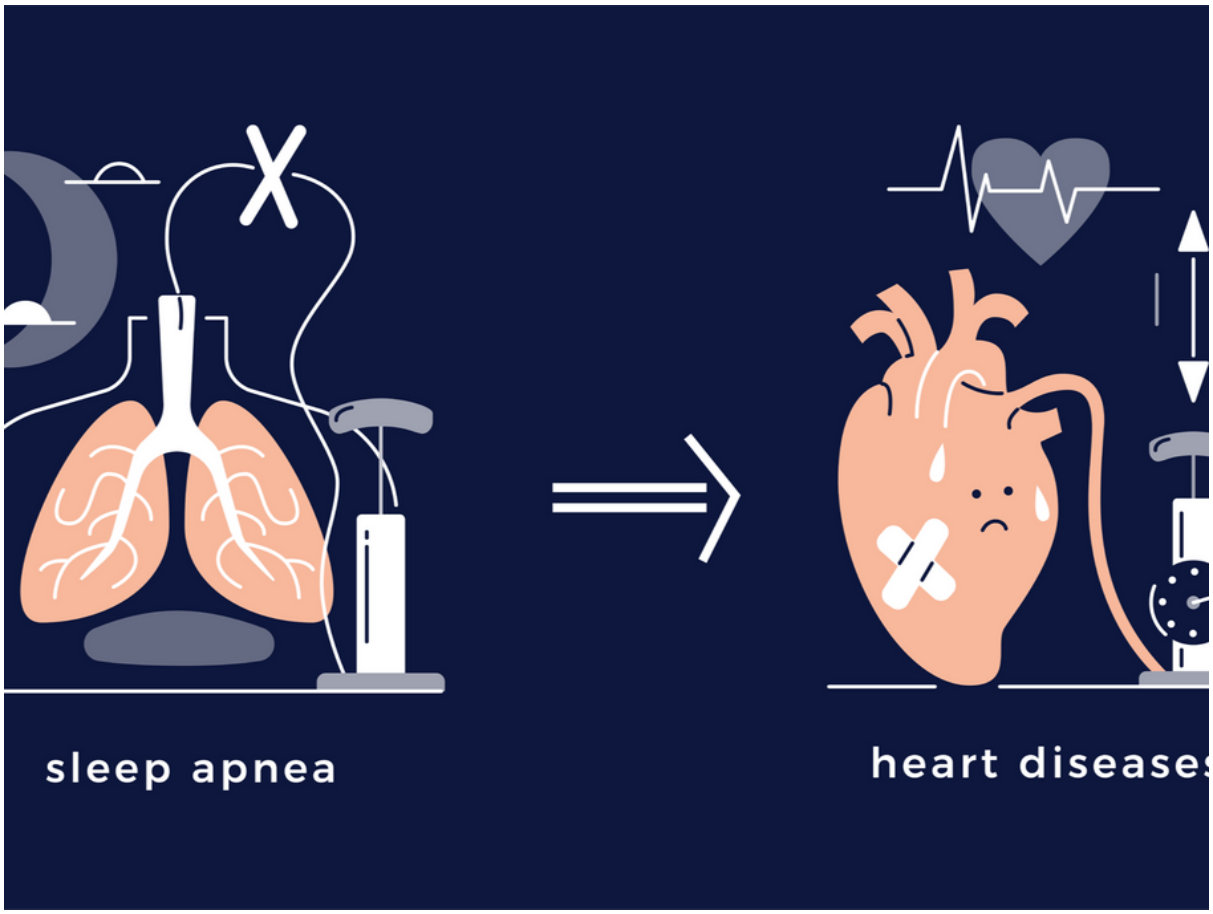
SLEEP & HEART CONNECTION



Uncovering the Link Between Sleep Disorders and Heart Failure: HSAT Implications for Improved Patient Care

Heart failure has emerged as the primary reason for hospitalization among individuals aged 65 and older in the United States. Statistics show that approximately 6.7 million Americans suffer from heart failure, and both the prevalence and mortality rates are increasing each year. Sleep apnea has been identified as a standalone risk factor for cardiovascular disease, and both OSA and CSA can directly or indirectly exacerbate the morbidity and mortality of HF (Heart Failure) through a variety of physiologic mechanisms, with the prevalence of obstructive sleep apnea combined with CHF(Congestive Heart Failure) as high as 11%.

With the continuous progress and development of telemedicine technology and wearable devices, such as a wrist pulse oximeter, the monitoring and early warning of cardiovascular diseases are no longer limited to hospitals. It is reasonable to believe that more realistic and accurate data samples and lower healthcare costs can be achieved based on a high degree of attention and aggressive treatment of CHF patients with comorbid OSA.

CHF patients

What is CHF (Congestive Heart Failure)?

Definition:

Congestive heart failure is a chronic condition. In this situation, the heart muscle is unable to adequately pump sufficient blood to fulfill the body's overall requirements for oxygen and blood. It tends to get worse gradually over time.

At first, the heart tries to make up for the situation in three ways:

- Building additional muscle mass involves an augmentation in muscle size as the contracting cells within the heart increase in size.
- The heart undergoes stretching to enhance its contraction strength, aiming to meet the increased demand for pumping more blood throughout the body. Consequently, this leads to the enlargement of the heart over time.
- Beating at a swifter rate serves to enhance the heart's output.

Other changes are happening in the body:

- Vasoconstriction. It is an attempt to keep blood pressure elevated to compensate for the weakened pumping ability of the heart.
- The kidneys preserve additional salt and water instead of expelling them in urine. This results in an elevated blood volume, contributing to the maintenance of blood pressure and enabling a more robust pumping action by the heart. However, prolonged retention of this surplus volume can strain the heart, exacerbating heart failure over time.

Symptoms:

The main symptoms of CHF (congestive heart failure) are:

- Breathlessness after activity or at rest.
- Experiencing persistent fatigue and finding physical activity draining.
- Swelling of ankles and legs.
- Experiencing dizziness and episodes of fainting.

Additional symptoms may include an enduring cough, an accelerated heart rate, and a diminished appetite, depending on the individual.

Causes:

Congestive heart failure frequently arises from the simultaneous impact of various issues affecting the heart. Common factors that may contribute to heart failure encompass:

- Hypertension. The heart of hypertensive patients commonly suffers from left ventricular hypertrophy, enlarged atria, etc. When the cardiac loading capacity is increased, the neuroendocrine mechanisms are also affected, and problems such as the activation of the renin-angiotensin-aldosterone system (RAAS) contribute to the onset and worsening of heart failure.
- Coronary heart disease. Cardiomyocytes in patients with coronary heart disease are often unable to accomplish effective contraction, and extensive necrosis of the myocardium caused by decreased contractile function is a major cause of heart failure.
- Arrhythmia. The irregular heart rhythms problem can induce an excessive or insufficient pace of heartbeats, imposing additional strain on the heart. Both fast and slow heart rates may contribute to the development of heart failure.
- Heart valve problems. The heart's valves play a crucial role in maintaining the proper flow of blood. When a valve malfunctions, the heart is compelled to exert more effort in pumping blood, leading to a gradual weakening of the heart over time.
- Congenital heart defect. In cases where the heart, its chambers, or valves haven't developed properly, the remaining components of the heart must compensate by

working more strenuously to circulate blood. This increased workload can potentially result in the onset of heart failure.

- Some other diseases. Obesity, anemia, excessive alcohol consumption, hyperthyroidism, or HIV infection can also lead to heart failure in some cases.

Correlation Between Congestive Heart Failure and Sleep Apnea Disorders (OSA)

With further understanding of the risk factors associated with HF, it has been found that OSA is strongly associated with increased HF morbidity, rehospitalization, and mortality.

OSA (Obstructive Sleep Apnea) is frequent hypoventilation or even apnea caused by upper airway obstruction during sleep. This sleep disorder can recur during the night, making it difficult for the body to receive adequate oxygen, which then induces problems such as intermittent hypoxemia, hypercapnia, and sleep architecture disorders.

Recent studies have shown that the underlying pathophysiologic mechanisms by which OSA accelerates the progression of HF are mainly related to the following 3 points:

1. Mechanical effects

This is one of the main possible mechanisms by which OSA accelerates the process of HF. Specifically, it refers to pharyngeal occlusion caused by factors such as retroflection of the tongue during nighttime sleep, resulting in dyspnea and, ultimately, a reduction in left ventricular beat volume and cardiac output. In OSA patients with normal cardiac function, beat-to-beat volume can return to baseline levels due to myocardial compensation for the temporary termination of respiration, whereas in HF patients with cardiac dysfunction, the mechanical effects of OSA increase the already reduced beat-to-beat volume and cardiac output, exacerbating myocardial damage and remodeling.

2. Nocturnal intermittent hypoxia effect

Recurrent nighttime upper airway obstruction leading to intermittent hypoxia and hypercapnia represents a significant potential mechanism through which obstructive sleep apnea (OSA) expedites the progression of heart failure (HF). This process, in turn, hastens the emergence of HF-related causes and contributory factors, thereby detrimentally impacting the prognosis of HF. This adverse effect is mediated by factors

such as disrupted sleep patterns, disturbances in autonomic function, heightened oxidative stress, metabolic irregularities, and other related mechanisms.

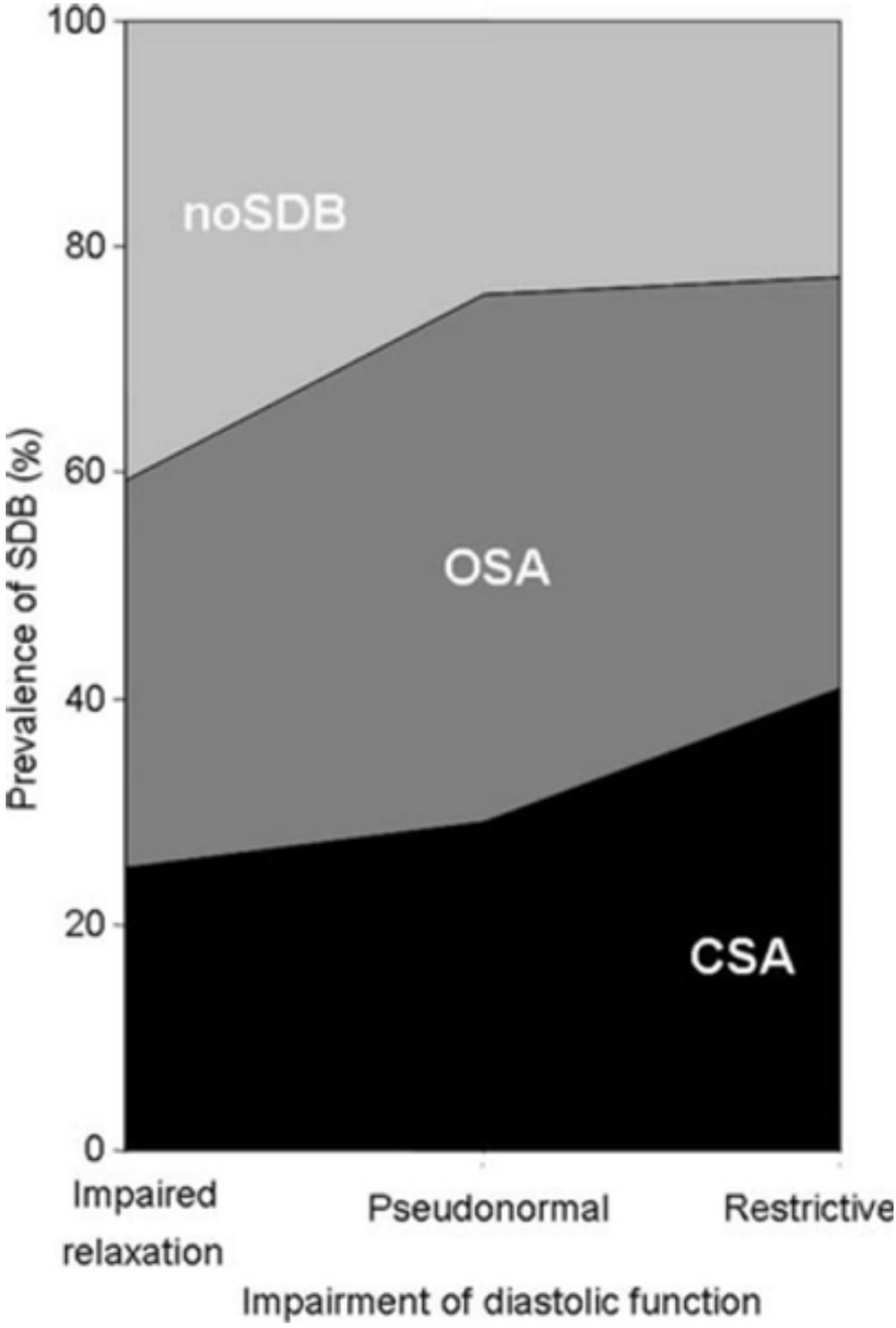
Fragmented sleep results from repeated awakenings caused by intermittent nocturnal hypoxia, disrupting normal non-rapid and rapid eye movement sleep patterns. During non-REM sleep, OSA leads to disturbances in autonomic nervous system activity, initially causing a slowing of the heart rate, followed by an increase in blood pressure and an increase in heart rate. In contrast, during REM sleep, there is a significant increase in sympathetic nervous system activity, leading to wider fluctuations in blood pressure and heart rate, and these changes greatly attenuate the restorative effects of sleep. In addition, in HF patients, the duration of sleep is reduced by about 1.3 hours compared with that of healthy individuals, which may further weaken the restorative effect of sleep and increase the risk of death in HF patients if combined with OSA at the same time.

OSA shifts the balance of the nocturnal autonomic nervous system toward sympathetic dominance in patients with HF and may be related to the stimulation of central and peripheral chemoreceptors by intermittent nocturnal hypoxia and CO2 retention. OSA episodes not only eliminate the inhibition of sympathetic activity induced by pulmonary traction but also decrease per-pulse volume and blood pressure, leading to a decrease in afferent impulses to carotid sinus pressure receptors, which ultimately results in increased sympathetic activity. In addition, both OSA patients and HF patients have a reduced rate of high-frequency heart rate variability, which indicates diminished vagal tone and increased mortality in patients with cardiovascular disease. If the reduced

effects of OSA and HF on HF HRV are superimposable, then the coexistence of OSA may further increase mortality in patients with HF.

3. Relationship between OSA and the cardiovascular event chain

In addition to directly exacerbating HF through the mechanisms described above, OSA can also lead to further increases in HF morbidity and mortality by promoting the development of the cardiovascular chain of events.



Necessity of Routine OSA Screening for Patients with CHF

As mentioned above, the body takes a number of actions to compensate for congestive heart failure. These interim measures serve to conceal the issue of heart failure, yet they do not address its underlying cause. Heart failure persists and progresses until these compensatory mechanisms are no longer effective. Eventually, the heart and body can't keep up, and patients experience fatigue, shortness of breath, or other symptoms that often prompt them to necessitate a visit to the doctor.

Sometimes, the patients affected with CHF(congestive heart failure suffer from sleep apnea without showing visible signs and symptoms, such as daytime sleepiness, dryness in the mouth, or obesity. As a result, most scientists are looking for ways to diagnose diseases at an early stage through sleep test data.

Put simply, it focuses on recording the brain waves, oxygen levels in blood, heart, and breathing rate of the patients while they sleep. This diagnostic procedure works as a milestone, leading to in-time detection of the condition, which can otherwise induce coronary heart disease or increased chances of mortality due to heart failure.

CheckMe O2Max for Sleep

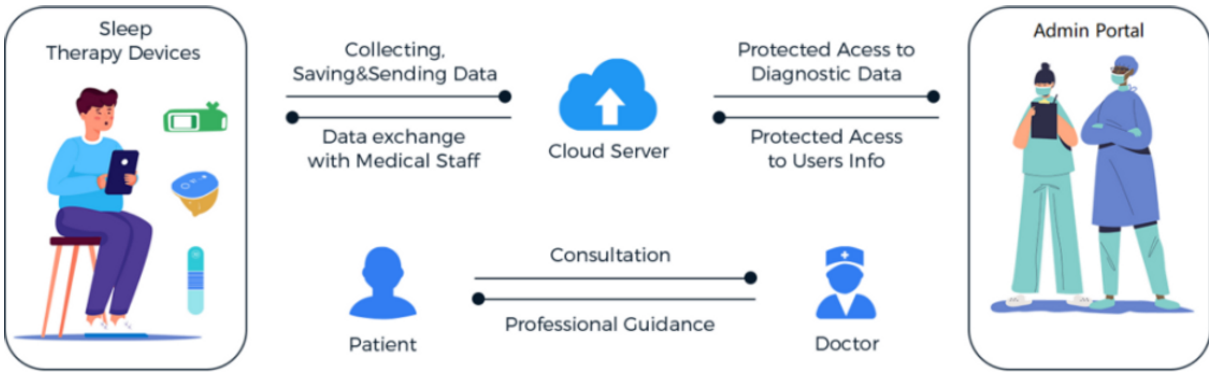
Tips for Home Sleep Apnea Test of CHF Patients

What Should Physicians Do To Serve and Help the Patient Remotely?

Ongoing support and assistance are the most vital elements for remote patient care. When concerns or issues arise during the testing procedure, doctors should ensure urgent availability to respond to them. Moreover, patients should receive guidance on interpreting the findings and spotting possible indicators that require treatment, such as SpO2 and heart rate.

Another crucial factor affecting the HSAT results is the incorrect pressure setting for your continuous positive airway pressure (CPAP) device. To help patients, physicians

should adjust the CPAP treatment according to their blood pressure or provide them with an auto-adjusting CPAP device for use at home.



Viatom remote sleep apnea treatment solution

Which Part of the Sleep Apnea Test Requires More Focus for Chronic Heart Failure Patients?

For chronic heart failure (CHF) patients, physicians should focus on assessing the following key points to diagnose the disease and recommend further treatment.

1. Oxygen Saturation

The average oxygen saturation (SPO2) levels for patients performing HSAT should fall around 97 or 99%, while levels below 94% indicate desaturation. Depending on the value obtained for desaturation per hour, sleep apnea ranges from mild to moderate or severe levels.

You can obtain this value by dividing the total number of desaturations by the total time. Here's how to interpret the results obtained through calculation:

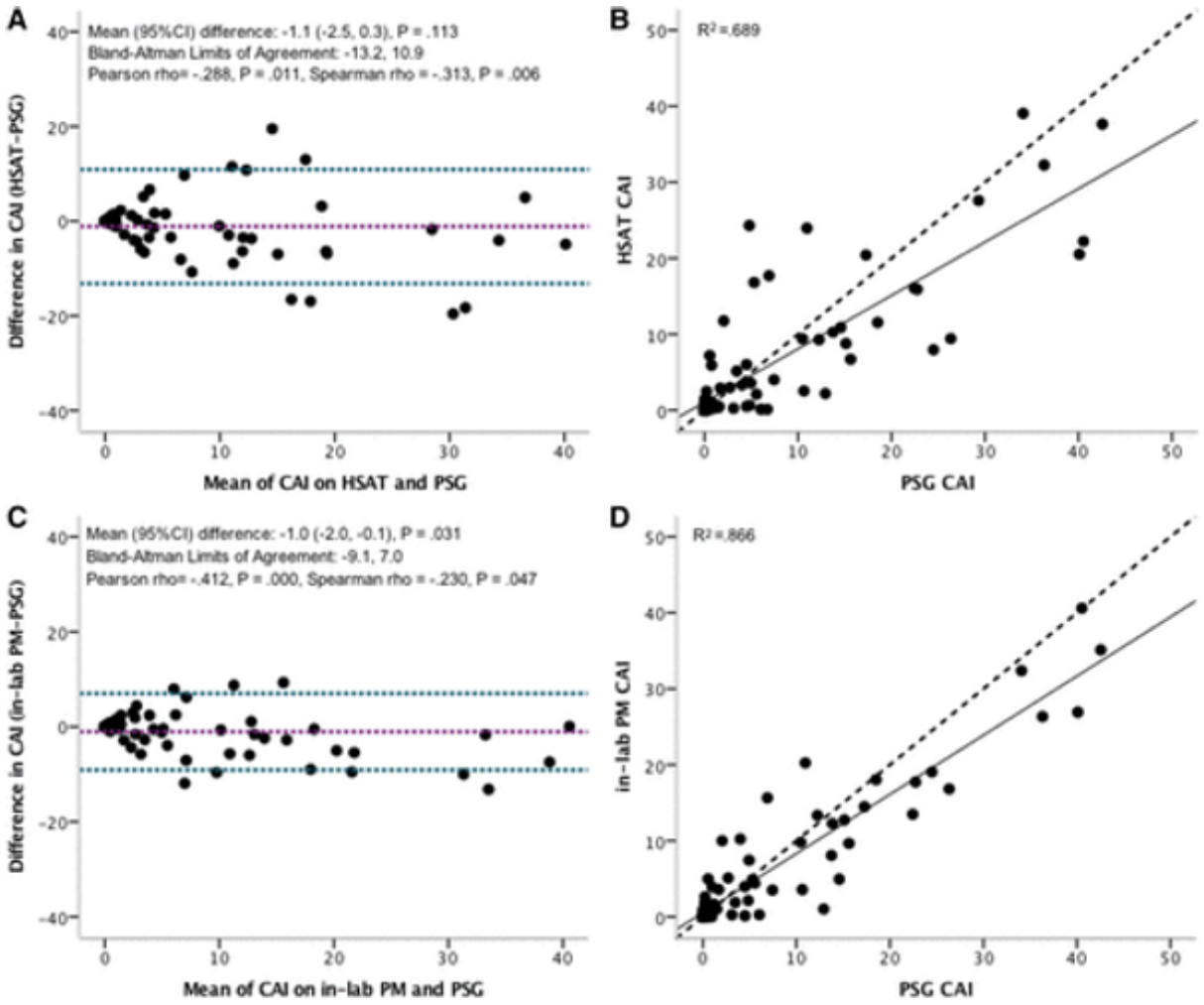
· A value of five or above will indicate a mild condition

· Between 15 and 30 will indicate a moderate condition

· A value above 30 will indicate a severe condition

2. Pulse Rate

An adult's regular sleeping pulse rate is about 60-100 beats/min, which can slow down during sleeping periods. However, the rates can show rapid fluctuation and can rise above ordinary when they suffer from sleep apnea or related disorders as an indication of CHF. Physicians can observe these changes to treat the patients accordingly.



Limitations of the Standard Wrist Pulse Oximeters

Although the standard overnight oxygen monitors are available at the sale price, they have several drawbacks for both the physicians and sleeping apnea solution suppliers, compromising the quality of results.

1. Small Battery

Many sleep oxygen monitors for HSAT offer a 16-hour operational battery life after a single charge, which is inadequate for reliable, continuous monitoring and fluid data collection. In the case of clinical monitoring after at-home testing, this small battery time can be a nuisance, leading to unwanted interruptions and frequent charging sessions to continue the procedure.

2. Inaccurate Results

The in-built recording intervals in wrist pulse oximeters have a huge impact on the accuracy of results after HSAT. Many low-priced oximeters take several seconds to record data. As a result, the sensors cannot register the minute physiological changes during sleep, leading to a substandard treatment plan and inaccurate diagnosis for the patients.

3. Complex Data Collection

Most sleep o2 monitors lack analytical reporting features and a simplified version of collected data to facilitate patients. It hinders the remote monitoring capabilities of patients while transitioning the correct and easy interpretation of the output to a challenging one.