NAME:ADEJUMO LATEEFAH MUHAMMAD

MATRIC NUMBER:18/MHS02/012

DEPARTMENT: NURSING

LEVEL:200 LEVEL

COURSE TITLE:PHYSIOLOGY

COURSE CODE:PHS 212

DISEASES OF THE RENAL SYSTEM

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.

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.

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.

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

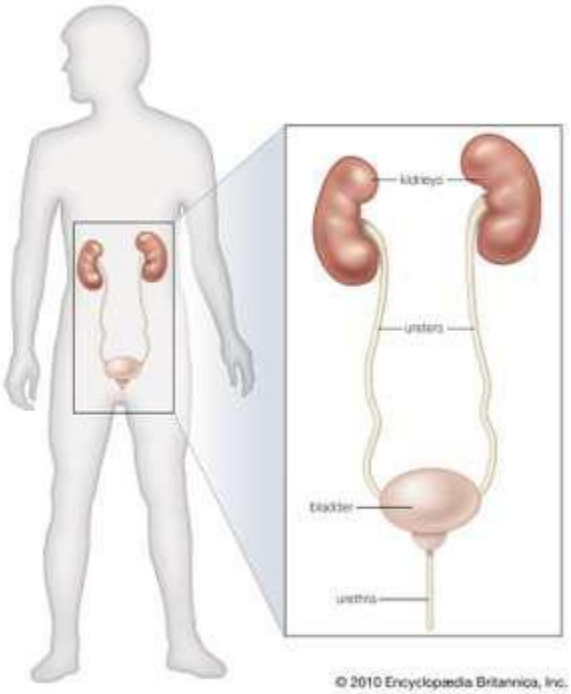
urinary infection. Intermittent drainage of the bladder with a catheter may be necessary, or firm pressure on the lower abdominal wall may be used to avoid overdistension and to develop an "automatic" bladder after some time. This is a small capacity organ (around 150 millilitres) with frequent emptying; 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 helpful.

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.

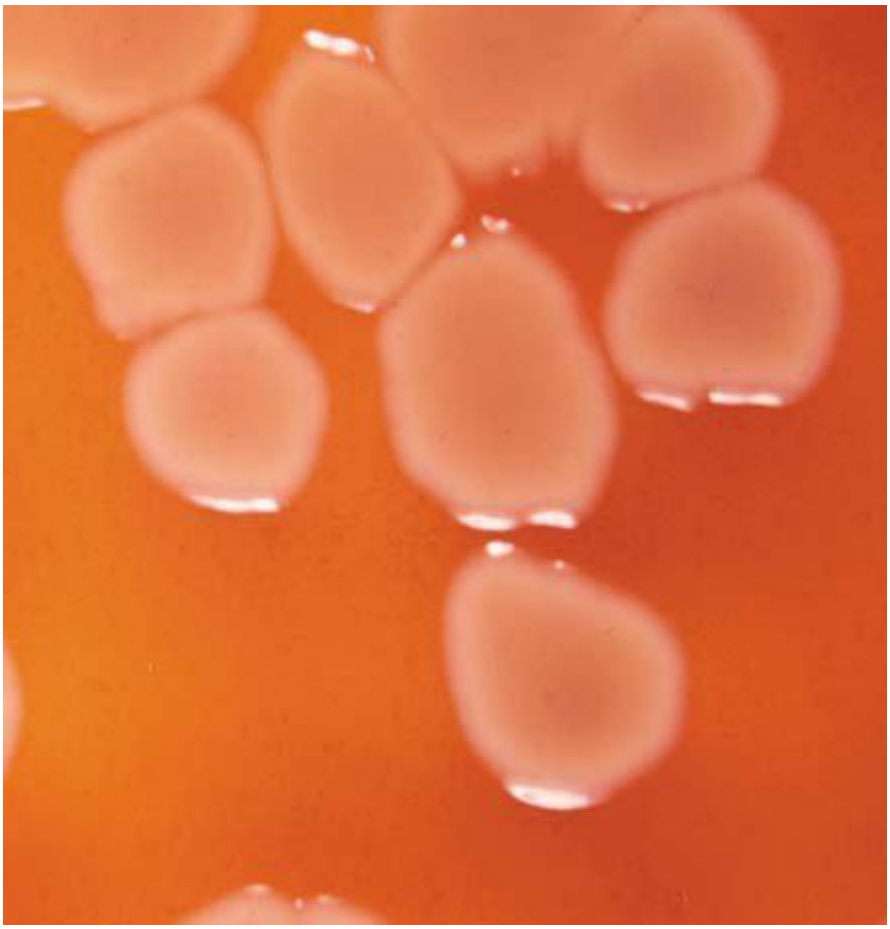


Acute renal failure

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