

on the difficulty of getting patients in general hospitals to admit that they have mental symptoms, and I am of the opinion that had I been better known to the patients in the Johns Hopkins Hospital whom I questioned as to having fear that I might have found a few more who would admit its presence. At the same time I believe that the majority of those who denied having apprehension were truthful, I was very careful about my questioning, leading up to the subject gradually and putting the question as to having fear in a number of forms so that there could be no doubt of the patient understanding what was meant.

In his text-book, Dr. Osler does not mention apprehension as associated with cardiac lesions, but in a personal letter states that it is "a pretty common symptom in heart disease, being especially common in the neurasthenic forms." It seems to me that this shows the general feeling of the clinician toward these associated conditions, that although it is often present it is a nervous phenomenon which can probably be explained by the personal individuality or peculiarity of the patient. This seems to be the opinion of others, as Gibson² narrates the case of a man who for years had suffered from tachycardia and had associated with this agoraphobia. Gibson considers the persistent tachycardia due to personal peculiarity, and states that in such cases there is usually some associated mental symptoms. In the same lecture he speaks of the mental distress associated with tremor cordis, caused by the sensation "as if the heart stopped and might not recommence its action again." There is a great deal of anxiety attendant on this condition and the patient shows apprehension in his facial expression. A feeble, fluttering action of the heart may be demonstrated both by palpation and by auscultation. It must be remembered that this condition is quite different from palpitation, both in its physical manifestation and its cause, for tremor cordis is probably produced by nervous disturbance but never by psychical excitement, while palpitation is frequently brought on by some emotional disturbance. In palpitation there seems to be no doubt that there is a loss of vagus control. We all know that with palpitation there is often present a feeling of anxiety and associated there may be ringing in the ears, flashes of light, deep, sighing respirations, and sometimes giddiness. The force, as well as the rate, is increased.

Stransky,³ after a study of the anxiety neuroses and their pathogenesis, is of the opinion that there must be a predisposed brain and a pathologic sensation acting in the region of the vagus in order to have apprehension associated with cardiac disease. This seems to me to sum up our present knowledge of these associated conditions, but I feel that it is not exact enough to be satisfying. The symptoms as observed in the colored race would seem to me to point to personal peculiarity, while the almost constant presence of apprehension in the neurasthenic heart cases seems to me to indicate a pathologic condition of the vagus control.

I hope to follow up this investigation more carefully. Many points have suggested themselves about which I am not able to speak at present. There seems to be little doubt that the physical symptoms present in our cases during the attacks of apprehension are similar to those which have been carefully studied in the anx-

iety neuroses, but more investigation is needed in these mild cases of apprehension.

The conclusions which I wish to make are:

1. The cardiac lesion is not the primary factor in causation of the associated state of apprehension.

2. For want of a better term, what we call the idiosyncrasy of the patient is largely responsible for apprehension associated with cardiac lesions.

3. In neurasthenic types a want of vagus control is an important etiologic factor.

4. Our knowledge of this subject is not yet exact enough. On this last point let me urge you to urge on your friends who are engaged more actively in clinical medicine investigation of this condition of apprehension in their heart cases.

SPASTIC DIPLEGIA FOLLOWING PERTUSSIS.*

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PHILADELPHIA.

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History.—The patient, a girl of 30 months, was brought to the clinic for nervous diseases at the St. Agnes Hospital in February, 1903. The mother stated that 8 months previously (May, 1902) the child had contracted pertussis, which lasted for 3 months. At the end of the first month of the disease the patient began gradually to lose the power of walking. Her mother first noticed that she became awkward, stumbled easily, and was very uncertain on her feet. By degrees the inability to walk increased until at the end of 4 months she could not walk unassisted. About this time the mother noticed that the child had nystagmus, but she had noted no other symptom until she brought her to the clinic 4 months later.

Family History.—The family history of the child was excellent. The parents were healthy young people, and there was no history of nervous disease in their families. This was the only child and the mother had never had any miscarriages. The child was delivered normally, the head presenting, after a pregnancy of nine months and one week. No instruments were employed, and there was no injury to the head. The child was reared on the breast and passed through her early infancy up to the age of 21 months without any of the usual digestive disturbances and without contracting any other disease than the one mentioned above. She developed in all ways normally, except that she did not walk until the nineteenth month.

Examination and Course of the Disease.—I saw the patient first in February, 1903, which was 8 months after the onset of her nervous disease. At this time she was fairly well preserved, intelligent, understood everything that was said to her, and could talk plainly. Her head was well formed; her palatal arch not unduly high, and there was an absence of the other physical defects which characterize the mentally deficient. In fact, there seemed to be every indication that the child was normal at birth, and had developed properly until the appearance of the weakness in her legs. The fontanelles had been closed since the end of the first year; the heads of the long bones were slightly enlarged, and some, though not marked, beading of the ribs was observed. When placed on the feet she sought at once for support, and would have fallen without it. Any attempt to walk unassisted was futile and developed a transient rigidity of both legs. When supported on each side by a person, her efforts to walk showed a tendency to cross-leg progression. The legs were slightly spastic at this time, though not always so. Often the test failed to show any stiffness whatever. Again, at the same examination, spasticity was present during a crying spell or on making voluntary effort. There was a tendency to hyperflexion of the feet, and both big toes were hyperextended. At this time the arms were

2. *The Nervous Affections of the Heart*, second series, Edin. Med. Jour., vol. xlii, p. 334.

3. *Monatsschrift für Psychiatrie und Neurologie*, vol. xiv, No. 2, August, 1903.

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-fifth Annual Session, June, 1904.

very little involved. When the patient picked up small objects there was some slight awkwardness of movement only. A constant small, irregular tremor was observed in the hand, and this was not increased by voluntary effort. The mother stated that the child twitched the shoulders and hands a good deal during sleep. There was a constant marked lateral nystagmus, and the pupils were equal and quite widely dilated. The knee jerks were increased and equal. The arm jerks were only slightly increased. Babinski's sign could not be developed, nor was there any clonus, and no sensory disturbance could be elicited. While the child was fretful and cried a good deal, there was no disturbance of any of the functions that could be noted. The appetite was good; the bowels regular; the heart was rapid and the first sound rough, but there was no murmur. The lungs were normal. No spinal deformity or tenderness could be observed. An examination of the eye-grounds by Dr. Shute gave negative findings.

From this time on the child was frequently observed and studied. The power to walk gradually failed, the spasticity increased, and in the arms there slowly developed a spastic condition, while at the same time there was a distinct and progressive loss of power. On March 25, 1903, the patient had, for the first time, a convulsive attack, which was general, preceded by a scream, and lasted only a few seconds. She was unconscious during the attack, though she did not bite her tongue nor lose control over the sphincters. Her condition was not materially changed immediately after the convulsion, but the spasticity and motor weakness increased more rapidly, the mind began to fail, her attention could not be fixed, any effort to grasp objects developed athetosis, and convulsive attacks became frequent. Some difficulty in swallowing was experienced shortly after the onset of the convulsions, and from this time until her death only liquids could be swallowed.

On September 27 an examination gave the following results: The child was greatly emaciated; the expression was anxious and vacant, and the attention could not be directed to the fingers moving before the eyes. The head was retracted and moved constantly from side to side; the eyeball rolled; the face was contorted as if in pain, and the pupils were contracted. The arms were held in a half-flexed position, the hands flexed at the wrist, the fingers hyperextended at the proximal joint and flexed at the other two joints, the thumb being held in the palm of the hand. The arms were twitching constantly, and at the same time moved slowly to and fro. The legs were spastic and crossed at the knees, while the big toe was hyperextended and the other toes flexed. The knee jerks were increased, but no clonus could be elicited.

The patient died on September 29, having had for a few days previous many convulsive attacks, general in character, but worse on the right side.

Autopsy.—At the autopsy only the brain and spinal cord were examined, and no gross lesions were discovered. The specimens were placed in formalin, and sections from various parts of the nervous system were stained by the Marchi and Weigert methods, and by hemalum and theonin.

Brain.—Throughout the cortex from the right and left frontal regions there were seen numerous small hemorrhages. These were especially clearly seen by the Weigert hematoxylin method. The pia was not abnormal.

In the right and left occipital regions the pia was thickened; there was an intense red-blood cell infiltration, and a moderate degree of round-cell infiltration. Numerous small hemorrhages were also observed throughout the cortex in these regions. The perivascular spaces were distended, and around the vessels there was an accumulation of mononuclear round cells. In one field a large vessel was seen, the walls of which were thickened and the lumen filled with blood.

In the paracentral regions the change was especially interesting and unique. The pia was slightly thickened; there was a moderate degree of round-cell infiltration; and the vessels were filled with blood cells.

In the cortex there were a few small hemorrhages, and there were also numerous areas where the perivascular spaces were distended. Around the blood vessels within these spaces was

a large number of round mononuclear cells, partly filling the spaces, in some instances quite filling them (Fig. 1). In some fields the accumulation of cells amounted to three times the diameter of the vessel. One perivascular space seen was filled with fibrous tissue containing numerous nuclei. Surrounding these spaces the neuroglia was deeply stained and seemed to be amorphous. Two obliterated vessels were seen in another field.

Everywhere in the cortex, but especially around these distended perivascular spaces, were found large numbers of large round mononuclear cells, the protoplasm of which was granular. These cells were probably granular cells. The distinction between the nerve cells and these was closely brought out by the theonin stain. In sections stained by the Marchi method the cells in the perivascular spaces took the stain intensely; those in the tissue around the vessels were distinctly, though less intensely, stained. In one field a vessel was seen containing what appeared to be separated epithelial lining (Fig. 2). Cells of Betz were present, but they were not numerous and stained diffusely. By the Marchi method marked degeneration was observed in one of the peduncles, and this was most intense in the center of the foot. The anterior pyramids stained but faintly by the Weigert method. The fibers of the internal capsule were very poorly medullated, and did not take the stain at all.

In the optic thalamus there were seen two small vessels in a space surrounded by cells which contained spindle and round-celled nuclei. In the brain tissue, directly posterior to the chiasm, numerous small hemorrhages were observed. The right optic nerve did not take the Weigert stain very deeply; in fact, there appeared a partial degeneration. It, however, was difficult to decide whether it was degeneration or the result of the formalin in which it was hardened.

Spinal Cord.—In the spinal cord from the upper thoracic region, which was the highest level studied, to the lower lumbar levels, the crossed pyramidal tracts stained but faintly. In the upper thoracic region the pyramidal tracts also took the stain poorly. The Marchi method gave negative results in the medulla and lumbar region. The anterior cells did not appear to be abnormal, and the spinal roots were intact. The neuroglia was increased in the affected areas, and the axis cylinder had disappeared.

RESUME.

A child of 21 months developed, during an attack of pertussis, a spastic diplegia, with, finally, imbecility and general convulsions, and died at the end of 17 months. A study of the brain and spinal cord revealed the presence of numerous small microscopic hemorrhages, distension of the perivascular spaces with cellular accumulation around the vessels and the presence of large mononuclear cells surrounding these spaces and elsewhere in the cortex of the paracentral region. The pia was in places thickened and the seat of a cellular infiltration. The motor tracts were diseased from the internal capsule to the lumbar cord, staining either poorly or not at all by the Weigert stain. Acute degeneration was observed in the cortex and one of the peduncles, as shown by the Marchi method. The lesions were those of hemorrhagic encephalo-meningitis.

The change in the nervous system which I have just described is indicative of some inflammatory process. This change is not peculiar to whooping cough, however, and would indicate that some toxic process had been at work on the blood vessels and their surroundings. I have not been able to trace any degeneration of the fibers in the cortex, nor to discover any foci of sclerosis. The blood vessels in places showed distinct thickening, some were obliterated, and many others were surrounded by large mononuclear round cells. The perivascular spaces were very much distended and filled with these round cells, while the neuroglia immediately around these distended perivascular spaces was crowded with the same sort of cells as were seen in the perivascular spaces themselves. These spaces were in some places so very large that they were visible to the naked

eye. The degeneration in the anterior pyramids was probably due to the destruction of the motor fibers here and there in the cortex. In Jarke's¹ case there was, microscopically, evidence of inflammatory change. The tissues were crowded with leucocytes; there was perivascular cellular infiltration; and the vessel walls were thickened and infiltrated. In this case, however, there is no mention of any degeneration of the motor tracts. His case was one of hemiplegia, moreover, and, while the microscopic changes in some respects were similar to those which I have just described as occurring in my own case, clinically the cases differ very much.

Neurath,² in a recent communication in which he makes a preliminary report of the study of the post-mortem records in a number of cases of pertussis, mentions the fact that he has found the pia distended by numerous foci of cells in the cortex, distension of the pericellular and perivascular lymph spaces, hemorrhages, severe hyperemia, and round-cell accumulation. These were the only references to pathologic findings which resembled those found in my case.

Many different nervous manifestations are described in the literature as occurring during the course of pertussis, and if we classify these for the purpose of study we find that there are cases: 1, exhibiting psychoses; 2, showing disorders of the cranial nerves; 3, with convulsive disturbances; 4, with cerebral hemorrhages; 5, with other lesions of the brain and spinal cord, under which heading my case properly comes; 6, with involvement of the peripheral nerves; and, finally, 7, cases which suffer from neuroses.

PSYCHOSES.

Meschede³ reported a case occurring in a child of 5¾ who, during an attack of whooping cough, developed hallucinations of sight, hearing and touch. Moebius⁴ described a case of melancholia occurring during an attack of pertussis and quotes Emminghaus, who recognized the occurrence of psychoses during the course of this disease. Ferber,⁵ Neurath,² Möller⁶ and others have described similar cases. In my own case imbecility was a late symptom.

DISORDERS OF THE CRANIAL NERVES.

The special senses have been affected in a few cases reported. Meissner,⁷ as early as 1844, recognized that blindness was a complication of pertussis. In Knapp's⁸ case there was total blindness, due, he believed, to ischemia of the retina. Alexander⁹ reported two cases, one in a child of 3 whose pupils were dilated and did not respond to light or in accommodation, and in whom there were bilateral neuritis and vomiting; the other, a child of 12, in whom, after headache, there was sudden loss of vision. The pupils were moderately large and reacted to light and in accommodation, but there was no abnormality of the media or eyegrounds. The former patient died without recovery of vision, and the second patient had improved at the time of reporting. Alexander believed that there was some intracranial process in these cases. There was no uremic condition in either.

In Duga's¹⁰ case of sudden complete blindness the

cause was a hemorrhage into the chambers of the eye. While there was no involvement of the nerves in this case it is deemed worthy of mention as a possible source of confusion in diagnosis.

Transient blindness may occur after scarlatina and typhoid fever, as well as pertussis. Jacoby¹¹ called attention to this fact, and claims that it is a uremic symptom. He states that it was first described by Ebert in 1868. In two cases cited by Sachs¹² there was transient blindness, but no uremic or nephritic symptoms. Steffen¹³ reported the case of a girl of 8 who, during an attack of pertussis, saw indistinctly, and refers to a case of blindness reported by Sebergondi.¹⁴

In Silex's¹⁵ case, a girl of 1¾, after convalescence from accidental poisoning by morphin, was found paralyzed on the left side, and to have divergent strabismus of the left eye. On the fourteenth day hemiopia was discovered, but the patient made a complete recovery 12 days later. Silex believed that the symptoms were due to hemorrhage into the posterior part of the internal capsule, while the hemiopia was probably the result of pressure on the optic tracts. Troitzky¹⁶ described a case of psychic blindness (*seelenblindheit*).

Hockenjos¹⁷ cites other cases of blindness occurring during pertussis, reported by Chotowitzky, Rau and Steffen. (I was unable to obtain the original references to these cases.)

In a few cases deafness has complicated pertussis. Falls¹⁸ reported 4 cases of deafness occurring during the course of whooping cough, only 3 of which were of nervous origin. Similar cases have been reported by Wilde and Friquet (quoted by Hockenjos¹⁷). Gibb¹⁹ reported the case of a boy of 4½ who bled from the ears during whooping cough, resulting finally in deaf-mutism. In 2,000 cases Gibb saw 4 in which bleeding from the ears took place, due to injury to the tympanic membrane. He quotes also the case of Clark.²⁰

A case of paralysis of the posterior crico-arytenoid muscles was reported by Jurasz,²¹ and in Craig's²² case there was paralysis of the sixth and seventh pairs of nerves, due, he believed, to hemorrhage into the pons.

CONVULSIVE DISTURBANCES.

The most frequent complication of pertussis is convulsions, according to Hochenbach²³ and May.²⁴ These may take the form of mere twitchings in the region of the facial nerve, spasm of the glottis, or general convulsive seizures. In a small percentage of cases the convulsions are due to eclampsia. Lenhartz²⁵ found in 75 cases of pertussis 4 cases of eclampsia, of which 3 were fatal. Spasm of the glottis was present in 2 cases. In 29 cases collected by Rilliet and Barthez²⁶ convulsions occurred 5 times, and during the second stage.

CEREBRAL HEMORRHAGE.

The literature of the nervous complications of per-

1. Jarke: *Archiv f. Kinderheilk.*, 1890, No. 20.
2. Neurath: *Wiener klin. Woch.*, 1904, No. 46, p. 1267; *ibid.*, 1896, No. 23, p. 488.
3. Meschede: *Allg. Zeitschr. f. Psychiatrie*, 30.
4. Moebius: *Centralbl. f. Nervenheilk.*, 1887, pp. 641 and 129.
5. Ferber: *Jahrbuch f. Kinderheilk.*, 1870, p. 229.
6. Möller: *Archiv f. Psychiatrie u. Nervenkrank.*, 13.
7. Meissner: *Gerhard's Handbuch*, vol. ii, p. 568.
8. Knapp: Quoted by Alexander.
9. Alexander: *Deut. med. Woch.*, 1888, No. 11.
10. Duga: Quoted by Hockenjos.

11. Jacoby: *New York Med. Monatsschrift*, February, 1891, p. 45.
12. Sachs: *Sammlung's klin. Vortrag. v. innere Med.*, 1-29, 1890-1894, p. 445.
13. Steffen: *Ziemssen's Handbuch*, 1896, vol. iv, No. 1, p. 285.
14. Sebergondi: Quoted by Schreifer.
15. Silex: *Berliner klin. Woch.*, 1888, p. 841.
16. Troitzky: *Jahrbuch f. Kinderheilk.*, 1890, No. 31, p. 291.
17. Hockenjos: *Jahrbuch f. Kinderheilk.*, 1900, No. 51, p. 4.
18. Falls: *Zeit. f. Ohrenheilkunde*, 1886, p. 303.
19. Gibb: *Brit. Med. Jour.*, 1861, p. 434.
20. Clark: Quoted by Gibb.
21. Jurasz: *Jahrbuch f. Kinderheilk.*, No. 14.
22. Craig: *Brit. Med. Jour.*, 1896, p. 1440.
23. Hochenbach: *Gerhard's Handbuch*, vol. ii, p. 568.
24. May: *Arch. f. Kinderheilk.*, 1900, p. 127.
25. Lenhartz: *Elstein's Handbuch*, vol. i, p. 223.
26. Rilliet et Barthez: *Traite des Malad. des Enfants*, 1891, vol. v, No. 3, p. 712.

tussis is largely taken up with reports of this condition. There have not been, however, a great many cases reported with autopsy. This is partly due to the fact that the prognosis in these cases is good. In 38 cases collected by Schreiber²⁷ up to 1899 only 10 were verified by autopsy. His deductions, I believe, will not hold good at the present time. From a study of these 38 cases he concluded that in most instances the hemorrhage is into the meninges, but may occur in the substance of the brain, either as capillary hemorrhages or in the form of hemorrhagic encephalitis. Cerebral hemorrhage occurred most frequently in children from 2 to 4 years of age; the youngest being 10 months and the oldest 10 years. The hemorrhage occurred usually during a severe paroxysm. He attributes the occurrence of cerebral hemorrhage in pertussis to increase in the blood pressure and to venous engorgement. Many cases of a similar character have been described by Bernhart,²⁸ Sachs,¹² MacKerron,²⁹ Neurath² and Jarke.¹ Hochenbach²³ collected 23 cases in which a diagnosis of brain hemorrhage was made with 9 autopsies. In 5 cases the hemorrhage was into the white matter, in 2 into the cortex, and in 2 other cases between the dura and the pons.

Hockenjos¹⁷ collected 41 cases of cerebral hemorrhage, and reported one case of his own. In only 11 of these was there an autopsy. Hemorrhage into the meninges was observed in 4 cases; into the cortex in one case; into the white substance in 5 cases; and undetermined in 1 case. He concludes that hemorrhage is most frequent under 6 years of age, and that the meninges is the site of preference for the hemorrhage.

Paul Valentine³⁰ collected from the literature 83 cases of nervous disturbances occurring during pertussis. Of these 16 were examples of monoplegia, 40 were hemiplegia, while only 5 or 6 cases were instances of paraplegia. He believes that the prognosis of paralysis in pertussis is very grave. Of 64 cases there were 14 who died, 22 who remained paralyzed, while the rest recovered almost completely.

Cases have been reported which came to autopsy without any—or only insignificant—pathologic change. For instance, Blache's³¹ case only showed slight engorgement of the brain and spinal meninges. He refers specially to the case of West³² and Wiesinger, in which the autopsy showed no pathologic change of the brain. Henoch³³ found some hyperemia of the brain in a child who had suffered from symptoms of meningitis. This was also true of a case of Jacobson's,³⁴ in which the child exhibited a left hemiplegia.

I have been able to collect in all only 20 cases in which an autopsy has been made. In Marshall's³⁵ case there was hemorrhage into the substance of the brain. The hemorrhage in Reimer's case was between the dura and the skull, as was also that of Cazin.³⁶ In the case of Barnier³⁷ (quoted by Barthez and Rilliet²⁶) the hemorrhage was into the meninges. In Hauner's³⁷ case a large vessel in the brain was ruptured, and there was also hemorrhage into the arachnoid. Dauchez³⁸ re-

ported a case of cerebral hemorrhage in a tubercular child. At the autopsy there was found a hemorrhagic area over the cortical surface of the left hemisphere, occupying the fissure of Rolando and the two frontal and parietal convolutions. In Stiebel's³⁹ case there was a large exudate in the arachnoid of the anterior lobes, and the gyri were filled with a grayish yellow substance. On section of the hemispheres hemorrhagic points were visible, and the ventricles were enlarged. External to the right ventricle there was extravasation of blood and some red softening around it.

Furbinger⁴⁰ describes a case in which there was capillary hemorrhage of the brain and cord, and also of the white substance. Strümpell⁴¹ (quoted by Hockenjos¹⁷) reported a case in which there was capillary hemorrhage into the brain.

In Vidal's⁴² case there were small hemorrhages of the white substance of the brain which were confluent in the occipital lobe.

Hockenjos¹⁷ reported a case in which hemorrhage of the floor of the left ventricle and punctiform hemorrhage of the pons were observed.

Jarke¹ described a case of hemiplegia in which the autopsy revealed the presence of inflammatory foci thickening and infiltration of the vessel walls, the presence of two small foci of softening in the frontal and parietal convolutions, symmetrical in character, a perivascular infiltration, and a diapedesis of white blood corpuscles.

In Thomas's⁴³ case there was hemorrhage into the anterior portion of the medulla and overfilling of the vessels of the meninges.

In Blache's³¹ case there was only slight engorgement of the meninges, brain and spinal cord.

West³² reported a case of convulsions in which there was no change visible postmortem.

In Henoch's³³ case, which was clinically one of meningitis, there was found postmortem only hyperemia of the brain.

In Guibert's⁴⁴ case (cited by Schreiber²⁷) there was no pathologic change observed. In a case reported by Cornelius May,²⁴ in which there were clonic convulsions, only edema and hyperemia of the brain were observed postmortem.

In Jacobson's³⁴ case of hemiplegia no pathologic change was found in the nervous system.

Simoninis⁴⁵ reported the case of a girl of 6 who exhibited bulbar symptoms, and the section showed hemorrhage into the brain.

Luce⁴⁶ cited a case in a boy of 5 who developed left hemiplegia. The autopsy showed the presence of hyperemia, and in the medulla some recent degeneration, as shown by the Marchi method. There was no perivascular hemorrhage, and no change in the blood vessel walls could be demonstrated.

Of these 21 cases—	Cases.
Hemorrhage occurred into the brain in.....	7
Hemorrhage occurred between dura and skull in.....	2
Hemorrhage occurred into the meninges in.....	1
Hemorrhage occurred into the substance of the brain and in meninges in	2
Capillary hemorrhage occurred in.....	2
Hyperemia occurred in	4
No pathologic change was found in.....	3

27. Schreiber: *Archiv f. Kinderheilk.*, 1899, p. 1.

28. Bernhart: *Deut. med. Woch.*, 1896, No. 20.

29. MacKerron: *British Med. Jour.*, 1897, No. 21.

30. Valentine: *Abstr. of Thesis in Lancet*, 1902.

31. Blache: *Schmidt's Jahrbuch*, vol. i, p. 307.

32. West: *British Med. Jour.*, 1889, No. 1, p. 157.

33. Henoch: *Vorlesung. Kinderkrank.*, 1903.

34. Jacobson: *Deut. Zeitschr. f. Nervenheilk.*, 1893, No. 4, p. 256.

35. Marshall: *Virchow-Hirsch*, vol. ii.

36. Bledert: *Lehrbuch f. Kinderheilk.*

37. Barnier: Quoted by Barthez and Rilliet.

38. Dauchez: *Prog. Méd.*, 1884, No. 12, p. 548.

39. Stiebel: *Journal f. Kinderheilk.*, 1856, No. 26.

40. Furbinger: Quoted by Schreiber.

41. Strümpell: Quoted by Hockenjos.

42. Vidal: *Prog. Méd.*, 1885, No. 2, p. 270.

43. Thomas: *Schmidt's Jahrbuch*, 1835, p. 139.

44. Guibert: Quoted by Schreiber.

45. Simoninis: *Schmidt's Jahrbuch*, v. 273, 1902, p. 51.

46. Luce: *Deut. Zeitschr. f. Nervenheilk.*, 1898, p. 272.

These statistics do not agree with the statements made by Schreiber, who, in 10 autopsies, found that the hemorrhage occurred in most cases into the meninges. In Hagenbach's²³ statistics, based on 23 cases collected from the literature with 9 autopsies, he found in 5 of these 9 cases which came to autopsy that the hemorrhage was into the white substance, thus agreeing with the conclusions to which I have come in my statistics.

OTHER LESIONS OF THE BRAIN AND SPINAL CORD.

Pertussis is a known cause of encephalitis, though probably not a frequent one. Nothnagel⁴⁷ states briefly that encephalitis may occur after whooping cough, as well as after the infectious diseases, and believes that

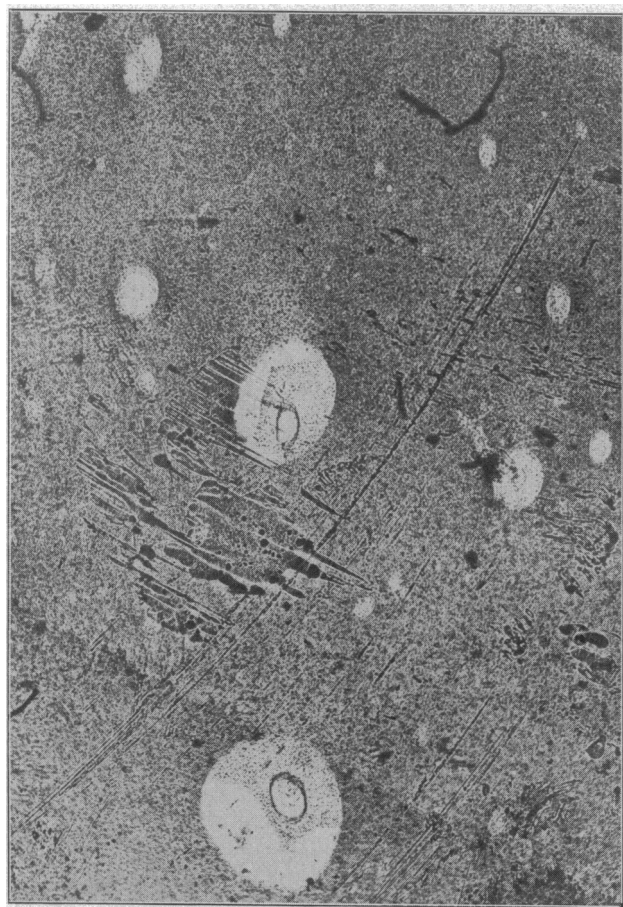


Fig. 1.—Cortex, showing distended perivascular spaces, round cell accumulations and obliterated vessels.

cases like those of Henoch,³³ Fritzsche and Troitzky¹⁶ belong to this class. Richardiere⁴⁸ (quoted by Marie) reported one case of "sclerosis encephalique."

Cases of spastic diplegia like that reported in this paper are rare in the literature; in fact, we can find but one—that reported by Sachs, which occurred in a child of 13 months who developed a gradual paralysis of both sides of the body, athetosis, increased reflexes, a high grade of imbecility, and defective speech. Unfortunately in this case there was no autopsy. Sachs,¹² who has studied the question of cerebral hemorrhage in children very carefully, states that whooping cough and pneumonia may frequently be causal factors. Out of 105 autopsies he found that in 23 cases of diplegia the lesions in 19 cases were atrophy, sclerosis and cysts, and

in 4 cases there was present a porencephalon. In most cases of lobar sclerosis he states that the primary lesion completely disappears, and it is impossible to discover where the sclerosis took its origin.

Bernhardt²⁸ reported an interesting case in a child of 5 who, after a severe paroxysm of coughing, suddenly became stiff in the lower extremities, so that standing and walking were quite impossible. There was no loss of consciousness, no convulsions, and no involvement of the arms. The plantar reflexes were increased, and the feet were held in a position of plantar flexion. There was no foot clonus nor muscular atrophy. Some slight disturbance of sensation was observed, and at the onset there was some involvement of the bladder functions. All the symptoms finally disappeared and the patient recovered. The author expresses the opinion that the condition was due to hemorrhage into the spinal cord.

A case of Michell's⁴⁹ deserves a passing mention here. In his case there was a transient monoplegia of the left arm. The patient recovered entirely in 8 days.

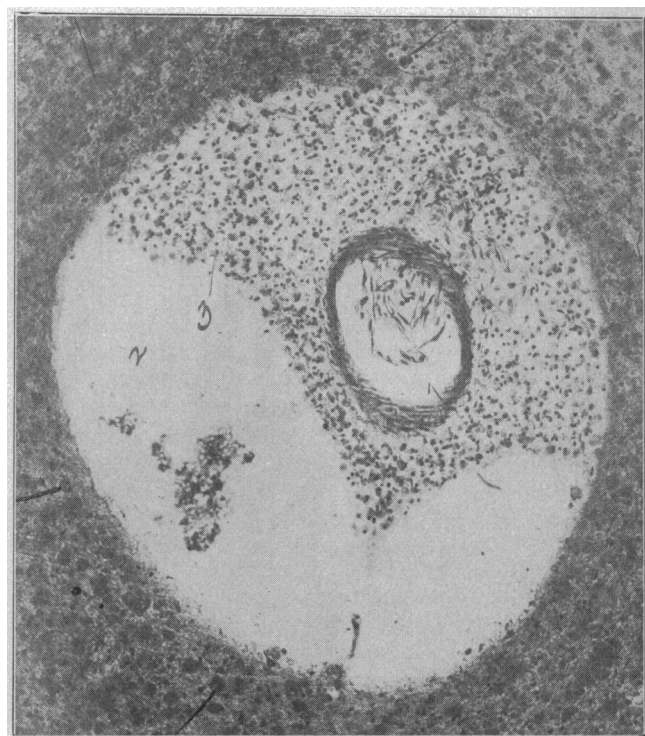


Fig. 2.—1. Lumen of a vessel. 2. Perivascular space, greatly distended. 3. Round cells. Around the spaces are seen numerous granular cells. (Marchi method.)

Luisade⁵⁰ described a case of paraplegia in a girl of 5 who finally recovered. In Hagedorn's⁵¹ case there was first paraplegia, ptosis and spasm of the facial muscles, finally all the limbs were paralyzed and the patient died. There was no autopsy. Mackey⁵² cites a case of Landry's paralysis with recovery. Hydrocephalus has followed pertussis in one case reported by Lombard⁵³ (Hochenbach).

PERIPHERAL NERVES.

We can find only one case that belongs under this heading, that reported by Moebius,⁴ of a child of 3 who developed, first, paralysis of the legs, then of the arms

47. Nothnagel: *Specielle Path. et Therap.*, vol. ix, 1901.

48. Richardiere: Quoted by Marie.

49. Michell: *Thèse de Paris*, 1891.

50. Luisade: *Schmidt's Jahrbuch*, 1899, No. 261, p. 232.

51. Hagedorn: *Schmidt's Jahrbuch*, 1903, No. 273, p. 51.

52. Mackey: *Brit. Med. Jour.*, Aug. 26, 1884.

53. Lombard: Quoted by Hochenbach.

and neck muscles, and finally of the diaphragm. He believed that the case was a toxic action analogous to that which produces paralysis in diphtheria and scarlet fever. The child recovered completely. This is the only case reported that pointed to involvement of the peripheral nerves.

NEUROSES.

Epilepsy has followed pertussis, and Theodor⁵⁴ and Steffen¹³ state that chorea may also be a complication. Steffen believes that in the latter case it is probably the result of some focal brain lesion.

Several theories have been advanced to explain the occurrence of paralysis. Kassirer,⁵⁵ Marie⁵⁶ and Moebius⁴ have claimed that the paralysis is due to infection. Moebius believed that it is the result of a toxin analogous to that of diphtheria. Neurath,² and particularly the French authors also, defend the toxic theory. Furbinger⁴⁰ believes that the blood vessel walls become altered by the toxin and are thus more susceptible to the increase in blood pressure.

Biedert³⁶ and Hagenbach look on whooping cough not as a general infection like scarlet fever and measles, but believe that it is a local infection of the bronchi in which the cramp-like attacks of coughing may be attributed to the irritation of the superior laryngeal nerve.

In a case reported by Henoch,³³ in which there were convulsions and strabismus, but in which the autopsy revealed only hyperemia of the brain and meninges as well as hemorrhages, he believed the hemorrhages were due to carbon dioxide intoxication.

Luce⁵⁷ believed that in acute attacks of hemiplegia occurring during the course of pertussis, either a meningeal hemorrhage is found or there is no anatomic basis for it. He claimed that it was not proven that an isolated hemorrhage into the internal capsule or elsewhere in the pyramidal tracts could occur as the result of increased blood pressure. He looks on the occurrence of hemiplegia in these cases as due to CO₂ intoxication.

I believe that in my case the wide-spread lesion in the cortex was the result of a toxin acting on the vessels and setting up an inflammatory process. This process resulted in the destruction of fibers here and there in the cortex, especially in the paracentral region, in consequence of which there followed a degeneration of the pyramidal tracts. The small hemorrhages in the cortex were probably due to the convulsive attacks which preceded death.

To Dr. W. G. Spiller is due my sincere appreciation for his assistance in the study of the sections.

DISCUSSION.

DR. T. H. WEISENBURG, Philadelphia, reported the case of a canary bird which was the inmate of a house where three children were sick with whooping cough. The bird had been sick for about five weeks; previously he had been perfectly well. He had spells of coughing and these were more frequent at night and would keep him from sleeping. He had attacks of panting, associated with coughing and choking. He died of diarrhea after glycerin had been placed in his drinking water. The brain was removed and sections stained by the hemalum-eosin stain showed the blood vessels to be very much congested; these stood out prominently and diapedesis of the red blood cells could be easily seen. This is an interesting finding in conjunction with Dr. Rhein's case where hemorrhages were marked.

DR. R. C. MOORE, Omaha, Neb., stated that in severe nervous troubles, following an acute disease, the question arises whether the nervous disorder is due to anything mechanically connected with the disease or whether it is due to the toxins. Of course, the sequelæ of scarlet fever are of toxic origin, but in this case the cerebral hemorrhage may possibly have been caused by the mechanical action due to the severity of the cough. In Dr. Moore's practice several years ago a little child about a year and a half old, during a severe attack of whooping cough, became totally blind. An ophthalmoscopic examination showed no disease of the eyeball, retina or anything connected with them. Therefore, the lesion must have been central. There was no spasm and the child developed no other severe symptoms. The lungs were normal and the child finally recovered. The probability is that in this child's brain there was a condition of those minute hemorrhages as described by Dr. Rhein, but the condition was not sufficiently severe to cause a fatal termination; the natural recuperative powers removed the results of the disease. Dr. Moore considered at that time that the child's condition was probably due to the severity of the cough, and that the products of the micro-organism of the disease had nothing whatever to do with it. Very likely in Dr. Rhein's case the slight hemorrhages were due to the same cause.

DR. JOHN H. W. RHEIN said that it would be interesting, if possible, to study the effect of whooping cough on the nervous system of the lower animals. After a brief review of the literature he has been unable to discover any data on this subject.

ACUTE INTESTINAL SURGERY, WITH REMARKS ON TECHNIC.*

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There is a class of abdominal cases which may justly be considered acute. Acute for the reason that the indications for immediate surgery are mandatory and in which the mortality largely depends on the time of operation and the technic of the work. Under this heading I wish to discuss:

1. Penetrating gunshot and stab wounds of the abdomen.
2. Severe abdominal contusions associated with rupture of the intestines or with other visceral injuries.
3. Strangulated hernia.

I shall confine myself as closely as possible to a consideration of the operative technic.

PENETRATING GUNSHOT AND STAB WOUNDS OF THE ABDOMEN.

At the last meeting of this Association I presented in detail a series of cases of gunshot and stab wounds of the abdomen on which I had operated at the St. Louis City Hospital. In that paper I endeavored to show the impossibility of determining the damage done to abdominal viscera by a bullet or knife wound penetrating in character by any known method save exploratory section. As my experience grows I am convinced that the conclusions arrived at in that paper were correct. Assuming that we accept the statement that all penetrating wounds should be explored, as is agreed by the majority of surgeons who have had any actual operative experience in this work, the technic of the work is well worthy of consideration and should be fully discussed.

The operation itself may be divided into: (a) Examination of wounds; (b) incision; (c) control of hemorrhage; (d) search for visceral injuries; (e)

54. Theodore: Arch. f. Kinderheilk., 1890, p. 219.

55. Kassirer: Centralbl. f. Kinderheilk., 1896, p. 37.

56. Marie: Progrès Méd., 1885, p. 167.

57. Luce: Deutsch. Zeitschr. f. Nervenheilk., 1898, p. 272.

* Read before the Mississippi Valley Medical Association, 1904, at Cincinnati, Ohio.