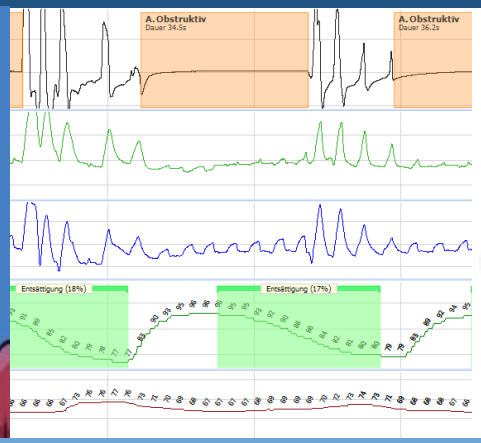
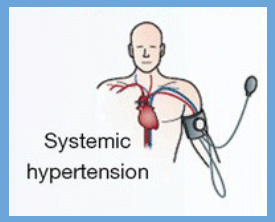
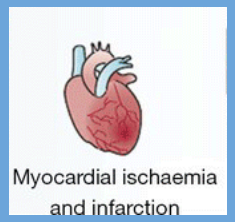
UNIVERSITÄT BERN

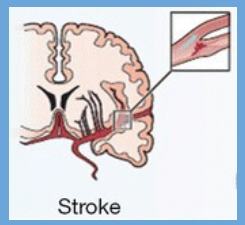
INSELSPITAL
Universitätsklinik für Pneumologie
Standorte Insel – Tiefenau

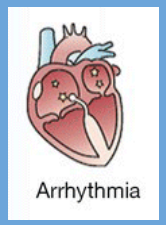
Vaskuläre Komplikationen des Schlaf Apnoe Syndroms

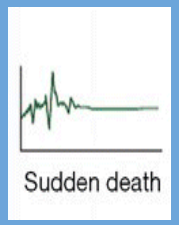


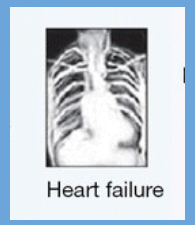






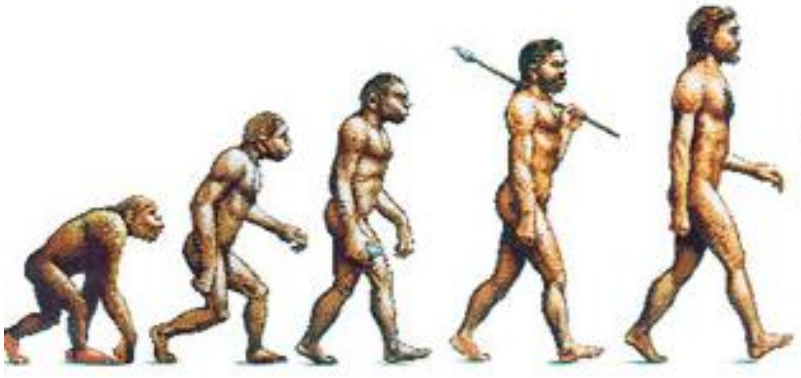






PD Dr. Sebastian R. Ott
Ärztlicher Leiter Schlaf- und Beatmungsmedizin, Universitätsklinik für Pneumologie, Inselspital

Das Problem der Evolution...



Agenda:

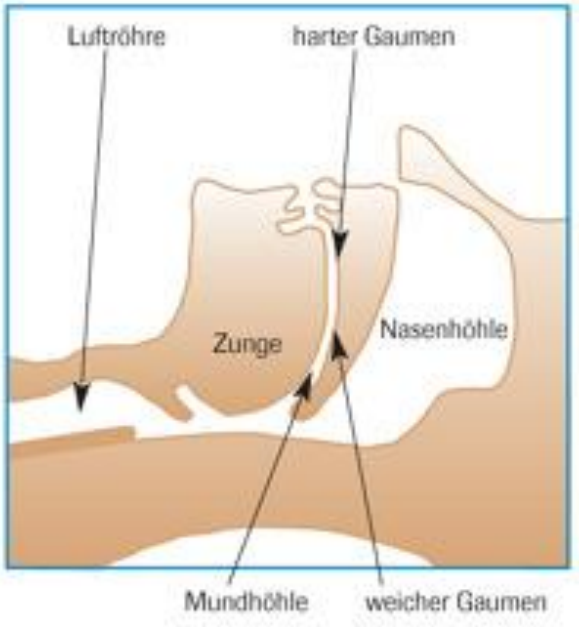
- Was ist Schlafapnoe?
- Pathophysiologie OSA und CV-Risiko
- Klinischer Zusammenhang OSA und CV-Risiko und Effekt von CPAP:
  - Observationsstudien
  - Randomisierte kontrollierte Studien (RCTs)
- Zusammenfassung

Der Pharynx – der Engpass?!

Funktionen:
Atmung, Sprechen, Schlucken:

- partiell kollaptischer „Muskelschlauch“
- partiell starre offene Röhre

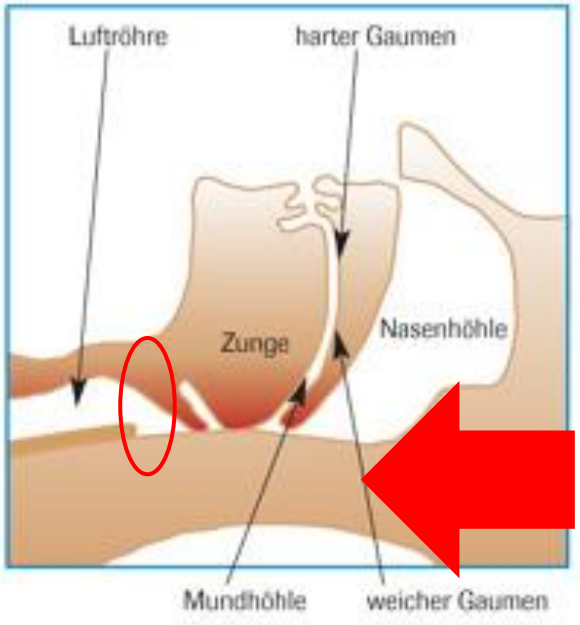
>20 Muskeln sind beteiligt



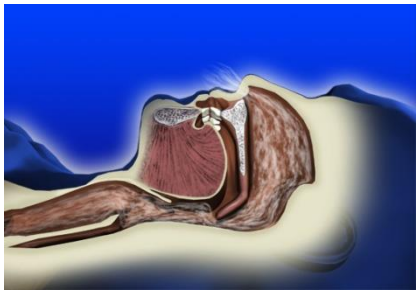
Was passiert beim Schlafen?

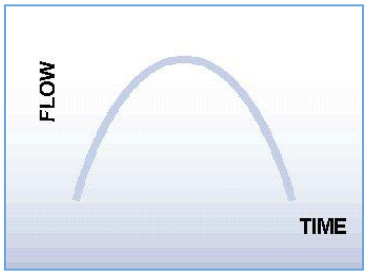
Erschlaffen der Muskulatur und des Weichteilgewebes im Hals / Rachenbereich.

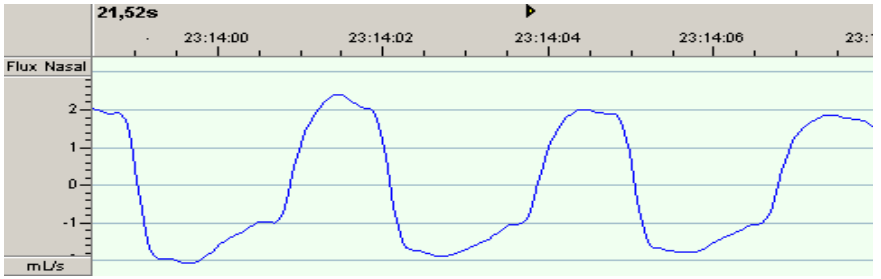
Obere Atemwege können zeitweise kollabieren
➔ Obstruktive Hypopnoe und Apnoe

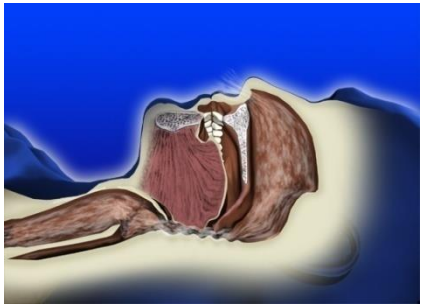


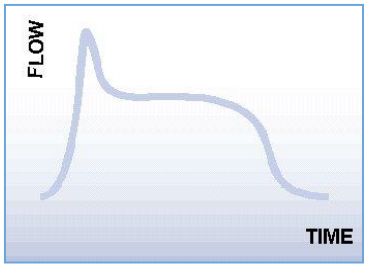
Einschränkung des Atemfluss im Schlaf

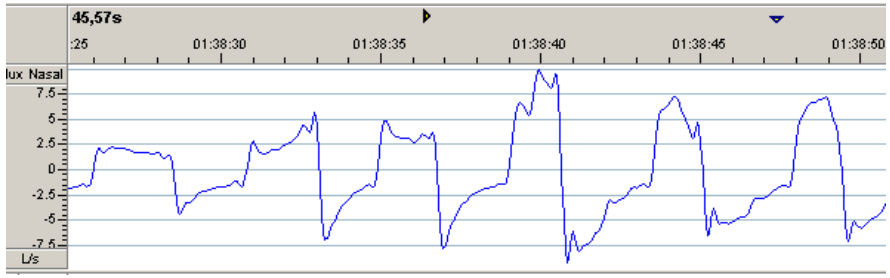


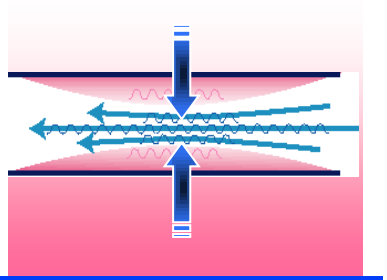


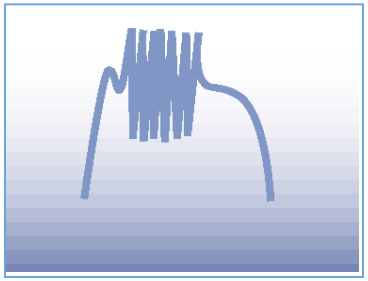


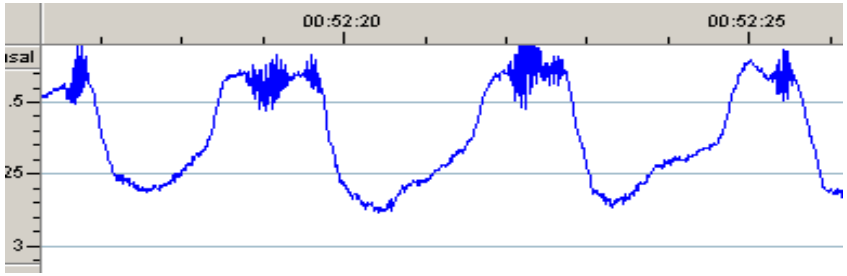


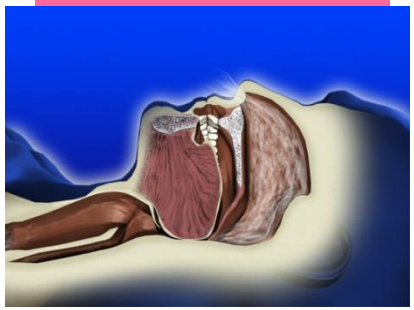


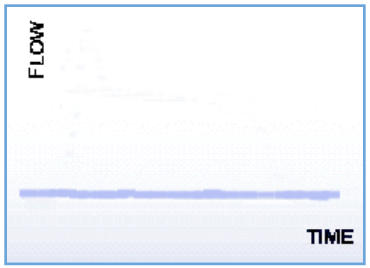


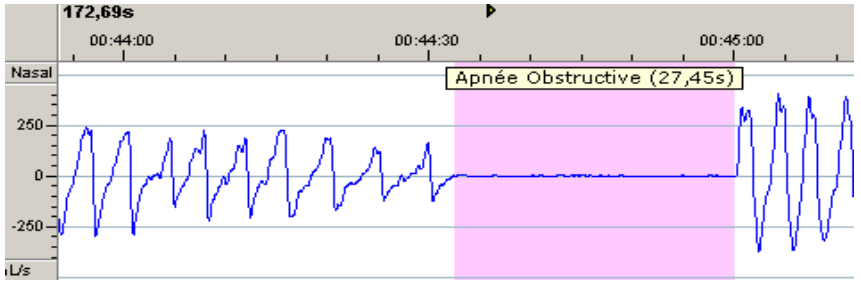


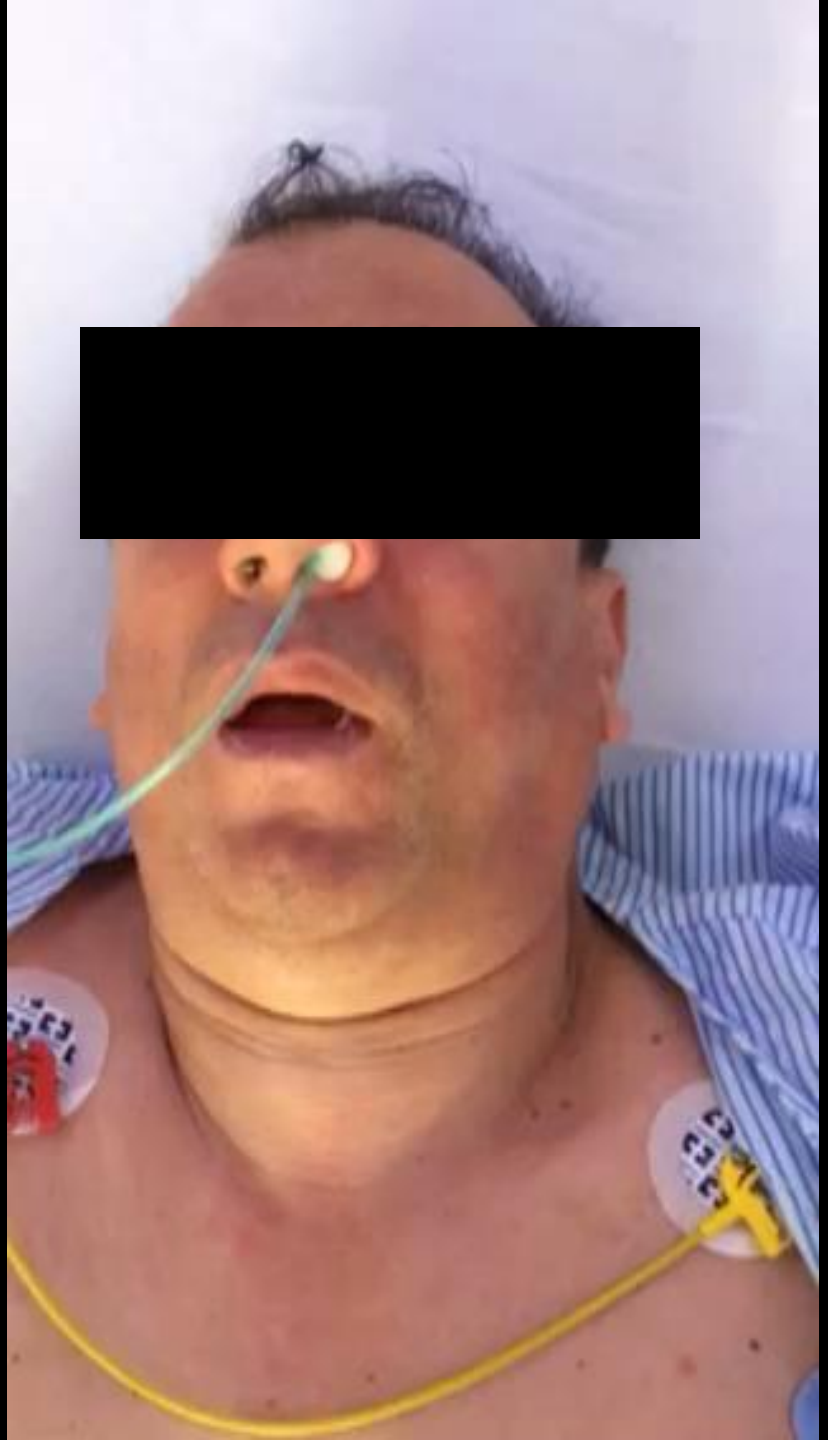




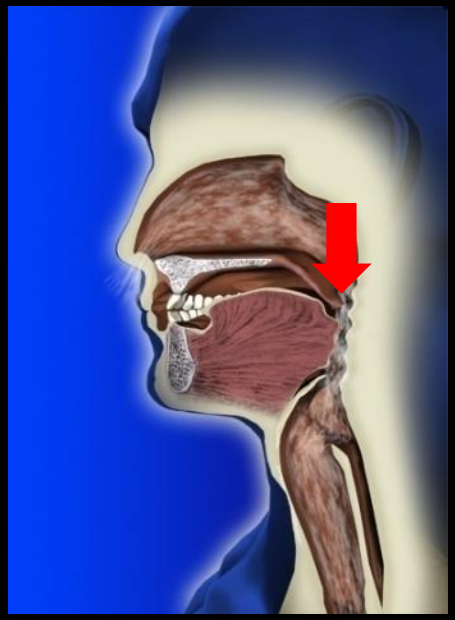


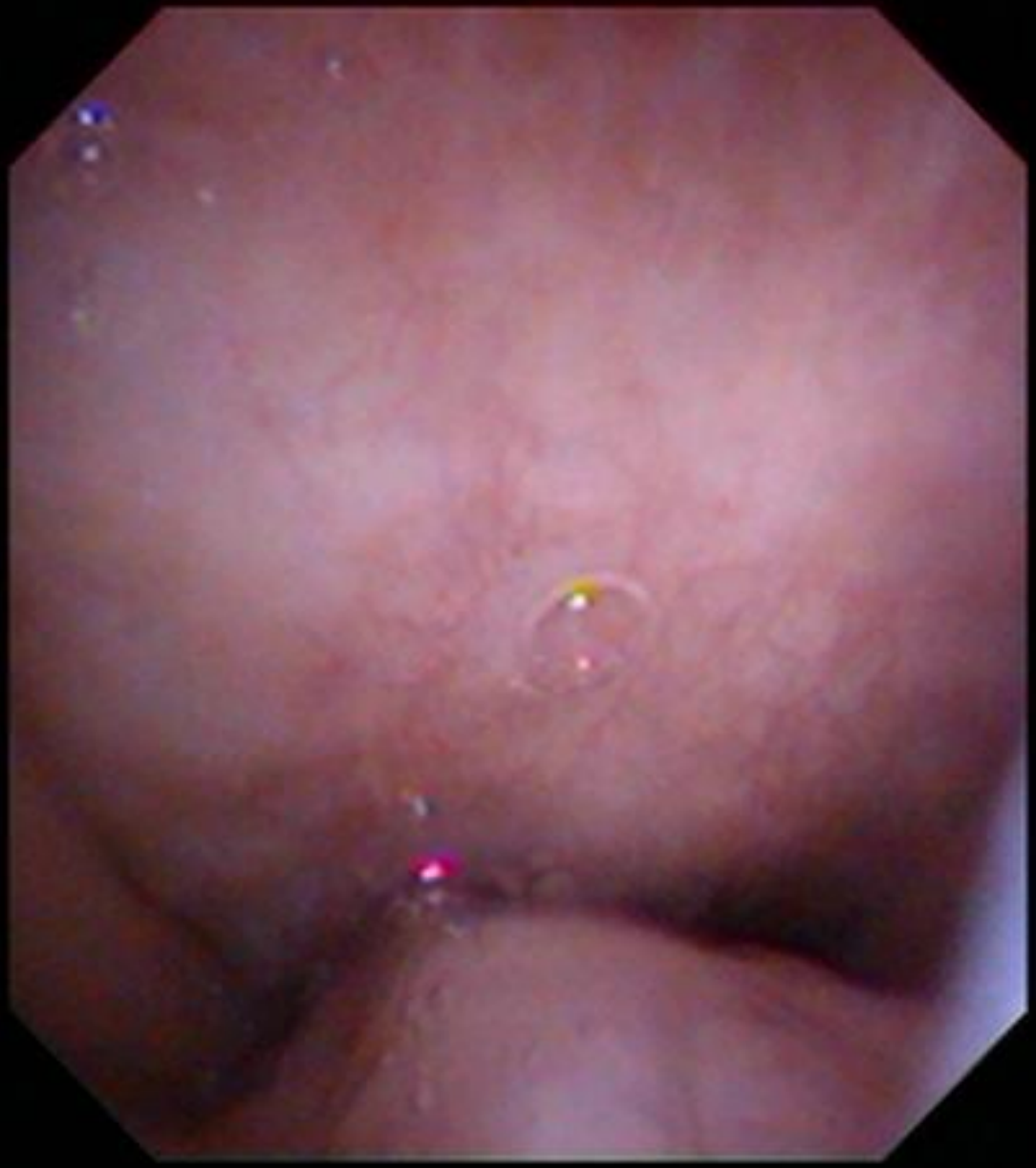






Obstruktive Hypopnoe und Schnarchen





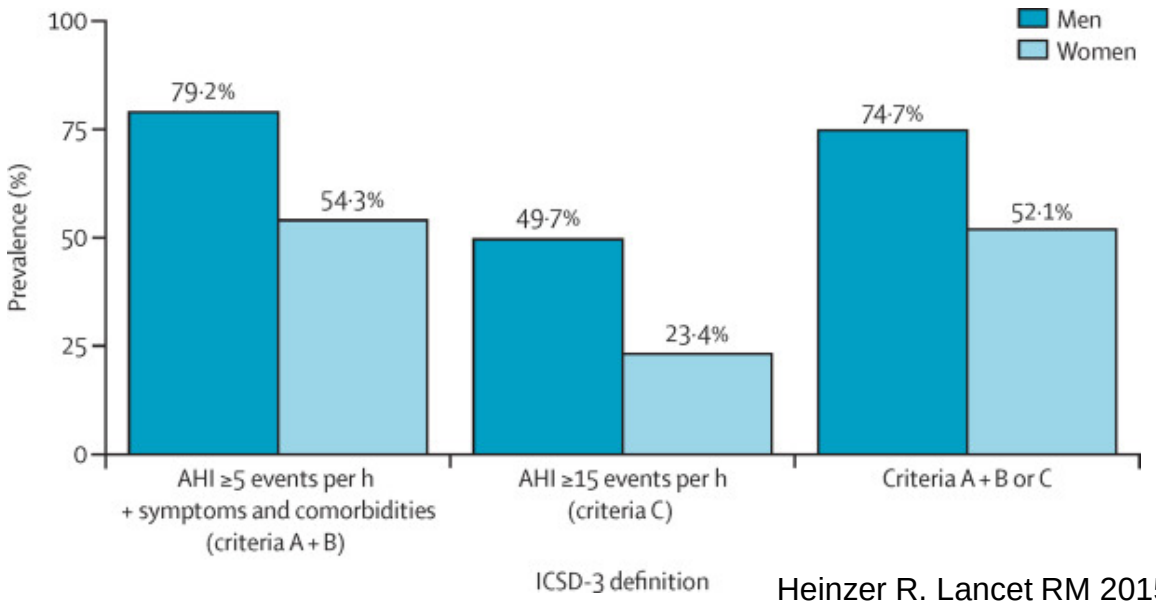
Die Prävalenz der OSA nimmt in der Allgemeinbevölkerung zu

F/U der Wisconsin Sleep Cohort Study (n=1520)

	Age	Prevalence in Men	Prevalence in Women
AHI ≥ 5	30-49	27%	9%
AHI ≥ 5	50-70	43%	28%
AHI ≥ 15	50-70	17%	9%

Peppard, Am J Epidemiol 2013; (Hypopnea criterium: RD 4%)

HypnoLaus cohort: 2121 Probanden mit home PSG



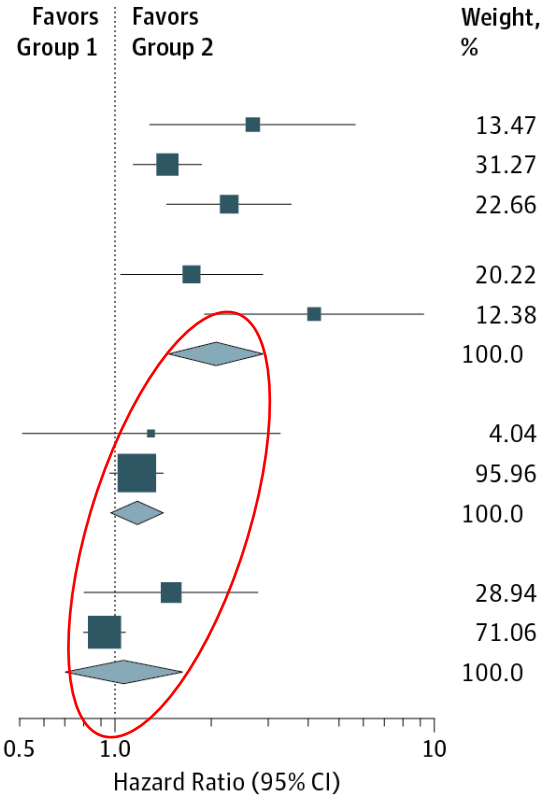
Heinzer R. Lancet RM 2015; (Scoring: 2012 AASM)

Screening for OSA in Adults. Evidence Report and Systematic Review for the US Preventive Services Task Force

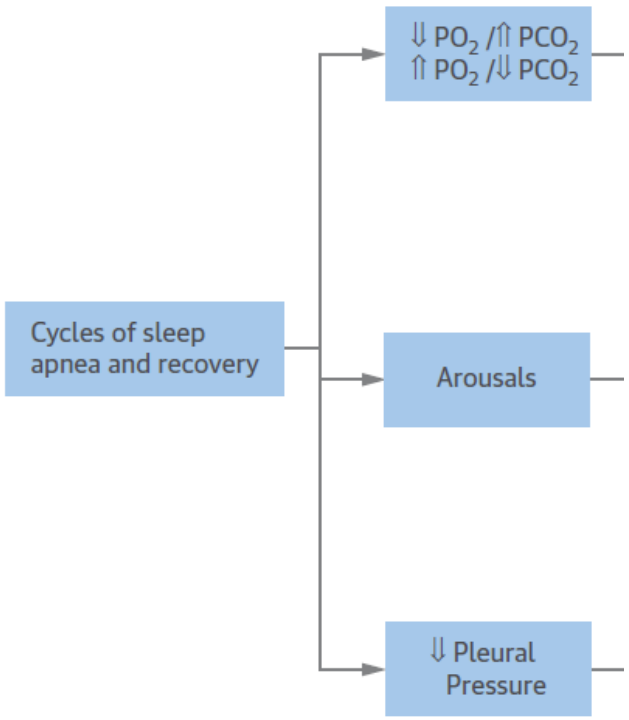
Jonas DE. et al JAMA 2017

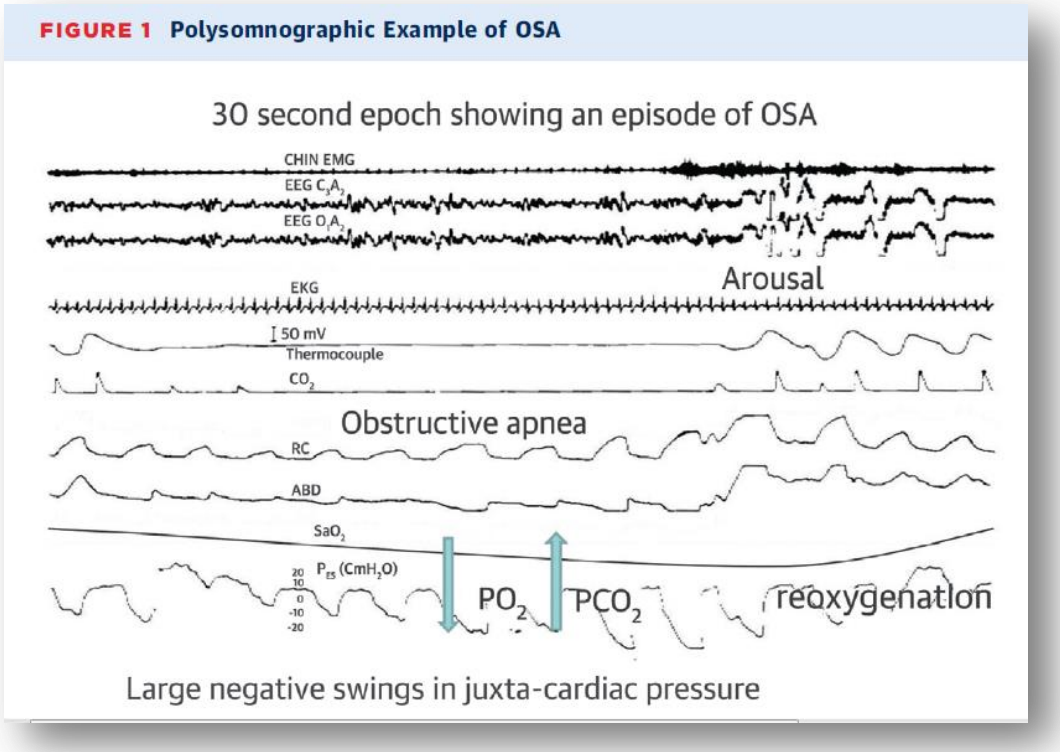
Es besteht ein Zusammenhang zwischen Apnoe-Hypopnoe-Index (AHI) und All-Cause Mortality

Source	No. of Deaths/ Total No. (%) Group 1	No. of Deaths/ Total No. (%) Group 2	Follow-up, y	Apnea-Hypopnea Index Comparison, Group 1 vs Group 2	Hazard Ratio (95% CI)	Weight, %
Severe OSA						
Young et al,[150] 2008	12/63 (19.0)	46/1157 (4.0)	13.8	≥30 vs <5	2.70 (1.29-5.65)	13.47
Punjabi et al,[151] 2009	86/341 (25.2)	477/3429 (13.9)	8.2	≥30 vs <5	1.46 (1.14-1.86)	31.27
Gooneratne et al,[147] 2011	35/42 (83.3)	59/119 (49.6)	13.8	≥20 and EDS vs <20 and no EDS	2.28 (1.46-3.57)	22.66
Ensrud et al,[144] 2012	25/209 (12.0)	155/2296 (6.8)	3.4	≥30 vs <30	1.74 (1.04-2.90)	20.22
Marshall et al,[153] 2014	10/18 (55.6)	54/294 (18.4)	20	≥15 vs <5	4.20 (1.91-9.24)	12.38
Subtotal (I^2 = 57.8%, P = .05)					2.07 (1.48-2.91)	100.0
Moderate OSA						
Young et al,[150] 2008	6/82 (7.3)	46/1157 (4.0)	13.8	15 to <30 vs <5	1.30 (0.51-3.29)	4.04
Punjabi et al,[151] 2009	165/727 (22.7)	477/3429 (13.9)	8.2	15 to <30 vs <5	1.17 (0.97-1.42)	95.96
Subtotal (I^2 = 0.0%, P = .83)					1.17 (0.97-1.42)	100.0
Mild OSA						
Young et al,[150] 2008	16/220 (7.3)	46/1157 (4.0)	13.8	5 to <15 vs <5	1.50 (0.80-2.81)	28.94
Punjabi et al,[151] 2009	319/1797 (17.8)	477/3429 (13.9)	8.2	5 to <15 vs <5	0.93 (0.80-1.08)	71.06
Subtotal (I^2 = 52.7%, P = .15)					1.07 (0.70-1.63)	100.0



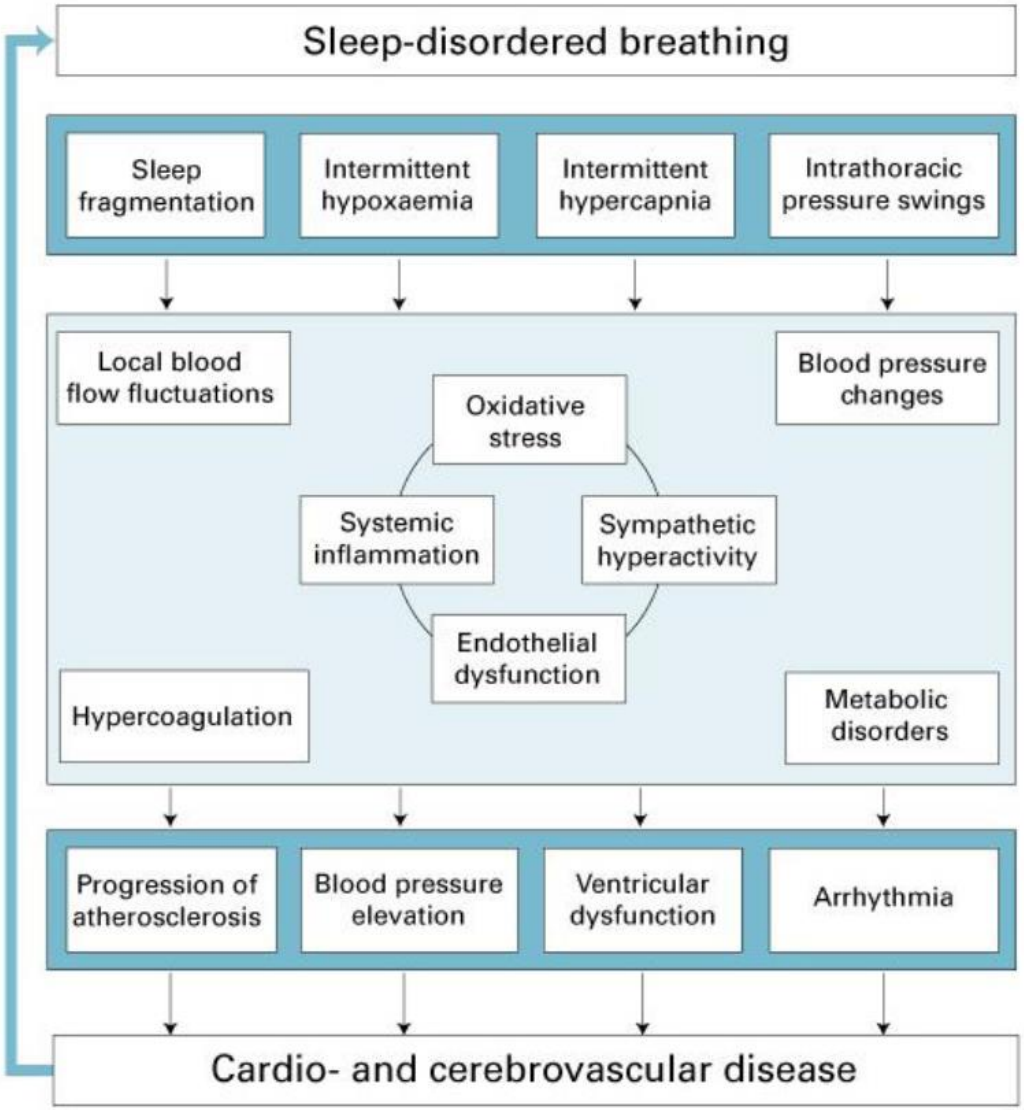
Pathophysiologie: Zusammenhang OSA und CV-Erkrankungen





Javaheri et al. *JACC* 2017;69:841-58

Pathophysiologie: Zusammenhang OSA und CV-Erkrankungen

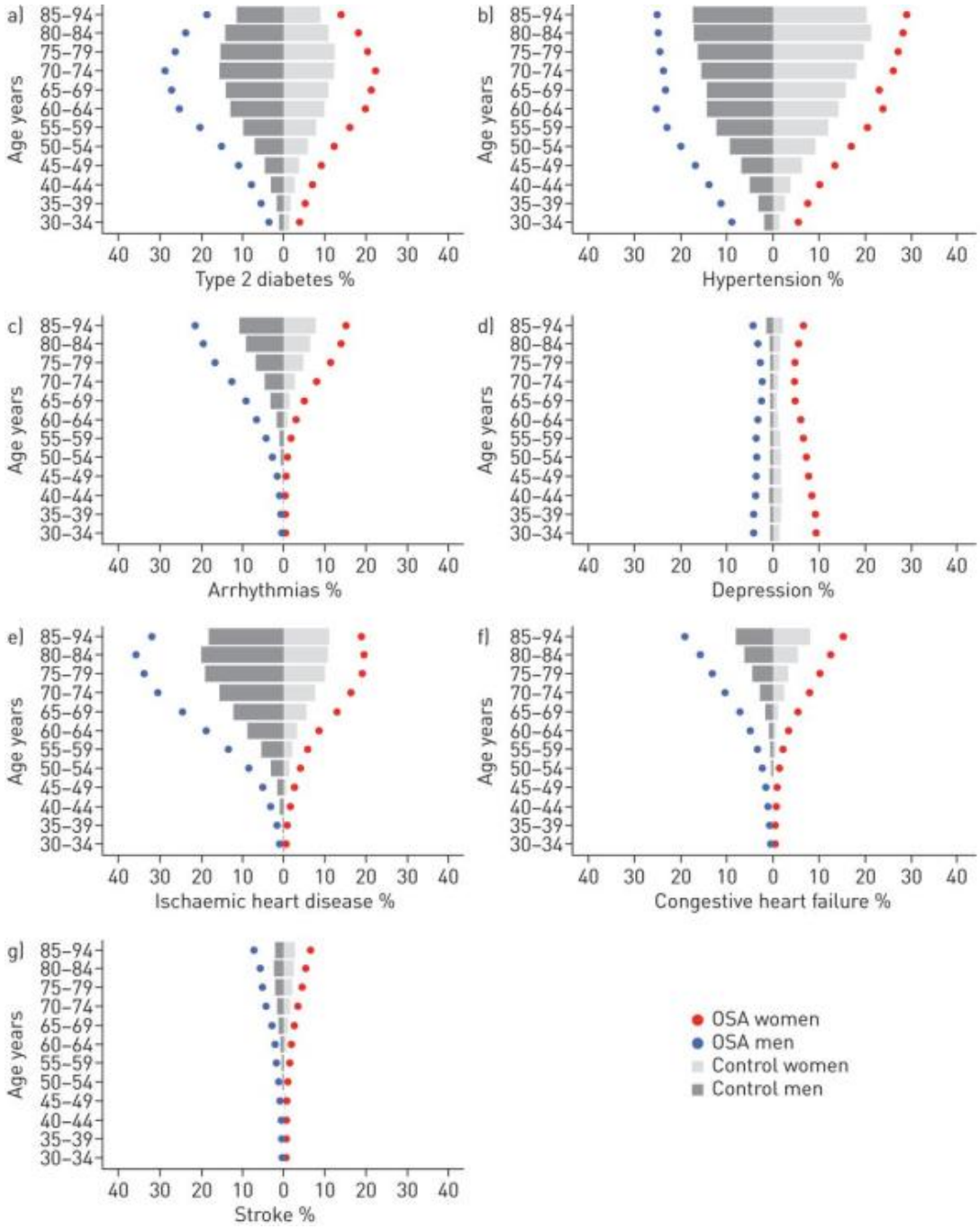


Ott SR et al. *Swiss Medical Weekly* 2017

Effect of sex and age on comorbidity in OSA: observational analysis from a nationwide US health claims database.

Mokhlesi B. ERJ 2016

- 1,704,905 Patienten mit OSA und gleich viele gematched Kontrollen
- Alle Co-Morbiditäten zeigten eine signifikant erhöhte Prävalenz bei Patienten mit OSA
- Prävalenz nahm zu mit Alter zu, insbesondere kardiovascular Erkrankungen, jedoch Abnahme der Depressionen
- Im adjustierten Model war die OR für alle Erkrankungen bei OSA erhöht



Prävalenz von schlafbezogenen Atemstörungen bei Patienten mit kardiovaskulären Erkrankungen

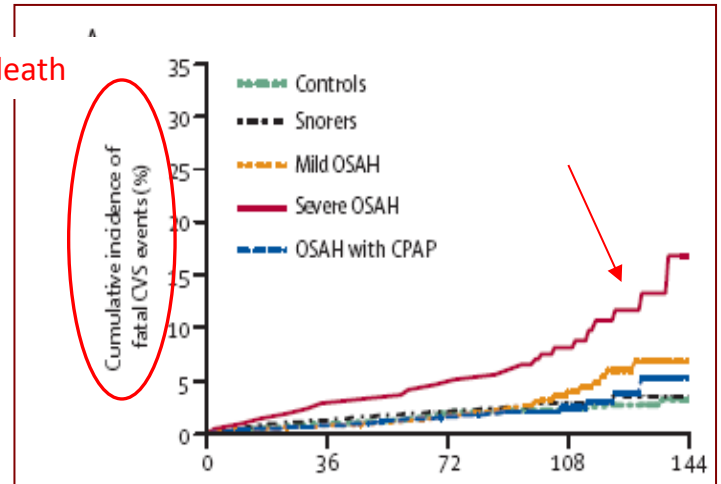
Pathology	Prevalence of SDB
Arterial hypertension	AHI ≥5/h: 58–74% AHI ≥ 15/h: 10–30%
Resistant arterial hypertension	AHI ≥ 5/h: 88% AHI ≥10/h: 60–83% AHI ≥30/h: 26–32%
Coronary artery disease (including acute myocardial infarction, post-revascularisation patients)	AHI ≥5/h: 83% AHI ≥10/h: 30–64% AHI ≥15/h: 64%
Congestive heart failure	AHI ≥10/h: 72% AHI ≥15/h: 60–64% AHI ≥20/h: 53% AHI ≥30/h: 36%
Heart rhythm and conduction disorders	AHI ≥5/h: 60–66% AHI ≥10/h: 59% AHI ≥15/h: 14–47% AHI ≥30/h: 20–27%
Atrial fibrillation	AHI ≥5/h: 70–74% AHI ≥10/h: 49% AHI ≥15/h: 25–43% AHI ≥30/h: 13%
Stroke and cerebrovascular disease	AHI ≥5/h: 79–86% AHI ≥15/h: 35–40%
Asymptomatic carotid stenosis	AHI ≥10/h: 69%
Pulmonary hypertension	AHI ≥10/h: 60% AHI ≥15/h: 42%

Ott SR et al. *Swiss Medical Weekly* 2017

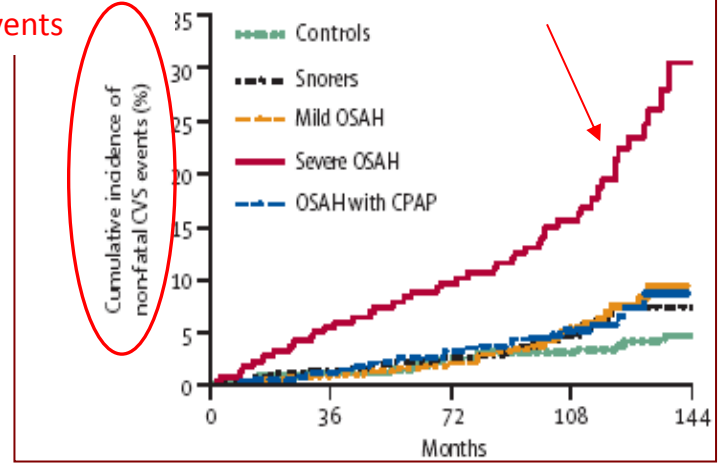
Association between OSA and CV mortality

Severe OSA, an independent risk factor of CV mortality, modifiable by CPAP therapy

CV death



Non fatal CV events



- Prospective observational trial, mean follow-up **> 10 years**.

  Patients untreated

  264 Control
  377 Snorer
  403 Mild to moderated OSA (AHI< 30)
  235 Sever OSA (IAH > 30)

  Patients treated

  372 OSA (AHI > 30 or AHI 5 to 30 with excessive daytime sleepiness

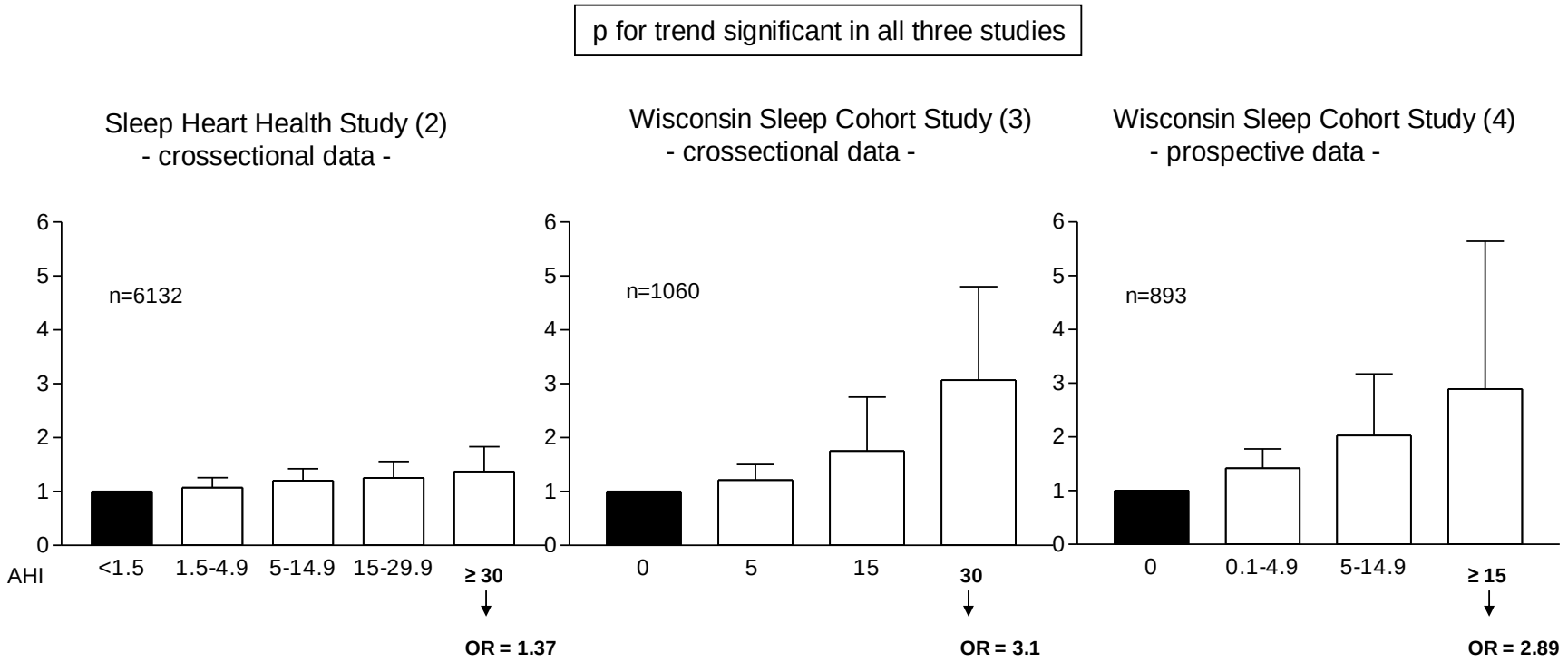
- **CV morbidity/mortality** increased for untreated severe OSA patients, compared to non OSA patient

  -Relative risk od death **2,87**
  -Relative risk of CV events **3,17**

- **Risk suppressed/normalized by CPAP therapy**

Jose M Marin, *Lancet* 2005

OSA und art. Hypertonus

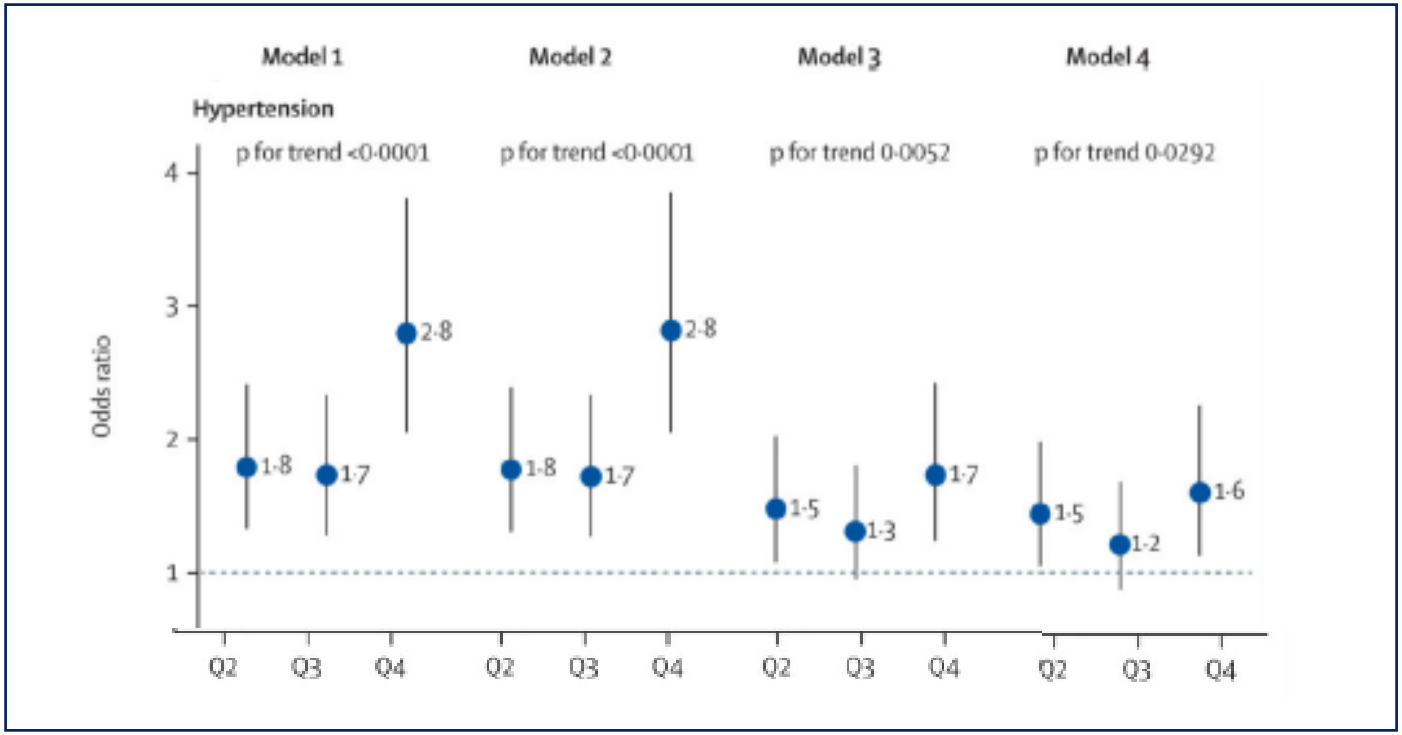


Legend of figure 1

Fully adjusted = odds ratio (OR) adjusted for confounders: age, sex, BMI, neck and waist circumference, alcohol, tabacco use and in case of the prospective study also baseline hypertension status; AHI = apnoea-hyponoea index; the graphics represent the OR and the upper 95% confidence interval

Prevalence of sleep-disordered breathing in the general population: the HypnoLaus study

R Heinzer, S Vat, P Marques-Vidal, H Marti-Soler, D Andries, N Tobback, V Mooser, M Preisig, A Malhotra, G Waeber, P Vollenweider, M Tafti*, and J Haba-Rubio*



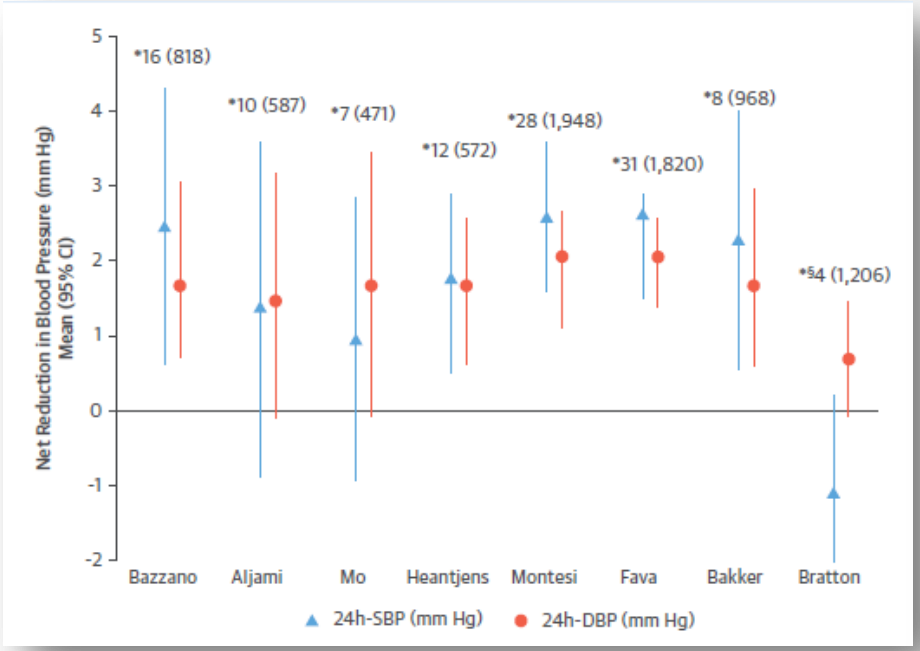
Q2 = AHI 4.3-9.9 Q3 = AHI 10-20.6 Q4 >20.6/h
Adj for age, sex, alcohol, tobacco (model 4) and +BMI (model 3)

Lancet Respir Med. 2015

Effekt von CPAP auf den Blutdruck bei Patienten mit Hypertonus

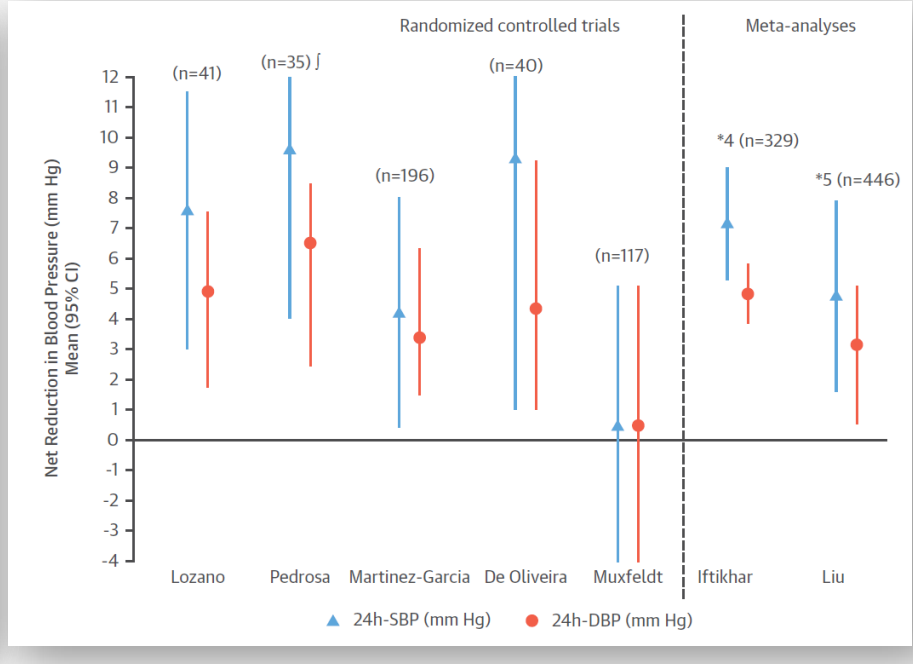
Ein langzeitig Absenkung des SBD um 2 - 3 mmHg führt zu einer 4 - 8% Reduktion des Risikos für zukünftige Schlaganfälle oder Herzinsuffizienz

HTN



SDP -2 to -2.5 mmHg
DBP -1.5 to -2 mmHg

Resistant HTN



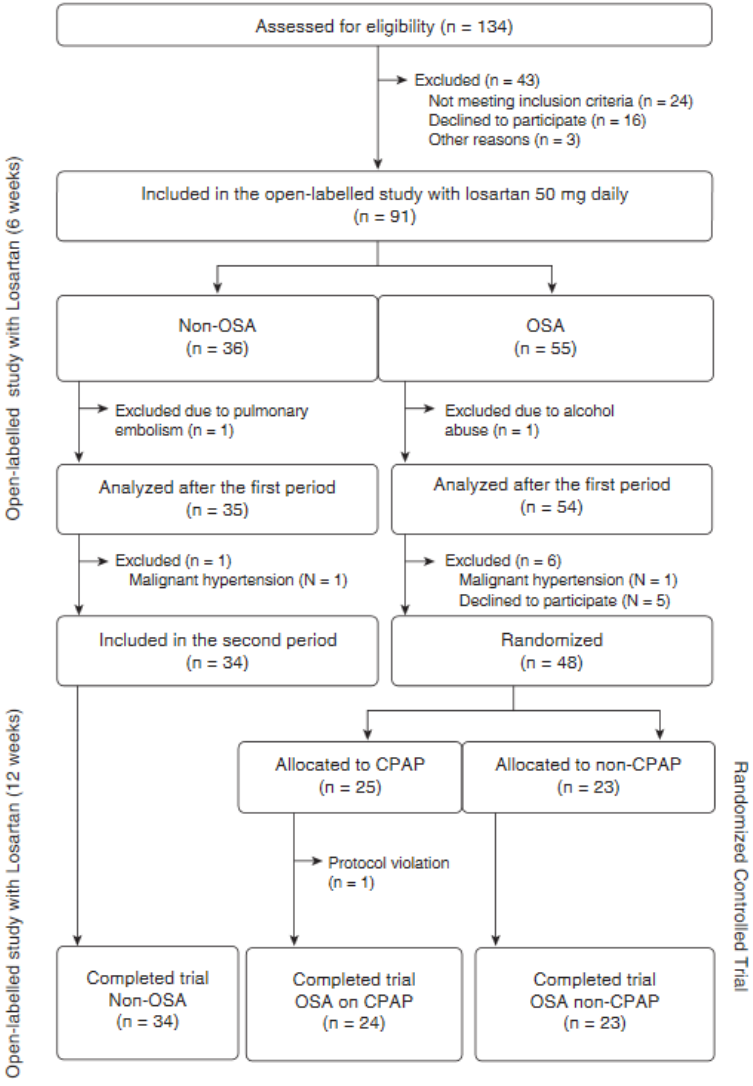
SDP -4.7 to -7.2 mmHg
DBP -2.9 to -4.9 mmHg

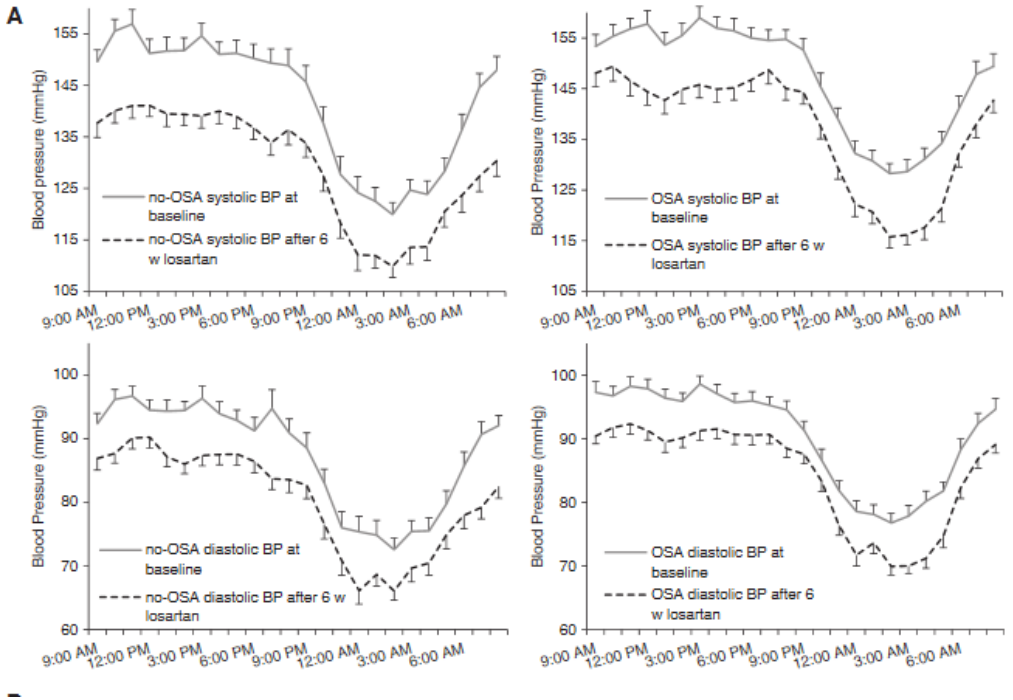
Javaheri et al. *JACC* 2017;69:841-58

Blood Pressure Response to Losartan and Continuous Positive Airway Pressure in Hypertension and Obstructive Sleep Apnea

Erik Thunström[1], Karin Manhem[1], Annika Rosengren[1], and Yüksel Peker[1,2]

Am J Resp Crit Care Med 2016





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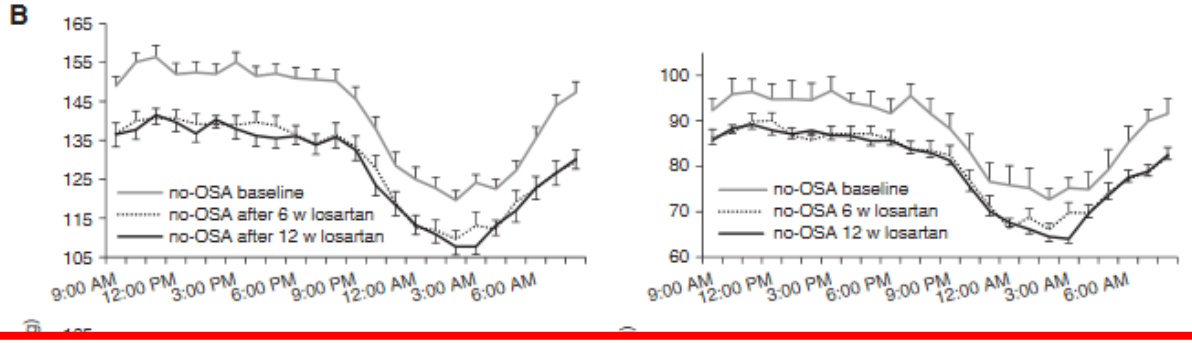
Am J Resp Crit Care Med 2016

No OSA

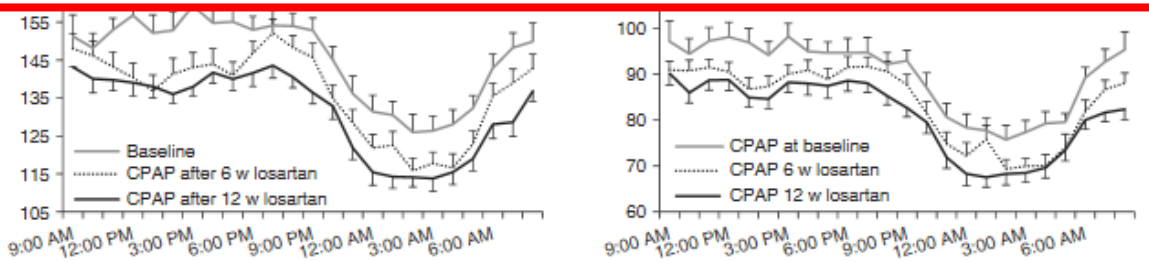


OSA
CPAP

Schlussfolgerung:

- Kombination einer anti-HTN Medikation mit CPAP hat einen synergistischen Effekt auf den Blutdruck
- HTN Pathophysiologie ist multidimensional in dieser Population

OSA
CPAP +



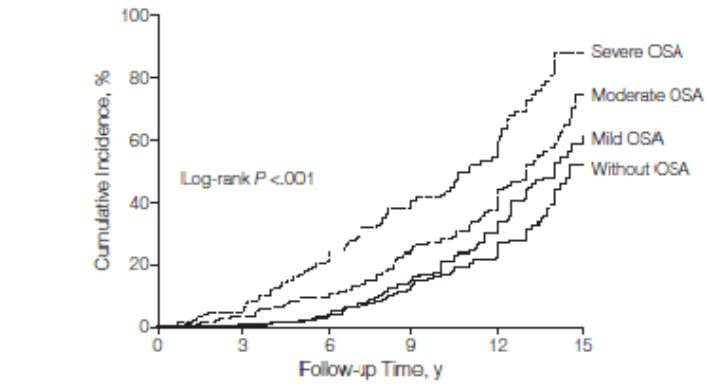
Mean nighttime BP					
Systolic					
At randomization	116.5 ± 12.9	120.2 ± 13.5	124.1 ± 13.2	0.335	
6 wk after randomization	114.9 ± 11.3	121.5 ± 14.4	119.4 ± 12.3	0.592	***5.9 (0.021)***
Mean morning BP					
Systolic					
At randomization	126.4 ± 16.3	133.8 ± 15.6	139.1 ± 16.1	0.245	
6 wk after randomization	126.4 ± 15.0	135.1 ± 19.1	131.2 ± 14.8	0.438	***9.1 (0.024)***

CPAP Behandlung senkt das Risiko eine Hypertonie zu entwickeln

Prospective cohort study of 1889 patients without HTN referred to a sleep lab mean follow-up 12y

Non treated OSA is significantly associated to a increased risk of incidence of HTN, related to the severity of OSA

Figure 2. Cumulative Incidence of Hypertension in Participants Without OSA and Untreated Patients With OSA



No. at risk

Severe OSA	199	184	141	119	62	37
Moderate OSA	258	222	202	162	114	67
Mild OSA	298	289	260	194	127	59
Without OSA	310	306	269	211	152	72

OSA indicates obstructive sleep apnea. Severity of OSA was defined by the apnea-hypopnea index (AHI) as mild OSA (AHI, 5.0-14.9), moderate OSA (AHI, 15.0-29.9), and severe OSA (AHI, ≥30.0). *P* value reflects an overall log-rank χ^3 test, providing an overall survival difference among the 4 study groups.

CPAP therapy is associated with a significant decreased of incidence of HTN

Table 2. Crude Rates of Incident Hypertension in Controls and Patients With Treated and Untreated OSA

		Patients With OSA			
	Controls[a] (n = 310)	Ineligible for CPAP Therapy (n = 462)	Declined CPAP Therapy (n = 195)	Nonadherent to CPAP Therapy (n = 98)	Treated With CPAP Therapy (n = 824)
AHI at baseline, mean (SD)	2.6 (1.3)	14.2 (6.6)	37.1 (16.3)	31.3 (13.4)	41.2 (19.9)
Incident hypertension, No. (%)	78 (25)	175 (38)	119 (61)	53 (53)	280 (34)
Total No. observed, person-years	3563	5239	2037	1015	9149
Crude incidence rate, No. per 100 person-years (95% CI)	2.19 (1.71-2.67)	3.34 (2.85-3.82)	5.84 (4.82-6.86)	5.12 (3.76-6.47)	3.06 (2.70-3.41)
P value		<.001	<.001	<.001	.003

Abbreviations: AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure; OSA, obstructive sleep apnea.
[a] Participants without OSA were controls. *P* values were calculated from 2-sided log-rank test comparing each of the patients with OSA groups with the control group.

Marin JM et al, *JAMA* 2012; 307(20):2169-76

Klinsch-observative Studien zeigen, dass das OSA-assoziierte CV-Risko durch CPAP normalisiert wird

	Exposure	Outcome	HR (untreated)	HR (treated)
Peker 2002	AI ≥5	MI, stroke, CV death	7.7	1.0
Marin 2005	AHI ≥30	Non-fatal MI, stroke	2.4	1.1
Campos-Rodriguez 2014	AHI ≥10	Incident MI, stroke	2.8	0.9
Campos-Rodriguez 2014	AHI ≥10	Incident stroke	6.4	1.3
Martinez-Garcia 2012	AHI ≥30	Recurrent stroke	2.0	0.9

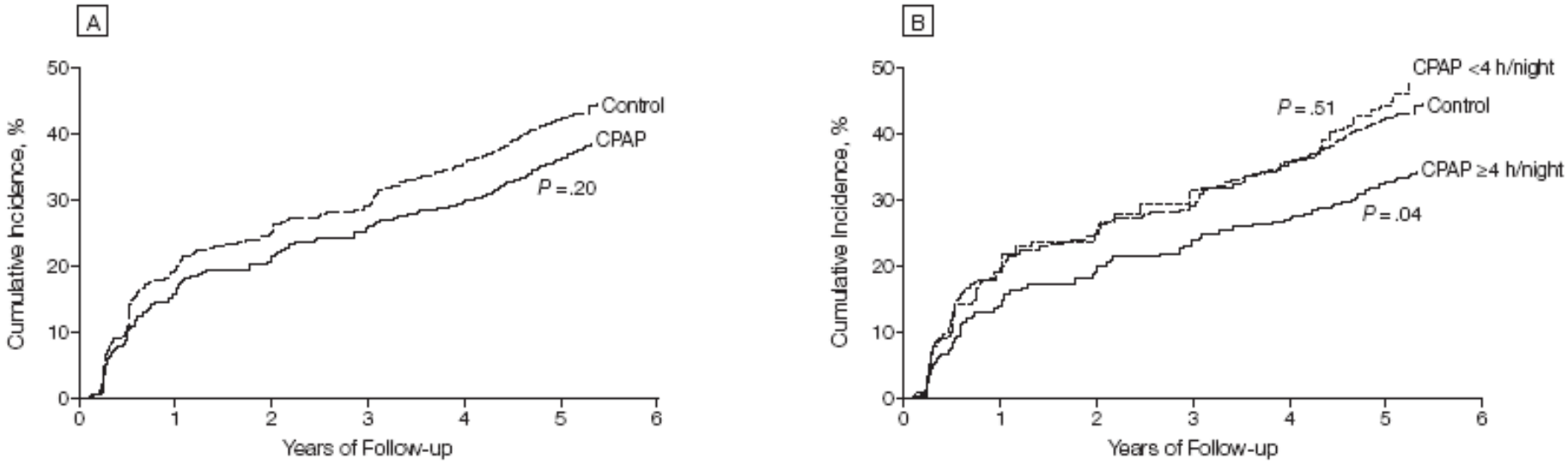
Zwischenbilanz…

- OSA Prävalenz ist hoch bei Patienten CV-Erkrankungen
- Observationsstudien deuten eindeutig auf ein erhöhtes, schwergrad-abhängiges CV-Risiko durch OSA hin
- CPAP kann den Blutdruck senken (RCTs)
- Longitudinale Observationsstudien (nicht-randomisiert!) zeigen klar eine CPAP-assoziierte Senkung des CV Risikos und verbessertes Überleben

So, wie sieht es mit prospektiven RCTs aus?

Incidence of HTN or CV events in non sleepy OSA patients (CERCAS trial)

Figure 2. Cumulative Incidence of Hypertension or Cardiovascular Events During Follow-up



- **Multicenter RCT with 725 patients with AHI>20 without sleepiness** (ESS≤10)
- In this cohort of non-sleepy patients (50% with initial HTN whom 25% treated for HTN), no beneficial effect of CPAP on incident HTN or CV events.
- In post hoc analysis, compliance > **4h/night leads to significant decrease**

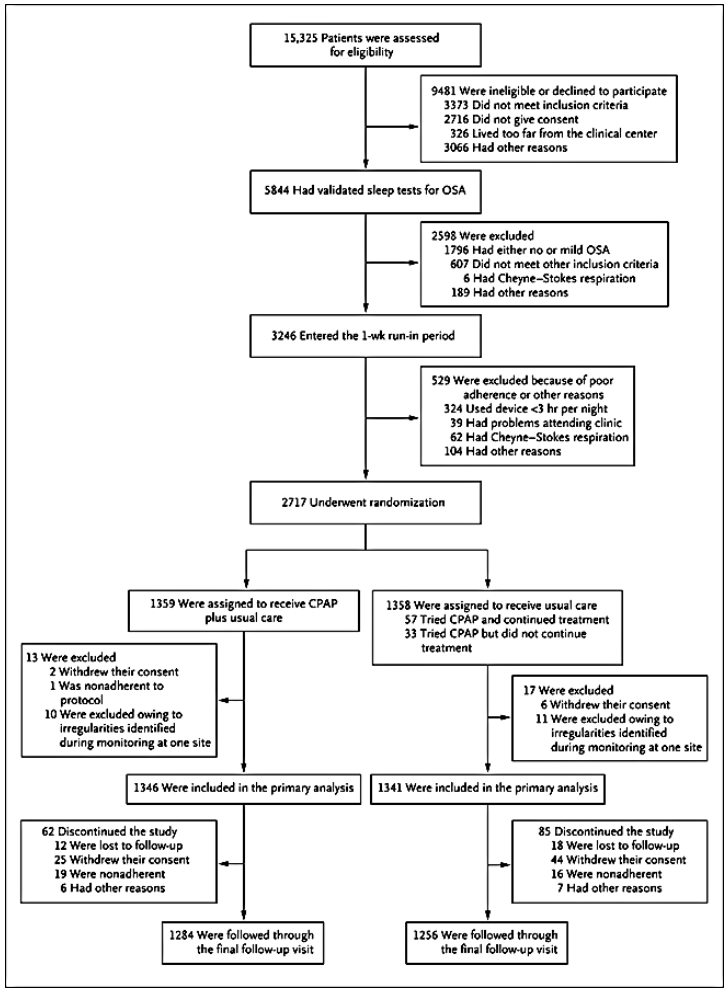
Barbé et al, *JAMA* 2012; 307(20):2161-8

CPAP for Prevention of Cardiovascular Events in OSA

McEvoy RD et al. N Engl J Med. 2016 Sep 8;375(10):919-31.

"SAVE-Trial"

Non-sleepy patients with pre-existing coronary or cerebrovascular disease



Secondary prevention trial
(almost half with prior stroke)

- 5844 sleep tests
- 3246 eligible for run-in (AHI ≥12)
- 2717 randomized (sham adherence ≥3 hrs/night)

- Mean AHI 29/hour
- Mean ESS 7.4

Mean f/u 3.7 years

Mean CPAP use 3.3 hours/night

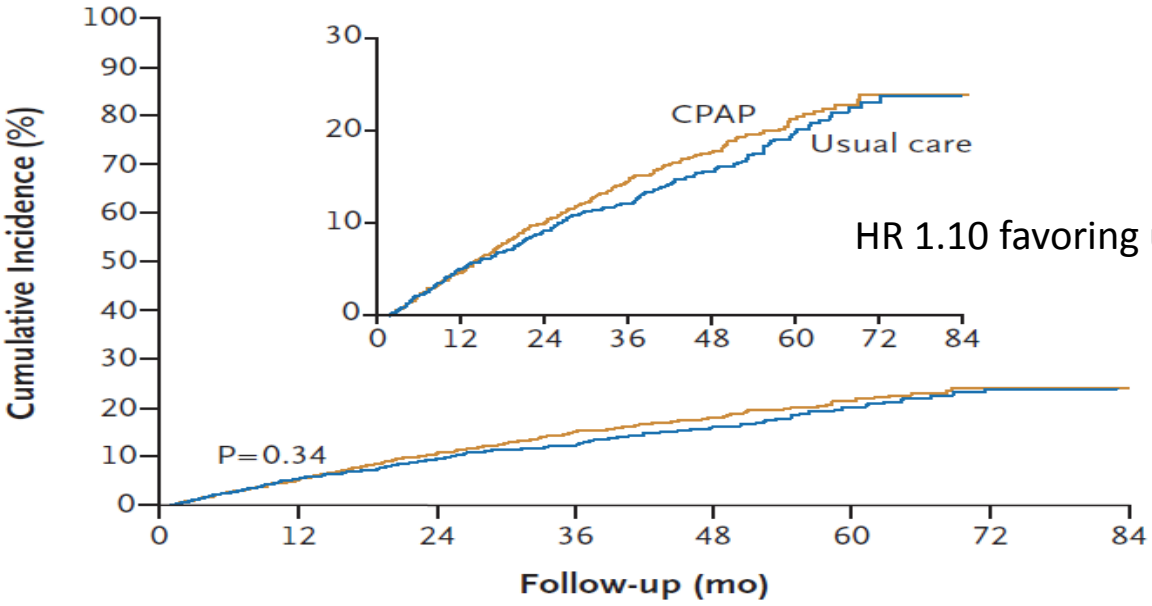
Primary outcome:
CV death, MI, stroke, or hospitalization for heart failure, unstable angina or TIA

McEvoy RD et al. N Engl J Med. 2016

CPAP for Prevention of Cardiovascular Events in OSA

McEvoy RD et al. N Engl J Med. 2016 Sep 8;375(10):919-31.

"SAVE-Trial"



HR 1.10 favoring usual care

No. at Risk								
CPAP	1346	1222	1118	754	482	278	146	146
Usual care	1341	1211	1108	727	499	290	103	103

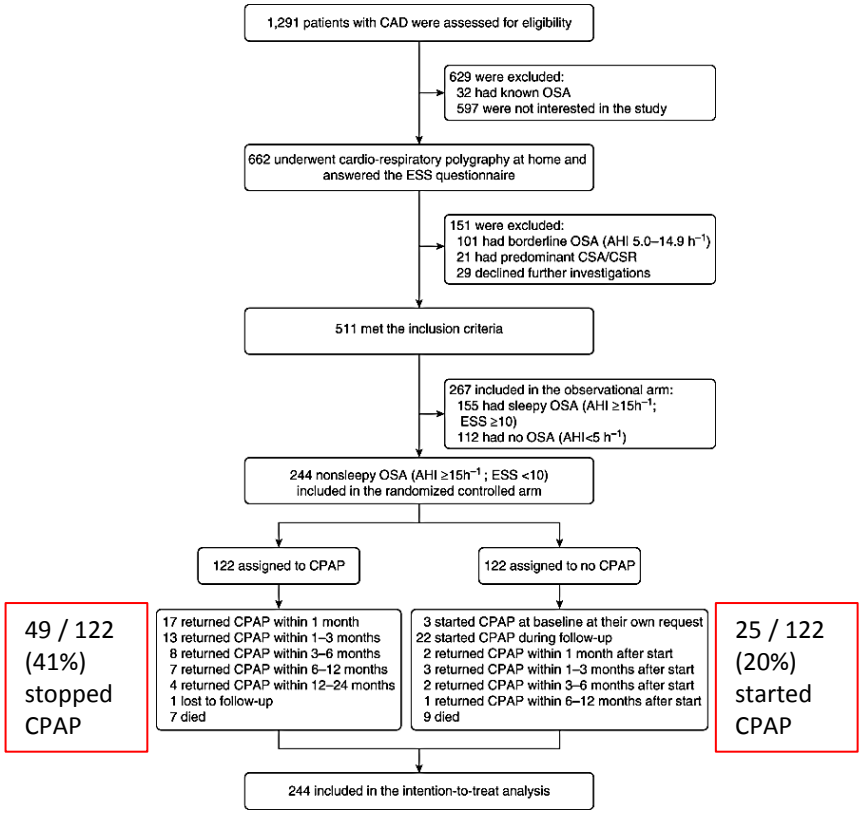
CVD Endpoints
229
207

Primary endpoint neutral. Average compliance only 3.3 hrs.
CPAP treated patients showed a significant drop in ESS, QoL, mood and in diastolic BP

McEvoy RD et al. N Engl J Med. 2016

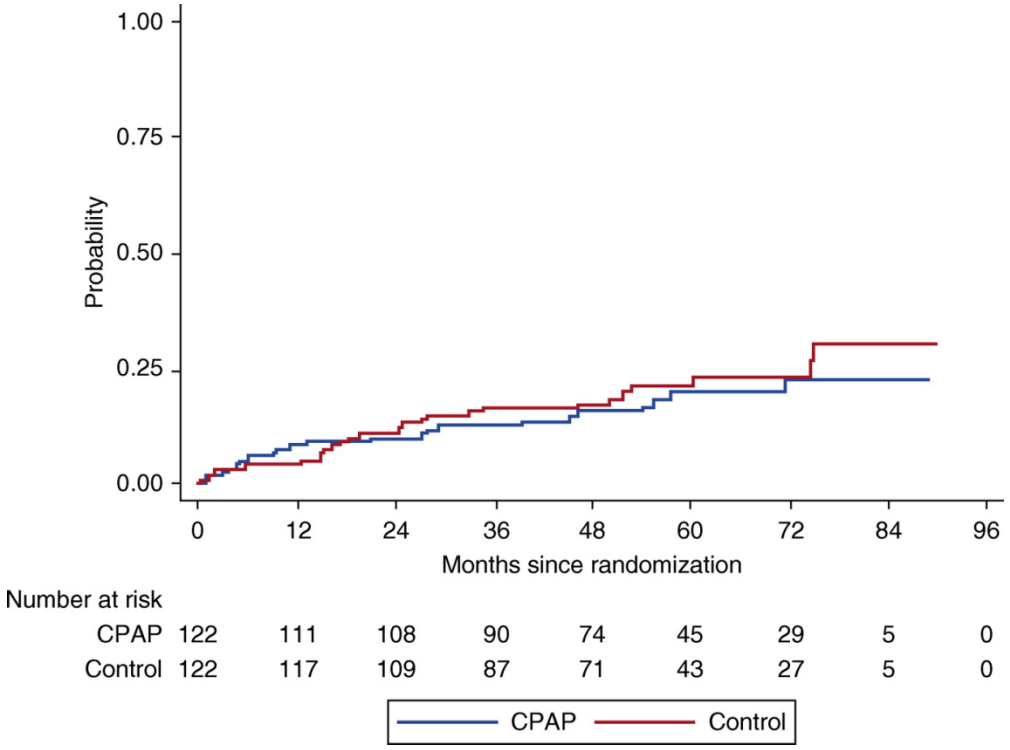
Effect of CPAP on Cardiovascular Outcomes in Coronary Artery Disease Patients with Nonsleepy OSA. The RICCADSA Randomized Controlled Trial.

Peker Y et al. Am J Respir Crit Care Med. 2016 Sep 1;194(5):613-20



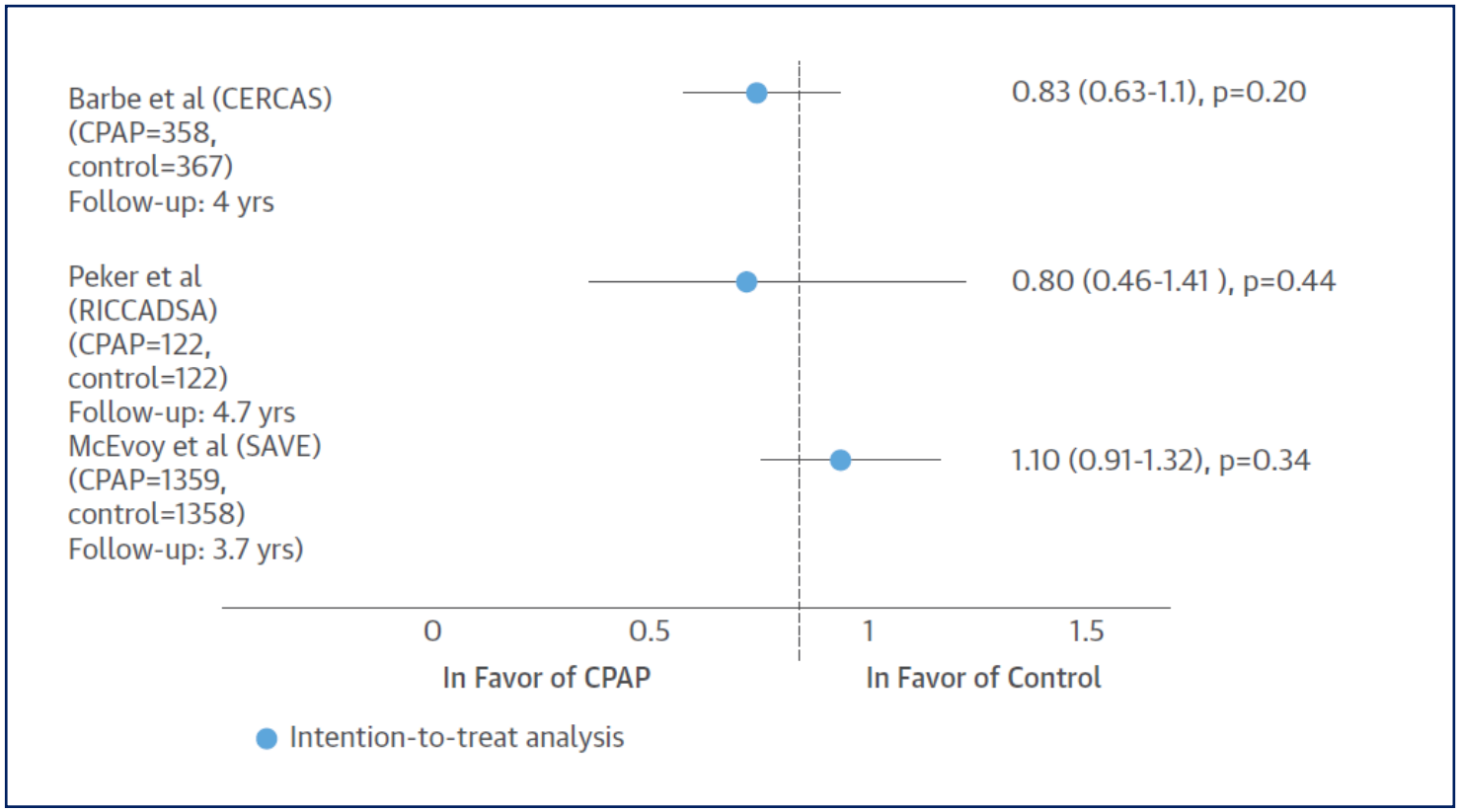
49 / 122 (41%) stopped CPAP

25 / 122 (20%) started CPAP



Schlussfolgerung: Keine Verbesserung des CV Outcome bei den CPAP behandelten Patienten. Aber, signifikanter Vorteil der CPAP-Therapie bei Patienten mit guter Compliance (>4 h/Nacht): HR 0.29 (CI 0.10-0.86 on multivariate analysis, P=0.026)

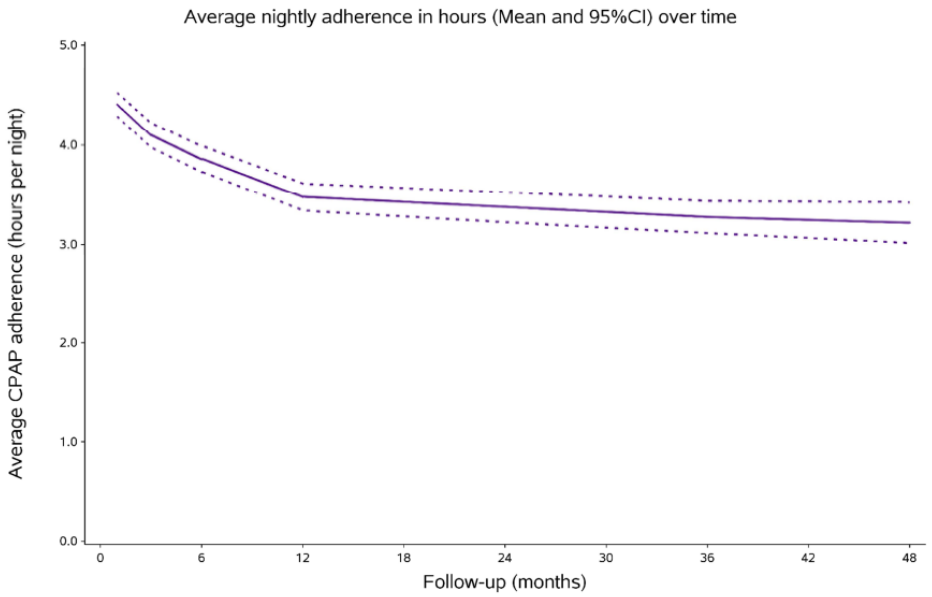
Zusammenfassung RCTs: CPAP-Effekt vs. Kontrolle



Javaheri et al. *JACC* 2017;69:841-58

Warum zeigte sich in den RCTs kein Vorteil für CPAP?

- Vielleicht reduziert CPAP das CV-Risiko gar nicht?
- Inadequate CPAP Nutzung?
  z.B. Adherence im SAVE-Trial:



Means hours of use per night at:

Sham run-in	5.2 hrs
1 month	4.4 hrs
12 months	3.5 hrs
Total study	3.3 hrs

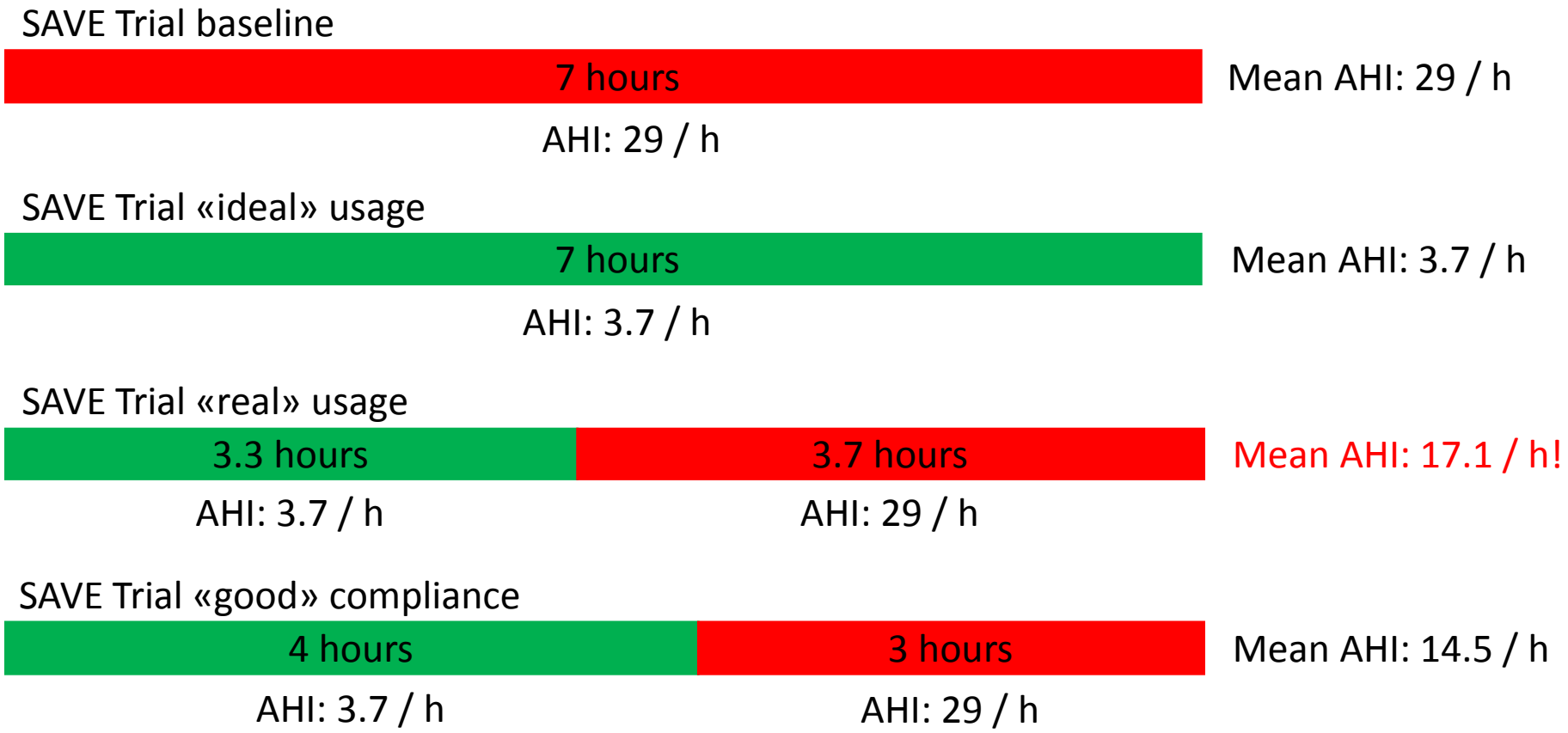
McEvoy RD et al. N Engl J Med. 2016

Warum zeigte sich in den RCTs kein Vorteil für CPAP?

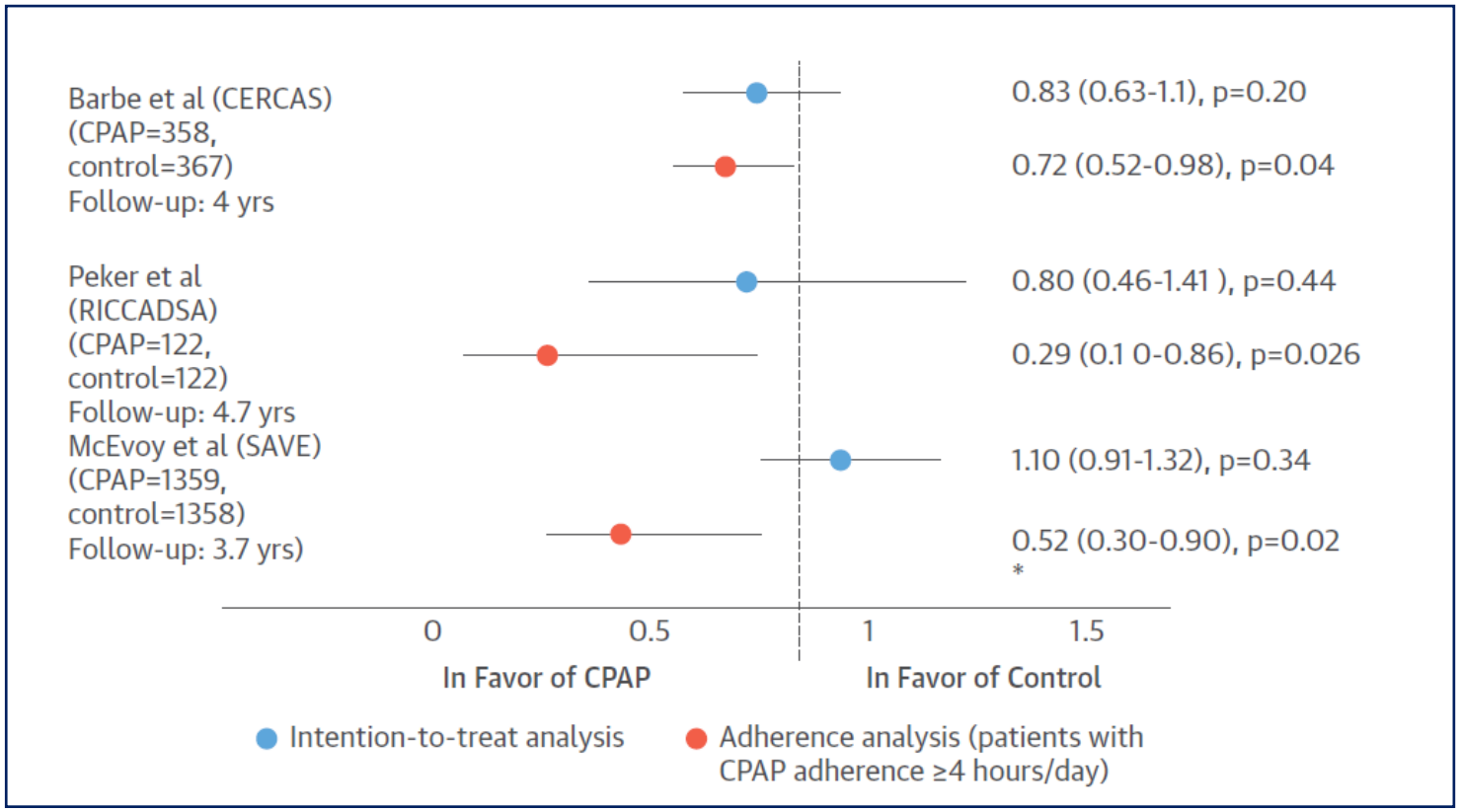
Einfluss der CPAP Adherence

SAVE-Trial

- Baseline AHI: 29 /h
- CPAP AHI: 3.7/h (mean usage per night: 3.3 h)
- Assumed "normal" sleep duration in adults: 6 – 8 hours per night (mean 7 h)



RCTs Summary of CPAP-Effect vs. Control



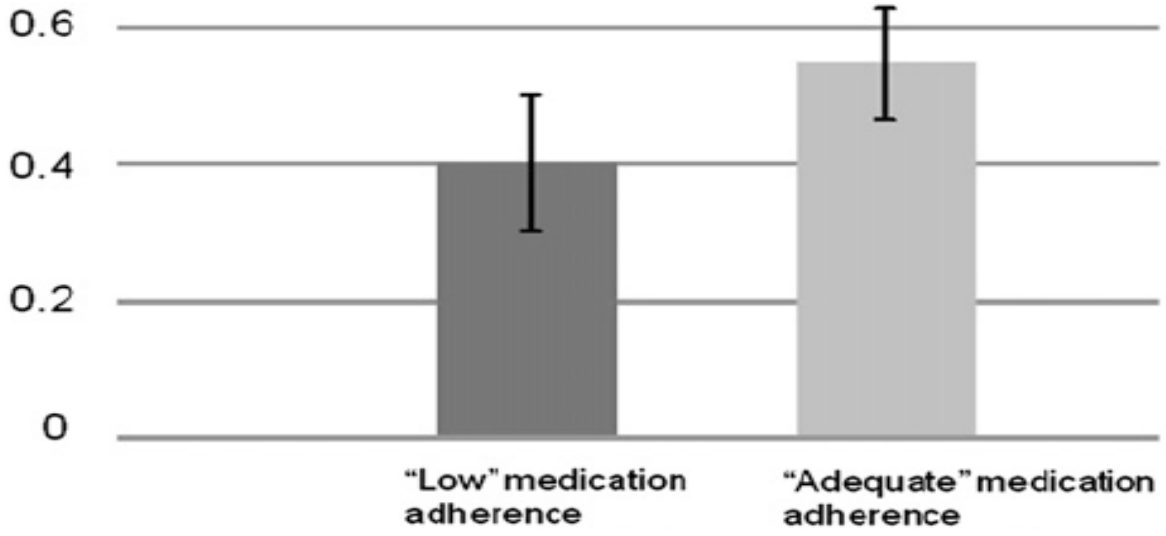
Javaheri et al. *JACC* 2017;69:841-58

Warum zeigte sich in den RCTs kein Vorteil für CPAP?

Einfluss der CPAP Adherence

“Healthy User Effect” und CPAP

Die Wahrscheinlichkeit einer guten CPAP-Nutzung (≥4 h/Nacht) lässt sich anhand der Therapietreue bei Lipidsenkern vorhersagen…



Platt, Chest 2010;137:102

Warum zeigte sich in den RCTs kein Vorteil für CPAP?

- Vielleicht reduziert CPAP das CV-Risiko gar nicht?
- Inadequate CPAP Nutzung?
- Sekundärprophylaxe ist möglicherweise der falsche Ansatz?
- Behandlung von “Mediatoren” des OSA-Effekts (Hypertonus, Diabetes, etc.) überdeckt das OSA-assoziierte Risiko?

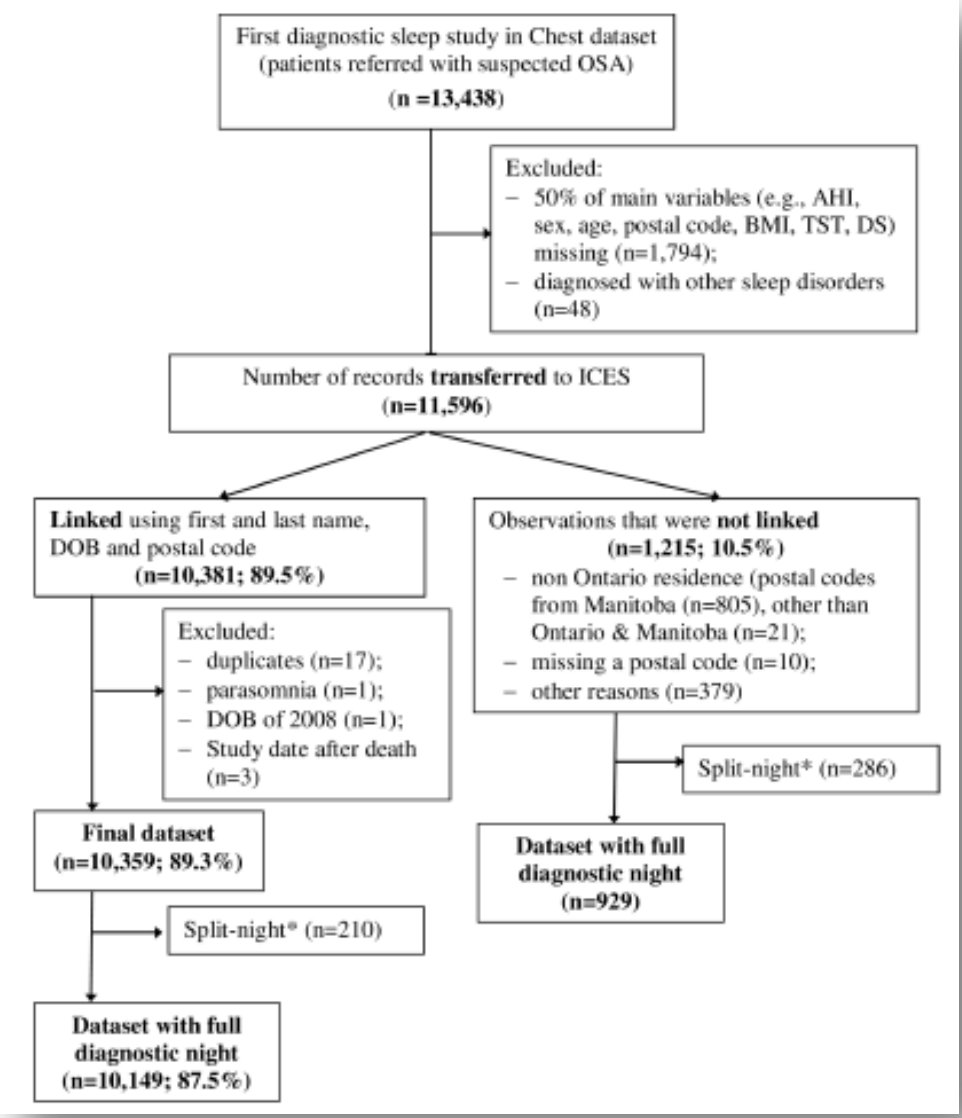
Medications — no./total no. (%)		
Antihypertensive agent	1049/1346 (77.9)	1040/1341 (77.6)
Statin or other lipid-lowering agent	762/1346 (56.6)	800/1341 (59.7)
Antidiabetic oral medication	291/1346 (21.6)	291/1341 (21.7)
Insulin	80/1346 (5.9)	83/1341 (6.2)
Aspirin or other antithrombotic agent	1009/1346 (75.0)	1009/1341 (75.2)

McEvoy RD et al. N Engl J Med. 2016

Warum zeigte sich in den RCTs kein Vorteil für CPAP?

- Vielleicht reduziert CPAP das CV-Risiko gar nicht?
- Inadequate CPAP Nutzung?
- Sekundärprophylaxe ist möglicherweise der falsche Ansatz?
- Behandlung von “Mediatoren” des OSA-Effekts (Hypertonus, Diabetes, etc.) überdeckt das OSA-assoziierte Risiko?
- AHI ist nicht der “richtige” Parameter, um das OSA-assoziierte CV-Risiko beurteilen zu können?

Ist der AHI der richtige Parameter zur Beurteilung der OSA-Schwere und des CV-Risikos?



Cohort study based on clinical database and health administrative data from Ontario (Canada)

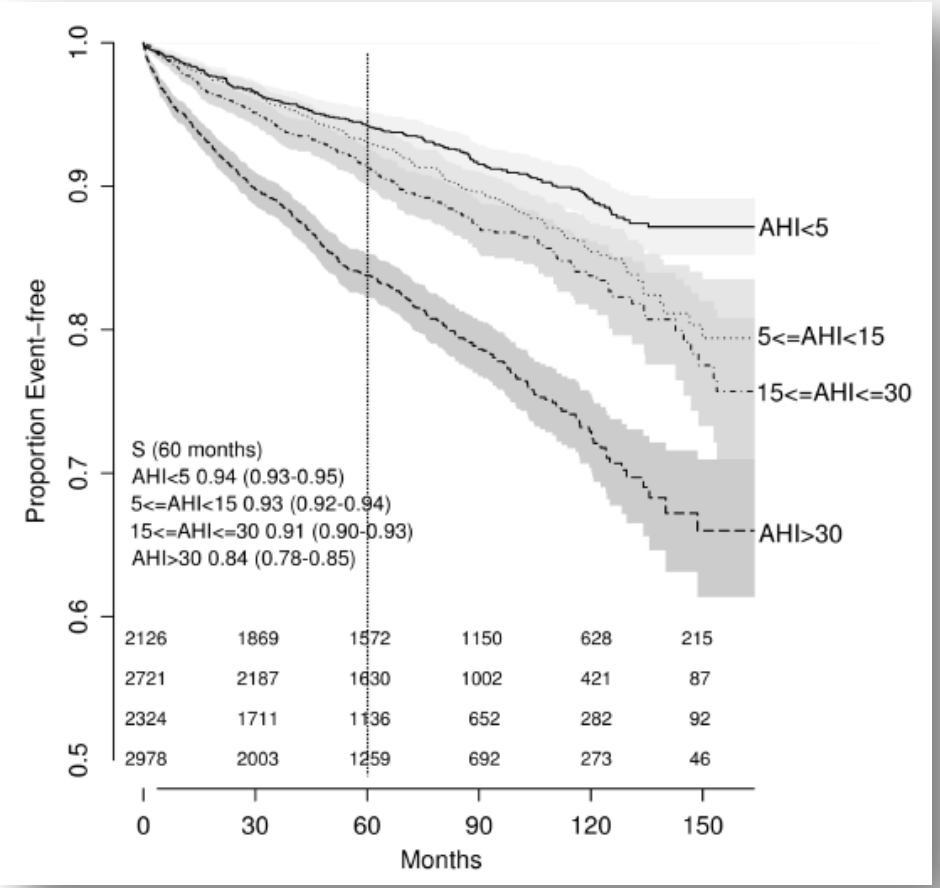
All adults referred for suspected OSA between 1994 and 2010 were followed until May 2011

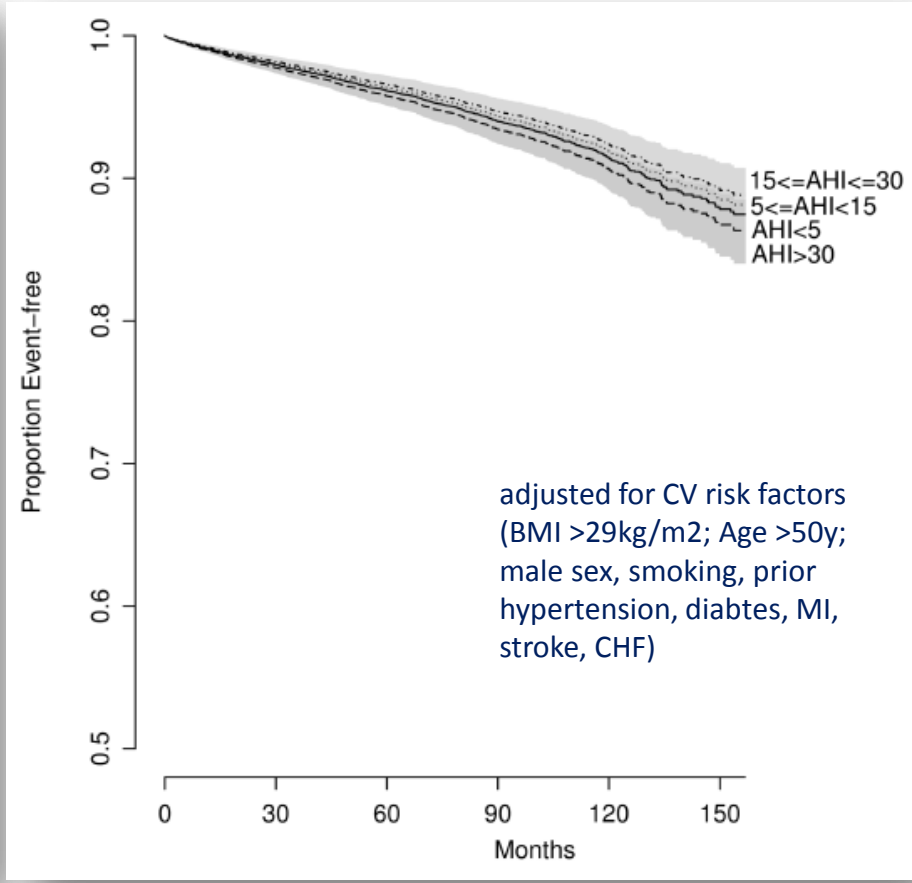
Endpoint:
Occurrence of a composite outcome

- myocardial infarction,
- Stroke
- CHF
- revascularization procedures
- death from any cause

Kendzerska et al. PLOS medicine 2014

Ist der AHI der richtige Parameter zur Beurteilung der OSA-Schwere und des CV-Risikos?

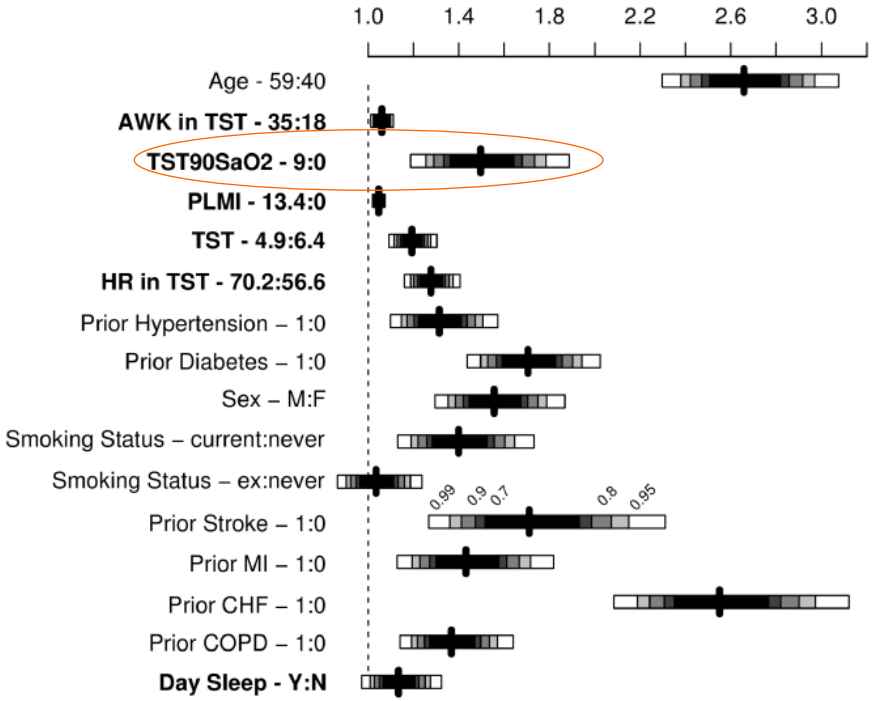




Kendzerska et al. PLOS medicine 2014

Ist der AHI der richtige Parameter zur Beurteilung der OSA-Schwere und des CV-Risikos?

In a fully adjusted model, other than AHI OSA-related variables were significant independent predictors: time spent with oxygen saturation 90%, sleep time, awakenings, periodic leg movements, heart rate and daytime sleepiness



Schlussfolgerung:
Andere OSA-assoziierte Faktoren (nicht AHI) waren wichtige Prädiktoren für das CV-Risiko bei OSA

Kendzerska et al. PLOS medicine 2014

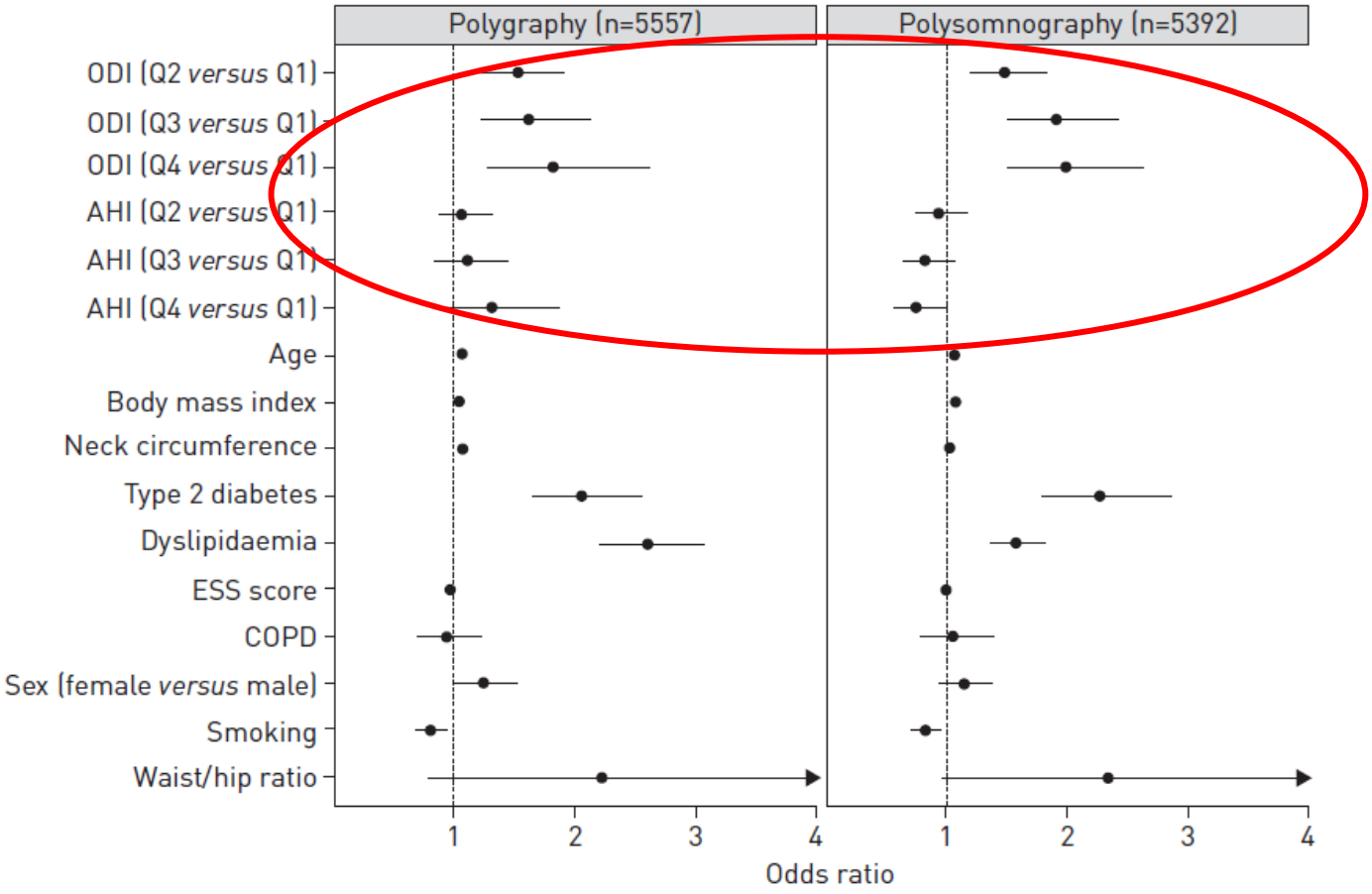
Nocturnal intermittent hypoxia predicts prevalent hypertension in the European Sleep Apnoea Database cohort study

Ruzena Tkacova[1,2], Walter T. McNicholas[3], Martin Javorsky[1,2], Ingo Fietze[4], Pawel Sliwinski[5], Gianfranco Parati[6,7], Ludger Grote[8] and Jan Hedner[8], on behalf of the European Sleep Apnoea Database study collaborators[9]

N=11 911
(2007 – 2013)

AHI Q2 6.0 – 17.4

ODI Q2 3.6 – 11.9



Eur Respir J. 2014

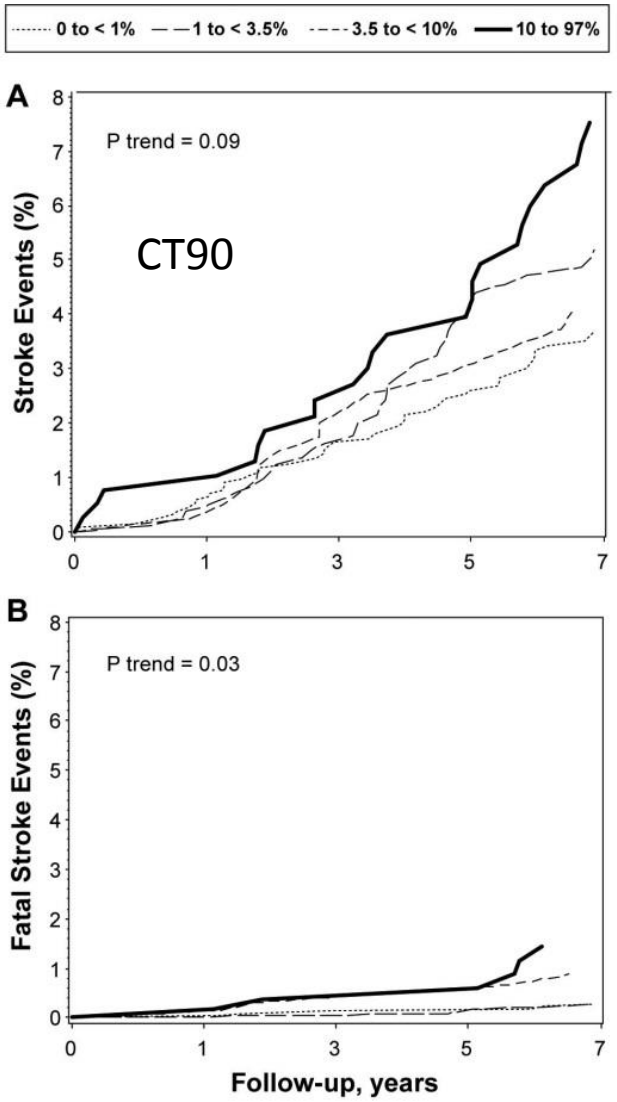
Sleep Disordered Breathing and Risk of Stroke in Older Community-Dwelling Men.

Stone, Sleep 2016.

2,872 elderly men having PSG.
156 (5.4%) had stroke during average 7.3 yrs follow-up.

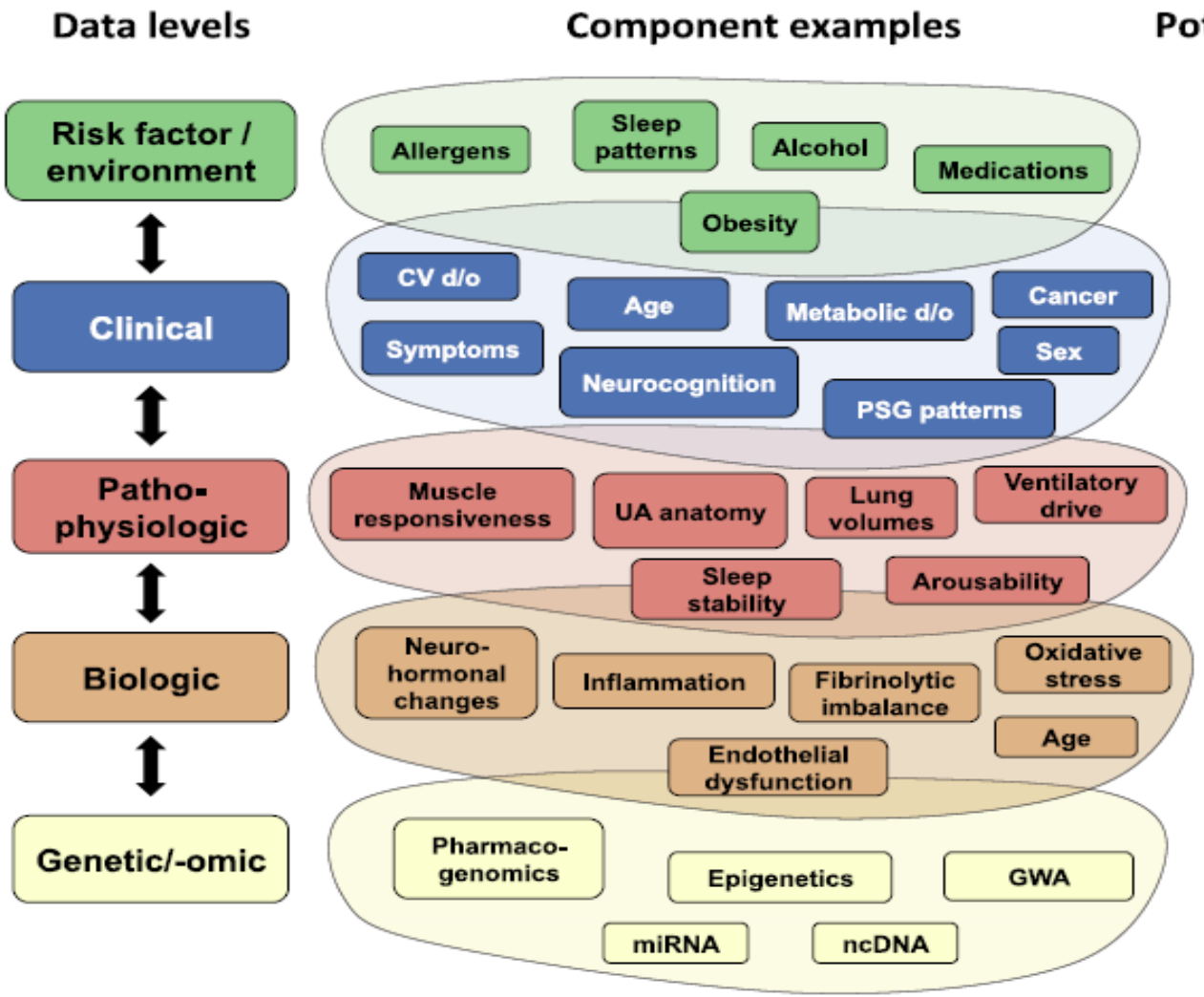
Severe nocturnal hypoxemia (CT90 >10%) had a 1.8-fold increased risk of incident stroke (P = 0.02).

AHI not associated with incident stroke.



	Follow-up, years				
No. at risk:	0	1	3	5	7
0 to < 1%	1,400	1,383	1,299	1,194	1,080
1 to < 3.5%	753	744	703	634	582
3.5 to < 10%	365	358	336	307	271
10 to 97%	354	346	317	282	239

Data levels in OSA phenotyping and their potential relevance



Potential clinical relevance
(selected examples)

Lifestyle
- Modifiable factors (weight loss)

Clinical phenotypes
- Integrated care
- Risk stratification (EDS, elderly)
- Comprehensive guidelines

Intermediate phenotypes
- Therapeutic targets (oxygen, sedatives)
- Diagnostic (PALM)
- Therapy response (CCC)

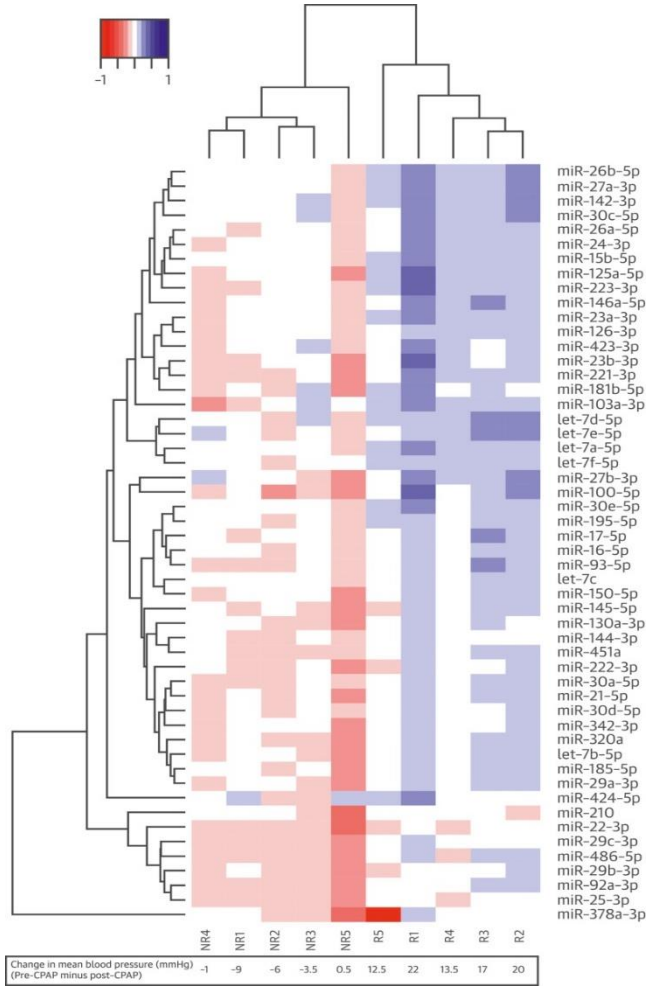
Biomarkers
- Diagnostic (IL-6, IL-10)
- Therapeutic targets
- Sequelae predisposition

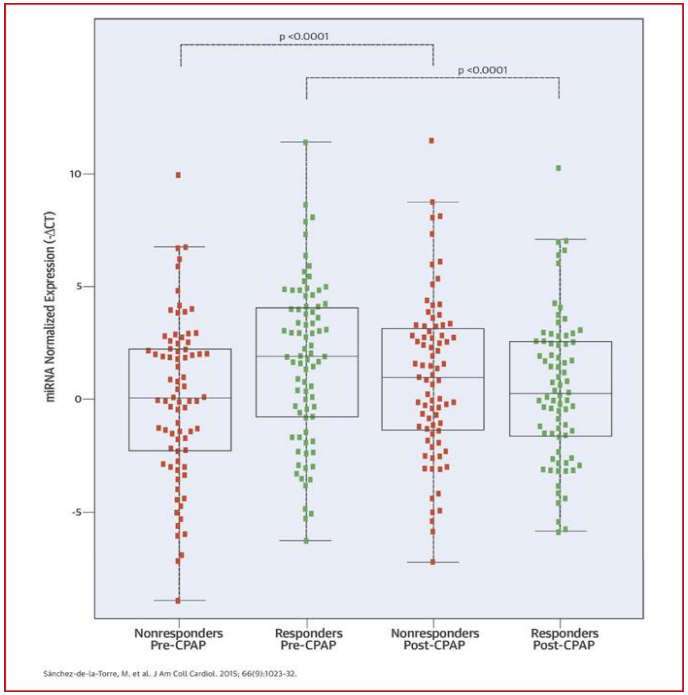
Genetic risk assessment
- OSA risk
- Sequelae predisposition
- Response to therapy (miRNAs & resistant HTN)

Zinchuk et al. Sleep Med Rev 2016

Precision Medicine:
Response to CPAP in Resistent Hypertension and OSA

Occurance of CVD-linked miRNA in relation to blood pressure change after CPAP
Prediction of the BP response to CPAP in patients with RH and OSA (proposed HIPARCO score)





Sanchez-de-la-Torre et al. The Spanish Sleep Network.
J Am Coll Cardiol. 2015, 66 (9):1023

Zusammenfassung

- Die Prävalenz der OSA hat in den vergangenen Jahren deutlich zugenommen
- OSA ist bei Patienten mit CV-Erkrankung überdurchschnittlich oft zu finden
- Gute Evidenz für die zugrunde liegenden Pathomechanismen
- Observationsstudien zeigen klar ein erhöhtes, schweregradabhängiges CV-Risiko bei OSA und einen deutlichen therapeutischen Effekt von CPAP (Senkung des CV-Risiko)
- Die jüngsten RCTs lassen allerdings an der Effektivität einer CPAP-Behandlung in der sekundäre Prophylaxe von CV-Erkrankungen zweifeln

  Aber: methodologische Schwächen geben Anlass zu Bedenken
  bzgl. Aussagekraft (z.B. schlechte Compliance, nur non-sleepy, etc.)
- Möglicherweise ist die aktuelle Definition von OSA (z.B. Schweregrad nach AHI, etc.) unzureichend zur Beurteilung der Schwere der OSA und des CV-Risikos
- OSA Phänotypisierung könnte helfen Risiko-Patienten besser zu erkennen.

„Laugh and the world laughs with you, snore and you sleep alone.“

Anthony Burgess (1917 - 1993), British Writer

sebastian.ott@insel.ch

Back-up

APPENDIX 1. AN OVERVIEW OF THE SCORING CRITERIA CHANGES WITH REGARDS TO APNEAS AND HYPOPNEAS IN ADULTS IN THE AASM MANUAL

	Chicago criteria (1999)	*AASM version 1.0 (2007)*	*AASM version 2.0 (2012)*	*AASM version 2.03 (2014)*
Apnea				
Recommended		≥90% signal drop for ≥10 s and ≥90% of the event duration meets amplitude reduction criteria	≥90% signal drop for ≥10 s	Same as in version 2.0
Hypopnea				
Recommended	≥50% drop or <50% with ≥3% desat or arousal	≥30% signal drop for ≥10 s and ≥4% oxygen desaturation and ≥90% of the event duration meets amplitude reduction criteria	≥30% signal drop for ≥10 s and ≥3% oxygen desaturation or an associated arousal	Same as in version 2.0
Alternative		≥50% signal drop for ≥10 s and ≥3% oxygen desaturation or arousal and ≥90% of the event duration meets amplitude reduction criteria	NA	NA
Acceptable*		NA	NA	≥30% signal drop for ≥10 s with ≥4% oxygen desaturation

AASM, American Academy of Sleep Medicine; NA, not available.
*The acceptable hypopnea scoring method was added to version 2.01 published in July 2013.