



# Acute Kidney Injury

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**GP education event**

**2 September 2017**



# AKI-Why does it matter

- › AKI is associated with 1 in 5 emergency hospital admissions
- › Higher incidence in Intensive care units with in hospital mortality ranging from 20%-50%
- › Associated with increased morbidity and mortality in the short and long term
- › Associated with higher risk of chronic kidney disease
- › Of all people with AKI up to 66% develop it in the community



# Stages of AKI

Stage	Serum creatinine	Urine output
<b>1</b>	Increase by greater than or equal to 26 micromol/Litre within 48 hours <b>OR</b> 1.5-1.9 times baseline	Less than 0.5 mL/kg/hour for 6-12 hours
<b>2</b>	2-2.9 times baseline	Less than 0.5 mL/kg/hour for 12 hours or more
<b>3</b>	3 times baseline <b>OR</b> Increase to greater than or equal to 354 micromol/Litre <b>OR</b> Decrease in eGFR to less than 35 mL/min/1.73m <sup>2</sup> in children and young people <b>OR</b> Initiation of renal replacement therapy	Less than 0.3 mL/kg/hour for 24 hours or more <b>OR</b> No urine output for 12 hours or more



# Stages Chronic Kidney diseases

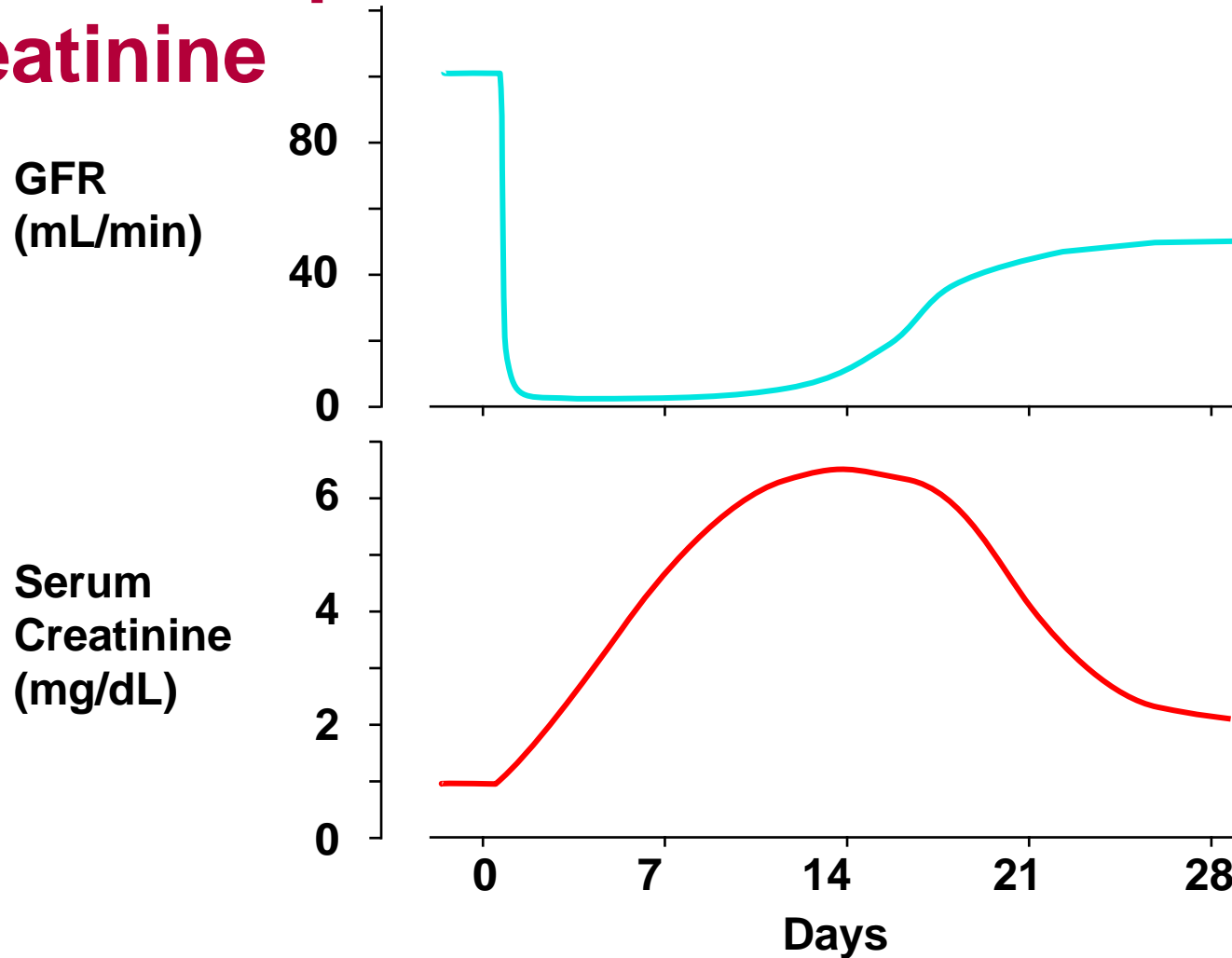
Prognosis of CKD by GFR and albuminuria category

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (ml/min/1.73 m <sup>2</sup> ) Description and range	G1	Normal or high	≥90	Green	Yellow	Orange
	G2	Mildly decreased	60-89	Green	Yellow	Orange
	G3a	Mildly to moderately decreased	45-59	Yellow	Orange	Red
	G3b	Moderately to severely decreased	30-44	Orange	Red	Red
	G4	Severely decreased	15-29	Red	Red	Red
	G5	Kidney failure	<15	Red	Red	Red

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red, very high risk.

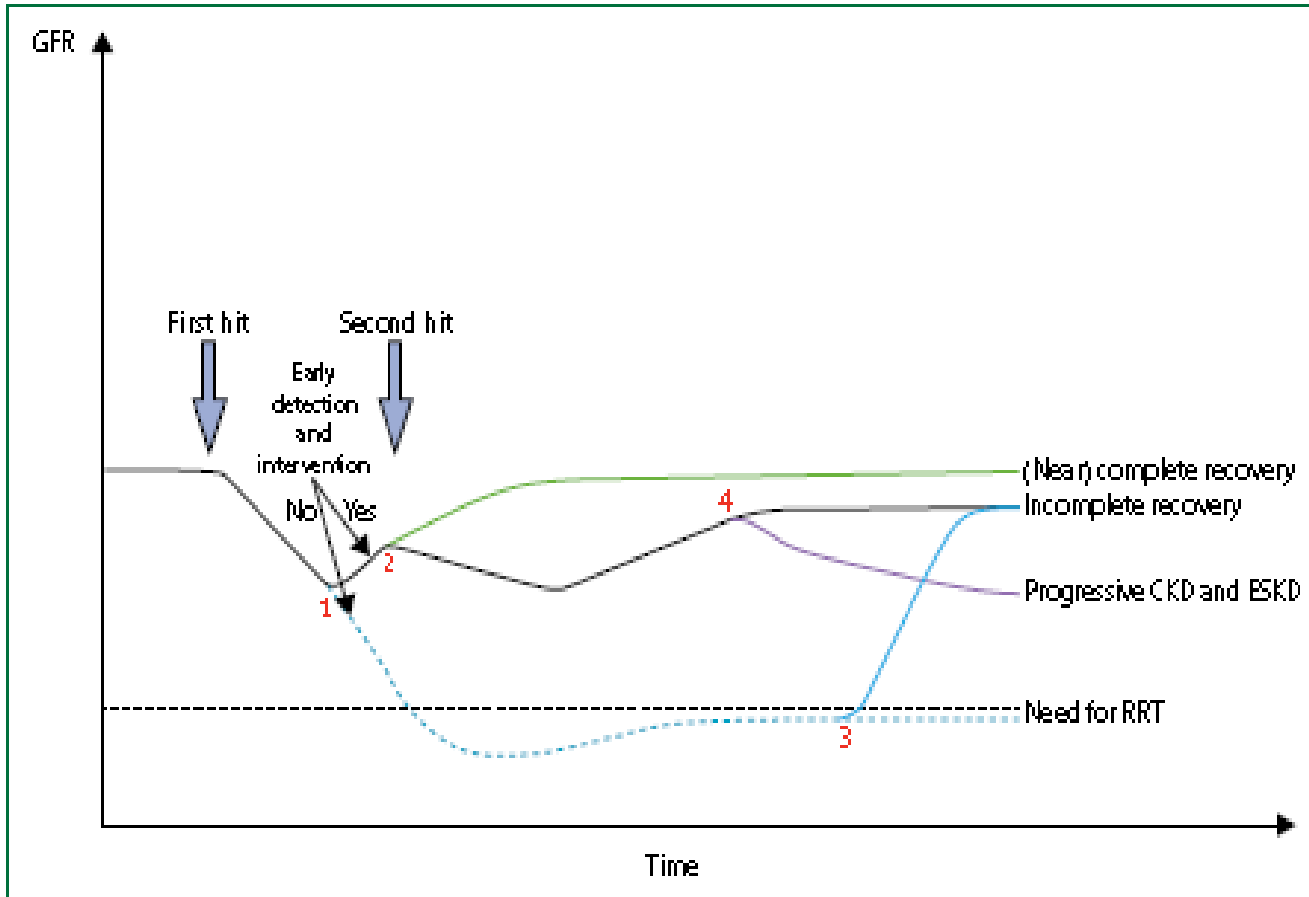


# Relationship Between GFR and Serum Creatinine





# Multiple Hit Theory



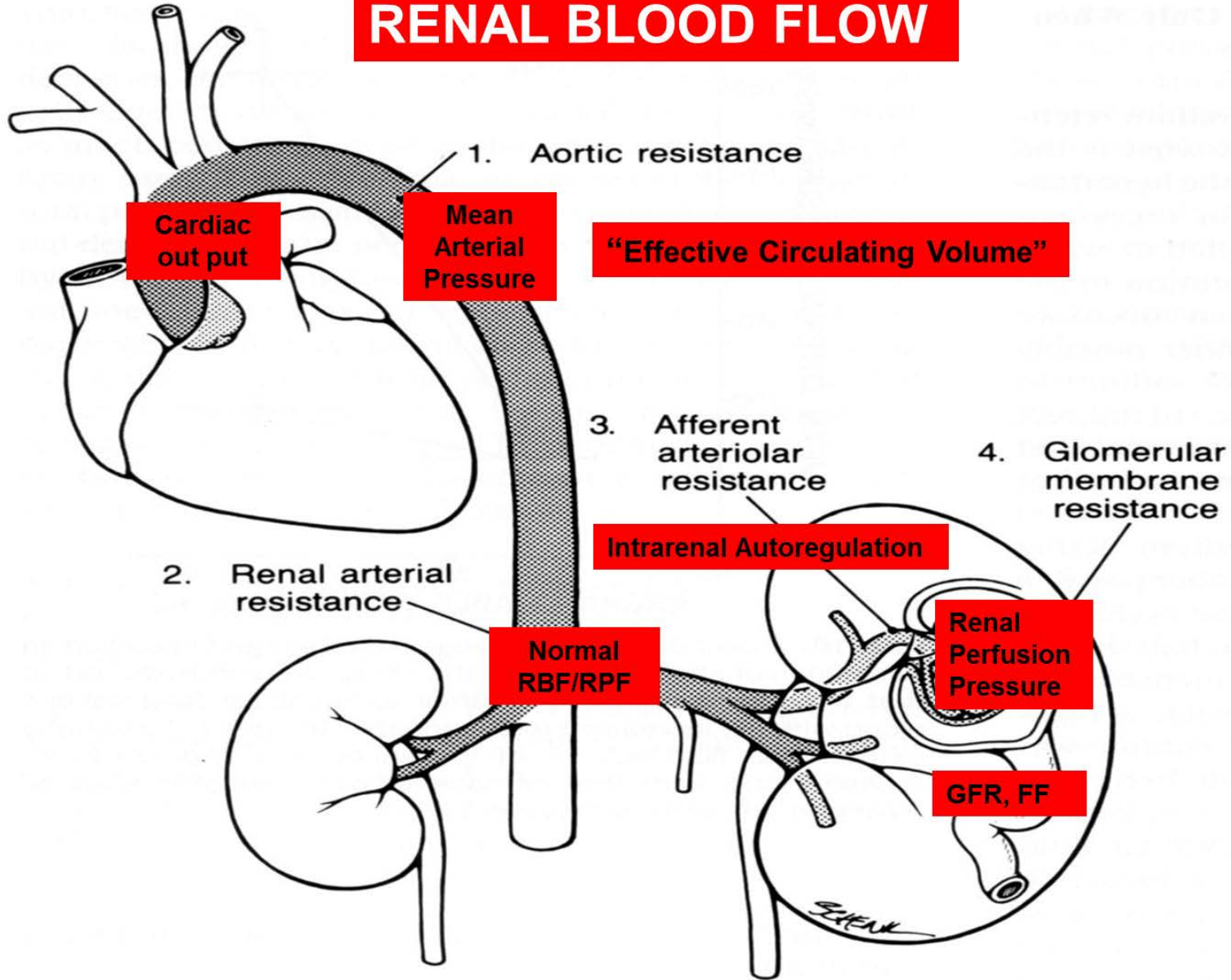


# Normal Renal Requirement

- › Normal Renal Blood flow
- › Functioning Glomeruli and tubules
- › Clear Urinary outflow tract



# RENAL BLOOD FLOW



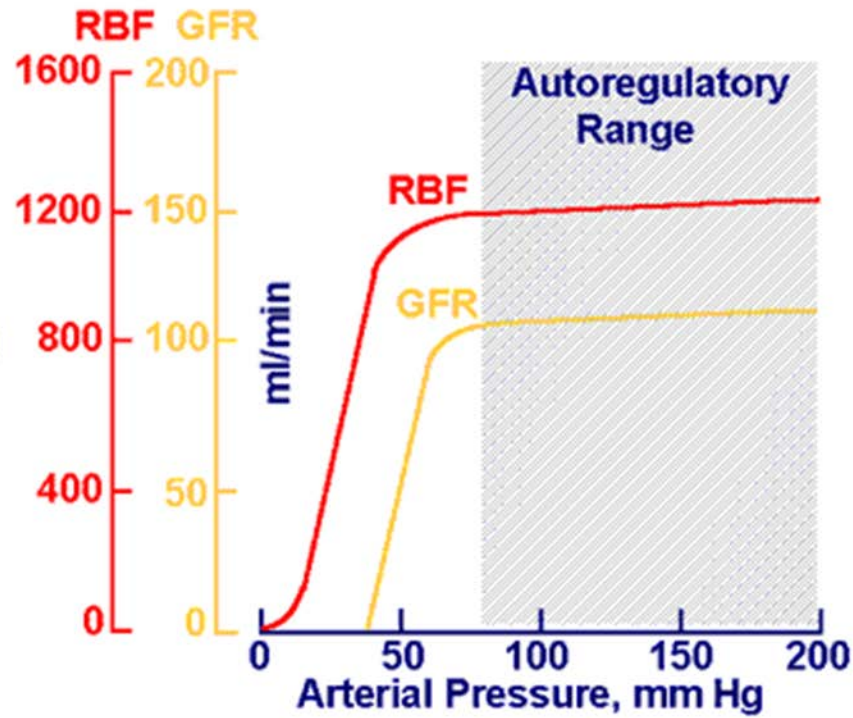




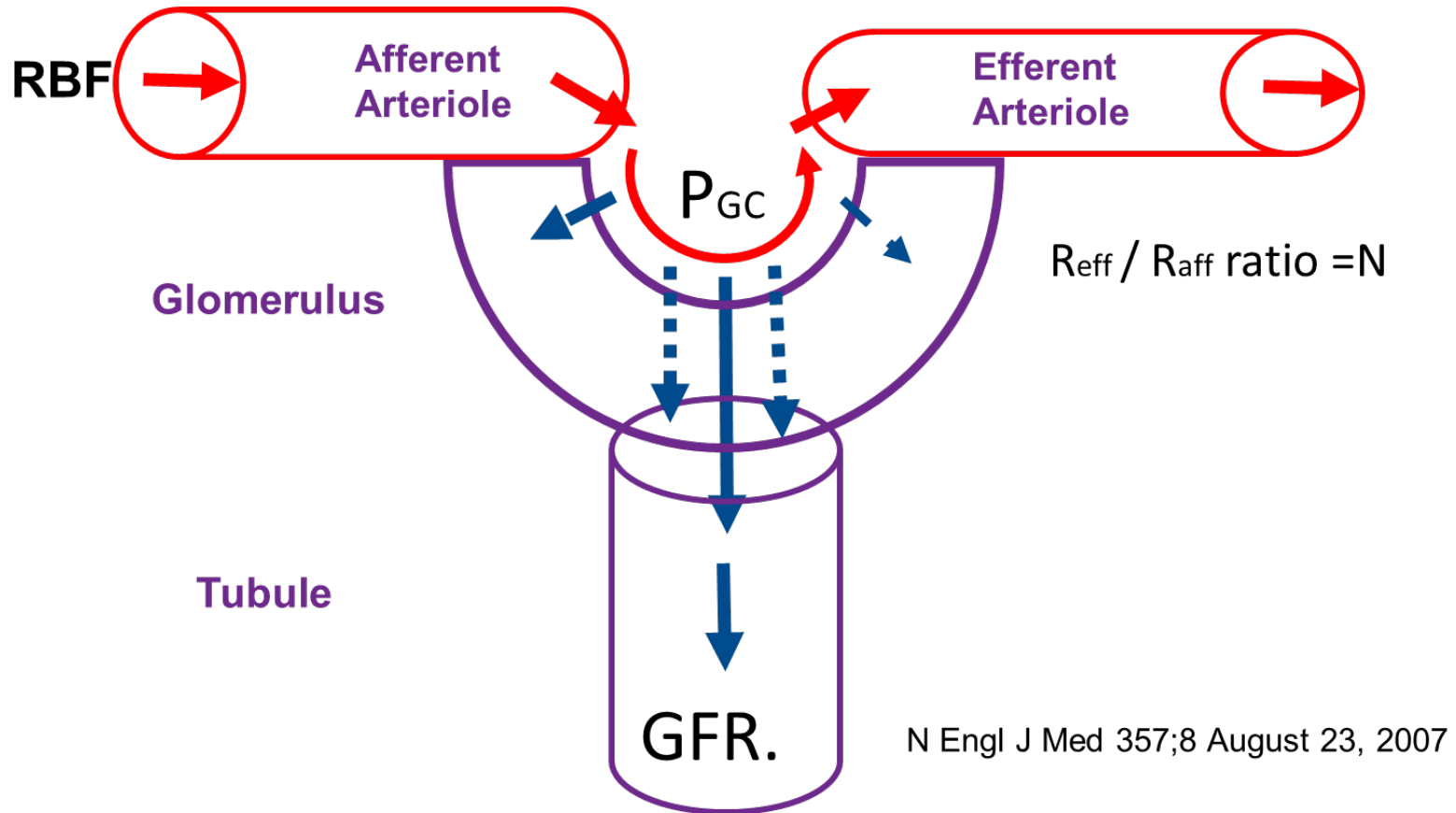
# Understanding Autoregulation

## AUTOREGULATION

In the absence of external neural and humoral factors, the kidney will maintain GFR and RBF relatively constant in the face of changes in arterial pressure within the range of 80-200 mm Hg.



# Intrarenal Mechanisms for Autoregulation



**Figure - shows normal conditions normal renal perfusion pressure and a normal GFR.**

# Intrarenal Mechanisms for Autoregulation under decreased Perfusion Pressure

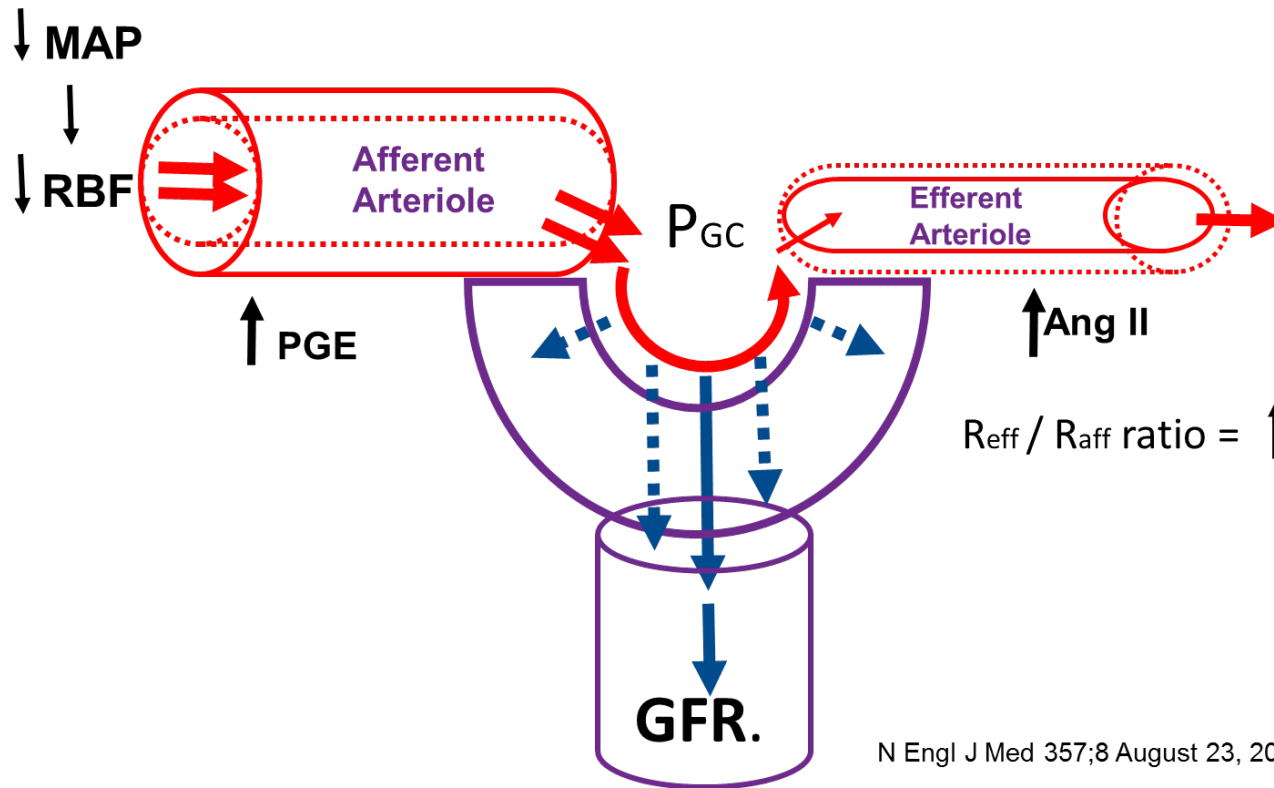


Figure: shows reduced perfusion pressure within the autoregulatory range. Normal glomerular capillary pressure is maintained by afferent vasodilatation and efferent vasoconstriction.



# Risk Factors

- › Who is at risk
- › When do people sustain AKI
- › How should patients with AKI be managed
- › What do people need to know
- › Vulnerability
- › Trigger
- › Response



# The Workshop's Aim to Understand

## Fixed Factors

- › The elderly
- › The Frail
- › Existing Comorbidities
- › Chronic Kidney disease
- › Previous history of AKI

## Modifiable risk factors

- › Drugs
- › Diuretics-Volume status
- › NSAID-Autoregulation
- › ACE/ARB and other BP medications –BP and autoregulation
- › Metformin side effect enhanced



# Diagnosis Investigations and Management of Case studies at Workshop

- › Case Study 1
- › Case Study 2



# Management Plan - Factors prompting early assessment and review

- › AKI stage 3
- › Any AKI with hyperkalaemia
- › Any AKI with suspected intrinsic kidney disease/suspected obstruction
- › AKI with underlying CKD or chronic heart failure
- › Dehydration/AKI not corrected in primary care
- › Lack of necessary support at home/community



# Suggested Management

- › Fluid – Check for signs of dehydration. Encourage oral fluid and assess need for support for hydration
- › With history and bladder inspection-exclude obstruction (catheterisation if appropriate)
- › Low BP –Check and withhold antihypertensive and diuretics(If history of angina/arrhythmia reduce dose rather than cease)





# Suggested Management

## › Withhold drugs and Toxins

ACE/ARB/NSAID/PPI/Antibiotics

## › Urine Microscopy –

Consider Intrinsic kidney disease if  
protein& blood in urine in absence of  
UTI/trauma



# Suggested Management Plan

- › If patient unwell and not responding clinically, or remains oliguric, requires IV therapy or high suspicion of intrinsic kidney disease refer to ED unless Advance Directive states otherwise.
- › If patient is well continue essential steps
- › Repeat UEC 24 -48 hours
- › Renal screen if haematuria/proteinuria in absence of UTI/trauma
- › Discuss with Renal on call or Acute Medical unit



# Post Recovery

- › Review Medications before restarting any potential nephrotoxics
- › Consider
  - Is it required
  - Is the patient suitable for restart
  - What is the clinical or biochemical risk vs benefit
- › UEC should be checked within 1-2 weeks after restarting any potential nephrotoxic medication
- › Discuss future risk reduction with patient /carers





# AKI Sick day guidance

## AKI -Who is at risk

- › CKD/history of AKI
- › Hypovolemia/sepsis
- › History of diabetes, heart failure, liver disease
- › Elderly
- › Use of drugs with potential for nephrotoxicity
- › Iodinated contrast use/surgery

## When to consider

- › Febrile illness
- › Protracted vomiting, diarrhoea
- › Dehydration



# AKI Sick day guidance

## What is the guidance

- › Stay Hydrated
- › At least 1500 ml of fluid a day(unless advised otherwise)
- › If Vomiting persists slow sips of water/fluid till symptoms have settled
- › Reduce or avoid Alcohol consumption

## Cease Nephrotoxics

- › NSAID
- › ACE
- › ARB
- › Diuretics
- › Metformin



# Key Points

- › AKI is a multifaceted syndrome that occurs in different settings.
- › The course of AKI can be variable from a single hit and complete recovery to multiple hits resulting in End stage renal disease.
- › No interventions to improve outcomes of established AKI have been developed so prevention and early diagnosis are key.
- › Awareness campaign and education for health professionals on diagnosis and management of AKI with attention to avoidance of volume depletion , hypotension and nephrotoxic interventions improve outcomes.
- › Electronic early warning systems where available can improve outcome

