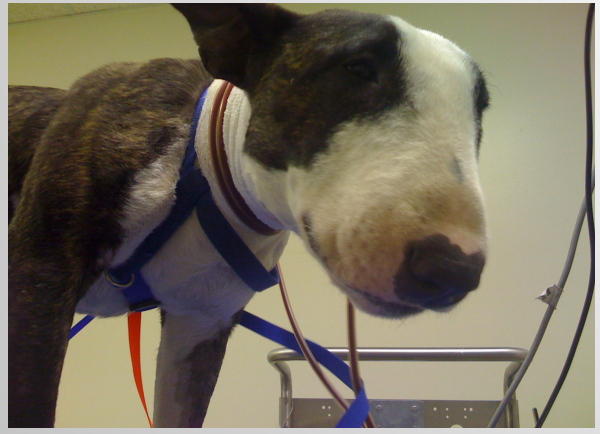
Acute Kidney Injury



CANADA WEST
VETERINARY SPECIALISTS

Carsten Bandt, DACVECC

Definition

"Acute and sustained reduction in renal function."

Definition

"Acute kidney injury represents a continuum of renal injury."

Classification Systems

Classification system in human medicine try to emphasize this continuing of renal injury:

AKIN

KDIGO

RIFLE

KDIGO Clinical Practice Guideline for Acute Kidney Injury

Staging

R isk

I njury

F ailure

L oss of function

E nd-Stage Renal disease

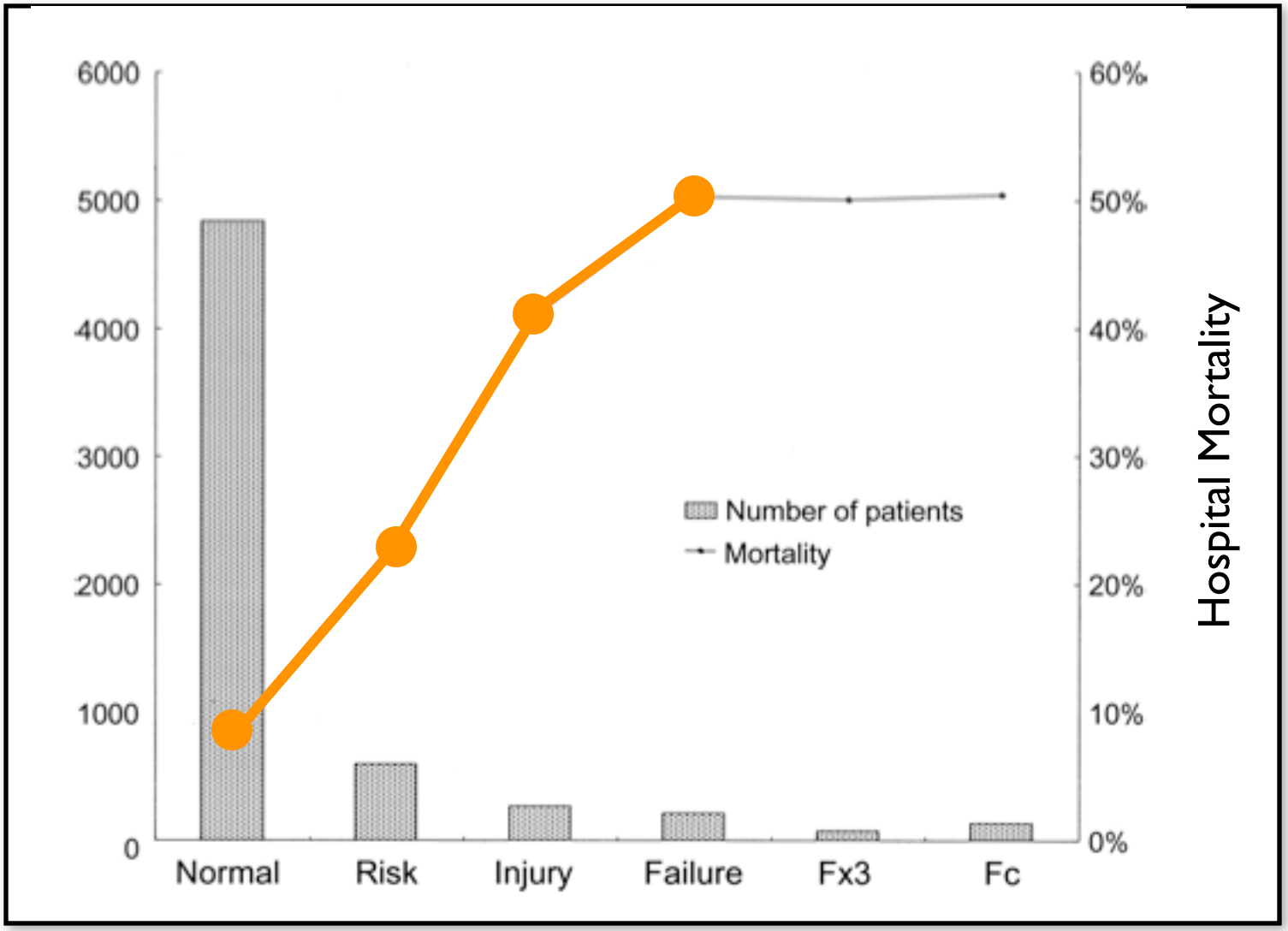
Risk: Inc Cr 50-100% or U.O. < 0.5 mL/kg/hr for > 6 hrs

Injury: Inc Cr 100-200% or U.O. < 0.5 mL/kg/hr > 12 hrs

Failure: Inc Cr > 200% or > 4 mg/dL or U.O. < 0.3 mL/kg/hr > 24 hrs or anuria for more than 12 hours

Loss of function: Need for dialysis for more than 4 weeks

End-Stage Renal disease : Need for dialysis for more than 3 months


6000
5000
4000
3000
2000
1000
0
60%
50%
40%
30%
20%
10%
0%
Number of patients
Mortality
Normal
Risk
Injury
Failure
Fx3
Fc
Hospital Mortality

Acute kidney injury in cats and dogs: A proportional meta-analysis of case series studies

Sabrina Almeida Moreira Legatti[1], Regina El Dib[2,3¤*], Emerson Legatti[4], Andresa Graciutti Botan[5], Samira Esteves Afonso Camargo[5], Arnav Agarwal[6], Pasqual Barretti[7], Antônio Carlos Paes[1]

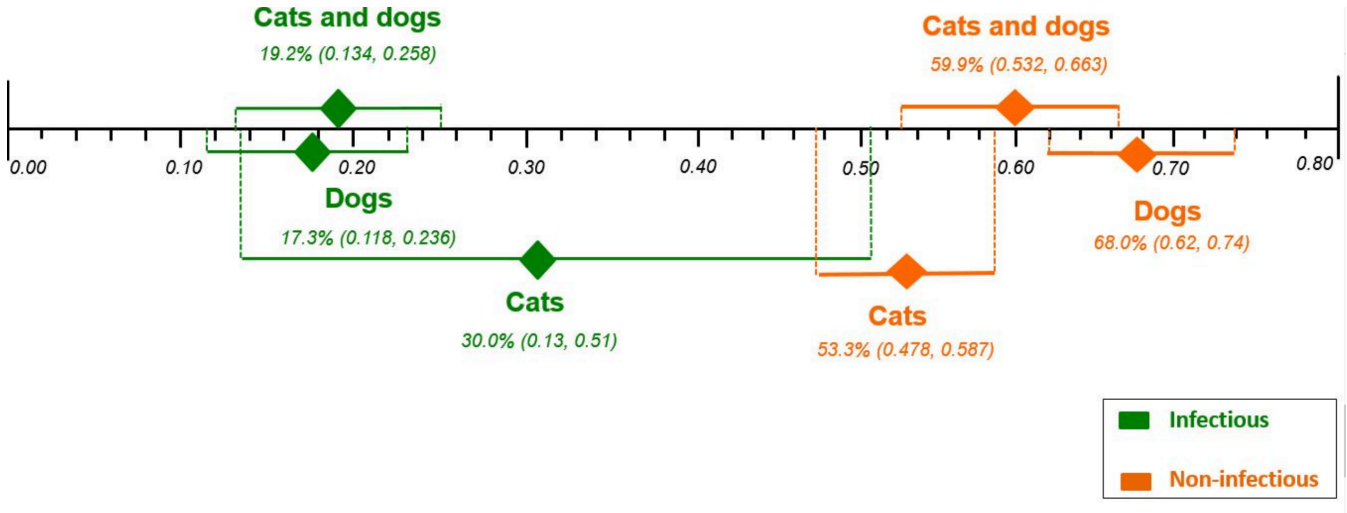
1 Department of Veterinary Hygiene and Public Health, School of Veterinary Medicine and Animal Science, Unesp – Univ Estadual Paulista, Botucatu, São Paulo, Brazil, **2** Department of Anaesthesiology, Botucatu Medical School, Unesp – Univ Estadual Paulista, Botucatu, São Paulo, Brazil, **3** McMaster Institute of Urology, McMaster University, Hamilton, Ontario, Canada, **4** School of Veterinary Medicine and Animal Science, Unesp – Univ Estadual Paulista, Botucatu, São Paulo, Brazil, **5** Institute of Science and Technology, Department of Biosciences and Oral Diagnosis, Unesp – Univ Estadual Paulista, São José dos Campos, São Paulo, Brazil, **6** Schoolof Medicine, University of Toronto, Toronto, Canada, **7** Department of Internal Medicine, Botucatu Medical School, Unesp – Univ Estadual Paulista, Botucatu, São Paulo, Brazil

AKI ETIOLOGY

Total #of (%)	
Cats	401 (33.4)
Dogs	800 (66.6)
Type of AKI (%)	
Oliguric	249(20.7)
Non-oliguric	304 (25.3)
Unknown	88 (7.3)
Total #of animals (%) per etiology	
• **Infectious**	353 (29.4)
Leptospirosis	154 (43.6)
Pyelonephritis	20 (5.7)
Pyometra	132 (37.4)
Sepsis	9 (2.5)
• **Non-infectious**	726 (60.4)
Nephrotoxic	220 (30.3)
Obstructive	115 (15.8)
Unknown	265 (36.5)

Acute kidney injury in cats and dogs: A proportional meta-analysis of case series studies

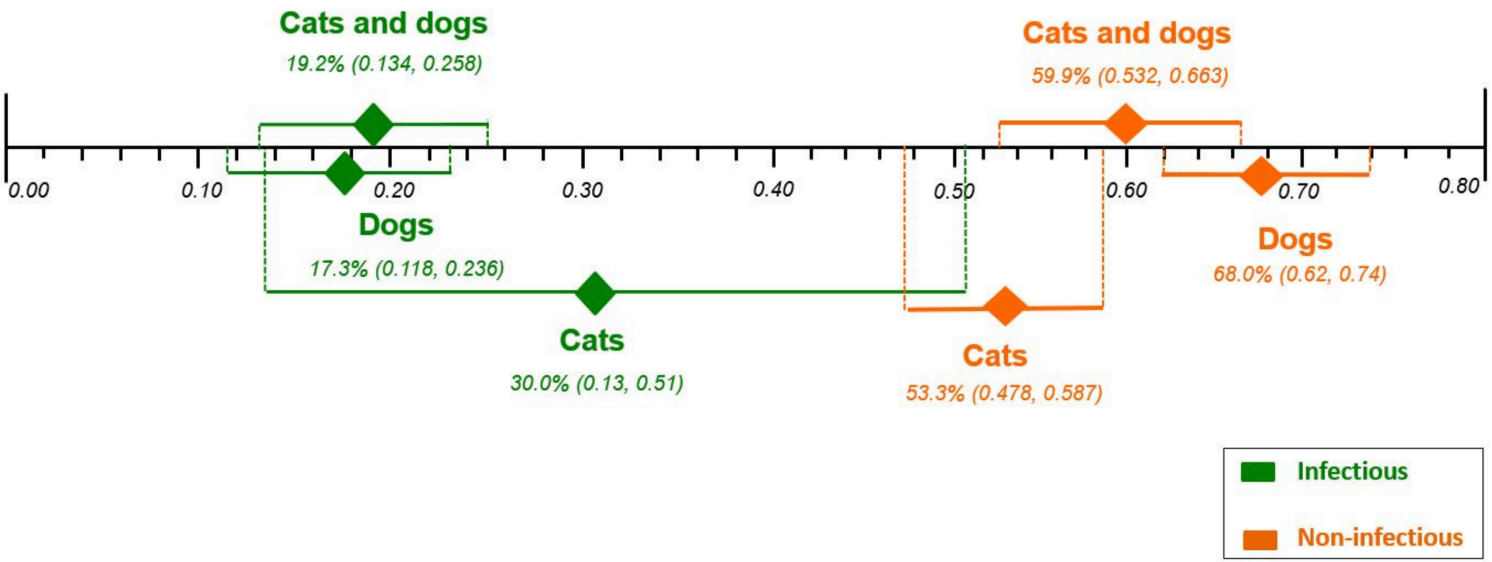
Dogs and cats with AKI due to a non-infectious etiology have higher mortality rates than those with AKI due to infectious etiologies, regardless of the treatment received



Acute kidney injury in cats and dogs: A proportional meta-analysis of case series studies

The combined mortality rate for dogs and cats with AKI is 47.2%

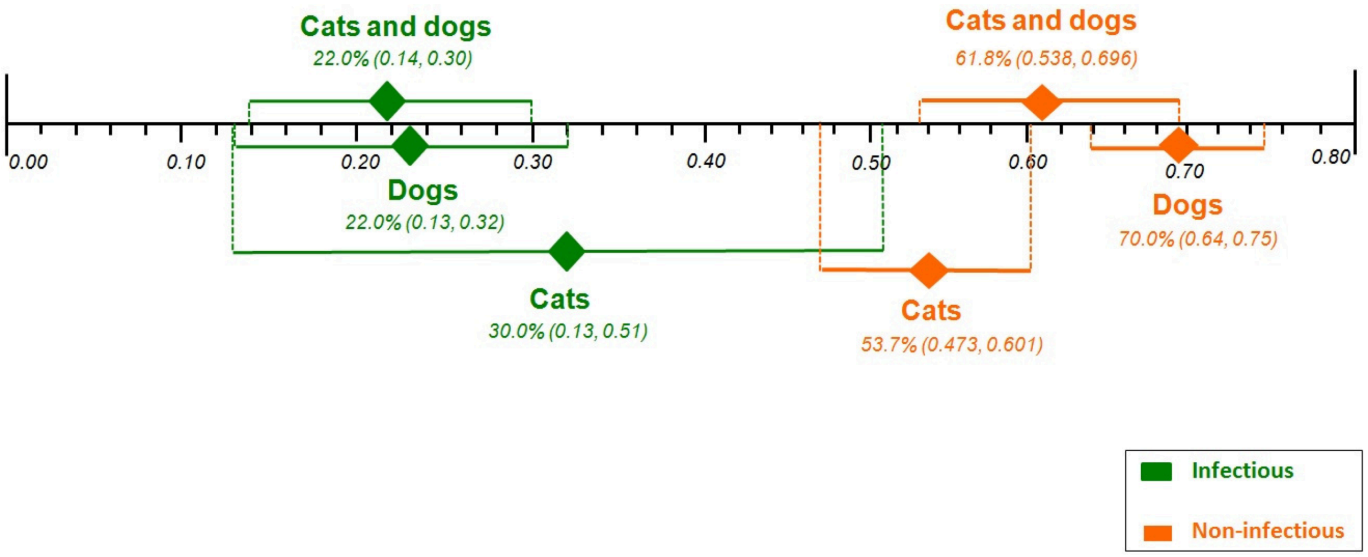
Regardless of AKI etiology and treatment, cats were found to have non-significantly higher AKI-associated mortality rates (53.1%) than dogs (45%)



Acute kidney injury in cats and dogs: A proportional meta-analysis of case series studies

Regardless of etiology, dialysis treatment is associated with higher mortality rates compared to conservative treatment among cats and dogs, though the association is not statistically significant.

The results may be potentially influenced by heterogeneity between studies, and by the increased severity of AKI for animals on dialysis relative to those being managed conservatively (88.4% vs. 11.6% rates of oliguria)



The Veterinary One

Continuum of renal injury from mild to severe

Emphasis on the concept of AKI as a continuum

The Veterinary One

Table 1: IRIS AKI Grading Criteria

AKI Grade	Blood Creatinine	Clinical Description
Grade I	<1.6 mg/dl (<140 µmol/l)	Nonazotemic AKI: a. Documented AKI: (historical, clinical, laboratory, or imaging evidence of AKI, clinical oliguria/anuria, volume responsiveness‡) and/or b. Progressive nonazotemic increase in blood creatinine: ≥ 0.3 mg/dl (≥ 26.4 µmol/l) within 48 h c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade II	1.7 – 2.5 mg/dl (141 – 220 µmol/l)	Mild AKI: a. Documented AKI and static or progressive azotemia b. Progressive azotemic: increase in blood creatinine; ≥ 0.3 mg/dl ≥ 26.4 µmol/l) within 48 h),or volume responsiveness‡ c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade III	2.6 – 5.0 mg/dl (221 – 439µmol/l)	Moderate to Severe AKI: a. Documented AKI and increasing severities of azotemia and functional renal failure
Grade IV	5.1 – 10.0 mg/dl (440 – 880 µmol/l)	
Grade V	>10.0 mg/dl (>880 µmol/l)	

Is It Really That Simple?

Creatinine as a lagging indicator

It is a lagging indicator

The actual damage occurs up to 48 hours before creatinine increases

Creatinine and GFR

The Big Assumption

Creatinine excretion rate is the product of creatinine concentration and GFR

A 50% decrease in GFR will be accompanied by a doubling of creatinine concentration

This holds true only if creatinine generation is constant!

ACUTE KIDNEY INJURY

Creatinine and AKI—through a glass, darkly

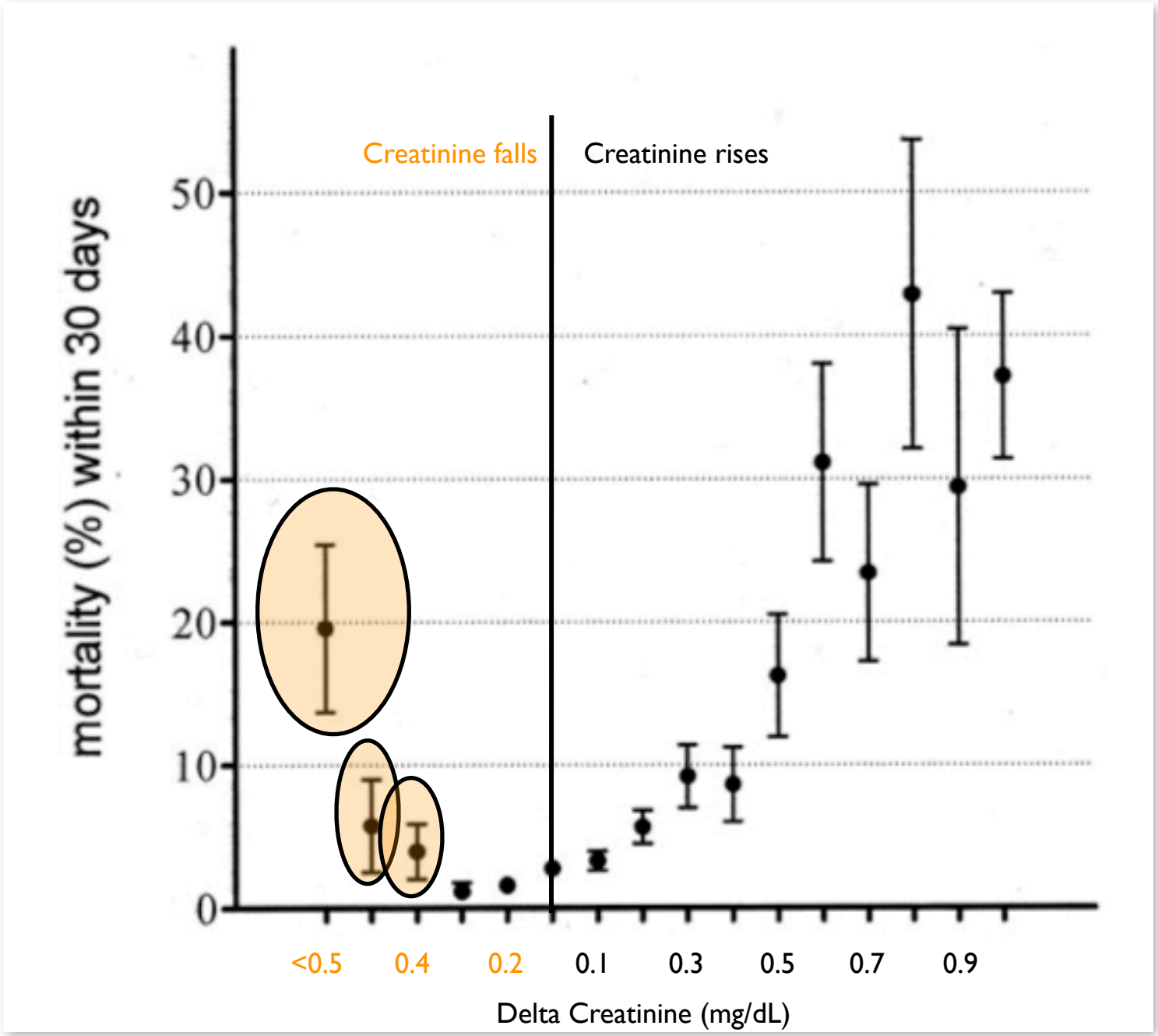
John R. Prowle

Creatinine generation rate is decreased by 25-50% in critical illness

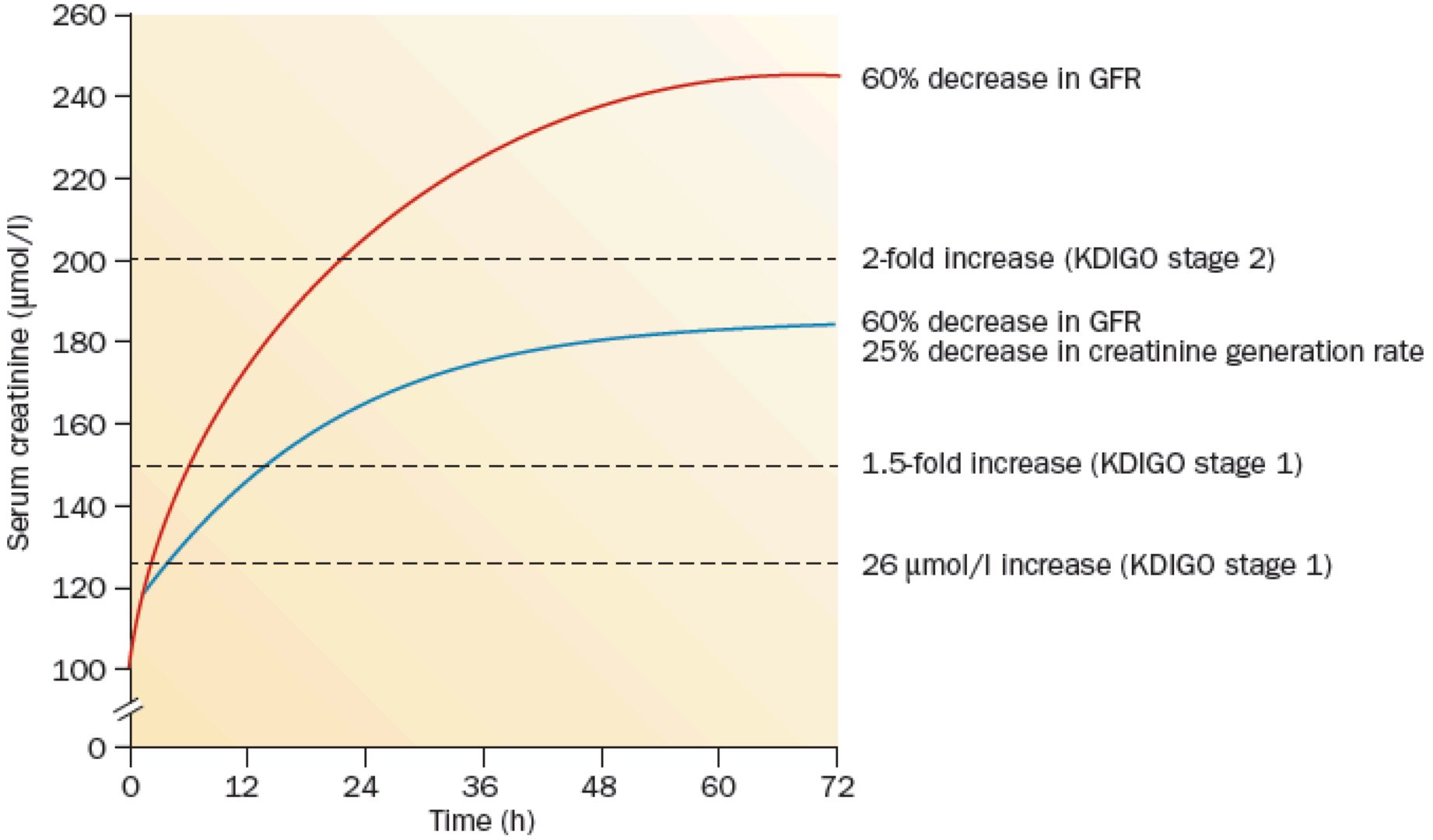
Severe reduction in GFR occurs in critically ill patients with unchanged creatinine levels

The magnitude of the decrease in creatinine generation correlates with illness severity

Prowle, J. R. Nat. Rev. Nephrol. 9, 193–195 (2013);



Prowle, J. R. Nat. Rev. Nephrol. 9, 193–195 (2013);


260
240
220
200
180
160
140
120
100
0
Serum creatinine (µmol/l)
0
12
24
36
48
60
72
Time (h)
60% decrease in GFR
2-fold increase (KDIGO stage 2)
60% decrease in GFR
25% decrease in creatinine generation rate
1.5-fold increase (KDIGO stage 1)
26 µmol/l increase (KDIGO stage 1)

...in Animals Too

Reduced Production of Creatinine Limits Its Use as Marker of Kidney Injury in Sepsis

Kent Doi, Peter S.T. Yuen, Christoph Eisner, Xuzhen Hu, Asada Leelahavanichkul, Jürgen Schnermann, and Robert A. Star

"Sepsis dramatically decreased production of creatinine in nephrectomized mice, without changes in BW, PCV, or EFV."

J. Am. Soc. Nephrol. 20, 1217–1221 (2009)

Take-Home Message

Small increases in serum creatinine count, especially in the very sick ones!!

ACUTE KIDNEY INJURY

Urine output in AKI—the canary in the coal mine?

Ravindra L. Mehta

UO < 0.3 ml/kg/h for > 6h predicts increase in CREA and dialysis requirement

Mehta, R. L. *Nat. Rev. Nephrol.* 9, 568–570 (2013)

Urine Output
-Post-Operative Changes-

- ADH secretion is increased following general anesthesia
- UO and free water clearance in the first 12 to 24 hours after surgery are reduced
- Poor value of USG, FWC, and FENa as measures of renal function in post-op

Crit Care Med 2008; 36: S141–5

CHRISTMAS 2010: THE LIVES OF DOCTORS

Urine output on an intensive care unit: case-control study

Objective: To compare urine output between junior doctors in an intensive care unit and the patients for whom they are responsible.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Oliguria is a common occurrence in patients admitted to intensive care and is associated with a marked increase in morbidity and mortality

WHAT THIS STUDY ADDS

Oliguria occurs twice as frequently in junior doctors on an intensive care unit as in their patients

This oliguria was not associated with increased mortality

Markers of acute kidney injury in junior intensive care unit doctors might diverge from those for the intensive care unit population as a whole

BMJ 2010;341:c6761 doi:10.1136/bmj.c6761

Oliguria as predictive biomarker of acute kidney injury in critically ill patients

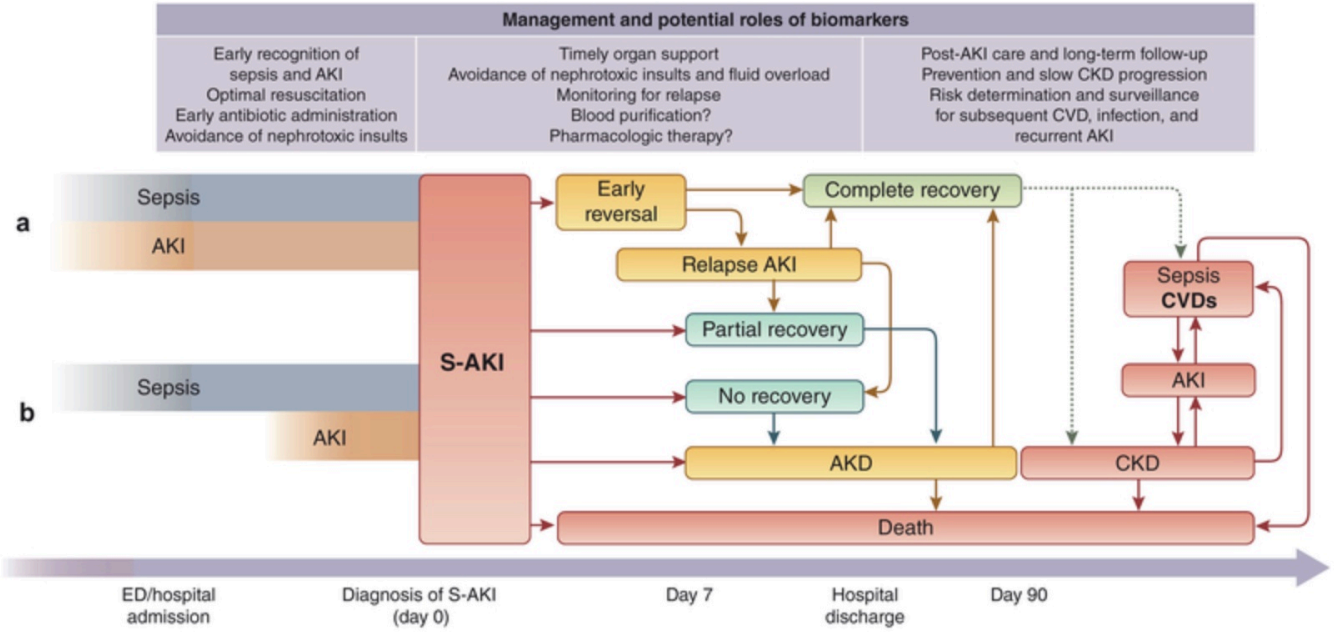
John R Prowle[1], Yan-Lun Liu[1], Elisa Licari[1], Sean M Bagshaw[2], Moritoki Egi[3], Michael Haase[4], Anja Haase-Fielitz[4], John A Kellum[5], Dinna Cruz[6], Claudio Ronco[6], Kenji Tsutsui[7], Shigehiko Uchino[7] and Rinaldo Bellomo[1,8*]

"Reduced UO for > 4 h good predictor of a subsequent increase in serum creatinine levels, but this increase occurs in only a fraction of patients who develop oliguria."

Critical Care 2011, 15:R172 http://ccforum.com/content/15/4/R172

Hospital Aquired AKI

Hospital aquired AKI



Kidney Int. 2019 Nov; 96(5): 1083–1099.

Sepsis AKI

- We used to attribute S-AKI to the decreased global renal blood flow and secondary tubular epithelial cell death, or acute tubular necrosis.
- One reason is that the leading causes of AKI (e.g., sepsis, major surgery, heart failure, and hypovolemia) are all associated with hypoperfusion and shock
- S-AKI develops in the absence of renal hypoperfusion and clinical signs of hemodynamic instability

S-AKI

- Histopathological findings in postmortem human observations and harvested animal organs are not as severe as expected and do not correlate with functional alterations.

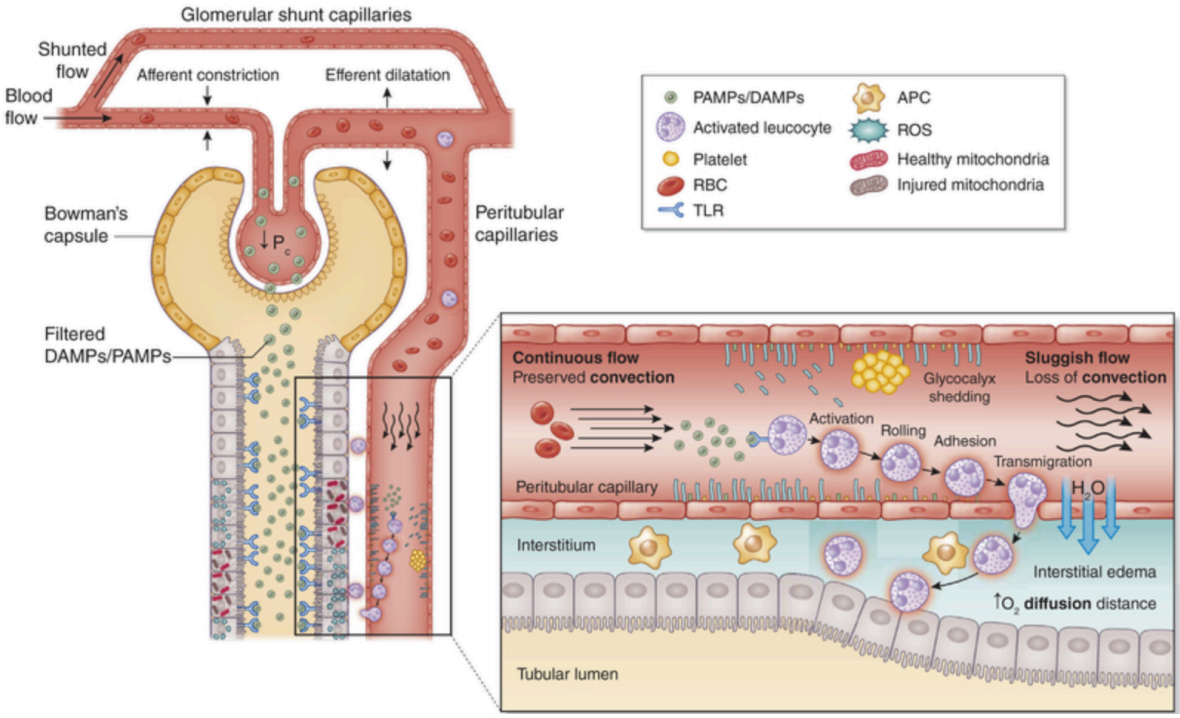
Renal histopathology during experimental septic acute kidney injury and recovery

Christoph Langenberg [1], Glenda Gobe, Sally Hood, Clive N May, Rinaldo Bellomo

"The lack of any tubular injury or increased apoptosis, the increased expression of all cortical nitric oxide synthase isoforms, and the link between inducible nitric oxide synthase and neuronal nitric oxide synthase with renal blood flow suggest in this experimental model that severe sepsis acute kidney injury can develop in the absence of histological or immunohistological changes and may be functional in nature."

Critical Care 2014 Jan;42(1):e58-67

Sepsis-associated AKI



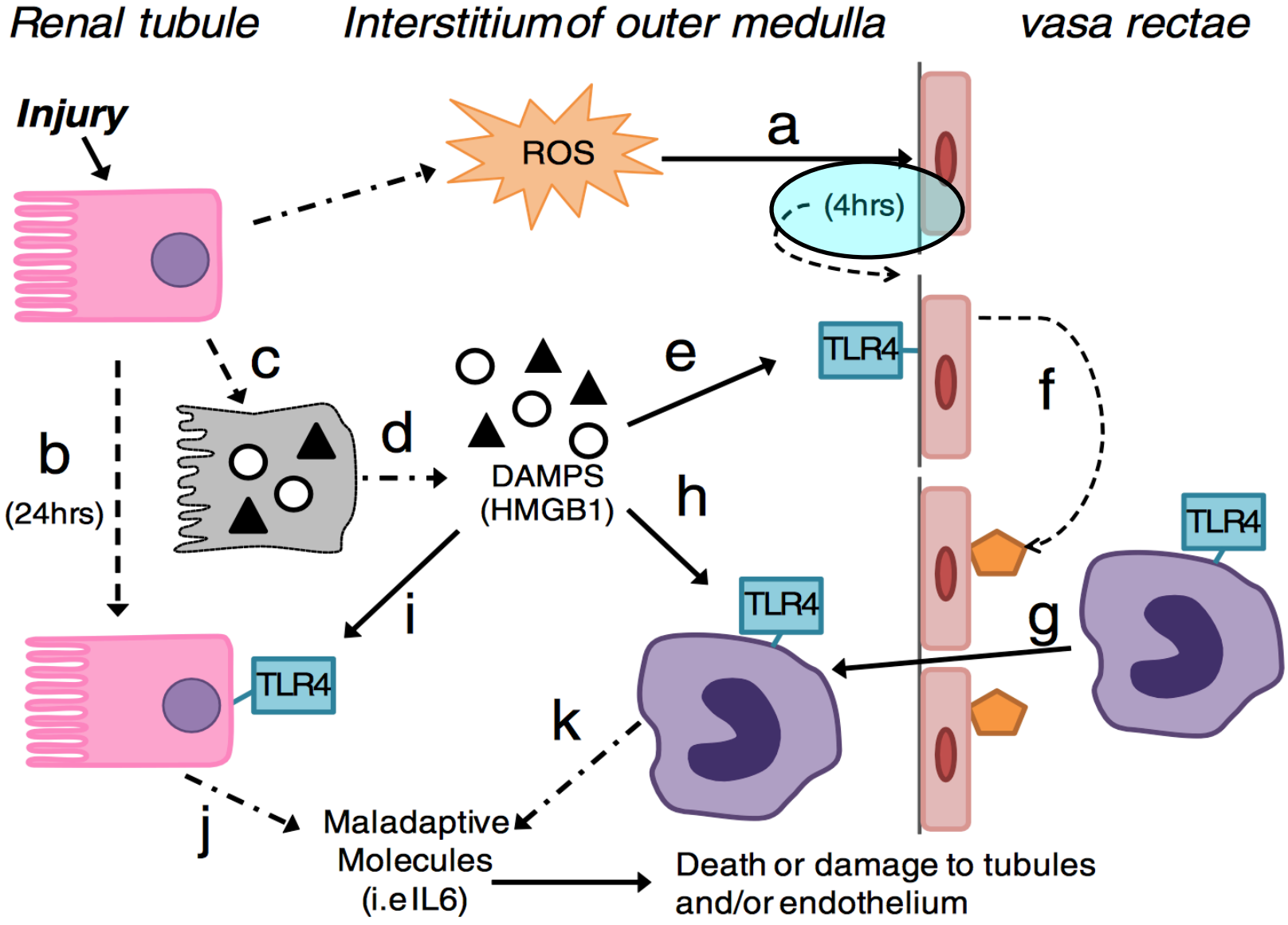
Kidney Int. 2019 Nov; 96(5): 1083–1099.

Acute kidney injury: a conspiracy of toll-like receptor 4 on endothelia, leukocytes, and tubules

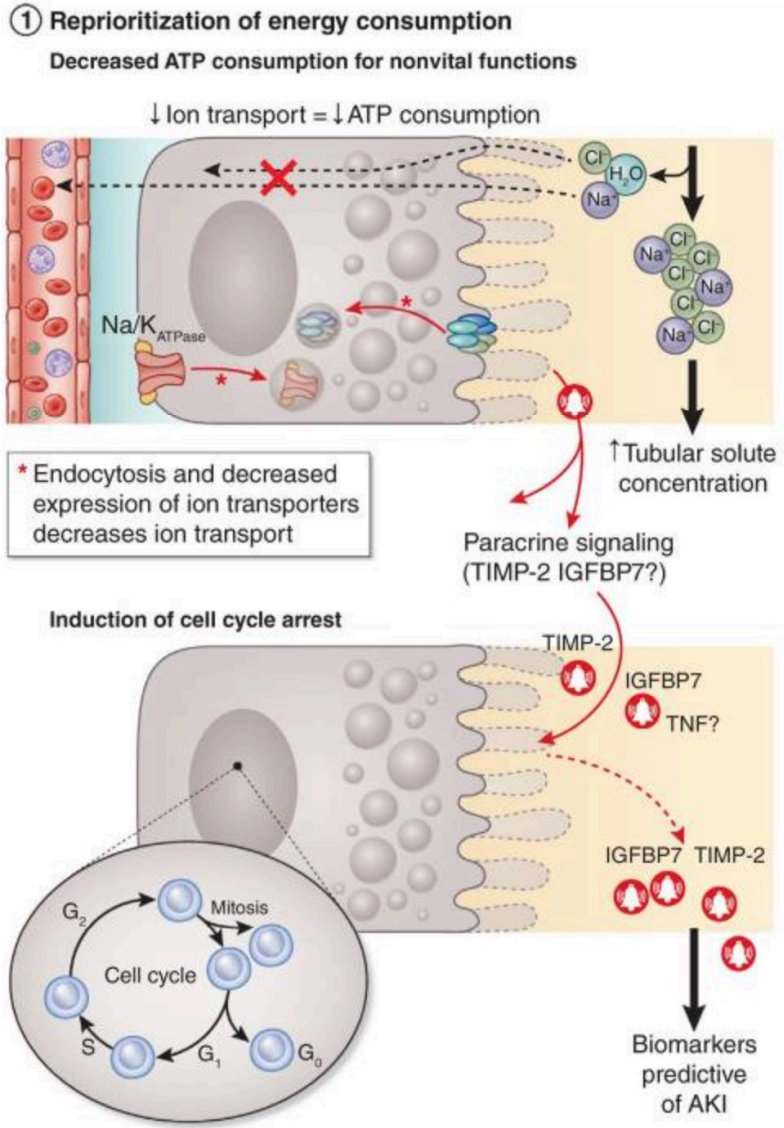
Christopher Y. Lu · Pamela D. Winterberg · Jianlin Chen · John R. Hartono

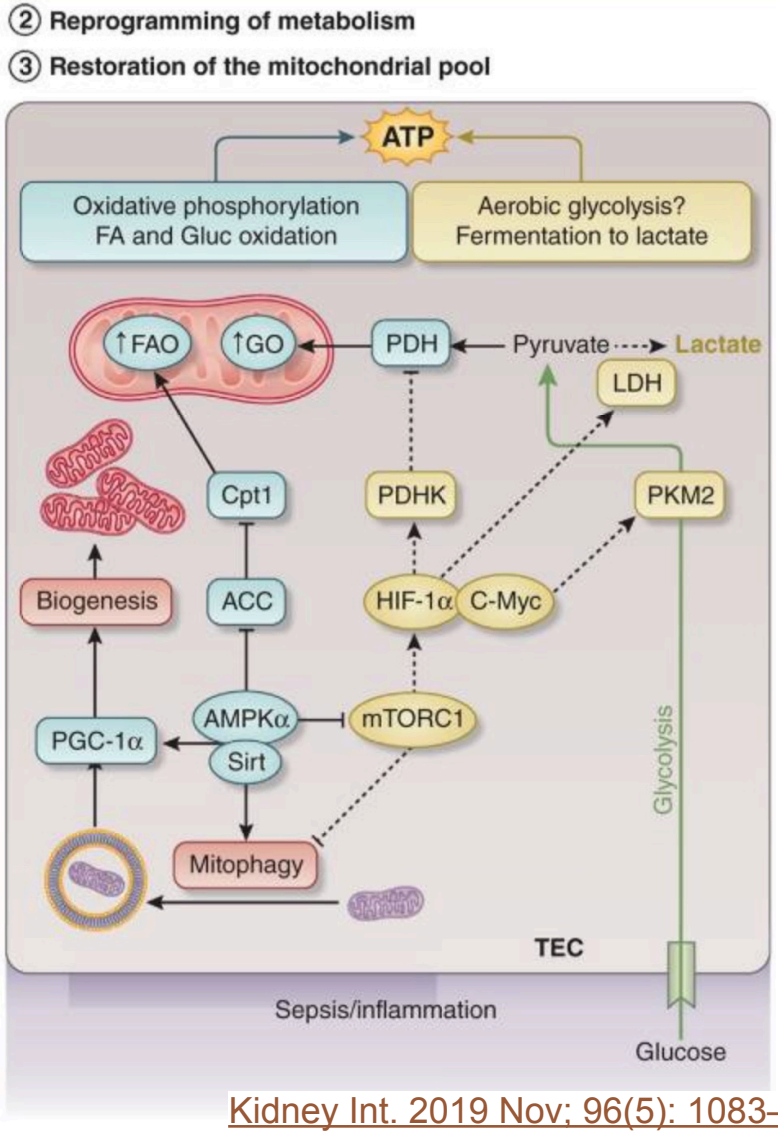
...”TLR4 are overexpressed on renal endothelium and epithelium following ischemic AKI”

Pediatr Nephrol . 2012 October ; 27(10): 1847–1854.


Renal tubule
Interstitium of outer medulla
vasa rectae
Injury
ROS
a
(4hrs)
b
(24hrs)
c
d
DAMPS
(HMGB1)
e
TLR4
f
h
TLR4
TLR4
i
g
TLR4
k
j
Maladaptive
Molecules
(i.e IL6)
Death or damage to tubules
and/or endothelium

Metabolic reprogramming





Kidney Int. 2019 Nov; 96(5): 1083–1099.

Common causes of AKI in ICU

- Sepsis
- Major surgery
- Low cardiac output
- Hypovolemia
- Medications
- Nephrotoxins

Prophylaxis is a way to get around the problem of late diagnosis due to the lack of an established biomarker.

Prevention of AKI in ICU

- Recognition of underlying risk factors
- Maintenance of renal perfusion
- Avoidance of hyperglycemia
- Avoidance of nephrotoxins

Prevention of AKI in ICU

- Avoid NSAIDs
- Avoid nephrotoxic medications
- Avoid potential nephrotoxic combinations
  - Acepromazine and NSAID's
  - NSAID's and diuretics

Management of AKI in ICU

- Maintain renal perfusion
- Correct metabolic derangements
- Provide adequate nutrition
- Avoid volume overload and role of diuretics
- Treat underlying reason for AKI

Therapy

Fluids!!!!

ACUTE KIDNEY INJURY IN 2012

Type of resuscitation fluid—it does matter!

Antoine G. Schneider and Rinaldo Bellomo

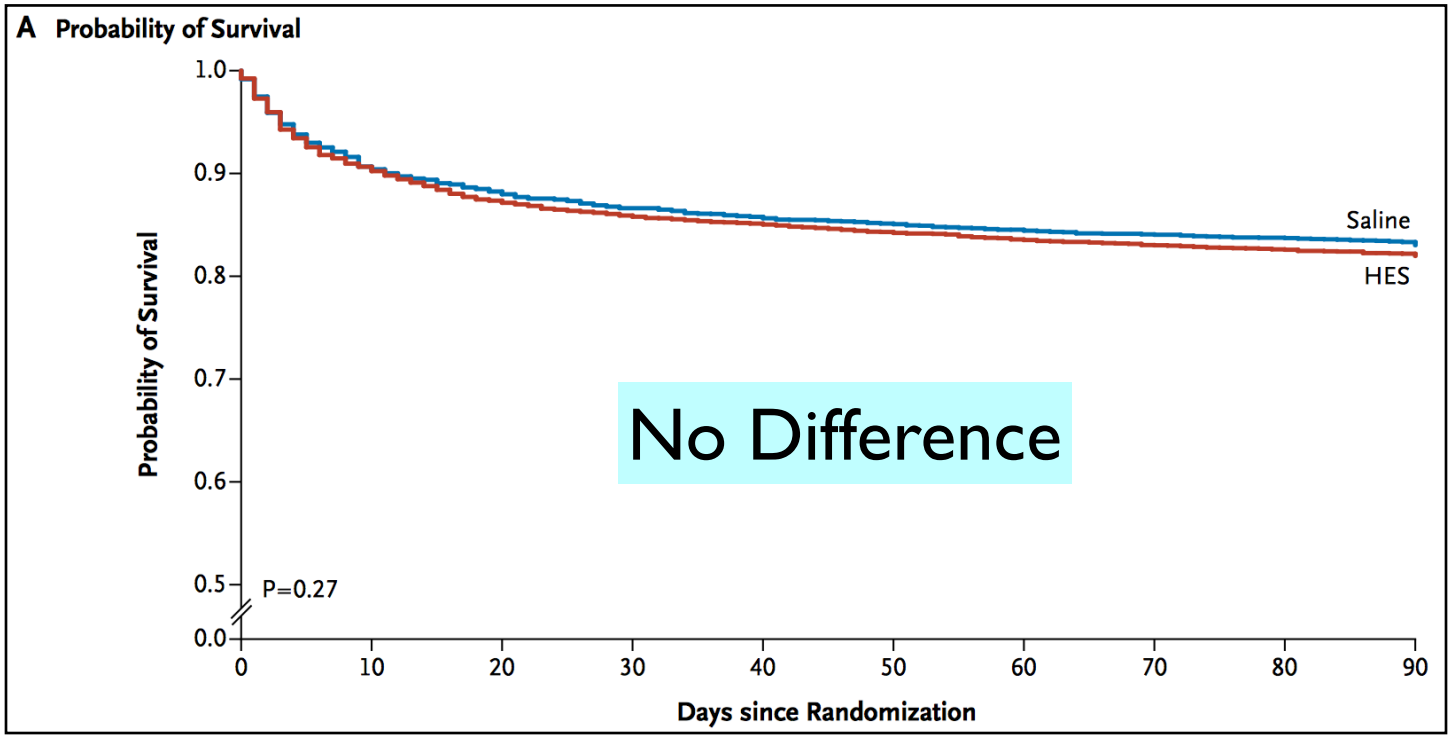
Key Points:

Nat. Rev. Nephrol. 9, 72–73 (2013)

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Hydroxyethyl Starch or Saline for Fluid Resuscitation in Intensive Care

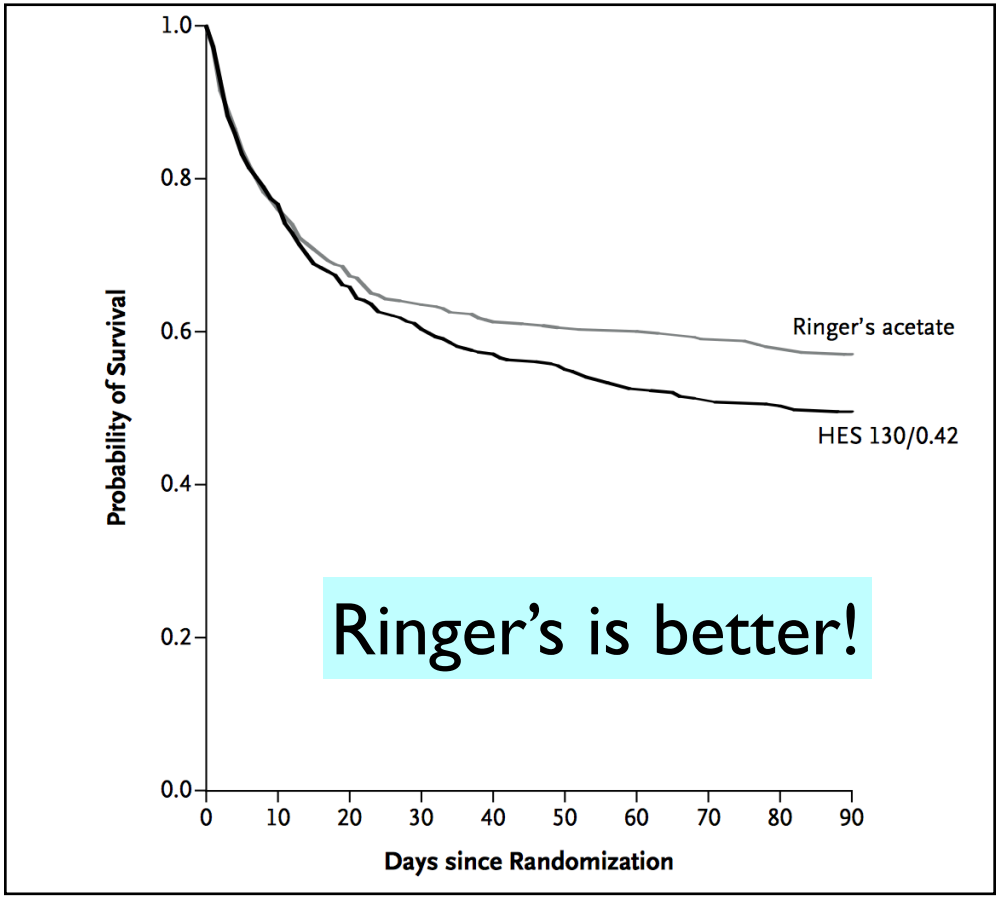


N Engl J Med 2012;367:1901-11.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis



Ringer's is better!

N Engl J Med 2012;367:124-34.

Association Between a Chloride-Liberal vs Chloride-Restrictive Intravenous Fluid Administration Strategy and Kidney Injury in Critically Ill Adults

Nor'azim Mohd Yunos, MD
Rinaldo Bellomo, MD, FCICM
Colin Hegarty, BSc
David Story, MD
Lisa Ho, MClinPharm
Michael Bailey, PhD

Conclusion: The implementation of a chloride-restrictive strategy was associated with a significant decrease in the incidence of AKI and use of RRT.

JAMA. 2012;308(15):1566-1572

Major Complications, Mortality, and Resource Utilization After Open Abdominal Surgery

0.9% Saline Compared to Plasma-Lyte

Andrew D. Shaw, MB, FRCA, FCCM, Sean M. Bagshaw, MD,† Stuart L. Goldstein, MD,‡ Lynette A. Scherer, MD,§ Michael Duan, MS,‖ Carol R. Schermer, MD,¶ and John A. Kellum, MD#*

Conclusions: The use of balanced crystalloids for replacement of fluid losses on the day of major surgery was associated with less postoperative morbidity than 0.9% saline.

Ann Surg 2012;255:821–829

Retrospective cohort study on the incidence of acute kidney injury and death following hydroxyethyl starch (HES 10% 250/0.5/5:1) administration in dogs (2007–2010)

Galina Hayes [1], Leontine Benedicenti [1], Karol Mathews [1]

Conclusions: HES administration was associated with increased risk of mortality or AKI.

JVECC Jan-Feb 2016;26(1): 35-40

Effects of Hydroxyethyl Starch 130/0.4 on Serum Creatinine Concentration and Development of Acute Kidney Injury in Nonazotemic Cats

N E Sigrist [1], N Kälin [1], A Dreyfus [2]

Conclusions: Hydroxyethyl-starch administration to critically ill nonazotemic cats seems to be safe. A larger prospective study is required to determine the effect of HES administration at higher dosages and for prolonged time periods.

JVIM 2017;255:821–829

Changes in Serum Creatinine Concentration and Acute Kidney Injury (AKI) Grade in Dogs Treated with Hydroxyethyl Starch 130/0.4 From 2013 to 2015

N E Sigrist [1], N Kälin [1], A Dreyfus [2]

Conclusions: HES-130/0.4-treated dogs were not more prone to develop AKI than HES-untreated, but the number of HES days was significantly associated with an increase in AKI grade within 10 days post-HES administration.

JVIM,2017 Mar;31(2): 434-441

Prospective randomized controlled blinded clinical trial evaluating biomarkers of acute kidney injury following 6% hydroxyethyl starch 130/0.4 or Hartmann's solution in dogs

Corrin J Boyd [1], Claire R Sharp [1], Melissa A Claus [1], Anthea L Raisis [1], Giselle Hosgood [1], Lisa Smart [1]

Conclusions: There were no differences in change over time of urine AKI biomarkers in dogs treated with 10 - 40 mL/kg HES or CRYST over 24 hours.

JVECC,2021 May;31(3) 306-314

How Much Fluids?

Fluid balance and acute kidney injury

John R. Prowle, Jorge E. Echeverri, E. Valentina Ligabo, Claudio Ronco and Rinaldo Bellomo

- Prolonged fluid resuscitation leads to renal edema

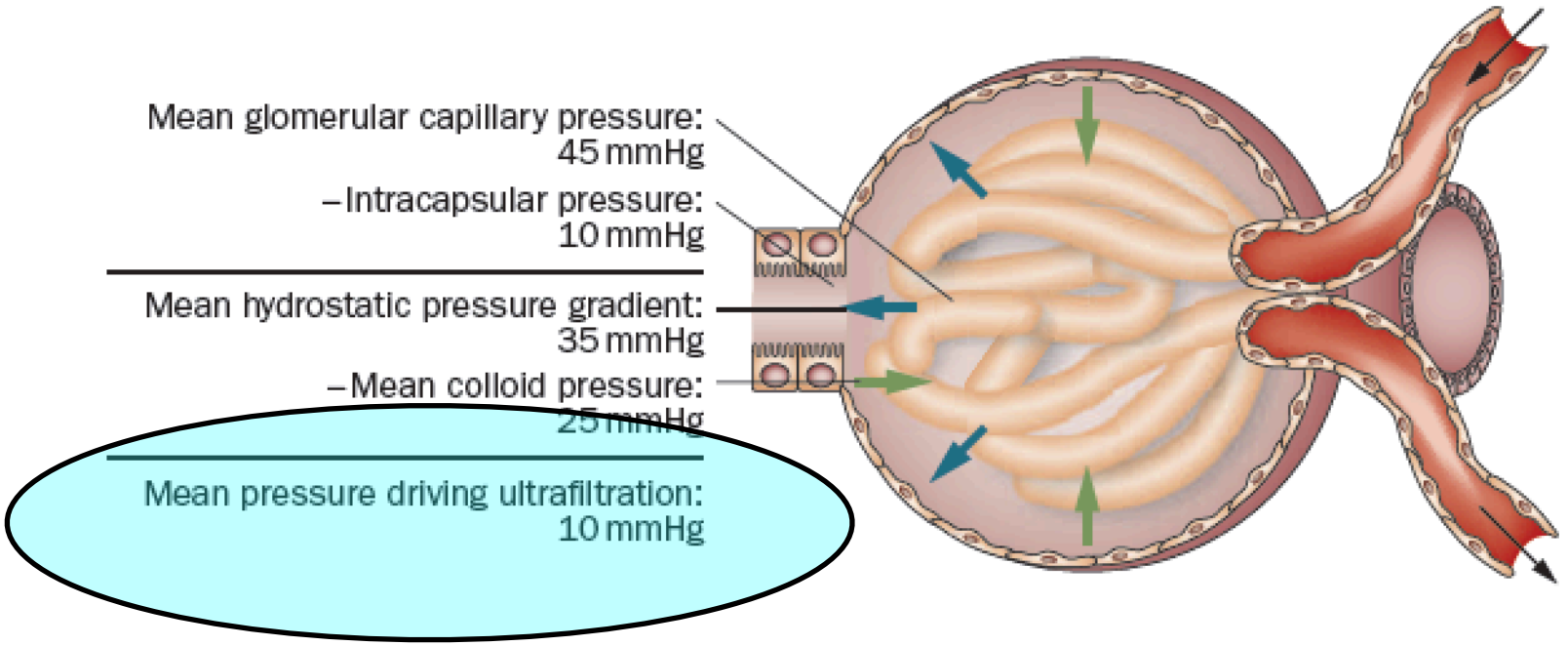
- Fluid overload is associated with increased morbidity

- An early transition to a fluid-restrictive strategy might be beneficial in patients with AKI

- Fluid removal in patients with or at risk of AKI should be implemented

Nat. Rev. Nephrol. 6, 107–115 (2010);

Why?


Mean glomerular capillary pressure:
45 mmHg
– Intracapsular pressure:
10 mmHg
Mean hydrostatic pressure gradient:
35 mmHg
– Mean colloid pressure:
25 mmHg
Mean pressure driving ultrafiltration:
10 mmHg

More fluid. More sick.

- longer ICU stay
- higher mortality
- more multi-organ dysfunction
- more likely to be intubated
- more inotropes
- more sepsis
- higher PRISM score

Table 2. Demographics and Clinical Information

	<10% Fluid Overload	≥10%-20% Fluid Overload	≥20% Fluid Overload	Total Cohort	*P*
No. of patients	153	51	93	297	
PICU length of stay (d)	15.7 ± 17.1	24.8 ± 30.0	29.5 ± 36.9	21.6 ± 27.6	<0.001
Mortality (%)	29.4	43.1	65.6	43.1	<0.001
Age (y)	10.4 ± 7.0	7.5 ± 6.8	6.1 ± 6.2	8.5 ± 7.0	<0.001
Weight (kg)	43.4 ± 32.1	29.1 ± 23.2	22.1 ± 23.1	34.3 ± 29.7	<0.001
Sex (%)					
Male	62.1	54.9	54.8	58.6	0.5
Female	37.9	45.1	45.2	41.4	
MODS diagnosis (%)	64.7	86.3	96.8	78.5	<0.001
Intubated	59.5	88.2	91.4	74.4	<0.001
Requiring ≥ 1 inotrope	49.7	76.5	80.7	64.0	<0.001
Requiring ≥ 2 inotropes	26.8	43.1	58.1	39.4	<0.001
eGFR < 60 mL/min/1.73 m^2	78.4	74.5	89.3	81.1	0.05
eGFR < 30 mL/min/1.73 m^2	49.0	47.1	58.1	51.1	0.3
Sepsis (%)	24.8	37.3	40.9	32.0	0.02
Oncologic process (%)	30.1	21.6	15.1	23.9	0.03
Inborn error of metabolism or intoxication diagnosis (%)	10.5	2.0	1.1	6.1	0.005
PRISM II score at PICU admission	13.1 ± 8.5	15.1 ± 7.9	15.9 ± 10.1	14.3 ± 9.0	0.04
Inotrope no. at CRRT initiation	0.9 ± 1.1	1.4 ± 1.0	1.7 ± 1.2	1.2 ± 1.2	<0.001
eGFR at CRRT initiation (mL/min/1.73 m^2)	47.5 ± 51.0	44.4 ± 33.0	33.9 ± 23.9	42.8 ± 41.6	0.05
CRRT indications included fluid overload (%)	69.3	82.4	88.2	77.4	0.002
CRRT modality (%)					0.04
Convective	60.1	49.0	44.1	53.2	
Diffusive	39.9	51.0	55.9	46.8	

Note: Values expressed as percentage or mean ± standard deviation. Univariate analysis of clinical parameters in each fluid overload group. $P < 0.05$ represents a significant association between increasing fluid overload severity and the respective variable (using analysis of variance for continuous variables and χ^2 for categorical variables). eGFR given in mL/min/1.73 m^2, factor for conversion to mL/s/1.73 m^2, ×0.01667.

Abbreviations: CRRT, continuous renal replacement therapy; eGFR, estimated glomerular filtration rate; MODS, multiorgan dysfunction syndrome; PICU, pediatric intensive care unit; PRISM, Pediatric Risk of Mortality.

3% increase in mortality for each 1% increase in degree of fluid overload

Oliguric

-Furosemide
-Treat primary cause
-Fluids only at maintenance rate

AVOID FLUID OVERLOAD AT ALL COSTS

Loop Diuretics

- Are They Nephrotoxic? -

- Aggressive diuretic usage does not result in increased structural injury.

- Aggressive use of loop diuretics to achieve greater volume removal is associated with improved outcomes

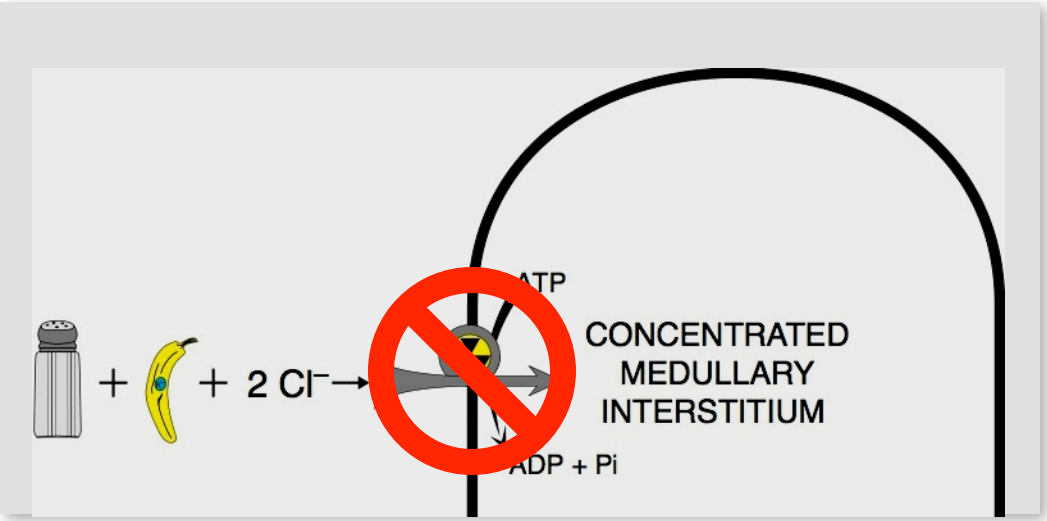
- Loop diuretic therapy achieved an equal amount of volume removal to ultrafiltration for kidney function outcomes

- Hypervolaemic patients with 'incipient AKI' should be treated with diuretics to achieve euvolaemia

Eur. J. Heart Fail. 14, 597–604 (2012).
Clin. J. Am. Soc. Nephrol. 6, 966–973 (2011).
N. Engl. J. Med. 367, 2296–2304 (2012).

Furosemide

- Decreased activity of the ascending loop of Henle
- Decreases renal oxygen demand



Loop Diuretics

- Are They Nephrotoxic? -

Answer: No they are not!

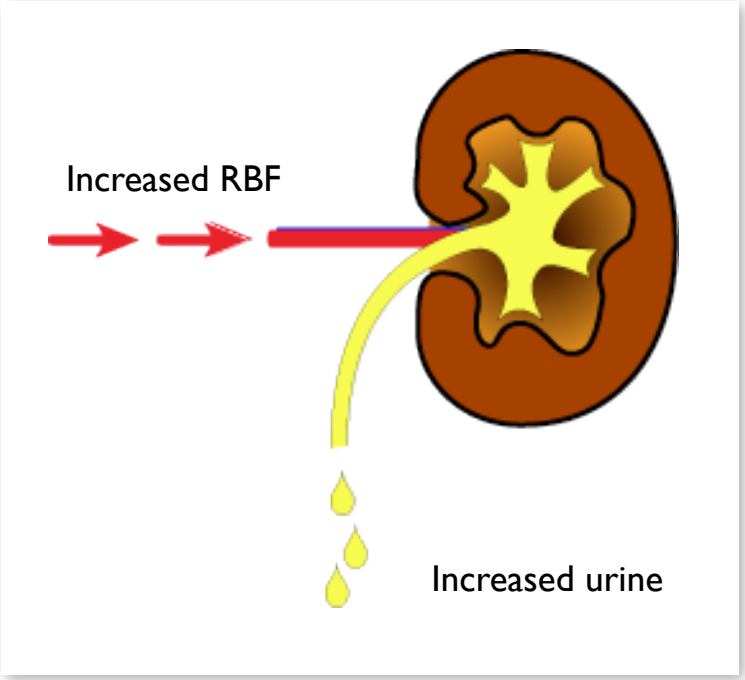
Helpful in converting some oliguric or anuric patients !

Nat. Rev. Nephrol. 9, 484–490 (2013)

Dopamine or no Dopamine?

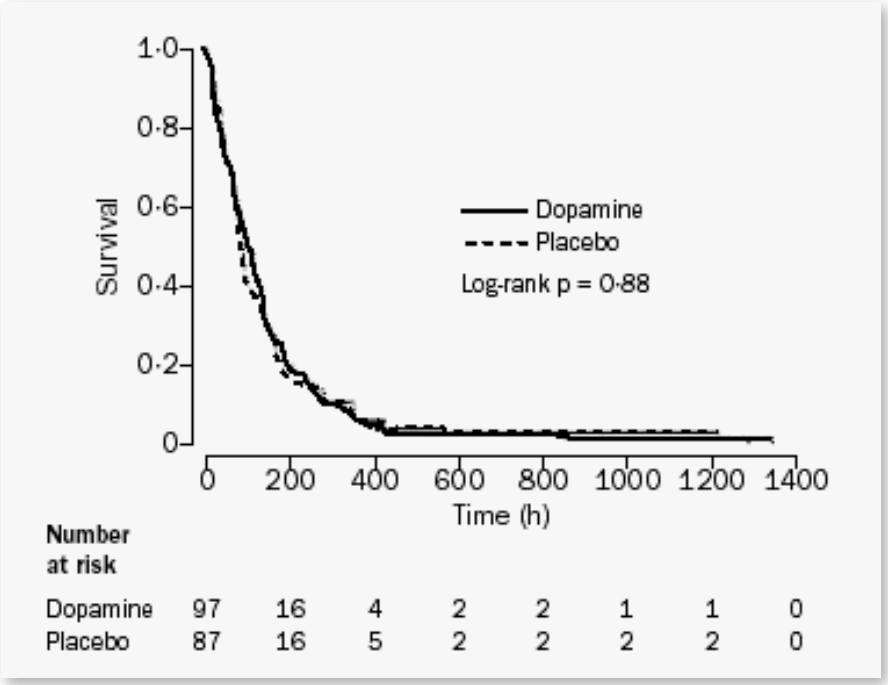
Dopamine: still doesn't work

- In healthy volunteers low dose dopamine increases renal blood flow and induces diuresis
- Patients in the intensive care unit do not respond this way.

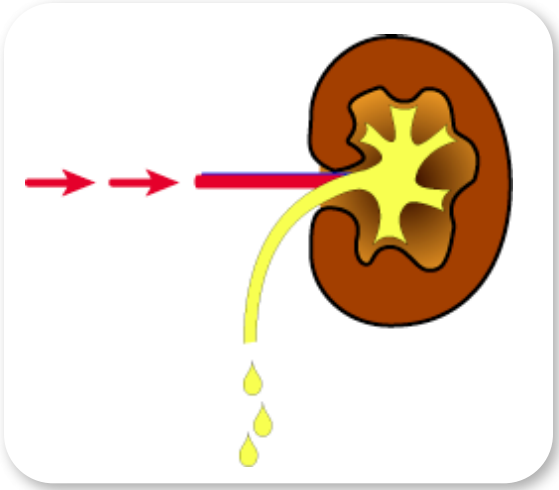


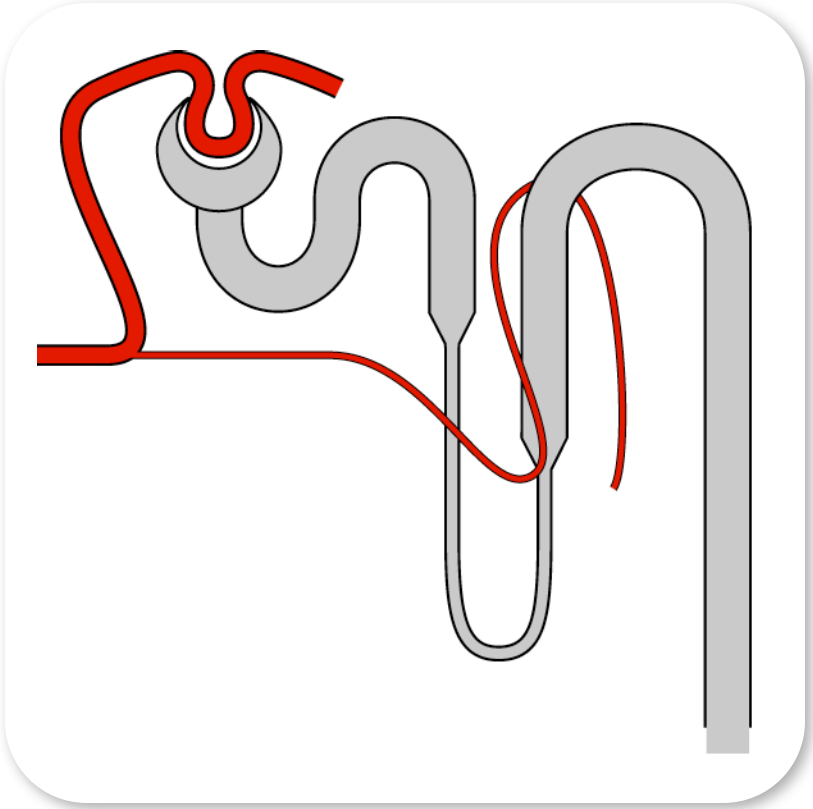
Dopamine: still doesn't work

- In healthy volunteers low dose dopamine increases renal blood flow and induces diuresis
- Patients in the intensive care unit do not respond this way.
  - RCT of 380 ICU patients with early renal failure



ANZICS Clinical Trials Group. Lancet 2000;356:2139-47.
Kellum JA, Decker JM.Crit Care 2001; 29:1526-31.





- Dopamine increases cortical blood flow more than medullary blood flow
  - Cortical blood flow increases GFR
  - Cortical blood flow increases renal oxygen demand

Complications of Low-Dose Dopamine

- Increase arrhythmias
- Increased myocardial oxygen demand
- Gut ischemia
- Suppressed respiratory drive
- Increased sensitivity to radiocontrast agents
- Decreases in T-cell activity

The Evidence 2008

- 53 studies, 2327 participants: 1293 received treatment and 1034 controls
- Dopamine and its analogues, diuretics, CCB, ACEi

...“We cannot draw conclusions about the effectiveness of these interventions in protecting patients’ kidneys during surgery”

Cochrane Database of Systematic Reviews 2008, Issue 4. Art. No.: CD003590.

The Evidence 2018

- 72 studies, 4378 participants: 2291 treatment and 2087 controls
- Dopamine and its analogues, diuretics, CCB, ACEi, NAC, ANP, NaHCO3, antioxidants and EPO

"No reliable evidence from the available literature suggests that interventions during surgery can protect the kidneys from damage"

Cochrane Database of Systematic Reviews 2013, Issue 9. Art. No.: CD003590

Vasopressors and target blood pressure

- Norepinephrine is current treatment of choice for septic shock
- Vasopresssin not associated with increased risk of AKI and in one study showed lower rates for need of RRT
- Target MAP is 65-70mmHg
- Patients with history of hypertension might benefit from MAP target of 80-85mmHg

Tx of Hyperkalemia

Drug	Indication	Dosage	Adverse Effects	Comments
Furosemide	Fluid overload, oliguria/anuria, hyperkalemia	2 to 5mg/kgIV bolus, may be repeated three to five times; 0.5 to 1mg/kg/hr CRI if urine production increased following bolus	Ototoxicity; volume depletion (unlikely if patient is monitored)	Results are frequently not satisfactory in cases of severe AKI but adverse effects minimal, so use in anuric AKI
Regular insulin	Hyperkalemia	0.5 units/kgIV or IM, may be repeated every 4 to 6 hours, provided hypoglycemia is avoided	Hypoglycemia	Hypokalemic effect modest and transient; IV dextrose must be administered concurrent with and following insulin administration
Dextrose	Hyperkalemia; avoidance of hypoglycemia following insulin administration	IV bolus of 2g/unit of insulin administered; bolus followed by CRI (the dextrose concentration and administration rate is dependent on serial blood glucose concentrations, patient's fluid status, and accessibility of central line)	Hyperglycemia, hyperosmolarity, hyponatremia, phlebitis with high dextrose concentrations	Dextrose should be diluted to avoid phlebitis; frequent changes in dextrose CRI frequently necessary based on serial blood glucose measurements
Calcium gluconate (10%)	Hyperkalemia; symptomatic hypocalcemia	0.5 to 1.5mL/kg of 10% solution or 50 to 150mg/kgIV slowly, to effect, while monitoring ECG; may be	Worsening bradycardia and ECG changes; hypercalcemia; soft tissue mineralization	ECG should be monitored during administration; will not affect extracellular potassium
Albuterol (inhaled)	Hyperkalemia	Four puffs; 90 microgram actuation via Aerokat device; repeated every 1 to 4 hours as necessary	Tachycardia, tremors, hyperexcitability	Adverse effects are uncommon at this dose; effects observed within 1 to 2 hours but may require multiple doses; effects can be sustained (several hours); only recommended for peracute hyperkalemia

Tx of Metabolic Acidosis

Drug	Indication	Dosage	Adverse Effects	Comments
Sodium bicarbonate	Severe acidemia	¼ to ⅓ of the base deficit over 30 to 60 minutes, followed by an additional ¼ over the next 4 to 6 hours; additional dosing based on serial blood gas analyses	Paradoxical central nervous system acidosis, hypernatremia, fluid overload, hypochloremia; may cause or exacerbate hypokalemia if patient is polyuric; may exacerbate hypocalcemia	Requires close monitoring of blood gases and electrolytes for effective treatment and avoidance of adverse effects

$$0.3 \times \text{body weight (kg)} \times \text{base deficit} = \text{bicarbonate (mEq)},$$

Timing for Renal Replacement Therapy

- Multiple studies have investigated in human medicine the ideal timing for RRT in AKI and S-AKI
  - IDEAL-ICU
  - START-AKI
  - ELAIN

Timing for Renal Replacement Therapy

- Guidelines are still unclear, but general understanding is hyperkalemia , fluid overload, and severe acidosis
- Most studies start with a a KDIGO stage of 2
- Most studies with S-AKI showed benefit in 90 day recovery and mortality with early start of RRT

Nutrition in AKI

- Adequate nutrition has been linked to better recovery rates in humans with AKI
- Dogs with severe AKI are commonly observed to lose 5–10% of their dry body weight during the first week of therapy
- Dogs with AKI have increased metabolic demand
- Goal is to administer 130% of resting energy requirement

Nutrition in AKI

- No evidence of positive effect for protein restriction
- Optimal dietary composition for veterinary AKI has not been determined
- Patients on hemodialysis require increased protein supplementation due to loss of amino acids in dialysate
- 1.5% x RER has been suggested for most patients on hemodialysis treatment

Nutrition in AKI

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Summary

- Prognosis is grim
- We now have a validated, consensus definition
  - R isk
  - I njury
  - F ailure
  - L oss of function
  - E srd
- Outpatient and inpatient acquired ARF differ in etiology
- Hospital acquired disease is our fault

Summary

- Prevention is the best therapy of AKI
- Use early dialysis if anuric
- Do not fluid overload your patient
- Dopamine doesn't work

Thank You

Thank you!

CANADA WEST
VETERINARY SPECIALISTS