New Advice for AKI Detection and Prevention in Primary Care

Kathryn E Griffith
RCGP Representative for Kidney Care
Member of Think Kidneys NHS England
AKI Project Board

NICE AKI Guideline August 2013
What is AKI?

- This is a loss of kidney function over hours or days
- Low levels of public and professional awareness
- Diagnosis starts with the identification of hypotension and falling urine output during acute illness, and arranging kidney function testing
- Urine should be dipstick tested for blood, leucocytes, protein, nitrites and glucose and remember acute nephritis
- Hydration and safe prescribing are priorities

Why does it matter?

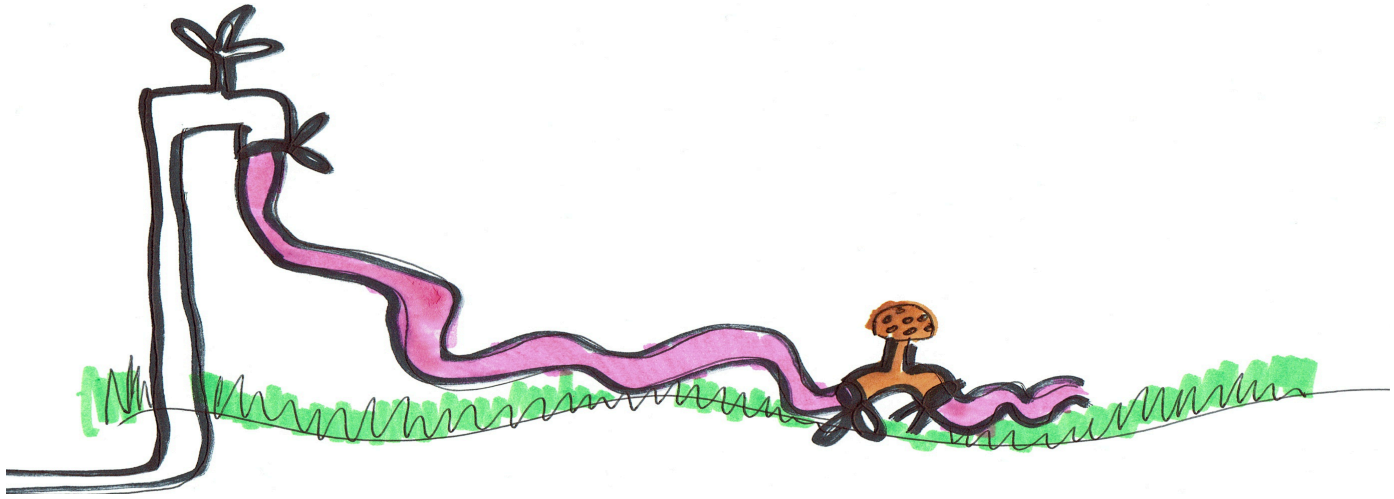
- AKI is associated with 1 in 5 emergency hospital admissions
- Off all people with AKI, 2/3 developed it in the community
- It is associated with increased mortality in short and long term, contributing to 100,000 deaths /yr
- It has poorer health outcomes
- People are more likely to have more CKD after AKI
- It is associated with longer lengths of hospital stay and more need for HDU and ICU care
- It is associated with increased RRT

Why does it happen?

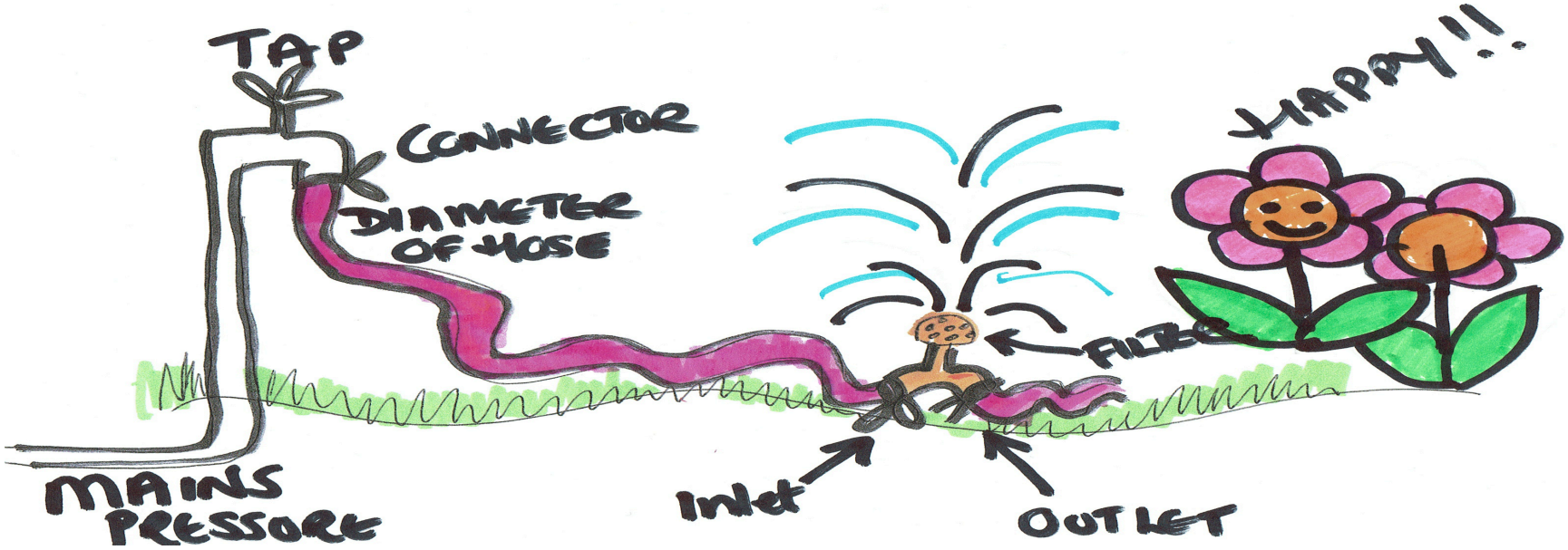
- Any condition associated with reduced perfusion of the kidney can be associated with AKI when the person is exposed to an acute illness
- AKI is more likely when the kidneys are more susceptible to damage for example older people with complex co-morbidity, existing CKD and multiple medications

Griffith Garden Sprinkler System

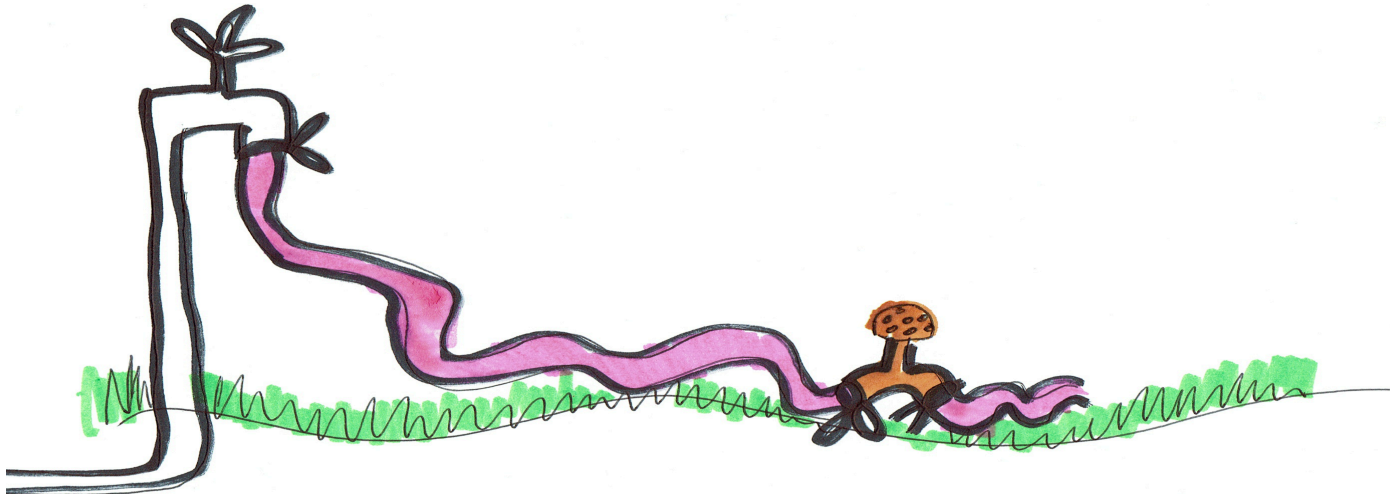
How do you make it work?



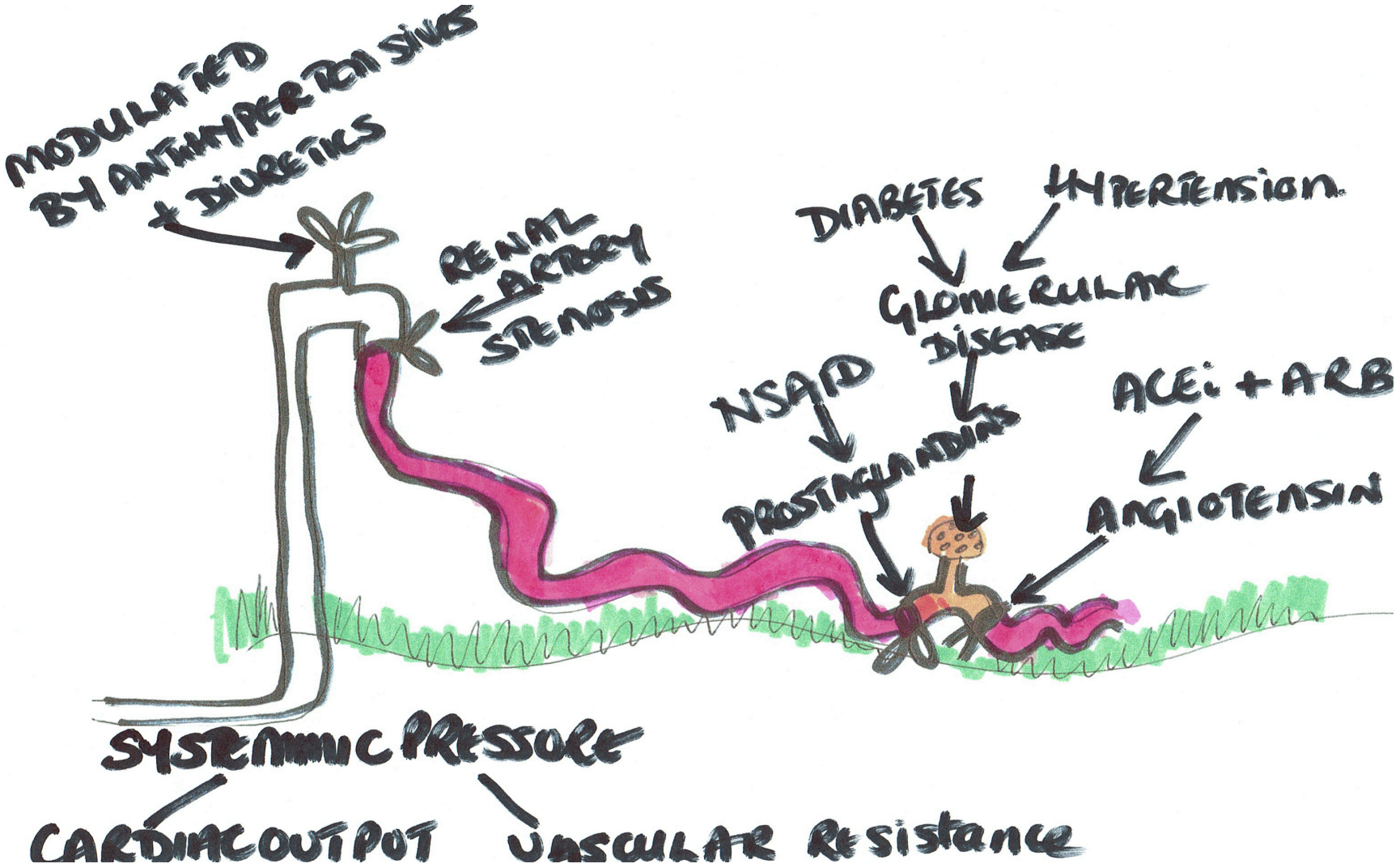
How does it work well??



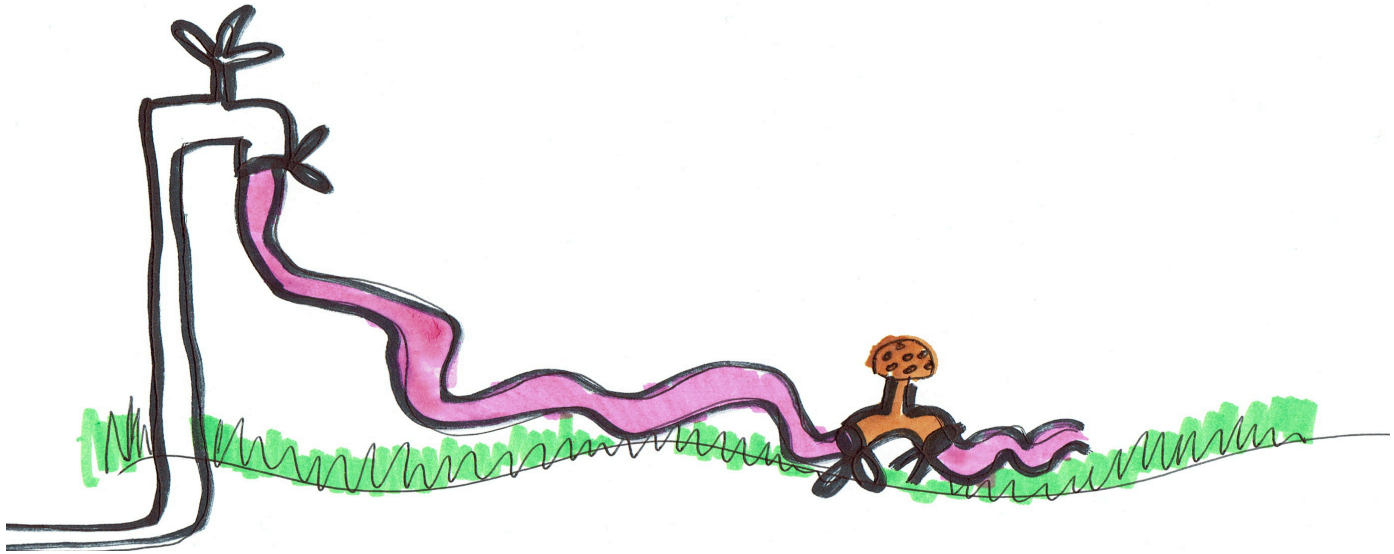
How do you make a kidney work?



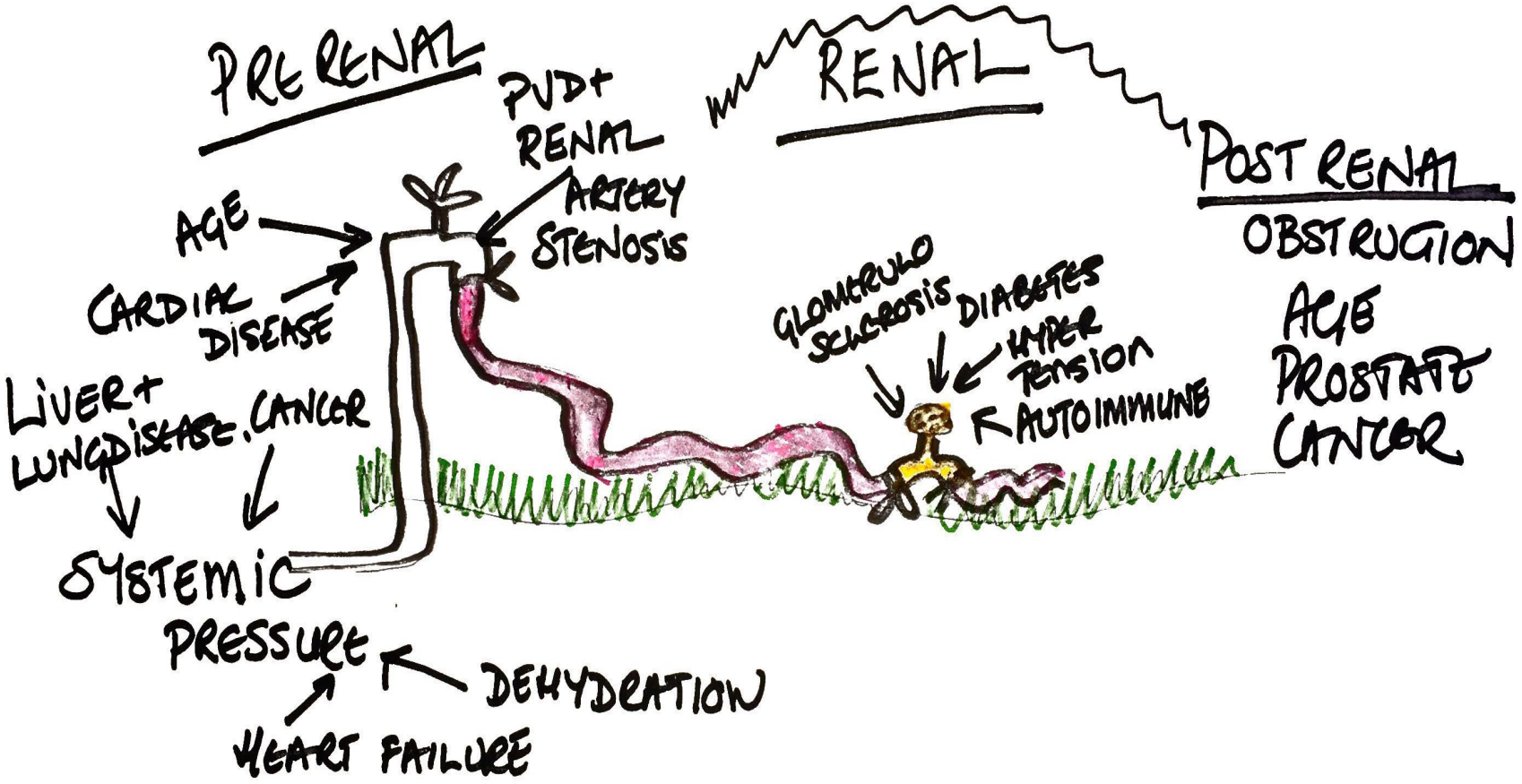
Kathryn's Kidney



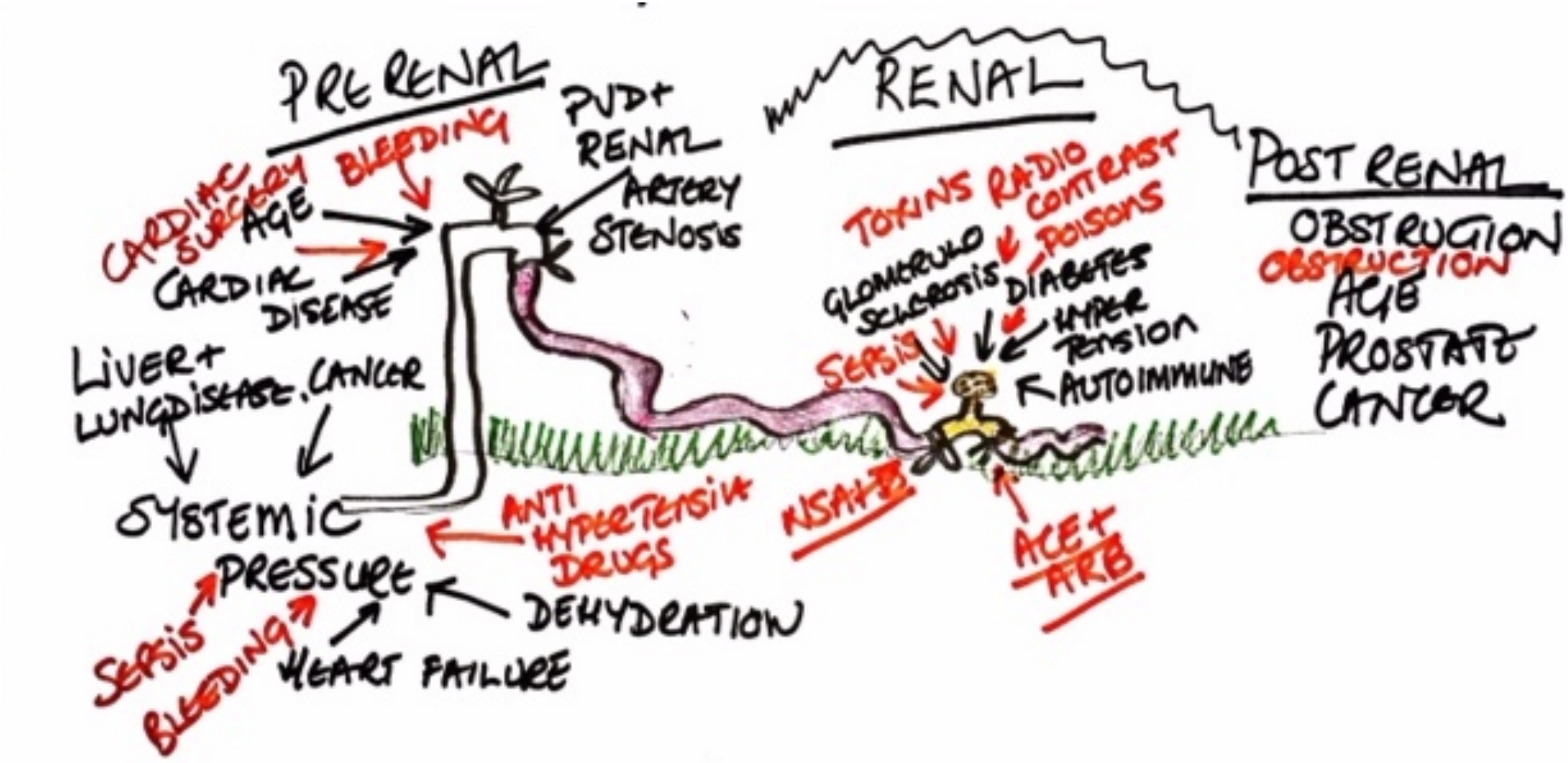
How do you make it easy to understand?
What makes a kidney susceptible to AKI?



How do you make it easy to understand?
What makes a kidney susceptible to AKI?



What exposures make AKI more likely?



Potential causes of AKI

Exposures (mostly reversible)	Susceptibilities (mostly irreversible)
Sepsis	Dehydration or volume depletion
Critical illness	Advanced age
Circulatory shock	Female gender
Burns	Black race
Trauma	CKD
Cardiac surgery especially bypass	Chronic heart, lung or liver disease
Major surgery	Diabetes mellitus
Nephrotoxic drugs	Cancer
Radiocontrast agents	Anaemia
Poisonous plants and animals	Hypertension

Diagnosis of Acute Kidney Injury

AKI Stage	Serum creatinine	Urine output
Stage 1	Increase of more than or equal to 26.5 umol/l or increase of 150-200% from baseline	Less than 0.5ml/kg/h for more than 6 hours
Stage 2	Increase of 200-300% from baseline i.e. 2-3 fold	Less than 0.5ml/kg/h for more than 12 hours
Stage 3	Increase to more than 300% i.e.3 fold increase from baseline or more than 354 umol/l	Less than 0.3ml/kg/h for more than 24 hours. Or anuria for 12 hours

Are we detecting this in primary care?

- **Stage 1**
- Increase of more than or equal to 26.5 umol/l in 48 hours or increase of 150-200% from baseline in previous 7 days
- **Stage 2**
- Increase of 200-300% from baseline i.e. 2-3 fold rise
- **Stage 3**
- More than 3x or higher than 354umol/l?
- Do we always check what baseline is???
- Are we diagnosing AKI at the moment?

Stage 3: Directive : AKI eAlert

- Recommendation for laboratory to report results when there is a change in creatinine in line with AKI
- Started in hospital 9th March 2015
- National roll out from April 2016
- This is NOT the diagnosis of AKI which requires clinical symptoms and signs as well
- Remember pseudo AKI with trimethoprim
- Remember increasing creatinine levels in a well person having CVD drugs up titrated
- How will it work?

How will it work?

- Detection of changes in creatinine by laboratory using standard algorithm
- Test results in AKI warning stage
- Communication to primary care using interruptive method (phone call), to practice or out of hours for Stage 2 and 3
- Non interruptive with routine blood results for Stage 1 with creatinine
- Expect result to be seen in 72 hours (Friday to Monday) need to cover for doctors away
- Action from primary care recommended within 24hrs of receiving the results
- How many alerts do you think that you will get?

Eric aged 82

- Telephone call 5:30pm
- Blood taken this morning
- Creatinine 200
- Previous value 90
- Potassium 5.8mmol/l
- AKI Alert
- What would you like to know?

Eric aged 82

- What will you do next??
- Have a system or template
- What would you put on it?
- How do you avoid broken door syndrome?

Action plan

- Could this be AKI?
- Compare with previous blood tests
- What stage of AKI is it?
- Look at change in creatinine
- What is the clinical context?
- Is the person sick or stable?
- Why was the blood taken?
- Was it a routine test in a stable patient? This means that there is a low pre test probability of AKI
- Was is an urgent test in a sick patient? This means that there is a high pretest probability of AKI

What next?

- What might the cause be?
- What medications is the patient taking?
- What is the patients fluid status and how do you assess hydration?
- Are they home alone or do they have help to maintain hydration?
- Do they need face to face review?
- If so how quickly?

Think Kidneys Guide for Primary Care Who should be assessed in 6 hours?

- Potassium >6.0 whatever stage of AKI
- People who are acutely unwell with potassium > 5.5 mmol/l
- People with AKI 2 and 3
- People with underlying heart failure or CKD 4 and 5
- People with poor urine output and fluid intake
- EVERYONE ELSE REVIEW WITIHIN 24 HOURS

Do they need admission

- What factors make you consider admission?

Is there hyperkalaemia?

- Level of AKI with potassium level determine the response to AKI
- High potassium above 5.5 mmol/lis associated with increased mortality
- Top normal range 5.3mmol/l
- > 5.5-6mmol/l considered mild hyperkalaemia and requires action
- Potassium >5.5mmol/l present in 14% admissions, but 21% Stage 3 CKD and 42% Stage 4 (Einhorn et al 2009)
- 6-6.5mmol/l is moderately severe and > 6.5mmol/l severe
- All people with potassium >= 6.5 mmol/l irrespective of kidney function should be referred for immediate assessment and treatment
- All people with elevated potassium, acute illness, poor fluid intake and poor urine output should be assessed urgently and considered for admission
- http://www.renal.org/guidelines/joint-guidelines/treatment-of-acute-hyperkalaemia-in-adults

Factors prompting early assessment and admission

- AKI warning stage 3 result
- Any AKI in the context of raised potassium >6.0
- Any AKI and suspected urinary tract obstruction
- Any AKI and suspected intrinsic renal disease
- AKI and underlying CKD or Chronic Heart Failure
- Clinical deterioration irrespective of stage of AKI
- Dehydration not corrected in primary care
- AKI and repeat creatinine getting worse
- Lack of necessary support at home

Medication Review with AKI

- The term ‘nephrotoxic ‘ should be used with caution as few medications have a direct toxic effect on the kidney
- It may be difficult to restart a drug which a patient thinks is toxic!!
- Some medications may impair kidney function in the context of CKD and hypovolaemia
- Renally excreted medications may accumulate causing increased side effects or toxicity to other organs
- These medications may need dose reduction or suspension or blood dose monitoring

SAD MAN: Drugs to be aware of if patient is hypotensive and unwell

- S
- A
- D

- M
- A
- N

- G
- O
- N

SAD MAN GON

- Sulphonylureas
- ACE and ARB
- Diuretics

- Metformin
- Aldosterone antagonists
- NSAID

- Gabapentin
- Opiates
- NOAC

Recommendations for Medicines Management

	Effects on renal/fluid/electrolyte physiology	Change in the side effect profile when renal function is reduced	Action in presence of AKI
Analgesics			
NSAIDs / COX II inhibitors	Altered haemodynamics within the kidney leading to underperfusion and reduced glomerular filtration Acute interstitial nephritis (rare)		Avoid these agents in people at high risk of AKI
Opioid analgesics		Accumulation of active metabolites in AKI (especially morphine, pethidine and codeine) – increased incidence of CNS side effects & respiratory depression	Avoid long acting preparations. Reduce dose and frequency Use opiates with minimal renal excretion e.g. fentanyl, oxycodone, hydromorphone, tramadol
Pregabalin & Gabapentin		Accumulation leading to an increase in CNS side effects	Reduce dose
Cardiovascular Medications			
Antihypertensives (including Ca-channel blockers, α-blockers, β-blockers, etc)	Hypotension may exacerbate renal hypo-perfusion	Risk of bradycardia with Beta Blockers	Consider withholding / reduce dose depending on blood pressure
ACEI / ARBs / Aliskiren	Hypotension Hyperkalaemia		In some situations, e.g. heart failure continuing them might actually be helpful In AKI consider with holding
Diuretics including Thiazide & Loop Diuretics	Volume depletion Acute interstitial nephritis (rare)	Loop diuretics (furosemide & bumetanide) preferred as thiazides less effective if GFR < 25ml/min. However thiazides can potentiate the effects of loop diuretics	If volume depleted, consider with holding
Potassium sparing diuretics amiloride, eplerenone and spironolactone	Volume depletion Hyperkalaemia		Stop if AKI
Statins	May cause AKI if rhabdomylolysis is present	Increased risk of rhabdomyolysis	Stop if AKI due to rhabdomyolysis. Stop if patient develops unexplained / persistent muscle pain
Digoxin	Hyperkalaemia	May accumulate in AKI leading to bradycardia, visual disturbances, mental confusion	Reduce dose Monitor potassium and drug levels

Acute Kidney Injury - potentially problematic drugs and actions to take in Primary Care

'THINK KIDNEYS'

Direct Oral Anticoagulants		May accumulate leading to increased risk of bleeding. Routine blood testing does not detect those people at high risk of bleeding	Consider withholding, particularly agents with high renal clearance.
Drugs to treat infection			
Aciclovir	Crystal nephropathy Acute interstitial nephritis (rare)	Drug accumulates in reduced renal function leading to mental confusion, seizures	Reduce dose Encourage patient to drink plenty
Trimethoprim And co-trimoxazole	Increased risk of hyperkalaemia (especially in combination with spironolactone or ACEI/ARB) Interferes with tubular secretion of creatinine leading to a rise in serum creatinine without a true change in GFR	Accumulation increases risk of hyperkalaemia (particularly with high doses), nausea and vomiting	Avoid or reduce dose (particularly if patient is already taking an ACEI, ARB or spironolactone)
Diabetes medications			
Hypoglycaemic Drugs		Accumulation in AKI may increase risk of hypoglycaemia	Avoid long acting preparations. Monitor blood glucose levels & reduce dose if necessary
Metformin		Risk of lactic acidosis increased Accumulation leading to hypoglycaemia	Avoid if GFR < 30 ml/min
Other agents			
Colchicine		Diarrhoea / vomiting causing hypovolaemia	Use lower doses or consider steroids. Do not use NSAIDs for gout
Lithium	Can cause nephrogenic diabetes insipidus Very rarely it is associated with neuroleptic malignant syndrome.	Accumulation increases risk of side effects Kidney impairment exacerbated in hypovolaemia and in combination with ACE inhibitors / ARB / NSAIDs	Avoid where possible Monitor lithium and electrolyte levels Encourage patient to drink plenty.
Phenytoin	Acute interstitial nephritis (rare)	Risk of phenytoin toxicity if patient has low serum albumin levels	Monitor levels. Correct phenytoin levels for uraemia and low serum albumin

Acute Kidney Injury Risk

'THINK KIDNEYS'

- You have been given this card because you are **at risk** of acute kidney injury (AKI)
- Show this card to your pharmacist when collecting or buying any medication
- If you are admitted to hospital show it to the doctors and nurses

If you notice any of these problems, refer to the other side of the card

Not drinking as much as usual

Vomiting/diarrhoea

Infection

Dark concentrated urine

Decreased urine output

If the holder of this card becomes acutely ill or is admitted to hospital, check their kidney function as they are at risk of ACUTE KIDNEY INJURY.

STOP Acute Kidney Injury

'THINK KIDNEYS'

Do you notice any of the following problems?

Fluids	- are you having trouble drinking normally?
Look	- do you look ill or feel ill?
Urine	- are you passing much less urine than normal? - is your urine dark and concentrated?
Infection	- could you have an infection or fever?
Drugs	- are you on blood pressure or water tablets? - if yes, do you feel light-headed?

If you answer 'yes' to any of these questions, please seek advice – contact your GP or phone NHS 111.

Potential Risks

- Rules is a very rigid term
- How well can people judge the level of their ill health?
- What do they understand about kidney function?
- Do they know when to restart?
- Are they at risk of decompensated heart failure or uncontrolled BP?
- Will they restart drugs they think are ‘nephrotoxic’?
- Will they fail to call for help?
- Could diabetes control deteriorate?
- How can they do this with a dossette box?

See Position Statements on 'Think Kidneys' Website

- Recommend individualised advice to those at high risk when they are unable to eat and drink
- Cards given by professional NOT help yourself
- NOT RULES but GUIDANCE
- Important to restart medications when patient and kidney function stable because of risk of uncontrolled BP, diabetes and heart failure
- Avoid using 'Nephrotoxic' for ACEi and ARB as all antihypertensive medication can exacerbate AKI including diuretics and this may reduce compliance/concordance in the future

'THINK KIDNEYS'

BRITISH KIDNEY Patient ASSOCIATION

improving life for kidney patients

RCGP Royal College of General Practitioners

Acute Kidney Injury Patient Leaflet

Who is this for?

This leaflet is for people who have been told that they have had Acute Kidney Injury and it's been designed to answer questions you may wish to ask about this condition

How do doctors know that I have had Acute Kidney Injury?

You have probably had a recent illness where you were unable to drink or eat properly, for example gastroenteritis, or after an operation. Your blood pressure may have been low and you had low urine output.

Take Home Messages

- Treat the patient not the test result
- Put the test result into clinical context
- What is the stage of AKI ?
- What is the potassium ?
- Think Cause
- Think Medication
- Think Fluids
- Think Review
- Can you correct things at home?
- Are they supported?
- Do they need admission?