Redécouvrir les diurétiques

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Congres CNCH 2017

Collège National des Cardiologues des Hôpitaux

DÉCLARATION DE RELATIONS PROFESSIONNELLES

Conférencier : Hrynchyshyn Nataliya, Pontoise

☑Je n'ai pas de lien d'intérêt potentiel à déclarer

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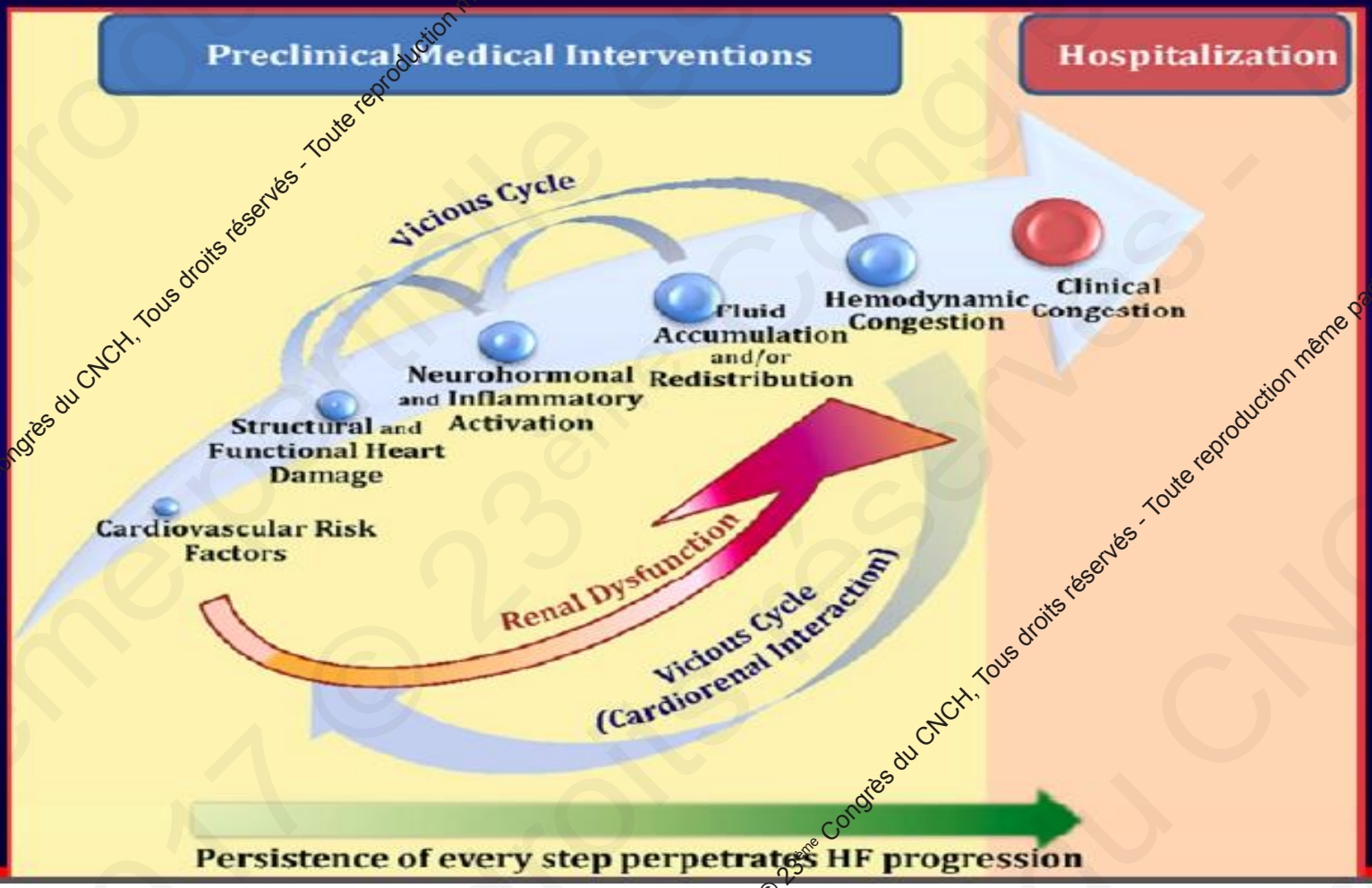
europa organisation

Water and Sodium in Heart Failure: A Spotlight on Congestion

Heart Fail Rev (2015) 20:13-24

Gaspare Parrinello · Stephen J. Greene · Daniele Torres · Michael Alderman · Joseph Vincent Bonventre · Pietro Di Pasquale · Luna Gargani · Anju Nohria · Gregg C. Fonarow · Muthiah Vaduganathan · Javed Butler · Salvatore Paterna · Lynne Warner Stevenson · Mihai Gheorghiade



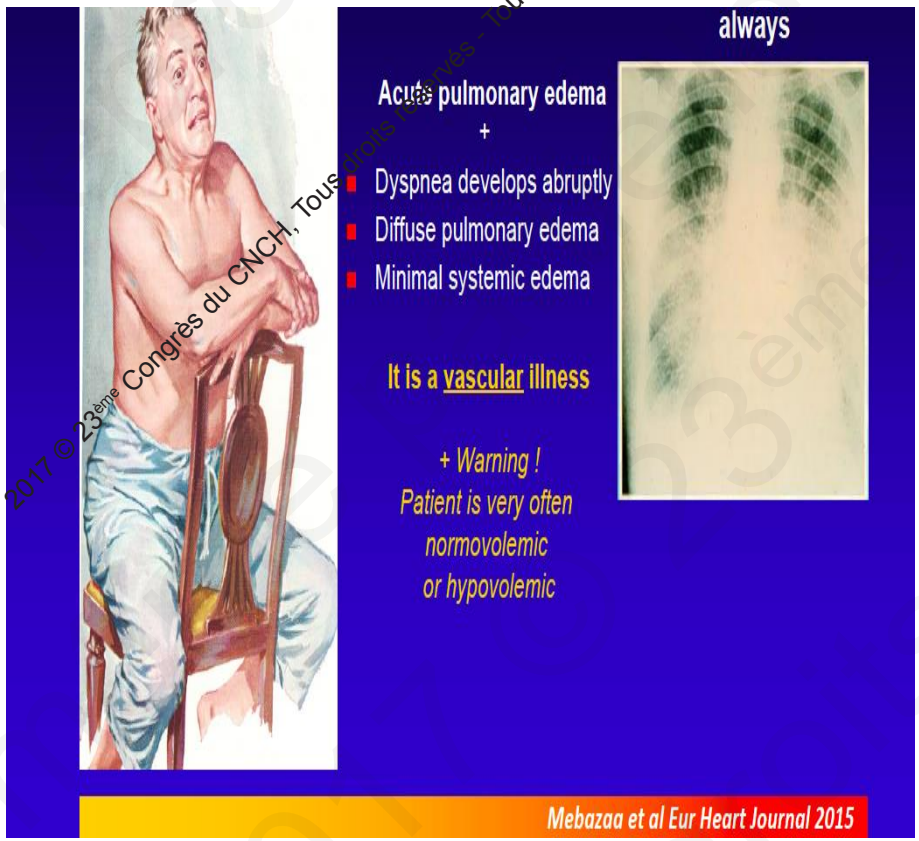
Congestion dans l'insuffisance cardiaque: une approche multi-paramètres

Variable	Score				
	−1	0	1	2	3
Bedside assessment					
Orthopnoea[a]		None	Mild	Moderate	Severe/worst
JVP (cm)	<8 and no hepatojugular reflux		8–10 or hepatojugular reflux	11–15	>16
Hepatomegaly	Absent in the setting of normal JVP	Absent	Liver edge	Moderate pulsatile enlargement	Massive tender enlargement extending to midline
Oedema		None	1+	2+	3+/4+
Laboratory					
Natriuretic peptides (one)					
BNP		<100	100–299	300–500	>500
NT pro-BNP		<400	400–1500	1500–3000	>3000
Dynamic manoeuvres					
Orthostatic testing	Significant decrease in SBP or increase in HR	No change in SBP or HR			
		No difficulty	Mild	Moderate	Severe/worst
6 min walk test	>400 m	300–400 m	200–300 m	100–200 m	<100 m
Valsalva manoeuvre	Normal response		Absent overshoot pattern	Square wave pattern	

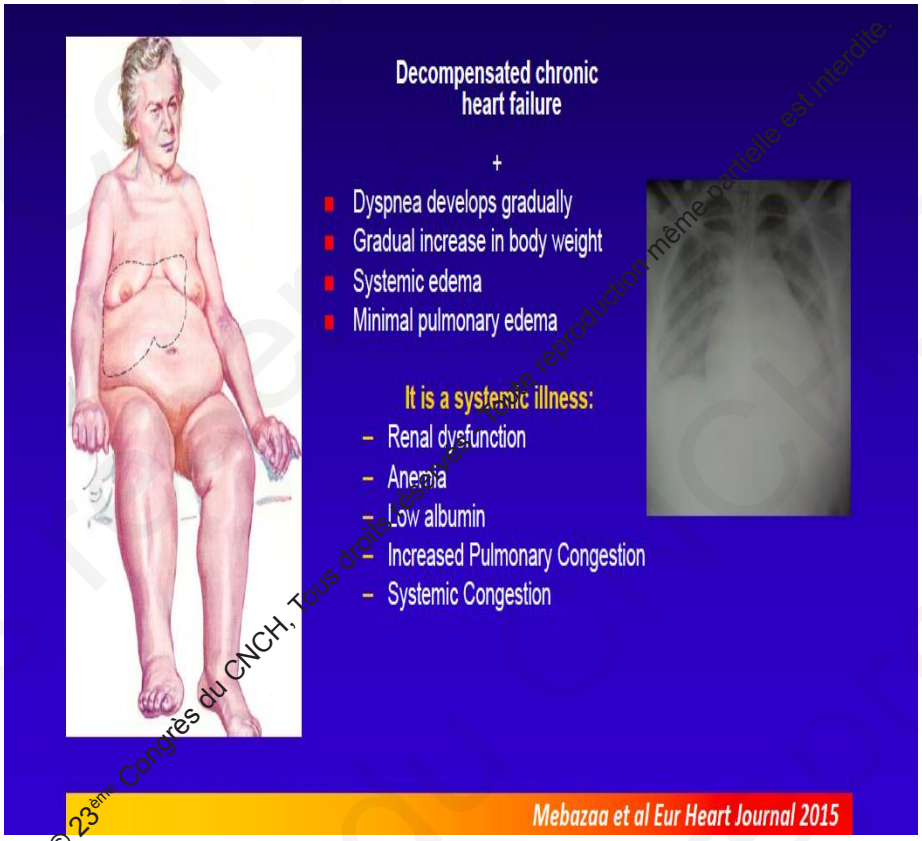
Gheorghiade et al. Eur J Heart Failure 2010

Les diurétiques sont-ils toujours utiles? Evaluez la volémie!

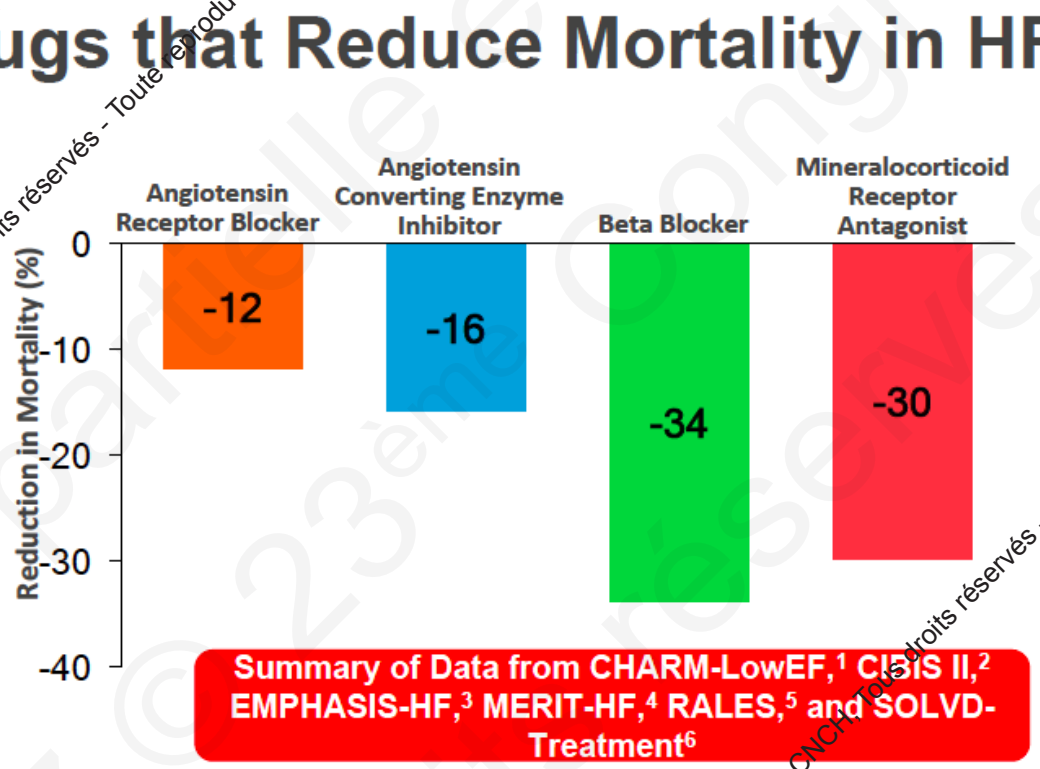
Dyspnée et/ou autres signes de congestion + HTA (>150 mmHg)



Dyspnée+ PAS 110-150 mmHG



Pas d'impact sur morbi-mortalité…?



1. Young JB, et al. *Circulation*. 2004;110:2618-26.
2. CIBIS II Investigators. *Lancet* 1999;353:9-13.
4. MERIT-HF Study Group. *Lancet* 1999;353:2001-7.
5. Pitt B, et al. *N Eng J Med*. 1999; 341:709-17
6. SOLVD Investigators. *New Engl J Med*. 1991

Time-to-Furosemide Treatment and Mortality in Patients Hospitalized With Acute Heart Failure

J Am Coll Cardiol 2017;69:3042-51

Yuya Matsue, MI … Kagiyama, MD,[c]
Tetsuo Yamaguc … e Kida, MD, PhD,[f]
Atsushi Mizuno, …
Ryuichi Matsuka … ıke, MD, PhD,[n]
Kenji Yoshioka, I … MD, PhD,[r]
Koji Murai, MD,[s] … MD, PhD[v,w]

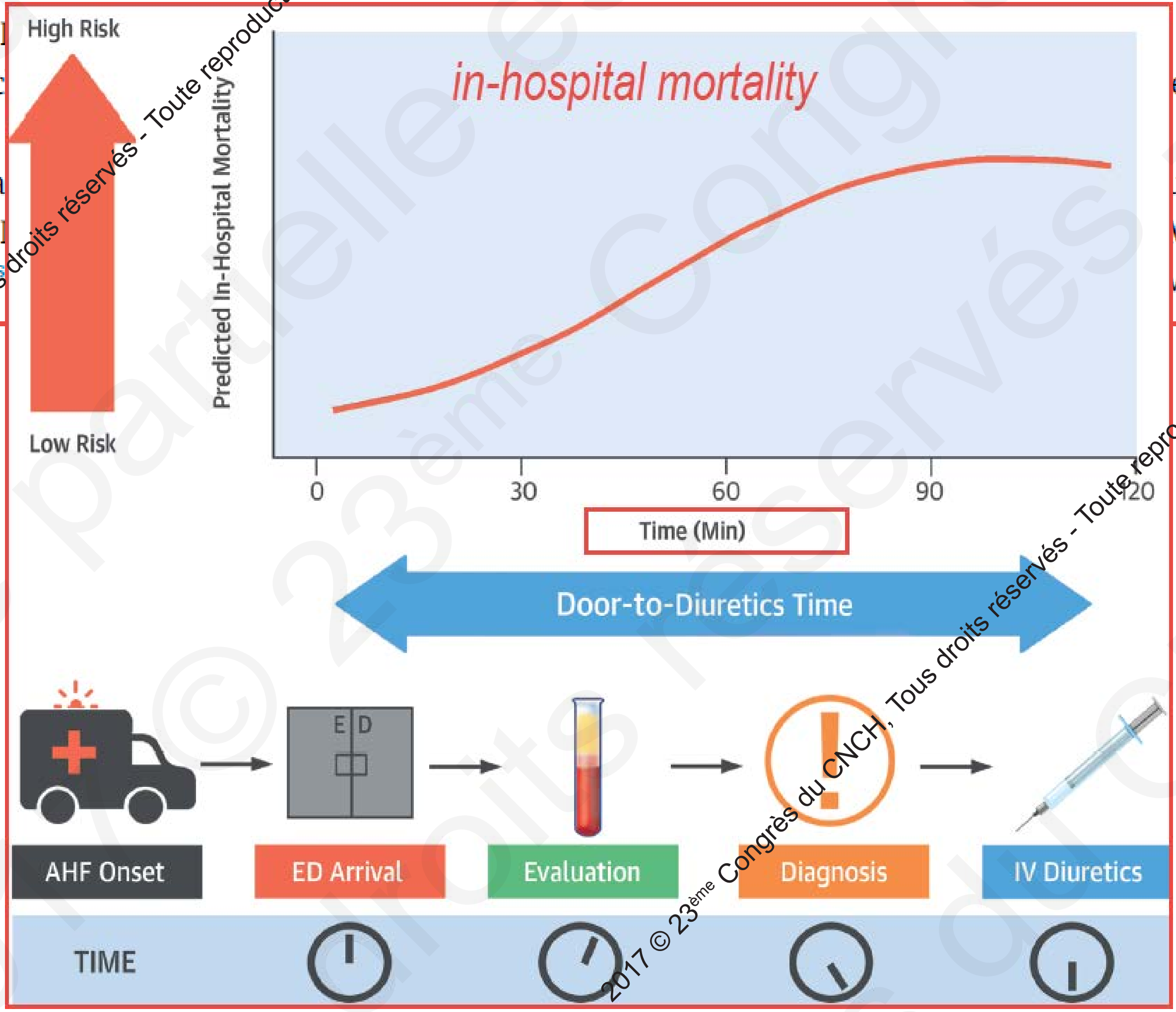


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Table 7.3 Doses of diuretics commonly used in patients with heart failure

Diuretics	Initial dose (mg)		Usual daily dose (mg)	
Loop diuretics[a]				
Furosemide	20–40		40–240	
Bumetanide	0.5–1.0		1–5	
Torasemide	5–10		10–20	
Thiazides[b]				
Bendroflumethiazide	2.5		2.5–10	
Hydrochlorothiazide	25		12.5–100	
Metolazone	2.5		2.5–10	
Indapamide[c]	2.5		2.5–5	
Potassium-sparing diuretics[d]				
	+ACE-I/ ARB	-ACE-I/ ARB	+ACE-I/ ARB	-ACE-I/ ARB
Spironolactone/ eplerenone	12.5–25	50	50	100–200
Amiloride	2.5	5	5–10	10–20
Triamterene	25	50	100	200

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

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The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 3, 2011 VOL. 364 NO. 9

Diuretic Strategies in Patients with Acute Decompensated Heart Failure

G. Michael Felker, M.D., M.H.S., Kerry L. Lee, Ph.D., David A. Bull, M.D., Margaret M. Redfield, M.D., Lynne W. Stevenson, M.D., Steven R. Goldsmith, M.D., Martin M. LeWinter, M.D., Anita Deswal, M.D., M.P.H., Jean L. Rouleau, M.D., Elizabeth O. Ofili, M.D., M.P.H., Kevin J. Anstrom, Ph.D., Adrian F. Hernandez, M.D., M.D., Abdallah G. Kfoury, M.D., Horng H. Chen, M.B., B.Ch., ran, M.D., Bradley A. Bart, M.D., Alice M. Mascette, M.D., D., and Christopher M. O'Connor, M.D., t Failure Clinical Research Network*

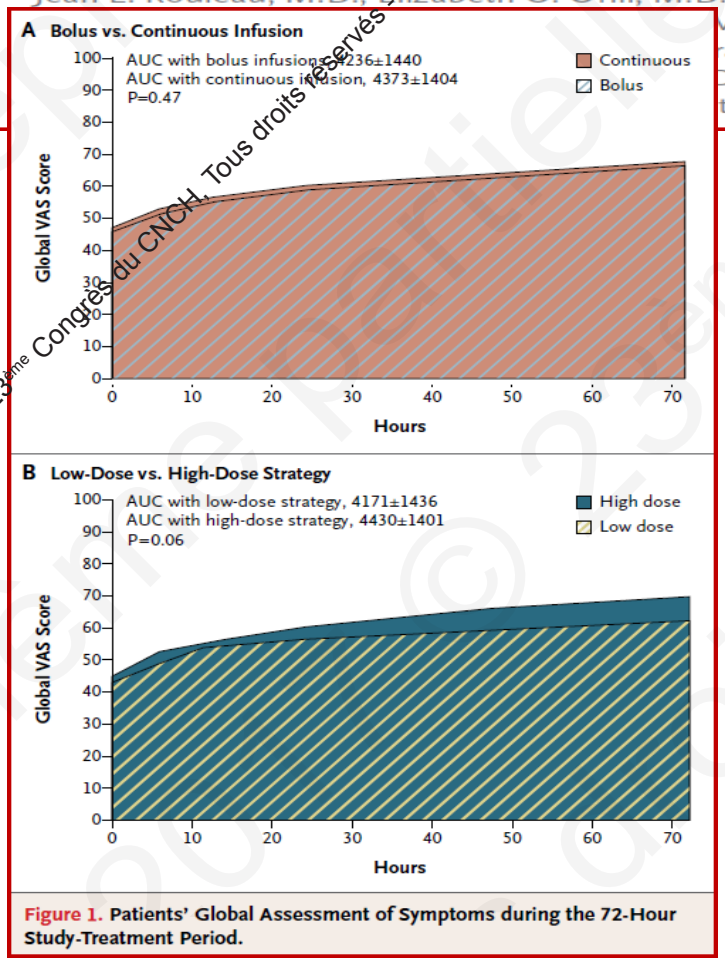
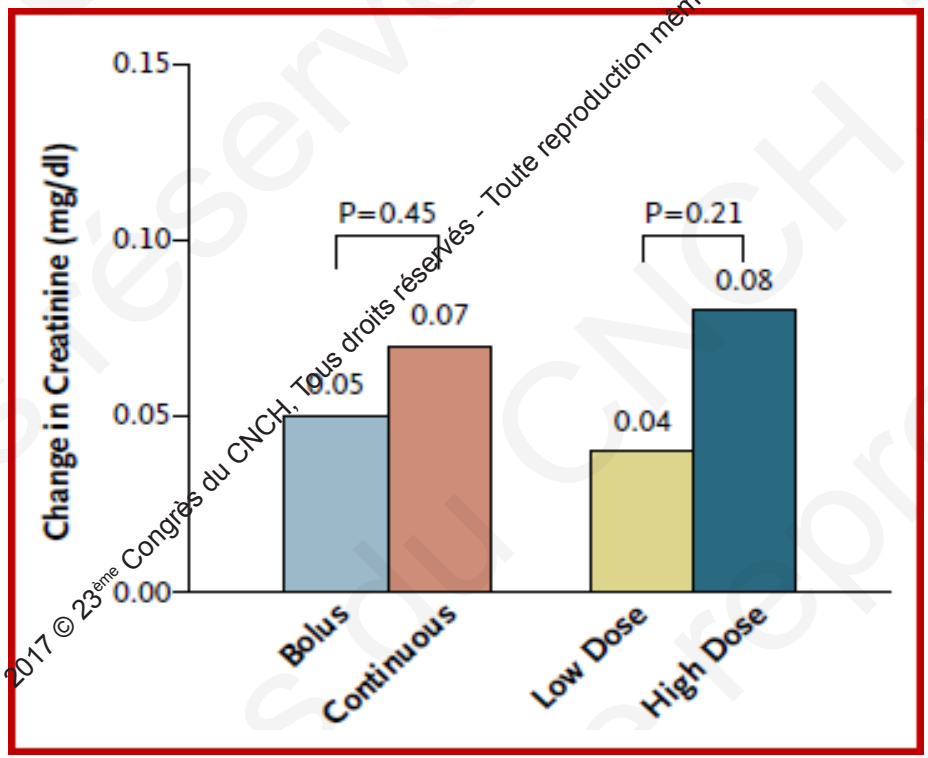


Figure 1. Patients' Global Assessment of Symptoms during the 72-Hour Study-Treatment Period.

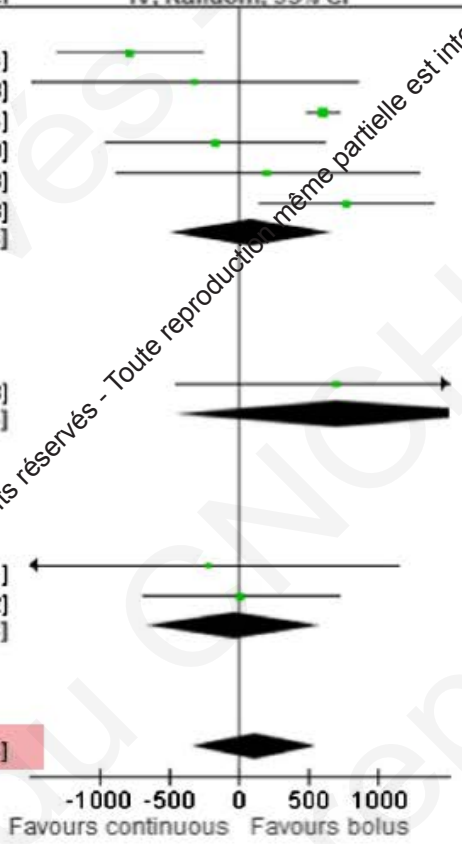


Loop diuretic strategies in patients with acute decompensated heart failure: A meta-analysis of randomized controlled trials ☆,☆☆

Mei-Yi Wu, MD [a], Nen-Chung Chang, MD, PhD [b], Chien-Ling Su, RT, MSc [c,d], Yung-Ho Hsu, MD [a], Tzen-Wen Chen, MD, PhD [e], Yuh-Feng Lin, MD, PhD [a,f], Chih-Hsiung Wu, MD, PhD [g], Kin-Wai Tam, MD, MSc [f,g,h,i,*]

Journal of Critical Care 29 (2014) 2-9

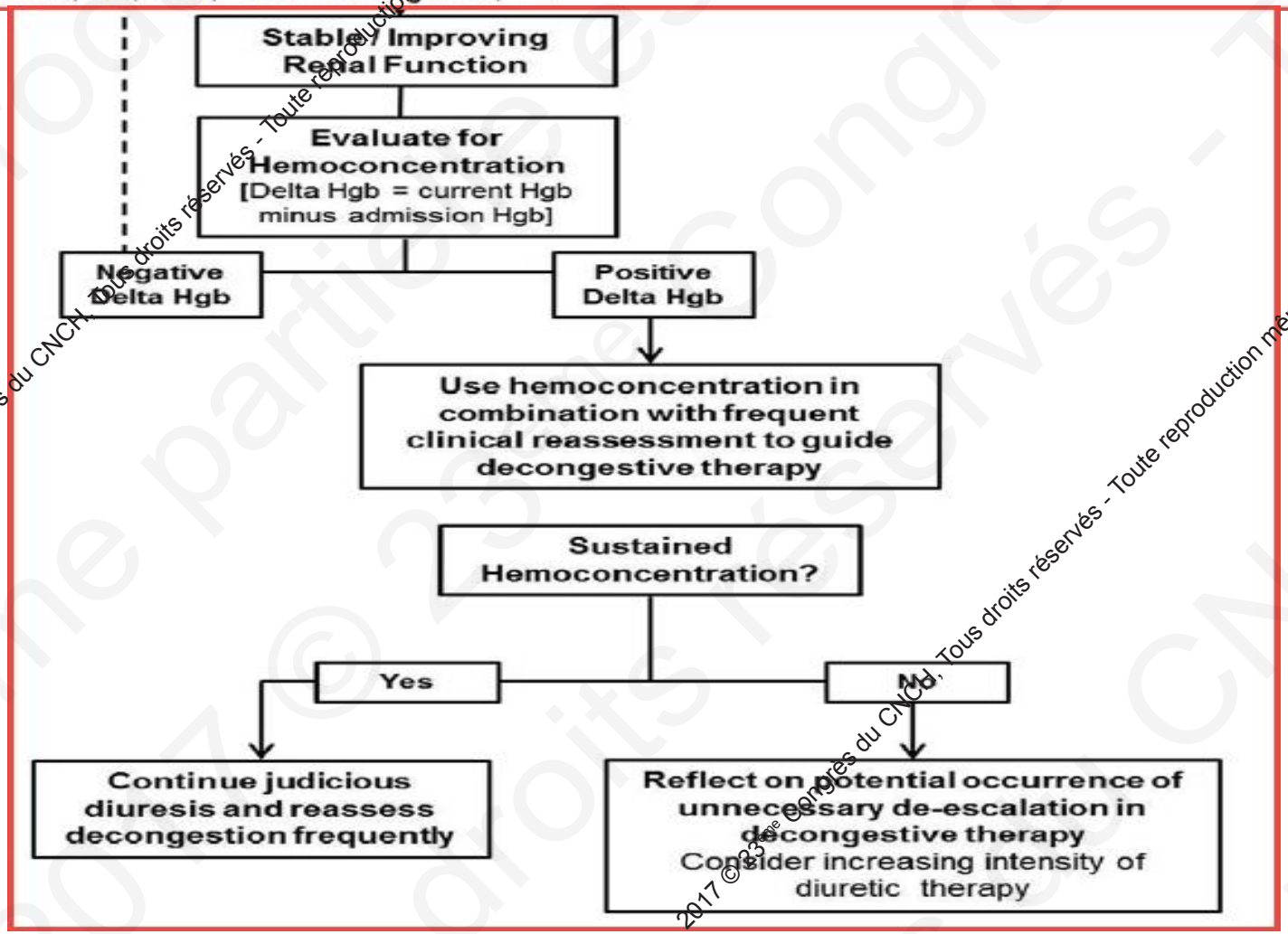
Study or Subgroup	Continuous Mean	SD	Total	Bolus Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI
2.1.1 Urine output (ml in 24hr)								
Aaser 1997	2,118	203.04	4	2,900	491.19	4	14.2%	-782.00 [-1302.86, -261.14]
Bagatin 1993	1,810	377.8	6	2,126.6	1,400	6	8.1%	-316.60 [-1476.88, 843.68]
Dormans 1996	2,860	240	20	2,260	150	20	17.3%	600.00 [475.96, 724.04]
Kramer 1996	1,727	672	8	1,891	903	8	11.5%	-164.00 [-943.99, 615.99]
Pivac 1998	3,250	1,175	10	3,046	1,302.5	10	8.7%	204.00 [-883.23, 1291.23]
Thomson 2010	3,726	1,121	26	2,955	1,267	30	13.1%	771.00 [145.52, 1396.48]
Subtotal (95% CI)			74			78	72.9%	84.10 [-500.67, 668.88]
Heterogeneity: Tau² = 395210.76; Chi² = 31.45, df = 5 (P < .00001); I² = 84%								
Test for overall effect: Z = 0.28 (P = .78)								
2.1.2 Urine output (ml in 48 hr)								
Lahav 1992	4,490	1,328.9	9	3,790	1,163	9	8.1%	700.00 [-453.73, 1853.73]
Subtotal (95% CI)			9			9	8.1%	700.00 [-453.73, 1853.73]
Heterogeneity: Not applicable								
Test for overall effect: Z = 1.19 (P = .23)								
2.1.3 Urine output (ml in 72 hr)								
Allen 2010	4,894	2,205	20	5,113	2,258	21	6.7%	-219.00 [-1585.21, 1147.21]
Felker 2011	4,249	3,104	152	4,237	3,208	156	12.3%	12.00 [-692.92, 716.92]
Subtotal (95% CI)			172			177	19.0%	-36.57 [-666.02, 589.88]
Heterogeneity: Tau² = 0.00; Chi² = 0.09, df = 1 (P = .77); I² = 0%								
Test for overall effect: Z = 0.11 (P = .91)								
Total (95% CI)			255			264	100.0%	110.98 [-338.00, 559.95]
Heterogeneity: Tau² = 298448.71; Chi² = 34.46, df = 8 (P < .0001); I² = 77%								
Test for overall effect: Z = 0.48 (P = .63)								
Test for subgroup differences: Chi² = 1.22, df = 2 (P = .54), I² = 0%								



Hemoconcentration-guided Diuresis in Heart Failure

The American Journal of Medicine (2014) 127, 1154-1159

Muthiah Vaduganathan, MD, MPH,[a] Stephen J. Greene, MD,[b] Gregg C. Fonarow, MD,[c] Adriaan A. Voors, MD, PhD,[d] Javed Butler, MD, MPH,[e] Mihai Gheorghiade, MD[b]



Altération de la fonction rénale

Dans l'ensemble,
l'aggravation de la fonction rénale,
principalement définie comme > 26.5 µmol/L
(>0.3 mg/dL) est associée à un mauvais
pronostic

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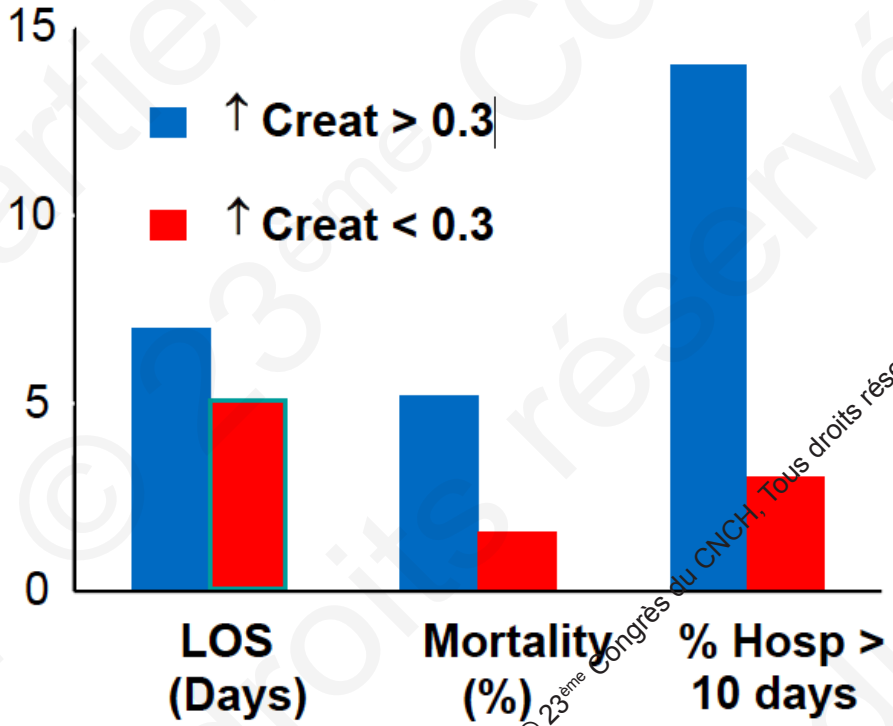
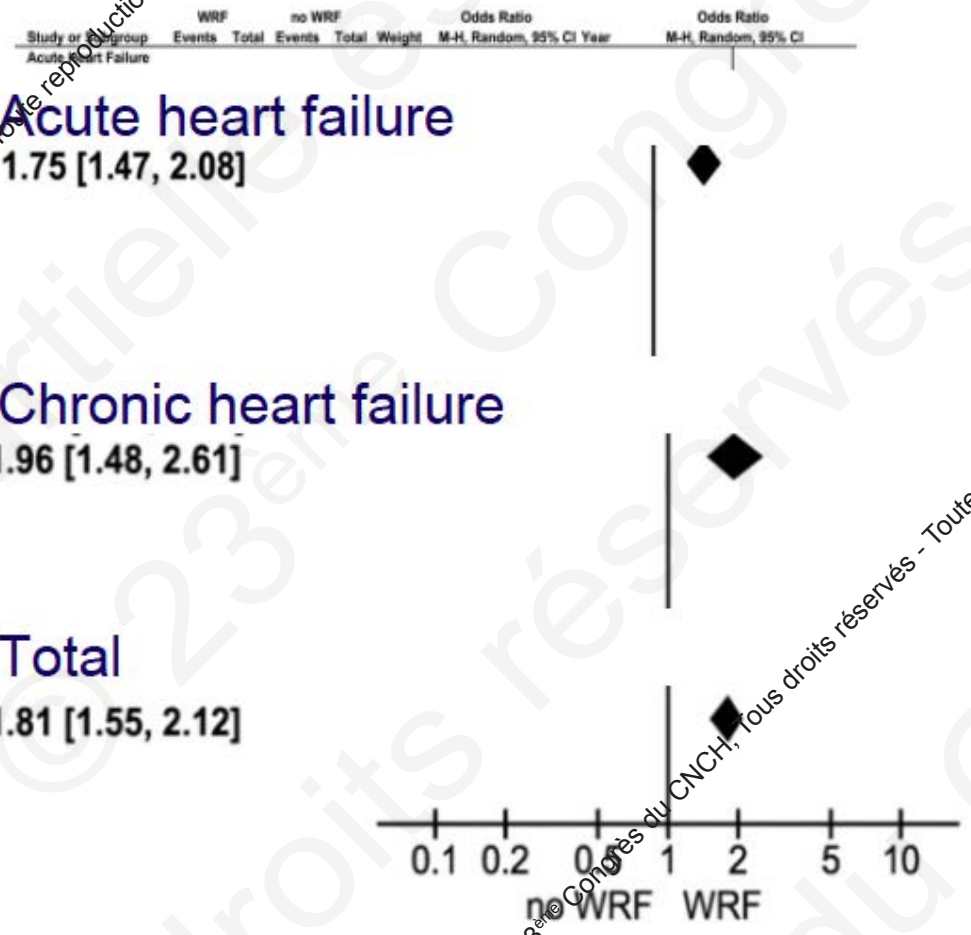
Effect of Worsening Renal Function in Hospitalized Heart Failure Patients



From Butler et al. AHJ 2004.

Forest plot of combined all-cause mortality for WRF vs. no WRF (28 studies, 49,890 patients) Damman K



Damman K et al. Eur Heart J 2014;35(7):455-69.

Syndrome cardio-rénal

Cardio-Renal syndromes

General Definition:

Pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other

CRS Type I (Acute Cardiorenal Syndrome)

Abrupt worsening of cardiac function leading to acute kidney injury

CRS Type II (Chronic Cardiorenal Syndrome)

Chronic abnormalities in cardiac function causing progressive and permanent chronic kidney disease

CRS Type III (Acute Renocardiac Syndrome)

Abrupt worsening of renal function causing acute cardiac disorders

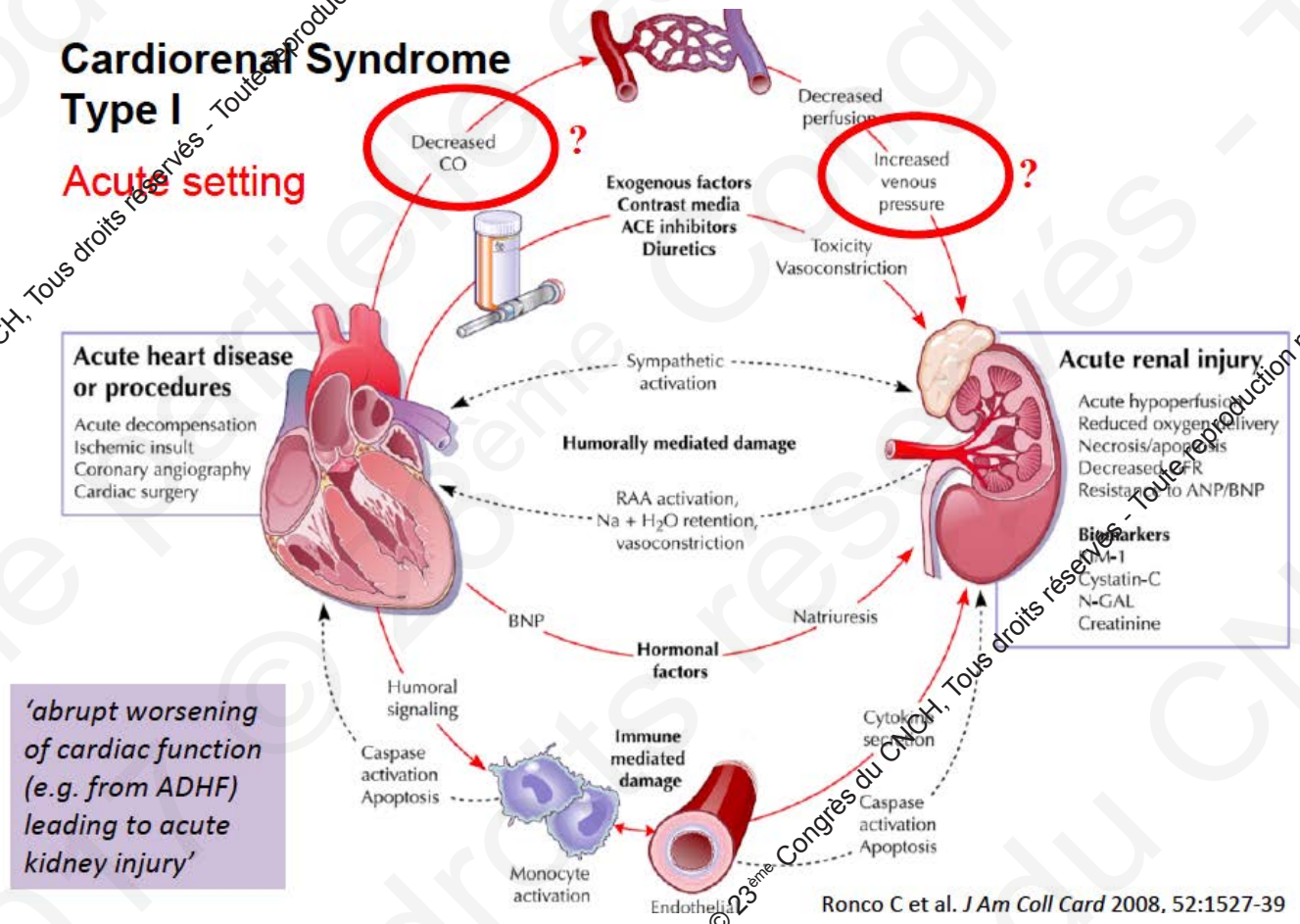
CRS Type IV (Chronic Renocardiac Syndrome)

Chronic kidney disease contributing to decreased cardiac function, cardiac hypertrophy and/or increased risk of adverse cardiovascular events

CRS Type V (Secondary Cardiorenal Syndrome)

Systemic condition (e.g. diabetes mellitus, sepsis) causing both cardiac and renal dysfunction

Syndrome cardio-rénal



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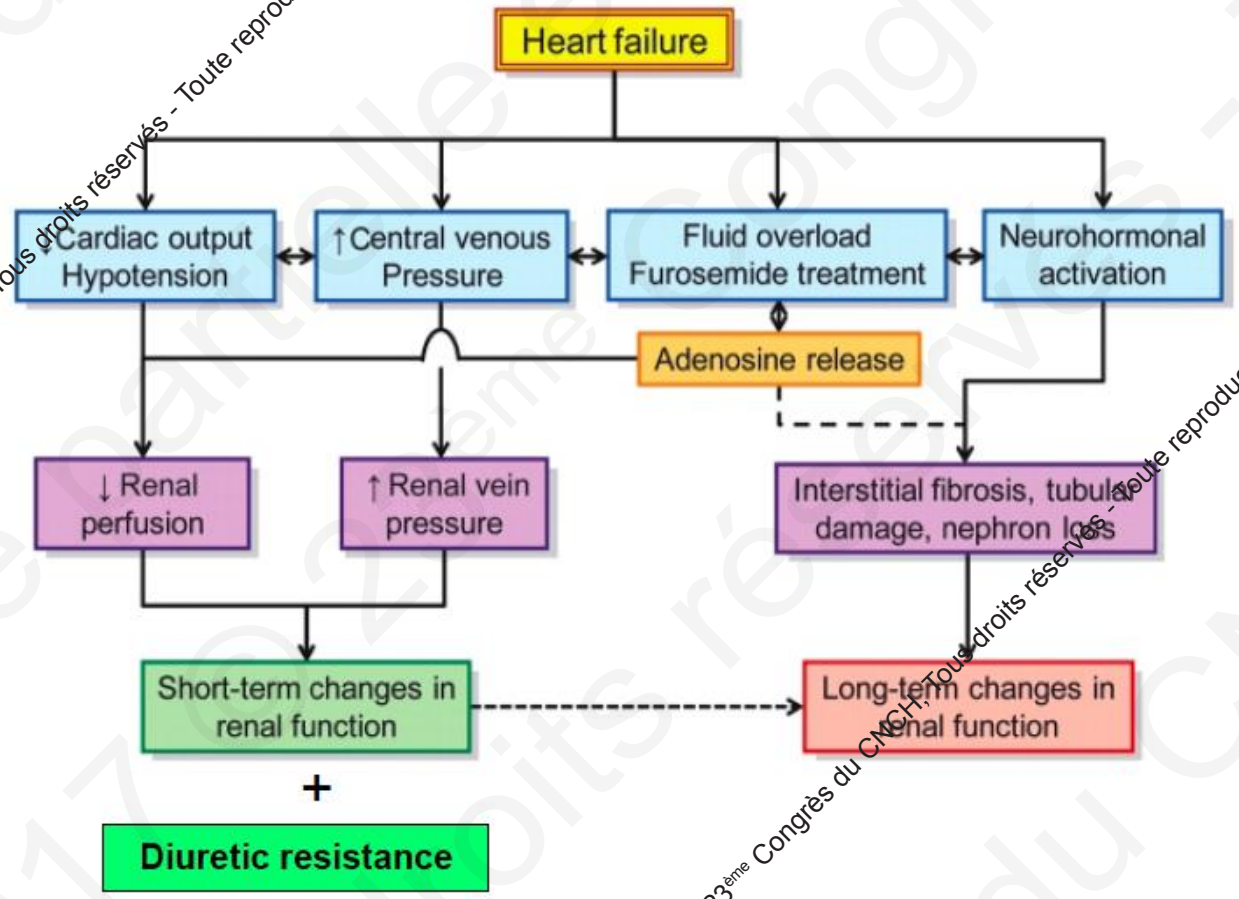
Impact d'insuffisance cardiaque sur la fonction rénale

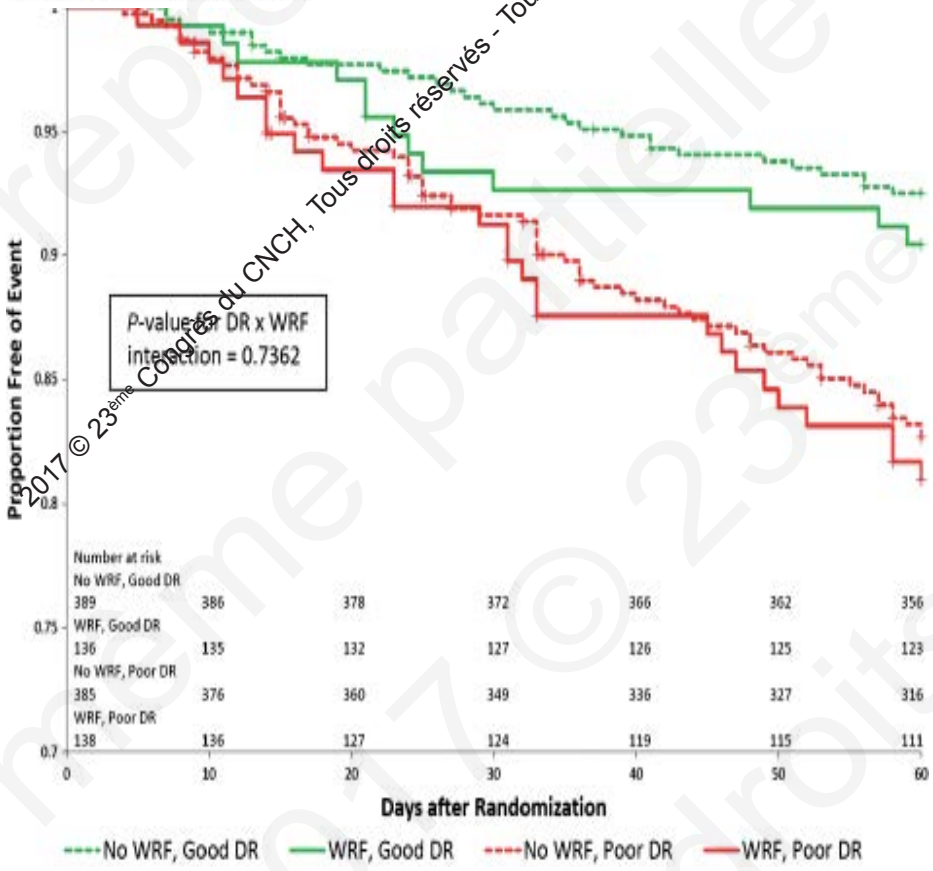


Metra et al. Eur Heart J 2012;33:2135-2142

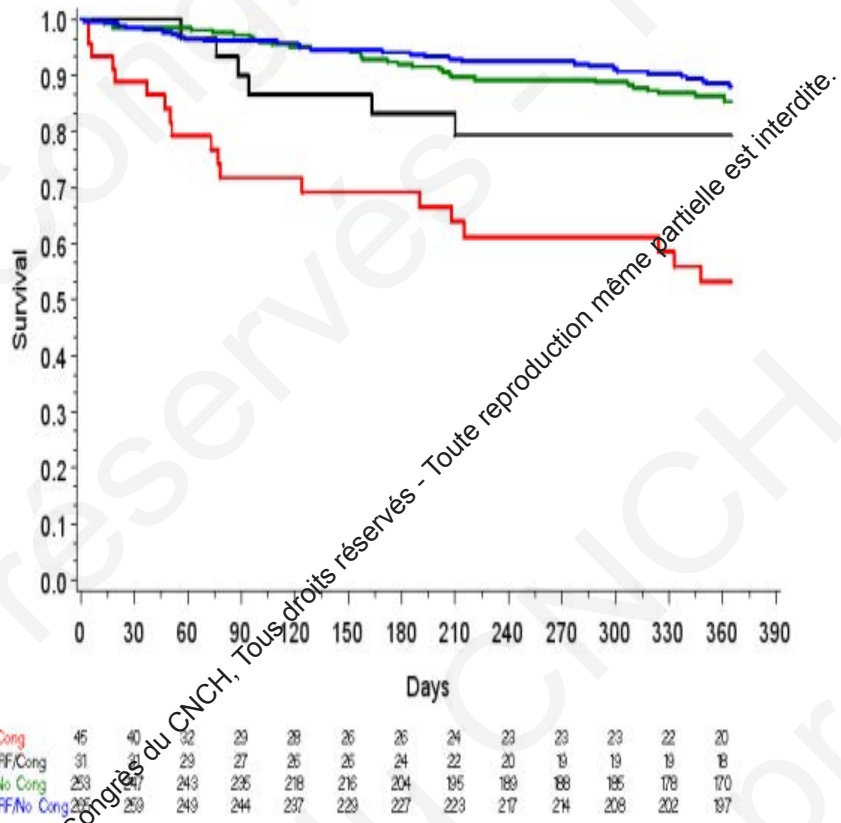
European Journal of Heart Failure (2014) 16, 1230–1240
doi:10.1002/ejhf.170

Diuretic response in patients with acute decompensated heart failure: characteristics and clinical outcome—an analysis from RELAX-AHF

Adriaan A. Voors[1]*, Beth A. Davison[2], John R. Teerlink[3], G. Michael Felker[4], Gad Cotter[2], Gerasimos Filippatos[5], Barry H. Greenberg[6], Peter S. Pang[7], Bruce Levin[8], Tsushung A. Hua[9], Thomas Severin[9], Piotr Ponikowski[10], Marco Metra[11], for the RELAX-AHF Investigators



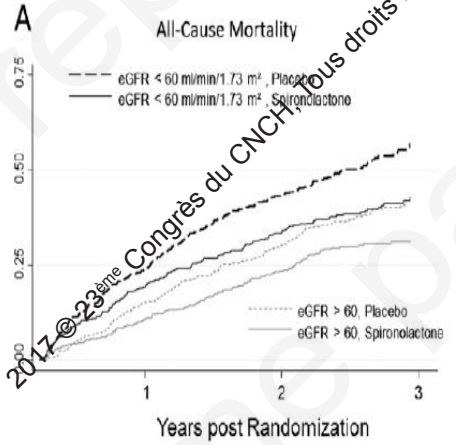
Death or urgent Tx in patients subdivided on the basis of volume status and WRF



Metra et al. Circ Haert Fail, 2012

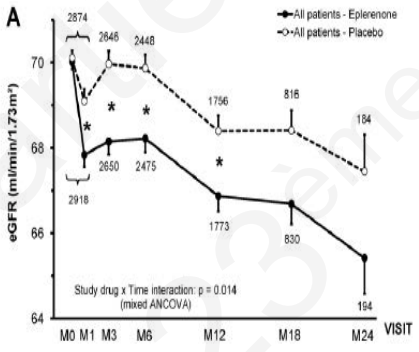
MRA and WRF

RALES



	Risk Associated With Worsening eGFR	
	Placebo (n = 60)	Spiro (n = 139)
Crude	HR 1.8 (1.3–2.5)	HR 0.99 (0.72–1.3)
Adjusted*	HR 1.9 (1.3–2.6)	HR 1.1 (0.79–1.5)

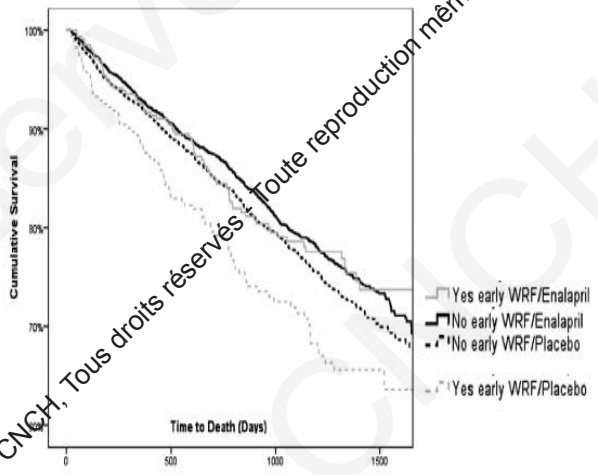
EPHESUS



Vardeny et al JACC 2012 / Rossignol et al Circ Heart Fail 2012

WRF – RAAS-inhibition

- WRF during ACEi/ARB initiation occurs in ≈ 10%, but is <u>not</u> associated with poor outcome



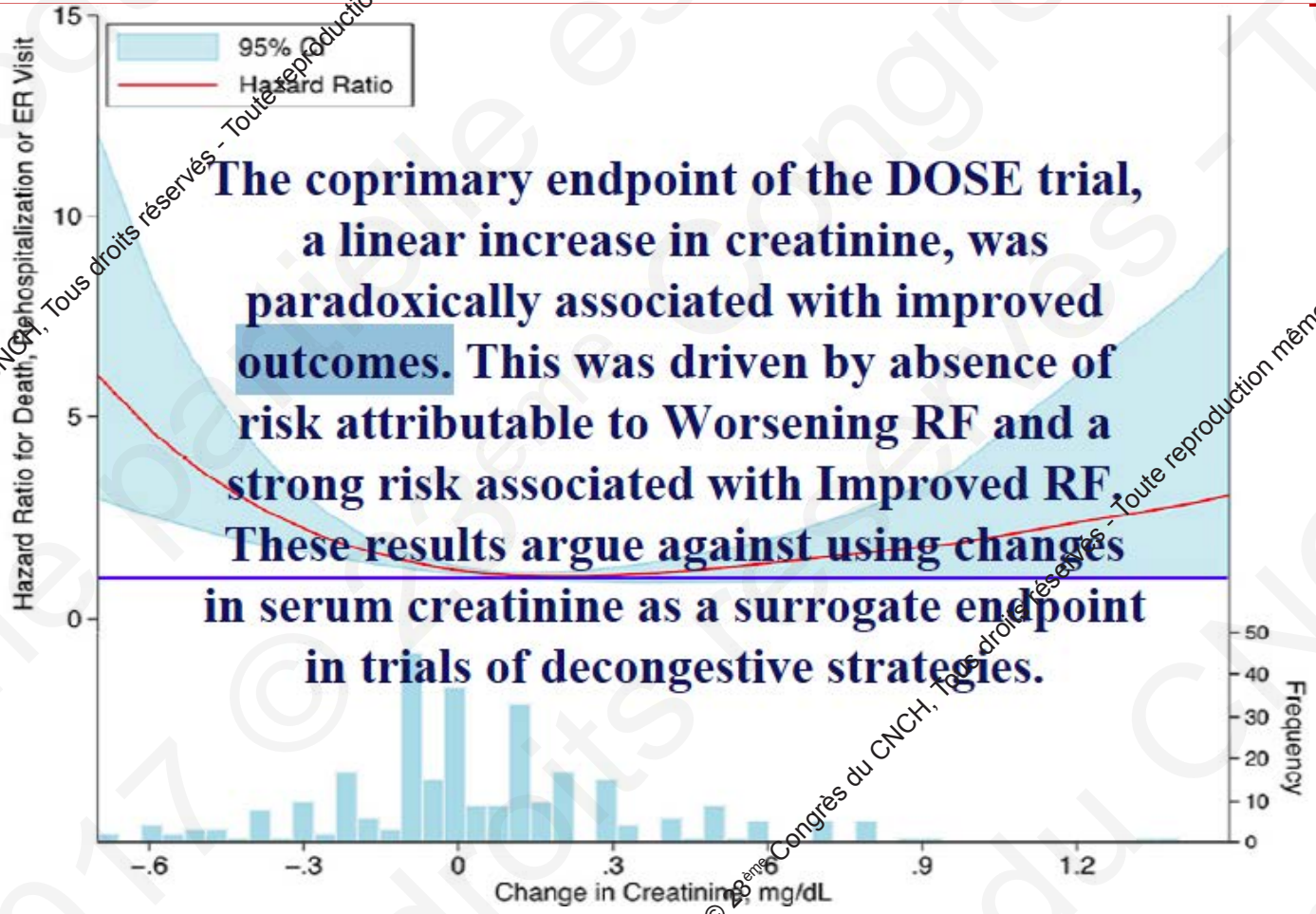
SOLVD

(Testani et al Circ Heart Fail 2011)

Relevance of Changes in Serum Creatinine During a Heart Failure Trial of Decongestive Strategies: Insights From the DOSE Trial

J Cardiac Failure, 2016

MEREDITH A. BRISCO, MD, MSCE,[1] MICHAEL R. ZILE, MD,[2] JENNIFER S. HANBERG, BA,[3] F. PERRY WILSON, MD, MSCE,[3,4] CHIRAG R. PARIKH, MD, PhD,[3,4] STEVEN G. COCA, DO, MS,[5] W.H. WILSON TANG, MD,[6] AND JEFFREY M. TESTANI, MD, MTR[3,4]



The coprimary endpoint of the DOSE trial, a linear increase in creatinine, was paradoxically associated with improved outcomes. This was driven by absence of risk attributable to Worsening RF and a strong risk associated with Improved RF. These results argue against using changes in serum creatinine as a surrogate endpoint in trials of decongestive strategies.

PROBLEM SOLVING

Worsening renal function and hyperkalaemia:

- Some rise in urea (BUN), creatinine, and potassium is to be expected after an ACE-I; if an increase is small and asymptomatic, no action is necessary.
- An increase in creatinine of up to 50% above baseline, or 266 µmol/L (3 mg/dL)/eGFR <25 mL/min/1.73 m^2, whichever is the smaller, is acceptable.
- An increase in potassium to ≤5.5 mmol/L is acceptable.
- If urea, creatinine, or potassium does rise excessively, consider stopping concomitant nephrotoxic drugs (e.g. NSAIDs[d]) and other potassium supplements or retaining agents (triamterene, amiloride) and, if no signs of congestion, reducing the dose of diuretic.
- If greater rises in creatinine or potassium than those outlined above persist despite adjustment of concomitant medications, the dose of the ACE-I (or ARB) should be halved and blood chemistry re-checked within 1–2 weeks; if there is still an unsatisfactory response, specialist advice should be sought.
- If potassium rises to >5.5 mmol/L or creatinine increases by >100% or to >310 µmol/L (3.5 mg/dL)/eGFR <20 mL/min/1.73 m^2, the ACE-I (or ARB) should be stopped and specialist advice sought.
- Blood chemistry should be monitored frequently and serially until potassium and creatinine have plateaued.

Algorithme de PEC d'aggravation de fonction rénale (AFR)

AFR

Diminution de congestion, hypoTA, initiation des IEC/ARA2

Persistance de congestion = AFR « VRAI »

Thérapie diurétique combinée: Thiazides /Spironolactone. NaCl hypertonique?

Persistance de congestion

Thérapies de remplacement rénale

OUI

AFR « FAUX »

Passer au traitement PO

Non

La résistance aux diurétiques

- La résistance aux diurétiques est définie comme l'incapacité d'obtenir un effet thérapeutique satisfaisant (diminution des oedèmes), malgré la dose maximale de diurétiques
- Hoorn EJ and Ellison DH. Am J Kidney Dis. 69(1):136-142.

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Définitions de résistance aux diurétiques et mesures réponse

Box 1 | Definitions of diuretic resistance

- Persistent congestion despite adequate and escalating doses of diuretic with >80 mg furosemide per day[107]
- Amount of sodium excreted as a percentage of filtered load <0.2%[108]
- Failure to excrete at least 90 mmol of sodium within 72 h of a 160 mg oral furosemide dose given twice daily[109]

Box 2 | Metrics of diuretic response

- Weight loss per unit of 40 mg furosemide (or equivalent)[5,51]
- Net fluid loss per milligram of loop diuretic (40 mg of furosemide or equivalent) during hospitalization[6]
- Natriuretic response to furosemide as the ratio of urinary sodium to urinary furosemide[52]

ter Maaten, J. M. et al. Nat. Rev. Cardiol. 12, 184–192 (2015);

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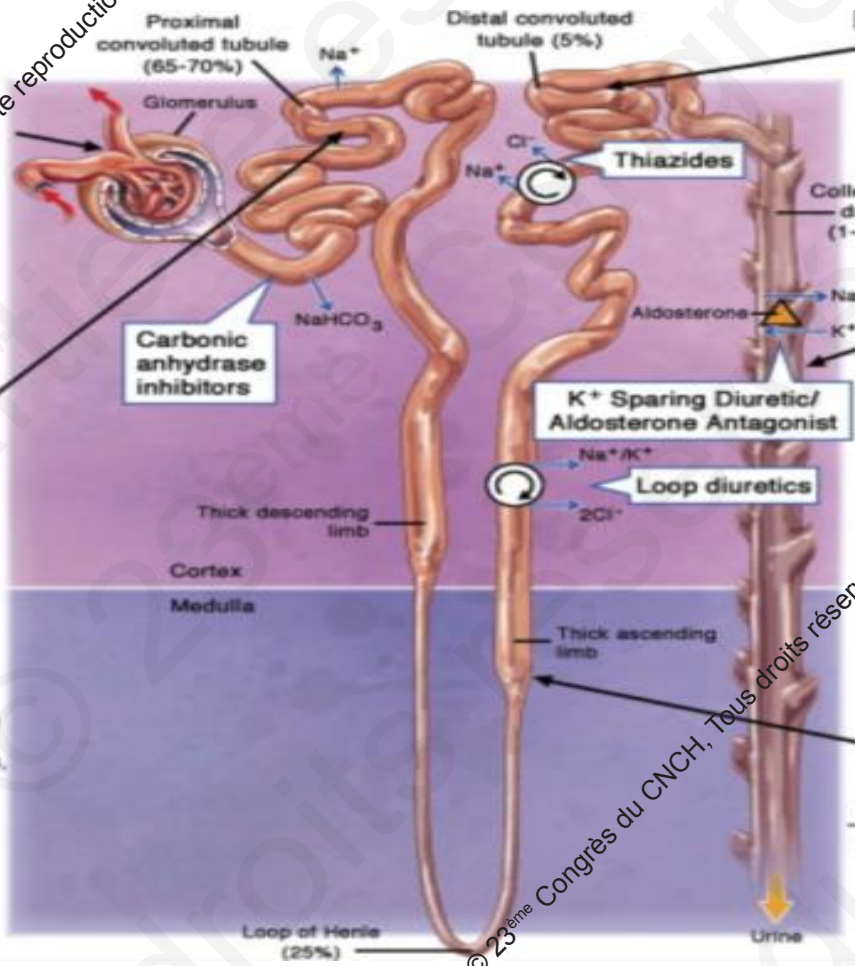
Diuretic Resistance and the Nephron

Reduced GFR:

Barriers	Potential solution
Abnormal glomerular hemodynamics	Discontinue NSAIDs, consider holding ACEI/ARB
Low cardiac output	Hemodynamic support
Chronic kidney disease or functional renal hypoperfusion	Increase LD dose

Proximal Tubule Hyperfunction:

Barriers	Potential solution
Neuro-hormonal activation	ACEI/ARB
Sodium-avid states	Increased LD doses, proximal tubule diuretics (i.e. acetazolamide)
Post-diuretic effect	Multiple daily doses, continuous LD infusion
Excessive daily sodium intake	Sodium restriction



Distal Tubule Hypertrophy:

Barriers	Potential solution
Rebound sodium retention	Sequential nephron blockade (Combination diuretic therapy)

Distal Nephron Hyperfunction:

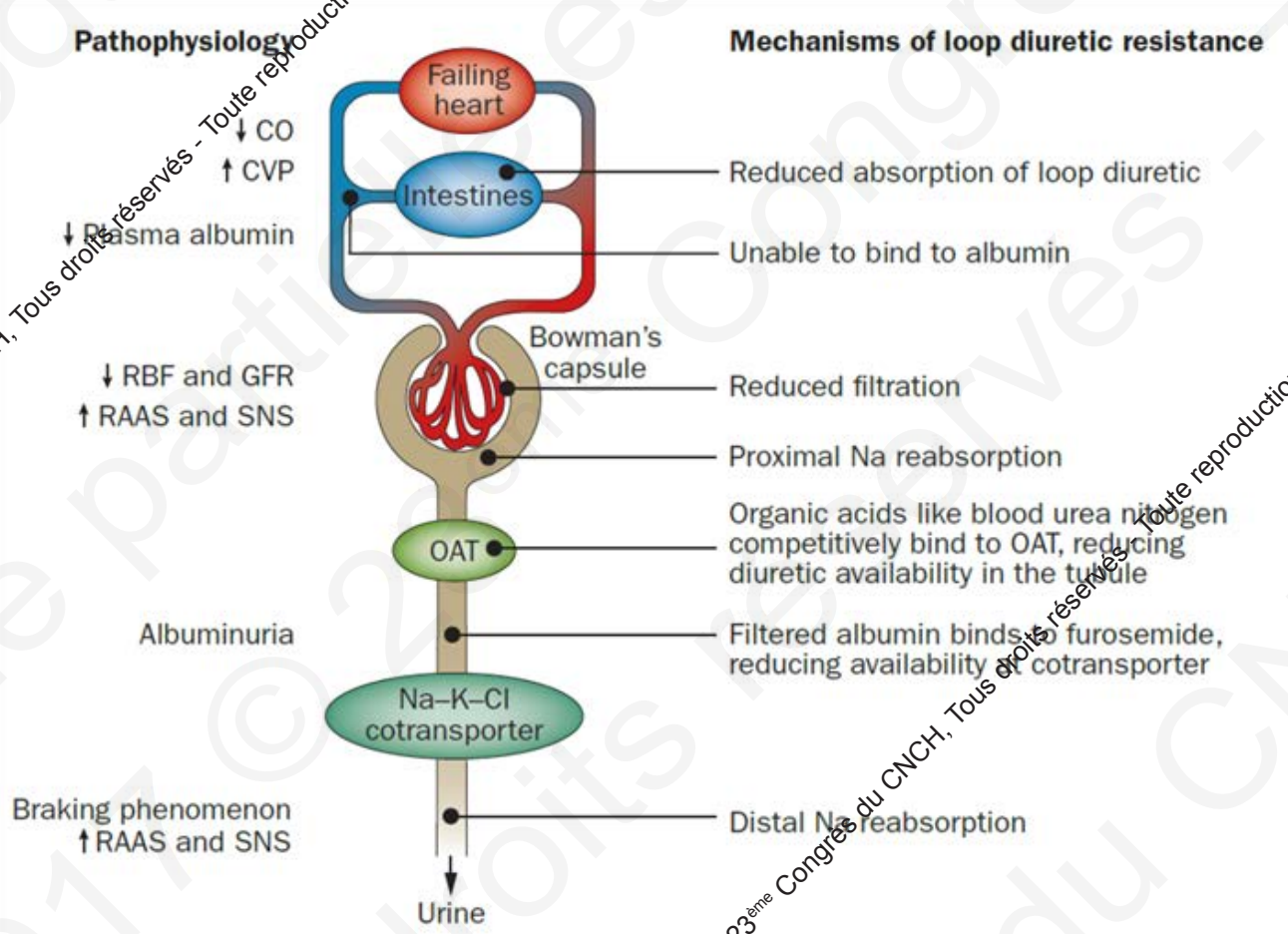
Barriers	Potential solution
Excessive aldosterone-mediated sodium retention	Aldosterone antagonist, K+-sparing diuretic (ENaC blocker)
Excessive vasopressin-mediated water retention	Vasopressin antagonist, free water restriction

Loop of Henle Hyperfunction:

Barriers	Potential solution
Braking effect	Higher LD doses

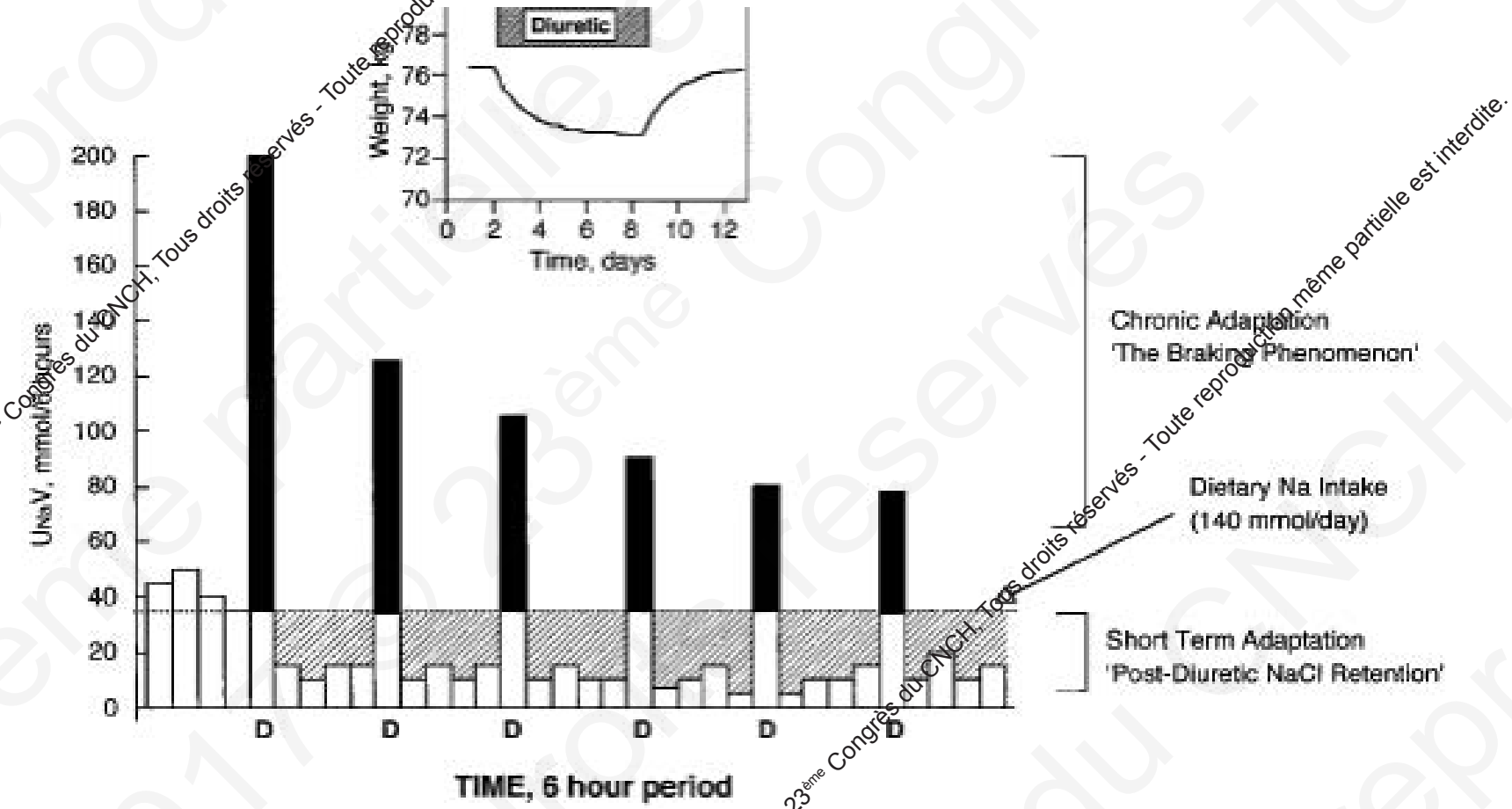
Jentzer, deWald, Hernandez. J Am Coll Cardiol. 2010;56(19):1527-1534.

Mécanisme de résistance aux diurétiques de l'anse



ter Maaten, J. M. et al. Nat. Rev. Cardiol. 12, 184–192 (2015);

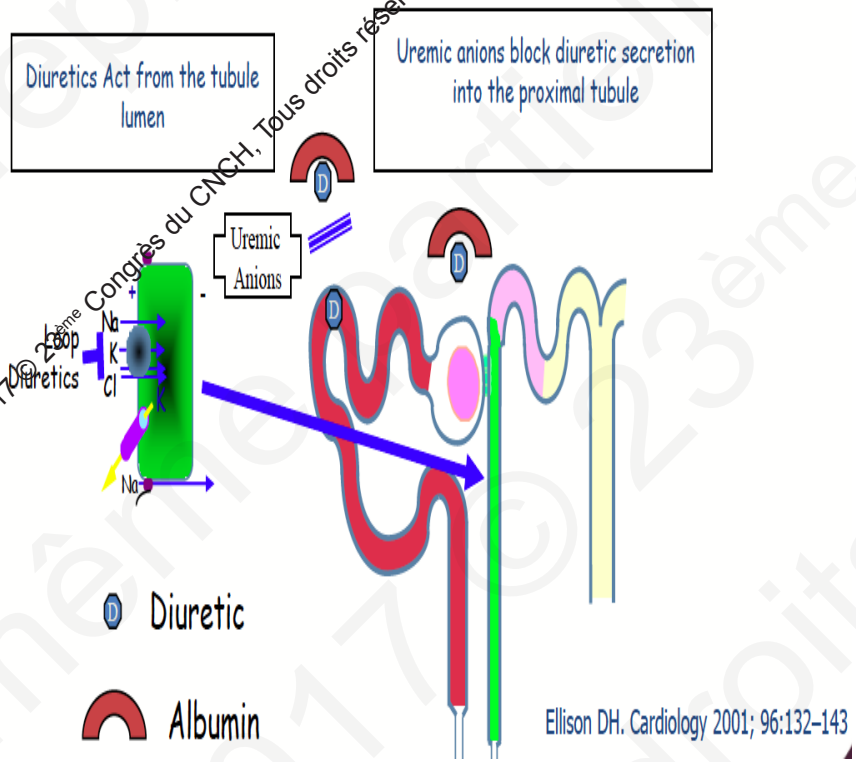
Phénomène de « freinage » et impact de consommation de sodium



Ellison DH. Cardiology 2001; 96:132–143

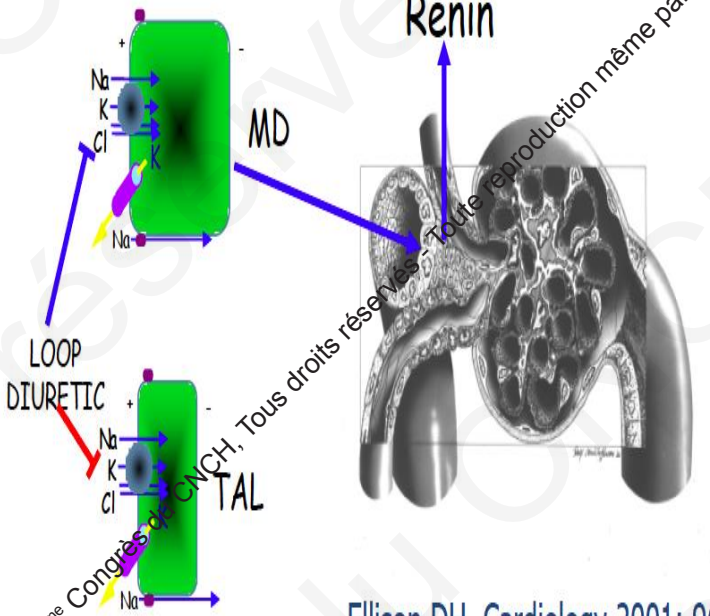
Insuffisance rénale chronique

Diuretic Secretion Is Impaired in CKD



Ellison DH. Cardiology 2001; 96:132–143

Loop Diuretics Stimulate Renin



Ellison DH. Cardiology 2001; 96:132–143

Pharmacocinétique de diurétiques de l'anse

Diurétique de l'anse	Biodisponibilité orale (%)		Elimination (demi-vie), h	
		Sujet normal	Patient avec insuffisance rénale	Patient avec insuffisance cardiaque
Furosemide	10-100	1,5-2	2,8	2,7
Bumetanide	80-100	1	1,6	1,3
Torsemide	80-100	3-4	4-5	6

Dans l'IC:
Congestion hépatique
Oedème intestinal
⬇Flux sanguin intestinal

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Combination decongestion therapy in hospitalized heart failure: loop diuretics, mineralocorticoid receptor antagonists and vasopressin antagonists

Expert Review of Cardiovascular Therapy,2015; 13:7, 799-

Muthiah Vaduganathan, Robert J Mentz, Stephen J Greene, Michele Senni, Naoki Sato, Savina Nodari, Javed Butler & Mihai Gheorghiade

Decongestion strategy	Strengths	Limitations
Loop diuretics	Ease of clinical use	Diuretic resistance
	Non-invasive strategy	Neurohormonal activation
	Operator experience is robust	Electrolyte disturbances
	Early dyspnea relief	Association with worsening renal function May not achieve complete decongestion when used alone
Mineralocorticoid receptor antagonists	Augmented natriuresis	Hyperkalemia
	May spare loop diuretic requirement	Limited provider experience with high doses
	Specific role in right-sided congestion due to reduced hepatic clearance of aldosterone[†]	Empiric data regarding inpatient use is lacking
Vasopressin antagonists	Early and sustained dyspnea relief	Benefits appear to be greatest in those with hyponatremia
	Reduction in body weight and clinical congestion	Cost consideration
	Augmented aquaresis	Non-natriuretic effects
	Improves hyponatremia	
	May spare loop diuretic requirement	
	Low risk of worsening renal function or hemodynamic instability	

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Changes in Brain Natriuretic Peptide Levels and Bioelectrical Impedance Measurements After Treatment With High-Dose Furosemide and Hypertonic Saline Solution Versus High-Dose Furosemide Alone in Refractory Congestive Heart Failure

A Double-Blind Study

Salvatore Paterna, MD,* Pietro Di Pasquale, MD,‖ Gaspare Parrinello, MD,† Ersilia Fornaciari, MD,*
Francesca Di Gaudio, MD,‡ Sergio Fasullo, MD,‖ Marco Giammanco, MD,§ Filippo M. Sarullo, MD,¶

Table 3. Laboratory Parameters of Renal Function at Discharge

	Furosemide Without HSS	Furosemide With HSS	
	Discharge	Discharge	p Value
Patient number	46	48	
Diuresis (ml/24 h)	1,550 ± 355	2,180 ± 545	<0.0001
Urinary Na (mEq/24 h)	119 ± 21	188 ± 25	<0.0001
Urinary K (mEq/24 h)	93 ± 29	87 ± 23	<0.26
BUN (mg/dl)	98 ± 12	65 ± 10	<0.0001
Serum creatinine (mg/dl)	1.97 ± 0.2	1.55 ± 0.05	<0.001
Uric acid (mg/dl)	10.8 ± 4.3	10.3 ± 3.7	<0.54

sults of the Study (30 Days): Side Effects,
s, and Mortality

	Furosemide Without HSS	Furosemide With HSS	p Value
Patient number	46	48	
Gender, F/M	16/30	18/30	
Side effects (tinnitus)	11	0	<0.05
Readmissions	12	0	<0.05
Mortality	3	0	
Sudden death	2	0	

Nouveau traitement natriurétique ou aquarétique

	Molécule	Délai prévu entre admission et début de traitement	Délai réel entre admission et début de traitement	Résultat
ASCEND	Nesiritide	≤ 48h	15,5h	Négative
EVEREST	Tolvaptan	≤ 48h		Effet + transitoire
VERITAS	Tezosentan	≤ 24h	11h	Négative
PROTECT	Rollofylline	≤ 24h		Négative
RELAX-HF	Serelaxine	≤ 16h	7h	Effet + transitoire
TRUE-AHF	Ularitide	≤	6h	Négative

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Ultrafiltration

Loop diuretics versus ultrafiltration in heart failure

Loop diuretics	Ultrafiltration
Increase isotonic urine	Removal of isotonic plasma water: larger natriuresis
Variable and unpredictable response	Predictable and adjustable response; better congestion relief / risks of excessive fluid loss ?
Neurohormonal activation	No neurohormonal activation unless intravascular volume depletion
Development of diuretic resistance	Restoration of diuretic response
Electrolyte abnormalities	No changes in serum K^+ or Mg^+
Intravenous treatment	Possible need of central catheter, vascular access complications, anticoagulation

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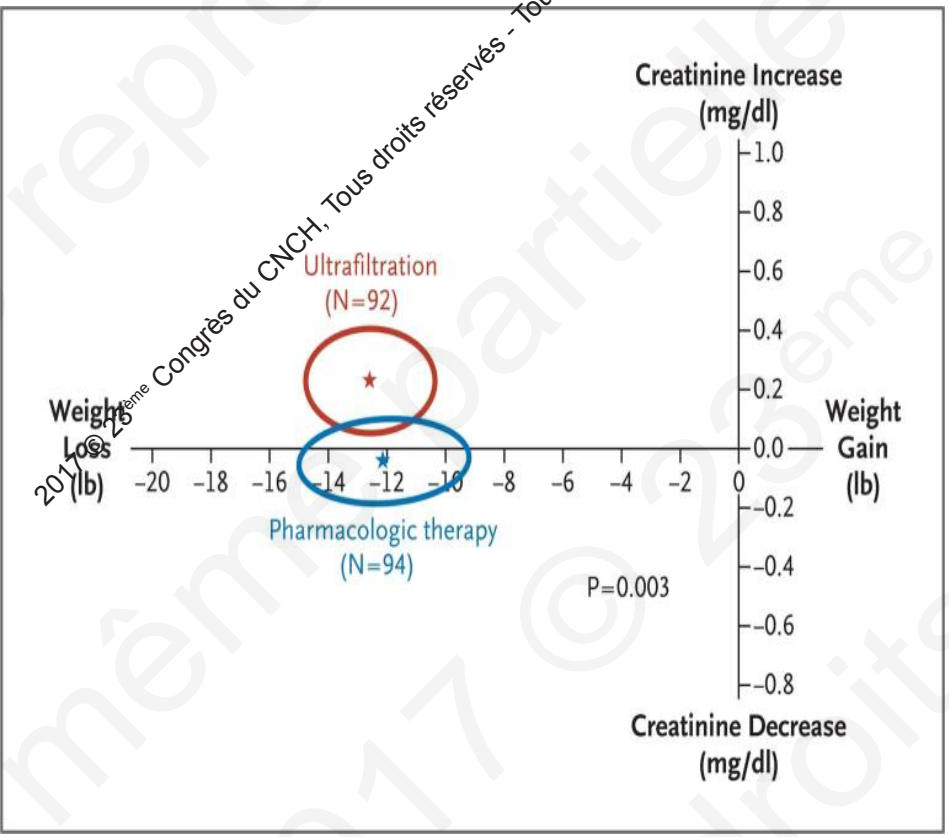
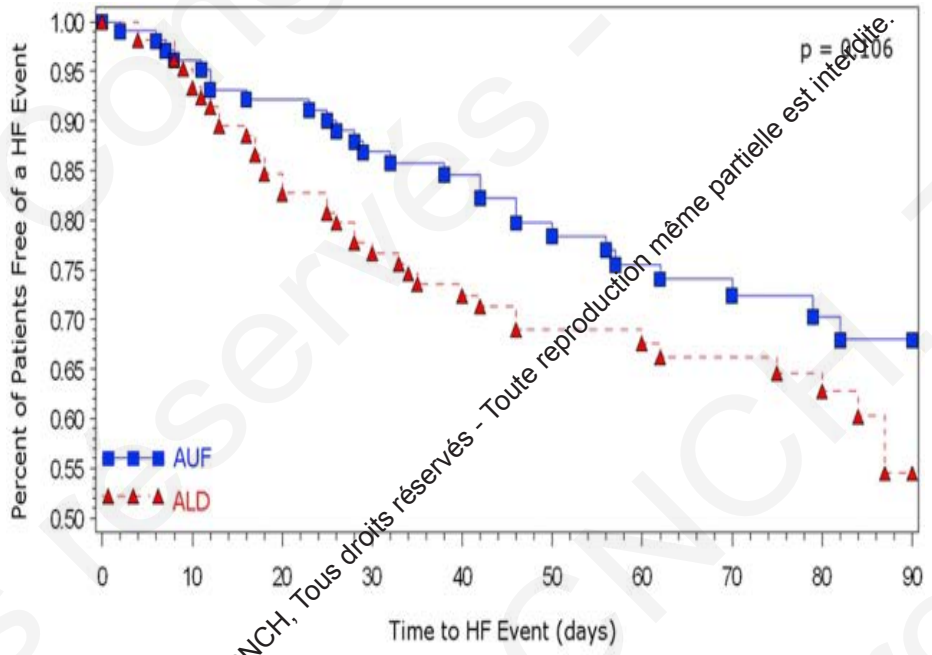
CARRESS-HF Primary endpoint. Changes in Serum Creatinine and Weight at 96 Hours



Bart BA et al. N Engl J Med 2012;367:2296-2304

AVOID-HF. Primary Endpoint: Time to HF Event after Discharge in the adjustable ultrafiltration (AUF) and adjustable loop diuretics (ALD) groups



	Baseline	30 Days	60 Days	90 Days
AUF	105	80	52	19
ALD	108	74	49	15

Maria Rosa Costanzo et al. JACC-HF 2016;4:95-105

pure-HF | Peripheral Ultrafiltration for the Relief from Congestion in Heart Failure

➔ 864 patients in 37 centers in 8 countries

Ultrafiltration group

Peripheral UF

+

low-dose i.v. diuretics

1-7 UF sessions

(6-10h/ day-time session, 1-10 days, OK to skip weekends)

Control group

Guideline-directed medical therapy

incl. i.v. diuretics

Treatment algorithm based on the current dose at index hospitalization, urine output and clinical assessment

Follow-up: 30 and 90-days

Primary endpoint:

→ Heart failure event in 90 days after discharge

→ Cardiovascular death in 90 days after randomization

Recommendations	Class [a]	Level [b]
Ultrafiltration may be considered for patients with refractory congestion, who failed to respond to diuretic-based strategies.	IIb	B
Renal replacement therapy should be considered in patients with refractory volume overload and acute kidney injury.	IIa	C

Ponikowski et al. Eur J Heart Fail. 2016 Aug;18(8):891-975.

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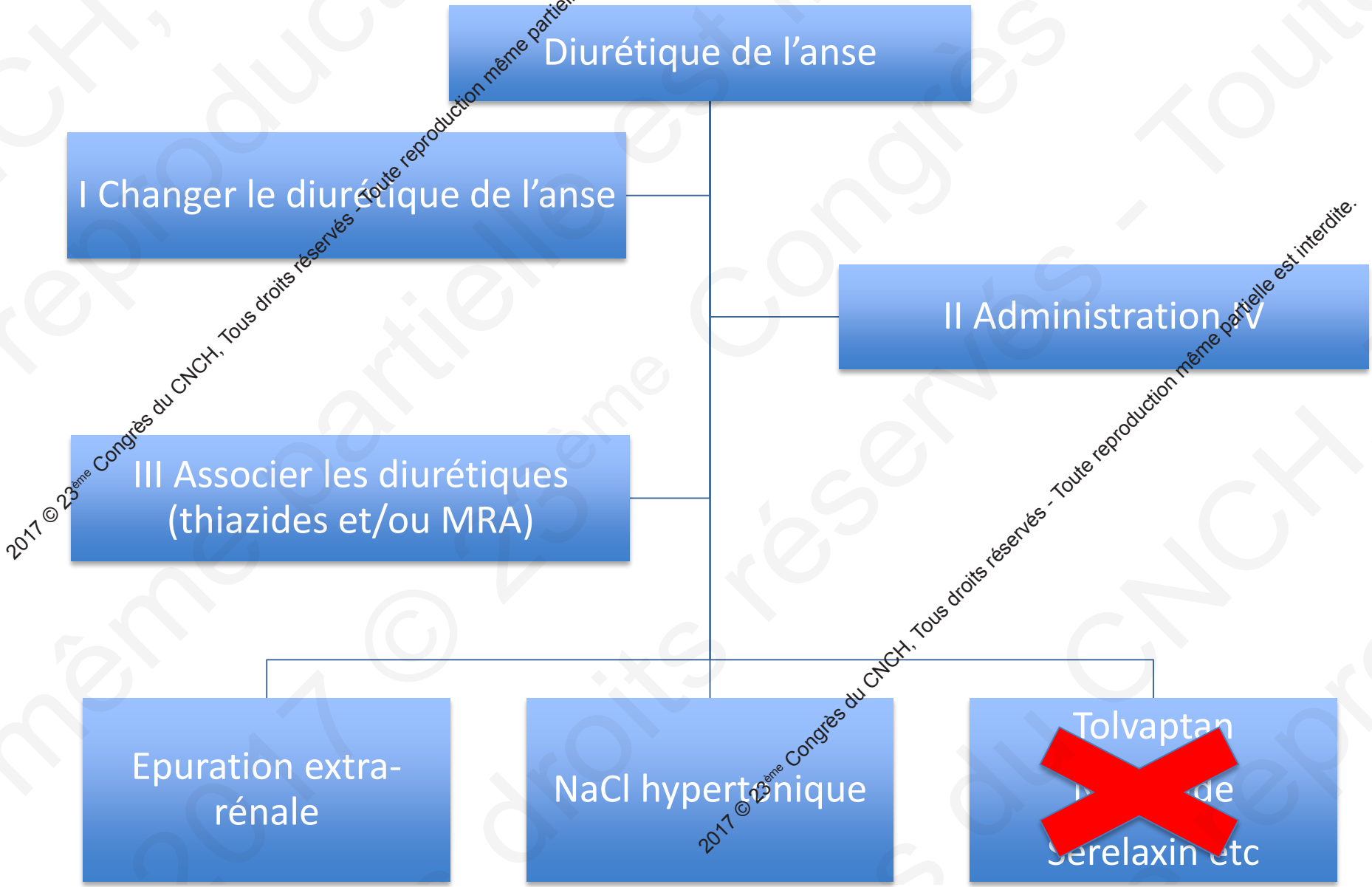
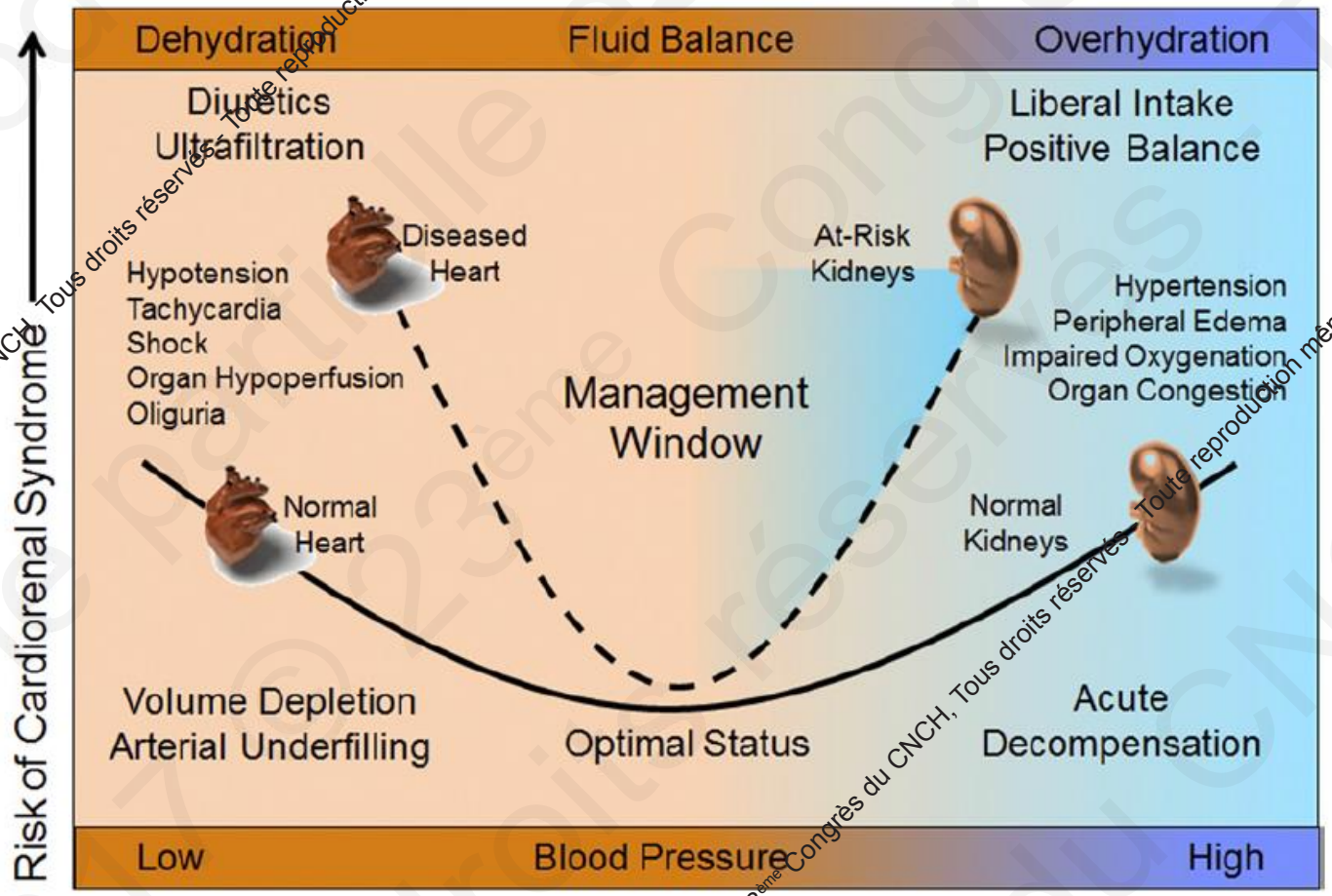
PEC de résistance aux diurétiques





Conclusions

- Il n'a jamais été démontré que les diurétiques réduisent la mortalité ou les hospitalisations chez les patients avec IC, mais ils sont utilisés pour diminuer les symptômes
- Les diurétiques peuvent conduire à l'activation de l'axe neuro-hormonal et devraient donc toujours être utilisés en combinaison avec des médicaments connus pour bloquer cette voie (ACE-I, beta bloqueurs, antagonistes de l'aldostérone)
- Le diurétique de premier choix est le diurétique de l'anse
- En cas d'œdème intestinal, il faut privilégier le diurétique avec la plus grande bio-disponibilité ou passer à l'administration IV
- Il n'y a pas de différence clinique entre l'utilisation de bolus versus perfusion continue

Conclusions

- Résistance aux diurétiques: diminution insuffisante des œdèmes malgré dose maximale
- Stratégie clé pour surmonter la résistance aux diurétiques : combinaison de 2 types de diurétiques (synergie diurétique).
- Ultrafiltration : alternative assurant une meilleure extraction du sodium sans activation neuro-hormonale excessive