Nephrology and Transplantation Department
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Hemodialysis & Heart failure; an unmet medical need

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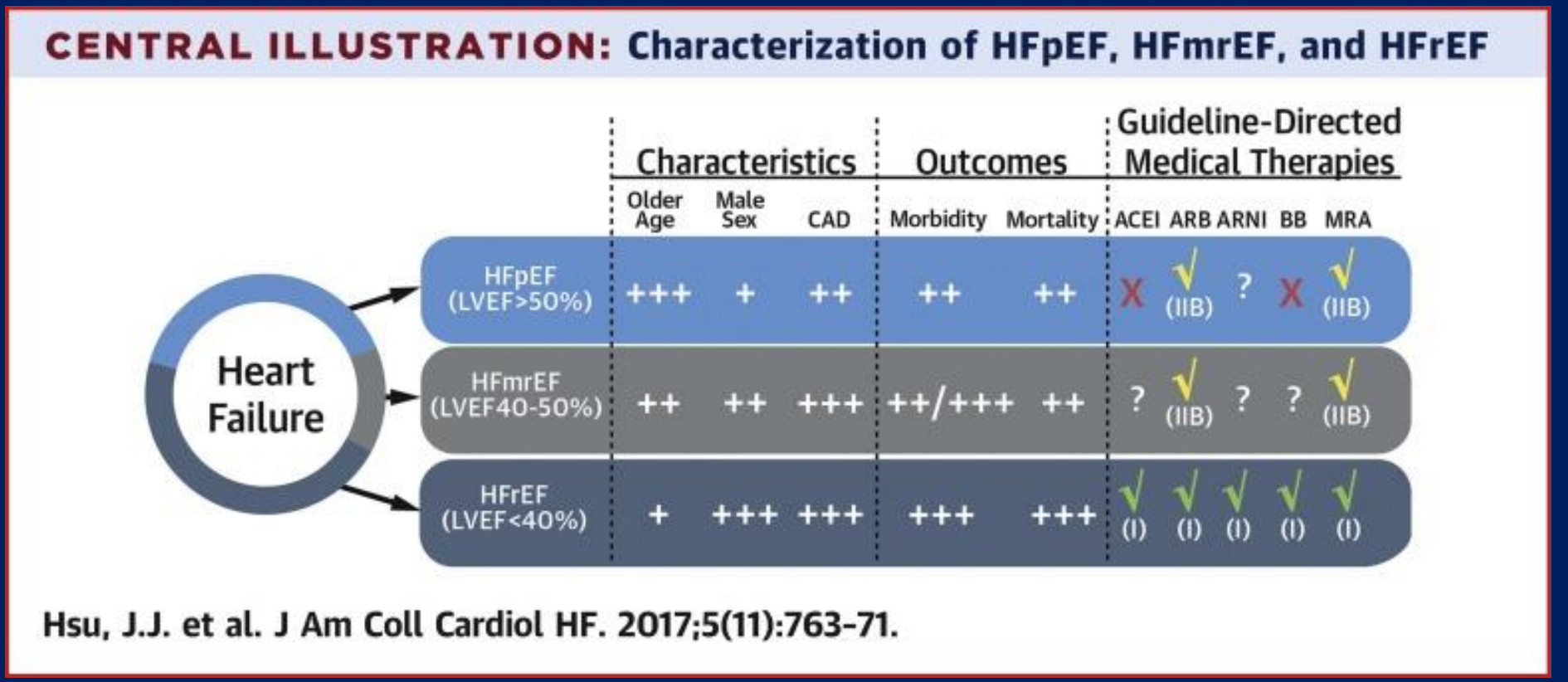
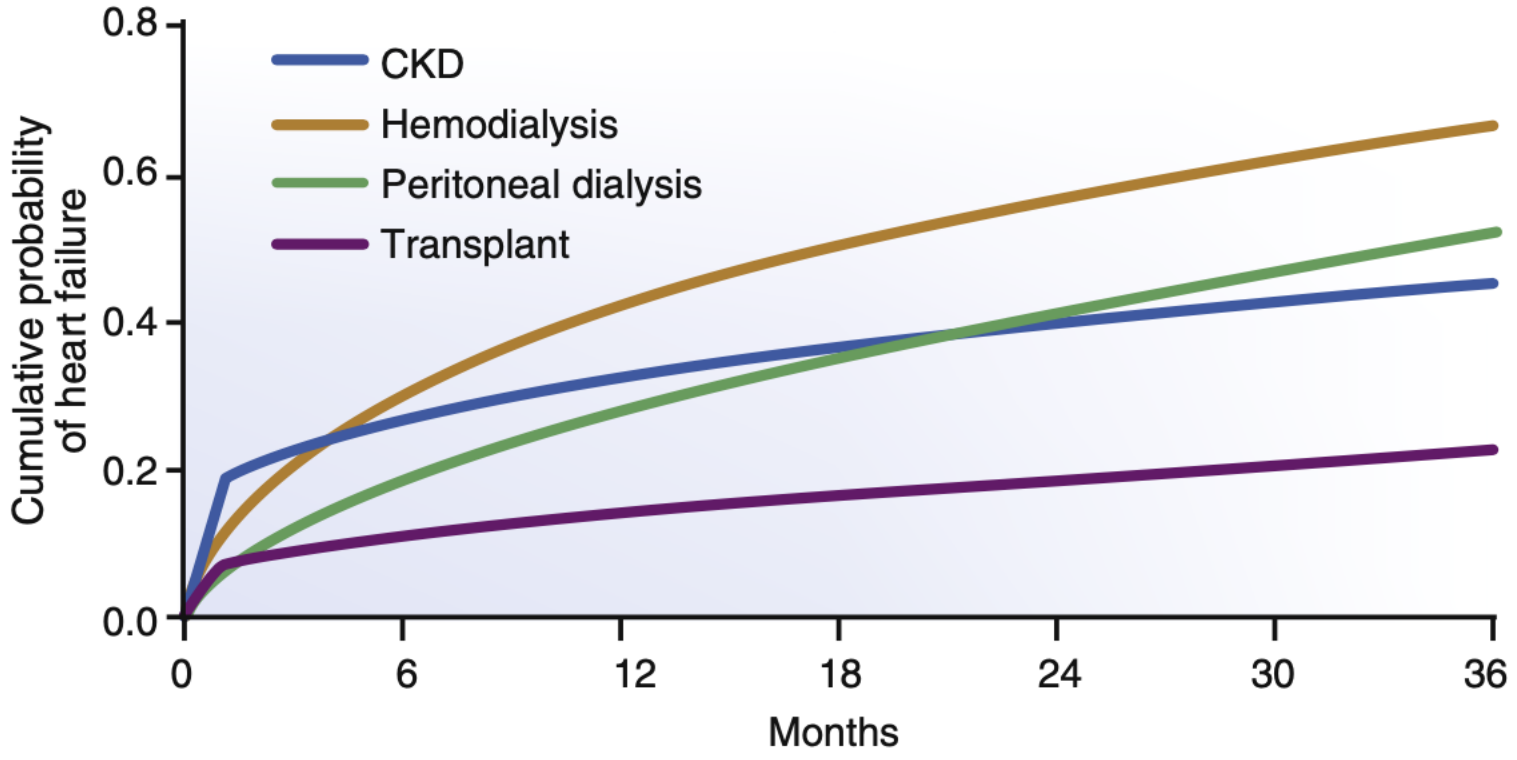
Outlines

- Definition
- Diagnosis
- Treatment:
  - Medical treatments
  - Dialysis

Definition


CENTRAL ILLUSTRATION: Characterization of HFpEF, HFmrEF, and HFrEF
Characteristics
Outcomes
Guideline-Directed Medical Therapies
Older Age
Male Sex
CAD
Morbidity
Mortality
ACEI
ARB
ARNI
BB
MRA
Heart Failure
HFpEF (LVEF>50%)
+++ + ++ ++ ++
X √ (IIB) ? X √ (IIB)
HFmrEF (LVEF40-50%)
++ ++ +++ ++/+++ ++
? √ (IIB) ? ? √ (IIB)
HFrEF (LVEF<40%)
+ +++ +++ +++ +++
√ (I) √ (I) √ (I) √ (I) √ (I)
Hsu, J.J. et al. J Am Coll Cardiol HF. 2017;5(11):763-71.



CKD: Incident general Medicare CKD patients, age 66 & older, 2001–2003 combined
ESKD: Incident ESKD patients, age 20 & older
Patients with CHF at baseline excluded. Probabilities unadjusted

Kidney International (2019) **95,** 1304–1317

Diagnosis

- KDOQI guidelines recommend:

- Echocardiograms to be performed 1–3 months after the start of dialysis and every 3 years thereafter, regardless of symptoms.

Factors contribute to HF in the dialysis patient include:

- Fluid overload,
- Poorly-controlled HTN increasing afterload,
- LV diastolic dysfunction (associated with LVH),
- Arterial stiffness,
- LV systolic dysfunction,
- Uremic toxin accumulation,
- Anemia,
- Valvular heart disease.
- A high-output state due to AVF

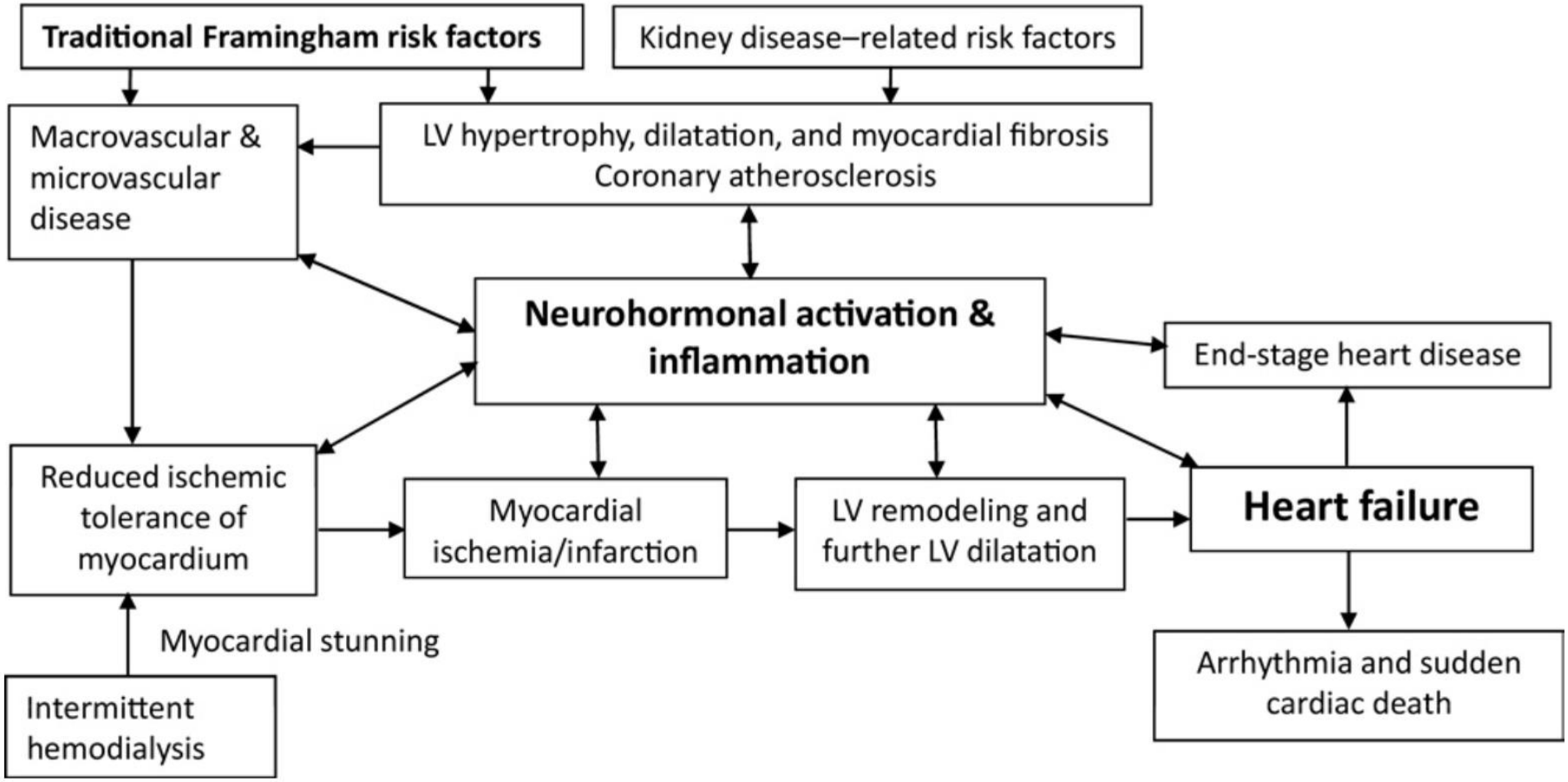
Pathogenesis of heart failure in long-term dialysis patients

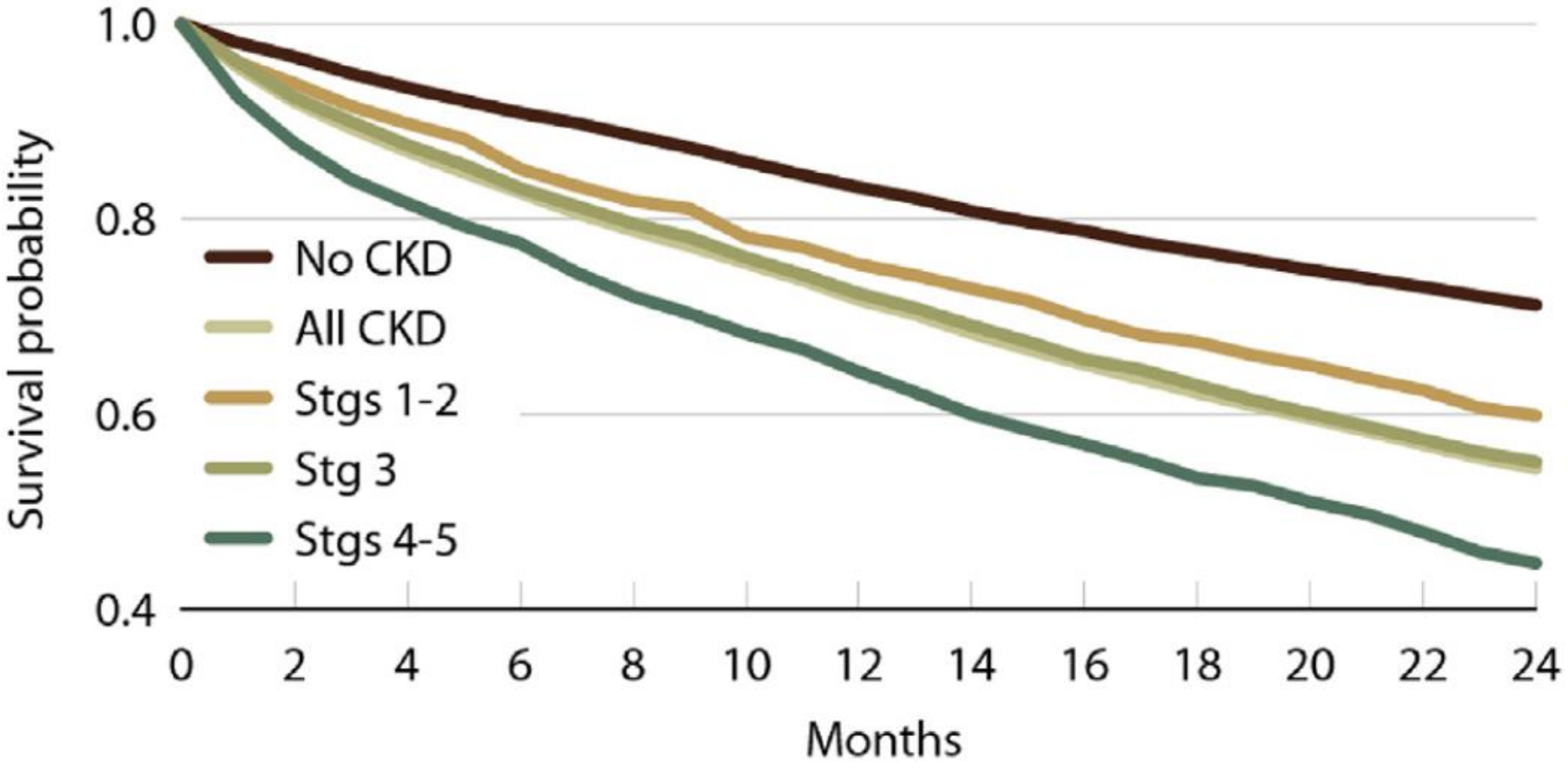
Traditional Framingham Risk Factors

- Hypertension
- Smoking
- Diabetes
- Aging
- Family history
- Dyslipidemia

Kidney Disease–Related Risk Factors

- Salt and volume overload
- Asymmetric dimethylarginine
- Sympathetic overactivity
- Arterial stiffening
- Oxidative stress
- Inflammation
- 25-Hydroxyvitamin D deficiency
- Hypoalbuminemia
- Uremic toxins
- Hyperphosphatemia
- Hyperparathyroidism
- Vascular/valvular calcification
- Anemia
- Insulin resistance


Traditional Framingham risk factors
Kidney disease–related risk factors
Macrovascular & microvascular disease
LV hypertrophy, dilatation, and myocardial fibrosis
Coronary atherosclerosis
Neurohormonal activation & inflammation
End-stage heart disease
Reduced ischemic tolerance of myocardium
Myocardial ischemia/infarction
LV remodeling and further LV dilatation
Heart failure
Myocardial stunning
Intermittent hemodialysis
Arrhythmia and sudden cardiac death


1.0
0.8
0.6
0.4
Survival probability
No CKD
All CKD
Stgs 1-2
Stg 3
Stgs 4-5
0
2
4
6
8
10
12
14
16
18
20
22
24
Months

Treatment

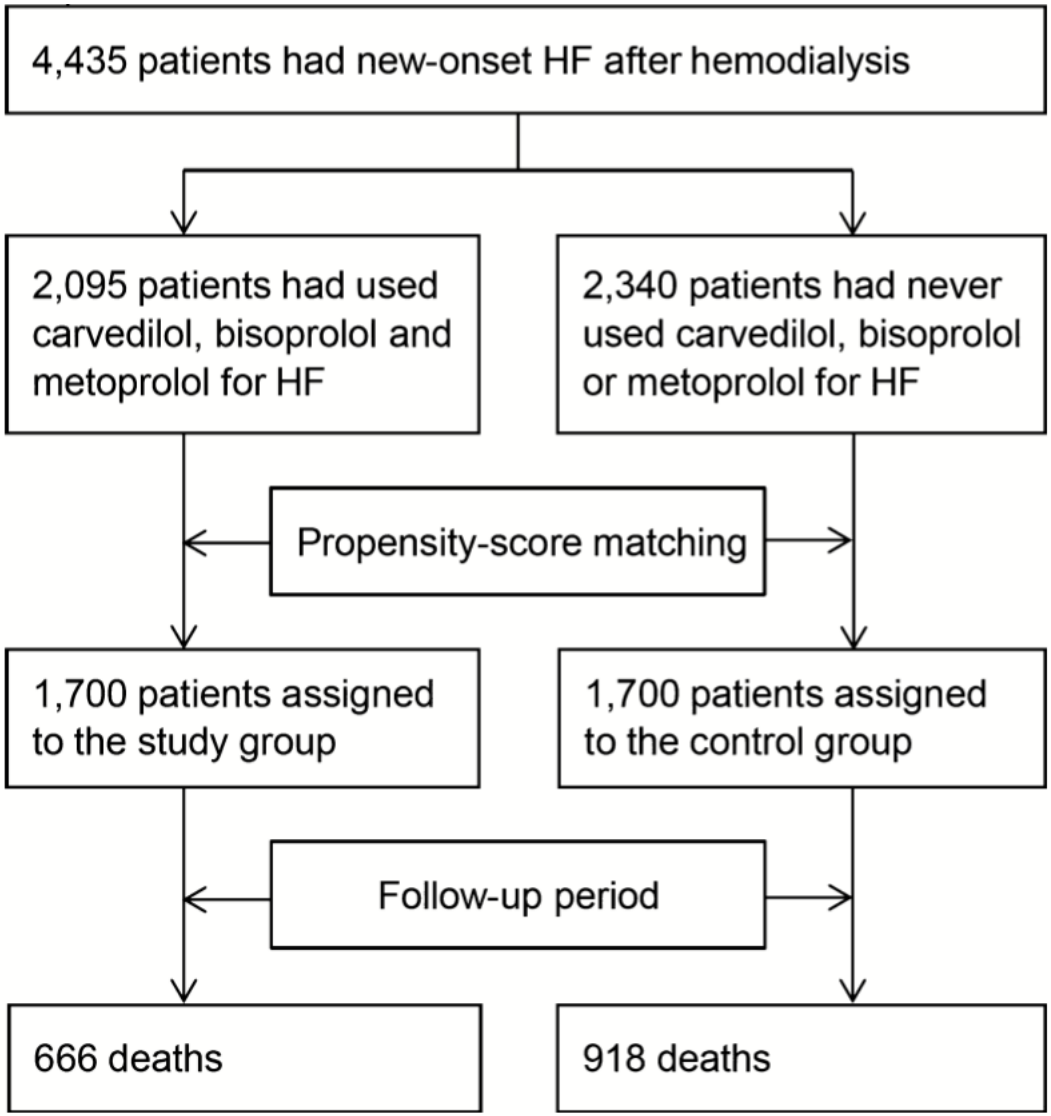
- HD patients have:
  - a more activated sympathetic nervous system,
  - a higher prevalence of HF and ischemic heart disease, a
  - a higher risk of sudden cardiac death.

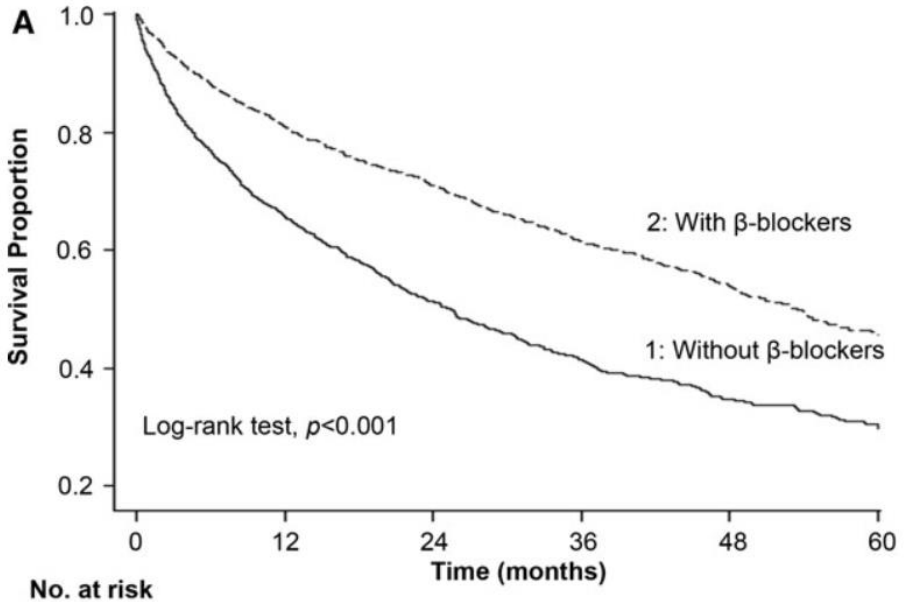
Medical Treatment:

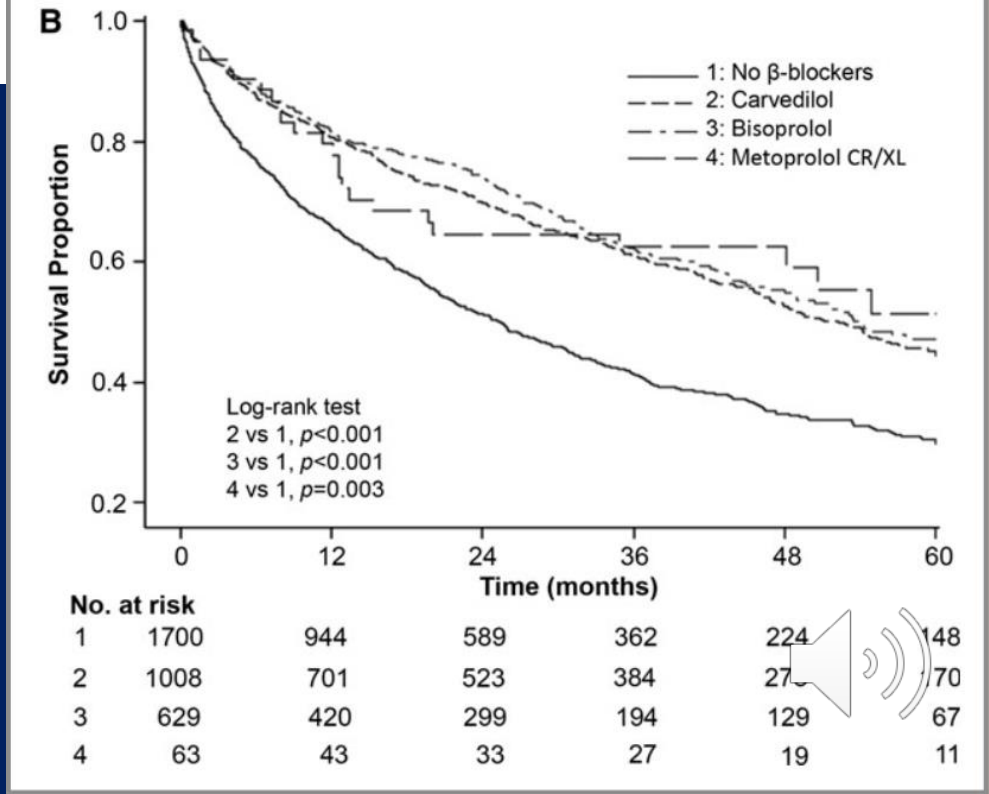
- HFrEF (LVEF ≤40%) & HFmrEF (40-50%):
  - A combination of β-blocker and ACEi
  - For dialysis patients with HFrEF who have persistent symptoms of HF despite treatment with optimally titrated beta blocker plus ACEi therapy, the role of additional pharmacologic therapy is uncertain.

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







J Am Heart Assoc. 2016;5:e002584

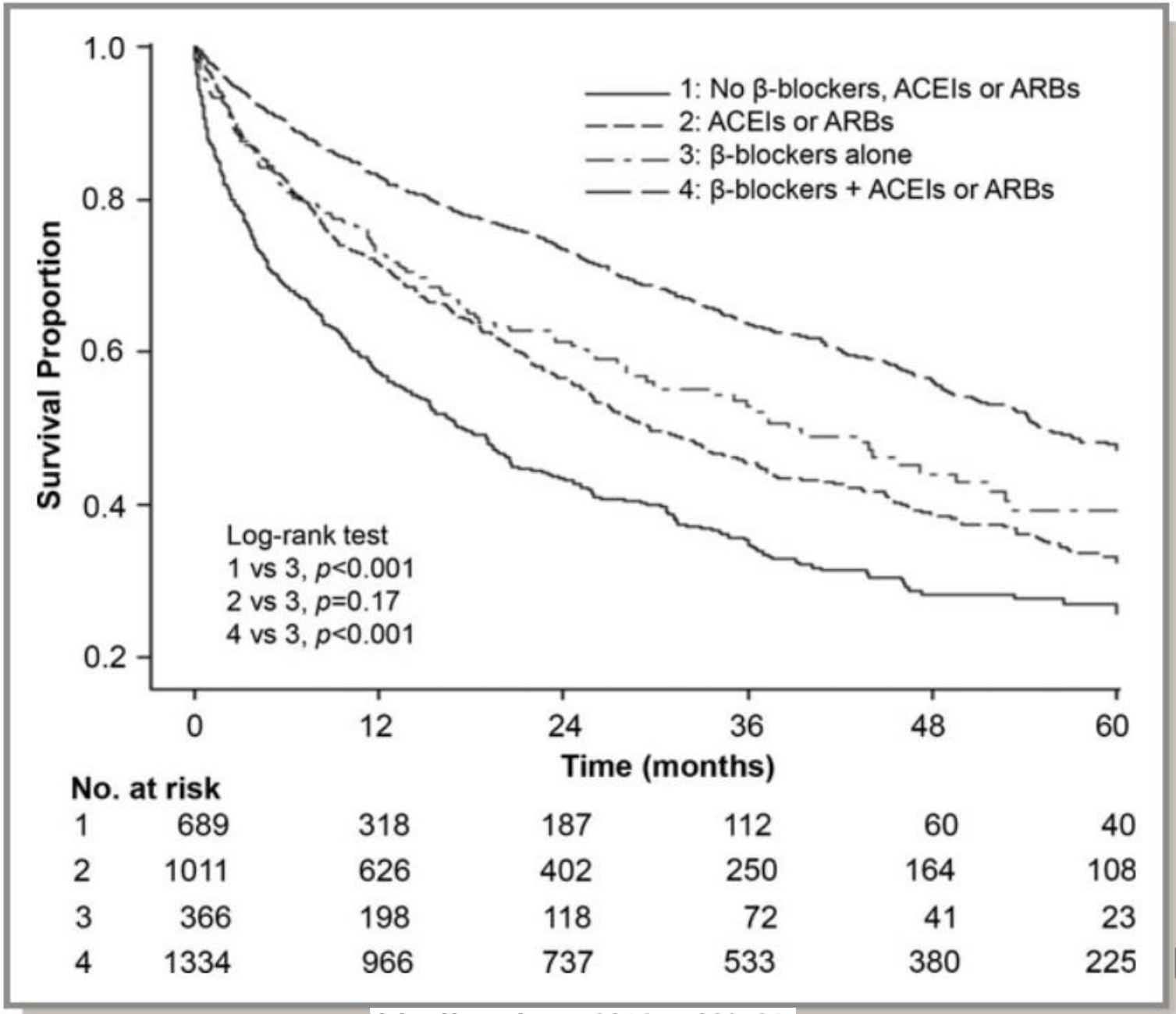
- β-blockers can have survival benefits on HD patients with HF, as demonstrated by the 20% reduction in all-cause mortality.
- A poorly dialyzed β-blocker may provide greater benefit than a highly dialyzable β-blocker.
- Carvedilol > Bisoprolol > Metoprolol XR

- Most common clinically significant arrhythmias in hemodialysis patients are bradyarrhythmias, and are most common in the first dialysis session of the week.

- The initiation and titration of these agents can be challenging in hemodialysis patients, particularly those with low blood pressures.

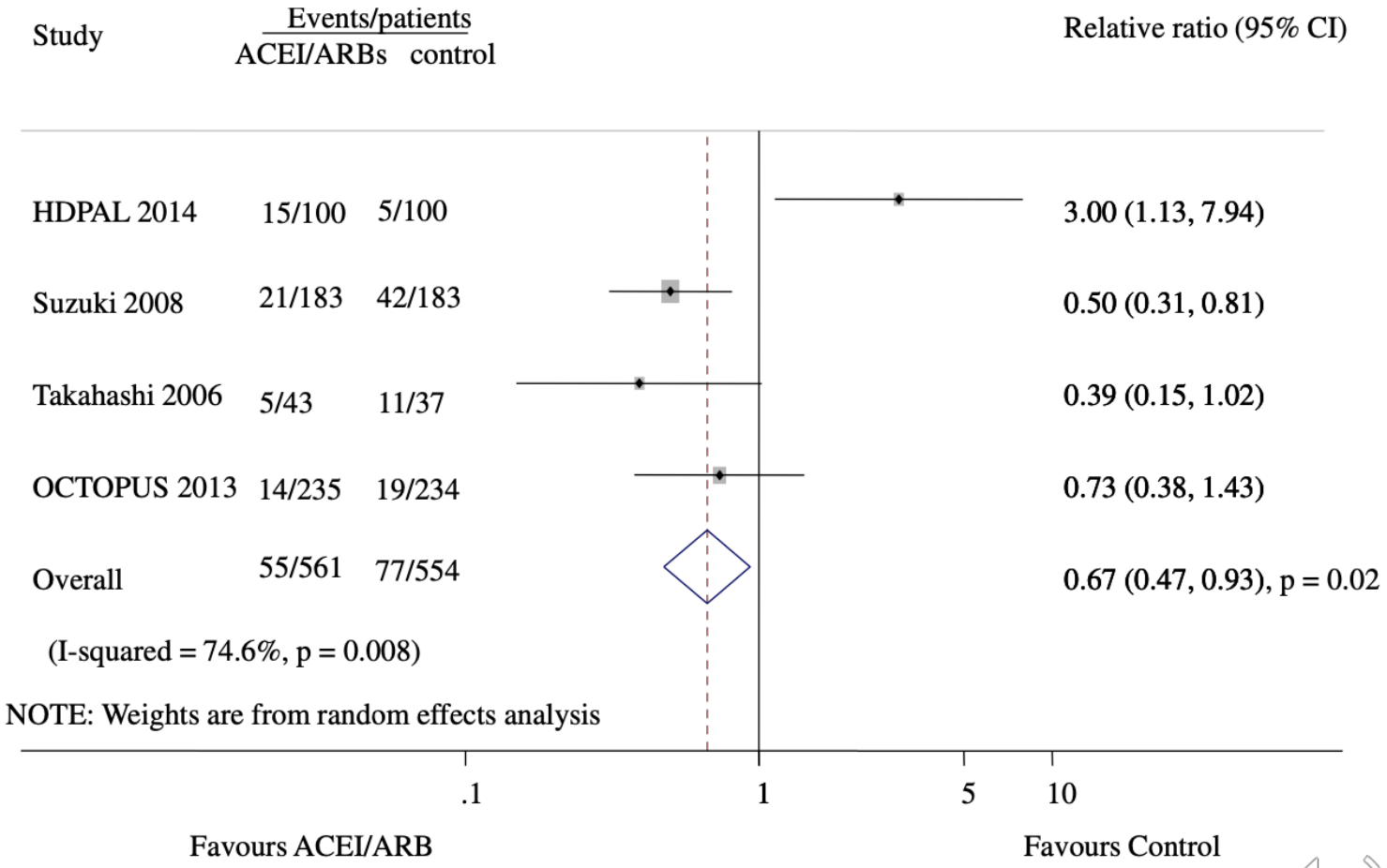
- In general, the higher the predialysis resting blood pressure, the easier it is to uptitrate these agents

- SBP< 100 mmHg: not administering these agents before dialysis on hemodialysis days.
- Low starting dose: Carvedilol 3.125 mg BID;
- Slow up-titration: Dose doubling every 2-4 weeks



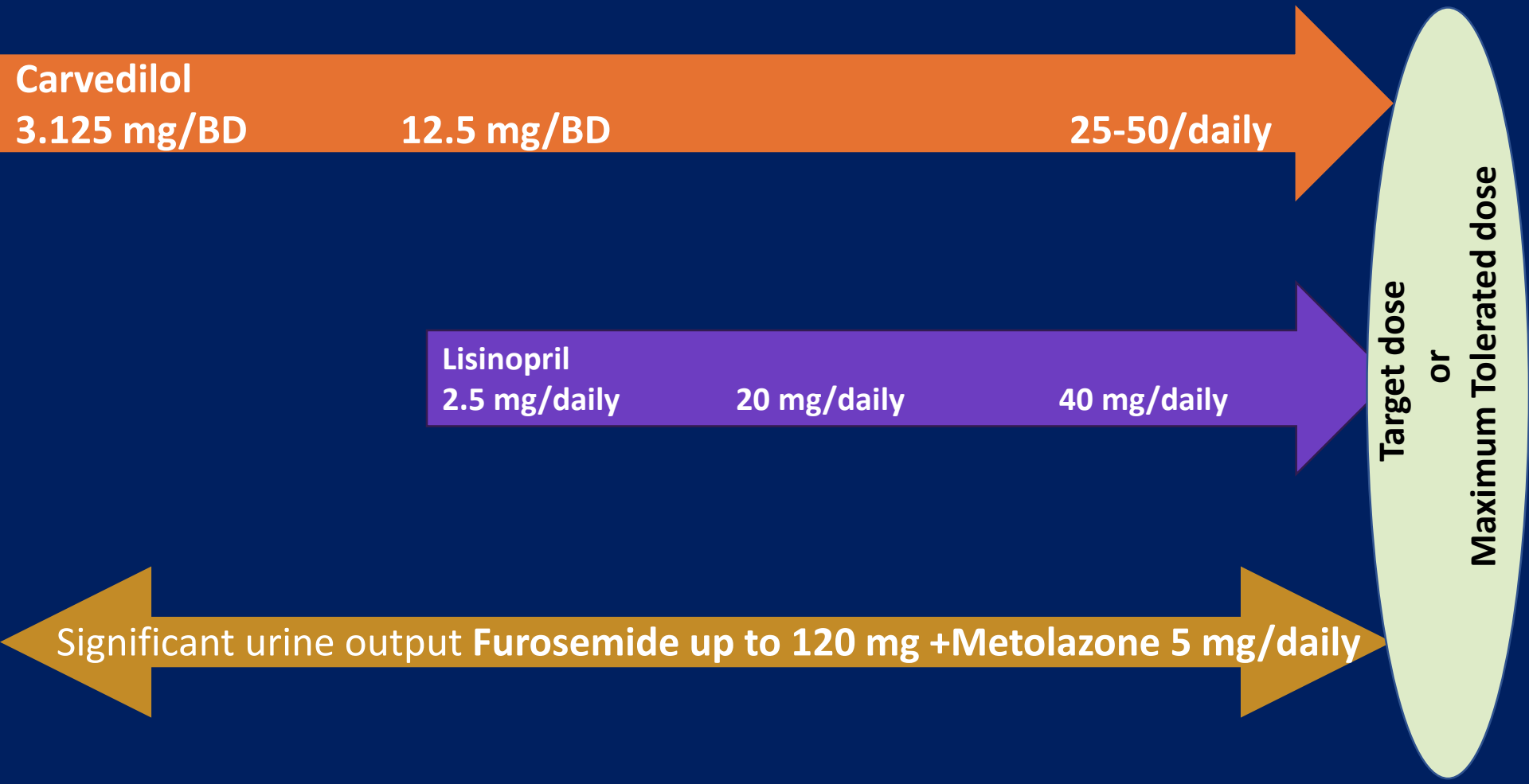
J Am Heart Assoc. 2016;5:e002584

a meta-analysis of randomised controlled trials



Effect of ACE-Is or ARBs compared with placebo or other active agents on heart failure

Liu *et al. BMC Nephrology* (2017) 18:206


Carvedilol
3.125 mg/BD
12.5 mg/BD
25-50/daily
Lisinopril
2.5 mg/daily
20 mg/daily
40 mg/daily
Target dose
or
Maximum Tolerated dose
Significant urine output Furosemide up to 120 mg +Metolazone 5 mg/daily

- In patients with intradialytic hypotension: dialyzable ACE-I (Captopril)
- In patients who experience intradialytic hypertension: an ARB or a nondialyzable ACE-I (Fosinopril)
- Dosing of most ACE-I should be daily;
- Nocturnal dosing of once daily medications.

Class	$T^{1/2}$ in ESRD	Range of dosing (initial to usual or maximum)	%Removal with hemodialysis
Angiotensin converting enzyme inhibitors			
Captopril	20–30 hours	12.5–50 mg q24 hours	Yes
Benazepril	?	5–40 mg q24 hours	20–50%
Enalapril	Prolonged	2.5–10 mg q24 to 48 hours	35%
Fosinopril	Prolonged	10–80 mg q24 hours	<10%
Lisinopril	54 hours	2.5–10 mg q24–48 hours	50%
Ramipril	prolonged	2.5–10 mg q24 hours	<30%
Angiotensin receptor blockers			
Losartan	4 hours	50–100 mg q24	None
Candesartan	5–9 hours	4–32 mg q24	None
Eprosartan	?	400–600 mg q24	None
Telmisartan	24 hours	40–80 mg q24	None
Valsartan	6 hours	80–160 mg q24	None
Irbesartan	11–15 hours	75–300 mg q24	None

Lisinopril has demonstrated good blood pressure control with thrice weekly administration following hemodialysis

MRA

- At this time, safety and positive effect of MRN in patients on dialysis remain unclear.

- The combination of an ACEi, an ARB, and an MRA is generally **avoided** given the risk of hyperkalemia and the lack of evidence of efficacy.

Aldosterone antagonists (spironolactone or eplerenone) versus control (placebo or standard care) for people with chronic kidney disease requiring dialysis

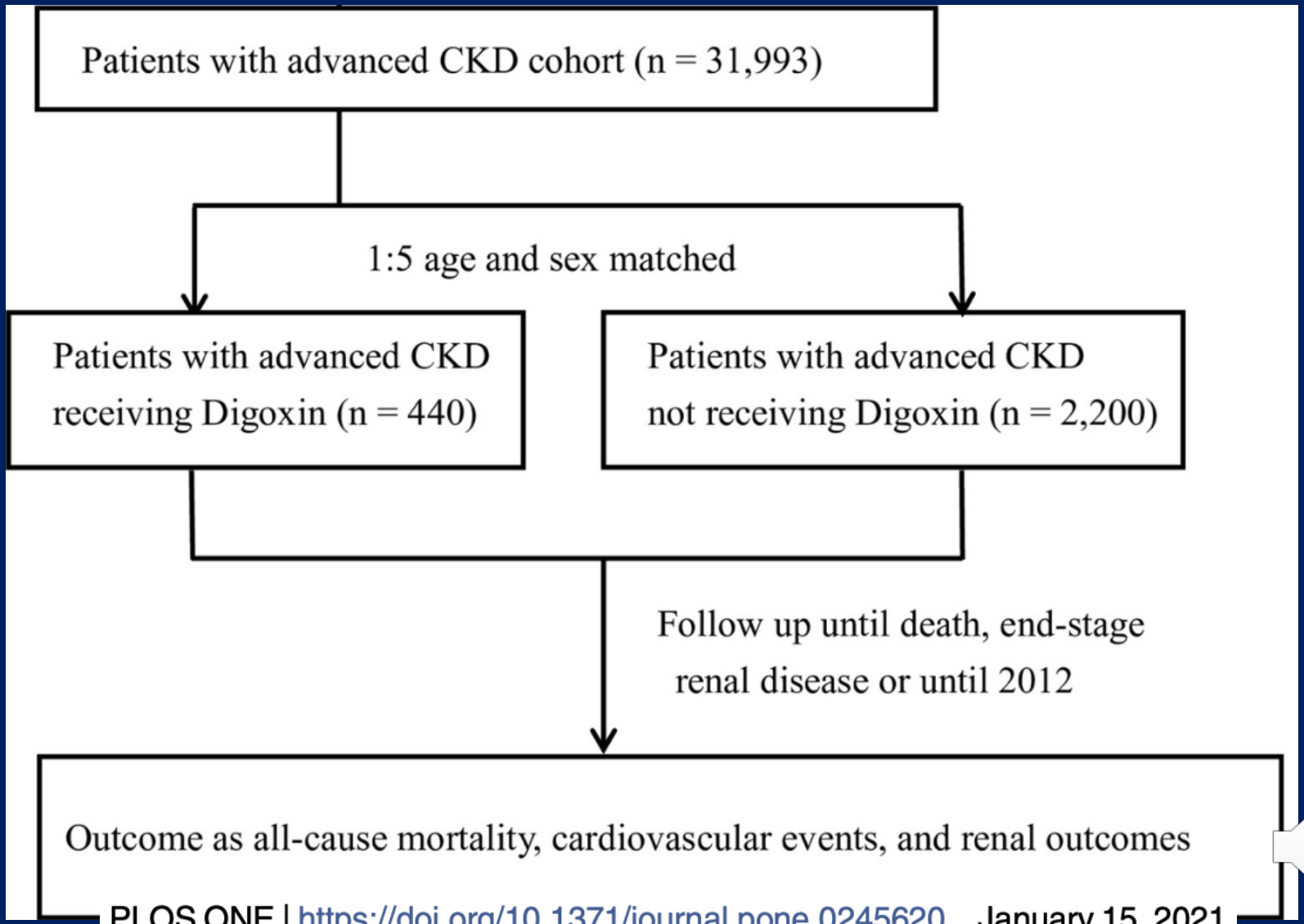
Patient or population: people with chronic kidney disease requiring dialysis
Setting: haemodialysis and peritoneal dialysis
Intervention: aldosterone antagonists (spironolactone or eplerenone)
Comparison: control (placebo or standard care)

Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of participants (RCTs)	Certainty of the evidence (GRADE)
	Risk with control (placebo or standard care)	Risk with aldosterone antagonists (spironolactone or eplerenone)			
Death (any cause)	131 per 1,000	59 per 1,000 (39 to 88)	RR 0.45 (0.30 to 0.67)	1119 (9)	⊕⊕⊕⊖ MODERATE [1]
Death (cardiovascular)	101 per 1,000	37 per 1,000 (22 to 65)	RR 0.37 (0.22 to 0.64)	908 (6)	⊕⊕⊕⊖ MODERATE [1]
Cardiovascular and cerebrovascular morbidity	133 per 1,000	51 per 1,000 (24 to 101)	RR 0.38 (0.18 to 0.76)	328 (3)	⊕⊕⊕⊖ MODERATE [1]
Hyperkalaemia	91 per 1,000	128 per 1,000 (66 to 253)	RR 1.41 (0.72 to 2.78)	981 (9)	⊕⊕⊖⊖ LOW [1 2]
Gynaecomastia	5 per 1,000	31 per 1,000 (10 to 95)	RR 5.95 (1.93 to 18.28)	768 (4)	⊕⊕⊕⊖ MODERATE [1]
Left ventricular mass Measured with different units in the different studies. Lower number mean less hypertrophy	Left ventricular mass in the aldosterone antagonist group was 0.42 **standard deviations** lower (0.05 to 0.78 lower) compared to placebo or standard care*		--	562 (7)	⊕⊖⊖⊖ LOW [1 3]

***The risk in the intervention group** (and its 95% confidence interval) is based on the assumed risk in the comparison group and the **relative effect** of the intervention (and its 95% CI).

Cochrane Database of Systematic Reviews 2021, Issue 2. Art. No.: CD013109.
DOI: 10.1002/14651858.CD013109.pub2.

Digoxin

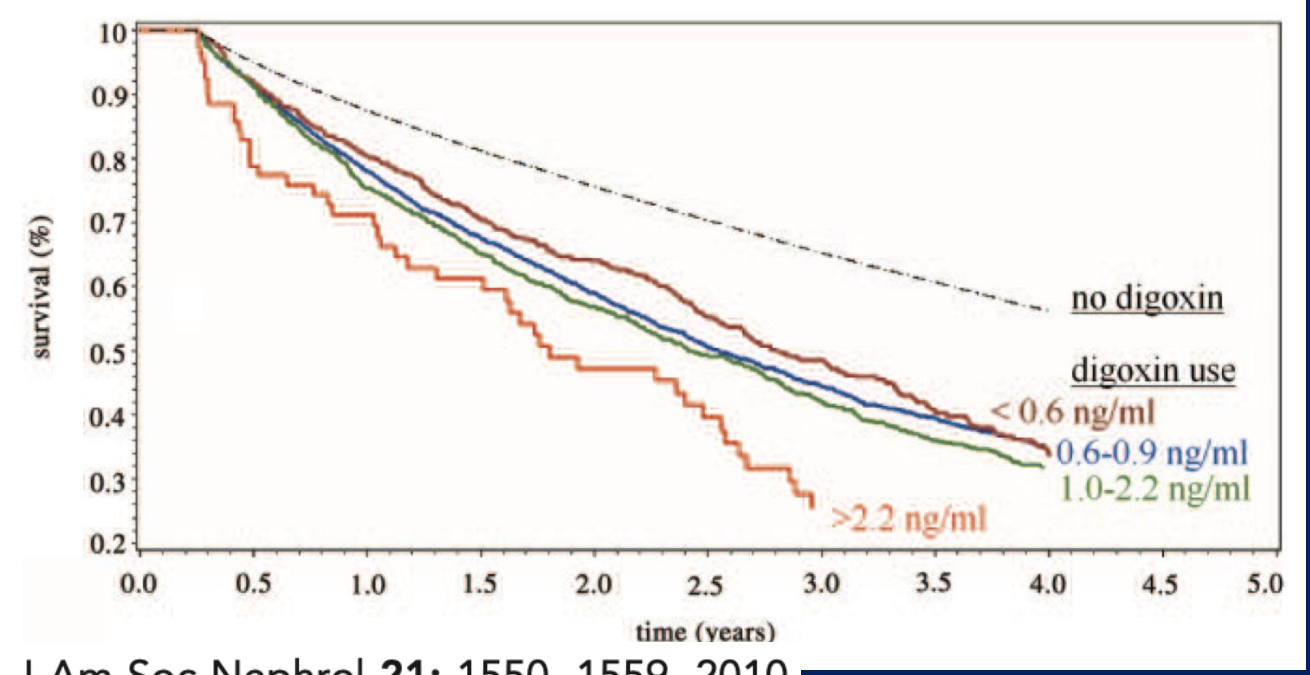


PLOS ONE | https://doi.org/10.1371/journal.pone.0245620 January 15, 2021

Table 2. Association between digoxin used or not and all-cause mortality, major cardiovascular events, and renal function decline in patients with chronic kidney disease using intention-to-treat analysis.

Variable	Overall events			Adjusted Hazard Ratio (95% CI)		
	With Digoxin use	Without Digoxin used	IRR (95% CI)	Model 1[a]	Model 2[b]	Model 3[c]
All-cause mortality[§]	113	268	2.25 (1.81–2.80)***	1.73 (1.32–2.27)***	1.86 (1.41–2.45)***	1.63 (1.23–2.17)***
Major cardiovascular events[¶]	73	212	1.88 (1.44–2.46)***	1.59 (1.13–2.22)**	1.70 (1.20–2.40)**	1.33 (0.95–1.86)
Acute coronary syndrome[¶]	40	116	1.86 (1.30–2.67)***	1.33 (0.85–2.09)	1.41 (0.89–2.24)	1.18 (0.75–1.86)
Ischemic stroke[¶]	32	90	1.91 (1.28–2.86)**	1.74 (1.04–2.91)*	1.79 (1.07–3.00)*	1.42 (0.85–2.37)
Hemorrhagic stroke[¶]	5	23	1.16 (0.44–3.06)	1.20 (0.44–3.26)	1.35 (0.48–3.77)	1.30 (0.44–3.87)
End-stage renal disease[¶]	55	370	0.79 (0.60–1.05)	0.65 (0.47–0.91)*	0.78 (0.55–1.12)	0.80 (0.55–1.14)
Rapid eGFR decline[¶#]	117	518	1.14 (0.93–1.39)	1.08 (0.85–1.37)	1.10 (0.86–1.39)	1.00 (0.78–1.27)
Acute kidney injury[¶]	88	284	1.70 (1.34–2.16)***	1.27 (0.93–1.72)	1.39 (1.02–1.90)*	1.20 (0.87–1.64)

PLOS ONE | https://doi.org/10.1371/journal.pone.0245620 January 15, 2021



J Am Soc Nephrol **21:** 1550–1559, 2010

- Digoxin use was associated with increased mortality.

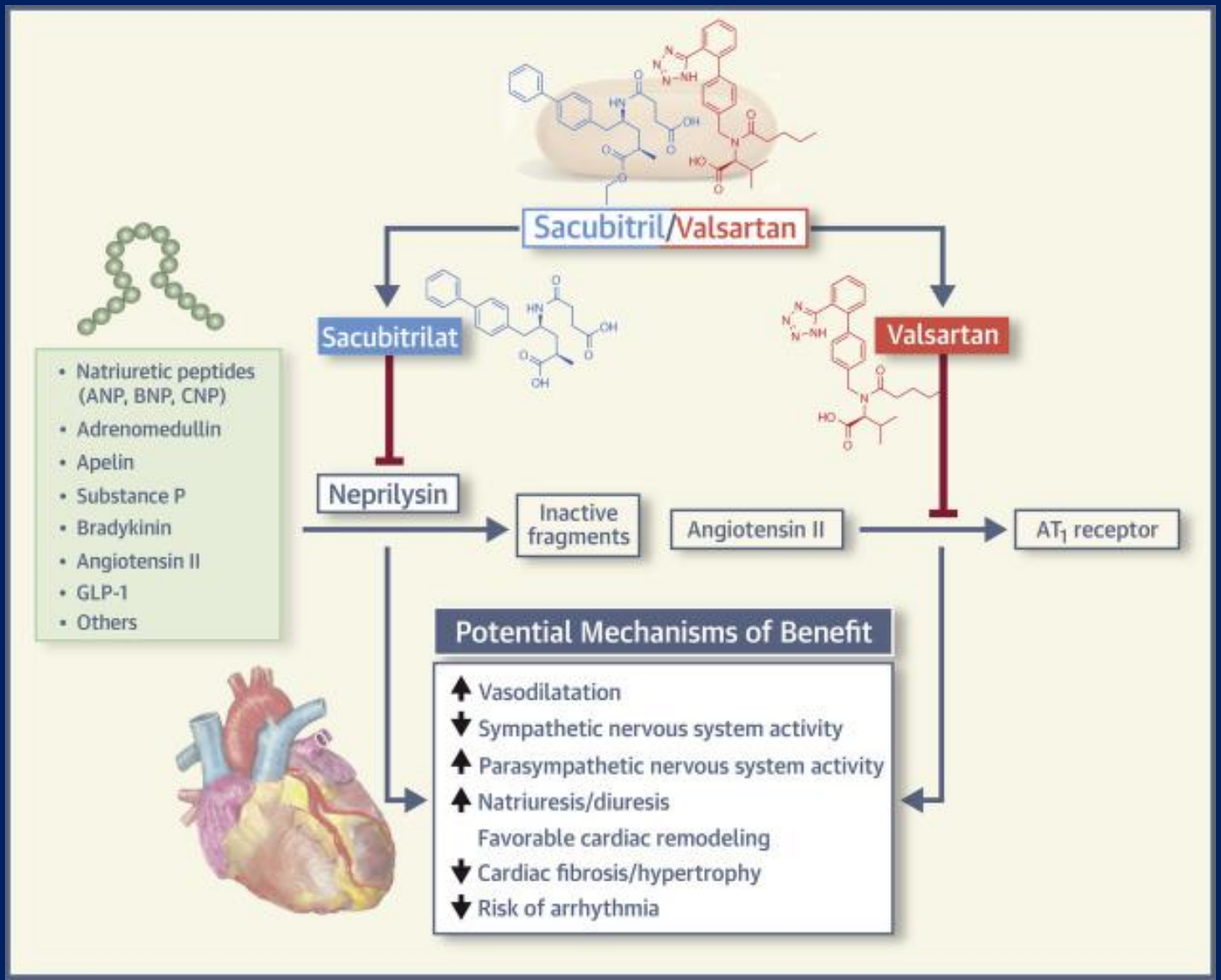
- Reserve digoxin use only for selected patients with AF who have not achieved adequate rate control with optimum β-blockers and who can be closely monitored to maintain digoxin level <1.0 ng/mL.

CCB

- If maximum tolerated disease of beta blocker plus ACE inhibitor is not sufficient for blood pressure control:

- dihydropyridine CCB (amlodipine 5 to 10 mg daily).

ARNI, angiotensin receptor- neprilysin inhibitor



Sacubitril/valsartan in heart failure and end-stage renal insufficiency

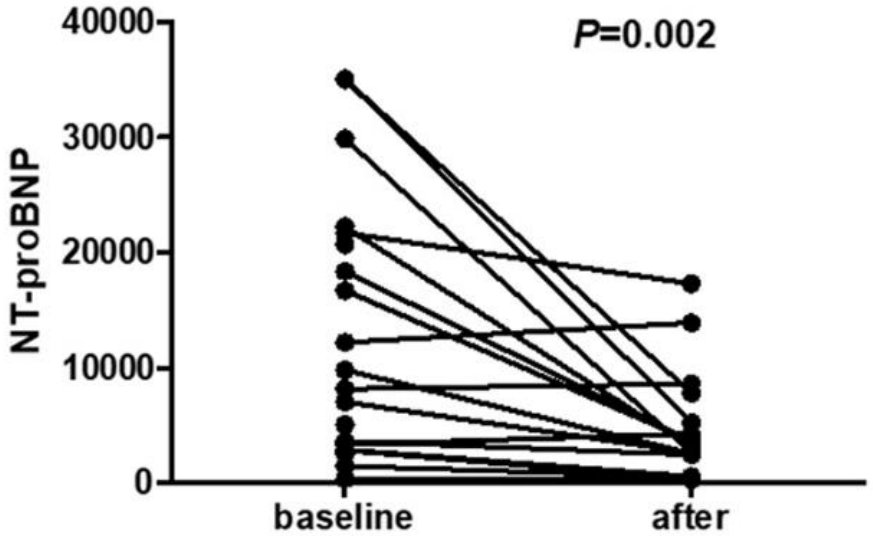
Alex Heyse[1]*, Lynn Manhaeghe[1], Elien Mahieu[2], Céline Vanfraechem[2] and Frederik Van Durme[1]

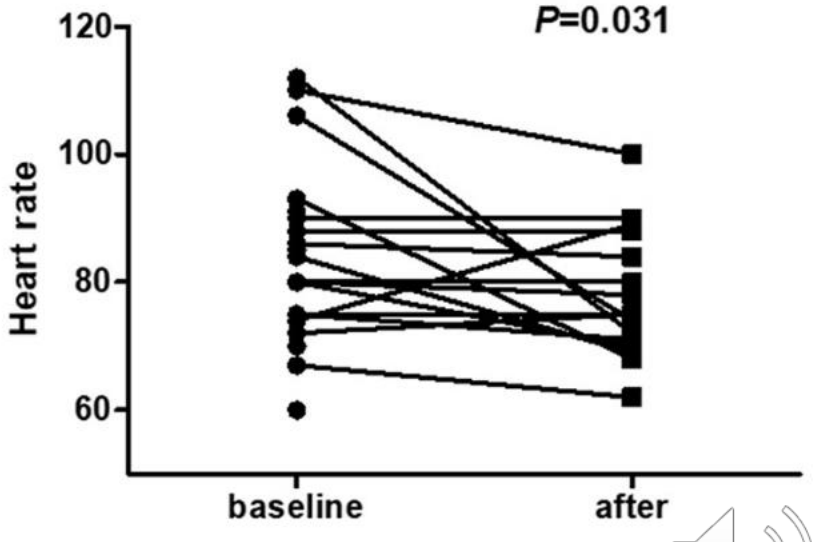
The aim of this report is to describe the feasibility and tolerability of medical treatment with sacubitril/valsartan in a patient treated with hemodialysis. We describe the case of a 67-year-old man with heart failure with reduced ejection fraction due to an ischemic cardiomyopathy and renal insufficiency undergoing hemodialysis. Because of worsening heart failure with no other therapeutic options, a treatment with sacubitril/valsartan was started. Although this patient had a very low systolic blood pressure, he could tolerate a moderate dose of 49/51 mg twice daily. After initiation of sacubitril/valsartan, there was a symptomatic improvement with a clear reduction NT-proBNP, accompanied by a decrease in filling pressures. In conclusion, in this patient with severe heart failure undergoing hemodialysis, treatment with sacubitril/valsartan was feasible, safe, and improved heart failure symptoms.

ESC Heart Failure 2019; **6**: 1331–1333

Effects of Sacubitril-Valsartan in Heart Failure With Preserved Ejection Fraction in Patients Undergoing Peritoneal Dialysis

Sha Fu[1†], Zhenjian Xu[1†], Baojuan Lin[1], Junzhe Chen[2], Qiuyan Huang[1], Yanchun Xu[1], Anping Xu[1], Yangxin Chen[3] and Ying Tang[4*]*





Medication	Benefits in patients with HFrEF not on dialysis	Benefits in patients with HFrEF on dialysis
Beta blockers	Carvedilol, metoprolol, and bisoprolol increase survival, improve symptoms and decrease HF hospitalization	Uncertain. Possible improvement in symptoms
ACEi/ARB	Increases survival, improves symptoms, and decreases HF hospitalization	Uncertain
Mineralocorticoid receptor antagonists	Increases survival, improves symptoms, and decreases HF hospitalization	Uncertain
Loop diuretics	Reduces symptom burden	Minimizes weight gains between treatments if residual kidney function is present
Digoxin	May improve symptoms, quality of life, and exercise tolerance in mild to moderate heart failure; no mortality benefit	Risks likely outweigh any potential benefits
SGLT2 Inhibitors	Increases survival and decreases HF hospitalization	Not studied; effects would likely need to be independent of kidney function
Angiotensin receptor-neprilysin antagonists	Increases survival and decreases HF hospitalization	Not studied

Medical Treatment:

- HFpEF; LVEF >50 percent:
  - Control of volume overload
  - Control of hypertension
  - Control of myocardial ischemia
  - For selected dialysis patients with HFpEF who can be carefully monitored: MRA therapy.

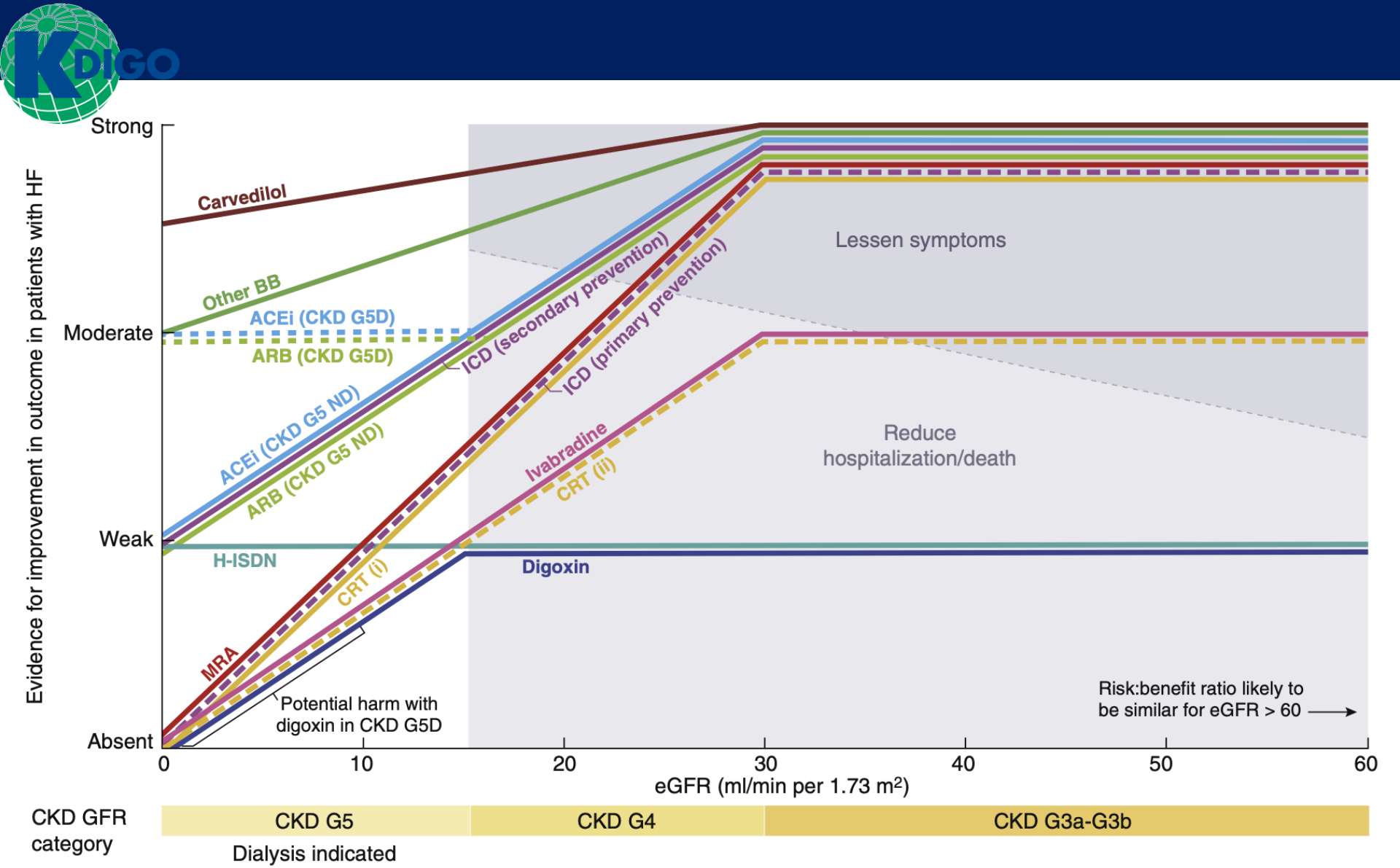
Control of hypertension

- Gradual targeting euvolemia
- Administering antihypertensive medications

- Goal: maintaining interdialytic self-recorded home blood pressure at <130/80 mmHg,

- β-blocker: carvedilol or labetalol or atenolol.
- β-blocker is not tolerated or is not sufficient to achieve target blood pressure: amlodipine
- If the β-blocker plus CCB is not sufficient: ACEi or ARB (candesartan or valsartan)

Class	$T^{1/2}$ in ESRD	Range of dosing (initial to usual or maximum)	%Removal with hemodialysis
Other			
Clonidine	18–41 hours	0.1–0.4 mg bid-tid	<5%
Hydralazine	7–16 hours	10–100 mg q8 hour	None
Isosorbide dinitrate	?	5–40 mg tid	Yes
Minoxidil	?	5–100 mg qd	Partially



CRT (i) = QRS > 120 ms, LBBB QRS morphology, EF ≤ 35%
or QRS > 130 ms, EF ≤ 30%
CRT (ii) = QRS > 150 ms

Loop diuretics (p.o./i.v.) (furosemide, bumetanide, torsemide) and thiazide diuretics (metolazone [p.o.], chlorothiazide [i.v.]) = benefit uncertain

Iron

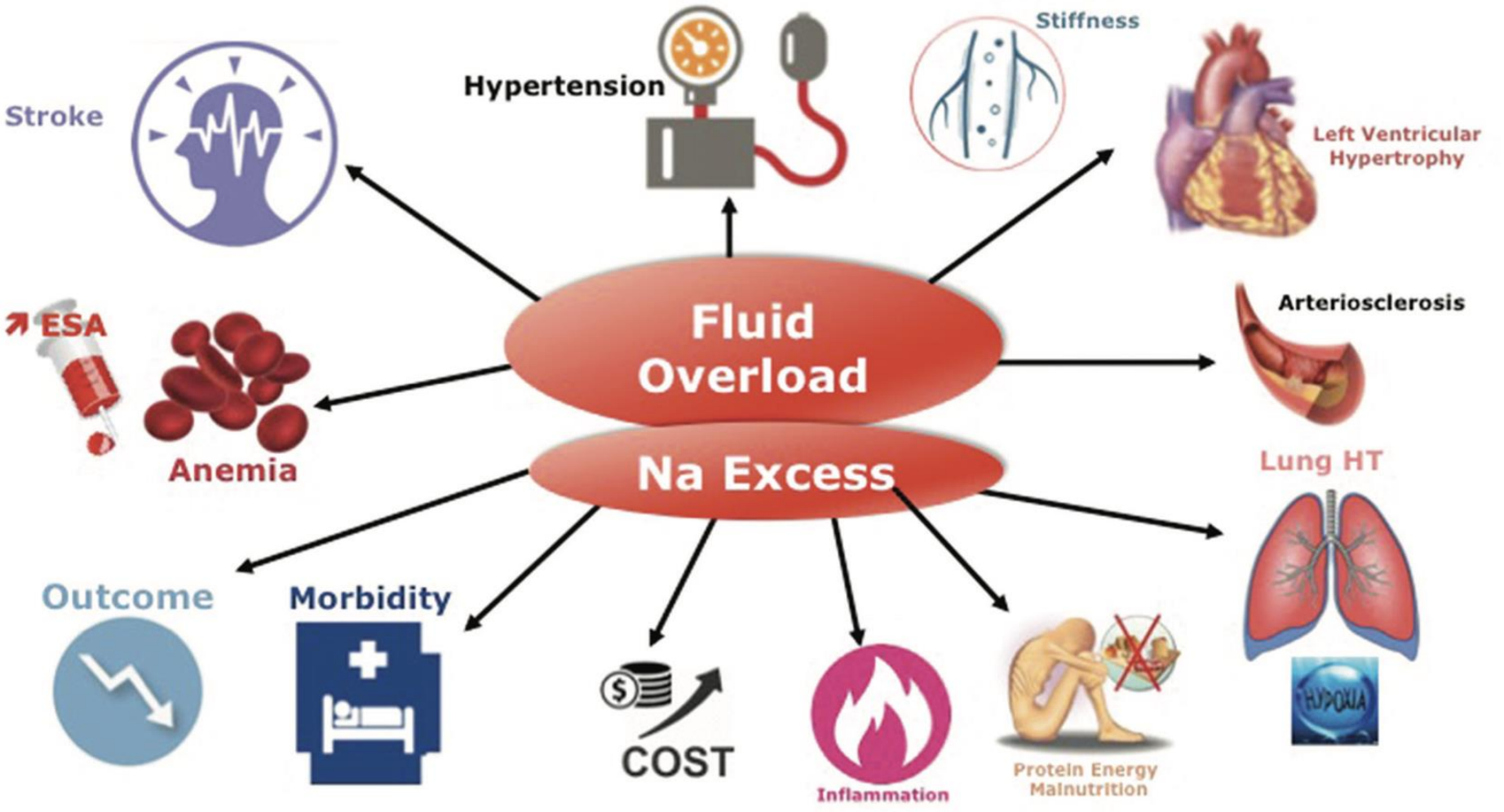
- Hospitalizations for HF and mortality were significantly decreased in the iron-treated group:

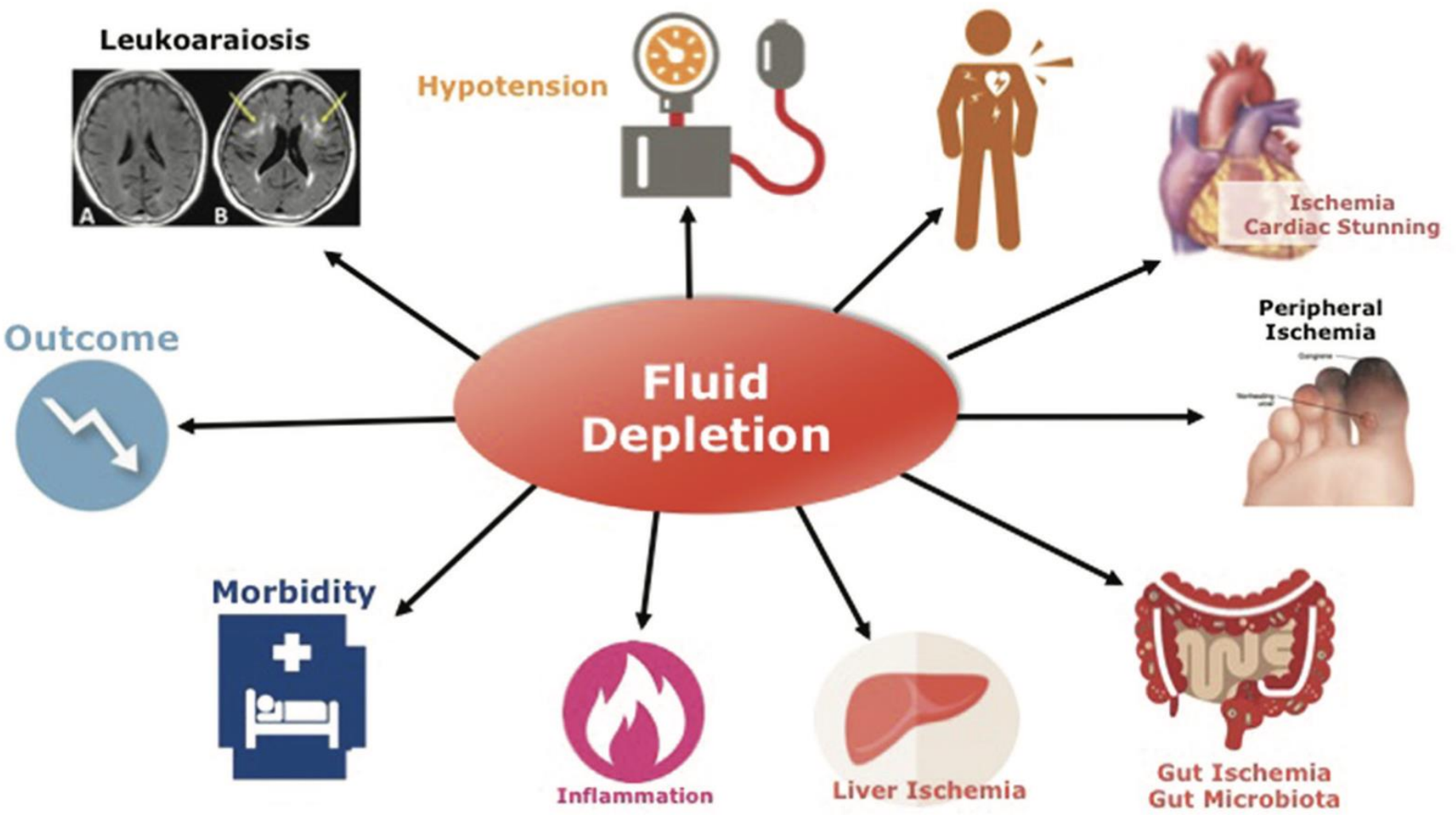
- Ferritin level <100 mg/l, or < 300 mg/l if transferrin saturation is <20%, irrespective of hemoglobin level.

Management of Iron Deficiency and Anemia in Patients with Heart Failure with Reduced Ejection Fraction and CKD

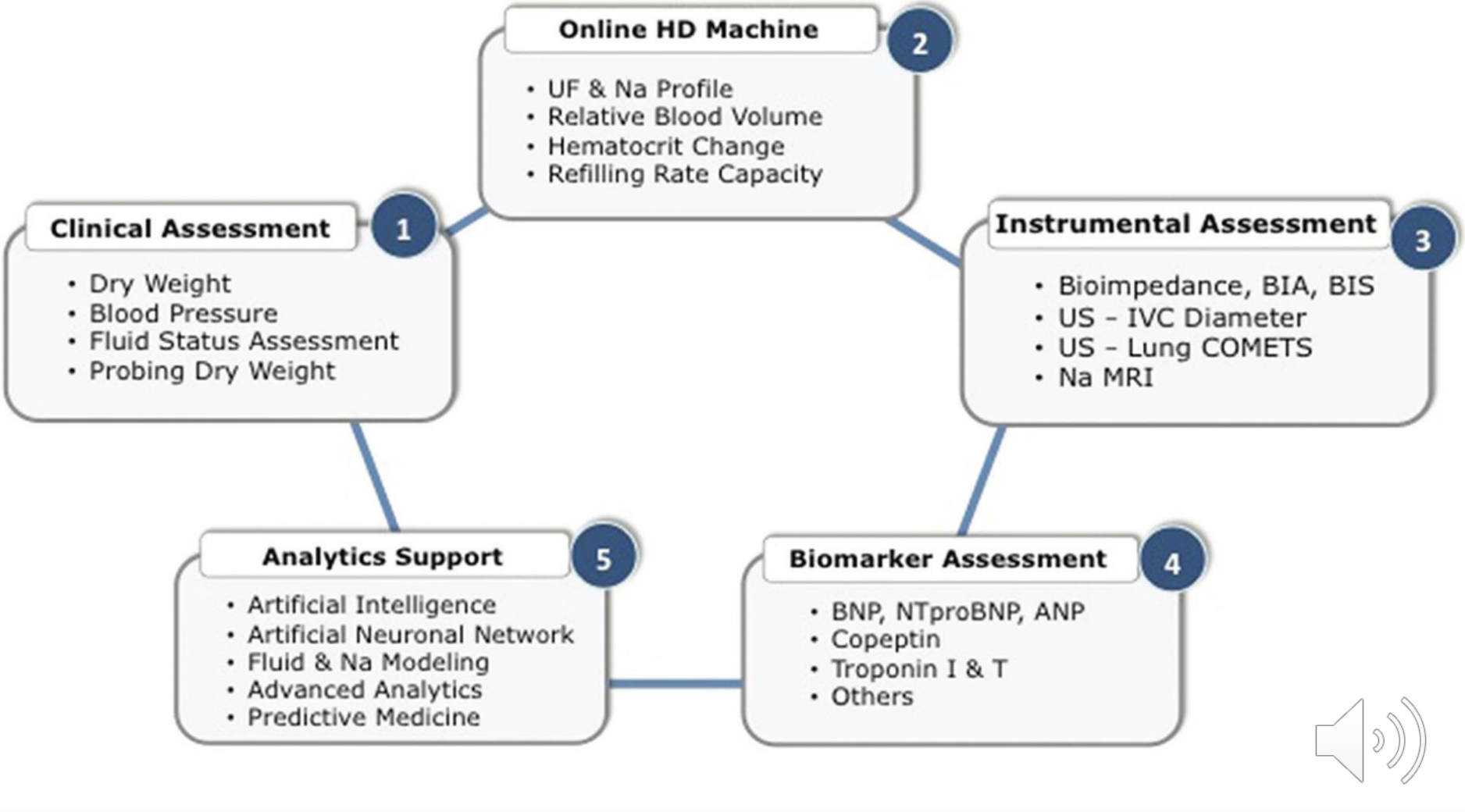
Nephrologists have been using intravenous iron for the past 3 decades to treat anemia in patients with CKD, both before and during dialysis. In the United Kingdom, a recent study in patients receiving dialysis has shown benefits of high-dose intravenous iron in reducing mortality and morbidity, together with heart failure hospitalizations by 44% (37). A previous trial of benefits of intravenous iron in patients with HFrEF included patients with early stages of CKD (38). A collaboration between cardiologists and nephrologists may assist the management of iron deficiency in patients with HFrEF and CKD.

Volume Overload


Stroke
Hypertension
Stiffness
Left Ventricular Hypertrophy
ESA
Anemia
Fluid Overload
Na Excess
Arteriosclerosis
Lung HT
Outcome
Morbidity
COST
Inflammation
Protein Energy Malnutrition
HYPOXIA


Leukoaraiosis
A
B
Hypotension
Ischemia
Cardiac Stunning
Peripheral
Ischemia
Outcome
Fluid
Depletion
Morbidity
Inflammation
Liver Ischemia
Gut Ischemia
Gut Microbiota

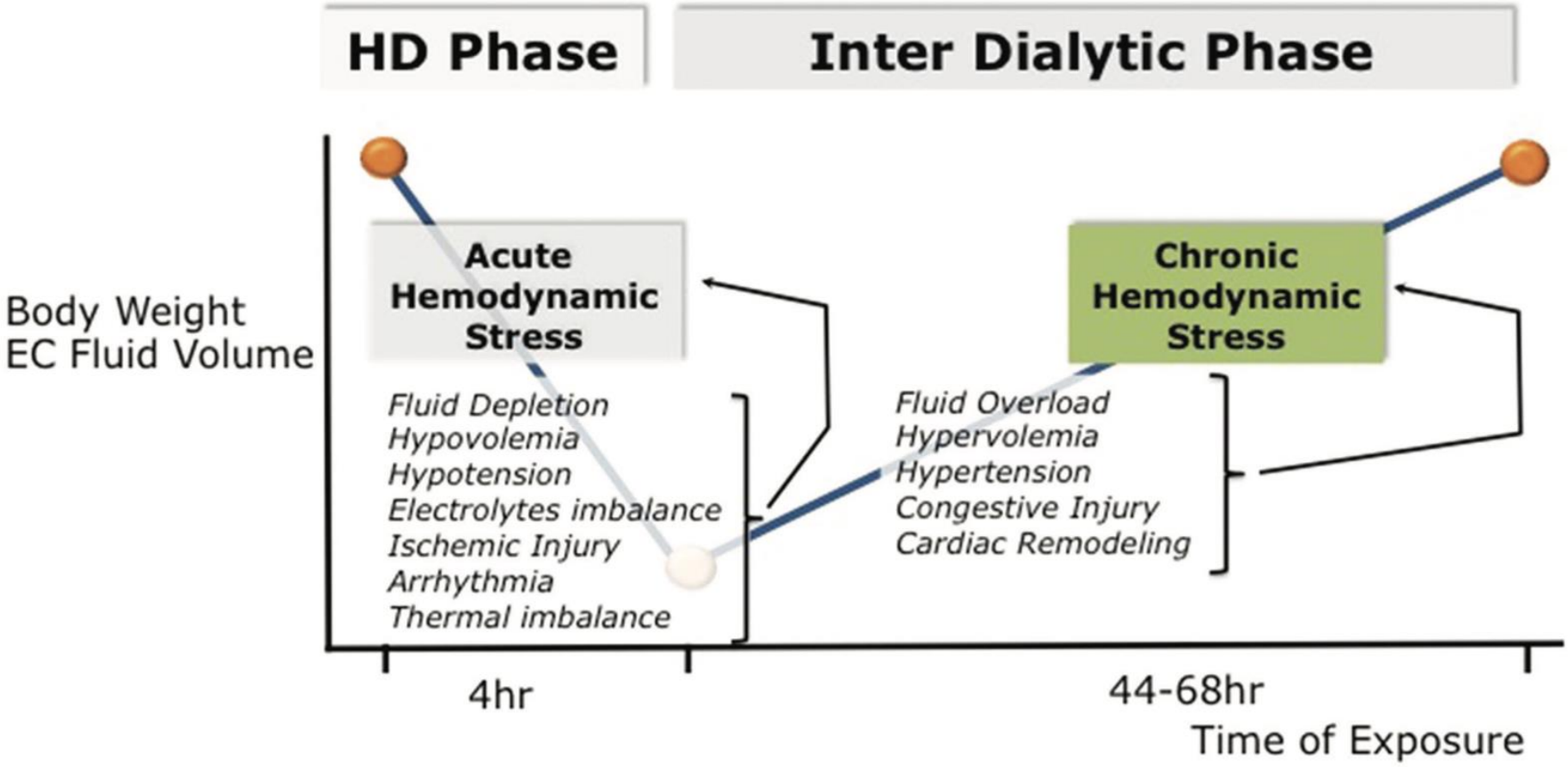
DETECTING VOLUME OVERLOAD

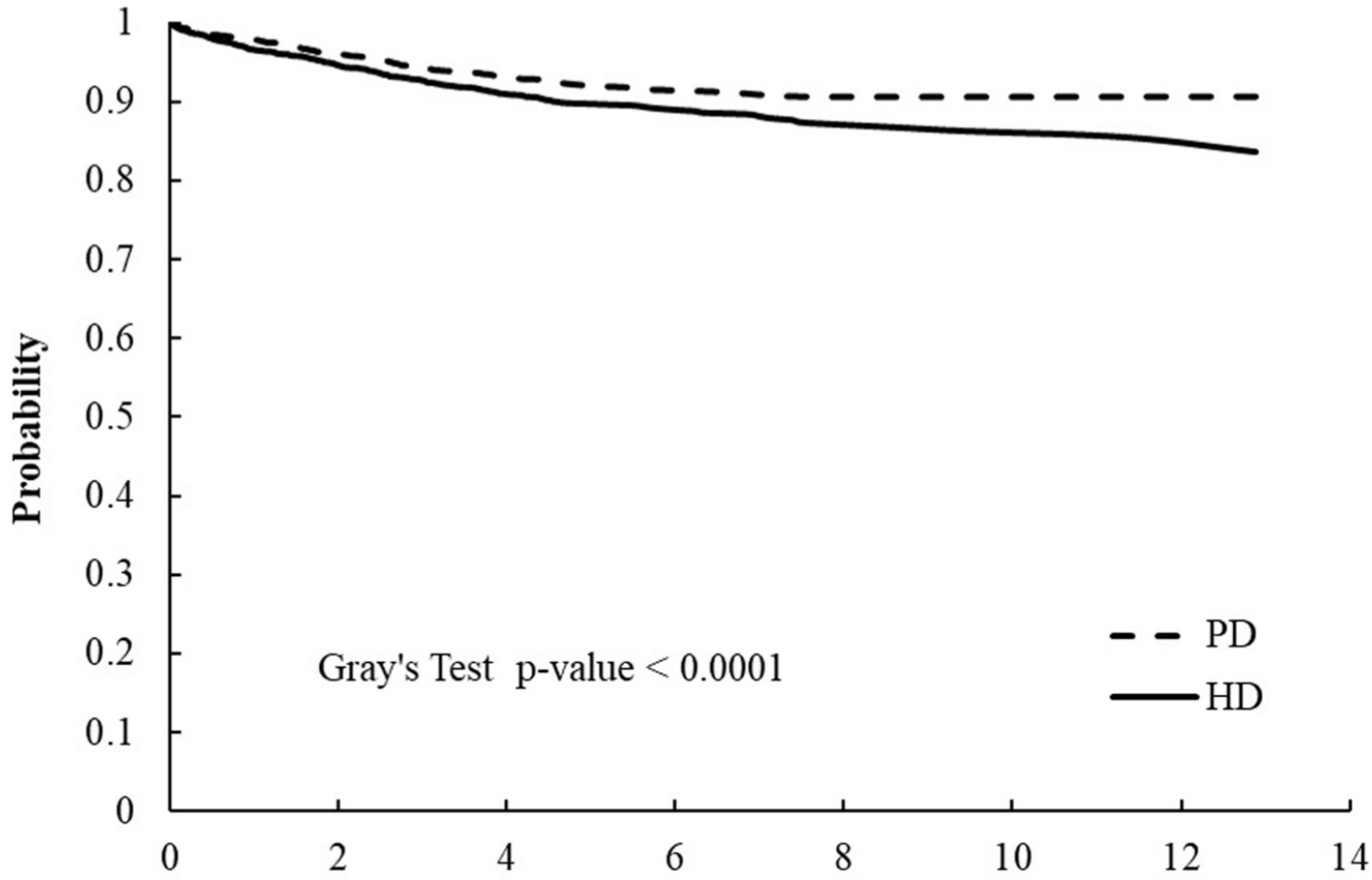


Dialysis

- Control of volume overload
  - In HFpEF
  - In HFrEF
- Removal of uremic toxins

Dialysis Modality


HD Phase
Inter Dialytic Phase
Body Weight
EC Fluid Volume
Acute Hemodynamic Stress
Chronic Hemodynamic Stress
Fluid Depletion
Hypovolemia
Hypotension
Electrolytes imbalance
Ischemic Injury
Arrhythmia
Thermal imbalance
Fluid Overload
Hypervolemia
Hypertension
Congestive Injury
Cardiac Remodeling
4hr
44-68hr
Time of Exposure



No. at risk:

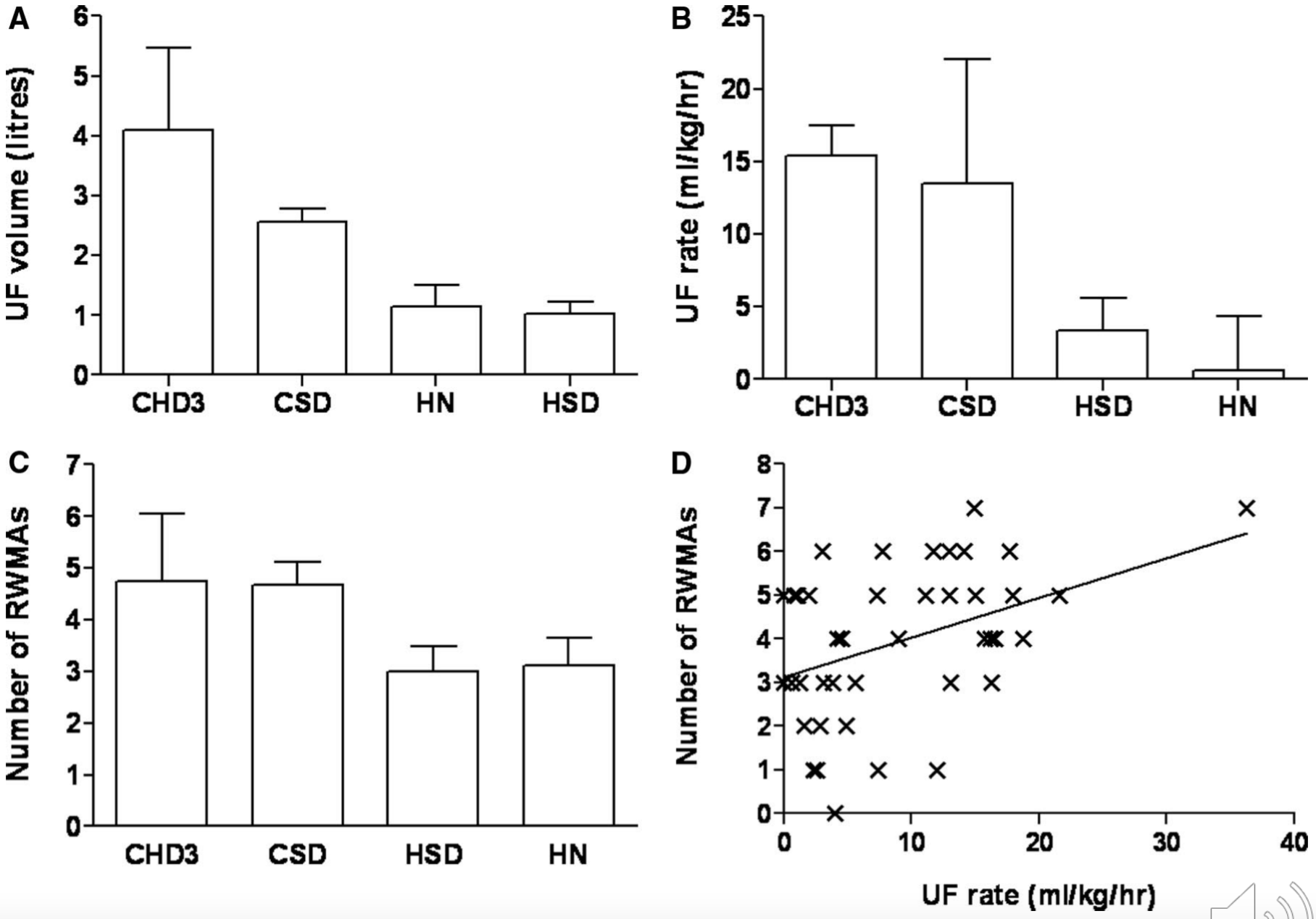
HD	4754	2798	1392	332	161	63	23
PD	4754	2530	1037	242	71	21	21

Performing hemodialysis in HFrEF

- Several potential strategies that may reduce myocardial stunning, reduce morbidity, and enhance the tolerability of hemodialysis in HFrEF.

- Treatment Time
- Frequency of treatment
- Ultrafiltration rate
- Dialysate cooling
- Dialysate Na
- Dialysate Ca

Treatment Time
Frequency of treatment
Ultrafiltration rate



Mean RWMAs per patient reduced with increasing dialysis intensity (CHD3 > CSD > HSD > HN).

Predictors of Change in Left-Ventricular Structure and Function in a Trial of Extended Hours Hemodialysis

	Change in SV (mL)			
	Univariable (95% CI)	*P* value	Multivariable (95% CI)	*P* value
Pathophysiologic pa				
Normalized UF rate (mL/hr/kg)	−0.91 (−1.73, −0.08)	0.032		
Weekly total weight gain (kg)	−1.06 (−1.91, −0.22)	0.014	−0.87 (−1.69, −0.05)	0.038
SBP (mmHg)	−0.04 (−0.33, 0.26)	0.80		
DBP (mmHg)	−0.20 (−0.67, 0.27)	0.40		
Phosphate (mmol/L)	0.32 (−8.19, 8.84)	0.94		
Total hours per week (h)	−0.27 (−1.14, 0.60)	0.56		
Sessional Kt/V	0.33 (−13.78, 14.44)	0.96		

Using more frequent haemodialysis to manage volume overload in dialysis patients with heart failure, obesity or pregnancy

- Increasing the frequency of dialysis treatments to five to six times per week, providing increased volume control by reducing intertreatment cardiac loading.

- The increased total weekly dialysis time reduces the UFRs, thereby also reducing the potential for intratreatment hypotension and cardiac stunning.

Nephrol Dial Transplant (2020) 35: ii11–ii17
doi: 10.1093/ndt/gfaa020

Table 1. Patient 1 treatment course for a 76-year-old female, 71 kg, with renal failure due to glomerulonephritis and with heart failure

Date	Echocardiographic data	Dialysis and cardiac medication	Symptoms
November 2014	EF < 20%	PD, Biventricular pacemaker	Blackout, SOB, 20 kg overloaded, mobility scooter
March 2015	EF 10%	ICHD, all meds stopped	Blacking out on HD, very low BP, increased overload
October 2015	EF 17.8%	4 times a week, small dose β-blocker (4 h, 40 L)	Not lowering BP, slowly reducing weight
February 2016	EF 31%	6 times a week at home, increase β-blocker (2.5 h, 20 L)	No oedema, maintaining BP, walking
June 2017	EF 35%	5 times a week at home (3 h, 30 L)	Improved mobility, stick only, not SOB

Table 2. Patient 2 treatment course for a 74-year-old female, 67 kg, with renal failure due to glomerulonephritis and with heart failure, pulmonary hypertension and hypotension

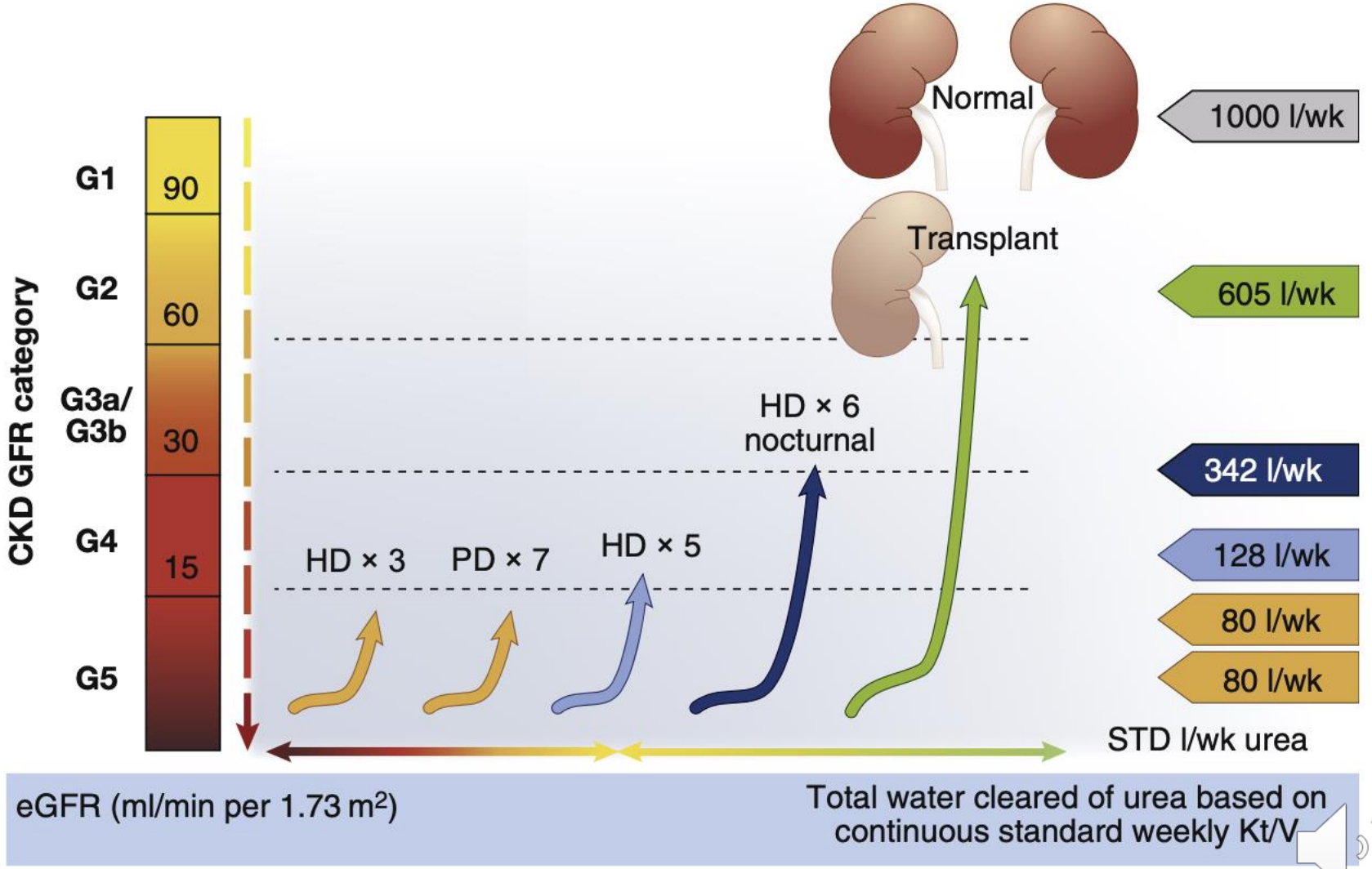
Date	Echocardiographic data	Dialysis and cardiac medication	Symptoms
2014	EF 41%, PAP 60 with severe TR	ICHD 3 × 4 h/week ARB stopped; calcium channel antagonist stopped	Symptomatic hypotension Not tolerating UF
2015	EF 60%, PAP 77, moderate–to–severe TR	Frequent HHD, 6 × 2 h with 20 L	Development of significant ascites, significant SOB
2016	EF 55–65%, PAP 21, mild TR	Frequent HHD, 6 × 2 h 45 min, 25 L, reintroduction of ARB	Reduction in ascites, improvement in SOB
2019	EF 60–65%, normal PAP, trivial TR	Frequent HHD, 6 × 2.5 h. ARB and calcium channel antagonist	No ascites, no SOB, improved exercise tolerance

Table 3. Patient 3 treatment course for a 53-year-old, 85-kg male with renal failure due to glomerulonephritis and a history of hypertrophic obstructive cardiomyopathy

Date	Echocardiographic data	Dialysis and cardiac medication	Symptoms
2015	EF 45–55% PAP 45 Moderate MR, hypertrophic obstructive cardiomyopathy	PD	Fluid overloaded
2016	EF 40–45% PAP > 55 Moderate–severe MR	Haemodialysis 3 × 4 h β-blocker	Hypotensive episodes on dialysis and SOB
2018 (February)	EF35% PAP38 Moderate–severe MR and TR	Perioperative cardiac event in hospital 3 times a week	Sever SOB, limited exercise tolerance
2018 (November)	EF 65%	Home HD 5 × 3 h, 30 LARB	Tolerating dialysis without issue Exercise improved

- PD or extended and more frequent hemodialysis in patients with HFrEF provide:
  - More consistent volume control with lower ultrafiltration rates
  - Greater hemodynamic stability,
  - Less myocardial stunning.

Weekly total water cleared of urea based on continuous weekly Kt/V according to chosen method of dialysis



Cooling of dialysate

- Cooling of dialysate is another strategy that may reduce intradialytic hypotension and myocardial stunning.

- Set the dialysate temperature 0.5 to 0.9 °C below each patient's body temperature (measured before starting the hemodialysis treatment) to a minimum of 35.5°C

Strategy	Benefits	Limitations
Longer or more frequent dialysis	Lower interdialytic weight gain allows for slower ultrafiltration with smaller amounts of volume removal at each session	Patient preference, cost
Dialysate cooling	May lead to less myocardial stunning, less intradialytic hypotension, and less cardiac remodeling	The optimal temperature has not been defined. Occasional patient discomfort.
Maintain dialysate calcium ≥ 2.5 mEq/L	May lead to less myocardial stunning, less intradialytic hypotension, and fewer HF hospitalizations	Possible promotion of vascular calcification.
Higher dialysate sodium	Less intradialytic hypotension, resulting in better single session tolerance	Increased interdialytic weight gain, thereby worsening heart failure
Dietary sodium restriction	May decrease fluid retention and congestive symptoms	May be associated with higher hospitalization rates and increased neurohormonal activation
Midodrine	Less intradialytic hypotension. May decrease dyspnea presumably through better volume removal during dialysis	May be associated with increased mortality

Note: Longer or more frequent dialysis refers to increasing dialysis time; this can be accomplished through longer hemodialysis sessions (including nocturnal), more hemodialysis sessions, and peritoneal dialysis.

Dialysis patients presenting with acute or chronic heart failure

- Rule out the presence of significant myocardial ischemia
- Early echocardiography to assess the type and severity of LV dysfunction and cardiac abnormality
- Assess volume status and blood pressure
- If evidence of volume overload, correct volume overload by –
    1. salt and fluid restriction
    2. intravenous frusemide (for those with urine output)
    3. extracorporeal or peritoneal ultrafiltration (for HD and PD patients, respectively)
    4. hemodiafiltration
- If evidence of significant myocardial ischemia causing cardiac dysfunction, consider early revascularization

- Improve blood pressure control if remains hypertensive after correction of volume overload
- Medications (to be added at a low dose and then stepped up gradually according to blood pressure response):


Thanks for your attention
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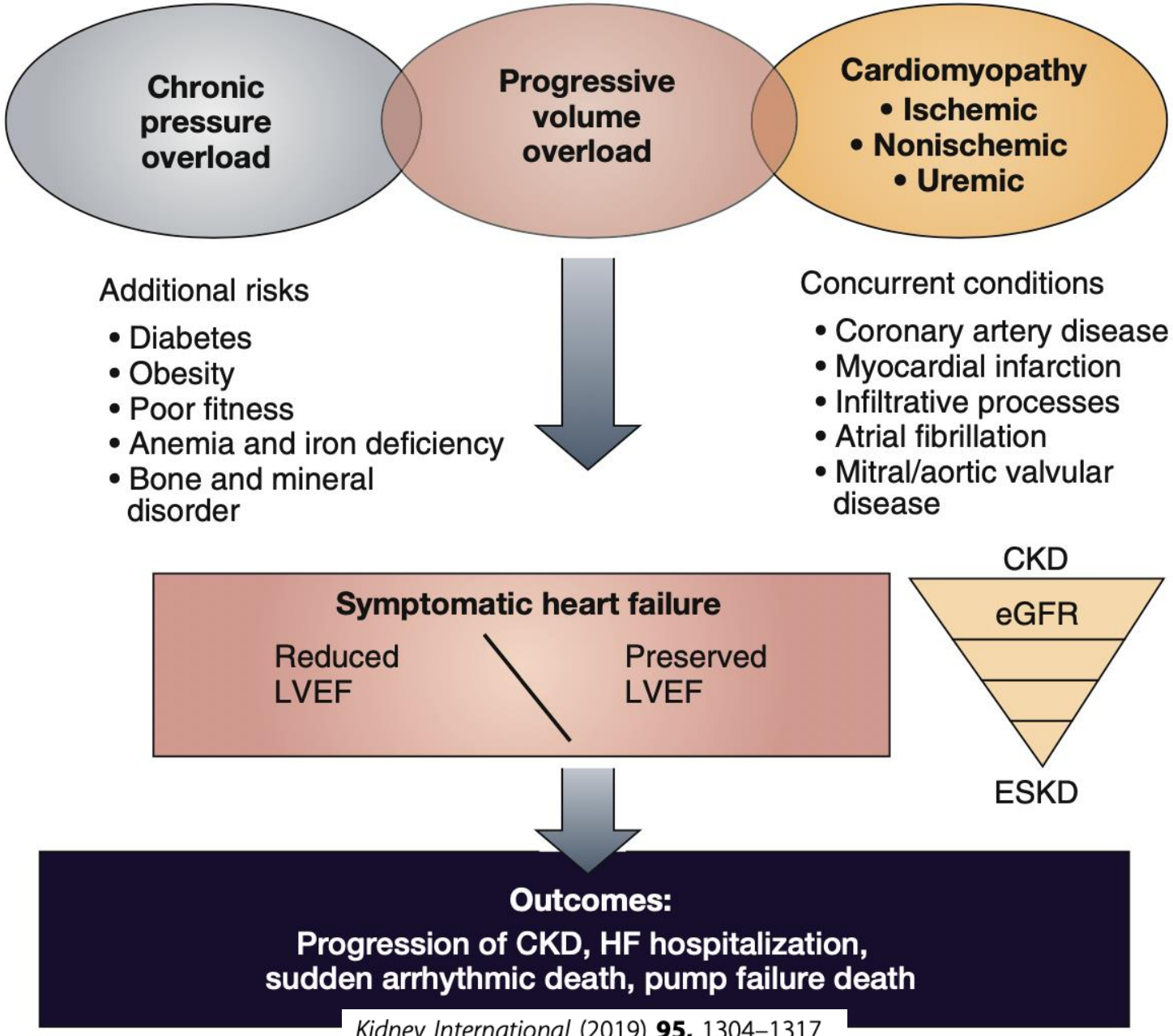
Pathophysiology of heart failure in CKD progressing to ESKD



Kidney International (2019) **95,** 1304–1317