

The Renal System & Acute Kidney Injury

AKI CLINICAL NURSE SPECIALIST TEAM

ROYAL WOLVERHAMPTON NHS TRUST

Aims of the session

- Review basic renal anatomy and physiology
- Look at classification of AKI
- Risk factors for AKI
- Causes and consequences of AKI
- Management of AKI

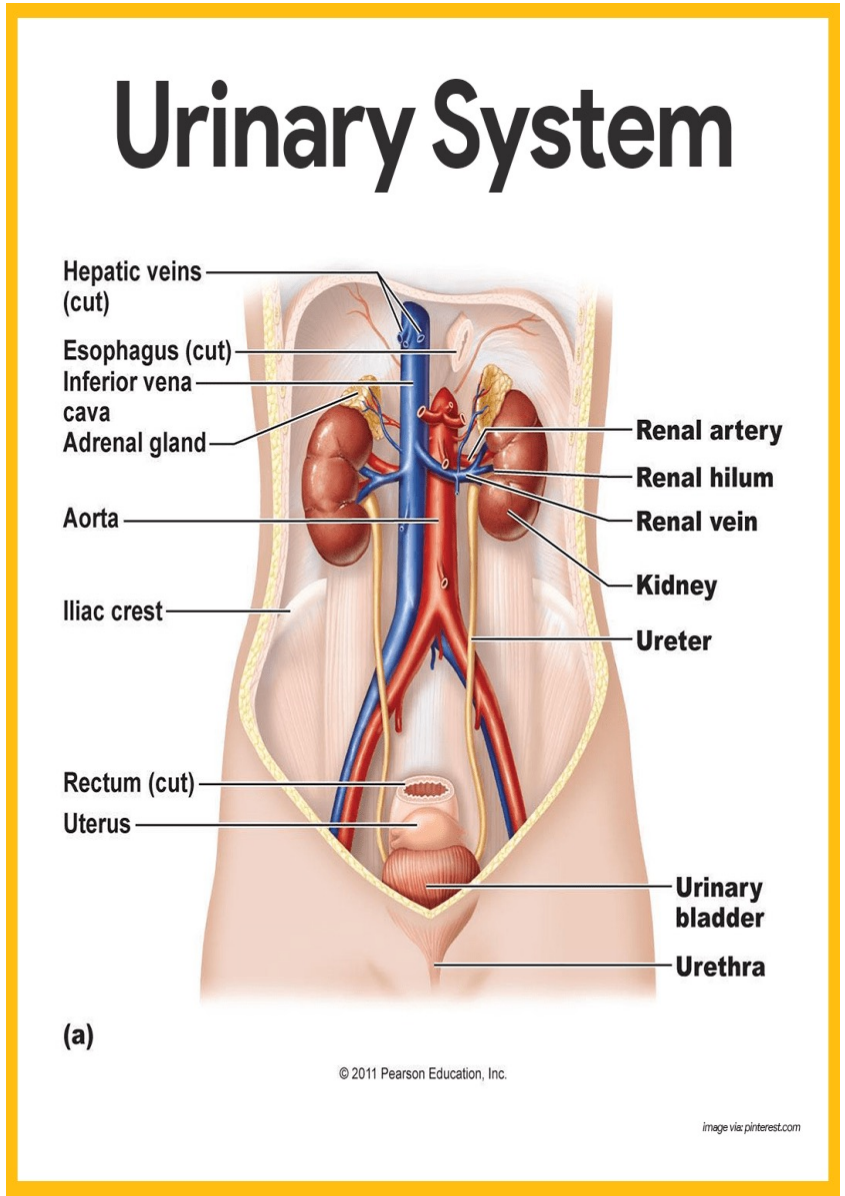
The Kidneys

Most people have two kidneys

They sit between the 12th thoracic and 3rd lumbar verebrae

Blood originates from the abdominal aorta and then into the renal artery

Blood travels away from the kidney via the renal vein & then the inferior vena cava



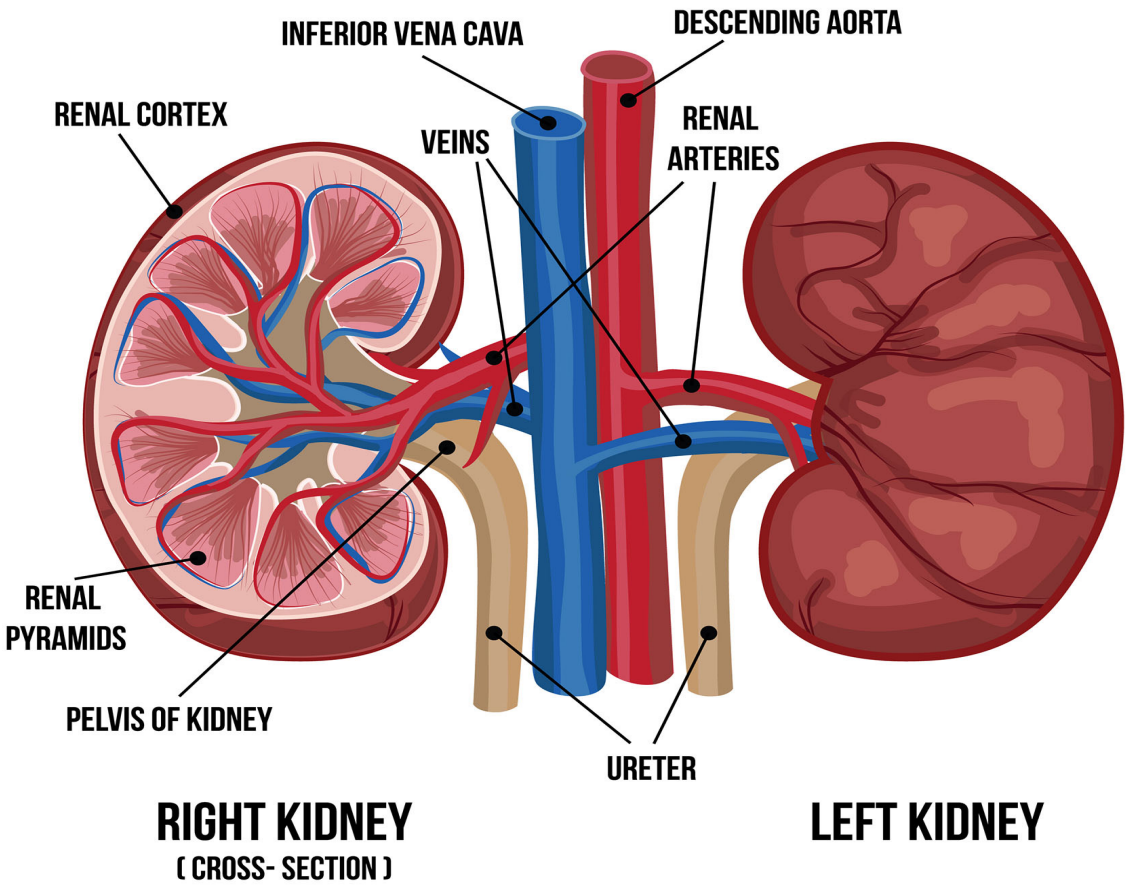
They filter your blood every minute of the day

Blood goes through your kidneys 40 times a day

They use 25% of cardiac output with each heartbeat

Kidneys make urine – about 2L per day

Structure of the kidney



Functions of the kidney



Removes waste products from the body, such as urea & creatinine



Removes toxins from the body



Helps maintain acid-base balance



Regulates electrolyte (salt) concentrations



Balances the body's fluids (ADH)



Regulates blood pressure (RAAS)

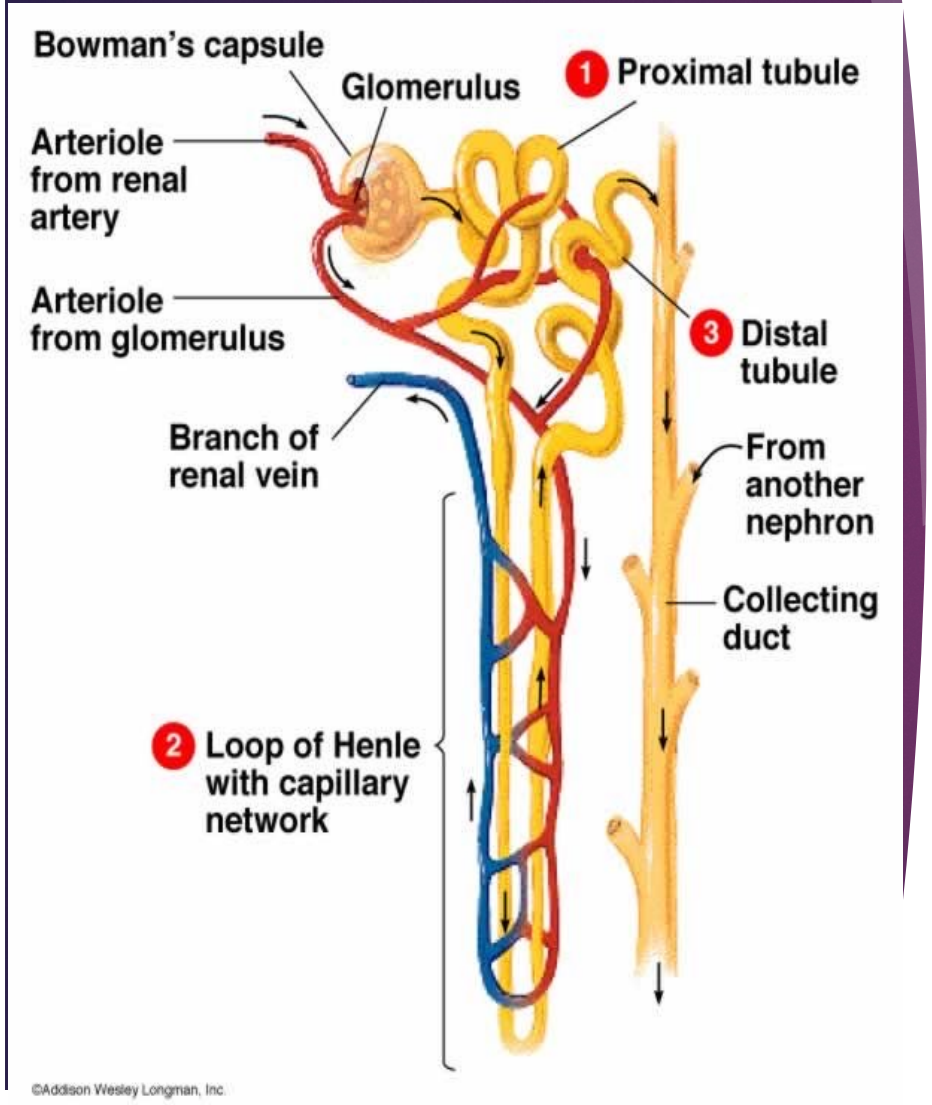


Activates vitamin D which promotes strong, healthy bones



Controls the production of red blood cells

The Nephron

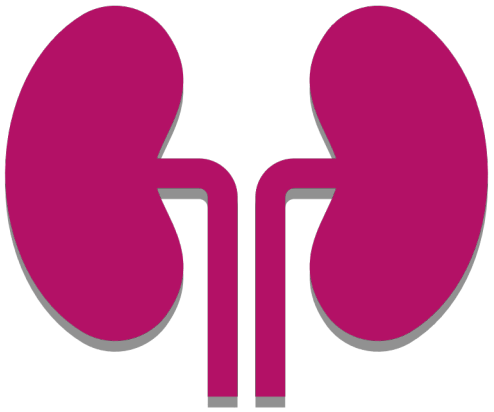


- Nephrons are the functioning part of the kidney, with approximately one million in each kidney

Three stages of urine formation:

- 1) **Filtration** – glomerulus
- 2) **Selective reabsorption** – tubules and loop
- 3) **Excretion** of waste products into collecting ducts.

Acute Kidney Injury

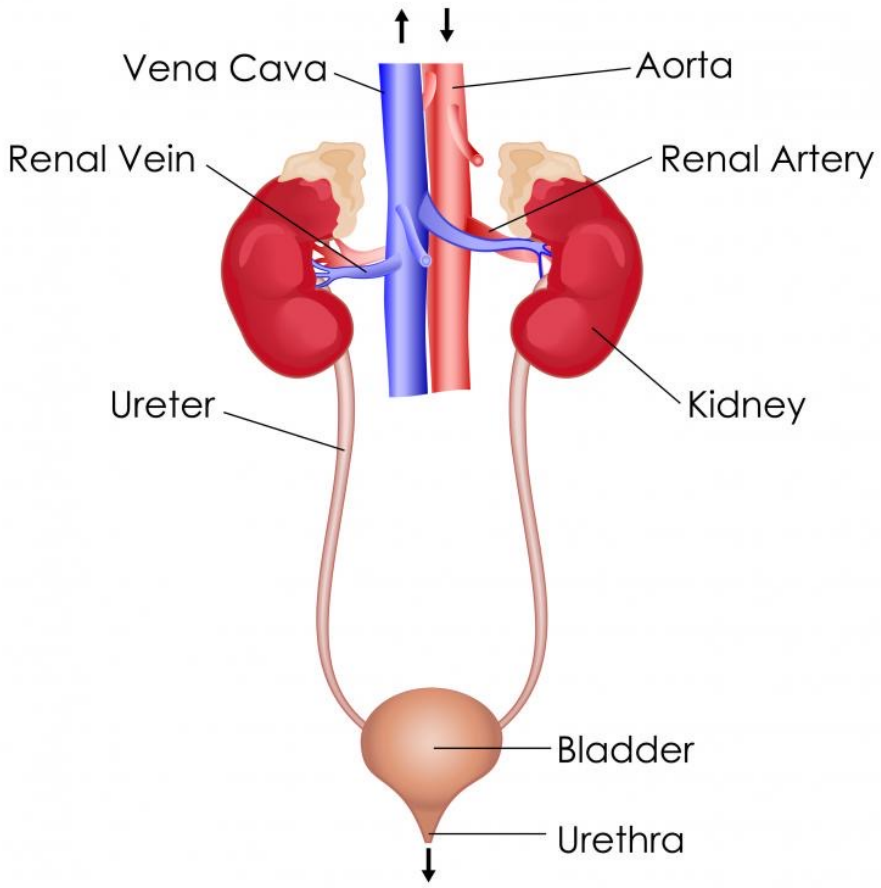


- **Acute Kidney Injury (AKI)** is the acute deterioration in kidney function that presents over a period of hours or days.
- Renal function will continue to deteriorate unless AKI is recognised and its cause identified and treated. Can be life-threatening.
- In the UK, up to 100,000 deaths each year in hospital are associated with AKI. Up to 30% could be prevented with the right care and treatment.
- It is estimated that one in five people with admitted to hospital each year as an emergency has AKI.
- Financial burden to the NHS estimated to cost > £1 billion pa (1% of the NHS budget)

AKI Staging – KDIGO

AKI stage	Serum Creatinine criteria	Urine output criteria
1	SCr increase ≥26 µmol/L within 48 hrs or SCr increase ≥1.5–2 fold from baseline	<0.5 mL/kg/hr for 6 consecutive hrs
2	SCr increase ≥2–3 fold from baseline	<0.5 mL/kg/hr for 12 hrs
3	SCr increase ≥3 fold from baseline or SCr increase ≥354 µmol/L or initiated on RRT (irrespective of stage at time of initiation)	<0.3 mL/kg/hr for 24 hr or anuria for 12 hr

To function, kidneys need….



Adequate blood pressure/blood volume

(*pre-renal – 70%*)

To be healthy, rather than damaged or inflamed by disease process

(*intrinsic – 20%*)

To drain urine freely through a functioning urinary tract

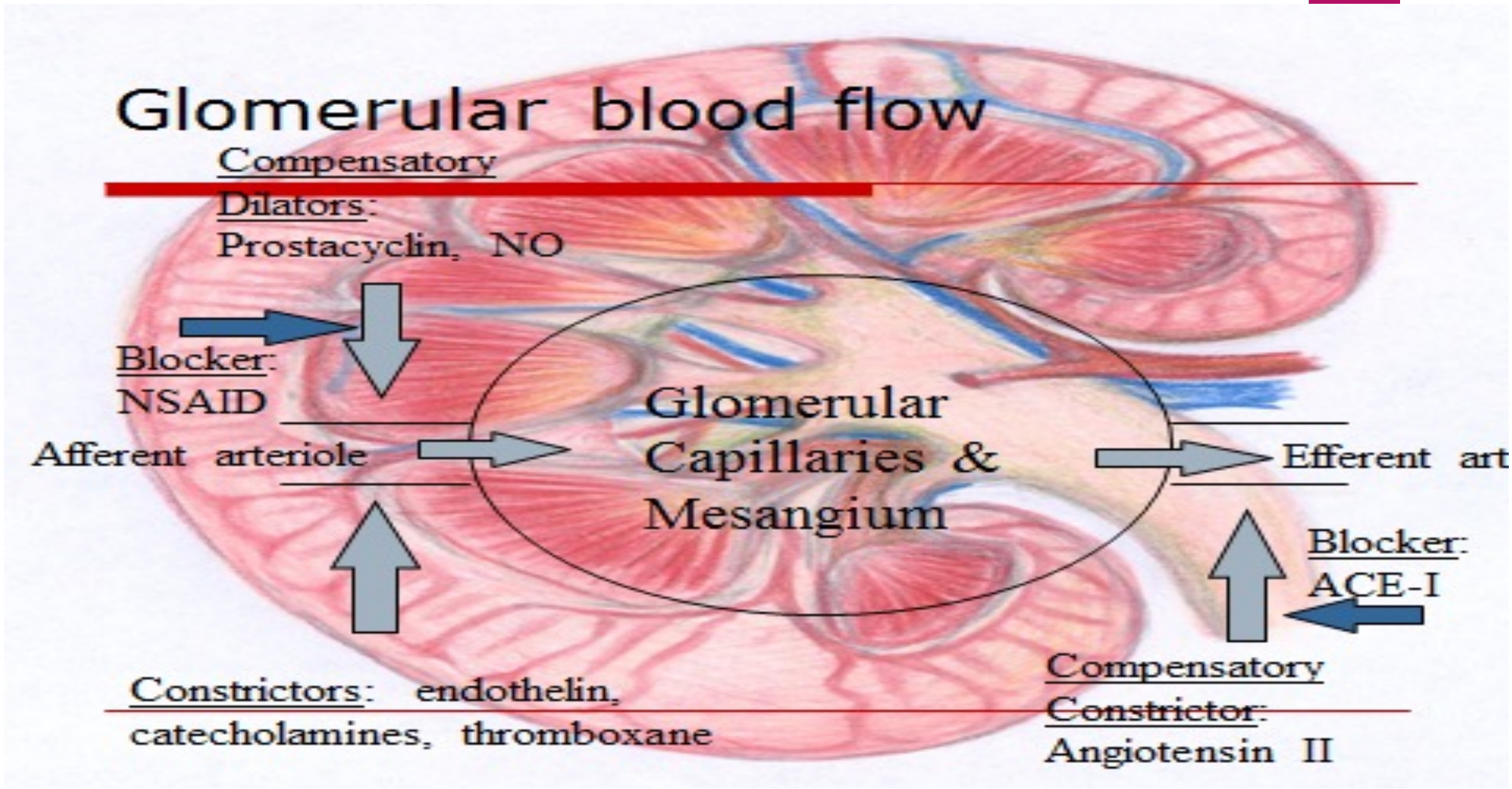
(*post-renal 5-10%*)

Risk Factors for AKI

Chronic Kidney Disease	Diabetes	Heart failure	Liver failure	Ileostomy
Age	Cognitive/ motor issues	Nephrotoxic medications	Enlarged prostate/pelvic cancers/stones	History of AKI

Pathophysiology of pre-renal AKI

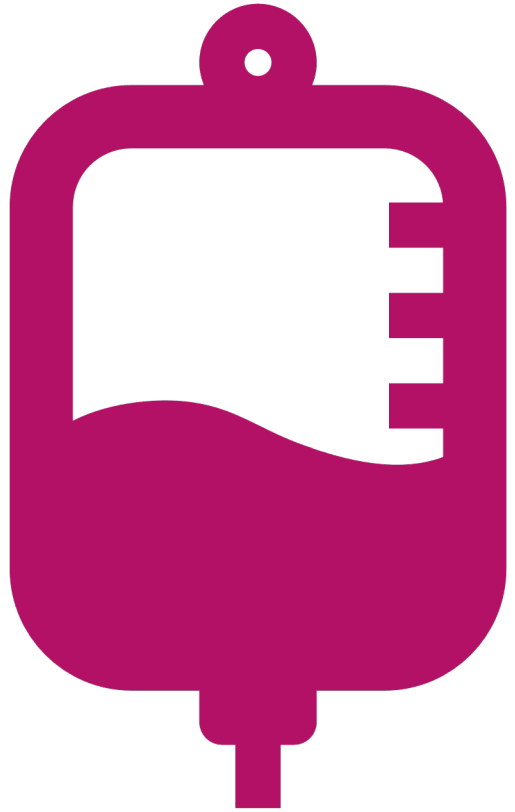
- Hypovolaemia/low cardiac output/vasodilation
- Baroreceptor activation
- Neurohormonal responses
- RAS/Vasopressin/sympathetic activation
- Prostaglandin synthesis/angiothensin 2/salt and water reabsorption/thirst
- **Goal – restore intravascular volume and BP and renal perfusion**


Glomerular blood flow
Compensatory
Dilators:
Prostacyclin, NO
Blocker:
NSAID
Afferent arteriole
Glomerular
Capillaries &
Mesangium
Efferent art
Blocker:
ACE-I
Compensatory
Constrictor:
Angiotensin II
Constrictors: endothelin,
catecholamines, thromboxane

Pre-renal causes of AKI

- **Hypovolaemia** - vomiting & diarrhoea, blood loss/ surgery, diuresis, burns, poor oral intake.
- **Low blood pressure/cardiac output** – for a variety of reasons (as above) infection, sepsis, cardiogenic shock, tamponade, heart failure, ACS, drugs.
- **Blockage to flow** – thrombosis, atherosclerosis.
- **Nephrotoxic drugs** –such as ACE inhibitors, ARBs, SGL2Ti, Metformin, NSAIDS, CT contrast, PCI

- **REVIEW HISTORY, ASSESS FLUID STATUS, REVIEW DRUG CHART, CHECK OBS, SEPTIC SCREEN**



Assessing Fluid Status

Pitting oedema – extent & position

Lying & standing BP/passive leg raise

Thirsty?

Chest auscultation - evidence of crepitiations

Review fluid balance - cumulative



CXR review – evidence of pulmonary oedema

CRT delayed?

Bloods review

Colour of urine

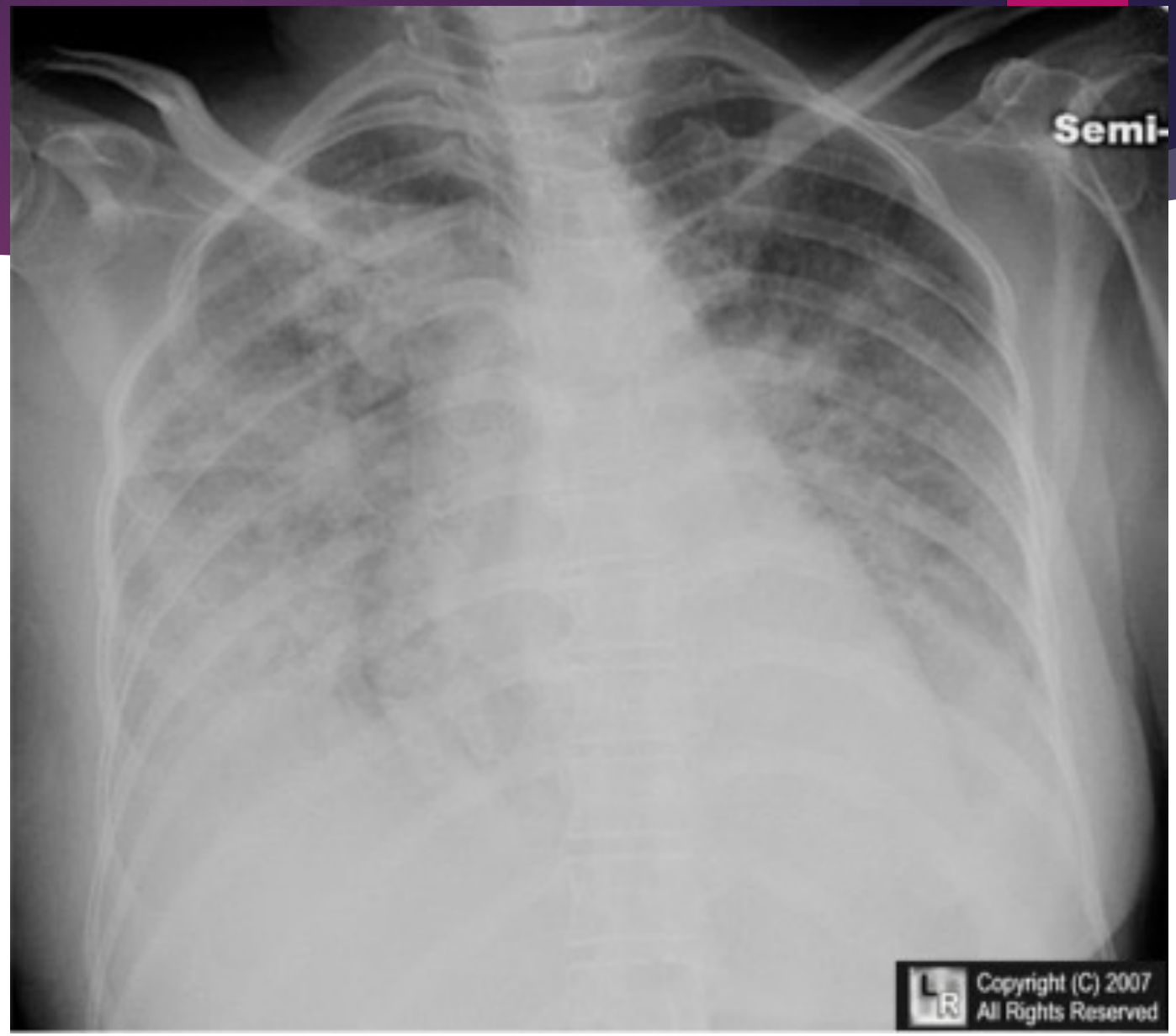
Daily weight

JVP/CVP raised

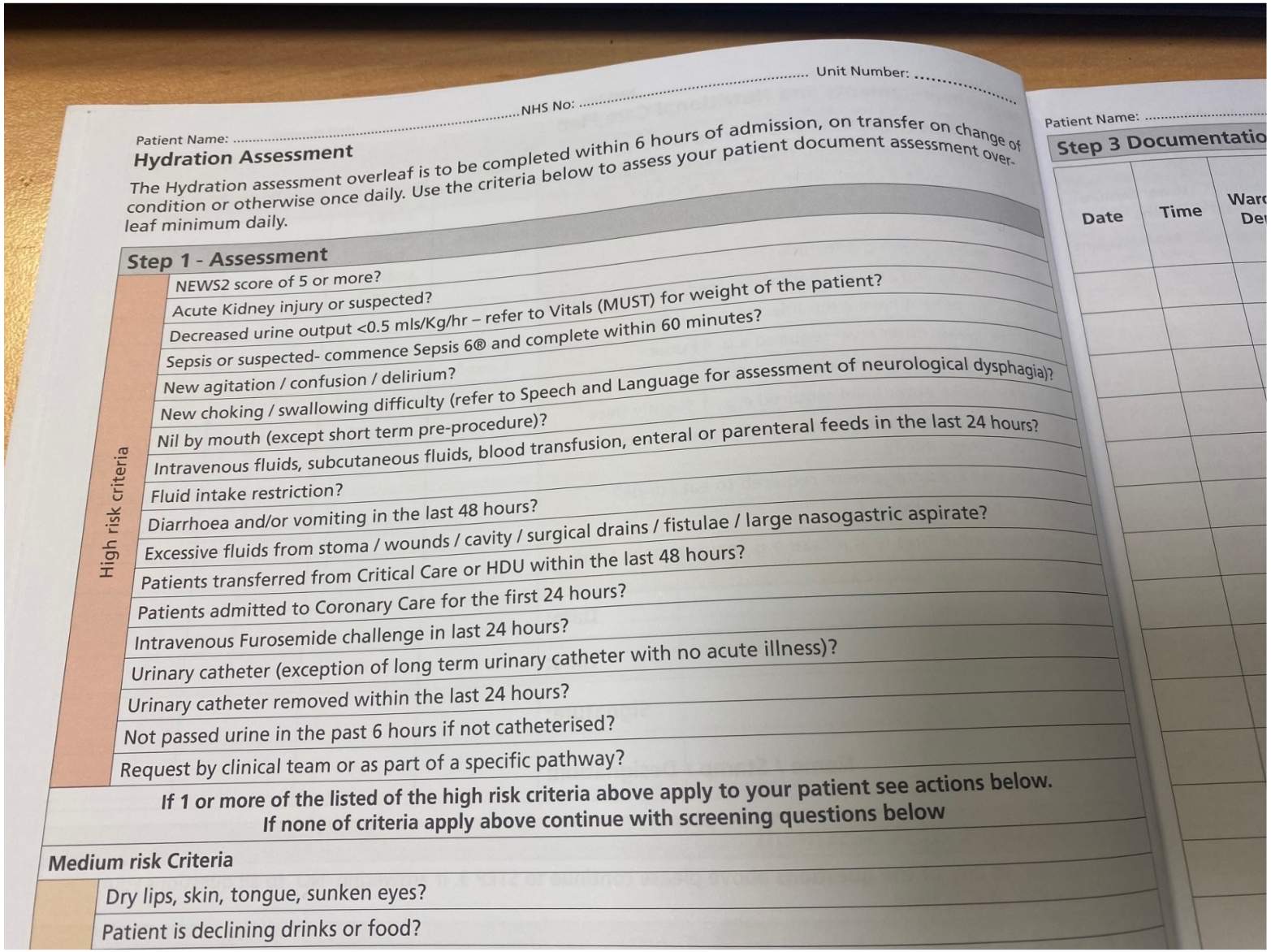
Loose skin turgor/dry mucous membranes?

Patient may require fluids, diuretics, fluid restriction

Hypervolaemia



Hydration Assessment Document



Patient Name: ..... NHS No: ..... Unit Number: .....

Hydration Assessment

The Hydration assessment overleaf is to be completed within 6 hours of admission, on transfer on change of condition or otherwise once daily. Use the criteria below to assess your patient document assessment overleaf minimum daily.

Step 1 - Assessment	
High risk criteria	NEWS2 score of 5 or more?
	Acute Kidney injury or suspected?
	Decreased urine output <0.5 mls/Kg/hr – refer to Vitals (MUST) for weight of the patient?
	Sepsis or suspected- commence Sepsis 6® and complete within 60 minutes?
	New agitation / confusion / delirium?
	New choking / swallowing difficulty (refer to Speech and Language for assessment of neurological dysphagia)?
	Nil by mouth (except short term pre-procedure)?
	Intravenous fluids, subcutaneous fluids, blood transfusion, enteral or parenteral feeds in the last 24 hours?
	Fluid intake restriction?
	Diarrhoea and/or vomiting in the last 48 hours?
	Excessive fluids from stoma / wounds / cavity / surgical drains / fistulae / large nasogastric aspirate?
	Patients transferred from Critical Care or HDU within the last 48 hours?
	Patients admitted to Coronary Care for the first 24 hours?
	Intravenous Furosemide challenge in last 24 hours?
	Urinary catheter (exception of long term urinary catheter with no acute illness)?
	Urinary catheter removed within the last 24 hours?
	Not passed urine in the past 6 hours if not catheterised?
	Request by clinical team or as part of a specific pathway?

If 1 or more of the listed of the high risk criteria above apply to your patient see actions below.
If none of criteria apply above continue with screening questions below

Medium risk Criteria	
	Dry lips, skin, tongue, sunken eyes?
	Patient is declining drinks or food?

Patient Name: .....

Step 3 Documentatio

Date	Time	Ward De

Hydration assessment and fluid balance monitoring



COMPLETE THE HYDRATION ASSESSMENT DAILY.



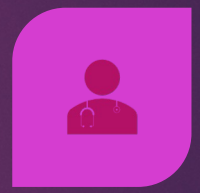
IF DEEMED AS HIGH RISK, PLEASE KEEP AN ACCURATE INPUT OUTPUT CHART. REMEMBER TO CUMULATE EVERY 6 HOURS.



ANYONE WITH AKI (1,2 OR 3) IS HIGH RISK SO NEEDS A FULL FBC



INCLUDE ALL DRAIN, NG AND STOMA OUTPUT ETC, AS WELL AS ORAL INTAKE, AND IVS.



INFORM PATIENTS THEY ARE ON A FULL FBC AND REMIND THEM TO MONITOR THEIR OWN ORAL INTAKE AND ASK FOR OUTPUT TO BE MEASURED

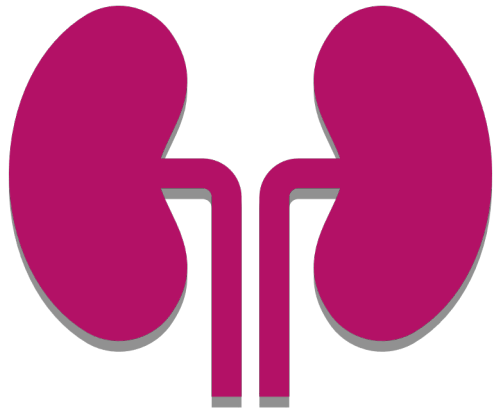


DAILY WEIGHT WILL HELP WITH FLUID ASSESSMENT.

Intrinsic kidney damage

- Damage to the structures of the kidney

- **Symptoms :**
- Peripheral oedema (in the absence of heart failure)
- New rash
- Epistaxis (nose bleeding)
- Haemoptysis (coughing up blood)
- Night sweats
- Splinter haemorrhages
- Positive urine dipstick for blood +/- protein



- ***May need a renal biopsy to confirm diagnosis***

Glomerular causes

- **Proliferative GN:**
- Typically presents as **nephritic** syndrome characterised by haematuria, mild proteinuria, HTN, oedema, elevated serum creatinine and oliguria, eg: IgA nephropathy, post-infectious GN (assoc. with strep), RPGN, Anti-GBM disease, vasculitic disorders (eg granulomatosis, polyangitis)

- **Non-proliferative GN:**
- Typically presents as **nephrotic** syndrome characterised by severe proteinuria (>3.5g/day), hypo-albuminuria and oedema. Eg minimal change disease, FSGS, membranous GN (may have normal creatinine).

Tubular

- **Acute tubular necrosis** (ATN) – usually occurs secondary to ischaemia of pre-renal AKI
- **Nephrotoxic drugs:** ACEis, ARBs, NSAIDS, Aminoglycosides (eg gentamycin), radiological contrast
- **Rhabdodomyolysis**
- **Multiple myeloma**

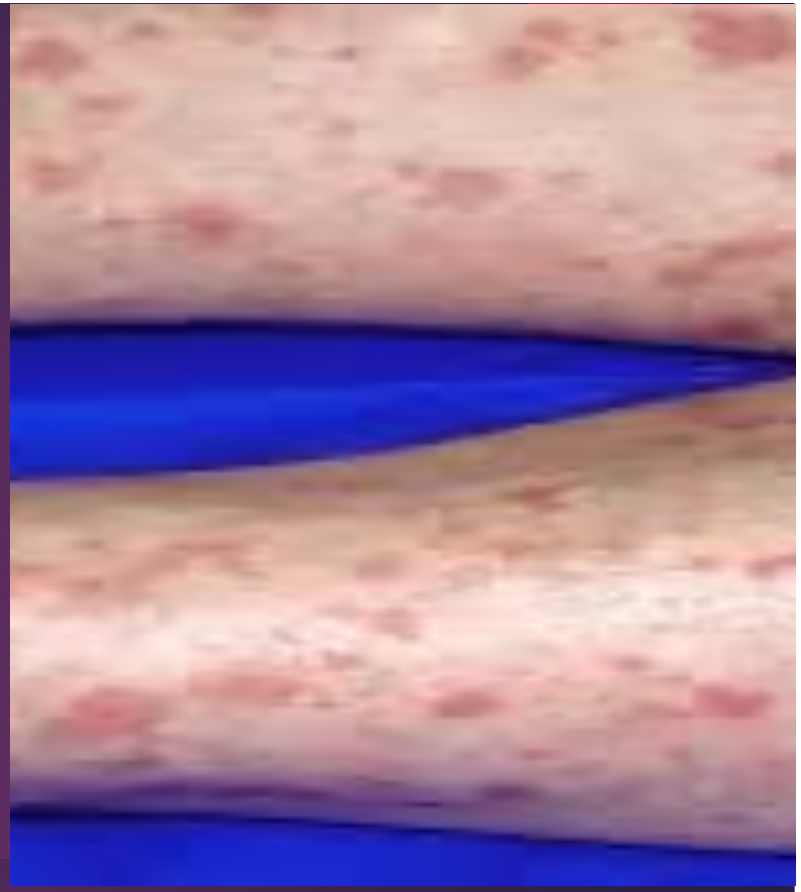
Interstitial

- **Acute Interstitial nephritis** – usually by a caused by a drug-induced allergic reaction eg, penicillin, NSAIDS, Abx
- **Automimmune disease-** eg systemic lupus erthromatosus
- **Infiltrative disease** – eg lymphoma, sarcoidosis

Vascular

- **Hypertensive nephropathy**
- **Vasculitides** (causing inflammation of blood vessels)
- **Haemolytic uraemic syndrome (HUS)**
- **Thrombotic thrombocytopenic purpura (TTP)**
- **Disseminated intravascular coagulation (DIC)**





Intrinsic signs

Urine dipstick



- Dip the urine and review after **one** minute
- Blood and protein enter the urine if the filtration system within the kidneys is not working effectively.
- 2+ of blood or 2+ protein (in the absence of leucocytes and nitrites) is a significant finding and should be documented and the AKI team informed.
- Can indicate intrinsic damage to the kidneys.
- Renal immunology screen and urine ACR
- **Refer to renal team**
- If urine dip is positive for leucocytes and nitrites as well as protein and/or blood, it indicates a UTI. It does **not** exclude intrinsic renal problems. You need to treat UTI and then repeat the urine dipstick.

Post-renal AKI

Due to obstruction of the urinary tract such as:

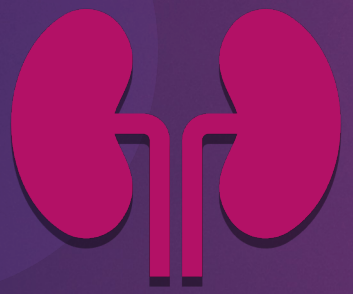
- Benign Prostate Hypertrophy (enlarged prostate)
- Bladder cancer
- Ureteric obstruction due to pelvic mass
- Bilateral renal calculi (kidney stones)
- Blocked urinary catheter/nephrostomy



- **Symptoms:** difficulty passing urine, dysuria, flank pain, tender loin, haematuria, may have palpable bladder if obstructed at that level

- **Actions**:
- Do bladder scan
- USS KUB **<6 hours** if obstruction highly suspected (contact I.R.)

Clinical approach to AKI



- Physical examination/history taking
- Drugs review
- U&Es and FBC
- VBG
- Urine dipstick +/- ACR, renal panel
- Urine osmolality/sodium – if needed to distinguish between pre-renal (high urine osmolality, low urinary sodium) and ATN (low osmolality, high urine sodium)
- Bladder scan
- Renal USS if obstruction likely
- Consider renal biopsy if intrinsic cause suspected.
- Others, eg: CXR, scans, septic screen etc

Other lab findings

- □ K, □ PO4, □ Ca
- □ CK and uric acid (rhabdomyolysis, tumour lysis)
- □ anion/osmolar gap
- Anaemia (haemolysis, myeloma, retroperitoneal haemorrhage)
- Thrombocytopenia (TTP, myeloma, DIC)
- Eosinophilia (allergic TIN, cholesterol emboli, Churg Strauss)
- Antibody titres (ANCA, ANA, anti-GBM, complements)

Management of AKI

Treat underlying cause (fluids, diuretics, Abx, steroids, nephrostomies etc)

Optimise haemodynamics

Optimise fluid status

Suspend/avoid nephrotoxic drugs



Correct electrolytes

Maintain accurate FBC/daily weight

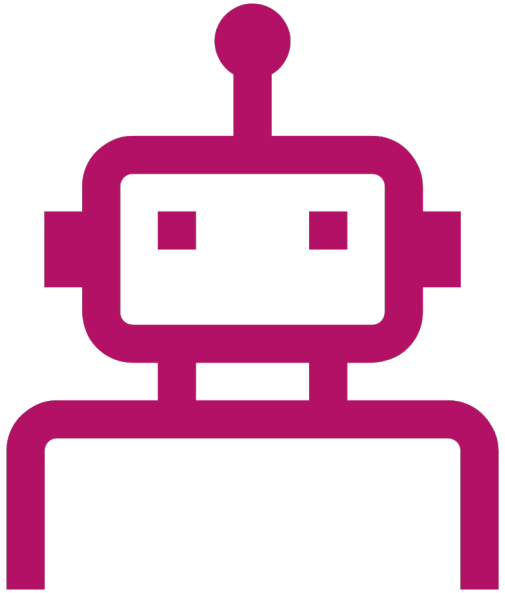
Daily bloods monitoring

Renal replacement therapy

Renally dose drugs, eg. enoxaparin, Abx, according to eGFR/CC)

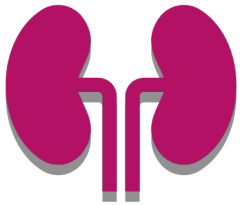
Types of renal replacement therapy

- Haemodialysis
- Peritoneal dialysis
- Continuous veno-venous haemodiafiltration (ITU only)

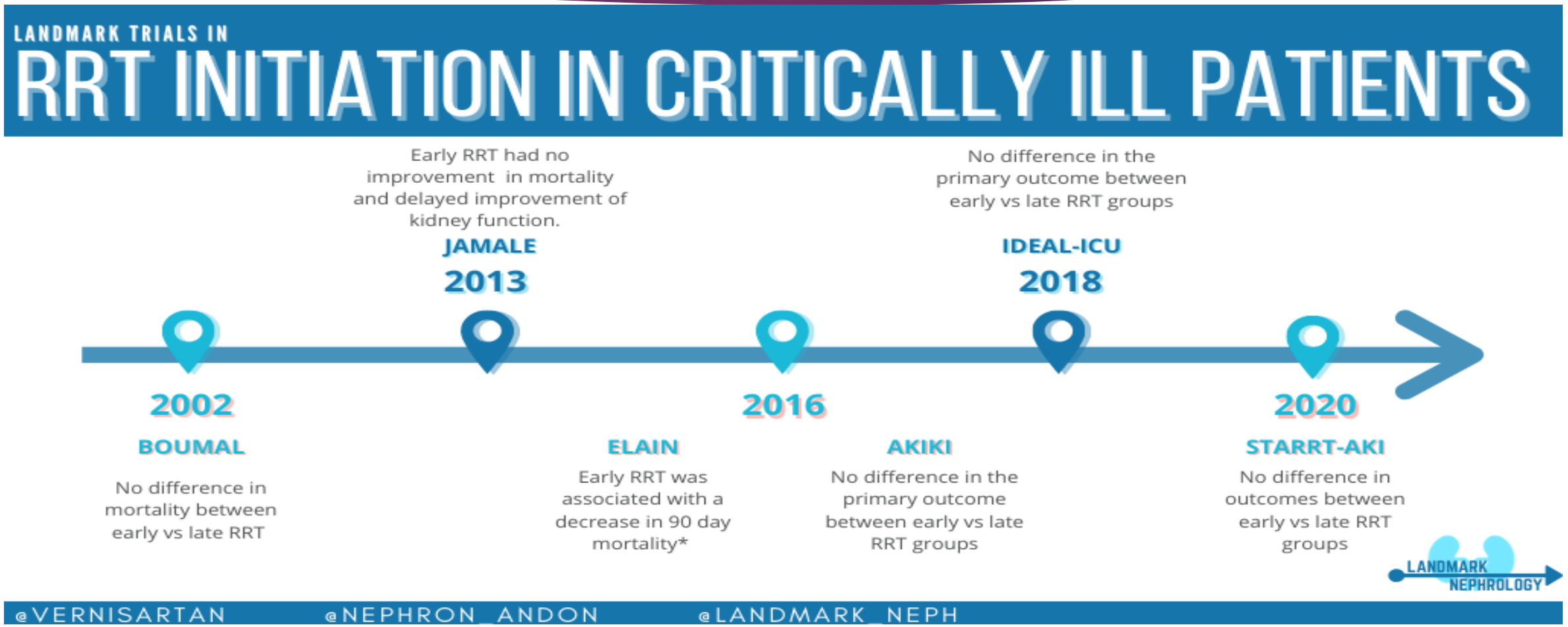


Indications for RRT

- **A** – acidosis (not responding to conventional treatment/worsening trend)
- **E** – Electrolytes (hyperkalaemia/hypercalcaemia which is refractory)
- **I** – Intoxication (drug toxicity)
- **O** – Oedema (pulmonary – not responding to diuretics)
- **U**- Uraemia (vomiting, fatigue, weight loss, muscle cramps, pericarditis, altered GCS, positive asterixis sign)

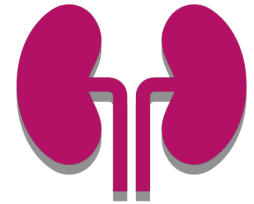


When to dialyse…….?



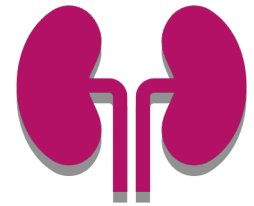
Case Study 1

- 78 –year-old male – brought to ED after found collapsed at home.
- BP = 82/48, HR 130– regular, RR 28, Sat 85% on room air, Temp 38.7C
- Creatinine 350
- (Base Creatinine 110) Urea 28, K 5.5, Na 152, eGFR 27, pH 7.23
- HPC – 10/7 history of cough. SOBOE. Reduced oral intake. Taking regular meds.
- PMH: T2DM, CKD 3, HTN, BPH
- Drugs: Lisinopril, Metformin, amlodipine, tamsulosin.
- O/E: Chest: reduced air entry/wheezing, JVP not raised. Minimal pedal oedema. Abdo soft, non-tender, no intrinsic signs, urine dipstick bland.
- **Cause** – likely chest sepsis (pre-renal). Need to rule out rhabdo
- **Tx**: Sepsis pathway – IV fluid resus & IV antibiotics//blood cultures/ O2/CXR/ Stop nephrotoxins, daily U&Es/ CK/ bladder scan/ USS KUB if no improvement.



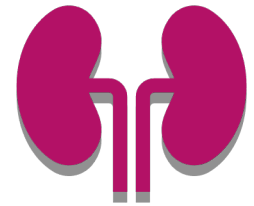
Case Study 2

- 79-year-old female – already an inpatient
- BP 95/56, HR 102, Sats 91% on room air, RR 24
- Creatinine 293
- (Base creatinine 90) Urea 24, Na 159, K 5.5, eGFR 29, WCC 9, Temp 37C
- HPC: Elective hip replacement 3 days ago
- PMH: HTN, COPD, OA
- Drugs: Losartan, Furosemide, Diclofenac, Montelukast
- Water jug empty, no FBC completed, Catheter in situ – draining, but urine output not measured.
- O/E: Lethargic, Concentrated, clear urine, Chest okay. No oedema. In pain. Thirsty. No intrinsic signs. Urine dipstick – bland. No palpable bladder.
- **Cause**: Likely pre-renal – dehydration, low BP, drugs
- **Tx:** IV fluids (bolus, infusion), stop nephrotoxins, USS KUB if not improved with fluids. Accurate FBC

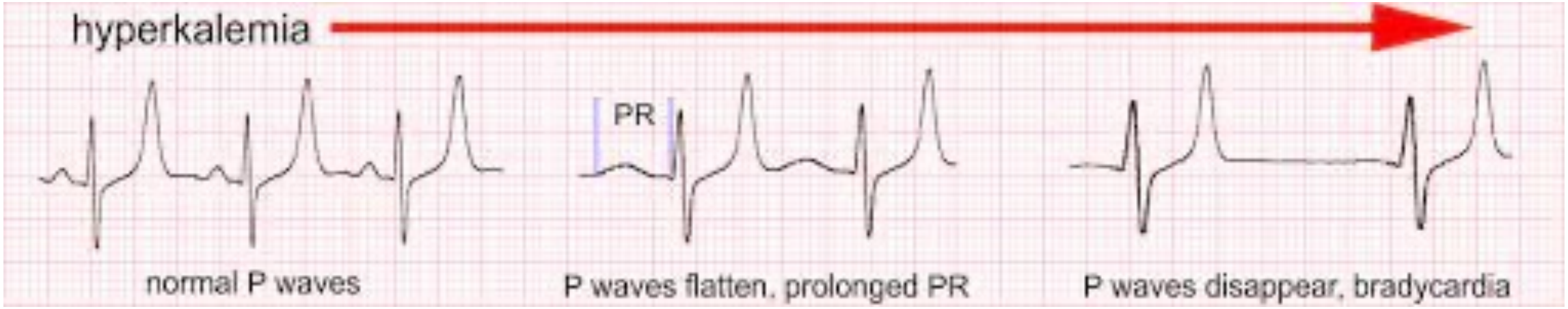


Case Study 3

- 82-year-old male, referred by GP to ED for abnormal bloods
- Creatinine 882, Urea 11, K 7.2, Na 138, eGFR 5, WCC 7
- Unknown base creatinine – assume stage 3 AKI
- Obs: BP 145/85, HR 85, Temp 36.8, Sat 95%, RR 26, slightly confused.
- PMH: HTN, BPH, T2DM
- Meds: Losartan, Metformin, Spironalactone, Tamsulosin.
- O/E: Appears euvolaemic, left flank pain, haematuria on dipstick, no protein. Difficulty passing urine.
- Cause: likely urinary obstruction (post-renal)
- Tx: urgent 12 lead ECG, Treat high K, Bladder scan, USS Abdo, VBG, stop meds
- IV bicarb, bilateral nephrostomies, ?bridging dialysis



ECG in hyperkalaemia



ROUNDUP

- **R** – Repeat U&Es daily
- **O** - Obstruction ruled out
- **U** – Urine dipstick
- **N** – Nephrotoxic meds suspended
- **D** – Dry or wet? Rehydrate or diurese
- **U** – Urine output- maintain accurate FBC
- **P** - Prescriptions reviewed

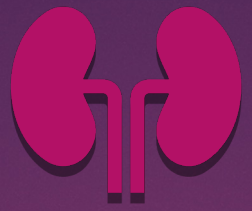
AKI team

Consists of:



- **AKI Renal Consultant (changes weekly)**
- **AKI Renal Registrar (changes weekly)**
- **AKI Clinical Nurse Specialist Team (1 x Band 7, 3 x Band 6s)**
- AKI alert list twice daily which we review and prioritise - running an 'In-reach' service
- Post discharge AKI follow-up/ AKI hot clinic
- Cardiorenal MDT
- Health promotion/information (prevention of AKI)
- Audit – prevalence of AKI/effectiveness of AKI Service
- Standardising AKI education across the Trust

In conclusion



Key features of AKI:

- Characterised by a sudden deterioration in renal function leading to an inability to maintain fluid, electrolyte and acid-base balance.
- Acute kidney injury can be life threatening if not treated in a timely and appropriate manner.
- Rapid recognition and treatment may prevent development or worsening of CKD & associated complications.
- Identification of AKI and prompt treatment leads to better outcomes for patients with better prognosis and lower mortality & improved patient experience.



QUESTIONS?