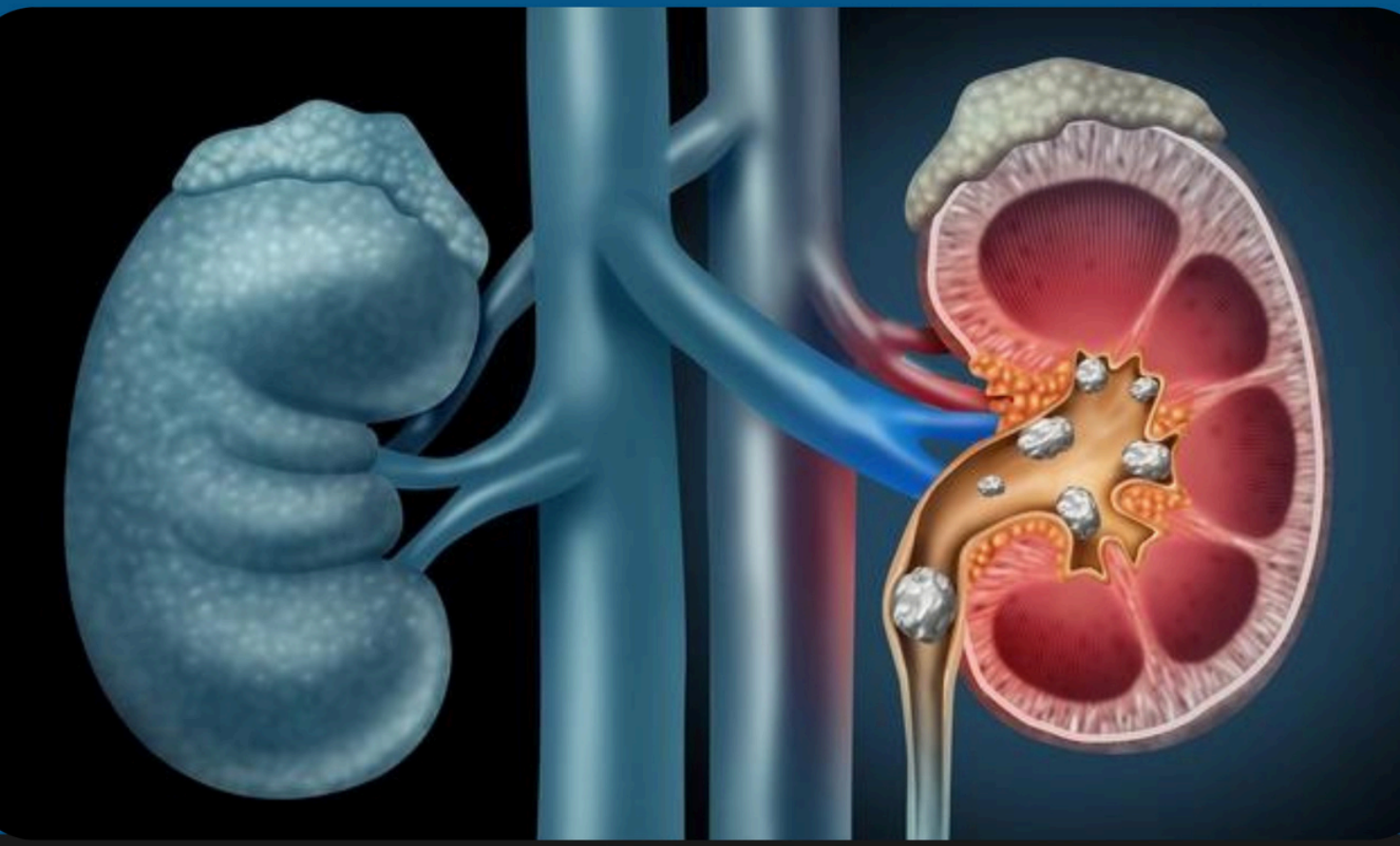
RENAL OR URINARY SYSTEM

ANATOMY, PHYSIOLOGY & PATHOLOGY



TAILORED FOR MEDICAL STUDENTS, USMLE, PLAB, PA & NURSING

4th EDITION

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PDF

131 PAGES

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Table Of Contents:

What's included: Ready-to-study anatomy, physiology and pathology notes of the **Genitourinary System** presented in succinct, intuitive and richly illustrated downloadable PDF documents. Once downloaded, you may choose to either print and bind them, or make annotations digitally on your iPad or tablet PC.

Anatomy & Physiology Notes:

Pathology Notes:

- ELECTROLYTE IMBALANCES
- FLUID IMBALANCES
- DIURETICS
- DRUGS ALTERING THE pH URINE
- POPULATION HEALTH & RENAL DISEASE
- MISCELLANEOUS POINTS
- UROGENIC PAIN
- CATHETERIZATION
- URINE ANALYSIS
- MCQS - URINARY TRACT DISEASE

FUNCTIONAL ANATOMY OF THE URINARY SYSTEM

FUNCTIONAL ANATOMY OF THE URINARY SYSTEM

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Urinary System - General Functions:

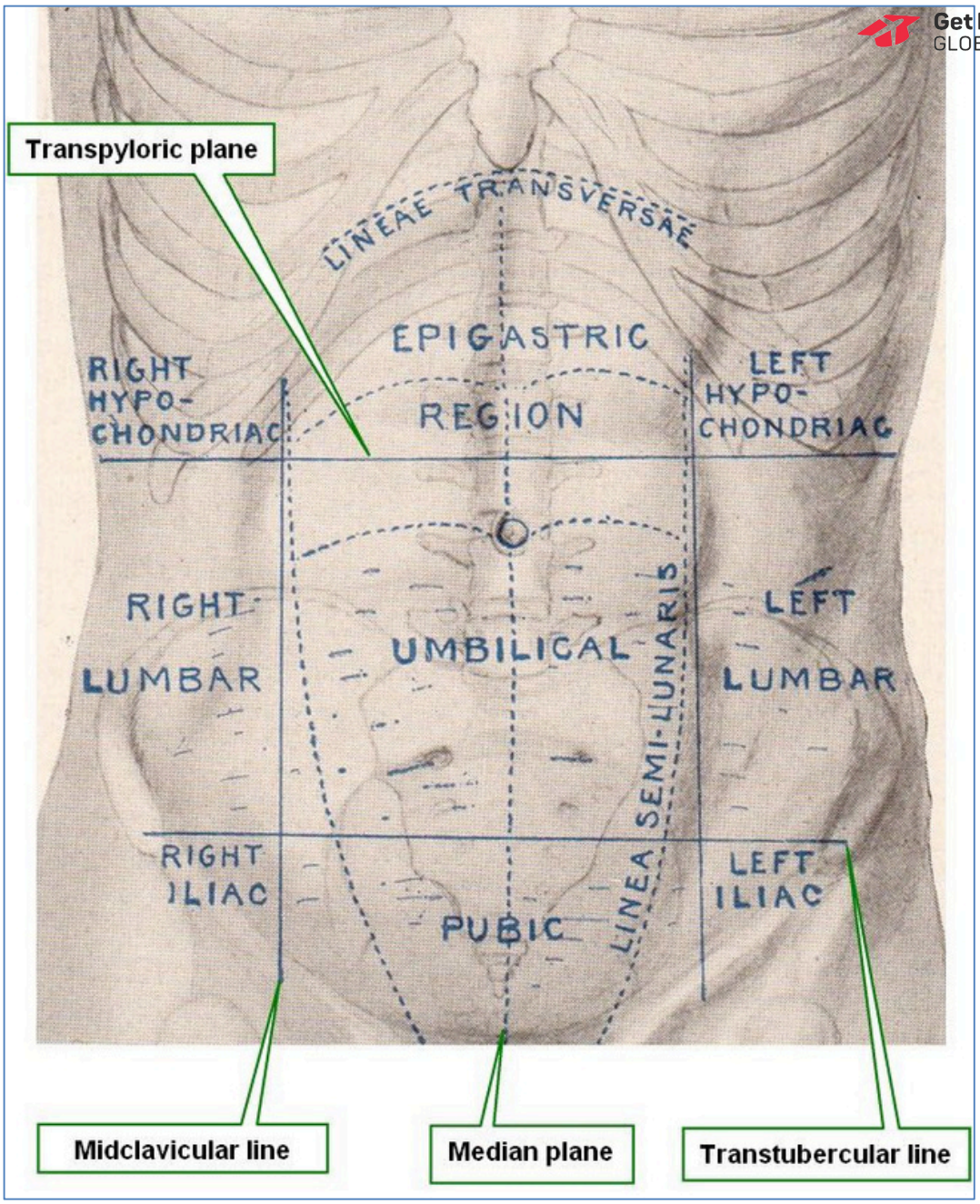
- Filter blood (Through "Ultrafiltration" – A filtration process using a porous membrane to remove
- particles, bacteria & viruses)
- Disposal of Metabolic Wastes & Drugs
- Regulate Water Balance
- Regulate Electrolyte Balance
- Regulate Body Fluid Osmolality & Electrolyte Concentrations
- Store & Eliminate Urine
- Maintain Blood Volume
- Regulate Acid/Base Balance (in Conjunction with Respiratory System)
- Regulate Arterial Blood Pressure
- Reproduction (Males)
- Endocrine Function – Excretion of Hormones
  Gluconeogenesis (Eg: From Amino Acids)

Surface Projections:

- ***Transpyloric Plane*** **(Transverse line @ T12):**
  - o Hilum of L-Kidney
  - o Superior Pole of R-Kidney
- ***Median Line*** **(Midline):**
  - o Hilum of Kidneys ≈ 5cm from Midline
  - o Slightly Splayed Outwards (further from midline at inferior pole)
  - o Ureters ≈ 5cm from Midline
- **Height:**
  - o Kidneys lie just deep to Ribs 11 & 12.
  - o Kidneys move up/down 2-3cm during deep breathing.
  - o Inferior Pole of R-Kidney = a finger's breadth superior to Iliac Crest
- **Right Vs. Left:**
  - o o Left = Higher than Right
  - o (by ≈2.5 cm) o Due to Liver (invades R-Abdomen) o Left Renal Artery – Shorter than Right (as Aorta lies to left of midline) o Left Renal Vein – Longer than Right (as IVC lies to right of midline)

Right = Lower (The Palpable One)

- **Dimensions:**
  - o 12 cm Long o
  - 3-4 cm Thick o
  - 5-6 cm Wide



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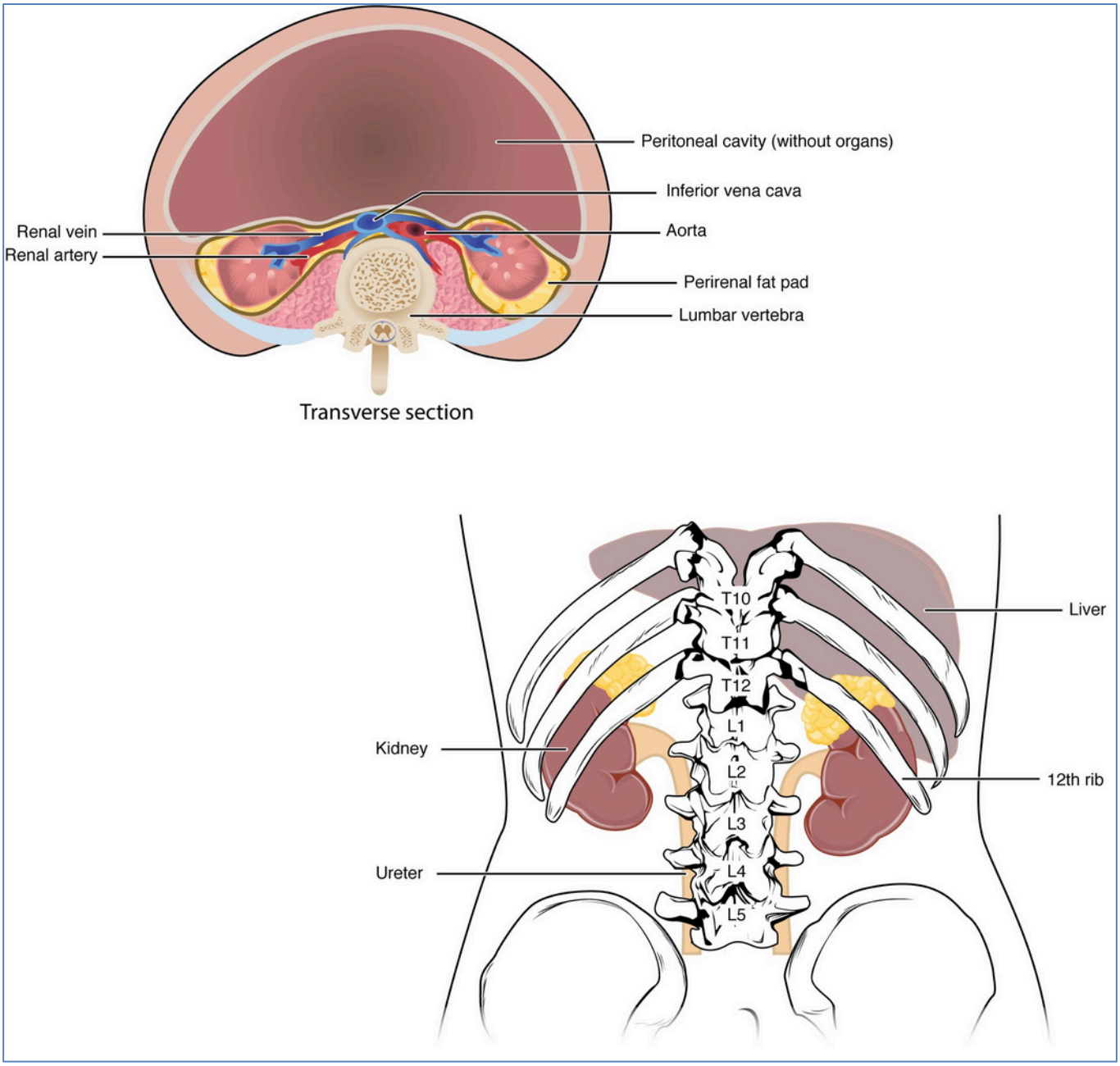
Position of Kidneys Within Abdomen:

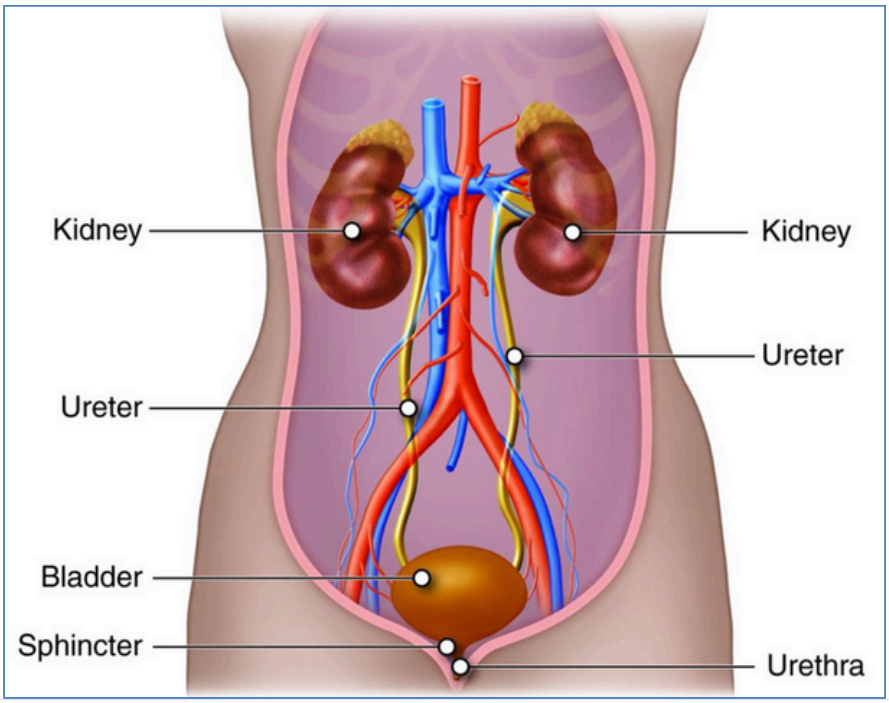
- Retroperitoneal
- Spleen on Lateral Border of L-Kidney
- Adrenal Glands on Superior Poles of Both Kidneys
- Pancreas on Anterior Margin of L-Kidney
- Duodenum on Anterior Margin of R-Kidney
- Liver on Superior Aspects of Both Kidneys
- Ascending Colon Anterior To R-Kidney
- Descending Colon Anterior To L-Kidney



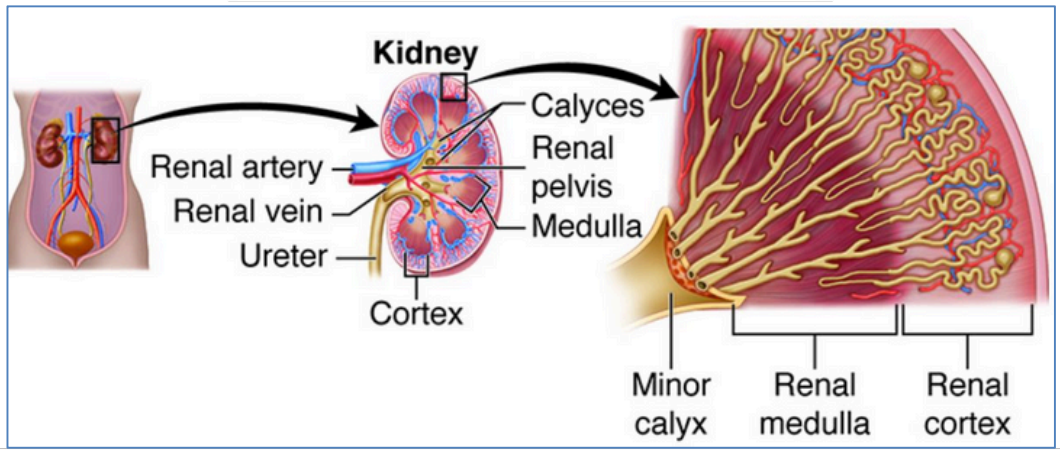
https://open.oregonstate.education/aandp/chapter/25-1-internal-and-external-anatomy-of-the-kidney/

Functional Components:

- **Kidneys:**
  - Filter Blood
  - Produce Urine
  - Blood pH/Volume/Pressure Homeostasis
- **Renal Veins:**
  - Anterior
  - Drain Blood From Kidneys
- **Renal Arteries:**
  - Supply Blood to Kidneys
  - Between Vein & Hilum
- **Renal Hilums ("Opening"):**
  - Beginning of Ureters
  - Posterior
- **Ureters:**
  - Transport Urine → Bladder
- **Bladder:**
  - Stores Urine
- **Urethra:**
  - Excretion of Urine



Urinary system organs. This work by Cenveo is licensed under a Creative Commons Attribution 3.0 United States (http://creativecommons.org/licenses/by/3.0/us/).



Internal structure of kidney. This work by Cenveo is licensed under a Creative Commons Attribution 3.0 United States (http://creativecommons.org/licenses/by/3.0/us/).

M acroscopic Anatom y of Kidneys:

- **Encased In Fascia & Fat:**
- o (Fat – Important in Stabilisation & protection)
- **Renal Capsule:**
  - o Tough, Fibrous layer surrounding the Kidney.

(Outer) Cortex:

- o Contains the Filtering Apparatus:
  - § Blood Vessels
  - § Renal Corpuscles
  - § Renal Tubules (excluding the Loop of Henle – in Medulla)

- **(Inner) Medulla:**
  - o Contains the Major Blood Vessels
  - o Made up of Renal Pyramids & Columns
  - o Contains Collecting Ducts – Deliver Urine to Minor Calyces.
- **Renal Pyramids:**
  - o Cone-shaped tissues
  - o Formed by straight parallel segments of Nephrons.
- **Renal Lobes:**
- o Portion consisting of a Renal Pyramid & the Renal Cortex Above.

Renal Columns:

- o Spaces between Renal Pyramids
- o Contains Interlobar Blood Vessels

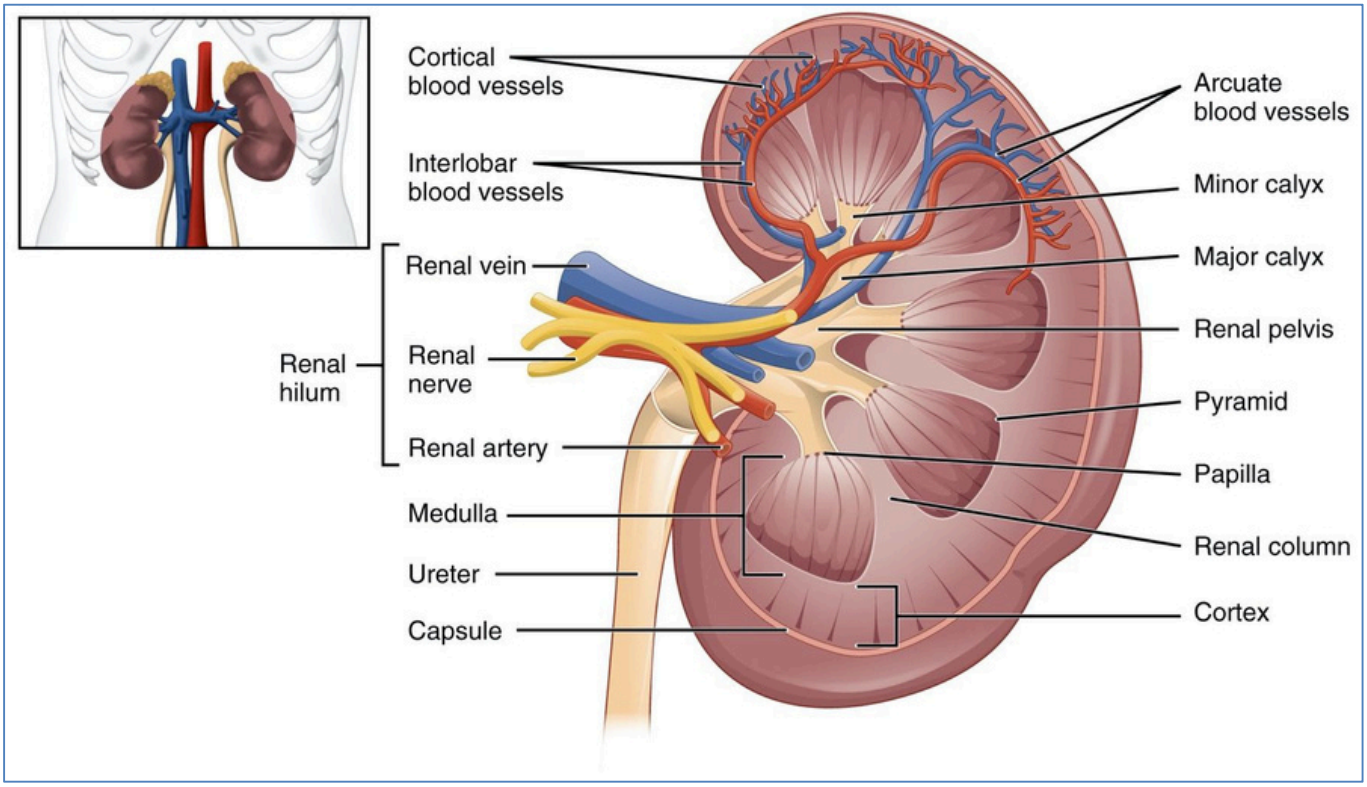
- **Renal Papilla:**
- o Where the Collecting Ducts of the Medullary Pyramids empty Urine into the renal pelvis.
- **Minor Calyx (Calyces):**
- o Transport Urine from Collecting Ducts → Major Calyces

Major Calyx (Calyces):

- o Transport Urine → Renal Pelvis

Renal Pelvis / Hilum:

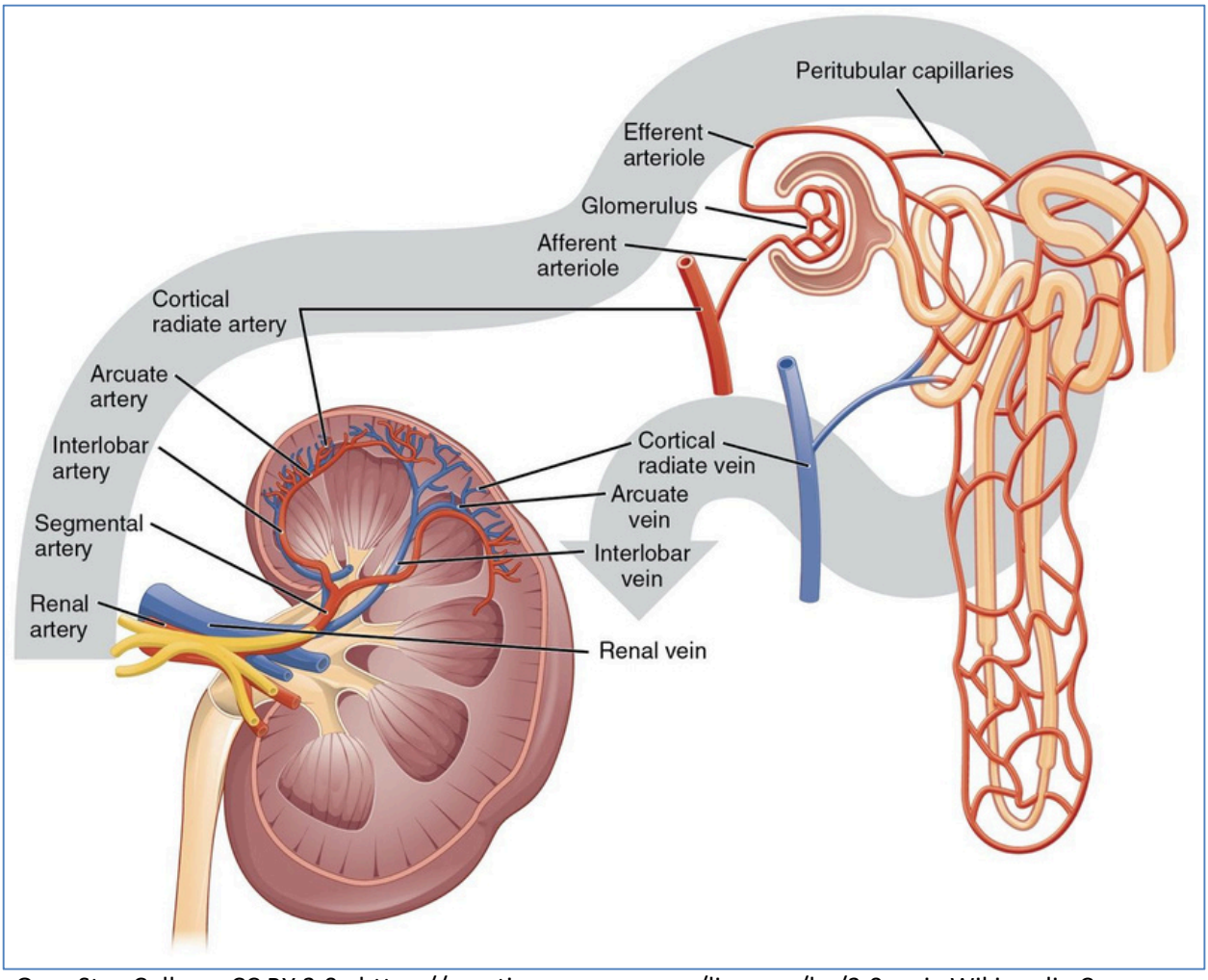
- o Convergence of all Calyces & Connecting Ducts
- o Becomes the Ureter as it Exits the Kidney.



Microscopic Anatomy of Kidneys:

- **Microvascular Supply:**
  - **Interlobar Arteries & Veins:**
    - Run up from the Medulla *Through* the Renal Columns
    - Each form an arc with Interlob*ular* Arteries/Veins.
    - 'horseshoe bends'
  - **Interlobular → Arcuate Arteries/Veins:**
    - Projections of the Interlo*bar* Arteries/Veins into the Cortex.
    - 'little dead-end streets'
  - **Afferent Arterioles:**
    - Carry blood from Interlobar Arteries → Corpuscle of the Nephron
    - 'driveways off little dead-end streets'
  - **Renal Corpuscle:**
    - ***The Glomerular Capillaries + Glomerular Capsule***
    - *Glomerular Capsule* = Little deeply-concaved membrane in which a convoluted mass of
    - *Glomerular Capillaries* are bundled.
    - **Note:** Glomerular Capillaries are *Highly Fenestrated* → 'Leaky' → Aids in filtration. Place of filtration
  - **Efferent Arterioles:**
  - Carry blood away from the Corpuscles → Peritubular Capillaries
  - **Peritubular Capillaries:**
    - Supply the rest of the Nephron (Renal Tubules & Ascending/Descending Limbs)

    Venules:
    - Drain filtered blood back to Inferior Vena Cava.
    - Peritubular Capillaries→ Interlobular Venules → Arcuate Veins → Interlobar Veins → Segmental Veins →Renal Vein → IVC.



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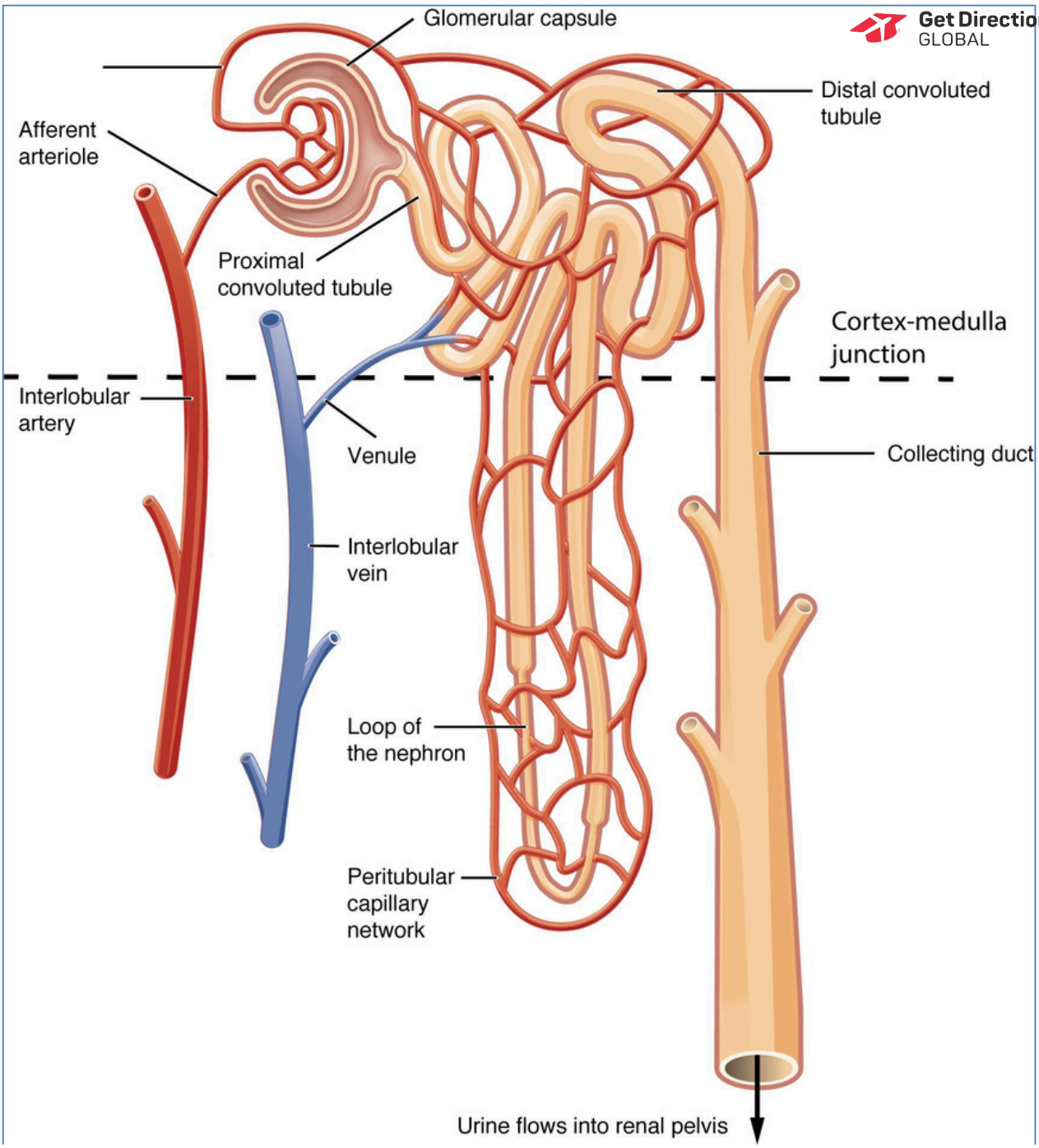

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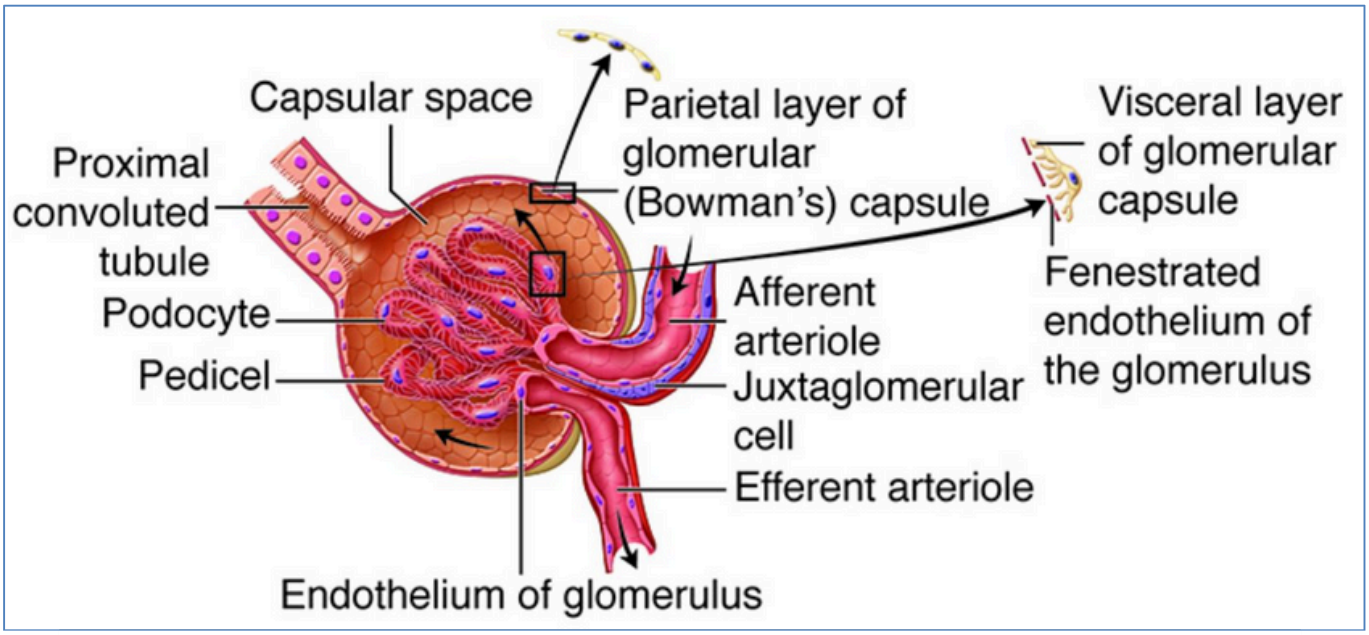
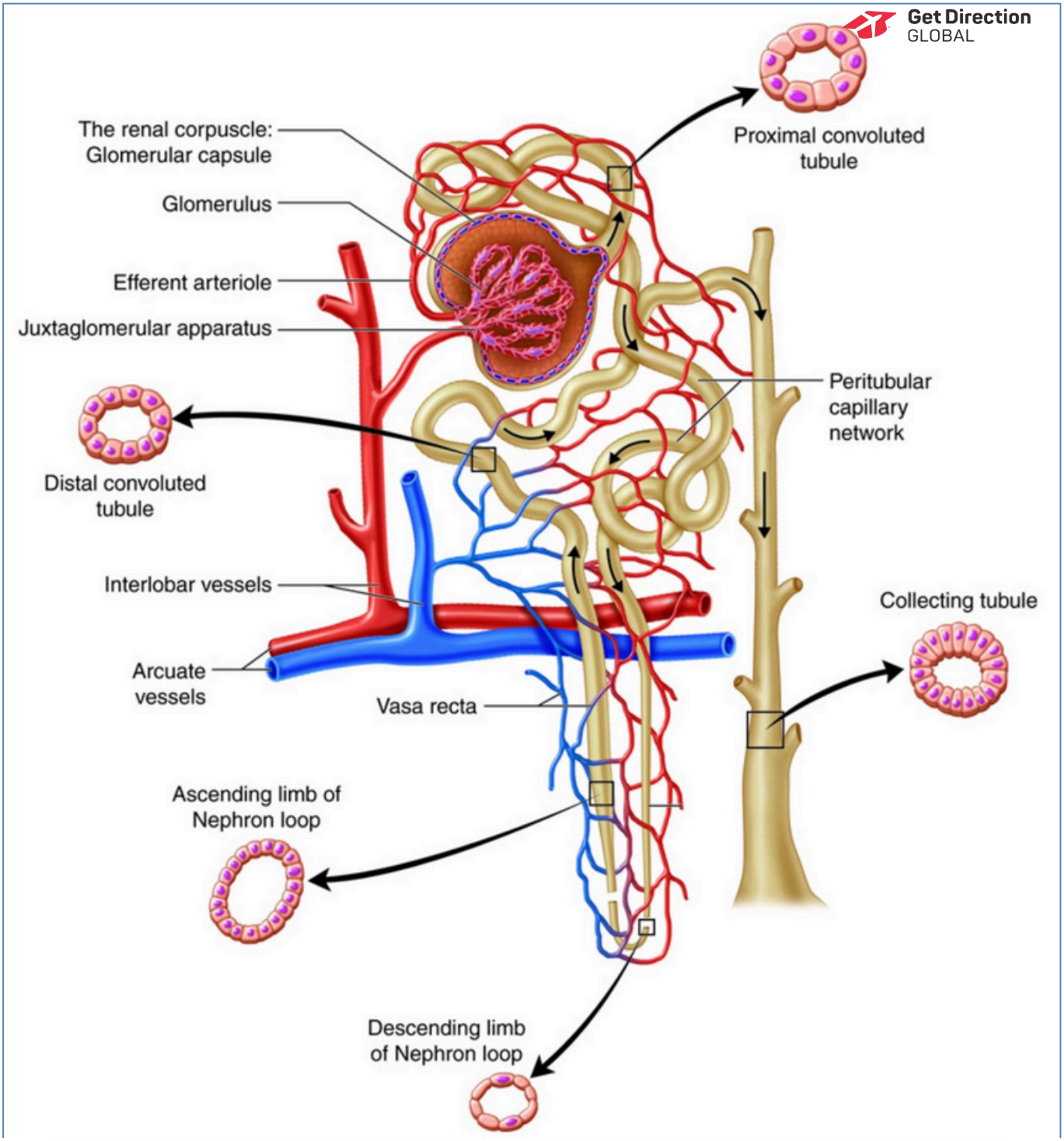
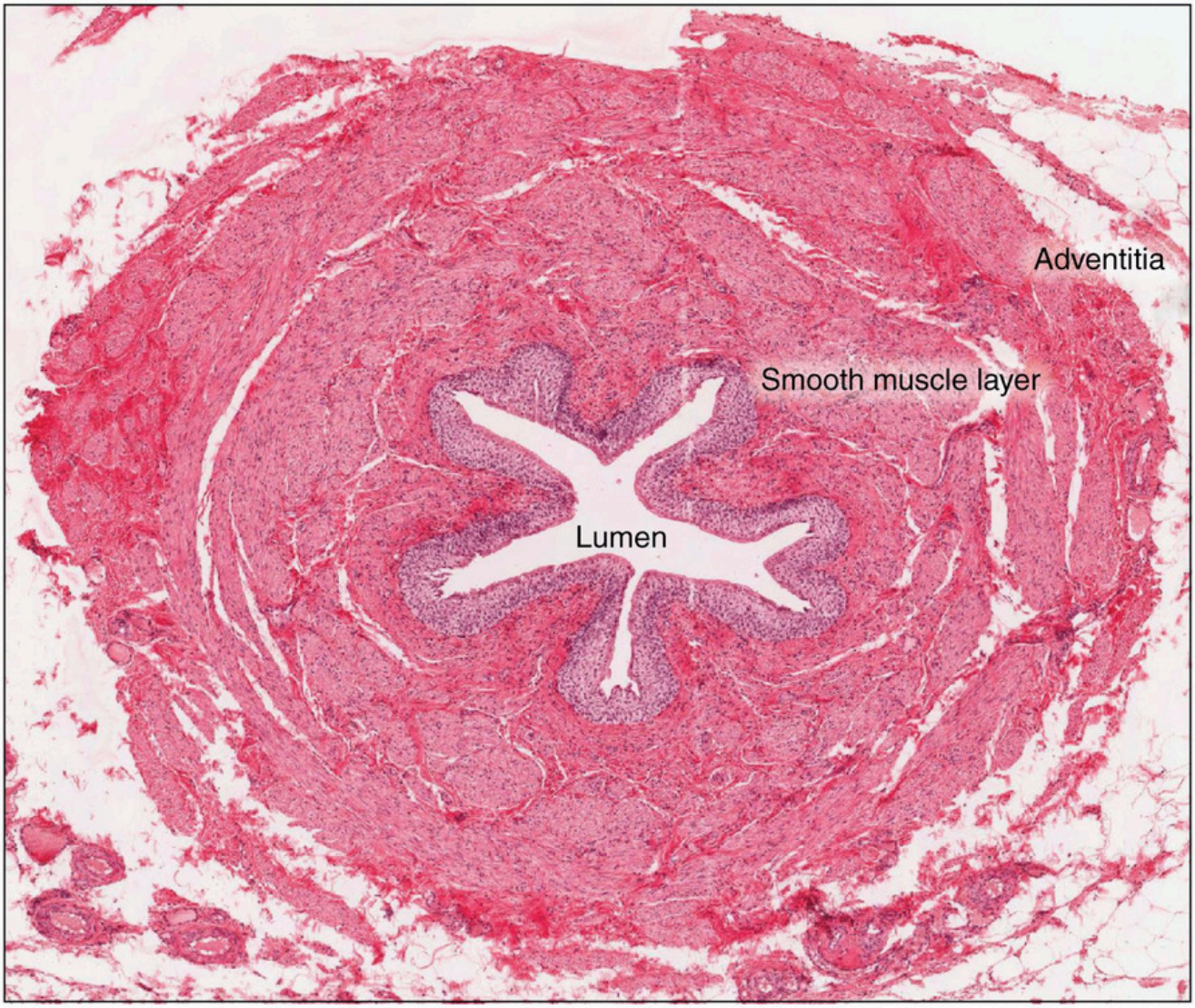
- **The Nephron:**
  - **Renal Corpuscle:**
    - ***The Glomerular Capillaries + Glomerular Capsule***
    - *Glomerular Capsule* = Little deeply-concaved membrane in which a convoluted mass of
    - *Glomerular Capillaries* are bundled.
      Place of filtration
  - **Renal Tubule:**
    - **Proximal Convoluted Tubule:**
      - Reabsorption of Water, ions & Organic Nutrients.
      - ***Histology:*** **Simple Cuboidal Epithelia with Microvilli** for bulk Reabsorption.
    - **Loop of Henle:**
      - **Descending Limb (Thick & Thin):**
        - Further Water Reabsorption
        - ***Histology:*** **Simple Squamous Epithelia** → H2O Reabsorption only.
      - **Ascending Limb (Thin & Thick):**
        - Na+ Reabsorption
        - Cl- Reabsorption
        - ***Histology:*** **Simple Cuboidal Epithelia** → Resorption of Ions.
    - **Distal Convoluted Tubule:**
      - Secretion of Ions, Acids, Drugs & toxins
      - Variable Reabsorption of Water, Na+ & Ca+ ions (under endocrine control)
      - ***Histology:*** **Simple Cuboidal Epithelia (No Microvilli)**→ Resorption of Ions.
  - **Collecting System:**
    - **Collecting Duct:**
      - Variable Reabsorption of Water
      - Reabsorption OR Secretion of Na+, K+, H+ & HCO-3.
      - ***Histology:*** **Simple Cuboidal – Columnar Epithelia** for reabsorption of H2O, Urea & other Ions.
    - **Papillary Duct:**
      - Carries urine to Minor Calyces.

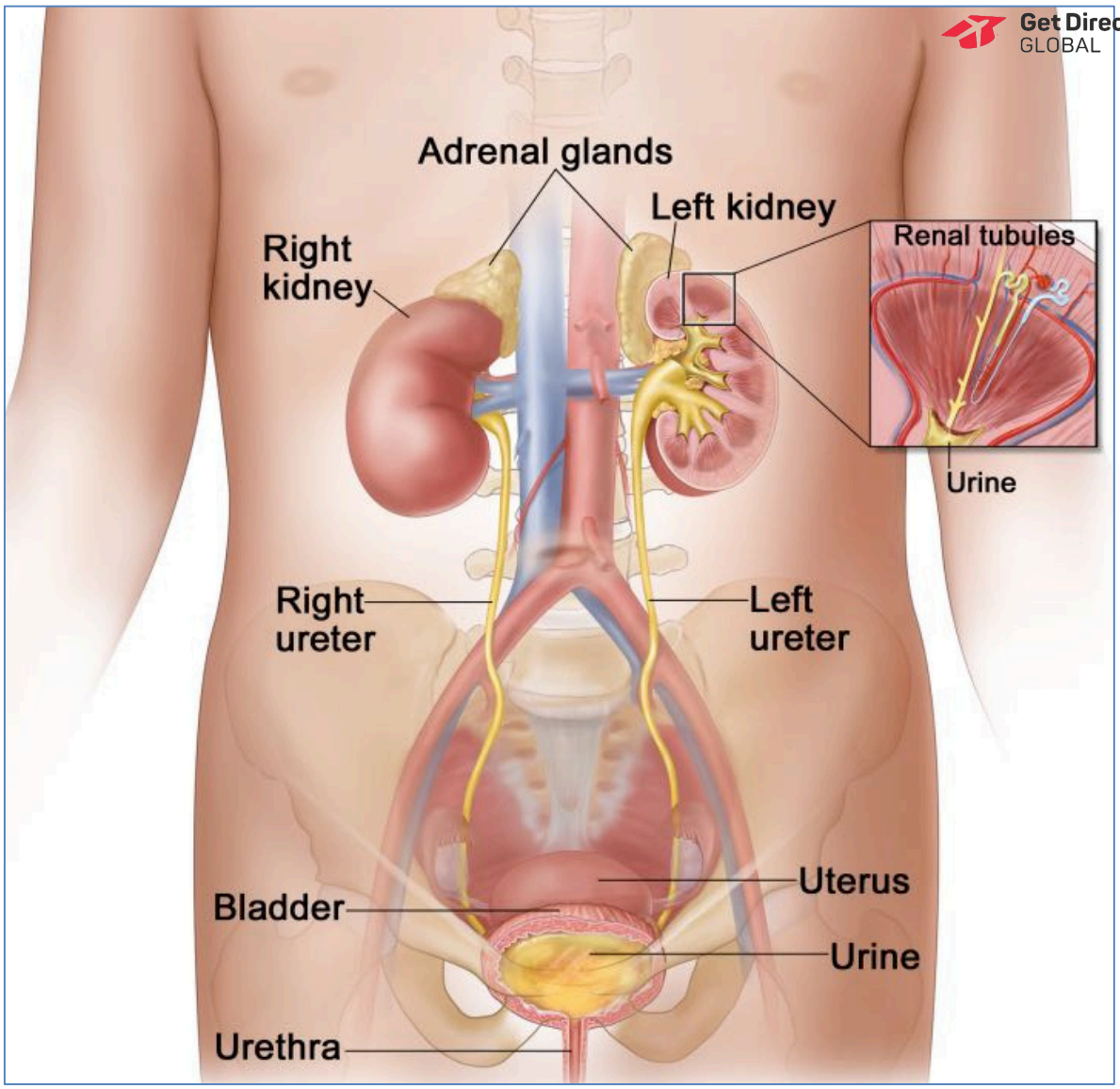




Loop of Henle. This work by Cenveo is licensed under a Creative Commons Attribution 3.0 United States (http://creativecommons.org/licenses/by/3.0/us/).

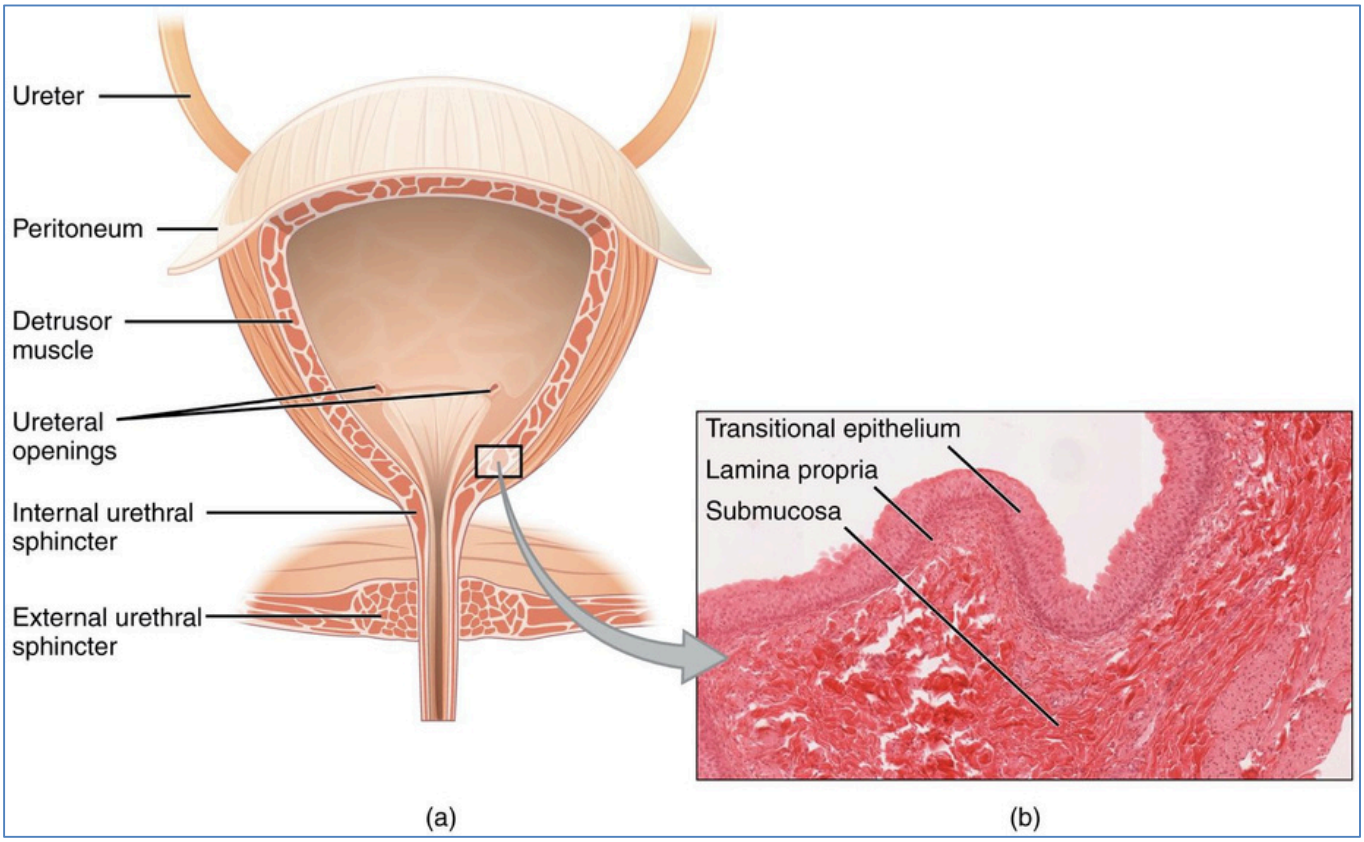
- **The Ureters**
  - o Carry Urine from Renal Pelvis → Bladder
  - o 30-35cm Long
  - o **Muscular Tubes:**
    - § Peristaltic Contractions – help urine flow
  - o **Histology:**
    - § Mucosa = Transitional Epithelium
    - § Smooth Muscle Outer Layer
  - o **Abdominal Part** – Runs just anterior to Psoas Major
  - o **Pelvic Part** – From below Bifurcation of Common Iliac Artery
  - o **3 Sites of Constriction:** - (where calculi can be caught)
    - § 1- Junction with Renal Pelvis (Hilum)
    - § 2- Entry to Bony Pelvis (Over the Pelvic Brim)
    - § 3- Entry to Bladder
  - o **Blood Supply:**
    - § Upper Ureter – Branch of Renal Artery
    - § Middle Ureter – Branches of Gonadal
    - § (Ovarian/Testicular), Aorta & Common Iliac Arteries.
      Lower Ureter – Branches of Internal Iliac





https://www.ncbi.nlm.nih.gov/books/NBK65953/

- **The Bladder:**
  - **General Info:**
    - Muscular-Walled Sac (**Detrusor Muscle)**
    - Inferior to Peritoneum
    - Ureter Openings – Just Below Pubic Tubercles.
  - **Notable Areas:**
    - **Trigone:**
      - Smooth Triangular Area on lower-posterior bladder wall
      - Triangle defined by openings of Ureters (top) & Urethra (bottom)
    - **Apex** at bottom
    - **Neck** – Entry to Urethra
    - Guarded by **Internal Urethral Sphincter**
    - **Body**
      Fundus – Above Ureteral Openings.
  - **Histology:**
    - Mucosa = Transitional Epithelium
    - Muscular Layer = Detrusor Muscle
    - Visceral Peritoneum
  - **Male:** **Rectovesical Pouch** – Space between Bladder & Rectum
    - **Blood Supply** – Internal Iliac Artery
  - **Female:**
    - **VesicoUterine Pouch** – Space between Bladder & Uterus
    - **Blood Supply** – Internal Iliac & Vaginal Arteries.



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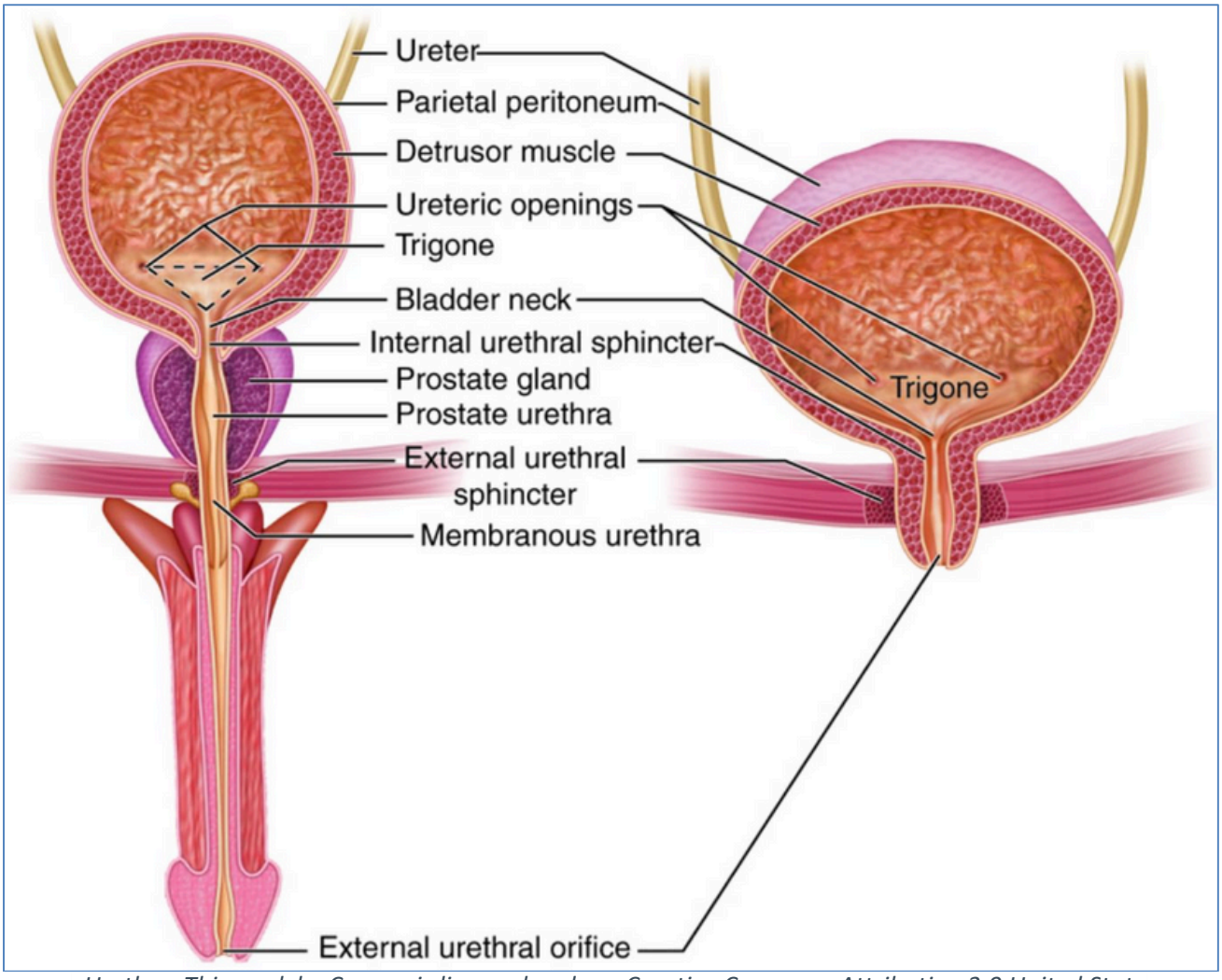
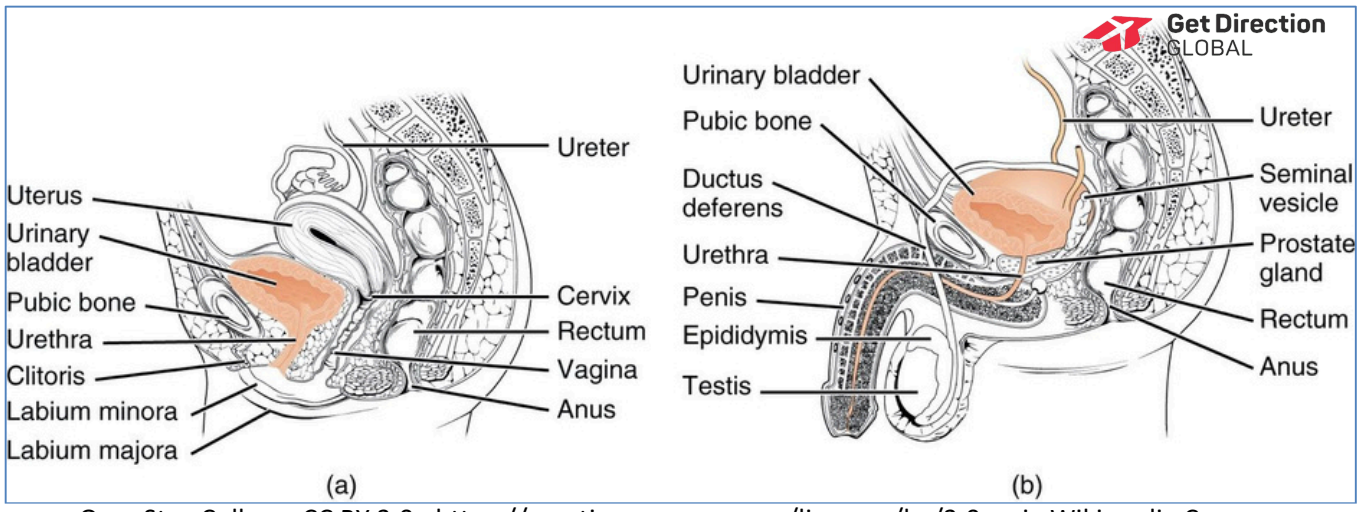
- **Urethra:**
  - o Thin-Walled Muscular Tube.
  - o Drains Urine from Bladder →Outside
  - o **Sphincters:**
    - § **Internal Urethral Sphincter**
      - @ Bladder-Urethra Junction
      - Prevents leakage between urinations.
    - § **External Urethral Sphincter**
      - @ Urethra-Pelvic Diaphragm Junction
      - Voluntary
  - o **Male:**
    - § 20cm Long
    - § Integrated with Reproductive System
    - § **3 Parts + Histology:**
      - Prostatic Urethra - Transitional Epithelium
      - Membranous Urethra - Pseudostratified Columnar Epithelium
      - Spongy (Penile) Urethra - Pseudostratified Columnar Epithelium
  - o **Female:**
    - § 2-3cm Long
    - § **Histology:**
      - Mostly Pseudostratified Columnar Epithelium
      - Stratified Squamous (external orifice)
    - § Separate from Repro. System





RENAL PHYSIOLOGY

RENAL PHYSIOLOGY:

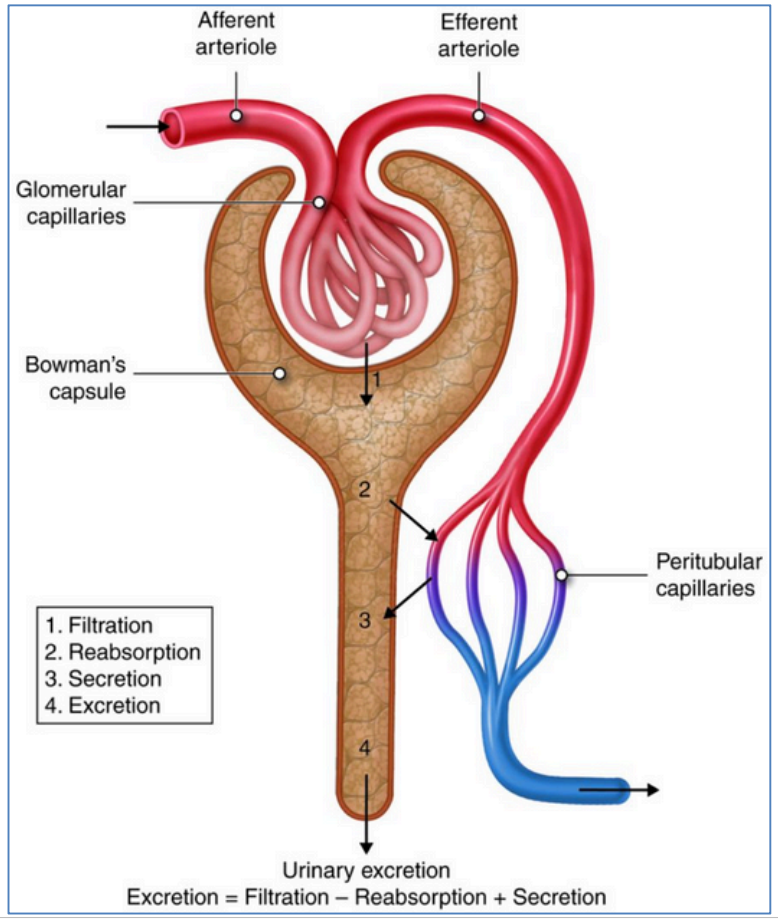
7 Physiological Functions of the Kidney:

- **Fluid Conservation**
- **Electrolyte Balance (Particularly Na+, K+, PO_4 & HCO_3)**
- **Waste Disposal (Urea, Creatinine, Urobilin/Bilirubin)**
- **Acid-Base Homeostasis (H+ Resorption/Excretion…OR HCO-3 Resorption/Excretion)**
- **Blood Pressure Regulation (Fluid Volume + Hormonal [Renin/Angiotensin])**
- **Haematopoiesis (Erythropoietin EPO)**
- **Vitamin D Activation**

Relevant Hormones:

- **Renin:**
  - o **Released by** Juxta-Glomerular Apparatus in response to Renal Hypoperfusion
  - o **Causes →** Conversion of Angiotensin-I to Angiotensin-II,
    - § →& Vasodilates Afferent Arteriole to ↑Kidney Perfusion
- **Angiotensin-II:**
  - o **Released by** Lungs in response to Renin
  - o **Causes →** Systemic Vasoconstriction → ↑BP
    - § →& Constriction of the Efferent Arteriole to ↑GFR
    - § → & Adrenal Release of Aldosterone
- **Aldosterone:**
  - o **Released by** Adrenal Glands in response to AT-II, HyperKalaemia, & HypoNatraemia.
  - o **Causes →** ↑Na+ Reabsorption (& K+ Excretion) (& H2O Reabsorption)
- **Anti-Diuretic Hormone (ADH):**
  - o **Released by** Posterior Pituitary Gland in response to ↑Plasma-Osmolality (Dehydration)
  - o **Causes →** ↑Water Resorption from the Collecting Ducts → ↑Plasma Volume & ↓Urine

URINE PRODUCTION AND EXCRETION

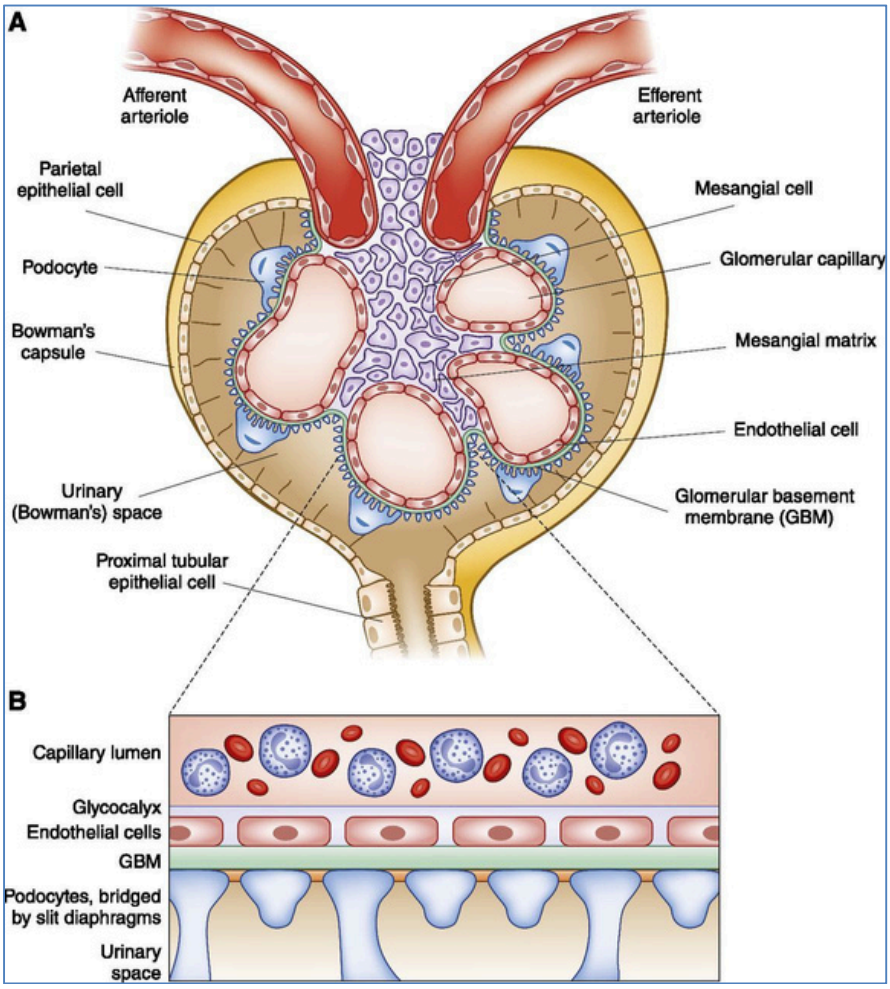


STEP 1 – GLOMERULAR FILTRATION:

- **Filtration of Large Volumes of Blood:**
- o Filtration is *Passive & Non-Selective* (Fluids & Solutes are forced through via Hydrostatic Pressure)

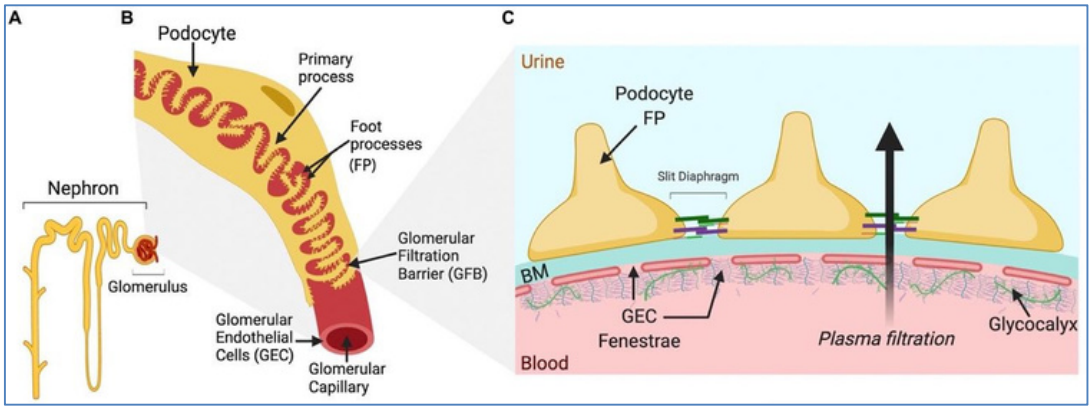
Filtration Through 3 Layers of Capillary (Glomerular) Membrane:

o Endothelium (Endothelial cells)
o Basement Membrane (GBM)
o "Podocytes" of Visceral Layer of Glomerular Capsule (Note: "Podocyte" = "Cells with Feet")

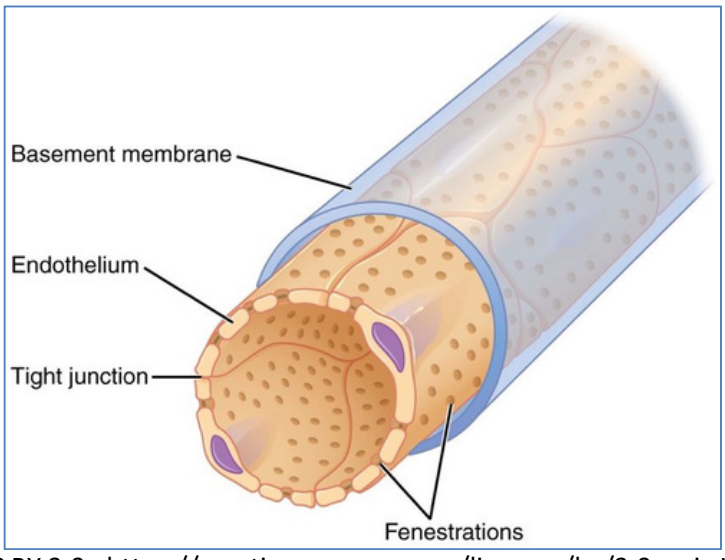


The Players: Cells Involved in Glomerular Disease; A. Richard Kitching, Holly L. Hutton; https://cjasn.asnjournals.org/content/11/9/1664

- **Filtrate:**
- o Ie: The Glomerular *FILTRATE* = Similar to Plasma (But *Without* the Proteins)
- **Permeability of Glomerular Membrane:**
- o Filterability of Solutes – Based on Size.
- o Small Chemicals are often bound to Plasma Proteins (Ca+, FA's, Drugs) – Hence not freely filtered.
- o **Note:** Visceral Membrane of Glomerular Capsule is *IMPEREABLE TO PROTEINS* – Ie: If Proteins/Cells appear in urine →Means Membrane is Damaged



Front. Physiol., 02 June 2021 | https://doi.org/10.3389/fphys.2021.689083



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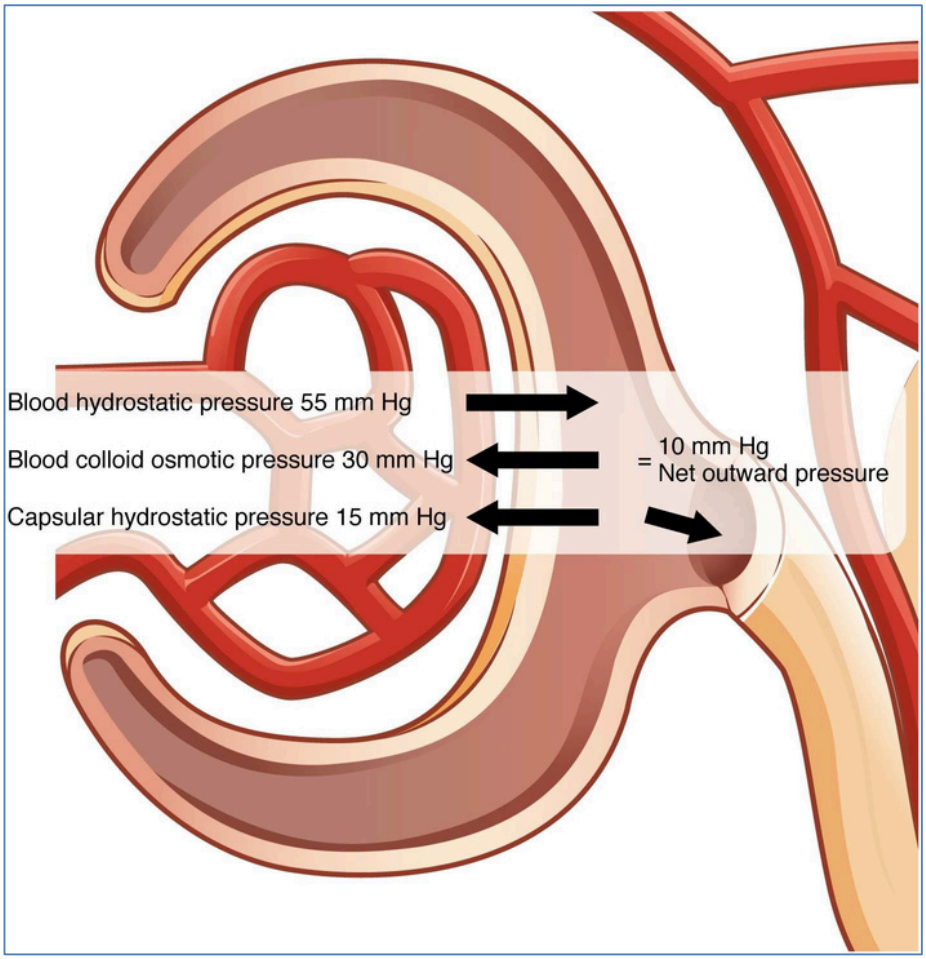
- **Glomerular Filtration Rate:** = Total Filtrate Formed/Per Minute
  - o **Determined by Net *Hydrostatic Pressure* and Net *Colloid-Osmotic Pressure*** Across Membrane.
    - § **Capillary Hydrostatic Pressure:**
      - The force the blood exerts against the capillary wall.
      - Tends to force fluids through the capillary
      - *Net Hydrostatic Pressure = Capillary Pressure – Interstitial Pressure.*
    - § **Colloid Osmotic Pressure:**
      - Opposes hydrostatic pressure
      - Due to non-diffusible molecules (In Plasma) drawing fluid into capillaries.
      - *Net Osmotic Pressure = Capillary Osmotic Pressure – Interstitial Osmotic Pressure.*



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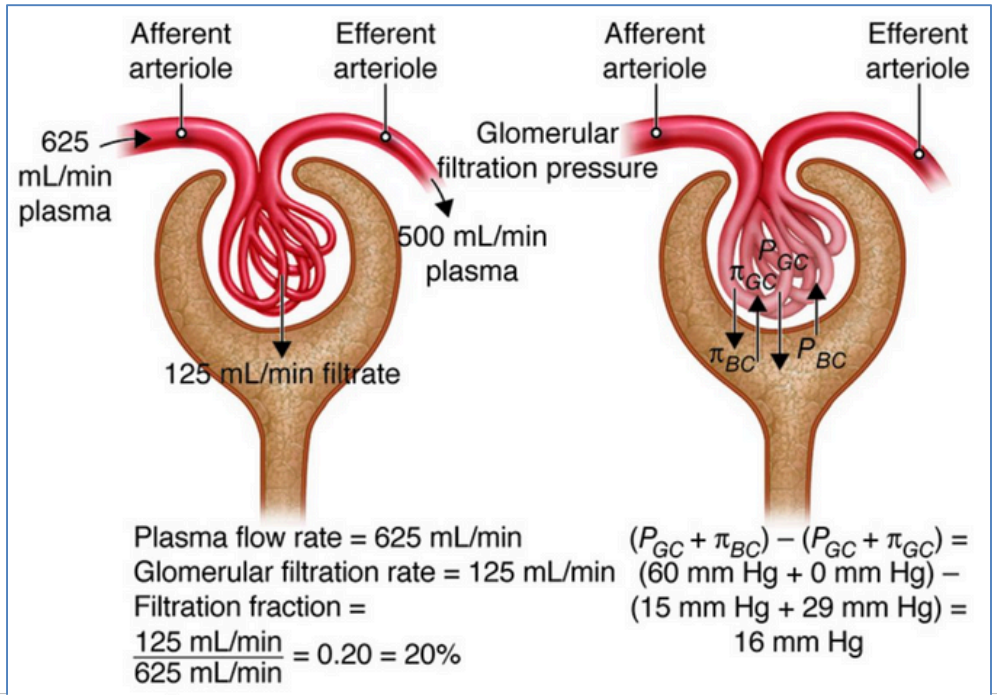
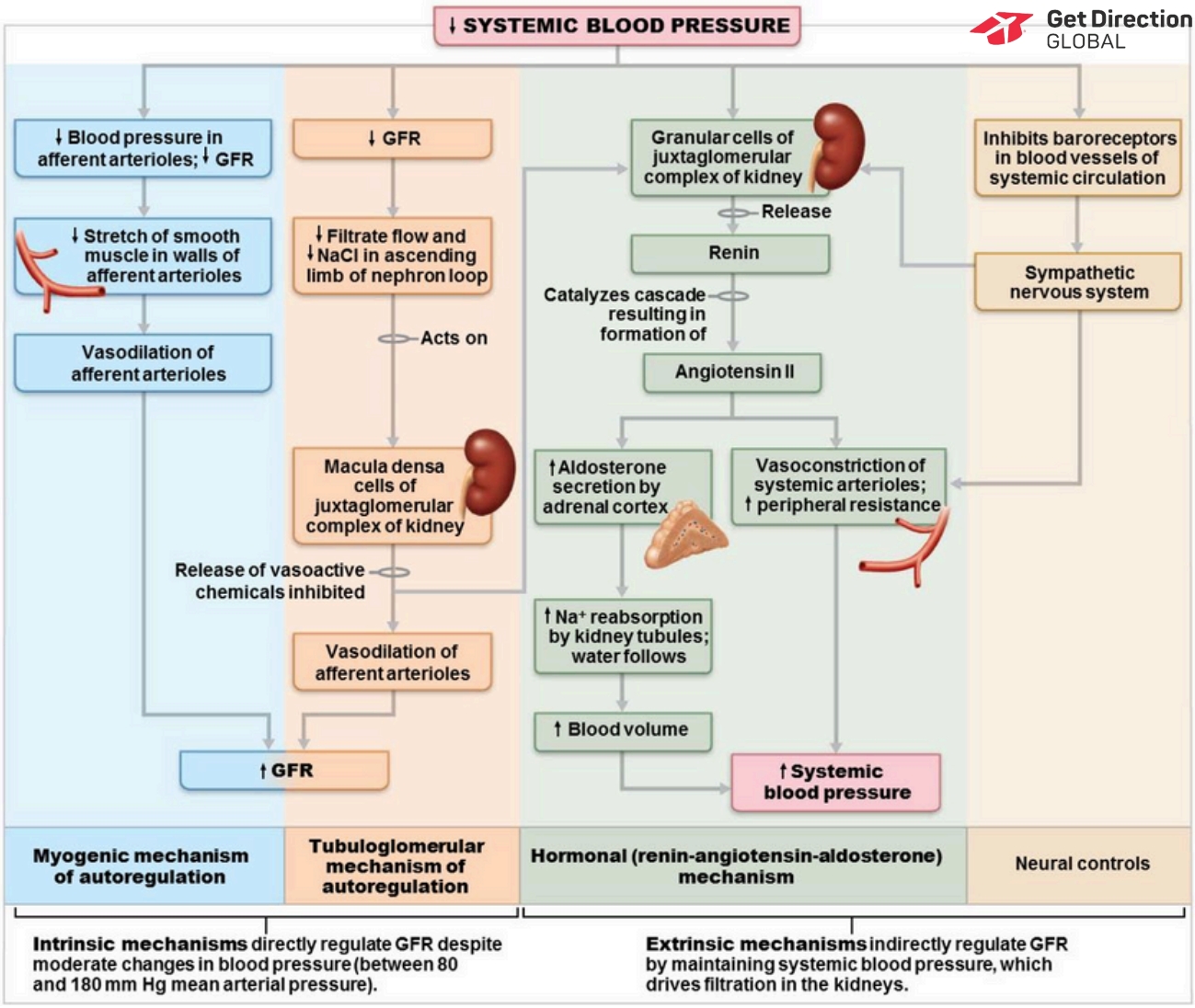
- **GFR Also Determined By:**
  - **Total Surface Area for Filtration**
  - **Membrane Permeability**
- **Kidneys receive ≈1/4 of Cardiac Output (1L of Blood/min)**;
  - Of that ≈125mL of Filtrate is Generated/Min → 180L of Filtrate/Day (From only 3L of
  - Plasma)
  - →Hence, The Blood Is Extremely Well Filtered.
    Note: Most of Filtrate is Reabsorbed into Blood (Via Renal Tubules)



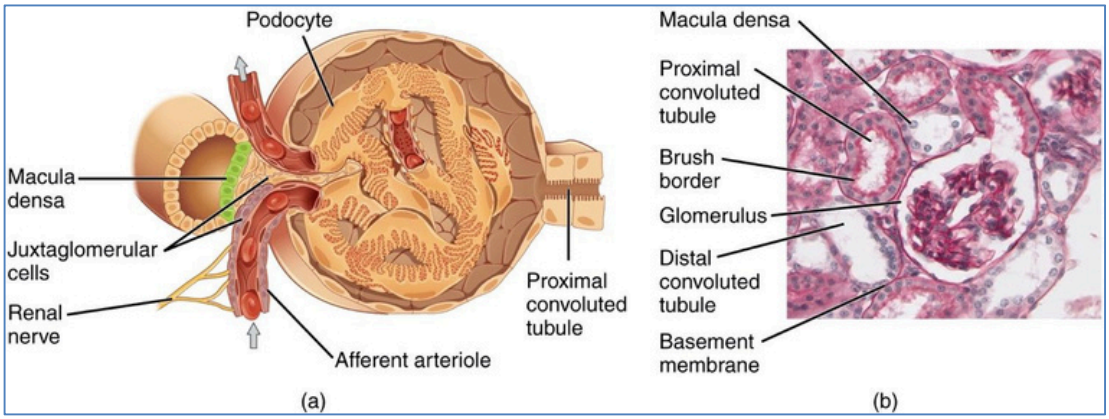
Glomerular filtration process. This work by Cenveo is licensed under a Creative Commons Attribution 3.0 United States (http://creativecommons.org/licenses/by/3.0/us/).

- **Control of GFR:**
  - **Sympathetic NS:** (Fight/Flight)
    - Constriction of Afferent & Efferent Arterioles.
    - →↓Renal Blood Flow
    - →↓GFR
  - **Hormones & Autocrine Secretions:**
    - **Causing Arteriole *CONSTRICTION*:**
      - (ADRENALINE, ENDOTHELIN...others)
        - →↓Renal Blood Flow
        - →↓GFR
    - **Causing Arteriole *DILATION*:**
      - (NITRIC OXIDE, PROSTAGLANDINS, BRADYKININ...others)
        - →↑Renal Blood Flow
        - →↑GFR
  - **Angiotensin II:**
    - Constriction of *EFFERENT ARTERIOLES*
      - →↓Renal Blood Flow
      - BUT – Maintains GFR (By keeping Glomerular Hydrostatic Pressure Up)



https://www.emr.ac.uk/wp-content/custom/case-72/blood-pressure-regulation.php

- **'Autoregulation' of Renal Blood Flow:**
  - o (The first of the body's regulators of Mean Arterial Pressure)
  - o Automatic Adjustment of Blood Flow to a Capillary Bed Relative to the Tissue's Requirements
    - § Maintains Normal Renal Function (GFR) Despite Changes in Arterial Pressure.
  - o **How? - Juxtaglomerular Apparatus is Sensitive to:**
    - § **Metabolic Controls: → Vasodilation:**
      - Low Oxygen / Nutrient levels
      - Nitric Oxide
      - Endothelin
    - § **Myogenic Control: → Vasoconstriction:**
      - Sheer Stress: Vascular Smooth Muscle Contracts When Stretched



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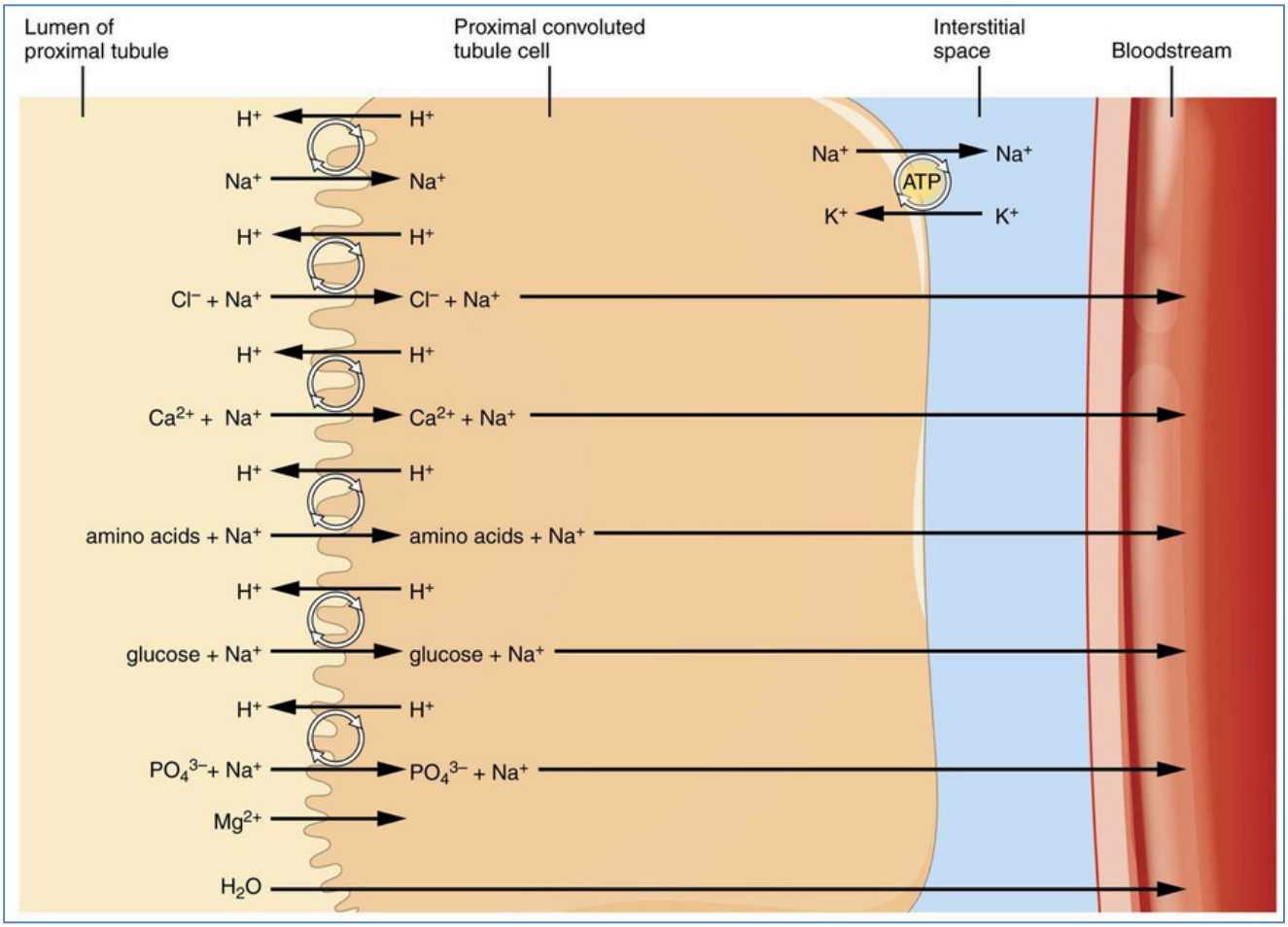
STEP 2 – TUBULAR REABSORPTION:

- Reabsorption of Certain Filtered Substances (In Renal Tubules) → Back into Blood
- Normally, 99% of Filtrate is Reabsorbed
- **-Is Highly Selective:**
  - o Some Substances (Eg: Glucose) are Almost Completely Reabsorbed.
  - o Some Substances (Eg: NaCl-) are Variable.
  - o Some Substances (Eg: Urea) are Not Reabsorbed at All.
- **-Is Passive & Active:**
  - o **Passive:**
  - o § Eg: Water – Via Osmosis
  - **Active:** Ie: Moving Solutes Against an Electrochemical Gradient. (Either Primary/Secondary)
    - § Eg: Na+ - (By Na+/K+-ATPase)
    - §
  - o Remember that all Active & Passive Transporters (Excluding Channels) Reach Saturation. (Max.V)
    - § Eg: Glucose doesn't normally appear in urine. However, if Filtered Load Exceeds Reabsorption, Urinary Excretion Occurs (Ie: In Uncontrolled Diabetes.)
- **Solutes May Be Reabsorbed Via 1 of 2 Routes:**
  - o **1-** Transcellular Pathway – Through The Cells
  - o **2-** Paracellular Pathway – Between Cells
- **Active Na+ Reabsorption:**
  - o Occurs in Ascending Limb of Loop of Henle.
  - o TransCellular Pathway
  - o Involves 3 Steps:
    - § Na+ *Passively* Diffuses from Tubule Lumen (Down an Electrochemical Gradient)→ Tubule
    - § Cell
    - § Na+ *Actively* Transported across Basolateral Membrane → Interstitium (By Na+/K+-ATPase) Na+ (+Water & Other Solutes) Reabsorbed from Interstitium → Peritubular Capillaries.

Substances Reabsorbed & Secreted by the PCT



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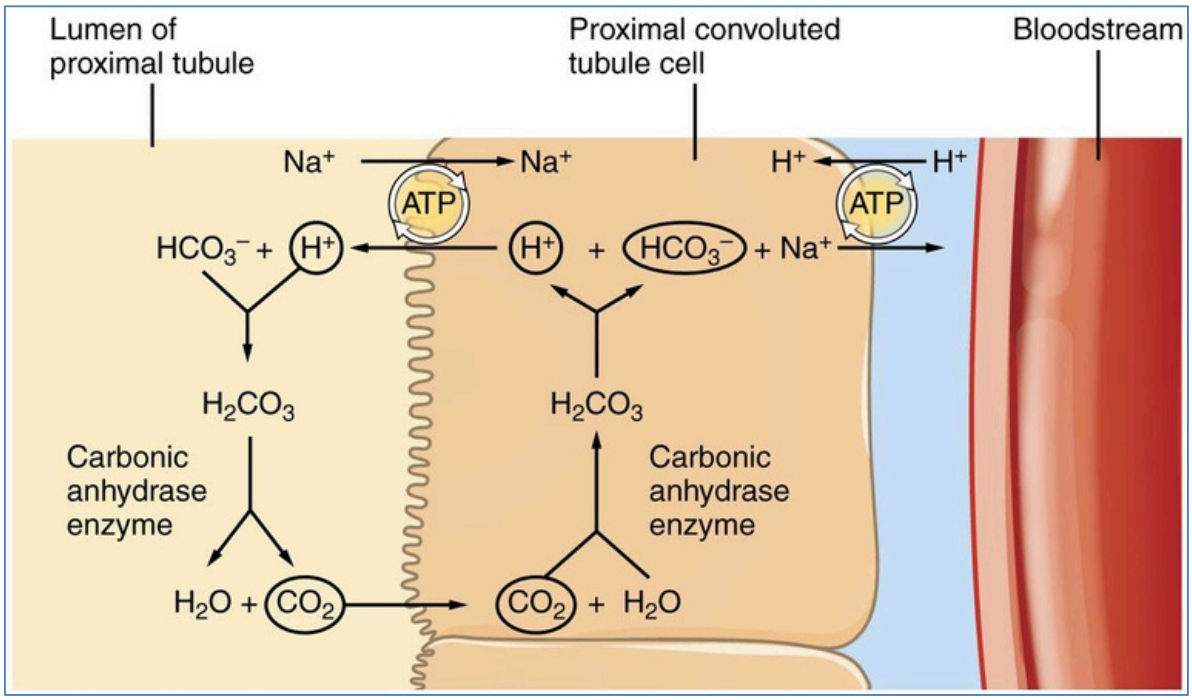
Reabsorption of Bicarbonate from the PCT

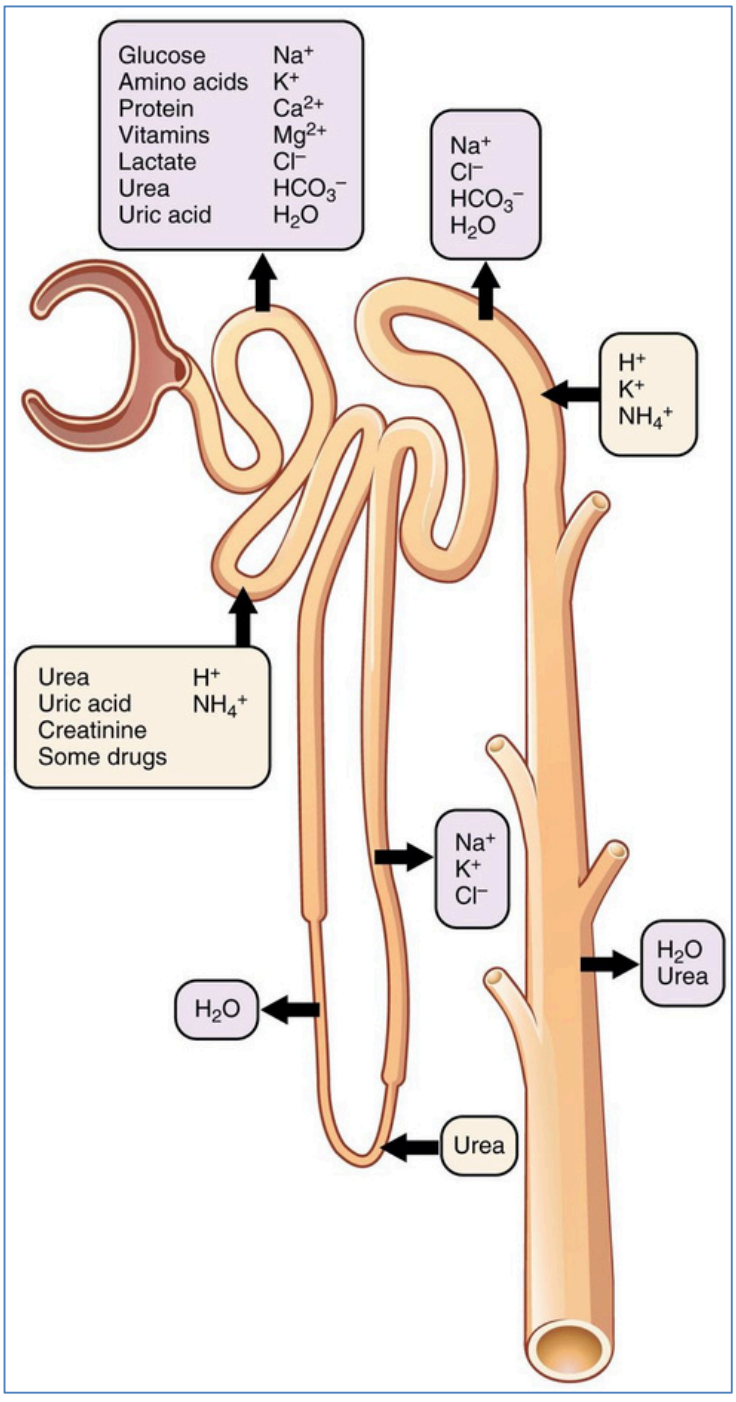


STEP 3 – TUBULAR SECRETION:

- Active Secretion of Substances From Peritubular Capillaries (Blood) → Into Renal Tubules
- **Important For:**
  - o Disposing of Substances That Weren't Filtered (or Weren't Filtered Enough)
    - § Eg: Drugs (Eg: Penicillin)
  - o Eliminating 'Bad' Substances that have been Passively Reabsorbed
    - § Eg: Urea, Uric Acid, etc.
  - o Removing Excess K+ ions
  - o Controlling Blood pH
- **Proximal Tubules:**
- o Site of Secretion of ***Organic Acids/Bases*** (Bile Salts, Oxalate, Uric Acid, etc)

Renal Tubules:

- o Secretion of K+
- o Secretion of H+
- o Secretion of Drugs/Toxins (Eg: Penicillin)



MICTURITION REFLEX (URINATION):

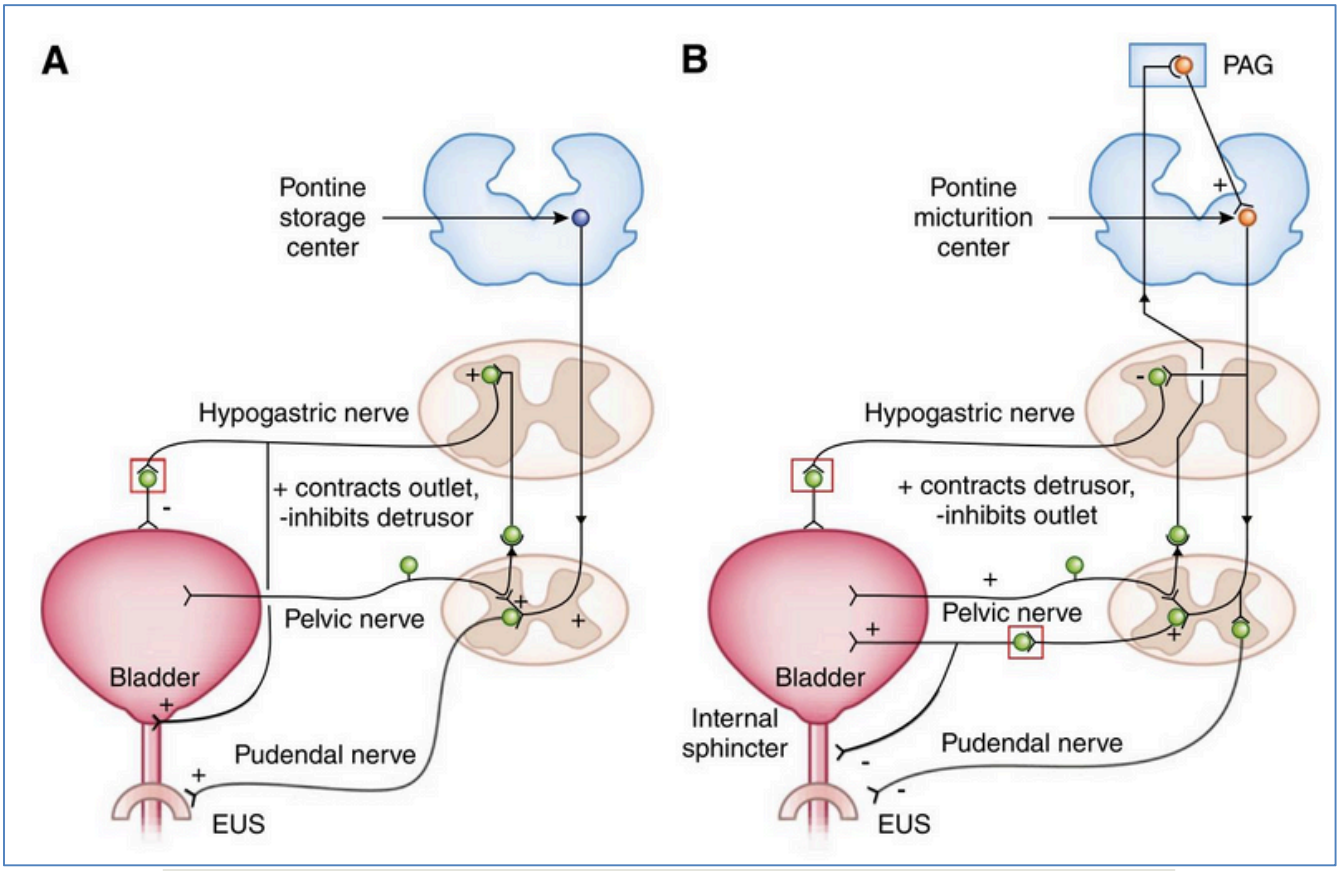
- **A Spinal-Cord Reflex**.
- o Bladder Fills Until Pressure Reaches a Critical Level → Initiates Micturition Reflex

Voluntary (in Health Adults)

- o Can be Inhibited by Higher Brain Centres.
  - § When Person is Ready to Urinate, Brain-Inhibition is removed
- o Involuntary (In Infants + Neurological Injury) → Urinary Incontinence
- o A *'Learned'* Process (develops @ 2-3yrs)

- **2 Phases:**
  - o Collection Phase
  - o Micturition Phase
- **Reflex Process:**
  - o Facilitated / Inhibited by Higher Brain Centres
  - o The *Phase* of the system - dependent on:
    - § 1- A Conscious Signal from the brain and
    - § 2- The *Firing Rate* of sensory fibres from the bladder and urethra.
  - o **Empty Bladder:** Afferent Firing Rate ↓→ excitation of the outlet (the sphincter and urethra), and relaxation of the bladder.
  - o **Full Bladder:** Afferent Firing Rate ↑ → Urinary Urge.
    - § **Voluntary Urination:** Person Consciously Initiates→ Bladder contracts +Sphincters relax.
    - § Urination Continues until Bladder is Empty → Bladder Relaxes + Sphincters Contract → Collection Phase

Nerves Innervating the Urinary System. A: Storage Reflex; B: Voiding Reflex



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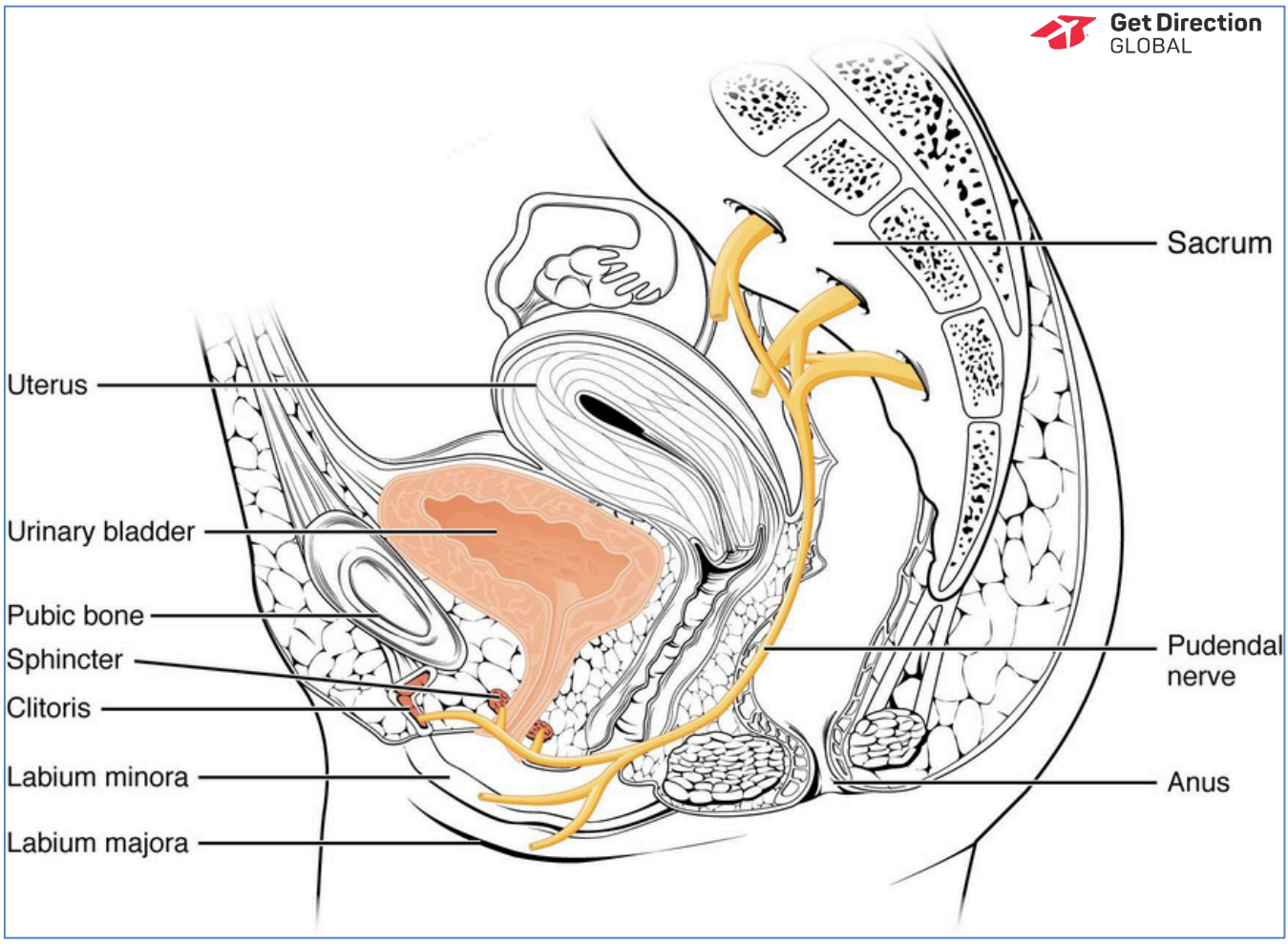

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ROLE OF THE KIDNEYS IN FLUID & ELECTROLYTE BALANCE

ROLE OF THE KIDNEYS IN FLUID & ELECTROLYTE BALANCE

W hy M aintain Fluid & Electrolyte Balance?:

- Critical for Normal Cell Function
- Critical for Chemical Stability (Homeostasis) of Surrounding Fluids
- *Electrolyte Balance (Particularly **Na+** & **K+**) – Critical for function of Excitable Tissues
- Critical for Blood Pressure Homeostasis

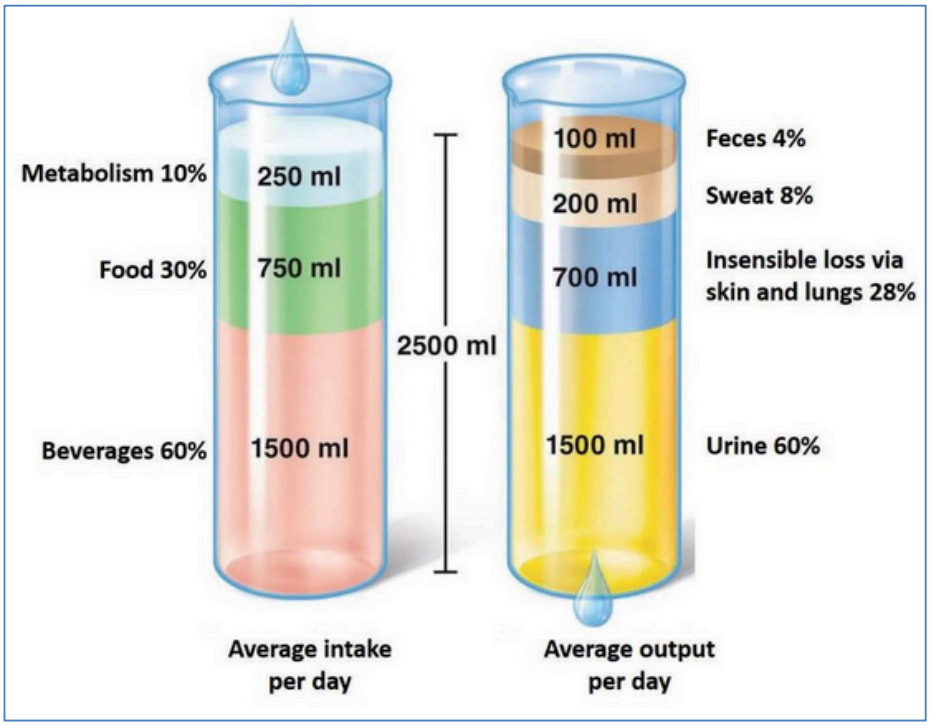
FLUID BALANCE:

Normal Adult Fluid Volume ≈ 40 Litres:

- **Extracellular =** 15 Litres
  - o 3 Litres = **Plasma**
  - o 12 Litres = **Interstitial Fluid**
- **Intracellular =** 25 Litres

Water Intake & Output:

- **Intake:**
  - o Produced in Metabolism
  - o Contained in Foods
  - o Consumed Fluids
- **Output:**
  - o Faeces (Obligatory)
  - o Sweat (Obligatory)
  - o Lungs (Obligatory)
  - o Urine



https://repository.ubn.ru.nl/bitstream/handle/2066/191611/191611.pdf

Regulation of Water Intake (Thirst):

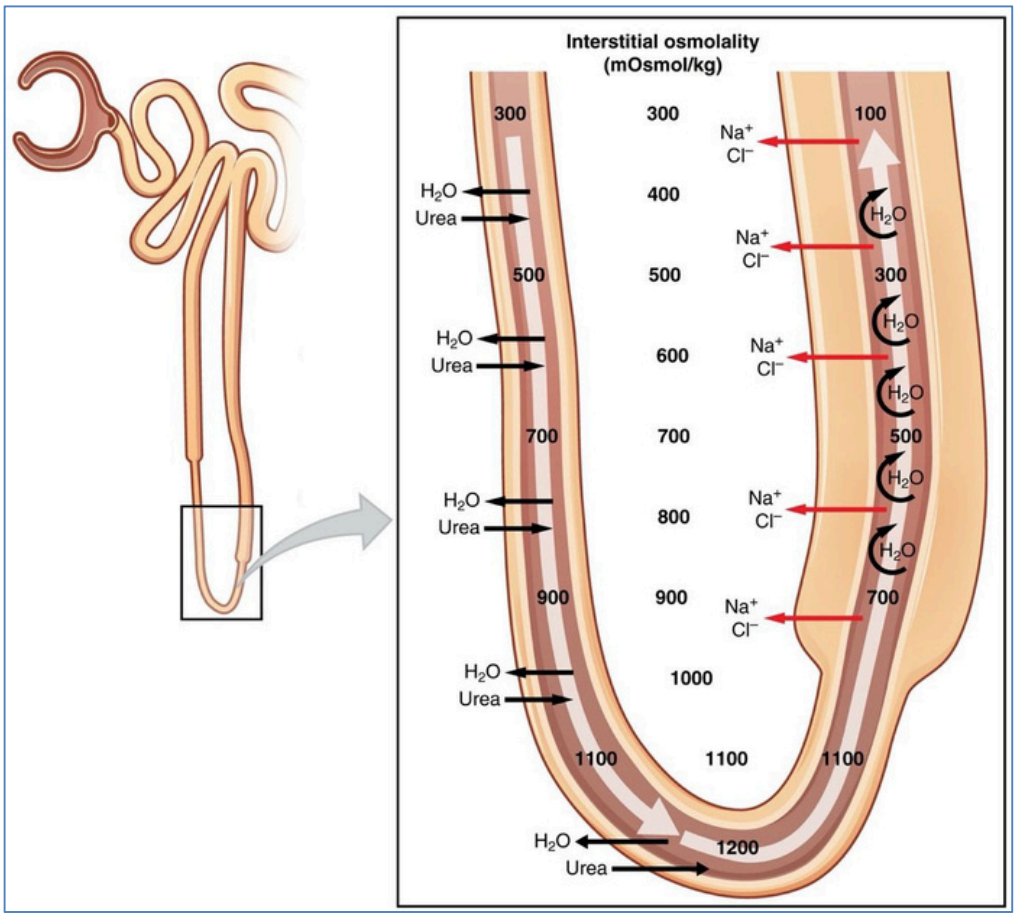
- **Thirst Triggered by 2 Things:**
  - o **1-** A 10%+ Decrease in Plasma Volume....OR
  - o **2-** A 1-2% Increase in Plasma Osmolality
- **1- Decreased Plasma Volume → Reduced Blood Flow to Salivary Glands → *"Dry Mouth"* → Triggers**
- **Thirst Centre in Hypothalamus.**

2- Increased Plasma Osmolality → Directly Triggers Thirst Centre in Hypothalamus.

Regulating Urine Volume:

- Kidneys aim to keep Solute-Load (OSMOLALITY) in Blood at around 300mOsm (miliosmols)
- The Kidneys can Regulate the Volume & Nature of Urine Produced...
- **Water Balance:**
  - **Conserve:**
  - § By Producing Low Volumes of Concentrated Urine.

    Excrete Excess:
    - § By Producing High Volumes of Dilute Urine.
- **The Loop Of Henle:**
  - o Actively Creates a High Osmotic Conc. Of Solutes in Interstitial Space of Medulla.
  - o **Descending Limb:**
    - § Permeable to Water – H2O Passively Flows into Interstitium (Then →Vasa Recta)
    - § Therefore, Descending Limb Contents Become Progressively More Hyperosmotic (Concentrated)
  - o **Ascending Limb:**
    - § Active Na+ Transport From Tubule Lumen→ Tubule Cell →Interstitium (Then→Vasa Recta)
    - § Therefore Ascending Limb Contents Become Progressively More Hypo-Osmotic (Diluted)
- **The 'Vasa Recta':**
  - o Runs ***"Counter-Current"*** to the Loop of Henle.
    - § Descending Vasa Recta = Parallel With Ascending Loop of Henle
    - § Ascending Vasa Recta = Parallel With Descending Loop of Henle
  - o **Descending Vasa Recta:**
    - § Absorbs the *Actively-Transported* Na+ (From Ascending Loop of Henle)
    - § Absorbs the *Co-Transported* K+ & Cl-
    - § *Loses* Some H2O
      - -Therefore Becomes More Hyper-Osmotic (As you go down)
  - o **Ascending Vasa Recta:**
    - § Absorbs the H2O (Lost through Descending Limb of Loop of Henle)
    - § *Loses* Some of the Salts/Ions into the Interstitium. (Na+, Cl-, K+)
      - -Therefore Becomes More Hypo-Osmotic (As you go Up)



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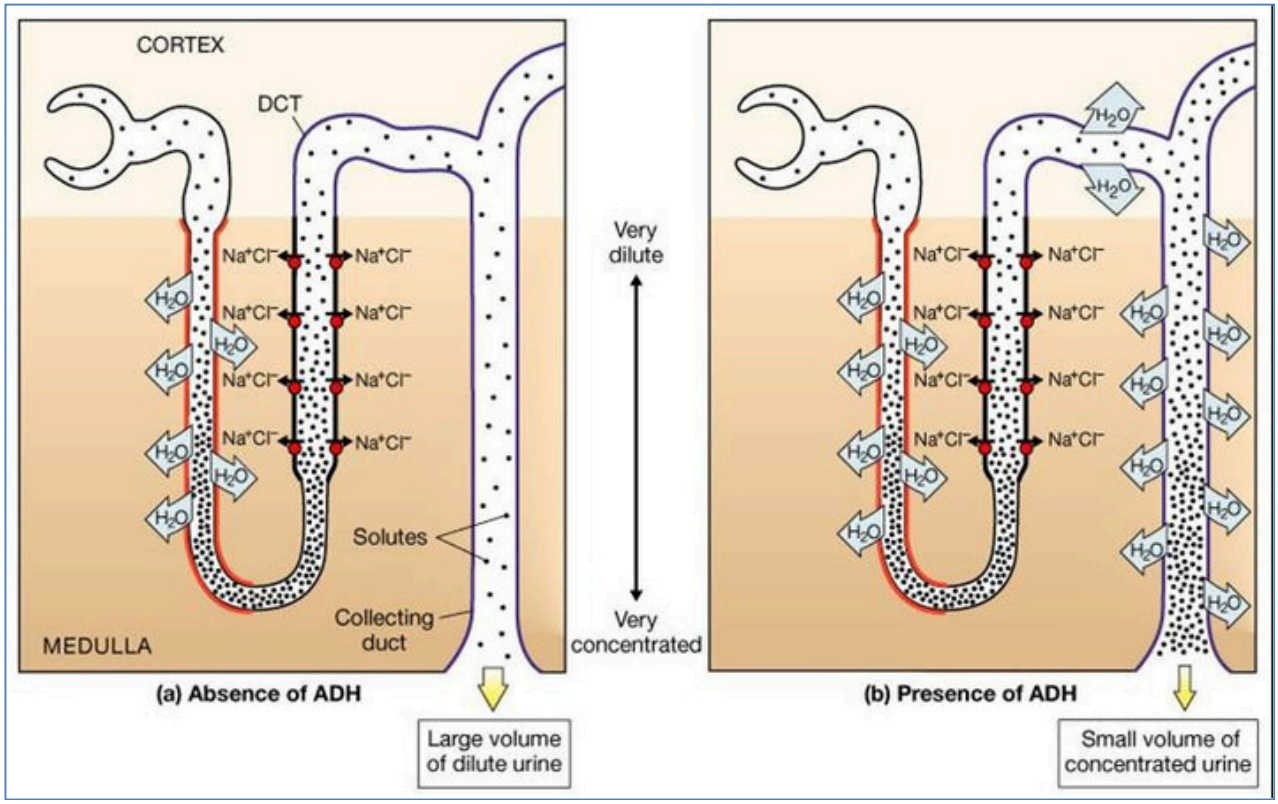
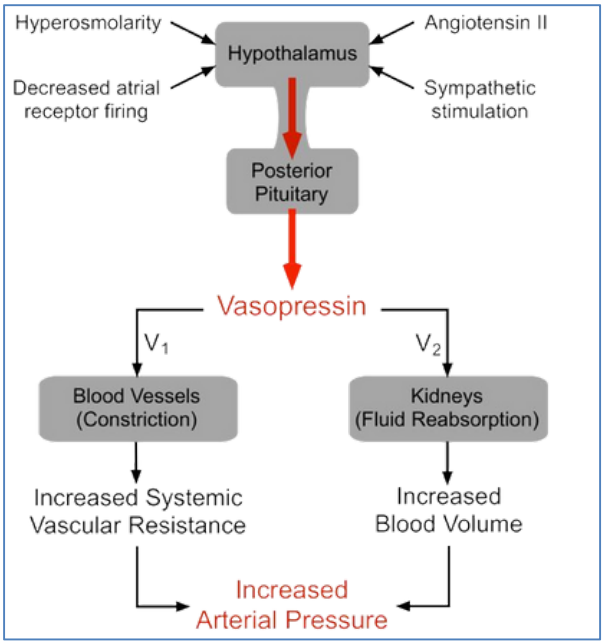
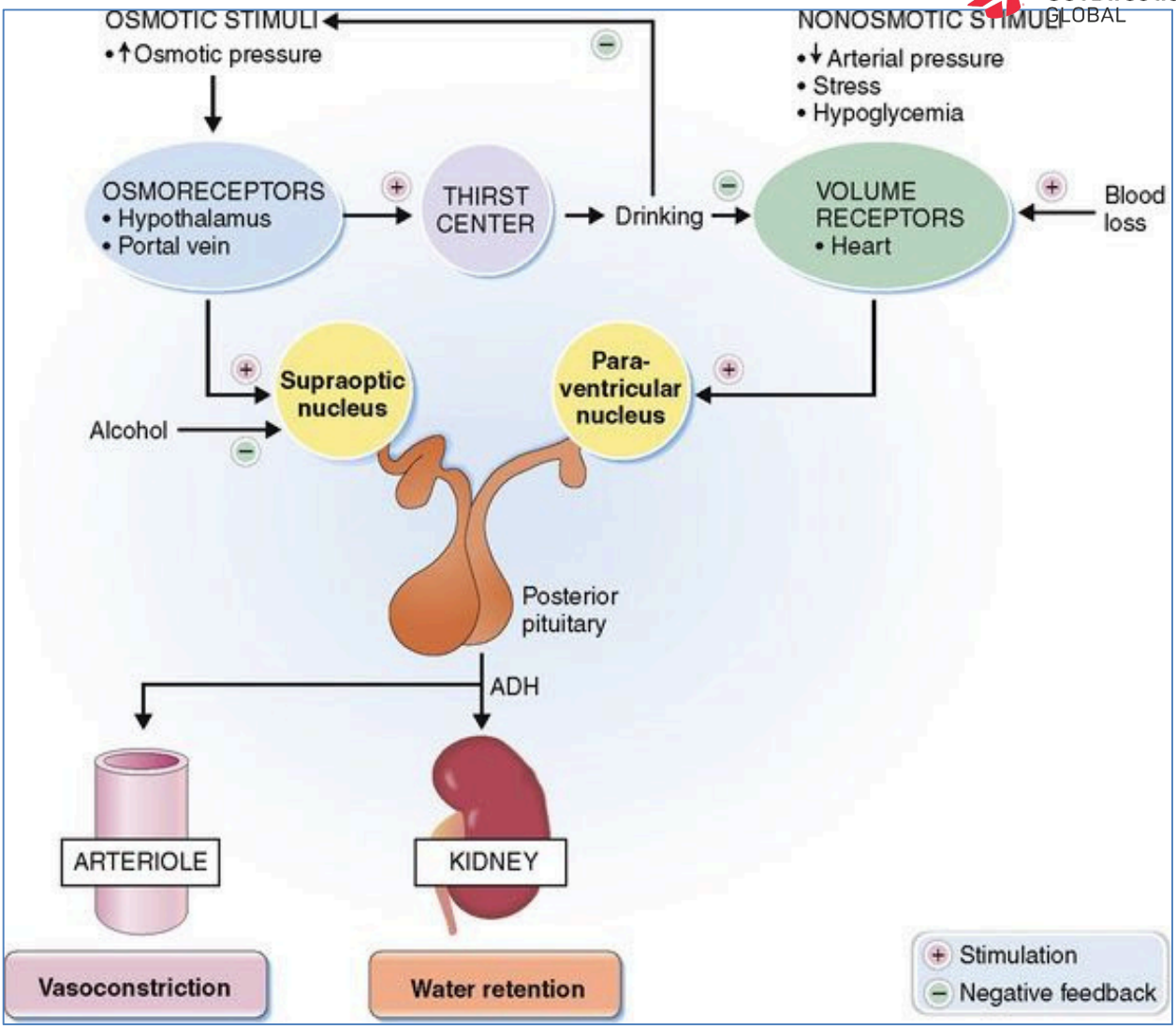
Anti-Diuretic Hormone (ADH) (Aka: "Vasopressin"):

- Acts to maintain Blood Volume.
- Made by Posterior Pituitary (In Response to Angiotensin-II)
- Primary Regulator of Urine Volume
- **Released in response to:**
  - o Stimulation of Osmoreceptors in Hypothalamus due to Increased Plasma Osmolality
  - o Stimulation of Hypothalamus by Angiotensin-II (Due to Renin Release by Kidneys)
- **Works by INCREASING H2O Permeability of Distal & Collecting Ducts:**
  - o Distal Tubules & Collecting Ducts are Normally *Impermeable to H2O*
  - o However, the Presence of ADH → ↑Permeability to H2O
    - § ↑Permeability to H2O + High [Solute] in Medulla →H2O Reabsorption (From Collecting Duct→ Interstitium → Blood)
- **Note: Aldosterone** (Released by Adrenal Gland in response to Angiotensin-II) Acts in conjunction with ADH by Increasing Na+ & Cl- Reabsorption (↑ Medullary Interstitial Osmolality) to facilitate more H2O Reabsorption.



https://www.austincc.edu/apreview/PhysText/Renal.html





https://basicmedicalkey.com/the-endocrine-system-7/

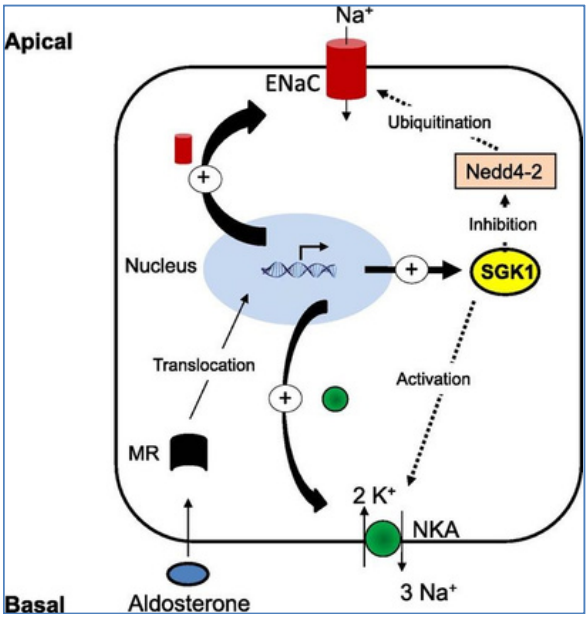
Aldosterone:

- **Secreted By:**
- o Zona Glomerulosa Cells of the Adrenal Glands

Primary Actions:

- o **Sodium Homeostasis** → Causes Na Reabsorption in the Renal Distal Tubules & Collecting Ducts
- o **Regulates extracellular fluid volume** (Via increasing blood Na concentration & renal absorption)
- o **Potassium Homeostasis** → Increases K Secretion in the Renal Distal Tubules & Collecting Ducts

- **Works by:**
  - o **ACTIVATING the Na/K-ATPases in the Principal Cells of Distal & Collecting Ducts:**
    - § Increases Na+ & Cl- Reabsorption
    - § Increases K+ Secretion
  - o **Promotes Na+-Channel (ENaC) Synthesis & Insertion into Luminal Membrane:**
    - § Facilitates the Na+ Reabsorption mentioned above.



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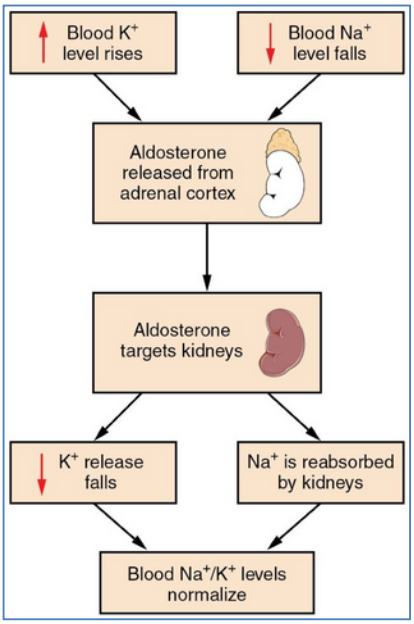
- **Release is Stimulated By Hypothalamus in Response to:**
  - o *Angiotensin-II, Part of the Renin-Angiotensin System (Due to Renin Release by Kidneys)
  - o *Hyponatraemia (Low Na+ in Blood)
  - o *Hyperkalaemia (High K+ in Blood)
  - o Stress
- **Release is Regulated By:**



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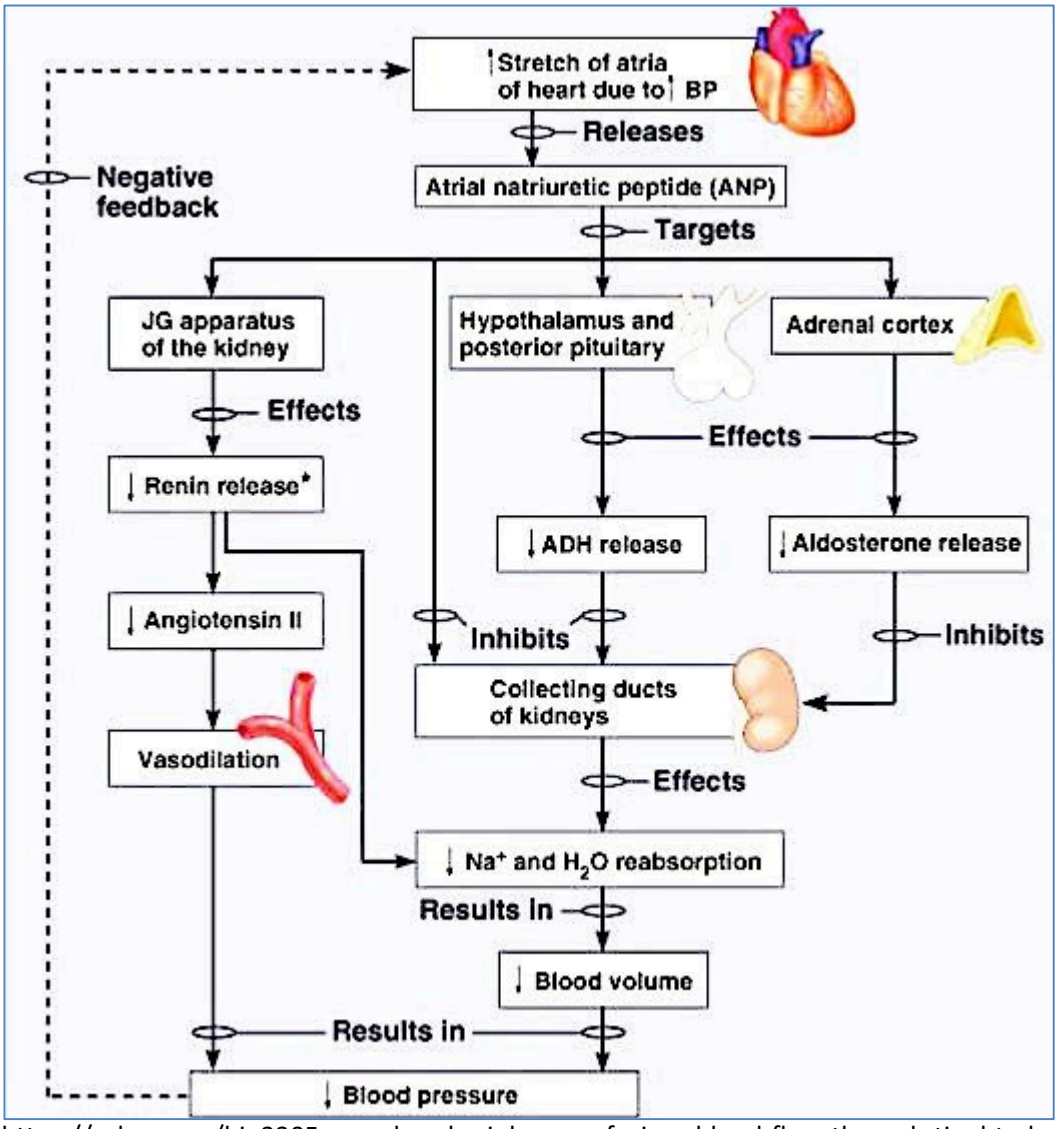
- **A trial N atriuretic Peptide (A N P) → ↑ W ater O utput:**
  - **Acts to:**
    - ↓blood volume
    - ↓Blood [Na]
  - **Secreted by** Atrial Myocytes of the Heart
  - **Released in response to:**
    - High Blood Pressure (Atrial Stretch)
  - **Works by:**
    - Dilating Afferent Glomerular Arteriole
    - Constricting Efferent Glomerular Arteriole
      - ↑Filtration Pressure → ↑ Filtration → ↑H2O & Na Excretion.
    - Inhibits Renin Secretion → Inhibits Renin-Angiotensin System
    - Inhibits Aldosterone Secretion from Adrenal Cortex.
    - Inhibits ADH Release from Post. Pituitary



https://azkurs.org/bio2305-vascular-physiology-perfusion--blood-flow-through-tiss.html

ELECTROLYTE BALANCE:

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Significant Electrolytes:

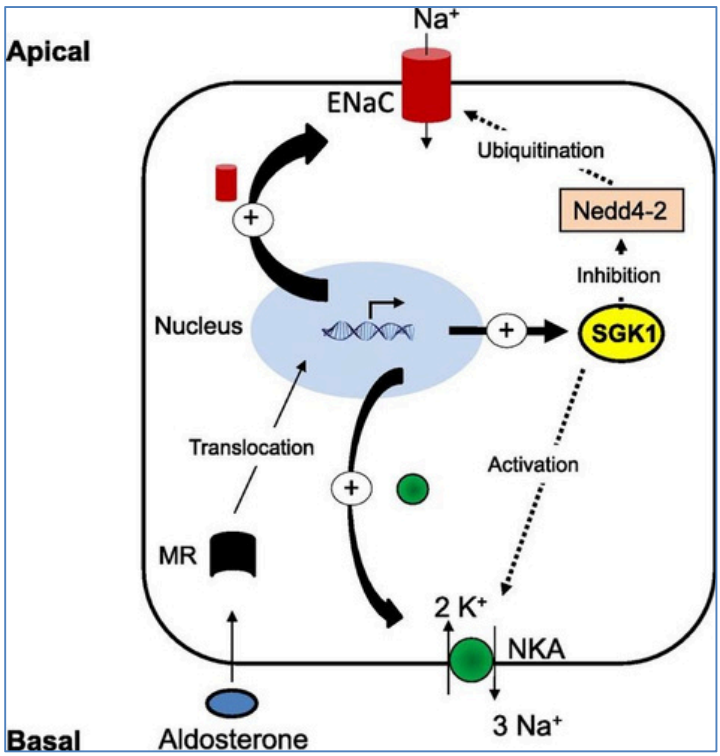
- **Na+** = High Extracellular Concentration
- **Cl-** = High Extracellular Concentration
- **K+** = High Intracellular Concentration (**Note:** too high *Extracellular* K+ interferes with Cardiac Function = Fatal)

W hy M aintain Electrolytes

- **Na+** = Important for Heart & Nerve Function/Cellular Transport
- **K+** = Important for Heart Function/Cellular Transport
- **Ca+** = Important for Muscle, Heart & Nerve Function/Bone Formation
- **Mg+** = Important for AcetylCholine Release → Important for Neural & Cardiac Function
- **HPO2-4** = Important for Bone Formation (Bone salts – primarily calcium & phosphates)

Regulation of Na+ - (The Main *Extracellular* Electrolyte):

- Primary role in Fluid & Electrolyte Balance (Because Water Follows Na+ Movement)
- Extracellular [Na+] is normally stable & is **Regulated by levels of Aldosterone:**
- **Regulated by: *ALDOSTERONE*:**
  - o **Aldosterone** = Steroid Hormone Released from The Adrenal Cortex.
  - o **Released in response to:**
    - § **Angiotensin-II, Part of the Renin-Angiotensin System (Due to Renin Release by Kidneys)
    - § *Hyponatraemia (Low Na+ in Blood)
    - § *Hyperkalaemia (High K+ in Blood)
    - § Stress
  - o **Works by:**
    - § **a) ACTIVATING the Na/K-ATPases in the Principal Cells of Collecting Ducts:**
      - Increases Na+ & Cl- Reabsorption
      - Increases K+ Secretion
    - § **b) PROMOTING Na+-Channel Synthesis & Insertion into Luminal Membrane:**
      - Facilitates the Na+ Reabsorption mentioned above.
  - o **The Effect =** Increased Na+ Reabsorption in Collecting Ducts of the Nephron.
    - § **If Aldosterone is High –** *All* Na in Filtrate is reabsorbed
    - § **If Aldosterone is Low** – *No* Na in Filtrate is reabsorbed



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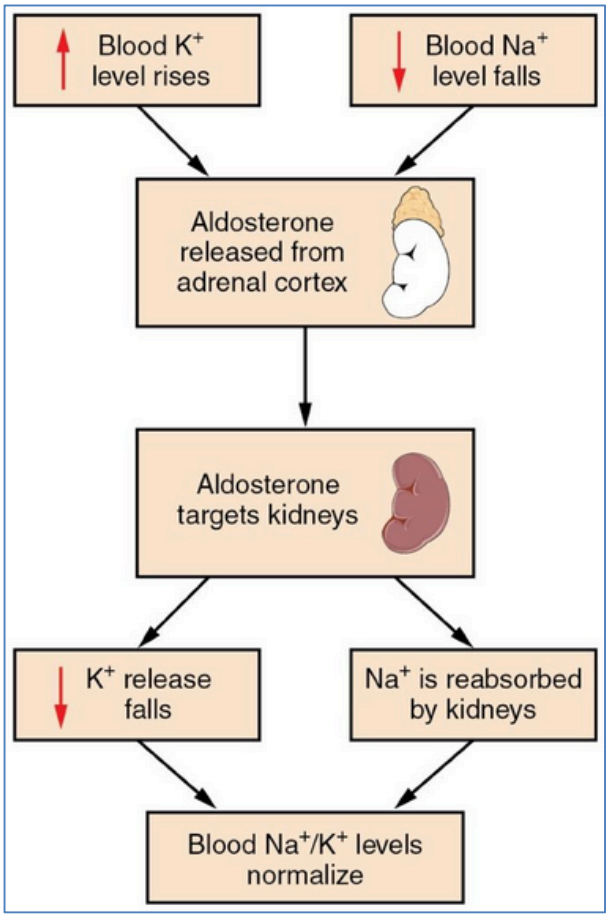
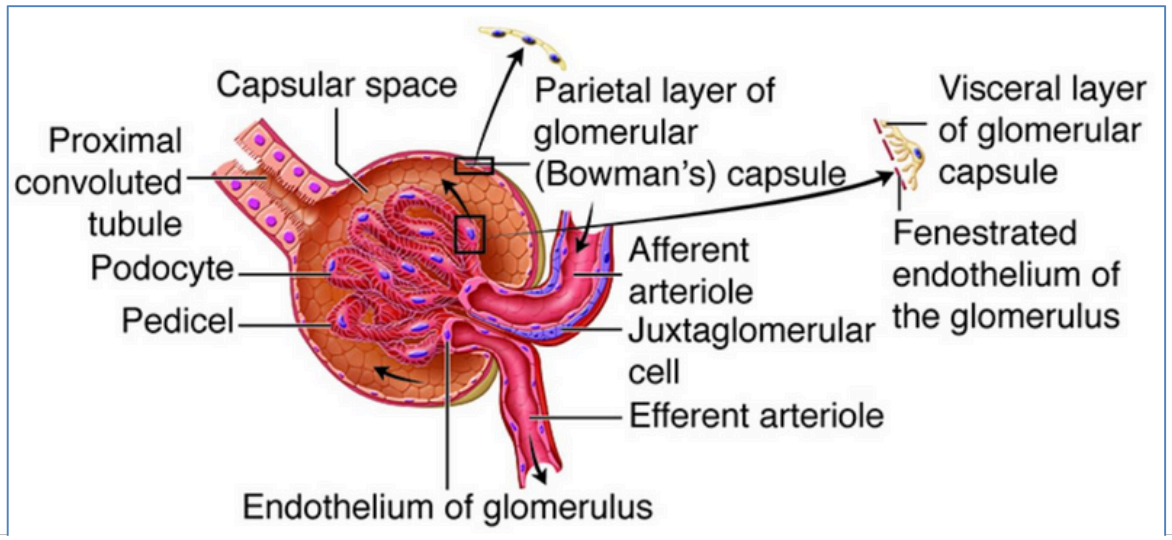
Regulation of K+: The Primary Intracellular Electrolyte:

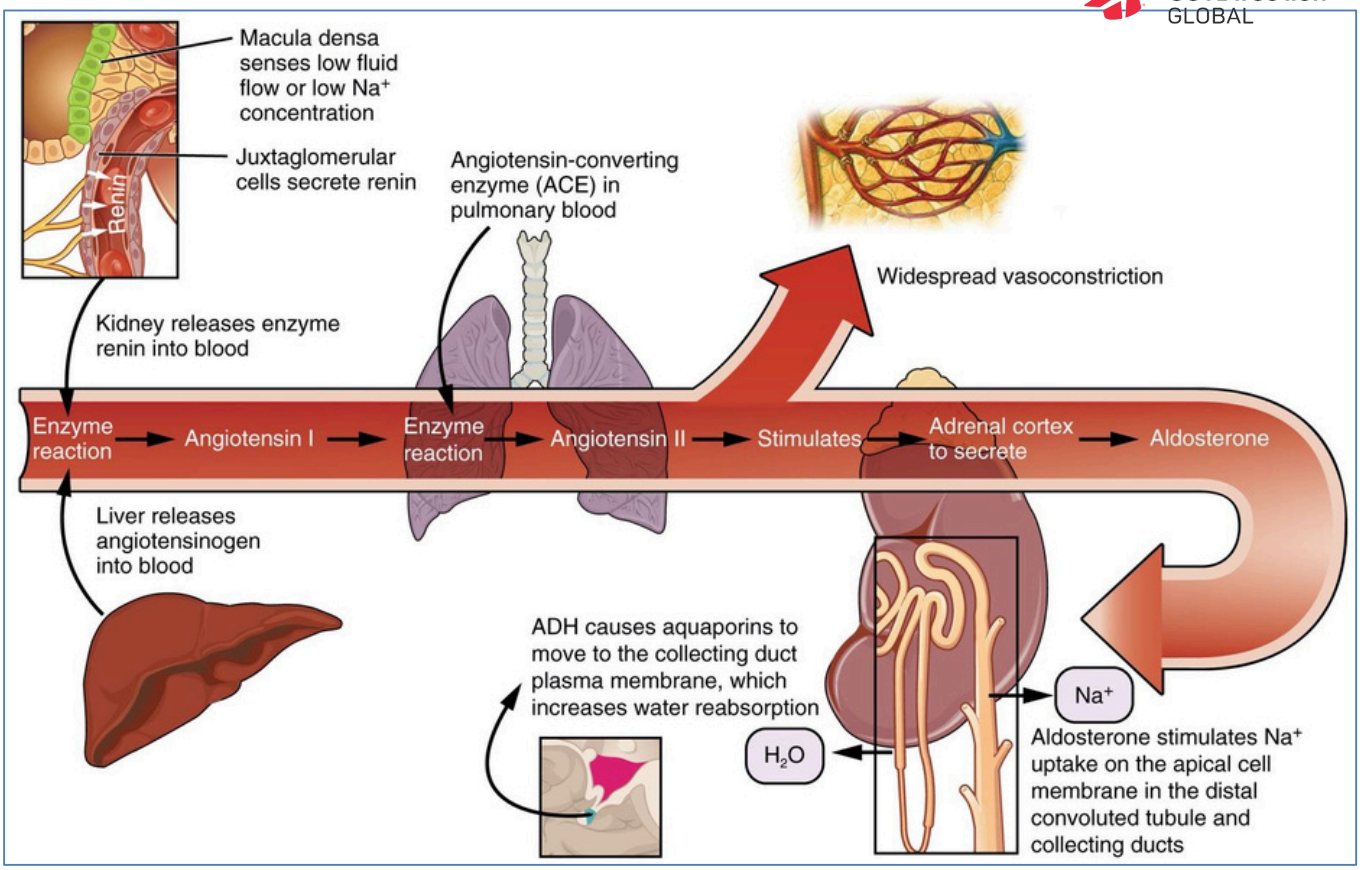
- Primary Roles in Normal Neuromuscular Function, Membrane Potentials & Membrane Transport.
- **Deficient Intracellular K+:**
  - o Cell membrane will be more Negative than normal (Ie: *Hyperpolarised*)
  - o Therefore it'll be harder to initialize an action potential as it takes more to reach threshold.
- **Excess Intracellular K+:**
  - o Cell membrane will be more Positive than normal (Ie: *Depolarised*)
  - o Therefore it'll be easier to initialize an action potential as it takes less to reach threshold.
- **Effect on the Heart:**
  - o The heart is particularly sensitive to K+ Levels
  - o Both Too High & Too Low K+ Levels will Disrupt Electrical Conduction of the Heart → Can be Fatal.
- **Regulating K+ Levels:**
  - o Relies solely on K+ Secretion by the ***"Principal Cells"*** in the Collecting Ducts of the Kidneys.
  - o **Principal Cells Detect [K+] in the Blood:**
    - § High Blood [K+] → K+ Secretion is Increased
    - § High Blood [K+] → K+ Secretion is Decreased
  - o **Adrenal Glands Detect [K+] in the Blood:**
    - § High Blood [K+] DIRECTLY Stimulates **Aldosterone** Release from Adrenal Cortex.
  - o **Aldosterone** → Activates Na+/K+-ATPase's in the Distal Tubules & Collecting Ducts:
    - § This Increases Reabsorption of Na+, Cl- & H2O from Distal Tubule→Interstitium
    - § But ALSO causes Secretion of K+ into the Filtrate.



The Renin-Angiotensin System (RAS): - Regulates Extracellular Fluid Volume & Systemic Blood Pressure

- **The Juxtaglomerular ("beside the glomerulus") Apparatus:**
  - The 'sensor' for the RAS.
  - A region in the Nephron containing 2 Types of Receptor Cells:
    - **1- Juxtaglomerular Cells:**
      - **Mechanoreceptors** – Detect Changes in Blood Pressure in Afferent Arteriole.
        - They are essentially enlarged Smooth-Muscle Cells
        - They contain Secretory Granules of 'Renin'.
      - **Release Renin** in response to:
        - *LOW BLOOD PRESSURE* in the *AFFARENT ARTERIOLE*. (Reduced Stretch – Maybe due to a significant drop in Systemic BP)
        - *DIRECT SYMPATHETIC STIMULATION* of JG-Cells (By Renal Sympathetic Nerves)
        - *ANGIOTENSIN-II* (Direct Stimulation of JG-Cells)
      - **Renin Release Leads To:**
        - Systemic Vasoconstriction (by Angiotensin-II) → Increase in Blood Pressure.
    - **2- Macula Densa:**
      - **Osmoreceptors** – Detect Osmolality of Distal Tubule Contents.
        - They are a modified epithelium of the Distal Tubule.
        - They are Tall & Densely packed (Compared to the normal Simple Cuboidal)
      - **Stimulate Renin Release** from JG-Cells in response to:
        - *HIGH FILTRATE OSMOLARITY.*
        - *HIGH FILTRATE FLOW RATE* (High flow rate gives the illusion of High Osmolality as more solutes come in contact with the cells per unit time.)
      - **Renin Release Leads To:**
        - Systemic Vasoconstriction (by Angiotensin-II)
          - Therefore Vasoconstriction of Renal Arteries
          - Therefore Decrease in GFR:
            - Decreases Filtrate Flow Rate
            - Decreases Filtrate Osmolality (as there is more time for solute reabsorption)
      - **Note: Macula Densa Also Plays a Role in "Tubuloglomerular" Autoregulation of GFR:**
        - High Filtrate Flow/Osmolality → Promotes Vasoconstriction of Afferent Arteriole
        - Low Filtrate Flow/Osmolality → Promotes Vasodilation of Afferent Arteriole





GENITOURINARY PATHOLOGY

GENERAL OVERVIEW OF RENAL PATHOLOGY

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Overview:

- **Renal Diseases are complex and are the result of abnormalities of one or more of the below:**
  - o Glomeruli
  - o Tubules
  - o Interstitium
  - o Blood vessels
- **Some of the above are more vulnerable to specific forms of injury**.
  - o Eg: Most Glomerular Diseases are *Immunologically Mediated.*
  - o Eg: Most Tubular Diseases are caused by *Toxic/Infectious Agents(Viruses/Bacteria).*
  - o Eg: Most Interstitial Diseases are caused by *Toxic/Infectious Agents (Viruses/Bacteria).*
  - o Note: Blood Vessel abnormalities are usually the result of one or more of the above.
- Note: Damage to one part always secondarily affects the others → Eventually leading to Chronic Renal Failure

Functional Reserve:

- Note: The Kidneys have a considerable *Functional Reserve:*
  - o You only need 1x Kidney to survive – (And even *it* has more function than the body needs)
  - o Therefore, with 2 kidneys, large-scale damage must occur for significant functional impairment.
  - o However, once the damage is done, it is irreversible and highly debilitating.

*4 Stages of Chronic Renal Failure:

Note: "Renal Failure" = Decreased Glomerular Filtration Rate (GFR)

- **1- Diminished Renal Reserve:**
  - o ***GFR = 50% of Normal**
  - o Blood Urea Nitrogen (BUN) – Normal
  - o Blood Creatinine – Normal
  - o – (Ie: Diminished functional reserve, but still enough to maintain bodily/blood homeostasis)
- **2- Renal Insufficiency:**
  - o ***GFR = 20-50% of Normal**
  - o Blood Urea Nitrogen (BUN) – Elevated
  - o Blood Creatinine – Elevated } "**Azotaemia**" (High levels of N-containing compounds)
  - o Anaemia – (↓[Hb] – Due to ↓Erythropoietin Release by Kidneys)
  - o Polyuria – (High Urine Output – due to poor H2O Retaining Abilities of damaged kidney)
  - o Hypertension – (due to fluid overload and production of vasoactive hormones)
- **3- Renal Failure:**
  - o ***GFR = <20% of Normal**
  - o Blood Urea Nitrogen (BUN) – Highly Elevated
  - o Blood Creatinine – Highly Elevated } "**Uraemia**" (More severe form of Azotaemia)
  - o Uraemia (Elevated Blood Urea) → Toxic to Brain & Nerves.
  - o Anaemia – (↓[Hb] – Due to ↓Erythropoietin Release by Kidneys)
  - o Polyuria – (High Urine Output – due to poor H2O Retaining Abilities of damaged kidney)
    - § Hypovolaemia
  - o Electrolyte Imbalances (K+, HPO_4, Ca^+)
    - § Hyperkalaemia (↑K+)
    - § Hyperphosphataemia (↑HPO4) – (Phosphate Retained by Failing Kidneys)
    - § Hypocalcaemia (↓Ca+) – (Due to the effects of Hyperphosphataemia & Poor activation of Vit-D in the kidney → $CaPO_4$ Deposition in Tissues & Poor Ca^+ Absorption in GIT – (As Active Vit-D is needed for Ca+ Absorption))
  - o Osteoporosis – (Due to Hypercalcaemia – {resulting from High Phosphate})
  - o Haematuria - (Blood in Urine)
- **4- End-Stage Renal Disease:**
  - o ***GFR = <5% of Normal**
  - o Terminal stage of **Uraemia**

The 2 Greatest Risk Factors For Renal Disease:

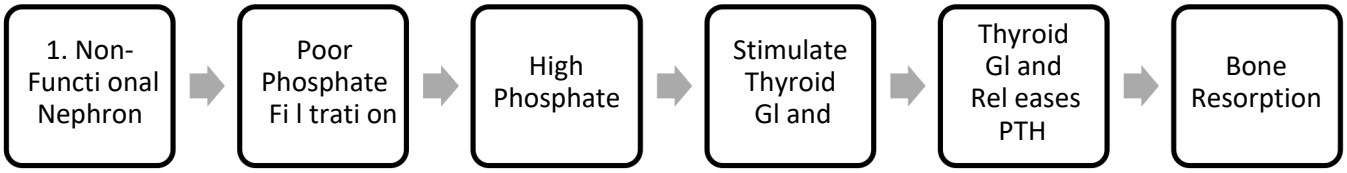
- **Hypertension** → Damage to Glomerular Capillaries → Sclerosis & Thickening of Capillary Wall → Tubular
- Necrosis → Inflammatory Response → Further Renal Disease
  Diabetes → ↑[Blood Glucose] → Blood proteins become *sticky* → deposit in small blood vessels → Vessel Inflammation, Damage & Scarring → Tubular Necrosis → Inflammatory Response → Further Renal Disease

Clinical Complications of Renal Disease

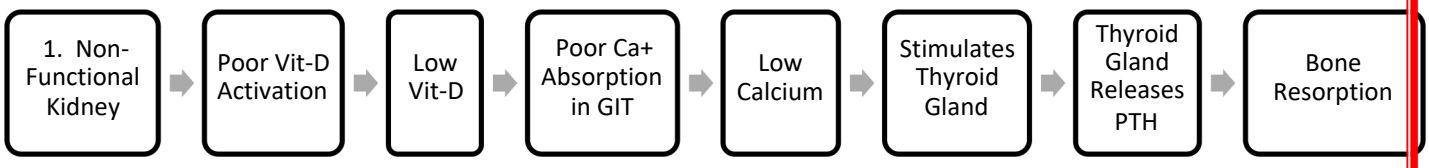
Electrolyte Imbalances:

- **Hyperphosphataemia (↑Phosphate):**
  - Blood Phosphate (which is usually removed by Kidney) is retained due to poor GFR.
    - →CaPO4 Deposition in Tissues (Tissue Calcification)
    - →Stimulates Thyroid Gland to secrete ParaThyroid-Hormone (PTH) → Bone Resorption.
- **Hyperkalaemia (↑Potassium):**
  - Blood Potassium (which is usually secreted into lumen) isn't being secreted because Nephrons are non-functional.
    - →Palpitations (Arrhythmias)
    - →Possible Death from Heart Failure
- **Hypocalcaemia (↓Calcium):**
  - Because The Active Form of Vitamin-D required for Ca+ Absorption in GIT (Which is usually produced by the kidney) Isn't being produced → Poor Ca+ Absorption in GIT.
    - →Stimulates Thyroid Gland to secrete ParaThyroid-Hormone (PTH) → Bone
    - Resorption (To Try to Increase Blood-Calcium Levels).
    - → Urinary Calculi
      →Arrhythmias

- **Renal Bone Disease:**
  - **Damaged Kidney Doesn't Remove Phosphate from Blood & Doesn't Produce Active Vitamin-D:**
    - **→ Hyperphosphatemia:**



    - **→ Poor Ca+ Absorption in GIT → Hypocalcaemia:**



- **Haem atologic Com plications:**
  - Anaemia (Due to ↓Erythropoietin → ↓RBC Synthesis)

- **Dehydration:**
  - Due to loss of kidney's ability to concentrate urine (Ie: Poor Reabsorption of Water)

- **Uraemia:**
  - High Blood Urea & Creatinine
    - → Toxic to Brain & Nerves
    - → Irritates the GIT → Vomiting & Nausea
    - → Can cause Uraemia Pericarditis (Deposition of Urea in Pericardium → Inflammation)
    - → Itch (Urea Excretion through skin)

CONGENITAL KIDNEY ABNORMALITIES

CONGENITAL KIDNEY ABNORMALITIES

Get Direction GLOBAL

Up to 10% of people are born with congenital malformations of the renal system – Some cause disease, some don't.

AGENESIS OF THE KIDNEY:

- **What is it?**
- o Agenesis = An absence of one or both kidneys at birth.

Risk Factors:

o More common in Intrauterine Growth Retardation (IUGR)
o More common in multi-gestations (Eg: Twins/triplets)

- **Implications:**
  - o Bilateral is incompatible with life and usually encountered in stillborn infants
  - o Unilateral is compatible with normal life if no other abnormalities exist
  - o Often associated with many other congenital disorders (limb defects, hypoplastic lungs) and leads to early death
- **Diagnosis:**
  - o Usually diagnosed on fetal ultrasound.
  - o The opposite kidney is usually enlarged as a result of compensatory hypertrophy
- **Prognosis:**
  - o Most kids with unilateral renal agenesis live normal lives.
  - o Some pts eventually develop progressive glomerular sclerosis in remaining kidney as a result of the adaptive changes in hypertrophied nephron and in time chronic kidney disease ensues

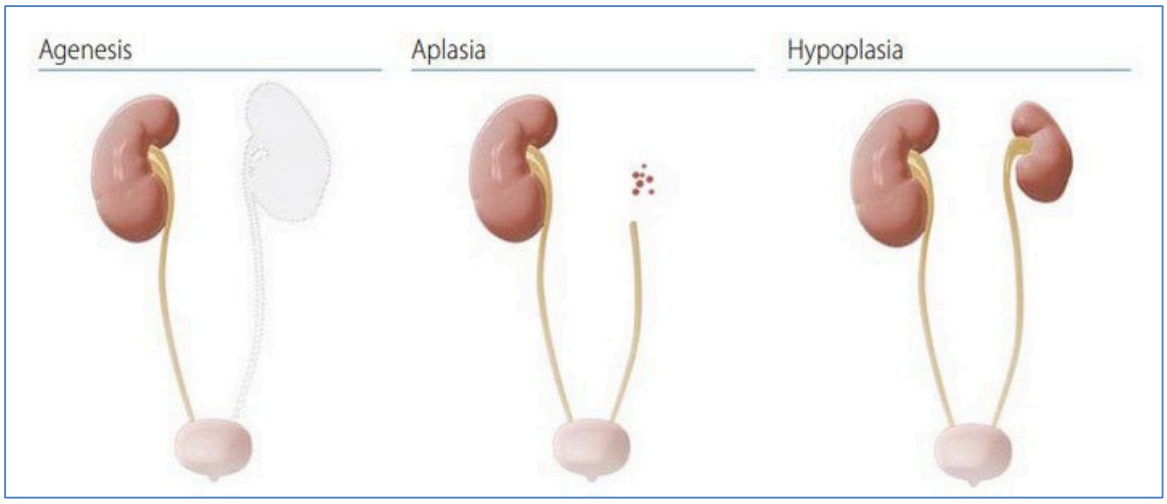
RENAL HYPOPLASIA:

- **What is it?**
- o Refers to failure of the kidneys to develop to a normal size

Risk Factors:

o Family history/genetics
o Certain medications in pregnancy

- **Implications:**
  - o Usually unilateral but may occur bilaterally resulting in renal failure in early childhood
  - o Higher risk of UTI's and HTN as an adult.
- **Diagnosis:**
  - o Antenatal ultrasound scan.
  - o Sometimes only picked up later in childhood.
- **Prognosis:**
  - o Depends on the presence/degree of chronic kidney impairment.



https://www.cdc.gov/ncbddd/birthdefects/surveillancemanual/quick-reference-handbook/congenital-anomalies-genital-urinary-organs.html

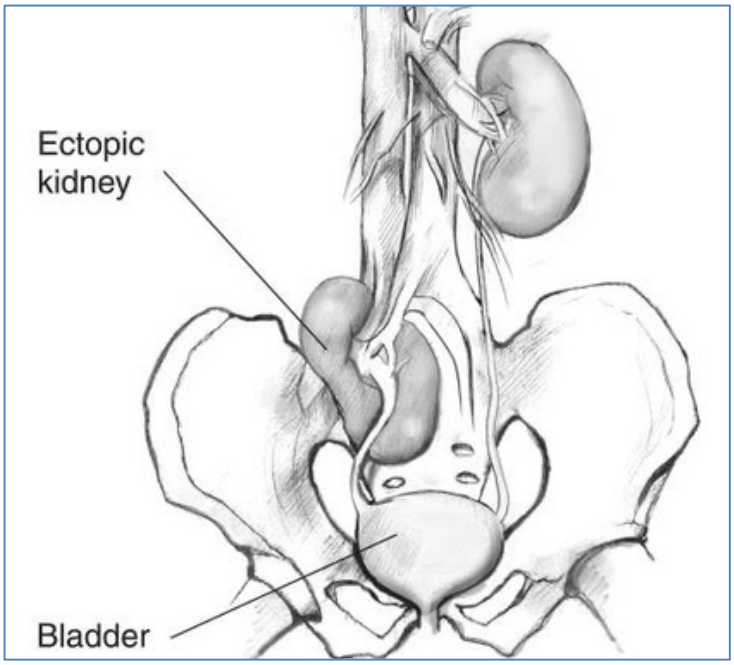
ECTOPIC KIDNEYS:

- **What is it?**
- o A birth defect in which a kidney is located in an abnormal position.

Implications:

- o Most people have no issues/symptoms.
- o Ectopic kidneys are usually normal or slightly small in size but otherwise not remarkable
- o Some ectopic kidneys may cause obstructive symptoms, predispose to UTIs & kidney stones.
- o Ectopic kidney is more vulnerable to trauma due to location

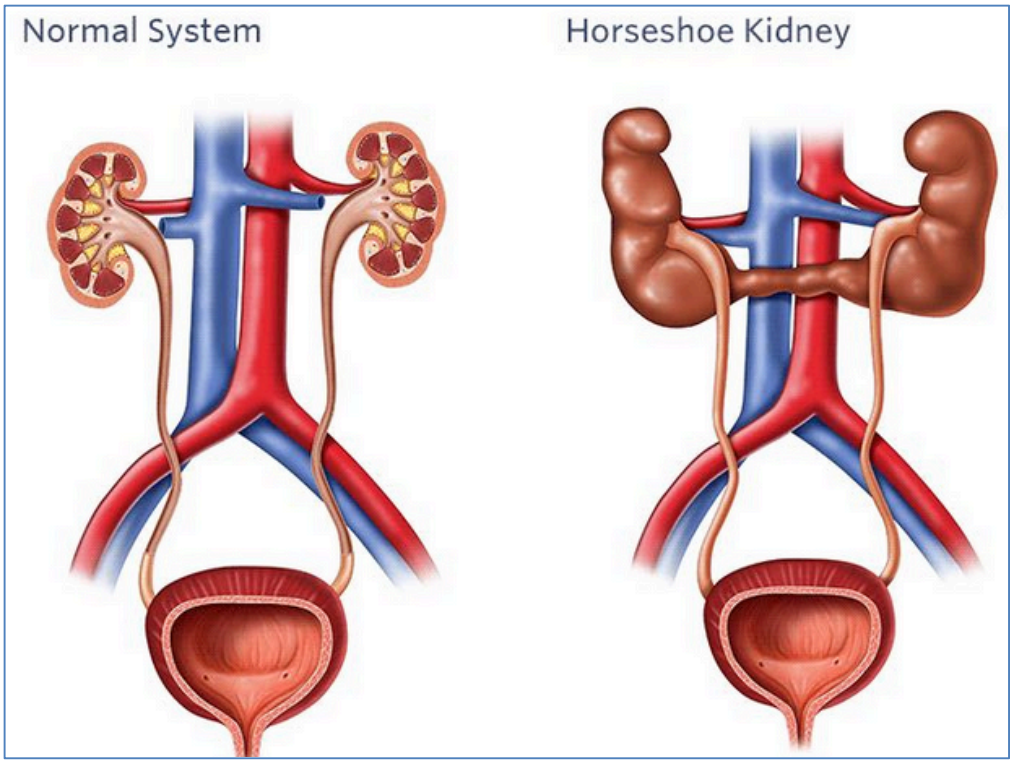
- **Diagnosis:**
  - o Ultrasound/Xray/CT
  - o Ectopics typically Lie either just above the pelvic brim or sometimes within the pelvis
- **Prognosis:**
  - o Generally no treatment necessary unless obstructive symptoms or other complications.



https://www.niddk.nih.gov/health-information/kidney-disease/children/ectopic-kidney

HORSESHOE KIDNEYS:

- **What is it?**
  - Fusion of the upper or lower poles of the kidneys produces a horseshoe-shaped structure that is continuous across the midline anterior to the great vessels
- **Risk Factors:**
  - Thought to be genetic
  - Associated with Turner's Syndrome & Edward Syndrome.
- **Prevalence:**
  - Common; found in about 1 in 500-1000
  - 90% of such kidneys are fused at lower pole, 10% fused at upper
- **Implications:**
  - May be asymptomatic
  - Some cause Abdo pain
  - Predisposed to kidney stones
  - Predisposed to UTI's
  - Must avoid contact sports
- **Diagnosis:**
  - Renal ultrasound
- **Prognosis:**
  - Typically doesn't affect life-expectancy.

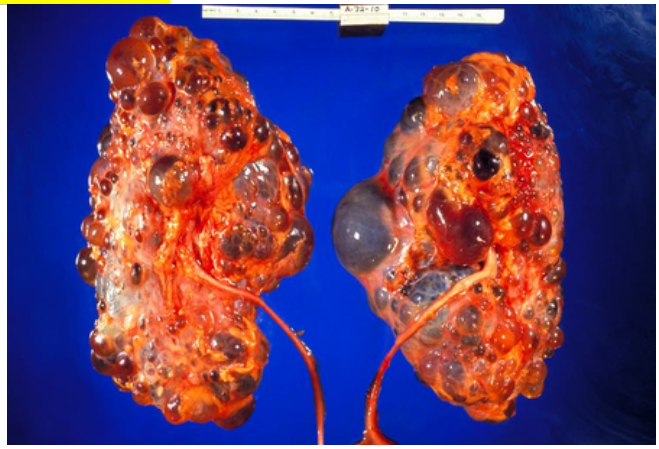


Children's hospital of Philadelphia: https://www.chop.edu/conditions-diseases/horseshoe-kidney

- **2 Types (& Modes of Inheritance):**
  - o Autosomal Dominant (Adult Variety)
  - o Autosomal Recessive (Childhood Variety)
- **Cysts:**
  - o = Bulging, filtrate-filled pouches of kidney.
  - o Caused by a Nephron not connecting to any collecting duct – (Ie: Filtrate has nowhere to go → Expands & Expands)
- **Clinical Features:**
  - o Abdominal Discomfort/Pain
  - o Haematuria (Blood in Urine – Eg: If a cyst ruptures)
  - o UTI's
  - o Renal Insufficiency:
    - § - Elevated Serum Creatinine
    - § - Anaemia – (↓[Hb] – Due to ↓Erythropoietin Release by Kidneys)
    - § - Polyuria – (High Urine Output – due to poor Concentrating Abilities of damaged kidney)
    - § - Hypertension

Adult: *AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD)*:

- **Aetiology:**
  - o Genetic – Autosomal Dominant
  - o (:. Fairly Common - 1:1000)
- **Pathogenesis:**
  - o Many tubules don't empty into Calyces → Obstruction → Cysts
- **Morphology:**
  - o Bilateral, large cystic kidney
  - o Some areas of Haemorrhage
  - o Some normal kidney tissue between cysts
- **Clinical Features:**
  - o **Onset** @ 30-40yrs
  - o **Symptoms:**
    - § Abdo/Flank pain (Stretching of the Renal Capsule → Pain)
    - § Intermittent Gross Haematuria (Cyst Rupture)
    - § Hypertension & Oedema (Fluid Retention)
- **Complications:**
  - o → **UTI**
  - o → **Renal Failure**/End Stage Disease At ~50yrs
  - o **Associated Features:**
    - § **Liver Cysts (30%)**
    - § **Cerebral Berry Aneurysms (20%)**
- **Treatment:**
  - o ***Dialysis/Kidney Transplant***



CDC/ Dr Edwin P. Ewing, Jr., Public domain, via Wikimedia Commons

Infantile: *AUTOSOMAL RECESSIVE POLYCYSTIC KIDNEY DISEASE (ARPKD)*:

- **Aetiology:**
- o Genetic – Autosomal Recessive (:.Very Rare – 1:30000)
- **Pathogenesis:**
- o 100% of Tubules are Affected :. Worse prognosis

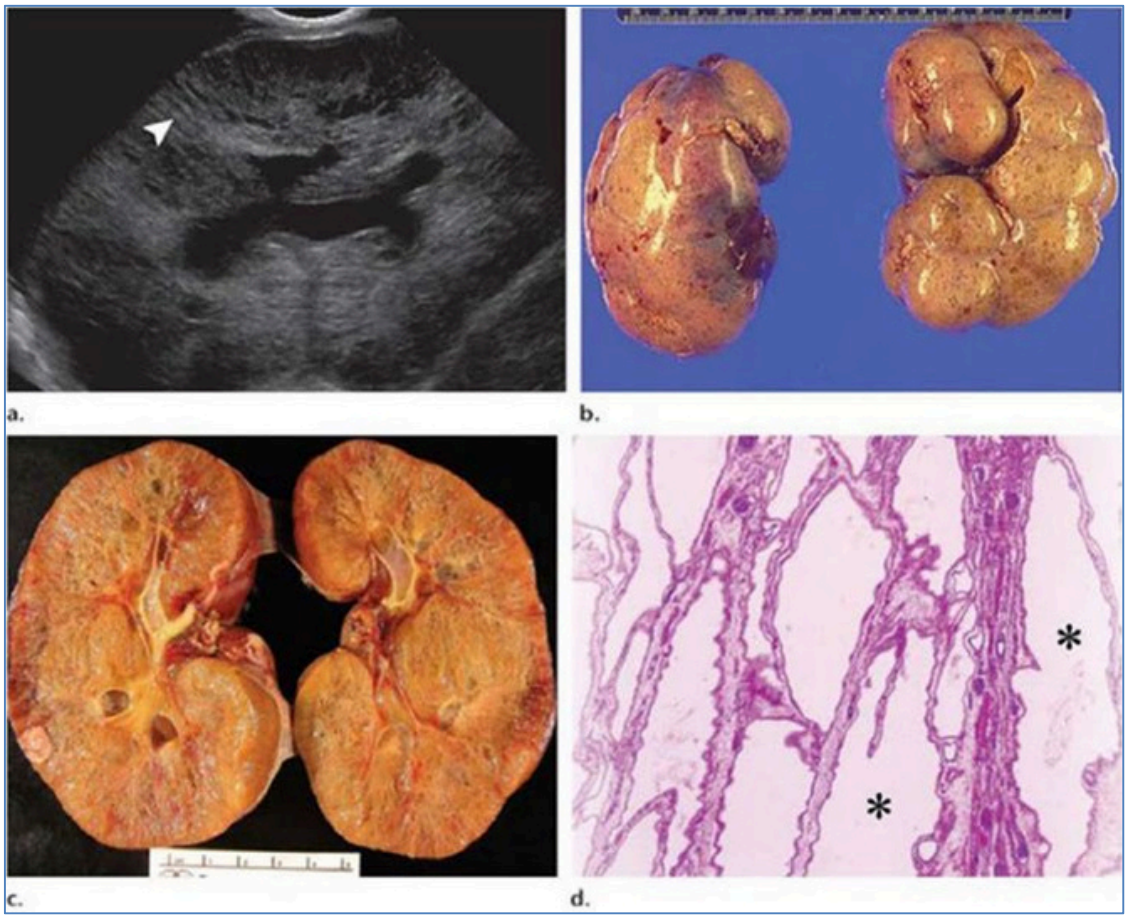
Morphology:

- o Regularly arranged, Spongy Kidney

Clinical Features:

- o Enlarged, Palpable Kidneys soon after birth (Bilateral Abdo Masses)
- o Poor Urinary Concentrating Ability
- o Metabolic Acidosis
- o Hypertension
- o Progression to ESRD by 15yrs

- **Poor Life Expectancy:**
- o 50% of Neonates Die
- o Most Surviving Babies develop End-Stage Kidneys by 15yrs
- **Treatment:**
- o ***Dialysis***
- o ***Kidney Transplant***



Patil A, Sweeney WE Jr., Avner ED, et al. Childhood Polycystic Kidney Disease. Available from: https://www.ncbi.nlm.nih.gov/books/NBK373381/figure/fig2_1/ doi: 10.15586/codon.pkd.2015.ch2

URINARY INCONTINENCE:

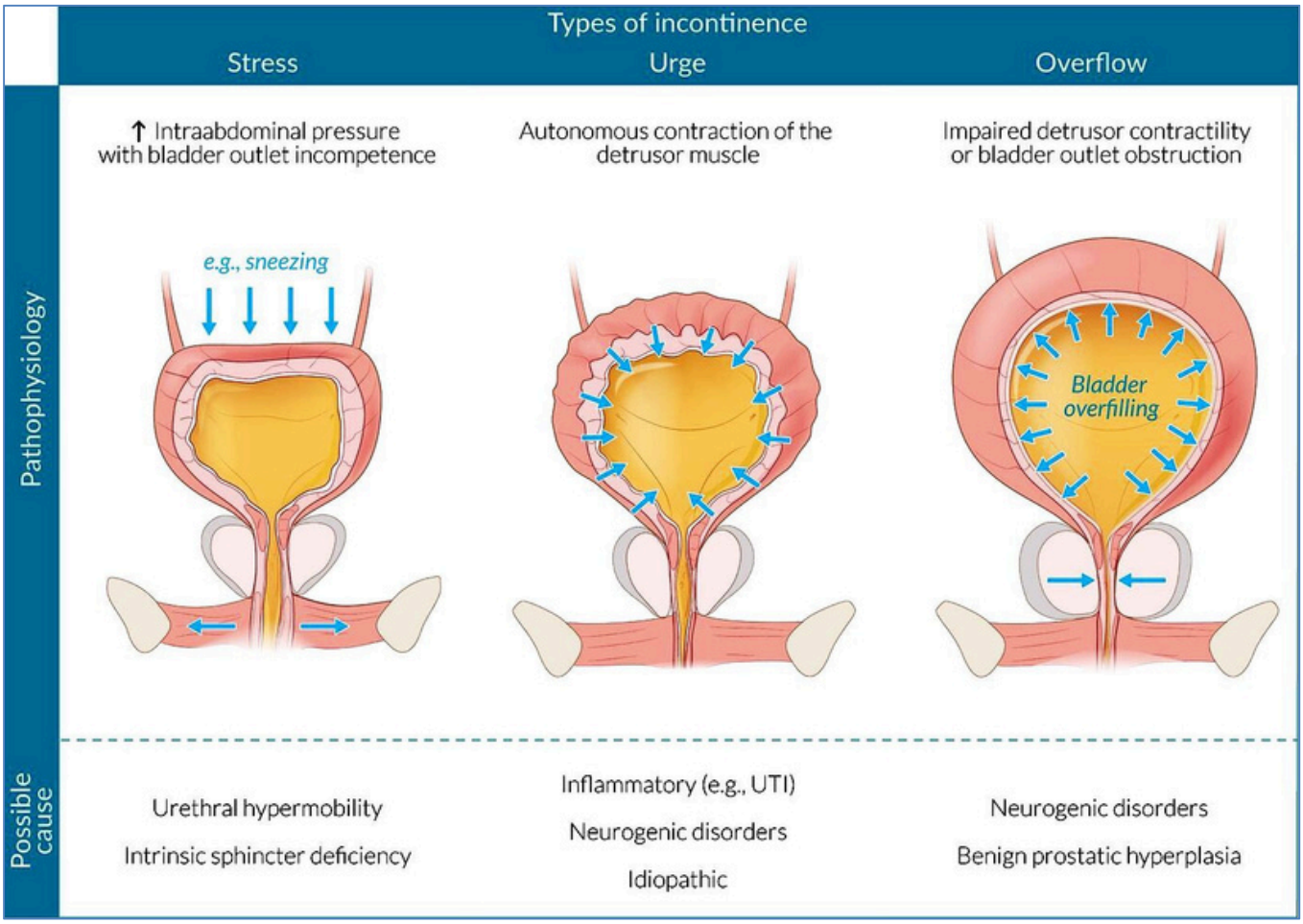
URINARY INCONTINENCE:

Urinary Incontinence = Inability to maintain micturition control → involuntary urine leakage

OVERFLOW INCONTINENCE:

- **Aetiology:**
  - Urinary Flow Obstruction (Eg: BPH, Prostate cancer, Urethral strictures, Cystocoele, uterine prolapse)
  - Detrusor Muscle disorder (Eg: Diabetic neuropathy, spinal cord injury, cauda equina syndrome, anticholinergics)
- **Pathogenesis:**
- Urinary retention → bladder pressure increases, exceeds urethral resistance

  Clinical Features:
  - Frequent loss of small amount of urine;
  - hesitancy;
  - weak/intermittent urinary stream
- **Diagnosis:**
  - Urologic History
  - Urodynamic studies
  - Abdo USS to identify anatomical anomalies
- **Treatment:**
  - Cholinergic agents (to increase bladder muscle tone)
  - Alpha blockers (Eg: Prazosin, tamsulosin → Relax bladder neck smooth muscle)
  - Surgery (if indicated by urologist/gynaecologist)
  - Intermittent self-catheterisation



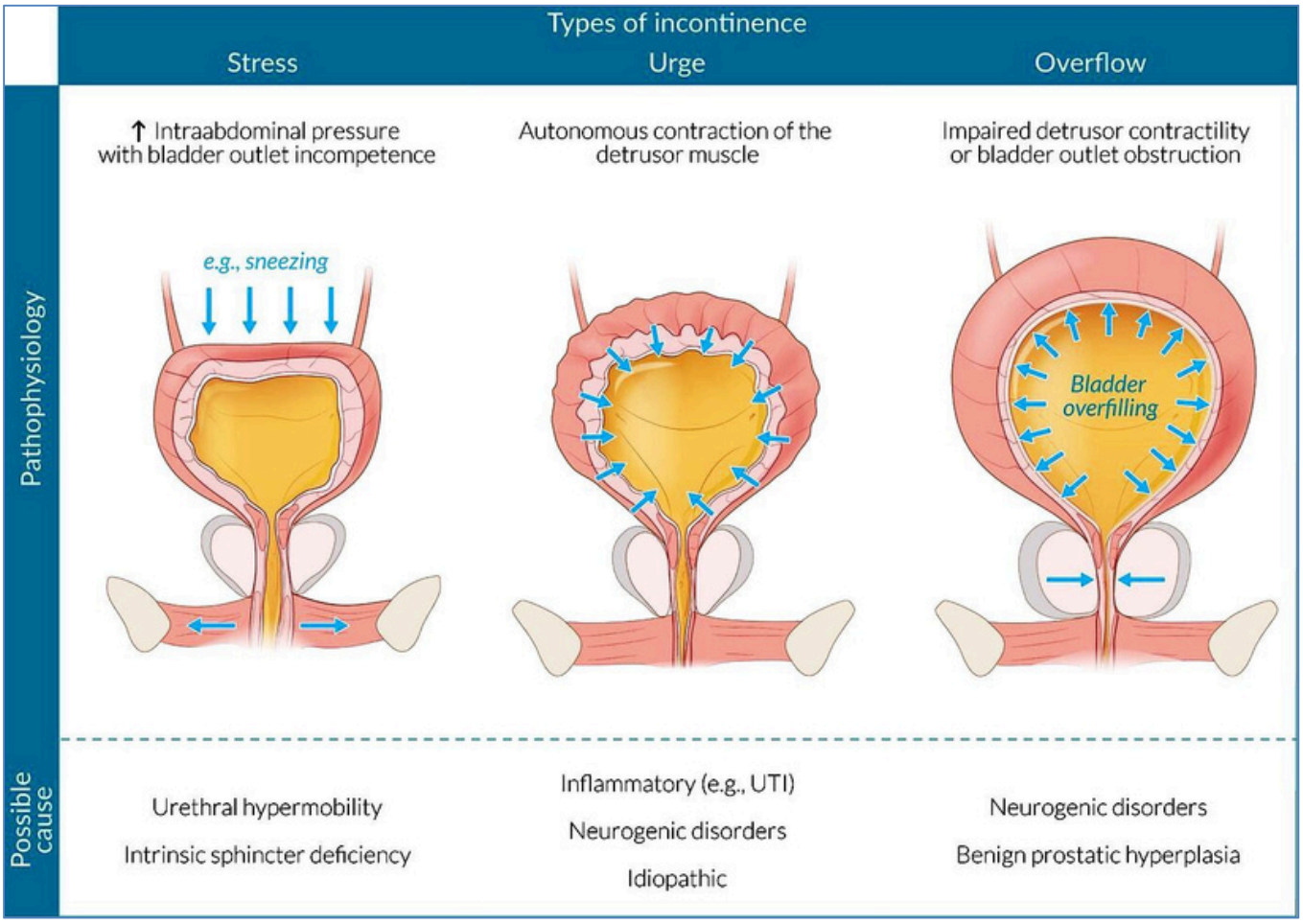
https://www.amboss.com/us/knowledge/Urinary_incontinence

STRESS INCONTINENCE:

- **Aetiology:**
  - o Pelvic Floor Weakness/laxity
  - o Most prevalent in Females <70yrs.
  - o Risk factors (Female, menopause, multiparity, pregnancy, obesity, previous pelvic surgery)
- **Pathogenesis:**
  - o Pelvic floor laxity → urethra loses support → increase in intra-abdominal pressure → overwhelms sphincter muscles
- **Clinical Features:**
- o Spurts of urine when intra-abdominal pressure increases (Eg: sneeze, cough, laugh, exercise)

Diagnosis:

- o Abdo USS to identify anatomical anomalies
- o Urodynamic studies
- o Urologic History
- **Treatment:**
  - o Oestrogen replacement therapy (HRT) for stress incontinence caused by menopause
  - o Lifestyle changes (weight loss)
  - o Kegel exercises (strengthens external sphincter and pelvic floor muscles)
  - o Surgery (Eg: Sling procedures)



https://www.amboss.com/us/knowledge/Urinary_incontinence

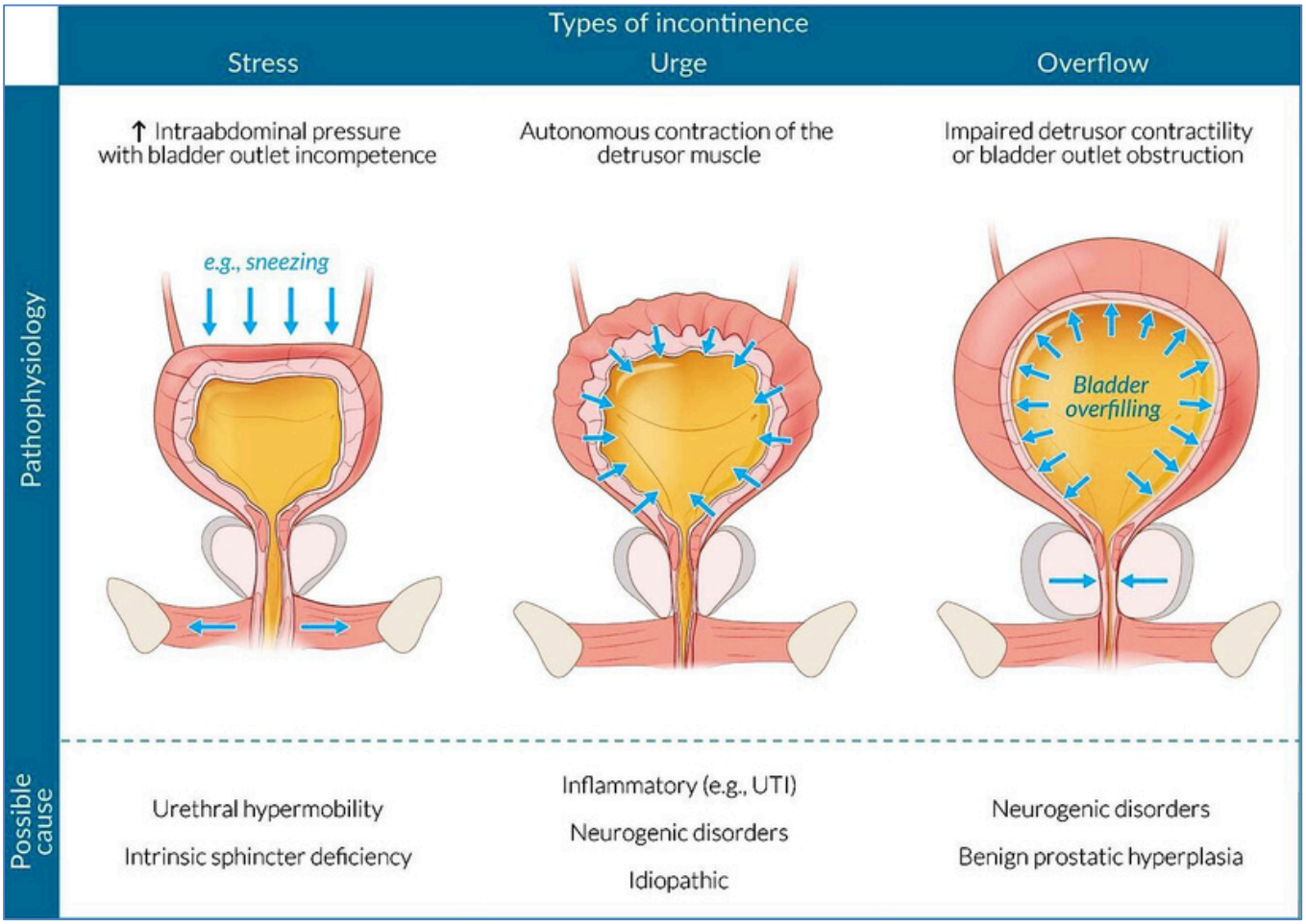
URGE INCONTINENCE:

- **Aetiology:**
  - Overactive Bladder (AKA: Detrusor Instability)
- **Pathogenesis:**
  - uninhibited detrusor muscle contracts randomly → Unintentional voiding

Clinical Features:

- Sudden/great urine leakage,
- strong/ immediate urge to void;
- frequency;
- nocturnal wetting

- **Diagnosis:**
  - Abdo USS to identify anatomical anomalies
  - Urodynamic studies
  - Urologic History
- **Treatment:**
  - Anticholinergic agents → inhibit detrusor overactivity by blocking muscarinic receptors
  - Tricyclic antidepressants (TCAs) → anticholinergic properties
  - Cystoscopic Injections with botulinum toxin → decrease detrusor muscle activity
  - Bladder Training
  - Kegel exercises
  - Sling procedures



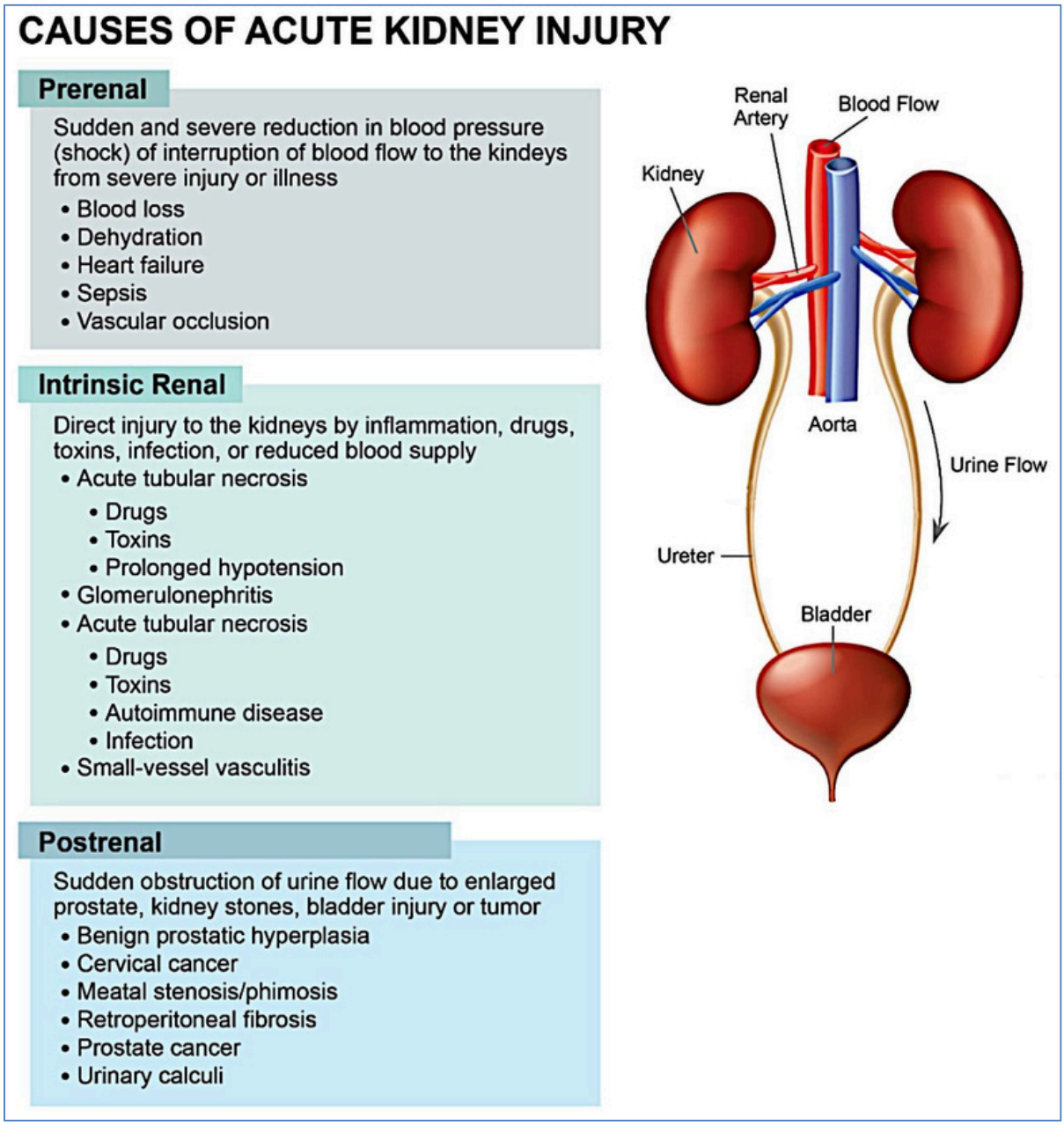
https://www.amboss.com/us/knowledge/Urinary_incontinence

ACUTE RENAL FAILURES

ACUTE RENAL FAILURES:

Acute Renal Failure – General Information:

- **Aetiology:**
  - ***= "Rapid loss of kidney function"***
  - **1- *Pre-Renal* Renal Failure: -** *Before the Blood Reaches the Kidney* (Ie: ↓Glomerular Perfusion)
    - § Eg: Hypovolaemia (Eg: Blood Loss)
    - § Eg: Decreased cardiac output (Eg: Heart Failure)
    - § Eg: Renal artery obstruction (Eg: Embolism)
  - **2- *Intra-Renal* Renal Failure -** The ***kidney itself is damaged***
    - § Eg: *Acute glomerular nephritis*
    - § Eg: *Tubular diseases* Eg: acute tubular necrosis
    - § Eg: *Interstitial diseases* Eg: auto immune disorders such as SLE
    - § Eg: *Vascular diseases* Eg: polyarteritis nodosa
  - **3- *Post-Renal* Renal Failure -** Due to ***outflow obstruction*** from the kidneys
    - § Eg: Cancer – Bladder / Prostate / Ureteric / Cervical
    - § Eg: Blood clot
    - § Eg: Calculi (Kidney stones – Bilateral)
    - § Eg: Accidental surgical ligation



J. Clin. Med. 2020, *9*(4), 1104; https://doi.org/10.3390/jcm9041104

- **Common Clinical Features:**
  - o **Uraemia** – (Fatigue, Malaise, Anorexia, Headache, Nausea, Vomiting)
  - o **Hyperkalaemia** → Brady-Arrhythmias
  - o **Fluid Retention** → Oedema (Peripheral & Pulmonary)
    - →...& RARELY, Hypertension & Cardiac Tamponade.
  - o **Haematuria** – Painless (Cancer) or Painful (Stones/LUTS)
  - o **Flank pain** (in specific conditions – Particularly Inflammatory or Ischaemic)

Clinical Complications of Renal Disease

- **General Effects/Problems Encountered in Renal Failure:**
  - o (Recall the functions of the kidney and then infer what happens when they are eliminated!)
    - § Acid Base Balance (Renal Failure → ***Met. Acidosis***)
    - § Electrolyte Balance (Renal Failure → ***Na+ & K+ Retention***)
    - § Fluid Balance (Renal Failure → ***Fluid Overload***)
    - § ↓Erythropoiesis (Renal Failure → ***Anaemia***)
    - § Renin Angiotensin System ***Renal Hypertension***
    - § Calcium Metabolism (Renal Failure → ***Osteoporosis & 2oHyper-Parathyroidism***)
    - § ***Uraemia***
    - § ↓Urine Output
    - § ↓Toxin Excretion (Renal Failure → Accumulation of ***Urea & Creatinine***)

Investigations:

- **Blood Urea:Creatinine Ratio – Distinguishing Between Intra/Pre/Post-Renal Failure:**
  - o Normal = 40:1 – 100:1
  - o Lower U:Cr = Likely *Intra-Renal* Failure
  - o Raised U:Cr = Either *Pre- OR Post-Renal* Failure
  - o (Note: Even though the absolute concentrations of Urea & Creatinine are raised in all types of renal failure, the **ratio** is useful in differentiating Intra-Renal Failures from Pre/Post-Renal Failures

Location	Urea: Cr	BUN: Cr
Pre-renal	>100:1	>20:1
Normal or Post-Renal	40-100:1	10-20:1
Renal	<40:1	<10:1

Test	Pre-renal	Renal (ATN)
BUN: Cr Ratio	>20:1	<10:1
Urea: Cr ratio	>100:1	<40:1
Urine Na	<20	>40
Urine osmolality	>500**	<350
Urine SG	>1.020	<1.010
Fractional excretion	<1%	>2%
Urinalysis	Normal or hyaline casts	Granular casts, tubular epithelial cells

**SIADH if urine Na high

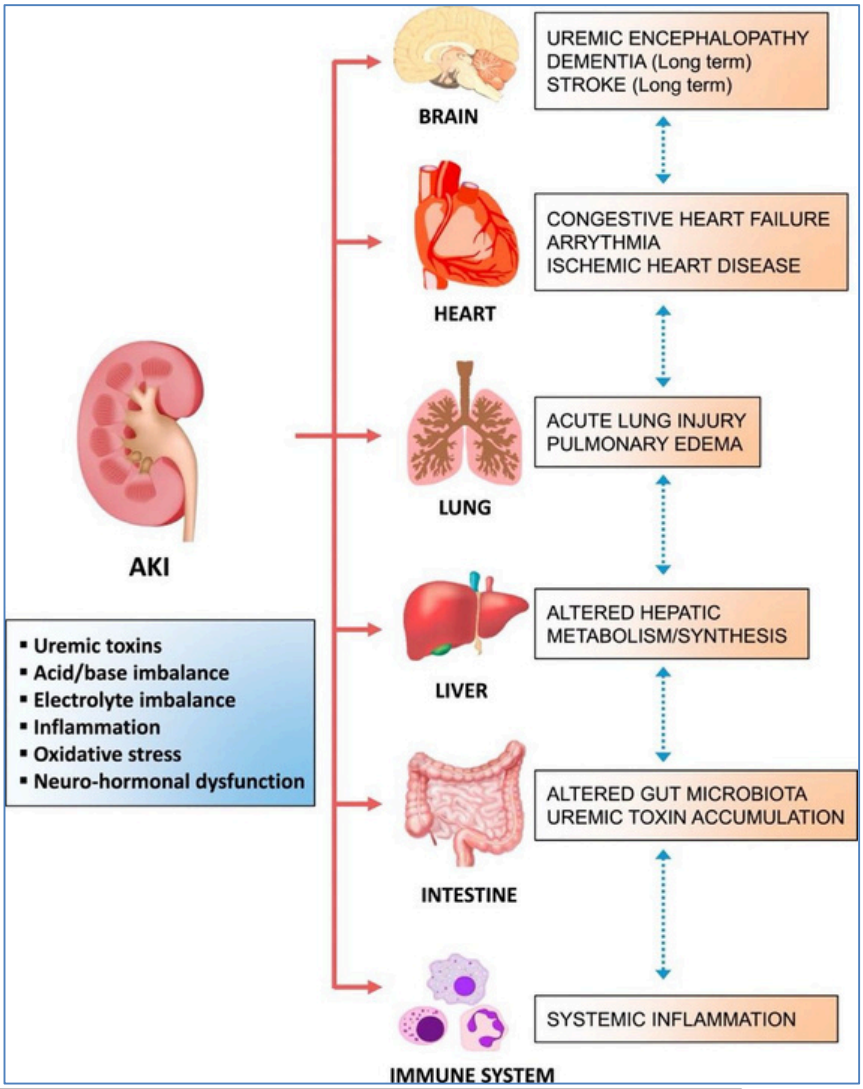
- **Urine Protein:Creatinine Ratio – Is there Proteinuria?**
  - o **Interpretation:**
    - § **Daily Creatinine Excretion is Constant, therefore a raised Pr:Cr ratio indicates an excess**
    - § **of ↑ Protein in Urine = Proteinuria**
    - § 30-300mg = **Microalbuminuria**
    - § >300mg = **Macroalbuminuria/"Proteinuria":**
      >3000mg = **Nephrotic Syndrome**

PRE-RENAL FAILURES

PRE-RENAL FAILURES

GENERAL INFO:

- **Aetiology:**
  - o ***Anything that ↓ Bloodflow to the Kidneys…Eg:***
    - § Hypovolaemia (Diarrhoea/Haemorrhage/Vomiting/Burns)
    - § Shock (Hypotension)
    - § Heart Failure (CCF/Ascites)
    - § Renal Artery/Vein Thrombosis/Stenosis
    - § Etc
- **Pathophysiology:**
  - o Renal Hypoperfusion → ↓GFR → Kidney Failure
    →Renal Ischaemia → Infarction of Tubules → ↓Kidney Function
- **Clinical Features:**
  - o Acute kidney injury (AKI)
  - o ↓GFR
    - § → Oliguria/Anuria
    - § → Uraemia/Azotaemia → Fatigue, Malaise, Headache
    - § → ↑Creatinine
  - o Thirst & Dehydration – if due to Fluid Depletion.
- **Complications:**
  - o Complete Renal Failure
  - o Other Multi-Organ Failure (if Shock)
  - o AKI Can lead to multiple system complications:



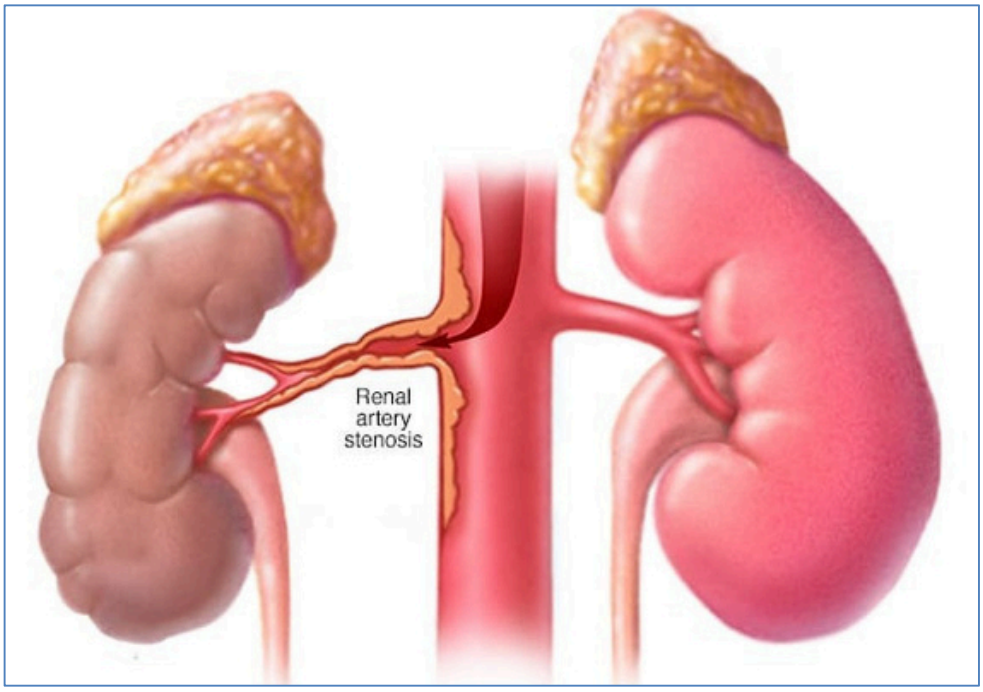
Lee, Sul A. et al. "Distant Organ Dysfunction in Acute Kidney Injury: A Review." *American journal of kidney diseases : the official journal of the National Kidney Foundation* 72 6 (2018): 846-856 .

RENAL ARTERY STENOSIS:

- **Aetiology:**
  - Typically atherosclerosis
  - Also sometimes Fibromuscular Dysplasia (females)
- **Pathophysiology:**
  - Progressive narrowing of renal artery → decrease in renal blood flow
    - → renin release by juxtaglomerular cells
    - → angiotensin II, aldosterone
    - → vasoconstriction, increased reabsorption of sodium, water
  - Contraction of blood vessels, increase in blood volume → blood pressure (BP) elevation
- **Clinical Features:**
  - Sudden onset of severe hypertension
    - Headaches
    - Blurred vision
  - Hypertension refractory to medications
  - Renal Bruit on abdo auscultation.
- **Diagnosis:**
  - Renal Arteriogram
  - o Renal Ultrasound + Doppler
  - MRA (magnetic resonance angiogram) if CT contrast is contraindicated

CT-KUB with contrast

- **Treatment:**
  - HTN - ACE inhibitors/Calcium channel blockers
  - Surgery – Renal angioplasty or bypass surgery.
- **Complications:**
  - Secondary hypertension (renovascular HTN)
  - Pre-renal failure
  - Renal Atrophy/fibrosis



https://www.stclair.org/services/mayo-clinic-health-information/diseases-and-conditions/CON-20305542/

RENAL CORTICAL NECROSIS:

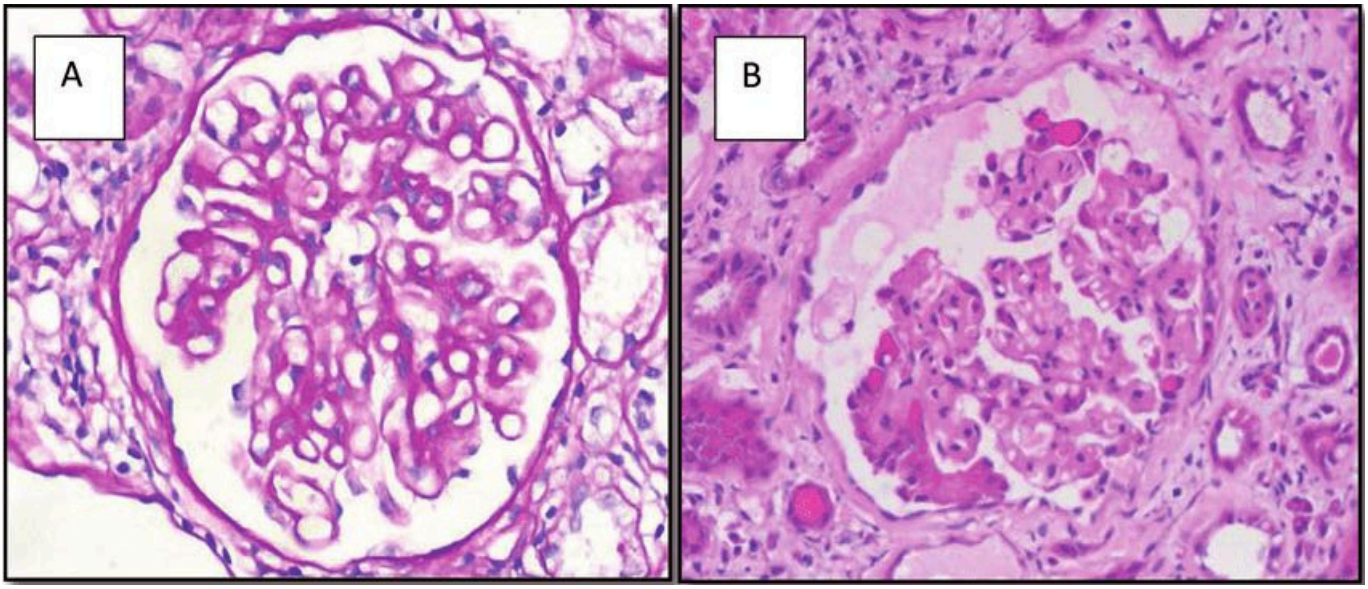
- **Aetiology:**
  - Sudden decrease in blood perfusion to renal cortex
    - Eg: Blood clots (Eg: DIC)
    - Eg: Vasospasm
    - Eg: Septic shock
- **Pathophysiology:**
  - Reduced blood supply to renal tubules → acute tubular necrosis
  - If ischemia persists → irreversible necrotic injury of renal cortex → renal cortical necrosis
- **Clinical Features:**
  - Sudden decrease in urine output (Oliguria/anuria)
  - Flank pain at costovertebral angle
- **Diagnosis:**
  - Non-contrast CT
  - Bloods (Elevated urea/creatinine, Hyperkalaemia, metabolic acidosis)
  - Ultrasound
  - Urine (Haematuria, proteinuria, casts)
  - Biopsy (Patchy necrosis; atrophy of renal cortex)
- **Treatment:**
  - IV Fluids
  - Treat underlying cause
  - May require dialysis if severe
- **Complications:**
  - Acute kidney failure

INTRA-RENAL FAILURES

GLOMERULONEPHRITIS:

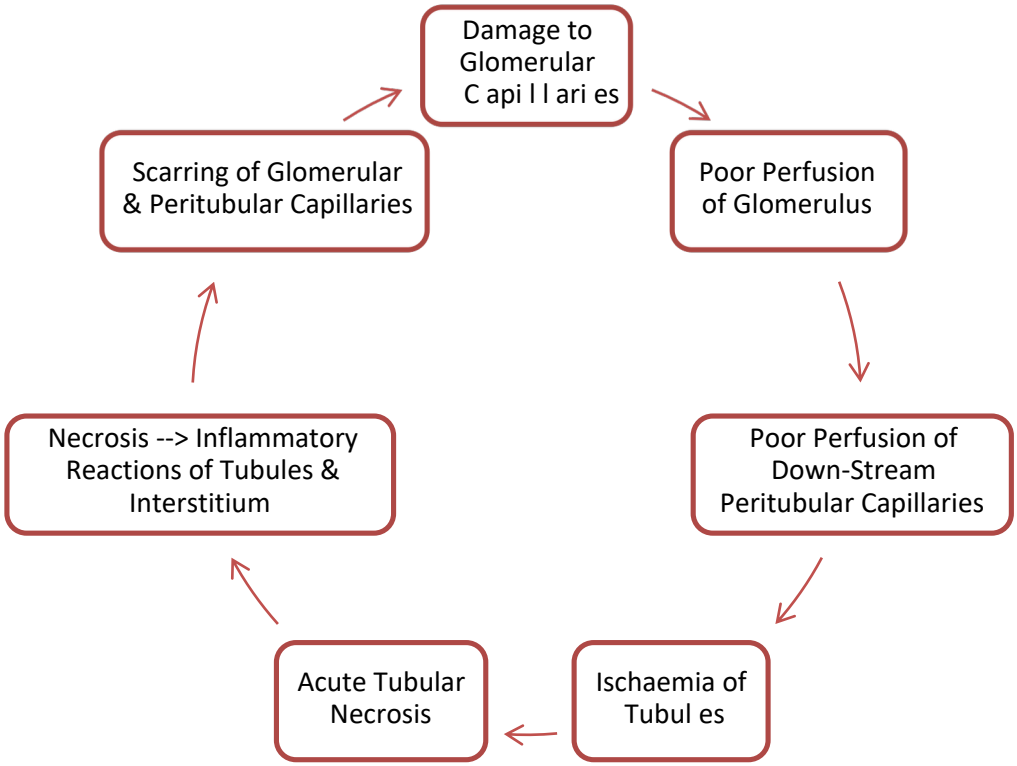
- **Aetiology:**
  - o May be post-infective (Eg: URTI/streptococcus/etc)
  - o Or may be Autoimmune
- **Pathophysiology:**
  - o **1- Antibody-Mediated Injury:**
    - § ***Antibody-Antigen Complexes*** form in the Glomerulus → Adheres to Capillary Wall → Causes Inflammation → Infiltration of Leukocytes → Attack the Basement Membrane of Glomeruli → Damage to Glomeruli → Subsequent Damage to Nephron, Vessels & Interstitium.
    - § ***Circulating Infectious/Toxic Agents*** Deposit in Glomerulus → Causes Inflammation → (Same as above) (Eg: Streptococcal Infections)
  - o **2- Cell-Mediated Injury:**
    - § Typically the reaction to an *Antibody-Antigen Complex* (As seen above)
  - o **3- Complement-Mediated Injury:**
    - § Complement (Cell-killing proteins released in inflammation) → Cause Glomerular Damage.
  - o **IF *Incomplete* Glomerular-Membrane Damage → NEPHROTIC SYNDROME:**
    - § → Selective Albuminuria, Proteinuria, (But NO Haematuria)
  - o **IF *Complete* Glomerular-Membrane Damage → NEPHRITIC SYNDROME:**
    - § → Oliguria (due to ↓↓Filtration), Haematuria & Hypertension
- **3 Basic Histological Alterations in Glomerulonephritis:**
  - o ***1- Hypercellularity:***
    - § Proliferation of Endothelial Cells
    - § Proliferation of Epithelial Cells
    - § Leukocyte Infiltration
    - § 'Crescents' of proliferating Epithelial Cells/Leukocytes.
  - o ***2- Basement Membrane Thickening:***
    - § Thickening of the Membrane between Endothelium of Capillaries & Podocytes of Bowman's Capsule.
  - o ***3- Hyalinization & Sclerosis (Scarring):***
    - § Accumulation of deposited Protein (Proteinaceous Material)

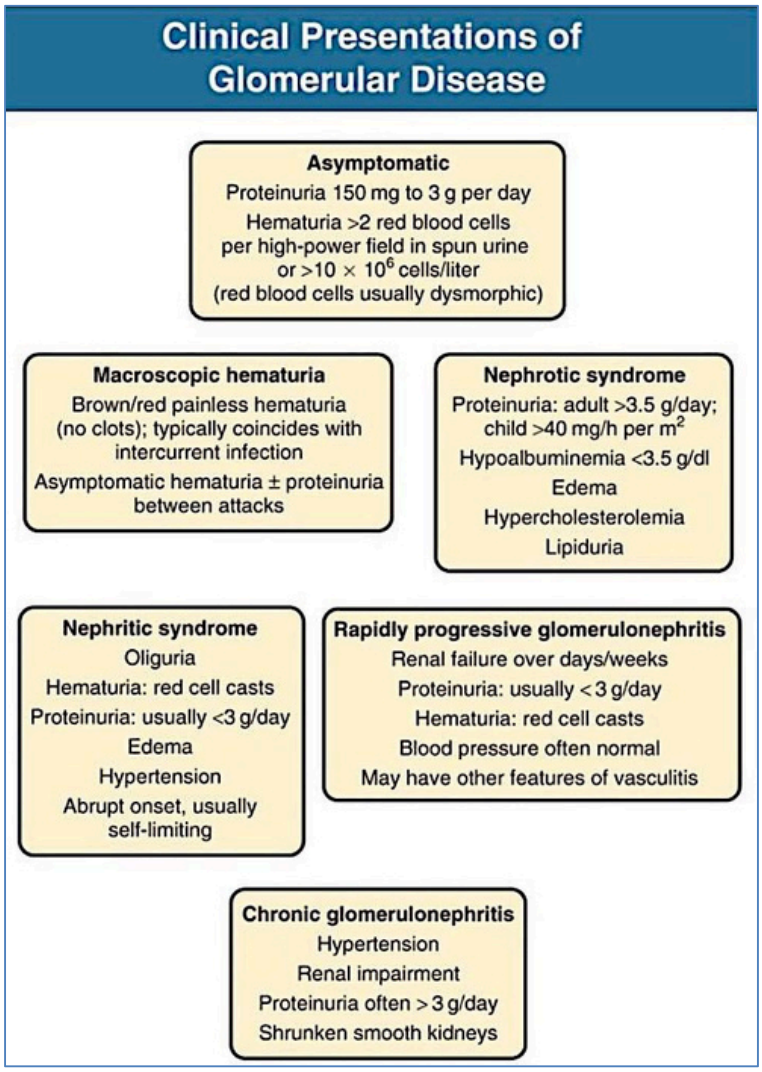
A: Normal Glomerulus; B: Focal Segmental Glomerulonephritis



Source: Wikimedia Commons

- **Progression of Glomerular Diseases:**
  - o Once damage causes a GFR reduction to 30-50%, a Vicious Cycle Starts.
    - § Focal Segmental Glomerulosclerosis & Tubulointerstitial Inflammation/Fibrosis → Reduction in Functional Renal Mass → Cycle (See Below)





NEPHROTIC SYNDROMES

NEPHROTIC SYNDROMES:

N ephrotic Syndrom es = Collection of diseases caused by inflam m ation, dam age to glom eruli of kidney; glom eruli become more permeable, allow proteins from blood into urine → proteinuria

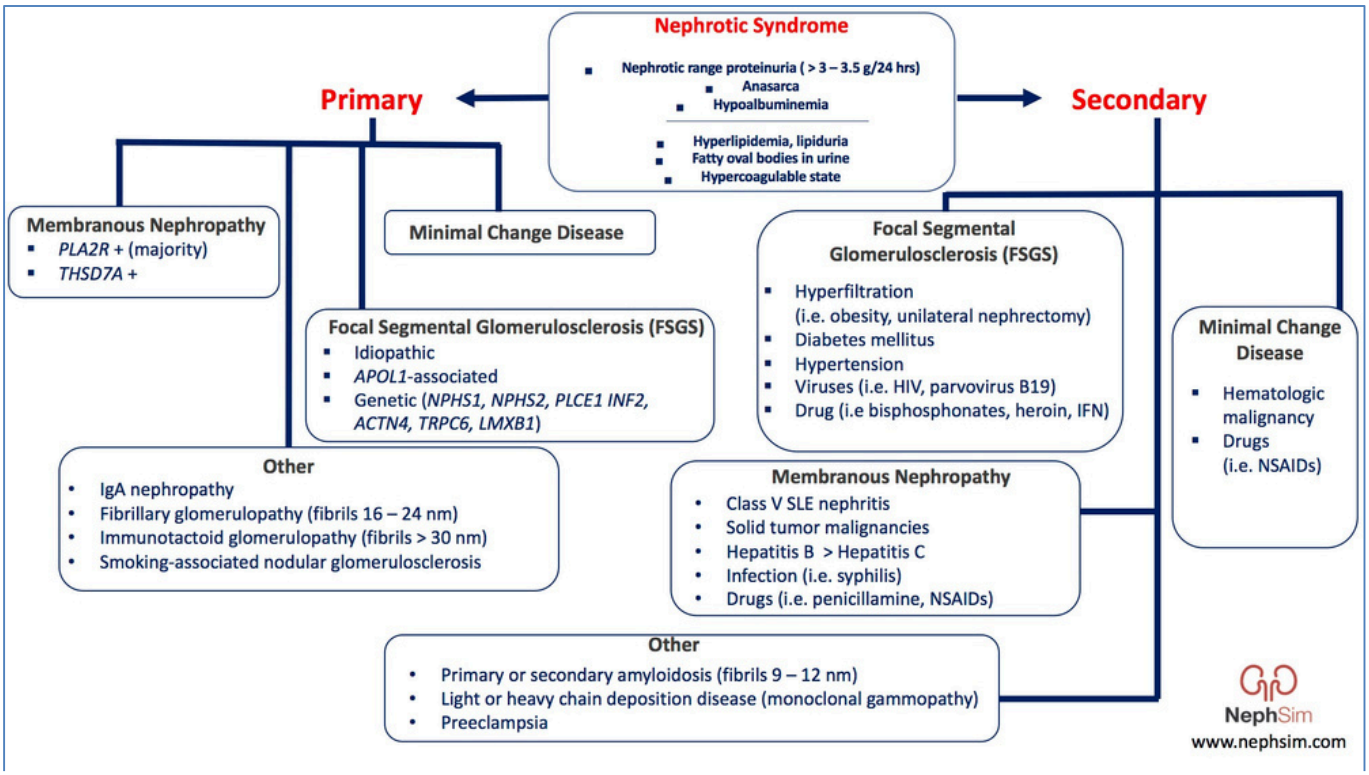
General Clinical Features:

- **Reduced GFR**
- **+++Polyuria**
- **++++ Proteinuria (>3000mg/day :. Nephrotic)**
  - o → **Granular (Protein) Casts**.
  - o → **Oedema (Especially Periorbital)**
  - o → **Hypercoagulability –** (Loss of Antithrombin-III in Urine)
  - o → **Immunocompromise –** (Loss of Ig in Urine)
  - o → **Hyperlipidaemia –** (Attempted Hepatic Compensation for ↓Plasma Osmolality)
- **↑Serum Creatinine –** Mildly Elevated
- **(Note: Dehydrated due to Polyuria; But Oedematous due to Proteinuria)**

General Treatment:

- **Diuretics** (Eg: Frusemide; for Oedema)
- **ACE-Inhibitors** (For BP control)
- **Reduce cholesterol/fat intake**
- **Heparin** (for hypercoagulability)
- **Immunosuppressants** (Eg: Cyclophosphamide, Prednisone)

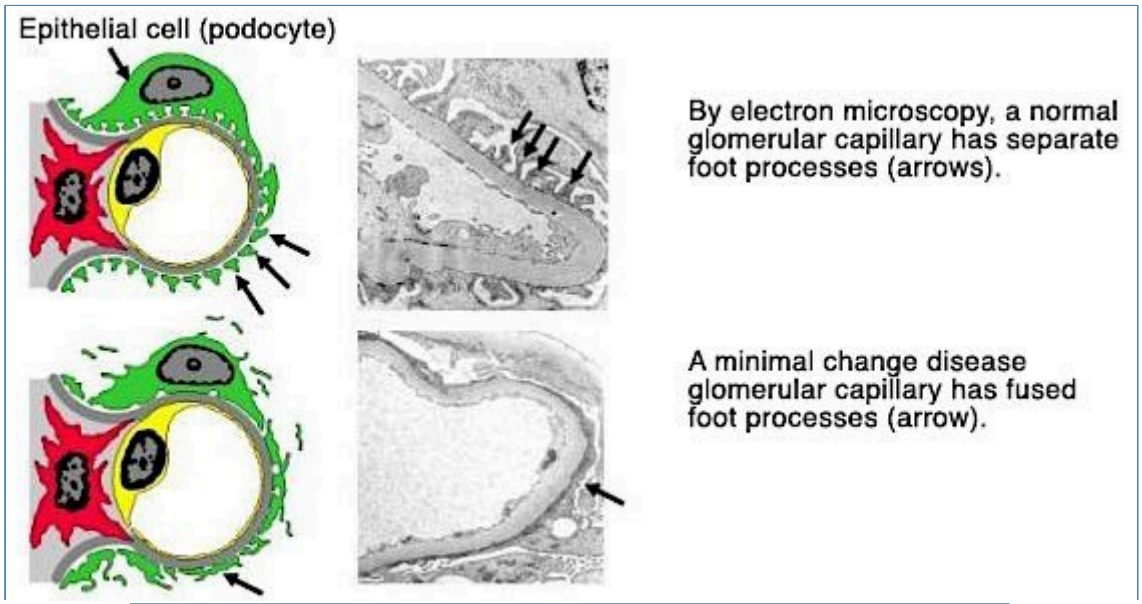
Numerous Types & Causes of Nephrotic Syndrome:

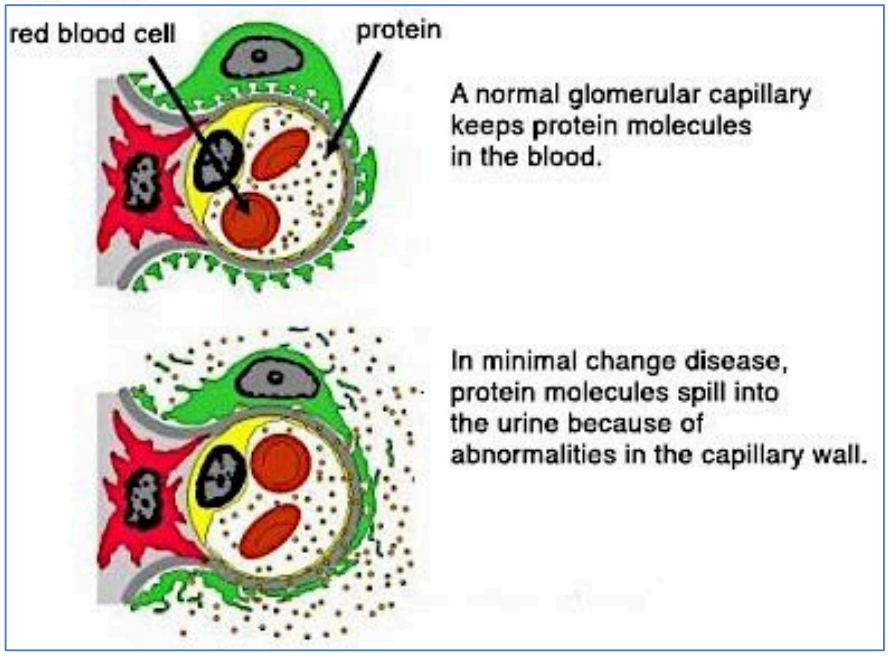


https://nephsim.com/case-50-diagnosis-conclusions/

MCD – MINIMAL CHANGE DISEASE (“*Foot Process Disease*”/“*Nil Disease*”):

- **MCD = *THE MOST COMMON Childhood cause of Nephrotic Syndrome* (1-8yrs)**
- **Aetiology:**
  - Often cause unknown
  - Can be Post-Infective (URTI)/Post-immunisation
  - Can be from NSAIDS
- **Pathophysiology:**
  - podocytes in glomeruli damaged by T cells cytokines
  - → podocytes damaged, flattened (AKA effacement) → lose function as barrier
  - → albumin permeates, bigger proteins cannot get through (selective proteinuria)
- **Clinical Features:**
  - **Eg:** 2yo Boy with sudden onset Polyuria, Oedema & Proteinuria following URTI.
  - **Children** – 1-8yrs
  - **Prognosis – Relatively Benign;** Spontaneous Remission in <70% of Pts; Some progress to FSGS.
- **Diagnosis:**
  - Proteinuria >3.5g/day
  - Renal biopsy
- **Treatment:**
  - Most cases respond well to **Prednisone**



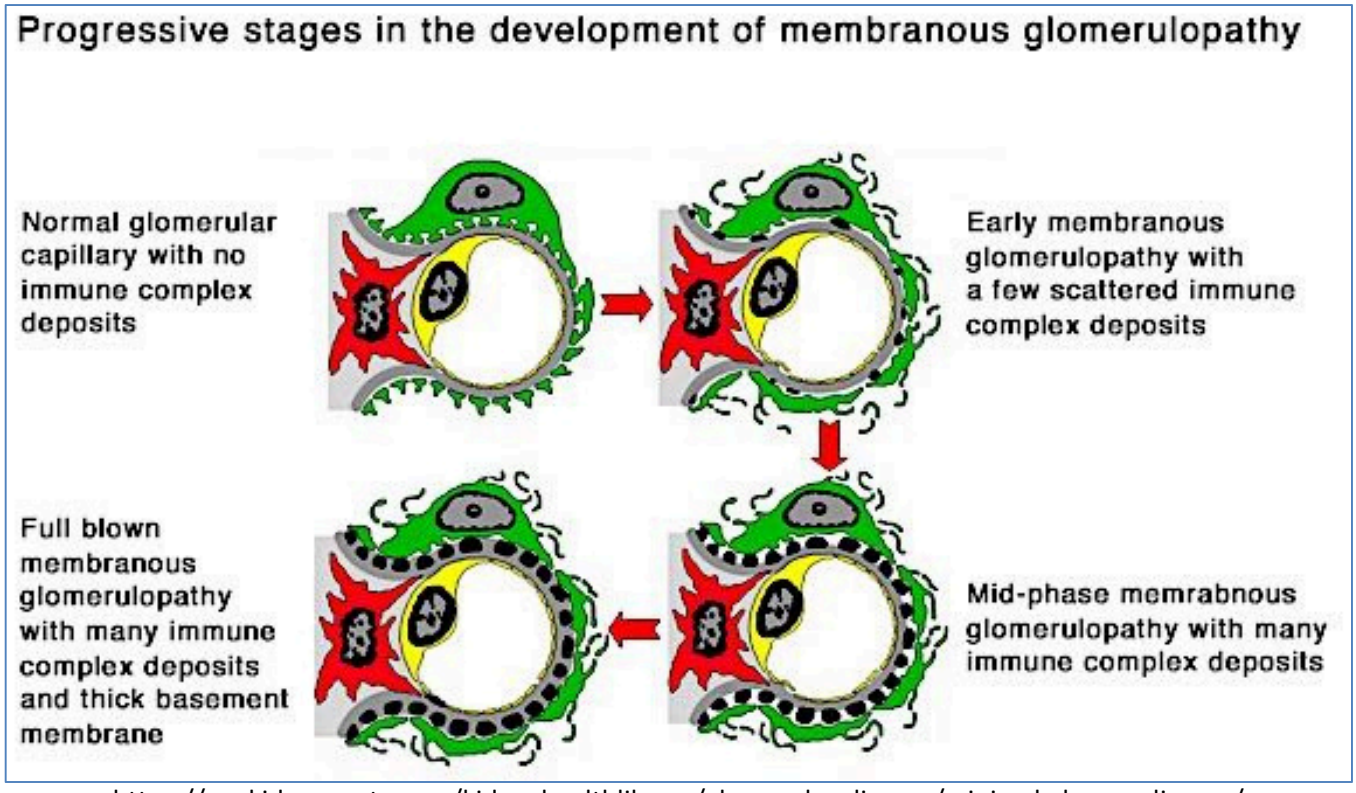


https://unckidneycenter.org/kidneyhealthlibrary/glomerular-disease/minimal-change-disease/

MGN – MEMBRANOUS GLOMERULONEPHRITIS:

- **MGN = *>50% of Adult Nephrotic Syndrome***
- **Aetiology:**
  - o Mostly idiopathic
  - o Autoimmune – Ag:Ab Complex Deposition
  - o Secondary – Hepatitis B/C, Syphilis, NSAIDS, SLE, Malignancy.
- **Pathophysiology:**
  - o Immune complex deposits in Glomerular Basement Membrane
  - o → Inflammation of glomerular basement membrane
  - o → damage to podocytes, mesangial cells
  - o → increased permeability, proteinuria → nephrotic syndrome
- **Clinical Features:**
  - o **Eg:** 35y female, Tired for years*, Worsened since two months. She has noted* swelling of her legs *and* **puffiness around eyelids (Periorbital Oedema – A classic sign of nephrotic syndrome)**.
  - o **Adults -** 40-60yrs
  - o **Nephrotic Syndrome** – Polyuria, +++ Proteinuria, Oedema.
- **Diagnosis:**
  - o Proteinuria
  - o Renal biopsy (Thickened glomerular basement membrane)
- **Treatment:**
  - o Diuretics (Frusemide)
  - o o ACE Inhibitors
  - o +/- Prednisone/Ciclosporin/Cyclophosphamide if at significant risk of ESRF. o Consider Rituximab
  - o Heparin
  - o Treat underlying cause

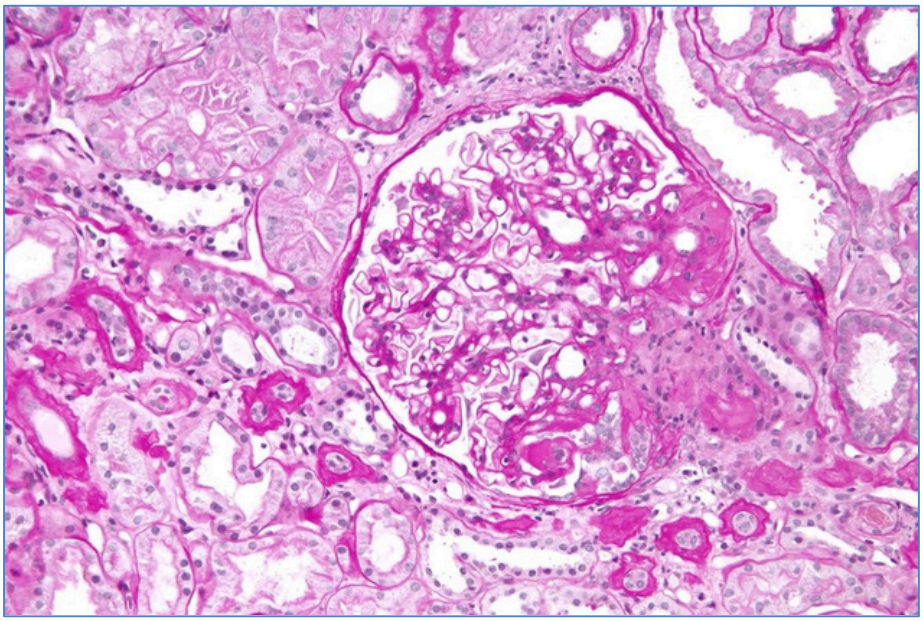
- **Prognosis:**
  - o **Spontaneous complete remission:** 5–30% at five years
  - o **Spontaneous partial remission:** 25–40% at five years
  - o **Occasionally progresses to ESRD**



https://unckidneycenter.org/kidneyhealthlibrary/glomerular-disease/minimal-change-disease/

FSGS – FOCAL SEGMENTAL GLOMERULOSCLEROSIS:

- **FSGS = *<35% of Adult Nephrotic Syndrome***.
- ***Very Similar to Minimal Change Disease, but in Adults***.
- **Aetiology:**
  - Often cause isn't identified.
  - **Can be secondary to:**
    - Sickle cell disease
    - HIV
    - Heroin Abuse
  - **Risk Factors:**
    - Black/African/Latin American Descent
    - Morbid Obesity
- **Pathogenesis:**
  - Affects parts (segmental) of some (focal) glomeruli of nephron; damage, scarring → proteinuria
  - Foot processes of podocytes damaged → plasma proteins, lipids permeate glomerular filter
  - Proteins, lipids trapped → build up inside glomeruli → hyalinosis (hyaline/ glassy view on histology) → scar tissue (glomerulosclerosis)
- **Clinical Features:**
  - **Eg:** 49y, Nephrotic Syndrome non-responsive.
  - **Nephrotic Syndrome**:
    - +++Proteinuria,
    - Hypoalbuminaemia,
    - Oedema,
    - Hyperlipidaemia/Lipiduria
    - Hypercoagulability
    - Polyuria
- **Diagnosis:**
  - Proteinuria
  - Kidney Biopsy – Segmental Sclerosis, Hyalinosis of glomeruli.
- **Treatment:**
  - ACE-Inhibitors (Blood pressure reduction)
  - Diuretics (For Oedema)
  - Prednisone if reasonable likelihood of reversibility.
- **Prognosis – Poor:** 30% Remission, 50% CKD & 20% RPGN.



(Focal and segm ental glom erulosclerosis: Scarred, obliterated capillaries and accum ulations of m aterial in part of the affected glomerulus)

NEPHROSCLEROSIS:

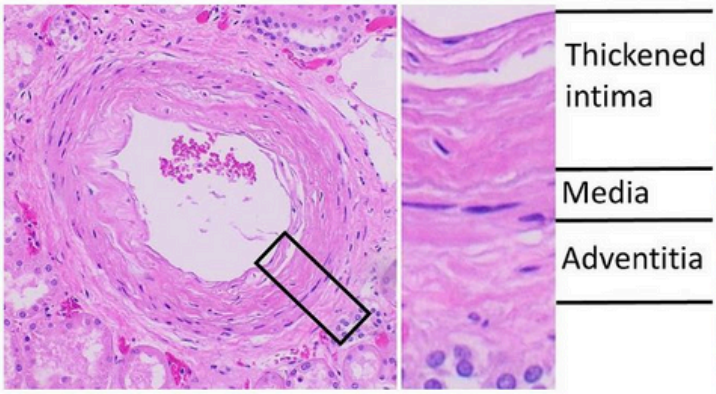
- = Scarring & stiffening of the Nephron (Primarily the Vasculature)
- **Aetiology:**
- o Diabetes &/Or Hypertension

Pathogenesis:

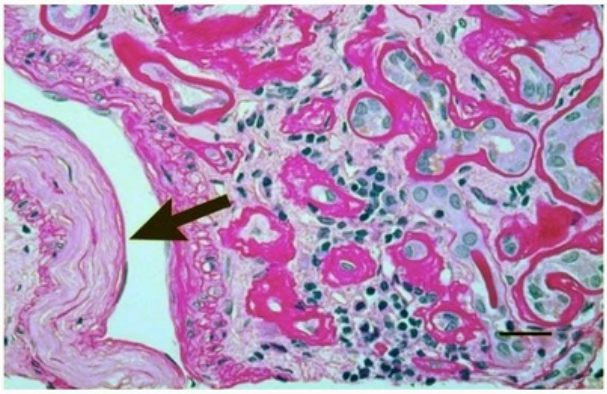
- o **(Diabetes** → ↑[Blood Glucose] → Blood proteins become *sticky* → deposit in small blood vessels → Vessel Inflammation, Damage & Scarring → Nephrosclerosis**)**
- o **(Hypertension** → Damage to Glomerular Capillaries → Sclerosis & Thickening of Capillary Wall → Nephrosclerosis)
- o **LEADING TO →Deposition of Protein in Vessel Wall → Thickening of Vessel Wall → Ischaemia → Necrosis**.

- **Clinical Features:**
  - o May be insidious as sufficient kidney reserve capacity may maintain adequate kidney function for m any years.
  - o Mild Chronic Kidney Failure Symptoms (Variably ↓GFR)
    - § Loss of appetite/nausea/vomiting
    - § Itching
    - § Confusion/sleepiness
    - § Weight loss
  - o Proteinuria (often in the **Nephrotic** range)
- **Histology:**
  - o Large renal arteries exhibit intimal thickening, medial hypertrophy & duplication of elastic layer.
  - o Small arterioles exhibit hyaline arteriolosclerosis (deposition of Hyaline) → Causes glomerular collapse & solidification.
- **Diagnosis:**
  - o Presence of chronic hypertension
  - o 24hr urine collection → Proteinuria/Albuminuria
  - o Definitive diagnosis via biopsy/histology.
- **Management:**
  - o o Tighter control of hypertension/diabetes.
  - o Weight reduction, exercise, reducing salt intake.

ACE Inhibitors/Angiotensin Receptor Blockers

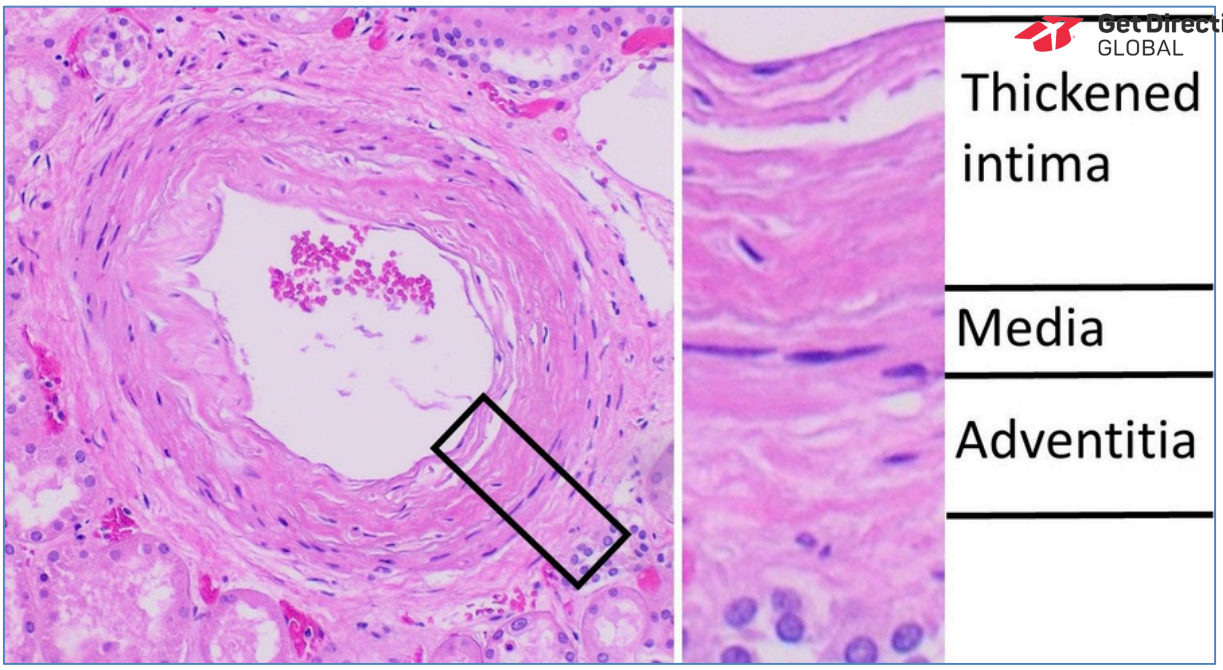


Histopathology of arcuate artery nephrosclerosis, seen as a thickened intima with an onion skin-like architecture. It is presumably a manifestation of hypertensive kidney disease.



Light micrograph showing signs of hypertensive nephropathy: interstitial fibrosis, tubular atrophy with thickened tubular basement membranes, and fibrous intimal thickening of a small artery (arrow).

Mikael Häggström, M.D. CC0, via Wikimedia Commons;
https://commons.wikimedia.org/wiki/File:Histopathology_of_arcuate_artery_nephrosclerosis,_annotated.jpg



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https://commons.wikimedia.org/wiki/File:Histopathology_of_arcuate_artery_nephrosclerosis,_annotated.jpg

DIABETIC NEPHROPATHY:

- **Aetiology:**
- o Type 1 or Type 2 Diabetes

Pathogenesis:

o **Excess Glucose in Blood → Glycosuria →**

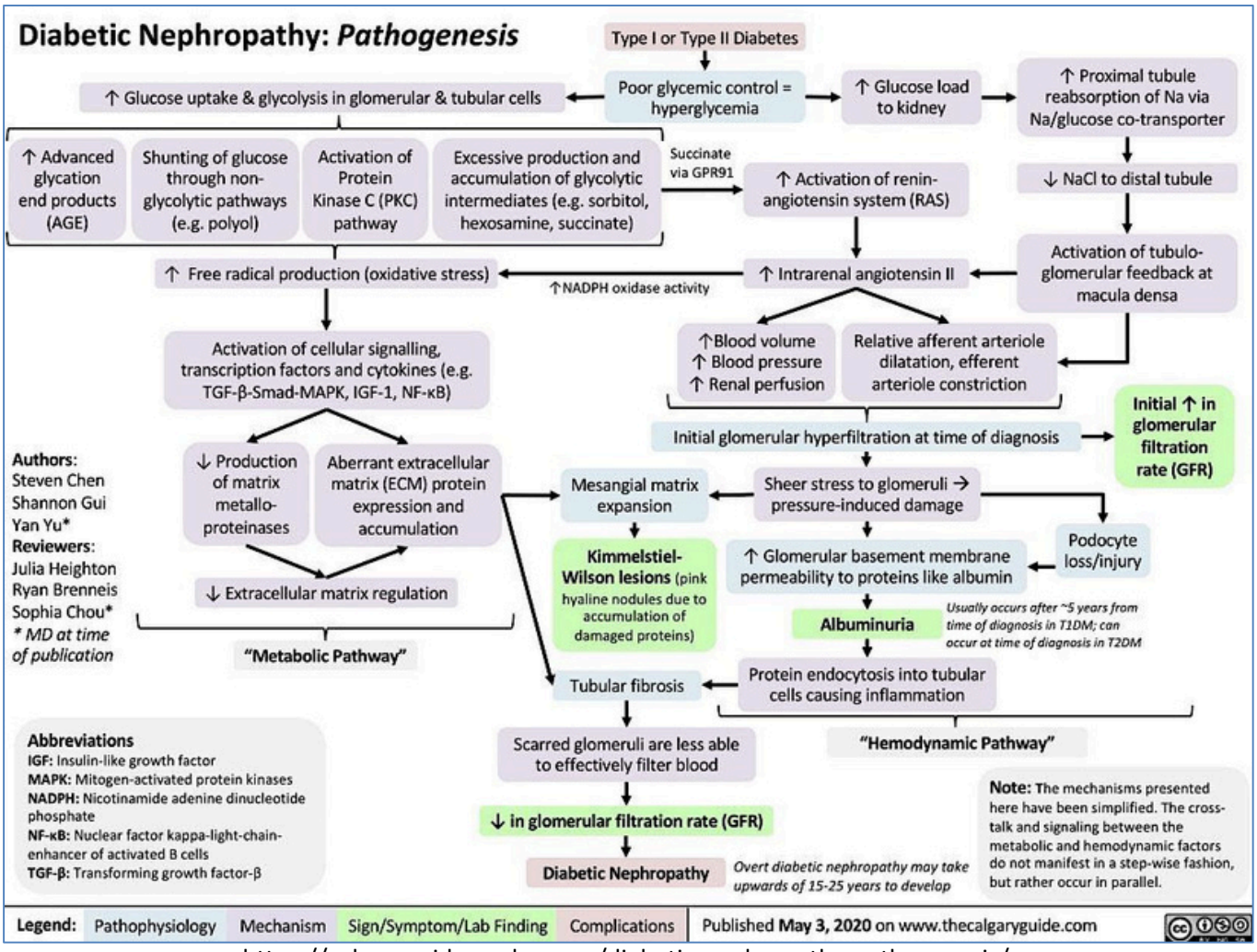
§ Glycosylation of proteins in basement membrane → **Hyaline Arteriosclerosis**
§ → Glomerular Hypertension → Increased GFR (initially)
§ → Thickening of Basement Membrane & Podocyte Injury → Damaged Glomeruli
§ → Tubular & Interstitial Fibrosis & Atrophy
§ → Decreased GFR (later stage)

- **Clinical Features:**
  - o Mostly asymptomatic
  - o Albuminuria
  - o Proteinuria
  - o (No haematuria)
  - o 50% of diabetics will have Nephrotic Syndrome
- **Diagnosis:**
- o Albuminuria

Treatment:

o Tight Control of BSL
o Initial Rx with ***Ace Inhibitors*** → reduce constriction of efferent arteriole → lower pressure in glomerulus

- **Prognosis:**
  - o 30% of Diabetics will → ESRF.



https://calgaryguide.ucalgary.ca/diabetic-nephropathy-pathogenesis/

SLE – LUPUS NEPHRITIS:

- = Inflammation of the kidney caused by SLE.
- **Aetiology:**
- o Complication of SLE (Autoimmune)

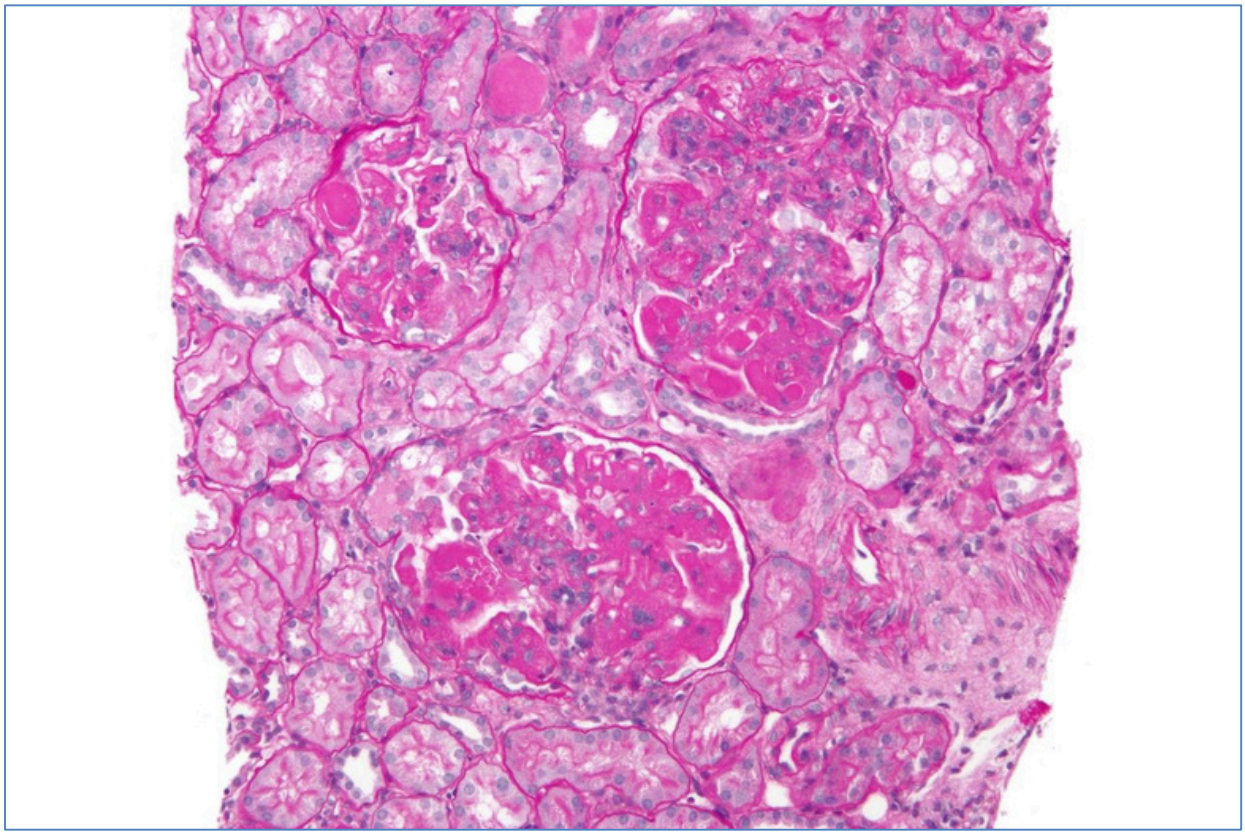
Pathogenesis:

o Immune Complex Deposition in Glomerulus → Inflammation → Glom.BM Damage – (incomplete) → Nephrotic Syndrome

- **Clinical Features:**
  - o Up to 60% of Lupus patients will develop Lupus Nephritis.
  - o Fluid Retention/ Oedema
  - o Swelling of legs, ankles and feet (Sometimes face and hands too)
  - o Weight gain
  - o o Hypertension
  - o Dark, frothy urine o **Nephrotic Syndrome** – +++ Selective Proteinuria, Oedema, Polyuria.

- **Diagnosis:**
  - o ANA Titre, ACCP Lupus Test
  - o Urinalysis (Protein/Albumin/Microscopy)
  - o Renal ultrasound
  - o Kidney biopsy
- **Treatment:**
  - o o ***Corticosteroids***
  - o **Immunosuppressives:** ***Methotrexate/Sulfasalazine/Cyclophosphamide***) o ***NSAIDS***
  - **Sometimes Dialysis → Transplant**

A high-magnification micrograph of diffuse proliferative lupus nephritis, class IV. Shows increased mesangial matrix and mesangial hypercellularity:



Nephron, CC BY-SA 3.0 <https://creativecommons.org/licenses/by-sa/3.0>, via Wikimedia Commons

NEPHRITIC SYNDROMES

NEPHRITIC SYNDROMES

Nephritic Syndromes = Collection of Diseases caused by inflammation, damage to glomeruli of kidney; become more permeable, allow red blood cells (RBCs) into urine → hematuria

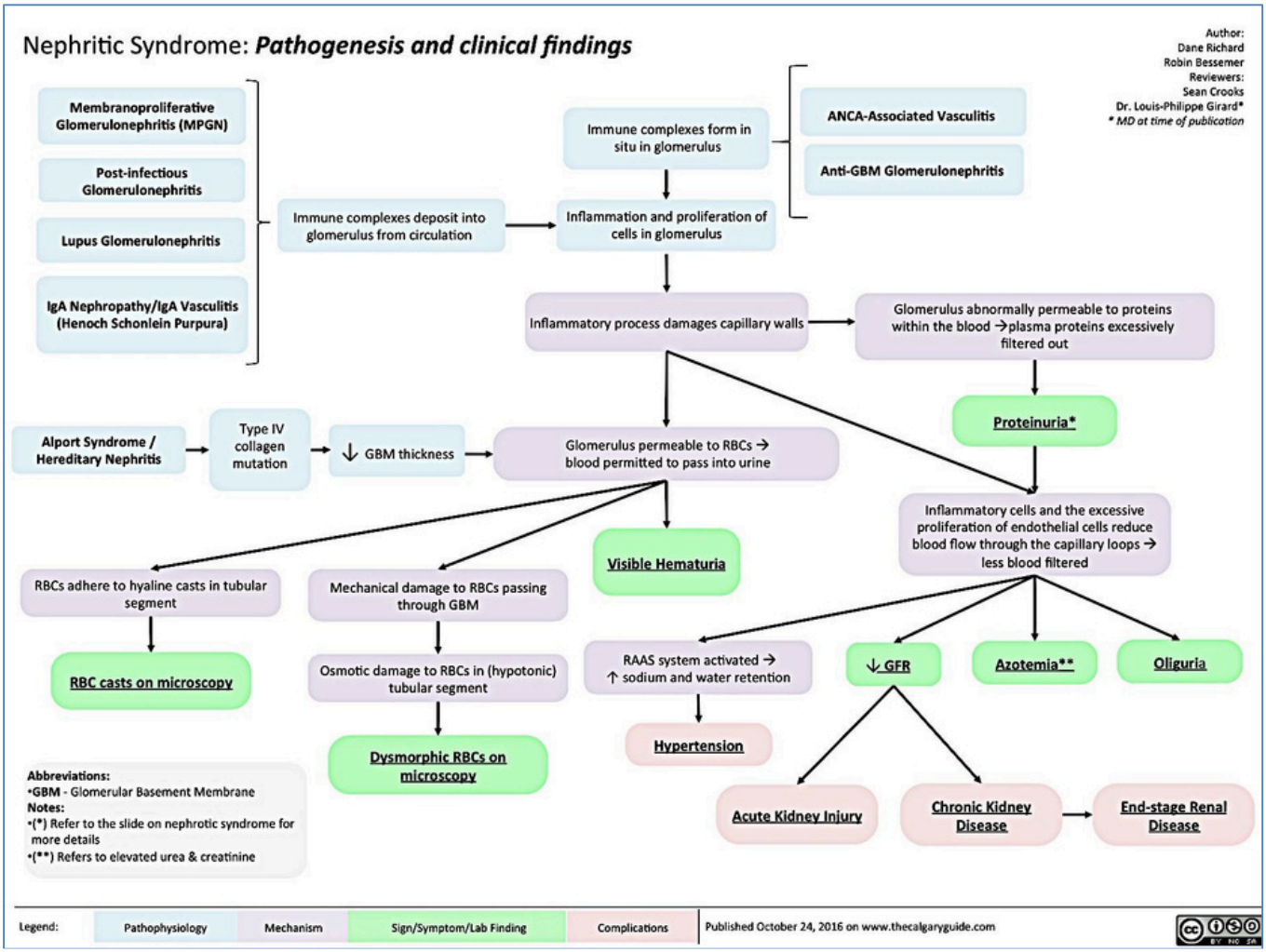
General Features of Nephritic Syndrome:

- **Acute Kidney Failure → ↓GFR:**
- **Oliguria**
  - o **→Renal Hypertension** (Hypoperfusion of JG Cells due to ↓GFR)
  - o **→Fluid Overload Oedema –** (↓Plasma Osmolality & Na + H2O Retention)
- **Microalbuminuria**
- **++++ Haematuria**
  - o **→*RBC (Cellular)* Casts in Urine**.
  - o **→ Anaemia**
- **↑ Creatinine & Uraemia**
- **(Note: Fluid Overloaded due to Oliguria; And Oedematous due to Fluid Overload)**

General Treatment:

- **Diuretics** (Eg: Frusemide; for Oedema)
- **ACE-Inhibitors** (For BP control)
- **Reduce dietary Na & K intake**

Numerous Types & Causes of Nephritic Syndrome:



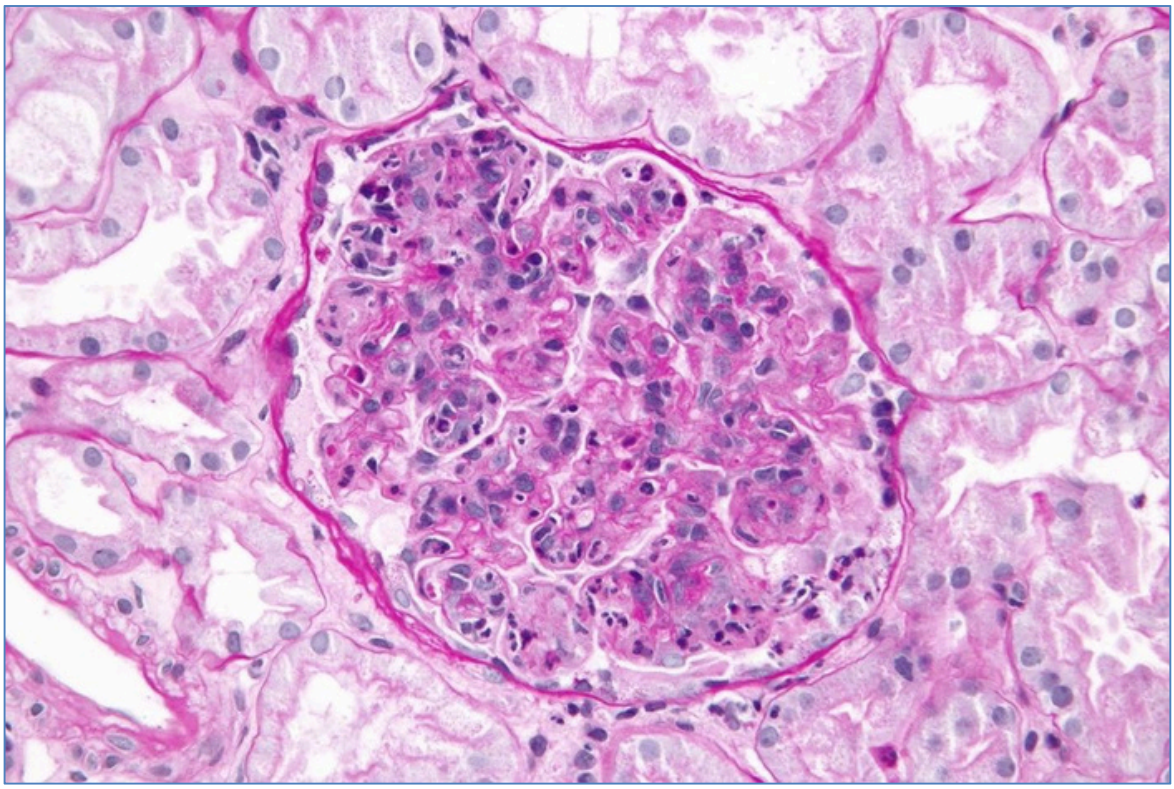
https://calgaryguide.ucalgary.ca/nephritic-syndrome-pathogenesis-and-clinical-findings/

PSGN – POST-STREP GLOMERULONEPHRITIS:

- **PSGN = *THE Childhood cause of Nephritic Syndrome (3-15yrs)***
- **Eg:** 8 year old girl with fever, oliguria, smoke coloured urine & hypertension following upper respiratory
- tract infection.
- **Aetiology:**
    - o Post-Infective *(GAß-Streptococcal Pharyngitis)* Ag:Ab Complex Deposition

  Pathogenesis:
    - o Immune complexes deposit in the Glomeruli → Proliferation of & damage to glomerular cells
    - o → Infiltration of leukocytes (mainly neutrophils) → Inflammatory damage.
    - o (Specific antigens implicated include Strep-Exotoxin-B & GAPHD; both have affinity for glomerular proteins)
- **Clinical Features:**
    - o **Timing:** Usually either 6wks after Impetigo, or 1-2wks after Strep Throat infection
    - o **Nephritic Syndrome –** Oliguria, Painless Haematuria, Non-Selective Proteinuria, Oedema, Hypertension
    - o **Prognosis–** Good Prognosis in Children (But progressive in Adults)
- **Diagnosis:**
    - o Protein/blood in urine
    - o Antibodies against GroupAStrep (Eg: Anti-DNaseB Antibodies; Anti-Streptolysin)
    - o Renal Biopsy
- **Treatment:**
    - o Usually supportive

PSGN: Glomerular hypercellularity caused by intracapillary leukocytes and proliferation of intrinsic glomerular cells. Note the red blood cell casts in the tubules.



Nephron, CC BY-SA 3.0 <https://creativecommons.org/licenses/by-sa/3.0>, via Wikimedia Commons

IGA NEPHROPATHY ("BERGER'S DISEASE"):

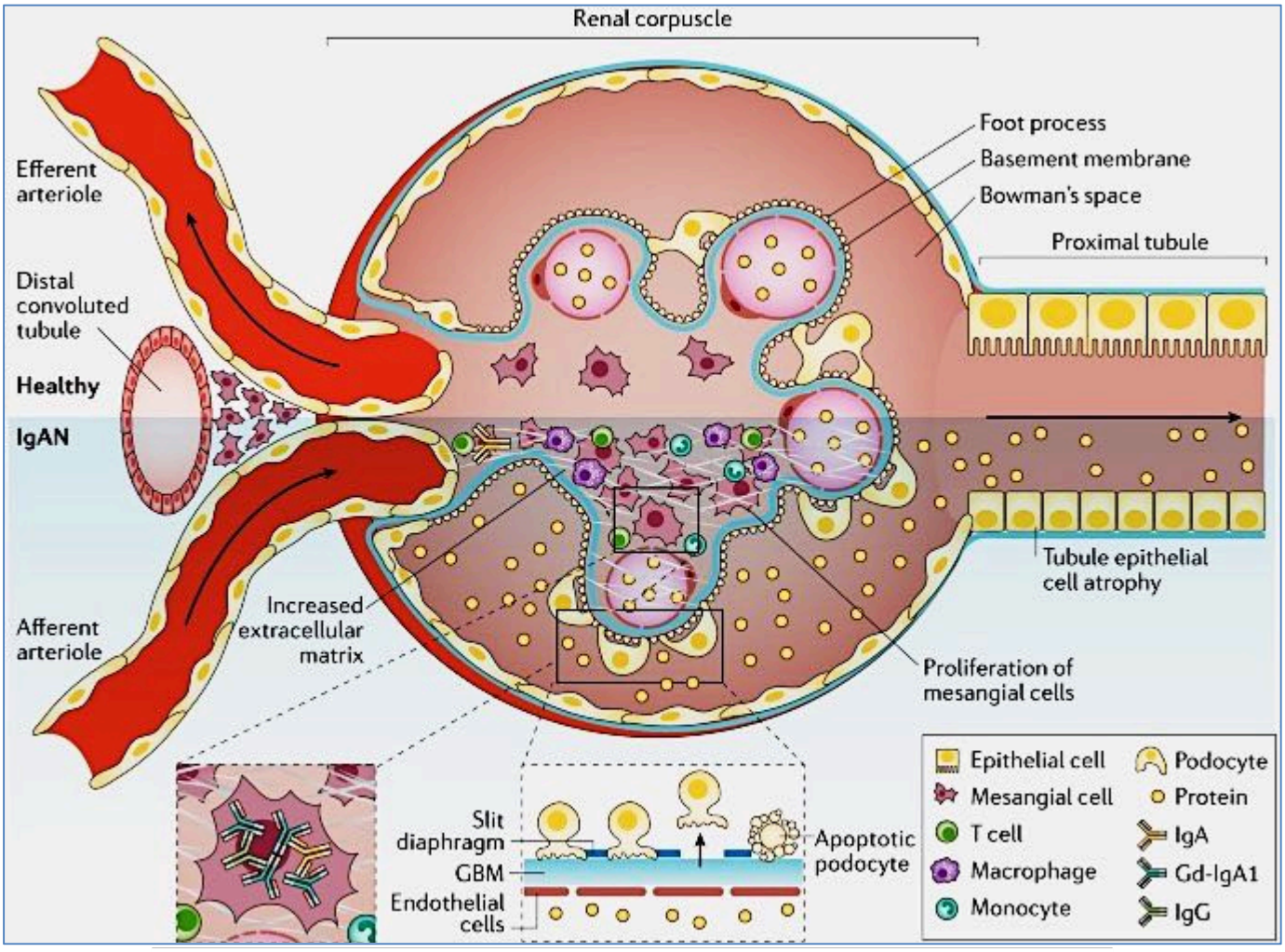
- **IgA-Nephropathy = *THE Adult (15-30yrs) Cause of Nephritic Syndrome***
- **Eg:** 18y male ***Recurrent, Episodic Painless +++Hematuria***, 3-6 days, usually following URTI.
- **Aetiology:**
- o Autoimmune - Ag:IgA Complex Deposition in Glomerulus

Pathogenesis:

- o Abnormal IgA Forms → deposits in kidneys → Inflammation → Glomerular Injury → RBC's leak into urine
- **Clinical Features:**
  - o **Nephritic Syndrome –** Oliguria, Painless Haematuria, Non-Selective Proteinuria, Oedema, Hypertension
- **Diagnosis:**
  - o o **Urine RBC's & RBC Casts**
  - o **High Serum IgA** **Renal Biopsy**
- **Treatment:**
- o Corticosteroids → Reduces IgA production.

Prognosis:

- o 30% → Slowly Progressive
- o 10% → Renal Failure



Lai, K., Tang, S., Schena, F. *et al.* IgA nephropathy. *Nat Rev Dis Primers* **2,** 16001 (2016). https://doi.org/10.1038/nrdp.2016.1

HEMOLYTIC-UREMIC SYNDROME (HUS):

- **Aetiology:**
- o Often caused by Shiga-Toxigenic E-Coli (STEC)/Enterohemorrhagic E-Coli (EHEC) Dysentery.

Pathogenesis:

o E-coli attaches to intestinal wall → secretes Shiga-like toxin →

o Shiga-like Toxin enters bloodstream → attaches to immune cells → toxins from white blood cells (WBCs) bind to endothelial cells of glomerular capillaries → many tiny blood clots in kidneys

o → kidney function decreases +Haemolysis→ urea levels in blood increase

- **Clinical Features:**

o o Typically Children <5yrs; Adults >75yrs

o Bloody Diarrhoea o Weakness, fatigue, lethargy, jaundice due to red blood cell destruction o Fever, blood clots: affect brain blood supply → visual disturbances, altered mental status, seizures, stroke → death

- **Diagnosis:**

o Thrombocytopenia

o microangiopathic hemolytic anemia (MAHA)

o acute renal failure

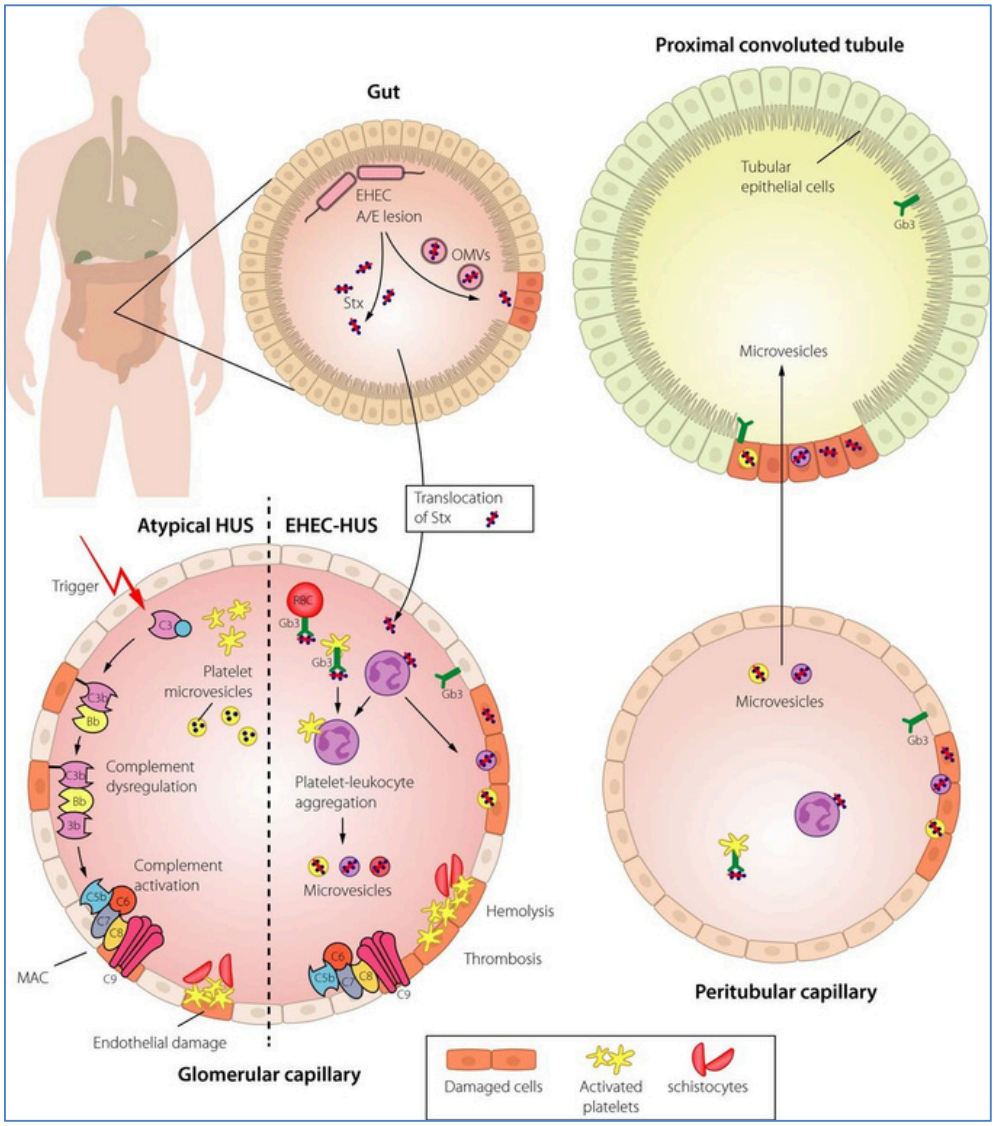
o Proteinuria/haematuria

o Schistocytes

o Positive Shiga Toxin PCR

- **Treatment:**

o Antibiotics not recommended as dead bacteria potentially release more toxins



https://onlinelibrary.wiley.com/cms/attachment/e0cfc955-713c-43aa-b6ca-2738904b741a/joim12546-fig-0002-m .jpg

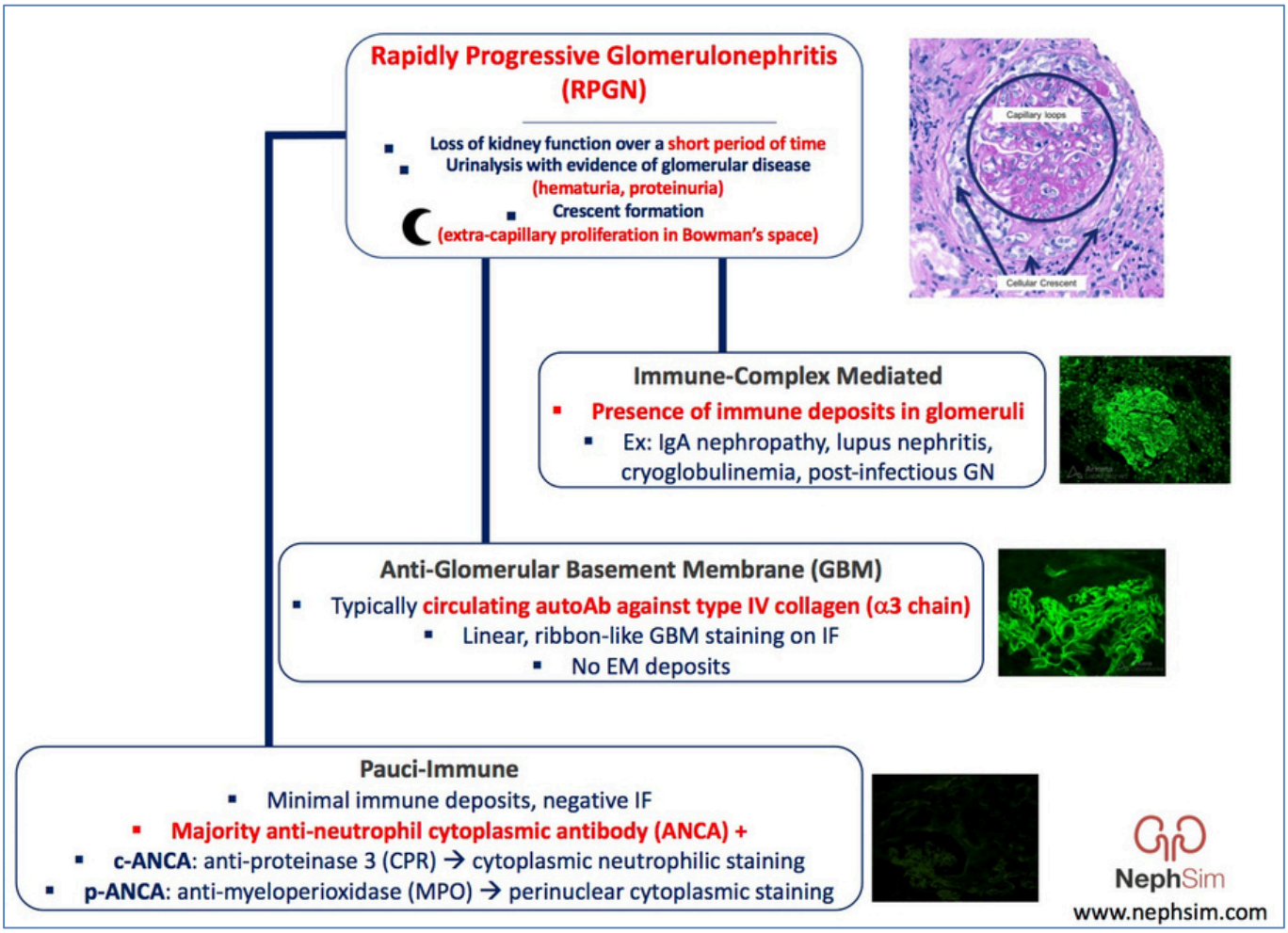
RPGN – RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS:

- **RPGN = *NOT a Separate Disease; ANY Glomerulonephritis can → RPGN***
- = Inflammation of glomeruli → Renal failure within weeks/months.
- **Aetiologies:**
  - (Progression of any Glomerulonephritis)
  - **Eg: Idiopathic**
  - **Eg: Secondary to:**
    - Immune Complex Deposition (Eg: PSGN, SLE, IgA Nephropathy)
    - Anti-GBM antibodies (Eg: Goodpasture Syndrome)
    - ANCA+ disorders (Eg: Wegener's Granulomatosis, Microscopic Polyangiitis, Churg-Strauss Syndrome)
- **Pathogenesis:**
  - Inflammation damages glomerular basement membrane
  - → inflammatory mediators, complement proteins, fibrin, monocytes macrophages pass into Bowman's space
  - → expansion of parietal layer of cells into thick, crescent-moon shape
  - → may undergo sclerosis/scarring.
- **Clinical Features:**
  - **Nephritic Syndrome –** Oliguria, Painless Haematuria, Non-Selective Proteinuria, Oedema, Hypertension
- **Diagnosis:**
  - **Kidney Biopsy** (Crescent shaped glomeruli, positive immunofluorescence)

Treatment:

- Pulse Methylprednisolone, then prednisone/cyclophosphamide/rituximab/plasmapheresis
- If irreversible renal failure → Dialysis/ Transplant.

- **Prognosis – Poor:** Quickly progresses to ESRF.



Source: Nephism

TUBULO-INTERSTITIAL DISEASES

ACUTE TUBULAR NECROSIS:

- The most common cause of Acute Kidney Injury (AKI) in hospitalised patients.
- **Aetiologies:**
  - o **Ischaemia** (Eg: Shock, heart failure, renal artery stenosis, malignant HTN, microangiopathies, HUS)
  - o **Nephrotoxins** (Eg: Aminoglycosides, cisplatin, amphotericin B, NSAIDS, Lead, Radiocontrast, etc)
- **Necrosis Caused by Ischaemic Or Toxic Injury to Tubules & Interstitium:**
  - o **- Ischaemia** (Poor Blood Flow) in the Peritubular Capillaries → Tubule Cell Death (Necrosis)
    - § Necrosis is *patchy* throughout PCT & Loop of Henle.
    - § • Most common in Proximal & Thick ascending tubules.
      'Casts' throughout the *entire* DCT & Part of Collecting Duct.
  - o **- Nephrotoxins** (Chemicals toxic to kidneys) → Tubule Cell Death (Necrosis)
    - § Necrosis is *consistent* throughout PCT & Desc.Loop of Henle.
    - § 'Casts' throughout the *entire* DCT & Part of Collecting Duct.
- **General Pathogenesis:**
  - o Death of tubular epithelial cells → Disruption of Basolateral Cell Surface → Sloughing & Obstruction of tubules → Increased Tubular Hydrostatic Pressure → Reduced GFR, Filtration & reabsorption → Reduced Urine Output → Oliguria → Azotaemia.
- **Clinical Features:**
  - o **Oliguric Phase (10-14 days)**
  - o **Diuretic Phase** – (>500mL of urine per day.)
  - o **Recovery Phase** (Return to normal urine output)
- **Diagnosis:**
  - o Kidney function tests (urea, creatinine, electrolytes)
  - o § Intrarenal AKI picture
    Urine Tests:
    - § Microscopy: Muddy-Brown 'Casts' of Cellular Debris in DCT & Collecting Ducts.
    - § Chemistry: Dilute urine
  - o Sometimes Biopsy
- **Management:**
  - o Avoid/Treat Precipitating Factor
  - o Supportive management for 1-2 weeks (Allow epithelial cells to regenerate)
  - o Dietary restrictions (limit sodium, potassium & fluid intake)
  - o Diuretics may be used
  - o Dialysis may be required.

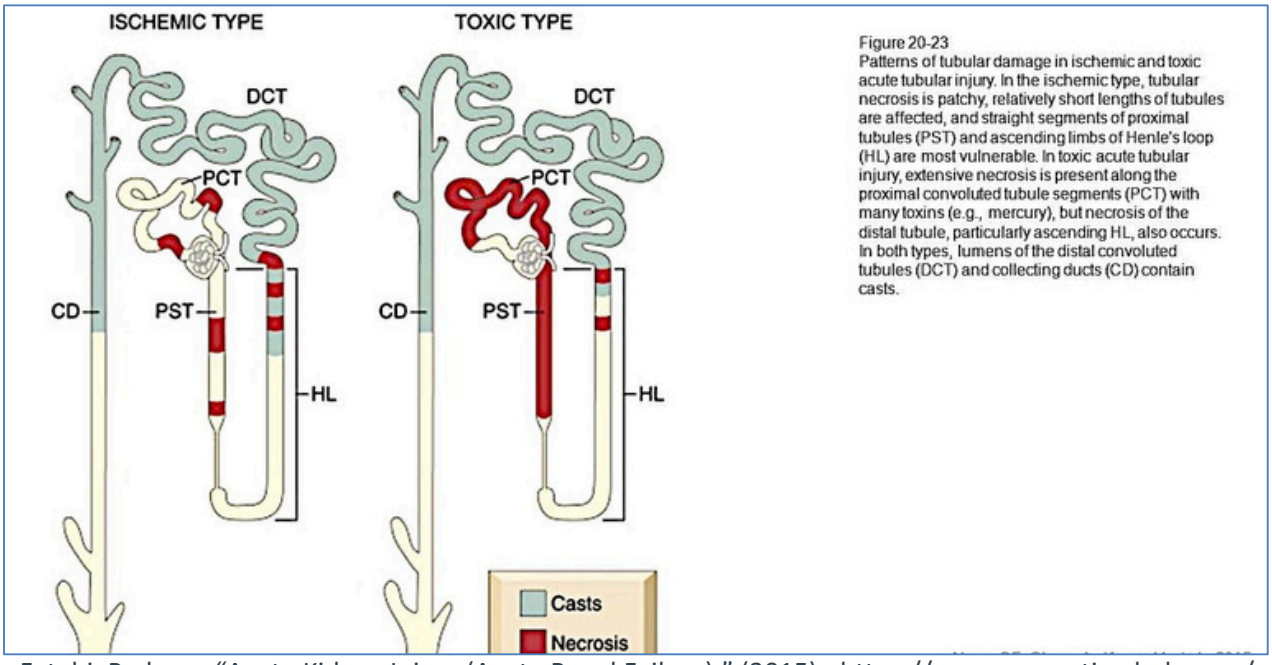
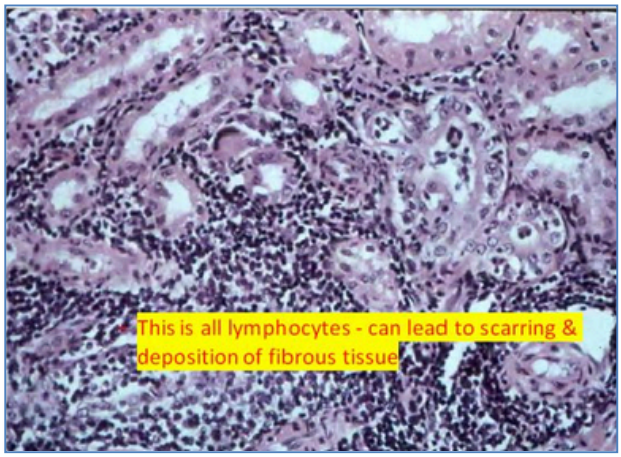


Figure 20-23
Patterns of tubular damage in ischemic and toxic acute tubular injury. In the ischemic type, tubular necrosis is patchy, relatively short lengths of tubules are affected, and straight segments of proximal tubules (PST) and ascending limbs of Henle's loop (HL) are most vulnerable. In toxic acute tubular injury, extensive necrosis is present along the proximal convoluted tubule segments (PCT) with many toxins (e.g., mercury), but necrosis of the distal tubule, particularly ascending HL, also occurs. In both types, lumens of the distal convoluted tubules (DCT) and collecting ducts (CD) contain casts.

Fatehi, Pedram. "Acute Kidney Injury (Acute Renal Failure)." (2015).; https://www.semanticscholar.org/

TUBULOINTERSTITIAL NEPHRITIS:

- = Inflammation of Tubules & Interstitium
- **Aetiologies:**
  - **Primary Causes – (Drugs/Toxins); Commonly an allergic reaction to a drug**.
    - Allopurinol
    - Some antibiotics (Penicillin, Cephalosporins, Rifampicin, Ciprofloxacin & sulfa drugs)
    - Frusemide
    - NSAIDs
    - Chemo drugs
    - PPI's (Eg: Omeprazole)
    - Heavy metals: Cadmium/Lead/Lithium
  - **Secondary Causes:**
    - Acute Tubular Necrosis
    - Multiple Myeloma
    - Polycystic Kidney Disease
    - Pyelonephritis
    - Vesicoureteral reflux
    - Sarcoidosis
    - Sickle Cell Disease
    - Sjogren Syndrome/SLE
- **Pathogenesis:**
  - Bacteria/Viruses/Drugs/Toxins/Phosphate Retention/etc. → Leukocytes Infiltration, Fibrous Tissue Deposition & Tubular Degeneration.
  - (Often Secondary to Acute Tubular Necrosis & Protein Cast deposition → Inflammation)
- **Clinical Features:**
  - Often results in kidney failure
  - **Acute Tubulointerstitial Nephritis:**
    - Electrolyte imbalances (Eg: Sodium & Potassium)
    - Polyuria due to reduced ability to concentrate urine.
    - Other symptoms directly related to cause (Eg: Fever, rash, dysuria, pain)
  - **Chronic Tubulointerstitial Nephritis:**
    - Pruritis
    - Fatigue
    - Reduced appetite
    - Nausea/Vomiting
    - Polyuria
- **Diagnosis:**
  - Bloods – BUN, electrolytes, metabolic acidosis, hypokalaemia
  - *Kidney biopsy is definitive.
- **Management:**
  - Treat underlying cause / eliminate exogenous insult.
  - Corticosteroids
  - Dialysis or transplant may be required.

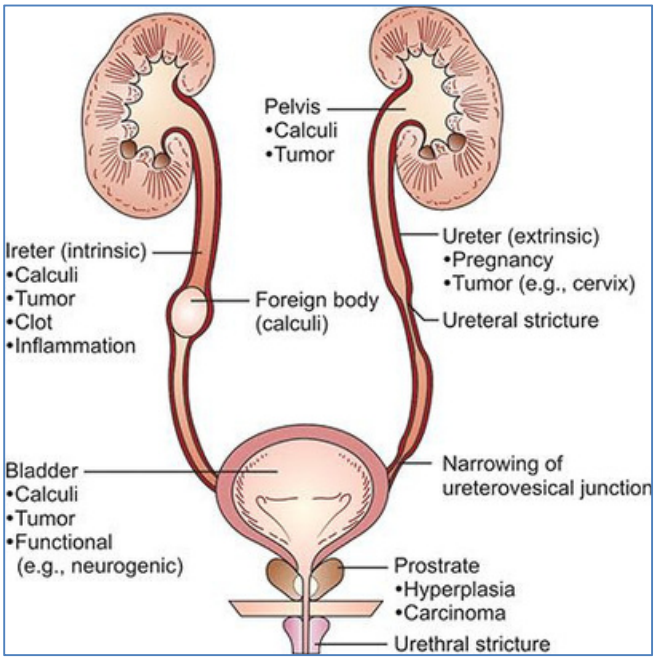


Source: Unattributable

POST-RENAL FAILURES

POST-RENAL FAILURE:

- **Aetiology:**
  - o ***Anything that Obstructs Urine Outflow from the Kidneys…Eg:***
    - § Papillary Necrosis
    - § Ureteric Obstruction
    - § Urethral Obstruction
    - § Calculi (Nephrolithiasis)
    - § Neurogenic Bladder Disease
    - § Prostatic Hypertrophy/Ca.
- **Pathophysiology:**
  - o **Urine Outflow Obstruction → Backup of Urine into the Kidney → "Hydronephrosis"**
    - § → ↑Pressure within the Kidney
      - • → Destruction of Delicate Filtration System
      - • → Compression of Tubule Vasculature → Renal Ischaemia
        - o → Progressive Atrophy of the Kidney
  - o **Kidney Stones (Calculi), Tumours, or Clots Typically tend to cause Obstruction**.
    - § – **Renal Pelvis**
    - § **– Ureter (**At the point where it enters the Bony Pelvis**)**
    - § **- Urethra**
  - o **– Prostate Hypertrophy/Cancer**
  - o **– Urethra – (Stricture/Cancer)**



Source: Unattributable

- **Clinical Features:**
  - o Kidney Stone → Severe Flank pain
  - o Nausea/Vomiting
  - o Urethral/Bladder-outlet Obstructions → Severe Suprapubic (Bladder) Pain
- **Diagnosis:**
  - o Bladder Ultrasound reveals ↑Post-Void Residual Volume.
  - o Oliguria, but NO dehydration.
- **Com plications:**
  - o Commonly UTI (due to ↓Urethral Flushing) → Fever, Pyuria & Haematuria.
  - o Complete Obstruction → Kidney Failure → ↑Creatinine, ↑Urea, & Electrolyte Imbalance.
- **Management:**
  - o Relieve Obstruction
  - o Fluid Restriction
  - o Treat any UTIs

RENAL STONES

Get Direction GLOBAL

NEPHROLITHIASIS & UROLITHIASIS:

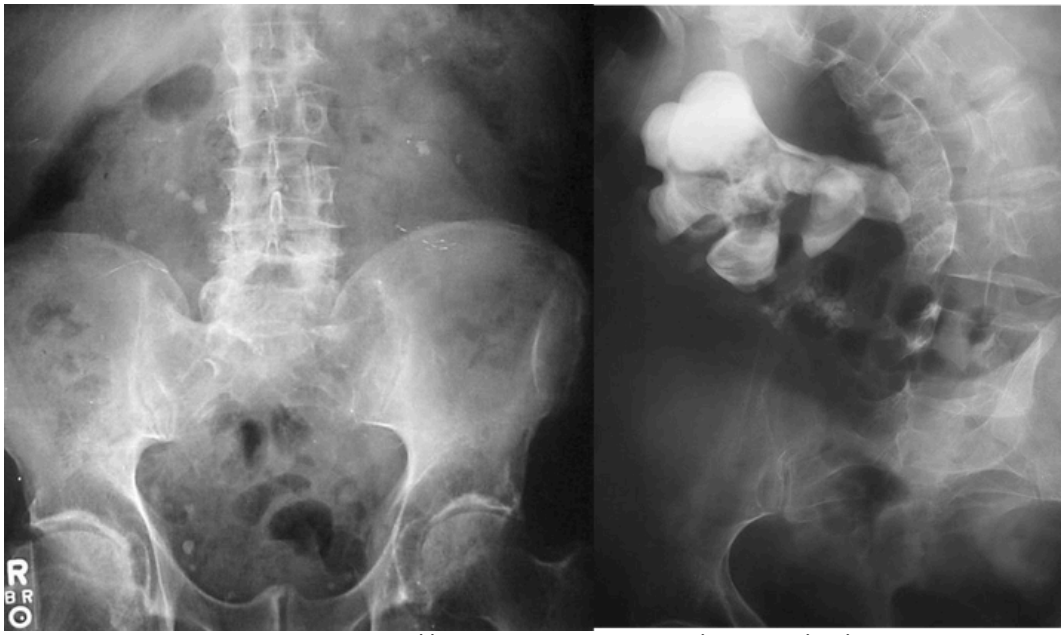
- **Basic Concept:** Stones form when **Solutes Precipitate Out** as crystals in the urine.
- **Aetiology:**
  - o **1- Hypercalcaemia (Eg: ↑Intake, or Hyper-PTH) → Calcium Stones 80%**
  - o **2- Chronic UTI → Triple Phosphate/Struvite/ "Staghorn" Stones 15%**
  - o **3- Uraemia → Urate Stones (+ Gout)**
  - o (Others: Cysteine stones, Xanthine stones)
- **Pathogenesis:**
  - o **1- Hypercalcaemia →** Calcium in Urine Precipitates out of Solution → **Calcium Stones 80%**
  - o **2- Chronic UTI →** Gram-Neg Rods (Proteus, Pseudomonas & Klebsiella – NOT E-Coli) → **Triple Phosphate/Struvite/ "Staghorn" Stones 15%**
  - o **3- Uraemia →** Urate binds sodium → Forms monosodium urate crystals → Stones.
  - o **(May → Urinary Obstruction → Hydronephrosis → Stretching of Renal Capsule → Pain)**
- **Risk Factors:**
  - o Family Hx
  - o o Vesicoureteral Reflux/Neurogenic bladder
  - o Congenital urinary tract malformations (Eg: Horseshoe kidney) o Hyperuricemia (Eg: High-purine diet; red meat, shellfish, anchovies etc) o High Cell Turnover conditions (Eg: Leukaemia, chemo drugs) o Hypercalcaemia (Eg: Hyperparathyroidism, inflammatory bowel disease, excessive calcium intake)

Recurrent UTIs

  - o High salt diet
  - o Obesity
- **Morphology:**
  - o **Calcium Stones 80%:**
    - § Small, hard Stones (1-3mm)
    - § Stones have sharp edges
    - § Radio-Opaque
  - o **Triple Phosphate/Struvite/ "Staghorn" Stones 15%:**
    - § Large Stones (Moulds to Renal Pelvis/Calyces) – Hence "Staghorn".
    - § Chronic Irritation of Epithelium surrounding Stone → Squamous Metaplasia
  - o **Urate Stones:**
    - § Pebble-like.
    - § Hard on outside, but softer on inside.
    - § Radiolucent

- **Clinical Features:**
  - Usually Unilateral
  - **Painful Hematuria** – Macro/Micro
  - ***"Writhing in pain, pacing about, and unable to lie still"***
  - **Hydronephrosis** → Stretching of Renal Capsule → Flank Pain & Tenderness.
  - **Stone in Ureteropelvic Junction →** Deep flank pain. No radiation. Distension of the Renal Capsule.
  - **Stone in Ureter** → Intense, Colicky Pain (Loin → Inguinal Region → Testes/Vulva) + N/V.
  - **Stone in Ureterovesical Junction** → Dysuria, Frequency, + Tip of penis pain
- **Com plications:**
  - Hydronephrosis
  - Post-Renal Failure
  - Infection – (UTI/Pyelonephritis/Perinephric Abscess)
- **Investigations:**
  - **Abdo USS –** (Confirm Stone) (Preferred for pregnant women)
  - **Abdo XR –** (Confirm Calcium Vs Radio-Lucent Stone)
  - **CT-KUB –** (Accurately detects size, location, density & category of stone)
  - **UECs –** (↑Calcium or ↑Urea)
  - **Urinalysis –** (Haematuria +/- crystals in urine)

Left: Radio-opaque stones in AXR; Right: Staghorn calculus



Bill Rhodes from Asheville, CC BY 2.0 <https://creativecommons.org/licenses/by/2.0>, via Wikimedia Commons
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- **Management:**
  - **Analgesics**
  - **Hydration**
  - **Most stones <5mm will pass spontaneously**.
  - **Conservative:**
    - Urine Alkalysers [Eg: Na-bicarb / K-Citrate] → Dissolve Urate Stones
    - Alpha blockers / Calcium channel blockers → Reduces ureteric spasms & pain.
  - **(ESWL)** ***Extracorporeal Shock-Wave Lithotripsy*** – (Good For Calcium Stones)
    - Non-invasive.
    - Uses acoustic pulses to break up stones into smaller fragments
  - ***Surgical*** **–** **(**For All Stones *Not* Amenable to the above**)**
    - Incl: Stents

RENAL SYSTEM CANCERS

(Adults) RENAL CELL CARCINOMA: *"Clear-Cell Carcinoma"*:

- **Aetiology:**
  - **Genetic** - VHL Gene Mutation
  - **Risk Factors** – Smoking, Obesity, Analgesic Abuse, M3:F1, >50yrs
- **Pathogenesis:**
- Carcinogenesis of Cells of the *PROXIMAL Convoluted Tubules*.

Morphology:

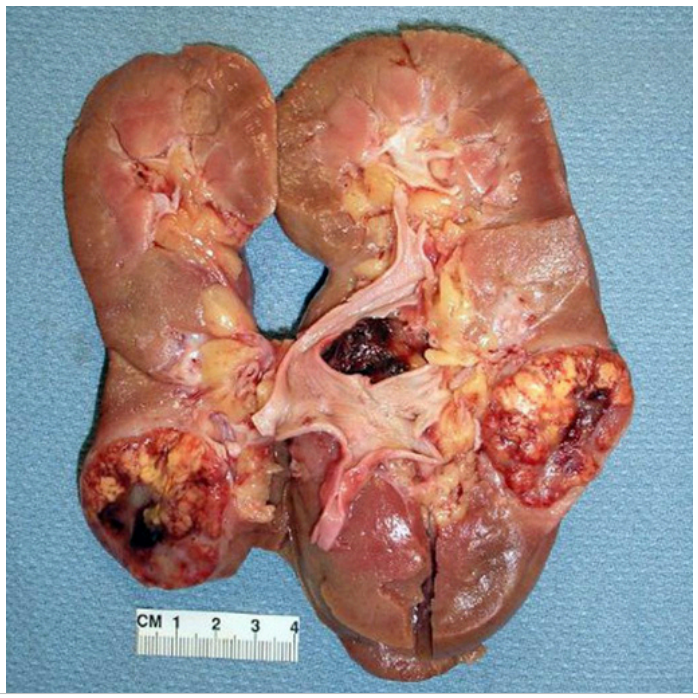
- Enlarged Kidney
- Yellowish-Orange Tumour (lots of fat)
- Looks Well Demarcated/Encapsulated
- Areas of Haemorrhage and Necrosis
- **+*Invasion into the Renal Vein**

- **Clinical Features:**
  - The Most Common Renal Malignancy.
  - Insidious onset (Often asymptomatic until late stage)
  - ***TRIAD* of Symptoms:**
    - **1- Painless Haematuria** – Most Common Symptom
    - **2- Flank pain**
    - **3- Palpable Mass in abdo/lower back**
  - **B Symptoms** – Fever, weight loss, night sweats.
  - **Weakness/Malaise**
  - **Renal vein invasion → Ipsilateral Varicocoele**.
- **Diagnosis:**
- **Abdo CT – (Diagnosis & Staging)**

Complications:

- **Metastasis** – Hematogenous Spread into Renal Vein + Local Abdominal Spread
- **Paraneoplastic Syndromes:**
  - **↑PTH → Hypercalcaemia** (Can → Calcium Stones)
  - **↑EPO → Polycythaemia**
  - **↑ACTH → Excess Cortisol → Cushing Syndrome**
- **Death** – 40% 5yr survival

- **Treatment:**
  - Nephrectomy
  - Chemotherapy/Immunomodulatory drugs (Eg: IFN, IL2, Monoclonal Ab's)



Gaillard, F. Renal cell carcinoma (gross pathology). Case study, Radiopaedia.org. https://doi.org/10.53347/rID-9888

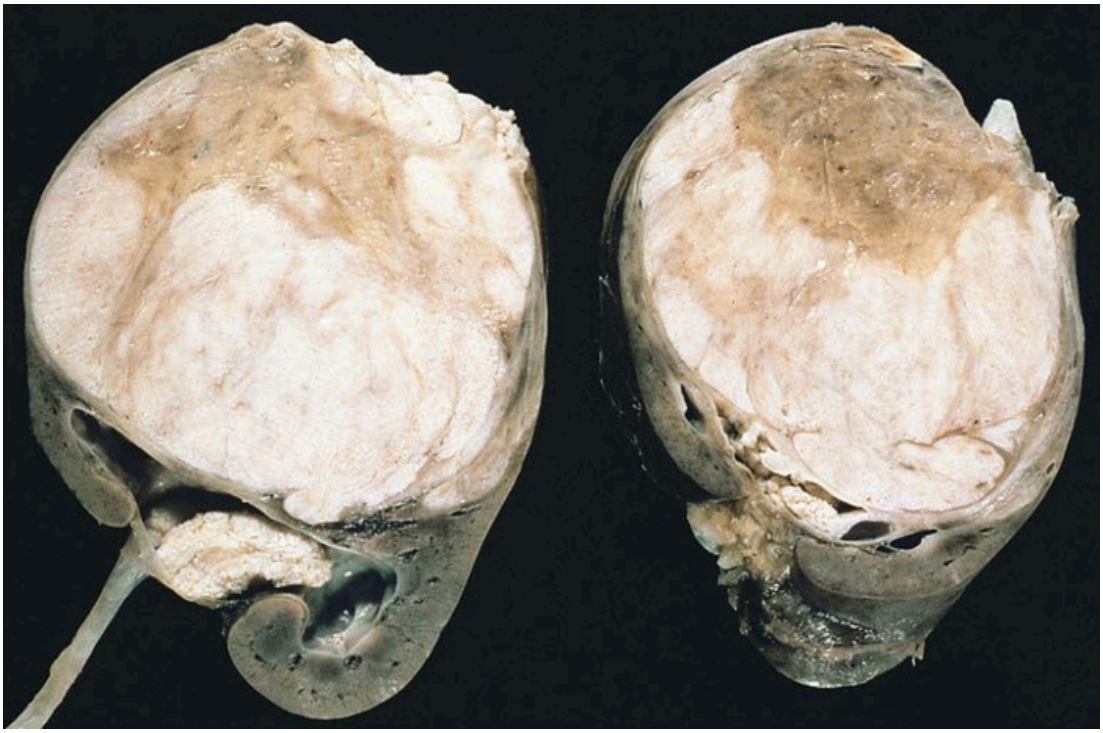
WILM'S TUMOUR / "NEPHROBLASTOMA":

- **Aetiology:**
  - **Sporadic** - Unilateral (80%)
  - **Familial** - Bilateral (20%)
    - Chromosome 11
    - Wilm's Tumour 1 (WT1) gene; Wilm's Tumour 2 (WT2) gene.
- **Pathogenesis:**
- A *Blast*oma – Ie: Carcinogenesis of embryonic Renal *Blast*-Cells.

Morphology:

- Huge, Pale, Gray-White Tumour Replacing Kidney Tissue
- Well Encapsulated
- Some focal Haemorrhage & Necrosis

- **Clinical Features:**
  - **Most common childhood kidney tumor (2-5y)**
  - **Symptoms:**
    - May have Hematuria
    - Palpable Abdo Mass
    - Abdo Pain
    - Anorexia, Nausea/Vomiting
  - **B Symptoms** – Fever, weight loss, night sweats.
- **Diagnosis:**
  - **Abdo USS – (Diagnosis & Check for renal vein infiltration)**
  - **Abdo CT – (Staging)**
  - **Chest CT – metastasis?**
  - **Guided needle biopsy**
- **Complications:**
  - **Metastasis** → Lung, Liver, Bone, Brain.
  - **Paraneoplastic Syndrome** → Renin secretion → Hypertension
- **Treatment:**
  - **Nephrectomy**
  - **Chemo/Radio-Therapy**
- **Prognosis:**
  - **80% 5yr Survival Rate**.



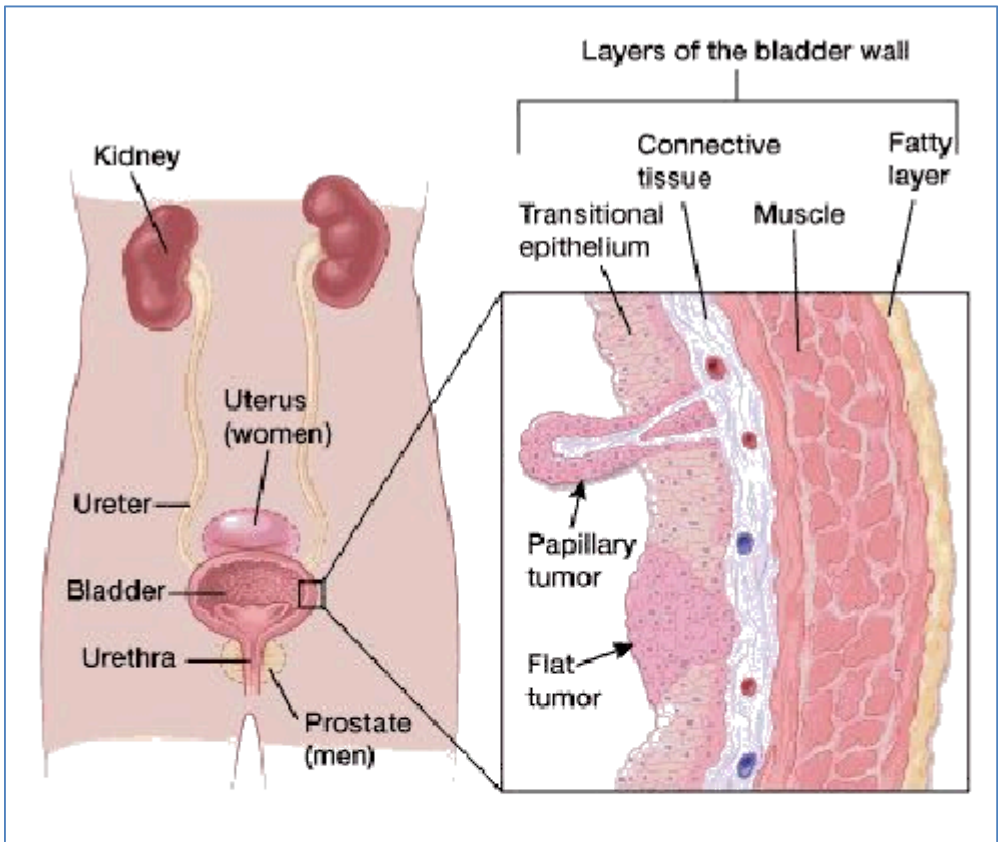
The Armed Forces Institute of Pathology, Public domain, via Wikimedia Commons; https://commons.wikimedia.org/wiki/File:Wilms_tumor.jpg

TRANSITIONAL CELL CARCINOMAS:

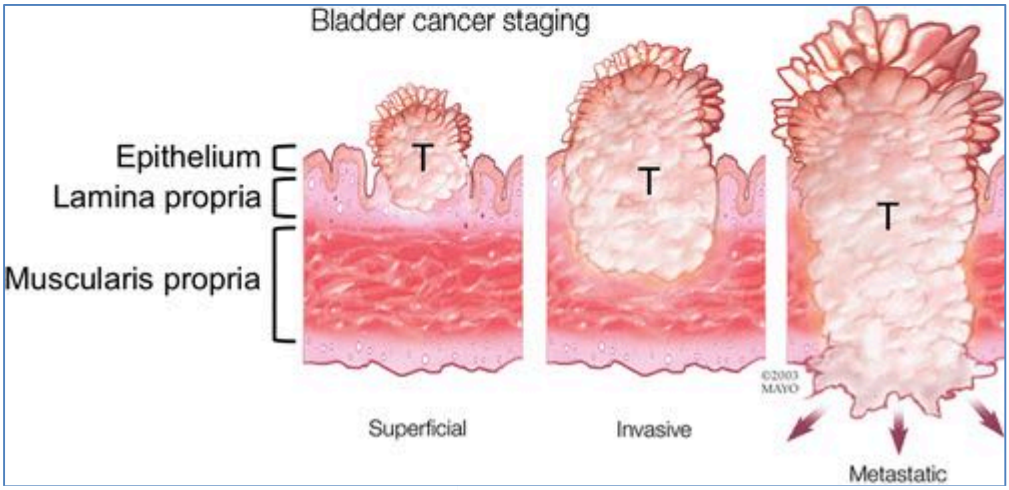
- **Aetiology:**
- o Risk Factors – Smoking, Chronic Cystitis, Male, Old Age
- **Pathogenesis:**
  - o Carcinogenesis of the Transitional-Cell Epithelium lining the Urinary Tract

  Morphology:
  - o **Commonest in bladder** → Can extend all the way from the bladder to the kidney
  - o **Papillary projections into hilum or ureters** → May cause Bladder Obstruction → Hydronephrosis
- **Clinical Features:**
  - o **Painless Haematuria**
  - o **Bladder Obstruction → Hydronephrosis**
- **Diagnosis:**
- o **Urine MCS** - Malignant cells in the urine

  Management:
  - o **Surgery + Chemo/Radiotherapy**



https://www.cancer.org/cancer/bladder-cancer/about/what-is-bladder-cancer.html

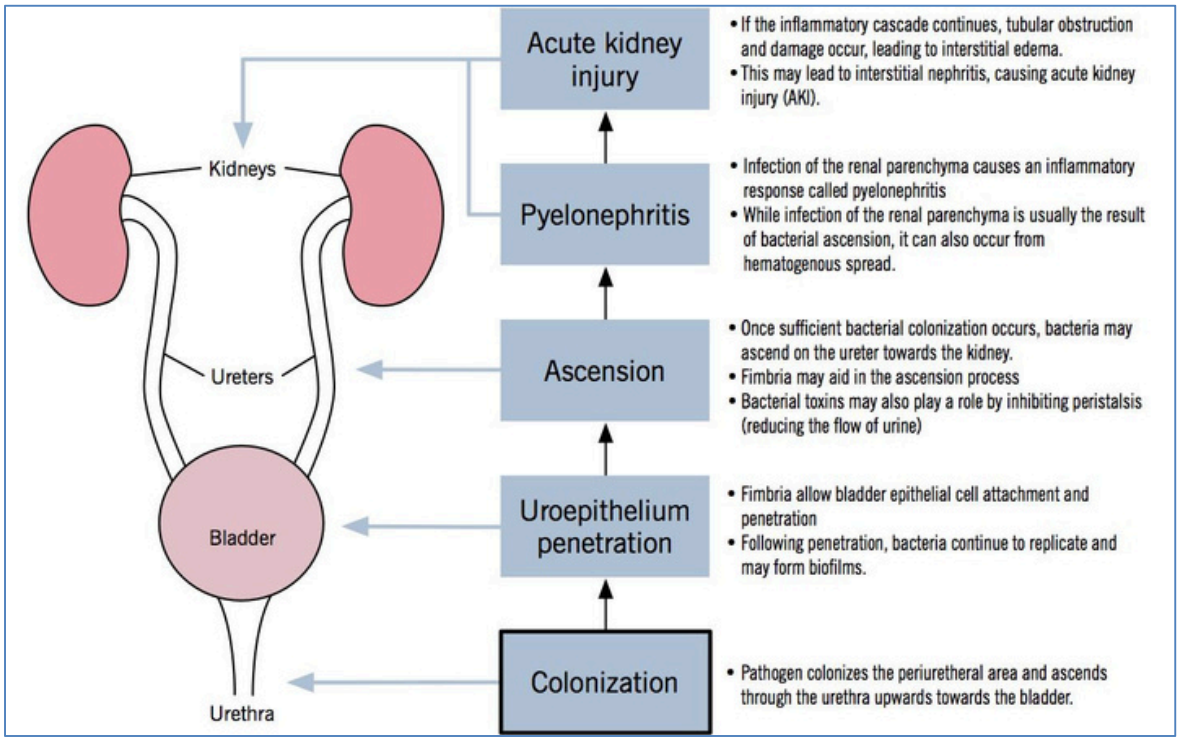


Daniel B. Green, et al; RadioGraphics 2019 39:1, 80-94; https://pubs.rsna.org/doi/full/10.1148/rg.2019180014

URINARY & KIDNEY INFECTIONS

PYELONEPHRITIS:

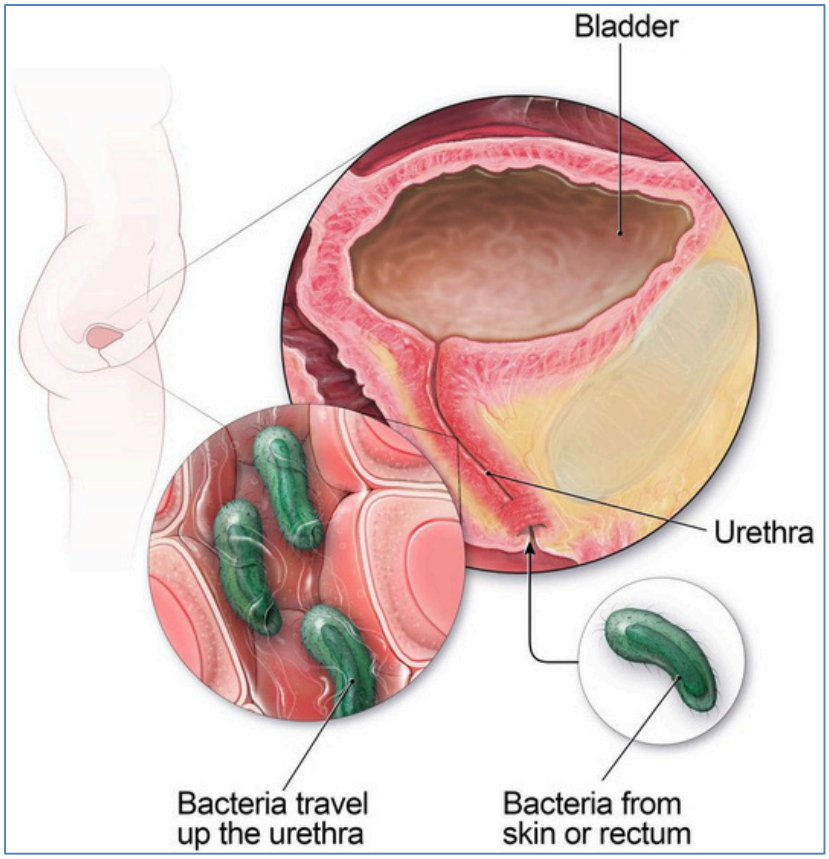
- = Inflammation of the Pyelum (Pelvis) of the Kidney (Which spreads to Tubules & Interstitium)
- **Aetiology:**
  - o Ascending UTI (***E-Coli = Most Common***)
  - o Sepsis (Seeding of bloodborne infection in to kidneys)
- **Risk Factors:**
  - o Anatomical urinary tract abnormalities
  - o o Vesicoureteric reflux (VUR)
  - o Immunocompromised
    - Urinary catheters
    - Diabetes
  - o Prostatic hypertrophy
- **Pathogenesis:**
  - o Ascending UTI → Inflammation
  - o Or – Bacteria in Blood Depositing in Kidneys → Inflammation
  - o → Heavy Inflammation of **Tubules & Interstitium** → Infiltration of Lymphocytes
  - o → Interstitial abscesses filled with pus
- **Clinical Features:**
  - o Fever, Nausea/Vomiting
  - o Pyuria +/- Haematuria
  - o Dysuria, Frequency, Urgency
  - o Flank→Groin Pain
  - o **Renal Angle Tenderness** (Murphey's Kidney Punch Positive)
- **Diagnosis:**
  - o Clinical diagnosis
  - o Pyuria/Haematuria, Bacteriuria
  - o Urine Culture & Sensitivity to guide antibiotic therapy
- **Management:**
  - o **Eg: Oral Antibiotics – *Eg: Ciprofloxacin, Augmentin, Bactrim***
  - o **Eg: IV Antibiotics -** fluoroquinolone, aminoglycoside, or a third-generation cephalosporin.
- **Complications:**
  - o Chronic Pyelonephritis (Repeated episodes → Fibrosis/scarring/atrophy)
  - o Sepsis
  - o Acute Renal Failure



Source: http://www.pathophys.org/uti/

URINARY TRACT INFECTIONS / ("CYSTITIS"):

- **Aetiology:**
  - Bacterial/fungal infection
  - Typically E-Coli / S-Saprophyticus.
- **Risk Factors:**
  - o Incontinence
  - Female (Shorter urethra)
  - Indwelling Catheter
  - Sexual intercourse
  - Diabetes
- **Pathogenesis:**
  - o Ascending infection → bacteria move from rectal area → urethra → bladder
  - o Descending infection → bacteria starts in blood/lymph → kidney → bladder, urethra
- **Clinical Features:**
  - o Suprapubic pain, dysuria, frequent urination/urgency, urine voids small in volume
  - **Babies**: fussy, fever, difficulties feeding
  - o **Elderly**: fatigue, incontinence, altered mental status
- **Diagnosis:**
  - o Urine Microscopy, Culture & Sensitivity
    - § Presence of bacteria
    - § Presence of Nitrites
    - § Presence of Pyuria
    - § Possibly haematuria
    - § Presence of leukocyte esterase.
- **Management:**
  - o Antibiotics: trimethoprim-sulfamethoxazole, ciprofloxacin, ceftriaxone, azithromycin, penicillin
  - o Minimise risk factors
  - Increase oral fluid intake
- **Complications:**
  - Pyelonephritis
  - Urosepsis



Source: https://www.cdc.gov/antibiotic-use/uti.html

RENAL AND PERINEPHRIC ABSCESS

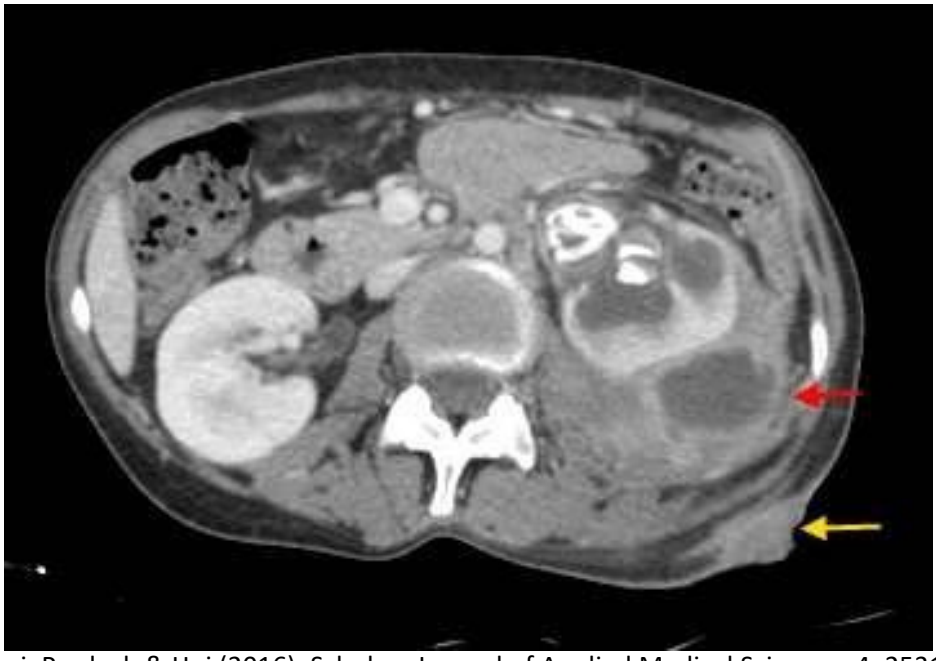
- **Aetiology:**
- o **Complication of pyelonephritis**. Commonly ***E. Coli***.

Presentation:

- o Similar to severe pyelonephritis:
- o fever, flank pain, abdominal pain, dysuria and/or frequency. A palpable mass may or may not be present
- o In perinephric abscess there may be an inflammatory reaction in the overlying skin.

- **Diagnosis:**
  - o Pyuria/Haematuria, Bacteriuria
  - o Urine Culture & Sensitivity to guide antibiotic therapy
  - o Imaging (CT/USS)
- **Treatment:**
  - o **FNA – (*Drain abscess*).**
  - o **Antibiotics – (*Trimethoprim-Sulphamethoxazole*)**
  - o ***Treat underlying cause*** **(if stones etc)**

Neglected staghorn calculus presenting with perinephric abscess and discharging lumbar sinus-a case report:



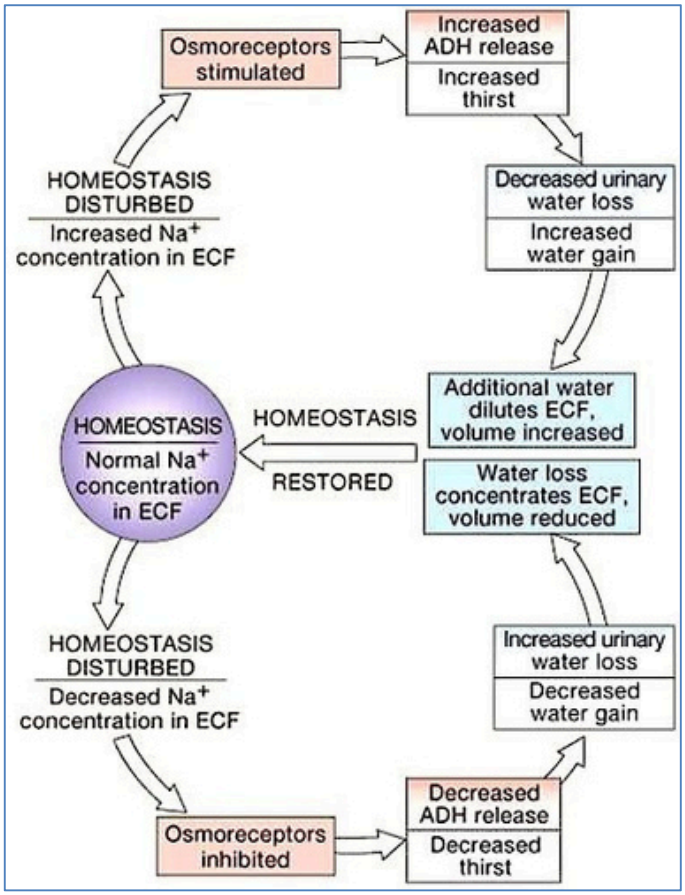
Sarangi, Pradosh & Hui (2016). Scholars Journal of Applied Medical Sciences. 4. 2531-2534. 10.21276/sjams.2016.4.7.47.

ELECTROLYTE IMBALANCES

OSMOLAR IMBALANCES:

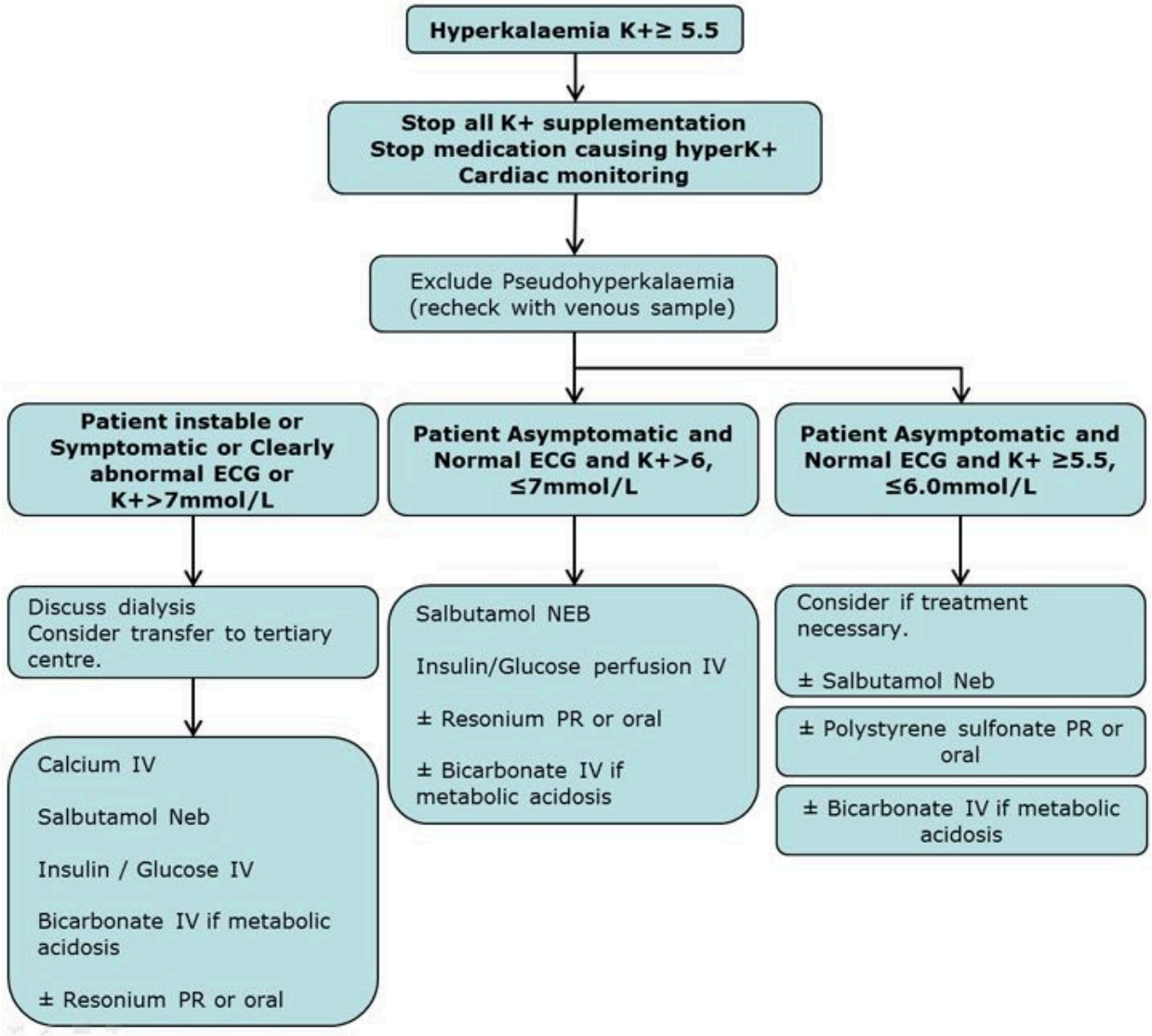
- **Sodium (Na+):**
  - **Hypernatraemia:**
    - § Higher-Than-Normal Blood [Na+]
    - § **May Be Due to:**
      - Decreased H2O Intake/Increased H2O Loss (Due to Reverse-Dilution Effect)
      - Over-Ingestion of Na+
      - Renal Insufficiency
    - § **Leads to:**
      - Cell-Shrinking (Due to Osmosis)
      - If due to H2O Loss, then Hypotension → Tachycardia (to ↑Cardiac Output)
      - Excessive Thirst.
    - § **Treatment:**
      - Water

  - **Hyponatraemia:**
    - § Lower-Than-Normal Blood [Na+]
    - § **May be Due to:**
      - Loss of Na+ from body Fluids...OR
      - Excessive Gain in Extracellular Water (Dilution Effect)
      - (Diuretic Therapy)
      - (Adrenal Insufficiency)
    - § **Leads to:**
      - Cell-Swelling (Due to Osmosis) → Oedema
      - Especially Cerebral Oedema → Headache → Eventually Coma
    - § **Treatment:**
      - Withdrawal of Diuretic
      - Reduce Fluid Intake

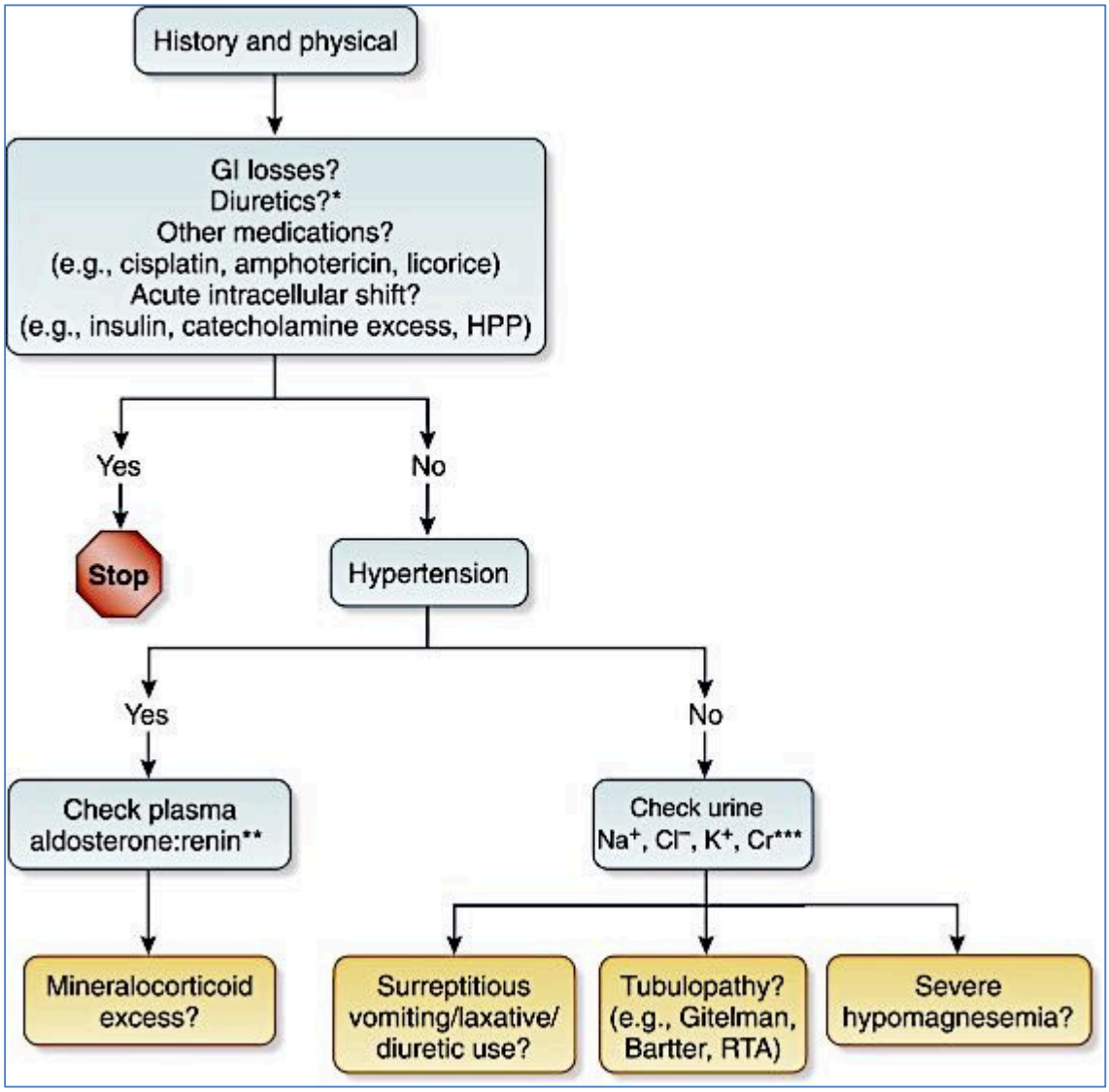


https://www.austincc.edu/apreview/EmphasisItems/Electrolytefluidbalance.html

- **Potassium (K+):**
  - (Note: K+ is needed to *repolarise* excitable membranes.)
  - **Hyperkalaemia:**
    - Higher-Than-Normal Blood [K+]
    - **May Be Due to:**
      - Excessive K+ Intake; OR
      - Renal Failure (Insufficient K+ Excretion in Urine)
      - Large Crush/Trauma Injuries (Rupturing of Cell membranes → Release of K+)
    - **Leads to:**
      - Slower/Poor Repolarisation of Excitable Membranes:
        - →Muscle Cramping
        - → ↓Conductivity of the Heart
    - **Treatment:**
      - Calcium Supplements – Not to lower K+, but to ↓Cardiac Excitability.
      - IV Insulin → Shifts K+ into the cells.
      - Bicarbonate Therapy – Stimulates Na/K-ATPase (Exchanges K+ for Na+)
      - Severe Cases may require Dialysis.



- **Hypokalaemia:**
  - Lower-Than-Normal Blood [K+]
  - **May Be Due to:**
    - Insufficient K+ Intake; OR
    - Excessive Loss of K+
    - (Use of Diuretics)
  - **Leads To:**
    - Faster/Hyper- Repolarisation of Excitable Membranes:
      - → Decreased Excitability of Muscle/Nerve Cells
      - → Cardiac Irritability → Dysrhythmias
  - **Treatment:**
    - Treat the Cause (Eg: Diet/Diarrhoea/Medication)
    - Or – Potassium Supplements.

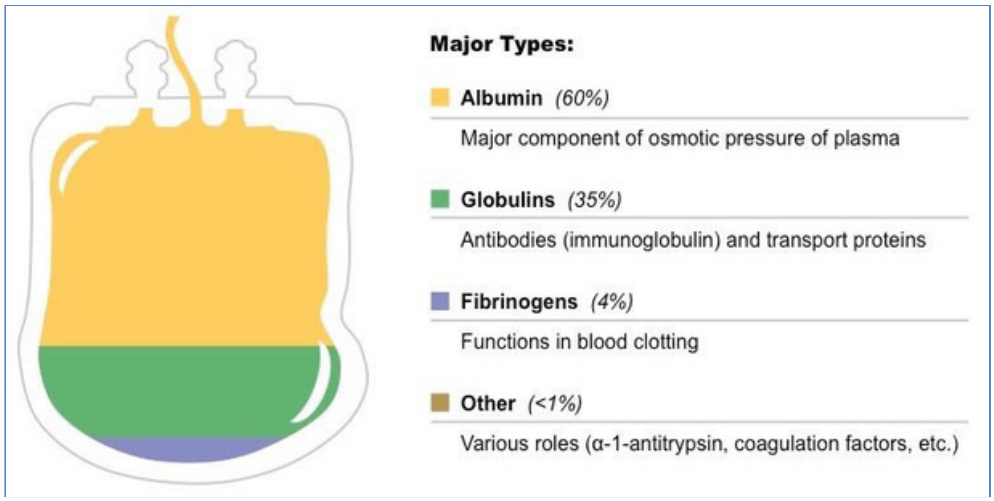


Source: Unattributable

- **Calcium (Ca+):**
  - (Note: Ca+ is needed for normal Heart/Cardiac-Nerve Function, as well as Bone Formation)
  - **Hypercalcaemia:**
    - Higher-Than-Normal Blood [Ca+]
    - **May be Due to:**
      - Increased Dietary Calcium
      - Decreased Ca+ Excretion
      - Shift from Bone → Extracellular Fluid.
    - **Leads to:**
      - Shortened AP-Plateau → Cardiac Arrhythmias
      - Muscle Weakness
    - **Treatment:**
      - Overhydration +Salt → Then Loop Diuretics to depress renal Ca+ Resorption
  - **Hypocalcaemia:**
    - Lower-Than-Normal Blood [Ca+]
    - **May be Due to:**
      - Insufficient Dietary Calcium
      - Increased Ca+ Excretion
    - **Leads to:**
      - Prolonged Depolarisation of Cardiac Action Potentials
      - Impaired Contraction
    - **Treatment:**
      - IV Calcium Replacement.

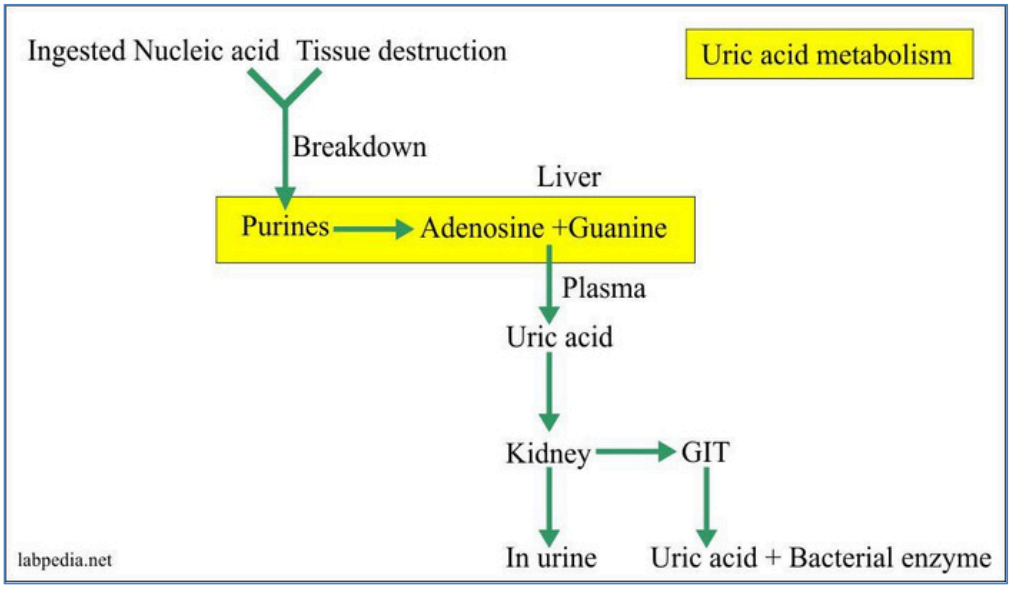
- **Phosphates (HPO2-4):**
  - (Note: HPO2-4 are important for bone formation – Bone Salts = calcium & phosphates)
  - **Hyperphosphataemia:**
    - Higher-Than-Normal Blood [HPO2-4]
    - **May be Due to:**
      - Hypo-Parathyroidism: Low (PTH) → Phosphate Reabsorption From bone.
      - Renal Failure: Increased Phosphate Retention in the Kidneys
    - **Leads to:**
      - Deposition of Ca+ Salts in Soft Tissues → Hypocalcaemia
    - **Treatment:**
      - Phosphate Binders (→↓Dietary Absorption of Phosphates)
      - Dietary Phosphate Restriction.
  - **Hypophosphataemia:**
    - Lower-Than-Normal Blood [HPO2-4]
    - **May be Due to:**
      - Decreased Intake
      - Chronic Alcoholism
      - Long-Term Antacid Use
    - **Leads to:**
      - Decreased ATP (As phosphates are needed for ATP synthesis)
        - →Muscle Weakness
        - →Impaired Cardiac Function
        - →Impaired Neural Function
    - **Treatment:**
      - IV Phosphate Replacement

- **Plasma Proteins:**
  - o (Note: Plasma Proteins – Important in regulating blood Volume & Viscosity/Pressure)
  - o **Hyperproteinaemia:**
    - § Higher-Than-Normal Blood [Protein]
    - § Rare
  - o **Hypoproteinaemia:**
    - § Lower-Than-Normal Blood [Protein]
    - § **May Be Due To:**
      - Liver Failure (As the liver makes the Plasma Proteins)
      - Protein Malnutrition
      - Burns
      - Kidney Failure (Proteinuria – Loss of Protein in Urine)
    - § **Leads to:**
      - Reduced Plasma Osmotic Pressure
        - o →Widespread Oedema



Source: Unattributable

- **Uric Acid:**
  - o (Note: Uric Acid = Metabolic Waste Product of Protein Metabolism. Excreted through Urine)
  - o **Hyperuricaemia:**
    - § Higher-Than-Normal Blood [Uric Acid]
    - § **May Be Due To:**
      - Renal Failure – Plasma Uric Acid isn't being excreted through kidneys.
    - § **Leads To:**
      - Gout: Deposition of Uric-Acid Crystals in Joints → Arthritis of Gout.



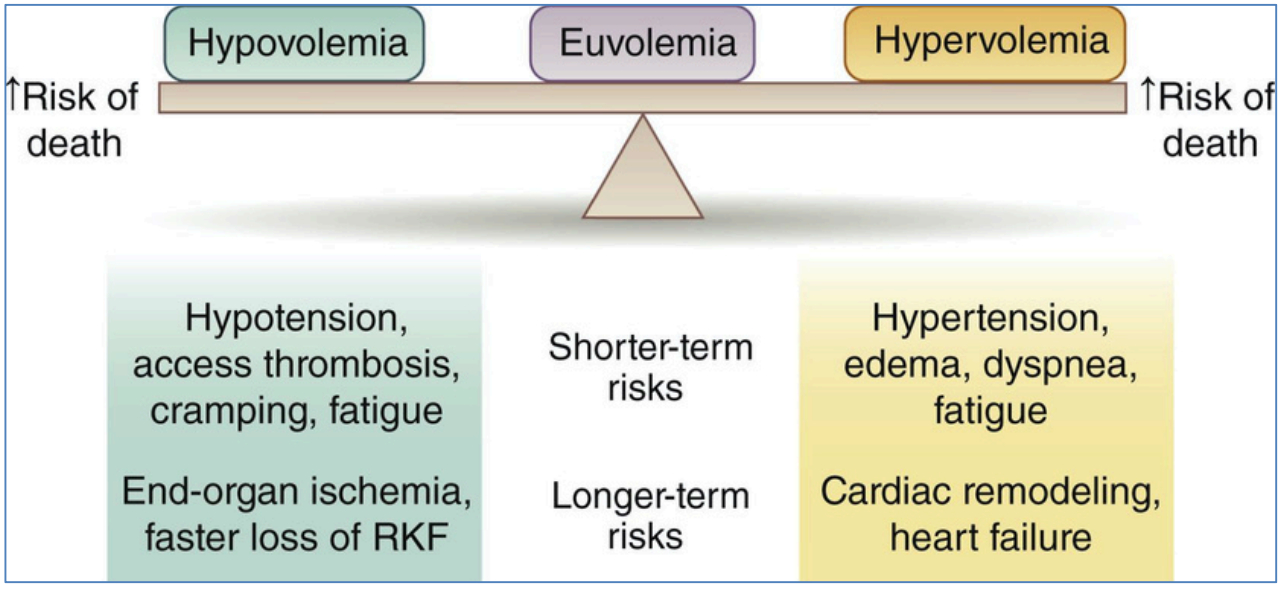
Source: https://labpedia.net/uric-acid-level-blood-uric-acid/

FLUID IMBALANCES

VOLUME IMBALANCES:

- **Hypervolaemia:**
  - A Gain of Extracellular Fluid (And an Associated gain in Na+)
  - **Symptoms:**
    - Hypertension
    - Oedema
  - **May Be Due To:**
    - Excessive Fluid Intake
    - Chronic Renal Failure (↓Urine Output)
    - Endocrine Imbalances (Eg: ADH & Aldosterone)
  - **Treatment:**
    - Diuretics

- **Hypovolaemia:**
  - A Loss of Extracellular Fluid (And an Associated loss of Na+)
  - **Symptoms:**
    - Hypotension
    - Tachycardia
    - High Resp. Rate
    - Thirst
  - **May Be Due To:**
    - Insufficient Intake of Fluids
    - Haemorrhage
    - Diarrhoea
    - Vomiting
    - Endocrine Imbalances (Eg: ADH & Aldosterone)
  - **Treatment**:
    - Fluid Replacement (Saline IV Fluids or Electrolyte Drink)



Source: https://www.researchgate.net/figure/Tension-in-balancing-volume-status-within-a-narrow-therapeutic-window-RKF-residual_fig1_339786229

DIURETICS

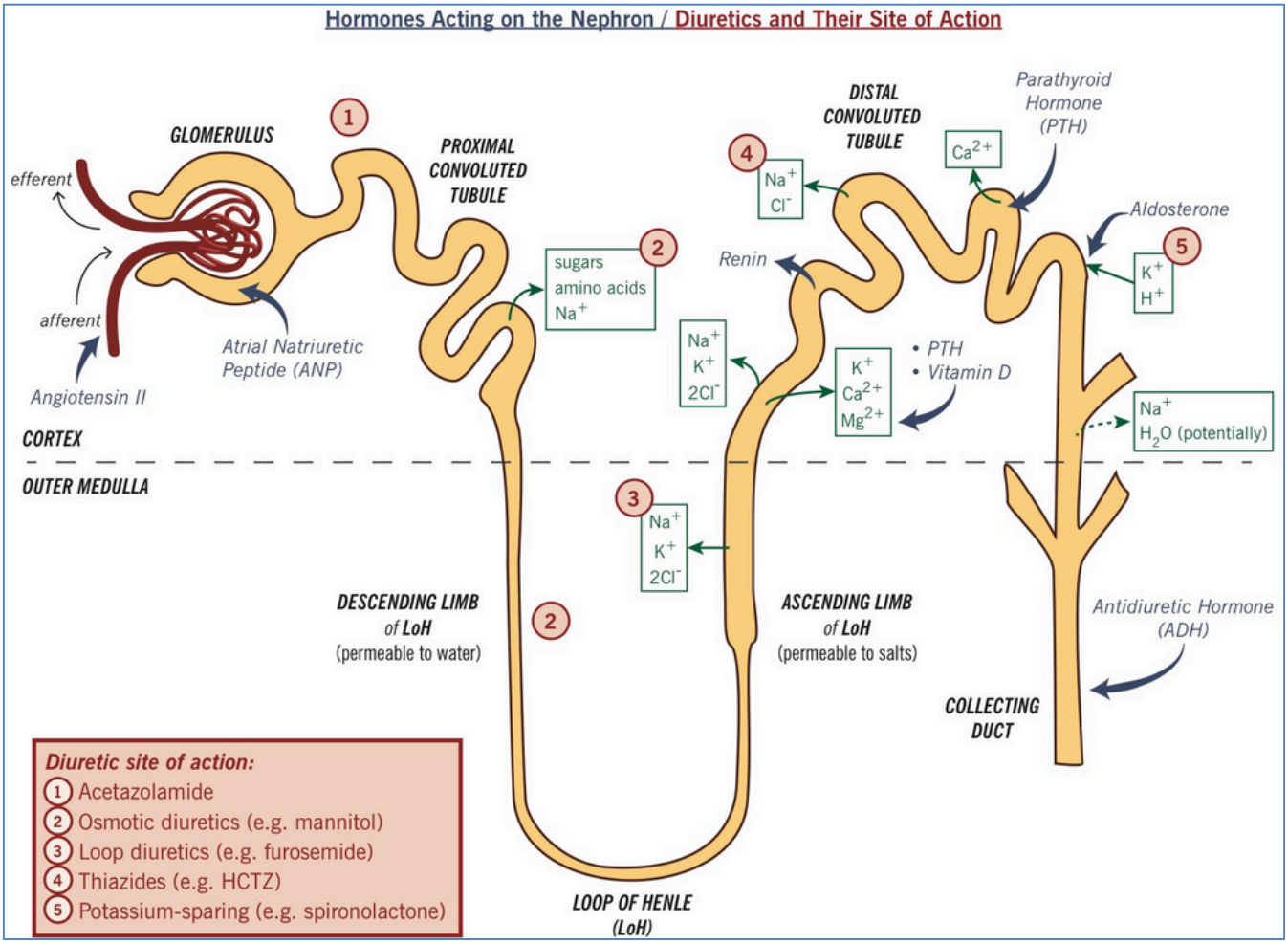
DIURETICS:

Drugs that ↓Na+ Reabsorption in the Kidneys → ↑H2O Excretion:

- **→ Net Loss of Na+ and therefore Water as well.**

Note: The [Na+] decreases as you travel down the Nephron:

- **Therefore, the *Effectiveness* of the Diuretic depends on its *Site of Action*:**
  - o **Eg: If Proximal Tubule (Osmotic Diuretics)** **[65% Na+]** – Very Effective
  - o **Eg: If Loop of Henle (Loop Diuretics)** **[25% Na+]** – Effective
  - o **Eg: If Distal Tubule (Thiazide Diuretics)** **[5% Na+]** – Low Effectiveness
  - o **Eg: If Collecting Ducts (K+ Sparing Diuretics)** **[2% Na+] –** Very Low Effectiveness



Source: http://www.pathophys.org/diuretics/

- **The Catch: It is difficult to *only* manipulate Na+; (Some are 'K+-Wasting'; some are 'K+-Sparing'):**
  - o Hence why Combinations – often used to balance K+ Movement
  - o **However,** *even a 'balanced diet' of Diuretics* can slowly lead to *Hypokalaemia* if not monitored

W hy Use Diuretics?:

- Treatment of *Mild* Hypertension:
- o Note: Diuretics are better than β-Blockers in *Every Way*. (↓Cost/Side Effects)
- Treatment of Acute Renal Failure
- Treatment of Oedema

Treatment of Congestive Heart Failure:

- o - to ↓Fluid Volume & ↓BP → ↓Preload → Treat Heart Failure.

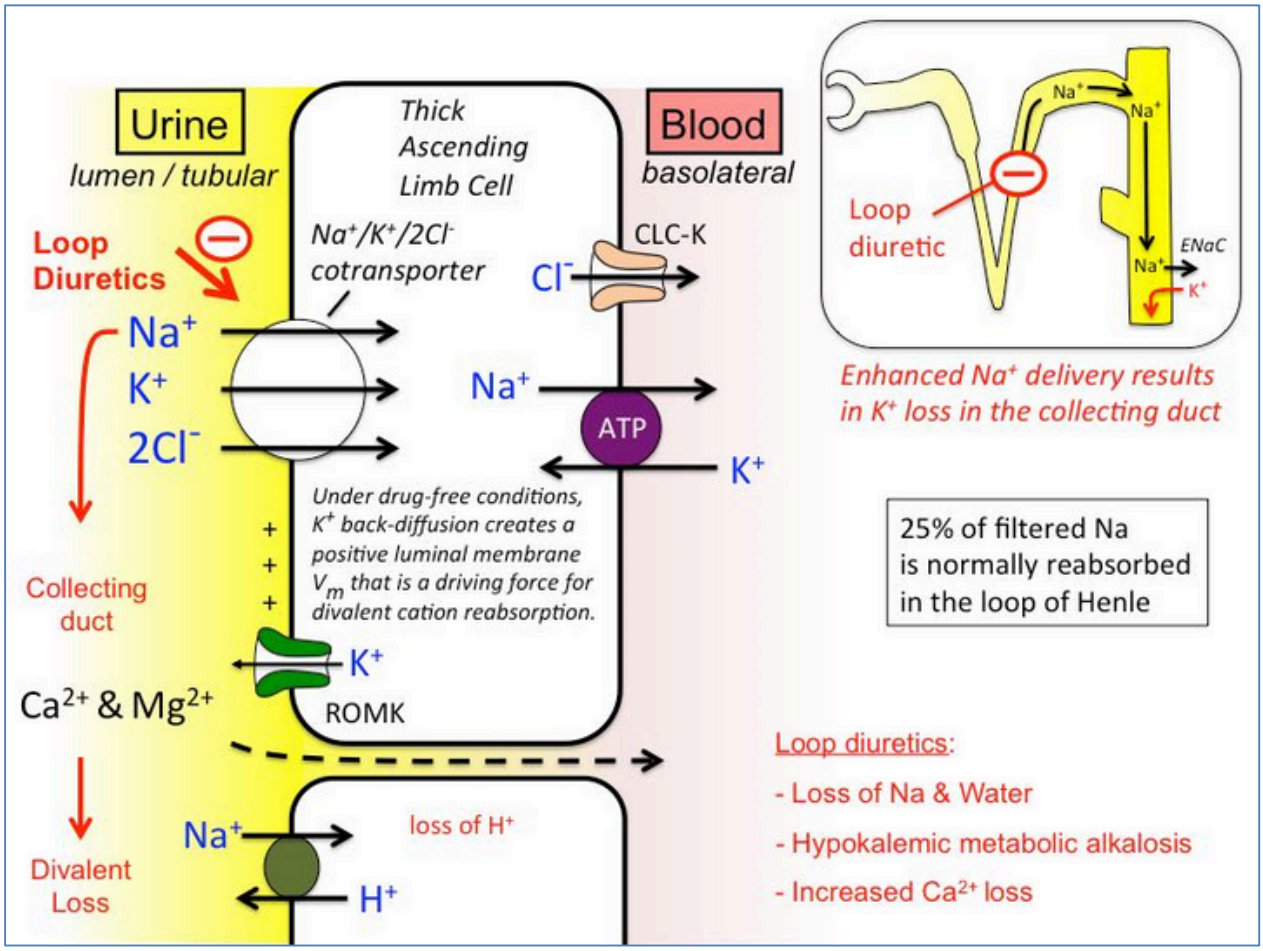
Loop Diuretics: (Most Powerful – *BUT* *Potassium-Wasting*)

- **Site of Action:**
  - The Thick Ascending Loop of Henle

Mechanism of Action:

- Inhibiting the Na/K/Cl-Transporter in the Thick-Ascending Loop of Henle.
- → prevents Na+ Resorption into Interstitium (Therefore Prevents H2O Resorption)
  - (Note: Also prevents K+ & Cl- Reabsorption)
- *→*Prevents* formation of the 'Hyperosmotic Medullary Interstitium' that ordinarily facilitates Water Resorption (under the influence of ADH).

- **Indications:**
  - Acute Pulmonary Oedema
  - Heart Failure
  - Ascites (due to hepatic cirrhosis)
  - Renal Failure
  - (Note: Thiazides are preferred for Hypertension.)
- **Side Effects:**
  - Hypovolaemia & Hypotension.
  - Hypokalaemia (Due to inhibition of K+ Reabsorption):
    - May require Potassium Supplements, Or coupling with **K+-Sparing Diuretics**.
    - (Note: Can increase Digoxin Toxicity)
  - Metabolic Alkalosis (Due to *reverse dilatation effect* of H2O loss, but *no* HCO3 Loss):
    - Aka: "Concentration Alkalosis".
  - Hyperuricaemia → Gout.
  - Reversible Hearing Loss (Same co-transporter is found in the Ear)
- **Classical Agents:**
  - ***Frusemide**
  - Bumetanide
  - Ethioyic acid



https://tmedweb.tulane.edu/pharmwiki/doku.php/loop_diuretics

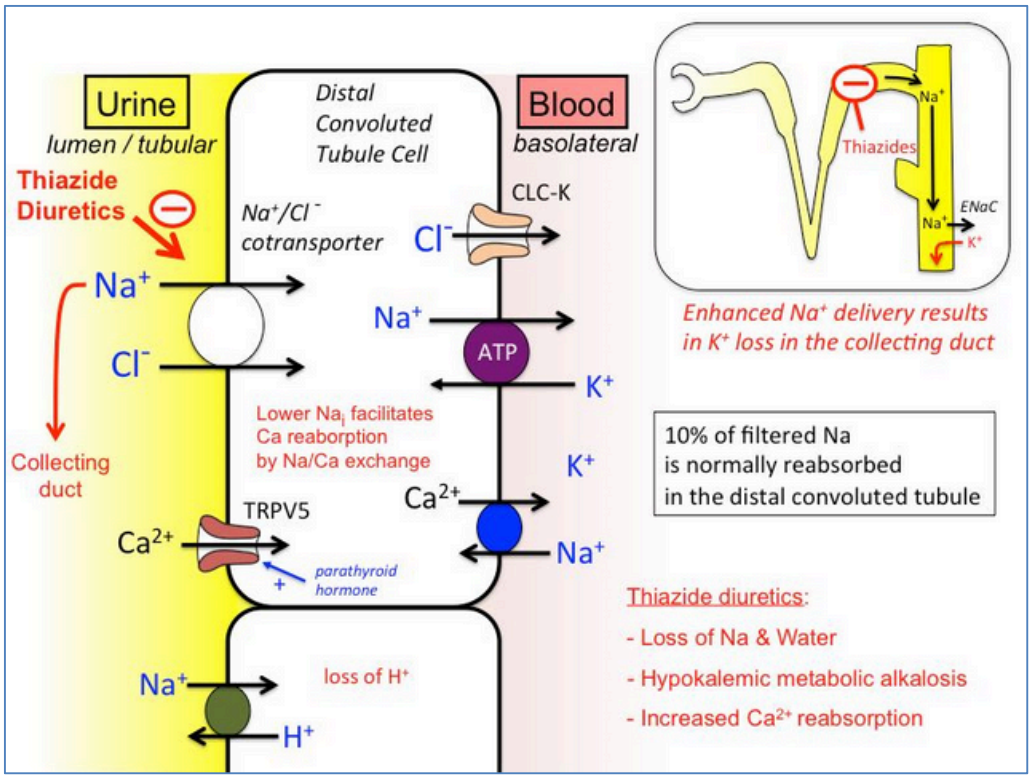
Thiazide Diuretics: (Not as powerful as Loop Diuretics – *And Still Potassium-Wasting*)

- **Site of Action:**
  - Distal Convoluted Tubules

Mechanism of Action:

- Inhibiting the Na/Cl Symporter in the DCT.
- → prevents Na+ Resorption into Interstitium (Therefore Prevents H2O Resorption)
  - (Note: Also prevents Cl- Reabsorption)
  - (Note: Still K+ Wasting)
- Maintains a High Filtrate Osmolality → Retaining Water in the Tubule.

- **Indications:**
  - **Uncomplicated Hypertension – (One of the 1st lines of treatment for hypertension)
  - Severe Resistant Oedema
  - Mild Heart Failure
  - Ascites (due to hepatic cirrhosis)
  - Renal Failure
- **Side Effects:**
  - Hypovolaemia & Hypotension.
  - Hypokalaemia:
    - May require Potassium Supplements, Or coupling with **K+-Sparing Diuretics**.
    - (Note: Can increase Digoxin Toxicity)
  - Hyponatraemia:
    - Can be Fatal.
  - Hypomagnesaemia
  - Hypocalciuria (Hypercalcaemia):
    - (Note: May be beneficial in elderly patients for Bone Metabolism)
  - Metabolic Alkalosis (Due to *reverse dilatation effect* of H2O loss, but *no* HCO3 Loss):
    - Aka: "Concentration Alkalosis".
  - Hyperuricaemia → Gout
  - Hyperglycaemia:
    - Can unmask latent Diabetes Mellitus.
  - Reversible Erectile Dysfunction
- **Classical Agents:**
  - ***Chlorothiazide**
  - Chlortalidone



https://tmedweb.tulane.edu/pharmwiki/doku.php/thiazides?s[]=thiazide

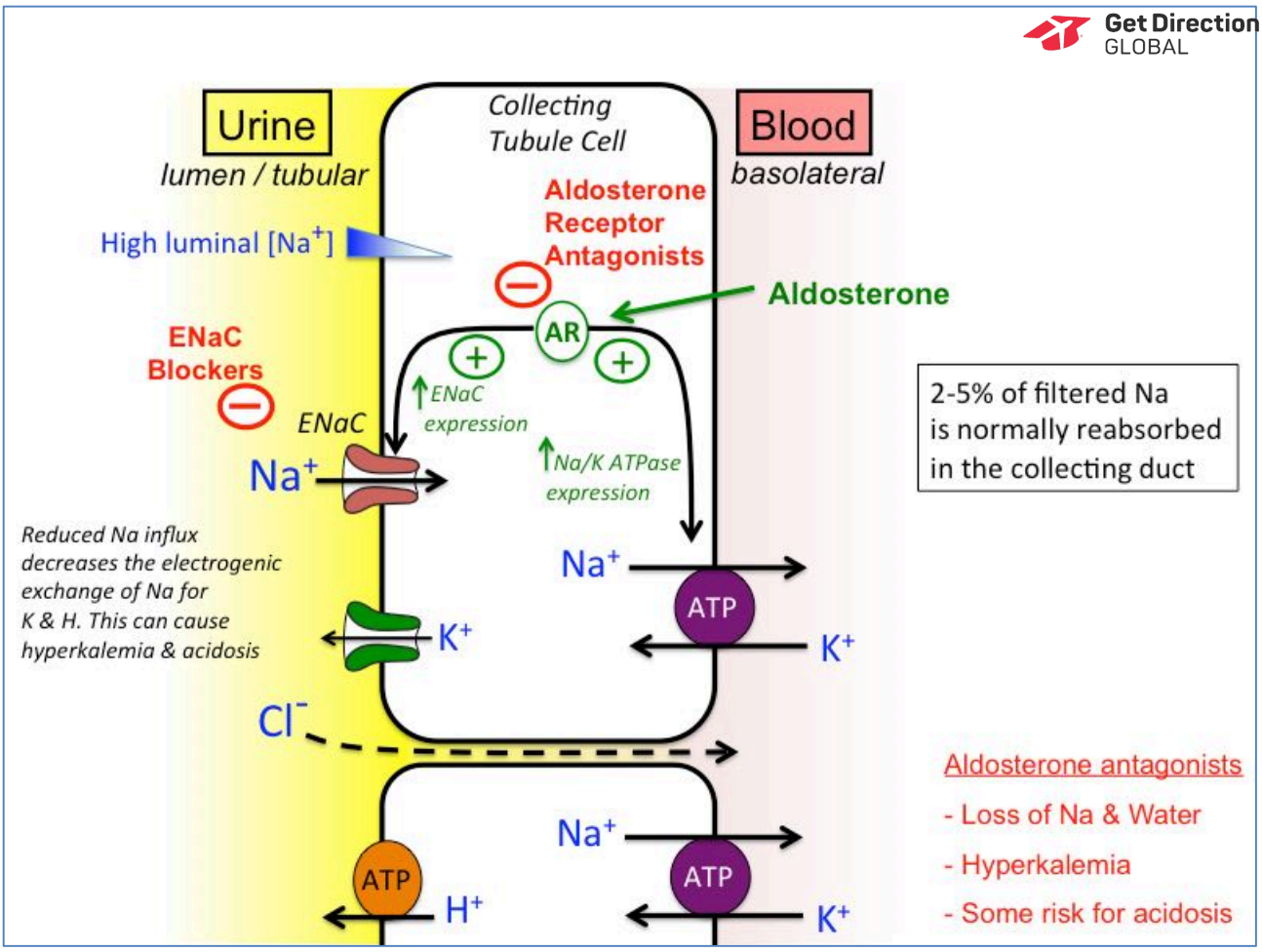
- **K+ Sparing Diuretics:**
  - **Site of Action:**
    - Collecting Ducts
  - **Indications – (Common for both):**
    - Used in Pts where K+ Loss is Hazardous – (Eg: Pts on Digoxin or Amiodarone)
    - Heart Failure
    - Hyperaldosteronism
    - Resistant Essential Hypertension (Eg: Low-Renin Hypertension)
    - Ascites (Due to Hepatic Cirrhosis)
  - **1- Epithelial Na+ Channel Inhibitors:**
    - **Mechanism of Action:**
      - **Directly Inhibits the *Aldosterone-Activated* Na+ Channels in walls of Collecting Ducts:**
        - → Inhibits H2O Resorption.
      - **K+ Sparing Effect** comes from a *Loss* of Na+-Concentration Gradient which normally powers a *Secondary-Active Na/K-Symporter* on Basal Membrane.
    - **Classical Agents:**
      - *Amiloride
      - Triamterene
    - **Side Effects:**
      - Hyperkalaemia – (Potentially Fatal)
      - Hence: Avoid in Pts with Renal Failure/ACE-Inhibitors/K+ Supplements

        Avoid NSAID Use – (Possible drug interaction)
  - **2- Aldosterone Antagonists:**
    - **Background on Aldosterone Function:**
      - Aldosterone is a Steroid Hormone → Causes Expression of Proteins:
        - Na+ Channel Proteins – (Responsible for Na+ Resorption).
        - TCA-cycle Enzymes → ↑ATP – (ATP is responsible for Na Pump).
      - Therefore, Aldosterone is Responsible for Na+ Resorption in Collecting Duct.
    - **Mechanism of Action of *Aldosterone Antagonists*:**
      - **Prevents Aldosterone from binding to its Nuclear Receptor → Prevents Expression of the Above Proteins**.
        - → ↓Na+ Channel Proteins → ↓Na+ Resorption → Inhibits H2O Resorption.
        - → ↓TCA Enzymes → ↓ATP → ↓Na+ Pump Function → ↓Na+ Resorption
      - **Ultimately → ↓ H2O Resorption**.
      - **Note:** ONLY works when Renin-Angiotensin System is Active.
        - Ie: Efficacy depends on Endogenous Aldosterone Level.

        K+ Sparing Effect comes from a *Loss* of Na+-Concentration Gradient which normally powers a *Secondary-Active Na/K-Symporter* on Basal Membrane.
    - **Classical Agents:**
    - *Spironolactone

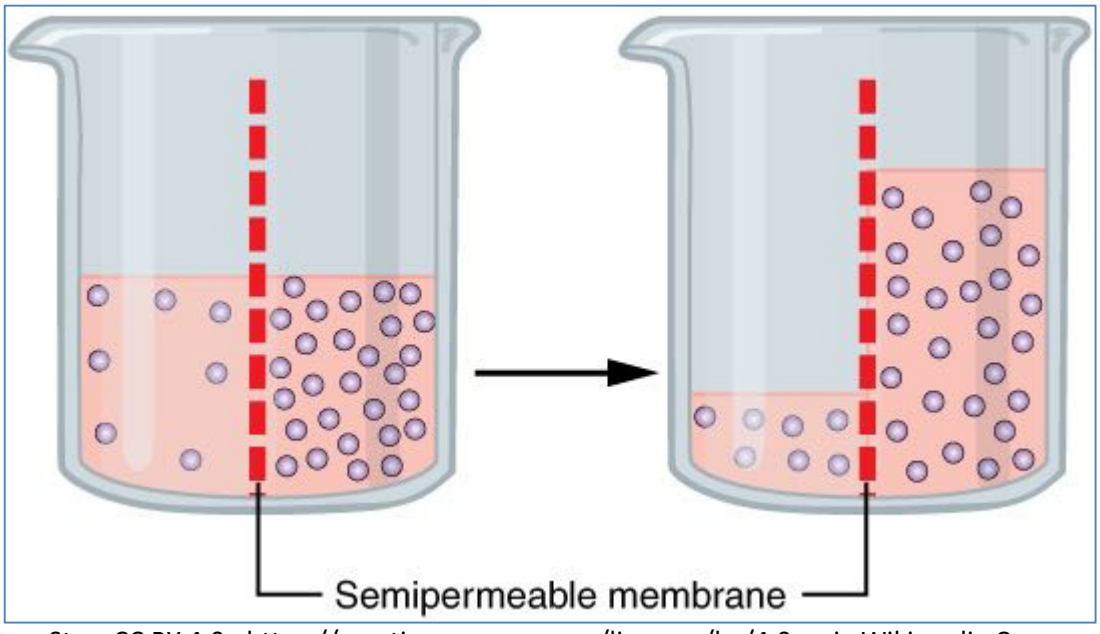
      Side Effects:
      - Hyperkalaemia – (Potentially Fatal)
        - Hence: Avoid in Pts with Renal Failure/ACE-Inhibitors/K+ Supplements
      - GI Upset
      - Gynaecomastia
      - Menstrual Disorders

        Testicular Atrophy



https://tmedweb.tulane.edu/pharmwiki/doku.php/potassium_sparing_diuretics

- **Osmotic Diuretic Drugs:**
  - **Site of Action:**
    - Filtered in the Glomerulus.
    - Affects Any Nephron that is Freely Permeable to Water.
    - **- Mainly The Loop of Henle
  - **Mechanism of Action:**
    - Inert Substances (Eg: Sugars) that are filtered by the Kidneys, but not reabsorbed.
      - →Increases Filtrate Osmolality to:
        - →Inhibit Passive Water Reabsorption.
        - →Facilitate Passive Water Excretion.
    - Ie: An example of *Physiological Antagonism*.
  - **Indications:**
    - Acute Renal Failure – Prevent kidneys from drying out.
    - Cerebral Oedema & Intraocular Pressure:
      - Simply by increasing Plasma Osmolality.
      - Relieves such pressures via osmosis.
  - **Classical Agent:**
    - *Mannitol
    - Isosorbide
    - Glycerin
  - **Side Effects:**
    - Transient Hypervolaemia (Ie: ↑Extracellular Fluid – due to ↑Plasma Osmolality)
      - Can →Dilution Hyponatraemia
      - Can →Heart Failure
      - Can →Pulmonary Oedema
    - Headache, Nausea & Vomiting.



DRUGS ALTERING THE pH URINE:

DRUGS ALTERING THE pH URINE:

Get Direction GLOBAL

Clinical Significance:

- The pH of the Urine affects the Excretion Rates of different Drugs. (Depending if drug is acidic or basic)
- **Urine Alkalinisation:**
  - **Excretion:**
    - Increases the Excretion of Weak-Acid Drugs. (Eg: Salicylates/Aspirin & Barbiturates)
      - Ie: Bicarbonate is sometimes used to treat Overdoses of the above.
    - Decreases the Excretion of Weak-Base Drugs.
  - **Precipitation:**
    - Can prevent Weak-Acid Drugs from Precipitating in the Urine (↓kidney stones).
    - Also decreases Precipitation of Uric Acid Crystals in the Urine (↓kidney stones).
- **Urine Acidification – (Rarely Ever Used):**
  - **Excretion:**
    - Increases the Excretion of Weak-Base Drugs.
    - Decreases the Excretion of Weak-Acid Drugs. (Eg: Salicylates & Barbiturates)
  - **Precipitation:**
    - Can prevent Weak-Base Drugs from Precipitating in the Urine (↓kidney stones).

Urinary Alkalizers:

- **Carbonic Anhydrase Inhibitors:**
  - **Mechanism of Action:**
    - Blocks Bicarbonate Reabsorption → Alkaline Urine (but Metabolic Acidosis)
- **Oral Citrate:**
  - **Mechanism of Action:**
    - Metabolised via TCA-Cycle → Produces Bicarbonate as a by-product.

Urinary Acidifiers – (Rarely Ever Used):

- **Ammonium Chloride:**
  - Only Used Clinically for an oral Acid-Loading test to Diagnose *Renal Tubular Acidosis*.

POPULATION HEALTH & RENAL DISEASE

POPULATION HEALTH & RENAL DISEASE

Get Direction GLOBAL

D e fin itio n s:

- **Renal Failure:**
  - o Sustained, Irreversible reduction in GFR (Glomerular Filtration Rate) to <60mL/min
  - o Raised Creatinine (200+ micro-mol/L)
  - o (On 2 Occasions; 1 Month Apart; With No Acute Illness)
- **End-Stage Renal Disease:**
  - o (GFR = <15ml/min)
  - o Kidney function *Incompatible With Life* → Require Dialysis/Transplant for Survival

Renal Disease: Significance?:

- Growing health issue
- Economic Costs:
  - o Public Health Service
  - o Out-of-Pocket (Patients)
- Personal Burden → ↓Quality of Life
- Medical Care → Positive Outcomes

M ost Com m on Renal *M orbidities*:

- **UTI's (Urinary Tract Infections)**:
  - o Both Children & Adults
  - o Often due to Diabetes → Sugar in Urine → Food For Bacteria
- **Urinary Tract Abnormalities:**
- o Eg: Urinary Reflux – (from Bladder → up the Ureters)

Urinary Incontinence:

- o Childhood Bedwetting
- o Females – Pelvic Floor Weakening (Eg: Following pregnancy)

- **Prostatic Hypertrohpy/Cancer:**
  - o Hypertrophy happens to all men → older
  - o Cancer = common

M ost Com m on Renal *M ortalities*:

- **Prostate Cancer**
- ****End-Stage Renal Disease**

ESRD Risk Factors:

- Childhood PSGN (Post-Streptococcal Glomerular Nephritis)
  - o Auto-immune response to Haemolytic Strep →Inflammatory response manifested by cellular P ro life ra tio n & O e d e m a o f th e G lo m e ru la r tu ft.
- Chronic UTI's
- Kidney/UT Stones
- Inter-Uterine Malnutrition:
  - o (Risk of ESRD begins in-utero – Foetuses that undergo metabolic Insults [poor nutrition/high Blood Sugar(Diabetic Mother)/etc] Actually grow *Less* Kidney Cells)
- Low Birth Weight
- Adult Obesity
- Diabetes (Poorly Controlled)
- Hypertension (Poorly Controlled)
- Smoking
- Poor Access to Services

Prevention of Renal Disease:

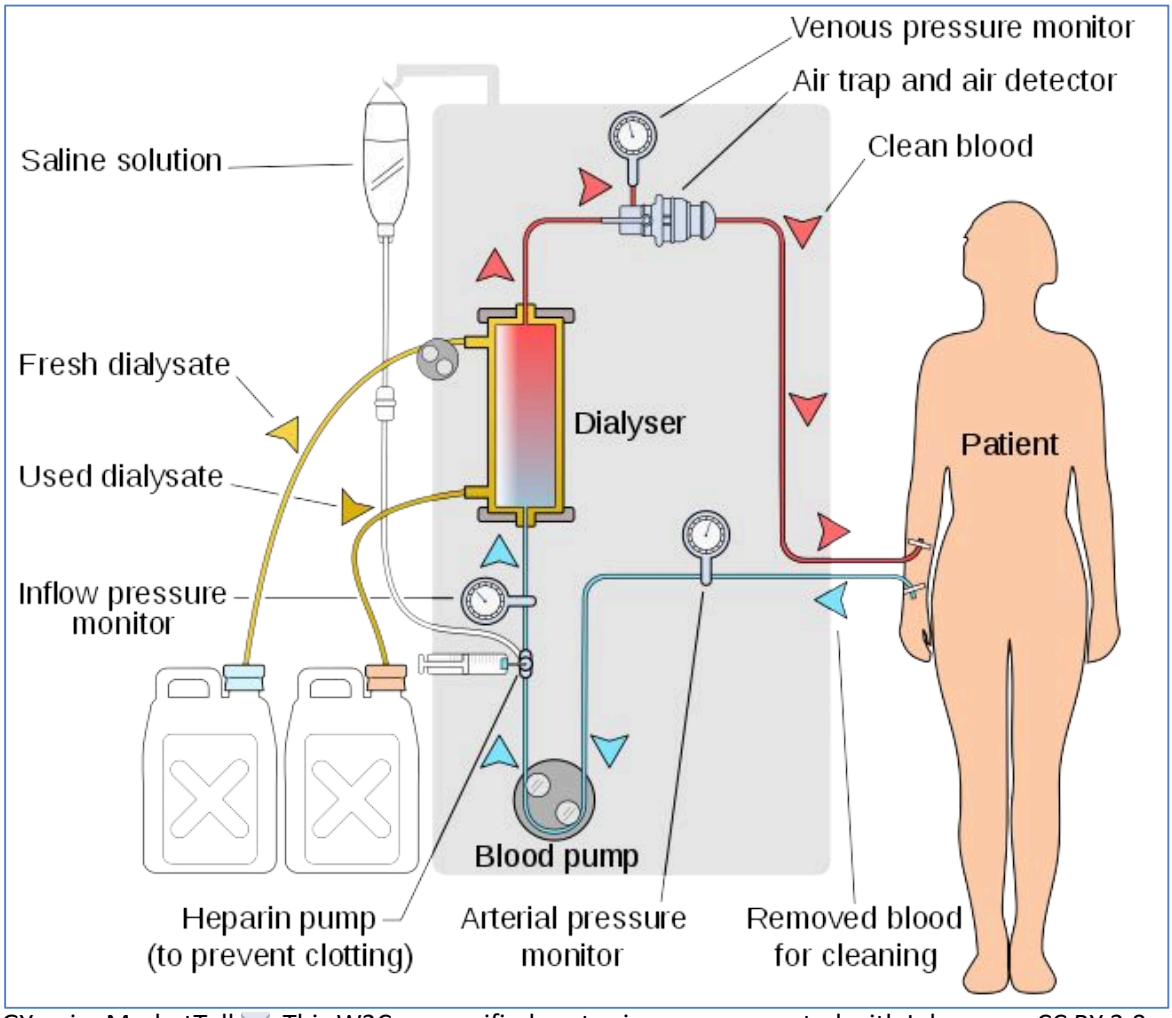
- **Primary Prevention:**
  - Preventing People Getting the Disease in the First Place.
  - **Improve Early-Life Health:**
    - **Note: Barker Hypothesis:** English Doctor – Found a Correlation between Antenatal Health
    - & Later-Life Health. – This relationship exists for Renal Disease.

      Antenatal Care:
      - Control of Mother's Diabetes
      - Nutritional Supplements for Extra Demands of Foetus
    - **Infant/Childhood Nutrition:**
      - Breast-Feeding - (Best for nutrients & immunity)
      - Avoid Obesity
    - **Exercise**
    - **Growth Monitoring**
  - **Reduce Risk Factors:**
    - Obesity
    - Smoking
    - Fatty Diets
    - Hypercholesterolemia
    - Alcohol

- **Secondary Prevention:**
  - Diagnosing the Disease Early to Optimise Management & Prognosis
  - **Screening For:**
    - Diabetes
    - Hypertension
    - Renal Dysfunction
  - **If Chronic Renal Disease is Present:**
    - Reduce Obesity
    - Exercise
    - Control Fat/Sugar Intake
    - Low GI Foods
    - Medications:
      - Antihypertensives
      - Diabetes Medication
  - **Monitor:**
    - Response to Meds
    - Blood Pressure
    - Blood Sugar Levels
    - Renal Function – Creatinine
  - **Refer:** To Renal Specialists (nephrologists)
    -

ESRD Treatment:

- **Kidney Transplant:**
  - o The Best Treatment
    - § Most Cost-Effective
    - § Most Permanent
- **Dialysis:**
  - o **Peritoneal Dialysis:**
    - § CAPD – Continual Ambulatory Peritoneal Dialysis
    - § Ambulatory Peritoneal Dialysis
  - o **Haemodyalisis:**
    - § Satellite
    - § Hospital
    - § Home



GYassineMrabetTalk✉ This W3C-unspecified vector image was created with Inkscape ., CC BY 3.0 <https://creativecommons.org/licenses/by/3.0>, via Wikimedia Commons

Cost of Renal Disease:

- ****Costs of Renal Disease Are PHENOMENAL****
- **Financial:**
  - o Hospital Services
  - o GP & Specialist Services
  - o Allied Health Costs
  - o Prescriptions
- **Personal Burden:**
  - o Relocation to Areas With Treatment (If Rural Patient)
  - o Loss of Income
  - o ↓Quality of Life
  - o ↓Social/Family Life (Due to Morbidity/Relocation)

Economics of Renal Disease Treatment:

- **Ie: Getting The *Best Outcomes* for The *Least Money*.**
  - Eg: Peritoneal Dialysis or Haemodialysis?
  - Dialysis or Transplant?
  - Dialysis or Diabetes Prevention?
- **How Do We Compare Outcomes of Different Actions?**
  - **Answer = QALY's (Quality-Adjust Life Years)**
    - **1x QALY** = 1 Full Year of Life @ Full Quality of Life
    - Used to Compare Quality & Length Of Life Gained from Different Interventions & the Costs of Doing So
  - **Calculating QALY's:**
    - Multiply the Years Gained From an Intervention by the Quality-Of-Life-Percentage.
    - (Where 100% = Full Health)
  - **Eg:**

Evaluating the benefits gained from screening for Proteinuria and how much each QALYS costs.

Screening for Proteinuria	QALYS gained per person	Cost per QALYS USD$
Well people	0.0022	282,818
With hypertension	0.03	18, 621

- ***Cost-Effectiveness* Analysis:**
  - Aim: To find the Cheapest Way to achieve A *Specific Desired Outcome*.

How we use quality of life years to decide the effectiveness of different treatments

Cost of One Quality Adjusted Life Year

	£
Home haemodialysis	17 260
CAPD	19 870
Hospital haemodialysis	21 970
Kidney transplant	4 710

So much more cost-effective.

- ***Cost-Utility Analysis*:**
  - Aim: To Compare Costs of Interventions With *Different Health Outcomes*.

	Cost per QALY (£)
Cholesterol testing and diet therapy	220
Advice to stop smoking from patient's own doctor	270
Hip replacement for arthritis	1 180
Kidney transplant	4 710
Breast cancer screening	5 780
Neurosurgery for malignant brain tumours	107 780

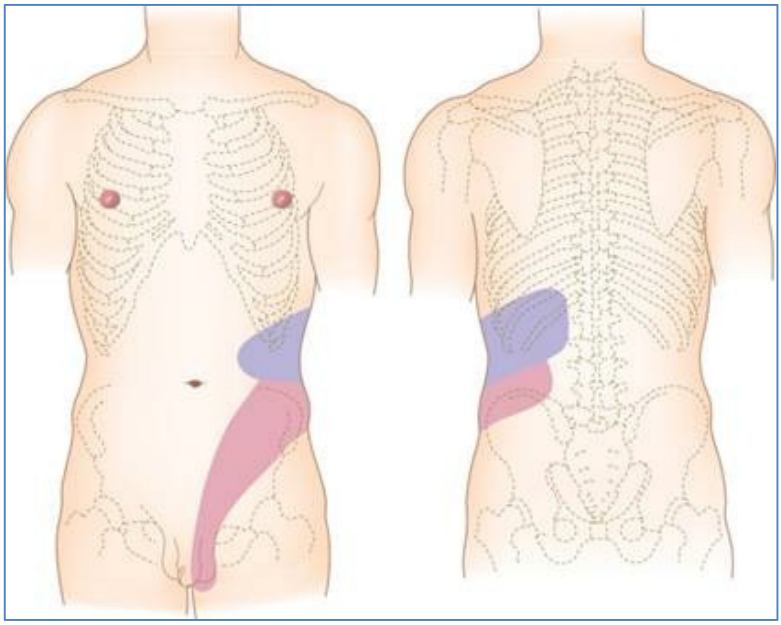
MISCELLANEOUS POINTS

UROGENIC PAIN

- **Nature of Pain may Vary:**
  - **Colicky Pain (Comes & Goes):**
    - Commonly caused by kidney stones
    - Pain comes in Waves due to Ureteric Peristalsis
  - **Constant Pain:**
    - Caused by a constant pathological process (Eg: Pyelonephritis, Ascending UTI, etc)
- **Location of Pain Varies Depending on Organ Affected:**
  - **Kidney Pain:**
    - Unilateral Flank/Back pain Radiating to Groin.

https://medlineplus.gov/ency/patientimages/000283.htm

  - **Ureteral Pain:**
    - Flank-Groin Colicky-Type (Comes & Goes) Pain

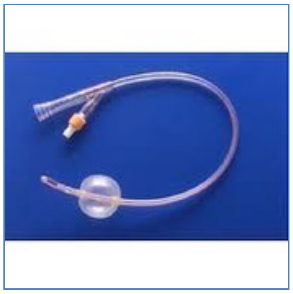


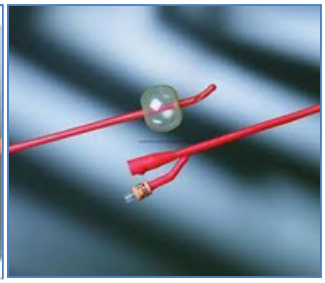
https://www.baus.org.uk/patients/conditions/6/kidney_stones/

  - **Bladder Pain:**
    - Suprapubic Pain
  - **Urethra Pain:**
    - Localised to the Urethra.

CATHETERIZATION (Females and Males):

- **Indications:**
  - Urinary retention
  - Urine Sample
  - Post-operative to assess urinary output, perfusion
  - Prostatic obstruction:
    - BPH [most likely]
    - CA of prostate
  - Other obstructions:
    - Clots
    - Stones
    - Bladder CA
  - Trauma
  - Paralysis
- **Peri-Urethral Structures that might Interfere with Catheterisation:**
  - Labia Foreskin
  - Prostate
  - Urethral Sphincters
  -
- **Different Types of Catheters:**
  - **Foley (Brown Latex):** Cheapest, Commonest
  - **Silastic (Clear Silicone):** can leave in longer than Foley with less chance of complications
  - **Robinson's:** Has no balloon, is used for Short term drainage
  - **Coude:** Angled for easier insertion around prostate



- **Basic Process of Catheterisation:**
  - Initial Steps:
    - Gather Equipment
    - Explain Procedure and get Consent
    - Lay pt into supine position + Spread Legs
    - Prepare Sterile Field + Apply Gloves
    - Cleanse Periurethral Mucosa with Cleansing Solution
  - Check Balloon for Patency
  - Coat the distal 2-5cm with Lubricant
  - Gently Insert Catheter into Urethra until 1-2inches *beyond* the point of Urine Flow.
  - Inflate Balloon with 10cc of Sterile Liquid
  - Connect To Drainage System + Make sure bag is below the level of the bladder.
    Gently Pull Catheter back until Balloon is snug against bladder neck.

- **Com plications:**
  - Tissue Trauma
  - Infection
  - Renal Inflammation
    Bacteriuria
  - Pyelonephritis
- **Suprapubic Catheters:**
  - If trans-urethral catheterization isn't possible.
  - Involves piercing the bladder (via the peritoneal cavity) with a syringe.

URINE ANALYSIS:

- **Purpose:**
  - To screen for diseases/pregnancy
  - To monitor treatment
  - To assess patient progress
- **Abnormal Urinary Constituents:**

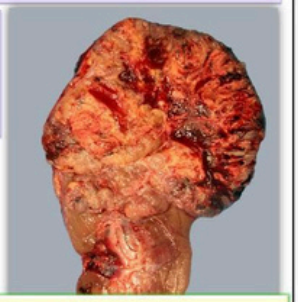
Substance	Condition	Possible Causes
Glucose	Glycosuria	Non-Pathological: Excessive intake of sugar. Pathological: Diabetes
Proteins	Proteinuria	Non-Pathological: Excessive physical exertion, pregnancy, high-protein diet. Pathological: Heart failure, severe hypertension, glomerulonephritis
Ketone bodies	Ketonuria	Excessive formation and accumulation of ketone bodies – starvation and untreated diabetes mellitus.
Hemoglobin	Hemoglobinuria	Transfusion reaction, hemolytic anemia, severe burns.
Bile pigments	Bilirubinuria	Liver disease (Hepatitis, cirrhosis), obstruction of bile ducts from liver to gallbladder
Erythrocytes	Haematuria	Bleeding in urinary tract – trauma, kidney stones, infection, neoplasm
Leukocytes (pus)	Pyuria	Urinary tract infection

MCQS - URINARY TRACT DISEASE

(Note: Green circle = correct answer)

A 59y woman presents with sudden onset of palpitations, flank pain and hematuria. ECG showed short Q-Tc interval, Hypercalcemia & Hypophosphotemia. Image shows her kidney removed at surgery. What is the most likely diagnosis ?

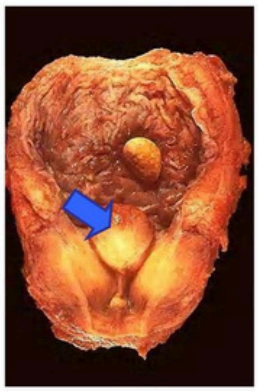
A. Adrenal gland carcinoma.
B. Metastatic medullary carcinoma.
C. Renal cell carcinoma.
D. Endstage kidney disease.
E. Renal papillary necrosis.



1. List all possible explanations for her Hypercalcemia?
2. Briefly discuss etiology and clinical features of this disease?
3. What other paraneoplastic syndromes are commonly seen in this disorder?

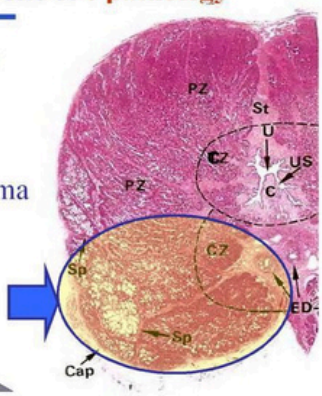
BPH: what feature is shown?

A. Bladder Wall Thickening
B. trabeculation
C. Stone formation
D. Ball valve obstruction
E. Enlarged lateral lobes



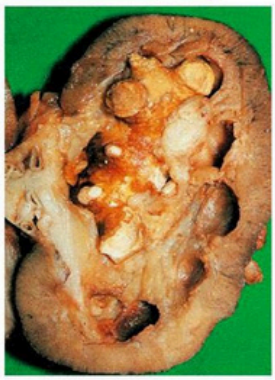
Prostate: Most likely site of ? pathology

A. Benign Hyperplasia.
B. Prostatitis
C. Stone formation
D. Adenocarcinoma
E. Transitional carcinoma



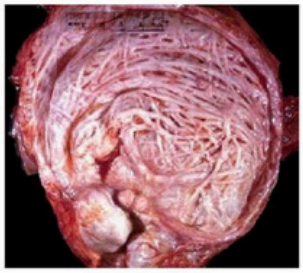
Kidney: What type of stone?

A. Oxalate & calcium
B. Calcium phosphate
C. Pure Uric acid
D. Triple phosphate
E. Cystine



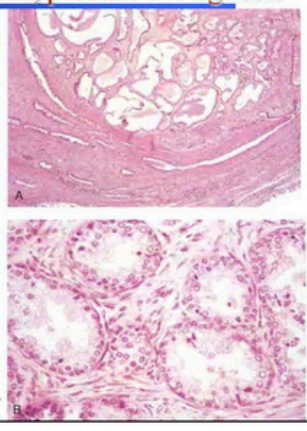
62y male chronic urinary retention. ? Diagnosis

1. Prostatic carcinoma
2. Benign P. Hyperplasia
3. Bladder carcinoma
4. Trabeculations
5. Bladder hypertrophy



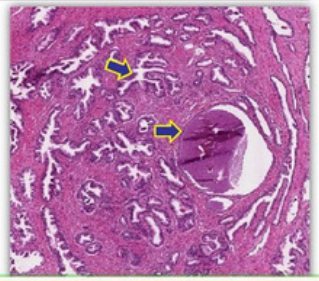
74y M, dysuria, hematuria, prostate ? Diagnosis

A. Prostatitis
B. Benign Prostatic Hyperpl.
C. Low grade carcinoma
D. Transitional carcinoma
E. High grade Carcinoma.



A 68y man investigated for nocturia, is fund on examination to have a slightly enlarged prostate and a PSA level of 7ng/ml (<4ng/ml). Image shows biopsy appearance of his prostate. What is the most likely diagnosis ?

A. Benign prostatic hyperplasia.
B. Mumps prostatitis.
C. Prostatic Cancer grade 4.
D. Metastatic adenocarcinoma.
E. Prostatic cancer.



1. Briefly discuss etiology and pathogenesis? (hormone, DHT, hyperplasia)
2. Explain his PSA level? (<4 normal, 4-10 BPH/prostatis.)
3. List complications? (*Obstruction, UTI, cystitis, stones, hydronephrosis/ureter*)
4. What is the prognosis? (*debilitating, not cancer*)

70y backpain, DRE-rock-hard, enlarged prostate. X-rays show multicentric, osteoblastic lesions of the lumbar vertebral bodies. An orchiectomy is performed. What is the rationale for this surgical procedure?

1. Leydig cells release androgenic factors.
2. Prostate carcinomas frequently metastasize to the gonads.
3. Sertoli cells release DHT.
4. The tumor is well known to invade the testes.
5. Tumor cells exhibit androgen-dependent growth.

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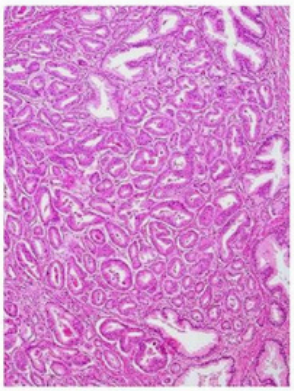
74y male, dysuria, hematuria, prostate ? Diagnosis

A. Prostatitis
B. BPH
C. Adenocarcinoma
D. Transitional carcinoma
E. BPH with carcinoma



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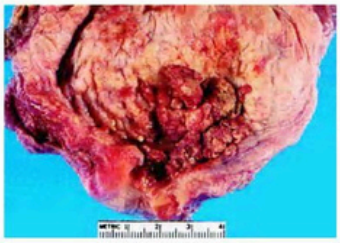
68y male, painless hematuria 4wk. Bladder image. What is the most likely risk factor?

1. Bladder calculi
2. Chronic HPV infection
3. Diabetes mellitus
4. Exposure to Azo dyes
5. Previous Prostatic ca.



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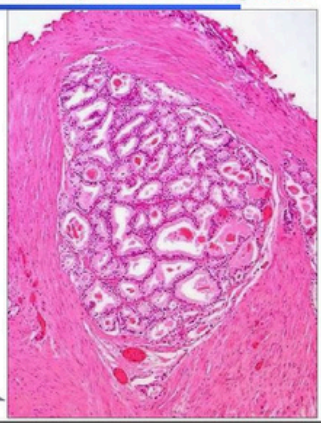
74y male, dysuria, hematuria, prostate ? Diagnosis

A. Prostatitis
B. BPH
C. Adenocarcinoma
D. Transitional carcinoma
E. BPH with carcinoma



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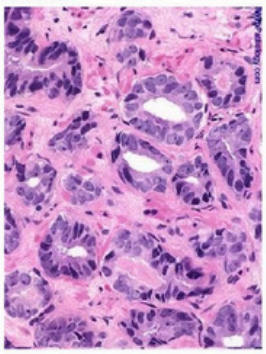
68y male, Image shows prostate biopsy. What is the most likely complication of this lesion?

1. Destructive vertebral lesions.
2. Bladder hypertrophy.
3. Calcium oxalate nephrolithiasis.
4. Gram negative septicaemia.
5. Lead to Prostatic carcinoma



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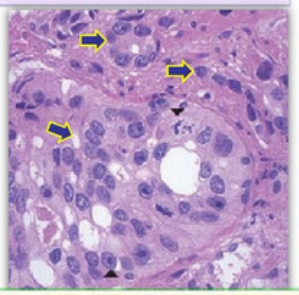
A 79y man with 1.5cm prostatic nodule found on DRE confined to prostate. Has a serum PSA of 13ng/ml (Ref. <4 ng/ml) Image shows biopsy specimen appearnce. What is the most likely diagnosis ?

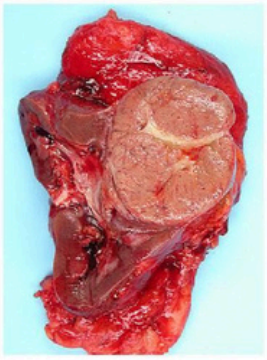
A. Benign prostatic hyperplasia.
B. Mumps prostatitis.
C. Prostatic Cancer grade 4.
D. Metastatic adenocarcinoma.
E. Prostatic cancer.



1. Briefly discuss etiology and pathogenesis?
2. What genetic abnormality common? (*Hypermethylation of GSTP1*)
3. What is the stage of disease? (*Stage-2, confined to prostate*)
4. What is the prognosis? (*good pronosis*)

61y Male, smoker, hematuria, loin pain, polycythemia. Kidney specimen. ? Most likely diagnosis

A. Transitional cell carcinoma.
B. Cortical adenoma
C. Renal cell carcinoma
D. Papillary carcinoma.
E. Wilm's tumor (Nephroblastoma)

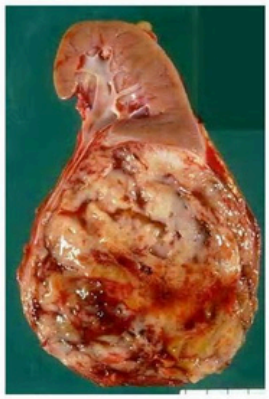


68y man elevated serum PSA (>6 ng/mL). Biopsy of the prostate reveals a poorly differentiated adenocarcinoma. Which of the following best describes the putative precursor of this neoplasm?

1. Basal cell hyperplasia
2. Chronic prostatitis
3. Obstructive uropathy
4. Nodular BPH
5. PIN.

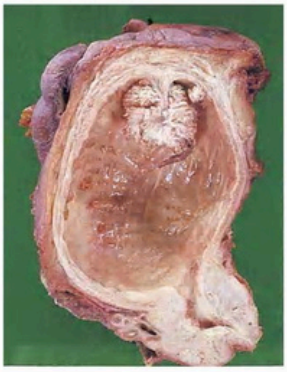
6y Male, Abdominal mass, weight loss Kidney ? diagnosis

A. Transitional cell carcinoma.
B. Cortical adenoma
C. Renal cell carcinoma
D. Metastatic to kidney.
E. Nephroblastoma



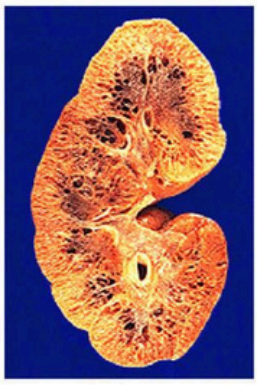
Urinary bladder tumor ? CORRECT STATEMENT

1. Benign papilloma
2. Polyposis of bladder
3. Patient has good prognosis
4. Papillary carcinoma
5. Adenocarcinoma



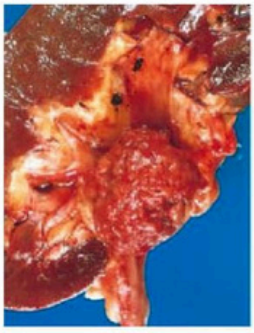
3m female renal & liver failure. Post mortem Kidney specimen ? Most likely diagnosis

A. Cystic Nephroblastoma.
B. Hereditary ARPKD
C. Hereditary ADPKD .
D. Uremic Medullary cystic D.
E. Cystic Renal Dysplasia



61y Male, smoker, hematuria, Kidney ? Diagnosis

A. Transitional cell carcinoma.
B. Cortical adenoma
C. Renal cell carcinoma
D. Metastatic to kidney.
E. Nephroblastoma



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64y man, 4m dysuria and hematuria for 4days. P/H of repeated bouts of acute cystitis. Urine cultures are positive for E. coli. A stone is found in the bladder diverticulum. Most likely predisposing condition ?

A. Diabetes mellitus
B. Malakoplakia
C. Nodular prostatic hyperplasia
D. Nephrolithiasis
E. Transitional cell carcinoma

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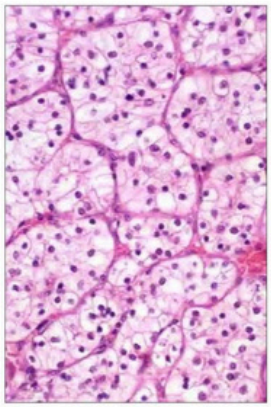
A. B. C. D. E.

56y Male, smoker, hematuria, loin pain. Kidney ? diagnosis

A. Transitional cell carcinoma.
B. Cortical adenoma
C. Clear cell carcinoma
D. Adenocarcinoma.
E. Papillary carcinoma



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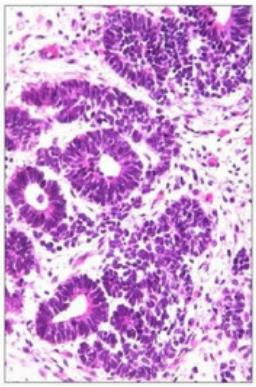
5y Male, Abdominal mass, hematuria, Kidney biopsy ? diagnosis

A. Transitional cell carcinoma.
B. Wilm's tumor
C. Clear cell carcinoma
D. Adenocarcinoma.
E. Papillary carcinoma



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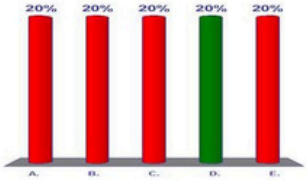
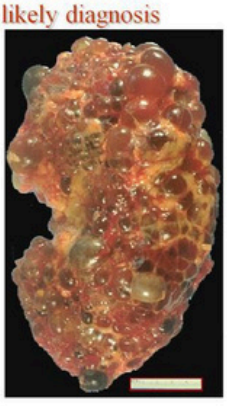
51y, Hypertension, Chronic renal failure on renal dialysis. Kidney specimen ? Most likely diagnosis

A. Chronic Glom. Nephritis.
B. AR-PKD
C. End stage Kidney dis.
D. AD-PKD - Adult
E. Dialysis induced cysts.





61y flank pain, Hematuria, Most likely diagnosis?

1. Acute glomerulonephritis.
2. Nephroblastoma
3. Renal Cell adenoma
4. Transitional cell carcinoma.
5. Clear cell carcinoma

Hct	57%
Hb	19 g/dL
BUN	12 mg/dL
Creat.	0.7 mg/dL
WBC	7,450/mm^3 normal differential
RBC cytology	3+, no casts. No Malignant cells.

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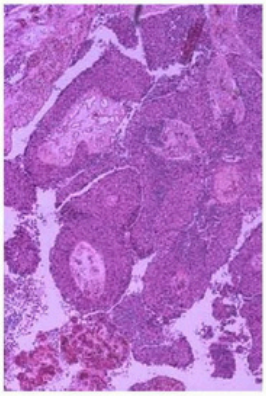
62y M, hematuria. 3cm growth in renal pelvis – Biopsy image ? diagnosis

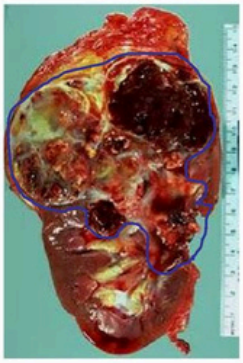
A. Papillary RCC
B. Nephroblastoma
C. Pyelonephritis
D. Transitional Cell Ca
E. Buergers Disesase



56y asymptomatic hematuria

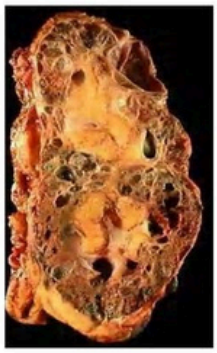
Get Direction GLOBAL

1. ADPKD with hemorrhage.
2. ARPKD
3. Renal cell carcinoma
4. Nephroblastoma
5. Transitional cell carcinoma



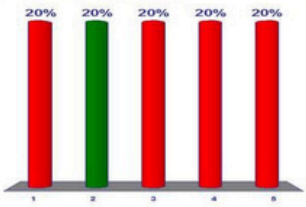
62y M, chronic renal failure on dialysis. Shrunken small kidneys. image ? diagnosis

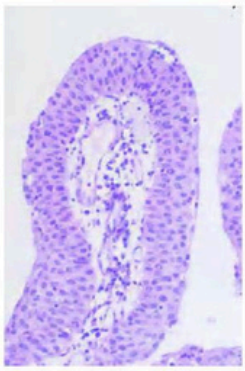
A. ADPKD
B. Endstage kidney disease.
C. Chronic Pyelonephritis
D. Uremic medullary cystic disease.
E. Dialysis associated cysts.



72y female, asymptomatic hematuria. 5 cm papillary tumor in bladder. Image shows biopsy. ?Diagnosis

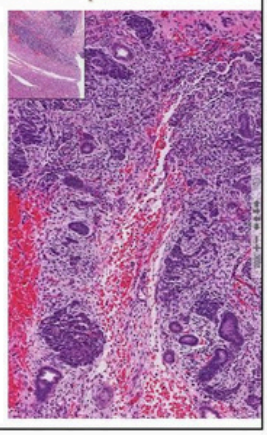
1. Adenocarcinoma
2. Transitional cell carcinoma.
3. Papilloma
4. Tubular adenoma
5. Villous adenoma





2y M, hematuria, hypertension. 6cm growth in lower pole of left kidney. Biopsy image ? diagnosis

A. Papillary RCC
B. Chronic glomerulonephritis
C. Benign Nephrosclerosis
D. Nephroblastoma
E. Acute glomerulonephritis

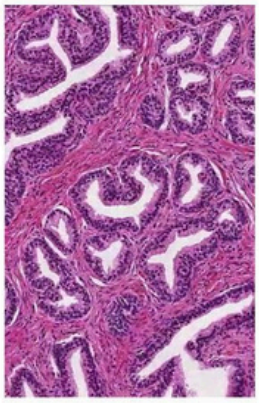


55y man, urinary urgency and frequency. DRE enlarged prostate. PSA of 4.9 (normal = 0–4). Needle biopsy - two cancer-positive needle cores: Gleason grades 4 and 5. Which of the following is the appropriate diagnosis?

1. **Adenocarcinoma**
2. **Nodular BPH**
3. **PIN-3**
4. **Squamous Carcinoma**
5. **Transitional Carcinoma**

78y male, Image shows prostate biopsy. What is the most likely complication?

1. Destructive vertebral lesions.
2. Bladder hypertrophy.
3. Calcium oxalate nephrolithiasis.
4. Gram negative septicemia.
5. Infertility.



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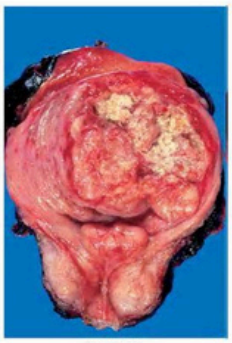
CPC-4.3– KFP Questions:

Get Direction GLOBAL

- **BPH – etiology, Pathogenesis, morphology & complications.**
- **Testosterone, DHT, Fenosteride.**
- **TURP – brief notes.**
- **Prostatic carcinoma – etiology, Pathogenesis, morphology & spread, metastases.**
- **Staging, Grading & Prognosis.**
- **Urolithiasis : Renal stones**
- **Other obstructive uropathy.**

68y male, Image shows Bladder & prostate. What complication is not shown?

1. Invasive bladder cancer.
2. BPH.
3. Ball valve obstruction.
4. Bladder diverticula.
5. Tumor necrosis & hemorrhage.



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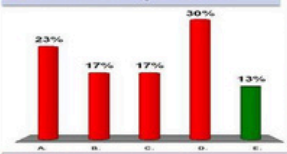
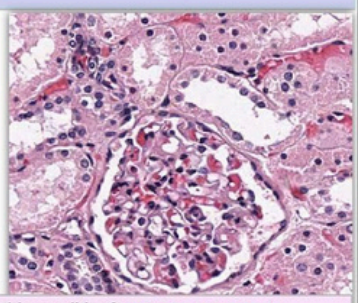
6 year girl presents with facial swelling, polyuria, polydipsia and massive proteinuria following recovery from upper respiratory tract viral infection. Image shows kidney biopsy appearance of her glomerulus. What is the most likely diagnosis?

A. Analgesic nephropathy.
B. Acute renal failure.
C. Membranous GN.
D. Proliferative GN.
E. Minimal change disease.

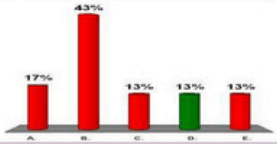


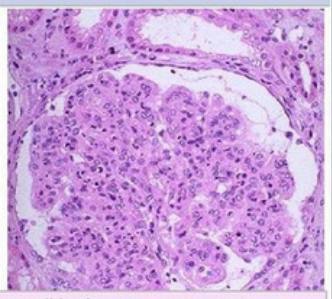


A. Briefly discuss pathogenesis of this condition? (podocyte foot process)
B. List gross and Microscopic feature of this disease? (normal)
C. Other features, Prognosis? (hyperlipidemia, lipiduria, hypoalbuminemia), good.

14 days following an episode of fever, pharyngitis treated with analgesics, a 16y boy presents with severe tiredness, fever. He is passing very little dark brown urine. Urine analysis shows non selective proteinuria, hematuria with many RBC casts. Image shows his renal biopsy appearance. What is the most likely diagnosis?

A. Analgesic nephropathy.
B. Acute streptococcal nephritis.
C. Membranous GN.
D. Diffuse proliferative GN.
E. Minimal change disease.

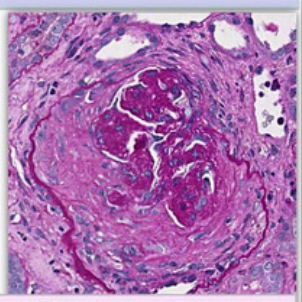




A. Briefly discuss pathogenesis of this condition? (autoimmune, IgG, BM)
B. List gross and Microscopic feature of this disease? (Inflam)
C. List other clinical features, prognosis? Hypertension, recover/CGN

43y man with 3 week history of hemoptysis, hematuria and fever. Past history revealed features of mixed connective tissue disorder treated with steroids in the past year. Labs show rapidly increasing urea & creatinine levels since 1 week. Image from renal biopsy shows appearance of one glomerulus. What is the most likely diagnosis?

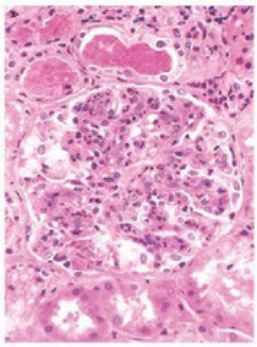
A. Global sclerosis.
B. Crescentic GN.
C. Membranous GN.
D. Diffuse proliferative GN.
E. Chronic GN.



A. Briefly discuss pathogenesis of this condition? (Goodpasteur sy)
B. List gross and Microscopic feature of this disease? (Inflam exudate)
C. List other clinical features, prognosis? Renal failure.

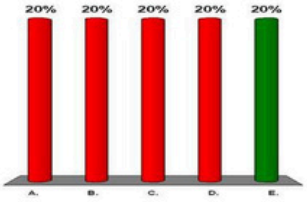
12y Fem, puffy face, Oliguria, smoky urine, hypertension. Recovering from URTI. Kidney biopsy ? Most likely diagnosis

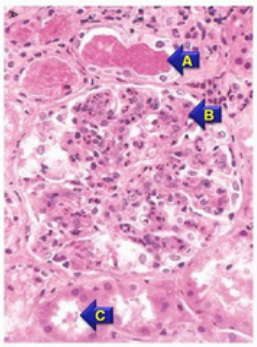
A. Diffuse proliferative GN
B. Membranous GN
C. Minimal change GN
D. Rapidly progressive GN
E. Membranoproliferative GN



12y Fem, puffy face, Oliguria, smoky urine, hypertension. Recovering from URTI. Kidney biopsy ? Feature "A"

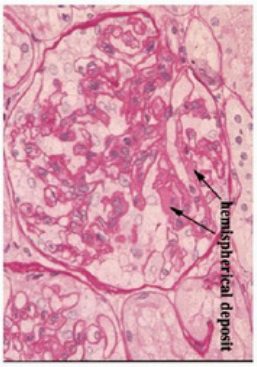
A. Proliferative GN
B. Proliferative GN (Neutrophils)
C. Diffuse glomerulosclerosis
D. WBC cast in tubule.
E. RBC cast in tubule.





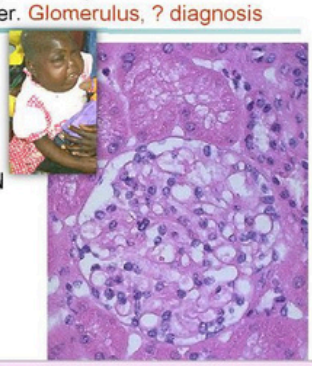
21y Male, hematuria, recovering from an URT infection. Had similar attack twice in last two years ? diagnosis

A. Diffuse proliferative GN
B. Membranous GN
C. Nodular Glomerulo sclerosis
D. Minimal change GN
E. Berger's Disease



2y girl, Severe albuminuria, facial & pedal edema. Recovering from a viral fever. Glomerulus, ? diagnosis

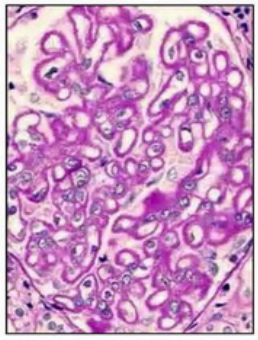
A. Diffuse proliferative GN
B. Membranous GN
C. Minimal change GN
D. Rapidly progressive GN
E. Membranoproliferative GN



A. Briefly discuss pathogenesis of this condition? (podocyte foot process)
B. List gross and Microscopic feature of this disease? (normal)
C. Other features, Prognosis? (hyperlipidemia, lipiduria, hypoalbuminemia), good.

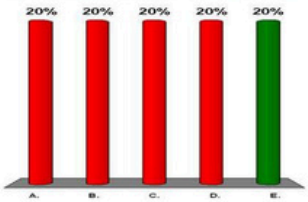
48y Male, proteinuria, lipiduria, pedal edema. On treatment for SLE arthritis. Kidney biopsy PAS stain ? diagnosis

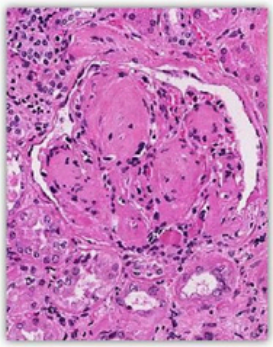
A. Diffuse proliferative GN
B. Membranous GN
C. Minimal change GN
D. Rapidly progressive GN
E. Membranoproliferative GN



54y Male, past MI, DM2, nocturia, polyuria, recurrent leg ulcers. Kidney biopsy ? diagnosis

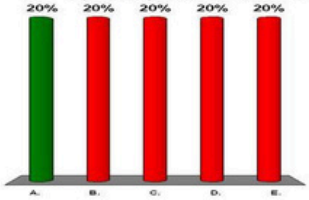
A. Diffuse proliferative GN
B. Membranous GN
C. Minimal change GN
D. Rapidly progressive GN
E. Nodular Glomerulosclerosis

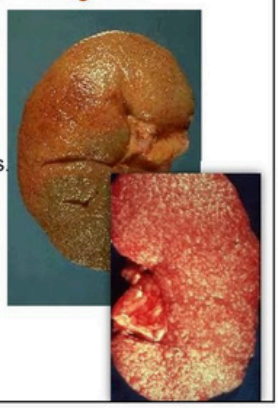




58y Male, Chronic hypertension. Slowly progressive renal failure since 2y. Kidney sp. ? diagnosis

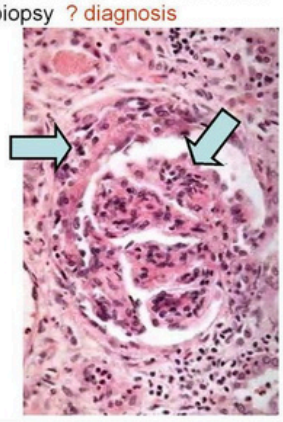
A. Benign nephrosclerosis
B. Papillary necrosis
C. Pyelonephritis with infarction
D. Nodular glomerulosclerosis
E. Pyelonephritis with abscesses





14y Male, severe acute renal failure, history of recent throat infection on treatment. Kidney biopsy ? diagnosis

A. Diffuse proliferative GN
B. Membranous GN
C. Minimal change GN
D. Rapidly progressive GN
E. Membranoproliferative GN

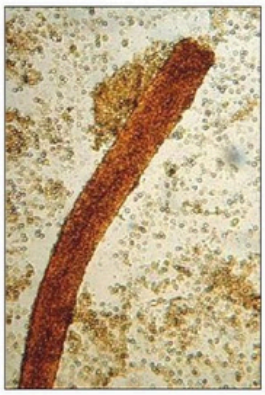


74y Male, Hypertensive, Oliguria & marked fatigue since 2y. Left Kidney gross ? diagnosis

A. Nodular Glomerulo sclerosis.
B. Chronic Pyelonephritis.
C. Polycystic kidney disease.
D. Rapidly progressive GN.
E. Chronic Glomerulonephritis.

46y Male, Hematuria. Urine cytology ? diagnosis

A. Urinary Tract Infection
B. Bladder cancer
C. Renal stones
D. Glomerulonephritis
E. Schistosomiasis



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46y Male, 3wk. lethargy. KFT ? diagnosis

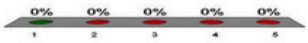
A. Nephritic Syndrome
B. Acute renal failure
C. Nephrotic syndrome
D. Chronic Renal failure
E. Renal cell carinoma.

- Selective proteinuria
- Hypoalbuminemia
- Hypercholesterolemia
- Serum creatinine normal

46y Male, 3wk. lethargy. KFT ? diagnosis

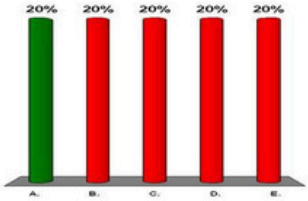
A. Nephritic Syndrome
B. Acute renal failure
C. Nephrotic syndrome
D. Chronic Renal failure
E. Rencal cell cancer.

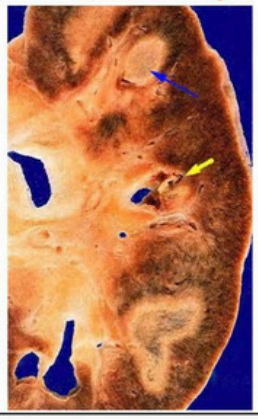
- Oliguria
- Hypertension
- Non Selective proteinuria
- Serum creatinine high
- RBC casts present.



46y Diabetic male. Fever, hematuria ? diag

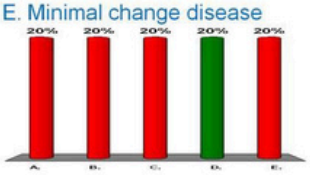
A. Papillary necrosis
B. Pyelonephritis & abscess.
C. Nodular glomerulosclerosis
D. Renal abscesses
E. Chronic Renal failure

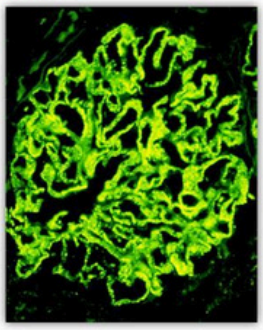




44y man, SOB, swelling of his legs and puffiness around his eyes & Ascitis. Total serum protein is 5.2 g/dL (reference = 5.5–8.0 g/dL), and albumin is 1.9 g/dL (reference = 3.5–5.5 g/dL). Serum cholesterol is elevated at 530 mg/dL. 5 g of protein in a 24-hour urine, with many granular casts but no RBCs or WBC. Image shows renal biopsy stained by direct immunofluorescence for IgG ? Diagnosis

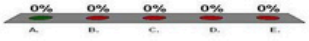
A. Proliferative GN
B. Focal Segmental GS.
C. Proliferative GN
D. Membranous GN
E. Minimal change disease

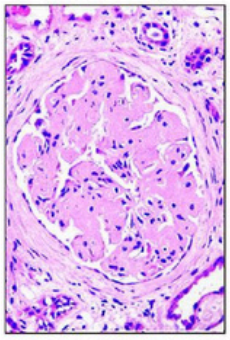




60y man, chronic back pain and fatigue, excessive urination, and increased thirst. X-ray - numerous lytic lesions in the lumbar vertebral bodies. Lab: hypoalbuminemia, 4+ proteinuria & A monoclonal Ig light-chain peak. A bone marrow biopsy 20% atypical plasma cells. Image shows kidney biopsy. ? Diagnosis

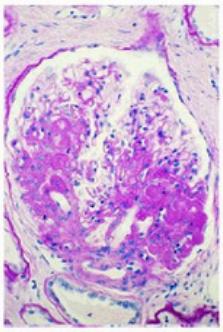
A. Amyloid nephropathy
B. Crescentic glomerulonephritis
C. IgA nephropathy (Berger disease)
D. Membranous glomerulonephritis
E. Nodular glomerulosclerosis.





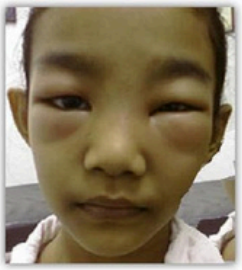
30y man with h/o drug addiction, 6/12 progressive edema & Ascitis, Marked proteinuria (>4 g/24 hours) but no WBC or RBCs in urine. Lab: Hyperlipidemia and hypoalbuminemia. Serum creatinine level is normal. The blood test for ANCA is negative. Recurrent attacks respond to corticosteroids, Upon the third recurrence, becomes steroid resistant. A renal biopsy is shown . ? Diagnosis

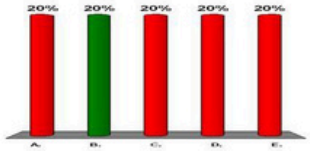
A. Acute glomerulonephritis
B. Amyloidosis
C. Crescentic glomerulonephritis
D. Diffuse proliferative glomerulonephritis
E. Focal segmental glomerulosclerosis



A 6-year-old boy complains of swelling of his face & feet for the last 3 weeks. He is otherwise healthy, with no known previous illness. Vital signs are normal. Physical examination reveals pitting edema of the lower legs and a swollen abdomen. Urinalysis shows 4+ protein but no RBCs or WBCs. ? Most likely Diagnosis.

A. Acute glomerulonephritis
B. Minimal change disease
C. Crescentic glomerulonephritis
D. Diffuse proliferative glomerulonephritis
E. Membranous Glomerulonephritis





9y boy, episode of hematureia 1wk after flulike illness. One month later his urine is red again. Urinealysis pH7, SG 1.015, Proteinuria 1+, 1+ hematuria. No ketones, glucose or urobilinogen. Serum urea & creat. Normal. Renal biopsy shows mesangial proliferation & Antibody complex deposition. Which of the following mechanisms is most likely to produce his symptoms?

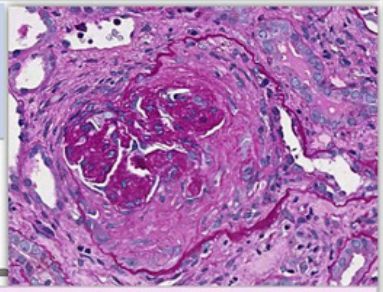
Explanation: Recurrent painless hematuria following a viral illness in a child or young adult is typically associated with IgA nephropathy (Berger's dis). Defective immune regulation causes excessive mucosal IgA synthesis in response to viral or other environmental antigens. IgA complexes are deposited in the mesangium and initiate glomerular injury. Antibodies against type IV collagen are seen in Goodpasture syndrome.

49y male, Ankle & Foot swelling for 2 months. 24h urine yielded 4.1g protein. No H/O DM, SLE or Hypertension. No response to steroid therapy. Renal biopsy showed diffusely thick capillary basement membrane with granular C3 deposition. Two years later he developed chronic renal failure. What is the most likely pathogenesis?

Explanation: This patient has idiopathic MGN & nephrotic syndrome. Diffuse basement membrane thickening caused by the deposition of immune complexes on the basement membrane, which activates complement. Antibodies that react with basement membrane give rise to a linear immunofluorescence pattern. Membranous glomerulopathy has no association with streptococcal infections. There is also no evidence of cytokine- or T-cell-mediated damage in this disease. In 85% of patients is unknown. In the remaining 15%, an associated systemic disease (e.g., SLE) or some known cause of immune complex formation (e.g., drug reaction, viral hepatitis) exists.

39y man with eight week history of cough, fever and skin rash is found to have nasopharyngeal ulcerations, nodular and vaitary lesions on chest x-ray. He develops rapidly progressive renal failure with hematuria and RBC casts in urine. A lung biopsy shows necrotizing vasculitis. Image shows appearance of affected glomerulus. What is the most likely diagnosis?

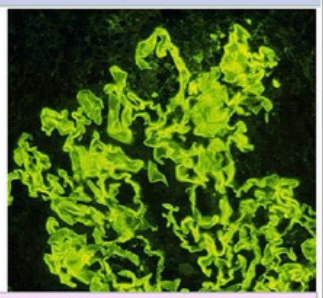
A. Goodpasteur syndrome.
B. Miliary tuberculosis.
C. Wegener's granulomatosis.
D. Endstage renal disease.
E. Berger's disease.



A. Briefly discuss pathogenesis of this condition? (Wegener's)
B. List 3 Microscopic features of this disease? (crescentic GN)
C. List etiology for this renal disorder (RPGN)?.

25y man presents with bout of hematuria, pedal edema and hypertension. On further questioning reveals recent attacks of coughing with blood streaked sputum. Urinalysis shows proteinuria and RBC casts. Image shows renal biopsy with Immunofluorescent stain for anti-IgG antibody. What is the most likely diagnosis?

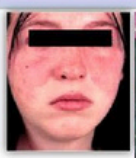
A. Post streptococcal GL.
B. HIV nephropathy.
C. Minimal chande disease.
D. Hep-B Inf. (membranous).
E. Goodpasture Syndrome.

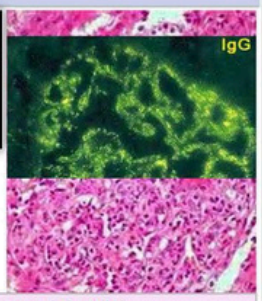


A. Pathogenesis? (*IgG to BM Collagen in lung & Kid → Acute Infl → RPGN*)
B. List gross and Microscopic feature of this disease? (*Inflam, RPGN,Linear*)
C. Clinical features? complications? prognosis? (*Nephritic, CGN, R.Failure*)

31y fem presents with fever , fatigue for 3 months on & off. She has 3kg wt loss. On PE malar rash, pain on deep inspiration, friction rub. Labs showed increased globulins, serum creatinine, decreased serum complements. Renal biopsy reported granular IgG deposits in the basement membrane. What is the most likely diagnosis?

A. SLE (lupus nephritis).
B. Post streptococcal GN.
C. Minimal change disease.
D. Membranous GN.
E. Goodpasture Syndrome.

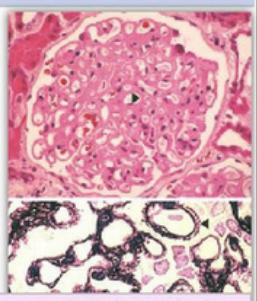




A. Pathogenesis? (*Genetic DR3 → Env+Hormones → AutoAb*)
B. List gross and Microscopic feature of this disease? (*Inflam, DPGN, Gran.*)
C. Clinical features? complications? prognosis? (*Nephritic, CGN, R.Failure*)

47year man, over the counter use of ibuprofen daily since [illegible] months following a road traffic accident. Presents with edema & hypertension. Urinalysis showed BA, anuria but no hematuria or glucosuria. Kidney biopsy (Image) view of his glomerulus is shown in the image with silver stain (bottom). What is the most likely diagnosis?

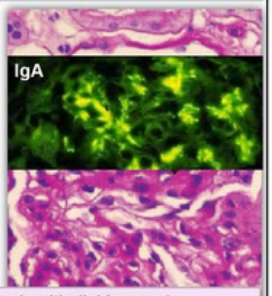
A. SLE (lupus nephritis).
B. Post streptococcal GN.
C. Minimal change disease.
D. Membranous GN.
E. Goodpasture Syndrome.



A. Pathogenesis? (*Drug+Ab complex deposited as subepithelial humps)*
B. List gross and Microscopic feature of this disease? (*Inflam, MGN, Gran.*)
C. What other drugs & Diseases? (*Penicillamine, captopril, Ca, HBV, HCV*)

21year man, a week following mild URI presents with profound weakness, very little dark urine. O/E hypertension, urinalysis showed hematuria with dysmorphic RBC. He recovers within a week, but develops four similar recurrences in the next year. Image shows his renal biopsy specimen. What is the most likely diagnosis?

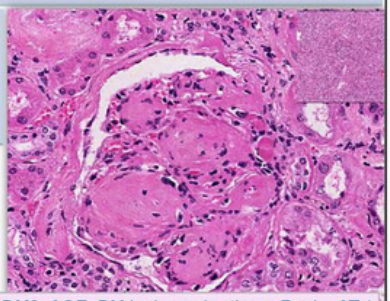
A. Membranous GN.
B. Post streptococcal GN.
C. Berger's disease
D. Minimal change disease.
E. Goodpasture Syndrome.



A. Pathogenesis? (*IgA complex deposited as subepithelial humps)*
B. Microscopic feature of this disease? (*Inflam, Mes-IgA*) Prognosis?
C. Other Disease association? (Pri/Sec, *Henoch Schonlein purpura, Celiac*)

68y man, BMI 41, Peripheral neuropathy, retinopathy and abdominal aortic aneurysm on therapy shows increasing serum creatinine. FBS 12.8 mol, Image shows his renal biopsy. What is the most likely diagnosis?

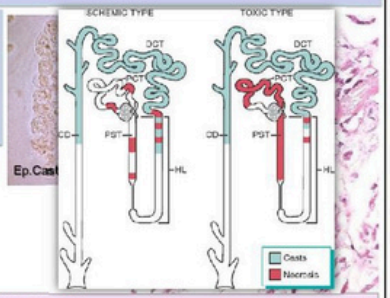
A. Membranous GN.
B. Goodpasture Syndrome.
C. Post streptococcal GN.
D. Berger's disease
E. Nodular glomerulosclerosis



A. Diagnosis? Pathogenesis? (*DM2, AGE, BM leak, nephrotic sy, Renin, AT..)*
B. Microscopic feature of this disease? (*NGS, artereolosclerosis, CGN)*
C. What other complications? (*Atherosclerotic PAD, stroke, MI, etc…)*

19y old boy, Post operative marked oliguria, nausea, malaise following spleenectomy for ruptured spleen following car crash. He was found in shock at the site of crash Immediate lapratomy revealed massive hemoperitoneum. (bladder not distended). Labs anemia, Increased BUN, Creatinine, U:C ratio 10:20. What is the diagnosis?

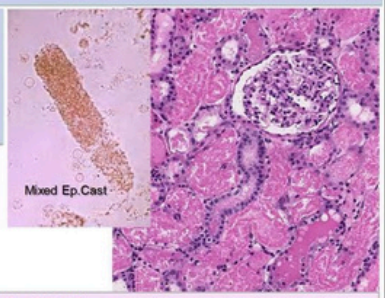
A. Toxic ATN.
B. Papillary necrosis.
C. Rapidly Progressive GN.
D. Berger's disease.
E. Ischemic ATN.



A. Pathogenesis? (*Ischemic ATN → ARF)*
B. Microscopic feature of this disease? (*Necrosis of tubules, more in PCT)*
C. Types of ATN/ARF, list differences? (*Toxic, Ischemic*)

32y woman, chronic headache, relieved by simple over the counter analgesics. Presents with high colored urine with brown pieces of tissue in her urine today. HPE reveals progressively increasing polyuria, anemia, diarrhoea and hypertension since 6 months. Image shows her urine sediment and renal biopsy. What is the likely diagnosis?

A. Toxic ATN.
B. Papillary necrosis.
C. Rapidly Progressive GN.
D. Berger's disease.
E. Ischemic ATN.



A. Pathogenesis? (*Ischemic ATN → ARF)*
B. *Drug induced interstitial nephritis / analgesic nephropathy.*
C. Briefly discuss prognosis? (*ARF, CRF, TCC*)