NAME: MABEOKWU PRECIOUS KOSISICHUKWU

MATRIC NUMBER: 18/ MHS02/ 105

DEPARTMENT : NURSING

Renal system disease, any of the diseases or disorders that affect the human urinary system. They include benign and malignant tumours, infections and inflammations, and obstruction by calculi.

organs of the renal system

organs of the renal system

Organs of the renal system.

Encyclopædia Britannica, Inc.

3d illustration human heart. Adult Anatomy Aorta Black Blood Vessel Cardiovascular System Coronary Artery Coronary Sinus Front View Glowing Human Artery Human Heart Human Internal Organ Medical X-ray Myocardium

Diseases can have an impact on the elimination of wastes and on the conservation of an appropriate amount and quality of body fluid. Many of the manifestations of renal disease can be accounted for in terms of disturbance of these two functions, and the alleviation of symptoms in those renal diseases that cannot be cured depends on knowledge of how these two functions are affected.

The eliminatory process does not, of course, end with the formation of urine; the urine has to pass down the ureters to the bladder, be stored there, and voided, usually under voluntary control. The whole mechanism can be deranged by structural changes in the lower urinary tract, by infection, or by neurological disorders that lead to abnormal emptying of the bladder. Disturbance of the lower urinary tract is an important cause of pain and distress, notably during pregnancy and in the elderly; and it can lead to serious and progressive damage to the kidneys, either by interfering with the drainage of urine or by allowing bacterial infection to have access to the kidney.

Bacteria such as Pseudomonas aeruginosa can cause infections of the urethra and bladder. These infections occur more often in women than in men and are typically associated with significant pain and distress.

Bacteria such as Pseudomonas aeruginosa can cause infections of the urethra and bladder. These infections occur more often in women than in men and are typically associated with significant pain and distress

Functional Aspects

Effects of abnormal renal function on body fluid

Renal disease in its diverse forms can lead to bodily deficits or excesses of water, sodium, potassium, and magnesium, and also to protein deficits occasioned by great losses of protein in the urine. Inability of the kidney to function normally may lead to retention in the blood of the waste products of protein metabolism, such as urea and uric acid, and of other nitrogenous compounds such as creatinine. There may be abnormally high levels of phosphates in the blood, which in turn can lead (for reasons about which there is still some disagreement) to low blood levels of calcium. The calcium deficiency can cause tetany, a condition marked by muscular spasms and pain, and calcium may be lost from the bones in the process of restoring normal calcium levels in the blood and tissue fluid. For descriptive purposes, changes in volume, changes in composition, and protein depletion of renal origin will be discussed separately, but these disturbances can and often do coexist.

Get exclusive access to content from our 1768 First Edition with your subscription.

Subscribe today

Though body fluid is most readily apparent in the bloodstream, it is present, and in larger amounts, in the tissues, both between the cells (interstitial fluid) and within them (intracellular fluid). Extracellular fluids, which include interstitial fluid and blood plasma, amount to 25 percent of body weight and contain sodium as their predominant cation (positive ion; metals and hydrogen in solution are cations). Intracellular fluids, amounting to 33 percent of body weight, have potassium as their predominant cation. These various “compartments” of body fluid are in osmotic equilibrium, so that if solute (e.g., sodium chloride) is added to the extracellular compartment so as to increase the concentration of the extracellular solution, water will join it to reduce the concentration, and that compartment will increase. An increase in extracellular fluid, if it is considerable, may be clinically apparent as edema, a swelling of the tissues by fluid, which can usually be displaced by firm pressure. Edema is present in acute inflammation of the kidney (nephritis), in protein deficiency of renal origin, and in chronic nephritis complicated by heart failure associated with abnormally high blood pressure; a factor common to all these states is failure of the kidneys to excrete sodium and water in adequate amounts.

The kidneys in such edematous states need not themselves be diseased; for example, normal kidneys, in a patient with heart failure, may retain sodium when handicapped in their function by poor circulation and by abnormal amounts of sodium-retaining hormones, such as aldosterone. Increase in extracellular fluids is the only volume change that is both common and easily discernible in renal disease, but the opposite condition, sodium depletion or clinical dehydration, is more commonly the result of vomiting and diarrhea when they are complications of terminal renal disease. Sodium and water depletion can be recognized by a lack of elasticity in the superficial tissues and by poor filling of the blood vessels, as well as by signs of impaired circulation, including a fall in blood pressure and an increase in pulse rate. Though changes in intracellular fluid volume occur in some diseases, especially when the potassium content of the body is affected, there is no easy way of detecting them.

Learn More!

Renal system disease

RELATED TOPICS

Human disease

Renal system

Disease

Bladder cancer

Urinary tract infection

Renal cell carcinoma

Bartter syndrome

Bright disease

Diabetic nephropathy

Urinary tract obstruction

Properties of body fluids

Because of the importance of osmotic forces in determining fluid distribution within the body, an important attribute of body fluid is its overall osmotic concentration, or osmolality. This depends on the concentration of solutes. While all solutes contribute to osmolality, small particles such as sodium or chloride ions are influential out of all proportion to their weight, and indeed account for over 90 percent of the osmolality of plasma. In the context of renal disease, changes in osmolality depend largely on how the kidney handles water. When the kidney either is incapable of conserving water or is not stimulated by ADH of the pituitary to do so, water is lost from the body, and a state of water depletion develops, characterized by increasing osmolality of body fluid. At other times, the kidneys may retain too much water, especially when too much hormone is present; in this case, water excess results, giving a clinical state of water intoxication, with decreased osmolality of body fluids.

Another important general property of body fluid is its degree of acidity or alkalinity. The kidneys are involved in the excretion of hydrogen ions, and imperfect function leads to their retention, the state of so-called renal acidosis. Renal acidosis may occur as part of general renal failure or as a specific disease of the renal tubules, one of whose functions is to convert the slightly alkaline glomerular filtrate into the (usually) acidic urine.

Apart from these general changes in body fluid, the pattern of individual constituents can be distorted in renal disease. For many substances, the problem is one of failure of excretion, with consequent increased concentration in body fluids. Insofar as excretion is achieved by filtration, the rise in concentration may assist excretion, permitting prolonged states of balance, at the cost of increased, but often tolerable, levels of concentration. For example, an individual in renal failure must put out as much urea as a healthy individual taking the same diet; but that person can only do so at a blood-urea concentration of 100 milligrams per 100 millilitres, instead of a normal blood-urea of 25 milligrams per 100 millilitres. Substances whose concentration increases in this way include urea, creatinine, uric acid, phosphate, sulfate, urochrome, and indeed all the usual constituents of urine apart from those that are “regulated” rather than simply “excreted.” Potassium should be mentioned because of the special danger associated with its retention, which can lead to fatal irregularity of cardiac action. This is a recognized danger of acute renal failure, now commonly prevented by use of the artificial kidney and its semipermeable membranes, and sometimes by the use of resins that will take up potassium in the alimentary tract.

Normal urine contains traces of protein, and in many forms of renal disease there is an increased excretion of protein in the urine, usually representing an increased permeability of the tuft of capillaries forming the glomerulus. This increased proteinuria (often, but less correctly, known as albuminuria) generally amounts to 0.5 gram per day or more. When it exceeds five grams per day and persists at this level, the loss of protein in the urine exceeds the capacity of the liver to produce new protein from the available materials; the concentration of protein in the blood decreases, and this leads to an increasing outflow of fluid from the bloodstream into the tissues (there is normally an equilibrium between the physical pressure in the capillaries, which tends to force fluids out, and the osmotic pressure of plasma proteins, the effect of which is to hold fluid in). This balance of forces is upset by a deficit of plasma proteins. The general loss of fluid into the tissues leads to massive edema, to which the kidneys contribute further by retaining salt and water. The combination of high levels of protein in the urine, low protein levels in the blood, and consequent edema is known as the nephrotic syndrome. This is a good example of a syndrome, defined as a recognizable pattern of manifestations that has not one but a number of possible causes. Other examples of syndromes in renal disease are acute renal failure and chronic renal failure.

Disorders of urine flow

If little or no urine appears, it may be because the kidneys are forming little urine (oliguria) or none (anuria); or it may represent a holdup in the bladder or urethra affecting the outflow from both kidneys. About one person in 500 is born with only one kidney, and loss of a kidney from disease or accident is not rare. The loss of a single kidney does not substantially affect an individual’s ability to eliminate wastes, as long as the other kidney functions normally. In cases in which complete obstruction of the remaining ureter occurs, patients will experience effects similar to obstruction of the entire lower urinary tract. Partial or complete failure to form urine is treated in the section on acute renal failure, obstructive conditions in the section on diseases of the urinary tract.

In instances of damage to nervous control, certain typical clinical situations may be differentiated, corresponding to different modes of disordered urinary flow: (1) Lack of conscious inhibition of micturition because of damage to the cerebral cortex or, more commonly, from psychological causes results in a need to micturate that cannot be suppressed even though the bladder volume may be quite small; micturition is precipitate and continues until the bladder is empty. (2) Transverse lesions or other damage to the spinal cord above the sacral reflex centres that cause paralysis of the lower half of the body produce at first a bladder that is atonic (lacking in physiological tone). This bladder becomes greatly distended; the detrusor relaxes and reflex micturition is abolished. Pressure finally rises sufficiently to overcome the spasm of the sphincters and urine is voided in small amounts. Further accumulation and partial voiding of the overflow recur (overflow incontinence). Under these conditions the bladder readily becomes inflamed, which may cause disability or death from chronic ascending urinary infection. Intermittent drainage of the bladder with a catheter may be necessary, or firm pressure on the lower abdominal wall may be used to avoid overdistension and to develop an “automatic” bladder after some time. This is a small capacity organ (around 150 millilitres) with frequent emptying; there is reflex control mediated through the sacral segments of the spinal cord; the higher centres do not restrain the detrusor, and the internal sphincter relaxes more readily. Voluntary assistance from the abdominal muscles helps in this situation if these too have not been paralyzed. There is, however, always some residual urine from incomplete emptying and a risk of infection. In some cases, pressure building within the bladder can be transmitted to the kidneys; without medications or more frequent bladder emptying to relieve the pressure, the kidneys will incur damage. (3) In contrast, there is the isolated, or “autonomous,” bladder resulting from damage to the central nervous system below the sacral cord reflex centres or to the nerves supplying the bladder and urethra. The bladder becomes tense but contracts only weakly so that, while small amounts of urine are voided, the residual urine may be as high as 200–300 millilitres. This condition is known as active incontinence as opposed to the overflow incontinence of the automatic bladder. Here again, more effective emptying of the bladder by catheter drainage may be h

Pain associated with urination (dysuria) can arise from bladder distension, which is then relieved by effective micturition; from inflammation of the lower urinary tract, commonly due to infection but rarely caused by chemical irritants in the urine; and from mechanical irritation by tumour or during the passage of stones. Dysuria is commonly, but not necessarily, associated with frequency of urination. This in turn may represent either an irritable or contracted bladder; or the actual amount of urine formed may be unusually large (polyuria), in which case voiding is likely to be painless. Sometimes polyuria may not be noticed by day but may manifest itself in the need to micturate on several occasions during the night (nocturia). The acute onset of dysuria and frequency suggests urinary infection; sustained polyuria is more likely to be due to renal failure (defective concentrating power) or to diabetes. In those who drink beverages into the night, nocturia is physiological.

Incontinence, the involuntary passage of urine (or feces), may be due to a faulty nerve supply, which either leaves the sphincters relaxed or allows them to be overcome by distension of the bladder. Comatose and disturbed patients, especially among the elderly, are commonly incontinent. Apart from nerve lesions, the sphincters that normally prevent the escape of urine may be damaged by repeated childbirth, by the growth of the prostate, or by other distortions of the bladder neck. Medications to relax the bladder and increase its capacity may be helpful. Alternately, more complete bladder emptying by intermittent catheterization may limit the amount of urine leakage. Procedures have been devised to stimulate the sphincters electrically, when their nerve supply is damaged; or to stimulate the bladder to empty itself at set times. For chronic incontinence, however, devices to catch the urine and prevent soiling of clothing are the most practical

Diseases And Disorders Of The Kidney

In this section, attention is directed not only to specific diseases of the kidney but also to the syndromes of acute and chronic renal failure, which have multiple causes. Infective disorders of the kidney are dealt with later, as part of the general problem of infection of the urinary tract.

3d illustration human heart. Adult Anatomy Aorta Black Blood Vessel Cardiovascular System Coronary Artery Coronary Sinus Front View Glowing Human Artery Human Heart Human Internal Organ Medical X-ray Myocardium

Acute renal failure

Acute renal failure occurs when renal function suddenly declines to very low levels, so that little or no urine is formed, and the substances, including even water, that the kidney normally eliminates are retained in the body. There are two main mechanisms that can produce acute renal failure. When the cardiac output—the amount of blood pumped into the general circulation by the heart—is lowered by hemorrhage or by medical or surgical shock, the renal circulation is depressed to an even greater extent. This leads directly to inefficient excretion, but, more importantly still, the kidney tissue cannot withstand prolonged impairment of its blood supply and undergoes either patchy or massive necrosis (tissue death). Given time, the kidney tissue may regenerate, and it is on this hope that the treatment of acute renal failure is based. The form of acute renal failure that is due to a poor supply of blood (ischemia) has many causes, the most common and most important being multiple injuries, septicemia (infections invading the bloodstream), abortion with abnormal or excessive bleeding from the female genital tract, internal or external hemorrhage, loss of fluid from the body as in severe diarrhea or burns, transfusion reactions, and severe heart attacks; a special case is the transplanted kidney, which commonly goes through a phase of acute renal failure that is independent of possible rejection.

The second common mechanism of acute renal failure is toxic. Many poisons are excreted by the kidney, and in the process, like other urinary constituents, they become concentrated and thus reach levels in the tubular fluid that damage the lining cells of the tubules. Though the tubular cells die and are shed in the urine, regeneration can take place and the patient survive, if he can be maintained during the period of depressed renal function and is not killed by other effects of the poison. Poisons that can affect the kidney in this way are numerous, but the main groups are heavy metals (mercury, arsenic, uranium); organic solvents (carbon tetrachloride, propylene glycol, methanol); other organic substances (aniline, phenindione, insecticides); and antibacterial agents (sulfonamides, aminoglycosides, amphotericin), and some fungi (e.g., Amanita phalloides). In addition to the ischemic and toxic causes of acute renal failure, mention must be made of fulminating varieties of acute renal illnesses that are generally mild (e.g., acute glomerulonephritis—see below) and of the acute form of immunologic rejection that can destroy a kidney irrevocably within minutes of transplantation. Another mechanism of acute renal failure is characterized by acute obstruction of the flow of urine from the kidneys; this condition is easily treated by restoring adequate urinary drainage from at least one kidney.

The course of acute renal failure can usefully be divided into three phases: an onset phase, a phase of established acute renal failure, and a recovery phase. In general, but not invariably, the second of these phases is characterized by a low output of urine (oliguria) and the third by an increasing urine output (polyuria). The onset phase is dominated by general illness, in which the episode of acute renal failure arises; at this stage there may be evidence of threatened renal damage such as blood in the urine or pain in the loins. At this early stage, renal damage may be reversible by prompt treatment of circulatory failure (e.g., by the transfusion of adequate amounts of plasma, whole blood, or electrolyte replacement fluids) and by maintaining adequate blood oxygen levels. Infection or any underlying causative disorder also must be treated quickly.

In the second phase, small amounts of urine, often containing red blood cells, or hemoglobin, are passed; complete absence of urine is not common and suggests that an obstruction is preventing urine from being passed. In quantitative terms, a urine volume of less than 500 millilitres per day constitutes significant oliguria; this is the least amount in which the excretory demand imposed by an ordinary diet can be met. In the actual situation of acute renal failure, the excretory demands may in fact be much greater, since many of the causes of acute renal failure also are causes of increased breakdown of the tissues in general. The blood urea increases, the rate of increase being conditioned both by the degree of renal failure and by the amount of tissue breakdown. Besides nitrogen, the kidney can no longer excrete adequate amounts of water, sodium, and potassium.

These various inadequacies point the way to the necessary management of acute renal failure—the elimination from intake of any dangerous substance that the kidney can no longer handle. The diet must either be free of protein or contain small amounts of high-quality protein to lessen tissue breakdown. It must also be free from sodium and potassium: many persons with renal failure have died from pulmonary edema, a correlate of sodium retention, and others from the acute toxic effects on the heart of a raised level of potassium in the blood. Water cannot be excluded from the intake but must be limited to an amount estimated to equal the unavoidable loss of water from the skin and in breathing. The weight of the patient and the concentration of sodium in the blood are good guides to the adequacy of water restriction. In the absence of continuing losses of sodium from the body, as might occur from vomiting or diarrhea, a progressive fall in serum sodium implies that too much water is being taken in. Kidney function may recover, often in seven to 10 days. The use of dialysis, the removal of waste products by straining the blood through semipermeable membranes, gives further time for renal recovery. Potassium can be removed from the body by resins, but this is less often required if dialysis is available.

Although by comparison with the oliguric phase the recovery phase presents fewer problems, the convalescent kidney takes time to recover its full regulatory function, and electrolytes and water may be lost at an unusual rate during this stage, requiring replacement. Most individuals who survive completely recover from acute renal failure, but residual renal damage persists in some persons. In a few, this is so severe as to bring them effectively into the category of chronic renal failure. The artificial kidney has transformed the outlook for many patients with acute renal failure, and this, together with developments in the control of infection with more powerful antibiotics, constitutes one of the miracles of medicine in the last few decades.

Chronic renal failure

The term uremia, though it is sometimes used as if it were interchangeable with chronic renal failure, really means an increase in the concentration of urea in the blood. This can arise in many acute illnesses in which the kidney is not primarily affected and also in the condition of acute renal failure described above. Uremia ought to represent a purely chemical statement, but it is sometimes used to denote a clinical picture, that of severe renal insufficiency.

Infections of the urinary tract are more frequent during pregnancy, and women who have acute infections of the bladder and kidneys while...

As with acute renal failure, there are many conditions that can lead to chronic renal failure. The two most common causes are pyelonephritis and glomerulonephritis (kidney inflammation involving the structures around the renal pelvis or the glomeruli), and other common causes are renal damage from the effects of high blood pressure and renal damage from obstructive conditions of the lower urinary tract. These primary disorders are described below. They have in common a progressive destruction of nephrons, which may be reduced to less than a 20th of their normal number. The quantitative loss of nephrons can account for the majority of the changes observed in chronic renal failure; the failure in excretion is due directly to loss of glomerular filters, and other features such as the large quantities of dilute urine represent a change in tubular function that could be accounted for by the increased load that each remaining nephron has to carry. There are many other causes of chronic renal failure aside from the four common ones. They include congenital anomalies and hereditary disorders; diseases of connective tissue; tuberculosis; the effects of diabetes and other metabolic disorders; and a number of primary disorders of the kidney tubules. Of the many causes, there are some that have importance out of proportion to their frequency, by virtue of their reversibility; these include renal amyloidosis (abnormal deposits in the kidney of a complex protein substance called amyloid), whose causes may be treatable; damage to the kidney from excessive calcium or deficiency of potassium; uric acid deposition in gout; the effects of analgesic agents (substances taken to alleviate pain) and other toxic substances, including drugs.

The person suffering from renal failure, especially in the early stages, may have no symptoms other than a feeling of thirst and a tendency (shared with many normal people) to pass urine at frequent intervals and through the night; or he may be in a coma, with occasional convulsions. The general appearance of the sufferer may be sallow because of a combination of anemia and the retention of urinary pigment. Even if not in actual coma, the affected person may be withdrawn; muscle twitchings and more general convulsions may occur. The coma is thought to represent poisoning, and convulsions are often related to the severity of the high blood pressure that commonly complicates advanced renal failure. Blurred vision is also a manifestation associated with high blood pressure. Bruising and hemorrhages may be noticeable.

Although the toxin (or toxins) of uremia has yet to be identified, the rapid improvement that follows dialysis points strongly to a toxic component. Urea itself is not notably toxic. Not all the chemical alterations in uremia are simple retentions. There is acidosis—a fall in the alkalinity of the blood and tissue fluids—reflected clinically in deep respiration as the lungs strive to eliminate carbon dioxide. The capacity of the kidney to adjust to variation in intake of salt, potassium, and water becomes progressively impaired, so that electrolyte disturbances are common. Poor appetite, nausea, vomiting, and diarrhea are common in uremic patients, and these in turn add another component to the chemical disturbance. Phosphate is retained in the blood and is thus associated with low blood levels of calcium; the parathyroids are overactive in renal failure, and vitamin D is less than normally effective because the kidneys manufacture less of its active form (1,25-dihydroxycholecalciferol). (Parathyroid hormone causes release of calcium from the bones, and vitamin D promotes absorption of calcium from the intestines.) These changes can lead to severe bone disease in persons suffering from renal failure, because bone calcium is depleted and the calcium stores are not adequately replenished.

In chronic renal failure, excessive production of renin by the kidney can lead to severe high blood pressure (hypertension), and the effects of this may even dominate the clinical picture. In addition to damage to the brain and the retina, the high blood pressure may lead directly to heart failure. Hypertension can also accelerate the progress of renal damage by its impact on the renal blood vessels themselves, setting up a cycle that can be hard to break. Anemia is also often severe due in part to a failure to produce erythropoietin.

The patient in advanced renal failure is vulnerable to infection and other complications, such as vomiting or diarrhea, which need special care. When symptoms of advanced renal failure appear, deterioration can be delayed by a strict low-protein diet, 18–20 grams of high-quality protein each day. In terminal renal failure, the affected person can be rescued only by some form of dialysis and then maintained by dialysis or transplantation.

Learn More!

Glomerulonephritis

Glomerulonephritis is the disorder commonly known as nephritis, or Bright’s disease. The primary impact of the disease is on the vessels of the glomerular tuft. The suffix “-itis” suggests an inflammatory lesion, and glomerulonephritis is indeed associated with infection, in the limited sense that it may begin soon after a streptococcal infection and may be aggravated in its later course by infections of various kinds. Nevertheless, there is convincing evidence that glomerulonephritis does not represent a direct attack on the kidney by an infective agent; it appears to be, rather, an immunologic disorder, in the sense of the formation of antibodies in response to the presence of a foreign protein (antigen) elsewhere in the body; these form antigen–antibody complexes that lodge in the glomerular tuft or, in a small number of cases, themselves become deposited on the capillary glomerular walls. In each case the antibody or the antigen–antibody complex reaches the kidney via the circulation, and the mechanism is usually referred to as circulating complex disease. Glomerular damage is a consequence of the reaction that follows within the glomeruli. These deposits of foreign protein and complexes react with other protein components of blood (see the article complement) and attract to the site white blood cells and platelets, which also are circulating in the blood; these in turn release protease enzymes and other chemical mediators of tissue injury.

This view of glomerulonephritis is based partly on analogy with the renal damage that can be induced in animals by allergic mechanisms and partly on finding that a protein component of the allergic reaction is deposited in the diseased glomerulus. Within the general concept of an immunologic disorder, there is ample room for a variety of primary stimuli and of later immunologic disease-causing mechanisms. These include the possibility of primary glomerular damage, causing the glomerulus itself to become antigenic and so to provide a secondary antibody response, and also the participation of (or lack of participation of) T lymphocytes. Such a diversity is strongly suggested not only by the variations in the glomerular tissues observed both with the ordinary and with the electron microscope but also by the varying manifestations of the disease observed in the affected person.

Typically, glomerulonephritis appears as an acute illness one to two weeks after a sore throat, or—less commonly—after a persistent streptococcal infection of the skin. Other infective agents may be responsible, however, including some viruses and protozoans. A small number of drugs that act as foreign macromolecules can also do so.

The affected person has puffiness of the face and ankles and at the same time scanty and noticeably blood-stained urine. On examination, loose tissues show edema, and the fluid is easily displaced by light pressure; both the blood pressure and the blood levels of urea are slightly or moderately increased. The illness is an alarming one, but the fact is that the acute attack of glomerulonephritis needs no particular treatment other than the eradication of the infection or withdrawal of the offending drug, with some restriction of fluid and protein. Nine out of 10 affected persons recover completely. Exceptional outbreaks, with a higher mortality, have sometimes been observed. A very few patients may die in the acute attack, however, or in a few months’ time, when the impact of the disease has been unusually severe. Another possibility is that the affected person may appear to have recovered completely, having lost all symptoms; but the disease process remains active, and there is progressive loss of nephrons, leading ultimately to chronic renal failure. This process may take many years, for most of which the person has no definite symptoms of latent nephritis except that the urine contains protein and small numbers of red blood cells. It need not be assumed, however, that the finding of protein in the urine (proteinuria) in the absence of symptoms means automatically that the patient has kidney disease; symptomless proteinuria has many causes and may indeed be found in young people who never develop any later evidence of renal disease.

In summary, glomerulonephritis can lead to renal failure within a few weeks or months, after many years of symptom-free proteinuria, or after a period of massive proteinuria, which causes the nephrotic syndrome. All of these manifestations may sometimes be seen in individuals who have never had, or cannot recall, an acute attack. Renal biopsies in many patients with glomerulonephritis show a range of glomerular reactions that include increased cellularity and basement membrane damage and thickening and varying degrees of progressive destruction of glomeruli. In those who recover, complete resolution of glomerular disease occurs.

A curious form of glomerulonephritis especially common in children is associated with little structural glomerular damage, at least as seen by the ordinary light microscope. Characteristic abnormalities affecting podocytes are revealed by electron microscopy. The condition is usually attended by heavy proteinuria and the nephrotic syndrome. Although the evidence for an immunologic cause of this form of glomerulonephritis is less certain than in other types, and the provoking antigen is unknown, paradoxically the disorder usually promptly resolves when the patient is treated with corticosteroids or other immunosuppressive drugs, and renal failure never occurs.

Learn More!

Vascular disease

In the discussion of chronic renal failure, attention was drawn to the cycle in which high blood pressure secondary to renal disease can produce further damage to the kidneys. Clearly, primary vascular disease—disease affecting the blood vessels—could equally well be a cause of renal damage.

The most dramatic instance of this is the condition known as malignant hypertension, or accelerated hypertension, which arises when the blood pressure attains extremely high levels, the diastolic figure (the blood pressure between heart contractions) being 140 millimetres of mercury or higher (the normal being around 80). Sustained levels of this magnitude cause serious damage to the arterioles, the smallest of the arteries; this damage is widespread, but as it affects the kidneys it produces rapid destruction of renal substance, with a scarred kidney. Unless the blood pressure is controlled, malignant hypertension can cause death in a few months; since treatment at an early stage is notably effective, the condition represents an important medical emergency. Since the retinas are damaged as rapidly as the kidneys, the affected person may first notice blurring or loss of vision and will typically have a severe headache. Prompt treatment is necessary to avoid stroke, as well as damage to other organs.

More modest, but still elevated, levels of blood pressure can cause more gradual renal damage in elderly people or in those made prematurely aged by widespread arteriosclerosis (“hardening of the arteries”). In this condition the damage is in the larger arteries rather than in the arterioles, and the condition is one of slowly progressive scarring. Renal damage can also arise, by various mechanisms, in a large number of diseases that impair the proper functioning of the blood vessels, such as diabetes mellitus, the collagen disorders, bacterial inflammation of the heart lining, and many more.

A specific renovascular cause of high blood pressure that, although uncommon, is important from the point of view of the control of blood pressure in healthy individuals involves the juxtaglomerular apparatus (JGA) and the secretion of renin. Occasionally, following trauma or arising spontaneously as a result of vascular disease, one or the other of the main renal arteries becomes constricted (renal artery stenosis). The fall in blood pressure beyond the constriction leads to increased secretion of renin from the JGA with the formation of the vasoactive angiotensin II. As a result, the blood pressure rises. Removal of the affected kidney, surgical repair of the constriction, or percutaneous transluminal angioplasty (a balloon catheter inserted through the skin and inflated in the artery to flatten plaque build-up) usually restores the blood pressure and the blood renin level to normal.

Tumours

Tumours in general are covered in the article Cancer. In this section, those tumours peculiar to the excretory system, and their local effects, are discussed briefly. In the case of benign tumours, these effects include pressure on local structures and obstruction to hollow organs; with malignant tumours, one must add the possibilities of local invasion and of spread by the bloodstream or lymphatics to other organs (metastasis).

Carcinoma

The most common tumour of the renal substance is a carcinoma, renal cell cancer (formerly called a hypernephroma), which is a malignant tumour, arising from epithelial cells (the cells of the bodily coverings and linings). It was formerly thought to arise from adrenal cortical cells lying within the kidney substance. This has since been disproved. One to 2 percent of all tumours are renal carcinomas, and most affected persons are aged from 40 to 60. The tumour may be symptomless or may first be apparent from the occurrence of metastases in the lungs, causing spitting up of blood; or in the bones, causing pathological fracture.

Much more commonly, the first evidence of the tumour is blood in the urine, which may be painless or may cause colic of the ureter, if clots are being passed. There may also be a dull pain in the loins, from stretching of the kidney capsule. The tumour may be directly palpable, or it may be revealed by X rays or ultrasonography. The silhouette of the kidney may be distorted by a rounded swelling; or the renal pelvis, made visible by the injection of a contrast medium, may be displaced or distorted. Less common first indications of renal carcinoma are an obscure fever, or polycythemia (excess of red blood cells in the blood), due to excessive production of erythropoietin. Direct visual examination of the urinary tract with an instrument called a cystoscope may demonstrate the side that is affected, blood coming from one ureteric opening only. Since this bleeding can equally arise from a tumour of the renal pelvis, examination of the renal pelvis is usually called for. An exploratory operation may sometimes be needed; if carcinoma is found to be present, the kidney must be removed. There is some evidence that the results of surgery may be somewhat improved by radiation therapy. The overall outlook is poor, with a five-year survival rate no better than 50 percent. This is, however, one of the forms of malignant tumour in which arrest or even regression has been described.

Nephroblastoma (Wilms’ tumour)

Nephroblastoma is a less common, but nevertheless an important, tumour in childhood, in which other forms of cancer are less common. About half the cases occur at ages two to four, but the tumour may be present even at birth. Early diagnosis, immediate surgery, and chemotherapy constitute the best possibility for a cure.

Other tumours

In addition to tumours of the renal substance, the renal pelvis may be affected by fernlike growths of the epithelium (papillomas). Benign tumours of the kidney substance occur, but rarely; on the other hand, cysts (abnormal sacs filled with liquid or semisolid substance) of the kidney are relatively common but are not tumours in any strict sense, being rather malformations brought about by failure of the embryonic tubules to achieve a proper outlet. Several forms of renal cystic disease, most of them fatal, occur in infancy. Various forms of solitary cyst occur, which may need local surgical treatment if they cause symptoms. The form of polycystic (multiple-cyst) renal disease that allows survival into adult life is a familial condition, in which several members of the family have little trouble until middle life but then are progressively affected by kidney malfunction. Episodes of blood in the urine and urinary infection are common, and the kidneys are large and irregular. Cysts of other organs—e.g., the liver—may be present. X rays show irregularity of the renal pelvis, through pressure from the cysts. Puncture of the cysts is possible, but the results are not encouraging; the general treatment is that of chronic renal failure, which may now include removal of the kidney and transplantation.

Learn More!

Obstruction To The Flow Of Urine

The causes of obstruction to the flow of urine lie in the lower urinary tract and are dealt with in a later section; here it is appropriate to consider the effects of urinary obstruction on the kidney (obstructive nephropathy). It should first be noted, however, that obstructions may arise at the junction of the renal pelvis and the ureter, either from faulty action of smooth muscle or from the pressure of an abnormal blood vessel crossing the pelvis; such cases can benefit from a plastic operation on the renal pelvis or from division of the abnormal vessel. Whether the obstruction arises in this way, or lower down, it can lead to renal pain, to the passage of irregular amounts of urine when obstruction is intermittent, and to a mass in the kidney when obstruction persists. As the renal pelvis swells, the renal tissue shrinks, leading to the condition called hydronephrosis, in which a greatly swollen sac is surrounded by a mere rind of atrophied renal tissue. A massive hydronephrosis, with negligible renal substance remaining, may suggest removal of the kidney.

The kidney may be wounded, usually along with other viscera; it may be bruised; or it may even be ruptured in closed injuries. Since the kidney receives about a fifth of the blood pumped by the heart, bleeding can be profuse, both into the urine and into the tissues and the kidney, forming a large mass of blood, called a hematoma, and leading to surgical shock. Some bleeding may follow the procedure of renal biopsy (taking a specimen of kidney tissue for examination), but with proper precautions this is not severe. In the past, massive irradiation to the kidney region led to chronic renal damage (radiation nephritis), but with adequate precautions, this is no longer so.

The usual signs of traumatic injury to the kidney are blood in the urine and the development of a tender mass in the loin, with progressive signs of shock (pallor, sweating, fall in blood pressure). Such signs call for resuscitation and for surgical exploration if the bleeding continues. The surgical treatment may be carried out to arrest the bleeding by closing the tear. The kidney must be surgically removed if it cannot be saved. Abnormal solitary kidneys are not unknown, and such kidneys are more exposed to trauma by their size or position. Removal of such a kidney can lead only to death unless transplantation is possible.

Substituting For Renal Function

The failure of a vital function normally, and by definition, leads to death; but in the case of the kidneys there are two methods of substituting for renal function: transplantation and dialysis.

Transplantation

In principle the simpler of these two is to transplant a kidney from a donor, ideally an identical twin. The immunologic and surgical problems of transplantation are dealt with in the article transplant. Here only the part played by renal transplantation in the total care of renal disease is considered. The question of a transplant does not arise in most cases of acute renal failure when the loss of function is largely recoverable; and in chronic renal failure it arises only when the residual renal function is barely adequate to support life.

Dialysis

Transplantation and dialysis are complementary rather than rival methods. Dialysis is used while a patient is awaiting transplant and during episodes of oliguria or of threatened rejection, while, on the other hand, patients who find dialysis a psychological burden can be offered a transplant. In addition to its complementary role in a transplant program, dialysis can be used independently in the maintenance of patients with chronic renal failure; and it can be used to preserve life in acute renal failure and in acute poisoning, to allow more time for recovery.

Patient undergoing dialysis treatment.

Patient undergoing dialysis treatment.

© Picsfive/iStock.com

There are two main techniques of dialysis in current use. In peritoneal dialysis, the patient’s own abdominal cavity is used as the container of fluid; the fluid is run in, allowed to reach equilibrium, and removed, taking with it urea and other wastes. The process has proved suitable for the short-term treatment of acute renal failure, especially in infants, and can be used in the treatment of individuals with chronic irreversible renal failure. New techniques have allowed many patients to conduct peritoneal dialysis on their own for limited periods of time.

Hemodialysis (filtration of the blood through semipermeable membranes) has also been used in the treatment of acute renal failure, since the method—the artificial kidney—was devised, in the 1940s; but, for chronic use, the problem was one of repeated access to the arterial bloodstream. This was largely solved by the introduction of a permanent shunt between an artery and a vein (an arteriovenous fistula), by which a suitable vein, usually in the arm, is connected directly to an adjacent artery. The vein becomes distended and so can be repeatedly punctured to gain access to blood, which can then be diverted through the “artificial kidney” when required. In the original artificial kidney, the patient’s blood was pumped through cellophane tubing immersed in a large bath of physiological fluid (solution of the same osmotic pressure as blood); in some later models, streams of blood and of dialyzing fluid are made to flow in opposite directions, separated by plastic sheets. This introduction of the “countercurrent” principle has allowed the apparatus to be smaller, and disposable versions of both patterns are now available. Some patients on intermittent hemodialysis have been kept alive for nearly 20 years. Most continued hemodialysis is still done in hospitals or special centres; but some patients using automatic equipment have been successfully trained to carry out the procedure in their own home.