Measures Which Tend to Prevent the Formation of Toxic Wastes.—*Diet.*—In the light of the conclusions submitted in the foregoing pages, it becomes evident, in this connection, (1) that while any food ingested is converted into nucleoproteid granules, we are dealing mainly with poisons formed during the breaking-down of worn-out living substances, *i.e.*, during catabolism; and (2) that these poisons form, owing to insufficiency of those constituents of the blood which carry on catabolism and destroy the toxic wastes in the blood* This, in turn, suggests as a fundamental principle, the imperative need of equipoise between these properties of the blood and the food-intake. In the majority of cases the latter is excessive, and the cellular elements are burdened with proteids which cannot be completely converted into eliminable products. The blood, therefore, is loaded with substances which irritate the vasomotor center and thus provoke the seizures.*

The practical indication is obvious, namely: to allow the patient only the quantity of food strictly necessary to the needs of the body. Indeed, neurologists of wide experience have found that the best results are obtained when the food consists mainly of cereals, milk, fruits and butter. Some include eggs, but others object to them. Meat, if allowed at all, should be strictly limited to the midday meal, and even then in small quantities. Fats, fried foods and pastry often prove harmful. Stimulants which tend primarily to cause a rise of the blood-pressure, *i.e.*, alcohol, coffee and tea, should not be allowed.* Gastro-intestinal disorders sometimes suffice to awaken the disease. Appropriate treatment, including daily colon-flushing with normal saline solution, has proven curative in such cases.

This represents the teachings of experience based on thousands of cases treated collectively. Yet experimental dietetics sometimes point in other directions. Schlöss,[191] for instance, divided 16 cases into four groups: two of these were given meat *exclusively* six weeks, and a milk-and-vegetable diet during the succeeding six weeks. During the meat period the seizures were fewer than before; during the milk-and-vegetable period they increased. The reason for this suggests itself: the nucleoproteid obtained from meat *only* was not equal to that of the milk and vegetables, while the nuclein intake was greater. As a result the phosphorus liberated during catabolism was relatively greater, and being added, on reaching the blood (via the lymphatics) to the phosphorus-laden nuclein in the auto-antitoxin, increased the plasma's proteolytic

* *Author's conclusion.*
[191] Schlöss: *Loc. cit.*

and therefore antitoxic activity. Indeed, Dana[192] gives glycerophosphates among other remedies, while B. Smith[193] has used pure phosphorus with advantage. The conclusion reached by some, that these and similar experiments indicate that patients should not be deprived of meat is erroneous, however, for it is because Schlöss's patients were placed on an *absolute* meat diet, that they were benefited. Meat given *besides* other foods cannot but aggravate the disease, in accord with the teachings of experience.

Alcohol and absinthe are recognized vasomotor stimulants and convulsivants, and abstinence therefrom, as shown by Forel and others, have proved curative. Fleury[194] cites several cases in which the cessation of alcohol and reduction of the diet alone very markedly reduced the number of seizures—the lapse in one instance being three years—although the treatment had not been modified. Tea was found by Haig,[195] and coffee by Marburg,[196] to aggravate the disease.

Drugs Which Tend to Increase the Destruction of Toxic Wastes.—These agents all act by stimulating the test-organ, and therefore by increasing the blood's proteolytic activity.

Thyroid and Parathyroid Gland.—Thyroid gland is useful especially in young subjects (doubtless because gliosis is less apt to be present), owing to its direct action on the anterior pituitary body as the physiological stimulant of this organ.* It not only increases, through the adrenals, the proportion of adrenoxidase in the blood, but also, and through this action, that of auto-antitoxin. All the antitoxic powers of the blood, phagocytic and plasmatic, being enhanced, the toxic wastes are more perfectly destroyed.* (See also p. 716, vol. i.)

In epilepsy, *small* doses are alone effective, since large doses, by too rapidly increasing the formation of adrenoxidase, cause excessive metabolic activity in all tissues and an augmentation of wastes.* The dose should not exceed 1 grain (0.065 gm.) of thyroid gland to begin with, and be slowly increased if need be, until not more than 3 grains (0.19 gm.) are taken three times daily, after meals. If extract of parathyroid is used, $^1/_{40}$ grain (0.0016 gm.) should be the initial dose, gradually increased to $^1/_{20}$ grain (0.0032 gm.).*

No depressants should be given at the same time, since they tend to antagonize the action of these extracts by reducing the sensibility of the test-organ. As the bromides, especially *sodium*

* *Author's conclusion.*
[192] Dana: Med. Record, May 13, 1905.
[193] B. Smith: Texas Med. Jour., Aug., 1891.
[194] Fleury: Bull. gén. de thér., Jan. 23, 1900.
[195] Haig: Brain, vol. xix, p. 68, 1896.
[196] Marburg: Wiener klin. Rundschau, Bd. xiii, S. 337, 1899.

or *strontium bromide,* only do so when given in full doses, however, 10 to 15 grains (0.64 to 1 gm.) may be given on retiring, to control the irritability of the vasomotor center, while the cause of this irritability is being counteracted by the thyroid extract.* The bromide salt may be combined with 5 grains (0.3 gm.) of *acetanilid.*

Under "Hæmophilia," the increase of oxidase caused by small doses (3 grains—0.19 gm.) *t.i.d.* of thyroid extract is graphically illustrated by tests in several cases treated by W. J. Taylor. That it can counteract convulsions has been emphasized mainly by experimental physiologists. "In some animals, as in most carnivora," says Chittenden,[197] "complete removal of the thyroid tissue is followed by a rapid development of symptoms indicating a marked irritation of the nervous and muscular systems, as manifested by tetanus, *epileptiform* convulsions, etc., and terminating in death." Thyroid extract, as is well known, cuts short these convulsions, the animal suffering in no way as long as it is administered. By stimulating the adrenal center, it enhances the production of oxidase and, therefore, that of auto-antitoxin. Browning[198] caused cessation of convulsions in three young epileptics, two of whom suffered from rickets and the third from "disturbed metabolism," by giving $^3/_4$ grain (0.048 gm.) doses of thyroid extract twice daily. He states that these cases "are due to or associated with disturbances in the general metabolism of the body" and refers to them as "pseudo-epilepsy." All cases of idiopathic epilepsy should be considered in the same light if the term "pseudo-epilepsy" were warranted at all, since, as we have seen, Pierce Clark and others ascribe the disease to "an accumulation of waste-products." Browning's cases were *bona fide* cases of epilepsy, and what they do teach is that small doses of thyroid extract are alone indicated. These, however, as my own observations have shown, must be *very gradually* increased according to the effect on the patient. I usually begin with one grain (0.065 gm.) at each meal, giving a small dose of sodium bromide on retiring at first, if necessary, and gradually reducing the dose, until none is given at all. Some cases are benefited by larger doses of thyroid. Thus, in a case treated by a colleague, under my supervision, the nurse, through a misinterpretation of instructions, doubled the dose; the result was considerable improvement. In adults, however, as it did in cases reported by Clarke[199] and Bourneville,[200] it failed to arrest the seizures. In fact, in the last-named neurologist's cases, it increased the number of seizures. But the cause of this is evident: the extract was administered in full doses, and excessive metabolism was produced, causing an excessive production of wastes. As the cases were adults, diffuse gliosis may have been present, recovery being then impracticable. It is in young subjects that the best results are obtained with thyroid extract used as above. Professor Osborne, of Yale,[201] wrote recently: "I have now on my records a number of cases of epilepsy treated successfully with thyroid. My attention was first drawn to the use of this preparation in epilepsy by several cases occuring in women at the time of the menopause, the attacks showing a suggestive periodicity.... I found that I could control and prevent the epileptic attacks as well with thyroid as could be done with bromides, and with much better results to the system."

* *Author's conclusion.*
[197] Chittenden: Trans. Congr. Amer. Phys. and Surgs., vol. iv, p. 87, 1898.
[198] Browning: Jour. of Nerv. and Mental Dis., Oct., 1902.
[199] Clarke: Amer. Jour. of Insanity, Oct., 1895.
[200] Bourneville: Progrès méd., vol. xxiv. p. 20, 1896.
[201] Osborne: Jour. Amer. Med. Assoc., Nov. 3, 1906.

Sodium Salicylate and Salicin.—The salicylates increase very actively the elimination of urea and uric acid, by actively stimulating the adrenal center. Moreover, they excite abnormally the sympathetic center and cause constriction of the peripheral arterioles and, therefore, reduce the convulsions by limiting the cerebro-spinal hyperæmia. In adults 15 grains (1 gm.) of sodium salicylate or salicin 5 grains (0.3 gm.) may be given three times daily, giving a small dose (10 grains—0.64 gm.) sodium bromide at bedtime.

The salicylates have been recommended by Haig[202] and used with advantage, but with potassium iodide. William Pepper likewise used the salicylates with success. A number of cases have been reported in which it reduced the number of seizures. W. J. Vincent,[203] in a severe and carefully studied case, in which all familiar remedial measures had been tried, arrested the seizures (nine months' test) with salicin 5 grains (0.3 gm.) three, then five times, in the twenty-four hours. Other cases were benefited.

Other drugs which tend to enhance catabolism and the destruction of wastes are *digitalis* and *strophanthus, adonis vernalis, iodide of sodium,* and *biniodide of mercury.* Their actual value will only be ascertained when given alone, *i.e.,* reserving depressants for the night. For the latter purpose *bromipin,* a brominized oil of sesamum, has been recommended in lieu of the bromides; *antipyrin, acetanilid,* and *urethan* likewise. The centers seem to lose their sensitiveness to remedies sooner in epilepsy than in other diseases, and a change of drugs often results in temporary improvement.

An *alkaline laxative,* preferably the citrate of magnesia, every other week, aids materially all the foregoing measures.*

Treatment of Status Epilepticus.—In this condition the main cause of the paroxysms is the intense vascular pressure, caused by a more or less great accumulation of toxic wastes. Here the bromides are of value when injected subcutaneously in the back, just below the angle of the scapula, 30 grains (2 gms.) of *sodium bromide* dissolved in one ounce (28 gms.) of sterile water being used repeatedly if need be. *Amyl nitrite* inhalations, which cause temporary paresis of the vasomotor center and relaxation of all the arteries, while useful to abort ordinary attacks when used during the aura, are seldom effective in status,

* *Author's conclusion.*
[202] Haig: "Uric Acid as a Factor in the Causation of Disease," London, 1892.
[203] W. J. Vincent: Jour. of Mental Sci., Apr., 1904.

but used in addition to the bromide injections, prove efficient. *Blood-letting* has been highly recommended; *hypodermoclysis* suggests itself as a valuable measure used immediately after the bleeding to aid in the elimination, through the marked diuresis it provokes, the elimination of waste-products.

The use of sodium bromide as above was found highly useful in the Mass. Hospital for Epileptics by Morton and Hodskins.[204] They also administer prophylatic injections of 20 minims each (in all 12 grains) when two seizures occur in succession. Chloroform is sometimes recommended, but Pierce Clark[205] states that chloroform should never be used during the sleep or stupor stage of the access, as it may cause coma and even death. According to Bondurant,[206] the best single remedy for status epilepticus is blood-letting.

TREATMENT OF JACKSONIAN AND REFLEX EPILEPSY.—When the lesion in Jacksonian epilepsy can be clearly located, the sooner operative procedures are resorted to the better. As a period of improvement follows almost any procedure, prudence in predicting a cure is desirable.

All cases of epilepsy should undergo a very careful examination lest any organ of special sense be the source of reflex seizures. Any abnormal condition should, if possible, be corrected, even if it happen not to be the spasmogenic factor, since it always tends to aggravate the disease.

EPILEPTOID DISORDERS.

INFANTILE ECLAMPSIA OR CONVULSIONS.—The seizures of this disorder, which resemble those of epilepsy, are ascribable to a similar morbid process,* autotoxins being, as a rule, derived from the gastro-intestinal canal. They may also be due to general hypocatabolism, however, as is observed in children suffering from rickets; or, they may occur as an intercurrent symptom of acute infections, where they are caused by the inability of the adrenal system to cope successfully with the aggregate of detritus, wastes, etc., which appear in the blood under such conditions.* The paroxysms may be brought on by reflex action, through dentition, precisely, as we have seen under Epilepsy, as is the case under the influence of nasal growths, phimosis, etc., *i.e.*, through irritation of the corresponding centers in the posterior pituitary.*

* *Author's conclusion.*
[204] Morton and Hodskins: Boston Med. and Surg. Jour., June 15, 1905.
[205] Pierce Clark: Pediatrics, Aug. 15, 1897.
[206] Bondurant: Amer. Jour. of Insanity, July, 1894.

The medicinal *treatment* of infantile convulsions is, on the whole, similar to that of the disorders studied in this chapter. An important feature, however, is the careful search for conditions, such as the teething, phimosis, rickets, etc., and their elimination as pathogenic factors. The convulsive, *i.e.*, epileptic, habit may be developed in children left untreated, since repeated hyperæmia of the neuroglia* may provoke a gliosis and place the case beyond our aid.

PUERPERAL ECLAMPSIA.

SYNONYMS.—*Puerperal Convulsions; Child-bed Fits.*

Definition.—Puerperal eclampsia, an acute disorder of pregnancy and parturition characterized by periodical convulsions, is due to an accumulation of toxic wastes in the blood, owing to inability of the adrenal system to convert the excess of wastes due to the presence of the fœtus, into benign and eliminable end-products. As these toxic wastes provoke inordinate vascular tension, an excess of blood is driven into all capillaries, including those of the spinal system and cortex. Both the latter being thus rendered hyperexcitable, a convulsion occurs when this hyperexcitability is suddenly increased by the appearance in the blood of an excess of auto-antitoxin, the result, in turn, of a sudden resumption of defensive activity by the adrenal system when the blood becomes sufficiently toxic to enforce it. The convulsion lasts until the toxic wastes are converted more or less efficiently into harmless and eliminable end-products.**

Symptoms and Pathology.—The symptomatology of puerperal eclampsia, as regards the convulsions, closely resembles that of epilepsy. It may be divided into three stages: (1) the invasion, (2) a short period of tonic spasm, and (3) the period of clonic convulsions.

The period of *invasion* is generally attended (besides other phenomena reviewed under "Etiology and Pathogenesis") with a severe frontal headache, sudden flashes, more or less blurred vision, vertigo, and mental torpor or excitement. The temperature gradually rises and the pulse and heart-beat become steadily stronger. Nausea and vomiting may also occur, the patient com-

* *Author's conclusion.*
** *Author's definition.*

plaining of pain in the epigastrium. More or less severe chills mark the approach of the seizure in most cases.

The *tonic* spasm may, however, occur suddenly, *i.e.*, without warning, and is usually very brief. The eyes remain wide-open and staring, the pupils being dilated, the face is very pale and consciousness is lost. The spasm consists in pronation and supination of the forearms, the fingers being tightly closed around the thumbs, and rigidity of the legs. The head and mouth are drawn to one side, generally the right, the breath is "held" through arrested action of the respiratory muscles, and opisthotonos may occur. The tonic spasm may appear alone, and recur, thus constituting the only form of convulsion witnessed. As a rule, however, it lapses into the clonic seizure.

The onset of the *clonic* stage is marked by a change in the appearance of the patient, the face becoming deeply congested, tumefied, then cyanotic. The eyeballs, fixed before, now move rapidly from side to side, the lids closing and opening rapidly, the pupils, from dilated, becoming contracted. The muscles of the face and jaws are violently contracted, the tongue being sometimes severely bitten; the patient foams at the mouth, and the head is tossed or rolled from side to side with great rapidity and violence. All the extremities are thrown around vigorously and rapidly flexed and extended, the back being arched in opisthotonos or to the side. The respiration is markedly impeded and often stertorous; the pulse and heart at first beat slowly and forcibly, the veins of the neck being turgid and distended, but later on the cardiac action may become irregular. The temperature in most cases rises to 104° F. (40° C.); in some it may reach much higher, *i.e.*, 110° F. (43.3° C.).

After a period varying from one-half to three minutes the paroxysm loses its violence, and the patient falls into a coma or a deep sleep. Sometimes a new paroxysm recurs while the patient is still comatose, and is followed by others in more or less rapid succession. Rarely, the patient dies during the first coma. In favorable cases, the seizures become less frequent or cease immediately after delivery.

Etiology and Pathogenesis.—The convulsions are due, as in epilepsy, to irritation, by blood-poisons, of the vasomotor and sympathetic centers. All the vessels of the body being violently

contracted, a wave of blood is forced into all capillaries, including the cellular elements and neuroglia of the cerebro-spinal system.* The activity of the cortex as a sensory organ being suddenly enhanced, a flood of impulses—of the voluntary type—is transmitted to every portion of the spinal system and the seizure occurs.*

The arterial pressure is so intense that the capillaries are sometimes found ruptured, hæmorrhagic lesions being found even in the placenta. This violent hyperæmia is the cause of the great fatality of eclampsia to the infant.*

The kinship between epilepsy and eclampsia is so close that some authors, Osthoff and Lantos,[207] for instance, consider the latter as an acute form of epilepsy. Others, again, compare it to hystero-epilepsy. Oliver[208] reported a fatal case of epilepsy in a young puerperal patient with no antecedent history of the disease.

That a marked and widespread vascular constriction and general capillary hyperæmia are present has been conclusively demonstrated. Not only does the facial congestion, the engorged veins of the surface, betoken the presence of these conditions, but as observed by Lubarsch,[209] multiple hæmorrhages are to be found in every part of the body: the liver, kidneys, stomach, large intestine, endocardium, lungs, etc., and in the pia mater and cortex. Schmorl[210] also found punctiform hæmorrhage of meninges and cortex, and moreover of the central ganglia. Similar lesions, as to the cortex, were noted by Leusden,[211] who also observed many ruptured capillaries, the blood flowing in the surrounding tissues, forming clots. Massen[212] found the veins of various regions completely thrombosed, and also "considerable dilatation of the cerebral capillaries," the blood having been forced into them, doubtless by the constricted arteries. All organs showed hæmorrhagic lesions of some kind. Cassaet and Chambrelent[213] found hæmorrhagic lesions in the still-born infants similar to those of the mother, and ascribe to this cause the great mortality of infants in eclamptics. The hepatic lesions of eclampsia have also received considerable attention from Jürgens,[214] Klebs, Pilliet,[215] Bouffe de St. Blaise and others, since all found in this organ hæmorrhagic and embolic foci. Finally, Blumreich and Zuntz[216] found experimentally that the brain of pregnant animals was much more susceptible to irritation than that of non-pregnant ones.

We thus have ample testimony to the effect that, as in epilepsy, the cortex is violently congested, and if in the latter disease this can provoke convulsions, there is no ground for doubt that the case is the same in puerperal eclampsia. The connection with the vasomotor center is well expressed by Herz:[217] "Even slightly toxic products in the blood

* *Author's conclusion.*

[207] Lantos: Archiv f. Gynaek., Bd. xxxii, S. 364, 1888.
[208] Oliver: Lancet, May 26, 1894.
[209] Lubarsch: Corr. f. schweizer Aerzte, Bd. xxi, S. 255, 1891.
[210] Schmorl: "Pathologisch-Anatomische Unter. u. Puerperal Eklampsia," Leipzig, 1893.
[211] Leusden: Virchow's Archiv, Bd. cxlii, S. 1, 1895.
[212] Massen: Ann. de gynécol. et d'obstét., vol. xl, p. 227, 1893.
[213] Cassaet and Chambrelent: Revue médico-chir. des mal. des Femmes, vol. xvii, p. 600, 1895.
[214] Jürgens: Berl. klin. Woch., Bd. xxiii, S. 874, 1886.
[215] Pilliet: Nouvelles arch. d'obstét. et de gynéc., vol. v, p. 600, 1900.
[216] Blumreich and Zuntz: Arch. f. Gynäk., Bd. lxv, S. 737, 1902.
[217] Herz: Wiener med. Woch., Bd. i, S. 113, 174, 227, 284, 326, 381, 1900.

of women in childbed are sufficient to irritate the vasomotor centers, which are then in a condition of increased excitability." Finally, Krönig[218] found by means of the Riva-Rocci sphygmomanometer that the blood-pressure was very high, especially in post-partum eclampsias, an observation confirmed by H. Richardson.[219] Vaquez[220] wrote recently that none of the theories in vogue in regard to the etiology of eclampsia took into account the main and essential feature, viz., arterial hypertension. He had evidently overlooked the abundant evidence to that effect in literature.

The spasmogenic poisons are intermediate products of tissue catabolism which accumulate in the mother's blood when her auto-protective mechanism, the adrenal system, is inefficient, *i.e.*, functionally hypoactive.*

During pregnancy the mother's blood becomes increasingly laden with waste-products, those of the developing fœtus being added to her own. To protect her organism, her adrenal system, including, of course, the thyroid apparatus, becomes increasingly active, owing to the exciting action of these products on the test-organ,* to insure destruction of all wastes as soon as they are found.* When the adrenal system does not become sufficiently active to enhance adequately the blood's antitoxic properties—including phagocytosis—the toxic wastes are allowed to accumulate in the blood* in sufficient quantities to provoke convulsions, *i.e.*, the eclamptic seizures.

Williamson, of Johns Hopkins,[221] in his recently published textbook says: "Up to the present time satisfactory proof has not been adduced in support of the bacterial nature of eclampsia, nor does it seem likely to be forthcoming," and he considers it probable that the "morbid process is caused by some as yet unknown poisonous substance circulating in the blood which may give rise to lesions of varying intensity in the several organs." The lesions to which he refers include those I have ascribed to excessive hyperæmia in all organs, the result of irritation of the general vasomotor center. After alluding to the fact that it was Bouchard who opened up the field of auto-intoxication, he writes: "Rivière, in 1888, was the first to put forward the theory that eclampsia was an auto-intoxication resulting from the heaping up of some substance in the system." Tarnier and Chambrelent[222] and others found the toxicity of the blood considerably increased, but this question is still *sub judice*. Nevertheless the kinship with epilepsy again suggests itself, since Ludwig and Savor[223] and Hofmann[224] regard carbonic acid as the spasmogenic agent, while Szili[225] ascribes this rôle to some "intermediate products from the proteid molecule." I would again suggest that several poisons should be incriminated, since the nervous system

* *Author's conclusion.*

[218] Krönig: Verb. d. deut. Gesellsch. f. Gynäk., Bd. ix, S. 313, 1901.
[219] Richardson: Amer. Medicine, Sept. 12, 1903.
[220] Vaquez: Semaine méd., vol. xxvii, p. 121, 1907.
[221] Williamson: "Obstetrics," p. 703, 1903.
[222] Tarnier and Chambrelent: Annales de gynéc. et d'obstét., vol. xxxviii, p. 321, 1892.
[223] Ludwig and Savor: Monats. f. Geb. u. Gyn., Bd. i, S. 447, 1895.
[224] Hofmann: Centralbl. f. inn. Med., July 16, 1898.
[225] Szili: Berl. klin. Woch., Oct. 22, 1900.

also supplies intermediate wastes under such conditions differing from those of other tissues. In a recent comprehensive biochemical study of the question, however, Zweifel[226] found that the eclamptic seizure followed the accumulation of lactic acid—which he ascribes to deficient oxygenation—in the blood, and that after the seizures this acid had disappeared.

That the mother's auto-protective resources are developed coincidently with the growth of the fœtus through a corresponding augmentation of the functional activity of the adrenal system is fully sustained by experimental evidence. As to the *pituitary body*, L. Comte[227] found microscopically that, during pregnancy, the *anterior* pituitary was hypertrophied; it was also very much heavier and larger. This was confirmed by Launois and Mulon[228] in two instances, one of the parturients having died eclamptic. They found, moreover, a marked increase of the cellular elements. In a more recent work[229] Prof. Launois reiterates his previous conclusion that in pregnancy the anterior lobe is in a state of marked "hyperactivity".

Lang,[230] in a series of 133 cases of pregnancy, found the *thyroid* enlarged in 108, the organ beginning to enlarge about the fifth month. This increase in volume ceased, however, if thyroid extract was administered, and began again when the extract was withdrawn. On the other hand, Verstraeten and Vanderlinden[231] and Nicholson[232] having concluded that in eclampsia nitrogenous metabolism is impaired, owing to insufficiency of the thyroid, the latter author tried thyroid extract and found that it counteracted the morbid symptoms—a fact repeatedly confirmed since, as shown under "Treatment."

As to the *parathyroids*, Vassale[233] reviewed recently his own labors and those of Pepere, Zanfrognini and others, which showed lesions of these organs after death from eclampsia; of Zanfrognini, Ernheim, Thaler and Adler, which showed that parathyroid insufficiency beginning during the last three months of pregnancy caused grave (experimental) eclampsia. In two out of three dogs in which the parathyroids had been removed, Vassale was able to prevent eclampsia by giving large quantities of parathyroid extract orally. Frommer[234] has confirmed the general trend of these views.

That stimulation of the *adrenals* by the pituitary body increases the antitoxic power of the blood, we have seen under "Epilepsy." The need of such a function in eclampsia is self-evident in view of the fact that Massin[235] found that at the end of pregnancy the blood contains an abundance of "partially oxidized products or leucomaines." It is only a question whether this function—carried on by the adrenal system—is equal to the occasion. As in epilepsy, also, the eclamptic stage is an effort of protective functions to destroy the poison. Indeed, Emery[236] observed that a polynuclear leucocytosis—the identical cells which we have seen furnish the blood its nucleo-proteid granules—occurs at the onset of the fever, and Kollmann[237] found a large increase of fibrinogen, a substance rich in nucleo-proteid, in the blood of eclamptics.

[226] Zweifel: Arch. f. Gynäk., Bd. lxxvi, S. 537, 1905.
[227] Comte: Thèse de Doctorat de Lausanne, 1898.
[228] Launois and Mulon: Ann. de gynéc. et d'obstét., 2 série, vol. i, p. 2, 1904.
[229] Launois: Thèse de la Faculté des Sciences de Paris, 1904.
[230] Lang: Zeit. f. Geburts. u. Gyn., Bd. xl, S. 34, 1889.
[231] Verstraeten and Vanderlinden: Ann. de la Soc. de méd. de Gand, vol. lxxvi, p. 72, 1897.
[232] Nicholson: Jour. of Obstet. and Gynec. for Brit. Empire, July, 1902.
[233] Vassale: Gaz. degli Ospedali, Aug. 5, 1906.
[234] Frommer: Monats. f. Geb. u. Gynäk., Bd. xxiv, S. 748, 1906.
[235] Massin: Centralbl. f. Gynäk., Bd. xix, S. 1105, 1895.
[236] Emery: Practitioner, Mar., 1905.
[237] Kollmann: Centralbl. f. Gynäk., Bd. xxi, S. 341, 1897.

Albuminuria is an index to the degree of vasoconstriction present.* When the vasoconstriction becomes excessive, there is added to the albuminuria, transudation of the albuminous portion of the blood (the serum) through the engorged capillaries, and œdema of the feet, legs, external genitals and face appears.* In marked cases, the trunk and the internal organs, especially the lungs, may likewise become œdematous.

The albuminuria of pregnancy, *when moderate,* is not due to a general constriction caused by the action of poisons on the general vasomotor center.* We have seen that it is the result of excessive vital activity in the muscular coats of the arteries, owing to the unusually stimulating properties of the blood supplied to them through the vasa vasorum and that it is purely mechanical.* Though albuminuria is not due to nephritis, renal irritation may be evoked by the inordinate work imposed upon the kidney. Moderate albuminuria alone is not a threatening condition, therefore; while moderate albuminuria *plus* renal casts may ultimately prove to be.* Albuminuria with œdema points to a marked vascular constriction which may become pathological.*

Lantos[238] found albuminuria in 60 per cent. of 600 newly delivered women, in over 70 per cent. of 268 primiparæ, and over 50 per cent. of 332 multiparæ, and concluded that it has no pathological significance. Palmer[239] also found albuminuria in about 50 per cent. of pregnant women examined at the Cincinnati Hospital. Pajot,[240] nearly twenty years ago, laid stress on the fact that many women who were highly albuminuric do not have eclampsia.

Willamson,[241] referring to the older view that nephritis was the fundamental cause of albuminuria, states that it was "gradually abandoned when it was found that only a small proportion of the women had eclampsia." This shows that the two phenomena are distinct entities. Indeed, as emphasized by Bar,[242] a normal action of the kidney does not prevent a fatal ending, while conversely, as shown by Van der Velde,[243] albuminuria may be absent notwithstanding the presence of marked renal disease. Nor do, as noted by Saft[244] and others, casts and albumin maintain a corresponding ratio. Just as the waste-products, as already stated, increase coincidently with the growth of the embryo, so did Saft, in his study of 707 cases, find that when albuminuria occurred, it was between the thirtieth and thirty-second weeks, and that while it was apt to become very considerable as the puerperium approached, it receded during the first days following labor. The general

* *Author's conclusion.*
[238] Lantos: *Loc. cit.*
[239] Palmer: Jour. Med. Coll. of Ohio, May, 1891.
[240] Pajot: Med. Press and Circular, Aug. 8, 1888.
[241] Williamson: *Loc. cit.*
[242] Bar: Bull. médical, vol. xiv, p. 77, 1900.
[243] Cited by Williamson: *Loc. cit.*
[244] Saft: Archiv f. Gynäk., Bd. li, S. 207, 1896.

vasoconstriction is likewise shown by the facts, pointed out by Nicholson,[245] that during pregnancy the right side of the heart is dilated, while the left side acts as though it were hypertrophied. Obviously, as he suggests, this is due to widespread and extreme vasoconstriction, a condition linked with the albuminuria of pregnancy by Allbutt, the blood being jammed into the venous system. The dilation of the right heart affords an idea of the intense centrifugal pressure to which the capillary system is submitted, and the familiar fact that the serum of the blood normally traverses the walls of the capillaries to penetrate the lymph spaces, indicates how it may traverse mechanically the renal filter, or invade the tissues and render them œdematous. Indeed, in a recent paper, Mynlieff[246] found marked intrarenal distention, sufficient to cause stasis at times, and limited only by the resistance of the capsule.

Although Charpentier[247] regards eclampsia without albuminuria as exceptional, he, Schroeder and Ingreslev[248] found numerous cases in literature in which it was absent. But this is readily accounted for by the fact that in some cases the brunt of the irritation is borne by the sympathetic centers. The arterioles being constricted, the circulation of the kidneys is inhibited as it is elsewhere, the pressure being only sufficient to sustain renal activity. In fact, the inhibition may exceed this limit: in 42 cases reported by T. K. Holmes,[249] the bladder contained no urine in 5 instances, although these cases had all been œdematous, while the only case in which albumin was absent, œdema was likewise absent. This case—in which the vasoconstriction was evidently not intense—had only one convulsion. Three cases attended with anasarca, and "solid" urine as to albuminuria, proved fatal.

The relative excretion of urea is an index to the proportion of the toxic waste-products that are present in the blood. The addition of fœtal products of metabolism to those of the mother necessarily involving an increase of them in the maternal blood, a corresponding increase in the urea excreted should occur. In eclampsia it is markedly reduced and the severity of a given case corresponds with the urea excreted, while improvement coincides with a material increase in the amount eliminated.

Butte[250] observed that when in eclampsia the proportion of urea excreted was normal, death usually followed; while recovery ensued as a rule, when it was doubled. Marx[251] has long emphasized the fact that "urea is always found markedly diminished in the so-called pure toxæmias of pregnancy" and that "the amount of urea excreted always goes hand in hand with the condition of the patient." E. P. Davis[252] emphasizes the same fact and states that although urea is not itself a poison, diminution in the quantity excreted indicates that toxins are being retained. Jewett[253] regards a marked falling off as of grave import. Hélouin[254] found that in eclampsia the nitrogen eliminated in

[245] Nicholson: Jour. of Obstet. and Gynec. for Brit. Empire, Jan., 1905.
[246] Mynlieff: Centralbl. f. Gynäk., Bd. xxix, S. 392, 1905.
[247] Charpentier: Bull. de l'Acad. de méd. de Paris, 3 série, vol. xxix, pp. 32, 54, 1893.
[248] Cited by Williamson: *Loc. cit.*
[249] T. K. Holmes: Med. Age, Feb. 10, 1896.
[250] Butte: Ann. de la policlin. de Paris, vol. iii, p. 164, 1893.
[251] Marx: Sajous's "Analytical Cyclo. of Pract. Med.," vol. v, p. 375, 1900; Med. Exam. and Pract., Mar., 1903.
[252] E. P. Davis: Amer. Gyn. and Obstet., July, 1899.
[253] Jewett: Brooklyn Med. Jour., Aug., 1899.
[254] Hélouin: Thèse de Paris, 1899.