

10th.—Awoke but twice during the night ; pulse, 86 ; temperature, 98·4° ; bowels freed by means of an enema. From this date there was nothing to record. She gained strength rapidly.

Although these two cases had the same favourable termination, they presented features very markedly different. In the first the tumour was not only of the simplest kind, but was free from adhesions, and was removed without the escape of one drop of fluid into the abdomen, the unpleasant symptoms subsequently occurring being apparently altogether due to the frequent use of the catheter. In the second case the dense adhesions which existed anteriorly rendered the removal of the cyst by the ordinary method impossible, and it was only by inverting the sac, and breaking the adhesions down from behind, that this was finally accomplished. In consequence of the rupture of the cyst the pelvis was filled with the fluid it had contained, and all this had to be removed by sponging, a process which occupied a long time ; but, notwithstanding these adverse circumstances, the patient made an excellent and rapid recovery.

In neither of these cases was any drug whatever administered, nor was any stimulant allowed ; but, on the other hand, the greatest care was taken with regard to diet, ice alone being allowed for the first few hours, and subsequently beef-tea and milk in very small quantities and at stated intervals. To this strict regimen, I believe, much of the favourable issue of these two cases was due. The greatest care was also taken to insure the best possible sanitary conditions, and no person was allowed to enter the ward subsequent to the operation, except the nurse, who had charge of the case, and two pupils, who, *not resident* in the hospital, gave their whole time for the first few days to watch the patients. To these gentlemen—Mr. Nelis and Mr. Meredith—I am much indebted for their constant care and attention to these patients.

ART. VI.—*Uræmia in Affections of the Liver.* By W. WHITLA, late Senior Resident Surgeon and Superintendent, Belfast Royal Hospital.*

AT the last meeting of the Ulster Medical Society we enjoyed the privilege of listening to the President's most interesting and

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valuable series of cases of puerperal eclampsia. The paper, which was an essentially practical one, was all the more valuable because the author chiefly confined himself to his most successful treatment of this very formidable and too often fatal affection. If I err not, he stated at the beginning of his remarks that it was not his intention to enter into the pathology of the disease. The few remarks in this paper on "uræmia" are merely intended as a preface to one on Puerperal Eclampsia.

With the symptoms of this poison (uræmia), and its most frequent cause, Bright's disease, it is wholly unnecessary to take up the time of a Society like this, where every one is so thoroughly acquainted with them, and it is merely my intention to show that the different forms of Bright's disease, or desquamative nephritis, are not the *sole* causes of this affection. Excluding diseases of the chest, there is no class of cases more common in the medical wards of an hospital than diseases of the kidneys; and perhaps next to these would come the various affections of the liver, especially cirrhosis and cancer. During the early part of last year there were in hospital several patients dying from Bright's disease, and at the same time there happened to be an unusual number of structural liver cases in the wards; and I was forcibly struck with the similarity of the symptoms of these two very different and opposite affections.

By closely watching the progress of a few patients in the advanced stage of kidney disease one becomes familiar with nearly all the phases of chronic uræmia, but here a condition presented itself identical in all respects with this, yet arising during the progress of an essentially different disease. At first I was led to believe that in these cases the kidneys were diseased in addition to the liver, but in some instances where I had the opportunity of making *post mortem* examinations I found these organs healthy and free from any organic change. In May, 1874, Dr. Murchison's Croonian Lectures on the functional affections of the liver were published in the *Lancet*, and in one of these valuable productions (which I regret being unable to lay hands on at present) he clearly points out a most important function of the liver.

Dr. Murchison, supported by high Continental authority, states that one of the chief duties of the liver is to lay hold of the effete albuminous compounds, the products of wear and waste in the blood, and to reduce these to their most soluble form, urea, in order to present them to the kidney in a condition capable of being

easily and rapidly eliminated. According to these authorities, one may say, if I understand them aright, that it is the liver which *manufactures* the urea, the office of the kidney being merely to throw it out after its elaboration; consequently, when this function of the liver is deranged, these products are not reduced to urea, but the process stops short, and intermediate compounds—tyrosin and leucin—are formed, substances which the kidney cannot so easily eliminate, and which consequently accumulate in the blood and give rise to the same symptoms as urea, from which, in chemical composition and physiological effect, they differ little, and may be practically regarded as modifications of this substance possessing lesser solubility.

When I use the words “uræmic poisoning,” I wish it then to be understood that I refer to two similar conditions produced by two very different causes—in one the diseased kidney refuses to throw out the already manufactured urea, in the other the healthy kidney is unable to excrete the deleterious products of disintegrating albumen less oxidised than urea. The present state of our pathological knowledge does not enable us in all cases to discriminate clinically the causes at work in producing these identical conditions, especially as we have reason to believe that both factors are operating in the majority of cases of uræmia.

Cirrhosis, by destroying the secreting texture of the liver, produces this derangement of its function, and the consequent retention of these substances gives rise to symptoms of uræmic poisoning. The convulsions and other symptoms met with in the rare disease, acute yellow atrophy of the liver, Dr. Murchison looks upon as owing to these poisons; and, if I mistake not, he goes so far as to say that this form of poisoning is not the uncommon wind-up of the long train of symptoms met with in cases of advanced structural disease of the liver. He believes in this way, too, is accounted for also the typhoid condition which characterises many of the profound lesions of the system. An excellent example of this typhoid condition, associated with symptoms of uræmia, presented itself in the case of Mary B., which I had the honour of bringing before the Society in April, 1874. In this case no trace of disease could be detected anywhere; her delirium was considered to be owing to typhoid fever; the patient died after a few days illness, evidently from some mysterious blood poison; and on opening the abdomen four-fifths of the liver was converted into a bag of pus, though not a single symptom pointed to this organ during life.

Of a considerable number of cases of cirrhosis of the liver, and a few of cancer of this organ which I watched in hospital and kept under observation after leaving it, in the majority death from uræmia occurred. Though these facts are very interesting from a pathological point of view, they are not of much practical importance if only occurring in the last stage of a disease, nor of much diagnostic value, since the affection is obvious before their development. But I am satisfied that uræmic poisoning may be the *first* symptom of structural disease of the liver, and it seems possible that it may arise also from a purely functional affection of this organ; and I hope to lay this before the Society in my next paper as an explanation of the pathology of puerperal eclampsia in those cases characterised by absence of structural kidney disease.

When we know positively that advanced structural disease of the liver nearly constantly ends in uræmic death (provided the patient be not cut off by some complication), and when we, over and over again, find in the bodies of persons dying from other causes livers largely diseased and extensively destroyed, without a single symptom pointing to derangement of this organ during life, it is easy to see of what vast practical importance this cause of uræmia becomes.

Long ago every obscure disease characterised by the presence of coma and convulsions was put down to apoplexy, but in later days, since the researches of Dr. Bright, it seems the fashion to class them as diseases of the kidney; it is not a very rare thing to hear of sudden deaths preceded by coma and convulsions, especially in children, where the *post mortem* examination reveals no traces of disease in either brain or kidneys; and we find it sometimes noted as a matter of little moment that the liver is found much enlarged.

In my own short experience of nearly two hundred autopsies during the last two years, I can recall but a few cases where this *now* seems to me the most satisfactory explanation, though at the time I could offer none. I will refer to my notes of one case. In January, 1874, a man, aged thirty-nine years, was admitted to the Belfast Royal Hospital suffering apparently from bronchial affection. He had been ill about a week. On examining his chest no evidence of disease was detected, though he suffered from considerable dyspnoea, and had a harsh barking cough. His body was evidently well nourished. Owing to the absence of physical signs, acute tuberculosis was suspected, and merely expectant treatment adopted. He remained in the same condition till about the tenth

day after admission, when sudden coma set in, with convulsions, from which he recovered under purgatives and the use of the blanket-bath—only, however, to be attacked the next day, when he died, after being nine hours comatose, with frequent convulsions. During life his urine was examined, but with negative results; unfortunately, tyrosin and leucin were not tested for. Careful examination showed complete absence of either casts or albumen.

Autopsy twelve hours after death.—*Lungs* healthy; nothing in them to explain the cause of the dyspnœa. *Brain* healthy. *Kidneys*—weight, normal; appearance, healthy; consistence, a little firmer than natural—left slightly lobulated. *Liver* fatty; weight, nearly seven pounds; under the microscope, cells filled with oil.

I could form no idea of the cause of death after examining every organ in the body; it seemed probably renal, till the kidneys were carefully examined under the microscope, but no disease was found in them. This case, read in the light of more recent pathology, is, perhaps, capable of explanation.

Looking at urea or its modifications as toxic poisons, and keeping in mind some of the most important functions of the liver, we are able to afford an explanation of many things very difficult to otherwise understand. Like many other poisons, after a time the system becomes very tolerant of its action, and the nerve centres, accustomed to the gradually-vitiated blood, are in a condition ready, as it were, to rebel at a moment's notice on the least additional increase of poisoned pabulum; when matters are thus evenly poised, suppose any trivial cause interferes with the function of the liver, the last straw is laid on, and the result is—a *convulsion*. On the other hand, we see patients smitten down and prostrated with this poison, and watch the progress of Death as he comes slowly and steadily on grasping his victim, who succumbs quietly, and passes away without one struggle. Is it not possible that *two* organs are at fault in the first case, and only one in the latter? Indeed, it may even come to be a disputed question whether the kidney alone, by its disease, is capable of producing death from uræmia.

Shortly after the first dawn of light was shed upon the pathology of kidney diseases, to find albumen and casts in the urine of a patient was to give him a very limited term of existence; now we know these are not incompatible with a lengthened and enjoyable period of life. We put under the microscope two samples of urine

from different patients the subjects of this disease; we see in one here and there a few small scattered casts, and these, too, found with difficulty; yet this patient may have but a few weeks to live, while in the other we find the field studded over with casts of such magnitude as to prove they are the models of tubes devoid of all epithelial or excreting tissue, and full of oil and fat cells, evidently from a gland whose structure is apparently almost destroyed; nevertheless, its owner may live and enjoy life for a good long time till perhaps cut off by some unexpected complication; we have no more reason to wonder at this than at the case of a patient who dies from a slight contraction of the mitral valve, while his neighbour lives with *his* mitral no longer a valve but a mere slit, till old age carries him home. Yet these admit of an explanation if we knew it; and is it not possible that the life of a diseased kidney is depending upon its liver? If so, a very material point is gained in prognosis and treatment.

A word about the uræmia of scarlatina. My experience is far too limited to generalise, but perhaps I shall be borne out in stating that convulsions occur more frequently in this form of desquamative nephritis than in the acute affection, the result of exposure and other causes. Dr. Samuel Fenwick and others have found in the stomach tubes and the Lieberkühnian follicles of the intestines in the bodies of patients killed by scarlatina, changes which prove that a process takes place in the epithelial lining of these glands analogous to the desquamation occurring on the surface of the body, while a very eminent authority affirms that this is the true pathological explanation of the lesion of the kidneys which, he says, is caused by the shedding of the epithelium lining in scarlatina, the convoluted tubes of these organs.

But I can go one step further, as I hope, from changes which I have noticed in scarlatinal blood, to demonstrate under the microscope, before the conclusion of this session, that the epithelial lining of the blood vessels of the body suffers the same destruction as the cuticle, and that partially broken-up cells and nuclei corresponding to the pavement on the fenestrated coat of Henle are sometimes to be found in the circulating fluid during the early desquamative stage of scarlatina. Many things go to show that at this stage of the disease the blood is charged to excess with excrementitious matters, consequently great extra work is required of the liver to elaborate urea from these, which work the liver does, though more quickly and less perfectly than it should, and the resulting

modifications or substitutes for urea cannot be thrown out sufficiently rapidly by the kidneys, already overtaxed and unhealthy, and uræmia results. If, then, this state of matters exists, we see what a very important part the liver plays in the uræmia of scarlatina.

The hypothesis of a condition somewhat like this will go far to explain many cases of puerperal eclampsia characterised by the absence of structural kidney affection. Supported by the researches of Murchison and others, whose labours have thrown considerable light upon the functions of the liver, one may venture to hope, as these functions are better understood and more generally known, that many of the pathological difficulties surrounding diseases of the kidneys will be cleared away.

That cases of disease in these organs are turning up now and then which baffle every scientific explanation the practice of most men will confirm; and it is not unlawful, having tried in vain to elucidate them by the light of all that is now known of renal pathology, to turn to the investigation of the conditions of other organs and there seek for hidden associations which may assist us. It was my intention to read the notes of a few cases of this kind which have given me some trouble, but I have already trespassed too much upon my reader's time, and will conclude by briefly referring to one. Nearly three years ago a physician in the country sent me a specimen of urine from a patient who had slight anasarca, and requested me to make a careful examination of it. Its specific gravity was 1012. On the addition of nitric acid it was found to contain nearly half the depth of the test tube of albumen; but, notwithstanding the most careful investigation, no traces of casts could be detected; his urine continued highly albuminous for some months, when I saw him and found him a man of fifty-five years of age, in excellent health and spirits, and capable of his usual amount of work—in fact, quite as well as he had been for years, excepting a trouble which I shall mention presently. There was no anasarca, heart, or liver affection. A sample of his urine has been sent me nearly every month since then—sometimes more frequently; it has always been highly albuminous, oftentimes alarmingly so; much time has been spent in examining it microscopically, but only once were anything like casts found in it; it has been examined repeatedly by other microscopists with the same result. This case seemed worth recording owing to the remarkable absence of *microscopic* evidence of structural disease; the urine of a healthy patient examined so often and

searchingly as his would hardly fail to show some well-formed casts occasionally. Last Christmas morning I received a sample of his urine, which contained one-third albumen; next day I received a letter stating he had been out shooting, exposed to frost and snow. Crystals of tyrosin have been found occasionally, and once little masses of leucin, in his urine, and, strange, the only trouble he ever experiences is owing to violent and excruciating attacks of hepatic colic, which reduce him very low indeed for days. Last time I examined him the extent of hepatic dulness in the mammary, axillary, and scapular lines was normal, and neither atrophy nor enlargement of the liver could be detected.

The supposition of a waxy kidney will not satisfactorily account for this state of matters, the only evidence of disease of this organ being found in the quantity of albumen passed daily; and it is worth while remembering the fact that if a healthy man fasts for twenty-four hours and then eats a quantity of highly albuminous food, such as eggs, large quantities of albumen appear in his urine; as the liver, having too much work to do, permits some of this substance to pass through in a condition unfit for supplying the wants of the blood. When blood so charged with this crude albumen reaches the kidneys it is at once purified by these emunctories. Is it going too far to suppose that some structural or functional derangement of the liver might convert this transient occurrence into a permanent symptom? If not, albuminuria with uræmia might be accounted for in many cases of puerperal eclampsia where careful microscopic examination reveals no casts.

But in a subject like this there is such scope for theorising that it is well to keep in mind the danger of passing through the region of the probable into the domain of impossibility. We arrive at much more accurate conclusions about the function of the liver from closely and narrowly watching the symptoms in cases where this organ is very extensively diseased than by any series of experiments on the lower animals, where many unfavourable conditions are necessarily induced, and the physiologist will soon learn that from clinical medicine alone must he seek for further light upon this subject. The facts stated upon the high authority of Dr. Murchison and others show beyond doubt that the liver is intimately associated with the elaboration of urea; and if these few remarks ever lead anyone to turn to the *liver* for an explanation of the many obscure cases of uræmia, its object will be more than fulfilled.

About the *diagnosis* of uræmia from brain disease, apoplexy, alcoholic poisoning, &c., considerable difficulty is sometimes met with, especially in those cases where a sudden attack is experienced for the first time, and where no history of any renal trouble can be found. In such cases great assistance will be had from careful examination of the condition of the heart, as nearly always distinctive modifications of the heart sounds will be heard, as reduplication of one or both, intensity of second sound, &c., differences also in the arterial tension and cardiac impulse. Of these none seem so constant or remarkable as muffling of the first sound. In a very interesting case of uræmia, which I saw with Dr. Bell, at Bangor, in June, 1875, in a lady aged forty-six years, the first sound was *entirely absent* for four days after the cessation of the convulsions, returning suddenly after a strong purgative; it seemed as if due to the resistance offered by the swollen artery to the entrance of blood during the contraction of the left ventricle, so that no wave of sufficient strength to beat against the wall of the artery occurred.

ART. VII.—*Questions in Ophthalmic Surgery.* By H. MAC-NAUGHTON JONES, M.D., M.Ch., L. & F.R.C.S., Irel. and Edin.; Surgeon, Cork Ophthalmic and Aural Hospital, &c.

I.—ANÆSTHESIA.

II.—ENUCLEATION AFTER INJURY.

I.—ANÆSTHESIA.

It must ever be a matter of considerable moment to the operator in conducting any eye operation, whether, in the first place, he shall use any anæsthetic at all; and secondly, if he does do so, what the nature of that anæsthetic shall be. The number of operations daily performed in any large ophthalmic hospital, and weekly in a small one, makes the question of the use of anæsthetics a very serious one to the ophthalmic surgeon. The responsibility involved in their administration in an hospital, such as Moorfields, where there is a daily average of at least six or eight operations, is, no doubt, a serious one. But it is much more so in a smaller hospital, or in private practice, when, especially if an experienced anæsthetiser cannot be had to administer the anæsthetic, the operator has to trust to inexperienced hands, and the anxiety is not relieved, and the responsibility divided, by a large staff, who surround and assist