

“What is the True Silent Killer” Are you at Risk?

Chicago Dental Society Regional Meeting

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Objectives

At the conclusion of this presentation, participants will be able to:

1. Understanding the difference between cardiac arrest and heart attack
2. Diabetes, Heart failure and OSA....What is the link?
3. Effects of oral inflammation on heart health.
4. “Assimilating Cardiovascular Knowledge into Dental Sleep Medicine” and Effectively Communicate a care plan with patients’ providers

-Case Scenario

OSA is the “TRUE SILENT KILLER”



Objectives

At the conclusion of this presentation, participants will be able to:

1. Acquiring critical medical knowledge to improve patient outcomes and identify GAPS and Barriers preventing development/building of a successful practice/referral system
2. Objectively develop/evolve your practice into an outcome/evidence-based healthcare service that will enhance referrals. However a few prerequisites have to be fulfilled which will be concisely outlined
3. Implement Strategies to enhance/transform your practice outcomes which drives REFERRALS.
 - *Understand the core of why **not treating sleep apnea is NOT AN OPTION.**
 - *Information on **KEY MUST KNOW DISEASES** that you need to explicitly understand to build legitimacy
 - *Understand the referral bias to improve referrals: Know your audience and their limitations (MDs, DOs, NPs, PAs)
 - *Implement disease modifying PATHWAYS to achieve desirable outcomes to share with healthcare providers

Heart Disease in the United States

- **Leading cause of death** in the United States.⁵
- **One person dies every 36 seconds**
- **659,000 people in the United States** die from heart disease each year—that's **1 in every 4 deaths**.⁶
- **\$363 billion** each year from 2016 to 2017.² (Health care services, medicines, lost productivity due to death.)

Coronary Artery Disease

- 360,900 deaths in 2019.⁷
- **18.2 million adults** age 20 and older have CAD (about 6.7%).⁸

Myocardial Infarction

- Heart attack occurs every 40 seconds.⁸
- **805,000 people in the United States** have a heart attack.⁸
- **About 1 in 5 heart attacks is silent**³

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7. Centers for Disease Control and Prevention, National Center for Health Statistics. [About Multiple Cause of Death, 1999–2019](#). CDC WONDER Online Database website. Atlanta, GA: Centers for Disease Control and Prevention; 2019. Accessed February 1, 2021.

8. Fryar CD, Chen T-C, Li X. [Prevalence of uncontrolled risk factors for cardiovascular disease: United States, 1999–2010](#) pdf icon[PDF-494K]. NCHS data brief, no. 103. Hyattsville, MD: National Center for Health Statistics; 2012. Accessed May 9, 2019.

LATEST STATS ON SUDDEN CARDIAC ARREST

• ADULTS

- Out of The Hospital Cardiac Arrest (OHCA) 347,322
- Survival with functional recovery: 0.8%-20.1
- Adults treated by EMS: 25% without prior sx
- Majority occur at home or residence 68.5%
- Public setting 21%
- Nursing Home 10.5%
- No witnesses 51%

SCD
6 AM -
Noon



• CHILDREN

- <18 years of age 7,037
- Survival to hospital discharge 10.7%
- Occurs at home 89.5% < 1 year old
- 77% 1-12 years old
- 72.9% 13-18 years old





Congestive Heart Failure (CHF): A Healthcare Epidemic

- **Prevalence: 6.7 million Americans**
 - 1.5 million are under 60 years of age
- **Incidence: 565,000 annually**
 - For those over 65 years: 15 – 20/1,000
- **11.5 million physician visits annually**
- Most common diagnosis in hospital patients ages 65 and older
 - **First-listed diagnosis in 890,000 hospitalizations annually**
 - 20% of patients return to the hospital within 30 days of discharge
 - **50% of patients return to the hospital within 6 months of discharge**
- More than ½ of all CHF patients die within 5 years of diagnosis
 - Approximately 350,000 deaths annually
- **Annual costs of over \$362 billion**

Figure 13.

New York Heart Association Classification of Heart Failure

Class	Patient Symptoms
Mild	<ul style="list-style-type: none"> ■ No limitation of physical activity ■ No undue fatigue, palpitation or dyspnea
Mild	<ul style="list-style-type: none"> ■ Slight limitation of physical activity ■ Comfortable at rest ■ Less than ordinary activity results in fatigue, palpitation, or dyspnea
Moderate	<ul style="list-style-type: none"> ■ Marked limitation of physical activity ■ Comfortable at rest ■ Less than ordinary activity results in fatigue, palpitation, or dyspnea
Severe	<ul style="list-style-type: none"> ■ Unable to carry out any physical activity without discomfort ■ Symptoms of cardiac insufficiency at rest ■ Physical activity causes increased discomfort

Criteria Committee of the New York Heart Association, 1964

Worldwide prevalence of sleep apnea in consecutive patients with heart failure

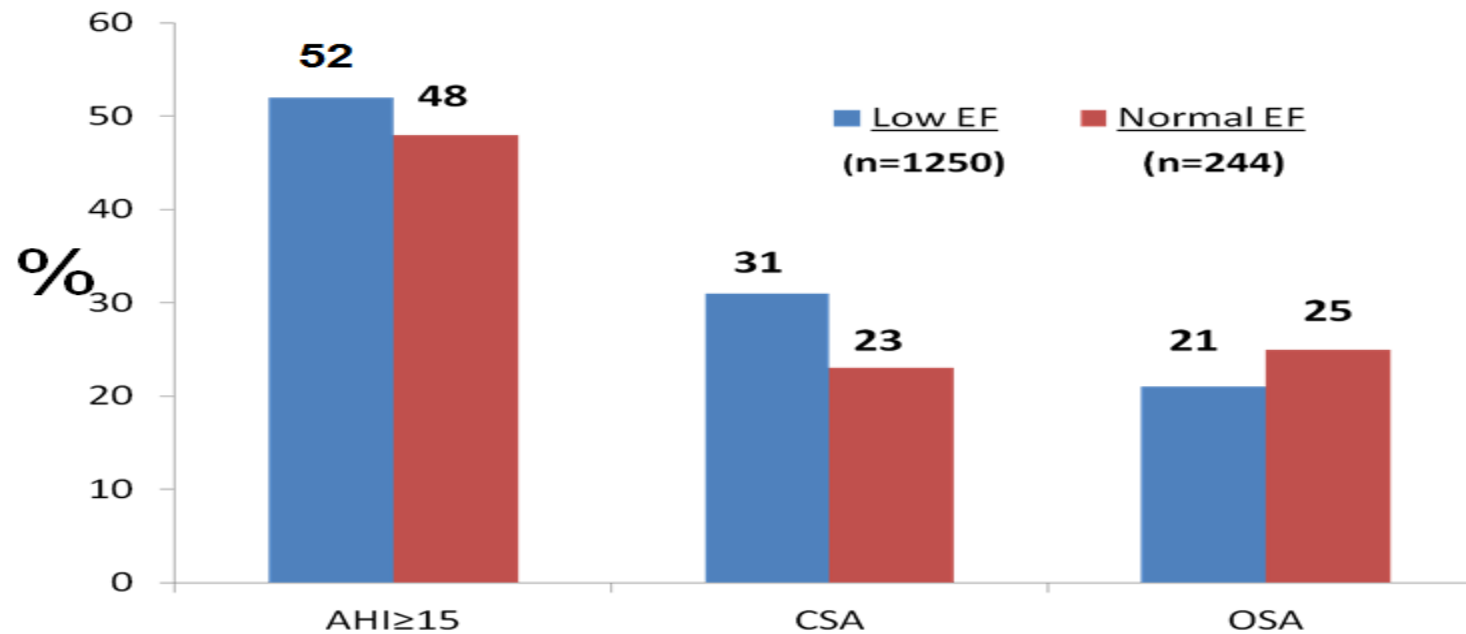


Figure 14. Worldwide Prevalence of Sleep Apnea in consecutive patients with Heart Failure

35. Javaheri S. Sleep-related breathing disorders in heart failure. In: Heart Failure, A Companion to Braunwald's Heart Disease, edited by Douglas L. Mann. WB Saunders, Philadelphia 2010; 471-487.

36. Javaheri S. Cardiovascular Disorders. In: Atlas of Clinical Sleep Medicine. Edited by Kryger MH, WB Saunders, Philadelphia 2010.

37. Javaheri S. Heart failure. In: Principles and Practices of Sleep Medicine, 5/e. Edited by Kryger MH, Roth T, Dement WC; WB Saunders, Philadelphia 2011; 1400-1415.

Prevalence and Incidence of Diabetes in the U.S.

- More than 16 million Americans have diabetes [9]
- 2/3 will die of cardiovascular complications
- By 2050, an additional 18 million with diagnosed diabetes in the U.S., and 37% of this increase will be due to changes in the age, sex, and race composition of the population, 27% will be due to population growth, and 36% will be due to changes in prevalence rates. [9]

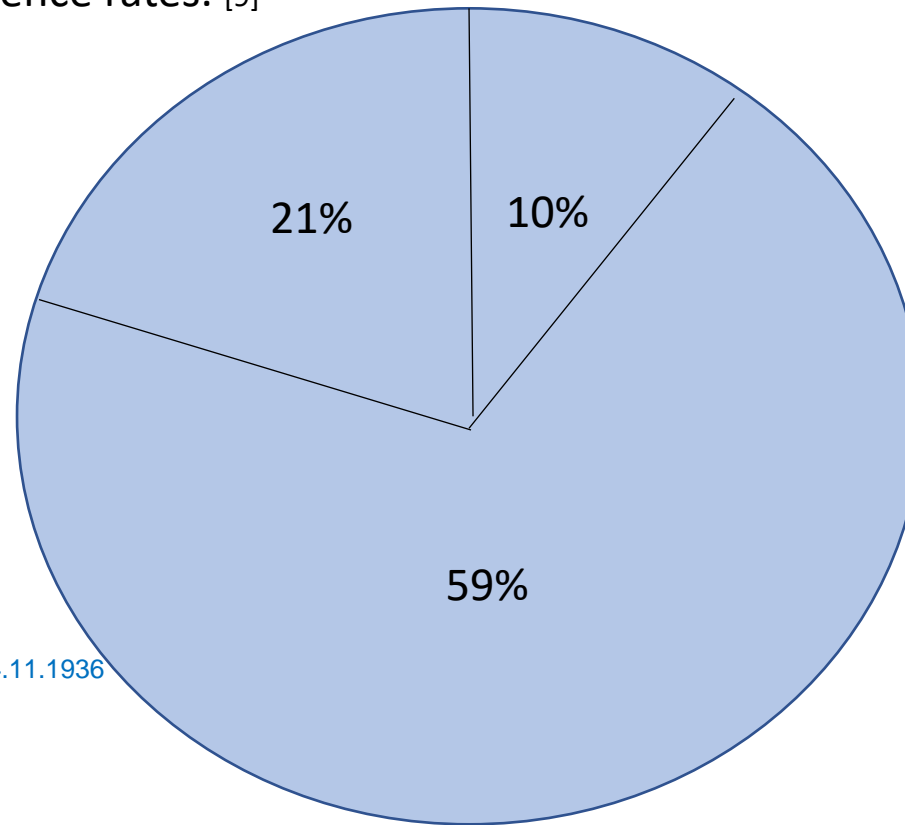


Figure 15.

Type 2: 800,000
new cases annually

Risk of Cardiac Death with Type 2 Diabetes Mellitus

Finnish Population-based cohort study
from 1982-1990 [14]

- 1,059 Diabetics
- 45-64 years of age

Compared with random sample of
1,373 nondiabetics

BOTTOM LINE

Diabetes type 2 is equivalent to having a
prior myocardial infarction

- Diabetic with prior MI had the worst prognosis
- Nondiabetic subjects without prior myocardial infarction had the best prognosis.
- **Diabetic without prior MI and nondiabetic with prior myocardial infarction had an intermediate survival rate, and these two groups had similar outcomes.**

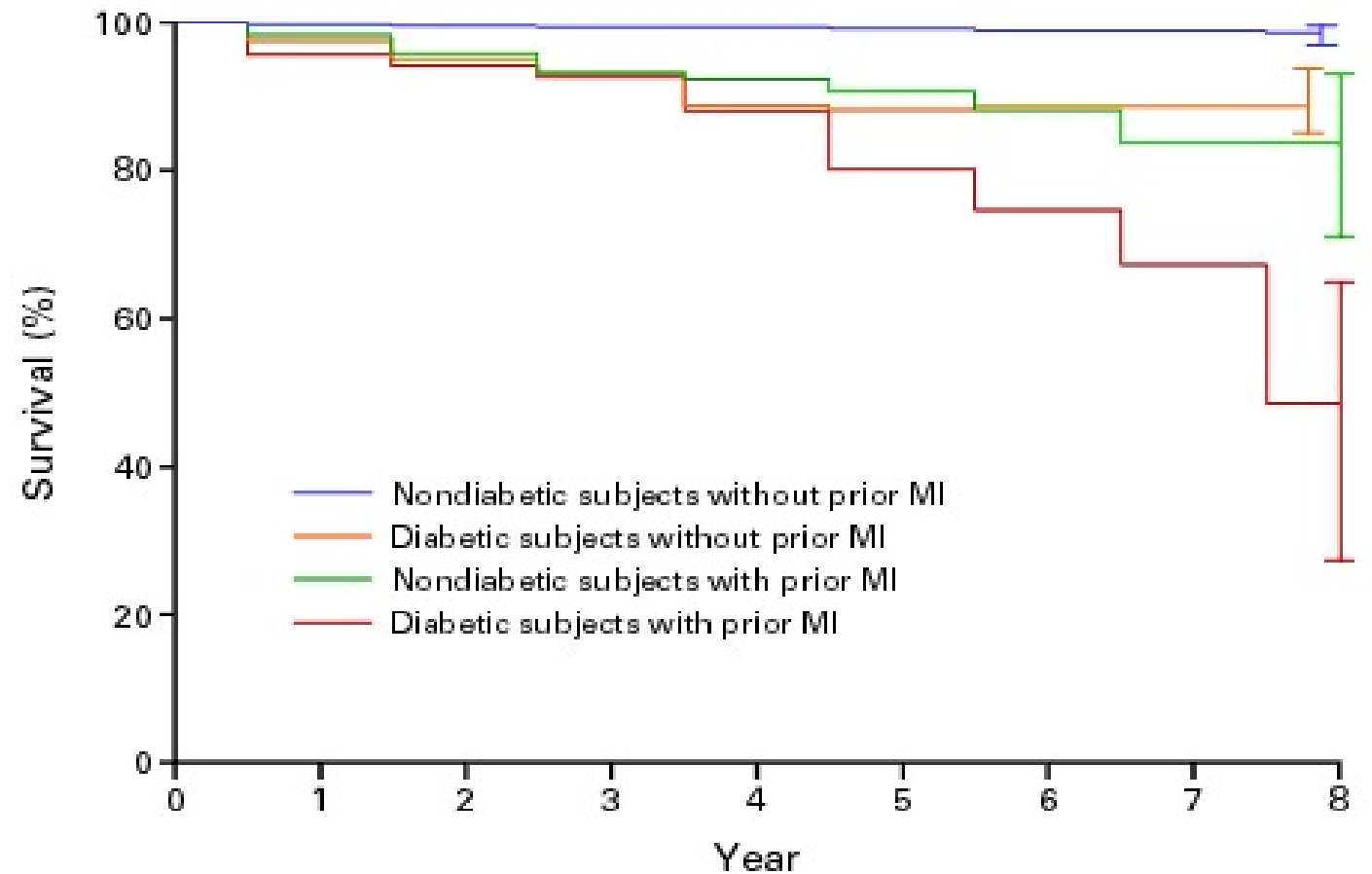


Figure 20. Kaplan-Meier estimates of the probability of death from coronary heart disease.

14. Haffner et al: N Engl J Med 1998; 339:229-234, DOI: 10.1056/NEJM199807233390404

The Relationship Between OSA and Diabetes Type 2

- **OSA is a comorbidity of type 2 diabetes:** more prevalent among diabetics than in non-diabetics. In the SHHS, a pooled study of multiple population-based cohorts in the USA, the prevalence of mild OSA ($5 \leq \text{AHI} < 15$) and moderate-to-severe OSA ($\text{AHI} \geq 15$) was 33.9% and 23.8%, respectively, in patients with self-reported diabetes, but 27.0% and 15.6% in non-diabetics, respectively [22]
- **Patients with more severe OSA were more likely to have diabetes** [22].

Insulin resistance and sensitivity

- Mitigating insulin resistance is critical. However, the findings regarding the effect of CPAP were inconsistent on fasting insulin levels alone [23][24][25][30][27][31]
- Change in bodyweight was the main determinant of insulin resistance in obese OSA patients [32]. Weight gain reported by some patients with increasing insulin resistance in that study, minimizing the impact of confounding factors is very important to examine the effect of CPAP therapy on insulin resistance.
- Harsch *et al.* [33]: beneficial effect of CPAP treatment on insulin sensitivity measured with the hyperinsulinemic euglycemic clamp. Patients with moderate-to-severe OSA ($n = 40$), **insulin sensitivity increased significantly after 2 days, and remained stable after 3 months of CPAP treatment** [33].
- De Lima *et al.* [34] **CPAP treatment for 2 months reduced insulin resistance in moderate-to-severe OSA patients.**

30. Lam JC, Lam B, Yao TJ, et al. A randomised controlled trial of nasal continuous positive airway pressure on insulin sensitivity in obstructive sleep apnea. *Eur Respir J* 2010; 35: 138–145.

31. Lindberg E, Berne C, Elmasry A, et al. CPAP treatment of a population-based sample—what are the benefits and the treatment compliance? *Sleep Med* 2006; 7: 553–560

32. Garcia JM, Sharafkhaneh H, Hirshkowitz M, et al. Weight and metabolic effects of CPAP in obstructive sleep apnea patients with obesity. *Respir Res* 2011; 12: 80. 28.

33. Harsch IA, Schahin SP, Radespiel-Troger M, et al. Continuous positive airway pressure treatment rapidly improves insulin sensitivity in patients with obstructive sleep apnea syndrome.

34. *Am J Respir Crit Care Med* 2004; 169: 156–162. 29. de Lima AM, Franco CM, de Castro CM, et al. Effects of nasal continuous positive airway pressure treatment on oxidative stress and adiponectin levels in obese patients with obstructive sleep apnea. *Respiration* 2010; 79: 370–376.

Pathophysiological Consequences of Sleep Apnea Adversely Affect Progression of Heart Failure

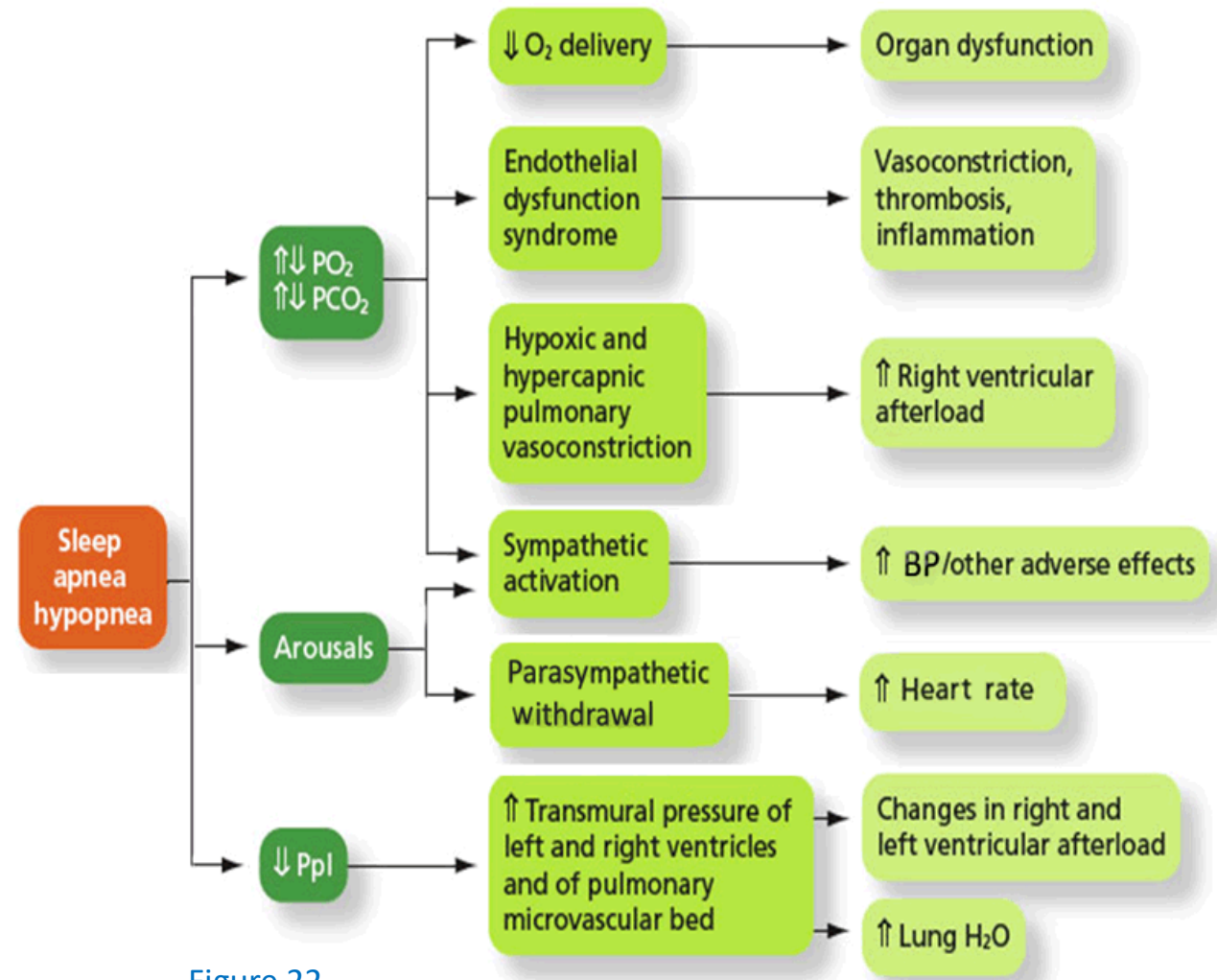


Figure 22.

35. Javaheri S. Sleep-related breathing disorders in heart failure. In: Heart Failure, A Companion to Braunwald's Heart Disease, edited by Douglas L. Mann. WB Saunders, Philadelphia 2010; 471-487.

1. Bacteria from periodontal plaques have been shown to induce platelet activation, aggregation, and thrombosis.
2. *Streptococcus mutans* and *S. sanguinis*, can lead to localized thrombus formation, and secretion of pro-inflammatory cytokines from the platelets lead to inflammation, atherogenesis, and thrombogenesis [42,43]. *Porphyromonas gingivalis* has also been shown to induce CVD by activating factor X, prothrombin and protein C, promoting thrombogenesis[44].
3. Systemic inflammation induced by periodontitis can promote molecular mimicry and generation of self-reactive antibodies targeting heat shock proteins (HSP). Bacterial invasion and systemic inflammation increase HSP production in endothelial cells [45]. Oral bacterial HSP generate antibodies that in turn attack the HSPs produced by the stressed endothelium [46].
4. Perschinka et al: CVD promotes expression of HSP60 and increased adhesion molecules by endothelial cells, facilitate formation of fatty streaks to irreversible atherosclerotic changes [47].

[42] J.R. Fitzgerald, T.J. Foster, D. Cox, The interaction of bacterial pathogens with platelets, Nat. Rev. Microbiol. 4 (6) (2006) 445–457, <https://doi.org/10.1038/nrmicro1425>.

[43] S.W. Kerrigan, D. Cox, Platelet-bacterial interactions, Cell. Mol. Life Sci. 67 (4) (2010) 513–523, <https://doi.org/10.1007/s00018-009-0207-z>.

[44] T. Imamura, J. Potempa, S. Tanase, J. Travis, Activation of blood coagulation factor X by arginine-specific cysteine proteinases (gingipain-Rs) from *Porphyromonas gingivalis*, J. Biol. Chem. 272 (25) (1997) 16062–16067, <https://doi.org/10.1074/jbc.272.25.16062>.

[45] G.J. Seymour, P.J. Ford, M.P. Cullinan, S. Leishman, K. Yamazaki, Relationship between periodontal infections and systemic disease, Clin. Microbiol. Infect. 13 (Suppl. 4) (2007) 3–10, <https://doi.org/10.1111/j.1469-0691.2007.01798.x>.

[46] G.J. Seymour, P.J. Ford, M.P. Cullinan, S. Leishman, K. Yamazaki, Relationship between periodontal infections and systemic disease, Clin. Microbiol. Infect. 13 (Suppl. 4) (2007) 3–10, <https://doi.org/10.1111/j.1469-0691.2007.01798.x>.

[47] J. Mollenhauer, A. Schulmeister, The humoral immune response to heat shock proteins, Experientia 48 (7) (1992) 644–649, <https://doi.org/10.1007/BF02118310>. [19] H. Perschinka, M. Mayr, G. Millonig, et al., Cross-reactive B-cell epitopes of microbial and human heat shock protein 60/65 in atherosclerosis, Arterioscler. Thromb. Vasc. Biol. 23 (6) (2003) 1060–1065, <https://doi.org/10.1161/01.ATV.0000071701.62486.49>

Native Valve Infective Endocarditis

Epidemiology

- 10K-15K new cases annually
- >50% are older than 54 years of age.
- Uncommon in children; usually associated with underlying structural heart disease, surgical repair of cong. Heart disease, or nosocomial catheter-related bacteremia.
- What about Periodontal disease????????????????????

Epidemiology

- Male >female (2.5:1)
- Chronic rheumatic valvular disease has been supplanted by MVP with regurg and degenerative aortic valve disease as the leading cardiac conditions underlying bacterial endocarditis in adults.
- Nosocomial endocarditis
- Mitral (85%)>aortic>tricuspid>pulmonary
- Tricuspid (0-6%), predominantly in IVDA
- PDA, VSD, Coarctation, Bicuspid AV, TOF, and PS underly 6-24% of cases.



Figure 22: C.D. Fobes, W.F Jackson; A Colour Atlas and Text of Clinical Medicine.1993

Pathophysiology

1. The atria are infiltrated by inflammatory cells, underlying the theory that systemic inflammation can drive the development of arrhythmia [48].
2. Patients with AF have increased levels of inflammatory markers (CRP, TNF- α , and plasma IL-6) [49]. CRP downregulates nitric oxide, promoting endothelial cell apoptosis [50]. TNF has also been shown to contribute to the pathogenesis of AF via augmenting pulmonary vein arrhythmogenicity and inducing abnormal calcium homeostasis [51].
3. Chen et al. found that patients with periodontitis had a 31 % higher risk of developing AF than patients without periodontal disease [52]

OBSTRUCTIVE SLEEP APNEA....THE TRUE SILENT KILLER

- 40-80% in patients with CHF, Hypertension, CAD, Pulmonary Hypertension, Atrial Fibrillation, and Stroke.
- Despite its high prevalence, “OSA is often under-recognized and undertreated in clinical practice”
- American College of Cardiology: recommends screening for OSA in patients with
 1. Uncontrolled hypertension, pulmonary hypertension
 2. Recurrent atrial fibrillation after either cardioversion or ablation.
 3. New York Heart Association class II-IV heart failure with suspicion of sleep-disordered breathing/daytime hypersomnolence, a formal sleep assessment is reasonable.
 4. Ventricular tachycardia or survivors of sudden cardiac death in whom sleep apnea is suspected, evaluation for sleep apnea needs to be considered.
 5. Post stroke.
 6. Myocardial infarction, arrhythmias, or appropriate shocks from implanted cardioverter-defibrillators are associated with OSA.

Oral appliances can be considered for those with mild to moderate OSA or for CPAP-intolerant patients.

Rules of Engagement

- Speak the language of physicians “Medical Speak”
- “Medical Speak”
- Ability to assimilate standardized, powerful medical knowledge into your dental practice and engagements with medical providers.
- Defines a meaningful conversation with providers
- Opportunity to become legitimate



ToolBox: Let's fix the problem!.....Implement.....Execute.....

How to engage with medical providers to ensure that Oral Appliance therapy is considered a viable option for patients:

1. Discuss with patients and respective healthcare providers (KNOW THE TEAM) cardiovascular implications of untreated OSA: Keep relationship with physicians/Providers up to date.

Target Referrals: Physician, Nurse Practitioners, Physician Assistants, Chiropractors, Podiatrists, Teachers

2. Demonstrate proficiency in baseline knowledge of how OSA negatively affects cardiovascular health
3. **Incorporating “Bottom Line” Fundamental Cardiovascular Health Knowledge into Sleep Dentistry Practice.**
4. **Perform routine health status surveys to define impact of oral appliance therapy: depression scale, improve medical compliance, reduce readmissions.**
5. **Understanding Cardiovascular Health promotes a truly objective conversation with physician and mid-level providers.**
6. **Be more up to date with Health Care Trends than providers who just read sleep studies.**
7. **Heart-Sleep CME for Providers.**



Key Takeaways and Pearls

1. **OSA** is the “**True Silent Killer**”
2. Implement **strategies** to develop and transform your practice into an objective Outcome-Based service to optimize patient safety and wellness.
3. Incorporate cardiovascular health wellness into your daily routine to become a critical component of the Medical Team and ultimately improve patients’ overall **Quality of life**, **Health status outcomes** and **Reduce READMISSIONS**.