Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.256144.3058 Volume 31, Issue 1.1, JAN. 2025, Supplement Issue
Manuscript ID: ZUMJ-2312-3058
DOI: 10.21608/ZUMJ.2024.256144.3058

REVIEW ARTICLE

Polysomnographic Parameters and Assessment of Cardiovascular Complications in Patients with Sleep Disordered Breathing

Mona Hassan[1*], Adel Hassan Ghoneim[1], Nagwan A. Ismail[1], Walid Ibrahim[1]
1: Chest Department, Faculty of Medicine, Zagazig University, El-Sharkia, Egypt

Corresponding Author*:
Mona Hassan

Email:
monmonty247@gmail.com

Submit Date: 19-12-2023
Revise Date: 01-01-2024
Accept Date: 08-01-2024

ABSTRACT

Sleep-Disordered Breathing (SDB) encompasses primary snoring, obstructive sleep apnea (OSA), central sleep apnea (CSA), and hypoventilation disorders. OSA, prevalent in approximately one billion people globally, is characterized by upper airway obstruction during sleep, leading to recurrent pauses in breathing and associated with factors such as obesity and structural abnormalities. CSA, less frequent but impactful, results from respiratory rhythm generator dysfunction and may be induced by various medical conditions.

Diagnostic methods, primarily polysomnography, play a crucial role in identifying and assessing the severity of sleep apnea. Untreated OSA is associated with a heightened risk of cardiovascular diseases, hypertension, atrial fibrillation, and sudden cardiac death. Additionally, OSA influences intrathoracic pressure and ventricular repolarization, potentially leading to sudden cardiac death. CSA risk factors include an increased mortality rate. Furthermore, SDB, especially OSA, has substantial implications for cardiovascular health, influencing coronary events and pulmonary arterial pressures. Nocturnal hypoxemia, measured by parameters like the Oxygen Desaturation Index, is linked to cardiovascular disease risk, hypertension, coronary artery disease, and stroke. Overall, the impact of SDB on cardiovascular health is multifaceted, involving complex interactions between respiratory disturbances during sleep and various cardiovascular conditions. Addressing sleep-related breathing disorders is crucial for mitigating associated cardiovascular risks and improving overall health.

This review aims to explore the link between sleep-disordered breathing (SDB), including obstructive and central sleep apnea, and cardiovascular complications. It emphasizes the role of polysomnography as a diagnostic tool in assessing the severity of sleep apnea and its cardiovascular implications.

Keywords: Sleep-Disordered Breathing; sleep apnea; central sleep apnea;Polysomnography.

INTRODUCTION

Sleep disordered breathing (SDB) affects 2–4% of the world's population, mostly obese. The rising incidence of sleep apnea and hypopnea has highlighted its mortality-raising effects. Even modest recurring sleep apnea or hypopnea burden the cardiovascular system, increasing the risk of hypertension, heart failure, coronary heart disease, arrhythmias, and stroke [1].

The "gold standard" for identifying sleep disordered breathing is polysomnography. This diagnosis requires an overnight sleep lab stay to examine several parameters. Measurements include oxygen saturation, heart rate, electroencephalography sleep stage, nasal and oral airflow, jaw muscle tone, sleep posture, and chest and abdominal movement. Comprehensive examination of these parameters identifies sleep-disordered breathing type and severity. Nighttime electrocardiography (ECG) during sleep apnea examinations may identify myocardial ischemia together with respiratory events, providing novel cardiovascular health insights [2].

The review aims to explore the complex relationship between sleep-disordered breathing (SDB), particularly obstructive sleep apnea (OSA) and central sleep apnea (CSA), and cardiovascular complications. It delves into how these sleep

disorders are diagnosed using polysomnography and discusses the potential cardiovascular risks associated with untreated SDB. The specific research objective is to assess the impact of polysomnographic parameters on the evaluation and prediction of cardiovascular complications in patients with SDB, thus providing a comprehensive view of the interplay between sleep pathology and cardiovascular health.

Sleep Disordered Breathing

Sleep-disordered breathing (SDB) encompasses various conditions like primary snoring (PS), obstructive sleep apnea (OSA), central sleep apnea (CSA), and hypoventilation syndromes. Primary snoring is characterized by snoring without oxygen desaturation or sleep arousal. Sleep apnea involves airflow cessation or significant reduction during sleep and includes OSA, CSA, and mixed disease. These SDB forms entail physiological disruptions, including intermittent hypoxemia, hypercapnia, sleep arousal, and catecholamine surges, contributing to downstream consequences like cardiovascular disease. Hypoventilation syndromes, worsened during sleep due to reduced ventilatory responsiveness, are incorporated into SDB classifications [3]. We observed limited comparative studies on the different types of sleep disordered breathing and their varying impacts on cardiovascular health.

Obstructive Sleep Apnea

Obstructive sleep-disordered breathing (SDB) is a condition when there is a problem with the upper airway during sleep. This leads to symptoms such as snoring and greater effort to breathe, caused by a higher resistance in the upper airway and a tendency for the throat to collapse. Obstructive Sleep Apnea (OSA), commonly referred to as obstructive sleep apnea-hypopnea, is characterized by the halt or substantial decrease of airflow accompanied by breathing effort [4, 5].

Obstructive Sleep Apnea is a condition in which the upper airway becomes more prone to collapsing during sleep, leading to a significant decrease (hypopnea) or complete absence (apnea) of airflow via the nose and/or mouth. This often results in a decrease in the amount of oxygen bound to hemoglobin, which is then followed by a short-lived period of awakening. Recurrent bouts of apnea lead to a continuous drop in the level of oxyhemoglobin saturation, disruption of sleep patterns, and a decrease in both slow-wave and rapid eye movement (REM) sleep [6].

Etiology

Obesity causes 58% of moderate-to-severe sleep apnea. Structural, nonstructural, and genetic factors induce OSA [7, 8].

Craniofacial bone anatomy predisposes persons to OSA by triggering pharyngeal collapse during sleep. Retrognathia, micrognathia, maxillo-mandibular hypoplasia, adenotonsillar hypertrophy (particularly in teenagers and young individuals), and a high, arching palate in women. Obesity, central fat distribution, male sex, age, postmenopause, alcohol, sedatives, and smoking are nonstructural OSA risk factors [9, 10]. Acromegaly, stroke, and hypothyroidism may induce OSA.

Smoking and alcohol use are connected to sleep apnea, although the evidence is limited, particularly in women. Drinking before bedtime promotes sleep apnea in males, whereas smoking induces snoring in women. In Japanese women, consuming more than 23 g of alcohol per day induces oxygen desaturation and snoring [11, 12].

Epidemiology

Nearly one billion people worldwide suffer with OSA, which stresses individuals and society. OSAS prevalence varies from 0.1% to 13%, with most studies reporting rates between 1% and 4%. An AHI of 15 or more indicates moderate to severe OSA in 50% and 25% of middle-aged men and women, respectively [13]. Over the last 30 years, obesity has soared, increasing OSA prevalence [14].

Pathophysiology

Normal sleep relaxes upper airway dilator muscles and narrows the airway. Without sleep disruptions, this physiological state is symptomless. Upper airway constriction from sleep disorders may produce airflow turbulence and recurrent pharyngeal obstruction. Despite increased breathing, obstructive hypopneas and apneas disrupt sleep. Neuromuscular activity in the Upper Airway (UA) decreases during sleep in OSA patients, particularly those at risk of structural collapse, where decreasing ventilatory motor output blocks the UA [15].

During airway restriction, the Bernoulli effect increases airflow velocity, affecting OSA pathogenesis. Airway collapse at transmural closure pressure may result from lower lateral wall pressure with higher airflow velocity. Obese persons with neck fat are more likely to develop OSA, hence this effect is crucial. OSA induces vascular dysfunction and hypertension, affecting cardiovascular health [16].

Common signs

According to [17, 18], obstructive sleep-disordered breathing (SDB) causes loud snoring, respiratory stoppage, disturbed sleep, increased nightly urination, and mouth breathing at night. OSA is characterized by sleep apneas. [19, 20] found daytime symptoms like nonrestorative

sleep, morning headache, dry or sore throat, excessive daytime sleepiness (EDS) during quiet activities, fatigue, cognitive deficits affecting memory and intellectual functioning, and sexual dysfunction like impotence and decreased libido

Snoring alone predicts sleep-disordered breathing (71% sensitivity). According to [17-20], disruptive snoring and observed apneas enhance SDB detection specificity to 94%.

- **Evaluation**

Sleep apnea diagnosis requires sleep tests, usually performed using polysomnography, either in a laboratory or ambulatory environment, to assess sleep and cardiorespiratory variables. During the examination of polysomnography, respiratory events are evaluated and an apnea is defined as a decrease in airflow by over 90% for at least 10 seconds. In addition, obstructive hypopneas are defined by a minimum 30% reduction in airflow, accompanied by a fall in arterial oxygen saturation of at least 3% and/or a microarousal [21].

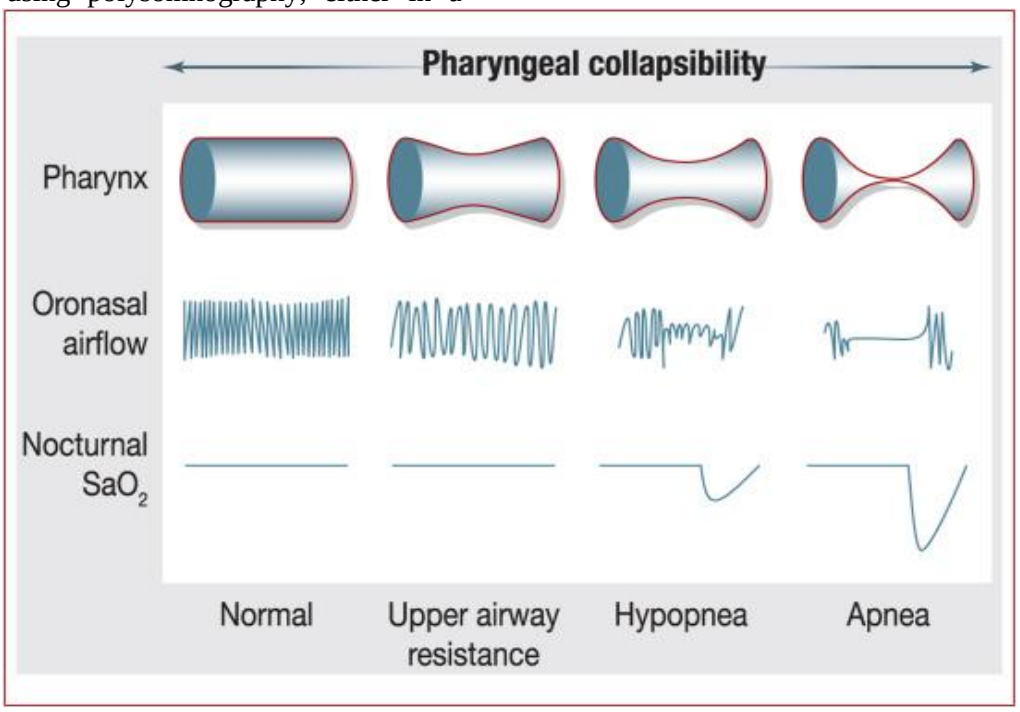

Fig. (1): Scheme of severity of obstructive sleep apnoea respiratory events dependent on pharyngeal collapsus. Disturbances of oronasal airflow are correlated with the degree of pharyngeal obstruction. In severe cases of pharyngeal collapsus, oronasal flow is stopped. The degree of obstruction and oronasal airflow reduction are subsequently correlated with a higher decrease in nocturnal oxygen saturation (SaO_2) [22].

For adult OSA patients, the American Academy of Sleep Medicine recommends polysomnography as the standard diagnostic test. A comprehensive sleep examination that addresses "at-risk populations"' symptoms and co-morbidities led to this advice [23]. Note that questionnaires and prediction algorithms are not recommended for adult OSA diagnosis.

Home respiratory polygraphy sleep apnea testing is recommended for uncomplicated adult patients with a high pre-test chance of moderate-to-severe OSA. If a single home sleep apnea test is negative or unclear, polysomnography is used to diagnose OSA. Literatures needs more exploration on the genetic factors contributing to OSA and its progression into severe cardiovascular conditions.

Central Sleep Disorder

CSA is pontomedullary respiratory rhythm generator dysfunction. Sleep breathing abnormalities. Disorder causes apnea, hypopnea, and hyperpnea sleep cycles. Production and induced hypocapnic central apnea constricts retropalatal upper airways without effort [24].

OSA is more common than CSA, yet both have symptoms. Clinical and polysomnographic criteria classify CSA in the International Classification of Sleep Disorders - Third Edition. CSA may be primary, with Cheyne-Stokes Breathing (CSB) from recurrent high-altitude breathing, drug-induced, or treatment-emergent [25].

Hypo and hyperventilation may produce central apneas in distinct ways. Alveolar ventilation classifies CSA. Heart failure patients may have hyperventilation-related central sleep apnea (CSA) while awake, whereas neuromuscular illnesses, opioid misuse, cervical spinal cord injury, and structural abnormalities including kyphoscoliosis can cause hypoventilation .

Causes

Central respiratory instability during sleep may cause central sleep apnea in patients with various medical problems. Atrial fibrillation (AF), heart failure (HF) with preserved or decreased EF, ischemic stroke, spinal cord injury, renal failure, and chronic opioid usage might temporarily impair respiratory output, producing central apnea. CSA is common in cardiovascular disease and risky. The etiology of idiopathic CSA is unknown [26].

Epidemiology

Age promotes central sleep apnea (CSA), especially in 65-year-olds. Central sleep apnea was found in 2.7% of 65-year-old males in a cross-sectional investigation utilizing ICSD-3. Chemical sensitivity may make older adults more susceptible to central apnea, particularly during NREM sleep [27]. CSA is less common in women and requires higher hypocapnia to induce central apnea [28].

The diagnosis

Central sleep apnea (CSA) disrupts sleep like other types. Their symptoms include poor sleep quality, overnight awakenings, sleep disruption, excessive daytime somnolence (EDS), morning headaches, exhaustion, and impaired attention span. Contrary to OSA, CSA does not cause snoring. OSA and CSA are separate illnesses, yet they may coexist and cause symptoms. [24]. discovered CSA patients are less fat than OSA patients.

Patients with hypercapnic central apnea may have additional symptoms. Heart failure (HF) patients may not have daytime symptoms while drowsy. The daytime sympathetic activity that alerts and lowers tiredness may help heart failure patients sleep. Heart failure mortality decreases with subjective daytime weariness [29]. Even without substantial daytime sleepiness, elderly HF patients who feel tired should be evaluated for sleep apnea. Early CSA diagnosis requires gold-standard procedures such nocturnal polysomnography (PSG) [30]. Self-reported symptoms may not suffice.

The STOP-BANG, Berlin, and Epworth Sleepiness Scales are used to assess sleep-disordered breathing (SDB) subjectively. The STOP-BANG questionnaire, which contains eight factors including snoring, daytime weariness, and BMI, is widely used but not recommended for diagnosis due to its poor specificity. It varies on OSA severity [31-33].

The 11-question Berlin questionnaire defines OSA risk by snoring, sleep disruption, daily fatigue, high blood pressure, and BMI. Television and passenger trips evaluate Epworth Sleepiness Scale daytime fatigue. Despite outmoded questions and coffee sensitivity, the ESS and Berlin questionnaires have lower specificity than the STOP-BANG questionnaire. After therapy, many normal ESS score patients improve dramatically [34-38].

Assessing sleep.

Main sleep testing techniques include polysomnography (PSG) and home sleep apnea testing (HSAT) (Scalzitti et al., 2017). Sleep lab polysomnography (PSG) is more accurate. Home Sleep Apnea Testing (HSAT) is cheaper and easier for healthy people. EEG does not monitor sleep, hence HSAT may underestimate the disorder's severity, particularly in insomnia. After a negative or inconclusive HSAT, polysomnography (PSG) is indicated to confirm sleep-disordered breathing [39].

PSG is recommended for cardiac, neurological, neuromuscular, and hypoventilation syndrome patients. HSAT is also being tested for sleep-disordered breathing in children and adolescents. HSATs measure airflow, chest wall expansion, and pulse oximetry. WATCH-PAT and other models compute the AHI using sympathetic activity indices [40].

Polysomnography

Nocturnal polysomnography (PSG) is used in sleep labs to identify respiratory issues, narcolepsy, movement disorders, and parasomnias. This test detects sleep-disordered breathing and other issues by monitoring the patient's physiological changes throughout sleep. Obstructive sleep apnea (OSA) diagnostic tests may be done at home without a professional in certain cases, although this is limited and does not replace PSG for other sleep disorders [40].

Technique

Insomnia and sleep fragmentation may result from caffeine before polysomnography (PSG). PSG patients should avoid caffeine in the afternoon and evening before the test. Before a sleep lab visit, caffeine is dangerous and impracticable [41].

OSA sufferers should take their medications, including sleep aids, on PSG night. For optimum result interpretation, the technician should record these prescriptions. Sleep drug zolpidem may improve sleep quality during PSG without aggravating sleep apnea in persons with severe insomnia or lab anxiety. Sedative users should take them in the lab and leave carefully [42, 43].

After extended-release zolpidem, the FDA advises avoiding driving for 24 hours. PSG for non-sleep-related respiratory issues may need different medication. Narcolepsy patients with PSG and MSLT may need to cease stimulants and other

psychoactive substances for accurate test findings. Before sleep testing, discuss medication discontinuation with the prescribing doctor and mention it in PSG/MSLT findings if clinically impossible for patient safety. Anxious sleep study participants may benefit from a pre-study laboratory tour and a family member or friend during setup. Comfortable cushions are recommended [44].

Protocols

General concerns

While patients sleep, polysomnography (PSG) involves several monitoring equipment. The sleeping chamber also contains an infrared camera and audio system for remote surveillance and communication. The technologist measures heart rate, respiration, oxygen saturation, snoring location, volume, and posture. In CPAP titration tests, researchers track masks, air pressure, and leaks [45].

Digitally collecting physiological parameters lets professionals assess sleep, wakefulness, and physiological events. Computers arrange and integrate video and polysomnographic records. Digital video synchronization identifies parasomnias and movement irregularities. It tracks atypical sleep patterns for inquiry. Time and sleep state are provided [46].

Split-night protocol

For severe clinical OSA suspicion, split-night testing is becoming more common. These investigations diagnose OSA and create positive airway pressure to prevent upper airway collapse. The AASM allows a split-night study instead of a full-night diagnostic polysomnography (PSG) followed by a night of positive airway pressure titration [47, 48].

A split-night study requires an apnea-hypopnea index (AHI) of 40 or more episodes per hour of sleep, recorded for at least 2 hours, or 20 to 39 occurrences per hour with significant OSA evidence. Changing positive airway pressure takes at least 3 hours. Watch and record REM and NREM sleep for considerable breathing improvements, especially on the back. If the foregoing conditions are not met, a second full-night polysomnography (PSG) is recommended for titration. Partial titration data may suggest auto-titrating CPAP device. Separate-night trials save money, eliminate scheduling delays, and ensure patient compliance [47, 49].

Indications

Adults use polysomnography (PSG) to diagnose OSA, manage positive airway pressure, and assess treatment success. Advanced cardiac patients and those suspected of having concurrent sleep disorders need PSG, particularly for central apneas [41].

Full-night and split-night PSG scans change OSA patients' positive airway pressure. During the PSG technique, CPAP is modified if breakthrough obstructive sleep apnea is observed. This method may also evaluate treatment-induced central sleep apnea and track OSA patients who use oral appliances or have surgery [43].

Cheyne-Stokes breathing, central apnea, COPD/OSA, and hypoventilation are diagnosed by PSG. It diagnoses parasomnias, REM sleep behavior disorder, and daytime drowsiness. Polysomnography (PSG) with prolonged EEG monitoring evaluates nocturnal seizures [40].

Contraindications

Polysomnography (PSG) without contraindications is safe. For optimal testing circumstances, numerous factors must be addressed. Testing requires medically stable people since medical treatment may be delayed. Get spinal cord injury patients ready for the sleep lab. Active respiratory infections, severe coughing, or high-dose opioid-treated acute pain need rescheduled visits. Testing may be delayed by lice or bed bugs. Regular opioid use and calm breathing seldom hinder testing. PSG may be possible with pacemakers, defibrillators, and left ventricular assist devices, but check the sleep lab. Consider the advantages and downsides of sending hospitalized patients to sleep laboratories. Some sleep tests are portable [43].

Complications

Polysomnography has little side effects. Electrode sticky cutaneous irritation is most prevalent. Cons include lab sleep issues, changing settings, monitoring devices, and bed discomfort. To prepare for medical crises during testing, labs need age-appropriate resuscitation equipment, well-trained personnel, and a defined emergency medical care system [41].

Variables The American Academy of Sleep Medicine (AASM) defines polysomnography (PSG) as a physiological measure of sleep [50]. EEG, EOG, and EMG control sleep. Submental EMGs identify hypotonia during REM sleep, whereas EEGs are collected from many brain locations. Esophageal manometry, RIP, and diaphragmatic/intercostal electromyography assess respiration. To overcome mouth breathing restrictions, nasal prongs and thermistors measure airflow. In pediatric research, end-tidal CO2 monitors detect hypoventilation, whereas neck microphones detect snoring. Electrocardiography detects arrhythmias using a modified lead II, whereas pulse oximetry measures oxygen saturation [51].

As indicated by the AASM, Patil et al. [52] recommend additional physiological measures during PSG. Monitor supine, left lateral, right lateral, and prone postures, particularly for individuals with position-specific anomalies. A sensor or video monitor tracks bed elevation and position. Electromyography (EMG) assesses limb kinematics by measuring anterior tibialis muscle activation in both legs. Two-way video monitoring with an infrared sensor enhances PSG patient supervision [53].

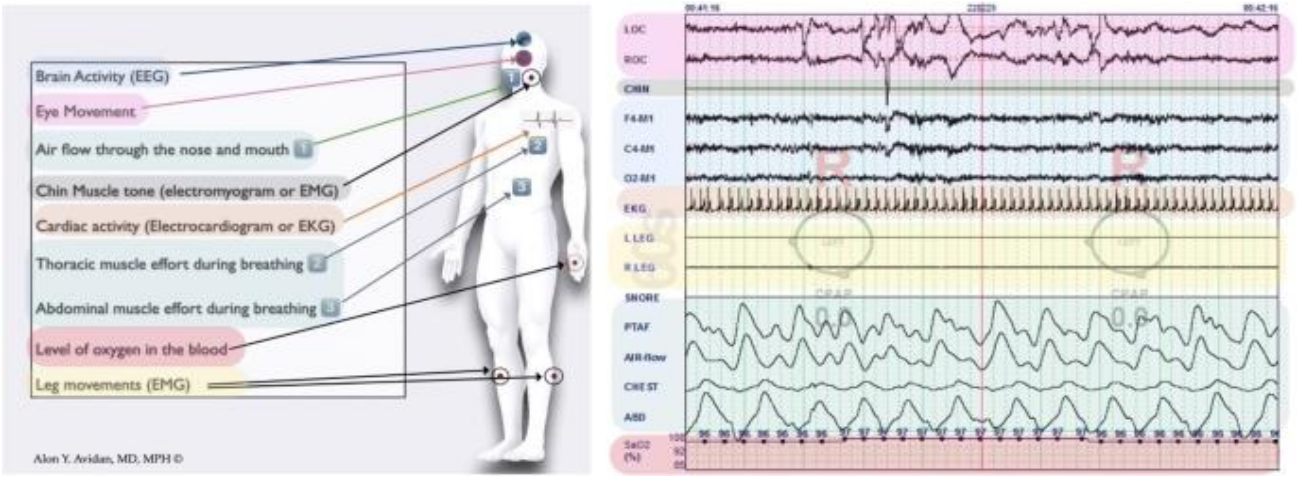

Fig. (2): A typical PSG setup includes EEG to measure brain activity, EOG to measure eye movements for sleep staging, EMG to measure chin and limb muscle tone, EKG to measure cardiac activity, respiratory channels to show airflow and effort, and pulse oximetry. Respiratory and pulse oximetry channels can diagnose sleep-related breathing issues [54].

Information Derived

Since PSG abnormalities typically indicate a diagnosis, sleep disorder professionals must analyze the substantial data from polysomnography (PSG). Sleep reports include several elements from the night [55, 56].

Attending PSG measures total recording time, total sleep time (TST), sleep efficiency (SE), sleep stage percentage (SSP), sleep latency, arousals, apneas, hypopneas, RERAs, Cheyne-Stokes breathing, and various indices that measure abnormal respiratory events during sleep. (Ingram et al., 2019). A microphone attached to the neck helps PSG determine a snoring index by measuring the amount and intensity of snoring. Body posture may indicate whether some events are position-dependent. Oxyhemoglobin saturation, limb movements, EEG abnormalities, and seizure activity are also assessed to determine sleep characteristics [57].

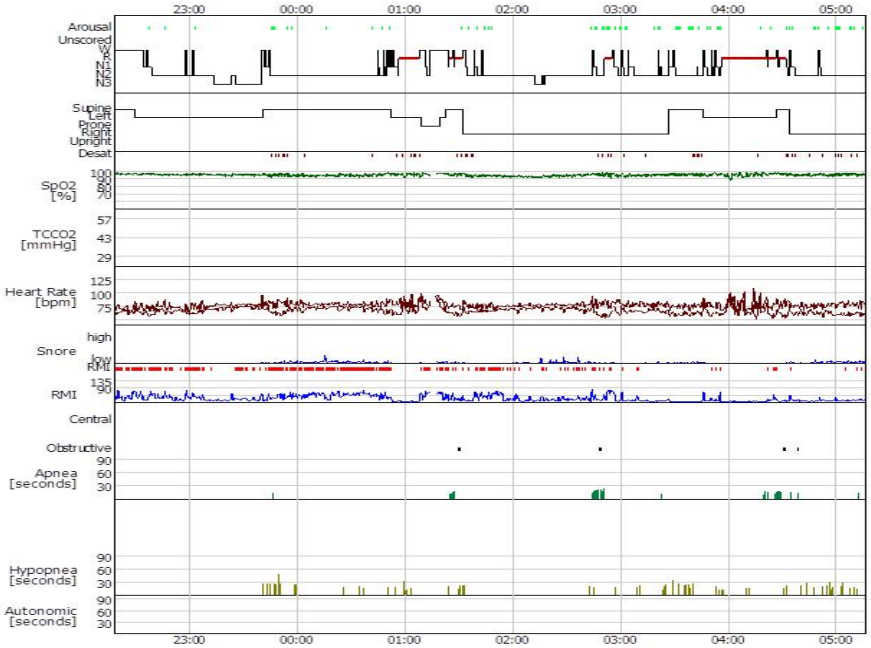

Fig (3): Visual summary of polysomnographic data. Graphical representation of an entire polysomnogram, with the beginning of data collection at left and the end of the study at right. The top line shows the variation in sleep stages through the night; directly below is a notation of

body position through the night, followed by oxyhemoglobin saturation, transcutaneous carbon dioxide levels (not measured in this particular study), and maximum/minimum heart rate. At the bottom are summary signals for respiratory events, including respiratory mechanics instability (RMI; a measurement of thoracoabdominal dyssynchrony), and events that have been scored as apneas or hypopneas [58].

BPM: beats per minute; RMI: respiratory mechanics instability.

PSG Typical OSA findings

To diagnose and assess OSA therapy, polysomnography (PSG) must last at least six hours [19]. PSG shows apneic episodes with respiratory muscle exercise, clinically meaningful apneic episodes lasting 10 seconds or more, and a combination of apneas and hypopneas or one of them solely suggesting OSA. Mixed apneas may occur.

OSA diagnosis relies on the AHI, which is derived by dividing apneas and hypopneas by sleep duration. Cutoff criteria define OSA severity in AHI standards. Mild OSA comprises 5-15 episodes per hour, moderate 15-30, and severe 30+. Drowsiness throughout the day is another indicator of sleep apnea that needs treatment. Figure 4 displays a severe obstructive sleep apnea (OSA) patient's polysomnogram (PSG) with frequent and cyclic airflow restriction and oxygen saturation drop.

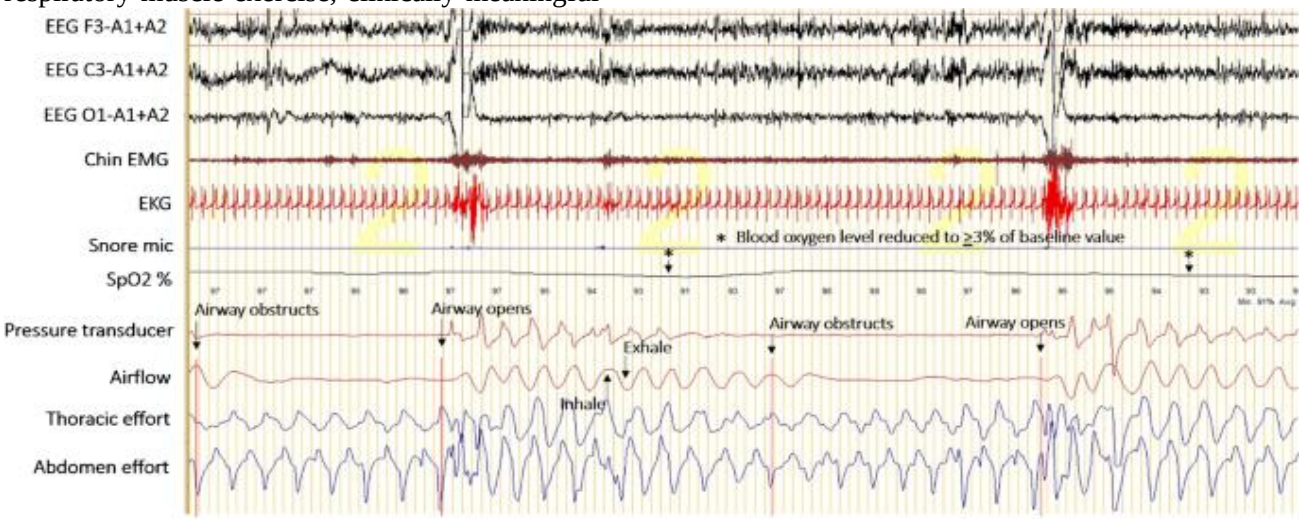

Fig. (4): An example of polysomnography for OSA. PSG demonstrates two obstructive apneic episodes with decreased airflow despite persistent respiratory effort, resulting in considerable oxygen saturation reductions during N2 sleep. Watch the airway occlusion and opening, which delays blood oxygen levels. EEG F3-A1+A2 for the left frontal area in referential montage, EEG C3-A1+A2 for the left central region, and EEG O1-A1+A2 for the left occipital region comprised the PSG epochs. Chin EMG is chin electromyography, EKG is electrocardiogram, snore mic is a snoring sensor or microphone, SpO2% is oxygen saturation, pressure transducer measures nasal airflow, thoracic effort is chest effort, and abdomen effort is abdominal effort [54].

Further research could be directed towards enhancing the predictive accuracy of polysomnography for cardiovascular complications.

Characteristic PSG results of CSA

Characteristic CSA PSG results

The American Academy of Sleep Medicine (AASM) developed the ICSD-3 Central Sleep Apnea diagnostic criteria, according to Sateia et al. [25]. Berry et al. [59] showed CSA-type-specific characteristics. Frequent central apneas on polysomnography (PSG) are required to diagnose CSA. You must rule out other diagnoses. While sleeping, central apnea stops airflow for 10 seconds without effort.

The main central sleep apnea (CSA) is diagnosed by polysomnography (PSG) demonstrating at least 5 central apneas and/or hypopneas per hour of sleep, accounting for more than 50% of the total respiratory events in the apnea-hypo You must also have a sleep complaint such severe exhaustion, difficulties falling asleep, waking up suddenly owing to breathing issues, loud snoring, or interrupted breathing [24].

Main CSA requires three or more central apneas or hypopneas followed by a 40-second crescendo-decrescendo respiratory rhythm.

Treatment-emergent central apnea requires early detection of Obstructive Sleep Apnea (OSA), defined as ≥5 obstructive respiratory events per hour of sleep. A PAP titration trial should eliminate obstructive apnea and reveal central

sleep apnea (CSA) without further problems or medications [24].

Cardiovascular pathophysiologies may result from nighttime breathing difficulties, such as sleep loss, periodic hypoxia, and chest pressure fluctuations. Death, atrial fibrillation, cerebral infarction, and heart failure from coronary artery disease are examples. In 2021, Cowie et al. [60] found that CPAP for Sleep Disordered Breathing (SDB) may lower systemic blood pressure and improve endothelial function.

Systemic inflammation, oxidative stress, and sympathetic nervous system stimulation cause endothelial dysfunction and SDB. These symptoms result from occasional hypoxia, sleep loss, and arousals. OSA intermittent hypoxia may cause atherosclerosis and inflammation. Endothelial dysfunction and arterial stiffness affect SDB severity, as seen by flow-mediated dilatation and cardio-ankle vascular index. Oxidative stress and inflammation thicken cerebral artery intima-media in SDB. Sleep-disordered breathing hypoxia and blood vessel intima-media thickness promote oxidative stress and inflammation [61].

OSA patients had thicker intima-media without arteriosclerosis risk factors. Nighttime hypoxia is based on intima-media thickness. Research suggests that doubling the Apnea-Hypopnea Index (AHI) elevates coronary artery calcium. It rose 19% for men under 65 and 17% for women of all ages. SDB is highly linked to Gensini score-measured coronary atherosclerotic load and increased troponin T, indicating silent myocardial ischemia and mild damage. Even in stable coronary artery disease patients, [62, 63].

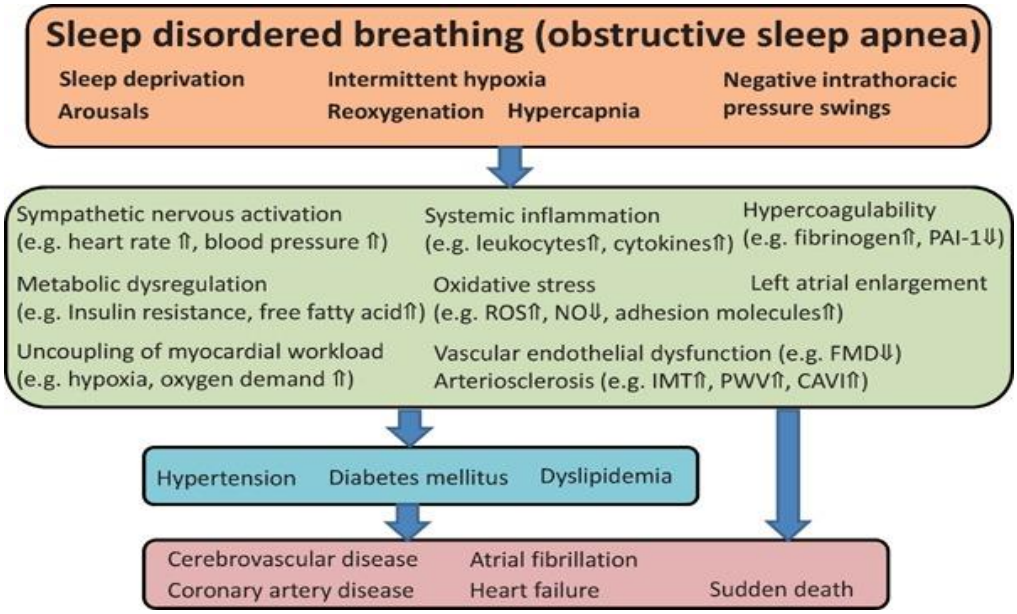

Fig. (5): Pathophysiology of the impact of sleep disordered breathing, OSA, on cardiovascular disease [64].

PAI-1 is plasminogen activator inhibitor. Reactive oxygen species (ROS). NO is nitric oxide. FMD is flow-mediated dilatation. IMT is intima-media thickness. PWV is pulse wave velocity. CAVI is cardio-ankle vascular index.

Mortality

Multi-Ethnic Study of Atherosclerosis included almost 5,000 healthy participants. Professionally diagnosed Obstructive Sleep Apnea (OSA) patients had a 2.4 times increased risk of death and CVD after 7.5 years. The relationships were higher in individuals with an Apnea-Hypopnea Index (AHI) greater than 30, whereas milder OSA patients showed lower or inconsistent correlations [65]. Males with untreated severe OSA had a 2.9-fold higher risk of fatal cardiovascular disease (CVD) events than those with mild or moderate OSA. According to Campos-Rodriguez et al. (2014), women with severe untreated OSA had a 3.5-fold greater death risk than controls. Patients with severe OSA had a three-fold greater risk of all-cause death. This group showed higher CVD mortality estimations [60].

Hypertension

Up to 50% of OSA patients have hypertension, and 30% have both. Over four years, untreated OSA doubled or tripled hypertension risk. Undiagnosed OSA, poorly managed hypertension, and accompanying comorbidities are common in African Americans, who have resistant hypertension [66]. A recent thorough review and meta-analysis linked essential hypertension to mild, moderate, and severe OSA (OR=1.18, 1.32, and 1.56, respectively) [67].

For OSA patients without significant sleepiness, continuous positive airway pressure (CPAP) did

not reduce hypertension or cardiovascular disease events during a median follow-up of 4 years [68].

Cardiac arrhythmia

OSA-related arrhythmias have a complex and shifting substrate. It involves structural remodeling and transitory electrical changes caused by apnea, is summarized in Figure 6 [69].

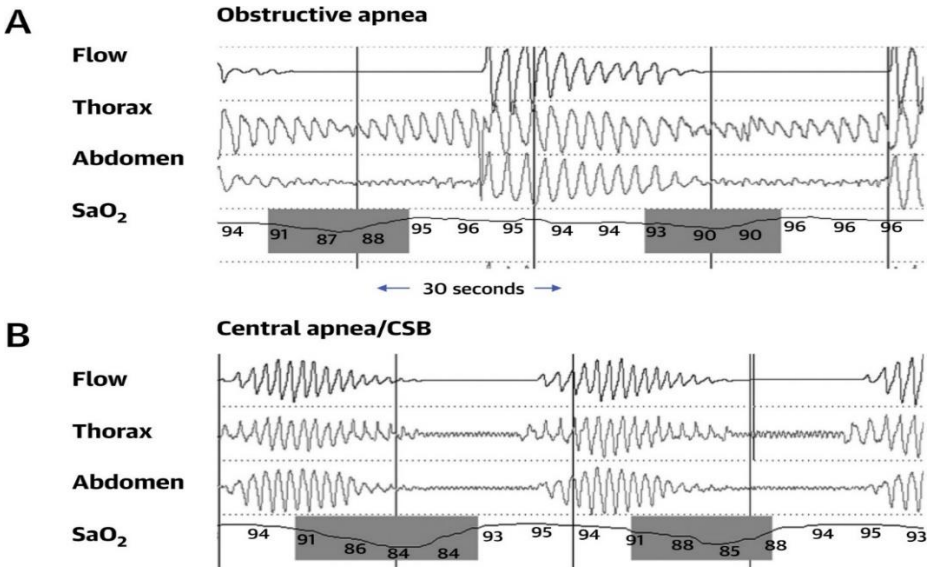

Fig. (6): Features of Home Sleep Apnea Testing Traces (A) Patient diagnosed with obstructive sleep apnea (OSA); (B) a patient diagnosed with central sleep apnea (CSA), displaying airflow, thoracic and abdominal wall movements, and PaO2 (arterial oxygen partial pressure). It is important to note that desaturation is delayed in central sleep apnea (CSA) because to the prolonged circulation time that occurs in individuals with heart failure. [60].

Atrial fibrillation

Obstructive Sleep Apnea (OSA) causes atria structural changes and conduction abnormalities, which increase the risk of atrial fibrillation. OSA may cause atrial fibrillation (AF) triggers, particularly in the pulmonary veins, despite the atria's persistent refractoriness. Obstructive respiratory episodes may also generate brief electrical arrhythmogenic changes, which may raise the risk of nocturnal AF paroxysms [70].

The VARIOSA-AF study demonstrated a significant 2.3-fold higher likelihood of experiencing at least one hour of atrial fibrillation (AF) on days with more severe sleep apnea compared to nights with better sleep quality [71, 72].

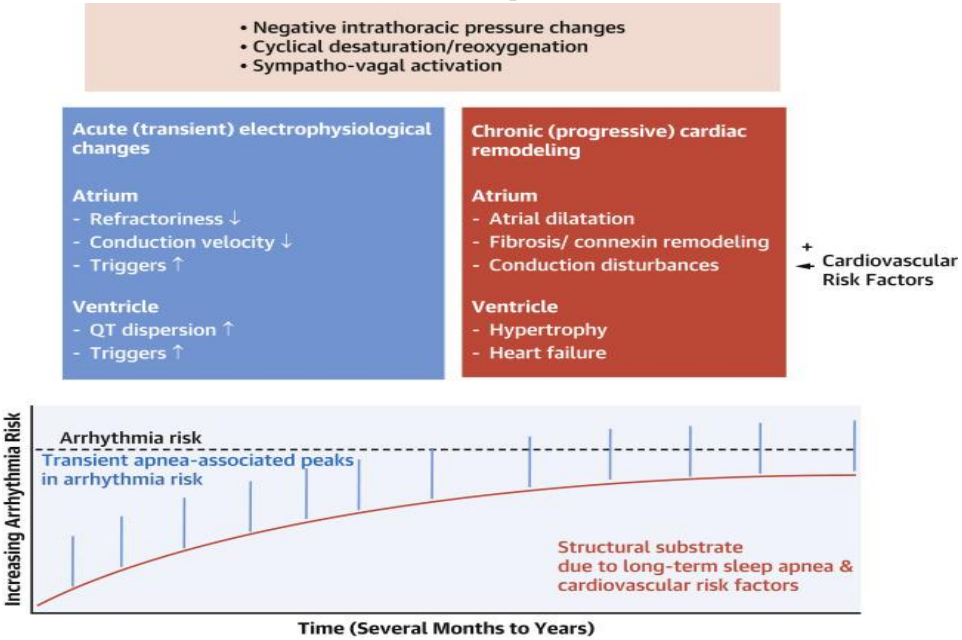

Fig. (7): The intricate and ever-changing foundation for arrhythmia caused by sleep apnea. (First) Sleep apnea leads to acute and temporary alterations in electrophysiology (blue box) and chronic and gradual remodeling of the heart (red box). The lowest part or position. Each instance of

acute sleep apnea results in temporary increases in the risk of arrhythmia (shown by blue lines), but if there is no underlying structural issue, the essential level to trigger an arrhythmia (represented by the dashed black line) cannot be achieved. Nevertheless, when there is structural remodeling caused by chronic sleep apnea and cardiovascular risk factors (shown by the red line), sudden bouts of sleep apnea might provoke arrhythmia [60].

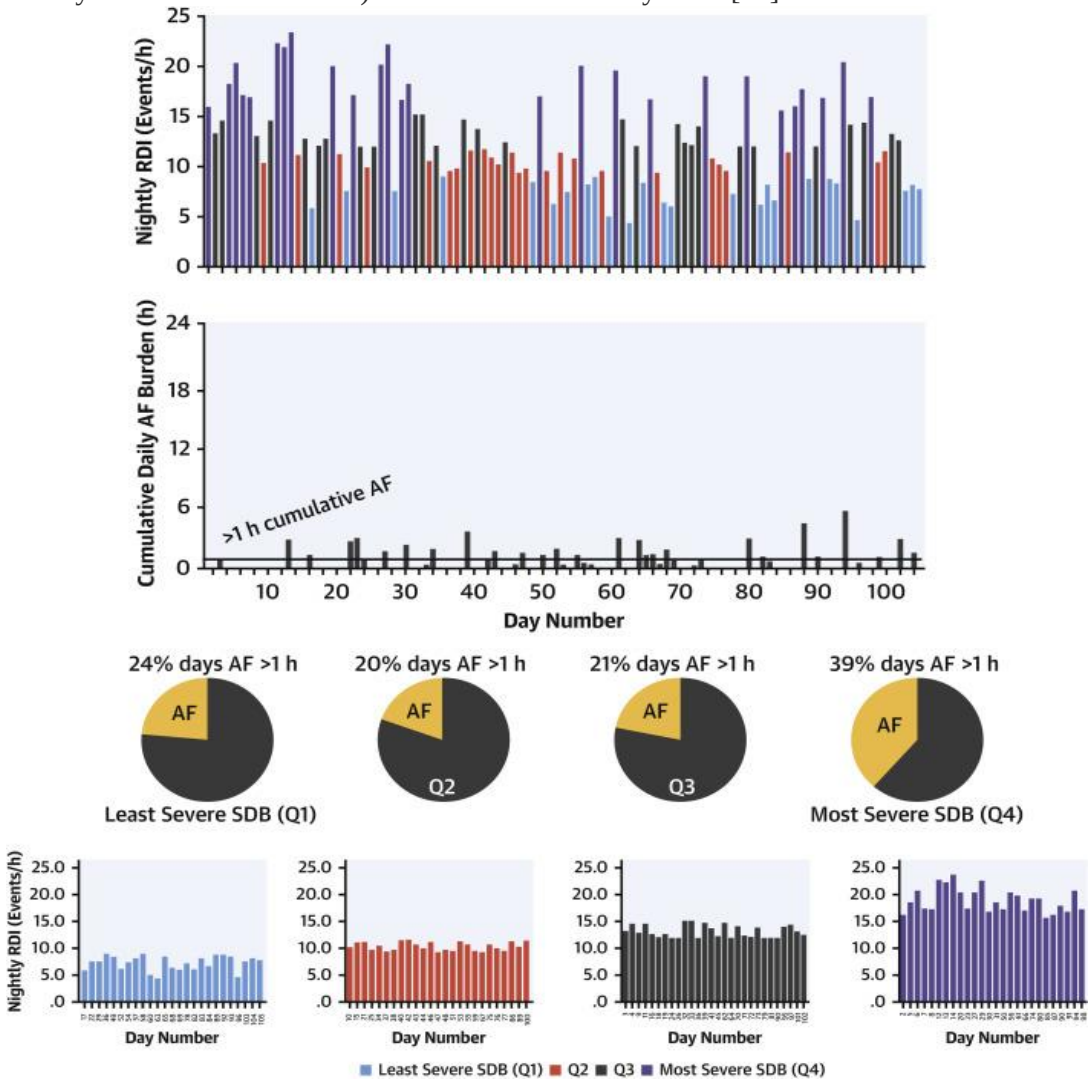

Fig. (8): Continuous sleep apnea and atrial fibrillation variations. Pacemaker patients exhibited nightly sleep apnea severity variations. Greater respiratory disturbance index (RDI) was connected to higher risk of atrial fibrillation (AF) on the same day [72].

AF patients had a higher rate of Sleep-Disordered Breathing (SDB) (21%–74%) than non-AF patients (3%–49%). OSA lowers antiarrhythmic medication response, and patients with OSA had a 31% greater probability of AF recurrence following pulmonary vein isolation. After cardioversion and pulmonary vein isolation, CPAP lowers AF recurrences. Behavioral changes to decrease weight, bariatric surgery, and abstaining from alcohol may help manage OSA and maintain a normal heart rhythm. HF produces respiratory instability owing to high pulmonary vascular pressure, increased breathing, and low carbon dioxide. This may explain the AF-CSB relationship. Idiopathic CSB and AF may result from autonomic dysfunction [73, 74]

Ventricular arrhythmia

OSA affects ventricular repolarization via intrathoracic pressure. Diseases and sympathetic nervous system activity may promote sudden cardiac death. In nearly 10,000 polysomnography patients, an AHI of 20/h independently predicted sudden cardiac death. MVA risk increases with HF and sleep apnea. Sleeping with severe OSA increases the risk of ventricular premature beats, nonsustained tachycardia, and sudden cardiac death. Registry evidence suggests that servoassisted breathing may reduce the requirement for implanted cardioverter-defibrillators in Central Sleep Apnea patients. A big randomized study found that this medication increased CVD and all-cause mortality [75, 76].

Coronary disease

Coronary events are linked to OSA. After risk factor adjustment, OSA quadrupled CVD events or fatalities in a 1,400-person sample [75, 77]. In 40% of ST-segment elevation MI patients, severe OSA went undiagnosed [77]. Undiagnosed hospitalised MI patients were more likely to develop OSA at night, suggesting acute stress [75, 77].

In a 4-year post-MI follow-up, nocturnal hypoxemia severity and EDS independently predicted MACE (Konishi et al., 2019). OSA promotes arterial stiffness, early atherosclerosis, coronary calcification, plaque instability, and susceptibility [77].

CAD is influenced by moderate-to-severe OSA regardless of risk. Acute pressor surges, hypoxemia, and adrenergic activation during apnea may induce myocardial ischemia or plaque rupture [78]. Obstructive apnea hypoxemia causes morning chest pain and ST-segment changes [78]. St-segment depression in OSA patients is better predicted by nocturnal oxygen desaturation severity than AHI [78]. OSA enhances MI mortality and CVD risk after coronary intervention [79]. Almost 45% of 1,300 polysomnography patients had an AHI ≥15/h, and OSA independently increased MACE risk [79]. Percutaneous coronary intervention increased OSA patients' MACE risk in the meta-analysis [79]

Heart failure

HFrEF 50%-75% SDB and sustained ejection percentage. 97%–44% have acute decompensated HF. In HFrEF patients, obesity, male sex, AF, age, and poorer left ventricular (LV) systolic function substantially correlated with SDB. HF syndrome and heart dysfunction raise CSA. SDB independently increases mortality [80-82].

Obstructive pulmonary hypertension

Vasoconstriction may raise pulmonary arterial pressures in acute OSA hypoxia. Possible transient pulmonary vascular resistance changes. OSA often coexists with pulmonary or systemic hypertension, although its function is unclear. OSA patients with pulmonary hypertension are at higher mortality risk, making identification crucial. About 20% of OSA patients develop pulmonary hypertension (PAP ≥20 mm Hg) without lung illness [83, 84].

The Apnea-Hypopnea Index does not predict sleep-disordered breathing mortality as well as overnight hypoxemia. Oxygen saturation cycles cause inflammation, oxidative damage, and SNS activity. Azarbarzin et al, Ma et al, and Cao et al. (2023) [85-87] studied polysomnographic characteristics such ODI, sleep duration, blood pressure dipping, T<90% saturation, alertness index, and pulse rate variability

Frequent and severe oxygen desaturation episodes during sleep increase cardiovascular disease risk, according to the Oxygen Desaturation Index. Hypertension, coronary artery disease, and stroke are associated to high ODI and oxygen desaturation [88, 89].

PSG measures total sleep time, essential for cardiovascular health. Poor sleep quality promotes sympathetic activity, inflammation, and endothelial dysfunction [41, 90].

SDB increases the risk of cardiovascular disorders such hypertension, left ventricular hypertrophy, and stroke owing to poor diastolic blood pressure drop during sleep.

Time Below 90% The duration of sleep with oxygen saturation below 90% (T<90%) suggests hypoxemia severity. Without oxygen, sleep promotes oxidative damage, endothelial dysfunction, and sympathetic activity, harming cardiovascular health [91].

Arousal Index: Frequent breathing might decrease sleep architecture, quality, and cardiovascular health. Sleep fragmentation promotes sympathetic activity, inflammation, endothelial dysfunction, and metabolic abnormalities [91, 92].

High PRV SDB patients have autonomic dysfunction and higher cardiovascular mortality. Recurrent apnea and hypopnea may cause alterations in heart rate and sympathetic activity [93-96].

Clinical Importance of PSG Parameters

Over 10,000 patient studies show that AHI alone does not predict mortality, but T<90 is more accurate. Inflammatory mediators increase nocturnal hypoxemia's risk of abrupt cardiac death and mortality. Other sleep-related hypoxemia indicators that predict mortality support oximetry-based risk classification [86, 97-101].

Complete Cardiovascular Complication Assessment

PSG parameters such as ODI, sleep duration, blood pressure decrease, T<90%, arousal index, and pulse rate variability per hour provide a comprehensive cardiovascular assessment in SDB patients. These assessments show SDB severity, hypoxemia, sleep disruption, and cardiovascular health impacts [85, 86, 91, 102].

More detailed studies are needed on the mechanisms through which sleep disordered breathing exacerbates or causes specific cardiovascular diseases.

Limitation in literature

In literature on polysomnographic assessment of sleep disordered breathing (SDB) and its cardiovascular implications, several critical gaps

persist. Firstly, longitudinal studies elucidating the causative correlation between specific SDB types, such as obstructive and central sleep apnea, and distinct cardiovascular pathologies are insufficient. This deficiency hampers the understanding of progression and interaction dynamics. Moreover, there is a lack of comprehensive analysis on the genetic contributors in the etiology of SDB, particularly in its exacerbation of cardiovascular risks. The predictive utility of polysomnography in prognosticating cardiovascular outcomes in SDB patients remains inadequately explored, necessitating further refinement and validation. Additionally, a deeper mechanistic insight into how SDB influences the onset and progression of various cardiovascular diseases is critically needed. These gaps underscore the necessity for advanced, multifaceted research to enhance the understanding and management of SDB-associated cardiovascular risks.

REFERENCES

1. Kadhim, K., Middeldorp, M. E., Elliott, A. D., Agbaedeng, T., Gallagher, C., Malik, V. et al. Prevalence and assessment of sleep-disordered breathing in patients with atrial fibrillation: a systematic review and meta-analysis. Canadian Journal of Cardiology, 2021; 37(11), p. 1846-1856.
2. Ma, Y., Sun, S., Zhang, M., Guo, D., Liu, A. R., Wei, Y. et al. Electrocardiogram-based sleep analysis for sleep apnea screening and diagnosis. Sleep and Breathing, 2020; 24, p. 231-240.
3. Sedky, K., Nazir, R., & Bennett, D. Sleep-Disordered Breathing. Sleep Medicine and Mental Health: A Guide for Psychiatrists and Other Healthcare Professionals, 2020; p. 131-150.
4. Zaibi, H., Safta, B., Dhahri, B., & Aouina, H. Impact of smoking on the severity of Obstructive Sleep Apnea Hypopnea Syndrome. Tunis Med, 2018; 96(8-9), p. 477-482.
5. Stöwhas, A.C., M. Lichtblau, and K.E. Bloch. [Obstructive Sleep Apnea Syndrome]. Praxis (Bern 1994), 2019; 108(2), p. 111-117.
6. Rundo, J.V. Obstructive sleep apnea basics. Cleveland Clinic journal of medicine, 2019; 86(9 Suppl 1), p. 2-9.
7. Awad, M. I., & Kacker, A. Nasal Obstruction Considerations in Sleep Apnea. Otolaryngol Clin North Am, 2018; 51(5), p. 1003-1009.
8. Lin, J., & Suurna, M. Sleep Apnea and Sleep-Disordered Breathing. Otolaryngol Clin North Am,
9. Mitra, A. K., Bhuiyan, A. R., & Jones, E. A. Association and risk factors for obstructive sleep apnea and cardiovascular diseases: a systematic review. Diseases, 2021; 9(4), p. 88.
10. Al-Qattan, H., Al-Omairah, H., Al-Hashash, K., Al-Mutairi, F., Al-Mutairat, M., Al-Ajmi, M. et al. Prevalence, risk factors, and comorbidities of obstructive sleep apnea risk among a working population in Kuwait: a cross-sectional study. Frontiers in neurology, 2021; 12, p. 620799.
11. Hsu, W. Y., Chiu, N. Y., Chang, C. C., Chang, T. G., & Lane, H. Y. The association between cigarette smoking and obstructive sleep apnea. Tobacco induced diseases, 2019; 17.
12. Ioannidou, D., Kalamaras, G., Kotoulas, S. C., & Pataka, A. Smoking and Obstructive Sleep Apnea: Is There An Association between These Cardiometabolic Risk Factors?—Gender Analysis. Medicina, 2021.; 57(11), p. 1137.
13. Benjafield, A. V., Ayas, N. T., Eastwood, P. R., Heinzer, R., Ip, M. S., Morrell, M. J. et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. The Lancet Respiratory Medicine, 2019; 7(8), p. 687-698.
14. Kuvat, N., Tanriverdi, H., & Armutcu, F. The relationship between obstructive sleep apnea syndrome and obesity: A new perspective on the pathogenesis in terms of organ crosstalk. The clinical respiratory journal, 2020; 14(7), p. 595-604.
15. McNicholas, W.T. Obstructive Sleep Apnoea: Focus on Pathophysiology, in Advances in the Diagnosis and Treatment of Sleep Apnea: Filling the Gap Between Physicians and Engineers. Springer. 2022; Springer, p. 31-42.
16. Jyothi, I., Prasad, K. R., Rajalakshmi, R., Kumar, R. S., Ramphanindra, T., Vijayakumar, T. M. et al. Obstructive sleep apnea: A pathophysiology and pharmacotherapy approach, in Noninvasive Ventilation in Medicine-Recent Updates. 2019; IntechOpen.
17. Verbraecken, J. More than sleepiness: Prevalence and relevance of nonclassical symptoms of obstructive sleep apnea. Current Opinion in Pulmonary Medicine, 2022; 28(6), p. 552-558.
18. Patel, S.R. Obstructive sleep apnea. Annals of internal medicine, 2019; 171(11), p. ITC81-ITC96.
19. Slowik, J. M., Sankari, A., & Collen, J. F. Obstructive sleep apnea, in StatPearls [Internet]. 2022; StatPearls Publishing.
20. Lal, C., Weaver, T. E., Bae, C. J., & Strohl, K. P. Excessive daytime sleepiness in obstructive sleep apnea. Mechanisms and clinical management. Annals of the American Thoracic Society, 2021; 18(5), p. 757-768.
21. Raphelson, J., Feldman, E., & Malhotra, A. Obstructive Sleep Apnea: Diagnosis with

Polysomnography and Portable Monitors, in Essentials of Sleep Medicine: A Practical Approach to Patients with Sleep Complaints. 2022; Springer, p. 111-128.
22. Arnaud, C., Bochaton, T., Pépin, J. L., & Belaidi, E. Obstructive sleep apnoea and cardiovascular consequences: Pathophysiological mechanisms. Archives of Cardiovascular Diseases, 2020; 113(5), p. 350-358.
23. Kapur, V. K., Auckley, D. H., Chowdhuri, S., Kuhlmann, D. C., Mehra, R., Ramar, K., et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American Academy of Sleep Medicine clinical practice guideline. Journal of Clinical Sleep Medicine, 2017; 13(3), p. 479-504.
24. Rana, A. M., & Sankari, A. Central sleep apnea, in StatPearls [Internet]. 2023; StatPearls Publishing.
25. Sateia, M.J. International classification of sleep disorders. Chest, 2014; 146(5), p. 1387-1394.
26. Javaheri, S., Barbe, F., Campos-Rodriguez, F., Dempsey, J. A., Khayat, R., Javaheri, S. et al. Sleep Apnea: Types, Mechanisms, and Clinical Cardiovascular Consequences. J Am Coll Cardiol, 2017; 69(7), p. 841-858.
27. Lee, Y.-C., K.-Y. Chang, and M.J. Mador. Racial disparity in sleep apnea-related mortality in the United States. Sleep Medicine, 2022; 90, p. 204-213.
28. Chowdhuri, S., Pranathiageswaran, S., Loomis-King, H., Salloum, A., & Badr, M. S. Aging is associated with increased propensity for central apnea during NREM sleep. J Appl Physiol (1985), 2018; 124(1), p. 83-90.
29. Kasai, T., Taranto Montemurro, L., Yumino, D., Wang, H., Floras, J. S., Newton, G. E. et al. Inverse relationship of subjective daytime sleepiness to mortality in heart failure patients with sleep apnoea. ESC Heart Fail, 2020; 7(5), p. 2448-2454.
30. Baillieul, S., Revol, B., Jullian-Desayes, I., Joyeux-Faure, M., Tamisier, R., & Pépin, J. L. et al. Diagnosis and management of central sleep apnea syndrome. Expert review of respiratory medicine, 2019; 13(6), p. 545-557.
31. Martins, E. F., Martinez, D., Cortes, A. L., Nascimento, N., & Brendler, J. Exploring the STOP-BANG questionnaire for obstructive sleep apnea screening in seniors. Journal of Clinical Sleep Medicine, 2020; 16(2), p. 199-206.
32. Hwang, M., Zhang, K., Nagappa, M., Saripella, A., Englesakis, M., & Chung, F. et al. Validation of the STOP-Bang questionnaire as a screening tool for obstructive sleep apnoea in patients with cardiovascular risk factors: a systematic review and meta-analysis. BMJ open respiratory research, 2021; 8(1), p. e000848.
33. Pivetta, B., Chen, L., Nagappa, M., Saripella, A., Waseem, R., Englesakis, M. et al. Use and performance of the STOP-Bang questionnaire for obstructive sleep apnea screening across geographic regions: a systematic review and meta-analysis. JAMA network open, 2021; 4(3), p. e211009-e211009.
34. Lipford, M. C., Wahner-Roedler, D. L., Welsh, G. A., Mandrekar, J., Thapa, P., & Olson, E. J. et al. Correlation of the Epworth Sleepiness Scale and sleep-disordered breathing in men and women. Journal of Clinical Sleep Medicine, 2019; 15(1), p. 33-38.
35. Lee, J. L., Chung, Y., Waters, E., & Vedam, H. The Epworth sleepiness scale: Reliably unreliable in a sleep clinic population. Journal of Sleep Research, 2020; 29(5), p. e13019.
36. Walker, N. A., Sunderram, J., Zhang, P., Lu, S. E., & Scharf, M. T. Clinical utility of the Epworth sleepiness scale. Sleep and Breathing, 2020; 24, p. 1759-1765.
37. Scharf, M.T. Reliability and Efficacy of the Epworth Sleepiness Scale: Is There Still a Place for It? Nature and Science of Sleep, 2022; p. 2151-2156.
38. Habib, M., Khan, A. H., Imtiaz, S., Ahmed, J., Ansari, M. G. J. K., & Dars, J. A. et al. Predictive Abilities of Epworth Sleepiness Scale and Stop Bang in Identifying Patients at High Risk of Obstructive Sleep Apnea. Pakistan Journal of Medical & Health Sciences, 2023; 17(03), p. 577-577.
39. Light, M. P., Casimire, T. N., Chua, C., Koushyk, V., Burschtin, O. E., Ayappa, I. et al. Addition of frontal EEG to adult home sleep apnea testing: does a more accurate determination of sleep time make a difference? Sleep and Breathing, 2018; 22, p. 1179-1188.
40. Caples, S. M., Anderson, W. M., Calero, K., Howell, M., & Hashmi, S. D.et al. Use of polysomnography and home sleep apnea tests for the longitudinal management of obstructive sleep apnea in adults: an American Academy of Sleep Medicine clinical guidance statement. Journal of Clinical Sleep Medicine, 2021; 17(6), p. 1287-1293.
41. Rundo, J. V., & Downey III, R. Polysomnography. Handbook of clinical neurology, 2019; 160, p. 381-392.
42. Messineo, L., Eckert, D. J., Lim, R., Chiang, A., Azarbarzin, A., Carter, S. G. et al. Zolpidem Increases Sleep Efficiency and the Respiratory Arousal Threshold in Obstructive Sleep Apnea, in D29. SRN: NOVEL THERAPEUTIC APPROACHES TOWARDS SDB: CAN

PATHOPHYSIOLOGY LEND A HELPING HAND? 2020, American Thoracic Society. p. A6453-A6453.
43. Ingram, D. G., Crane, S. C., & Halbower, A. C. Polysomnography. Sleep in Children with Neurodevelopmental Disabilities: An Evidence-Based Guide, 2019; p. 27-43.
44. Carberry, J. C., Grunstein, R. R., & Eckert, D. J. The effects of zolpidem in obstructive sleep apnea–An open-label pilot study. Journal of Sleep Research, 2019; 28(6), p. e12853.
45. Badr, M.S. Central sleep apnea: risk factors, clinical presentation, and diagnosis. 2021; UpToDate.
46. Kramer, N. R., & Millman, R. P. Overview of polysomnography in adults. 2011, UpToDate.
47. Liendo, A., & Liendo, C. H. Comments on split-night polysomnography. Revista de la Facultad de Medicina, 2019; 67(1), p. 7-8.
48. Gerdung, C. A., Castro-Codesal, M. L., Nettel-Aguirre, A., Kam, K., Hanly, P. J., MacLean, J. E. et al. Feasibility of split night polysomnography in children to diagnose and treat sleep related breathing disorders. Sleep Medicine, 2022; 96, p. 107-112.
49. Rouse, J. K., Shirley, S. R., Holley, A. B., Mysliwiec, V., & Walter, R. J. Split-night polysomnography overestimates apnea-hypopnea index in high-risk professions. Military medicine, 2019; 184(5-6), p. e137-e140.
50. Jacobowitz, O., Afifi, L., Penzel, T., Poyares, D., Marklund, M., & Kushida, C. et al. Endorsement of:"treatment of adult obstructive sleep apnea with positive airway pressure: an American academy of Sleep Medicine Clinical Practice Guideline" by World Sleep Society. Sleep Medicine, 2022; 89, p. 19-22.
51. Chung, Y. M., Huang, W. K., & Wu, H. T. Topological Data Analysis Assisted Automated Sleep Stage Scoring Using Airflow Signals. arXiv preprint arXiv:2306.02857, 2023.
52. Patil, S. P., Ayappa, I. A., Caples, S. M., Kimoff, R. J., Patel, S. R., & Harrod, C. G. et al. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine clinical practice guideline. Journal of Clinical Sleep Medicine, 2019; 15(2), p. 335-343.
53. Schätz, M., Procházka, A., Kuchyňka, J., & Vyšata, O. et al. Sleep apnea detection with polysomnography and depth sensors. Sensors, 2020; 20(5), p. 1360.
54. Markun, L. C., & Sampat, A. Clinician-Focused Overview and Developments in Polysomnography. Current Sleep Medicine Reports, 2020; 6(4), p. 309-321.
55. Shrivastava, D. Polysomnography II: Sample Reports with Explanation of Various Terminologies. Making Sense of Sleep Medicine, 2022; p. 141-145.
56. Boulos, M. I., Jairam, T., Kendzerska, T., Im, J., Mekhael, A., & Murray, B. J. et al. Normal polysomnography parameters in healthy adults: a systematic review and meta-analysis. The Lancet Respiratory Medicine, 2019; 7(6), p. 533-543.
57. Rashid, N. H., Zaghi, S., Scapuccin, M., Camacho, M., Certal, V., & Capasso, R. et al. The value of oxygen desaturation index for diagnosing obstructive sleep apnea: a systematic review. The Laryngoscope, 2021; 131(2), p. 440-447.
58. Kramer, N.R. and Millman, R. P. Overview of polysomnography in adults. 2023; UpToDate.
59. Berry, R. B., Brooks, R., Gamaldo, C., Harding, S. M., Lloyd, R. M., Quan, S. F. et al. AASM Scoring Manual Updates for 2017 (Version 2.4). J Clin Sleep Med, 2017; 13(5), p. 665-666.
60. Cowie, M. R., Linz, D., Redline, S., Somers, V. K., & Simonds, A. K. et al. Sleep disordered breathing and cardiovascular disease: JACC state-of-the-art review. Journal of the American College of Cardiology, 2021; 78(6), p. 608-624.
61. Chen, J., Lin, S., & Zeng, Y. An update on obstructive sleep apnea for atherosclerosis: mechanism, diagnosis, and treatment. Frontiers in Cardiovascular Medicine, 2021; 8, p. 647071.
62. Faria, A., Macedo, A., Castro, C., Valle, E., Lacerda, R., Ayas, N. et al. Impact of sleep apnea and treatments on cardiovascular disease. Sleep Science, 2022; 15(2), p. 250.
63. Kidawara, Y., Kadoya, M., Morimoto, A., Daimon, T., Kakutani-Hatayama, M., Kosaka-Hamamoto, K. et al. Sleep Apnea and Physical Movement During Sleep, But Not Sleep Duration, Are Independently Associated With Progression of Left Ventricular Diastolic Dysfunction: Prospective Hyogo Sleep Cardio-Autonomic Atherosclerosis Cohort Study. Journal of the American Heart Association, 2022; 11(19), p. e024948.
64. Yoshihisa, A. and Takeishi, Y. Sleep disordered breathing and cardiovascular diseases. Journal of atherosclerosis and thrombosis, 2019; 26(4), p. 315-327.
65. Xie, C., Zhu, R., Tian, Y., & Wang, K. Association of obstructive sleep apnoea with the risk of vascular outcomes and all-cause mortality: a meta-analysis. BMJ open, 2017; 7(12), p. e013983.
66. Johnson, D. A., Thomas, S. J., Abdalla, M., Guo, N., Yano, Y., Rueschman, M. et al. Association between sleep apnea and blood pressure control

among blacks: Jackson Heart Sleep Study. Circulation, 2019; 139(10), p. 1275-1284.
67. Hou, H., Zhao, Y., Yu, W., Dong, H., Xue, X., Ding, J. et al. Association of obstructive sleep apnea with hypertension: a systematic review and meta-analysis. Journal of global health, 2018; 8(1).
68. Barbé, F., Durán-Cantolla, J., Sánchez-de-la-Torre, M., Martínez-Alonso, M., Carmona, C. et al. Effect of continuous positive airway pressure on the incidence of hypertension and cardiovascular events in nonsleepy patients with obstructive sleep apnea: a randomized controlled trial. Jama, 2012; 307(20), p. 2161-2168.
69. Linz, D., McEvoy, R. D., Cowie, M. R., Somers, V. K., Nattel, S., Lévy, P. et al. Associations of obstructive sleep apnea with atrial fibrillation and continuous positive airway pressure treatment: a review. JAMA cardiology, 2018; 3(6), p. 532-540.
70. Anter, E., Di Biase, L., Contreras-Valdes, F. M., Gianni, C., Mohanty, S., Tschabrunn, C. M. et al. Atrial substrate and triggers of paroxysmal atrial fibrillation in patients with obstructive sleep apnea. Circulation: Arrhythmia and Electrophysiology, 2017; 10(11), p. e005407.
71. Linz, D., Brooks, A. G., Elliott, A. D., Kalman, J. M., McEvoy, R. D., Lau, D. H. et al. Nightly variation in sleep apnea severity as atrial fibrillation risk. Journal of the American College of Cardiology, 2018; 72(19), p. 2406-2407.
72. Linz, D., Brooks, A. G., Elliott, A. D., Nalliah, C. J., Hendriks, J. M., Middeldorp, M. E. et al. Variability of sleep apnea severity and risk of atrial fibrillation: the VARIOSA-AF study. JACC: Clinical Electrophysiology, 2019; 5(6), p. 692-701.
73. Linz, D., Baumert, M., Desteghe, L., Kadhim, K., Vernooy, K., Kalman, J. M. et al. Nightly sleep apnea severity in patients with atrial fibrillation: potential applications of long-term sleep apnea monitoring. IJC Heart & Vasculature, 2019; 24, p. 100424.
74. Moula, A. I., Parrini, I., Tetta, C., Lucà, F., Parise, G., Rao, C. M. et al. Obstructive sleep apnea and atrial fibrillation. Journal of clinical medicine, 2022; 11(5), p. 1242.
75. Di Fusco, S. A., Pignalberi, C., Santini, L., Colivicchi, F., & Santini, M. Arrhythmias and sleep apnea: physiopathologic link and clinical implications. Journal of Interventional Cardiac Electrophysiology, 2020; 57(3), p. 387-397.
76. Acharya, R., Basnet, S., Tharu, B., Koirala, A., Dhital, R., Shrestha, P. et al. Obstructive sleep apnea: risk factor for arrhythmias, conduction disorders, and cardiac arrest. Cureus, 2020; 12(8).
77. Konishi, T., Kashiwagi, Y., Funayama, N., Yamamoto, T., Murakami, H., Hotta, D. et al. Obstructive sleep apnea is associated with increased coronary plaque instability: an optical frequency domain imaging study. Heart and vessels, 2019; 34(8), p. 1266-1279.
78. Ishiwata, S., Tomita, Y., Ishiwata, S., Narui, K., Daida, H., & Kasai, T. et al. Association between obstructive sleep apnea and SYNTAX score. Journal of Clinical Medicine, 2020; 9(10), p. 3314.
79. Qu, H., Guo, M., Zhang, Y., & Shi, D. Z. et al. Obstructive sleep apnea increases the risk of cardiac events after percutaneous coronary intervention: a meta-analysis of prospective cohort studies. Sleep and Breathing, 2018; 22, p. 33-40.
80. Pak, V. M., Strouss, L., Yaggi, H. K., Redeker, N. S., Mohsenin, V., & Riegel, B. et al. Mechanisms of reduced sleepiness symptoms in heart failure and obstructive sleep apnea. Journal of sleep research, 2019; 28(5), p. e12778.
81. Coniglio, A. C., & Mentz, R. J. Sleep breathing disorders in heart failure. Cardiology Clinics, 2022; 40(2), p. 183-189.
82. Javaheri, S., & Javaheri, S. Obstructive Sleep Apnea in Heart Failure: Current Knowledge and Future Directions. Journal of Clinical Medicine, 2022; 11(12), p. 3458.
83. Duan, A., Huang, Z., Hu, M., Zhao, Z., Zhao, Q., Jin, Q. et al. The comorbidity burden and disease phenotype in pre-capillary pulmonary hypertension: The contributing role of obstructive sleep apnea. Sleep Medicine, 2023; 101, p. 146-153.
84. Adir, Y., Humbert, M., & Chaouat, A. Sleep-related breathing disorders and pulmonary hypertension. European Respiratory Journal, 2021; 57(1).
85. Cao, W., Luo, J., Huang, R., & Xiao, Y. Implication of a novel measure of obstructive sleep apnea severity for cardiovascular morbidity. Sleep Medicine, 2023; 103, p. 204-210.
86. Ma, C., Zhang, Y., Liu, J., & Sun, G. A novel parameter is better than the AHI to assess nocturnal hypoxaemia and excessive daytime sleepiness in obstructive sleep apnoea. Scientific Reports, 2021; 11(1), p. 4702.
87. Azarbarzin, A., Sands, S. A., Stone, K. L., Taranto-Montemurro, L., Messineo, L., Terrill, P. I. et al. The hypoxic burden of sleep apnoea predicts cardiovascular disease-related mortality: the Osteoporotic Fractures in Men Study and the Sleep Heart Health Study. European heart journal, 2019; 40(14), p. 1149-1157.
88. Sunkonkit, K., Alzaid, M., Al-Saleh, S., & Amin, R. et al. The Clinical Utility of the Oxygen Desaturation Index to Predict Sleep-Disordered Breathing in Children, in B49. SLEEP, CHEST

WALL, AND NEUROMUSCULAR-OH MY! 2022; American Thoracic Society. p. A2992-A2992.
89. Wang, L., Ou, Q., Shan, G., Lao, M., Pei, G., Xu, Y. et al. Independent Association Between Oxygen Desaturation Index and Cardiovascular Disease in Non-Sleepy Sleep-Disordered Breathing Subtype: A Chinese Community-Based Study. Nature and Science of Sleep, 2022; p. 1397-1406.
90. Sreedharan, S. E., Agrawal, P., Rajith, R. S., Nair, S., Sarma, S. P., & Radhakrishnan, A. et al. Clinical and polysomnographic predictors of severe obstructive sleep apnea in the South Indian population. Annals of Indian Academy of Neurology, 2016; 19(2), p. 216.
91. Wali, S. O., Abaalkhail, B., AlQassas, I., Alhejaili, F., Spence, D. W., & Pandi-Perumal, S. R. et al. The correlation between oxygen saturation indices and the standard obstructive sleep apnea severity. Annals of thoracic medicine, 2020; 15(2), p. 70.
92. Kim, J. B., Seo, B. S., & Kim, J. H. Effect of arousal on sympathetic overactivity in patients with obstructive sleep apnea. Sleep Medicine, 2019; 62, p. 86-91.
93. Sabil, A., Gervès-Pinquié, C., Blanchard, M., Feuilloy, M., Trzepizur, W., Goupil, F. et al. Overnight Oximetry–derived Pulse Rate Variability Predicts Stroke Risk in Patients with Obstructive Sleep Apnea. American Journal of Respiratory and Critical Care Medicine, 2021; 204(1), p. 106-109.
94. Sequeira, V. C. C., Bandeira, P. M., & Azevedo, J. C. M. Heart rate variability in adults with obstructive sleep apnea: a systematic review. Sleep Science, 2019; 12(3), p. 214.
95. Mejía-Mejía, E., May, J. M., Torres, R., & Kyriacou, P. A. Pulse rate variability in cardiovascular health: A review on its applications and relationship with heart rate variability. Physiological Measurement, 2020; 41(7), p. 07TR01.
96. Blanchard, M., Gerves-Pinquie, C., Feuilloy, M., Le Vaillant, M., Trzepizur, W., Meslier, N. et al. Association of nocturnal hypoxemia and pulse rate variability with incident atrial fibrillation in patients investigated for obstructive sleep apnea. Annals of the American Thoracic Society, 2021; 18(6), p. 1043-1051.
97. Fazeli, M. A., Najafi, T., & Ghadami, M. R. Polysomnographic characteristics in patients with obstructive sleep apnea with blood pressure" dipping" and" non-dipping" pattern. Scandinavia Journal of Sleep Medicine, 2022; 2(1).
98. Wang, Q., Zhang, C., Jia, P., Zhang, J., Feng, L., Wei, S. et al. The association between the phenotype of excessive daytime sleepiness and blood pressure in patients with obstructive sleep apnea-hypopnea syndrome. International journal of medical sciences, 2014; 11(7), p. 713.
99. Kendzerska, T., Gershon, A. S., Hawker, G., Leung, R. S., & Tomlinson, G. et al. Obstructive sleep apnea and risk of cardiovascular events and all-cause mortality: a decade-long historical cohort study. PLoS medicine, 2014; 11(2), p. e1001599.
100. Gami, A. S., Olson, E. J., Shen, W. K., Wright, R. S., Ballman, K. V., Hodge, D. O. et al. Obstructive sleep apnea and the risk of sudden cardiac death: a longitudinal study of 10,701 adults. Journal of the American College of Cardiology, 2013; 62(7), p. 610-616.
101. Smagula, S. F., Stone, K. L., Redline, S., Ancoli-Israel, S., Barrett-Connor, E., Lane, N. E. et al. Actigraphy and polysomnography measured sleep disturbances, inflammation, and mortality among older men. Psychosomatic medicine, 2016; 78(6), p. 686.
102. Varghese, L., Rebekah, G., Oliver, A., & Kurien, R. Oxygen desaturation index as alternative parameter in screening patients with severe obstructive sleep apnea. Sleep science, 2022; 15(Spec 1), p. 224.

Citation

Mahmoud, M., Ghoneim, A., Ismail, N., Ibrahim, W. Polysomnographic Parameters and Assessment of Cardiovascular Complications in Patients with Sleep Disordered Breathing. ***Zagazig University Medical Journal,*** **2025; (507-522): -. doi: 10.21608/zumj.2024.256144.3058**