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Exploration of sleep quality and rest-activity rhythms characteristics in Bilateral Vestibulopathy patients

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ABSTRACT

Sleep and circadian timing systems are constantly regulated by both photic and non-photic signals. Connections between the vestibular nuclei and the biological clock raise the question of the effect of peripheral vestibular loss on daily rhythms, such as the sleep-wake cycle and circadian rhythm. To answer this question, we compared the sleep and rest-activity rhythm parameters of 15 patients with bilateral vestibulopathy (BVP) to those of 15 healthy controls. Sleep and rest-activity cycle were recorded by a device coupling actimetry with the heart rate and actigraphy at home over 7 days. Subjective sleep quality was assessed by the Pittsburgh Sleep Quality Index (PSQI). Sleep efficiency and subjective sleep quality were significantly reduced, and sleep fragmentation was increased in BVP patients compared to controls. BVP patients displayed a damped amplitude of the rest-activity rhythm and higher sleep fragmentation, reflected by a higher nocturnal activity compared to controls. These results suggest that both rest-activity and sleep cycles are impaired in BVP patients compared to healthy controls. BVP patients good sleep at night compared to controls. BVP pathology appears to affect the sleep-wake cycle and disturb the circadian rhythm synchronization. Nevertheless, these results need further investigation to be confirmed, particularly with larger sample sizes.

circadian rhythms and sleep regulation [8-12].

this idea of non-photic stimuli influencing the biological clock, recent research has underscored the influence of the vestibular system on

circadian rhythms, suggesting it may serve as a stimulus affecting both

circular canals responsible for detecting angular head acceleration and

two otolithic organs responsible for detecting linear head acceleration.

This system is mainly responsible for postural control and gaze stabili-

zation during locomotion [13,14]. Animal and human studies have

suggested links between the vestibular system, sleep, and circadian

timing systems, with anatomical connections between the medial

vestibular nuclei and the SCN [15]. First, physiological evidence sup-

ports these links: (a) the vestibular system can impact rapid eye move-

ments (REM) during sleep [16-19]; (b) vestibular inputs to neurons in

the pontine reticular formation play a role in mediating transitions

The vestibular system, located in the inner ear, comprises three semi-

1. Introduction

Sleep and wake state alternate is regulated by the homeostatic and circadian timing system [1]. Together these two processes determine most aspects of sleep and related variables like sleepiness and alertness. Circadian timing system establishes an individual temporal organization [2] for each organism, enabling optimal task completion throughout the day-night cycle. For mammals, the primary temporal cue is the alternation between day and night brought about by Earth's rotation. Photoreceptors within the retina transmit light signals to the biological clock located in the suprachiasmatic nucleus (SCN) via the retinohypothalamic tract [3]. Additionally, non-photic cues such as feeding schedules and social interactions have been demonstrated to reliably synchronize the biological clock [4,5], and physical activity patterns can also impact the daily rhythm's amplitude and phase [6,7]. Following

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between sleep states [15,20,21]; and (c) the medial vestibular nucleus projects to cortical regions involved in arousal and certain aspects of sleep, receiving orexinergic inputs from the lateral hypothalamus [15]. Second, human studies have shown that vestibular stimulation by rocking can accelerate the transition between wakefulness and sleep and enhance sleep quality during napping [22,23]. Stimulation of the otoliths via linear bed movements has shown potential effects on drowsiness [24,25]. A study in mice has additionally confirmed the positive impact of vestibular stimulation on the wake-sleep transition [26].

Studies have highlighted a bidirectional association between vestibular symptoms and sleep disorders. For instance, individuals with vestibular disorders were found to exhibit abnormal sleep durations, such as very short or very long sleep durations, and experienced more severe sleep problems compared to those without vestibular disorders [27,28]. Using the Pittsburgh sleep questionnaire index (PSQI), studies revealed that patients with vestibular disorders reported significant sleep disturbances [29,30]. Polysomnography conducted over one night indicated reduced sleep efficiency (SE) and proportions of REM sleep in patients with vestibular migraines, have been linked with sleep disorders [33–35]. Wu et al. (2020) [31] demonstrated increased wake after sleep onset (WASO) in vestibular migraine patients compared to healthy controls. Finally, poor sleep quality was associated with more severe vestibular symptoms [29].

Most of these studies have been conducted on individuals suffering from various vestibular disorders, and mainly on patients with incomplete vestibular lesions. The heterogeneity of these disorders and the potential effects of the underlying diseases have made it challenging to interpret the link between the vestibular system and sleep. In the present study, we aimed to investigate sleep patterns in patients with bilateral vestibulopathy (BVP), a condition characterized by bilateral hypofunction of the vestibular organ or nerves with an unknown etiology [36] and not attributed to another disease as per the Barany Society diagnostic criteria [37]. BVP patients often exhibit significant disturbances in the vestibulo-ocular reflex during low-frequency movements, leading to symptoms such as oscillopsia [36,38,39]. They frequently report difficulties with gait initiation [40], balance issues in dark or uneven terrain, a sense of unsteadiness, and abnormal head and body alignment responses [39,41,42]. To our knowledge, no previous study has specifically examined sleep patterns in BVP patients, and only one investigation by Martin et al. (2016) [28] focused on rest-activity rhythm using actigraphy, coupled with temperature and cortisol profile recordings. They found that BVP patients exhibited fragmented sleep periods, increased nocturnal activity, and shorter sleep duration compared to healthy controls [28]. While actimetry is a reliable method for studying sleep in various populations [43,44], it does not provide detailed information about sleep architecture. Polysomnography, while the gold standard for measuring sleep architecture [45], is cumbersome and typically limited to recording sleep over a few nights. New devices, that combine actigraphy with beat-to-beat heart rate analysis, offer a promising approach to quantifying sleep architecture. This device has been shown to provide results comparable to manual scoring of polysomnography [46-48], offering a more practical and informative way to assess sleep patterns over several nights.

The first objective of this study was to assess the objective sleep quality of BVP patients compared to healthy control participants. We hypothesized that BVP patients would exhibit lower sleep quality and alterations in sleep architecture when compared to healthy controls. The second objective was to evaluate the rest-activity rhythms and subjective sleep quality of BVP patients compared to healthy control participants. We hypothesized that BVP patients would demonstrate disturbances in rest-activity rhythms and report poorer subjective sleep quality compared to the control group.

2. Materials and methods

2.1. Study design

This study constituted a single-center exploratory investigation involving BVP patients and age-, sex- and education-level-matched healthy controls. The study protocol received approval from a French ethics committee (CPP Nord Ouest III, IDRCB 2022-A01513-40). All participants provided written informed consent following a comprehensive explanation of the study procedures.

2.2. Participant

The BVP patients group was composed of 15 adults, comprising 6 men and 9 women, with a mean age of 53.3 ± 11.8 years. BVP was based on the criteria established by the Classification Committee of the Barany Society [37]. Specifically, each BVP patient exhibited bilateral reduction or absence of vestibulo-ocular reflex (VOR) angular function, as confirmed by video-head impulse test (vHIT) or caloric test. Additionally, none of the patients reported any hearing loss or associated neurological symptoms.

The control group included individuals selected from a non-published laboratory database with a matching sleep protocol. Fifteen healthy controls (9 women and 6 men, with a mean age of 51.9 ± 10.9 years) were selected, matching BVP patients based on sex, age, and education level.

All participants underwent a screening visit and had to meet the following inclusion criteria: (1) aged between 18 and 75 years old, and (2) diagnosed with BVP for the patients group or being healthy volunteers for the control group. Exclusion criteria included illiteracy; uncontrolled cardiovascular affection, psychiatric or neurologic affections; current or prior history of shift work; diagnosed sleep disorders (obstructive sleep apnea, chronic apnea, restless legs syndrome, narcolepsy-cataplexy); vestibular or hearing affections (for the controls group only); use of psychotropic or bradicardizing medications; substance abuse including alcohol, drug or caffeine; and pregnancy or breastfeeding. The demographic characteristics of the participants are presented in Table 1.

2.3. Measurements

2.3.1. General procedure

Two test sessions were conducted in the laboratory, one for the BVP patients and another for the healthy controls. During each session, participants completed questionnaires covering socio-demographics information, clinical status, and the PSQI. Additionally, they were provided with an actigraph device to record their rest-activity rhythms characteristics and a sleep architecture recording device to wear at night. Participants received training on how to use these devices before returning home for data collection. The recording conditions for both groups involved a 7-day period during which the activity-rest cycle was continuously monitored using actigraphy, coupled with the wearing of a Somno-Art device (Somno-Art®, PPRS, Colmar, France) for 7 consecutive nights. Subsequently, participants sent back the devices and the daily sleep diary to the laboratory for data analysis.

 Table 1

 Participant characteristics of BVP and Control groups.

	BVP (n = 15)	Controls ($n = 15$)
Age (years)	53.3 ± 11.8	51.9 ± 10.9
Gender (number of women)	9 (60.0 %)	9 (60.0 %)
BMI	$\textbf{24.4} \pm \textbf{4.1}$	$\textbf{24.2} \pm \textbf{2.3}$
Sleep medications as reported by PSQI (n)	0 (0 %)	0 (0 %)

2.3.2. Subjective sleep evaluation

The PSQI evaluates self-perceived sleep quality and disturbances experienced over the past month [49]. It covers various parameters including work schedule, sleep habits, disturbances during sleep, subjective sleep quality, use of sleep medication, and daytime sleepiness. A PSQI score exceeding 5 is indicative of potential sleep disorders [49].

2.3.3. Sleep parameters

The Somno-Art device combines the analysis of both actigraphy and heart rate at a beat-to-beat resolution. Participants were instructed to wear the Somno-Art armband on their non-dominant forearm for seven consecutive nights. The armband was worn before participants turned off the lights to sleep and removed upon waking up. The device recorded actimetry and heart rate data at a resolution of 1 Hz, while sleep stage classification was conducted at a 1-s epoch resolution.

2.3.4. Rest-activity rhythm parameters

Actigraphy involves recording body movements using a wrist-worn triaxial accelerometer. Participants wore an actigraph device (Motion Watch 8, CamNTech Ltd, Cambridge, UK) on their non-dominant wrist continuously for seven days. Each movement was recorded and stored within the device's memory, with movements above a 0.01G threshold being recorded. The device was set with filters in the range of 3–11 Hz to ensure accurate movement detection. After the seven-day recording period, the data were transferred and analyzed using Motion Watch Ware® software (V.1.3.33, Cambridge Neurotechnology Ltd, Cambridge, UK). According to general recommendations [50], both the BVP and control groups were asked to keep a daily sleep diary during the study period, reporting bedtime, waking-up time, naps, and any other relevant events such as device removal from the wrist. This self-report data was utilized for interpreting actigraphy data and to address potential errors.

2.4. Data analysis

2.4.1. Sleep parameters

The sleep classification algorithm utilized the Somno-Art Software v.1.6.1 employing expert rules for analysis [48]. Data collected from the Somno-Art device were processed using this software (v.1.6.1, PPRS, Colmar, France). Sleep classification was based on various parameters including sleep onset (hh:mm), wake-up time (hh:mm), total sleep time (TST, min), sleep onset latency (SOL, min), wake after sleep onset (WASO, min), sleep efficiency (SE, %), percentage of time (%) spent in each sleep stage (non-rapid-eye movements (NREM): S1, S2, S3; and REM).

2.4.2. Rest-activity rhythm parameters

Non-parametric circadian rhythm analysis (NPCRA) was conducted over the seven days of recording using actigraphy data processed with MotionWare® software (V.1.3.33, Cambridge Neurotechnology Ltd, Cambridge, UK). Two experimenters (E.M & T.M) analyzed the restactivity rhythm using NPCRA. Five parameters were measured [51]: (a) the inter-daily stability (IS); (b) intra-daily variability (IV); (c) L5 (average of the least active 5-h periods); (d) M10 (average of the most active 10-h); and (e) relative amplitude (RA).

The IS quantifies the regularity of the rest-activity pattern, ranging from 0 to 1, where 0 indicates no rhythm and 1 indicates perfect stability. A low IS suggests a weaker circadian rhythm. The IV measures the fragmentation of activity-rest periods. The variable has a theoretical range of 0–2 with higher values indicating higher fragmentation. L5 activity provides the average activity level during the least active 5-h, reflecting the restfulness and regularity of sleep periods; a lower L5 value suggests more restful sleep. M10 activity provides the average activity level during the activity and regularity of wake periods. RA ranges from 0 to 1, with higher values indicating a rhythm with a higher amplitude.

The dichotomy index (I < O) measures the percentage of time (ranging from 0 to 100 %) that activity during in-bed periods falls below the median level of activity during out-of-bed periods [52,53]. While this marker has mainly been validated in the cancer clinical population, we chose to utilize it due to the high degree of rest-activity rhythm fragmentation observed in BVP patients [28]. The I < O measure assesses the reduction in physical activity during in-bed periods (I) compared to activity levels during out-of-bed periods (O). A high I < Ovalue, close to 100 %, indicates a robust and prominent circadian rhythm, indicating restful sleep both at night and during the day. Conversely, a low I < O value indicates weak circadian rest-activity rhythmicity [52,54]. To categorize patients with circadian disruption, we applied a cut-off point for I < O of 99 % [55]. Thus, when I < O was lower or equal to 99 %, we inferred the presence of circadian rest-activity rhythm disruption. Conversely, when I < O exceeded 99 %, we concluded that the circadian rhythm was maintained.

2.5. Statistical analysis

PSQI score, rest-activity rhythm parameters, and sleep parameters were compared between the two subject groups using independent Student T-tests. A non-parametric Mann-Whitney *U* test was used when data were unequally distributed and the variances were unequal. Statistical analyses were performed using JASP 0.16.1.0 software. *p*-values less than 0.05 were considered statistically significant.

3. Results

3.1. Self-reported sleep measures

Significant differences in PSQI scores were observed between the two groups (Table 2). The PSQI score of the BVP patients was significantly higher compared to controls (+2.6; p = 0.006). 40 % of BVP patients (6.67 % of controls) had scores greater than the established cutoff score of 5, indicating decrements in sleep quality and the presence of sleep disorders.

3.2. Sleep parameters measures

All results concerning sleep parameters analysis are displayed in Table 3. WASO was significantly longer (+15.1min; p = 0.010) in BVP patients than in controls. SE was significantly lower (-4.0 %; p = 0.024) in BVP patients than in controls. A trend for a later wake-up time in the BVP group was also observed (-0.27min; p = 0.078). The 7-day analysis of sleep parameters did not reveal any significant differences between the two groups in sleep onset, wake-up time, TST, SOL, and the percentage of time in each sleep stage.

3.3. Rest-activity rhythm parameters

All results related to rest-activity rhythm analysis are presented in Table 4. L5 values were significantly higher (+365 counts; p = 0.045) in BVP patients than in controls. RA and I < O values were significantly lower (respectively, -0.06; p = 0.009; -2.92 %; p = 0.002) in BVP patients than in controls. I < O score was found below the 99 % cut-off in 14 out of 15 BVP patients and in 8 out of 15 controls. No significant differences were observed between the two subject groups for M10, IS, and IV values.

Table 2	
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Score of PSQI questionnaire (Mean \pm SD) for BVP patients and Controls.

	BVP (n = 15)	Controls ($n = 15$)	р
PSQI	5.3 ± 2.9	2.7 ± 1.3	0.006 ^a

^a Indicates a significant difference with p < 0.05.

Table 3

Mean Somno-Art sleep parameters (Mean \pm SD).

Measurements of sleep parameters for sleep onset, wake-up time, total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE), and percentage of time in each sleep stage (S1+2, S3, and REM).

	BVP (n = 15)	Controls ($n = 15$)	р
Sleep onset (hh:mm)	$00{:}08\pm01{:}51$	$23{:}31\pm00{:}38$	0.595
Wake-up time (hh:mm)	$\textbf{07:39} \pm \textbf{00:46}$	$\textbf{07:}12 \pm \textbf{00:}32$	0.068
TST (min)	397.8 ± 52.2	417.0 ± 55.9	0.336
SOL (min)	23.1 ± 25.1	17.7 ± 12.9	0.967
WASO (min)	$\textbf{44.3} \pm \textbf{16.4}$	$\textbf{29.2} \pm \textbf{13.3}$	0.010 ^a
SE (%)	$\textbf{85.7} \pm \textbf{4.7}$	89.7 ± 4.5	0.024 ^a
S1+2 mean percentage (%)	60.4 ± 5.7	59.8 ± 4.5	0.764
S3 mean percentage (%)	19.2 ± 5.3	18.7 ± 3.2	0.764
REM mean percentage (%)	$\textbf{20.4} \pm \textbf{3.4}$	21.5 ± 2.6	0.306

^a Indicates a significant difference with p < 0.05.

Table 4

Mean rest-activity rhythm parameters (Mean \pm SD).

Measurements of rest-activity rhythm parameters using actigraphy for IS (interdaily stability), IV (intra-daily variability), L5 activity (least active 5 h), M10 activity (most active 10 h), RA (relative amplitude), and I < O (dichotomy index).

	BVP patients ($n = 15$)	Controls ($n = 15$)	р
IS IV L5 activity (counts) M10 activity (counts)	$\begin{array}{c} 0.53 \pm 0.14 \\ 0.90 \pm 0.19 \\ 749 \pm 661 \\ 14\ 144 \pm 4254 \end{array}$	$\begin{array}{c} 0.56 \pm 0.13 \\ 0.79 \pm 0.17 \\ 384 \pm 277 \\ 16 \ 407 \pm 4105 \end{array}$	0.506 0.111 0.045 ^a 0.149
RA I < 0	$\begin{array}{l} 0.90 \pm 0.08 \\ 95.57 \pm 4.01 \end{array}$	$\begin{array}{c} 0.96 \pm 0.03 \\ 98.49 \pm 1.14 \end{array}$	0.009^{a} 0.002^{a}

^a Indicates a significant difference with p < 0.05.

4. Discussion

The primary main of this study was to evaluate the objective sleep quality of BVP patients compared to healthy control participants. The secondary objective was to assess the rest-activity rhythm and subjective sleep quality of BVP patients compared to healthy control participants. Our main hypotheses were that BVP patients would exhibit structural changes in sleep compared to healthy controls, specifically (a) a lower proportion of total sleep time, slow wave sleep (S3), and REM sleep; (b) a higher proportion of sleep latency, sleep lighter (S1+S2) and sleep fragmentation; and (c) a shifted phase of sleep.

BVP patients presented significant sleep modifications characterized by lower sleep quality, higher sleep fragmentation, and increased sleep complaints compared to controls. BVP patients displayed significant rest-activity rhythm alterations, including lower amplitude and I < Ovalues, and higher L5 period than controls.

4.1. Sleep disturbances

PSQI is commonly used as a clinical scale to assess subjective sleep quality [49]. Epidemiological studies suggest that approximately 20 % of adults report experiencing poor sleep quality [56]. In line with our hypothesis, we found that the PSQI score was higher in BVP patients compared to the controls. This finding is consistent with previous studies by Kim et al. (2018) [30] and Smith et al. (2018) [57], indicating that BVP patients are more likely to experience sleep complaints. Similar findings have been reported in studies examining other vestibular pathologies such as vestibular migraine, further supporting the results of our study [29,31].

To our knowledge, sleep parameters have not been previously evaluated in BVP patients before the present study. Previous investigations have focused on other vestibular pathologies such as vestibular migraine [31,32,58] and Ménière's disease [59]. These studies utilized polysomnography (PSG), which is considered the gold standard for sleep

assessment, but its use remains cumbersome. In our study, our methodological choice was to use Somno-art. This device offers a less invasive and more cost-effective means of monitoring sleep architecture at home over several consecutive nights. Importantly, studies have demonstrated that the results obtained with the Somno-Art device are comparable to those obtained through manual scoring of PSG [46-48]. Our findings reveal a decrease in sleep efficiency and an increase in WASO proportion among BVP patients compared to controls, which is consistent with the results obtained from the PSQI assessment. These results align with existing literature, as several studies have reported significantly lower sleep efficiency and higher sleep fragmentation in patients with vestibular migraine [31,32,58] and Ménière's disease [59] compared to controls. Interestingly, these two pathologies involving spontaneous episodic vestibular syndrome and without hypofunction of the vestibular system, or a peripheral organ hypofunction caused similar sleep alterations. This observation suggests a potential hypothesis that daily altered vestibular signals, originating either from the peripheral organ or modified centrally (e.g., in the vestibular nuclei), may contribute to sleep disturbances.

The vestibular nuclei indirectly project to the SCN [15,60], which regulates the sleep/wake cycle circadian rhythm. Supra-optical nucleus and paraventricular nucleus of the hypothalamus respond to the vestibulo-cochlear nerve and caloric stimulation of the labyrinth [61]. The locus coeruleus [62,63] and the intergeniculate leaflet (IGL) [62] serve as potential relays of vestibular inputs to the hypothalamic structures involved in circadian rhythms and sleep regulation. The IGL being involved in the transmission of both photic and non-photic stimuli to the SCN [64-66], could constitute a neuronal relay of vestibular inputs to the SCN, through the geniculohypothalamic tract (GHT). Based on these findings, it has been hypothesized that BVP affects the synchronization of the circadian rhythm and disrupts the sleep-wake cycle. In addition, hypocretin/orexin neurons in the hypothalamus, which are involved in maintaining wakefulness and alertness during the day [67-69], have projections from the medial vestibular nucleus [15]. These neurons promote the release of excitatory neurotransmitters such as norepinephrine, dopamine, and serotonin, which stimulate the brain and maintain wakefulness [68,69]. Dysfunction of hypocretin/orexin neurons could disrupt this regulation, contributing to an imbalance in the sleep-wake balance, including increased nighttime awakenings and thus decreased sleep efficiency.

The absence of changes in Wake-up time, Sleep onset, TST, and SOL values between our two groups is consistent with actigraphy findings reported by Martin et al. [28]. These results may indicate that BVP patients can maintain their daily routines, such as office work, business activities, domestic chores, and physical activity, without experiencing significant phase shifts despite their pathology symptoms, such as gait ataxia and postural balance disorders. While studies have established that poor sleep quality is a recognized risk factor for anxiety and major depressive disorders, and that the severity of dizziness and sleep-related problems are closely linked, both contributing to a reduced quality of life and emotional stress [70–73], a review of the literature suggests that BVP patients may not experience heightened anxiety distress compared to the other vestibular pathologies [74]. Nevertheless, many BVP patients report significant fatigue due to the symptoms associated with the disease, particularly those related to maintaining balance [16,57,75]. Therefore, it is plausible that the lack of difference in SOL and TST observed in our study may be attributed to this chronic fatigue.

We did not observe a difference in REM and NREM sleep in the BVP patients compared to the controls. This finding contrasts with previous studies that have reported significantly lower percentages of NREM and REM sleep in patients with vestibular migraine [31,32,58] and Ménière's disease [59]. However, these studies have focused on individuals experiencing incomplete and intermittent vestibular disorders. Furthermore, we cannot exclude that the absence of alteration in sleep architecture among BVP patients in our study may be limited by statistical underpowering, which remains thus puzzling and requires more

investigation.

4.2. Rest-activity rhythm disturbances

The influence of the vestibular system on circadian rhythms has been studied in rodents, primarily through methods such as 2G hypergravity, which have been shown to disrupt circadian rhythms of temperature and locomotor activity [8,11,76,77]. Data indicate a similar disruption of circadian rhythms in rats through chemical induction of a bilateral vestibular lesion [76]. In humans, the impact of vestibular stimulation on circadian rhythms has been characterized by a shift in sleep phases in healthy adults exposed to vestibular stimulation on a rotating chair [78]. Martin et al. (2016) [28] also showed a disruption of rest-activity and temperature circadian rhythms in BVP patients. Disruption of the circadian rhythm can lead to disturbances in the sleep-wake cycle and inversely [79,80]. Based on these findings, we hypothesized that BVP patients would exhibit rest-activity rhythm disorders characterized by reduced amplitude, increased sleep fragmentation with higher activity during the L5 period, and lower activity during the M10 period.

The actigraph is a highly reliable tool for characterizing the restactivity rhythm, validated for various populations [44,81,82], including those in free-living condition [50]. Using actigraphy, we observed a reduced amplitude of rest-activity rhythm in BVP patients compared to controls, which constitutes a marker of disrupted sleep, as commonly seen in populations with sleep disorders [50]. BVP patients showed higher movement during the L5 period compared to controls. Lower amplitude and increased movement during the L5 period are commonly associated with the trough of the sleep-wake cycle. We did not find differences in IS values between BVP patients and controls. However, IS values recorded for the BVP patients were similar to those reported in a previous study for insomniac people. Natale et al. (2014) [83] found no differences in IS values between insomniac people and healthy subjects, suggesting that altered rest-activity rhythm is not a systematic feature among patients with sleep disorders.

The dichotomy index I < O, utilized as a measure to assess both daytime activity and nighttime rest, has been established as a reliable marker of circadian rhythmicity alteration and is commonly employed in sleep research in patients with chronic diseases, such as cancer or insomnia [55,84]. Our findings indicate a high circadian disruption, which aligns with higher WASO, lower sleep efficiency, and diminished subjective sleep quality measured with the PSQI. This I < O score observed in BVP patients (95.57 \pm 4.01) was even under the cut-off of 97.5 %, which has been identified as a clinically relevant marker for the quality of life and survival outcomes in patients having chronic diseases such as cancer [54]. The alteration in rest-activity rhythms among BVP patients might be a key component strongly linked to sleep disruption and fragmented sleep-wake patterns. However, one must note that the I < 0 values of the control participants (98.49 \pm 1.14) appear to be slightly below the 99 % cut-off, even if 46.7 % of controls were above this cut-off. This result might question the quality of the rhythmicity of our control patient but is not surprising since a dramatic decline in the quality and quantity of sleep has been observed in the general healthy population and more than 8 out of 10 French people are affected by nocturnal awakenings as highlighted by a recent French national survey [85].

M10 values recorded in the BVP patients did not differ from those of the controls, indicating that the BVP patients were similarly as active as the controls during the M10 sequence. These results are consistent with the findings on sleep parameters obtained with the Somno-Art device and reflect the ability of our BVP patients to complete their daily routine (e.g. office work, business, domestic activities, physical activity) despite their pathology and its associated symptoms. In this study, patients had been diagnosed several years prior, suggesting that certain dynamic compensatory processes had already been completed [41,86], allowing for normal postural adjustments in common situations. This could also explain why patients in our study maintained similar M10 values to the

control group during the 7-day home actigraphy.

4.3. Limitations

Due to the rarity of the BVP pathology (120/100 000) [87], we cannot exclude that our study suffers from statistical underpowering limitation, therefore these results requires more investigation and need to be confirmed in studies with larger sample sizes. Consequently, we did not consider the subtypes of BVP due to the same size of our sample. Additionally, despite their common pathology, the subjects are varied in age, physical activity, and lifestyle. Furthermore, we did not account for the level of physical activity when matching our BVP patients with controls. Although we observed trends for wake-up time, we cannot discount the possibility that the small number of subjects prevented us from observing other significant results in this study. However, our sample size was sufficient to detect significant differences in some sleep and rest-activity rhythm parameters. The absence of a thorough PSG, typically including respiratory parameters, may be a limitation due to the potential presence of participants with undiagnosed obstructive sleep apneas - a condition previously associated with vestibular weakness [88] - which could have introduced bias to the study sample.

5. Conclusion

The present study highlights that BVP patients experience poorer objective and subjective sleep compared to healthy participants. BVP patients seem to have greater difficulty maintaining good sleep at night, which is consistent with greater and more pronounced sleep complaints in BVP patients compared to controls. These findings support the concept that good overall health correlates with better sleep quality and robust circadian motor rest-activity rhythms.

Evaluating sleep parameters and rest-activity rhythm may contribute to a better understanding of vestibular involvement in sleep. These results may also help to establish more effective therapeutic interventions, such as vestibular rehabilitation and stimulation for sleep improvement [73,89,90]. The present data could be a further step in promoting additional clinical attention and research into sleep disturbances in BVP patients, but, these results warrant further investigations to be confirmed, particularly with larger sample sizes. For future work, we plan to conduct a larger-scale study using polysomnography, the gold standard for sleep measurement.

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Ethics statement

All participants provided written informed consent to take part in the study, which was conducted in line with the Declaration of Helsinki and approved by the ethics committee (CPP Nord Ouest III), n° ID-RCB: 2022-A01513-40.

Data availability statement

Data will be provided upon reasonable request to the corresponding author.

CRediT authorship contribution statement

E. Milot: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **T. Martin:** Writing – review & editing, Writing – original draft, Visualization,

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Declaration of competing interest

We confirm that this work is original, has not been published elsewhere, and is not currently under consideration for publication elsewhere. All authors have approved the manuscript and agree with its submission to Sleep Medecine.

We have no conflicts of interest to disclose.

We have no financial/personal/competing interests that could affect our objectivity.

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References

- Borbély AA. A two process model of sleep regulation. Hum Neurobiol 1982;1(3): 195–204.
- [2] Pittendrigh CS. Circadian rhythms and the circadian organization of living systems. Cold Spring Harbor Symp Quant Biol 1960;25:159–84. https://doi.org/10.1101/ sqb.1960.025.01.015.
- [3] Moore RY. Organization of the mammalian circadian system, vol. 183. Ciba Found Symp; 1995. p. 88–99. ; discussion 100-106.
- [4] De Araujo LD, Roa SL, Bueno AC, et al. Restricted feeding schedules modulate in a different manner the expression of clock genes in rat hypothalamic nuclei. Front Neurosci 2016;10:567. https://doi.org/10.3389/fnins.2016.00567.
- [5] Mistlberger RE, Skene DJ. Social influences on mammalian circadian rhythms: animal and human studies. Biol Rev Camb Phil Soc 2004;79(3):533–56. https:// doi.org/10.1017/s1464793103006353.
- [6] Atkinson G, Edwards B, Reilly T, Waterhouse J. Exercise as a synchroniser of human circadian rhythms: an update and discussion of the methodological problems. Eur J Appl Physiol 2007;99(4):331–41. https://doi.org/10.1007/ s00421-006-0361-z.
- [7] Challet E. Minireview: entrainment of the suprachiasmatic clockwork in diurnal and nocturnal mammals. Endocrinology 2007;148(12):5648–55. https://doi.org/ 10.1210/en.2007-0804.
- [8] Fuller PM, Jones TA, Jones SM, Fuller CA. Neurovestibular modulation of circadian and homeostatic regulation: vestibulohypothalamic connection? Proc Natl Acad Sci USA 2002;99(24):15723–8. https://doi.org/10.1073/pnas.242251499.
- [9] Fuller PM, Fuller CA. Genetic evidence for a neurovestibular influence on the mammalian circadian pacemaker. J Biol Rhythm 2006;21(3):177–84. https://doi. org/10.1177/0748730406288148.
- [10] Martin T, Zouabi A, Pasquier F, Denise P, Gauthier A, Quarck G. Twenty-four-hour variation of vestibular function in young and elderly adults. Chronobiol Int 2020; 38:1–13. https://doi.org/10.1080/07420528.2020.1835941.
- [11] Murakami DM, Erkman L, Hermanson O, Rosenfeld MG, Fuller CA. Evidence for vestibular regulation of autonomic functions in a mouse genetic model. Proc Natl Acad Sci USA 2002;99(26):17078–82. https://doi.org/10.1073/pnas.252652299.
- [12] Quarck G, Ventre J, Etard O, Denise P. Total sleep deprivation can increase vestibulo-ocular responses. J Sleep Res 2006;15(4):369–75. https://doi.org/ 10.1111/j.1365-2869.2006.00550.x.

- [13] Grossman GE, Leigh RJ, Bruce EN, Huebner WP, Lanska DJ. Performance of the human vestibuloocular reflex during locomotion. J Neurophysiol 1989;62(1): 264–72. https://doi.org/10.1152/jn.1989.62.1.264.
- [14] Peterson BW, Richmond FJ. Control of head movement. Oxford University Press; 1988.
- [15] Horowitz SS, Blanchard J, Morin LP. Medial vestibular connections with the hypocretin (orexin) system. J Comp Neurol 2005;487(2):127–46. https://doi.org/ 10.1002/cne.20521.
- [16] Altena E, Buguet E, Higginson C, et al. Vestibular symptoms are related to the proportion of REM sleep in people with sleep complaints: a preliminary report. J Vestib Res 2023;33(3):165–72. https://doi.org/10.3233/VES-220113.
- [17] Eisensehr I, Noachtar S, Strupp M, von Lindeiner H, Brandt T, Büttner U. Absence of nystagmus during REM sleep in patients with vestibular neuritis. J Neurol Neurosurg Psychiatry 2001;71(3):386–9. https://doi.org/10.1136/jnnp.71.3.386.
- [18] Hobson JA, Stickgold R, Pace-Schott EF, Leslie KR. Sleep and vestibular adaptation: implications for function in microgravity. J Vestib Res 1998;8(1):81–94.
- [19] Morrison AR, Pompeiano O. Vestibular influences during sleep. VI. Vestibular control of autonomic functions during the rapid eye movements of desynchronized sleep. Arch Ital Biol 1970;108(1):154–80.
- [20] Puligheddu M, Figorilli M, Serra A, et al. REM Sleep without atonia correlates with abnormal vestibular-evoked myogenic potentials in isolated REM sleep behavior disorder. Sleep 2019;42(9):zsz128. https://doi.org/10.1093/sleep/zsz128.
- [21] de Natale ER, Ginatempo F, Laccu I, et al. Vestibular evoked myogenic potentials are abnormal in idiopathic REM sleep behavior disorder. Front Neurol 2018;9:911. https://doi.org/10.3389/fneur.2018.00911.
- [22] Bayer L, Constantinescu I, Perrig S, et al. Rocking synchronizes brain waves during a short nap. Curr Biol 2011;21(12):R461–2. https://doi.org/10.1016/j. cub.2011.05.012.
- [23] Omlin X, Crivelli F, Näf M, et al. The effect of a slowly rocking bed on sleep. Sci Rep 2018;8(1):2156. https://doi.org/10.1038/s41598-018-19880-3.
- [24] Crivelli F, Omlin X, Rauter G, von Zitzewitz J, Achermann P, Riener R. Somnomat: a novel actuated bed to investigate the effect of vestibular stimulation. Med Biol Eng Comput 2016;54(6):877–89. https://doi.org/10.1007/s11517-015-1423-3.
- [25] Woodward S, Tauber ES, Spielmann AJ, Thorpy MJ. Effects of otolithic vestibular stimulation on sleep. Sleep 1990;13(6):533–7. https://doi.org/10.1093/sleep/ 13.6.533.
- [26] Kompotis K, Hubbard J, Emmenegger Y, et al. Rocking promotes sleep in mice through rhythmic stimulation of the vestibular system. Curr Biol 2019;29(3): 392–401.e4. https://doi.org/10.1016/j.cub.2018.12.007.
- [27] Albathi M, Agrawal Y. Vestibular vertigo is associated with abnormal sleep duration. J Vestib Res 2017;27(2–3):127–35. https://doi.org/10.3233/VES-170617.
- [28] Martin T, Moussay S, Bulla I, et al. Exploration of circadian rhythms in patients with bilateral vestibular loss. PLoS One 2016;11(6):e0155067. https://doi.org/ 10.1371/journal.pone.0155067.
- [29] Mutlu B, Topcu MT. Investigation of the relationship between vestibular disorders and sleep disturbance. Int Arch Otorhinolaryngol 2022;26(4):e688–96. https://doi. org/10.1055/s-0042-1742763.
- [30] Kim SK, Kim JH, Jeon SS, Hong SM. Relationship between sleep quality and dizziness. PLoS One 2018;13(3):e0192705. https://doi.org/10.1371/journal. pone.0192705.
- [31] Wu J, Liu C, Yu H, et al. Clinical characteristics of sleep disorders in patients with vestibular migraine. Sleep Breath 2020;24(4):1383–8. https://doi.org/10.1007/ s11325-019-01994-1.
- [32] Karthik N, Sinha S, Taly AB, Kulkarni GB, Ramachandraiah CT, Rao S. Alteration in polysomnographic profile in 'migraine without aura' compared to healthy controls. Sleep Med 2013;14(2):211–4. https://doi.org/10.1016/j.sleep.2012.10.019.
- [33] Tiseo C, Vacca A, Felbush A, et al. Migraine and sleep disorders: a systematic review. J Headache Pain 2020;21(1):126. https://doi.org/10.1186/s10194-020-01192-5.
- [34] Çelebisov N, Ak AK, Ataç C, et al. Comparison of clinical features in patients with vestibular migraine and migraine. J Neurol 2023;270(7):3567–73. https://doi.org/ 10.1007/s00415-023-11677-3.
- [35] Akdal G, Ozge A, Ergör G. The prevalence of vestibular symptoms in migraine or tension-type headache. J Vestib Res 2013;23(2):101–6. https://doi.org/10.3233/ VES-130477.
- [36] Fujimoto C, Yagi M, Murofushi T. Recent advances in idiopathic bilateral vestibulopathy: a literature review. Orphanet J Rare Dis 2019;14:202. https://doi. org/10.1186/s13023-019-1180-8.
- [37] Strupp M, Kim JS, Murofushi T, et al. Bilateral vestibulopathy: diagnostic criteria consensus document of the classification committee of the bárány Society1. J Vestib Res. 27(4):177-189. doi:10.3233/VES-170619.
- [38] Baloh RW, Fife TD, Zwerling L, et al. Comparison of static and dynamic posturography in young and older normal people. J Am Geriatr Soc 1994;42(4): 405–12. https://doi.org/10.1111/j.1532-5415.1994.tb07489.x.
- [39] Sargent E, Goebel J, Hanson J, Beck D. Idiopathic bilateral vestibular loss. Published online, https://journals.sagepub.com/doi/abs/10.1016/S0194-5998 9770318-8. [Accessed 30 August 2023].
- [40] Sasaki O, Asawa S, Katsuno S, Usami S, Taguchi K. Gait initiation in bilateral vestibular loss. Auris Nasus Larynx 2001;28(4):295–9. https://doi.org/10.1016/ s0385-8146(01)00094-3.
- [41] Horak FB, Nashner LM, Diener HC. Postural strategies associated with somatosensory and vestibular loss. Exp Brain Res 1990;82(1):167–77. https://doi. org/10.1007/BF00230848.

- [42] Rinne T, Bronstein AM, Rudge P, Gresty MA, Luxon LM. Bilateral loss of vestibular function: clinical findings in 53 patients. J Neurol 1998;245(6–7):314–21. https:// doi.org/10.1007/s004150050225.
- [43] de Souza L, Benedito-Silva AA, Pires MLN, Poyares D, Tufik S, Calil HM. Further validation of actigraphy for sleep studies. Sleep 2003;26(1):81–5. https://doi.org/ 10.1093/sleep/26.1.81.
- [44] Sadeh A. The role and validity of actigraphy in sleep medicine: an update. Sleep Med Rev 2011;15(4):259–67. https://doi.org/10.1016/j.smrv.2010.10.001.
- [45] Rundo JV, Downey R. Polysomnography. Handb Clin Neurol 2019;160:381–92. https://doi.org/10.1016/B978-0-444-64032-1.00025-4.
- [46] Thiesse L, Staner L, Fuchs G, et al. Performance of Somno-Art Software compared to polysomnography interscorer variability: a multi-center study. Sleep Med 2022; 96:14–9. https://doi.org/10.1016/j.sleep.2022.04.013.
- [47] Thiesse L, Staner L, Bourgin P, et al. Validation of Somno-Art Software, a novel approach of sleep staging, compared with polysomnography in disturbed sleep profiles. Sleep Adv 2022;3(1):zpab019. https://doi.org/10.1093/sleepadvances/ zpab019.
- [48] Muzet A, Werner S, Fuchs G, et al. Assessing sleep architecture and continuity measures through the analysis of heart rate and wrist movement recordings in healthy subjects: comparison with results based on polysomnography. Sleep Med 2016;21:47–56. https://doi.org/10.1016/j.sleep.2016.01.015.
- [49] Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatr Res 1989;28(2):193–213. https://doi.org/10.1016/0165-1781(89)90047-4.
- [50] Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. Sleep 2003;26(3):342–92. https://doi.org/10.1093/sleep/26.3.342.
- [51] Bonmati-Carrion MA, Middleton B, Revell VL, Skene DJ, Rol MA, Madrid JA. Validation of an innovative method, based on tilt sensing, for the assessment of activity and body position. Chronobiol Int 2015;32(5):701–10. https://doi.org/ 10.3109/07420528.2015.1016613.
- [52] Minors D, Akerstedt T, Atkinson G, et al. The difference between activity when in bed and out of bed. I. Healthy subjects and selected patients. Chronobiol Int 1996; 13(1):27–34. https://doi.org/10.3109/07420529609040839.
- [53] Mormont MC, Waterhouse J, Bleuzen P, et al. Marked 24-h rest/activity rhythms are associated with better quality of life, better response, and longer survival in patients with metastatic colorectal cancer and good performance status. Clin Cancer Res 2000;6(8):3038–45.
- [54] Innominato PF, Giacchetti S, Bjarnason GA, et al. Prediction of overall survival through circadian rest-activity monitoring during chemotherapy for metastatic colorectal cancer. Int J Cancer 2012;131(11):2684–92. https://doi.org/10.1002/ ijc.27574.
- [55] Natale V, Innominato PF, Boreggiani M, et al. The difference between in bed and out of bed activity as a behavioral marker of cancer patients: a comparative actigraphic study. Chronobiol Int 2015;32(7):925–33. https://doi.org/10.3109/ 07420528.2015.1053909.
- [56] Morin CM, Jarrin DC. Epidemiology of insomnia: prevalence, course, risk factors, and public health burden. Sleep Med Clin 2022;17(2):173–91. https://doi.org/ 10.1016/j.jsmc.2022.03.003.
- [57] Smith L, Wilkinson D, Bodani M, Bicknell R, Surenthiran SS. Short-term memory impairment in vestibular patients can arise independently of psychiatric impairment, fatigue, and sleeplessness. J Neuropsychol 2019;13(3):417–31. https://doi.org/10.1111/jnp.12157.
- [58] Xue H, Wang B, Meng T, et al. Differences of sleep disorders between vestibular migraine and benign paroxysmal positional vertigo. Front Psychiatr 2021;12: 726038. https://doi.org/10.3389/fpsyt.2021.726038.
- [59] Nakayama M, Suzuki M, Inagaki A, et al. Impaired quality of sleep in Ménière's disease patients. J Clin Sleep Med 2010;6(5):445–9.
- [60] Cavdar S, Onat F, Aker R, Schirli U, San T, Yananli HR. The afferent connections of the posterior hypothalamic nucleus in the rat using horseradish peroxidase. J Anat 2001;198(Pt 4):463–72. https://doi.org/10.1046/j.1469-7580.2001.19840463.x.
- [61] Azzena GB, Melis F, Caria MA, Teatini GP, Bozzo G. Vestibular projections to hypothalamic supraoptic and paraventricular nuclei. Arch Ital Biol 1993;131(2–3): 127–36.
- [62] Horowitz SS, Blanchard JH, Morin LP. Intergeniculate leaflet and ventral lateral geniculate nucleus afferent connections: an anatomical substrate for functional input from the vestibulo-visuomotor system. J Comp Neurol 2004;474(2):227–45.
- [63] Pompeiano O, Manzoni D, Barnes CD. Responses of locus coeruleus neurons to labyrinth and neck stimulation. Prog Brain Res 1991;88:411–34. https://doi.org/ 10.1016/s0079-6123(08)63826-1.
- [64] Harrington ME. The ventral lateral geniculate nucleus and the intergeniculate leaflet: interrelated structures in the visual and circadian systems. Neurosci Biobehav Rev 1997;21(5):705–27.
- [65] Janik D, Mrosovsky N. Intergeniculate leaflet lesions and behaviorally-induced shifts of circadian rhythms. Brain Res 1994;651(1–2):174–82. https://doi.org/ 10.1016/0006-8993(94)90695-5.

- [66] Muscat L, Morin L. Intergeniculate leaflet: contributions to photic and non-photic responsiveness of the hamster circadian system. Neuroscience 2006;140(1): 305–20.
- [67] Luppi PH, Fort P. Neuroanatomical and neurochemical bases of vigilance states. Handb Exp Pharmacol 2019;253. https://doi.org/10.1007/164_2017_84.
- [68] Ohno K, Sakurai T. Orexin neuronal circuitry: role in the regulation of sleep and wakefulness. Front Neuroendocrinol 2008;29(1):70–87. https://doi.org/10.1016/ j.yfrne.2007.08.001.
- [69] Inutsuka A, Yamanaka A. The physiological role of orexin/hypocretin neurons in the regulation of sleep/wakefulness and neuroendocrine functions. Front Endocrinol 2013;4:18. https://doi.org/10.3389/fendo.2013.00018.
- [70] Konomi U, Suzuki M, Ogawa Y, et al. Assessment of sleep disturbance using the Pittsburgh Sleep Quality Index in patients with dizziness. Equilib Res 2014;73(6): 502–11. https://doi.org/10.3757/jser.73.502.
- [71] Buysse DJ, Angst J, Gamma A, Ajdacic V, Eich D, Rössler W. Prevalence, course, and comorbidity of insomnia and depression in young adults. Sleep 2008;31(4): 473–80. https://doi.org/10.1093/sleep/31.4.473.
- [72] Weidt S, Bruehl AB, Straumann D, Hegemann SCA, Krautstrunk G, Rufer M. Healthrelated quality of life and emotional distress in patients with dizziness: a crosssectional approach to disentangle their relationship. BMC Health Serv Res 2014;14: 317. https://doi.org/10.1186/1472-6963-14-317.
- [73] Sugaya N, Arai M, Goto F. The effect of vestibular rehabilitation on sleep disturbance in patients with chronic dizziness. Acta Otolaryngol 2017;137(3): 275–8. https://doi.org/10.1080/00016489.2016.1244859.
- [74] Brandt T, Dieterich M. 'Excess anxiety' and 'less anxiety': both depend on vestibular function. Curr Opin Neurol 2020;33(1):136. https://doi.org/10.1097/ WCO.0000000000000771.
- [75] Karabulut M, Van Laer L, Hallemans A, et al. Chronic symptoms in patients with unilateral vestibular hypofunction: systematic review and meta-analysis. Front Neurol 2023;14:1177314. https://doi.org/10.3389/fneur.2023.1177314.
- [76] Martin T, Mauvieux B, Bulla J, et al. Vestibular loss disrupts daily rhythm in rats. J Appl Physiol 2015;118(3):310–8. https://doi.org/10.1152/ japplphysiol.00811.2014. 1985.
- [77] Holley DC, DeRoshia CW, Moran MM, Wade CE. Chronic centrifugation (hypergravity) disrupts the circadian system of the rat. J Appl Physiol 2003;95(3): 1266–78. https://doi.org/10.1152/japplphysiol.00707.2002. 1985.
- [78] Pasquier F, Bessot N, Martin T, et al. Effect of vestibular stimulation using a rotatory chair in human rest/activity rhythm. Chronobiol Int 2020;37(8):1244–51. https://doi.org/10.1080/07420528.2020.1797762.
- [79] Moore RY. Suprachiasmatic nucleus in sleep-wake regulation. Sleep Med 2007;8 (Suppl 3):27–33. https://doi.org/10.1016/j.sleep.2007.10.003.
- [80] Hastings MH, Maywood ES, Brancaccio M. Generation of circadian rhythms in the suprachiasmatic nucleus. Nat Rev Neurosci 2018;19(8):453–69. https://doi.org/ 10.1038/s41583-018-0026-z.
- [81] Lieberman HR, Wurtman JJ, Teicher MH. Circadian rhythms of activity in healthy young and elderly humans. Neurobiol Aging 1989;10(3):259–65. https://doi.org/ 10.1016/0197-4580(89)90060-2.
- [82] Osse RJ, Tulen JHM, Bogers AJJC, Hengeveld MW. Disturbed circadian motor activity patterns in postcardiotomy delirium. Psychiatr Clin Neurosci 2009;63(1): 56–64. https://doi.org/10.1111/j.1440-1819.2008.01888.x.
- [83] Natale V, Léger D, Bayon V, et al. The consensus sleep diary: quantitative criteria for primary insomnia diagnosis. Psychosom Med 2015;77(4):413–8. https://doi. org/10.1097/PSY.00000000000177.
- [84] Madsen MT, Huang C, Gögenur I. Actigraphy for measurements of sleep in relation to oncological treatment of patients with cancer: a systematic review. Sleep Med Rev 2015;20:73–83. https://doi.org/10.1016/j.smrv.2014.07.002.
- [85] Sondage OpinionWay pour INSV/MGEN Enquête sommeil 2023 réalisé du 02 au 12 décembre 2022 auprès de 1004 personnes représentatives des Français âgés de 18-65 ans interrogées selon la méthode des quotats par questionnaire autoadministré en ligne sur système CAWI.
- [86] McCall AA, Yates BJ. Compensation following bilateral vestibular damage. Front Neurol 2011;2:88. https://doi.org/10.3389/fneur.2011.00088.
- [87] Della Santina CC, Migliaccio AA, Hayden R, et al. Current and future management of bilateral loss of vestibular sensation - an update on the Johns Hopkins Multichannel Vestibular Prosthesis Project. Cochlear Implants Int 2010;11(Suppl 2):2–11. https://doi.org/10.1179/146701010X12726366068454. Suppl 2.
- [88] Micarelli A, Liguori C, Viziano A, Izzi F, Placidi F, Alessandrini M. Integrating postural and vestibular dimensions to depict impairment in moderate-to-severe obstructive sleep apnea syndrome patients. J Sleep Res 2017;26(4):487–94. https://doi.org/10.1111/jsr.12516.
- [89] Krystal AD, Zammit GK, Wyatt JK, et al. The effect of vestibular stimulation in a four-hour sleep phase advance model of transient insomnia. J Clin Sleep Med : JCSM : official publication of the American Academy of Sleep Medicine 2010;6(4): 315–21. https://doi.org/10.5664/jcsm.27871.
- [90] Micarelli A, Viziano A, Carbini V, et al. Effects of vestibular rehabilitation on body composition and daily-living physical activity in chronic unilateral vestibular hypofunction. J Vestib Res 2023;33(1):71–83. https://doi.org/10.3233/VES-220019.