

PUERPERAL HEMIPLEGIA

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SINCE 1904, when Rudolph von Hösslin, a German neurologist, published a monumental study of the various puerperal paralyses, in which he reviewed, among other types, 146 cases of cerebral palsy, scant attention has been paid puerperal hemiplegia either in obstetric or in neurologic literature. When mentioned in textbooks it is usually dismissed briefly as one of the rare complications of eclampsia. While the condition is avowedly uncommon, the number of cases that Hösslin was able to collect, as well as the evidence afforded by the seven cases in this report, indicate that it occurs with sufficient frequency to merit careful consideration, particularly in the matters of etiology and prognosis.

Puerperal hemiplegia is sufficiently rare to render statistics of its incidence scanty, and when available, conflicting. Thus, Villa observed only two examples of the condition in twenty-seven years among 10,000 deliveries, while Immelmann in the Frauenklinik of Berlin saw eight cases within two years. Hösslin's collection of 146 cases in 1904 suggests that the condition is not uncommon, but this figure probably approximates very closely all the recorded cases prior to that date; it moreover includes a group of cases, which, while central in origin, were not hemiplegic, namely, cases 1 to 27 (hysteria), and 28 to 34 (myasthenia gravis). Talley and Ashton in 1925 were able to collect only seventeen cases recorded since 1896 but excluded from their list all cases associated with eclampsia or cerebral hemorrhage. The evidence at hand would seem to indicate that puerperal hemiplegia is considerably more common than are peripheral maternal palsies due to birth trauma. In Charpentiere's study embracing 114 cases of various puerperal paralyses, he listed fifty-seven as hemiplegic and only twelve as traumatic (mostly due to forceps pressure on nerve roots). Hösslin included eighty-one cases due to trauma (forceps, breech extraction and prolonged labor) as against 112 due to spontaneous vascular accidents in the brain. Our records show similar percentages; during the four year period in which we observed seven cases of central puerperal palsy, we saw only one of local, traumatic origin, a difficult forceps case that was delivered elsewhere.

The immediate causes of puerperal hemiplegia are of course those of apoplexy in general, namely, (1) intracranial hemorrhage from rupture of a blood vessel (hemorrhagic apoplexy), and (2) acute

cerebral softening from embolism or thrombosis (embolic or thrombotic apoplexy). It has long been known that neither of these causal agents is primary, the former being secondary usually to arterial changes, nephritis or syphilis and the latter to infection, circulatory stasis or anemia. It will be noted in the course of this paper that, with the exception of syphilis, any one or all of these contributory elements may be furnished by the common complications of pregnancy and labor (infection, toxemia, hemorrhage).

PUERPERAL HEMIPLEGIA FROM CEREBRAL HEMORRHAGE

Brain hemorrhage, incident to the toxemias of late pregnancy, has long been recognized as a common cause of puerperal hemiplegia and is probably responsible for most cases occurring in close conjunction with labor. This relationship is substantiated not only by numerous autopsy observations on fatal cases of hemiplegia but also by the high incidence of cerebral changes in patients dying of eclampsia. Thus Prutz noted edema in 42 per cent, hyperemia in 35 per cent, and apoplexy in 13 per cent, while the brain was apparently normal in only 10 per cent of his cases. Quite recently Gammeltoft made a careful study of the brains of twenty-four fatal cases of eclampsia and found in nine, hemorrhages of sufficient size to be of significance in the lethal issue; in four others there was edema with small superficial hemorrhages. While the rôle of brain hemorrhage in these cases seems clear, the exact mechanism by which the toxemias produce this phenomenon is not so obvious. Since normal blood vessels do not rupture without external trauma, the simple factors of elevated blood pressure and the bearing-down efforts incident to labor are not sufficient to account for it, and in order to arrive at a plausible explanation, it is necessary to postulate a third factor, namely, structural damage to the vessel walls. That such changes do occur is in accord with Zangemeister's theory of eclampsia and as well with the recent findings of several Continental investigators. For instance, Jaffe reports that in cases of death from eclampsia the cerebral vessels show extensive changes, varying from slight swelling and poor staining nuclei to an actual hyalinization of the vessel wall with disappearance of the nuclei. It is his opinion that the media is first involved, later the intima, and he is in agreement with Hinselmann in concluding that the injury to the vessel walls is due to spasm of the arterioles which results finally in necrosis of the smaller branches. In the opinion of Domagk, endothelial changes play a major rôle in eclampsia and are responsible, directly or indirectly, for a great part of the clinical picture; pulmonary edema, so frequently met in eclampsia, is particularly mentioned as being due to previous capillary injury; the well-known liver changes are interpreted in the same light. If these views may be accepted, they explain on a very definite basis the occasional

occurrence of cerebral hemorrhage in the toxemias of pregnancy. Conversely, it may be noted, cases of puerperal hemiplegia from brain hemorrhage apparently constitute a very convincing substantiation of the important rôle that vessel damage may play in eclampsia and the other toxemias of late pregnancy.

Puerperal hemiplegia from cerebral hemorrhage is characterized clinically by its tendency to occur during or shortly after labor, by its suddenness of onset and by the coexistence of a toxemia of pregnancy. The paralysis usually attains its full extent within a few hours. The condition appears to be more common in the latter years of the child-bearing period. Among the cases of hemiplegia due to brain hemorrhage that were reviewed by Hösslin, the majority were over thirty years of age and seven were forty or over. Of our two cases of this type, one was twenty-nine, the other forty-four.

CASE 1.—A married Chinese primigravida, age twenty-eight, at term and in labor, was admitted on the morning of July 4, 1923. She had not had prenatal supervision, but gave no history of toxic symptoms and showed no edema. B.P. 145/90. Labor appeared to progress normally until at 10 P.M., when the cervix was almost fully dilated, she suffered a typical eclamptic convulsion followed by coma. As she regained semiconsciousness, it was discovered that the right arm and leg were paralyzed. Catheterized urine showed albumin plus 4, and hyaline and granular casts; B.P. 150/90. Labor was terminated with low forceps; child living and well. There were no further convulsions.

The patient remained in semicoma for 5 days. There was no active motion of the right arm or leg; tendon reflexes on right were absent. The eyes were deviated to the left. There was dull response to pin prick on right side of body, prompt response to stimulation on left. Lumbar puncture: 5 c.c. of spinal fluid under increased pressure were withdrawn, pink in color with well mixed red blood cells. Retinal examination revealed papilloedema, bilateral. Blood chemical studies showed definite nitrogen retention: N.P.N. 46 (mg. per 100 c.c.), urea N. 30, uric acid 9, creatinine 1.5, CO₂ combining power 53 per cent. Blood and spinal Wassermanns negative. Blood pressure remained the same; the urinary albumen decreased. Temp. elevated (39.5 on July 6 and 7). Pulse 100-120.

On July 9, the fifth day postpartum, consciousness gradually returned, but with it the evidence of complete aphasia. The mouth muscles, however, could be moved sufficiently to suck fluids through a tube. The paresis of the ocular muscles had disappeared.

During the ensuing weeks the following changes transpired: the aphasia began to improve 20 days after delivery and on discharge (46 days postpartum) the patient could speak intelligibly but not distinctly. All facial paralysis had disappeared. Voluntary movements of the right leg started 25 days after delivery and when the patient was discharged the leg could be used for walking, but there was a decided limp. The right arm remained parietic. The urinary albumen disappeared until only a "trace" was present on dismissal; the blood pressure gradually fell to 135/85; the nitrogen retention decreased only slightly (N.P.N. 42). The retina returned to normal.

The patient has been watched carefully during the past 4 years and the following significant events have occurred: October, 1923 (4 months p.p.) attack of Jacksonian epilepsy, involving mostly right side and sufficiently severe to throw patient to floor dislocating left shoulder. Similar attacks in December, 1923, January, 1924

and October, 1924. Various manifestations of slight mental deterioration appeared: irritability, forgetfulness, etc.; these persisted for about a year and then subsided. The blood pressure returned entirely to normal and the urinary albumen completely disappeared. In 1927 the patient again became pregnant and supra-vaginal hysterectomy was done at the 3rd month for purposes of therapeutic abortion and permanent sterilization.

Four and one-half years after delivery the right arm remains sufficiently paralyzed to be of no practical use, in spite of prolonged physiotherapy. There is still a slight loss of function of the right leg. Mentality normal; speech shows detectable "slur." No convulsive seizures during past two years.

Final Diagnosis: Hemorrhage of middle cerebral artery into internal capsule incident to toxemia of pregnancy.

CASE 2.—A married Chinese woman, age forty-four, para ix, seven months' pregnant, was admitted on the morning of September 13, 1926 in deep coma. She was said to have had increasing edema of feet, hands and face for twenty days; during the previous four days, she had suffered blurring of vision, headaches and on the evening before, distinct lethargy. At 6 A.M. on the morning of admission there was said to have been a convulsion of the upper extremities followed by unconsciousness. No history of previous nephritis.

Examination on admission: Patient comatose with labored, stertorous respiration; marked edema of ankles, hands and face. B.P. 180/110. Catheterized urine: Albumen plus 4, many granular casts. Blood chemical findings: N.P.N. 35 (mg. per 100 c.c.), uric acid 5.7, creatinine 1.5, CO₂ combining power 54 per cent. Blood Wassermann negative. Retinal examination revealed marked bilateral papilloedema with multiple hemorrhages.

There were no convulsions after admission, but the coma continued. Patient was treated with glucose intravenously and insulin. During the day she went into labor and delivered at 10 P.M. a stillborn 1254 gram fetus. (Fetal heart had not been heard.)

The patient remained in semicoma. At 9 P.M. on September 14th, twenty-three hours after delivery, the patient suddenly showed marked rigidity of the upper extremities and jaws associated with deepening of the coma, rapid pulse and involuntary passing of urine and stools. The following morning when consciousness had improved, it was discovered that the left upper limb showed flaccid paralysis; the left lower limb was moved voluntarily but was weak. Tendon reflexes present and about equal on both sides; abortive ankle clonus on left. Sensation present on both sides. Spinal puncture: fluid clear and transparent; pressure 190 mm.; cell count 1 or 2. B.P. had risen to 220/120.

During the ensuing two weeks the patient's neurologic symptoms improved markedly and on discharge, 19 days after delivery, she had full use of her left leg and could move the left arm voluntarily; the latter, however, was distinctly weak. Renal function tests showed definite kidney damage and on discharge the urinary albumen was still 1 gram per liter and the B.P. 150/100.

One year after delivery the left hand is still weak. B.P. 205/120. Urinary albumen plus 2; a few hyaline casts.

Final Diagnosis: Cerebral hemorrhage incident to nephritic toxemia of pregnancy.

Both the immediate and ultimate prognosis of puerperal hemiplegia following brain hemorrhage is grave. Of the forty cases summarized by Hösslin, twenty-nine died, the majority within a few days. Patients who survive the hemiplegia may later succumb to an underlying

nephritis. A very definite relationship seems to exist between the severity of an attack and its proximity to labor, cases of cerebral hemorrhage that occur a few days after delivery tending to be more benign than the intrapartum variety. Of sixteen cases cited by Hösslin in which the brain hemorrhage took place during or immediately after parturition, all the patients died; while of twenty cases in which the seizure occurred several days postpartum, over half survived. This generalization applies not only to the immediate prognosis for life, but also to the ultimate prognosis of the paralysis. The latter point is borne out by the histories of Cases 1 and 2; the puerpera in Case 2, in which the seizure occurred twenty-three hours postpartum, recovered more quickly and more completely than the patient in Case 1, although the former was older and had more definite signs of a permanent nephritis. Deep and prolonged coma, absence of reflexes, marked elevation of temperature and other evidences of an extensive hemorrhage, point of course to a lethal issue; in the event the patient survives, such signs, particularly if aphasia also occurs, forecast inevitably a certain degree of permanent paralysis. The ultimate disability, however, is never so great as the initial palsy. Rapid improvement may be expected for about three months and then a less marked progress for a year; as in other forms of apoplexy the aphasia, the face, leg and arm paralyzes clear up in the order named. Few cases escape a certain degree of finger or hand disability. Attacks of Jacksonian epilepsy and varying degrees of mental impairment, as evidenced in Case 1, may be expected in patients who have suffered extensive hemorrhages with resultant softening of larger or smaller areas of brain tissue.

Concerning the prognosis for future pregnancies after this type of puerperal hemiplegia, scant data are available. Immelmann has reported a case in which hemiplegia, incident to eclampsia (?), occurred in two successive pregnancies, the second seizure being fatal; autopsy showed massive hemorrhage into both ventricles. It will be noted that both our cases gave clear evidence of renal damage, the first by persistent, prolonged nitrogen retention and the latter by elevated blood pressure and urinary albumin a year after delivery. While we have no statistics to support such a view, we feel that the majority of cases of puerperal cerebral hemorrhage are likewise consequent to a toxemia that is essentially nephritic and that, on this ground alone, therapeutic abortion with permanent sterilization is probably justifiable.

PUERPERAL HEMIPLEGIA FROM CEREBRAL THROMBOPHLEBITIS

While the earlier writers considered brain hemorrhage, incident to eclampsia and nephritis, as the most common cause of puerperal hemiplegia, our present knowledge points rather to cerebral thrombo-

phlebitis as the most frequent etiologic agent. Talley and Ashton, in their recent study of the centric puerperal palsies, corroborate this view and recall that in puerperal infection, however mild, all the elements necessary to thrombus formation are present—bacteria to injure the intima, and sluggish circulation to favor the deposition of blood platelets on the necrotic wall, a change which initiates the processes leading to clotting. Two other factors, biochemical in nature, augment this tendency, namely, the marked increase during the puerperium of plasma fibrin and the correlated acceleration in the sedimentation rate of the red blood corpuscles. Fibrin increases gradually during pregnancy until in the first week of the puerperium, it reaches 0.45 per cent, whereas readings in normal nonpregnant women average 0.31 per cent (Plass). In puerperal sepsis the figures are still higher and in a series of plasma fibrin determinations we have been making on such cases in this clinic, values of 0.7 and 0.8 per cent are not uncommon. Closely paralleling the plasma fibrin changes, the sedimentation rate of the red cells increases rapidly during gestation until on the third or fourth day of the puerperium it varies between thirty and forty minutes (Linzenmeier method); in infected puerpera, as shown in a previous communication by the writer, the rate is still further accelerated, occasionally reaching ten minutes. The importance of these two changes, as contributory factors to thrombus formation, seems obvious.

Puerperal cerebral thrombophlebitis is probably always mycotic in origin, secondary to infectious thrombophlebitis in the uterine, iliac, or femoral veins. It is important, however, particularly from the obstetrician's viewpoint, to recall that the pelvic infection may be either frank or concealed. Of singular interest in this connection is the minutely studied case of Hunt's: A primipara, twenty-one years old, on the twentieth day after an apparently normal delivery and puerperium, was seized with severe headaches, vomiting and general convulsions. The convulsive seizures continued and were both general and Jacksonian in type. Paralysis of the right side of the face, right arm and motor aphasia supervened. The patient gradually sank into a stupor and succumbed on the tenth day of the disease. Autopsy showed extensive thrombosis of the dural sinuses, the superior longitudinal, lateral and petrosal sinuses; also thrombosis of the cerebral veins with extensive softening of the cerebral cortex. *The uterus was small, firm and clean. The appendages were normal.* Almost identical cases have been reported by Zangemeister and Collier. In certain other instances the hemiplegia may occur before the pelvic phlebitis can be demonstrated, as in a case of Talley's in which the apoplectic seizure preceded by three days the detectable pelvic phlebitis.

Postpartum cerebral thrombosis is characterized clinically by, (1) tardiness of onset, the seizure occurring usually five to twenty days

postpartum, sometimes later; (2) the presence of prodromata—headache, numbness in the fingers, hands, or face, and difficulty in speech; (3) slowness of onset, the hemiplegia developing gradually, taking several hours or days, and often coming on with remissions; and (4) the persistence of a slight, unexplained elevation of temperature after delivery with a gradual, progressive, prolonged increase in pulse rate. But to decide in a given case that puerperal hemiplegia is due to cerebral thrombophlebitis, embolism, or hemorrhage, is difficult if not sometimes impossible, and when the case does not come to autopsy, the diagnosis must usually be held as largely presumptive.

CASE 3.—A married Chinese woman, age 29, para v, eight months' pregnant, was admitted on October 5, 1926, on account of increasing edema of the extremities and face of a month's duration, associated with headache, vertigo and occasional blurring of vision. B.P. 155/125. Urinary albumin 0.1 per cent; no casts. Urinary output low, about 400 c.c. daily. Blood chemistry normal. Fundi normal. Wassermann negative. Despite usual therapeutic measures, the edema and albumen increased, the CO₂ combining power dropping from 50 to 38 per cent. On October 12 she went into labor and delivered spontaneously a 2775 grams still-born fetus.

The patient appeared to improve decidedly for six days after delivery, the blood pressure returning to normal, the urinary output increasing and the albumen disappearing. The pulse remained persistently rapid, however, varying between 110 and 120; there was no elevation of temperature. On the morning of the seventh day she complained of headache over the right half of the head and numbness of the left arm. At noon she had obvious difficulty in speaking. At 4 P.M. she complained of numbness of the right arm. Examination showed slight weakness of lower left face and both limbs; faint perception of left face and upper left limb was much reduced. At 3:30 A.M. on the eighth day she was heard to emit a high-pitched scream following which she went into a general convulsion which terminated in coma, the latter lasting ten minutes. Upon return to consciousness there was marked weakness of both handgrips, particularly the right; no aphasia. At 7 A.M. another convulsion, general and clonic in type; at 9 A.M. a third convulsion, followed by deep coma. Upon regaining consciousness there was complete paralysis of the right arm and distinct weakness of the right leg. She remained stuporous for 24 hours. Temp. 39, pulse 136.

For the remainder of the patient's stay in the hospital, there persisted occasional attacks of Jacksonian epilepsy involving the right upper limb and face and lasting ten to twenty minutes. On discharge, fifty-one days after delivery, there remained a slight loss of power in the right leg and a moderate loss of function of the right hand.

The report one year after delivery is of multiple attacks of Jacksonian epilepsy, six during the previous two months. Still complains of numbness and weakness of the right hand. Thenar and hypothenar eminences of the right hand demonstrably atrophied. Personality "hysterical."

Final Diagnosis: Cerebral thrombophlebitis, probably secondary to an unrecognized puerperal infection.

The toxemia of pregnancy was such an outstanding feature of Case 3, particularly prior to delivery, that the question arises as to its possible relationship to the later cerebral thrombosis. While the obscure state of our knowledge concerning the toxemias of pregnancy

precludes definite answer of this inquiry, the following considerations seem pertinent. In several cases recorded by others a toxemia of pregnancy has preceded the development of cerebral thrombophlebitis. For instance, Zangemeister has recently reported the case of a twenty-five-year-old secundigravida in whom forceps delivery had been done for threatened eclampsia; on the thirteenth day postpartum, when elevated blood pressure, albuminuria and other signs of toxemia had disappeared, stupor and left-sided convulsions set in. Death ensued within a few hours. At autopsy there was complete thrombosis of the longitudinal and transverse sinuses. Zangemeister feels that the toxemias of pregnancy produce certain changes in the intima of the blood vessels which predispose to thrombosis. Enough has already been said concerning the likelihood of vessel damage in such conditions to make it conceivable that the injured areas might, in the presence of bacteremia, serve as foci for thrombus formation.

The prognosis of puerperal cerebral thrombosis is not only grave but extremely uncertain. In the case of brain hemorrhage the paralysis is usually complete soon after the onset so that a rough estimate of the extent of the final tissue damage may be attempted soon after the first seizure, but in thrombosis, where one is dealing with a gradually progressive process, it is difficult, if not impossible, to ascertain to what limits it will extend. The severity of the attack seems to bear no relationship either to the time of onset or to the degree of the primary infection. In the above case, for instance, the pelvic infection appeared negligible, whereas in Hunt's fatal case even autopsy study failed to reveal changes in the uterus or pelvis. The prognosis of the chronic stage of puerperal cerebral thrombophlebitis, is much the same as that of hemorrhage and depends altogether on the amount and location of the brain tissue damage, as evidenced by neurologic symptoms and signs.

PUERPERAL HEMIPLEGIA FROM CEREBRAL EMBOLISM

That endocarditis, acute or recurrent, may lead to puerperal palsy from brain embolism is obvious, but this occurrence probably represents a coincidental event rather than a complication intrinsically related to the puerperal state. Other interpretations of the phenomenon seem hardly in keeping, at any rate, with the well-known frequency of chronic endocarditis in pregnancy on the one hand and with the rarity of puerperal cerebral embolism on the other. Recently, Corwin, Herrick, Valentine and Wilson tabulated the results of a careful personal study of 172 cases of chronic cardiac valvular disease complicating pregnancy and reached the conclusion that "liability to embolism, so frequent in younger women with mitral stenosis, seems not to be increased by pregnancy and labor."

Interest in puerperal cerebral embolism, however, has centered not

so much about the type of case just mentioned, as around a group of cases in which emboli have apparently reached the brain from thrombotic foci in the pelvis, the result of puerperal sepsis. The cause of the interest has lain largely in the problem of how such detached fragments of pelvic or femoral thrombi can pass the pulmonary capillaries, a question that has still not received adequate answer, but upon which several plausible suppositions have been advanced. Among these may be mentioned: (1) "clumps of bacteria" may circulate with the blood corpuscles and plug small terminal arteries in the brain; (2) primary localization of the infection may occur in the lungs or at some site on the systemic side of the circulation, whence new metastases may pass to the brain; (3) emboli may pass through a patent foramen ovale. In connection with temporary pulmonary localization it will be noticed that the patient in Case 5 showed definite lung signs several days prior to her seizure. The third theory awaits autopsy confirmation. Whatever the exact mechanism of the metastasis, its occurrence is attested by numerous necropsy studies and its actuality must be conceded.

Puerperal hemiplegia from cerebral embolism is characterized clinically by its sudden onset, by the coexistence of valvular heart disease or thrombotic processes in the pelvis or thigh, and by the slighter degree and shorter duration of the coma. Many patients are in the earlier years of the child-bearing period. Cases secondary to pelvic foci naturally occur later in the puerperium when puerperal septic processes have had time to assert themselves.

CASE 4.—A married Russian woman, age 28, was admitted October 3, 1927, complaining of paralysis of the left arm and leg of thirteen months' duration. At 15 she had had acute rheumatic fever being in bed a month with high temperature and painful swelling of one knee. Other past history irrelevant.

One year prior to admission, when in the last month of her fourth pregnancy (about twenty days before the expected confinement), the patient was suddenly seized with paralysis of the left face and the left upper and lower limbs with partial loss of consciousness. For two weeks she was aphonic and recognized no one. At the end of this period she underwent an easy, spontaneous delivery of a normal living child. After delivery consciousness returned, but the face paralysis persisted for three months and the arm and leg paralysis to the time of admission. For the previous two months she had been noting swelling of the ankles, most marked in the evening.

Examination revealed the following positive findings: Heart definitely enlarged to the left with palpable presystolic thrill; loud, rough presystolic murmur at apex and base. Spleen palpable, liver enlarged. Fingers distinctly clubbed. There was moderate muscular atrophy of left upper and lower limbs. Left upper limb spastic with power diminished, especially in extensors of hand. Left lower limb also spastic with slight impairment of motion. Tendon reflexes were much exaggerated on the left side with patellar and ankle clonus present. Wassermann negative.

Final Diagnosis: Embolism of middle cerebral artery, rheumatic cardiovascular disease with mitral stenosis and insufficiency.

CASE 5.—A married Chinese woman, age 40, a secundigravida, was admitted on the morning of September 8, 1926, on account of prolonged and ineffectual labor. The patient's one previous pregnancy had been terminated by an operative delivery done by native "physicians" in which the baby, born dead, had been severely mutilated and the maternal soft parts extensively damaged. Upon examination dense cicatricial atresia of the vagina was found barely permitting the passage of a 16 French catheter. Cesarean section was done shortly after admittance.

The patient ran a febrile course following operation, the temperature reaching 38 daily. On September 12 she developed a cough with thick purulent sputum. The lungs showed slight dullness at left base with diminished breath sounds and occasional crepitant râles. The condition was thought to be an acute respiratory infection. On September 20 (the twelfth day) at 1:20 A.M. when the baby was brought to breast, the patient was found semiconscious. She recognized neither the child nor the nurse and could not talk. She was incontinent of urine and feces. The following morning her mental condition remained the same; the temperature had reached 39.8. Examination showed marked weakness of right lower face and of the right extremities. Voluntary motion of the right arm was distinctly impaired, of the right leg slightly so. Tendon reflexes present and about the same on both sides. The semicomatose condition persisted for a week, at the end of which period, control of the sphincters returned, consciousness became clearer and the paralysis gradually subsided. Upon discharge on November 8, (30 days after delivery) the patient seemed free of hemiplegic symptoms.

Condition one year after delivery: No neurologic symptoms or signs.

Final Diagnosis: Embolism of middle cerebral artery from thrombotic focus in pelvis.

The prognosis of puerperal embolism is somewhat better than that of hemorrhage and thrombosis and the sixteen cases secondary to endocarditis which are reviewed by Hösslin include only two with fatal termination. When puerperal thrombosis is the primary issue, the pelvic process rather than the cerebral may be the cause of death. In many instances the hemiplegia is transitory, lasting only a few hours or days and may even be mistaken for hysteria.

UNCLASSIFIED CASES

It has not seemed advisable to classify the following cases since neither was seen until long after the paralysis had been established and the histories, as well as the findings, were inconclusive.

CASE 6.—A married Chinese woman, age 27, was admitted on February 21, 1927, complaining of stiffness of and inability to use the right arm and leg. One year prior to admission the patient delivered spontaneously a living child; the last three months of the pregnancy were said to have been complicated by headache, vertigo and marked edema of the face and extremities. On the fifth day of the puerperium, the patient became suddenly comatose (without convulsions) and remained so for three hours. Upon regaining consciousness, she found herself unable to speak and unable to move the right arm and leg. Both the aphasia and the paralysis were practically complete for one month. Gradual improvement then ensued, particularly in the ability to speak. During the six months prior to admission, however, the condition had remained stationary.

Examination revealed marked spasticity of the right upper and lower limbs and complete loss of function of the right hand and fingers. Movement of the right

elbow, shoulder, toes, knee and hip were distinctly impaired. Gait spastic. Speech indistinct and slurred. No anatomic heart disease could be detected. Wassermann negative.

Impression: Possibly cerebral embolism.

CASE 7.—A married Chinese woman, age 29, was admitted on September 16, 1927, complaining of paralysis of the left upper and lower limbs, of sixteen months' duration. In January, 1926, she underwent an apparently normal delivery in the care of a native Chinese midwife. Following confinement she continued for three months to have "excessive vaginal bleeding with clots." She was said to have been very weak during this period but was not thought to have had fever. Toward the end of the three months the left face and the left upper and lower limbs became gradually paretic. There was no loss of consciousness, no convulsions and no speech defect. The patient was unable to walk until nine months after delivery and then awkwardly. A year after confinement she suffered a convulsive seizure lasting twenty minutes, probably Jacksonian epilepsy.

Examination showed grip of left hand very weak with complete loss of finger motion. Wrist, elbow and shoulder movements on left were markedly impaired; the left arm and fingers were kept flexed. The left lower limb was slightly spastic with considerable loss of power; the toes could not be moved. Tendon reflexes were exaggerated in the left upper and lower extremities. Gait spastic. Sensation to brush, touch and pin prick was decreased on the left side, including the face. Wassermann, negative.

Impression: Probably cerebral thrombophlebitis.

Therapeutic measures in puerperal hemiplegia unfortunately yield scant results and are limited to symptomatic treatment in the attack and physiotherapy during the chronic stage. Massage and active or passive motion are of course contraindicated if there is the slightest likelihood of embolic detachment. In some cases electrical stimulation seems to hasten the return of function, but the most important element in the chronic stage is probably time.

It is a pleasure, as well as a duty, for the writer to express his cordial thanks to Dr. Andrew H. Woods, Professor of Neurology, Peking Union Medical College, for his valuable advice throughout this study.

SUMMARY

Seven cases of puerperal hemiplegia were seen at the Peking Union Medical College Hospital within a period of four years. Six of the patients were Chinese women. Two cases were apparently due to cerebral hemorrhage, two to cerebral thrombophlebitis, and three to cerebral embolism.

Puerperal hemiplegia from brain hemorrhage usually comes on during or shortly after labor, probably always as the result of a co-existing toxemia of pregnancy. The mechanism of the hemorrhage is best explained on the ground that the toxemias of pregnancy produce structural damage to the vessel walls which become so weakened that the elevated blood pressure and the bearing-down efforts incident to labor cause rupture.

Puerperal hemiplegia from cerebral thrombosis, possibly the most common type of the condition, occurs most often in the second or third week of the puerperium. It is probably always secondary to a pelvic infection, although the latter may be so slight as to escape detection. In many instances the cerebral thrombosis is preceded by a toxemia of pregnancy and it is suggested that here, as in cerebral hemorrhage, vessel damage may play an important etiologic rôle. Certain changes in the colloidal state of the blood further augment the tendency to thrombosis in the puerperium.

Puerperal hemiplegia from cerebral embolism may be due either to detached cardiac vegetations or to emboli of pelvic origin. The former probably represents a coincidence not directly related to the puerperal state. The mechanism by which detached pelvic thrombi may reach the brain through the pulmonary capillaries is obscure, but numerous necropsy studies attest its occurrence and its actuality must be conceded.

Although the seven cases of this report all survived, the prognosis of puerperal hemiplegia is usually grave. Cerebral hemorrhage occurring during labor is particularly likely to prove fatal. Patients who survive the apoplectic seizure seldom escape a certain degree of permanent paralysis.

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