so, as the muscles of mastication were powerful and paresis of the soft palate was very slight. Ataxia of the muscles of the face and tongue and of mastication became so great that when the patient attempted to speak he showed great contortion of the face. All these disturbances were probably the result of alteration of the fifth nerve. In another case in which implication of the fifth nerve existed, chewing and swallowing were difficult, the patient did not know whether any food were in her mouth, and she could extend the tongue properly only when she watched the movements of the tongue in a looking-glass. In both these cases the spinal root of the fifth nerve was degenerated on each side.

Oppenheim¹ says that Duchenne spoke of implication of the fifth nerve in tabes. He also says that the symptoms attributed to degeneration of the spinal root of the fifth nerve have varied considerably in different cases.

The constant biting of the cheeks, for the relief of which in 1886 Dr. Cohen's patient had several teeth extracted, and the absence of pain upon their removal, as well as the loss of food in the mouth, were probably the result of degeneration of the spinal roots of the fifth nerves.

The descending root of the vago-glossopharyngeus is occasionally found degenerated in tabes. Cassirer and Schiff collected twelve cases in which degeneration of this bundle was observed, but only in six of these was the microscopical examination thorough. The absence of disturbance of taste in these six thoroughly studied cases seems to show that integrity of the solitary bundle is not necessary for the function of taste. Disturbances of the functions of the larynx observed in these six cases were not necessarily the result of degeneration of the solitary bundle, because there was also degeneration of the bulbar nuclei or of the peripheral portions of the vagus and glossopharyngeus nerves. In one of the twelve cases the solitary bundle was degenerated, but clinical signs indicative of this degeneration had not been observed. Cassirer and Schiff conclude that the solitary bundle has no relation to the function of taste, or of sensation or motion in the larynx, or to deglutition. The degeneration of this bundle has no relation to gastric crises or disturbances of respiration. Cassirer and Schiff found the large fibres in the solitary bundle degenerated, while the fine fibres were preserved and formed a group on the dorso-median portion of the bundle. In Schlesinger's² case both descending glossopharyngeal roots were degenerated, and, according to Schlesinger, the degeneration of these roots has been found in tabes especially when laryngeal disturbances had been present, and yet he merely calls attention to the frequency of the association without expressing himself very positively in regard to the importance of it. It seems from his investigations, also, that degeneration of these roots does not cause disturbance of taste.

It is possible, however, that the feeble and irregular conduction in Dr. Cohen's patient was the result of the degeneration of the descending root of the left vagoglossopharyngeus.

The dorsal nucleus of the vagus has been found degenerated in tabes, and was so in one of Cassirer and Schiff's cases, but these authors were unable to observe positive degeneration of the nucleus ambiguus, and I also have been unable so to do in the sections studied by me.

Cases of bulbar tabes with necropsy are very rare, and v. Reusz,¹ in publishing a case of this kind, says only two other such cases have been reported since the appearance of the paper by Cassirer and Schiff, viz., a case by Grubner and Oppenheim and one by Zeri, but the case by Zeri is probably referred to by Cassirer and Schiff.

SOME OBSERVATIONS ON TYPHOID FEVER COMPLICATED BY CROUPOUS PNEUMONIA, WITH REPORTS OF FOUR FATAL CASES.

BY HENRY M. FISHER, M.D.,
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MANY text-books do not even mention croupous pneumonia as a complication of typhoid fever.

As Niemeyer says,² it can easily be overlooked in the same way as the pleurisy with effusion that also occasionally occurs as a complication of typhoid, and it, like the latter, is often unattended either with pain, chill, or increased fever.

That it is not a very unusual complication is shown by the study of Liebermeister's³ cases in the epidemic of typhoid fever at Basle, 1865-1868, when consolidation of the lungs, not due to hypostatic congestion, was noted fifty-two times among 1420 cases, giving a percentage of the complication of 3.6. That it is a very serious complication is shown by the fact that of these fifty-two cases reported by Liebermeister⁴ and Betke,⁵ twenty-nine died, showing a mortality, therefore, of 55.7 per cent. Gnllisart de Mnrigne⁶ reports thirteen cases with only three recoveries, a mortality for the complication, therefore, of 77 per cent.

Other French observers, as L. Girard,⁷ Coste,⁸ and G. Destaix,⁹ all report a number of cases with a low percentage of mortality. I think,

¹ Archiv f. Psychiatrie, 1899, vol. xxii., No. 2, p. 535.

² Lehrbuch der speciellen Pathologie und Therapie.

³ Ziemssen's Cyclopaedia.

⁴ Loc. cit.

⁵ Die Complicationen des Abdominal Typhus. Berlin, 1870.

⁶ Pneumonie lobaire. Survenant dans le cours de la Fièvre Typhoïde. Paris, 1877.

⁷ De la fièvre typhoïde à début pneumatique. Paris, 1882.

⁸ Contribution à l'étude des accidents pulmonaires de la fièvre typhoïde. Paris, 1879.

⁹ Considerations sur quelques accidents pulmonaires, et sur la pneumonie pseudolobaire en particulier, survenant dans le cours de la fièvre typhoïde.

however, that this low mortality must have occurred in an epidemic of an unusually mild type. When we consider the extreme prostration of patients in the second, and particularly the third, week of a severe attack of typhoid fever, an involvement of any large area of their lungs by the pneumonic process can hardly fail to increase very much the gravity of the prognosis.

The etiology of the complication is an interesting one.

It seems probable that in many cases the pneumonitis is due to a mixed infection. In fact, both typhoid bacilli and pneumococci have been recovered from the lungs of patients who have died of pneumotyphus. In other cases it seems reasonable to believe that the pneumonitis is purely septic. Such are the cases where a pneumonitis develops with great rapidity and with fulminating symptoms, perhaps late in the course of the typhoid fever, and is rapidly fatal. One such case occurred during my late service at the Episcopal Hospital, the fourth on my list. This developed in a boy who, after several weeks of fever of rather an indefinite type, suffered from an acute otitis media. Two or three days later his lungs, which had been clear before, rapidly filled up, and he died within twenty-four hours after the first signs of pulmonary congestion. Unfortunately, in this case cultures were not made from the lungs. The intestines showed apparently recently healed typhoid ulcers.

Dr. D. Betke's¹ observation would seem to show that the complication has been more frequent since the cold bath treatment has been in vogue than before, but that the mortality from the complication since the treatment has been instituted has been considerably less. While it may be difficult or impossible to make the diagnosis of typhoid fever when pneumonitis occurs, as it sometimes does in the various stages of typhoid fever and masks the symptoms of the latter, a high degree of leucocytosis occurring in the second or third week of typhoid fever ought to make us suspect the occurrence of this complication.

As Destaix² observes, the normal leucocytosis of the first week of typhoid fever diminishes rapidly in uncomplicated cases after the seventh day of the disease. Often, too, but by no means invariably, the onset of the croupous pneumonia is announced by a marked rise in the temperature and an increase in the respiration rate. The sharp pain in the side and the rusty sputa are seldom observed.

In the following case the diagnosis was unfortunately not made, the pulmonary symptoms having been, perhaps, unusually pronounced. The marked abdominal pain and tenderness were supposed to have been caused by a rapidly developing tubercular peritonitis.

¹ Loc. cit.

² *Considerations sur quelques accidents pulmonaires survenant dans le cours de la fièvre typhoïde.* Paris, 1877.

It will be observed in this case that while there was a distinct leucocytosis noted on the third day after her admission, on only one occasion, three days before death, was a weak positive Widal reaction noted.

CASE I.—Jennie R., aged sixteen years, was admitted to the Episcopal Hospital under my care on January 31, 1901. Notes by Drs. Steel and Francis Sinkler.

Two years ago the patient was in the hospital with nervous prostration. One week before admission she was seized with a sudden pain in the right side, accompanied by a chill and fever. Cough began three days ago, unaccompanied by expectoration. Upon admission she complained of muscular pains, shortness of breath, and cough. Her bowels were opened three times on the day of her admission. Complete anorexia and insomnia. Temperature on admission was 99.4°. Breathing labored and rapid; face flushed, extremities cold. Friction sounds heard all over right chest except at apex, and mucous râles over base posteriorly, and tubular breathing heard over a limited area of the left lower lobe posteriorly, and mucous râles over upper lobe. Abdomen negative. Urine shows a faint trace of albumin, but no casts.

CHART 1.

February 1st. Condition fair. Leucocytes 17,800.

2d. Rigidity and extreme tenderness of the whole abdomen, with slight tympanites. Pulse weaker.

4th. Tubular breathing and crepitant râles over whole left lung.

5th. Widal reaction negative.

6th. Urine shows a faint trace of albumin, one granular cast, granular epithelium, and chains of bacteria.

9th. Widal reaction still negative; less cough. Abdomen tympanitic and very sensitive to the touch. No spots. Pulse rapid and slightly irregular. Breathing rapid and entirely costal. Two loose stools.

11th. Leucocytes, 9000. Oxygen administered last night; improved pulse and respiration temporarily, though latter still much labored.

13th. Widal reaction still negative. Leucocytes, 6200.

16th. Weak positive Widal. Slight leucocytosis.

18th. Widal negative; no leucocytosis; patient weaker; respiration shallow.

19th. Patient died at 6.30 A.M.

Autopsy revealed bed-sore over sacrum. Abdomen full of foul-smelling pus, partly free and partly in abscess lying between and around the organs. Pelvis contained about one pint of pus. Mesenteric glands enlarged.

Intestines. Ileum shows ulcerated Peyer's patches. There is one perforation at the hepatic flexure of the colon and another about three inches above the sigmoid flexure. Both perforations are at the seat of ulcerated Peyer's patches.

Liver enlarged and shows fatty degeneration.

Spleen large and soft. Pulp semi-liquid.

Kidneys. Right: atrophic, of the size of the last joint of the thumb. Left: about twice the normal size; no pathological change.

Thorax. Visceral pleura adherent to parietal over entire surface except at base of chest. Half a pint of sanguino-purulent fluid in upper surface of diaphragm. Hepatization of lower lobes of both lungs.

Heart. Pericardium not inflamed; no increase of normal fluid; mitral valve showed vegetations on each leaflet.

In the following case congestion of the lungs was noted eight days after admission or probably toward the end of the second week of the fever. Distinct evidences of pneumonia were not, however, found until the twenty-first day after her admission to the hospital. In this case the attack of pneumonia was ushered in by a distinct chill, and the patient had also rusty sputa and dyspnoea.

CASE II. *Typhoid fever, complicated by croupous pneumonia* (from hospital record of Dr. Steel).—Amelia P., aged nineteen years, single, born in England, mill hand, was admitted to the Episcopal Hospital, January 3, 1900. Has suffered from chlorosis, with a good deal of gastric and intestinal indigestion. Three weeks before her admission to the hospital she was obliged to stop work on account of weakness, vertigo, and headache. No epistaxis or diarrhoea, but insomnia.

On admission her temperature was 103.2°. She was pale, but appeared fairly well nourished. Her conjunctivæ slightly jaundiced; her cheeks flushed; her tongue coated, but red at the tip and edges; slight prolongation of the expiratory murmur at the right apex; a soft systolic apex murmur. Her abdomen was slightly tympanitic, and there was tenderness in both iliac fossæ. No rose spots. Splenic dulness extends upward to the seventh space. Liver dulness normal.

Blood Examination. Widal, negative; hyperleucocytosis, 10,000; red blood-corpuscles, 4,608,000; hæmoglobin, 60 per cent.; index, 65.2 per cent.

Urine. Specific gravity, 1014; acid; no albumin; no glucose; no casts; epithelium and leucocytes.

January 7th. Blood shows a positive Widal reaction to-day.

9th. Stools light, partly formed. Crops of rose spots developed to-day. Spleen palpable on deep inspiration.

11th. Hard cough for past twenty-four hours. Lungs show considerable congestion posteriorly. General condition good.

14th. Diminished resonance with sibilant râles all over left lung. Complaints of constant headache. Red blood-corpuscles, 4,728,000; hæmoglobin, 65 per cent.; index, 70 per cent.

22d. Cough not so severe and looks decidedly better. Lungs still full of sibilant râles. As the cold sponges fail to reduce temperature, this afternoon at 7, patient's temperature being 104° , she received a plunge bath, and her temperature fell to 101.6° . There was a little shivering after the bath. On the 23d she had three plunge baths, her temperature having risen to 104.8° . At 4 P.M. she had a distinct chill.

24th. Patient's temperature rose to 105° to-day, and she was slightly delirious. Baths stopped. Bases of lungs more congested, and there is a distinct area of tubular breathing just below and to the inner side of the lower angle of the left scapula; sputum rusty. She had another chill last night.

25th. Temperature rose at 4 A.M. to 105.5° . This morning tubular breathing is heard over a more extended area of the left lung. Dyspnea has increased, and at 4 P.M. her respirations were 40 in the minute. Examination of the blood shows a marked leucocytosis. Urine shows a distinct trace of albumin, but no casts.

CHART 2.

26th. At 7 this morning patient's pulse was 148, and her respiration 40. This afternoon at 4 o'clock her pulse had dropped to 116 and her temperature to 101.5° . There were signs of consolidation of the whole upper lobe of the left lung and of the upper two-thirds of the lower lobe. Bowels loose, and again of the "pea-soup" character. At about 5 o'clock in the afternoon there had been rather a sudden marked change for the worse. Her pulse was very weak—160 to 180—and she was in a state bordering on collapse. My resident, Dr. Steel, exposed the median basilic vein and injected about one pint of hot normal salt solution, but during the operation dyspnea increased and her color became livid. Immediately afterward strychnine, grain one-twentieth; nitroglycerin, grain one-fiftieth, and whiskey, twenty minims, were injected hypodermically, and this injection was repeated in about an hour.

The injections and salt solution appeared to give slight temporary relief, but in spite of very free stimulation no marked improvement occurred. She died at 6 o'clock in the morning.

27th. In reference to the clinical notes I see that the patient had

been receiving strychnine sulphate, grain one-sixtieth to one-fortieth, and ammonium carbonate, grains ten, every alternate four hours since January 21st; also whiskey in half-ounce doses every three hours, and on the 23d or 24th she was also given tincture of musk in twenty minim doses every three hours. No autopsy was permitted.

In the next case the occurrence of rather a high degree of leucocytosis—45,300—on the tenth day after the patient's admission, or probably toward the end of the third week, coincidently with the physical signs of pneumonia and marked rise in temperature, was interesting. It is to be regretted that an autopsy could not be secured.

CASE III.—Edward M., aged sixteen years, was admitted to the Episcopal Hospital under Dr. D. J. Milton Miller's care, January 29, 1901. Notes by Dr. Ziegler. Patient had been ill for seventeen days and confined to bed for a week. No epistaxis and no cough, but some diarrhoea. Constant headache and occasional delirium. Face flushed and has a listless expression. Lungs clear, heart negative. Spleen

CHART 3.

somewhat involved. Abdomen moderately distended and tympanitic, and numerous rose spots on its surface. A good deal of hebetude, and at times somewhat delirious. Both buttocks are reddened and excoriated, and over the right great trochanter there is a small area of superficial ulceration. Urine shows a decided trace of albumin, but no casts. Diazo-reaction present. Widal positive. Leucocytosis absent.

January 31st. Has had small doses of morphine for the last two nights to give him rest. This has been effectual, though he moans a good deal in his sleep. Is very delirious. Bowels very loose, and stools thin and yellowish and passed involuntarily.

February 2d. Noisy at night; involuntary passage of urine and feces. Bed-sore on his back.

5th. Low muttering delirium. Did not react well to plunge. Trional and chloral were given to secure sleep. He slept after this, but moaned and talked in his sleep. Peripheral circulation weak.

6th. Condition improved. Plunges were given at temperature of 90°.

7th. Suggestion of tubular breathing; no other change observed, except some dyspnoea.

8th. Still noisy at night and sleep disturbed. Restless also during the day; very nervous. Reflexes increased; ankle-clonus present. Complaints of pain in his head. Leucocytes, 45,300.

9th. Pulmonary second sound accentuated. Right heart dilated, right border extending to right border of sternum. Upper border of heart dullness begins at third rib. Liver dullness stops two inches above the central margin in mid-clavicular line. Whole left chest from spine of scapula down is dull on percussion. Tubular breathing over whole lower lobe. About 11 o'clock face and hands became cyanosed. Respiration and heart's action much embarrassed. Radial pulse scarcely perceptible. Under influence of oxygen and hypodermic of camphor condition improved, and patient broke out into a heavy sweat. Continued to perspire all day, and he seemed somewhat better during the afternoon. At about 7.30 p.m. he grew rapidly weaker and died at 8.30 p.m. Temperature before death rose to 108°.

In the following case the absence of positive symptoms of typhoid fever while the patient was at the hospital, and the absence of a marked Widal reaction at any time, would have rendered the diagnosis doubtful. The post-mortem findings appeared, however, to show pretty conclusively that the patient was convalescing from that disease, and I have, therefore, felt justified in including the case in my list.

CASE IV.—H. C. G., aged six years, colored, was admitted to the Episcopal Hospital on January 19, 1901. Patient had been ill for three weeks, complaining of pain in chest and abdomen, and had vomited almost all his food for the past ten days. His bowels were constipated. He had been very drowsy. Had had severe epistaxis for three days. Temperature on admission 104°. His tongue was heavily coated, the mucous membrane of his pharynx much congested, and his tonsils were swollen. A few moist râles were heard over the bases of both lungs. The apex-beat of his heart was in the sixth space within the nipple line, and a soft systolic murmur was heard over the body of the heart. There was tenderness in both iliac fossæ. Liver and splenic dullness normal; no rose spots. Stools frequent, loose, dark brown, and contained some greenish flakes and curds.

January 22d. Had severe nose-bleed last night, losing three to four ounces of blood. Widal reaction negative. No leucocytosis.

27th. Widal still negative. Apparently slight leucocytosis. Urine normal.

30th. Temperature has been normal or subnormal at times during the past two days, with slight febrile exacerbations. Stools dark and partly formed.

February 4th. Leucocytes, 20,000. Heart and lungs negative. No abdominal symptoms. Throat tender externally. Severe epistaxis

occurred several times, and it became necessary to pack the nasal cavities.

6th. Thin, clear discharge appeared from the right ear this morning. Had complained of pain in this ear for twenty-four hours previously.

7th. Ear still discharging profusely. Abdomen and chest negative.

8th. Complained this morning of intense pain in the left ear. Vomited milk once to-day.

9th. Still complains of ear, and also this morning for the first time of severe pain in chest. His temperature at 1 o'clock rose to 105.5° and his respiration to 40. Fine moist râles were heard over both lungs, and tubular breathing heard over the whole of the lower lobe of the right lung. Abdomen rigid and tender. Pulse became rapidly weaker, and he died at 8.45 on the morning of February 10th.

Autopsy. Both pleural cavities contain about one pint each of clear serum; the right also some lymph flocculi. The right lung was the seat of croupous pneumonia, involving the lower lobe of the apex of the upper lobe anteriorly. Plastic lymph on visceral pleura over consolidated area, and there was a gelatinous puriform collection anteriorly between the lobes.

Pericardial cavity contained a normal amount of clear serum, but there were numerous bright-red petechial spots in the visceral layers of the pericardium and on the outer surface of the heart.

Liver shows cloudy swelling.

Spleen firm and not much swollen.

Kidneys show cloudy swelling.

Intestines. Peyer's patches swollen, some resembling typhoid ulcers at an early stage of the disease, and others presenting the appearances commonly seen in the seventh, eighth, or ninth weeks after typhoid. The mesenteric glands and solitary follicles were also considerably enlarged.

Brain weighed forty-nine and one-quarter ounces, and there was a small subdural hemorrhage over the median fissure in the parietal region.

REVIEWS.

A SYSTEM OF PHYSIOLOGIC THERAPEUTICS. A Practical Exposition of the Methods Other than Drug-giving Useful in the Treatment of the Sick. Edited by SOLOMON SOLIS COHEN, A.M., M.D., Professor of Medicine and Therapeutics in the Philadelphia Polyclinic; Lecturer on Clinical Medicine at the Jefferson Medical College, etc. Vols. I. and II., Electrotherapy, by GEORGE W. JACOBY, M.D., Consulting Neurologist to the German Hospital, New York City; to the Infirmary for Women and Children, etc. In two books. Book I., Electrophysics. Book II., Diagnosis, Therapeutics. Illustrated. Philadelphia: P. Blakiston's Son & Co.

IN bringing out the first system bearing upon the therapeutical measures other than the administration of drugs which are not the command of the physician, the editor has coined a most expressive title, "Physiologic Therapeutics," which is to be commended as expressing much in few words. Dr. Cohen has prefaced the series with an interesting "Foreword," written in the philosophical manner so characteristic of its author, and detailing the plan to be pursued in the issuance of the work. We think he has done wisely in "preferring compact books by single writers to bulky tomes of composite authorship." Dr. Johnson said that the books one could hold in one's hand were the best, and there is no doubt that ease of handling renders a book more apt to be consulted. It is a pity that the volumes, however, were not made of lighter material, as they are of considerable weight for the amount of matter they contain.

The first two volumes are by Dr. George W. Jacoby, of New York, and deal with that most interesting and complex subject, electrotherapy. Dr. Jacoby begins with a very lucid and concise exposition of the principles of electrophysics. The reviewer knows from practical experience as a teacher how little the average medical student knows concerning the elemental laws of electricity, and how hard it is to attempt to teach him its therapeutical applications unless he possesses some knowledge previously acquired. Dr. Jacoby's book is, of course, unsuitable for use by the student, but it will prove of the greatest value to the teacher as a guide in his work of instruction. Dr. Jacoby does not fill up his pages with discussions of theories and problems, but gives a description of the various ways of obtaining electricity, the varieties of electrical apparatus, and the physical results produced by the different methods of applying the current.

In the second volume the physiological effects of the electrical current on the different organs and tissues of the body are first considered, followed by a section devoted to the principles of electrodiagnosis and electroprognois in diseases of the motor and sensory apparatus. The rest of the book is devoted to the direct application of our knowledge