Les Hôpitaux Universitaires de STRASBOURG

Université de Strasbourg

CARDIOPATHIE URÉMIQUE ET INSUFFISANCE CARDIAQUE EN DIALYSE

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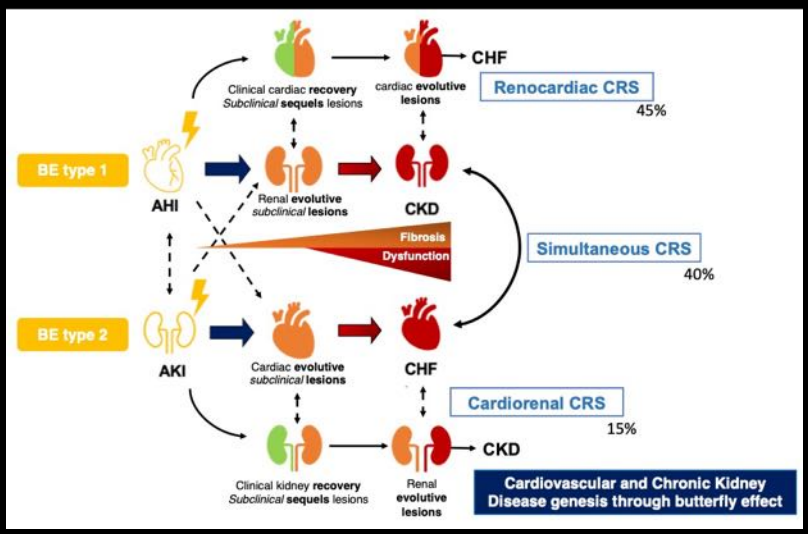
Service de néphrologie, dialyse, transplantation, Strasbourg

1

LE SYNDROME CARDIORÉNAL

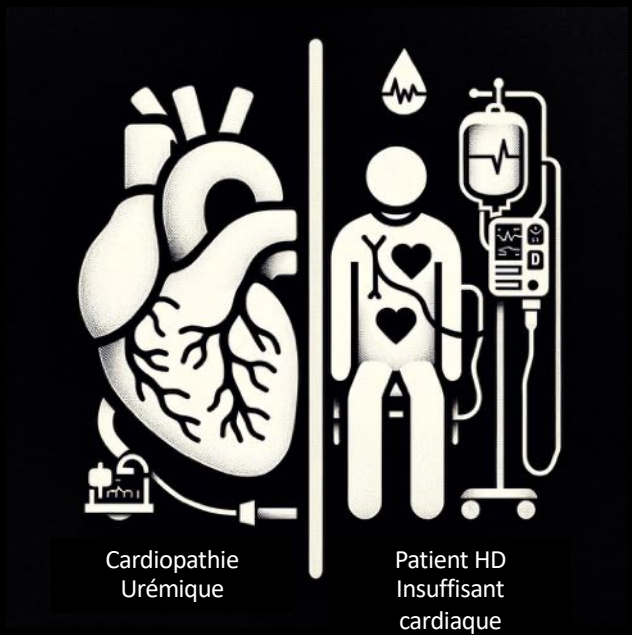
La maladie cardiovasculaire et rénale chronique



Bedo D, Beaudrey T and Florens N, In press

2

DEUX SITUATIONS EN OVERLAP



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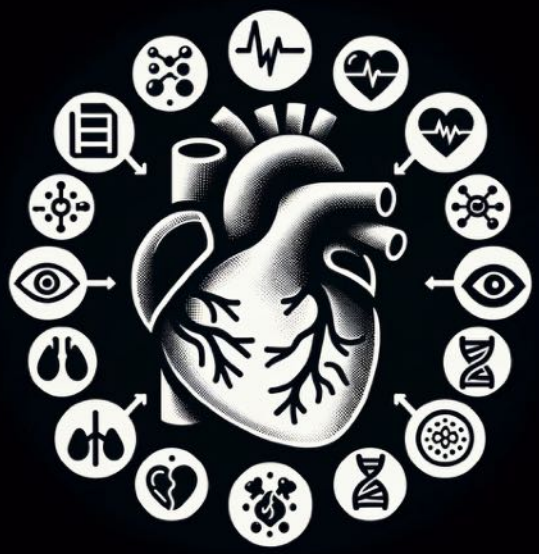
LA CARDIOPATHIE URÉMIQUE



•Définition : Cardiopathie associée à l'insuffisance rénale chronique et terminale, caractérisée par une hypertrophie ventriculaire gauche, une dysfonction diastolique, et une fibrose myocardique.

•Prévalence : Fréquente chez les patients avec maladie rénale chronique, en particulier ceux au stade terminal.

•Impact : Risque élevé de morbidité et de mortalité cardiovasculaires, incluant les arythmies, l'insuffisance cardiaque et la mort cardiaque subite.

4

LA CARDIOPATHIE URÉMIQUE

+ effets de l'hémodialyse

•Définition : Cardiopathie associée à l'insuffisance rénale chronique et terminale, caractérisée par une hypertrophie ventriculaire gauche, une dysfonction diastolique, et une fibrose myocardique.

ec maladie terminal.

•Impact : Risque élevé de morbidité et de mortalité cardiovasculaires, incluant les arythmies, l'insuffisance cardiaque et la mort cardiaque subite.

5

PHYSIOPATHOLOGIE

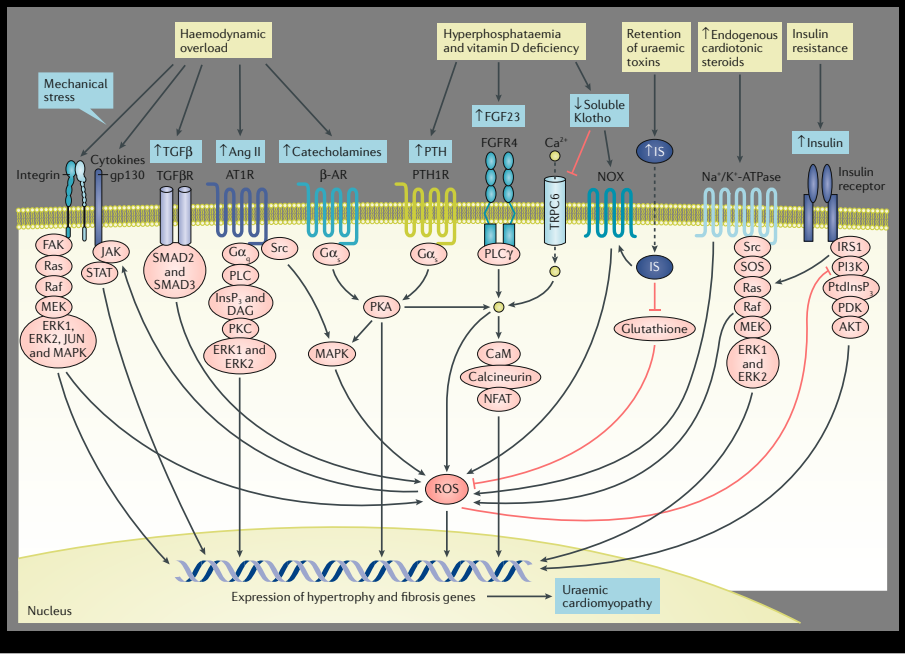
•Facteurs de Pathogenèse : Implique l'hypertension, l'hyperlipidémie, et le diabète; spécifiques à la CKD sont les toxines urémiques, l'anémie, l'hypervolémie, le stress oxydatif, l'inflammation, et la résistance à l'insuline.

•Dysfonctionnement Cardiaque : La cardiopathie urémique provoque une hypertrophie ventriculaire gauche, une dysfonction diastolique, une fibrose interstitielle diffuse et des dysfonctionnements systoliques et diastoliques.

•Métabolisme et Toxines Urémiques : Modifications du métabolisme cardiaque, impact des toxines urémiques, et déséquilibre du métabolisme minéral.

6

PHYSIOPATHOLOGIE

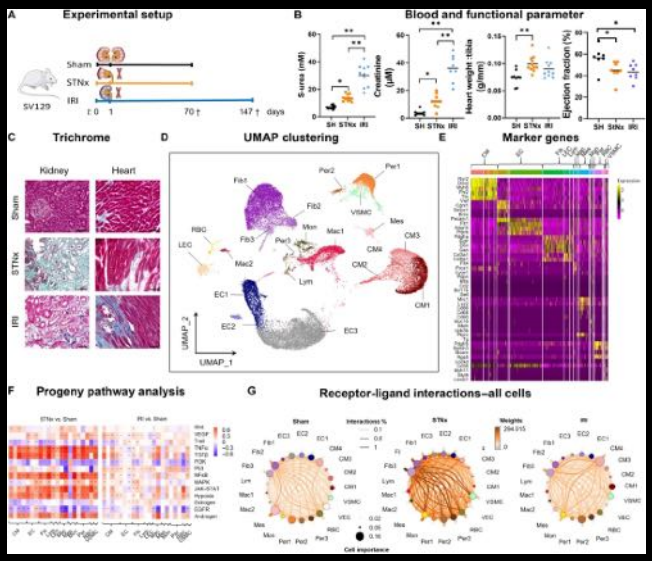


Evolving concepts in the p[illegible]genesis of uraemic cardiomyopathy

Xiaoliang Wang and Joseph I. Shapiro*

7

PHYSIOPATHOLOGIE



SCIENCE ADVANCES | RESEARCH ARTICLE

HEALTH AND MEDICINE

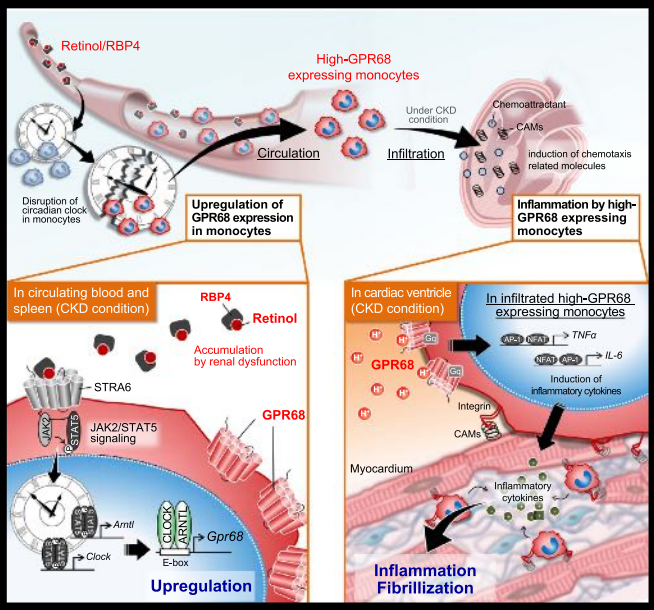
Mapping cardiac remodeling in chronic kidney disease

Nadine Kaesler[1,2†], Mingbo Cheng[3†], James Nagai[3], James O'Sullivan[4], Fabian Peisker[2], Eric M. J. Bindels[5], Anne Babler[2], Julia Moellmann[6], Patrick Droste[1,7], Giulia Franciosa[8], Aurelien Dugourd[9], Julio Saez-Rodriguez[9], Sabine Neuss[7,10], Michael Lehrke[6], Peter Boor[1,7], Claudia Goettsch[6], Jesper V. Olsen[8], Thimoteus Speer[11], Tzong-Shi Lu[12], Kenneth Lim[13], Jürgen Floege[1], Laura Denby[4], Ivan Costa[3‡], Rafael Kramann[1,2,14‡*]

Rôle de la voie JAK-STAT et du TNF-alpha

8

PHYSIOPATHOLOGIE



ARTICLE

https://doi.org/10.1038/s41467-021-23050-x OPEN

Alteration of circadian machinery in monocytes underlies chronic kidney disease-associated cardiac inflammation and fibrosis

Yuya Yoshida[1,6], Naoya Matsunaga[1,2,6], Takaharu Nakao[1], Kengo Hamamura[1], Hideaki Kondo[3], Tomomi Ide[4], Hiroyuki Tsutsui[4], Akito Tsuruta[1], Masayuki Kurogi[1], Michio Nakaya[5], Hitoshi Kurose[5], Satoru Koyanagi[1,2] & Shigehiro Ohdo[1,6]

9

DIAGNOSTIC

•Techniques d'Imagerie : Utilisation de l'échocardiographie, et de la résonance magnétique pour évaluer la structure et la fonction cardiaques.

•Biomarqueurs : Détection de biomarqueurs spécifiques pour évaluer le risque et la progression de la cardiopathie.

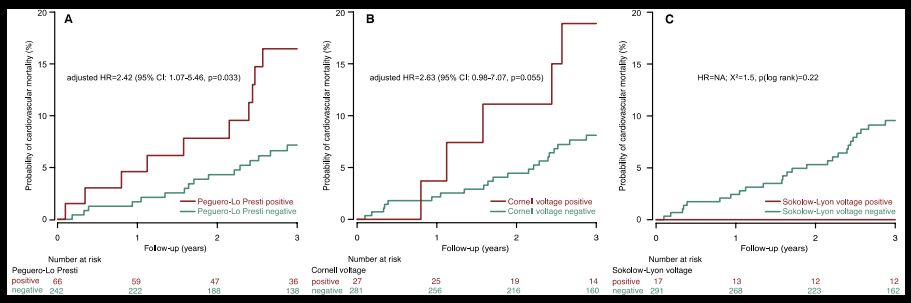
•Évaluation Clinique : Importance de l'évaluation clinique pour détecter les symptômes précoces et suivre la progression de la maladie.

10

ELECTROCARDIOGRAMME

Electrocardiographic parameters of left ventricular hypertrophy and prediction of mortality in hemodialysis patients

Matthias C. Braunisch[1] · Peter Gundel[1,2] · Stanislas Werfel[1] · Christopher C. Mayer[3] · Axel Bauer[4,5] · Bernhard Haller[6] · Roman Günthner[1] · Georg Lorenz[1] · Susanne Angermann[1] · Julia Matschkal[1] · Carolin Schaller[1] · Christopher Holzmann-Littig[1,7] · Stephan Kemmner[1,8] · Johannes Mann[9,10] · Axel Krieter[11] · Lutz Renders[1] · Siegfried Wassertheurer[3] · Georg Schmidt[12] · Uwe Heemann[1] · Marek Malik[13,14] · Christoph Schmaderer[1]



11

ELECTROCARDIOGRAMME

Electrocardiographic Predictors of Mortality and Sudden Cardiac Death in Patients with End Stage Renal Disease on Hemodialysis

Jonathan W. Waks, MD[1], Larisa G. Tereshchenko, MD, PhD[2], and Rulan S. Parekh, MD, MS[3]

Intervalle QT Prolongé
- Risque accru de Mort Subite Cardiaque (MSC) et de mortalité.
- Indicateur possible de pathologies cardiaques graves (ex : hypertrophie ventriculaire gauche).

Angle QRS-T Spatial
- Corrélation avec le risque cardiovasculaire.

ECG Moyenné par Signal (SAECG)
- Détection des anomalies subtiles du signal ECG.
- Lié aux risques d'arythmies.

Variabilité de la Fréquence Cardiaque (VFC)
- Réduction de la VFC associée à une mortalité plus élevée.
- Alternance de l'Onde T (TWA)
- Marqueur des arythmies ventriculaires et risque de MSC.

Limites
- Incertitude sur le lien direct entre ces anomalies ECG et la MSC.
- Difficultés dans l'application de la stratification du risque ECG en hémodialyse.

12

ECG ET ETT

Cardiac Disease in Chronic Uremia: Clinical Outcome and Risk Factors

Robert N. Foley and Patrick S. Parfrey

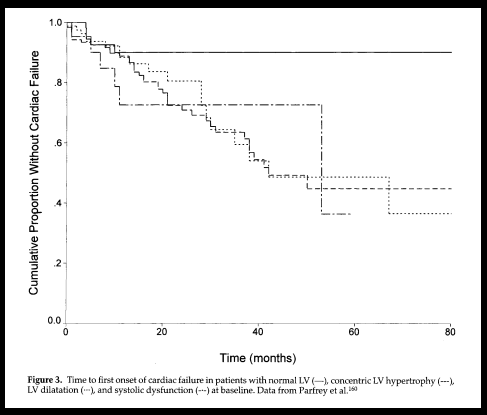


Figure 3. Time to first onset of cardiac failure in patients with normal LV (—), concentric LV hypertrophy (---), LV dilatation (··), and systolic dysfunction (-·-) at baseline. Data from Parfrey et al.[160]

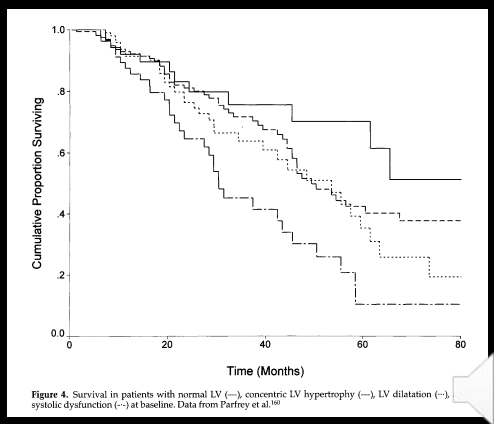


Figure 4. Survival in patients with normal LV (—), concentric LV hypertrophy (---), LV dilatation (··), systolic dysfunction (-·-) at baseline. Data from Parfrey et al.[160]

13

FEVG

Prognostic Value of Reduced Left Ventricular Ejection Fraction at Start of Hemodialysis Therapy on Cardiovascular and All-Cause Mortality in End-Stage Renal Disease Patients

Shigeki Yamada,* Hideki Ishii,† Hiroshi Takahashi,‡ Toru Aoyama,‡ Yasuhiro Morita,† Hirotake Kasuga,* Keiko Kimura,* Yutaka Ito,* Ryo Takahashi,* Takanobu Toriyama,‡ Yoshinari Yasuda,§ Mutsuharu Hayashi,§ Hideki Kamiya,§ Yukio Yuzawa,‖ Shoichi Maruyama,‖ Seiichi Matsuo,‖ Tatsuaki Matsubara,¶ and Toyoaki Murohara†

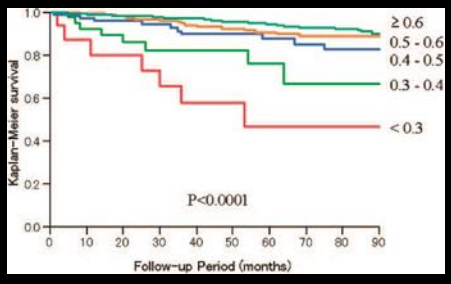


Figure 1. Kaplan-Meier estimates: Event-free survival from cardiovascular death.

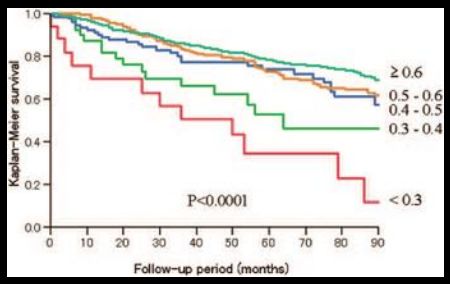


Figure 2. Kaplan-Meier estimates: Event-free survival from all-cause death.

14

DYSFONCTION DIASTOLIQUE

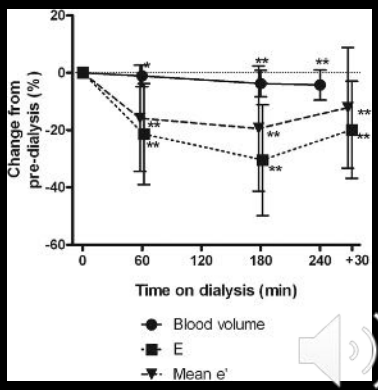
Changes in Left Ventricular Diastolic Function During Hemodialysis Sessions

Solmaz Assa, MD,[1] Yoran M. Hummel, BSc,[2] Adriaan A. Voors, MD, PhD,[2] Johanna Kuipers,[3] Henk Groen, MD, PhD,[4] Paul E. de Jong, MD, PhD,[1] Ralf Westerhuis, MD, PhD,[1,3] and Casper F.M. Franssen, MD, PhD[1]

	Predialysis	60-min Intradialysis	180-min Intradialysis	30-min Postdialysis	P[a]
E (m/s)	0.93 ± 0.24	0.71 ± 0.22[b]	0.63 ± 0.20[b]	0.73 ± 0.23[b]	<0.001
A (m/s)	0.86 ± 0.24	0.82 ± 0.21[c]	0.80 ± 0.2[b]	0.84 ± 0.23	0.001
E:A	1.11 ± 0.34	0.88 ± 0.28[b]	0.82 ± 0.37[b]	0.88 ± 0.28[b]	<0.001
DT (s)	224 ± 83	258 ± 86[b]	282 ± 94[b]	253 ± 70[b]	<0.001
IVRT (s)	94 ± 25	115 ± 33[b]	112 ± 35[b]	107 ± 31[b]	<0.001
Mean e′ (cm/s)	6.6 ± 2.1	5.6 ± 2.2[b]	5.3 ± 2.0[b]	5.8 ± 2.0[b]	<0.001
E:e′	15.1 ± 5.6	14.3 ± 7.1[d]	13.6 ± 7.3[b]	14.0 ± 6.9[b]	<0.001

	Normal	DD grade I	DD grade II	DD grade III
E/*A* ratio	0.8–2	< 0.8	0.8–2	≥2
e′ (cm/second)	> 8	< 8	< 8	< 8
Average *E*/*e*′	< 8	≤8	9–12	≥13
LAVi (ml/m²)	< 28	< 34	≥34	> 34
Ar – A (ms)	< 30	< 30	≥30	> 30

E, early mitral flow velocity; *A*, atrial mitral flow velocity; *e*′, early mitral annulus velocity; LAVi, left atrium volume index; Ar – A, time difference between duration of pulmonary venous atrial reversal wave and duration of A wave.

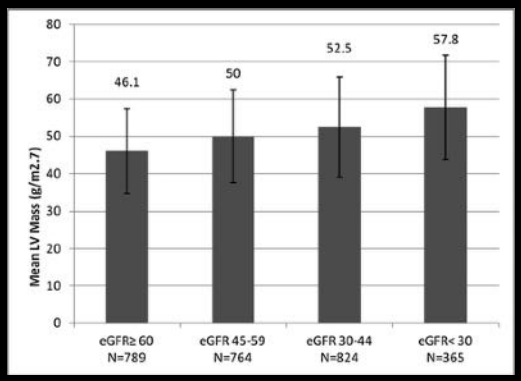


15

HVG ET MASSE VG

Associations between Kidney Function and Subclinical Cardiac Abnormalities in CKD

Meyeon Park,* Chi-yuan Hsu,* Yongmei Li,† Rakesh K. Mishra,‡ Martin Keane,§ Sylvia E. Rosas,‖ Daniel Dries,§ Dawei Xie,¶ Jing Chen,** Jiang He,†† Amanda Anderson,¶ Alan S. Go,‡‡§§ Michael G. Shlipak,†§§ and Chronic Renal Insufficiency Cohort (CRIC) Study Group



- Prévalence de l'hypertrophie VG :
  - 40 à 80% en prédialyse
  - > 90% en dialyse
- HVG et masse VG associées à
  - Augmentation du risque d'arrythmies A et V
  - Mortalité toute cause et mort subite

16

HVG ET MASSE VG

Is left ventricular hypertrophy a powerful predictor of progression to dialysis in chronic kidney disease?

Ernesto Paoletti[1], Diego Bellino[1], Anna Maria Gallina[2], Marco Amidone[1], Paolo Cassottana[3] and Giuseppe Cannella[1]

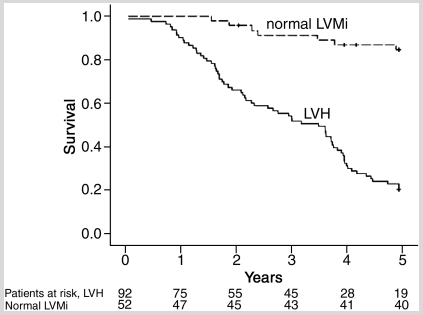


Fig. 1. Kaplan–Meier analysis of combined end point-free survival in patients with LVH and in those with normal LVMi at baseline (log-rank $P < 0.001$).

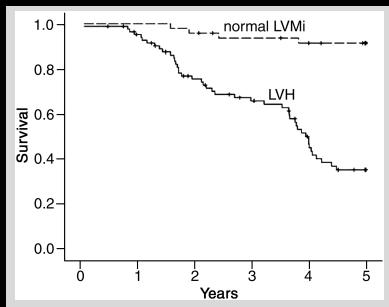


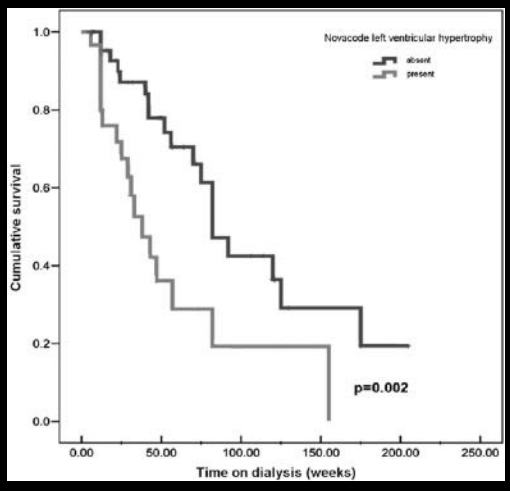
Fig. 2. Kaplan–Meier analysis of dialysis-free survival in patients with LVH and in those with normal LVMi at baseline (log-rank $P < 0.001$).

17

HVG ET MASSE VG

The Prognostic Value of Electrocardiographic Estimation of Left Ventricular Hypertrophy in Dialysis Patients

Adrian C. Covic, M.D., Ph.D., F.R.C.P. (London), F.E.R.A.,*† Laura-Dumitriţa Buimistriuc, M.D.,† Darren Green, M.D.,‡§ Alina Stefan, M.D.,† Silvia Badarau , M.D.,† and Philip A. Kalra, M.D., F.R.C.P‡§

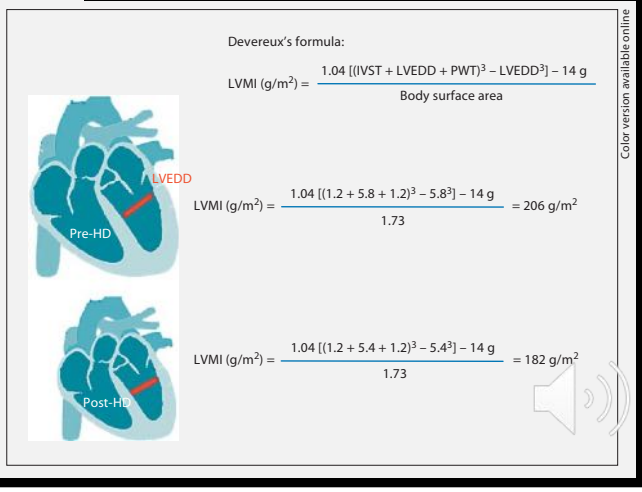


18

HVG ET MASSE VG

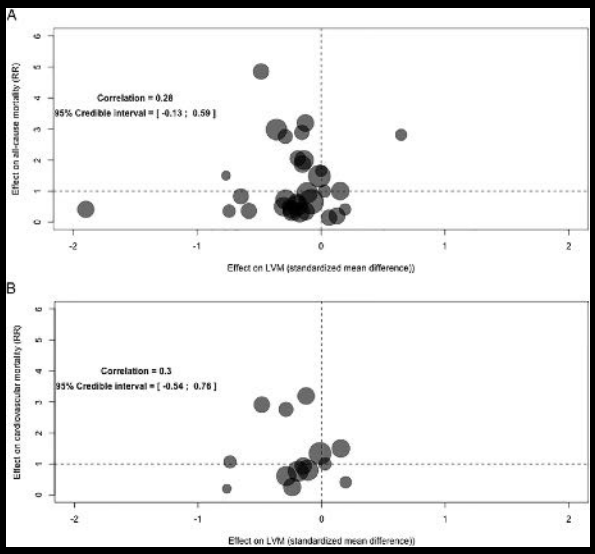
Left Ventricular Mass Index as an Outcome Measure in Clinical Trials in Dialysis Patients: A Word of Caution

Carmine Zoccali



19

HVG ET MASSE VG



The Validity of Left Ventricular Mass as a Surrogate End Point for All-Cause and Cardiovascular Mortality Outcomes in People With CKD: A Systematic Review and Meta-analysis

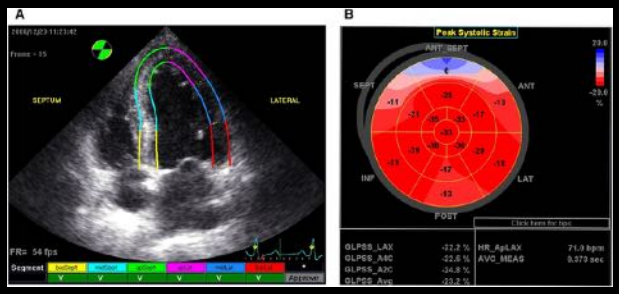
Sunil V. Badve, MD,[1,2,3,4] Suetonia C. Palmer, PhD,[1,5] Giovanni F.M. Strippoli, PhD,[6,7,8] Matthew A. Roberts, PhD,[1,9] Armando Teixeira-Pinto, PhD,[8] Neil Boudville, M Med Sci,[1,10] Alan Cass, PhD,[1,11] Carmel M. Hawley, M Med Sci,[1,2,12] Swapnil S. Hiremath, MD, MPH,[13,14] Elaine M. Pascoe, M Biostat,[1,2] Vlado Perkovic, PhD,[1,4] Gillian A. Whalley, PhD,[15] Jonathan C. Craig, PhD,[1,6,16] and David W. Johnson, MD, PhD[1,2,12]

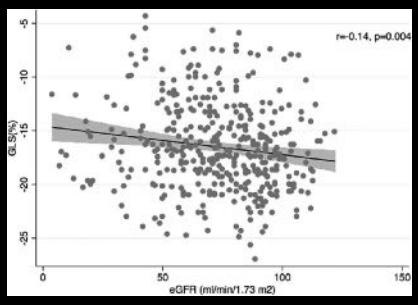
20

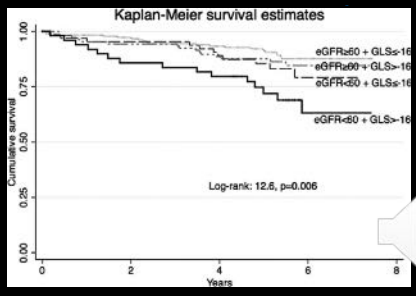
GLS : MESURE DE LA DÉFORMATION

The association between left ventricular global longitudinal strain, renal impairment and all-cause mortality

Rathika Krishnasamy[1], Nicole M. Isbel[1], Carmel M. Hawley[1], Elaine M. Pascoe[2], Rodel Leano[3], Brian A. Haluska[3] and Tony Stanton[3]





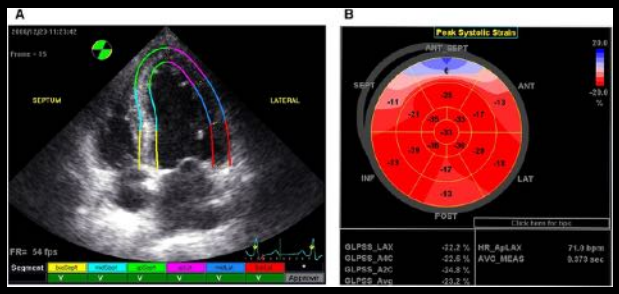


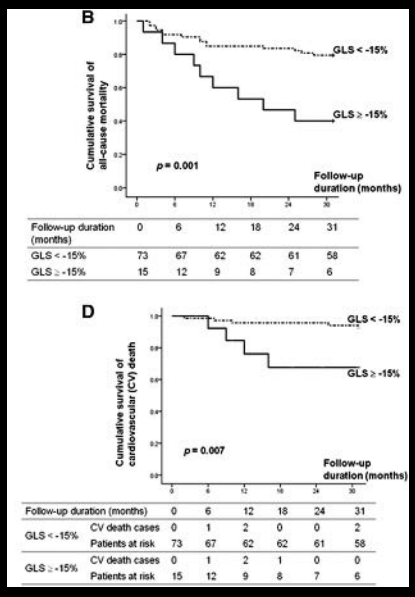
21

GLS : MESURE DE LA DÉFORMATION

Association of Left Ventricular Longitudinal Strain with Mortality among Stable Hemodialysis Patients with Preserved Left Ventricular Ejection Fraction

Yen-Wen Liu, Chi-Ting Su,† Junne-Ming Sung,‡ Saprina P.H. Wang,§ Yu-Ru Su,‖ Chun-Shin Yang,¶ Liang-Miin Tsai,* Jyh-Hong Chen,* and Wei-Chuan Tsai**



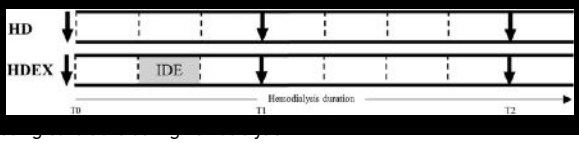


22

GLS : MESURE DE LA DÉFORMATION

Cardioprotective Effect of Acute Intradialytic Exercise: A Comprehensive Speckle-Tracking Echocardiography Analysis

Matthieu Josse,[1] Laure Patrier,[2,3] Myriam Isnard,[4] Cécile Turc-Baron,[2,5] Antoine Grandperrin,[1] Stéphane Nottin,[1] Stéphane Mandigout,[6] Jean-Paul Cristol,[2,5,7] Claire Maufrais,[1] and Philippe Obert[1]



Variable	HD			HDEX			Linear Mixed-Effects Model: Condition×Sequence	Linear Mixed-Effects Model: Condition×Time		
	T0	T1	T2	T0	T1	T2	*P* Value	Estimated Difference[a]	95% CI	*P* Value
Primary outcome										
GLS (%)	−18.2±3.1	−16.2±3.3	−15.2±3.7	−18.1±3.0	−16.8±3.5	−16.3±3.4	0.94	−1.16	−0.31 to −2.02	0.009
P value[b]		<0.001	<0.001		<0.001	0.007				
		<0.001			<0.001					

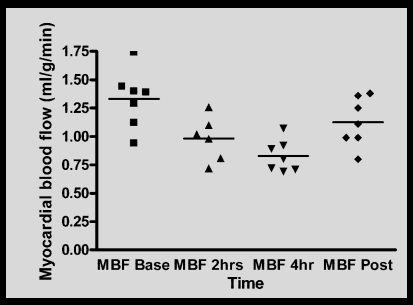
23

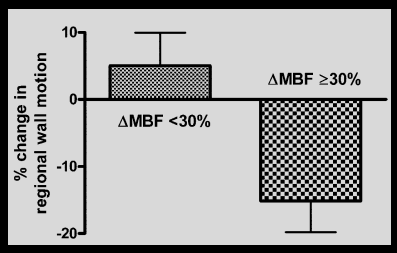
ANOMALIES DE CONTRACTIONS RÉGIONALES

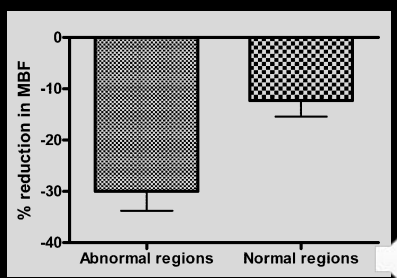
Hemodialysis-Induced Cardiac Dysfunction Is Associated with an Acute Reduction in Global and Segmental Myocardial Blood Flow

Christopher W. McIntyre,*† James O. Burton,* Nicholas M. Selby,* Lucia Leccisotti,‡ Shvan Korsheed,* Christopher S.R. Baker,‡ and Paolo G. Camici‡

•Reliées à une ischémie myocardique per-HD





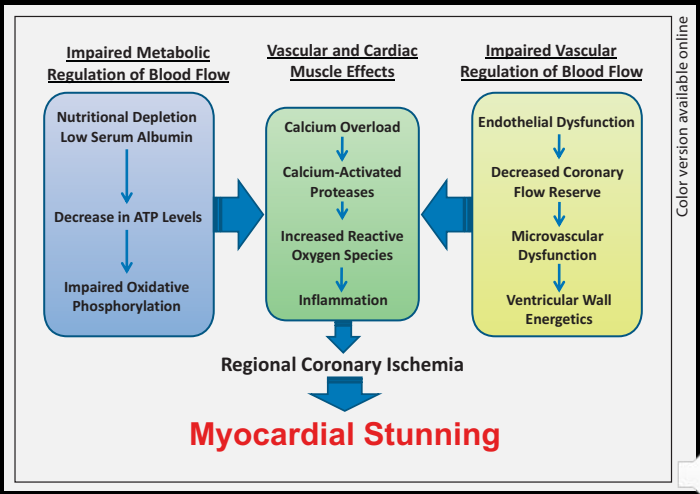


24

SIDÉRATION MYOCARDIQUE

Myocardial Stunning with Hemodialysis: Clinical Challenges of the Cardiorenal Patient

Mozow Y. Zuidema[a,c] Kevin C. Dellsperger[a–c]

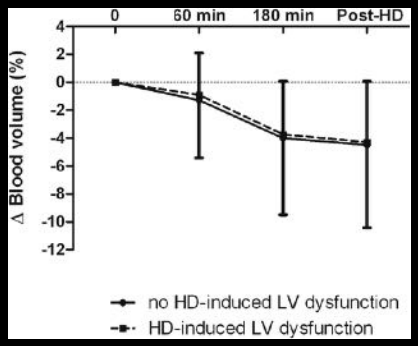


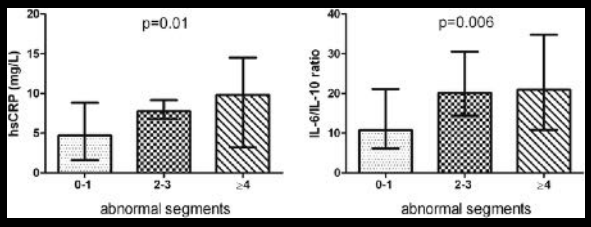
25

SIDÉRATION MYOCARDIQUE

Hemodialysis-Induced Regional Left Ventricular Systolic Dysfunction and Inflammation: A Cross-sectional Study

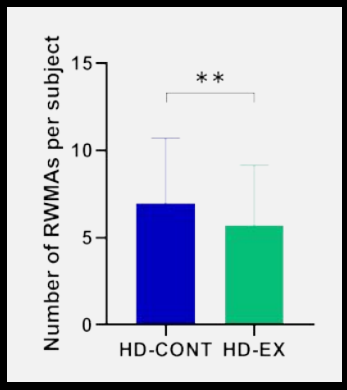
Solmaz Assa, MD,[1] Yoran M. Hummel, BSc,[2] Adriaan A. Voors, MD, PhD,[2] Johanna Kuipers,[3] Ralf Westerhuis, MD, PhD,[1,3] Henk Groen, MD, PhD,[4] Stephan J.L. Bakker, MD, PhD,[1] Anneke C. Muller Kobold, PhD,[5] Wim van Oeveren, PhD,[6,7] Joachim Struck, PhD,[8] Paul E. de Jong, MD, PhD,[1] and Casper F.M. Franssen, MD, PhD[1]

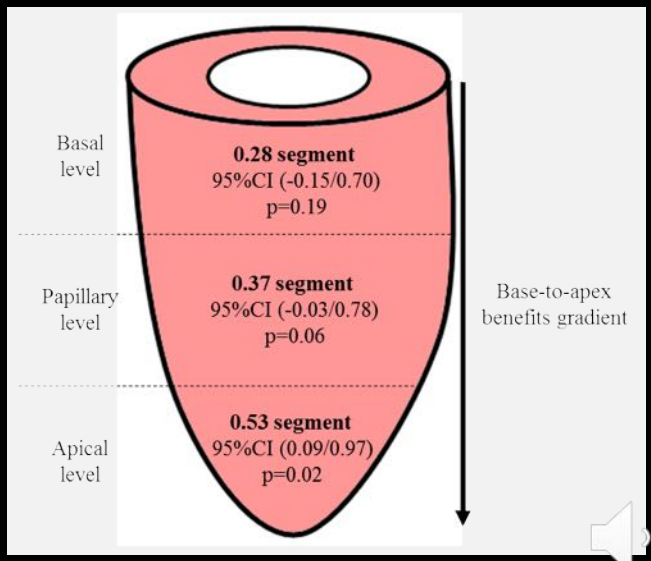




26

SIDÉRATION MYOCARDIQUE





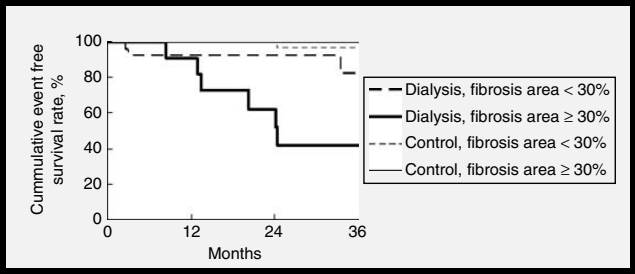
Josse et al, in press

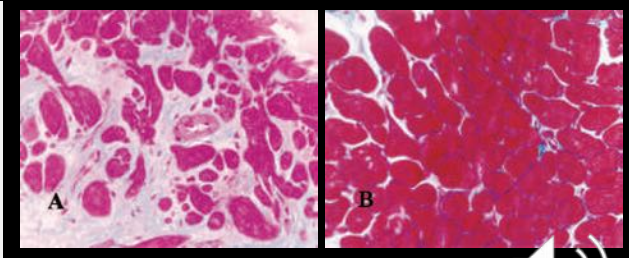
27

BIOPSIE MYOCARDIQUE

Clinical and pathologic characteristics of dilated cardiomyopathy in hemodialysis patients

Jiro Aoki, Yuji Ikari, Hiroyoshi Nakajima, Masaya Mori, Tokuichiro Sugimoto, Mitsuharu Hatori, Shuzou Tanimoto, Eisuke Amiya, and Kazuhiro Hara



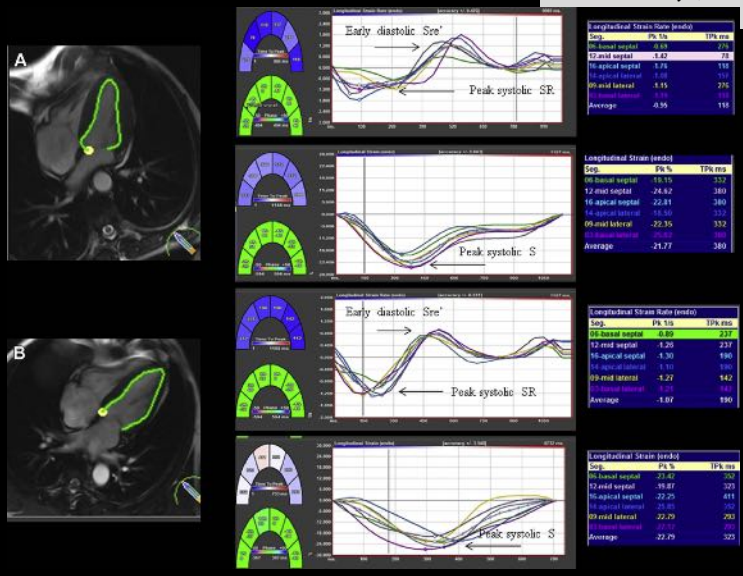


28

IRM CARDIAQUE

Diffuse Interstitial Fibrosis and Myocardial Dysfunction in Early Chronic Kidney Disease

Nicola C. Edwards, PhD[a,b,*], William E. Moody, MBChB[a,b], Mengshi Yuan, MBChB[b], Manvir K. Hayer, MBChB[c], Charles J. Ferro, MD[a,c], Jonathan N. Townend, MD[a,b], and Richard P. Steeds, MD[a,b]

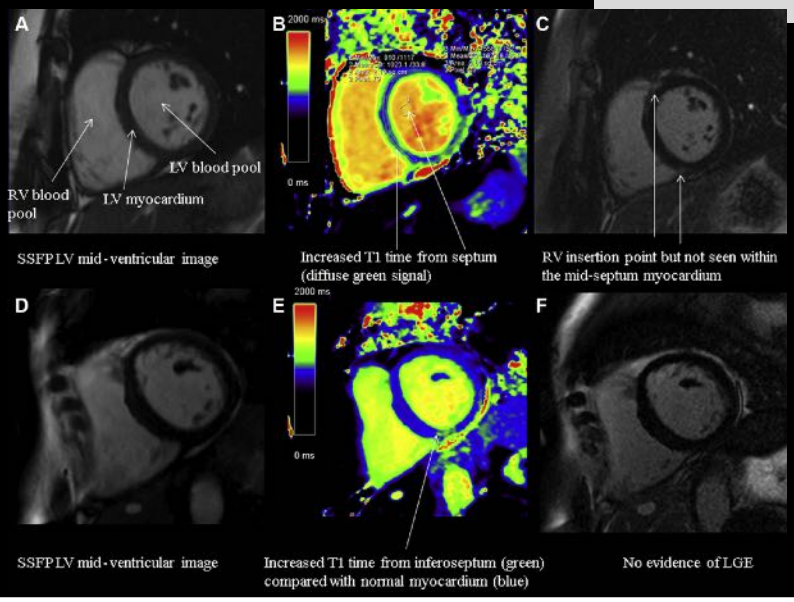


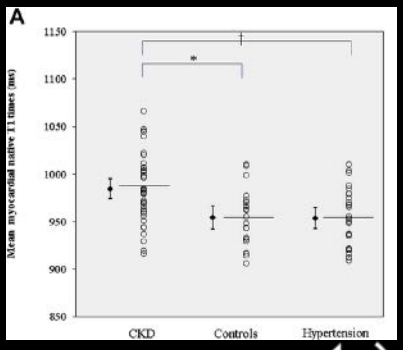
29

IRM CARDIAQUE

Diffuse Interstitial Fibrosis and Myocardial Dysfunction in Early Chronic Kidney Disease

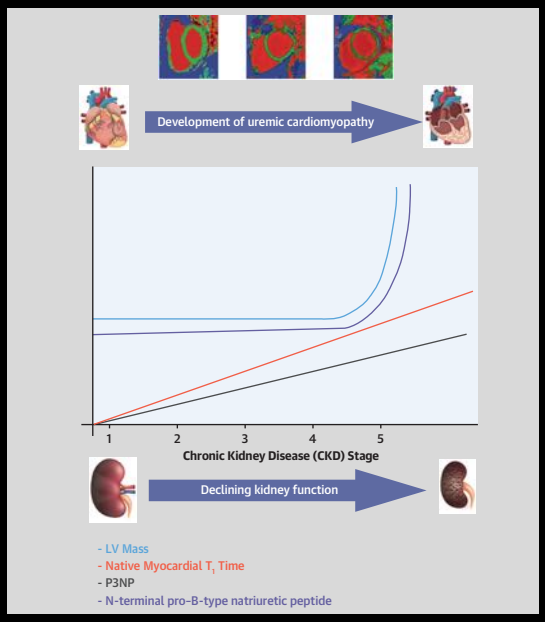
Nicola C. Edwards, PhD[a,b,*], William E. Moody, MBChB[a,b], Mengshi Yuan, MBChB[b], Manvir K. Hayer, MBChB[c], Charles J. Ferro, MD[a,c], Jonathan N. Townend, MD[a,b], and Richard P. Steeds, MD[a,b]





30

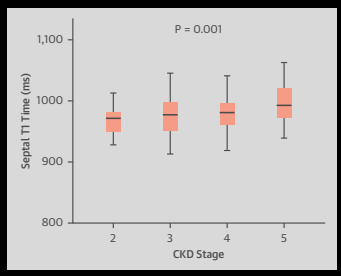
IRM CARDIAQUE

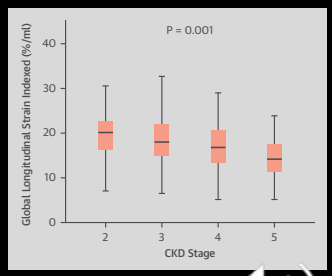


Defining Myocardial Abnormalities Across the Stages of Chronic Kidney Disease

A Cardiac Magnetic Resonance Imaging Study

Manvir K. Hayer, MBChB,[a,b] Ashwin Radhakrishnan, BMBS,[a,c] Anna M. Price, MBChB,[a,b] Boyang Liu, MBBS,[a,c] Shanat Baig, MBBS,[a,c] Christopher J. Weston, PhD,[d,e] Luca Biasiolli, DPhil,[f] Charles J. Ferro, MD,[a,b] Jonathan N. Townend, MD,[a,c] Richard P. Steeds, MD,[a,c] Nicola C. Edwards, PhD,[a,g] on behalf of the Birmingham Cardio-Renal Group*





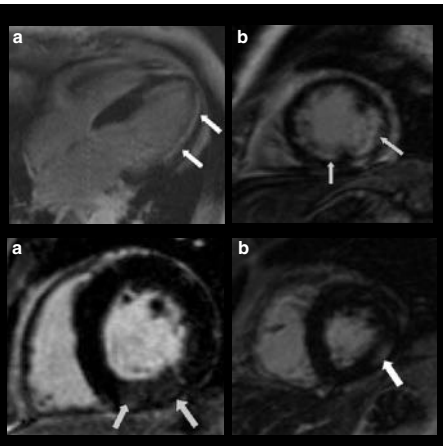
31

IRM CARDIAQUE

Redefinition of uremic cardiomyopathy by contrast-enhanced cardiac magnetic resonance imaging

PB Mark[1,2], N Johnston[3], BA Groenning[3], JE Foster[3], KG Blyth[3], TN Martin[3], T Steedman[3], HJ Dargie[3] and AG Jardine[1,2]

Variable	All patients	Gadolinium negative	Gadolinium positive	Subendocardial gadolinium	*Diffuse Gadolinium*
LVH (%)	96 (71.6)	61 (63.5)	35 (92.1)**	17 (89.5) *	18 (94.7)***
LV dilatation (%)	15 (11.2)	5 (5.2)	10 (27.7)***	7 (36.8)***	3 (15.8)
LVSD (%)	11 (8.2)	0 (0)	11 (57.9)**	8 (44.4)**	3 (15.8)**
EF (%) (s.d.)	68.5±9.9	70.5±7.0	63.6±13.8*	59.7±13.9**	67.5±12.9
LV mass (g)	172.3 (67.8)	154 (64.0)	186.5 (50.8)**	185.0 (66.0)*	195 (47)***
LV mass/BSA (g/m^2)	94.4 (32.0)	84.4 (31.0)	104.0 (19.9)**	100.5 (27.9)	105.0 (16.0)***
ESV (ml)	43.1 (30.2)	34.2 (21.5)	57.7 (41.7)**	64.0 (33.0)**	50.7 (34.3)*
ESV/BSA (ml/m^2)	23.5 (14.0)	19.0 (12.5)	30 (21.9)**	35.5 (21.0)**	22 (17.9)
EDV (ml)	131.8 (64.6)	120 (56.8)	152.5 (67.5)**	174.0 (57.0)***	140.0 (78.0)*
EDV/BSA (ml/m^2)	71.9 (30.8)	69.0 (29.7)	84.0 (32.3)***	89.6 (32.5)**	74.0 (31.8)
Mass of LGE	—	0	9.0±4.9	9.3±6.2	8.7±3.4
Signal intensity of myocardium	—	6.6±1.7	24.4±12.3**	30.6±13.6**	18.2±6.9**
Signal intensity index	—	1	3.8±1.9	2.8±0.10**	5.3±1.8**
Normal angiogram (%)	20 (40.8)	13 (56.5)	7 (26.9)	0 (0)	7 (58.3)
No. of patients with mild/moderate CAD (%)	10 (20.4)	6 (26.1)	4 (15.4)	0 (0)	4 (33.3)
No. of patients with severe CAD (%)	19 (38.8)	4 (17.4)	15 (57.7)***	14 (100)**	1 (8.3)



32

BIOMARQUEURS : BNP/NT_{PRO}BNP

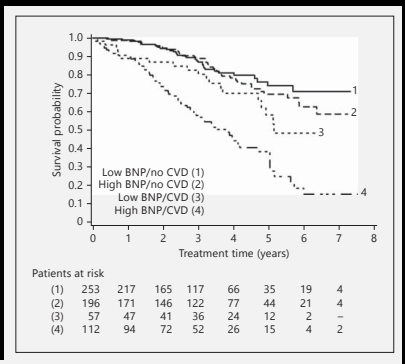
N-Terminal Pro–B-Type Natriuretic Peptide Variability in Stable Dialysis Patients

Magid A. Fahim,[a,e] Andrew Hayen,[b] Andrea R. Horvath,[f,g] Goce Dimeski,[f,e] Amanda Coburn,[a] David W. Johnson,[a,e] Carmel M. Hawley,[a,e] Scott B. Campbell,[a] and Jonathan C. Craig[d,e]

Table 1. (Continued)

Characteristic	Value (*n*=55)
NT-proBNP (pg/ml)	
Median (interquartile range)	1698 (718–3742)
Distribution (%)	
<300	7
300–899	20
900–4999	51
5000–19,999	11
≥20,000	11

D/P creatinine, ratio of creatinine concentration in dialysate to plasma; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin type 1 receptor blocker; NT-proBNP, N-terminal pro–B-type natriuretic peptide.



The Relationship of NT-proBNP and Dialysis Parameters with Outcome of Incident Haemodialysis Patients: Results from the Membrane Permeability Outcome Study

Francesco Locatelli[a] Thierry Hannedouche[b] Alejandro Martin-Malo[c] Stefan H. Jacobson[d] Raymond Vanholder[e] Claudio Ronco[f] Vincenzo La Milia[a] Juan M. Lopez Gomez[g] Sergio Stefoni[h] Hervé Maheut[i] Marian Klinger[j] Thierry Krummel[b] Annemie Dhondt[e] Isabel Berdud[c] Adelheid Gauly[k] for the Membrane Permeability Outcome (MPO) Study Group

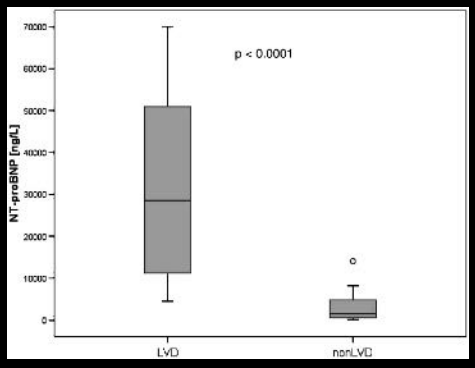
	n	NT-proBNP (pg/ml) mean ± SD	median (min.–max.)	p value
All patients	618	6,224±8,953	2,124 (5–35,000)	
High-flux	301	5,100±7,827	1,854 (5–35,000)	<0.001
Low-flux	317	7,291±9,798	2,919 (88–35,000)	
Serum albumin ≤4 g/dl	470	6,443±9,159	2,367 (5–35,000)	0.07
Serum albumin >4 g/dl	148	5,330±8,253	1,833 (88–35,000)	
Albumin ≤4 g/dl				
High-flux	235	5,139±7,962	1,829 (5–35,000)	.001
Low-flux	235	7,746±10,066	3,402 (110–35,000)	
Albumin >4 g/dl				
High-flux	66	4,963±7,380	1,971 (134–35,000)	0.71
Low-flux	82	5,987±8,913	1,778 (88–35,000)	

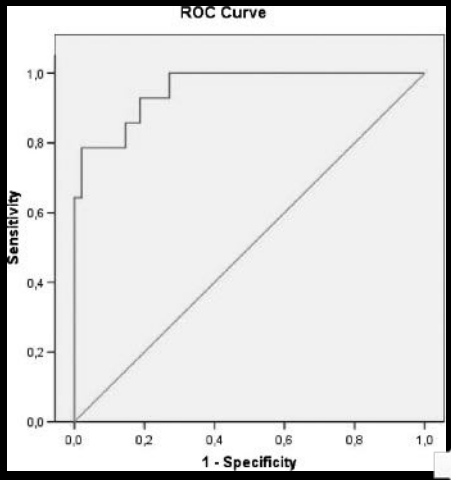
33

BIOMARQUEURS : BNP/NT_{PRO}BNP

Diagnostic value of N-terminal pro-B-type natriuretic peptide (NT-proBNP) for left ventricular dysfunction in patients with chronic kidney disease stage 5 on haemodialysis

Sascha David[1], Philipp Kümpers[1], Vega Seidler[1], Frank Biertz[2], Hermann Haller[1] and Danilo Fliser[1]



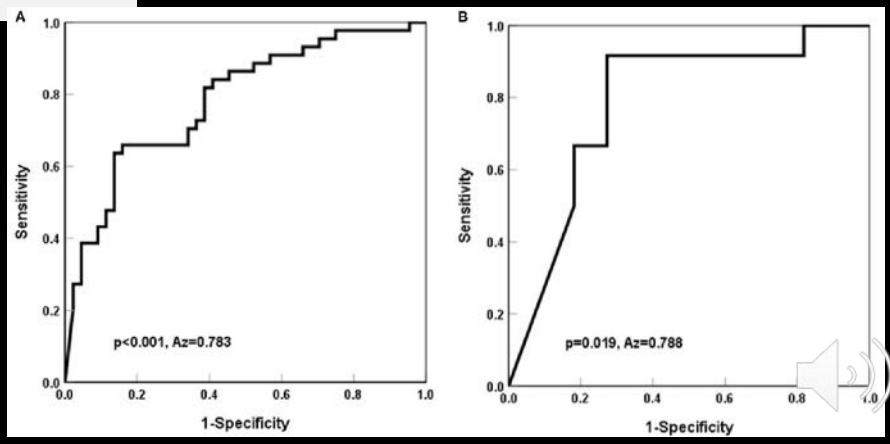


34

BIOMARQUEURS : BNP/NT_{PRO}BNP

Association of N-Terminal Pro-brain Natriuretic Peptide With Volume Status and Cardiac Function in Hemodialysis Patients

Yaqiong Wang [1,2,3,4,5], Xuesen Cao [1,2,3,4,5], Jinbo Yu [1,2,3,4,5], Yongmei Zhang [1,2,3,4,5], Xianzhe Li [1,2,3,4,5], Xiaohong Chen [1,2,3,4,5], Jianzhou Zou [1,2,3,4,5], Bo Shen [1,2,3,4,5]† and Xiaoqiang Ding [1,2,3,4,5]†

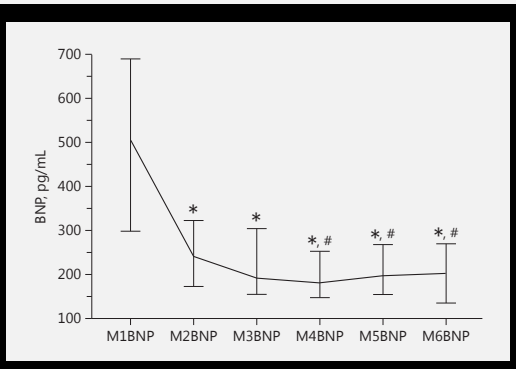


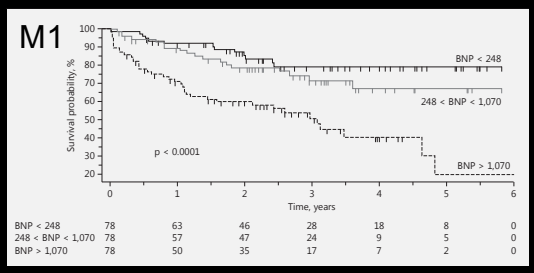
35

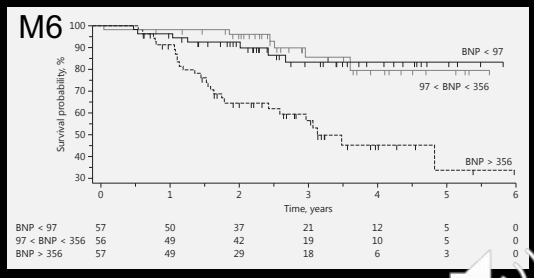
BIOMARQUEURS : BNP/NT_{PRO}BNP

Brain Natriuretic Peptide Is a Marker of Fluid Overload in Incident Hemodialysis Patients

Charles Chazot[a, b] Margaux Rozes[a] Cyril Vo-Van[a] Patrik Deleaval[a] Jean-Marc Hurot[a] Christie Lorriaux[a] Brice Mayor[a] Eric Zaoui[c] Guillaume Jean[a]



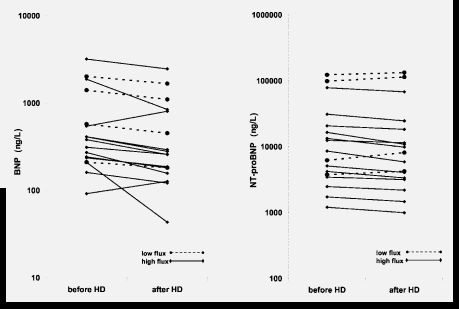




36

BIOMARQUEURS : BNP/NT_{PRO}BNP

Elimination of the Cardiac Natriuretic Peptides B-Type Natriuretic Peptide (BNP) and N-Terminal proBNP by Hemodialysis, *Hans Günther Wahl,[1*] Stephanie Graf,[2] Harald Renz,[1] and Winfried Fassbinder[2]* ([1] Klinikum der Philipps-Universität Marburg, Department of Clinical Chemistry and Molecular Diagnostics, 35033 Marburg, Germany; [2] Klinikum Fulda, Department of Internal Medicine III, Fulda, Germany; * author for correspondence: fax 49-6421-2865594, e-mail hg.wahl@med.uni-marburg.de)



	All (n = 46)		Low-flux membranes (n = 21)		High-flux membranes (n = 25)	
	Pre-HD	Post-HD	Pre-HD	Post-HD	Pre-HD	Post-HD
NT-proBNP, pg/mL	5615 (2344–24,637)	5199* (1420–26,352)	7425 (2536–29,626)	8033* (2872–33,525)	5577 (2328–22,918)	3817** (1408–16,521)
Albumin, g/L	35.3 (27.2–39.8)	38.0** (33.1–46.5)	33.6 (26.9–36.7)	37.0** (31.6–43.0)	36.0 (29.9–39.8)	39.1** (35.8–47.6)

Values are shown as median and 10th–90th percentiles distribution. *p < 0.05, **p < 0.01 vs. pre-HD.

Influence of haemodialysis on the NT-proBNP plasma concentration

37

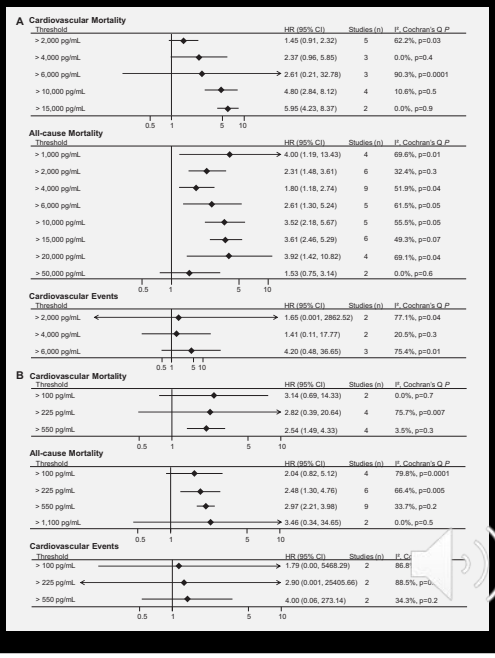
BIOMARQUEURS : BNP/NT_{PRO}BNP

Association of NT-proBNP and BNP With Future Clinical Outcomes in Patients With ESKD: A Systematic Review and Meta-analysis

Tyrone G. Harrison, Caley B. Shukalek,* Brenda R. Hemmelgarn, Kelly B. Zarnke, Paul E. Ronksley, Nicolas Iragorri, Michelle M. Graham, and Matthew T. James*

	Pooled Unadjusted HRs (95% CI) for Peptide Thresholds		
	CV Mortality	All-Cause Mortality	CV Events
NT-proBNP threshold			
> 1,000 pg/mL		4.00 (1.19-13.44)	
> 2,000 pg/mL	1.45 (0.91-2.32)	2.31 (1.48-3.61)	1.65 (0.001-2,862.5)
> 4,000 pg/mL	2.37 (0.96-5.85)	1.80 (1.18-2.74)	1.45 (0.11-17.77)
> 6,000 pg/mL	2.61 (0.21-32.78)	2.61 (1.30-5.24)	4.20 (0.48-36.65)
> 10,000 pg/mL	4.80 (2.84-8.12)	3.52 (2.18-5.67)	
> 15,000 pg/mL	5.95 (4.23-8.37)	3.61 (2.46-5.29)	
> 20,000 pg/mL		3.92 (1.42-10.82)	
> 50,000 pg/mL		1.53 (0.75-3.14)	
BNP threshold			
> 100 pg/mL	3.14 (0.69-14.33)	2.04 (0.82-5.12)	1.79 (0.01-5,468.28)
> 225 pg/mL	2.82 (0.39-20.64)	2.48 (1.30-4.76)	2.90 (0.001-25,405.66)
> 550 pg/mL	2.54 (1.49-4.33)	2.97 (2.21-3.98)	4.00 (0.06-273.14)
> 1,100 pg/mL		3.46 (0.35-34.65)	

Note: Pooled unadjusted HRs are presented along with 95% CIs for both NT-proBNP and BNP at peptide-specific thresholds. Threshold-specific HRs were estimated using restricted maximum likelihood random-effects meta-analysis and CIs corrected for between-study variance with the Hartung-Knapp-Sidik-Jonkman method.[28]
Abbreviations: BNP, brain natriuretic peptide; CI, confidence interval; CV, cardiovascular; HR, hazard ratio; NT-proBNP, N-terminal pro-BNP.

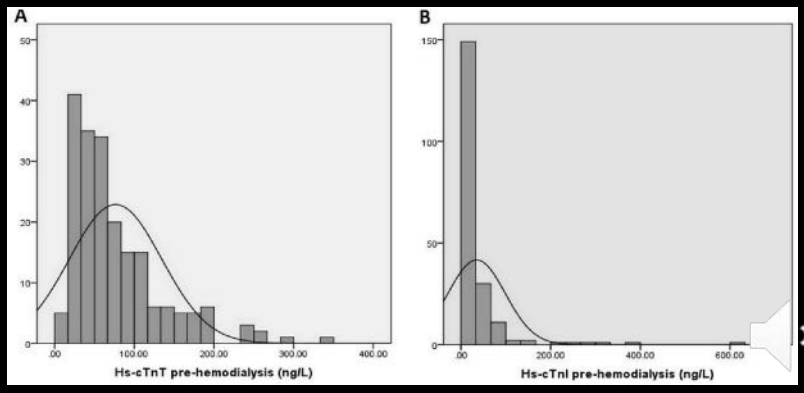


38

BIOMARQUEURS : TROPONINE

Variability of high-sensitivity cardiac troponin T and I in asymptomatic patients receiving hemodialysis

Wanwarang Wongcharoen[1], Teetad Chombandit[1], Arintaya Phrommintikul[1] & Kajohnsak Noppakun[2,3]✉



39

BIOMARQUEURS : TROPONINE

Effect of hemodialysis on levels of high-sensitivity cardiac troponin T

Michael Chen, MD MSc[1]; Howard Gerson, MD[1]; Shaun Eintracht, MD[1]; Sharon J. Nessim, MD[2]; Elizabeth MacNamara, MD[1]

Table 1. Effect of hemodialysis on levels of high-sensitivity cardiac troponin T

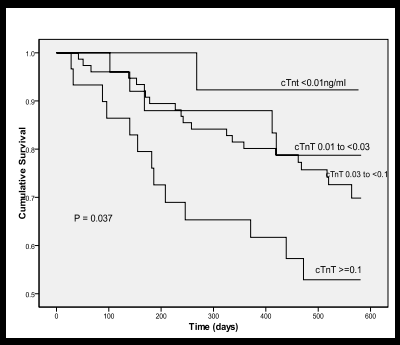
			Variation during dialysis (%)			Variation immediately post-dialysis (%)		
Age	Sex	Baseline troponin (ng/L)	Total protein	Creatinine	Troponin	Total protein	Creatinine	Troponin
64	M	44	3	-46	-7	4	-65	-2
64	F	52	2	-53	-15	2	-65	-21
71	F	55	2	-23	-18	5	-49	-20
83	F	61	0	-53	-10	-1	-70	-8
75	M	100	5	-45	-14	6	-62	-18
79	M	113	5	-47	-27	6	-63	-34
78	M	142	-1	-42	-5	6	-53	-3
89	M	148	4	-51	1	7	-70	-3
80	M	149	2	-47	-4	4	-62	-6
88	M	250	3	-50	-8	6	-55	-5
Mean % change (95% confidence interval)			3 (0.1 to 4)	-46 (-53 to 40)	-11 (-16 to 5)	5 (2 to 7)	-61 (-69 to -57)	-12 (-19 to -5)

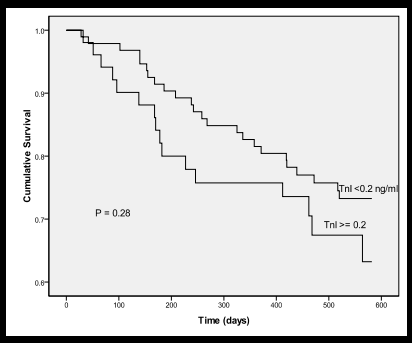
40

BIOMARQUEURS : TROPONINE

Predictive Value of Cardiac Troponin T and I in Hemodialysis Patients

Faiza Rawas Kalaji, Sami Albitar



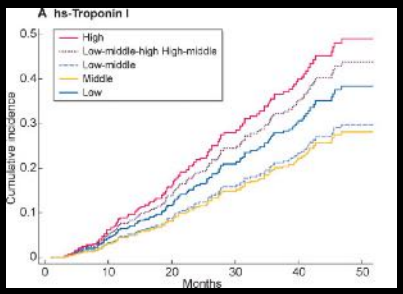


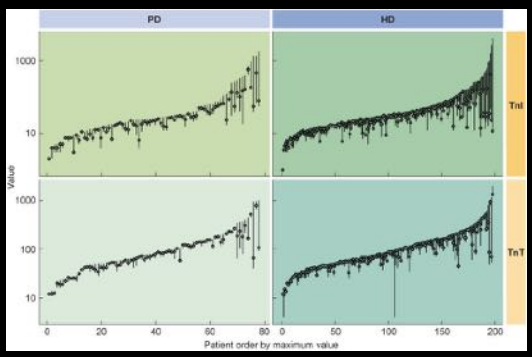
41

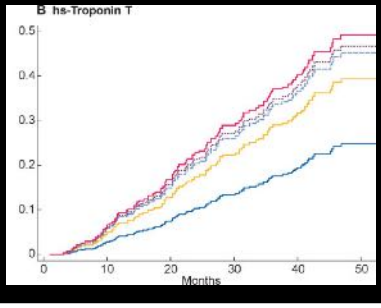
BIOMARQUEURS : TROPONINE

High-sensitivity troponins in dialysis patients: variation and prognostic value

Sunna Snaedal[1,2], Peter Bárány[1], Sigrún H. Lund[3], Abdul R. Qureshi[4], Olof Heimbürger[1], Peter Stenvinkel[1], Christian Löwbeer[5,6] and Karolina Szummer[7,8]





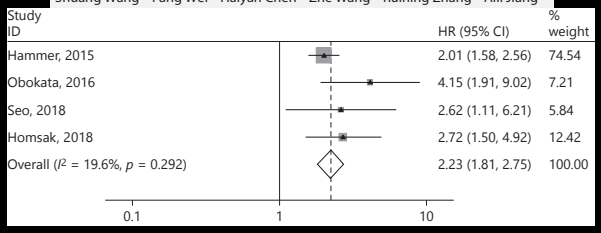


42

BIOMARQUEURS : AUTRES

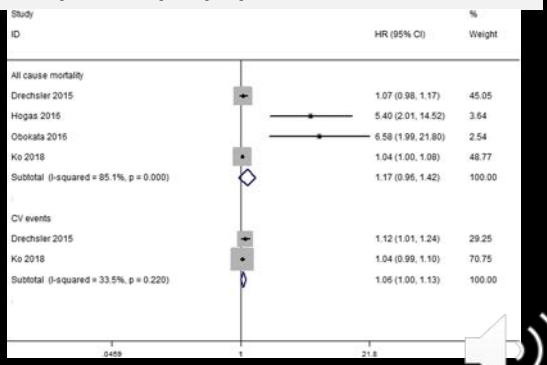
The Prognostic Value of Soluble ST2 in Maintenance Hemodialysis Patients: A Meta-Analysis

Shuang Wang Fang Wei Haiyan Chen Zhe Wang Ruining Zhang Aili Jiang



Prognostic impact of galectin-3 in chronic kidney disease patients: a systematic review and meta-analysis

Tao Zhang[1] · Shili Cao[1] · Hongtao Yang[1] · Jing Li[2]



43

APPROCHES THÉRAPEUTIQUES

•Traitement Médicamenteux : Utilisation d'agents antihypertenseurs, de diurétiques, et d'autres médicaments pour gérer les symptômes.

•Dialyse : Rôle crucial de la dialyse dans la gestion des toxines urémiques et du déséquilibre hydrique.

•Transplantation Rénale : La transplantation rénale comme intervention ultime pour améliorer la fonction rénale et les résultats cardiaques.

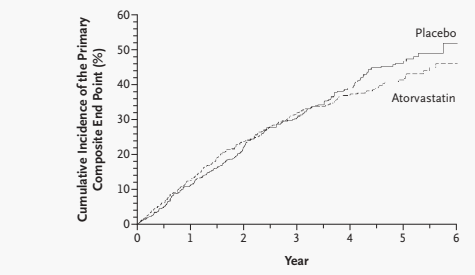
44

PHARMACOLOGIQUE

ORIGINAL ARTICLE

Atorvastatin in Patients with Type 2 Diabetes Mellitus Undergoing Hemodialysis

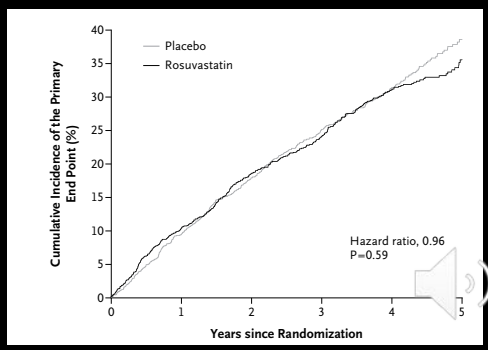
Christoph Wanner, M.D., Vera Krane, M.D., Winfried März, M.D., Manfred Olschewski, M.Sc., Johannes F.E. Mann, M.D., Günther Ruf, M.D., and Eberhard Ritz, M.D., for the German Diabetes and Dialysis Study Investigators*



ORIGINAL ARTICLE

Rosuvastatin and Cardiovascular Events in Patients Undergoing Hemodialysis

Bengt C. Fellström, M.D., Ph.D., Alan G. Jardine, M.D., Roland E. Schmieder, M.D., Hallvard Holdaas, M.D., Ph.D., Kym Bannister, M.D., Jaap Beutler, M.D., Ph.D., Dong-Wan Chae, M.D., Ph.D., Alejandro Chevaile, M.D., Stuart M. Cobbe, M.D., Carola Grönhagen-Riska, M.D., Ph.D., José J. De Lima, M.D., Ph.D., Robert Lins, M.D., Ph.D., Gert Mayer, M.D., Alan W. McMahon, M.D., Hans-Henrik Parving, M.D., D.M.Sc., Giuseppe Remuzzi, M.D., Ola Samuelsson, M.D., Ph.D., Sandor Sonkodi, M.D., Ph.D., D. Sci., Gultekin Süleymanlar, M.D., Dimitrios Tsakiris, M.D., Ph.D., Vladimir Tesar, M.D., Ph.D., Vasil Todorov, M.D., Ph.D., Andrzej Wiecek, M.D., Ph.D., Rudolf P. Wüthrich, M.D., Mattis Gottlow, M.Sc., Eva Johnsson, M.D., Ph.D., and Faiez Zannad, M.D., Ph.D., for the AURORA Study Group*

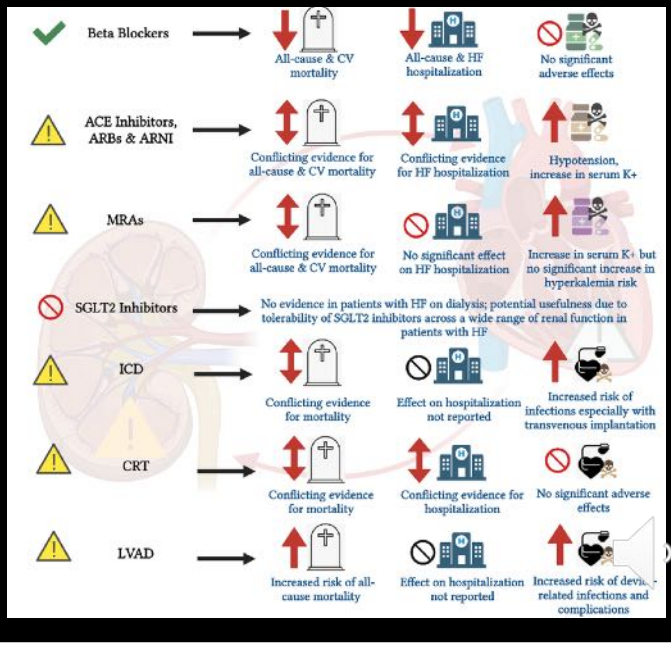


45

PHARMACOLOGIQUE

Managing Heart Failure in Patients on Dialysis: State-of-the-Art Review

MUHAMMAD SHAHZEB KHAN, MD, MSC,[1] AYMEN AHMED, MBBS,[2] STEPHEN J. GREENE, MD,[1,2] MONA FIUZAT, PharmD,[1] MICHELLE M. KITTLESON, MD, PhD,[4] JAVED BUTLER, MD, MPH, MBA,[5,6] GEORGE L. BAKRIS, MD,[7] AND GREGG C. FONAROW, MD[8]

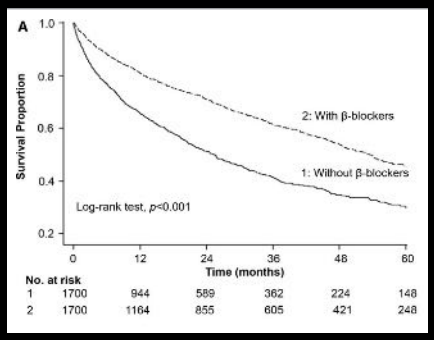


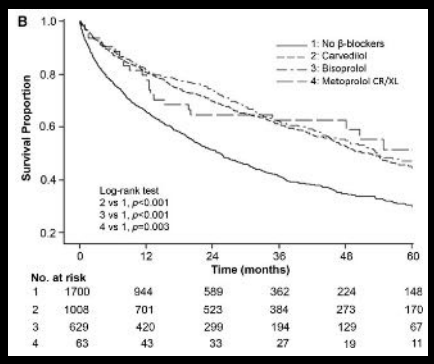
46

PHARMACOLOGIQUE

Prognostic Benefits of Carvedilol, Bisoprolol, and Metoprolol Controlled Release/Extended Release in Hemodialysis Patients with Heart Failure: A 10-Year Cohort

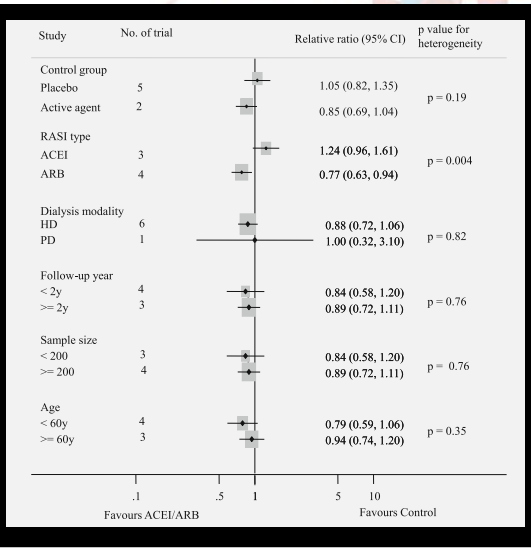
Chao-Hsiun Tang, PhD; Chia-Chen Wang, MD; Tso-Hsiao Chen, MD, PhD; Chuang-Ye Hong, MD, PhD; Yuh-Mou Sue, MD





47

PHARMACOLOGIQUE

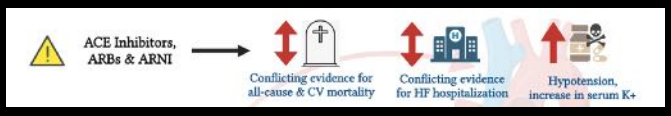


Effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers on cardiovascular events and residual renal function in dialysis patients: a meta-analysis of randomised controlled trials

Youxia Liu[1†], Xinxin Ma[2†], Jie Zheng[3], Junya Jia[1] and Tiekun Yan[1*]

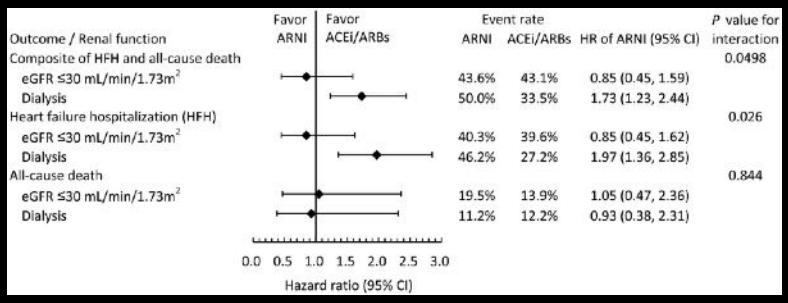
48

PHARMACOLOGIQUE



Angiotensin Receptor-Neprilysin Inhibitors in Patients With Heart Failure With Reduced Ejection Fraction and Advanced Chronic Kidney Disease: A Retrospective Multi-Institutional Study

*Fu-Chih Hsiao [1], Chia-Pin Lin [1], Chun-Chen Yu [2], Ying-Chang Tung [1] and Pao-Hsien Chu [1]**



49

PHARMACOLOGIQUE

Rossignol P, Zannad F Massy ZA, et al. ALdosterone Antagonist Chronic HEModialysis Interventional Survival Trial (ALCHEMIST): Primary Results. Paper Presented at: American Society of Nephrology Kidney Week 2023. November 1-5, 2023

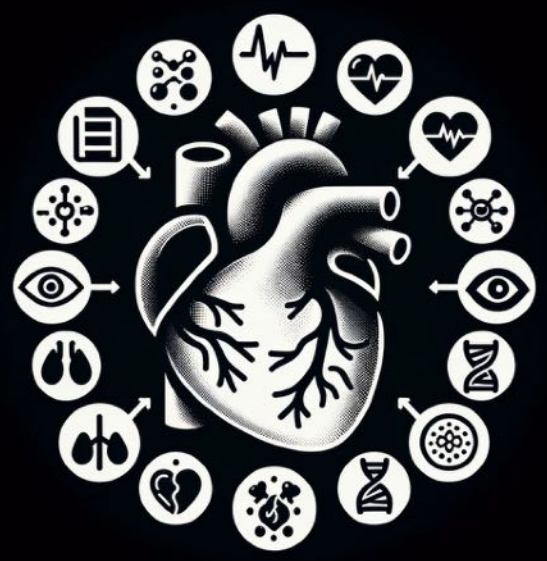
Etude ALCHEMIST 644 patients
Randomisation SPIRONOLACTONE vs PLACEBO
Primary composite endpoint of ALCHEMIST

- nonfatal myocardial infarction
- acute coronary syndrome
- hospitalization for heart failure
- nonfatal stroke
- cardiovascular mortality

hazard ratio [HR], 0.996; confidence interval [CI], 0.729-1.362; P=.9819).
incidence of hospitalization for heart failure HR, 0.412; CI, 0.171-0.995; P=.0487

50

PHARMACOLOGIQUE



iSGLT2 et Finérénone ?

Inhibiteurs sST2 ? Gal 3 ?

51

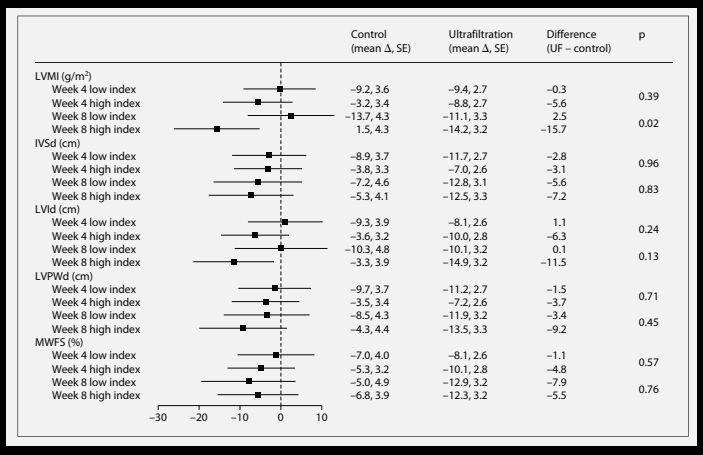
GESTION DU POIDS SEC

Dry-Weight Reduction in Hypertensive Hemodialysis Patients (DRIP)
A Randomized, Controlled Trial

Rajiv Agarwal, Pooneh Alborzi, Sangeetha Satyan, Robert P. Light

Probing Dry-Weight Improves Left Ventricular Mass Index

Rajiv Agarwal[a, b] J. Michael Bouldin[a] Robert P. Light[a] Ashok Garg[c]



	Control (mean Δ, SE)	Ultrafiltration (mean Δ, SE)	Difference (UF – control)	p
LVMI (g/m²)				
Week 4 low index	−9.2, 3.6	−9.4, 2.7	−0.3	0.39
Week 4 high index	−3.2, 3.4	−8.8, 2.7	−5.6	
Week 8 low index	−13.7, 4.3	−11.1, 3.3	2.5	0.02
Week 8 high index	1.5, 4.3	−14.2, 3.2	−15.7	
IVSd (cm)				
Week 4 low index	−8.9, 3.7	−11.7, 2.7	−2.8	0.96
Week 4 high index	−3.8, 3.3	−7.0, 2.6	−3.1	
Week 8 low index	−7.2, 4.6	−12.8, 3.1	−5.6	0.83
Week 8 high index	−5.3, 4.1	−12.5, 3.3	−7.2	
LVId (cm)				
Week 4 low index	−9.3, 3.9	−8.1, 2.6	1.1	0.24
Week 4 high index	−3.6, 3.2	−10.0, 2.8	−6.3	
Week 8 low index	−10.3, 4.8	−10.1, 3.2	0.1	0.13
Week 8 high index	−3.3, 3.9	−14.9, 3.2	−11.5	
LVPWd (cm)				
Week 4 low index	−9.7, 3.7	−11.2, 2.7	−1.5	0.71
Week 4 high index	−3.5, 3.4	−7.2, 2.6	−3.7	
Week 8 low index	−8.5, 4.3	−11.9, 3.2	−3.4	0.45
Week 8 high index	−4.3, 4.4	−13.5, 3.3	−9.2	
MWFS (%)				
Week 4 low index	−7.0, 4.0	−8.1, 2.6	−1.1	0.57
Week 4 high index	−5.3, 3.2	−10.1, 2.8	−4.8	
Week 8 low index	−5.0, 4.9	−12.9, 3.2	−7.9	0.76
Week 8 high index	−6.8, 3.9	−12.3, 3.2	−5.5	

−30 −20 −10 0 10

52

MODALITÉ DE DIALYSE

Short Daily Hemodialysis: Blood Pressure Control and Left Ventricular Mass Reduction in Hypertensive Hemodialysis Patients

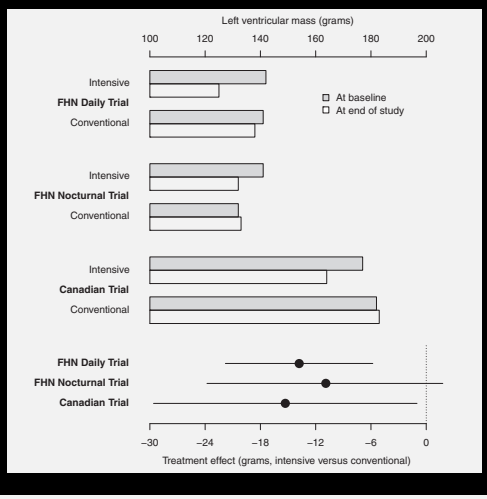
Riccardo Maria Fagugli, MD, Gianpaolo Reboldi, MD, PhD, Giuseppe Quintaliani, MD, Paolo Pasini, MD, Giovanni Ciao, MD, Beatrice Cicconi, MD, Franca Pasticci, RD, Jean Marie Kaufman, MD, PhD, and Umberto Buoncristiani, MD

Table 4. Echocardiographic Parameters During SHD and DHD

	SHD	DHD	*P*
IVS (mm)	13.7 ± 3.4	11.9 ± 2.3	0.001
PWT (mm)	8.9 ± 2.5	9.9 ± 3.5	NS
LVIDD (mm)	52.3 ± 7.8	46.8 ± 8.3	<0.01
LVM (g)	239 ± 91.7	192 ± 90.4	0.01
LVMI (g/m^2)	148 ± 59.7	120 ± 60.4	0.01

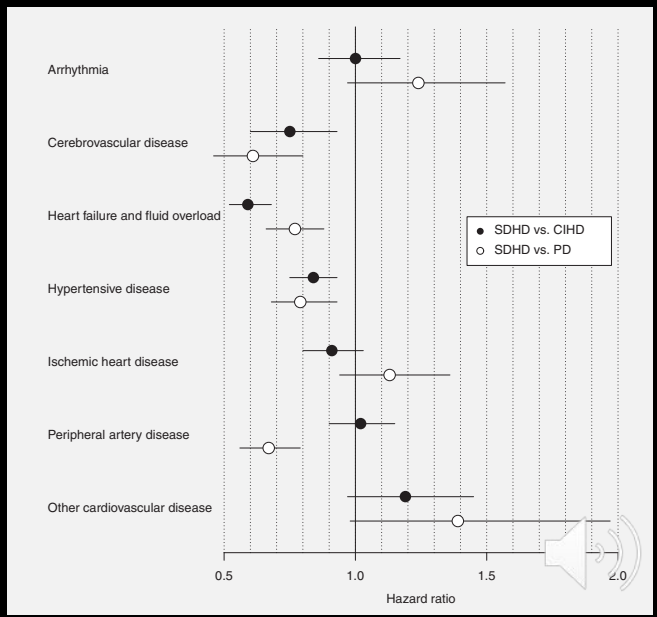
53

MODALITÉ DE DIALYSE



Intensive Hemodialysis, Left Ventricular Hypertrophy, and Cardiovascular Disease

Peter A. McCullough, MD, MPH,[1,2,3,4] Christopher T. Chan, MD,[5] Eric D. Weinhandl, PhD, MS,[6] John M. Burkart, MD,[7] and George L. Bakris, MD[8]

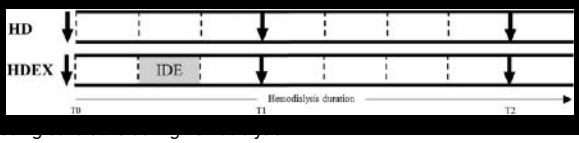


54

EXERCICE PER-DIALYTIQUE

Cardioprotective Effect of Acute Intradialytic Exercise: A Comprehensive Speckle-Tracking Echocardiography Analysis

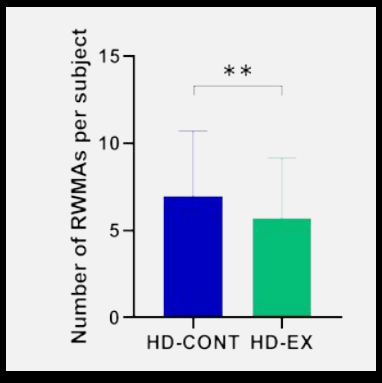
Matthieu Josse,[1] Laure Patrier,[2,3] Myriam Isnard,[4] Cécile Turc-Baron,[2,5] Antoine Grandperrin,[1] Stéphane Nottin,[1] Stéphane Mandigout,[6] Jean-Paul Cristol,[2,5,7] Claire Maufrais,[1] and Philippe Obert[1]

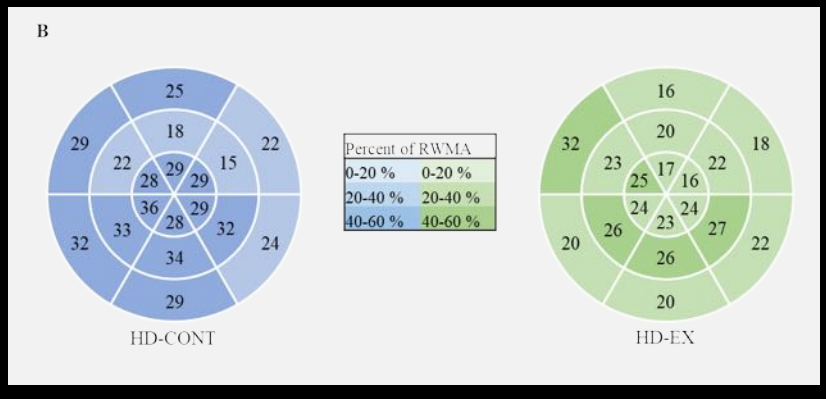


Variable	HD			HDEX			Linear Mixed-Effects Model			
							Condition×Sequence	Condition×Time		
	T0	T1	T2	T0	T1	T2	*P* Value	Estimated Difference[a]	95% CI	*P* Value
Primary outcome										
GLS (%)	−18.2±3.1	−16.2±3.3	−15.2±3.7	−18.1±3.0	−16.8±3.5	−16.3±3.4	0.94	−1.16	−0.31 to −2.02	0.009
P value[b]		<0.001	<0.001		<0.001	0.007				
		<0.001			<0.001					

55

EXERCICE PER-DIALYTIQUE





Josse et al, in press

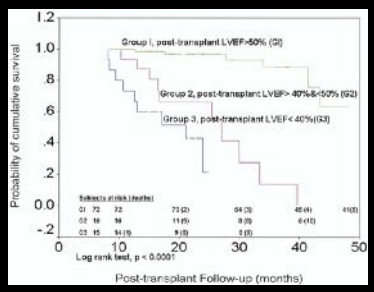
56

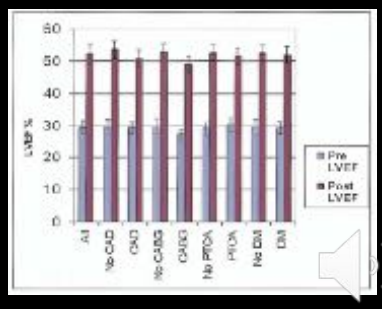
TRANSPLANTATION

Effect of Kidney Transplantation on Left Ventricular Systolic Dysfunction and Congestive Heart Failure in Patients With End-Stage Renal Disease

Ravinder K. Wali, MD,* Gregory S. Wang, MD,† Stephen S. Gottlieb, MD,‡ Lavanya Bellumkonda, MD,* Riple Hansalia, MD,† Emilio Ramos, MD,* Cinthia Drachenberg, MD,‖ John Papadimitriou, MD,‖ Meredith A. Brisco, MD,† Steve Blahut, PHD,* Jeffrey C. Fink, MD,* Michael L. Fisher, MD,‡ Stephen T. Bartlett, MD,§ Matthew R. Weir, MD*

	All Patients (N = 103)	Groups Based on Post-Transplant LVEF% LVEF ≥50% Group 1 (n = 72)	LVEF ≥40% to <50% Group 2 (n = 16)	LVEF <40% Group 3 (n = 15)	p Value Group 1 vs. 2	Group 2 vs. 3	Group 3 vs. 1
Pretransplant evaluation							
LVEF% (initial transplant evaluation)							
Mean ± SD	31.6 ± 6.7	31.7 ± 6.7	31.6 ± 7.6	31.2 ± 6.1	1.00	0.98	0.98
(95% CI)	(30.3–32.9)	(30.1–33.3)	(27.5–35.6)	(27.7–34.6)			
LVEF% (repeat evaluation before transplant surgery)*							
Mean ± SD	29.3 ± 6.2	29.0 ± 6.0	30.5 ± 8.0	29.6 ± 5.3	0.87	0.97	0.98
(95% CI)	(28.1–30.6)	(27.6–30.5)	(26.2–34.8)	(26.6–32.5)			
Post-transplant evaluation							
Post-transplant LVEF% (at six months)							
Mean ± SD	47.2 ± 10.7	52.5 ± 6.9	39.2 ± 6.1	30.6 ± 6.5	<0.0001	0.002	<0.0001
(95% CI)	(45.1–49.3)	(50.8–54.1)	(35.9–42.5)	(26.9–34.2)			
Post-transplant LVEF% (at 12 months)†							
Mean ± SD	52.2 ± 12.0	58.8 ± 6.8	42.1 ± 2.4	31.6 ± 4.9	<0.001	0.001	<0.001
(95% CI)	(49.9–54.6)	(57.2–60.4)	(40.8–43.4)	(28.9–34.4)			



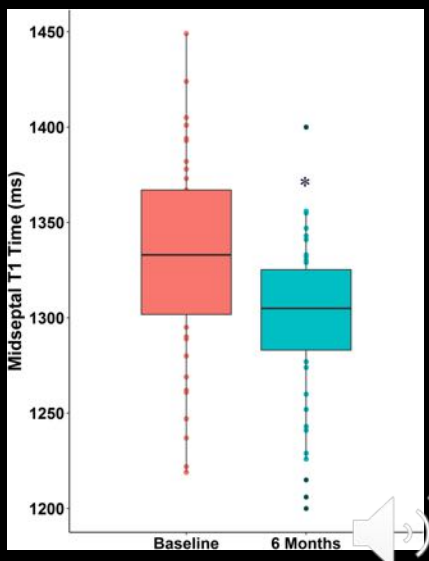


57

TRANSPLANTATION

Kidney transplantation is associated with reduced myocardial fibrosis. A cardiovascular magnetic resonance study with native T1 mapping

	Baseline (n = 44)	6 months (n = 44)	p
Ejection Fraction (%)	64 ± 12	67 ± 10	0.07[a]
EDV (ml)	155 ± 43	152 ± 35	0.61[a]
ESV (ml)	52 [38–69]	50 [39–63]	0.49[b]
SV (ml)	96 ± 21	100 ± 20	0.27[a]
LVM (g)	154 ± 40	152 ± 35	0.76[a]
EDVi (mL/m^2)	88 ± 23	85 ± 19	0.29[a]
ESVi (mL/m^2)	30 [21–40]	28 [22–36]	0.28[b]
SVi (mL/m^2)	55 ± 12	56 ± 11	0.42[a]
LVMi (g/m^2)	87 ± 20	85 ± 16	0.36[a]
IS (mm)	12.9 ± 3.3	12.7 ± 3.2	0.71[a]
LVPW (mm)	10.2 ± 2.2	9.6 ± 1.8	0.07[a]
LVED (mm)	53 ± 7	52 ± 7	0.21[a]
LVES (mm)	34 ± 9	33 ± 6	0.20[a]



58

LIMITES THÉRAPEUTIQUES

•Efficacité Limitée : Les traitements actuels ne parviennent pas à résoudre complètement les complications cardiaques liées à la maladie rénale.

•Besoin de Recherche : La nécessité d'une recherche continue pour développer de meilleures stratégies thérapeutiques.

•Gestion Globale : L'importance d'une approche globale incluant la gestion des facteurs de risque de la maladie rénale et cardiaque.

59

IMPLICATIONS POUR LE CLINICIEN

•Approche Individualisée : Nécessité d'une approche de traitement individualisée pour chaque patient.

•Surveillance Continue : Importance de la surveillance régulière pour détecter et gérer les complications précocement.

•Éducation du Patient : Rôle crucial de l'éducation du patient sur la gestion de la maladie rénale et la prévention des complications cardiaques.

60

ET POUR DEMAIN ?

•Thérapies Innovantes : Exploration de nouvelles stratégies thérapeutiques et médicaments.

•Compréhension Approfondie : Besoin de recherches approfondies sur la pathophysiologie de la maladie.

•Intégration des Technologies : Utilisation de technologies avancées pour le diagnostic et le suivi.

61

62