Arterielle Hypertonie - Epidemiologie

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Epidemiologie

Was kann man evaluieren ?

Prävalenz und Inzidenz

Einfluss von Alter, Geschlecht, Umweltfaktoren, Lebensstil

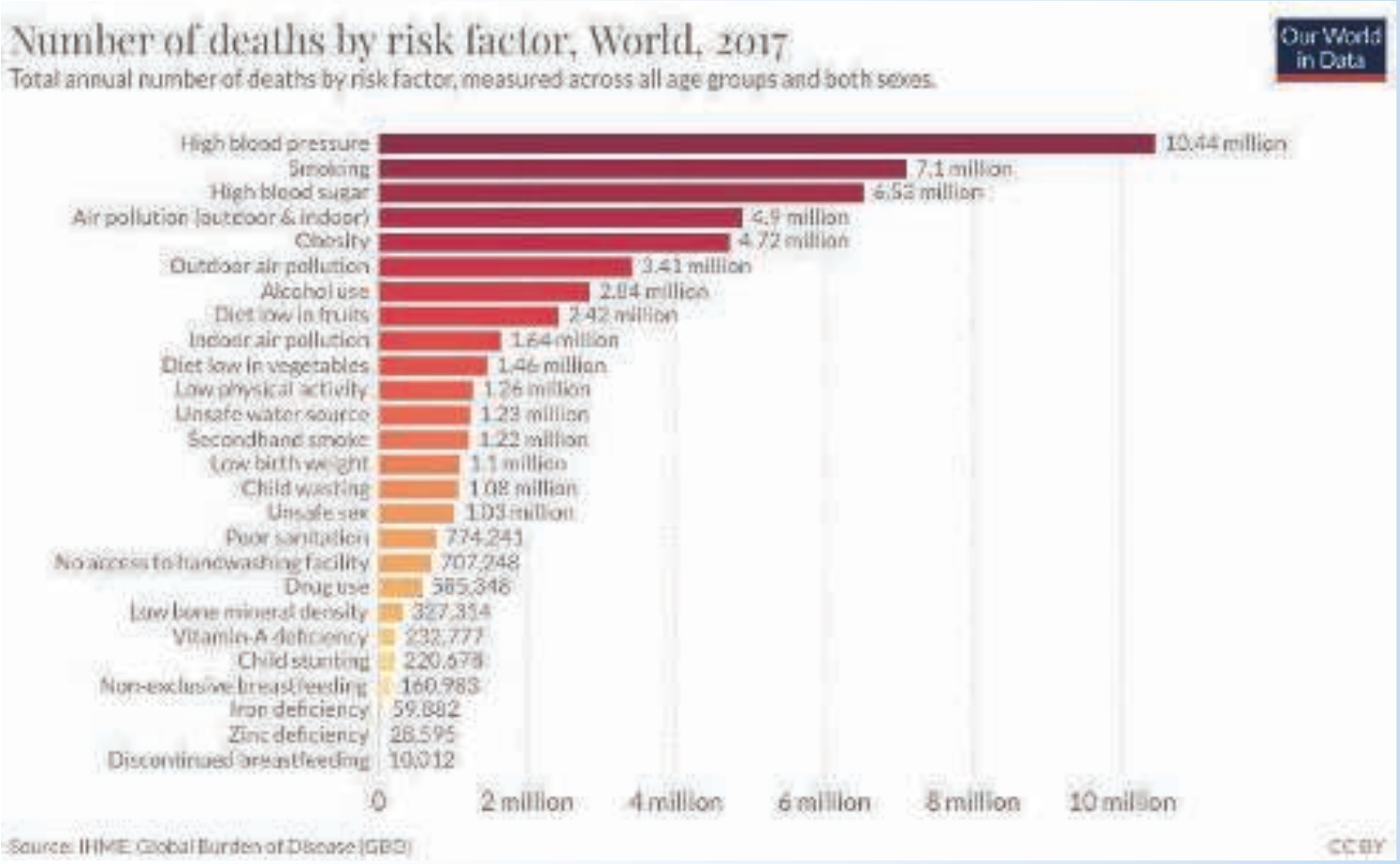
Folgeerkrankungen

Lebenszeitrisiken

Veränderungen im zeitlichen Verlauf, Therapieeffekte


MAY
MEASUREMENT
MONTH
International
Society of
Hypertension
EINE EINFACHE
MESSUNG,
DIE LEBEN RETTET
Wußten Sie, dass...
Bluthochdruck
der Nr. 1
Risikofaktor
für Herzinfarkt und
Schlaganfall ist
Bluthochdruck für
10 Mio.
unnötige
Todesfälle
jährlich verantwortlich ist
Bluthochdruck nur der
½ der
Betroffenen
bekannt ist

Todesursachen



„Came out of clear sky“

Leibarzt Admiral Ross T. McIntire

THE JOHNSTOWN DEMOCRAT

JOHNSTOWN, PA., FRIDAY MORNING, APRIL 13, 1945

ROOSEVELT DEAD!

End of Nazi Army Held Probable in Few Days

Yanks Cross Elbe, Strike For Leipzig

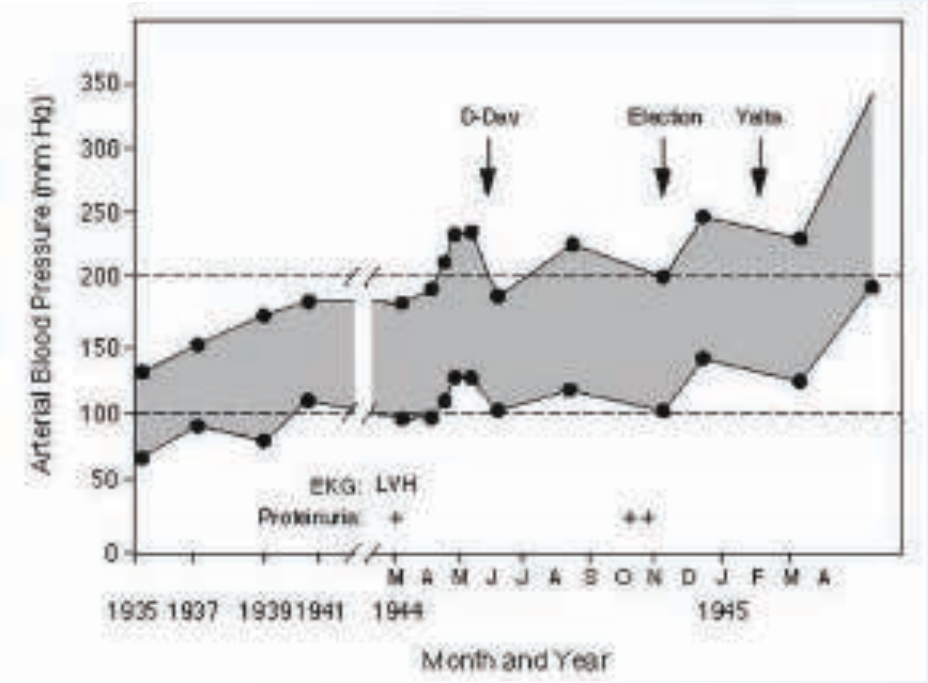
Oath Given Truman by Judge Stone

President Expires Unexpectedly Of Cerebral Hemorrhage After Speaking of 'Terrific Headache'

Chief Executive Dies on Vacation At Warm Springs

„Came out of clear sky“

Leibarzt Admiral Ross T. McIntire



Franklin D. Roosevelt starb am 12. April 1945 an einer Hirnblutung

gemessener Blutdruck: 340 / 190 mmHg ??

1937: Paul Dudley White

„Hypertension may be an important compensatory mechanism which should not be tampered with, even were it certain we could control it“

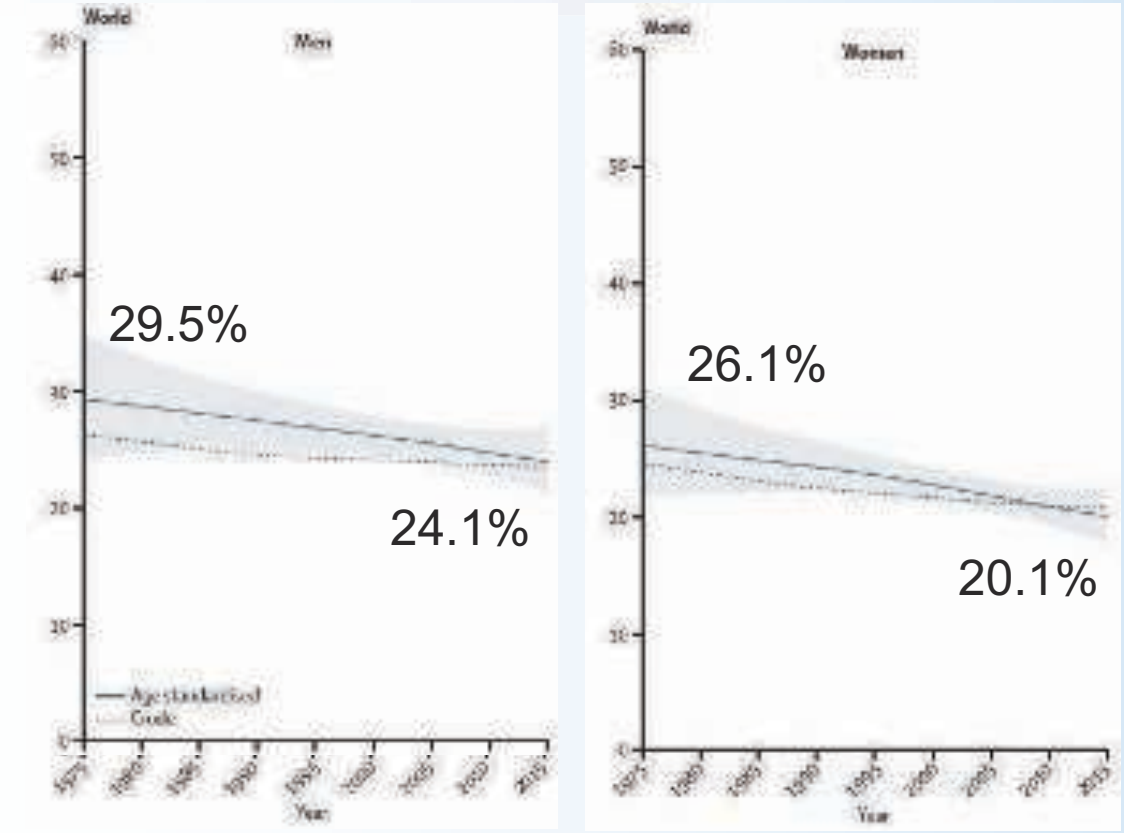
Messerli FH. This day 50 years ago. NEJM 1995; 332: 1038

Moser H. J Clin Hyperten. 2006; 8: 15

Lüscher TF. Eur Heart J. 2017; 38: 12791

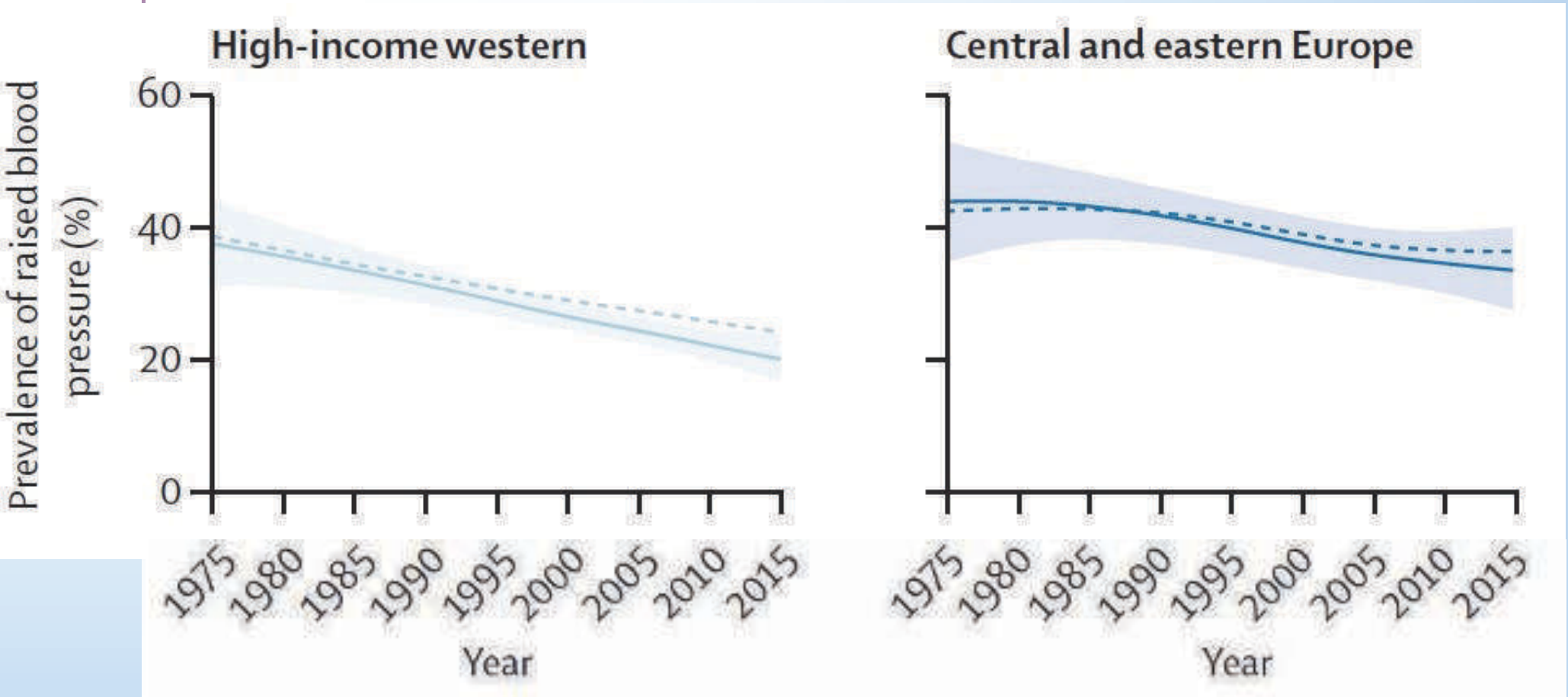
Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants

- 2015: 1.13 Milliarden Menschen betroffen – insgesamt sinkt die Prävalenz langsam mit starken regionalen Unterschieden !
- direkte Todesursache in 8,5%



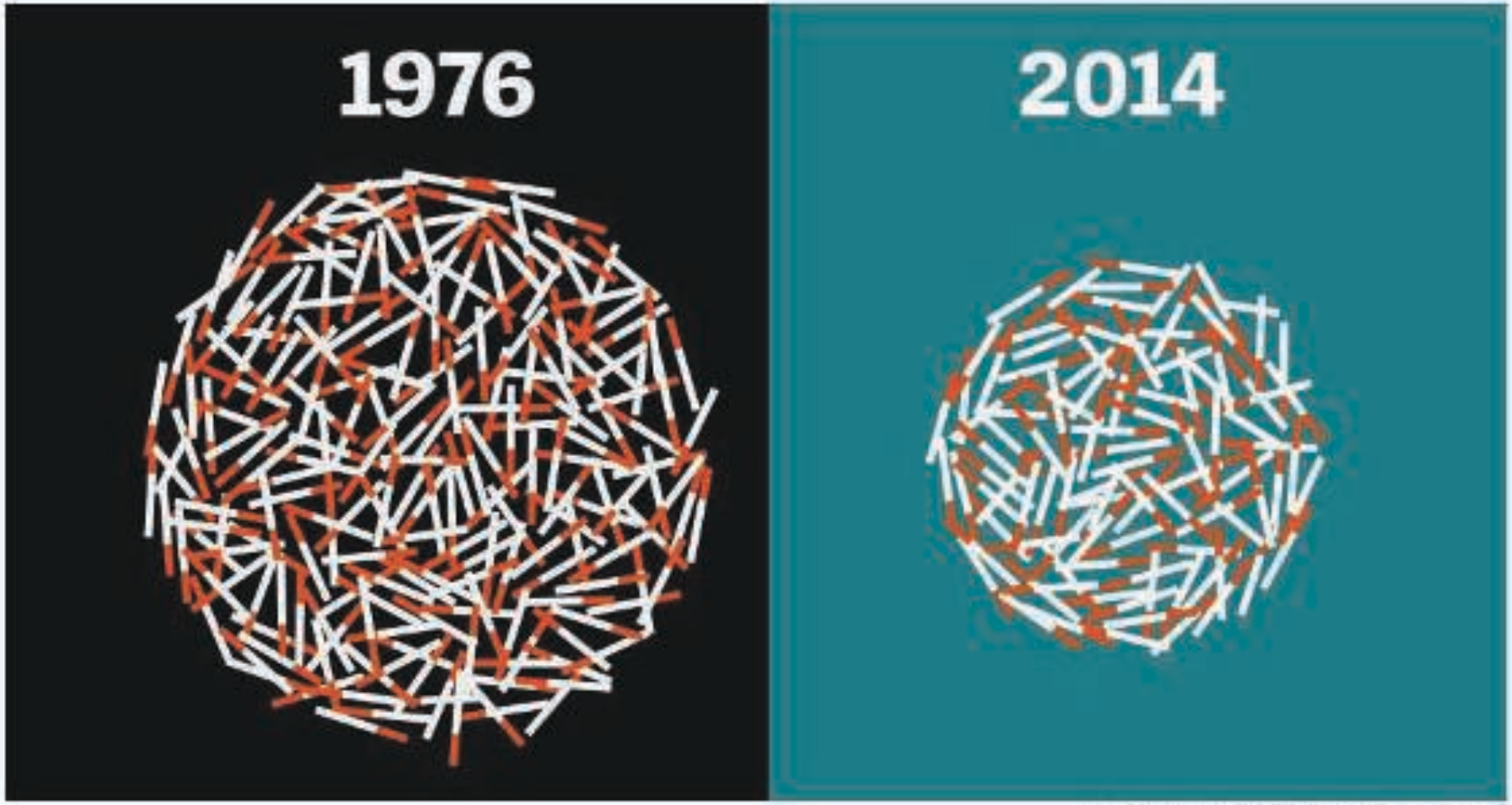
Lancet 2015

Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants



Lancet 2015

Früher was vieles schlechter



Jahrlicher Pro-Kopf-Verbrauch von Zigaretten (1976 in Westdeutschland)

Schweden: Prävalenz der CV Risikofaktoren zwischen dem 25. – 64. Lebensjahr

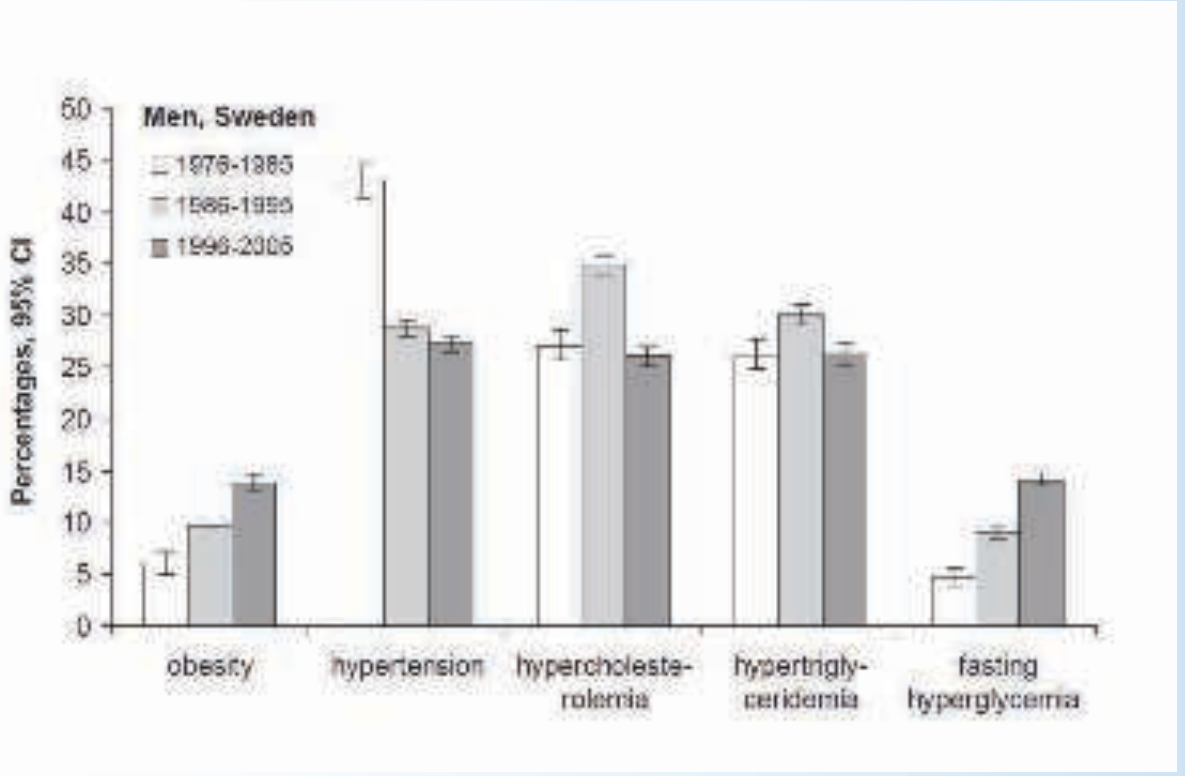
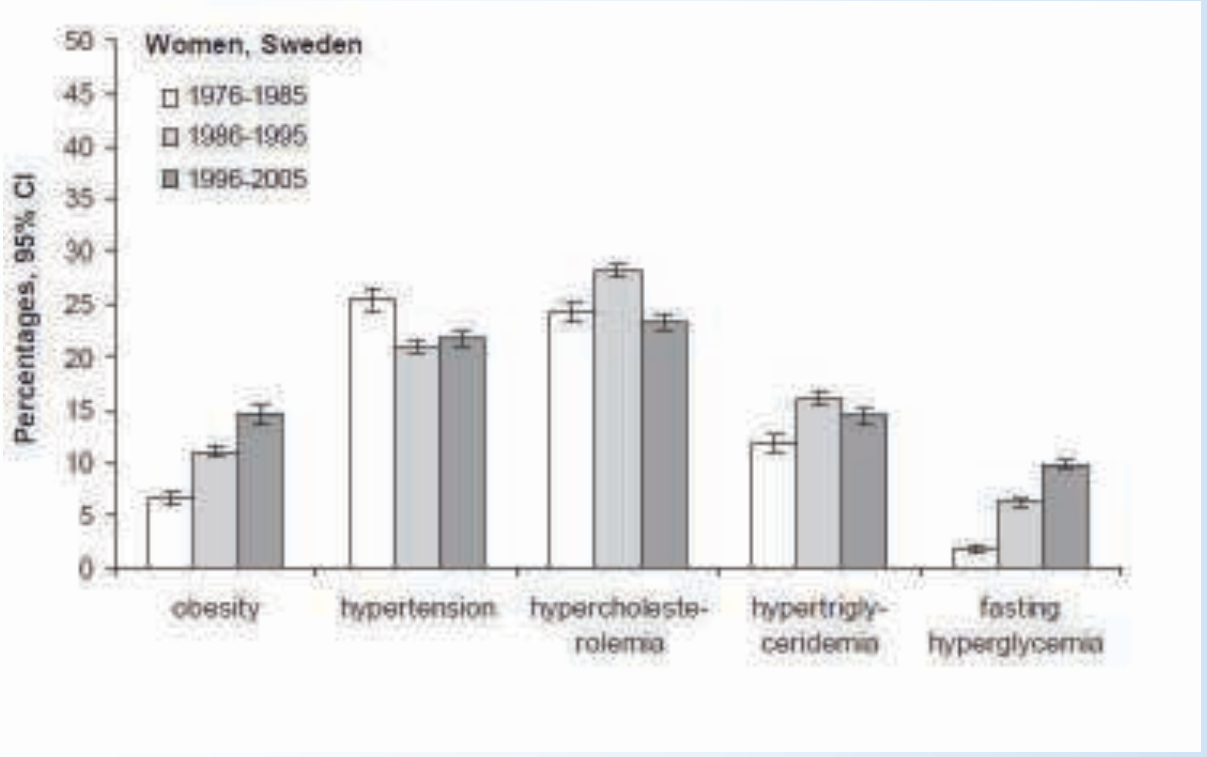


Fig. 1. Prevalence and 95% CI of obesity (BMI ≥30), hypertension (systolic blood pressure ≥140 mmHg or diastolic ≥90 mmHg), hypercholesterolemia (total cholesterol >6.1 mmol/l), hypertriglyceridemia (triglycerides ≥1.69 mmol/l) and hyperglycemia (fasting glucose >6.1 mmol/l) by decades in women and men, aged 25–64 years, Austria, Sweden

Borena W et al. Wi Kli Wo 2009; 121: 623-630

Schweden: Prävalenz der CV Risikofaktoren zwischen dem 25. – 64. Lebensjahr



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Borena W et al. Wi Kli Wo 2009; 121: 623-630

S´Ländle

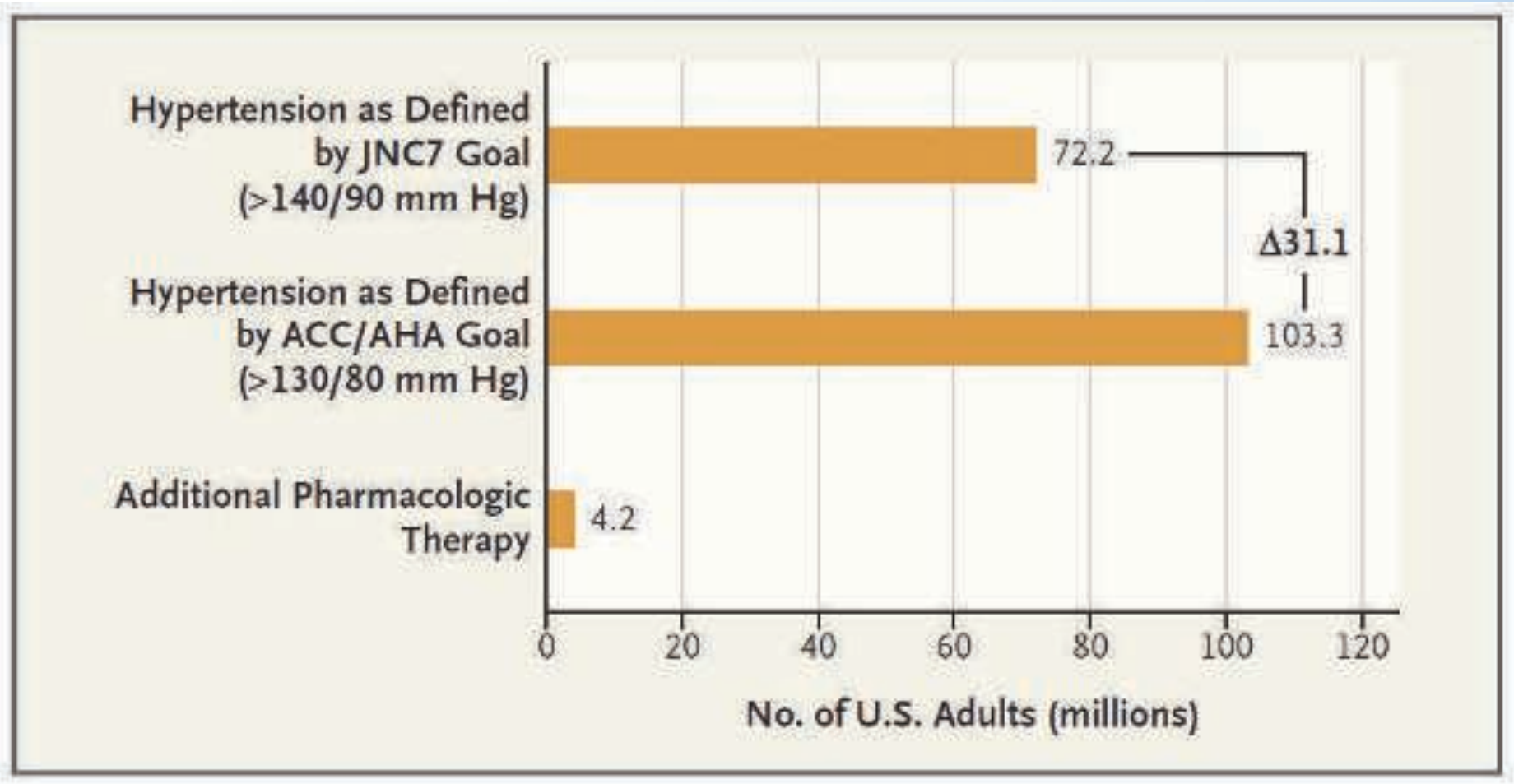
The Vorarlberg Health Monitoring & Promotion Programme (VHM&PP)

Das Vorkommen von Bluthochdruck ist von 1975 bis 2015 bei **Männern von 40,8% auf 25,2%** und bei **Frauen von 31,0% auf 16,8%** deutlich gesunken.

Schauen wir über den großen Teich …

2017: ACC / AHA senken Richtwert für Hypertonie

NEJM 2018



2017: ACC / AHA senken Richtwert für Hypertonie

ÖAZ 22.11.2017

Experten der American Heart Association haben den Grenzwert für arterielle Hypertonie in den USA herabgesetzt: als systolischer Grenzwert gilt nun 130 mmHg, als diastolischer 80 – anstatt bisher 140/90 mmHg.

Mit den neuen Richtlinien würde anerkannt, dass schon „bei diesen niedrigeren Werten" Komplikationen auftreten könnten.

Nach den neuen Richtwerten ist fast die Hälfte der US-Bevölkerung von Hypertonie betroffen; zuvor waren es 32 Prozent.

Whelton PK, et al. 2017 High Blood Pressure Clinical Practice Guideline: Executive Summary

2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults

2018: EHS/ESC Guidelines

Very high risk	People with any of the following:
	Documented CVD, either clinical or unequivocal on imaging • **Clinical CVD** includes acute myocardial infarction, acute coronary syndrome, coronary or other arterial revascularization, stroke, TIA, aortic aneurysm, and PAD • **Unequivocal documented CVD on imaging** includes significant plaque (i.e. ≥50% stenosis) on angiography or ultrasound; it does not include increase in carotid intima-media thickness
	• **Diabetes mellitus with target organ damage**, e.g. proteinuria or a with a major risk factor such as grade 3 hypertension or hypercholesterolaemia
	• **Severe CKD** (eGFR <30 mL/min/1.73 m^2)
	• **A calculated 10 year SCORE of ≥10%**
High risk	People with any of the following: • **Marked elevation of a single risk factor**, particularly cholesterol >8 mmol/L (>310 mg/dL), e.g. familial hypercholesterolaemia or grade 3 hypertension (BP ≥180/110 mmHg)
	• **Most other people with diabetes mellitus** (except some young people with type 1 diabetes mellitus and without major risk factors, who may be at moderate-risk)
	Hypertensive LVH
	Moderate CKD eGFR 30-59 mL/min/1.73 m^2)
	A calculated 10 year SCORE of 5-10%
Moderate risk	People with: • **A calculated 10 year SCORE of ≥1 to <5%** • **Grade 2 hypertension** • **Many middle-aged people belong to this category**
Low risk	People with: • **A calculated 10 year SCORE of <1%**

2018: EHS/ESC Guidelines

Table 7 Correction factors for the Systemic COronary Risk Evaluation (SCORE) cardiovascular risk estimates in first-generation immigrants to Europe[35]

Region of origin	Multiplication factor
Southern Asia	1.4
Sub-Saharan Africa	1.3
Caribbean	1.3
Western Asia	1.2
Northern Africa	0.9
Eastern Asia	0.7
Southern America	0.7

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Table 6 Risk modifiers increasing cardiovascular risk estimated by the Systemic COronary Risk Evaluation (SCORE) system[35]

Social deprivation, the origin of many causes of CVD
Obesity (measured by BMI) and central obesity (measured by waist circumference)
Physical inactivity
Psychosocial stress, and fatigue/vital exhaustion
Family history of premature CVD (occurring at age <55 years in men and <65 years in women)
Autoimmune and other inflammatory diseases
Major psychiatric disorders
Treatment for infection with human immunodeficiency virus
Atrial fibrillation
LV hypertrophy
CKD
Obstructive sleep apnoea syndrome

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BMI = body mass index; CKD = chronic kidney disease; CVD = cardiovascular disease; LV = left ventricular.

2018: EHS/ESC Guidelines

Hypertension disease staging	Other risk factors, HMOD, or disease	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 or DBP ≥110
Stage 1 (uncomplicated)	No other risk factors	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 (asymptomatic disease)	HMOD, CKD grade 3, or diabetes mellitus without organ damage	Moderate to high risk	High risk	High risk	High to very high risk
Stage 3 (established disease)	Established CVD, CKD grade ≥4, or diabetes mellitus with organ damage	Very high risk	Very high risk	Very high risk	Very high risk

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Risikofaktoren

Risk factors
Male sex
Age (men ≥55 years; women ≥65 years)
Smoking
Dyslipidaemia
Total cholesterol >4.9 mmol/L (190 mg/dL), and/or
Low-density lipoprotein cholesterol >3.0 mmol/L (115 mg/dL), and/or
High-density lipoprotein cholesterol: men <1.0 mmol/L (40 mg/dL), women <1.2 mmol/L (46 mg/dL), and/or
Triglycerides >1.7 mmol/L (150 mg/dL)
Fasting plasma glucose 5.6–6.9 mmol/L (102–125 mg/dL)
Abnormal glucose tolerance test
Obesity [BMI ≥30 kg/m^2 (height^2)]
Abdominal obesity (waist circumference: men ≥102 cm; women ≥88 cm) (in Caucasians)
Family history of premature CVD (men aged <55 years; women aged <65 years)

Endorganschaden

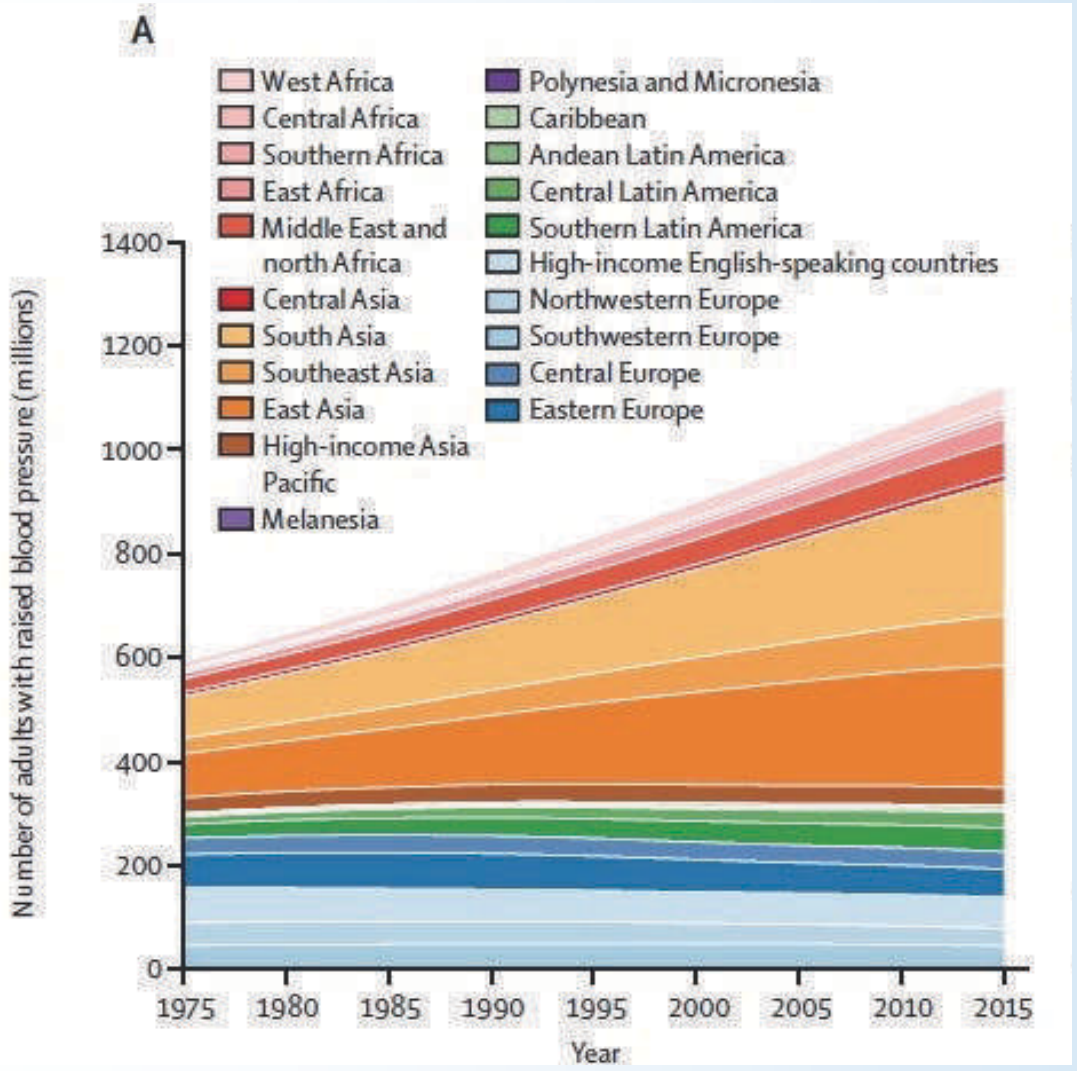
Asymptomatic organ damage
Pulse pressure (in the elderly) ≥60 mmHg
Electrocardiographic LVH (Sokolow–Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms), or
Echocardiographic LVH [LVM index: men >115 g/m^2; women >95 g/m^2 (BSA)][a]
Carotid wall thickening (IMT >0.9 mm) or plaque
Carotid–femoral PWV >10 m/s
Ankle-brachial index <0.9
CKD with eGFR 30–60 mL/min/1.73 m^2 (BSA)
Microalbuminuria (30–300 mg/24 h), or albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine)

IMMER
Hochrisikopatienten

Established CV or renal disease
Cerebrovascular disease: ischaemic stroke; cerebral haemorrhage; transient ischaemic attack
CHD: myocardial infarction; angina; myocardial revascularization with PCI or CABG
Heart failure, including heart failure with preserved EF
Symptomatic lower extremities peripheral artery disease
CKD with eGFR <30 mL/min/1.73m^2 (BSA); proteinuria (>300 mg/24 h).
Advanced retinopathy: haemorrhages or exudates, papilloedema

Schwellenländer…

Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants



Lancet 2015

Lebenszeitrisiko

(bei 140/90 mmHg – was wär erst bei 130/80 mmHg ?!?)

MESA (Multi-Ethnic Study of Atherosclerosis): Risiko, zwischen dem 45. und dem 85. Lebensjahr eine arterielle Hypertonie zu entwickeln

Afroamerikaner (93%) > Hispanics (92%) > Kaukasier (86%) > Asiaten (84%)

Framingham: 90% aller 55-bis 65- Jährigen entwickeln in ihrem Leben eine arterielle Hypertonie

Parikh NI, Pencina MJ, Wang TJ, et al. A risk score for predicting near - term incidence of hypertension: the Framingham Heart Study. Ann Intern Med. 2008;148:102 - 10.

Alter, Alter, Alter ...

Bis zu ¾ der über 75-jährigen leiden an Bluthochdruck

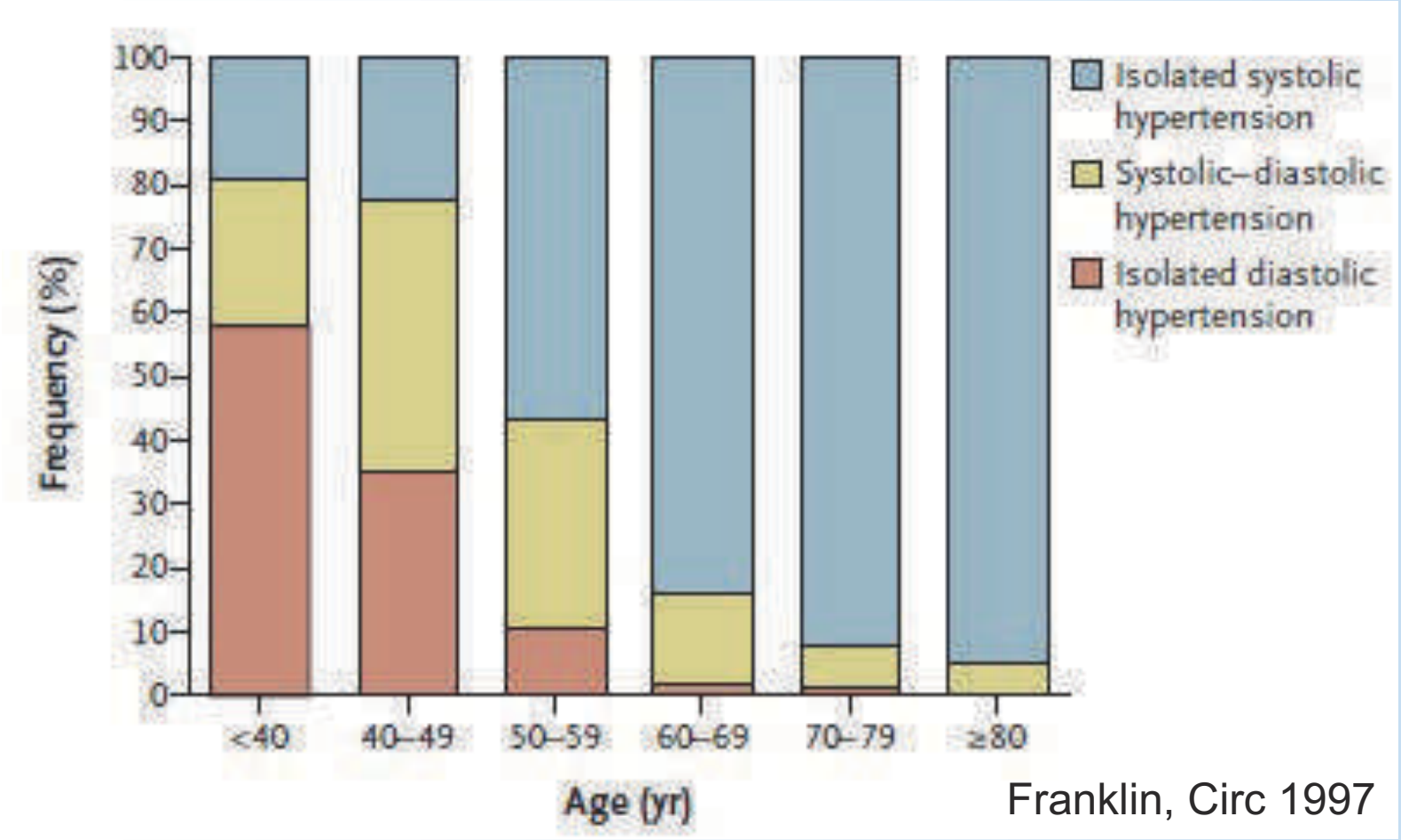
Alterungsprozesse

- Gefäße werden steifer
- Abnahme der Dilatationskapazität
- endotheliale Dysfunktion

erschwerte Diagnose

- bis zu einem Drittel Weißkittelhypertonie
- aber auch maskierte Hypertonie

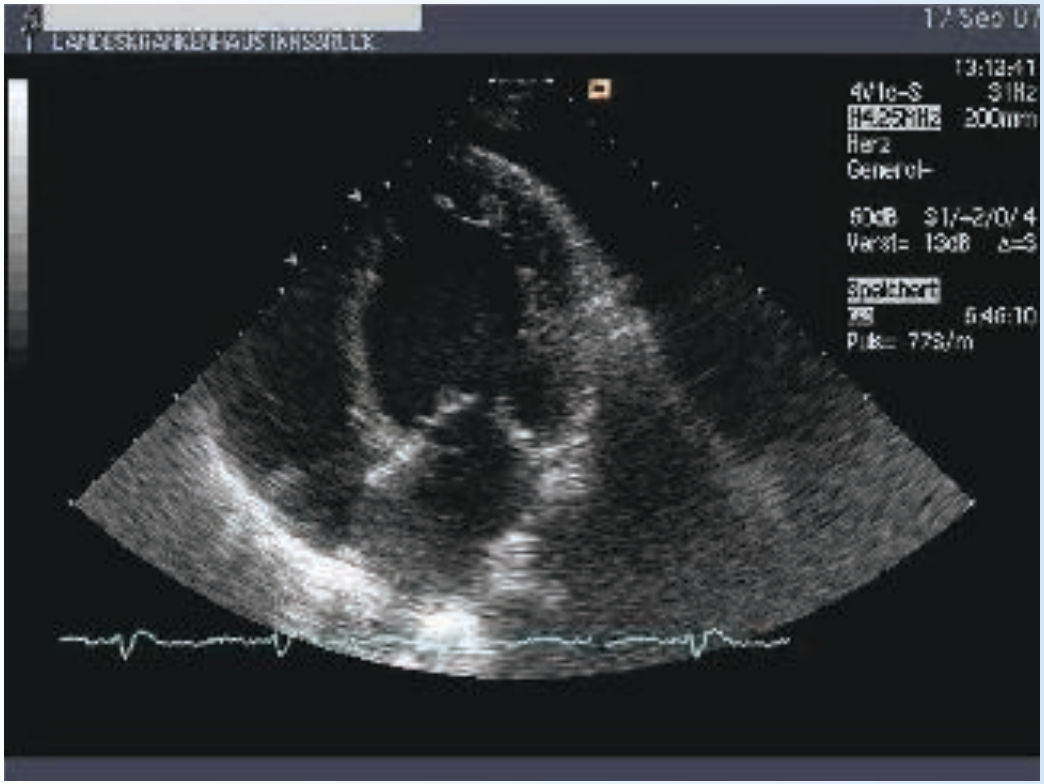
Target: systolischer Blutdruck



Franklin, Circ 1997

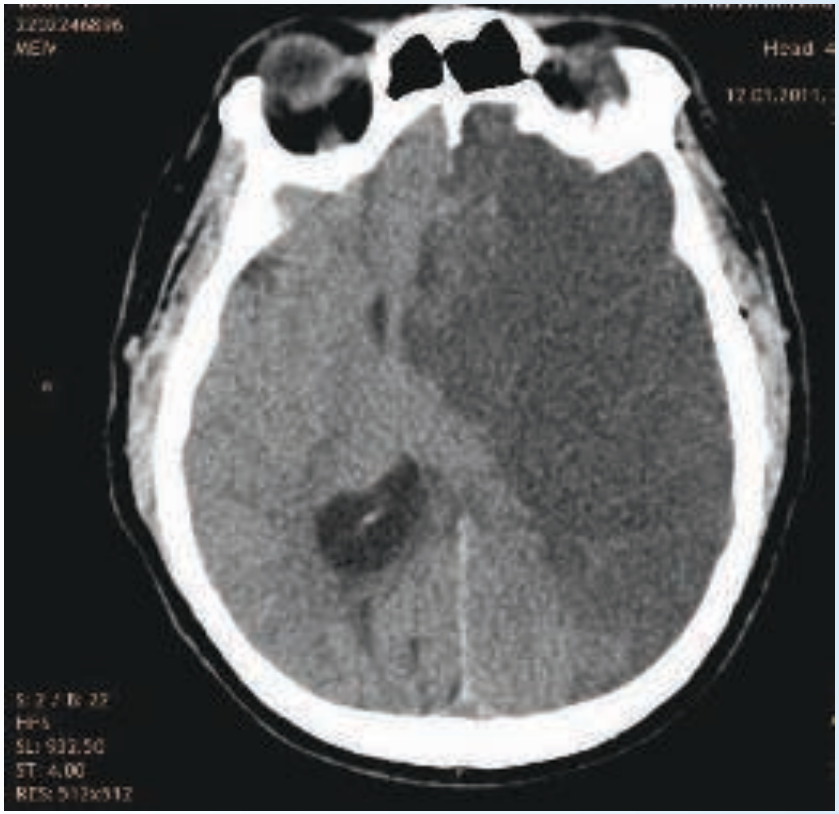
Gefäßsteifigkeit steigt, diastolischer Blutdruck sinkt im Alter

Was bringt die antihypertensive Therapie ?



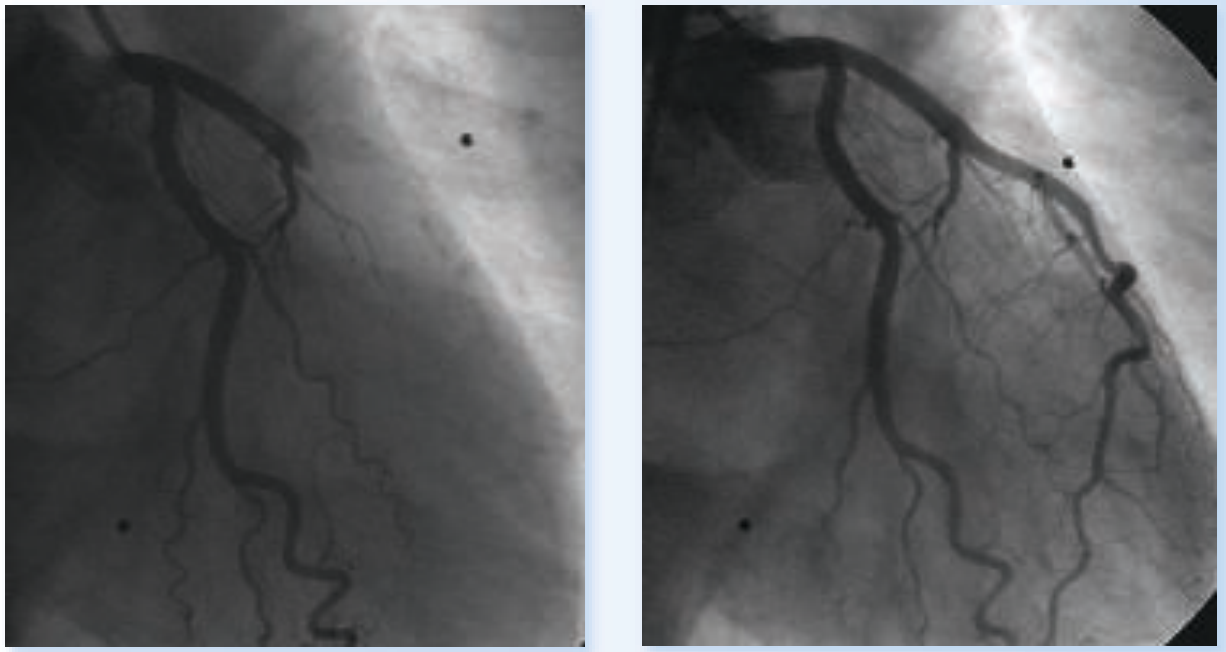
Herzschwäche (- 64%)

Was bringt die antihypertensive Therapie ?



Schlaganfall (- 40%)

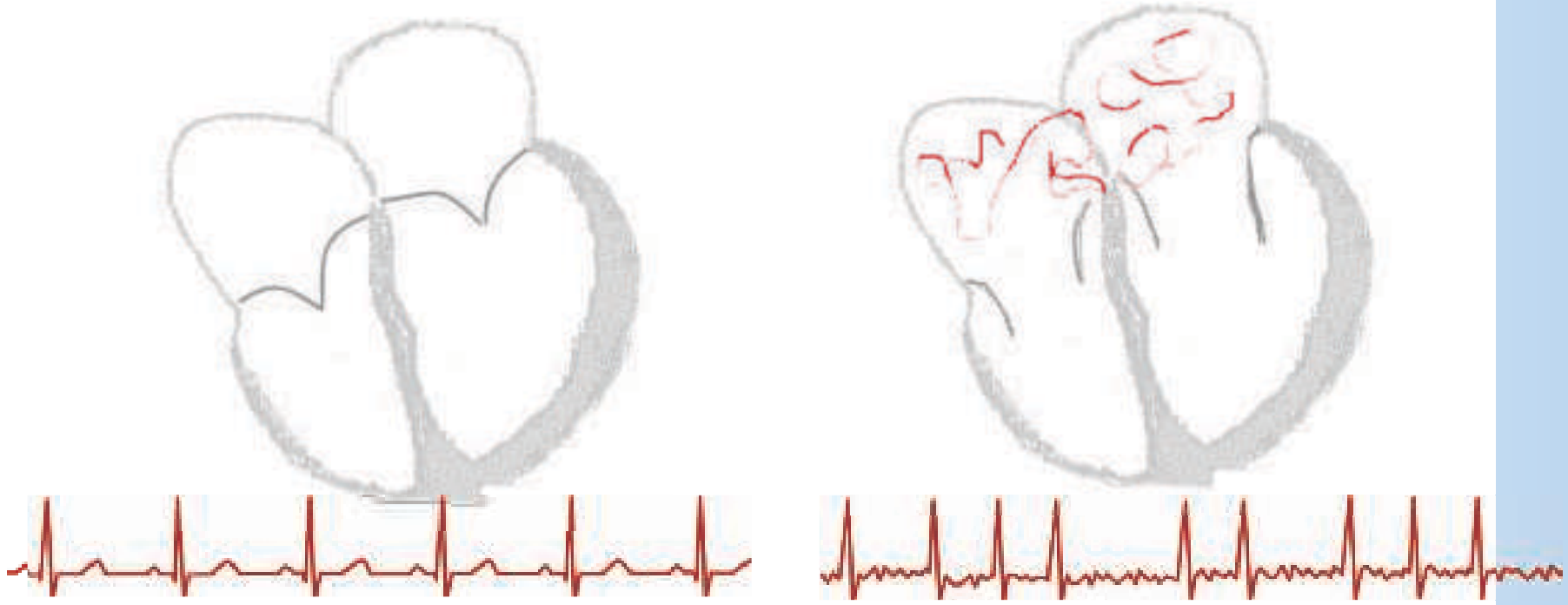
Was bringt die antihypertensive Therapie ?



Herzinfarkt (- 20%)

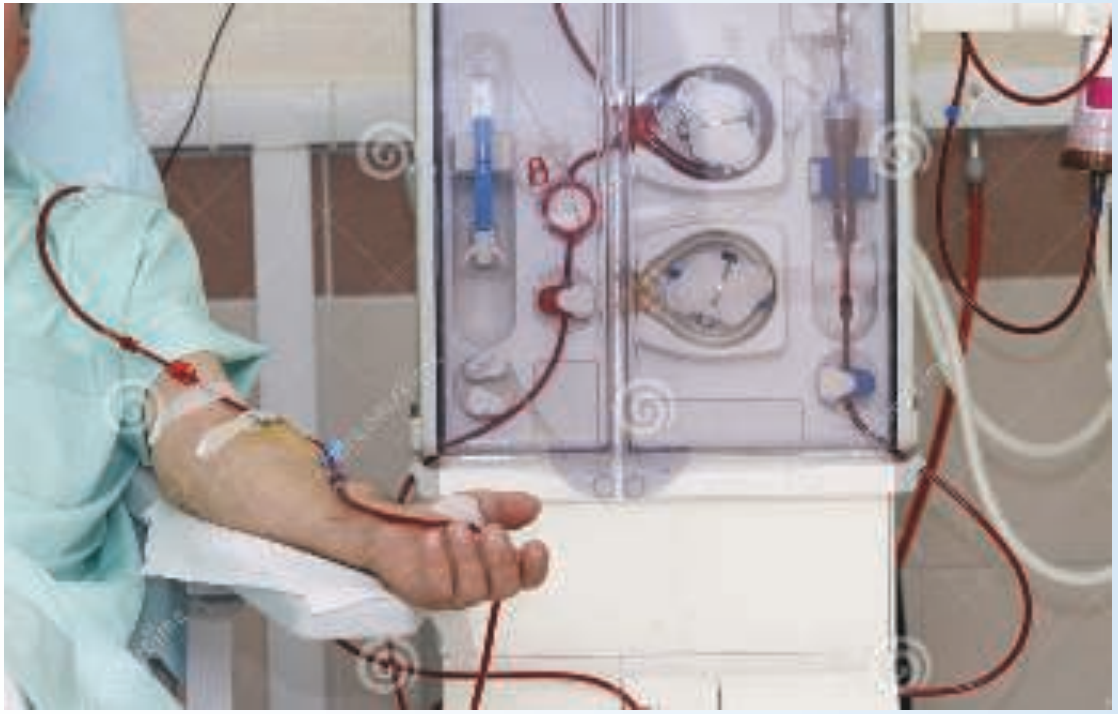
Was bringt die antihypertensive Therapie ?

Sinusrhythmus	Vorhofflimmern



Vorhofflimmern (- 15%)

Was bringt die antihypertensive Therapie ?



Verlangsamung der Niereninsuffizienz

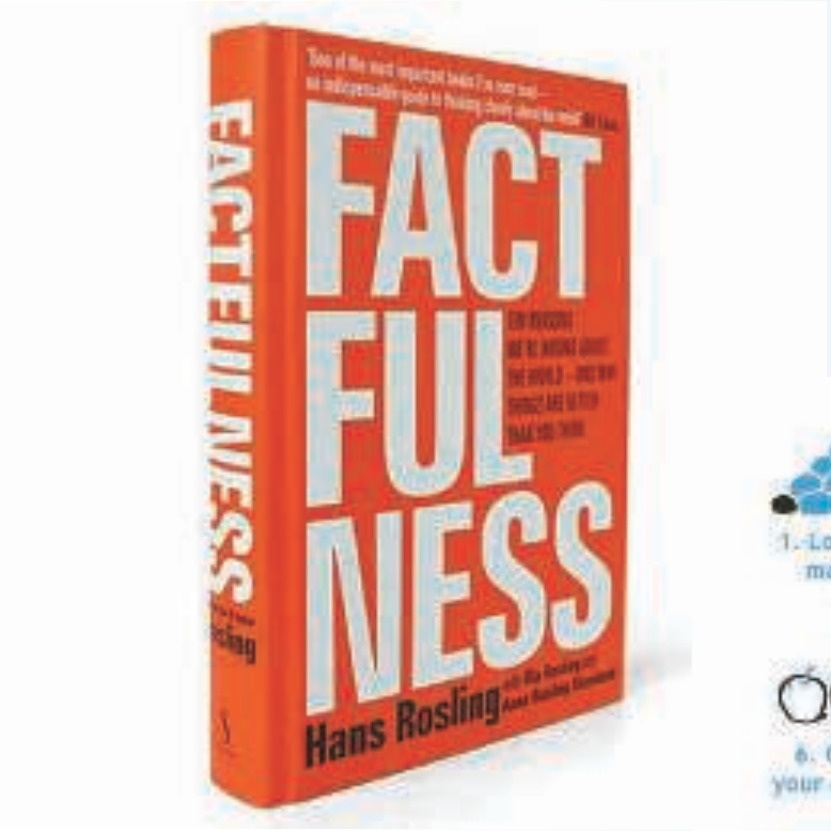
Kleine Unterschiede der kardiovaskulären Risikofaktoren LDL-Cholesterin und systolischer Blutdruck sind klinisch hochrelevant, wenn sie über einen langen Zeitraum bestehen

Falls LDL Cholesterin – 39 mg/dl (= 1 mmol/L) & systolischer Blutdruck – 10 mmHg

- 80%ige Reduktion des lebenslangen Risiko für ein kardiovaskuläres Event
- 75%ige Risikoreduktion bzgl. Herzinfarkt
- 68%ige Risikoreduktion bzgl. kardiovaskulärem Tod

UK-Biobank 440.000 Personen, bei denen fundierte Genanalysen liegen – "Mendelsche Randomisation" ; 2 bevorzugte genetische Gruppen (LDL Cholesterin um 15 mg/dl weniger als Kontrolle; systolischer Blutdruck um 3 mmHg weniger als Kontrolle)

Früher was vieles schlechter



Risikostratifizierung, Zielwerte

PD Dr Sabine Perl

Kardiologie Graz

Österreichische Gesellschaft für Hypertensiologie

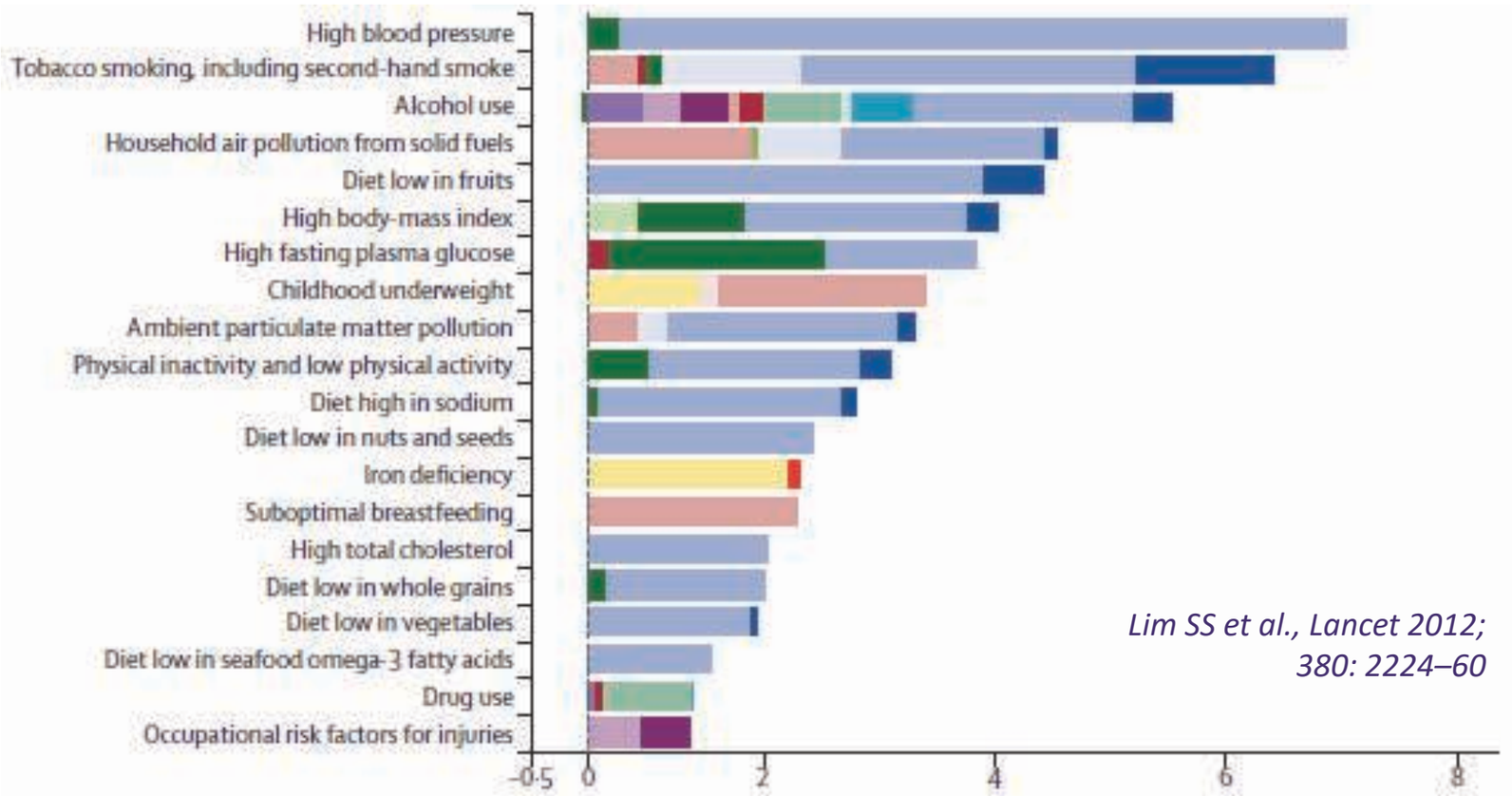
Disclosures

- Vortrags- und Konsulententätigkeiten

  Pfizer-BMS, MSD, Servier, Sanofi Aventis, Menarini, Takeda, Novartis, Neucomed, Amgen, Ratiopharm

Kardiovaskuläres Risiko

Einfluss durch die 20 führenden Risikofaktoren 2010

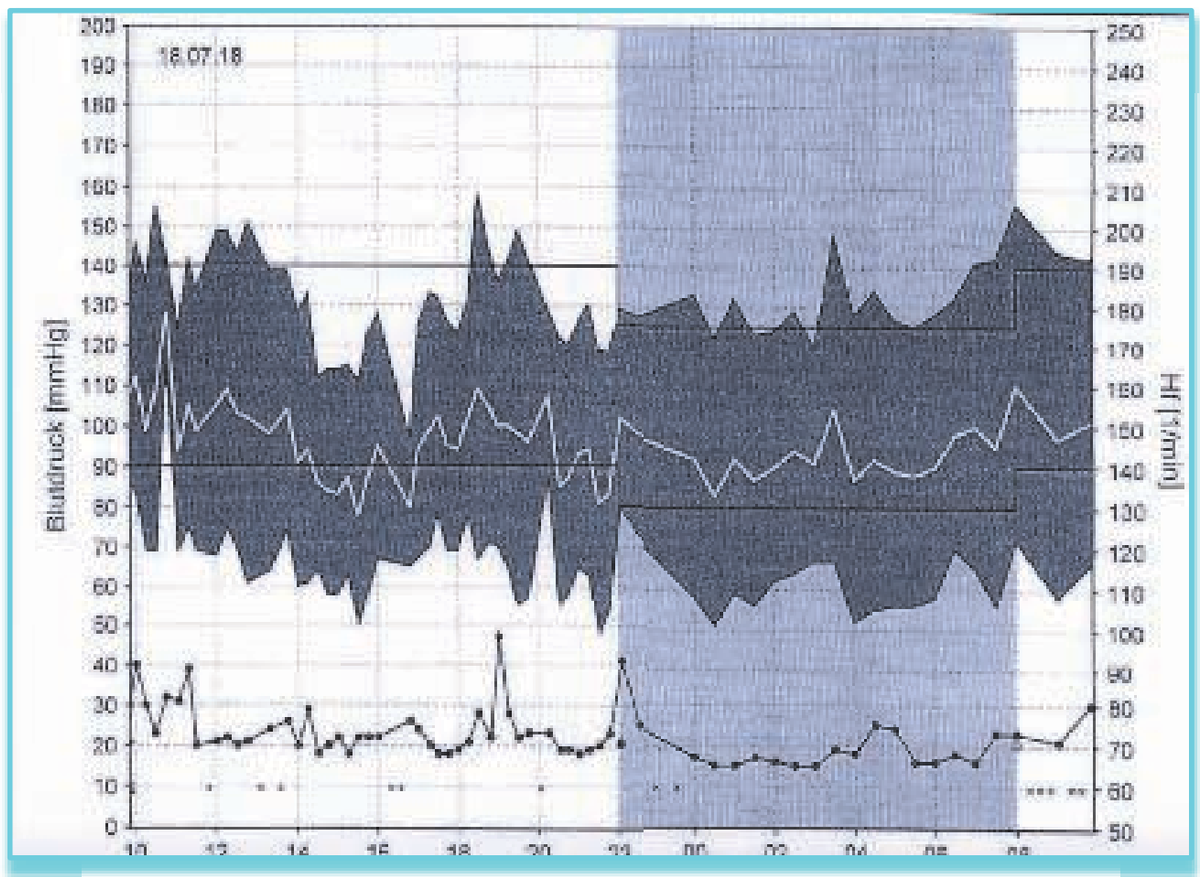


Lim SS et al., Lancet 2012; 380: 2224–60

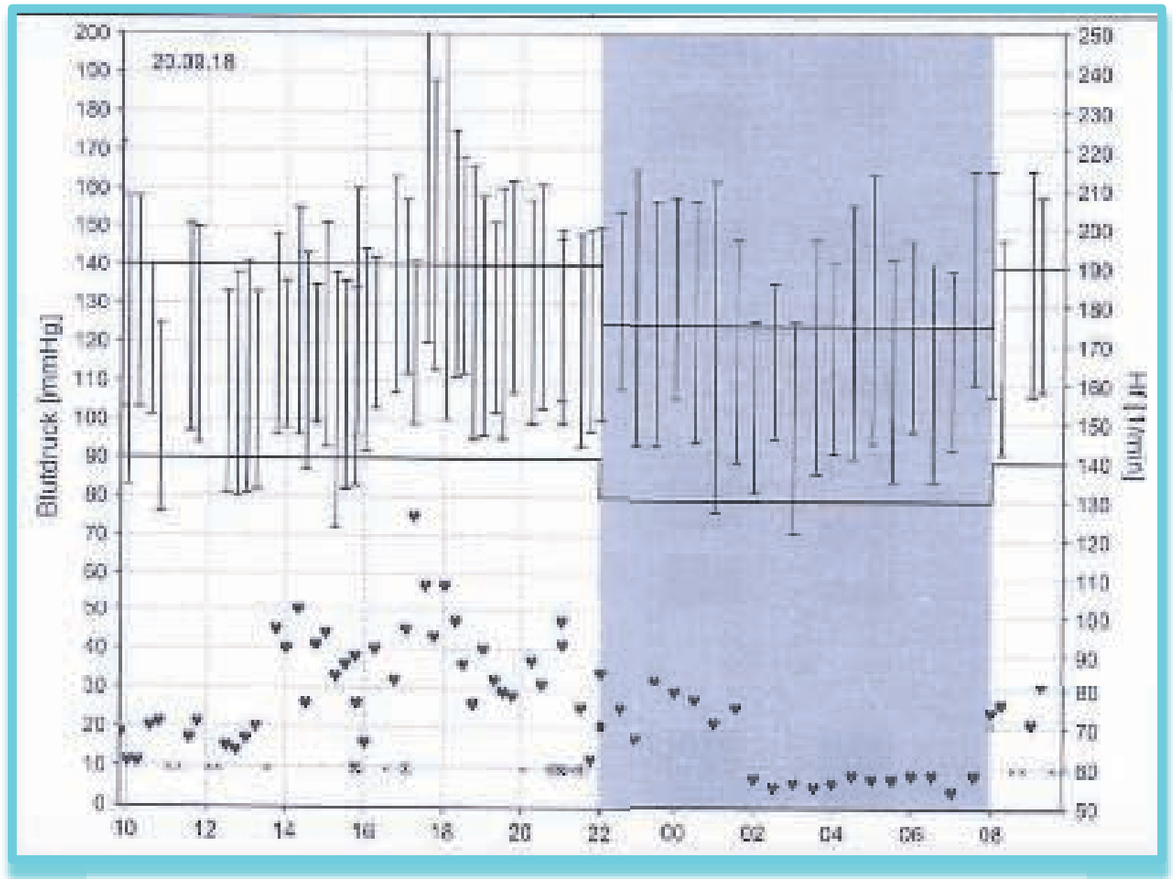
- RR 152/87mmHg

- RR 152/87mmHg

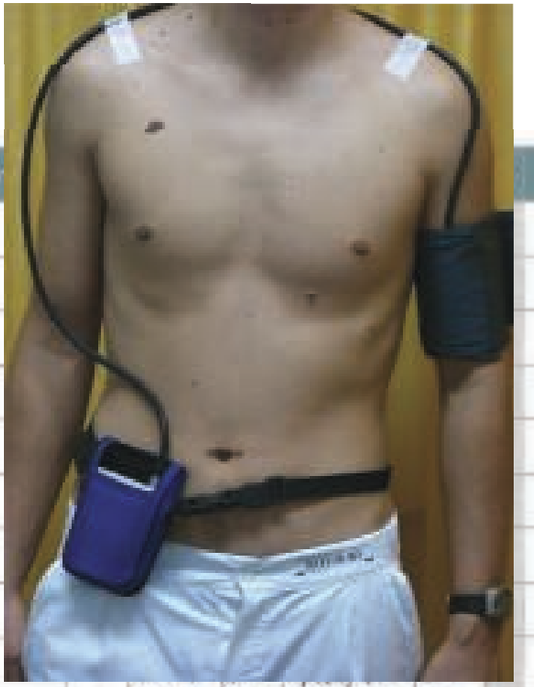
- RR 152/87mmHg



24 h 132/66mmHg



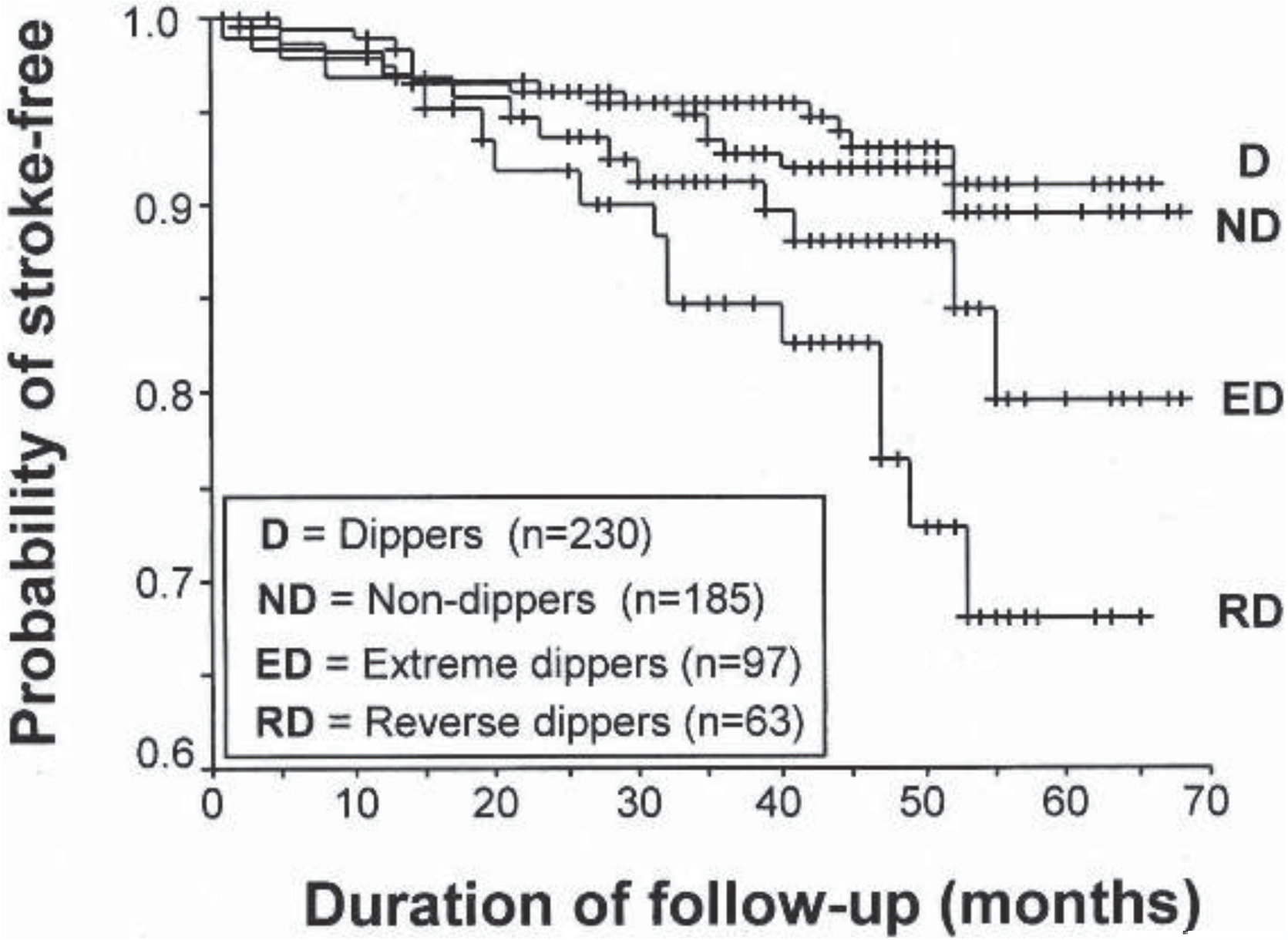
24 h 153/96mmHg



Datum	Zeit	Blutdruck	Puls	✱	A
4.1	9⁰⁰	130/90	96		
4.1	21⁰⁰	127/88	72		
5.1	9⁰⁰	114/93	82		
5.1	21⁰⁰	134/100	67		
6.1	8⁰⁰	130/82	69		
6.1	21⁰⁰	128/86	71		
7.1	7⁰⁰	114/82	87		
7.1	20⁰⁰	129/85	72		
		/90	81		
		/72	71		
		9/87	96		
		/78	69		
		/80	81		
		2/80	70		
		9/85	79		
		6/82	72		

17.1	8³⁰	122/85	80
17.1	21⁰⁰	122/82	69
18.1	8³⁰	117/81	80
18.1	20³⁰	138/90	64
19.1	9⁰⁰	109/83	78
19.1	20³⁰	123/	
20.1	9⁰⁰	122/	
Mittelwert			

Datum	Zeit		
20.1	20³⁰		
21.1	9⁰⁰		
22.1	9⁰⁰		
22.1	21⁰⁰		
23.1	9⁰⁰	124/90	75
23.1	21⁰⁰	131/94	70
24.1	9⁰⁰	117/87	78
24.1	21⁰⁰	121/94	68
26.1	8³⁰	122/82	83
27.1	8⁴⁵	118/86	78
27.1	21⁰⁰	134/91	69
28.1	9⁰⁰	118/86	76
29.1	9⁰⁰	107/82	82
29.1	20³⁰	131/86	66



Kario; Hypertension 2001;38:852

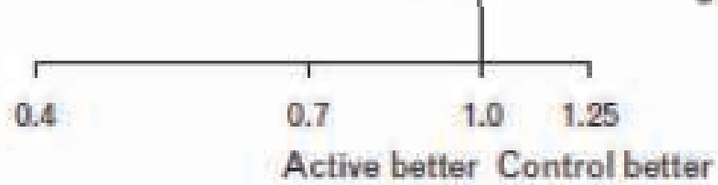
Effects of blood pressure lowering on outcome incidence in hypertension. 1. Overview, meta-analyses, and meta-regression analyses of randomized trials

Costas Thomopoulos[a], Gianfranco Parati[b,c], and Alberto Zanchetti[d,e]

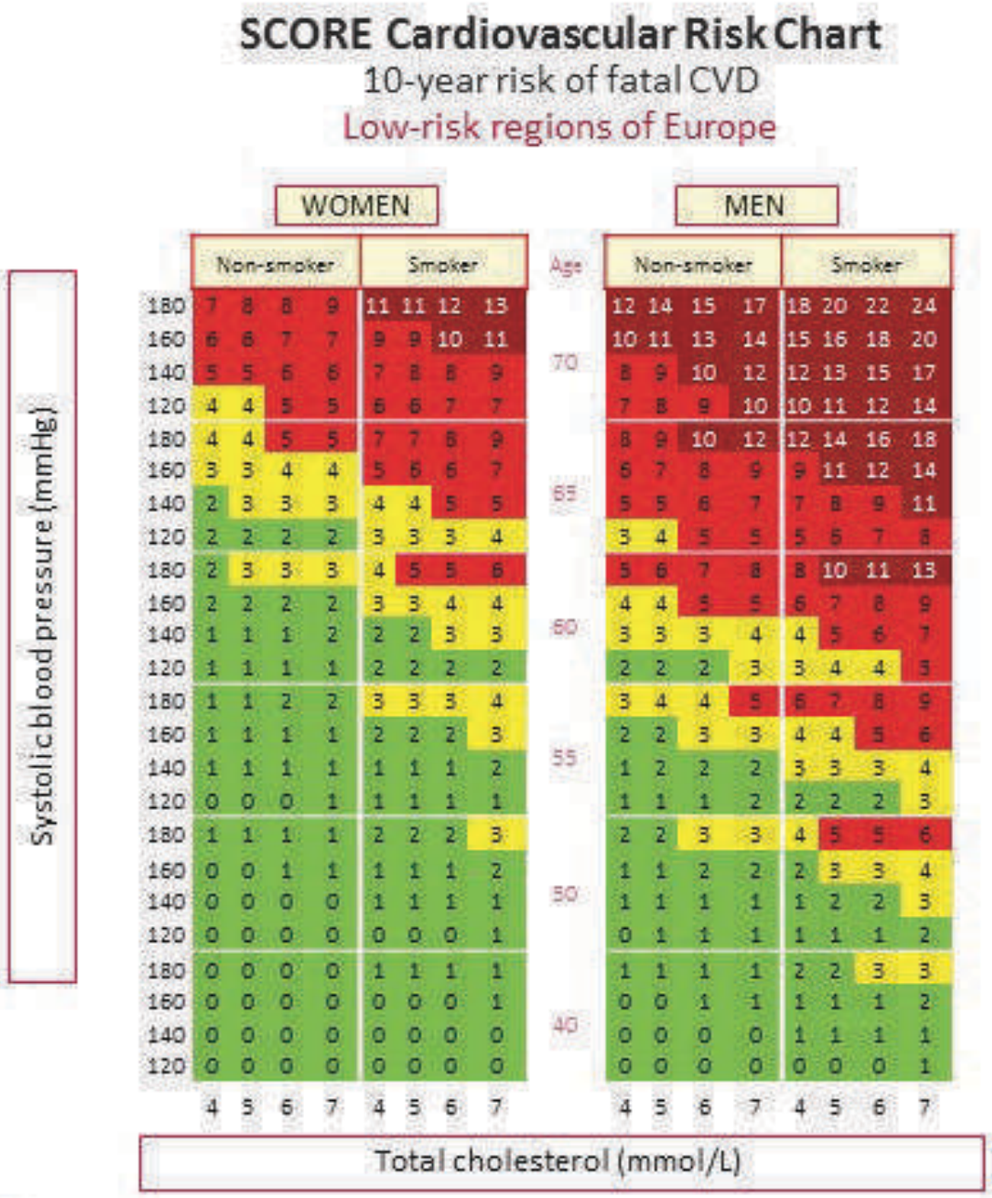
68 Studien, 245.885 Patienten

Pro Reduktion
10/5 mm Hg

Outcome	Standardized RR (95% CI)	Standardized RR (95% CI)	Absolute Risk reduction 1000 pts/5 years (95% CI)	NNT 5 years (95% CI)
Stroke	0.63 (0.56–0.71)		–15 (–18, –12)	67 (57, 84)
CHD	0.78 (0.72–0.85)		–8 (–11, –6)	118 (94, 171)
HF	0.54 (0.45–0.64)		–12 (–14, –10)	81 (70, 101)
Stroke + CHD	0.75 (0.71–0.79)		–17 (–20, –15)	57 (50, 68)
Stroke + CHD + HF	0.74 (0.69–0.81)		–25 (–31, –19)	40 (32, 52)
CV death	0.80 (0.73–0.88)		–7 (–10, –5)	134 (100, 221)
All-cause death	0.88 (0.82–0.93)		–8 (–12, –5)	124 (83, 212)



Journal of Hypertension 2014, 32:2285–2295



SCORE-Chart Niedrig-risikoländer

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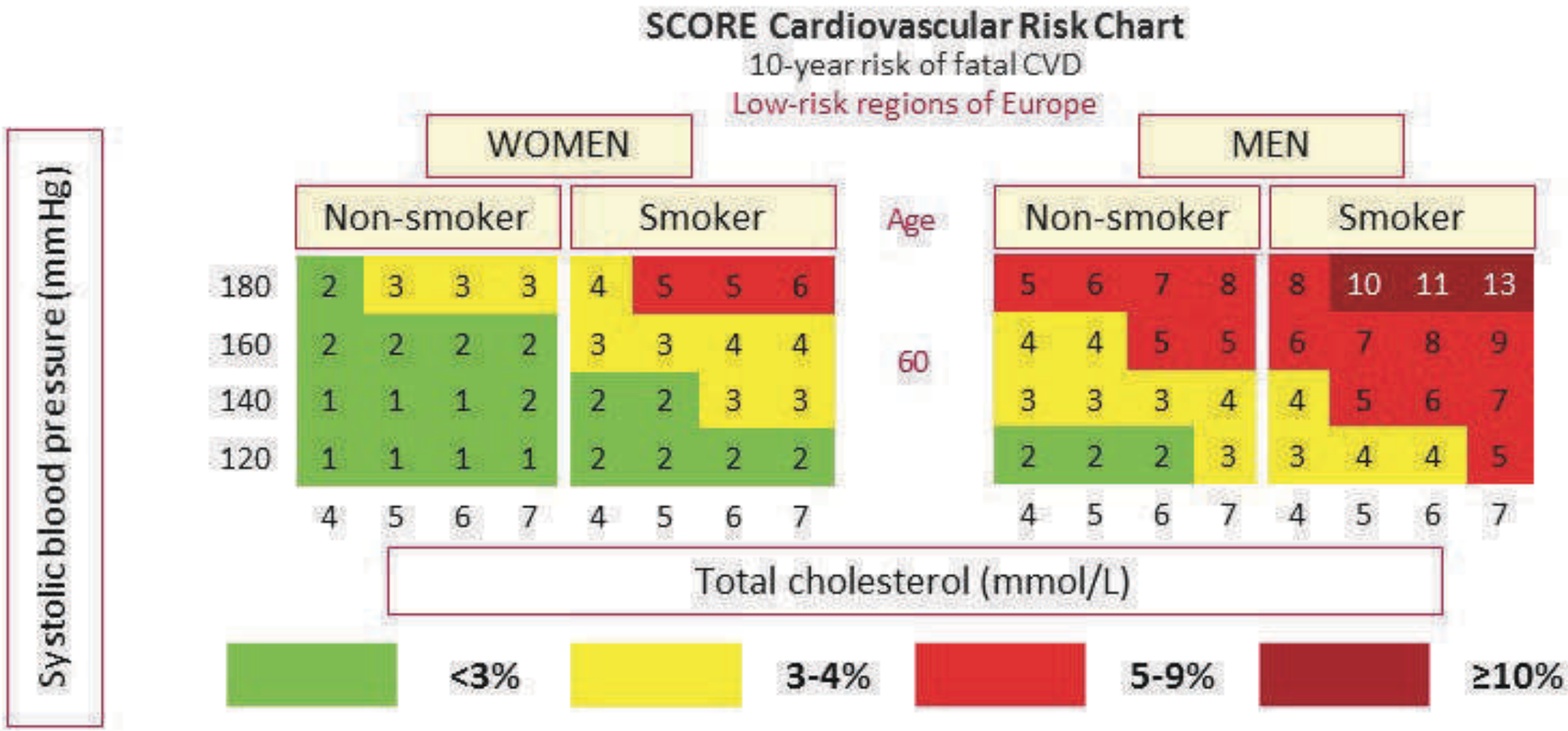
2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

SCORE-Chart Niedrig-Risikoländer

EAS ESC European Society of Cardiology



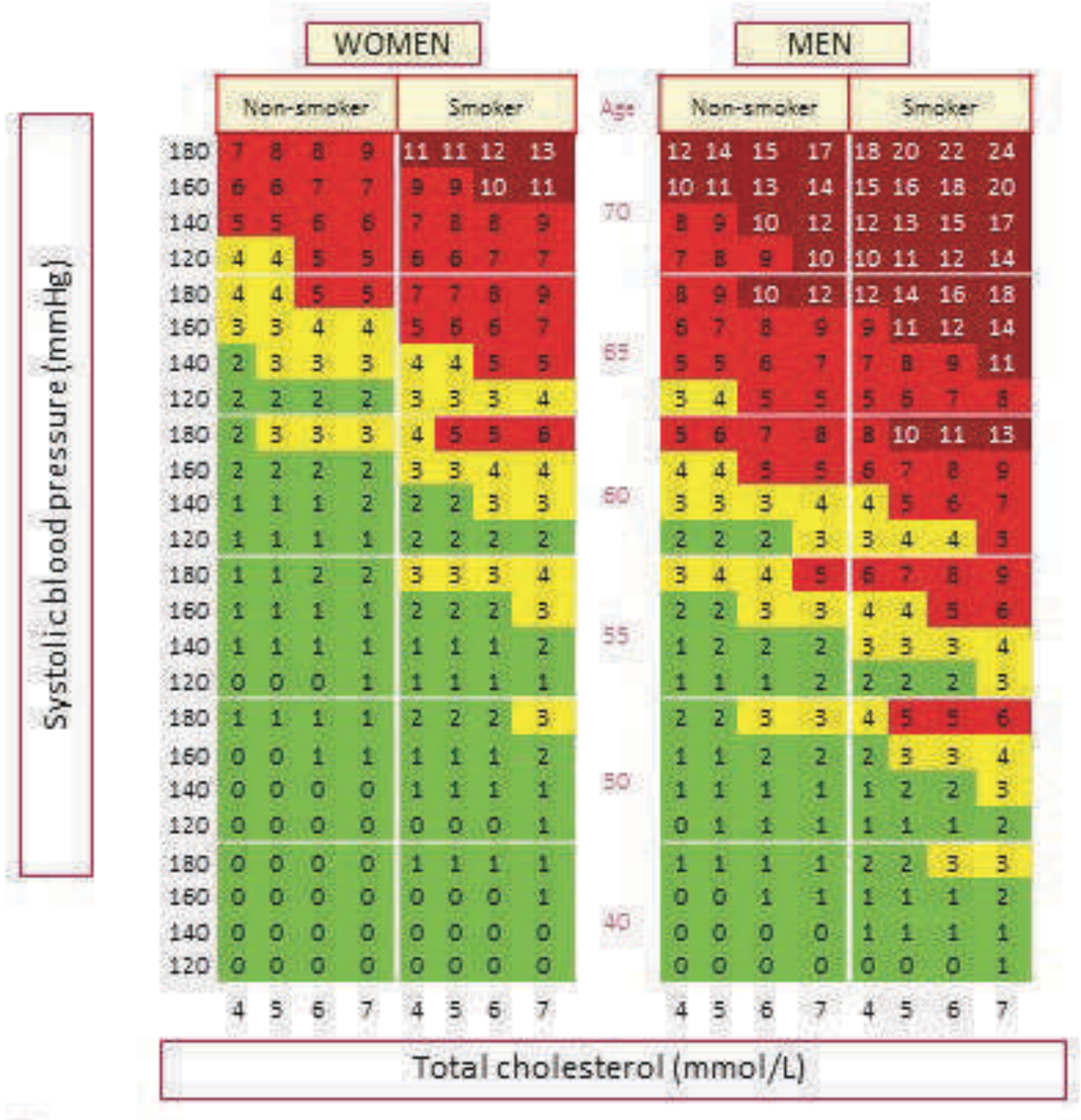
www.escardio.org/guidelines

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

- RR 152/87mmHg
- Hypercholesterinämie

- RR 152/87mmHg
- Raucher

- RR 152/87mmHg
- Keine RF



- 62a männl. Patient
- Raucher
- RR 150/90mmHg

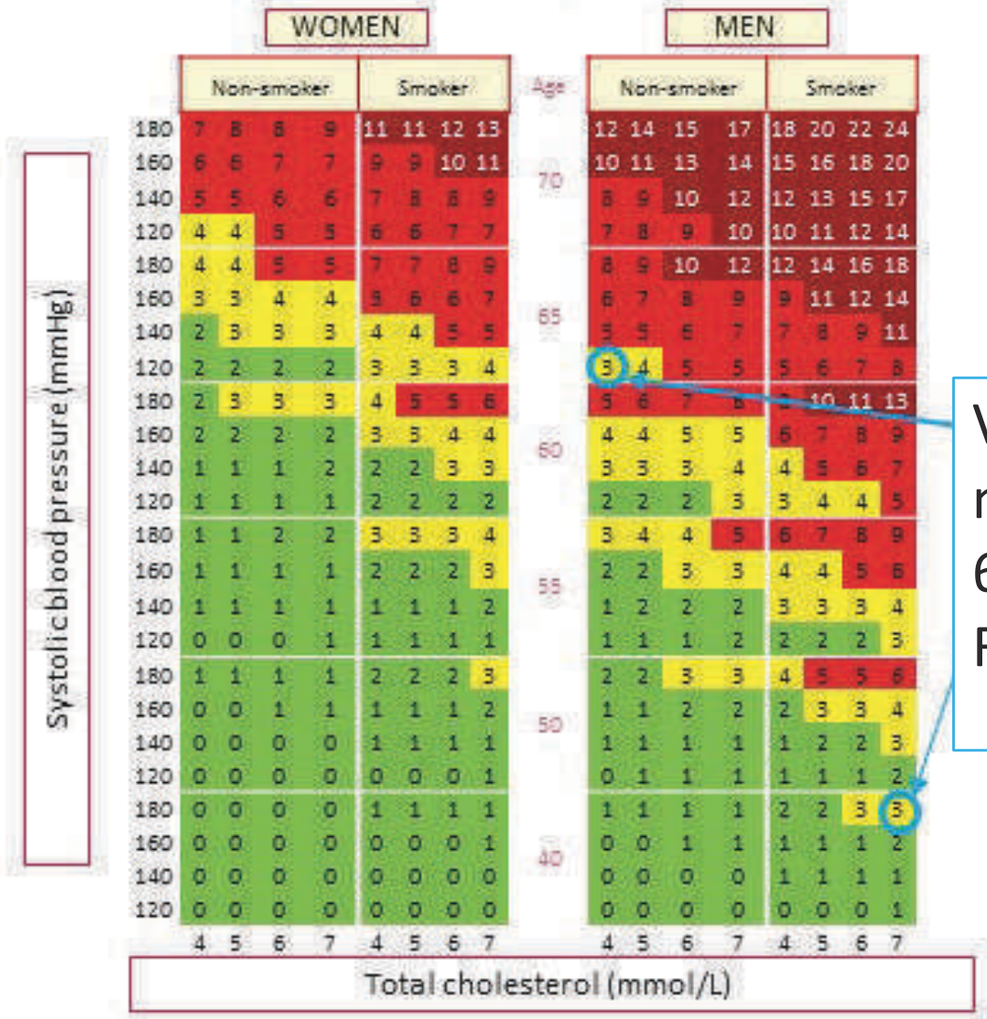
41a Frau
Hypercholesterinämie 235mg/dl (6mmol)

75a Frau
Keine Risikofaktoren

10 Jahres Risiko für fatales Ereignis

10-year risk of fatal CVD

Low-risk regions of Europe (age interactions included)



Vergleich 40 jähriger Raucher mit Risikofaktoren vs
65 Jähriger mit optimierter Risikosituation

©ESC

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

Genetik und Lebensstil

- Geschlecht
- Alter
- Nikotin
- Gesamtcholesterin und HDL-C
- Harnsäure
- Diabetes
- Adipositas
- Ernährung, Salzkonsum
- Pos FA für frühes CV Ereignis (♂<55a ♀<65a)
- Pos FA early onset Hypertonie
- Frühe Menopause
- Bewegungsmangel
- Psychosoziale und sozioökonomische Faktoren (Bildung, Einkommen, Depressio, Gesundheitsversorgung,...)
- Herzfrequenz (Ruhe >80)

CV RISIKO

Asymptomatischer Endorganschaden (HMOD)

- Arterielle Gefäßsteifigkeit (Pulsdruck≥60mmHg; c-f PWV >10m/s)
- LVH (EKG:Skolow-Lyon >35mm; Echo: LVMI ♂>50g/m♀>47g/m)
- Imaging: Stenose>50% coronar od. Carotis
- Mikroalbuminurie (30-300mg/24h)
- Niereninsuffizienz eGFR 30-59ml/min/1.73m2
- ABI <0.9
- Fortgeschrittene Retinopathie

Symptomatischer Endorganschaden

- Ischämischer oder hämorrhagischer Insult, TIA
- MCI, KHK, AP, PCI und Stenting, CABG
- Herzinsuffizienz systolisch oder diastolisch
- PAVK
- VHFA
- Niereninsuffizienz eGRF <30

- RR 152/87mmHg
- Hypercholesterinämie
- Diabetes mellitus
- Proteinurie

- RR 152/87mmHg
- Raucher
- Z. n. LAD Stent

- RR 152/87mmHg
- Keine RF

CV Risiko

Hypertension disease staging	Other risk factors, HMOD, or disease	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 or DBP ≥110
Stage 1 (uncomplicated)	No other risk factors	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 (asymptomatic disease)	HMOD, CKD grade 3, or diabetes mellitus without organ damage	Moderate to high risk	High risk	High risk	High to very high risk
Stage 3 (established disease)	Established CVD, CKD grade ≥4, or diabetes mellitus with organ damage	Very high risk	Very high risk	Very high risk	Very high risk

©ESC/ESH 2018

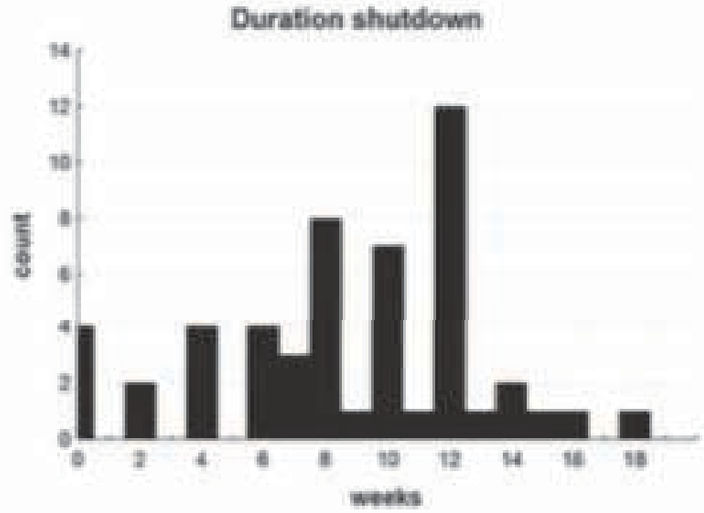
CV Risiko

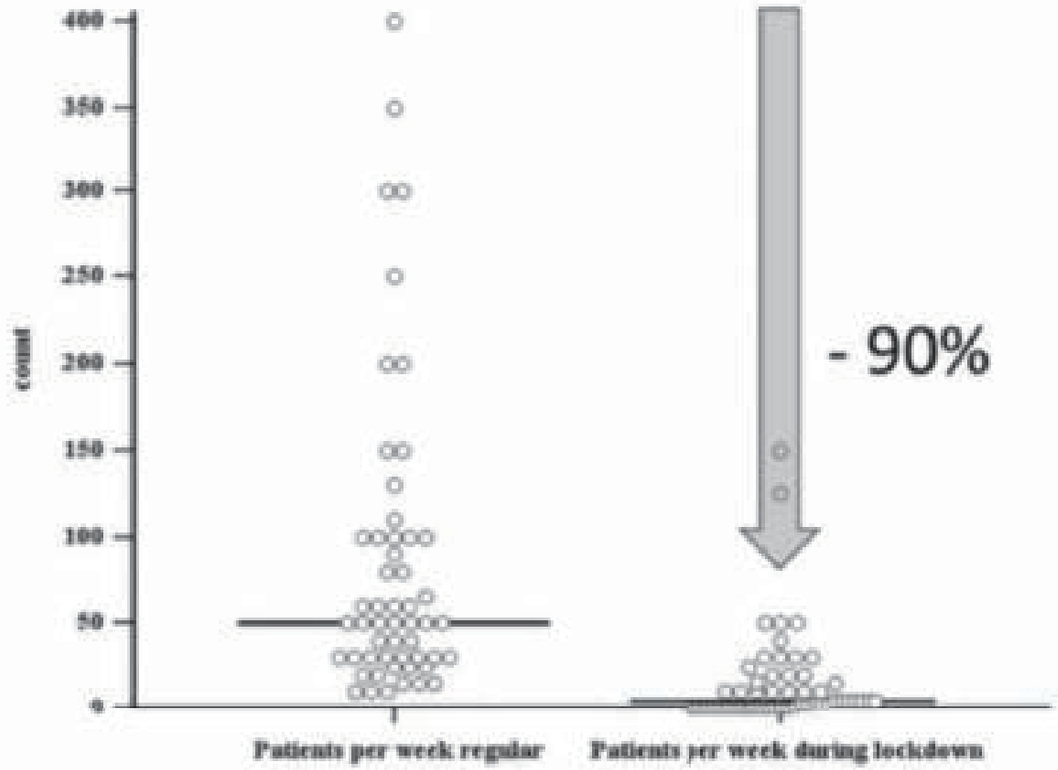
Very high risk	**People with any of the following:**
	Documented CVD, either clinical or unequivocal on imaging • **Clinical CVD** includes acute myocardial infarction, acute coronary syndrome, coronary or other arterial revascularization, stroke, TIA, aortic aneurysm, and PAD • **Unequivocal documented CVD on imaging** includes significant plaque (i.e. ≥ 50% stenosis) on angiography or ultrasound; it does not include increase in carotid intima-media thickness • **Diabetes mellitus with target organ damage**, e.g. proteinuria or a with a major risk factor such as grade 3 hypertension or hypercholesterolaemia • **Severe CKD** (eGFR < 30 mL/min/1.73 m^2) • **A calculated 10 year SCORE of ≥ 10%**
High risk	**People with any of the following:** • **Marked elevation of a single risk factor**, particularly cholesterol > 8 mmol/L (> 310 mg/dL), e.g. familial hypercholesterolaemia or grade 3 hypertension (BP ≥ 180/110 mmHg) • **Most other people with diabetes mellitus** (except some young people with type 1 diabetes mellitus and without major risk factors, who may be at moderate-risk)
	Hypertensive LVH
	Moderate CKD (eGFR 30-59 mL/min/1.73 m^2)
	A calculated 10 year SCORE of 5-10 %
Moderate risk	**People with:** • **A calculated 10 year SCORE of ≥ 1 to <5%** • **Grade 2 hypertension** • **Many middle-aged people belong to this category**
Low risk	**People with:** • **A calculated 10 year SCORE of < 1%**

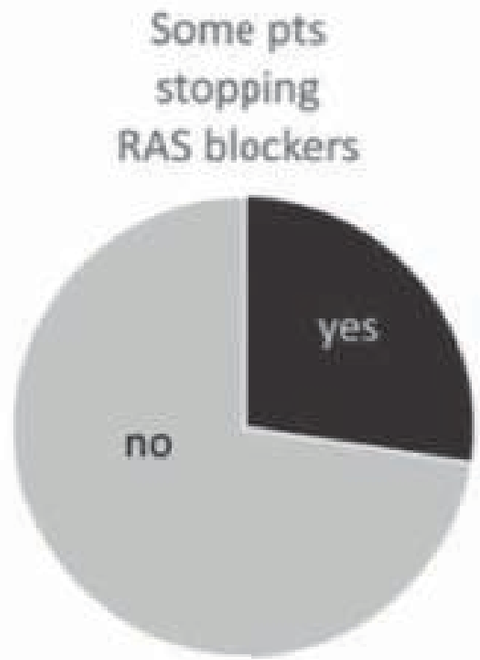
Wolters Kluwer

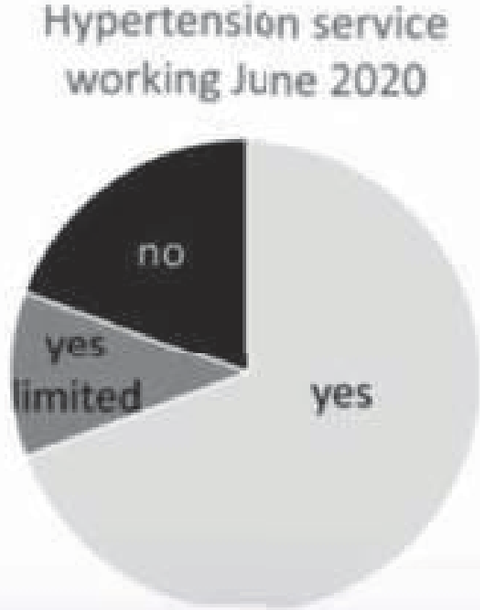
2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018 and J Hypertens

Corona-Virus 2019: 52 Excellence Centers



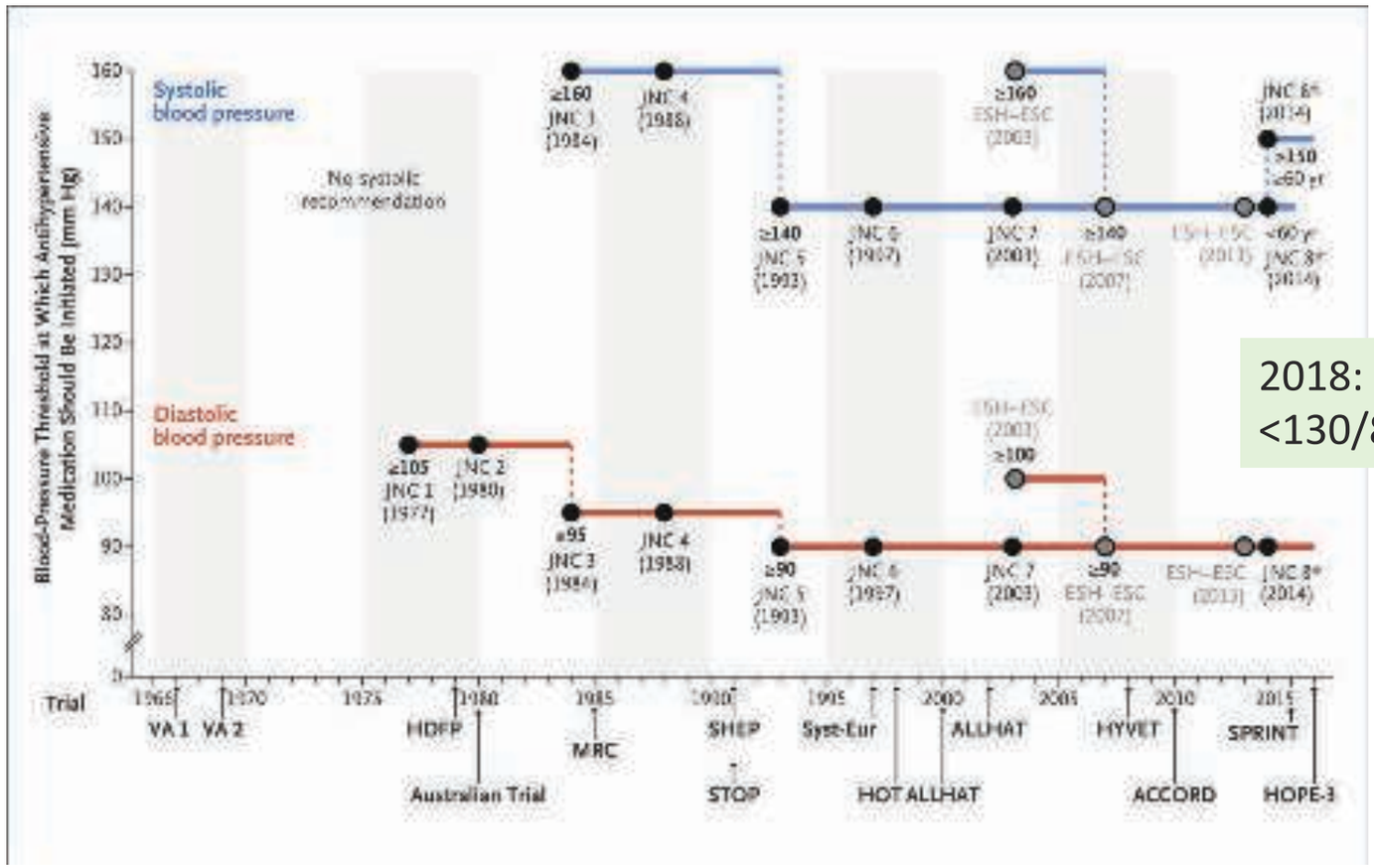






Weber et al. J Hypertens 2021,39:190-195

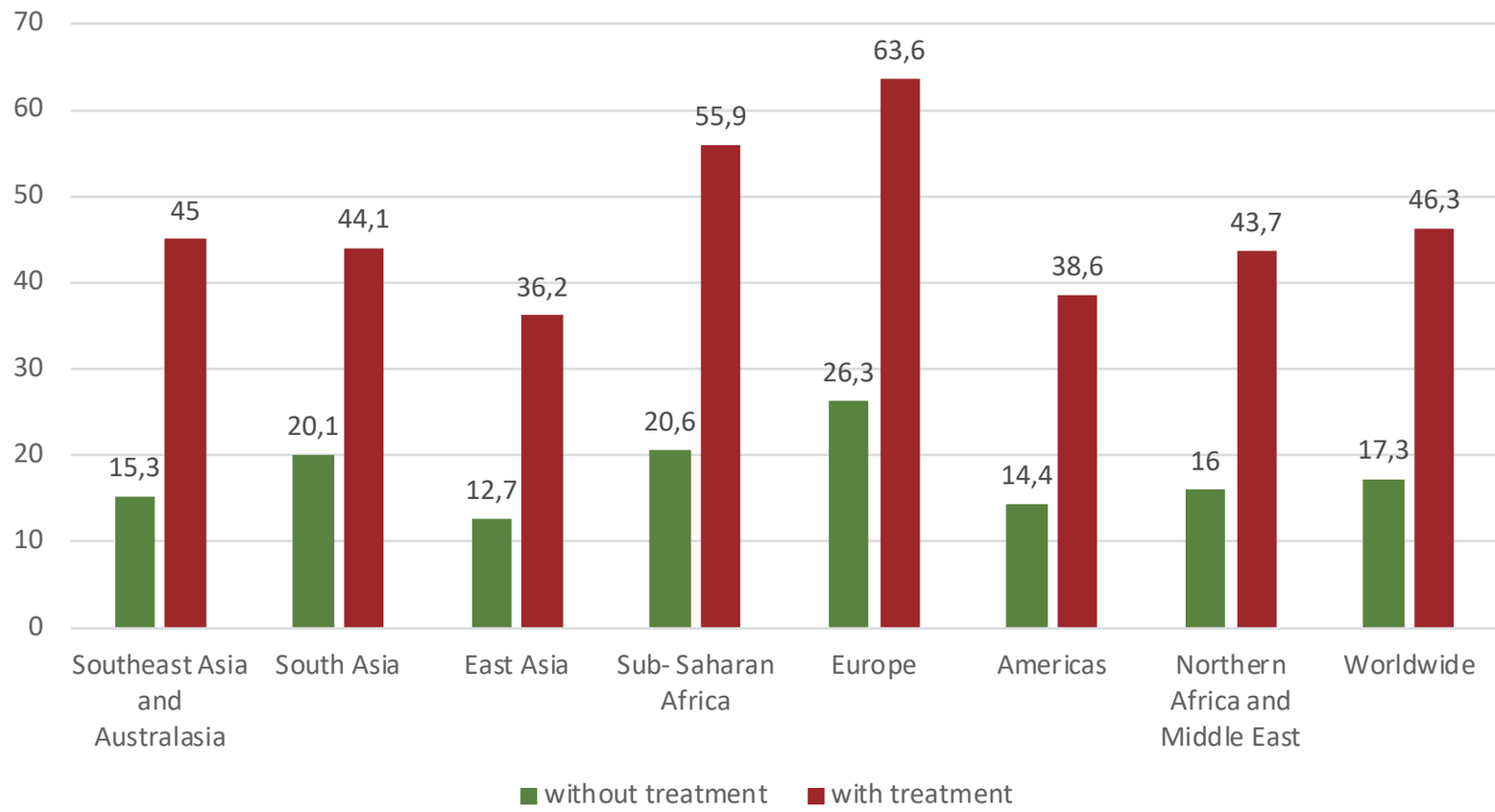
Zielwerte



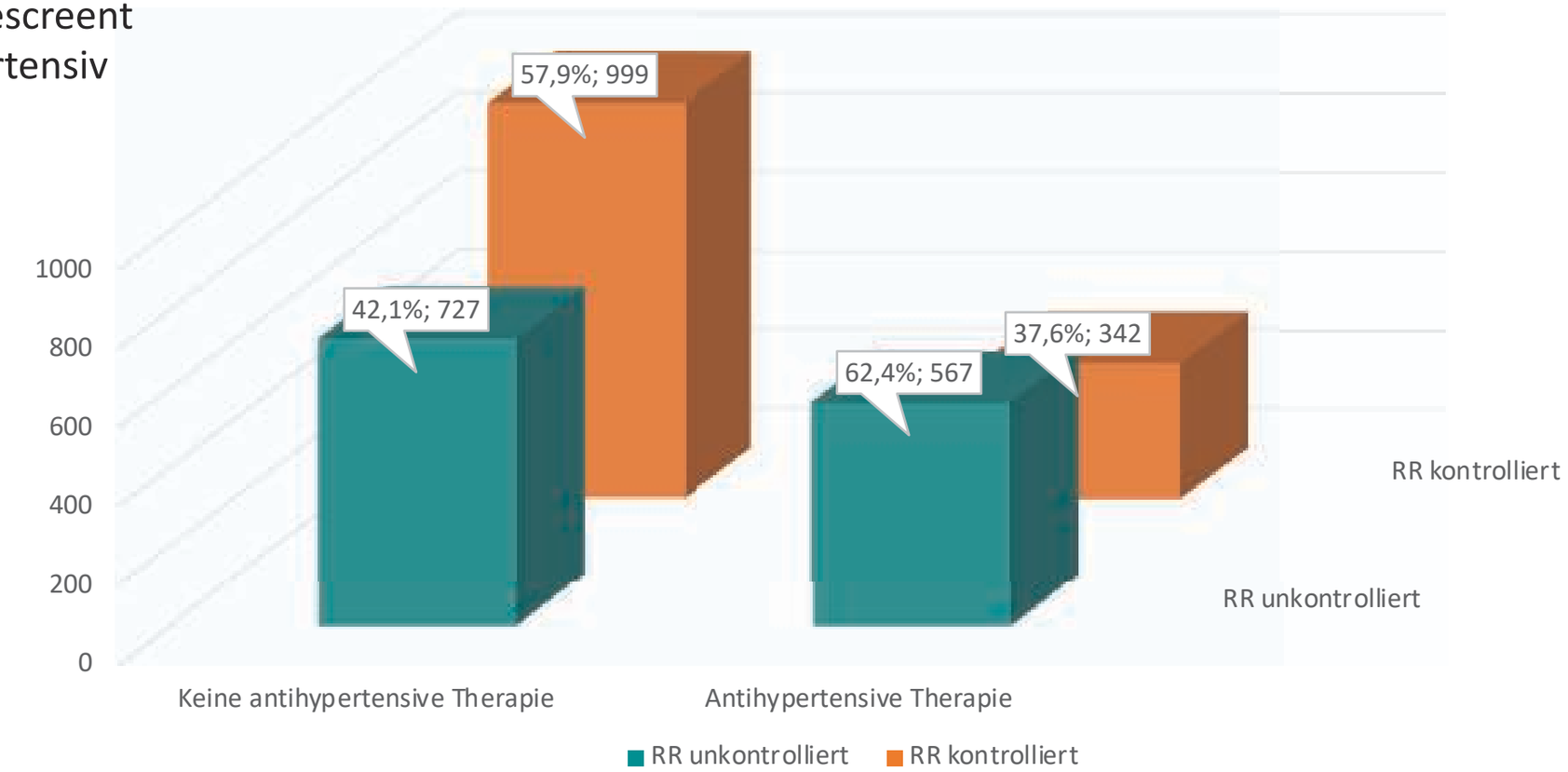
2018:
<130/80mmHg???

Pfeffer MA, Mc Murray JJ. NEJM 2016:375:1756-1766

RR>140/90 ohne und mit Therapie

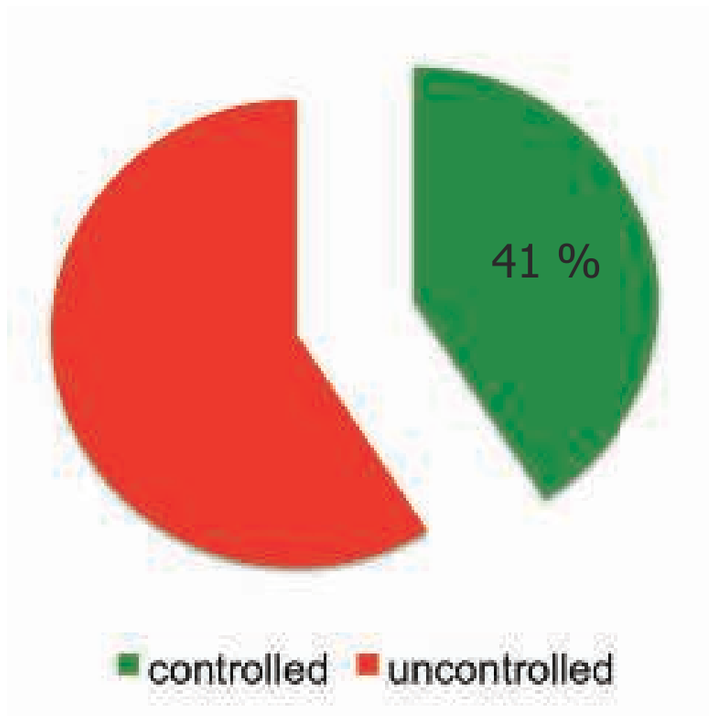


2711 Pt gescreent
62% hypertensiv

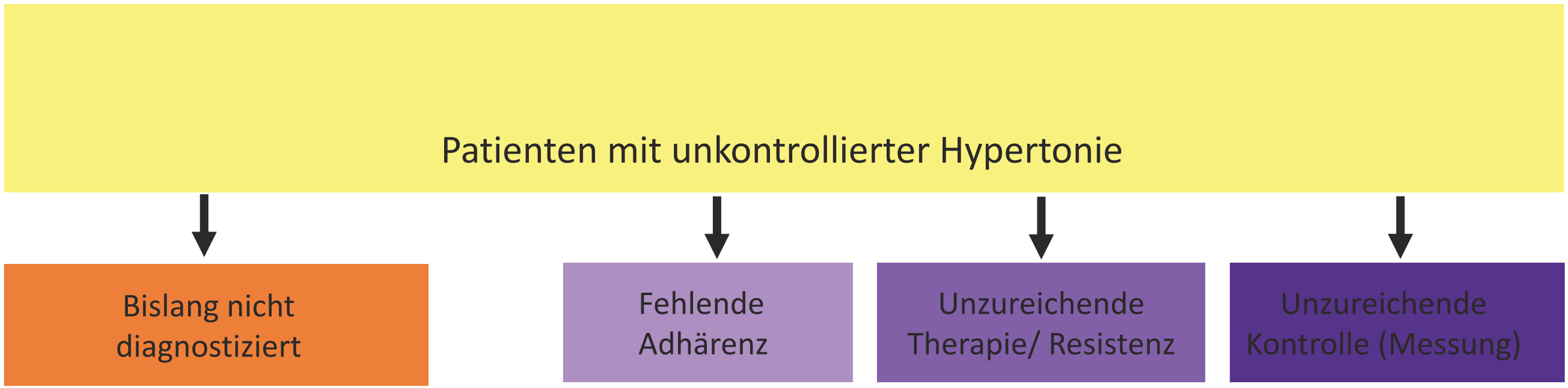


Perl et al. EHJ Suppl 2019 (MMM2017)

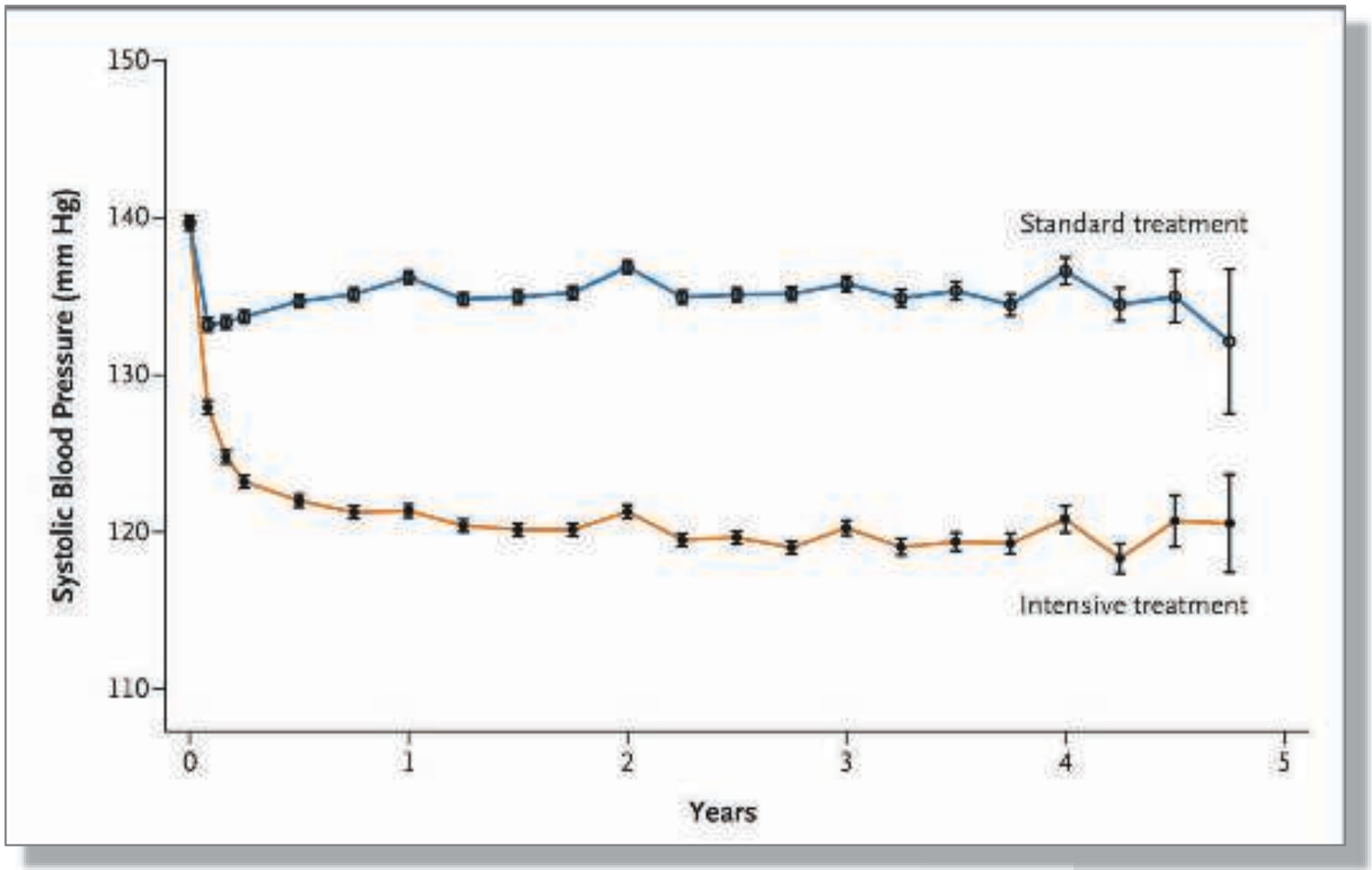
> 4000 Hypertoniker in 150 Apotheken



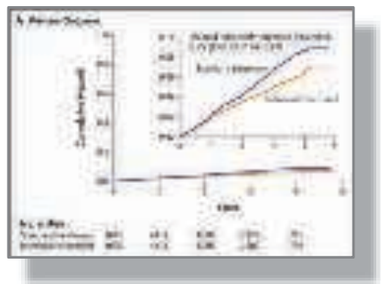
Rohla et al, J Hypertens 2016

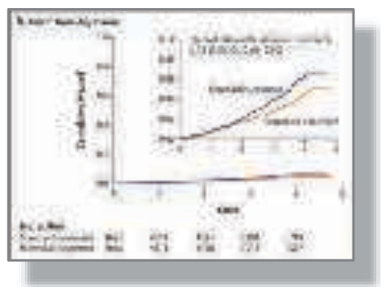

Patienten mit unkontrollierter Hypertonie
Bislang nicht diagnostiziert
Fehlende Adhärenz
Unzureichende Therapie/ Resistenz
Unzureichende Kontrolle (Messung)

SPRINT: Systolischer Blutdruck



mean syst. RR nach 1 Jahr:
136.2 vs 121.4 mmHg



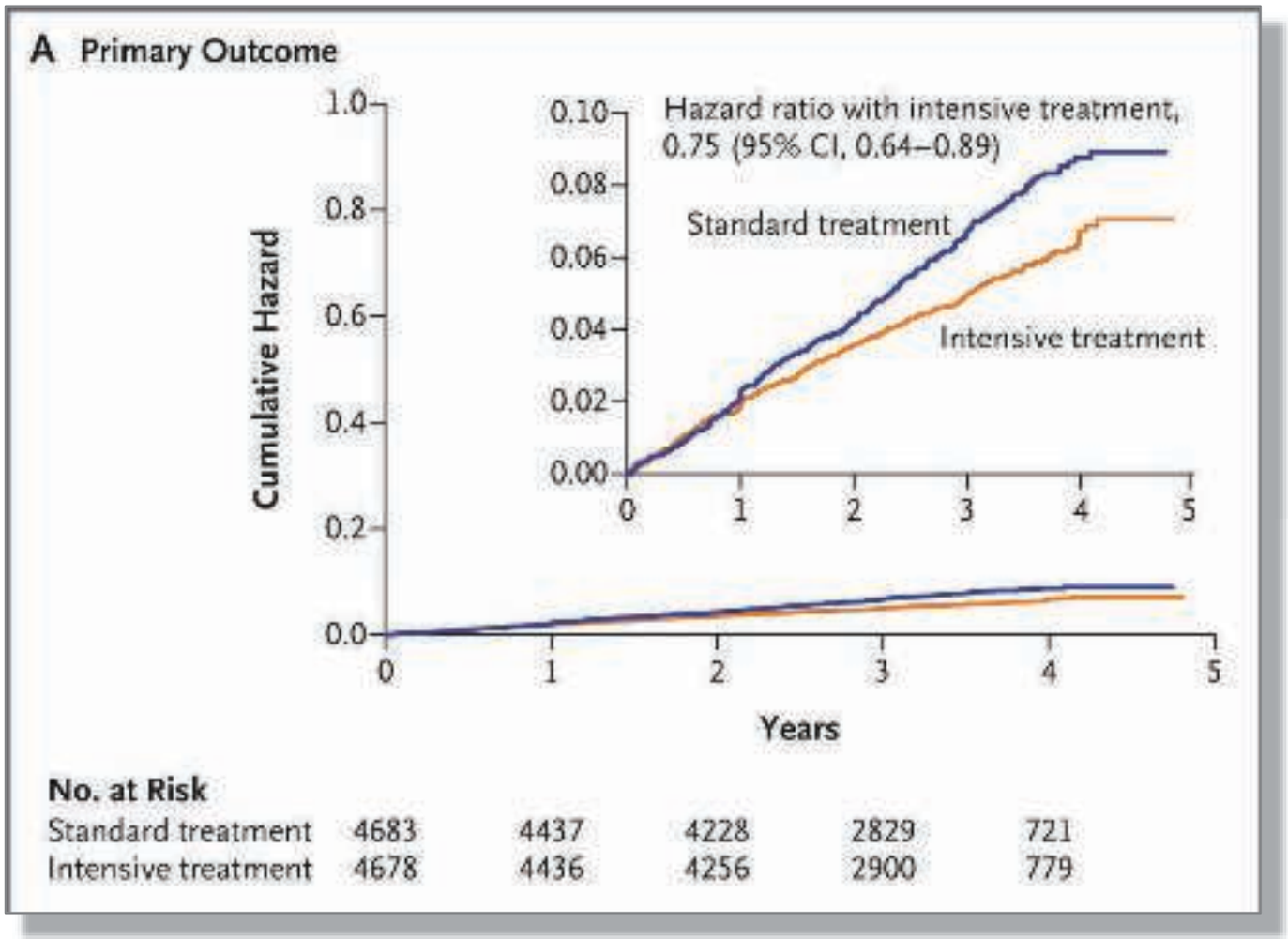


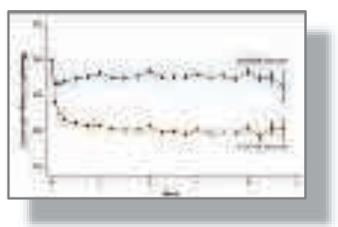
SPRINT Research Group. N Engl J Med 2015;373:2103-2116

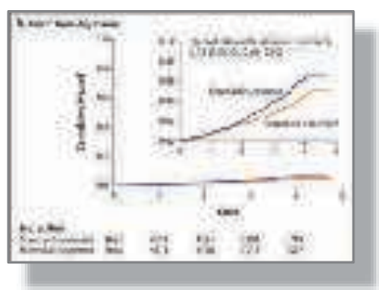
SPRINT: Systolischer Blutdruck

Vorzeitiger Abbruch
nach 3.26 J Follow up

Prim. Outcome p<0.001



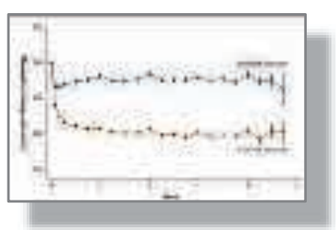


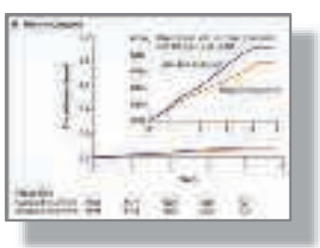


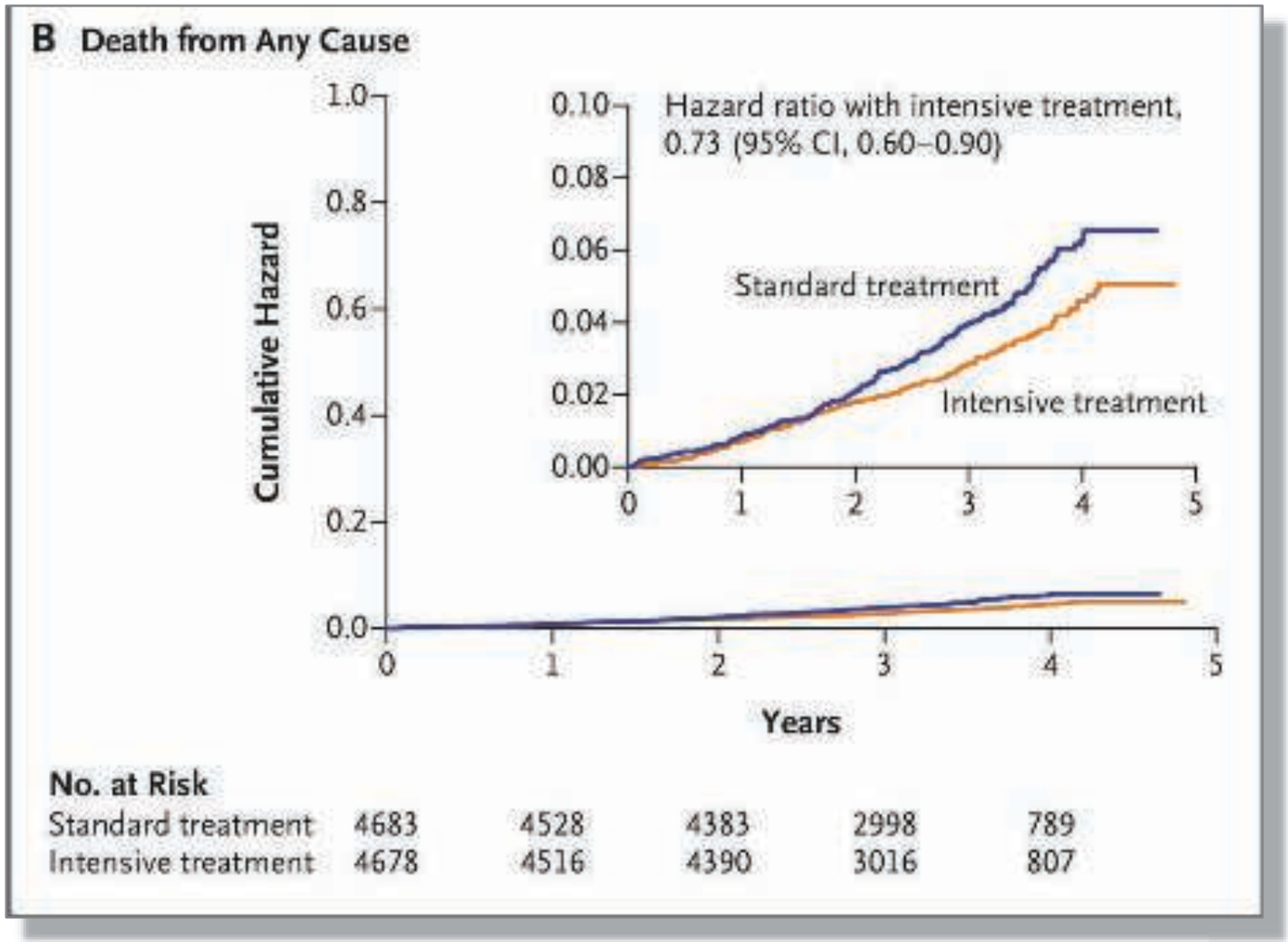
SPRINT Research Group. N Engl J Med 2015;373:2103-2116

SPRINT: Systolischer Blutdruck

Mortalitätsreduktion p=0.003







The SPRINT Research Group. N Engl J Med 2015;373:2103-2116

AHA/ACC

	Therapieeinleitung	**Therapie - Ziel**
bestehende CV Erkrankung	≥ 130 u/o ≥ 80	<130/80
CV 10-Jahres Risiko ≥ 10%	≥ 130 u/o ≥ 80	<130/80
CV 10-Jahres Risiko < 10%	≥ 140/90	<130/80
Alter ≥ 65 nicht pflegebedürftig	≥ 130 systolisch	<130 syst
DM, CKD, HI, KHK, PAVK	≥130/80	<130/80

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke[a]/TIA	
18–65 years	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	**Target to <140 to 130** *if tolerated*	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	70–79
65–79 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
≥80 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
Office DBP treatment target range (mmHg)	70–79	70–79	70–79	70–79	70–79	

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke[a]/TIA	
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65–79 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
≥80 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
Office DBP treatment target range (mmHg)	70–79	70–79	70–79	70–79	70–79	

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke[a]/TIA	
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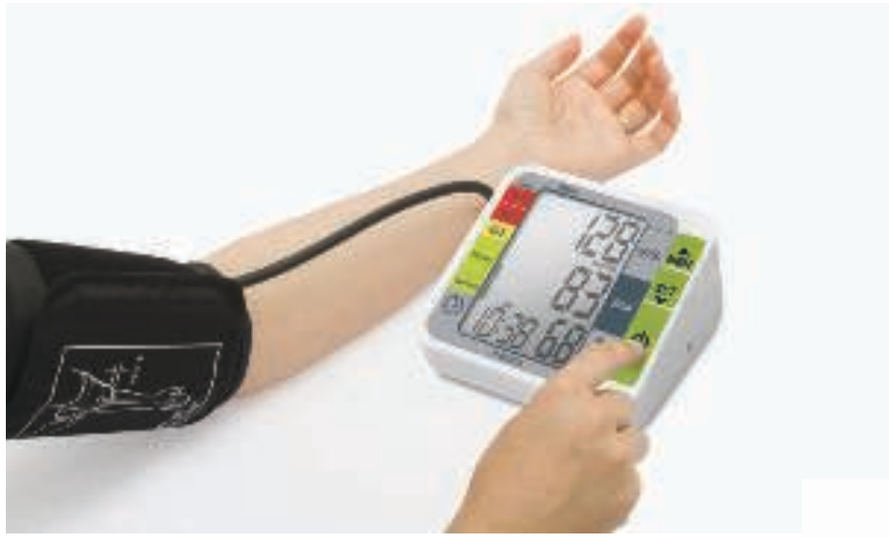
65–79 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
≥80 years[b]	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	**Target to 130–139** *if tolerated*	70–79
Office DBP treatment target range (mmHg)	70–79	70–79	70–79	70–79	70–79	

Zielwerte bei unkomplizierter Hypertonie

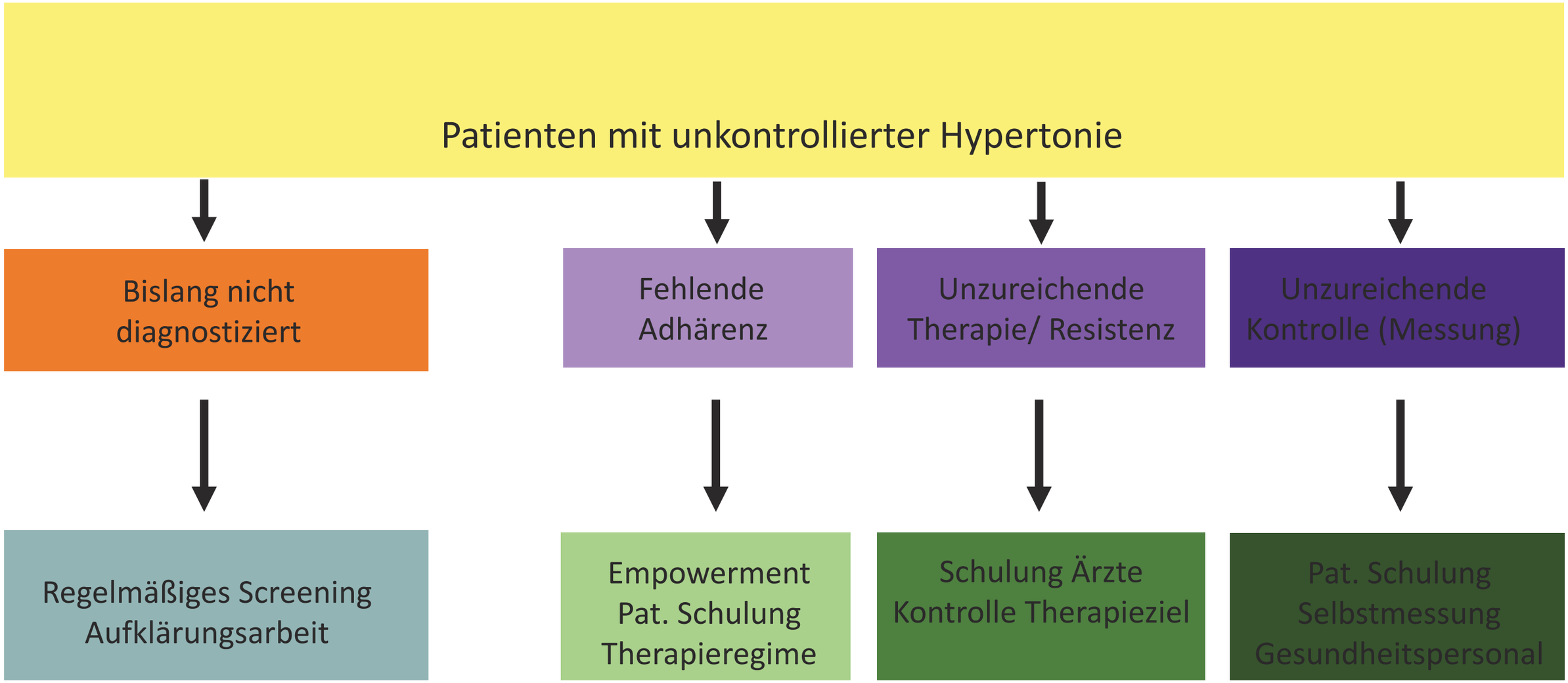
Art der Blutdruckmessung	*Zielwert mm Hg*
OFFICE BLUTDRUCK	
Systolischer Blutdruck	1. Ziel: 130 2. Ziel: 120-129 (bei guter Verträglichkeit)
Diastolischer Blutdruck	70-79
UNBEOBACHTETE AUTOMATISCHE OFFICE-MESSUNG	
Mittelwert dreier Messungen	120-125 / 70
24-STUNDEN BLUTDRUCK MONITORING	
24-Stunden Mittelwert	125 / 70
Tages-Mittelwert	125-129 / 70-75
Nacht-Mittelwert	115-120 / 65
BLUTDRUCK SELBSTMESSUNG	
Mittelwert	125-129 / 70-75

Blutdruckkonsens Österreich 2019

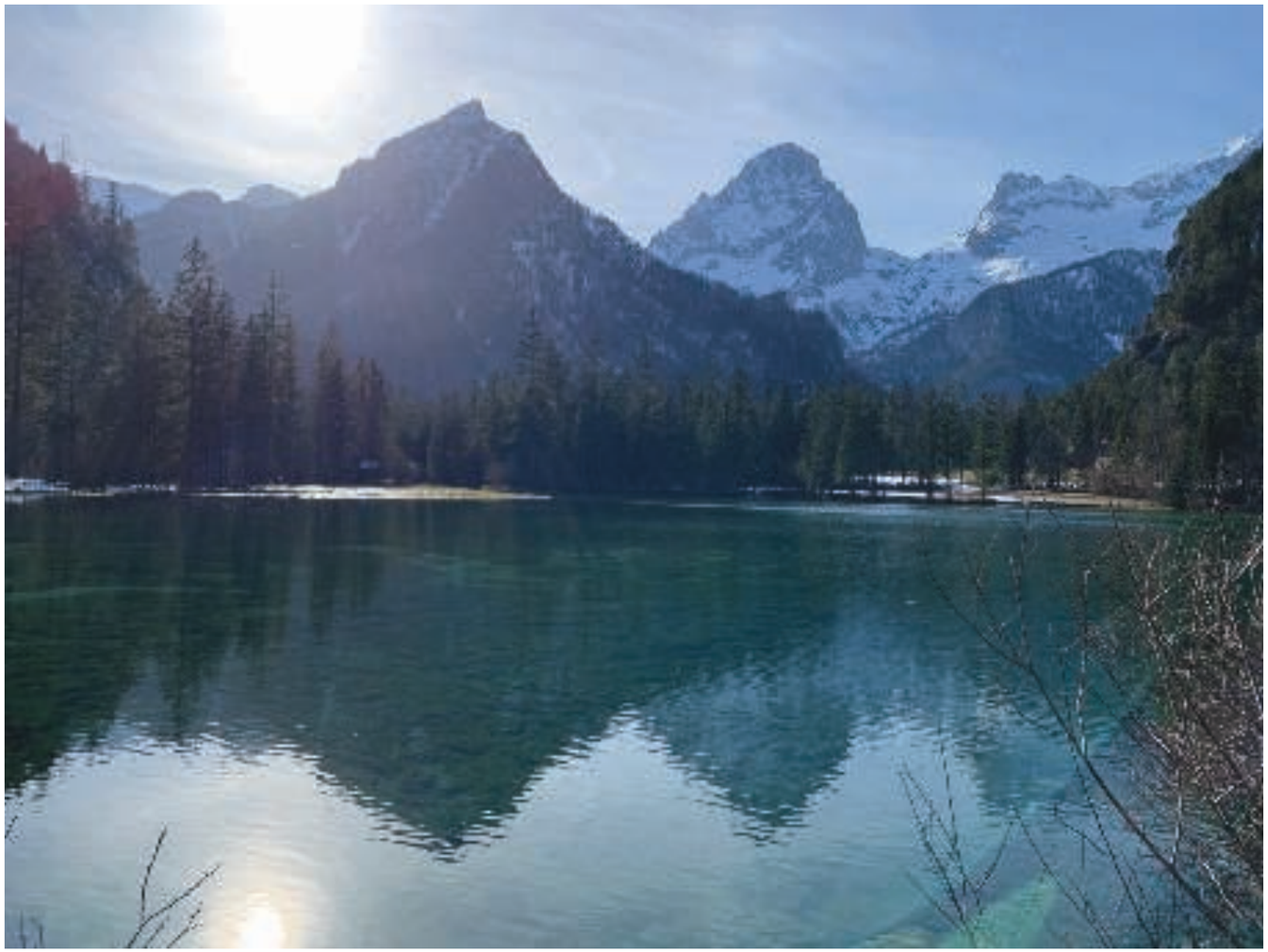

120
83


625


PDF


Patienten mit unkontrollierter Hypertonie
Bislang nicht diagnostiziert
Fehlende Adhärenz
Unzureichende Therapie/ Resistenz
Unzureichende Kontrolle (Messung)
Regelmäßiges Screening Aufklärungsarbeit
Empowerment Pat. Schulung Therapieregime
Schulung Ärzte Kontrolle Therapieziel
Pat. Schulung Selbstmessung Gesundheitspersonal

Med Uni Graz



PRO
Kompetenzzentrum
für Gesundheit

Universitäres Herzzentrum Graz

Österreichische Gesellschaft für
Hypertensiologie

PRODOC+
ÄRZTEZENTRUM
GRAZ-EGGENBERG

Vorhofflimmern und arterielle Hypertonie

Prim. Univ. Prof. Dr. Michael M. Hirschl

Abteilung für Innere Medizin

Landesklinikum Zwettl

Vorhofflimmern

- Assoziation zwischen arterieller Hypertonie und dem Auftreten von VHF
- Epidemiologie des VHF
- Paroxysmal/Persistierend/Permanent
- Komplikationen des VHF
- Diagnostik
- Therapie (allgemein)
  - Antiarrhythmische Therapie
  - Antikoagulation
- Therapie in spezifischen klinischen Situationen
  - Herzinsuffizienz
  - Akutes koronares Syndrom mit/ohne PCI

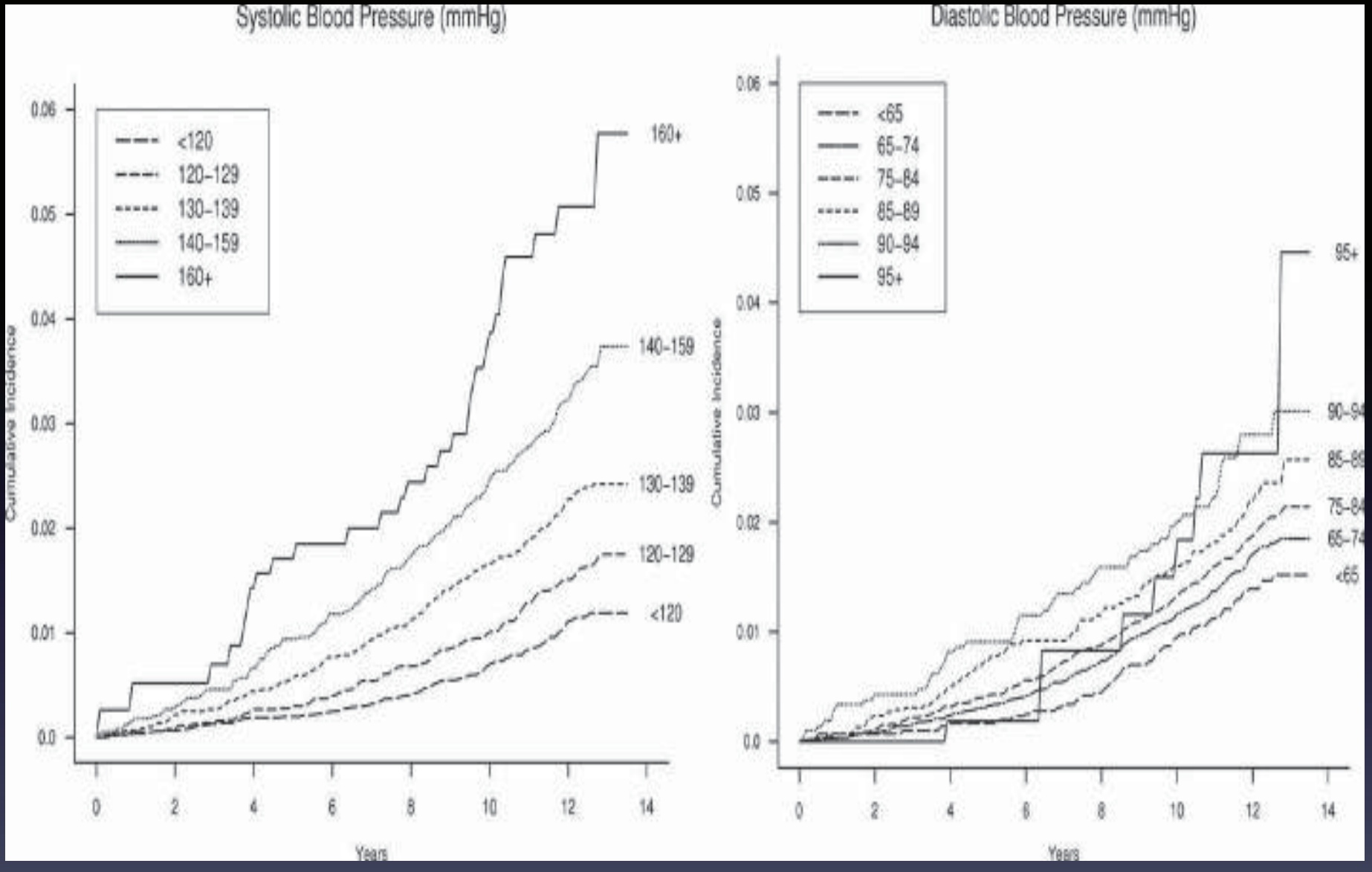
Epidemiologische Daten hinsichtlich Hypertonie und AF

- Arterielle Hypertonie findet sich bei 60% aller Patienten mit Vorhofflimmern.
- Bei ca. 15% der Patienten ist die arterielle Hypertonie der alleinige Auslöser für das Vorhofflimmern.
- In den letzten 15 Jahren wurden eine Reihe von epidemiologischen Studien publiziert, die eine Assoziation zwischen der Höhe des Blutdruckes und dem Auftreten von Vorhofflimmern zeigten.

Risikofaktoren bei AF

	First detected (*n*=978)	Paroxysmal (*n*=1517)	Persistent (*n*=1167)	Permanent (*n*=1541)
Age ≥75 years	255 (26)	325 (21)	271 (23)	591 (38)
Heart failure or LVEF ≤35%	291 (30)	415 (28)	454 (39)	796 (52)
Hypertension	620 (63)	942 (62)	772 (66)	984 (63)
Mitral stenosis	34 (4)	37 (3)	62 (5)	188 (13)
Valve surgery	27 (3)	75 (5)	72 (6)	172 (11)
Stroke/TIA	70 (7)	143 (10)	101 (9)	228 (15)
Age 60–74 years, and diabetes or CAD	212 (22)	292 (19)	230 (20)	334 (22)
At least one of the above	808 (83)	1200 (79)	1010 (87)	1447 (94)

European Heart Journal (2005) 26, 2422–2434



David Conen et al. Circulation. 2009;119:2146-2152

American Heart Association.

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Risiko für AF in Abhängigkeit vom Blutdruck

Variables	Hazard Ratio (95 % CI)			
	Q1	Q2	Q3	Q4
Systolic BP (mm Hg)	88–116	118–126	128–138	140–220
Multiple adjusted*	1.00	1.02 (0.69–1.53)	1.51 (1.05–2.20)	1.62 (1.10–2.39)
Diastolic BP (mm Hg)	54–78	80–86	88–92	94–130
Multiple adjusted*	1.00	1.69 (1.15–2.53)	1.81 (1.20–2.76)	1.94 (1.30–2.95)

Hypertension. 2012; 59: 198-204

Cardiovascular and other conditions independently associated with atrial fibrillation (1)

Characteristic/comorbidity	Association with AF
Genetic predisposition (based on multiple common gene variants associated with AF)	HR range 0.4–3.2
Older age 50–59 years 60–69 years 70–79 years 80–89 years	HR: 1.00 (reference) 4.98 (95% CI 3.49–7.10) 7.35 (95% CI 5.28–10.2) 9.33 (95% CI 6.68–13.0)
Hypertension (treated) vs. none	HR 1.32 (95% CI 1.08–1.60)
Heart failure vs. none	HR 1.43 (95% CI 0.85–2.40)
Valvular heart disease vs. none	RR 2.42 (95% CI 1.62–3.60)
Myocardial infarction vs. none	HR 1.46 (95% CI 1.07–1.98)
Thyroid dysfunction Hypothyroidism Subclinical hyperthyroidism Overt hyperthyroidism	(reference: euthyroid) HR 1.23 (95% CI 0.77–1.97) RR 1.31 (95% CI 1.19–1.44) RR 1.42 (95% CI 1.22–1.63)
Obesity (body mass index) None (<25 kg/m²) Overweight (25–30 kg/m²) Obese (≥31 kg/m²)	HR: 1.00 (reference) 1.13 (95% CI 0.87–1.46) 1.37 (95% CI 1.05–1.78)
Diabetes mellitus vs. none	HR 1.25 (95% CI 0.98–1.60)

HR = hazard ratio; RR = risk ratio

Continued on next slide

European Heart Journal - doi:10.1093/eurheartj/ehw210

EUROPEAN SOCIETY OF CARDIOLOGY®

Cardiovascular and other conditions independently associated with atrial fibrillation (2)

Characteristic/comorbidity	Association with AF
Chronic obstructive pulmonary disease FEV1 ≥80% FEV1 60–80% FEV1 <60%	RR: 1.00 (reference) 1.28 (95% CI 0.79–2.06) 2.53 (95% CI 1.45–4.42)
Obstructive sleep apnoea vs. none	HR 2.18 (95% CI 1.34–3.54)
Chronic kidney disease None Stage 1 or 2 Stage 3 Stage 4 or 5	OR: 1.00 (reference) 2.67 (95% CI 2.04–3.48) 1.68 (95% CI 1.26–2.24) 3.52 (95% CI 1.73–7.15)
Smoking Never Former Current	HR: 1.00 (reference) 1.32 (95% CI 1.10–1.57) 2.05 (95% CI 1.71–2.47)
Alcohol consumption None 1–6 drinks/week 7–14 drinks/week 15–21 drinks/week >21 drinks/week	RR: 1.00 (reference) 1.01 (95% CI 0.94–1.09) 1.07 (95% CI 0.98–1.17) 1.14 (95% CI 1.01–1.28) 1.39 (95% CI 1.22–1.58)
Habitual vigorous exercise Non-exercisers <1 day/week 1–2 days/week 3–4 days/week 5–7 days/week	RR: 1.00 (reference) 0.90 (95% CI 0.68–1.20) 1.09 (95% CI 0.95–1.26) 1.04 (95% CI 0.91–1.19) 1.20 (95% CI 1.02–1.41)

European Heart Journal - doi:10.1093/eurheartj/ehw210

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Pathophysiologie von Vorhofflimmern und arterieller Hypertonie I.

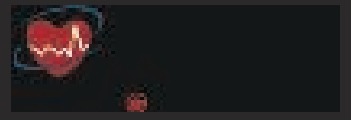
i. Arterielle Hypertonie führt zu
   i. Abnahme der LV-Compliance
   ii. Diastolischer Relaxationsstörung
   iii. Linksventrikulärer Hypertrophie
ii. Die linksventrikuläre Hypertrophie führt zu
   i. Einer Aktivierung des Sympathikus
   ii. Einer Aktivierung des RAAS

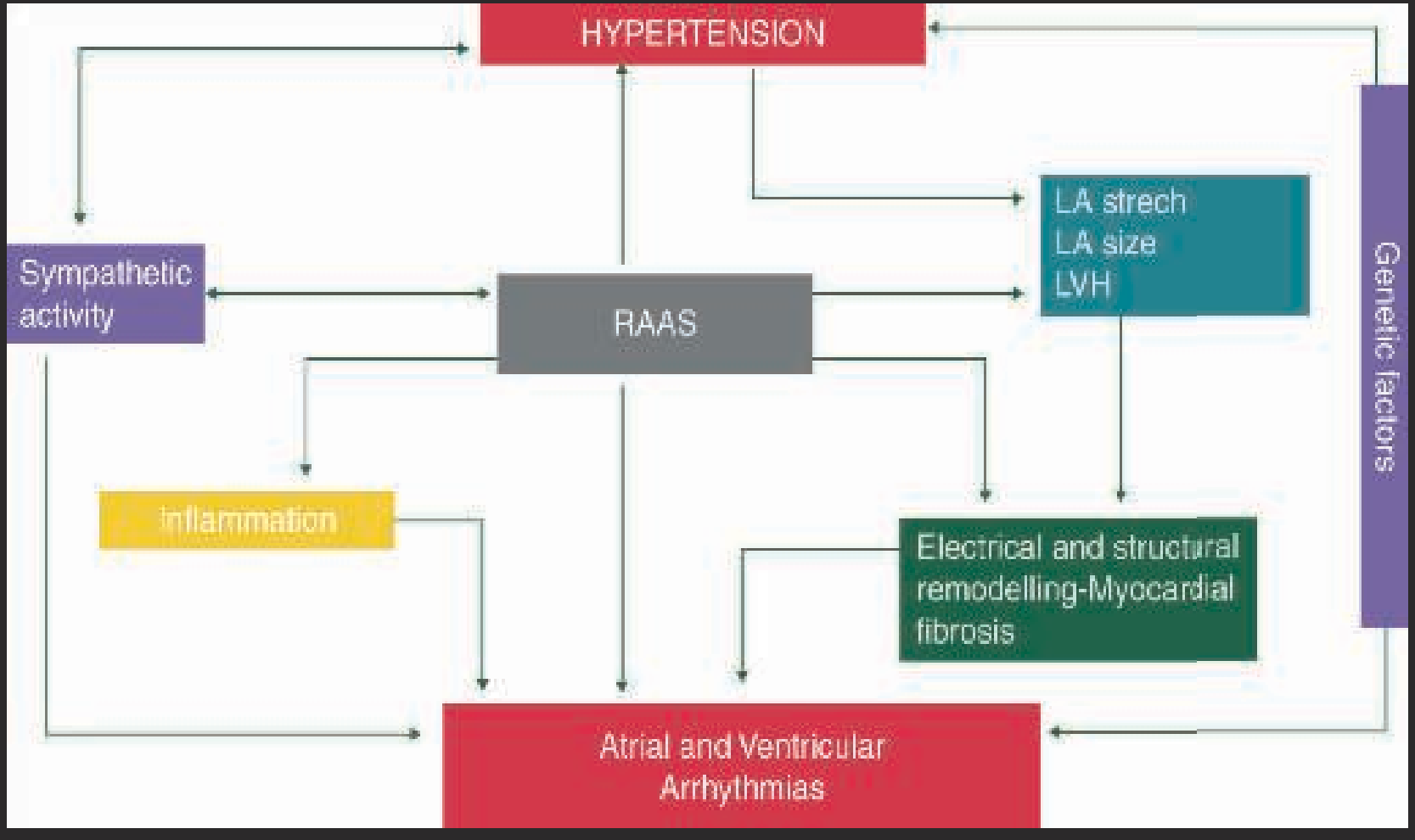
führt zu einer höheren Rate an AF

Pathophysiologie von Vorhofflimmern und arterieller Hypertonie II.

i. Arterielle Hypertonie führt auch zu
    i. einem atrial remodeling
        i. zu einem Anstieg von Enzymen (Metalloproteinasen), die Fibrose fördernd sind
        ii. Kardiale Fibrosierung führt zu elektrischen Störungen im Vorhof und zu einem erhöhten Risiko von AF

Figure 1 Mechanisms of arrhythmias in hypertension. LA, left atrium; LVH, left ventricular hypertrophy; RAAS, ...





EP Europace, Volume 19, Issue 6, June 2017, Pages 891–911, https://doi.org/10.1093/europace/eux091

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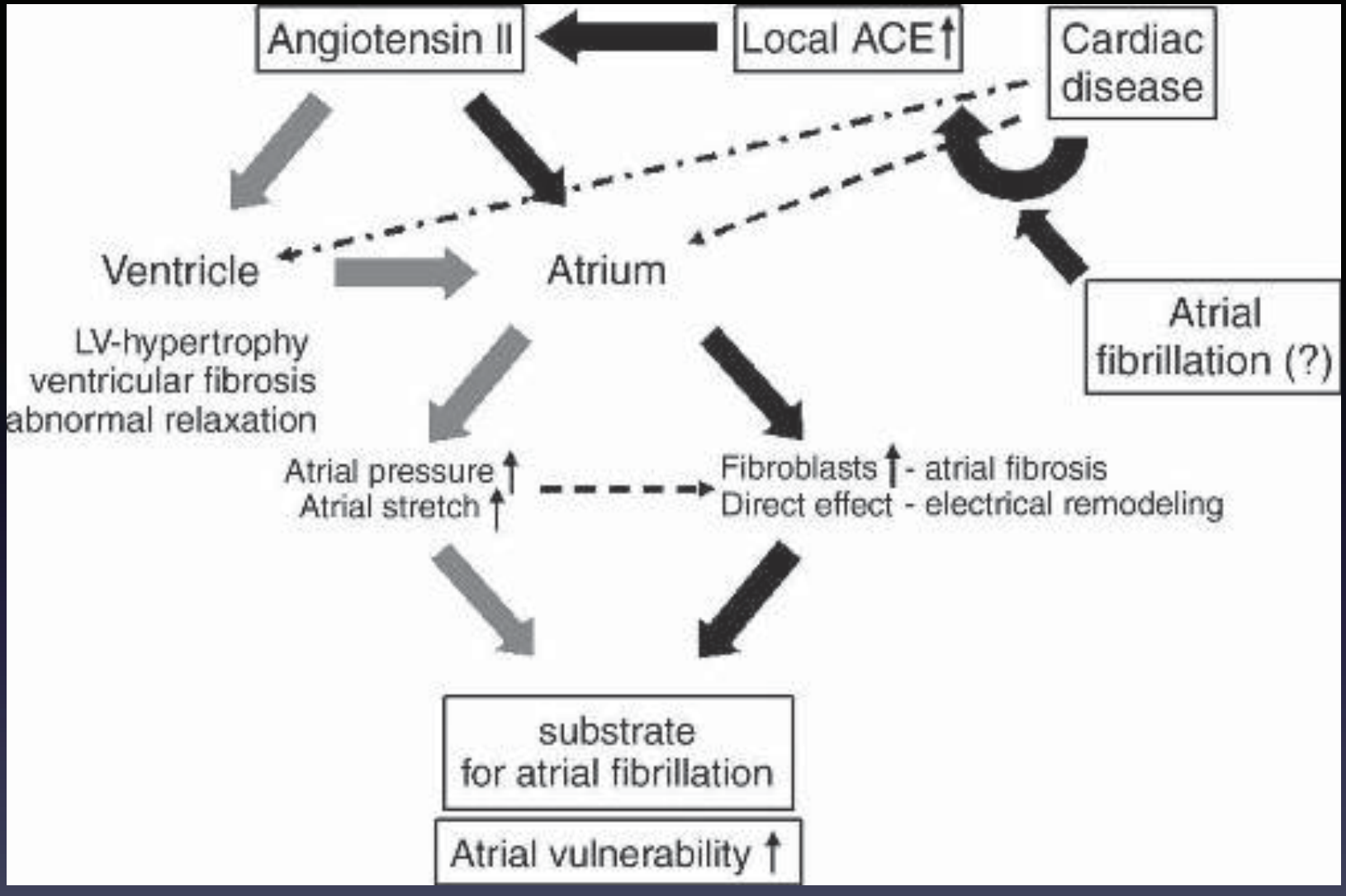
Figure 2 Direct and indirect atrial actions of angiotensin-II.



Joachim R. Ehrlich et al. Eur Heart J 2006;27:512-518

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European Heart Journal

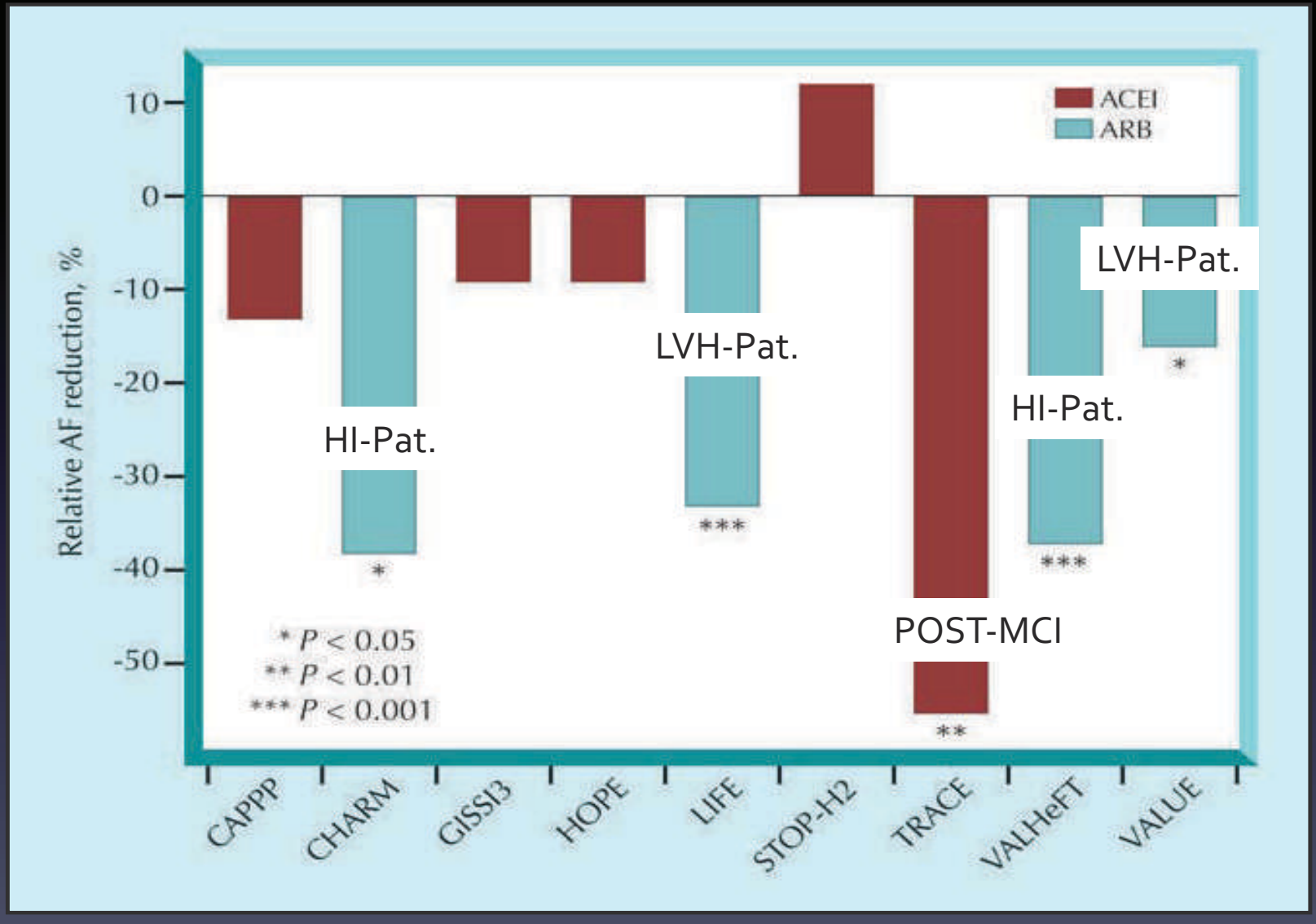
Effekte antihypertensiver Therapie auf das Auftreten von AF

- Kann durch eine antihypertensive Therapie das Auftreten von Vorhofflimmern verhindert werden?
  - Ist dieser Effekt auschließlich durch eine Reduktion der Blutdruckhöhe zu erzielen

ODER

  - Gibt es Unterschiede hinsichtlich der antihypertensive wirksamen Substanzklassen

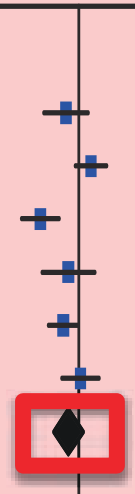
Current Hypertension Reports 2008, **10:**175–181



Prevention of Atrial Fibrillation by Renin-Angiotensin System Inhibition : A Meta-Analysis

Review: ARB/ACE for Prevention of AF
Comparison: 01 ARB/ACE for Prevention of AF
Outcome: 01 Atrial Fibrillation

When considering new-onset AF in patients with arterial hypertension, our analysis of 6 large clinical trials failed to demonstrate any overall effect.



Study or sub-category	Treatment n/N	Control n/N	OR (random) 95% CI	Weight %	OR (random) 95% CI
01 Hypertension Studies					
Hansson (CAPPP)	117/5456	135/5459		6.39	0.86 [0.67, 1.11]
Hansson (STOP-2)	200/2088	357/4215		6.97	1.14 [0.95, 1.37]
Wachtell (LIFE)	150/4298	221/4182		6.73	0.65 [0.52, 0.80]
Salehian (HOPE)	86/4291	91/4044		5.95	0.89 [0.66, 1.20]
Schmieder (VALUE)	252/6872	299/6888		7.06	0.84 [0.71, 1.00]
Yusuf (TRANSCEND)	182/2844	180/2857		6.72	1.02 [0.82, 1.26]
Subtotal (95% CI)	25849	27645		39.82	0.89 [0.75, 1.05]

Total events: 987 (Treatment), 1283 (Control)
Test for heterogeneity: $Chi^2 = 17.98$, df = 5 (P = 0.003), $I^2 = 72.2\%$
Test for overall effect: Z = 1.39 (P = 0.17)

Prevention of Atrial Fibrillation by Beta-Blockade : A Meta-Analysis

European Heart Journal (2007) 28, 457–462

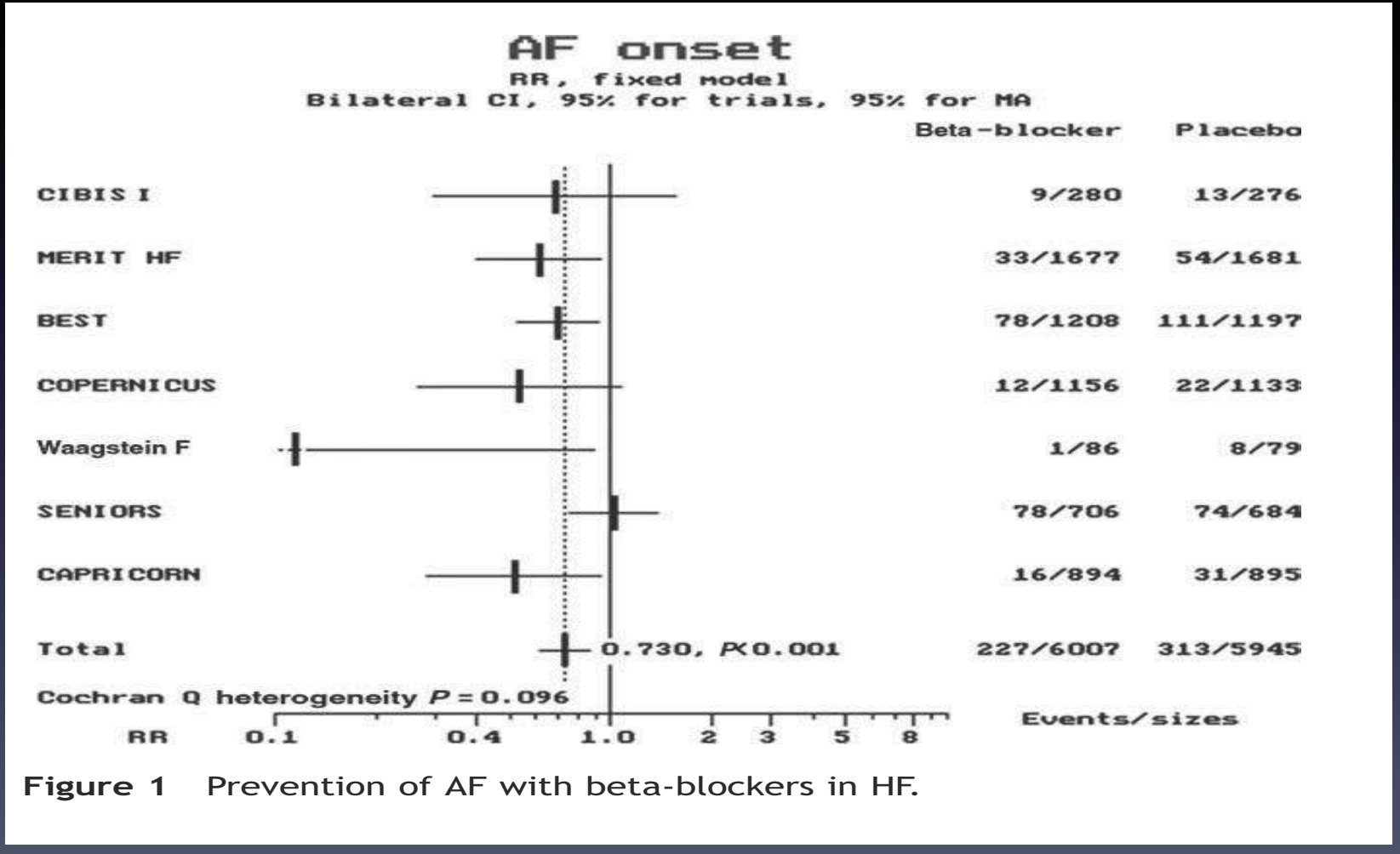
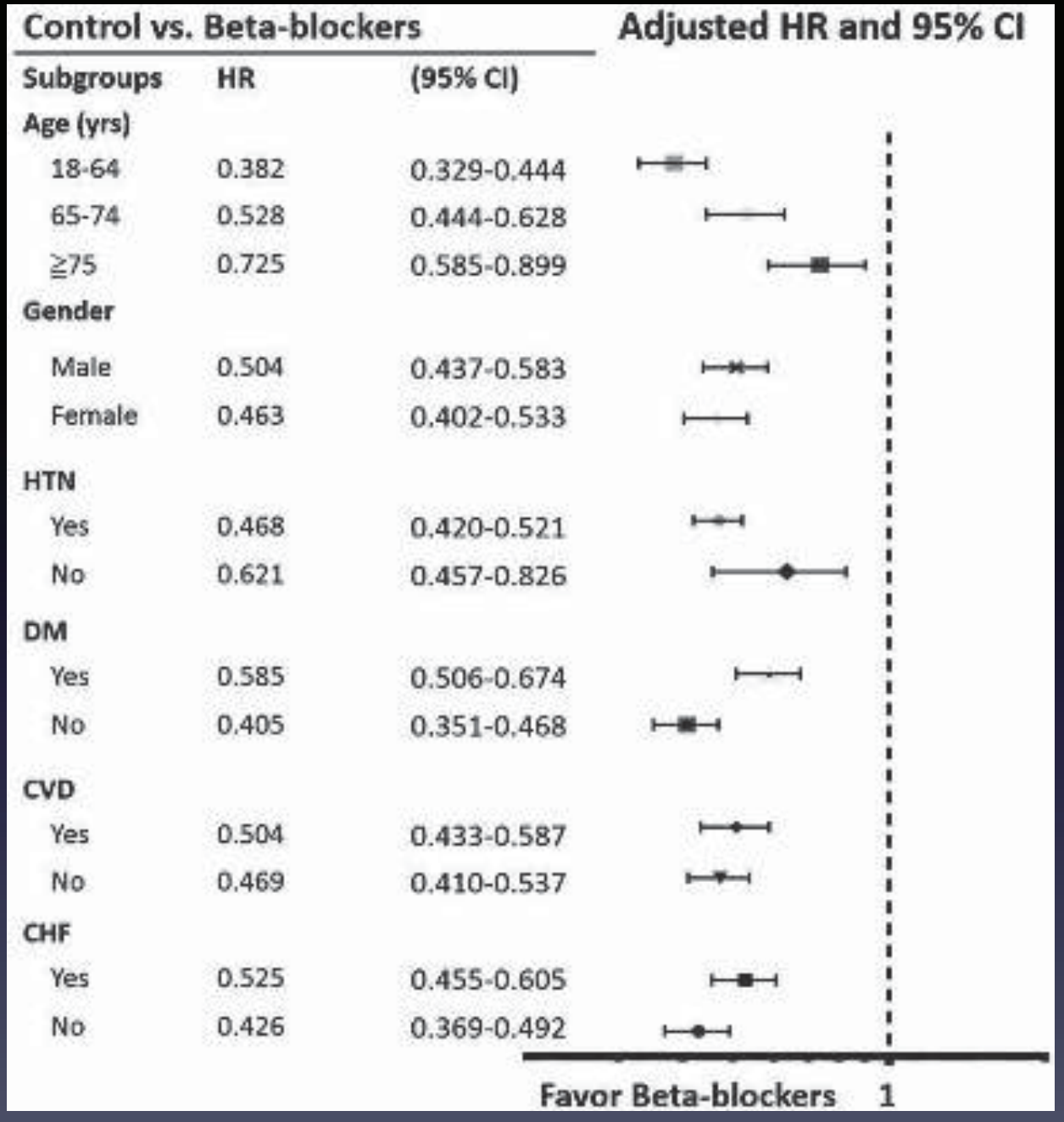


Figure 1 Prevention of AF with beta-blockers in HF.

www.nature.com/scientific reports/

Scientific **RepoRts** | 5:17731 | DOI: 10.1038/srep17731 (2015)



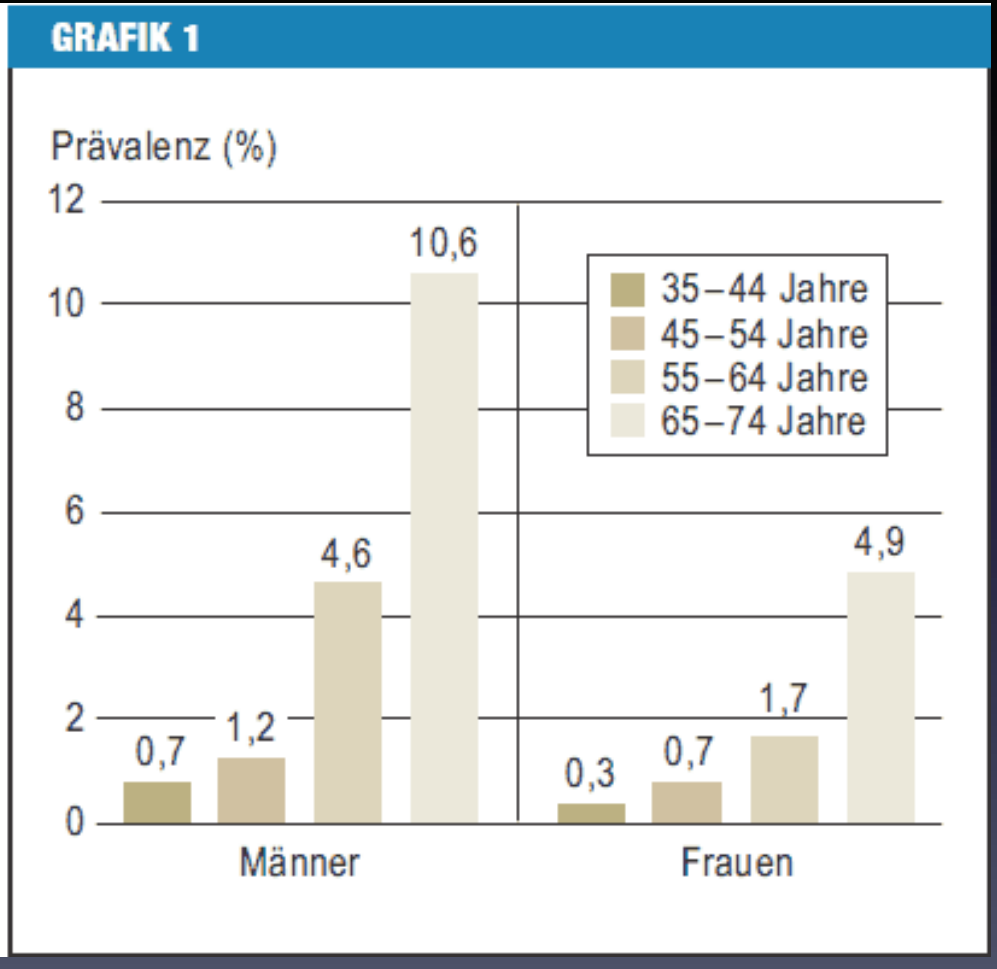
Assoziation zwischen Hypertonie und VHF

- Die Assoziation zwischen arterieller Hypertonie ist unstrittig!
- Ein wesentlicher pathophysiologischer Mechanismus ist die Fibrosierung im Bereich der Vorhöfe als Folge der erhöhten Druckbelastung (mechanisch) und der Einwirkung des Sympathikus bzw. des RAAS auf das Myokard des Vorhofs.
- Demzufolge sind RAAS-Inhibitoren und Betablocker effektiv in der primären Prävention des Vorhofflimmerns.

Epidemiologie des Vorhofflimmerns

Dtsch Arztebl Int 2012; 109(16): 293-9;

Prävalenz von Vorhofflimmern bei Patienten der Gutenberg-Gesundheitsstudie (GHS) nach Altersdekaden und Geschlecht dargestellt



(Circulation. 2014;129:837-847.)

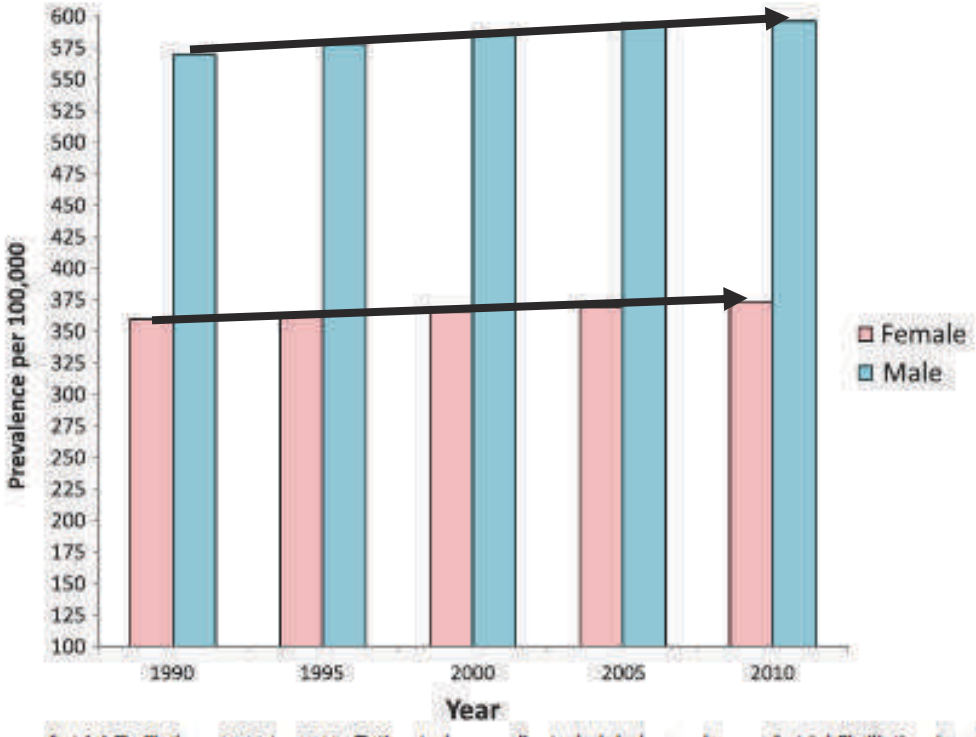
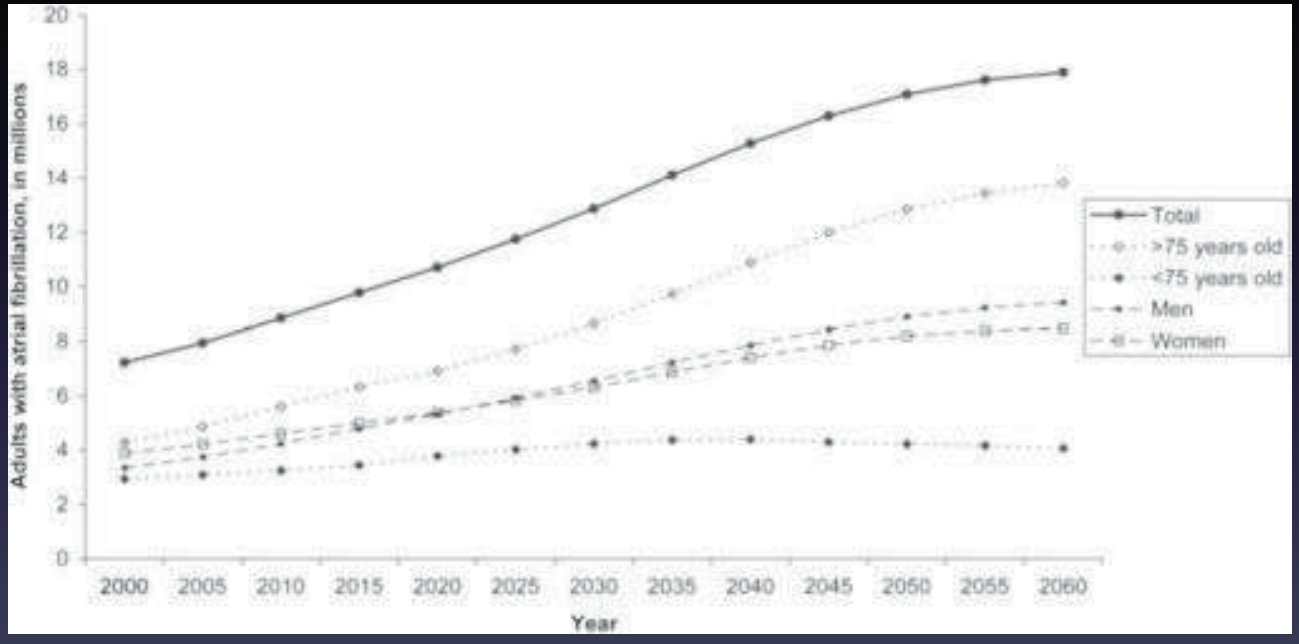


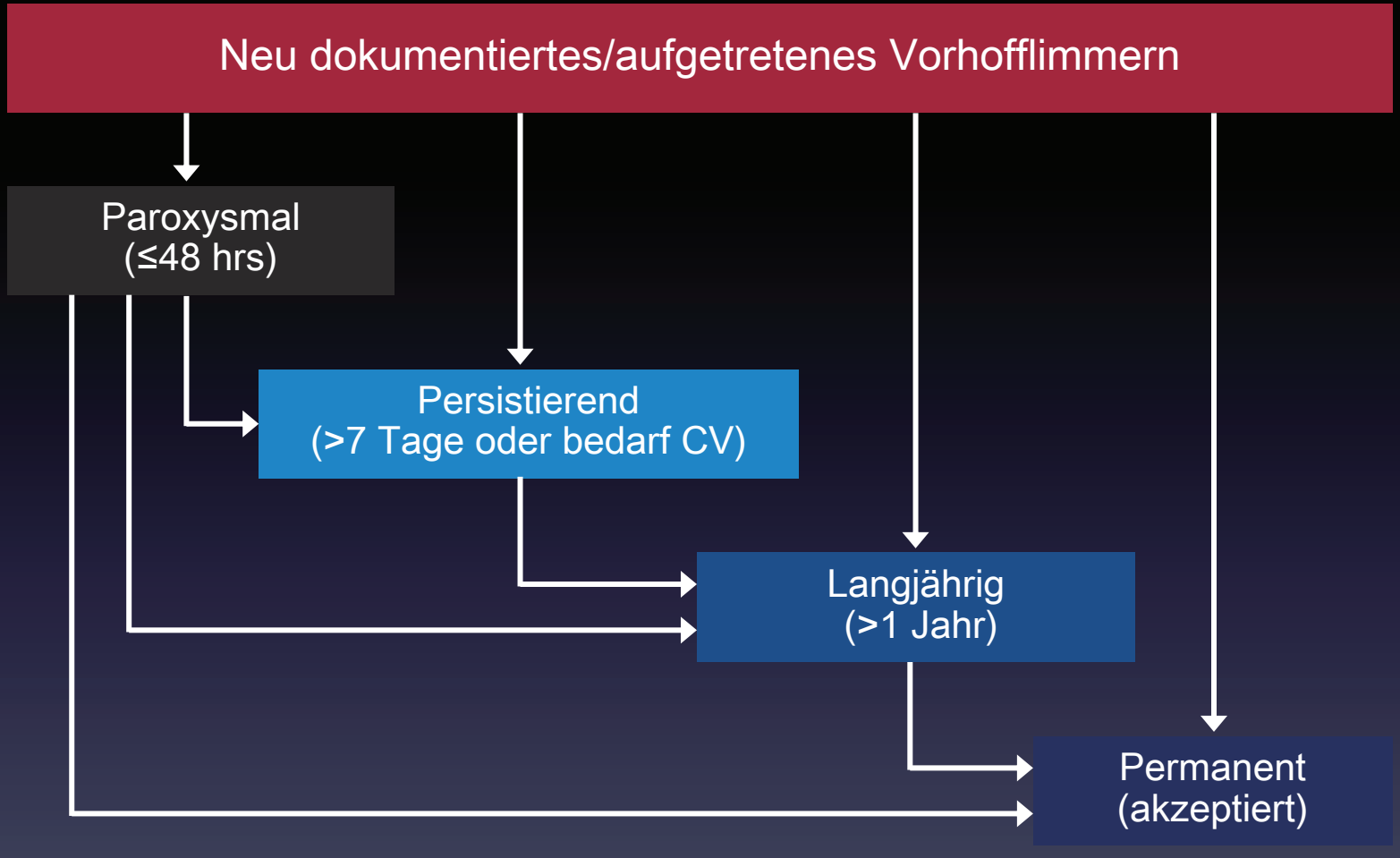
Figure 3. Prevalence of atrial fibrillation: 1990 to 2010. Estimated age-adjusted global prevalence of atrial fibrillation (per 100 000 population) for men and women from 1990 to 2010.

Die Prävalenz des Vorhofflimmerns hat in den letzten 20 Jahren bei beiden Geschlechtern zugenommen!

Ruigómez A., Johansson S., Wallander M.A., Rodríguez L.A. Incidence of chronic atrial fibrillation in general practice and its treatment pattern. J Clin Epidemiol. 2002;55(4):358–363



Klassifikation von Vorhofflimmern (ESC Guidelines 2010)



CV = Kardioversion

ESC guidelines: Camm J et al. Eur Heart J 2010;31:2369–429

Komplikationen des unbehandelten Vorhofflimmerns

- Objektivierbare Komplikationen:
  - Insult
  - Kardiomyopathie (tachykardie-assoziiert)
  - Periphere Embolien
    - Mesenterialarterienembolie
    - Embolie in die Extremitätenarterien
- Nicht objektivierbare Komplikationen
  - Reduktion der Lebensqualität
  - Reduktion der Leistungsfähigkeit

Risiko für Insult und periphere Embolie

	Insult	Periphere Embolie
ATRIA STUDY	2.3%	0.16%
AFI STUDY	2.3%	0.2%
SPAF	3.3%	
FRAMINGHAM COHORT	1.7%	

Eine relevante Komplikation des Vorhofflimmerns ist der Morbus embolicus!

Cowan C, Healicon R, Robson I, Long WR, Barrett J, Fay M, et al.. The use of anticoagulants in the management of atrial fibrillation among general practices in England. Heart 2013;99(16):1166–72.

Figure 2. Proportion of atrial fibrillation patients prescribed anticoagulation and antiplatelet agents among general practices in England, according to Cowan C and colleagues, Heart 2013 [Ref. [16]], original figure, with permission].

Table 1. Results of the BAFTA trial [Ref. [38]].

		Warfarin ($n = 448$)		Aspirin ($n = 485$)		Warfarin vs. aspirin	
		n	Risk per year (%)	n	Risk per year (%)	RR (95% CI)	p
Stroke		21	1.6	44	3.4	0.46 (0.26–0.79)	0.003
By severity	Fatal	13	1.0	21	1.6	0.59 (0.27–1.24)	0.14
	Disabling non-fatal	8	0.6	23	1.8	0.33 (0.13–0.77)	0.005
Type of stroke	Ischemic	10	0.8	32	2.4	0.30 (0.13–0.63)	0.0004
	Hemorrhagic	6	0.5	5	0.4	1.15 (0.29–4.77)	0.83
	Unknown	5	0.4	7	0.5	0.69 (0.17–2.51)	0.53
Other intracranial bleeding		2	0.2	1	0.1	1.92 (0.10–113.3)	0.65
Systemic embolism		1	0.1	3	0.2	0.32 (0.01–3.99)	0.36
Total number of events		24	1.8	48	3.8	0.48 (0.28–0.80)	0.0027

Diagnostik des Vorhofflimmerns

- Puls messen ????
- 12-Ableitungs-EKG
- Holter-EKG
- (Blutdruckmessung zu Hause)
- (Nicht 12-Ableitungs-EKG (via Smartphone))

Opportunistic Screening

Recommendations for screening AF		
Recommendations	**Class[a]**	**Level[b]**
Opportunistic screening for AF in patients ≥65 years of age using pulse-taking followed by an ECG is recommended to allow timely detection of AF.	I	B

[a]Class of recommendation. [b]Level of evidence.
AF = atrial fibrillation; LoE = level of evidence.

European Heart Journal 2012;33:2719-2747 - doi:10.1093/eurheartj/ehs253

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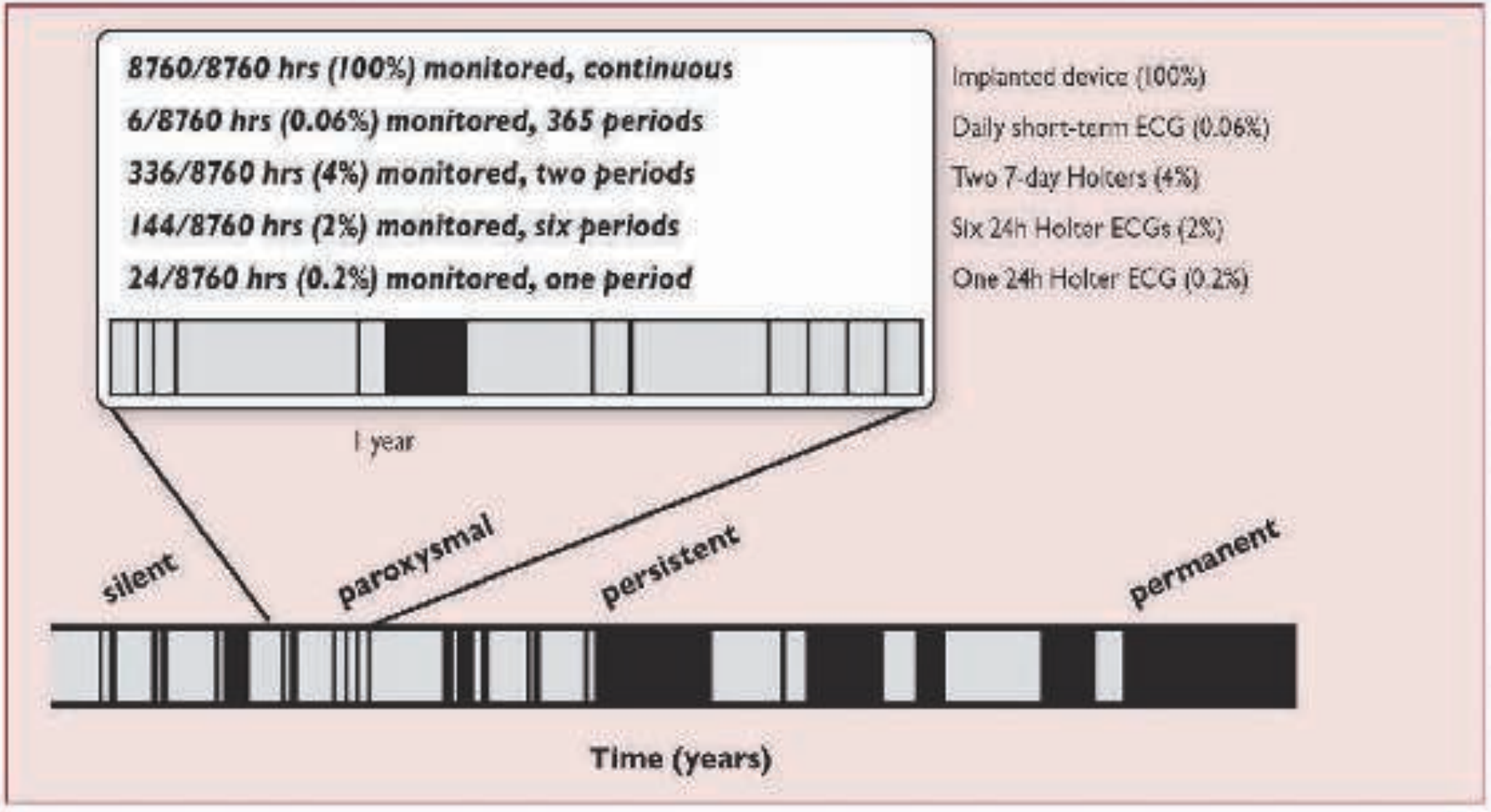
Diagnostic yield of different ECG screening techniques for paroxysmal or silent atrial fibrillation



European Heart Journal - doi:10.1093/eurheartj/ehw210

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BPMs and non-12-lead ECG devices had a greater accuracy for detecting pulse irregularities caused by AF than pulse palpation. Newer technologies may therefore be a pragmatic alternative to pulse palpation for identifying patients with suspected AF as part of national screening programmes.

European Journal of Preventive Cardiology 2016, Vol. 23(12) 1330–1338

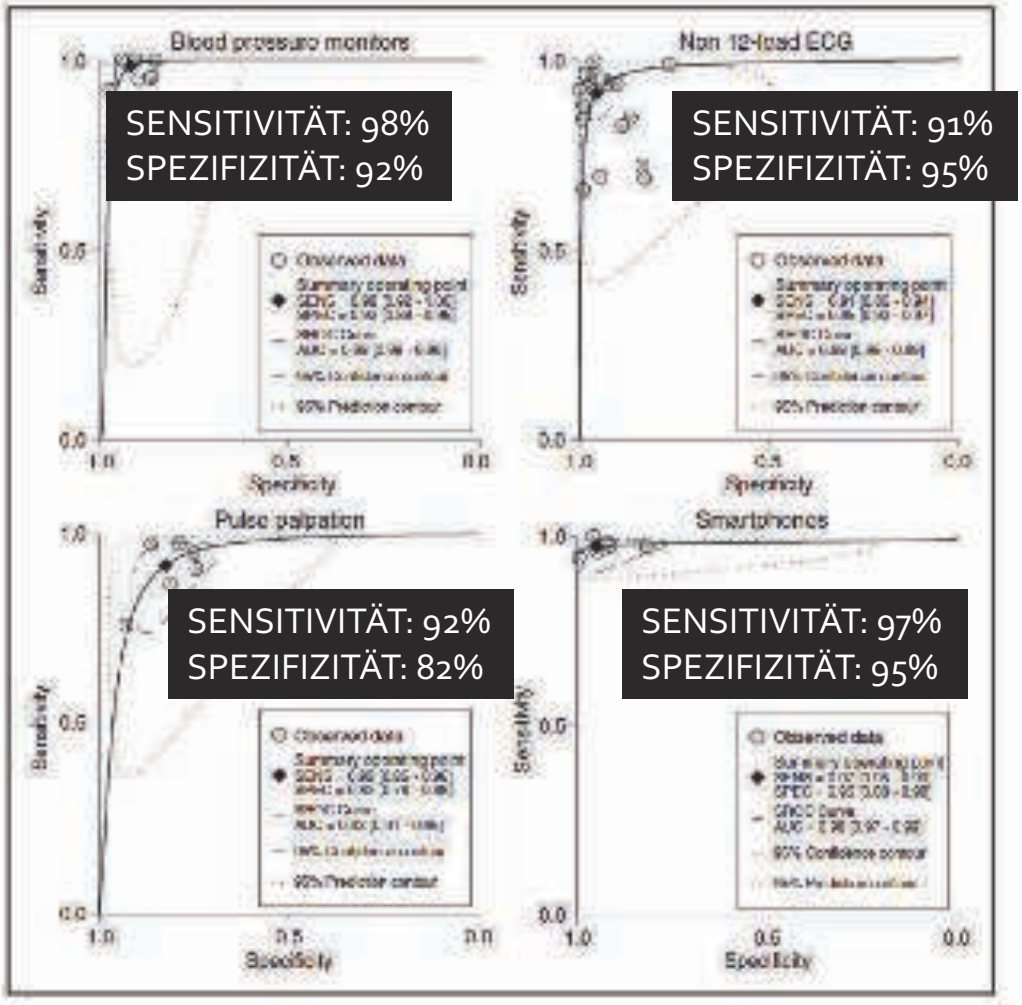
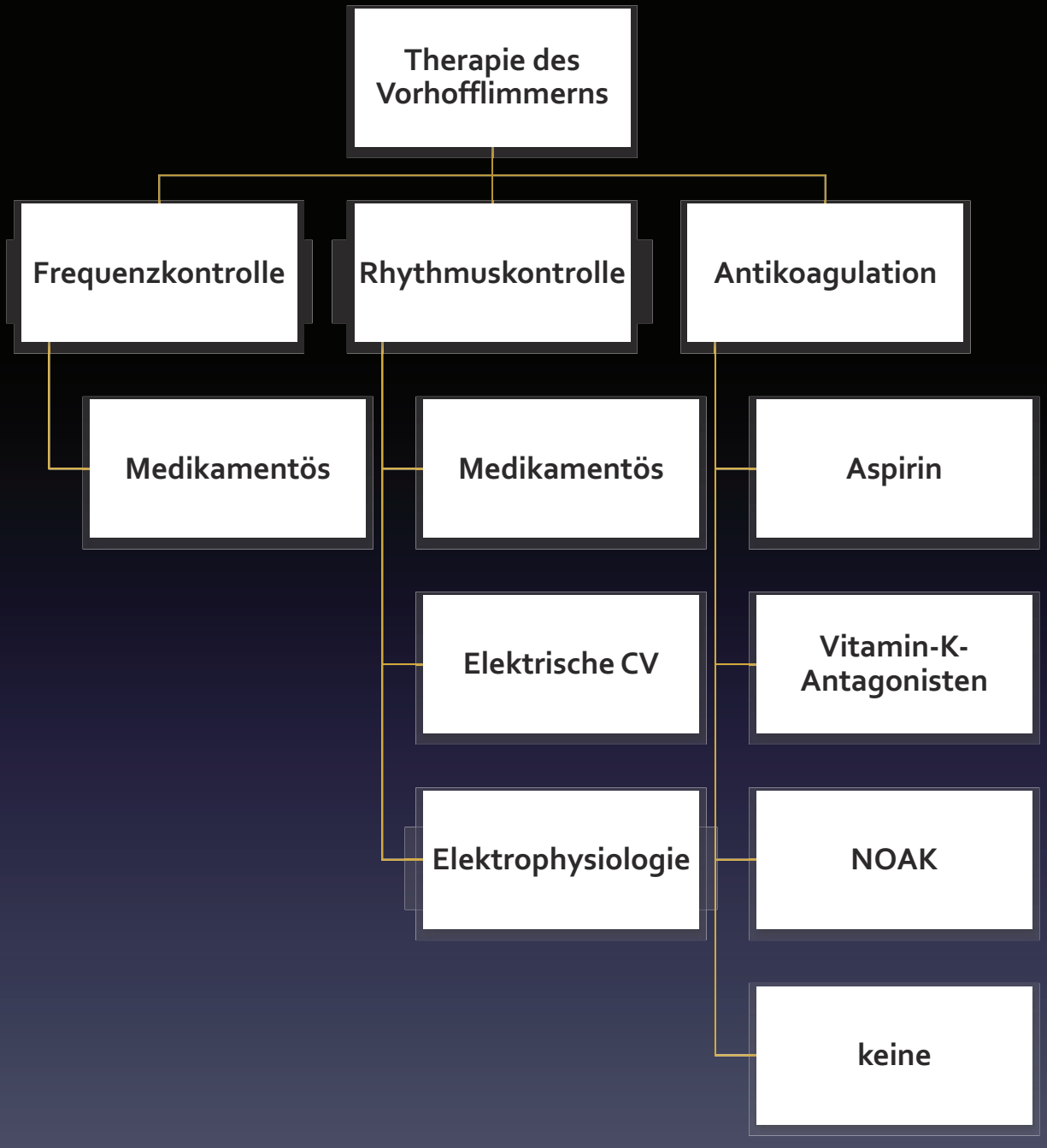


Figure 3. Summary receiver operating characteristic plots of interventions for the detection of atrial fibrillation.


Therapie des Vorhofflimmerns
Frequenzkontrolle
Rhythmuskontrolle
Antikoagulation
Medikamentös
Medikamentös
Aspirin
Elektrische CV
Vitamin-K-Antagonisten
Elektrophysiologie
NOAK
keine

Rhythmus-Kontrolle versus Frequenzkontrolle

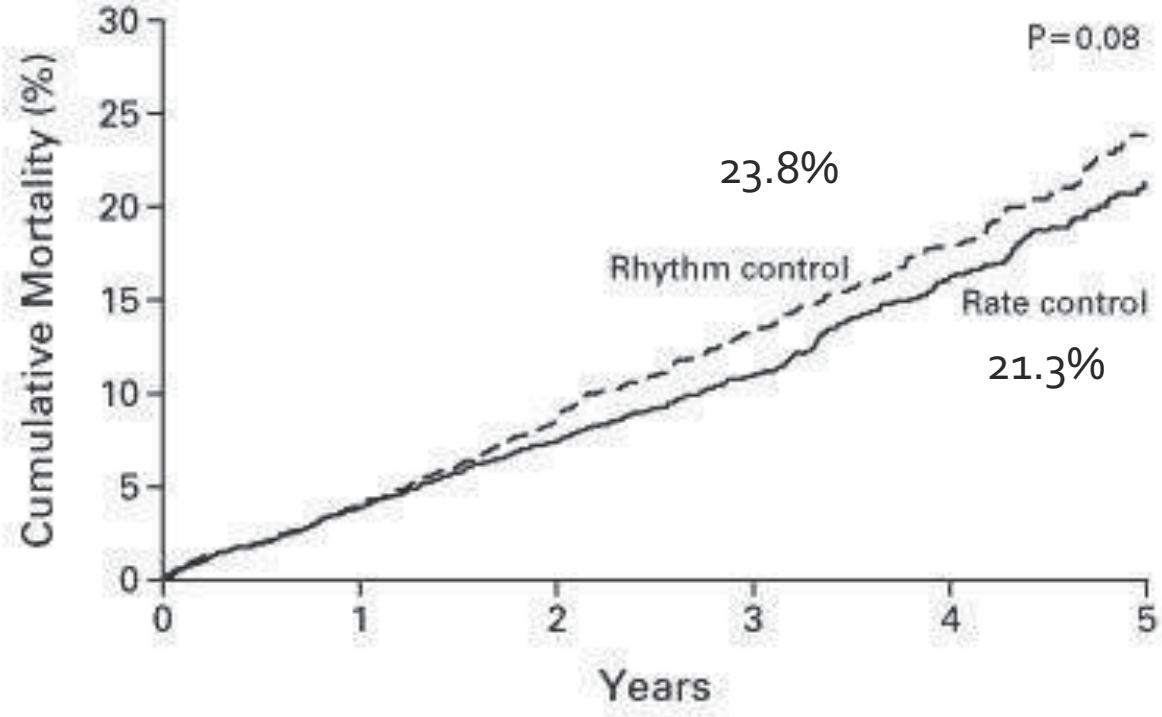
- Vorteile der Rhythmuskontrolle:
  - Bessere Lebensqualität
  - Bessere Leistungsfähigkeit
- Vorteile der Frequenzkontrolle:
  - Nebenwirkungen der Antiarrhythmika geringer

AFFIRM Trial

- Patients who were at least 65 years of age or who had other risk factors for stroke or death could be enrolled in this study. The overriding criteria for enrollment were that (in the clinical judgment of the investigators) atrial fibrillation was likely to be recurrent; atrial fibrillation was likely to cause illness or death; long-term treatment for atrial fibrillation was warranted; anticoagulant therapy was not contraindicated
- In the rhythm-control group, the antiarrhythmic drug used was chosen by the treating physician.[24,25] Attempts to maintain sinus rhythm could include cardioversion as necessary. The following drugs were acceptable for use, according to the protocol: amiodarone, disopyramide, flecainide, moricizine, procainamide, propafenone, quinidine, sotalol, and combinations of these drugs. When dofetilide became available, it also could be used. Specific guidelines for the use of antiarrhythmic drugs were imposed.[22,26]
- **Rate-Control Strategy**
- The therapeutic target in this group was heart-rate control. Drugs that were acceptable in the protocol for this purpose were beta-blockers, calcium-channel blockers (verapamil and diltiazem), digoxin, and combinations of these drugs. Heart-rate control during atrial fibrillation was assessed both at rest and during activity, which usually consisted of a six-minute walk.[22,27] The goal was a heart rate not higher than 80 beats per minute at rest and not higher than 110 beats per minute during the six-minute walk test.

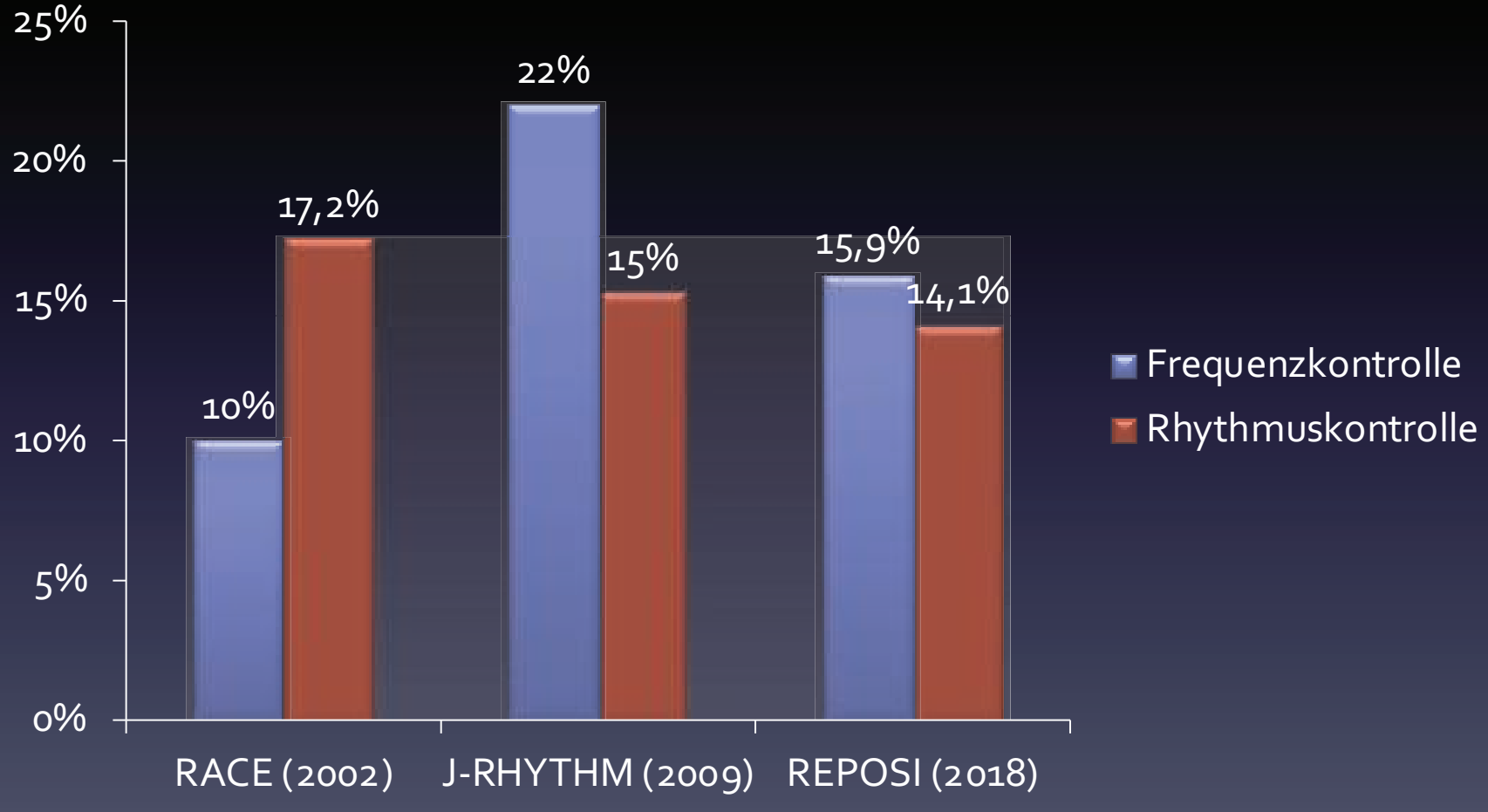
AFFIRM Trial

N Engl J Med 2002; 347:1825-1833



No. of Deaths		number (percent)				
Rhythm control	0	80 (4)	175 (9)	257 (13)	314 (18)	352 (24)
Rate control	0	78 (4)	148 (7)	210 (11)	275 (16)	306 (21)

Vergleich Frequenz- versus Rhythmuskontrolle



Rhythmuskontrolle

- Ziel ist die dauerhafte Wiederherstellung eines Sinusrhythmus
- Geeignete PatientInnen:
  - Neu aufgetretenes Vorhofflimmern
  - Kurze Dauer des Vorhofflimmerns
  - Symptomatisches Vorhofflimmern
    - Subjektiv
    - Objektiv – eingeschränkte Leistungsfähigkeit

Optionen der Rhythmus-Kontrolle

- Medikamentöse Therapie
  - Die Wahl des Antiarrhythmikums hängt von kardialen Situation ab.
- Elektrische Kardioversion
  - Ohne orale Antikoagulation möglich, wenn **zweifelsfrei** der Beginn des Vorhofflimmerns festgestellt werden kann und dieser **kürzer als 72 Stunden** zurückliegt.
  - Alternativ: Orale Antikoagulation für zumindest 4 Wochen mit einem INR-Wert zwischen 2 und 3 bzw. bei NOAKs Therapiedauer von zumindest 3 Wochen.

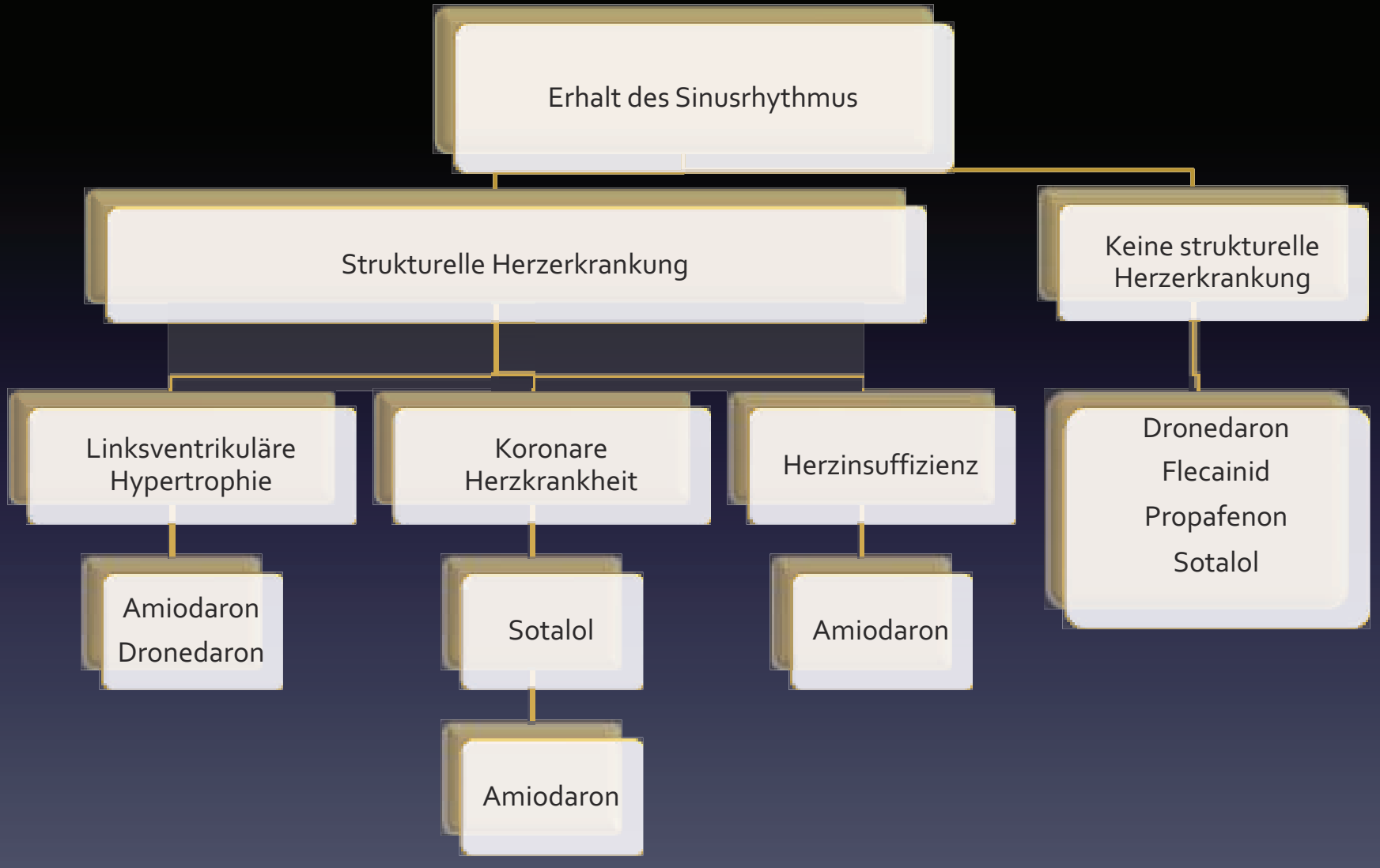
Entscheidungsdiagramm bei rezent aufgetretenem Vorhofflimmern

Dosierungs - ABC

	Amiodaron Sedacoron ®	**Flecainid Aristocor ®**	**Propafenon Rytmonorma ®**
Applikation	intravenös	intravenös	intravenös
Initiale Dosierung	300 mg	1 mg/kg KG	0.5-1 mg/kg KG
Repetitive Dosierung	300 mg	0.5 mg/kg KG	0.5-1 mg/kg KG

Die Entscheidung zur Kardioversion beinhaltet auch die Entscheidung den Sinusrhythmus durch dauerhafte Einnahme von Antiarrhythmika zu erhalten!!

Entscheidungsdiagramm zum Erhalt des Sinusrhythmus

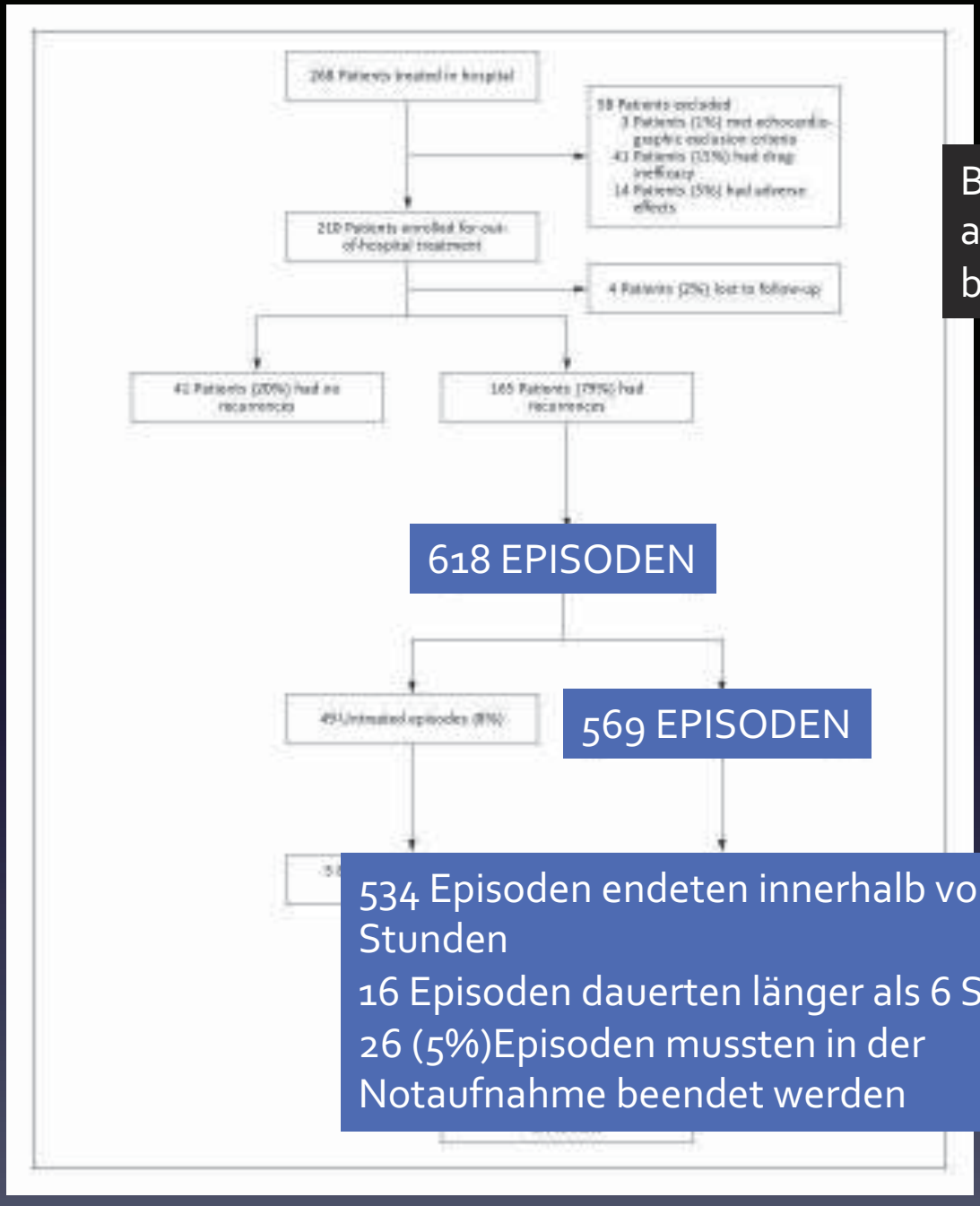


Dosierungs-ABC

Substanz	Dosierung	Dosierungsintervall
Sedacoron® (Amiodaron)	200 mg	24 Stunden
Aristocor ® (Flecainid)	100 mg Initial: 50 mg	12 Stunden
Rytmonorma ® (Propafenon)	150-200 mg Tagesdosis: 450-600 mg	8-12 Stunden
Sotacor ® (Sotalol)	80-160 mg Initial: 40 mg	12 Stunden
Multaq ® (Dronedaron)	400 mg	12 Stunden

Pill-in-the-Pocket

- Das therapeutische Prinzip basiert auf einer situationsbezogenen Einnahme von Antiarrhythmika und erlaubt dem Patienten ein hohes Maß an Autonomie.
- Geeignete Substanzen:
  - Flecainid
  - Propafenon
- Voraussetzung:
  - keine strukturelle Herzerkrankung
  - Test der Effektivität im Krankenhaus unter Monitoring



Behandlung mit

a) Aristocor 200-300 mg p.o.
b) Rytmonorma 450-600 mg p.o.

534 Episoden endeten innerhalb von 6 Stunden

16 Episoden dauerten länger als 6 Stunden

26 (5%)Episoden mussten in der Notaufnahme beendet werden

N Engl J Med 2004; 351:2384-2391

Frequenzkontrolle

- Ziel der Frequenzkontrolle ist die Erhaltung eines normfrequenten Vorhofflimmern.
- Geeignete PatientInnen:
  - Symptomloses Vorhofflimmern
  - Mehrmalige frustrane Kardioversionsversuche
  - Permanentes Vorhofflimmern

Entscheidungsdiagramm bei PatientInnen mit Frequenzkontrolle

Akute Frequenzkontrolle

- Klinische Situation mit tachykardem Vorhofflimmern bei PatientInnen mit einem permanenten Vorhofflimmern:
  - Betablocker oder Calciumantantagonisten von Non-Dihydropyridin-Typ
  - Digitalis oder Amiodaron

Antikoagulation

- Vor Beginn einer Antikoagulation bei Vorhofflimmern ist das Verhältnis zwischem dem Nutzen (=Verhinderung eines embolischen Ereignisses) und dem Risiko (=Auftreten einer schwerwiegenden Blutungskomplikation) zu klären.
- Hierfür wurden in der Zwischenzeit gute Risikoscores entwickelt.

CHA_2DS_2-VASc Score empfohlen von der ESC

	Condition	Points
C	Congestive Heart Failure	1
H	Hypertension = BP consistently > 140/90 mm Hg	1
A_2	Age ≥ 75 years	2
D	Diabetes mellitus	1
S_2	Prior Stroke or TIA or Thromboembolism	2
V	Vascular Disease (previous MI, peripheral arterial disease or aortic plaque)	1
A	Age 65-74	1
S	Sex category (female=1, male=0)	1

Relation zwischen CHA_2DS_2-VASc-Score und Insult-Risiko

CHA_2DS_2-VASc-SCORE	Adjusted stroke rate (%/year)
0	0%
1	1.3%
2	2.2%
3	3.2%
4	4.0%
5	6.7%
6	9.8%
7	9.6%
8	6.7%
9	15.2%

Update ESC Guidelines 2012

Stratifizierung des individuellen Blutungsrisikos

HAS-BLED	Score	CHA_2DS_2-VASc - Score
Hypertension	1	1
Abnormal renal or liver function (1 point each)	1 or 2	
Stroke	1	2
Bleeding	1	
Labile INRs	1	
Elderly (e.g. age >65 yrs)	1	1 - 2
Drugs or alcohol (1 point each)	1 or 2	
Maximal-Score	9	

Ein HAS-BLED Score $\geq$ 3 weist auf ein erhöhtes Blutungsrisiko hin.
Es sind regelmäßige Kontrollen vorzusehen.

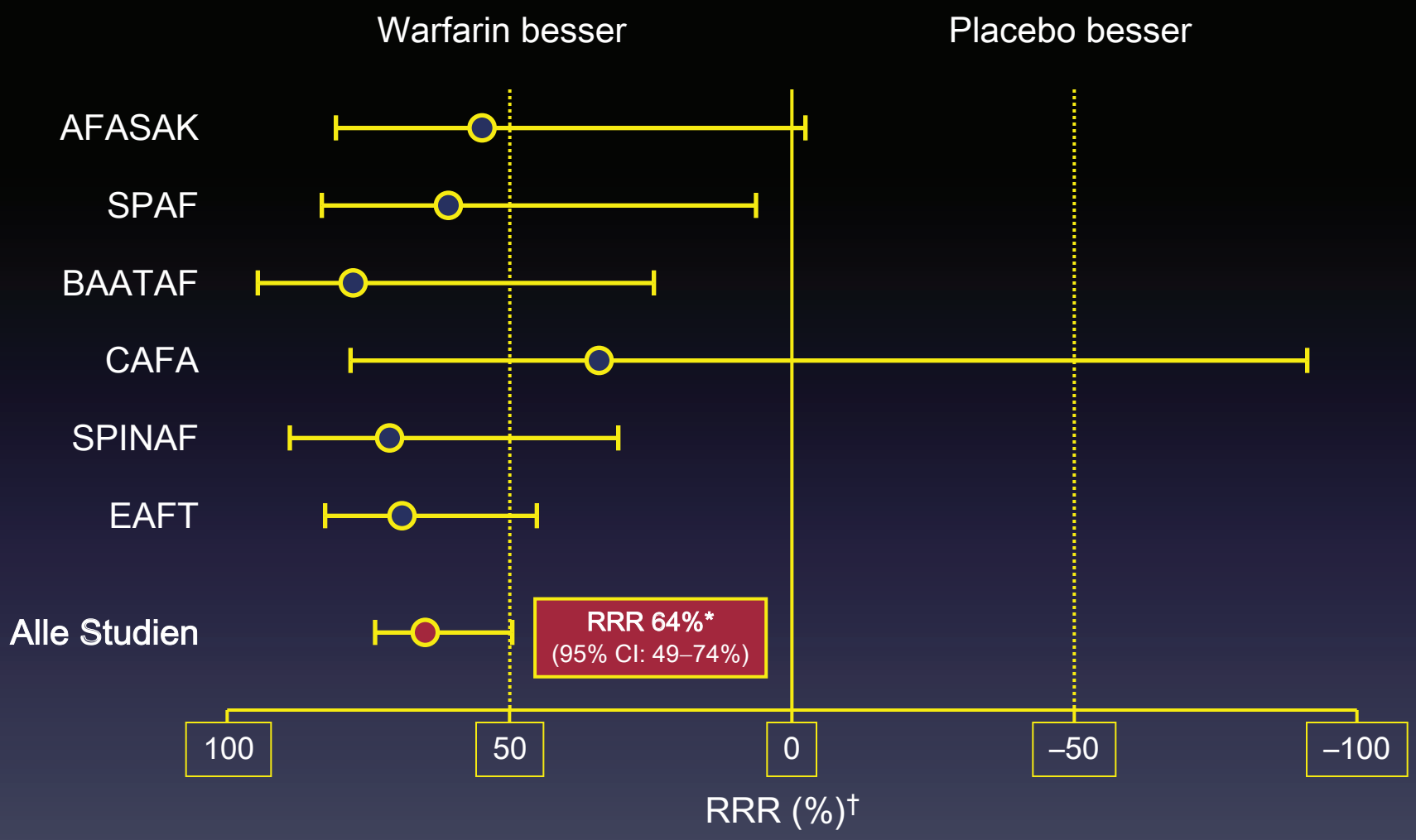
Eur Heart J 2012; doi:10.1093/eurheartj/ehs253

Antikoagulation

- Vitamin-K-Antagonisten
- NOAKs
  - Thrombin-inhibitoren: Dabigatran
  - Orale Xa-Inhibitoren: Rivaroxaban, Apixaban

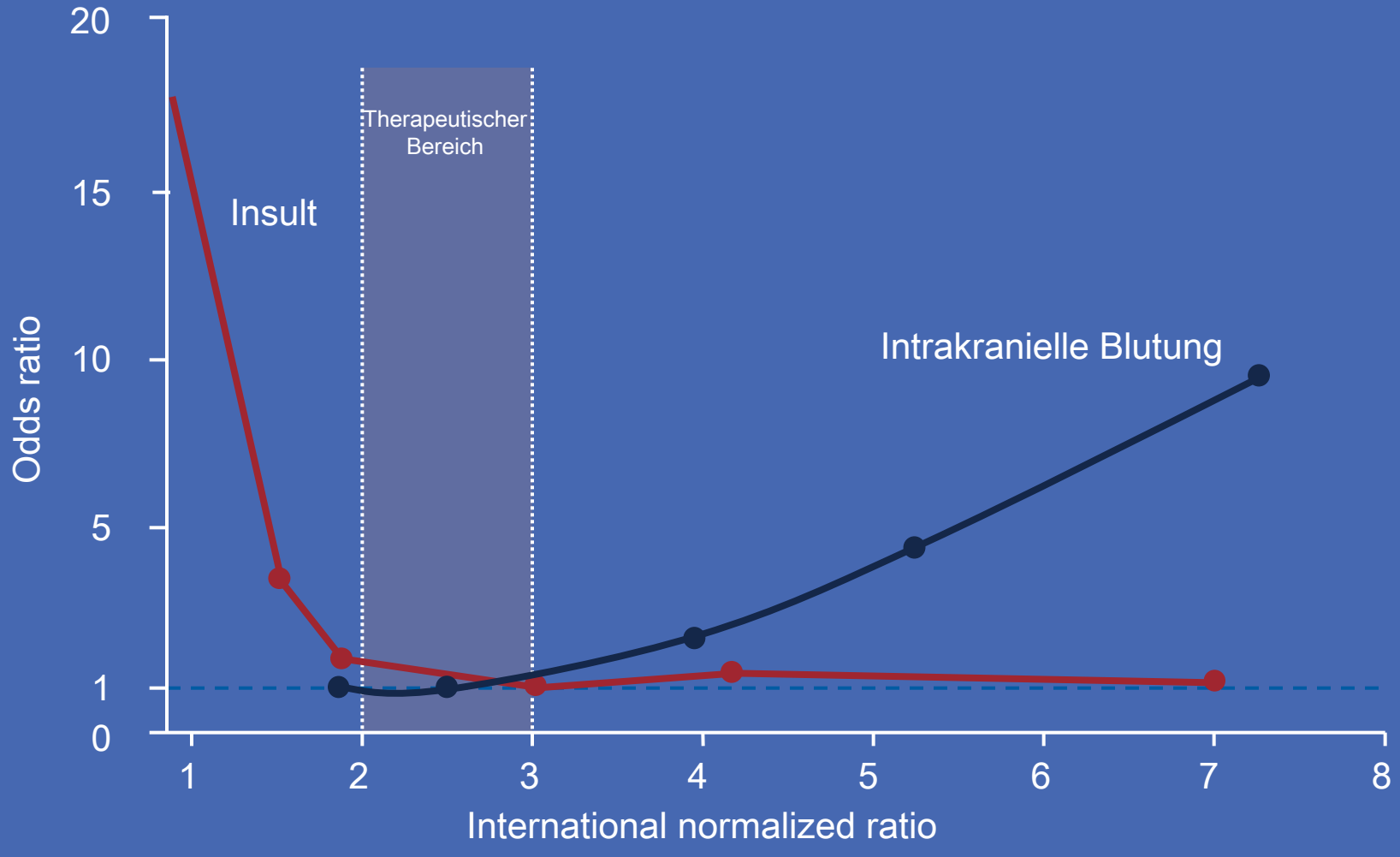
Warfarin reduziert das Schlaganfallrisiko bei Patient mit VHF um 64%

Hart RG et al. Ann Intern Med 2007;146:857–67



Random effects model; Error bars = 95% CI; *P>0.2 for homogeneity;
†Relative risk reduction (RRR) for all strokes (ischaemic and haemorrhagic)

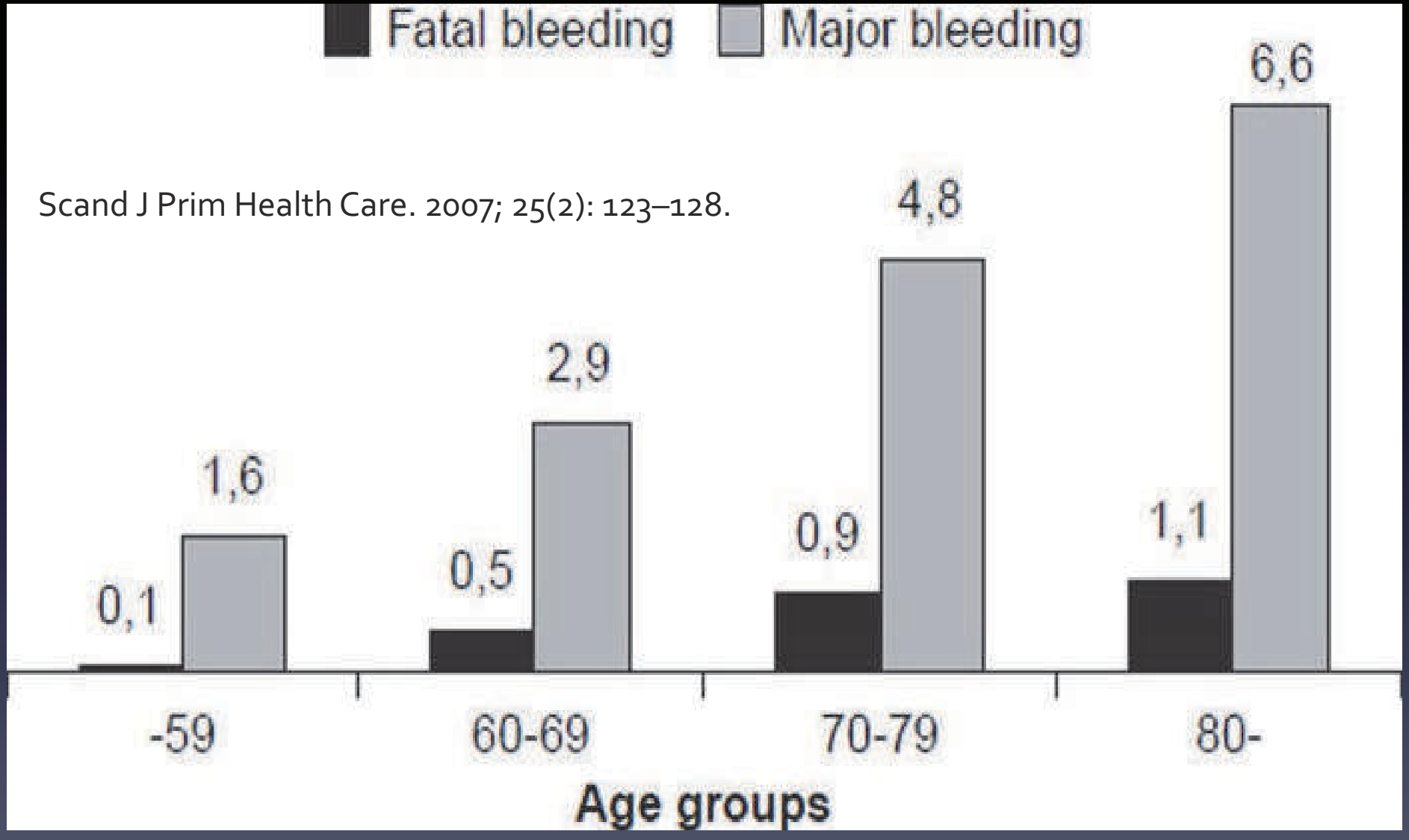
Grundproblem:
Schmale therapeutische Bereich der VKA-Therpie



ACC/AHA/ESC guidelines: Fuster V et al. Circulation 2006;114:e257–354 & Eur Heart J 2006;27:1979–2030

Blutungskomplikationen unter Vit-K-Antagonisten

Scand J Prim Health Care. 2007; 25(2): 123–128.



Lokalisation der Blutungen

Scand J Prim Health Care. 2007; 25(2): 123–128.

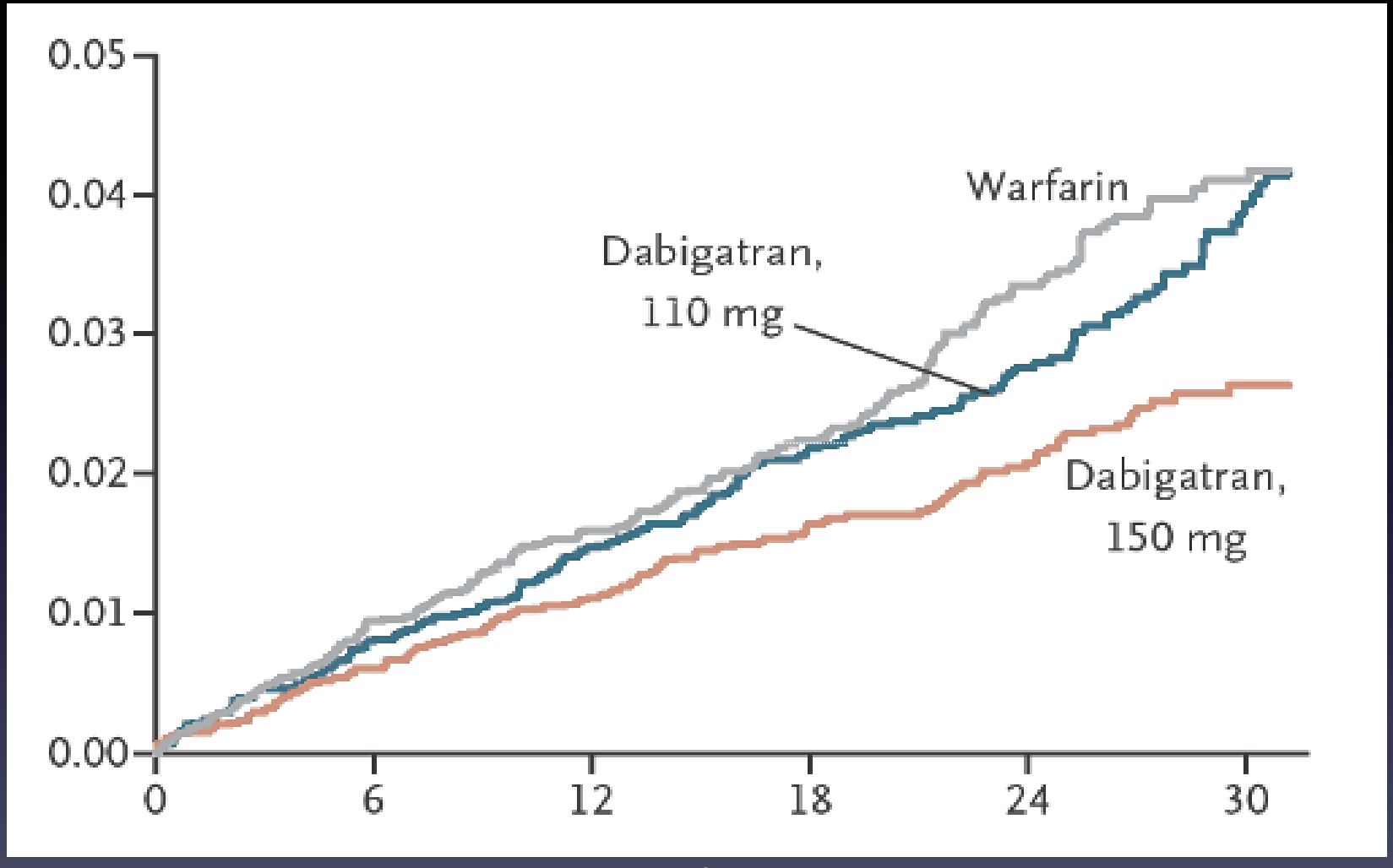
Bleeding event	Major	Fatal
Gastrointestinal haemorrhage	72	4
Intracerebral haemorrhage	37	21
Epistaxis	17	0
Haematuria	18	0
Abdominal aortic aneurysm	8	7
Other	43	2

Antikoagulation

- Vitamin-K-Antagonisten
- NOAKs
  - Thrombin-inhibitoren: Dabigatran
  - Orale Xa-Inhibitoren: Rivaroxaban, Apixaban, Edoxaban

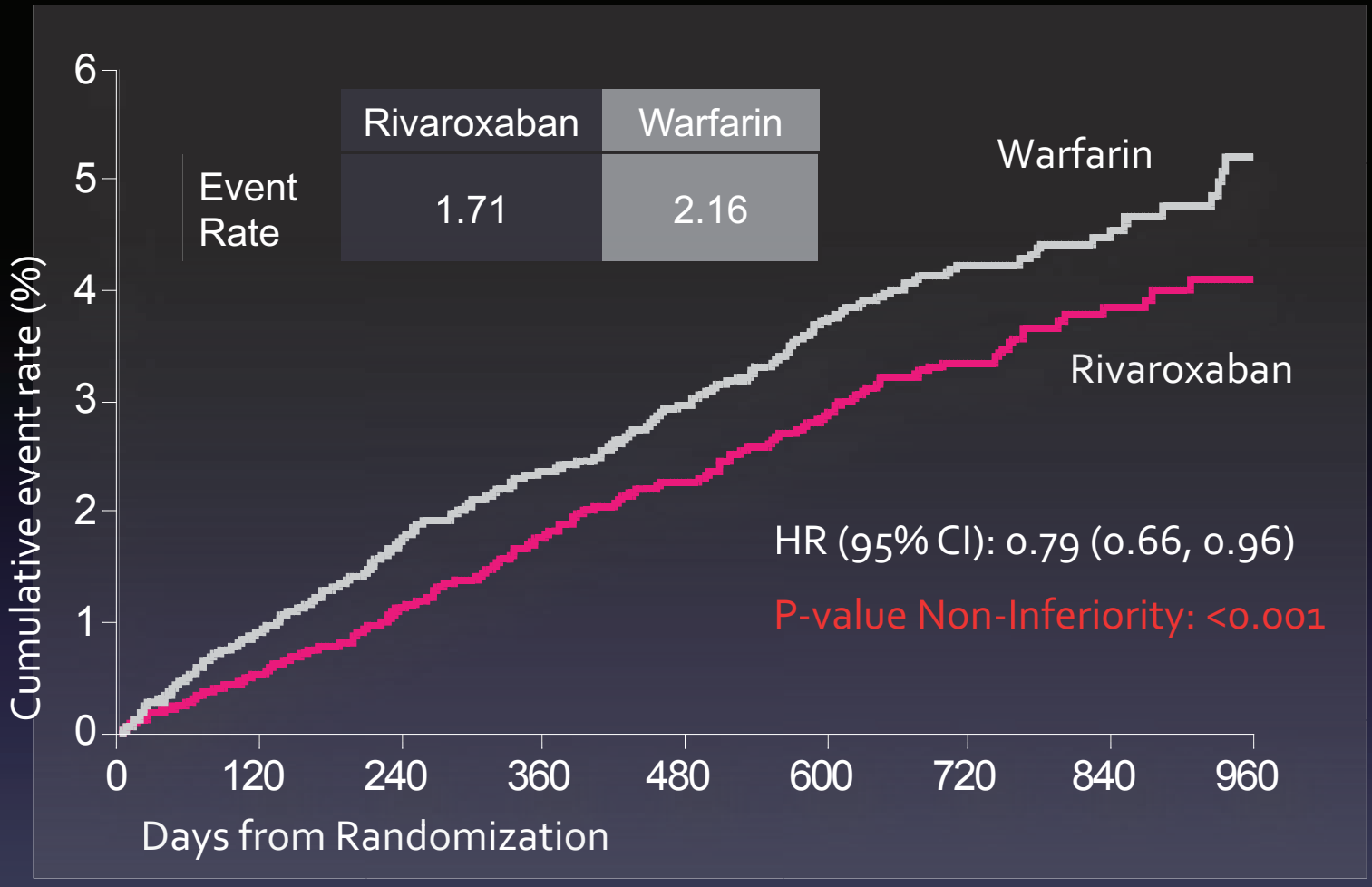
RE-LY Study (mean CHADS2 score: 2.1)

Primary Outcome: Systemic Embolism/Ischemic Stroke



Connolly SJ, New Engl J Med 2009

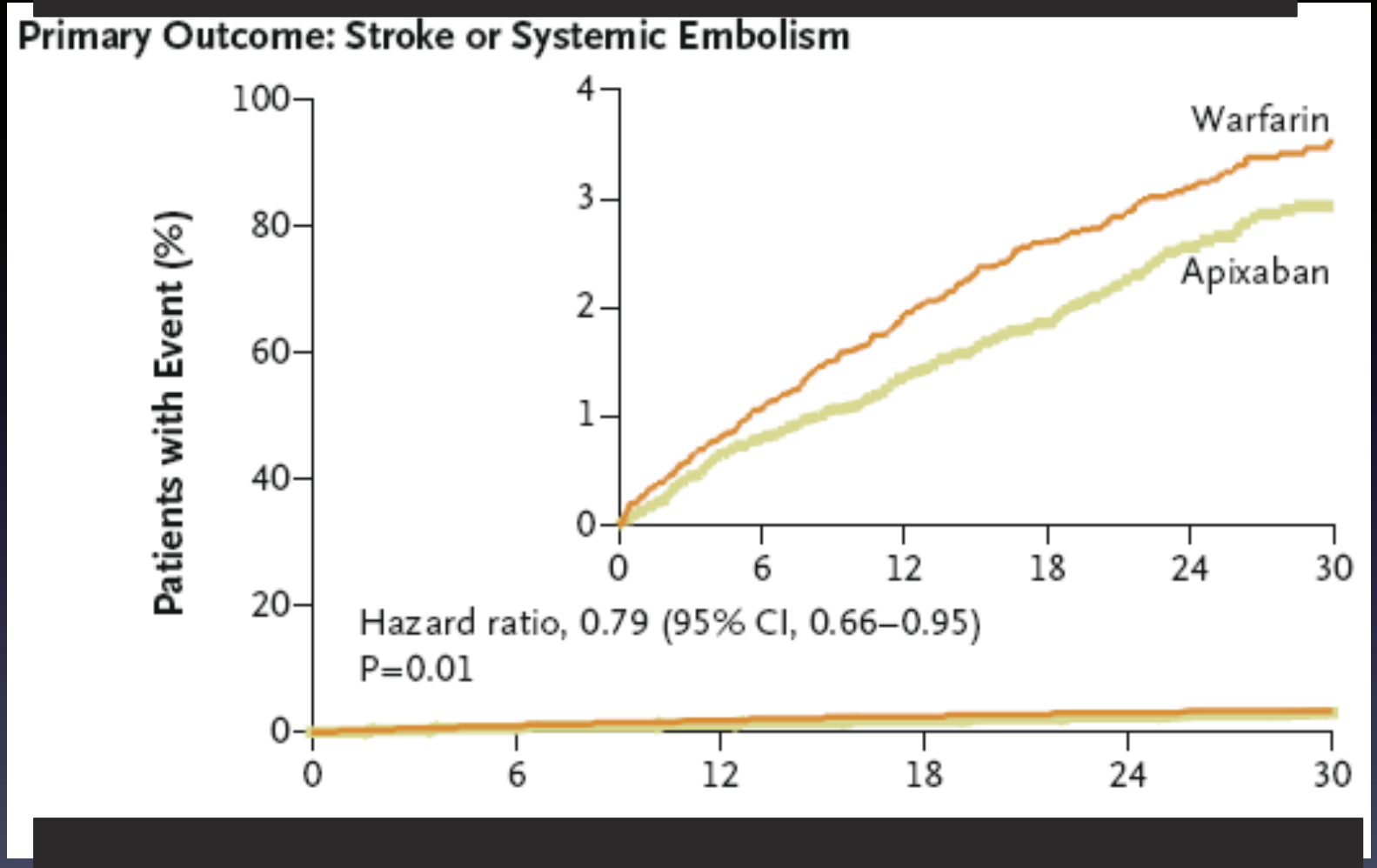
ROCKET-AF Study (mean CHADS2 score: 3.5)



Kenneth W. Mahaffey

Event Rates are per 100 patient-years
Based on Protocol Compliant on Treatment Population

ARISTOTLE – Apixaban vs Warfarin



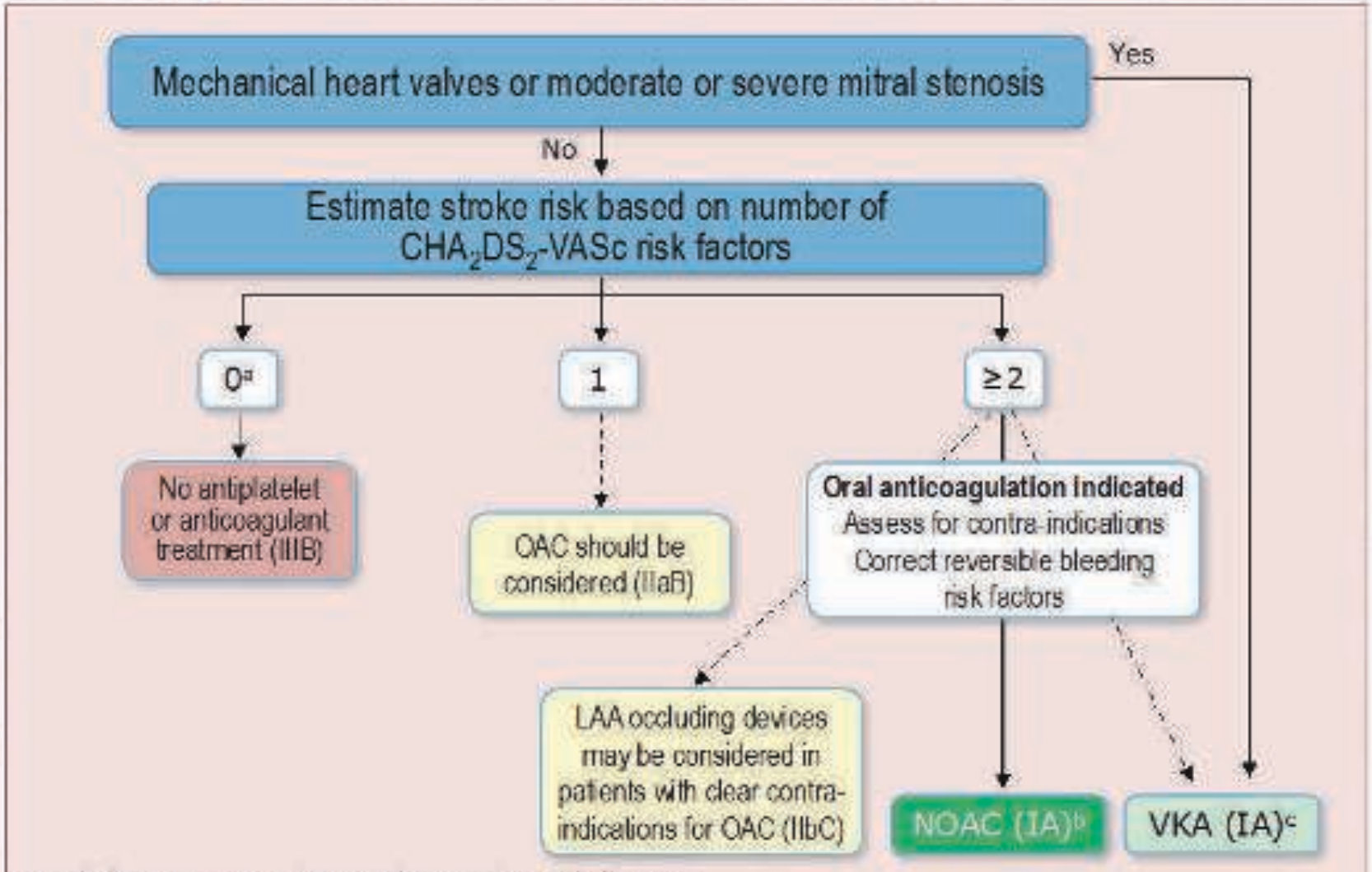
Wirksamkeit der NOAKs

	Dabigatran 110 mg	Dabigatran 150 mg	Rivaroxaban	Apixaban
Insult/syst. Embolie	-9%	-34%	-12%	-21%
Ischäm. Insult	+11%	-24%	-1%	-8%
Hämorrhag. Insult	-69%	-74%	-42%	-49%

Sicherheit und Netto-Benefit

	Dabigatran 110 mg	Dabigatran 150 mg	Rivaroxaban	Apixaban
Major Bleeding	-20%	-7%	+4%	-31%
ICB	-77%	-60%	-33%	-58%
GI-Bleeding	+12%	+50%	+45%	-11%
MCI	+29%	+27%	-19%	-12%
Ges. Mortalität	-9%	-12%	-8%	-11%
Netto-Benefit	-8%	-9%	???	-15%

Stroke prevention in atrial fibrillation



European Heart Journal - doi:10.1093/eurheartj/ehw210

EUROPEAN SOCIETY OF CARDIOLOGY®

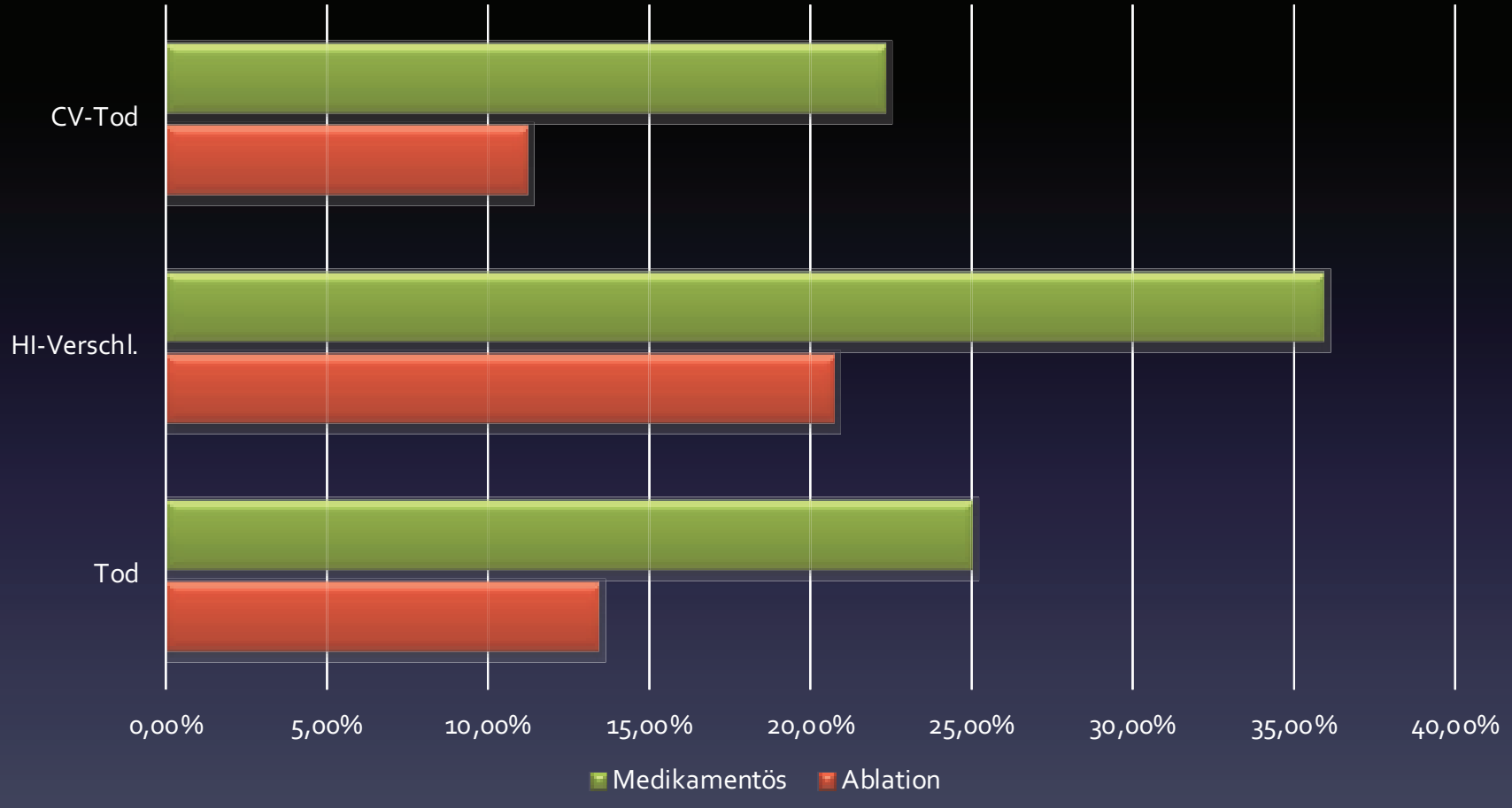
Management der VHF bei Herzinsuffizienz

Mögliche richtungsweisende neue Daten

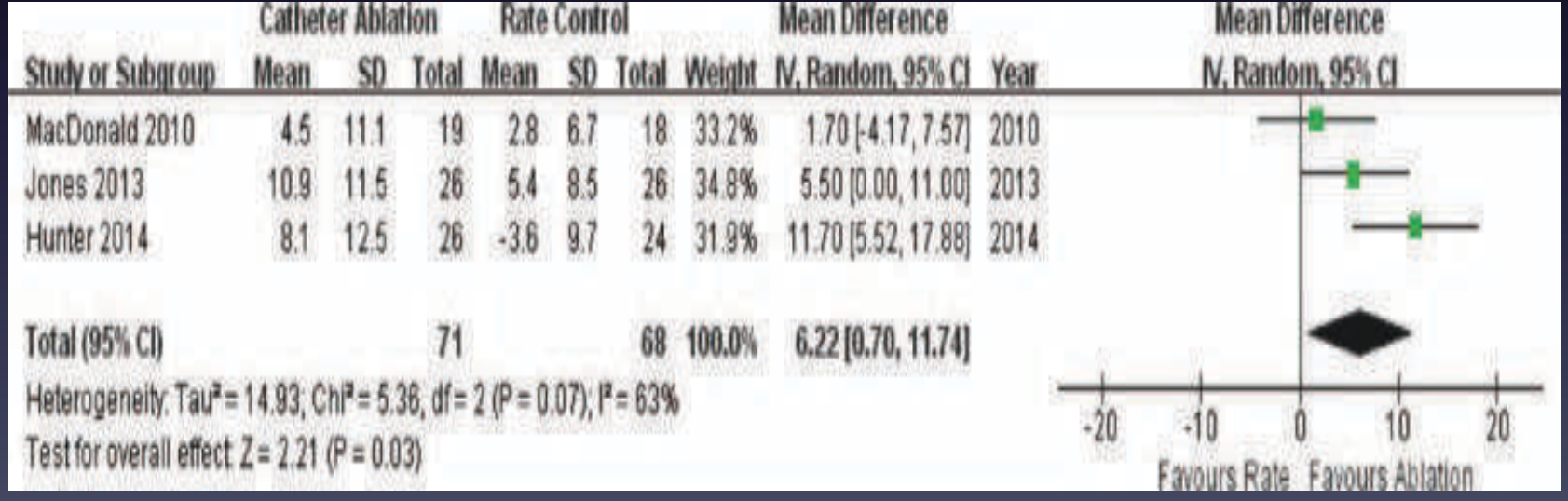
CASTLE-AF NEJM 2018, Februar

- 363 Patienten mit Herzinsuffizienz wurden entweder zur Katheterablation oder zur medikamentösen Therapie des Vorhofflimmerns randomisiert.
- Endpunkt waren Tod und/oder Verschlechterung der bestehenden Herzinsuffizienz.

CASTLE-AF


CV-Tod
HI-Verschl.
Tod
0,00%
5,00%
10,00%
15,00%
20,00%
25,00%
30,00%
35,00%
40,00%
Medikamentös
Ablation

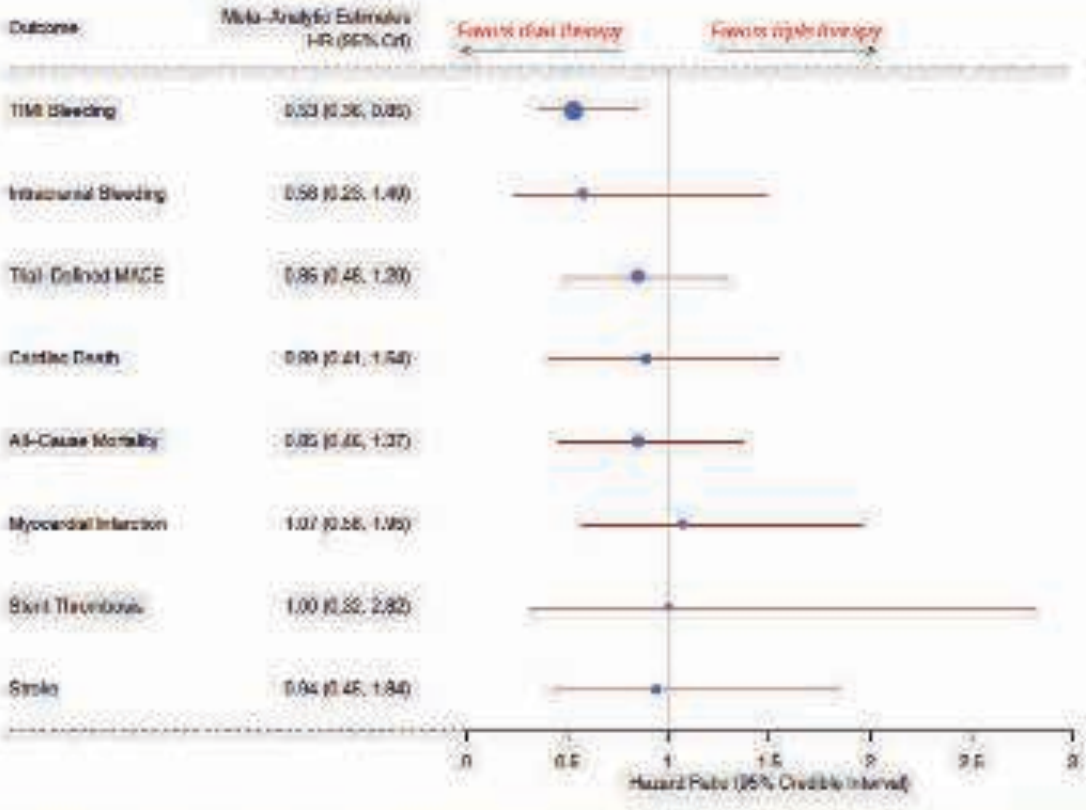
Ablation bei Herzinsuffizienz Veränderung der Lebensqualität



VHF und PCI

Wie geht es weiter?

Safety and efficacy of dual vs. triple antithrombotic therapy in patients with atrial fibrillation following percutaneous coronary intervention: a systematic review andmeta-analysis of randomized clinical trials



Take home figure Summary of bleeding and ischaemic risks for dual versus triple antithrombotic therapy.

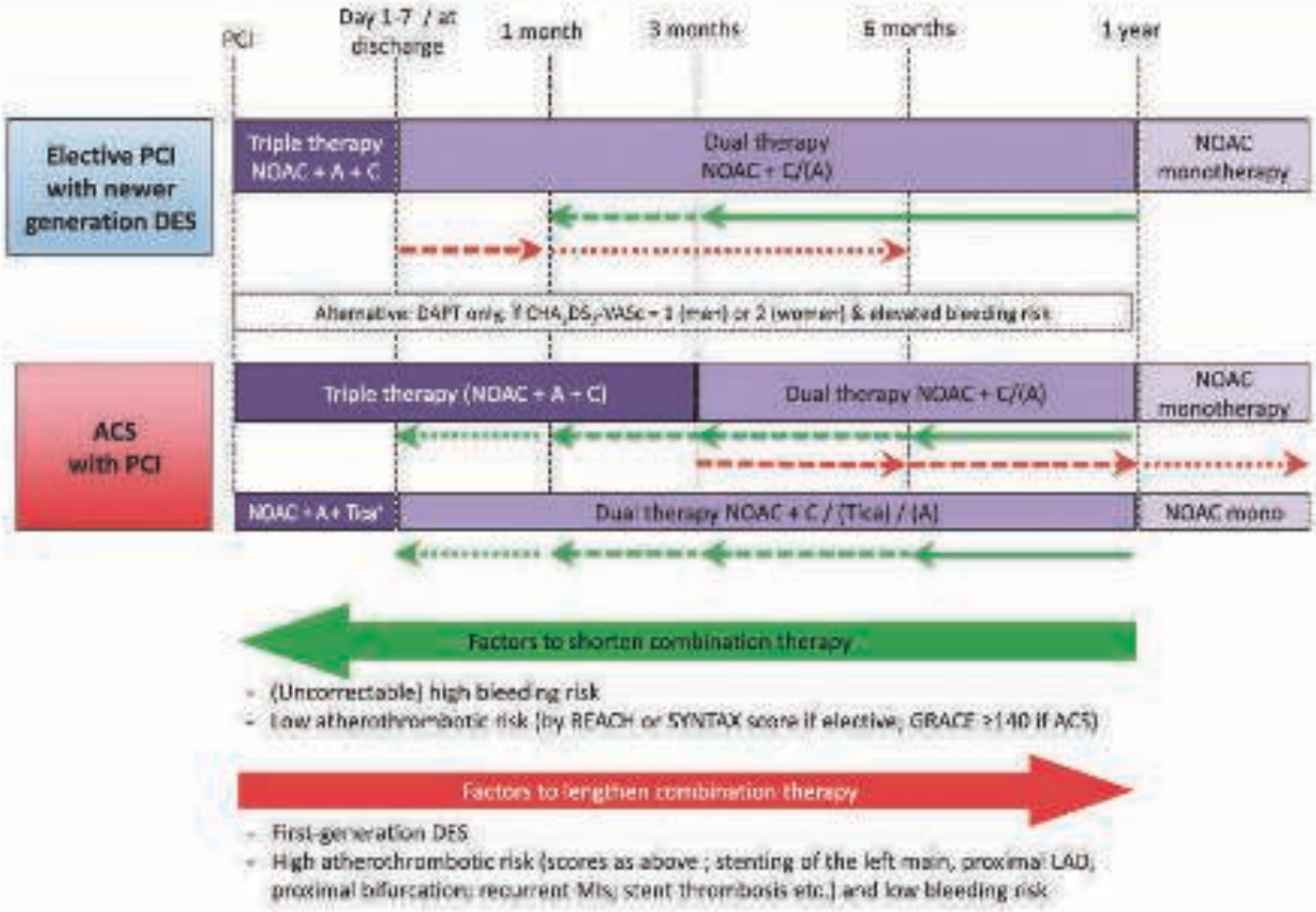
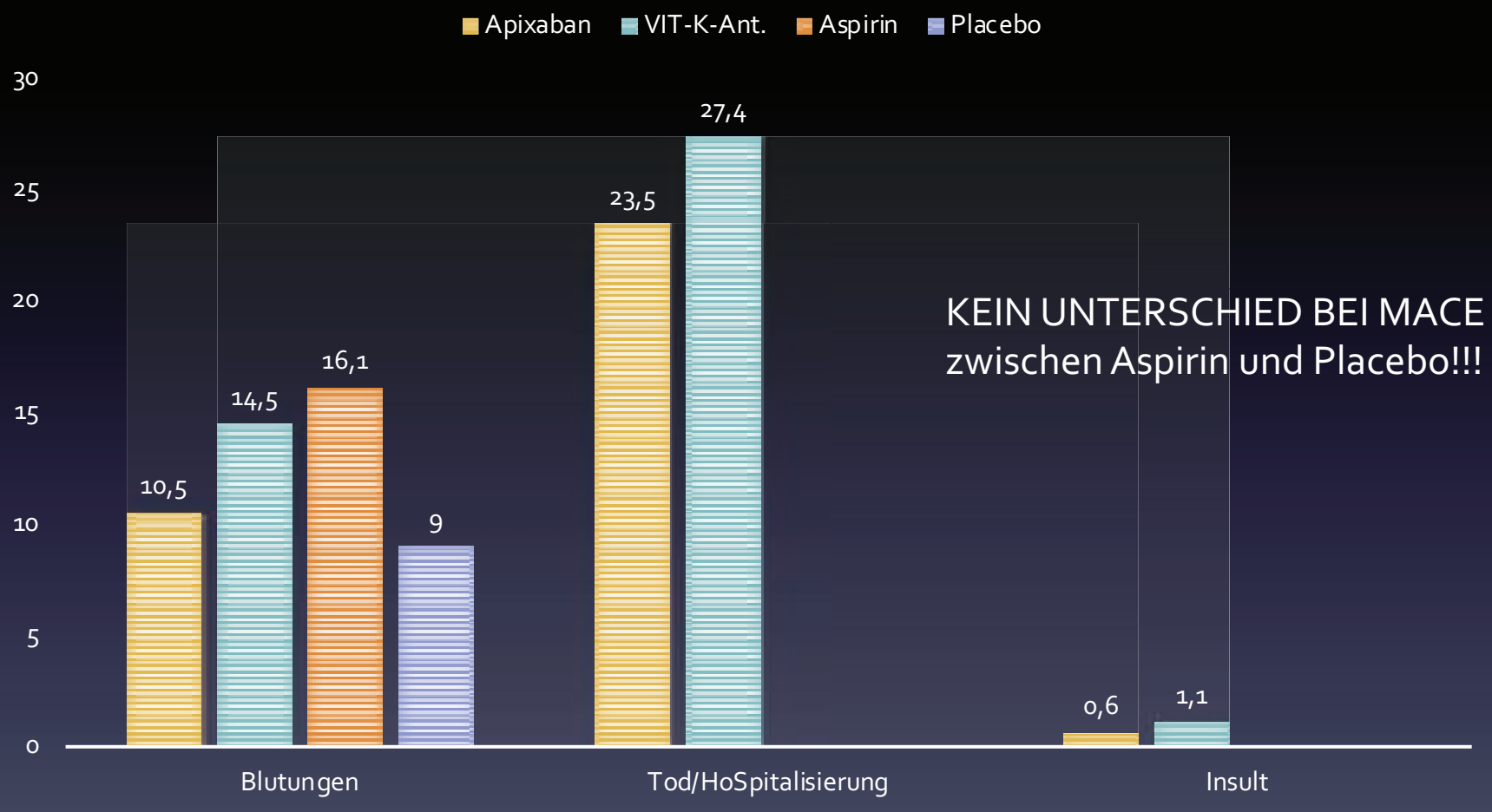


Figure 11 Long-term treatment of patients on non-vitamin K antagonist oral anticoagulant therapy after elective percutaneous coronary intervention or acute coronary syndrome. There are innumerable possible variations on this global theme, as discussed in the text. Patient characteristics and institutional practices should be taken into account to individualize the approach to each and every single patient. This figure wants to create a 'backbone' as guidance for such tailored approaches. A: aspirin 75–100 mg OD; C: clopidogrel 75 mg OD; Tica: Ticagrelor 90 mg BID. *If triple therapy needs to be continued after discharge clopidogrel is preferred over ticagrelor (due to lack of data).

AUGUSTUS TRIAL



KEIN UNTERSCHIED BEI MACE
zwischen Aspirin und Placebo!!!

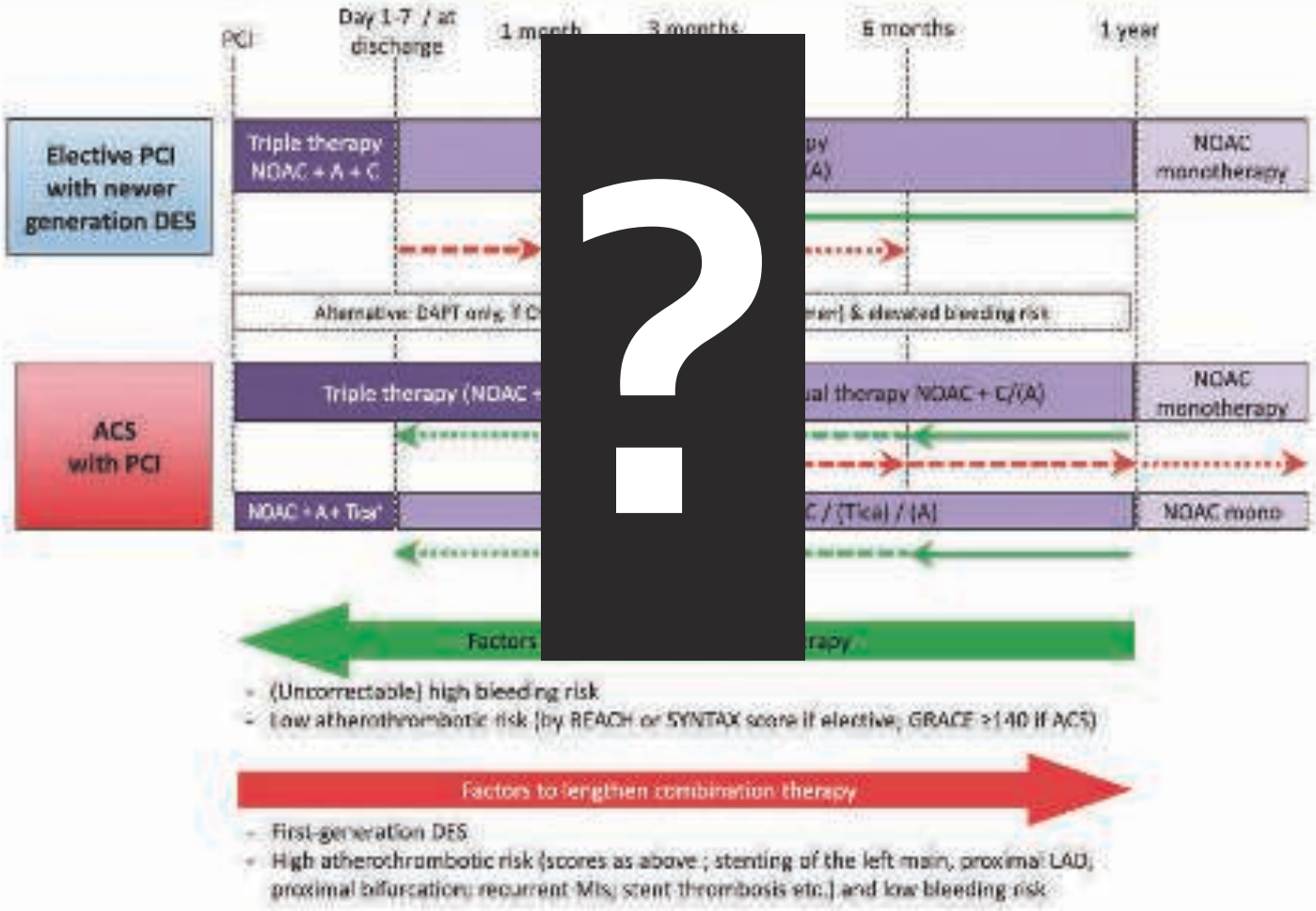
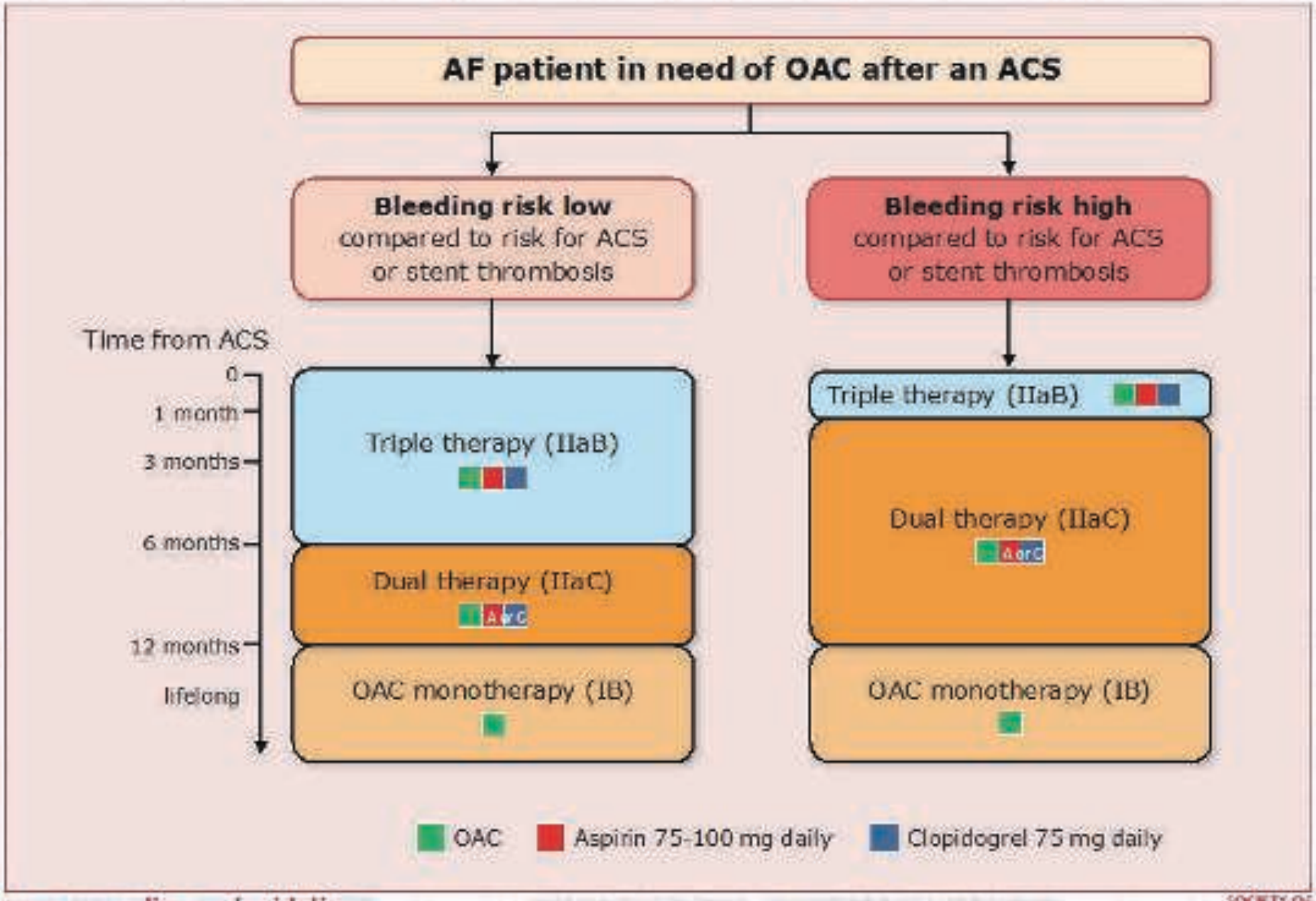
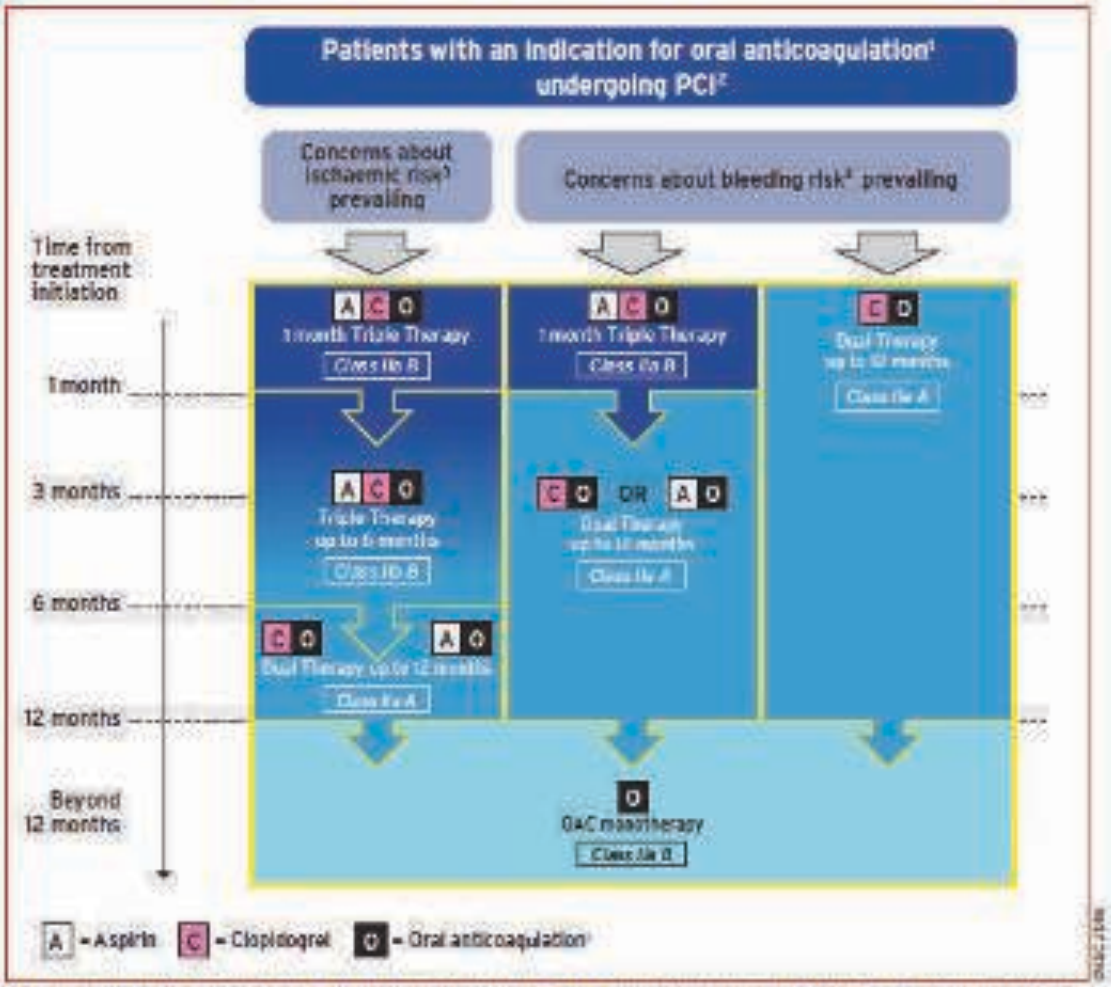


Figure 11 Long-term treatment of patients on non-vitamin K antagonist oral anticoagulant therapy after elective percutaneous coronary intervention or acute coronary syndrome. There are innumerable possible variations on this global theme, as discussed in the text. Patient characteristics and institutional practices should be taken into account to individualize the approach to each and every single patient. This figure wants to create a 'backbone' as guidance for such tailored approaches. A: aspirin 75–100 mg OD; C: clopidogrel 75 mg OD; Tica: Ticagrelor 90 mg BID. *If triple therapy needs to be continued after discharge clopidogrel is preferred over ticagrelor (due to lack of data).

Antithrombotic therapy after an acute coronary syndrome in atrial fibrillation patients requiring anticoagulation



European Heart Journal - doi:10.1093/eurheartj/ehw210
SOCIETY OF CARDIOLOGY



Clinical scoring refers to the number of concomitant antithrombotic medications. Triple therapy denotes treatment with DAPT plus oral anticoagulant (OAC). Dual therapy denotes treatment with a single antithrombotic agent (aspirin or clopidogrel) plus OAC.
ABC = Age, Biomarkers, Clinical history; AF = atrial fibrillation; HAS-BLED = Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly; VKA = vitamin K antagonist.
[1]Non-vitamin K antagonist oral anticoagulant (NOAC) preferred over VKA in patients with non-valvular AF. (Class I adv).
[2]Periprocedural administration of aspirin and clopidogrel during PCI is recommended irrespective of the treatment strategy.
[3]High ischaemic risk is considered as an acute clinical presentation or anatomical/procedural features which might increase the risk for myocardial infarction.
[4]Bleeding risk can be estimated by HAS-BLED or ABC score.

Figure 11 Algorithm for dual antiplatelet therapy in patients with an indication for oral anticoagulation undergoing percutaneous coronary intervention.

TAKE HOME Message

- Eine effektive Behandlung der arteriellen Hypertonie ist eine suffiziente Primärprävention hinsichtlich des Auftretens von Vorhofflimmern.
- Die Kriterien für eine OAK sind durch den CHA_2DS_2-VASc-Score klar definiert.
- Dies gilt auch für die Abschätzung des Blutungsrisikos durch den HAS-BLED-Score.
- Aktuell zur Diskussion stehen die Fragen hinsichtlich der optimalen Therapie:
  - des Vorhofflimmerns bei Herzinsuffizienz und
  - nach PCI