Original Article

Cardio-autonomic functions and sleep indices before and after coronary artery bypass surgery

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Abstract:

BACKGROUND: Earlier studies showed a short-term impairment of cardio-autonomic functions following coronary artery bypass grafting (CABG). There is a lack of consistency in the time of recovery from this impairment. Studies have attributed the post-CABG atrial fibrillation to preexisting obstructive sleep apnea (OSA) without an objective sleep assessment. The aim of this study was to evaluate the effect of CABG on cardio-autonomic and hemodynamic functions and on OSA indices in patients with ischemic heart disease (IHD).

METHODS: Cardio-autonomic function using heart rate variability indices, hemodynamic parameters, and sleep studies were performed in 26 patients with stable IHD before, on day-6, and day-30 post-CABG surgery.

RESULTS: The high-frequency powers of normalized R-R intervals significantly (P = 0.002) increased from the preoperative value of 46.09 to 66.52 on day-6 and remained unchanged on day-30 postsurgery. In contrary, the low-frequency powers of normalized R-R interval decreased from 53.91 to 33.48 during the same period (P = 0.002) and remained unchanged on day 30 postsurgery. Baroreceptor sensitivity, obstructive and central apnea indices, desaturation index, and lowest O_2 saturation were not significantly different between preoperative, day-6, and day-30 postsurgery.
CONCLUSION: Our study revealed that recovery of autonomic functions following CABG occurs as early as 30 days of postsurgery. CABG does not seem to have short-term effects on sleep study indices. However, long-term effects need further evaluation.

Keywords:

Apnea/hypopnea index, heart rate variability (HRV), ischemic heart disease (IHD), sympathetic component, vagal component

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Several studies showed a strong interaction between myocardial ischemia and autonomic nervous system. Excessive sympathetic stimulation may precipitate myocardial ischemia which can trigger the activation of cardiac sympathetic nerves.[1,2] Imbalance in autonomic cardiovascular function has been shown to increase the risk for ventricular arrhythmias and sudden death in patients with coronary artery disease (CAD) and after myocardial infarction (MI).[3] On the other hand, myocardial ischemia was shown to increase sympathetic adrenergic tone and reduce parasympathetic activity,[4] a combination that augments ventricular workload and oxygen demand leading to an increase in ischemic events.

Analysis of HRV is a useful tool to assess the relationship between autonomic function and other heart diseases such as myocardial ischemia.[1] Some studies have shown that reduced HRV, mainly related to the reduced cardiac vagal activity, is associated

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with poor prognosis after MI and could predict the risk of mortality.[1,2] Restoring the blood supply of the myocardium in patients with severe IHD by coronary artery bypass surgery (CABG) has proven to be effective in reducing mortality and other complications.[5] However, the surgical procedure of CABG which includes median sternotomy, cardiopulmonary bypass (CPB), and thoracic manipulation is believed to be responsible for changes in cardiorespiratory and autonomic function observed after surgery.[6] Few studies have reported impairment in cardiac autonomic function following CABG, but there was a significant variation in the time of occurrence of this modulation, especially recovery time.[7]

Obstructive sleep apnea (OSA) is common in patients with CAD[8] and reported to increase the risk of hypertension, IHD, and stroke by increasing the sympathetic activity[9] and progression of atherosclerosis.[10] Higher risk of post-CABG atrial fibrillation was attributed to the high prevalence of OSA among IHD patients.[11] However, there was no assessment of sleep apnea indices conducted to support this attribution.

The aim of this study was to evaluate the effect of CABG on cardio-autonomic and hemodynamic functions and on OSA indices in patients with established IHD.

Methods

Study design and patients

In this cross-sectional study, all patients (aged ≥30 years) admitted for CABG in the Department of Cardiothoracic Surgery during the study period were screened for study eligibility. Patients with stroke and severe disability, decompensated heart failure (ejection fraction <35%), current arrhythmia, valvular heart disease, diabetes with HbA1C >10, uncontrolled hypertension (blood pressure [BP] >140/90 mmHg), and those on supplemental oxygen or who had changes in medication over a month prior to the study were excluded from the study. Patients underwent a detailed clinical evaluation, physical examination, and biochemical investigations. A day before the surgery (preoperative, day-0), they had an echocardiography, autonomic function test, and an overnight polysomnography in the hospital. All tests were repeated on day-6 and day-30 postsurgery. There was no change in the regular medications of the patients during the follow-up period. The protocol was approved by the Medical Research Committee of College of Medicine and Health Sciences, and was conducted in accordance with the ethical guidelines of the 1975 Helsinki Declaration. A written informed consent was obtained from all participants.

Laboratory evaluation

Biochemical investigations included HBA1C, lipid profile, electrolyte, urea, and creatinine. A two-dimensional echocardiography with M-mode recording (Vivid E9; GE Healthcare, Norway) was performed according to the American Society of Echocardiography.[12] Echocardiography studies were interpreted (blind review) by a specialized cardiologist.

Hemodynamic and autonomic measurements

Beat-to-beat hemodynamic and autonomic parameters were obtained noninvasively using the validated Task Force® Monitor (TFM V2.2, CNSystems, Graz, Austria).[13] Hemodynamic and autonomic measurements were recorded for 10 min, while the patient is at rest and breathing spontaneously. HR was calculated from 6-lead electrocardiogram (ECG) with sampling frequency of 1000 Hz. A beat-to-beat BP was also recorded using finger cuffs. Other hemodynamic parameters such as stroke volume, cardiac volume, end-diastolic volume, and total peripheral resistance were obtained using impedance cardiograph. These parameters were indexed for body surface area as stroke index (SI), cardiac index, end-diastolic index (EDI), and total peripheral resistance index (TPRI).[14]

Frequency domain analysis

An adaptive autoregressive model was used to compute HRV using beat-to-beat spectral analysis of R-R interval in the frequency domain. Ten minutes of ECG tracing free of ectopics was selected for spectral analysis. Only two of our patients had occasional ventricular ectopics on day-6. The TFM automatically computed total power of frequency domain and the absolute values and normalized units (nu) of the low frequency (LF) 0.05–0.15 Hz (RRI-LFnu) and high frequency (HF) bands 0.15–0.40 Hz (RRI-HFnu) of R-R intervals' variability and the LF 0.05–0.15 Hz band of systolic BP variability (SBP-LFnu).[15] The RRI-HFnu represented the parasympathetic modulation of the sino-atrial node. The RRI-LFnu and SBP-LFnu reflected the sympathetic modulation of the vasomotor tone.[13] Sympathovagal balance (LF/HF) was calculated as the ratio of SBP-LFnu and RRI-HFnu. Baroreceptor reflex sensitivity (BRS) was estimated using the sequence method.[13] The above autonomic parametric analyses were carried out by a blinded physician.

Time domain analysis

The RRI sequence was obtained from the TFM. Sections of these RRIs with large visible artifacts were discarded from the onset. The LF baseline trend was removed using a smoothness prior method. The following time domain parameters for HRV signal were calculated for each patient in the three groups using MATLAB Software (MathWorks Inc., Natick, MA, USA): SDNN, the standard deviation of all normal RR intervals; RMSSD, the square root of the mean of the squared successive differences in R-R intervals; and pNN50, relative number of intervals differing more than 50 ms.[16] SDNN reflects the overall

HRV and correlates with the total power from frequency domain analysis.[2] The RMSSD and pNN50 are estimates of short-term components of HRV and correlates with the HF component of frequency domain analysis.[2]

Polysomnography (sleep studies)

Portable polysomnography (PSG) was performed in all participants in an isolated and quiet room in the medical ward 1–2 days before CABG, using level 2 PSG (Alice pidex, Philips Respironics, USA). The PSG recordings include two electroencephalography leads, electro-occulogram leads, two leg electromyogram leads, two ECG chest leads, nasal airflow, chest impedance, and pulse oximetry. The PSG results were scored and reported based on the American Association of Sleep Medicine guidelines.[17] Sleep studies were repeated on day-6 and day-30 postsurgery. The PSG data obtained included number of obstructive, central, and mixed apneas, apnea-to-hypopnea index (AHI), minimum SaO_2, and desaturation index. All sleep studies were scored blindly by one experienced polysomnographer.

Surgery

All patients underwent cardiac catheterization before surgery. Results showed that 15% of the patients had left main CAD, 88% had triple vessel block, 100% had left anterior descending, and 92% had right coronary artery disease. All surgeries were done on elective basis and all patients underwent isolated on-pump coronary artery bypass grafting (CABG).

The general anesthesia protocol was similar for all patients. Patients were premedicated with an oral dose of 1–2 mg lorazepam on the night before the surgery and 1 mg on the day of surgery. Balanced anesthesia was induced with intravenous sufentanil, midazolam, and propofol titrated according to the response. They were administered rocuronium for muscle relaxation before endotracheal intubation. The anesthesia was maintained with sevoflurane.

Standard CABG was carried out under CPB, hypothermia at 30°C–32°C, and cardioplegic arrest by ante- and retro-grade multidose of cold blood cardioplegia which was used for myocardial protection during aortic cross-clamp time.

The necessity of inotropic support and the choice of inotropic drugs to be administered during weaning from CPB and in Intensive Care Unit (ICU) were noted.

Patients were transferred to the ICU after the surgery and extubated after hemodynamic and respiratory stability. Noninvasive ventilation CPAP + PS/BiPAP modalities were successfully used in case of respiratory failure after extubation as required during the ICU stay or in high dependency unit.

During the immediate postoperative period, all patients were monitored continuously using telemetry in the surgical ICU until they were clinically suitable for transfer to cardiothoracic surgery unit.

Statistical analysis

The descriptive and comparative analyses were performed using Statistical Package for the Social Sciences (SPSS version 20). Parametric data were expressed as Mean ± standard deviation. $P < 0.05$ was considered statistically significant.

Beat-to-beat measurements obtained with TFM were averaged for time periods of 10 min of rest. Normality of continuous variable was assessed with one-sample Kolmogorov–Smirnov test. One-way analysis of variance with Scheffe's *post hoc* analyses was used for comparisons of normally distributed variables of preoperative, day-6, and day-30 of hemodynamic, autonomic, PSG, and echocardiography parameters. Nonnormally distributed variables were analyzed using Kruskal–Wallis test. Autonomic parameters including total power, RMSSD, SDNN, and pNN50 were log transformed before applying statistical tests. Spearman's correlation test was used to study the relation of AHI and desaturation index with RRI-LFnu.

Results

A total of thirty patients met the selection criteria. Three patients were excluded due to off-pump surgery and one patient did not attend the postoperative follow-up studies. A total of 26 patients completed all measurements and follow-up studies. Table 1 shows the medical history, demographic data, and biochemical profiles of the studied patients. Most of the participants were males (84.6%). The mean age was 62.6 ± 9.7 years with a mean BMI of 27.6 ± 4.7 kg/m^2.

Hemodynamic parameters

Table 2 and Figure 1 summarize the changes in hemodynamic, autonomic, echocardiography, and sleep study parameters on day-0, day-6, and day-30.

There was a significant increase in HR from preoperative period to day-6 and day-30 ($P < 0.05$). Systolic, diastolic, and mean blood pressure as well as TPRI were similar pre- and post-CABG ($P > 0.05$ for all). There were significant reductions in SI ($P = 0.004$) and EDI ($P = 0.007$) from preoperative period to day-6 but showed a nonsignificant increase from day-6 and day-30. The left ventricular ejection time was significantly reduced from preoperative period to day-6 followed by significant increase from day-6 to day-30 ($P = 0.0001$) [Table 2].

Table 1: Anthropometric measurements, biochemical parameters, risk factors, and comorbidities of the patients in the study

Variable	Mean (SD)
Anthropometric parameters	
Gender (%)	
Male	22 (84.6)
Age (years)	62.6 (9.7)
Weight (kg)	74.6 (12.1)
BMI (kg/m^2)	27.6 (4.7)
Biochemical parameters	
Total cholesterol (mmol/l)	4.08 (1.0)
HDL (mmol/l)	1.04 (0.34)
LDL (mmol/l)	2.34 (0.80)
TG (mmol/l)	1.55 (0.68)
Na+ (mmol/l)	139 (2.60)
K+ (mmol/l)	4.43 (0.36)
Urea (mmol/l)	5.00 (1.49)
Creatinine (mmol/l)	72.27 (16.49
HbA1c	7.35 (1.9)
Risk factors and comorbidities (%)	
Smoking	9 (35)
Alcohol consumption	3 (11)
MI	14 (54)
Angina	26 (100)
CCF	7 (27)
DM	18 (69)
Hyperlipidemia	26 (100)
HTN	22 (85)
Respiratory disease	7 (27)
Pulmonary HTN	2 (8)

BMI=Body mass index, TG=Triglycerides, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, Hb=Hemoglobin, MI=Myocardial infarction, CCF=Congestive cardiac failure, DM=Diabetes mellitus, HTN=Hypertension

There was no change in ejection fraction and fraction shortening post-CABG ($P > 0.05$) as assessed by echocardiography [Table 2].

Autonomic parameters

The total power was significantly reduced from pre-CABG to day-6 ($P < 0.05$) and later increased from day-6 to day-30 post-CABG ($P > 0.05$). There was a significant increase in RRI-HFnu from preoperative measurement to day-6 measurement (46.09 vs. 66.52, $P = 0.002$) and remained at the same level at day-30 post-CABG. Simultaneously, there was a significant decrease in RRI-LFnu between preoperative measurements and day-6 and day-30 ($P = 0.002$ for both) [Figure 1]. There was a decrease in LH/HF ratio from pre-CABG to post-CABG day-6 and day-30; however, the differences were not statistically significant ($P = 0.87$) [Table 2]. The BRS decreased on day-6 but returned to the preoperative level on day-30, but the changes were not statistically significant ($P = 0.92$).

In time domain analysis, SDNN significantly ($P < 0.05$) decreased from pre-CABG to day-6 post-CABG. Although RMSSD and pNN50 showed a trend of decrease from pre-CABG to day-6 and increase from day-6 to day-30 post–CABG, the differences were not statistically significant.

Sleep indices

Before surgery, all the patients had an AHI of ≥30 with SaO_2 of <80% [Table 2], indicating that all the studied patients suffered from severe OSA. None of the patients had other sleep disorders.

We found no significant difference in AHI, obstructive apnea, central apnea, desaturation index, and lowest O_2 saturation between preoperative period (day-0) and postsurgery (day-6 and day-30) ($P > 0.05$ for all). In addition, there was no correlation between sleep and autonomic parameters on day-0, day-6, or day-30 [Table 3].

Discussion

The results of the present study have shown that CABG is followed by an improvement in cardiac autonomic modulation as demonstrated by the improvement in HRV parameters that started from day-6 and persisted to day-30 postoperatively. These changes were not associated with improvement in cardiac hemodynamic parameters assessed by echocardiography or any changes in OSA indices.

Hemodynamic and autonomic parameters

Some studies showed that, following cardiac procedures such as cardiac valve surgery or CABG, measurements such as HRV, LF, HF, SDNN, and RMSSD might be reduced.[6,7,18,19] The decrease in HRV is attributed to the combined effects of surgical manipulation of the heart and allied structures, anesthesia, cardioplegia, and extracorporeal circulation.[6,7,20] Our study showed an overall decrease in HRV, however, with a significant increase in RRI-HFnu (vagal component) and a significant decrease in RRI-LFnu (both sympathetic and vagal component) between preoperative measurements and day-6 and day-30. This is contrary to the previous observation[7] which showed an increase in LF after CABG. Our study showed an insignificant decrease in LF/HF and RMSSD. Previous studies have also shown post-CABG decrease in RMSSD.[7,18] The RMSSD is an indicator of vagal component, and decrease in RMSSD may indicate decrease in parasympathetic control over the heart which may not be true. We propose that proportion of RMSSD out of SDNN (RMSSD/SDNN: vagal component out of the total variability from time domain analysis) is a better representation of vagal control. This study indicated average increase of RMSSD/SDNN from pre-CABG to day-6 post-CABG (pre-CABG: 0.838; day-6 CABG: 1.103; and day-30 CABG: 0.8475). Although

Table 2: Hemodynamic, autonomic, and sleep parameters before and after coronary artery bypass surgery

	Mean (SEM)			*P*
	Pre-CABG (*n*=25)	Day-6 (*n*=25)	Day-30 (*n*=25)	
Hemodynamic parameters				
Heart rate (bpm)	70.12 (2.43)	87.45 (1.60)	80.71 (2.53)	0.0001*,§
Systolic BP (mmHg)	111.90 (3.45)	116.29 (3.14)	115.41 (2.77)	0.69
Diastolic BP (mmHg)	68.28 (3.62)	68.78 (3.54)	74.16 (1.58)	0.22
Mean BP (mmHg)	81.53 (2.66)	82.24 (2.52)	84.07 (1.75)	0.77
Stroke index (ml/M^2)	32.03 (1.53)	26.30 (0.97)	29.71 (1.17)	0.007*
Cardiac index (L/min×m^2)	2.187 (0.78)	2.289 (0.08)	2.349 (0.09)	0.39
Total peripheral resistance index (dyne×s×m^2/cm^2)	2989.1 (141.8)	2902.5 (153.1)	2897.8 (112.6)	0.73
End diastolic index (ml/m^2)	52.79 (2.09)	44.39 (1.44)	49.94 (1.65)	0.004*
Left ventricular ejection time (ms)	297.0 (4.58)	269.4 (3.27)	285.5 (5.17)	0.0001*,#
Thoracic fluid content (1/ohm)	25.35 (0.627)	30.01 (1.06)	28.12 (0.90)	0.002*
Echocardiography parameters				
Ejection fraction (%)	53.15 (3.04)	52.69 (2.48)	56.00 (2.03)	0.08
Fraction shortening (%)	29.42 (1.45)	27.80 (1.86)	25.46 (2.49)	0.34
Frequency domain autonomic parameters				
RRI-LFnu (%)	53.91 (3.66)	33.48 (4.73)	36.44 (4.24)	0.002*,§
RRI-HFnu (%)	46.09 (3.66)	66.52 (4.73)	63.56 (4.24)	0.002*,§
Total power (ms^2)^	6.41 (1.75)	3.83 (1.20)	5.23 (1.85)	0.0001*,#,§
LF/HF	1.776 (0.28)	1.516 (0.59)	1.214 (0.42)	0.64
Total baroreceptor sensitivity (ms/mmHg)	13.02 (2.92)	6.195 (1.31)	12.01 (3.97)	0.42
Time domain autonomic parameters				
SDNN (ms)^	2.78 (0.63)	2.30 (0.54)	2.56 (0.86)	0.030*
RMSSD (ms)^	2.73 (0.10)	2.58 (0.57)	2.68 (0.92)	0.182
pNN50 (ms)^	1.01 (1.9)	0.11 (1.56)	0.95 (2.12)	0.514
Sleep parameters				
AHI	37.05 (4.95)	36.75 (5.59)	34.88 (5.84)	0.88
Obstructive apnea	21.29 (6.08)	37.13 (21.11)	29.69 (11.51)	0.76
Central apnea	12.71 (4.02)	6.88 (3.90)	32.75 (18.14)	0.19
Mean saturation index	3.475 (1.15)	7.812 (6.57)	0.9375 (0.32)	0.24
Hypopnea	121.71 (18.87)	118.63 (21.62)	106.06 (21.51)	0.66
Desaturation index	21.38 (3.27)	19.21 (3.13)	23.94 (5.27)	0.52
Lowest O_2 saturation	82.81 (1.46)	80.29 (1.21)	83.56 (2.14)	0.27

*Pre-CABG versus day-6=*P*<0.05, #Day-6 versus day-30=*P*<0.05, §Pre-CABG versus day-30=*P*<0.05, ^Total power is expressed in log. NS=Nonsignificant, BP=Blood pressure, LH=Low frequency, HF=High frequency, AHI=Apnea-to-hypopnea index, CABG=Coronary artery bypass surgery, SEM=Standard error mean, SDNN=The standard deviation of all normal RR intervals, RMSSD=The square root of the mean of the squared successive differences in R-R intervals

Table 3: Correlation of apnea-to-hypopnea index and desaturation index with RRI-LFnu

	AHI			Desaturation index		
	Day-0 *r* (*P*)	Day-6 *r* (*P*)	Day-30 *r* (*P*)	Day-0 *r* (*P*)	Day-6 *r* (*P*)	Day-30 *r* (*P*)
RRI-LFnu						
Day-0	0.061 (0.77)			0.308 (0.15)	0.279 (0.29)	0.256 (0.32)
Day-6	0.192 (0.09)			0.432 (0.07)	0.298 (0.22)	0.199 (0.41)
Day-30	0.072 (0.73)	0.014 (0.95)	0.044 (0.85)	−0.187 (0.43)	0.170 (0.47)	−0.010 (0.96)

r (*P*)=Correlation (*P* value), There was no correlation between sleep and autonomic parameters on day-0, day-6, or day-30. AHI=Apnea-to-hypopnea index

there is a trend supporting the improvement of vagal control from time domain analysis, these changes were not significant. Our proposal needs further evaluation. Altered HR dynamics have been associated with myocardial ischemic episodes in patients post-CABG.[21] Sympathetic activation increases cardiac oxygen demand causing myocardial ischemia in patients with CAD. This suggests a key role of the autonomic nervous system in the pathogenesis of myocardial ischemia in the postoperative phase of CABG.[21] Patients recovering from CABG might, therefore, be at an increased risk of myocardial ischemia and arrhythmias at about postoperative day 3, when adrenergic influence is higher and vagal modulation is at its lowest. In our study, the postoperative improvement of vagal component of HRV might be at least partly attributed to the improvement in cardiac function as reflected by the significant reduction of SI and EDI. These findings may indicate the recovery of cardiac muscle function resulting from postsurgical myocardial reperfusion.[22] Patients with reduced left

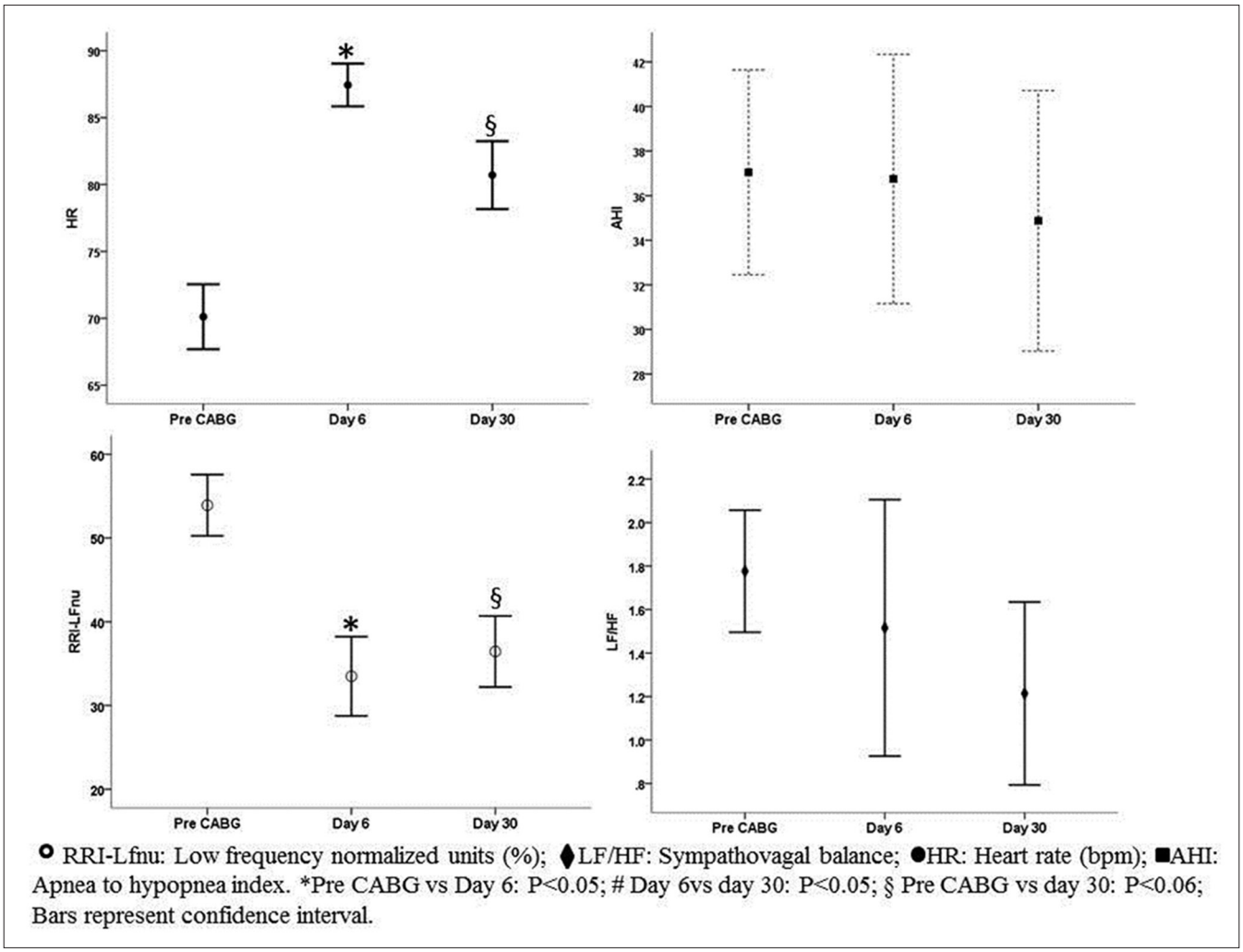

Figure 1: Changes in RRI-LFnu, low frequency/high frequency, heart rate, and apnea-to-hypopnea index before coronary artery bypass grafting and on day-6 and day-30 postsurgery

ventricular ejection fraction (LVEF) were found to have reduced long-term survival after CABG.[23] It is possible that post-CABG improvement in RRI-HFnu and RRI-LFnu in our study could be due to the selection of patients with good LV function, controlled diabetes, and without valvular disease. Greatly reduced LV function and low EF,[5] uncontrolled diabetes,[24] and valvular disease[25] are known to be associated with morbidity and mortality after CABG. Our findings are unlikely to be due to the type of surgery as all patients in our study underwent on-pump CABG. Furthermore, studies have shown similar effects of off- and on-pump CABG surgeries on autonomic impairment and adrenergic activity.[20,26]

Our findings are in accordance with other studies who reported autonomic recovery after CABG.[6,7] Our results showed that most of the recovery occurred after 30 days compared to studies that reported recovery of cardiac functions after 60 days.[7] Late recovery of HRV may be due to resolution of ischemia or the use of angiotensin-converting enzyme inhibitors.[6] Studies have shown that reduced HRV after CABG may not have relevance in predicting mortality as it has in predicting outcome in patients with sustained MI.[20]

An estimation of baroreflex sensitivity (BRS) is one of the important measures of autonomic nervous system balance in the body.[27] La Rovere *et al*.[3] studied the independent prognostic value of BRS to reassess the value of HRV when added to simple clinical factors such as LVEF and ventricular premature complexes which are already known to predict outcome after MI. The BRS estimated on day-16 of CABG by injecting phenylephrine clearly indicated its prognostic value independent of LVEF and ventricular arrhythmias. We did not find any significant difference in BRS before and after CABG.

Sleep parameters

All participants of this study had severe OSA (AHI ≥30). Previous study also reported high prevalence of

sleep breathing disorder in patients with IHD.[28] The independent association of OSA with postoperative complications has been documented.[29] The available evidence suggested that treatment of patients with CADs with continuous positive airway pressure (CPAP) can reduce cardiovascular events and mortality.[30] Therefore, screening for sleep disorders and treatment of OSA with CPAP should be considered in all patients referred for CABG.

There were no significant differences in respiratory or central events of sleep disturbances at all the three stages of this study which suggested that CABG surgery may not worsen sleep apnea indices. Earlier studies reported a higher risk of post-CABG atrial fibrillation and it was attributed to the high prevalence of OSA among IHD patients.[11] However, postsurgery assessment of OSA was not conducted in that study. Other studies have reported an association of post-CABG atrial fibrillation with other factors such as low levels of Vitamin D.[31] There is a possibility of fluid overload during on-pump CABG surgery. It is not known if CABG would cause fluid congestion leading to upper airway obstruction. Studies showed that these complications could be partially reverted with CPAP treatment.[10] CPAP was recommended to all patients of this study although it was not initiated during the study period, as the study was not designed to assess the effect of CPAP.

Limitations

The results of our study should be interpreted within the context of its limitations. Our study included patients with good LV function, controlled hypertension, and diabetes. Therefore, the result could not be extrapolated to patients with more severe comorbidities. Patients were followed up for 30 days postoperatively and this does not reflect the long-term effect of CABG on the autonomic, hemodynamic, and sleep parameters.

Conclusion

Our study revealed that recovery of autonomic functions following CABG could occur as early as 30 days postsurgery. CABG does not seem to have short-term effects on sleep study indices. Further studies are needed to evaluate the long-term effect of CABG on sleep and autonomic indices.

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Conflicts of interest

There are no conflicts of interest.

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