International Journal of Research in Medical Sciences
Chakrabarty M et al. Int J Res Med Sci. 2025 Oct;13(10):4551-4558
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20253219

Review Article

The usefulness of various subjective and objective measures of circadian phase in sleep medicine diagnostics

Mousumi Chakrabarty[1]*, Vivekananda Lahan[2]

[1]Department of Neurophysiology and Sleep Medicine, GNRC Hospitals, Guwahati, Assam, India
[2]Department of Psychiatry and Sleep Medicine, GNRC Hospitals, Guwahati, Assam, India

Received: 28 July 2025
Accepted: 05 September 2025

***Correspondence:**
Dr. Mousumi Chakrabarty,
E-mail: mcmousumi94@gmail.com

ABSTRACT

Circadian rhythm sleep disorders (CRSDs)can be difficult to diagnose as they usually present with a wide array of complaints ranging from insomnia to excessive sleepiness or impairment in the quality of life. Malalignment of the endogenous circadian clock to the external day-night cycle leads to these disorders. There are various subjective and objective methods for assessing these disorders. Both individual subjective and objective measures of circadian phase have their own merits and limitations and a combination of both- especially sleep logs and questionnaires along with actigraphy usually suffice for diagnosing most cases. Dim light melatonin onset (DLMO) and core body temperature (CBT) minimum are also very useful in some cases. Recent advances in the knowledge of neurobiology and genetics of the circadian rhythm should hopefully lead us to improved diagnostic tools and provide us with evidence base practical guidelines for diagnosis and treatment of CRSDs.

Keywords: CRSDs, Circadian clock, Zeitgebers, Subjective and objective measures

INTRODUCTION

Among the various sleep disorders which present in a sleep clinic, Circadian rhythm sleep disorders (CRSDs) form a substantial percentage and need assessment of sleep. These disorders result primarily from a desynchrony between the exogenous light-dark cycle and the endogenous biological clock. The endogenous clock regulates body temperature, levels of different hormones such as Growth hormone, Prolactin, Cortisol, Melatonin, glucose, Insulin etc. and primarily the sleep-wake cycle. Each cycle of this endogenous clock is a little more than 24 hours. The circadian pacemaker or SCN is set by some external cues also known as 'zeitgebers' , of which light is primary. Light entrains the 'internal clock' to the external environment, to a 24 hours rhythm. According to the international classification of sleep disorders (ICSD-3), "the essential feature of CRSDs is a chronic or recurrent pattern of sleep-wake rhythm disruption primarily caused by an alteration in the endogenous circadian timing system or misalignment between the endogenous circadian rhythm and the sleep-wake schedule desired or required".[1] The ICSD-3 classifies CRSDs into six types, namely, delayed sleep phase disorder (DSPD), Advanced sleep phase disorder (ASPD), Irregular sleep-wake phase disorder (ISWD), Free-running disorder (FRD), Jet-lag disorder (JLD), Shift-work disorder (SWD), Circadian sleep-wake disorder not otherwise specified. CRSDs can be difficult to diagnose as they usually present with complaints of insomnia or excessive sleepiness or impairment in the quality of life. In such patients, the circadian phase needs to be determined to correctly diagnose the cause, so that proper treatment which is also appropriately timed can be administered. Circadian phase can be assessed by subjective as well as objective methods. The subjective methods are the various questionnaires available as the MEQ. (Morningness-eveningness questionnaire) and the MCTQ (Munich chronotype questionnaire), sleep logs or diaries. These are self-assessment tools which the patients or an observer (e.g., relative) are asked to fill up. The objective measures include actigraphy, the circadian phase markers as

melatonin. (Salivary, plasma or urinary 6 suphatoxymelatonin), Dim light melatonin onset (DLMO), core body temperature (CBT), serum cortisol and the 'gold standard' of sleep diagnostics -polysomnography (PSG). Before going into detailed analysis of the usefulness and limitations of these methods, the initial part of this essay will discuss concisely the core concepts of (a) the circadian system, which is one of the two main determinants of our sleep-wake cycle; and (b) the different types of CRSDs are concisely discussed below.

THE CIRCADIAN SYSTEM

The word 'circadian' denotes a full cycle of 24 hours.[3] If the cycle is denoted by a waveform, then 'phase' denotes the position in time on the waveform. There is a master-clock situated in the SCN of the hypothalamus. The cells of this region show near 24-hour oscillations individually. The coordinated output of these individual clocks generates the circadian rhythm which in turn control timing of many physiological, behavioural and metabolic functions, including that of sleep.[2] Studies on Drosophila, mutant mice and hamsters finally led to the discovery of the 'clock genes' which generate the circadian rhythm. The two proteins BMAL1 and CLOCK regulate transcription and production of 'Per' and 'Cry' proteins. These proteins then form a complex in the cytoplasm and enter the nucleus. When the concentration of this complex rises in the nucleus, they inhibit the CLOCK-BMAL1 mediated transcription. The PER/CRY complex is then degraded which stimulates CLOCK-BMAL1 to restart transcription. This cyclical phenomenon occurs every 24 hours.

Further studies led to the discovery that such clocks also exist in peripheral tissues, the activity of all of which is synchronized by the master clock through neuronal and chemical signals. The peripheral clocks send feedback to the SCN which integrates the information and resets the circadian rhythm. The biological clock is influenced by some external cues of which light is the principal one. When light falls on the eye, signals are sent via the Retin hypothalamic tract to the SCN. This leads to release of the neurotransmitter PACAP (Pituitary adenylate cyclase-activating polypeptide) and glutamate and activates a cascade, ultimately leading to the synthesis of PER1 and PER2 proteins. Increased levels of PER 1 and 2, supress CLOCK/BMAL1 activity. In this way light entrains activity of the clock. A desynchrony between this endogenous clock and external environment gives rise to the various CRSDs.

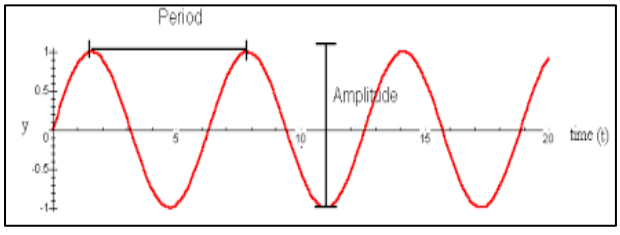

Figure 1: Circadian rhythm.

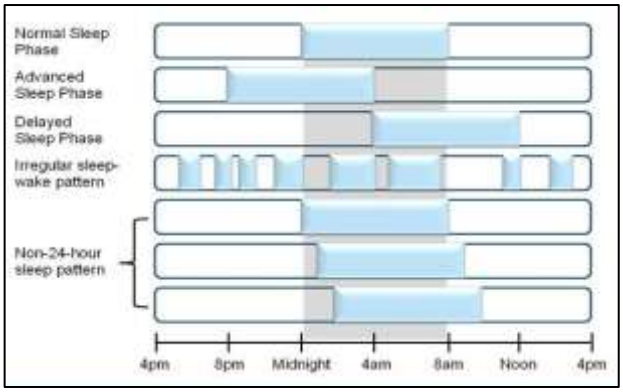

Figure 2: Circadian rhythm sleep disorders.

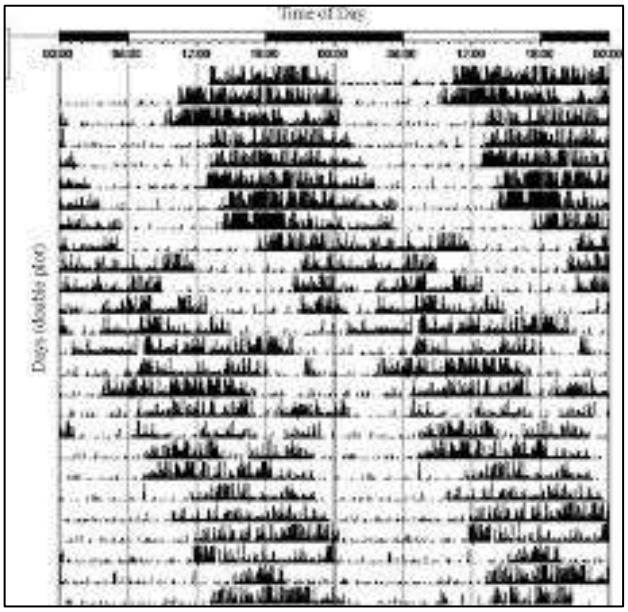

Figure 3: Actigraphy in free running rhythm.

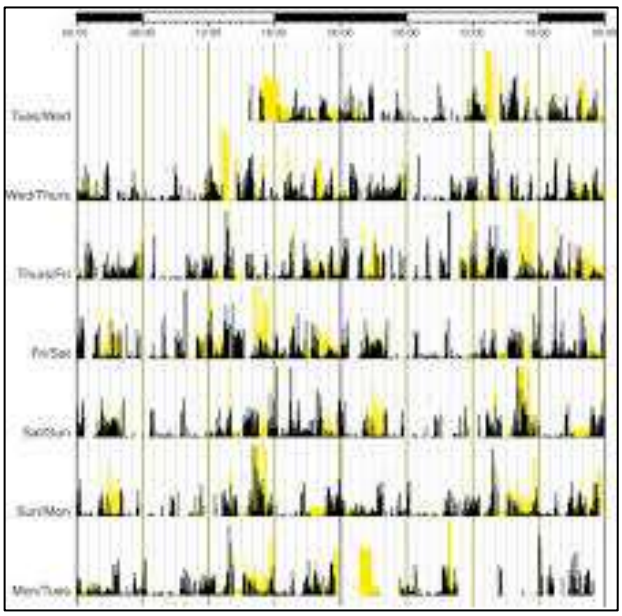

Figure 4: Irregular sleep-wake rhythm.

CIRCADIAN RHYTHM SLEEP DISORDERS

As already mentioned, the ICSD 2 describes seven types of CRSDs, which are as follows.

Advanced sleep phase disorder

This is characterized by a regular sleep schedule, but which is several hours ahead of the normal or conventional sleep time. It is generally seen as age advances, although a definite genetic link has also been described.[4,5] Sleep logs and actigraphy have been found to be useful tools for assessing ASPD.[6-8] PSG maybe necessary to rule out other causes of insomnia.

Delayed sleep phase disorder

This is characterized by a stable sleep schedule, but which is several hours later than the conventional time. They have difficulty in initiating sleep (sleep onset insomnia), as well as extreme difficulty in rising at conventional times. It is generally seen in adolescents, although there could be other factors responsible for the phase delay such as sensitivity to evening light, a diminished ability to compensate for sleep deprivation or a masking effect.[9-14] Assessment tools include primarily actigraphy and sleep logs, although salivary DLMO (sDLMO) may also be estimated for diagnosis.[7,15] PSG done twice- one on a week day and one at an weekend can be conclusive.[16] Multiple sleep latency tests (MSLTs) is also useful to diagnose daytime sleepiness which is one of the presenting symptoms.

Irregular sleep-wake rhythm

ISWR is characterized by an absence of a definite pattern in the sleep-wake cycle. They have frequent episodes of short bouts of sleep distributed throughout the day and night very irregularly and present with neuropsychiatric disorders as mental retardation in children and dementia in elderly.[6] Damage to the SCN is a likely cause.[17] Assessment is mainly based on sleep logs and actigraphy.[18] CBT, Melatonin levels have a limited role in diagnosis of ISWR.

Free-running disorder or non-24-hour sleep-wake syndrome

The free- running rhythm is seen in people who are totally blind and fail to entrain to the light-dark cycle.[19] The patients experience several weeks of good, consolidated sleep, followed by several weeks of poor night-time sleep, daytime sleepiness and napping.

Onset is usually around teenage years in sighted patients, but variable in totally blind people.[19] Assessment tools are sleep logs or diaries and actigraphy. Multiple serial determinations of DLMO and CBT may have a restricted role.

Jet lag disorder

It is a disorder which occurs due temporary desynchrony between the internal circadian rhythm and the light-dark cycle as a result of long-distance travel. The adaptation of the circadian system to the new destination takes few days and depends on the number and direction of time zones crossed. The patients present with symptoms of insomnia, fatigue, cognitive dysfunctions, gastrointestinal problems etc. They can be assessed by Columbian Jet Lag Scale, estimation of Phase markers such as CBT, Melatonin levels, Growth hormone, Cortisol levels etc. PSG and actigraphy have very limited role.[20,21]

Shift work disorder

Shift work is a work schedule in which at least 50% of work is required to be done outside of the standard 'day - work-hours'. It maybe early morning starts, late shift finishes, evening shifts or night shifts.[22] The patients experience sleep disturbances, fatigue, cognitive and memory dysfunctions, gastro-intestinal problems, greater risk of accidents, cardiovascular diseases, diabetes and even some types of cancer. Assessment can be done by sleep logs or diaries and actigraphy.

The main section of the essay will discuss in detail, the various subjective and objective assessment tools of circadian phase and their relative merits and limitations in sleep medicine diagnostics.

Subjective assessment tools

Circadian phase can be assessed subjectively using MEQs, MCTQ and sleep logs or diaries.

Role of MEQ

The MEQ was developed by Horne and Ostberg in 1976. This self-assessment tool helps to determine what time of the day a person is productive and when is the best time for him to sleep. It contains a series of 19 MCQs each having 6 choices with scores from 0-5. Total scores range from 16-86 with lower values from 16-30 designated 'definite evening types', 31-41 designated 'moderate evening types', 42-58 as 'intermediate types', 59-69 as 'moderate morning types' and 70-86 as 'definite morning types'.

Several research studies have utilized the MEQ as a principal assessment tool to detect morningness or eveningness. In a study by Christopher R. Jones et al, in 1999, 29 persons in a family were identified to have ASPD and 46 were declared to be unaffected.[5] They used the HO questionnaire as a primary assessment tool to categorize them into 'morning types or 'evening types'. The questionnaire findings correlated well with PSG, actigraphy and circadian phase markers findings. The affected family members in their study scored highly in the

MEQ, thus validating the usefulness of HO questionnaire in diagnosis of ASPD.

In another study by Satoh et al, Mishima et al, in Japan in 2003 on two families in Japan, the association of Per 2 gene mutation with familial ASPD was examined.[8] The HO questionnaire was used to detect the affected members. Though the MEQ scores were more than 70 in affected subjects, there was no significant advance shift of sDLMO. Their findings showed that it is difficult to diagnose ASPD patients using currently available tools. Further studies in larger population are needed and MEQ scoring criteria for younger persons has to be determined.

A study by Palmer et al tested the effectiveness of evening light therapy on ASPD patients.[23] The researchers confirmed that MEQ scores of 91% of their subjects correlated with 'Morningness' (i.e. greater than or equal to 59) and 53% with 'definite morning types' i.e. MEQ greater than or equal to 70. These findings suggest that though MEQ can provide confirmatory evidence for ASPD diagnosis, it cannot be the sole diagnostic criteria.

There is insufficient evidence for the role of MEQs in diagnosis of DSPD, FRD, ISWR, SWD or JLD.[6] A study on DSPD patients and Extreme Diurnal Preference subjects showed association of length polymorphism on Per3 gene with DSPD and Extreme Diurnal Preference.[28] Diurnal preference of the study subjects and DSPD was determined using the Horne-Ostberg Questionnaire. Their study identified a potential genetic marker for patients with DSPD.

A study on NASA shift workers by Stewart K T et al, in 1995, showed that MEQs have little predictive value in assessing adaptability to shift work.[24] They evaluated the effect of exposure to light or darkness on phase shifting. Further research is needed before using MEQ scores as a valuable tool for assessing adaptability to night shift work, they concluded.

Role of MCTQ

The munich chronotype questionnaire is another self-assessment tool, but determines the actual sleep timings to diagnose chronotypes. It also addresses free and work days separately. When validated against MEQ, it shows good correlation.[25] It was developed by Roenneberg in 2003 and has 14 questions. Chronotype is estimated by subtracting 'midpoint of sleep on workdays' (MSW) from 'midpoint of sleep on free days' (MSF). In a study by Marc Wittmann et al, in 2006 on social jetlag, how individual chronotypes affect sleep quality and mental wellbeing was determined.[26] 501 subjects were asked to fill up the MCTQ and the correlation between chronotype, sleep quality, addiction to stimulants and psycho-social wellbeing was estimated. The study subjects were from a wide age group range. They stated that the sleep corrected mid-sleep on free days (MSFsc) is an excellent predictor for chronotypes.

Another study by Thomas Kantermann et al compared MEQ and MCTQ to DLMO scores to evaluate effectiveness of the questionnaires in determining the circadian phase.[27] They concluded that DLMO correlated significantly with both MEQ scores and MSFsc. The strongest predictor of DLMO was MSFsc they stated. However none of the questionnaires can be used exclusively to aid timing of light or melatonin therapy. Thus, they concluded, if given a choice MCTQ is better than MEQ.

Role of sleep logs/diaries

Sleep logs and diaries are useful tools for diagnosis of CRSD. However there are no standardized formats resulting in wide variability between diaries used by clinicians across the globe. They can assess sleep both qualitatively and quantitatively. Sleep logs are very useful for documenting sleep patterns in patients with FRDs.

In a study conducted by Lockley in 1999, a comparison between the subjective and actigraphy measurement of sleep and sleep rhythms was done.[29] They reported several inconsistencies in the sleep assessment by the two methods- diaries and actigraphy. They opined that sleep and nap diaries are necessary to interpret actigraphy data which often over estimates nap and total sleep times. Their results showed that sleep onset latency was ,much less by actigraphy as compared to sleep logs. As in diaries subjects are asked to note 'bedtime' and 'time when they tried to fall asleep', so actual time spent in sleep can be separated from total time spent in bed. Similarly nap timings are also overestimated by actigraphy. So simultaneous use of diaries to correct the actigraphy data will help in accurate diagnosis, they concluded.

In a clinical trial by Czeisler et al in 2005, electronic sleep diaries were used to assess SWD.[30] They used e-diary to asses sleepiness during- a) their night shift work b) during their home after the night shift and also c) sleep efficiency during daytime after the nightshift work. Such a technique if applied in everyday clinical practice could be very useful to detect symptoms of SWD. Thus sleep diaries undoubtedly have a definite role in diagnosis and treatment of almost all CRSDs.

Objective assessment tools

These include a) actigraphy b) circadian phase markers which include DLMO, aMT6s, melatonin levels, CBT measurement and c) 'the gold standard'-PSG.

Role of actigraphy

Actigraphy is a procedure in which bodily movements, especially limb movements are recorded over a period of days to weeks by a device which can be worn usually on wrist or ankle and the data integrated to estimate sleep and wakefulness. It can be used to estimate sleep latency (SL), total sleep time (TST), wake after sleep onset (WASO) and

sleep efficiency (SE=TST/Time in bed).[31] However the sleep architecture or the respiratory events cannot be estimated. Actigraphy has been shown in numerous studies to be a useful tool in diagnosis of ASPD/DSPD, non-24-hour sleep disorder and SWD.

In a study Lockley et al subjective and actigraphy measurement of sleep in 49 blind subjects with varied circadian rhythms was done.[29] They obtained good correlations between subjective and actigraphy data in relation to sleep onset, offset, night sleep and daytime nap duration. There was also good correlation between the methods when compared to aMT6s and sleep changes in both normal and blind subjects.

A study by Einstein et al in 1999 on 322 subjects, it was opined that actigraphy monitoring was indispensable for diagnosis of CRSDs.[10] They also strongly advocated that actigraphy should replace PSG for diagnosis and monitoring prognosis of CRSDs. As CRSDs require study of at least a few days of sleep-wake pattern of subjects, it cannot be accomplished by PSG. It is only more practical to use actigraphy instead. Moreover actigraphy monitoring should be done during 'free running like' conditions, when person is not duty bound or socially restricted such as during vacations which is necessary for correct diagnosis.

In a study by Pickering et al on patients of Craniopharyngioma and healthy controls, sleep onset and offset and melatonin secretion was assessed using sleep logs and actigraphy.[32] Their study showed that sleep onset time is slightly delayed as assessed by sleep logs in comparison to actigraphy. Sleep offset time was also comparable by both the methods. However the quality of evidence was poor due to the small sample size.

In another study done in a hospital setting on liver cirrhosis patients, sleep and circadian rhythm was evaluated using actigraphy and sleep logs.[33] The study also showed good correlation between the two methods as regards to sleep onset and offset. Actigraphy is quite acceptable to patients, can be used for extended periods and though slightly more expensive than sleep logs, is much cost-effective as compared to PSG. Moreover it can be used in home settings and therefore patient compliance as compared to PSG is definitely more. In case of children with CRSDs, actigraphy is more sensitive at detecting sleep loss.[31]

Role of circadian phase markers

These include Melatonin levels- salivary, plasma, urine. DLMO (dim light melatonin onset), urinary aMT6s (a urinary metabolite of melatonin), CBT and cortisol secretion.

Some useful indices of the phase markers are time of onset and offset. Time at which it reaches lowest point (nadir). Time at which it peaks (acrophase). Area under the curve which is the total secretion in 24 hours (e.g. cortisol, melatonin). Amplitude-halfway between peak and nadir. Phase relationships between various markers Phase angle-time of endogenous to exogenous phase (e.g., light off to melatonin onset). Phase response curve-relationship between stimulus (light, melatonin) and shift in the circadian rhythm.

There are various determinants of the circadian cycle. These include melatonin secretion and phase and amplitude of CBT. Plasma, salivary levels of melatonin or urinary metabolites of melatonin aMT6s can be estimated by immunoassays. Again, melatonin secretion starts in the dark, so onset of secretion provides a clue to the circadian phase (DLMO). The CBT dips at night and reaches its nadir in the early hours of morning at around 5 am. However due to the masking effects, CBT may be misinterpreted and therefore needs to be measured in the laboratory under 'constant routine protocol'.[34] In clinical practice, salivary DLMO and urinary aMT6s are preferred if at all necessary.

In a publication by Lewy et al in 1989, usefulness of DLMO as a marker of circadian phase was determined.[35] It was concluded that measurement of plasma melatonin provides useful information about circadian phase when obtained under dim light conditions and can even be clinically useful. Phase shifting effects of bright light therapy can also be monitored using DLMO, they observed.

Another study by Wyatt et al in 2016 on diagnosed cases of DSPD on young adults and equal number of controls used sDLMO as a predictor of circadian phase.[36] They recommended the collection of saliva samples for DLMO under controlled conditions as 'gold standard' for correctly interpreting the circadian phase of patients with suspected DSPD.

In a study in 2003 by Palmer et al, efficacy of evening light was ascertained in elderly patients with ASPD. HO-MEQ, sleep diaries, actigraphy and urinary aMT6s was used for the study.[37] As commented by Sack et al, in an AASM review article, 'serial phase determinations using melatonin onset can define FRD with a very high level of diagnostic consensus'.[6]

Role of polysomnography

PSG is multiparametric study as the name suggests and regarded as the 'gold standard' for sleep medicine diagnostics. The different parameters monitored are EEG, EOG, EMG, ECG, respiratory parameters as airflow, chest and abdominal movements and pulse oximetry. It is the only test with the aid of which sleep staging can be done. PSG is not directly useful in diagnosing CRSD, but may be used to diagnose other associated sleep disorders.

In a study Jones et al on Familial ASPD, PSG was used to demonstrate advancement of sleep onset and offset and preservation of sleep quality and duration.[5] However in case of patients with DSPD, two PSGs-one to simulate

'workday' and another to simulate 'free day' could help to prove increased TST and preserved sleep architecture as shown in a study by Thorpy et al.[16] Thus though this could provide an ideal clinical tool for assessment of such patients, financial constraints could be a limitation. Another study by Khusida et al compared use of actigraphy, PSG and subjective methods for sleep assessment in sleep disordered patients.[38] TST and SE data from PSG, actigraphy and subjective measures were quite comparable. They recommended that along with actigraphy, subjective data could be useful in estimating TST and SE.

Though several studies on Alzheimer's disease patients have used PSG, it has been shown that it is not necessary for patients with ISWR disorders. It is also not indicated for diagnosis of FRD, SWD or JLD patients as a clinical tool. However, it can be used to rule out other sleep disorders or for research purposes.[6]

The Circadian rhythm is generated by the pacemaker located in the SCN of the hypothalamus and entrained via 'external zeitgebers' or cues of which light is primary. Molecular studies have revealed the presence of circadian clock gene proteins, levels of which oscillate by a feedback loop and generate a near 24 hours rhythm.

Malalignment of the endogenous clock to the external day-night cycle leads to CRSDs. They can be easily misdiagnosed as their complaints are atypical. The Core Consensus diary is a very useful tool for assessing almost all types of circadian rhythm. Several studies have documented the usefulness of sleep logs specially in FRDs and SWDs.[29,30] They are inexpensive, simple, can be used in home settings and can give a very valuable sleep history of the patient. Thus it helps the clinician to arrive at a baseline diagnosis in patients who present with vague complaints of insomnia, excessive daytime sleepiness or social jetlag, all of which may actually be due to circadian rhythm disruption. However the sleep logs can be quite misleading in patients who have a sleep state misperception. In clinical practice therefore it becomes necessary to supplement sleep logs with objective measurements of sleep such as actigraphy.

The MEQ, MCTQ are also self-assessment tools recommended by several studies for diagnosing CRSD.[5,8,23,25-27] The MCTQ, where MSFsc can be measured, is a better predictor of chronotype. However, none of these questionnaires can be used as the sole diagnostic tool and have to be combined with other objective measures. Among the objective measures of circadian phase, actigraphy is the most acceptable to patients. The pattern of sleep can be obtained which is very essential in CRSDs. The costs are much less than PSGs and so can be repeated to determine the treatment response. Actigraphy helps to distinguish normal delayed but stable pattern seen in adolescents from DSDP patients. Along with sleep logs, it also helps in diagnosis of ASPD, FRD and SWD.

Circadian phase markers provide a clue to the endogenous circadian phase of a person. DLMO is a useful marker for FRD when multiple serial estimations separated by at least a week are done. Phase delay in CBT maybe of importance in diagnosing ISWR. The key circadian phase markers are DLMO and CBT minimum. In clinical practice, monitoring of phase shifting in response to melatonin administration or phototherapy is useful to monitor treatment response. The role of PSG in diagnosis of circadian phase is limited as the sleep pattern cannot be estimated by a single night's study. It is only of value to rule out comorbidities such as sleep apnoea.

CONCLUSION

To conclude, though both individual subjective and objective measures of circadian phase have their own merits and limitations in clinical diagnostics, a combination of both especially sleep logs and questionnaires along with actigraphy usually suffice for diagnosing most cases. Phase markers such as DLMO and CBT minimum are also very useful in some cases. Recent advances in the knowledge of neurobiology and genetics of the circadian rhythm should hopefully lead us to improved diagnostic tools and provide us with evidence base practical guidelines for diagnosis and treatment of CRSDs.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Sateia MJ. International classification of sleep disorders. Chest. 2014;146(5):1387-94.
2. Lockley SW, Foster RG. Sleep: A very short introduction. Oxford University Press. 2012.
3. Wulff K. Chronobiology: Biological rhythms that influence sleep. Sleep. Multi-professional perspectives. London: Jessica Kingsley Publishers. 2012:41-67.
4. Carrier J, Monk TH, Buysse DJ, Kupfer DJ. Sleep and morningness-eveningness in the 'middle'years of life (20–59y). J Sleep Res. 1997;6(4):230-7.
5. Jones CR, Campbell SS, Zone SE, Cooper F, DeSano A, Murphy PJ, et al. Familial advanced sleep-phase syndrome: A short-period circadian rhythm variant in humans. Nat Med. 1999;5(9):1062-5.
6. Sack RL, Auckley D, Auger RR, Carskadon MA, Wright KP, Vitiello MV, et al. Circadian rhythm sleep disorders: part II, advanced sleep phase disorder, delayed sleep phase disorder, free-running disorder, and irregular sleep-wake rhythm. Sleep. 2007;30(11):1484-501.
7. Sadeh A. The role and validity of actigraphy in sleep medicine: an update. Sleep Med Rev. 2011;15(4):259-67.

8. Satoh K, Mishima K, Inoue Y, Ebisawa T, Shimizu T. Two pedigrees of familial advanced sleep phase syndrome in Japan. Sleep. 2003;26(4):416-7.
9. Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. Sleep. 1998;21(8):871-81.
10. Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. Sleep. 1998;21(8):871-81.
11. Dagan Y, Stein D, Steinbock M, Yovel I, Hallis D. Frequency of delayed sleep phase syndrome among hospitalized adolescent psychiatric patients. J Psychos Res. 1998;45(1):15-20.
12. Aoki H, Ozeki Y, Yamada N. Hypersensitivity of melatonin suppression in response to light in patients with delayed sleep phase syndrome. Chronobiol Int. 2001;18(2):263-71.
13. Ozaki S, Uchiyama M, Shirakawa S, Okawa M. Prolonged interval from body temperature nadir to sleep offset in patients with delayed sleep phase syndrome. Sleep. 1996;19(1):36-40.
14. Uchiyama M, Okawa M, Shibui K, Liu X, Hayakawa T, Kamei Y, et al. Poor compensatory function for sleep loss as a pathogenic factor in patients with delayed sleep phase syndrome. Sleep. 2000;23(4):553-8.
15. Wyatt JK, Stepanski EJ, Kirkby J. Circadian phase in delayed sleep phase syndrome: predictors and temporal stability across multiple assessments. Sleep. 2006;29(8):1075-80.
16. Thorpy MJ, Korman E, Spielman AJ, Glovinsky PB. Delayed sleep phase syndrome in adolescents. J Adoles Health Care. 1988;9(1):22-7.
17. Mistlberger RE. Circadian regulation of sleep-in mammals: role of the suprachiasmatic nucleus. Brain Res Rev. 2005;49(3):429-54.
18. Bliwise DL, Hughes M, McMahon PM, Kutner N. Observed sleep/wakefulness and severity of dementia in an Alzheimer's disease special care unit. J Gerontol Series Biol Sci Med Sci. 1995;50(6):303-6.
19. Sack RL, Lewy AJ, Blood ML, Keith LD, Nakagawa HI. Circadian rhythm abnormalities in totally blind people: incidence and clinical significance. J Clin Endocrinol Metabol. 1992;75(1):127-34.
20. Spitzer RL, Terman M, Williams JB, Terman JS, Malt UF, Singer F, Lewy AJ. Jet lag: clinical features, validation of a new syndrome-specific scale, and lack of response to melatonin in a randomized, double-blind trial. American J Psych. 1999;156(9):1392-6.
21. Sack RL, Auckley D, Auger RR, Carskadon MA, Wright KP, Vitiello MV, et al. Circadian rhythm sleep disorders: part I, basic principles, shift work and jet lag disorders. Sleep. 2007;30(11):1460-83.
22. Hossain JL, Reinish LW, Heslegrave RJ. Subjective and objective evaluation of sleep and performance in daytime versus nighttime sleep in extended-hours shift-workers at an underground mine. J Occup Environment Med. 2017;46(3):212-26.
23. Palmer CR, Kripke DF, Savage Jr HC, Cindrich LA, Loving RT, Elliott JA. Efficacy of enhanced evening light for advanced sleep phase syndrome. Behav Sleep Med. 2003;1(4):213-26.
24. Stewart KT, Hayes BC, Eastman CI. Light treatment for NASA shiftworkers. Chronobiol Internat. 1995;12(2):141-51.
25. Zavada A, Gordijn MC, Beersma DG, Daan S, Roenneberg T. Comparison of the Munich Chronotype Questionnaire with the Horne-Östberg's morningness-eveningness score. Chronobiol Int. 2005;22(2):267-78.
26. Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. Chronobiology Int. 2006;23(1):497-509.
27. Kantermann T, Sung H, Burgess HJ. Comparing the morningness-eveningness questionnaire and munich chronotype questionnaire to the dim light melatonin onset. J Biol Rhyt. 2015;30(5):449-53.
28. Archer SN, Robilliard DL, Skene DJ, Smits M, Williams A, Arendt J, et al. A length polymorphism in the circadian clock gene Per3 is linked to delayed sleep phase syndrome and extreme diurnal preference. Sleep. 2003;26(4):413-5.
29. Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. J Sleep Res. 1999;8(3):175-83.
30. Czeisler CA, Walsh JK, Roth T, Hughes RJ, Wright KP, Kingsbury L, et al. Modafinil for excessive sleepiness associated with shift-work sleep disorder. New England J Med. 2005;353(5):476-86.
31. Smith MT, McCrae CS, Cheung J, Martin JL, Harrod CG, Heald JL, Carden KA. Use of actigraphy for the evaluation of sleep disorders and circadian rhythm sleep-wake disorders: an American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. J Clin Sleep Med. 2018;14(7):1209-30.
32. Pickering L, Jennum P, Gammeltoft S, Poulsgaard L, Feldt-Rasmussen U, Klose M. Sleep–wake and melatonin pattern in craniopharyngioma patients. European J Endocrinol. 2014;170(6):873-84.
33. De Rui M, Middleton B, Sticca A, Gatta A, Amodio P, Skene DJ, Montagnese S. Sleep and circadian rhythms in hospitalized patients with decompensated cirrhosis: effect of light therapy. Neurochem Res. 2015;40(2):284-92.
34. Minors DS, Waterhouse JM. Separating the endogenous and exogenous components of the circadian rhythm of body temperature during night work using some ‘purification’ models. Ergonomics. 1993;36(5):497-507.
35. Lewy AJ, Sack RL. The dim light melatonin onset as a marker for orcadian phase position. Chronobiol Int. 1989;6(1):93-102.
36. Wyatt JK, Stepanski EJ, Kirkby J. Circadian phase in delayed sleep phase syndrome: predictors and temporal stability across multiple assessments. Sleep2006;29(8):1075-80.

37. Palmer CR, Kripke DF, Savage HC. Efficacy of enhanced evening light for advanced sleep phase syndrome. Behav Sleep Med. 2003;1(4):213-26.
38. Kushida CA, Chang A, Gadkary C, Guilleminault C, Carrillo O, Dement WC. Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep-disordered patients. Sleep Med. 2001;2(5):389-96.

Cite this article as: Chakrabarty M, Lahan V. The usefulness of various subjective and objective measures of circadian phase in sleep medicine diagnostics. Int J Res Med Sci 2025;13:4551-8.