CDA JOURNAL, VOL 40, Nº2

Dentistry-Based Approaches to Sleep-Disordered Breathing, Algorithms, and Multidisciplinary Perspectives

MICHAEL S. SIMMONS, DMD; ROY ARTAL, MD; MIGUEL A. BURCH, MD; RICHARD P. CAIN, MD; RUWANTHI CAMPANO, MD, MS; HARRY G. COHEN, MD; CHRISTOPHER A. MANN, PHD; TODD MORGAN, DMD; JUDITH A. OWENS, MD, MPH; ANDREW PULLINGER, DDS, MSC; ROBERT RELLE, DDS; JOHN M. ROESLER, MD; JOHN SOTOS, MD; JOHN A. YAGIELA, DDS, PHD; RONALD B. ZIMAN, MD; AND JERALD H. SIMMONS, MD

ABSTRACT Sleep disorders affect more than 20 percent of the U.S. population, but less than 7 percent have been medically diagnosed. Dentists are ideally positioned to identify many patients who fall under the grouping of sleep-disordered breathing. This paper presents perspectives on sleep-related issues from various medical specialties with a goal to broaden the dentist's appreciation of this topic and open avenues of communication. Algorithms are proposed to guide dentists following positive screenings for sleep-disordered breathing.

AUTHORS

Michael S. Simmons, DMD, is a diplomate ABOFP, FAGD, lecturer at the University of California, Los Angeles, a clinical assistant professor at the University of Southern California, serves on the Board of AADSM and ASAA, and is engaged in furthering dentistry's involvement in sleep medicine.

Roy Artal, MD, FCCP, FAASM, is a diplomate in sleep medicine and pulmonology, clinical instructor, University of California, Los Angeles, School of Medicine; medical director of Tower Sleep Medicine in Los Angeles.

Miguel A. Burch, MD, is the associate director of general surgery and minimally invasive surgery at Cedars-Sinai Medical Center in Los Angeles.

Richard P. Cain, MD, is a cardiologist, a fellow at Wadsworth VA, director of cardiac rehabilitation at Providence Tarzana Medical Center, and in private practice in Tarzana, Calif.

Ruwanthi Campano, MD, MS, otorhinolaryngology/head and neck surgery/and facial plastics and reconstructive surgery, is in private practice in Lancaster, Calif.

AUTHORS CONTINUE ON 169

According to a 2006 landmark report by the Institute of Medicine, sleep disorders and sleep deprivation remain an enormous unmet public health problem, yet sleep disorder "awareness among the general public and health care professional is low, given the magnitude of the burden."[1] About 50 to 70 million people were reported in 2003 to be chronically affected by sleep disorders in the United States, with these numbers increasing in concert with the obesity epidemic.[2,3] The International Classification of Sleep Disorders (ICSD-2) lists approximately 100 different sleep disorders.[4] The four main sleep disorder categories, according to most people affected, include insomnia (5 percent to 35 percent of the U.S. population) circadian rhythm problems (7 percent to 16 percent), sleep-related movement disorders (4 percent to 12 percent), and sleep-related breathing disorders (SRBDs, 3 percent to 25 percent).[4-12] These ranges in prevalence vary by the population sample and the inclusionary diagnostic criteria. For example, Schroeder et al. determined that obstructive sleep apnea (OSA, a most serious form of SDB) occurred in 50 percent of individuals >65 years old in a random community sample.[13] Shochat et al. found the prevalence of insomnia to be 69 percent in a primary care population, and Sack et al. reported circadian rhythm disorders in >50 percent of the totally blind.[14,15] In addition to those directly affected by sleep disorders and sleep deprivation are bed partners, nearby sleepers, parents of affected children, and even children of affected parents. It

is therefore reasonable to conclude that at least one-third of the U.S. population will be impacted by a significant sleep disturbance during their life.[16]

In terms of morbidity and mortality, the most important ICSD-2 sleep disorder diagnostic category is SRBD (which includes OSA), and it is here that dental professionals can make significant contributions as part of the sleep medicine team.[17] Only about 5 percent to 10 percent of patients with OSA are diagnosed; overlooked are 82 percent of males and 93 percent of females with moderate to severe and symptomatic OSA[18,19] Even fewer patients receive adequate treatment with therapies such as continuous positive airway pressure (CPAP).[20] Given the 300 million dental patient visits per year in the United States, the dental office may be an ideal environment to apply routine SDB or sleep wellness screening, possibly identifying many affected individuals before the onset of serious medical and psychosocial consequences.[21]

In spite of the high prevalence of sleep disorders, there is a great deficit in our health care educational system addressing sleep medicine. In 1978, Orr et al. reported less than one hour was spent on sleep medicine during the four-year MD educational process.[22] By 1993, educational time had increased to about two hours, and by 1998 reported time spent was up to 2.11 hours.[23,24] With such limited exposure it is no wonder that physicians are generally not well-prepared to identify and address the gamut of sleep disorders. On the positive side, there is a burgeoning interest in sleep medicine. The American Academy of Sleep Medicine (AASM) now boasts more than 9,100 members, with 3,655 diplomates of the American Board of Sleep Medicine. Additional boarded sleep specialists are recognized since 2007 in the medical disciplines of internal medicine, family practice, pediatrics, otolaryngology and neurology/psychiatry. These would all be considered medical sleep specialists (MSS). Additionally, the American Academy of Dental Sleep Medicine (AADSM), the premier dental group devoted to sleep issues, now has a membership of more than 2,500 with more than 160 diplomates, and is one of the fastest-growing professional sleep organizations. In support of this interest by dentistry is the increasing education given at the predoctoral level, where the time devoted to sleep medicine has increased from a mean of about 30 minutes in 2003 to almost three hours in 2009.[25]

If a dentist wishes to include sleep medicine as a part of his/her clinical practice, gaining perspectives of clinicians from other medical specialties provides insights that can enhance multidisciplinary care. Primary care physicians (PCPs), along with dental sleep medicine experts (DSEs), can together adequately manage a large number of the simpler SDB cases. For those dentists who would prefer to just screen for SDB, they may consider the PCP, DSE, or MSS for referral to manage the case. More involvement by the dentist still requires reliance on the MSS and/or other medical specialists to assist in management, depending on the complexity of each case.

Specialist Input

Anesthesiologist

OSA is an independent risk factor for anesthetic mortality and is linked to various comorbidities that also have implications for anesthesia care: morbid obesity, pulmonary and treatment-resistant hypertension, congestive heart failure, cardiac dysrhythmias, metabolic syndrome and type 2 diabetes mellitus, hypothyroidism, gastroesophageal reflux disease (GERD),

AUTHORS, CONTINUED FROM 168

Harry G. Cohen MD, FACP, is assistant professor of family medicine at Keck School of Medicine at the University of Southern California, and a visiting physician for Vitas Hospice.

Christopher A. Mann, PHD, FAASM, interprets sleep studies for multiple sleep centers and performs site visits for the American Academy of Sleep Medicine.

Todd Morgan, DMD, is a diplomate ABDSM, chief of dentistry, Scripps Memorial Hospital-Encinitas. He also is an inventor and collaborator, NIH research: oral appliance therapy, and a board member of AADSM.

Judith A. Owens, MD, MPH, DABSM, is the director of Sleep Medicine at Children's National Medical Center in Washington, D.C.

Andrew Pullinger, DDS, MSC, is a professor at the University of California, Los Angeles, School of Dentistry, Section of Oral Medicine and Orofacial Pain, co-director UCLA Orofacial Pain and Oral Medicine Faculty Practice.

Robert Relle, DDS, is a diplomate ABOMS, practices in Century City, Calif., focusing on orthognathic surgery, and is a lecturer at University of California, Los Angeles, School of Dentistry.

John M. Roesler, MD, is an assistant clinical professor in otolaryngology at University of California, Los Angeles' David Geffen School of Medicine and in private practice in Tarzana, Calif.

John Sotos, MD, is chief executive officer of Apneos Corporation, a cardiologist, and an adjunct professor of medicine at the University of North Carolina.

John A. Yagiela, DDS, PHD, is a distinguished professor emeritus, Section of Dental Anesthesiology, University of California, Los Angeles, School of Dentistry, in Los Angeles.

Ronald B. Ziman, MD, FACP, FAAN, is board-certified in internal medicine, neurology, vascular neurology, a clinical associate professor of neurology at University of California, Los Angeles, and practices in Northridge, Calif.

Jerald H. Simmons, MD, FAASM, is a neurologist and a sleep disorders specialist who trained at Stanford University. He is past co-director of University of California, Los Angeles' Sleep Disorders Center, and currently directs multiple sleep centers in Texas.

TABLE 1

OSA and Anesthesia Management

- Screen patients for OSA preoperatively. Refer patients for preoperative evaluation and treatment where the probability of OSA is high, surgery is elective, and there is a likely need for postoperative opioid analgesia or sedation.
- Develop an anesthetic plan tailored to the severity of the patient's OSA and the planned procedure.
- When diagnosed with OSA and compliant with PAP, encourage its use preoperatively and ensure it is available for perioperative use.
- Where previously diagnosed but not compliant with PAP, reinstruct in its use.
- Avoid sedative premedication unless the patient's ventilation is being continuously monitored.
- Use regional anesthesia and analgesia where feasible.
- When general anesthesia is used, intubation is preferred. Be prepared for difficult intubation and other problems in airway maintenance. Use techniques that allow early return of consciousness (e.g., desflurane, nitrous oxide, propofol, remifentanil).
- Try to minimize postoperative opioid analgesia or sedation (consider acetaminophen, nonsteroidal analgesics, dexmedetomidine, low-dose ketamine).
- Keep patients on continuous ventilatory monitoring postoperatively and resume PAP as soon as feasible. Continuous monitoring may be discontinued if oxygen saturations during sleep remain above 90 percent while breathing room air. Patients requiring ongoing opioid analgesia or sedation should remain monitored until this need abates.
- Avoid the supine position. Use lateral positioning, a nasopharyngeal airway, and oxygen therapy where PAP is refused and upper airway obstruction is problematic.
- Inform patients with unanticipated difficult airways of the possibility of OSA and refer for sleep studies when clinically indicated.

Modified from references Nos. 27 and 29.

and head and neck cancer. Anesthetic concerns specific to OSA begin preoperatively and extend well into the postoperative period. The same anatomic and neurophysiologic derangements in people with OSA that promote repeated collapse of the airway during sleep underlie the respiratory complications associated with anesthesia. These derangements include a narrow supraglottic airway and excessive relaxation of muscles that, during consciousness, maintain pharyngeal wall tension (e.g., sternohyoid) and prevent the tongue (e.g., genioglossus) and soft palate (e.g., tensor veli palatini) from occluding the airway.

Benzodiazepines and other sedative drugs prescribed or administered for preoperative anxiety control may easily induce sleep in chronically fatigued individuals. Because these drugs obtund the protective arousal response that restores airway patency following acute obstruction and hypoxia/hypercarbia, patients with OSA are at increased risk of anoxic death. During induction, there is a much greater likelihood of encountering a difficult airway, which can complicate both ventilation and intubation. The greatest danger, however, lies in the postoperative period. Airway obstruction and apnea are particularly likely when parenteral opioids are required for postoperative pain relief. Case reports document lethal outcomes when such patients are not intensively monitored for extended periods after anesthesia.[26]

In 2006, the American Society of Anesthesiologists (ASA) published guidelines for the perioperative management of patients with OSA; subsequently, algorithms for the preoperative evaluation and perioperative management of patients with known or suspected OSA have been developed.[27,28]

Several instruments exist that can help clinicians identify and classify patients with OSA, including the Berlin Questionnaire, the ASA Checklist, and the Stop-Bang Questionnaire.[28] Of these, the Stop-Bang Questionnaire is easy to use and has the highest sensitivity with acceptable specificity. **TABLE 1** outlines the anesthetic management of patients with OSA.[27,29]

The recommendations listed in **TABLE 1** directly apply to dentists (oral surgeons, dentist anesthesiologists) who administer deep sedation/general anesthesia. For other dentists, the implications inherent in **TABLE 1** strongly suggest that patients with OSA are poor candidates for in-office sedation using oral or parenteral benzodiazepines or similar agents. Even the prescription of oral opioids for postoperative pain relief may be problematic and should be approached with caution. This speaks clearly to the dentist minimally screening for SDB.

Bariatric Surgeon

The most common modifiable cause of sleep apnea is obesity, which is defined as a body mass index (BMI) >30 kg/m^2. For every 10-point increase in BMI there is a 32 percent increase in the apnea-hypopnea index (AHI, the number of episodes of sleep-disordered breathing per hour).[30] As the epidemic of obesity continues to spread, one-third of Americans are now considered obese, and the fastest-growing subset is the super morbidly obese (BMI >50, or 150 pounds above the ideal body weight).[29,31,32] Management approaches utilizing oral appliances and even upper airway soft- and hard-tissue surgery have their greatest failures within this population as a result of the anatomical and functional restrictions of the obese airway. Only tracheostomy is a predict-

able upper airway surgical approach.

Although a loss of 10 percent of body weight was found to decrease AHI by 26 percent, only intensive medically supervised diets produced 10 to 20 kg of weight loss within six months, and this weight was universally regained within weeks of ceasing dieting.[30,33,34] Bariatric surgery, either gastric bypass or lap-band surgery, has been shown to maintain weight loss for 10 years or longer and to reduce overall mortality by 24 percent. Sleep apnea resolution after bariatric surgery occurs in concert with the weight loss and can start in the first three months after surgery.

A recent meta-analysis showed that postoperatively the AHI improved from 55 to 16 episodes per hour.[35] Despite these improvements, the AHI remains sufficiently high for some patients to warrant continued therapy. Since OSA is grossly underdiagnosed even in patients with morbid obesity who present for bariatric surgery, mandatory testing of all patients for OSA with polysomnography before bariatric surgery is recommended.[36]

Cardiologist

OSA has potentially deleterious effects on the cardiovascular system. A recent review identified nine different physiological mechanisms by which apneas can derange cardiovascular function.[37] Moreover, these mechanisms impact all major cardiovascular disease entities: hypertension, heart failure, dysrhythmias, atherosclerosis, and ischemia. Clinical studies show about a 50 percent prevalence of OSA in patients with hypertensive cardiovascular disease, 33 percent with coronary artery disease, 30 percent to 40 percent with heart failure, and 50 percent with stroke. About half of the patients with atrial fibrillation have OSA.[38] It is believed the severe repeated oxygen desaturations occurring with OSA can cause various dysrhythmias, such as severe bradycardia, heart block, and premature ventricular contractions. Finally, OSA has been associated with increased risk of death from nocturnal cardiac events, in particular from midnight to 6 a.m., when sudden death from cardiac causes occurred in 46 percent of people with OSA compared to 21 percent of people without OSA.[39]

Despite these associations, it is not possible to describe succinctly the degree to which OSA causes or exacerbates cardiovascular disease or the effect of OSA treatment on cardiovascular disease, for two reasons: 1) the relationship depends on the severity of both the OSA and the cardiovascular disease; and 2) data from the limited clinical studies to date are often equivocal.[40,41] Formal communication is recommended as cardiologists are, in general, increasingly conscious of the detrimental effects of OSA.[41] Cardiology patients with severe OSA and/or severe cardiovascular disease should generate the most immediate concern. For example, some patients with advanced heart failure are very sensitive to changes in blood pressure, fluid status, or other demands on the heart. Because dental treatment can transiently alter blood pressure and cardiac demand for the worse, coordination with the cardiologist is advisable in these patients. Patients taking multiple blood pressure or heart medicines are another population of concern, for the same reasons. Finally, in the unlikely event that a dentist is asked to treat central sleep apnea, cardiological consultation should be obtained first as the apnea may be a sign of inadequately treated heart failure.

A RECENT REVIEW identified nine different physiological mechanisms by which apneas can derange cardiovascular function.

Neurologist/Psychiatrist

There are many neurologic and psychiatric conditions that overlap with sleep disorders. Morning headache, for example, can stem from the sleep pathology of OSA.[42] Similarly, studies show a great range in association of depression (7 percent to 63 percent) and anxiety (11 percent to 70 percent) with OSA.[43] Several conditions are discussed below to assist the dentist's knowledge of conditions they may encounter when delving into a patient's sleep history.

Movement disorders may occur during any part of the wake and sleep cycle, and restless legs syndrome (RLS) occurs in both. It is a common condition presenting as an irresistible urge to move the symptomatic limbs, which then provides temporary relief. RLS affects about 10 percent of the adult population (increasingly in the elderly) and 2 percent of children. It occurs in women almost twice as often as in men. A patient lying in the dental chair who keeps shifting may have RLS even though appearing to have anxiety or another psychological condition. RLS responds well to dopamine agonists and, if unaddressed, can disrupt sleep to a significant degree. Periodic limb movements during sleep (PLMS) are usually complaints by the bed partner rather than by the patient, although the movements may disrupt both sleepers sufficiently to cause excessive daytime sleepiness (EDS). Clues include disrupted bedding and bed partner-witnessed kicking, flailing, or

other recurrent or potentially violent movements during the middle of the night. If such a sleep-related movement history is obtained, it should be considered quite significant and followed up with referral. The majority of patients with PLMS also have daytime RLS.[44]

Nocturnal myoclonus is in some ways similar to PLMS except that movements are more isolated, not periodic and usually occur in the transition between wakefulness and sleep.

REM behavior disorder (RBD) may appear as a movement disorder but is a parasomnia in which the patient acts out dream content such as talking, swinging arms, or exiting the bed in elaborate imaginary confrontations. A lack of the normal muscle atonia during REM sleep is the cause for this condition. There are a variety of etiologies and recent studies have described RBD as an early symptom of Parkinson's disease. Additionally under the topic of movement disorders is nocturnal or sleep bruxism (SB), which may be a forme fruste of a focal, mild, or more generalized dystonia. SB is intimately linked with SDB. The presence of temporomandibular joint dysfunction or arthritis in these patients may be a result of bruxism or be incidental to it.

Sleep-induced seizures often arise out of instability in the brain as it transitions between wakefulness and sleep. While some of these patients also have daytime seizures, those with only nocturnal seizures may go undiagnosed for years, and, unless their bed partner is able to give a clear history, they may never be identified. Such patients, however, may complain of muscle aches on awakening, enuresis (bedwetting or nocturnal urinary incontinence), traumatized oral tissues (e.g., bitten tongue), unexplained bruises or unexplained confusion on awakening. Other brain-related manifestations of OSA may include cognitive deficits especially in higher levels of executive functioning.[45]

Chronic pain conditions such as fibromyalgia have long been recognized as associated with poor sleep, which lowers the underlying pain threshold. Patients who present with a diagnosis of fibromyalgia or chronic pain without identifiable pathology should be considered for screening of sleep disturbances. Narcolepsy is fairly renowned despite its limited prevalence (less than 0.05 percent of the population[46]). Although this condition is notorious for extreme hypersomnolence, its most specific characteristic is cataplexy, the sudden loss of muscle strength brought on by an emotional event, such as laughing. This muscle weakness is attributed to the precipitous onset of a REM atonic state.

REDUCED SLEEP
in psychiatric conditions generally relates to reduced time in REM sleep, reduced total sleep time and reduced sleep efficiency.

Many psychiatric conditions, such as depression, are associated with sleep disorders. Falling asleep may be easily accomplished, but the affected individual awakens early, typically around 2 to 4 a.m., and is then unable to fall back asleep. Sleep-onset insomnia is more associated with anxiety, obsessive compulsive disorder, mania, hypomania, bipolar disorder, cyclothymic personality (where mood fluctuates but not to the extent as seen in bipolar disorder), and some forms of schizophrenia.[47] Reduced sleep in psychiatric conditions generally relates to reduced time in REM sleep, reduced total sleep time and reduced sleep efficiency. Medications addressing some psychiatric disorders influence sleep, as do some over-the-counter medications used for sleep. For example, antihistamines often taken as a sleep inducer can cause cognitive decline and memory problems in the elderly, which may not develop for weeks to months after starting the medication.[46] Opioids, anxiolytics, and sedative-hypnotics taken for insomnia, may similarly contribute to cognitive disorders, especially in the elderly who are more sensitive to such adverse effects. Of course, these drugs may also aggravate SDB.

Oral and Maxillofacial Surgeon

Oral and maxillofacial surgeons (OMFSs) have made important contributions to sleep disorder therapy by introducing surgical procedures for OSA and reporting on their outcomes. No treatment for sleep apnea is easily accomplished and acceptable to all patients.[38] Furthermore, the long-term compliance required of patients being treated with positive airway pressure (PAP) and oral appliances is not an issue for patients successfully cured with surgery.[47,48] It is therefore likely that surgery will maintain an important role in the treatment of OSA for those patients who cannot tolerate or who fail or decline nonsurgical treatment.[49]

No one surgical treatment algorithm has been adopted by the surgical community. Some surgeons treat OSA in an escalating manner, beginning with phase 1 surgery such as variations of adenoidectomy, tonsillectomy, uvulopalatopharyngoplasty, nasal or tongue surgery, and hyoid myotomy and suspension. Patients failing these procedures undergo maxillomandibular advancement (MMA) as a second

phase. Others advocate MMA as a primary definitive surgery, particularly for those patients with diffuse upper airway obstruction or facial skeletal anomalies.[50-54]

Over the last two decades, the trend toward MMA as a definitive primary surgery has gained support.[50] The Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine stated that, while MMA can improve sleep study parameters comparable to PAP, most other sleep apnea surgeries may improve clinical outcomes but are rarely curative for OSA.[55]

Referral to the OMFS for surgery is appropriate when a diagnosis of OSA has been established and when other treatment modalities such as PAP and oral appliances have been deemed inappropriate or ineffective by the clinician or declined by the patient. In addition to conservative therapy failure, OSA patients who may be referred to the OMFS practice include, most commonly, the adolescent or adult patient with a facial skeletal anomaly, particularly retrognathia, who has been referred by an orthodontist for orthognathic surgery. A diagnosis of OSA has often already been established, and the patient is seeking surgery to address both concerns. Otherwise, sleep disturbance information is elicited to confirm a possible diagnosis of OSA, and formal sleep consultation and polysomnography are obtained as indicated. This work-up is especially important for the cleft population, where OSA is known to be more prevalent.[56] All patients undergoing MMA must be informed of the risk of complications, and the discussion should include the heightened risk of sensory disturbance in the adult patient, stability issues and potential temporomandibular joint problems owing to the relatively larger maxillary and mandibular advancements commonly performed to treat OSA.

A less frequent but important referral to the OMFS is the infant or child with severe micrognathia who has been referred for mandibular distraction osteogenesis. Although patients with Pierre Robin sequence or a craniofacial malformation with associated micrognathia do not have OSA, there is a much more immediate risk of death from airway obstruction. Many other medical and surgical specialists are generally involved in the care of these children, who often have additional life-threatening anomalies and comorbid conditions. For these patients, decisions about alternative airway management, such as nasopharyngeal intubation, tongue-lip adhesion and tracheostomy, must be made by the multidisciplinary team of clinicians and the parents.[57,58] Even though mandibular distraction osteogenesis offers the possibility of definitive treatment, the benefits of this surgery must be weighed against the risks, including damage to tooth buds, mandibular nerve injury and premature consolidation of the osteotomy requiring reoperation. Its success is highly dependent upon the diligence of caregivers to perform daily activation of the distraction devices, and it requires a certain degree of patient cooperation.

IT IS IMPORTANT that dentists be aware of the many separate anatomic factors that contribute to these disorders.

Otolaryngologist

Snoring and OSA are caused by upper airway obstruction. It is important that dentists be aware of the many separate anatomic factors that contribute to these disorders. Snoring or OSA can result from nasal airway obstruction, obesity, mouth breathing, lesions within the upper airway, hypertrophy or enlargement of the adenoids, tonsils and base of the tongue, and vibration of structures such as the uvula and soft palate. Otolaryngologists can be useful partners to dentists who have an interest in screening for or who specifically focus on sleep disorders in their practices. While general dentists most commonly focus on ameliorating obstruction of airflow by the fabrication of custom oral appliances that advance the mandible and/or tongue base, otolaryngologists are accustomed to evaluating and managing the nasal and oropharyngeal blockage leading to upper airway obstruction. For example, adenotonsillectomy is a commonly employed procedure effective for SDB in children.[59,60]

Otolaryngologists perform several core surgical procedures to improve nasal breathing, including septoplasty (straightening of the nasal septum), turbinate reduction, and nasal valve reconstruction (adding internal cartilage grafts that support the nasal sidewalls to prevent collapse of the nostrils). These can be important to aid the use of nasal CPAP, reducing dependence on more cumbersome full-face masks.[61] Other soft-tissue surgical procedures that may help reduce snoring and alleviate OSA in appropriately selected patients include adenotonsillectomy, UPPP, radiofrequency palatoplasty, placement of soft-palate Pillar implants, lingual tonsillectomy, radiofrequency tongue-base reduction and hyoid suspension.[62] The overall efficacy of these individual procedures is still under inves-

tigation, and although there has been a trend toward concurrent multilevel approaches, some procedures have lost favor following long-term results analysis.[63,64]

Additionally, otolaryngologists are considered a last resort for unmanageable SDB for the morbidly obese, failure of all other reasonable therapies or, in rare instances, those who are born with congenital central hypoventilation syndrome.[65,66] The management of these severe disorders requires performance of a surgical bypass of the collapsible upper airway, i.e., tracheostomy. Although this approach is not as socially acceptable, it is life-saving, and patients can learn adaptive techniques for masking the surgical site. Finally, other upper airway pathology, such as throat cancer, epiglottitis and Ludwig's angina, may cause acute upper airway obstruction and require the creation of a surgical airway in the neck.

Pediatrician

The prevalence of OSA in the pediatric population is estimated to be 1 percent to 3 percent for children 2 to 18 years old.[67] The prevalence of primary snoring in children is believed to be 9 percent to 12 percent.[68] OSA occurs in all age groups, from infancy through senescence, but the peak age of presentation is 2 to 6 years, during which time the tonsils and adenoids are proportionately large relative to the airway. Ethnicity also influences the prevalence of OSA, with higher rates reported in African-Americans and Asians. Obesity has become an increasingly important factor in childhood OSA as obesity continues to increase in the pediatric population. Obesity may further narrow the upper airway because of fat deposition, mass loading of the neck with subcutaneous tissue, decreased upper airway caliber from additional loading of the chest wall and decreased lung volumes and, in some cases, abnormal central respiratory control.

Studies comparing neuropsychological functions in children with OSA have found impairments in tasks involving reaction time and vigilance, attention, executive functions, motor skills and memory, as well as impairments in school performance.[69,70] Measures of behavior and neuropsychological functioning in children following treatment (usually adenotonsillectomy) for SDB have documented significant improvement in daytime sleepiness, behavior and academic performance and neuropsychological measurements of attention, vigilance, reaction time, and cognitive functions.[71] Finally, several studies examining the prevalence of SDB symptoms in children with identified behavioral and academic problems have found an increased prevalence of snoring in children with behavioral concerns and in children being evaluated for or diagnosed with attention-deficit hyperactivity disorder.[72] A recent report indicates that children with OSA are heavy consumers of health care resources and that early diagnosis and treatment may be cost-effective in reducing the associated morbidity and mortality.[73]

ETHNICITY ALSO influences the prevalence of OSA, with higher rates reported in African-Americans and Asians.

Routine screening for snoring should be a part of normal health care visits.[74] Various testing methods, including imaging and home sleep testing, have not been validated; thus, full-night polysomnography in accredited sleep laboratories remains the "gold standard" for diagnosing children of all ages and development using age-appropriate equipment and well-trained staff.

Adenotonsillectomy is usually the first line of treatment for pediatric OSA. Reported cure rates after adenotonsillectomy range from 75 percent to 100 percent in normal healthy children.[75] Cure rates in obese children may not be as good, but even obese children benefit from adenotonsillectomy. The severity of the OSA affects the timing of surgery and the perioperative care. Children with very severe OSA who are at high risk of operative mortality related to surgery warrant stabilization beforehand. A sleep specialist should be involved in such circumstances. PAP is an effective and reasonably well-tolerated treatment option for a wide range of children with OSA, including infants, children with craniofacial syndrome, Down syndrome and those with developmental delay.[76] PAP has been successfully used as the initial treatment for children in whom surgery is not an option, in children who continue to have OSA despite surgery and also to stabilize the child medically before surgery.

Pulmonologist

Many respiratory conditions can affect sleep quality. Nocturnal dyspnea from a multitude of conditions, including chronic obstructive pulmonary disease (COPD), chronic cough and asthma, can lead to repetitive nocturnal awakenings, poor quality sleep, and EDS.[77] Patients with restrictive pulmonary disease, such as those who are morbidly obese, or individuals with neurologic disease and concommitant respiratory muscle weakness, may hypoventilate during sleep. Patients with chronic lung disease, whether it be

obstructive or restrictive, may suffer from chronic dyspnea leading to impaired sleep consolidation and nonrestorative sleep.[78] Such patients are also predisposed to suffer from abnormalities of gas exchange, making them more susceptible to the development of nocturnal hypoxemia and/or hypercarbia. These complications may arise with or without concomitant upper airway obstruction or apnea.[79]

As a general statement, patients with more complex heart-lung disease and concomitant OSA will require treatment with positive pressure ventilation and are not appropriate candidates for initial treatment with oral appliances.[80,81] CPAP-intolerant patients however, may be appropriate candidates for oral appliance therapy (OAT). Patients with a history of cardiopulmonary disease and sleep complaints are best advised to speak with their PCP or a pulmonologist.

Primary Care Physician (Internist/Family Practice)

Fortunately, attitudes toward the importance of sleep health are changing. The result is a broader recognition among both the health care community and patients that how much and how well we sleep matters. Patients increasingly self-refer to sleep disorders centers. The Internet is replete with sleep health websites covering topics ranging from insomnia to snoring to sleep apnea.

A dentist who is involved with using appliances for the treatment of snoring or OSA needs to recognize that patients with medical conditions should be followed in conjunction with their PCP. Hopefully, PCPs will be increasingly receptive to recognizing sleep disturbances and directing patients toward proper care.

The Medical Sleep Specialist

Sleep specialists come from many different medical specialties; when properly trained, they should be able to handle the full spectrum of sleep disorders. The MSS can be considered the quarterback for patients who are found to have a sleep disorder. A properly trained MSS should recognize the important role OAT plays in the treatment armament. The goal for each dentist expanding his/her practice to include dental sleep medicine is to partner with a MSS so that the dentist is not functioning beyond the scope of dentistry while involved in this field of clinical practice.

A BAD NIGHT'S sleep on an infrequent basis is different from insomnia that lasts a month or more.

Behavioral Sleep Specialist — Psychologist

Insomnia is often associated with SDB, has additive negative effects and therefore should be addressed to enable better triage by the dentist.[82,83] A bad night's sleep on an infrequent basis is different from insomnia that lasts a month or more. There are many causes of insomnia, with psychological, medical, and environmental contributors. Patients with insomnia report having insufficient sleep and often present with symptoms of daytime hypersomnolence, moodiness, reduced affect, and decreased work productivity. The two primary treatment strategies are pharmacotherapy and cognitive/behavioral therapy (CBT). Medications, including hypnotics, anti-anxiety agents and antidepressants, are used particularly in acute cases, but CBT is more effective over extended periods.[84-87] Combining both therapies can also be effective.[88,89] Health care providers on a routine basis see patients who have insomnia complaints. Patients with problems remaining asleep likely require a sleep study, especially if experiencing EDS.

Determining the primary diagnosis of sleep disturbances can be challenging. Many patients referred to sleep centers often take medication for depression or anxiety. For some, the primary problem has to do with sleep, or the lack of it. Once the sleep disorder is treated, the patient no longer requires psychoactive medication. Other EDS can be related to time-shift changes or difficulty adapting to night or swing shifts. These circadian rhythm sleep disorders are diagnosed and treated by sleep specialists usually with behavioral approaches. When possible, the patient's sleep/wake schedule is progressively shifted (normalized) with use of artificial light to stimulate the brain wake times; medications such as melatonin may also help. Individuals certified in behavioral sleep medicine by the American Board of Sleep Medicine are highly competent in the utilization of CBT and other treatments for insomnia.

The Dentist's Role in SDB

Since many patients see their dentist more regularly than their primary care physician, the dentist may be the first health care provider to interface with the patient about sleep-related difficulties, especially regarding snoring, OSA, and bruxism. With this increasingly visible role of dentists in the management paradigm of SDB comes an increased responsibility to include sleep-related concerns within the scope of their health survey. Three flowsheets in this paper summarize for dentists the general overview approach to SDB (**FIGURE 1**) and specific aspects of screening (**FIGURE 2**) and co-treatment (**FIGURE 3**). In comparison with other health care providers, the dentist may have a firmer founda-

Overview Algorithm

DDS collaborating with MSS on sleep disorders

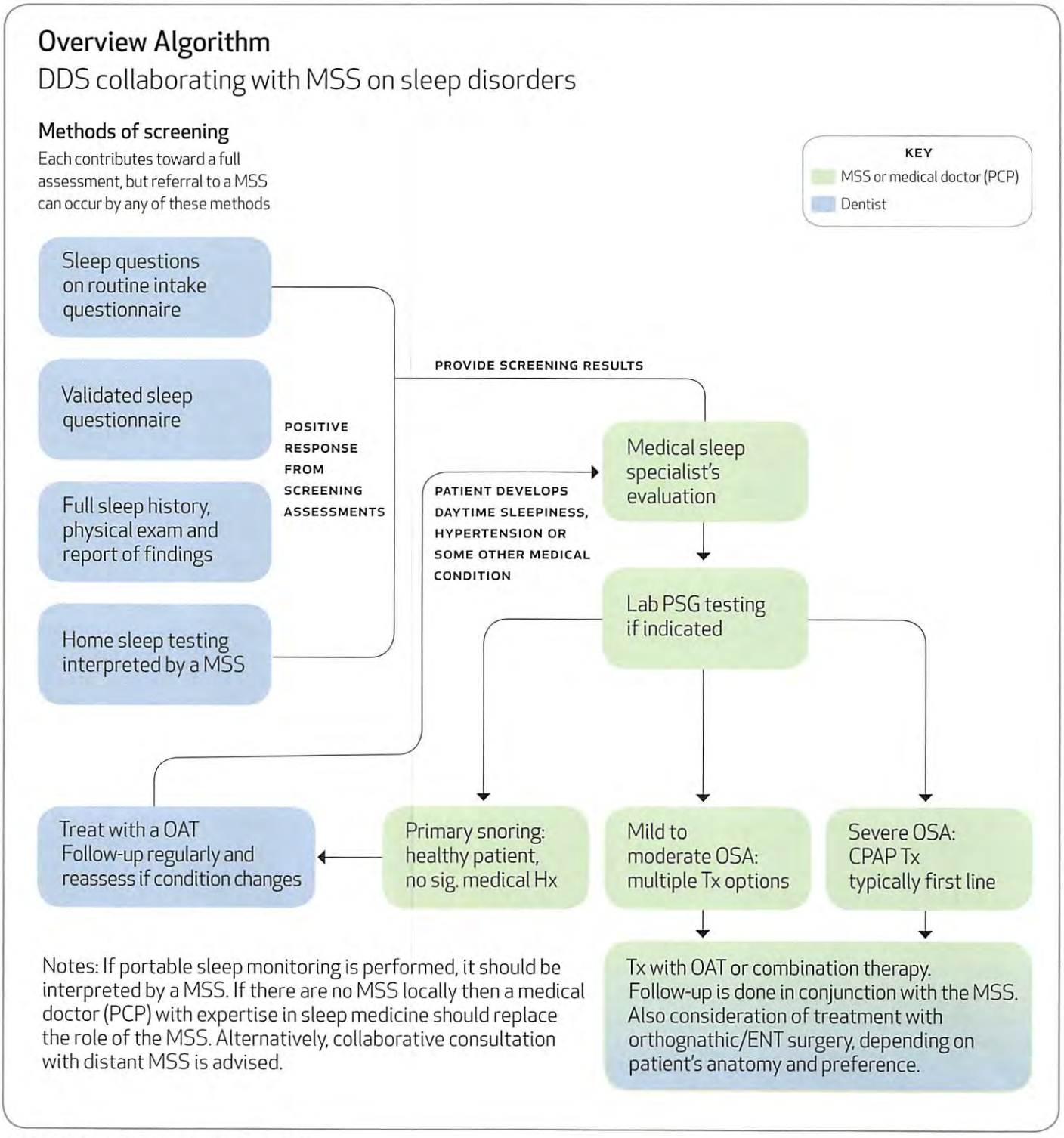

FIGURE 1. General overview algorithm for dentists.

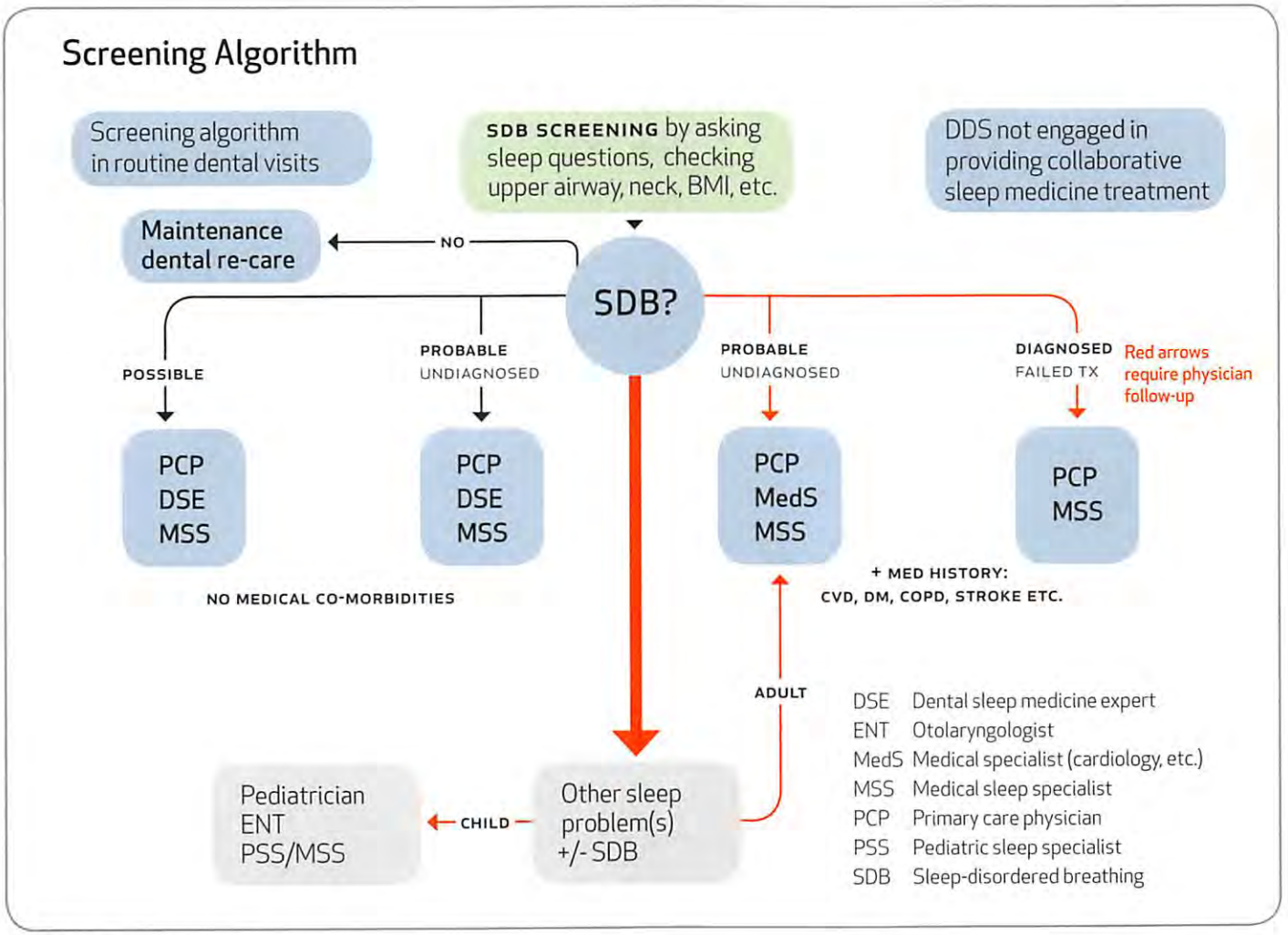

FIGURE 2. Screening algorithm for dentists.

tion on many sleep disorder presentations and be more comfortable in engaging other doctors in a multidisciplinary model.

An important issue for the dental practitioner is to determine when snoring is just simple primary snoring (PS) versus when is it nonbenign snoring, a marker for OSA or another nocturnal respiratory disturbance. The definition of PS is audible snoring without complaints of insomnia, EDS, or sleep disruption attributable to snoring or airflow limitation. The noises are not associated with significant airflow limitation, significant arousals from sleep, oxygen desaturation, or cardiac dysrhythmias.[5] Snoring may emanate from the nasopharynx down through the hypopharynx and glottis and be caused by such disparate conditions as allergic rhinitis, tonsillar hypertrophy, and inspiratory pharyngeal collapse. OAT might be quite successful for velopharyngeal airway collapse but it would likely not provide any airway benefit for allergic rhinitis. Designating a patient with a diagnosis of PS requires clear exclusion of any associated medical abnormalities.

A history must be void of conditions such as hypertension, daytime sleepiness, fatigue, etc., and a sleep study, at minimum home sleep monitoring, needs to yield normal results. Also not everyone who snores and is tired has sleep apnea. Just like any other medical condition, the diagnosis and management of a sleep-related complaint calls for a complete history and physical examination. Patients who snore but have only limited or marginal concerns related to their sleep, no comorbidities, a normal BMI, and a normal head and neck examination should probably only be encouraged to discuss their condition with their PCP or dentist sleep expert. If there are concomitant medical problems, such as hypertension, coronary artery disease, prior myocardial infarction or cerebrovascular accident, GERD, diabetes mellitus, or respiratory illness (e.g., asthma or COPD), they should raise red flags for the dentist. These problems, along with complaints of EDS and/or physical findings strongly correlated with OSA (e.g., obesity or crowding of the airway), should also heighten

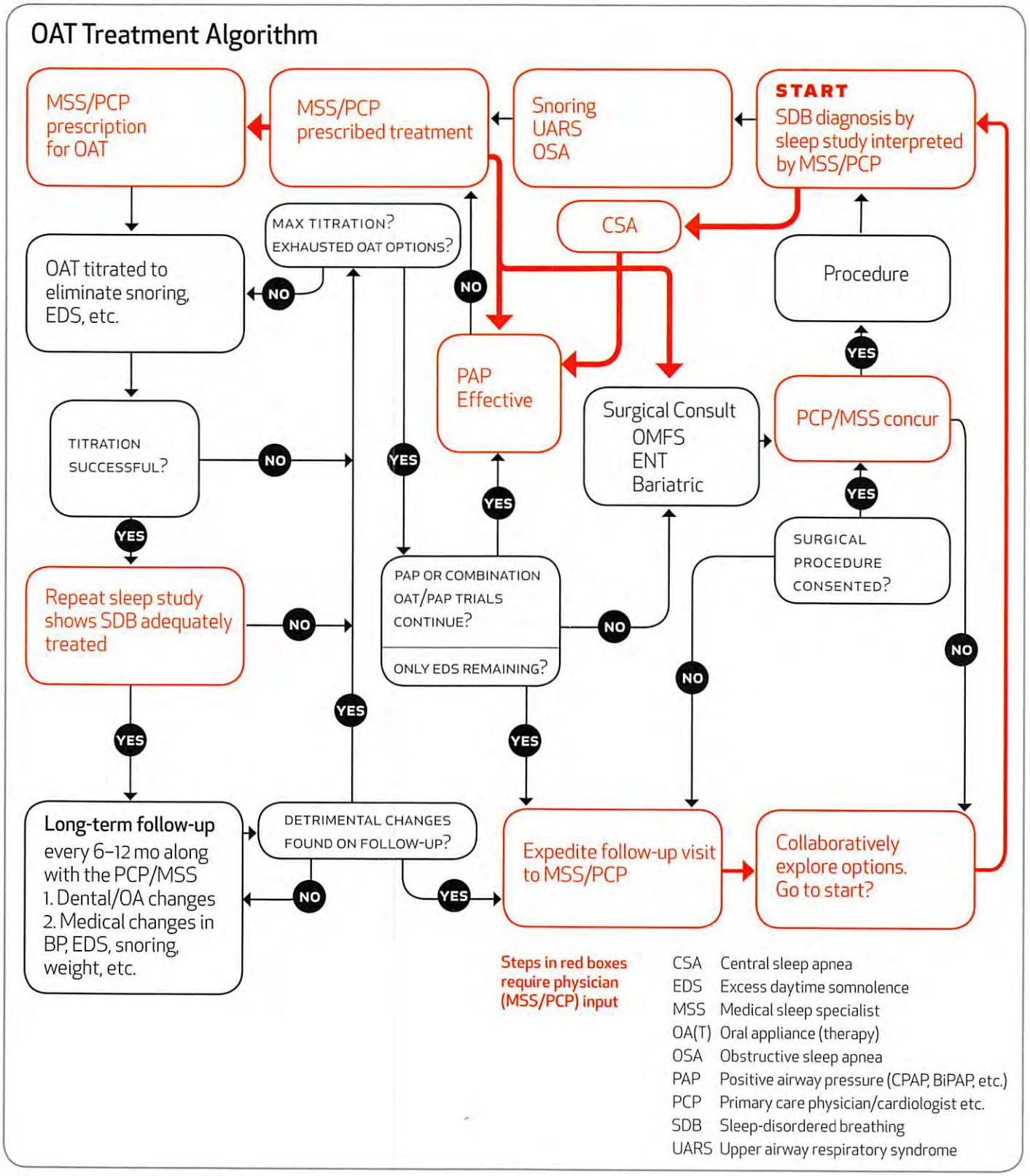

FIGURE 3. Treatment algorithm for dentists.

concern for the presence of significant SDB.

Knowledge of these signs and symptoms should stimulate communication with the patient's PCP. It might be necessary for the dentist to educate the physician regarding SDB. If this is the case, tact and support are essential to gently educate the PCP about sleep medicine. Another area needing dentist input is CPAP compliance. This area is an "orphan" not well-managed by sleep technicians or even prescribing physicians. Since less than 50 percent of CPAP patients consistently use their devices as prescribed, there is an obvious vacuum to be filled. CPAP treatment of OSA has suffered greatly from lack of focus on this ubiquitous problem. It is logical that the dentist could become an effective "compliance officer" for CPAP. A collaborative relationship between health professionals engenders shared confidence, and participation in holistic care is beneficial to both professionals and patients.

Conclusion

In its 2006 report regarding increasing awareness among health professionals of sleep-related problems, the Institute of Medicine stated, "In particular, medical, nursing, dentistry, and pharmacy students require greater exposure to the public health burden of sleep loss and sleep disorders."[2] Sleep medicine has proposed various strategies for the screening, treatment, and collaborative care of SDB by various health practitioners who limit dentist involvement to treating cases involving failure of CPAP.[56] By suggesting screening and treatment protocols for sleep disorders with the dental community's perspective in mind, the authors offer a potentially enhanced approach to patient care. Evolving treatment paradigms can enable the multidisciplinary field of somnology to move forward with a consistent, coordinated, and collaborative therapeutic approach benefiting all patients with SDB. Dentists are uniquely positioned as front-line health care practitioners to educate and engage patients. As a primary care screener and active participant in the identification and/or treatment of SDB, the dentist can play an integral role alongside the PCP and MSS. ■■■■

REFERENCES

1. Committee on Sleep Medicine and Research, Board on Health Sciences Policy, Institute of Medicine, Colten HR, Altevogt BM, eds., Sleep disorders and sleep deprivation: an unmet public health problem. Washington, D.C., The National Academies Press, 2006.
2. National Center on Sleep Disorders Research, 2003 Report on National Sleep Disorders Research Plan. U.S. Department of Health and Human Services NIH publication No. 03-5209, 2003. http://www.nhlbi.nih.gov/health/prof/sleep/res_plan/index.html. Accessed Dec. 9, 2011.
3. Collop NA, Obstructive sleep apnea syndromes. *Semin Respir Crit Care Med* 26(1):13-24, 2005.
4. American Academy of Sleep Medicine, The international classification of sleep disorders: diagnostic & coding manual, second ed., Westchester, Ill., American Academy of Sleep Medicine, 2005.
5. Ohayon MM, Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev* 6(2):97-111, 2002.
6. Ohayon MM, Hong SC, Prevalence of insomnia and associated factor in South Korea. *J Psychosom Res* 53:593-600, 2002.
7. Regestein QR, Monk TH, Delayed sleep phase syndrome: A review of its clinical aspects. *Am J Psychiatry* 152:602-8, 1995.
8. Montplaisir J, Allen RP, et al, Restless legs syndrome and periodic limb movements during sleep, in: Kryger MH, Roth T, Dement WC, eds., Principles and practice of sleep medicine, fourth ed., Philadelphia, Elsevier/Saunders pages 839-52, 2005.
9. Ohayon MM, Prevalence of restless leg syndrome and periodic leg movement disorder in the general population. *J Psychosom Res* 53:547-54, 2002.
10. Stiasny K, Oertel WH, Trenkwalder C, Clinical symptomatology and treatment of restless legs syndrome and periodic limb movement disorder. *Sleep Med Rev* 6:253-65, 2002.
11. Young T, Palta M, et al, The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 328:1230-5, 1993.
12. Jelic S, Le Jemtel TH, Inflammation, oxidative stress, and the vascular endothelium in obstructive sleep apnea. *Trends Cardiovasc Med* 18(7):253-60, 2008.
13. Schröder CM, O'Hara R, Depression and obstructive sleep apnea (OSA). Ann Gen Psychiatry 4:13, 2005. annals-general-psychiatry.com/content/4/1/13. Accessed Dec. 9, 2011.
14. Shochat T, Umphress J, et al, Insomnia in primary care patients. *Sleep* 22(supp 2):S359-65, 1999.
15. Sack RL, Lewy AJ, et al, Circadian rhythm abnormalities in totally blind people: incidence and clinical significance. *J Clin Endocrinol Metabol* 75(1):127-34, 1992.
16. Ram, S, Seirawan H, et al, Prevalence and impact of sleep disorders and sleep habits in the United States. *Sleep Breath* 14(1):63-70, 2010.
17. Philipson, EA, Sleep disorders, in: Murray JF, Nadel JA, eds., Textbook of respiratory medicine, Philadelphia, WB Saunders, (2)1841-60, 1988.
18. Kapur V, Strohl KP, et al, Underdiagnosis of sleep apnea syndrome in U.S. communities. *Sleep Breath* 6(2):49-54, 2002.
19. Young T, Evans L, et al, Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep* 20(9):705-6, 1997.
20. Weaver TE, Grunstein RR, Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *Proc Am Thorac Soc* 5:173-8, 2008.
21. Manski RJ, Moeller JF, Maas WR, Dental services: an analysis of utilization over 20 years. *J Am Dent Assoc* 132(5):655-64, 2001.
22. Orr WC, Stahl ML, et al, Physician education in sleep disorders. *J Med Educ* 55:367-9, 1980.
23. Rosen RC, Rosekind M, et al, Physician education in sleep and sleep disorders: a national survey of U.S. medical schools. *Sleep* 16:249-54, 1993.
24. Rosen R, Mahowald M, et al, The taskforce 2000 survey on medical education in sleep and sleep disorders. *Sleep* 21(3):235-8, 1998.
25. Simmons MS, Pullinger A, Education in sleep disorders in U.S. dental schools DDS programs. Presented at the American Academy of Dental Sleep Medicine, June 5, 2010.
26. Bolden N, Smith CE, Auckley D, Avoiding adverse outcomes in patients with obstructive sleep apnea (OSA): development and implementation of a perioperative OSA protocol. *J Clin Anesth* 21(4):286-93, 2009.
27. Gross JB, Bachenberg KL, et al, Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American Society of Anesthesiologists Task Force on Perioperative Management of patients with obstructive sleep apnea. *Anesthesiology* 104(5):1081-93, 2006.
28. Seet E, Chung F, Obstructive sleep apnea: preoperative assessment. *Anesthesiol Clin* 28(2):199-215. 2010.
29. Hillman DR, Platt PR, Eastwood PR, Anesthesia, sleep, and upper airway collapsibility. *Anesthesiol Clin* 28(3):443-55, 2010.
30. Peppard PE, Young T, et al, Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 284(23):3015-21, 2000.
31. Sturm R, Increases in clinically severe obesity in the United States, 1986-2000. *Arch Intern Med* 163(18):2146-8, 2003. Flegal KM, Carroll MD, et al, Prevalence and trends in obesity among U.S. adults, 1999-2008. *JAMA* 303(3):235-41, 2010.
32. Sturm R, Increases in clinically severe obesity in the United States, 1986-2000. *Arch Intern Med* 163(18):2146-8, 2003.
33. Wadden TA, Sternberg JA, et al, Treatment of obesity by very low caloric diet, behavior therapy and their combination: a five-year perspective. *Int J Obes* 13(suppl 2):39-46, 1989.
34. Dansinger ML, Gleason, JA, et al, Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA* 293(1):43-53, 2005.
35. Greenburg DL, Lettieri CJ, Eliasson AH, Effects of surgical weight loss on measures of obstructive sleep apnea: a meta-analysis. *Am J Med* 122(6):535-42, 2009.
36. Hallowell PT, Stellato TA, et al, Potentially life-threatening sleep apnea is unrecognized without aggressive evaluation. *Am J Surg* 193(3):364-7, 2007.
37. Somers VK, White DP, et al, Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation scientific statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing in