

conditions: O. D. + .25 + .62 x 90, O. S. + .25 + .50 x 90, and 3° of exophoria. Prescribed above glasses to be worn constantly, in addition to which I advised employment of prismatic exercise for the improvement of his weakened internal recti. Within one month he noticed a marked betterment in his physical condition, which, without the use of internal remedies, continued to so improve that he was finally enabled to pursue his professional course of study with satisfaction.

CASE 9.—Miss E. S., aged 16, presented a history as follows: Since 6 years of age has had indistinct vision for distance and difficulty in reading which has gradually become worse, attended with a rolling of eyes, twitching of lids, spasmodic contortions of the face and shoulders, and an increasing nervous irritability. Examination of the eyes revealed the following conditions: O. D. — 3.00 — 1.50 x 180, O. S. — 3.00 — 2.00 x 180, and 1° of left hyperphoria. She was instructed to wear the above correction constantly whereby the muscular imbalance was also remedied by deceleration of left lens. In the course of a year, without the use of any medication, the above symptoms practically disappeared and she gained markedly in weight. Two years later, however, she began to notice an irritation of the eyes, a blurring of distant vision, and some twitching about the eyes. Examination showed the following conditions: O. D. — 4.00 — 1.25 x 180, O. S. — 4.00 — 2.25 x 180, and 3° of left hyperphoria instead of above. With the correction of these defects the symptoms again disappeared in the course of a few months.

CASE 10.—Mr. C. L. C., aged 28, gave the following history: During the past five years he has complained of a pulling sensation in his eyes, feeling of constriction about the head, gastric disorder, loss of weight, and a marked nervous exhaustion, which finally terminated in an acute attack of melancholia. During the past six months he has been unable to use his eyes for ordinary reading. An examination of his ocular condition revealed the following: O. D. + 3.00 + .88 x 90, O. S. + 2.00 + .75 x 90, and 4° of esophoria. A partial correction was prescribed for constant use, and gradually increased until he was able to employ the full correction. In the course of a few months his former symptoms practically disappeared, and he was enabled to carry on his former occupation with satisfaction. Occasionally he would notice a return of some of his symptoms; but these were readily traced to a malposition of his lenses, due to a bending of his frame, on the correction of which the symptoms would disappear.

In conclusion, I wish to emphasize the fact that the foregoing illustrative cases of eye-strain are not rare in occurrence nor are the reported favorable results of ocular treatment exceptional, but they are frequently being encountered and successfully treated by those ophthalmologists who are doing thorough, scientific and conscientious refractive work. In contradistinction to these facts, however, I am sorry to admit that there is an alarming amount of humbuggery practiced in the "fitting of glasses." In making several thousand examinations of the eyes I have found that over 90 per cent. of refractive errors are astigmatic, one-quarter of a diopter or more, while 50 per cent. of the lenses prescribed from various sources are merely spherical. In other words, probably 75 per cent. of the glasses worn to-day do not accurately correct the ocular defects of the wearer, not taking into consideration muscular imbalances. When it is remembered, however, that the greater percentage of these glasses are furnished by pseudo-specialists, "jewelers and opticians," "optical specialists," "doctors of optics," "optical companies" and quacks in general, whose chief equipment consists of gross pretensions, commercial aggressiveness and the ubiquitous sign:

EYES EXAMINED FREE,

whereby the gullible and credulous are led to believe that they will receive something for nothing, it is small wonder, indeed, that the anticipated results of

ocular treatment are so frequently unrealized and that the uninformed laity and indiscriminating physician often confuse the claims of the ophthalmologist with those of the pseudo-specialist.

It is gratifying to note, however, that during the past few years an increasing interest and broader knowledge has been exhibited by the medical profession in the diagnosis and rational treatment of eye-strain. Even in the conservative medical centers of Europe, as I recently learned in an extended tour of her hospitals, considerable importance is being attached to ocular defects in the production of neuro-gastric disorders, but there is plenty of room for improvement in this direction, both at home and abroad.

AN ANALYTIC STUDY OF UREMIA.

WITH SOME GENERAL CONCLUSIONS IN REGARD TO ITS CAUSES AND TREATMENT.*

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CRITIQUE OF CURRENT UREMIA THEORIES.

If the current belief that uremia is due to renal inadequacy and the retention in the blood and tissues of excrementitious urinary bodies were correct, then (1) complete anuria should always produce uremia, and (2) the blood of uremic patients should always show an increase and the urine a corresponding decrease of urinary substances.

Anuria Without Uremia.—Many cases of complete anuria, due to various causes, are, however, recorded, in some instances persisting for several weeks, in which none of the characteristic phenomena of uremia developed. Most of these patients for days complained of nothing more than a feeling of extreme lassitude, death resulting suddenly from heart failure without preceding coma or amaurosis, and without eclamptic spasms or typical changes of the blood pressure. On the other hand, uremia not infrequently occurs when the flow of urine is abundant and the excretion of urinary solids and water does not appreciably deviate from the normal.

Similar results are seen in animals after experimental nephrectomy or occlusion of both ureters. The animals die, but not uremic.

Uremia and Urinemia.—Death produced in animals by the injection of urine also produces a syndrome that differs materially from the uremic symptom complex. One must, therefore, distinguish clinically between uremia and urinemia. In uremia we often, but not invariably witness many of the signs of urinemia, but also many other signs besides. These other signs are the most constant and the most characteristic symptoms of uremia and the fact that they do not occur in urinemia forces one to the conclusion that they must be produced by other factors than simple urine poisoning. This view is borne out by a study of the solids of the blood and urine in uremia.

1. *The Nitrogenous Constituents.*—With the recent refinement of chemical technic these bodies (comprising chiefly urea, uric acid and its chemical congeners, the purin bases, ammonia salts, amido acids, creatin and others) have been diligently studied. The accumulated material is enormous, but disorderly. A careful sifting of the analytic data leads to a negation of the retention theory of uremia.

* This is a preliminary synopsis, full analytic data and clinical protocols will be published later.

For we see (1) as many cases of uremia develop without as with an abnormal accumulation of nitrogenous constituents in the blood; (2) we find uremia when the nitrogenous waste products of the blood are below the normal average, and (3) we inversely often fail to witness the appearance of uremia when the accumulation of these products in the blood is high above normal.

Similar conditions are determinable in the urine. Uremia may develop when (1) the patient is in nitrogen equilibrium (i. e., when the nitrogen output corresponds to the nitrogen intake) or (2) when the nitrogen output is far above normal, or (3) when the nitrogen output is below the normal average as compared to the intake; but it does not appear from a careful sifting of the analytic data published (including also some unpublished studies of my own) that uremia is more common when the nitrogen output is reduced below normal than when it is normal or even increased. Finally, many cases of true nitrogen retention are on record in which uremia failed to occur.

A quantitative study of the circulating and excrementitious urinary nitrogen bodies, therefore, teaches practically nothing of the causes of uremia. Only when we determine separately the various groups of nitrogen bodies that occur in the blood and the urine do we encounter certain deviations from the normal that may be considered fairly characteristic. I refer chiefly to two findings, viz.: (1) A relative increase of the ammonia salts, both of the blood and the urine as compared to normal average values, and as compared to the circulating and excrementitious urea. (2) A relative decrease of the urea both of the blood and urine as compared to average normal values and to the total nitrogen contained in circulating and excrementitious nitrogenous waste products.

These two findings I consider to be of the greatest importance, as I will have occasion to show.

It is well to bear in mind that there may be present in minute quantities among the nitrogen bodies of the blood and urine certain highly toxic albuminoid or alkaloid bodies that might produce an enormous effect without appreciably changing the nitrogen content of the blood and urine nor the relative proportion of the better known nitrogenous constituents to one another. This point also will be referred to again.

II. *The Inorganic Saline Constituents.* In the case of the salts of the blood and the urine our data are for the most part obtained from physical methods of examination, viz., cryoscopy and electric conductivity tests, determinations that are far more accurate than chemical methods of analysis. The electric conductivity in particular is a function of the salt content (molecular concentration) alone of the blood and urine and is altogether independent of the nitrogenous bodies that may be circulating at the same time; hence this latter method gives us especially valuable information in regard to the salt economy of the body in uremia. The slight inaccuracies inherent even in these physical methods are, I believe, overbalanced by the mass of figures collected in each case, for the technic when once properly acquired is so simple and rapid of execution that the operator is enabled to perform a great many determinations in the same patient within a short time.

The collected data show a marked degree of uniformity and indicate clearly two things: (1) That uremia often occurs when the salt values in the blood are normal or under normal; (2) that uremia need not occur even when the increase of salts in the blood and the corresponding decrease in the urine are very marked.

It appears, in other words, that increased molecular concentration of the blood is by no means a constant phenomenon in uremia. (This refutes Lindemann's seductive "physical" theory of uremia.) There is, in fact, less numerical evidence to show that uremia is due to salt retention than to the retention of nitrogenous waste products. One might argue again as in the case of the latter that certain salts were exquisitely toxic and were present in such minute quantities that they might poison the patient without appreciably altering the molecular concentration of the blood; this postulate can not be absolutely negated by the evidence at hand; but if such salts play a commanding rôle in uremia (one might think for instance of cyanids) then they are not identical with any of the salts that normally appear in the urine, for none of the latter can exercise a pronounced toxic effect in quantities that would be so small as to escape detection by the chemical methods that are being employed to look for them.

This applies in particular to the potassium salts of the urine that have so often been accused of producing uremia. True, these salts are poisonous and they undoubtedly play an important part in urinemia (presumably causing death in this affliction by their action on the heart), but they can not be incriminated with causing the characteristic symptoms of uremia. Thus we are forced to the conclusion that neither the retention of the nitrogenous bodies nor of the salts that normally occur in the urine generally produces uremia. So much for the negative side of the argument.

The questions now arise: (1) What is the character and the origin of the poisons that can be accused of producing uremia? (2) What function or functions must become perverted; what organs (if not the kidneys) must become diseased in order that these poisons may be formed? For poisons there must be, as no one will gainsay.

THE CAUSES OF UREMIA.

It is well known that in uremia the urea secretion is often decreased; this fact is commonly interpreted to signify that the elimination of this body has become deficient, in other words, that it is being retained. If its elimination, however, were merely interfered with, it should always be correspondingly increased in the blood, and this is not the case.

I am inclined to the belief that the decreased urea secretion is not so much due to retention as to non-formation. This postulate is borne out by the finding referred to in a preceding paragraph, viz.: That with a decrease of the urea in the urine and blood, we very often find an increase of the ammonia. This points to the liver as the affected organ, for the bulk of the circulating ammonia is normally converted into urea in the liver.

There is other chemical evidence that points to the liver as the deranged organ. I refer, e. g., to the occasional increase of the purin base excretion relative to the uric acid excretion (the purin bases forming a precursor of uric acid that should normally be converted into the latter in the liver), and the increase of uric acid relative to urea (the former being a precursor of the latter, the conversion also occurring in the liver), the appearance of various amido-acid compounds, and compound glycuronates; of members of the acetone body group in uremia. The reduced tolerance for carbohydrates with alimentary glycosuria that I have noticed in several cases of chronic uremia also seems to point to some liver derangement.

All this chemical evidence that supports the idea of an hepatic instead of a renal origin of uremia is somewhat scanty; and our knowledge of the normal intermediary metabolism within the liver is at best still so deficient that we must approach the interpretation of its perversions with much trepidation. The clinical evidence, however, and what may be called the pathogenetic evidence in favor of the hepatic origin of many cases of uremia is in my judgment very strong and very convincing. For we know clinically that disturbances of the liver and of the kidneys often go hand in hand and I believe we would find this to be still more frequently the case if the liver were examined with the same degree of thoroughness as the kidneys in each case of nephritis or of Bright's disease that came to autopsy; and if mild, functional disorders of the liver were as easily recognizable as those produced by renal disorders of like severity.

That the liver, i. e., hepatic insufficiency, plays an important rôle in the pathogenesis of Bright's disease is acknowledged¹; that, further, the same infectious or toxic agencies that produce nephritis also affect the liver in most cases is clear. For to the liver and to the kidneys jointly is relegated the rôle of ridding the organism of toxins, the former eliminating them into the bowel by the bile, the latter into the bladder by the urine. Toxins, moreover, of gastroenteric origin (and they constitute the most important of the exogenous poisons) must pass through the liver to reach the general circulation and the kidneys beyond. The same applies to poisons derived from metabolic perversions (endogenous poisons) for they are either for the most part generated in the liver or are carried to it before they reach the kidneys.

Assuming now for the sake of argument that the kidneys were always primarily affected in cases that ultimately determine towards uremia, so that their power of eliminating circulating toxins were diminished, then the bulk of the disintoxicating function would be thrown on the liver and in time hepatic insufficiency, i. e., overtaxation, fatigue of the liver cells, would develop. The character of the hepatic lesion would depend in each case on the virulence of the circulating toxins and the length of time during which they circulated.

There is still another idea that must be considered in this connection. It may be called the defective filter idea. It is a well-known fact that nephritic kidneys, while on the one hand less permeable than normal kidneys for a variety of circulating bodies, are on the other hand more permeable than normal for certain other bodies. In the case of the kidneys we are dealing with a living selective filter that is intended to hold back certain substances that are still useful to the organism and to allow the passage of other compounds that have outlived their usefulness, that possess no more potential energy, that are completely oxidized, dead, mere ash, and that should be gotten rid of as soon as possible.

When the kidneys are diseased, the filter leaks. Useful bodies that could still furnish a modicum of heat and energy are wasted. Among such bodies many are nitrogenous derivatives of albumin, as ammonia salts, amido acids, etc., bodies that should normally have been carried back to the liver for further elaboration to terminal products, like urea, uric acid, etc. When

such suboxidized nitrogenous compounds are wasted, the body, we must assume, is forced to disintegrate more of albuminous pabulum in order to make up the deficit and the bulk of this additional labor is thrown on the liver. In time, this organ must consequently become fatigued, so that the further disintegration of albumins either proceeds more slowly than normal or becomes altogether perverted into abnormal channels.

When this happens less urea and more incompletely disassimilated intermediary products of albuminous metabolism are thrown into the circulation; or bodies are split off from the albumin molecules that are completely foreign to normal blood and that are poisonous. Substances of this character are actually found in the blood of uremic cases.

One may further say axiomatically that the less complete the disassimilation of the albumins the more do the generated products maintain an albuminoid character (ptoma peptones, peptotoxins), and the more intensely toxic do they become.

That the general metabolism of albumins in particular is perverted in uremia is further manifested by the frequent appearance of acidosis in terminal uremia and even in pre-uremic states; in fact, an acid intoxication must be incriminated with producing many of the fulminating signs of uremic, as well as of diabetic, coma. The increased excretion of ammonia that has been referred to may even be explained on the basis of a chronic acidosis, for we know that ammonia is thrown into the circulation whenever abnormal amounts of acids begin to circulate. This is a protective process, inasmuch as it aids in maintaining the normal alkalinity of the body fluids and the alkali content of the cells. The ammonia salts formed in this way are sacrificed at the cost of urea; hence more ammonia and less urea in the blood and urine of uremic cases. As urea is the most potent physiologic diuretic the flow of urine is at the same time often reduced.

This in rough outline is the pathogenesis of a self-intoxication, either acute or chronic, that may follow or accompany chronic renal disease, and still be due more to hepatic than to renal inadequacy. That the liver is not the only organ affected is probable, and uremia interpreted as above is not so much the result of hepatic derangement alone as of a general metabolic disturbance. This view is forcibly borne out by the observation of an occasional case of uremia, in which the kidneys are found practically normal after death, and in which essentially no evidence of renal disease or even of functional inadequacy on the part of the kidneys presented itself during the life of the patient.

The manifold factors that may precipitate an acute attack of uremia in an individual suffering from hepatic insufficiency (pre-uremia) need not be enumerated in detail; the determining insult may be severe as, for instance, some virulent infection or intoxication (chloroform anesthesia) suddenly throwing a mass of work on the liver or causing degeneration of its cells, or it may be apparently insignificant, and consist of nothing more than an attack of gastric or enteric indigestion or merely some psychic or emotional shock that acutely deranges the liver function.

How important it is, therefore, to be able to recognize early, even mild degrees of hepatic insufficiency (particularly in renal cases and in pregnant women), and to safeguard the patient against all the agencies that might suddenly throw a strain on the fatigued liver!

1. Croftan: "The Circumstances and Treatment of Bright's Disease." THE JOURNAL A. M. A., June 24, 1905.

GENERAL PRINCIPLES GOVERNING THE TREATMENT OF UREMIA.

We are wont to treat uremia by stimulating the flow of urine, by purging and by sweating, with the intention of forcing the kidneys to resume their work, and of ridding the body of the urinary bodies that we imagine to be circulating in excess. In addition, we attempt to regulate the diet in such a way that there shall accumulate in the blood the smallest possible amount of residual nitrogenous bodies ("Harnschlacken"). If, now, uremia is not due to the circulation in excess of such bodies, the above therapy is wrongly directed.

It is questionable, moreover, whether any of the above measures can at best do more than rid the body of water, some sodium chlorid and possibly a little urea; the loss of water one might imagine to do more harm than good, for it should promote the concentration of the poisoned body fluids, and hence render them more toxic. There is surely no exact evidence to show that the sweat or the urine of uremic cases after the use of diaphoretics and diuretics becomes more toxic than before; nor has any one ever succeeded in demonstrating in such sweat or urine any of the albuminoid or alkaloid bodies that must be accused of causing the most fulminating symptoms of uremia.

The chief object of treatment should be to prevent the development of acute uremia by giving attention to those organs and functions that threaten to fail. In order to do this intelligently, *the renal idea should be relegated to the background*, and more attention should be bestowed in pre-uremic states on the liver and the general metabolism. The liver, above all, should have a rest. To stimulate the liver in chronic uremia is bad practice. The same principles should obtain here as in the treatment of a fatigued heart or a fatigued stomach or a fatigued nervous system.

First rest, then graduated exercise until the normal tone is regained. Stimulation should be reserved for emergencies and as a last resort. To accomplish this end all articles should at first be eliminated from the diet that can irritate the liver or stimulate it to increased functional activity; every effort should be made to reduce intestinal putrefaction to a minimum, for intestinal toxins as they reach the liver severely strain the disintoxicating function of the organ. No cholagogues should be administered.

After a period of rest—and starvation for a few days seems a rational plan—the liver may be gently stimulated in the hope that it may be coaxed gradually to resume its functions. This should be attempted by the carefully graded administration of salicylates, of bile acids, possibly of calomel; in addition, various dietetic and physical means may be adopted that we know to be capable of stimulating the various functions of the liver. All this treatment should be carried on with careful supervision and daily control of the effect of these measures on the functional activities of the liver as evidenced by the composition of the stools and the urine and the general condition of the patient. The details of the various means mentioned and of the symptomatic evidences of the state of the liver function can not be discussed within the narrow frame of this article. I content myself with establishing general principles.

The treatment of the acute uremic attack is always an ungrateful task; for it is immaterial whether we are dealing with a disorder that is primarily or in its ultimate consequences due to renal or hepatic or general

metabolic insufficiency, in any case we are dealing with a terminal syndrome, that is due to the crumbling of the whole cellular edifice. To arrest this collapse, essentially means to revive a dying organism. That this may occasionally be done for the time being can not be denied; and as the recuperative powers of the human body border on the phenomenal no effort should be spared to bring an acutely uremic patient back to life.

Of sweating, purging and the stimulation of the kidneys I have already spoken. The administration of liver stimulants by mouth seems hardly feasible, and I have never seen any good effects in uremia that I could fairly attribute to this practice. The most sensible procedure is blood-letting. This useful measure constitutes a lost art nowadays, and it is time that it should be revived; we are dealing in uremia with a toxemia (for no organic lesions that could explain uremia are discovered after death), and to eliminate some of the poisons that are present in the blood by bleeding is always indicated; whether the injection of a saline to replace the lost fluid is useful or necessary is not yet established; it can certainly do no harm, particularly if some saline is injected that can stimulate the hepatic function, e. g., salicylate of soda in normal salt, or a solution of sodium citrate or phosphate in appropriate molecular concentration. Symptomatically, the use of such infusions has been useful in my hands, and has occasionally caused the most alarming symptoms of an uremic attack to disappear when other measures, I think, would have failed.

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A CASE OF TRAUMATIC ANEURISM OF THE RIGHT RENAL ARTERY, WITH A REVIEW OF THE LITERATURE.

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PHILADELPHIA.

Because of its rarity and the few cases that have been successfully operated on, I report this case of traumatic aneurism of the right renal artery that occurred during my service as house surgeon at the German Hospital. This patient was operated on by Dr. John B. Deaver, with whose permission I present the report:

History.—J. D., male, aged 26, single, machinist, was admitted to the surgical wards of the German Hospital March 17, 1905. There is no hemophilic, tubercular, neoplastic, cardiac or nephritic history in the family. The man smokes constantly and has used alcoholic beverages freely until lately. He denies venereal history. His appetite has been variable; bowels have usually been regular.

Present Illness.—When 12 years of age he was thrown from a horse and landed heavily on the ground, the brunt of the blow being received in the right lumbar region. The patient went to bed on account of shock, and complained of sharp, cutting pain in the right lumbar region, followed by hematuria. There was no vomiting. After being confined to bed for about twenty-four hours, the patient got up and went about his business. About a month after the accident he was seized again with a sharp, cutting pain in the right lumbar region, which lasted about two hours. He has had similar attacks of pain, usually with intervals of five days; often six months would elapse without any symptoms whatsoever. Each attack was severer than its predecessor.