Discussion

The radiologist is not always asked to examine patients suspected of appendicitis. This is in part due to the fact that the radiology of the appendix has been up to now suspect. One of the reasons for this is that in the past we have failed to fill the normal appendix owing to defective technique, and there has been a tendency to rely on indirect signs such as pyloric spasm, abnormal filling of the caecum, and localized tenderness, etc. These indirect signs are frequently misleading. An unfilled appendix should only be diagnosed as abnormal if it fails to fill after repeated examination.

The normal appendix gives characteristic appearances in the radiogram, and, *per contra*, the abnormal appendix can also readily be detected, as a rule, from alteration in its contour, emptying rate, position, and other changes. Adhesions are revealed by fixation of the appendix in an abnormal position in relation to the caecum, or by the retention of unusual curves or kinks in the serial radiograms. It must be remembered, however, that in many patients without signs of disease there is diminution of the swaying or pendulum movements of the appendix. The presence of adhesions must therefore be diagnosed with caution, the more so since a short meso-appendix will limit the movements of the organ.

A large single faecalith, if it contains calcium, may be visible in a plain radiogram, and is usually seen close to the right iliac crest.⁴ But it is more often the case that the faecaliths when present are invisible, and are only detected as filling defects in the barium-filled appendix. Inconstant filling defects are often seen in normal cases, and are apparently due to accumulated secretion. The filling defects caused by faecaliths retain their contour and position in all the radiograms. The lumen is generally slightly enlarged where many faecaliths are present.

The most characteristic changes are seen when the appendix is the seat of subacute inflammation. There is obvious dilatation of the lumen, especially at the distal extremity, as in Case III; fixation is present in one position, and, owing to the diminution or absence of peristalsis, there is delayed emptying of the barium, the appendix being generally the last part of the bowel to empty. Localized tenderness is frequently present.

When the appendix lies behind the caecum it is concealed from view in the earlier radiograms. As the caecum begins to empty, however, it comes into view, and it is then sometimes possible to obtain films showing its shape and position. These cases are some of the most difficult from the point of view of the radiologist. The size of the lumen of the appendix appears to vary with the condition of the colon. When the colon is spastic the appendix tends to be contracted and the lumen is narrow. On the other hand, when the colon is found to be atonic, the atony appears to affect the appendix at the same time, and it fills with considerable ease and shows a large lumen. It will often fill in such a case under the pressure exerted by a barium enema. This, however, is not usual, since the appendix is normally only visible after the enema has been partially evacuated. Dilatation of the lumen of the appendix appears to be one of the most important radiological signs of disease, especially when it affects the distal extremity and is accompanied by retention of the barium.

Summary and Conclusions

1. It is suggested that the appendix, unless blocked by fibrosis or adhesions, will always be filled with barium if careful attention be paid to the radiological technique.

2. A careful study of the radiographic appearances of the filled appendix enables a reasonably accurate radiological diagnosis to be made in most cases.

3. The following radiological signs are suggestive of disease: (a) dilatation of the lumen, (b) fixation in one position, (c) delay in emptying, (d) clear-cut filling defects, and (e) non-filling after repeated examinations.

I should like to take this opportunity of acknowledging the assistance received from Mr. Rast and other colleagues at the German Hospital who have provided me with clinical material and operation results.

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SOME OBSERVATIONS ON SURGICAL
URAEMIA

BY

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The surgical type of uraemia may be defined as that state which supervenes when there is disease or functional derangement of the urinary tract and a failure to excrete toxins present in the blood and tissues of the patient. This implies, first, a derangement of the urinary mechanism, and secondly, an accumulation of toxins as the result. I shall therefore limit myself to a type of case in which there is obstruction to the urinary outflow, accumulation of toxins, and no known previous disease of the kidneys themselves.

In the cases seen in the surgical wards the condition as defined above arises from two causes: (1) mechanical obstruction, most frequently seen in the peripheral passages, as in prostatic cases, and (2) septic changes which involve the kidney itself and injure its function. The latter group of phenomena are often superimposed on the former and complicate the picture, so that for the purposes of this discussion I have concentrated on the former type in which uraemia, uncomplicated by sepsis, is seen in obstructed cases.

Pathology of Urinary Tract Obstruction

What are the toxins which accumulate in a case of urinary obstruction? From time to time someone claims to have isolated a body which is asserted to be the substance responsible for the uraemic state; so far, however, no such single specific substance has been discovered which stands the test of critical examination, and it is probably safe to assume that the phenomena are due to a general accumulation of products, which are normally rejected, but which clog and derange the working of the whole organism by their presence in excess. Among the products accumulating in this way, the ones which primarily focus our attention are due to protein metabolism, and are all of a nitrogenous character. We may regard the condition as due to the soaking of the various tissues of the body in waste metabolic and katabolic products, so that we ought to go a stage further than the examination of the blood, and determine, if possible, when uraemia develops in relation to the accumulation of waste products in the tissues. This is a difficult subject, and there are as yet no definite data available.

The following facts emerge: in the first place, cases with high blood nitrogen and no symptoms of uraemia are common; secondly, a persistent high blood nitrogen ultimately leads to uraemic manifestations; and thirdly, after the removal of the obstructing cause the blood nitrogen does not always show an immediate fall, but a

gradual one. There is therefore a latent period between the rise of nitrogenous products in the blood and the onset of uraemia, and also a period of high blood nitrogen after the removal of the causative obstruction, whilst the tissues are unloading their accumulations and the excretory organs are unable to carry these away fast enough.

With regard to the determination and recording of these cases I shall merely mention the methods of estimating renal function. The important factors are the general clinical condition of the patient, the concentrating power of the kidneys, the daily fluid output, and the level of urea in the blood. The last-named must be closely watched in the presence of known defect of function.

Post-mortem microscopical examinations of these cases reveal no appearances suggestive of a specific derangement of the uriniferous elements. The glomeruli and tubules retain their characteristics, and are only influenced by the general congestion or the presence of an inflammatory infiltration throughout the organ. They may be the subject of atrophic changes due to the presence of fibrotic elements after sepsis or to the pressure of a hydronephrotic sac. Even in the septic cases the changes are clearly those which are due to the presence of the septic process, and are not limited in any way to the essential portions of the urinary mechanism. There is nothing, therefore, in these cases, so far as the kidneys are concerned, to account for the state of uraemia; and when we come to contrast the clinical picture of the surgical with the medical type of uraemia we observe that we are dealing with a somewhat simpler picture.

Clinical Picture of Uraemia

The kidneys having been deranged there is a general cutting down of the excretion of the substances presented to them for elimination. These accumulate in the blood, and are insidiously soaked up by the tissues until a hypothetical point of tissue saturation is reached, when the symptoms of uraemia supervene. Such a state is seen in the many recorded instances of complete calculous anuria, the striking feature of which is their relative symptomlessness. The patient remains in a comparatively quiet state, is mentally alert, is nauseated, vomits from time to time, tends to be rather drowsy, and has a dull headache. There is little else for a week or ten days, and then the rapid onset of coma and death.

In the more chronic cases a typical uraemic patient, as we see him, is frequently a man of prostatic age with some urinary obstruction. He has been carrying on his usual life until increased frequency of micturition, dysuria, or an attack of retention drives him to seek relief. His unstable condition is perhaps immediately revealed, because the mere upset of admission to hospital or the use of a catheter may throw him into a state of mild mental confusion. It will be discovered that for a time he has been losing weight and strength, and has been growing restless in his habits. He has been irritable, off his food, and his mental alertness may have been noticeably impaired.

On examination he is seen to have a yellowish, unhealthy appearance; the pulse is elevated, 80 to 90; the tongue is dry and dirty; and the daily output of urine 20 to 30 ounces. The urea concentration is below 1 per cent., and the blood urea 90 to 100 mg. per cent. or more. Such is the common picture. Occurrences such as wild delirium, convulsions, deep coma, etc., are absent. Occasionally one meets with abnormal symptoms, such as in a case which occurs to me, in which an acute abdomen was simulated. There was, however, an advanced degree of sepsis in this case, and one suspects that the unusual happenings are often due to simultaneous lesions, among which sepsis is of the greatest importance.

The genito-urinary surgeon is familiar with many cases of uraemia which present some symptoms of the medical type, but they are in a minority. To take an example, I would suggest that elevation of blood pressure is evidence not of uraemia, but of the coexistence of a true nephritis in addition to the state arising from the urinary obstruction. There is no rise of blood pressure in cases of total nephrectomy in animals or in human beings.

It appears, therefore, that uraemic cases of the surgical type present a less complex picture than do those seen in cases of true intrinsic disease of the kidneys. There are fewer symptoms, and these are usually milder. We appear to be dealing with a complete or incomplete cutting off of the ability to excrete the poisonous metabolic substances in the blood. The "excretory tap" is turned off, as it were, absolutely or partially, permanently or temporarily.

Effect of Complete Anuria

Let us see what happens when the excretory flow is shut off absolutely. Double nephrectomy in animals has shown that the symptoms are not those of the active uraemic type as occurs in cases of intrinsic nephritis. There is no production of oedema, the metabolic products accumulate in the blood, and the animal dies frequently without any pronounced symptoms.

In this connexion the case has been described by Thompson in the *British Medical Journal*¹ of a young man who injured his kidney playing football. Haemorrhage could not be controlled, and a nephrectomy was performed. Complete anuria supervened, and it was found that he was a case of solitary kidney, which had been excised. He lived for ten days, during which he had no convulsions. His symptoms were a dull headache, a good deal of vomiting, fibrillary twitchings of the muscles, drowsiness, and a terminal coma during the last one or two days. During this period the blood showed the following changes:

	At commencement	Terminal
Non-protein nitrogen in mg. per 100 c.cm. whole blood	20	168
Urea	20	272
Ammonia	0.1	130
Chloride	450	351
Ca : P ratio	4	0.53
Alkali reserve in c.cm. CO ₂ bound by 100 c.cm. plasma	50-80	39
Blood pressure in mm. Hg	116/62	110/40 (day before death)

I will describe a parallel case in which the kidneys were destroyed by lithiasis until terminal uraemia resulted in death.

A young woman had had stones removed on three or four previous occasions from both kidneys, and when I saw her for the first time on the last day of her last illness both pelves and both ureters were again blocked almost solidly with calcareous material. The urinary output had fallen to a few ounces of low specific gravity fluid, which was little else but water. She was nauseated and vomiting from time to time. She was particularly clear mentally, and only distressed by the vomiting. Blood urea was well over 200 mg. per cent., and she died a few hours after I saw her and advised against operation.

These two cases are alike in that in both renal function was completely cut off: in the first by the removal of the remaining kidney, and in the second by stone. Both exhibited similar terminal symptoms.

The Everyday Case

Such are extreme cases, but the same appears to happen in our prostatic uraemic cases. There appears to be a "turning off of the excretory tap," irreversible in the two cases I have just mentioned, but as I shall now show reversible in many cases of lesser degree. The

¹ December 24th, 1932, p. 1134.

majority of mildly uraemic cases, such as one sees in prostatics, obstructions due to stricture, calculi, etc., only fail to make a good recovery in a minority of instances. The prognosis in these cases depends on the amount of real destruction of renal substance which has occurred, and not so much on the actual presence of uraemic symptoms.

I will give a short description of three cases which illustrate some of these points.

Case 1, a taxi-driver aged 54, was admitted under my care in 1923 with acute retention due to an impassable urethral stricture. There had been complete retention for thirty-six hours, and on admission his general condition was extremely bad. He was unable to answer questions, his tongue was dry and furred, and he was nearly comatose. As no catheter could be passed a suprapubic cystotomy was done, no attempt being made to deal with the stricture. After two or three weeks' severe illness he recovered completely, had his stricture excised, and is now as well as ever he was. He went back to his work as a taximan, and still comes to the hospital once or twice a year "to make sure that his stricture is not re-contracting."

Case 2 was that of a man of 62 with retention due to enlargement of the prostate. His general condition was poor, with a urea concentration below 2 per cent., and as he did not respond to catheter drainage he was opened suprapubically. For a short time he did well, but for financial reasons was anxious to leave the nursing home, and so passed out of my direct observation. He must have got into a condition of mild uraemia, because such reports as I had of him were that he would not eat, was irritable, and generally miserable. He gradually became worse, and after about two months was persuaded to come into hospital. It was then noticed that the daily output of urine was below 20 ounces, and the blood urea was found to be above 200. The hospital residents thought that he would die. The whole difficulty was that, although the suprapubic was working well, he was refusing to take sufficient fluid by the mouth. His error was pointed out to him, and after some persuasion an adequate diuresis was established, and he made a perfect recovery.

Case 3, occurring nine months ago, was that of a man with prostatic obstruction and renal insufficiency. A preliminary suprapubic was done, but the expected recovery did not take place, and he developed a persistent high pyrexia. Further investigation revealed a small bilateral hydronephrosis, which was infected. The pyrexia continued for nearly four months, in spite of a variety of treatments, and it was at last decided to drain one of the kidneys, as it was thought that the drainage by way of the ureters might not be sufficient. A right nephrostomy was done, from which urine appeared twenty-four hours later. The pyrexia disappeared, and after a few weeks' drainage I was able to deal with the prostate by endoscopic resection. Now both suprapubic and renal sinuses have closed, he feels well, and is back at light work.

Lastly, Professor Jean Brau of Montpellier, at a medical congress at Vittel in 1930, quoted a case of anuria which came on after the removal of a pelvic tumour. Three days later a nephrostomy was done on the left side, which acted successfully and saved the patient. The right kidney, however, did not recommence its activities for thirty-four days.

Object of Treatment

A perusal of these cases—which one could multiply indefinitely—three of which are from my own practice and one from the literature, demonstrates several interesting facts. We have seen that in the obstructed cases there is a "turning off of the excretory tap" by the kidneys, more or less complete according to the type of case. In mild established prostatic obstruction the decrease of renal activity is moderate, whilst in the anuric cases it is practically complete, and the picture compares with those of absolute destruction or ablation of the kidneys.

We have seen that on adequate measures being taken the renal apparatus returns to normal, and in many instances often appears to be as good as ever. One is led, in fact, to infer a functional retardation of excretion rather than actual damage as the first factor when the kidneys are placed in difficulties due to obstruction lower down in the urinary passages, the bulk of the renal substance remaining potentially normal. Actual destruction supervenes as time passes in accordance with the particular circumstances of the case.

To turn on the "excretory tap" we must first secure adequate drainage. In many cases an indwelling catheter for some days will suffice; if not, a suprapubic fistula must be provided. It is essential to have a perfectly free outflow, and often a catheter is not enough. In addition, the intake of fluid must be forced. I try to get my patients to drink two to three quarts of water, preferably of the diuretic type, during the twenty-four hours. I keep them on a light diet and give them alkalis, which I think helps to establish diuresis. Of these two essentials, the necessity of forcing the fluid intake was noted in the case of the man with the suprapubic (*Case 2*, above), who did not take sufficient fluid, so that his blood urea rose above 200 mg. and fell only when enough liquid was given. With regard to the other essential, if, for any reason, suprapubic drainage is insufficient, as it may be in hydronephrotic or infected cases, a nephrostomy may have to be done before the kidneys will respond, as I have shown in *Case 3*.

Discussion

So far, we have seen that the surgical type of uraemia appears to result from a functional retardation, and that it is comparatively easy to reverse this unless there is severe destruction of the renal substance. It will be useful, therefore, to consider briefly the possible causes which govern this inhibition of renal function with a view to possible improvements in treatment.

It is doubtful if the time-honoured theory of "back pressure" really gives us an adequate explanation, because in many cases the uretero-vesical valves are efficient, actual dilatation of the upper urinary passages is absent, and yet uraemia may occur without any evidence of urinary reflux. Back pressure probably only contributes its share in the later stages of gross alteration of the urinary mechanism. Again, the accumulation of toxins due to the stagnation and fermentation of the urine in the bladder, while probably playing a part in contributing to the general renal discomfort, cannot be regarded as a complete explanation. There is little evidence of the early absorption of toxins from the bladder; in many cases the renal pelves and ureters appear to remain intact, and yet uraemia occurs. Further, the highly specialized renal cells can remain in their functionless state for much longer periods than one would suppose probable if they were rendered inert by poisoning, and yet they recover immediately on the removal of the causative influence. One cannot put the effect of toxins on one side, but I do not think it gives us a complete explanation of the whole problem.

A third suggestion has not yet been fully explored, and I feel that until it has been much further investigated we shall not reach a complete solution of our difficulties. The phenomena of renal excretion are much more dominated by the sympathetic nervous system than has yet been realized. We have seen that the accumulation of the metabolic products in the blood and tissues is due to a sort of arbitrary shutting down of the renal activities, and that the microscopical examination of the kidneys reveals no sufficient reason for this behaviour. We know also that this suspension of activities can be brought about, without any apparent

disease of the organs, by a simple stimulation producing a partial or complete anuria, as in the well-known instance of a calculus in one ureter causing anuria not only of the affected side but also of the opposite one; uraemic symptoms may occur when no appearance suggestive of obstruction can be found; such a simple proceeding as the passage of a catheter often appears to be the immediate cause of a tendency to anuria in predisposed cases; and a period of anuria is sometimes provoked by manipulations of the ureters by the passage of ureteric instruments. One may recall here Case 3, in which the manipulations of the kidney, necessary to establish a nephrostomy, were sufficient to cause complete anuria for twenty-four hours, after which it excreted freely.

One is inclined to suggest, therefore, that stimuli from a prostate, stricture, calculus, etc., may provoke a primary reflex inhibition of renal function by way of the sympathetic nervous system, the degree of inhibition being in proportion to the severity of the causative stimulus. It is difficult to believe that cells such as those which make up the renal excretory apparatus can be rendered functionless for prolonged periods by toxic influences alone without suffering permanent damage, and yet recovery is often rapid and apparently complete. Poisoning of the renal cells must often, of course, exist, and to it one must attribute the permanent damage which occurs, but I feel certain that for a full understanding of these cases evidence of the effect of irritation, mechanical or toxic, on the sympathetic control of the kidneys must be sought for before we shall be able to give an exact explanation of all the phenomena. It is noteworthy that the inhibitory effect of toxins on the nervous tissues of the brain is often prolonged, and recovery is frequently slow. It may be months before symptoms such as those of mental depression disappear and the patient is normal.

In illustration of this, and in conclusion, I will quote a portion of the preface of a novel, *Aphrodite in Aulis*, by George Moore, and dedicated to Sir John Thomson-Walker, who had removed the author's prostate:

"At the end of two months and a half I staggered out of the Home into a cab, a shrunken piece of wreckage, craving for the seashore. The sea and its salt breezes restored me to health, and soon I began to write; but I burnt as much as I wrote, and when you came to see me before leaving London for a holiday, I told you that I had given up all hope of writing *Aphrodite in Aulis*. 'The words won't stick together,' I cried, with doleful accent. 'My gift of writing, such as it was, has left me. I am strong in health, but mentally paralysed.' 'The poison of uraemia,' you answered, 'affects the creative parts of the brain for at least six months.' 'Another three months of empty phrases!' I wailed; and you went away to Scotland, leaving me to continue to write and to burn. But every month the burning diminished, and now I am dedicating *Aphrodite in Aulis* to you."

M. Tripodi (*Il Polclinico*, Sez. Med., February 1st, 1935, p. 111) studied the behaviour of the alkaline reserve in the course of hypoglycaemia produced by injections of insulin in normal and diabetic subjects. He found that the alkaline reserve was lowered in normal subjects and in patients with mild diabetes on the occurrence of hypoglycaemic symptoms, while in severe diabetes with acidosis it rose at first and then fell, but as a rule not below the original level before the administration of insulin. The intravenous injections of large doses of bicarbonate of soda on the appearance of hypoglycaemic symptoms did not prevent the occurrence of more severe symptoms or relieve severe symptoms when they had once developed. Tripodi concludes that the production of hypoglycaemic symptoms caused by administration of insulin cannot be connected with the excessive concentration in the blood of ketonic and lactic acids, as maintained by some writers.

Clinical Memoranda

Acriflavine Bowel Instillation for Relapsing Fever (*B. abortus*)

This case report should prove of interest from the therapeutic point of view.

A schoolboy, aged 12, during the latter part of a term at school, began to feel out of sorts, and gradually lost 7 to 8 lb. in weight. At home he was thought to be run down, but he began to develop a swinging temperature, eventually rising to 102° in the evening, returning to normal in the morning. Typhoid fever was suspected, but the Widal reaction was negative. However, the agglutination test for *B. abortus* of relapsing fever was found to be positive. Salicylates and quinine seemed to be of no account, but *B. abortus* vaccine improved the condition somewhat, but it may only have been one of the usual intermission periods of a lower degree of swinging temperature. Instillation of half a pint of one five-thousandth neutral acriflavine solution into the colon, repeated daily for one week, brought the temperature speedily to normal, and it has remained so ever since. From that time the boy's condition has improved steadily, and he has now quite recovered from his illness, the duration of which was about three weeks.

Acriflavine is rapidly absorbed, and is being excreted through the kidneys within half an hour; the presumption is that it exercises an antiseptic action on the blood stream.

Prestwick.

ARNOLD JONES,

A Case of Intestinal Obstruction

The following case of apparent volvulus with faecal vomiting, the patient living without being operated on, seems worthy of record.

A male East Indian of about 23 years walked into the Mabaruma Hospital, North-West District, British Guiana, at 8.30 one night and reported that a man had struck him on the back with a stick. On examination a small abrasion about the size of a threepenny-piece was seen over the sacral region. No injury to the spinal column could be detected. The bladder was distended. A rubber catheter was passed and left in. The next day the patient had faecal vomiting and looked very ill. The stipendiary magistrate was informed and the patient's dying deposition taken. I may here state that, as a result of inquiries made by the police, they were satisfied that the patient had not been assaulted. Laparotomy was not considered, as most authorities agree that when faecal vomiting occurs it is really to be regarded as a sign of impending death. However, the patient was given several turpentine enemas with no result. A rectal tube was passed, still with no result. The patient was placed in the knee-elbow position and turpentine enemas repeated frequently. On the fourth day after admission coffee-ground stools were obtained after enemas. On the fifth day small amounts of offensive faeces resulted from enemas. During this period the patient was still vomiting offensive faecal matter, the large intestine being distended and no peristalsis being visible. Pain around the umbilicus was complained of, and the patient was very restless. His temperature never exceeded 99.2° F., pulse 100, and respirations 30. On the seventh day after admission the bowels were moving normally, stools being black and very offensive, and the motions frequent and painful. Bloody-looking stools continued for a fortnight. Patient gradually improved, and was discharged as recovered fifty days after admission.

I am indebted to Dr. J. A. Henderson, surgeon-general, for permission to publish these notes.

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