Supportive care of the AKI patient

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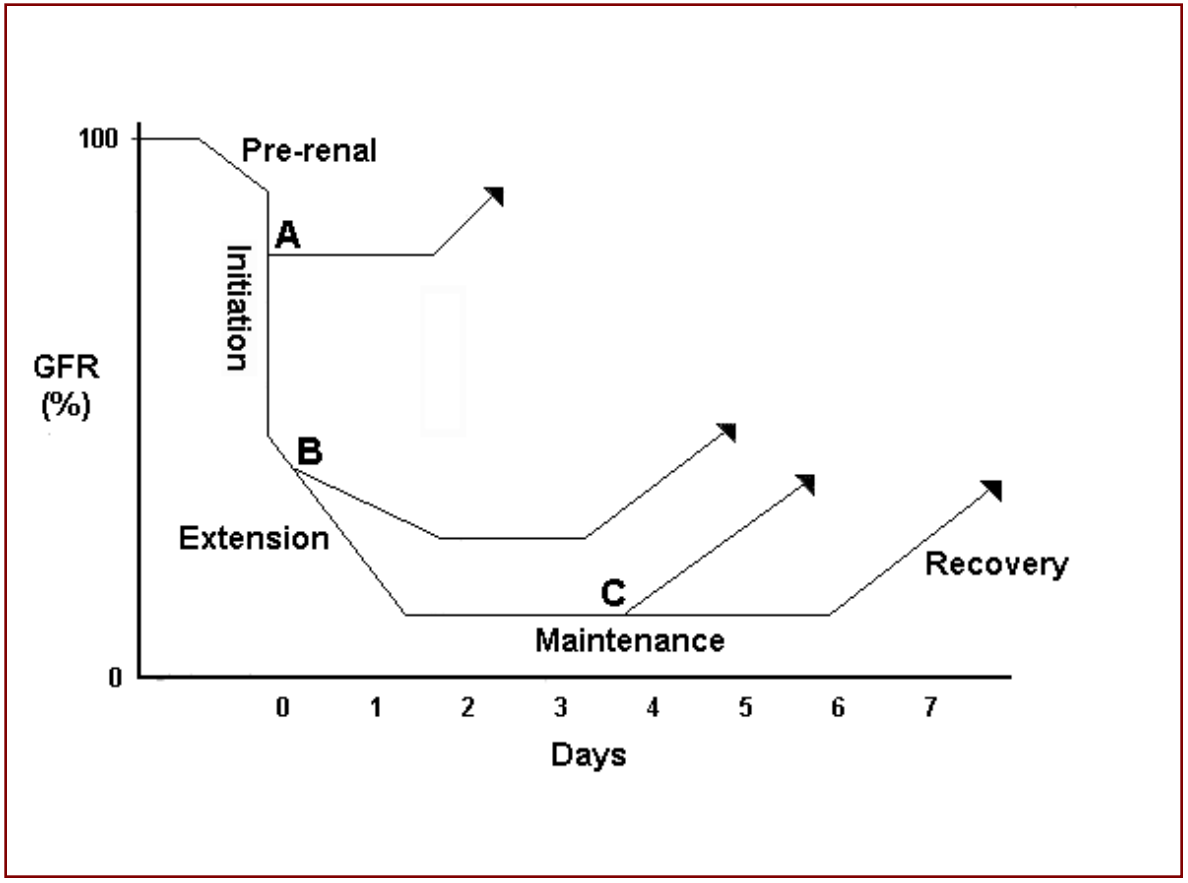
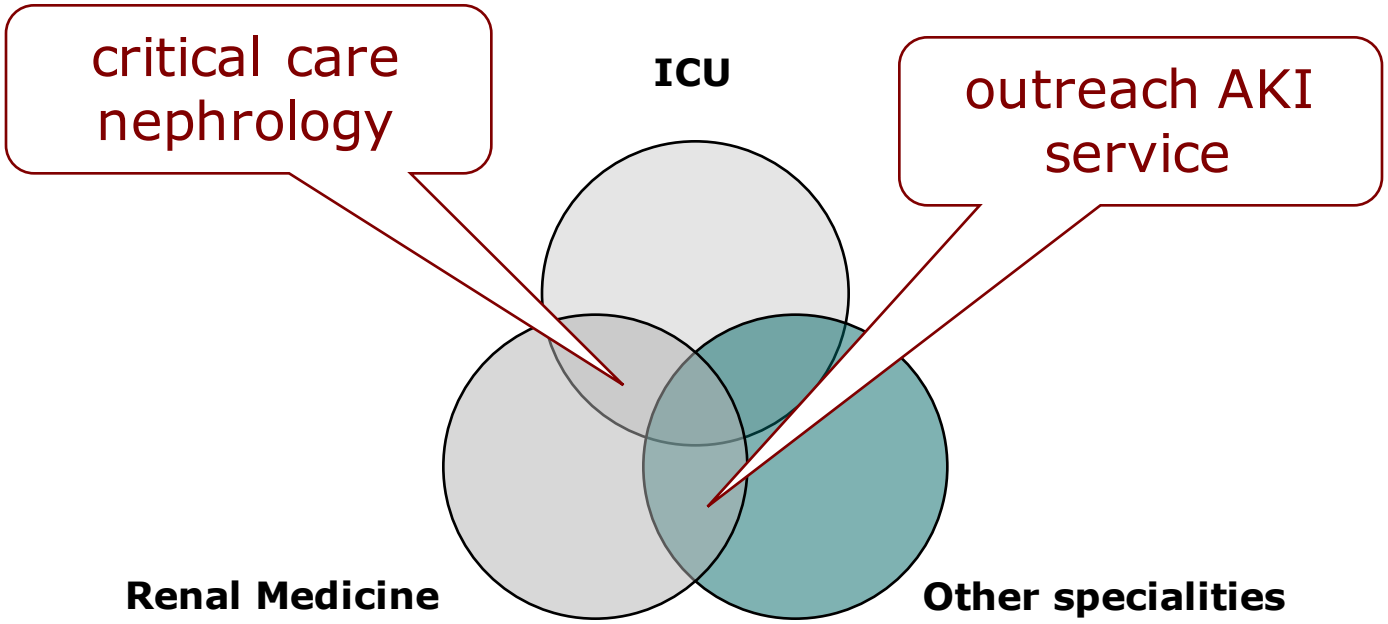
The Newcastle upon Tyne Hospitals NHS
NHS Foundation Trust

Phases of ischaemic AKI

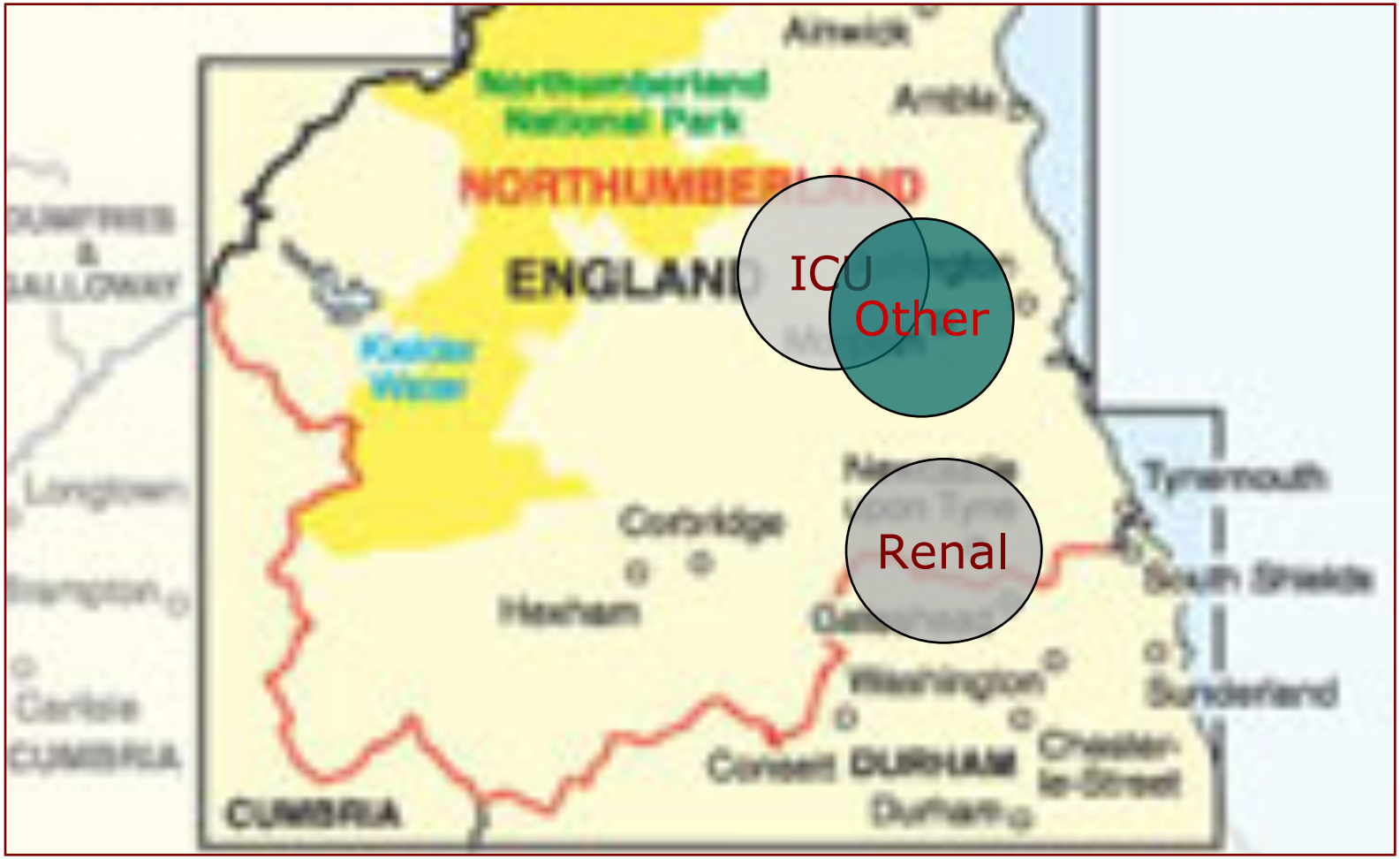


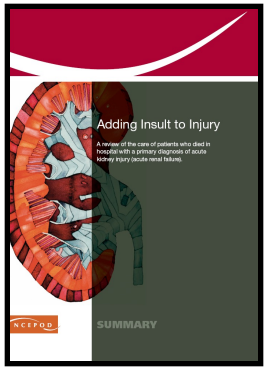
Molitoris, B. A. (2003) *J Am Soc Nephrol,* **14,** 265-7.

Venue


critical care nephrology
ICU
outreach AKI service
Renal Medicine
Other specialities

Venue


Alnwick
Northumberland National Park
Amble
NORTHUMBERLAND
ENGLAND
ICU
Other
Renal
Tynemouth
Corbridge
Hexham
Washington
Sunderland
Consett
DURHAM
Chester-le-Street
Durham
CUMBRIA



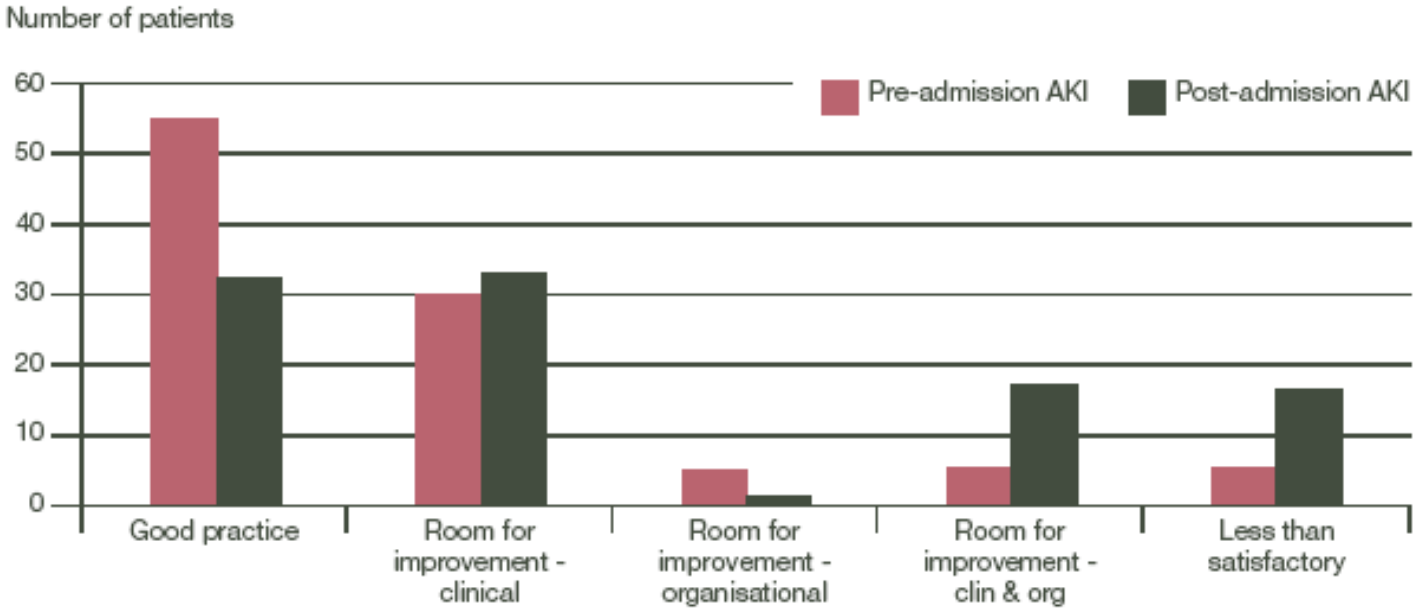


Figure 3.2 Overall assessment of care (pre-admission vs post-admission AKI)

n = 457 and 107 for pre- and post-admission AKI respectively

Sub-optimal delivery of renal aspects of care after step-down from critical care to non-specialist wards

TJ Hardy[1], AL Rhodes[1], N Kaudeer[1], SE Wright[2], SV Baudouin[2,3], NS Kanagasundaram[1]

Evaluation against markers of good renal practice:

- daily fluid balance, weights, regular bloods
- safe prescribing (avoidance of nephrotoxins; dose adjustments)
- long-term follow-up of persisting renal impairment

Results

N = 94 patients (including 6 chronic HD)

- 1 in 5 had daily fluid balance
- 1 in 5 had a clinical assessment of volume status
- 1 in 50 had daily weights

- at hospital discharge, 45% had stage 3 or 4 CKD - only 2 patients had documented plans for CKD management

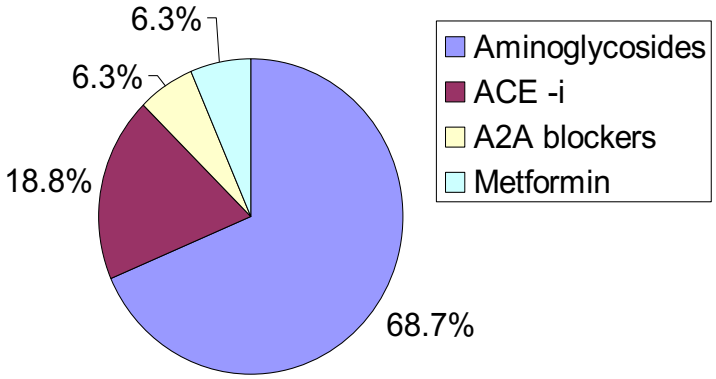
- of the 55% supported from **nephrology outreach service**, only 25% received daily fluid balance monitoring

Renal Association, annual meeting, 2008

Sub-optimal delivery of renal aspects of care after step-down from critical care to non-specialist wards

TJ Hardy[1], AL Rhodes[1], N Kaudeer[1], SE Wright[2], SV Baudouin[2,3], NS Kanagasundaram[1]

- 1 in 6 were prescribed at least one class of potentially nephrotoxic drug



- 44.4% were on doses of drugs inappropriate to their level of kidney function

- A decision on the use of radiocontrast prophylaxis was rarely documented in those to whom this applied

Renal Association, annual meeting, 2008

What stops the “very basics of care” being delivered?

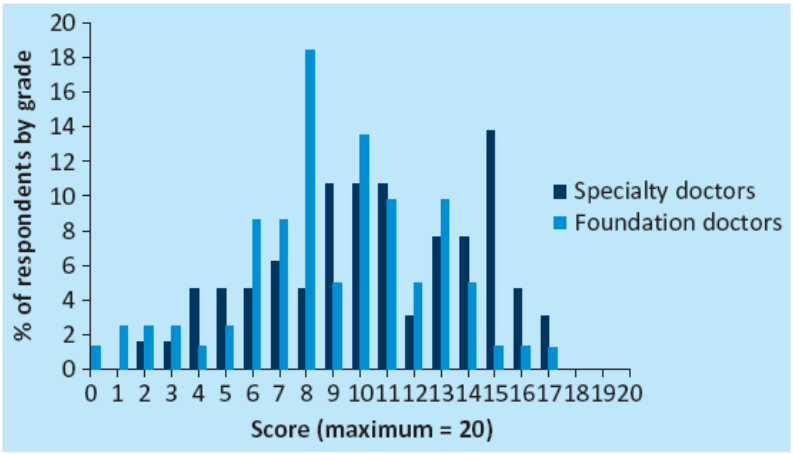


Fig 1. Questionnaire scores broken down by grade.

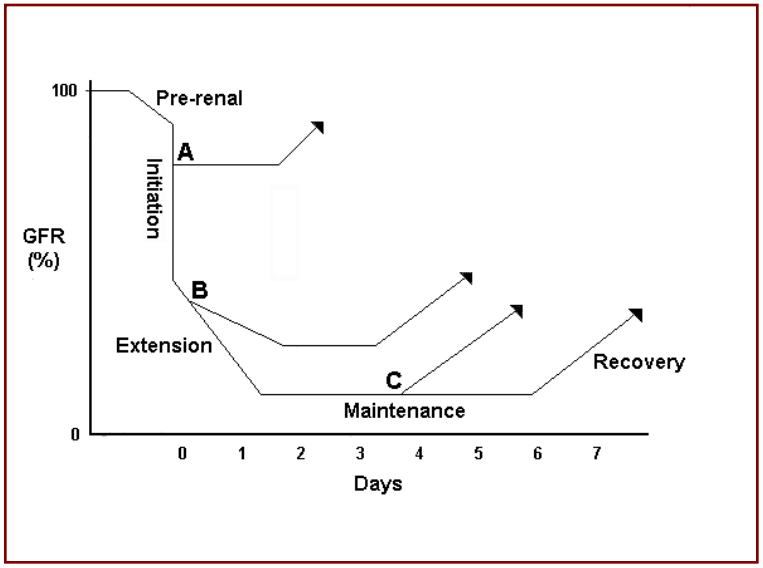
•40% could not name even 1 indication for seeking renal advice

• 35% said they had never had teaching on AKI

Clinical Medicine 2012, Vol 12, No 3: 216–21

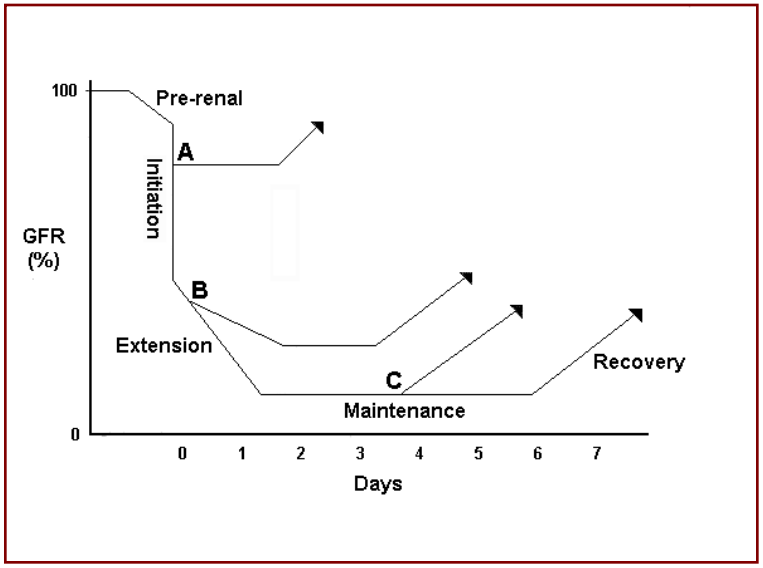
- Competing pressures
- Continuity of care

Supportive care of the AKI patient



- **Haemodynamic support**
  - Vulnerability of the acutely injured kidney
  - Volume assessment, fluid therapy, haemodynamic monitoring
- **Nutritional support**
- **Prescribing**
- **Esoteric causes**
- **Transfer of care**
- **Long term follow up**
- **Monitoring**
  - Biochemistry, haematology
  - Clinical variables: urine output, haemodynamic indices (non-invasive, invasive), fluid balance, daily weights, regular clinical assessment of volume status
  - Vigilance for sepsis

Supportive care of the AKI patient



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Haemodynamic support

pre-renal azotaemia vs. ischaemic AKI

Pre-renal azotaemia → **ischaemic AKI**

renal cell injury

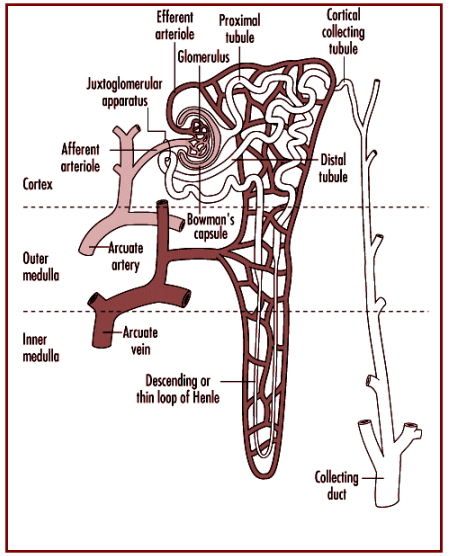
rapid restoration of excretory capacity

< 20	urine Na^+	> 40 mmol/L
< 1%	$FENa^+$	> 2%

Haemodynamic support
regional alterations in renal blood flow

- Decrease in total renal blood flow, alone, does not entirely account for reduction in GFR
- Regional alterations in RBF are probably more important
- Outer medullary blood flow is reduced disproportionately in experimental models
  - local oedema
  - structurally vulnerable capillary network

  compound…….
  - relative outer medullary hypoxia in health



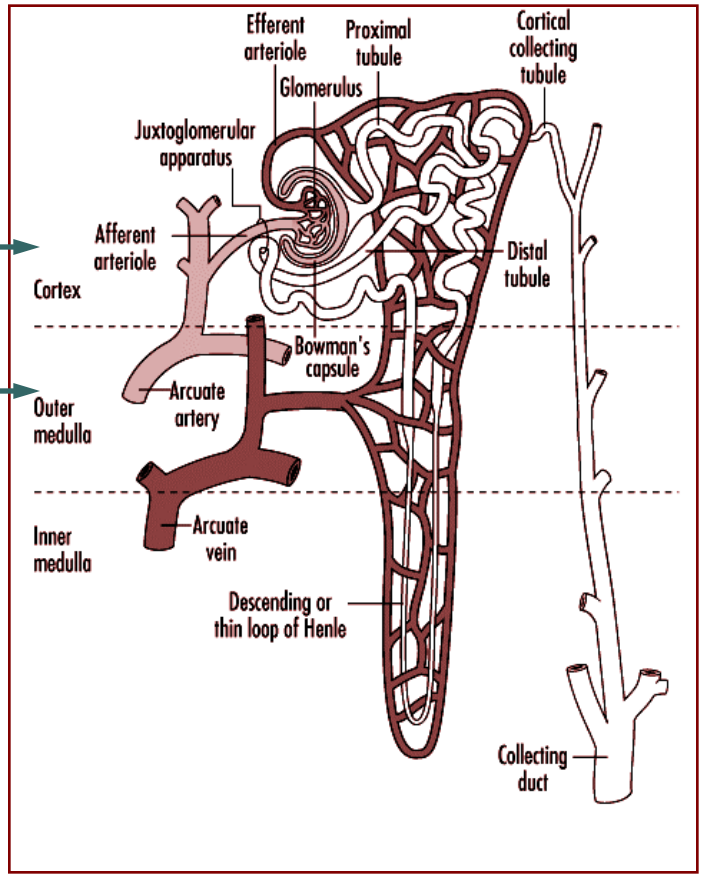
Haemodynamic support

relative outer medullary hypoxia

25% cardiac output

PO_2 6.65 – 13.3 kPa

PO_2 1.3 – 2.9 kPa



Haemodynamic support
renal auto-regulation

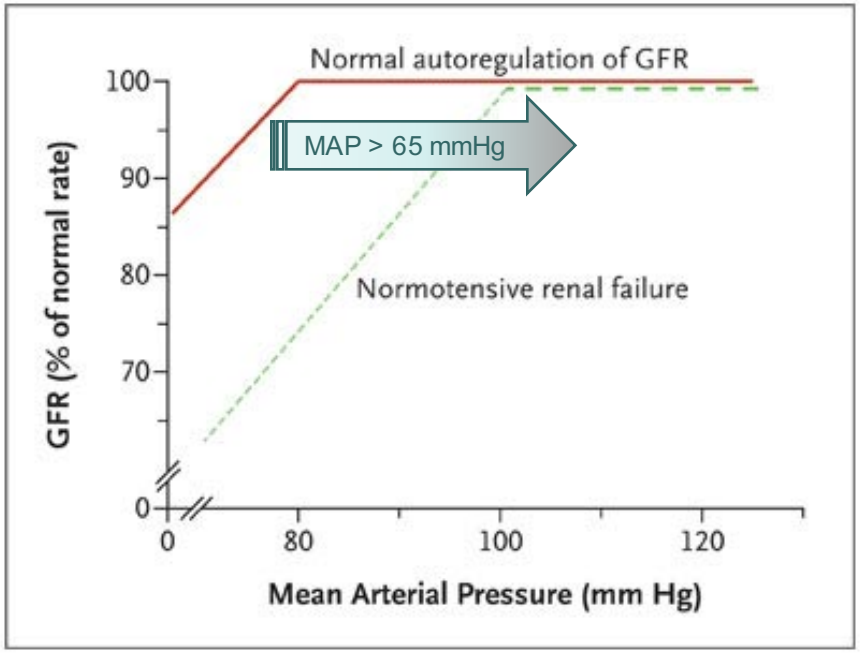


Table 1. Factors Increasing Susceptibility to Renal Hypoperfusion.

Failure to decrease arteriolar resistance

Structural changes in renal arterioles and small arteries
- Old age
- Atherosclerosis
- Chronic hypertension
- Chronic kidney disease
- Malignant or accelerated hypertension

Reduction in vasodilatory prostaglandins
- Nonsteroidal antiinflammatory drugs
- Cyclooxygenase-2 inhibitors

Afferent glomerular arteriolar vasoconstriction
- Sepsis
- Hypercalcemia
- Hepatorenal syndrome
- Cyclosporine or tacrolimus
- Radiocontrast agents

Failure to increase efferent arteriolar resistance

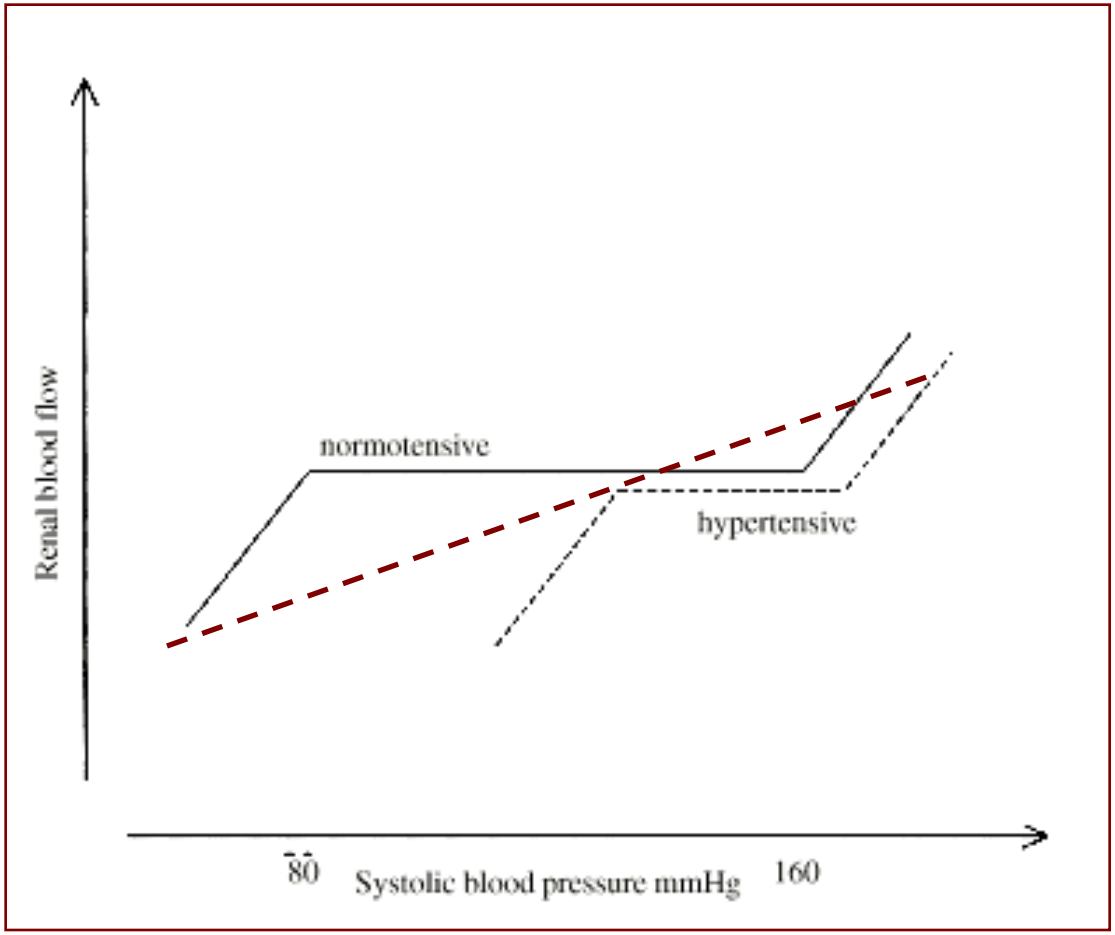
Angiotensin-converting–enzyme inhibitors
Angiotensin-receptor blockers

Renal-artery stenosis

From Abuelo, J.G. *NEJM* 2007: 357(8):797

Haemodynamic support

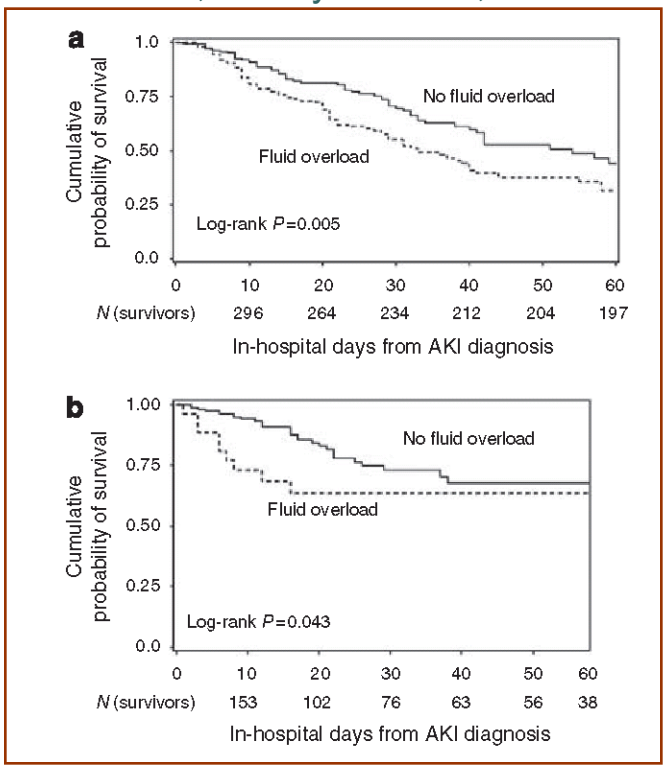
disrupted renal auto-regulation



Haemodynamic support
fluid therapy

A positive fluid balance is associated with increased mortality in AKI:

Bouchard J, Kidney Int 2009; 76: 422–427:

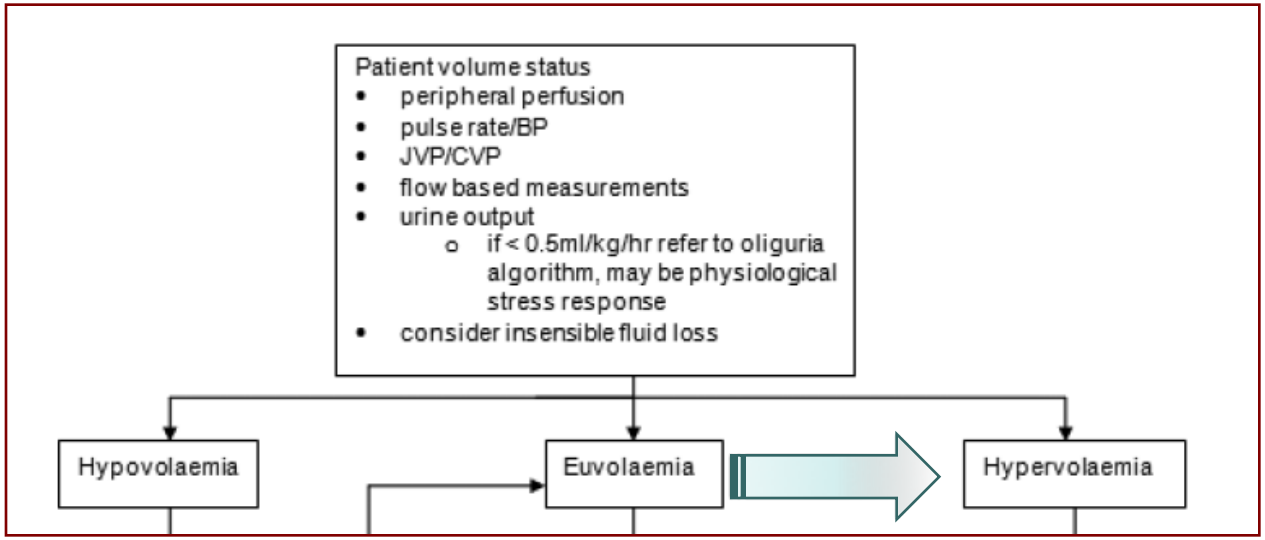


Fluid overload status at RRT initiation

Fluid overload status at AKI diagnosis (non-RRT patients

Also: Payen D, de Pont AC, Sakr Y, et al. A positive fluid balance is associated with a worse outcome in patients with acute renal failure. Crit Care 2008; 12: R74.

Haemodynamic support
fluid therapy



Assessment **(assessment, assessment)**

MAP 65 mmHg **(???)**

Stability **(avoid fluctuations)**

Judicious use of vasopressors

Haemodynamic support
diuretics

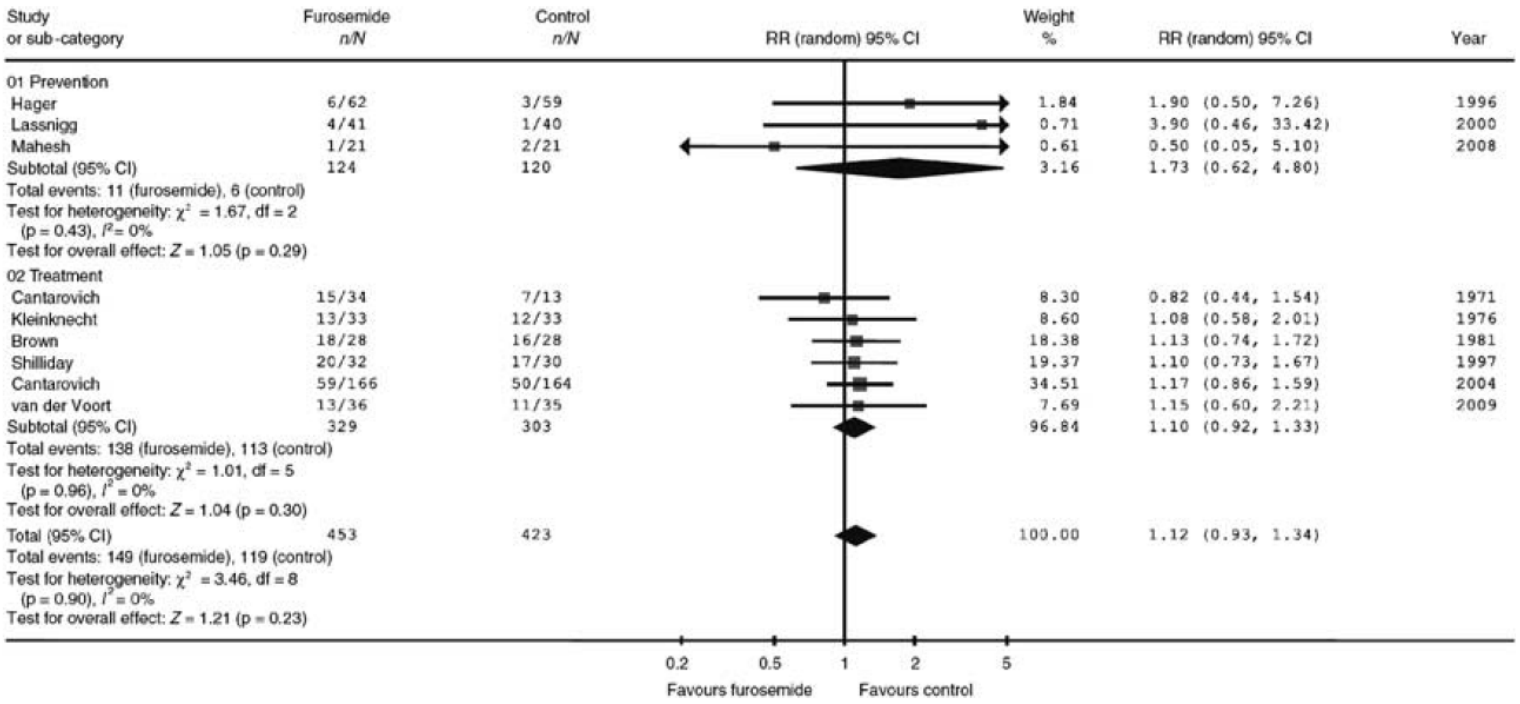


Figure 9 | Effect of furosemide vs. control on all-cause mortality. Reprinted from Ho KM, Power BM. Benefits and risks of furosemide in acute kidney injury. Anaesthesia 2010; 65: 283–293 with permission from John Wiley and Sons[193]; accessed http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2009.06228.x/full

Haemodynamic support
diuretics

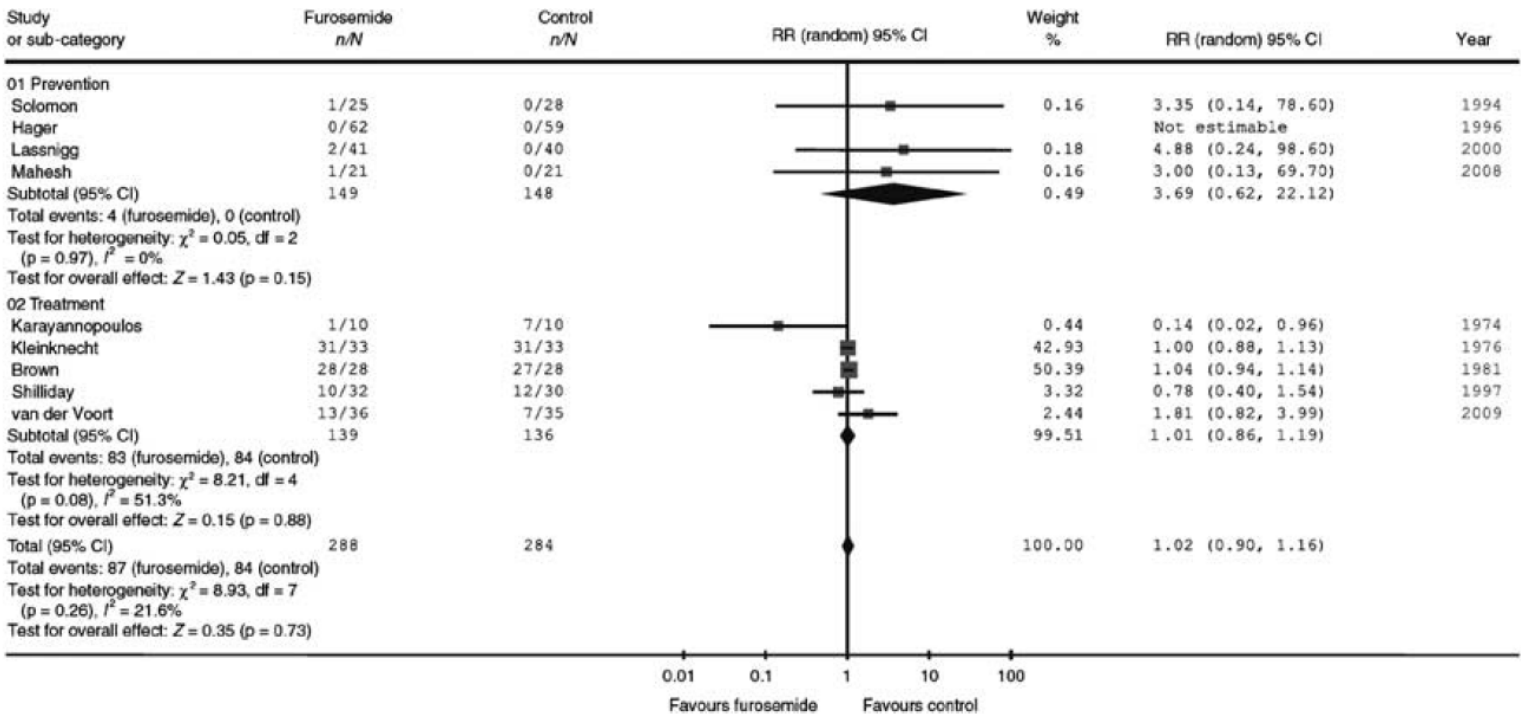


Figure 10 | Effect of furosemide vs. control on need for RRT. Reprinted from Ho KM, Power BM. Benefits and risks of furosemide in acute kidney injury. Anaesthesia 2010; 65: 283–293 with permission from John Wiley and Sons[193]; accessed http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2044.2009.06228.x/full

Haemodynamic support
fluid management

BP and volume control – salt and water management

Review dialysate sodium prescription	Reduce dialysate sodium towards usual pre-dialysis sodium Avoid sodium profiling
Review dietary salt intake	Reduce daily sodium intake to 80 – 100 mmol (a 'no added salt' diet)
Review fluid restriction	Aim for inter-dialytic weight gain ≤ 3% 'active' body weight • If non-obese, active and actual body weight are pragmatically equivalent • If obese, active body weight = 25*height^2 (weight in kg, height in meters; 'fat-free' BMI assumed as 25)

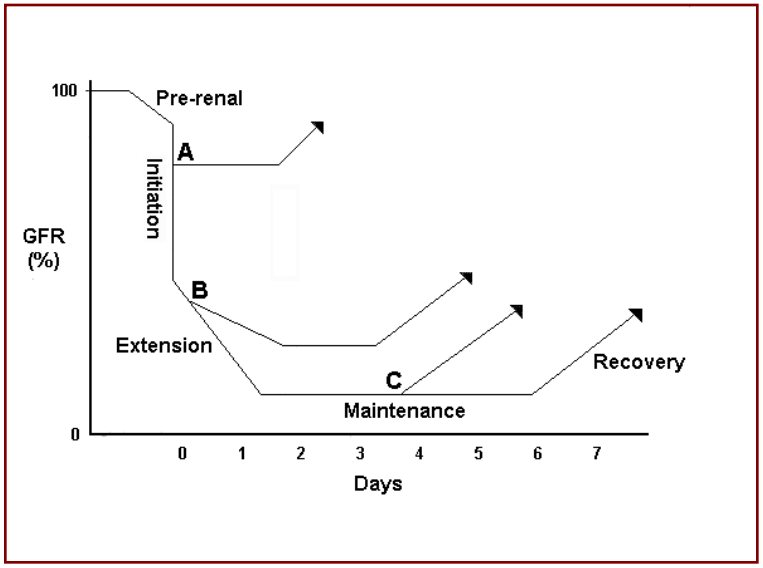
Haemodynamic support
fluid management

BP and volume control – salt and water restriction and patient education

Emphasise key role of sodium in hypertension, volume overload, thirst
Differentiate between sensations of thirst and dry mouth
Review drugs that may be contributing to dry mouth
Patient tips and tricks to manage restrictions:

- check sodium content of processed food
- take tablets with food where possible
- use small volume cups
- sip and savour, don't gulp
- use ice cubes, ice lollies, sweets or gum (n.b. sugar content), mouth washes, artificial saliva
- use pre-filled measuring jug to help guide daily fluid intake

Supportive care of the AKI patient



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Nutritional support
maintenance and replacement

Protein-calorie malnutrition is common in patients with AKI, affecting 42% at admission to one renal unit

Fiaccadori, E. J Am Soc Nephrol 1999; 10: 581–593.

AKI:

- is pro-inflammatory
- is often associated with hypercatabolism:
  normalised protein catabolic rate (nPCR) ~ 1.8 g/kg/day
  (nPCR ~1.2 in steady state chronic HD – largely represents dietary intake)
- may require RRT which contributes through:
  nutrient losses (water-soluble essential nutrients e.g. 10 – 15 g amino acids / day on CRRT
  induction of hypercatabolic state (8 – 10 g protein lost per IHD session)

Compounded by:

- under-prescription of nutrient replacement

Sparse literature

Critical care literature**:** enteral > parenteral; early initiation if possible; re-feeding syndrome

Nutritional support
maintenance and replacement

Carbohydrate metabolism
AKI is associated with hyperglycaemia

We suggest achieving a total energy intake of 20–30 kcal/kg/d in patients with any stage of AKI (2C)

Administer, predominantly, as carbohydrate due to inhibited lipolysis in critical illness

Res)%
of n

Suf sis)

Nea
kca

Macias, W.L. J. Ren. Nutr. Enter. 20: 56

No difference in nitrogen balance with 30 vs. 40 kcal/kg/day b e
produced more hyperglycaemia and hypertriglyceridaemia:

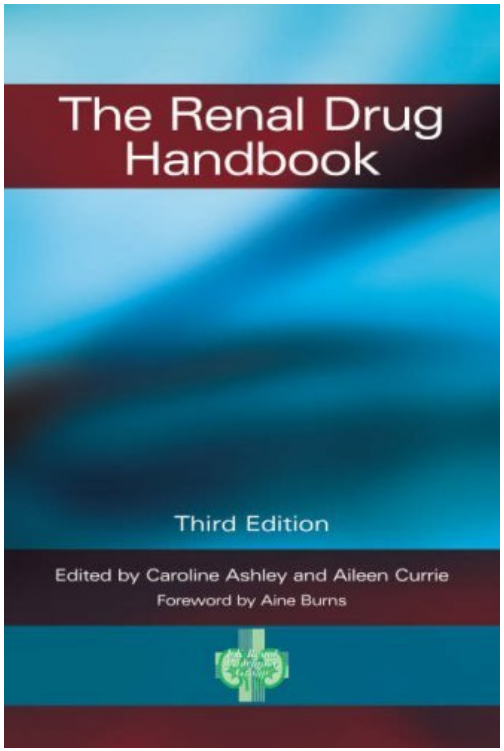
Fiaccadori, E. Nephrol Dial Transplant 2005; 20: 1976

Nutritional support
maintenance and replacement

We suggest protein supplementation of:

- 0.8–1.0 g/kg/day in non-dialysis requiring, non-catabolic AKI patients
- 1.0–1.5 g/kg/day in patients with AKI on RRT
- up to a maximum of 1.7 g/kg/day in patients on CRRT / who are hypercatabolic

Supportive care of the AKI patient



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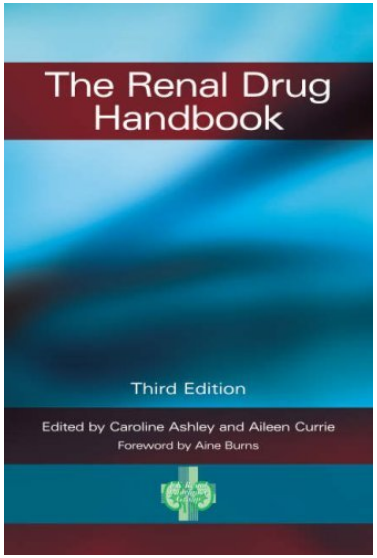
Prescribing

nephrotoxins and other drugs potentially deleterious to renal function

- Justify initiation or continuation, e.g.:
  - RAS modifying agents
  - Radio-contrast
  - Aminoglycoside antibiotics
- Seek alternatives if possible

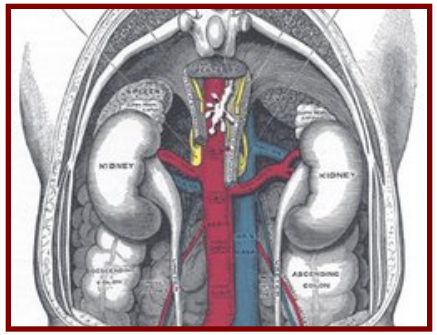
Prescribing
dose adjustment in renal support

- Drug dosing in renal support
- Advice broken down according to RRT technique, too

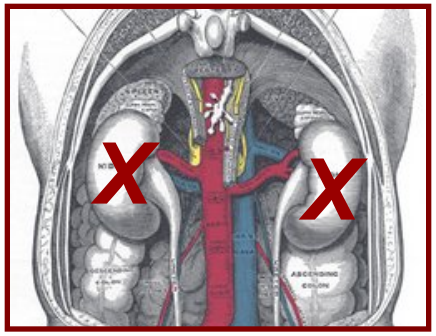


Prescribing

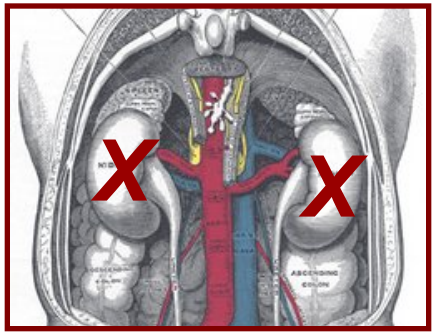
dose adjustment – AKI patients independent of RRT and assumptions about CrCl



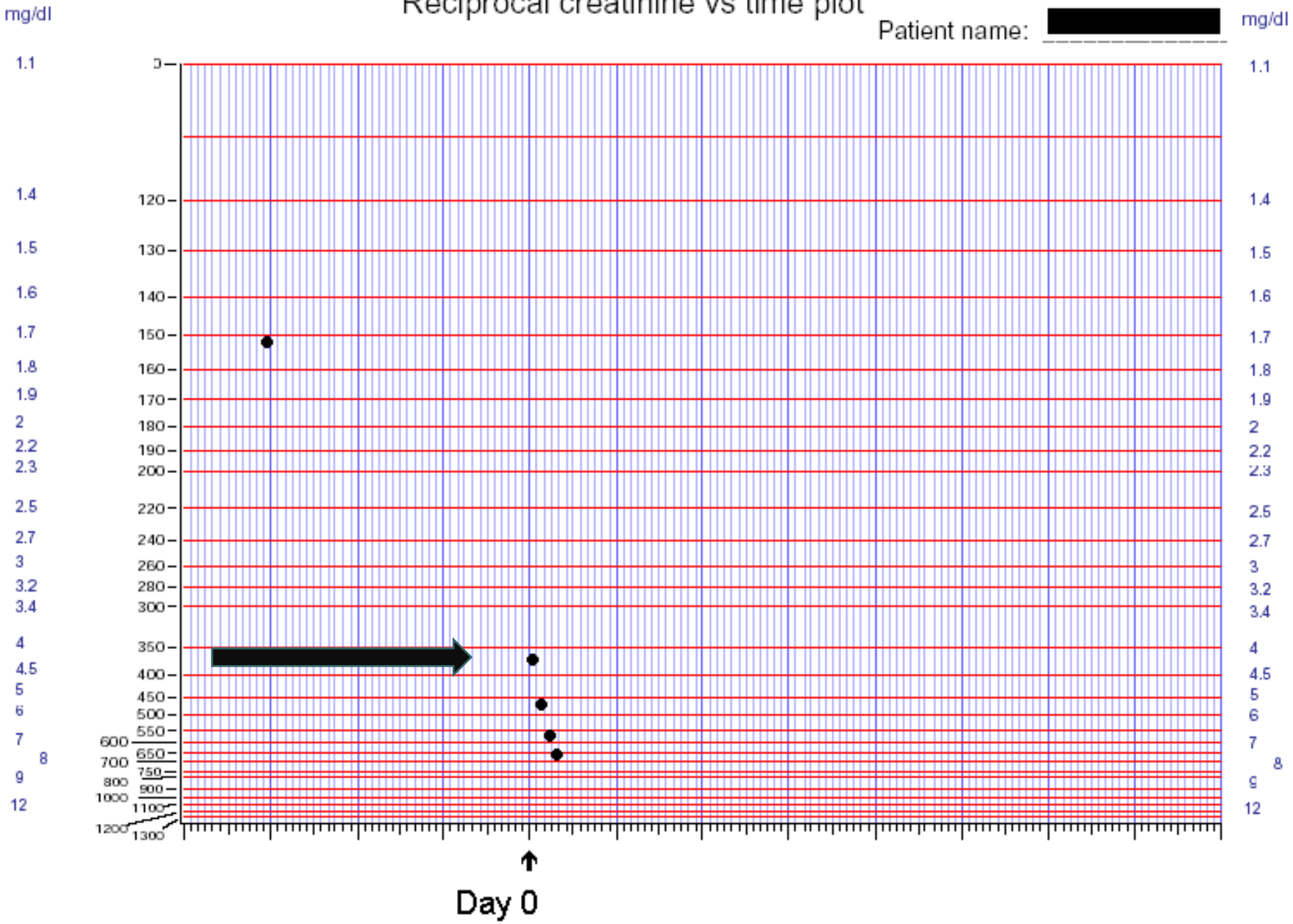
- time 0 hrs
- serum creatinine 90
- eGFR 99
- actual GFR normal



- time 24 hrs
- serum creatinine 200
- eGFR 39
- actual GFR 0



- time 72 hrs
- serum creatinine 800
- eGFR 8
- actual GFR 0



Supportive care of the AKI patient

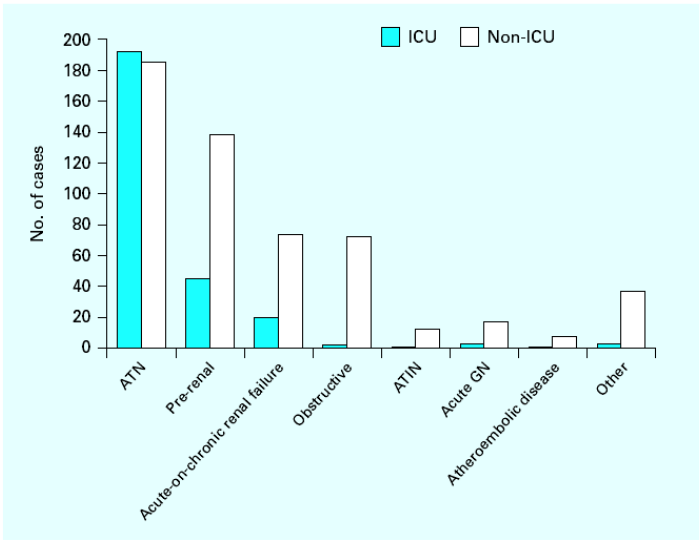


Fig 1. Spectrum of acute renal failure in intensive care unit (ICU) (253 patients) and non-ICU (495) settings. More than one type of ARF could be recorded for each ARF episode; the main ARF aetiologies are illustrated (ATN = acute tubular necrosis; ATIN = acute tubulo-interstitial nephritis; GN = glomerulonephritis (adapted from Ref 7).

Spectrum of ARF in ICU and non-ICU settings.
Kidney International 1998; 53:S16

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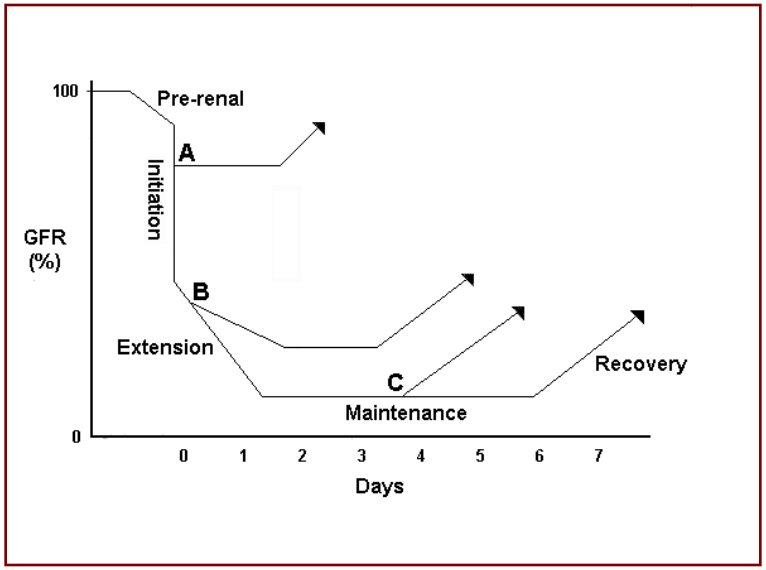
Esoteric causes
aetiology



Molitoris, B. A. (2003) *J Am Soc Nephrol,* **14,** 265-7.

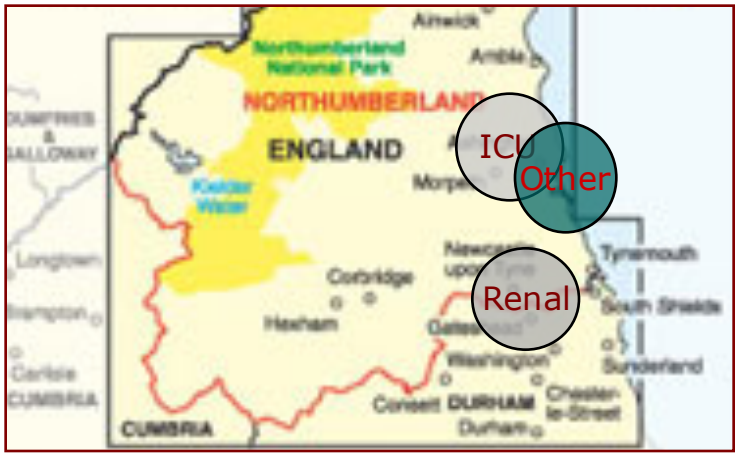
May be suggested by:

- Lack of clear, precipitating insult(s)
- Prolonged time-course
  - ATN usually recovers in 7–21 days
  - May be later, compounding insults

Maintain vigilance for obstruction

- May have been missed, at 1st, by initially non-compliant renal tract or superimposed ATN (urine is needed to dilate)

Supportive care of the AKI patient



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Transfer of care
Renal involvement

- If an esoteric diagnosis is possible
- If dialysis is indicated
- If AKI is prolonged, even if dialysis is not indicated

Emergency transfer of in-patients to renal services – admission MEWS scoring may identify those at risk of needing early critical care

N.S. Kanagasundaram, S.A. Tee, M.Brady, L.Grant, J.F. Cosgrove

Findings

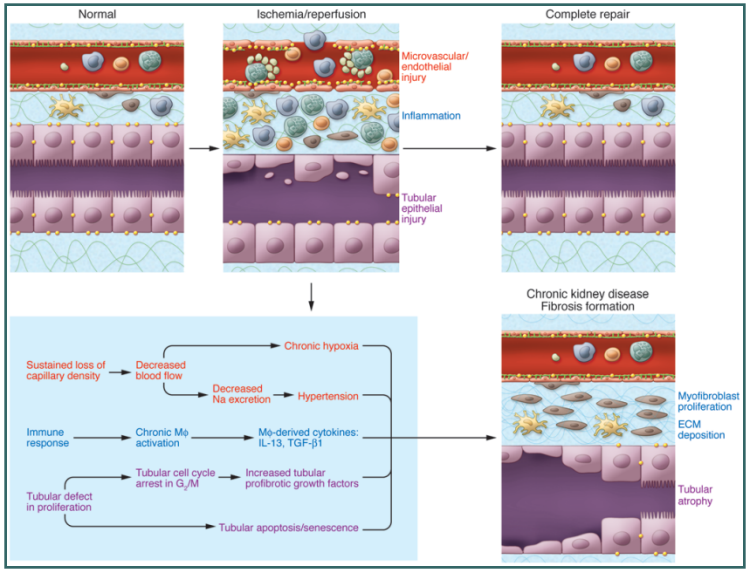
- 136 emergency transfers (127 patients inc. 36 chronic dialysis, 65 AKI):

- 4 patients required a step-up care on the day of admission (3 AKI, 1 ESRD, 2 from ward-level care, 2 from A+E)

MEWS	Stepped up on day of transfer (n = 4)
0 (n = 79)	0%
≥ 1	7%
≥ 2	10%
≥ 3	27%

- MEWS now used to facilitate discussions with ICU
- All transfers discussed with senior on-call nephrologist
- All critical care step-down transfers also discussed with senior intensivist

Supportive care of the AKI patient



From Bonventre, J.V. and Li Yang, L. *J Clin Invest.* 2011;121(11):4210.

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Long term follow up

Persisting CKD

Long-term prognosis after acute kidney injury requiring renal replacement therapy

Nephrol Dial Transplant (2009) 24: 2186–2189

Pierre-Alain Triverio[1], Pierre-Yves Martin[1], Jacques Romand[2], Jerome Pugin[2], Thomas Perneger[3] and Patrick Saudan[1]

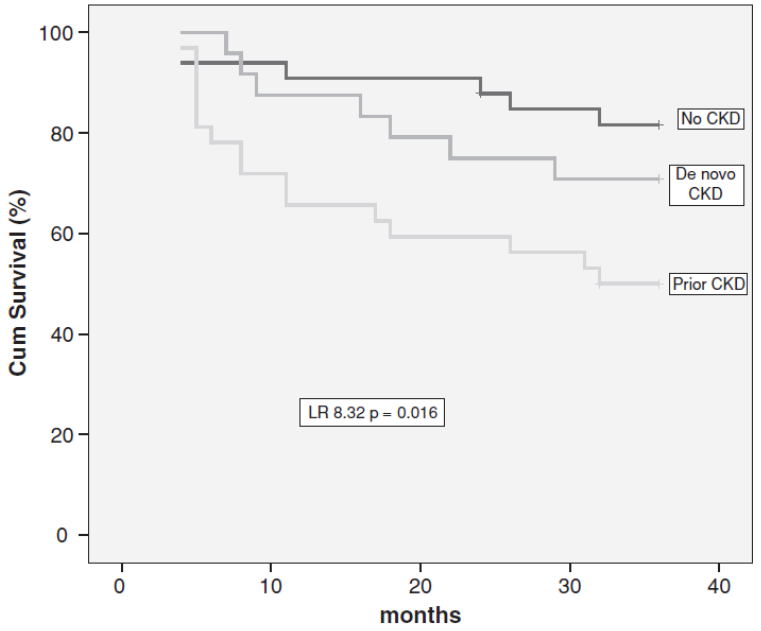

Fig. 1. Kaplan–Meyer analysis of 3-year survival rates in patients without (no CKD prior to AKI) and with prior (prior CKD to AKI) or *de novo* CKD after AKI.

- **N= 89 at 3 years**
- **ESRD developed in 9**
  - 8 had prior CKD

Long term follow up

Discharge advice

- Advice to patient and primary care:
  - Intercurrent illness
    - Early and regular monitoring of biochemistry
    - Drug holiday

Supportive care of the AKI patient

- **Haemodynamic support**
  - Vulnerability of the acutely injured kidney
  - Tightrope walk between dangerous hypo- and hyper-volaemia
- **Nutritional support**
  - Malnutrition is common in AKI
  - Maintenance and replacement needs to account for associated hypercatabolism and the effects of renal support
- **Prescribing**
  - Justify initiation / continuation of nephrotoxins
  - Consider need for drug dose adjustment if on renal support and according to renal support technique
  - Remember to play 'catch-up' with drug dosing when renal function is changing, quickly
- **Esoteric causes**
  - May be suggested by aberrance from expected natural history
- **Transfer of care**
  - Should be safe
- **Long term follow up**
  - CKD management where appropriate
  - Advice for intercurrent illness