spleen, high-colored urine, a frequent, full, and often bounding pulse, pains in the back and limbs, restlessness, and occasionally delirium. These symptoms abruptly terminate by an exceedingly copious perspiration between the fifth and the eighth day; and after a complete apyrectic interval (during which the patient may be so well as to get up and walk about), an abrupt relapse supervenes on the *fourteenth* day from the first commencement. The relapse runs a similar course to that of the primary paroxysm, and terminates between the third and the eighth day. In some cases a second, third, fourth, and even fifth relapse may occur. Death is apt to happen from sudden syncope, especially after the excessive perspiration; or from suppression of urine and coma. No constant eruption and no specific lesion are associated with the fever."

The temperature generally rises to 104° or 105° F. on the second day, and reaches its maximum the day before the defervescence. Then, as the other symptoms subside, it sinks rapidly, to about 98°. When the relapse occurs, on or near the 14th day, the heat again rises to 104°, 105° or more; descending afterwards with convalescence.

History.—Drs. Christison and Welsh first described relapsing fever in Scotland, 1817–18; Barker and Cheyne in Ireland about the same time. Rutty appears to have given an account of it in Dublin as early as 1739. In 1844 Dr. M. Clymer recognized it in some Irish emigrants coming to the Philadelphia Hospital.[1] Dr. Dubois reported the occurrence of a few cases in New York, mostly among emigrants, in 1847–8. In the same years it was epidemic in Glasgow, Edinburgh, London, and some parts of Germany; in 1855, in the Crimea, during the war; and since that time in Russia and elsewhere, on the European continent. Since 1856 it appears to have almost disappeared from Great Britain. During the summer of 1870, in New York and Philadelphia, hundreds of cases occurred in local "nests" of unsanitary conditions. In the Philadelphia Hospital, from April to November, 1870, 517 cases were admitted, with 80 deaths. Of this mortality (15½ per cent.) the greater part was amongst negroes; of whom 26 per cent. affected with relapsing fever died; of white patients, only 5 per cent.[2] In Great Britain the deaths have been reported as about 1 in 40; in Russia, 10 or 11 per cent. of the cases.

Diagnosis.—The most distinctive features of this fever are, the "crisis," from the fifth to the seventh or eighth day, and the relapse, on the fourteenth, or at all events between the twelfth and the twentieth day. In its general symptoms it resembles, otherwise, remitting fever; but with less decided daily abatement and exacerbation of the febrile movement, and with greater severity of "rheumatic" pains in the joints and muscles. The convalescence is slow, and is not unfrequently attended by a severe ophthalmia.

[1] Fevers, etc., by Meredith Clymer, M.D., Philadelphia, 1846, p. 99.
[2] Philadelphia Med. Times, March, 1871. The total number of deaths in the city, reported to the Board of Health, was 162; of which 107 were of colored people.

Pathology and Causation.—It would seem that relapsing fever must be a specific disease. Associated in nearly all cases with circumstances of destitution in large cities, it has been called in Europe, "famine fever." Contagion is asserted of it by Prof. J. Simon, Dr. E. Harris, and others. In the Philadelphia Hospital, in 1870, it did not spread to other inmates.

Anatomically, it has been observed, in fatal cases, that the *spleen* is often enlarged to a greater degree than in any other fever. Dr. Hand, of Philadelphia, has found the red corpuscles granulated and crenated; with (Cormack, A. Thompson) increase of white corpuscles. Dr. Obermeier, of Berlin, asserted the observation of minute mobile filiform bodies in the blood in this disease.

Treatment.—After a mild cathartic at the beginning, and, if headache be severe, a few cups or leeches to the back of the neck, cooling diaphoretics may be given, as solution of citrate of potassium or acetate of ammonium. After the crisis, quinine may be used, in moderate doses, at least until the relapse. It has been proved that this (the relapse) will not be *prevented* by any amount of quinine. Many patients will require support, especially in the third week, by beef-tea, etc.; some, alcoholic stimulation.

CEREBRO-SPINAL FEVER.

Synonyms.—*Cerebro-spinal Meningitis; Spotted Fever; Malignant Purpuric Fever.* The name adopted above is preferred by me, in the absence of sufficient preponderance of authority or reason in favor of either of the other names. The disease is a *fever*, or systemic disorder; not a mere local phlegmasia. It has no more claim to be called cerebro-spinal meningitis[1] than typhoid fever has to be called *enteritis*, or scarlet fever *faucio-pharyngitis*. Yet the term spotted (or petechial, Wood) fever is not fully justified as distinctive—because, only in a minority of cases it exhibits any eruption, and something like the same is also at times seen in typhus.

History.—Often obscurely described, this disease appears to have been known in France in 1310 and 1482; over Europe, or parts of it, in 1503, '10, '16, '17, '28, '45, '59, (Sicily) '64, 68, (Paris) '69–'74. In 1580, it was at Rome, Venice, and Madrid, with great mortality; again over Europe in 1582; at Trent, 1591, Florence, 1592; at various places, 1616 and 1624. Sydenham described it in 1661. In 1661 and '93 it was in Italy; and in England, 1698, 1710, 1741; in Prussia, 1704. Other years named for it are 1720, '60, '61; 1757 and 1788. A well-known outbreak of it occurred at Geneva, 1805; one in the Prussian army, 1806–7; in Sicily, 1808; at Dantzic, 1811; Brest and Mayence, 1813–14; Grenoble, 1814, and the same year at Paris; 1815 at Metz; elsewhere in 1816 and 1823. Afterwards in Europe its historians (under the names *méningite cérebro-spinal épidemique*, *cerebral typhus*, and *tifo apoplettico tetanico*) speak of it in 1832, '37, '39, '40, and almost every year till 1850, extending over many places in succession as far as Gibraltar and Algiers at the south, and Scotland and Ireland

[1] I regret being obliged to differ here from so high an authority as Professor A. Stillé. See his "Treatise on Cerebro-spinal Meningitis," Philadelphia, 1867.

at the north. From 1854 to 1861, in Sweden, Norway, and Holland. In North Germany and Russia, it is said to have prevailed in 1863–4–5 ; and in Ireland, 1866 and 1867.

In the United States, its first recorded visitation was in 1806, in Massachusetts. Then it gradually spread through the New England States, New York and Canada, from 1807 to 1812, when it had reached Philadelphia. After that it was met with at various places until 1820 ; but not with great frequency.[1] Between 1840 and 1850 it was epidemic in several of the Middle, Western, and Southern States (Kentucky, Indiana, Illinois, Michigan, Missouri, Tennessee) ; also in 1852 and 1858. Next we hear of it in 1862–3 ; most clearly in the descriptions of Dr. Gerhard[2] in the latter year, as it occurred in the neighborhood of Philadelphia. Since that time (at which cases were seen especially at Frankford, Falls of Schuylkill, Manayunk, and Norristown, but only a few in the city) it has been observed in a number of places in Pennsylvania, New York, Ohio, Indiana, Michigan, Missouri, Rhode Island, Vermont, Massachusetts, Maryland, and the District of Columbia.

Fig. 85.

Cerebro-spinal fever. (J. Lewis Smith.)

Symptoms and Course.—The attack is nearly always sudden. Chilliness, terrible pain in the head, extending to the back of the neck, nausea and vomiting, are the earliest symptoms. Delirium follows ; ending not unfrequently in coma. Tetanic spasm or rigidity of the muscles of the back of the neck (and sometimes of the back and limbs), is common. Convulsions are much less so, but do occur, particularly in the young. Painful sensitiveness (hyperæsthesia) of the whole surface of the body is present in most cases, when there is no coma. Loss of sight and

[1] Early American writers upon it were, North and Strong, 1811; Gallop, 1815; Miner, 1825.

[2] Transactions of the College of Physicians of Philadelphia, 1863.

hearing may take place during the middle period of the attack. The pulse is at first slow, then accelerated, but diminished in volume and strength. Respiration is slower than natural in most, but not in all cases. The tongue is usually at first white and moist ; sometimes natural ; in prolonged cases it may become yellow or brown. The bowels are costive or natural.

The skin has almost always at the beginning an abnormally low temperature. When reaction occurs it does not become very hot, as a rule. Burdon Sanderson has found it as high as 102° to 104° in children. Near the time of death, Wunderlich has known it to reach 107°, 108°, 110°. Dr. J. Lewis Smith has found the temperature morbidly elevated in the rectum, when it was not so in the axilla. Dryness of the surface is most common, although late in the attack profuse perspiration may occur. Often there is an herpetic eruption about the lips.

In a minority of the cases, though varying in proportion in different epidemics, *spots* (petechiæ) appear, on the second or third day, or later ; on the neck, breast, or limbs ; seldom on the face. They are of different dimensions, from the size of a pin's head to three-quarters of an inch in diameter, and distinct ; but not elevated nor disappearing on pressure. Their color is red, purple, or black. Sometimes they remain after death. They are either congested portions of the skin, or subcutaneous extravasations of blood.

The *duration* of fatal cases of this disease is generally short. Some die in three or four hours ; many within twelve or twenty-four. That period of time overpassed, the danger becomes less, but a fatal result may still occur, even after a number of days. The first four days are the most perilous to life. After recovery, sometimes blindness or deafness may remain ; sometimes, without these, a staggering gait is observed.[1]

Morbid Anatomy.—The blood, during life, is found to have an excessive proportionate amount of fibrin and corpuscles. After death, when it has taken place on the first or second day, no anatomical changes, even in the brain, have, in several instances, been found. Most generally, however, the brain and spinal cord show some alteration. It is the *pia mater* especially in which congestion, at least, is nearly always present. At the base of the brain most of all, is this, often with serous and plastic exudation, observed. The surface of the hemispheres may also be diseased ; and, next in frequency, the pia mater of the cervical portion of the cord. The ventricles of the brain have usually an excess of fluid in them ; serum, either clear or mingled with blood or pus. The substance of the brain is more or less injected or congested ; the spinal cord occasionally so. Softening of the brain is reported in protracted cases.

No other lesion or appearance is shown to be usual in this disease. A few observers record the presence of rather firm fibrinous clots in the heart ; a larger number, enlargement of the spleen.

Diagnosis.—From typhus fever, this is known by the sudden-

[1] S. W. Mitchell, Phila. Med. Times, Jan. 31, 1874.

ness of its onset, the early period of danger, and, in favorable cases, the rapid recovery; as well as by the peculiarity of the eruption. From ordinary inflammation of the brain, while the diagnosis may be very difficult, it differs in the unexplained abrupt attack, severe from the start; in the lowness of temperature during the first day or two; in the early tetanic tendency and the eruption in many cases. Malignant scarlet fever resembles it considerably at the onset; and so does the chill of pernicious intermittent. Locality and season will designate the latter; age and exposure, especially the former. Fortunately, the principle of treatment is not essentially different in these affections, at the stage which may present a doubt.

Prognosis.—More than half the cases die. Those who survive three days have a fair, though not certain, prospect of recovery.

Causation.—Of either sex more children, and of adults more males, die of this disease. Coincident with the circumstances of war, or military *régime*, most of its epidemics have been, though not all of them. The analogy which it presents to typhus suggests a probable relation of the disease in causation to local or atmospheric contamination. I can think of only one plausible hypothesis; that it depends upon a peculiar zymotic material, or "morbid poison," generated by a slow change in human or animal emanations, such as, in camp or garrison life, the long unwashed clothing of soldiers may particularly engender. Exposure to *cold* is thought to predispose to it.

According to Hirsch, in central Europe, very much the largest number of epidemics of it have occurred in winter; next, in spring; and the same has been found true in the United States. In 1871–2, a number of cases in New York City were traced by Dr. Moreau Morris to very *unsanitary local conditions*.[1]

There is no proof whatever of personal contagiousness in cerebro-spinal fever.

Treatment.—We must lament the unsatisfactory condition of the evidence upon this subject. Almost all agree that *asthenia* characterizes the disease, most of all at the beginning. The resemblance to pernicious fever has suggested the use of quinine; and several very positive statements are made of success with it in large doses, as two to four grains every hour or half hour until cinchonism is produced, or until from thirty to sixty grains have been taken; afterwards a grain or two every two or three hours. Some practitioners, upon trial, have abjured quinine altogether in this disease. Were the diagnosis sure in any case from the commencement, I should feel inclined to continue the trial of it, from what has been reported, in this city particularly, of its success.

Opium has equally enthusiastic (Boudin) advocates and opposers. *Early*, if it be given, must be the time. The idea of those who urge it is to give of it a grain every two or three hours, until an *opiate sleep* is produced; then to withdraw it and give it in much smaller doses.

[1] See also Clymer, on Cerebro-spinal Meningitis, 1872.

Bromide of potassium is favored by Dr. J. Lewis Smith[1] and others in the early stage, especially in children. The use of it is justified by the opinion that it tends to produce "contraction of the arterioles of the encephalon," and thus to relieve cerebral congestion. Dr. J. L. Smith has been disappointed with the effects of quinine in this disease. He recommends the application to the head in the early stage, of bladders or bags filled with bran mixed with pounded ice. Dr. Borland, of Boston, has successfully used bromide of potassium, with *ergot*.

Stimulation with brandy or whisky is generally employed in the first stage with freedom. Dr. Stillé considers, however, that general experience does not warrant its being used in all cases, but only when the signs of failure in nervous power occur. External stimulation is of course indicated; by mustard, direct heat, friction with red pepper and brandy, or hot whisky and salt, the *hot bath*, etc. Dry cupping, or in some cases cut cups (when reaction occurs), to the back of the neck, will be proper; followed by a blister at the same place. In Germany leeching behind the ears is said to have often proved useful.

Cantharides (20 to 40 drops of the tincture every hour till reaction), camphor, choloroform, sulphite of sodium, and hydrate of chloral,[2] have each had laudation from some who have used them. But more positive experience is needed to give the profession much confidence in the treatment of this affection.[3]

TYPHUS FEVER.

Synonyms.—*Ship Fever; Camp Fever; Jail Fever.*

Symptoms and Course.—For a day or two premonitory weakness, headache, and loss of appetite occur. Then a cold stage, of variable distinctness, begins the attack. In rare instances it is said that death takes place in this stage without reaction. Much more commonly fever follows, with severe headache, great heat of skin, pulse 120 (110 to 130), but compressible, tongue whitish or yellowish, bowels costive. Delirium is common, especially at night. The temperature in the axilla is from 102° to 108°; generally after the third day, 105°–6° in the morning, 106°–7° in the evening. Muscular debility is very decided.

For a number of days this condition lasts; the patient lying in a stupid half-sleep much of the time, muttering to himself, easily roused, but soon relapsing again; the face having a dusky flush of redness. Hardness of hearing is present in most cases. Positive coma is a very bad prognostic, but is not infrequent. Suppression of urine may take place in the worst cases; retention occurs in many severe ones. The tongue grows darker as the attack progresses; brown, even black; often cracked or fissured; and it as well as the teeth may be covered with sordes.

Toward the end of the first week, in most cases, a rash appears, of little and numerous red papulæ (miliary eruption), all over the

[1] Amer. Journal of Med. Sciences, Oct. 1873.
[2] Patton, Indiana Journal of Medicine, July, 1870.
[3] See J. S. Jewell, M.D., Report on Cerebro-Spinal Meningitis, Chicago, 1866; J. J. Levick, M.D., "Report on Spotted Fever, so called," Trans. Am. Med. Assoc., 1866; A. Stillé, M.D., Treatise on Cerebro-Spinal Meningitis, 1867.

chest, abdomen, and upper parts of the limbs. They are accompanied by *sudamina* (minute vesicles) in many instances, by *petechiæ* in a few. Sometimes a strong odor comes from the body; but I have never noticed this, even in the cases of ship-fever from Ireland in the Pennsylvania Hospital, in 1847–8, at which time I took the disease myself from them.

The urine is scanty. Generally it contains an excess of urea and uric acid, with a deficiency of the chlorides. Sometimes there is actually less than the normal amount of urea eliminated; excreta may then be supposed to accumulate in the blood, promoting coma. *Costiveness* is the general rule in typhus.

The *dicrotous* or double pulse, and *subsultus* or twitching of the tendons at the wrist, are common. Weakness of the impulse of the heart is often noticeable; sometimes so much so as to justify Dr. Stokes's diagnosis of "typhous softening." *Hypostatic* pneumonia (*i. e.*, beginning with passive congestion of the lungs posteriorly) is the most frequent complication of the fever.

The *duration* of an attack of typhus is generally three weeks. The critical period is usually about the eleventh day; after which *defervescence* (the decline of the fever) may be looked for. Occasionally death may take place within five days, or recovery within fifteen from the commencement.

Morbid Anatomy.—Absence of lesion of the solids has been repeatedly noticed. The blood is always altered during life; after the early stage it is less coagulable and darker in color than in health. Passive congestion in various organs is observed, as in the lungs, brain, liver, etc., but without anything characteristic.

Pathology and Causation.—No disease affords more reason for pronouncing it a disease of the blood than typhus. Its cause, demonstrably in many cases, is *ochlesis* or crowd-poison; the effluvia from human bodies, accumulated, especially in cold weather, in small and ill-built dwellings of the poor, and most of all in filthy towns, ships, jails, or camps. Having once been thus generated, it becomes contagious; one patient having, in his morbid emanations, the poisoning power of a whole crowd. Yet the contagion is not very strong; many who are exposed often escaping the disease. In giving this account of it, candor requires me to add that the spontaneous origin of typhus in any case, apart from direct, specific, personal contagion, is denied by such eminent British authorities as Drs. W. Budd, Parkes, and others.

Diagnosis.—After the first two or three days (during which there may well be doubt as to its character) the only probable question will be between typhus and typhoid fever. All medical authorities are not yet agreed as to the non-identity of the two forms of slow continued fever.[1] A large majority, however, regard them as quite distinguishable during life, and separated pathologically by the absence in typhus of the morbid alterations of Peyer's glands, and those of the mesentery, characteristic of typhoid fever. I have many times seen typhus and typhoid cases in the same ward, lying side by side, and should feel confident of being generally able to diagnosticate them by the countenance alone. Under the head of *Typhoid Fever*, the clinical differences will be enumerated.

[1] Dr. J. Hughes Bennett, for example, still maintains their identity; and some German writers call typhoid "abdominal typhus." The definite history of typhoid or "enteric" fever began with Prost, of Paris, 1804. Louis, 1839, studied it elaborately, showing the constancy of the intestinal lesions. In 1823, Dr. Enoch Hale, of Massachusetts, described two forms of continued fever. Dr Gerhard, upon careful autopsies in the Philadelphia Hospital, announced evidence of the distinctness of typhus from the typhoid or "dothinenterite" of Louis in 1835. Dr. A. P. Stewart, of Glasgow, published similar conclusions in the same year. In 1846, Dr. W. Jenner commenced an investigation into the subject, whose results most physicians have accepted as decisive. He concluded that typhus and typhoid fevers are clinically and anatomically distinct, as well as different in causation. Dr. Gairdner has recorded cases in which patients convalescent from typhoid fever have taken typhus upon exposure to its contagion.

Prognosis.—Murchison states the mortality in the hospitals of Great Britain, from typhus, to be one death in five cases. Cheyne and others in private practice have found it but one in twenty or more. I have not seen many deaths from it, in private or hospital practice. Probably one in ten or fifteen would be a fair general estimate. Bad signs are, great feebleness or extreme rapidity of the pulse; profound coma; hiccough; suppression of the urine; involuntary defecation. Pneumonia complicating the attack increases its danger, though I have known several recoveries notwithstanding this.

Treatment.—More than half the cases of typhus, according to my observation, require alcoholic stimulation, as well as concentrated nourishment, after the fourth day. But not all the cases; as my own, among others, proved. I was bled on the second day, the diagnosis not being made out; and leeched on the third day, freely, on the back of the neck; yet no stimulus was required, after the typhous nature of the attack was clearly shown; recovery following at the usual time. Drs. Russell and Gairdner[1] have shown (in the Glasgow Fever Hospital), that typhus may be treated, in many cases, *without alcohol*. In nearly a thousand cases, their mortality was about nine per cent.

We may begin the treatment of an ordinary case of typhus with a mild laxative—*e. g.*, a *moderate* dose of solution of citrate of magnesium, on the second day. The diet at first may be of gruel, toast-water, etc.; but very soon must milk and beef-tea or chicken or mutton-broth (or an alternation of these) be given to support the strength. Before the first week is out, half the cases will need wine in moderation; some, brandy or whisky. In the second and third week, more than half the cases will require steady support of a positive kind. In such cases, the proper routine is, a tablespoonful of brandy or whisky punch (one part of spirit to three, two, or one of milk) every two, three, or four hours, and, the alternate hours, a tablespoonful or two of beef-essence or beef-tea.

Of medicines, quinine has had the most extended trial in typhus. It acts well as a tonic, in one or two grain doses, four or five times daily, after defervescence has begun; *i. e.*, after the tenth or twelfth day usually. Dr. Dundas's plan of treating typhus early with *large* doses of quinine is, I am satisfied, after seeing some trial of it, futile and even unsafe.

[1] British Medical Journal, Aug. 22, 1868.

Mineral acids have acquired much reputation in typhus. Dr. Flint advises dilute sulphuric acid. Nitro-muriatic acid I have known to produce an excellent effect in the depression of the middle stage. Large doses are not required; but the acid should be given several times in the day. Some prefer dilute nitric acid [F. 165, 166]. *Chlorine water* is lauded highly by others. Sulphite of sodium and carbolic acid may be worthy of trial.

But the great point of skill will be to determine when and how far to stimulate. Delirium favors the probability of its being needed; especially a low, muttering delirium. Of course a very feeble pulse indicates it. On trial, when the pulse grows slower, the skin more moist, and the restlessness or delirium is quieted, the stimulus has done good, and should be continued. If, on the contrary, a more hurried or a *harder* pulse follow, with heat of head and dryness of skin, and wilder delirium or deeper stupor, it should be stopped, for a while at least, or, if given, be diminished in amount.

Catheterism may be needed for retention of urine. Inquiry and inspection should determine every day the state of the bladder. Constipation, through the attack, may be overcome by enemata, or by small doses of oil, Rochelle salt, or other mild laxative.

When the coma is very deep, a blister to the back of the neck may do good; and so may sinapisms to the extremities. Great heat of the head may render proper, especially in the first week, the application of cold water to the head. Sponging the whole body daily (best at night) with whisky and water, warmed, is extremely comforting and beneficial.

Hypostatic pneumonia in typhus cannot be treated actively. Even abstraction of blood by cups is hardly ever to be ventured upon. *Dry* cups, between the shoulders, and a blister upon the breast, are about all the special treatment allowable. It is, however, possible generally to *prevent* hypostatic pneumonia, by not allowing the patient ever to lie for many hours together upon his back. Let him be turned, once in a while, upon one or the other side.

Prophylaxis.—Thorough *ventilation* is the only security against the generation of typhus fever; and this is capable also of almost disarming its contagion.

TYPHOID FEVER.

Synonyms.—*Slow Nervous Fever; Common Continued Fever; Enteric Fever; Abdominal Typhus* (Pythogenic fever of Murchison).

Symptoms and Course.—After a more gradual approach than that of any other fever, with languor, and debility, anorexia and headache, for several days—bleeding at the nose and a bronchial cough are almost pathognomonic early symptoms. The patient takes to bed, with fever of considerable violence. The face acquires a dark purple flush. He lies dozing, perhaps muttering, unless disturbed, all day; but is more or less wakeful and delirious at night. Hardness of hearing is common from the middle of the second week. Swelling of the belly (tympanites) comes on towards

the end of the first week; diarrhœa about the same time. Rose-colored lenticular spots (*taches rouges*), disappearing on pressure, are discoverable, few in number, and on the abdomen only, toward the end of the second week; they continue a week or two. Tenderness on pressure in the right iliac region, with gurgling under the hand, generally exists. Sudamina over the chest are not unusual. The duration of the typhoid pyrexia is seldom, from the start, much less than two weeks, and it is often more; the whole attack of typhoid fever may be protracted, as I have seen it, to two or three months. One month may be considered the average time, from taking to bed to leaving it convalescent.

Late symptoms in severe cases are, the dicrotous pulse, subsultus tendinum, retention (perhaps suppression) of urine, hemorrhage from the bowels; and, if death be imminent, hiccough, cold sweats, involuntary discharges.

In protracted cases, great emaciation and bed-sores may supervene. Even during convalescence, abscesses in various parts of the body may give trouble. These usually affect the glands or connective tissue, but may occasionally involve the long bones.

Danger of perforation of the intestine, from deep ulceration of the glands of Peyer, exists always after the first week, until late in convalescence. Patients out of bed for a week or two have sometimes died, after imprudence, from this cause. Dr. Harlan reports[1] a case in which fatal perforation took place four months after the commencement of an attack of typhoid fever. Dr. Da Costa mentions it as happening after seven months, from error in diet. The occurrence of perforation is recognized by symptoms of severe peritonitis, with collapse. The result of this is almost inevitably fatal; the only recorded exception being reported by Prof. G. B. Wood. I saw a case of suppurative peritonitis, opening externally, which recovered, in the Philadelphia Hospital, several years ago; but I was not able to learn the antecedents of the case.

Temperature.—This has, of late, been made a special study in typhoid fever. The rise from 98.5° (the normal degree) is gradual, during the first four or five days; reaching 104° on the evening of the latter; sometimes 104.5°. An attack of disease in which on the second day the heat in the axilla is as high as 104°, is not typhoid fever; and the same exclusion applies if from the fourth to the eleventh day the temperature falls below 103°. A difference of 1° or 1.5° between morning and evening (greatest heat, the latter) is usual; the reverse is not a good sign. The marked tendency to a morning fall and evening rise of temperature is almost pathognomonic of typhoid fever. Toward the end of the second week, lowering of the heat below 103° is always favorable; persistence at 104°, 105°, or 106°, shows a severe case; the higher the worse. Sudden increase of temperature indicates a complicating inflammation; as pneumonia.

Discharges.—Liquidity of the stools is a characteristic of this disease, even if there be but one daily. Generally, after the middle of the first week, there are two or three passages, brownish with a slight yellowish tinge, every day. From the very begin-

[1] Trans. Philada. Pathol. Soc., 1859.

ning of the attack, the bowels are unusually susceptible to the action of purgatives; a teaspoonful of castor oil operating readily. Excessive diarrhœa, at a middle or late stage, not unfrequently adds to the prostration of the patient. Hemorrhage from the bowels, when it occurs, is most apt to be met with in the second or third week.

The *urine*, through the attack, is commonly scanty, high-colored, excessive in the amount of urea, deficient in the chlorides, and sometimes albuminous in severe cases.

Complications.—*Pneumonia*, especially the hypostatic form (as in typhus), is the most frequent. It has been, by some writers, denied that true pneumonitis, anything more than passive congestion, occurs in these cases. But, in the analogous instance of typho-malarial fever, especially when the scorbutic diathesis was also present, I have seen, after death, more than once, suppuration as well as hepatization, confined altogether to the posterior portions of both lungs. I do not doubt the same happening in typhoid as well as in typhus fever.

Inflammation of the brain may complicate typhoid, more often than typhus; but it is not common.

Peritonitis follows always when perforation of the ileum takes place. Examples of its occurrence without that accident are said to have been, though very rarely, observed.

Sequelæ.—Prolonged debility, or a very slow convalescence, is common. The mental faculties are sometimes enfeebled for weeks or months. Paralysis is an occasional sequela. Abscesses have been mentioned. Periostitis, followed by necrosis, of the tibia, femur, or humerus, may happen, though I have known of but two such cases. Perforation of the bowel may, as already stated, occur after convalescence has seemed to be established.

Morbid Anatomy.—Omitting variable and unessential or occasional appearances, the parts characteristically affected in typhoid fever are, the agminated glands or patches of Peyer in the small intestine, the mesenteric glands, and the spleen. Careful study of Peyer's glands, by many observers, has shown that, at first, the glands thicken and become elevated from one to three lines above the membrane around them. They are generally at this time reddened; but with variable depth of hue. Sometimes, after this, a sort of induration occurs; in other instances, softening. Later, ulceration affects many, though not all, of the altered glands; and this process may go on until, as above said, it may perforate all the coats of the intestine. This, however, is exceptional. The healing of the ulcers by granulation is the general rule. Dr. Murchison has reported fatal cases of *sloughing* of the intestine *without perforation*.[1]

The *solitary closed glands* of the small intestine are also commonly enlarged, and often softened or ulcerated. The *mesenteric* glands are almost uniformily enlarged, congested, and softened; occasionally they suppurate.

The *muscles*, especially the *recti abdominis*, in protracted cases, have been shown to undergo a granular, or sometimes a waxy or

[1] Trans. of Pathol. Soc. of London, 1866, p. 130.

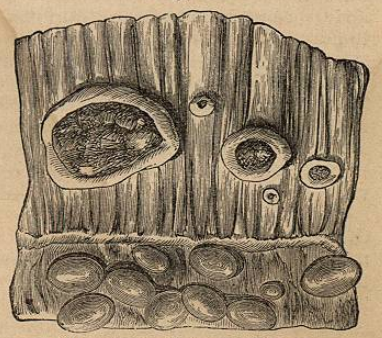
amyloid degeneration; resulting, in the rectus, occasionally, in rupture of its fibres.

Pathology.—Typhoid fever is believed by most authorities to be a general or systemic disorder, with a characteristic secondary local lesion in the intestines. How far the matter deposited in the patches of Peyer before ulceration is *specific*, is a question. Rokitansky and Carl Wedl believe it to be peculiar—the former comparing it to that of encephaloid cancer, the latter to tubercle. I do not believe that there is anything properly to be called *specific* in its nature.

Dr. G. B. Wood holds the opinion that an inherent predisposition to the disease exists in many persons, analogous to the tuberculous, gouty, and rheumatic diathesis. This seems very probable. Dr. H. Kennedy, of Dublin, has (1873) advanced a similar opinion.

Another view is, that the affection of the intestine is primary; and that the "typhoid" symptoms result from the absorption into the blood of morbid, putrescent material from the glands of Peyer, producing a *septæmia* or *ichoræmia*. This view does not appear to me to be confirmed by the usual order of events in the disease.

Fig. 86.



Ulceration of glands of Peyer.

Causation.—More doubt exists as to this in typhoid fever than in regard to any other common disorder. Depressing causes of all kinds seem to promote it; foul air, removal from home, fatigue, anxiety, etc. Yet it will occur in the entire absence of all such causes. No locality limits it; all climates allow it; from the Arctic regions to those bordering upon the tropical; from the cities of the East to the Rocky Mountains. The "mountain fever" of hunters in the far West was found in the autopsies of Dr. Hammond to present the lesions of Peyer's and the mesenteric glands.

Such universality is very much in the way of the "pythogenic" theory of Murchison (*i. e.*, its reference always to foul air, as that of sewers), or that of Budd, that its only cause is a specific matter, passed from the bowels of those having it, and by water or air, conveyed into the systems of others.

Contagion of this kind is, nevertheless, widely believed in now, especially in England. Some facts asserted (Flint, Canstatt, Budd, Watson) in proof of it are hard to explain without admitting such a mode of propagation (*e. g.*, by the discharges of a patient getting into a well, etc., so as to contaminate drinking-water). Dr. Ballard has reported[1] instances in which it appeared to be propa-

[1] Brit. Med. Journal, Nov. 26, 1870; Lancet, April 5, 1873.

gated by the milk served by dairymen. The pans used for the milk appear to have been washed with water exposed to contamination. A more extensive series of cases in the west end of London, in the summer of 1873, was traced to the same mode of propagation.[1] Prof. I. Buckman asserts the observation of a peculiar "fungoid or confervoid" growth in water, contaminated by sewage or otherwise, and productive of typhoid fever. But the large majority of cases allow of no such explanation; most of all those occurring in the open country.

There is no doubt that typhoid and typhus fevers may *coexist* as epidemics; sometimes affecting the same patient, the one fever shortly after the other (Gairdner); and occasionally together, as a hybrid disease. This may help us to account for some instances in which foul air has appeared to generate typhoid, and where the latter has seemed to be contagious. My own experience leads me to adopt the view expressed by Niemeyer, Anstie, and others, that "typhoid fever is certainly not contagious in the same sense as typhus is." Dr. Murchison[2] states that, in the Fever Hospital, in fourteen and a half years, with 2506 cases of typhoid fever admitted, only eight new cases originated in the hospital.

Dr. Jas. E. Reeves, of Wheeling, Virginia, asserts his conviction, based upon twenty years' observation of typhoid fever in village and rural practice, that while it may originate *de novo*, it is also positively, though feebly, contagious. In some places in the country, suspicion has recently fallen (not altogether without reason) upon the too close proximity of *privies*, or foul drains, to *drinking wells*, as a promotive cause of typhoid fever. Dr. Austin Flint[3] writes thus: "under ordinary circumstances it is not diffused by contagion;" "facts appear to show conclusively the spontaneous generation of the causative agent in the great majority of cases."

Typhoid fever is rarest in old age; not frequent in childhood; most common between fifteen and thirty years. Few have it under ten or over forty; almost none beyond fifty. It scarcely ever (relapses apart) occurs a second time in the same person.

Diagnosis.—From *remittent* fever, typhoid is known by the absence of vomiting and of sallowness of the skin, the slower onset, more protracted course, the *hebetude* or mental dulness and drowsiness, and the abdominal symptoms. Vomiting occurs in children.

From *typhus* fever, the distinctive points are as follows:—

In Typhus.	*In Typhoid.*
No epistaxis nor bronchitis;	Epistaxis and bronchitis;
Bowels constipated;	Diarrhœa;
Belly seldom tympanitic;	Tympanites, gurgling, etc.;
Miliary eruption, 5th to 7th day;	Lenticular rose spots;
Progress moderately slow;	Progress very slow;
Death often within ten days;	Death rarely within 14 days;
Countenance dusky red;	Countenance purplish red;
Causation mostly obvious;	Origin obscure;
Anatomy not peculiar.	Lesions characteristic.

[1] Lancet, Aug. 16, 1873.
[2] On Fevers, p. 428.
[3] Practice of Medicine, 4th edition, 1873, p. 883.

As Dr. J. W. Miller[1] has pointed out, the duration of elevated temperature is, in typhus, rarely longer than eighteen days; in typhoid, seldom less than twenty-one days, and often more than thirty. In typhus, also, the evening temperature is, not unfrequently, lower than that of the morning; in typhoid, the evening temperature is almost constantly higher than that of the morning.

Cases called "febricula," or "irritative fever" (formerly "synochus") are described by some writers, and met with once in a while in practice, which give a good deal of trouble in diagnosis. Some of these, probably most of them, are mild examples of typhoid fever. Some may be called *walking* cases; the patient being able to keep out of bed.

Prognosis.—The mortality from this disease varies greatly under different circumstances. The possibility of perforation of the ulcerated bowel gives an element of uncertainty to every case. Probably one death in twenty cases will represent its average mortality. The favorable and unfavorable symptoms, other than those common to typhus or other febrile affections, have been indicated sufficiently already in our account of the disease. The state of the tongue, especially at the period of defervescence (end of second week, about), should always be noticed, as it aids our observation of the abdominal symptoms in concluding upon the progress of the intestinal lesion.

Treatment.—Self-limited as typhoid fever is, no *cutting short* of it is possible. We must *conduct* the patient through it as safely as possible. For this, little medication, perhaps none, will suffice, with good nursing, in many cases. I have treated the disease with so little medicine, that it might be said to have been left to nature, supported by regulated liquid nourishment alone. Yet this is not always proper or safe.

The course of treatment learned in the Pennsylvania Hospital more than twenty years since, has been followed throughout my practice with generally successful results. My only deviations from it have been in the direction of diminishing the amount of medicine given. It was, upon the average, as follows:—

In the course of the first few days, if the bowels were costive, a teaspoonful of castor oil was given; after that, no laxative. During the first week, while the fever was highest, the tongue furred and often dry, skin hot and without perspiration, small doses of blue mass with ipecacuanha were prescribed, with the view of favoring freedom of the secretions. Afterwards, or at the same time, spiritus mindereri (liquor ammonii acetatis) was given, a tablespoonful (diluted) every two or three hours, from noon till near midnight, as a diaphoretic.

Liquid food is necessary from the first. Oatmeal gruel, toast-water, rice-water, the first three or four days; then milk may be added, one or two tablespoonfuls every two or three hours. *Less than half* the cases of typhoid fever which I have seen have required alcoholic stimulation at any stage; not more than one-fourth of the cases need it before the middle of the second week.

[1] Brit. and Foreign Medico-Chir. Review, Oct. 1868, p. 464.