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Would the Actigraph Always be Sufficient for Sleep Analysis in Exercise-Based Studies? A Case Report of Negative Response of Sleep to Exercise

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Abstract

Introduction Sleep deprivation is common after coronary artery bypass grafting (CABG). It is mostly managed well by exercise. The number of reported post-CABG cases that respond negatively to exercise is scanty. The etiology is usually associated with the underlying sleep pathology, and how it responds to exercise. Cases with undiagnosed central sleep apnea post CABG have not been reported before.

Case description A medically stable male patient, 63 years old, hypertensive, but not diabetic, had entered coronary artery bypass grafting (CABG) 8 weeks before attending the outpatient cardiac rehabilitation unit and was referred for a cardiac rehabilitation program at this time. He entered a study in the cardiac rehabilitation center utilizing either aerobic or combined aerobic and resistance training for 10 weeks to improve sleep architecture and functional capacity post-CABG. After randomization, he entered the group doing combined aerobic and resistance exercises. All of the patients in this group improved except him, his sleep quality worsened, but his functional capacity improved. After a complete analysis of sleep on polysomnography, it was revealed that the patient had central sleep apnea that was mostly worsened by resistance training. The patient was withdrawn from the study by the 8th week, and his sleep condition improved gradually. After then, he was asked to attend the cardiac rehabilitation center again to share in aerobic exercise, having evidence that central sleep apnea does not respond negatively to this form of training. After 12 months of follow-up, the patient still shows no signs of sleep deprivation.

Conclusion Sleep deprivation is prevalent in post-CABG patients, but with different presentations and it can generally improve by exercise. Identification of the underlying cause of the sleeping difficulty is a cornerstone of targeted treatment.

Keywords
- actigraph
- central sleep apnea
- polysomnography
- coronary artery bypass grafting (CABG)

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Introduction

Evidence demonstrated that patients may experience sleep deprivation weeks or months after coronary artery bypass grafting (CABG).[1–3] In a few instances, the underlying reason is understandable, however, in the majority of cases, it is not. That raised a new definition in sleep medicine called non-specific insomnia or sleep deprivation (SD).[4,5]

Exercise that was proven to positively affect sleep post-CABG has numerous variations. Several studies investigated the effect of aerobic, resisted, combined forms, and it was proven that morning exercise can be used as a safe technique for controlling sleep disorders post CABG without any adverse effects. Few studies reported that exercising at night may lead to exercise-induced insomnia.[6] To our knowledge, no data on adverse reactions to morning exercise are documented.[5,7]

In the current case, we are presenting a case of a patient who did not respond well to the morning exercise done post-CABG. The patient had his sleep quality worsened with complaints of SD.

Patient Information and Clinical Findings

This case conforms with the recommendations in the CARE guidelines for case reports.[8] Approval of the Research Ethical committee of the faculty of physical therapy, Cairo University was taken for this study on 3/12/2017 under number: P.T. REC/012/001805 and written informed consent for patient information and images to be published was provided by the patient.

A medically stable mixed African male patient, 63 years old, hypertensive, but not diabetic, had entered coronary artery bypass grafting (CABG) 8 weeks before attending the outpatient cardiac rehabilitation unit and was referred for a cardiac rehabilitation program at this time.

A complete history taking, and clinical examination were conducted to ensure complete post-surgical recovery and adequate blood pressure control. History taking showed that the patient blood pressure was 140/90, had a family history of cardiovascular disease (CVD), and had no previous history of anxiety and/or sleeping disorder before the CABG. He had a normal body mass index (BMI) of 26 Kg/m2, a history of transient ischemic attack (TIA) that happened 2 months before the surgery, and a complaint of a significant decline in exercise capacity, the fact that led him to consult the cardiologist and consequently underwent the CABG.

The patient entered a study in the cardiac rehabilitation center utilizing either aerobic or combined aerobic and resistance training for 10 weeks to improve sleep architecture and functional capacity post-CABG. After randomization, he entered the group doing combined aerobic and resistance exercises. All the patients in this group improved except him, his sleep quality worsened, but his functional capacity improved. ►**Fig. 1**

Diagnostic Assessment

As a part of the cardiac rehabilitation study, functional capacity was examined by the 6 Minute Walk Test which is a sub-maximal exercise test used to assess aerobic capacity and endurance. The distance covered over a time of 6 minutes is used as the outcome by which to compare changes in functional capacity.[7,9]

After then, Sleep Quality was assessed by Pittsburgh Sleep Quality Index (PSQI) which is a self-report questionnaire that assesses sleep quality subjectively. Actigraphy as a valid and objective tool was used to assess sleep patterns objectively.[10]

Actigraph recorded sleep latency, total sleep duration, deep sleep duration, light sleep duration, sleep fragmentation index, and sleep efficiency. Actigraph was placed on the non-dominant wrist and activities were monitored continuously and recorded at one-minute intervals during wakefulness and sleep. The times of wakefulness and sleep were determined utilizing a diary kept by the patient, together with the times calculated by the actigraph monitor. The patient kept the device for 96 hours before the first and last training sessions. The device was removed only at bath time and replaced on the wrist immediately after. The information was collected by the device and recorded by a validated algorithm (Minimitter Company - USA®) and transferred to closed technology software installed on a computer. After analysis of the exams, the data were plotted on a spreadsheet for statistical treatment.[11,12]

Therapeutic Interventions

Regarding the exercise program of the patient's group, it included: Aerobic exercise: with 13-15 intensity on a modified BORG scale, 5 minutes warm-up, 30 minutes of moderate aerobic exercise, and 10 minutes cool down. in 3 sessions/week for 10 weeks. That was in addition to resistance exercise: 30% then progress to 50-60% of one-repetition maximum(1RM). Resistance training included 5 minutes of warm-up, and 30 minutes of nine exercises selected in the following order: 1- leg press 45 degrees. 2- Bench press. 3- Extensor Bench. 4- Handle Front. 5- Flexor Bench- sitting. 6- Upright Row. 7- Planter Flexion. 8- Seated Row and 9- Abdominals and 10 minutes cool down for 3 sessions/week for 10 weeks.

All the patients in this group improved dramatically in their sleep and functional capacity with exception of this patient. He complained that his sleep was getting worse. Therefore, in the 8th week of the rehabilitation program, we -as the medical team- decided to withdraw this patient from the study, and do a detailed level examination for him, trying to explain why he responded negatively to the treatment.[7,9]

In the 8th week, we proposed doing diagnostic overnight polysomnography (PSG) assessment for this study to monitor sleep and body functions during sleep.

This rarely happens, and we couldn't reach a similar case who experienced exercise-induced insomnia on conducting a morning exercise.

Follow-up and Outcomes

In the 8th week, a follow-up assessment was done. Physical examination showed that the patient had slightly elevated

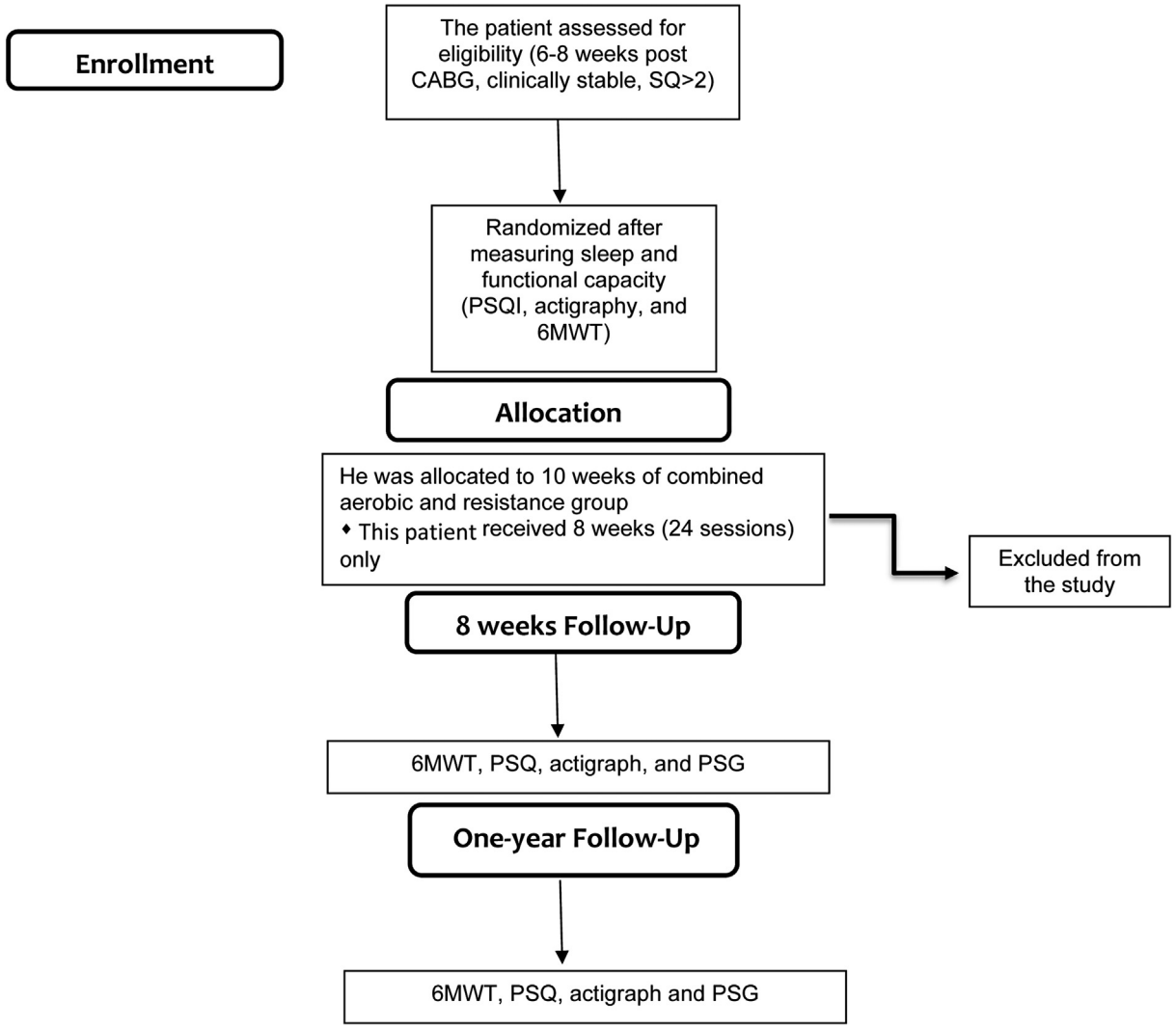

Fig. 1 Flow Diagram of the study.

blood pressure (150/90), otherwise, he was medically stable. Pittsburg sleep quality index (PSQI) scores were higher than those recorded in the initial assessment (moved from 2 to 3) (the scale ranges from 0 to 3, and higher scores mean worse sleep quality). Actigraphy was worn for 4 days by the patient and the sleep-related outcomes were recorded. Sleep latency increased (SL: 120 to 180 min), total sleep duration was the same (TSD: 3 hours), but the proportion of deep sleep within the total sleep duration had decreased (DSD: 60 to 45 min), and the proportion of light sleep had increased (LSD: 120 to 135 min). Also, the sleep pattern became more fragmented (i.e., the patient woke up frequently at night) and the sleep fragmentation index moved from (52.92 to 72.12 %). In addition, sleep efficiency deteriorated (16 to 13%).

On the opposite hand, his 6MWD had improved dramatically (220 to 400 meters). Additionally, polysomnography (PSG) analysis was done, and it showed signs of central sleep apnea in the form of apnea-hypopnea index (AHI) of 12 seconds (i.e. cessation of airflow for 12 seconds without any identifiable respiratory effort). ►**Table 1**

After the literature review, there was solid evidence that aerobic training improves the severity of central sleep apnea and does not negatively affect this form of sleep apnea.[13,14] On the other hand, there was controversial evidence about the efficacy of resistance training on central sleep apnea, raising concerns about the safety and efficacy of this form of training on central sleep apnea.[5]

The patient was withdrawn from the study by the 8th week, and his sleep condition improved gradually. After then, he was asked to attend the cardiac rehabilitation center again to share in aerobic exercise, having evidence that central sleep apnea does not respond negatively to this form of training. He attended 6 weeks of aerobic exercise with the same frequency, intensity described previously. After the end of the 6-week program, the patient reported a subjective improvement in his sleep quality (SQ:1).

A one-year follow-up assessment was done for the patient to ensure he got the most out of his aerobic training. Physical examination was repeated at the National Heart Institute hospital and showed that the patient had controlled blood

Table 1 Represents the changes in the outcomes throughout the follow-up period.

Outcome	Initial assessment (IA)	8th-week assessment	% of change	1-year assessment	% of change (in comparison with IA)	% of change (in comparison with the 8th week)
SBP (mmHg)	140	150	+7.14	140	0.00	-6.66
DBP (mmHg)	90	90	0.00	90	0.00	0.00
SQ	2	3	+50.00	1	-50.00	-66.67
SL (min)	120	180	+50.00	49	-59.17	-72.78
TSD (min)	180	180	0.00	259	+43.89	+43.89
DSD (min)	60	45	-25.00	58	-3.33	+28.89
LSD (min)	120	135	+12.50	168	+40.00	+24.44
SFI (%)	52.92	72.12	+36.28	0	-100.00	-100.00
SE (%)	16	13	-18.75	21	+31.25	+61.58
6MWD (m)	220	400	+81.81	438	+99.09	+9.50
AHI	N/A	12	N/A	10	N/A	-16.67

*IA: Initial assessment, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SQ: Sleep quality, SL: Sleep latency, TSD: Total sleep duration, DSD: Deep sleep duration, LSD: Light sleep duration, SFI: Sleep fragmentation index, SE: Sleep efficiency, 6MWD: Six-minute walking distance, AHI: Apnea-hypopnea index.

pressure (140/90) and was medically stable. PSQI scores were lower than those recorded previously (moved from 3 to 1) (The same number reported after the 6-week aerobic program). The patient was instructed to wear the actigraph for 4 days again and the sleep-related outcomes were re-recorded. In comparison with the 8th week results, sleep latency decreased (SL: 180 to 49 min), and total sleep duration increased dramatically (TSD: 4 hours and 19 min). The deep sleep became 58 min after being only 45 min (the proportion of deep sleep remained the same % of TSD), and the light sleep became 2 hours and 48 minutes after being 135 min (the proportion of light sleep moved from 75% to 64% of TSD). Also, the sleep pattern became less fragmented (i.e., the patient woke up less frequently at night) and the sleep fragmentation index moved from 72.12 to 0%. In addition, sleep efficiency improved (13 to 21%). ►**Fig. 2**

Moreover, his 6MWD had improved (400 to 438 meters). A polysomnography (PSG) analysis showed signs of central sleep apnea (central apnea index >5 apnea/hour)[5] in the form of an apnea-hypopnea index (AHI) of 10 seconds.

That clearly showed that aerobic training might have a long-term positive effect on sleep and functional capacity outcomes in this patient. Also, aerobic training affected the central apnea severity recorded on the PSG (AHI decreased from 12 to 10 seconds). The changes that happened to the outcomes throughout the whole period are illustrated in ►**Table 1**.

Discussion and Conclusion

Among the reported cases, this is the first time for a morning exercise-based study to report such an adverse effect of exercising on sleep outcomes in cardiovascular patients. The exercise-based studies usually utilize actigraphy and sleep questionnaires to have a comprehensive subjective and objective assessment of sleep. In most cases, there is no need for the PSG to be used in these forms of studies, and the actigraph is preferred for many reasons.

First, an actigraph is a portable device that the patient can wear for 4-7 days and that helps the researchers doing an exercise-based study to know the bidirectional relationship between exercise and sleep, however, this is not the case with the traditional forms of PSG. Second, actigraph can record some cardiovascular parameters during activity such as training heart rate and duration of training. It records also exercise-related outcomes such as the cutpoints which represent the performance level of the subject, increasing its preference over the PSG in exercise-based studies. Consequently, as a feasible and portable option, most of the exercise-based studies aiming to improve sleep preferred the utilization of the actigraphic approach.[15–18]

In our reported case, we emphasized a well-known piece of information that it is not possible to measure sleep patterns only with actigraphy. As it is not possible to measure circadian parameters with PSG. They are related but the information they provide is not equivalent. They can be considered as complimentary to each other. Also, it's important to remind that it actigraph does not diagnose sleep apnea. In other words, if the patient has sleep deprivation due to sleep apnea, this will not be detected by the actigraph.

A novel point that can be concluded from this case is that insomnia might respond negatively to morning exercise, and this is an unfamiliar response if compared with the existing literature.[19] Also, regarding results, it's well known from the literature that functional capacity mostly correlates positively with sleep outcomes.[8–10] However, in this case, we have noticed that the functional capacity improved dramatically on doing the combined aerobic and resistance training for 8 weeks, however, his sleep outcomes deteriorated.

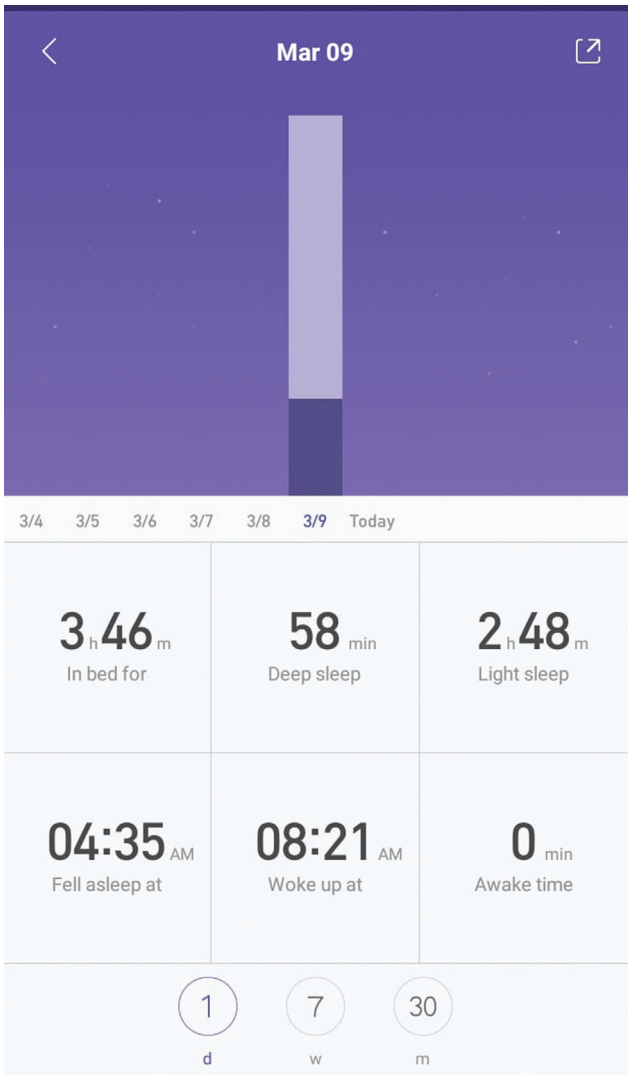

Fig. 2 Sleep parameters on the actigraph after 12 months of follow up. The actigraph display shows sleep parameters of the patient after 12 months of follow up. It shows sleep latency, fragmentation, and deep and light sleep durations.

Therefore, unexpectedly, there is no association/correlation between functional capacity and sleep outcomes.

Therefore, there are practical notes here, first, not to use actimetry (or actigraphy) to assess sleep apnea, and perhaps include other actigraphy parameters to evaluate the relation between activity during the day and increased central sleep apnea events. Secondly, if a researcher is doing an exercise-based study to improve sleep in a non-sleep apnea population, actigraph with oximetry is usually the first option of choice as a suitable methodology. However, if patients are predominately males, older adults (>60), have a history of chronic CVD, report fatigue, insomnia, hypersomnia, attention deficits, and/or morning headaches together with frequent breathing abnormalities/snoring/apnea during sleep, most probably there is a need here for further investigation (i.e. PSG) to rule out if there is any component of central sleep apnea or not. According to our case and literature as well, if the PSG confirmed the diagnosis of central sleep apnea, then higher intensities of exercise would not be recommended.[20]

However, as a limitation of this study, we could not control and/or track the confounders that might affect the sleep quality of the patient through the nine months following the termination of the follow-up aerobic training. Also, the tracking of the sleep using the actigraph was conducted on a 4 day basis per the recommendations found in the literature at the time of conduction of this study.[21] However, recent recommendations recommended a 7 days actigraph measurement to include the weekends.[22] Therefore, further research is needed to rule out the acute and long-term effects of aerobic training on central sleep apnea.

In conclusion, cardiac rehabilitation is an effective treatment post CABG. It can improve functional capacity and sleep deprivation reported after the surgery. However, in a few instances such as central sleep apnea, patients might respond negatively. Therefore, the identification of the underlying cause of the sleeping difficulty is a cornerstone of targeted treatment. Also, further research is needed to investigate the efficacy and safety of resistance training on different types of sleep apnea, and to investigate whether the use of the PSG is mandatory in these forms of exercise-based studies.

Patient Perspective

The patient's feedback was positive. He mentioned that he enjoyed the cardiac rehabilitation program and that he was happy with the quality of care provided for him.

Conflict of Interest

None declared.

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