textbook in English that makes any mention of the bilobed conception, and in the 1942 edition McWhorter's (1917) paper is quoted. On the other hand, the French school of anatomists have long accepted the newer teaching, and in their standard textbook (Rouvière, 1943) the account of the parotid salivary gland is replete with a description of the facial nerve lying between a superficial and a deep lobe. In this connexion it should be emphasized that the American surgeon McWhorter (1917) admitted that he derived his ideas from the French surgeon Grégoire (1912).

Masterly sharp dissection with a knife that is deftly honed at frequent intervals is a heritage of which members of the Anatomical Society of Great Britain and Ireland are justly proud. To display the arborescing facial nerve by cutting formalin-hardened, dehydrated if not desiccated salivary tissue calls for much honing and great skill. I believe that if the supple parotid gland of a fresh subject was distended by injecting an appropriate blue-stained fluid into Stensen's duct, after employing the knife to disengage the peri-parotid fascia and to free the excised mastoid process with the sterno-mastoid muscle attached, the scalpel could be laid aside, and under these conditions, by haemostat and blunt dissection only, the main trunk of the facial nerve could be followed to its bifurcation and further without incising any (blue-stained) salivary tissue.

I am deeply indebted to Dr. L. R. Yealland for examining the muscle reactions and accurately prognosticating in a number of instances where recovery from facial palsy was doubtful; to Professor Anson and his pupils for their encouraging anatomical substantiation of the bilobed structure of the parotid gland; to Mr. C. Eisinger for great assistance in translating foreign works on anatomy; and to Dr. Donald Teare for his careful histological study of the tumours I have removed.

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Hull Royal Infirmary was founded on March 22, 1781, and received its first patients in September of the following year. To mark the transition which took place on July 5 the Managing Committee has issued a handsomely produced booklet which has been prepared by Mr. K. J. Lowson. This tells the story of the Infirmary from its foundation to the present day and includes brief biographical notes on all the members of the honorary medical staff. Also included in the booklet are pictures of the hospital in 1784 and in 1850 and there are photographs of the present and past honorary staffs.

ACUTE URAEMIA

BY

S. G. ZONDEK, M.D.

(From the Department of Internal Medicine of the Hadassah Municipal Hospital, Tel-Aviv)

Extrarenal uraemia—or “prerenal uraemia” according to Fishberg (1939) and “renal anoxia” according to Maegraith *et al.* (1945)—is probably the most frequent form of acute uraemia (Zondek, 1944, 1946; Maegraith *et al.*, 1945). This view is supported by the results referred to in this paper.

Extrarenal uraemia is a non-specific body-reaction—i.e., a pathological process met with in entirely different diseases. The uraemia in question is called “extrarenal” (other possible terms will be discussed later), as it occurs in diseases not primarily involving the kidneys and in patients who may not have previously suffered from kidney trouble. The renal failure in these cases seems to be of a functional nature; another, and essential, characteristic is its association with extrarenal azotaemia, a process caused by increased endogenous breakdown of protein which can be diagnosed (Zondek, 1944, 1946) by symptoms such as an increase of non-protein nitrogen in the blood in addition to a dissociation of the urea-sodium-chloride excretion (in the urine, great quantities of urea and small quantities of sodium chloride).

If quite different groups of diseases, comprising not only general affections such as burns, crushing, surgical shock, and general infections (e.g., Weil's disease) but also affections of special organs, such as liver diseases and myocardial infarctions, can cause either pure external azotaemia or extrarenal azotaemia plus renal failure—i.e., extrarenal uraemia—might not primary renal disease, too, occasionally be the cause of this symptom-complex? The following case may prove this assumption correct.

Case 1

A man aged 48 underwent a calculus operation of the urinary bladder a few months before the onset of his present illness. Three days before his admission to hospital cystoscopic examination was carried out because of new troubles and was followed immediately by chills and high temperature. Death occurred 48 hours after admission. While in hospital he had two more chills and the usual symptoms of severe infection. X-ray examination revealed the presence of a calculus in the lower part of the right ureter. The urine contained only traces of albumin and leucocytes. A blood examination showed: white cells 13,400 per c.mm., with a marked shift to the left (juvenile cells 34%). Urea on the day of admission was 136 mg. per 100 ml.; two days later (the day of death) it was 153 mg. Uric acid was 8 and 9.7 mg. per 100 ml. respectively. The urine output for 24 hours amounted only to 1,200 ml. Its NaCl concentration was 0.8 g. per litre and its urea concentration 18.4 g. per litre; accordingly the total excretion on this day was NaCl 0.96 g., urea 22 g. The NaCl-urea ratio was 1:23. Though death resulted from severe septic infection the uraemia certainly was of clinical importance; the nature of the latter did not differ from typical extrarenal uraemia as seen in the course of various non-renal diseases, which is proved by the dissociation of the urea-NaCl excretion and to some degree also by the fact that the urine was actually free from albumin and contained no casts and no cells except leucocytes.

Extrarenal azotaemia is probably also a manifestation of non-specific pathological processes caused by quite different diseases and appearing even in persons who may never before have suffered from any kidney troubles. But what will happen if a person suffering from chronic kidney trouble contracts one of the various diseases potentially able

to cause extrarenal azotaemia or extrarenal uraemia? The occasional coincidence of chronic renal disease and extrarenal azotaemia or extrarenal uraemia seems natural if our basic conception is correct. May we expect interference from both these processes, and what will their clinical peculiarities be? Extrarenal uraemia appearing in a person suffering from chronic renal disease has already been reported (Zondek, 1944). Two more cases, each of a special type, may now be quoted.

Case 2

A man aged 30 was found to be suffering from chronic nephritis two years before the present illness started. Since then he has had permanent albuminuria of 1-3 g. per litre, with leucocytes and casts. Four days before admission to hospital a fever started which lasted five weeks. The clinical picture resembled that of typhoid fever; all serological and bacteriological examinations, however, were negative. Amoebic hepatitis could not be entirely ruled out in view of the fact that temperature eventually subsided after treatment with emetine. Throughout the period of fever the quantity of albumin in the urine remained at about the same level as before, and there was no change after the patient's recovery; the same applies to the cells and casts found in the urine. Blood pressure during the fever period, and later, was not raised; systolic pressure was 100-110 mm. Hg, diastolic pressure about 70 mm. Hg. For the first 8-10 days after admission to hospital—the period with the highest temperature (102-103° F. (38.9-39.4° C.) in the morning and evening)—the findings in blood and urine were typical of extrarenal azotaemia, as seen in the following table.

Details of Case 2

Day of Illness:	5	6	7	8	9	10	14	16	18
Blood:									
Urea (mg. per 100 ml.)	60		50	36	28	24			3.6
Uric acid	7.5			7.2		5.8			
Urine:									
NaCl (g. per litre)	1.7	1.5	4.2	1.1	1.1	1.2	3.2	3.1	6.0
Urea	40.8	41.6	36.0	34.4	28.8	31.2	18.4	12.8	10.6
Urinary excretion daily:									
NaCl (g.)	0.85	0.8	2.1		0.6	0.72	1.92	3.4	
Urea (g.)	20.4	22.5	18.0		14.4	18.7	11.0	13.0	

This case is one of extrarenal azotaemia caused by general infection in a person suffering from chronic nephritis. The extrarenal azotaemia and the chronic nephritis existed separately and did not interfere with each other. Following the accepted lines of diagnosis, the rise of urea and uric acid in the blood would be considered to have been associated with the existing nephritis, and accordingly a state of true uraemia would be assumed. There is, however, no doubt that the condition in question is one of extrarenal azotaemia, which may occur in patients suffering from a similar infection without the kidneys being previously affected. The extrarenal azotaemia is proved by the strong dissociation of the sodium-chloride-urea excretion (NaCl-urea ratio up to 1 : 28) and a daily excretion of urea greatly exceeding that resulting from protein intake. The rise of urea and uric acid in the blood is probably due to increased endogenous breakdown of protein only; considering the high urea concentration in the urine (up to 41 g. per litre) renal insufficiency can hardly be assumed; at least the insufficiency, if it existed, was much less pronounced than it might have been under similar conditions in persons not suffering from any chronic kidney troubles.

Case 3

A woman aged 46 had had her right kidney removed 16 years ago on account of renal calculus. Since then there had been no more complaints concerning the kidneys. Some weeks before admission she had a gall-bladder attack, with chills and pyrexia lasting a few days. Another and particularly severe attack started a week before admission; at the time of admission she had very high temperatures of septic type, chills,

jaundice, and local peritoneal symptoms which lasted five days; from the sixth day all these symptoms subsided. Bile pigments, including bilirubin, which were present in abundance at the height of the illness, likewise disappeared from the urine. At the time of the high temperature the urine contained albumin up to 1 g. per litre; after that only traces were found; in addition there were always a moderate number of leucocytes, but no other pathological elements. For sodium chloride and urea in blood and urine see the table. After an interval of three months,

Details of Case 3

Day of Illness:	8	9	11	12	14
Blood:					
Bilirubin (mg. per 100 ml.)	12.0				2.0
Urea	110	96	60		32
Urine:					
NaCl (g. per litre)	2.6	1.6	1.4	2.0	2.4
Urea	7.0	7.6	12.0	18.0	24.0
Urinary excretion daily:					
NaCl (g.)	2.6	1.9	3.2	4.0	7.2
Urea (g.)	7.0	9.0	29.0	36.0	72.0

during which the patient felt comparatively well, she had another gall-bladder attack with the same clinical symptoms as the previous one. The sodium chloride and urea findings in blood and urine were also similar.

Case 3 is one of severe acute cholecystitis complicated by a state of acute uraemia. The urinary excretion, at least in the first three days, of only small amounts of urea in spite of a high urea level in the blood suggests that the renal insufficiency must have been rather severe. But a process of extrarenal azotaemia, too, must be assumed. This, however, was not recognizable until, from the third day onwards, the daily urinary excretion of urea rose (to 37-70 g.) while the urinary excretion of sodium chloride still remained low; the sodium-chloride-urea ratio was up to 1 : 10.

Extrarenal uraemia is a rather frequent occurrence in severe liver diseases complicated by jaundice (hepato-renal syndrome); therefore at first sight Case 3 does not seem to present any uncommon feature. But further examination revealed some findings deserving close consideration. X-ray examination of the kidneys showed that the pelvis of the left kidney (the right kidney had been removed) was almost entirely filled by calculus masses. A urea-clearance test carried out several weeks after the patient's complete recovery from the acute cholecystitis and the acute uraemia showed 22% of the normal value.

From these findings there can be no doubt that the patient must have been suffering from chronic renal disease with renal insufficiency for a long time, though the latter was not severe enough to raise the urea level of the blood so long as there was no special call upon the kidneys. But the condition changed definitely when, in consequence of the acute liver affection, a process of extrarenal azotaemia set in, thus forcing upon the kidneys the task of eliminating a much greater quantity of substances containing non-protein nitrogen. Yet there is an additional factor to be considered. Since in severe liver disease acute renal insufficiency associated with extrarenal azotaemia may occur even in persons who have never before suffered from any kidney disease, the chances of its appearance may of course have been greatly enhanced because of the already impaired function of the kidneys. But, however severely the pre-existent chronic renal disease may have affected the course and the severity of the acute uraemic state, the latter must be considered to belong to the group of extrarenal azotaemia plus acute non-specific renal insufficiency—i.e., extrarenal uraemia.

Discussion

Chronic and acute uraemia differ from each other not only in their clinical course but in the pathological

processes upon which they are based. Chronic uraemia may occur in the advanced or final stage of all chronic renal diseases. It is a remarkable fact that the morphological changes do not vary greatly according to the primary cause of the renal disease. Whichever the latter may be (chronic nephritis, malignant hypertonia, pyelonephritis, or even renal diseases not belonging to the group of Bright's disease), the morphological process met with when chronic renal insufficiency makes its appearance is that of contracted kidneys. Nor can there be any doubt that renal insufficiency in these cases is closely related to the morphological process, and that chronic renal insufficiency has no other origin than the pre-existence of renal affection and its histological manifestations.

The genesis of acute uraemia, however, is of a different nature. In acute glomerulonephritis, for instance, it is the affection of the glomerular vessels, eventually leading to glomerular ischaemia, which must be held responsible for a possible occurrence of acute renal failure. But, whether the original nature of this affection be a functional spasm (Volhard, 1931) or a true endocapillaritis (for literature see Fishberg, 1939), the pathological process represents, as generally assumed, nothing but a special body-reaction ("allergic" according to Schick) to an extrarenal process—i.e., streptococcal infection in general. Glomerulonephritis, however, most certainly represents the background of only a minority of all cases of acute uraemia, while the majority belong to that group of renal failure defined here as extrarenal uraemia. Actually the theory that acute uraemia is due to glomerulonephritis and the latter to an extrarenal process is not quite correct, since the former is to some extent also extrarenal. In spite of this the terms should not be changed, at least not at present. Since acute glomerulonephritis and what it stands for represent a body-reaction to a rather specific cause—i.e., streptococcal infection—while extrarenal uraemia owes its occurrence to a variety of diseases, the big difference in the incidence of the two forms of acute uraemia cannot be surprising. How wide this variety of causative diseases is may be realized when it is said that even acute uraemia occurring in patients suffering from chronic renal diseases may be of the type of extrarenal uraemia. The occurrence of acute uraemia in cases of chronic renal disease has always been a difficult matter to explain, since the latter, according to its morphological characteristics, supposedly leads only to chronic uraemia. Perhaps the observations reported here may throw some light on this question.

In cases of extrarenal uraemia renal insufficiency may be regarded as functional. Though marked and well-defined morphological changes may be met with (Zondek, 1944, 1946), particularly of the tubular apparatus (degeneration or even necrosis of the epithelium, chiefly that of the ascending loop of Henle and of the distal convoluted tubules), they do not, even in the severest cases, represent a necessary finding; accordingly pathological elements such as albumin, cells, and casts in the urine may be absent. A striking difference must be assumed between the pathological processes underlying the uraemia in cases of acute glomerulonephritis and that in cases of extrarenal uraemia. While the former condition is characterized by an affection of the glomerular capillaries (functional spasm or endocapillaritis), sometimes verging on total glomerular ischaemia, in the latter a similar process has never been detected by microscopical examination. In addition, in cases of extrarenal uraemia blood pressure is found to be normal or even particularly low, while it is high in cases of uraemia connected with acute glomerulonephritis. This rise in blood pressure is only an expression of the vascular process mentioned above and is not restricted to the kidney

vessels alone. On the other hand, disturbances of the blood supply to the kidneys may also be responsible for the renal failure in cases of extrarenal uraemia (Fishberg, 1939; Maegraith *et al.*, 1945; Trueta *et al.*, 1946); they are, however, of a different nature from those typical of acute glomerulonephritis.

According to Fishberg, reduced renal blood flow is the decisive change found in "prerenal" uraemia; Maegraith *et al.* and Trueta *et al.*, in addition to the reduction in the total blood flow, particularly stress a redistribution of the blood flow within the kidneys, which latter, in their opinion, is characterized by a whole or partial diversion from the cortex. According to Trueta *et al.* this change in blood supply is due to abnormal nervous reflexes—a suggestion advanced on the strength of very interesting and important experiments. The conception of a redistribution of blood flow within the kidneys fits in very well with the morphological renal findings as described by many authors (for literature see Maegraith *et al.*) and recently again confirmed by Darmady (1947)—namely, that in cases of extrarenal uraemia the cortex is always found to be pale and anaemic, while there is a marked congestion of the juxta-medullary area and an engorgement of the vessels in the medullary zone.

The fact that extrarenal uraemia is always associated with increased endogenous breakdown of protein led to the assumption that metabolites originating from this process may affect the kidneys and be responsible for the occurrence of renal failure (Zondek, 1944, 1946). This hypothesis does not contradict that of Maegraith *et al.* and Trueta *et al.*, who regard the disturbances of the blood supply as the decisive causative factor. Possibly the supposed metabolites—as also considered by the above-mentioned authors themselves—may affect the nervous centres and thus become the cause of the abnormal blood circulation within the kidneys.

Obstruction of the urinary passages has until recently been regarded as the exclusive cause of acute uraemia, such as may occur after intravascular haemorrhages—e.g., blackwater fever, transfusion of incompatible blood, and other acute haemolytic processes—as well as that sometimes occurring in the course of treatment with sulphonamides. Precipitated pigment and precipitated crystals of sulphonamides were respectively supposed to obstruct mechanically the tubules (intrarenal obstruction) and the lower parts of the urinary passages—i.e., the pelves of the kidneys and the ureters (extrarenal obstruction). Such a mode of action may account, indeed, for certain cases of uraemia—i.e., those where great masses of crystals of sulphonamides obstruct the pelves and particularly the ureters. According to more recent investigations (Murphy and Wood, 1943; Foy *et al.*, 1943; Maegraith and Findlay, 1944), however, the possibility of intrarenal obstruction as the only or even the main cause of uraemia must be abandoned, and this holds true in cases of uraemia associated with haemolytic processes as well as those caused by sulphonamides, etc. The direct toxic action of drugs (sulphonamides, compounds of mercury) on the renal tissue may be considered possible. But this can hardly be the case in acute haemolysis. Since haemolytic processes always represent diseases of a serious character, it seems from the first more likely that renal failure in these cases is of the nature of extrarenal uraemia such as occurs in so many other diseases characterized by a turbulent course. Support for this assumption can be found in the fact that the morphological renal changes met with in cases of blackwater fever and transfusions of incompatible blood have been observed to resemble closely those which may be detected in cases of undoubted extrarenal uraemia such as crushing and pyloric stenosis (for literature see Maegraith *et al.*).

There may remain cases of acute uraemia which do not belong to any of the groups referred to, but they cannot be numerous. On the other hand we may assume that the great majority of all possible cases of acute uraemia have been met with and been grouped, and in the result extrarenal uraemia has been found to account for most of the cases in question.

There is general agreement that contracted kidney is the pattern for *chronic* uraemia. It may not be an exaggeration to assert that extrarenal uraemia is, to say the least, the most frequent pattern for *acute* uraemia. As to the pathological processes typical of extrarenal uraemia, the following triad of specific features—increased endogenous breakdown of protein, disturbance in the blood supply to the kidneys and certain morphological changes in those organs—has so far been recognized. They, all or in part, form the background for the renal failure, the nature of which, however, even if recognized as functional, is not as yet clearly defined. There is, for instance, no certainty whether the chief cause of the functional disturbance is glomerular filtration or tubular reabsorption or whether it comprises both of these basic processes; nor is it certain what the peculiarities of these processes are. These questions certainly open a wide field for investigation.

It cannot be denied that the term "extrarenal uraemia" (and also "prerenal uraemia") may give rise to some misunderstanding in so far as it may be considered to infer that there exists a state of uraemia where renal insufficiency does not represent the basic process. Such a conception, of course, does not fit in with the facts. In "extrarenal" uraemia renal insufficiency does not play a smaller part than in any other condition determined as "uraemia." The term "extrarenal" refers to the primary disease, which in extrarenal uraemia is not necessarily a renal affection as in the classic form of uraemia. The eventual misunderstanding will certainly be avoided by accepting the term "renal anoxia" as proposed by Macgrath *et al.* (1945). But, even admitting that the uraemia in question may actually be caused by renal anoxia only, the two processes can hardly be identified with each other: the fact that this uraemia is caused by renal anoxia certainly does not justify the conclusion that in turn renal anoxia necessarily leads to renal failure. For comparison, the subject of cardiac pathology may be referred to. Though acute cardiac failure can be caused by coronary failure, the latter does not necessarily involve the former; therefore the term "coronary failure" cannot replace that of "cardiac failure." For the same reason the term "renal anoxia" can hardly be applied when a state of renal failure is to be expressed.

In a previous publication "extrarenal uraemia" was designated also as "functional renal failure"; but there may be arguments against this term also: first, in view of the morphological renal changes found in cases of extrarenal uraemia the functional nature of the latter might not be regarded as absolutely proved; secondly, other forms of uraemia, such as that which occurs in cases of glomerulonephritis, may be functional too. If "extrarenal uraemia" for the reason given above should no longer be regarded as wholly appropriate, the term "acute non-specific uraemia" might best fit in with all the facts.

Summary

Acute uraemia occurring in cases of chronic renal disease may be of the type of extrarenal uraemia.

Extrarenal uraemia represents the pattern for most of the cases of acute uraemia.

The term "extrarenal uraemia" may be replaced by "acute non-specific uraemia."

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FIBROSITIS

BY

JAMES CYRIAX, M.D.

Physician to the Department of Physical Medicine,
 St. Thomas's Hospital, London

Fibrositis has been divided into primary and secondary. This is a separation with which I am in the fullest agreement; for in my opinion primary fibrositis is an imaginary disease and secondary fibrositis is a real entity.

Primary Fibrositis

Controversy has gone on for many years about the nature and identity of the different disorders included by common consent under this heading. The existence of fibrositis is affirmed by most clinicians, denied by most pathologists, but in the absence of an alternative explanation for the symptoms and signs purely negative views have carried little weight.

It is my purpose to draw attention to the ready solution to the problem of the existence or not of fibrositis that can be obtained by going back to first principles. This involves taking a detailed history, making a clinical examination of the patient, and drawing deductions on accepted lines from the physical signs discovered in each case. If this is done the conclusion is forced upon the unprejudiced observer that the symptoms so readily ascribed in the past to "rheumatic fibrositis" (i.e., fibrositis coming on for no apparent reason) are all in fact the result of articular lesions. I say "unprejudiced observer" advisedly; for I started my professional life as the very reverse, and it is only recently that I have been able to convince myself that the condition has no real existence.

Historical Note

In 1816 Balfour described fibrous thickenings occurring in the muscles in chronic rheumatism; Froriep, in Weimar, again drew attention to them in 1843. Sir William Gowers, in an article on lumbago in 1904, coined the term "fibrositis" to denote inflammatory changes in the fibrous structure of the sacrospinalis muscles. He went on to point out—perfectly correctly, as time has shown—that subsequent sciatica is "primarily an affection of the fibrous sheath of the nerve." Ever since, the disease has been recognized by medical men as a true entity. For example, Stockman (1920) shows photomicrographs of inflammatory changes detected in excised fibrous tissue from patients with fibrositis. Steinberg (1942) illustrates polymorphonuclear infiltration and proliferation of fibroblasts in a piece of the trapezius muscle excised from a woman who for 14 years had had generalized muscular stiffness ascribed by several