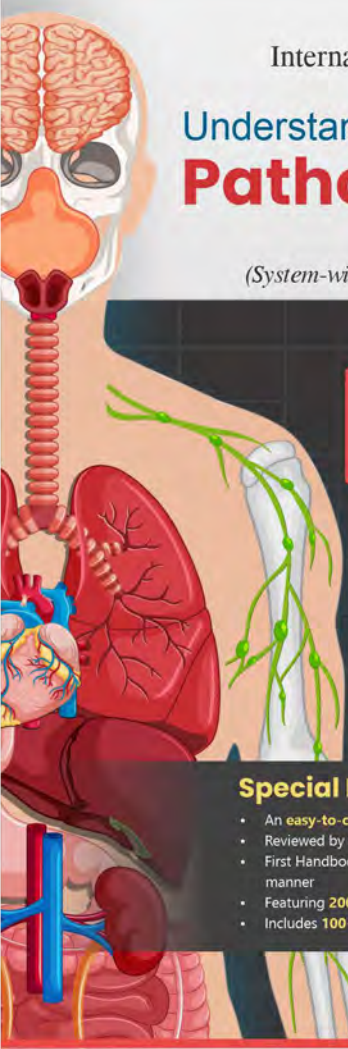
International Standard Colored Edition

Understanding Pathophysiology of Diseases

(System-wise disorders as per INC Syllabus)



Special Features

- An **easy-to-carry** compendium
- Reviewed by the Top **Nursing Faculties/Luminaries PAN India**
- First Handbook on **Pathophysiology** of all the diseases in system-wise manner
- Featuring **200+** flowcharts, line arts, and real-time photographs
- Includes **100+** pathophysiology of diseases

Kanika Rai

Foreword

Sukhpal Kaur

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Understanding
Pathophysiology
of Diseases

(System-wise disorders as per INC Syllabus)

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Preface

Pathophysiology is an essential topic for the nurses to understand. The essence of critical thinking is the ability of nurses to act according to their knowledge and understanding. Because of this reason, the nursing students as well as nurses should understand the pathophysiological changes that take place in any disease. This helps them to think critically and apply that knowledge while taking care of the patients. Considering the importance of learning and understanding the concepts related to pathophysiology, it is an honest effort to bring out a compact but an enriched content for making the nursing students well-versed with the changes that occur in various disease conditions.

Pathophysiology helps build a strong foundation of nursing practice enabling nurses to provide quality care. The book has been organized into 17 chapters divided in accordance with the curriculum given by Indian Nursing Council, parallel to the chapters of Medical Surgical Nursing. The content has been designed in an interesting manner with the help of illustrations, flowcharts and figures for a better understanding of pathophysiological concepts.

The motivation behind writing this book is my own interest in the pathophysiological concepts as I am specialized in Medical Surgical Nursing. Moreover, I have observed the students facing issues regarding an organized content related to pathophysiology and searching from multiple sources. I am pretty confident that not only the nursing students and nurses, but the undergraduate students of other medical disciplines will also be benefitted from the content presented in this book.

I sincerely thank the Almighty for His blessings and all near and dear ones for their constant support and motivation at the time of writing this book.

Kanika Rai

Special Features of the Book

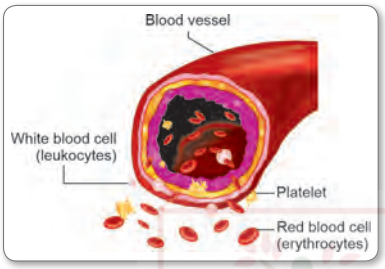
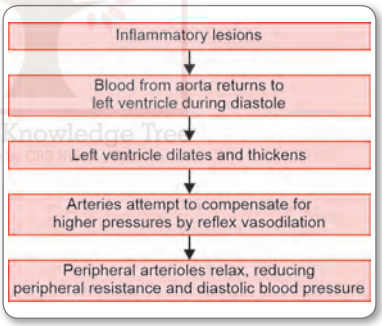


Fig. 1: Components of blood

Self-explanatory **images** included to enhance understanding of topic

Flowchart



Contains **flowcharts** to make the pathophysiological concepts more clear and easy to grasp

Table 1: Differences between rheumatoid arthritis and osteoarthritis

Parameter	Rheumatoid arthritis	Osteoarthritis
Age at onset	Young to middle age	Usually >40 years of age
Nodules	Present, especially on extensor surfaces	Heberden's (DIPs) and Bouchard's (PIPs) nodes

Useful **tables** are included from clinical and diagnosis Point of View

SARS CORONAVIRUS-2

The Severe Acute Respiratory Syndrome (SARS) coronavirus-2 is a novel coronavirus belonging to the family Coronaviridae. It is known to be responsible for the outbreak of a series of recent acute atypical respiratory infections originating in Wuhan, China. The disease caused by this virus, termed coronavirus disease 19 or simply COVID-19, rapidly spread throughout the world at an alarming pace and was declared a pandemic by the World Health Organization (WHO) on March 11, 2020. Let us discuss the pathophysiology of Covid-19 and its different stages.

A highly relevant topic on **SARS CORONAVIRUS-2** Pathophysiology has been included to keep the readers abreast of the various aspects of this deadly disease

Also Know

If the fibrous cap is **thick**, it can resist the stress from blood flow and vessel movement. If the fibrous cap is thin, lipid core may grow causing it to rupture and hemorrhage into plaque allowing a thrombus to form. This is called atherothrombosis.

Also Know boxes have been included in between the text to enhance the knowledge of the readers

Contents

SARS Coronavirus-2

The Severe Acute Respiratory Syndrome (SARS) Coronavirus-2 is a novel coronavirus belonging to the family Coronaviridae. It is known to be responsible for the outbreak of a series of recent acute atypical respiratory infections originating in Wuhan, China. The disease caused by this virus, termed coronavirus disease 19 or simply COVID-19, rapidly spread throughout the world at an alarming pace and was declared a pandemic by the World Health Organization (WHO) on March 11, 2020. Let us discuss the pathophysiology of Covid-19 and its different stages.

In the asymptomatic phase, SARS-CoV-2, received via respiratory aerosols, binds to the nasal epithelial cells in the upper respiratory tract. Local replication and propagation of virus occur in the conducting airways along with an infection of the ciliated cells. The duration of this stage is around two days with a limited immune response. The individuals tend to be highly infectious at this stage despite having a low viral load.

In the next stage, the virus migrates to the upper respiratory tract from the nasal epithelium through the conducting airways. With the involvement of upper airways, manifestations, like fever, dry cough and malaise appear. The immune response in this stage is very high involving the release of C-X-C motif chemokine ligand 10 (CXCL-10) and interferons (IFN-β and IFN-λ) from the cells infected by the virus. This mounted immune response is enough to contain the spread of infection.

About 1/5th of infected patients progress to the next stage in which there occurs an involvement of lower respiratory tract with an onset of acute respiratory distress syndrome (ARDS). The virus is able to invade the type-2 alveolar epithelial cells and starts replicating. The virus-laden pneumocytes start releasing different cytokines and inflammatory markers such as interleukins (IL-1, IL-6, IL-8, IL-120 and IL-12), tumor necrosis factor-α (TNF-α), IFN-λ and IFN-β, CXCL-10, monocyte chemoattractant protein-1 (MCP-1) and macrophage inflammatory protein-1α (MIP-1α). Owing to this cytokine storm, neutrophils, CD4 helper T cells and CD8 cytotoxic T cells begin to get sequestered in the lung tissues, leading to inflammation and injury to lung tissues. The persistent injury caused by the sequestered inflammatory cells and viral replication lead to loss of both types 1 and 2 pneumocytis. This diffuses alveolar damage ultimately culminated into ARDS.

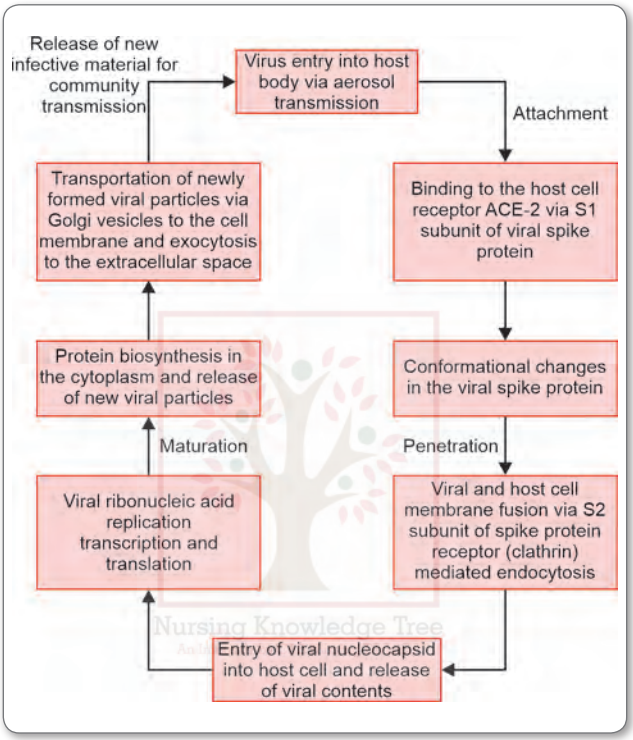


Fig. 1: The severe acute respiratory syndrome coronavirus-2 life cycle

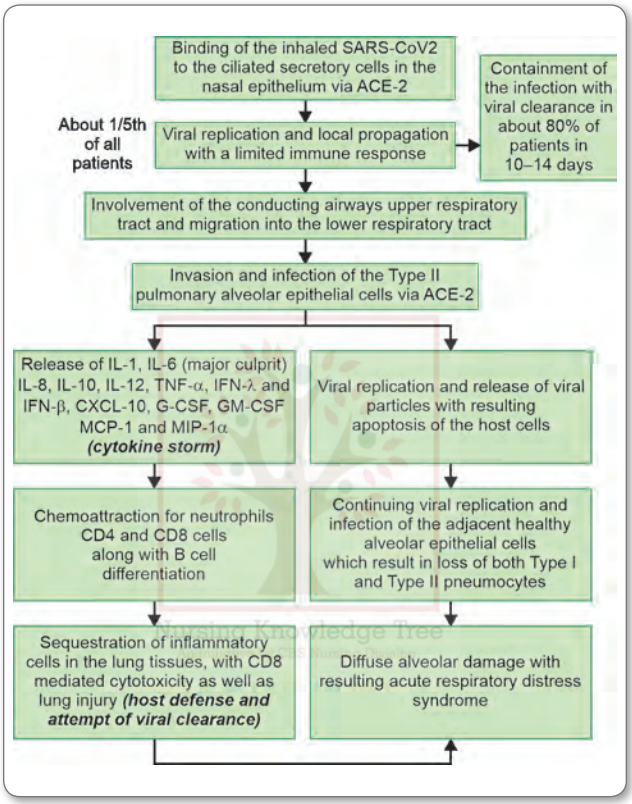
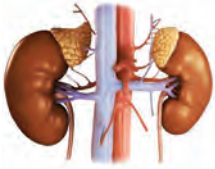


Fig. 2: Pathophysiology of COVID-19

Abbreviations: CXCL-10, C-X-C motif chemokine ligand 10; G-CSF, granulocyte colony-stimulating factor; GM-CSF, granulocyte-macrophage colony-stimulating factor; IFN, interferon; IL, interleukin; MCP-1, monocyte chemoattractant protein-1; MIP-1α, macrophage inflammatory protein-1α; SARS-CoV-2, severe acute respiratory syndrome coronavirus-2; TNF-α, tumor necrosis factor-α



CHAPTER

6

Pathophysiology of Renal System

INTRODUCTION

The renal or genitourinary system functions to remove the excess amount of fluid and toxic waste products from the bloodstream. The main functions of the renal system are to excrete out the wastes from the body, thereby, regulating the volume and pressure of blood. This system also maintains the electrolyte concentration and regulation of blood pH level (Fig. 1).

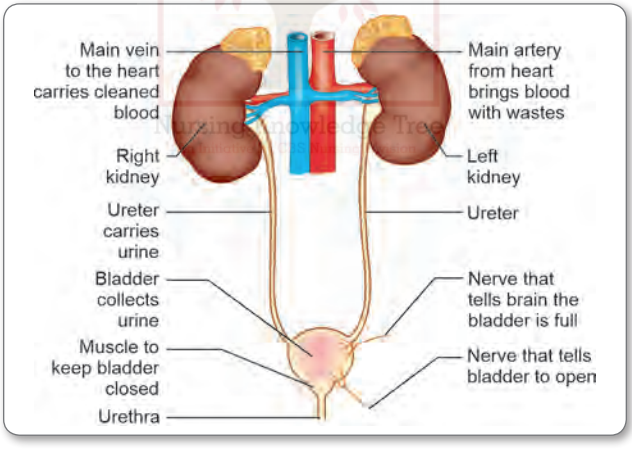


Fig. 1: Parts of renal system

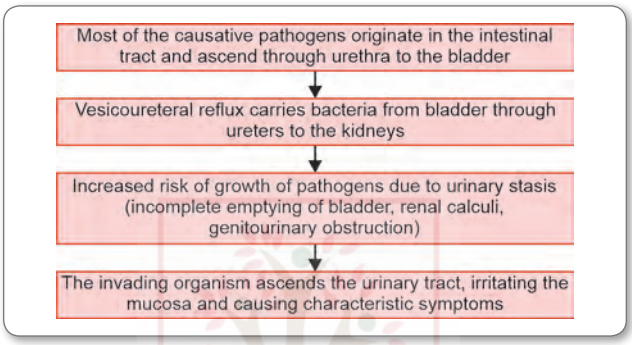
Let us discuss about the pathophysiological changes occurring in various disorders related to renal system.

URINARY TRACT INFECTIONS

Infections mostly occur when bacteria gain access to the bladder, get attached and colonized in the epithelium of the urinary tract so that

they don't get washed off with voiding, and are able to evade host defense mechanisms, and initiate inflammation.

A large number of bacteria can be cleared off from the bladder by increasing the slow shedding of epithelial cells lining the bladder.



CYSTITIS

Acute or chronic inflammation of urinary bladder is termed as Cystitis. The bladder serves as a storage/reservoir for urine and also is lined with mucus membrane having a protein layer above it, that makes it resistant to infection. But the infection of the bladder occasionally is the result of infection of neighboring areas as vagina, urethra and kidneys in females and urethra and prostate gland in males. Obstruction, tumors, traumatic injury or stones in bladder also make it susceptible to infection. Acute cystitis mostly occurs in the course of urinary tract infection whereas chronic cystitis is a recurrent or persistent inflammation of the bladder. Pathophysiology of cystitis is depicted in Figure 2.

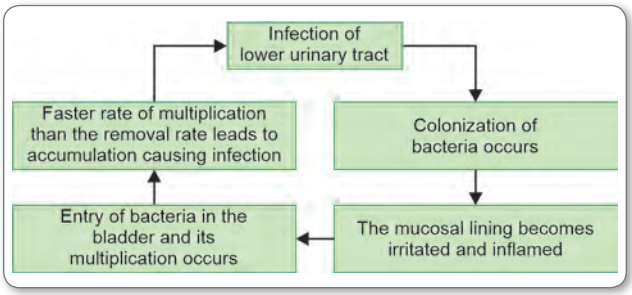


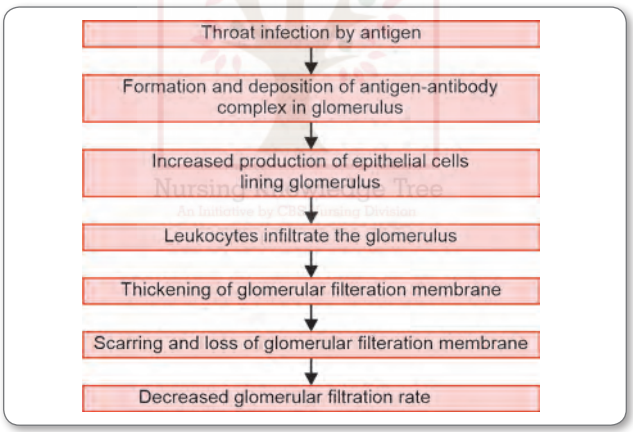
Fig. 2: Pathophysiology of cystitis

GLOMERULONEPHRITIS

An immune-mediated injury acts as a triggering factor for the occurrence of glomerulonephritis. With an onset of immune response, lymphocytes and macrophages infiltrate the glomeruli. The formation of immune deposit occurs in the glomerulus membrane. The circulating antibodies also become trapped in the glomerulus and get deposited there.

Acute Glomerulonephritis

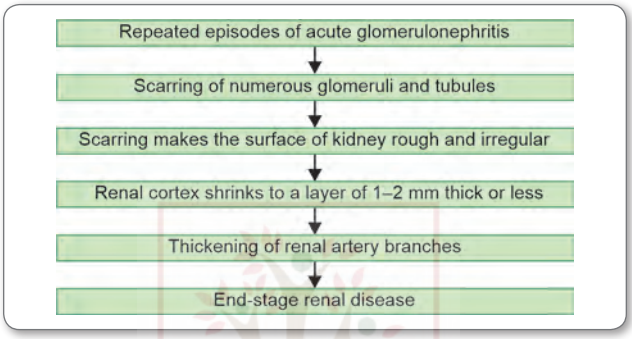
Acute glomerulonephritis is mainly characterized by severe inflammation, renal insufficiency, swelling, increased blood pressure, and severe back pain. Kidneys get damaged because of infection and may progress on to subacute and chronic stages. Swelling of the kidneys occurs in the acute form of disease, the surface becomes smooth and grey and the capsule covering the kidneys is stretched.



Chronic Glomerulonephritis

The repeated episodes of acute glomerulonephritis lead to occurrence of chronic glomerulonephritis, hypertensive nephrosclerosis, hyperlipidemia, chronic tubulointerstitial injury, or hemodynamically medicated glomerular sclerosis. The kidneys are reduced to as little as one-fifth their normal size (consisting primarily the fibrous tissue). The cortex shrinks, making the surface of the kidney rough and irregular. Numerous glomeruli and their tubules become scarred, and the branches of the renal artery are thickened.

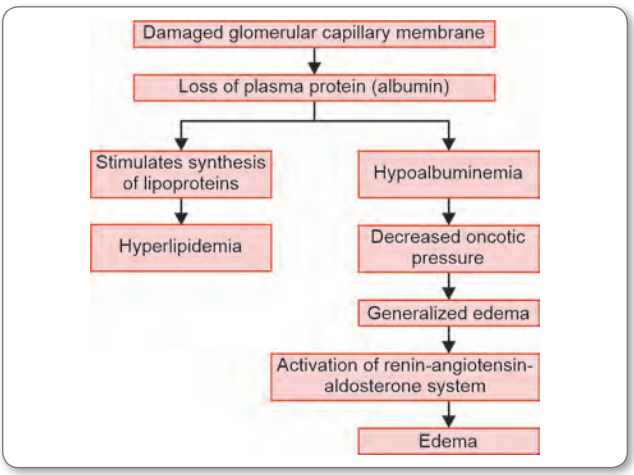
The result is severe glomerular damage that results in end-stage renal disease (ESRD). Failure of kidneys to filter the waste products from the blood and accumulation of abnormal quantities of nitrogenous waste products is known as uremia.



NEPHROTIC SYNDROME

Nephrotic syndrome results from the disease conditions that primarily affect the glomerulus, chronic glomerulonephritis, systemic lupus erythematosus and renal vein thrombosis being the main conditions.

Nephrotic syndrome is characterized by the loss of plasma protein in the urine. Hypoalbuminemia eventually results with an inability of liver to balance the loss of albumin through the kidneys.



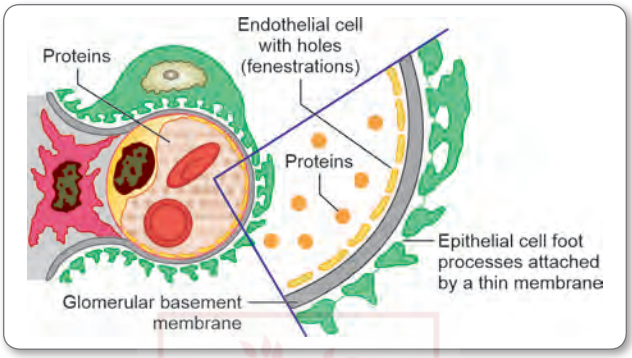
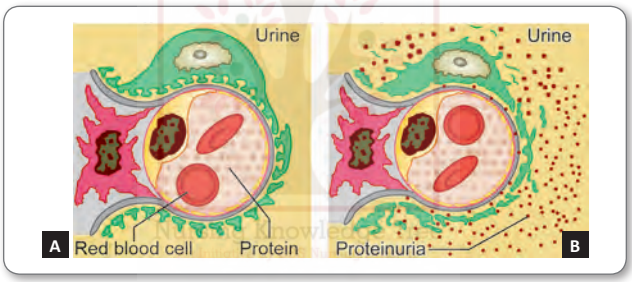


Fig. 3: Barriers that keep protein and blood cells out of the urine



Figs 4A and B: **A.** Normal glomerular capillary; **B.** Capillary with proteinuria

RENAL CALCULI

The stones may be formed anywhere in the upper or lower urinary tract. Increased concentration of calcium oxalate, calcium phosphate, and uric acid whereas decrease in concentration of citrate, magnesium, nephrocalcin, and uropontin favors the formation of calculi.

Other factors contributing to the formation of stones include dehydration, infection, urinary stasis and episodes of immobility (Fig. 5).

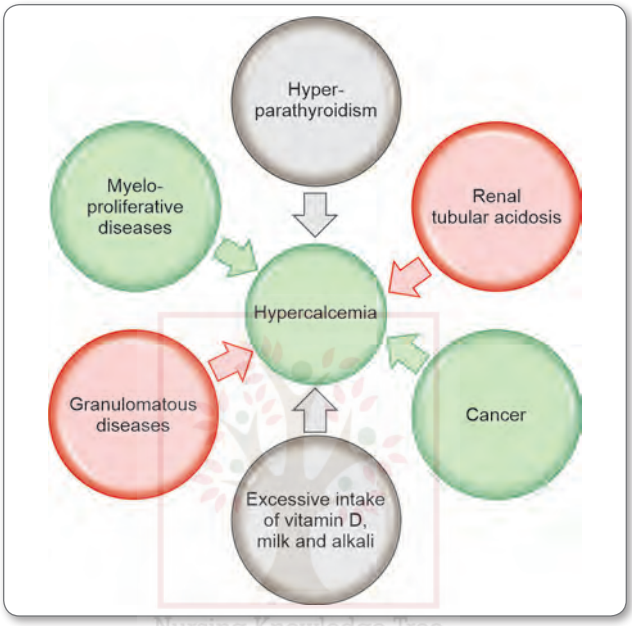
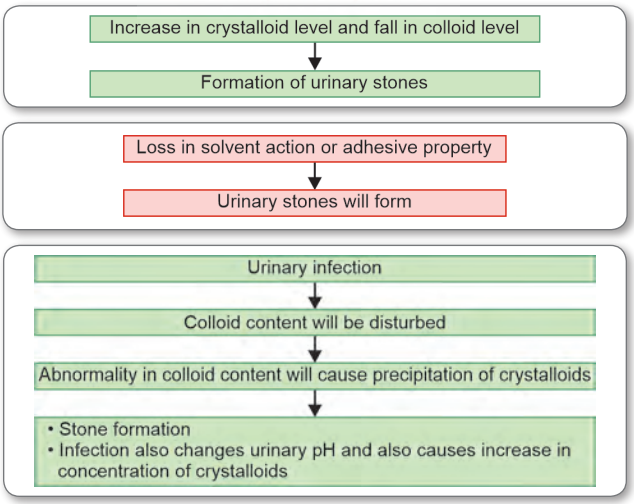
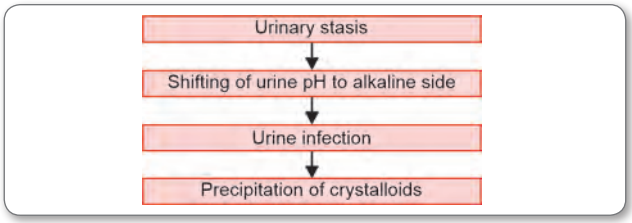
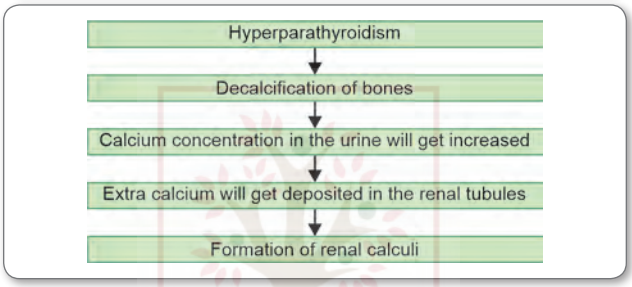


Fig. 5: Causes of hypercalcemia







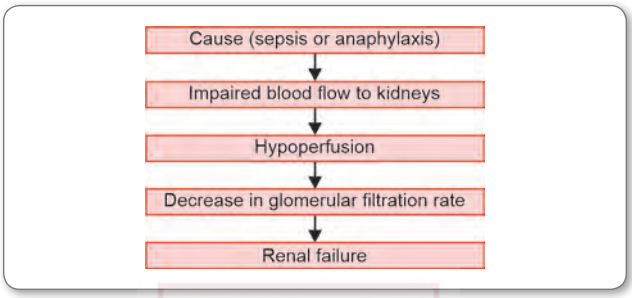
ACUTE RENAL FAILURE

Acute renal failure (ARF) is a sudden and nearly complete damage of kidney function. Oliguria (<400 mL/day of urine) is the most common manifestation seen in ARF; whereas anuria (<50 mL/day of urine) and normal urine output occur rarely. Even if the patient excretes normal urine output that normally doesn't occur, there will be an increase in the level of blood urea nitrogen (BUN) and serum creatinine including retention of other metabolic waste products.

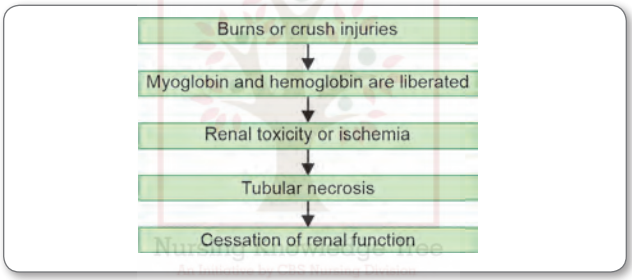
The kidneys require an adequate blood supply, properly functioning glomeruli and renal capillaries and a normal elimination of urine from the body. Any interruption of these processes will lead to the occurrence of acute renal failure.

The classification of causes is in accordance with the disorders leading to disruption of these processes. Functional and structural causes that prevent a smooth supply of blood to kidneys are classified as prerenal. These include extracellular fluid (ECF) volume contraction, congestive heart failure (functional) and renal artery stenosis (structural). Diseases causing actual damage to the kidneys or any associated structure, are categorized as intrarenal causes like acute glomerulonephritis, acute tubular necrosis etc. The conditions causing an interference with normal drainage and excretion of urine are classified as postrenal, i.e., benign prostate hypertrophy or tumor of the prostate.

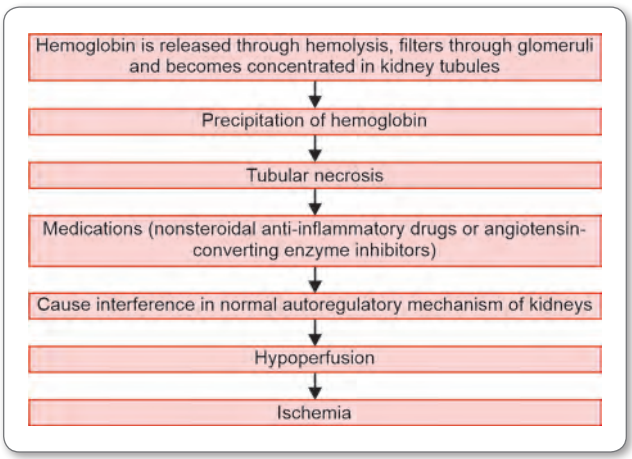
- Prerenal



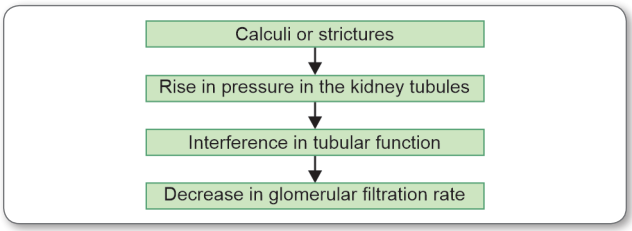
- Intrinsic



- Transfusion reaction



- Postrenal



Phases of ARF

- **Initiation:** Begins with the injury of kidneys and ends with oliguria development.
- **Oliguria:** This period is accompanied by a rise in serum concentration of substances normally excreted by kidneys (urea, creatinine, uric acid, organic acids, intracellular cations). Hyperkalemia may also develop.
- **Diuresis:** Patient experiences gradually increasing urine output signaling that glomerular filtration has started to recover. Lab values start decreasing. Renal function may still be abnormal. Observe for dehydration during this period.
- **Recovery period:** It signals the improvement of renal function and may take 3–12 months.

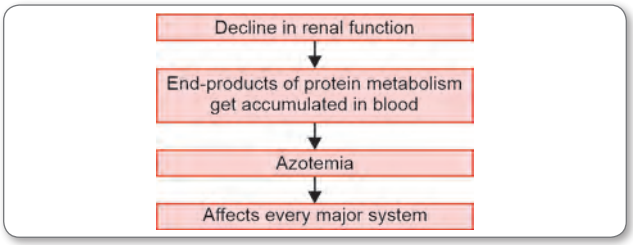
CHRONIC RENAL FAILURE

Chronic renal failure also known as end-stage renal failure or ESRD is a progressive, irreversible deterioration in renal function in which the body's ability to maintain metabolic and fluid and electrolyte balance fails, resulting in uremia.

With the decline in renal function, the metabolic waste products start accumulating in the blood. Increase in blood urea nitrogen levels (uremia) results from this accumulation affecting every major system of the body. The severity of symptoms depends upon the extent of build-up of such waste products. The chronic renal disease is categorized in three stages as reduced renal reserve, renal insufficiency, and ESRD.

Any underlying disease condition, presence of hypertension, loss of protein in urine, all exacerbate the progression of chronic renal failure and increased decline in renal function.

Pathophysiological Changes



Stages of ESRD

- **Reduced renal reserve:** 40–75% loss of nephron function. Patient usually does not have any symptom because remaining nephrons are able to carry out the normal functions of the kidney.
- **Renal insufficiency:** 75–90% of nephron function is lost. At this point, BUN and serum creatinine rise, kidney loses its ability to concentrate urine and anemia develops. Patient may report polyuria and nocturia.
- **ESRD:** It occurs when there is less than 10% nephron function remaining. Evidenced by elevated creatinine and BUN levels as well as electrolyte imbalances.

URETHRAL STRICTURE

A stricture refers to the narrowing of the lumen, which may be congenital or acquired. Urethral strictures arise from various causes and can result in a range of manifestations, from an asymptomatic presentation to severe discomfort secondary to urinary retention.

- Narrowing of the urethra can result from chronic infection that leads to inflammation of mucus membrane.
- The inflammation causes hyperplasia of the lining resulting in the development of stricture.
- Urethral anastomosis can also cause stricture.
- Pressure from a tumor against the exterior of urethra can result in the stricture of the lumen.
- A congenital stricture results from inadequate fusion of the anterior and posterior urethra, is short in length, and is not associated with an inflammatory process.

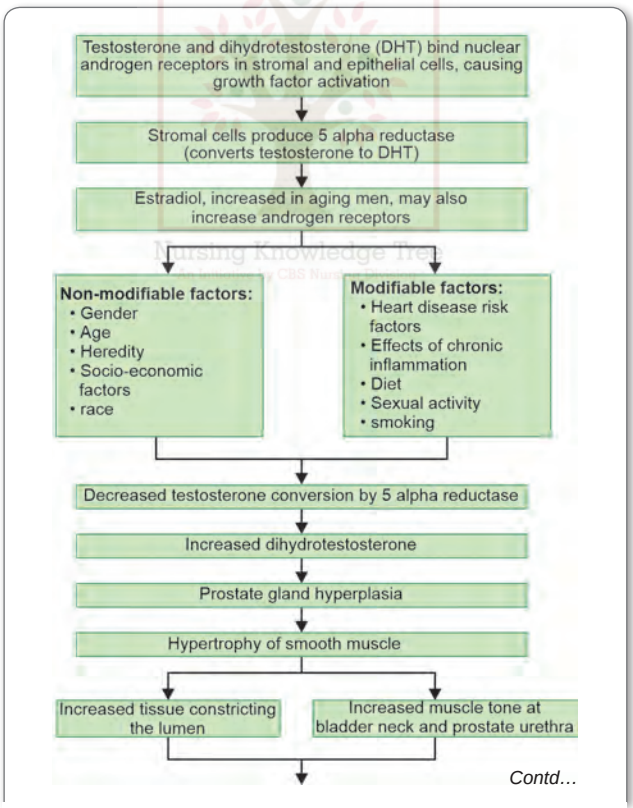
Urethral Stricture in Males

- **Posterior urethral stricture:** Posterior urethral strictures occur due to an injury related to pelvic fracture and is located in the first two inches of the urethra. There is a disruption of the urethra that may either be separated or completely cut due to which urine is not able to pass through.

- **Anterior urethral stricture:** Main causes of anterior urethral stricture that is present in the last two inches of the urethra include direct traumatic injury to penis or catheterization.

BENIGN PROSTATE HYPERPLASIA/HYPERTROPHY

Benign prostatic hyperplasia (BPH) is a proliferative process of cellular elements of prostate, enlarged prostate or a voiding dysfunction that results from enlargement of prostate and bladder obstruction. The dominant role of the androgen system and the androgen receptor is well defined in the pathogenesis of BPH. Androgen receptors are expressed in BPH tissues in which they are activated by the potent androgen dihydrotestosterone. Synthesis of dihydrotestosterone is under control of the 5α-reductase enzyme, activity of which is antagonized by finasteride and dutasteride.



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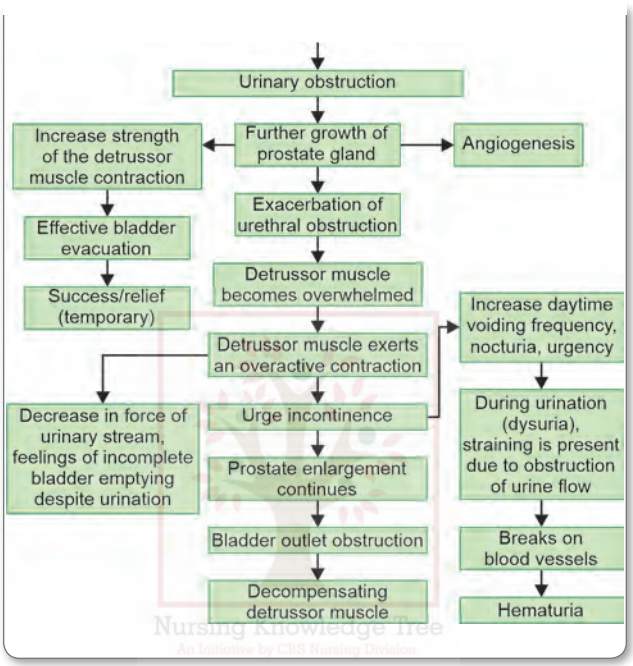
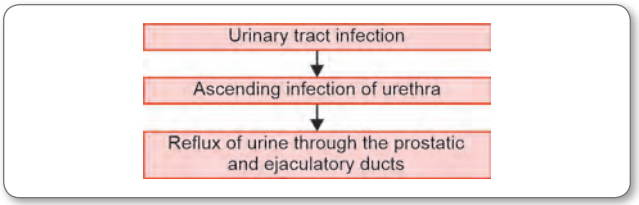


PROSTATITIS

An ascending urethral infection is responsible for causing acute bacterial prostatitis, through direct or lymphatic spread from the rectum or hematogenous spread via bacterial sepsis. Chronic bacterial prostatitis is chronic bacterial infection of the prostate with or without symptoms and also occurs because of inadequate treatment of acute prostatitis.

- **Acute bacterial prostatitis:** Acute prostatitis does not yet have a fully understood pathogenesis.



- **Chronic bacterial prostatitis:** Ascending infection from the distal part of urethra to the prostate is the possible cause of chronic prostatitis. Sometimes, any anatomical abnormality in the intra-prostatic ducts may also be responsible for the retrograde spread of infection.

Understanding **Pathophysiology** of Diseases

Salient Features

- A comprehensive handbook on Pathophysiological changes of various medical and surgical disorders written by an Indian Author
- Intended for undergraduate, postgraduate nursing students and the students from other medical disciplines
- Contains colorful illustrations and flowcharts for an easy understanding of the psychophysiology concept of respective disease condition
- Written parallel to the units of Medical Surgical Nursing as per INC Curriculum
- Text organized into logical sequence for smooth flow of content throughout.

About the Author

Kanika Rai, presently working as a Professor & HOD (Medical Surgical Nursing) at Maharishi Markandeshwar College of Nursing, Maharishi Markandeshwar (deemed to be) University, Mullana, Ambala. She possesses 12 years of teaching experience. She is the founder of Kanika's Nursing Academy, Chandigarh. The author has done her BSc Nursing from NINE, PGIMER, Chandigarh (2007) and MSc Nursing (Cardiovascular and Thoracic Nursing) from Maharishi Markandeshwar College of Nursing, Maharishi Markandeshwar (deemed to be) University, Mullana, Ambala. She is a Gold Medalist in MSc (N). She has completed her PhD Nursing from Chitkara University, Rajpura, Punjab. Besides, she is a member of TNAI, NRSI and Society of Indian Neuroscience Nurses. She has published more than 25 research papers in National and International Journals.

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