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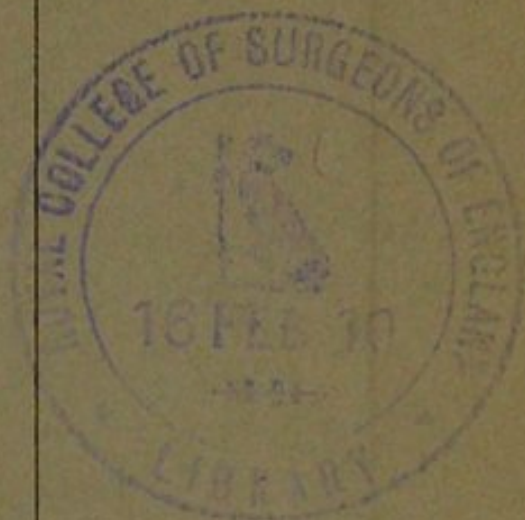
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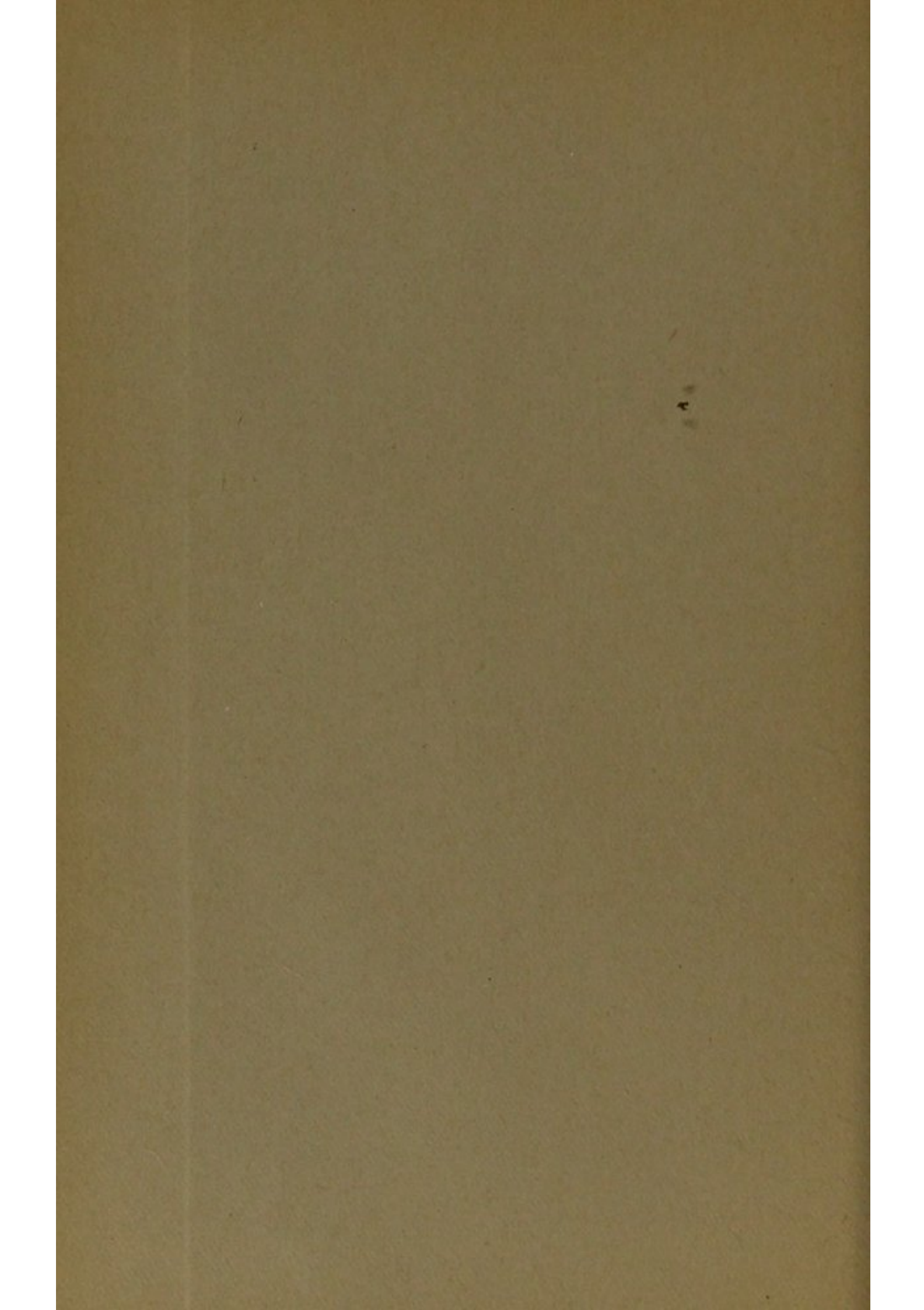
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## ON COMA AND THE VALUE OF THE OCULAR SIGNS OBSERVED THEREIN.\*

By BURTON CHANCE, M.D.,

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THERE is, I suppose, no condition in the daily rounds of the busy physician requiring more clear-headed understanding, and more prompt and efficient handling, than that confronting him when called to attend a person who has suddenly become unconscious, or one who has lapsed into coma, without having displayed any premonitory signs.

When we speak of coma we mean the complete loss of consciousness. The individual appears to be in the profoundest sleep; his perceptions and the exercise of his will are suspended. The face wears a confused look; his mouth is open and the tongue is dry. All conscious and unconscious response to sensory irritation is lost. Shouting and shaking will not arouse him. The extremities are relaxed. The breathing may be rhythmic, but it is frequently irregular; at times it is retarded and full, at others it has the Cheyne-Stokes character; while towards the close it becomes stertorous and stridulous. The reflexes are abolished. Swallowing is impossible. The sphincters are no longer resistant, and incontinence of urine and feces develops. The eyes may deviate from their normal

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axes; the pupils are dilated, or unequal and sluggish, or sharply contracted and immobile.

Coma always betokens a serious disturbance of the functions of the brain presiding over the vital activities of the body. It is brought about either through a blotting out of the cerebral centers, as in edema, or when an effusion presses upon them, or when the centers have been inhibited through inanition which may have been caused by defective circulation in the brain, or by the effects of poisons circulating in the blood in the course of a general toxemia. The brain may suffer from a deficient supply of arterial blood, or from an excess of venous blood, or from both these conditions variously combined.

This state of unconsciousness may come on in the course of a great variety of diseases; it may be at the end of a prolonged illness of definite character and be marked by distinct preliminary manifestations; or it may seize a man suddenly while he is in the midst of his daily business. It may be recovered from and the individual may have repeated attacks, or it may shortly end in death without manifesting a single sign to give one a clew as to its cause. The question paramount is obviously an immediate and exact diagnosis, particularly if the person is in a deep coma when seen for the first time. The value of a leading sign which may be reasonably depended upon is evident. Such a sign may be found, as I shall show in this paper, from the examination of the eyes. For, from the examinations of many cases, I am led to believe that coma arises from cardiovascular disturbances and that the characteristics elicited from the careful examination of the intra-ocular vessels are indicative of and entirely comparable to those found in the brain of such subjects.



Let us consider then the conditions under which coma may arise.

The most complete coma is seen in gross disturbances of the cerebral circulation when it may come on quickly without premonitory signs. Embolism and thrombosis of the cerebral vessels will produce symptoms similar to those occasioned by their rupture. In a typical apoplectic stroke the patient falls into deep unconsciousness from which he cannot be aroused. The face is injected, sometimes cyanotic, or of an ashen hue. The respiration is noisy and the pulse is slow. We must therefore ask ourselves the question when called to a case of suspected apoplexy whether the coma has been caused by an obstruction of the vessels or by their rupture.

The age of the patient may guide us in determining the probable cause and help us in our hope for his recovery. We may suspect arterial obstruction if the patient is young or is in early middle life, if there is fibrosis of the arteries, or if within a brief period several incomplete attacks have occurred before the completely comatose condition has set in.

The diagnosis at times may be cleared by a study of the ocular symptoms. In a typical seizure ptosis of the upper eyelid is a not very uncommon symptom, and the eyes may be fixed in the position of "conjugate deviation" which has been brought about as the result of the suspension, for the time, of all the functions of the affected hemisphere, so that no stimuli can be transmitted to the visual center—which may be paralyzed itself also. The pupils vary; usually they are dilated, and sometimes unequal. In deep coma they are always inactive. If there be a loss of reflex on one side and the pupil is rigid, doubtless the coma is due to a hemiplegic lesion. When a hemorrhage has flooded the ventricles, or is



in the pons, however, there may be marked contraction of the pupils from irritation of the oculomotor nerve nucleus. An ophthalmoscopic examination will reveal in what an analogous condition the walls of the cerebral vessels must be. The retinal vessels are seen to be stiffened and more tortuous, and where they cross one another they show strangulations or constrictions with a diminution of their lumen. Above and below the points of constriction are found dense whitish streakings; and where the vessels cross, the blood columns are narrow and the vessel wall is either invisible or it presents a fusiform thickening. When a stiffened artery presses upon a softer vein, the vein is seen to curve under and around the artery. Here and there along the lines of the vessels and at the points of constriction, there may be small hemorrhages without apparent inflammatory changes. In extreme cases, the veins also may show analogous strangulations and saciform dilatations. These symptoms should be studied together with the results of the examination of the patient's cardiovascular system. By this time it may have been learned from other sources that the individual is the subject of renal disease. A large proportion of the cases of apoplexy occur in those with affected kidneys, especially those with granular contracted kidneys. Hemorrhage therefore is likely, if the heart is found to be hypertrophied and if the disease of the kidneys co-exists.

Consciousness may be lost in an attack of angina pectoris, and syncope may follow the epileptiform convulsive seizures attending profound disturbance of the heart's action which have been seen notably in instances of heart-block. The unconsciousness is doubtless due to cerebral anemia. In the case of the Adams-Stokes syndrome, the abnormally slow and



disordered pulse; the stiffened arteries or signs of cardiac disease; the age of the patient; the unwounded tongue; and the unrelaxed sphincters, are points to be regarded to confirm us in our hypothesis that it is not true epilepsy with which we have to deal.

Drink and narcotic poisons may produce coma which may be most difficult to distinguish from the coma of apoplexy, and, to make it still more difficult, sometimes a person who has been drinking may be stricken with apoplexy. In a simple drunken stupor there may be complete unconsciousness, yet the person may not be bereft of all power of motion; he certainly is not paralyzed. The pulse is not slow; it is usually frequent. The pupils are generally dilated. The eyes are injected and show no lateral deviation. When apoplexy comes on in a drunkard, the coma is deeper, stertor is present usually, and there may be evidences of hemiplegia in the greater flaccidity of the limbs on one side.

In narcotic poisoning, especially from opium, we are likely to encounter a gradual intensification of the coma. The coma is scarcely less complete, but as a rule it does not appear so suddenly as that of apoplexy. The pupils are much contracted; yet a hemorrhage into the pons may cause comatose symptoms with marked contraction of the pupils. In each of these conditions convulsive seizures may have preceded the coma; and because of the quiet respiration, and because paralytic symptoms may not be present early in apoplexy and pontine effusions, the diagnosis may be exceedingly difficult.

The coma attending uremia is as a rule less sudden in its onset than that of apoplexy, or that from narcotic poisoning, and it is only in the rarest instances that it has not been preceded by convulsions.



The convulsions are always general and not localized as in cerebral apoplexy; and the coma is not always profound; it may come on and pass off suddenly. The stertor is peculiar. It appears to come from the mouth, the loud sound of the expirations is not like the low guttural tones of apoplexy, but is pitched in a much higher key. Cheyne-Stokes respirations may be present. The ankles are swollen and the eyelids are puffy.

The pupils are as a rule widely dilated, though they may be normal and the reactions to light preserved. The preservation of the light reflex proves that the blindness is not caused by disease of the eyes or of the optic nerves, but is due to the disturbance of the cerebral centers—excretory matters retained in the blood circulating about them have poisoned them. If the pupillary light-reflex is absent, the action of these poisons may be upon both the central and peripheral parts of the visual paths, or only on the peripheral parts, the optic tracts, and the nerves.

The ophthalmoscopic examination may be negative, that is to say, there may be none of the signs of that form of retinitis commonly seen accompanying disease of the kidneys. The association of nephritic retinitis and uremic amaurosis is not common. The retinal inflammation noticed in Bright's disease is generally persistent and the recovery of vision seldom takes place, whereas, the blindness in uremia has been caused by toxemia, and the sight is restored rapidly and completely on the elimination of the poison.

In the coma of Bright's disease the cause is made manifest by the finding of albumin and casts in the urine and by there having been more or less definite attacks of uremia. Of course, the preceding history



may aid us greatly and the examination of the urine may conduce to certainty in the diagnosis, yet for obvious reasons they cannot always do so at once. Albumin may be present after apoplectic strokes and after convulsions not associated with uremia, for a slight amount of albumin may be found following violent epileptic seizures.

It is well to remember, however, that coma may develop without convulsive seizures. Twitchings of the muscles of the face and hands may occur, yet there may be cases in which these muscles are not involved. In such cases there may be no indications of previous renal disease and their nature may be overlooked unless the urine has been examined. In this connection it must be remembered that the absence of albumin must not be taken as proof that no renal disease exists.

Are we not coming more and more to believe that the presence of albumin in the urine does not always specifically indicate the health of the kidneys? Albumin and other compounds may be absent in the urine in definite lesions of the kidneys and they may be found present in cases of apparently healthy kidneys which have been examined after death from other causes. So we may be confident that the presence of albumin is not a criterion of the localization in the kidneys of a process which of itself alone is sapping the vitality of the individual in whom it is present. On the other hand the urine may be taken as only one of the indexes of the state of the general bodily health and we may infer from it that the organs from which it has been received are diseased in company with many others in the branches of the vascular tree.

In all cases of albuminuria we must consider carefully the condition of the cardiovascular system. The



heart must be examined for evidences of hypertrophy or dilatation; the arteries must be examined to ascertain whether their coats present evidences of thickening or other disease. The coexistence of such signs indicates a general arteriosclerosis while their absence renders it probable that the albumin is derived from the kidneys, or has been produced by some functional disturbance.

Except in the cases of coma following acute poisoning, in which those from alcohol and from opium are included, arterial degeneration is associated with all of them. Miliary aneurysms are often associated with sclerosis of the large arteries of the brain, though it was once difficult to explain the reason for such an association. From the study of the eye-grounds of a large number of arteriosclerotics, I have been led to believe the characteristics elicited from careful examination of the retinal vessels are indicative of, and entirely comparable to those found in the brain of such subjects; and the frequent association of these changes with manifest disease of the cerebral vessels only proves the contention. It must be remembered that in these examinations we have been dealing with visible pulsating blood-vessels and not with one only palpated with the fingers.

For years I have been skeptical of the accepted dictum that the causal relation is direct between visual disturbance and disease of the kidneys in the so-called "uremic amaurosis." As pointed out above, blindness in Bright's disease does not appear until late, and the visual disturbance is permanent, because portions of the retina and choroid have been blotted out by distinct hemorrhagic and inflammatory processes which we denominate "albuminuric retinitis."

On the other hand, the eye-grounds of the uremic



present no such changes, as a rule. The authorities state that "the fundus is here negative." Alas! the conditions are *not* negative. I have noticed distinct alterations in the appearance of the retinal vessels, and the chief importance of these retinal changes lies in their association with disease of practically a similar nature in the kidneys and the brain, as well as in the liver, and accompanying a more or less general arterial sclerosis. Many cases of supposed uremia are not specifically renal in origin at all, although albumin has been found in the urine. Such cases, I believe, are dependent upon a form of toxemia brought about by the retention of compounds in the vascular currents which would have been eliminated had not the coats of the vessels become impervious; and, further, I believe that to treat such affections upon the supposition that they are based solely upon deficient renal activity is to jeopardize the life of the patient.

Coma occurs sometimes in connection with diabetes also, and when that disease ends suddenly it is likely to do so by the so-called "diabetic coma." The comatose condition is preceded by vomiting usually, and by abdominal pains. The respiration is labored and the breath has a sweetish chloroform odor.

Diabetes develops in neurotic individuals, particularly in those who have lived a sedentary life and are the subjects of mental emotion from worrying or from excessive devotion to business. It is therefore a disease chiefly of the upper classes of society. It appears to be caused by the presence of acetone in the blood. Acetone is derived from acetic acid or from beta butyric acid, and the coma is doubtless due to an excess of these compounds. Certain it is that the disease is due to some ex-



tremely acid toxic agent in the blood, for examination of the urine will reveal the presence of sugar, acetone, and perhaps diacetic acid also.

The difficulties attending the diagnosis of diabetic coma are not many. It rarely happens that an individual affected with diabetes becomes unconscious without some warning; or that we are called to minister to one about whom we are unable to obtain some knowledge of the previous conditions. The ocular features are not of especial concern. The state of the pupils varies, they may be dilated, or they may be unequal. The retina usually presents evidences of chronic alterations of structure; and for our purposes in this discussion, diabetes and Bright's disease may be considered together.

Akin to diabetes is that state of bodily ill health brought about by the disordering of the gastrointestinal digestion which we now call "intestinal auto-intoxication." Here, from defective chemistry, there has been developed a general toxemia, the elimination of organic compounds has become perverted in quality and quantity, and there have been thrown into the system elements which have proved noxious to it.

Persons thus afflicted often have periods of hebetude, lapses of memory, and in some instances complete loss of consciousness. The difficulty in the last case arises in deciding whether or not we have to deal with uremia; indeed, in considering these cases I have been led to place them in very close analogy with the sudden "uremic" ones.

In this group no systematic classification has been made of the ocular symptoms present at the time of coma. They may be such as we find in simple "uremia," or there may be distinct evidences of congestion and stasis in the choroid with degeneration



of the vitreous. I have noticed in chronic melancholics, who have become stuporous, unequal and irregular pupils, cloudiness of the aqueous and vitreous, and congestion of the internal tunics. I would expect to find similar conditions in the autotoxic cases, for melancholics are invariably the victims of a depraved metabolism dependent upon gastrointestinal derangement.

Occasionally we are called to attend a person of advancing years, of sedentary habits, who has suddenly lost consciousness while at table after a hearty meal. This, too, is from a form of intestinal auto-intoxication, though it has been a common inference that uremia of renal origin is at the bottom of these attacks. A careful examination of such individuals will disclose signs of arteriosclerotic changes in the retinal vessels as well as disturbances in the vitreous and choroid, as outlined above. The urinous odor emanating from these prostrate persons is not due to uremic exhalations, but rather to vesical incontinence.

Ptomain poisoning belongs here, as it, too, may give rise to profound unconsciousness. Sometimes cerebral symptoms may be present in hepatic cirrhosis, when they manifest themselves in convulsive seizures, and a delirium which ends in coma. They, too, resemble similar attacks in uremia; yet a careful study will show that they have been caused by a toxemia comparable to that observed in the common form of intestinal intoxication.

A subject in the secondary stage of the general paralysis of the insane may sometimes be seized with syncopal attacks in which he falls, and, for a variable period, may lie unconscious. And such attacks with vertigo have been noticed in multiple sclerosis. In the early periods of these diseases,



these lapses of consciousness may be mistaken for hysterical fits. The eyes of the hysteric are healthy, however, and the visual paths to the brain are open, though luminous impressions are not perceived; the pupils, therefore, react to light. The paretic, on the other hand, commonly presents manifest pupillary changes, and during the syncopal attacks the optic nerves and retinas remain pallid; while in the case of the multiple sclerotic there may be distinct nystagmus and the nerve heads atrophic.

The coma following convulsive seizures in epilepsy may be due to a general toxemia, perhaps, as well as from the exhaustion of cerebral centers. The generation of muscle acids may be so excessive and enter the circulation in such quantities as to be profoundly poisonous. There may be no ocular changes whatever, but, occasionally, hemorrhages from the conjunctival vessels have been noticed, with congestion, as well as hemorrhages, in the choroids and retinas.

Only two more conditions remain to be touched upon: the coma following the convulsions of pregnancy, and the coma induced by inhalation of poisonous gases. Except in the case of an epileptic who has become pregnant, the convulsions and coma are without doubt due to a general toxic state dependent upon profound disturbance in the fluids of the abdominal circulation. I am excluding cases in which the women are the subjects of distinct Bright's disease.

In the matter of diagnosis we are inclined to place more dependence in the circumstantial evidence attending a case of gas poisoning than in the actual physical conditions present. There may be profound coma with deep, loud, snoring respirations, free from the odor of alcohol. The face is bright



red and there may be deeper colored patches in the skin. The mucous membranes are dark purplish and the scleral vessels are injected. I have examined the eyes of only one patient suffering from gas poisoning. The fundus in each eye of this patient was of an intense lake color. The pupils were widely dilated.

In studying this group of nervous phenomena, attention has been given to the prominence of the ocular symptoms in certain of the diseases marked by coma; and, as these symptoms are easily observable, their importance in diagnosing the probable causative factors becomes manifest. As we view the suddenly prostrated individual who lies apparently lifeless before us, the necessity for a distinct and signal symptom becomes urgent, in order that we may be led out of the confusion, as well as to provide for the patient's recovery without entailing further damage to his already embarrassed vitality. For, assuredly, the management of an apoplectic seizure must not be the same as that for the coma attending opium or other narcotics. Thus, the deviation of the axis of the eyes, or the condition of the pupils, may designate whether there is a suppression of the cerebral centers or only their irritation from disturbed nutrition.

The finding of albumin in the urine of an unconscious person, or the detection of an urinous odor emanating from his body, is not sufficient proof to guarantee the presumption that we have before us a case of uremia of distinctly renal origin. We have seen how albumin can occur in cases without renal insufficiency, that it may be present in obstruction of the cerebral vessels, and we know that it is frequently found in the comatose condition following epileptic convulsions.



Uremic amaurosis is still an obscure subject, but I have no hesitation in saying that, when all the cases are analyzed, we shall find the large number of them dependent upon a general toxemia generated by a disturbance in the chemic and metabolic processes in the gastrointestinal tract. Remedies and other measures designed to eliminate the festering products in the intestinal tube, very commonly are effectual in relieving the blindness, too; and, except in the case of late Bright's disease, in which ocular disturbances have existed long before coma has set in, there are no markedly visible changes in the intraocular tunics. It is my belief, however, that careful examination of the retinal blood-vessels, from the disks out to their finest extremities, will disclose periarterial and endoarterial changes. These vascular changes are comparative indexes of the state of the cerebral circulation.

In studying cases of coma it is our duty to attend to the careful examination of the ocular fundus. Except in the case of acute narcosis following the ingestion of well-known poisons, there are distinct arterial changes to be seen; and as transient visual disturbances may arise from an interference in the nutrition of the cells of the retina, so in like manner, cerebration, psychic and physical, may be interrupted through nutritional changes. Again, as the extremity of arterial changes in the eye may culminate in the rupture of the vessels, whereby areas of the retina are blotted out, so may there be rupture of the cerebral vessels whereby not only may regions of the brain be affected but life itself may be blotted out.

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