THE

URÆMIC CONVULSIONS

OF

PREGNANCY, PARTURITION,

AND

CHILDBED.

DR CARL R. BRAUN,

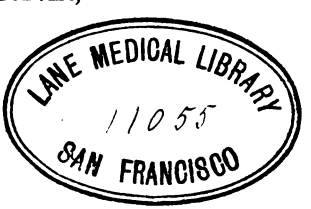
PROFESSOR OF MIDWIFERY, VIENNA.

TRANSLATED FROM THE GERMAN, WITH NOTES

BY

J. MATTHEWS DUNCAN, F.R.C.P.E.,

LECTURER ON MIDWIFERY, ETC., ETC.



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PREFACE BY THE TRANSLATOR.

THE following volume consists of the translation of a single chapter of Dr Braun's new text-book of Midwifery. (*Lehrbuch der Geburtshülfe mit Einschluss der operativen Therapeutik, der übrigen Fortpflanzungs-functionen der Frauen und der Puerperalprocesse.* Von Dr Carl R. Braun, K.K.O.Ö., Professor der theoretischen Geburtshülfe und Geburtshülflichen Klinik für Ärzte an der K. K. Universität in Wien, etc. *Mit* 150 *Holzschnitten. Wien* 1857. *Wilhelm Braumuller, K. K. Hofbuchändler.*) It has appeared to me so valuable, from its completeness and erudition, that I have first published a translation of it in the *Edinburgh Medical Journal* for 1856–57; and now, partly in consequence of the reception it has already met with, and partly from the utter deficiency of any similar treatise in the English language, I republish it in this separate form.

Students in every branch of Medicine, must be painfully aware of the deficiency, in this country, of labourers in the field of the organic chemistry of human pathology, perhaps the most important department of that great science. Dr Braun's work shows, in some degree, the immense advantages derivable and derived from a more intimate alliance between such scientific studies and the pursuits of practical physicians.

The notes which I have subjoined, in brackets, must speak for themselves. They make no pretension to completeness, and are

added merely to connect Dr Braun's memoir more intimately with our own authors, and to afford a medium for a few observations of my own.

Whatever estimate may be made of my comparatively unimportant labours in this matter, there can be no doubt of the honour due to Dr Braun, for his exposition of a subject which is at present one of the most prominent in the whole range of practical medicine, and in connection with which so much research is now going on, as to render it impossible to produce a perfect account of it.

I have been much indebted to a clerical friend for assistance in the whole translation, and I am also under obligations to my learned friend Dr Charles Wilson, for occasional aid of much value.

EDINBURGH, October 1857.

URÆMIC ECLAMPSIA.

CHAPTER I.

DEFINITION AND SYMPTOMS OF URÆMIC ECLAMPSIA.

ECLAMPSIA PUERPERALIS is an acute affection of the motor function of the nervous system (an acute neurosis of motility), characterized by insensibility, tonic and clonic spasms, and occurs only as an accessory phenomenon of another disease, generally of Bright's disease in an acute form (diabetes albuminosus, nephritis diffusa seu albuminosa), which, under certain circumstances, spreading its toxæmic effects on the nutrition of the brain and whole nervous system, produces those fearful accidents. The toxæmia (or blood poisoning), in eclampsia gravidarum, parturientium et puerperarum, is commonly produced by uræmia, *i.e.*, by a change of the urea which has been retained in the blood, or by retention of excrementitial extractive matter of the urine. Hence, according to the present state of our knowledge, true eclampsia during pregnancy is designated uræmic, without, however, implying that it is peculiar to pregnancy or child-bearing, because the same disease, with similar phenomena, may manifest itself also in women not pregnant, in children, and even in males under certain circumstances favourable to it.

Under the common appellation of "Eclampsia," several pathological processes have hitherto been comprehended, which do not even present an identical series of symptoms, and which have only this in common, that there exist tonic, and specially clonic spasms, along with loss of sensibility, in which the life of the patient is ordinarily in very great danger, and which very soon come to a termination.

From the eclampsia which arises from uræmia, are to be distinguished, that from defective purification of the blood, defective elimination of carbonic acid through the lungs, of bile from the blood (cholæmia), or of other matters which ought to have been secreted, as happens, for instance, in typhus.

Cerebral or apoplectic eclampsia is also spoken of, originating in meningitis, encephalitis, apoplexia intermeningealis or capillaris cerebri, thrombosis of the longitudinal sinuses, or in hyperæmia of the brain, of the spinal cord or medulla oblongata. Irritations of the terminal nerves may, by a reflex action on the spinal marrow and medulla oblongata, produce so-called hysterical convulsions, which, under certain circumstances, come closely to resemble an eclamptic attack.

Epileptic fits may happen during the period of pregnancy, as well as at other times; may occur only rarely, or several times in a day, and then again disappear.

They may, from the nature of the spasms, be very like a true eclampsia,[1] and hence may very easily produce deception and mistakes. Vogel, for instance, would have every eclampsia regarded as an acute epilepsy.

A chemically and histologically changed condition of the blood-serum and blood-corpuscles, such as hydræmia, leukæmia, hyperinosis, etc., may produce eclampsia.

Anæmia, after rapid and abundant losses of blood, produces death with the phenomena of eclampsia.

Mineral, animal, and vegetable poisons, such as preparations of lead, strychnine, conium maculatum, cicuta aquatica, oenanthe crocata, etc., inhaling of carbonic acid and carbonic oxide, have the power of producing conditions similar to eclampsia.

We are very far from denying that the most different causes may, during the period of pregnancy, as well as out of it, produce phenomena closely resembling those of uræmic eclampsia; but after a careful examination of literary and statistical observations by myself[2] and others, I think I am entitled to maintain my assertion already made—that, as a rule, eclampsia vera puerperalis is found intimately connected with diabetes albuminosus.

Uræmic eclampsia occurs in every period of pregnancy, as well as at other times, and even in males. It is distinguished by quick repetition of the fits, and complete insensibility during the fit, as well as generally during the interval. The face and neck appear swollen and injected during a paroxysm. The eyelids are prominent, and open or closed; the eyeballs exhibit quick rolling motions in the most different directions, or are fixed in an upward stare; the vessels of the conjunctiva are mostly injected; the mouth is at first widely opened and distorted; the tongue is protruded; then trismus follows, in which, if proper care be not taken, the protruded tongue is often bitten through, and hence a bloody foam often flows out of the mouth.

In the muscles of the face, lively distorting convulsions are observed, whereupon the upper extremities get bent, the trunk is twisted to one side, and then all the extremities are thrown into jerking motions.

Respiration often altogether ceases for many seconds. The carotids show strong pulsations; the veins of the neck and face swell on account of stoppage of the blood from muscular spasms. The colour of the face is cyanotic. All the

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[1] Eclampsia, from λαμβάνω, Fut. Ion. Med. λαμψομαι—to scrape together, to shake one's self. This etymology appears to be more correct than that from ἐκλαμψις, λαμπω, Fut. λαμψω, to flash or flare up. Synonyms of eclampsia puerperalis are, puerperal convulsions, acute epilepsy, renal spasms, uræmic convulsions, epilepsia renalis, albuminurica, Fraisen (Boër), Gichter (Hoffman); epilepsia ex utero of the ancients, allgemeine Krämpfe, schwere Convulsionen (Wigand); dystocia convulsiva (Young); dystocia epileptica (Merriman); encephalopathia albuminurica (Legroux).

[2] Braun, C.: Ueber Eclampsie und Albuminurie in der Zeitsch. d. G. d. Wiener Aerzte, 1851, Bd. 1. S. 57.

Braun, C.: Zur Lehre und Behandlung der in der Fortpflanzungsperiode, d. w. G. v. convulsionen mit, i. B. z. Hysterie, Epilepsie, Gehirnleiden, Vergiftungen und uramischer Intoxication bei Morbus Brightii. Klinik der Geburtsh. und Gynäkologie, Erlangen, 1855, S. 249.

muscles of respiration, especially the diaphragm, are in a state of contraction; and in consequence of this, asphyxia may occur. The urine and fæces are involuntarily excreted. Vomiting rarely precedes the first fit. The skin remains dry, or may be covered with perspiration, and its temperature is either increased or diminished. The reflex sensibility is suspended during the fit. The pulse is frequent or slow; the arteries are large or small.

After this group of symptoms, there follows a soporose condition, in which the patient continues for a shorter or longer time, and lies motionless; the extremities stretched out and stiff; the respiration frequent and difficult, and at first stertorous, afterwards slower and snoring. Generally there is absence of consciousness and sensation.

The duration of each fit, including the convulsive tonico-clonic part and the soporose part, extends commonly to half an hour, or to one or two hours, and only in very rare cases does the sopor of the first fit last a whole day.

If the paroxysm do not terminate in death, a remission takes place; the respiration becomes more slow and free, and less rattling; the rigidity of the muscles diminishes; the frequency of the pulse becomes considerably less; consciousness either does not return at all, or only very slightly, mostly remaining dim, so that a proper but short answer may be got to a question in a loud voice, but recollection of what has happened is altogether wanting. The abdomen is sensitive to touch, and the reflex sensibility is often intense during this lighter sopor.[1] After awaking, patients generally complain of a confused, dull headache, and of great languor, which continue till a renewal of restlessness, stretching, extending, slow tremulous bending of the upper extremities, jerking of the facial muscles, with reddening of the face, announce a new paroxysm. The fits may be repeated several times in a day—sometimes as much as seventy times. Generally after a few fits, complete unconsciousness supervenes, and this continues till recovery or death.

Sometimes there are phenomena foreboding the coming on of fits.[2] They

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[1] [Described by Dr Hamilton as supersensation. See his "Practical Observations on Midwifery," Part ii.]

[2] [Sometimes, in cases of uræmia, the premonitory symptoms of eclampsia are present to an extreme degree, without eclampsia really occurring. Such cases are, pathologically, nearly identical with eclampsia, and require the same care and treatment. The following very interesting case of this description was very lately under my professional attendance:—A lady had given birth, at the full time and naturally, to four healthy children. In her fifth pregnancy, she was taken about the sixth month with epistaxis, frequently and to a great extent. About the same time, symptoms of the same nature appeared as in her sixth pregnancy, now to be described. These disappeared soon after the birth of a dead child, about the end of the seventh month. In her sixth pregnancy, she was in Edinburgh, under my care. About the seventh month, she was again very ill, and had been so for some time before I was sent for. No epistaxis occurred. The face and hands were slightly œdematous. The cheek was coloured a bright purplish red. The legs were much swollen. There was much complaint of noise in the head and of weakness of the lower limbs. The first symptom to appear and the last (except the albuminuria) to disappear, was pain in the back near the base of the chest, round the sides, and in the epigastrium, where also there was tenderness on pressure. The urine was clear and of high

consist of headache, giddiness, wandering of the ideas, increased heat of skin, mental illusions, seeing of lights, imperfectness of vision even to total blindness (amblyopia, amaurosis), with an enlarged pupil and staring look, noises in the ears, difficulty of speaking, lowness of spirits, pains in the præcordium, nausea, vomiting, palsies, irregular pulse, and great languor without any known cause.

The fits commonly appear for the first time during the last three months of pregnancy, more frequently during labour, and in childbed. Generally, for several weeks before the first fit, œdematous swellings may be observed in the most different but single parts of the body; it is rare not to find them, and sometimes they are very extensive.

Œdema in the upper part of the body, the face, hands, and arms, is in uræmic eclampsia not unfrequent; but is very commonly observed in the ankles, and feet, and in the labia majora. The œdema of the face, when it occurs, is generally connected with increased heat of skin, redness of the cheeks, injection of the conjunctiva, in consequence of which it may assume a swollen appearance. These œdemata are not very constant, appearing when the patients are recumbent on the back, and disappearing when they get up, and *vice versa*. They sometimes decrease towards the end of pregnancy, and not unfrequently disappear altogether, while the albuminous contents of the urine, and the morbid process in the kidneys themselves, are sometimes increasing.

The skin of the non-œdematous parts of the body appears very dry, and as white as chalk (chlorotic, hydræmic, leukæmic), and has a low temperature. Only those œdemata of pregnant women which exist contemporaneously with albumen, fibrin cylinders, and fatty degenerated scales of Bellini's epithelium in the urine, have a connection with uræmic eclampsia. The œdema of the lower extremities, ascites, and hydramnios, which are not complicated with albuminous urine containing fibrin cylinders, are not followed by uræmic eclampsia in pregnancy or labour. The affection of the kidneys with disease cannot with certainty be inferred from the appearance of dropsy, as distinct causes may, at the same time, or one after the other, produce dropsies.

The urine, removed by the catheter, in cases of uræmic eclampsia, is generally

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colour, not very scanty, loaded with albumen. Very soon, signs of death of the fœtus became distinct, and it was soon after born dead. The process of birth occupied only a few hours in an evening. But, from the morning of that day, and for about thirty-six hours afterwards, no urine was passed. The symptoms, caused by local pain and tenderness of the kidneys, were at the same time very acute. The persistent giddiness was intense, and although the light was painful to the eyes, and it was difficult to keep them open, she had to make constant efforts to do so in order to assure herself of the unreality of the frightful dreams and visions that afflicted her. The first urine passed was in small quantity, and very turbid from lithates contained in it; and, when heated, became almost a solid clot. During this condition, the pulse was 96. Under the microscope, the urine showed crystals of uric acid, urate of ammonia, some blood corpuscles, and patches of epithelium from the tubuli uriniferi. It was of sp. gr. 1030.

In ten days the albuminuria and symptoms of nephritis had almost completely disappeared. There was then a slight reaccession of albuminuria, with fibrin cylinders in the urine. But about the fourteenth day, all traces of the disease had vanished.]

acid, and when nitric acid is dropped into it, or when the fresh urine is boiled, exhibits a large quantity of albumen; and when examined by the microscope, the well-known fibrin cylinders are observed, often also blood-corpuscles. The quantity of albumen has generally an intimate relation to the extent, intensity, and duration of the acute Bright's disease, but not so constantly to the violence of the eclampsia. The albumen in the urine, however, generally increases at the end of pregnancy, during labour, and during the eclamptic fits.

To demonstrate the albuminuria and diffuse exudation in the substance of the kidneys, we use moderately diluted nitric acid, as the fuming nitric acid re-dissolves and decomposes the coagulated albumen. The acid is dropped in at the edge of a test-glass, whereupon the albumen is separated in yellowish-white thick flakes, which, being heated to the boiling point, undergo no change. Further addition of diluted nitric acid produces no change in the coagulated albumen, but any sediment of phosphate of lime (bone-earth) is dissolved by it. The addition of ammonia precipitates the phosphate of lime, but not the albumen, the coagulated flakes of which are, on the contrary, completely dissolved. If, by the above reaction, the urates (urate of ammonia) also were precipitated, they are re-dissolved by a boiling heat.

It is only in urine having an acid reaction that albumen is precipitated in large quantity by boiling, for, in alkaline urine, the ammonia, which is always present, retains the albumen in solution. Hence, to every alkaline urine, previous to boiling, a few drops of acetic acid must be added, until blue litmus paper is distinctly reddened by it. Boiling precipitates both the albumen and phosphate of lime. The further addition of acetic acid leaves the albumen unchanged, but quickly dissolves the phosphate of lime.

Of the normal constituents of the urine, the urea is constantly diminished, even sometimes altogether wanting; the uric acid is also ordinarily in small quantity—the uroxanthin in increased quantity; all the other components, the sulphates, phosphates, and urophœin, vary very much; and the chlorides alone are not in the ordinary quantity, but without any striking difference. The specific gravity of the urine varies from 1010 to 1030. In the thick sediment of the urine there are found, during the first twenty-four hours, besides blood and mucous corpuscles, and epithelial cells of the urinary passages, those fibrin cylinders (cylindrical clots) first described by Henle, Nasse, Simon.[1] They are, however, absent in alkaline urines, as the bi-carbonate of ammonia, which is formed in the decomposition of the urine, dissolves the fibrin.

Frerichs[2] describes these cylinders as $\frac{1}{60}'''$ thick, and $\frac{1}{4}'''$[3] long, consisting of amorphous fibrin, and containing sometimes crystals of oxalate of ammonia and of uric acid, and believes them to be constant companions of the different

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[1] [See Simon's Animal Chemistry, Sydenham Transl., vol. ii., p. 235; also Gairdner, Pathology of the Kidney, 1848; and Johnston, Diseases of the Kidney, 1852.]

[2] Frerichs, F. Th.: *Brightsche Nierenkrankheit*. Braunschweig, 1851.

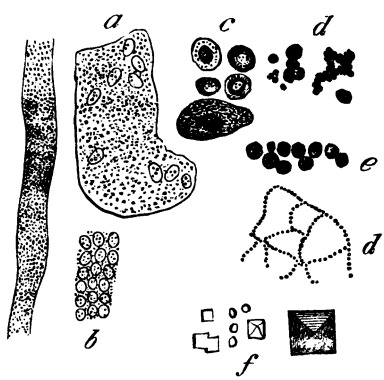
[3] [A Paris line ''' is the ·088 inch.]

degrees of disease of the kidneys.[1] He delineates five different kinds of these cylinders, according as they are either covered with a coating of exfoliated epithelial cells of the tubuli uriniferi, or of shrivelled up homogeneous epithelial cells; or resemble an amorphous clot with numerous fatty granules, or are covered with blood-corpuscles and crystals of oxalate of lime, or exhibit an amorphous clot with purulent contents.

The more acute the Bright's disease, the darker is the urine, and the more numerous, generally, are the blood-corpuscles.

For a microscopic demonstration of the fibrin cylinders, it is best to examine fresh urine about an hour after it has been drawn off by the catheter. By means of a glass tube 4″ long and $\frac{1}{2}$‴ wide, or with a pipette, some drops of urine are taken from the bottom of the vessel, and subjected to microscopic investigation. By this means, fibrin cylinders present, even sparingly, are easily discovered.

Fig. 1.



The bodies represented in the above figure (*i.e.*, the exudation clot), from the albuminous urine of a woman suffering from eclampsia, do not, according to Wedl's[2] view, deserve the name of fibrin cylinders, since a molecular form of fibrin is doubtful. It is easy to avoid confusing these so-called fibrin cylinders with the epithelium of the tubuli uriniferi (Bellini's epithelium), inasmuch as it (Fig. 1, *b*) always exhibits an entire series of oval nuclei, arranged at equal distances, and which are held together by an intermediate molecular substance.

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[1] Fig. 1. Different bodies in the urine of an eclamptic patient: (*a*) cylindrical exudation clots (so-called fibrin cylinders); (*b*) epithelium of the tubuli uriniferi; (*c*) flat brownish epithelial cells; (*dd*) urate of ammonia; (*e*) drusy crystals; (*f*) muriate of soda (blue, like the corncockle, from an admixture of uroxanthin), distinguishable from oxalate of lime by its solubility in water. Enlarged 350 times. (after Wedl).

[2] Wedl: *Grundzüge der path. Histol. Wien*, 1854, S. 301.

The brown-red colour of solitary, smooth, epithelial cells, which appears partly upon, partly around the nuclei, arises from the urine containing hæmatin, and is to be distinguished from that of the blood-corpuscles, which are, in this case, never arranged like packets of coin—by the definite size, flattened shape, and cup-like depressions of the latter.

As to the occurrence of these cylindrical clots, it is just as with the albumen and blood-contents of the urine; at one time a considerable quantity of them may be present in all those forms of disease which are included under the name of Bright's disease; at another time, even after careful examination, scarcely one of them, or even none at all, may be found. It is, indeed, well known, as Wedl has repeatedly pointed out, that the exudations advance by intermitting attacks, and that hence there occur periods when no exudation takes place in the kidneys. The relative quantity of the clots also depends on the varying quantity of water in the urine.

The occurrence of epithelium of the tubuli uriniferi exclusively, or in great proportional quantity, indicates an exudation just beginning to take place (just as we observe the same in inflammations of mucous membranes), the fluid exudation and epithelium being at first washed away in great quantity. It is, however, also possible that a merely congestive condition may induce a somewhat increased transudation, and thereby also a mortification of the epithelium—a process which comes soon to a termination, without leaving any evil consequences behind. We find, in fact, that in erysipelas, scarlatina, pneumonia, etc.,[1] a great quantity of the epithelium of the tubuli uriniferi is discharged with the urine, and, nevertheless, speedy recovery takes place. Höfle and several others have observed even exudation-clots in the urine, in cases which followed a favourable course. The exudation has, in these cases, quickly ceased without the case reaching a more advanced stage of Bright's disease (Wedl).

In this manner, those cases also may be accounted for in which the most violent albuminuria, during pregnancy, and later, or during parturition, entirely disappears, and also those where a violent albuminuria during parturition entirely disappears during the confinement in child-bed, and also those cases of death from ten to fourteen days after the eclampsia, in which sometimes no disease of the kidneys can be found.

Along with these changes of the urine, there is frequently observed great tenderness of one or both kidneys, on being subjected to pressure through the abdominal walls, a circumstance which Litzmann[2] has found to be not characteristic of pregnant women in a state of health. The circumstance of pressure on the lumbar portion of the spinal column causing pain is a matter of no moment, as this is often observed to occur in healthy pregnancy.

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[1] [The peculiar condition of the urine, in certain acute diseases, and more particularly its temporary coagulability, and the presence of epithelium, in greater or less amount, has of late years received much attention in this country. See a paper on Temporary Albuminuria, by Dr Warburton Begbie, in the *Monthly Journal of Medical Science*, for October 1852; also, an article, "Albumen in Urine," by the same author, in the *British and Foreign Medico-Chirurgical Review*, for July 1853.]

[2] Litzmann: *Deutsche Klinik*. Berlin, 1852. Nr. 19–31.

Gastric symptoms, as a foul tongue, pains about the stomach, constipation, have seldom any connection with eclampsia.[1]

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[1] [Complete or nearly complete suppression of urine during the day of the labour and of the fits, is not unfrequently observed. Pain and tenderness around the whole body, in the region of the kidneys, are very characteristic symptoms, and often present. These are much more easily referred to the inflamed kidneys in cases where pregnancy is not far advanced or when delivery is already completed. In such cases there is also a marked undefined fulness frequently present in the hypochondriac regions. In a very interesting case of the kind under consideration, in which I was lately called in consultation, this symptom was very marked and easily made out, in consequence of the inflammatory affection being confined to the right kidney (See Christison on Granular Degeneration of the Kidneys, p. 20), while, on the other side, there was easy compressibility without tenderness. In this case the albuminuria was persistent. There had been no fits during the confinements, and only very inconsiderable œdema of the ankles.]

CHAPTER II.

PATHOGENESIS OF URÆMIC ECLAMPSIA.

SEVERAL years ago I analyzed the numerous views entertained as to the cause of eclampsia, and then arrived at the conclusion, that acute Bright's disease and uræmic intoxication of the blood were the causes of eclampsia, not its results. In other quarters, the opposite view is held.

In order to get a clear view of the present state of the subject, I shall first discuss all the arguments in favour of the identification of eclampsia and uræmic intoxication, and then enter upon the refutation of the opposite view, that the disease of the kidney is entirely the effect of the eclampsia, the latter being the result of some disease of the blood still unknown—perhaps hydræmia, leukæmia, puerperality, etc.

The coincidence of eclampsia and albuminuria is an undisputed fact, which is verified by the very numerous observations of Lever,[1] Simpson,[2] Devilliers,[3] Regnault, Dubois, Danyau, Cazeaux,[4] Cormack,[5] Blot,[6] Helfft,[7] Frerichs, Litzmann, the author, and many others, as also by daily observation. This has opened a new path to the knowledge and treatment of this most dangerous disease, so that the eclamptic convulsions of women during pregnancy must be considered to be identical with the fits of adults in general, that are produced by uræmia in the course of acute Bright's disease. This I publicly declared in January 1851, and at the same time Frerichs published his most convincing classical treatise on the subject. It must now be considered an axiom in theory as well as in practice.

Frerichs has often found the œdema already developed in the third month of pregnancy, although far more frequently it is first observed in the four last months, often, however, without attracting attention, as the other less obvious features of the morbus Brightii are overlooked, although they are present. The more the pregnancy advances, the more the albumen in the urine increases,

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[1] Lever: *Guy's Hospital Reports.* April, 1843.
[2] Simpson: *Monthly Journal.* 1847, p. 288.
[3] Devilliers und Regnault: *Archives Generales de la Med.* 1848, Mai.
[4] Cazeaux: *L'Art des Accouch.* Paris, 1850, p. 465.
[5] Cormack: *Medical Times.* 1850, p. 26.
[6] Blot: *Bulletin de Therap.* xxxviii. p. 421.
[7] Helfft: *Neue Zeitschrift für Geb.* 1851. Heft. 3.

but disappears quickly after labour, sometimes within a few days, unless the exudative process in the kidneys makes further progress, and assumes the condition of chronic Bright's disease. But dropsical swelling is not necessarily connected with this albuminuria, just as, on the other hand, there not unfrequently occurs, during pregnancy, a dropsy in which the urine is found quite normal.

A hyperæmia, affecting one or both kidneys, caused by congestion of venous blood, is the primary stage of acute Bright's disease. It is soon followed by extravasation of a fibrous exudation into the Malpighian capsules, which to some extent invades the interstitial tissue, partly covers the knot of vessels, partly flows into the urinary canaliculi, and is often secreted, in a fluid form, with the urine. But, generally, the albumen only of the exudation passes off with the urine, while the greater portion of the fibrinous matter coagulates in the tubuli of the cortical substance, remains in them a longer or shorter time, till it is propelled from thence, the epithelium being at the same time often exfoliated and discharged with the urine in the form of the rectilinear tubes of Bellini and Ferrein. By this inflammatory process, and by the continual production of an abnormal plasma around the epithelial cells of the tubuli uriniferi, the nutrition of these last at length suffers; they commence a retrogressive metamorphosis into cells with fatty granules, and dissolve into a detritus, rich in fat, whilst, at the same time, a similar change affects the fibrinous substance which has been retained a length of time. The tubuli, deprived of their epithelium, now collapse; their walls come into contact, and, instead of the glandular parenchyma, there arises from the remains of the basement membrane an indistinct fibrous cicatricial substance, which produces, as a result, the depressions observable on the surface of the kidney; while other tubuli, still filled with fat, are seen on the external surface, and in a section of the cortical substance. The further this collapse of the renal tubuli proceeds, the more, of course, is the volume of the kidneys diminished, and at length atrophy of them exists, especially as, at the same time, a portion of the nutritive circulatory apparatus is destroyed by obliteration.

The essence of the Bright's disease, lying at the root of the eclampsia, is then, according to this view, an inflammatory process (*Nephritis diffusa* of Reinhard), an explanation which Frerichs considers to hold good only so far as the exudation of blood-plasma is connected with a paralytic-like dilatation of the capillaries; whilst, in cases where mere pressure of the pregnant uterus upon the renal veins is the cause of the exudation, he will not admit the existence of inflammation proper.[1]

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[1] [The influence of simple pressure by the pregnant uterus seems to be much exaggerated. For in cases of ovarian dropsy, fibrous tumour, and a variety of other affections, no such result is observed from a pressure exactly like that of the gravid uterus, and often more severe and longer continued. Moreover, it is well known that a great number of characteristic cases occur before the uterus has so increased in size as to cause much pressure, and many after all extraordinary pressure is removed by complete delivery.

But of the influence of pressure on the renal veins, when really exerted, the

The symptoms of uræmia, which are observed in animals, after the extirpation of both kidneys, are the same as those of the acute Bright's disease of pregnant women, in whom the diseased condition of the secretion and excretion of the kidneys causes pollution of the blood with excrementitial elements of the urine, a state which frequently, after headache, giddiness, drowsiness, delirium, coma, and convulsions, leads on to death.

The acute form of uræmia, which manifests itself by convulsions or sopor, admits in general of a more favourable prognosis than the gradually and slowly developed disease. Among the symptoms of uræmia, are also the diminished power of vision, amblyopia, and complete amaurosis. A pregnant woman, after headache, vomiting, or a condition of stupefaction, may be found altogether blind. We have also to enumerate here the humming in the ears, or the suddenly supervening partial deafness of pregnant women; also a fever very like typhus, with diminution or complete suppression of urinary excretion (*Febris urinosa* of Frerichs); in addition, a painful vomiting of tough or watery substance, in which the addition of an alkali reveals the presence of ammoniacal compounds, but never of undecomposed urea.

These various phenomena appearing in the different regions of the body during life, leave no changes constantly discoverable after death. The brain and its membranes are then either exsanguine, natural, or engorged with blood; the sac of the arachnoid, and the cerebral ventricles, contain sometimes, but not always, a serous effusion. The globe of the eye is, after uræmic intoxication and amaurosis, sometimes normal; sometimes it exhibits an increased secretion of aqueous humour (Cucuel,[1] Abeille,[2] Crocq,[3] Collard,[4] Marchal), sometimes an exudation on the retina (Türk[5]).[6]

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experiments on rabbits of Meyer (*Arch. f. Phys. Heilk.* Bd. 3. S. 116–119) leave no doubt. "On compressing with a ligature, sometimes the renal vein on one side and sometimes the inferior vena cava, by which the increased hydrostatic pressure of the blood must dilate the renal capillaries, he always found albumen in the urine collected after the operation; and on tying the renal vein on one side, he only found albumen in the urine that escaped from the exposed ureter of the side on which he operated, and here it was very abundant." Lehmann's *Physiological Chemistry*. Dr Day's Translation for the Cavendish Society. Vol. ii., p. 423.]

[1] Cucuel: *Union méd.* 1850, Janv.

[2] Abeille: *Revue clinique.* 1850, Avril.

[3] Crocq: *Presse med. de Bruxelles.* 1850.

[4] Collard: *Union méd.* 1850. Nr. 41.

[5] Türk: *Zeitschrift d. Ges. d. Wiener Aerzte.* 1850.

[6] [This condition must be distinguished from the amaurosis from undue lactation, described by Drs Ashwell (*Practical Treatise on the Diseases Peculiar to Women.* Third Edition, p. 761) and Mackenzie (*Glasgow Medical Journal.* April 1854). Mialhe (*Chimie appliquée a la Physiologie et a la Thérapeutique.* 1856. P. 164) attributes the weakness of vision in albuminuria and diabetes to milkiness of the serum of the blood, caused by the presence of caseiform albumen in it, in a state of suspension not of perfect solution, and interfering with the transparency of the humours of the eye. This theory would, if verified, explain only cases of indistinct vision not of complete amaurosis. In diabetic patients, there is a tendency to cataract, which probably will lend some help to the real explanation of the disturbance of vision in such cases.]

The degree of coagulation of the blood, when drawn from the veins, varies; it has a tinge of violet colour, sometimes an ammoniacal smell, distinctly reminding one of putrid urine, and it contains carbonate of ammonia (often in such quantity, that it effervesces on the addition of muriatic acid), with generally some remains of undecomposed urea.

The uræmic fits do not originate, as was formerly supposed, only from the blood being poisoned by urea. Filtered urine, injected into the veins of animals, has been tolerated without evil consequences.[1]

In regard to uræmic intoxication, Frerichs has been led, by a series of carefully performed experiments, to conclusions, which we have found confirmed in the cases of several pregnant women who suffered from eclampsia and acute Bright's disease.

(*a*) The phenomena of uræmic intoxication are produced neither by urea or any other ingredient of the urine, nor by the united excretory matters, as such, of this fluid; but they commonly arise from this circumstance, that the urea accumulated in the blood is transformed into carbonate of ammonia under the influence of some peculiar ferment.

(*b*) Carbonate of ammonia is the baneful power which produces these disturbances of the functions of the nervous system.

For the production of uræmic intoxication, it is therefore necessary to have in the blood quantities of urea, and the presence of some ferment, by means of which the urea may be changed into carbonate of ammonia.

If the fermenting material is awanting, then the blood may be for a long time impregnated with urea, without any injurious consequences appearing. In this way the fact is accounted for, that in the bodies of persons dead of Bright's disease, the blood may be found saturated with urea, without any uræmic phenomena having been observed during life.

The cause of this fermentation is, as yet, not altogether known.

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[1] [See Johnston on Diseases of the Kidney, p. 199, etc.]

CHAPTER III.

THE CONNECTION BETWEEN ECLAMPSIA AND THE PAINS OF LABOUR.

THE phenomena of uræmic intoxication take the form of eclampsia, often, no doubt, when active uterine pains are present; but they are as certainly observed in the course of pregnancy, as well as in the puerperal state, in women not pregnant, and also in young men.

By the observations of Böer, Baudelocque, Prestat, Scanzoni, of the author, of Wegscheider,[1] Litzmann, Lever, Beer,[2] Wieger,[3] Chailly,[4] Gustav. Braun, Morel,[5] Lobach,[6] Thomas,[7] Crède, Hohl,[8] and many others, it has been established as a matter beyond doubt, that eclampsia may appear independently of uterine pains. Even with the most violent eclampsia, sometimes no pains are observed; the fits may cease, and pregnancy go on for weeks;[9] or pains already become active may disappear, and days may pass ere parturition take place spontaneously; or, during the continuance of the eclampsia, complete absence of pains may be remarked, the patient may die of the fits before labour is ended, and a dead fœtus be extracted by Cæsarean section from the corpse.

Pains must not be considered as the cause, but with more truth as the effect of the eclampsia, *i.e.*, of the uræmic intoxication. With eclampsia, spontaneous premature labours are not unfrequently observed.[10] This may arise from the

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[1] Wegscheider: *Verhandlungen d. Berliner Gesellsch. f. Geb.* 5. J.
[2] Beer: *Oesterr. Wochenschrift.* 1843, Nr. 38.
[3] Wieger: *Gaz. de Strasbourg.* 1854, Nr. 6–12.
[4] Chailly: *L'art des Accouch.* Paris, 1853, p. 196.
[5] Morel: *Gaz. de Hôp.* 1853, Nr. 155.
[6] Lobach: *Verh. d. phys. med. Ges. zu Würzburg.* 1852, iii. 2.
[7] Simon Thomas: *Nederl. Lancet.* Dec. 1853, en Jan. 1854; *und Schmidt's Jahrb.* Bd. 84, s. 321.
[8] Hohl: *Lehrb. f. Geb.* 1855, s. 288.
[9] Thus Morel witnessed a case where a dead child was born twenty days after the most violent eclampsia; and Gustav Braun, on the 12th May 1854, in the Midwifery Clinique in Vienna, saw a case of uræmic eclampsia, after which the albuminuria even disappeared; and fourteen days having elapsed, a living child was born.
[10] [Uræmia is, when present, often a cause of death of the fœtus and premature labour. For an example, see the case recorded in footnote, p. 7, where it occurred in two successive pregnancies.]

circumstance, that in acute Bright's disease and uræmia the nutrition of the nervous system is carried on in a morbid manner by undepurated blood. Hence, premature labours may be induced by eclampsia, just as by other acute diseases, as pneumonia, typhus, etc.

By exciting pains, and increasing their strength, fits cannot be produced at will, nor even aggravated. For we have made the observation that, under a high degree of reflex sensibility, convulsions cannot be induced at will at definite periods by violent irritation of the uterus; and that, by the energetic use of the caoutchouc-bladder plug, very strong pains may indeed be produced, but no increased intensity, and no more frequent returns of the eclamptic fits, and that during colpeurysis[1] the convulsions themselves sometimes altogether cease, and the birth of the child takes place rapidly.

The frequent occurrence of eclampsia during the first days of child-bed, and up to six weeks after delivery, as has often been observed by Lever, Devilliers, Regnault, M'Clintock, Hardy, the author, Simpson, and others, evidently proves that there is a very subordinate relation between active pains and uræmic eclampsia.

Repeated returns of eclampsia in subsequent pregnancies in the same individuals have been observed by many. Litzmann reports a very rare example, in which, in nine pregnancies, uræmic symptoms appeared under the severe form of convulsions and amaurosis. In general, however, relapses are rare, although uræmic eclampsia may happen in every pregnancy (not in the first only), and even twice in the course of one child-bed. A short time ago, I had a very rare case of most violent uræmic eclampsia, ending in recovery, occurring in the fourteenth pregnancy of a woman who had already borne, without any difficulty, fifteen blooming children (several years ago, she twice had twins). In this case, the idea of relapse is not to be mentioned.

Among the most constant premonitory symptoms of eclampsia, are nausea, retching, vomiting. These are to be attributed to the uræmia, and not to sympathetic nervous irritation, only when in the vomited matters, by means of a chemical reaction with an alkaline solution, carbonate of ammonia is shown to be present. Of humming in the ears, patients do not often complain.

Lowness of spirits, melancholia, unruliness, ardent restlessness, unmannerly conduct, frequent cold rigors, are, as uræmic symptoms, sometimes also forerunners of the breaking out of eclampsia. Tossing about in bed during pains indicates a perception of painfulness, which, however, on account of the diseased condition of the brain, does not reach to full consciousness. When the paroxysms are numerous, sometimes the appearance of a fit is contemporaneous with the acme of a pain, sometimes with the interval between pains.

Convulsions, when they appear suddenly, are probably produced by the sudden decomposition of a great quantity of urea.

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[1] [Colpeurysis is an operation which consists in distending the vagina by means of a colpeurynter, or bag of caoutchouc into which water is forcibly injected and there retained. It is a method of inducing premature labour, recommended by Dr Braun.]

Without uræmia, the most violent and painful uterine contractions produce no eclampsia in a case of Bright's disease; as, after long experience, we have frequently observed in women suffering from Bright's disease during pregnancy and natural labour.

If it happens that the uræmia is removed during pregnancy or labour, then the later and stronger pains generally are followed by no more fits.

With the disappearance of uræmia, the pains sometimes become weaker. During uræmia the uterus is never seized with tetanus. The consideration of these numerous facts makes it certain that the pains play a very subordinate part in cases of uræmic eclampsia.

Between eclampsia and the serous effusions there does not exist any constant relation. On the contrary, when the dropsy is very extensive, eclampsia is very rare, for the blood gets rid of a part of the retained urea by the serous secretions, and the uræmic intoxication may thus be kept off.

Uræmia exerts its influence first upon that portion of the brain which physiologically still belongs to the spinal marrow, *i.e.*, the medulla oblongata. This is clearly indicated by the disturbance of the sensitive parts (headache, giddiness), and of the motor parts (convulsions).

Hemeralopia occurs with or without eclampsia, with or without acute Bright's disease, and generally disappears a few hours after delivery.

Tyler Smith[1] has tried to account for the considerable lividity of the face, which generally comes on during a paroxysm, by a tonic spasm of the heart, specially of the right auricle, whereby the return of the blood by the veins is stopped; and he thinks the emptiness of the ventricles after death supports his view.

But this view is contradicted by the state of the pulse during the paroxysm; for, as Litzmann has very correctly observed, the pulse becomes slower in the beginning of the paroxysms, and then rises very quickly to 120 or 150 in the minute, and becomes smaller and quicker, according as the intervals between the fits become shorter, and according as the eclamptic attacks become more frequent.

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[1] [Parturition and Obstetrics, p. 319.]

CHAPTER IV.

INFLUENCE OF ECLAMPSIA ON THE LIFE OF THE FŒTUS.

It is an unquestioned fact, that the eclampsia of pregnant and parturient women exerts upon the fœtus an influence dangerous to life. It is only as to the way of accounting for this fact that opinions are still divided. Kiwisch supposes that the stoppage of the circulation in the maternal vessels of the placenta during the fits, is a part of the cause of the death of the fœtus, but does not think that the fœtus, at the same time, dies of eclampsia.

We cannot assent to this explanation, since many convulsive fits, originating in habitual epilepsy, and appearing repeatedly in the same day, never exercise any injurious influence on the life of the fœtus when albuminuria is altogether absent.

But, after even one or two eclamptic fits, it is sometimes found that the fœtal motions have diminished in liveliness, that the fœtal heart-sounds have ceased, and that the fœtus is dead; and it is in exceptional cases only that the fœtus remains alive after numerous paroxysms. If the mother dies during pregnancy, under uræmic symptoms, it is almost always a dead child that is brought to light by the abdominal section. If, after numerous uræmic convulsive fits, the child is born still alive, a large quantity of urea is found in the blood taken from the umbilical cord; but if it is born dead, we can, immediately after the birth, demonstrate the presence of carbonate of ammonia in the fœtal blood. If it happens that the uræmic symptoms have been entirely removed during the pregnancy and delivery, or if the fœtus has been cautiously and in good time removed from the uterine cavity, then the life of the child may be permanently saved, if it is mature; but children not quite at the full time often succumb, even under these circumstances, in the first days of life. If, during the eclampsia of pregnant women, the child does not die at once, it generally does so a few days afterwards; so that, when at a subsequent period delivery takes place, only dead children are born. The opposite happens only in an exceptional case.

The cause of death of the fœtus of eclamptic mothers is, for this reason, chiefly to be sought for in the passage of carbonate of ammonia into the fœtal circulation. But the uræmia itself of the mother may, without eclampsia occurring, destroy the fœtus, as we have observed in acute Bright's disease of pregnant women without eclampsia.

Besides the uræmic poisoning of the blood, other injurious influences may

endanger the life of the fœtus, as interruption of the placental circulation by violent pains, in cases where insuperable difficulty is produced by transverse presentations, disproportion in size, pressure on the cord, premature discharge of the waters, and deficient preparation and softening of the neck of the womb. Albuminuria and Bright's disease of the kidneys of children, born of eclamptic mothers, have been observed by Simpson. We have not unfrequently observed apoplectic masses in the cavity of the skull and spine in cases of children dying of convulsions in the first days of life.[1]

The mortality of children during the fits, and during delivery, amounts to forty-five per cent. During the period immediately following delivery, the mortality is forty per cent. among those born at the full time, and sixty-four per cent. among the premature.

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[1] [Some remarkable cases of children born of mothers suffering from albuminuria have attracted my notice. In one, the child, above a year old, suffered at the same time from laryngismus and albuminuria.]

CHAPTER V.

ETIOLOGY OF URÆMIC ECLAMPSIA.

THE causes which may produce acute Bright's disease and uræmia, are to be regarded as also the causes of eclampsia. As regards the proximate cause of the uræmic intoxication in Bright's disease, conjectures only can at present be expressed, for acute Bright's disease is not always accompanied by uræmia and eclampsia. Of 100 cases of acute Bright's disease, only from 60 to 70 are seized with uræmic eclampsia. The quantity of urea retained in the blood, the extent and intensity of the disease in the kidneys, but not its duration, may, in this respect, be of the greatest importance.

Just as Frerichs has tried to explain the production of uræmia by supposing the existence of some ferment, so does Litzmann by the beginning of the act of parturition. But opposed to this last hypothesis is the frequent occurrence of eclampsia during pregnancy and after childbirth. According to my own statistical data, and those of Wieger, these last kinds of cases are as numerous as those during labour; viz., twenty-four per cent. before the commencement of pains; fifty-four per cent. during labour; twenty-four per cent. after the birth of the child.[1] Accidental diseases can hardly be considered as causes of eclampsia, because no connection between them can generally be observed. The commence-

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[1] [The really preponderating importance of the act of parturition as a proximate exciting cause of eclampsia, is concealed by the bareness of Dr Braun's statistical statement. No doubt, as he and Wieger make out, cases of uræmic convulsions, during pregnancy and childbed, may be as numerous as those occurring during parturition. But when it is remembered that pregnancy lasts nine months, and the childbed condition for about six weeks, while parturition does not occupy a single day, the bare statistical statement loses much of the force which Dr Braun would attribute to it. To be fair, it should be made thus:—During parturition, a process occupying less than one day or than half a day, there occur as many cases of uræmic eclampsia as happen during pregnancy and childbed, occupying about 320 days. In this way, the immense importance of labour as a cause of convulsions is at once evident, and the use of the name "Puerperal Convulsions," ordinarily applied to the disease in our language, is to a great extent justified. That name, strictly used, applies only to cases occurring during labour; but in obstetric works it is generally made to include cases occurring at any time during pregnancy, parturition, or childbed.]

ment of an abnormal puerperal process[1] cannot be considered as a cause of eclampsia; because, among eclamptic cases fatal in childbed, the existence of an abnormal puerperal process can be demonstrated only in exceptional instances, and the frequency of eclampsia does not increase with the spreading of epidemics of puerperal fever.

The disappearance of œdema, which sometimes precedes the eclamptic seizure, is considered by Frerichs as specially increasing the liability to uræmia, because the purification of the blood is arrested by the cessation of the serous exudations, and so much the greater quantity of urea will be retained in a short time.

Among the accidental causes of eclampsia, are violent emotions, fright, anger, grief, overloading of the stomach (Litzmann, Chaussier), asphyxia (Benedix), and climatic influences.

Cold,[2] abuse of alcoholic drinks, bad food and clothing, can hardly be accused of being frequent causes of Bright's disease and eclampsia.

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[1] *Puerperal-process* is a very indefinite German word used by Dr Braun. We have translated it *puerperal process* or *abnormal puerperal process*. It is intended to imply "any anomaly of childbed, in which the typical powers of the female organism are exerted under extraordinary circumstances."—*Clinik der Geburtshilfe und Gynækologie*. Von Drn J. Chiari, Carl Braun, und J. Spæth, p. 424.

[2] [I have lately been able, in several cases, to attribute the occurrence of slight and temporary attacks of nephritis, in the early weeks of suckling, to exposure to cold. These attacks have been, in some cases, accompanied by all the symptoms of a weed, and followed by slight œdema of the ankles and legs. In all the cases, the urine presented fibrin cylinders under the microscope, and was in some proved to be albuminous. This nephritis, no doubt, offers the key to the pathology of many cases of what has hitherto been considered a simple weed or ephemeral fever, and gives some support to the opinion, that in no case is weed a simple feverish attack, but a fever symptomatic of the onset of an inflammation. In many instances, the inflammation is in the mamma or uterus. Such cases are not characteristic examples of the weed. Inflammation of the kidneys will still further curtail the category of characteristic examples of this disease.

In the following instance, eclamptic convulsions were evidently the result of a temporary nephritis produced by cold. Mrs M., ten days after her confinement with her sixth child, went out of doors for the first time. It was an extremely cold and bitter day, coming after a few days of prematurely fine and mild weather in spring. She was soon seized with violent headache and pain in the pit of the stomach, and corresponding part of the back. On Monday, the day of exposure to cold, she passed water as usual. But after going to bed that evening, it was entirely suppressed for more than twenty-four hours. On Tuesday, she took the first fit at about 9 A.M.; another came on about 11 A.M.; another at 3 P.M.; a fourth at 7 P.M.; and the last, a slight one, about 1 A.M. of Wednesday. Between the fits she was almost completely comatose. Her tongue was much bitten. Chloroform, given during the fourth fit and in small quantity, seemed to aggravate the stertor and lividity of countenance. Purgatives, doses of calomel, and repeated doses of sweet spirits of nitre, were administered during Tuesday, and at midnight they acted copiously; the action of the bowels preceding for some time the discharge of urine. After this there was only the slight fit of Wednesday morning. She rapidly became sensible, and quickly recovered completely.

The urine of Wednesday morning contained only a very slight trace of albu-

Among the more remote causes may be included the peculiar changes in the blood of pregnant women, the pressure of the pregnant uterus, and the stoppage of the venous blood in the kidneys caused thereby; for it has been proved by experiments, that this produces an escape of albumen, fibrin, and blood, into the tubuli uriniferi. Of cases of eclampsia, eighty per cent. occur in first pregnancies, in which, on account of the greater resistance of the abdominal walls, a powerful counter-pressure on the kidneys is generally produced. In cases of repeated pregnancy, the pressure connected with a plural pregnancy, with deformed pelvis, hydramnios, large size of the fœtus, and a high position of the womb, are frequently met with where eclampsia occurs. A very painful delivery does not always precede the appearance of eclampsia.

Accumulation of urine in the ureters, and in the pelves of the kidneys, may of itself, and without direct pressure of the womb on these organs, produce uræmic eclampsia, probably by the resorption of carbonate of ammonia; as also, decomposed urine retained in the bladder, as I once[1] had opportunity to observe in a case of retroversion of the uterus, and as Picard[2] also has witnessed in a young man suffering from stricture of the urethra.

Dubois has directed attention to the circumstance, that obliquity of the pelvis and rickets may be predisposing causes of eclampsia. The possibility of this cannot be disputed; but, generally speaking, eclampsia is rare among cripple women.

Faulty position of the child is so seldom found in eclampsia, that we are not warranted in assuming a causal connection between the two.

The commencement and continuance of labour cannot well be considered a cause of eclampsia, since in forty-eight per cent. of the cases it occurs during pregnancy and in child-bed.

The cases in which albuminuria occurs, during the first half of pregnancy, cannot be referred to pressure on the renal veins, but may, according to Litzmann, be traced back to catarrhal irritation of the urinary passages; or, in the case of really existing Bright's disease, to a complication existing before the pregnancy; and here mistakes may easily arise from the admixture with the urine of a purulent mucus.

Congestion of venous blood in the kidneys is, however, not the only cause of Bright's disease; for among cases of eclampsia, there are 20 per cent. of premature births; and abortions, even in the fifth and sixth months of pregnancy, having fatal terminations, have been observed by Velpeau, Harris, Pätsch,[3] and others.

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men. Fibrin cylinders were found in it in great abundance, and also some crystals of urate of ammonia. On Thursday, the albumen slightly increased, and in two days afterwards disappeared. The urine at this last time was very turbid, contained few fibrin cylinders, some brown epithelial masses from the kidney, and a little urate of ammonia and uric acid.

The influence of cold, as a cause of Bright's disease, is clearly pointed out by Dr Christison, and in a manner strongly corroborative of the above remarks on weed. See his work on Granular Degeneration of the Kidneys, p. 108.]

[1] Braun: *Klinik der Geburtshilfe*, etc., S. 322.

[2] Picard: *Gaz. de Strasbourg.* 1855, Nr. 7.

[3] Pätsch: *Verh. d. Berliner Ges. f Geb.* 4, *Jahrg.*

It is reserved for future inquirers, to answer the question whether or not eclampsia of the first half of pregnancy appears always contemporaneously with Bright's disease.

The circumstance, that after the womb is evacuated, and at the same time the impediment to the free circulation of venous blood is removed, the albuminuria disappears with surprising rapidity—a fact known to Rayer, and at present contested by no one—clearly shows the intimate connection between the two, and furnishes also the key for explaining, what has been asserted by several obstetricians, how even the most violent eclampsia generally soon ceases, if, after a few fits, the practitioner succeeds in cautiously emptying the womb.

Frerichs has directed attention to a second and more subordinate cause, viz., the altered condition of the blood of pregnant women, and specially pointed out the increase of water and of fibrin (hydræmia, hyperinosis), the diminished quantity of albumen (hypalbuminosis), diminution of the quantity of red (oligocythæmia) and increase of colourless corpuscles (leukæmia).[1]

It is quite in accordance with this, that, as general observation attests, the majority of pregnant women affected with albuminuria have a chlorotic aspect, a strange paleness of the lower limbs, a bloated and frequently reddened face, and the other appearances of chlorosis.

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[1] [In the etiological chapter, it appears to me that Dr Braun has made a great omission in neglecting that portion of the study of the causes, which may very well be called the theory of the disease. Convulsions, of whatever kind, are induced by some nervous action. In eclamptic convulsions, the explanation of the function of the nervous system belongs to the theory of the disease. The term theory is very justly used here, as indicating the position such disquisitions, most valuable as they are, should hold. While Dr Braun has neglected these portions of his subject, Dr Tyler Smith has, in my opinion, placed too much and too exclusive weight upon them, as the causes of the disease, in his recently published and excellent lectures in the *Lancet*. (See vol. ii., 1856, p. 451.) The subject is too extensive to be entered upon here. I shall content myself with referring the reader to the works of Marshall Hall and Carpenter, on the physiology of the nervous system in health and disease, and more especially to Dr Tyler Smith's volume on "Parturition and Obstetrics." I may add, in one word, that in uræmia, the most important point is the circulation of a morbid fluid in the nervous system, which probably does not act as a direct excitant of the convulsive motions, so much as it increases the irritability of the nerves, and the consequent liability to convulsions from exciting causes which, under other circumstances, would produce no noticeable disturbance. Ingenious experiments have, as is well known, been performed on frogs, which seem to demonstrate an analogous condition to exist under poisoning by strychnia, at least when moderate quantities of the poison are administered.]

CHAPTER VI.

PATHOLOGICAL ANATOMY OF URÆMIC ECLAMPSIA.

The autopsy of the bodies of those dying of uræmic eclampsia reveals the following conditions:—

In the brain we most frequently find anæmia, œdema, and diminished consistence, even when no evacuation of blood took place during life. Hyperæmia of the membranes is not frequently met with, and still more rarely inter-meningeal apoplexy, which Helm[1] and Kiwisch[2] consider, very justly, as a secondary phenomenon produced by impeded circulation of blood, and which Litzmann considers a result of the uræmia.

Examinations of the spinal cavity have only rarely been made. Bluff once found much serum in it.[3]

In the lungs œdema is constantly found, and sometimes emphysema, as was long ago observed by Bōer, and is at present regarded as always a secondary result of the fits.

The heart is commonly empty and flaccid.

The spleen exhibits the large dimensions it possesses in pregnancy and child-bed.

In the kidneys there is most constantly found one or other stage of the three forms of Bright's disease, so minutely described by Frerichs.

In the first stage, that of hyperæmia and commencing exudation, the surface of the kidney is smooth, the capsule is easily removed, the plexus of veins on the surface of the kidneys is dilated and full of dark blood.

The cortical substance is brownish-red, soft, and friable; from the surface of a section there flows a sticky bloody fluid, with which the parenchyma is infiltrated.

The pyramidal masses are likewise hyperæmic, and their injection is striped. The mucous membrane of the pelves and infundibula is swollen, and covered with vascular arborescence; and they contain a bloody fluid. Apart from hyperæmia, the finer structures of the kidneys do not appear to be essentially injured. Hæmorrhagic effusions are very frequently observed, which sometimes take their rise from the glomeruli, sometimes from the vascular plexus of the tubuli uriniferi, sometimes from the veins on the surface of the cortical substance.

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[1] Helm, Th.: *Med. Jahrbücher.* Wien, 1839, Bd. xx., S. 202.

[2] Kiwisch: *Beiträge z. Geburtsk.* Würzburg, 1846.

[3] [The serum found in various parts of the body often contains urea. See Christison on Granular Degeneration of the Kidneys, p. 8, etc.]

The generally regular, round vesicles in the cortical substance originate in convoluted tubuli uriniferi being distended with blood.

The epithelium of the tubuli uriniferi is, in the first stage, not yet essentially altered, but may generally be distinguished by the ease with which it is separated. The tubuli uriniferi are filled with coagulated or fluid exudation, appear as homogeneous transparent cylinders, and sometimes contain blood-corpuscles. These so-called fibrin cylinders occur less frequently in the chronic form of the disease. By their presence Bright's disease is, in the dead body, distinguished from simple hyperæmia. But hyaline fluid exudation cannot as yet be demonstrated, either by dissection or experiments of coagulation (Wedl).

The second stage, that of exudation, and of the commencing fatty metamorphosis of the same, is characterized by this, that the cortical substance appears of a dull yellow colour, the striped vascular ramifications and the red spots in it disappear. The kidney is bulky, and far exceeds its normal weight.

It then gets softer, more friable, milky, and dark. Its surface appears sometimes smooth, sometimes granulated, covered with elevations of the size of a poppy seed. The latter is the case when single tubuli uriniferi, whose walls lie on the surface, are excessively distended with exudation.

The capsule of the kidney can likewise be easily separated. The pyramidal masses are dark red. The infundibula have a dirty-red mucous surface. The glomeruli (vascular knots, Malpighian corpuscles), which may be drawn out with a curved pin, are covered with a fine granular matter, and partly with solitary or grouped fatty corpuscles, which, by the addition of acetic acid, become transparent. Between the glomerulus and the capsule lies a thick stratum of firm exudation, of granular structure, and having fat droplets, and sometimes crystals of cholesterine.

Fig. 2.[1]

The interior of the epithelial cells of the tubuli uriniferi is, in extreme cases, filled with fat droplets, becomes turbid, and at last the cells themselves are decomposed into aggregations of granules, *i.e.*, fatty degeneration of the contents of the epithelial cells of the tubuli uriniferi takes place.

In the third stage, that of retrogression, and dissolution of the glandular substance (atrophy), the kidneys again become smaller, are reduced to their normal dimensions, and often sink even far below it, down to a weight of three to one and a half ounces. The capsule of the kidney, which appears dirty white, and thickened in some parts, is closely united to the cortical substance, may be drawn off, but with difficulty, and in so doing, parts of the cortical layer are removed with it.

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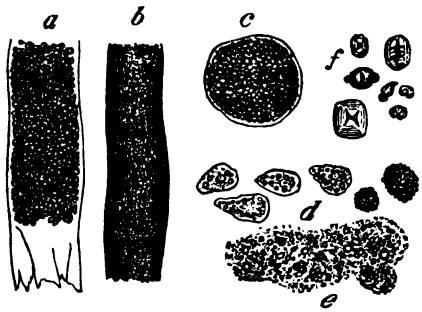
[1] Fig. 2. Malpighian corpuscle, with granular covering, from the kidney of a person who died of eclampsia parturientium (after Wedl). Magnified 300 times.

The surface of the kidney has lost its smoothness, is become uneven and tuberculated, and often shows deep, furrow-like indentations, dividing it into lobes.

The colour of the surface of the kidney is, in general, dirty yellow, the cicatrix-like indented parts commonly pale, in some cases of a bluish-black colour, from old extravasations of blood. Generally some parts of the organ are found which still retain their normal brown coloration. The consistence of the kidney has lost its former friability; instead of that, it acquires a toughness like that of leather, which is the more marked the more the atrophy increases.

The surfaces of sections of the organ show a greater or less disappearance of the cortical substance.

Fig. 3.[1]



In the depth of the cortical layer, just as on the surface, granulations of different sizes are seen.

The renal tissue of the cortical layer becomes atrophic, from the circumstance, that the tubuli uriniferi collapse, as soon as, in consequence of the fatty degeneration and expulsion of the exudation, they are completely deprived of epithelium; while, at the same time, the capsules of Malpighi shrink up after the obliteration of their vascular structures. Of the destroyed tubuli uriniferi only the basement membrane remains, which then crumples up.

In some rare cases, when, during the first two stages, a part of the exudation passes into the interstitial tissue, it becomes more or less completely organized into connective tissue, which surrounds the tubuli uriniferi and Malpighian capsules with concentric layers, and by a cicatrix-like contraction affords an essential cause of the production of atrophy. When Bright's disease assumes a chronic form, it is only very rarely that we find the radiated colloid bodies of Wedl, which represent a part of the cystic contents of atrophic kidneys.

The atrophy of Malpighi's and Ferrein's pyramids is less than takes place in the cortical layer. At their basis, we find granulations interspersed between the straight uriniferous tubules, which are pressed apart from each other.

The pelves of the kidneys are generally wider than natural. Their mucous

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[1] Fig. 3. Fatty degeneration of the epithelium of the tubuli uriniferi (after Wedl). *a.* Tubulus crammed with greater and lesser fatty globules; the membrana propria is seen folded downwards; *b.* Tubulus filled with small fatty globules, in some parts quite opaque; *c.* Transverse section of a tubulus, the transparency destroyed by the accumulated fat globules; *d.* Transition forms of the fatty degenerated epithelial cells; *e.* Contents of several tubuli uriniferi, the basis hyaline, with superadded fatty globules; *f.* Imperfect brownish-yellow crystals of uric acid from an urinary tubulus. Magnified 350 times.

membrane is swollen, and pervaded by varicose vessels of an uniform greyish-blue colour. The layer of fat in which the kidney is imbedded diminishes in quantity when atrophy of the gland commences.

In those who die of uræmic eclampsia during pregnancy, atrophy of the kidneys is less frequently observed than the first two stages of Bright's disease.

CHAPTER VII.

PROOF OF THE INTIMATE CONNECTION BETWEEN ECLAMPSIA AND URÆMIA.

THE theory of the identity of uræmic intoxication in acute Bright's disease and puerperal eclampsia has been energetically defended by Frerichs, Litzmann, the author, Wieger, Oppolzer, and many others; but it has been assailed by Marchal,[1] Siebert,[2] Depaul,[3] Legroux,[4] L'Huillier,[5] Stoltz, Seyfert,[6] Levy,[7] in very valuable articles, and also by Scanzoni.[8] With much anxiety, these have tried to prove that the Brightian degenerations of the kidneys, which, it cannot be denied, are found in the bodies of those who have died of eclampsia, are consequences merely of the convulsions, only accidental secondary phenomena of the hyperæmias caused by the eclampsia, and of hydræmia (plethora serosa).

The result of analytical investigations is thus summed up by Scanzoni:—

1. In the most recent times, the post-mortem examinations of persons dying of eclampsia have shown, only in a minority of the cases, so profound a degeneration of the kidneys as to justify the diagnosis of Bright's disease. (?)

2. It is not proved that albumen in the urine and the presence of fibrin-cylinders always precede the outbreak of the convulsions. On the contrary, there are circumstances which show that this anomaly is frequently developed for the first time during the delivery or the convulsions. (?)

3. The arguments which have been brought forward to prove that uræmic intoxication has taken place, are not by any means equally strong arguments for our holding that the true eclampsia parturientium is always the result of uræmic intoxication originating in Brightian degeneration of the kidneys. (?)

4. Eclampsia puerperalis presents general clonic convulsions of the voluntary

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[1] Marchal: *Gaz. des Hôpit.* 1851, Dec.
[2] Siebert: *Deutsche Klinik.* 1851, Nr. 44.
[3] Depaul: *Bulletin de l'Academie.* 1854, Nr. 7.
[4] Legroux: *Die Eclampsia albuminurica (L'Union* 87, 1855*).*
[5] L'Huillier: *Gaz. de Strassbourg.* 1854, Nr. 3.
[6] Seyfert: *Wien. mediz. Wochensch.* 1853, Nr. 12.
[7] Levy: *Hospitals—Meddelelser Bd.* 4, H. 4, *Schmidt's Jahrb.* 1853, Nr. 4, S. 49.
[8] Scanzoni: *In den klinisch. Vorträg. uber spec. Path. u. Ther. d. K. des weibl. Geschlechtes. Von Kiwisch v. Rotterau. Prag.* 1854, S. 436, III. Bd.

muscles, proceeding from the spinal cord, with removal of consciousness. These convulsions have their immediate cause in the irritability of the motor system of nerves which has been induced by pregnancy, and increased by the act of delivery. (?)

Wieger[1] and Litzmann[2] lost no time in answering, as early as last year, the objections just mentioned, and in confirming by new observations the identity of eclampsia vera and uræmia. As both of these gentlemen have most extensively availed themselves of the most recent literature on the subject, in order to vindicate this doctrine, it only remains for me to adduce a few additional arguments in proof of it.

I. On taking a wider survey of the literature of the subject, we find that the post-mortem examination of those who have died of eclampsia shows extremely often, and in an undoubted manner, the existence of Bright's disease of the kidneys.

Of the 45 cases of eclampsia published by me, fifteen terminated fatally; but post-mortem examination was made only in twelve. In seven of these cases, which are described under atrophy, fat-metamorphosis, diffuse nephritis, and œdema of the kidneys, Bright's degeneration of the kidneys was always found by the microscopic examinations of Wedl and others; and it was only for the sake of brevity that I made use of the above expressions. In the other cases there was hyperæmia of the kidneys, but from accidental obstacles no microscopic examination was made. This, however, cannot be considered any proof of the absence of Bright's renal exudation.

None of the nine cases of eclampsia observed by me during the last three years ended fatally, and therefore I cannot here refer to them in support of my view. According to the observations communicated to me by Gustav Braun, six cases of eclampsia had a fatal issue during the last three years in the first midwifery clinique of Vienna; and in three of them the second stage of fat-metamorphosis of kidneys affected with Bright's disease was demonstrated beyond a doubt.

According to Wedl,[3] who has carefully described the histology of Bright's kidney, the circumstance that in several cases of death from eclampsia no fatty metamorphosis of the kidneys can be found, may be accounted for by the fact that in many cases a dissolution of the Malpighian bodies is effected by the fluid exudation; and hence in every diffuse inflammation of the kidneys an evident fat-metamorphosis of the contents of the Malpighian capsules does not ensue.

Lumpe[4] has published a case in which eclampsia occurred in the first, second, and fifth deliveries. Three hours after the last, death took place, and on a

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[1] Wieger, F.: *Recherches critiques sur l'éclampsie urœmique. Gaz. méd. de Strasbourg.* Nr. 6–12. Juin, 1854.

[2] Litzmann in Kiel: *Ueber den ursächlichen Zusammenhang zwischen Urämie und Eclampsie i. d. deutschen Klinik.* Berlin, 1855.

[3] Wedl: *Grundzüge der pathol. Histologie. Wien,* 1854. S. 306.

[4] Lumpe: *Zeitsch. d. Gesellsch. d. Aerzte zu Wien.* 1854, August.

dissection being made, there was demonstrated beyond any doubt, in the left kidney, the second stage, that of fat-metamorphosis; and in the right kidney, the third stage, that of atrophy of Brightian kidneys.

Hecker,[1] in two cases of eclampsia ending in death, found in one, atrophy of the left kidney, and recent Brightian fatty metamorphosis in the right; in the other, both kidneys presenting an exquisite example of Bright's disease in the stage of fat-infiltration.

Devilliers and Regnauld, in four cases of eclampsia with fatal issue, found granular degeneration of the kidneys, and Simpson[2] has in three cases found the same on dissection. Blot,[3] Cahen, Wieger, Litzmann, Credè[4] have, each in one case, Sabatier[5] and Hohl,[6] each in two cases, found the Brightian fat metamorphosis of the kidneys after eclampsia.

Scanzoni has been able to discover, only in one case, traces of exudative nephritis. Hasse never saw eclampsia puerperalis without Bright's disease. There are above thirty cases known to me, offering positive proofs of the intimate connection between Bright's disease and eclampsia, and they are far too numerous to admit of the opinion, that there is only an accidental connection between the two, being still entertained. Their value as arguments is very much enhanced by the circumstance, that in negative observations the histology of the kidneys was examined microscopically only in very rare instances.

II. Acute Bright's disease is the first link of a chain of morbid changes leading on to puerperal eclampsia.

This view is supported by the following arguments founded on experience:—

a. In an observation made by Oppolzer[7] and myself, we prognosticated the outbreak of eclampsia, two days before its occurrence, from the presence of abundant albuminuria, and from the blood containing a large quantity of urea.

b. Devilliers, Regnauld, the author,[8] Litzmann, and Wieger have published a complete series of observations, in which albuminuria and exudation-clots occur along with acute Bright's disease during pregnancy, lead to spontaneous premature labour under different uræmic phenomena, and then terminate in speedy recovery, having no further injurious consequences, and producing no eclampsia.

The circumstance that eclampsia does not occur in every case of Bright's disease during pregnancy, may, as Litzmann has very correctly remarked, be accounted for by this result following only when the blood has been very consi-

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[1] Hecker: *Verhandl. d. Gesellsch. s. Geb. in Berlin.* VII.
[2] Simpson: *Obstetric Memoirs and Contributions.* Edinburgh, 1855. P. 821.
[3] Blot: *L'Union.* 1850, Nr. 122.
[4] Credè: *Klinische Vorträge über Geburtshilfe.* Berlin, 1854, S. 484.
[5] Sabatier: *L'Union Méd.* 1853.
[6] *Lehrb. f. Geb.* 1855, S. 403.
[7] *Clinik der Geburtshilfe, etc.* S. 352.
[8] C. Braun: *Klinischer Bericht aus Trient.* 1854. *In Scanzoni's Beiträgen,* Bd. 3, S. 20. Würzburg, 1855.

derably impregnated with the excrementitial elements of the urine,[1] which always implies a very profound, or at least extensive, disease of the renal tissue.

c. When the urine is examined for the first time after the appearance of eclampsia, as is the general rule, and after one or at most two fits, the urine is found charged with albumen to such an extent that, on boiling, the whole mass not unfrequently clots into a yellowish-white coagulum; and the microscope discovers in the fluid a large quantity of exudation clots covered with abraded glandular epithelium, already in part undergoing fatty degeneration, or even passing into detritus. No one can really believe, as Litzmann has strikingly pointed out, that a disease in the kidneys, furnishing such products, has been just developed within the last few hours.

d. A complete solution of this question, sufficient to silence the most obstinate doubter, will be arrived at only when the urine of pregnant women in lying-in hospitals shall have been submitted to an exact chemical examination. But even from the observations already made, we may assume that it is certain that acute Bright's disease often precedes eclampsia, and that it is a probability bordering on certainty that a connection exists between the two.

e. Erroneous inferences have been drawn from the circumstance, that albuminuria occurs more frequently among parturient and lying-in women than among the pregnant. For, in this argument, cases in which the urine contains only small quantities, or even only traces, of albumen without admixture of exudation clots, and in which it will occur to no one to think of the possibility of uræmia, have been classed as equally important with those where so large an excretion of albumen and cylindrical clots takes place as is constantly found in eclamptic patients. Litzmann was the first correctly to appreciate this occurrence of traces of albumen among lying-in women as a catarrh of the bladder, on account of the simultaneous presence of pus globules, and the absence of exudation clots. He believes, also, that this cannot be regarded even as a symptom of commencing Bright's disease. Ludw. Mayer,[2] on the other hand, inferred from the albuminuria, with microscopically demonstrable exudation clots, which appeared during

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[1] [On this subject, see Johnson on Diseases of the Kidney, p. 198. "It is (he says) in the highest degree probable that urea is a poisonous agent, but we have no proof that it is more so than other urinary constituents, which must, with the urea, be retained and accumulate in the blood, when the kidneys are so much disorganized as they are often found to be. Dr Christison states, that in some cases the daily discharge of solids by the urine may, for weeks together, be reduced to one-fourth of the natural amount, without any symptom of an affection of the head supervening, and, moreover, when an analysis of the blood shows that it is loaded with urea. Dr Bright relates a case to the same purpose. A person labouring under disease of the kidney, lived for four or five years under his occasional observation. The blood was analyzed in the earlier stage, and found to contain a large quantity of urea; yet this patient had no fits till towards the close of his life. And Dr Rees states, that he examined the blood of a patient who had his senses about him to the last moment of his life, and whose blood was more impregnated with urea than that of any case of Bright's disease that ever came under his notice."]

[2] Mayer, Lud: *Analecta ad gravidarum, parturientium et puerperarum albuminuriam. Dissert. inaug. Berol.* 1853.

childbed in a woman operated on by me, that Bright's disease occurred during childbed. But I cannot at all agree with him, for in the case referred to in consequence of an abnormal puerperal process, a limited fibrinous exudative action, a so-called metastatic secondary deposit, was forming in the kidneys; for which reason this observation cannot in any way be used to invalidate the theory of the identity of Bright's disease and eclampsia.

f. As respects the commencement of Bright's disease during labour, specially as regards the sudden and abundant appearance of albumen, and of large quantities of fatty degenerated cylindrical clots, no convincing microscopical observations have been anywhere published. And even if these phenomena were demonstrated, they would prove only that Bright's disease may begin in every stage of the function of reproduction.

g. According to my own observations, and those of Frerichs[1] and Hasse[2], the most violent attacks of epileptic and hysterical convulsions, even if occurring several times in one day, never have, as a consequence, albuminuria or excretion of cylindrical clots. The remark made by some, respecting the occurrence of traces of albumen and exudation clots after epileptic fits, does not prove that Bright's disease can be induced by a secondary hyperæmia of the kidneys, the result of the fit. For as yet no case is known in which there was no trace of renal exudation immediately before an epileptic fit, but after it large quantities of albumen and cylindrical clots. Exceptionally, in women affected with habitual epilepsy, eclampsia may supervene, and Bright's disease be at the same time demonstrable during pregnancy, labour, or childbed; and it may happen that an uræmic eclampsia may even yet, on account of some uncommon symptoms, be not very rarely designated epilepsia renalis, as was very lately done by Robert B. Todd.[3]

h. Eclampsia appearing in childbed originates in Bright's disease being already developed during pregnancy, as several observations prove that have been recorded by Simpson, Lumpe,[4] Hecker, Duchek,[5] Wieger, the author, and others. Leudet[6] observed albuminuria to appear during pregnancy, eclampsia during delivery, the albuminuria continuing for two months, and at that time violent eclampsia again return.

i. A case of retroversion of the uterus published by me[7] and Bamberger ended fatally under eclamptic attacks, in consequence of Bright's degeneration of the kidneys and secondary uræmia. A similar case was observed by Picard[8] in a man affected with stricture of the urethra, who died of albuminuria and eclampsia, and in whose dissection Brightian fat-metamorphosis of the kidneys was demonstrated.

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[1] Frerichs: *Wiener mediz. Wochenschrift.* 1854, S. 469.
[2] Hasse: *In Virchow's Path. u. Therap.* Bd. IV., Abth. 1, S. 254.
[3] *Med. Times and Gaz.* 1854, August.
[4] Lumpe: *Wiener mediz. Wochenschrift.* 1853, Nr. 51, and 1854, Nr. 29–31.
[5] Duchek: *Prager Vierteljahrsschrift.* 1853, Bd. X. 1.
[6] Leudet: *Gaz. hebdomadaire.* 1854, I. 28.
[7] Braun: *Clinik der Geb. etc.*, S. 322.
[8] Picard: *Gaz. de Strasb.* 1855, Nr. 7.

j. The first stage of Brightian kidneys, in which the exudation in the tubuli uriniferi is still fluid, and cannot be demonstrated by the microscope, or when the coagulated exudation appears periodically in the urine in large quantities as cylindrical clots, and then again disappears for some time, must be regarded as a blood-poisoning disease, equally dangerous as the fatty metamorphosis of the kidneys. For Brücke's[1] ingenious investigations on the causal connection of albuminuria and uræmia, have shown that the occurrence of uræmia depends not so much on the *intensity* of the textural charges as on the *extent* of the morbid exudation in the kidneys.[2]

Brücke refers to a discovery formerly made by himself, according to which, when, through the shell-membrane of an egg, water is separated from a solution of albumen, the salts of the latter may go through with the water without being accompanied by the albumen itself; as also to a discovery of Valentin's, that if, under hydrostatic pressure, albuminous solutions are passed through animal membranes, the part transmitted contains less albumen than what remains. Hence, he thinks that the membrana propria of the tubuli uriniferi and of the Malpighian capsules is so constructed, that in its normal condition, and under the ordinary pressure of the blood, only those portions of albumen necessary for nourishing the epithelium pass through, and that the same is true of the fibrin.

Now, when from any cause the covering of a glomerulus allows albumen to traverse it, the urine secreted in the capsule forms already a concentrated solution (normally, it is very attenuated). The diffusion in the tubuli uriniferi (by means of which, in the normal condition, a state of equilibrium of the conditions of contraction is approached) will now no more have its ordinary intensity; and hence urea, salts, etc., will no longer impregnate the urine in the usual way. The relative diminution in quantity of the various substances will be different. Analyses hitherto performed show a decrease chiefly of the urea (and of the chlorides).

The reaction of the urine is less acid, as the albuminoid substances in their combinations with alkalis are no longer retained or kept back.

The quantity of the urine may be increased, or normal, or diminished. The mere escape of albumen along with the urine will in itself increase the quantity of the latter, because the albumen opposes the process of diffusion, which concentrates the urine. But the fibrin, which is secreted at the same time, and which mostly coagulates in the capsules and tubuli uriniferi, and obstructs them, opposes the secretion of urine; and a similar influence is exerted by partial stases, and destruction of individual parts of the tissue of the kidneys. The quantity of urea secreted within twenty-four hours is still more variable than the

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[1] Brücke: *Zeitsch. der Wiener Aerzte.* 1855. *Heft.* 1, XI.

[2] [Dr Christison, also, points out that coma and convulsions may come on in the very earliest stage of Bright's disease, and that then, indeed, they advance more rapidly than when the degeneration is more advanced. He also mentions their occurrence independently of any dropsical effusion, and their occasionally coming on shortly after dropsy has been dispelled.—*Granular Degeneration of the Kidneys*, p. 93, etc.]

quantity of albumen; sometimes quantities of urine, apparently sufficient and with moderate impregnation of albumen, are evacuated, and after a short time uræmia appears; at other times the urine is in small quantity, much loaded with albumen, and uræmia does not make its appearance.

These apparent contradictions are to be accounted for by the size of the kidneys exceeding the ordinary wants. It is only in those cases where the transudation of albumen takes place everywhere, that the process of diffusion in the tubuli uriniferi is everywhere disturbed, and accumulation of urea in the blood takes place if that condition continues, and if the diminished quantity of urea is not compensated for by the quantity of urine.

The gradual impoverishing of the blood in albuminous substances is in itself favourable to the occurrence of uræmia, only from the activity of the process of diffusion diminishing while the hydræmia increases. The process of diffusion would altogether cease, if blood and urine contained equal quantities of albumen at the same time.

In a theoretical point of view, it therefore seems advisable in such cases to give the patient as much to drink as possible, so that, under this relatively diminished quantity of urea in the urine, through the excretion of greater proportional quantities of urine, the blood may be protected from accumulations of urea, which can naturally only ensue under certain grades of attenuation.

k. Further, if we ascribe any value to the careful chemical researches of Mialhe,[1] we find in them sufficient grounds for the opinion that copious albuminuria is not a product of hydræmia, but a primary exudation from the kidneys.

According to this view, and that of Lehmann, albumen proper is incapable of endosmosis, since in serum, as in white of egg, it is in a molecular and merely nascent condition. Ordinary albumen, coagulable by heat, and precipitable by nitric acid, is to be distinguished from albuminose (peptone of Lehmann), which does not exhibit this reaction, but is precipitated by the well-known metallic salts only. Between these two, Mialhe makes out an intermediate condition of the substance, in which it is not coagulated by heat, but is so precipitated by nitric acid as to be redissolved by an excess of it. This last modification is formed from ordinary albumen, not only in the commencement of the operation of natural or artificial gastric juice on it, but also in great attenuations of ordinary albumen under the simultaneous influence of heat, or by miasmatic or poisonous materials which may have got into the blood. The passage of one or other of these three modifications of albumen into the urine, or other animal fluids, is different in various forms of disease. Albumen proper appears only in secretions and excretions in true inflammations, and generally where real destruction of tissue is taking place; and hence only in acute Bright's disease, or degeneration of the kidneys.

Modified albumen, which Mialhe has called caseiniform, is capable of endosmosis; it is formed when the blood is greatly attenuated, whether that has been brought about by great fluid discharges or by sudden suppression of the function of the skin, as after scarlatina, erysipelas, etc., and in the chronic

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[1] Mialhe: *Chimie appliquée à la physiologie et à la thérapeutique.* Paris, 1856.

form of Bright's disease; and hence the observation, frequently made, that the albumen of urine in Bright's disease, when precipitated by nitric acid, is again dissolved by an excess of the same.

In the normal condition, albuminose appears in all the secretions and excretions, but only in small quantity, as it is assimilated in the blood.

In cholera, according to Mialhe, a fermenting substance is formed in the blood, which, like pepsin, changes normal albumen into albuminose. In forming a prognosis in cholera, it is hence of great importance to try whether the dejections contain albumen that may be precipitated by boiling, or at least by nitric acid, or only albuminose. In cases which prove fatal, the latter only is generally found.

l. An association of London physicians[1] holds those forms of eclampsia, which they call renal puerperal convulsions, to be a consequence of hyperæmia of the kidneys, and blood-poisoning produced thereby. Pregnancy, according to them, produces an increased necessity for purification of the blood through the congested kidneys, and in this way defective secretion of poisonous excrements from the blood—*i.e.*, toxæmia—is induced. The direct influence of this morbid blood on the brain, spinal cord, and medulla oblongata, causes convulsions.

m. If we hold, with Frerichs, Litzmann, the author, Wieger, and others, that the ordinary cause of Bright's disease in pregnancy is the retardation of the stream of venous blood in the kidneys from the compression of the venous trunks by the gravid uterus, then it is obvious that in this circumstance we have the cause of the abnormal process of exudation being more or less uniformly spread over the whole organ, without any immediate necessity for its texture undergoing more profound changes. On the contrary, such will probably appear only gradually, and after a long duration of the diseased action. It must always be kept in mind that it is possible to have one kidney only, or at least principally, affected, from the uterus having a peculiar position, as several cases already cited demonstrate.

In this way also, partly, the circumstance may be accounted for, as Litzmann has already remarked, that sometimes, in spite of the intensity of the local morbid process, no signs of uræmia appear, while in other cases uræmia is observed when the disease is much less intense, but probably affects both kidneys, in which case the quantity of the urine does not always undergo a marked diminution.

n. Although the analyses of the urine of pregnant women suffering from Bright's disease, published by the author, Scanzoni,[2] Gegenbauer, Harvey, and others, have shown that there is a considerable diminution of the quantity of urea, and generally also of uric acid, yet it remains for future investigations to decide the exact relations of these changes to the intensity and to the extent of the disease of the kidneys and of the uræmia.

The physiological, chemical, microscopical, and clinical studies here propounded, leave no doubt whatever on this point, that the albuminous contents

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[1] *Assoc. Med. Journ.*, Dec. 8, 1854, S. 1102. [Dr Cormack.]
[2] Gegenbauer: *In Scanzoni's Würzburger Beiträgen.* Bd. I., S. 206.

and the presence of exudation clots in the urine precede the eclampsia and the labour, and that the Bright's disease is the first link of a chain of morbid changes leading on to eclampsia.

III. Eclampsia parturientium is commonly the result of uræmic intoxication arising from Bright's disease of the kidneys, and produced mostly by carbonate of ammonia in the blood, perhaps also by extractive matters of the urine.

This appears from the following analysis:—

a. All observers at present agree that urea retained in the blood is not, as such, the cause of the uræmic symptoms.

b. Lehmann[1] and Frerichs, almost simultaneously and independently of one another, arrived at the conviction that the cause of the uræmic phenomena is to be sought for in the ammoniacal contents of the blood, produced by the transformation of urea into carbonate of ammonia.

c. The investigations of Frerichs, Litzmann, the author, Heller, Kletzinsky, Oppolzer, Gegenbauer, and others, have demonstrated that in the eclamptic, urea and carbonate of ammonia developed by its decomposition, are generally found in considerable quantity in the fresh blood; that from the presence of these materials in the blood the occurrence of uræmic eclampsia may be prognosticated, and that these substances are observed also in the blood of children born of uræmic mothers.

d. Chemical analysis, however, cannot always, even during the most violent eclampsia, discover the presence of carbonate of ammonia in fresh blood, as is shown in an observation made on the 14th August 1854, by Gustav Braun and Heller, and communicated to me. The blood, drawn from a vein after the sixteenth eclamptic fit, separated itself into clear serum and a light red, bulky, moderately consistent clot, covered with spongy fibrin tinged yellow by a gall-pigment. The strongly alkaline serum had a specific gravity of 1·025, and contained much casein and biliphæin. The serum, filtered after being treated with alcohol, contained little urea and sugar, much cholesterin and choleic acid. The blood then had not the uræmic but the cholæmic constitution, as it contained no carbonate of ammonia, but all the elements of bile. This observation, although it does not stand alone, cannot be used to invalidate the theory of the very frequent coincidence of eclampsia and uræmia. It rather points out that the excrementitial elements of bile may, producing cholæmia in the living body, be likewise a cause of eclampsia.

e. According to the very careful experiments of Mettenheimer,[2] Beneke, and Reuling,[3] all healthy and sick individuals have the power of expiring ammonia under certain circumstances. In the lungs of the healthy, ammonia is in general absorbed rather than expelled; and hence the vapours produced on holding before the mouth a glass rod wetted with dilute muriatic acid, are never capable of indicating the degree of uræmia and Bright's disease.

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[1] Lehmann: *Physiol. Chemie.*, II. S. 245.
[2] Mettenheimer, C.: *Archiv f. wissenschaftliche Heilk.*, I. 4.
[3] Reuling, W.: *Inaug. Diss.—Giessen.* 1854.

f. Normal blood, when fresh-drawn, does not contain any ammonia, as Reuling has proved by a very simple, but very delicate, qualitative method with logwood paper.

In several diseases, as caries of the teeth, angina tonsillaris, typhus, pyæmia, ischuria, and blennorrhœa of the urinary bladder, we sometimes find in the blood carbonate of ammonia, just as in uræmia and Bright's disease. Hence carbonate of ammonia in the blood cannot be regarded as a characteristic indication of uræmia, and in many constitutions uræmia may be produced by extractive matters in the blood.

g. After weighing the objections raised against the theory of the intoxication of the blood by carbonate of ammonia, Litzmann has arrived at the following conclusions:—

The fresh blood of healthy individuals never contains ammonia. In the majority of the cases of uræmia, the blood does contain ammonia, which has been formed by the decomposition of urea formed in the blood and retained in it, or by the decomposition of urea that has been secreted into the urinary passages, and has returned into the circulation by absorption.[1]

The presence of ammonia in the blood is indicated by the increased ammoniacal contents of the expired air; but this increase cannot of itself be considered a proof, for it may be produced by the admixture of ammonia formed in the cavity of the mouth by decomposition of the oral secretions, and of remains of the food, in cases of carious teeth, etc.

In the vomited fluids and the contents of the intestine, in cases of uræmia, carbonate of ammonia is not unfrequently discovered. Sometimes there is an alkaline reaction of the sweat (Litzmann, Rühle). The urine may contain ammonia even while it has an acid reaction (Brücke).

But, on the one hand, presence of ammonia in the blood is not by any means a sign of uræmia exclusively, for it has been exceptionally observed (Reuling) in other diseased conditions (typhus, pyæmia), where the urinary secretion was not disturbed. On the other hand, cases undoubtedly occur where, notwithstanding obstructed secretion of urea by the kidneys, and the occurrence of all the characteristic symptoms of uræmia, the blood does not contain any ammonia, and the ammoniacal contents of the exhaled air are not increased (Reuling); but where, on the contrary, undecomposed urea is found in the transudations from the blood, and in the sweat specially, may be in such quantity as to be left on the skin in the form of a white crystalline dust (Schottin,[2] Fiedler[3]).

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[1] [On this point see Christison on Granular Degeneration of the Kidneys, p. 34. "Many specimens (he says) of urine, in this stage are much more prone to decay than the healthy secretion. In some I have observed a decided ammoniacal odour, so soon after its discharge, that in all probability decay had commenced within the body; and, frequently, so much carbonate of ammonia is formed in eight or ten hours, that a powerful ammoniacal odour is exhaled, earthy phosphates are thrown down in abundance, brisk effervescence is caused by acids, and another character, to be stated presently, coagulation by heat, may be prevented from being developed."]

[2] Schottin: *Arch. f. physiol. Heilkunde*, X., XI., XII.

[3] Fiedler: *De secretione urinæ per cutem. dissert. Inaug. med. Lipsiæ.* 1854.

The cause of the uræmic phenomena cannot therefore be sought for in the decomposition only of the urea retained in the blood into carbonate of ammonia.

h. Whether the accumulation of extractive matters in the blood, in consequence of suppression of the urinary secretion, is the cause of eclampsia (Schottin, Reuling) or not, is a question for the future to decide.

Hoppe found, in the case of an uræmic person, besides urea, three times the usual quantity of extractive matters in the serum of the blood, and also the muscles saturated with their excretory products. The quantity of kreatin obtained from them was five times as much as the normal amount.

i. Wieger, on these grounds, regards uræmia not so loosely as the ancients, who held it to be a metastasis of the urine, and not so exclusively as Frerichs, who considers it an intoxication by carbonate of ammonia, but as a consequence of Brightian exudation into the kidneys, which in its chemical relations is characterized by retention of water and excrementitial matters in the blood, which itself, from the loss of blood-corpuscles and albumen, is impoverished in these elements.

But it is uncertain whether the chief part in the combined operation is to be ascribed to the excess of serum, or to the diminution of the albumen and blood-corpuscles.

Hydræmia, however, is never the cause of the nervous symptoms, but has only a predisposing action.

j. From the results of chemical analyses, it is certain that in uræmia carbonate of ammonia and urea are often found in the fresh blood, and are only rarely awanting.

Although the essential nature of uræmia is always coming into clearer light, in consequence of the rapid strides of organic chemistry, yet we know enough already to assure us that eclampsia parturientium is always caused by the presence in the blood of an excess of excrementitial matters, and also generally by uræmic intoxication.

IV. Eclampsia puerperalis is not a consequence of hydræmia, of pregnancy, or of pains. For the following reasons, Bright's disease cannot be regarded as the consequence of eclampsia.

a. Blot's observations show that the average quantity of albumen in the urine is, in albuminuric individuals not suffering from eclampsia, 33 per cent.; in the eclamptic, sometimes 74 per cent. But from the quantity of albumen in the urine, we cannot decide whether eclampsia will occur or not, because the whole quantity of urine secreted in twenty-four hours, and of urea accumulated in the blood, may greatly vary.

b. From the analyses of Becquerel, Rodier, Devilliers, and Regnauld, it appears that the albumen in the blood of pregnant women is diminished 4 per cent., and in albuminuria 16 per cent. But hydræmia and diminished albuminous contents cannot be the cause of the escape of albumen into the urine, because, as Wieger has shown, it would be impossible to explain why hydræmic blood secretes a small quantity of urine,—why blood-globules often, and exudation clots always, appear in the urine,—why the absolute quantity of urea and of extractive matters secreted within twenty-four hours is diminished—why, in

post mortem examinations of eclamptic patients, the kidneys so often exhibit extensive degenerations,—and how it can happen that in cholera, with an early stage of nephritis, condensation of the blood co-exists with excretion of albumen.

c. Depaul has advanced the opinion that albuminuria is a consequence of eclampsia, inasmuch as, when it exists during pregnancy, it disappears a few hours after delivery, while it is often not till some days afterwards that eclampsia begins, and causes the reappearance of the albumen.

Wieger, founding on numerous observations made by the author, Blot, Regnauld, and Devilliers, has controverted this opinion in detailed statistical tables. He shows that, in cases of distinct albuminuria and of eclampsia, the albumen does not disappear a few hours after delivery; that this does not happen till two days at least, and often not till a week after; that it often persists, and even increases; that in women dying comatose the albuminuria continues till death; and that the danger of eclampsia not unfrequently increases with the intensity of the albuminuria.

Dropsies unaccompanied by albuminuria, during the labour, although very extensive, are not accompanied by eclampsia.

d. The cases adduced by L'Huillier, Depaul,[1] Dubois, Mascarel,[2] and the author, in which, during puerperal eclampsia with fatal issue, no traces of albuminuria could be discovered, prove only that in pregnant women convulsions may in exceptional cases be produced by various causes, as meningitis, burns, typhus, capillary apoplexy, habitual epilepsy, anæmia, cholæmia, etc. They do not force us to take refuge, in a manner that cannot be justified, in a mysterious hypothesis of some peculiar condition of puerperality, in hydræmia, leukæmia (Bengel[3]), oligocythæmia, hyperinosis (Oldham), hypalbuminosis, etc., for the explanation of the ordinary form of the far more frequently occurring uræmic eclampsia.

The proofs that hydræmia cannot be the cause of eclampsia, and this again not the cause of Bright's disease in pregnant women, are so manifold, that we stand impregnable on the principles which we have above so comprehensively defended, and must base upon them the rules to guide us in practice.

The fundamental characters of uræmic eclampsia brought out here are the result of careful statistical investigations; and it affords me great pleasure to observe that the distinguished neurologist of Heidelberg, Hasse,[4] recognises the importance of the following propositions:—1. All cases of albuminuria in pregnancy and childbed are not referable to Bright's disease; 2. Only a few cases of Bright's disease go so far as to produce uræmia; 3. It is not a necessary result of uræmia that every one suffering from it will have eclampsia; 4. The appearance of this affection is not necessarily connected with any particular stage or extent of degeneration of the kidneys; 5. The function of labour is not the immediate cause of eclampsia, nor do the spasms during the paroxysms stand in any connection with the coming on of uterine pains.

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[1] Depaul: *Union méd.*, 1854, Nr. 2, 3, 5, 7.
[2] Mascarel: *Bull. de therap.*, 1853, *Août.*
[3] Bengel: *In Merhlingen—Würtemb. Corr. Bl.*, 1855, Nr. 47.
[4] Hasse: *Im Handb. d. spec. Path. u. Ther. Red. v. Virchow.* IV. Bd. 1, Abth. S. 292.

CHAPTER VIII.

DIFFERENTIAL DIAGNOSIS OF URÆMIC ECLAMPSIA AND OTHER AFFECTIONS OF THE MOTOR SYSTEM OF NERVES.

URÆMIC ECLAMPSIA may be distinguished from all other convulsions which may arise from habitual epilepsy, hysteria, apoplexy, meningitis, thrombosis of the sinuses, typhus, poisons, anæmia, cholera, cholæmia, etc., by the following circumstances:—

1. In uræmic eclampsia the urine is rich in albumen and cylindrical clots, deficient in uric acid and urea, and sometimes appears of a red colour from blood globules, or from hæmatin that has been set free; œdematous infiltrations of the face and of the extremities are seldom wanting, but often are only slight; considerable swelling of the spleen is never present except when the disease has been preceded by intermittent fever. The fits come on suddenly, without any nervous symptoms having been present for any length of time; sometimes they are anticipated by headache, giddiness, amblyopia, amaurosis, nausea, and vomiting. The fits are very acute, and return in short intervals of minutes or hours, often in one day. They often occur only once in a lifetime, and it is only rarely that they return in several successive pregnancies. Labour is generally induced by them after they have continued for several hours; they exercise a very injurious influence on the life of the fœtus, and are not unfrequently followed by puerperal diseases. Insensibility generally supervenes after the first few fits, and often returns with the commencement of cure after a comatose condition may have continued for a few days. The symptoms of Bright's disease generally disappear after a few days, sometimes twelve days after delivery, or the cessation of the eclampsia. If the disease ends in death, then generally, in the post mortem examination, we find œdema and anæmia of the brain, œdema of the lungs, and Brightian degeneration of the kidneys; death, therefore, is generally the effect of the uræmic condition of the blood, and it is only very seldom the consequence of a secondary apoplexy of the brain.

2. Cholæmic eclampsia arises from the blood being overcharged with the constituents of bile and the products of their decomposition, and is connected with acute atrophy of the liver (Rokitansky), icterus typhoides (Lebert), pyæmia and

puerperal diseases of pregnancy, labour, and child-bed. It generally terminates fatally after continuing several hours or days.[1]

Acute atrophy of the liver is recognised,—during life, by the indications of a rapidly advancing diminution in bulk of the liver (in consequence of parenchymatous inflammation, according to Bamberger and Wedl); after death, by microscopical evidence of destruction of the glandular tissue of the liver (Rokitansky, Budd).

Convulsions, icterus, and fever, are the ordinary group of symptoms of acute atrophy of the liver. But the same sometimes occur in typhus and puerperal disease; and therefore, in appreciating the various causes of cholæmic eclampsia, the greatest stress is to be laid on the evidently diminishing size of the liver as discovered by percussion. Its appearance is generally very unexpected, because the premonitory symptoms are, from their insignificant character, generally overlooked. A slight icterus generally precedes it for some days, and then, on the occurrence of some slight baneful circumstance, complete insensibility comes on, or a lethargy which rapidly increases to insensibility; often also severe pain in the region of the liver, delirium, constant tossing about, violent shrieking and raving, convulsions, considerable acceleration of pulse, sometimes vomiting of blood and bloody stools. After a short time, deep coma generally comes on, or the patients pass from an apathetic and lethargic state into coma, single convulsive attacks at the same time appearing. The pulse is generally very quick and small, sometimes slow, perspiration bursts out on the skin, and death quickly succeeds.

The liver becomes flatter and thinner in its antero-posterior diameter. Over the whole surface, where in health moderately strong tapping produced the completely dull sound of the liver, percussion now affords certainly a deadened sound, but not perfectly dull, and more or less tympanitic.

Manipulation of the region of the liver, even during the deepest sopor, causes

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[1] [For a recent description of this disease, see an article by Professor Lebert of Zurich, in Virchow's Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, VII. Bd. p. 343, 1854. He describes coma, and not unfrequently convulsions, as coming on towards the end of the third or advanced stage of the disease. These last have sometimes the character of alternating tonic and clonic spasms, sometimes more that of tetanus. They affect the muscles of the face and then of the upper and lower extremities. Occasionally they are confined to one half of the body. Sometimes they are quite partial, in the form of trismus, spasms of the mouth, abdominal, or respiratory muscles, and occasionally of cramps in the limbs. A case from Kerkring is recorded in Bonnet's sepulchretum, where an icteric mother miscarried at the eighth month. Kerksig records three cases of abortion from this cause, of which two were fatal. Ozanam describes a fatal case of this disease in a woman six months pregnant. Wisshaupt describes a case from Oppolzer's clinique, where the disease came on and proved fatal in the seventh month of pregnancy; and another case is described where, after premature labour in the seventh month, the disease proved fatal. These cases are considered by Professor Lebert as sufficient to prove that icterus, in pregnant women, is a disease equally dangerous to mother and child. This list of cases might be enlarged by the records of experience nearer home. But cases of the kind are rare, and the complication with characteristic convulsions is still more uncommon.]

contractions of the muscles of the face, groaning, and movements to ward off the pressure. Swelling of the spleen is generally present, but it is often not sufficiently great to be recognised with certainty during life. In the dead body, we discover under the microscope, instead of the normal liver-cells among the ultimate vascular ramifications, and the connective tissue accompanying them, abundance of larger or smaller fat-drops, molecular masses, and nuclei probably belonging to the destroyed liver-cells. According to Wedl, crystals of hæmatoidin and brownish-black particles of hæmatin are also found.

Sometimes particular portions of the gland have their liver-cells still entire, but they are almost always in a state of fatty degeneration.

According to observations made by Spaeth,[1] Bamberger, and myself, acute atrophy of the liver occurs scarcely once in 10,000 deliveries, but in our climate still more rarely apart from pregnancy, while we have found uræmic eclampsia occur once in every 500 deliveries. It is still unknown which elements of the bile, or what products of their decomposition, exercise the baneful influence on the nervous system. The formation of leucin and tyrosin—crystalline products of the decomposition of albuminous substances—may, according to Frerichs' views, produce cholæmic eclampsia. Virchow,[2] however, feels himself unable to grant this, because leucin and tyrosin are also found in typhus and exanthematous diseases, and may possibly be formed not till after death. Bamberger[3] thinks it more probable that cholæmic cerebral phenomena are produced by the acids of bile resin (taurocholic and glycocholic acid), and by the possible products of their decomposition, than by the biliary pigments.

The skin is of a light sulphur colour. The urine contains a large quantity of the colouring matter of the bile, and the fæces are generally coloured with bile.

As is well-known, the presence of biliary pigment in the urine is demonstrated by mixing it with nitric acid (the best for the purpose being what contains some nitrous acid). A green colour is produced, which generally quickly passes into violet, blue, red, and orange. Instead of nitric acid, a mixture of equal portions of nitric and sulphuric acids may be used, and, in this way, with smaller amounts of biliary colouring matter, the reaction often comes out more distinctly.

When cholæmia and Bright's disease occur together, the following method may, according to Heller, be used with urine containing albumen: He puts a few grammes of muriatic acid into a cup-shaped glass, and passes into it drop by drop the urine to be examined, until the albumen begins to coagulate; then, keeping the fluids agitated, nitric acid is added, whereupon, if biliary pigment be present, a distinct green colour appears. The acids of the bile (taurocholic and glycocholic acid) are almost never found in the urine in cholæmic eclampsia. This may be proved to any one by the negative results of Pettenkofer's test. Two or three drops of a solution of sugar (one part of sugar to four parts of water) are added to the urine, and then gradually pure concentrated sulphuric acid, up to

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[1] Spaeth: *Zeitsch. d. Ges. Wiener Aerzte.* 1854.
[2] *Clinik der Geburtsh. Erlangen.* 1855, S. 246.
[3] Bamberger, H.: In Virchow's *Handb. d. spez. Path. u. Therap.* Bd. VI. Abth. I. Zweite Hälfte. S. 525–590.

five times the volume of the quantity of urine to be examined. Excessive heating of the mixture is to be avoided by plunging the test glass in a cool medium. When the acids of biliary resin are present, a purple colour gradually appears, but often not till after some hours.

But as a decided reaction is only rarely produced in the urine as it is passed, it is in every case preferable to evaporate the urine in a water-bath, and then produce the above-mentioned reaction with the alcoholic extract, which will, by its bitter taste, betray the presence of biliary acids.

3. Hysterical convulsions occur, during very painful deliveries, in women who, in the course of pregnancy, chiefly at the times menstruation might have occurred, suffer from the well-known hysterical affections, as spasm of the glottis, of the pharynx (Globus hystericus), dyspnœa, tendency to coughing, anæsthesia of the skin, etc. They are not accompanied by complete insensibility, and have no injurious influence on the life of the fœtus or of the mother. The urine is deficient in solid contents, but contains no albumen or cylindrical clots, and generally contains sugar (R. Wagner, Valentiner).[1]

In the severest attacks, the psychical life is never affected so as to produce loss of consciousness and perception. A hysterical patient falls into a convulsive, tetanic, or cataleptic attack with a scream, and closes the eyes when a dazzling light is presented to them. In this way these attacks may be clearly distinguished from epilepsy and eclampsia.

The paroxysms appear in shorter or longer intervals, sometimes during labour when the child's head is passing the os uteri or os vaginæ. They often stand in evident connection with the pains, and sometimes appear at irregular periods during pregnancy, or apart from it during the presence of the most different diseases of the uterus, or during attempts at replacing a retroverted gravid uterus (Romberg). In the intervals, a bodily and mental irritability and weakness are characteristics, but consciousness always persists. In hysterical convulsions, diseased conditions of the central organs of the nervous system, or indeed other palpable changes in the organism, cannot be demonstrated.

The hysteric fits manifest themselves in a manner varying according to the period at which pregnancy has arrived. In the first four months of pregnancy, they have most likeness to ordinary hysteric fits—globus hystericus, oppression,

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[1] Valentiner: *Die Hysterie und ihre Heilung*. Erlangen, 1852. [See a curious case of hysteria, with albumen and xanthic oxide in the urine, reported by Dr Douglas Maclagan in the *Edinburgh Monthly Journal of Medicine*, vol. xiii., 1851, p. 121. The statement in the text in regard to Glycosuria, may require to be modified in consequence of the researches of Blot and Kistner, which seem to show that the occurrence of sugar in the urine of pregnant women is common, in the urine of parturient and suckling women is constant. De la Glycosurie, physiologique chez les femmes en couches, les nourrices et un certain nombre de femmes enceintes. *Comptes Rendus de l'Acad. des Sc.*, tome xliii., 1856, p. 676. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, 1857, Paris. See also an observation by Lehmann, Physiol. Chemistry. Cavendish Society's Translation, vol. ii., p. 427. In a paper just published in the *Archives Generales de Medicine* for August, Dr Leconte denies the accuracy of the researches of Blot, showing that he has used the sugar tests without sufficient care to avoid sources of error.]

difficulty of breathing, bending backwards of the body, convulsive motions of the limbs; or they may assume the milder forms of anguish and oppression in the chest, which find vent in involuntary weeping or laughing, or may increase to the ecstatic form of hysteria.

In the second half of pregnancy, hysteric convulsions appear on very slight irritations; for example, motions of the child causing pain while there is a state of hyperæsthesia of the internal surface of the womb; and permanent nervous diseases sometimes come on, as paraplegia, or paralysis of one or more of the extremities, which, however, do not disturb the pregnancy, and are often perfectly cured after delivery at the full time (Gendrin).[1]

4. Idiopathic epileptic convulsions are habitual, chronic, and often recur during pregnancy, with intervals of days or weeks, and rarely happen several times in one day; they do not interfere with the pregnancy, and have no injurious influence upon the life of the fœtus, or the health of the mother in other respects.

Epileptic fits, even when they have occurred repeatedly during pregnancy, make their appearance only rarely during labour; and when they do so, cause no interruption to the advance and delivery of the child and placenta. The aura epileptica generally precedes these attacks, which are characterized by insensibility, and distinct consciousness generally soon returns after the fit.

All Brightian and uræmic symptoms are altogether wanting, except in the case of an epileptic being afterwards seized with Bright's disease. Hitherto, such complications have been very rarely observed.[2]

Loss of consciousness, with persistence of reflex sensibility, continues from the beginning to the end of the paroxysm; for touching the eyelids causes motions of them, and sprinkling the face with cold water causes, during the fit, a starting of the whole body—phenomena not observed in uræmic eclampsia. The spasm of the pharynx, which is constantly present, hinders the respiration, and causes congestion of the head, swelling of the veins, and a cyanotic appearance of the face. In consequence of the presence of trismus, trachelismus, and laryngismus, the saliva accumulates in the mouth, and passes out of it as foam. Attacks of this kind often terminate in deep sleep, sometimes without it, and in a few hours afterwards the patients are very well. Epileptic cases are very little influenced by pregnancy, labour, or child-bed; and sometimes are ameliorated, sometimes aggravated, in the course of the function of reproduction. The chronic character is sufficient to distinguish epilepsy from the ordinary acute uræmic or apoplectic convulsions, if there be a similarity in the convulsive attacks.[3]

Death very rarely occurs, during a paroxysm of epilepsy, from secondary asphyxia, rupture of a vessel in the brain or in the lungs, but mostly at a later period and unconnected with the fits. In the dead bodies of epileptics, no patho-

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[1] Gendrin: *Gaz. des Hôp.* 1854. Nr. 1, 5. [On this subject, see Churchill on the Diseases of Women, including those of Pregnancy and Childbed. Fourth Edition. 1857. P. 782.]

[2] [In a paper by M. Seyfert, which I have not seen, but of which an abstract appeared in the *Edinburgh Monthly Journal* for 1854, vol. xviii., p. 168, a quite different statement is made, but apparently without satisfactory evidence of its accuracy.]

[3] Romberg: *Nervenkrankheiten.* Berlin, 1851.

logical changes are observed which can be regarded as having any constant relation to the disease.

5. Apoplectic or cerebral convulsions are characterized by these circumstances: that the spasms continue and endure; that with their sudden appearance and frequent (often in a few minutes) recurrence consciousness is destroyed, and the pulse becomes slow and hard; that paralysis of the facial muscles and of the extremities of one side (hemiplegia), and clonic spasms, come on in the paralysed parts; that the comatose condition precedes the convulsions instead of following them, as in uræmic eclampsia; and that the breathing is much slower and quieter than in the intervals of uræmic attacks. Enlargement of the spleen is not met with. The symptoms of disease of the kidneys and of uræmia are absent, and the phosphates are sometimes found in the urine in great quantity. Apoplexy ends generally in idiocy or death.

During pregnancy, apoplexy of the brain occurs as seldom as during labour, so that no intimate connection between them can be established.

6. Convulsions originating in meningitis and encephalitis are distinguished by premonitory pain in the head, radiating to the shoulders; by the accompaniment of violent fever, great heat of skin, and the quick supervention of quiet or furious delirium; by absence of enlargement of the spleen; by remarkable increase of the phosphates, high specific gravity of the urine, its acid reaction, generally inconsiderable diminution of the chlorides; and by the absence of Brightian and uræmic symptoms.

These convulsions are sometimes followed by paralysis of the right side when the left half of the brain is affected, and *vice versa*.

Thrombosis of the longitudinal sinuses can scarcely be distinguished from encephalitis during life (Mikschick).[1]

7. In acute tuberculosis of the membranes of the brain, there is no albumen in the urine; it has a high specific gravity (1028–35), acid reaction; that is, little or no diminution of the chlorides, but there is a large quantity of urea, uric acid, uro-erythrin, moderate increase of the alcoholic extract, and never any diminution of the quantity of phosphates.

Hearing and speaking are rarely interfered with. During the convulsions, the pulse is frequent; during the coma, slow. The coma generally comes on after delirium. Cramps sometimes occur in the neighbourhood of the neck; but they and the convulsions cease when the coma appears, and paralysis, specially of the urinary bladder and bowels, come on.

8. Convulsions originating in typhus are known by fever, languor, confused headache, and loss of appetite preceding them for a longer or shorter time. A so-called papular typhus exanthem (Roseola) is generally to be found on the chest, and startings of the tendons are remarked. After the convulsive attacks, there does not come on a lethargic condition characterized by deep stertor, but a state of heaviness with occasional delirium. A more or less considerable enlargement of the spleen may be demonstrated by percussion, which cannot be accounted for by the history of the case indicating a previous attack of intermitting fever.

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[1] Mikschick: *Wiener med. Wochensch.* 1855, Nr. 15.

In the acid urine of typhus patients carbonate of ammonia is found. The reaction of the fresh urine is generally alkaline. The urea and uric acid are never increased, the uro-erythrin is in small quantity or altogether wanting, the alcoholic extract is considerably increased, and the chlorides are diminished to a very remarkable degree. The smell is ammoniacal; the phosphates are in small quantity or altogether wanting. In the sediment is found much urate of ammonia with a little triple phosphate. The specific gravity is low (1017). Traces of albumen are met with only in the most dangerous and protracted cases of typhus, according to the very numerous researches of Heller[1] and Tomowitz.[2]

9. Convulsions arising from anæmia are distinguished by the symptoms of the latter condition,—waxy yellow colour of face, the redness of the lips completely blanched, top-murmur in the vessels, coldness of skin, small threadlike quick pulse, small spleen, etc., as well as by the easily obtained history of the case. The convulsive motions of the extremities are in most cases only trifling; generally, indeed, the spasms affect only single muscles. Anæmic convulsions are justly regarded as a symptom of the last agony.

10. Eclampsia toxica, quickly supervening on eating, or introduction into the system otherwise, of mineral, vegetable, and animal poisons, has the greatest resemblance to uræmic eclampsia, but is distinguished from it by absence of all symptoms of diabetes albuminosus and uræmia, by pains in the region of the stomach and swelling of the same part, vomiting, gastritis, the chemical evidence of the existence of poison in the evacuations, and by various symptoms characteristic of the different kinds of poisoning.

a. Eclampsia saturnina (plumbismus) is distinguished by the gum having slate-grey markings, slow pulse, hardness, dryness, and icteric coloration of the skin, and absence of diabetes albuminosus (Grisolle, Tanquerel des Planches).

b. In eclampsia argyrialis (poisoning with nitrate of silver), intense colicky pains are absent.

c. In eclampsia mercurialis, the mercurial tremor is almost never awanting.

d. In stibismus and

e. Cuprismus cerebro-spinalis, intestinal symptoms are almost always awanting, and cerebral symptoms occur, not at the end, but the beginning of the poisoning.

f. In arsenicismus cerebro-spinalis, when the poison is applied to the stomach, vomiting always comes on. When it is absorbed through wounds of the skin, all symptoms of intestinal disorder are awanting; and it is then to be distinguished from narcotic poisoning only by chemical examination of the evacuations of the poisoned.

g. Oxalysmus cerebro-spinalis cannot be distinguished from strychnismus except by the interrupted pulsation of the heart.

h. Hydrocyanismus is known by the smell of bitter almonds diffused at every expiration.

i. Acute alcoholismus, appearing in the form of eclampsia in young individuals,

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[1] Heller: *Archiv. f. phys. u. path. Chemie u. Mikroskopie.*
[2] Tomowitz: *Zeitsch. d. Ges. Wiener Aerzte*, II. Bd. Wien. 1851.

in consequence of intoxication with spirituous drinks, is recognised by the history of the case, by the alcoholic smell of the expired air, by acid eructations, and by the absence of all the phenomena of albuminuria and uræmia.

j. Poisoning by strychnine and brucine is characterized by great susceptibility to terror through insignificant irritations (noise, light, draught of air, touch), by the tetanic form of the spasms, by agitative movements of individual sets of muscles and of the eyes, by continued retention of consciousness, and by remarkable paralysis of nerves after the disappearance of the spasm.

k. Eclampsia from poisoning with picrotoxin, from the berries of menispermum cocculus, is characterized by tetanic attacks succeeded by spasms of the muscles for mastication, salivation, and peculiar clonic spasms of the limbs (swimming motions).

l. Poisoning by hemlock (conicismus) has this peculiarity, that anæsthesia and adynamia begin at the feet, which make the gait staggering, and afterwards walking altogether impossible (from paralysis). Then inability to utter articulate sounds, loss of sight, with great heaviness of the eyes, come on, while consciousness remains entire.

m. Nicotismus (poisoning by tobacco) is distinguished by sensations of choking, vomiting, diarrhœa, convulsive trembling, unfrequent small pulse, pallor of the skin, which is covered with a cold sweat, salivation, and asphyxia.

n. Aconitismus manifests itself either by suddenly occurring paralysis, asphyxia, or syncope. The extremities are pale and ice-cold. Consciousness is long of disappearing—shortly before death.

o. Colchicismus resembles either a distinct gastro-enteritis or Asiatic cholera, and tetanic convulsions close the scene.

p. In atropismus (Atropa belladonna, Datura stramonium, Hyoscyamus niger) the prominent symptoms are, extraordinary dryness of the mouth and throat, which are of a lively red colour, completely suppressed secretion of saliva, dysphagia, pulsation of the vessels of the neck, pseudopsia, diplopia, hallucinations, sardonic laughing, delirium, and madness, with tendency to get up and run away.

q. Convulsions from acute poisoning by phosphorus (phosphorismus cerebrospinalis) are distinguished from uræmic eclampsia by this circumstance, that the matters vomited, the fæces, the urine, the pulmonary exhalations, and the sweat, contain phosphorus, and glow in the dark.

r. The diagnosis of morphinismus is arrived at only by analysis of the evacuations from the body. But even in this way the object is not always gained.

s. In ergotismus convulsivus the patient complains of suddenly-appearing giddiness, blindness, trembling of the limbs, convulsive motions, tonic spasmodic contractions of the flexor muscles, choking, vain attempts to vomit, cramplike tension of the abdomen, retention of urine and fæces. The pulse is small and contracted; the expression of the face is disfigured and sallow. Death happens during insensibility and convulsions.

t. Botulismus (poisoning by sausages) is recognised by the occurrence of giddiness and stupefaction, dryness and livid coloration of the conjunctiva, angular arrangement of the edge of the pupils, pain in the eye-balls, and paralytic condition of the eyelids. Comatose drowsiness, burning in the throat, difficulty in

swallowing, obstinate constipation, exfoliation of the epidermis, asphyxia, or slight convulsions, precede death.

u. Echidnismus[1] (poisoning by the bites of snakes) can scarcely be confounded with eclampsia puerperalis, as the anamnesis, the presence of a variously coloured and very painful swelling, the form of the wound with one or two fine stings or scratches ¼″ to ½″ distant from one another, languor, faintings, loss of strength, convulsive spasms of the face or other parts of the body, even irregular or epileptic convulsions, fearful cardiac anguish, with frequent intercurrent swoons, entire pulselessness, and almost invariably frequent vomiting of bilious mucous matters, prevent any doubt being had as to the correct diagnosis (Falck).[2]

11. Chorea gravidarum (Scelotyrbe)[3] appears as aimless spasmodic movements of single or several groups of muscles, and is distinguished from the general convulsions that we have described by this, that the violent convulsive motions are quite partial in the upper or lower extremities, and come on at a definite time of the day, the consciousness and reason are not in the least disturbed, the disease may continue for months, all the functions of the body go on undisturbed, and it is generally completely cured only after delivery at the full time or prematurely. Frank,[4] Ingleby,[5] Lever,[6] Romberg,[7] Scanzoni,[8] Duncan.[9]

12. Fainting fits (dystocia lipothymica) often appear during or shortly after delivery, oftener after losses of blood, more rarely from great force of pains, suffering, fright, too great heat or bad ventilation of the chamber, inhalation of carbonaceous fumes, or a long-continued noise. It is characterized by sudden falling down, disappearance of consciousness, striking paleness of face, short duration, and the absence of all general or partial convulsions. Hence it is hardly possible to confound it with eclamptic coma.

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[1] ἡ ἐχιδνα—the viper.
[2] Falck in Marburg: In Virchow's *Handb. d. spez. Path. u. Ther. Erlangen.* 1855, II. Bd. 1. Abth.
[3] Το σκελος—limb, ἡ τυρβη—restlessness.
[4] Frank, Jos.: *Prac. Med. Præcepta,* p. ii. v. i.
[5] Ingleby: *The Lancet,* Nr. 860.
[6] Lever: *Guy's Hosp. Rep.,* II. Ser. v. v. vi.
[7] Romberg: l. c. S. 178.
[8] Scanzoni: In *d. Forts. d. klinischen Vorträge von Kiwisch. Bd. III. S.* 433.
[9] Matthews Duncan: *Edinburgh M. and S. Journal,* 1854. [See Appendix.]

CHAPTER IX.

PROGNOSIS OF URÆMIC ECLAMPSIA.

In eclampsia the prognosis must always indicate danger to life, since certainly, hitherto, 30 per cent. of the cases have proved fatal. From the nature of the influence exerted by the uræmic intoxication, the prognosis embraces several considerations, as, in reference to the convulsions of Bright's disease, and the subsequent conditions, as mania, hemiplegia, amaurosis, hemeralopia, and abnormal puerperal processes,—in reference, also, to the life of the fœtus, and the influence of the disease in causing premature labour and abortion.

Uræmic eclampsia terminates more frequently in complete recovery or death, than in consequent long-protracted sickness. Its dangerous character depends on the following circumstances :—

a. From its commencing during pregnancy, or at the beginning of labour, where the obstructions to the extraction of the fœtus, and consequent diminution of the volume of the uterus, presented by the cervix and os uteri, are still very great ; and when, consequently, congestion of venous blood in the kidneys cannot be removed, as happens after earlier or later expulsion of the fœtus. For the fits completely cease after evacuation of the uterus in 37 per cent., become weaker in 31 per cent., and in 32 per cent. only continue of the same severity.

b. Upon the occurrence and continuance of complete unconsciousness during the intervals of the paroxysms.

c. Upon extraordinary restlessness and exalted reflex sensibility in the intervals.

d. Upon deficiency or gradual weakening of the pains, or from their inefficiency in the period of dilatation.

e. Upon the pulse quickly rising in frequency, and on œdema of the lungs extending after every attack, and the dyspnœa produced by it.

f. Upon serous exudation in the brain, effusion into the ventricles, or apoplexy, with its consequent hemiplegia, coming on during the fits, in consequence of secondary hyperæmia of the meninges.

g. Upon extensive dropsical effusions.

But if the paroxysms become regularly less frequent and violent, if no secondary disease of the brain and lungs has been produced by the fits, if the

pulse continues full and quiet, the prognosis assumes a more favourable aspect, especially when coma that has continued for several hours or days disappears, when abundant diuresis goes on, and the action of the heart approximates to its natural condition.

The mortality among the sufferers does not vary according as the eclampsia has come on early or for the first time at the end of pregnancy.

Unless the uræmia proves dangerous to life, the acute Bright's disease of pregnant women seldom has so unfavourable a course as the other forms of the disease.

The affection of the kidneys often passes off without causing any striking disturbances, and is not even suspected unless the urine has been examined chemically and microscopically.[1]

Bright's disease often induces premature labour, when no other cause is present, and this happens approximatively in 25 per cent.

The process of parturition is very painful, and gives evidence of increased reflex sensibility in cases of acute Bright's disease without uræmia.

It can be shown that during labour, in consequence of the increased mechanical obstruction, the albuminous contents of the urine sometimes increase, and the exudation clots are found in great quantity. During child-bed, the albuminous contents always diminish, and often so quickly, that after two or three days none can be discovered.

After from six to ten days, if the child-bed patient continues to go on well, there is generally no trace of albumen to be discovered. If during child-bed the albuminuria continue for weeks, it arises either from the admixture of pus from an acute catarrh of the bladder, or from nephritis metastatica, or from a far advanced destruction of the kidneys being present, and the Bright's disease being chronic.

The cylindrical clots are, in the first days of child-bed, passed in great quantities, but disappear from the urine sooner than the albumen, and are not found at all in simple catarrh of the bladder.

Diuresis increases from after delivery till the recovery. In this way the œdema is generally made to disappear rapidly, so that generally after eight days no trace of it can be discovered; and as the bloated condition of the face generally disappears with it, patients assume a very much changed and generally more pleasant expression of countenance. If the decrease or disappearance of the existing œdema takes place without improvement of the disease in the kidneys, no good prognosis can be given, because uræmic eclampsia sometimes comes on in the period of child-bed without it.

If the symptoms of Bright's disease, albuminuria, cylindrical clots, and œdema, are not gone several weeks after delivery, the disease assumes a chronic character; but, even in these unfavourable circumstances, a cure is effected, although after a prolonged illness, more frequently than in Bright's disease arising from other causes.

The prognosis of the evil consequences of uræmic eclampsia is generally less

[1] [See footnote, p. 23.]

unfavourable. The mania, sometimes occurring after awaking from the comatose condition, generally admits of a favourable prognosis, if it be not confounded with the delirium, which is a symptom, in many cases, of puerperal pyæmia. The mania seldom lasts more than three days, generally ends in complete recovery, is almost never followed by a continued derangement of mind, and commonly assumes a cheerful character. (Helm, the Author, Litzmann and others.[1])

The amaurosis of pregnancy is dangerous, because it is often followed by eclampsia, and blindness after delivery often lasts for several months.

After hemeralopia evil results are rarely observed.

Hemiplegia is among the saddest occurrences, because it indicates that extravasation of blood in the brain has already taken place.

Morbid puerperal processes after eclampsia are to be the more dreaded, because, while in Bright's disease, in general, exudations into the pleura, peritoneum, and lungs are apt to take place, in cases of eclampsia the most dangerous puerperal diseases are easily induced, especially if an epidemic of zymotic diseases prevails.

The life of the fœtus is endangered so long as it is nourished by the uræmic blood of the mother.[2] If it has sustained no injury during labour, and if it is mature and viable, little fear need be entertained for the suckling's life; for the possibility of the hereditary transmission of eclampsia, uræmia, and Bright's disease of the kidney to a suckling, has not yet been demonstrated, and only Simpson has found albuminuria in a suckling born of an eclamptic mother.

Metrorrhagia is very dangerous from the hydræmia which is generally present after uræmic eclampsia, but it occurs only very rarely, if the conduct of the labour has been carefully attended to.

The dangers of eclampsia are greatly increased by complications with diseases of the heart and lungs, rupture of the uterus, etc.

The prognosis in other kinds of eclampsia is the same as when pregnancy has not occurred. Cholæmic, apoplectic, toxic, and anæmic eclampsiæ, are very often fatal; hysteric and epileptic attacks, and chorea, almost never so.

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[1] [Some cases of puerperal mania, accompanied by albuminuria, and where no eclamptic attacks had occurred, are alluded to by Dr Simpson. *Edinburgh Medical Journal* for 1856, p. 761.]

[2] [If the uræmia persists in a nursing woman, urea may be present in the milk, as has been shown by several observers, and may disturb the health of the suckling.]

CHAPTER X.

TREATMENT OF BRIGHT'S DISEASE AND URÆMIC ECLAMPSIA.

A. The prophylaxis consists of the medical and obstetrical treatment of Bright's disease during pregnancy.

Complete cure of Bright's disease is rarely obtained during pregnancy, because the cause of it, the obstruction of the venous circulation in the kidneys, is not easy of removal.

Hydræmia, developing itself at an early stage of pregnancy, is somewhat ameliorated by nutritious diet, vegetable tonics, and preparations of iron. Increase of the secretion of urine does not generally produce this result. Favourable influences are sometimes observed from tepid baths, and especially vapour baths.

For the neutralization of the carbonate of ammonia in the blood, produced by the decomposition of urea, we may, according to Frerichs, make use of benzoic acid, lemon juice, or tartaric acid.

To obviate congestion of the head, costiveness should be prevented by vinegar injections, aloes, jalap, etc.

When exudation has taken place into the Malpighian capsules, and the tubuli of Bellini and Ferrein, the cylindrical clots must be removed from them, and the formation of new ones prevented. If the current of fluid proceeding from the vascular knot of the Malpighian bodies into the Malpighian capsule be strong, then the copious use of a large quantity of diluents is alone sufficient sometimes to wash away the cylindrical clots, and recovery ensues.

But if the secretion of urine be very scanty, and uræmic intoxication threaten to come on, then the force of the current of fluid proceeding from the Malpighian bodies must be increased, and the cylindrical clots removed; for which purpose, besides the acids above-mentioned, the mineral waters of Selters or Vichy are best adapted.

According to the example of Frerichs, pills of tannin and extract of aloes are to be used for restoring the normal tone.

Since, by medical treatment, acute Bright's disease during pregnancy is generally only mitigated, not cured, the question has to be considered, whether, on account of the Bright's disease, the induction of artificial premature labour be

admissible, in order to avert the venous congestion and the advancement of degeneration of the kidneys.

It must be laid down as settled, that, in Bright's disease, artificial premature labour is not to be thought of so long as no symptoms of uræmia have appeared, and no danger to life is present. But when the duration of the disease, the severity of the albuminuria, the quantity of cylindrical clots, a high degree of hydræmia, considerable dropsical swellings, along with disturbances, dangerous to life, of the functions of the heart, lungs, brain, etc., entitle us to fear the existence of profound and advancing degeneration of the kidneys, it is quite rational to proceed to the induction of premature labour. When several symptoms indicate that the fœtus is already dead, we are the more justified in proceeding, all the sooner, to this operative interference, because the dead fœtus is sometimes retained for weeks in the uterus, and the danger to the mother's life may be thereby increased in a way that cannot be justified.

Observation of the proceedings of nature indicates to us this method of proceeding, for, in acute Bright's disease, pregnancy is often spontaneously interrupted; and, in that case, a fatal issue of the childbed rarely results.

When, in Bright's disease, labour comes on without eclampsia, Chailly[1] recommends, in order to prevent the outbreak of convulsions, the use of a slight degree of narcotism by chloroform. I have not yet had occasion to make observations in regard to this point, but would make use of it in metralgia and protraction of labour in those suffering from Bright's disease.

B. Medical and obstetrical treatment of uræmic eclampsia.

The medical treatment of uræmic eclampsia is conducted in a similar manner in pregnancy, labour, and childbed. The chief object to be attained is to diminish as much as possible the reflex excitability, to weaken the paroxysms, in order to diminish the dangers, and to gain time for entering upon rational treatment.

In this respect, we have observed results from chloroform-narcotism which have surpassed all expectations. In uræmic eclampsia, the chloroform narcotism is to be induced instantly when indications of an impending paroxysm show themselves—as great restlessness, increasing rigidity of the muscles of the arms, expiry of the interval between former paroxysms, fixity of expression, or tossing hither and thither. The narcotism is to be kept up until the premonitory symptoms of the paroxysm disappear and quiet sleep follows; a result generally attained in one minute. But if it be not possible to cut short the paroxysm, then the chloroform inhalation is not to be kept up during the convulsive attacks and the comatose condition, in order to let an abundant supply of fresh atmospheric air reach the lungs. The chloroform inhalation moderates the imminently dangerous cramps of the muscles of the neck, epiglottis, and tongue, and may be continued even during a persistent trismus, when other medicines cannot be introduced into the stomach, and when loud mucous râles indicate the development of œdema of the lungs.

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[1] Chailly: *L'Union Médic.* 1853.

In sixteen cases of eclampsia, occurring in succession, which I treated with chloroform and acids, complete recovery always took place. As by anæsthesia we are put into a condition suited for remarkably accelerating delivery, the preservation of the life and health of the offspring is promoted in a very gratifying manner.

Before using chloroform, it should be tested, especially by smelling and by sulphuric acid, in order, by its bad smell or its assuming a brown colour, to discover if it has been prepared from wood spirit, and has the poisonous qualities arising therefrom.

Whether chloroform operates so beneficially merely as a sedative, or whether by chemical action it produces innocuous changes in the toxæmic blood, is as yet undecided.

Simpson is of the latter opinion, for this reason, that chloroform inhalation, according to chemical analysis, produces a transitory diabetes mellitus—hence sugar certainly appears in the urine (and also in that of animals, according to Hartmann's[1] researches), and probably also in the blood; and because, out of the human body, a very small quantity of sugar added to the urine prevents the ordinary change of urea into carbonate of ammonia. Although the direct action of chloroform upon uræmia is still doubtful, yet it is certain that in eclampsia chloroform is the best palliative, inasmuch as it moderates the paroxysms—the waiting for and performance of operations is shortened and facilitated—the danger to the lives of mother and child is essentially diminished; and hence it is that the already announced commendations of chloroform in puerperal eclampsia by Simpson,[2] Channing,[3] the Author, Seyfert,[4] Chailly-Honoré, Scanzoni, Sedywick,[5] Wieger, Meisinger,[6] Hoogeweg,[7] Leudet, Dechambre,[8] and others, are constantly gaining a wider recognition. In the intervals of the fits, the direct treatment of the uræmia is proceeded with—either 5–10 grain doses of benzoic acid being administered, or lemon juice, or table-spoonful doses of a solution of tartaric acid, with ice-water, when copious diuresis generally soon appears.

To moderate the secondary congestions of the head which come on during and after the paroxysms, the application of ice is useful, and also smart sprinkling with cold water (Recamier, Booth[9]); and, better still, the cold douche on the head, during which operation the head of the patient is held over the side of the bed, and the ice-water falls into a basin held beneath it.

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[1] Hartmann, F.: *Beitrag zur Literatur über die Wirkung des Chloroforms.* Geissen, 1855.
[2] Simpson: *Anæsthesia, or the Employment of Chloroform, etc.* Philadelphia, 1849. P. 207.
[3] Channing: *A Treatise on Etherization.* Boston, 1848.
[4] Seyfert: *Wien med. Wochensch.* 1853, Nr. 12.
[5] Sedywick: *Bull. de Ther.* 1850. P. 83.
[6] Meisinger: *Wiener mediz. Wochenschrift.* 1853, Nr. 40.
[7] Hoogeweg: *Pr. Verhandl. Zeit.* 1852, Nr. 51; und 1853, Nr. 51.
[8] Dechambre: *Gaz. hebdomadaire.* 1855, Nr. 5.
[9] Booth: *Journ. der conn. méd. chir.* 1853.

Tepid baths of the whole body cause too much trouble when the patients are completely insensible, and therefore we never employ them.

The local application of cold has a more powerful and lasting influence against secondary hyperæmia of the meninges than the use of leeches, which, on account of the restlessness of the patients, cannot be got to stick on the region of the mastoid process, where any considerable depletion can be effected through the great blood sinuses; and on the forehead no essential and direct depletion of superfluous blood from the brain is possible by this means.

Sponging the skin with tepid vinegar produces a most desirable diaphoresis, and is easily accomplished.

General depletion of blood easily produces, in uræmic eclampsia, an injurious effect, because the cyanosis of the face coming on in eclamptic women is only a consequence of the spasm; because, by bleeding, the hydræmia is further increased, the nervous fits are not improved, puerperal thrombosis and pyæmia in child-bed are much to be feared; and because, not unfrequently, the paroxysms are aggravated by it, and exhaustion, fainting, and very slow reconvalescence are thereby produced.

As to the very doubtful, and sometimes even injurious, effects of venesection in uræmic eclampsia, Maygrier, Peterson,[1] Kiwisch, King, Bloot, Sedywick, the Author, Churchill, Litzmann, Williams, Miquel,[2] Schwartz,[3] Legroux, Thomas, have very strongly expressed their conscientious opinions; and myself avoiding venesection, I have found, after long-continued observation, the best results confirm the opinion already expressed, that a "general depletion of blood in uræmic eclampsia had very seldom any valuable effect on symptoms, and generally produces irreparable injury."

We cannot reconcile with their theory the circumstance that the adherents of the hypothesis, that eclampsia is produced by hydræmia, recommend venesection as a cardinal remedy. But experience has established that, when a cautious selection of single cases is made, one moderate general blood-letting is not injurious in the case of strong, full-blooded women, when there is violent pulsation of the carotids, and the face continues dark red even a considerable time after the fit, and œdema of the lung is commencing, and when, at the same time, all anæmia, chlorosis, and bodily weakness, etc., are absent; on the contrary, in rare cases, a cessation or longer interval between the fits is observed.

Since the days of Dewees, Burns, and Hamilton, it has been in many places, and still is, the custom to find the only panacea against eclampsia in abundant general blood-lettings often repeated in the course of a day—a proceeding which can be justified as little by the present state of theoretical knowledge in regard to this disease, as it is by the great mortality of mothers and children constantly produced by this method of treatment.[4]

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[1] Peterson: *Lond. Med. Gaz.* 1844.

[2] Miquel, A.: *Traité des Convulsions*, etc. Paris, 1823.

[3] Schwartz: *Riga'er.* Beit. I. 2. 1850.

[4] [In spite of such statements as occur in the text, and as are now common in the mouths of obstetricians, I am not disposed to consider the old treatment by bleeding as having been so bad and absurd as it is now often called. I have too

Cazeaux has observed, in several cases, remarkably favourable results from hæmospasia (Junod's boots), in the way of soothing the convulsions by the derivative action on the head.

I consider this proceeding much more rational than dealing profusely in general blood-letting, because the blood is only momentarily withdrawn from the circulation, and the production of a transitory œdema of the extremities may free the blood for a certain time of morbid and altered serum.

The internal use of 1 to 6 grains of opium, of ¼ to 1 grain of acetate of morphia within six hours, and at the same time of 20 to 30 drops of anodyne tincture as a lavement, is specially to be recommended in those cases where chloroform and acids do not operate quickly and permanently enough, when the delivery is over, and the eclamptic fits still continue in childbed. My own observations in regard to this agree completely with those of Kiwisch, Scanzoni, Kilian, Wieger, Hohl, Feist, Credé, and others.

Expectative treatment can be recommended only where delivery is nearly completed, where the attacks are not severe, and consciousness returns in the intervals.

Coma, after the cessation of the paroxysms, is most safely and best treated by complete rest of the body, mind, and organs of sense; careful avoidance of all frights; the use of benzoic and vegetable acids and much cold drink, as has already been justly remarked by Harvie, Betschler, Wieger, and others; and by moderate diaphoresis, because the comatose condition of the brain is not produced by its being congested with blood, but by serous infiltration and uræmia.

From revulsive measures, as sinapisms on the calves of the legs, hot foot and hand baths, blisters on the back of the neck, and the like, no marked result is to be expected. After the abuse of phlebotomy, musk may be of some service as an anti-spasmodic.

But this remedy is always to be dispensed with, if no vampyrismus has previously occurred in the particular case, and if the anæmia produced is not dangerous to life.

Lobach[1] asserts that he has seen, in the eclampsia of pregnant women, vomiting stopped, and obstinate constipation removed, by tincture of nux vomica (four drops given every two hours), and the tinctura cupri acetici useful for the general spasms. On these points I have made no observations.

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much faith in the joint testimony of the able and experienced physicians who recommended this treatment, to adopt, without modification, the modern criticisms on their practice. While the custom of blood-letting in puerperal convulsions was still rife, I had occasion, in more than one instance, to express my disbelief in its efficacy; and I am now inclined to adopt, in great measure, the modern opposition to blood-letting. How to reconcile this opinion with my defence of the old practice of venesection I shall not here attempt to show. I shall only refer to the very interesting discussion at present going on (see *Edinburgh Medical Journal* 1856–57) in regard to a similar change in the usual treatment of inflammatory complaints in general, and to the evidence therein adduced of a change of type in the inflammatory diseases of to-day as compared with those of thirty years ago.]

[1] Lobach: *Verh. d. phys. med. Ges. zu Würzburg.* 1852. III.

Hasse approves of free evacuation of the contents of the bowels by calomel in large doses, castor-oil, and especially clysters with assafœtida and vinegar, infusion of senna, croton oil, and the like, as a good derivative measure.

Hohl regards a strong emetic dose of ipecacuanha as very valuable, if the attack was preceded by an error of diet. But, by other authorities, the use of emetics is altogether rejected. We have ourselves been unable to recognise any decided influence upon the course of an eclampsia from spontaneous vomiting.

The employment of tartar emetic as a vomit has been earnestly discommended by Mauriceau and Kilian, and we have never seen any good from its use in small doses.

Regarding Vanoye's [1] treatment of eclampsia with ammonia (20 drops of spirits of salt, with 250 grammes of distilled water and syrup, every half hour) we have no experience. Krause [2] says that, under the use of carbonate of ammonia, he has seen elamptic attacks, not in connection with pregnancy, disappear on the occurrence of menstruation.

In every eclampsia, care is to be taken that a patient who has fallen on the ground be as soon as possible placed in bed, and protected from injuries of the head.

During the paroxysm, free movement of the extremities is to be allowed, and only the rolling of the body out of the bed is to be prevented. Every precaution must be used for the protection of the tongue, which is always protruded in the beginning of the paroxysm. This is best done by pushing it back with the side of a finger. When trismus comes on, complete shutting of the jaws must not be prevented by anything, least of all by the handle of a spoon covered with linen, as formerly used so often to be done, to the very great injury of the teeth.

Formerly, too little was known of the nature of uræmic eclampsia to allow of a decision in regard to the value of directly interrupting the progress of the pregnancy and accelerating delivery; and this is the reason why, hitherto, no unanimity could be attained in reference to the indication of artificial premature labour and acceleration of delivery.

A great number of physicians, as Puzos, Osiander, Hasse, Feist,[3] Siebold, Meissner, Mad. Lachapelle, Langheinrich, Casier,[4] Krause, Caleb Rose,[5] Grenser, Gendrin, P. Dubois, and others, consider prompt careful evacuation of the uterus as the main point in the treatment of eclampsia.

The obstetrical treatment of eclampsia must be discussed from various points of view, according as the labour is far advanced, or has not yet commenced at all.

A. The treatment of eclampsia in the expulsive stage of labour is considered from the same point of view by the most different authors. All agree in this, that when the head is in a situation suitable for the application of the forceps, it

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[1] Vanoye: *Abeille Méd.* 1851. Nov.
[2] Krause, A.: *Die Theorie u. Praxis, etc.* Berlin 1853. II. Bd. S. 503.
[3] Feist: *Gem. deutsch. Zeitsch.* Bd. vi.
[4] Casier: *Presse Med.* 1853. 3.
[5] Caleb Rose: *Med. Times and Gaz.* 1852.

should be extracted in a most cautious manner, because thereupon the attacks often completely cease, and the children are generally born living, if there have not been very many fits before the operation.[1]

Presentations of the shoulder and pelvis, which are very rarely observed at this stage of labour in eclamptic cases, are to be treated according to general principles, and require an acceleration of delivery.

B. Oxytocic treatment in the stage of dilatation is of very great importance; for, in it, the mode of interference by operation must be chosen according to two conditions.

a. Treatment of eclampsia when the stage of dilatation is considerably advanced.

If the neck of the womb is completely dilated by the advancing head and the pains, before the arrival of the physician—if the external orifice is opened up to from 1 to 2″,[2] and the membranes are unruptured, then it is most rational, while the woman is on her side, to burst the membranes, and watch the result of the flowing off of the first waters and of the continued pains.

If the paroxysms do not cease thereupon, and if regular progress is not remarked in the further dilatation of the cervix and advance of the child, then the orifice should be fully opened up by dilatation by means of the finger.

If this be done a few minutes after the appearance of the comatose condition, or during the chloroform narcotism, new fits are never produced by it.

If, after some pains, the presenting head does not advance into the brim of the pelvis, and if some fits come on, then it is most advisable, even when the position of the head is high, and every obstetric disproportion is absent, to apply the forceps according to Hatin's[3] method, and carefully extract the child. By using the forceps under these circumstances, we have succeeded in extracting a greater number of living children, than when, in cases of eclampsia, the hand is, with-

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[1] [Although, at first sight, it may appear excessively cautious to warn the practitioner not entirely to neglect the progress of the labour, yet experience shows that the advice is necessary. The first case of puerperal convulsions which I saw, while yet a student, was under the care of two physicians, of whom one was old and experienced. We bled the woman, and cupped her, and applied sinapisms, and were, in short, very diligent till the convulsions, which were severe and frequent, ceased and the woman became quite quiet. It was only then that the physicians thought of the labour, and were thunderstruck on finding the abdomen empty. Indeed, the fœtus was found, only after some searching, among the bedclothes, confused by the woman's previous restlessness. It was dead, and had been flattened beneath the woman's hips.]

[2] [″ indicates a Paris inch.]

[3] [Hatin's method consists, essentially, in introducing the whole hand into the passages, and as high as the blades of the forceps go, in order to ensure the right direction and application of them. First suggested by Flamant, it was regularly proposed by Hatin (*Cours complet d'accouch.* Paris, 1835, p. 263) and extensively practised. It has been more completely described by Chailly (*Traité Prat. de l'art des Accouch.*, p. 637. 1853. Paris), and defended against the objections of M. P. Dubois. I shall not here enter on the questions of the extent of its feasibility and propriety. I am sure that it can be required only in very rare cases; and in these even, it is a question whether turning would not be a safer and better operation.]

out the power of choosing circumstances, introduced through a dilatable os uteri, and the extraction of the child completed by podalic version.

On account of eclampsia alone, without obstetrical disproportion, it is never justifiable to proceed to the operation of craniotomy.

Podalic version should, in cases of eclampsia, be confined to those cases only in which there is at the same time contraction of the pelvis, or an obstetrical disproportion on the part of the child.

Scarification of the cervix uteri, at a more or less advanced period of the stage of dilatation, for the purpose of facilitating labour, has been recommended by Paré, Mesnard, Coutouly, Lauverjat, Dubois, Kiwisch, Kilian, Credé, and others, and it is done either with a probe-pointed bistoury, a uterostomatome (two scarificator blades), or long bent scissors. In the hands of a practised operator, the making of these incisions, under the above-mentioned conditions, does not involve any danger, and contributes greatly to rapid dilatation when the cervix is peculiarly rigid and indilatable. But when we consider the question of the necessity of this proceeding, it is, indeed, in very rare cases only to be justified, inasmuch as in the literature of the subject only very few observations are recorded in which the performance of hysterostomatomia was found to be indispensable, and that more on theoretical than practical grounds.

b. If the treatment of eclampsia is commenced at the beginning of the stage of dilatation, when the cervix has disappeared, but the os is still very contracted, or when the cervix and os are still contracted, and there are scarcely any signs of labour, as not unfrequently happens in cases of eclampsia with spontaneous coming on of premature labour; then, in regard to treatment, choice has to be made among these different plans; *α.* either exciting energetic pains to completely dilate the cervix and os uteri in a physiological manner; *β.* or by operative bloody interference to open up a way for the fœtus through the lower segment of the uterus; *γ.* or to remain altogether passive until spontaneously supervening pains bring the labour to a termination, in cure or death of the mother or fœtus.

α. The results produced by increase of the physiological activity of the pains, in cases of eclampsia, are so favourable to the preservation of the lives of both mother and child, that the acceleration of labour in the stage of dilatation is warmly defended and recommended by the author, Kiwisch, Litzmann, Grenser, Stoltz, Chailly,[1] Credé, and many others; opinions being divided only regarding the choice of the means to be used.

According to my opinion, all the methodical rules which are in general suited for the most rapid bringing on of artificial premature labour, are here to be carefully considered.

Plugging the vagina with a caoutchouc bag has been very strongly recommended by me for attaining the above-mentioned object, after making numerous observations. In my proposal I have been very warmly supported by the opinions and experiments of Kiwisch, Holst, Wieger, Grenser, Simon Thomas,[2] Schil-

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[1] Chailly: *L'Art. des Accouch.* Paris 1853. P. 195.
[2] Simon Thomas: *Nederl. Lancet*, 1853, *und Schmidt Jahrb*, 1854. Nr. 12.

linger,[1] Litzmann, and many others; so that now, in a case of eclampsia, in the period of dilatation, where the pains are slow, it would scarcely be justifiable to abstain from making use of colpeurysis, and to proceed to *accouchement forcé*, when the lower segment of the uterus is closed.

The rapid dilatation of the soft genital passages obtained in this manner is in many cases chiefly to be ascribed to colpeurysis, because, in the eclamptic or anæsthetic coma, a greater distension than usual of the caoutchouc bag is sometimes made use of, and because, after inducing strong pains, labour proceeds very rapidly, even after removal of the colpeurynter.

The introduction and retention of an elastic catheter between the chorion and the walls of the body and fundus uteri, is a very simple, sure, and quickly operating means of inducing energetic pains; wherefore I must urgently recommend its use in eclampsia during the stage of dilatation.

It exerts this influence more rapidly than bursting the membranes and letting off the waters, and produces no injury. When there is evident danger to the life of the mother and fœtus, I would recommend the simultaneous employment of colpeurysis and uterine catheterization.[2]

The tepid uterine douche has been highly recommended by Kiwisch, Holst,[3] Grenser, Wieger, Simon Thomas, and Legroux, as a means of accelerating labour in the stage of dilatation. And there is no doubt that the douche, with a powerful jet of water, sometimes leads to results more rapidly in cases of eclampsia than in cases of contraction of the pelvis, in which even it has already happened that by one sitting labour has been induced and brought into full activity. Other methods of accelerating labour, as the use of secale cornutum, irritation of the nipples, carbonic acid douche, etc., are too uncertain in their action to admit of their being employed in a case of dangerous delivery.

In the most intense cases of eclampsia, good results have been observed from rupturing the membranes, by P. Dubois, Busch,[4] Rul-Ogez,[5] and others.

Waterhouse, Ashwell, Mitchel, and Villeneuve, consider energetic pains a very favourable event in eclampsia, and recommend the use of secale cornutum in order to accelerate delivery. But Kilian,[6] Velpeau,[7] and Masson,[8] have in the most earnest manner objected to this treatment, and, judging by our own experience, we must agree with them.

β. The second plan of treatment, to force the passage of the fœtus through the

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[1] Schillinger: *Ungar. Zeitschr*, V. 21. 1854.

[2] [The use of the colpeurynter and of uterine catheterization (especially the latter), require much more consideration than is devoted to them in the text. The other measures (especially the douche and sponge tent) for attaining the same ends, are more to be recommended in consequence of their recognized efficiency and decided innocuity. I have known untoward results arise from this use of uterine catherization.]

[3] Holst: *N. Z. f. Geburtsk.* Bd. 32. S. 85.

[4] Busch: *Schmidt's Jahrb.* 1849.

[5] Rul-Ogez: *Gaz. Méd. de Paris.* 1852.

[6] Kilian: *Geburtslehre.* 2 Bd. II. Thl.

[7] Velpeau: *Des convulsions chez 65 femmes*, Paris, 1834.

[8] Masson: *Union Méd.* 1853.

soft genital passages by mechanical power, is deserted by accoucheurs of modern times, almost without exception.

When artificial delivery (*accouchement forcé*) is attempted by introducing the hand in a conical form through a narrow os uteri, and when the cervix is narrow or very little dilated, it is generally found to be altogether impossible, or it sometimes leads to uterine ruptures dangerous to life; and thus the mother is subjected to greater dangers from the operation than from the eclampsia itself, of which no one can say whether any more paroxysms may come on and cause death.

But when the stage of dilatation is far advanced, when the cervix has completely disappeared, and the mouth of the womb opened from 1 to 2″, forcible delivery is no longer necessary, because the dilatation of the fine border of the mouth of the womb is commonly easily accomplished with the fingers, and delivery goes on quickly and spontaneously, or it may without danger be easily accelerated. For these reasons, we must decidedly join with those who oppose resort to the *accouchement forçé* as Böer,[1] Betschler, Nægele, and others, have done.

The dilatation of the soft parts by incision, which has been recommended by Baudelocque, is without danger only when merely the external os uteri or the vaginal portion of the cervix are incised a few lines deep; it is very dangerous if the os uteri be very thick, or if the undilated part of the cervix still form a canal from ½ to 1″ long, because then the incisions can no longer be exactly controlled, but penetrate too deeply; and the subsequent introduction of the hand, or extraction of the fœtus, may produce uterine lesions dangerous to life, and under which the patient may sink, after she has recovered from the eclampsia.

Hysterostomatomia should be confined to those cases only of eclampsia when there exists at the same time an organic stenosis of the external os uteri.

Our opinion, therefore, is, that forced delivery, with bloody or bloodless dilatation of the cervix, is never to be resorted to when any injury from it is to be feared; and we think the wise consideration of the success, from the above described methods of increasing the pains, affords sufficient reason to induce us to abstain from doing any harm either by rash officiousness or irresolute passiveness.

The commendation by Jörg[2] of the *accouchement forcé*, in preference to artificially induced premature labour, has lately been called in question, in an excellent manner, by Krause, who rightly points out that the *accouchement forcé* is possible only when the os uteri is so far opened as at least completely to admit one finger, and when it feels to a certain degree pliable, and that, to reach this condition, artificial premature labour is pre-eminently applicable; so that the two methods by no means exclude one another, but each may be used at its proper time.

γ. The third mode of treatment of the stage of dilatation in eclampsia consists,

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[1] Böer, L. J.: *Aphorismen über Fraisen besonders bei Schwangern und Gebärenden.* Abh. Bd. II. 1807.

[2] Jörg: *Zwangsmitteln gegen die Natur.* Leipzig, 1852.

according to the examples of Baudelocque[1] and Betschler,[2] in carefully abstaining from all operative interference, and never undertaking anything which can be effected by nature herself.

Careful critical examination of statistical statements has already shown the injurious consequences of such passive conduct on the part of the physician, and therefore we do not feel called upon to enter upon further reasons of disapproval.

C. Treatment of eclampsia during pregnancy.

Eclampsia appearing in the second half of pregnancy has generally premature labour as a consequence; or, in exceptional cases, yields completely to medical treatment. Great attention is, therefore, to be paid to making an exact diagnosis of uræmic eclampsia, to the violence, frequency, and danger of the paroxysms, and to the results of the use of medicines; and artificial premature labour is to be resorted to only when there is some probability of the mother being thereby saved, and so much the more, if death of the fœtus has already occurred.

Colpeurysis and uterine catheterization we consider, in this case, the most secure method.

If the agony comes on after a fit occurring during pregnancy, or the early part of labour, it is inadmissible to risk an *accouchement forçé*. In this case, it is much better to wait till the mother is dead, and thereupon to deliver the child by the Cæsarean section.

D. Treatment of eclampsia in the stage of the after-birth.

The treatment of the stage of the after-birth, in cases of eclampsia, is to be conducted on general principles; only attention must be paid not to delay too long the careful removal of the placenta, in order quickly to procure most valuable rest to the patient.

E. Treatment of eclampsia in childbed.

Cases of eclampsia occurring in childbed are to be treated according to ordinary principles. Large doses of opium are, as a general rule, very useful at this time. But here, as at other times, we advise the greatest caution in resorting to phlebotomy, and would only make use of it if opium and cold affusions are without effect, and if cyanosis of the face and frequency of the pulse increase to a very alarming extent in a very strong constitution.

Regarding the influence of hæmostasis (ligature of the extremities) in puerperal and childbed eclampsia, no observations are known to me. Since, according to Vogel's calculation, a leg may, by this plan, be made to contain thirty ounces of blood more than is normal, a revulsion or derivation equivalent to that from a large venesection is obtained. I therefore feel myself bound to direct attention to the investigation of hæmostasis in regard to its influence upon secondary hyperæmiæ and cyanosis, which generally follow the most violent eclamptic attacks.

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[1] Baudelocque, A. C.: *Sur les Convulsions.* Paris, 1822.
[2] Betschler: *Programm über die Eclampsie der Gebärenden.* Breslau, 1831.

When a comatose condition comes on in childbed, we maintain the greatest quietness of the patient and of those around her, and take care that too bright light and all noise be avoided; we set aside all medicines, as well as cold applications to the head, and encourage abundant diaphoresis by covering the body well; for, at this period, more is to be feared from serous infiltration of the brain than from true hyperæmia.

When the discharge of urine is scanty, the catheter is to be used, in order to avert retention and decomposition of urine in the bladder, resorption of carbonate of ammonia, and repeated outbreak of convulsive paroxysms.

When cholæmic eclampsia occurs, Bamberger discommends here also general blood-lettings, because, as in other similar cases of blood poisoning—for example, typhous, puerperal, alcoholic uræmiæ, etc.—they aggravate the tendency to rapid collapse, to further dissolution of the blood, and to hemorrhage. Strong purgatives (calomel, senna, jalap, croton oil, irritating and purging clysters) are most useful. When symptoms of depression prevail, then stimulating treatment is to be used to its fullest extent (cold affusion and douche on the head, embrocations of croton oil; internally, wine, ether, musk, camphor, and preparations of ammonia). On the other hand, if violent fever and symptoms of excitement prevail, then cold applications to the head, cold spongings, morphia, inhalation of chloroform, quinine, and mineral acids are to be used. All these means are, however, generally without any results, as well as the use of aconite, recommended by Ozanam as a specific.

Other convulsions resembling eclampsia—the apoplectic, cerebral, anæmic, toxic, and general spasms of typhus—are to be treated according to the general principles applicable when pregnancy has not existed, and also in cases of males. Obstetric interference in such cases must be decided on with great caution, in order not to increase the already existing danger to life.

Hysteric and epileptic convulsions do not generally require any treatment during pregnancy, because the use of medicines is generally without any results; and because it can never be justifiable to bring on artificial premature labour, because notwithstanding suffering from these affections, if the mother enjoy otherwise good health, the development of the fœtus is brought to completion, and because frequently, during childbed or after it, no improvement of these nervous affections is observed.

The chorea of pregnant women is not unfrequently connected with hydræmia; hence preparations of iron are to be employed. Although it is known that after the completion of delivery the disease generally ceases, yet we do not consider that, in that circumstance, there is sufficient to indicate the induction of artificial premature labour, as the disease is not dangerous to the life of the mother.

APPENDIX.[1]

TWO CASES OF PARTIAL CHOREA IN PREGNANCY; WITH REMARKS.

CHOREA is peculiarly a disease of childhood, but is occasionally observed also in the adult. When it does thus occur in the adult female, it is frequently found in connection with pregnancy. Numerous cases of this description are recorded by Lever, Meissner, Romberg, and others.

In some the disease is found to have continued from childhood, or from the period of commencing menstruation on till the occurrence of pregnancy. In others it has commenced with or during pregnancy, and in another class of cases it has a less distinct individuality, being found merely as the precursor or follower of paralysis of the limbs. The cases specially under consideration are peculiarly interesting, *firstly*, because the attacks, although severe, were partial, the lower limbs being the parts affected with the involuntary movements; and, *secondly*, because the attacks were periodical in their character, the movements in one case occurring only in the evening and early part of the night, and especially on awaking after the first sleep of an hour or less; in the other case, the motions being troublesome also only during the night.

This affection is one of a class of nervous disorders now better known than formerly, although their pathology is still confessedly obscure. In his work on Pregnancy (p. 4), Dr Montgomery takes notice of them after speaking of the anatomical changes in the hypogastrium consequent on pregnancy, and their more direct results. He says, that "from the stretching of the round ligaments of the uterus, as well as from the increased sensibility of the nerves which they contain, considerable uneasiness is felt in the direction of these cords, and about their termination at the sides of the pubes. This uneasiness extends also along the nerves of the thigh, producing numbness, cramps, and even considerable pain along the limb; which latter symptoms are often observed among the earliest indications of uterine irritation, whether arising from functional derangement, organic disease, or the healthy excitement of pregnancy. It is not unusual, under

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[1] See p. 50.

such circumstances, to find the power of one or both of the lower limbs considerably impaired; and in some few rare instances they have become partially or completely paralytic, and even hemiplegia has been observed, but to what degree the mere enlargement of the uterus is the agent in the production of such a state, seems very doubtful."

The observations, now sufficiently numerous, of similar nervous disorders during pregnancy seizing various regions of the upper half of the body instead of the lower, demonstrate that the mere mechanical enlargement and consequent pressure of the uterus on the neighbouring parts cannot be in all cases their direct cause. As examples of these affections, we may refer to the cramps or paralyses of one or both arms or hands, or even of single fingers,—to cases of amaurosis or other derangements of vision, also of deafness, as well as the more ordinary disturbance of the function of taste and of the purely cerebral functions.

Moreover, cases are constantly met with where extreme distension of the abdomen and consequent pressure on the hypogastric regions do not induce disturbance of the nervous functions of the lower limbs, although the circulation may be seriously obstructed. Of this, large fibrous or ovarian tumours are familiar examples. I lately met with a case of pregnancy aptly illustrative of the effects of the pressure of the gravid uterus upon the circulation, without disturbing the innervation of the lower limbs. A young woman, unmarried, employed as a housemaid, came to consult me in regard to swelling of the lower limbs. On examination the veins were found in the highest varicose condition, large, convoluted, and prominent from the ankle upwards. She had not menstruated for seven months, and, on inquiry, she admitted the existence of an abdominal tumour. In it a fœtal heart's pulsations were easily detected. The uterus was developed transversely, the head of the child lying in one iliac fossa, and the body directed across to the corresponding part on the other side. It appeared that she had been using her stays so as violently to compress her figure, and hide the abdominal swelling. The result of this was the abnormal position of the child *in utero*, and its powerful pressure against the parts about the brim of the pelvis, causing obstruction of the returning current of blood. There were no cramps in the limbs, nor did she complain of any want of strength in them.

Although the weight and pressure of the gravid uterus affords no sufficient explanation of these affections, there can be no doubt of the fact that the lower limbs are more liable to be implicated than any other part. The cases of chorea related in this paper, affecting the lower limbs, afford striking illustrations of this; for this disease is scarcely known to attack the inferior extremities alone under any other circumstances. Romberg, indeed, says that "the inferior extremities are never attacked alone."[1] But this restricted statement is proved incorrect by the cases occurring during pregnancy, and by other observations.[2]

Mrs H. was first affected with chorea when she was sixteen years of age. Menstruation did not commence at this time, but was delayed till she was

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[1] *Diseases of the Nervous System.* Sydenham. Transl., vol. ii., p. 54.

[2] Nous l'avons vue une fois limitée aux membres inférieurs, mais plus marquée du coté gauche.—*Grisolle, Pathol. Int.*, tome ii., p. 611.

eighteen. The motions began in a very slight form, affecting first the ankles only, but soon the whole legs and also the hands and forearms. They were always much aggravated during summer, and continued more or less severe till about the beginning of 1850, when they were either entirely absent or so little troublesome as not to attract her attention. In the course of 1850 she was married, at the age of thirty-two years. After this she continued in very good health till about the middle period of her first pregnancy, which ended favourably in the birth of a male child in July 1851. She had passed through the first half of the pregnancy in good health, but after that, she began to be troubled with the choreic motions of the entire lower limbs, and chiefly, as on former occasions, of that of the left side. The movements were described as very annoying, only at night, and chiefly on lying down in bed, when they continued so long as to deprive her of much sleep. When the motions were worst in the limbs, there were occasionally also some affecting the hands, the forearms being still. The movements occurred only in the evening and at night, unless she sat in one position for a long time, as in church, when they sometimes commenced.

The motions had been very troublesome for about six weeks before I was consulted. There was no other complaint. The morning sickness was only occasional. Her appearance was that of a delicate dyspeptic female; the skin was pallid, dry, and harsh; the tongue whitish, and deeply fissured in all directions; the bowels were regular and the urine natural. I ordered the use of large doses of carbonate of iron (ten grains) thrice daily, and a moderate dose of laudanum to be taken at bed-time; also the use of a full diet, with good beer. After this plan of treatment had been continued for some days, she was generally able to get to sleep in a short time after going to bed, without the laudanum. The iron and diet were persevered in till within a few days of her confinement; but for nearly a month before this, the motions had entirely disappeared. Her confinement was natural; only, during the inhalation of chloroform, the choreic movements were reproduced in a troublesome way. The recovery was not unfavourable, except that an abcess formed in one mamma. She nursed her child. In the end of 1852 she was again confined under favourable circumstances; and since the attack in the first pregnancy she has not had any motions worth mentioning. A third pregnancy has also recently terminated favourably, and without any appearance of chorea.

Mrs R., a lady of middle age, was travelling in Scotland during the summer of 1854. She was pregnant, and was gradually becoming worse and worse in general health, from symptoms which she connected with her being in that condition. In consequence she came to Edinburgh on her way home to England. Mrs R. has generally enjoyed good health, and never has had chorea,—is the mother of several children, the last only of which was stillborn. She is now in the sixth month of pregnancy,—has the look of a woman of originally good constitution, but is at present thin and of unhealthy appearance. Her pulse varies in frequency, is never rapid, but weak; the skin dry and scurfy; she complains of burning heat in hands and feet,—tongue not foul,—suffers greatly from vomiting after every meal,—no derangement of the heart's action, or of the function of the kidneys, can be discovered. There is a copious muco-purulent discharge from the vagina, which is much softened and dilated, and the *cervix uteri* is in a

swollen and abraded condition. Late in the evening involuntary movements of the lower limbs come on, causing great annoyance, and continuing till she goes to bed. Generally she falls into a short sleep, which is, however, soon interrupted, and then the motions recommence and continue for hours till she again falls asleep. On awaking, the choreic movements are again entirely absent till near evening.

The treatment pursued in this case was the same as that described in the former, only in addition the *cervix uteri* was twice cauterized with nitrate of silver, with an interval of three days between the operations, and an astringent lotion of decoction of oak bark with borax was injected to the amount of three ounces morning and evening. By these means the leucorrhœa was entirely arrested. For the irritability of stomach, a belladonna plaster was applied over the epigastrium, and was followed by great improvement. In about a fortnight the choreic movements disappeared, and my patient left Edinburgh, greatly improved in health. She subsequently was delivered of a dead child, a little before the full period of pregnancy.

The state of albuminuria, which has been shown to be intimately connected with some of the most important nervous affections of pregnancy and parturition, did not exist in the two preceding cases. Neither presented any of the signs of disease of the heart, nor had either of them ever suffered from rheumatism. They were both in the better classes of society, and were therefore well fed and cared for, and both were subject only to favourable psychical influences. But in both there was a distinct asthenic state of body, characterized by pallor of the surface, weakness of pulse, dryness of skin, and the other unmistakeable signs of general bad health; and in one the previous occurrence of chorea formed a strong predisposition.

The only condition with which this affection is at all liable to be confounded is fidgets. This latter is not unfrequently observed in pregnant females, existing in the lower limbs to a degree so intense as to assume the importance of a disease. It is generally brought on by either voluntary or constrained abstinence from exercise, and is aggravated by the engorged state of the vessels of the lower limbs, induced partly by the anatomical conditions of pregnancy, and partly by the absence of the contractions of the muscles of the limb. It may be regarded as a muscular affection, and is removed by exercise of the limbs, or by rubbing and kneading the affected parts.

In the two cases just related, the treatment pursued was simply that of symptoms. The numerous peculiar plans of treatment of chorea were disregarded. It was my object, by the use of iron, to remedy the asthenic condition generally, and also to improve the state of the blood indicated by the pallor of the lips and general surface. But I am inclined to regard the use of opiates to procure sleep as having been the chief agent in improving the condition of these females. Both were suffering from want of sleep, of which also they complained. Small doses of laudanum were placed at their bedsides, and if they found the movements continue so as to prevent sleep, they were enjoined to have recourse to the opiate. After a time they improved, so as to procure sleep without resorting to this means.

In addition to iron and opiates, various other remedies were used intermedi-

ately to regulate the bowels, to palliate sickness, and to meet other passing indications. In one of the cases an aggravated leucorrhœa demanded special attention. The abraded *os uteri* was twice cauterized with nitrate of silver, and by this, along with the use of an astringent lotion twice daily in small quantity, and gently injected, the discharge was arrested, a result manifestly favourable to recovery.

It has frequently been remarked by authors, that cases of chorea in pregnancy do not recover till after confinement. Both of these patients, however, got completely rid of the motions after a few weeks of treatment.—*Edinburgh Medical and Surgical Journal, January* 1854.

THE END.

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