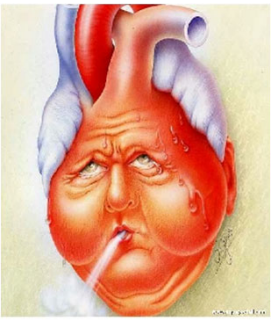
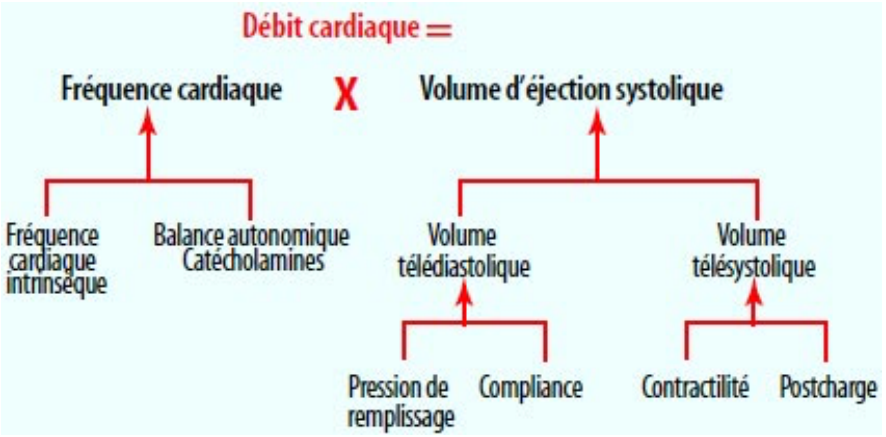
FONCTION RÉNALE ET CHOC CARDIOGÉNIQUE

Dr Anouk ALAUX
Service cardiopathies acquises (Pr OUATTARA)



DÉFINITION

Physiologiquement

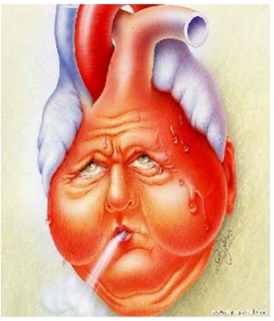


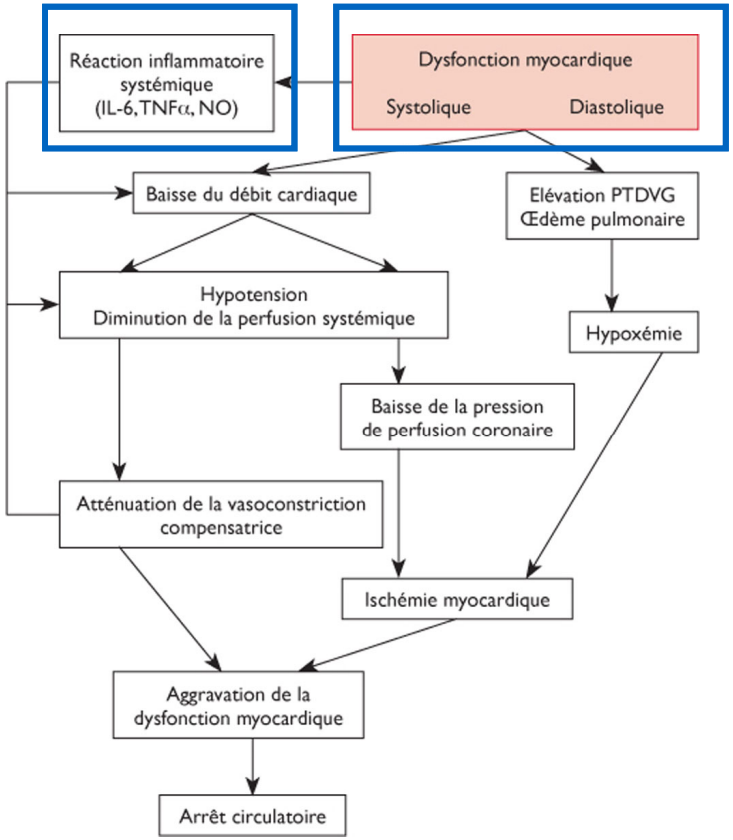
Hémodynamiquement

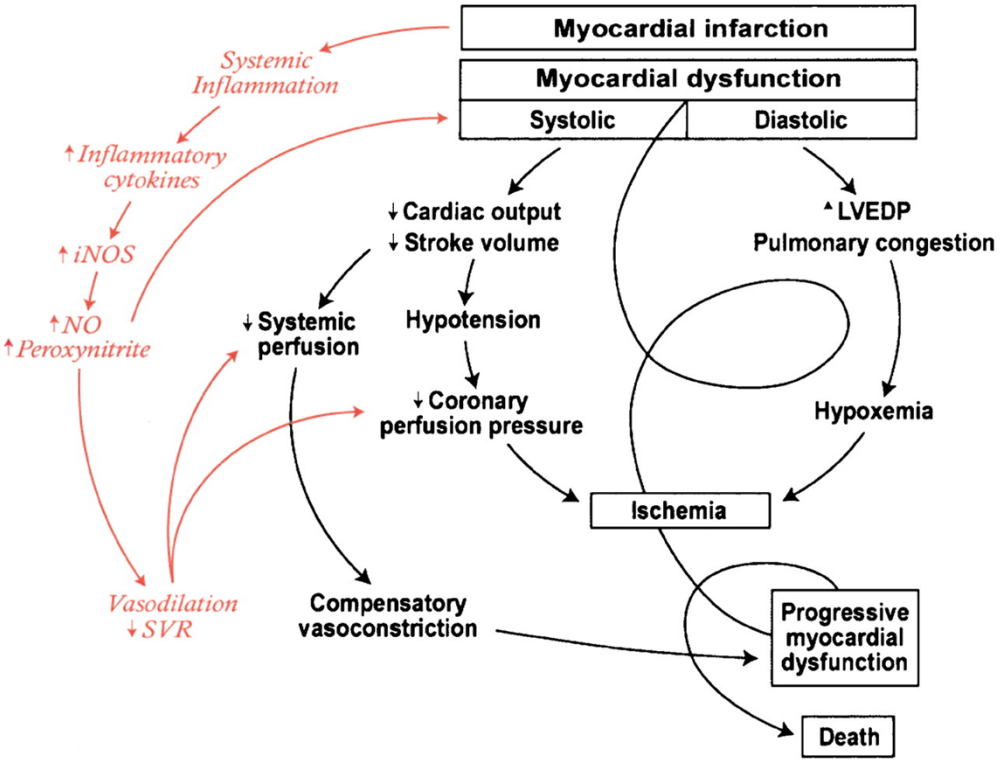
DC = PAM / RVS

CHOC CARDIOGÉNIQUE

→ Inadéquation apport O2 (DO2) VS consommation (VO2) **+** pré/post-charge




Réaction inflammatoire systémique (IL-6, TNFα, NO)
Dysfonction myocardique
Systolique
Diastolique
Baisse du débit cardiaque
Elévation PTDVG Œdème pulmonaire
Hypotension Diminution de la perfusion systémique
Hypoxémie
Baisse de la pression de perfusion coronaire
Atténuation de la vasoconstriction compensatrice
Ischémie myocardique
Aggravation de la dysfonction myocardique
Arrêt circulatoire


Myocardial infarction
Myocardial dysfunction
Systolic
Diastolic
Systemic Inflammation
↑Inflammatory cytokines
↑iNOS
↑NO
↑Peroxynitrite
↓Cardiac output
↓Stroke volume
↑LVEDP
Pulmonary congestion
↓Systemic perfusion
Hypotension
↓Coronary perfusion pressure
Hypoxemia
Ischemia
Vasodilation
↓SVR
Compensatory vasoconstriction
Progressive myocardial dysfunction
Death

ÉPIDÉMIOLOGIE

Étiologie choc cardiogénique: SCA ++

Khan. J Pak Med Assoc 67:1693–1697

Reynolds (2008). Circulation. 117:686–697

AKI 20-35% lors choc cardiogénique

19% nécessite EER

Lauridsen (2015) Crit Care 19:452

↗ mortalité (surtout si EER)

Ho (2014) Int J Cardiol Heart Vessel 3:88–89

Koreni (2002) Am J Med 112:115–119

Mishra J.*Lancet*. (2005) 365:1231–8

Chertow GM. *Am J Med*. (1998) 104:343–8

FdR indépendant si choc

ÉPIDÉMIOLOGIE

AKI = 17,6% post-CEC réglée

mortalité = 8,3-50% selon stade VS 1,8% sans AKI *Kallel. Néphrologie & Thérapeutique.2013;9:108–114*

↑ ventilation mécanique ↑ durée séjour ICU

↓ fonction cardiaque au décours

Krawczeski CD. *J Am Coll Cardiol*. (2011) 58:2301–9

Li S. *Crit Care Med*. (2011) 39:1493–9

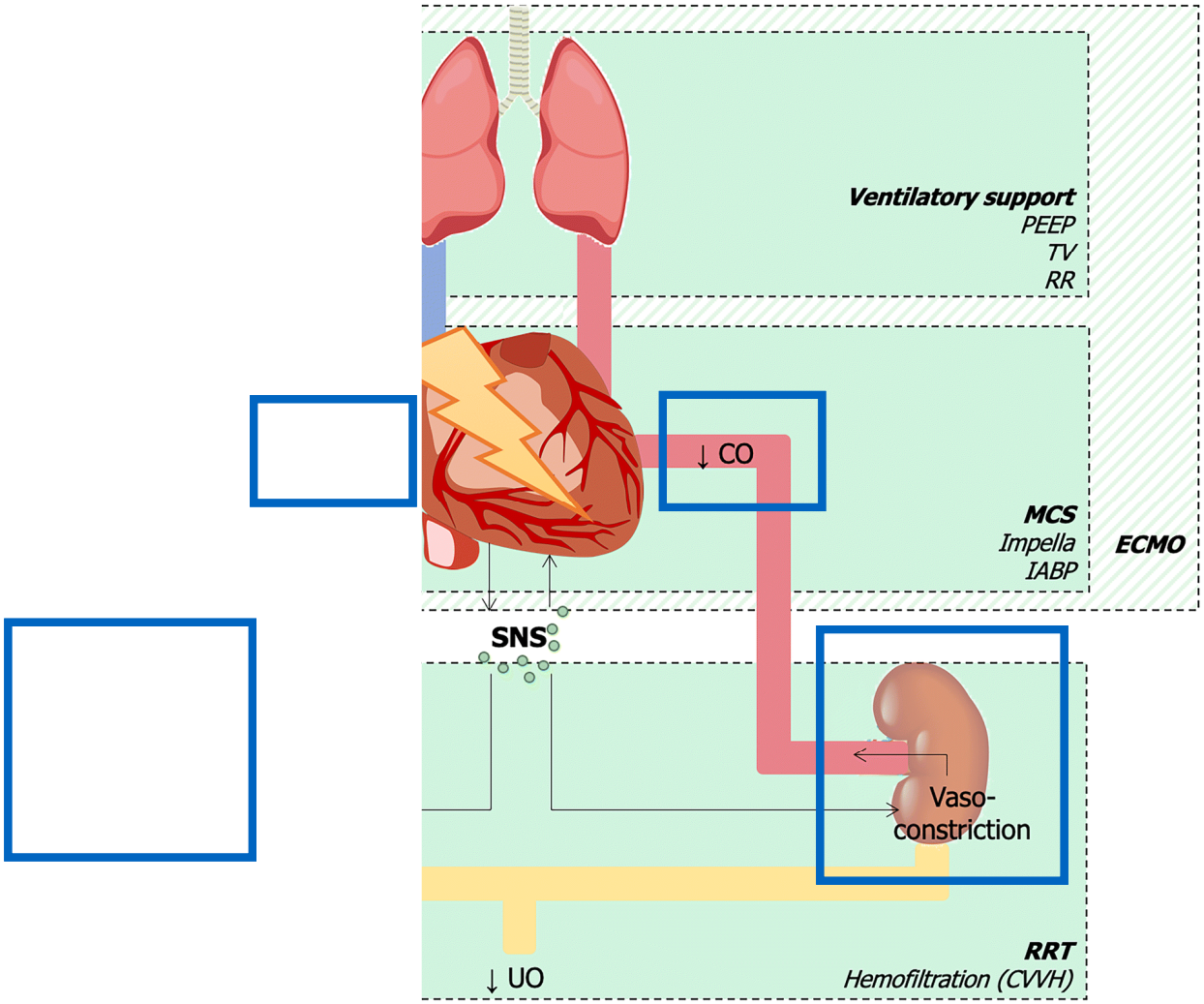
Blinder JJ. *J Thorac Cardiovasc Surg*. (2012) 143:368–74

IRA si IRC → FDR cardio-vasculaire

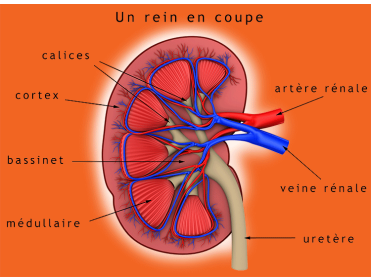
Askenazi. *Kidney Int*. (2006) 69:184–9

Greenberg. *JAMA Pediatr*. (2016) 170:1071–8

The classic shock paradigm



van den Akker(2019). J Crit Care 50:11–16



PHYSIOPATHOLOGIE

life saving BUT detrimental

Ghionzoli Heart Fail Rev (2021) 26:487–496

1. Hypoperfusion → sécrétion **adrénergique** massive

   contractilité + débit périphérique + mVO2

   pro-arythmique + effets cardiomyotoxiques

Hochman. Circulation. 2003; 107: 2998–3002

Hollenberg. Ann Intern Med. 1999; 131: 47–59.

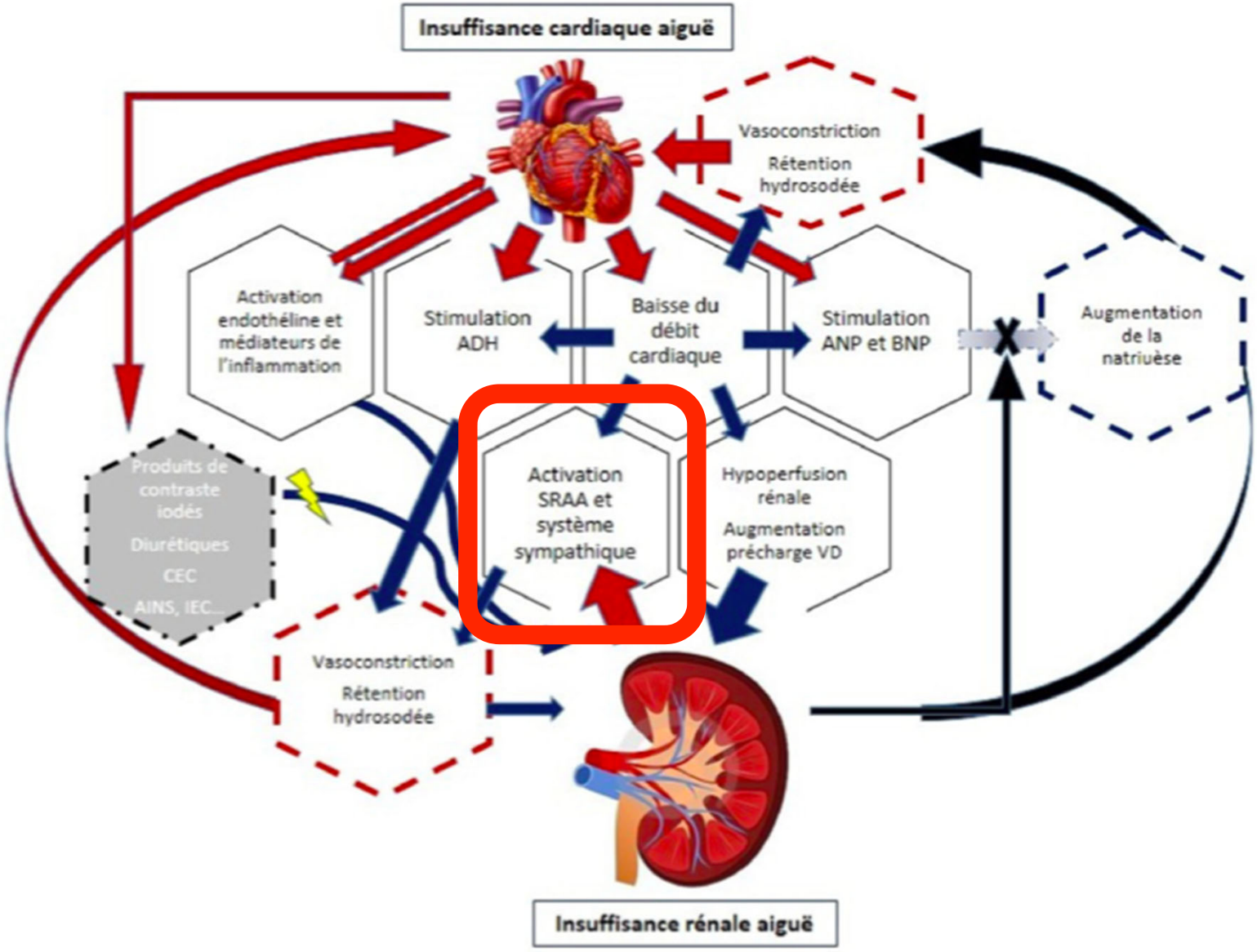
2. Vasoconstriction: ischémie + post-charge

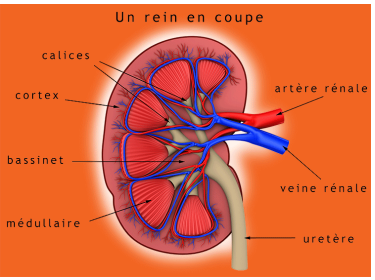
Metra (2008). Eur J Heart Fail 10:188e95

3. β-1 stimulation rénale → libération **rénine**

   → activation SRA: **rétention hydro-sodée**

Karth (2010) Crit Care Med 38:699–700


Insuffisance cardiaque aiguë
Vasoconstriction
Rétention hydrosodée
Activation endothéline et médiateurs de l'inflammation
Stimulation ADH
Baisse du débit cardiaque
Stimulation ANP et BNP
Augmentation de la natriurèse
Produits de contraste iodés
Diurétiques
CEC
AINS, IEC...
Activation SRAA et système sympathique
Hypoperfusion rénale
Augmentation précharge VD
Vasoconstriction
Rétention hydrosodée
Insuffisance rénale aiguë



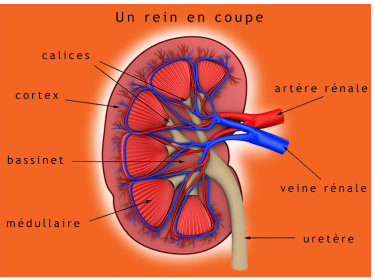
PHYSIOPATHOLOGIE

SRAA

1. ↗ activité Angiotensine II:
hypertrophie ventriculaire + rétention hydro-sodée

2. ↗ activité Aldostérone: réabsorption hydro-sodée

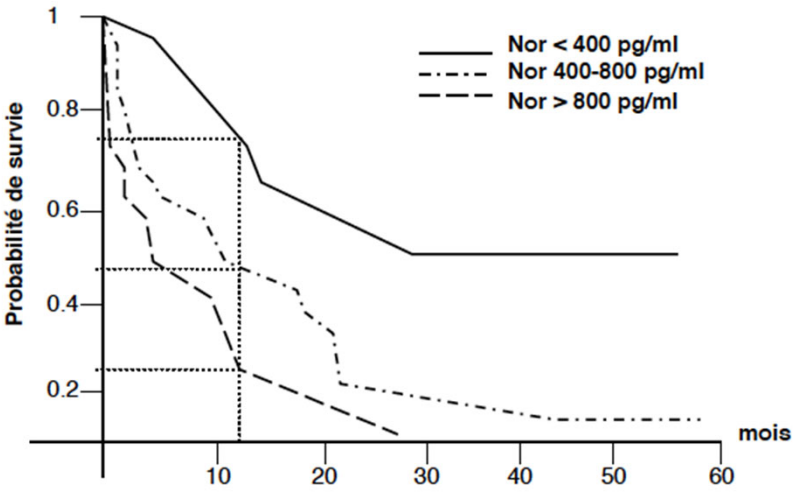
3. SYSTÈME VASOPRESSINERGIQUE:
   - ↗ inappropriée AVP «up-regulation» récepteurs V2
   - ↗ effet-antidiurétique



PHYSIOPATHOLOGIE

SNA

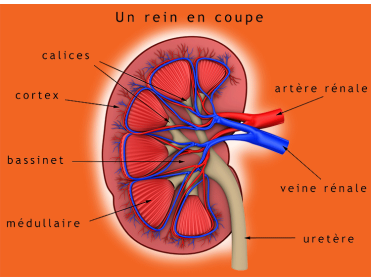
Activation SNA via ↗ activité AII (relargage pré-synaptique de noradrénaline)



<u>Catécholamines</u>:

→ réabsorption directe eau-Na^+

→ toxicité cellulaire directe (↗Ca^{++} intracellulaire)



PHYSIOPATHOLOGIE

Inflammation

1. **Ischémie**:

↗ cytokines pro-inflammatoires ✚ production massive de NO

Neumann. Circulation 1995 (92)

Rev Med Suisse 2014; volume 10. 1495-1500

2. **Cascade inflammatoire**:

↘ contractilité myocardique,

↘ réponse aux catécholamines

\+ vasodilatation systémique

Sleeper. Am Heart J. 2010 Sep;160(3):443-50

IMPACT inflammatoire !!!

Hochman. Circulation. 2003 Jun 24;107(24):2998-3002

Sleeper. Am Heart J. 2010 Sep;160(3):443-50

Acute Myocardial Infarction Complicated by Systemic Hypoperfusion without Hypotension: Report of the SHOCK Trial Registry

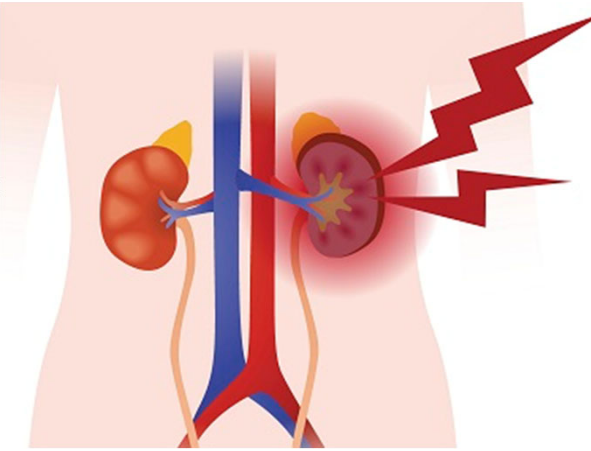
Menon. Am J Med. 2000 Apr 1;108(5):374-80


choc cardiogénique:

Sans / avec hypotension = **43%** VS **66%** *(p=0,001)*

FACTEURS DE RISQU

AKI si choc



Angiographie: NON

Schmucker (2018) Bremen STEMI Registry. Eur Heart J Acute Cardiovasc Care 7:710–722

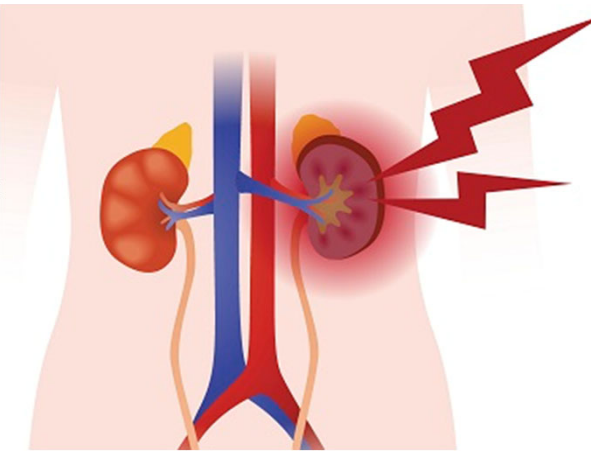
Methodist Debakey Cardiovasc J. 2022; 18(4): 73–85.

CPBIA: NON / OUI

Sukhodolya (2013). ASAIO J 59:593–599

FACTEURS DE RISQU

en CEC



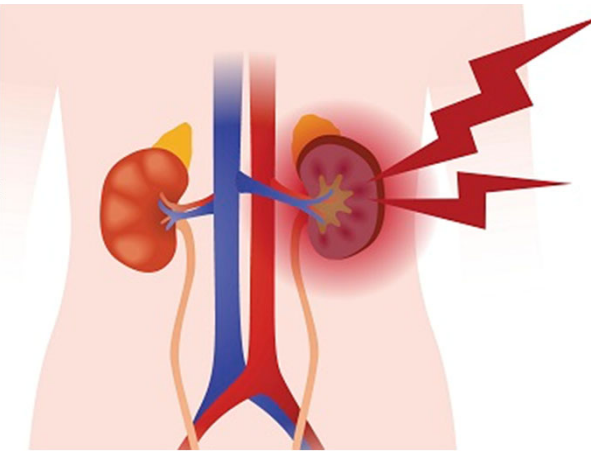
CEC: hémolyse (shear forces et surfaces non biocompatibles) →lésions tubulaires

Devarajan P. J Am Soc Nephrol. (2006) 17:1503–20.

Rosner. J Intensive Care Med, 2008;23(1):3-18.

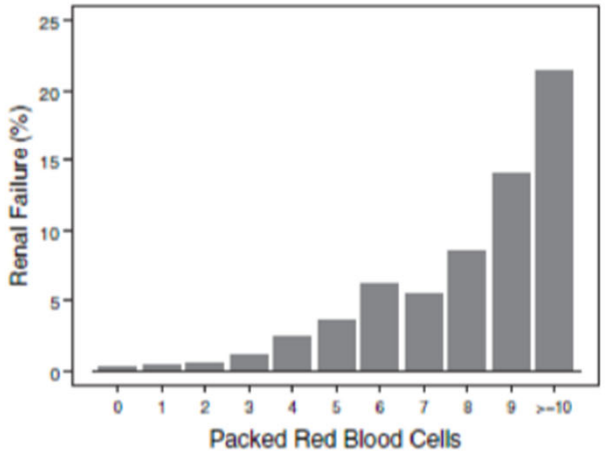
FACTEURS DE RISQU

en CEC



OR = 1.22 [1.19–1.26], p < 0.001 pour chaque CGR additionnel

Furnary, Circulation 2007



Bonnot. Néphrologie & Thérapeutique. 2016;12:345–346

DFG préopératoire (OR 0,977 *p=0,006*)

colloïdes (OR 3,712 *p=0,0001*)

CGR transfusés (OR 1,15 *p=0,02*)

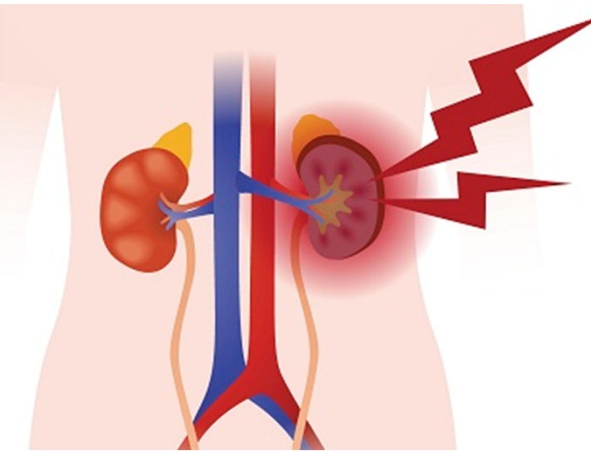
NT-ProBNP préopératoire (OR 1,00386 *p=0,019*)

âge (OR 1,0385 *p=0,014*)

IMC (OR 1,08 *p=0,026*)

FACTEURS DE RISQU

en CEC



Kallel. Néphrologie & Thérapeutique.2013;9:108–114

âge, EUROscore, IRA pré-op (DFG<63 mL/min), clampage et CEC, CRP (>158 mg/L)

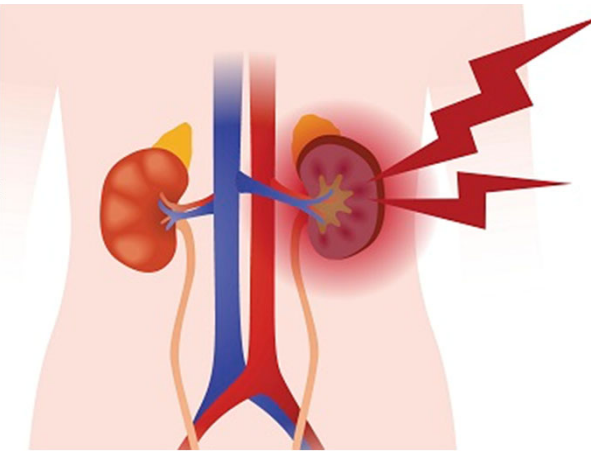
Pickering et al. Am J Kidney Dis, 2015

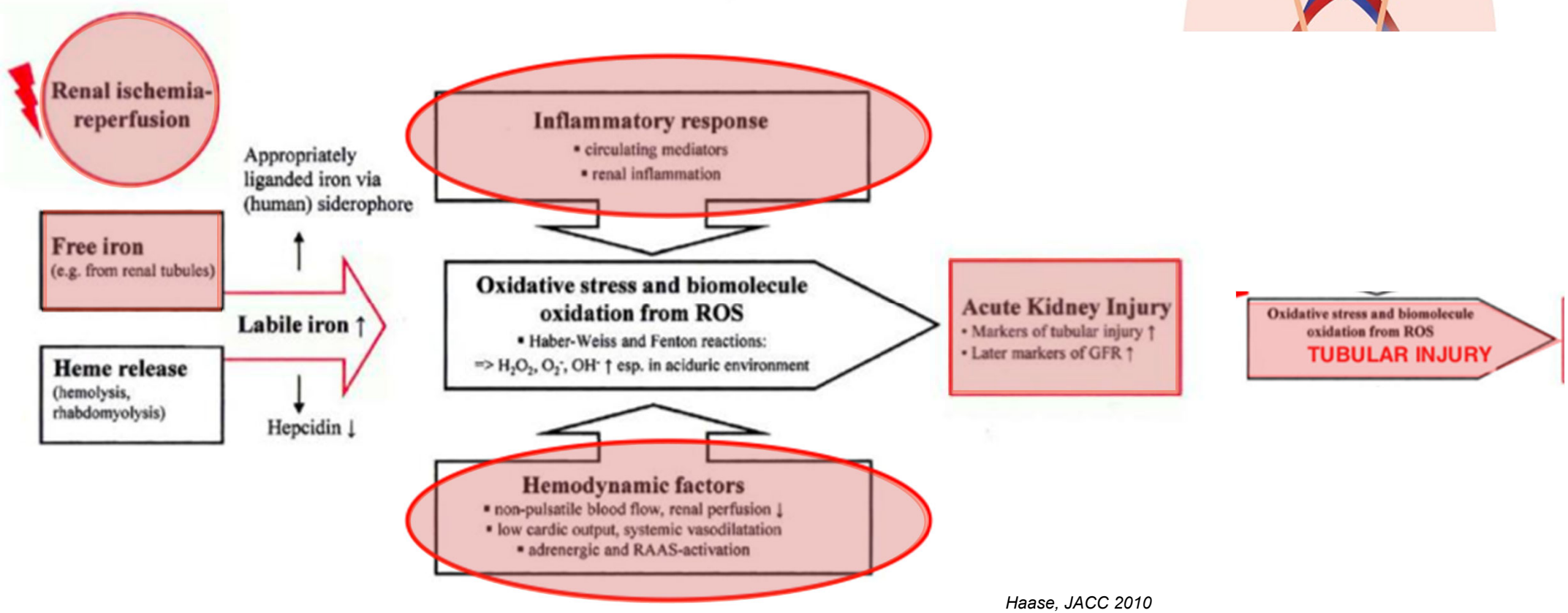
sepsis (50%), IRC (30%)

Mortalité x4 post CEC si AKI

FACTEURS DE RISQU

en CEC

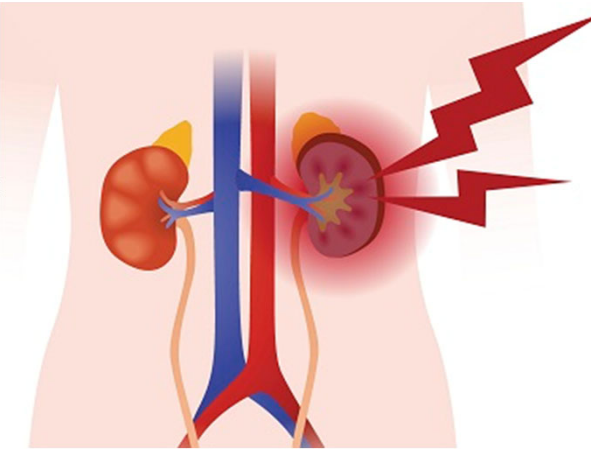




Haase, JACC 2010

FACTEURS DE RISQU

chocs médicaux



Sepsis

Pedersen. Dan Med J. 2012 Feb 1;59(2):177e178

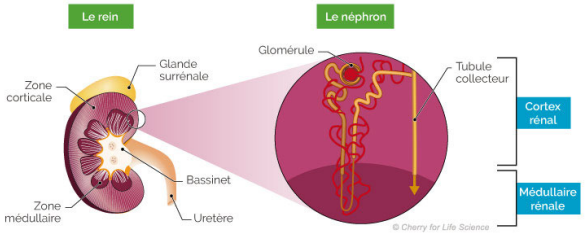
Tolwani. J Am Soc Nephrol. 2008 Jun 1;19(6):1233e1238.

Chirurgie, insuffisance hépatocellulaire

Harada.Crit Care Res Pract. 2019, 2019 Nov 20.

APACHE et SOFA score sont facteurs pronostics si AKI

Wang. Ren Fail. 2020;42(1):638e645.



DIAGNOSTIC AKI

Recommandations *KDIGO 2012* pour AKI

STADES	Cr	Diurèse
1	4/804/<{#Fu#edvh#RX#A59#kP	?3/8p O1nj1k#shqgdqw#9#â#45k
2	505/<{#Fu#edvh	?3/8p O1nj1k#A45k
3	A6{#Fu#edvh#RX#A687#kP #RX#gŸexw#HU	?3/6p O1nj1k#A57k#RX#bqxuh#A45k

SFNDT si IRA: UV/P

$$\text{Clairance} = \frac{U \times V}{P}$$

Si les unités suivantes sont utilisées :

U = concentration urinaire de créatinine (en mmol/l pour ce calculateur)

V = débit urinaire (en ml par 24h soit 1440 min)

P = concentration plasmatique de créatinine (en µmol/l)

SYNDROME CARDIO-RENAL

Classification du SCR

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 (e.g. chronic congestive heart failure) causing progressive and permanent chronic kidney disease*
CRS Type III (Acute Renocardiac Syndrome) *Abrupt worsening of renal function (e.g. acute kidney ischaemia or glomerulonephritis) causing acute cardiac disorders (e.g. heart failure, arrhythmia, ischemia)*
CRS Type IV (Chronic Renocardiac Syndrome) *Chronic kidney disease (e.g. chronic glomerular 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. DM, sepsis) causing both cardiac and renal dysfunction*

1. Ronco C et al. *Eur Heart J* 2009;Dec 25 [epub ahead of print]

SYNDROME CARDIO-RENAL

CRS Type I (Acute Cardiorenal Syndrome)
Abrupt worsening of cardiac function leading to acute kidney injury

diminution DC: hypoperfusion rénale ✵ ischémie → NTA

Ronco. J Am Coll Cardiol 2012;60:1031–42

1. pas corrélation DFG et Index Cardiaque

Cody. Kidney Int 1988;34:361–7

2. pas corrélation ↘ DC-gravité IRA

Nohria. J Am Coll Cardiol 2008;51:1268–74

3. adaptation rénale si hypoperfusion

Abuelo. N Engl J Med 2007;357:797–805
Ljungman. Drugs 1990;39:10–21

SYNDROME CARDIO-RENAL

auto-entretien SCR via SRAA: hypoperfusion rénale majorant rétention hydro-sodée

→ diminuant inotropsime cardiaque via:

1. **ENDOTHÉLINE**:
   - puissant vasoconstricteur sécrété par l'endothélium vasculaire
   - médiateur pro-inflammatoire et profibrosant
     → atteinte cardiaque et rénale (microangiopathies)

Macedo. Curr Opin Crit Care 2009;15:467–73

2. **MACROPHAGES**:

Mills. Am J Cardiol 2003;92:222–6

SYNDROME CARDIO-RENAL

cœur droit ?

Défaillance VD sur congestion veineuse: **corrélation inverse PVC-DFG**

Maxwell. J Clin Invest 1950;29:342–8
Damman. J Am Coll Cardiol 2009;53:582–8

PVC meilleur indice que Index Cardiaque pour pronostic rénal

Mullens. J Am Coll Cardiol 2009;53:589–96

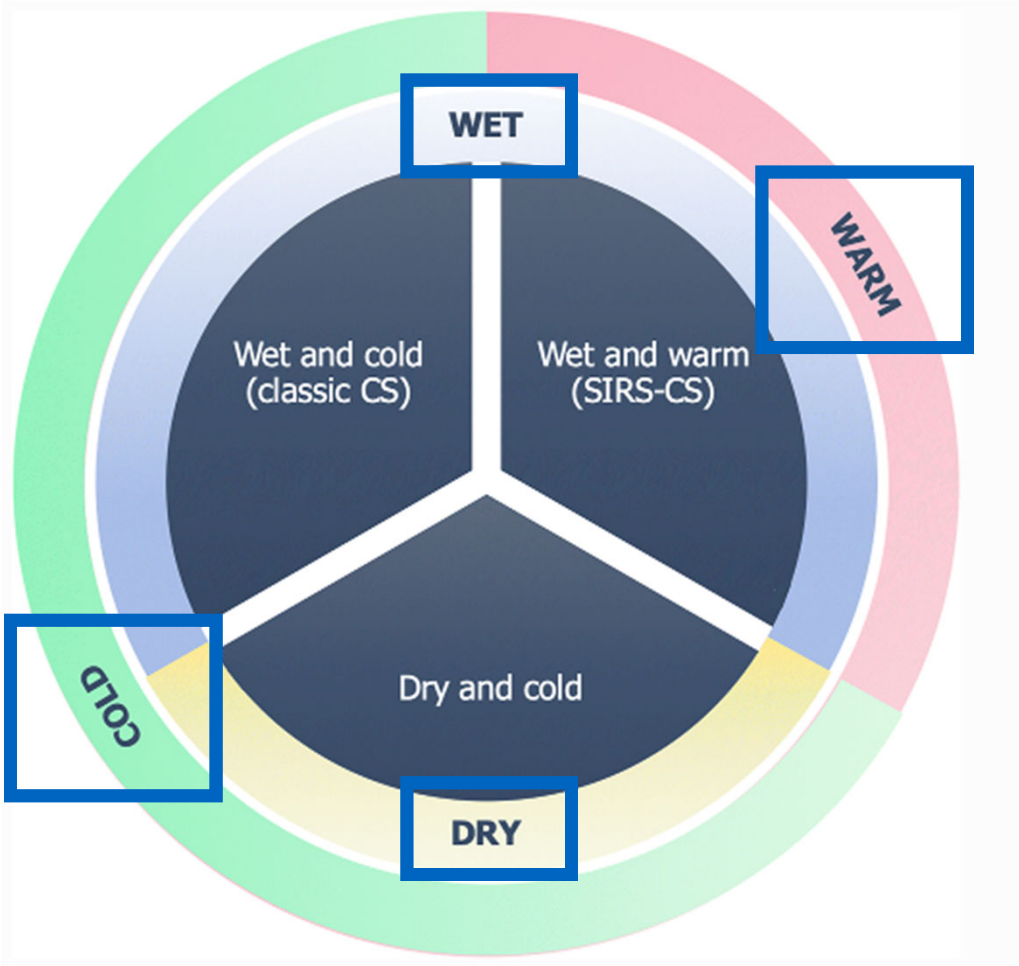
PVC meilleur indice que PAM sur AKI

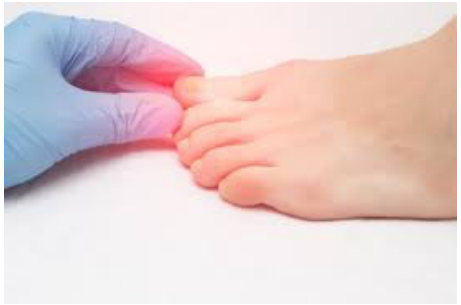
Legrand. Crit Care. (2013) 17:R278
Algaze. Pediatr Crit Care Med. (2017) 18:34–43

PVC: facteur prédictif indépendant d'AKI si choc

van den Akker(2019). J Crit Care 50:11–16

3 phénotypes de choc cardiogénique





van den Akker (2019). J Crit Care 50:11–16

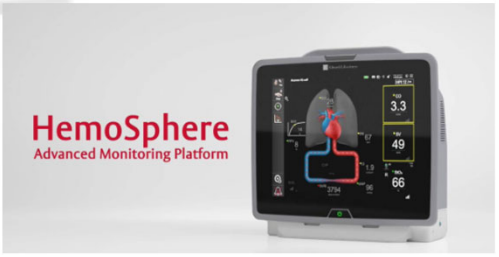
SYNDROME CARDIO-RENAL

dépistage ?

CRS Type I (Acute Cardiorenal Syndrome)
Abrupt worsening of cardiac function leading to acute kidney injury

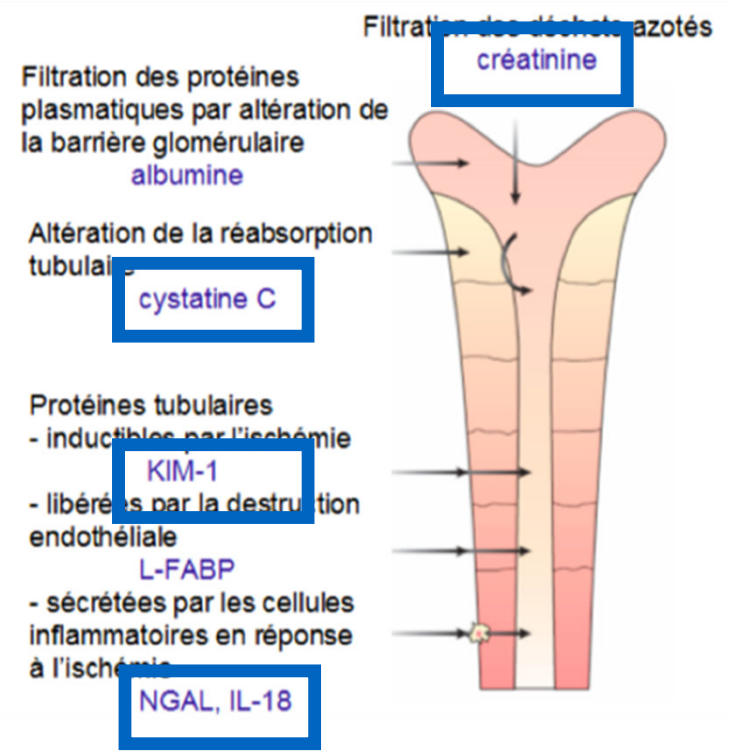
→ **CONJONCTURE**:

- monitorage hémodynamique: Swan-Ganz / PVC
- diurèse, créatininémie
- BNP *Rodseth. J Am Coll Cardiol 2013.*
- NGAL plasmatique et urinaire plus précoce si NTA *Wagener. Anesthesiology 2006;105:485–91*



SYNDROME CARDIO-RENAL

dépistage



BIOMARQUEURS RÉNAUX:

1. **Fonctionnels** de filtration glomérulaire: inuline, créatinine, albuminurie…
2. **Lésionnels** (atteinte tubulaire): cystatine C, KIM-1, NGAL

Briggs JP. Kidney Int. 2008: 74, 987–989

Wagener. Anesthesiology 2006;105:485–91

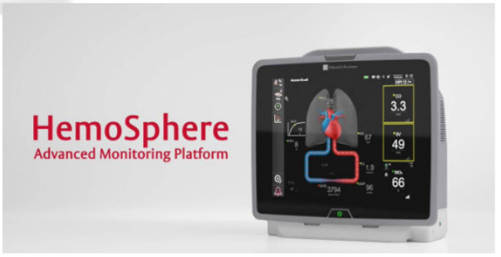
SYNDROME CARDIO-RENAL

dépistage ?

CRS Type I (Acute Cardiorenal Syndrome)
Abrupt worsening of cardiac function leading to acute kidney injury

→ **CONJONCTURE**:

- monitorage hémodynamique: Swan-Ganz / PVC
- diurèse, créatininémie
- BNP *Rodseth. J Am Coll Cardiol 2013.*
- NGAL plasmatique et urinaire plus précoce si NTA *Wagener. Anesthesiology 2006;105:485–91*



Autres marqueurs: *Anaesthesia Critical Care & Pain Medicine.Volume 37, Issue 4,2018, Pages 335-341*

- doppler rénal
- TIMP-2 et IGFBP7

A combined approach for the early recognition of acute kidney injury after adult cardiac surgery

Cédrick Zaouter [a,*], Julien Potvin [a], Marie-Lise Bats [b], Marie-Christine Beauvieux [b], Alain Remy [a], Alexandre Ouattara [a,c,d]

Anaesthesia Critical Care & Pain Medicine.Volume 37, Issue 4,2018, Pages 335-341

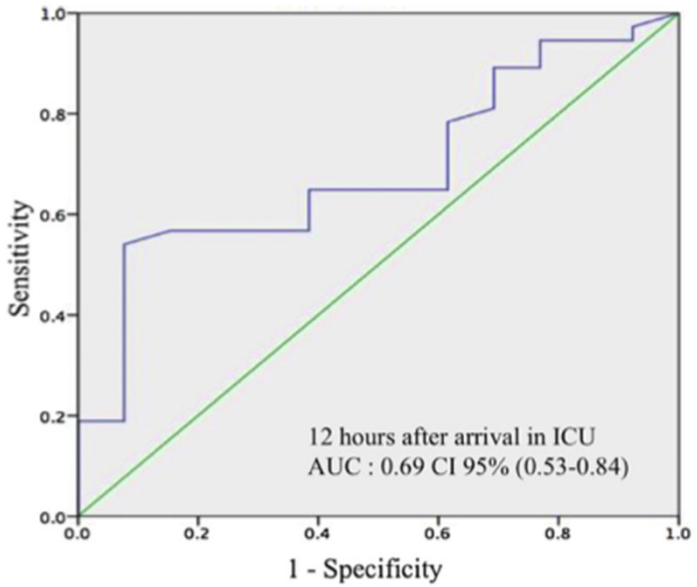


Fig. 2. This figure depicts the receiver operating characteristic (ROC) curve for the prediction of AKI using the urinary [TIMP-2]*[IGFBP7] measured 12 hours after arrival in intensive care (blue line). The line of no-discrimination is represented in green.

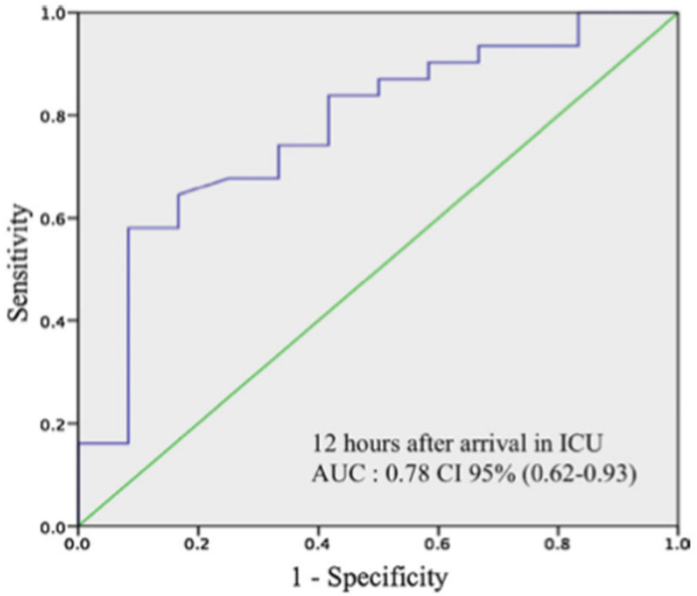


Fig. 3. This figure depicts the receiver operating characteristic (ROC) curve for the prediction of AKI using the Doppler RRI and combined the urinary [TIMP-2]*[IGFBP7] measured 12 hours after arrival in intensive care (blue line). The line of no-discrimination is represented in green. AKI: Acute kidney injury; AUC: area under the curve; CI: confidence interval.

Prevention of cardiac surgery-associated AKI by implementing the KDIGO guidelines in high risk patients identified by biomarkers: the PrevAKI randomized controlled trial

Meersch. I*ntensive Care Med*. (2017) 43:1551– 1561

Intérêt du “KDIGO bundle”:

- [TIMP-2]·[IGFBP7] urinaire > 0.3
- Optimisation hémodynamique
- Éviter les drogues néphrotoxiques
- Prévenir hyperglycémie

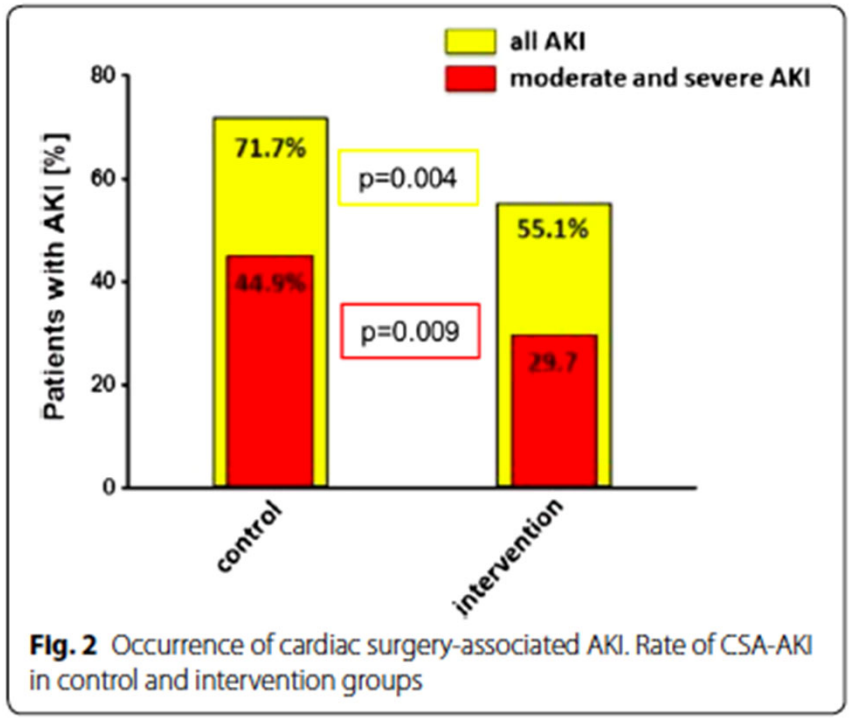


Fig. 2 Occurrence of cardiac surgery-associated AKI. Rate of CSA-AKI in control and intervention groups

PROPHYLAXIE

Bellomo, Ronco. Cardiology. 2001;96(3-4):169-76.

1. Perfusion rénale
2. Eviter surcharge volémique: PVC, PC

Oedème ↘ EO2 locale Lim. Clin Cardiol (2016) 39:477–483

3. Dépister quand dépléter Beaubien-Souligny et al. Ultrasound J (2020) 12:16
4. Eviter nephro-toxiques:

AINS induisent atteinte rénale sans AKI chez enfant Nehus E. *J Pediatr*. (2017) 189:175–80

Methodist Debakey Cardiovasc J. 2022; 18(4): 73–85.

Aucun impact positif

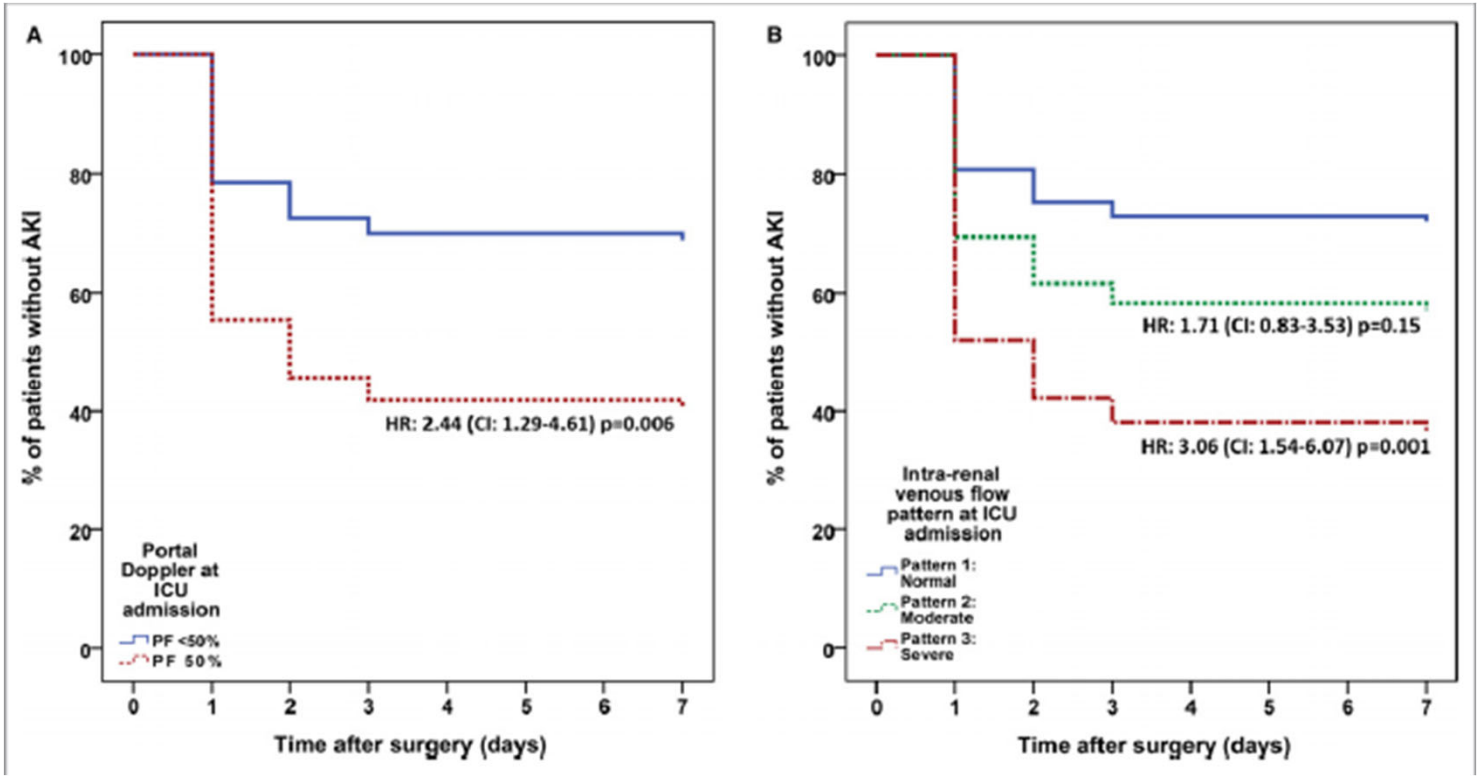
vasodilateurs, chelateurs Fer, anti-apoptotiques, diurétiques

Park M. *Am J Nephrol*. (2010) 31:408–18

Alterations in Portal Vein Flow and Intrarenal Venous Flow Are Associated With Acute Kidney Injury After Cardiac Surgery: A Prospective Observational Cohort Study

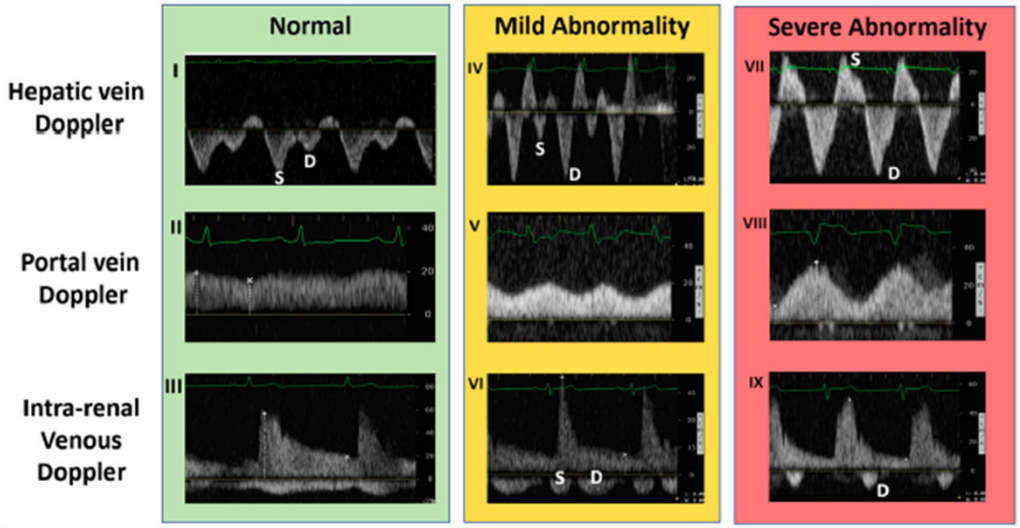
William Beaubien-Souligny, MD; Aymen Benkreira, MD; Pierre Robillard, MD; Nadia Bouabdallaoui, MD; Michaël Chassé, MD, PhD; Georges Desjardins, MD; Yoan Lamarche, MD, MSc; Michel White, MD; Josée Bouchard, MD; André Denault, MD, PhD

Beaubien-Souligny et al. Am Heart Assoc. 2018;7:e009961.



Quantifying systemic congestion with Point-Of-Care ultrasound: development of the venous excess ultrasound grading system

The Ultrasound Journal



	VExUS A	VExUS B	VExUS C	VExUS D	VExUS E
Grade 0	IVC < 2 cm	IVC < 2 cm	IVC < 2 cm		
Grade 1	IVC ≥ 2 cm **Normal** patterns *(All three of : I, II, III)*	IVC ≥ 2 cm **Normal** patterns *(All three of : I, II, III)*	IVC ≥ 2 cm **Normal** patterns **or mild** abnormalitie(s) *(Any combination of : I, II, III, IV, V, VI)*	**Normal** patterns *(All three of : I, II, III)*	**Normal** patterns **or mild** abnormalitie(s) *(Any combination of : I, II, III, IV, V, VI)*
Grade 2: Mild congestion	IVC > 2 cm **Mild** abnormality in **at least one** pattern *(At least one of : IV, V, VI)*	IVC > 2 cm **Mild or severe** abnormality in **at least one** pattern *(At least one of : IV, V, VI, VII, VIII, IX)*	IVC > 2 cm **Severe** abnormalities in **at least one** pattern *(At least one of : VII, VIII, IX)*	**Mild or severe** abnormalities **in at least one** pattern *(At least one of : IV, V, VI, VII, VIII, IX)*	**Severe** abnormalities **in at least one** pattern *(At least one of : VII, VIII, IX)*
Grade 3: Severe congestion	IVC > 2 cm **Severe** abnormalities **in at least one** pattern *(At least one of : VII, VIII, IX)*	IVC > 2 cm **Mild or severe** abnormalities **in multiple** patterns *(At least two of : IV, V, VI, VII, VIII, IX)*	IVC > 2 cm **Severe** abnormalities **in multiple** patterns *(At least two of : VII, VIII, IX)*	**Mild or severe** abnormalities **in multiple** patterns *(At least two of : IV, V, VI, VII, VIII, IX)*	**Severe** abnormalities **in multiple** patterns *(At least two of : VII, VIII, IX)*

Beaubien-Souligny et al. Ultrasound J (2020) 12:16

THÉRAPEUTIQUES *du choc*

Sédation / Ventilation mécanique

Price (2010). Crit Care 14:R169

Gurm. Crit Care Clin 2007 (23)

Inotropes

Holmes. Curr Opin Crit Care 2005 (11)

Ellender. Emerg Med Clin N Am 2008

Traitement étiologique

Revascularisation précoce si choc cardiogénique ischémique

SHOCK trial. Congest Heart Fail. 2003;9(1):35-9.

Causes	Traitements
Syndrome coronarien aigu	Revascularisation coronarienne
Valvulopathie aiguë	Chirurgie de remplacement valvulaire
Tamponnade	Drainage péricardique
Embolie pulmonaire	Thrombolyse, embolectomie
Insuffisance chronotrope sur bradycardie	Stimulateur cardiaque
Tachyarythmie	Cardioversion pharmacologique ou électrique
Sepsis	Antibiotiques, amines
Dissection aortique	Prise en charge chirurgicale
Toxique, médicamenteuse	Antidotes
Dysfonction du ventricule droit prédominante	• Médicament inotrope et vasopresseur • Expansion volumique

Rev Med Suisse 2014; volume 10. 1495-1500

THÉRAPEUTIQUES
décongestion: les diurétiques

Diurétiques ne ↘ pas mortalité:

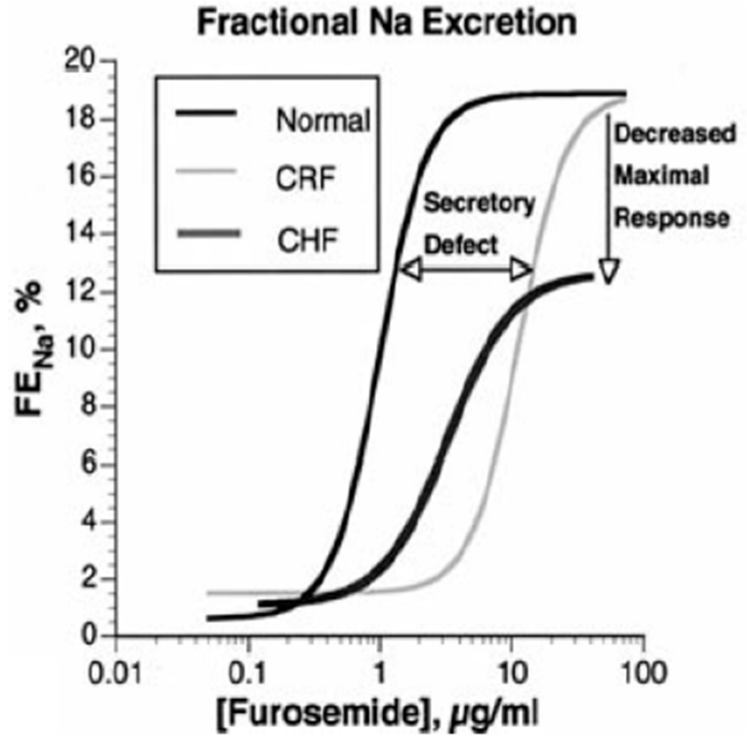
- Efficaces SSI stade modéré, sinon effets indésirables Ho. *BMJ*. (2006) 333:420
- Risque non récupération rénale et mortalité Mehta. JAMA.2002 Nov 27;288(20):2547-53.

NON excrétés lors bas débit, pour exercer action rénale

van der Vorst. *Paediatr Drugs*. (2006) 8:245–64

THÉRAPEUTIQUES
les diurétiques

RÉSISTANCE AUX DIURÉTIQUES



Elison DH Cardiology 2001;96:132–43

THÉRAPEUTIQUES

EER

↘ Mortalité J30 si EER lors choc cardiogénique

Fahad. Cureus. 2020 Jan; 12(1): e6591.

UF plus efficace si échec diurétiques

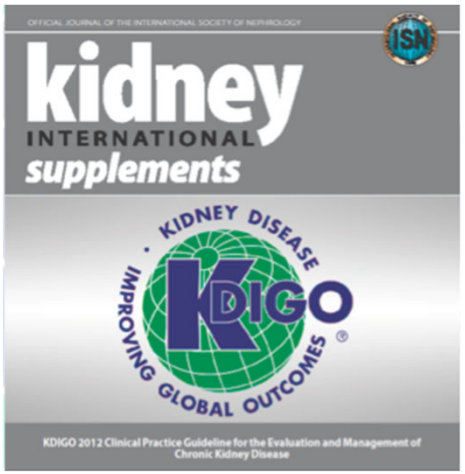
Agostoni. Am J Med. 1994 Mar; 96(3):191-9.

Ronco. Cardiology. 2001; 96(3-4):155-68.

UF n'active pas SRAA VS diurétiques

💡 *n'active pas SRAA via la macula densa*

Agostoni. Am J Med 1994;96:191–199.



THÉRAPEUTIQUES

EER quel stade ?

Recommandations KDIGO 2012

KDIGO AKI Work Group (2012). Kidney Int Suppl 2(1):1–138

→ Plus précoce ? Pré-émptif ?

✔ Méta-analyse: *Zou. Crit Care. 2019 Apr 25;23(1):142*

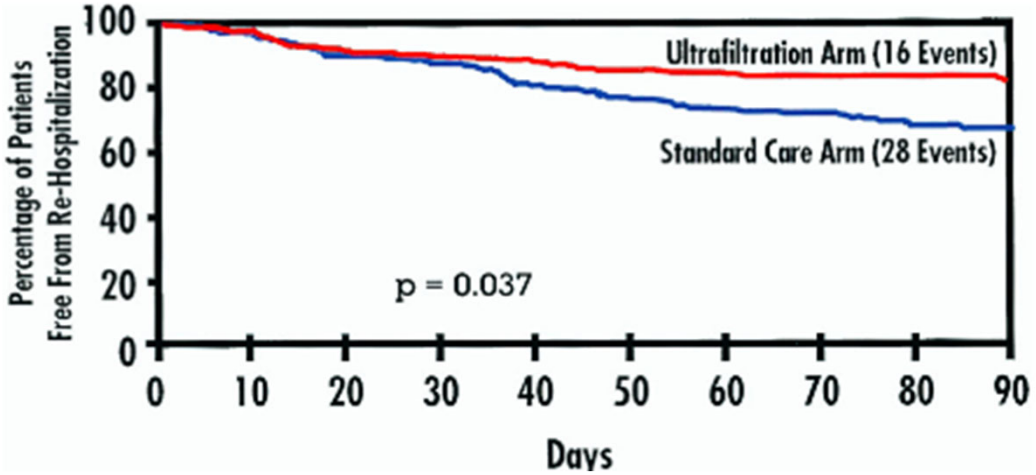
⊖ Méta-analyse: *Yang. Nephrol. 2017 Aug 7;18(1):264*

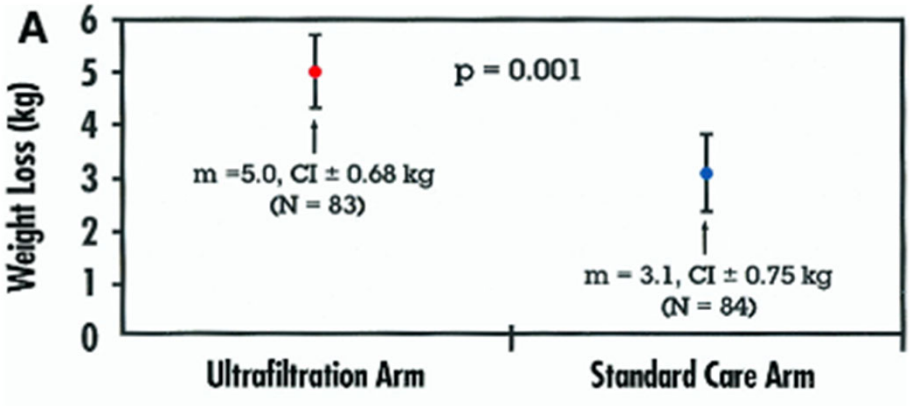
Méta-analyse: *Lai. Ann Intensive Care. 2017 Dec;7(1):38*

Ultrafiltration Versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Heart Failure

Constanzo. J Am Coll Cardiol. 2007 Feb 13; 49(6):675-83.

The UNLOAD (Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Congestive Heart Failure) trial was a prospective, random-

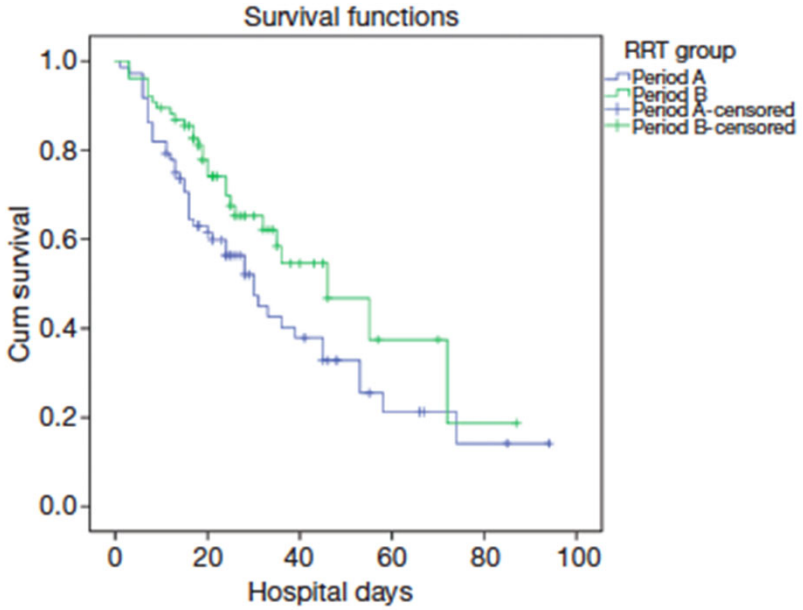




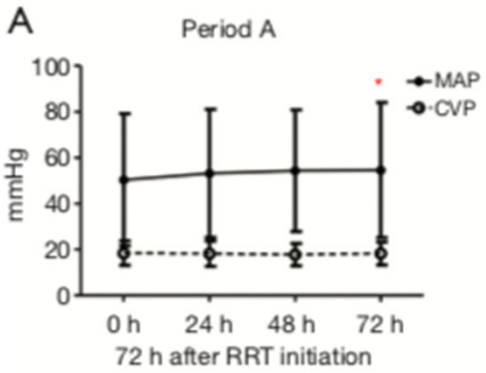
Preemptive renal replacement therapy in post-cardiotomy cardiogenic shock patients: a historically controlled cohort study

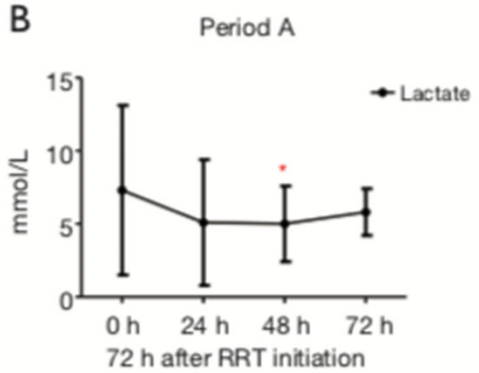
Tu. Ann Transl Med. 2019 Oct; 7(20): 534

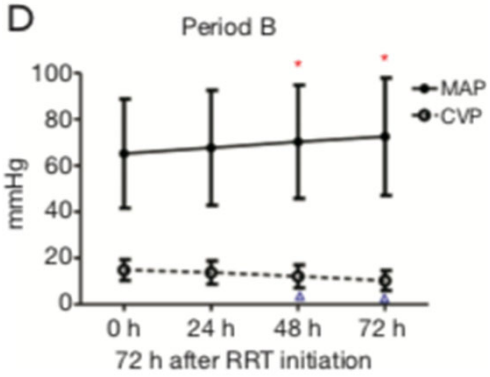
2 périodes: EER tardive ou précoce [47h (20-127) VS 23h (17-66) *p<0,01*]

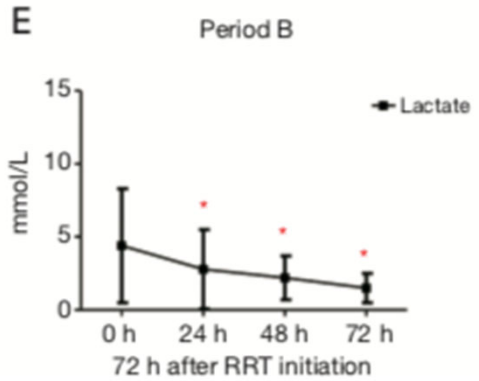


↘ mortalité si EER précoce [38.0% vs 59.2% *p<0,01*]





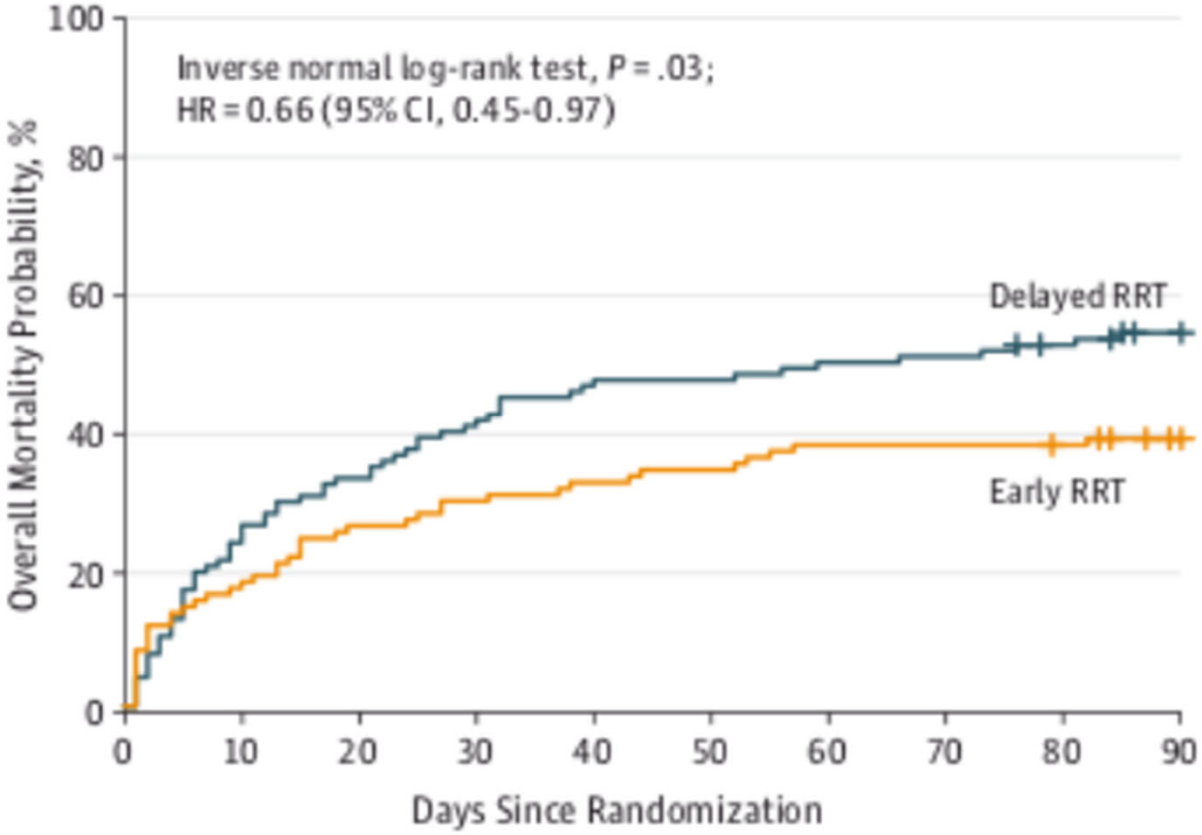




Effect of Early vs Delayed Initiation of Renal Replacement Therapy on Mortality in Critically Ill Patients With Acute Kidney Injury

The ELAIN Randomized Clinical Trial

Zarbock A JAMA. 2016 May 24-31;315(20):2190-9



↘ mortalité J90 si EER précoce

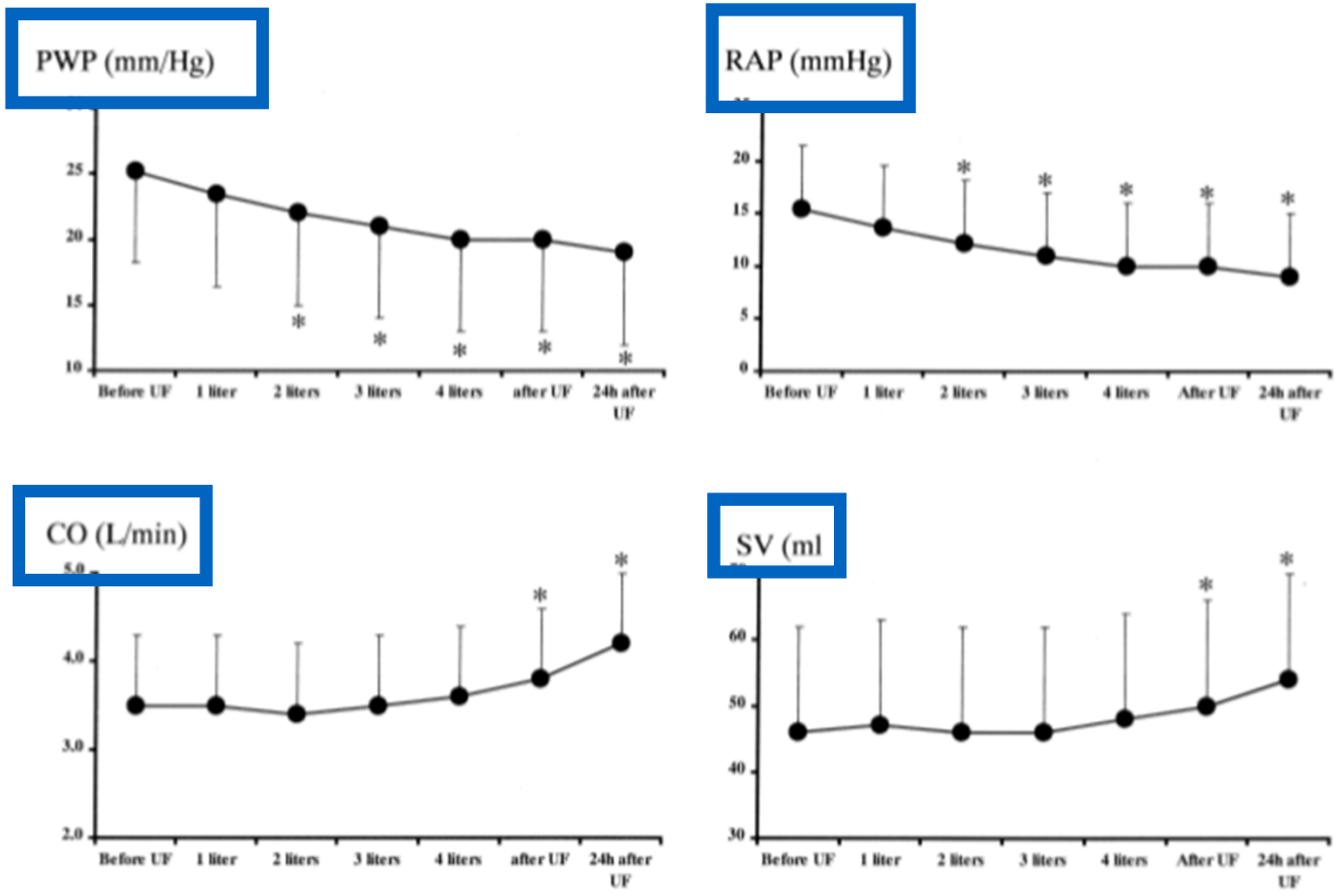
39,3% VS 54,7% *(p=0,03)*

Délai médian 6h VS 25,5h *(p<0,001)*

CJS: ↘ durée EER et VM, récupération rénale

Circulatory Response to Fluid Overload Removal by Extracorporeal Ultrafiltration in Refractory Congestive Heart Failure

Marenzi. J Am Coll Cardiol 2001;38:963–8



Nombreuses publications favorables …

Post-Opératoire

Bent et al. Ann Thorac Surg 2001; 71: 832-7

Eur J Cardiothorac S 004 Nov;26(5):1027-31.

Médical

Bart. J Am Coll Cardiol. 2005 Dec 6;46(11):2043-6.

Niagara®

Cathéter 2 voies court terme pour hémodialyse et plasmaphérèse

Power-Trialysis®

Cathéter 3 voies court terme pour hémodialyse et plasmaphérèse

Cathéter périphérique ?

Jaski. J Card Fail. 2003 Jun;9(3):227-31.

Bart. J Card Fail. 2012 Mar;18(3):176-82.

Cardiorenal Rescue Study in Acute Decompensated Heart Failure: Rationale and Design of CARRESS-HF, for the Heart Failure Clinical Research Network

Bart. J Card Fail. 2012 Mar;18(3):176-82.

Bart. N Engl J Med 2012;367:2296–304.

Serious Adverse Events.

Event	Pharmacologic Therapy (N = 94)	Ultrafiltration (N = 94)
	no. of patients (%)	
Any	54 (57)	68 (72)
Heart failure	28 (30)	31 (33)
Other cardiovascular disorder	5 (5)	6 (6)
Renal failure	14 (15)	17 (18)
Anemia or thrombocytopenia	5 (5)	8 (9)
Catheter-site hemorrhage	0	2 (2)
Electrolyte disorder*	3 (3)	0
Gastrointestinal hemorrhage	3 (3)	7 (7)
Pneumonia or other respiratory disorder	6 (6)	10 (11)
Sepsis, bacteremia, or cellulitis	4 (4)	8 (9)
Other	19 (20)	17 (18)

*Included in this category are hyperkalemia, hypokalemia, hypernatremia, hypoatremia, and hyperuricemia.

UF<diurétiques à H96 (*p=0,003*)

aggravation AKI

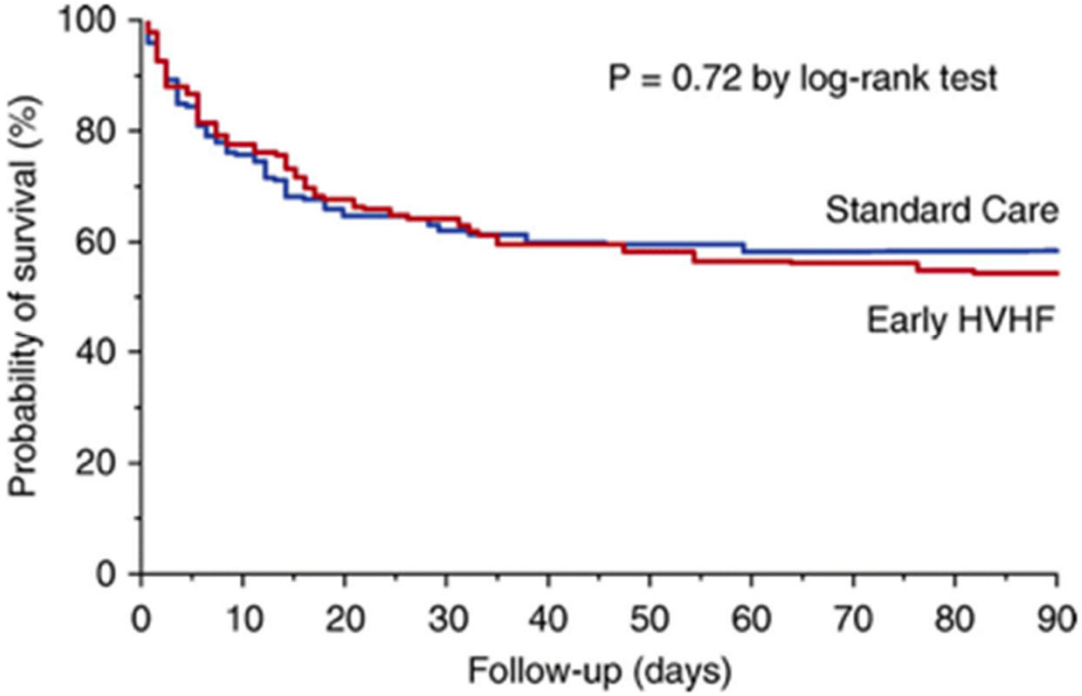
évènements indésirables (72 vs 57%, *p=0,03*)

Early High-Volume Hemofiltration versus Standard Care for Post–Cardiac Surgery Shock

The HEROICS Study

Alain Combes[1], Nicolas Bréchot[1], Julien Amour[2], Nathalie Cozic[3], Guillaume Lebreton[4], Catherine Guidon[5], Elie Zogheib[6], Jean-Claude Thiranos[7], Jean-Christophe Rigal[8], Olivier Bastien[9], Hamina Benhaoua[10], Bernard Abry[11], Alexandre Ouattara[12], Jean-Louis Trouillet[1], Alain Mallet[3], Jean Chastre[1], Pascal Leprince[4], and Charles-Edouard Luyt[1]

H Am J Respir Crit Care Med; 2015 Nov 15;192(10):1179-90.



Toujours des interrogations …

Composante inflammatoire nuancerait résultats ?

Contexte médical ou post-opératoire ?

Selon le phénotype de choc cardiogénique ?

Pré-emptif ? Car timing trop faible en précoce-tardif …

Traiter AKI ou casser cercle vicieux ?

THÉRAPEUTIQUES
Lévosimendan

↘ mortalité lors choc cardiogénique: **17,4** VS **23,1%** (*p=0,001*)

→ améliore DC et ↘ inotropes

→ via amélioration inotropisme VD ?

Landoni. Crit Care Med 2012; 40:634–646.

THÉRAPEUTIQUES
autres

Étude ASCEND-HF: Nésiritide = BNP recombiné

→ pas d'amélioration clinique

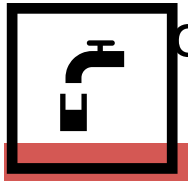
Fenoldopam = agoniste R dopa-1

↘ AKI lors CEC OR=0,46 [0,27-0,79] MAIS NS sur mortalité-durée ICU

Gillies. Crit Care. 2015 Dec 25;19:449.

CONCLUSION

Avant AKI:



optimiser perfusion → risques

Après AKI:

congestion ?  → déplétion + support VD

AUCUNE PREUVE

Supériorité diurétiques ou UF précoce ?