Insuffisance Rénale Aiguë

Définition, monitorage et traitement NON dialytique

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25 octobre 2014

Définition de l'IRA

- Défaillance rapide (quelques heures, qq jours) des fonctions glomérulaires et tubulaires,
- due à des agresseurs
- isolés ou plus souvent multiples.

Definition

AKI is defined as any of the following (*Not Graded*):

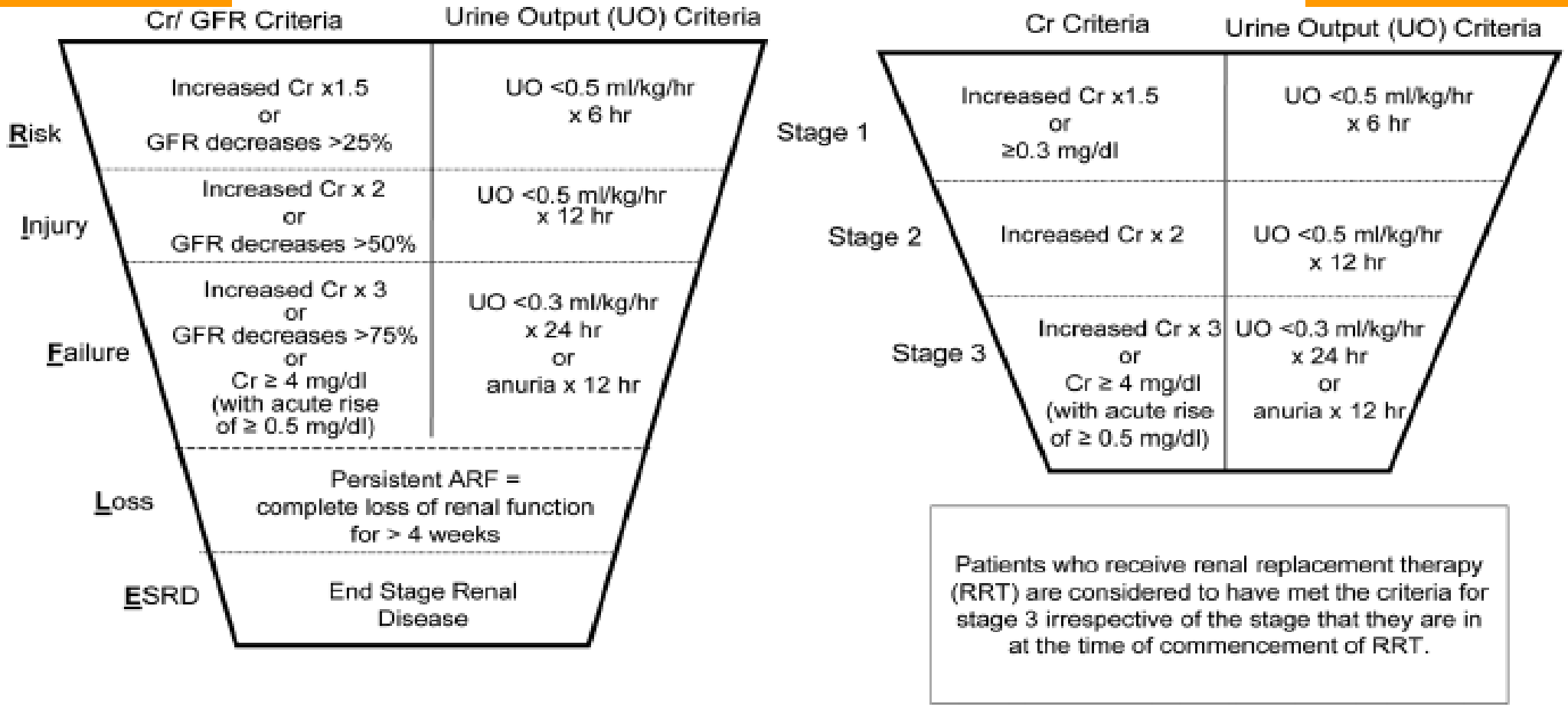
- Increase in SCr by >0.3 mg/dl within 48 hours; or
- Increase in SCr by >1.5-fold above baseline, which is known or presumed to have occurred within 7 days; or
- Urine volume <0.5 ml/kg/h for 6 hours.

The cause of AKI should be determined whenever possible.

Figure 1

< 7 j

< 48h



RIFLE and AKIN classifications for acute kidney injury. Risk–Injury–Failure–Loss–Endstage renal disease (RIFLE) and Acute Kidney Injury Network (AKIN) classifications for acute kidney injury (adapted from [6,7]). ARF, acute renal failure; Cr, creatinine; GFR, glomerular filtration rate.

Table 2. Pediatric Modified RIFLE (pRIFLE) Criteria for Diagnosis and Classification of AKI in Children

Class	eCCr	Urine Output
Risk	eCCr decrease by >25%	Urine output <0.5 mL/kg/h for >8 h
Injury	eCCr decrease by >50%	Urine output <0.5 mL/kg/h for >16 h
Failure	eCCr decrease by >75%; or eCCr <35 mL/min/1.73 m^2	Urine output <0.3 mL/kg/h for >12 h; or anuria for >12 h
Loss	Persistent failure for >4 wk	
End Stage	Persistent failure for >3 mo	

Abbreviations and definitions: AKI, acute kidney injury; eCCr: estimated creatinine clearance using the Schwartz formula; RIFLE, risk, injury, failure, loss, end-stage disease.

Am J Kidney Dis. 2013;61(5):649-672

Patients plus à risque d'IRA

TERRAIN	CIRCONSTANCES	NEPHROTOXIQUES
Age, sexe F	Sepsis	Produits CI
Diabète, cancer	Type de Chirurgie	Antibiotiques
Insuf cardiaque Artériopathie MI	Etat de choc	Chimiothérapie
Hypoalbuminémie	Transplantation	Empoisonnement
IRC	Troisième secteur	ISRA, AINS
	Ventilation artif	

Murugan, R. & Kellum, J. A. *Nat. Rev. Nephrol.* 7, 209–217 (2011)

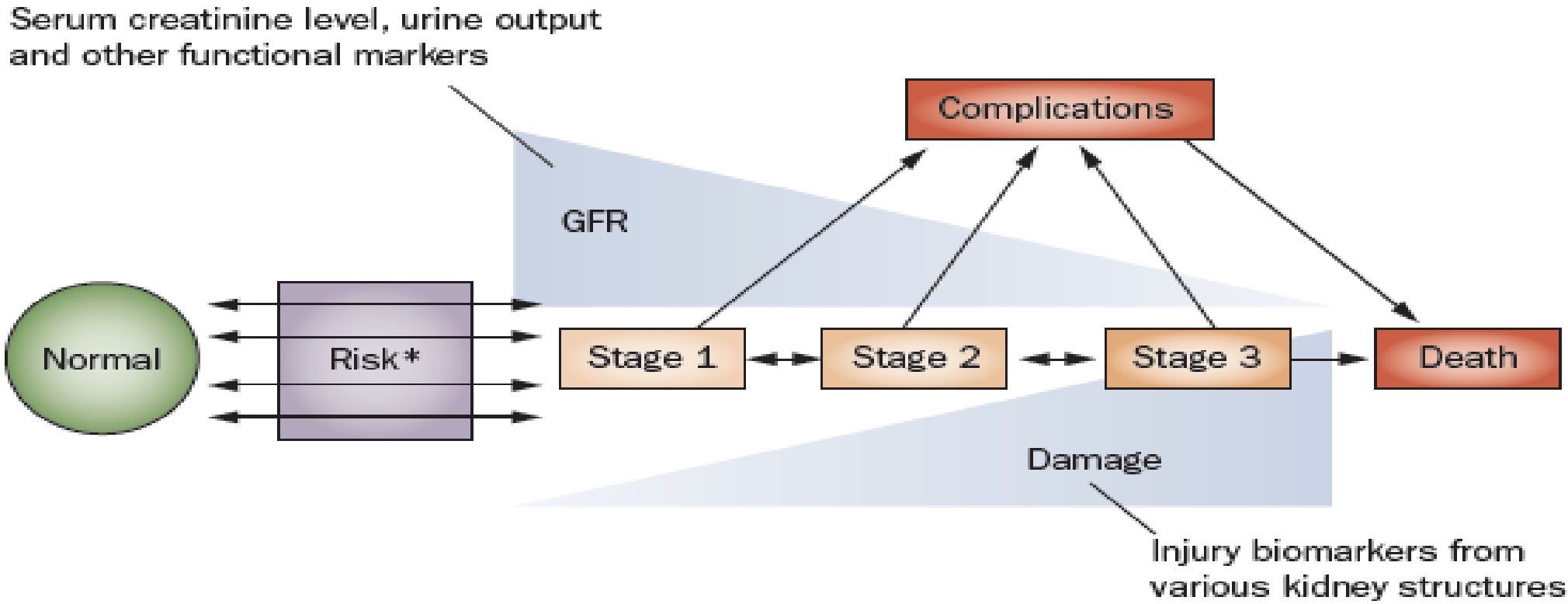


Figure 1 | Conceptual model of AKI. The new conceptual model of AKI incorporates changes in renal function and structure. It also illustrates the potential inverse relationship that may exist between changes in renal function as well as renal structure as captured by injury biomarkers. *Risk incorporates both patient susceptibilities (for example, advanced age) as well as exposures (for example, sepsis). When susceptibilities are great, exposure may be limited but still result in AKI. Abbreviations: AKI, acute kidney injury; GFR, glomerular filtration rate.

Long term outcome of patients with severe AKI (Oeyen et al Acta Clin Belgica 2007;62)

- AKI in ICU: 30-60%
- Hospital mortality: 45-71%
- Mortality at 1y: 57-78%

- Renal recovery :

  in most cases of survivors with normal renal function before AKI but 10% develop CKD

Figure 2. Mortality in AKI patients admitted to the intensive care unit. Mortality could be traced for all patients at 1 and 2 years after discharge. Delayed mortality = mortality during the first/second year after release.

Clin J Am Soc Nephrol 5: 1755–1762, 2010.

Epidemiology of AKI CJASN2008

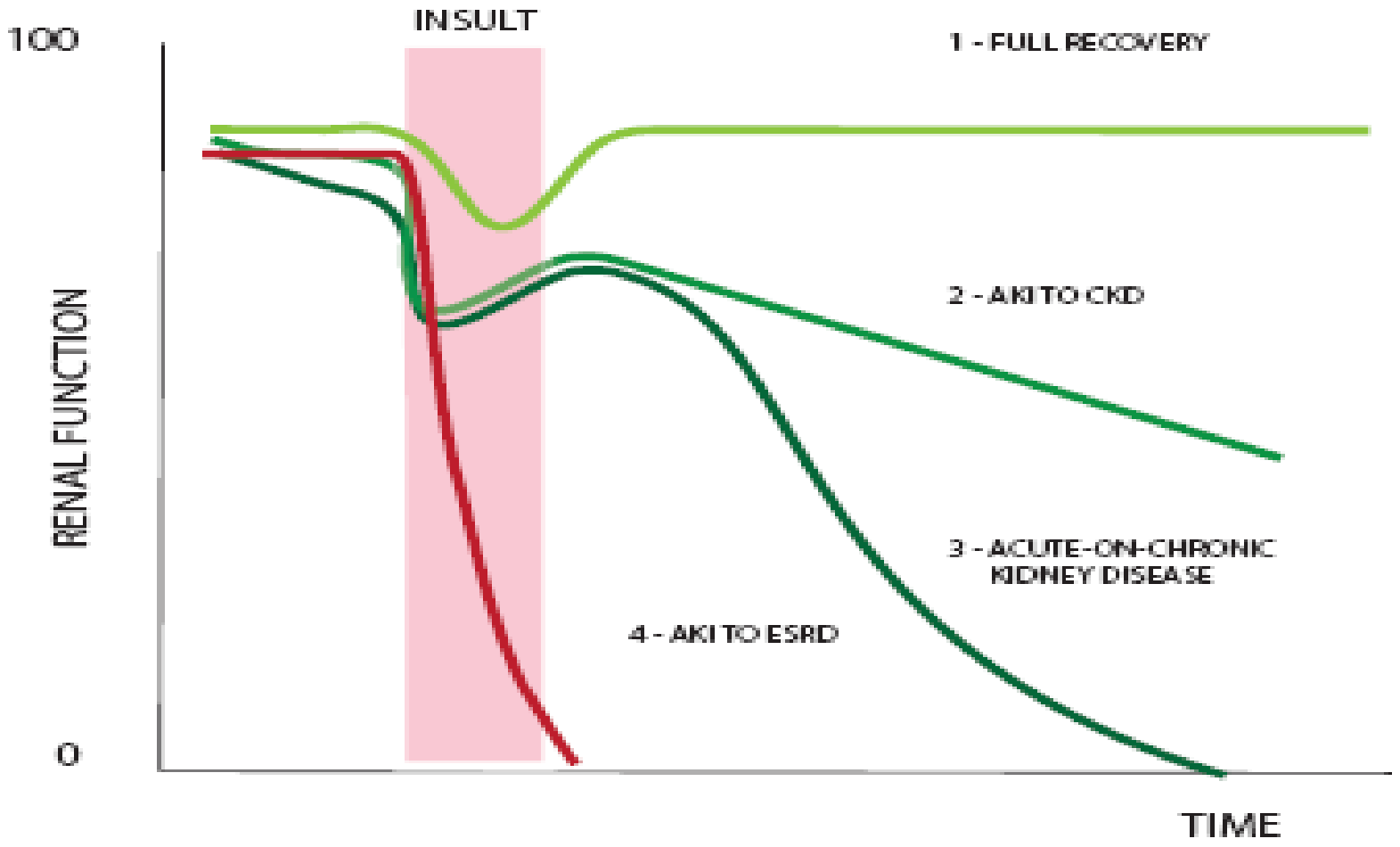


Figure 2. Natural history of AKI. Patients who develop AKI may experience (1) complete recovery of renal function, (2) development of progressive chronic kidney disease (CKD), (3) exacerbation of the rate of progression of preexisting CKD; or (4) irreversible loss of kidney function and evolve into ESRD.

Importance de l'IRA chez l'hospitalisé
CJASN 2006

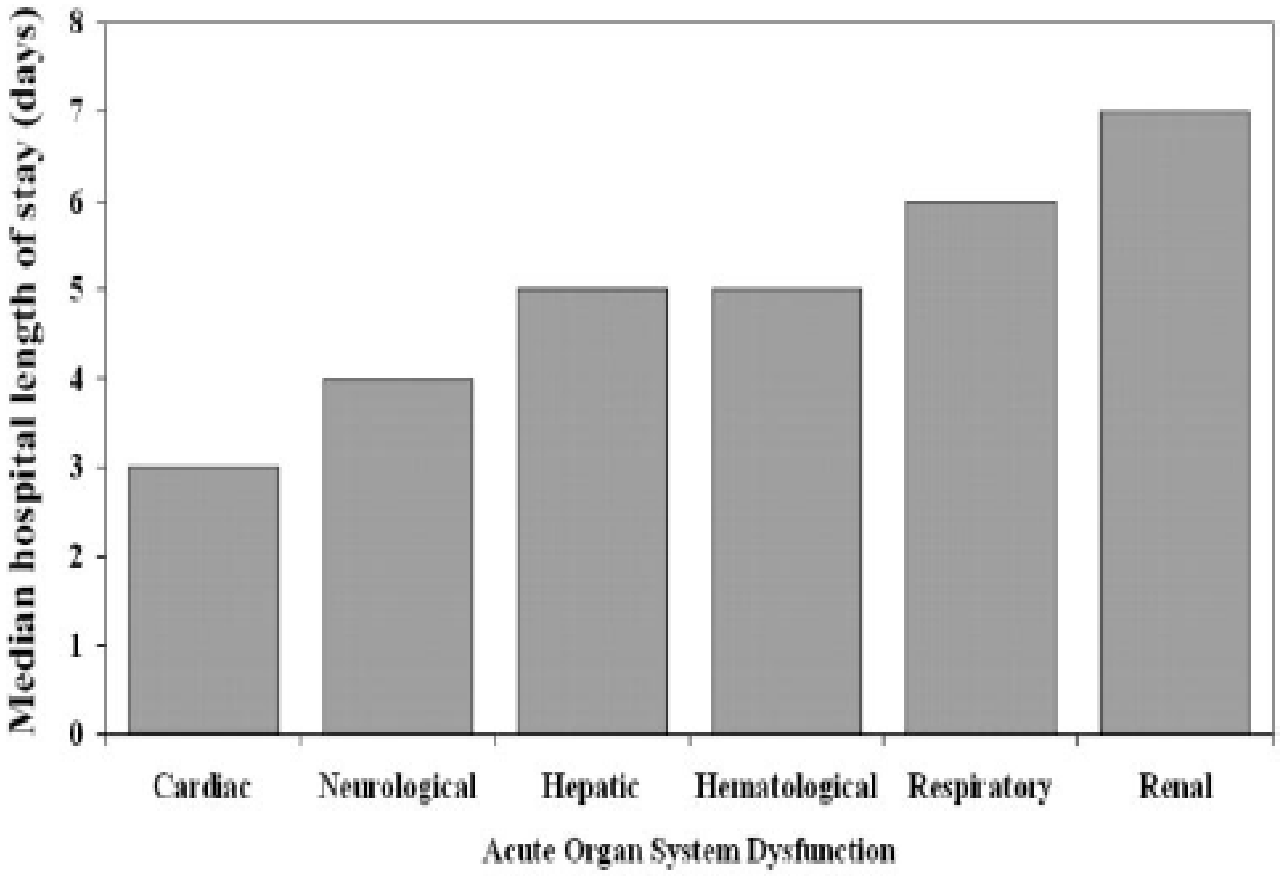


Figure 1. Median hospital length of stay (LOS) stratified by single acute organ system dysfunction (AOSD), including acute renal failure (ARF).

Mechanisms of Acute renal failure

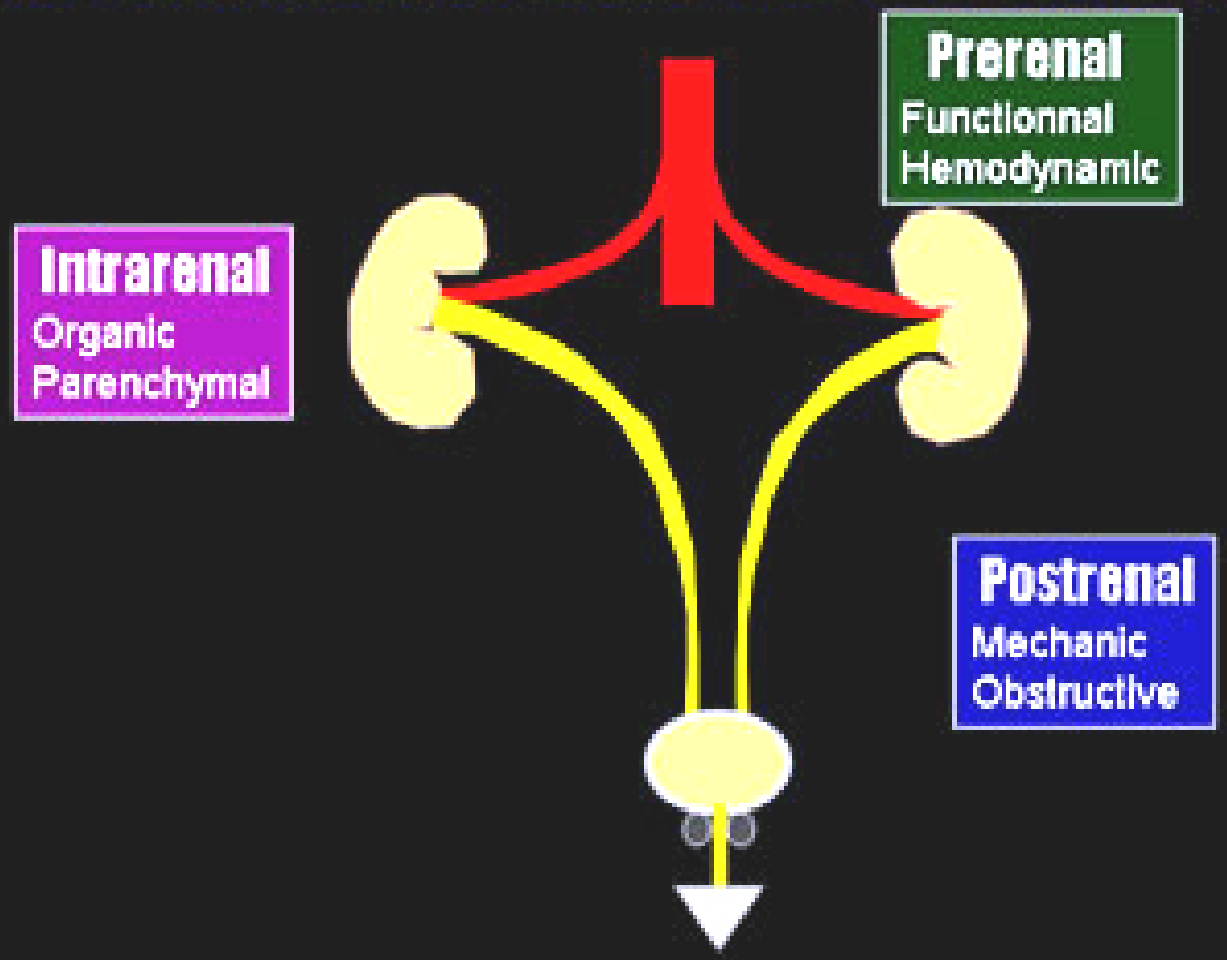
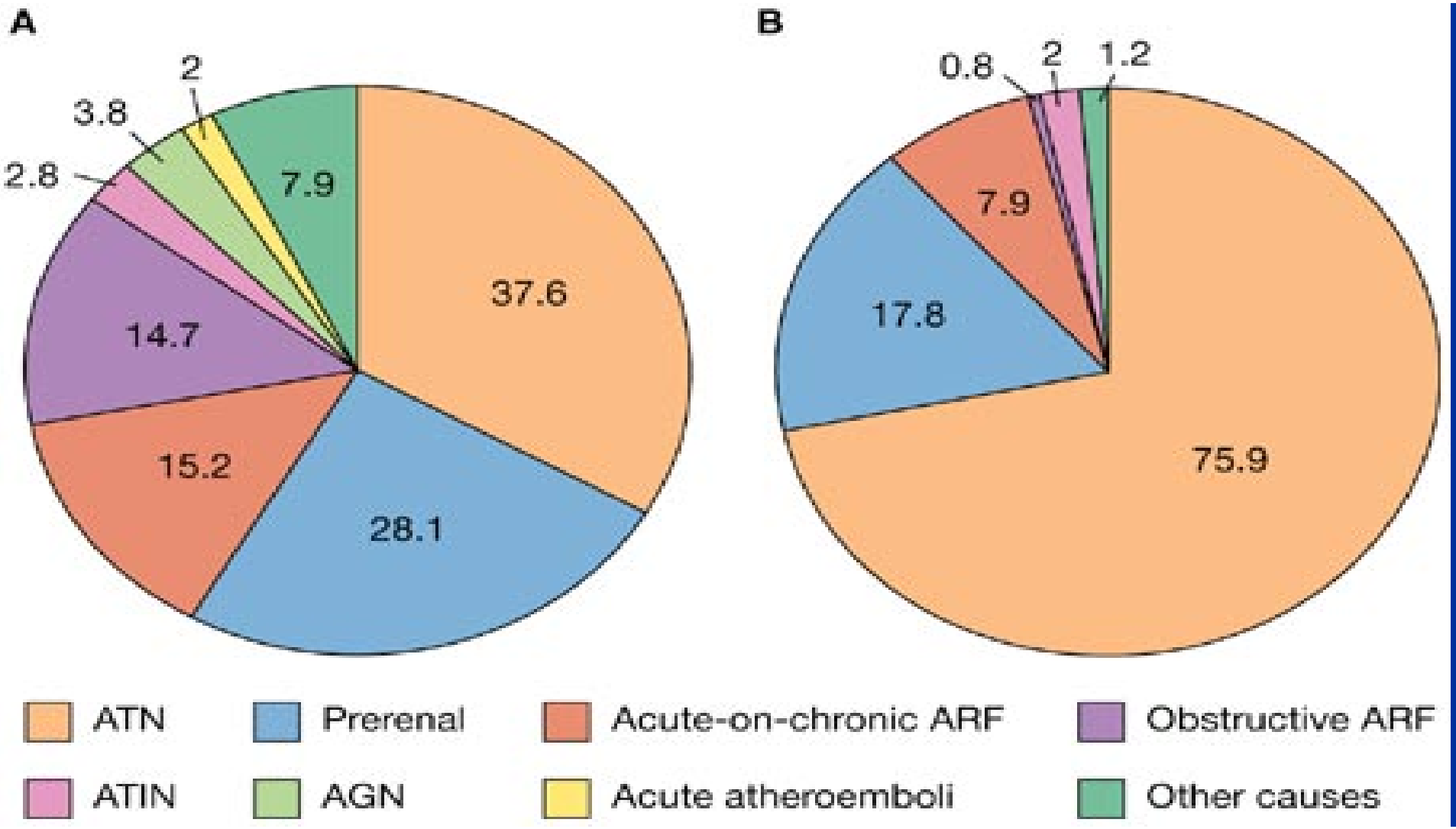


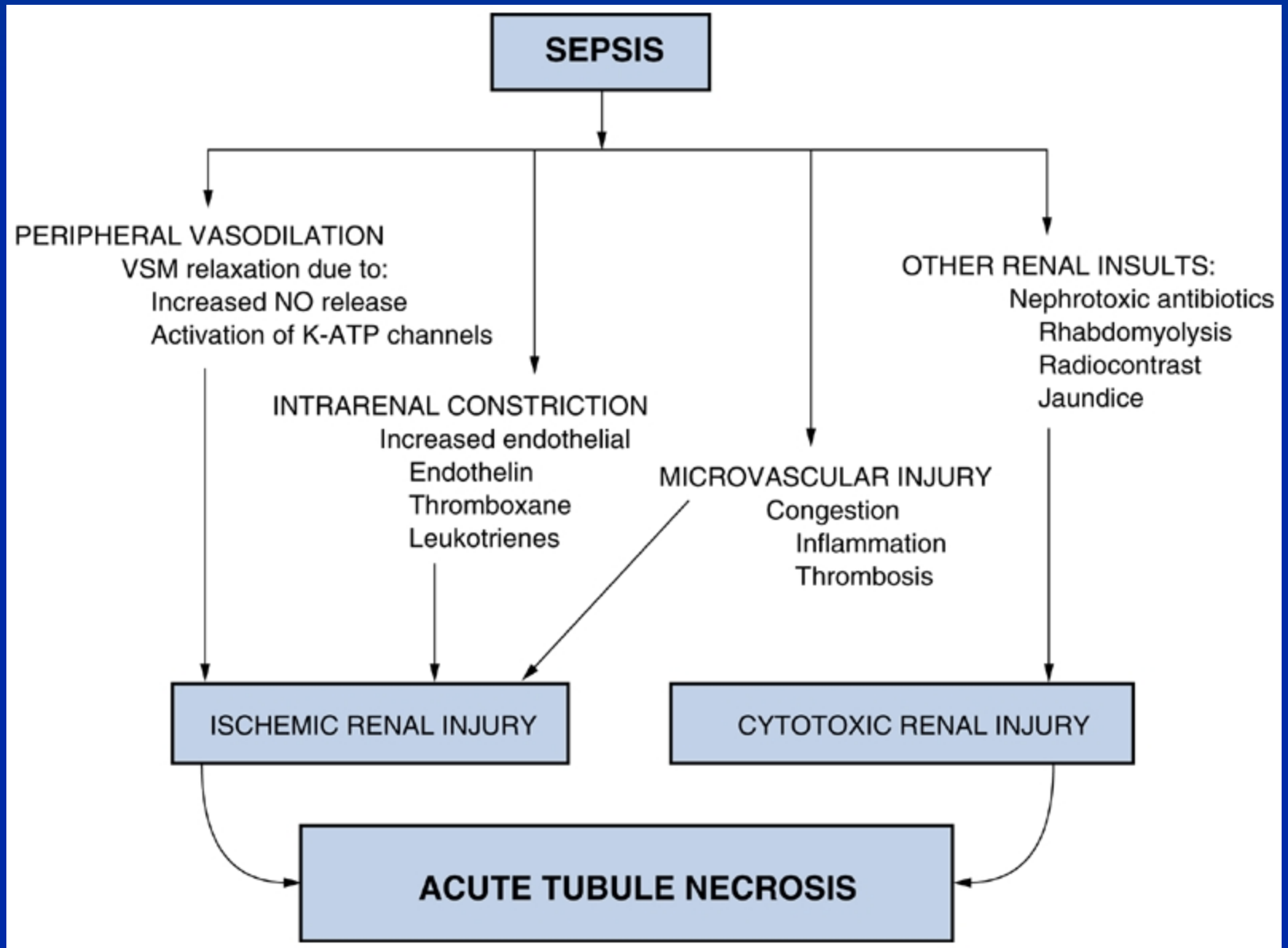
Figure 2 Percentage distribution of causes of acute renal failure in (**A**) non-ICU and (**B**) ICU settings



Modified with permission from Liano F *et al*. (1998) The spectrum of acute renal failure in the intensive care unitcompared with that seen in other settings. The Madrid Acute Renal Failure Study Group. *Kidney Int Suppl* **66**: S16–S24. © (1998) Nature Publishing Group.

Causes d'IRA aux SI

1. Sepsis (35-50%)
2. Chirurgie (25%)
3. Post-traumatique (crush S 25%))
4. Produits de contraste (5-10%)
5. Pathologie hépatique (20%)
6. Hyperpression abdominale
7. Nephrotoxiques



(From Abernethy VE, Lieberthal W: Acute renal failure in the critically ill patient. Crit Care Clin 18:203-222, 2002.)

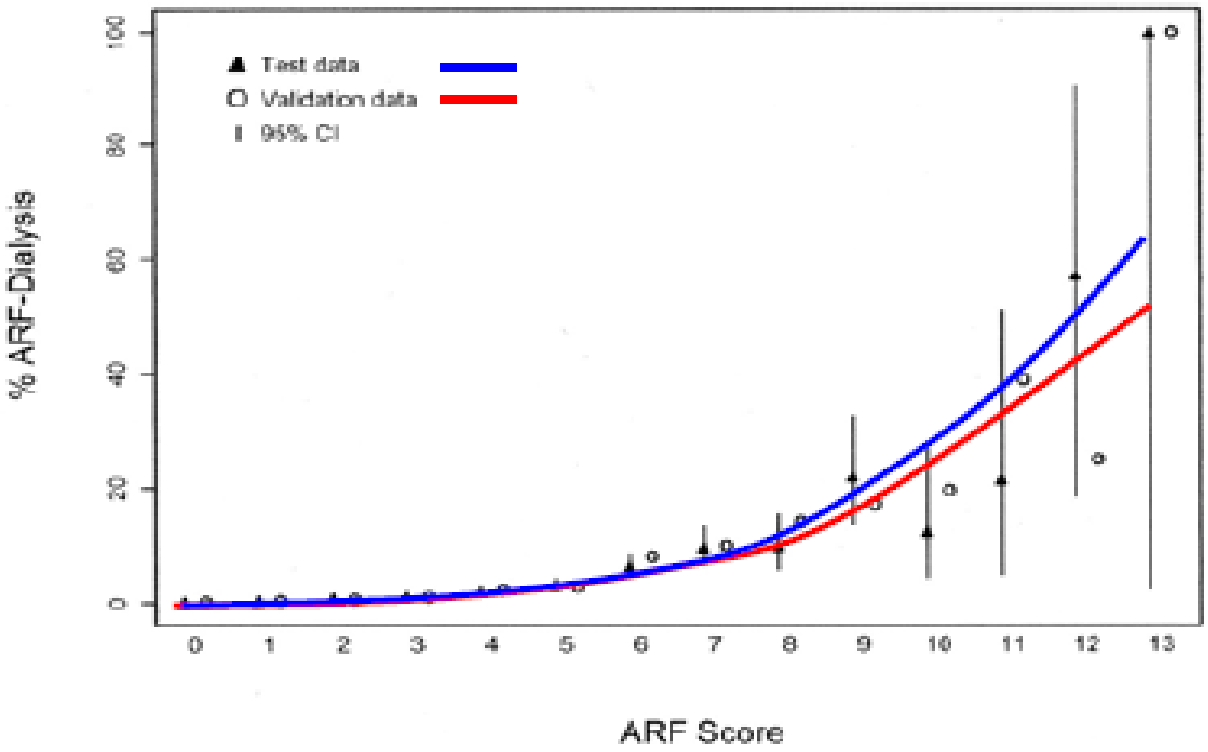
Causes d'IRA en chirurgie

- ISCHEMIQUE:
  - toutes les causes prérénales
  - chirugie aortique, cardiaque, hépato-biliaire
- TOXIQUE:
  - antibiotiques, produit de contraste
- PIGMENTS:
  - hémolyse, rhabdomyolyse

Risk Assessment of ARF Post CABG (1992-2002)

Risk factors	Score
Female gender	1
Congestive heart failure	1
Left ventricular ejection fraction <35%	1
Preoperative use of IABP	2
COPD	1
Insulin-requiring diabetes	1
Previous cardiac surgery	1
Emergency surgery	2
Valve surgery only (reference to CABG)	1
CABG + valve (reference to CABG)	2
Other cardiac surgeries	2
Preoperative creatinine 1.2 to <2.1 mg/dl (reference to 1.2)	2
Preoperative creatinine 2.1 (reference to 1.2)	5

Minimum score, 0
Maximum score, 17



C. Thakar J. Am Soc. Nephrol 16:162-168, 2005

Predictive Models for Acute Kidney Injury Following Cardiac Surgery

Am J Kidney Dis. 59(3):382-389. © *2012*

Study Design: Prospective observational cohort.

Settings & Participants: 25,898 patients who underwent cardiac surgery at Cleveland Clinic in 2000-2008.

	Doubling of SCr or Dialysis	*P*	Dialysis	*P*
Variable (reference)				
Race (white)		0.007		<0.001
Black	1.35 (1.06-1.72)		1.77 (1.20-2.62)	
Other	1.33 (1.03-1.73)		1.94 (1.31-2.89)	
Body mass index	1.03 (1.02-1.04)	<0.001		
Pulmonary disease	1.22 (1.02-1.47)	0.04	1.65 (1.26-2.15)	<0.001
Congestive heart failure	1.40 (1.22-1.62)	<0.001		
Diabetes mellitus	1.59 (1.32-1.91)	<0.001	1.50 (1.12-2.00)	0.006
Hypertension	1.23 (1.05-1.43)	0.003		
Emergent surgery	2.43 (1.97-3.00)	<0.001	1.95 (1.42-2.68)	<0.001
Estimated GFR[a]	0.98 (0.98-0.99)	<0.001	0.24 (0.18-0.32)[b]	<0.001
Estimated GFR[2,a,c]	1.0001 (1.00009-1.00013)	<0.001		
Albumin	0.71 (0.62-0.81)	<0.001	0.70 (0.58-0.84)	0.001
Potassium			1.29 (1.04-1.59)	0.02
Bicarbonate	0.97 (0.95-0.99)	0.002		
Sodium	0.98 (0.96-0.998)	0.06		
Serum urea nitrogen	1.01 (1.005-1.02)	<0.001	1.02 (1.01-1.02)	<0.001
Hemoglobin	0.97 (0.93-1.003)	0.08		
Platelet count	0.998 (0.997-0.999)	0.001		
Bilirubin[b]			1.45 (1.22-1.71)	<0.001
CPB time (≤80 min)		<0.001		<0.001
81-120 min	1.21 (0.997-1.48)		1.30 (0.89-1.90)	
121-150 min	1.34 (1.07-1.67)		1.52 (1.01-2.29)	
151-180 min	1.31 (1.01-1.71)		2.09 (1.36-3.21)	
>180 min	2.67 (2.07-3.43)		3.85 (2.54-5.82)	
Bypass not used	0.90 (0.66-1.24)		1.17 (0.68-2.00)	
Intrasurgical packed RBC transfusions (none)		<0.001		<0.001
1-2 units	1.48 (1.23-1.79)		1.75 (1.26-2.43)	
3-4 units	2.22 (1.80-2.74)		2.86 (2.05-3.99)	
5-6 units	3.25 (2.49-4.24)		4.18 (2.82-6.19)	
>6 units	6.53 (5.08-8.40)		9.24 (6.44-13.28)	
Intrasurgical vasopressor use	1.35 (1.16-1.58)	<0.001	2.22 (1.64-3.00)	<0.001
Intrasurgical urine output[b]	0.76 (0.71-0.81)	<0.001	0.76 (0.69-0.84)	<0.001

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Potassium			1.59)	0.02
Bicarbonate				
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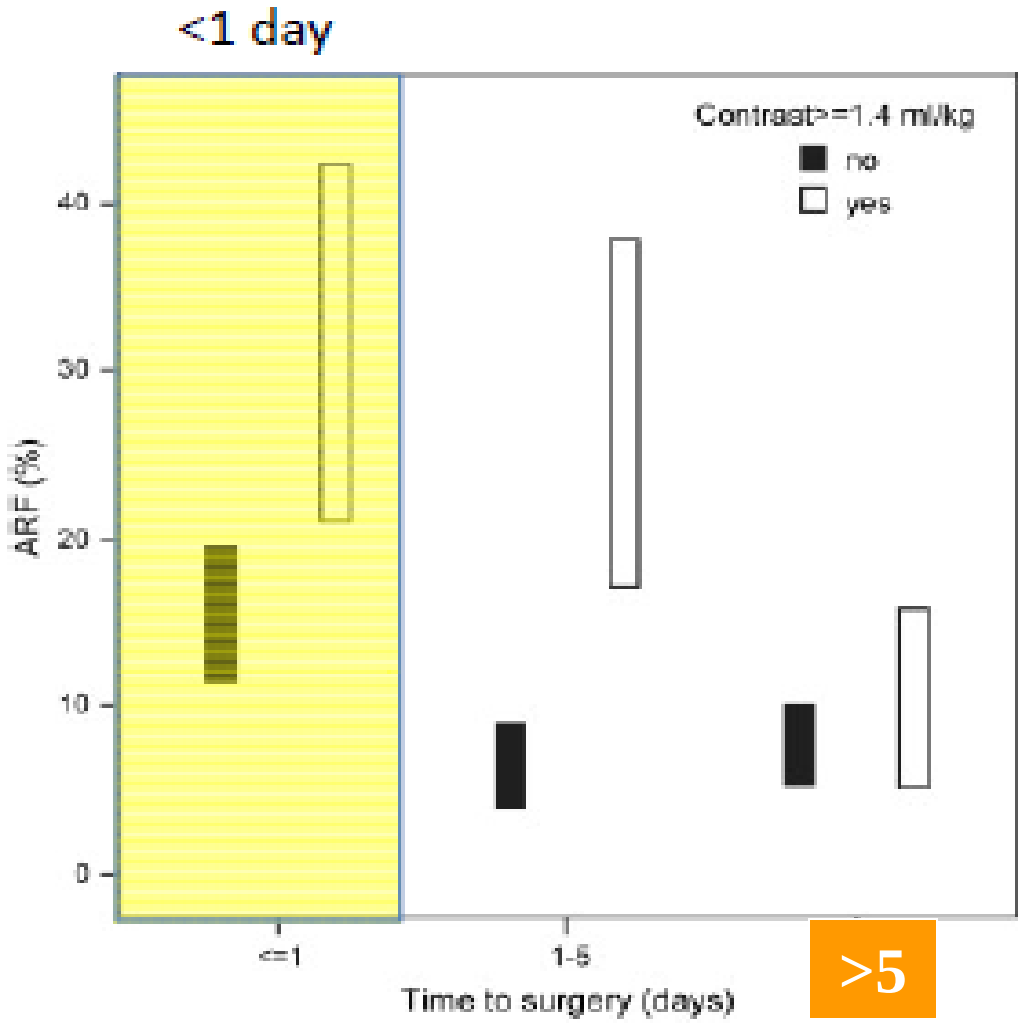
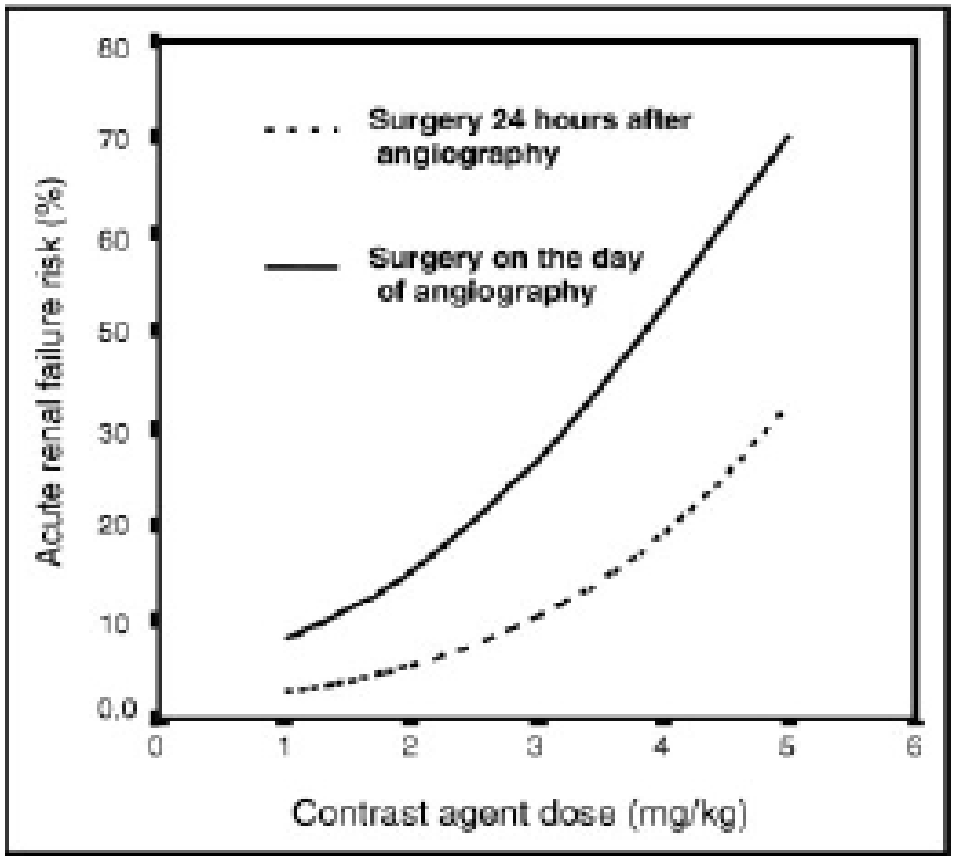
Comorbidités
CKD
Urgence, chirurgie multiple
Durée CEC
Nb unités transfusions

AKI post cardiac surgery

Timing of angiography and contrast dose



B. Medalion et al. (J Thorac Cardiovasc Surg in press 2010)



M. Ranucci, et al. Am J. Cardiol. 101:1112-1118 2008

Preventing nephropathy induced by CM

Barrett and Parfrey NEJM 2006

Table 24. CIN risk-scoring model for PCI

Risk factors	Integer score (calculate)
Hypotension	5
IABP	5
CHF	5
Age >75 years	4
Anemia	3
Diabetes	3
Contrast-media volume	1 per 100 ml
SCr >1.5 mg/dl *or*	4
eGFR <60 ml/min per 1.73 m^2	2 for 40-60 4 for 20-39 6 for <20

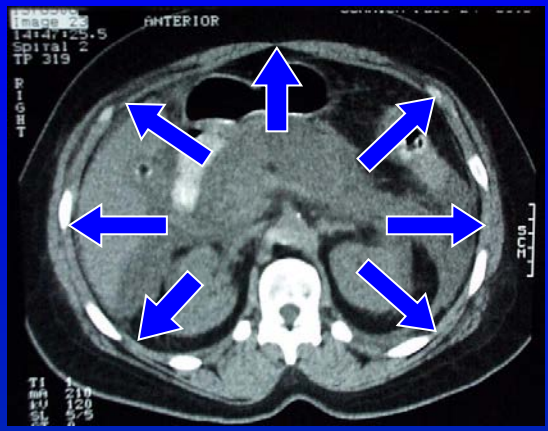
Preventing nephropathy induced by CM

Barrett and Parfrey NEJM 2006

Total Risk Score*	Risk of an Increase in Serum Creatinine Levels of >0.5 mg/dl (44 μmol/liter) or >25 Percent	Risk of Dialysis
	percent	
≤5	7.5	0.04
6 to 10	14.0	0.12
11 to 15	26.1	1.09
≥16	57.3	12.6

Importance of INTRA-ABDOMINAL PRESSURE?

- **"Intra-abdominal pressure (IAP) is the steady-state pressure concealed within the abdominal cavity."**
  - Elevated IAP is a common finding in the ICU
  - IAP increases and decreases with respiration
  - IAP is directly affected by:
    1. Solid organ or hollow viscera volume
    2. Space occupying lesions
       - Ascites, blood, fluid, tumors
    3. Conditions that limit expansion of the abdominal wall
       - Burn eschars, third-space edema



RECOMMENDATIONS: RISK FACTORS & SURVEILLANCE FOR IAH/ACS

Patients should be screened for IAH / ACS risk factors upon ICU admission and in the presence of new or progressive organ failure (Grade 1B)

- Independent risk factors for IAH / ACS include:
  - Large volume fluid resuscitation (> 3.5 L / 24 hrs)
  - Acidosis
  - Hypothermia
  - Coagulopathy / polytransfusion
  - Pulmonary, renal, hepatic dysfunction
  - Ileus
  - Abdominal surgery / primary fascial closure

Drug use and nephrotoxicity in the intensive care unit

Mark A. Perazella[1]

Table 4 | Common forms of drug-induced AKI in the ICU

Hemodynamic AKI
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- RAAS inhibitors
- Calcineurin inhibitors (cyclosporine, tacrolimus)
- Vasopressors

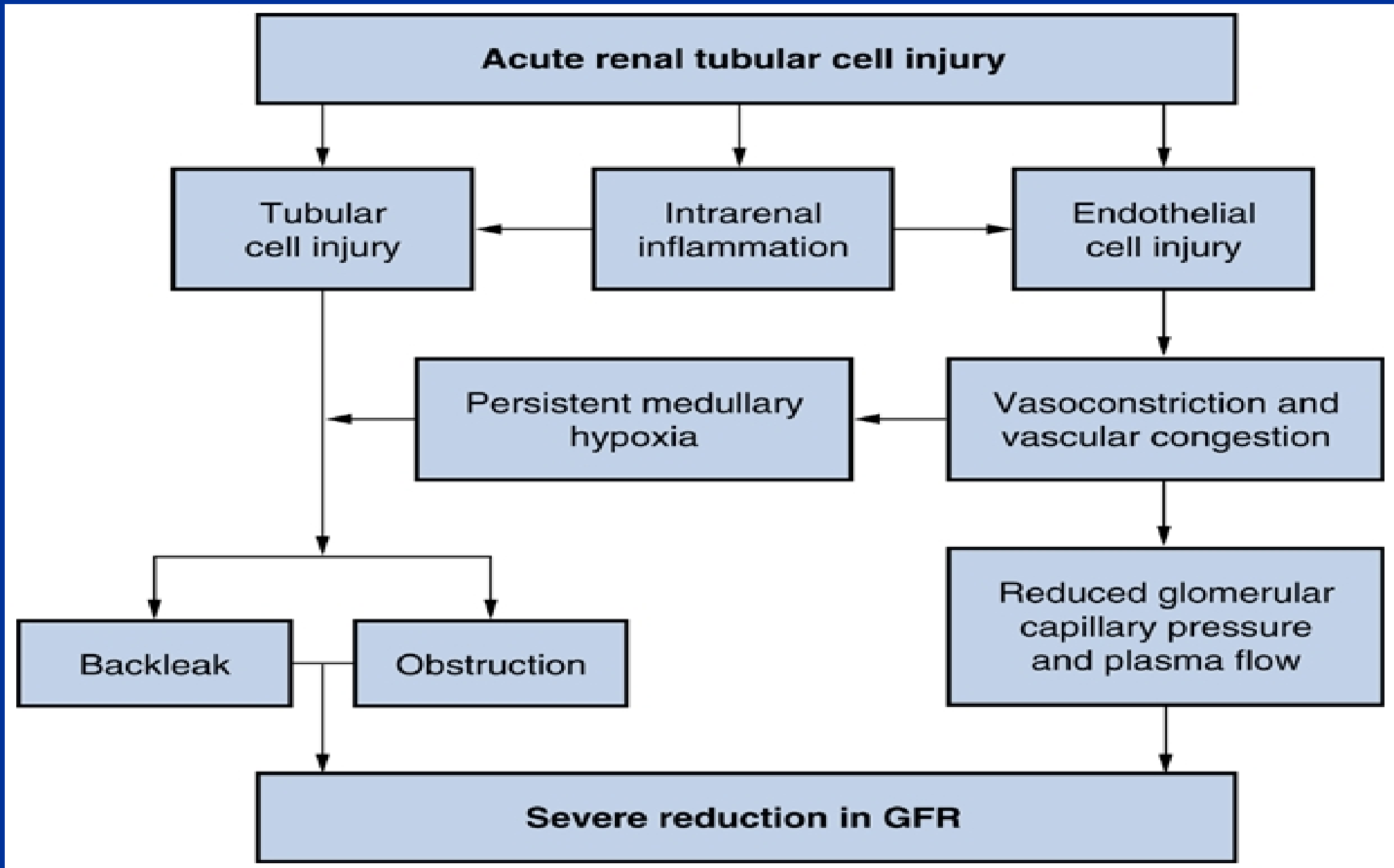
Acute tubular necrosis
- Radiocontrast
- Nephrotoxic antimicrobials

Osmotic nephropathy
- Hydroxyethyl starch (HES)
- Intravenous immunoglobulin (IVIG containing sucrose)

Crystal nephropathy
- Highly active anti-retroviral therapy (HAART)
- Acyclovir
- Ciprofloxacin
- Sodium phosphate purgatives

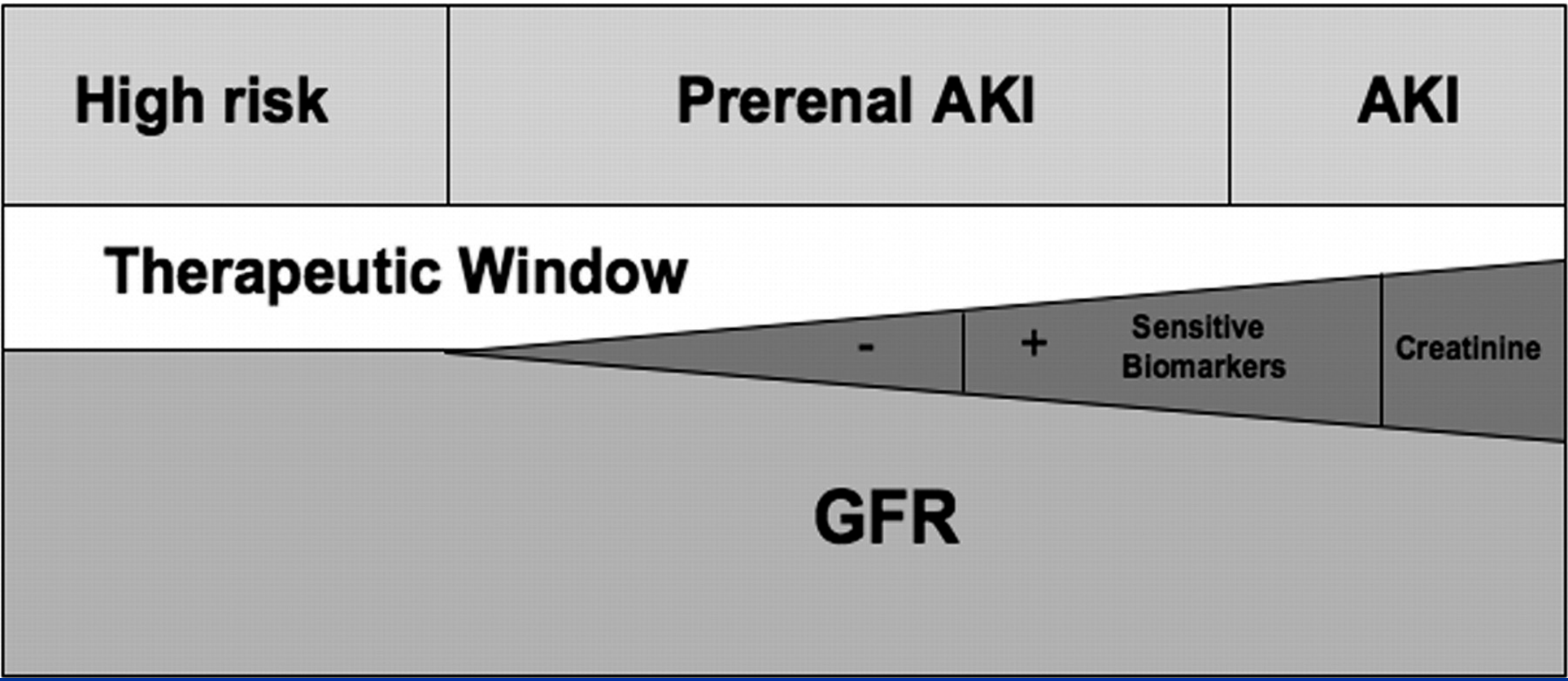
Acute interstitial nephritis
- Antibiotics (β-lactams, sulfa-based, quinolones)
- Proton pump inhibitors, H_2 antagonists
- Anti-convulsants

Abbreviations: AKI, acute kidney injury; H_2, histamine-2; ICU, intensive care unit; NSAIDs, nonsteroidal anti-inflammatory drugs; RAAS, rennin–angiotensin–aldosterone system.



Narrow Therapeutic Window

Need for Sensitive Biomarkers

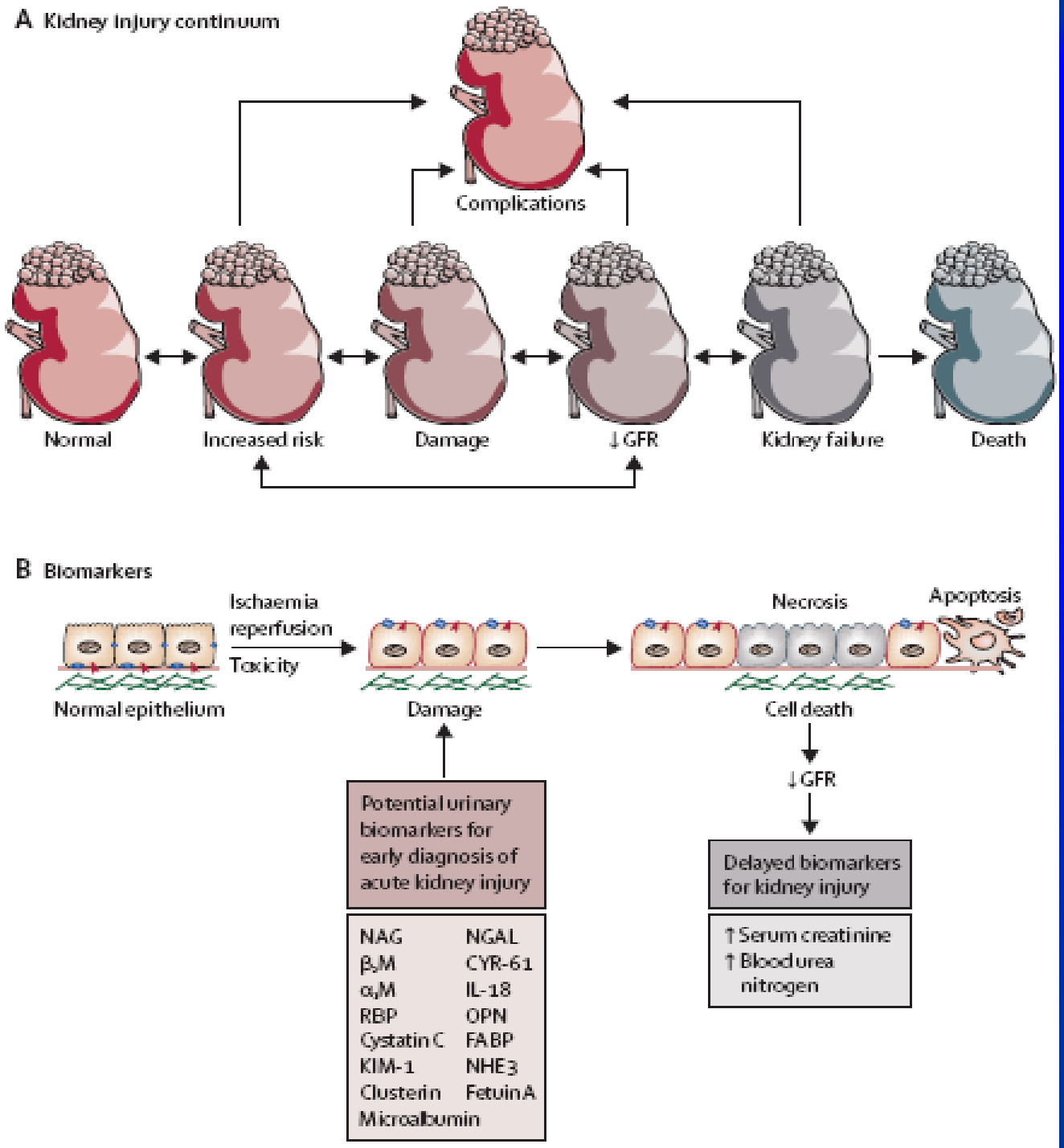


Jo, S. K. et al. Clin J Am Soc Nephrol 2007;2:356-365

Table 18. Urinary indices

Indices	Prerenal	Renal
Urine sediment	Hyaline casts	Abnormal
Specific gravity	>1.020	~1.010
Urine osmolality (mOsm per kg H_2O)	>500	<350
U_{Na} (mmol/L)	<20	>40
Fractional excretion		
Sodium (%)	<1	>2
Urea (%)	<35	>35
Uric acid (%)	<7	>15
Lithium (%)	<7	>20
Low molecular weight proteins	Low	High
Brush border enzymes	Low	High

www.thelancet.com Vol 372 November 29, 2008



Biomarkers kinetics in AKI

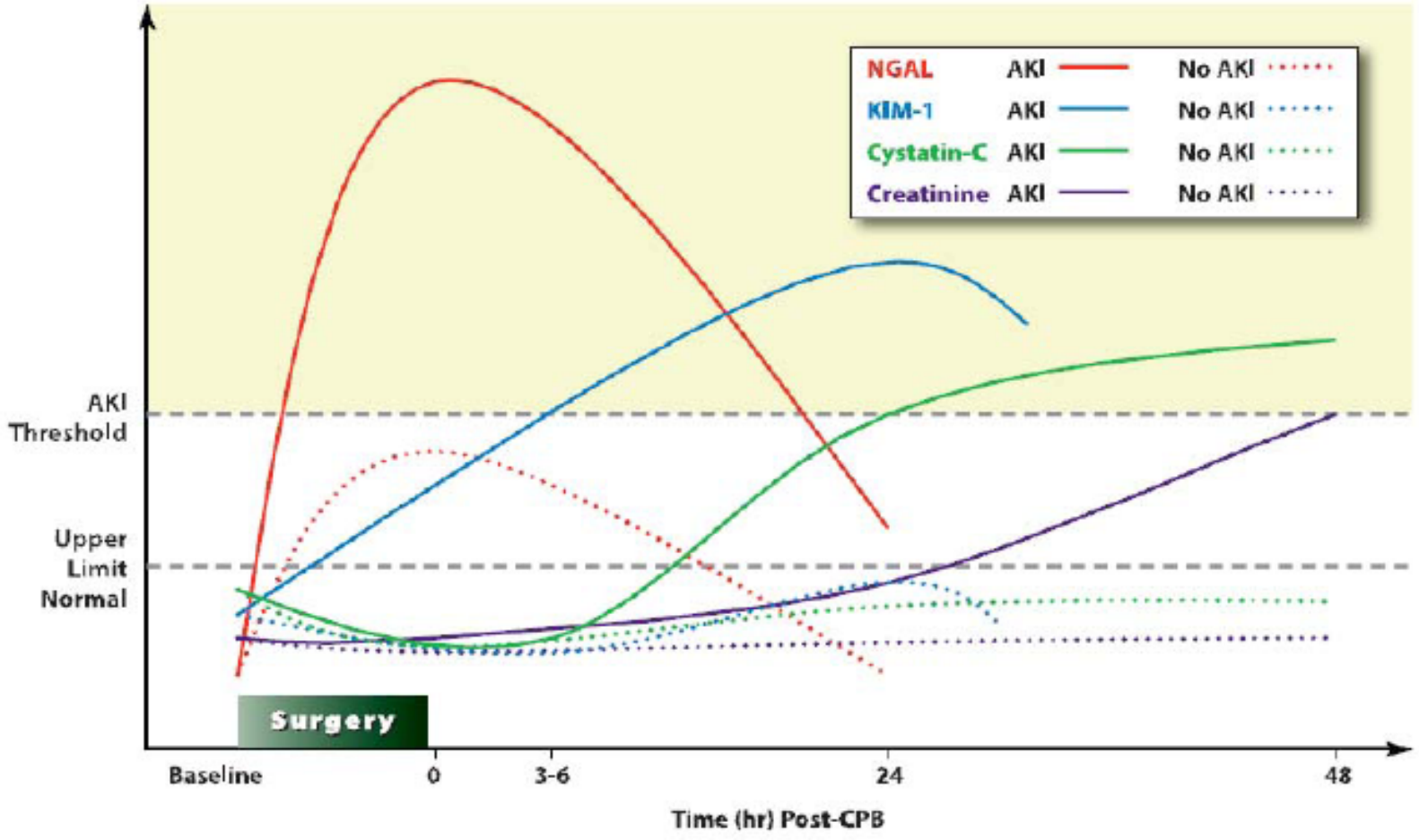


Figure 22. The theoretical evolution of the time course of several biomarkers in AKI following cardiac surgery. AKI, acute kidney injury; KIM-1, kidney injury molecule 1; NGAL, neutrophil gelatinase–associated lipocalin. Reprinted with

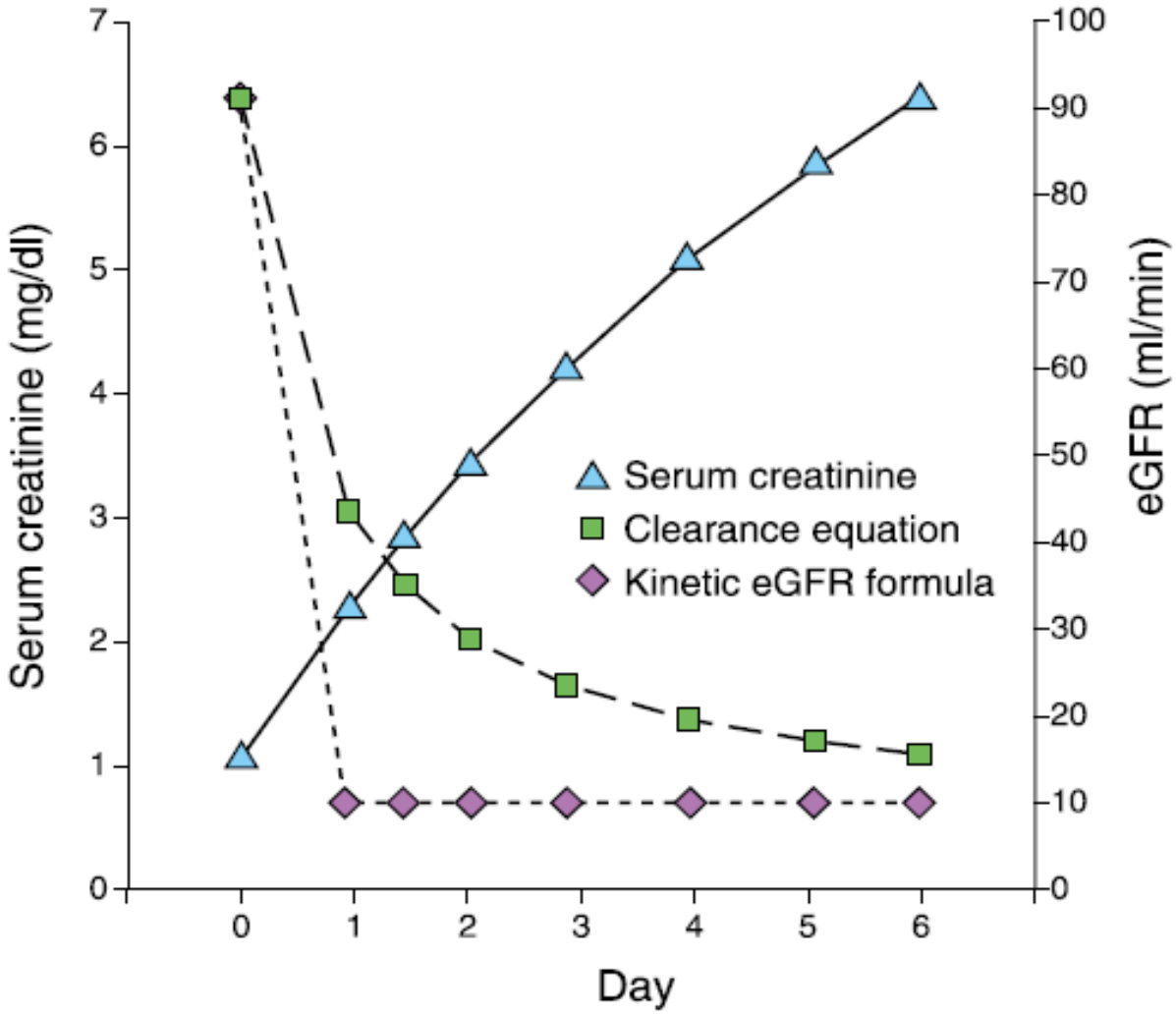


Figure 1. Graph of the plasma creatinine rise in AKI and the corresponding clearances given by the kinetic formula and the clearance equation. Table 1 is plotted for the plasma creatinine (▲) and the formula-derived eGFR (◆), along with the value that would be obtained by the clearance equation (■). For a typical rate of creatinine rise in severe AKI, the eGFR as predicted by the kinetic formula declines immediately and then remains diminished the rest of the time. On the other hand, the eGFR as interpreted through the clearance equation declines more gradually and slowly approaches the true magnitude of the renal function impairment. In fairness, the clearance equation in its classic form is not equipped to handle the nonsteady state.

J Am Soc Nephrol 24: 877–888, 2013.

Retooling the Creatinine Clearance Equation to Estimate Kinetic GFR when the Plasma Creatinine Is Changing Acutely

Table 1. Example of an increasing plasma creatinine in step decrement AKI

Day	Hours between Creatinine Measurements	Plasma Creatinine (mg/dl)	Kinetic eGFR Formula (ml/min)
0.00		1.10	91
	23		
0.96		2.29	10.17
	12		
1.46		2.85	9.87
	14		
2.04		3.45	9.99
	20		
2.88		4.22	10.02
	26		
3.96		5.09	9.99
	27		
5.08		5.86	9.94
	22		
6.00		6.39	10.04

The plasma creatinine rises precipitously in severe AKI, usually by >0.5 mg/dl per day. The differing numbers of hours between creatinine measurements are listed, resulting in fractional days, but 6 days of data are shown. Between successive pairs of creatinines, the eGFR by the kinetic formula has been calculated in the last column. The rate of creatinine rise starts to slow down eventually, but the abrupt drop in kidney function persists, reminiscent of the shape of a step, hence step decrement.

$$KeGFR = \frac{SSP_{Cr} \times CrCl}{MeanP_{Cr}} \times \left(1 - \frac{24 \times \Delta P_{Cr}}{\Delta Time(h) \times Max\Delta P_{Cr}/Day}\right)$$

$$= \frac{1.10 \times 91}{1.695} \times \left(1 - \frac{24 \times 1.19}{23 \times 1.5}\right)$$

$$= 10.17 \frac{ml}{min}$$

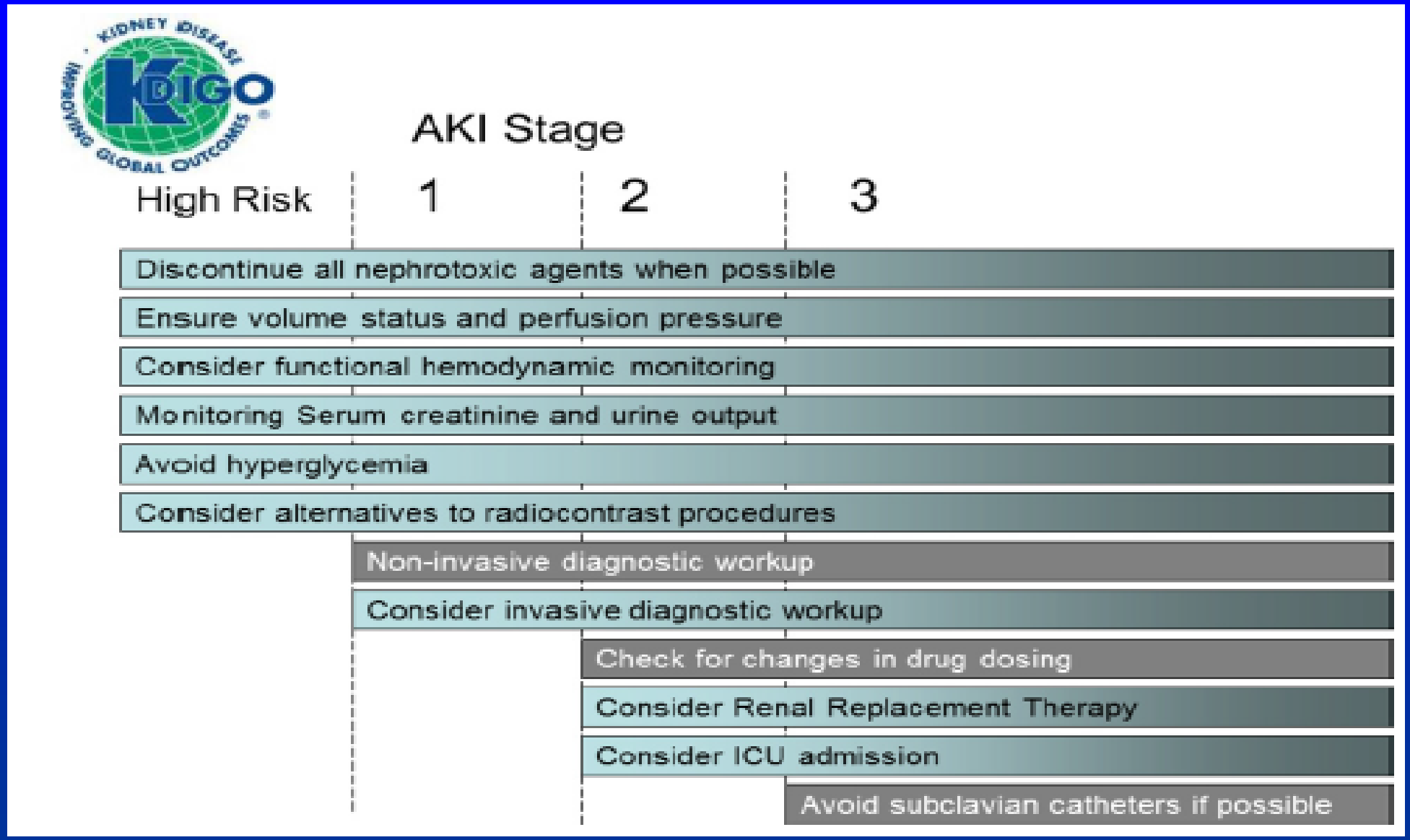
Intensive Care Med (2010) 36:379–380
DOI 10.1007/s00134-009-1683-1

EDITORIAL

Andrew Davenport

Clinical guidelines for the protection of kidney function and prevention of acute kidney injury in the intensive care unit: common sense rather than magic bullets?

KDIGO AKI guidelines 2010



Useful prophylactic interventions

Optimize hemodynamic and oxygenation

Use vasopressors + fluids in patients with vasomotors shock (mainly NE)

- Dopamine: no (risk of arythmia)
- Furosemide: only for treating fluid overload
- Control of glycemia: avoid too strict control
- Realize a monitoring of intra-abdominal pressure

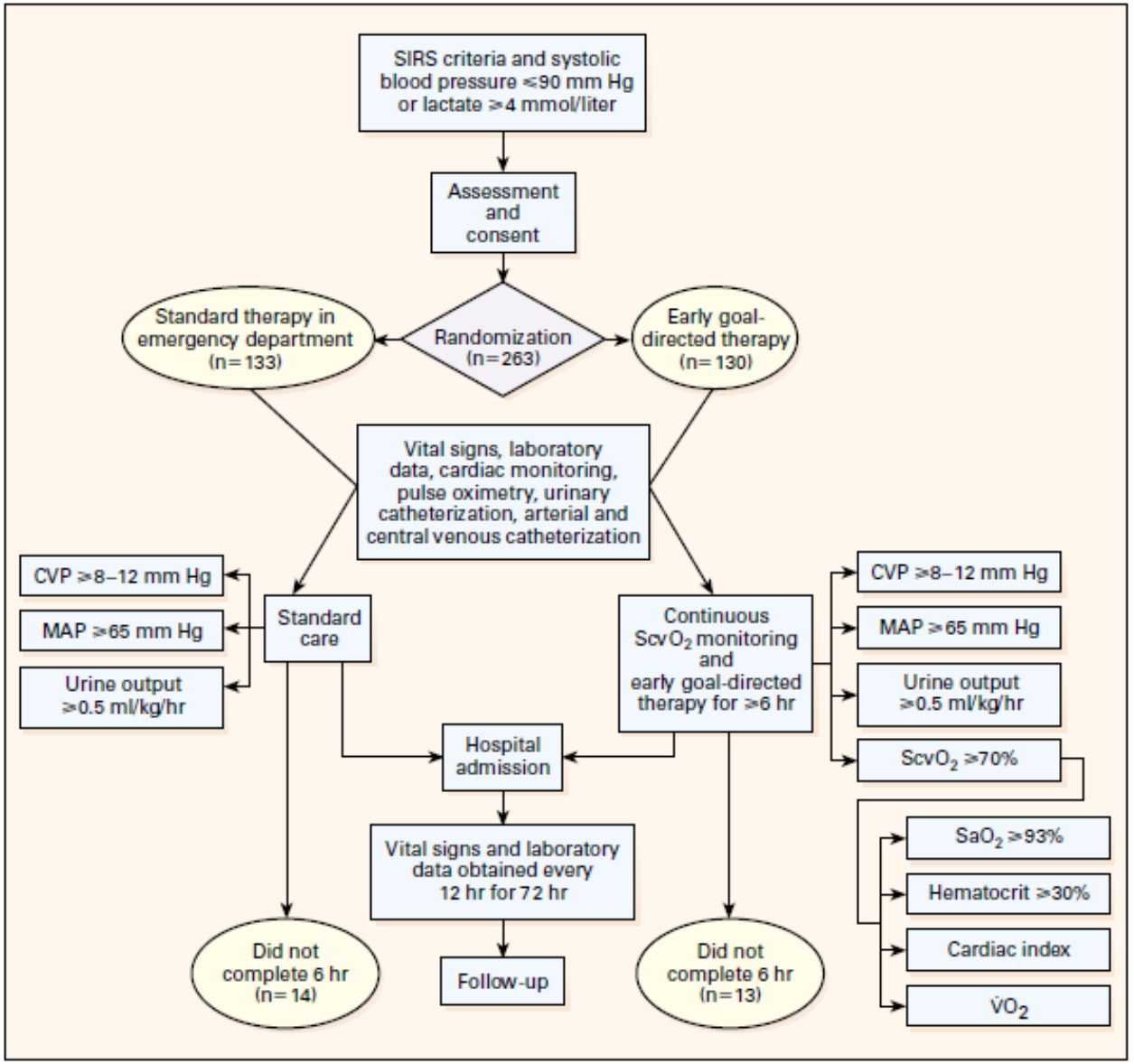
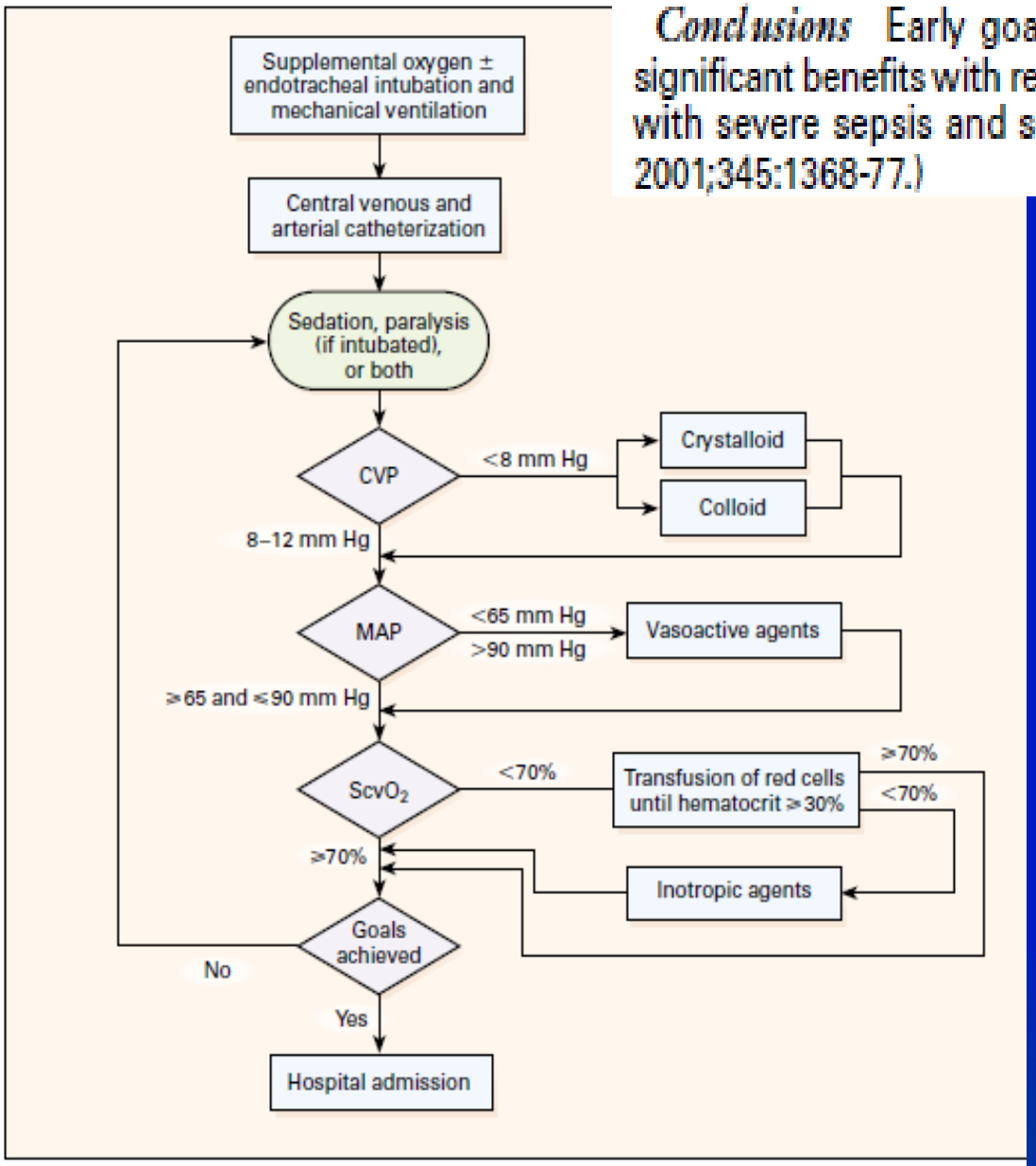


Figure 1. Overview of Patient Enrollment and Hemodynamic Support.

SIRS denotes systemic inflammatory response syndrome, CVP central venous pressure, MAP mean arterial pressure, $ScvO_2$ central venous oxygen saturation, SaO_2 arterial oxygen saturation, and $\dot{V}O_2$ systemic oxygen consumption. The criteria for a diagnosis of SIRS were temperature greater than or equal to 38°C or less than 36°C, heart rate greater than 90 beats per minute, respiratory rate greater than 20 breaths per minute or partial pressure of arterial carbon dioxide less than 32 mm Hg, and white-cell count greater than 12,000 per cubic millimeter or less than 4000 per cubic millimeter or the presence of more than 10 percent immature band forms.



Conclusions Early goal-directed therapy provides significant benefits with respect to outcome in patients with severe sepsis and septic shock. (N Engl J Med 2001;345:1368-77.)

Figure 2. Protocol for Early Goal-Directed Therapy.
CVP denotes central venous pressure, MAP mean arterial pressure, and $ScvO_2$ central venous oxygen saturation.

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MAY 1, 2014 VOL. 370 NO. 18

A Randomized Trial of Protocol-Based Care for Early Septic Shock

The ProCESS Investigators*

METHODS

In 31 emergency departments in the United States, we randomly assigned patients with septic shock to one of three groups for 6 hours of resuscitation: protocol-based EGDT; protocol-based standard therapy that did not require the placement of a central venous catheter, administration of inotropes, or blood transfusions; or usual care. The primary end point was 60-day in-hospital mortality. We tested sequentially whether protocol-based care (EGDT and standard-therapy groups combined) was superior to usual care and whether protocol-based EGDT was superior to protocol-based standard therapy. Secondary outcomes included longer-term mortality and the need for organ support.

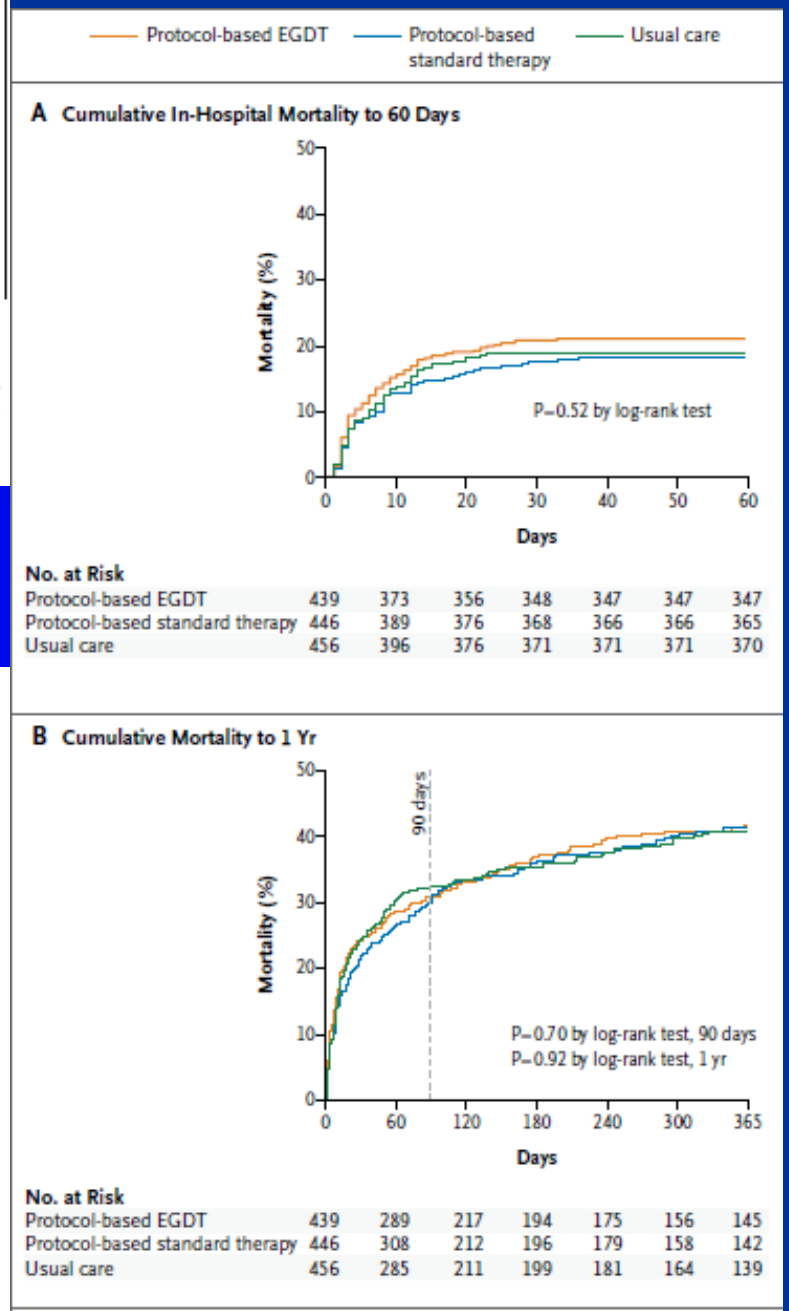


Figure 2. Cumulative Mortality.

Panel A shows cumulative in-hospital mortality, truncated at 60 days, and Panel B cumulative mortality up to 1 year after randomization.

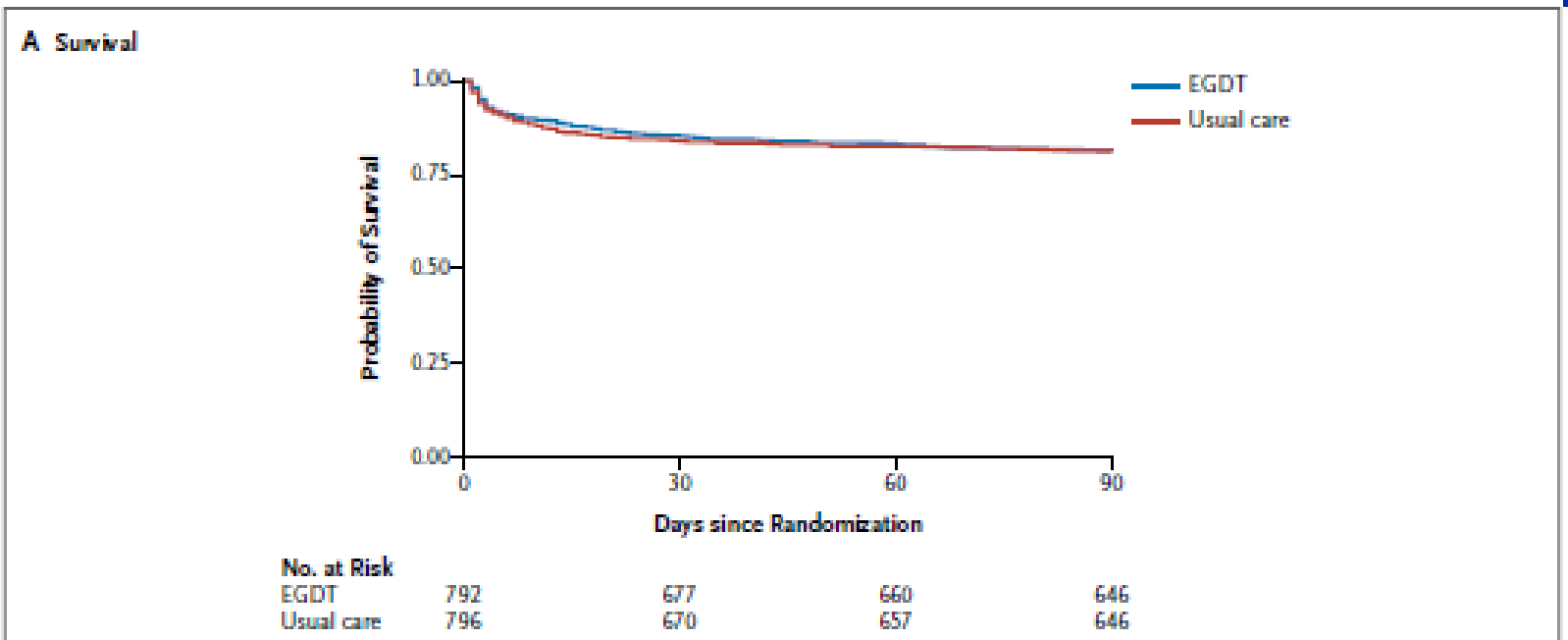
Goal-Directed Resuscitation for Patients with Early Septic Shock

The ARISE Investigators and the ANZICS Clinical Trials Group*

N Engl J Med 2014;371:1496-506.

METHODS

In this trial conducted at 51 centers (mostly in Australia or New Zealand), we randomly assigned patients presenting to the emergency department with early septic shock to receive either EGDT or usual care. The primary outcome was all-cause mortality within 90 days after randomization.



CONCLUSIONS

In critically ill patients presenting to the emergency department with early septic shock, EGDT did not reduce all-cause mortality at 90 days. (Funded by the National Health and Medical Research Council of Australia and the Alfred Foundation; ARISE ClinicalTrials.gov number, NCT00975793.)

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 24, 2014 VOL. 370 NO. 17

High versus Low Blood-Pressure Target in Patients with Septic Shock

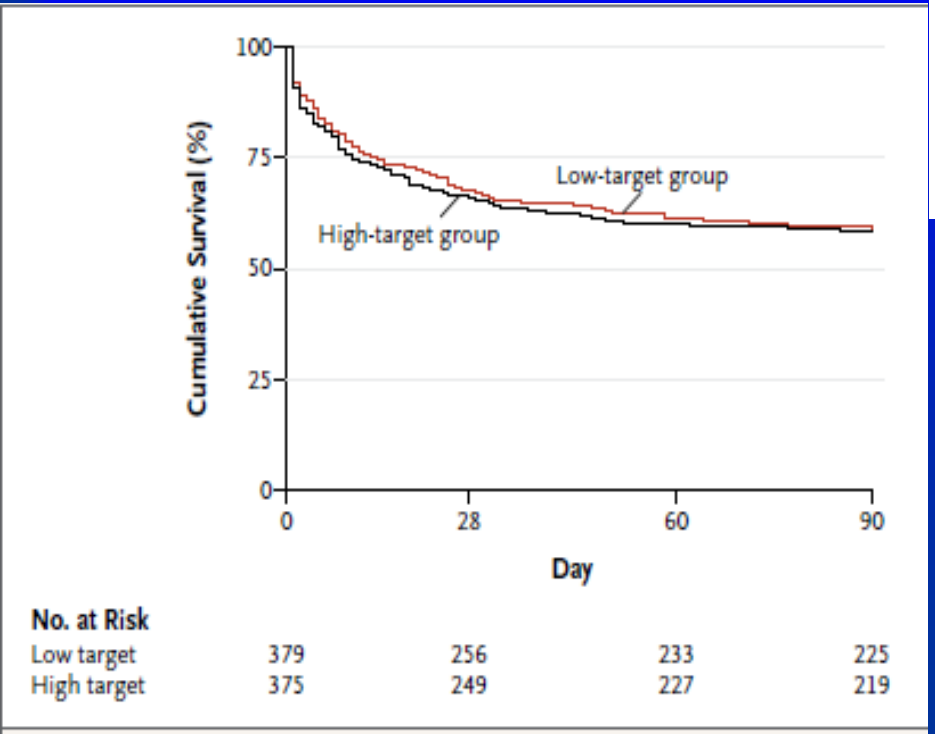


Figure 3. Kaplan–Meier Curves for Cumulative Survival.

CONCLUSIONS

Targeting a mean arterial pressure of 80 to 85 mm Hg, as compared with 65 to 70 mm Hg, in patients with septic shock undergoing resuscitation did not result in significant differences in mortality at either 28 or 90 days. (Funded by the French Ministry of Health; SEPSISPAM ClinicalTrials.gov number, NCT01149278.)

The NEW ENGLAND JOURNAL of MEDICINE

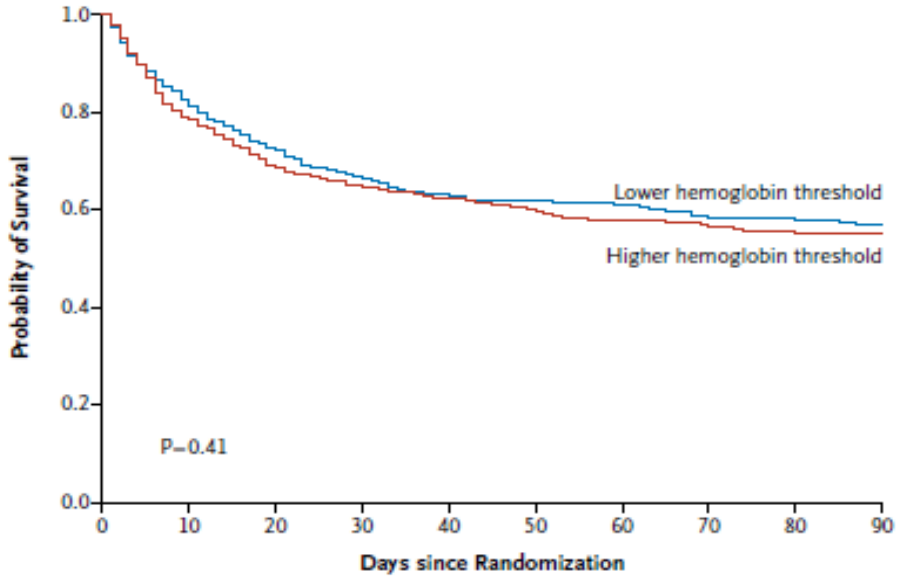
ESTABLISHED IN 1812 OCTOBER 9, 2014 VOL. 371 NO. 15

Lower versus Higher Hemoglobin Threshold for Transfusion in Septic Shock

METHODS

In this multicenter, parallel-group trial, we randomly assigned patients in the intensive care unit (ICU) who had septic shock and a hemoglobin concentration of 9 g per deciliter or less to receive 1 unit of leukoreduced red cells when the hemoglobin level was 7 g per deciliter or less (lower threshold) or when the level was 9 g per deciliter or less (higher threshold) during the ICU stay. The primary outcome measure was death by 90 days after randomization.

A Time to Death



No. at Risk				
Lower hemoglobin threshold	502	334	306	286
Higher hemoglobin threshold	496	321	287	273

B Relative Risk of the Primary Outcome

Subgroup	Lower Hemoglobin Threshold	Higher Hemoglobin Threshold	Relative Risk (95% CI)	P Value for Heterogeneity
	no. of events/no. of patients in subgroup			
Age				0.85
>70 yr	93/173	98/185	0.98 (0.79–1.18)	
≤70 yr	123/329	125/311	0.94 (0.75–1.14)	
Chronic cardiovascular disease				0.25
Yes	42/75	33/66	1.08 (0.75–1.40)	
No	174/427	190/430	0.90 (0.75–1.06)	
SAPS II at baseline				0.06
>53	112/207	139/226	0.83 (0.64–1.04)	
≤53	104/295	84/270	1.10 (0.91–1.30)	
All patients	216/502	223/496	0.94 (0.78–1.09)	

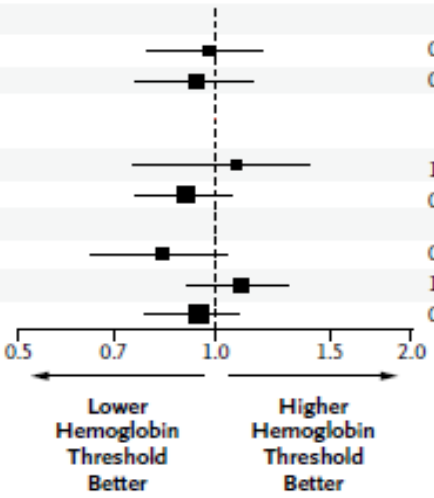
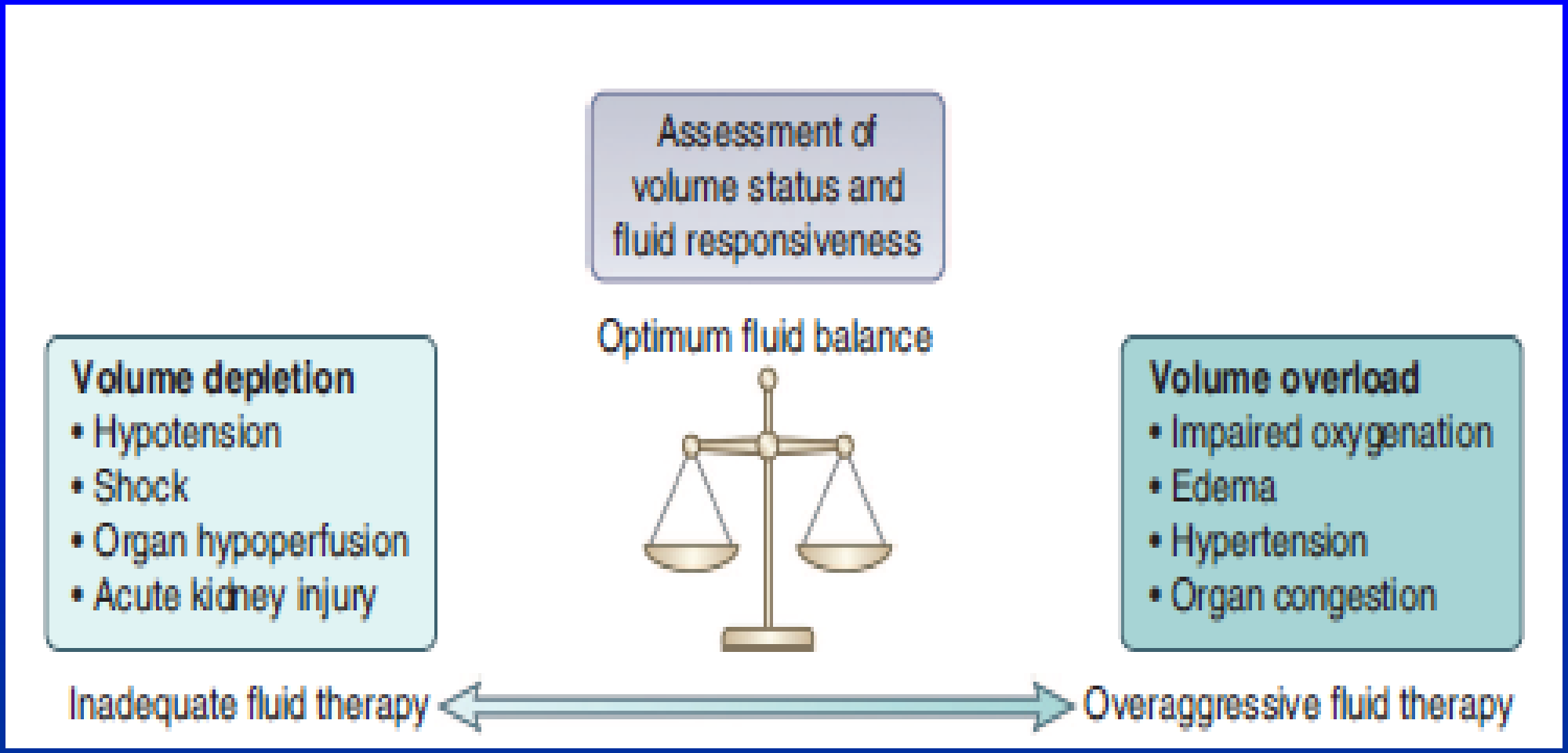


Figure 3. Time to Death and Relative Risk of Death at 90 Days.

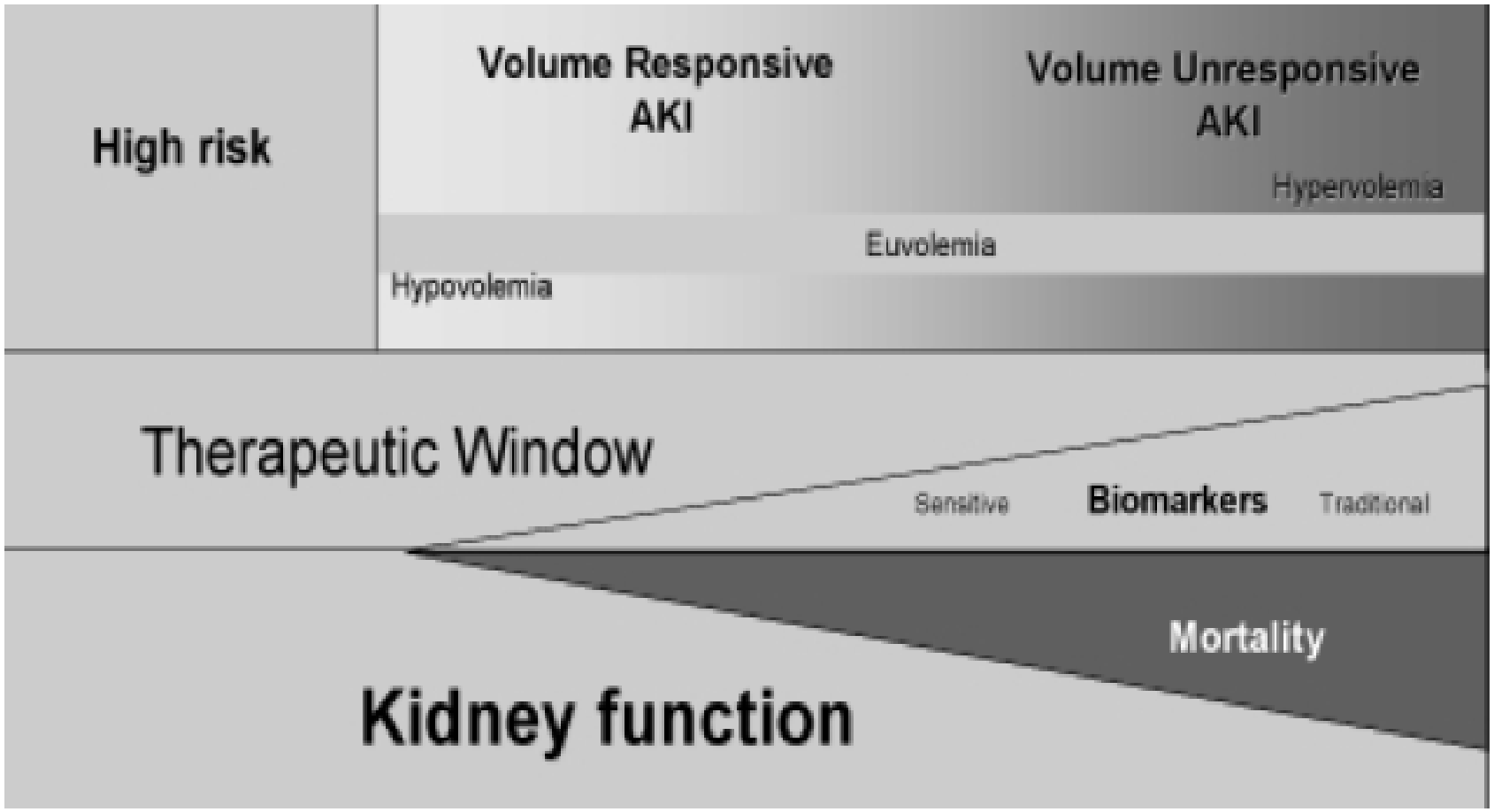
Assessment of intravascular volume status and volume responsiveness in critically ill patients

Kambiz Kalantari[1], Jamison N. Chang[1], Claudio Ronco[2] and Mitchell H. Rosner[1]



Kidney International advance online publication, 9 January 2013; doi:10.1038/ki.2012.424

KDIGO CLINICAL PRACTICE GUIDELINE FOR ACUTE KIDNEY INJURY



http://www.kidney-international.org

Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury

Josée Bouchard[1], Sharon B. Soroko[1], Glenn M. Chertow[2], Jonathan Himmelfarb[3], T. Alp Ikizler[4], Emil P. Paganini[5] and Ravindra L. Mehta[1], Program to Improve Care in Acute Renal Disease (PICARD) Study Group

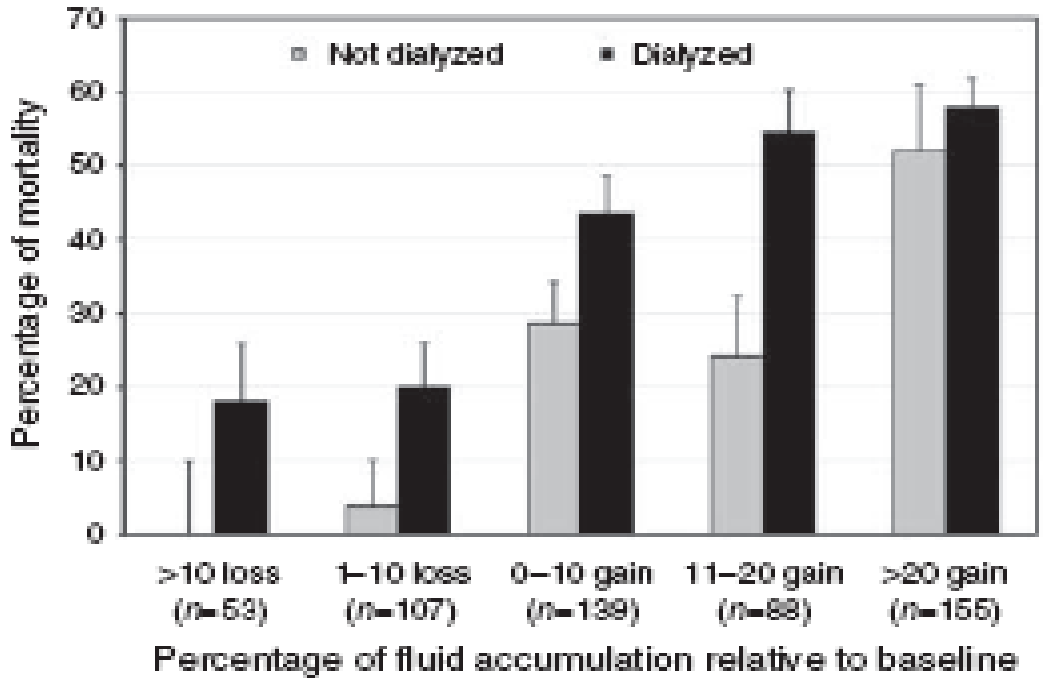


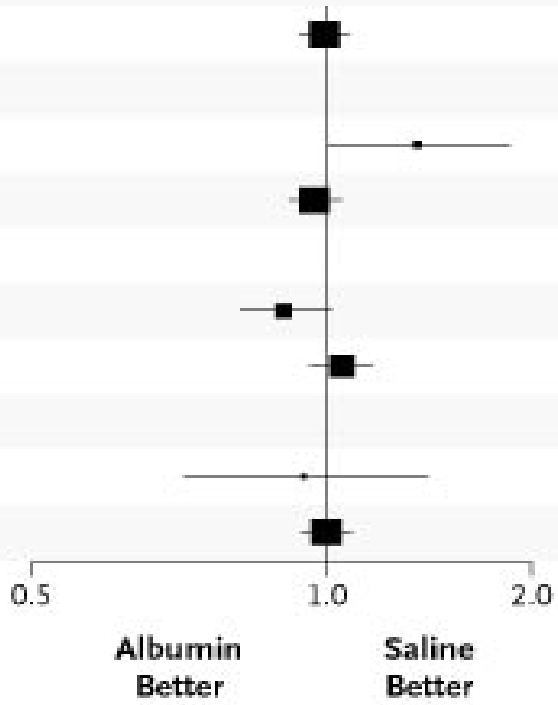
Figure 2 | Mortality rate by final fluid accumulation relative to baseline weight and stratified by dialysis status.

FLUIDS

3.1.1: In the absence of hemorrhagic shock, we suggest using isotonic crystalloids rather than colloids (albumin or starches) as initial management for expansion of intravascular volume in patients at risk for AKI or with AKI. (*2B*)

Relative Risk of Death from Any Cause among All the Patients and among the Patients in the Six Predefined Subgroups

Patients	Albumin Group	Saline Group	Relative Risk (95% CI)
	no. of deaths/total no.		
Overall	726/3473	729/3460	0.99 (0.91–1.09)
Trauma			
Yes	81/596	59/590	1.36 (0.99–1.86)
No	641/2831	666/2830	0.96 (0.88–1.06)
Severe sepsis			
Yes	185/603	217/615	0.87 (0.74–1.02)
No	518/2734	492/2720	1.05 (0.94–1.17)
ARDS			
Yes	24/61	28/66	0.93 (0.61–1.41)
No	697/3365	697/3354	1.00 (0.91–1.09)



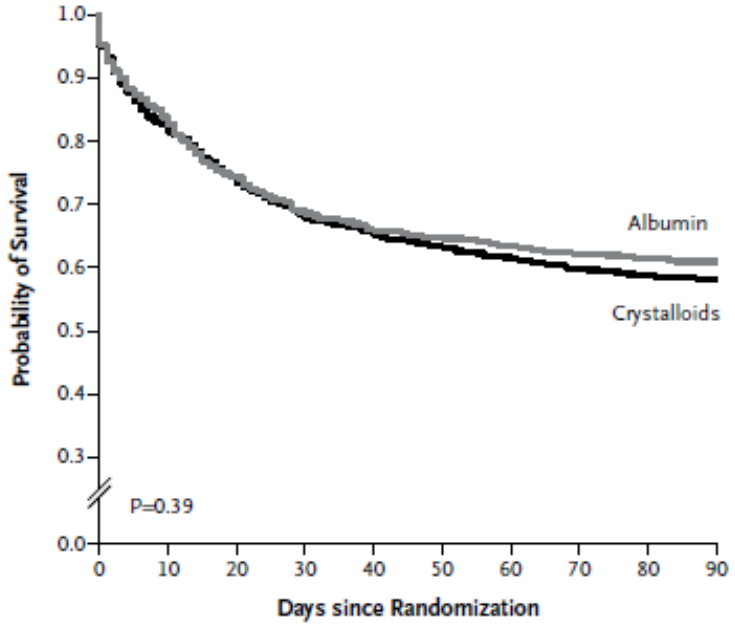
The SAFE Study Investigators. N Engl J Med 2004;350:2247-2256

Albumin Replacement in Patients with Severe Sepsis or Septic Shock

This article was published on March 18, 2014, at NEJM.org.

METHODS

In this multicenter, open-label trial, we randomly assigned 1818 patients with severe sepsis, in 100 intensive care units (ICUs), to receive either 20% albumin and crystalloid solution or crystalloid solution alone. In the albumin group, the target serum albumin concentration was 30 g per liter or more until discharge from the ICU or 28 days after randomization. The primary outcome was death from any cause at 28 days. Secondary outcomes were death from any cause at 90 days, the number of patients with organ dysfunction and the degree of dysfunction, and length of stay in the ICU and the hospital.



No. at Risk										
Albumin	903	733	647	597	567	556	545	535	529	523
Crystalloids	907	729	652	598	676	551	538	521	511	504

Figure 2. Probability of Survival from Randomization through Day 90.
The graph shows the Kaplan–Meier estimates for the probability of survival among patients receiving albumin and crystalloids and among those receiving crystalloids alone. The P value was calculated with the use of the log-rank test.

CONCLUSIONS

In patients with severe sepsis, albumin replacement in addition to crystalloids, as compared with crystalloids alone, did not improve the rate of survival at 28 and 90 days. (Funded by the Italian Medicines Agency; ALBIOS ClinicalTrials.gov number, NCT00707122.)

Hydroxyethyl Starch 130/0.4 versus Ringer's Acetate in Severe Sepsis

This article was published on June 27, 2012, at NEJM.org.

CONCLUSIONS

Patients with severe sepsis assigned to fluid resuscitation with HES 130/0.4 had an increased risk of death at day 90 and were more likely to require renal-replacement therapy, as compared with those receiving Ringer's acetate. (Funded by the Danish Research Council and others; 6S ClinicalTrials.gov number, NCT00962156.)

6S (Scandinavian Starch Severe Sepsis/ Septic Shock) trial 2012

B Relative Risk of the Primary Outcome

Subgroup	HES 130/0.4	Ringer's Acetate	Relative Risk (95% CI)	P Value for Heterogeneity
	no. of events/no. in subgroup			
Shock at the time of randomization				0.22
Yes	179/336	148/337	1.21 (1.04–1.42)	
No	23/62	25/63	0.93 (0.60–1.46)	
Acute kidney injury at the time of randomization				0.60
Yes	72/142	63/140	1.13 (0.88–1.44)	
No	130/256	110/260	1.20 (1.00–1.45)	
All patients	202/398	173/400	1.17 (1.01–1.36)	

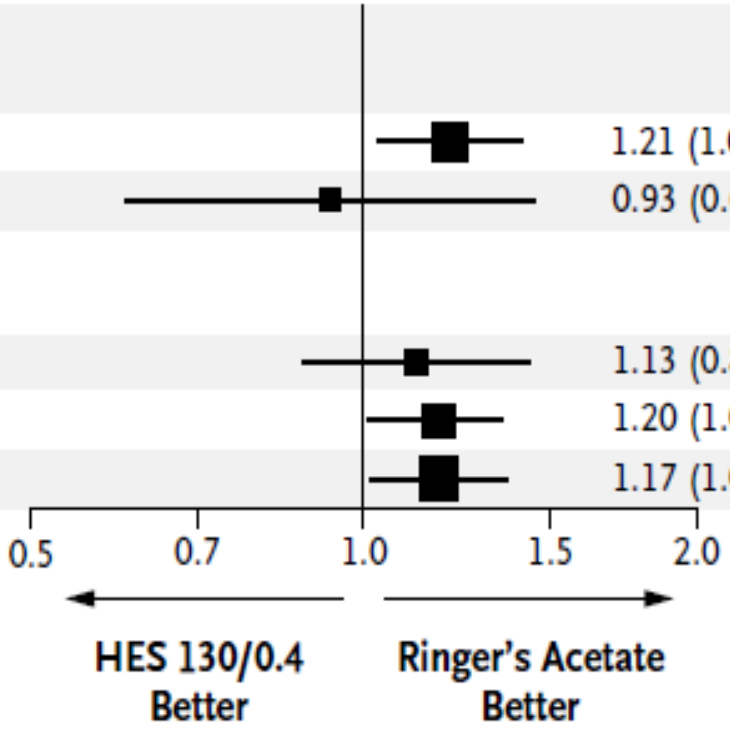
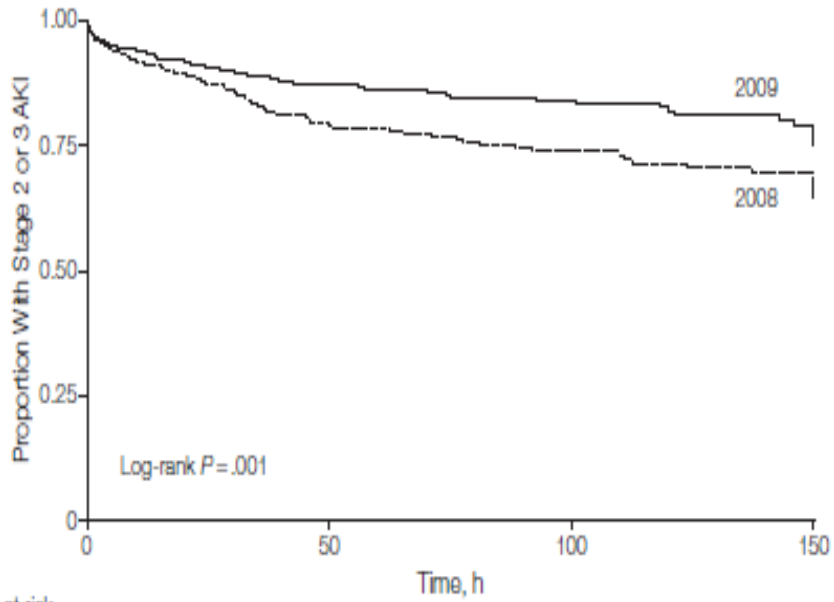


Figure 2. Time to Death and Relative Risk of the Primary Outcome.

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

JAMA, October 17, 2012—Vol 308, No. 15

Figure 1. Development of Stage 2 or 3 Acute Kidney Injury (AKI) While in the Intensive Care Unit (ICU)



Stage 2 or 3 defined according to the Kidney Disease: Improving Global Outcomes clinical practice guideline.

Figure 2. Renal Replacement Therapy (RRT) in the Intensive Care Unit (ICU)

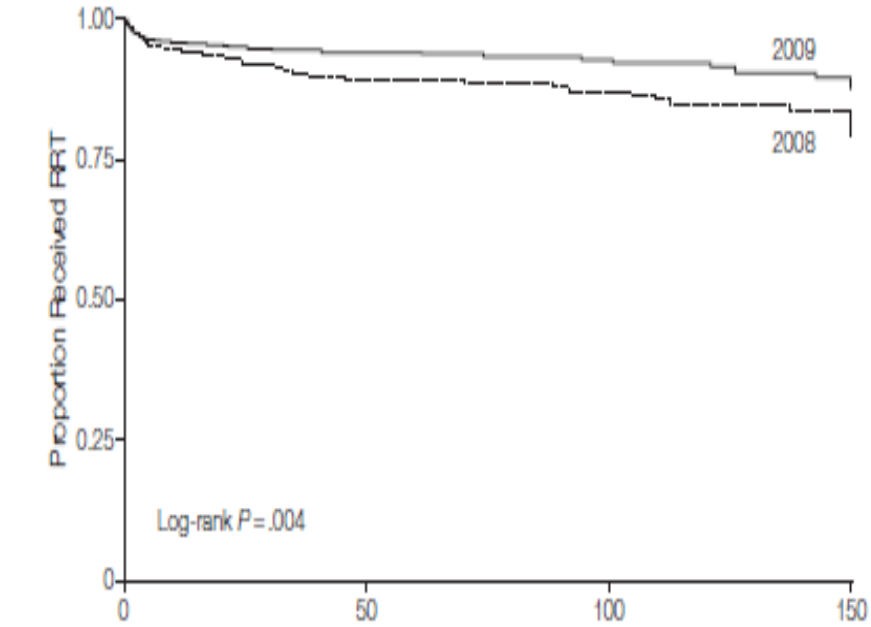


Table 1. Crystalloid Solutions

	Na^+ (mEq/L)	K^+ (mEq/L)	Ca^{++} (mEq/L)	Mg^{++} (mEq/L)	Cl^- (mEq/L)	Buffers (mEq/L electrolyte)	Glucose (g/L)	pH	POsm (mOsm/L)
Plasma	141	4.5	5	2	103	Bicarbonate, 26; protein, 16	0.7-1.1	7.4	290
Isotonic									
Normal saline	154	—	—	—	154	—	—	6.0	308
Lactated Ringer's solution	130	4	4	—	109	Lactate, 28	—	6.5	274
Plasma-Lyte	140	5	—	3	98	Acetate, 27; Glucose, 23	—	7.4	294

KDIGO CLINICAL PRACTICE GUIDELINE FOR ACUTE KIDNEY INJURY

CHAPTER 3.3: GLYCEMIC CONTROL AND NUTRITIONAL SUPPORT

3.3.A: GLYCEMIC CONTROL IN CRITICAL ILLNESS: RENAL EFFECTS AND OUTCOMES

3.3.1: In critically ill ICU patients, we suggest insulin therapy (target plasma glucose <150 mg/dl [8.33 mmol/L] rather than 180-200 mg/dl [9.99-11.1 mmol/L]), using a protocol to prevent hypoglycemia. (*2B*) To prevent hypoglycemia, the frequently monitored blood glucose levels should not be lower than 110 mg/dl (6.1 mmol/L).

Preventable Risk Factors for Acute Kidney Injury in Patients Undergoing Cardiac Surgery

Pradeep Arora, MD,*† Hari Kolli, MBBS,† Neha Nainani, MD,† Nader Nader, MD,‡ and James Lohr, MD*†

Journal of Cardiothoracic and Vascular Anesthesia, Vol 26, No 4 (August), 2012: pp 687-697

Preoperatively:

1. Evaluate preoperative renal function. If the serum creatinine is above baseline, look for a reversible cause and allow creatinine to return to baseline before elective surgery.
2. If the patient has an angiogram, allow 5 days if possible before surgery.
3. Discontinue NSAIDs (except for aspirin) 3 days before surgery. Aspirin should be continued.
4. Discontinue ACEIs/ARBs 3 days before surgery. Restart in postoperative period.
5. Optimize glucose control before surgery in diabetics. Maintain glucose <180 without causing hypoglycemia in the perioperative period.
6. Statin therapy may decrease the incidence of AKI after cardiac surgery (weak evidence) but has been shown to improve other outcomes; thus, the authors would recommend this be given pre- and postoperatively.
7. In patients with baseline CKD, consider off-pump versus on-pump surgery.

Intraoperatively:

1. Avoid hematocrit of <21% during CPB.
2. Administer $NaHCO_3$ for a 24-hour period to reduce the incidence of AKI.

Postoperatively:

1. Use antibiotics judicially.
2. Avoid aminoglycosides if possible.

Prophylactic Perioperative Sodium Bicarbonate to Prevent Acute Kidney Injury Following Open Heart Surgery: A Multicenter Double-Blinded Randomized Controlled Trial

Michael Haase[1,9], Anja Haase-Fielitz[1*,9], Michael Plass[2], Hermann Kuppe[2], Roland Hetzer[3], Claire Hannon[4], Patrick T. Murray[4], Michael J Bailey[5], Rinaldo Bellomo[6*], Sean M. Bagshaw[7]

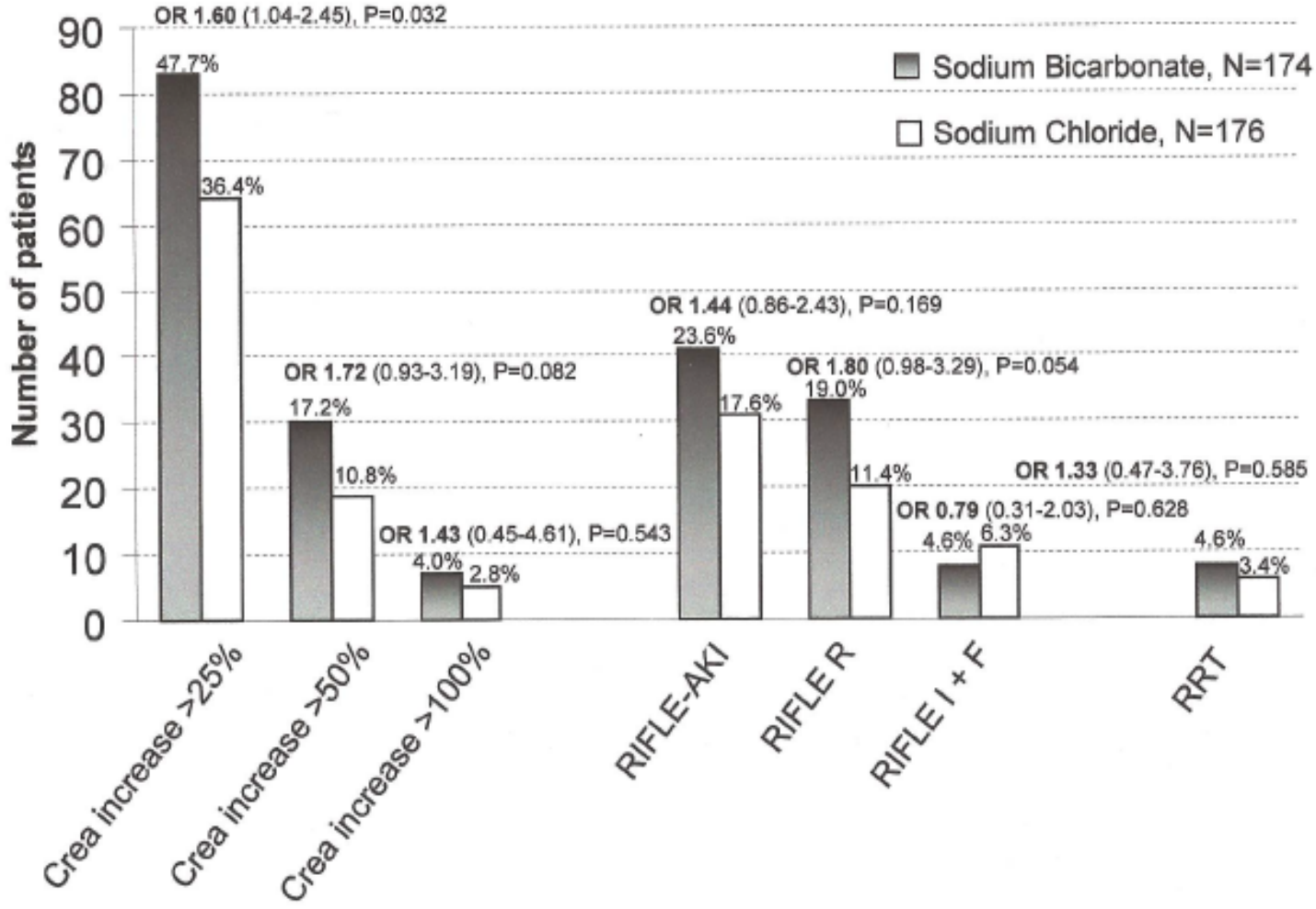
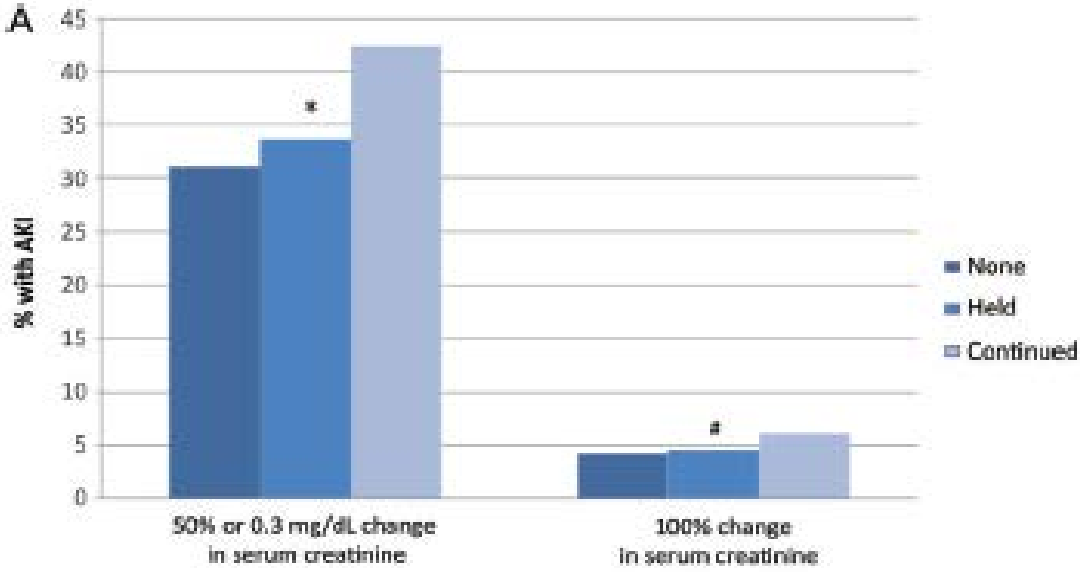


Figure 2. Renal endpoints for patients receiving sodium bicarbonate versus sodium chloride. Number of patients receiving sodium bicarbonate (black bars) developing acute kidney injury after open heart surgery compared to patients receiving sodium chloride (white bars). The

Preoperative angiotensin-converting enzyme inhibitors and angiotensin receptor blocker use and acute kidney injury in patients undergoing cardiac surgery

Nephrol Dial Transplant (2013) 28: 2787–2799
doi: 10.1093/ndt/gft405
Advance Access publication 29 September 2013

Steven G. Coca[1,2],
Amit X. Garg[3],
Madhav Swaminathan[4],
Susan Garwood[5],
Kwangik Hong[1,2],
Heather Thiessen-Philbrook[3],
Cary Passik[6,7],
Jay L. Koyner[8]
and Chirag R. Parikh[1,2]
On behalf of the TRIBE-AKI Consortium



Nephrol Dial Transplant (2013) 28: 2787–2799
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Advance Access publication 29 September 2013

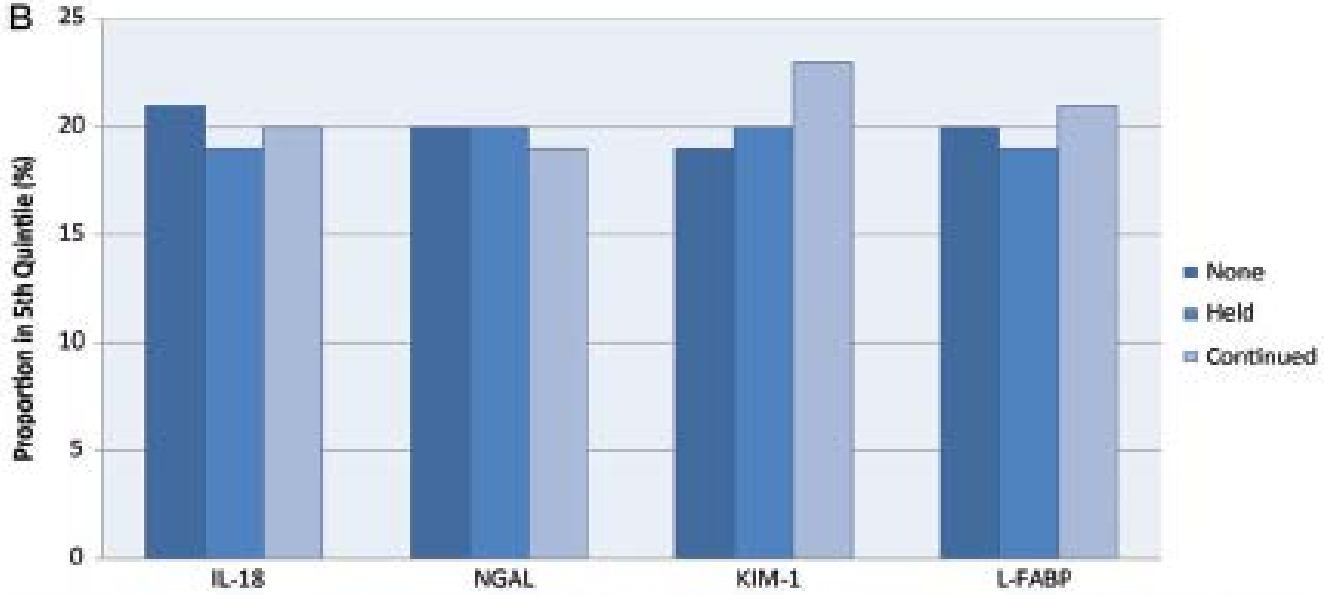


FIGURE 2: Incidence of AKI by ACEI/ARB status. (A) Serum creatinine-based definitions of AKI. *P for trend = 0.005; # P for trend = 0.41. (B) Biomarker-based definitions of AKI (5th Quintile of Peak Postoperative Concentration). P for trend not significant for all biomarkers. IL-18, interleukin-18; NGAL, neutrophil gelatinase-associated lipocalin, KIM-1, kidney injury molecule-1; L-FABP, liver-fatty acid binding protein.

Preoperative angiotensin-converting enzyme inhibitors and angiotensin receptor blocker use and acute kidney injury in patients undergoing cardiac surgery

Nephrol Dial Transplant (2013) 28: 2787–2799
doi: 10.1093/ndt/gft405
Advance Access publication 29 September 2013

In conclusion, continued preoperative ACEi/ARB administration resulted in more functional AKI, as evidenced by greater increases in postoperative serum creatinine; however, there was no evidence that ACEi/ARB exposure on the morning of cardiac surgery resulted in more structural kidney injury, as assessed by multiple biomarkers of tubular damage over the first 5 postoperative days. Moreover, there was no impact observed on nonrenal outcomes. Thus, the clinical significance of AKI in patients on ACEi/ARBs undergoing cardiac surgery is unclear, and future studies should assess optimal strategies for administration of these medications in the perioperative setting.

Prévention de l'IRA quand produit de contraste

- Limiter cette injection si risque >
- Bien hydrater (NaCl 0,9% 1 ml/kg/h 12 h avant et 12h après)
- Ajouter lysomucil 2X 1200 mg/j (?)
- Ajouter Statine à forte dose en aigu (avant et après injection) (?)

A randomized comparison of 1-h sodium bicarbonate hydration versus standard peri-procedural saline hydration in patients with chronic kidney disease undergoing intravenous contrast-enhanced computerized tomography

Nephrol Dial Transplant (2014) 29: 1029–1036
doi: 10.1093/ndt/gfu025
Advance Access publication 27 February 2014

Table 1. Patient and procedure characteristics

	Sodium bicarbonate ($n = 267$)	Saline ($n = 281$)
Mean age, years	71.6 (9.8)	72.5 (9.5)
Sex, male	160 (59.9)	171 (60.9)
Outpatients	252 (94.4)	253 (90.0)
Mean eGFR	49.9 (13.4)	50.9 (13.9)
eGFR > 45 mL/min/1.73 m^2	186 (69.7)	191 (68.0)
eGFR 30–45 mL/min/1.73 m^2	59 (22.1)	71 (25.3)
eGFR 15–30 mL/min/1.73 m^2	19 (7.1)	18 (6.4)
eGFR < 15 mL/min/1.73 m^2	3 (1.1)	1 (0.4)

Table 2. Incidence of CI-AKI according to the AKI criteria

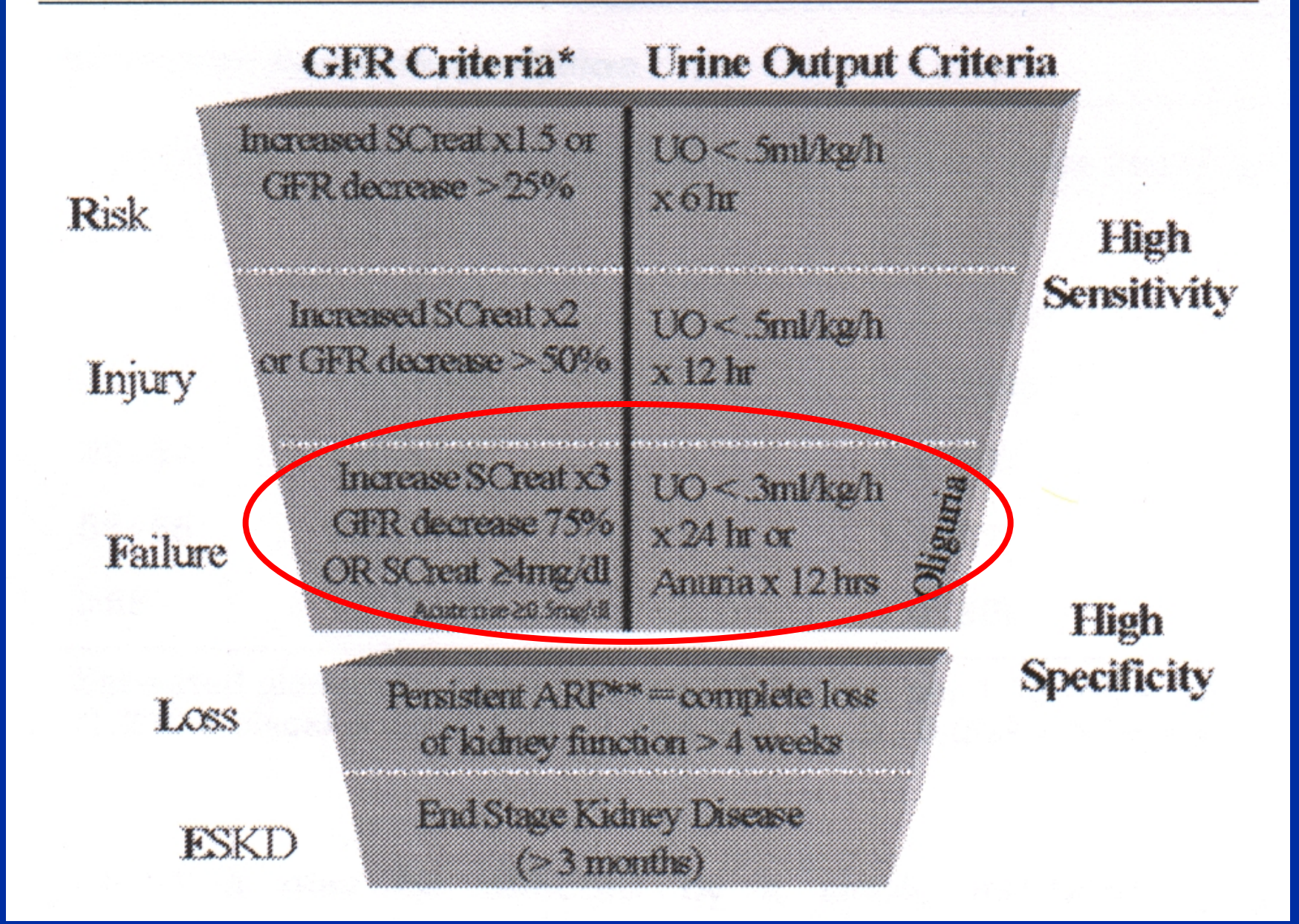
AKIN stage	Sodium bicarbonate (95% CI)		Saline (95% CI)		RR (95% CI)	
I (increase > 26.5 μmol/L or 150–200% from baseline)	11/263, 4.2%	(2.3–7.4)	17/273, 6.2%	(3.9–9.8)	0.7	(0.3–1.4)
II (increase 200–300% from baseline)	0/263	(0.0–1.7)	0/273	(0.0–1.7)	1.0	(0.0–52.1)
III (increase > 300% from baseline, or ≥354 μmol/L, or on RRT)	0/263	(0.0–1.7)	0/273	(0.0–1.7)	1.0	(0.0–52.1)

AKIN, acute kidney injury network.

Intra Abdominal Pressure (IAP)

- *"Normal IAP is approximately 5-7 mmHg in critically ill adults."*

- An IAP in excess of 15 mmHg can cause significant end-organ dysfunction, failure, and patient death. **Oliguria is one of the first visible signs of elevated IAP**



DEFINITION OF AKI

Bellomo R. et al., *Critical Care*, 2004, **8**: R204-R212.

Defining kidney FAILURE

- ARFailure is often defined by the initiation time of RRT, but the decision to start this therapy is subjective
- **Treatment of ARF is largely supportive:** avoid volume overload, correct acid-base and electrolytes disturbances, adaptat drug dosing, start RRT in due time.

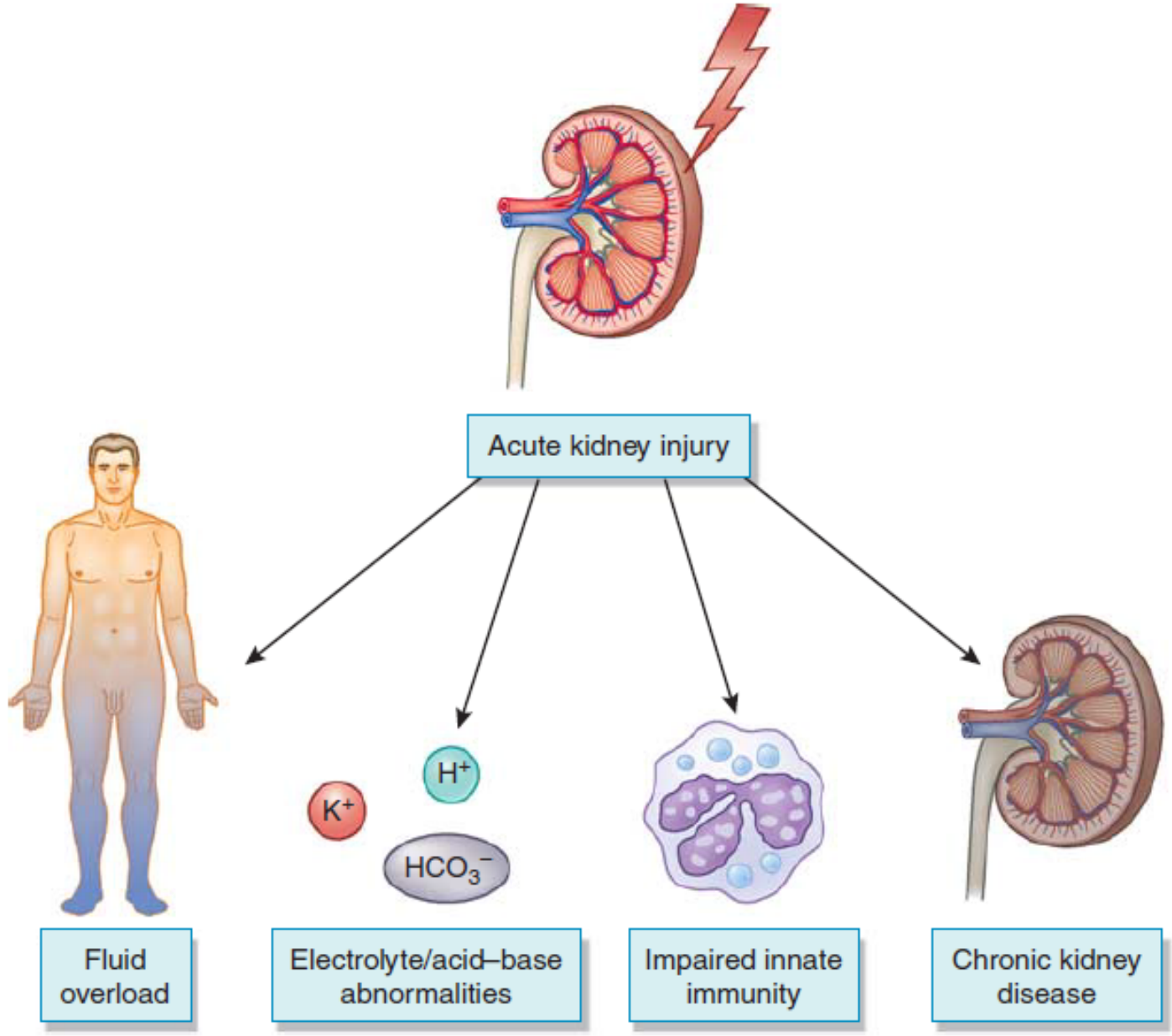


Figure 2 | Acute kidney injury (AKI) can have both immediately recognizable consequences as well as less noticeable or delayed consequences. Fluid overload and electrolyte/acid–base abnormalities represent well known, easily recognized consequences of AKI. Contrary, impaired innate immunity and chronic kidney disease do not manifest themselves immediately.

EVOLUTION ET PRONOSTIC

Evolution défavorable : complications

	Incidence (%)	Cause de décès (%)
1. Infectieuses	70	30-70
2. Cardiovasculaires	35	5-30
3. Digestives	50	5-20
4. Pulmonaires	40	5-10
5. Hématologiques	-	6
6. Neurologiques	-	2
7. Hyperkaliémie	-	2

JNEPHROL 2008; 21: 645-656
www.sin-italy.org/jnonline – www.jnephrol.com

Nutritional support in acute kidney injury

Enrico Fiaccadori, Elisabetta Parenti, Umberto Maggiore Department of Clinical Medicine, Nephrology and Prevention Science, University of Parma, Parma - Italy

Preferentially use enteral route

TABLE II
NUTRITIONAL REQUIREMENTS IN PATIENTS WITH ACUTE KIDNEY INJURY

Energy	
Nonprotein calories	25 kcal/kg BW per day*
Carbohydrates	5 g/kg BW per day
Fat	0.8-1.2 g/kg BW per day
Protein (essential and nonessential amino acids)	
Conservative therapy, mild catabolism	0.8 g/kg BW per day
Extracorporeal therapy, moderate catabolism	1.0-1.5 g/kg BW per day
CRRT or SLED, severe hypercatabolism	1.5-2.0 g/kg BW per day

BW = body weight; CRRT = continuous renal replacement therapy; SLED = sustained low-efficiency dialysis.
*Adapted to catabolism levels and to individual needs in case of underweight or obesity.

Et dans un avenir proche?

- Cellules stromales mésenchymateuses
- Levosimendan: vasodilatateur préservant la fonction mitochondriale
- Dexmedetomidine: alpha 2 agoniste utile en chir cardiaque
- Phosphatases alcalines: utilité dans le sepsis
- Statines: effets pléiotropes

Acute Kidney Injury

- Importance of the problem

- Consequences for the patient and higher cost

- Danger of infection

- Need for early diagnosis (new biomarkers + RIFLE)
- **Prevention first**: limitation of contrast agents use, caution about medical therapy, IAP monitoring, control of fluid volume and of glycemia
- Treatment: avoid complications when AKI installed, nutrition !
- Early RRT (?)(rapid uremic control or fluid balance?)
- Follow the patient after (caution with eGFR data!)