PLACE DES DIURÉTIQUES DANS L'INSUFFISANCE CARDIAQUE AIGUE

Pr Nebiha BORSALI FALFOUL
Urgences la Rabta, Tunis

ATR, novembre 2014

IC : DÉFINITION

- Incapacité du cœur à assurer un débit cardiaque suffisant aux besoins métaboliques des tissus.
- Syndrome complexe initié par une dysfonction cardiaque associant :
  - Une inadéquation des conditions de charge du cœur
  - Des phénomènes congestifs
  - Des déficits énergétiques des tissus
  - Une mise en jeu excessive des mécanismes de régulation

DÉCOMPENSATION AIGUE D'IC

- ICA est définie par l'installation rapide ou progressive de **symptômes d'IC** indiquant **l'hospitalisation** de mécanismes physiopathologiques divers.

- Insuffisance cardiaque aiguë est une affection fréquente associée à une morbi-mortalité dans le monde*.

*Munger MA. Pharmacotherapy 2006;26:131S–8S.

ÉPIDÉMIOLOGIE

- 5 millions d'Américains en IC
- ≈ un million d'hospitalisations par an aux États-Unis*.
- La principale cause de l'hospitalisation HF est la congestion symptomatique.
- 50% des patients réhospitalisées dans les 6 mois
- 25% à 35% de mortalité à 1 an.
- ADHERE : Près de 50% des patients ont encore des symptômes de congestion à la sortie**.

*Lloyd-Jones, *et al.Circulation* **2009**, *119*, 480–486.
Adams, K.F, et al. ADHERE. *Am. Heart J.* **2005, *149*, 209–216

Les mécanismes adaptatifs

PAm = Qc x RVP

VES x Fc x RVP

Rétention hydro sodée++
Dilatation du VG

Tachycardie

Vasoconstriction

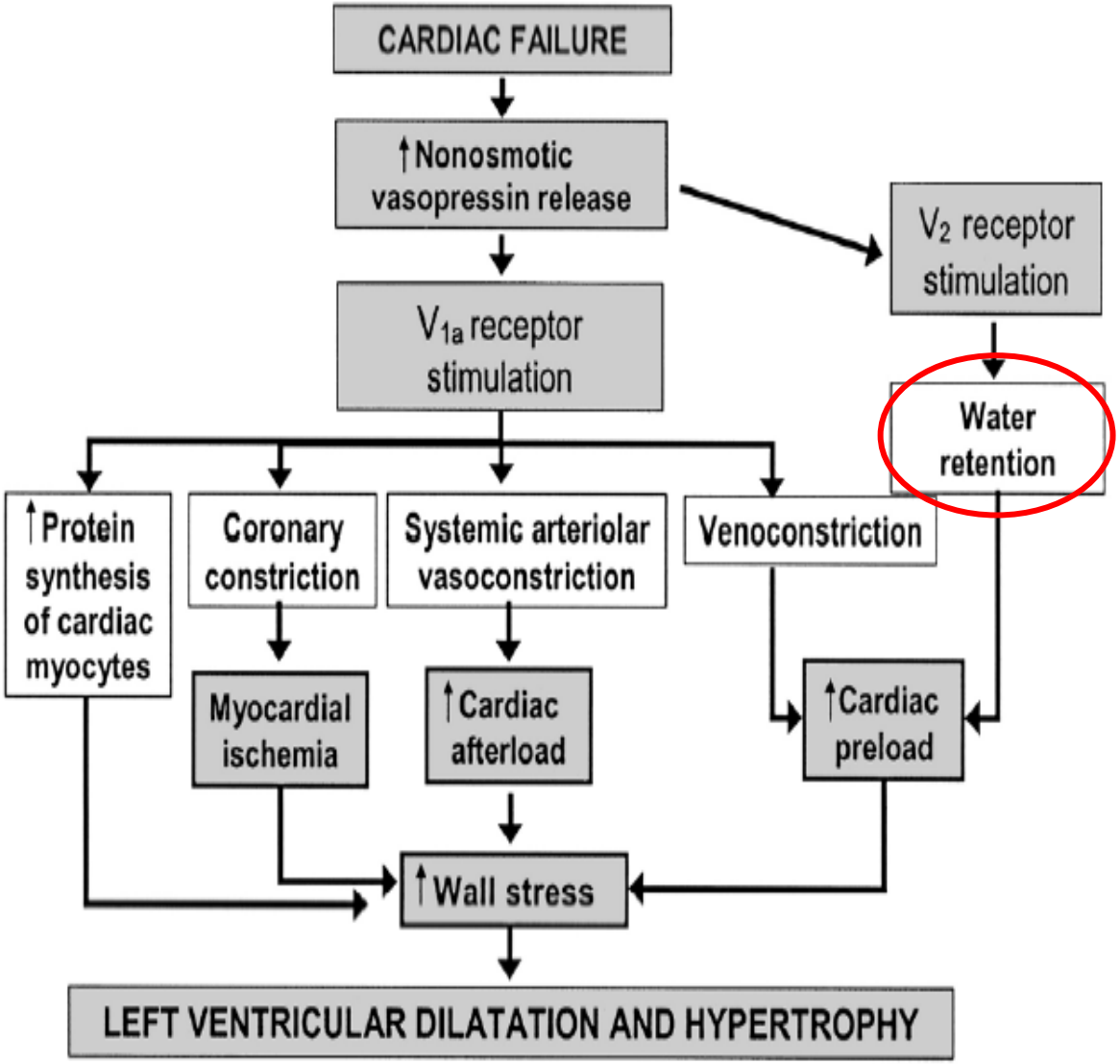
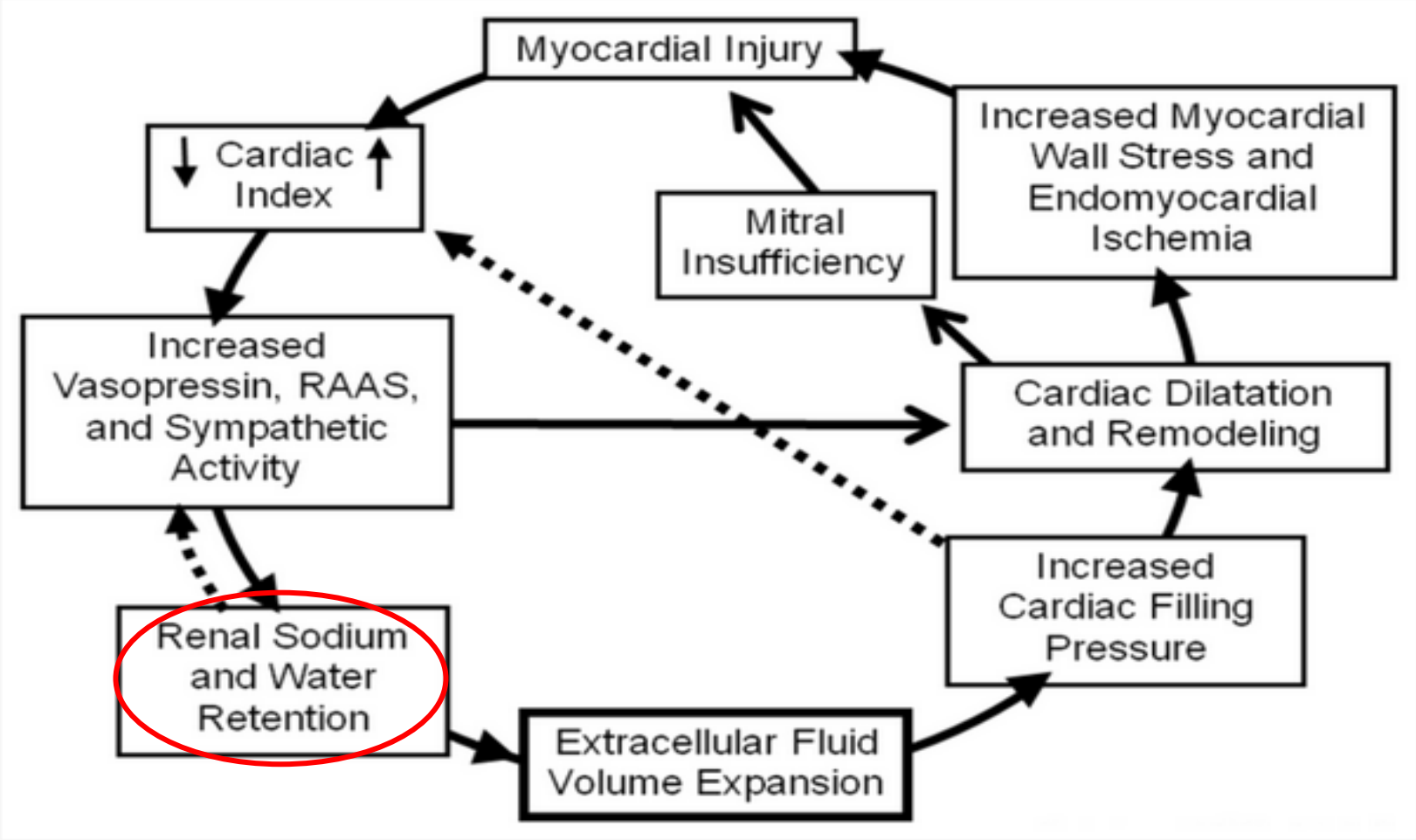


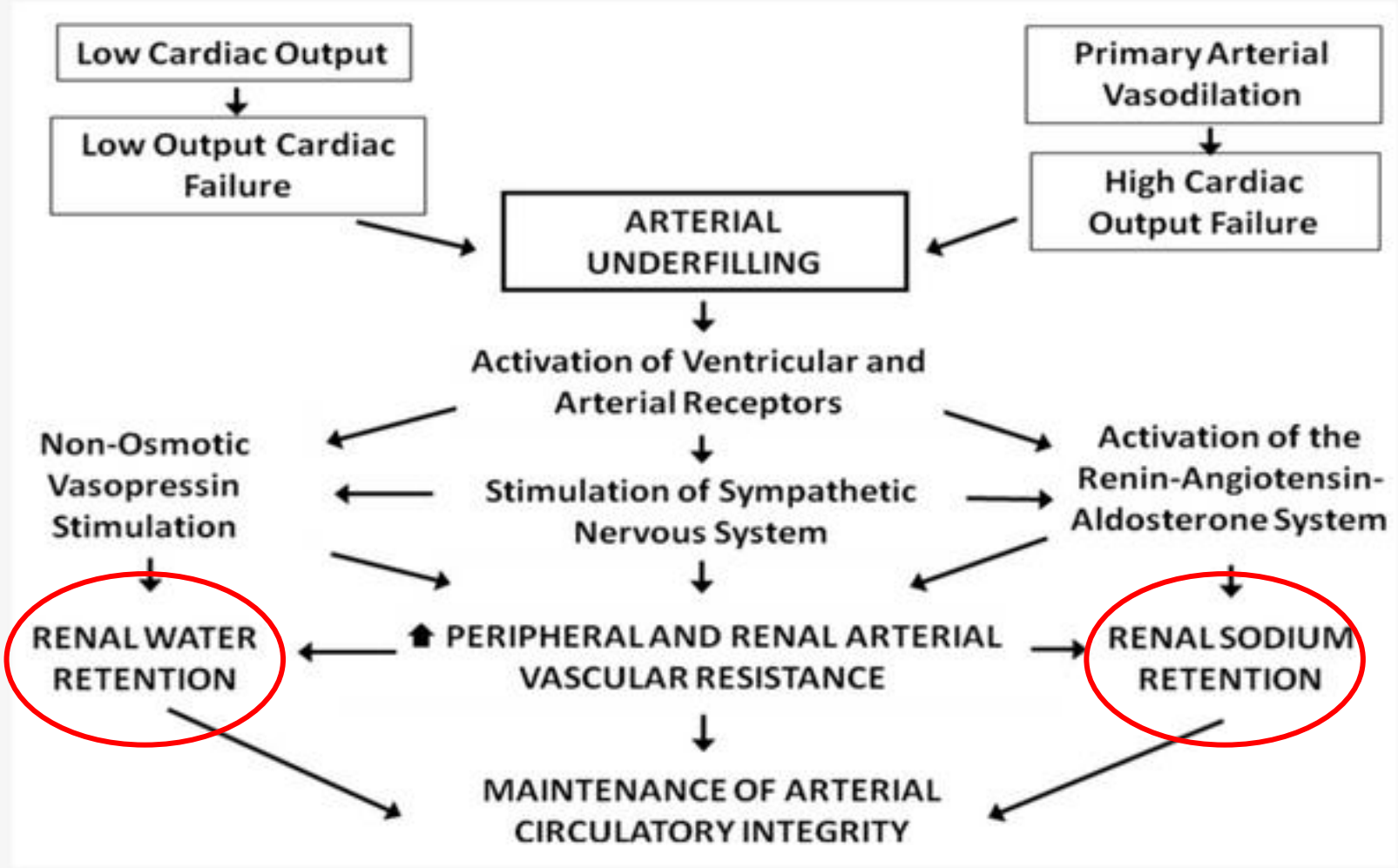
Figure 3 Potential effects of vasopressin on V_1 and V_2 receptors, which can worsen cardiac function by increasing cardiac preload and

Figure 1. Vicious cycle of chronic heart failure. Reproduced from [8] with permission.



Schrier, R.W. *J. Am. Coll. Cardiol.* **2006**, *47*, 1–8

Figure 2. Pathogenesis of low and high cardiac output heart failure. Reproduced from [12] with permission.



*Dmitry Shchekochikhin , Fawaz Al Ammary , JoAnn Lindenfeld and Robert Schrier. *Pharmaceuticals* **2013**, *6*(7), 851-66. **Schrier, R.W. Body fluid volume regulation in health and disease: A unifying hypothesis. *Ann. Intern. Med.* **1990**, *113*, 155–9.

Insuffisance cardiaque : cercles vicieux

Dysfonction Ventriculaire

Baisse de la fonction pompe

Mise en jeu de mécanismes inadaptés

Stimulation neuro-hormonale excessive
Charge sodique et calcique
Stress oxydatif
Modification du génome (programmes fœtaux)
Apoptose

Altérations physiopathologiques

Diminution du débit cardiaque
Diminution de la PA
Hypoperfusion tissulaire
Pressions auriculaires excessives

Tentative d'adaptation physiologique

Régulation de la PA
Régulation de la volémie
Stimulation sympathique
Baroréflexe
Hormones natriurétiques

6. Schrier RW, Abraham WT. Hormones and hemodynamics in heart failure. *N Engl J Med.* 1999;341:577–585.

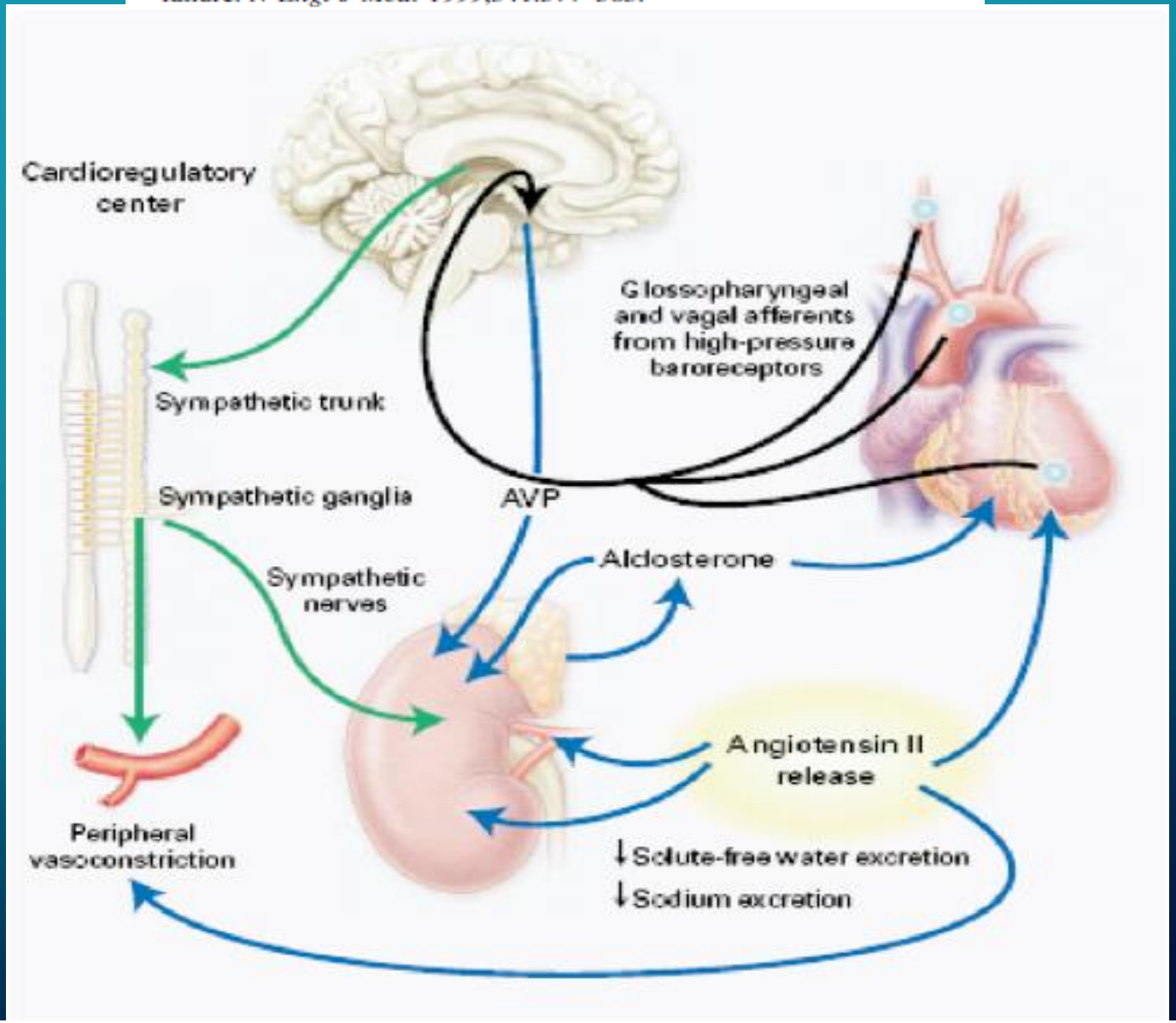
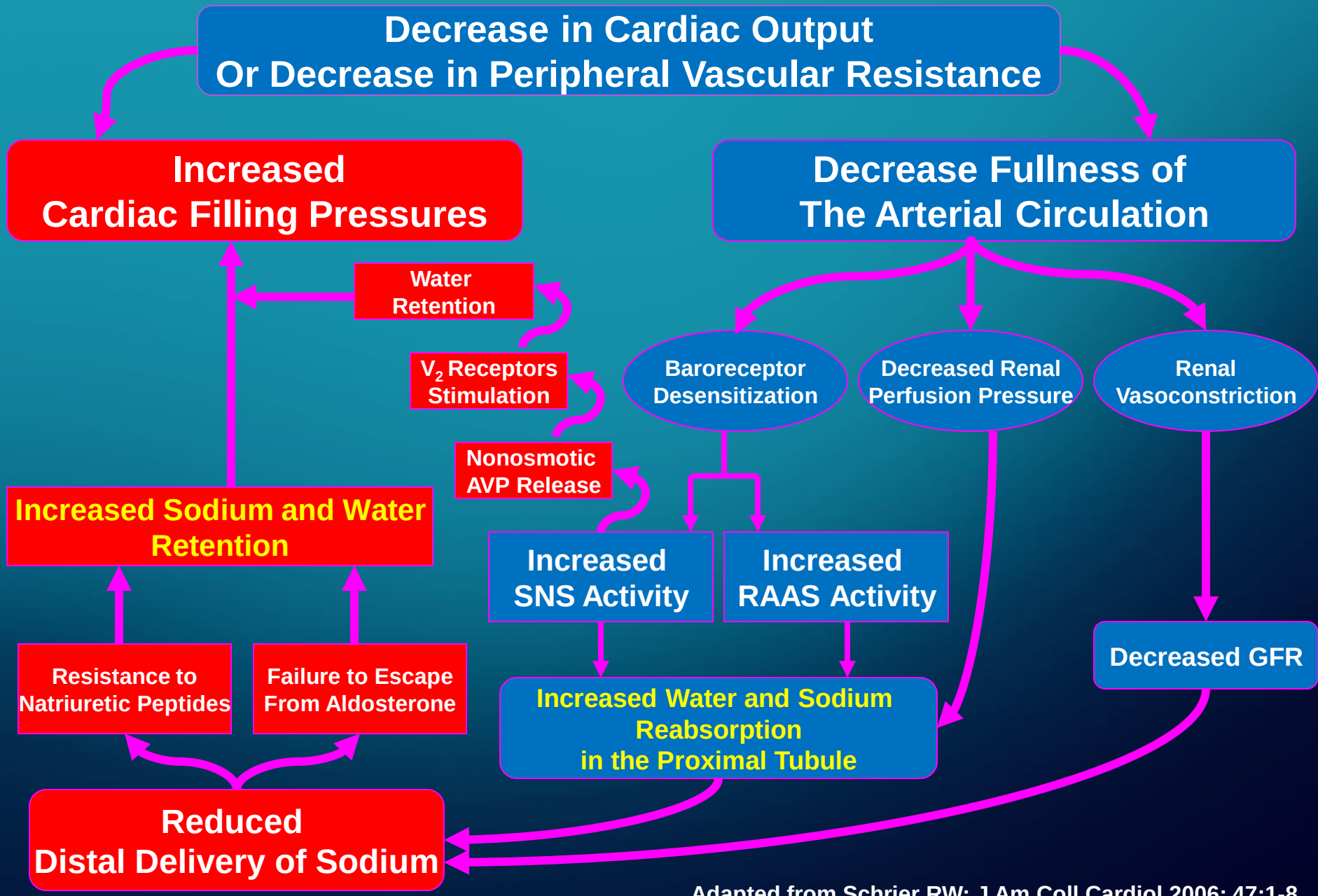


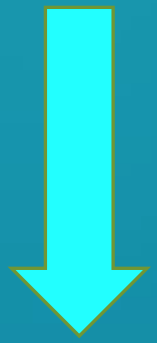
Figure 2 Unloading of high-pressure baroreceptors (*blue circles*) in the left ventricle, carotid sinus, and aortic arch generates afferent signals (*black arrows*) that stimulate cardioregulatory centers in the brain, resulting in the activation of efferent pathways in the sympathetic nervous system (*green arrows*). The sympathetic nervous system appears to be the primary integrator of the neurohumoral vasoconstrictor response to arterial underfilling. Activation of renal sympathetic nerves stimulates renin release, thus activating the renin-angiotensin-aldosterone system. Concomitantly, sympathetic stimulation of the supraoptic and paraventricular nuclei in the hypothalamus results in the nonosmotic synthesis and release of arginine vasopressin (AVP). Sympathetic activation also causes peripheral and renal vasoconstriction, as does angiotensin II. Angiotensin II also stimulates the release of aldosterone from the adrenal gland and increases tubular sodium reabsorption in addition to remodeling cardiac myocytes. Aldosterone enhances cardiac fibrosis and increases the reabsorption of sodium and the secretion of potassium and hydrogen ions in the collecting duct. The blue arrows designate circulating hormones. (Reprinted with permission from *N Engl J Med.*[6])

MECHANISMS OF SODIUM AND WATER RETENTION IN HEART FAILURE



Adapted from Schrier RW: J Am Coll Cardiol 2006; 47:1-8

Dysfonction cardiaque



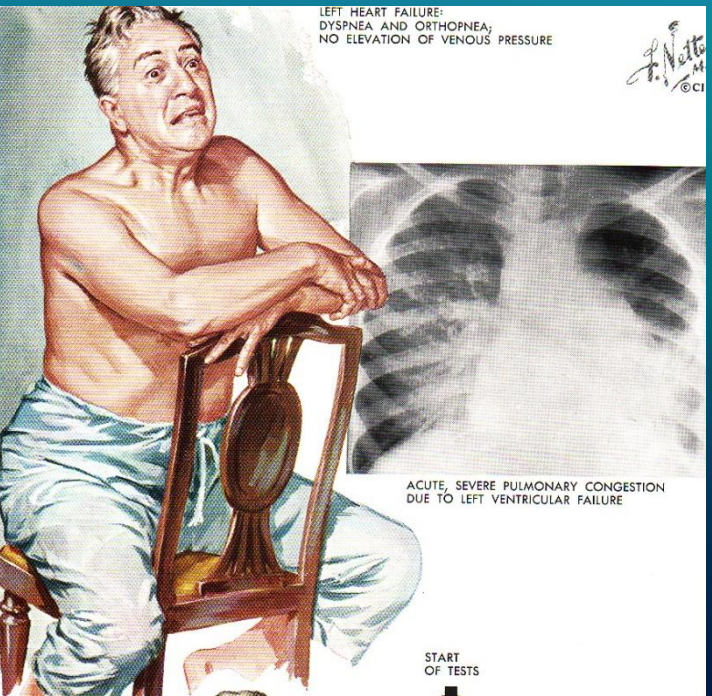
Mécanismes adaptatifs
délétères

Cibles thérapeutiques

Rétention d'eau et de sodium: 2 tableaux cliniques

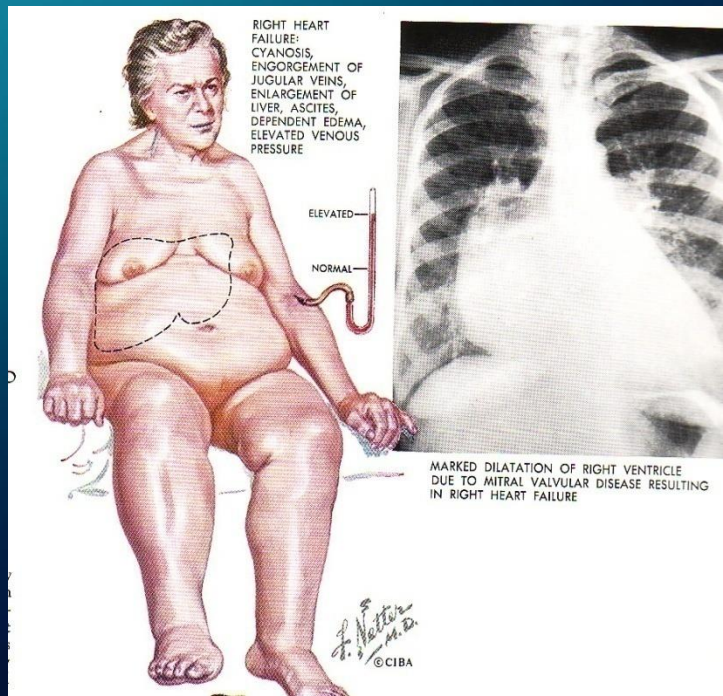
⇒ Symptômes de congestion pulmonaire : dyspnée, orthopnée, dyspnée paroxystique nocturne

IVG : OAP



⇒ la congestion veineuse systémique : œdème, ascite, hépatomégalie.

IC Drt



Le traitement diurétique est la pierre angulaire du traitement de l'insuffisance cardiaque aigue.

≈ 90% des patients hospitalisés pour décompensation cardiaque aiguë ont reçu un traitement diurétique par voie intraveineuse*.

* Peacock WF, et al. the ADHERE registry. Cardiology .9–113:12;2009.

Les diuretiques

Les diurétiques inhibent la rétention d'eau et de sodium

⇒Réduisent la volémie

⇒Réduisent la précharge cardiaque

⇒Améliorent l'efficacité de la pompe cardiaque

⇒ ↑ du débit cardiaque

⇒ réduisent l'œdème dû à l'insuffisance cardiaque

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Contents lists available at ScienceDirect

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journal homepage: www.jccjournal.org

Renal/Coagulation

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], Ka-Wai Tam, MD, MSc [f,g,h,i,*]

[a] Division of Nephrology, Department of Internal Medicine, Taipei Medical University–Shuang Ho Hospital, Taipei, Taiwan
[b] Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan
[c] School of Respiratory Therapy, Taipei Medical University, Taipei, Taiwan
[d] Department of Thoracic Internal Medicine, Taipei Medical University–Shuang Ho Hospital, Taipei, Taiwan

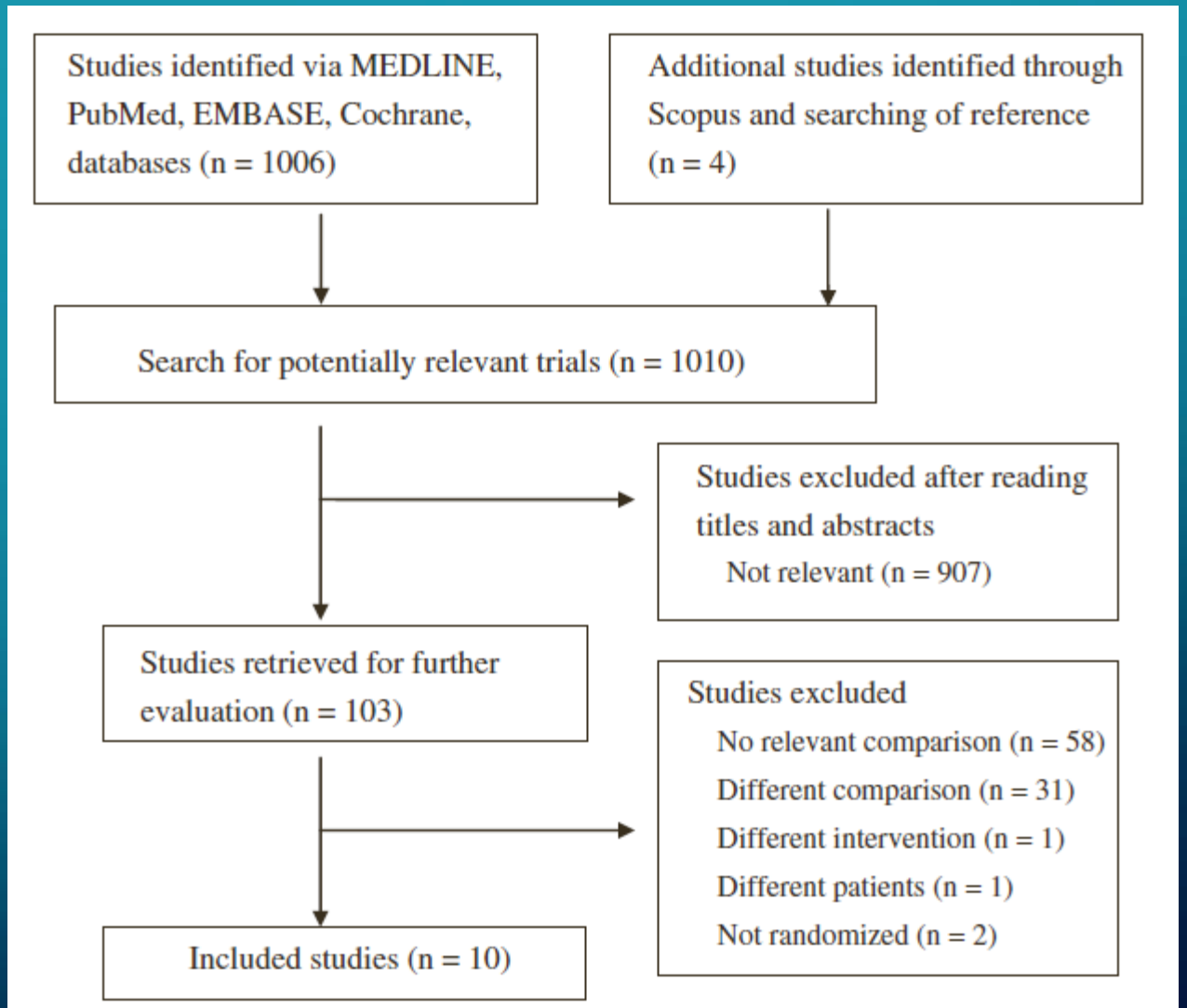

Fig. 1. Flowchart for selection of studies.

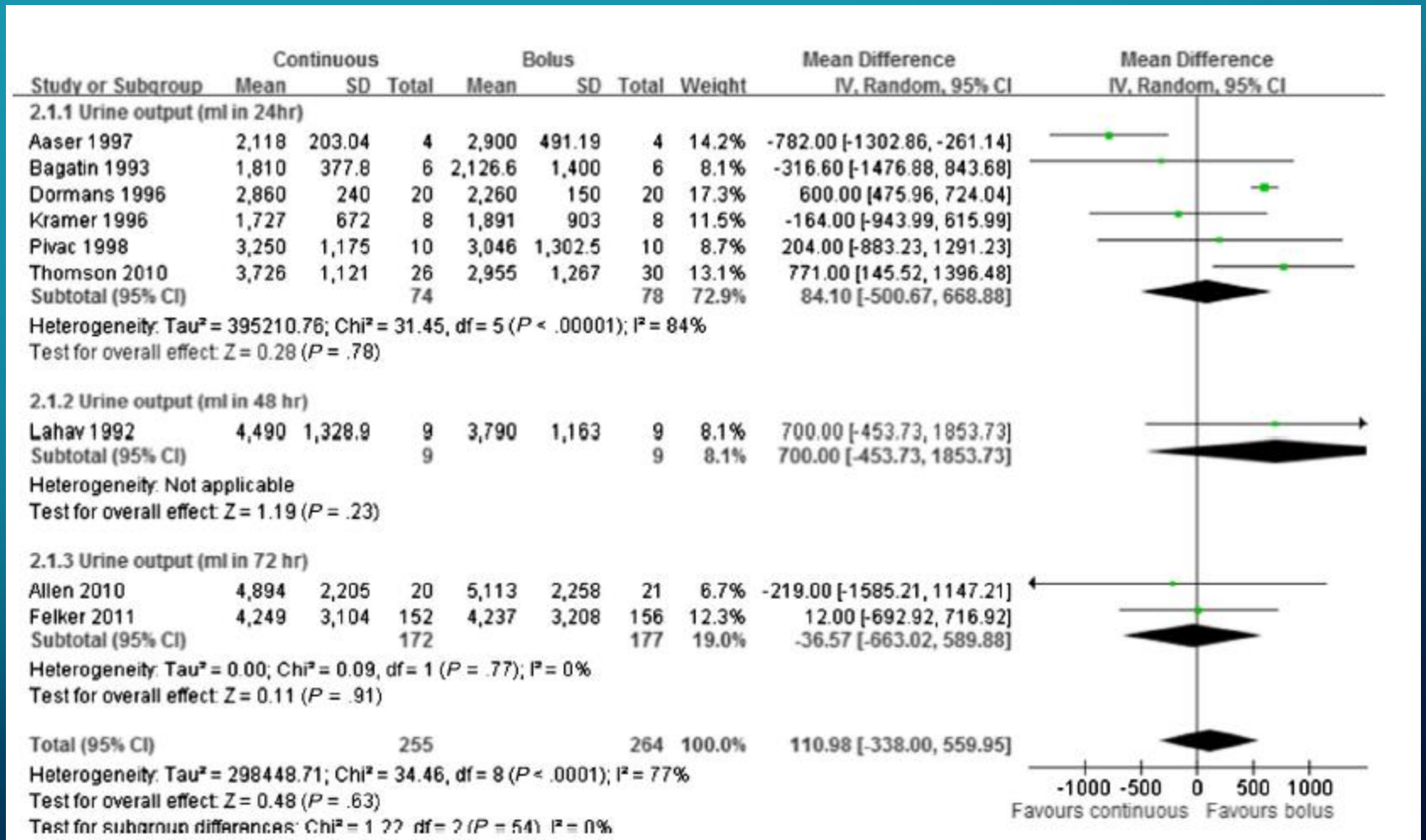

Fig. 2. Forest plot of comparison: continuous vs bolus. Outcome: urine output.

Study or Subgroup	Mean
Allen 2010	2.66
Felker 2011	3.67
Thomson 2010	6.8
Total (95% CI)	

Heterogeneity: Tau² = 0.00;
Test for overall effect: Z = 2.0

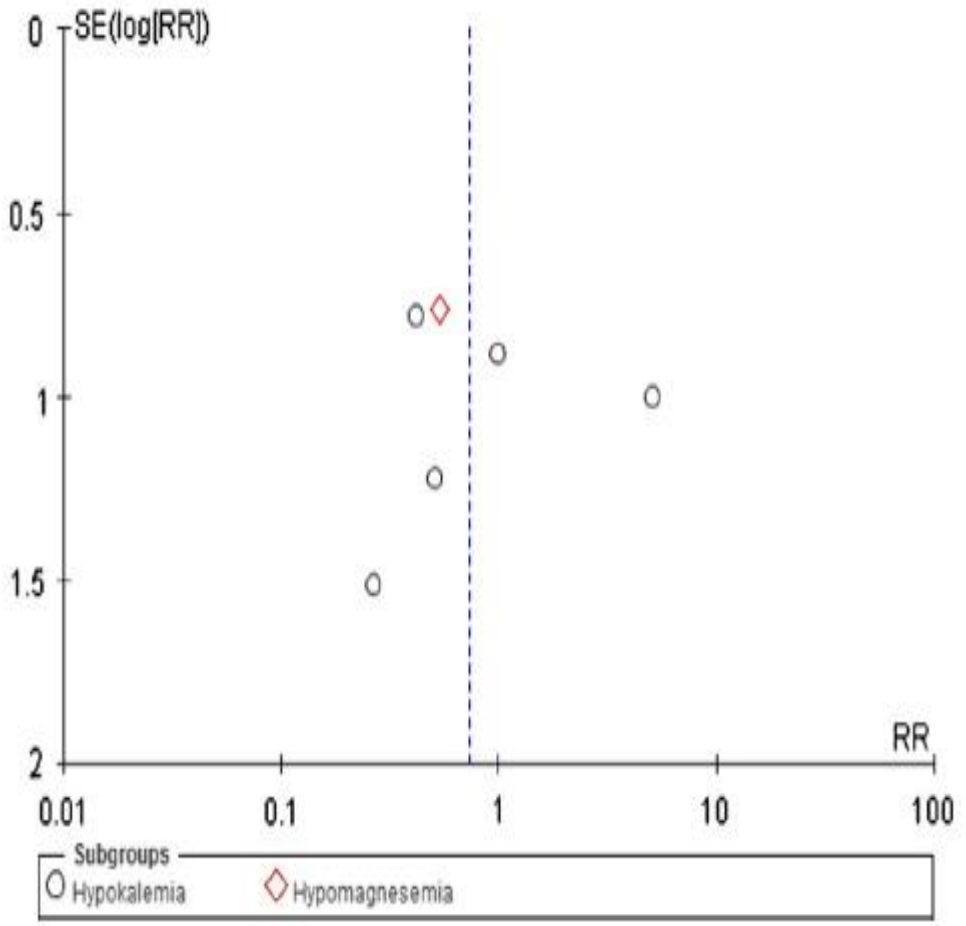

Fig. 5. Funnel plot for the bolus and continuous groups in the incidence of electrolyte imbalance. No asymmetry was found as indicated by the *P* value of Egger test.

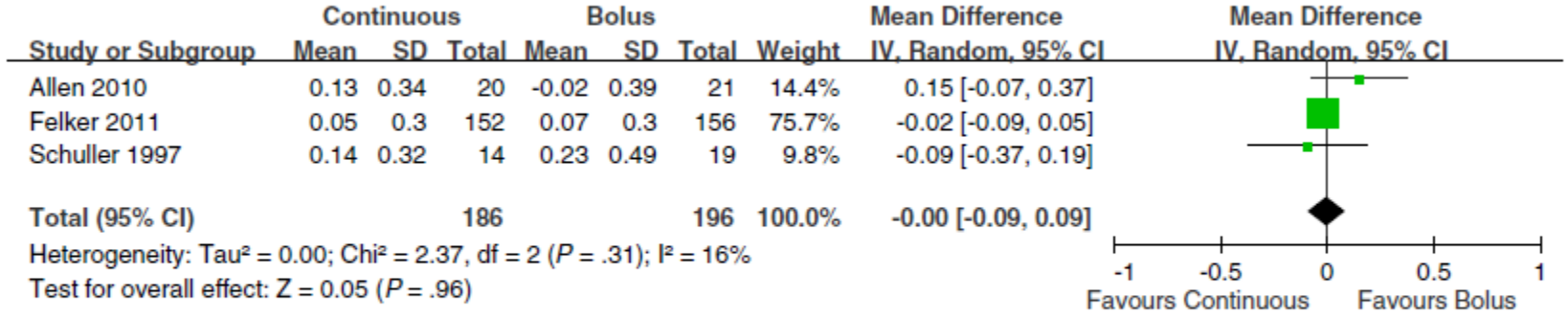

Fig. 6. Forest plot of comparison: continuous vs bolus. Outcome: incidence of increase in creatinine.

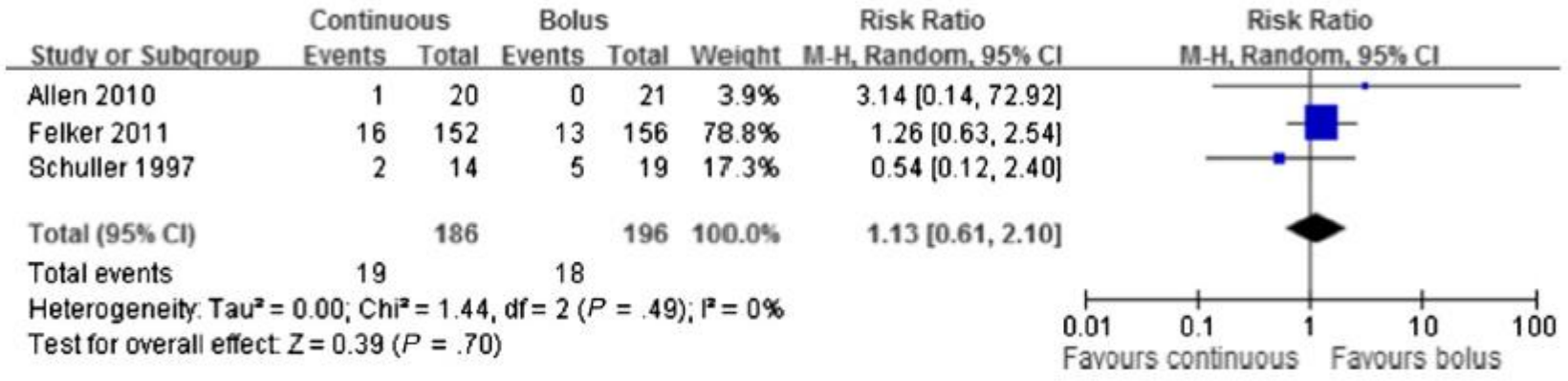

Fig. 8. Forest plot of comparison: continuous vs bolus. Outcome: all-cause mortality.

Despite the improvements made in the current review, our research was subject to certain limitations. Population characteristics, small patient numbers, crossover designs without an adequate washout period, differing diuretic schedules and dosages, use of concomitant drugs, varied outcome analysis, and the methodological weakness inherent in some studies we reviewed may have resulted in a somewhat speculative interpretation of our subgroup analysis. These differences will have contributed to the observed heterogeneity. In addition, the efficiency of diuresis as measured by urine output per dose of diuretic appears to be more representative than urine amount or body weight along. There is a wide range of publication dates in the included studies. The concomitant use of angiotensin-converting enzyme inhibitors and β-blockers might alter both the efficacy and the toxicity of diuretics in individual patients.

Circulation
Heart Failure

American Heart Association.

Loop Diuretics in Acute Decompensated Heart Failure: Necessary? Evil? A Necessary Evil?

G. Michael Felker, Christopher M. O'Connor and Eugene Braunwald
for the Heart Failure Clinical Research Network Investigators

Circ Heart Fail. 2009;2:56-62
doi: 10.1161/CIRCHEARTFAILURE.108.821785
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Table 1. Observational Studies of Diuretics and Outcomes in Heart Failure

Study	Population	N	Comparison	End Point	Risk	95% CI
			diuretics	(change of 0.3 mg/dl)	of furosemide	
Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization	Advanced HF in-patients	395	Dose of IV loop diuretics	Mortality	1.15 per doubling of dose	1.025–1.28
			doses			
Mielniczuk et al[27]	Chronic HF	183	Oral diuretic dose	HF events	1.53 for dose >80 mg	0.58–4.03

Études rétrospectives,
population hétérogène
Dysfonction VG avec ou sans IC
IC Ch

Am J Cardiol. 1997 Aug 15;80(4):519-22.

Association between diuretic use, clinical response, and death in acute heart failure.

Philbin EF[1], Cotto M, Rocco TA Jr, Jenkins PL.

Author information

Abstract

Because the impact of diuretic use on mortality in acute congestive heart failure (CHF) is not known, we examined the association between drug use, fluid balance, and death among 1,150 patients hospitalized for evaluation and treatment of CHF. After adjusting for other relevant intergroup differences, we observed that less net weight loss and a greater number of intravenous drug doses retained significant predictive value for death, suggesting that more frequent diuretic dosing or diuretic resistance may be related to mortality in acute CHF.

PMID: 9285672 [PubMed - indexed for MEDLINE]

Am Heart J. 2004 Feb;147(2):331-8.

Relationship between heart failure treatment and development of worsening renal function among hospitalized patients.

Butler J[1], Forman DE, Abraham WT, Gottlieb SS, Loh E, Massie BM, O'Connor CM, Rich MW, Stevenson LW, Wang Y, Young JB, Krumholz HM.

⊕ Author information

Abstract

BACKGROUND: Among patients who are hospitalized with heart failure (HF), worsening renal function (WRF) is associated with worse outcomes. Whether treatment for HF contributes to WRF is unknown. In this study, we sought to assess whether acute treatment for patients who were hospitalized with HF contributes to WRF.

METHODS: Data were collected in a nested case-control study on 382 subjects who were hospitalized with HF (191 patients with WRF, defined as a rise in serum creatinine level >26.5 micromol/L [0.3 mg/dL], and 191 control subjects). The association of medications, fluid intake/output, and weight with WRF was assessed.

RESULTS: Calcium channel blocker (CCB) use and loop diuretic doses were higher in patients on the day before WRF (25% vs 10% for CCB; 199 +/- 195 mg vs 143 +/- 119 mg for loop diuretics; both P <.05). There were no significant differences in the fluid intake/output or weight changes in the 2 groups. Angiotensin-converting enzyme (ACE) inhibitor use was not associated with WRF. Other predictors of WRF included elevated creatinine level at admission, uncontrolled hypertension, and history of HF or diabetes mellitus. Higher hematocrit levels were associated with a lower risk. Vasodilator use was higher among patients on the day before WRF (46% vs 35%, P <.05), but was not an independent predictor in the multivariable analysis.

CONCLUSIONS: Several medical strategies, including the use of CCBs and a higher dose of loop diuretics, but not ACE inhibitors, were associated with a higher risk of WRF. Although assessment of inhospital diuresis was limited, WRF could not be explained by greater fluid loss in these patients. Determining whether these interventions are responsible for WRF or are markers of higher risk requires further investigation.

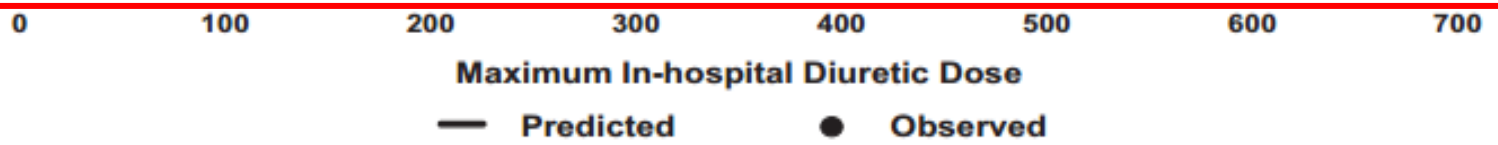

Figure 1. Relationship between maximum in-hospital diuretic dose and mortality in the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness study. Reprinted with permission from Reference 23.

Butler J, et al. Am Heart J. 2004;147:331–338.

Table 2. Randomized Trials of Bolus Versus Continuous Infusion of Diuretics in Heart Failure

Study	N	Design	Intervention	Duration	End Point(s)	Findings
Aaser et al[42]	8	Randomized, cross-over, unblinded	Continuous infusion vs BID IV bolus	24 hours	Urine output	Bolus better
Dormans et al[44]	20	Randomized, cross-over, unblinded	Continuous infusion vs single IV bolus	24 hours	Urine output	Infusion better
Kramer et al[45]	8	Randomized, cross-over, unblinded	Continuous infusion vs single IV bolus	24 hours	Urine output	No difference
Lahav et al[46]	9	Randomized, cross-over, unblinded	Continuous infusion vs Q8 bolus	48 hours	Urine output	Infusion better (trend)
Licata et al[48]	107	Randomized, single blind	Continuous infusion+hypertonic saline vs Q12 bolus	6–12 days	Urine output at 24 hours LOS Mortality	Infusion better on all end points
Pivac et al[43]	20	Randomized, single blind, crossover	Q12 4-hour infusion vs Q12 bolus	24 hours	Urine output	Infusion better
Schuller et al[47]	33	Randomized, unblinded	Continuous infusion vs bolus	72 hours	Mortality	No difference

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morbidity, and costs. Although loop diuretics are the mainstay of therapy for ADHF, much uncertainty remains about the safety and efficacy of various doses as well as means of administration. Although observational data can provide clues to the safety and efficacy of therapies, a true assessment of the risks and benefits can only be achieved with an appropriately powered, prospective randomized clinical trial.

Defining the optimal strategy for diuretic administration will therefore not only impact current clinical care but will aid in the development and evaluation of new ADHF therapies moving forward.

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PMCID: PMC3412356
NIHMSID: NIHMS278788

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., Steven E. McNulty, M.S., Eric J. Velazquez, M.D., Abdallah G. Kfoury, M.D., Horng H. Chen, M.B., B.Ch., Michael M. Givertz, M.D., Marc J. Semigran, M.D., Bradley A. Bart, M.D., Alice M. Mascette, M.D., Eugene Braunwald, M.D., and Christopher M. O'Connor, M.D., for the NHLBI Heart Failure Clinical Research Network*

N Engl J Med. 2011 Mar 3;364(9):797-805. doi: 10.1056/NEJMoa1005419.

Diuretic strategies in patients with acute decompensated heart failure.

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Collaborators (79)

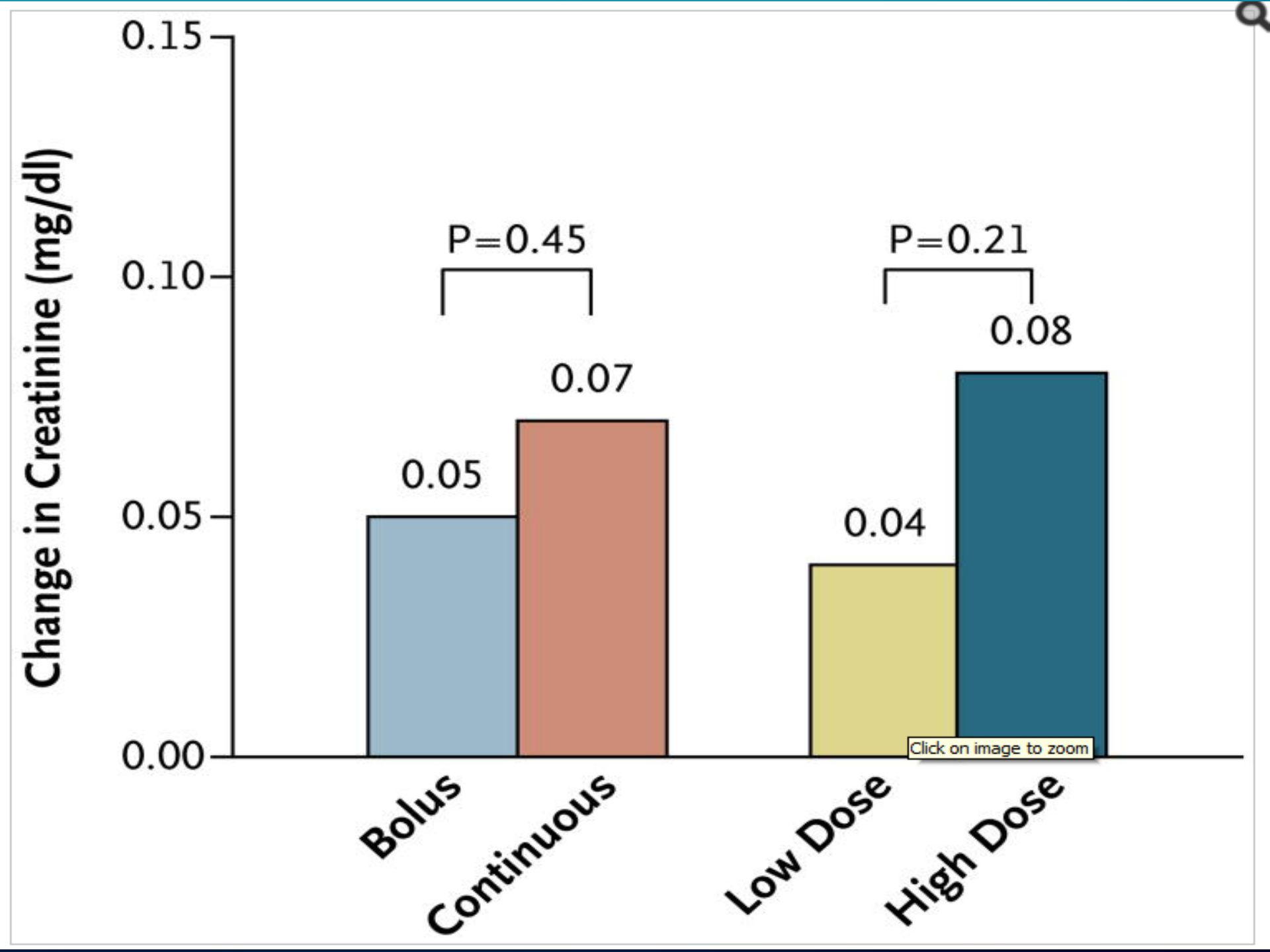
METHODS: In a prospective, double-blind, randomized trial, we assigned 308 patients with acute decompensated heart failure to receive furosemide administered intravenously by means of either a bolus every 12 hours or continuous infusion and at either a low dose (equivalent to the patient's

METHODS: In a prospective, double-blind, randomized trial, we assigned 308 patients with acute decompensated heart failure to receive furosemide administered intravenously by means of either a bolus every 12 hours or continuous infusion and at either a low dose (equivalent to the patient's previous oral dose) or a high dose (2.5 times the previous oral dose). The protocol allowed specified dose adjustments after 48 hours. The coprimary end points were patients' global assessment of symptoms, quantified as the area under the curve (AUC) of the score on a visual-analogue scale over the course of 72 hours, and the change in the serum creatinine level from baseline to 72 hours.

RESULTS: In the comparison of bolus with continuous infusion, there was no significant difference in patients' global assessment of symptoms (mean AUC, 4236±1440 and 4373±1404, respectively; P=0.47) or in the mean change in the creatinine level (0.05±0.3 mg per deciliter [4.4±26.5 µmol per liter] and 0.07±0.3 mg per deciliter [6.2±26.5 µmol per liter], respectively; P=0.45). In the comparison of the high-dose strategy with the low-dose strategy, there was a nonsignificant trend toward greater improvement in patients' global assessment of symptoms in the high-dose group (mean AUC, 4430±1401 vs. 4171±1436; P=0.06). There was no significant difference between these groups in the mean change in the creatinine level (0.08±0.3 mg per deciliter [7.1±26.5 µmol per liter] with the high-dose strategy and 0.04±0.3 mg per deciliter [3.5±26.5 µmol per liter] with the low-dose strategy, P=0.21). The high-dose strategy was associated with greater diuresis and more favorable outcomes in some secondary measures but also with transient worsening of renal function.

CONCLUSIONS: Among patients with acute decompensated heart failure, there were no significant differences in patients' global assessment of symptoms or in the change in renal function when diuretic therapy was administered by bolus as compared with continuous infusion or at a high dose as compared with a low dose. (Funded by the National Heart, Lung, and Blood Institute; ClinicalTrials.gov number, NCT00577135.).

Comment in


0.15
0.10
0.05
0.00
Change in Creatinine (mg/dl)
P=0.45
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0.05
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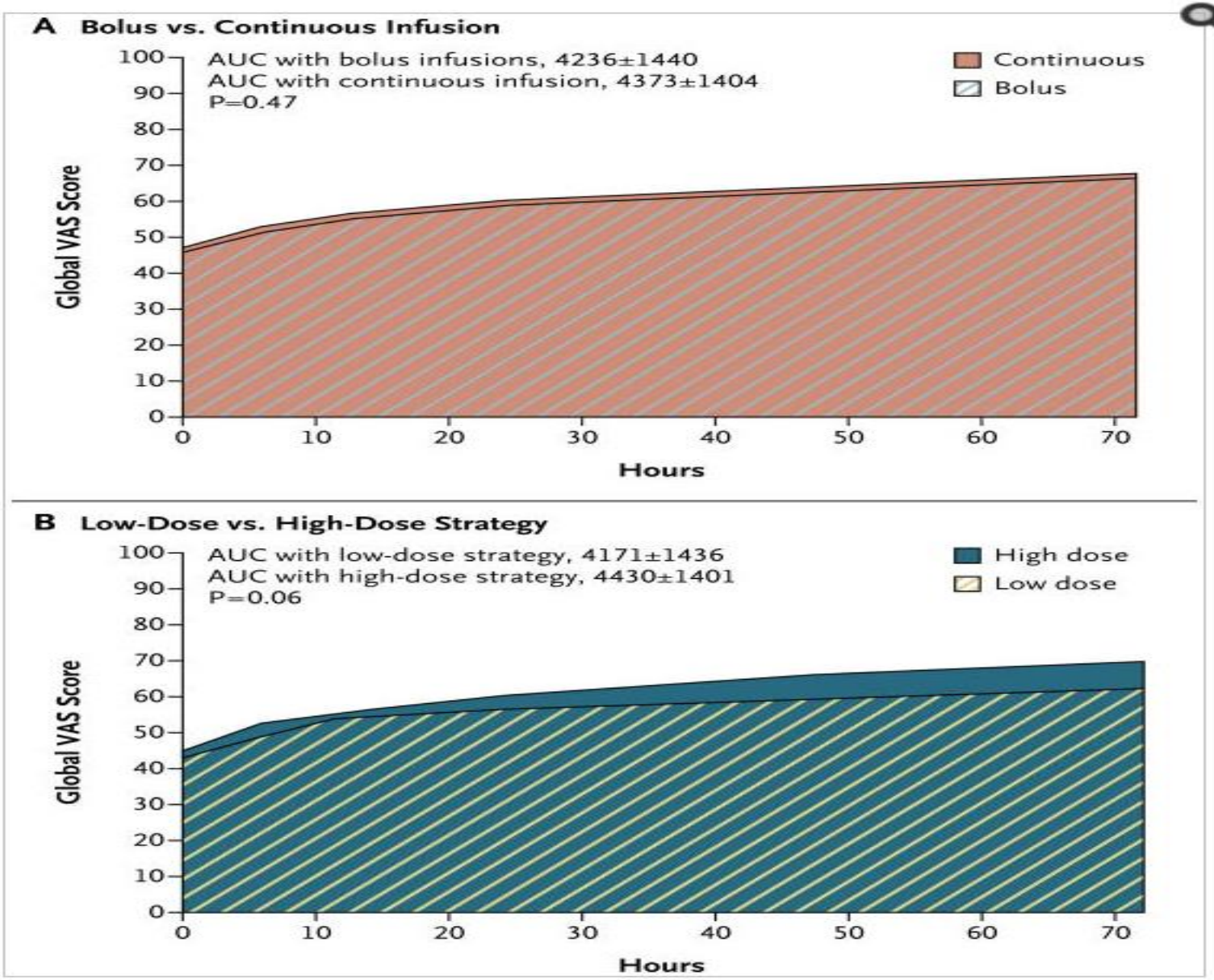
Click on image to zoom
Bolus
Continuous
Low Dose
High Dose



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

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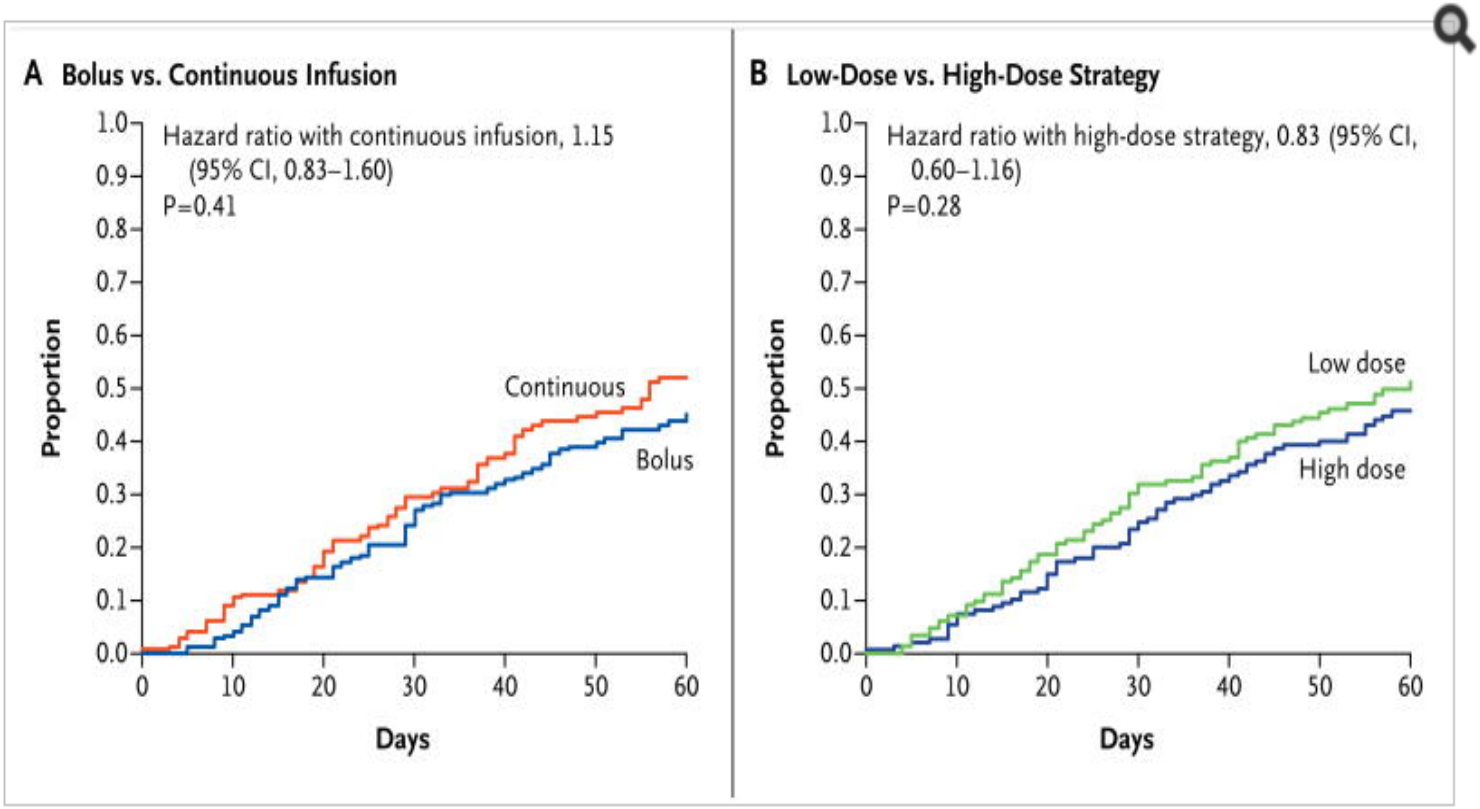
doi: 10.1056/NEJMoa1005419

Figure 3



Kaplan–Meier Curves for the Clinical Composite End Point of Death, Rehospitalization, or Emergency Department Visit

Kaplan–Meier curves are shown for death, rehospitalization, or emergency department visit during the 60-day follow-up period in the group that received boluses every 12 hours as compared with the group that received a continuous infusion (Panel A) and in the group that received a low dose of the diuretic (equivalent to the patients' previous oral dose) as compared with the group that received a high dose (2.5 times the previous oral dose) (Panel B).

N Engl J Med. 2011 Mar 3;364(9):797-805. doi: 10.1056/NEJMoa1005419.

Diuretic strategies in patients with acute decompensated heart failure.

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as compared with a low dose. (Funded by the National Heart, Lung, and Blood Institute; ClinicalTrials.gov number, NCT00577135.)

Comment in

Curr Treat Options Cardiovasc Med. 2014 Aug;16(8):330. doi: 10.1007/s11936-014-0330-5.

Treatment Approaches to Congestion Relief in Acute Decompensated HF: Insights After DOSE-AHF and CARRESS-HF.

Shakar SF[1], Lindenfeld J.

Author information

Abstract

OPINION STATEMENT: Most patients admitted to the hospital with ADHF do not achieve adequate relief of signs and symptoms of congestion. Patients with inadequate decongestion are known to be at higher risk of readmission for heart failure and mortality, although it is uncertain whether this is a cause or simply a marker of increased risk. Nonetheless, adequate decongestion is critical for improving quality of life. Based on the DOSE-AHF and CARRESS-HF studies, a high-dose diuretic regimen consisting of 2.5 times the daily dose of loop diuretic in furosemide equivalents, administered in twice-daily bolus doses, is reasonable to achieve a goal of 3-5 liters of urine output per day. Transient increases in creatinine in the first 4-5 days of diuresis should not be a limiting factor, but a prolonged progressive increase in creatinine signals a high-risk patient. Current goals for decongestion should be resolution of orthopnea, jugular venous pressure of < 8 cm of water, and trace to no peripheral edema. The hope is that better measures of assessing complete decongestion will reduce the progression to heart failure and mortality. While the best noninvasive method to assess speed of congestion has not been determined, it is clear that hemoconcentration (an increase in hematocrit) reflects a decrease in plasma volume and decongestion. In-line monitoring of hemoconcentration may improve the results of ultrafiltration therapy by preventing too large and/or too rapid a fall in intravascular volume and consequent triggering of neurohormonal activation. Several additional strategies such as serelaxin, high-dose mineralocorticoid receptor antagonists, and new forms and combinations of natriuretic peptides have shown promising results in the relief of congestion in patients with ADHF.

Table 5 Clinical Profiles

Clinical Presentation	Incidence*	Characteristics	Targets† and Therapies‡
Elevated BP (above 160 mm Hg)	~25%	Predominantly pulmonary (radiographic/clinical) with or without systemic congestion. Many patients have preserved EF.	Target: BP and volume management Therapy: vasodilators (e.g., nitrates§, nesiritide, nitroprusside) and loop diuretics
Normal or moderately elevated BP	~50%	Develop gradually (days or weeks) and are associated with systemic congestion. Radiographic pulmonary congestion may be minimal in patients with advanced HF.	Target: volume management Therapy: loop diuretics ± vasodilators
Low BP (<90 mm Hg)	<8%	Mostly related to low cardiac output and often associated with decreased renal function.	Target: cardiac output Therapy: inotropes with vasodilatory properties (e.g., milrinone, dobutamine, levosimendan); consider digoxin (intravenous and/or orally) ± vasopressor medications ± mechanical assist devices (e.g., IABP)
Cardiogenic shock	<1%	Rapid onset. Primarily complicating acute MI, fulminant myocarditis, acute valvular disease.	Target: improve cardiac pump function Therapy: inotropes ± vasoactive medications ± mechanical assist devices, corrective surgery
Flash pulmonary edema	3%‖	Abrupt onset. Often precipitated by severe systemic hypertension. Patients respond readily to vasodilators and diuretics.	Target: BP, volume management Therapy: vasodilators, diuretics, invasive or NIV, morphine¶

Heart. 2008 May;94(5):573-80. doi: 10.1136/hrt.2007.117978. Epub 2008 Jan 20.

The Hong Kong diastolic heart failure study: a randomised controlled trial of diuretics, irbesartan and ramipril on quality of life, exercise capacity, left ventricular global and regional function in heart failure with a normal ejection fraction.

Yip GW[1], Wang M, Wang T, Chan S, Fung JW, Yeung L, Yip T, Lau ST, Lau CP, Tang MO, Yu CM, Sanderson JE.

Author information

Abstract

BACKGROUND: Although heart failure with a preserved or normal ejection fraction (HFNEF or diastolic heart failure) is common, treatment outcomes on quality of life and cardiac function are lacking. The effect of renin-angiotensin blockade by irbesartan or ramipril in combination with diuretics on quality of life (QoL), regional and global systolic and diastolic function was assessed in HFNEF patients.

METHODS: 150 patients with HFNEF (LVEF >45%) were randomised to (1) diuretics alone, (2) diuretics plus irbesartan, or (3) diuretics plus ramipril. QoL, 6-minute walk test (6MWT) and Doppler echocardiography were performed at baseline, 12, 24 and 52 weeks.

RESULTS: The QoL score improved similarly in all three groups by 52 weeks (-46%, 51%, and 50% respectively, all $p<0.01$), although 6MWT increased only slightly (average +3-6%). Recurrent hospitalisation rates were equal in all groups (10-12% in 1 year). At 1 year, LV dimensions or LVEF had not changed in any group, though both systolic and diastolic blood pressures were lowered in all three groups from 4 weeks onwards. At baseline both mean peak systolic (Sm) and early diastolic (Em) mitral annulus velocities were reduced, and increased slightly in the diuretic plus irbesartan (Sm 4.5 (SEM 0.17) to 4.9 (SEM 0.16) cm/sec; Em 3.8 (SEM 0.25) to 4.2 (SEM 0.25) cm/sec) and ramipril (Sm 4.5 (SEM 0.24) to 4.9 (SEM 0.20) cm/sec; Em 3.3 (SEM 0.25) to 4.04 (SEM 0.32) cm/sec) groups (both $p<0.05$). NT-pro-BNP levels were raised at baseline (595 (SD 905) pg/ml; range 5-4748) and fell in the irbesartan (-124 (SD 302) pg/ml, $p = 0.01$) and ramipril (-173 (SD 415) pg/ml, $p = 0.03$) groups only.

CONCLUSIONS: In this typically elderly group of HF patients with normal LVEF, diuretic therapy significantly improved symptoms and neither irbesartan nor ramipril had a significant additional effect. However, diuretics in combination with irbesartan or ramipril marginally improved LV systolic and diastolic longitudinal LV function, and lowered NT-proBNP over 1 year.

1. Lindenfeld J, Albert NM, Boehmer JP et coll. HFSA 2010 Comprehensive Heart Failure Practice Guideline. *J Card Fail* 2010 ; 16 (6) : e1-e194.

8. Arnold JM, Liu P, Demers C et coll. Canadian Cardiovascular Society consensus conference recommendations on heart failure 2006: diagnosis and management. *Can J Cardiol* 2006 ; 22 (1) : 23-45.

Tableau II

***Traitements pharmacologiques étudiés contre l'insuffisance cardiaque à FEVG préservée*[1,8,10-14]**

Molécules	Mortalité	Hospitalisations pour insuffisance cardiaque	Dyspnée et qualité de vie	Recommandations[1,8]
Diurétiques	⇔	⇔	Améliorées	Oui, en cas d'hypervolémie
Périndopril	⇔	⇓	Améliorées	Oui, surtout en présence d'une autre indication
Candésartan	⇔	⇓	⇔	
Irbésartan	⇔	⇔	⇔	
Nébivolol	⇔	⇔		Oui, s'il y a une autre indication
Inhibiteurs des canaux calciques	⇔	⇔	Possiblement améliorées	
Digoxine	⇔	⇓	⇔	

⇓ : diminution ; ⇔ aucun changement

- Les diurétiques sont nécessaires pour réduire la volémie et les symptômes.
- Les diurétiques thiazidiques (hydrochlorothiazide) ou de l'anse (furosémide) peuvent être utilisés en première intention.
- Les diurétiques de l'anse sont plus puissants

$\Rightarrow$Hypervolémie importante.

ACC HEART FAILURE GUIDELINES SLIDE SET

Based on the 2009 Focused Update Incorporated Into the ACCF/AHA 2005 guidelines for the Diagnosis and Management of Heart Failure in Adults:

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

Developed in Collaboration With:

International Society for Heart and Lung Transplantation

HEART FAILURE GUIDELINES

2013 ACCF/AHA Guideline for the Management of Heart Failure

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

http://circ.ahajournals.org/content/early/2013/06/03/CIR.0b013e31829e8776.citation

2005 - 2010 GUIDELINES
ACUTE HF—TREATMENT GOALS

- **Table 12.3 Treatment Goals for Patients Admitted for ADHF**
  - Improve symptoms, especially congestion and low output symptoms
  - Restore normal oxygenation
  - **Optimize volume status**
  - Identify etiology
  - Identify and address precipitating factors
  - Optimize chronic oral therapy
  - Minimize side effects
  - Identify patients who might benefit from revascularization or device therapy
  - Identify risk of thromboembolism and need for anticoagulant therapy
  - Educate patients concerning medications and self assessment of HF
  - Consider and, where possible, initiate a disease management program

RECOMMENDED THERAPIES FOR HOSPITALIZED HF PATIENTS

Table 22. Recommendations for Therapies in the Hospitalized HF Patient

Recommendation	COR	LOE	References
HF patients hospitalized with fluid overload should be treated with intravenous diuretics	I	B	(310,311)
HF patients receiving loop diuretic therapy should receive an initial parenteral dose greater than or equal to their chronic oral daily dose; then should be serially adjusted	I	B	(312)
HF*r*EF patients requiring HF hospitalization on GDMT should continue GDMT unless hemodynamic instability or contraindicated	I	B	(307-309)
Initiation of beta-blocker therapy at a low dose is recommended after optimization of volume status and discontinuation of intravenous agents	I	B	(307-309)
Thrombosis/thromboembolism prophylaxis is recommended for patients hospitalized with HF	I	B	(22,324-328)
Serum electrolytes, urea nitrogen, and creatinine should be measured during titration of HF medications, including diuretics	I	C	N/A

http://circ.ahajournals.org/content/early/2013/06/03/CIR.0b013e31829e8776.citation

RECOMMENDED THERAPIES FOR HOSPITALIZED HF PATIENTS

When diuresis is inadequate, it is reasonable to a) give higher doses of intravenous loop diuretics; or b) add a second diuretic (e.g., thiazide)	IIa	B B	(37,312) (313-316)
Low-dose dopamine infusion may be considered with loop diuretics to improve diuresis	IIb	B	(317,318)
Ultrafiltration may be considered for patients with obvious volume overload	IIb	B	(319)
Ultrafiltration may be considered for patients with refractory congestion	IIb	C	N/A
Intravenous nitroglycerin, nitroprusside, or nesiritide may be considered an adjuvant to diuretic therapy for stable patients with HF	IIb	A	(320-323)
In patients hospitalized with volume overload and severe hyponatremia, vasopressin antagonists may be considered	IIb	B	(330,331)

http://circ.ahajournals.org/content/early/2013/06/03/CIR.0b013e31829e8776.citation

THE HOSPITALIZED PATIENT

Treatment With Intravenous Loop Diuretics

Patients admitted with HF and with evidence of significant fluid overload should be treated with intravenous loop diuretics. Therapy should begin in the emergency department or outpatient clinic without delay, as early intervention may be associated with better outcomes for patients hospitalized with decompensated HF *(Level of Evidence: B).*

If patients are already receiving loop diuretic therapy, the initial intravenous dose should equal or exceed their chronic oral daily dose. Urine output and signs and symptoms of congestion should be serially assessed, and diuretic dose should be titrated accordingly to relieve symptoms and to reduce extracellular fluid volume excess. *(Level of Evidence: C).*

New

THE HOSPITALIZED PATIENT

Intensifying the Diuretic Regimen

When diuresis is inadequate to relieve congestion, as evidence by clinical evaluation, the diuretic regimen should be intensified using either:

New

a. higher doses of loop diuretics;

b. addition of a second diuretic (such as metolazone, spironolactone or intravenous chlorthiazide) or

c. Continuous infusion of a loop diuretic.

Diuretic Therapy in ADHF

Table 5. Intravenous Diuretic Medications Useful for the Treatment of Severe Heart Failure

Drug	Initial Dose	Maximum Single Dose
Loop Diuretics		
Bumetanide	1.0 mg	4 to 8 mg
Furosemide	40 mg	160 to 200 mg
Torsemide	10 mg	100 to 200 mg
Thiazide Diuretics		
Chlorothiazide	500 mg	1000 mg
Sequential Nephron Blockade		
Chlorothiazide	500 to 1000 mg (IV) once or twice plus loop diuretics once; multiple doses per day	
Metozalone (as Zaroxolyn or Diulo)	2.5 to 5 mg PO once or twice daily with loop diuretic	
IV Infusions		
Bumetanide	1-mg IV load then 0.5 to 2 mg per hour infusion	
Furosemide	40-mg IV load then 10 to 40 mg per hour infusion	
Torsemide	20-mg IV load then 5 to 20 mg per hour infusion	

IV indicates intravenous; kg, kilograms; mg, milligrams; and PO, by mouth.

Hunt et.al. 2009. J Am Coll Cardiol 53;15:1-90

2005 - 2010 GUIDELINES
ACUTE HF—TREATMENT GOALS

- *Recommendation 12.7*
- Careful repeated assessment of signs and symptoms of congestion and changes in body weight is recommended, because clinical experience suggests it is difficult to determine that congestion has been adequately treated in many patients.
- *Strength of Evidence = C*

HFSA 2010 PRACTICE GUIDELINE
ACUTE HF—DIURETIC SIDE EFFECTS

- *Recommendation 12.9 (1 of 2)*
- Careful observation for development of a variety of side effects, including renal dysfunction, electrolyte abnormalities, symptomatic hypotension, and gout **is recommended** in patients treated with diuretics, especially when used at high doses and in combination.
- Patients should undergo routine laboratory studies and clinical examination as dictated by their clinical response.

Strength of Evidence = C

HFSA 2010 PRACTICE GUIDELINE
ACUTE HF—DIURETIC ALTERNATIVES

- *Recommendation 12.11*
- When congestion fails to improve in response to diuretic therapy, the following options **should be considered**:
  - Re-evaluating presence/absence of congestion,
  - Restricting sodium and fluid,
  - Increasing doses of loop diuretic,
  - Continuous infusion of a loop diuretic,
  - Or addition of a second type of diuretic orally (metolazone or spironolactone) or intravenously (chlorothiazide).

  Another option, ultrafiltration, **may be considered**.

Strength of Evidence = C

Table 28. Recommendations for Therapies in the Hospitalized HF Patient

Recommendations	COR	LOE	References
HF patients hospitalized with fluid overload should be treated with intravenous diuretics	I	B	737, 738
HF patients receiving loop diuretic therapy should receive an initial parenteral dose greater than or equal to their chronic oral daily dose; then dose should be serially adjusted	I	B	739
HF*r*EF patients requiring HF hospitalization on GDMT should continue GDMT except in cases of hemodynamic instability or where contraindicated	I	B	195, 735, 736
Initiation of beta-blocker therapy at a low dose is recommended after optimization of volume status and discontinuation of intravenous agents	I	B	195, 735, 736
Thrombosis/thromboembolism prophylaxis is recommended for patients hospitalized with HF	I	B	21, 770–774
Serum electrolytes, urea nitrogen, and creatinine should be measured during titration of HF medications, including diuretics	I	C	N/A
When diuresis is inadequate, it is reasonable to	IIa		
a. give higher doses of intravenous loop diuretics; or		B	38, 739
b. add a second diuretic (eg, thiazide)		B	740–743
Low-dose dopamine infusion may be considered with loop diuretics to improve diuresis	IIb	B	744, 745
Ultrafiltration may be considered for patients with obvious volume overload	IIb	B	752
Ultrafiltration may be considered for patients with refractory congestion	IIb	C	N/A
Intravenous nitroglycerin, nitroprusside, or nesiritide may be considered an adjuvant to diuretic therapy for stable patients with HF	IIb	A	760–763
In patients hospitalized with volume overload and severe hyponatremia, vasopressin antagonists may be considered	IIb	B	787, 788

COR indicates Class of Recommendation; GDMT, guideline-directed medical therapy; HF, heart failure; HF*r*EF, heart failure with reduced ejection fraction; LOE, Level of Evidence; and N/A, not available.

AGGRESSIVE FLUID AND SODIUM RESTRICTION IN ACUTE DECOMPENSATED HEART FAILURE
A RANDOMIZED CLINICAL TRIAL

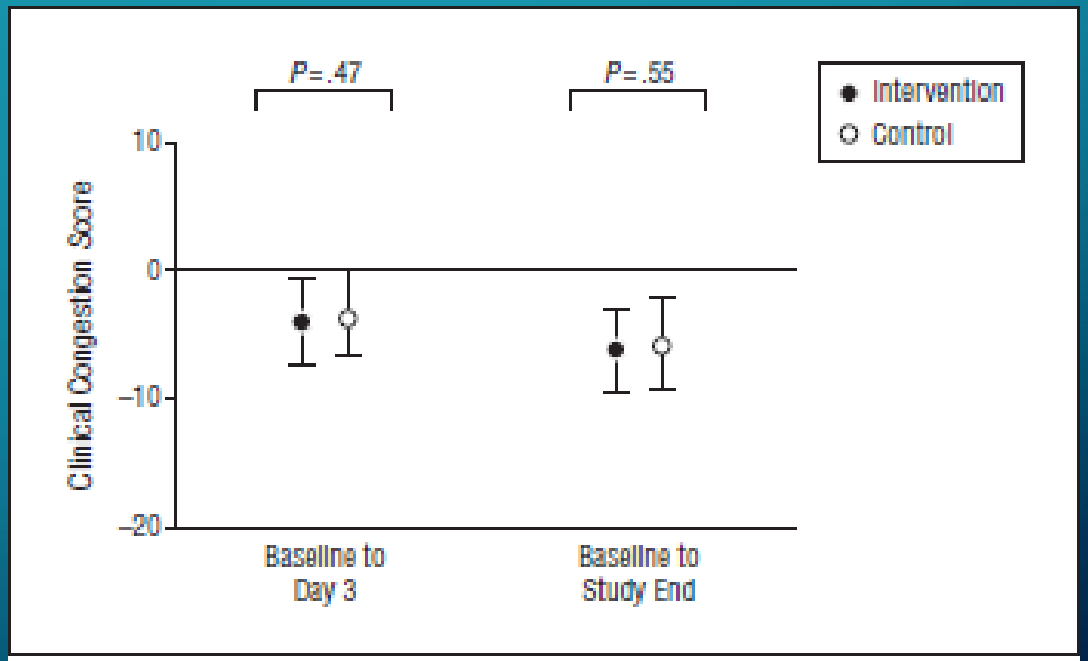


Figure 3. Change in clinical congestion score from baseline to 3-day reassessment and from baseline to the end of the study period in the intervention and control groups. Significance was determined using the Mann-Whitney test. Data points indicate the mean values; whiskers indicate SD.

JAMA Intern Med. 2013;173(12):1058-1064

Les diurétiques de l'anse ⇒

- diminution de filtration glomérulaire, par activation du système rénine- angiotensine-aldostérone

- activation du système nerveux sympathique*

Les effets indésirables des diurétiques* :

- variations volémie : eau intravasculaire,

- aggravation de la fonction rénale,

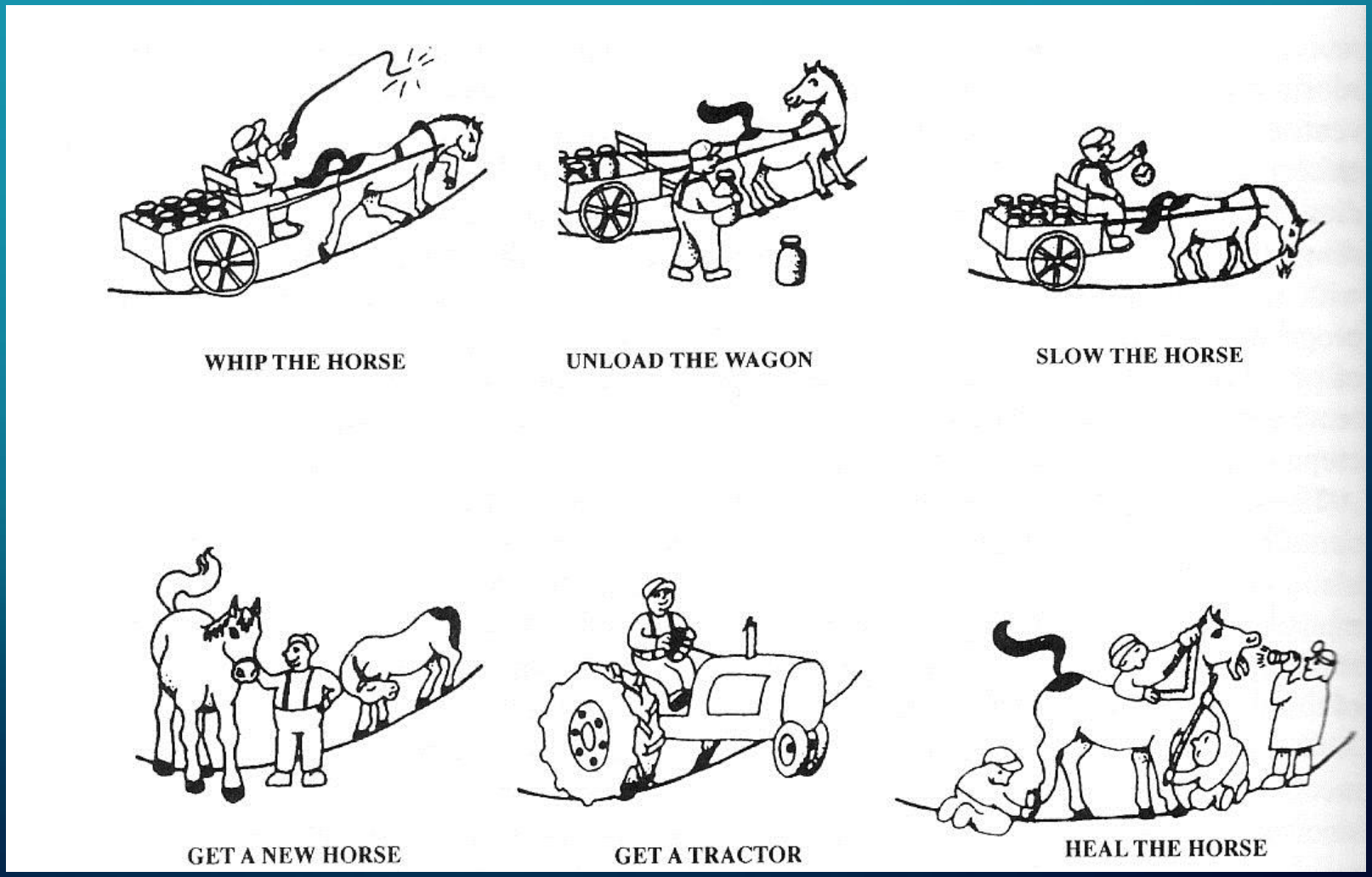
- troubles électrolytiques.

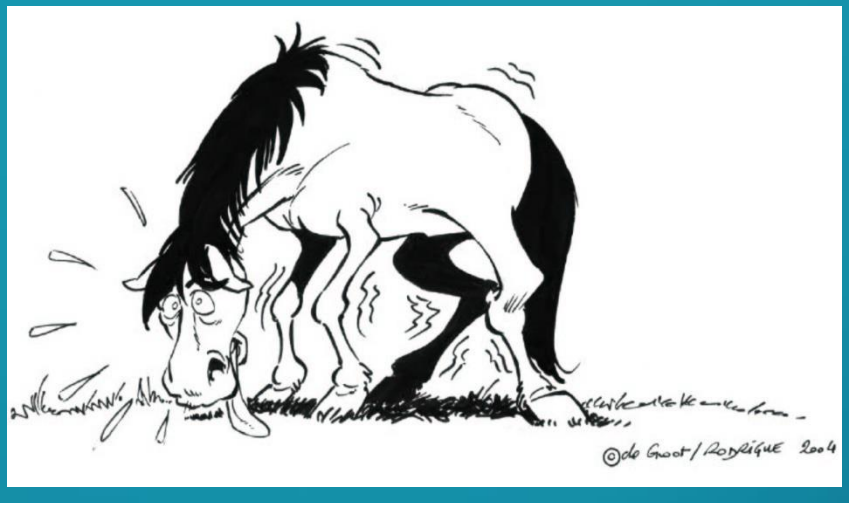
*Gottlieb SS, et al. Circulation 2002;105:1348–53.

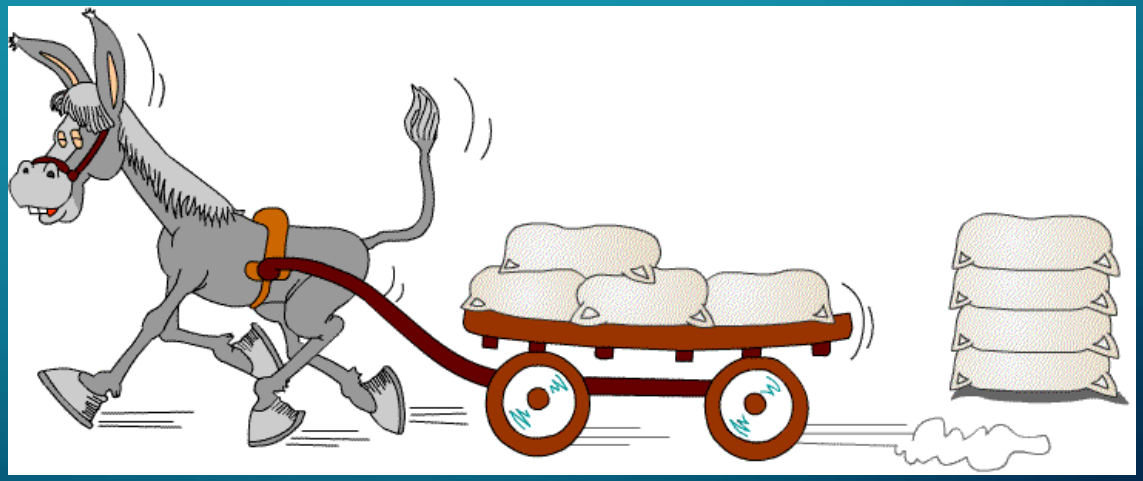
Développement de stratégies pour un traitement diurétique efficace et sécuritaire permettant l'amélioration des symptômes*.

*Freda BJ, et al. Am J Kidney Dis 2011;58:1005–17.

Insuffisance cardiaque : principes du traitement







Reduce the number of sacks on the wagon

DIURETIQUES, IEC (ARA2)

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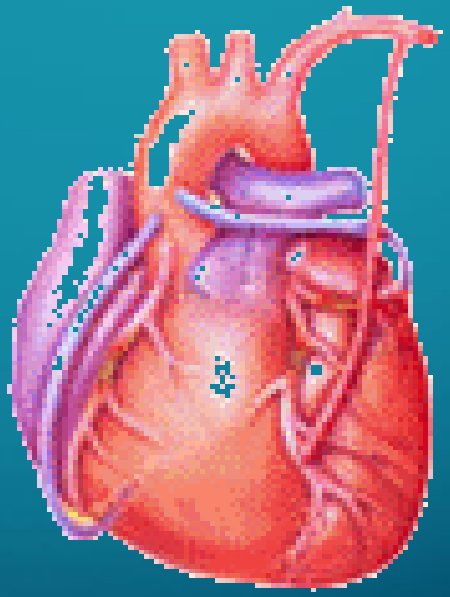
Urgences



Merci de votre attention