

Nierfunctie de basis:



definitie en meting

Definitie nierfalen

verminderde glomerulaire filtratie snelheid = GFR

en/of urinair eiwitverlies = albumine

Chronisch: bij **herhaling na 3 maanden**



Acuut: ontstaan **binnen de 7 dagen** en **reversibel**

Chronisch nierfalen: stadia

CKD Progresses in Stages (Defined by eGFR)

CKD Stage	Description	GFR (mL/min/1.73 m ²)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60-89
3	Moderate ↓ GFR	30-59
4	Severe ↓ GFR	15-29
5	Kidney failure	<15 or dialysis

Nierfalen: Wie screenen?



Nierfalen: Hoe screenen?

Nefrouupdate congres
9/2023

eGFR and Albuminuria for Risk Assessment

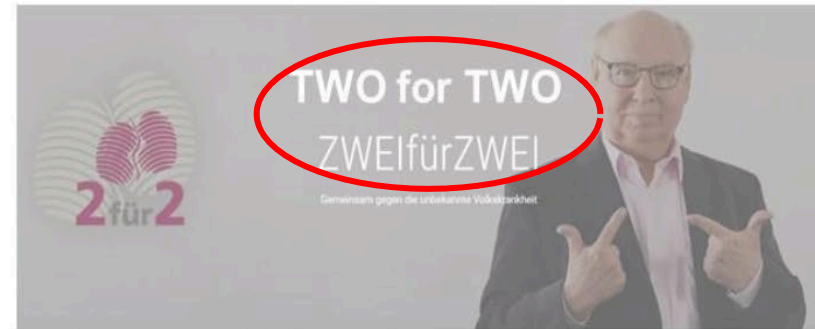
CV risk profile assessment

		Albuminuria Categories (mg/g)			
		A1	A2	A3	
		<30	30-299	≥300	
GFR Categories (mL/min/1.73 m ²)	G1	>90	30.0%	1.9%	0.4%
	G2	60-89	30.3%	2.2%	0.3%
	G3a	45-59	3.6%	0.8%	0.3%
	G3b	30-44	1.0%	0.4%	0.2%
	G4	15-29	0.2%	0.1%	0.1%
	G5	<15	0.0%	0.0%	0.0%

ABCDE profile
↑ **a**lbuminuria
blood pressure
cholesterol
diabetes status
eGFR

plus obesity and smoking

Campaign of the German Kidney Foundation



<https://www.nierenstiftung.de/2fuer2/>

Ortiz et al. *Europ J Prev Card.* 2022; 9:2211-2215.

Nierfalen: Waarom screenen?

KDIGO Classification and Prognosis of Chronic Kidney Disease 2012

Prognosis of CKD by GFR and albuminuria category

Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012

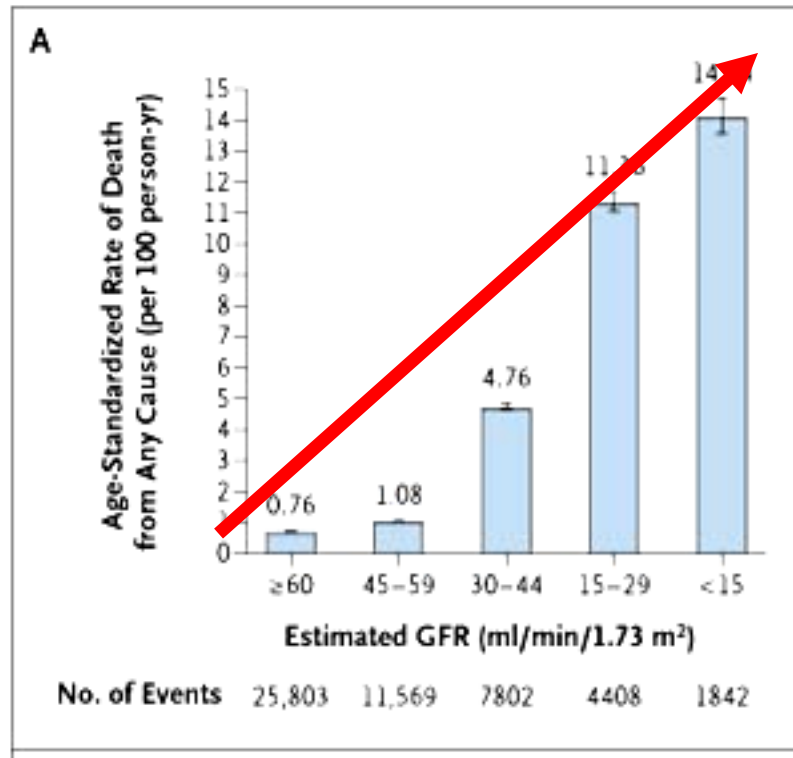
				Persistent albuminuria categories description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (ml/min/1.73 m ²) description and range	G1	Normal or high	≥90	Green	Yellow	Orange
	G2	Mildly decreased	60–89	Green	Yellow	Orange
	G3a	Mildly to moderately decreased	45–59	Yellow	Orange	Red
	G3b	Moderately to severely decreased	30–44	Orange	Red	Red
	G4	Severely decreased	15–29	Red	Red	Red
	G5	Kidney failure	<15	Red	Red	Red

Green: low risk (if no other markers of kidney disease, no CKD); yellow: moderately increased risk; orange: high risk; red: very high risk.

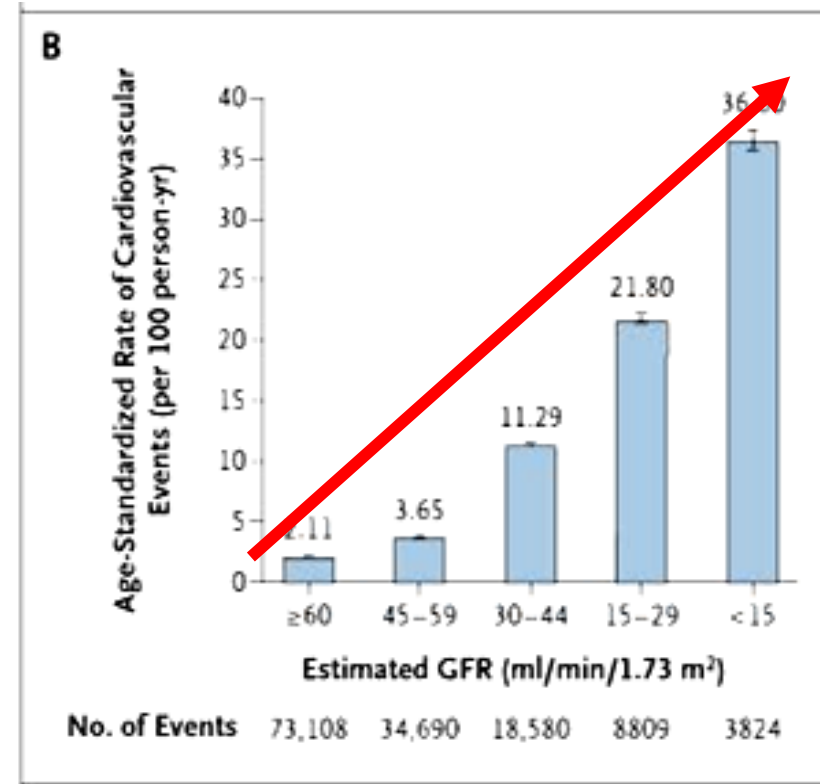
Nierfalen: waarom screenen?

GFR onafhankelijke risicofactor mortaliteit

Mortaliteit

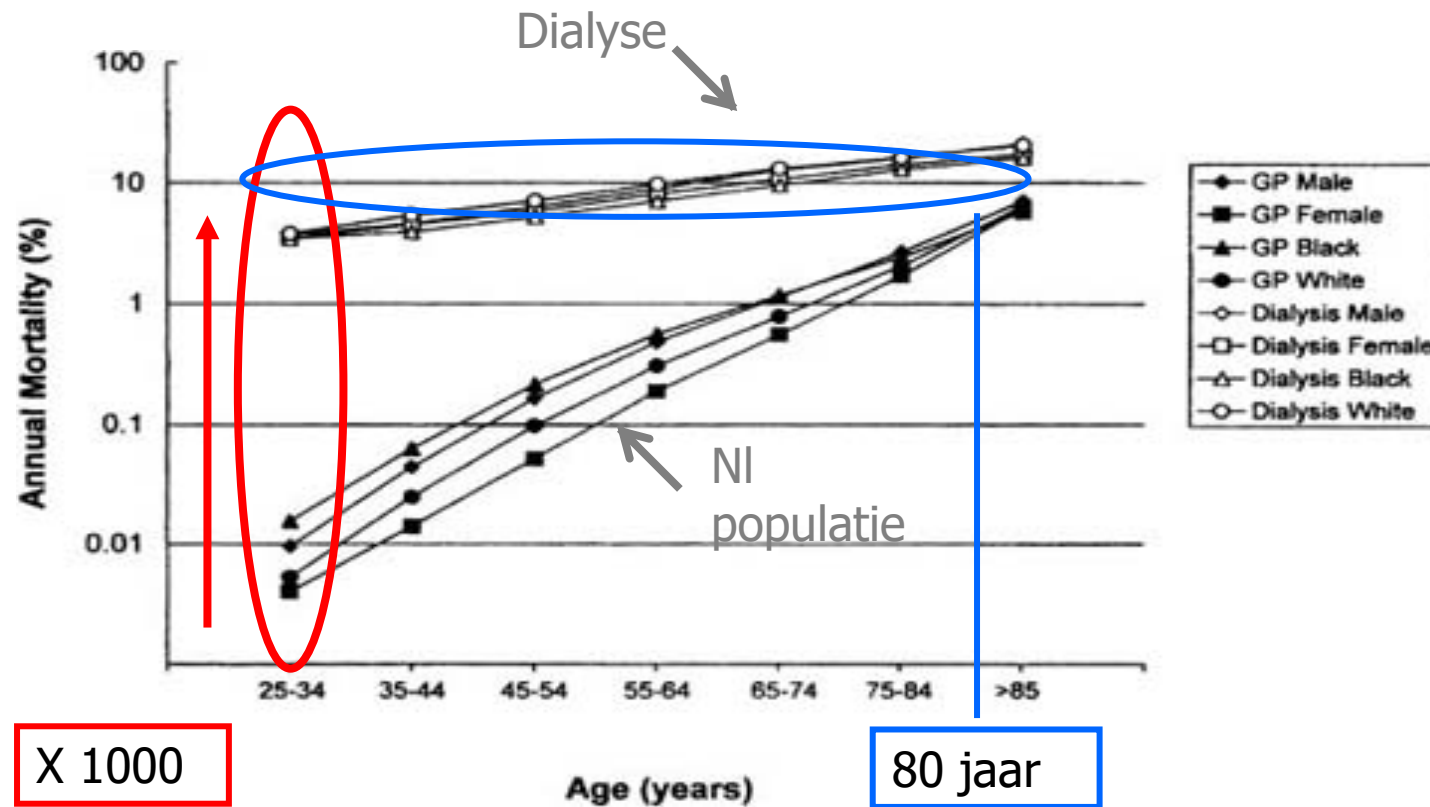


Cardiovasculaire events



Nierfalen: waarom screenen?

GFR onafhankelijke risicofactor mortaliteit



Foley et al AJKD,
1998

Nierfalen: waarom screenen?

GFR en **proteinurie**: belangrijkste onafh parameters die risico op dialyse voorspellen

	eGFR ≥60	eGFR 45–59	eGFR 30–44	eGFR 15–29
<i>Adjusted relative risk</i>				
Normal ACR	1.0 (ref.)	11.7	25.3	120
Microalbuminuria	18.2	60.9	183	765
Macroalbuminuria	110	302	934	982
<i>Absolute risk</i>				
Incidence rate (per 100,000 person-years)	Reference	Low risk	Moderate risk	High risk
	4	69	586	3,938

**Risico op dialyse als
macroproteinurie:**

X 40

X 10



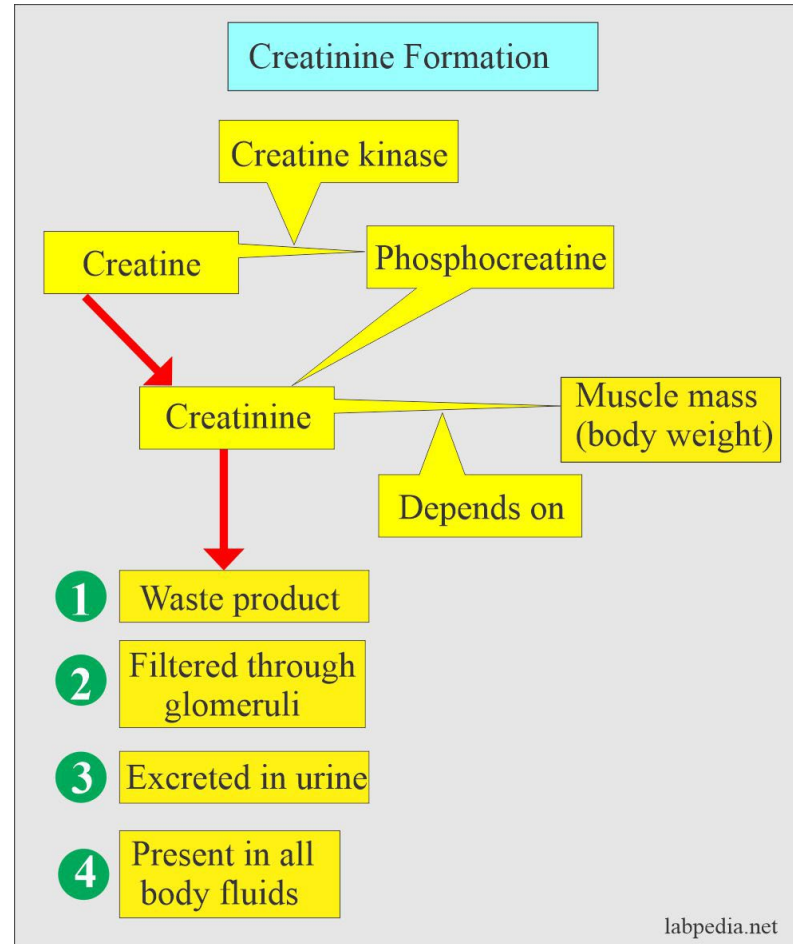
Meting nierfalen

- Serumcreatinine
- Formules
- 24 uurse urine collectie

Nefrouupdate congres
9/ 2023

- **Nieuwer cystatine:** niet in gebruik bij ons, Nederland universiteiten wel..

Creatinine



vrij gefilterd door glomerulus
niet gereabsorbeerd
Niet gesecreteerd door tubuli

Dus creatinine = 'balans tussen aanmaak in spieren en filtratie door glomerulus '

Creatinine

- **voeding** (oa stoofvlees)
 - **Spiermassa**
 - **referentiewaarden** verschillen..
 - **tussen labo's onderling**
 - **tussen geslacht/ras/leeftijd**
- *vrouw: 0.5-1.1 mg/dL*
- *man: 0.6-1.2 mg/dL*
- *kind: 0.3-0.7 mg/dL*



Ps: **ureum niet meer standaard gegeven** (recent riziv beperking tenzij e GFR<30)

Meting nierwerking: e GFR "schattingen"

Cockcroft Gault Equation	MDRD	CKD-EPI equation
$eCrCl = \frac{(140 - \text{Age}) \times \text{Weight (kg)}}{72 \times \text{Creatinine}_{\text{serum}} \text{ (mg/dL)}} \times 0.85 \text{ if female}$	<p>Equation for estimating GFR in patients with lower level of GFR.</p> $\text{GFR (mL/min/1.73m}^2\text{)} = 170 \times [P_{\text{CR}}]^{-0.999} \times [\text{Age}]^{-0.176} \times [\text{SUN}]^{-0.170} \times [\text{Alb}]^{+0.318} \times 0.762 \text{ if patient is female} \times 1.180 \text{ if patient is black}$	<p>Equation for patient with normal or near GFR, higher BMI, elderly, transplant and diabetic patients.</p> <p>CKD-EPI</p> <ul style="list-style-type: none">• $e\text{GFR} = 141 \times \min([\text{creat}/\kappa, 1])^\alpha \times \max([\text{creat}/\kappa, 1])^{-1.209} \times 0.993^{\text{Age}} \times 1.018$ [if female] $\times 1.159$ [if black]• $\kappa = 0.7$ if female, $\kappa = 0.9$ if male.• $\alpha = -0.329$ if female, $\alpha = -0.411$ if male• min = the minimum of Scr/κ or 1, max = the maximum of Scr/κ or 1• Probably more accurate than MDRD• Certainly better if GFR > 60mls/min• New (2009) and MDRD remains the NICE approved formula

bijsluiters medikatie

bij slechte nierwerking

bij goede nierwerking

Casus

- Mariette
- 84 jaar
- Ernstige urosepsis
- 4 weken Inzo



Labo: Creatinine 1,2 mg/dl (ref 0,9-1,2 mg/dl vrouw)

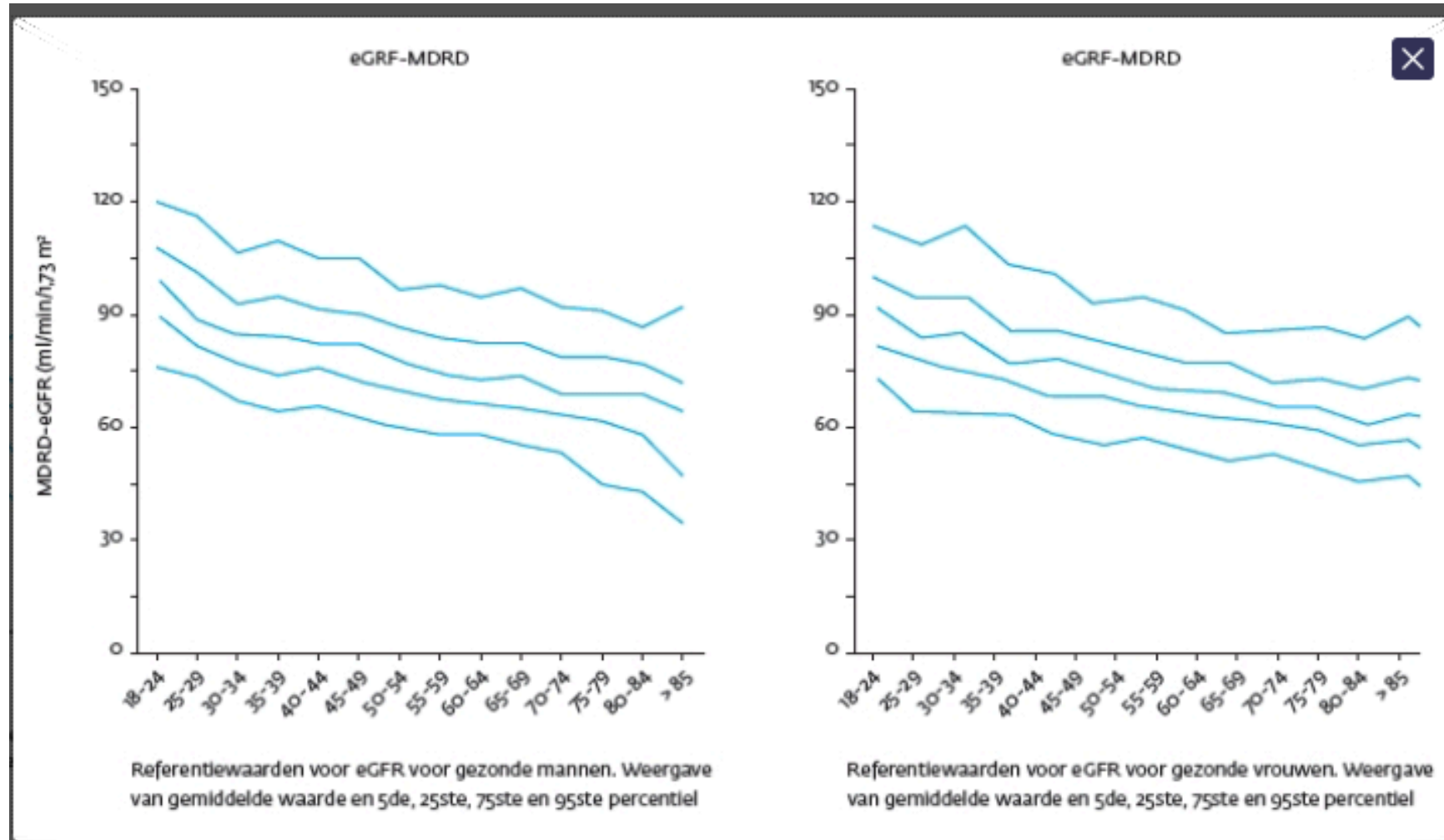
eGFR 50ml/MIN/1,73 m² (> 60 ml/MIN/1,73 m²)

Urine: proteinurie, E coli +

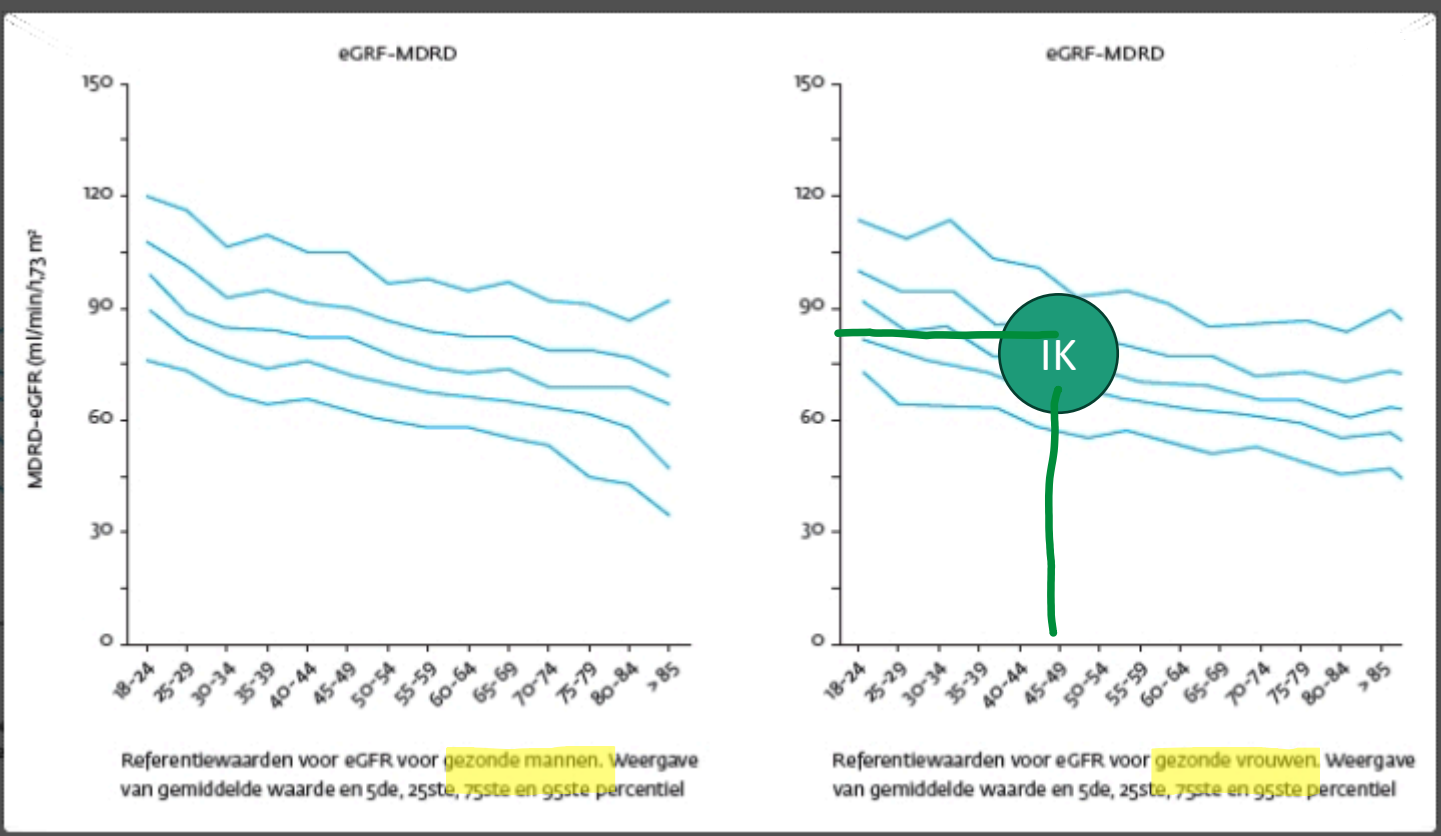
Urine collectie: 0 diuresis

Conclusie nierfunctie nihil

Meting nierfunctie: leeftijdsgebonden



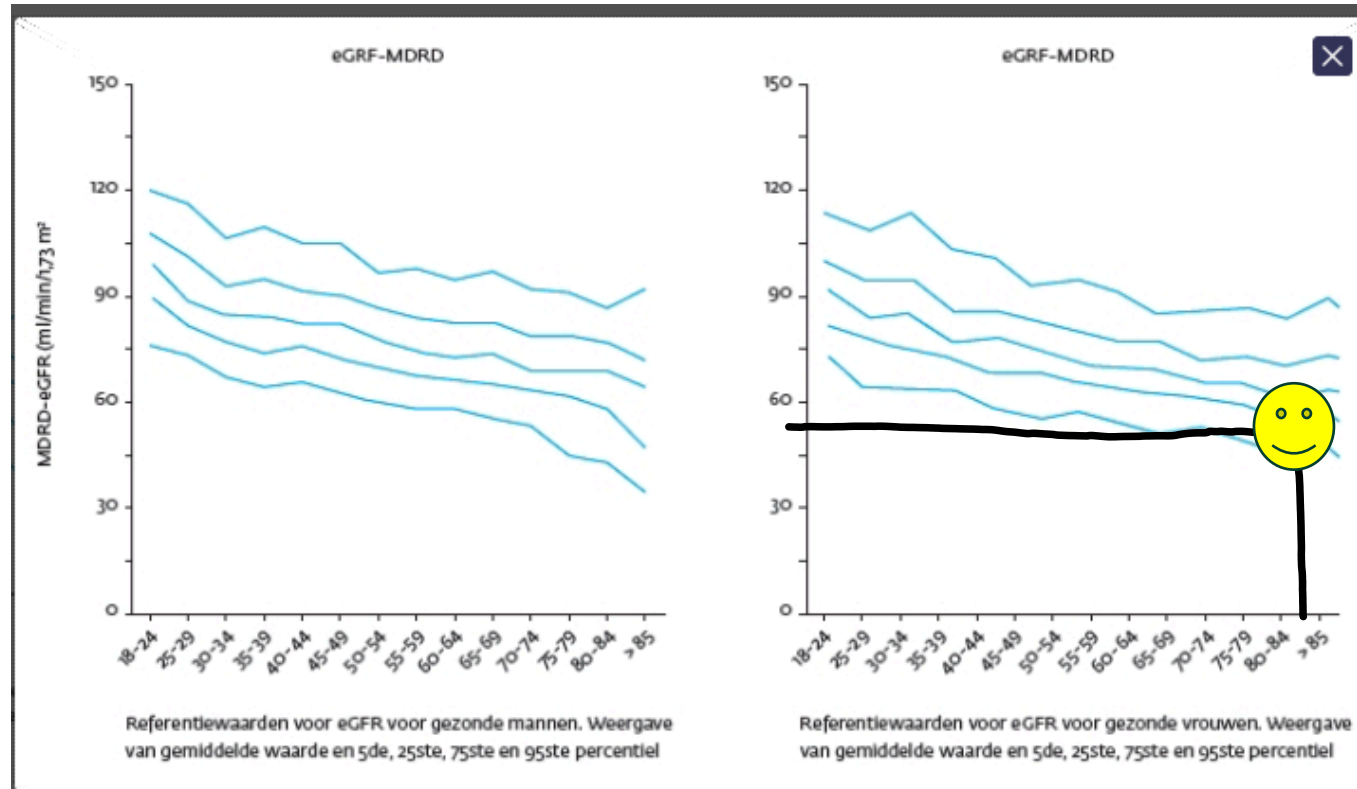
Meting nierfunctie: leeftijdsgebonden



Vrouw 46 j
eGFR **epi 88**
alb neg
nierfunctie normaal



Meting nierfunctie: leeftijdsgebonden



87 j

eGFR 48

alb neg

= nierfunctie normaal



Gemeten creatinine klaring met urine collectie

Creatinine clearance formula

$$\text{GFR} = \text{Clearance}_{\text{creatinine}} \text{ (mL/min)} = \frac{[\text{Creatinine}_{\text{urine}} \text{ (mg/dL)}] \times [24\text{-Hour Urine Volume (mL/day)}] / 1440 \text{ (min/day)}}{[\text{Creatinine}_{\text{blood}} \text{ (mg/dL)}]}$$

Zelf berekenen:

$$\frac{\text{urine creat}}{\text{serum creat}} \\ 14,4$$

- Nog zeker **nuttig** (ipv eGFR formules) bij:
 - Amputaties
 - zeer gespierde mensen
 - Paraplegien
 - zeer oud
 - Kinderen
 - Zwangeren
 - Spierziekten
- Als **e GFR <15** : wat **tubulaire secretie creatinine** ..
maw 'bij slechtere nierwerking dus **overschatting** nierwerking
- Bij **levende donatie** nuttig doch **'inuline'** klaring= nog correcter!

Meting proteinurie

proteinurie	24h debiet Eiwit/creat ratio	> 300mg/d > 0,2 (= 200mg/g creat)
microalbuminurie	24h debiet Alb/ creat ratio	> 30-300 mg/d > 0,02 (= 20 mg/g creat)

Niet nefrogene oorzaken albuminurie

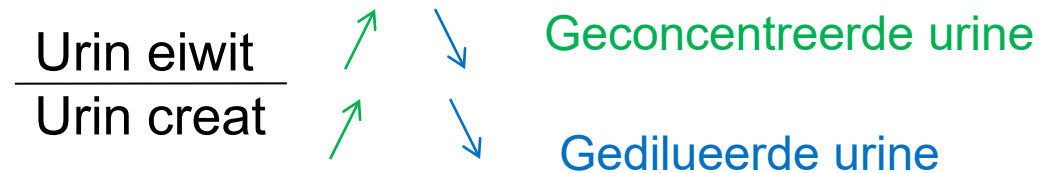
- *extreme Inspanning*
- *Infecties*
- *Koorts*
- *ontregelde diabetes*
- *extreme hematurie*



Meting proteinurie

proteinurie:

- eiwit/creat ratio op ochtendstaal
 - en niet: eiwit in mg/dl > cfr oiv hydratatietoestand
 - goede correlatie met 24h proteinurie / geen collectiefout



microalbuminurie: bij DM of HT en afwezigheid macroprot

- microalb/creat ratio

casus

1

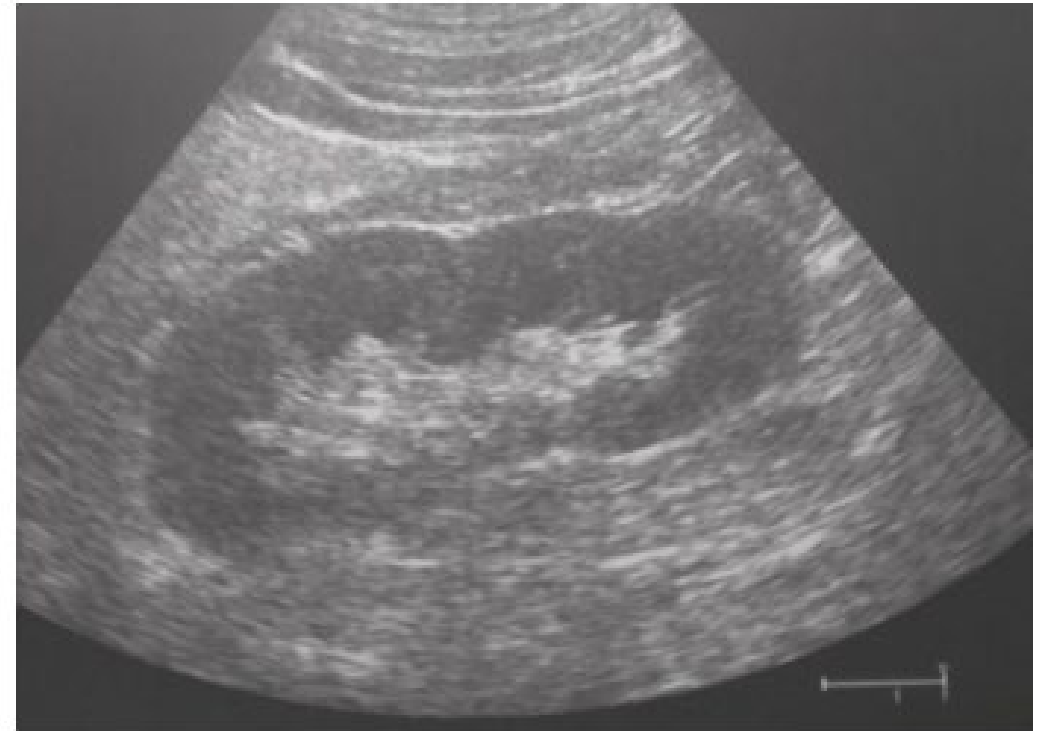
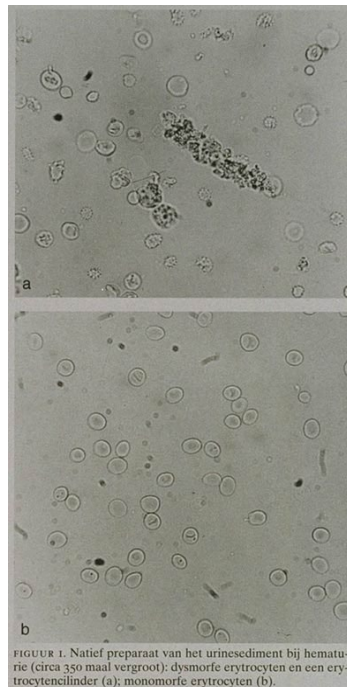
- 30 jaar, zeer gespierd, sprinter, wedstrijd gelopen
- Anamnese: geen klachten, laat toevallig dag na wedstrijd labo nemen op arbeidsgeneeskunde en is ongerust
- Medikatie: geen, supplementen voor spieren te versterken
- KO: gespierd, BD 12/80 1 m 80 100 kg
- Labo creatinine 1,5 mg/dl
eGFR: 58
- Urine: eiwit positief +++



Acuut versus chronisch?

Extra informatie nodig!

- Vergelijkende labo's voor en na
- Sediment: eiwit én bloed
- Echo nieren ev al
- Ev 24 uurse urinecollectie



Casus

1

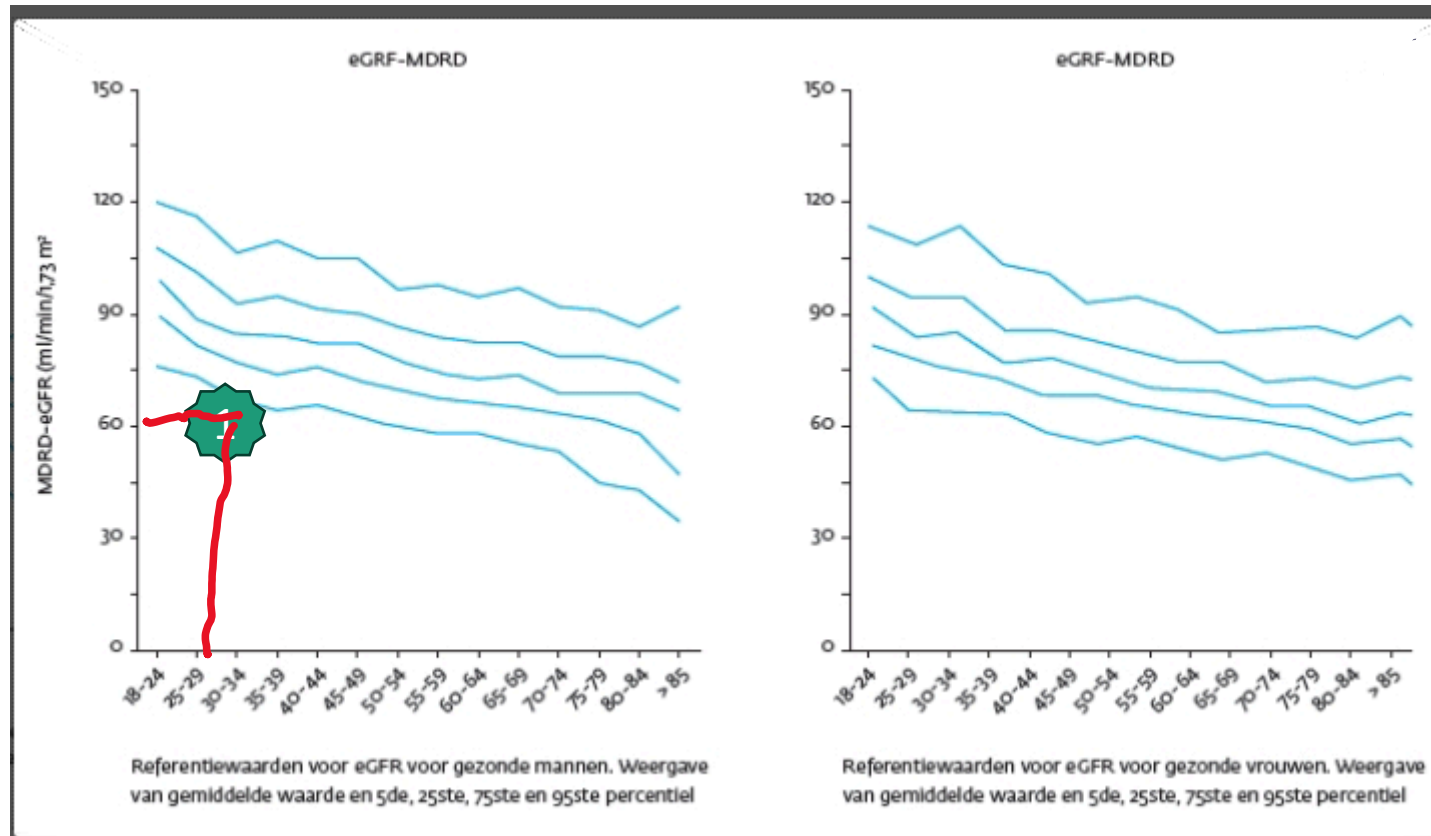
- Labo voorheen 2022 eGFR 56
- Labo na 1 week e GFR 59
- Urine na 1 week eiwitten 0 bloed 0
- Echo nieren: normaal
- 24 uurse collectie creat klaring 110 ml/MIN/1,73 m²

-> conclusie

Gerustelling, normale nierfunctie, eiwit door inspanning
afwijkende spiermassa plus Creatin inname!!



Meting nierfunctie: onderschatting met eGFR



Meting nierwerking: 'ras issue'

Nefrouupdate congres
9/2023

State of the Art – Estimating GFR

- Different formulas to estimate GFR have been developed
Cockrauft-Gault (Crea-Clearance), MDRD IV, CKD-EPI
- For the **same mGFR** serum **creatinine values in Black** people in the US are **higher** than in non-Black people; therefore a “**race-coefficient**” has been introduced to account for this difference
- 2021: **new** CKD EPIcr and CKD EPIcys **equations** were developed without a “race coefficient”
- Consequences:
 - New formulas **underestimate GFR in self-identified Black people** and **overestimate GFR in non-Black people**
 - Applying the new formulas **in Non-Black people decreases CKD prevalence** !!
 - Recommendation to measure cystatin C more frequently

Europa: doet niet mee..

formules blijven correctie voor zwarte ras gebruiken

Casus



- Nigeriaanse man, 44 jaar
- Anamnese : marathon goed uitgelopen, wat ziekjes gevoeld dagen ervoor infectie viraal maar niet 100 %, gaat naar huisarts voor controle en pijnstilling
- Antecedenten: als kind bij schoolonderzoek albumine in urine en hoge BD, nadien niet opgevolgd
- Medikatie: amlodipine 10, huisarts stelt voor eerst nieuw labo
- KO: normaal, erg gespierd, BD 15/90
- Labo: creat 1,6 mg/dl eGFR 51
- Urine: eiwit +++ en bloed +++



Acuut versus chronisch?

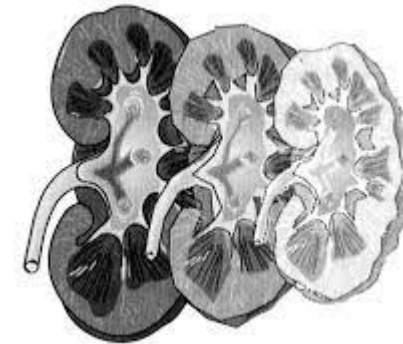
Casus



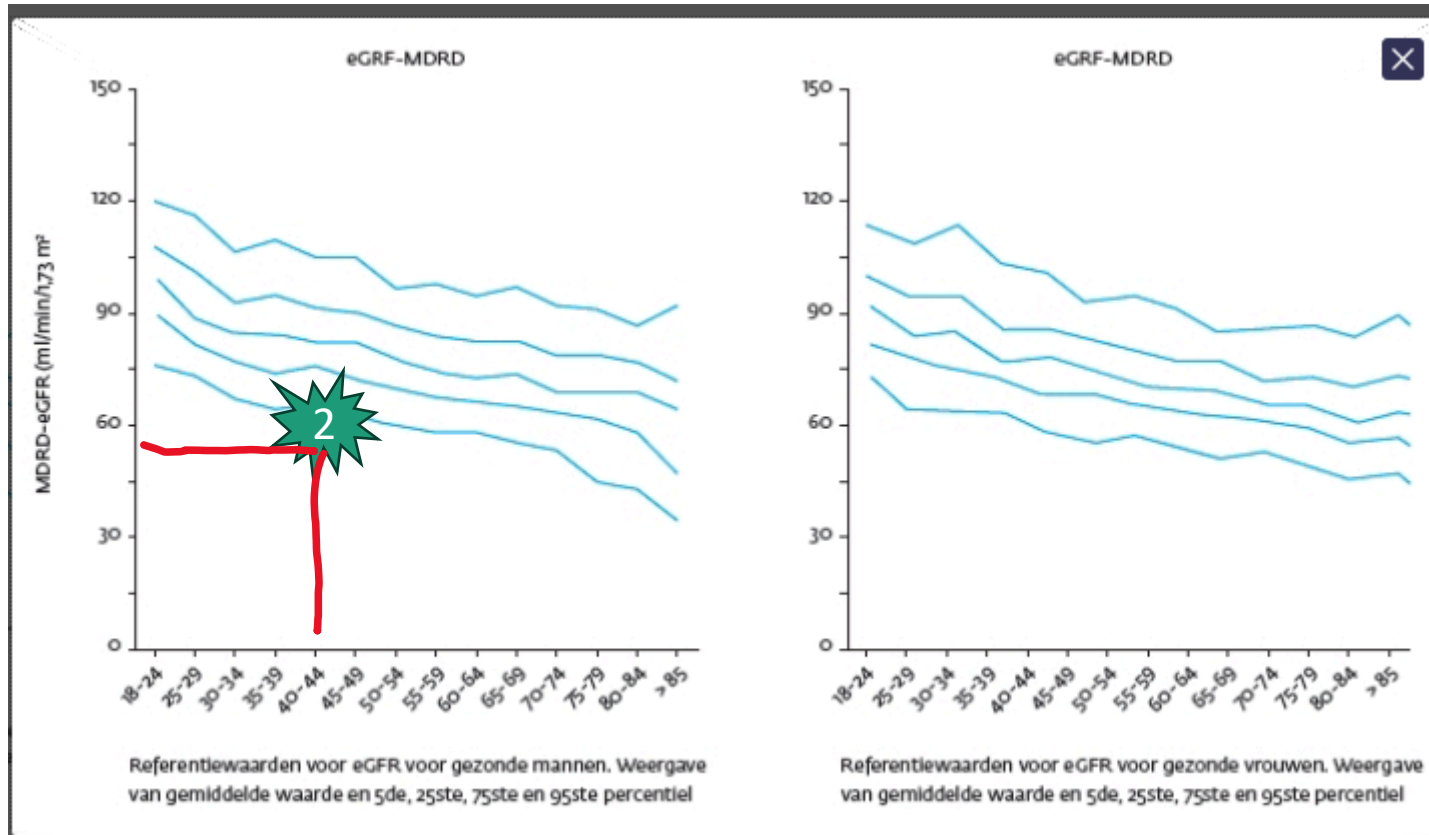
- Oude labo 2020 e GFR > 50
- Nieuw labo > 1 week e GFR > 51
- Urine sediment: microalb 750mg/ g creat
rode bloedcellen ++
kweek negatief
- Echo nieren: iets kleinere nieren al met wat uitgedunde cortex
- 24 uurse urine collectie: GFR 59
- Labo na 3 m e GFR 52
- Urine na 3 m proteinurie 1000 mg/ g creatinine

-> Conclusie :

- afwijkend vooraf bestaand 'chronisch' nierlijden
- Nierbiopsie: IGA nefropathie + hypertensie schade, switch coveram 5/5



Meting nierfunctie



casus

- man 75 jaar
- Goede conditie, op 45 j gestopt met roken
- Anamnese:
 - Hevige flankpijn na 1 uur , wedstrijd moeten stoppen, naar spoed gebracht door ambulance..
- Antecedenten: diabetes mellitus type II, jicht
- Medikatie: allopurinol, asaflow, bisoprolol, ramipril, forxiga
- Ko: nierslagpijn rechts, BD 10/70
- Labo: creat 2,5 mg/dl
eGFR 25
- Urine: bloed, microalbumine 2000 mg/ g creat



Acuut versus chronisch?

Casus



- Oude labo 2021
- Nieuw labo na 1w
- Urine sediment:
- Echo nieren:

e GFR 40
e GFR 41
albumine 1,2 g/g creatinine
bloed positief
gestuwde nier rechts
-> ct scan steen ureter

-> conclusie :

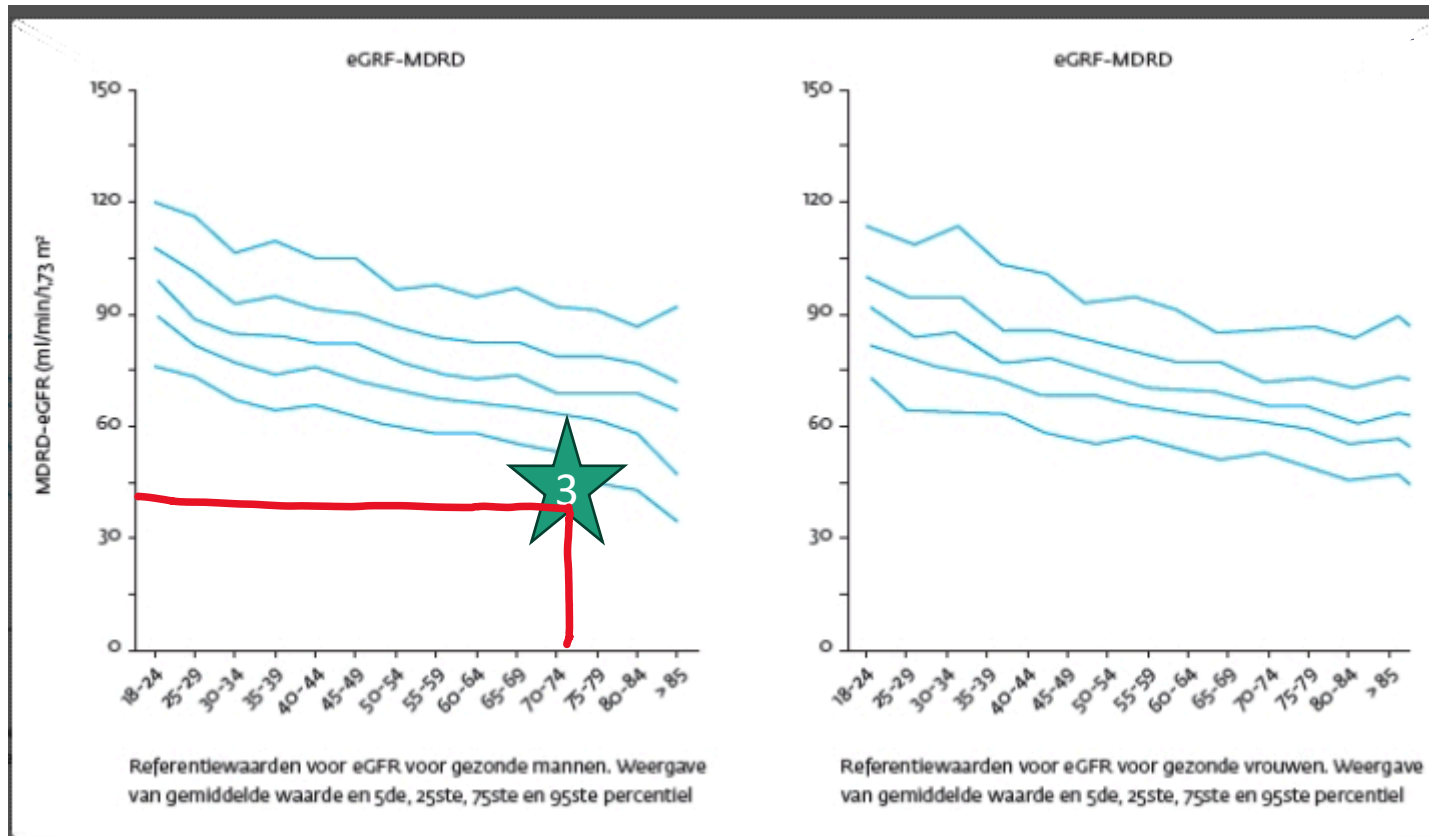
acut nierfalen > niersteen/ verwijzing uroloog

acut nierfalen > ACEI en Forxiga /stop tijdelijk

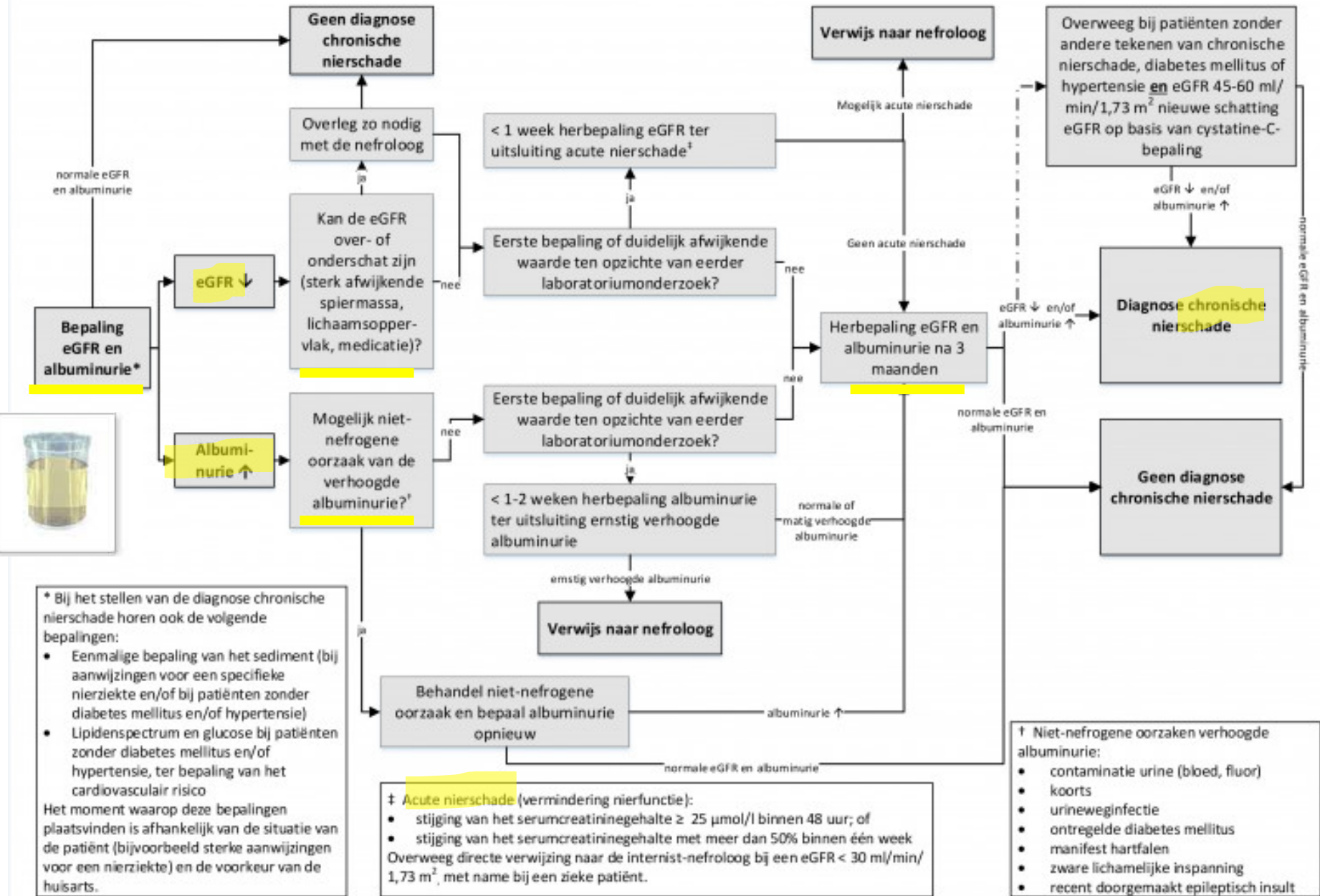
- Labo na 6 m e GFR 40
- Urine na 6 m bloed 0 , albumine 800 mg /g creat
- Voorafbestaand chronisch nierfalen: verwijzing nefroloog



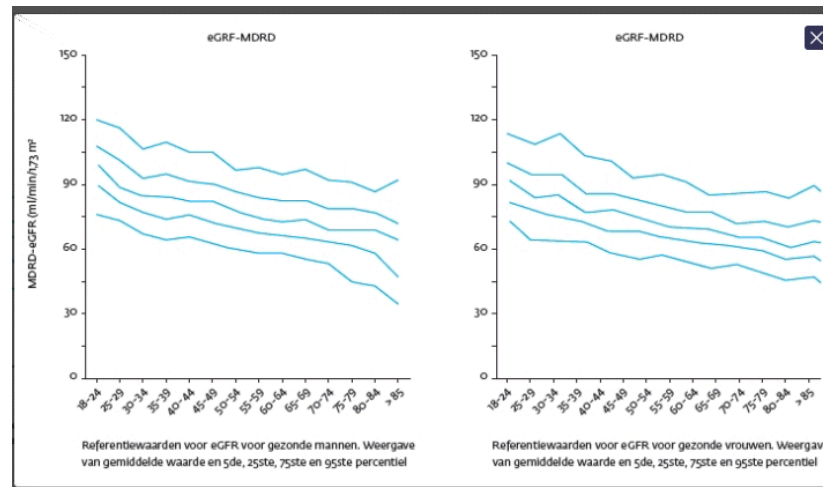
Meting nierfunctie



Mogelijke flowchart voor huisartsen



Take home



Altijd TWEE!!!
urine (eiwit/creat ratio) én eGFR

spiermassa/leeftijd belang bij interpretatie eGFR





danku voor uw luisteren

De nieuwe 2023 richtlijnen van de ESH voor de behandeling van arteriële hypertensie

Practopics-Plus online
27 oktober 2023

Dr. Manu Henckes
Nefrologie – multidisciplinaire hypertensie eenheid

Europese 'clinical practice' hypertensie-richtlijnen

2003 ESC-ESH Guidelines for the management of AHT

2007 ESC-ESH Guidelines for the management of AHT

2009 (revised)

ESH and ESC Guidelines

Management of AHT

ESC European Society of Cardiology
European Heart Journal (2018) 00, 1-98
doi:10.1093/eurheartj/ehy339
ESC/ESH GUIDELINES
2018 ESC Guidelines for the management of arterial hypertension
The Task Force for the management of arterial hypertension of the European Society of Cardiology

2023 ESH Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Hypertension

.../...

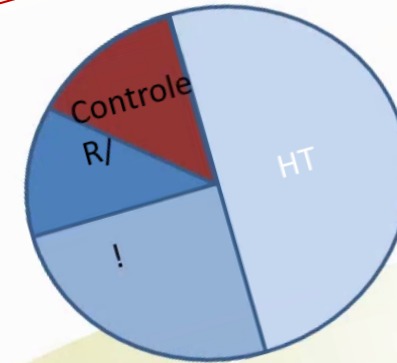
1735. Silva GFS, Fagundes IP, Teixeira BC, Chiavegatto Filho ADP. Machine Learning for Hypertension Prediction: a systematic review. *Current Hypertension Reports* 2022; 24:523–533.
1734. Niu M, Wang Y, Zhang L, Tu R, Liu X, Hou J, *et al.* Identifying the predictive effectiveness of a genetic risk score for incident hypertension using machine learning methods among populations in rural China. *Hypertens Res* 2021; 44:1483–1491.
1735. Oikonomou EK, Spatz ES, Suchard MA, Khera R. Individualising intensive systolic blood pressure reduction in hypertension using computational trial phenomaps and machine learning: a post-hoc analysis of randomised clinical trials. *Lancet Digit Health* 2022; 4:e796–e805.
1736. Koren G, Nordon G, Radinsky K, Shalev V. Machine learning of big data in gaining insight into successful treatment of hypertension. *Pharmacol Res Perspect* 2018; 6:e00396.

2023 ESH Guidelines

- Definitie van hypertensie
- Relatie met CVD
- Meting van CVD
- Vanaf wanneer behandelen
- Bloeddruk doelen
- Medicaties
- 'True resistant hypertensie'
- Hypertensie bij bepaalde klinische condities

Implementatie van de CPG

- 50% van de pt met HT weet het
- 50% van zij die het weten worden behandeld
- 50% van zij die behandeld worden zijn onder controle
- ...



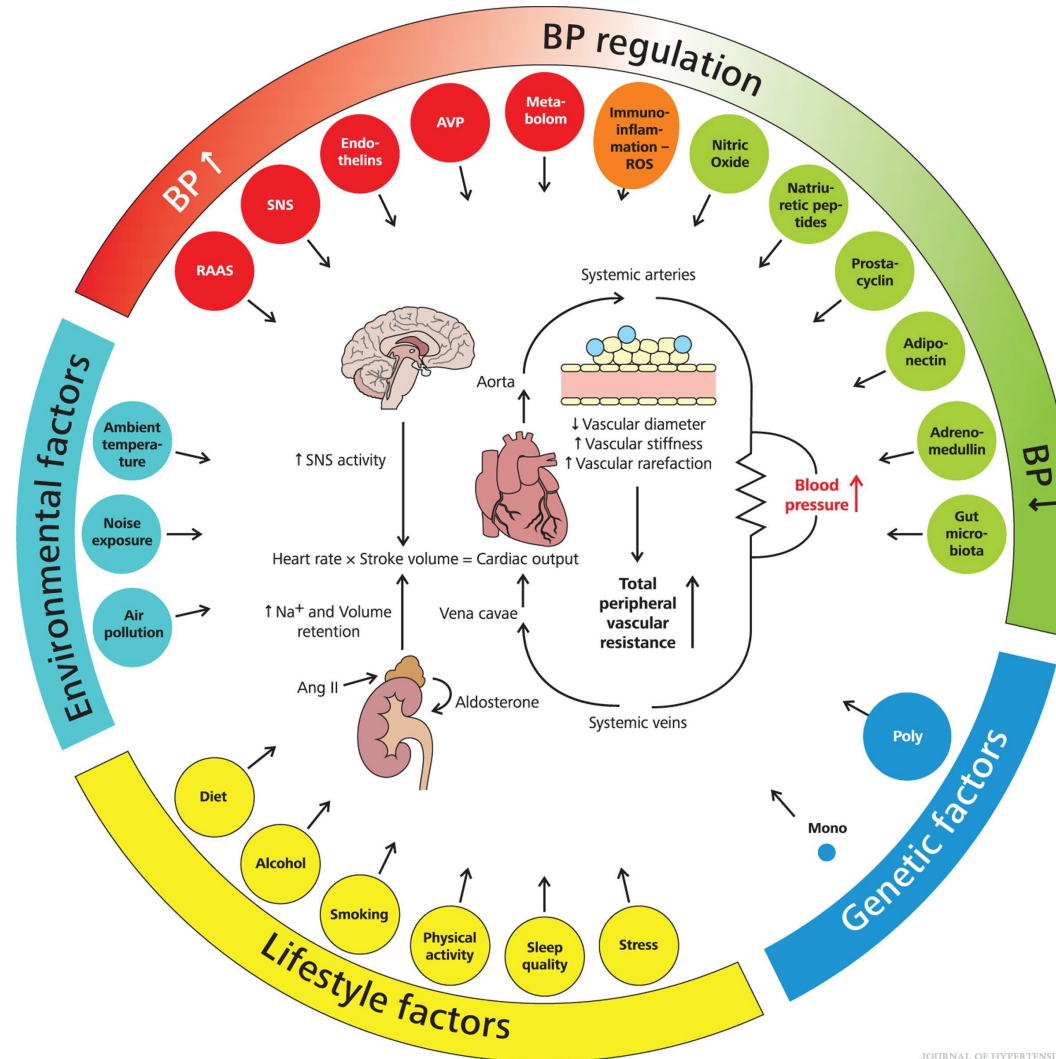
Behoud van controle op lange termijn

2023 ESH Guidelines

Gemodificeerde en gesimplifieerde criteria voor de gradatie van de evidentie voor de richtlijnen

Class of Recommendation		Level of Evidence		
	Definition	Definition	Interpretation	
I	Evidence or general agreement that a treatment/test/procedure is beneficial, useful or effective AND that potential benefits clearly outweigh potential risk	A	<ul style="list-style-type: none"> - RCT or meta-analysis of RCTs with CVD outcomes - Single trial enough if sufficient power and without important limitations^a 	Strong evidence. Evidence of high certainty. Unlikely that future studies will change the effect estimate substantially.
II	Conflicting evidence or opinion about the benefit, usefulness and effectiveness of a treatment/test/procedure OR uncertainty about benefit-risk balance	B	<ul style="list-style-type: none"> - RCT with surrogate measures (BP, HMOD) - Observational studies with CVD outcomes and no major limitations^a - Meta-analyses including the above study types 	Moderate evidence. Evidence with some uncertainty. Future studies may modify, at least the magnitude of, the effect estimate.
III	Evidence or general agreement that a treatment/test/procedure is not beneficial, useful or effective OR that potential risks outweigh the potential benefit	C	<ul style="list-style-type: none"> - Observational studies of surrogate measures - Any study type may be downgraded to level C due to limitations^a - Expert opinion (EO) 	Weak evidence. Evidence of low certainty. Future studies may change the effect estimate substantially.

2023 ESH Guidelines



Definitie van hypertensie

2023 ESH Guidelines

TABLE 3. Definitions and classification of office blood pressure levels (mmHg)^a




Idem

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

^aThe blood pressure (BP) category is defined by the highest level of BP, whether systolic or diastolic. Isolated systolic hypertension should be graded 1, 2, or 3 according to systolic BP values in the ranges indicated.

Totaal cardiovasculair risico

Hypertension disease staging	Other risk factors, HMOD, CVD or CKD	BP (mmHg) grading			
		High-normal SBP 130–139 DBP 85–89	Grade 1 SBP 140–159 DBP 90–99	Grade 2 SBP 160–179 DBP 100–109	Grade 3 SBP ≥ 180 DBP ≥ 110
Stage 1	No other risk factors ^a	Low risk	Low risk	Moderate risk	High risk
	1 or 2 risk factors	Low risk	Moderate risk	Moderate to high risk	High risk
	≥3 risk factors	Low to moderate risk	Moderate to high risk	High risk	High risk
Stage 2	HMOD, CKD grade 3, or diabetes mellitus	Moderate to high risk	High risk	High risk	Very high risk
Stage 3	Established CVD or CKD grade ≥4	Very high risk	Very high risk	Very high risk	Very high risk

	<50 years	60–69 years	≥70 years
	<2.5%	<5%	<7.5%
	2.5 to <7.5%	5 to <10%	7.5 to <15%
	≥7.5%	≥10%	≥15%

Complementary risk estimation in Stage 1 with SCORE2/SCORE2-OP



<https://www.escardio.org/Education/Practice-Tools/CVD-prevention-toolbox/SCORE-Risk-Charts>

European Society of Cardiology > Education > Practice Tools & Support > CVD Prevention Toolbox

Practice Tools & Support

CVD Prevention Toolbox

EACVI Imaging Toolboxes

ACVC Clinical Decision-Making Toolkit

Pocket Guidelines App

Guidelines Summary Cards

ACS Trials Comparison Tool

Atrial Fibrillation Patient Website

Heart Failure Patient Website

ESC Science In Your Language

ESC Prevention of CVD Programme

SCORE2 and SCORE2-OP

Risk assessment models to estimate the 10-year risk of cardiovascular disease in Europe.

SCORE2 and SCORE2-OP

Two new algorithms, SCORE2 and SCORE2-OP (older persons), were published in June 2021:

SCORE2

SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe

SCORE2 working group and ESC cardiovascular risk collaboration

European Heart Journal, ehab309, <https://doi.org/10.1093/eurheartj/ehab309>

SCORE2-OP

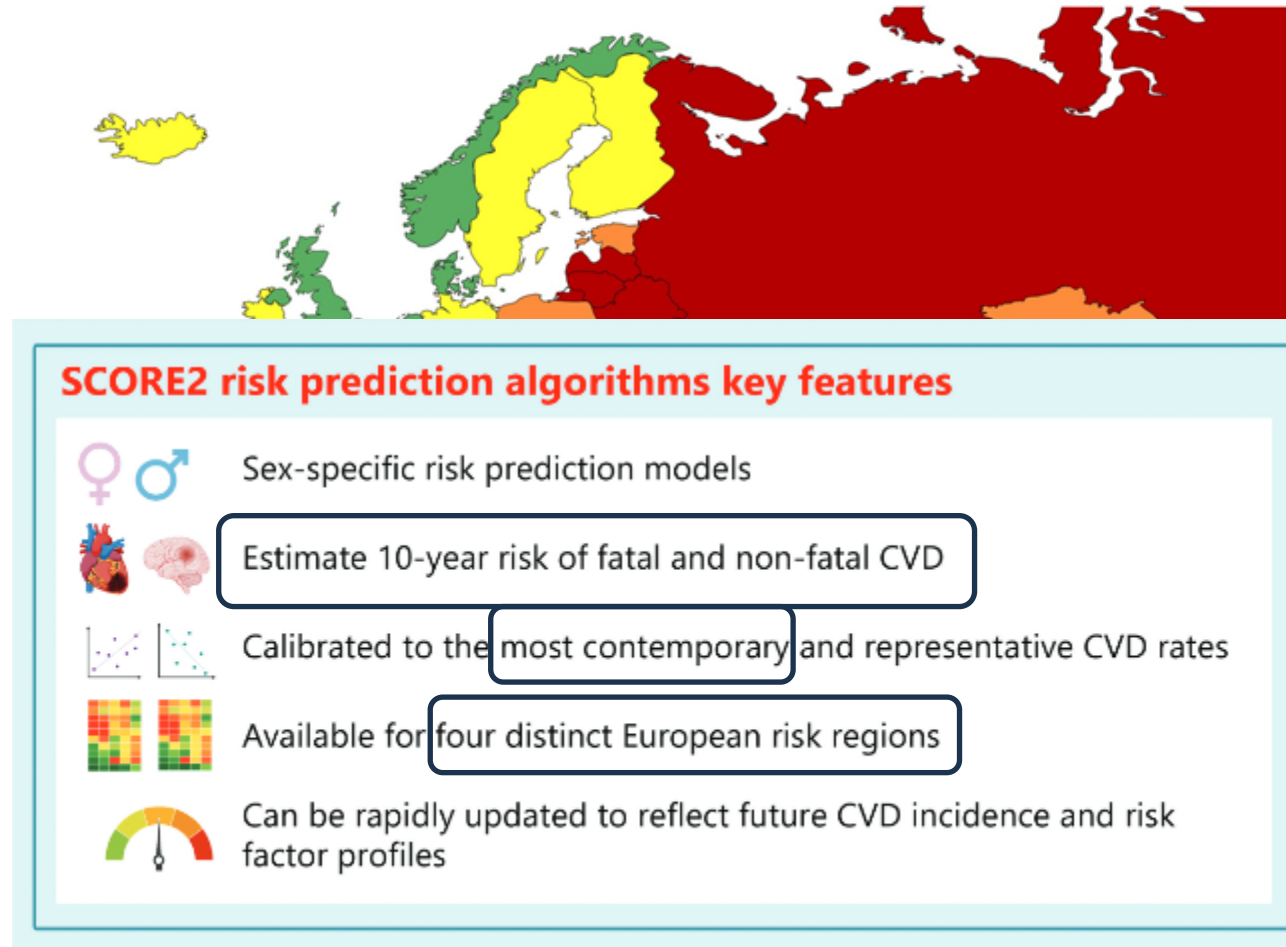
SCORE2-OP risk prediction algorithms: estimating incident cardiovascular event risk in older persons in four geographical risk regions

SCORE2-OP working group and ESC cardiovascular risk collaboration

European Heart Journal, ehab312, <https://doi.org/10.1093/eurheartj/ehab312>

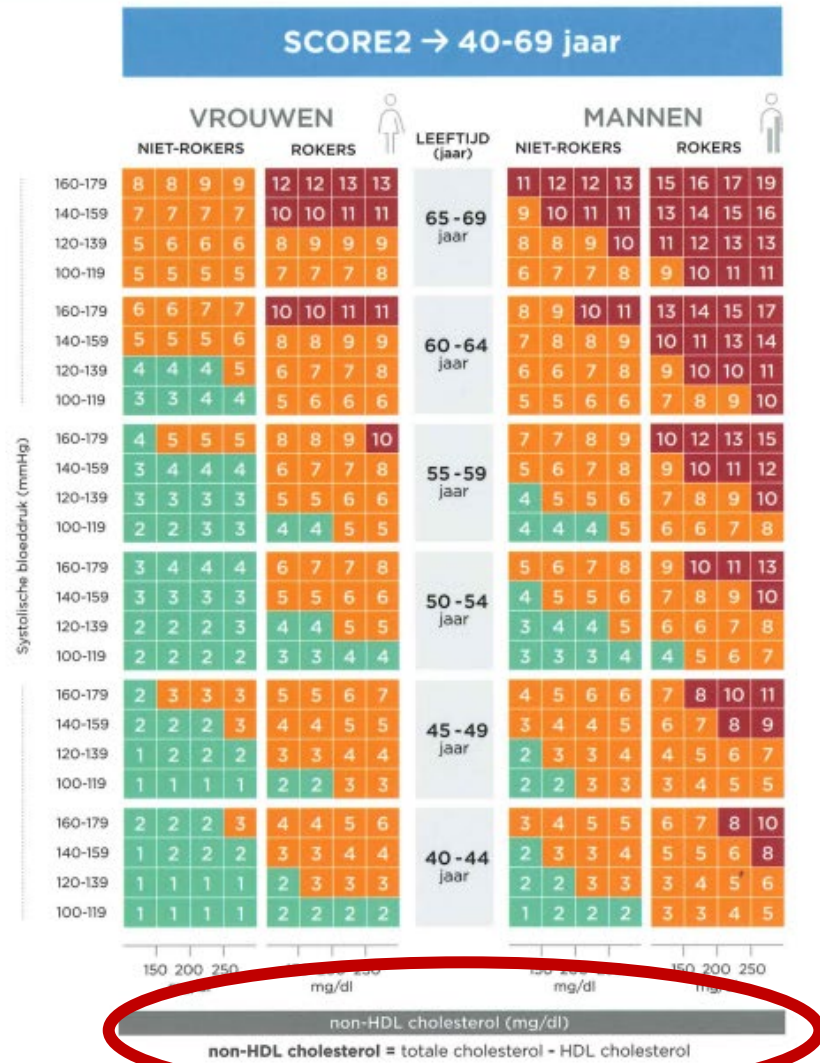
SCORE2 and SCORE2-OP interactive version

Figure 2 Risk regions based on standardised cardiovascular disease mortality rates. Countries were grouped into four ...



10 jaar risico op cardiovasculaire evenens (fataal en niet-fataal)¹

bij schijnbaar gezonde personen zonder vastgestelde arteriële hypertensie, diabetes, chronische nierinsufficiëntie of familiale hypercholesterolemie



Naast de klassieke CVZ-risicofactoren, zoals leeftijd, geslacht en roken, zijn opgenomen, kunnen ook andere risico-factoren of individuele informatie het berekende risico wijzigen. De beoordeling van potentiële risico-modificatoren lijkt relevant indien het risico van de betrokkene dicht bij een beslissingsdrempel ligt.¹



1. Visseren, Frank L J et al. "2021 ESC Guidelines on cardiovascular disease prevention in clinical practice." European heart journal vol. 42,34 (2021): 3227-3337.
2. Mach François et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS), European Heart Journal, vol. 41, 1, 1 (2020): 111-188.

Diagnose van hypertensie

- Office **SBD** > 140
of **DBD** > 90 mmHg
- Op 2 of 3 verschillende Rpl
gemeten
- Én rekening houden met
thuis-BD meting en/of
24u ABPM

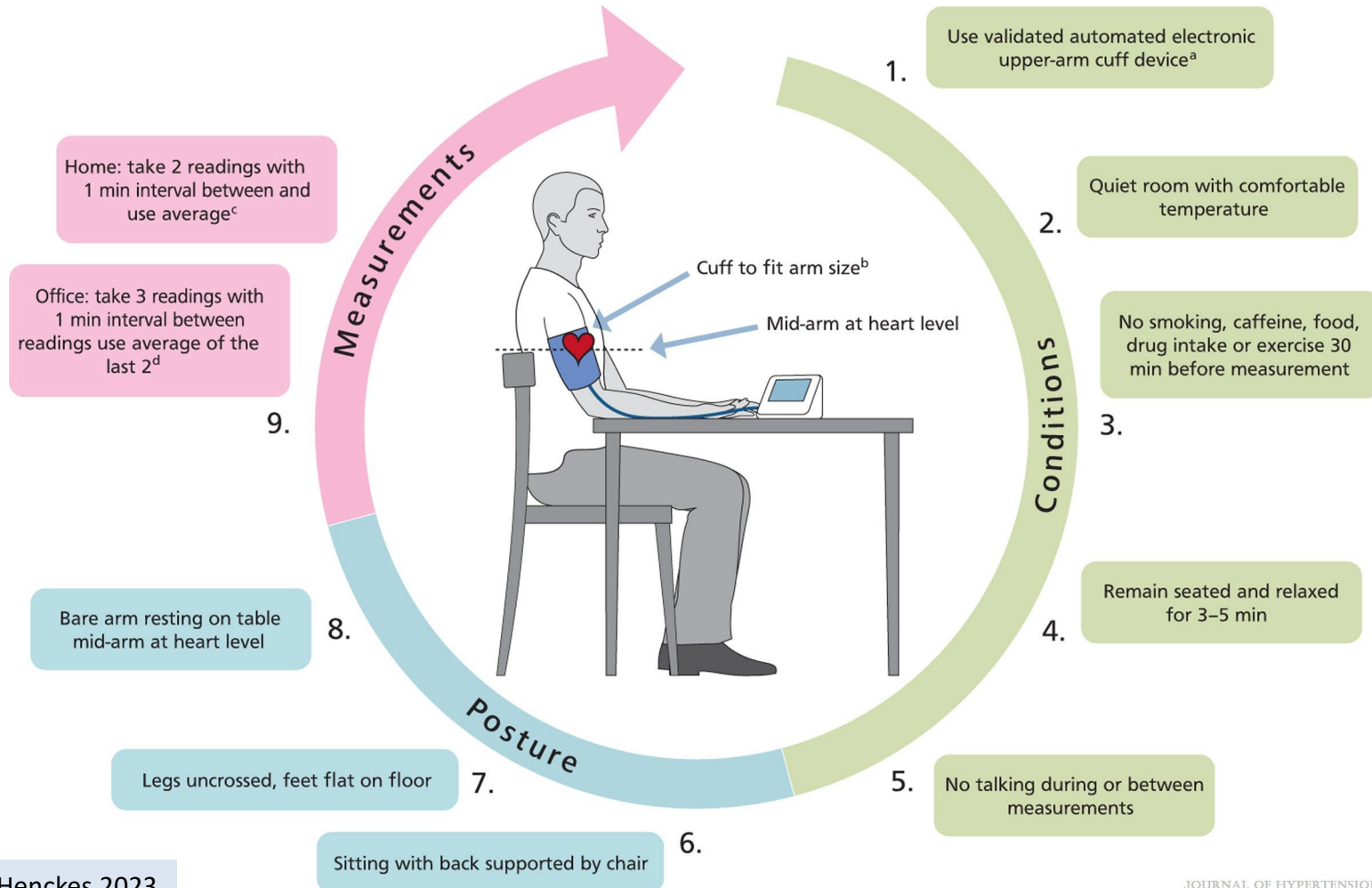
OBPM



HBPM
ABPM

2023 ESH Guidelines

Meting van de bloeddruk



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Meting van de bloeddruk



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Recommendations and statements	CoR	LoE
Office BP is recommended for diagnosis of hypertension, because it is the one method by which hypertension-related risk, benefits of antihypertensive treatment, and treatment-related BP thresholds and goals are based.	I	A
Office BP measurements should be performed in <u>standardized</u> conditions, using a standard measurement protocol. <u>Triplicate</u> measurements should be taken and <u>the average of the last two</u> should be referred to as the representative value.	I	C
It is recommended to diagnose hypertension <u>during at least 2</u> <u>separate office visits (within 4 weeks)</u> unless office BP indicates <u>grade 3 hypertension ($\geq 180/110$ mmHg)</u> or <u>patients presents with hypertension related symptoms or there is evidence of HMOD or CVD.</u>	I	C
At the <u>first office visit, BP should be measured in both arms.</u> A consistent between-arm SBP difference $>15-20$ mmHg suggests atheromatous disease and is associated with increased CV risk. All subsequent measurements should be made on the arm with the highest BP readings.	I	C
Out-of-office BP is a source of multiple BP-related information before and during treatment. It is therefore recommended to <u>obtain additional information on BP values by ABPM or HBPM</u> or both if available.	I	C



Meting van de bloeddruk

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HBPM

- Veel literatuur over!
- Predictief voor CVD
- D/ Gemaskeerde hypertensie
D/ Witte jas hypertensie
- **Belangrijk in de opvolging van de BD**
- Gevalideerd, automatisch (oscill.), bovenarm
- Cut off waarde : 135/85 mmHg (ipv 140/90 OBPM)
- Laatste 7 dagen voor Rpl
2 x 2 metingen per dag met 1' tussen
(eerste 's morgens)



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ABPM

- Veel literatuur over!
- D/ Gemaskeerde hypertensie
D/ Witte jas hypertensie

TABLE 4. Definitions of hypertension according to the correspondence of home and ambulatory BP values with office BP

Method	SBP (mmHg)		DBP (mmHg)
Office BP ^a	≥140	and/or	≥90
Ambulatory BP			
Awake mean	≥135	and/or	≥85
Asleep mean	≥120	and/or	≥70
24 h mean	≥130	and/or	≥80
Home BP mean	≥135	and/or	≥85

^aRefers to standard office BP measurements (not unattended measurements). Data compare the averages from cohorts of untreated and treated individuals. Given the low correlation between office and out-of-office BP values, individuals can have considerable discrepancies from the averages.

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- Anamnestisch
- Klinisch
- Labo
 - Hb, Htc
 - Gluc, HbA1c
 - Chol, TG, HDL, LDL, non-HDL
 - Natrium, Kalium
 - Urinezuur
 - Creatinine
 - Calcium
 - URINESTAAL (ochtend) voor multi-dip stick en albumine/creatinine Evt. + Sediment

BD?
Co-morbiditeit?
HMOD? CVD?
Secundaire HT?



Evaluatie van hypertensie

- Hypertensie- gemedieerde orgaanschade :
 - ECG
 - Creatinine en urine alb/creat
 - Echo nieren (evt doppler)
 - Enkel/arm index
 - Evt :
 - Echocardio
 - PWV
 - Echo halsvaten
 - Coronaire calciumscore (CT)
 - Oogfundus
 - MMSE, CT/MRI hersenen

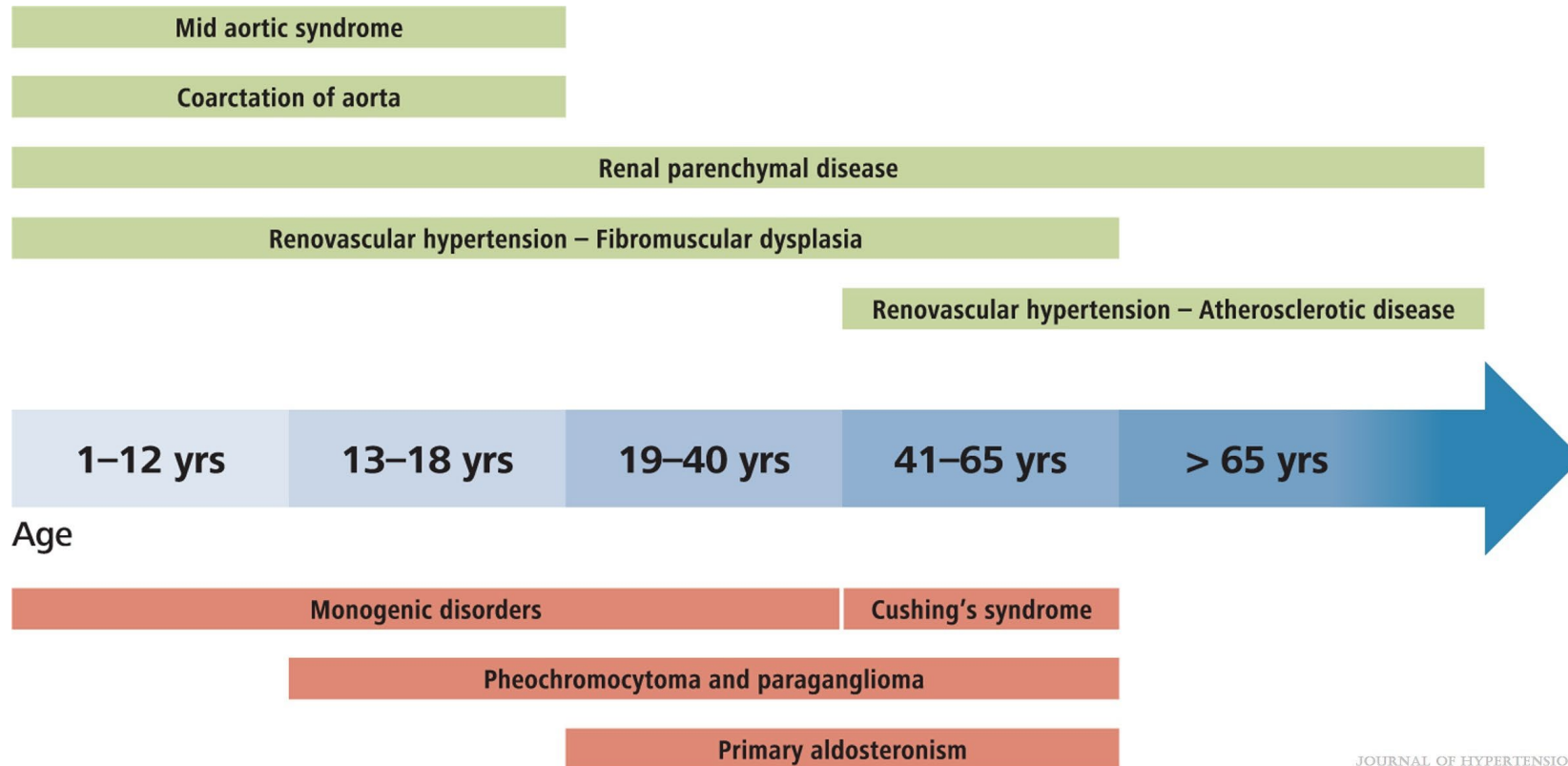
Doorverwijzing naar hypertensie-specialist

TABLE 12. When to refer a hypertensive patient to a specialist or to hospital

- Patients in whom secondary hypertension is suspected
- Young patients (<40 years) with grade 2 or 3 hypertension in whom secondary hypertension should be excluded
- Patients with sudden onset or aggravation of hypertension when BP was previously normal
- Patients with treatment-resistant hypertension
- Need of more detailed assessment of HMOD, which might influence treatment decision
- Requirement of more in-depth specialist evaluation from the referring doctor
- Hypertensive emergencies (inpatient care will usually be needed)

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Secundaire hypertensie



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Secundaire hypertensie

(A) Atherosclerotic renovascular disease

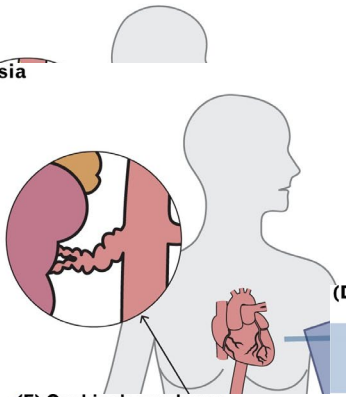
Prevalence:
6–14%^a
(B) Fibromuscular Dysplasia

Suggestive symptoms, signs and findings
Early-onset/severe hypertension
Migraine
Pulsatile tinnitus

1st choice screening test^b
Renal artery duplex ultrasound;
otherwise angio-CT or angio-MR

Treatment
Antihypertensive treatment
Angioplasty without stenting^{c,d}

Follow-up
• Whole body angio-CT or angio-MR at diagnosis^e
• Indefinite follow-up



Cardiovascular phenotype

Cardiovascular phenotype (C) Primary aldosteronism

Prevalence:
6–20%^a
Suggestive symptoms:
24h ABPM – early morning hypertension

(D) Pheochromocytoma and paraganglioma

Prevalence:
<1%^a

(E) Cushing's syndrome

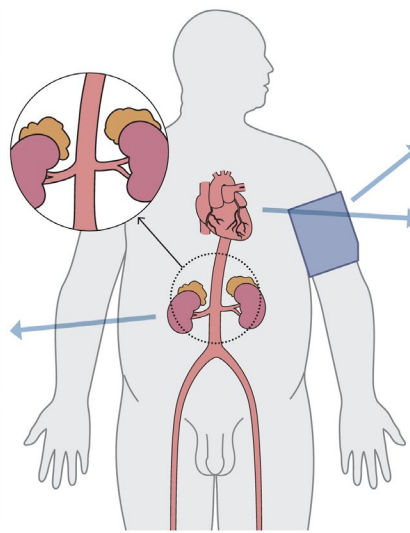
Prevalence: 2–5%^a

Suggestive symptoms and signs
Resistant hypertension
Easy bruising, facial plethora, 'moon' face, skin thinning
Proximal myopathy
Weight gain with centripetal distribution of body fat
Diabetes mellitus

1st choice screening test^b
Overnight 1 mg dexamethasone suppression test
24-h urinary free cortisol
Late-night salivary cortisol

Further work-up
Morning plasma ACTH
ACTH stimulation by CRH or desmopressin
CT

Treatment
Medical – normalization of cortisol levels
Surgical – first line treatment for Cushing's disease, ectopic Cushing's syndrome and ACTH-independent hypercortisolism



Cardiovascular phenotype

24h ABPM – frequent non-dipping
Short-term BP variability

• LVH
• Decreased systolic function
• Decreased diastolic function

Increased CV Risk and mortality



Cardiovascular phenotype

24 ABPM – true resistant hypertension, frequent non-dipping

• LVH
• Decreased diastolic function
• Myocardial fibrosis (MRI)

Increased CV Risk and mortality

Cardiovascular phenotype

24h ABPM – frequent non-dipping

• LVH
• Decreased systolic function
• Myocardial fibrosis (MRI)


Increased CV Risk and mortality

Secundaire hypertensie

TABLE 14. Rare genetic causes of secondary hypertension [343]

Condition	Phenotype	Mechanism and Treatment
Liddle syndrome	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC	Increased renal tubular ENaC activity; responds to treatment with amiloride
Apparent mineralocorticoid excess	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC	Decreased 11 β -hydroxysteroid dehydrogenase isoenzyme 2; responds to spironolactone
Gordon syndrome	Hyperkalemia, metabolic acidosis, low PRA or PRC, low/normal PAC	Overactivity of the sodium-chloride cotransporter; responds to thiazides
Geller syndrome	Pregnancy-exacerbated hypertension, low PRA or PRC, low PAC	Agonist effect of progesterone on the mineralocorticoid receptor (which is constitutively active); responds to amiloride, spironolactone activates instead of blocking the receptor
Glucocorticoid-remediable aldosteronism (familial hyperaldosteronism type 1)	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Chimeric <i>CYP11B1/CYP11B2</i> gene; responds to glucocorticoids
Familial hyperaldosteronism type 2	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Increased activity of CLCN2 chloride channel; responds to steroidal MRA
Familial hyperaldosteronism type 3	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Loss of selectivity of KCNJ5 potassium channel; patients who do not respond to steroidal MRA require bilateral adrenalectomy
Familial hyperaldosteronism type 4	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Increased activity of CACNA1H calcium channel; responds to steroidal MRA
PASNA syndrome (primary aldosteronism, seizures and neurological abnormalities)	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC; neurological defects coexists	Increased activity of CACNA1D calcium channel; responds to steroidal MRA and CCB
11beta-hydroxylase deficiency	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC, virilization of female individuals	Reduced activity of 11 β -hydroxylase with increase of DOC and androgens; responds to glucocorticoids
17alpha-hydroxylase deficiency	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC, pseudohermaphroditism in male individuals	Reduced activity of 17 α -hydroxylase with increase of DOC and reduction of androgens; responds to glucocorticoids
Autosomal dominant hypertension with brachydactyly [342]	Brachydactyly type E (BDE), short stature, severe hypertension (salt-independent, age-dependent), high risk of death from stroke before age 50	PDE3A mutations upregulated the cAMP-hydrolytic activity that results in lower cAMP levels in vascular smooth muscle cells

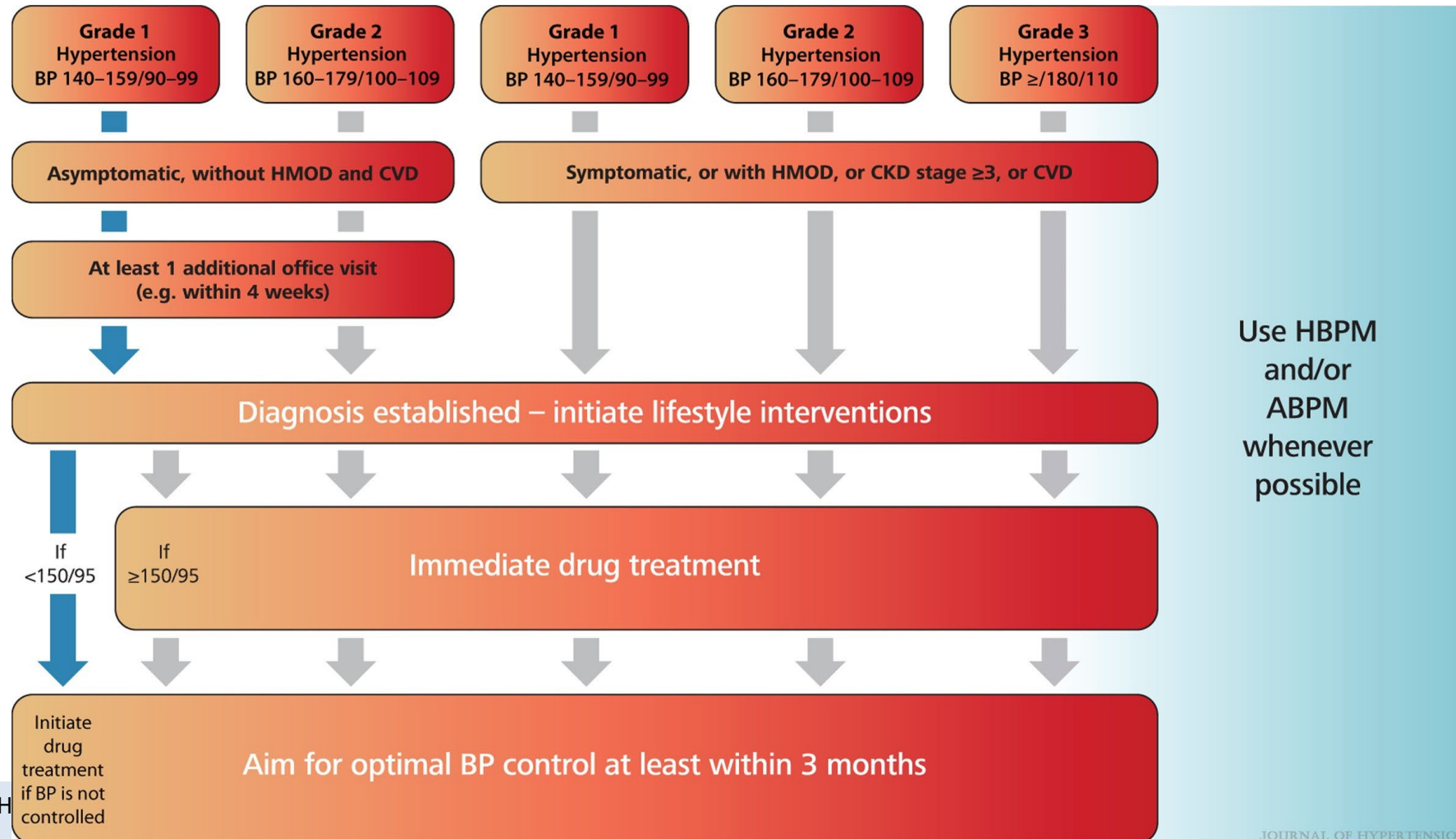
Levensstijl – interventies

Zo overgewicht: vermageren	I	A
Voorkeur voor groenten, fruit, bonen, noten, zaden, plantaardige olieën, vis en gevogelde. 'Gezond dieet meer vegetarisch en minder dierlijk'	I	B
Zoutbeperking (NaCl < 5 g/d)	I	A
Zo hoge zoutinname, vervangen door KCl substituten	I	B
 Meer kalium consumptie (tenzij gevorderde CKD)	I	B
Dagelijkse fysieke activiteit. Liefst > 150-300 min aerobe oefeningen, 'vigoureuze intensiteit'. Minder sedentaire tijd. Dynamische weerstandsoefeningen, 2-3x/week	I	B

Levensstijl – interventies

Zo alcoholconsumptie >3 cons/w: advies om te stoppen (tot bijna niets) => zal BD doen dalen	I	A
Vermijd excessief (binge) drinken = vooral risico voor hemorragisch CVA en plotse dood	III	B
Alcohol wordt best niet aangeraden als CVD preventie.	III	B
Rookstop	I	B
Stress vermindering via gecontroleerde ademhalings-oefeningen, mindfulness gebaseerde oefeningen en meditatie kunnen overwogen worden	II	C

Wanneer starten met behandeling?



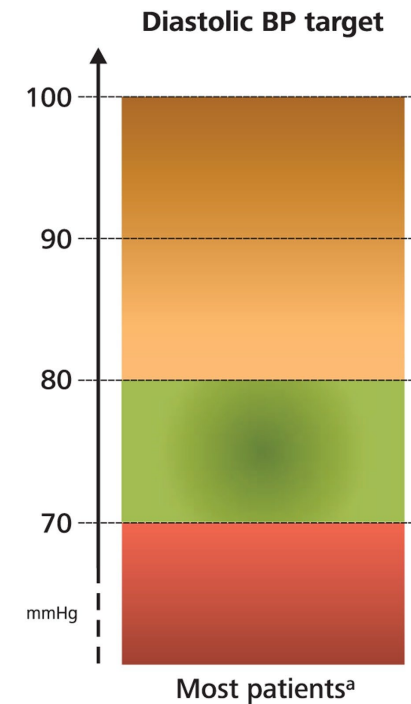
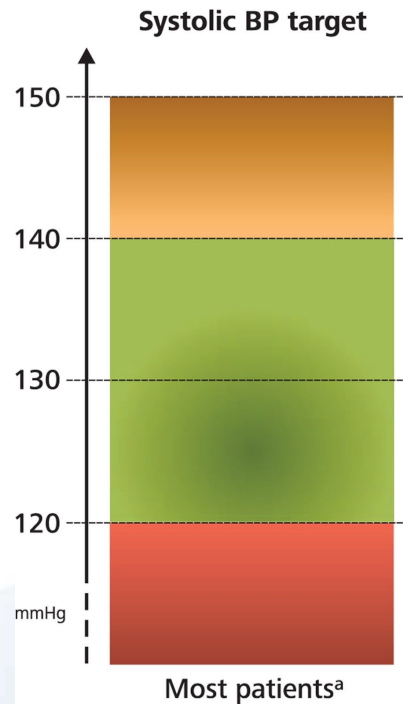
M H

Wanneer starten met behandeling?

- **Algemeen : zo BD > 140/90 mmHg (OBPM)**
- *Zo > 80 j : zo SBD > 160 mmHg (OBPM)*
- *Op individuele basis bij > 80 j:
zo SBD 140 – 159 mmHg*
- *Bij fragiele patiënten : individualiseren!*
- **Bij volwassenen met CVD (vooral CAD)
starten zo BD > 130/80 mmHg**

Bloeddruk-doelen bij behandeling

< 140/80 mmHg



Bloeddruk-doelen bij behandeling

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na ESH 2023						
	Age	18 - 64		>65	>80	
Office SBP	CFS ?	1 tot 4				> 5 (fragiel)
SBD < 160	mmHg					i d n u e d a e i l r v i ! i s
SBD < 140-150	mmHg			ISH	> 80 j	
< 140/80	mmHg	18 - 64 j		65 - 79 j	zo goed verdragen	
< 130/80	mmHg	bij CVD (CAD)		zo goed verdragen		
< 130/80		CKD en jong - A/C > 0,3 - hoog CVrisico				
< 120/70	mmHg	Niet				
DBD < 70	mmHg	Zo DBD<70 en SBD boven doel : voorzichtig verder dalen.				
Standing BP ?						!
Home BP ?		5 mmHg SBD/DBD lager				
24 u BP ?		overdag 5 mmHg SBD/DBD lager				

Medicamenteuze behandeling



Prescribing patterns:

- Start with dual combination therapy in most patients
- Uptitrate to maximum well tolerated doses and to triple therapy if needed
- **Once daily (preferred in the morning)**
- **Add further drugs if needed**
- **Preferred use of SPCs at any step**



Additional drug classes

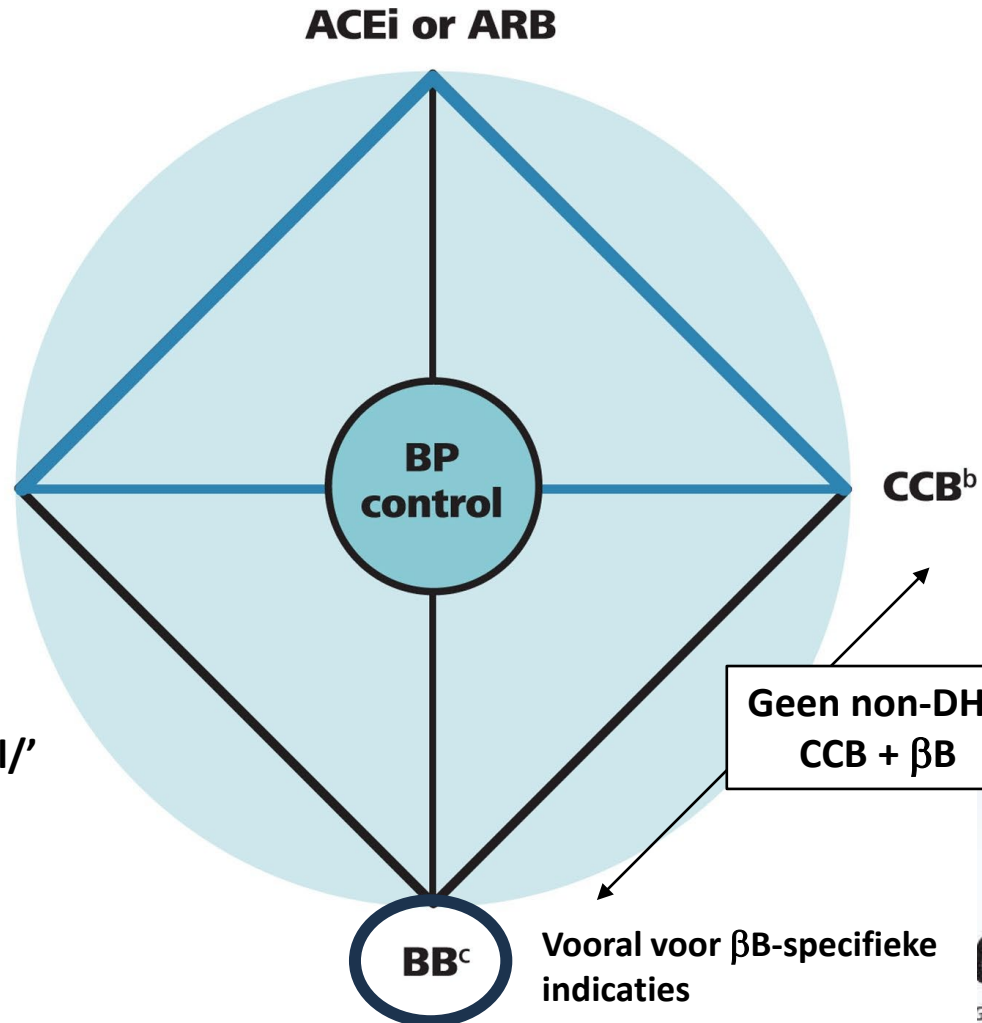
General antihypertensive therapy:

- Steroidal MRA
- Loop Diuretic → **Bij eGFR < 30 ml/'**
- Alpha-1 Blocker
- Centrally acting agent
- Vasodilator

Special comorbidities:

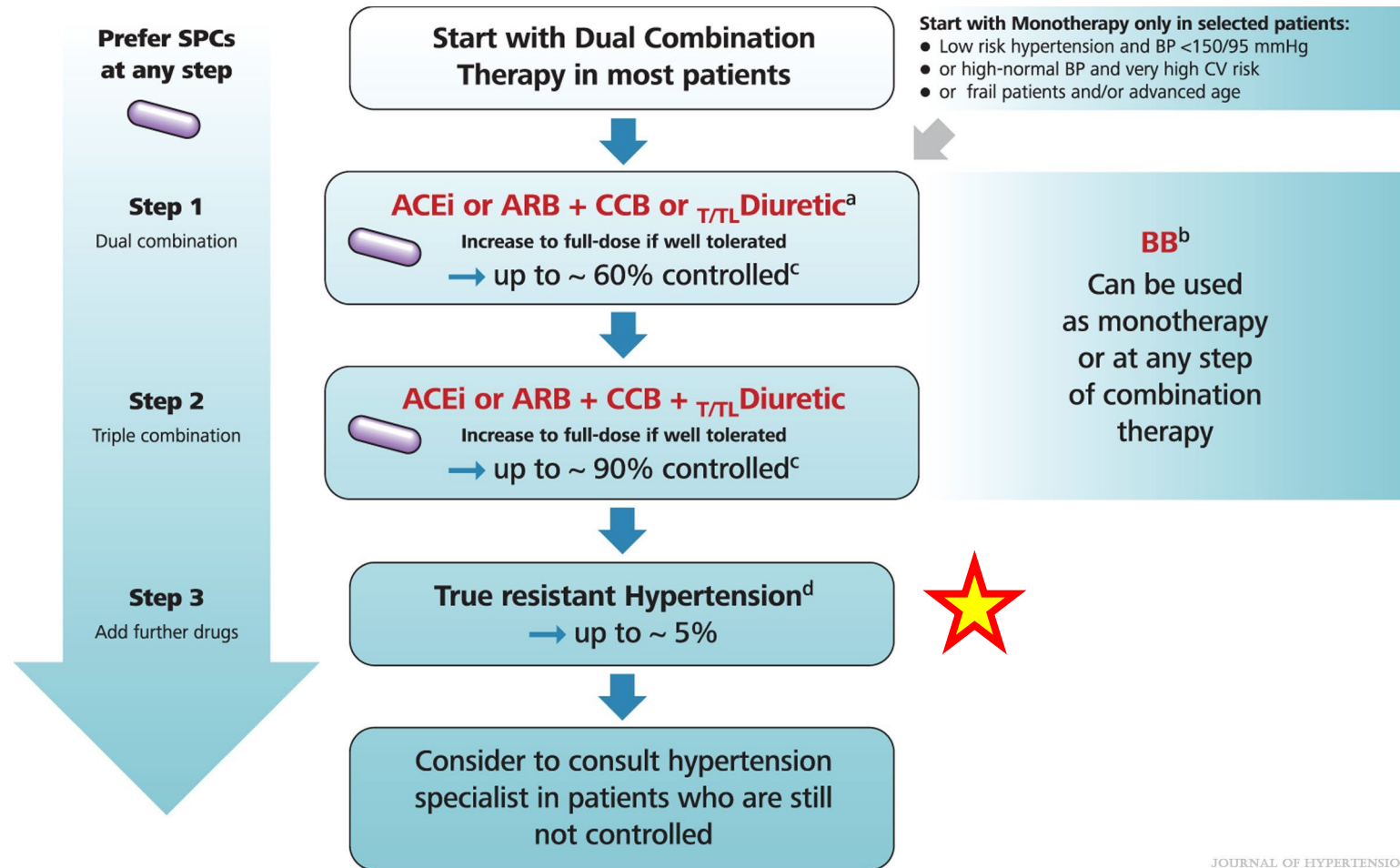
- ARNi
- SGLT2i
- Non-Steroidal MRA

Entresto
Forxiga/Jardiance
Kerendia



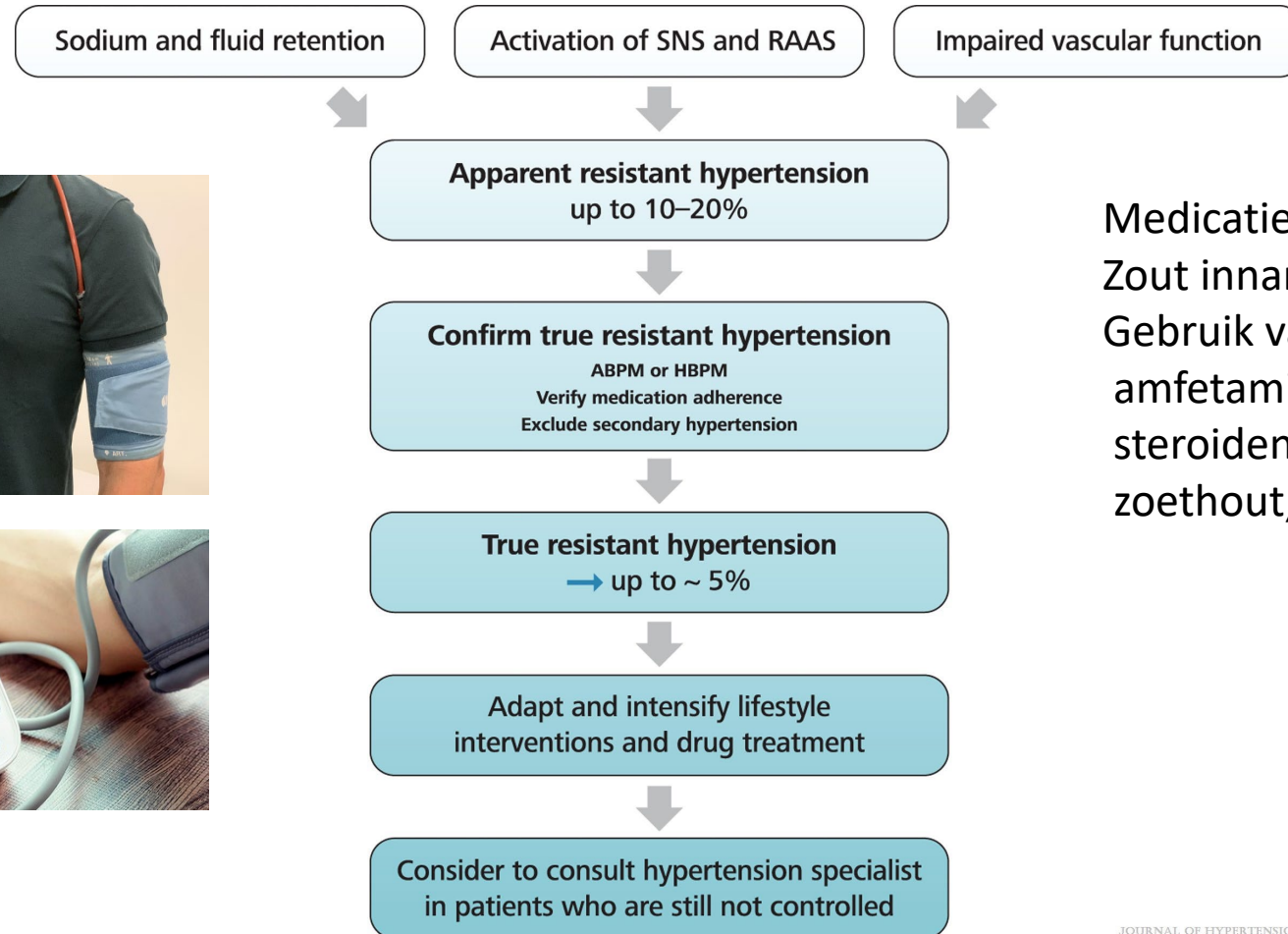
Medicamenteuze behandeling

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'True resistant hypertension'

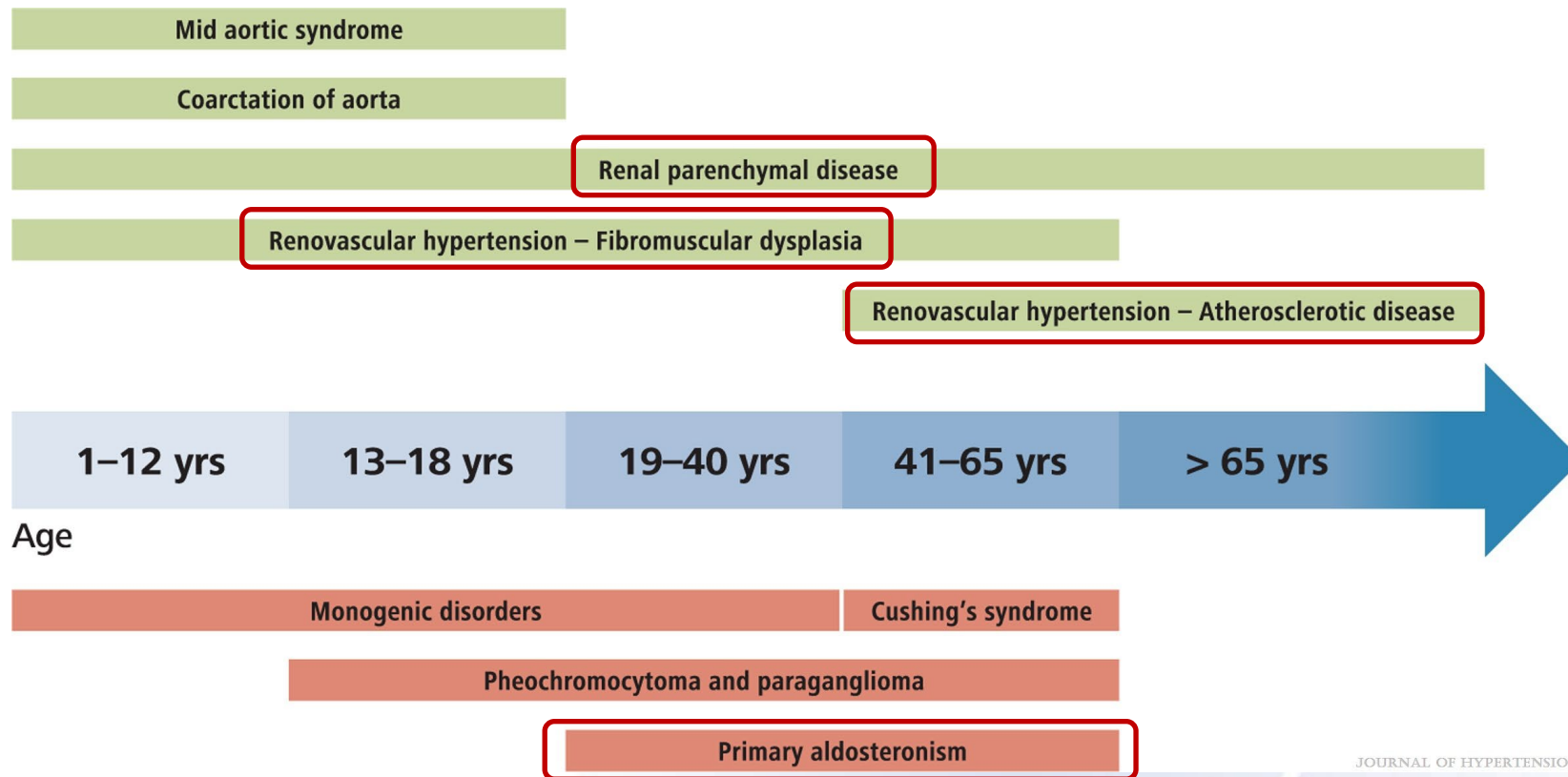


Medicatie inname?
Zout inname?
Gebruik van NSAï,
amfetamine's, drugs,
steroiden, B-mimetica,
zoethout, ...

TABLE 20. Medications and other substances that may increase BP

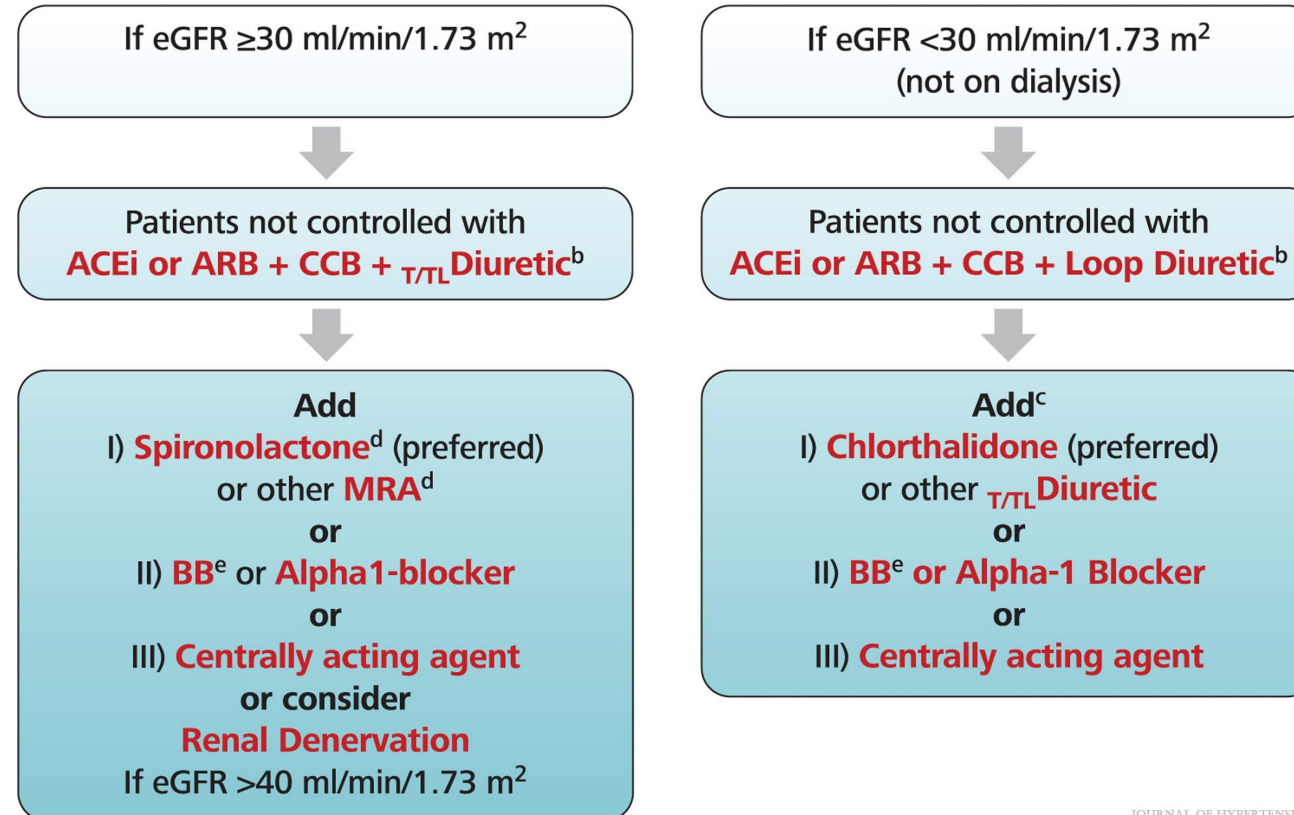
Medication/substance	Proposed mechanism	Comments
NSAIDs	Inhibition of COX-1 and 2 decreasing PG I2 and E2 synthesis with subsequent reduction in urinary Na excretion and an increased systemic vascular resistance.	Mild, dose-dependent increase in BP. Increased risk with age, preexisting hypertension, salt-sensitive patients, patients with renovascular hypertension.
Paracetamol (acetaminophen)	Presumably via inhibition of cyclooxygenases and reduced production of prostaglandins.	Increased relative risk of 1.34 of hypertension with almost daily paracetamol use.
Estrogens and progestins	Increased renin synthesis (by estrogens) leading to RAS activation and subsequent Na ⁺ and water retention.	Mild, sustained increase in BP (6/3 mmHg increase with high doses of estrogen >50 µg of estrogen and 1–4 µg progestin) but can be severe, common in premenopausal women, cause hypertension in 5% of women.
Glucocorticoids	Enhanced Na ⁺ reabsorption and fluid retention via stimulation of mineralocorticoid receptors. Increased systemic vascular resistance due to upregulation of AT1 receptors on vascular smooth muscle cells.	Dose-dependent, low doses have less effect on BP, more common in older patients, or with a family history of primary hypertension.
Calcineurin inhibitors	Reduced NO production, ET-1 overproduction, systemic and renal vasoconstriction, renal Na ⁺ retention.	Dose-dependent, mild-to-moderate increase in BP. Severe hypertension has been reported. Increased risk with preexisting hypertension, elevated creatinine levels and maintenance therapy with corticosteroids. See Section 20.8.2
Antidepressants SNRIs	Increased noradrenaline release causing adrenergic activation and increased SNS activity.	Dose-dependent, mild (2/1 mmHg) increase in BP.
Nasal decongestants	Vasoconstriction due to stimulation of alpha-1 receptors on vascular smooth muscles.	Dose-dependent, sustained increase in BP.
Erythropoietin-stimulating agents	Increased thromboxane, reduced prostacyclin levels and activation of the local RAS. Increased ET-1 production, decreased NO synthesis with subsequent vasoconstriction.	Dose-dependent, mild increase in BP, increased risk with preexisting hypertension, or when the initial hematocrit level is low. See Section 20.8.2
Stimulants		
- Modafinil - Amphetamines - Methylphenidate	Block noradrenaline or dopamine reuptake. Promote release of catecholamines	
VEGF inhibitors	Decreased NO production via VEGFR-2 antagonism and stimulation of ET-1 receptors promoting vasoconstriction.	A class effect. The incidence of hypertension is dose-related, risk is increased by preexisting hypertension, old age and overweight. See Section 20.8.2.
Substances of abuse		
- MDMA - PCP - Methamphetamine	Increased release and inhibited reuptake of monoamine neurotransmitters with subsequent SNS activation. Increased CNS catecholamine release with decreased neuronal uptake.	Cocaine induces both acute and chronic increases in BP.
- Cocaine	Cocaine induces acute sympathomimetic effects and chronic HMOD, i.e. an increase in arterial wall stiffness.	Alcohol causes a dose-dependent, sustained increase in BP.
- Alcohol	Alcohol increases SNS and RAS activity.	
Herbal products		
- Licorice - Ephedra - St. John's wort - Yohimbine - Ginseng (high doses) - Ma huang	Chronic excessive liquorice use mimics hyperaldosteronism by stimulating the mineralocorticoid receptor and inhibiting cortisol metabolism. Ephedra activates the alpha-1 receptor increasing SNS activity.	Licorice: Dose-dependent, sustained increase in BP characterized by hypokalemia, metabolic alkalosis and suppressed plasma renin activity and aldosterone levels Yohimbine causes acute, dose-dependent increase in BP.
Diet pills		
- Sibutramine - Phenylpropanolamine	Increased levels of norepinephrine with subsequent activation of noradrenergic transmission	Mild increase in BP.

??? Secundaire hypertensie ???

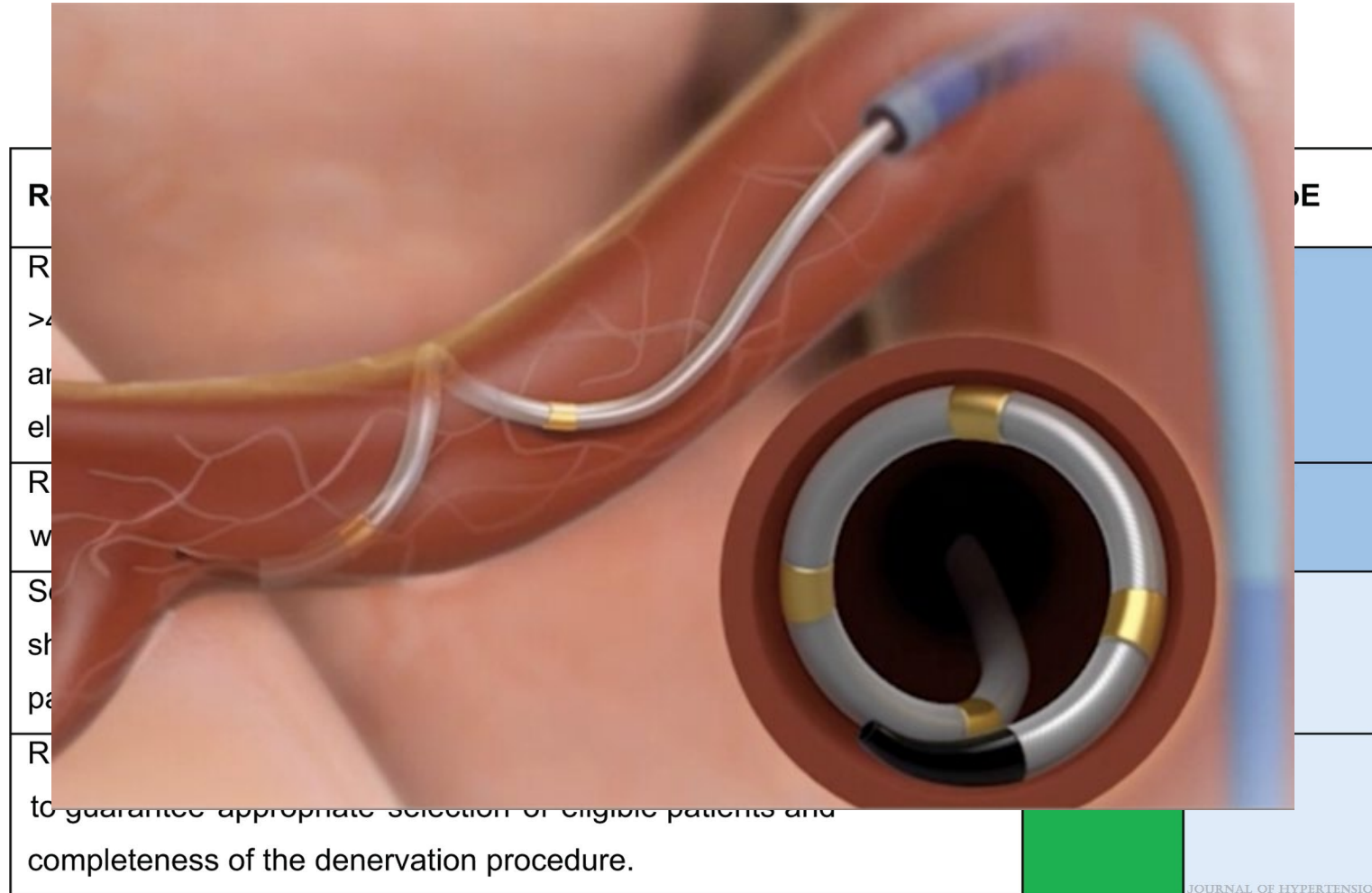


Behandeling van 'true resistant hypertension'

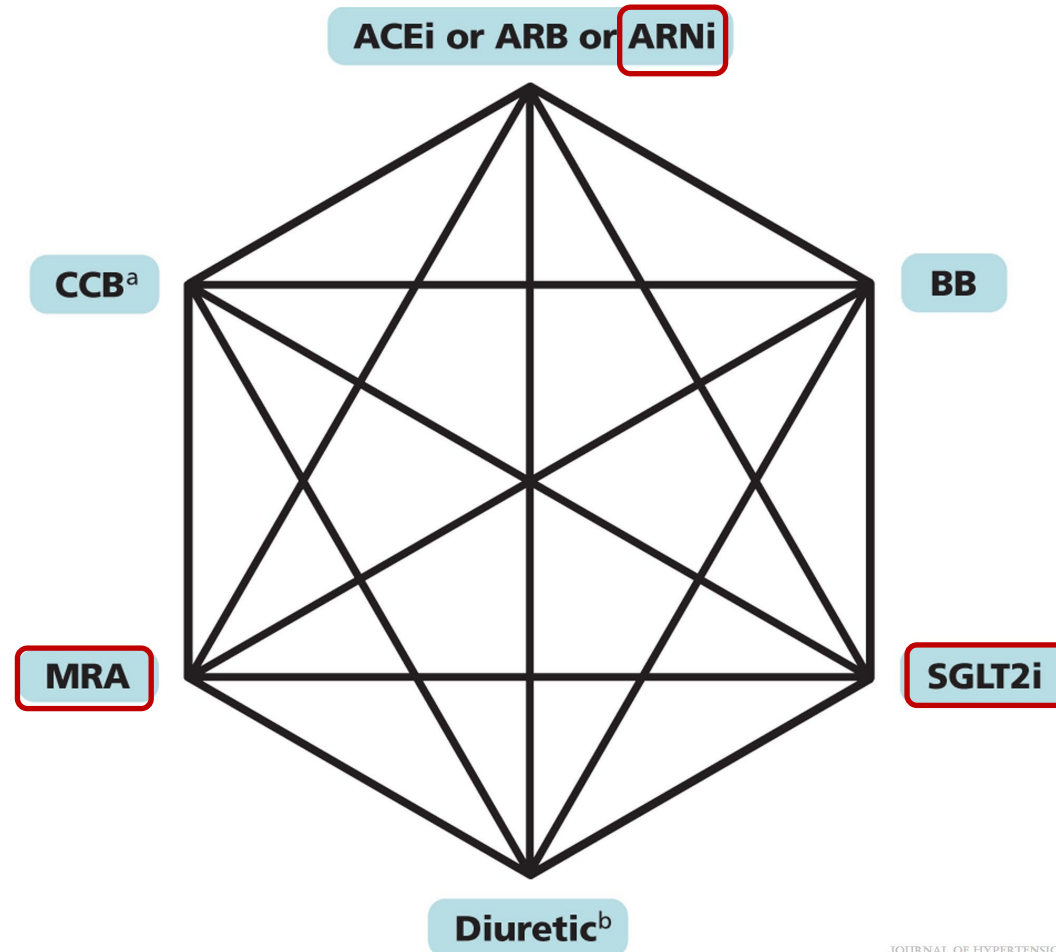
BP-lowering therapy in true resistant hypertension^a



Renale denervatie



Medicatie in HT en hartfalen (HFrEF)



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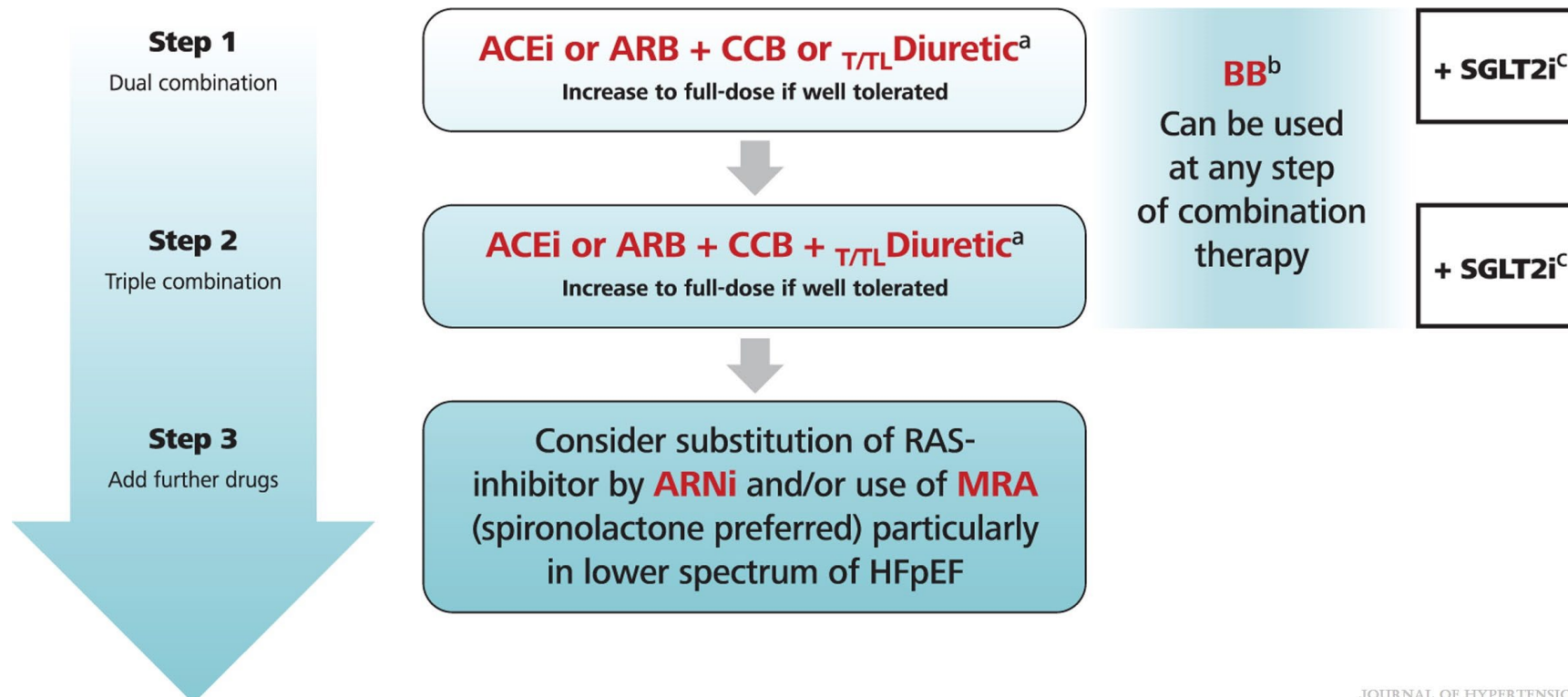
Thiaziden of T-Like

Lisdiuretica zo eGFR < 30 – 45 ml/, zo longoedeem

Medicatie in HT en hartfalen (HFpEF)

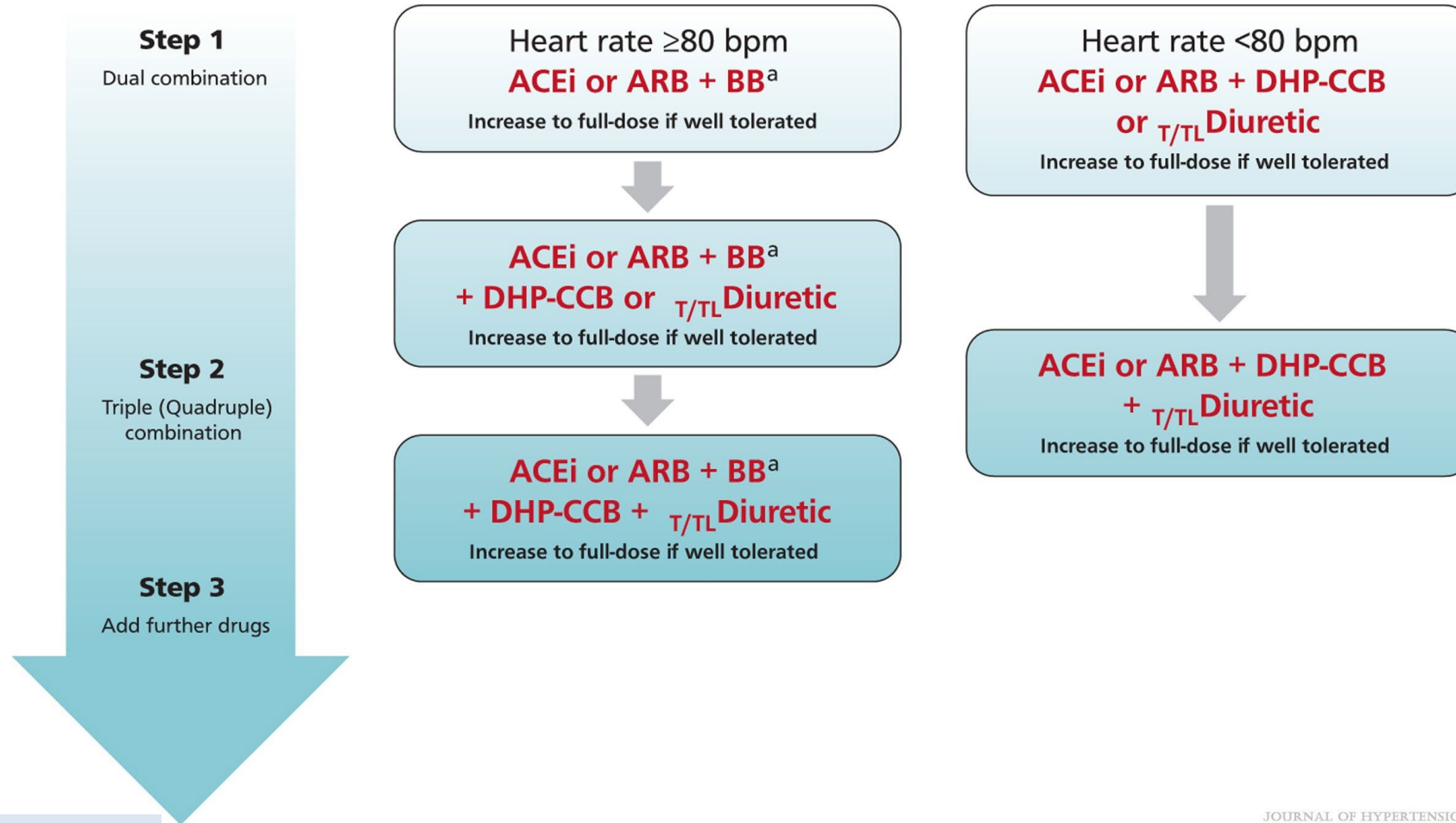
AHT en LVH is hier !!!

➔ Best naar SBD < 130 mmHg!



Medicatie in HT en VKF

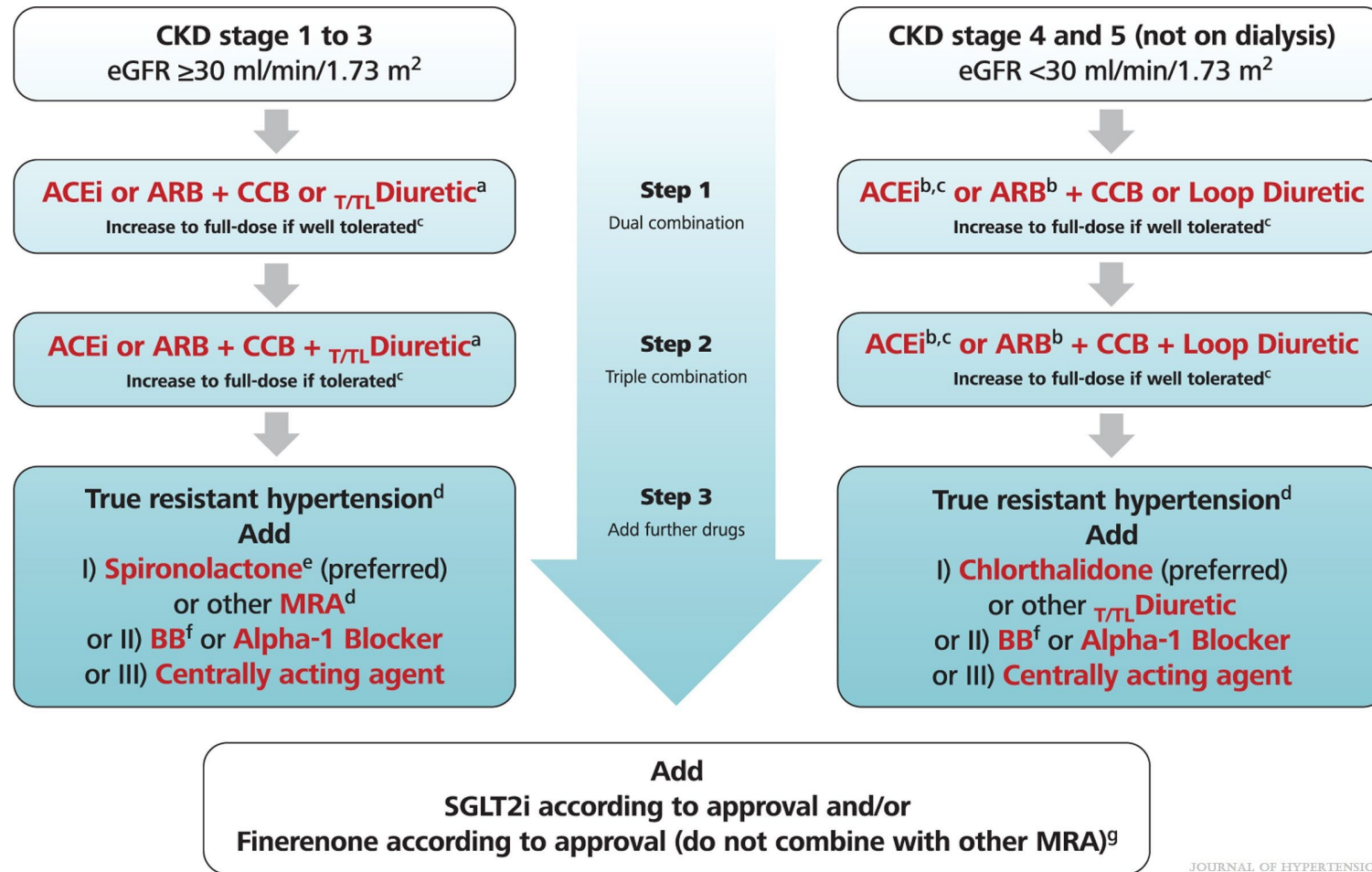
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Recommendations and statements	CoR	LoE
BP should be monitored to detect hypertension in all patients with diabetes, because it is a frequent comorbidity associated with an increase CV risk and risk for kidney events.	I	A
<u>Non-dipping or elevated night-time BP</u> are frequent in type 2 diabetes and should be monitored by ABPM or HBPM.	I	B
Antihypertensive treatment in type 2 diabetes is recommended to <u>protect against macrovascular and microvascular complications.</u>	I	A
Immediate lifestyle interventions and antihypertensive drug treatment are recommended for people with type 2 diabetes when office SBP is ≥ 140 mmHg and DBP is ≥ 90 mmHg.	I	A
Drug treatment strategies in patients with type 2 diabetes should be the same as for patients without diabetes and the <u>primary aim is to lower BP below $<130/80$ mmHg.</u>	I	A
<u>SGLT2is</u> are recommended to reduce cardiac and kidney events in type 2 diabetes.	I	A
The <u>non-steroidal MRA finerenone</u> can be used, because of its nephroprotective and cardioprotective properties in patients with diabetic CKD and moderate to severe albuminuria.	I	A
There are only limited data on the potential benefits of combining SGLT2is and finerenone.	II	C

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In all patients with CKD the primary goal is to lower office BP to <140 mmHg systolic and <90 mmHg diastolic.	I	A
In most CKD patients (<u>young patients, patients with an albumin/creatinine ratio \geq 300 mg/g, high CV risk patients</u>) office BP may be <u>lowered to <130/80 mmHg if tolerated.</u>	II	B
In <u>kidney transplant patients with hypertension</u> , office BP may be lowered to <130 mmHg systolic and <80 mmHg diastolic.	II	B
In patients with CKD, a BP target of less than 120/70 mmHg is not recommended.	III	C
An <u>ACEi or an ARB, titrated to the maximum tolerated doses</u> is recommended for patients with CKD and moderate (UACR 30 to 300 mg/g) or severe (UACR > 300 mg/g) albuminuria.	I	A

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Dual combination of an ACEi with an ARB is not recommended.	III	A
SGLT2is inhibitors are recommended for patients with diabetic and non-diabetic nephropathies CKD if eGFR is at least 20 ml/min/1.73 ² . ^a	I	A
The non-steroidal MRA finerenone is recommended in patients with CKD and albuminuria associated with type 2 diabetes mellitus if eGFR is at least 25 ml/min/1.73 ² and serum potassium <5.0 mmol/L.	I	A
In CKD patients with hyperkalemia a potassium binder can be used to maintain normal or near normal serum potassium levels (<5.5 mmol/L) in order to allow optimal treatment with a RAS-blocker or a MRA to continue.	II	B

^aAdditional eGFR and albuminuria criteria apply for initiation of treatment with different SGLT2is according to their respective approval.

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