

adductor reflex on the other side. A result which might have been anticipated if the foregoing conclusion is correct. This dissociation of the homolateral from the contralateral reflex was present in another case which clinically resembled the one reported in this paper, except that the symptoms were descending. I have not found this dissociation present in cases of hemiplegia, syringomyelia, amyotrophic lateral sclerosis nor transverse myelitis.

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SYMPTOM-COMPLEX OF A SERIES OF INTES- TINAL CASES WITH PATHOLOGIC FINDINGS.*

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During the years from 1903 to 1906 a number of cases came under observation which till that time had not been encountered and a description of which has not been found in the text-books, nor in current literature so far as I have investigated.

The first two cases seen by me occurred in the practice of Dr. Morris of Birmingham, and so unusual were the symptoms, so prompt the fatal ending, that poisoning was suspected, though no known poison would seem to explain all the symptoms. Other cases occurring soon after, we were forced to the conclusion that we had encountered a symptom-complex new in our experience. Conversation with our older practitioners shed no light, as they had seen or had recognized no such cases.

Eighteen cases have come under my personal charge, or in consultation. Of these twelve patients died, six recovered. The youngest was 6 months old, the oldest 20 months. All were, or had been, breast raised. Fourteen were nursing their mothers at the time of illness. All but two were vigorous children, previously healthy.

The symptoms that marked the cases were rapid abdominal breathing (air hunger), fast pulse, cessation of bowel movement, enlargement of liver and lowered temperature, restlessness and expiring moan.

As a rule, the attack was preceded by mucous or bloody mucous action with slight temperature for a few days, then the supervention of the typical type of breathing. From the time of inception of symptoms there has been no question in the minds of parents, nurse or doctor of seriousness of condition. The restlessness, the expiring moan, the labored breathing marked at once a seriously ill child.

Tympany has been marked in some of the cases. Vomiting has existed in a good proportion. In only one case of the series has there been any loose bowel action without purgatives. The temperature has become subnormal at some time after the attack began in most of the cases. In some it has risen high at some period.

The two things that have been found in every case, save one mild one that did not show enlargement, have been the type of breathing and the enlargement of the liver. The rapidity of enlargement has been a phenomenon that has excited interest. In twelve hours a liver not palpable below the ribs will be distinctly felt and, in forty hours, in some cases be two inches below. This has been demonstrated to physicians in a number of instances—a liver shown not to be enlarged at

first visit and next day demonstrated to be an inch and more below the rib.

The urine has been normal in quantity in all the cases, save the V. baby, in which vomiting was so persistent as to preclude absorption of water. Albumin in small quantity was found, in two cases, in urine taken from bladder postmortem. In the other cases it was not obtained. Jaundice was absent in all the cases.

A leucocyte count of 25,000 was made in one case. Of the patients that have recovered, every one, with the exception of Baby B., whose liver did not appreciably enlarge, has shown prolonged convalescence, loose bowels and a very tardy restoration of the digestive functions.

The cases have occurred in various sections of our city and in the suburbs and in families which provide their children hygienic surroundings. Of the series eight have been brought to postmortem.

The postmortem findings of organs, other than liver and mucosa of intestine, have been negligible. The intestinal mucosa in some of the examined cases showed hyperemia. In others very little or no appreciable hyperemia. The liver has been enlarged to greater or less extent in every one and sections from all examined show fatty degeneration. In some of the specimens there are fields in which no normal cells can be seen and very few nuclei. In others there are areas of fatty degeneration in the fields. In every one the fatty degeneration exists. The gall bladder in every case contained a dark green, ropy bile. This same dark green material passed in the actions when the bowels were moved by purgatives during the course of the typical symptoms.

Unsuccessful attempts have been made to work up the bacteriology, due to the unpreparedness of our local bacteriologists, and inoculated tubes from the intestines and different organs have been sent to eastern laboratories without result.

What the etiologic factor is I am unable to state. It would seem, but only seem, reasonable to conclude that a toxin is generated in the intestinal tract which produces the fatty degeneration of the liver. How the air hunger is produced, whether directly by the toxins breaking down the natural barrier afforded by the healthy liver and thus entering the circulation, or whether indirectly by secondary products, is a matter for speculation.

If it be the Shiga bacillus that generates the toxin, as might be suggested from the mucous or bloody mucous actions commonly preceding the onset of symptoms, then the natural inquiry arises, why does not the Shiga bacillus generally cause these cases, for we are constantly encountering the severest types of ileocolitis in our spring and summer seasons.

I feel certain that such cases have not been confined to our locality, but that they have been occurring elsewhere in the south. From a report that has reached me I have every reason to conclude that a baby in another southern city died with the identical type of symptoms. It is with a view to calling attention to these cases that now, after observing them for four seasons, I have brought them to the attention of this section.

The treatment pursued has been eliminative. Hypodermoclysis has been used in some of the cases. Camphor has been given by mouth, where the stomach was not revolting too much. Alcohol has not been given, as it seems the liver already had burdens enough to carry. Food has been withdrawn and the patients recovering have been kept on water till the symptoms seemed to warrant a return to food.

* Read in the Section on Diseases of Children of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

The impression gained from the series has been that when the dose of poison has been sufficiently large medication is useless. In the cases where the dosage has not been lethal, elimination and symptomatic medication only have been employed. The effort has been rather to give the natural recuperative forces of the child fair play in the fight with the enemy. In this type, as in the severe cases of ileocolitis usually met with, the conclusion has been forced on me that, as in typhoid, the problem is to keep the patient alive till the natural forces can get the better of the disease.

All the patients with the exception of one, Baby P., in 1906, have gone on to recovery after the type of breathing has ceased. In this case recovery was predicted on cessation of this symptom, but it reappeared after some twenty-four hours and the child was dead in some thirty hours more.

It is known to you that, in cases of ileocolitis coming to postmortem after a protracted course, a fatty degeneration of the liver is frequently encountered.

The monograph on "Diarrheal Diseases of Infancy," issued in 1904 by the Rockefeller Institute, speaks of fatty degeneration of the liver occurring in dysenteric cases in these words: "The liver is fatty in about the same percentage as in other infantile conditions" (Howland); but none of the many histories given correspond with the cases above cited. None of these show the symptom-complex nor the rapid enlargement of the liver.

Unsupported, I would not care to subject myself to criticism in reporting this series of observations however certain I might be of my own opinion, but it so happens that cases have been seen by and with a number of our local physicians. A report to our local society was made in 1903 giving symptoms-complex and pathologic findings, and as a result of that report I have been called into consultation by our physicians who recognize the description. Moreover, three of these fatal cases have occurred in the children of as many of our physicians.

Baby G. M. W., aged 6 months. Plump and strong up to thirty-six hours preceding time of consultation, when an ileocolitis, apparently of mild grade, had developed. Temperature was 100 and there had only been a few actions, showing, however, bloody mucus. The child seemed very slightly ill.

After retiring to consult, Dr. Morris, who was in charge, stated that another child had died since I was called to see the first in May and that it began to be ill just as had this one and the first.

On the day following, June 10, this child developed labored, rapid abdominal breathing, the bowel actions ceased, the temperature became subnormal and the child was dead in 48 hours after the labored breathing set in. The abdomen became very tympanitic before death and temperature a few hours preceding rose to 104.

A postmortem was obtained and done in a couple of hours. Other than an enlarged liver and a slightly hyperemic intestinal mucosa nothing was found to throw light on the cases.

Here were three patients dying in one house, all healthy, strong, breast-fed babies and all of healthy young parents, with symptoms unrecognized by any of the three physicians in attendance.

On July 3 I was called to see L. P., male, white, aged 20 months. The symptoms were constant vomiting and a profuse watery diarrhea containing undigested vegetables. The father was a very strong man, who stated that he believed in raising children to eat everything. There was no elevation of temperature, the pulse was strong and by withdrawing all food and giving water *ad libitum* the child, by evening seemed to be relieved. Next day he was sitting up playing in bed when seen, seeming to be relieved of his attack. His mother was

instructed to withhold food till the following morning as a precautionary measure. That night—the 4th—labored breathing developed, but I was not called till the following morning. At 9 a. m. respirations 44, labored abdominal in character, temperature 99.5, abdomen distended. Child very restless. The liver was enlarged to an inch below the border of ribs. The bowels had not moved for 36 hours. Unconsciousness came on during the day and after ten hours of extreme restlessness child died the following morning at 7 o'clock. Postmortem one hour after death. Heart and lungs normal. Kidneys somewhat congested, with an infarct in one kidney, mucous membranes of intestinal tract slightly hyperemic along whole course, but no lesion present. A dark green substance was found in the small intestine, the source of which was ascertained by opening the distended gall bladder, for here was the same material—dark, thick, green bile. The liver was enlarged, firm, with yellowish streaks across surface.

Baby R., male, white, aged 11 months, breast fed, robust child, son of a physician. On July 29, 1903, bowels slightly disordered, for which mother prescribed. During the next two days the bowels were loose and greenish, but baby did not seem ill nor give any uneasiness to father. During the night of the 31st the father telephoned condition of child. Suggestions made and child seen early on August 31. Respirations 64, labored abdominal, just as one badly shocked. At each expiration a moan. Pulse 140. Temperature 99.5. Lungs normal. Liver to be felt below ribs, stools few in number, mucus and green. Extremely restless, Saturday night respirations 70, pulse 180, temperature 99.5. Sunday in desperate condition all day, unconscious, dying at 7 p. m.

Postmortem one hour after death. Stomach empty, but for a little greenish liquid. Intestines contained only dark-green thick bile. Mucous membrane apparently normal. No distension with gas. Spleen not enlarged. Lungs normal, except for congestion at base of right lower lobe. Gall bladder distended with same material as found in intestines. Liver not greatly enlarged—yellowish streaks across surface.

Baby V., male, white, aged 8 months, breast-fed, vigorous child till taken sick on Aug. 6, 1903. First seen in consultation with Dr. Sellers, August 10. History was that baby had been taken ill on the 6th with vomiting and actions from bowels of foul odor. Seen by Dr. Sellers August 7. Temperature 100, respirations 36, pulse 130. On the 8th and 9th bowels difficult to move and stomach irritable. Afternoon of 9th liver found enlarged, bowels tympanitic, vomiting at frequent intervals. Temperature 98, respirations 48, labored, pulse 150. When seen on the 10th temperature 67.5, pulse 140, respirations 44, labored. Vomiting at frequent intervals. This kept up till the morning of the 12th, when the child died at 7 a. m. The physician in charge seemed confident, from the distension, the vomiting and non-acting bowels, that peritonitis had developed, but on the strength of former cases no hesitation was felt in predicting the condition to be encountered on postmortem examination, namely, an enlarged, firm liver, distended gall bladder, filled with thick dark-green bile, normal lungs, spleen and heart and very slight macroscopic change in the intestinal mucous membrane. Such proved to be true on postmortem examination made some four hours subsequent to death.

None of these cases showed the least trace of jaundice. All had abundant flow of urine except Baby V., who vomited so persistently that water could not be taken and absorbed to supply needs of system. In none of these cases, however, was the urine obtained for examination.

On June 22, 1903, was called to see baby H. C., male, white, aged 10 months, breast fed. One action reported that afternoon, loose, with some mucus. No elevation of temperature and child did not look sick. Had been affected in April preceding with a proctitis lasting some two weeks, but never appeared ill. The mother was instructed to leave off nursing for the night and give water in place. Next morning early a telephone message came that baby was very ill. On arrival it was learned that mother had disobeyed orders by nursing him at 5 o'clock, but that he had vomited soon afterward; that

he had been restless and wakeful all night, passing several bloody mucus stools. Temperature 105. All food discontinued, water *ad libitum*, hydrotherapy to control temperature. Temperature kept in bounds during day and night by frequent baths and on following morning had fallen to 101. During course of the 24th—second day of illness—liver showed below border of ribs, temperature went subnormal, labored, abdominal breathing developed. This type of breathing continued for three days. The temperature came to normal, then gradually rose and for ten days ranged from 100 to 102. A slow, protracted convalescence.

Baby B, female, white, 7 months. Breast fed and large for age. Mother noticed bowels slightly upset on Aug. 12, 1903. First seen on 15th. Green mucus actions with a little blood. Baby apparently little sick, temperature and pulse normal. Castor oil given, all nourishment withdrawn, water *ad libitum*. Next day temperature 101.5, pulse 150, respirations 60, labored, abdominal. Liver not palpable below border of ribs. Abdomen not distended. No vomiting. Two dark-green actions from castor oil. Castor oil, half ounce, ordered night and morning. Water to be freely given. On the 17th temperature 99.5, respirations 60. Condition continued practically the same till the 20th, when temperature reached normal, pulse 130, respirations 54, but no longer labored abdominal in type. On Wednesday rice water was allowed and on the 21st allowed to return to mother's breast. Two and three green actions only had been daily obtained from the ounce of castor oil given daily in two doses. This was a mild case, apparently, without noticeable enlargement of liver, returning to mother's milk in six days.

During 1904 one fatal case was seen. This was first seen some three hours before death, but despite the most persistent effort, no postmortem could be obtained. Three cases were encountered in which recovery followed. The measures employed were castor oil often enough to keep the bowels acting, withdrawal of all food till conditions changed, water *ad libitum*. Very little medicine was given beyond castor oil.

The last case seen in 1904 was in October.

Baby M., female, white, aged 15 months, bottle raised in large part. Seen in consultation. History gave two days of greenish actions, then labored breathing. Antitoxin was administered by the attending physician in the belief that the difficulty in breathing came from throat and might be diphtheritic. A throat specialist had been called, who failed to find any membrane in larynx. As soon as seen the case was recognized, the enlarged liver demonstrated, the normal lungs demonstrated and the absence of any throat involvement. This child was in desperate condition for days and for some weeks could assimilate the very smallest quantities of food. Slow convalescence.

During the season of 1905 two cases were encountered, but notes have been misplaced.

Baby H., male, white, aged 6 months, breast fed, vigorous, well-developed child of a physician. Asked to see the baby and found its bowel actions had been green and mucous for a couple of days, but baby did not seem ill. Rectal temperature 100. The following day it developed labored, abdominal, rapid breathing, its liver enlarged, its bowels ceased acting and were only made to act by large doses of oil. The child died unconscious in 54 hours.

The same month, June, 1905, Baby S., male, white, aged about 15 months, was seen at 11 a. m. For one hour previous labored abdominal respiration had existed. The child maintained this labored type of breathing for 56 hours, when it died unconscious.

During the season of 1906 four cases were seen, all fatal. Baby W., male, white, aged 20 months. Breast fed till more than one year old. Chicken-pox recently. Whooping cough few months preceding. Never very robust child, but strongest in family of three. Digestion not good for a week previous to present illness. Seen May 13, in morning. Temperature normal; apparently the child was but slightly ill, though there was a history of several loose actions the preceding night.

Next morning early message came that baby was breathing fast since 5 a. m.—seen at 7 a. m. Respirations 56, labored abdominal type of breathing. Pulse 120, temperature 100. Liver not palpable. Child very restless and had been so all night. At 10 p. m. temperature 100, pulse 140, respirations 56. Liver palpable below border of ribs. Several bowel movements, mucous and bloody tinged; kidneys active. Thirsty; vomited at times.

May 15, temperature varied little from 99.5, respirations from 60 to 64, pulse 150 to 160. Very restless. Apparently conscious, expiratory moan and occasionally cries out. May 16, 7:30 a. m., pulse 170, respirations 62, temperature subnormal; unconscious, pupils dilated. Liver enlarged, reaching within inch of umbilicus. During night bowels had moved in very small quantity a number of times. Child died at 11:30 a. m., 54½ hours after fast breathing developed. Autopsy one and a half hours following. Heart and lungs normal. Spleen not enlarged. Organs apparently normal, except enlarged liver.

Baby P., 1906, male, white, aged 13 months, breast raised, son of physician. Loose bowels on Sunday afternoon, Monday and Tuesday. Not sick enough to excite any notice. Wednesday, June 27, in afternoon labored breathing began. Seen at 10 p. m. the 27th with Dr. Wilson. Father met me with the statement that his baby was extremely ill and he feared would die. Respirations 60, very labored and abdominal, expiratory moan. Pulse irregular and difficult to count at wrist. Temperature 99.1. Liver not palpable below border of ribs. Oil had been given during the afternoon and action resulting was very dark green; 400 c.c. normal salt solution given hypodermatically.

On 28th baby somewhat better. On 29th apparently much better. Temperature 99, pulse 95, respirations 34 and natural. Actions from oil on 29th much lighter in color. On 30th breathing again labored. Pulse so irregular as to preclude counting. Temperature 102. By 10 p. m. 30th liver full two inches below border of ribs and nearly to umbilicus across epigastric region. Very restless, vomiting, and died at 2 a. m. night of 30th. Demonstrations made to father and Dr. W.

Baby Robertson, male, white, aged 15 months, breast fed, seen in consultation with Dr. Wyman and Robertson, Aug. 31, 1906; sick two days. Labored abdominal breathing, developed day previous. Fast abdominal breathing, expiratory moan. Liver enlarged. Died 50 hours after symptoms set in. Seen only once in consultation.

Baby P., male, white, aged 13 months. Breast fed, always been healthy. Seen in consultation with Dr. Mobley, Sept. 9, 1906. Sick for four days. Labored breathing, only 40 to 42 in this case, developed the day previous to being seen. Temperature 99.5, had been subnormal earlier in day. Liver enlarged an inch below margin of ribs. Had been vomiting. Very restless and with expiratory moan. Died following day, about 50 hours from time labored breathing developed.

DISCUSSION.

DR. I. A. ABT, Chicago, said that several years ago he reported two cases almost identical with those of Dr. Parke, and, although he asked many men if they had ever seen such cases, few had recognized them. Dr. Abt emphasized the symptoms the essayist referred to: Children who have been previously healthy fall ill suddenly, with rapid breathing, great prostration, rapid pulse, dyspnea, and examination shows a very large liver. Those cases followed to autopsy show extensive fatty degeneration, so extensive that one of the pathologists to whom he referred such a case reported that the liver resembled the liver of phosphorus poisoning, or acute yellow atrophy. As Dr. Abt's patients approached the fatal termination they showed evidence of obstruction of the bowels; they became very tympanitic and were unable to pass feces or flatus, and in one case immediate laparotomy was advised for a supposed intussusception. The autopsy showed that there was no mechanical obstruction, but a paralytic ileus. In one case albumin leucin and tyrosin were found in the urine, indicating the degenerative changes which had taken place.

DR. R. B. GILBERT, Louisville, Ky., has seen three cases very much like those described. In one of them the autopsy

disclosed an intussusception in a 4 months old infant, with a large liver and other characteristic findings. Another patient died in 36 hours, presenting all the usual symptoms. The liver reached nearly to the umbilicus. Another patient, seen recently, presented rapid breathing, with the liver enlarging. The urine was highly colored with bile. The following treatment was instituted: High irrigation of the colon with normal saline solution, every eight hours. Five grain doses of solution of phosphate of soda in hot water. The baby (5 months old) improved promptly.

DR. J. L. MORSE, Boston, Mass., said that the symptomatology of these cases is quite characteristic of acid intoxication with the acetone bodies, whatever may be the cause of the formation of these bodies. In a number of cases he has found acetone bodies in the urine, although this finding was not of great importance, because the babies were vomiting and taking no food. Several patients treated with bicarbonate of soda in large doses, however, improved considerably.

DR. S. J. WALKER, Chicago, said that while this is a definite symptom-complex, most characteristic is the character of the breathing, the air hunger these patients present, moaning, gasping, irregular breathing, and then alarming symptoms of collapse. Of course, he said, it is a profound intoxication of some sort.

DR. A. C. MERCER, Syracuse, N. Y., asked Dr. Parke whether he knew the pulse and respiration rate of these patients.

DR. C. G. KERLEY, New York City, did not consider the cases Dr. Parke described as being particularly common. He has known of cases that come up to the symptomatology described very well, but so far as his observation goes, the enlargement of the liver is not at all unusual. This, he said, is found in cases which show rapidity of respiration as well as in those that do not. Fatty liver is found in many toxic conditions, whether prolonged or acute. He has always thought that these cases are due to some peculiar form of toxemia. There are many intestinal bacteria that are capable under favorable conditions of producing different kinds of toxemia, as has been shown by Herter and others. Dr. Kerley thought that the rapidity of the respiration was due to some particular form of toxemia that affected the respiratory center and produced unusual stimulation. Some drugs produce such results.

DR. T. D. PARKE, Birmingham, Ala., said that there were fields in several of the sections of liver that showed no normal cells had no nuclei, hardly conforming with fatty infiltration. As to obstruction in any case, the postmortem definitely excluded it from all of the cases coming to section. There was no mouth symptom in any case. The color of the stools was a very dark green, in most instances so dark as to appear black at first sight. The pulse and respiration ratio was variable. Dr. Parke thought that the question of toxemia is an open one.

THE CYTOLOGY OF GONORRHEAL DISCHARGES.*

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Within the past five or six years considerable attention has been directed to the study of the finer structural details, in other words, the cytology of the elements of the discharge of urethritis. This line of study, as well as the methods employed, is practically an application of the recent advances in the morphology of the blood. It is somewhat difficult to see what particular purpose this work subserves, for it has not helped us in understanding the nature of the process nor has it given any aid from a prognostic standpoint. And after reviewing the subject and supplementing it with some

personal observations I have come to the conclusion that any clinical aid or therapeutic guidance from this whole line of work, although sustained by a large volume of literature, is of very doubtful value. It is not susceptible of practical application.

I shall endeavor to lay the subject before you, however, through these personal observations, which, although limited to some 28 cases of ordinary gonorrhea, 5 cases of vulvo-vaginitis in children and 2 cases of gonorrheal ophthalmia, seem to me to cover the field sufficiently for our purposes, as well as would the study of cases by the hundreds. Considerable time has been expended on the relative advantages of various staining mixtures, the methods of fixing the smears, etc., which merely involves the consideration of rather immaterial details. I have found Wright's method of staining blood smears the most practical, since it is very convenient and differentiates the various cellular elements in the discharges and exhibits perfectly their structural details.

As every one knows, the predominant cellular element in gonorrheal discharge all through its course and even in the stage of the threads is the ordinary pus cell or leucocyte, the neutrophilic, polymorphonuclear leucocyte. The only exception to this is the exceedingly early initial and evanescent stage when there may be a considerable proportion of desquamated epithelial cells. These leucocytes are seen in Figures 1 to 5 and 31, and are the cells in which, as shown in Figure 7, the gonococci are most frequently, indeed almost invariably found, although, of course, they may be found in uninuclear cells and on the surface of epithelial cells.

Occasionally among the leucocytes the eosinophilic variety is found, which never contain gonococci and are pictured in Figure 6. These very striking structures, crowded with coarse, brilliant, red granules, have naturally attracted attention from nearly all writers on the subject, and they have endeavored to connote their presence with some particular stage of the disease. There is an impression that this type of leucocyte is present in greatest number in the first month of the discharge. The several statements on this point are very discordant. Both C. and H. L. Posner¹ regard the advent of the eosinophiles as occurring up to the fourth or sixth week of the discharge. After that they gradually decline. Joseph and Polano² also subscribe to this statement and have persuaded themselves into the belief that the decline of the eosinophiles indicate that the process has reached its culmination. On the other hand, Bettman has found voluminous eosinophiles late in the disease, in the gleety stages. Among their tables Joseph and Polano record finding them in the eleventh month of the discharge. In our observation we have found that they are just as liable to be absent as present in the first four weeks of the affection. So, as a matter of fact, there is no definite period which is distinctly related to the advent of the eosinophiles. They may be absent in the early period and, on the other hand, they may be found all through the course of the disease. According to Pella-gatti,³ the eosinophiles appear in the first few days of the malady; they then disappear, only to reappear later on in small numbers. When the discharge is treated,

1. Posner (H. L.): Zur Cystologie des gonorrhoeischen Eiters, Berlin, klin. Wochschr., Oct. 22, 1906, 1401.

2. Joseph (Max) and Polano (M. E.): Cytodiagnostische Untersuchungen Gonorrhoeischer Sekrete, Archiv. für Dermatologie und Syphilis, lxxvi, 1905, 65.

3. Pellagatti (M.): La Costituzione anatomica del Pus blennorrhagico, Giornale Ital. delle Mutattie Veneree e della Pelle, Fasc. 3, 1906, 237.

* Read in the Section on Cutaneous Medicine and Surgery of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.