cerebrospinal meningitis *is* contagious, not like measles and scarlatina, but very closely resembling phthisis pulmonalis. Kiefer,[74] Ghon and others have almost conclusively proven that the specific germ exists in the buccal and nasal secretions; hence it stands to reason that where, through uncleanliness or neglect, these secretions are

scattered broadcast, it is quite easy for the organisms to enter the nasal and buccal cavities of persons exposed, and, the proper conditions being present, for the disease to develop. The nature of the bacterium explains why the disease is apparently, in some instances, contagious, while it is not so in others. For instance, as is shown in the section on bacteriology, the germ will only grow for a limited length of time; this explains why persons, moving into rooms previously occupied by a person having the disease, do not contract cerebrospinal meningitis. The reason why the disease does not spread in the wards of hospitals is because the infective material is not scattered around. The disease is more prevalent during or following wet weather, because the germ retains its life longer in a moist medium than in a dry one. In fact, by a careful consideration of the mode of growth of this germ a majority of the apparent inconsistencies in the history of the spread of this disease will become clear.

The question whether cases should be isolated is of interest, both to the sanitarian and to the practitioner. By the medical officers of the great military establishments of Germany, Great Britain and France, cases are strictly isolated as a precautionary measure. This is done on account of the fact that epidemics of the disease in barracks have been by no means of rare occurrence. During the recent limited outbreak of cerebrospinal meningitis among the troops at Chickamauga Park, the cases were also isolated, with the apparent result of a stoppage of the spread of the disease. As a rule, in large city hospitals the cases were admitted into the general wards without any other inmates taking the disease. Cerebrospinal meningitis at the present time is not considered contagious by the health boards of our cities, and physicians are not required to report the cases occurring in their practice. The same state of affairs had existed until a short time ago in regard to phthisis pulmonalis, but recently some of the health authorities have, in

view of the constant increase of pulmonary tuberculosis, taken measures that cases of this disease be investigated and controlled in a manner similar to that by which cases of the more generally acknowledged contagious disease have been controlled. Although epidemic cerebrospinal meningitis does not as a rule spread by direct contact, still it has been proven to do so in exceptional cases when the conditions for its propagation are favorable. These conditions are uncleanliness and a general unsanitary condition of the environment of the patient. It therefore seems probable that, although all harsh measures are to be deprecated, if a certain amount of control could be exerted by the public health authorities, over these cases, the spread of the disease could be limited to some extent. Isolation should be insisted upon whenever practicable and sanitary defects remedied. During an epidemic it might be advisable to caution those exposed to use a mild antiseptic nose-and-throat spray or wash, in accordance with the more generally accepted view that the specific germ gains entrance through these passages.

Influence of sanitary defects.—This disease is no exception to the rule that the spread of epidemic diseases is increased by unsanitary conditions. It has been stated that the disease occurred among all classes and conditions of people, but many instances which prove the relation between cerebrospinal meningitis and unsanitary conditions could be cited, and a few examples may not be out of place:

In his description of an epidemic in Norwich, N. Y., Brooks[78] states that the following conditions existed: Norwich lies in a valley about a mile wide, between high hills and practically surrounded by them; at the west side of the valley, at the foot of the hills, is a large creek, and at the foot of the eastern hills is a river of medium size. The creek referred to comes through a narrow valley and starts in the town of Plymouth where cases of cerebrospinal meningitis occurred coincident with the epidemic in

Norwich. This creek empties into the Chenango River at the lower end of Norwich village. Eight miles below Norwich, on the same river and in the same valley, is Oxford, where twelve cases occurred. Eleven miles above Norwich, on the same river and in the same valley, is located Sherbourne, the location of a severe epidemic of cerebrospinal fever in 1860. North Norwich lies six miles above Norwich, in the same valley. Eighteen cases occurred here, where cases also occurred in 1860. The creek mentioned passes through the center of the town and makes the greater part of it into an island, it being low and swampy. The soil of the valley referred to is composed of a layer of loam, beneath which is a layer of sand and gravel, the whole being underlaid by a thick layer of blue clay. This clay is impenetrable and retains everything above it There was no system of sewerage, drains and closets being made in the soil. A system of water-supply exists, the reservoir being three miles from the village, and for three years the extra material has been washed into the soil. The general direction of the water current is from the northwest toward the southeast. At the northwest end of the village was a slaughter-house and another at the northern part, both kept in a filthy condition and surrounded by large hog-yards. The northeast part of the village is most thickly settled and contains numerous stables, shops, a tannery, and an abandoned basin nearly a quarter of a mile in length, that has been used for cesspool purposes for several years without cleaning. The northwest and the southeastern portions were next in order of unsanitary conditions. The severe winter caused the ground to be frozen to an unusual depth which, together with the natural construction of the soil, caused an accumulation in the cesspools and vaults instead of draining off.

In the northeast section, with a population of 1599 inhabitants, there were 29 cases; in 17 of the houses where cases occurred the sanitary condition was very

bad, in 5 fair, but not good, and in the remaining 7 there was no fault. In the northwest section, with a population of 1395, there were 18 cases, 11 of the houses were in a bad condition, 3 fair and 4 good; in the southeastern section, with a population of 1204, there were 13 cases, 9 of the houses were in a bad condition, 2 fair and 2 good, and in the southwestern section, with 780 inhabitants, there were 4 cases, 3 of the houses being unsanitary, while one was in a fair condition.

Brooks arrives at the conclusion that while unsanitary conditions do not produce the disease, they have much influence in localizing it when the proper causative condition exists. Very characteristic is also the description of the unsanitary condition of Lonaconing, by Flexner and Barker.[58] In this town a severe epidemic raged in 1893. Lonaconing is a mining town of 5000 inhabitants, located in a valley on the sides of steep hills, at whose bottom runs a muddy creek. Privies are above the houses and flat upon the ground. All refuse, etc., is washed down the mountain side by the houses and through the yards, after a rain or thaw, and down into the creek, which acts as a sewer. The drinking water is derived from surface wells and cisterns. In the valley the wells are situated but a few feet from the creek. The slaughter-house is in the heart of the town. Carcasses of horses are frequently floating in the stream or lying on the banks. There was great overcrowding, eight to eleven persons living in a house of four small rooms. In one house where a fatal case occurred, eleven persons slept in three bed-rooms. The houses often stand very close together, not leaving room for a footpath between them, and were themselves usually in a filthy condition.

During the Chicago epidemic fifty-one houses where cases of meningitis had occurred were inspected by the writer. In twenty-one of the houses or their surroundings unsanitary conditions were found which, in some instances, were very bad. In one house

where a fatal case occurred, nine families were living in a ten-room house, which was littered with filth of every description. Defective plumbing was discovered in a number of instances, and remedied through the health department. In eighteen of the houses the sanitary condition was fair, while twelve were in a good condition. The majority of the cases occurred in the poorer quarters, inhabited chiefly by foreigners.

From a consideration of our present knowledge of epidemic cerebrospinal meningitis, the writer arrives at the following conclusions:

1. That epidemic cerebrospinal meningitis is to be classed among the contagious diseases, belonging in this respect to the same category as phthisis pulmonalis.

2. That unsanitary conditions exert great influence in affording a proper nidus for the growth of the germ of this disease.

3. That the health authorities should receive notice of the occurrence of a case of this disease in order that sanitary defects may be remedied.

4. Persons afflicted with this disease should, whenever possible, be isolated, and all evacuations rendered sterile by the use of antiseptics.

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