ECLAMPSIA WITHOUT CONVULSIONS.

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The disease which is now universally designated as eclampsia was described by the earliest medical authors, but its present apellation dates from comparatively modern times. The word itself derived from the Greek εκ, forth, and λαμπειν, to shine, was used in classical literature to describe a flash of lightning or the sudden beaming of the sun through the clouds. When first introduced into medical terminology the word eclampsia was not used in connection with the disease with which it is now associated, but was employed to denote a sudden rise in temperature. It was used in this sense by Hippocrates and throughout the Middle Ages.

According to Knapp and Kossman, the first instance in which the term was applied to convulsive seizures occurs in The Nosology of Boissier von Sauvage, published in 1760. Under the caption Eclampsia, the latter classified all non-periodic fits other than epileptic, but made a separate group of the convulsions occuring during pregnancy, which were designated as "Eclampsia parturientium." Gehler, a professor of physiology in Leipzig, adopted this term and made use of it in theses written in 1776 and 1777. For some time the use of the term remained purely local, but it gradually gained favor, so that fifty years later its use had become current throughout the continent.

The convulsive attacks, which finally gave the disease its enduring name, have always been its most prominent clinical feature, and this they remain. However, it is of prime importance to recognize that they are not the disease itself, but merely constitute one of its symptoms. Disregard of this fact leads to the diagnosis of eclampsia in all conditions in which convulsions occur during pregnancy. Should the pathologist accept without question such clinical diagnoses, his findings at autopsy would vary so greatly that it would be impossible to regard any lesion as specific; whereas, in truth, the pathological anatomy of eclampsia is most characteristic and offers the only means by which an unimpeachable diagnosis can be made.

Since the appearance of Schmorl's monograph in 1893, it has been generally conceded that the typical anatomical findings in eclampsia consist of degenerative changes in the kidney and myocardium, hæmorrhages into the brain and focal necroses in the liver. The last constitute the most important eclamptic lesion and would seem to be areas of infarction dependent upon thrombosis of the smaller portal vessels. The areas of necrosis may be either hæmorrhagic or anæmic in character, but always maintain a regular topographical disposition, appearing first in the portal spaces and invading the peripheral zone of the lobule. Schmorl's observations have been abundantly substantiated by Jürgens, Klebs, Pilliet, Bouffe de Saint Blaise, Prutz, Lubarsch, Pels Leusden, Bar, Williams, Jung, Chiari, Sandberg, and Lindfors and many others.

The changes in the liver are most characteristic, and Schmorl regards them as absolutely pathognomonic, having noted them in 70 out of 72 cases, and in the two negative instances there was total thrombosis of the portal vein. In a recent study of the liver in 500 autopsies at the Johns Hopkins Hospital, Opie noted such lesions only in eclampsia; while Williams has met with them so regularly at autopsy upon eclamptics that he considers that their presence justifies the diagnosis of the disease, even in the absence of a clinical history.

Unfortunately, we are not in possession of any clinical phenomenon in eclampsia which may be fairly said to point to the occurrence of this specific change in the liver. All the signs and symptoms, as we know them now, may occur in a number of other conditions. Thus, Lloyd has lately recorded an instance of uræmia during pregnancy in which the clinical picture was identical with that obtaining in typical eclampsia, yet the autopsy revealed a profound acute necrosis of both kidneys without any change in the other viscera. Likewise, acute yellow atrophy of the liver may be indistinguishable from eclampsia at the bedside, while occasionally it may be impossible to make a differential diagnosis between it and epilepsy or even hysteria.

Again the obstetrician should bear in mind the fact that the toxic action of certain drugs may simulate eclampsia, particularly as women who are illegitimately pregnant may take poison with suicidal intent. Knapp has met with cases of strychnine poisoning and Schild with intoxication from nitro-benzol which could not be distinguished from eclampsia by the symptomatology. Lead, phosphorus, carbolic acid, corrosive sublimate and mushroom poisoning also give rise to toxic manifestations which may be characterized by convulsions and coma.

The difficulty in making a positive clinical diagnosis of eclampsia is still further increased by the fact that the disease sometimes appears in an atypical form. Thus, the customary convulsive attacks, the very symptom which has given the disease its name, may be absent throughout its course. Two such cases have come under observation in the Obstetrical Department of the Johns Hopkins Hospital, and I am indebted to Dr. Williams for the opportunity of making this report. The diagnosis in each instance was obscure, until the characteristic hepatic changes were demonstrated at autopsy; and in view of their presence, it was evident that both cases were examples of the somewhat paradoxical condition—eclampsia without convulsions.

CASE I.—Obstetrical No. 909. K. B., negro, IV p., aged 27.

The patient applied for registration in the out-patient department April 25, 1901. She stated that her four previous pregnancies had been normal. Her labors and convalescence had always been free from any complication. During the present pregnancy she has been quite well.

May 2. She visited the dispensary to bring a specimen of urine previously requested. This showed 0.05 gm. of albumin to the liter, but contained no casts. There were no subjective symptoms.

June 4. She complained of swelling of the feet and legs, but had no headache, visual disturbance or other subjective symptoms. The urine presented a large amount of albumin without casts. She was advised to enter the hospital, but declined.

June 12. Delivered by the Out-patient Service at 10.30 p. m., after a normal labor of 18 hours. Child was a male, 50 cm. in length, and weighed 7¼ pounds. The mother's condition was good when left at midnight.

June 13. When the nurse called at 9 a. m. to pay her daily visit, she found the woman in a semi-conscious condition, with a normal temperature and a pulse of 46 to the minute. The family stated that no convulsion had occurred, and attached little importance to the drowsy condition of the patient, but after considerable delay, reluctantly consented to have her removed to the hospital.

Examination on admission to the hospital two hours later. The patient is a stout colored woman presenting marked œdema, especially of face, hands, and feet. She is semi-conscious and arouses slightly when questioned, yet cannot be made to answer. The breathing is stertorous. Pupils contracted. Temperature, 98.4°. Pulse, 64. Heart sounds clear. There are definite signs of fluid in both pleural cavities and in the abdomen. The urine, 520 cc. by catheter, is neutral in reaction and has a specific gravity of 1010. The albuminous precipitation is heavy. On microscopical examination, casts of varied type and red blood cells are found. Soon after entering the hospital 400 cc. of blood were taken from one of the veins of the arm and an equal amount of normal saline solution transfused. Following this a sweat bath was given. The patient did not respond; gradually became profoundly comatose and died, without having had a convulsion, at 4.45 p. m. the same day.

Autopsy (Path. No. 1749).—Permission was obtained for a post-mortem examination with great difficulty, so that it could not be made until the day after death. As an agonal infection with the gas bacillus had occurred, all the tissues and organs of the body presented an emphysematous appearance, which in a measure marred the anatomical findings. The autopsy was performed by Dr. W. G. MacCallum, who kindly placed the following notes at my disposal.

Anatomical diagnosis.—Puerperal uterus. Hydronephrosis and hydro-ureter. Chronic nephritis with hæmorrhage. Hæmorrhages into liver. Acute spleen tumor. Œdema of lungs. Distension of veins with gas. Emphysematous organs. Interstitial emphysema of lungs.

Body of a stoutly built negro woman, 165 cm. long. The abdomen is distended, and on being opened is found to contain blood-stained fluid. The peritoneal surfaces are smooth everywhere.

Both pleural cavities as well as the pericardial cavity contain bloody fluid.

Heart weighs 340 gm. Endocardium smooth. Valves are normal.

Both lungs present a smooth surface. No areas of consolidation are found.

Spleen weighs 230 gm. No macroscopic lesion noted.

Liver measures 25 x 18 x 9½ cm. Over almost the whole surface of the right lobe an extensive subserous hæmorrhage is seen. Smaller hæmorrhages are scattered over the remaining surface of the organ. On section, the lobules are extremely pale and opaque. The greater part of the right lobe shows conglomerate hæmorrhages throughout its substance. These obscure the lobulations. Small gray points can be seen among the fresh hæmorrhages and here the consistence is much firmer. These are evidently areas of liver tissue. Other hæmorrhages occur throughout the liver.

Kidneys. Left: Capsule strips off easily. On section, one notes hæmorrhages into the substance of the organ. The pelvis of the kidney as well as the ureter is distended. Right: Somewhat larger than left, but presents the same general appearance. The ureter is slightly distended, but much less so than on left side. No obstruction, other than the uterus, can be found blocking the ureters.

Stomach is distended with gas, but otherwise is normal.

Pancreas is emphysematous, but otherwise normal.

Brain: On opening the skull, no puerperal osteophytes are found.

The cortex is normal in appearance, except for an atrophic area behind the fissure of Rolando on the left side. (No notes were made as to the quantity of cerebro-spinal fluid. The condition of the brain substance or the condition of the lateral ventricles.)

The uterus rises 16 cm. above symphysis. Its surface is smooth and glistening.

Microscopical examination.—Liver: A large portion of the organ is made up of necrotic tissue in which the nuclei are indistinct or totally missing. The cell protoplasm stains deeply with eosin. Between the dead cells there is an extensive infiltration with blood.

Other sections show the liver cells in a better state of preservation as a whole, but numerous foci of degeneration and necrosis are noted. The fresher ones are accurately confined to the portal spaces with the bile duct approximately in their centers, while the adjacent blood vessels are thrombosed. A few of these areas are anæmic, but for the most part they are infiltrated by hæmorrhage.

Larger areas of necrosis predominate, involving all or parts of several lobules. They appear to have resulted from the fusion of smaller areas of infarction, or from a secondary extension from a single portal space. Practically everywhere the liver is infiltrated with blood.

Kidney: A general increase in connective tissue is apparent throughout the section.

The cells of the straight and particularly of the convoluted tubules are the seat of marked degeneration. The cell nuclei having disappeared, the cytoplasm is converted into hyaline material. Some of the glomeruli are contracted, and lie in what are apparently retention cysts. In other places they are swollen and completely fill the surrounding capsule.

Spleen: Pulp is engorged with blood. The vessels in the center of the Malpighian corpuscles contain thrombi and are surrounded by masses of hyaline material, frequently occupying half of the tuft. Under the high power these are seen to be made up of areas of agglutinated red blood corpuscles.

Heart: The myocardium presents a mild parenchymatous degeneration.

Lung: Blood vessels distended. Alveoli show œdematous changes, but do not contain any inflammatory exudate.

CASE II.—Obstetric No. 1722, L. C., white, primigravida, aged 33.

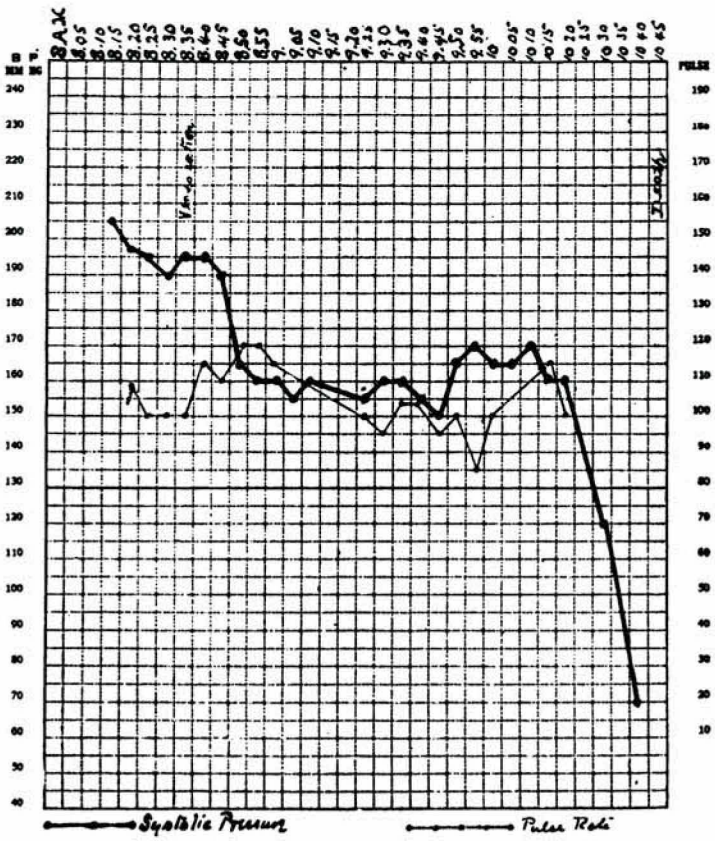
The history prior to admission was obtained from the patient's mother, an intelligent woman, whose statements gave the impression of accuracy and trustworthiness.

The patient has always been rather delicate. As a child she had diphtheritic croup and measles. Both these attacked her with more than ordinary severity. No other serious illness can be remembered. She frequently had "spells," which were usually associated with the mentsrual period, and in which she suffered from nausea, vomiting, headache, swelling of the feet and legs, and puffiness about the face.

The last menstruation occurred in August, 1903. Since then she has never felt well and has complained of a good deal of headache and difficulty in reading, on account of "seeing double." She is habitually constipated. Her whole body has been swollen for some weeks. The patient has never mentioned passing less urine than usual, nor any other peculiarity of micturition. A decided change for the worse in her condition occurred about the middle of February, when she "caught cold." Since then the headache has been more severe and the swelling has increased.

Two weeks ago the patient was threatened with premature labor, and had considrable bleeding, which ceased after a few days. She did not have abdominal pain on this occasion, or at any other time. The day before admission she was more uncomfortable than usual, but was not sick enough to go to bed. After retiring at the customary hour, she awakened her mother about 3 a. m., when she complained of "sick stomach" and was violently nauseated for half an hour, but did not vomit. Immediately afterwards she fell into a stupor and did not regain consciousness. The patient's husband, mother and father, who came to the hospital with her, all agreed in the statement that she had not had a convulsion of any kind.

Examination on admission.—March 12, 1904, 7.15 a. m. Patient is a stout woman, weighing about 200 pounds. She is in profound coma and all attempts to arouse her fail to elicit the slightest response. Face is cyanotic and swollen. Pupils are contracted. Conjunctival reflex is absent. Lips are quite blue and do not present any abrasion. Tongue is uninjured. There is a sweetish odor to the breath. Lungs are negative on percussion and auscultation. Respirations 20 to minute, are alternately shallow and deep, closely resembling the Cheyne-Stokes' type. Heart sounds are clear at apex and base. Pulse is full, of high tension, regular in force and rhythm. Systematic observations as to its rate and the systolic blood pressure (Riva Rocci apparatus) are given in the accompanying chart.



Abdomen is distended by the pregnant uterus which rises three fingers breadth above the umbilicus. There is dullness in both flanks. Feet and legs are markedly swollen and readily pit on slight pressure. Temperature by rectum is 99.2°. Vaginal touch shows the cervix long and snout-like. The external os is intact. The cervical canal is approximately 3 cm. in length.

Urinalysis.—Two hundred cubic centimeters of amber colored urine were obtained by catheterization. Microscopical examination showed an unusually large number of hyaline and granular casts, with many red blood cells and a few leucocytes.

The chemical analysis of the specimen was as follows: Reaction, acid; specific gravity, 1025; no sugar. Total nitrogen (Kjeldahl) = 2.66 grams; albumen (Esbach) = 2.5% (25 grams per liter); therefore, the non-albuminous nitrogen = 1.91 grams. Ammonia (Schlössing) = .16 gm. = 6.8%.

Nitrogen division.—Nitrogen precipitated by phosphotungstic acid = 1.31 grams.

Therefore,

Non-albuminous precipitate — N. =	.564 gm.	= 29.5%
Urea — N. =	1.189 "	= 62.2%
Amido acid — N. =	.141 "	= 7.3%

Treatment.—The patient was so ill on admission that an *accouchement forcé* was inadvisable. Induction of labor by a slow method with the simultaneous use of medicinal measures was thought to offer the only possible means of relieving her condition, though little if any hope was entertained of her recovery.

After stretching the internal os with a Goodell dilator until it had a diameter of 1 to 2 cm., a small rubber balloon was introduced into the uterus and inflated with normal salt solution, after which the cervix and vagina were packed with sterile gauze. Anæsthesia was not necessary during these precautions.

A vein was opened in the forearm and 500 cc. of blood allowed to escape, which was immediately replaced by a similar amount of 1% sodium bicarbonate solution given subcutaneously. A quantity of bile-stained fluid was washed out by gastric lavage, after which two ounces of Epsom salts and one drachm of sodium bicarbonate were left in the stomach. The patient was given a sweat bath at 9 a. m., but did not respond satisfactorily. Her condition gradually grew worse and she died at 10.45 a. m.

During the three and a half hours from the time of admission to the hospital until death she had no convulsions and was continually in profound coma.

Autopsy (Path. No. 2265) was performed four hours after death by Dr. W. G. MacCallum, whose notes were as follows:

Anatomical diagnosis.—Pregnancy, 7 months; chyliform ascites; generalized œdema; basal atalectasis of lung; focal necrosis in liver; erosion of mucosa of stomach; accessory spleen

Body of a very stout woman, 172 cm. in length.

On opening the peritoneal cavity, it is found to contain about two liters of an opalescent fluid. The peritoneum is smooth and glistening.

The pleural cavity contains a little of the same kind of fluid and its surfaces are smooth. The pericardium is free from excess of fluid, but all the tissues are œdematous.

Heart is not enlarged. Foramen ovale is open. All the valves are normal. The coronary arteries are not sclerotic.

Lungs: The left lung is everywhere crepitant, except in the lower and posterior portions where it is collapsed. The right lung presents a similar appearance.

Spleen weighs 220 gms. It is somewhat enlarged, and soft and flabby, but apparently normal on section. Adjacent to it are found two small accessory spleens.

Liver weighs 1590 gms., is remarkably firm and distinctly diminished in size, measuring 21 x 18 x 8.5 cm. Under the markedly thickened capsule numerous small, irregular hæmorrhages are seen upon the surface of the upper and lower parts of the right lobe.

On section inconspicuous focal areas of alteration in the liver tissue may be seen after careful inspection. They are opaque, yellow in appearance, and would seem about the size of three lobules. In general the lobulation is everywhere quite distinct. A few very small hæmorrhages are scattered throughout the liver substance.

Kidneys: The two organs are practically alike. The capsule strips off readily, leaving a smooth, rather pale, grey surface, on which the stellate veins stand out prominently. Fœtal lobulation is quite evident. On section the cortex is found to be 5 to 6 mm. in thickness. Its striations are quite straight. The labyrinthine portion is somewhat opaque. The straight tubules stand out definitely and are translucent. The glomeruli appear as prominent greyish dots.

Stomach is normal in appearance. Just below the pylorus there is a small erosion 4 mm. in diameter. The mucous membrane of the duodenum is œdematous. Pancreas is œdematous, but otherwise normal.

Thyroid is distinctly enlarged and measures 6 cm. in length and 3.5 cm. in width. In section, an increase in connective tissue is evident, otherwise there is nothing abnormal in the right lobe. In the left lobe there is a cyst containing a brown, colloid fluid. Parathyroids are apparently normal.

Œsophagus and trachea are normal. The gall ducts are normal.

Uterus measures 25 x 20 x 13 cm. and is normal in its external appearance. Cervix practically undilated and only admits tip of little finger. Canal intact. On opening the organ, the placenta is found attached to its anterior wall, and is separated from its attachment for a distance of 3 cm. from its lower edge. Beneath the placenta there is a blood clot 1 x 2 x 2.5 cm. and other clots are found between it and the internal os, the membranes having been dissected from the uterine wall. The partial separation would readily explain the bleeding from which the patient suffered during the last two weeks of life. Fœtus is a male, measures 24 cm. from vertex to sacrum and does not present any external abnormality.

Tubes and ovaries are normal. The left ovary contains the corpus luteum of pregnancy.

Microscopical examination.—Liver: Immediately around the portal vein a varying number of liver cells are necrotic and have been converted into a mass of hyaline material through which leucocytes are scattered. Sometimes these areas are infiltrated by hæmorrhage. The centers and middle zones of the lobules present a normal appearance, save that here and there foci occur in which the liver cells are swollen and almost transparent.

Spleen: Central portions of Malpighian bodies show fragmentation of the cells with formation of a coagulum. In the splenic pulp the vessels are sharply marked out. The fibrous tissue appears to be slightly increased.

Kidney: The glomeruli are normal. Epithelium of convoluted tubules appears somewhat ragged. The tubules contain some débris. There is an inflammatory exudate in the kidney.

Thyroid: Alveoli greatly enlarged, cells lining them are well preserved as a rule, though here and there they melt into hyaline masses with irregular outline, in which the nuclei are extremely small, deeply stained and shrunken; the colloid material is sometimes seen to extend between the cells.

Lungs: In some places the lungs show complete atalectasis, but for the most part they present no other changes than those seen in pulmonary œdema.

Thymus: Considerable remains are found in the form of long strands.

Bacteriology: Cultures from the various organs and the blood are negative.

The first record of a case in which eclamptic lesions were found in the liver at autopsy without a preceding clinical history of convulsive attacks is to be found in the Paris thesis of Bouffe de Saint Blaise in 1891. His patient was suffering from a profound toxæmia of pregnancy and was delivered by *accouchement forcé*. Death occurred a few days later from peritonitis. A similar instance was reported by Wendt in 1898, where the operation was immediately followed by a fatal post-partum hæmorrhage.

These cases were of considerable importance at the time in silencing the objections of Winkler and others, who urged that the hæmorrhages in the liver and elsewhere were the result of the eclamptic seizures and therefore not pathognomonic of the disease. They are also of great interest to us now, as they indicate that the characteristic organic changes in the liver may occur in pre-eclamptic toxæmia; but at the same time they cannot be placed in the same category with the two cases which I have just reported, since there is no evidence that convulsions might not have occurred had the patients not succumbed to complications incident to the mode of delivery.

In 1902, Schmorl placed on record three cases of eclampsia without convulsions, in which death occurred in coma, and autopsy revealed characteristic lesions. Subsequently two similar cases were reported by Meyer-Wirtz and Esch, respectively. These five cases are analogous to our own and supply a basis for comparison, as well as material for an analytical study of the condition. Labhardt, in a recent communication, incidentally mentions having met with a similar instance, but fails to give any clinical or pathological data.

CLINICAL COURSE.

The incidence of this type of eclampsia bears no relation to age, as the statistics indicate that women are subject to it throughout the childbearing period. A predisposition on the part of primigravidæ, and the onset of the condition in the second half of the pregnancy is seen, but in these respects it is entirely analogous to the usual form of eclampsia. Again, there is nothing characteristic in the relation of the disease to labor. While it has been observed more frequently before parturition, it may also occur during labor and in the puerperium.

The clinical history in all the cases gives evidence of the existence of a toxæmia prior to the appearance of coma. Such manifestations, however, are extremely variable and may persist through several months or last only a few days.

Headache and albuminuria with casts were invariably noted, although in one of Schmorl's cases albumin did not appear until shortly before death. Œdema, jaundice, nausea, and vomiting may occur as premonitory signs, but not necessarily. Preliminary visual disturbances were very common and would seem to possess a particular significance. Usually the patients complained of black spots before the eyes, dimness of sight or double vision, while Esch's case became totally blind and one of Schmorl's cases showed a left oculo-motor paralysis.

Consciousness usually disappears slowly. At first the patient is drowsy and stupid, later she becomes unconscious,

and finally passes into profound coma, the gradual transition occupying several hours. Occasionally, however, the woman has passed very suddenly from normal mentality to complete coma. Thus, in Meyer-Wirtz's case, the patient complained of intense headache and nausea two hours after delivery and quickly lapsed into deep coma. The duration of the coma has varied from 5 to 11 hours, except in one of Schmorl's cases, in which it seems to have persisted for a longer time, possibly 24 hours, although its exact duration cannot be determined from the data given.

CLINICAL SUMMARY OF CASES.

No.	Reported by	Age of Pt.	Para.	Variety.	Duration of Pregnancy.	Premonitory Symptoms.	Duration of Coma.	Obstetrical Condition.	Clinical Diagnosis.	Pulse.	Temp.	Respiration.
1	Schmorl.	22	I	Ante-partum.	6 mos.	Headache, jaundice, œdema, albumen and casts.	7 hours.	Undelivered.	Uræmia.	120	37.3 C.	Dyspnœa.
2	... Do....	23	III	...Do. ...	6 mos.	Headache, double vision, vertigo, left oculomotor paralysis, urine negative until just before death, no œdema.	Not stated.	.....Do.....	Cerebral syphilis.	Rapid.	No note.	No note.
3	....Do.. ..	37	X	...Do.....	6 mos.	Headache, vomiting, sleeplessness, albuminuria with casts.	6 hours.	..... Do.....	Uræmia.	..Do....	37.3 C.	...Do.....
4	Meyer-Wirtz.	38	I	Post-partum.	9 mos.	Headache, cyanosis, albumin with casts.	5 hours.	Delivered spontaneously.	....(?)	96	37.5 C.	Temporary cessation.
5	Esch.	25	I	Intra-partum.	10 mos.	Headache, dim vision, vomiting, albuminuria, total loss of sight, no œdema.	11 hours.	Undelivered.	....(?).....	84	39.7 C.	Deep and stertorous.
6	Siemons.	27	IV	Post-partum.	10 mos.	Headache, œdema, albuminuria with casts.	6 hours.	Delivered spontaneously.	....(?).....	46	98.4° F.	Stertorous.
7	... .Do.....	33	I	Ante-partum.	7 mos.	Headache, œdema, double vision, vomiting, albuminuria with casts, premature separation of placenta.	7 hours.	Undelivered.	Uræmia.	84	99.2° F.	Periodic.

The temperature is usually normal, but finally became elevated to 102° and 104° in the cases of Meyer-Wirtz and Esch, respectively. The pulse rate is not characteristic; more frequently it is slow at the onset of the coma and later becomes rapid, ranging between 120 and 160 to the minute. The usual findings are similar to those represented in the preceding chart, which also gives the only blood pressure observations that have been made in any of the cases. Irregularities in the force or the rhythm of the pulse have received slight mention.

The character of the respiration was commented upon in five of the seven cases. Deep stertorous breathing was noted by Esch, as well as in our first case, while dyspnœa occurred in one of Schmorl's cases. Respiration ceased in Meyer-Wirtz's patient at the onset of coma, but was re-established by artificial means; while our second case exhibited most marked irregular breathing, which differed from typical Cheyne-Stokes' respiration only in that there was no period of complete apnœa.

Pathology.

The pathological findings in all these cases have conformed to the anatomical picture of eclampsia, so that there can be no doubt as to the accuracy of the diagnosis. Indeed, the only question is, as to why the disease should occasionally run its course unattended by convulsions. Schmorl's explanation is that the eclamptic toxin is analogous in its action to certain drugs which have a stimulating effect when given in small doses, but cause depression and paralysis when administered in larger amounts. He is, therefore, inclined to regard such cases as manifestations of a more profound intoxication than occurs in the usual type of eclampsia, and ascribes the absence of fits to an overpowering of the central nervous system.

The truth of this hypothesis cannot be confirmed or denied until we have the poisonous principle in our hands. Meanwhile, it is of interest to study the anatomical changes exhibited by the seven cases reported with a view of ascertaining whether any organic lesion is constantly accentuated in non-convulsive eclampsia; and if so, what?

Pathological Summary.

Renal lesions were demonstrated in all cases, excepting that of Esch, in which the kidneys were not examined; but even in his case the clinical analysis of the urine afforded conclusive proof of the existence of a nephritis, as it showed a very large amount of albumin and numerous tube casts. In three instances there was evidence of a chronic nephritis with superimposed acute changes, while the three remaining cases presented only an acute parenchymatous degeneration. The intensity of the renal affection however, is notably inconstant, varying from a very mild, cloudy swelling of the epithelium to extensive destruction of tissue by hæmorrhage and necrosis.

The liver, without a single exception, presented characteristic necroses at the periphery of the lobule. Sometimes there was only a slight involvement of the organ, the necrosis not passing beyond the cells immediately adjoining the portal spaces. In other instances, large areas of the parenchyma were thrown out of function by degenerative processes and the extravasation of blood. Between these extremes, various gradations were noted, so that no connection can be established between the non-convulsive type of eclampsia and the intensity of the liver lesion. Similarly the heart and lungs

fail to show any more marked involvement than in typical eclampsia, although degeneration of the myocardium, and pulmonary œdema were observed in all the cases. Likewise, serous effusions into the peritoneal, pleural and pericardial cavities occurred in all the cases in which an especial autopsy note was made concerning the condition.

In contradistinction to the other organs, the brain in these cases was the seat of unusually severe lesions, which seem to be of such importance that I give a somewhat detailed account of the findings recorded in the literature.

SUMMARY OF EXTRAVASCULAR HEMORRHAGES.

No.	Reported by	Kidney.	Liver.	Spleen.	Pancreas.	Stomach.	Lung.	Heart.	Serous Effusion.	Brain.	Bacteriology.
1	Schmorl.	Acute upon chronic nephritis.	Peri-portal necroses.	Enlarged.	No note.	Punctiform hæmorrhages.	Œdema.	Parenchymatous degeneration and punctiform hæmorrhages.	No note.	Hæmorrhage and œdema.	Negative.
2	....Do.....	Parenchymatous degeneration.	....Do.....	No note.	Focal necroses.	....Do.....	....Do.....	....Do.....	....Do.....	Dura distended by cerebro-spinal fluid hæmorrhage.	....Do.....
3	....Do.....	Acute upon chronic nephritis.	....Do.....	....Do.....	....Do.....	....Do.....	....Do.....	....Do.....	....Do.....	Hæmorrhage into cerebrum, cerebellum, pons and medulla.	....Do.....
4	Meyer-Wirtz.	Parenchymatous degeneration.	....Do.....	....Do.....	No note.	Negative.	Negative.	Fatty degeneration.	Pleural and pericardial.	Cerebral apoplexy.	....Do.....
5	Esch.	Not examined.	....Do.....	Enlarged.	....Do.....	No note.	(Edema.	Punctiform hæmorrhages.	....Do.....	Hæmorrhage into lateral ventricles.	No note.
6	Siemons.	Acute upon chronic nephritis.	....Do.....	Coagulum in malpighian tufts.	....Do.....	Negative	....Do.....	Parenchymatous degeneration.	Pleural, pericardial, peritoneal.	Details not obtainable.	B. aerogenes capsulatus.
7	....Do.....	Parenchymatous degeneration.	....Do.	....Do.....	(Edematous.	Small fresh ulcer.	....Do.....	....Do.....	....Do.....	No examination.	Negative.

Schmorl's cases. No. 1: Meninges hæmorrhagic and œdematous. On the cortex multiple punctiform extravasations of the blood. In the central ganglia bluish red areas of softening, the size of a lentil. A similar focus in the left hemisphere of the cerebellum. No. 2: Dura under marked tension. Flattening out of convolutions. A hæmorrhagic area 3 to 4 cm. in diameter at base of left frontal lobe in subarachnoid space. Destruction of left olfactory and oculomotor nerves by hæmorrhage. Thrombosis of left cavernus sinus. Apoplectic focus, size of walnut in left frontal lobe. Multiple punctiform hæmorrhages over cortex. Areas of softening in cerebellum and central ganglia of variable dimensions, largest the size of a pea. No. 3: Punctiform hæmorrhages unusually abundant, and areas of softening throughout cerebrum, central ganglia, pons and medulla.

Meyer-Wirtz's case. Pia œdematous. Diffuse hæmorrhages on right side of temporal and parietal convolutions. 30 cc. of bloody fluid in posterior cranial fossa. Diffuse subpial hæmorrhages at base of brain in temporal region, as well as at boundary between cerebellum and pons. Blood clots on posterior surface of medulla and at point of exit of vagus. Lateral ventricles distended, containing bloody fluid. Substance of hemisphere pale, but of good consistence. Hæmorrhage into corpus striatum on right side, which extends into lenticular nucleus and internal capsule. Nucleus caudatus free, as also pedunculus cerebri. Extensive hæmorrhage in pons. Aqueduct of Sylvius and fourth ventricle filled with clots. Cerebellum negative.

Esch's case. Under posterior part of right parietal lobe there is a large extravasation of blood. On sectioning the brain, one finds single, punctiform hæmorrhages scattered throughout its entire substance. Hæmorrhage size of hazelnut in posterior part of right corpus striatum. Both lateral ventricles, and especially the right, are distended with bloody fluid.

The profound nature of the brain lesions noted here affords a probable anatomical and physiological basis for the unusual clinical course in this variety of eclampsia, and suggests at least two possibilites. In the first place the location and extent of the hæmorrhage into the brain substance could be such as to explain both the onset of coma and the absence of convulsions. The material at present available, however, speaks against the very frequent occurrence of this, and would rather seem to favor a second possibility, namely, that the phenomena in question are due to an increase in the intracranial tension, which in turn depends upon hæmorrhagic or œdematous changes in the brain or its enclosing membranes.

Rosenstein, who formulated his theory in accordance with the experiments of Traube and of Munk, formerly laid great emphasis upon the part played by increased intracranial pressure in the production of eclampsia. He held that increased arterial pressure and hydræmia were commonly observed in eclampsia; and that both of them caused an increase in intracranial tension; so that all of the symptoms of the disease could be interpreted as results of these changes. His hypothesis was ultimately abandoned, because the cerebral condition which supplied its basis was frequently lacking at autopsy. However it would seem that the brain lesions observed in these cases of eclampsia without convulsions strongly indicate, if they do not absolutely assure, a greatly increased intracranial pressure. Thus, hæmorrhage into the ventricles or beneath the meninges, œdematous changes in the brain

itself, or in the meninges have been found with great regularity. The dura has been seen to be markedly distended, the convolutions of the brain flattened out, and the cerebro-spinal fluid present in excessive amount; and all of these phenomena lead to the production of a high intracranial pressure.

The effect of an increase of tension within the skull is dependent upon the anæmia of the brain and medullary centers which follows it. This bears a very direct relation to the mean arterial pressure, as Cushing has shown that the blood supply to the brain is cut off when the resistance within the skull is greater than the force impelling the blood stream. The existence of this state of affairs may be accurately demonstrated only by comparing the pressure in the peripheral arteries with that in the cerebro-spinal fluid after trephining the skull and directly estimating the pressure within the cranium. However, no such observations have been made in cases of eclampsia without convulsions. Neither are we in possession of clinical data obtainable by a less radical procedure which would afford presumptive evidence of its existence, namely, a comparison of the pressure in the peripheral arteries with that in the spinal canal as determined by lumbar puncture. The importance of such observations in the future for the interpretation of the pathological physiology underlying eclampsia without convulsions is evident.

In our own cases the anatomical data at hand throw no light on the subject of intracranial tension. In the first case the intracranial conditions were not thoroughly studied at autopsy; while in the second, permission could not be obtained to open the skull. However, in this latter patient, who came under my personal observation, the clinical manifestations indicated the existence of an increased intracranial tension; as a marked elevation in blood pressure associated with periodic breathing is especially significant according to recent studies on Cheyne-Stokes' respiration.

Following the work of Cushing, which demonstrated in the event of an increase in the intracranial tension that the arterial pressure tends to rise and exceed the former, Eyster has been able to explain the association of "Periodic Breathing" with such conditions. The phenomenon would seem to depend upon "an alternation of anæmia and blood supply to the brain and medullary centers," as is shown by the blood pressure consecutively mounting above and falling below the line of intracranial tension, thus giving rise to the so-called Traube-Hering waves.

From his experimental and clinical work Eyster concludes that the existence of increased intracranial pressure in a patient may be definitely recognized when Cheyne-Stokes' respiration occurs "accompanied by a rise of blood pressure and an increase of pulse rate during the dyspnœic periods." The diagnostic value of these data and the importance of the changes in the eye grounds lately studied by Cushing and Bordley were not appreciated at the time our cases occurred, and we are, therefore, unable to make any statement concerning them. We do feel, however, that the clinical observations which were made, very strongly indicate a high tension within the cranium.

Diagnosis.

The clinical diagnosis in these cases has invariably been wrong. Most often the condition has been mistaken for uræmia, which is not surprising in view of the frequent history of long standing renal disturbance and the autopsy findings of chronic nephritis. Suicidal poisoning, cerebral syphilis and acute yellow atrophy were seriously considered several times, until the anatomical lesions revealed the true nature of the disease.

For the present we must admit that only the pathologist can make an absolutely positive diagnosis of eclampsia either with or without convulsions; and that his ability to do so will depend upon the demonstration of peri-portal necroses in the liver. This lesion, constituting the one constant feature of the disease, must be present to justify the diagnosis. The absence of it in the case lately reported by Reinecke effectually proves that his was not a case of eclampsia without convulsions.

Clinically, we remain unable to identify the disease positively at the bedside; since as I have already indicated, the obstetrician may meet on the one hand with various conditions besides eclampsia which are associated with convulsions and coma; while on the other, eclampsia may occur without giving rise to a single convulsive movement. Very fortunately, our inability to make an accurate clinical diagnosis is of but slight practical importance, since most of the affections with which eclampsia may be confused are manifestations of some variety of toxæmia of pregnancy and are, therefore, amenable to the same therapeutic measures. From a scientific standpoint, on the other hand, it is exceedingly important to differentiate, as far as possible, the several varieties of toxæmias, and at present this can be effected only upon a pathological basis. Failure to bear this in mind will result in great confusion and naturally delay the fuller recognition of our great ignorance and the subsequent development of more correct ideas.

Indeed, the anatomical evidence is so essential for the differentiation of every toxæmia of pregnancy that it is impossible to classify with certainty the cases reported by Lebenstein, Binder, Schlutius, and Jardine, in which recovery finally occurred. All of their patients were comatose for a time, having no convulsive seizures, while the urine changes were indicative of nephritis. It cannot be denied that these may have been instances of eclampsia without convulsions; but on the other hand, since positive anatomical proof as to their exact nature cannot be adduced, it would appear wiser to leave the diagnosis *sub judice*.

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