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The Scoring of Cardiac Events During Sleep

Sean M. Caples, D.O.[1]; Virend K. Somers, M.D.[1]; Carol L. Rosen, M.D.[2]; Win K. Shen, M.D.[1]; Apoor S. Gami, M.D.[1]; William Cotts, M.D.[3]; Michael Adams[4]; Parvin Dorostkar, M.D.[2]; Kalyanan Shivkumar, M.D.[5]; Timothy I. Morgenthaler, M.D.[1]; Edward J. Stepanski, Ph.D.[6]; Conrad Iber, M.D.[7]

[1]Mayo Clinic College of Medicine, Rochester, MN; [2]Case Western Reserve University School of Medicine, Cleveland, OH; [3]Northwestern University, Chicago, IL; [4]Holston Valley Medical Center, Kingsport, TN; [5]David Geffen School of Medicine at UCLA, Los Angeles, CA; [6]Rush University Medical Center, Chicago, IL; [7]University of Minnesota, Minneapolis, MN.

Abstract: Standardized guidelines for polysomnography (PSG) have not specified methods for acquiring or interpreting electrocardiographic (ECG) data. The practice of single lead ECG monitoring during PSG may allow identification of simple measures of cardiac rhythm but reduces the ability to detect myocardial ischemia and to define cardiac intervals. Although simple measures of cardiac rhythm such as heart rate and cardiac pauses are inherently reliable, there is limited data regarding outcome measures relative to sleep related heart rates and cardiac events during sleep. Several observational and cross-sectional studies demonstrate that average heart rate drops nearly 50% from infancy through young adulthood and that the average heart rate slows during sleep compared with wakefulness; the definitions of sinus bradycardia and sinus tachycardia should therefore be lower during sleep than wakefulness. Asystoles of up to 2 seconds are seen in normal populations during sleep. Although there may be an increased risk of certain arrhythmias at night, particularly in sleep disordered breathing, there is no evidence that supports different definitions for these arrhythmias during sleep compared with wakefulness. When the quality of tracings permits, the standard definitions of narrow- and wide-complex tachycardias and atrial fibrillation may be employed. In the future, expansion to multiple ECG leads and the use of alternative tools may provide better definition of heart rates and cardiac events during sleep.

Keywords: Sleep, sleep disorders, electrocardiography, heart rate, arrhythmia, sinoatrial node, atrioventricular node, myocardial ischemia

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1.0 HISTORICAL PERSPECTIVE

Cardiac monitoring during polysomnography (PSG) has been largely based upon local tradition and resources, in both monitoring methods and reporting of cardiac variables on PSG reports. Detailed methods for acquiring or interpreting electrocardiographic (ECG) data are not currently incorporated into standardized guidelines for PSG,[1-4] nor are they included in sleep scoring by standardized Rechtschaffen and Kales methodology.[5] Despite this lack of standardized methods for ECG, cardiac events are commonly encountered during routine PSG, with pathologic cardiac events reported in 18%-48% of referred patients with obstructive sleep apnea.[6,7]

There has been increasing interest in cardiovascular aspects of sleep and sleep disorders, particularly in relation to obstructive sleep apnea (OSA) and central sleep apnea (CSA). A variety of newer technologies (continuous ambulatory blood pressure [BP] monitoring, peripheral arterial tonometry, measures of endothelial function) are being used as clinical research tools to explore complex sleep and cardiovascular relationships, but their role in patient care has yet to be defined. The single lead ECG appears as a virtual "standard" in the performance of clinical PSG, yet scoring and reporting for these data have not been standardized. Thus, there is a compelling need to develop a clinically applicable set of basic standards for monitoring and reporting cardiac variables during PSG. While this review of cardiovascular PSG scoring guidelines is limited to the information available from the single lead ECG, it is anticipated that future revisions will reflect the evolving complexity of cardiovascular monitoring during polysomnographic sleep studies.

2.0 METHODS

This review paper has been developed by the Cardiac Task Force (see page 153) of cardiologists and sleep medicine experts convened by the American Academy of Sleep Medicine for the purpose of developing standard scoring rules for cardiac events occurring during sleep. The paper focuses when possible on available evidence for reliability and validity of measurements, tempered by practicality of methods in the setting of routinely practiced PSG.

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Address correspondence to: Sean Caples, D.O., Mayo Clinic, 200 First Street SW, Rochester, MN 55905; Tel: (507)-255-8343; Fax: (507)-284-1161; caples.sean@mayo.edu

Table 1—Evidence Table

Evidence Levels	Study Design
I	Randomized well-designed trials with low-alpha & low-beta errors
II	Randomized trials with high-beta errors
III	Nonrandomized controlled or concurrent cohort studies
IV	Nonrandomized historical cohort studies
V	Case series

This will include a single channel ECG display and the limits imposed by this choice. The authors recognize that evolving technology and evidence will require future revisions of our positions. The scope of the review will be limited to methods and events in cardiac monitoring that are most relevant to current practices in PSG:

1. Choice of single ECG channel
2. Scoring bradycardia
3. Scoring asystole
4. Visual scoring of tachyarrhythmias, including wide and narrow complexes and atrial fibrillation

The search strategy was performed on Medline and Embase between 1966-2004 and restricted to English language publications and the following key words: sleep, sleep stages, sleep disorders, electrocardiography, heart rate, sinoatrial node, atrioventricular node, heart block, heart conduction system, arrhythmia, myocardial ischemia, cardiac pacing, cerebrovascular accident, pacemakers, cardiovascular agents.

The search strategy yielded 1,683 abstracts/articles, which were then screened by the committee members. Data were extracted from 285 papers found to be of potential importance relative to the Cardiac Task Force objectives. From these, the Task Force members further distilled the articles to include those that were found to have relevance for specific criteria defined a priori: 1) the validation of a single lead ECG and 2) normative data across age groups. Most of the articles were not found to be useful for development of scoring rules. Many published consensus statements in this topic are based on nonsystematic reviews or established practice and can therefore not be assigned an evidence grade. Evidence grading was modified from Sackett[8].

Only 14 articles pertinent to the reliability or validity of scoring rules were considered for evidence grading in this review and included observational cohorts, case series, or cross-sectional studies with level V evidence. (Table 1) Two additional data sets from previously published cross-sectional studies with level V evidence were used for discussion and consensus purposes. Two studies are cited which contain nonsystematic reviews and consensus statements. For the purposes of this review, the Cardiac Task Force 1) used evidence-based medicine to grade evidence and 2) developed a reference paper including background and evidence review that would serve as an annotated supporting text for developing rules and guiding consensus. This paper and the results of consensus balloting were then forwarded to the steering committee for drafting of cardiac scoring rules. As a result of limited evidence, scoring rules for cardiac events were derived largely by consensus. The Cardiac Task Force met by conference call 16 times between September 2004 and December 2005 to discuss evidence review and to complete RAND/UCLA Appropriateness Method consensus balloting.[14]

3.0 RELIABILITY AND VALIDITY OF TECHNICAL CONSIDERATIONS

ECG, the most commonly performed cardiac diagnostic test, is safe, simple, and reproducible.[9] In the case of heart rate determination for bradycardia and tachycardia, ECG is the standard of measurement and thus has inherent reliability and validity.

3.1.1 Electrocardiographic lead choice

Standards for ECG use, indications, and competency in interpretation in the ambulatory setting have been devised by the American Heart Association (AHA) and American College of Cardiology (ACC).[9-11] These recommendations were designed to guide use of 24- to 48-hour recorders, particularly multichannel Holter monitors. These recordings are considered the gold standard for the noninvasive diagnosis of rhythm and conduction disturbances, for which test sensitivity and specificity are high. Multiple-channel recordings are considered a requirement to accurately assess changes in ST and T tracings, which are limited in their specificity.[9] While a useful general reference, these recommendations do not comment on single channel monitoring or use of ECG during polysomnography.

In considering the goals of cardiac monitoring during PSG as currently practiced with the single lead ECG, we have placed primacy on determination of heart rate (HR), basic rhythm recognition (sinus or otherwise), and detection of ectopy.

3.1.2 Single electrocardiographic leads

In some cases, use of multi-lead Holter monitoring or even full 12 lead ECG was reported. Most papers did not specify lead placement, although it may be inferred that many utilized a single lead. Moreover, much of the data are derived from research exploring more advanced techniques of cardiac rhythm analysis such as heart rate variability and spectral analysis, QT dispersion, and signal averaging. The clinical importance of these measures during sleep is unclear.

Our literature review yielded scant data on the use of specific single ECG leads during sleep upon which to assess reliability and validity in detecting pathological events. Those papers that did specify a single lead cited the use of a variety of lead placements, including I, II, MCL1, V1, and V6. A limited number of references from the pediatric sleep literature have utilized single limb as well as precordial leads for compiling normative HR data.

3.1.3 Electrocardiographic lead placement

Classically, electrode placement for frontal-plane channels (I, II, III) during resting 12-lead ECG specifies the distal upper extremities. However, the advent of ambulatory and exercise ECG necessitated modification of electrode placement to more proximal sites, such as near bony prominences at the base of the upper limbs;[12] This placement is also well suited to the sleep lab environment where distal extremity electrodes are prone to dislodge. Ambulatory Holter ECG monitoring frequently utilizes 4 limb electrodes plus an additional chest electrode to allow monitoring of a precordial lead such as V1, V5, or V6. The use of modified chest lead 1 (MCL1) has been used as a bipolar alternative to V1, although it appears to be inferior to V1 at differentiating wide

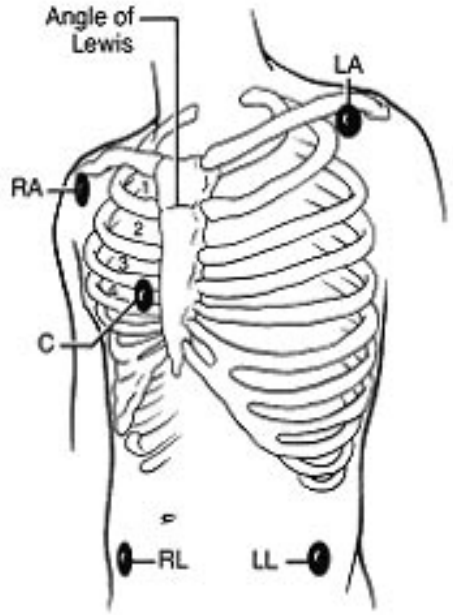

Figure 1—Four extremity electrodes (right and left arm, RA and LA, respectively plus right and left legs, RL and LL, respectively) allows recording of any of the six limb leads. An extra parasternal electrode here allows for recording of precordial lead V1. (Reprinted with permission from Lippincott Williams & Wilkins, Baltimore, MD. – LWW.com)

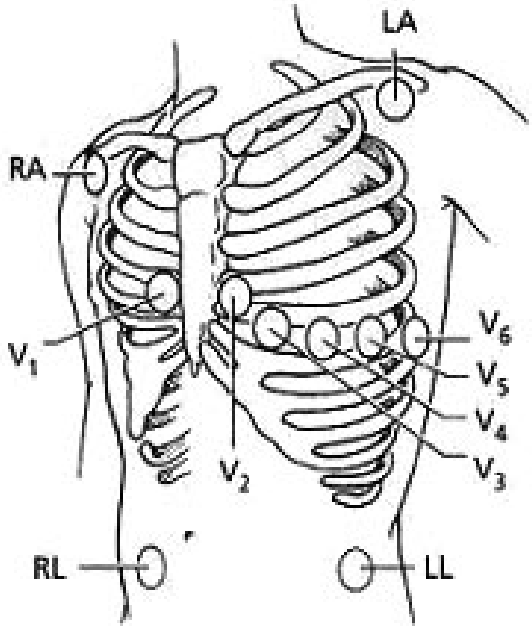

Figure 2—The Mason-Likar 12-lead ECG system includes anatomic locations for precordial leads identical to those used in a standard 12-lead ECG. The extremity electrodes are situated proximally for use in exercise tesing. (Reprinted with permission from Lippincott Williams & Wilkins, Baltimore, MD. – LWW.com)

complex tachycardias.[13]

Figures 1 and 2 demonstrate basic electrode position for commonly used ECG channels in ambulatory electrocardiography, for which practice standards have been published by the American Heart Association.[12] Because of the relative ease of electrode placement on the upper torso and reasonable utility at detecting basic (sinus) cardiac rhythm, the use of ECG bipolar leads I or II (or a modification of these) likely predominates across sleep labs, although systematic analyses to confirm this assumption do not exist. The Sleep Heart Health Study (SHHS), a cross-sectional study employing PSG monitoring in randomly selected cohorts,[27] used a modified lead II, although technicians had the option of placing V1 if the participant did not object to chest exposure. It is probable that leads other than I and II are utilized in some labs during routine PSG. Our literature search did not find convincing evidence of the superiority of one specific lead over another for determining basic rhythm or detecting atrial activity via P waves. The area of the chest encompassed by the bipolar leads, forming Einthoven's triangle[16] (Figure 3), may suggest a theoretic advantage of lead II, the electrical vectors of which traverse the atria.

The modern PSG routinely calls for electrode placement at both upper extremities (or bilaterally on the upper chest) for ECG leads and on the legs for evaluation of limb movements during sleep. These electrodes allow monitoring of any bipolar limb lead (I, II or III), which should adequately monitor HR and the presence of atrial activity.

With the transition of most sleep laboratories from analog to digital polysomnographic monitoring systems, it is possible to assess modified versions of other frontal plane bipolar limb leads in real time. While this approach may functionally provide additional ECG leads within the same patient, there is no evidence at present for its clinical utility.

3.1.4 Summary: lead choice and placement for PSG.

Based upon the simplicity of electrode placement as well as the potential advantage over other leads in determining basic cardiac rhythm (sinus vs. non-sinus), a single lead II using torso electrode placement is preferable for routine polysomnography. (Figure 4) Electrode position and lead may be adjusted or added at the practitioner's discretion to optimize signal and/or R wave voltage.

3.1.5 Limitations of single electrocardiographic leads—myocardial ischemia and origin of the wide QRS complex

A number of publications have reported on ST segment changes during sleep with OSA.[17-20] However, all reported utilization of multichannel ECG recordings that included at least one precordial lead. The validity of single lead monitoring in myocardial ischemia is poor. In one prospective study of 463 myocardial ischemic events comparing single-lead to 12-lead monitoring, the sensitivity of a single lead was 33% (95% CI, 29%-37%).[21] Myocardial ischemia is often regional and thereby vector dependent for detection on the surface lead electrocardiogram. Therefore, a number of practical factors render the sleep lab environment unsuitable for ST segment monitoring for myocardial ischemia.

Changes in body position, frequent and sometimes mandated during diagnostic PSG, are known to result in ST segment fluctuations,[22] thus rendering the tracing unreliable for inclusion or exclusion of ischemic changes. Because the amplitudes of clinically significant ST changes are as small as 1 mm, a degraded signal related to muscle or movement artifact would hinder reliability in assessing waveform changes. It would be anticipated, furthermore, that an excessive amount of technician time and effort would be spent tending to loose chest or limb electrodes. Finally, a consensus following a nonsystematic review (no evidence grade) by the American Heart Association (AHA) in 2004

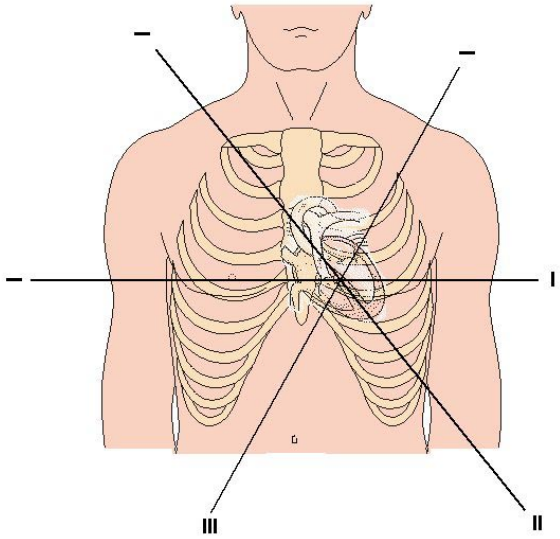

Figure 3—The vectors of the bipolar leads I, II and III form Einthoven's triangle. That of lead II (right arm negative to left leg positive) passes through the atria.

recommended more than one precordial lead to accurately detect ECG changes of acute myocardial ischemia.[23] Nevertheless, in the setting of chest pain occurring during polysomnographic monitoring, the ST segments or T waves may be used by a physician to supplement the clinical evaluation.

A further potential shortcoming of the single ECG channel relates to the widened QRS complex. There are limited data to suggest that limb leads without an accompanying precordial lead, such as V1, pose limitations for differentiating ventricular from supraventricular origin in evaluating wide complex tachycardias.[13]

3.1.6 Sampling Frequency

The American Heart Association has published recommendations for bandwidth (defined as the frequency range between low-frequency and high-frequency cutoffs) and sampling frequency in digital signal processing of the ECG. In 1990, the AHA recommended a minimum bandwidth of 125 Hz and, utilizing a rule of thumb of at least 2 to 3 times this value, a sampling frequency of 500 Hz.[24] Although a recent level V evidence paper reporting ECG sampling rates from a pediatric sample suggested inadequacy of these values and recommended a sampling frequency of 1000 Hz,[25] further evidence to support a frequency >500 Hz is currently lacking.

4.0 RELIABILITY AND VALIDITY OF DEFINITIONS

Validating threshold criteria for heart rate abnormalities during sleep requires comparison to heart rates associated with outcomes, and comparison to the range of normative data acquired during sleep. The Cardiac Task Force found that studies of cardiac rhythm and events during sleep were limited to case series and cross-sectional studies (level V evidence), and that outcome measures were seldom identified.

4.1.1 Bradycardia in adults

The lowest HR encountered in normal individuals over a 24-hour period is most likely to occur during NREM sleep, due to

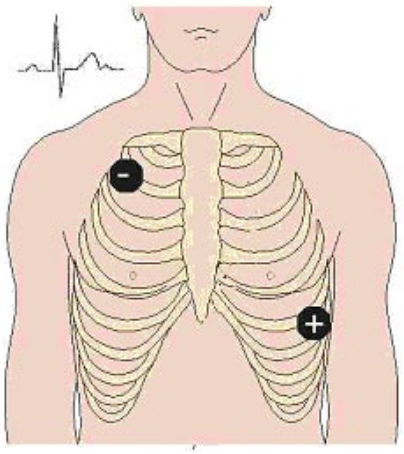

Figure 4—Electrode placement for the recommended ECG lead II. While classically placed on the right arm and left leg, the electrodes may be placed on the torso aligned in parallel to the right shoulder and left hip.

increase in parasympathetic tone and sympathetic withdrawal.[26]

Unpublished data from 2,067 adult subjects (1,448 females and 619 males, mean age 62.1 years) not on cardiac/antihypertensive medications who were found to have an apnea-hypopnea index <5 during unattended PSG in the SHHS were analyzed (evidence level V). Utilizing 95% confidence intervals, 2 standard deviations below the mean HR yielded a minimum normative value of 43 beats per minute (bpm) in men and 47.5 bpm in women.[28]

Other normative data on the lower limits of HR during sleep are derived from extended (12- to 24-hour or longer) electrocardiographic Holter recordings (level V evidence) where sleep is inferred, although not confirmed by PSG. Among the earliest descriptions of minimal nocturnal HR, Brodsky et al reported findings from 50 healthy young (23- to 27-year-old) males.[29] Heart rate mean was 43 bpm, ranging from 33 to 55 bpm. A minimum HR of <40 bpm was exhibited in 24%. More dramatic reductions in HR during sleep have been documented in endurance athletes (evidence level V).[30]

As in the SHHS data, females have consistently been shown to have higher average HR than men during sleep. Twenty-four hour Holter monitoring of 50 healthy young women (evidence level V) showed a mean minimum HR of 48 bpm (range 37-59).[31] Only 8% of the women demonstrated a minimum HR of <40 bpm.

4.1.2 Bradycardia in children

In childhood, heart rates change dramatically, decreasing almost 50% from infancy to young adulthood. While there are normative data for ECG-derived resting heart rates in children of different ages[32] (Table 2), there is a paucity of comparable sleeping heart rate data from which the lower limits of normal might be extrapolated. These limited data from sleep are summarized in Table 3.

Because vagal tone is often higher in children and adolescents, heart rates during sleep are expected to be significantly lower compared with resting wakefulness. For example, the mean resting heart rate during wakefulness for 8- to 11-year-old children (n = 233) has been reported to be 91 bpm (62 to 130 bpm for 2nd to 98th percentiles, respectively).[32] By comparison, slower sleeping heart

Table 2—Normal values for resting heart rate during wakefulness*

Age	Minimum	Maximum	Mean	2nd to 98th percentile	No. of Subjects
Under 1 day	88	168	123	93-154	189
1-2 days	57	170	123	91-159	179
3-6 days	87	166	129	91-166	181
1-3 weeks	96	188	148	107-182	119
1-2 months	114	204	149	121-179	112
3-5 months	101	188	141	106-186	109
6-11 months	100	176	134	109-169	138
1-2 years	68	165	119	89-151	191
3-4 yr	68	145	108	73-137	210
5-7 yr	60	139	100	65-133	226
8-11 yr	51	145	91	62-130	233
12-15 yr	51	133	85	60-119	247

*The above data showing normal resting average heart rate data limits were obtained from 12-lead ECGs in a study of 2,141 white children between birth and 16 years. Data were derived from computer assisted methods with a sampling rate of 333 samples per second, so there may be some underestimation of upper limits of normal (see reference 32).

rates were noted in unpublished data (level V evidence) extracted from a cross-sectional urban community-based cohort, Cleveland Children's Sleep and Health Study (CCSHS).[33] This study provided bipolar ECG data from 772 children (376 females and 396 males; mean age 9.5 years, range 8-11 years) without major comorbidities and with an apnea-hypopnea index of <5 during unattended limited channel overnight PSG. In the CCSHS sample, the mean heart rate during sleep was 73±10 bpm with significant sex differences—70±9 bpm in boys compared with 75±9 bpm in girls ($p < 0.0001$). This mean heart rate during sleep was almost 20% lower than the resting heart rate during wakefulness in the same aged sample. Utilizing 95% confidence intervals, 2 standard deviations below the mean yielded a sleeping value of 51 bpm in boys and 57 bpm for girls in the 8- to 11-year-old CCSHS sample.[33]

4.1.3 Bradycardia in the geriatric population

The reduction in HR associated with the state change from wakefulness to sleep has also been demonstrated in individuals >80 years of age, although the change is not as great as that encountered in younger subjects. In a cohort study (level V evidence) of 50 elderly subjects (44 female) without apparent cardiovascular disease, only 12% demonstrated nocturnal sinus arrhythmia (to include bradycardia).[34] The mean HR was 64 bpm, with a minimum recorded HR of 43 bpm. However, ectopic beats were frequent and the incidence of brief arrhythmias was significant, suggesting that normative data in this age group are difficult to interpret. In a cohort study with level V evidence, of 260 healthy subjects aged 40 to 79 years, only 3% demonstrated a minimum HR <40 bpm.[35] The effect of endurance training on HR may persist into older age, as demonstrated by a far more common incidence of HR <35 bpm seen in veteran athletes compared with age-matched controls.[36]

4.1.4 Bradycardia in sleep-related breathing disorders

Obstructive sleep apnea has been associated with a number of bradyarrhythmias, including severe slowing of HR; this may be sinus in origin or associated with atrioventricular block.[7,37-39] Bradycardia in OSA has been found to correlate with the degree of oxyhemoglobin desaturation[7,37] and to be more pronounced during REM sleep.[37,40,41] The American Heart Association/American College of Cardiology designates AV block associated with apneic events during sleep a Class III indication for a pacemaker (not useful/effective or may be harmful).[42]

4.1.5 Summary: bradycardia during PSG

The Cardiac Task Force felt that a lower threshold for bradycardia should be established during sleep as compared with wakefulness, but that there were not sufficient data to establish a different threshold in older children compared with adults. The Cardiac Task Force agreed that bradycardia during sleep should be defined in those 6 years and older as a sustained HR <40 bpm, based upon normative data from the SHHS and the Cleveland cohort, as well as other smaller studies.

4.2 Asystole

The normative data cited above (level V evidence)[29,31] regarding young healthy subjects found sinus pauses to be longer during sleep than wakefulness and more prolonged in males (mean 1.62 secs, range 1.20–2.06 sec) than in females (mean 1.47 secs, range 1.08-1.92 sec). Thirty-seven percent of trained athletes demonstrated sinus pauses between 2 and 3 seconds during sleep (evidence level V).[30] In Bjerregaard's cohort of 40- to 79-year-olds (level V evidence), the longest pause during sleep was 2.04 seconds, while the minority of 80+ year olds with bradycardia demonstrated sinus pauses from 1.8 to 2.0 secs.[35]

The influence of sleep stage on sinus pauses in otherwise healthy individuals was demonstrated by Guilleminault et al who described asystolic periods lasting up to 9 seconds during REM sleep.[43]

Clinical conditions commonly found in sleep laboratory referrals, such as severe OSA, have been associated with longer sinus pauses. Forty-three of 400 (11%) OSA patients in one study manifested asystole for 2.5 to 13 seconds (evidence level V).[7] It must be emphasized, however, that the reliability of the incidence of sinus pauses or bradyarrhythmias is constrained by limited recording times during these studies and interindividual variability from night to night (evidence level V).[38]

The 1998 guidelines of the American College of Cardiology and the American Heart Association for pacemaker implantation suggest that asymptomatic episodes of sinus bradycardia (with the heart rate as low as 30 bpm, sinus pauses ≤3 seconds, and type 1 second degree (Wenckebach) atrioventricular nodal block are not necessarily regarded as pathologic and may be considered to be within the normal range.[42] Similar recommendations were repeated in the 2002 revision, although some suggested that the lower "normal" HR limit should be raised to 35 or 40 bpm.[44] Guidelines for healthy children without congenital heart disease were not presented.

4.2.1 Summary: asystole during PSG

In normal adults, asystoles ≤2 seconds are observed during sleep. Asystoles ≤3 seconds during sleep are relatively common in athletes. AHA guidelines define asystoles of >3 seconds as pathologic in symptomatic individuals; this is consistent with the range observed during sleep in normal adults and athletic individuals. Asystoles >3 seconds may be observed in patients with

Table 3—Age specific heart rates during sleep in normal children over 1 year of age

Age (yr)	Mean bpm	SD	Median, Range	IQR	PSG	n	Author/ref/comment
1-1.5	114	11			N	88	Kelly/55
2			98 73-120		N	42	Poets/56/from 24-hr recordings
2-6			86, 69-118	79-93	N	33	Poets/57/ heart rate
7-11			75, 59-93	71-71		22	derived from pulse
12-16			68, 58-85	66-71		15	wave form, overnight
2-4	93	13			N	18	Finley/58/QS and
4-6	82	9				8	AS, not explained,
10-12	68	9				10	only QS shown
8-10	69	7.1			Y	14	Pivik/59 only males, no significant state differences
8-11	72.5	9.7	73 (40-112)		Y	772	Rosen/33
8.9±0.6	73	1			Y	7	Villa/60
10-13			median not provided 60-110 max 30-70 min		N	131	Scott/61/males only, 24-hr Holter recordings
13.4 mean (10-17.5)	69	N/A			Y	10	Ferri/62

Table abbreviations: AS = active sleep; IQR = interquartile range; N = no; N/A = not applicable or not available; OSA = obstructive sleep apnea; PSG = polysomnography; QS = quiet sleep; SD = standard deviation; Y = yes

sleep apnea.

4.3 Sinus tachycardia

Sinus tachycardia in adults has traditionally been defined as HR >100 bpm, although some have advocated lowering this threshold to 90 bpm during wakefulness.[45] Because of the autonomic changes that occur with sleep, applying similar ranges to HR during both sleep and wakefulness would be inappropriate. Data extracted from the SHHS cohort offers some insight into the upper limits of normal HR during sleep (unpublished data; evidence level V).[28] The upper limit of the 95% confidence interval for average heart rate was 84.7 bpm in women and 80.8 bpm in men. The importance of an increased HR of sinus nodal origin is traditionally thought to relate to extracardiac factors such as fever, metabolic disease, or psychogenic disorders. In the absence of such secondary causes, the significance of a supranormal HR during sleep, however defined, is not known.

Maximal normal HR with exercise in children has been estimated to be within 10% of the child's age subtracted from 220. Since this formula was designed to apply during wakefulness, maximal HR during sleep would be expected to be much lower, as in adults, due to mechanisms related to autonomic tone. Only scant data are available in pediatric populations to set the upper limits of normal during sleep at different ages (see Table 3). Data extracted from the CCSHS in 8- to 11-year-old children showed that 2 SD above the mean heart rate during sleep, utilizing 95% CI, yielded values of 94 bpm in girls and 89 bpm in boys. These values were almost 30% lower than the 98th percentile for a resting heart rate in wakefulness in the same sample (evidence level V).[32]

4.3.1 Summary: sinus tachycardia during PSG

Average heart rate is lower during sleep than wakefulness and higher in females than males during sleep. In adults, the Cardiac Task Force agreed that the threshold for defining sinus tachycardia during sleep should be defined as a sustained heart rate >90 bpm, based upon normative data from the SHHS. Sinus heart rate in children varies substantially by age.

4.4 Tachyarrhythmias other than sinus

Detailed definitions of and criteria for tachyarrhythmias are beyond the scope of this paper and can be referenced to the cardiac literature describing those encountered during wakefulness.[46,47] Atrial fibrillation is characterized by the replacement of P waves with rapid oscillations or waves that vary in size, shape, and timing, resulting in an irregularly irregular ventricular rhythm.[47] By standard convention, narrow-complex and wide-complex tachycardias can be differentiated by a 120 msec QRS duration. However, accurate determination of this interval may not be achievable from a single lead ECG obtained during PSG, thereby limiting the reliability of this interval definition.

Much of the literature on rhythm disturbances during sleep in adults originates from the study of sample populations that include participants or patients with OSA and/or CSA. The occurrence of cardiac ectopy and tachyarrhythmias, including supraventricular tachycardia and atrial fibrillation/flutter, is frequent in those with OSA.[7,48-50] A circadian variation in the occurrence of sudden cardiac death has been demonstrated in a group of patients with OSA with the highest risk during the nighttime.[15] While the occurrence of arrhythmias may relate to the prevalence of underlying cardiac disease in these patients, there is some evidence that effective therapy of underlying sleep disordered breathing may reduce the occurrence of arrhythmias.[51-53]

Evidence describing dysrhythmias related to sleep independent of disordered breathing events is scant. There are some data (evidence level V) showing a circadian rhythm for ventricular fibrillation in the Brugada syndrome, manifesting as increased arrhythmias and risk of death during the nighttime hours.[54]

4.4.1 Summary: tachyarrhythmias other than sinus

The Cardiac Task Force agreed that conventional definitions for abnormal tachycardias could be modified for the purposes of interpretation during PSG. A wide complex tachycardia is a sustained rhythm lasting more than 3 cardiac cycles with a QRS duration ≥120 ms and a rate >100 bpm. A narrow complex tachycardia is a sustained rhythm lasting more than 3 cardiac cycles with a QRS duration <120 ms and a rate >100 bpm. Atrial fibrillation is an irregularly irregular ventricular rhythm associated with the replacement of P waves with rapid oscillations or waves that vary in size, shape, and timing. This definition of atrial fibrillation is consistent with AHA guidelines.

5.0 UNRESOLVED AREAS AND FUTURE RESEARCH

Technical advances in PSG currently allow more complex monitoring than was available at the time of Rechtschaffen and Kales. The capability and burden of expanding ECG monitoring to include multiple ECG leads during PSG for the better definition of myocardial ischemia and arrhythmias should be considered, especially for targeted populations at risk for cardiac events. Blood pressure monitoring and indirect measures of sympathetic tone and cardiovascular function may enrich the cardiovascular information once these devices have demonstrated validity, reliability, and practical application.

CARDIAC TASK FORCE MEMBERS

The Cardiac Task Force members participating in consensus decisions to derive scoring rules included: Sean M. Caples, co-chair, Virend K. Somers, co-chair, Michael E. Adams, William G. Cotts, Parvin Dorostkar, Thomas Kara, Tim Morgenthaler, Carol L. Rosen, Edward J. Stepanski, Win K. Shen, Kalyanan Shivkumar, Apoor S. Gami, and Conrad Iber.

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