task to make clear that the protection afforded by barrier creams is questionable, and that it has not yet been scientifically substantiated; we should demand that the experts employed by manufacturers publish the evidence on which their views are based in such a form and in such detail that it can be critically assessed; and we should insist that the formulae of these products are made known. The claims of those who make and sell these preparations are specious and their literature voluminous, but I have shared Porter's experience that these firms yield little information when one seeks the precise evidence on which their claims are based, and when one tries to find out the composition of their products.

I am not saying that all barrier creams are completely useless; only that we do not know and at present have no means of knowing. If this conclusion is correct, then no employer should be blamed or held negligent for failing to provide them. The present unsatisfactory situation is partly our fault, and we should reconsider our position. First, we should actively advocate the use of barrier cream only when we know that the proof of its value is soundly based (at the present time I suggest this means never); anything short of this should be regarded as an experiment and should be planned accordingly. Second, we should adhere to the general principle that we cannot recommend the use of an application whose exact composition is not made known to us. At the present time proprietary barrier creams are secret remedies.

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OLIGURIC RENAL FAILURE OF SURGICAL ORIGIN

BY

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Renal failure, an omnibus term used to describe events which follow inadequate renal control of the internal environment of the body, may develop as an acute phenomenon in patients with previously normal kidneys or in patients with overt or occult antecedent renal disease. In its acute oliguric form it is a relatively rare complication of major surgical and traumatic conditions, and, perhaps for this reason, may be temporarily overlooked. While a high fatality rate may be inevitable as a direct result of the primary condition, delayed recognition and inadequate treatment of the secondary acute renal failure makes the overall prognosis significantly worse.

Acute oliguric renal failure has been well documented during the years since the second world war (Legrain, 1951; Swann and Merrill, 1953; Aoyama and Kolff, 1957; Hamburger *et al.*, 1958; Salisbury, 1958; Parsons and McCracken, 1959), but, apart from the wartime experiences recorded by Smith *et al.* (1955), Balch *et al.* (1955), and others, the surgical aspect of the problem, in the main, has received little specific attention. This despite the fact that a significant proportion of all cases of acute oliguric renal failure have a "surgical" basis.

Moreover, conclusions drawn from experience gained in the management of "medical" and "obstetric" patients with acute renal failure are not necessarily applicable: not only is the prognosis significantly worse in the surgical patients but their detailed management is often much more complicated and difficult. Oral feeding may be impracticable. Abdominal distension or vomiting may require prolonged use of an indwelling gastric tube with risk of pharyngo-oesophageal irritation, ulceration, bleeding, and the development of chest infection. Repeated gastro-intestinal aspiration may add to the difficulties of fluid and electrolyte management. Poor wound-healing, wound dehiscence, infection, secondary haemorrhage, and the development of intestinal fistula may occur. Incontinence may develop, and it may be virtually impossible to prevent the development of pressure sores.

The so-called "conservative" management of patients with acute oliguric renal failure (Borst, 1948; Bull *et al.*, 1949), by fluid and protein restriction, administration of carbohydrate, insulin, and calcium (Meroney and Herndon, 1954), ion-exchange resins (Evans *et al.*, 1953), and sodium lactate, as a rule easily applicable to "medical" and "obstetric" cases, is used whenever possible: there are, however, some potential difficulties in the "surgical" cases. It may not be easy to assess extrarenal fluid and electrolyte losses from fistulae, skin burns, or skin and intestinal ulceration: oral ion-exchange resins may not be effective in cases of ileus (they may even be dangerous because gastric casts

may form); and it may be difficult to find adequate veins for continued parenteral treatment and daily blood-sampling for biochemical control, especially when multiple limb injuries are present and plaster-of-Paris casts are required. Peritoneal dialysis may be impracticable and even dangerous in post-operative abdominal cases. And when haemodialysis by an artificial kidney is required heparinization in the presence of recent wounds or intracranial injury may be dangerous. It may be difficult to gain access to suitable veins or arteries for the haemodialysis, and the dialysis may have to be carried out without continual systolic and diastolic blood-pressure recordings.

Acute oliguric renal failure is more a functional designation than a diagnosis. It connotes an acute onset, an inadequate urine output, and renal inability to maintain the internal environment of the body: it does not imply a specific pathological lesion. In normal circumstances, when the kidneys are not diseased, urine volumes of 400 to 500 ml./24 hours may be adequate to rid the body of unwanted products of protein metabolism such as urea, potassium and inorganic acids such as phosphates and sulphates: when the urine volumes are less than 400 to 500 ml./24 hours the condition is designated oliguria. Oliguria may be classified as (*a*) extrarenal pre-renal, (*b*) renal, and (*c*) post-renal obstructive (Aird, 1957), or considered to result from (*a*) extrinsic renal failure (which includes pre-renal and post-renal oliguria), and (*b*) intrinsic renal failure (Waugh, 1959).

Polyuric renal failure may occur in surgical patients. It may complicate the recovery stage of acute tubular necrosis, the commonest form of acute intrinsic renal failure, it may follow instrumental or surgical relief of post-renal obstruction (see below), and it may present as a manifestation of chronic renal disease.

Extrinsic Renal Failure

Pre-renal Oliguria

Pre-renal oliguria, sometimes called extrarenal azotaemia but preferably designated acute renal circulatory failure, occurs when the renal blood flow and glomerular filtration rate are depressed. The condition may complicate the oligaemia of haemorrhage or it may follow plasma depletion in patients with extensive burns or ulceration of the skin or bowel. It may be a sequel to water and sodium depletion in gastro-intestinal conditions, particularly when there is excessive vomiting, gastric aspiration, diarrhoea, or loss through intestinal, biliary, or pancreatic fistulae. It may complicate hypercalcuria, or metabolic alkalosis and hypokalaemia.

The urine in pre-renal oliguria has a relatively high specific gravity, usually not less than 1018, a urea concentration not less than 1.5 g./100 ml., and a sodium concentration not greater than 30 mEq/l. Overt clinical evidence of blood loss, plasma loss, or water and sodium depletion may be present, and hypotension, tachycardia, and a lax skin may be prominent features; a high P.C.V. and a raised serum sodium concentration reflect dehydration.

The diagnosis of pre-renal oliguria is confirmed when, after replacement of blood, plasma, or water and sodium, there is a good diuresis and rapid restitution of the abnormal blood biochemistry. However, prolonged vasoconstriction of the renal vessels, a homoeostatic mechanism invoked to maintain blood-pressure, may cause acute tubular necrosis, an ischaemic lesion of the kidneys which is probably the commonest cause of acute intrinsic renal failure and a much more serious condition.

Pre-renal extrinsic renal failure may occur in patients with known or previously unsuspected chronic renal disease. In such circumstances a sustained diuresis, at least 3 l./24 hours, is required before unwanted products of protein metabolism can be cleared: the urine specific gravity is then low, usually not greater than 1014, and the urea concentration less than 1 g./100 ml. This urine may thus resemble qualitatively that excreted in the oliguric phase of acute tubular necrosis.

Post-renal Obstructive Oliguria

Post-renal oliguria is an obstructive oliguria. The obstruction is often complete, functionally if not organically, and there is anuria rather than oliguria. It is to be distinguished from retention of urine, which complicates prostatic, bladder-neck, or urethral obstruction and which is manifested by the presence of a distended bladder; this physical sign may, however, be obscure in obese patients, particularly post-operative and traumatic cases, and urethral catheterization may be needed for diagnosis.

Except for those rare cases which follow accidental injury during pelvic surgery, bilateral ureteric lesions responsible for the development of post-renal oliguria are only rarely simultaneous: in most instances one ureter has been previously subjected to a "silent" obstruction for some time, and only subsequently, when the second ureter becomes involved, does the condition reveal itself by the onset of oliguria or anuria. This sequence of events may be seen in cases of carcinoma of the cervix, prostate, and bladder.

When oliguria or anuria develops as a sequel to calculous obstruction of a solitary functioning kidney, in cases of congenital absence of one kidney the aetiology may be obscure until cystoscopic examination reveals trigonal deficiency and absence of one ureteric orifice: the problem is not as great when it is known that nephrectomy has preceded the event. Although it is possible for a patient with a solitary functioning kidney to develop acute intrinsic renal failure from acute tubular necrosis, or indeed acute nephritis or cortical necrosis, loin pain and tenderness, a palpable kidney, anuria rather than oliguria, and radiographic evidence of calculi strongly suggest post-renal obstruction. Unilateral calculous obstruction associated with reflex inhibition of function of the contralateral organ is a great rarity, and the exact mechanism responsible for this phenomenon remains obscure.

Cystoscopy, ureteric catheterization, and retrograde pyelography should be attempted whenever the diagnosis is in doubt (it is unwise to omit pyelography and to rely only on conclusions drawn from the passage of ureteric catheters), but oedema and bruising of the bladder base may obscure the ureteric orifices in post-operative cases and it may be impossible to catheterize the ureters. Ureteric catheterization may also fail when the ureters are lax, tortuous, but yet unobstructed, and lead to false conclusions regarding the aetiology of the acute oliguric renal failure. Post-renal obstruction, however, usually produces some degree of loin tenderness, although such tenderness may at times be very slight in degree and difficult to elicit in a drowsy patient. The post-renal oliguria of idiopathic retroperitoneal fibrosis raises

diagnostic difficulties because, paradoxically, ureteric catheters may be freely passed up to the kidneys: however, the presence of intermittent oliguria or anuria, and the absence of calculi and prostatic or uterine malignancy, should suggest the diagnosis, particularly when there is elevation of the blood sedimentation rate. Surgical exploration may be required, not only to establish the diagnosis but also to effect treatment.

Intrinsic Renal Failure

Renal Oliguria

Renal oliguria may result from lesions of the renal arteries, veins, glomeruli, or tubules (Milne, 1957). The renal arteries may be occluded by a dissecting aneurysm of the aorta or by intimal stripping at aortography (Gaylis and Laws, 1956): the renal veins may thrombose in infants with gastro-intestinal disease (Regan and Crabtree, 1948) or in adults with amyloidosis which complicates chronic sepsis or a reticulosis. Glomerular lesions, rare in surgical patients, may be a reflection of glomerulonephritis or cortical necrosis.

Acute tubular necrosis is the commonest cause of acute oliguric intrinsic renal failure in surgical cases (and non-surgical cases, too) and may develop as a complication of (*a*) surgery or surgical conditions and (*b*) accidental trauma. Transfusion of incompatible blood has been incriminated as a cause (Barlas and Kolff, 1959), but the lesion almost always follows an episode of hypotension, the significance of which may be overlooked until the subsequent development of oliguric renal failure; indeed, relevant records of the blood-pressure during or shortly after surgical operations and in traumatic cases before the patient reaches hospital may not be available. Subsequent compensatory vasoconstriction with restitution of the blood-pressure may mislead the observer. Jaundiced patients, particularly when submitted to surgery under general anaesthesia (Shackman *et al.*, 1953), seem peculiarly susceptible to the effects of hypotension, and acute oliguric renal failure due to acute tubular necrosis may develop in circumstances which seldom produce such changes in non-jaundiced patients. The term "hepato-renal failure" is applied to this phenomenon.

The urine in acute tubular necrosis has a relatively low specific gravity, usually not greater than 1014, a urea concentration less than 1 g./100 ml., and a sodium concentration greater than 30 mEq/l.: albuminuria, with a moderate excess of white blood cells and a few hyaline casts, is usual. When the urine sodium concentration is less than 30 mEq/l., the renal lesion is more likely to be glomerular, and acute nephritis or cortical necrosis should be suspected.

Post-operative Oliguria

Le Quesne (1954) has drawn attention to the post-operative oliguria, associated with sodium retention, which may follow surgical operations. It is pre-renal in origin and, in the context of the present paper, has no special significance except that it may, on occasion, lead the observer to overlook the possibility that acute tubular necrosis or post-renal obstruction may, in fact, be really responsible for the oliguria. The urine specific gravity and sodium concentration partly resolves the issue: a high urine specific gravity (at least 1018) and a low sodium concentration (not greater than 30 mEq/l.) is indicative of a pre-renal post-operative oliguria but does not necessarily exclude an incomplete post-renal obstruction. Ureteric catheterization with retrograde pyelography may be required to establish the true diagnosis.

Since October, 1956, when an artificial kidney unit was established at Hammersmith Hospital, 106 surgical patients have been treated for acute renal failure. Most of the patients were transferred, at various stages of their illnesses, from other hospitals: some degree of artificial and unintentional case selection was thus inevitable. In the main, only severe cases and those likely to require dialysis were received.

During the same period, 123 "medical" patients and 48 "obstetric" patients with acute renal failure were admitted.

Aetiology of Pre-renal Extrinsic Renal Failure

Only four patients (Table I) with pre-renal extrinsic renal failure were seen (there is little doubt, however, that several more such cases occurred and were successfully treated at the parent hospitals during the period under review); pre-existing chronic renal insufficiency was present in three and post-operative pyloric obstruction was present in one. Fluid and electrolyte depletion followed acute intestinal obstruction in two of the patients (one referred to above and one other with abdominal adhesions and severe bilateral chronic pyelonephritis), rapid decompression by suprapubic cystostomy in one with chronic prostatic obstruction, and excessive vomiting in one with severe bilateral polycystic kidneys.

TABLE I.—*Cases of Pre-renal Extrinsic Renal Failure*

Case No.	Surgical Condition	Age and Sex	No. of Dialyses	Outcome
1	Suprapubic cystostomy with chronic prostatic obstruction	71 M	0	Survival
2	Intestinal obstruction; chronic pyelonephritis	38 F	0	,,
3	Polycystic kidneys	45 M	1	,,
4	Post-operative pyloric obstruction	46 M	1	Death

Case History

A medical practitioner aged 45 (Case 3) was able to conduct a busy general practice until a week before he was admitted to a hospital in Surrey with malaise, vomiting, and weakness. He realized that his general health had gradually deteriorated in the previous 12 months, and indeed he had suffered a transient attack of right hemiparesis, right homonymous hemianopia, and dysplexia during this time: a significant hypertension remained unexplained. He was transferred to Hammersmith Hospital in a stuporous state with the blood urea 415 mg./100 ml., serum potassium 3.8 mEq/l., and CO_2-combining power 10 mEq/l. It was soon clear that he had bilateral loin swellings, and these were shown by retrograde pyelography to be polycystic kidneys. A diagnosis of pre-renal extrinsic renal failure superimposed on chronic renal insufficiency was made and haemodialysis was carried out with dramatic clinical and biochemical improvement. He was subsequently able to ingest a 20-g. protein diet and sufficient fluids to produce a daily urine output of 3 l., with the result that the blood urea was sustained at 150 mg./100 ml. He was discharged to convalescence and was subsequently able to spend most of the summer on his boat off the south coast of England until he rapidly deteriorated again six months after haemodialysis. He found it increasingly difficult to drink large volumes and the urine output fell. The blood urea, which had been maintained between 120 and 150 mg./100 ml. during the summer, rose to 410 mg./100 ml. and the CO_2-combining power fell to 15 mEq/l. He was readmitted to Hammersmith Hospital and again successfully treated by

haemodialysis on the artificial kidney but died four days later from an antibiotic-resistant septicaemia. Post-mortem examination confirmed the diagnosis of severe bilateral polycystic kidneys and showed cerebral atherosclerosis with local infarction in the left parieto-occipital region.

Aetiology of Post-renal Extrinsic Renal Failure

There were 23 patients with post-renal obstructive extrinsic renal failure (Table II): obstruction was due to malignant disease in 11, calculi in four, retro-peritoneal fibrosis in four, tuberculosis in two, and surgical complications in two. Of the 11 patients with malignant disease, four had carcinoma of the bladder, four carcinoma of the uterus, and three carcinoma of the prostate. The ureteric obstruction resulted from direct bilateral spread of the primary tumour in the pelvis in nine patients, metastatic spread into lymph nodes at the bifurcation of the iliac vessels in one patient with a solitary functioning kidney, and metastatic spread into para-aortic nodes with involvement of the proximal ureter of a solitary kidney in one.

TABLE II—*Cases of Post-renal Extrinsic Renal Failure*

Case No.	Surgical Condition	Age and Sex	Treatment	Outcome
5	Carcinoma of bladder ..	61 M	Laparotomy: no treatment	Death
6	,, ,, ,, ..	74 M	Pyelostomy	Survival
7	,, ,, ,, ..	71 M	Uretero-colic anastomosis	Death
8	,, ,, ..	45 M	Nephrostomy	Survival
9	,, ,, cervix .	66 F	Uretero-colic anastomosis	Death
10	,, ,, ,, ..	54 F	Pyelostomy	Survival
11	,, ,, ,, ..	37 F	Uretero-colic anastomosis	,,
12	,, ,, ,, ..	72 F	,, ,,	Death
13	,, ,, prostate	73 M	Pyelostomy; oestrogens	Survival
14	,, ,, ,, ..	56 M	Oestrogens	,,
15	,, ,, ,, ..	59 M	,,	,,
16	Retroperitoneal fibrosis	67 M	Nephrostomy	,,
17	,, ,, ..	55 F	Ureterolysis	,,
18	,, ,, ..	50 F	Ureterostomy	,,
19	,, ,, ..	58 M	Nil	Death
20	Calculus anuria: solitary kidney	42 F	Ureterolithotomy	Survival
21	,, ,, ,,	59 F	,,	,,
22	Reflux anuria: ureteric calculus	68 M	,,	,,
23	Calculus stricture left ureter; atrophic right kidney	50 M	Nephrostomy	,,
24	Haemonephrosis; nephrolithotomy	24 M	,,	,,
25	Renal tuberculosis ..	17 M	Ureterostomy	,,
26	,, ,, ...	34 M	,,	Death
27	Ureteric ligation; total hysterectomy	61 F	Laparotomy. Anaesthetic death	,,

Of the four patients with calculous obstruction, two were known to have had previous nephrectomy, one had an aplastic contralateral kidney, and one, in whom subsequent excretion urograms showed good function on both sides, had reflex anuria after calculous impaction on one side.

Of the four patients with retroperitoneal fibrosis, two had idiopathic periureteric fibrosis (Stueber, 1959), one had fibrosis following organization of a retroperitoneal haematoma after aortic grafting, and one had fibrosis after total hysterectomy and pelvic irradiation.

Tuberculous stricture of the ureter of a solitary functioning kidney produced post-renal obstructive failure in two patients: tuberculostatic drug therapy had been employed in both cases and no doubt expedited the fibrotic process responsible for the obstruction.

Post-operative post-renal obstruction occurred in two patients: one had both ureters accidentally ligated at hysterectomy and one had an obstructive blood clot in the renal pelvis of a solitary kidney after nephrolithotomy.

A high proportion of our patients with post-renal obstructive renal failure were over the age of 55, and the primary cause was malignant disease: indeed, the onset of acute renal failure in elderly patients should suggest this possibility, though examples of acute nephritis, acute tubular necrosis, oligaemic pre-renal extrinsic renal failure, and non-malignant post-renal obstructive failure have been seen in patients in this age-group.

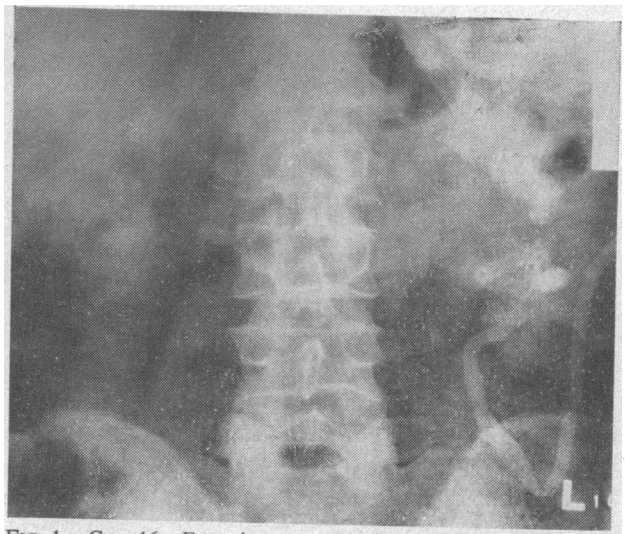

FIG. 1.—Case 16. Excretion urogram after emergency left nephrostomy for acute obstructive post-renal failure due to periureteritis fibrosa. Bilateral hydronephrosis is present and the nephrostomy tube can be seen.

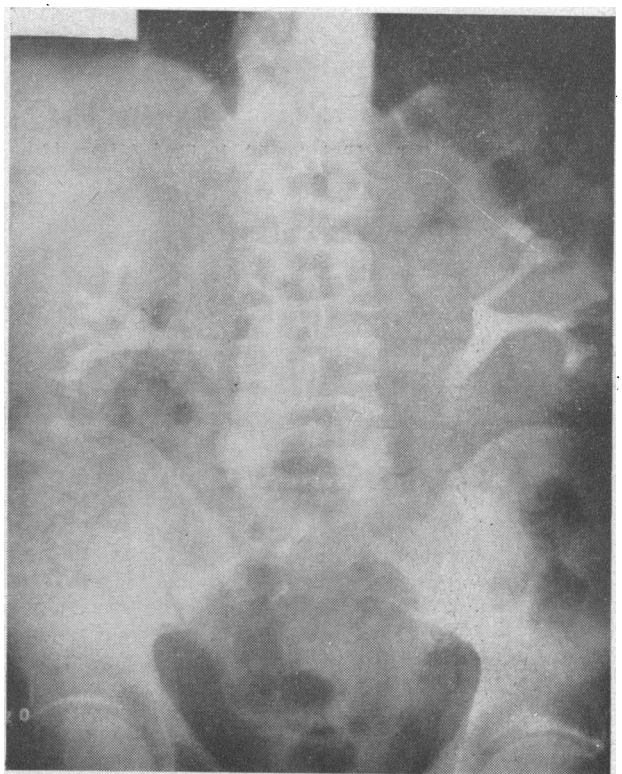

FIG. 2.—Case 16. Excretion urogram several months after bilateral ureterolysis. There is no trace now of the previous bilateral hydronephrosis.

Case History

A farmer aged 67 (Case 16) noticed a gradual onset of malaise, low back pain, and a tendency to constipation approximately eight months before admission to Hammersmith Hospital. Clinical examination elsewhere had shown no abnormality, and a barium-enema *x*-ray examination was reported to be normal. He was advised to take a long sea cruise, and while on his way home from South Africa he noticed a decrease in his daily urine output; finally, two days before admission he became virtually anuric.

On examination he was well nourished, drowsy, and a little pale. There was no loin tenderness, the bladder was not distended, and the prostate felt normal on rectal examination. The blood-pressure was 180/105, the blood urea 316 mg./100 ml., the serum potassium 7.6 mEq/l., the CO_2-combining power 15 mEq/l., the E.S.R. 100 mm., the haemoglobin 9.8 g., and the W.B.C. 7,000. He was treated by oral "resonium-A" and intravenous 40% dextrose with insulin. Ureteric catheterization was attempted but was unsuccessful: the catheters would not pass beyond 1 cm. Accordingly it was decided to explore the left kidney under general anaesthesia (this was an arbitrary decision, as loin tenderness and swelling were conspicuously absent). At operation the left kidney was found to be moderately enlarged and somewhat tense. A nephrostomy was carried out and the urine was found to have a specific gravity of 1025. Rapid restitution of the clinical and biochemical state paralleled a copious flow of urine from the nephrostomy, and after three weeks the blood urea had become 32 mg./100 ml., the serum potassium 3.8 mEq/l., and the CO_2-combining power 24 mEq/l.; the blood-pressure had fallen to 130/80. Excretion urography was then possible and showed bilateral hydronephrosis (Fig. 1). The cause of the hydronephrosis was obscure; there was no clinical, radiographic, or cystoscopic evidence of prostatic obstruction, and ureteric catheters could now be passed freely up to both kidneys. Accordingly the abdomen was explored through a long paramedian incision six weeks after the nephrostomy and both ureters were found to be embedded in dense retroperitoneal fibrosis; they were freed without great difficulty. It was not long before normal micturition was re-established and the nephrostomy wound closed spontaneously: indeed, he was discharged home three weeks after the exploration. Excretion urography carried out nine months later showed no abnormality (Fig. 2); the blood urea was 30 mg./100 ml., the E.S.R. was 3 mm., and the patient had regained his normal health.

Aetiology of Intrinsic Renal Failure

Intrinsic renal failure was present in 79 patients; it developed as a complication of (*a*) surgery or surgical conditions in 50 and (*b*) accidental trauma in 29. Acute tubular necrosis was present in 77 cases and cortical necrosis in two.

Surgery or Surgical Conditions

Of the 50 patients in this group (Table III), 28 had been subjected to elective surgery, 18 had been admitted to hospital as abdominal emergencies, two had been admitted with diabetic coma and developed pancreatitis as a complication (Trever and Cluff, 1958) one was

TABLE III.—*Extrarenal Conditions Responsible for the Onset of Acute Intrinsic Renal Failure*

Case No.	Surgical Condition	Operation	Age and Sex	No. of Dialyses	Outcome	Day of Death
28	Post-op. peritonitis; acute pancreatitis	Partial pancreatectomy	43 M	2	Death	23
29	,, ,,	Cholecystectomy; exploration C.B.D.	61 F	4	,,	22
30	,, ,,	Partial pancreatectomy	63 M	0	,,	9
31	Post-op. biliary peritonitis; acute pancreatitis	Exploration C.B.D.	50 M	0	,,	11
32	Peritonitis; acute pancreatitis	Nil	66 F	0	,,	13
33	,, ,,	Laparotomy	52 M	0	,,	99
34	,, ,,	,,	49 F	2	,,	18
35	Peritonitis; acute pancreatitis; diabetic coma	Nil	19 F	0	,,	12
36	,, ,,	,,	64 M	0	,,	13
37	Post-op. peritonitis; acute pancreatitis	Emergency gastrectomy	44 M	0	,,	5
38	Post-op. biliary peritonitis	Cholecystectomy	55 F	1	,,	32
39	,, ,,	,,	58 M	3	Survival	
40	,, ,,	Cholecystectomy; exploration C.B.D.	63 M	2	Death	17
41	,, ,,	,, ,,	65 F	2	,,	24
42	Peritonitis; perforated diverticulitis coli	Colostomy and drainage	61 F	0	,,	9
43	,, ,,	,, ,,	58 F	0	Survival	
44	Peritonitis; perforated colon	Retropubic prostatectomy	67 M	0	Death	5
45	Peritonitis; ulcerative colitis	Laparotomy and drainage	70 F	0	,,	20
46	Post-op. peritonitis	Colostomy	42 F	0	,,	11
47	Post-op. peritonitis; perforated diverticulitis	Closure of colostomy	57 M	1	,,	8
48	Post-op. peritonitis	Gastro-enterostomy. Vagotomy	52 M	0	,,	14
49	Peritonitis; P.G.U.	Suture of perforation	57 M	0	,,	7
50	Post-op. peritonitis; haematemesis	Emergency gastrectomy	63 M	1	,,	5
51	Peritonitis; acute appendicitis	Appendicectomy	28 M	0	,,	15
52	Pelvic peritonitis: post-appendicectomy bleeding	,,	19 F	0	Survival	
53	,,	,,	27 M	1	Death	18
54	Peritonitis	Total hysterectomy	37 F	2	,,	12
55	Wound infection; renal tuberculosis	Nephrectomy	24 M	2	Death	21
56	Wound infection; renal calculus	Partial nephrectomy	49 M	1	,,	35
57	Wound infection; peripheral vascular disease	Lumbar sympathectomy	50 M	2	Survival	
58	Septicaemia; peptic ulcer	Gastrectomy	45 M	0	Death	4
59	Pericarditis and pneumonia; Fallot's tetralogy	Repair ventricular septal defect	15 M	1	,,	8
60	Ascending cholangitis; gall-stones	Nil	53 F	1	,,	8
61	,, ,,	,,	64 F	1	,,	25
62	Hypotension; haematemesis	Emergency gastrectomy	71 M	0	,,	10
63	Hypotension; peptic ulcer	Gastrectomy	48 M	4	,,	23
64	Hypotension; intrahepatic obstructive jaundice	Exploration C.B.D.	40 M	1	,,	20
65	Hypotension; gall-stones	,, ,,	64 F	0	,,	7
66	Hypotension; thrombocytopenic purpura	Splenectomy	45 M	1	Survival	
67	Hypotension; urethral stone	Transurethral lithopaxy	53 M	2	Death	11
68	Hypotension	Excision of cervix	49 F	0	,,	10
69	Hypotension; haematemesis and melaena; gastro-jejunal ulcer	Laparotomy	37 M	0	,,	8
70	Hypotension; dissecting aneurysm	Transection and resuture aorta	37 M	1	,,	9
71	Hypotension; ca. kidney	Nephrectomy	60 M	0	Survival	
72	Hypotension; benign prostatic hypertrophy	Transurethral resection prostate	63 M	0	,,	
73	Hypotension; intimal stripping	Aortography	49 M	2	Death	20
74	Hypotension; cardiac arrest	Laparotomy	49 F	1	,,	18
75	Total nephrectomy; horseshoe kidney		54 M	1	,,	18
76	I.V.C. thrombosis; varicose veins	Bilateral stripping varicose veins	60 F	0	,,	34
77	I.V.C. thrombosis; ca. kidney	Nephrectomy	62 M	0	Survival	

admitted with a dissecting aneurysm of the aorta, and one had been submitted to an aortography for investigation of vascular disease.

Peritonitis was present in 27 of the patients and was associated with pancreatitis in 10 of them; the pancreatitis followed pancreatic or biliary tract surgery in four, it presented primarily as an acute abdominal emergency in three, it complicated diabetic coma with acidosis in two, and followed an emergency gastrectomy for bleeding ulcer in one. The peritonitis was biliary in five instances (including one of the cases of post-operative pancreatitis); it complicated lesions of, or operations on, the large bowel in six instances (pre-operative three, post-operative three), the stomach and duodenum in four (pre-operative one, post-operative three), and the appendix in three (pre-operative one, post-operative two); it followed abdominal hysterectomy in one.

The acute intrinsic renal failure which occurred in the 27 patients with peritonitis was thought to have been due directly to the peritonitis in 25. In the remaining two the peritonitis was pelvic and followed infection of post-operative pelvic haematomata; significant hypotension occurred in both patients at the time of the bleeding, and the acute intrinsic renal failure clearly reflected this phenomenon.

Significant infection, other than peritonitis, was thought to be the cause of the acute intrinsic renal failure in seven patients: retroperitoneal cellulitis and wound infection with an antibiotic-resistant *Staph. aureus* occurred in three instances (lumbar nephrectomy two, lumbar sympathectomy one), calculous obstructive jaundice with cholangitis occurred in two (no surgery), a *Pseudomonas pyocyanea* septicaemia complicated an otherwise straightforward gastrectomy for benign ulcer in one, and an antibiotic-resistant *Staph. aureus* pericarditis and pneumonia occurred in one patient after a heart operation.

Although jaundice, of varying degrees, was present in 12 patients, and may have been contributory, in no instance was it considered to have been primarily responsible for the development of the acute intrinsic renal failure.

Case History

An American Marine colonel aged 40 (Case 64) developed jaundice while on active service in Europe and was submitted to laparotomy under general anaesthesia because the jaundice had persisted for six weeks and the biochemical tests and the liver biopsy strongly suggested obstructive jaundice. The biliary passages were explored, and, although there was no overt extrahepatic biliary obstruction, a T-tube was inserted into the common bile-duct for drainage. A prolonged hypotensive phase followed the operation and oliguria was noted. He was transferred to Hammersmith Hospital three days after the laparotomy and was found to be fully conscious, restless, and deeply jaundiced; the blood-pressure was 140/75, the blood urea 280 mg./100 ml., the serum potassium 6.6 mEq/l., the CO_2-combining power 12.4 mEq/l., and the serum bilirubin 16.5 mg./100 ml. He was given 40% dextrose with insulin and vitamins B and K through a caval catheter, and within two days began to void significant amounts of urine: on the ninth day after operation he voided 1.3 l. Despite diuresis, however, the blood urea continued to rise and reached 635 mg./100 ml. on the eleventh post-operative day: this was accompanied by epistaxis and melaena and led to a decision to carry out haemodialysis. This was successfully achieved and the blood urea was lowered to 180 mg./100 ml. The gastro-intestinal bleeding, however, persisted despite a normal prothrombin time (13 seconds) and transfusions of fresh blood. The urine output fell temporarily, as usual, after dialysis, but reached 0.75 l. on the sixth day after the dialysis (Fig. 3). However, the blood urea rose again to over 500 mg./100 ml. and he became comatose, dying 20 days after the original operation. Further dialysis was not undertaken because it was impossible to control the gastro-intestinal bleeding. Post-mortem examination showed intrahepatic biliary obstructive jaundice, acute tubular necrosis, and uraemic intestinal ulceration.

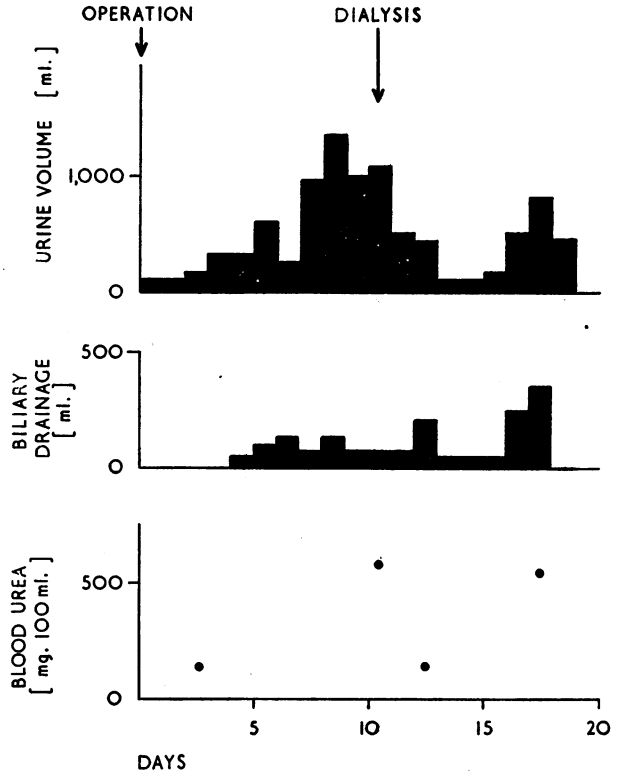

FIG. 3.—Case 64. Daily urine volumes and biliary drainage in a man aged 40 who developed acute tubular necrosis after surgical exploration and drainage of the biliary tract for obstructive jaundice. Haemodialysis was required on the 11th day after operation despite urine outputs of 1.7–1.3 l. The blood-urea values are shown. Uncontrollable gastro-intestinal bleeding precluded further dialysis, and death occurred 20 days after operation.

In 14 patients hypotension due to loss of blood or surgical shock was known to have occurred, and was thought to have been responsible for the development of the acute intrinsic renal failure. It complicated subtotal gastrectomy for benign peptic ulceration in two, exploration of the biliary tract for obstructive jaundice in two, post-appendicectomy intraperitoneal bleeding in two, splenectomy for thrombocytopenic purpura in one, nephrectomy for carcinoma in one, transurethral resection of the prostate in one, transurethral manipulation of an impacted posterior urethral calculus in one, perineal excision of the cervix in one, and cardiac arrest during laparotomy in one: it occurred pre-operatively in one patient who bled from a gastro-jejunal ulcer and in one who developed a dissecting aneurysm of the aorta.

Acute oliguric renal failure followed inferior vena-cava thrombosis in two patients (one after operative stripping of varicose veins and one after nephrectomy for carcinoma), total nephrectomy for carcinoma in a horseshoe kidney in one, and intimal stripping at aortography in one.

Accidental Trauma

There were 29 patients who sustained accidental trauma and developed acute intrinsic renal failure (Table IV). Road accidents accounted for 14, run-over

TABLE IV.—*Extrarenal Conditions Responsible for the Onset of Acute Intrinsic Renal Failure*

Case No.	Injuries	Cause	Age and Sex	No. of Dialyses	Outcome	Day of Death
78	Multiple fractures: pelvis, ribs, long bones. Visceral injuries: right kidney, liver	Road accident	30 M	2	Death	17
79	Multiple fractures: ribs, long bones. Visceral injuries: l. kidney, spleen	,,	20 M	1	,,	12
80	Multiple fractures: ribs, long bones. Visceral injuries: small bowel. Cerebral contusion	,,	59 M	0	,,	10
81	Multiple fractures: pelvis, long bones. Visceral injuries: r. kidney, spleen	,,	44 M	2	Survival	
82	Fractured pelvis	,,	35 M	0	,,	
83	Multiple fractures: pelvis, ribs	,,	16 M	2	,,	
84	Multiple fractures: skull, long bones	,,	27 M	2	Death	11
85	Multiple fractures: pelvis, long bones	,,	63 M	0	,,	6
86	Fractured pelvis: extraperitoneal rupture of bladder	,,	4 M	0	Survival	
87	Ruptured solitary kidney	,,	18 M	0	Death	10
88	Fractured ribs. Visceral injuries: liver, spleen, stomach	,,	21 M	2	,,	9
89	Fractured spine. Ruptured left kidney	,,	50 F	2	,,	17
90	Fractured pelvis: rupture of posterior urethra	,,	54 M	2	,,	20
91	Multiple fractures: long bones	,,	17 M	0	Survival	
92	Multiple fractures: traumatic amputation arm and leg. Intracranial haemorrhage	Run-over accident	52 M	1	Death	7
93	Multiple fractures: pelvis, long bones	,,	37 M	1	,,	12
94	Traumatic amputation leg	,,	60 M	0	Survival	
95	Fractured pelvis: extraperitoneal rupture of bladder	,,	55 M	0	Death	8
96	Multiple visceral injuries: left kidney, liver, pancreas, stomach	G.S.W.	20 M	2	,,	16
97	Lacerated small bowel and I.V.C.	,,	21 M	0	,,	6
98	Lacerated liver	,,	27 M	0	,,	17
99	Multiple visceral injuries: right kidney, liver, transverse colon	,,	18 M	0	,,	7
100	Extensive burns		20 M	2	,,	9
101	,, ,, ...		42 M	1	,,	5
102	Fractured spine	Fall	23 M	1	,,	18
103	Fractured pelvis; extraperitoneal rupture of bladder	,,	36 M	4	,,	20
104	Muscle necrosis; coal-gas poisoning	Domestic accident	23 F	0	,,	9
105	Muscle necrosis; dislocated shoulder	,,	54 M	0	,,	6
106	Multiple fractures: long bones	Pit accident	40 M	1	Survival	

accidents (train, tractor, mobile crane) for four, gunshot wounds for four, extensive burns for two, falls from heights for two, domestic accidents (muscle necrosis after a fall down stairs and complicating dislocation of a shoulder in a recluse, and muscle necrosis complicating coal-gas poisoning in a young woman (Loughridge *et al.*, 1958), for two, and a pit accident for one.

Abdominal visceral injuries (excluding bladder injuries) were present in 11 patients, multiple fractures without visceral injury in eight, pelvic fractures with rupture of the posterior urethra or extraperitoneal rupture of the bladder in four, extensive muscle necrosis in two, and extensive surface burns in two.

Of the 11 patients with abdominal visceral injuries, there were six with multiple visceral injuries and five with concomitant fractures of ribs, pelvis, or long bones. A ruptured kidney was present in seven instances, the liver was ruptured in five, the spleen in three, the pancreas in one, the stomach in two, the small bowel in two, the large bowel in one, and the inferior vena cava in one. Laparotomy was successfully carried out under general anaesthesia in 10 of the 11 patients and there is little to suggest, in retrospect, that it contributed significantly to the development of acute intrinsic renal failure: indeed, there is good reason to believe that prolonged hypotension immediately after the injury was adequate alone to explain the subsequent course of events.

CASE HISTORY

An American non-commissioned officer aged 44 (Case 81) was involved in a road accident while on active service in Europe. He sustained a comminuted fracture of the upper third of the right femur, an open fracture of the right elbow, a fractured pelvis, multiple skin lacerations, a rupture of the spleen and a rupture of the right kidney. Laparotomy under general anaesthesia was carried out within a few hours; the spleen was removed and the right kidney sutured; the fractures were treated and immobilized in plaster-of-Paris. He was given 5 l. of blood. Post-operative oliguria was noted and he was transferred to Hammersmith

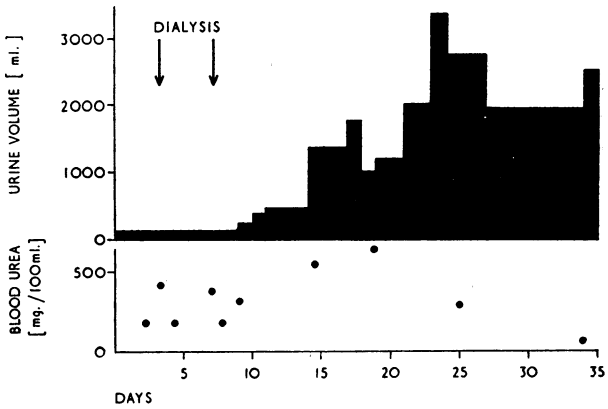

FIG. 4.—Case 81. Daily urine volumes in a man aged 44 who developed acute tubular necrosis after multiple injuries sustained in a road accident. Haemodialysis was required on two occasions before the onset of spontaneous diuresis on the 11th day and subsequent recovery. The blood-urea values are shown.

Hospital two days after his accident, when the blood urea was found to be 170 mg./100 ml., the serum potassium 5 mEq/l., and the CO_2-combining power 20 mEq/l. The blood-pressure was 160/90. A caval catheter was inserted and he was given 40% dextrose with insulin and vitamin B, but, because there was a rapid rise of blood urea, haemodialysis was required on the fourth day after the accident (Fig. 4). A second dialysis was required four days later, and, although a third dialysis was contemplated, spontaneous diuresis began 11 days after the accident, and, despite the fact that the blood urea continued to climb and reached 650 mg./100 ml., restitution gradually ensued. A spontaneous leak of urine occurred from the right loin, and this, together with the hip spica plaster cast required for the treatment of the femoral and pelvic fractures, made nursing extremely difficult and, not surprisingly, he developed a sizable pressure sore over the sacrum. He was finally transferred back to the United States Military Hospital approximately 10 weeks after the initial accident. He was then ambulatory and clinically well, and his blood urea was 30 mg./100 ml. There was, however, a large staghorn calculus in the right kidney which had developed while under treatment and for which subsequent surgery was required.

Prolonged hypotension, a direct result of injury (Clarke, 1957), was thought to have been responsible for the development of acute intrinsic renal failure in the eight patients with multiple fractures without visceral injury, the four patients with pelvic fractures and rupture of the posterior urethra or extraperitoneal rupture of the bladder, and the two patients with extensive burns; myoglobin (Bywaters and Dible, 1942)

liberated from damaged muscles may have been contributory in the two patients with muscle necrosis.

Treatment of Extrinsic Renal Failure

No useful purpose would be served by recording a detailed acount of the treatment, by blood transfusion, water and electrolyte replacement, used in the four patients with pre-renal extrinsic renal failure and the 23 patients with post-renal obstructive failure: it varied from case to case, although it was always based on regular clinical, biochemical, and haematological observations. The patients were seen at least once a day and repeated, usually daily, biochemical observations were made not only on the blood but also on the urine and, when required, on gastro-intestinal aspirate. When post-renal obstruction was present, fluid and electrolyte restriction, as used for the treatment of patients in the oliguric phase of acute intrinsic renal failure, was instituted and maintained until the obstruction was relieved and diuresis occurred.

Haemodialysis by the artificial kidney was carried out in two of the four patients with pre-renal extrinsic renal failure: it was used as a prelude to corrective surgery in one patient with severe post-operative pyloric obstruction and hypokalaemic alkalosis because it proved impossible to correct the biochemical abnormality by potassium administration, and in one with severe bilateral polycystic kidneys, vomiting, and dehydration.

Oestrogen therapy was successfully used in three patients with post-renal obstructive failure due to prostatic carcinomatous involvement of the ureters (although a temporary pyelostomy under general anaesthesia was required in one); 19 of the remaining 20 patients with post-renal obstruction due to causes other than prostatic malignancy were submitted to surgery, under general anaesthesia in 14 instances, spinal anaesthesia in four, and local anaesthesia in one (one patient with retroperitoneal fibrosis due to a leaking aortic graft was considered unsuitable for surgery). Nephrostomy was carried out in four patients, uretero-colic transplantation in four, pyelostomy in three (including one of the patients with prostatic cancer treated by oestrogens), ureterostomy in three, uretero-lithotomy in three, and ureterolysis in one; definitive surgery was not possible in one patient with bladder cancer, and cardiac arrest occurred in one other during operation under general anaesthesia.

After relief of the post-renal obstruction, the daily urine volumes, together with the sodium and potassium contents, were carefully determined so that comparable replacement of water and electrolytes, together with a basal daily requirement of 500 ml. of water, could be given, orally when possible, during the succeeding day.

Treatment of Intrinsic Renal Failure

Oliguric Phase

Excepting eight cases in which it was possible to use the oral route—and here 20% lactose in water is preferable—the primary treatment of the acute intrinsic renal failure was based on the administration of 40% dextrose in water containing insulin (1 unit per 10 g. of dextrose), 10 ml. of 20% calcium gluconate, and a vitamin B compound through a caval catheter; 45 of the 50 "post-surgical" and 26 of the 29 "post-traumatic" cases were treated in this way. The daily allowance was determined by the sum of the basal daily requirement (500 ml. in a 70 kg. patient) and volumes equal to any urine, gastro-intestinal aspirate, vomit, loss from fistulae or diarrhoea; it was sometimes expedient to weigh dressings and the stools. Extrarenal losses of potassium from the gastro-intestinal tract were often significant. Ion-exchange resin (resonium-A, 15 g. four-hourly) for hyperkalaemia was required in less than half the cases; when ileus was present, a watery solution of the resin was administered through an indwelling gastric catheter and was aspirated one hour later. Only rarely was sodium lactate (M/3 or M/6) given as a temporary measure to control acidosis and decrease the serum potassium.

Haemodialysis by the artificial kidney was carried out in 25 of the 50 "post-surgical" and 16 of the 29 "post-traumatic" cases. Multiple dialyses were required in 22: on two occasions in nine "post-surgical" and nine "post-traumatic" cases, on three occasions in one "post-surgical" case, and on four occasions in two "post-surgical" and one "post-traumatic" case. Early dialysis was often required; it was carried out on the fourth day of oliguria in eight patients (two "post-surgical" and six "post-traumatic") and was required before the lapse of one week in 26 (14 "post-surgical" and 12 "post-traumatic").

Urgent transfusions of whole blood were sometimes required to correct oligaemic hypotension. Anaemia, when the haemoglobin concentration was less than 11 g./100 ml., was treated by transfusions of packed cells, which, when delay was justified, were preferably given during dialysis. Surgical treatment was carried out during the oliguric phase in 17 instances.

Diuretic Phase

With the onset of diuresis, which was generally characterized by a rapid improvement in the clinical state, the fluid replacement was increased to balance the urine output, and when significant losses of sodium and potassium were observed comparable replacement was instituted. The caval catheter was withdrawn as soon as possible, and a 20-g. protein diet was given when the blood urea showed a sustained fall and reached 150 mg./100 ml.

Major surgical treatment was carried out during the diuretic phase in seven instances.

Results in Extrinsic Renal Failure

Of the four patients with pre-renal extrinsic renal failure, one recovered and underwent a successful second-stage prostatectomy a year after the acute episode, two others (one with severe bilateral chronic pyelonephritis and one with severe bilateral polycystic kidneys) recovered immediately but died within six months, and one died from pulmonary complications a few days after an emergency gastro-enterostomy for post-operative pyloric obstruction. The series is too small to justify broad conclusions, and, moreover, three of the four patients had significant chronic renal disease.

Of the 23 patients with post-renal obstructive failure immediate success was achieved in 16, by surgery in 14 and by administration of oestrogens in two. No definitive surgery was possible in three others (one died during general anaesthesia, one had an extensive infiltrating bladder cancer, and one had an extensive retroperitoneal fibrosis after a leaking aortic graft). There were three deaths from peritonitis after uretero-colic anastomosis in patients with uterine cancer, and one from persistent renal failure after ureterostomy in a patient with tuberculous ureteric stenosis of a solitary

functioning kidney. Recovery occurred in four patients treated by nephrostomy, three by pyelostomy, three by ureterolithotomy, two by ureterostomy, one by ureterolysis, and one by uretero-colic anastomosis.

Results in Intrinsic Renal Failure

Surgery or Surgical Conditions

Only 8 of the 50 patients who developed acute intrinsic renal failure after surgery or surgical conditions survived to leave hospital. Of the 42 fatal cases, however, death occurred in 28 patients with blood-urea values less than 450 mg./100 ml., serum potassium values less than 6.5 mEq/l., and serum bicarbonate values greater than 14 mEq/l.—values which would not necessarily be expected to prove lethal but which, in the presence of significant and often multiple extrarenal lesions, adversely affected the prognosis. Serious chest complications, pneumonia or purulent tracheobronchitis, occurred in 18 instances, peritonitis in 13, septicaemia in 11, and septic pericarditis in three; severe gastro-intestinal bleeding developed in two patients, a haemothorax in one, and a cerebral haemorrhage in one.

Of 14 patients who were considered to have died of renal failure, there were only four without significant extrarenal complications: in the remaining 10, pneumonia or purulent tracheobronchitis was present in 10 instances, severe gastro-intestinal bleeding in four, peritonitis in three, septicaemia in three, suppurative cholangitis in one, and intracranial bleeding in one.

Of the eight survivors, significant extrarenal complications occurred in six.

Accidental Trauma

Only 7 of the 29 patients with post-traumatic acute intrinsic renal failure survived to leave hospital: one of the seven died a year later from chronic nephritis, which was first recognized during the recovery phase of the superadded acute tubular necrosis.

Of the 22 fatal cases, 14 deaths occurred in patients with non-lethal values of blood urea, serum potassium, and serum bicarbonate although it is not denied that the renal factors contributed to the mortality. Serious chest complications, pneumonia or purulent tracheobronchitis, occurred in 10 instances, pericardial effusions in four, cerebral haemorrhage in two, retroperitoneal haemorrhage in two, peritonitis in one, septicaemia in one, gastro-pleural fistula in one, gas-gangrene in one, cerebral, renal, and pulmonary fat embolism in one, and cerebral oedema in one; anaesthetic deaths occurred in two instances.

Extrarenal complications were relatively few in the eight patients who were considered to have died of renal failure: serious chest complications occurred in three instances, peritonitis in two, and retroperitoneal haemorrhage in one.

Of the seven survivors, only two had significant extrarenal complications.

Discussion

The incidence in civilian practice of acute oliguric renal failure, a serious complication of surgery and surgical conditions, is difficult to assess: it is probably low. Most of the patients collated in the present series were transferred from a comparatively large number of widely separated hospitals, and, although approximately 25 such patients were received each year (a similar number of non-surgical cases of acute renal failure were also admitted), it is not unreasonable to conclude that these represented only a small fraction of the total number of surgical cases seen and treated at the various hospitals during the period under review. An unknown number of less severe cases of acute renal failure may have been successfully managed by "conservative" methods alone at the parent hospitals and did not require the special facilities available in an artificial kidney unit: it is not possible to estimate their number. The fact that only four cases of pre-renal extrinsic renal failure were received suggests that such was probably the case, for patients with uncomplicated pre-renal extrinsic renal failure are easily managed by simple and readily available measures: haemodialysis is seldom required, although it was used in two of the four such patients we received.

Similarly, haemodialysis is seldom required for the treatment of patients with post-renal extrinsic renal failure; indeed, it was not used in the management of the 23 patients in the present series. The mechanical obstruction responsible for the post-renal failure was relieved by surgical measures in the majority of cases; general anaesthesia was used when there was no electrocardiographic evidence of hyperkalaemia, although spinal or local anaesthesia was sometimes preferred. When surgical relief of the post-renal obstruction was followed by a significant diuresis a regime of balanced water and electrolyte replacement was instituted.

The majority (74%) of the surgical patients with acute renal failure had acute intrinsic renal failure, which, with the exception of two hypertensives who died and were shown to have had patchy cortical necrosis, had resulted from acute tubular necrosis. It developed as a complication of surgery or surgical conditions in 47% and followed accidental trauma in 27%. Hypotension, sometimes not recorded, was regarded as the predisposing cause in all cases: mismatched transfusions were conspicuously absent.

Uncomplicated acute intrinsic renal failure in non-surgical patients has a relatively good prognosis: Parsons and McCracken (1959) and Loughridge *et al.* (1960) record a recovery rate of approximately 80%. In contrast, the prognosis is far worse in surgical patients. The recovery rate in the present series was only approximately 20%, and, although somewhat better results have been recorded by others (Smith *et al.*, 1955; Bluemle *et al.*, 1959; Parsons and McCracken, 1959), the differences may not be significant, because there are variations in the nature and degree of severity of the primary surgical conditions, and it cannot be assumed that the ensuing extrarenal complications were comparable in each series of cases. Furthermore, it is not certain that fatalities due to the primary surgical conditions or extrarenal complications were always included in the mortality figures. The high mortality in the present series includes two patients who had been subjected to total nephrectomy and in whom death was inevitable, and 42 who died with significant, but not necessarily lethal, values of blood urea, serum potassium, and serum bicarbonate; in other words, more than half the patients succumbed with, but not from, acute renal failure.

As has been noted by others (Bluemle *et al.*, 1959), we found infection to be the commonest significant complication: it developed in 77% of our patients with acute intrinsic renal failure. Septicaemia, peritonitis,

wound infection, and infection in the urinary tract all occurred with varying frequency: chest infection, either pneumonia or severe purulent tracheobronchitis, was the most frequent and lethal form. Gastro-intestinal and intracranial bleeding, wound dehiscence, and bedsores were also seen.

The fact that haemodialysis was required in 41 of the 79 patients with acute intrinsic renal failure, and that multiple dialyses were carried out in 22 of the 41, more than suggests that there was a high and sustained metabolic response to the surgical and accidental trauma and infection, and that there was rapid biochemical deterioration; indeed, the difference between the metabolic response of surgical and non-surgical patients with acute renal failure is striking (Milne, *et al.*, 1960). The presence of wounds, devitalized tissue, and extravasated blood, apart from the direct effect they produce on biochemical deterioration, provide a portal of entry and a suitable medium for infection which not only provokes an increased metabolic response but also adds directly to the morbidity and mortality.

There is no evidence to suggest that there is any qualitative difference between the renal lesions in surgical and non-surgical cases of acute intrinsic renal failure. There is also, unfortunately, little doubt that a higher mortality rate is inevitable in the surgical patients. While complications of surgery and accidental trauma aggravate the renal aspect of the problem the uraemic state itself encourages their development. The presence of an indwelling gastric tube and mouth-breathing encourage chest infection; prolonged intravenous therapy and poor wound-healing invite bacterial contamination; prolonged recumbency and drowsiness, together with incontinence or an intestinal fistula, encourage the development of pressure sores; and an indwelling urethral catheter is inevitably followed by urethritis, cystitis, and sometimes pyelonephritis: all aggravate the uraemic state. There is, therefore, a dilemma. Surgical patients with acute intrinsic renal failure are peculiarly liable to develop complications which aggravate and are aggravated by the uraemic state. It is to be hoped that the dilemma will be resolved, but this is not likely unless there is a fresh approach to the problem. Haemodialysis effectively controls the biochemical abonormalities of acute oliguric renal failure of surgical origin, but the control is only temporary. Continuous haemodialysis may have more to offer in such cases. Infection, the major hazard, is unlikely to be contained until properly designed isolation facilities with air-conditioned single rooms and separate toilet accommodation are available, and there is an adequate supply of trained nursing staff for duty not only during the day but also during the night. Successful homotransplantation of a kidney might well resolve the problem.

Summary

Acute extrinsic oliguric renal failure (which includes pre-renal and post-renal obstructive oliguria) and acute intrinsic oliguric renal failure may complicate surgical and traumatic conditions.

Experience gained from the management of 106 such cases is presented: extrinsic renal failure was present in 27 (4 pre-renal and 23 post-renal) and intrinsic failure in 79.

Intrinsic renal failure was due to acute tubular necrosis in 77 cases and cortical necrosis in 2; it complicated surgery or surgical conditions in 50 and followed accidental trauma in 29.

The majority of the patients had been transferred at various stages of their illness, and were severe cases likely to require treatment by an artificial kidney. This probably explains the apparent low incidence of pre-renal extrinsic failure.

Post-renal obstruction was a complication of pelvic malignant disease, urinary calculi, retroperitoneal fibrosis, urological tuberculosis, and surgical operations. Treatment by surgery, and oestrogens in cases of prostatic cancer, was successful in 15 of 24 such cases. Haemodialysis was not required.

Of 50 patients with acute intrinsic renal failure following surgery or surgical conditions, the predisposing cause was peritonitis in 25, hypotension in 14, and infection (other than peritonitis) in 7. Renal-vein thrombosis, total nephrectomy, and intimal stripping at aortography accounted for the other four cases. Haemodialysis, often multiple, was required in 25 of the 50 patients.

Hypotension was responsible for the acute intrinsic renal failure in the 29 post-traumatic cases. Road and vehicular accidents occurred in 18. Haemodialysis, usually multiple, was required in 16 of the 29 patients.

Success was achieved in only 34 of the 106 patients. The survival rate in the cases of acute intrinsic renal failure was only approximately 20%, but a high proportion of the fatalities occurred in patients with non-lethal biochemical abnormalities: extrarenal lesions contributed significantly to the high mortality. Infection was the most serious hazard; serious complications included gas-gangrene, multiple fat embolism, gastro-intestinal and intracranial haemorrhage.

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