

Review Article

Sleep with the Rhythm of your Heart

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Abstract

Arrhythmias during sleep are often detected on routine cardiac monitoring. Specialist cardiology opinion and input is often sought in order to assess whether nocturnal arrhythmias are clinically relevant or not. For the most part, bradyarrhythmias in the young fit individual are a variation of normal and require no further investigations. However even in normal sleep there are cardiovascular diseases that predispose patients to malignant arrhythmias, including heart failure, coronary heart disease and certain cardiac channelopathies. Primary sleep disorders such as obstructive sleep apnoea and alterations in sleep patterns during shift work have also been shown to have a proarrhythmogenic effect. This review aims to provide the reader with a broad overview of which arrhythmias are simply a reflection of normal changes in cardiac physiology during sleep, and which abnormal potentially dangerous arrhythmias require referral and treatment.

ABBREVIATIONS

AF: Atrial Fibrillation, AHI: Apnoea-Hypopnea Index, CCF: Congestive Cardiac Failure, CPAP: Continuous Positive Airway Pressure, CSA: Central Sleep Apnoea, ICD: Implantable Cardiac Defibrillator, NREM: Non Rapid Eye Movement, OSA: Obstructive Sleep Apnoea, PVC: Premature Ventricular Complex, REM: Rapid Eye Movement, SCD: Sudden Cardiac Death, LQTS: Long QT Syndrome.

INTRODUCTION

Sleep is generally considered a protective process, a chance for the human body to repair and restore itself. Despite this, up to 15% of sudden cardiac deaths occur at night [1]. Physiological fluctuations of the autonomic nervous system on cardiac rhythm can lead to the development of both benign and malignant arrhythmias. It is important for physicians to have an understanding of which arrhythmias are normal and abnormal during sleep in order to prevent over treatment and premature death.

Normal cardiovascular physiology during sleep

Normal sleep is divided into two stages, non rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. These stages occur in a cyclical manner, with approximately 4 to 5 NREM/REM sleep cycles occurring each night. Each stage is characterised by its own autonomic profile leading to fluctuations in sympathetic and parasympathetic activity. NREM sleep is characterised by a decrease in sympathetic activity with an accompanying shift towards parasympathetic predominance. This leads to associated bradycardia, reduced blood pressure and

cardiac output. During REM sleep, sympathetic tone dominates resulting in heightened restlessness, disordered breathing and an increase in heart rate and blood pressure at similar levels to when awake.

Normal sleep and associated arrhythmia

Bradycardia, 1st degree heart block and Wenckebach

The relative shift from sympathetic to parasympathetic neuronal dominance during normal sleep leads to alterations in cardiac electrophysiology. Studies of young individuals free of cardiovascular disease have shown that bradyarrhythmias consisting of sinus bradycardia, sinus pauses, first degree and second degree (Mobitz type 1) heart block are not uncommon [2], and are seen more frequently in athletes [3]. These arrhythmias are, for the most part, both asymptomatic and are of no prognostic significance. No further investigations or interventions are usually required unless accompanied by symptoms. With aging, healthy individuals demonstrate an overall decrease in vagal tone leading to an increase in resting heart rate. Over the age of 60 nocturnal sinus pauses, first degree heart block and second degree (Mobitz type 1) heart block becomes increasingly uncommon (Table 1) [4]. The clinical significance of finding such bradyarrhythmias remains uncertain but it may trigger a search for conduction system disease, especially in individuals who present with symptoms of syncope.

Ventricular arrhythmias and sudden cardiac death

The incidence of ventricular arrhythmias is reduced during sleep, as is the incidence of myocardial infarction, sudden cardiac death (SCD) and implantable cardiac defibrillator (ICD)

Table 1: Prevalence of bradyarrhythmias in healthy subjects during sleep [4].

Study	Brodsky et al [2]	Sabotka et al [5]	Fleg and Kennedy [6]	Kantelip et al [7]
Size	50	50	98	50
Age range (years)	23 - 27	22 - 28	60 - 85	80 - 100
Sex (M:F)	50:0	0:50	69:29	6:44
Bradycardia <40bpm	24%	8%	2%	0%
Sinus pauses > 1.5s	68%	36%	2%	12%
1 st Degree AV block	8%	12%	-	-
Wenckebach AV block	6%	4%	1%	2%

AV: Atrio Ventricular

discharges. An overall decrease in sympathetic tone and shift towards a parasympathetic control during NREM sleep seems to have a protective effect. Despite this relatively protected period, up to 15% of SCD's occur at night, with a peak incidence in the early morning hours after waking [1]. The usual nocturnal decline in sympathetic activity is altered in individuals with coronary heart disease and congestive cardiac failure (CCF), due to increased baseline sympathetic tone. Authors propose that surges of unopposed cardiac sympathetic nerve activity from an elevated baseline in REM sleep can trigger ventricular fibrillation and ventricular tachycardia [5]. The elevation of both arterial blood pressure and sympathetic tone associated with nocturnal apnoeas has also been linked to onset of non-sustained ventricular tachycardia [6,7]. The detection of non-sustained ventricular tachycardia however should promote further assessment of a patient's symptoms and ejection fraction for consideration of ICD therapy.

Cardiac Channelopathies

Brugada and Long QT syndrome are two cardiac channelopathies that demonstrate a nocturnal predisposition to ventricular arrhythmias and sudden cardiac death. Long QT syndrome (LQTS) is a genetic arrhythmogenic disorder characterised by prolonged QT interval on an electrocardiogram and a propensity to ventricular arrhythmias. Several genotypes have been identified and the incidence of nocturnal events varies with each. In LQT1 only 3% of deaths occur during sleep, whereas there is a much higher incidence rate in LQT2 at 29% and 39% in LQT3 [8]. Sinus node pauses of up to nine seconds have been documented in young adults with normal cardiac function during REM sleep. Prolonged periods of a systole can trigger early after depolarization and in turn ventricular arrhythmias in patients with predisposed risk such as those with LQTS [5].

Brugada syndrome is an autosomal dominant inherited disorder; it is characterized by right bundle branch block, ST elevation in leads V1-V3 and an increase risk of SCD. Mutations of genes coding for single sodium channels, particularly SCN5A, predispose to progressive conduction disease [9-11]. A study of 12 patients with Brugada syndrome demonstrated that ventricular fibrillation occurred most frequently during sleep between the hours of midnight and 6am [12]. Patients with Brugada syndrome who survive ventricular arrhythmias appear to have low nocturnal vagal tone [13]. The link between sleep and arrhythmogenesis in this patient group needs further investigation.

Primary sleep disorders and associated arrhythmias

Obstructive sleep apnoea: Obstructive sleep apnoea (OSA) is a primary sleep disorder associated with cardiovascular disease. OSA is characterised by repetitive interruption of ventilation during sleep secondary to upper airway collapse on inspiration. Frequent cessation in ventilation results in intermittent periods of hypoxemia and hypercapnia, which provokes arousal from sleep. Each apnoeic episode results in autonomic, haemodynamic and neuroendocrine consequences, which may help explain the link between OSA and a range of cardiovascular disease (Figure 1). Cardiac arrhythmias occur commonly in patients with OSA, however the exact mechanisms underlying the relationship remain unclear. It is thought there may be a temporal relationship, with arrhythmias occurring more frequently after a respiratory event. The most commonly associated arrhythmia observed in patients with OSA is cyclic variation of heart rate. This describes progressive bradycardia in periods of apnoea, the rate of which is proportional to degree of hypoxaemia [14]. Other commonly described bradyarrhythmias in OSA are atrioventricular block, sinus pauses and a systole (up to 13 seconds in duration) [15]. OSA is an independent risk factor for atrial fibrillation (AF). Data from the Sleep Heart Study has shown that the odds of AF in patients with severe sleep disordered breathing are four times higher compared to subjects without sleep disordered breathing [16]. Currently limited data is available to suggest that treatment of OSA with continuous positive airway pressure (CPAP) will reduce the risk of recurrent AF. An observational study of 130 patients electrically cardioverted from AF showed that patients with untreated OSA had a higher recurrence of AF at 83%, compared with recurrence rates of 42% for treated OSA and 53% in patients without a diagnosis of OSA [17]. Main stay treatment of OSA is nasal CPAP. The positive airway pressure at night maintains airway patency and significantly reduces apnoeic episodes. Proven treatment benefits include a reduction in daytime somnolence, an improved quality of life, lower blood pressure and enhanced mood. The use of CPAP as an adjunct for arrhythmia prevention in patients with apnoea is discussed later in this text.

Congestive cardiac failure

CCF is a common disease associated with significant economic cost, morbidity and mortality. A well-documented co-morbidity of this patient group is sleep-related breathing disorders and is associated with poor outcome [18]. Up to 20% of heart failure

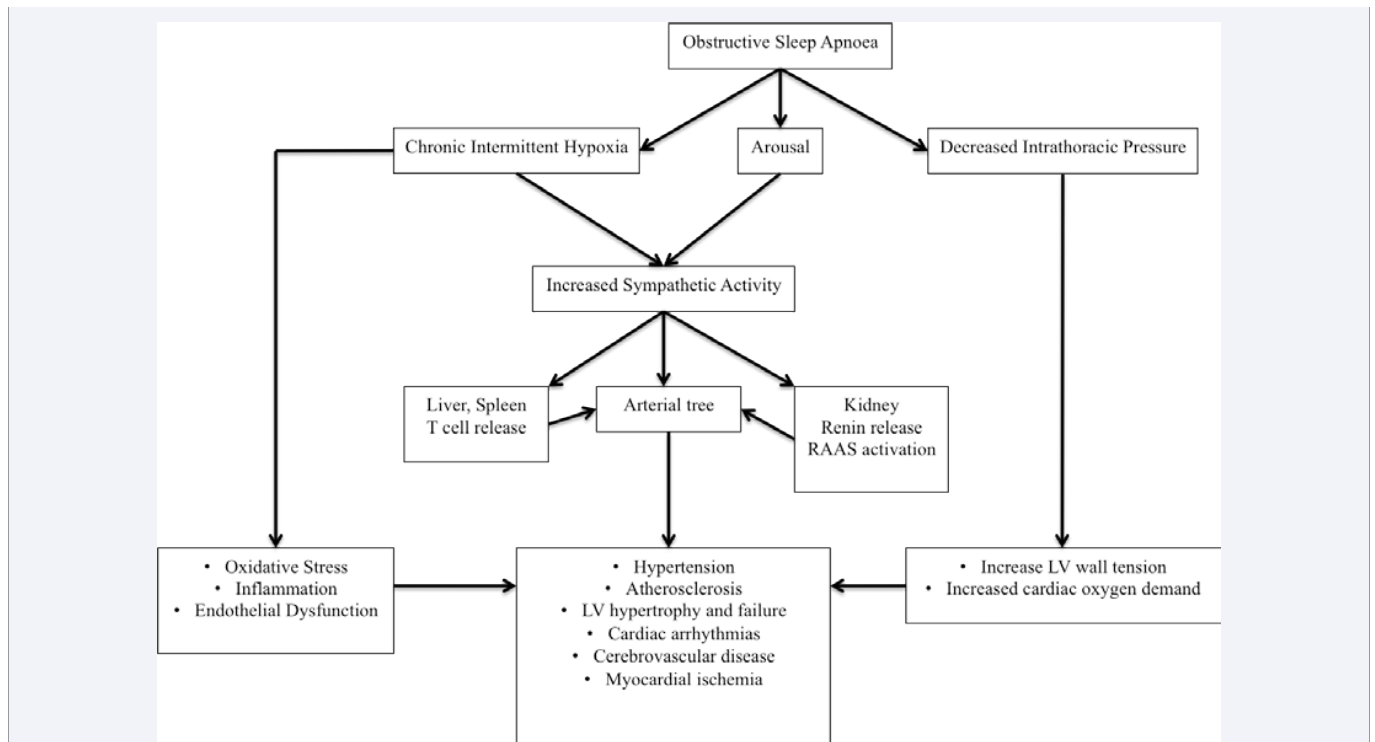


Figure 1 Pathophysiological effects of obstructive sleep apnoea.
 RAAS: Renin-Angiotensin-Aldosterone system LV: Left Ventricle

related deaths occur during the night [19]. CCF is associated with disordered nocturnal breathing with up to 50% of patients suffering from either OSA or central sleep apnoea (CSA) [20]. CSA describes a heterogeneous group of sleep related breathing disorders in which temporary withdrawal of respiratory drive leads to cessation of breathing, hypoxia and finally arousal from sleep. It differs from OSA in that it has a central nervous system component and distinct profile characterized by Cheyne-Stokes breathing. This cyclical process leads to a progression of heart failure and in turn elevation of sympathetic tone and associated arrhythmias [21-23]. Both OSA and CSA have been linked with higher incidences of nocturnal arrhythmias and an overall increase in mortality rates in patients with CCF [24-27]. Although arrhythmias are a recognised complication of CCF, there are no definitive methods to assess potential risk for malignant arrhythmias. The apnoea-hypopnea index (AHI) is an independent predictor of poor prognosis in patients with CCF [28], and low daytime arterial carbon dioxide is associated with an increased risk of nocturnal ventricular tachycardia [29]. In patients with daytime hypocapnia, or who have an AHI index score of ≥ 15 it may be suggested that they are further investigated with 24-48 hours of Holter monitoring.

Therapeutic approaches to apnoea

The main stay of treatment for patients with both OSA and CSA remains mechanical breathing assist devices. The use of CPAP and adaptive servo-ventilation (ASV) therapy in patients with OSA and CSA improves exercise tolerance and relieves heart failure symptoms but its role in arrhythmia prevention remains controversial [30-33]. In contrast to OSA, treatment

response to CPAP therapy in patients with CSA is less uniform [34,35]. Furthermore, the benefits of these long-term therapies are limited by patient compliance. For these reasons, alternative therapies have been investigated. Phrenic nerve pacing offers an alternative approach to regulating respiratory patterns. Therapy is currently targeted at patients with respiratory paralysis from cervical cord injury and patients with central alveolar ventilation syndrome [36,37]. Supportive outcome data has led to research in other selected patient groups including those with OSA or CSA and underlying CCF. Chronic nerve stimulation through transthoracic surgical placement of cuff electrodes is not well suited in patients with advanced CCF [38]. A novel implantable pacing system offers an alternative approach to avoid such limitations. Early research has focussed on targeted therapy in patients with CSA and CCF. Prospective studies to date have demonstrated unilateral phrenic nerve pacing as a safe, feasible and effective approach for the treatment of CSA [39-41]. More recently, Abraham et al. demonstrated additional improvements in cardiac symptoms, sympathetic surges and patient outcomes [42]. Further work is required to assess the efficacy of this therapy.

Sleep duration and shift work patterns

It is well documented that shift work has many health related effects including disturbed sleep. Shift work is the most common cause of circadian rhythm disturbance, leading to misalignment between an individual's environment and sleep wake cycle. In a survey of 1010 people conducted by the American National Sleep Foundation shift workers on average slept less during the week (6hours and 30minutes) compared to regular day

workers (6hours and 54minutes) [43]. Shorter sleep duration and shift work increases an individual's risk of cardiovascular disease by up to 40% [44]. Data from the Coronary Artery Risk Development in Young Adults (CARDIA) cohort study showed a shorter duration of sleep was associated with a greater likelihood of developing hypertension [45]. Furthermore longer sleep duration is associated with a significantly reduced incidence of coronary artery calcification [46].

Shift work may have adverse effects on autonomic balance. Sympathetic tone is controlled by stages of sleep whilst parasympathetic activity by circadian rhythms. In a study comparing Holter monitor results in individuals after a night of sleep and then a subsequent night deprived of sleep, there was no difference in the degree of sinus arrhythmia [47]. This suggests that parasympathetic driven sinus arrhythmias will continue to occur in shift workers whilst awake overnight. Individuals experiencing these natural arrhythmias as palpitations do not necessitate any further management other than reassurance. Lastly, the number of nights worked by an individual has been shown to have a cumulative effect on the number of ventricular extra systoles seen [48] as well as QTc prolongation on the electrocardiogram [49]. It is likely therefore that shift work and sleep deprivation has a pro-arrhythmic effect.

CONCLUSION

The majority of sleep arrhythmias detected during sleep are benign. Appropriate investigation and treatment of nocturnal arrhythmias remains a challenge to physicians. Commonly observed sleep arrhythmias in the well population with normal cardiac function include sinus bradycardia, sinus pauses, first degree and type-1 second degree AV block. Arrhythmias linked to structural and coronary heart disease typically warrant further investigation and specialist referral. Despite the protective period of increased nocturnal vagal tone, the rates of ventricular arrhythmias, ICD shocks and sudden cardiac death peak during the early hours of the morning between the hours of midnight and 6am. The aetiology of nocturnal arrhythmias is likely related to abrupt changes in autonomic tone. Those with underlying cardiovascular disease as outlined in this text are more sensitive to these autonomic changes. Both obstructive and central sleep apnoea are prevalent in patients with congestive heart failure. The use of mask-based mechanical assisted breathing devices remains the mainstay of symptom management in this patient group however their role in arrhythmia prevention remains controversial. The novel therapy of transvenous phrenic nerve pacing offers an alternative strategy and crucially is not limited by patient compliance. Early studies suggest unilateral phrenic nerve pacing is a safe, feasible and effective approach for the treatment of CSA but its role in arrhythmia prevention needs further investigation.

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