ACUTE ANTERIOR POLIOMYELITIS COMPLICATING PREGNANCY*

LEON S. McGoogan, M.D., OMAHA, NEBR.

(From the Department of Obstetrics and Gynecology, University of Nebraska College of Medicine)

A CUTE anterior poliomyelitis, apparently unknown as a definite disease entity prior to Underwood's¹ paper in 1784, has assumed a more and more prominent position in medicine during the past third of a century. Although much has been written concerning this disease, there are only a few scattered articles pertaining to the relationship of acute anterior poliomyelitis to pregnancy and to its occurrence during pregnancy. There are several possible explanations for the lack of literature dealing with the latter subject.

Acute anterior poliomyelitis is primarily a disease of childhood. In Vermont from 1912 to 1925 there were 848 cases of poliomyelitis, of which 176 or 23.5 per cent were over fifteen years of age. In Massachusetts from 1914 to 1923 there were 1594 cases reported, and only 145 or 9.09 per cent were over fifteen years of age. According to Wickman³ and others the disease is slightly more common in males than females. Aycock² in a study of the age incidence of the disease concluded that "the majority of persons become immunized either subclinically or through unrecognized attacks of the disease" before they reach adult age.

Believing that an immunity to the virus of poliomyelitis in individuals who gave no history of having passed through an attack of the disease might be present, Aycock and Kramer⁴ tested 75 normal individuals in Massachusetts and Vermont. Forty-six of the individuals were from urban communities and 29 were from rural districts. Of the urban population 69.6 per cent were immune by the neutralization test and only 20.7 per cent of the rural population were immune. In the age group fifteen to nineteen years, 85.7 per cent of the urban population and 50 per cent of the rural population were immune. In the adult group, 87.5 per cent of the urban and 40 per cent of the rural population were immune. The authors concluded that there is a wide spread immunity to poliomyelitis among individuals not known to have had the disease, and believe that this immunity originates in exposure to the virus, and from the extent to which it occurs and the order in which it develops that the virus spreads by person to person contact, the actual disease occurring in only a small portion of those exposed.

^{*}Note: For lack of space, the complete paper of Dr. McGoogan cannot be included in the Journal but may be had in the author's reprints.

That the immunity to poliomyelitis is equally extensive in warmer and cooler climates is shown in that the serum of 18 out of 21 normal adults in Georgia neutralized the virus.⁵

From these studies it must be concluded that the occurrence of the disease in women during pregnancy must be rare. If the rare occurrence is not recognized, or if recognized is not reported in the medical literature then the disease becomes, on paper, relatively more rare.

Alpers⁶ in an extensive study of the cerebral and spinal complications occurring during pregnancy states that poliomyelitis complicating pregnancy is of rare occurrence.

Three cases of acute anterior poliomyelitis during pregnancy have been observed in Omaha, Nebraska, during the past five years. This led to a survey of the literature upon the subject and five similar cases were found.

The first case reported is that of Schell's, in 1906. Although he diagnosed the case as one of myelitis, a review of the history warrants the reclassification to that of acute anterior poliomyelitis.

Renault and Martingays reported the second case in 1911.

Millerº in 1924 reported two more cases.

Foulkrod¹⁰ in 1924 reported a case as probably poliomyelitis, but after a careful review of the case history, it should be classified as an acute infectious myelitis rather than a poliomyelitis.

Hornung and Creutzfeldt11, 12 reported the fifth case in 1930.

The three cases occurring in Omaha follow, and the first two are from the private service of Charles W. Pollard.

Case 1.—Mrs. J. The patient was a primipara aged twenty-four, who had her last menstrual period May 10, 1926. She was well except for mild morning nausea until August 23 when she developed rather severe nausea. The following day she felt faint and tired. On August 25 she developed tenderness in the back, pain on motion in the back and legs and was unable to stand. There was moderate vomiting and severe headache. These symptoms persisted for four days after which time the patient developed urinary retention and paralysis of the right leg. The urinary paralysis lasted only two days. The pain in the back and legs persisted for three weeks then began to abate and the paralysis began to improve. She was admitted to the Omaha Maternity on September 6, 1926, two weeks after onset of her disease, and an examination at that time revealed absence of the right abdominal reflex, weakness of the right leg and a bilateral Kernig sign. A lumbar puncture was done, the pressure being 4 mm. mercury. The protein content was 25 mg. per 2 c.c. of spinal fluid, and a cell count showed 14 cells per c. mm. A spinal fluid Wassermann was negative and a colloided gold curve was not remarkable.

The patient improved with massage to the right and left legs and after two months was walking without support. She went into labor on February 3, 1927, and after a normal labor delivered a male child weighing 6 pounds and 12 ounces. The placenta was expelled normally. The puerperium was normal. The child was apparently normal and is well at the present time. The patient has had one normal pregnancy since.

Case 2.—Mrs. N. The patient was a para ii, aged thirty-two, who had her last menstrual period on May 14, 1930. She was well except for slight morning nausea and vomiting until August 24, 1930, when she developed a generalized muscle sore-

ness in back and legs as if she had influenza, and she became very drowsy. She felt well for the next two days when she had a recurrence of the symptoms. The following day she felt so ill that she was confined to her bed and developed chills, fever, and epigastric pain with nausea and vomiting. These symptoms continued for seventy-two hours. One week after the onset of her illness she developed shooting pains in both legs and back. The nausea, vomiting, and epigastric distress ceased and the temperature became normal. Toward evening of that day her left leg became so "tired" that she could not move it. The following morning she felt so well that she got out of bed and while making her toilet she collapsed and found that she could not arise. The following day she noticed that she could not arise from a sitting posture or sit down, but if she were helped up she could walk by holding onto a chair, pushing it before her.

She was admitted to the Bishop Clarkson Hospital on that day, and an examination by Dr. Robert Shrock showed weakness of the abdominal muscles, more on the left than on the right, bilateral weakness of the hip muscles, no complete paralysis of the legs although the thigh muscles were the weakest. Dr. Shrock concurred in the diagnosis of acute anterior poliomyelitis, involving the last four dorsal segments of the cord. The patient was treated by massage and exercise and after three months had returned to two-thirds of her normal muscular power of the muscle groups involved.

The pregnancy continued uneventfully until January 5, 1931, when the patient developed a rise in blood pressure to 140/90, with three-plus albuminuria and slight edema of the extremities and face. She was treated at the Omaha Maternity Hospital from January 18 to 22 for toxemia, and was greatly improved on her discharge. She had a return of symptoms however, and was readmitted to the Hospital February 6 with blood pressure 150/90, albuminuria 2-plus, slight edema of face and hands and slight headache. She again improved under toxemia treatment and on February 10 was awakened at 2 a. m. by mild lower abdominal pains. She delivered herself at 5:30 a.m. of a normal female child weighing 6 pounds. The placenta was expelled normally and there was little bleeding. The puerperium was uneventful. Six weeks following delivery the patient had recovered 90 per cent of the function and power of her involved muscles. The child was well and showed no deformities.

Case 3.—This case is reported through the kindness of Dr. Frank J. Munk. The patient was a primipara, aged twenty-four, and in the third month of gestation. On April 28, 1926, she developed pains in lower abdomen, pain and soreness in the back, both legs, and the right arm. There was an associated intense headache, and the temperature was 102°. The patient became paralyzed in both legs and the right arm the day after onset and developed urinary retention and constipation. There was no impairment of sensation. A lumbar puncture showed a slight increase in pressure, but otherwise normal findings. On April 30, May 1, and May 2 the patient was given Rosenau's serum intraspinally. She required catheterization of the bladder during this time. The temperature gradually subsided and on May 7 was normal. At this time the patient began showing some recovery of motion in the right arm and return of bladder function, and of bowel function.

She was treated with massage and plaster splints to her legs with slight improvement.

On September 21, the patient went into normal labor, and delivered a premature (eight months) child, weighing 5 pounds. The child lived only two days, the cause of death is unknown but was thought to be prematurity. An autopsy was not done.

The mother has regained very little function of her lower extremities, and is able to get about only with the aid of supports. She has not been pregnant since.

The mode of infection in this case is interesting. Living in the patient's home was a graduate nurse who was nursing a case of acute poliomyelitis.

EFFECT OF THE DISEASE UPON PREGNANCY

Any disease arising as a complication during pregnancy may affect the pregnancy. In the eight cases reported, the pregnancy itself was in no way affected. In the seven cases in which the disease occurred during the early months of gestation, there was no spontaneous interruption of the pregnancy nor was there intrauterine death of the child during the acute stage of the infection itself. What the ultimate effects in Miller's second case would have been are problematical, and undoubtedly Hornung's patient would have died undelivered had there not been the intervening cesarean section. One case (Munk's) delivered prematurely but five months after the onset and subsidence of the acute stage, and in view of the frequent occurrence of premature labor, it is doubtful if there was any relationship between the premature labor and the poliomyelitis. Although the series is small, it may be concluded that acute poliomyelitis occurring during pregnancy has no effect upon the pregnancy itself.

Pregnancy upon the other hand may be a factor in the severity and ultimate outcome of any disease already existing at the onset of gestation or occurring as an intercurrent infection. In only two cases out of the entire series, Miller's second case and Hornung's case, does pregnancy seem to have any deleterious effect upon the progress of the disease or its outcome. In Miller's second case, the patient was greatly improved following the interruption of the pregnancy. The patient had a paralysis of the bladder with a fulminating type of cystitis. The enlarged uterus evidently prevented a return of the normal tonus of the bladder, and a cystitis intervened as so frequently happens in those cases in which retention of urine, secondary to lesions in the spinal cord, occurs in paralytic bladders. Her long continued cystitis was undoubtedly the cause of her poor physical condition. Interruption of the pregnancy with resultant improvement of the patient's condition followed. In the remainder of the cases, although bladder paralysis was present, it was of short duration.

In Hornung's case the importance of the relationship of the extent of the paralysis to the duration of the pregnancy is clearly demonstrated. The patient developed an ascending paralysis involving the diaphragm with an increasing cyanosis. The aeration of the lungs in a normal pregnant woman at term is accomplished by a costal type of respiration rather than abdominal due to the restricted excursion of the diaphragm¹³ and with emptying of the uterus, the chest is relieved. If the diaphragm becomes paralyzed then respiration is accomplished entirely by the accessory muscles. In an effort to obtain better aeration of the lungs already embarrassed by diaphragmatic paralysis, Hornung performed a cesarean section. The patient's condition appeared to have been temporarily improved as a result of this procedure in as much as she was not cyanotic. The disease was progressive and

nervous system from the nasopharynx has not been definitely determined. The two possible routes are, (1) via blood stream, and (2) via the perineural lymph spaces of the olfactory nerve. Experimental demonstrations have supported the latter route (Draper³²). If the latter route is the only route and the virus does not enter the blood stream, then it seems as if it would be anatomically impossible for the fetus in utero to develop acute anterior poliomyelitis.

The virus has not been found at any stage of the infection in the blood in human beings, though in the monkey, on the first day of symptoms it has been detected in one instance when large amounts of the virus were inoculated. After intravenous injection of the virus into the blood, the virus tends to disappear so that after one hundred and twenty hours it is no longer present. He finding of the virus in the lymph glands (Amoss³⁵), (Levaditi³⁶), supports the theory that the virus must be blood borne at some stage of the disease, possibly very early and its presence of very short duration. If the virus is blood borne at some stage of the infection before it localizes in the central nervous system, then it seems possible that it might be transmitted via placenta to a fetus in utero.

In only one of the eight cases reported is there any suggestion of intrauterine poliomyelitis and that is in Miller's first case in which the child was born with a bilateral club foot. Miller, however, points out that in view of the frequent occurrence of this deformity, it cannot be concluded that it is the result of a transmitted intrauterine poliomyelitis. One infant, delivered before the period of viability, was in no way deformed; and one was premature and died in forty-eight hours, yet it showed no external evidence of deformity. The other five children were perfectly formed.

EFFECT OF THE DISEASE UPON LABOR

The effect of the paralysis and bony deformities subsequent to an attack of poliomyelitis upon labor depends upon a number of factors, (1) the extent of the paralysis, (2) the size and shape of the pelvis, (3) the size of the child and the relationship of the size of the head and the pelvis to one another, and (4) the general condition of the mother.

* * * *

Miller⁹ discussed the possibility of prolonged bladder paralysis interfering with the second and third stages of labor. A full bladder might interfere with the normal contractions of the third stage and increase the possibility of a postpartum hemorrhage.

That the onset of uterine contractions occurs in spite of spinal cord lesions has been shown by many experiments and clinical cases. Normal delivery can and will occur with all the nerves to the uterus severed. That the third stage may also be without incident is shown by Good's experiments.⁵³ He performed transection of the cord upon a pregnant guinea pig twenty-four hours before a cesarean section was done. Following the cesarean section the uterus contracted promptly without hemorrhage, and he concluded that the sympathetic nervous system and not the spinal cord controlled uterine contractions. Rudolph and Ivy⁶⁵ studied the uterine contractions in the postpartum uterus of the dog and came to the conclusion that there is an extrinsic and intrinsic mechanism, one resident in the uterine wall and the other in the uterovaginal ganglia, the former being the most important.

Very little difficulty should be expected with delivery in any of its stages, unless there is disproportion or malposition present, in a woman who has had poliomyelitis either preceding or during pregnancy. The six patients, who delivered via naturalis in the above reported series in which poliomyelitis occurred during pregnancy, delivered without difficulty, labor was not unduly prolonged, and the third stage was normal as to time and blood loss.

DIAGNOSIS OF ACUTE ANTERIOR POLIOMYELITIS

Luther and Aycock⁶⁶ in 1929 studied a large series of cases in order to determine the incubation period of the disease and from their studies concluded that the apparent incubation period falls within a period of from six to twenty days. They found evidence that "the infectious period of the disease may extend from the fourteenth day preceding the onset of symptoms to at least the fifth day of the disease."

Luther and Aycock^{67, 68, 60} have given methods of making the diagnosis in the preparalytic stage. The patient is taken ill with fever, headache, and occasional gastrointestinal disturbances, nausea, vomiting, and constipation. Drowsiness and a desire to be let alone are frequently observed. These findings resemble most any acute infection and one should be on the watch for the following symptoms: flushed face, a rapid pulse, more prostration than the temperature warrants, and evidence of meningeal irritation, such as a coarse tremor, slight rigidity of the neck, stiffness of the spine, a very slight Kernig's, but with hyperactivity of the deep reflexes which later diminishes. A lumbar puncture with spinal fluid examination reveals the fluid to be under slightly increased pressure, an increase of white cells usually 50 to 250 per c.mm., increase of globulin and normal sugar, and the colloidal gold curve shows an early slight rise.⁷⁰ The patient may or may not develop a flaccid paralysis within the next forty-eight to seventy-two hours.

DIFFERENTIAL DIAGNOSIS OF ACUTE ANTERIOR POLIOMYELITIS

Acute anterior poliomyelitis may be confused with several other diseases which run a similar course or which show weakness or paralysis simulating that which occurs in poliomyelitis. A careful history and physical examination with study of the reflexes and sensory changes, accompanied by spinal fluid studies, will aid in making the correct diagnosis. Many of the acute infections in their early stages simulate the

early stage of poliomyelitis before the onset of the paralysis. One of the common diseases so confused in influenza, which the patient, reported as Case 2 of the Omaha series, thought she had before the onset of her paralysis. Renault and Martingay's patient had a similar condition. Acute articular rheumatism and typhoid fever are also commonly confused with poliomyelitis. Epidemic encephalitis with its prodromal symptoms of fatigue, headache and myalgia may so closely resemble poliomyelitis as to make the differential diagnosis difficult. However, encephalitis is primarily a disease of the higher nervous centers, and poliomyelitis a disease of the spinal cord. The presence of an epidemic aids in making the diagnosis.

Epidemic cerebrospinal meningitis may be confused with the encephalitic form of poliomyelitis as the symptoms of headache, vomiting, lassitude, fever and rigidity of the neck usher in both diseases. Spinal puncture and the finding of the meningococcus makes the diagnosis. The paralyses are different, as that of poliomyelitis is flaceid and of meningitis spastic.

An important differential lies between acute anterior poliomyelitis and the neuritides, the mononeuritis and the polyneuritis. In mononeuritis any nerve may be involved. Fink⁷¹ reports a series of cases in which the nerves of the shoulder girdle, the facial nerve, the ulnar nerve, the median nerve, and the lumbar nerves respectively were the site of the disturbance.

TREATMENT

Treatment of acute anterior poliomyelitis should be directed toward lessening the damage to the central nervous system. Early diagnosis and institution of therapy early are of great importance. The general opinion is that the use of human convalescent serum seems the most rational form of treatment, following the technic of Aycock, Luther, and Kramer.⁸¹

Rosenau had used antistreptococcic horse serum in 1,300 cases and reports favorable results. The best results are obtained with serum therapy in the preparalytic stage but it may be used with less favorable results in the early paralytic stage. After the paralysis appears, the diagnosis becomes quite apparent, and treatment should be directed toward the prevention of deformities and increasing the patient's general condition. MacAusland describes in great detail the treatment of this phase of the disease and what methods should be used to prevent the development of deformities.

The presence of a pregnancy, after reviewing the series of cases presented, in no way should alter the accepted forms of treatment for poliomyelitis. In the majority of cases when the disease occurs early during the gestation period, the pregnancy can be disregarded. If it occurs late as in Hornung's case, or causes a cystitis as in Miller's case then interruption of pregnancy should be considered. Pregnancy following a recent poliomyelitis should be advised against, because of the possibility of the pregnancy affecting the rapidity of recovery as is suggested in Fernandez's case.

SUMMARY

- 1. Eight cases of acute anterior poliomyelitis complicating pregnancy are referred to, five from the literature and three additional cases.
- Acute anterior poliomyelitis complicating pregnancy has no effect upon the pregnancy itself.
- 3. Pregnancy may have some effect upon the rapidity of the patient's recovery from the disease. It may increase the severity of the poliomyelitis, or its complications, such as, cystitis, and diaphragmatic paralysis.
- 4. Interruption of pregnancy should not be done except in those instances in which the uterus encroaches upon the diaphragm and there is diaphragmatic paralysis; or in the case of a severe cystitis, or other complications.
 - 5. Intrauterine poliomyelitis if it does occur must be very rare.
 - 6. Passive transmission of maternal immunity to the child does occur.
- 7. Very little difficulty should be expected with delivery in any of its stages, unless there is malposition or disproportion present in a woman who has had poliomyelitis either preceding or during her pregnancy.
- 8. Pregnancy following a recent poliomyelitis should be advised against.

Although eight cases is a small series from which to draw conclusions, much of interest has been learned from them. As more cases are reported, and there undoubtedly will be as the disease is apparently becoming more common, a better understanding of the pathology of acute anterior poliomyelitis and pregnancy will be reached.

REFERENCES

(1) Underwood: Diseases of Children, Philadelphia, 1784. (2) Aycock, W. L.: Am. J. Hyg. 8: 35, 1928. (3) Wickman, I.: Nerv. & Ment. Disease Monographs Series 16: 12, 1913. (4) Aycock, W. L., and Kramer, S. D.: J. Prev. Med. 4: 189, 1930. (5) Aycock, W. L.: J. Prev. Med. 4: 201, 1930. (6) Alpers, B. J., and Palmer, H. D.: J. Nerv. & Mental Dis. 70: 456, 1929. (7) Schell, J. T.: N. Y. Med. J. 83: 657, 1906. (8) Renault, J., and Martingay: Bull. et mém. Soc. méd. d. hôp. de Paris 28: 344, 1911. (9) Miller, N. F.: J. Mich. S. M. S. 23: 58, 1924. (10) Foulkrod, C.: Am. J. Obst. & Gynec. 5: 329, 1923. (11) Hornung, R., and Creutzfeldt, H. G.: Deutsche med. Wchnschr. 56: 1470, 1930. (12) Hornung, R.: Ztschr. f. Geburtsh. u. Gynäk. 97: 546, 1930. (13) DeLee, J. B.: Principles and Practice of Obstetrics, ed. 5, Philadelphia, 1928, Saunders, p. 110. (14) Drinker, P., and Shaw: J. Clinic. Invest. 7: 229, 1929. (15) Draper, G.: Nelson Loose-Leaf Med. 2: 75, 1920. (16) Amoss, H. L.: Practice of Med., Tice 5: 336, 1923. (17) Peabody, F.: The Oxford Med. 1: 115, 1927. (18) Fishberg, A. M.: Hypertension and Nephritis, ed. 2, Philadelphia, 1931, Lea & Febiger, p. 271. (19) Carr, W. L.: Arch. Ped. 34: 591, 1917. (20) Blanton, W. B.: J. Med. Rev. 36: 1, 1917. (21) Larkin, J. H.: Arch. Ped. 34: 601, 1917. (22) Frauenthal and Manning: A Manual of Infantile Paralysis, London, 1914, Davis. (23) Stander, H. S.: Medicine 8: 1, 1929. (24) Fernandez, V.: Semana Med.,

Buenos Aires 19: 693, 1912. (25) Mercier et al.: Bull. Acad. de Med., Paris 85: 625, 1921. (26) Marinesco: Rev. Neurol. 37: 1055, 1921. (27) Jorge, R.: Paris Med. 39: 453, 1921. (28) Levaditi et al.: Compt. rend. Soc. de biol. 84: 957, 1921. (29) Paterson, D., and Carmichael, E.: Brain 47: 207, 1924. (30) Virchow: Virchow's Arch. f. Path. Anat. 38: 129, 1867. (31) Kramer, G. B., and Wright, W.: J. A. M. A. 71: 729, 1913. (32) Draper, G.: Acute Poliomyelitis, Philadelphia, 1917. (33) Clark, P. F., Amoss, H. L., and Fraser, F. R.: J. Exper. Med. 19: 223, 1914. (34) Flexner, S., and Amoss, H. L.: J. Exper. Med. 20: 249, 1914. (35) Amoss, H. L.: Practice of Med., Tice 5: 336, 1923. (36) Levaditi, G., and Willemin, L.: Ann. de l'Inst. Pasteur 46: 223, 1931. (37) McCarthy, J. D.: Rep. Henry Phipps Ins. 5: 437, 1907-8. (38) Batten, F. S.: Brain 33: 149, 1910. (39) Potts, C. S.: Arch. Neurol. & Psych. 21: 288, 1929. (40) Aycock, W. L., and Kramer, S. D.: J. Exper. Med. 52: 457, 1930. (41) MacAusland, W. R.: Poliomyelitis, Philadelphia, 1927, Lea & Febiger, 213. (42) DeLee, J. B.: Principles and Practice of Obstetrics, ed. 5, Philadelphia, 1928, Saunders, 717. (43) Williams, J. W.: Obstetrics, ed. 5, New York, 1923, Appleton, p. 895. (44) Lacomme, Ravina and Philouze: Bull. de Soc. d'Obst. et Gynec. 17: 508, 1928. (45) MacAusland, W. R.: Poliomyelitis, Philadelphia, 1927, Lea & Febiger, 221. Ollivier: Traite de la Moelle Epinere, ed. 2, 784, 1827. (47) Nasse, F., and H.: Untersuchungen zur Phys. u. Path., Bonn 2: 268, 1935. (48) Routh, A.: Obst. Trans., London 39: 191, 1897. (49) Benicke, F.: Ztschr. f. Geburtsh. u. Gynäk. 1: 28, 1874. (50) Scanzoni: Lehr. der Gerburt 3: 500, 1851. (51) Paget: Quoted 1. 25, 1874. (50) Scanson: Lehr. der Gerburt 3. 300, 1831. (51) Payer: Quoted by Bruce. (52) Bruce, Alex: Scot. Med. & Surg. J. 11: 107, 1902. (53) Good, F. L.: J. A. M. A. 83: 416, 1924. (54) Renz: Krankeiten des Ruckenmarks in der Schwangerschaft, Wiesbaden, 1886. (55) Windscheid, F.: Arch. f. Gynäk. 72: 361, 1904. (56) Meyer, C.: Zentralbl. f. Gynäk. 44: 238. (57) Taylor, E. W.: J. Nerv. & Ment. Dis. 33: 583, 1906. (58) Elkins, D. C.: J. A. M. A. 78: 25, 1920. (50) Rehimson, F. D. Prop. Payel. Sca. Med. 32: 21, 1918, 19. (60) Rec. J. Nerv. & Ment. Dis. 33: 383, 1990. (38) Etkins, D. C.: J. A. M. A. 76: 20, 1820. (59) Robinson, F. D.: Proc. Royal Soc. Med. 3: 22, 1918-19. (60) Pou, R. E.: Am. J. Obst. 69: 1052, 1914. (61) Heinz, H.: Boston M. & S. J. 197: 714, 1927. (62) Brachet: Recherches, ed. 2, Paris, 1837, pp. 299, 313. (63) Lewis, Dean: J. A. M. A. 83: 418, 1924. (64) DeLee, J. B.: Principles and Practice of Obstetrics, ed. 5, Philadelphia, 1928, Saunders, 79. (65) Rudolph, L., and Ivy, A.: Am. J. Obst. & Gynec. 21: 65, 1931. (66) Aycock, W. L., and Luther, E. H.: I. Pray. Med. 2: 102, 1929. (67) Luther, E. H.: Boston M. & S. J. 197: 187, A.: AM. J. OBST. & GYNEC. 21: 65, 1931. (66) Aycock, W. L., and Luther, E. H.: J. Prev. Med. 3: 103, 1929. (67) Luther, E. H.: Boston M. & S. J. 197: 1175, 1927. (68) Aycock, W. L., and Luther, E. H.: J. A. M. A. 91: 387, 1928. (69) Aycock, W. L.: Bull. N. Y. Acad. Med. 4: 1150, 1928. (70) Jungeblut, C. W., and Khorazo, D.: J. Immunol. 19: 283, 1930. (71) Fink, K.: Med. Welt. 1: 1593, 1927. (72) Acosta-Sison, H.: J. Philippine Islands M. A. 8: 230, 1928. (73) Chlopicki and Stepowski: Polska gaz. lek. 8: 633, 1929. (74) Ledoux, E.: Presse Chiopicki and Stepowski: Polska gaz. 1ek. 8: 633, 1929. (74) Ledoux, E.: Presse méd. 37: 516, 1929. (75) Sage, E.: To be reported. (76) Cawer, P.: Ztschr. f. Geburtsh. u. Gynäk. 97: 539, 1929. (77) Creutzfeldt, H. G.: Ztschr. f. Geburtsh. u. Gynäk. 97: 553, 1929. (78) Gueissaz E.: Gynec. et obst. 11: 446, 1925. (79) Krupp, S.: Zentralbl. f. Gynäk. 43: 915, 1919. (80) Pakozdy, K.: Deutsche med. Wehnschr. 55: 1509, 1929. (81) Aycock, W. L., Luther, E. H., and Kramer, S. D.: J. A. M. A. 92: 385, 1929.

420 SOUTH TWENTY-FIFTH AVENUE.