Acute Kidney Injury (AKI)
Undergraduate nurse education

Year Two

Developed Summer 2017

THINK KIDNEYS

THINK KIDNEYS NHS

Think Kidneys is a national programme led by NHS England in partnership with UK Renal Registry

Aims and Objectives

To review physiology of the kidney

- Identify illness and disease that can affect renal function
- To be able to identify causes of Acute kidney injury
- To identify evidence based nursing interventions and management of patients with Acute Kidney Injury

Media Awareness

- Thousands dying of thirst on NHS: Watchdog forced to issue guidelines on giving patients water
- Tens of thousands dying in hospital from kidney failure linked to dehydration
  NHS officials calculate up to 42,000 deaths a year could be avoided
- NICE issuing guidelines to help staff prevent deaths from condition
- Estimated a fifth of patients attending A&E every year suffer from the illness

What is the problem?

- AKI seen in 13-18% of all people admitted to hospital. Older adults most affected
- AKI in increasingly being seen in primary care without any acute illness.
- If AKI is highlighted early (community) this may prevent hospital admission or reduced length of stay.
- Associated mortality from 10-80%.
- Financial burden to the NHS estimated between £420 million and £600 million per year – equivalent of 20,000 District Nurses!

Findings from Ipsos MORI poll June 2014

Table 5.1. Which, if any of the following things do you think your kidneys do?

	Remove waste	Make Urine	Remove excess fluid from the body	Control the body's chemical balance	Process medicines	Help control blood pressure	Pump fluid/blood around the body	Help to make red blood cells	Control the bodies temperature	Help keep bones healthy	Cleans/filters/purifies blood	Other	Don't know
All responses	1208 (60%)	1020 (51%)	705 (35%)	486 (24%)	231 (12%)	193 (10%)	168 (8%)	150 (70%)	75 (4%)	56 (3%)	22 (1%)	19 (1%)	133 (7%)
Working	685 (64%)	572 (54%)	368 (35%)	270 (25%)	142 (13%)	100 (9%)	69 (6%)	79 (7%)	42 (4%)	33 (3%)	11 (1%)	10 (1%)	51 (5%)
In education	72 (53%)	58 (43%)	46 (34%)	35 (25%)	8 (6%)	16 (11%)	8 (6%)	10 (7%)	7 (5%)	1 (1%)	1 (1%)	0 (0%)	16 (12%)
Not working	451 (56%)	390 (49%)	291 (36%)	182 (23%)	81 (10%)	77 (10%)	91 (11%)	60 (7%)	26 (3%)	22 (3%)	10 (1%)	9 (1%)	66 (8%)

Findings from Ipsos MORI poll June 2014

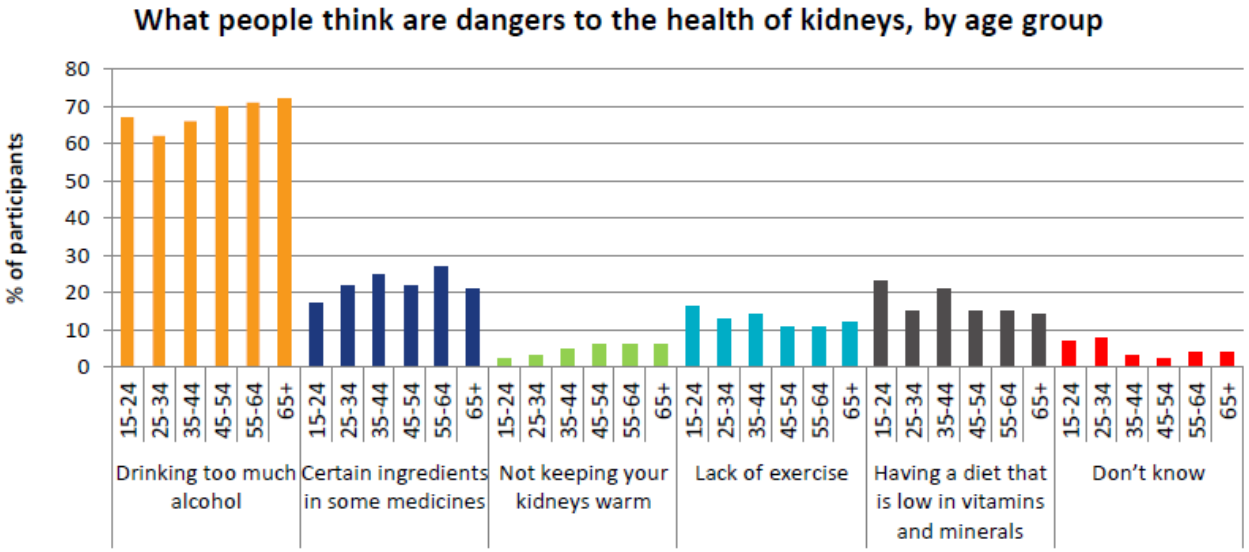


Figure 5.2

Findings from Ipsos MORI poll June 2014

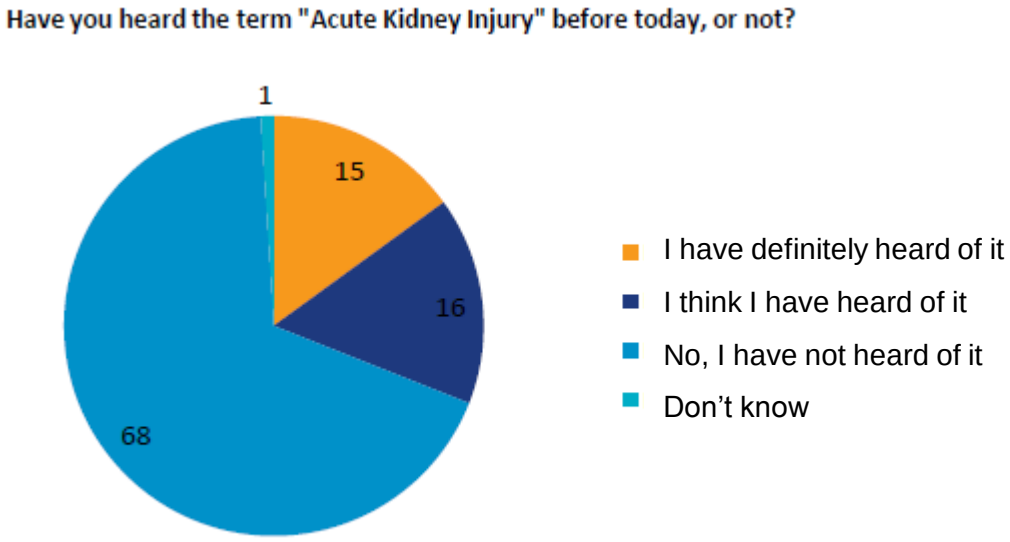


Figure 5.3

Findings from Ipsos MORI poll June 2014

5.5. What do people think best describes AKI?

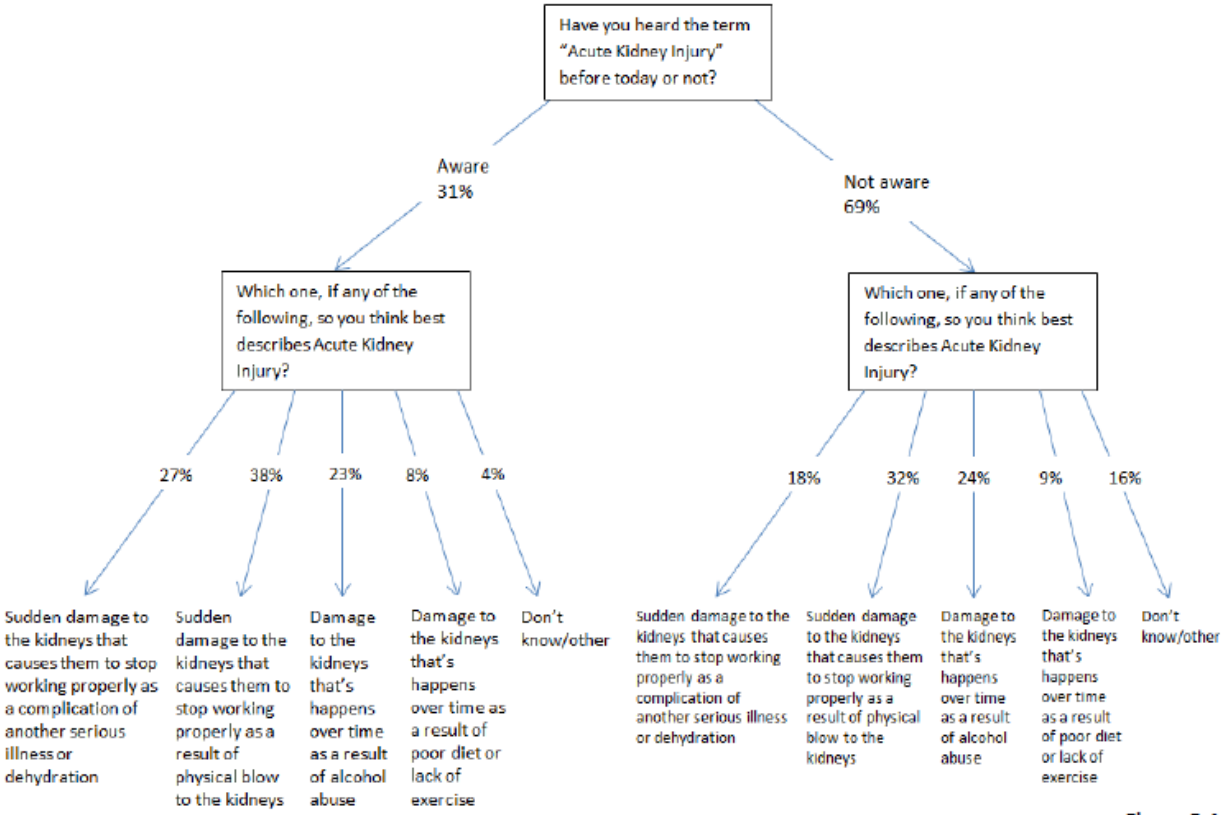


Figure 5.4

What does this mean?

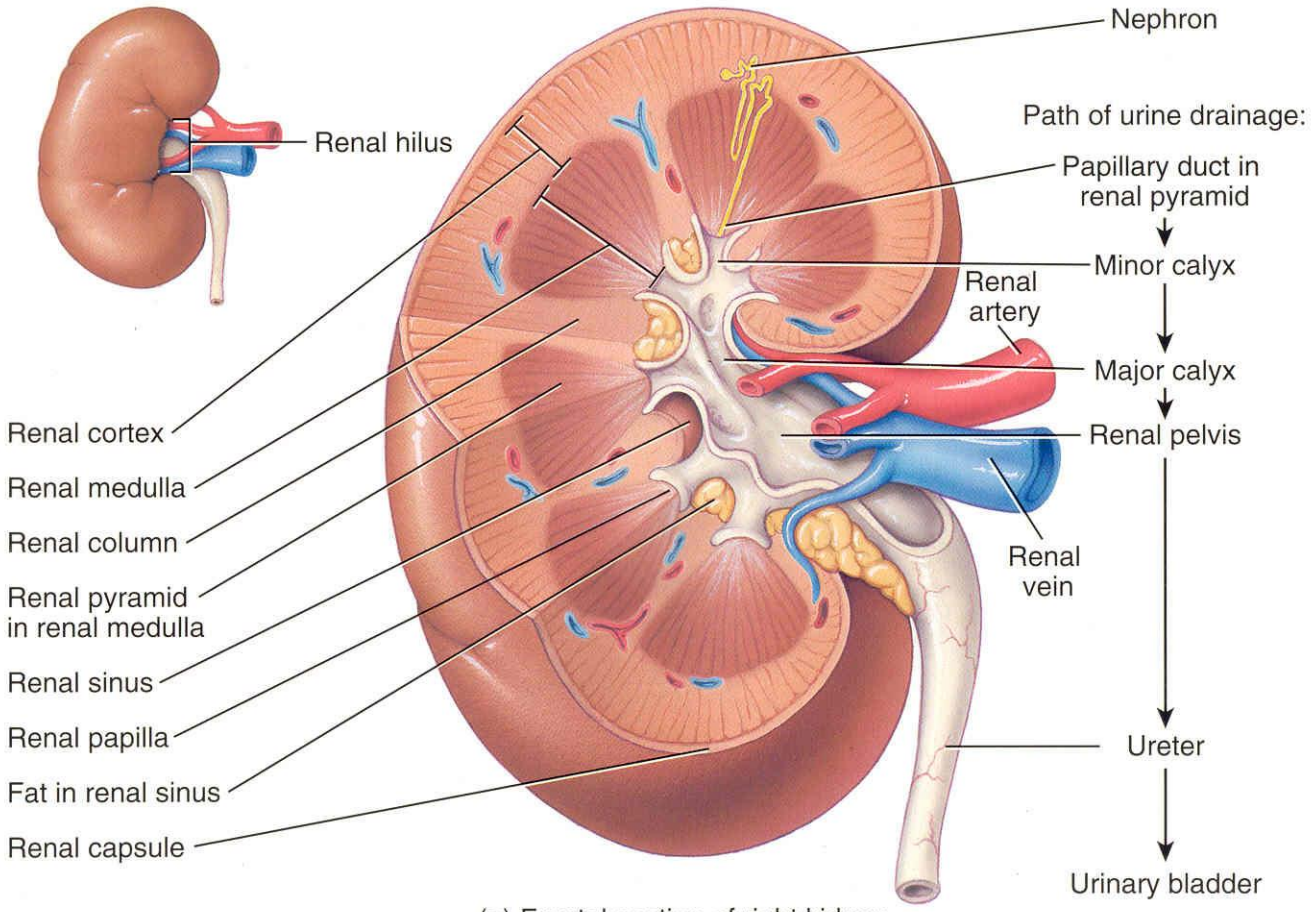
- ‘It is clear from analysis of the survey results that public knowledge levels about the normal functioning of the human kidney are low. Kidneys do not appear to be considered by the public as vital organs that need to be considered and kept healthy.’

  - ThinkKidneys/Ipsos MORI 2014

- Almost Everything You Need to Know About Your Kidneys

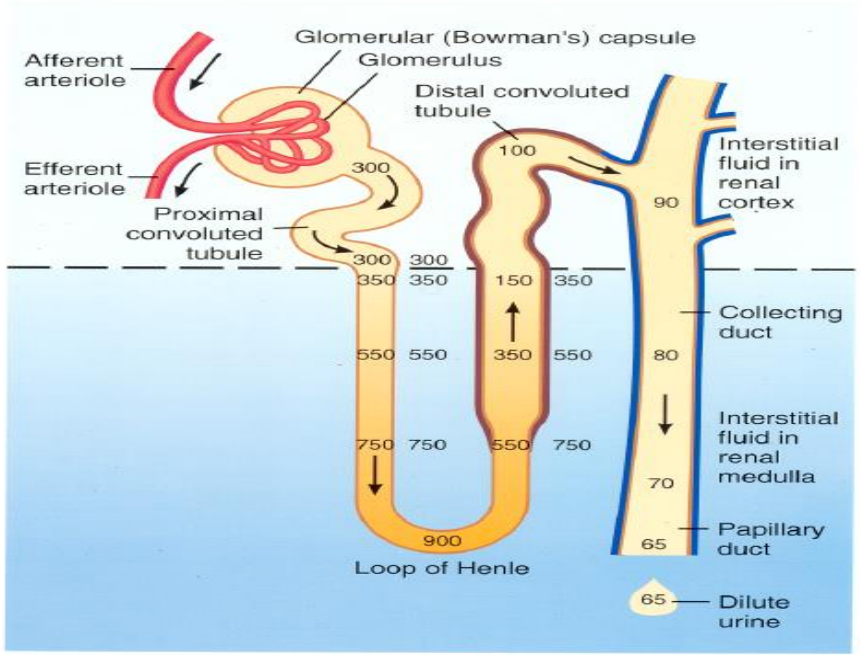
Recap:
Physiology and functions of the kidney

Internal anatomy of the kidneys (Figure 26.3a)



(a) Frontal section of right kidney

Nephron



THINK KIDNEYS

Functions of the Kidney

1. Excretion of nitrogenous wastes
2. Fluid homeostasis,
3. Electrolyte homeostasis,
4. Control of Blood pressure,
5. Acid base balance,
6. Erythropoisis,
7. Vitamin D conversion,
8. Calcium and Phosphate homeostasis,
9. Excretion of drugs and toxins.

Definition and risk factors of AKI

Definition and staging

The renal association defines AKI as...

"*AKI is characterised by a rapid reduction in kidney function resulting in a failure to maintain fluid, electrolyte and acid base balance homeostasis*" (2011, page 3).

AKI can be staged using a variety of methods from RIFLE, AKIN and KDIGO these range from 1-3.

UK Renal Association Acute Kidney Injury Clinical Practice Guidelines – 5th Edition, 2011 (New version expected to be published Autumn 2017)

https://www.thinkkidneys.nhs.uk/aki/wp-content/uploads/sites/2/2016/02/Acute-Kidney-Injury-5th-edition.pdf

Risk Factors for AKI

Age (above 75 years)

Chronic Kidney Disease

Cardiac Failure

Atherosclerotic Peripheral Vascular Disease

Liver Disease

Diabetes

Nephrotoxic medications

Severe Burns

Prolonged bouts of Diarrhoea and vomiting

Profuse sweating

Diuretic Abuse

Who is at risk of developing acute kidney injury?

- The prevalence of CKD and AKI increases with age. Between one-quarter and one-third of all adults aged over 64 years have CKD.
- The incidence of severe AKI is more than fifty times higher in people aged over 80 years than in people aged under 50 years.

AKI Classification

- **Pre-renal:** Failure to receive an adequate blood supply **(40-80%)**

Sepsis, hypovolaemia, heart disease. Low cardiac output

- **Renal:** Intrinsic damage to the kidney tissue. This is actual damage of renal cells, commonly referred to as ATN (Acute Tubular Necrosis) **(20-40%)**

Infection, diabetes, hypertension, disease. (Ischaemia)

- **Post-renal:** Impaired renal drainage (obstruction of the renal tract) **(2-10%**)

Calculi (stones) prostatic hyperplasia. Inability to pass urine…?

Causes of AKI: Pre-renal

Most common cause of AKI -
Flow disruption to the kidney

For example:

Hypovolaemia - vomiting & diarrhoea, blood loss/ surgery, sepsis

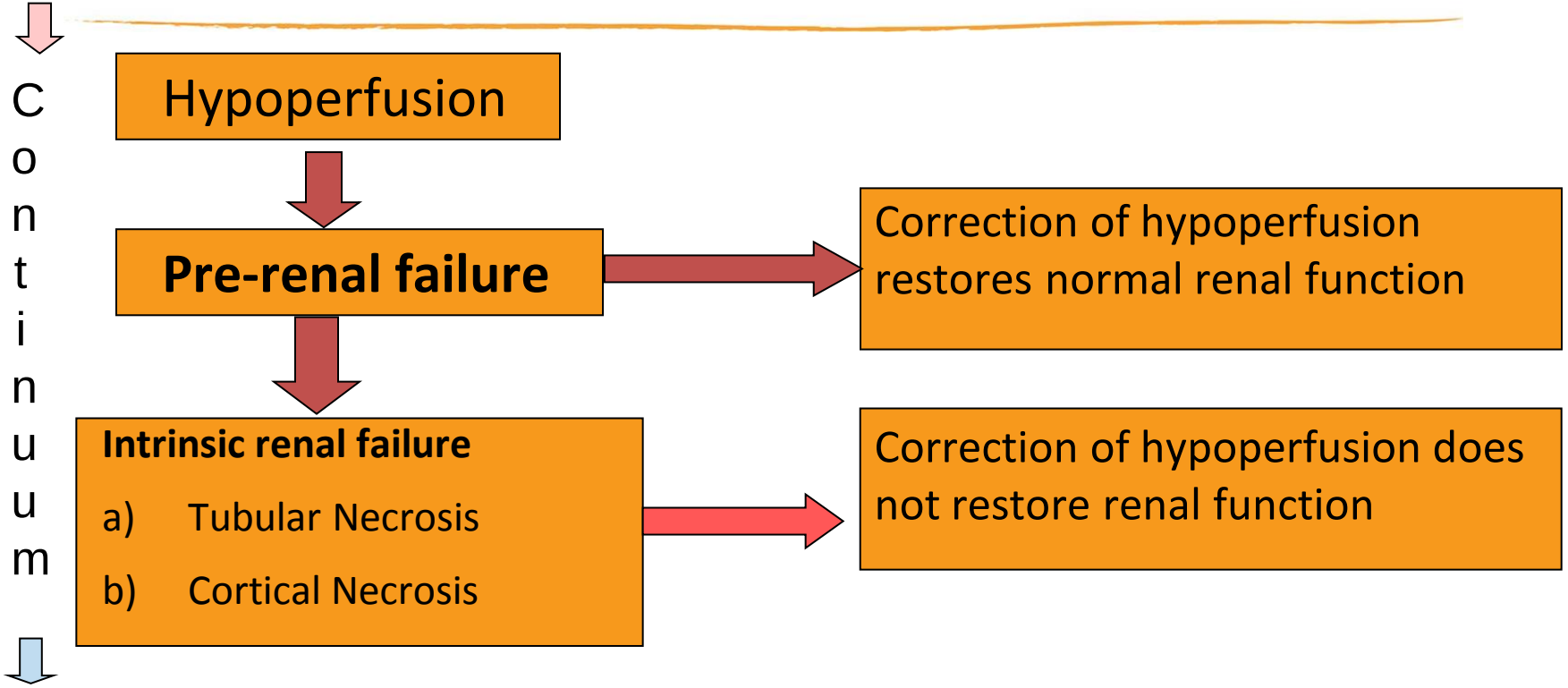
Low blood pressure – for a variety of reasons (as above) fluid loss, bleeding, sepsis, heart failure.

Blockage to flow – thrombosis, atherosclerosis.

Drugs - ACE inhibitors

Decrease in effective circulatory volume - low cardiac output - septic shock - cirrhosis

Pre-renal AKI



Causes of AKI: Intrinsic

Damage to the kidney itself

For example:

Glomerulonephritis, infection.

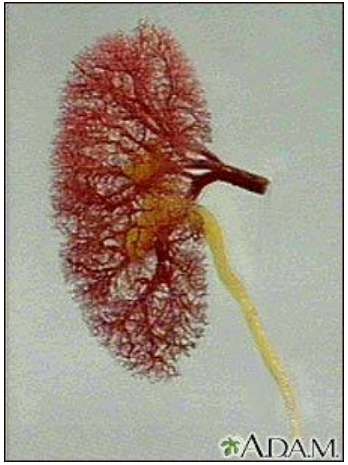
The main cause of intrinsic damage is from **Acute Tubular Necrosis (ATN)** caused by:

- **Ischaemic injury:** (can occur if blood flow to the kidneys is not improved following one of the pre-renal insults)
- **nephrotoxic injury:** exposure to many drugs, poisons & endogenous compounds in high concentrations

Occurs when there is damage to the structures of the nephron, such as the glomeruli, tubules, vessels or interstitium

Acute Tubular Necrosis

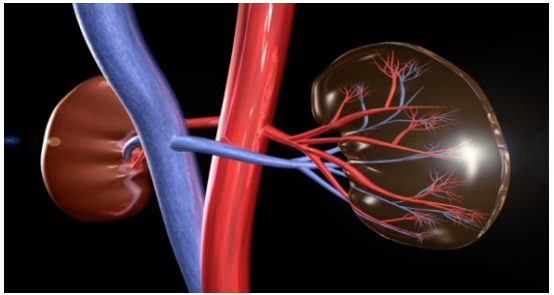
- Pre-renal and intrinsic renal. Lack of blood flow and oxygen can result in cell injury and acute tubular necrosis (ATN).
- Recovery of AKI is dependant upon the severity and extent of ATN.
- Diagnosing ATN is one of exclusion.



Causes of AKI

Post Renal

- A consequence of urinary tract obstruction.
- For example:
- Blocked catheter
- Renal calculi
- Bladder tumours
- Retroperitoneal fibrosis
- Prostatic hypertrophy
- Cervical carcinoma
- Urethral stricture
- Intra-abdominal hypertension



Identification of AKI

Identification of AKI

Measuring changes in serum creatinine as well as changes in urine volume in patients who have risk factors can help identify AKI.

The markers used are;

- Urine output
- Serum creatinine
- eGFR (estimated glomerular filtration rate)
- Some hospitals have AKI alerts (when creatinine is elevated by 26µmol/L)

KDIGO Staging of AKI

Stage	Serum Creatinine	Urine Output
1	1.5-1.9 times baseline **OR** **26.5 µmol** increase from baseline	<0.5 ml/kg/h for 6-12 hrs
2	2.0 -2.9 times baseline	<0.5 ml/kg/h for ≥12 h
3	3 times baseline **OR** **Increase in SrCr** **353.6µmol** **OR** Initiation of RRT	<0.3 ml/kg/h for ≥24 h **OR** Anuria for ≥12 h

Diagnosis: Urine

Minimum expected urine output per day **0.5mls/kg/hr.**

How could this be measured?

- Consider **acute hospitals**
- Consider **community**

Acute fluid balance charts/ urine colour charts and fluid estimations could be documented in patients' assessment sheets and alert nursing staff to a problem.

Patients who are incontinent – easy. Weigh pads 1ml / 1g

Patients who are continent – education regarding importance of urine output and how to recognise dehydration.

Urine as a marker of AKI

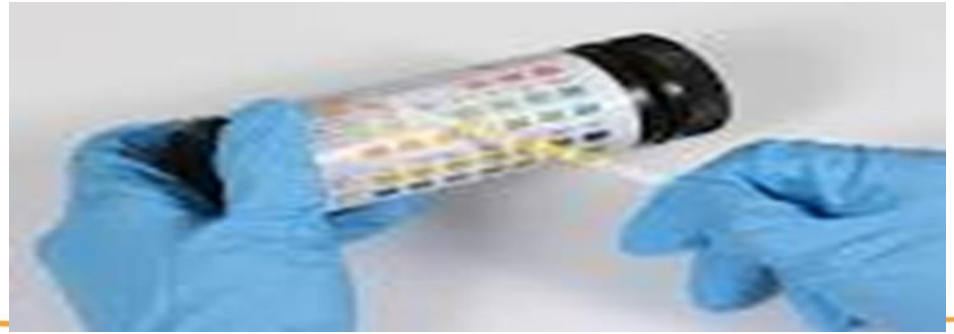
- Urine output is used as a decrease in output is often seen before changes in creatinine (Levin et al., 2007).
- Urine output is however, less specific than creatinine
- Urine output can remain normal in the present of severe AKI especially in the presence of diuretics.

Urinalysis

Assessment of urine can provide vital information as to whether the damage is within the kidneys or as a result of pre renal cause.

Blood and protein enter the urine if the filtration system within the kidneys is not working effectively.

2+ of blood or 2+ protein is a significant finding and should be documented and appropriate individual informed.



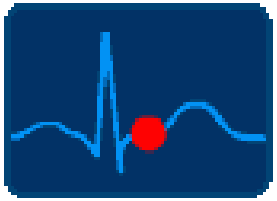
Complications and Management

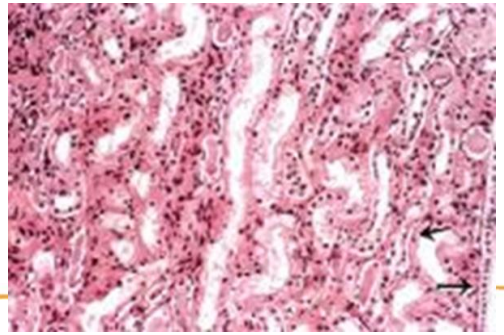
Treatment of AKI

- Acute kidney injury is frequently **reversible.**
- Rapid **recognition** and **treatment** may prevent irreversible nephron loss.
- Patients with underlying renal insufficiency may be prevented from reaching end- stage renal failure.

Specific complications of AKI:

- Fluid overload
- Hyperkalaemia
- Acidosis,





Emergency treatment of AKI: Fluid

- Fluid management is extremely important as hypovolaemia exacerbates AKI. However rapid fluid infusion can result in overload which is also life threatening.

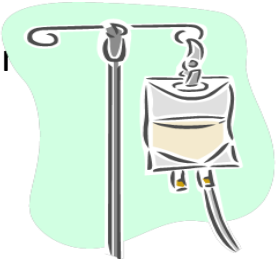
- Fluid challenge: What does this mean?
- Improving urine output will help resolve issues with hyperkalaemia/ fluid acidosis

- Consider the role of Diuretics

Fluid Therapy

Assess; A-E assessment heart rate, blood pressure, capillary refill (should be <3 secs), conscious level.

Fluid status:



- If hypovolaemic a bolus of fluid (e.g. 250-500mls) should be considered, regular review and repeat if required.
- If the patient is euvolaemic (normal fluid status) - maintenance fluids may be prescribed. This is based on estimated output plus 500mls.
- If the patient is overloaded and there is evidence of oedema- diuretics or Haemodialysis may be required if no response.

Clinical course

- **Oliguric/ non- oliguric phase**
  - <400mls urine/24hrs
- **Diuretic Phase:**

  Increased urine output. 3000mls/24hrs. Hydration and electrolyte management essential
- **Recovery Phase:**

  Tubular function is restored, diuresis subsides kidneys function as normal.

AKI Management Bundle

Do not routinely administer loop diuretics

Consider loop diuretics for treating fluid overload or oedema

- while awaiting renal replacement therapy or
- renal function is recovering without renal replacement therapy

Emergency treatment of AKI: ACIDOSIS and Hyperkalaemia

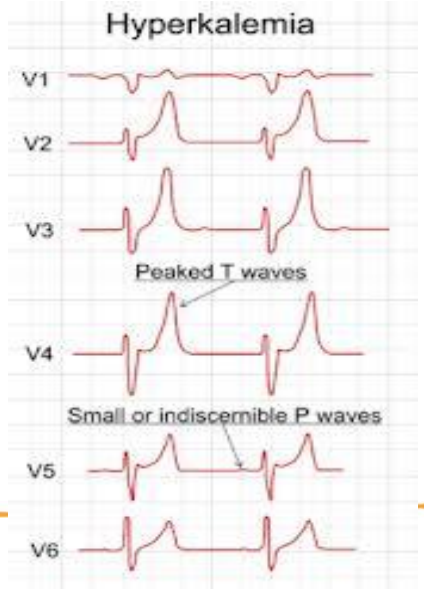
If the kidney is failing it can no-longer maintain **acid-base balance** by producing bicarbonate to buffer the acid and excreting hydrogen.

Treatment is to administer **fluid** to try to manage the cause of AKI.

Improving urine output will help resolve issues with hyperkalaemia although other measures are required.

Emergency treatment of AKI: Hyperkalaemia

1. **Protect the heart**: Calcium gluconate 10% in 10mls: It protects the heart from excess potassium excitability
2. **Shift potassium into cells**:
   - Insulin and dextrose
   - Salbutamol
3. **Remove potassium**
   - Fluid
   - Renal replacement therapy
   - Oral calcium resonium
4. **Monitor** potassium and glucose
5. **Prevent** recurrence



Management of Hyperkalemia

Cardiovascular Monitoring

Calcium Gluconate

- Antagonizes action of K at membrane
- Quick on set
- Protects myocardium from arrthymias
- Short duration

Dextrose & Insulin

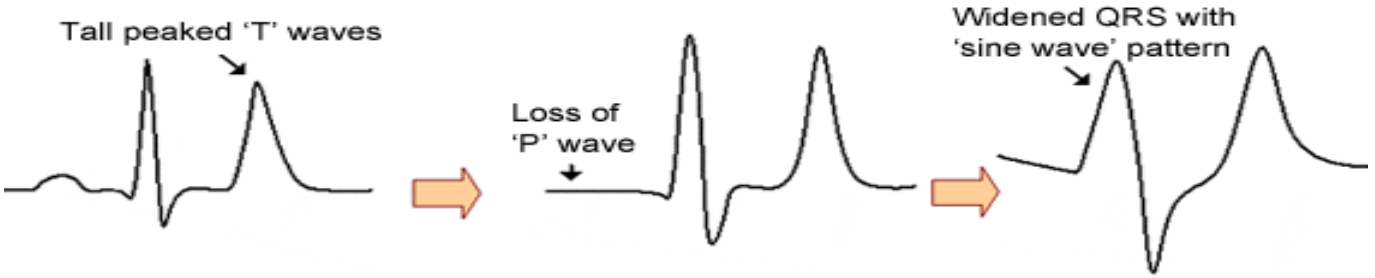
- Shifts K back into the cells
- Monitor blood sugar

ß2 Atonists

- Monitor heart rate

Cardiovascular - Hyperkalemia Signs & Symptoms

Arrhythmias peaked t waves loss of p waves widened QRS => VF &/or asystole



Management of Hyperkalemia

Sodium Bicarbonate:

- Shifts K+ back into cell
- Can increase extracellular volume (hypervolaemia)
- Watch for alkolosis

Calcium Resonium:

- Tends to be non emergency treatment Exchanges sodium for potassium in the intestinal tract
- Causes constipation (give lactulose)

Other complications: Infection

- Main cause of death
- Impaired immunological response Impaired WC function
- Impaired wound healing + patient catabolic, Prophylactic Antibiotics is not recommended
- Skin and pressure area care
- Regular observations

Renal Replacement Therapy:

What, When & Why?

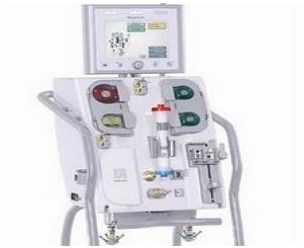
When to initiate RRT?

1. U&E's unstable
2. Metabolic acidosis
3. Fluid overloaded
4. Ureamia

AKI classification do not indicate if or when to initiate RRT

3 basic types;

1. Haemodialysis (usually intermittant 2-4hrs - rapid removal of waste products & fluid)
2. Haemofiltration (CRRT) (continous - gentle removal of waste products & fluid)
3. Peritoneal Dialysis (Catheter inserted into peritoneum - peritonium acts as filter - gentle used more in paeds)



'THINK KIDNEYS'

Care bundles may support management of a patient with AKI

Think Kidneys is a national programme led by NHS England in partnership with UK Renal Registry

AKI Care Bundle

This is a Medical Emergency

Full set of physiological obs
Assess for signs of shock
If NEWS triggering give O2, begin resuscitation and contact outreach team

Fluid Therapy

Assess heart rate, blood pressure, jugular venous pressure,
capillary refill (should be <3 secs), conscious level.
If hypovolaemic give bolus fluids (e.g. 250-500mls) until volume replete with regular review of response.
Middle grade review if >2 litres filling in oliguria.
If the patient is euvolaemic give maintenance fluids (estimated output plus 500mls)
and set daily fluid target.

Monitoring in AKI

Do arterial blood gas and lactate if venous bicarbonate is low or evidence of severe sepsis or hypoperfusion.
Consider insertion of urinary catheter and measurement of hourly urine volumes.
Measure urea, creatinine, bone, other electrolytes and venous bicarbonate
at least daily while creatinine rising.
Measure daily weights, keep a fluid chart and perform a minimum of 4 hourly observations.
Perform regular fluid assessments and check for signs of uraemia

AKI Care Bundle (cont)

Investigation of AKI

Investigate the cause of all AKI unless multi-organ failure or obvious precipitant

Urine dipstick. If proteinuria is present perform urgent spot urine protein creatinine ratio (PCR).
USS should be performed within 24 hours unless AKI cause is obvious or AKI is recovering or within 6 hours if obstruction with infection (pyonephrosis) is suspected.
Check liver function (hepatorenal), CRP and CK (rhabdomyolysis). If platelets low do blood film/LDH/Bili/retics (HUS/TTP). If PCR high, consider urgent Bence Jones protein & serum free light chains.

Supportive AKI care

Treat sepsis - in severe sepsis intravenous antibiotics should be administered within 1 hour of recognition.
Stop NSAID/ACE/ARB/metformin/K-sparing diuretics and review all drug dosages.
Give proton pump inhibitor and perform dietetic assessment.
Stop anti-hypertensives if relative hypotension. If hypovolaemic consider stopping diuretics.
Avoid radiological contrast if possible. If given follow prophylaxis protocol.

Causes Think 'STOP AKI'

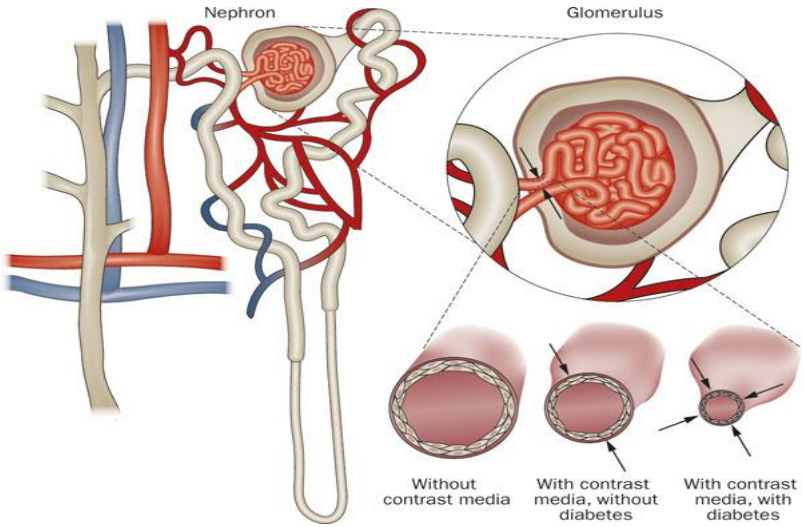
Sepsis and hypoperfusion, Toxicity (drugs / contrast), Obstruction, parenchymal kidney disease (acute GN)

On going management

Management of AKI

- Identify cause.
- Fluid management, fluid balance and daily weights
- Blood pressure management.
- Avoid nephrotoxic agents and avoid radio-contrast procedures
- Nutritional support.
- Treat infection, remove lines, catheters.

Contrast AKI



3rd biggest cause of AKI

It occurs within 72 hours of receiving the contrast and recovers over five days.

Its incidence increases in patients with risk factors i.e. renal dysfunction, diabetes and elderly

The kidney injury results from a combination of afferent arteriolar vasoconstriction and direct toxicity of the contrast media to the tubular epithelial cells.

Prevention

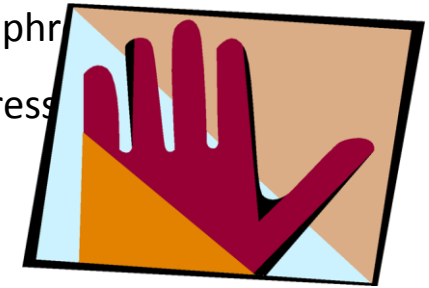
Prevent AKI - The 4 ‘M’s

1. **Monitor Patient**
   obs and EWS, regular blood tests, pathology alerts, fluid charts, urine volumes
2. **Maintain Circulation**
   hydration, resuscitation, oxygenation
3. **Minimise Kidney Insults**
   e.g. nephrotoxic medications, surgery or high risk interventions, iodinated contrast and prophylaxis, hospital acquired infection
4. **Manage The Acute Illness**
   e.g. sepsis, heart failure, liver failure

Prevention is better than cure

Up to 30% AKI maybe preventable by:

- volume replacement
- discontinuing and/or avoiding certain potentially nephr
- earlier recognition of conditions causing rapid progress
- NEWS



“Predictable and avoidable AKI should never occur”

Ncepod (2009)

Conclusion

Key features:

- Rapid onset - usually reversible.
- Characterised by ureamia– accumulation of nitrogenous waste in the blood (urea and creatinine).
- Oliguria and anuria.

Acute kidney injury is life threatening if not treated in a timely and appropriate manner.

Identification of AKI and prompt treatment leads to better outcomes for patients with better prognosis and lower mortality.

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