Part 2

Update on renal replacement therapy in AKI and post AKI concerns

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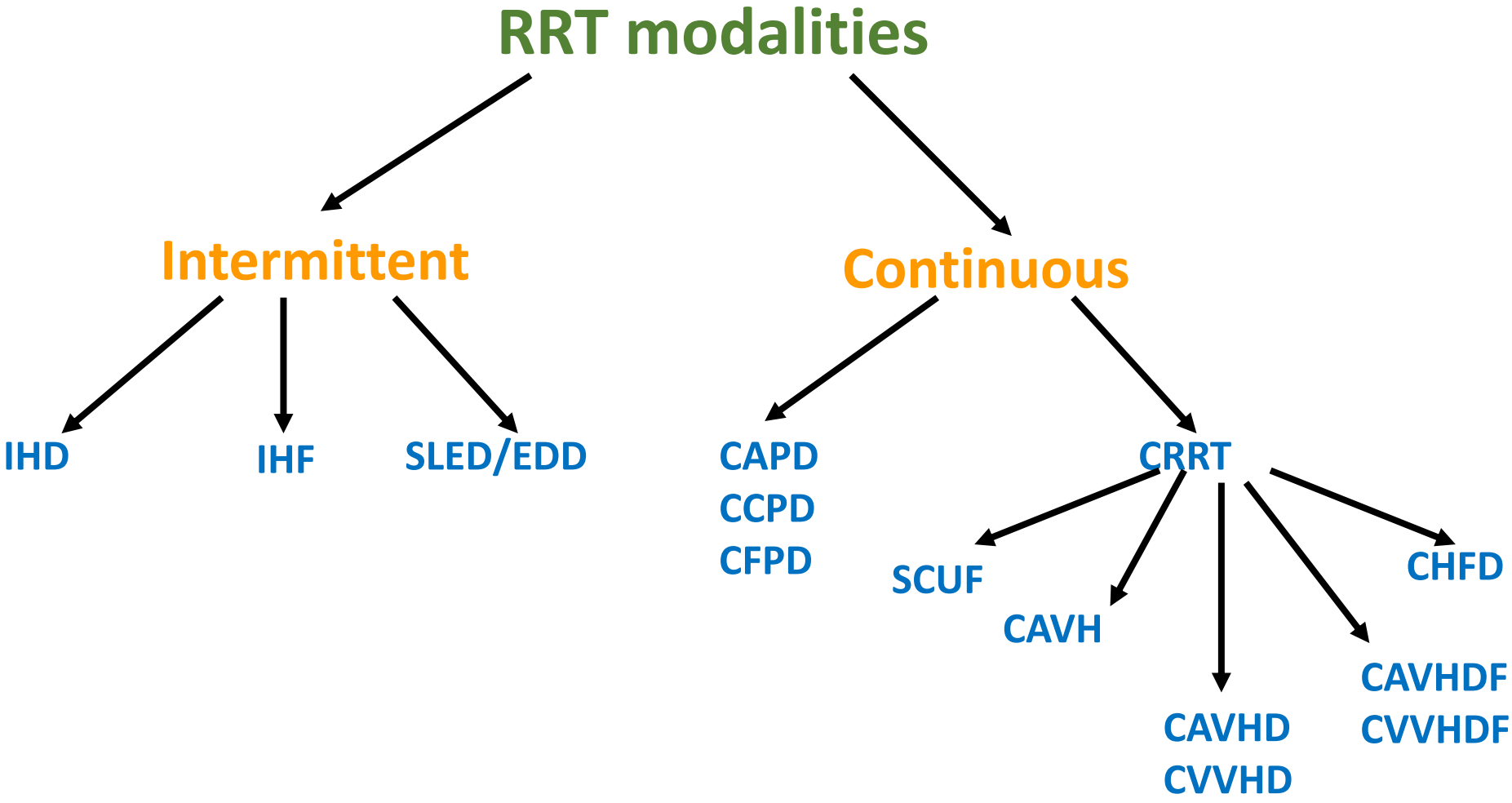
Classification of renal replacement therapies in AKI



Characteristics of different modalities of extracorporeal RRT

	IHD	PIRRT	CRRT
Mechanism of Clearance	Diffusion	Diffusion, convection, or both	Diffusion, convection, or both
Duration (h)	3–4	6–12	Continuous
Frequency (d/wk)	3-7	3–7	Continuous
Timing of Procedure	Usually daytime	Daytime or nighttime	Continuous
Anticoagulation	Not necessary	Not necessary	Necessary
Vascular Access	AVF, AVG, CVC	CVC	CVC
Patient Location	Ward, ICU, step-down	ICU, step-down	ICU

PIRRT: prolonged intermittent RRT

Heung, Yessayan, Crit Care Clin 33 (2017) 365–378

CHAPTER 5.4: VASCULAR ACCESS FOR RRT IN AKI

- **5.4.1: We suggest initiating RRT in patients with AKI via an uncuffed non-tunneled dialysis catheter, rather than a tunneled catheter. (*2D*)**
- **5.4.2: When choosing a vein for insertion of a dialysis catheter in patients with AKI, consider these preferences (*Not Graded*):**

  First choice: right jugular vein;

  Second choice: femoral vein;

  Third choice: left jugular vein;

  Last choice: subclavian vein with preference for dominant side.

CHAPTER 5.1: TIMING OF DIALYSIS

- **5.1.1: We recommend initiating dialysis emergently when life threatening changes in fluid, electrolyte and acid-base balance exist. (*1A; applicable to adults & children*)**
- **5.1.2: When determining when to start dialysis, base therapeutic decisions on the broader clinical context and trends of laboratory tests rather than on single BUN and creatinine thresholds alone. (*Not graded*)**

Generally recognized absolute indications for initiation of RRT

- Oliguria or anuria for more than 72 hours

- Serum urea concentration >40 mmol/l,
- Serum potassium concentration >6 mmol/l,
- Serum potassium concentration >5.5 mmol/l persisting despite medical treatment (bicarbonate or glucose-insulin infusion),
- pH <7.15 in a context of pure metabolic acidosis ($PaCO_2 < 35$ mmHg) or in a context of mixed acidosis with $PaCO_2 \geq 50$ mmHg without possibility of increasing alveolar ventilation,
- Acute pulmonary edema due to fluid overload leading to severe hypoxemia requiring oxygen flow rate >5 l/min to maintain $SpO_2 > 95\%$ or $FiO_2 > 50\%$ in patients already undergoing invasive or noninvasive mechanical ventilation and despite diuretic therapy.

Differences in design and outcomes between the ELAIN and AKIKI trials

Characteristic	ELAIN trial[7]	AKIKI trial[6]
Country	Germany	France
Number of Sites	1	31
Number of participants	231	620
ARR for sample size calculation	18%	15%
Criteria for early intervention strategy	KDIGO stage 2	KDIGO stage 3
Criteria for delayed intervention strategy	KDIGO stage 3	Specific criteria*
Mean SOFA score of enrolled patients	~16.0	~10.9
Primary end point	90 day mortality	60 day mortality
End point mortality – early intervention strategy	39.3%	48.5%
End point mortality – delayed intervention strategy	54.7%	49.7%
Proportion of patients in the delayed intervention group who received RRT	90.8%	51.0%

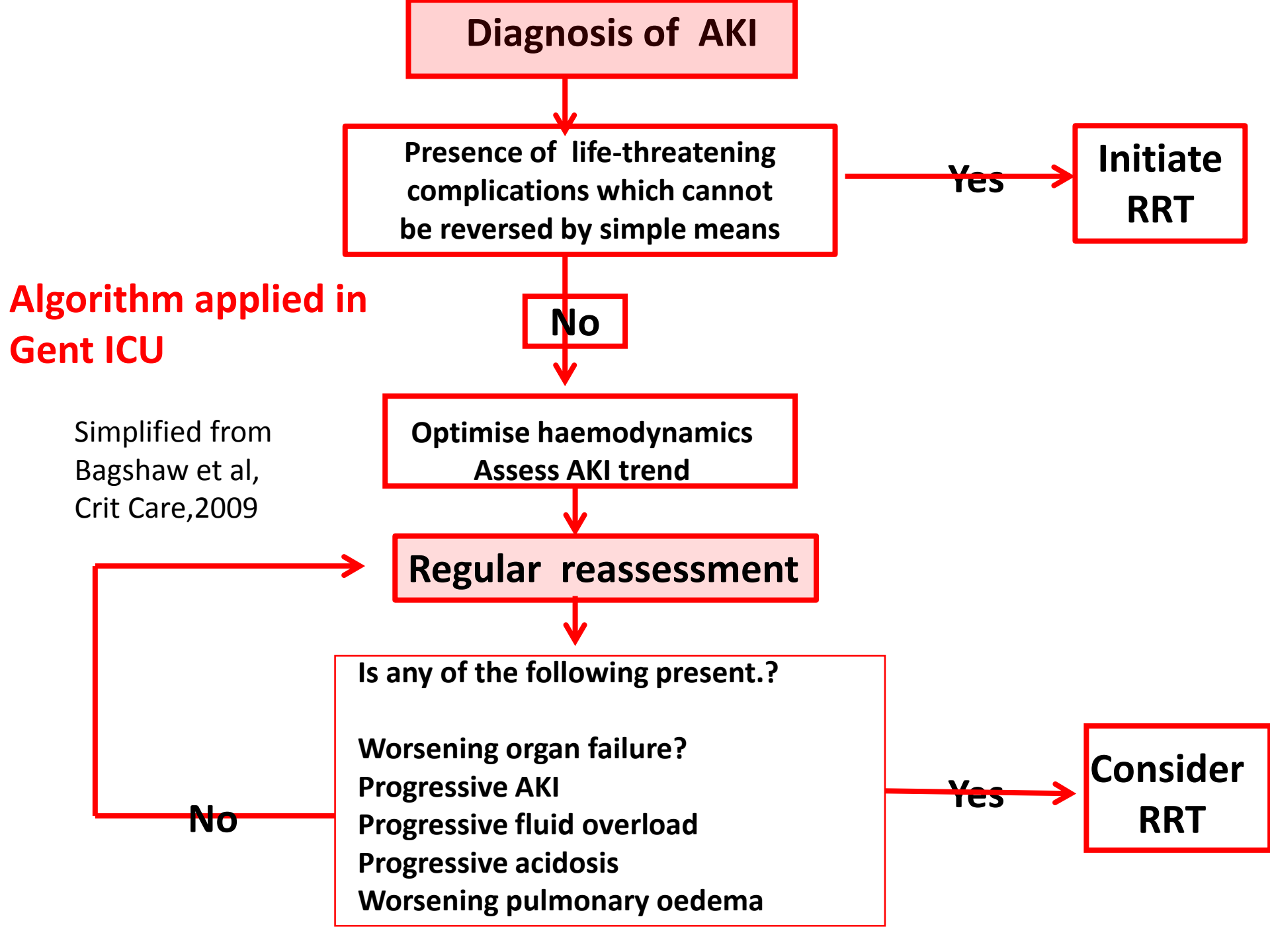
ARR, absolute risk reduction in primary end point; KDIGO, Kidney Disease: Improving Global Outcomes RRT, renal replacement therapy; SOFA, sepsis-related organ failure assessment. *Oliguria or anuria for >72 h, serum urea >40 mmol/l, serum potassium >6 mmol/l (or >5.5 mmol/l despite medical intervention), blood pH <7.15, or acute pulmonary oedema causing significant hypoxaemia.

Bagshaw, Wald, Nature Reviews in Nephrol 2016

Advantages and shortcomings of earlier RRT initiation in AKI

Advantages	Shortcomings
More effective reversal of volume expansion, particularly in diuretic-resistant patients	Exposure to complications associated with supplemental vascular access (both at time of insertion and therafter- infections, thrombosis, emboli,…)
Better control of electrolyte and acid base status	Exposure to complications associated with RRT (e.g. intradialytic hypotension, dysrhythmias, clearance of antibiotics- hypokalemia, hypoglycemia…..)
Pro active clearance of toxic low and middle molecular weight solutes	Higher cost, especially if patient was destined to recover kidney function
Avoidance of AKI-related emergencies, (eg cardiac dysrhythmias related to hyperkalemia	

Adapted from Wald, Bagshaw, Semin Nephrol 36:78-84, 2016



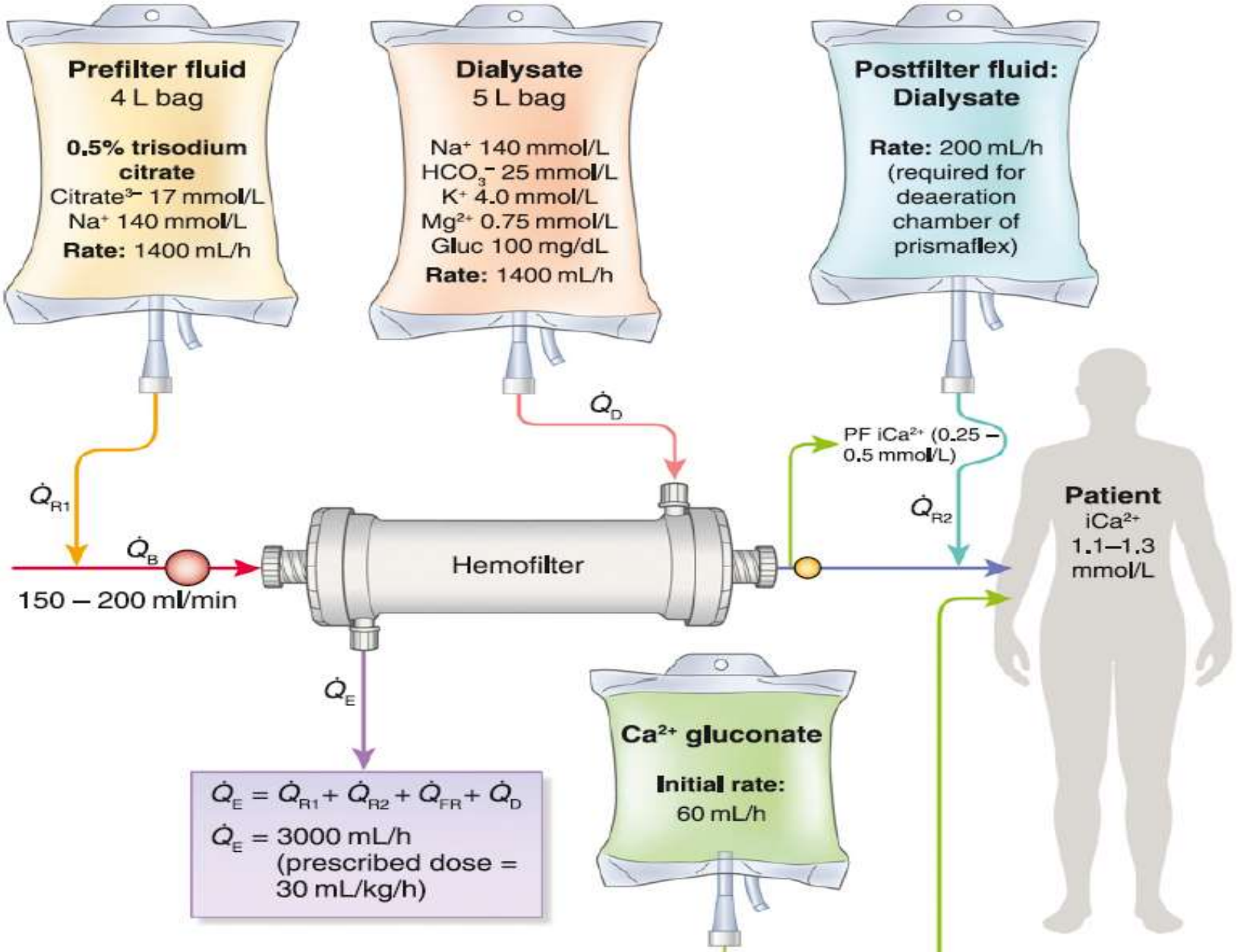
Algorithm applied in Gent ICU

Simplified from Bagshaw et al, Crit Care,2009

Anticoagulation strategies for RRT in AKI patients

Strategy	Advantages	Disadvantages
No anticoagulation	• No bleeding risk	• Lowest filter patency rates
Regional citrate anticoagulation	• Highest filter patency rates • No systemic anticoagulation, and therefore no increased bleeding risk	• Complexity of protocols and required monitoring • Risks include citrate toxicity, metabolic alkalosis
Unfractionated heparin	• Ease of use, availability • Ability to monitor • Ability to reverse with protamine	• Systemic anticoagulation and bleeding risk
Low-molecular-weight heparin	• Can be prescribed as fixed dose or titrated to activity	• Systemic anticoagulation and bleeding risk • Expense, need for specialized monitoring (anti-Xa levels) • Less effective reversal with protamine

Heung, Yessayan, Crit Care Clin 33 (2017) 365–378



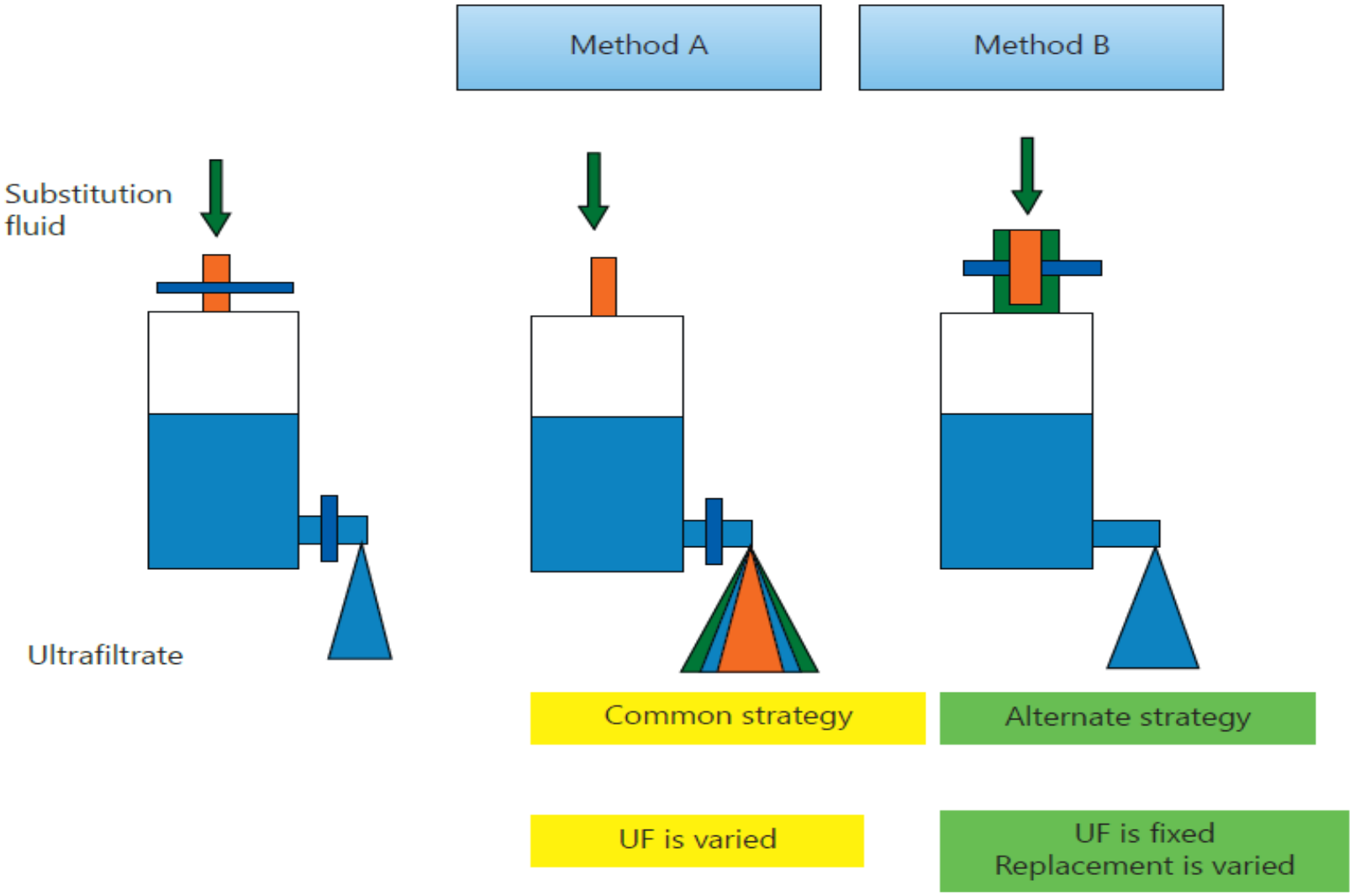
CVVHDF schematic for a given clinical patient of 100 kg BW with liver failure, lactic acidosis , sepsis

Advantages and disadvantages of pre-and post-filter substitution

Pre-filter	Post-filter
Advantages	
UF rate is not limited by Q_B	Clearance of solutes is directly related to UF rate
Enhanced elimination of urea from RBC's	A higher solute clearance rate is produced
Filter life is increased as the Hct throughout the filter remains low	Delivery of specified solutes and concentrations directly to the solution
Filter life is increased which may increase filter lifespan and solute clearance, even though hourly solute clearance is decreased	
Disadvantages	
Solute concentrations are decreased and thus clearance is decreased	UF rate is limited by Qb. You cannot order too much UF because the end-filter Hct will be too high
	Because UF rate is limited by FF you may not reach optimal dose
	Filter life may be decreased by high end-filter Hct

Huang et al, in Ronco, Bellomo, Kellum (eds): Crit Care Nephrol, ed 2.Saunders, 2009, 1370–1374.

Two different methods to achieve the target fluid balance with CRRT



Murugan et al. Blood Purif 2016;42:266–278

Effects of dialysis modality on bleeding complications and transfusion requirements in critically ill patients with AKI

	IHD	CVVH	P-value
Number of patients	130	122	
Major all-cause bleeding	23%	26%	0.95
Rate of RRT-related blood loss events	30.4%	57.4%	0.01
Total blood volume loss per patient (ml)	112.5±222.7	222.3±291.9	P<0.01
Overall transfusion rates			NS

Pschowski et al , Anaesth Intensive Care. 2015, 43:764-770.

Renal Replacement Therapy for AKI - treatment dose -

- retrospective analysis of dosing patterns of continuous renal replacement therapy in 115 critically ill patient treated in a single large tertiary care hospital in the U.S.

- average treatment time: 16.1 ± 3.5 hours per day
- average treatment dose: 1.36 ± 0.3 liter per hour
- mean delivered dose: 68% of prescribed dose

Venkataraman R et al. *J Crit Care* 2002; 17: 246

RCTs comparing the hemodynamic tolerability of CRRT vs IHD

	CRRT (n)	IHD (n)	Definition of Hemodynamic Tolerability	Outcome
Misset et al [a][4]	27	27	Amplitude of MAP change (lowest recorded every hour to highest recorded), and episodes of MAP reduction by > 10 mmHg	No significant difference (p = 0.72 and 0.73, respectively)
Augustine et al [6]	40	40	Difference between mean MAP in 12 hours prior to RRT and during RRT	Significant drop in MAP during IHD (p = 0.04)
Uehlinger et al [7]	70	55	Number of hypotensive events (MAP < 65) during RRT	No significant difference (p = 0.36)
Vinsonneau et al [8]	175	184	Number of hypotensive events (SBP < 80, or drop of greater than 50 mmHg) during RRT	No significant difference (p = 0.47)

[a]A cross-over study with a 24-hour wash-out period.

Fieghen et al. BMC Nephrology 2010, 11:32

Hemodynamic tolerability of SLED vs CRRT

	CRRT (n = 86)	SLED (n = 39)	p-value
MAP prior to treatment session (mmHg)	74.1 ± 10.0	76.4 ± 13.1	0.34
Vasopressor requirement prior to RRT session (%)	62 (72.1)	19 (48.7)	0.01
Volume ultrafiltered per session (mL)	1823 ± 1464	1915 ± 1302	0.74
Sessions associated with > 20% reduction in MAP (%)	16 (18.6)	15 (38.5)	0.02
Sessions with vasopressor escalation (%)[a]	34 (39.5)	10 (25.6)	0.13
Unstable sessions[b] (%)	43 (50.0)	22 (56.4)	0.51

Continuous variables are presented as mean ± SD. Categorical variables are presented as number of sessions (%).

SLED = sustained low efficiency dialysis.

CRRT = continuous renal replacement therapy.

MAP = mean arterial pressure.

[a]Includes any increase in pressor dosage, as well as initiation of pressors.

[b]Defined as a treatment associated with a > 20% intra-treatment reduction in MAP or a treatment on which an escalation in pressor requirement occurred.

Fieghen et al. BMC Nephrology 2010, 11:32

Guidelines to improve hemodynamic tolerance of IHD in critically ill patients with AKI

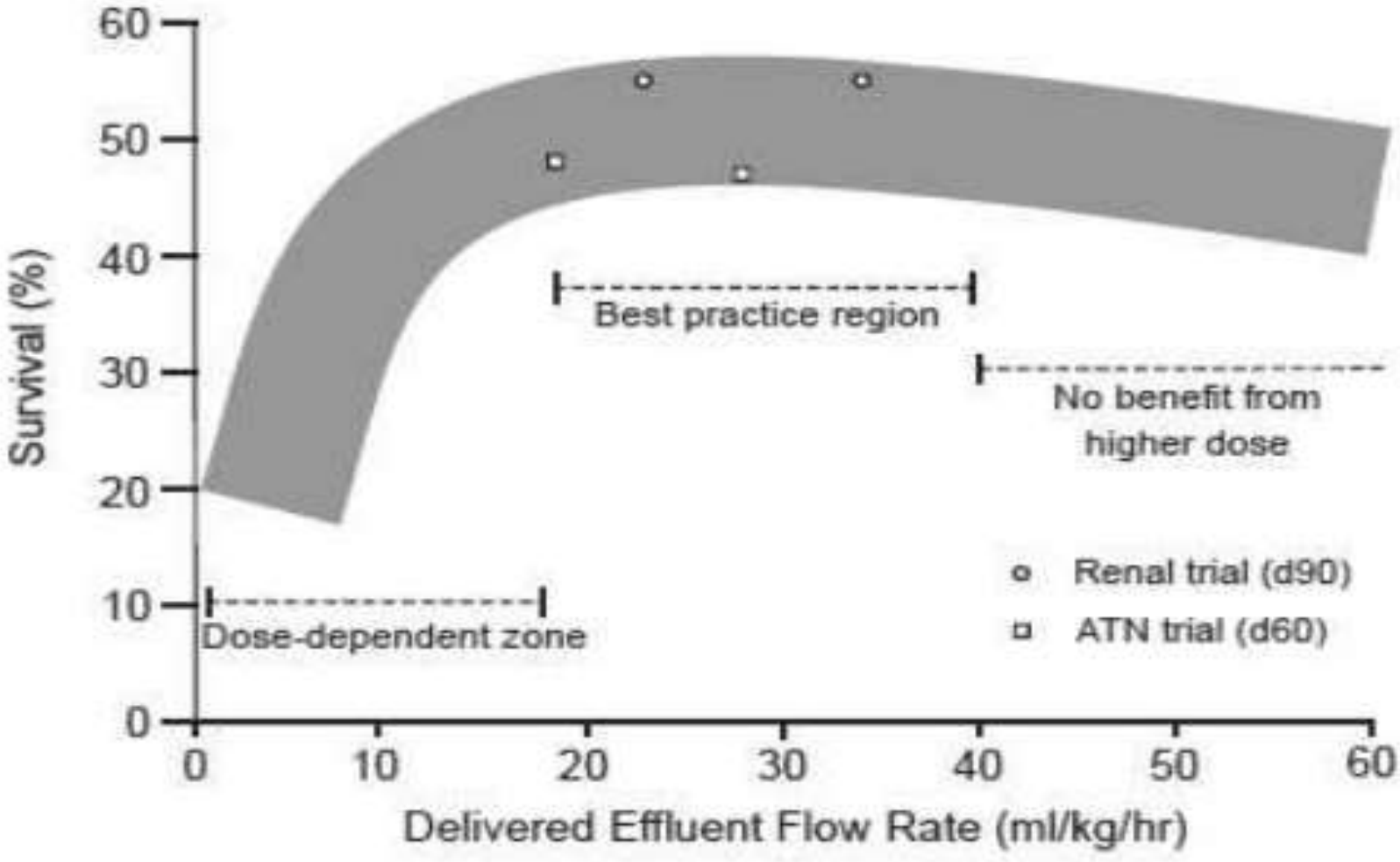
Recommendations for systemic use	-Use only modified cellulosic membranes rather than cuprophane -Connect the two lines of the circuit, which have been filled with 0.9% saline, to the catheter simultaneously -Set dialysate sodium concentration to ≥145 mmol/L -Limit the maximal blood flow to 150 mL/min with a minimal session duration of 4 hrs -Set dialysate temperature to ≤37° C
Advice for the most hemodynamically unstable patients	-Start session by dialysis and continue with ultrafiltration alone -Cool dialysate at 35° C
Additional recommendations	-Stop vasodilator therapy -Start session without ultrafiltration, then adapt UF/h rate according to hemodynamic response -Strictly adapt ultrafiltration order to patient's volemia and weight loss requirement

RCTs of RRT intensity , start of RRT and modality selection for CS AKI

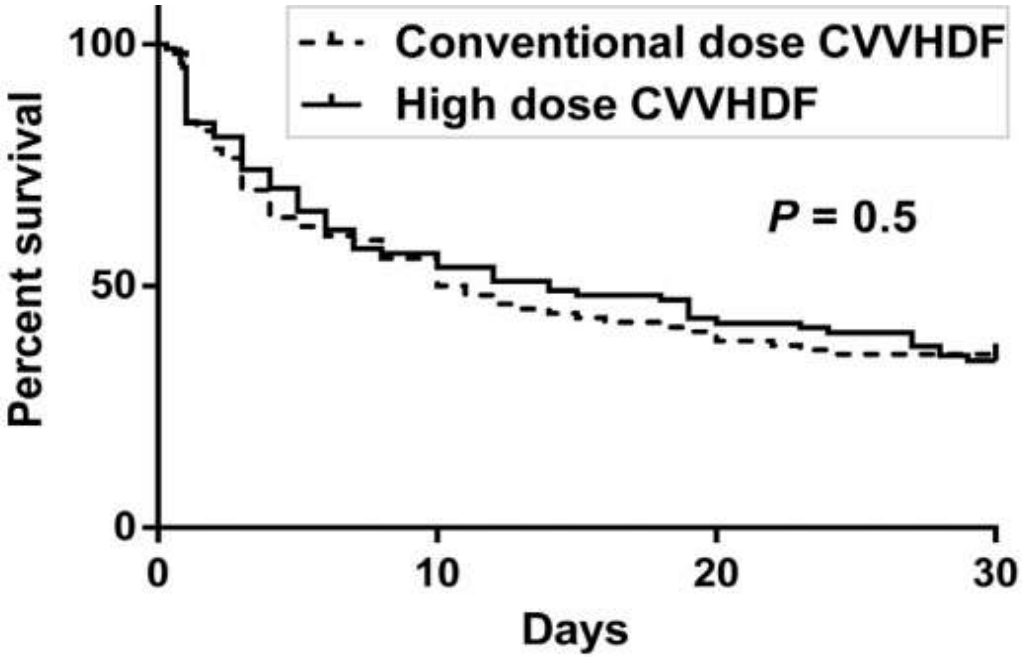
Trial	Multicentre (Yes/No)	Double blinded (Yes/No)	No. of patients		Intervention	Primary outcome data
			Total	Cardiac surgery		
RENAL (2009)	Yes	No	1,508	269 (17.8%)	Intensity of RRT	High-intensity continuous RRT did not reduce all-cause mortality at 90 days after surgery
ATN (2008)	Yes	No	1,124	463 (41.2%)	Intensity of RRT	No difference in mortality, rate of recovery of kidney function, duration of RRT and evolution of nonrenal organ failure between high-intensity and low-intensity RRT
AKIKI (2016)	Yes	No	620	126 (20.3%)	Timing of initiation of RRT	No difference in all-cause mortality between early and delayed initiation of RRT; 49% of patients in the delayed RRT group did not need RRT
ELAIN (2016)	No	No	231	108 (46.8%)	Timing of initiation of RRT	Early RRT initiation reduced mortality in the first 90 days after cardiac surgery
Kielstein *et al.* (2004)	No	No	39	NA	CVVH versus EDD	No significant difference in average mean arterial blood pressure, heart rate, cardiac output, systemic vascular resistance, urea reduction rate and catecholamine dose between interventions
CONVINT (2014)	No	No	252	13 (5.16%)	CVVH versus IHD	No survival difference after 30 days between CVVH and IHD group
Vinsonneau *et al.* (2006)	Yes	No	360	107 (29.7%)	CVVHDF versus IHD	No survival difference after 60 days between CVVHDF and IHD group
Badawy & Samir (2013)	No	No	80	80 (100%)	CVVHDF versus EDD	No significant difference in mortality and renal recovery between CVVHDF and EDD group; EDD seemed to be more cost-effective

Wang, Bellomo, Nature Rev Nephrol ,Adv Access,2017

Possible relationship between delivered dose of CRRT and survival, with results from the ATN and RENAL trials.



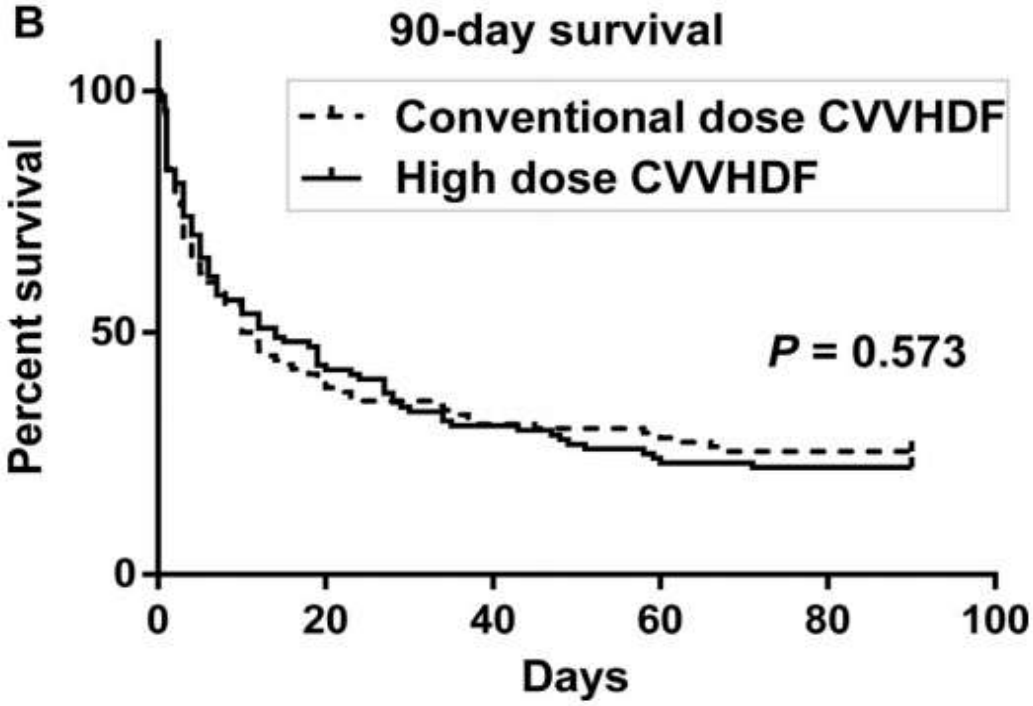
Adapted from Prowle JR et al, Crit Care. 2011;15:207-214.



Very high vs conventional CVVHDF dose in septic AKI: an RCT

Conventional dose : 40ml/kg/h

High dose: 80 ml/kg/h

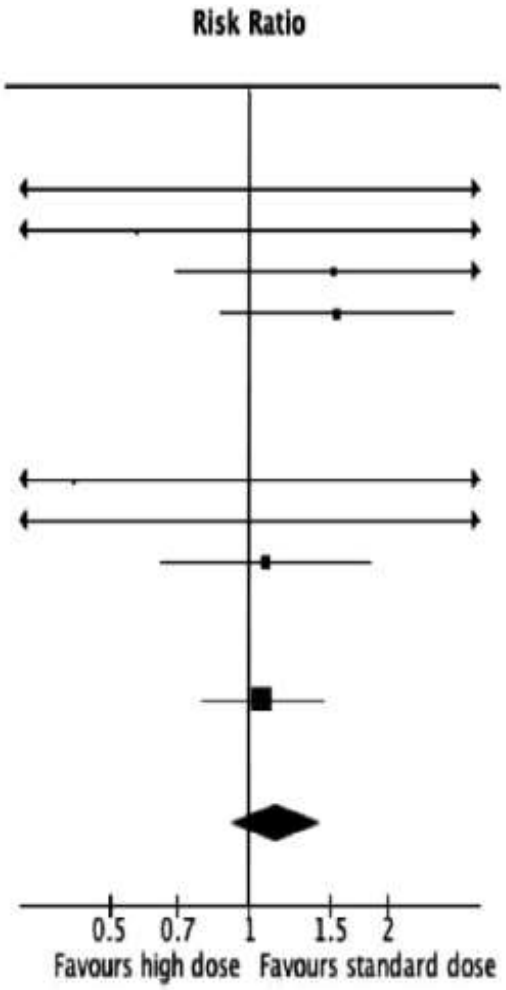


Jun Tak Park et al, AJKD 68:599-608, 2016

Effect of high dose vs. standard dose RRT on dialysis dependence- meta-analysis

Study	High dose Events	Total	Standard dose Events	Total	Weight	Risk Ratio
CRRT						
Bouman 2002	0	22	0	39		Not estimable
Jiang 2005	0	15	1	13	0.5%	0.29 [0.01, 6.60]
Saudan 2006	2	61	2	35	1.4%	0.57 [0.08, 3.90]
Tolwani 2008	11	36	8	40	8.0%	1.53 [0.69, 3.37]
Bellomo 2009	27	399	18	411	14.9%	1.55 [0.86, 2.76]
IHD or SLED						
Conger 1975	0	5	0	2		Not estimable
Gillum 1986	0	7	1	9	0.5%	0.42 [0.02, 8.91]
Schiffl 2002	2	58	0	43	0.6%	3.73 [0.18, 75.74]
Faulhaber-Walter 2009	18	45	17	46	18.5%	1.08 [0.64, 1.82]
Combined						
Palevsky 2008	65	256	64	269	55.7%	1.07 [0.79, 1.44]
Total (95% CI)		**904**		**907**	**100.0%**	**1.15 [0.92, 1.44]**

P = 0.23, I^2 = 0%



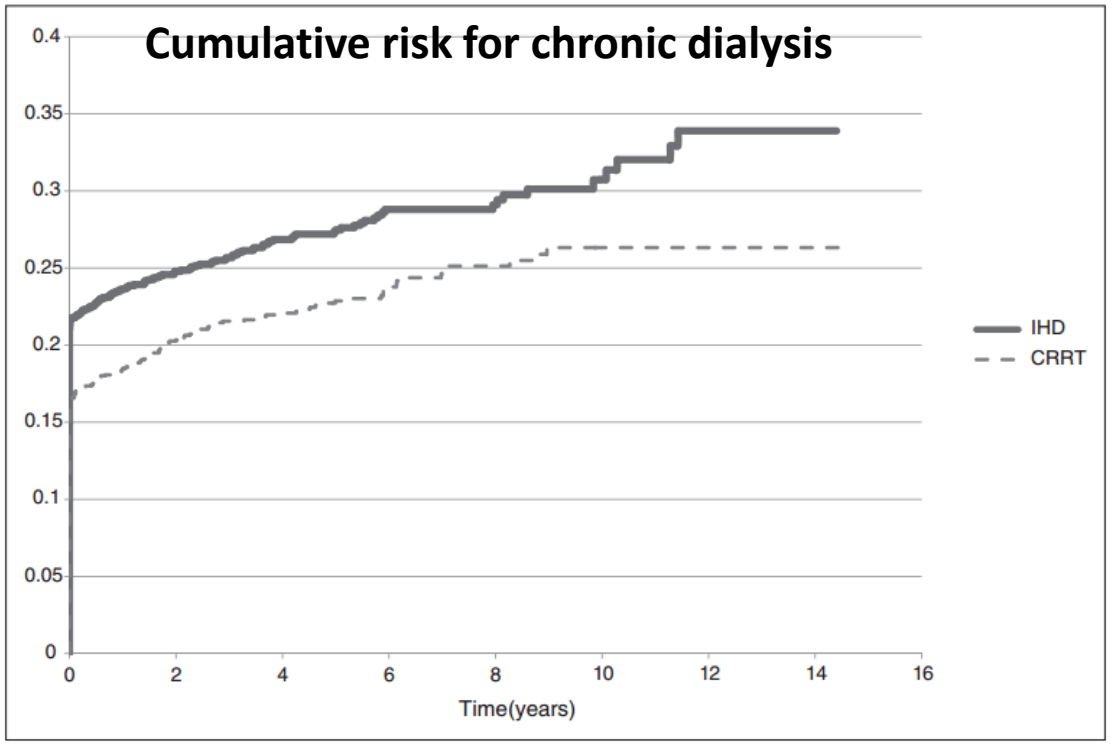
Van Wert et al, Crit Care Med 2010; 38:000–000

CHAPTER 5.8: DOSE OF RRT IN AKI

- **5.8.3: We recommend delivering a Kt/V of 3.9 per week when using intermittent or extended RRT in AKI. (*1A*)**

- **5.8.4: We recommend delivering an effluent volume of 20-25 ml/kg/h for CRRT in AKI *(1A).* This will usually require a higher prescription of effluent volume. (*Not Graded*)**

Late outcomes in critically ill AKI patients surviving 90 days and initiated on CRRT vs IHD



Outcome	CRRT ($n = 2,004$)		IHD ($n = 2,004$)		Hazard Ratio (95% CI) for CRRT vs IHD	p
	n (%)	Incidence Rate per 100 Person-Years	n (%)	Incidence Rate per 100 Person-Years		
Chronic dialysis	435 (22)	6.5	533 (27)	8.2	0.75 (0.65–0.87)	< 0.0001
Death	883 (44)	11.2	905 (45)	11.4	1.02 (0.91–1.14)	0.73

Wald et al, Crit Care Med 2014; 42:868–877

Specific indications for each modality of RRT

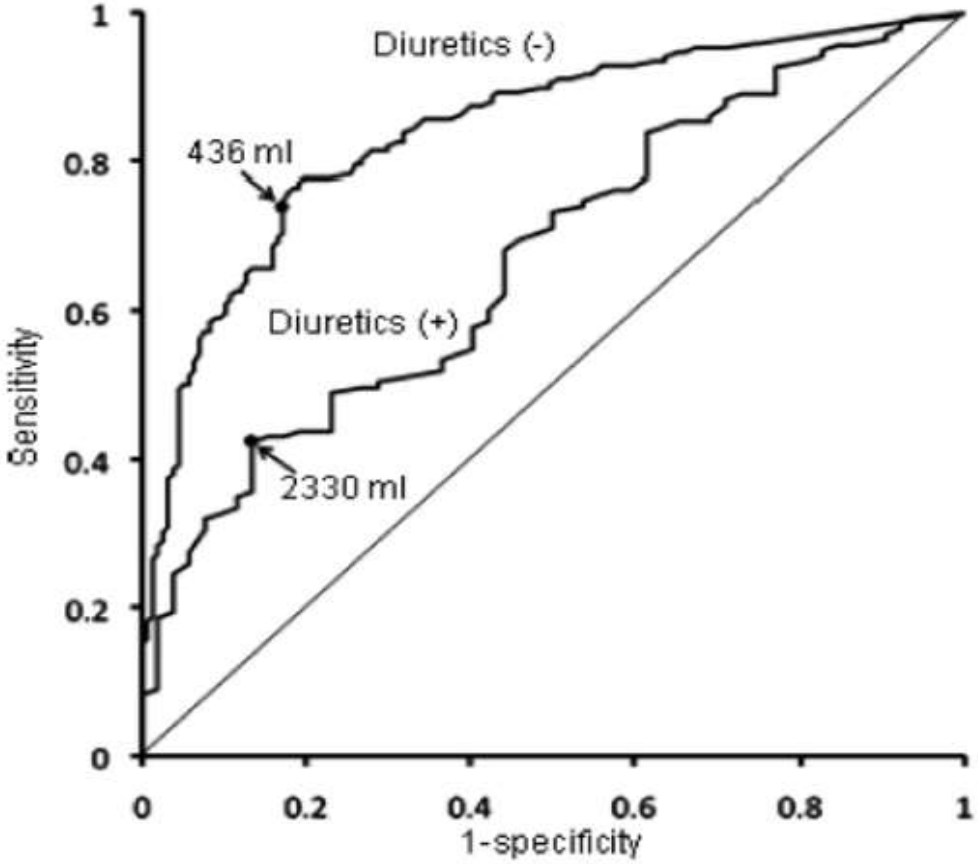
Goal	Hemodynamic Status	Preferred Modality
Urea clearance	Unstable	CRRT or PIRRT
	Stable	IHD
Severe hyperkalemia	Unstable/stable	IHD first
Metabolic acidosis	Unstable	CRRT/PIRRT or IHD first if hemodynamic instability caused by acidosis
Severe dysnatremia	Unstable/stable	CRRT
Severe hyperphosphatemia	Unstable/stable	CRRT
Brain injury	Unstable/stable	CRRT
Acute liver failure	Unstable/stable	CRRT

Heung, Yessayan, Crit Care Clin 33 (2017) 365–378

CHAPTER 5.6: MODALITY OF RRT FOR PATIENTS WITH AKI

- **5.6.1: Use continuous and intermittent RRT as complementary therapies in AKI patients. (*Not Graded*)**
- **5.6.2: We suggest using CRRT rather than standard intermittent RRT, for hemodynamically unstable patients. (*2B*)**
- **5.6.3: We suggest using CRRT, rather than intermittent RRT, for AKI patients with acute brain injury or other causes of increased intracranial pressure or generalized brain edema. (*2B*)**

Analysis for successful discontinuation of CRRT-role of diuretics



Impact of diuretics use on predictive ability of urine output. The area under the receiver operating characteristics curve of urine output for successful discontinuation of continuous renal replacement therapy was 0.671 (0.585–0.750) with diuretics and 0.845 (0.799–0.883) without diuretics. Urine output of 436 mL/day for patients without diuretics and of 2330 mL for those with diuretics had the highest accuracy.

Uchino et al, Crit Care Med 2009; 37:2576 –2582

Points of concern in post-AKI follow up

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THE LATE PROGNOSIS IN ACUTE TUBULAR NECROSIS

AN INTERIM FOLLOW-UP REPORT ON 14 PATIENTS

K. G. LOWE

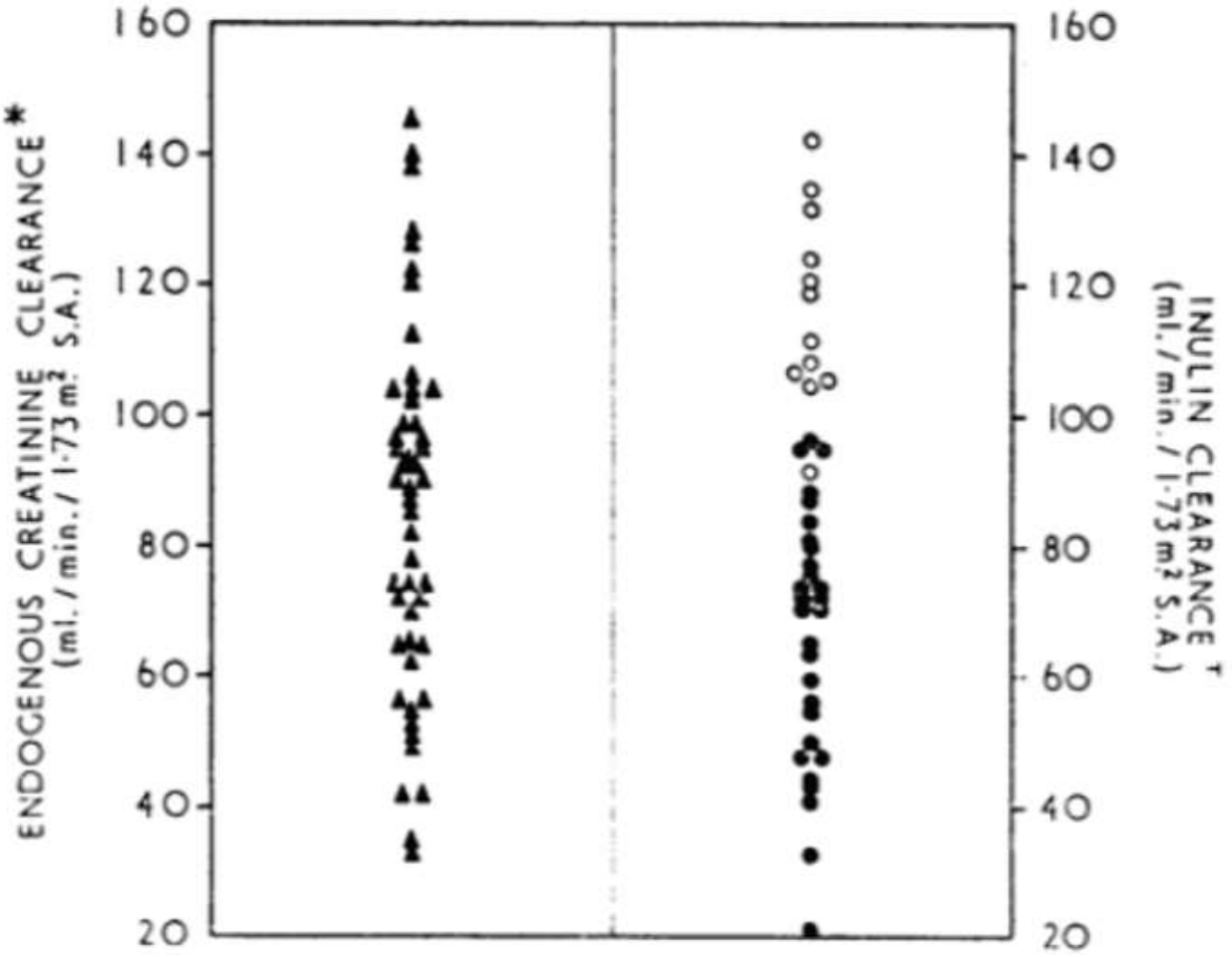
M.D. St. And., M.R.C.P.

LATE MEDICAL REGISTRAR AND TUTOR IN MEDICINE,
POSTGRADUATE MEDICAL SCHOOL OF LONDON

The slightly subnormal renal function in the late follow-up period is of interest. It may well be due to residua of the acute episode (scarring, vascular damage, &c.), but this remains open to speculation.

Lowe/Lancet/1952

Creatinine and inulin clearances after AKI

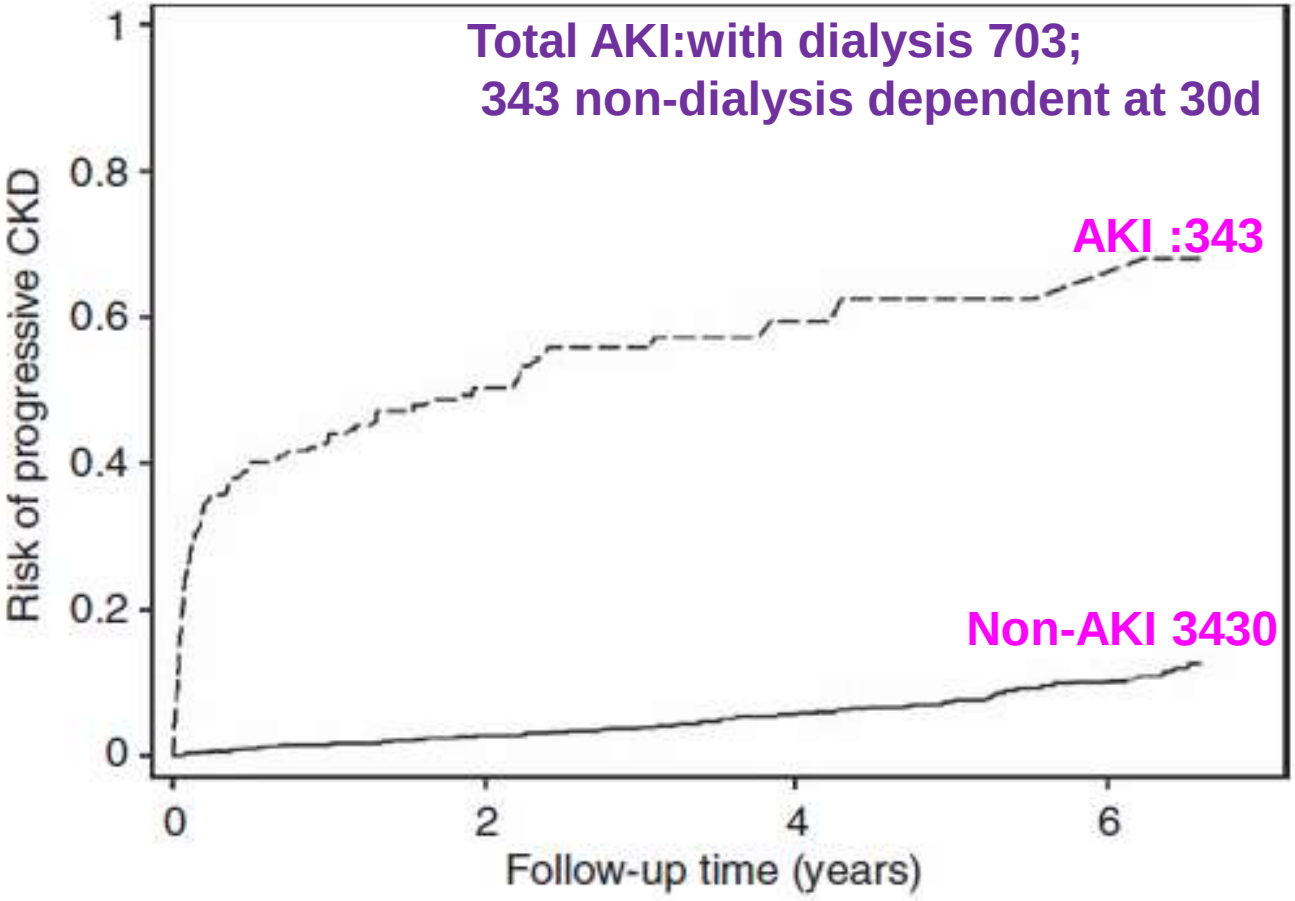


"Although it is widely accepted that the prognosis in general is good in patients who survive an episode of acute tubular necrosis, there remains some doubt about the exact degree to which glomerular and tubular function will ultimately recover."

50 patients were studied on average of 35 months after episode of AKI

Briggs et al , Brit Med J 3 : 513-516, 1967

Long-term renal prognosis after survival of dialysis requiring AKI –Kayser Permanente



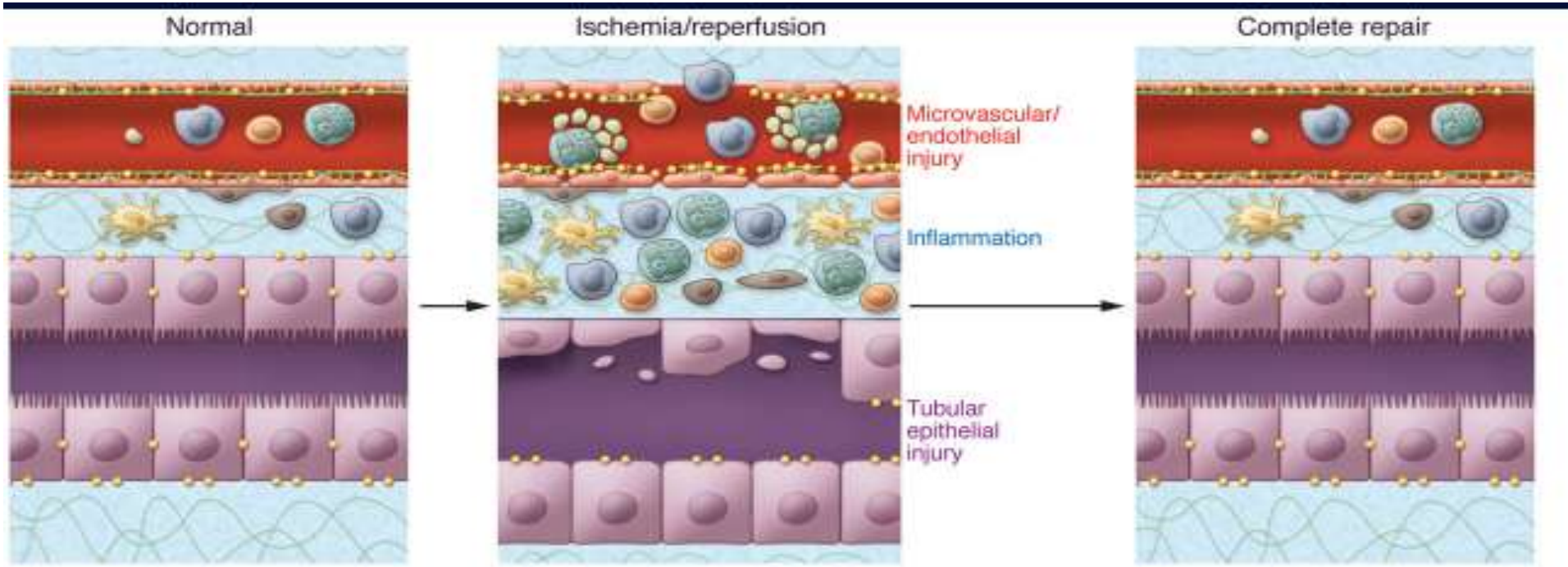
Pre-admission baseline
GFR > 45 ml/min/1.73 m²

Risk
28 x CKD

Risk for progressive CKD-stage 4 or higher

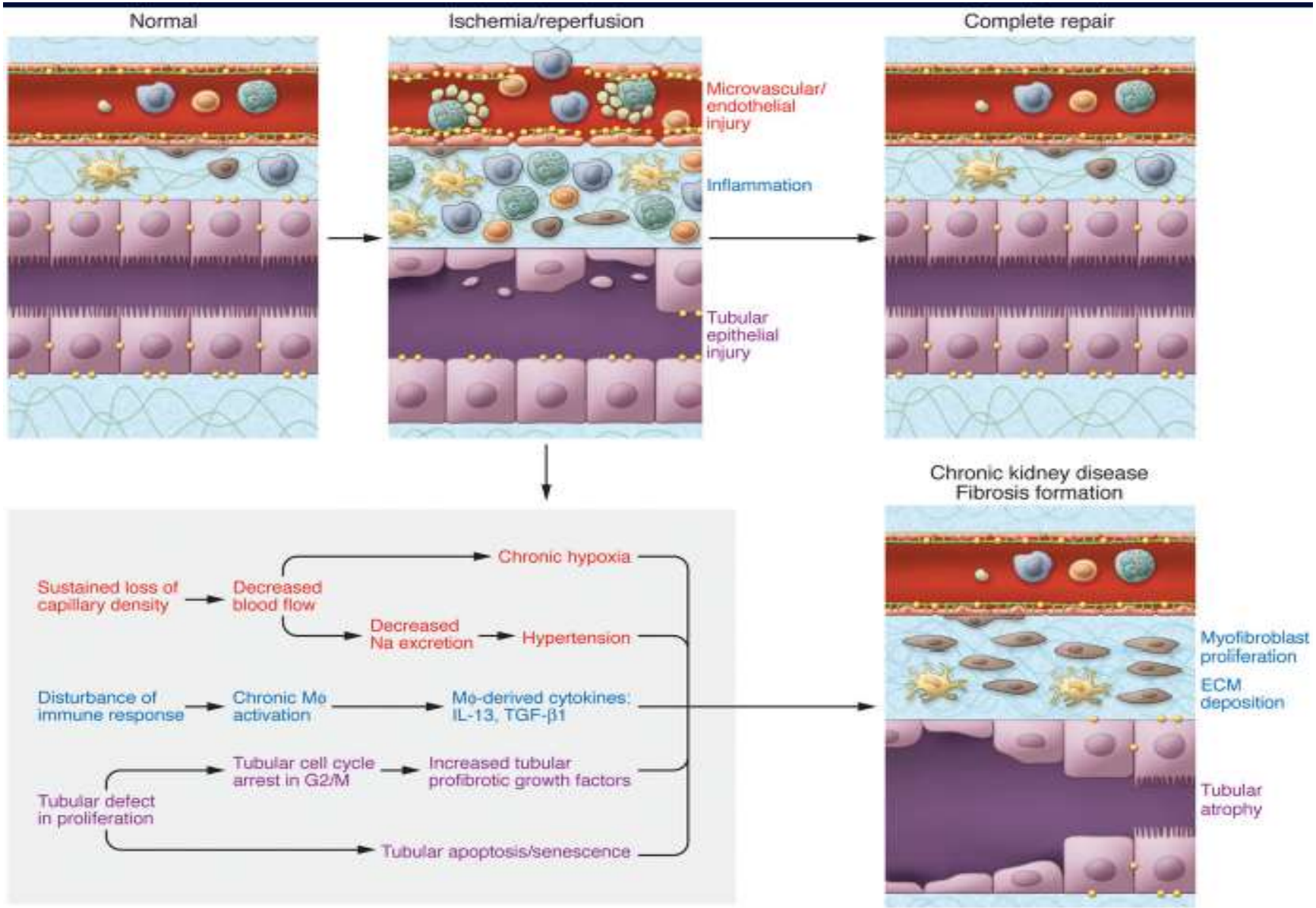
Lo et al, Kidney Int 2009, 76: 893–899

Normal repair in ischemic AKI



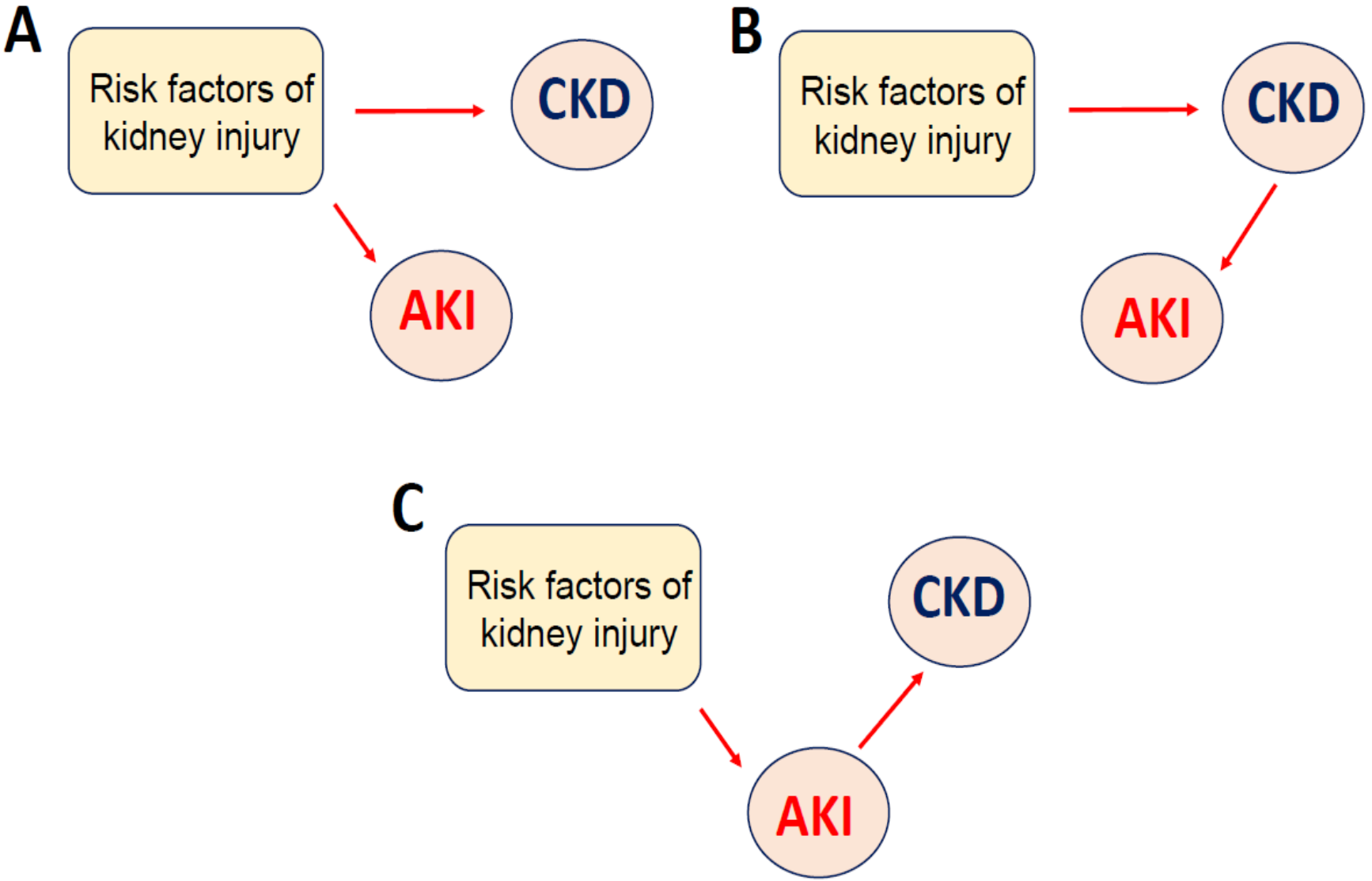
Bonventre , Chang J Clin Invest 121: 4210-4221, 2011

Abnormal repair in ischemic AKI

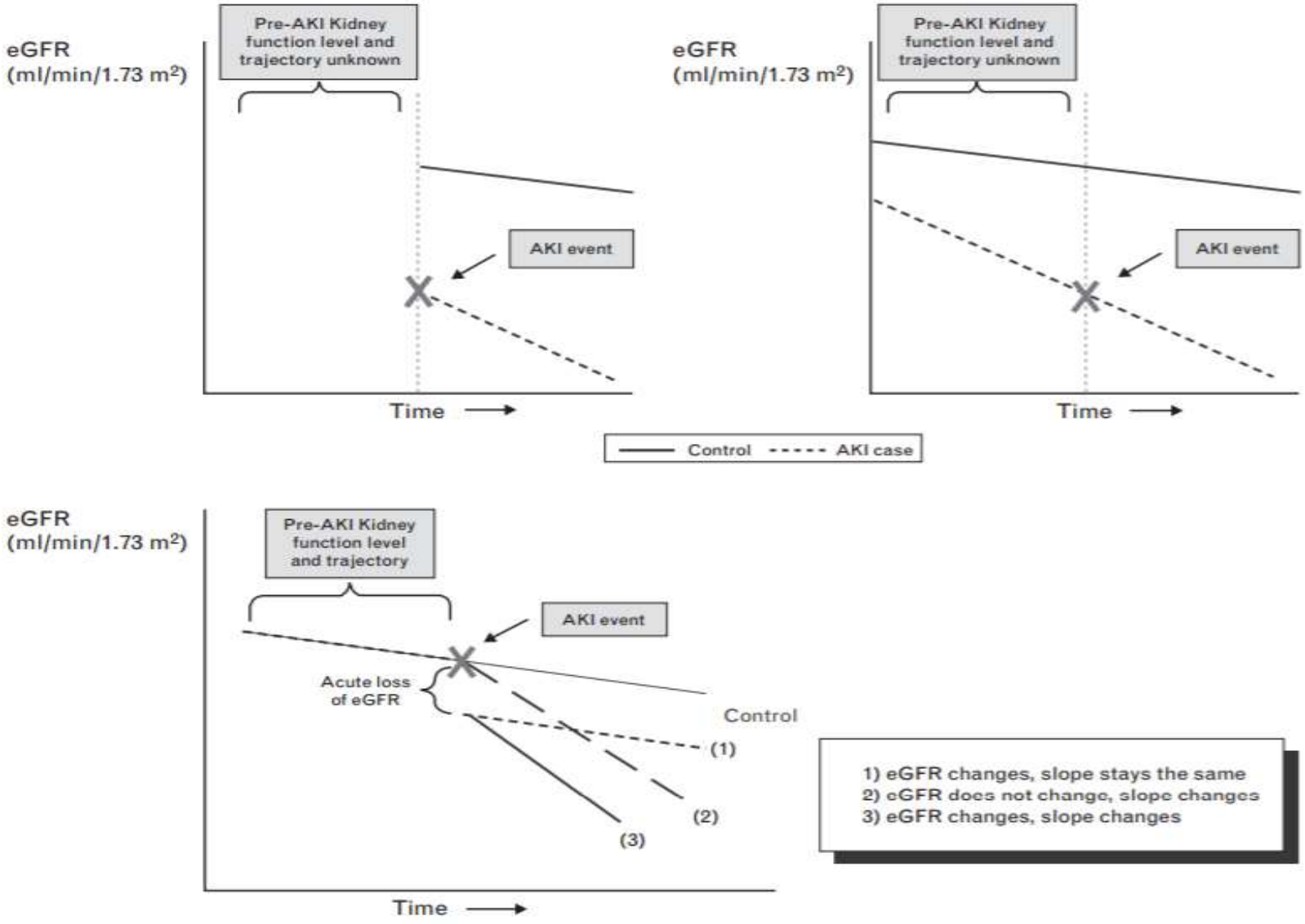


Bonventre , Chang J Clin Invest 121: 4210-4221, 2011

The interelationship AKI-CKD-AKI

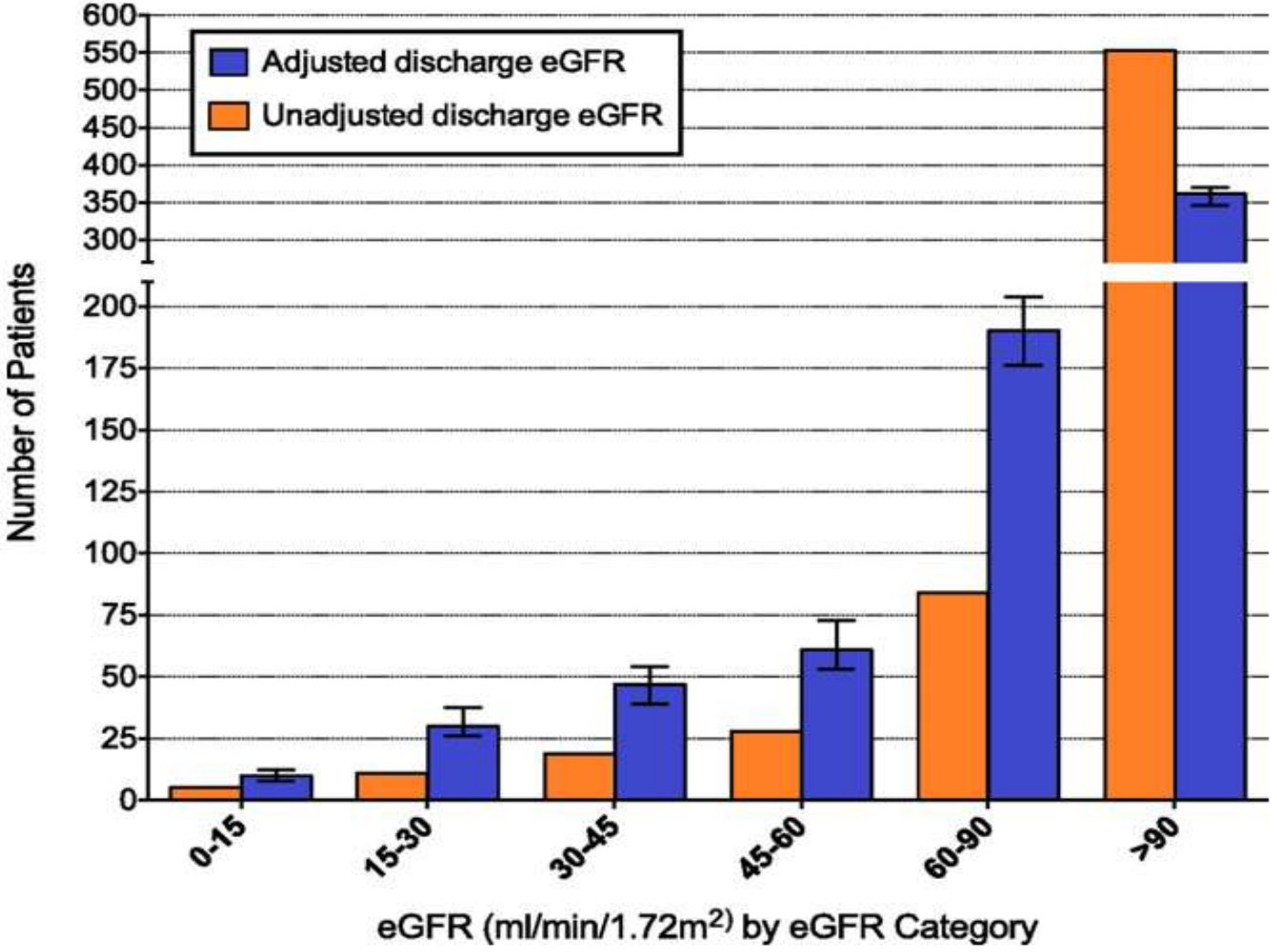


Only knowledge of baseline renal function and prior trajectory allows to define the impact of an AKI event on subsequent renal trajectory



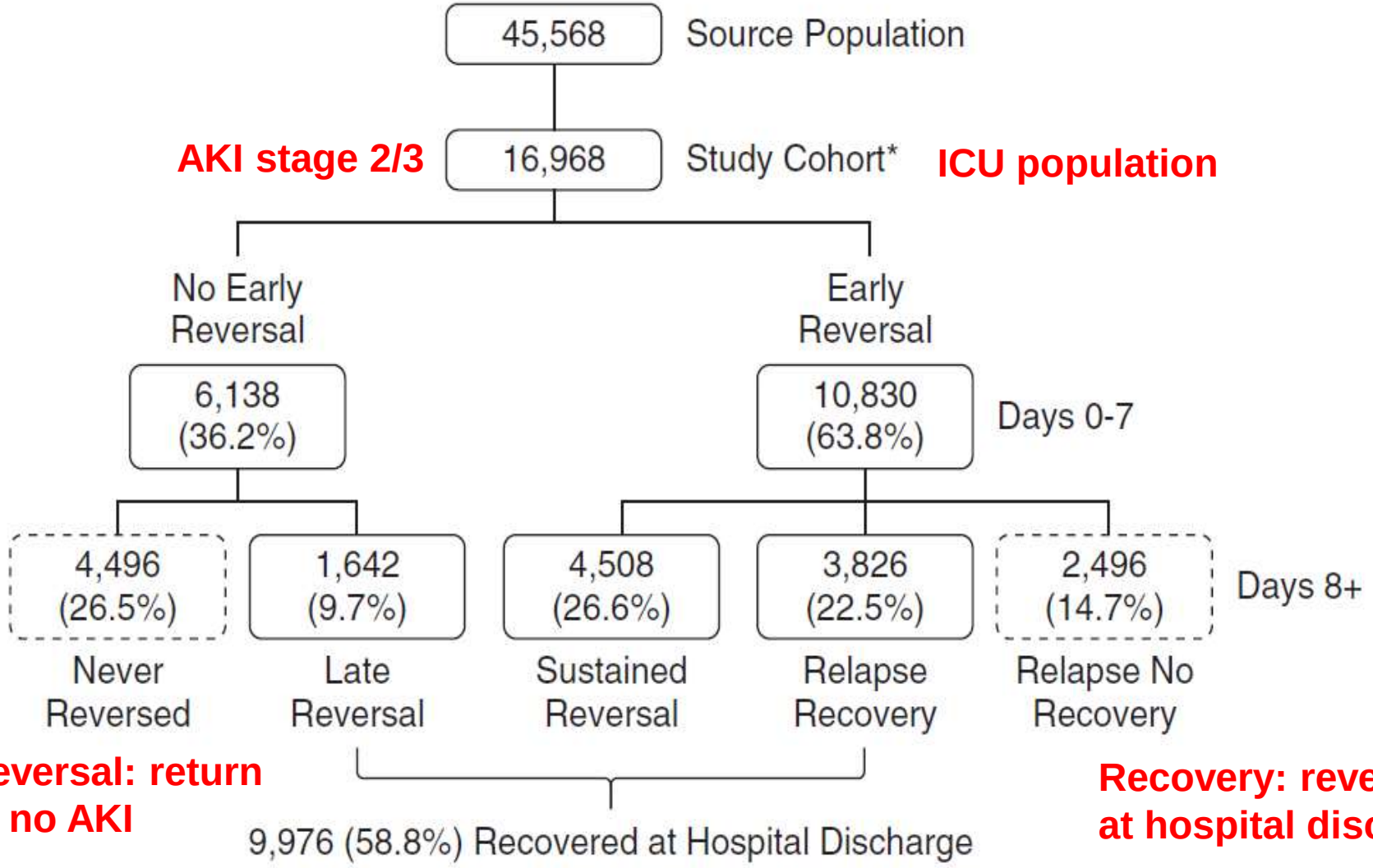
Liu et al, Curr Opin Nephrol Hypertens 18:241–245, 2009

CKD category at hospital discharge in 700 survivors of critical illness based on actual or for critical illness adjusted discharge creatinine



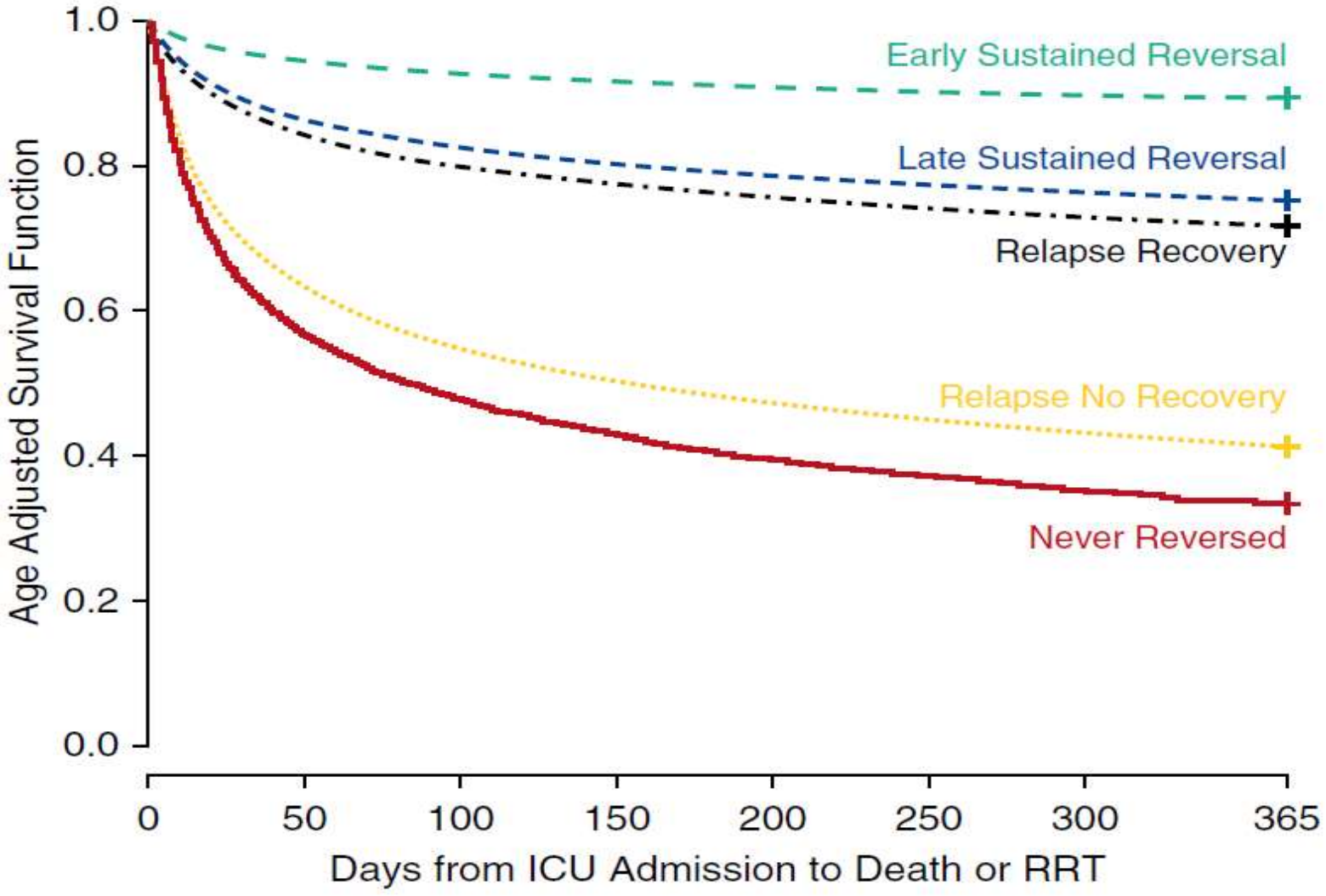
Prowle et al, cJASN, 9: 1015-1023; 2014

Definitions and epidemiology of reversal and recovery of AKI in critically ill



Kellum et al, Am J Respir Crit Care Med 195, 784–791, 2017

Different types of recovery after AKI-survival to death or RRT



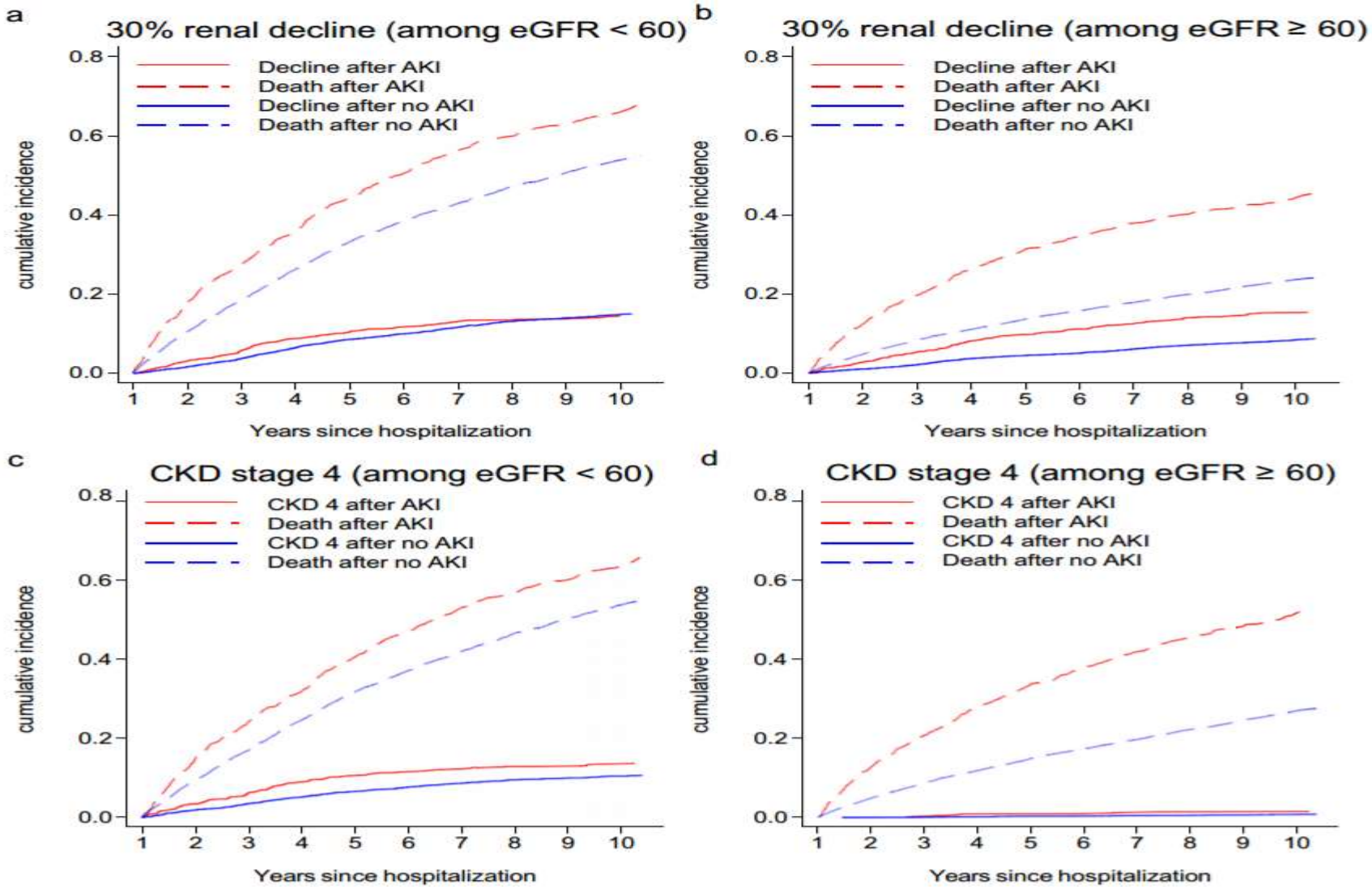
Kellum et al , Am J Respir Crit Care Med 195, 784–791, 2017

Development of CKD stage 3 or higher by 1 year after discharge according to AKI recovery pattern

	Stage 1 AKI[a]		Stage 2 AKI[a]		Stage 3 AKI[a]	
	RR (95% CI)	*P*	RR (95% CI)	*P*	RR (95% CI)	*P*
AKI by recovery pattern[a]						
No AKI	1.00 (reference)	—	1.00 (reference)	—	1.00 (reference)	—
≤2 d	1.40 (1.32-1.48)	<0.001	1.85 (1.30-2.64)	<0.001	1.80 (1.33-2.45)	<0.001
3-10 d	2.05 (1.86-2.27)	<0.001	1.58 (1.02-2.44)	0.04	1.83 (1.41-2.37)	<0.001
Still elevated after 10 d	2.33 (2.10-2.59)	<0.001	3.00 (2.32-3.88)	<0.001	3.48 (2.96-4.10)	<0.001
No Scr measure in 10-d postpeak period	—	—	—	—	—	—
Baseline albuminuria						
0-<30 mg/g	1.00 (reference)	—	1.00 (reference)	—	1.00 (reference)	—
30-300 mg/g	1.11 (1.09-1.13)	<0.001	1.40 (1.30-1.50)	<0.001	1.39 (1.30-1.49)	<0.001
>300 mg/g	1.35 (1.28-1.43)	<0.001	1.71 (1.50-1.95)	<0.001	1.72 (1.51-1.95)	<0.001

Heung et al, Am J Kidney Dis. 67(5):742-752, 2016

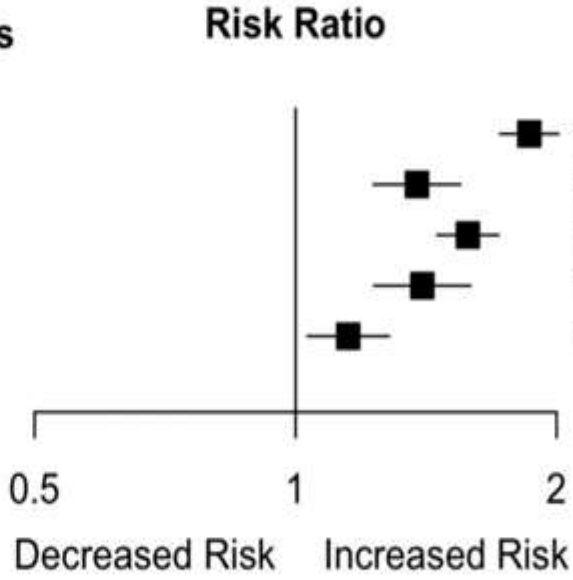
Cumulative incidences of subsequent renal progression (solid line) for those with (red) and without (blue) an AKI admission in 2003, grouped by postdischarge eGFR and accounting for the competing risk of death (dashed line) – Grampian study population



Sawhney et al Kidney Int (2017) 92 : 440–452, 2017

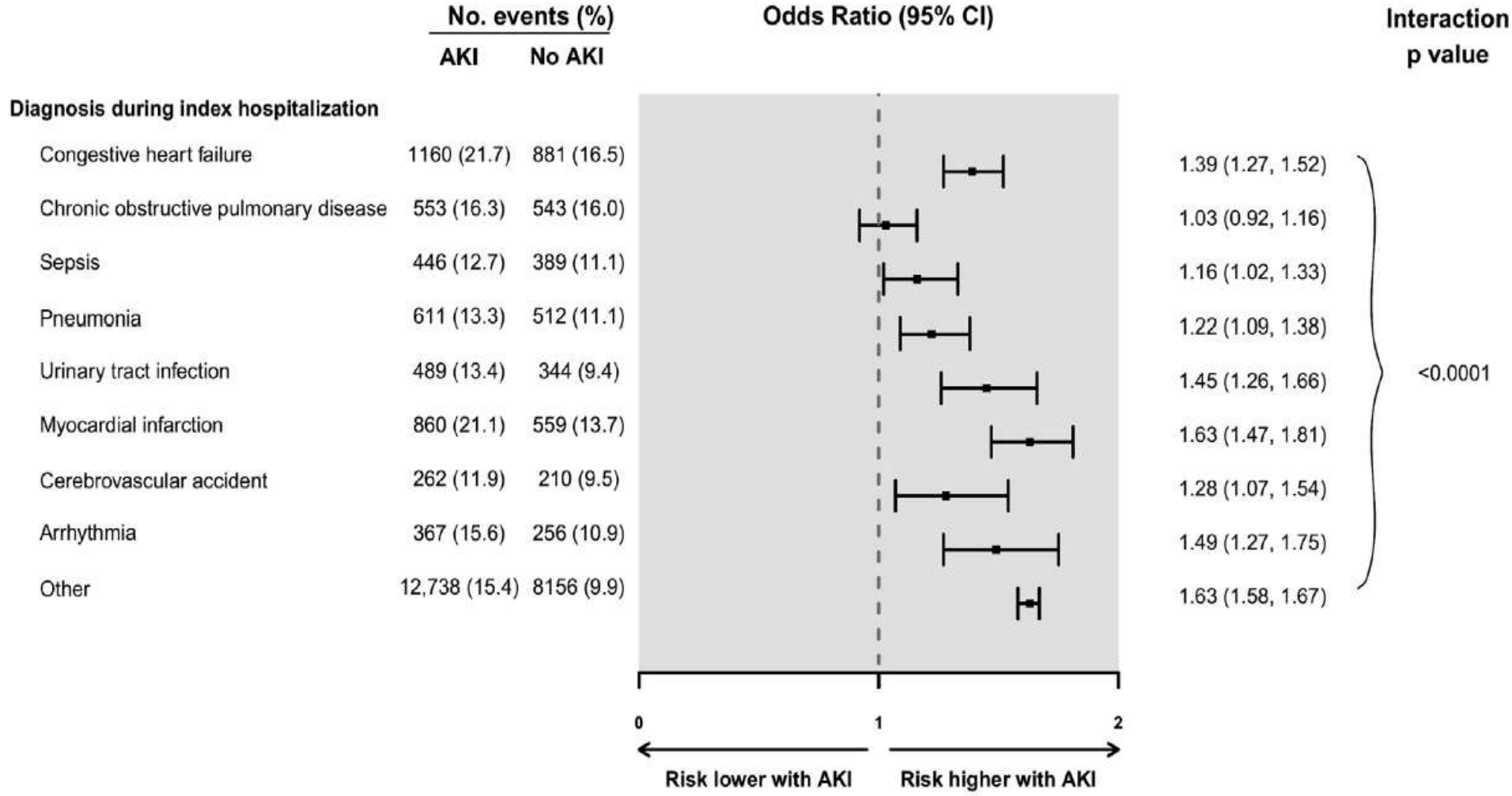
Increased risk of cardiovascular events post-AKI

Study	Studies	Participants	Events	RR (95% CI)	I2 (%)
CVD Mortality	6	54641	4195	1.86 (1.72-2.01)	0
MACE	6	40489		1.38 (1.23-1.55)	47
CHF	10	90251	3866	1.58 (1.46-1.72)	10
AMI	10	90498	5057	1.40 (1.23-1.59)	43
Stroke	5	72347	1955	1.15 (1.03-1.28)	0



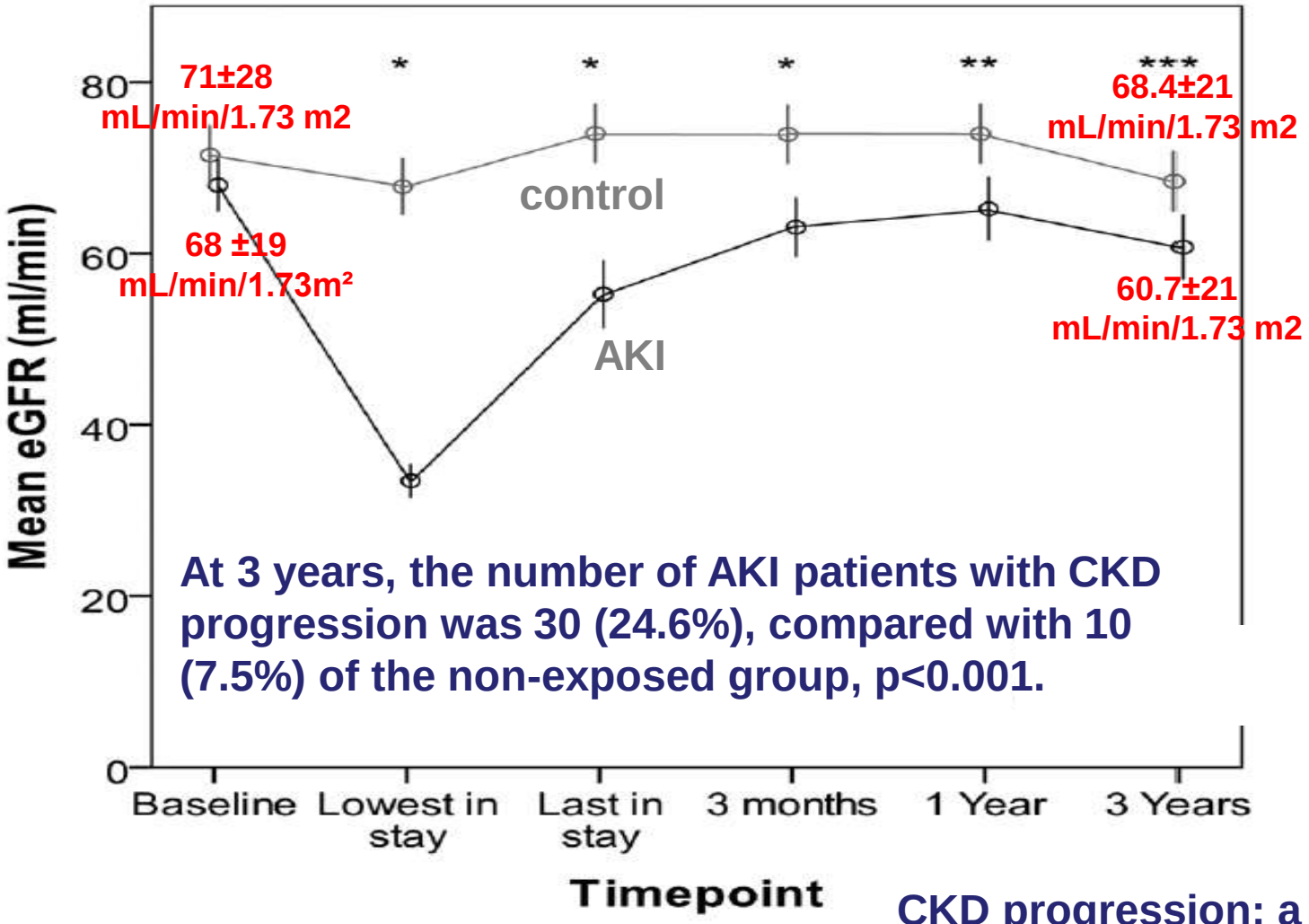
Odutayo et al, JASN, 28:377-387; 2014

Association of AKI with 30-day hospital readmission in a propensity-matched cohort-most responsible diagnosis during index hospitalization



Silver et al, The American Journal of Medicine (2017) 130, 163-172

Three-year outcomes after AKI: results of a prospective parallel group cohort study



At 3 years, the number of AKI patients with CKD progression was 30 (24.6%), compared with 10 (7.5%) of the non-exposed group, p<0.001.

Horne KL, et al. BMJ Open 2017;7:e015316

CKD progression: a decrease in eGFR of ≥25% associated with a decline in eGFR stage.

Three-year outcomes after AKI: results of a prospective parallel group cohort study

Number (%) of acute kidney injury and non-exposed groups with albuminuria (ACR ≥3 mg/mmol) and proteinuria (PCR ≥15 mg/mmol) at each time point

	3 months		1 year		3 years	
	AKI (n=150)	Non-exposed (n=150)	AKI (n=137)	Non-exposed (n=147)	AKI (n=125)	Non-exposed (n=138)
Albuminuria number (%)	48 (36.6)*	30 (20.5)	40 (31.5)	36 (25.4)	46 (40.7)***	26 (21.1)
Proteinuria number (%)	71 (50.7)*	53 (36.3)	50 (38.8)**	31 (22.1)	53 (46.5)**	36 (29.8)

Significance of comparisons between the AKI group and non-exposed group: *p<0.05, **p≤0.01, ***p≤0.001.

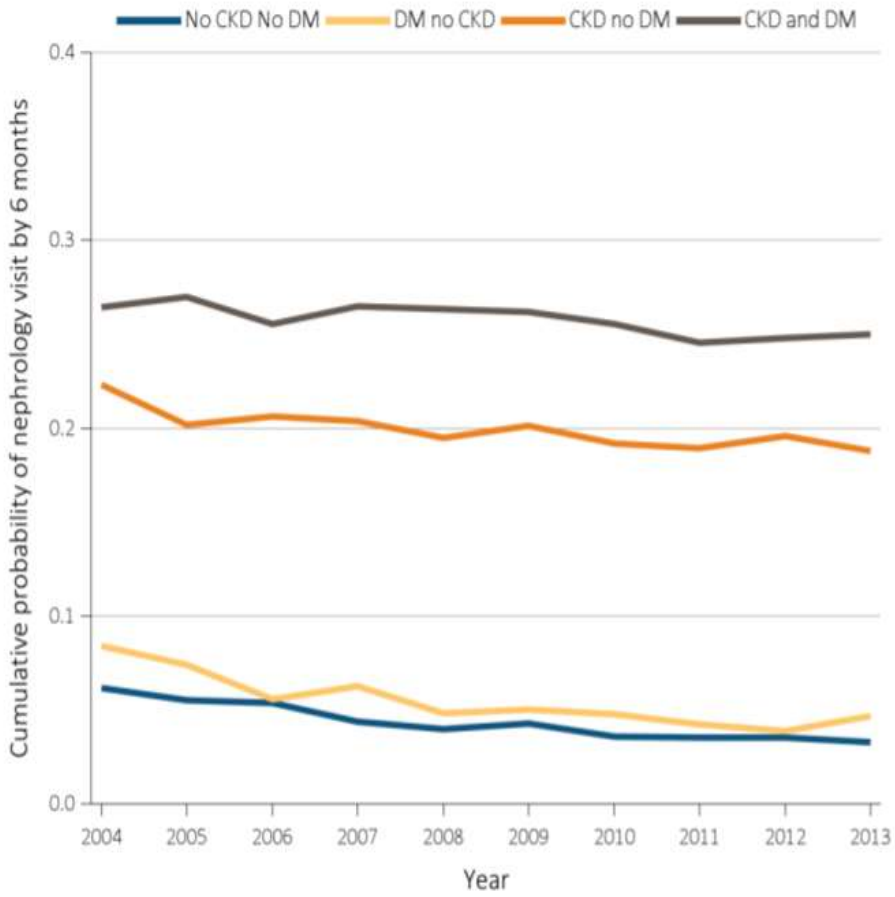
Horne KL, et al. BMJ Open 2017;7:e015316

CKD progression: a decrease in eGFR of ≥25% associated with a decline in eGFR stage.

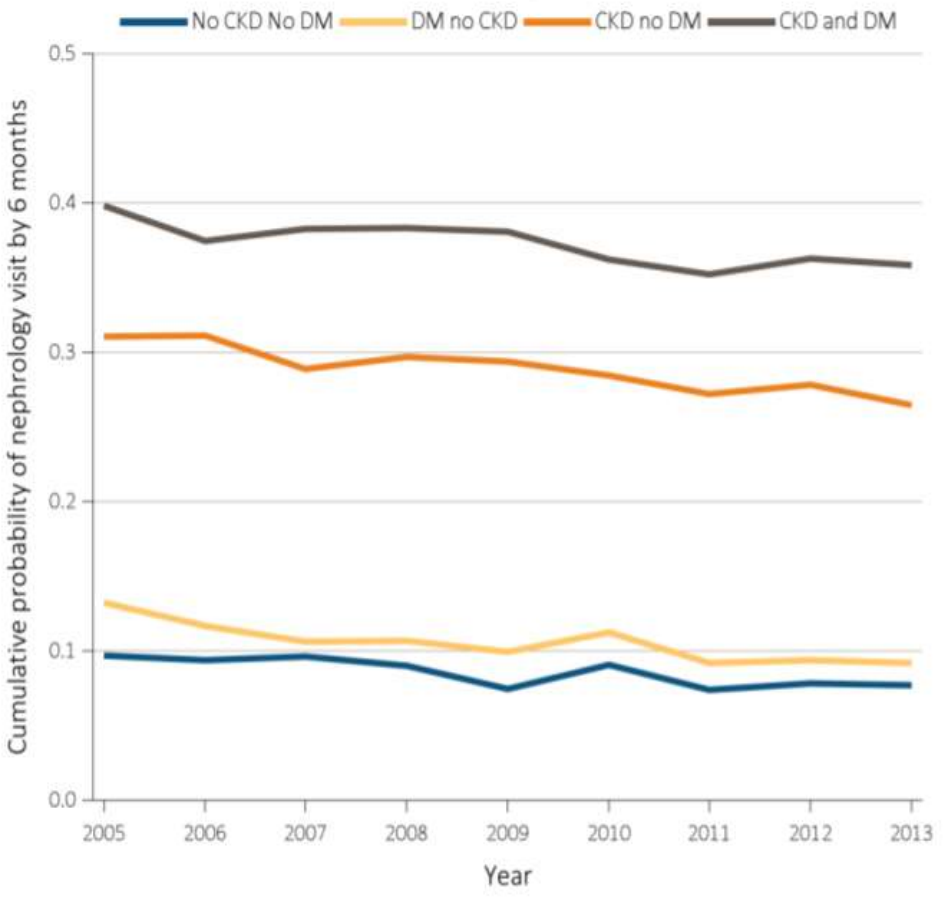
Risk factors for kidney disease progression after an AKI episode

- Old age
- Higher Charlson Co- Morbidity score
- Baseline CKD
- Developing proteinuria
- Diabetes
- Hypertension
- Heart Failure
- Low serum albumin level
- AKI severity and duration
- Discharge Serum Creatinine
- Recurrent AKI episodes

Cumulative probability of a claim for an outpatient nephrology visit within six months of live discharge from first AKI hospitalization, by CKD, DM, 2004-2013 –USRDS 2016



Medicare (66+)



Clinformatics (22+)

Silver *et al. Canadian Journal of Kidney Health and Disease* (2015) 2:36
DOI 10.1186/s40697-015-0071-8

Canadian Society of Nephrology/
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CANADIAN JOURNAL OF
KIDNEY HEALTH AND DISEASE
Journal canadien de la santé et de la maladie rénale

REVIEW Open Access

Ambulatory care after acute kidney injury: an opportunity to improve patient outcomes

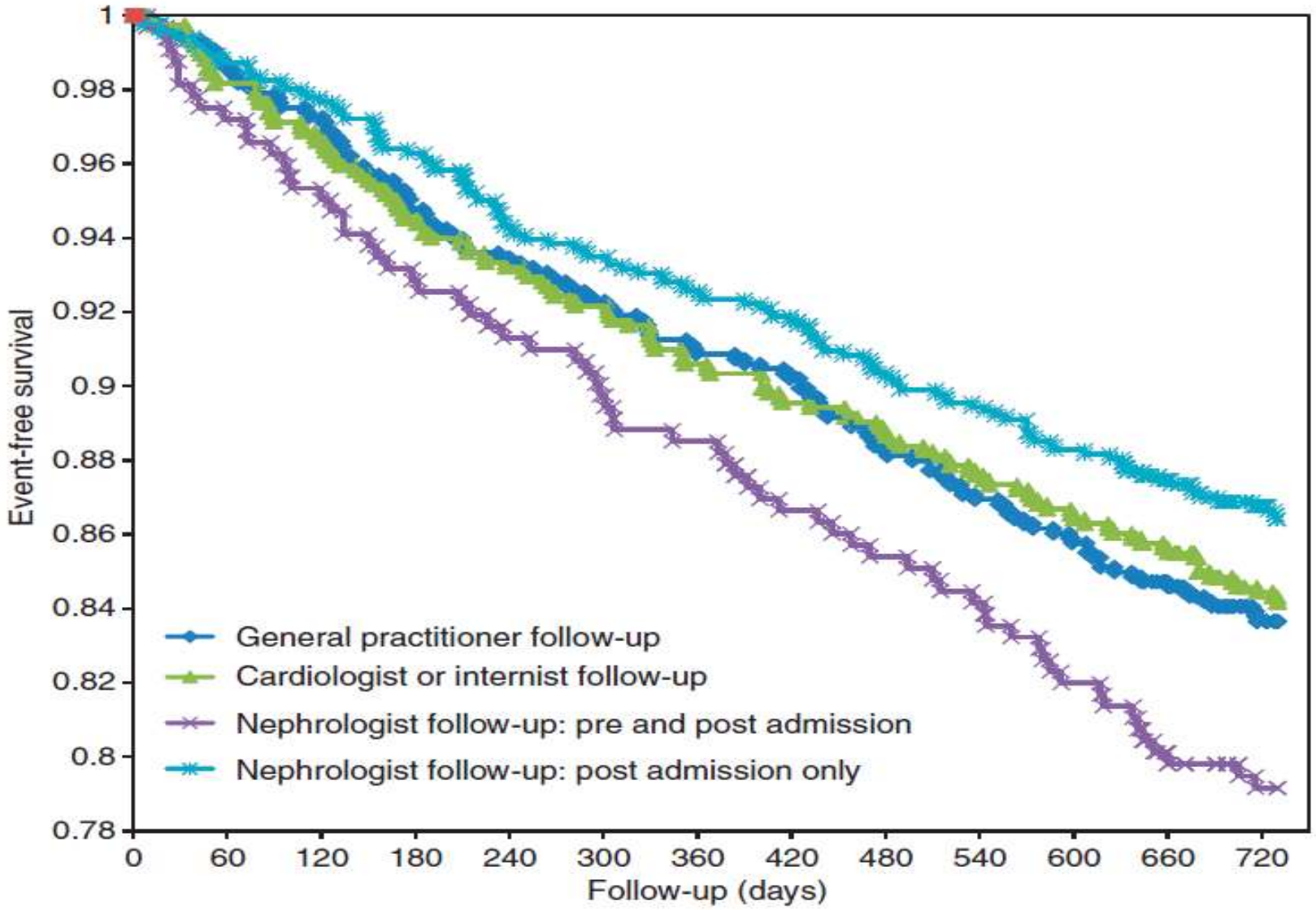
Samuel A. Silver[1,10*], Stuart L. Goldstein[2], Ziv Harel[1,3], Andrea Harvey[1], Elizabeth J. Rompies[2], Neill K. Adhikari[4], Rey Acedillo[5], Arsh K. Jain[5], Robert Richardson[6], Christopher T. Chan[6], Glenn M. Chertow[7], Chaim M. Bell[8,9†] and Ron Wald[1,3†]

Patients who should be prioritized for early outpatient follow-up with a nephrologist after an episode of hospital-acquired AKI

- **Baseline chronic kidney disease**
- **Diabetes mellitus**
- **Congestive heart failure**
- **Patients who suffered from RRT-treated AKI**
- **Hospital discharge serum creatinine > 25% greater than pre-AKI baseline**

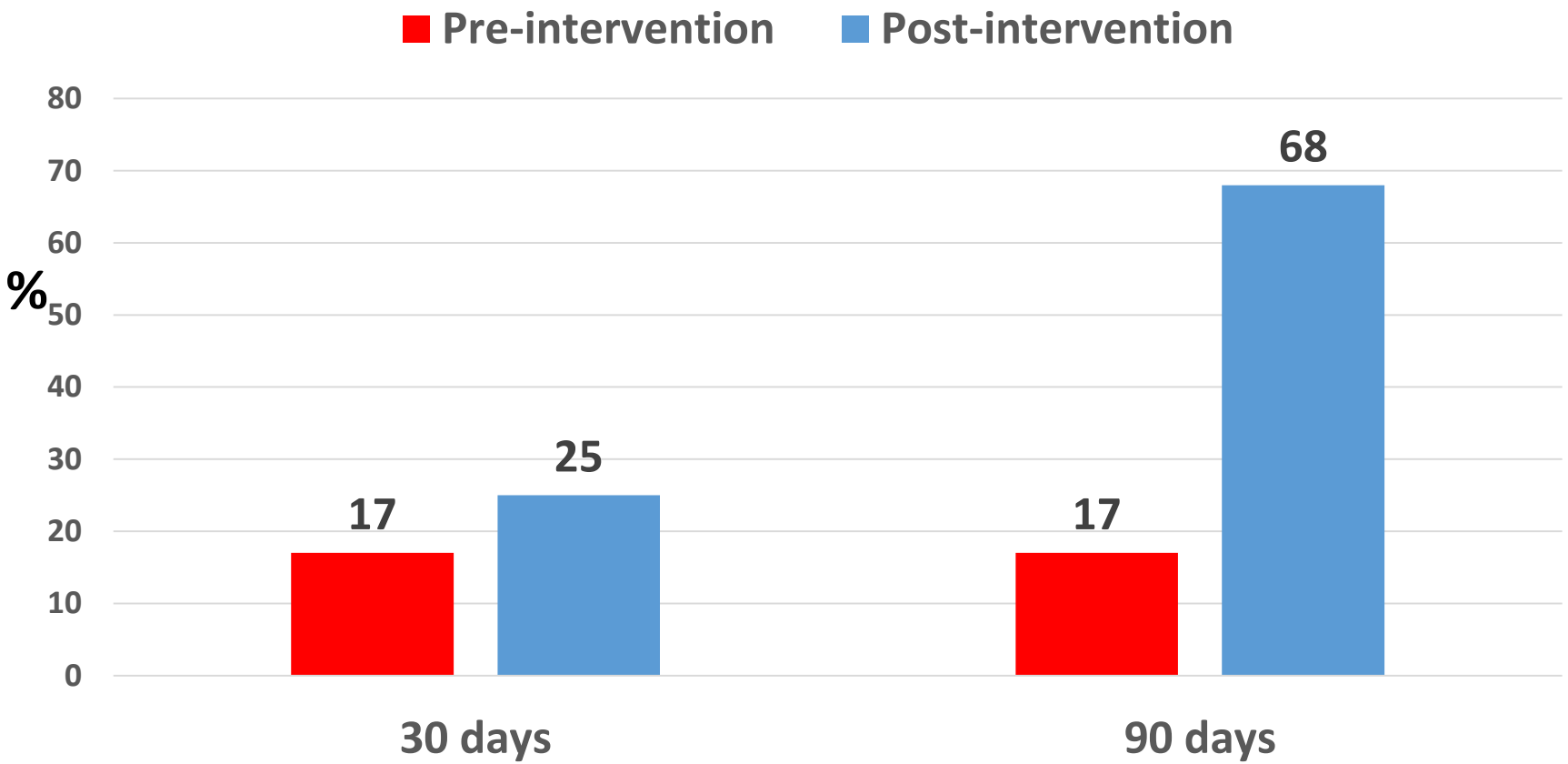
Adapted from Silver, Wald Curr Opin Crit Care 2015, 21:500–505

Survival during FU by specialty of severe AKI adult patients in Ontario



Harel et al, Kidney Intl (2013) 83, 901–908

Improving Care after AKI (stages 2 or 3)
A Prospective Time Series Study in a post AKI Clinic



pre-intervention period: electronic reminders to the nephrology consults and CV surgery services to refer

post-intervention period: patients were automatically scheduled into the post AKI Clinic at discharge

Fifty-five of 79 patients (70%) received at least 1 medical intervention at their first AKI Follow-Up Clinic visit.

Silver et al, Nephron 2015;131:43-50

Points of attention in post AKI patients

- Resume pre-AKI therapies (especially antihypertensives, anticoagulants, antiaggregants) if arrested during course and if not contraindicated
- Treat de novo hypertension
- Treat de novo proteinuria
- Avoid nephrotoxicity
- Arrest therapies (e.g. ACEi) if incipient de novo AKI
- Adapt drug doses to kidney function