Furosemide và AKI

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Giới thiệu

- Tổn thương thận cấp (AKI) thường gặp ở bệnh nhân ICU, góp phần tăng tỉ lệ tử vong
- Non-oliguric AKI có tiên lượng tốt hơn oliguric AKI
- Tích luỹ dịch làm tăng tỉ lệ tử vong ở bệnh nhân ICU

→ sử dụng Furosemide:

- ngăn ngừa AKI tiến triển
- chuyển oliguric AKI thành non-oliguric AKI
- kiểm soát quá tải dịch.

→ **"cải thiện"** tiên lượng của bệnh nhân.

Furosemide

- Là acid yếu
- Thanh thải chủ yếu bởi thận (85%)
- Tác động **ức chế** thụ thể Na-K-Cl2 tranporter ở đoạn dày cành lên quai Henle. → giảm nhu cầu oxy tuỷ thận
- Tăng nồng độ prostaglandin máu → cải thiện tưới máu thận (RBF)

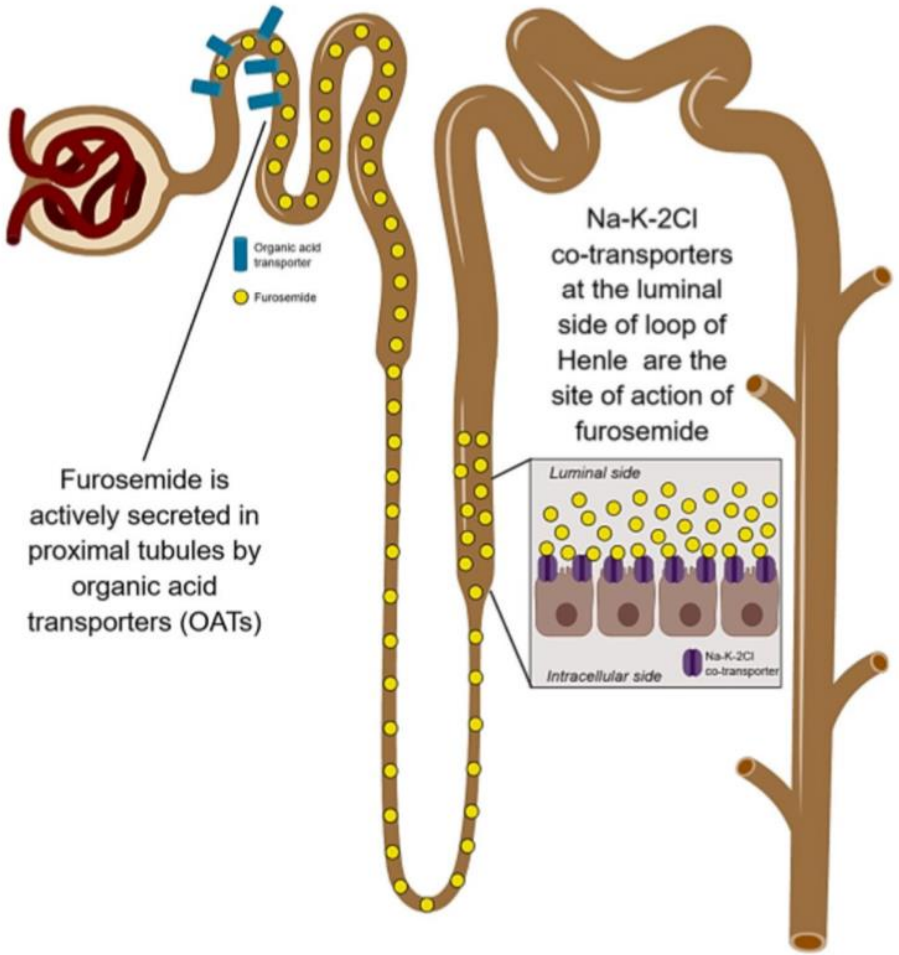

Fig. 1 Visual representation of furosemide secretion and site of action in the nephron

Furosemic có bảo vệ thận????

"Furosemide as a functional marker of acute kidney injury in ICU patients: a new role for an old drug" Filippo Mariano1 · Alberto Mella1 · Marco Vincenti2 · Luigi Biancone1

Giả thuyết furosemide

RESEARCH ARTICLE | *Cardiovascular and Renal Integration*

Furosemide reverses medullary tissue hypoxia in ovine septic acute kidney injury

Naoya Iguchi,[1,2,3] Yugeesh R. Lankadeva,[1] Trevor A. Mori,[6] Eduardo A. Osawa,[2] Salvatore L. Cutuli,[2] Roger G. Evans,[4] Rinaldo Bellomo,[5] and Clive N. May[1]

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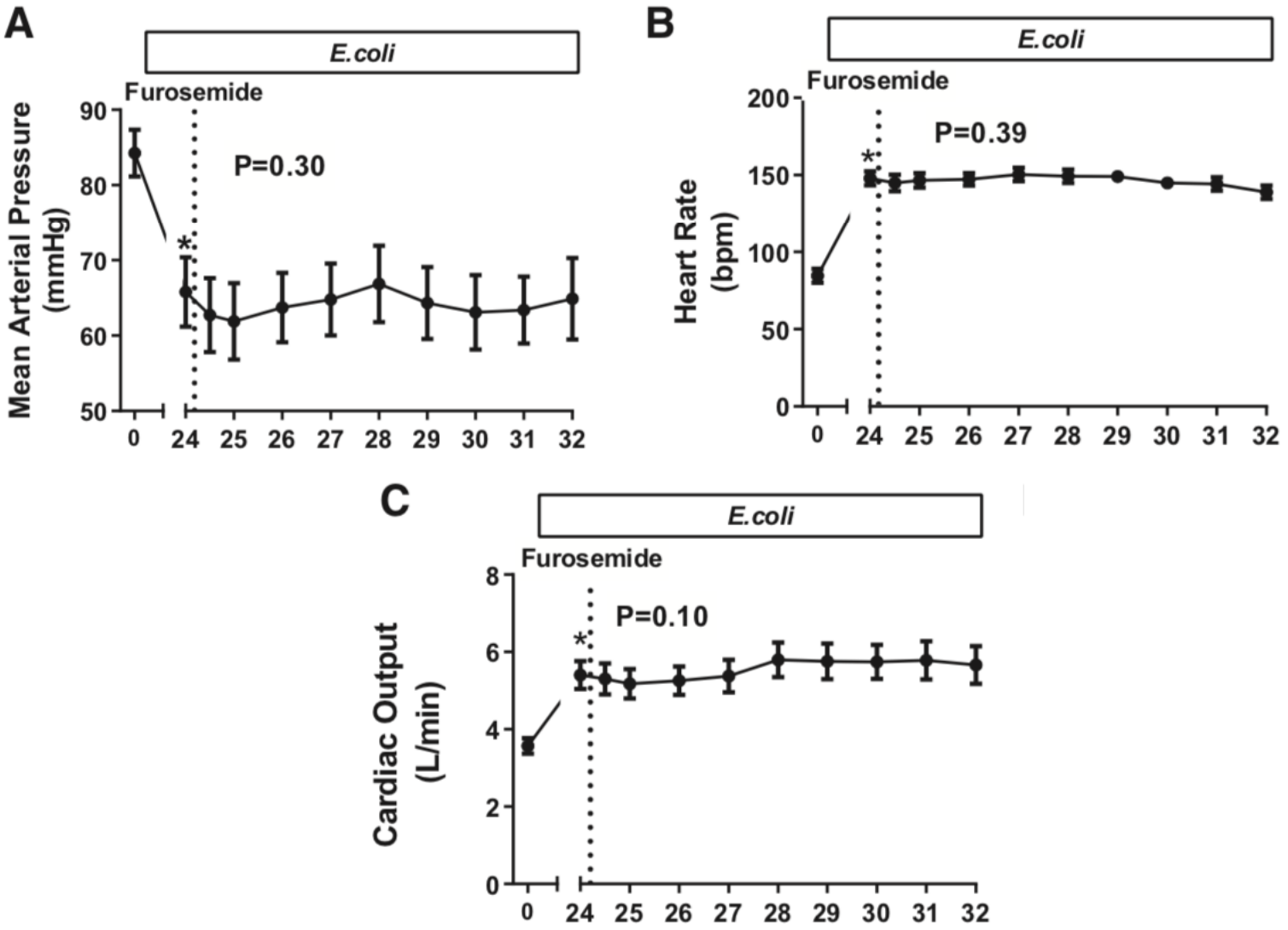
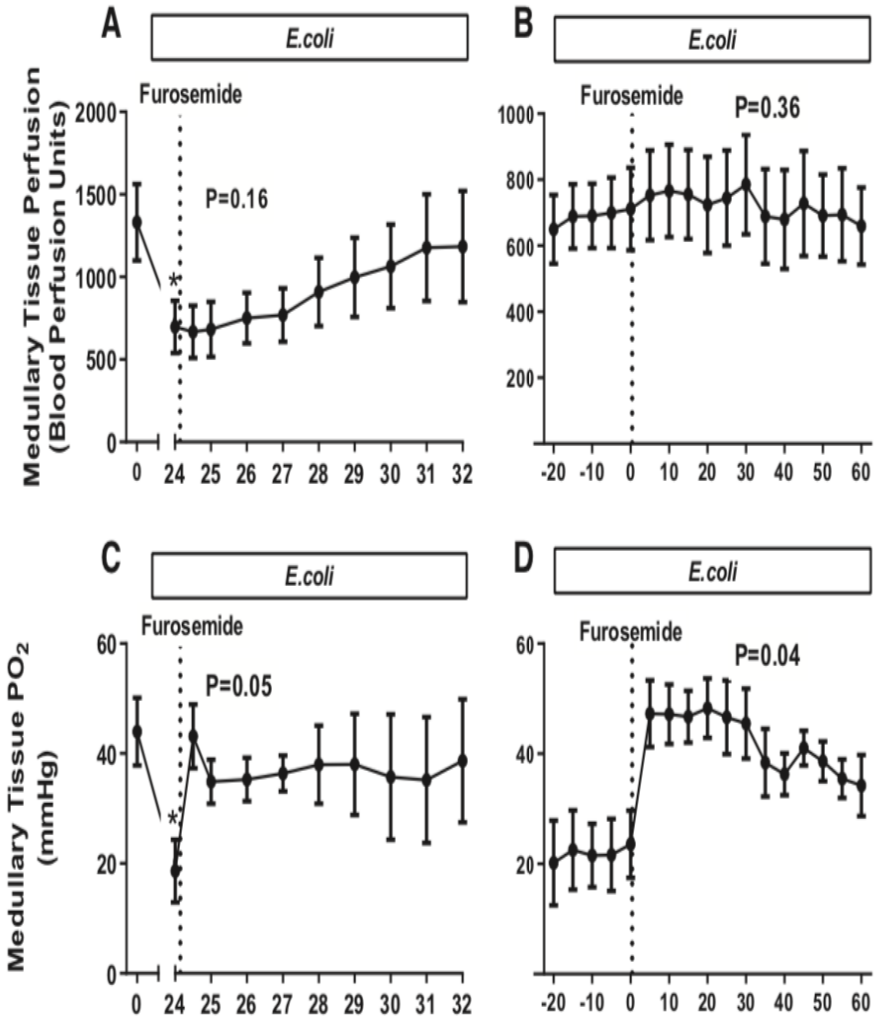
Giả thuyết furosemide

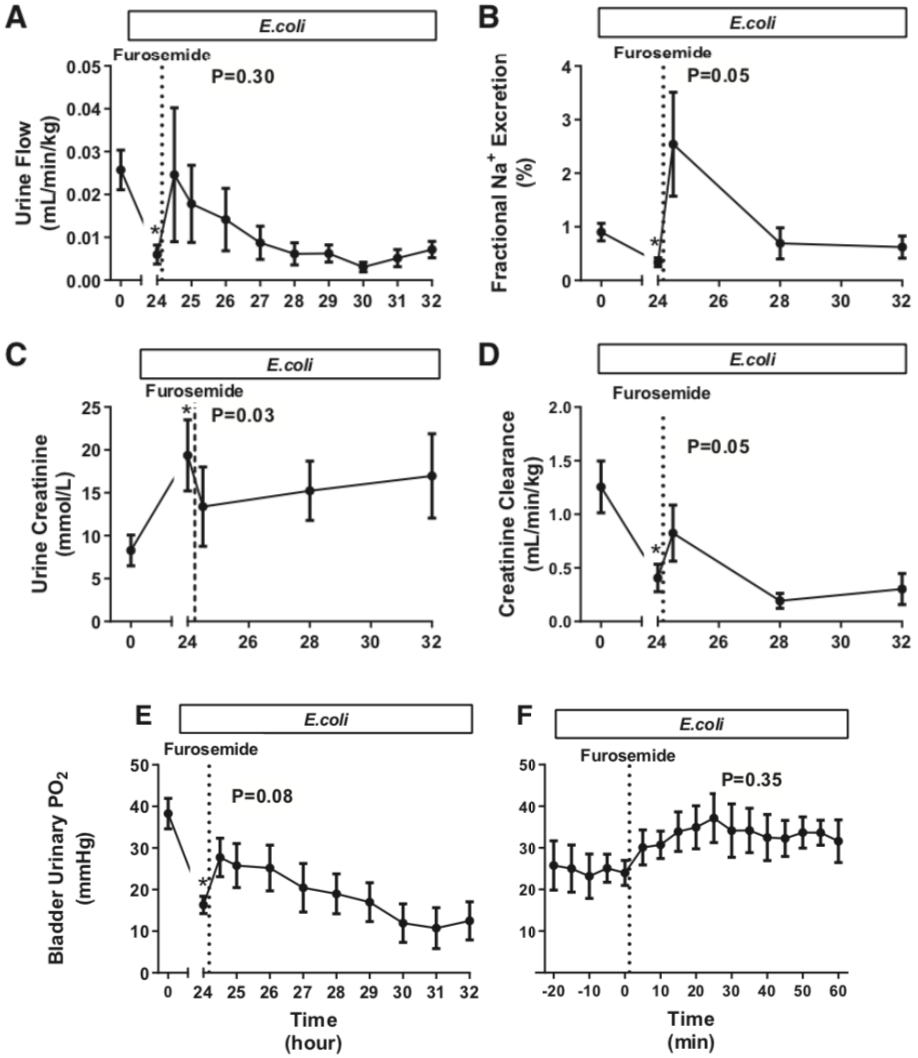
- Thực nghiệm trên 7 con cừu
- Gây nhiễm khuẩn huyết và AKI bằng E.coli
- Duy trì NaCl (0.9% wt/vol) at 1 ml/kg/1h cho đến khi kết thúc can thiệp.
- Sau 24h sepsis, Cừu được bolus tĩnh mạch furosemide (Lasix, 20 mg; Sanofi-Aventis), theo sau là bolus saline (10 ml)

Giả thuyết furosemide



Giả thuyết furosemide





Giả thuyết furosemide

Table 1. *Renal oxygenation and function, blood lactate, and body temperature*

Variables	Baseline	Sepsis, h			
		24	24.5	28	32
Cortical tissue P_{O_2}, mmHg ($n = 6$)	32.6 ± 3.8	48.6 ± 4.8*	51.8 ± 5.4	44.4 ± 8.1	45.5 ± 6.6
Cortical tissue perfusion, BPU ($n = 6$)	1,492 ± 124	1,688 ± 378	1,532 ± 205	1,844 ± 458	1,968 ± 625
Medullary tissue P_{O_2}, mmHg ($n = 6$)	44.0 ± 15.1	18.6 ± 14.0*	43.1 ± 14.2	38.0 ± 17.4	38.7 ± 27.4
Medullary tissue perfusion, BPU ($n = 6$)	1,331.5 ± 569.7	698.0 ± 388.4*	667.4 ± 389.8	909.0 ± 504.6	1,183.4 ± 828.4
Renal O_2 delivery, ml $O_2 \cdot min^{-1} \cdot kg^{-1}$, ($n = 7$)	0.82 ± 0.08	0.94 ± 0.07	0.93 ± 0.09	0.99 ± 0.09	1.07 ± 0.13
Renal O_2 consumption, ml $O_2 \cdot min^{-1} \cdot kg^{-1}$ ($n = 4$)	0.10 ± 0.02	0.07 ± 0.01	0.07 ± 0.01	0.06 ± 0.01	0.07 ± 0.01
Renal O_2 extraction ratio, % ($n = 4$)	12.2 ± 2.8	8.00 ± 1.5	8.0 ± 0.4	6.3 ± 1.1	7.4 ± 1.2
Plasma creatinine, μmol/l ($n = 6$)	86.3 ± 9.4	186.0 ± 40.8*	201.7 ± 39.3	205.3 ± 42.5	218.2 ± 44.0
Urinary sodium excretion, mmol/h ($n = 7$)	5.48 ± 0.84	1.25 ± 0.95*	3.41 ± 2.15	1.06 ± 0.58	0.98 ± 0.55
Blood lactate, mmol/l ($n = 6$)	0.6 ± 0.2	2.5 ± 0.8*	2.0 ± 0.6	4.3 ± 2.7	2.3 ± 1.4
Core temperature, °C ($n = 7$)	40.0 ± 0.2	41.5 ± 0.2*	41.3 ± 0.2	41.2 ± 0.2	40.9 ± 0.3

Values are means ± SE; n, number of sheep. BPU, blood perfusion units; P_{O_2}, oxygen tension. Measurements were made at the end of the baseline period, at 24 h of sepsis, and then after a bolus infusion of furosemide given at 24 h of sepsis in conscious sheep. *$P < 0.05$, significant differences between baseline and 24-h of sepsis from a Student's paired t-test.

Bằng chứng....

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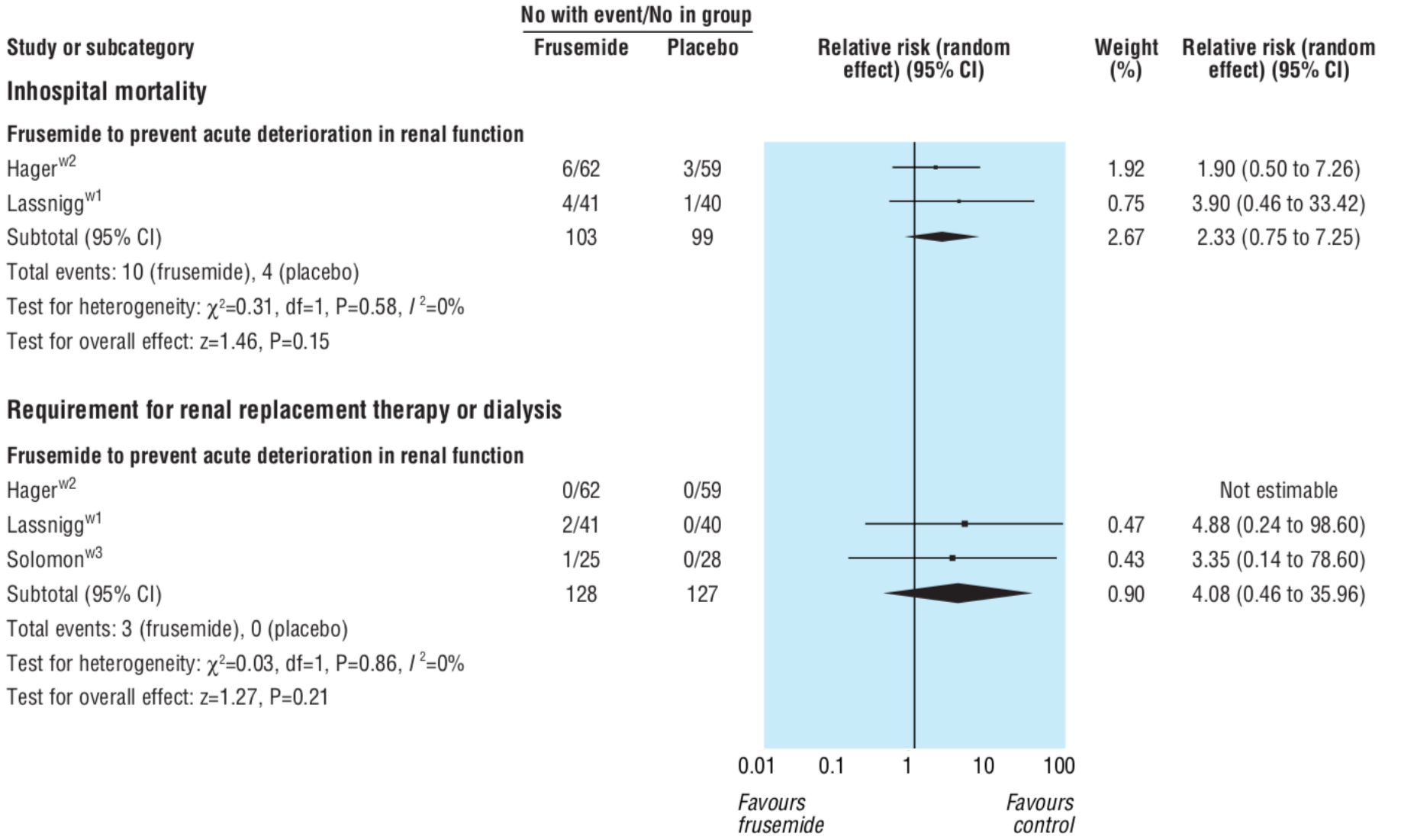
Research

BMJ

Meta-analysis of frusemide to prevent or treat acute renal failure

Kwok M Ho, David J Sheridan

Bằng chứng....


No with event/No in group
Study or subcategory
Frusemide
Placebo
Relative risk (random effect) (95% CI)
Weight (%)
Relative risk (random effect) (95% CI)
Inhospital mortality
Frusemide to prevent acute deterioration in renal function
Hager[w2] 6/62 3/59 1.92 1.90 (0.50 to 7.26)
Lassnigg[w1] 4/41 1/40 0.75 3.90 (0.46 to 33.42)
Subtotal (95% CI) 103 99 2.67 2.33 (0.75 to 7.25)
Total events: 10 (frusemide), 4 (placebo)
Test for heterogeneity: χ²=0.31, df=1, P=0.58, I²=0%
Test for overall effect: z=1.46, P=0.15
Requirement for renal replacement therapy or dialysis
Frusemide to prevent acute deterioration in renal function
Hager[w2] 0/62 0/59 Not estimable
Lassnigg[w1] 2/41 0/40 0.47 4.88 (0.24 to 98.60)
Solomon[w3] 1/25 0/28 0.43 3.35 (0.14 to 78.60)
Subtotal (95% CI) 128 127 0.90 4.08 (0.46 to 35.96)
Total events: 3 (frusemide), 0 (placebo)
Test for heterogeneity: χ²=0.03, df=1, P=0.86, I²=0%
Test for overall effect: z=1.27, P=0.21
0.01
0.1
1
10
100
Favours frusemide
Favours control

Khuyến cáo

Intensive Care Med (2017) 43:730–749
DOI 10.1007/s00134-017-4832-y

CONFERE

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M. Joannidis
H. M. Oudemans-va
mann[6],

CrossMark

Diuretics

Recommendations

1. We *recommend* against loop diuretics given solely for the prevention of acute kidney injury (Grade 1B).
2. We *suggest* using diuretics to control or avoid fluid overload in patients that are diuretic-responsive (Grade 2D).

Shen *et al. Critical Care* (2019) 23:9
https://doi.org/10.1186/s13054-019-2309-9

Critical Care

RESEARCH Open Access

Early diuretic use and mortality in critically ill patients with vasopressor support: a propensity score-matching analysis

Yanfei Shen[1*], Weimin Zhang[2] and Yong Shen[3]

Table 1 Comparisons of the baseline characteristics between patients with and without diuretic use

Variables	All patients ($n = 7828$)	No diuretic use ($n = 6359$)	Diuretic use ($n = 1469$)	P
Age (years)	67.0 ± 14.2	66.5 ± 14.5	70.0 ± 12.3	< 0.001
Male, *n* (%)	4588 (58.6)	3749 (58.9)	839 (57.1)	0.196
Weight (kg)	82.3 ± 22.6	81.7 ± 22.3	84.9 ± 24.0	< 0.001
Emergency, *n* (%)	6072 (77.5)	5075 (79.8)	998 (67.9)	< 0.001
Comorbidities, *n* (%)				
Diabetes mellitus	2390 (30.5)	1850 (29.1)	540 (36.7)	< 0.001
Hypertension	3831 (48.9)	3025 (47.5)	806 (54.8)	< 0.001
Cardiac disease	3431 (43.8)	2603 (41.2)	848 (57.7)	< 0.001
Acute or chronic heart failure	2986 (38.1)	2329 (36.6)	657 (44.7)	< 0.001
Intracranial hemorrhage	273 (3.5)	249 (3.9)	24 (1.6)	< 0.001
Fluid balance				
Fluid intake (ml/kg/48 h)	110.3 ± 73.9	113.8 ± 77.1	95.5 ± 55.6	< 0.001
Urine output (ml/kg/48 h)	49.0 ± 36.6	48.5 ± 38.2	51.2 ± 28.4	0.009
Fluid balance (ml/kg/48 h)	45.1 ± 76.7	49.9 ± 79.7	24.2 ± 57.9	< 0.001
MAP after ICU admission				
MAP on ICU admission (mmHg)	77.4 ± 17.3	77.7 ± 17.6	76.2 ± 15.8	0.002
Mean MAP (mmHg)	74.3 ± 8.1	74.6 ± 8.2	73.0 ± 7.1	< 0.001
Maximum MAP (mmHg)	115.6 ± 40.1	115.9 ± 38.7	114.4 ± 45.5	0.19
Minimum MAP (mmHg)	48.2 ± 14.7	48.3 ± 14.5	47.7 ± 15.3	0.139
Disease severity scores, median (IQR)				
SOFA score on ICU admission	6 (4–8)	6 (4–8)	6 (4–9)	0.0122
Maximum SOFA score during ICU stay	10 (8–13)	10 (8–13)	11 (8–13)	0.2377
SAPS II on ICU admission	41 (32–52)	41 (32–52)	41 (34–51)	0.0151
GCS score on ICU admission	7 (3–14)	8 (3–14)	4 (3–11)	< 0.001
Maximum GCS score during ICU stay	15 (15–15)	15 (15–15)	15 (15–15)	0.001

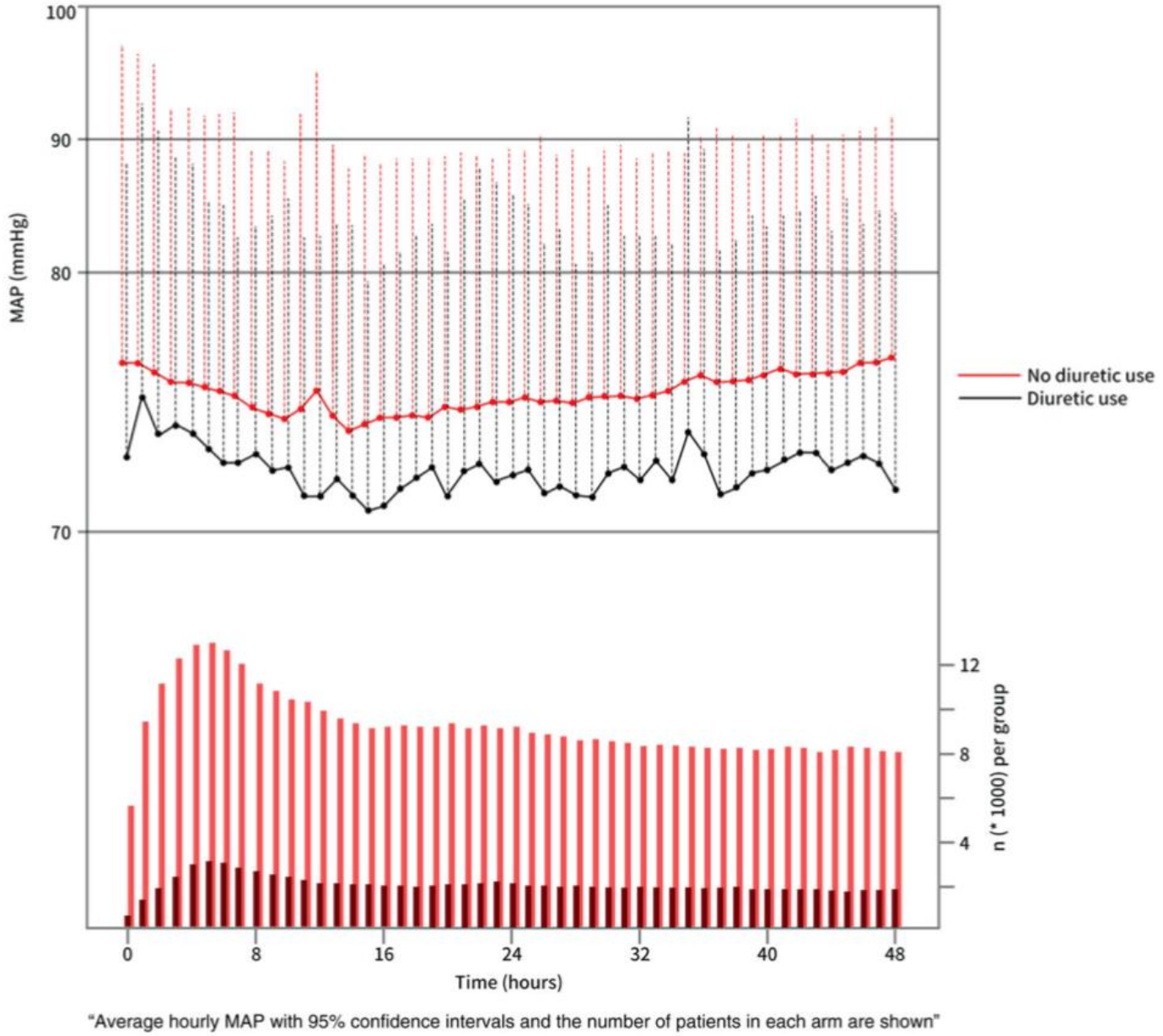
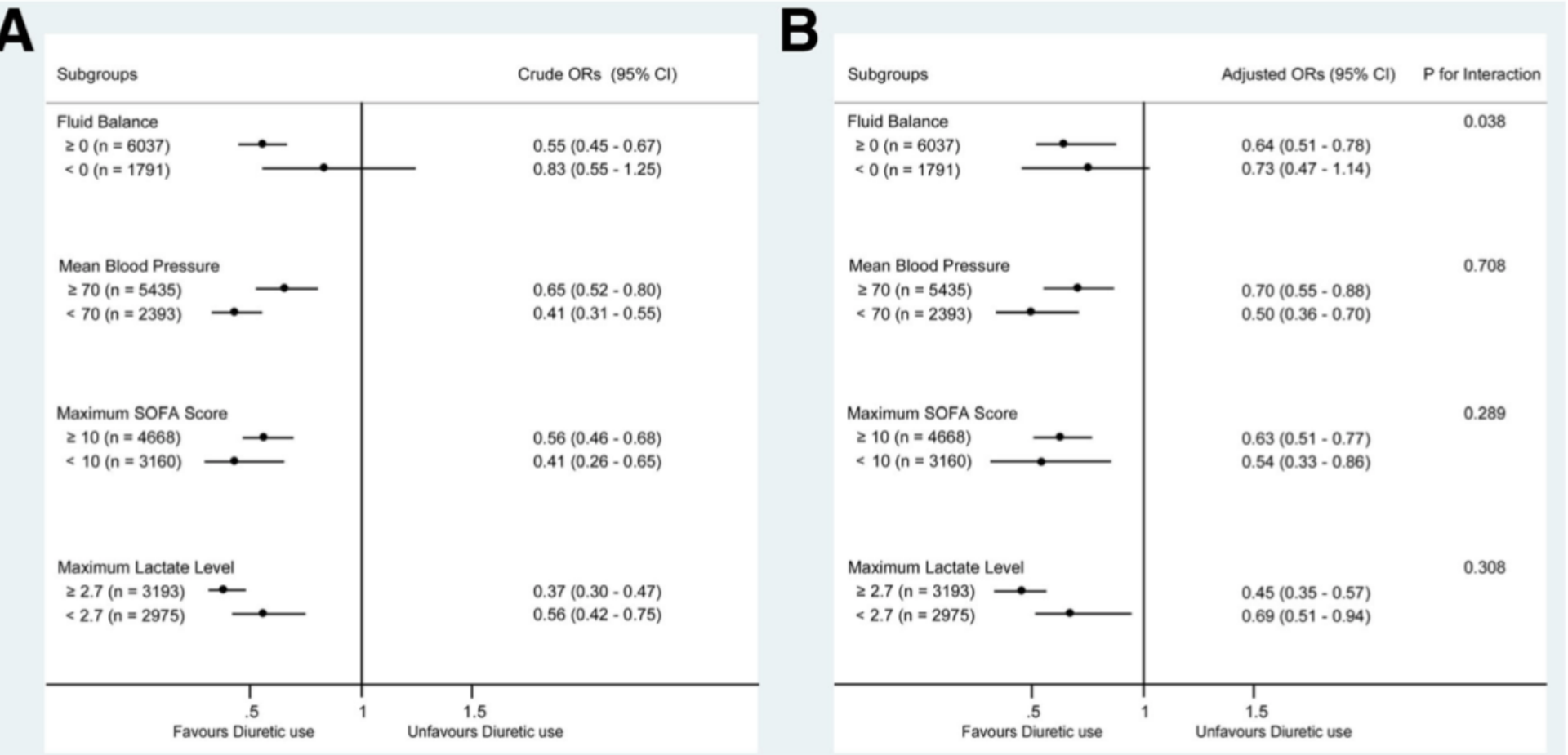


Fig. 2 Trends of the average hourly mean arterial pressure (MAP) in patients with or without diuretic use within 48 h after ICU admission



"CI confidence interval, OR odds ratio, SOFA sequential organ failure assesment"

g. 3 Subgroup analysis of the association between hospital mortality and diuretic use

Furosemide, oliguric AKI và non-oliguric AKI

ORIGINAL CONTRIBUTION

Diuretics, Mortality, and Nonrecovery of Renal Function in Acute Renal Failure

Ravindra L. Mehta, MD
Maria T. Pascual, RN, MPH
Sharon Soroko, MS
Glenn M. Chertow, MD, MPH
for the PICARD Study Group

Context Acute renal failure is associated with high mortality and morbidity. Diuretic agents continue to be used in this setting despite a lack of evidence supporting their benefit.

Objective To determine whether the use of diuretics is associated with adverse or favorable outcomes in critically ill patients with acute renal failure.

Design Cohort study conducted from October 1989 to September 1995.

Table 1. Baseline Patient Characteristics on First Day of Nephrology Consultation*

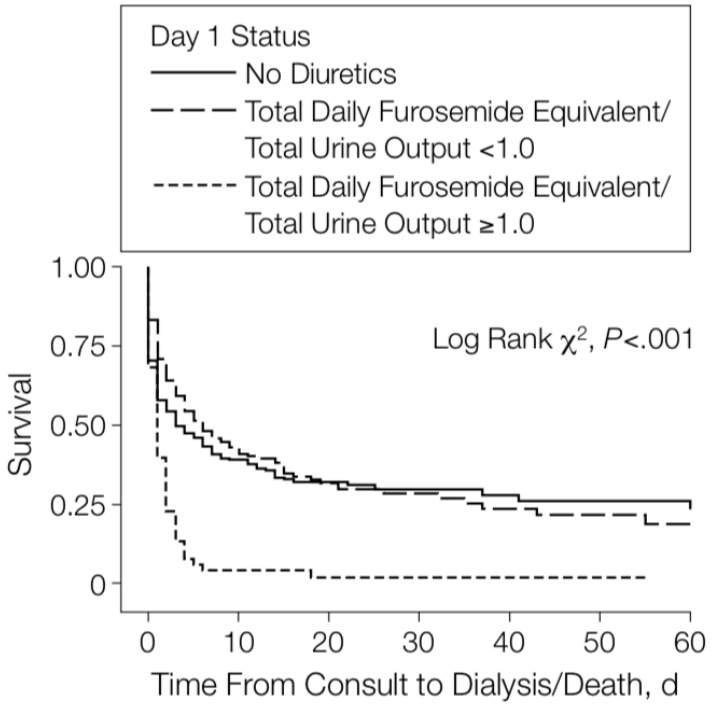
Demographics and History	No Diuretic (n = 226)	Diuretic (n = 326)	*P* Value
Age, mean (SD), y	53.8 (18.0)	58.1 (17.1)	.005†
Male, No. (%)	168 (74)	230 (71)	.33
Race, No. (%)			
White	125 (55)	203 (62)	.12†
African American	50 (22)	46 (14)	
Hispanic	2 (1)	5 (2)	
Asian	21 (9)	37 (11)	
Other or unknown	28 (12)	35 (11)	
Surgical, No. (%)	77 (65)	96 (62)	.28
Oliguria, No. (%)	71 (32)	100 (31)	.75
ARF on CRI, No. (%)	56 (25)	83 (26)	.86
Hyperkalemia, No. (%)‡	17 (8)	29 (9)	.57
History of CHF, No. (%)	30 (13)	87 (27)	<.001†
History of liver disease, No. (%)	49 (22)	54 (17)	.13†
Etiology of acute renal failure, No. (%)			
Ischemic	98 (43)	128 (40)	.34
Nephrotoxic	28 (12)	61 (19)	.05†
Multifactorial	43 (19)	49 (15)	.22†
Unknown	57 (25)	88 (27)	.64
Renal function			
Mean (SD) BUN, mg/dL	72.3 (43.4)	61.6 (34.6)	.001†
Mean (SD) creatinine, mg/dL	4.1 (3.3)	3.6 (1.9)	.02†
Median urine output, mL/d	955	888	.49
Physiologic indicators			
Temperature, mean (SD), °C	37 (1.2)	37 (1.1)	.63
Heart rate, mean (SD), beats/min	102 (24)	100 (22)	.24†
Systolic blood pressure, mean (SD), mm Hg	122 (33)	117 (29)	.07†
Diastolic blood pressure, mean (SD), mm Hg	61 (17)	59 (17)	.30
Arterial pressure, mean (SD), mm Hg	81 (21)	78 (20)	.19†
Central venous pressure, mean (SD), mm Hg§	15 (7)	15 (6)	.77
Pulmonary artery wedge pressure, mean (SD), mm Hg§	18 (8)	20 (7)	.04
Cardiac output, mean (SD), L/min§	8.5 (3.9)	6.9 (3.1)	<.001
Cardiac index, mean (SD), L/min/m^2§	4.6 (2.0)	3.7 (1.6)	<.001
Systemic vascular resistance, mean (SD), dynes·s·cm^{-5}§	728 (429)	903 (811)	.02
Po_2, mean (SD), mm Hg§	102 (48)	98 (49)	.43
Pco_2, mean (SD), mm Hg§	35 (9)	37 (9)	.11
pH, mean (SD)§	7.3 (0.1)	7.4 (0.1)	.21
APACHE III score, mean (SD)§	86.7 (32.9)	86.1 (30.5)	.84
APACHE II score, mean (SD)§	19.0 (7.8)	18.8 (7.4)	.54
Organ system failure, No. (%)			
Respiratory	143 (64)	241 (74)	.01†
Cardiac	75 (33)	148 (45)	.005†
Liver	75 (33)	109 (33)	.98
Hematologic	73 (32)	92 (28)	.29
Central nervous system	82 (36)	112 (34)	.61

Table 2. Effect of Diuretics on Mortality and Nonrecovery of Renal Function Compared With No Diuretic Use*

Variable	OR (95% CI)		
	Unadjusted	Covariate Adjusted	Covariate and Propensity Score Adjusted
In-hospital mortality	1.37 (0.97-1.92)	1.65 (1.05-2.58)	1.68 (1.06-2.64)
Nonrecovery of renal function	1.53 (1.08-2.15)	1.70 (1.14-2.53)†	1.79 (1.19-2.68)§
Death or nonrecovery	1.48 (1.02-2.12)	1.74 (1.12-2.68)‡	1.77 (1.14-2.76)‖

*Covariate adjusted for age; sex; log urine output; serum creatinine level; blood urea nitrogen level; respiratory, hepatic, and hematologic failure; and heart rate. The referent group was no diuretics; time was first day of intensive care unit consultation. OR indicates odds ratio; CI, confidence interval.
†Area under receiver operating characteristic (ROC) curve = 0.76; goodness-of-fit χ^2 P = .89.
‡Area under ROC curve = 0.82; goodness-of-fit χ^2 P = .39.
§Area under ROC curve = 0.85; goodness-of-fit χ^2 P = .84.
‖Area under ROC curve = 0.81; goodness-of-fit χ^2 P = .58.

Figure 2. Time to Death or Dialysis From Day of Consultation in Intensive Care Unit



No. at Risk						
No Diuretics	170	63	31	18	14	10
Total Daily Furosemide Equivalent/Total Urine Output						
<1.0	188	73	28	21	12	9
≥1.0	53	2	1	1	1	1

Loop diuretics in the management of acute renal failure: a systematic review and meta-analysis

Sean M Bagshaw, Anthony Delaney, Michael Haase, William A Ghali and Rinaldo Bellomo

Results: Of 62 studies reviewed, five RCTs, enrolling 555 patients, were eligible and analysed. These trials enrolled a mix of patients, but only two included critically ill patients. Overall trial quality was low. There was no statistical difference in mortality (odds ratio [OR], 1.28; 95% CI, 0.89–1.84; $P = 0.18$) or renal recovery (OR, 0.88; 95% CI, 0.59–1.31; $P = 0.5$) with use of loop diuretics compared with control. However, loop diuretics were associated with a shorter duration of RRT (weighted mean difference, –1.4 days; 95% CI, –0.2 to –2.3 days; $P = 0.02$), shorter time to spontaneous decline in serum creatinine level (weighted mean difference, –2.1 days; 95% CI, –0.4 to –3.7 days; $P = 0.01$) and a greater increase in urine output from baseline (OR, 2.6; 95% CI, 1.4–4.9; $P = 0.004$). Insufficient data were available on acid–base status, hospital length of stay or health costs. Four studies reported toxicity, most commonly transient tinnitus and deafness.

SCIENTIFIC REPORTS

nature research

OPEN

Optimal timing of initiating continuous renal replacement therapy in septic shock patients with acute kidney injury

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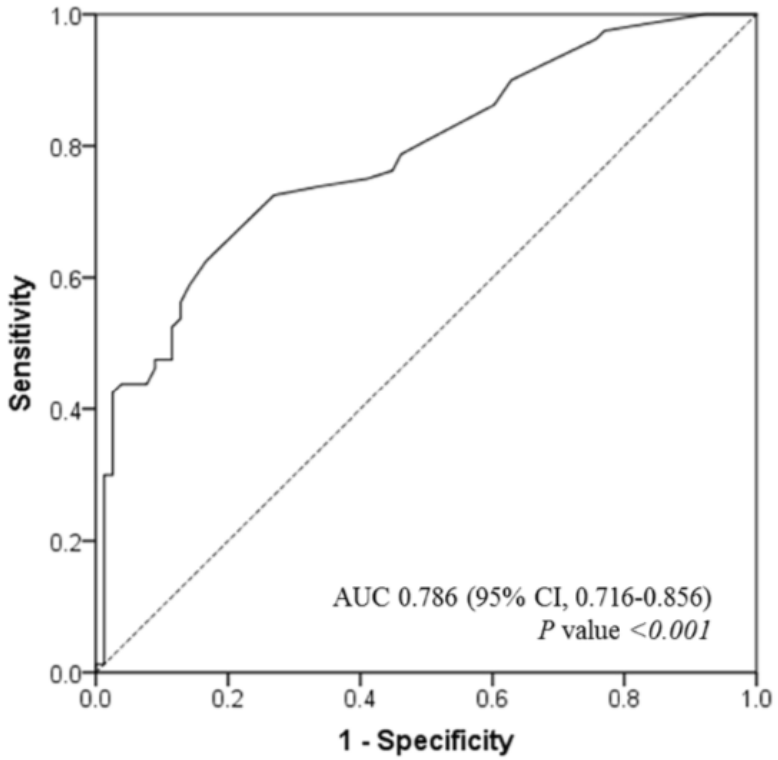
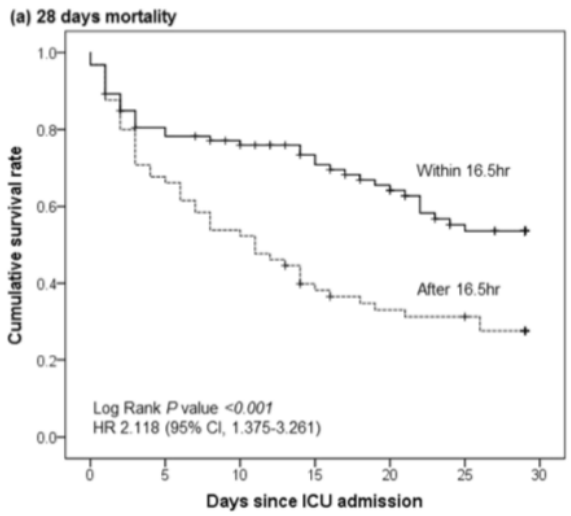
Bo Ra Yoon, Ah Young Leem, Moo Suk Park, Young Sam Kim & Kyung Soo Chung

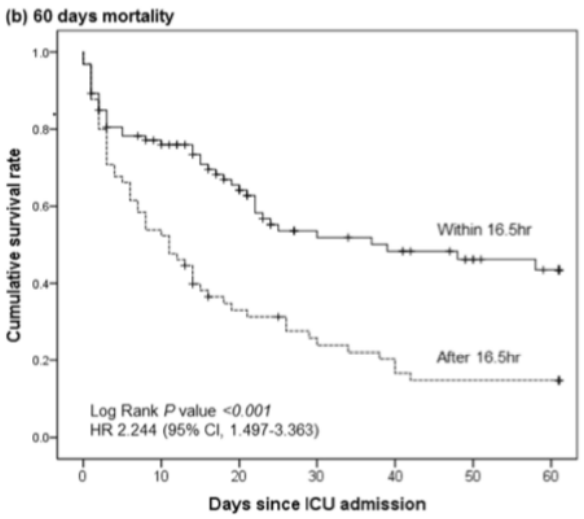
	Survivors n = 78 (49.4%)	Non-survivors n = 80 (50.6%)	*P* value
At acute kidney injury occurrence			
Lactate (mmol/L)	4.9 (2.8, 9.8)	7.0 (3.7, 12.6)	*0.113*
BUN (mg/dL)	50.0 (30.5, 70.6)	40.5 (26.2, 57.5)	*0.047*
Creatinine (mg/dL)	2.54 (1.54, 3.66)	1.88 (1.27, 2.88)	*0.003*
Potassium (mmol/L)	4.7 (1.0, 5.8)	4.4 (3.7, 5.1)	*0.024*
Urine output (ml/day)	1,493 (505, 2,140)	1,114 (467, 2,487)	*0.698*
pH	7.295 (7.236, 7.389)	7.299 (7.209, 7.395)	*0.963*
GFR (ml/min/1.73 $m^{2¥}$)	24 (16, 39)	31 (19, 53)	*0.020*
GFR (ml/min/1.73 $m^{2€}$)	23 (15, 42)	32 (18, 57)	*0.015*
At ICU admission			
Lactate (mmol/L)	3.4 (2.2, 5.6)	9.7 (4.4, 15.3)	*<0.001*
BUN (mg/dL)	44.0 (31.2, 65.7)	44.5 (31.9, 63.7)	*0.836*
Creatinine (mg/dL)	2.44 (1.45, 3.34)	2.34 (1.55, 3.07)	*0.766*
Potassium (mmol/L)	4.3 (3.6, 5.1)	4.3 (3.7, 5.0)	*1.000*
Urine output (ml/day)	585 (196, 1,381)	520 (188, 1,070)	*0.321*
pH	7.379 (7.306, 7.430)	7.300 (7.251, 7.374)	*<0.001*
At CRRT initiation			
Lactate (mmol/L)	4.3 (2.6, 8.9)	7.5 (4.2, 15.5)	*0.001*
NGAL (ng/mL)	1,004 (465, 1,695)	820 (367, 2,083)	*0.488*
Cystatin C (mg/L)	2.76 (1.87, 3.89)	2.89 (2.04, 3.93)	*0.640*
GFR (ml/min/1.73 $m^{2≠}$)	14 (8, 28)	14 (8, 25)	*0.829*
Interval time from AKI to CRRT initiation (hours)	9 (6, 14)	26 (11, 66)	*<0.001*
CRRT duration (hours)	78 (52, 146)	67 (31, 212)	*0.607*

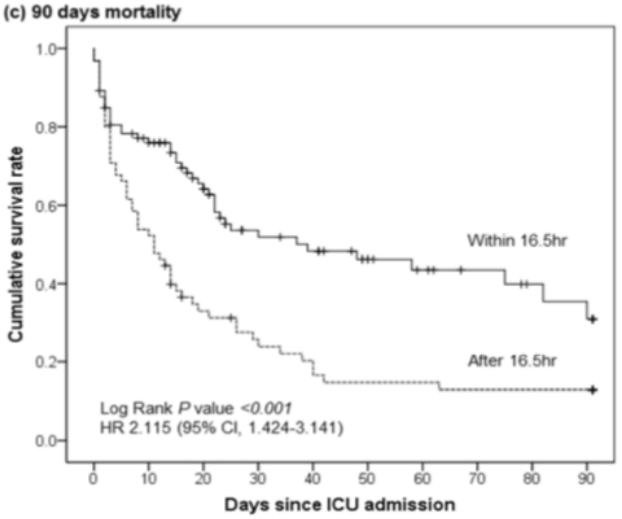


** Cutoff 16.5 hours (Sensitivity 0.638, Specificity 0.821)

Figure 1. AUROC curve of optimal CRRT initiation time for ICU mortality. The cut-off value of the optimal interval time for ICU mortality.







(d) Cumulative survivors

	Number of survivors															Survival rate	Mortality rate
	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28		
Within	93	79	75	73	72	71	71	69	66	64	62	58	56	55	55	59.1%	40.7%
After	65	52	44	40	35	34	30	26	24	23	22	21	21	19	19	29.2%	70.8%
	158	131	119	113	107	105	101	95	90	87	84	79	77	74	74	46.8%	53.2%

Figure 3. 28 days-, 60 days- and 90 days- overall mortality of septic shock patients who initiated CRRT within and after 16.5 hours. The comparison of overall mortalities at 28, 60, and 90 days for early and late CRRT initiation groups. Within: time interval from AKI to CRRT initiation <16.5 hours; After: time interval from AKI to CRRT initiation ≥ 16.5 hours.

Furosemide ở bệnh nhân AKI

- Sử dụng furosemide chỉ làm tăng thể tích nước tiểu, không cải thiện được tỉ lệ tử vong, tỉ lệ RRT, tỉ lệ phục hồi chức năng thận
- Việc trì hoãn CRRT có thể làm tăng tỉ lệ tử vong

Treat AKI not treat urine output

Furosemide giúp ngưng RRT

- Ở những bệnh nhân AKI được sử dụng RRT, việc tăng thể tích nước tiểu thường là một trong những triệu chứng giúp cân nhắc ngưng RRT.
- Việc sử dụng lợi tiểu, để tăng thể tích nước tiểu thường dùng cho mục đích này.

Furosemide does not improve renal recovery after hemofiltration for acute renal failure in critically ill patients: A double blind randomized controlled trial*

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Objective: To study the potential beneficial role of furosemide in resolving renal failure after hemofiltration in mechanically ventilated critically ill patients.

Design: Single-center randomized, double blind, placebo-controlled study.

Setting: A 13-bed mixed intensive care unit (ICU) in a teaching hospital.

Patients: Patients who had been treated with continuous venovenous hemofiltration were included.

Interventions: After the end of continuous venovenous hemofiltration, the urine of the first 4 hours was collected for measuring creatinine clearance. Patients were subsequently randomized for furosemide (0.5 mg/kg/hr) or placebo by continuous infusion. To prevent hypovolemia, the rate of fluid infusion was adapted every hour and was set as the urinary production of the previous hour.

Measurements and Main Results: End points were renal recovery (creatinine clearance more than 30 mL/min or stable serum creatinine without renal replacement therapy) in the ICU and in the hospital. Seventy-two patients were included and 71 were eligible for the analysis. The 36 furosemide-treated patients had a significantly increased urinary volume compared with the 35 placebo-treated patients (median 247 mL/hr (interquartile range [IQR] 774 mL/hr) vs. 117 mL/hr (IQR 158 mL/hr), $p = 0.003$) and greater sodium excretion (median 73 mmol/L (IQR 48) vs. 37 (IQR 48) mmol/L, $p = 0.001$). In the furosemide group 25 patients and in the placebo group 27 patients showed recovery of renal function at ICU discharge ($p = 0.46$). Two patients of the furosemide group needed long-term dialysis dependency ($p = 0.23$).

Conclusion: Furosemide by continuous infusion in the recovery phase of hemofiltration-dependent acute kidney failure did increase urinary volume and sodium excretion but did not lead to a shorter duration of renal failure or more frequent renal recovery. (Crit Care Med 2009; 37:533–538)

KEY WORDS: furosemide; acute renal failure; hemofiltration; recovery; critically ill; mechanical ventilation

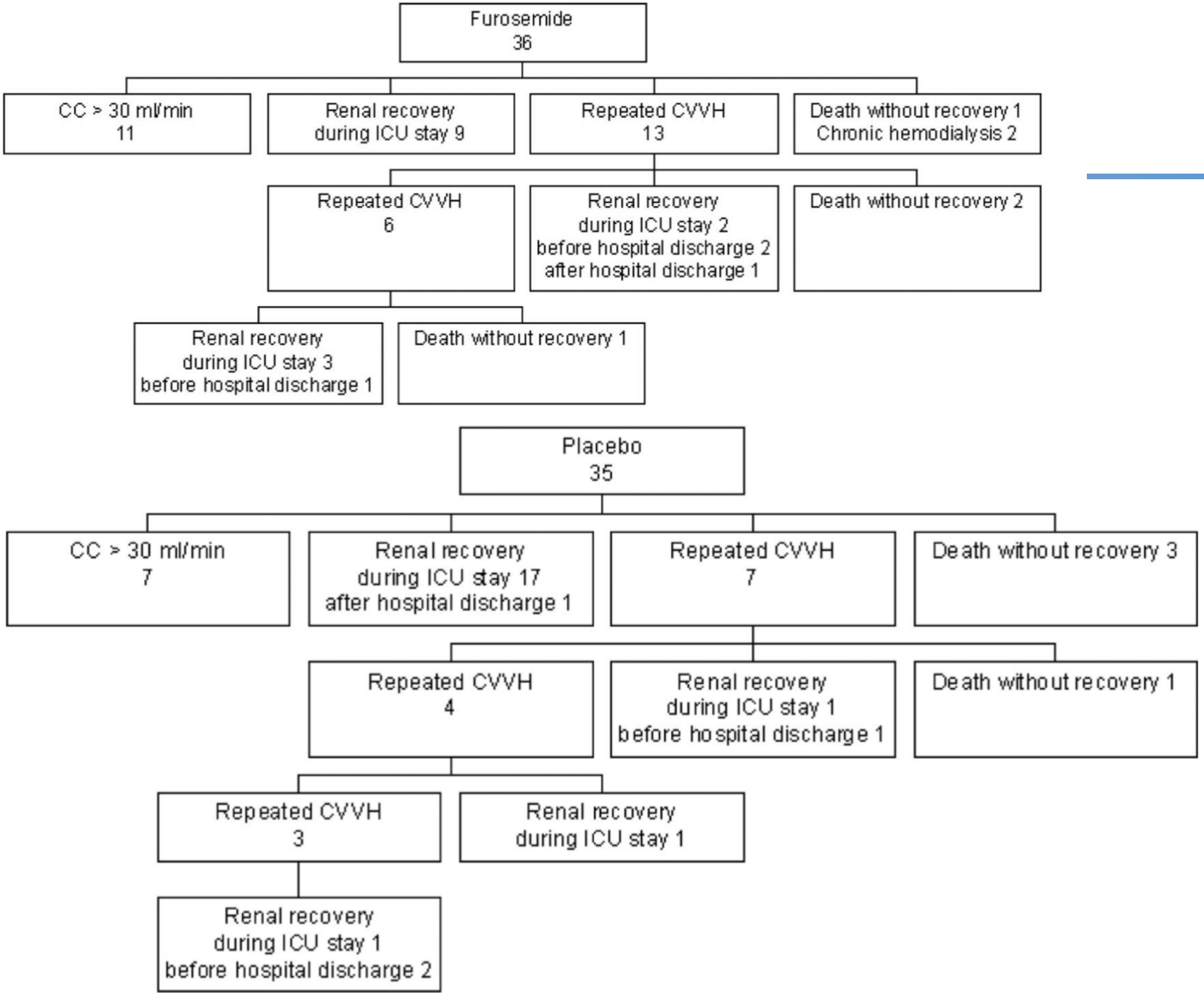
Furosemide
36
CC > 30 ml/min
11
Renal recovery
during ICU stay 9
Repeated CVVH
13
Death without recovery 1
Chronic hemodialysis 2
Repeated CVVH
6
Renal recovery
during ICU stay 2
before hospital discharge 2
after hospital discharge 1
Death without recovery 2
Renal recovery
during ICU stay 3
before hospital discharge 1
Death without recovery 1
Placebo
35
CC > 30 ml/min
7
Renal recovery
during ICU stay 17
after hospital discharge 1
Repeated CVVH
7
Death without recovery 3
Repeated CVVH
4
Renal recovery
during ICU stay 1
before hospital discharge 1
Death without recovery 1
Repeated CVVH
3
Renal recovery
during ICU stay 1
Renal recovery
during ICU stay 1
before hospital discharge 2

Table 3. Main results

	Furosemide	Placebo	*p*
Fluid balance over study episode (mL)	−169 (958)	−75 (871)	0.25
Sodium excretion (mmol/L)	72.9 (48.4)	37.5 (48.5)	0.001
Serum chloride (mmol/L)	105 (4)	105 (4)	0.91
Number of days on CVVH	8.2 (12)	7.0 (10)	0.74
Creatinine Clearance (mL/min)	15.4 (26.2)	16.1 (18.3)	0.88
Renal recovery on ICU discharge			
All patients (N)	25/36	27/35	0.46
In ICU survivors (N)	23/31	24/29	0.42
Renal recovery on hospital discharge			
All ICU survivors (N)	26/31	27/29	0.43
In-hospital survivors (N)	19/23	23/24	0.14
Renal recovery 2 months after hospital discharge (N)	21/23	24/24	0.23
Duration of mechanical ventilation in days	20 (16)	16 (16)	0.13
ICU length of stay in days	24 (18)	20 (24)	0.28
ICU mortality (%)	14	18	0.7
Hospital mortality (%)	36	32	0.8

Furosemide - Albumin

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Renal/Metabolic

Co-administration of furosemide with albumin for overcoming diuretic resistance in patients with hypoalbuminemia: A meta-analysis ☆

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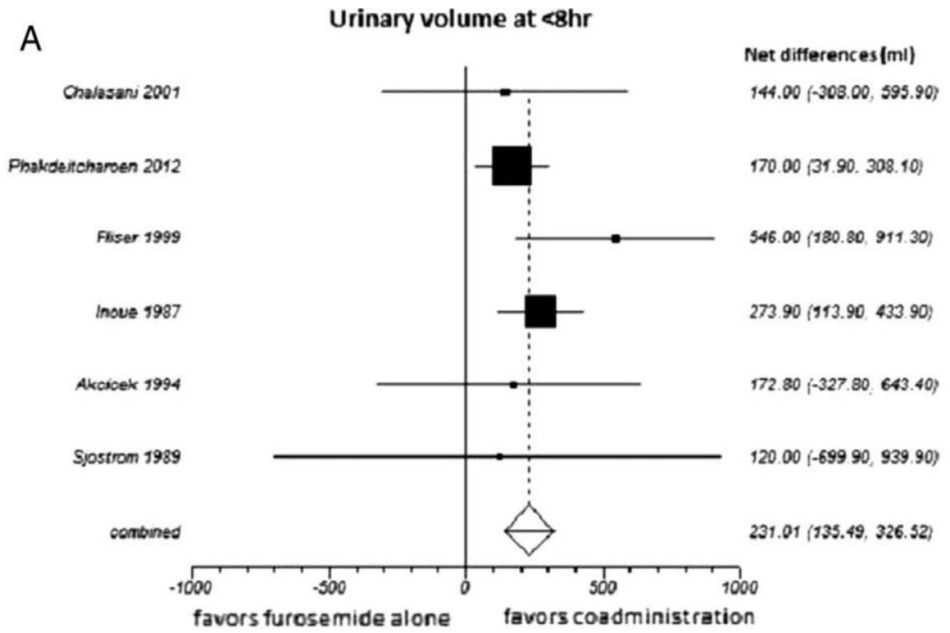
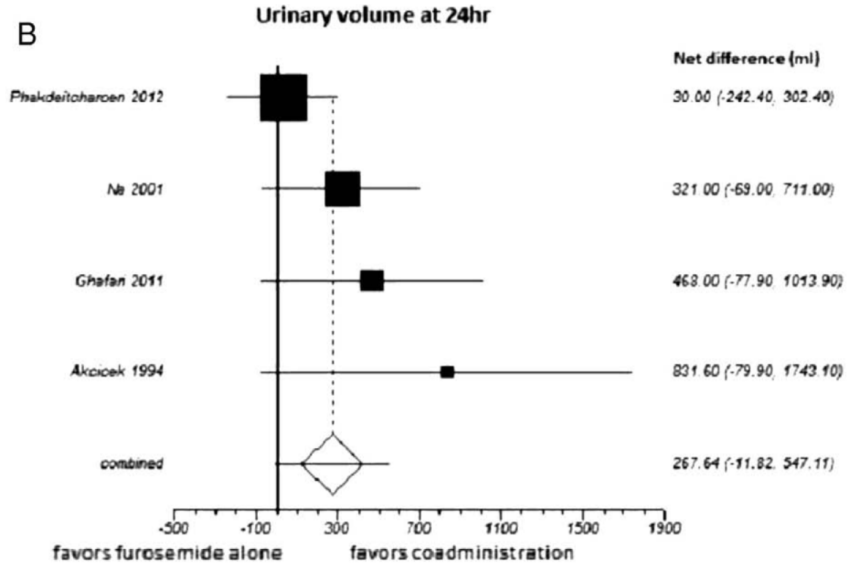
Furosemide - Albumin

Table 2
Meta-analysis results for the outcomes of urinary volume and urinary Na excretion at 8 and 24 hours for the main analysis and the nephrotic syndrome patients subgroup

Outcome	Main analysis		Nephrotic syndrome subgroup	
	Summary Difference (95% CI), N of studies	Heterogeneity (I^2, p_Q)	Summary Difference (95% CI), N of studies	Heterogeneity (I^2, p_Q)
Urinary volume <8 h (mL)	**231.0** (**135.5-326.5**), **n = 6**	$I^2 = 0$, $P = .53$	**378.4** (**103.4-653.4**), **n = 3**	$I^2 = 0$, $P = .39$
Urinary volume 24 h (mL)	267.6 (-11.8-547.1), n = 4	$I^2 = 365$, $P = .21$	**420.5** (**120.8-720.2**), **n = 3**	$I^2 = 0$, $P = .58$
Urinary Na <8 h (mEq)	**15.9** (**4.9-26.8**), **n = 5**	$I^2 = 0$, $P = .66$	24.2 (-13.4-61.8), n = 3	$I^2 = 4$, $P = .35$
Urinary Na 24 h (mEq)	23.4 (-13.0-59.9), n = 4	$I^2 = 84$, $P = .003$	34.9 (-3.9-73.9), n = 3	$I^2 = 410$, $P = .18$

Statistically significant results are shown in bold





Furosemide liên tục hay ngắt quãng

Anaesthesia
Peri-operative medicine, critical care and pain
Association of Anaesthetists

Review Article | Free Access

Continuous infusion vs. intermittent bolus injection of furosemide in acute decompensated heart failure: systematic review and meta-analysis of randomised controlled trials

K. T. Ng, J. L. L. Yap

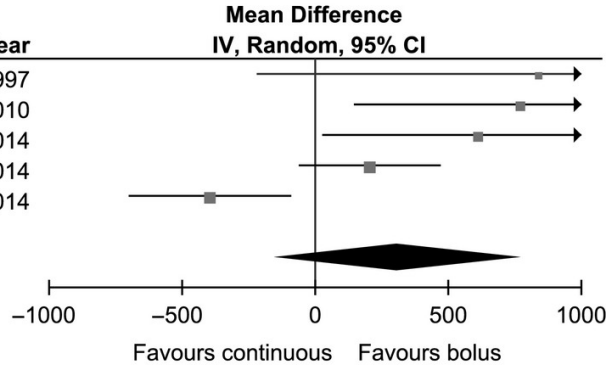
First published: 22 September 2017 | https://doi.org/10.1111/anae.14038 | Cited by: 5

Furosemide liên tục hay ngắt quãng

(a)

Study or Subgroup	Continuous Mean	SD	Total	Bolus Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
Makhoul 1997	3,672.5	1,353.7	10	2,833	1,042.7	10	11.4%	839.50 [–219.56, 1898.56]	1997
Thomson 2010	3,726	1,121	26	2,955	1,267	30	18.6%	771.00 [145.52, 1396.48]	2010
Llorens 2014	3,705	1,340	36	3,093	1,208	37	19.4%	612.00 [26.25, 1197.75]	2014
Palazzuoli 2014	2,295	775	43	2,090	421	39	25.6%	205.00 [–61.68, 471.68]	2014
Shah 2014	721.57	447.99	30	1,117.15	726.7	30	25.0%	–395.58 [–701.06, –90.10]	2014
Total (95% CI)			**145**			**146**	**100.0%**	**311.61 [–153.71, 776.93]**	

Heterogeneity: Tau² = 201414.68; Chi² = 19.93, df = 4 (p = 0.0005); I² = 80%
Test for overall effect: Z = 1.31 (p = 0.19)



(b)

Study or Subgroup	Continuous Mean	SD	Total	Bolus Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
Makhoul 1997	3,672.5	1,353.7	10	2,833	1,042.7	10	8.5%	839.50 [–219.56, 1898.56]	1997
Thomson 2010	3,726	1,121	26	2,955	1,267	30	20.0%	771.00 [145.52, 1396.48]	2010
Llorens 2014	3,705	1,340	36	3,093	1,208	37	22.0%	612.00 [26.25, 1197.75]	2014
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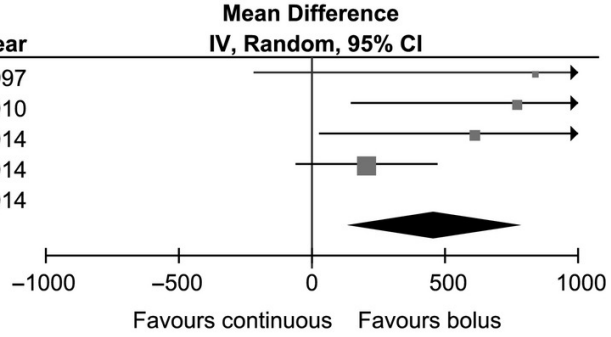
Shah 2014	721.57	447.99	30	1,117.15	726.7	30		–395.58 [–701.06, –90.10]	2014
Total (95% CI)			**115**			**116**	**100.0%**	**461.54 [133.67, 789.41]**	

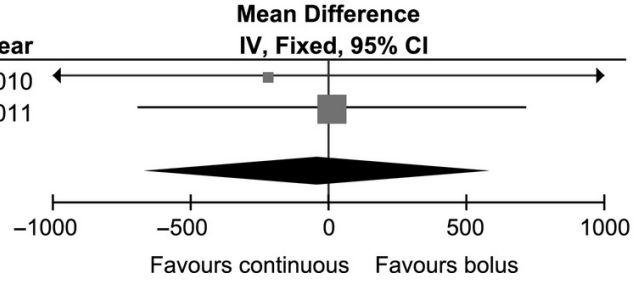
Heterogeneity: Tau² = 37990.23; Chi² = 4.45, df = 3 (p = 0.22); I² = 33%
Test for overall effect: Z = 2.76 (p = 0.006)



(c)

Study or Subgroup	Continuous Mean	SD	Total	Bolus Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI	Year
Allen 2010	4,894	2,205	20	5,113	2,258	21	21.0%	–219.00 [–1585.21, 1147.21]	2010
Felker 2011	4,249	3,104	152	4,237	3,208	156	79.0%	12.00 [–692.92, 716.92]	2011
Total (95% CI)			**172**			**177**	**100.0%**	**–36.57 [–663.02, 589.88]**	

Heterogeneity: Chi² = 0.09, df = 1 (p = 0.77); I² = 0%
Test for overall effect: Z = 0.11 (p = 0.91)



Thực tế sử dụng Furosemide

Ronco C, Bellomo R, Kellum JA (eds): Acute Kidney Injury.
Contrib Nephrol. Basel, Karger, 2007, vol 156, pp 236–249

Diuretics in the Management of Acute Kidney Injury: A Multinational Survey

Sean M. Bagshaw[a,g], *Anthony Delaney*[b,c], *Daryl Jones*[d], *Claudio Ronco*[f], *Rinaldo Bellomo*[a,e]

[a]Department of Intensive Care, Austin Hospital, Melbourne, Vic., [b]Department of Intensive Care, Royal North Shore Hospital, Sydney, [c]Northern Clinical School, University of Sydney, St. Leonards, [d]Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Vic., [e]Department of Medicine, Melbourne University, Melbourne, Vic., Australia, and [f]Department of Nephrology, St. Bortolo Hospital, Vicenza, Italy, [g]Division of Critical Care Medicine, University of Alberta Hospital, University of Alberta, Edmonton, Alta., Canada

Table 3. Summary of the responses pertaining to physiologic indications for use of diuretics in the management of ARF

Factor	Almost never, %	Infrequently %	Sometimes %	Frequently %	Almost always, %
Increasing SCr (n = 313)	29.4	21.1	31.6	14.4	3.5
Oliguria when SCr not known (n = 314)	29.9	22	22.9	19.1	6.1
Oliguria when SCr is increasing (n = 313)	16	19.8	32.3	21.4	10.5
Pulmonary edema (n = 314)	0.32	1	12.4	38.9	47.5
Metabolic acidosis (n = 314)	34.7	31.2	29.3	4.1	0.64
Hyperkalemia (n = 315)	7	16.5	38.1	25.1	13.3

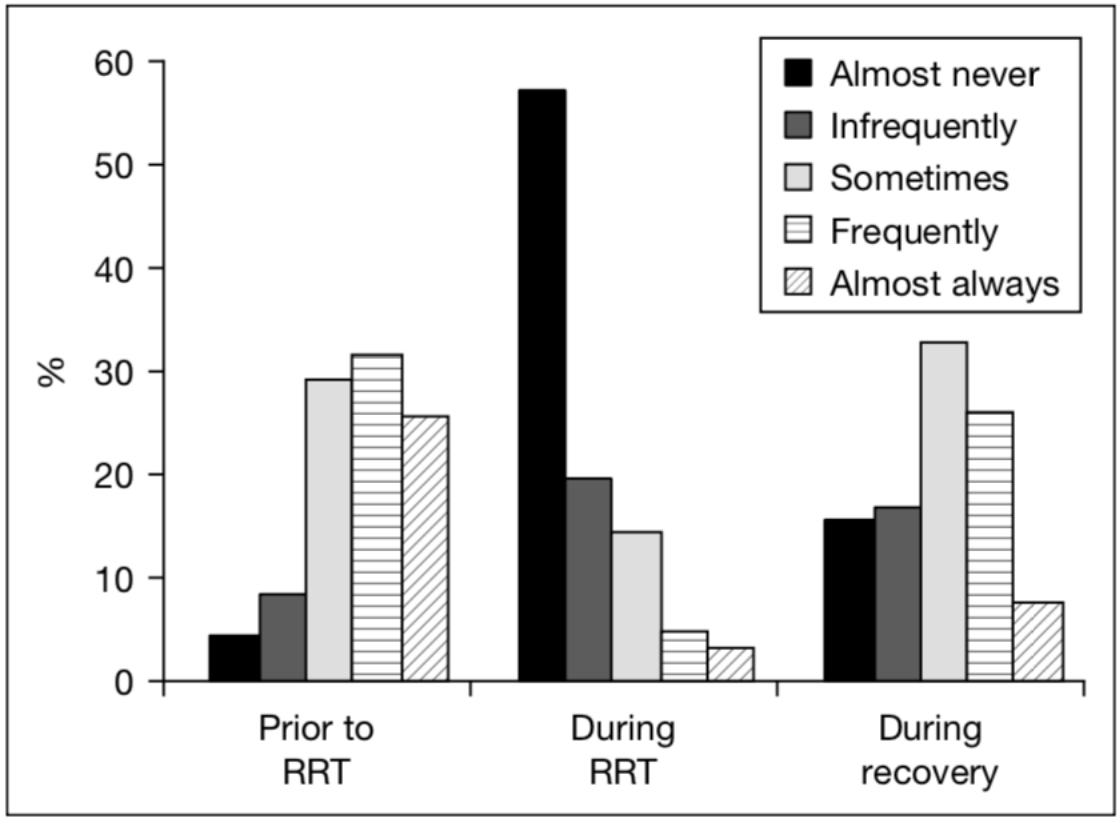


Fig. 3. Summary of responses pertaining to the timing of diuretic use in the management of ARF.

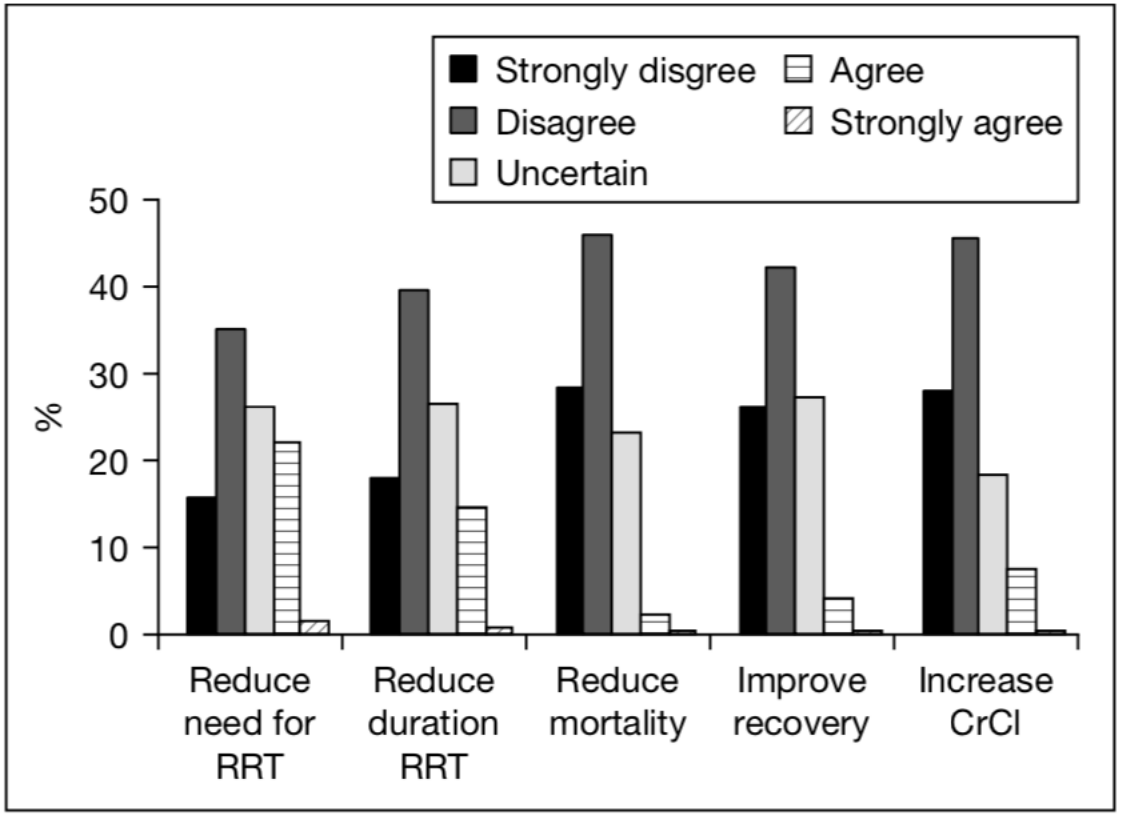
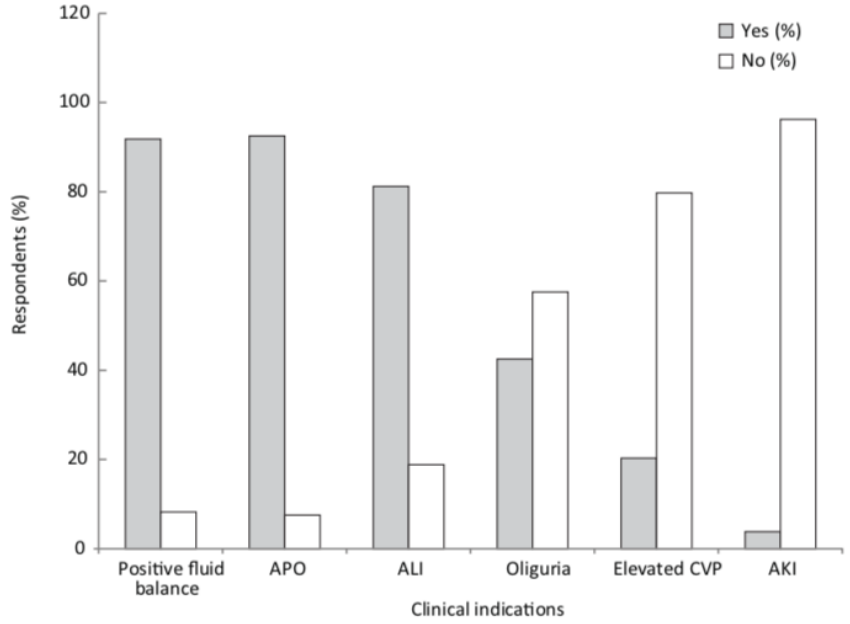


Fig. 4. Summary of responses pertaining to the beliefs about outcomes with the use of diuretics in the management of ARF.

Loop diuretic therapy in the critically ill: a survey

Sarah L Jones, Johan Mårtensson, Neil J Glassford, Glenn M Eastwood and Rinaldo Bellomo

Figure 1. Surveyed clinical indications for loop diuretic therapy and percentage of respondents who would give it



APO = acute pulmonary oedema. ALI = acute lung injury. CVP = central venous pressure. AKI = acute kidney injury.

Tóm lại

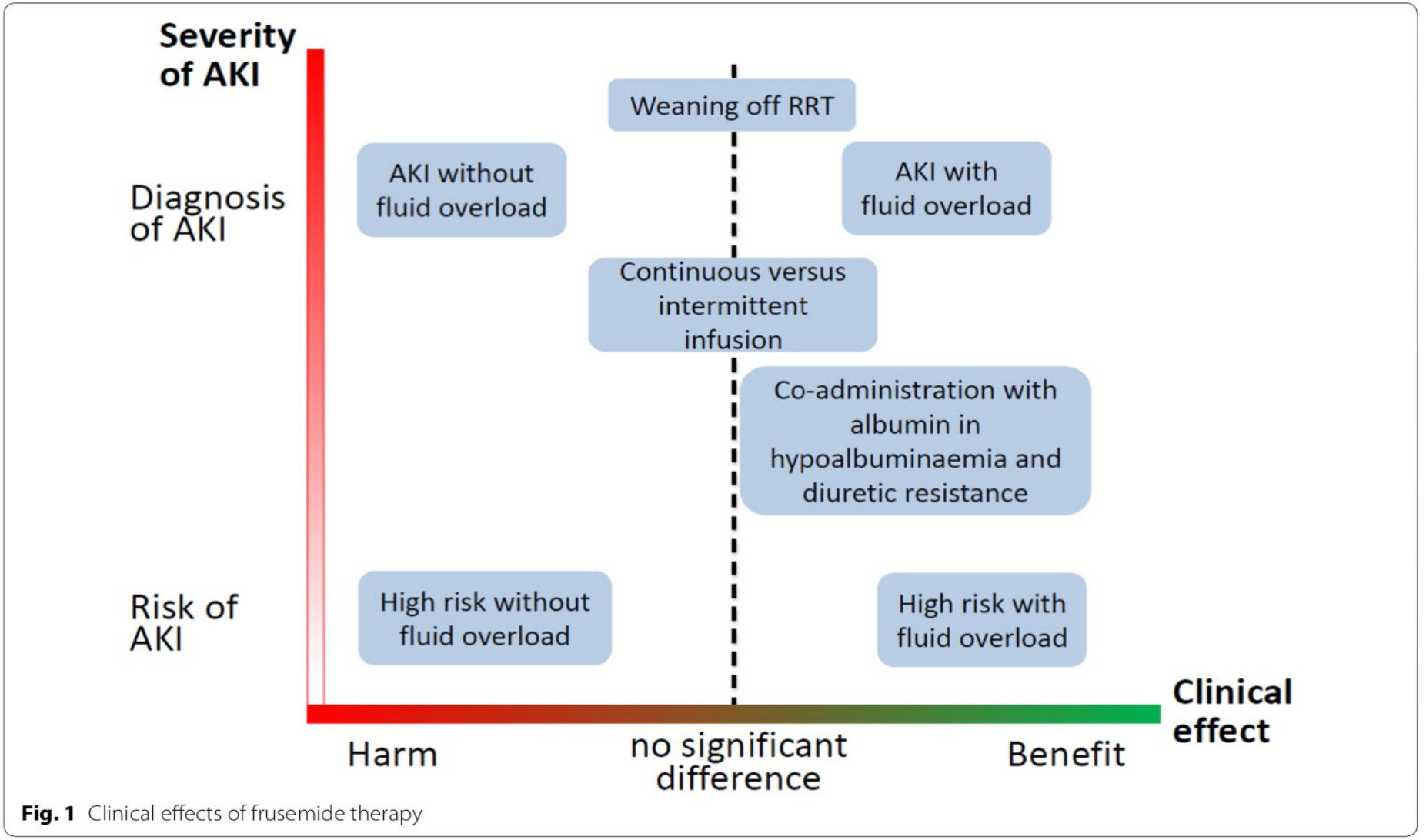


Fig. 1 Clinical effects of frusemide therapy

"10 myths about frusemide" Michael Joannidis1* , Sebastian J. Klein1 and Marlies Ostermann2

Thank you for listening!