THE ETIOLOGY OF PACHYMEINGITIS HEMORRHAGICA INTERNA IN INFANTS*

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CHICAGO

Since the first accurate description of pachymeningitis hemorrhagica interna by Virchow¹ in 1856, observers have differed in their views regarding the exact nature of this condition. Virchow believed that the membrane formation on the inner surface of the dura precedes the hemorrhage. Huguenin² maintained that the hemorrhage is primary, the organization of the clot causing the membrane formation. The work of Melnikow-Raswedenkow³ and others supported Virchow's view, which has gradually become the prevailing one.

It was thought that most of the cases seen in the adult⁴ resulted from sclerotic changes incident to chronic diseases of the brain, heart, kidneys and lungs. Similarly, regarding cases occurring in the infant, the prevailing opinion seemed to be that pachymeningitis is secondary to other diseases, the cerebral changes being usually discovered at necropsy in infants dying from various infectious diseases, or in chronic cachectic conditions.⁵

Kremiansky⁶ performed experiments on dogs, showing the importance of alcoholism as an etiologic factor in the production of this disease. Needless to say, cases occurring in infancy cannot be explained by this hypothesis.

It has been maintained by many observers that cerebral hemorrhage occurring at birth is an important factor in these cases. Recent workers though emphasizing other factors in adults, still attribute to

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1. Virchow, Rudolf: Das Hämatom der Dura Mater, Verhandl. d. phys. med. Gesellsch. in Würzburg, 1857, vii, 134.

2. Huguenin, G.: Handbuch d. Speciellen Pathologie und Therapie (Ziemssen), 1876, xi, 342.

3. Melnikow-Raswedenkow, N.: Histologische Untersuchungen über den normalen Bau der Dura Mater und über Pachymeningitis interna, Beitr. zur path. Anat. u. zur allgemeinen Path. (Zeigler), 1900, xxviii, 217.

4. Ciarla, E.: Beitrag zum pathologisch anatomischen u. klinischen Studium der Pachymeningitis cerebri hemorrhagica, Arch. f. Psychiat., 1913, lii, 439.

5. Holt, L. Emmett: Diseases of Infancy and Childhood, New York, 1913, p. 698. Steffen, A.: Handbuch d. Kinderkrankheiten, Tübingen, 1880, v, 380.

6. Kremiansky, J.: Ueber die Pachymeningitis interna hemorrhagica bei Menschen und Hunden, Virchow's Arch. f. path. Anat., 1868, xlii, 129 and 321.

such hemorrhage great importance in the production of this disease in infants.⁷

Boeckmann⁸ made a study of numerous cases coming to necropsy following various cranial operations. Most of these had had considerable hemorrhage following the operation. In no case could Boeckmann demonstrate any pachymeningitis; its absence, he thinks, disproving the importance of trauma as a cause for pachymeningitis.

Marie⁹ and his co-workers were unable to produce pachymeningitis hemorrhagica in dogs and rabbits by the subdural injection of blood.

In a series of 5,998 necropsies on children, Kowitz¹⁰ found intracranial hemorrhage present in 16.9 per cent., pachymeningitis in only 3.9 per cent. Among other causes given for this disease are syphilis,¹¹ scorbutus,¹² hemorrhagic diseases,¹³ and pertussis.⁷

Some observers¹⁴ have laid more or less stress on digestive disturbances in infancy as of possible etiologic significance, while others¹⁵ have noted that cases are occasionally brought to a hospital on account of alimentary symptoms, which often obscure the true nature of the disease.

The possibility of pachymeningitis being the result of a hematogenous infection is suggested by the case reported by Schottmüller¹⁶ of a woman 28 years old who died following a puerperal septicemia of streptococcus origin. The fluid obtained by lumbar puncture during life showed an excess of globulin, the cell count was 181, cultures

7. Hada, B.: Ueber die gehirnkomplicationem des Keuchhustens mit besonderer Berücksichtigung der Pachymeningitis productiva interna, *Virchow's Arch. f. path. Anat.*, 1913, ccxiv, 206. Wohlwill, F.: Ueber Pachymeningitis hemorrhagica interna, *Virchow's Arch. f. path. Anat.*, 1913, ccxiv, 388.

8. Boeckmann, M.: Ein Beitrag zur Ätiologie der Pachymeningitis interna hemorrhagica, *Virchow's Arch. f. path. Anat.*, 1913, ccxiv, 380.

9. Marie, P.: Roussy, G., and Laroche, G.: Sur la reproduction expérimentale des pachyméningites hémorragiques, *Compt. rend. Soc. de biol.*, 1913, lxxiv, 1303.

10. Kowitz, H. L.: Intrakranielle Blutungen und Pachymeningitis hemorrhagica chronica interna bei Neugeborenen und Säuglingen, *Virchow's Arch. f. path. Anat.*, 1914, ccxv, 233.

11. Heubner, O.: Pachymeningitis hemorrhagica bei hereditärer Syphilis, *Virchow's Arch. f. path. Anat.*, 1881, lxxxiv, 267.

12. Sutherland, G. A.: On Hematoma of the Dura Mater Associated with Scurvy in Children, *Brain*, 1894, xvii, 27.

13. Oppenheim, H.: *Lehrbuch d. Nerven krankheiten*, Berlin, 1908, p. 849.

14. Misch, P.: Zwei Fälle von Pachymeningitis hemorrhagica interna, *Jahrb. f. Kinderh.*, 1905, lxii, 229. Poulet, Anna: Ein fall von Pachymeningitis nicht traumatischen natur im Kindesalter, *Inaug. Dissert.*, Zurich, 1902.

15. Göppert, F.: Drei Fälle von Pachymeningitis hemorrhagica mit Hydrocephalus externus, *Jahrb. f. Kinderh.*, 1905, lxi, 51. Meigs, Grace: *Jour. Am. Med. Assn.*, 1913, lxi, 2186.

16. Schottmüller, H.: Pachymeningitis interna infectiosa acuta und Meningismus, *München. med. Wehnschr.*, 1910, lvii, 1984.

were sterile. Postmortem examination revealed a pachymeningitis. Göppert thinks that Krücke's¹⁷ case of pachymeningitis demonstrated in a child dying from pharyngeal and laryngeal diphtheria illustrates a similar possibility. Krücke's case lacks the clinical history prior to death, nor was Krücke able to demonstrate diphtheria bacilli in the lesions, attributing his failure to the fact that the necropsy was not done until sixty hours after death.

Until recently there were few cases of this disease reported in infancy. Finkelstein¹⁸ and Göppert¹⁵ not only described a definite clinical picture occurring in pachymeningitis, but were the first to call attention to its more frequent occurrence in infancy than had been previously recognized.

Rosenberg,¹⁹ reporting thirty-eight cases, divides the disease into three types:

1. A latent form, in which class of cases there may be seen slight restlessness, no nervous symptoms, enlargement of the head, with separation of the sutures and fontanel.

2. A form with acute nervous symptoms. In these cases, the onset occurs with vomiting. Restlessness, the "hydrocephalic cry" and convulsions follow. The reflexes are exaggerated, and there may be spasm of the extremities.

3. A fulminating form in which the patient is in coma, has frequent convulsions and rigidity of the neck. These are the cases which are usually diagnosed as meningitis. There is usually not much fever.

The enlarged head and separation of sutures, suggesting hydrocephalus, is present in all types.

Retinal hemorrhage is of great diagnostic importance, occurring in one-third of Rosenberg's cases. Optic atrophy is occasionally found; choked disk may be present.

Lumbar puncture usually gives a clear fluid under increased pressure. Occasionally, one may get bloody spinal fluid, which always indicates a tear in the arachnoid.²⁰ Hemorrhage due to accidental puncture of a vein during lumbar puncture must be excluded. Hemorrhagic fluid obtained on fontanel puncture is of great diagnostic value. This fluid may be truly hemorrhagic, or exhibit a brownish or yellowish coloration due to the presence of old blood. Rosenberg

17. Krücke, L.: Ein fall von eitrig entzündeter Pachymeningitis hemorrhagica bei Difterie, Inaug. Dissert., Kiel, 1902.

18. Finkelstein, H.: Ueber Pachymeningitis hemorrhagica im Kindesalter, *Jahrb. f. Kinderh.*, 1911, lxxiv, 451; *Berl. klin. Wchnschr.*, 1904, xli, 403.

19. Rosenberg, O.: Ueber Pachymeningitis hemorrhagica im Kindesalter, *Berl. klin. Wchnschr.*, 1911, I, 2272.

20. Quincke, H.: Ueber Lumbalpunktion, *Deutsche klin.*, Berlin, 1906, vi, Part 1, p. 351.

found that 80 per cent. of his cases showed the presence of rhinitis, from which the diphtheria bacillus was isolated. He attributes to this rhinitis considerable etiologic importance. The ethmoidal veins which supply the nasal mucous membrane empty into the ophthalmic veins, which empty into the cavernous sinus. Rosenberg thinks there is a possibility of erosions on the nasal mucous membrane causing a sinus thrombosis, which may produce the pachymeningitis. He attempts to explain the case reported by Heubner, which the latter had attributed to congenital syphilis, on similar grounds, believing the coincident rhinitis to be the possible explanation for the presence of pachymeningitis.

Only recently has more attention been called to the possible infectious nature of pachymeningitis in infancy.

Göppert, in 1905, reporting three cases, expressed the opinion that a primary serous subdural exudation occurred in pachymeningitis. He found no similar suggestion in the previous literature on the subject.

Hahn²¹ reported a case in which he was able to demonstrate an actual serofibrinous exudate.

In a study of the relationship of pertussis to pachymeningitis, Hada⁷ concludes that toxins or bacteria first produce an infection which is subsequently followed by hemorrhages.

The actual occurrence of symptoms of serous meningitis preceding the development of hydrocephalus in a case, which after death showed pachymeningitis, suggests to Freund²² the possibility of pachymeningitis being of an infectious nature.

Though Rosenberg demonstrated a streptococcus in the bloody fluid obtained from two patients with pachymeningitis, one of whom recovered, and one of whom died with a streptococcus meningitis, he lays no stress on the isolation of the streptococcus in these cases.

Excepting this finding of Rosenberg, in all recent reported cases²³ of pachymeningitis which have included bacterial examinations, no organisms have been demonstrated in the spinal fluid.

Finkelstein¹⁸ concludes that bacteriologic study of this disease has not shown it to be of an infectious nature.

21. Hahn, H.: *Klinischer Beitrag zur Lehre von der Pachymeningitis hemorrhagica interna im frühen Kindesalter*, Deutsch. med. Wchnschr., 1911, xxxvii, 1518.

22. Freund, W.: *Zur Therapie des Hydrocephalus zugleich ein Beitrag zur Kenntnis der Pachymeningitis hemorrhagica im Säuglingsalter*, Monatschr. f. Kinderh., 1909, vii, 613.

23. Gordon, A.: *Internal Pachymeningitis in Young Children*, Jour. Nerv. and Ment. Dis., 1914, xli, 382. Hartje, E.: *Zur Casuistik der Meningitis basilaris posterior*, Arch. f. Kinderh., 1912, lvii, 333. Schindler, R.: *Ein Fall von Meningocele spuria traumatica kombiniert mit Pachymeningitis hemorrhagica interna*, Jahrb. f. Kinderh., 1912, lxxvi, 160.

The diagnosis of the case to be reported was that of pachymeningitis. It is based on the clinical picture entirely.

In this picture there stand out prominently the repeated bloody character of the spinal fluid, the severe retinal hemorrhages, and the fact that the subdural fontanel puncture yielded an amber colored fluid. All of these features have been accepted by Rosenberg and others as indicating the presence of pachymeningitis. Naturally there exists the possibility that we are dealing with a particular case of leptomeningitis, or encephalitis with pronounced hemorrhagic lesions. The lack of a necropsy does not permit the absolute exclusion of such possibilities. If so, then a number of cases reported in the literature as pachymeningitis on purely clinical evidence are subject to the same objections.

Even accepting such possibilities, these cases become important, inasmuch as they may be gradations between the usual form of leptomeningitis and pachymeningitis.

The case herewith reported is one in which the meningococcus was demonstrated in the spinal fluid on several separate examinations.

REPORT OF CASE

History.—Jennie P., Polish girl, born January, 1915, was admitted to The Children's Memorial Hospital, May 10, 1915, on account of convulsions and diarrhea.

Family History.—Parents living and well. No other children. No miscarriages. No known history of tuberculosis in family.

Past History.—Protracted labor, no forceps used. Birth weight 9¼ pounds. No respiratory trouble following delivery. Breast fed one week, following which the infant was fed on various milk mixtures. Feeding intervals irregular. At two weeks she had furunculosis. At four months the patient had three convulsions, cause unknown. Was treated in Out-Patient Department three weeks prior to admission for fracture of left humerus.

Present Illness.—Began May 3, 1915, with convulsions, which recurred frequently for twenty-four hours. Then followed two days, during which time, no convulsions occurred.

May 6, 1915, convulsions recurred, continuing since. Had had a diarrhea; stools described as foul, green, watery. Had had a slight cough; no vomiting.

Physical examination showed an under-nourished baby with no abnormal findings.

During a four-days' stay in the hospital, the temperature was normal, the stools appeared normal, the infant had no convulsions, and gained 9 ounces on a mixture of ⅔ whole milk and ⅓ water, with 3 per cent. additional dextrin-maltose.

The case was considered one of convulsions due to improper feeding and the patient was discharged May 14, 1915. May 17, 1915, she was readmitted. The mother stated that the child had a convulsion shortly after returning home and cried much that day and the next. On the second day home the patient vomited and had vomited persistently since. For the previous two days had appeared too weak to cry. Bowel movements normal.

Physical Examination.—Semicomatose. Extremely pale. Does not resist examination. Respirations regular but shallow. Emaciated. Very poor tissue turgor. Corneae dull, eyes fixed.

Head: All sutures of skull widely separated. The anterior fontanel was tense and bulged considerably. Slight craniotabes present. Head appeared large in comparison to body.

Eyes: Pupils equal, left did not react to light. Right pupil reacted sluggishly.

Ears, Nose, Mouth and Throat: Nothing abnormal noted.

Thorax: Rachitic rosary present.

Heart: No abnormality noted.

Lungs: Few mucous râles scattered over chest. No dulness. No abnormal breath sounds.

Abdomen: Level and tympanitic. Liver edge felt 4 cm. below costal margin. Spleen not felt.

Extremities: Slight rigidity.

Laboratory Reports.—White cell count, 20,600.

Hemoglobin (Tallquist), 65 per cent.

Differential Count:

Polynuclears, 76 per cent.; small mononuclears, 14 per cent.; large mononuclears, 6 per cent.; basophils, 4 per cent.

Wassermann: Negative.

Vaginal Smear: No gonococci found.

Urinalysis: Nothing abnormal found.

Throat and Nasal Culture: Negative for diphtheria bacilli.

Von Pirquet reaction negative.

Roentgenogram of head and chest negative.

Lumbar Puncture: Sixty c.c. bloody fluid under increased pressure. Hang-drop shows field full of red blood cells, no organisms seen. Cultures made show growth, within twelve hours, of a large bacillus (contamination).

May 18, 1915. Lumbar puncture: 3 c.c. bloody fluid, no examination made. Left lid drooping. Left foot and hand show occasional twitching.

May 19, 1915. Fronto-occipital circumference, 40.5 cm.

May 20, 1915. Ophthalmoscopic examination (Dr. Alfred Hall): Chorio-retinitis with marked hemorrhage throughout both retinæ.

May 22, 1915. Lumbar puncture: 5 c.c. bloody fluid under increased pressure. Cultures made in bouillon. After thirty-six hours' growth, diplococcus isolated.

May 25, 1915. General condition somewhat improved. Color better. Responds more to stimuli. Has fairly normal cry during examination. Opens eyes, but apparently does not see. Fontanel tense. Pupils unchanged. Suggestive Kernig's sign on left side. Brudzinsky sign present. Slight spasticity of extremities.

May 27, 1915. Lumbar Puncture: 20 c.c. bloody fluid under increased pressure. Cultures made in human blood agar and plated. After twenty-four hours diplococcus isolated.

May 29, 1915. Lumbar Puncture: 40 c.c. amber colored fluid, under increased pressure. Unstained smears show crenated and degenerated red cells. No organisms; 10 c.c. antinephritis serum given. Cultures made in bouillon and human blood agar; diplococcus isolated.

May 30, 1915. Patient lies very quietly, but cries when disturbed. Piercing shriek when extremities are manipulated to elicit reflexes. Knee jerks not obtained. No Kernig's sign. Marked spasticity of extremities. Fronto-occipital circumference 42.5 cm. General appearance of hydrocephalus.

May 31, 1915. Lumbar Puncture: Sixty c.c. amber-colored fluid under increased pressure. Cultures made in litmus milk and on plain agar. No growth obtained.

June 2, 1915. Lumbar Puncture: Forty c.c. amber-colored fluid under increased pressure. Smears show many white and red cells and an occasional diplococcus. Cultures made on human blood agar; diplococcus isolated.

June 3, 1915. Ophthalmoscopic Examination (Dr. Alfred Hall): Extreme atrophy of both optic nerves. Hemorrhages of retina clearing up.

June 4, 1915. Lumbar Puncture: Forty c.c. clear yellow fluid under increased pressure. Smears show increased number of leukocytes, polynuclears predominating. No organisms. Cultures on human blood agar; no growth.

June 8, 1915. Lumbar Puncture: Forty c.c. bloody fluid under increased pressure. Cultures in bouillon; diplococcus isolated.

June 11, 1915. Lumbar Puncture: Thirty c.c. clear fluid under increased pressure. Cultures in bouillon; no growth.

June 26, 1915. Lumbar Puncture: Sixty c.c. fluid; first 30 c.c. clear, last 30 c.c. a pale yellow; pressure greatly increased. Cultures in bouillon; no growth. Furunculosis of scalp; furuncles incised; cultures made. Staphylococcus present.

July 13, 1915. Facial expression not as drawn, though there is still marked pallor and universal loss of tissue turgor. Sutures and fontanel are beginning to approximate themselves. Fronto-occipital circumference 41.5 cm.

July 27, 1915. Fontanel bulging somewhat. Lumbar puncture and fontanel puncture done. The fluid obtained at both points was under increased pressure. That from the fontanel (subdural) was amber colored, and on standing showed a deposit of blood. Cultures made in bouillon; no growth. The fluid obtained by lumbar puncture was perfectly clear. Cell count 50. Noguchi: Faint turbidity. Permanganate reduction index, 3.4. Excepting for a short interval when patient had furunculosis, the temperature remained normal throughout illness. Weight practically stationary. Aug. 14, 1915, patient began losing weight, failed steadily, and died August 23. No necropsy granted.

Bacteriology.—The organism isolated from this case of pachymeningitis was demonstrated in cultures of the cerebrospinal fluid obtained from five different lumbar punctures. The organism grew well on human blood agar and in bouillon. Subcultured, it grew on most of the ordinary laboratory culture mediums. On agar slants the growth appeared as slightly larger than pin-head, round, glistening, grayish-white, discrete, elevated colonies, which as growth continued formed a confluent streak with slightly irregular edges.

By Gram staining it appeared as a large gram-negative coccus, occurring in diplo, tetrad or group arrangement, which characteristics it retained in further subcultures.

Several observers²⁴ have reported the finding of other gram-negative diplococci than the meningococcus in the cerebrospinal fluid. Furthermore, the importance recently ascribed to the parameningococcus²⁴ as a causative agent in resistant cases of epidemic meningitis, made it necessary to determine by differential tests the exact nature of the organism isolated.

It has been shown by von Lingelsheim²⁵ that the meningococcus can be differentiated from other gram-negative diplococci by its reactions to various sugars. According to his work, the meningococcus changes dextrose and maltose, but not levulose, saccharose, galactose and lactose.

The medium used for the differentiation was prepared in the usual manner.²⁶

24. Kutscher: *Kolle-Wassermann Handbuch d. path. Mikro-organismen*, Jena, 1912, p. 589. Dopfer, M.: *Diagnostic bactériologique des méningites cérébrospinales à méningocoques et à parameningocoques*; Société méd. d. Hôpitaux d. Paris, Series 3, 1914, xxxvii, 1217.

25. v. Lingelsheim, W.: *Klin. Jahrb.*, 1906, xv, 373.

26. Kolle-Hetsch: *Die Experimentelle Bakteriologie und du Infektionskrankheiten*, Berlin, 1911, p. 378.

The growth on this medium was slow in appearing, no culture showing growth before forty-eight hours, while some showed no growth before sixty hours. The colonies on the dextrose and maltose ascites agar soon turned pink, while those in the other sugars remained colorless or slightly blue colonies.

Albrecht and Gohn²⁷ were the first to note the occurrence of specific agglutinins in the blood serum of animals receiving repeated injections of meningococci.

Specific agglutinins in the serum of patients ill or convalescent from epidemic meningitis were first noted by Bettencourt and Franca.²⁸

Following the work of Dopter and others on the parameningococcus, agglutination through specific serum reactions is now considered the most trustworthy guide in the identification of the meningococcus.²⁹

While normal serums may agglutinate the meningococcus in dilutions of 1 to 20, serums containing specific agglutinins will agglutinate the meningococcus in greater dilutions.²⁸

The serums used in the present instance were as follows:

1. Jennie: Patient's own serum.
2. Stephanie: Serum of a convalescent epidemic meningitis patient who had responded to treatment with Flexner's serum.
3. Antimeningitis serum: New York State Board of Health.
4. Normal serum (author's blood).

A suspension of the organism having been made in 0.9 per cent. salt solution, various dilutions of the serum were then made to which were added the same amount of suspension. The tubes were placed in the incubator at 37 C. for two hours, then transferred to the ice chest for twenty-four hours.

The accompanying table gives the results of the agglutination determinations:

RESULTS OF AGGLUTINATION DETERMINATIONS

	1 to 40	1 to 60	1 to 80	1 to 100	1 to 120	1 to 160	1 to 320	1 to 640
Jennie's serum	+	+	—	—	±	—	—	—
Stephanie's serum	++	++	+	—	—	—	—	—
Antimeningitis serum	+	+	±	—	—	—	—	—
Normal serum	—	—	—	—	—	—	—	—

27. Albrecht, H., and Ghon, A.: Ueber die Aetiologie und path. Anatomie der Meningitis cerebrospinalis epidemica, Wien. klin. Wchnschr., 1901, xiv, 984.

28. Bettencourt, A., and Franca, C.: Ueber die Meningitis cerebrospinalis epidemica und ihren Erreger, Ztschr. f. Hyg. u. Infectiönskr., 1904, xlv, 463.

29. Flexner, Kraus and Levaditi: Handb. d. Technik, u. Methodik d. Immunitätsforsch., Jena, 1914. Quoted by Wollstein, M.: Jour. Exper. Med., 1914, xx, 201.

DISCUSSION

It appears probable that pachymeningitis hemorrhagica as it occurs in infancy is an infectious disease, which may be caused by the meningococcus or other organisms. That this possibility should have passed unnoted by students of pachymeningitis seems all the more remarkable when it is recalled that Weichselbaum³⁰ in his early work with the meningococcus was able to produce pachymeningitis and leptomeningitis and acute encephalitis by the injection of this organism.

His experiments were done on dogs, the injection of the meningococcus being made subdurally after trephining the skull. The dogs appeared well for several hours. After five or six hours they appeared sick. One dog died the same evening. The second, acutely ill, was killed on the third day. The third dog died on the twelfth day. The first two dogs showed fluid blood between the dura and the cerebrum, a hemorrhagic area of softening in the cerebrum, with much injection of the meninges. The third dog showed a thick reddish exudate between the dura and the brain, while in the brain substance there was an abscess the size of a hazelnut, and numerous hemorrhages.

It seems probable that pachymeningitis and leptomeningitis are but different manifestations of a single process, the meningococcus being the offending organism in both diseases.

In the light of Rosenow's³¹ work with the streptococcus, it seems possible that the meningococcus, in like manner, at one time may produce leptomeningitis, at another pachymeningitis. That transition forms of meningitis do occur, suggesting such a possibility, is evident from the studies of various epidemics of the disease. While the predominating feature of the usual case is an acute fibrinopurulent exudate involving the pia-arachnoid of the brain and cord, the dura is usually vascular,³² and hemorrhages in the brain and other organs are common. The extent of these hemorrhages varies considerably in various epidemics. Thus Bettencourt and Franca²⁸ in their studies on epidemic meningitis comment on the frequency of hemorrhages in their series, the histologic picture of the brain being often that of meningo-encephalitis hemorrhagica. Subarachnoidal hemorrhages are also noted by these observers.

As there are various forms of meningitis, produced by various organisms, it is quite probable that similar divisions occur among the

30. Weichselbaum, A.: Ueber die Aetiologie des akuten Meningitis cerebrospinalis, *Fortschr. d. Med.*, 1887, v, 620.

31. Rosenow, E. C.: *Jour. Infec. Dis.*, 1915, xvi, 240; *ibid.*, xvi, 367; *ibid.*, xvii, 219.

32. Westenhoeffer, M.: *Pathologisch-anatomischen Ergebnisse der Oberschlesischen Genick-starre epidemie von 1905*, *Klinisches Jahrb.*, 1906, xv, 657.

cases of pachymeningitis. Indeed, the cases reported by Rosenberg of pachymeningitis in which streptococci were isolated, and the case reported by Dupérié³³ in which tuberculous meningitis and pachymeningitis were present in the same case, confirm this idea. That organisms have not been isolated in these cases heretofore is probably due as much to the lack of search, as to the difficulty often encountered in their isolation.

33. Dupérié, R., and Dubourg, E.: Pachyméningite hémorragique. Hémorragie méningée et tuberculose encéphalo-méningée chez un enfant de deux ans, *Gaz. hebdomadaire de médecine et de chirurgie*, 1913, xxxiv, 157.