

Uykuda Solunum Bozuklukları

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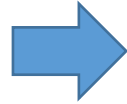
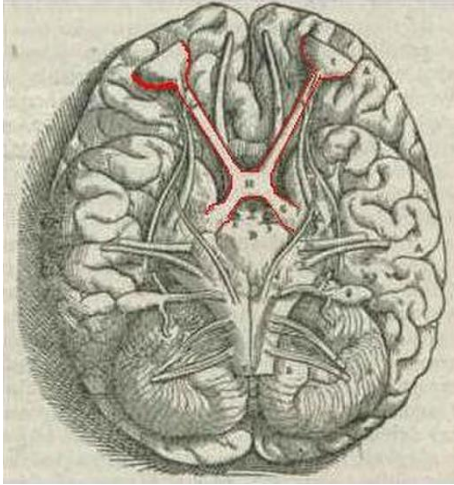
UYKU

- Tüm canlılar uyur
- Memeliler için uyku; Çevre ile iletişimin *geçici, kısmi, periyodik* olarak kesilmesi

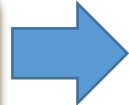


UYKU

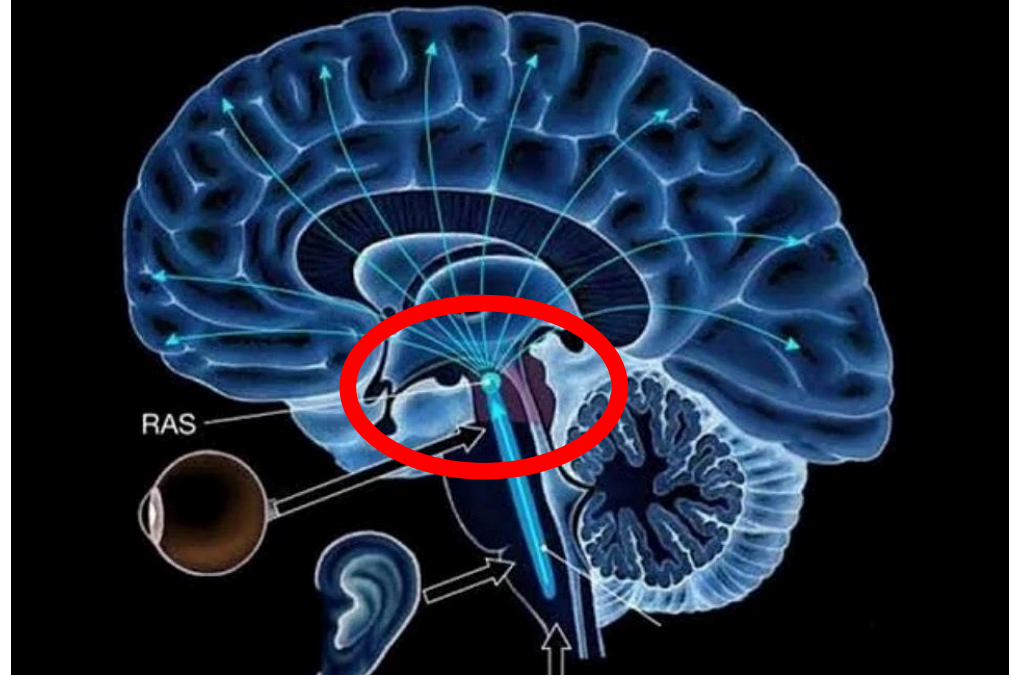
Zamanlama



Sirkadien ritim



Adenozin
metabolizması



UYKU

Değişimler

- Sys vazodilatasyon
- Dipping
- Hormonal regülasyon(Gherilin, leptin, vaspin, lipokin, glukagon, kortizol,GH)
- Duygusal unutkanlık
- Uzun hafıza

Parasempatik hakimiyet/antiinsülin aktivite/Sex hormon depresyonu

Evre 1

Evre 2

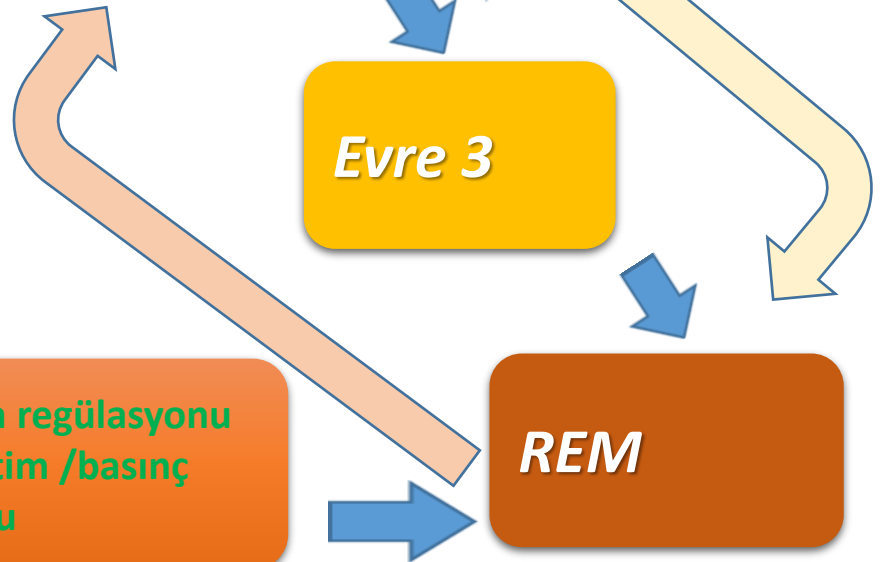
Evre 3

REM

EEG dalga değişimi

Talamus üzerinden motor sinir arkının bloklanması

- Uzun hafıza regülasyonu
- Kardiyak ritim /basınç regülasyonu



UYKU

Solunum ritmi ve hızı bozulur

ÜSY'da direnç artar

Kan basıncı değişiklikleri

Serebral kan akımı ve metabolizma

Santral solunum yanıtı azalır

Uykuyla İlişkili Solunum Hastalıkları

Uluslararası Uyku Bozuklukları Sınıflaması (ICSD- 3)

1.İnsomniler

2.Uykuyla İlişkili Solunum

3. Hipersomnia
hastalıklar

4. Sirkadiyen ritm

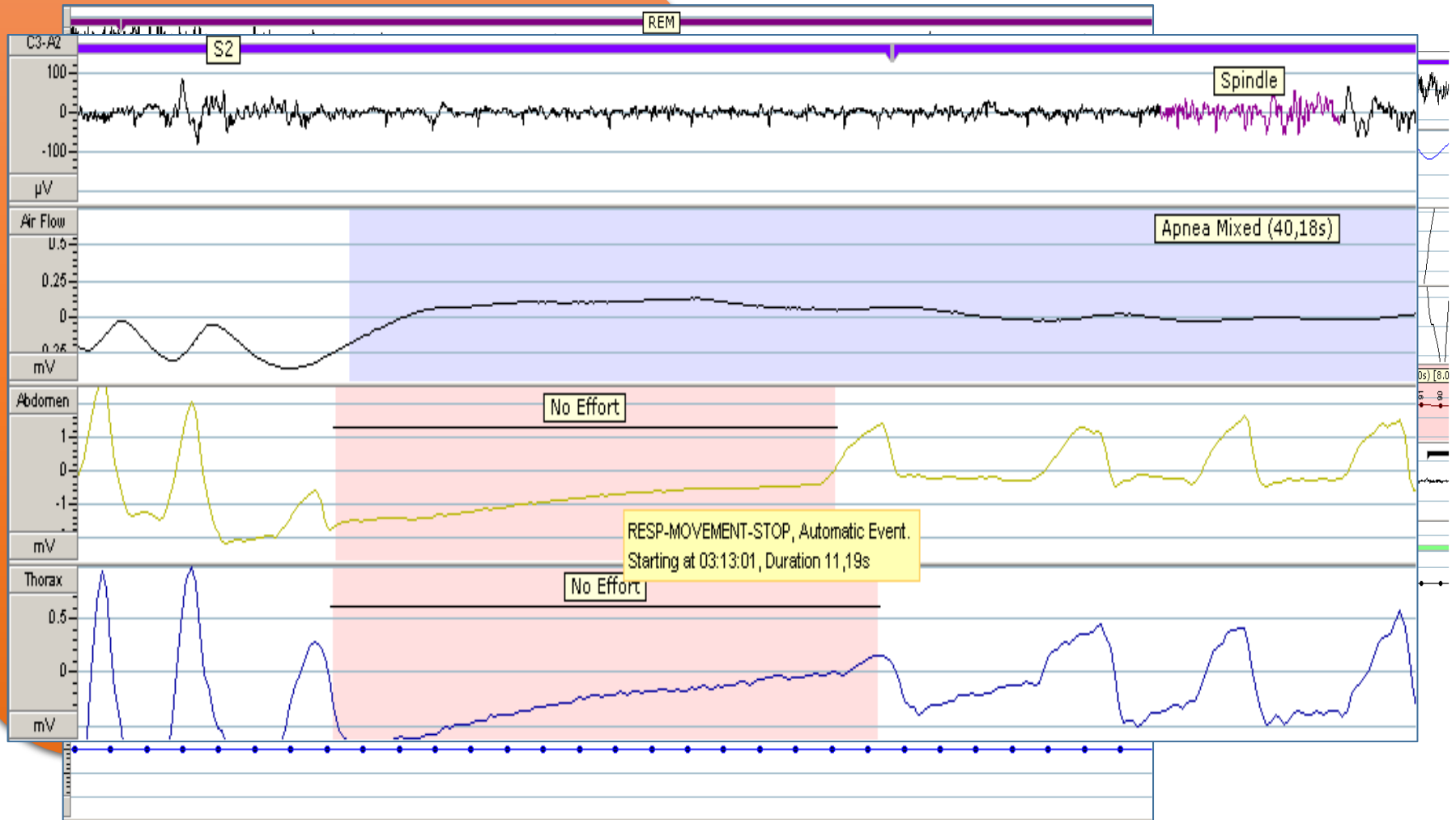
5.Parasomniler

6.Uyku ile ilişkili ha

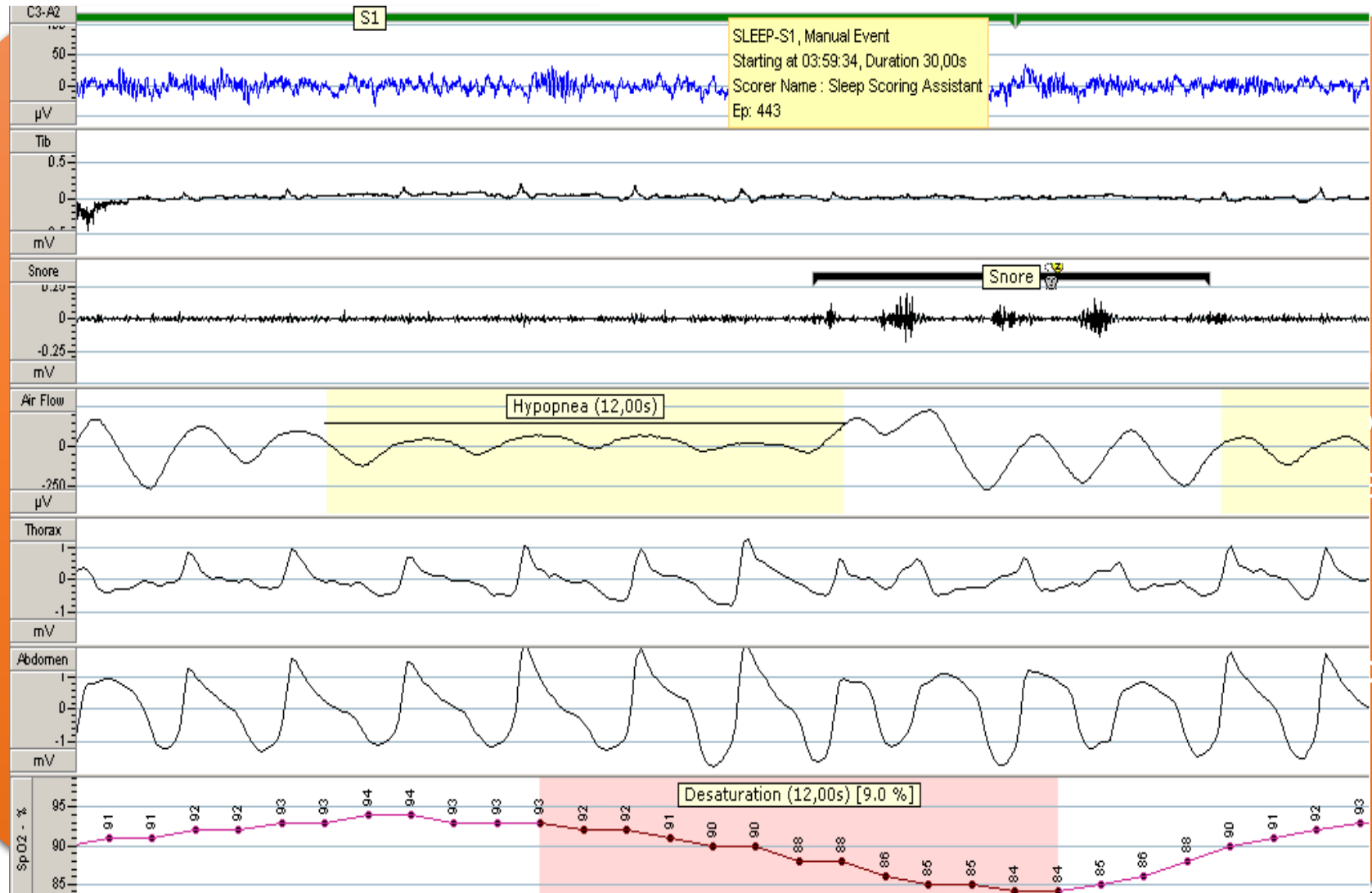
7. Diğer uyku hastalıkları

- ✓ Obstruktif uyku apne sendromu
- ✓ Santral uyku apne sendromları
- ✓ Uyku ilişkili hipoventilasyon hastalıkları
- ✓ Uyku ilişkili hipoksemi hastalığı
- ✓ İzole semptomlar ve normal varyasyonlar
 - ✓ Horlama
 - ✓ Katatreni

Terminoloji

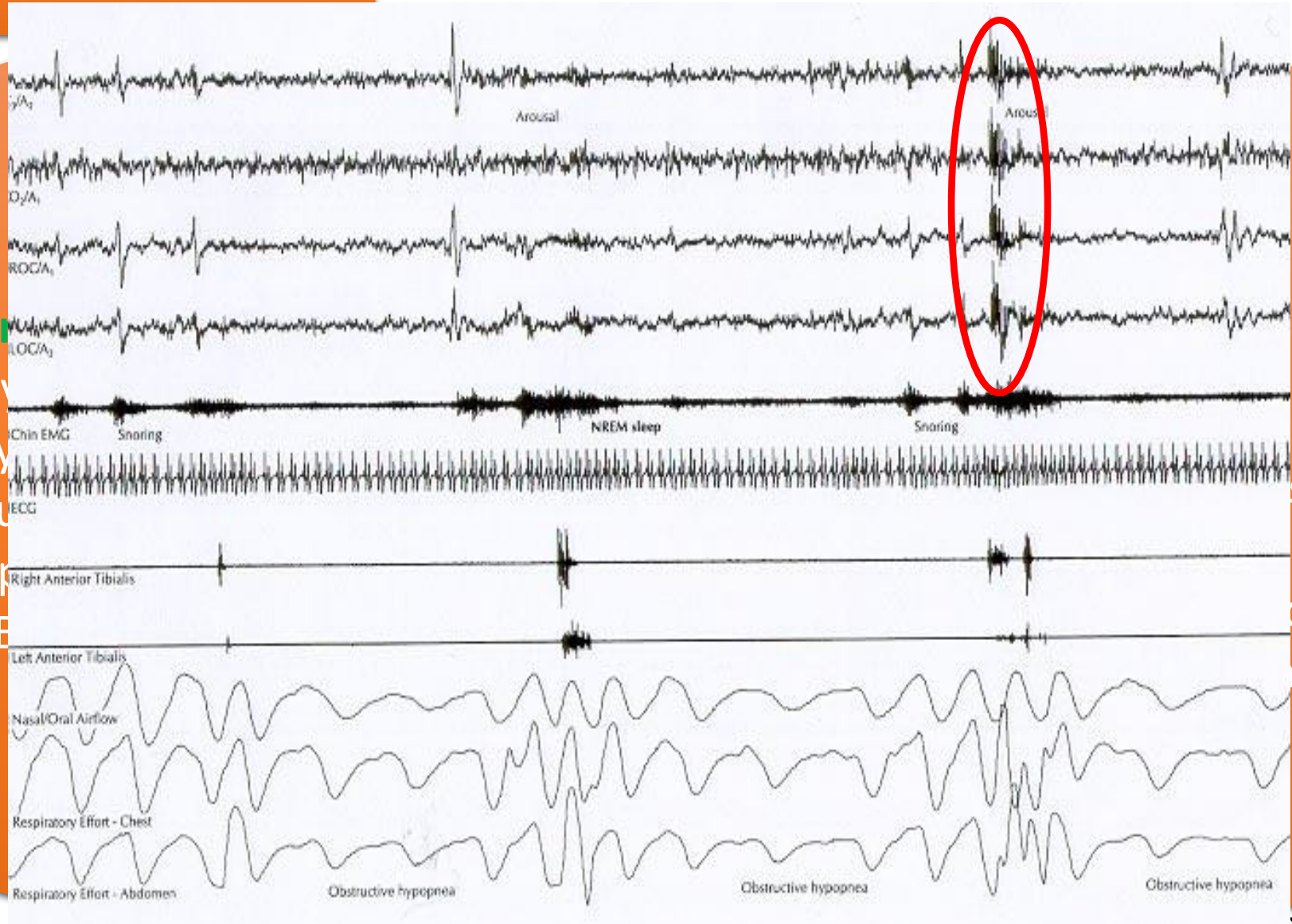


Terminoloji



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Terminoloji



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Terminoloji

Apne İndeksi: Uykuda saat başına düşen apne sayısı

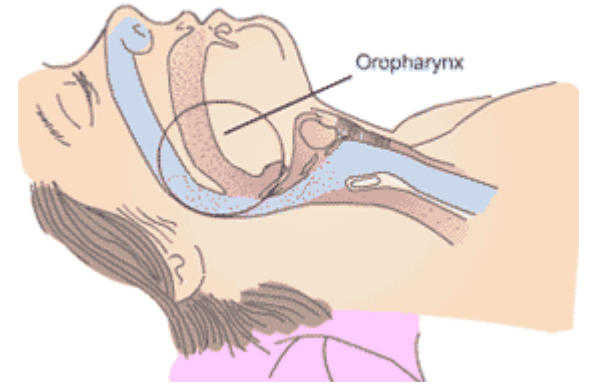
AHi: Uykuda saatteki Apne+ Hipopne sayısı

RDI: Uykuda saatteki Apne+ Hipopne sayısı+ RERA

OSA

OSA

Uyku sırasında tekrarlayan üst solunum yolu (ÜSY) obstrüksiyonu episodları ve sıklıkla kan oksijen saturasyonunda azalma ile karakterize bir sendromdur



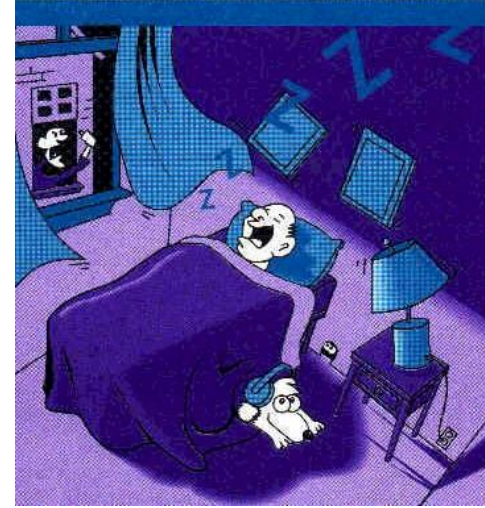
EPİDEMİYOLOJİ

- Obstrüktif uyku apne sendromu % 1-5 oranında görülür
- OSAS' ın, orta yaş erkeklerde (40-60 y) prevalansı % 4-8 dir



Risk faktörleri

- Yaş
- Cinsiyet
- Obesite
- Horlama
- İlaçlar
- Genetik



Obesity

OSA Semptom ve Bulguları

Gece semptomları

- Tanıklı apne
- Horlama
- Dinlendirmeyen uyku
- Gece terlemesi
- Noktüri, enürezi
- İnsomniya
- Nondipper hipertansiyon

Gündüz Semptomları

- Yorgunluk
- Baş ağrısı
- Ağız kuruluđu
- Konsantrasyon bozukluđu
- Hafıza deđişiklikleri
- Duygu durum bozukluđu
- Hormonal anomaliler

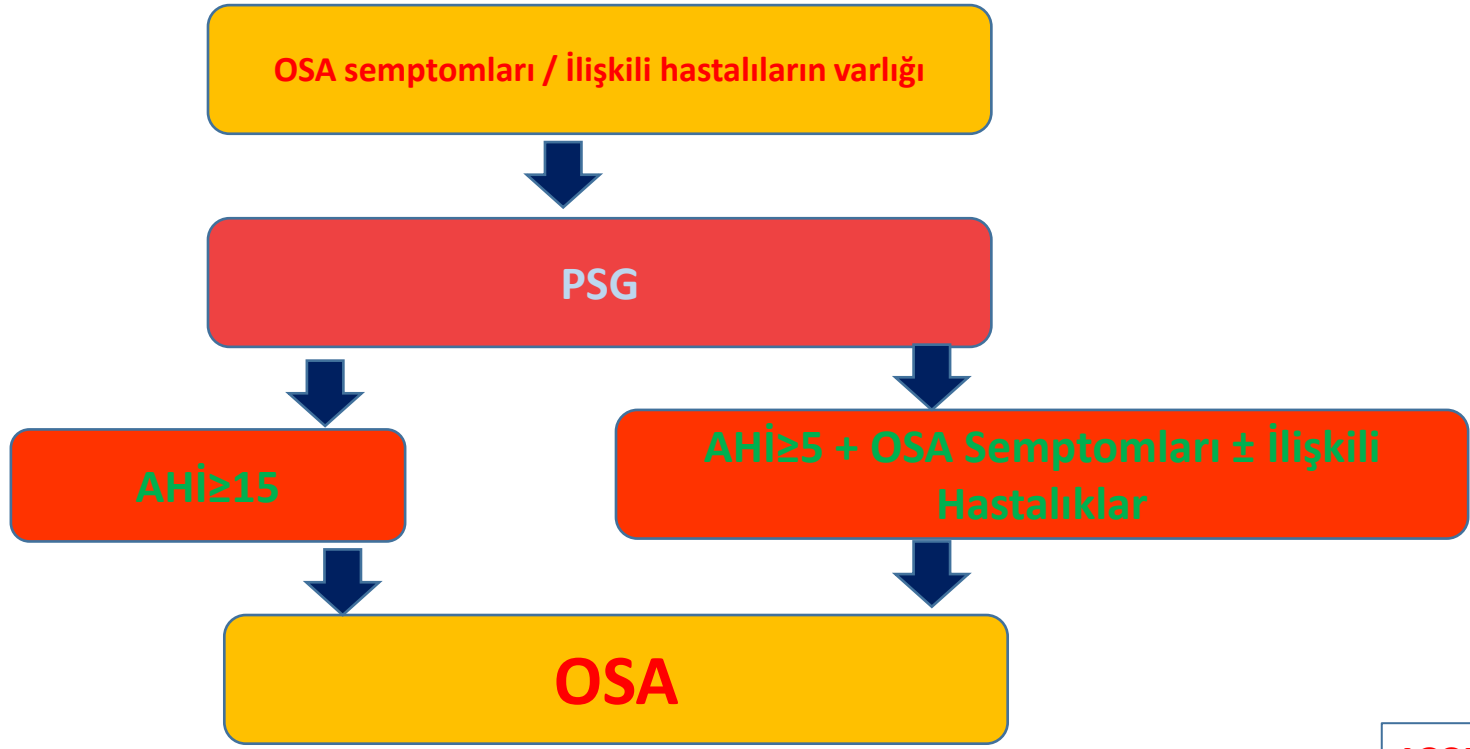
Patofizyoloji

OSA

- ✓ Aralıklı hipoksemi
- ✓ Toraks içi negatif basınç artışı
- ✓ Sempatik aktivasyon
- ✓ VK ve periferik direnç artışı
- ✓ Oksidatif stres
- ✓ Endotel disfonksiyonu

- ✓ KAH
- ✓ Ventrikül hipertrofisi
- ✓ KKY
- ✓ HT
- ✓ Aritmiler
- ✓ İnme
- ✓ Pulmoner HT

Tanı



$AHI < 5 \longrightarrow$ NORMAL

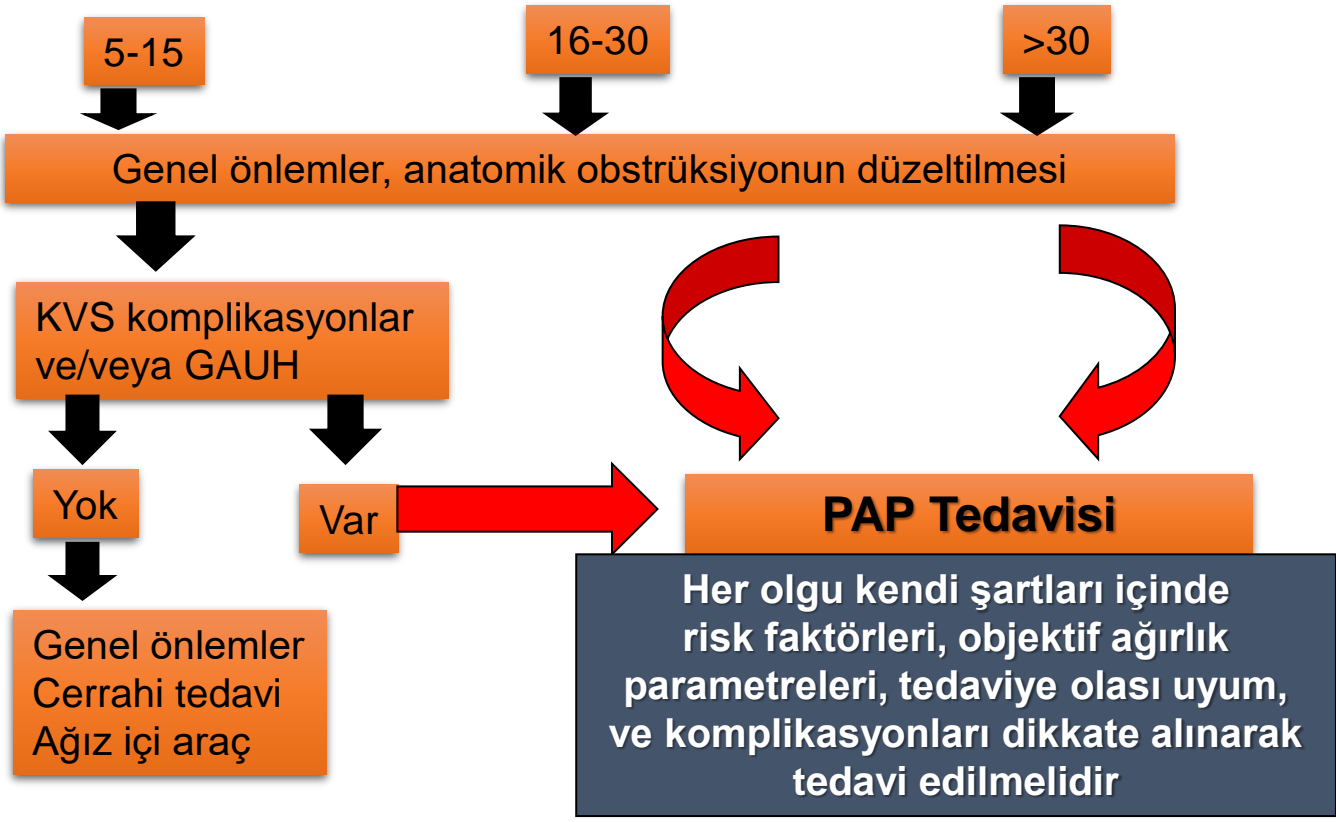
$AHI \ 5 - 15 \longrightarrow$ HAFİF

$AHI \ 16 - 30 \longrightarrow$ ORTA

$AHI \ > 30 \longrightarrow$ AĞIR

Tedavi

AHi



PAP Tedavisi

- CPAP (%78,6 Başarılı)
- APAP
- BPAP
 - Dakika hacmini artırır
hipokapni yapar
 - CompSAS'ta kullanılmaz
- BPAP –ST
- ASV
- AMV

OSA Sonuçları

1. Kardiyovasküler
2. Endokrinolojik
3. Nefrolojik
4. Gastrointestinal
5. Hematolojik
6. Psikiyatrik
7. Göz
8. Sosyo-ekonomik
9. Ani ölüm

Kardiyovasküler Sonuçları

- HİPERTANSİYON (HT)
- İNME (SVO)
- ARİTİMİ
- SOL KALP YETMEZLİĞİ (KKY)
- KORONER ARTER HASTALIĞI (KAH)
- SAĞ KALP YETMEZLİĞİ, PULMONER HİPERTANSİYON (PHT)

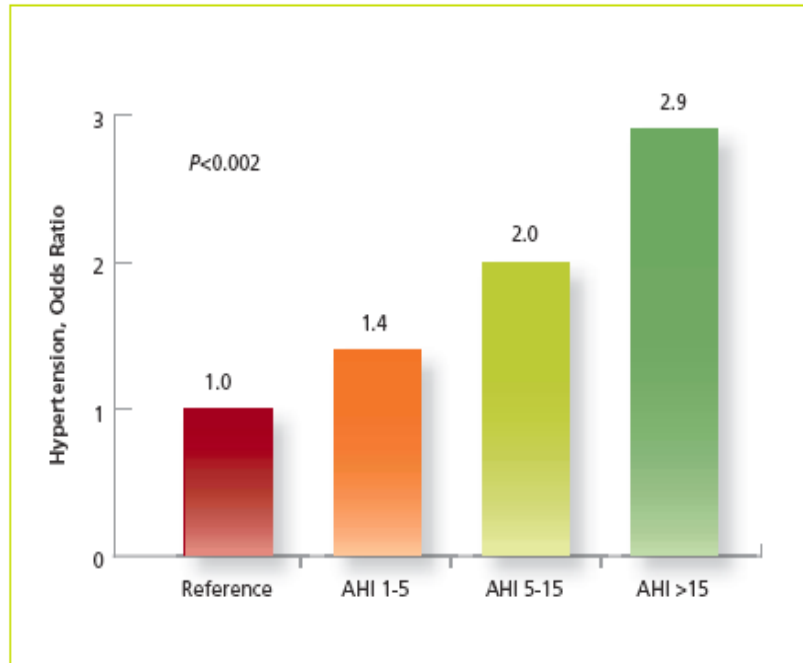
Kardiyovasküler Sonuçları

- HİPERTANSİYON (HT)
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- SOL KALP YETMEZLİĞİ (KKY)
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- SAĞ KALP YETMEZLİĞİ, PULMONER HİPERTANSİYON (PHT)

OUAS ve HT

- Hafif derecede
- OUAS hastalığı
- HT tedaviye

Wisconsin
Peppard
Logan



HT gelişmesi

Figure 5. Odds ratio for hypertension as a function of apnea hypopnea index.

Note the significant dose-dependent relationship ($P=0.002$ for trend).

Adapted from Peppard PE, et al. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med.* 2000;342:1378-1384.

OUAS ve Aritmi

En sık sinüs bradikardisi (%7),
sinüs arrest(%8), A-V bloğu
(%11)

Ventriküler prematür atımlar,
ventriküler taşikardi (%1-3),
sinüzal taşikardi (%1-3)

Gula LJ. Heart 2004

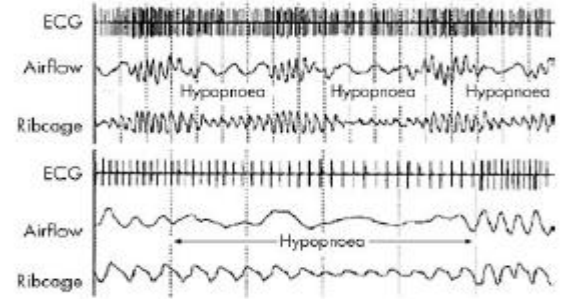


Figure 3 Illustration of cyclic variation of heart rate in relation to arterial oxygen saturation in obstructive sleep apnoea.

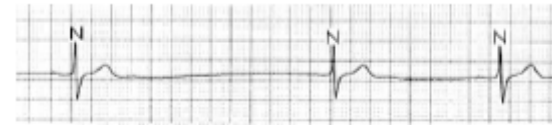


Figure 4 Sinus pause during episode of apnoea in an OSA patient.

OUAS ve SVO

- İnme hastalarının %45-90'inde OUAS

Mohsenin V. Arch Phys Med Rehabil 1995; 76: 71–76.

Wessendorf TE. J Neurol 2000; 247: 41–47.

Bassetti C. Sleep 1999; 22; 217–223.

- Horlayanlarda SVO 1.8 kat fazla

Hu FB. J Am Coll Cardiol 2000;35:308-313

- 167 inme vakasının 1/3'ünde OSA

Palomaki H. Stroke 1989;10:1311-1315

OUAS ve KKY

- KKY olanların %50'sinde OUAS

Alchanatis M. Eur Respir J 2002; 20: 1239–124

- OUAS'da 2.38 kat fazla KKY riski

Nieto FJ. Sleep Heart Health Study. JAMA 2000; 283: 1829–1836.

OUAS ve KAH

- Anjiyografi ile KAH saptanan kadın hastaların %30'unda, erkek hastaların %37'inde OSAS

Moore T. Chest. 1996;109:659-663.

Moore T. Am J Med. 1996;101:251-256.

- KAH olan hastaların %50'sinde OUAS

Andreas S. Cor Art Dis. 1996;7:541-545

OUAS ve Endokrin Komplikasyonlar

- **İnsulin Rezistansı**
- **Growth Hormon Salınımında Azalma (Obesite)**
- **Lipido Azalması, İmpotans**
- **Metabolik Sendrom**

OUAS ve DM/ İnsülin Rezistansı

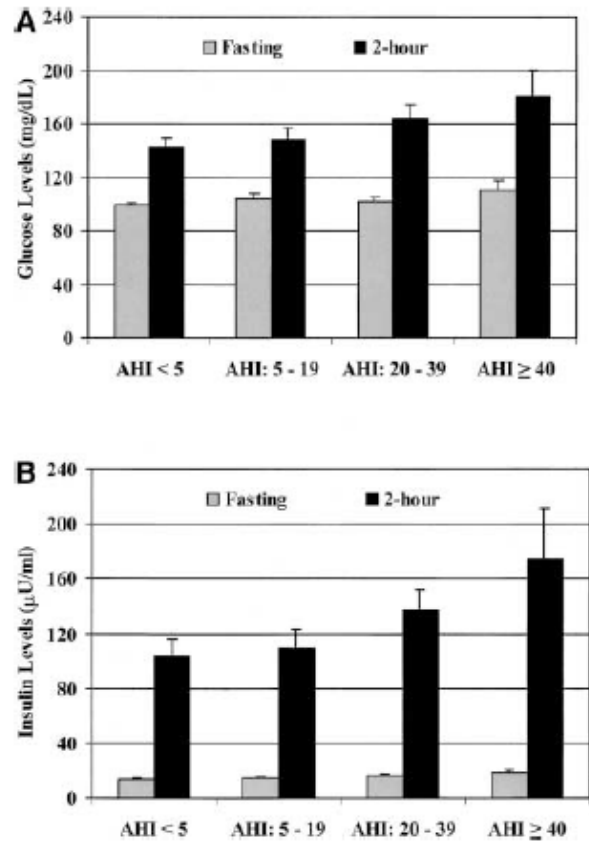


Figure 1. Fasting and 2-h insulin and glucose levels by apnea-hypopnea index (AHI) category (values represent means \pm SE). Significant trends ($p < 0.05$) were noted across AHI categories in the fasting insulin level and 2-h glucose and insulin levels.

Naresh M. Punjabi. Sleep Heart Health Study. Am J Epidemiol. 2004; 160:521-530.

OUAS ve Metabolik Sendrom

Table 3 Death and cardiovascular events in obstructive sleep apnea (OSA) patients according to the presence of metabolic syndrome

Event	OSA with metabolic syndrome (<i>n</i> = 47)	OSA without metabolic syndrome (<i>n</i> = 42)	<i>P</i>
Death	0 (0)	1 (2)	0.901
Acute coronary syndrome	1 (2)	0 (0)	0.907
Newly-diagnosed chronic angina	0 (0)	4 (10)	0.085
Coronary revascularization	0 (0)	1 (2)	0.901
Cerebrovascular event	1 (2)	1 (2)	0.448
Peripheral vascular event	0	0	–
Venous thromboembolism	1 (2)	3 (7)	0.527
Any event	3 (6)	10 (24)	< 0.05

Data are *n* (%).

Vaspin and lipocalin-2 levels in severe obstructive sleep apnea

Muharrem Kiskac¹, Mehmet Zorlu¹, Muhammed Emin Akkoyunlu², Elif Kilic³, Cumali Karatoprak¹, Mustafa Cakirca¹, Erdinc Yavuz⁴, Cuneyt Ardic⁴, Ahmet Adil Camli¹, Mehmetali Cikrikcioglu¹, Levent Kart²

¹Internal Medicine Clinic, Bezmialem Vakif University, Faculty of Medicine, 34093 Fatih, Istanbul, Turkey; ²Department Of Pulmonology, ³Department Of Medical Biochemistry, Bezmialem Vakif University, Faculty of Medicine, Fatih 34093, Istanbul, Turkey; ⁴Family Health Care Center, Rize 53100, Turkey

Correspondence to: Muharrem Kiskac, MD. Internal Medicine Clinic, Bezmialem Vakif University, Faculty of Medicine, Fatih 34093, Istanbul, Turkey.
Email: dr_kiskac@mynet.com.

Background: Vaspin and lipocalin-2 are less-known recent members of adipocytokine family. There are ongoing studies investigating the role of vaspin ve lipocalin-2 in metabolic syndrome (MS). Obstructive sleep apnea syndrome (OSAS) is independently associated with an increased prevalence of MS. We aimed to measure the levels of vaspin and lipocalin-2 which are secreted from adipocytes in patients with severe OSAS and examine the relationship between these two adipocytokines and OSAS.

Methods: The study consisted of two groups: severe OSAS patients with an apnea-hypopnea index (AHI) of >30/h (OSAS group, 34 subjects) and age-matched healthy volunteers with a AHI <5/h (control group, 25 subjects) Serum levels of vaspin and lipocalin-2 in these two groups were compared.

Results: Serum levels of vaspin were significantly lower in OSAS group patients with severe OSAS compared with control group; healthy volunteers (OSAS group: 0.69 ± 0.5 vs. control group: 1.24 ± 1.13 ; $P=0.034$). The difference between the two groups in terms of serum levels of lipocalin-2 has not reached statistical significance (OSAS group: 61.6 ± 18.2 vs. control group: 68.5 ± 20.1 ; $P=0.17$).



OUAS ve Growth Hormon Salınımı

Growth hormon salınımının OUAS'lılarda azalması nedeniyle lipoliz e azalır

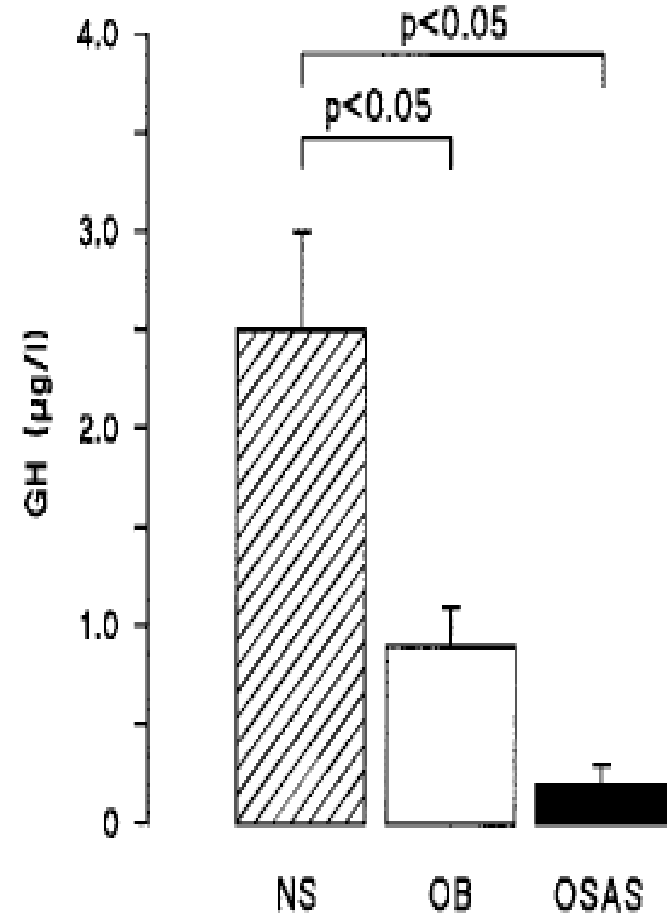
J Clin Endocrinol Metab. 2002

n-CPAP kullanımı ile uyku yapısı düzelir , GH salınımı normale döner ve lipoliz hızlanır

OUAS'lı çocuklarda GH eksikliğine bağlı büyüme ve gelişme geriliği

Tonsillektomi sonrası hızla gelişme o.ç.

Leiberman A. Int J Pediatr Otorhinolaryngol 2006



OUAS ve Empotans

- OUAS'luların % 28-50'sinde empotans
- OUAS'lularda empotans nedenleri : Depresyon, hipotalamik-hipofizer-testiküler fonksiyon bozukluğu
- Empotans ile AHİ korele

Margel D. Urology. 2004 Mar;63(3):545-9.

- Hafif OUAS'luların % 53.8'inde, ağır OUAS'luların ise % 54.5'unda libido azalması

Köktürk ve ark.

- CPAP ile testesteron düzeyi artar

Goncalves MA. Sleep Med. 2005 Jul;6(4):333-9.

OUAS ve Kaza Riski

- Uykulu araba kullanma trafik kaza riskini 8.2 kat artırmakta

Connor J. Accid Anal Prev33;31-41

Respiratory medicine

- (

ORIGINAL RESEARCH ARTICLE

Open Access

- (Investigation of obstructive sleep apnoea syndrome prevalence in long distance drivers from Turkey

ve riskin 6.1

OSAS prevalansı :1,5-2,4

Uzun yol şoförleride %14,9

Muhammed E Akkoyunlu¹
and Meltem Tor²

- (

Abstract

Background: The aim of this study was to assess the prevalence of Obstructive sleep apnoea syndrome (OSAS) in



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Obstructive Sleep Apnea and the Risk of Sudden Cardiac Death: A Longitudinal Study of 10,701 Adults

Apoor S. Gami, M.D., M.Sc., F.A.C.C.^{1,2,5}, Eric J. Olson, M.D.^{3,4,5}, Win K. Shen, M.D., F.A.C.C.^{2,5}, R. Scott Wright, M.D., F.A.C.C.^{2,5}, Karla V. Ballman, Ph.D.⁶, Dave O. Hodge, M.S.⁶, Regina M. Herges, B.S.⁶, Daniel E. Howard, M.D.⁵, and Virend K. Somers, M.D., Ph.D., F.A.C.C.^{2,5}

¹Midwest Heart Specialists – Advocate Medical Group, Elmhurst, IL

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³Sleep Disorders Center, Rochester, MN

⁴Division of Pulmonary and Critical Care Medicine, Rochester, MN

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Abstract

Objective—To identify the risk of sudden cardiac death (SCD) associated with obstructive sleep apnea (OSA).

Background—Risk stratification for SCD, a major cause of mortality, is difficult. OSA is linked to cardiovascular disease and arrhythmias, and has been shown to increase the risk of nocturnal SCD. It is unknown if OSA independently increases the risk of SCD.

Methods—We included 10,701 consecutive adults undergoing their first diagnostic polysomnogram between 7/1987 and 7/2003. During follow-up up to 15 years, we assessed incident resuscitated or fatal SCD in relationship to the presence of OSA, physiological data including the apnea-hypopnea index (AHI) and nocturnal oxygen saturation (O₂sat) parameters, and relevant comorbidities.

Results—During an average follow-up of 5.3 years, 142 patients had resuscitated or fatal SCD (annual rate 0.27%). In multivariate analysis, independent risk factors for SCD were age, hypertension, coronary artery disease, cardiomyopathy or heart failure, ventricular ectopy or nonsustained ventricular tachycardia, and lowest nocturnal O₂sat (per -10%, HR 1.14, P=0.029). SCD was best predicted by age >60 years (HR 5.53), AHI >20 (HR 1.60), mean nocturnal O₂sat <93% (HR 2.93), and lowest nocturnal O₂sat <78% (HR 2.60, all P<0.0001).

Conclusions—In a population of 10,701 adults referred for polysomnography, OSA predicted incident SCD, and the magnitude of risk was predicted by multiple parameters characterizing OSA severity. Nocturnal hypoxemia, an important pathophysiological feature of OSA, strongly

