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Alterations in Temporal Patterns of Heart Rate Variability after Coronary Artery Bypass Graft Surgery

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Background: Preliminary studies have indicated that autonomic nervous system dysfunction may be present in patients after cardiac surgery. The purpose of this study was to evaluate cardiac autonomic nervous system function, as assessed by analysis of heart rate variability (HRV), in adult patients undergoing uncomplicated coronary artery bypass graft surgery.

Methods: Longitudinal changes in HRV were determined perioperatively by continuous electrocardiographic monitoring in 40 adult patients undergoing elective coronary artery bypass graft surgery and were compared with HRV in two groups of control subjects: 15 patients undergoing nonthoracic major vascular surgery and 19 healthy volunteers. Exclusion criteria were diabetes, renal failure, recent or perioperative myocardial infarction, or use of inotropic drugs. HRV data during electrocardiographically documented episodes of myocardial ischemia were omitted.

Results: There were no differences in any measurement of preoperative HRV between groups during the day, but HRV was greater at night (12:00 AM to 5:00 AM) in volunteers than in patients in either surgical group. In the hour after induction of anesthesia (before cardiopulmonary bypass), the components of HRV were decreased compared with those in the preoperative daytime but were similar in the two surgical groups. After surgery, HRV in the group undergoing nonthoracic vascular surgery remained at about the same level as that observed after induction of anesthesia, whereas in the group undergoing coronary artery bypass graft surgery, HRV was further reduced and was approximately 40–50% less than that in the vascular surgery group ($P < 0.05$). In the coronary artery bypass group, the reduction in HRV compared with the pre-

operative daytime measurements persisted on postoperative day 5.

Conclusions: HRV is reduced after uncomplicated coronary artery bypass graft surgery. Although we cannot exclude the effects of uncontrolled variables in this reduction of postoperative HRV, the observed changes in HRV did not appear to result from general anesthesia, perioperative stress responses, and other factors associated with the early postoperative period. These data are consistent with the supposition that cardiac autonomic nervous system function is impaired after cardiac surgery. (Key words: Autonomic nervous system. Heart: heart rate: heart rate variability. Surgery: cardiac.)

IMPAIRMENT of cardiac autonomic nervous system (ANS) function after cardiac surgery involving cardiopulmonary bypass (CPB) has been observed in several laboratory and clinical investigations.^{1–5} Extensive evaluation of cardiac autonomic function after cardiac surgery in patients, however, has not been reported. Analysis of heart rate variability (HRV) provides information about cardiac sympathetic and parasympathetic nervous system function.^{6–9} Preliminary evidence suggests reductions in spectral measurements of HRV for as long as 6 weeks after cardiac surgery.^{4,5} Although such reductions in HRV may represent a manifestation of impaired ANS function after cardiac surgery, these HRV changes also may be explained by other factors, including nonspecific effects of recovery from general anesthesia, perioperative stress responses, pain, recent myocardial infarction, myocardial ischemia, reduced left ventricular function, or concomitant medication (e.g., inotropic or anticholinergic drugs).^{1–3,10–23}

There is growing appreciation of the relation between cardiac sympathovagal imbalance and adverse outcomes in other disease processes of the heart as well as after noncardiac surgery.^{16–19,22} Thus, understanding whether reduced HRV after cardiac surgery is the result of nonspecific perioperative factors or the result of perturbations associated with cardiac surgery could be of clinical importance. The purpose of this study,

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HEART RATE VARIABILITY AFTER CORONARY ARTERY SURGERY

therefore, was to evaluate cardiac ANS function after cardiac surgery by the use of HRV analysis in adults having uncomplicated coronary artery bypass graft (CABG) surgery and to compare it with HRV in patients undergoing nonthoracic major vascular surgery and in a cohort of healthy, nonhospitalized volunteers.

Materials and Methods

The protocol used in this study was approved by our institutional Human Studies Committee, and procedures were performed after receiving individual informed consent. Forty adult patients scheduled for elective CABG surgery (group CABG) and 15 patients scheduled for nonthoracic major vascular surgery (group VASC) underwent continuous electrocardiographic (ECG) monitoring with AM Holter recorders (Marquette Electronics, Milwaukee, WI) using leads CC₅ and II. Eleven patients from group VASC underwent repair of an infrarenal abdominal aortic aneurysm, and 4 underwent aortobifemoral bypass grafts for aortic occlusive disease. Holter monitoring was initiated on the afternoon before surgery and was continued until the 2nd postoperative day, including the intraoperative period but excluding CPB in group CABG. Further Holter monitoring was performed in 10 CABG patients on the 5th postoperative day. In addition, a group of healthy nonhospitalized volunteers underwent 24-h Holter monitoring as outpatients. Volunteers had a normal resting 12-lead ECG and had no history of coronary artery disease or hypertension (blood pressure <160/95 mmHg) and were receiving no medication.

Exclusion criteria for surgical patients were digoxin use, diabetes mellitus, renal failure requiring hemodialysis, congestive heart failure, myocardial infarction within 6 months of the study, atrial fibrillation, or presence of a permanent cardiac pacemaker before surgery. Patients also were excluded if they required perioperative inotropic support, an intraaortic balloon pump, or tracheal intubation for ventilatory support after postoperative day 1, or if postoperative myocardial infarction developed (new ECG Q wave and increases in MB isoenzymes of creatine kinase >50 or >12 IU for cardiac and noncardiac surgical patients, respectively).

Anesthetic Management

All cardiac medications, including nitroglycerin, β -adrenergic receptor, and calcium channel-blocking drugs, were continued until the time of surgery. With

the exception of nitrates, cardiac medications were not continued after surgery. In group CABG patients, premedication included intramuscular or subcutaneous morphine sulfate (0.08–0.1 mg/kg) and oral lorazepam (1–2 mg) given 90 min before induction of anesthesia. In these patients, anesthesia was induced and maintained with fentanyl 30–40 μ g/kg and supplemental isoflurane. Vecuronium and metocurine were given for skeletal muscle relaxation. After orotracheal intubation, patients' lungs were mechanically ventilated at 10 breaths/min and at an inspired oxygen fraction of 1.0. Lorazepam 1–2 mg was administered intravenously at the start of rewarming during CPB.

In group VASC, patients were premedicated with a benzodiazepine; anesthesia was induced with thiopental (4 mg/kg) or etomidate (0.2–0.3 mg/kg). Patients also received fentanyl (30–60 μ g/kg) in divided doses, vecuronium, and isoflurane. At the completion of surgery anticholinergic and anticholinesterase drugs were not administered to reverse the effects of muscle-relaxing drugs. Before and after surgery group VASC patients were given morphine epidurally *via* a lumbar catheter. After surgery, all patients were admitted to an intensive care unit (ICU) and received ventilatory support until postoperative day 1. Patients from both surgical groups received routine postoperative care as determined by attending physicians, including administration of parental morphine as needed for pain and phenylephrine and sodium nitroprusside for systemic systolic hypo- and hypertension, respectively.

Surgical Management

CPB was accomplished with centrifugal pumps (Bio-Medicus, Minneapolis, MN) and membrane oxygenators (Cobe, Denver, CO). High-potassium, cold cardioplegia was administered after aortic cross-clamping during systemic hypothermia (venous temperature of approximately 25–28°C). Myocardial temperatures were intermittently measured and typically were 11–14°C after the administration of cardioplegia. Topical myocardial cooling techniques included application of cold saline after aortic cross-clamping or use of topical iced "slush" or external cooling "jackets" (Shiley, Irvine, CA).

Analysis of Heart Rate Variability

Holter tape analysis was performed with a computerized scanner (Marquette 8000) with version 5.8 software and standard QRS labeling techniques. Computer classifications were edited by a single investigator

to ensure proper QRS labeling. Spectral analysis of HRV was performed on normally classified beats (*i.e.*, excluding ectopic and paced beats resulting from temporary cardiac pacing in group CABG) with Marquette software version 002A using fast-Fourier transformation. Spectra were reported as power amplitude (milliseconds per Hertz) every 2 min and divided into low-frequency (LF) (0.04–0.15 Hz), high-frequency (HF) (0.15–0.40 Hz), and total power amplitudes (TPA) (0.01–1.0 Hz). Also, the LF/HF ratio was determined. Analysis of HRV was performed on hourly spectral summaries. Because of the potential effects of myocardial ischemia on HRV, hourly HRV measurements in which a myocardial ischemic episode occurred, as detected by shifts of the ST segment, were omitted from analysis.^{19,20,23} The criteria used for the diagnosis of myocardial ischemia were ST-segment depression greater than 1 mm or elevation greater than 2 mm occurring 60 ms after the J-point, as previously described.²⁴

Statistical Analysis

The perioperative period was divided into pre-, intra-, and postoperative study intervals. The preoperative data and those from the healthy volunteers were classified as daytime (12:00 PM to 12:00 AM) or nighttime (12:00 AM to 5:00 AM) intervals to account for diurnal fluctuations in HRV.^{25,26} To examine effects of anesthetics on HRV, findings are also reported from the hour after induction of anesthesia. The immediate ICU and postoperative day 1 periods were divided into the following intervals: ICU evening, ICU admission to 12:00 AM; night, 12:00 AM to 5:00 AM; morning, 5:00 AM to 12:00 PM; afternoon, 12:00 PM to 6:00 PM; and evening, 6:00 PM to 12:00 AM. The measurement periods from the 5th postoperative day were divided into daytime (12:00 PM to 12:00 AM) and nighttime (12:00 AM to 5:00 AM) intervals.

Spectral amplitudes were converted to their natural logarithms and subjected to one-way analysis of variance with Bonferroni's *post hoc* test. Categorical data between groups were compared by chi-square testing and other between groups comparisons were by analysis of variance with Bonferroni's test. A significant difference was considered to exist when $P < 0.05$. All values are expressed as mean \pm standard error.

Results

Patient characteristics and preoperative medications for each group are listed in table 1. There were no

Table 1. Patient and Nonsurgical Volunteer Demographics and Medication

	Group CABG (n = 40)	Group VACS (n = 15)	Volunteers (n = 19)
Age (yr) (mean \pm SE)	59.3 \pm 1.6	61.3 \pm 1.62	62.7 \pm 1.81
Male/female	35/5	11/4	14/5
History of hypertension	16	7	0
Medication			
Beta-blockers	20	0	0
Calcium-channel blockers	24	5	0
Nitrates	29	1	0
ACE inhibitors	1	2	0

differences between patient groups with respect to age or gender; however, preoperative medications did vary between groups. Nine group CABG patients received lidocaine for less than 18 h immediately after surgery. Heart rate results observed in volunteers and in patients is shown in table 2. There were no differences in heart rates between groups after surgery.

Holter monitoring was performed in group CABG for 17.9 \pm 4.1, 3.7 \pm 0.9, and 36.8 \pm 10.4 h in the pre-, intra-, and postoperative periods, respectively. In group VASC, Holter monitoring was performed for 16.1 \pm 1.8, 4.5 \pm 1.2, and 34.5 \pm 9.1 h in the pre-, intra-, and postoperative periods. In the preoperative period 13 patients in group CABG experienced 41 ECG episodes of myocardial ischemia, and 3 patients in group VASC had 11 ischemic episodes. Intraoperatively, 6 ECG ischemic episodes were detected in 5 group CABG patients and 5 in 1 group VASC patient. Postoperatively, 21 patients in group CABG and 2 in group VASC had 39 and 20 ECG ischemic episodes, respectively. The durations of the myocardial ischemic episodes detected by ECG in the pre-, intra-, and postoperative periods were 23.2 \pm 12.7, 0.81 \pm 0.3, and 103.7 \pm 34.7, respectively, in group CABG and 9.1 \pm 6.8, 1.6 \pm 1.6, and 20.2 \pm 14.3 min, respectively, in group VASC.

Preoperative and Volunteer Heart Rate Variability

Results of HRV analysis from the preoperative periods and from the outpatient Holter session performed in the volunteers are listed in table 3. There were no significant differences among groups in any measurement of HRV during the day. Volunteers demonstrated a significant increase in all measurements of HRV at night compared with that during the day. In volunteers, LF

HEART RATE VARIABILITY AFTER CORONARY ARTERY SURGERY

Table 2. Perioperative and Volunteer Heart Rates

	Group CABG	Group VASC	Volunteers
	Preoperative		
Day	67 ± 1	73 ± 2*	81 ± 2†
Night	62 ± 1	71 ± 3*	62 ± 1‡
Anesthesia	62 ± 2	63 ± 1	—
ICU evenings	96 ± 1	94 ± 2	—
ICU night	93 ± 2	94 ± 2	—
POD1 morning	92 ± 2	99 ± 4	—
POD1 afternoon	93 ± 2	100 ± 4	—
POD1 evening	93 ± 2	102 ± 3	—
POD1 night	92 ± 2	104 ± 3	—

Values are mean ± SE (beats/min).

ICU = intensive care unit; POD1 = postoperative day 1.

* $P < 0.05$ group VASC versus group CABG.

† $P < 0.05$ Volunteer versus groups CABG and VASC.

‡ $P < 0.05$ Volunteer versus group VASC.

and TPA but not HF for the night period were significantly greater than those during the night in group CABG. All measurements of HRV from the night period in the volunteer group were significantly greater than those observed at night in group VASC. The LF/HF ratio in volunteers was greater during the day (2.6 ± 0.6) than it was in groups CABG (2.1 ± 0.6) and VASC (1.9 ± 0.4) ($P < 0.05$) but was not different at night (volunteers 2.2 ± 0.6 , group CABG 2.1 ± 0.6 , and group VASC 2.0 ± 0.6).

Perioperative Heart Rate Variability

Comparisons of LF, HF, and TPA obtained throughout the perioperative periods in groups CABG and VASC are shown in figure 1. There were no differences in HRV between surgical groups in the hour after induction of anesthesia. In the immediate postoperative period, mean LF and TPA in group CABG were approximately 50% less and HF was 40% less than that observed in group VASC ($P < 0.05$). The LF/HF ratio in group CABG was less than that observed in group VASC during the day in the ICU (1.6 ± 0.6 vs. 2.0 ± 0.4), during the evening in the ICU (1.5 ± 0.6 vs. 2.0 ± 0.9), and postoperative day 1/morning (1.5 ± 0.5 vs. 2.0 ± 0.8) ($P < 0.05$). Differences in postoperative HRV between surgical groups persisted when performing analysis only on data from those patients without ECG evidence of myocardial ischemia at any time after surgery. The temporal patterns of HRV in groups VASC and CABG also appeared to be different after surgery when com-

pared with the hour after induction of anesthesia and with HRV measured within each group preoperatively (fig. 1). In group VASC, postoperative indexes of HRV remained at about the same level as that observed in this group the hour after induction of anesthesia. In contrast, in group CABG all indexes of HRV were less after surgery when compared with those after induction of anesthesia and with HRV measured during the preoperative day.

Longitudinal changes in all measurements of perioperative HRV from the 10 patients in group CABG who underwent additional Holter monitoring on the 5th postoperative day are shown in figure 2. In this group, HRV on the 5th postoperative day remained decreased compared with the preoperative day values. These postoperative reductions in HRV on postoperative day 5 observed in this subset of group CABG patients, when compared with the preoperative day values, were significant for LF and TPA during the daytime (12:00 PM to 12:00 AM).

Perioperative HRV values from patients in group CABG who were receiving β -adrenergic-receptor blocking drugs before surgery are compared with values from patients who were not receiving these drugs in figure 3. During all preoperative study periods, measurements of HRV were significantly greater in patients receiving β -blocking agents compared with those who were not, with the exception of TPA during the preoperative night period. These differences persisted in the hour after induction of anesthesia. After surgery,

Table 3. Preoperative and Nonsurgical Volunteer HRV

	Group CABG	Group VASC	Volunteers
Day			
LF	16 ± 1	15 ± 1	18 ± 1
HF	9 ± 1	8 ± 1	7 ± 1
TPA	26 ± 2	25 ± 2	28 ± 2
Night			
LF	19 ± 1	15 ± 2	26 ± 1*†
HF	10 ± 1	8 ± 1	12 ± 1†‡
TPA	31 ± 2	26 ± 2	40 ± 2*†

Values are mean ± SE (power amplitude).

LF = low frequency (0.04–0.15 Hz); HF = high frequency (0.15–0.40 Hz); TPA = total power amplitude (0.01–1.0 Hz).

* $P < 0.05$ Volunteer versus groups CABG and VASC.

† $P < 0.05$ versus Day within group.

‡ $P < 0.05$ Volunteer versus group VASC.

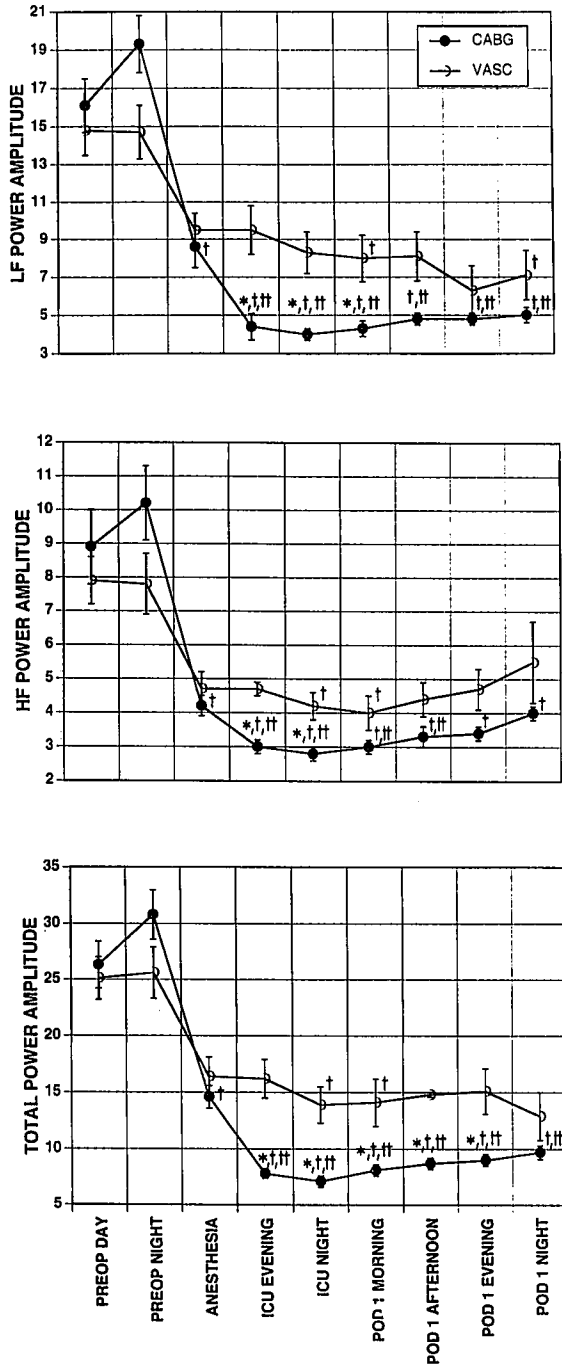


Fig. 1. Low frequency (LF) (0.04–0.15 Hz), high frequency (HF) (0.15–0.40 Hz), and total power amplitude (0.01–1.0 Hz) by surgical group for each perioperative period. ICU = intensive care unit; POD = postoperative day. * $P \leq 0.05$ group undergoing coronary artery bypass graft surgery (CABG) versus group undergoing nonthoracic vascular surgery (VASC); † $P \leq 0.05$ versus preoperative (PREOP) day period within group; †† $P \leq 0.05$ versus anesthesia period within group.

blocking agents was not different than that in patients not receiving these drugs.

Discussion

The results of this study show that after CABG, HRV is reduced compared with that observed before surgery and after induction of anesthesia, as well as compared with HRV reductions observed after major vascular surgery.

Investigations of the physiologic processes influencing HRV have focused, for the most part, on the LF and HF components of the heart rate spectrum. The HF component of HRV is influenced by the parasympathetic nervous system, whereas the LF component is influenced by both the sympathetic and parasympathetic nervous systems.^{6–9} Acute increases in sympathetic activity can increase the LF component of HRV; sustained sympathetic activation has been shown to reduce it.^{6–9,27} The parasympathetic modulation of LF is demonstrated by increases and decreases in LF after the administration of β -adrenergic-receptor and muscarinic-receptor blocking drugs, respectively.^{6–9,26,28}

HRV can be affected by many factors, including age, myocardial ischemia and infarction, poor left ventricular function, and possibly inotropic drugs.^{9,16–21,29,30} We have attempted to control for the effects of most of these variables by including two control groups and by observing strict inclusion criteria including the exclusion of patients with recent or perioperative myocardial infarction. Comparison of preoperative day HRV in surgical patients with that in volunteers suggests that our baseline HRV results are representative of a group similar in age and gender. Thus, the changes in postoperative HRV we observed are a manifestation of some combination of disease process and factors related to the intra- and postoperative periods.

In the current study we also have attempted to control for the effects of myocardial ischemia on HRV by omitting HRV data obtained during periods of ST-segment shifts compatible with myocardial ischemia. Due to the inherent limitations of myocardial ischemia detection with ECG compared with other diagnostic techniques (e.g., echocardiography), differences in the frequency of myocardial ischemia between groups cannot be excluded as influencing our HRV results. Differences in postoperative HRV between groups CABG and VASC persisted, however, even after excluding from analysis patients with ECG ischemic episodes at any time postoperatively. This suggests that ischemic induced

HEART RATE VARIABILITY AFTER CORONARY ARTERY SURGERY

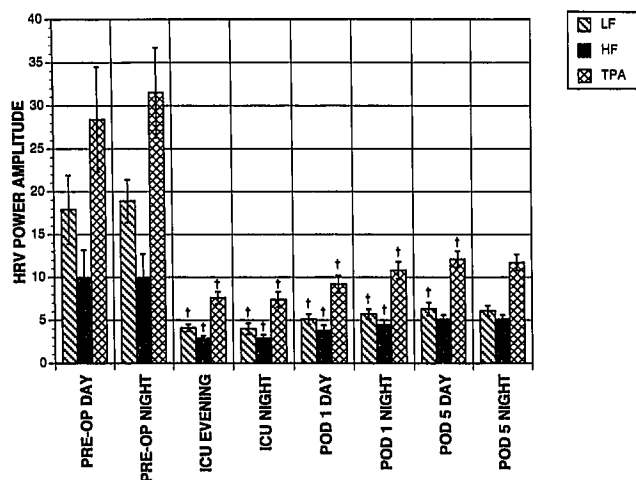


Fig. 2. Frequency components of heart rate variability for the group undergoing coronary artery bypass graft surgery (CABG) by perioperative period, including postoperative day 5. ICU = intensive care unit; POD = postoperative day; LF = low frequency (0.04–0.15 Hz); HF = high frequency (0.15–0.40 Hz); TPA = total power amplitude (0.01–1.0 Hz). † $P \leq 0.05$ versus preoperative (PREOP) day period.

changes in HRV persisting after the resolution of an ECG myocardial ischemic episode probably had little effect on the reduced HRV in group CABG. Moreover, differences in heart rates (table 2) does not explain the postoperative HRV differences between groups.

Residual effects of anesthetics and analgesics could also account for the reductions in HRV observed in the postoperative period.^{10–15} However, similar anesthetics were administered in demographically comparable patients undergoing noncardiac procedures. Also, postoperative HRV was reduced compared with that observed after induction of anesthesia in group CABG further suggesting that residual anesthetics cannot explain the magnitude of reduction in postoperative HRV (fig. 1). Although not directly compared in the past to our knowledge, sympathetic nervous system activation occurs after both cardiac and noncardiac vascular surgery and maybe influenced by epidural morphine administration.^{2,31–33} Severity in postoperative pain was not assessed in the current study and could have potentially been different between surgical groups in light of the differences in types of surgical procedures and postoperative pain management techniques. Although we cannot disprove this as a factor causing the differences in postoperative HRV between groups, we speculate that the magnitude and duration of the reduction in HRV in group CABG is not solely explained by differ-

ences in pain or differences in the perioperative stress response.

The effect of β -adrenergic-receptor blocking drugs on HRV was also examined. As have others, we observed an increase in all preoperative measurements of HRV in group CABG patients receiving compared with those not receiving β -adrenergic-receptor blocking agents (fig. 3).^{6,9,26,28} Although β -blocking drugs were continued up to the morning of surgery, they were not administered postoperatively. Nonetheless, the differences in HRV observed preoperatively and after induction of anesthesia in patients receiving and those not receiving β -blocking agents were not evident after surgery, suggesting that HRV-reducing factors were operative in both groups at this time. Some patients had been receiving calcium channel-blocking drugs or angiotensin-converting-enzyme inhibitors, but the former have been demonstrated to have little effect on HRV and the latter may actually increase HRV.^{28,34} Also, nine patients in group CABG received an infusion of lidocaine in the immediate postoperative period. Antiarrhythmic drugs with vagolytic properties may reduce HRV.³⁵ The effects of lidocaine on HRV, however, are not known. Thus, perioperative administration of cardioactive drugs cannot explain the postoperative reduction in HRV we observed. Acute administration of sodium nitroprusside increases LF, whereas phenylephrine can be expected to increase HF.^{6–9} The use of the latter drugs for short intervals after surgery would appear to have little influence on our HRV results.

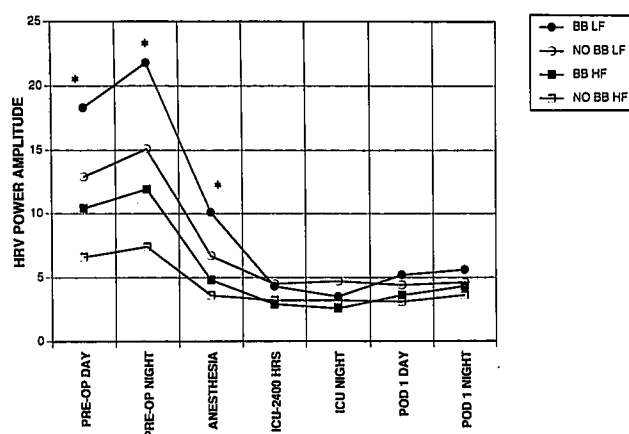


Fig. 3. Perioperative low- and high-frequency components of heart rate variability (HRV) power amplitude in patients undergoing coronary artery bypass graft surgery and receiving or not receiving β -adrenergic-blocking drugs (BB or NO BB, respectively). * $P \leq 0.05$, BB versus NO BB.

Thus, it is conceivable that the reduction in HRV we observed after surgery in group CABG was the result of factors associated with CPB and the surgical procedure. Effects of aortic cross-clamping and cardioplegia also cannot be excluded as contributing to the reduction in group CABG.^{20,23} It is unknown whether such potential causes of reduced postoperative HRV in group CABG would persist for a period as extended as that we observed.

Analysis of HRV provides information only about target-organ responses to cardiac ANS activity and not information about the actual functioning of autonomic nerves. Thus, the exact mechanism of the reduction in HRV in group CABG cannot be identified with the methods used in this study. Nonetheless, the reduced HRV we observed after CABG surgery may reflect a manifestation of cardiac autonomic dysfunction, as previously suggested.¹⁻³ In this regard, Murphy and Armour¹ have observed blunted effects of stellate ganglion stimulation on atrial and ventricular function in animals after sham cardiac surgery. Because the chronotropic and inotropic responses to isoproterenol were preserved, alterations of β -receptors or direct cardiac myocyte damage could not explain the blunted sympathetic nervous systems response observed by Murphy and Armour.^{1,36,37} Investigations of cardiac parasympathetic function have shown no change or decreased vagally mediated activity in an animal model and patients after cardiac surgery.^{1,3}

Injury to the phrenic nerve during the course of cardiac surgery is well described and believed to result from contact of the nerve with intrapericardiac iced "slush."³⁸ Autonomic nerves have been demonstrated to remain structurally intact after hypothermic CPB with cold cardioplegic arrest.³⁹ Murphy and Armour¹ have speculated that the more superficial epicardial course of sympathetic postganglionic fibers, in contrast to the more subendocardial location of parasympathetic fibers, might increase the susceptibility of the sympathetic nerves to injury from topical cooling during cardiac surgery.¹ Finally, our data suggest that recovery of autonomic function, although not complete, occurs after surgery, corroborating that there was no permanent impairment of the autonomic nerves resulting from surgical transection.

Clinical Implications

ANS dysfunction after CABG surgery has several important clinical implications, including the possible cardiovascular instability that could result from ob-

tunded ANS reflex augmentation of heart rate and myocardial contractility in response to the multiple hemodynamic and pharmacologic perturbations that may occur perioperatively. Because ANS dysfunction may increase susceptibility to anesthetic induced hypotension and bradycardia, our findings suggest that patients with prior cardiac procedures maybe predisposed to these anesthetic hemodynamic derangements, especially immediately after surgery.^{14,40} The presence of impaired autonomic regulation of heart rate after CABG surgery could explain, in part, the tachycardia that is frequently observed in the immediate postoperative period. Also, autonomic nerve dysfunction has been shown to be associated with silent myocardial ischemia in patients with diabetes mellitus.^{41,42} Impairment of cardiac autonomic nervous function after CABG surgery, if present, could have the potential to contribute to altered perception of myocardial ischemic pain.⁴³⁻⁴⁶

Finally, reduced indexes of HRV have been shown to be a predictor of mortality after myocardial infarction.¹⁶⁻¹⁹ Whether reduced HRV immediately after CABG surgery is prognostic of adverse postoperative cardiac outcomes was not examined in the current study and, thus, remains to be elucidated. Considering our postoperative HRV findings and the unknown time course of HRV recovery after CABG surgery, potential difficulty could occur in interpreting low HRV in patients with prior CABG surgery and new ischemic events. That is, in the setting of a new ischemic event in a patient with prior CABG surgery low HRV could be the result of prognostically important cardiac autonomic imbalances or could represent residual effects of CABG surgery itself.

We observed reductions in HRV after uncomplicated CABG surgery compared with that before surgery and after induction of anesthesia and compared with HRV observed in patients after major vascular surgery. Although we cannot exclude the effects of uncontrolled variables reducing postoperative HRV, the observed changes in HRV appeared not to be the result of general anesthesia, perioperative stress responses, and other factors associated with the early postoperative period. These data are consistent with the supposition that cardiac ANS function is impaired after cardiac surgery.

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HEART RATE VARIABILITY AFTER CORONARY ARTERY SURGERY

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