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**ASSIGNMENT TITLE: RENAL PHYSIOLOGY FOR  
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**COURSE TITLE: RENAL PHYSIOLOGY BODY  
FLUID AND TEMPERATURE REGULATION**

**COURSE CODE: PHS 303**

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Question

Second assignment

1. Discuss the pathophysiological process involves in renal failure?

## **RENAL FAILURE**

Kidney failure, also known as end-stage kidney disease, is a medical condition in which the kidneys are functioning at less than 15% of normal capacity, causing a decrease of not only glomerular and tubular function but also endocrine renal function occurs. Kidney failure is classified as either acute kidney failure, which develops rapidly and may resolve; and chronic kidney failure, which develops slowly.

### **ACUTE KIDNEY FAILURE**

Acute kidney failure is a rapidly progressive loss of renal function, generally characterized by oliguria (decreased urine production); and fluid and electrolyte imbalance. It can result from a variety of causes, generally classified as prerenal, intrinsic, and postrenal. Many people diagnosed with paraquat intoxication experience it, sometimes requiring hemodialysis. The underlying cause must be identified and treated to arrest the progress, and dialysis may be necessary to bridge the time gap required for treating these fundamental causes.

### **CHRONIC KIDNEY FAILURE**

Chronic kidney failure can also develop slowly and, initially, show few symptoms. It can be the long term consequence of irreversible acute disease or part of a disease progression like diabetic nephropathy and glomerulonephritis.

### **CAUSES OF KIDNEY FAILURE**

Causes of acute kidney failure include low blood pressure, blockage of the urinary tract, certain medications, muscle breakdown, and hemolytic uremic syndrome.

Causes of chronic kidney failure include diabetes, high blood pressure, nephrotic syndrome, and polycystic kidney disease.

### **DIAGNOSIS**

Diagnosis of acute kidney failure is often based on a combination of factors such as decrease urine production or increased serum creatinine.

Diagnosis of chronic kidney failure is based on a glomerular filtration rate (GFR) of

less than 15 or the need for renal replacement therapy.

## **COMPLICATIONS**

Complications of acute and chronic failure include uremia, high blood potassium, and volume overload. Complications of chronic failure also include heart disease, high blood pressure, and anemia.

## **THE PATHOPHYSIOLOGICAL PROCESSES OF KIDNEY FAILURE**

### **ACUTE KIDNEY FAILURE**

Acute kidney failure usually occurs when the blood supply to the kidneys is suddenly interrupted or when the kidneys become overloaded with toxins. Causes of acute kidney failure include accidents, injuries, or complications from surgeries in which the kidneys are deprived of normal blood flow for extended periods of time. Heart-bypass surgery is an example of one such procedure.

Drug overdoses, accidental or from chemical overloads of drugs such as antibiotics or chemotherapy, bee stings may also cause the onset of acute kidney injury. Unlike chronic kidney failure, however, the kidneys can often recover from acute kidney failure, allowing the person to resume a normal life. People suffering from acute kidney injury require supportive treatment until their kidneys recover function, and they often remain at increased risk of developing future kidney failure.

Among the accidental causes of acute renal failure is the crush syndrome, when large amounts of toxins are suddenly released in the blood circulation after a long compressed limb is suddenly relieved from the pressure obstructing the blood flow through its tissues, causing ischemia. The resulting overload can lead to the clogging and the destruction of the kidneys. It is a reperfusion injury that appears after the release of the crushing pressure. The mechanism is believed to be the release into the bloodstream of muscle breakdown products – notably myoglobin, potassium, and phosphorus – that are the products of rhabdomyolysis (the breakdown of skeletal muscle damaged by ischemic conditions).

Treatment of acute failure depends on the underlying cause.

### **CHRONIC KIDNEY FAILURE**

Chronic kidney failure has numerous causes. The most common causes of chronic failure are diabetes mellitus and long-term, uncontrolled hypertension. Polycystic kidney

disease is another well-known cause of chronic failure. The majority of people afflicted with polycystic kidney disease have a family history of the disease. Other genetic illnesses cause kidney failure, as well.

Overuse of common drugs such as ibuprofen, and acetaminophen (paracetamol) can also cause chronic kidney failure.

Some infectious disease agents, such as hantavirus, can attack the kidneys, causing kidney failure.

Chronic renal failure is caused by a progressive decline of all kidney functions, ending end with terminal kidney damage. During this time, there is modulation and adaptation in the still-functional glomeruli, which keeps the kidneys functioning normally for as long as possible. The remaining glomeruli, therefore, experience a rise in pressure through hyperfiltration. The release of various cytokines and growth factors leads to hypertrophy and hyperplasia. At the same time, the function of the glomeruli suffers due to the excessive demands on them, leading to increased permeability and proteinuria. Increased protein concentrations in the proximal tube system are direct nephrotoxins and can further impair kidney function.

There are four phases of chronic renal failure, namely:

1. Reduction in Excretory Function: Breakdown of excretory function is the consequence of an accumulation of endogenous and extraneous substances. This leads to changes in pharmacokinetics and an increase in the concentration of various medications.
2. Reduction in Incretory Renal Function: Because the kidney plays a part in the regulation of many important hormonal cycles, chronic renal failure also has endocrinal consequences. Through a shortage of erythropoietin, there is a reduction in erythrocyte synthesis, which leads to renal anemia; uremia then leads to a reduction of functional erythrocytes due to hemolysis or hemorrhages. Vitamin D production is also impaired, and phosphate excretion is reduced. Secondary hyperparathyroidism and the associated renal osteopathy ('high-turnover' osteopathy) develop as a result of hyperphosphatemia. Parallel to this, other pathomechanisms lead to a disruption in bone metabolism: osteomalacia occurs due to a disruption of mineralization, and adynamic bone disease occurs due to a reduction in bone cell activity (particularly in dialysis patients).

3. Over-hydration and the Disruption of Electrolyte Balance: As long as the glomeruli can manage to compensate, diuresis and fractional sodium excretion rise. If the glomerular filtration rate noticeably drops, then the ability to compensate is exhausted, leading to increased retention of water and electrolytes. Hypertension, pulmonary edema, and peripheral edema result from overhydration. Water and salt excretion are thereby inextricably linked. Diuretics can aid in water and salt excretion where critical glomerular damage is present. Early loss of salts as a result of the disturbance in the resorption process can actually be made worse by the use of diuretics. Thus, as the glomeruli adapt to compensate, the tubular transport mechanisms also adapt in order to prevent hyperkalemia through increased potassium secretion. Hyperkalemia only develops as a result of hyperstimulation of the resorption capacity.
4. Toxic Organ Damage as a Result of Retention of Urinary Excreted Metabolites: Toxic organ damage can be explained under the umbrella term 'uremic syndrome.' The rise in urinary excreted metabolites in the blood is called azotemia. These metabolites include urea, creatinine, beta-2 microglobulin, parathyroid hormone, among others. Uremic syndrome (uremia) principally describes a systemic disruption of all organ functions, especially the circulatory system, central nervous system, blood, and membranes. Uremia can also lead to hemolysis with anemia. Simultaneously, thrombocyte and leukocyte dysfunctions or deficiencies can arise. Seizures can occur. Uremia also causes polyneuropathy with paresthesia.

People with chronic renal failure have a generally increased risk of atherosclerosis with an elevated cardiovascular risk. This leads to media calcification caused by calcium phosphate and to intima calcification through inflammatory factors and cholesterol plaques. Hypertension is common, along with edemas and pulmonary congestion.

Treatment of chronic failure may include hemodialysis, peritoneal dialysis, or a kidney transplant.

## 2. With the aid of suitable diagrams discuss the types of dialysis you know?

### DIALYSIS

Dialysis is the process of removing excess water, solutes, and toxins from the blood in people whose kidneys can no longer perform these functions naturally. It is also called renal replacement therapy.

Dialysis is used in patients with rapidly developing loss of kidney function, called acute kidney failure, or slowly worsening kidney function, called chronic kidney failure. Dialysis is used as a temporary measure in either acute kidney failure or in those awaiting kidney transplant and as a permanent measure in those for whom a transplant is not indicated or not possible.

### PRINCIPLE

Dialysis works on the principles of the diffusion of solutes and ultrafiltration of fluid across a semi-permeable membrane. Diffusion is a property of substances in water; substances in water tend to move from an area of high concentration to an area of low concentration. Blood flows by one side of a semi-permeable membrane, and a dialysate, or special dialysis fluid, flows by the opposite side. A semipermeable membrane is a thin layer of material that contains pores of various sizes. Smaller solutes and fluid pass through the membrane, but the membrane blocks the passage of larger substances (for example, red blood cells and large proteins). This replicates the filtering process that takes place in the kidneys when the blood enters the kidneys and the larger substances are separated from the smaller ones in the glomerulus.

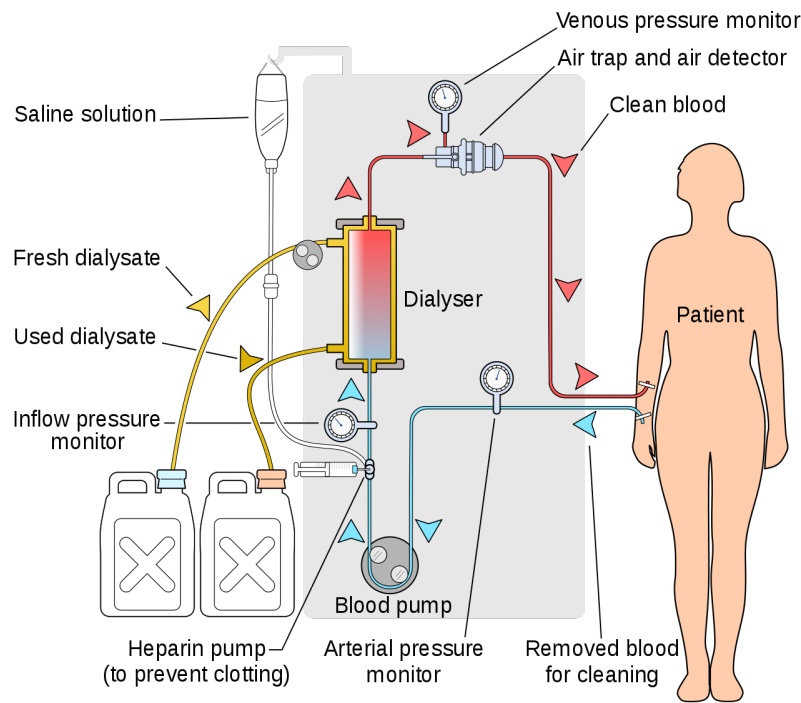
### Types

There are two primary and three secondary types of dialysis, namely:

### PRIMARY DIALYSIS

**-Hemodialysis:** Hemodialysis removes wastes and water by circulating blood outside the body through an external filter, called a dialyzer, that contains a semipermeable membrane. The blood flows in one direction and the dialysate flows in the opposite. The counter-current flow of the blood and dialysate maximizes the concentration gradient of solutes between the blood and dialysate, which helps to remove more urea and creatinine from the blood. The concentrations of solutes normally found in the urine (for

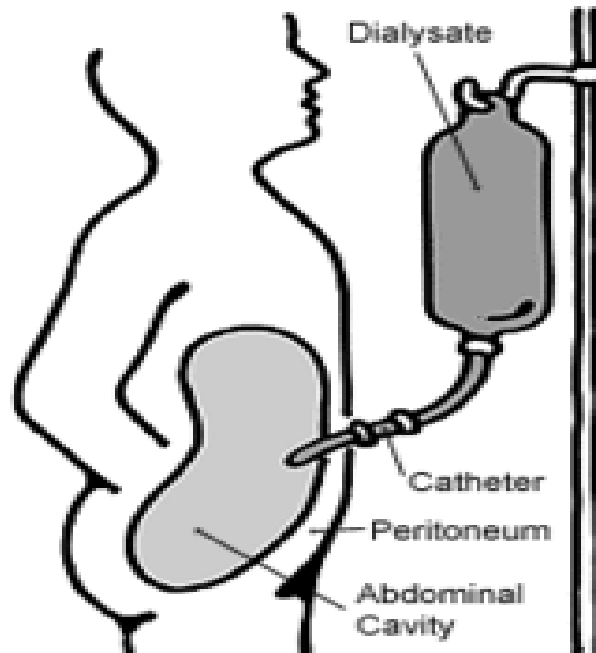
example potassium, phosphorus and urea) are undesirably high in the blood, but low or absent in the dialysis solution, and constant replacement of the dialysate ensures that the concentration of undesired solutes is kept low on this side of the membrane. The dialysis solution has levels of minerals like potassium and calcium that are similar to their natural concentration in healthy blood.



In hemodialysis, the patient's blood is pumped through the blood compartment of a dialyzer, exposing it to a partially permeable membrane. The dialyzer is composed of thousands of tiny hollow synthetic fibers. The fiber wall acts as the semipermeable membrane. Blood flows through the fibers, dialysis solution flows around the outside of the fibers, and water and wastes move between these two solutions. The cleansed blood is then returned via the circuit back to the body. Ultrafiltration occurs by increasing the hydrostatic pressure across the dialyzer membrane. This usually is done by applying a negative pressure to the dialysate compartment of the dialyzer. This pressure gradient causes water and dissolved solutes to move from blood to dialysate and allows the removal of several litres of excess fluid during a typical 4-8 hour treatment, 5 to 7 times a week.

**-Peritoneal dialysis:** In peritoneal dialysis, wastes and water are removed from the blood inside the body using the peritoneum as a natural semipermeable membrane. Wastes and excess water move from the blood, across the peritoneal membrane and into a

special dialysis solution, called dialysate, in the abdominal cavity.



In peritoneal dialysis, a sterile solution containing glucose (called dialysate) is run through a tube into the peritoneal cavity, the abdominal body cavity around the intestine, where the peritoneal membrane acts as a partially permeable membrane.

This exchange is repeated 4–5 times per day; automatic systems can run more frequent exchange cycles overnight. Peritoneal dialysis is less efficient than hemodialysis, but because it is carried out for a longer period of time the net effect in terms of removal of waste products and of salt and water are similar to hemodialysis. Peritoneal dialysis is carried out at home by the patient, often without help. Peritoneal dialysis can be performed with little to no specialized equipment (other than bags of fresh dialysate).

## **SECONDARY DIALYSIS**

**-Hemofiltration:** Hemofiltration is a similar treatment to hemodialysis, but it makes use of a different principle. The blood is pumped through a dialyzer or "hemofilter" as in dialysis, but no dialysate is used. A pressure gradient is applied; as a result, water moves across the very permeable membrane rapidly, "dragging" along with it many dissolved substances, including ones with large molecular weights, which are not



cleared as well by hemodialysis. Salts and water lost from the blood during this process are replaced with a "substitution fluid" that is infused into the extracorporeal circuit during the treatment.

**-Hemodiafiltration:** Hemodiafiltration is a combination of hemodialysis and hemofiltration, thus used to purify the blood from toxins when the kidney is not working normally and also used to treat acute kidney injury

**-Intestinal dialysis:** In intestinal dialysis, the diet is supplemented with soluble fibres such as acacia fibre, which is digested by bacteria in the colon. This bacterial growth increases the amount of nitrogen that is eliminated in fecal waste. An alternative approach utilizes the ingestion of 1 to 1.5 liters of non-absorbable solutions of polyethylene glycol or mannitol every fourth hour.