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Normothermic Continuous Blood Cardioplegia Improves Electrophysiologic Recovery after Open Heart Surgery

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Background: **Myocardial protection during open heart surgery is based on administration of oxygenated blood cardioplegia, the preferred temperature of which is still under debate. The current randomized study was designed to prospectively evaluate the quality of myocardial protection and the functional recovery of the heart with either normothermic (group N) or hypothermic (group H) oxygenated blood cardioplegia.**

Methods: **Under continuous electrocardiographic Holter monitoring, 42 patients were randomly scheduled to receive either normothermic (33.5°C) or hypothermic (10°C) cardioplegia solutions during coronary bypass grafting surgery. Blood samples for creatinine phosphokinase, creatinine phosphokinase-MB, lactate, epinephrine, and norepinephrine were withdrawn during cardiopulmonary bypass *via* a coronary sinus cannula.**

Results: **Active cooling in group H on initiation of cardiopulmonary bypass was characterized by transition through ventricular fibrillation in 75% of patients, whereas in group N atrial fibrillation occurred in 65% of patients. On myocardial reperfusion, sinus rhythm spontaneously resumed in 95% of group N patients compared to 25% in group H ($P = 0.0003$). In the latter, 75% of patients developed ventricular fibrillation often followed by complete atrioventricular block, which necessitated temporary pacing for a mean duration of 168 ± 32 min. Both groups showed a similar incidence of intraventricular block and ST segment changes. However, the incidence of ventricular premature beats in the first 16 h after cardiopulmonary bypass was significantly greater in group H ($P < 0.05$), 20 ± 26/h, compared to 3 ± 5/h in group N. Blood concentrations of lactate, creatinine phosphokinase, epinephrine, and norepinephrine increased gradually during the operation, but the differences between the groups were not significant.**

Conclusions: **The current prospective human study suggests that the increased susceptibility for ventricular fibrillation and dysrhythmia, and the delayed recovery of the conduction system after hypothermic myocardial protection, are related to temperature-induced changes in vital cellular functions of the conduction tissue in the postischemic period. Both cardioplegic methods provide adequate myocardial protection, but normothermic oxygenated blood cardioplegia may accelerate recovery of the heart after cardiopulmonary bypass.**
(Key words: Creatinine kinase: blood. Heart: cardiac arrest; cardioplegic solutions. Lactate: blood. Monitoring: Holter electrocardiography. Surgery, cardiac: coronary artery bypass grafting. Sympathetic nervous system, catecholamines: blood.)

OXYGENATED blood cardioplegia has been shown to improve myocardial preservation during functional recovery after open heart surgery.[1–2] Induction of cardiac arrest by antegrade administration of cardioplegia combined with intermittent retrograde delivery of cardioplegic solution through the coronary sinus seems to improve protection of the myocardium supplied by the left anterior descending coronary artery and of the interventricular septum.[3–4]

Controversy exists about the preferred temperature of the cardioplegia administered to obtain an optimal oxygen supply-consumption balance.[5–6] Cessation of

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electromechanical work at normothermia accounts for 90% of the decrease in myocardial oxygen consumption, whereas only a minimal further decrease is achieved in the arrested decompressed heart by decreasing myocardial temperature to 11°C.[7–8] Profound hypothermia to the heart is associated with detrimental consequences to tissue oxygen uptake, enzymatic function, and glucose utilization, which may contribute to tissue hypoxemia even in the presence of continuous delivery of oxygenated hemoglobin.[8–9] Nevertheless, cold blood cardioplegia combined with systemic hypothermia is a widely accepted method because it results in safe and reliable myocardial preservation, together with protection of other organs such as the brain and the kidneys.

Retrograde warm blood cardioplegia is an appealing approach because it circumvents the detrimental effects of hypothermia to the heart, while the continuous supply of oxygenated blood reduces the magnitude of ischemia and therefore the subsequent reperfusion injury.[2,5,8,10] Several clinical and experimental studies have suggested that warm blood cardioplegia is preferred for myocardial protection as judged by the recovery of systolic and diastolic myocardial performance and reduced release of creatinine kinase.[11–12]

The current study was designed to investigate whether the electrophysiologic abnormalities observed after hypothermic cardioplegic arrest are related to the effect of ischemia, or to the incomplete recovery from hypothermia. Animal studies have shown that both normothermic and hypothermic cardioplegic techniques resulted in similar depletion of adenosine triphosphate stores,[13] and that the changes in electrophysiologic characteristics were mainly related to rheologic composition, that is, more in crystalloid than in blood cardioplegia, rather than to temperature effects.[14]

In the current clinical investigation we prospectively compared the quality of myocardial protection induced by hypothermic or normothermic blood cardioplegia solutions administered during coronary artery bypass grafting. The effects of temperature on the electrocardiographic (ECG) behavior during cardioplegic arrest and reperfusion were assessed by continuous electrocardiographic recording (Holter). Biochemical indicators of ischemic myocardial metabolism were obtained from the effluent coronary sinus venous blood. Thus, the data obtained might differentiate between specific changes in the conduction system in response to hypothermic and normothermic cardioplegia regimens, and those related to the development of ischemia.

Materials and Methods

Patient Selection

After obtaining approval by the Institutional Committee of Human Investigation and written informed consent from patients, 42 patients scheduled for elective coronary artery bypass grafting surgery were studied in a prospective and randomized manner. The patients were assigned to one of two regimens of myocardial preservation: normothermic oxygenated blood cardioplegia (group N) or hypothermic oxygenated blood cardioplegia (group H). Exclusion criteria included patients with poor left ventricular function (ejection fraction less than 40%), concomitant valvular disease, antidysrhythmic medications, and those with left bundle branch block. Patients requiring reexploration for bleeding also were excluded, because their myocardial function, their hemodynamic condition, and the amount of anesthesia administered to them differed substantially from the others.

Six of the 42 patients enrolled in the study were excluded from analysis: three were returned to the operating room for reexploration for persistent bleeding, and three others had an uninterpretable Holter recording owing to inadvertent lead disconnection or battery malfunction.

Anesthetic Management

Before arrival at the operating room, the patients received 0.15 mg/kg diazepam by mouth, 0.15 mg/kg morphine sulfate intramuscularly, and 3 μg/kg scopolamine intramuscularly, as well as their usual cardiac medications. Continuous monitoring was obtained from two ECG leads (modified V5 and L2), radial artery blood pressure, internal jugular central venous pressure, oxygen saturation, end-tidal carbon dioxide, and esophageal, rectal, and myocardial temperatures.

In all patients, anesthesia was induced with 15 μg/kg fentanyl, 50 μg/kg midazolam, and 150 μg/kg pancuronium bromide. A continuous infusion of fentanyl ($2\ \mu g \cdot kg \cdot h^{-1}$) and midazolam ($20\ \mu g \cdot kg \cdot h^{-1}$) was then started. Additional boluses of fentanyl (3 μg/kg) and midazolam (20 μg/kg) were given to maintain blood pressure in a ± 15% range of the patient's baseline. When hypotension occurred, phenylephrine was given in 50-μg boluses as needed. No volatile anesthetic was administered.

Electrocardiography

Patients were monitored continuously using a three-channel Holter ECG recorder (Series 8500 Holter Recorder, Marquette Electronics, Milwaukee, WI) using pregelled ECG electrodes (Type 4050, Nikomed, Denmark). The recording was started 30 min before induction of anesthesia and continued for 24 h. Two modified bipolar leads V5 and L3 were monitored. Each anesthetic procedure and operative stage was marked on the Holter tape by electrical signal.

Holter tapes were analyzed off-line for ST-segment deviation, QRS abnormalities, presence of atrioventricular (AV) blocks and dysrhythmias using a dedicated computer (Marquette Series 8000 analysis system, Marquette Electronics). Changes appearing at the trend recording also were observed by full disclosure printing and separately printed at 25 mm/s for detailed analysis, and these ECG strips were independently reviewed by two investigators unaware of the patient's identity, the procedure performed, or the type of cardioplegia employed. Paced patients were evaluated for recovery of sinus rhythm by the nursing staff every 30 min, or on observing the recovery of the heart rhythm on the ECG monitor. The decision to turn off the pacemaker was made with the on-duty physician, who also was unaware of the study groups. Significant ST changes were defined as either $\geq$0.1 mV (1 mm) horizontal or downsloping ST segment depression persisting for at least 1 min in one lead, measured at 60 ms after the J point, or $\geq$0.2 mV ST segment elevation. Intraventricular block was defined as a prolongation of QRS duration $\geq$ 120 ms. Three or more consecutive ventricular premature beats were tabulated as ventricular tachycardia.

A 12-lead ECG was obtained the night before surgery, immediately after, and during the first postoperative day. Myocardial infarction was defined as ECG evidence of myocardial infarction with new Q wave or persistent ST-T changes and an increase in creatinine phosphokinase (CPK)-MB isoenzyme value above 60 IU/l in at least two samples.

Biochemical Indicators

Blood samples were taken from the central venous pressure catheter and from the coronary sinus. A coronary sinus cannula with a manually inflating balloon (Research Medical, Midvale, UT) was inserted by the surgeon from the right atrium according to the closed technique.

Blood samples for lactate, norepinephrine, epinephrine, CPK, and CPK-MB were collected at the following time intervals: before induction (baseline), before initiation of cardiopulmonary bypass (CPB), 15 min after aortic cross-clamping, immediately before declamping, immediately after declamping, and 5 min after termination of the CPB.

Creatinine phosphokinase and CPK-MB values also were measured 12 h after completion of surgery.

Lactate and CPK values were measured by Kodak Ektachem Clinical Chemistry Slide (Eastman Kodak, Rochester, NY). Normal values and coefficient of variation for CPK are 20–116 units/l $\pm$ 9.2%; for lactate 0.5–2.2 mM $\pm$ 0.08. Plasma catecholamines were measured by high performance liquid chromatography (Hewlett-Packard, Texas), having a sensitivity of 100 pg/ml.

Operative Technique

All the operations were performed by the same two surgeons; each used both regimens of myocardial preservation. Cardiopulmonary bypass was performed with a membrane oxygenator (Cobe Membrane Lung, 050-124-500, Arvada, CO) and the aorta was cross-clamped immediately after achieving satisfactory CPB. The patients were prospectively randomized into two groups: group N (normothermia; n = 20) included patients who received normothermic oxygenated blood cardioplegia at CPB blood temperature (no active cooling was performed). Group H (hypothermia) (n = 16) included patients in whom cardioplegia administration started when body temperature decreased by the CPB to 32°C. They received hypothermic oxygenated blood cardioplegia at 10°C, the heart was kept cool with topical application of slushed ice, and body temperature was maintained at 24°C. In group N, body temperature was not allowed to drift below 32°C. The composition of the cardioplegic solution, the pressure and sequence of administration were similar in both groups and they differed only in temperature. In both groups, the cardioplegia was turned off during the construction of the distal anastomosis.

Two cardioplegia solutions were used: high potassium content (high K; 100 mEq/l) and low potassium content (low K; 20 mEq/l), which were delivered antegradely and retrogradely in all patients. High K blood cardioplegia (St. Thomas Hospital modified cardioplegic solution containing no glucose) was delivered into the aortic root at a rate of 300 ml/min for 2 min. Low K blood cardioplegia was then delivered intermittently, in both groups, through the coronary sinus cannula and the vein grafts at a rate of 150–200 ml/min at a pressure of 25–50 mmHg. The low K solution was perfused unless myocar-

Table 1. General Data

	Normothermia (n = 20)	Hypothermia (n = 16)	*P*
Age (yr) (range)	62 ± 11 (38–78)	65 ± 6 (55–75)	NS
Sex (%)			
Male	80	94	NS
Female	20	6	NS
Preoperative medications (%)			
β blockers	70	69	NS
Calcium channel blockers	75	75	NS
Nitrates	95	94	NS
Catheterization data (%)			
Two-vessel disease	10	6	NS
≥ Three-vessel disease	90	94	NS
Anesthetic medications			
Fentanyl ($\mu g \cdot kg^{-1}$)	35.5 ± 3.4	32.3 ± 2.1	NS
Midazolam ($mg \cdot kg^{-1}$)	0.15 ± 0.03	0.13 ± 0.04	NS
Grafts (n)	4.4 ± 1.0	4.2 ± 0.9	NS
Cross-clamp time (min)	85.9 ± 24.9	80.2 ± 32.2	NS
Total bypass time (min)	132.2 ± 35.4	143.5 ± 26.3	NS
High K solution (ml)	647.2 ± 163.1	415.0 ± 205.5	<0.005
Low K solution (ml)	861.1 ± 582.2	547.5 ± 188.0	NS
Total K administration (mEq)	79.7 ± 13.1	47.6 ± 15.9	0.0002
Postbypass serum K concentration (mM)	5.55 ± 0.84	4.54 ± 0.95	<0.01

Values are mean ± SD.

NS = not significant.

dial electrical activity necessitated an additional bolus of high K cardioplegic retrogradely. In both groups, the heart was perfused with warm blood reperfusion, without cardioplegic solution, before aortic declamping, such that on termination of CPB, body temperature in both groups was equal.

Statistical Methods

All data are presented as mean ± standard deviation. Student's *t* test was used to test the differences between the means of each data point in the two groups. Parametric data were analyzed by two-way analysis of variance. Wilcoxon-rank-sign test was used to compare nonparametric data. Differences were significant when *P* was less than 0.05.

Results

General Data

Both groups had similar distribution of clinical and demographic data as shown in table 1. The severity of cardiac disease, as expressed by their catheterization data and preoperative medications, also was similar.

During surgery, the two groups were similar in regard to the number of coronary grafts, aortic cross-clamp time, and total bypass time. In spite of the difference in body temperature, the amount of anesthesia administered also was similar (table 1).

The quantity of potassium administered in the cardioplegia solution in patients of group N (79.7 ± 13.1 mEq) was significantly greater than in group H (47.6 ± 15.9 mEq; $P < 0.0002$), which resulted in plasma potassium concentration in the immediate postbypass period of 5.55 ± 0.84 mM in group N and 4.54 ± 0.95 in group H ($P < 0.01$). The different potassium requirements have a close association to the myocardial temperature during CPB, *i.e.*, 33.3 ± 0.7°C in group

Table 2. Temperature Data during Cardiopulmonary Bypass

	Core	Cardioplegia	Myocardium
Normothermic group			
Bypass	34.2 ± 1.8	33.5 ± 1.1	33.3 ± 0.7
Declamping	37.0 ± 0.7	37.0 ± 0.6*	37.2 ± 0.6
Hypothermic group			
Bypass	24.0 ± 2.1	10.0 ± 0.4	9.0 ± 1.4
Declamping	36.8 ± 1.6	37.0 ± 0.8	37.1 ± 0.6

* Antegrade blood perfusion without cardioplegic solution.

N compared to 9 ± 0.4°C in group H (table 2). Although regional differences in myocardial temperature are possible, the similarity between body and heart temperature in group N and the use of topical cooling with ice in group H ensure small differences in regional myocardial temperature.

Electrocardiographic Findings

Preoperative ST-segment changes characteristic of ischemia were demonstrated on the Holter monitoring in three group N patients (15%) and in two group H patients (12.5%; P = NS). These findings regressed after induction of anesthesia. Nine patients (45%) in group N, who were free of ischemic ST changes before initiation of CPB, demonstrated changes compatible with ischemia after bypass, as compared to eight patients (50%) in group H (P = NS; fig. 1). Most of these ST-T abnormalities resolved in the subsequent hours.

The major difference between the two cardioplegic methods was in their effect on the conduction system. On initiation of CPB, three different patterns of transition from normal sinus rhythm to cardiac standstill were observed in response to cardioplegia administration (fig. 2). The sequence of events was as follows: (1) sinus rhythm to atrial fibrillation to idioventricular rhythm to sinus arrest, (2) sinus rhythm to idioventricular rhythm to sinus arrest, (3) sinus rhythm to atrial fibrillation to ventricular fibrillation to sinus arrest.

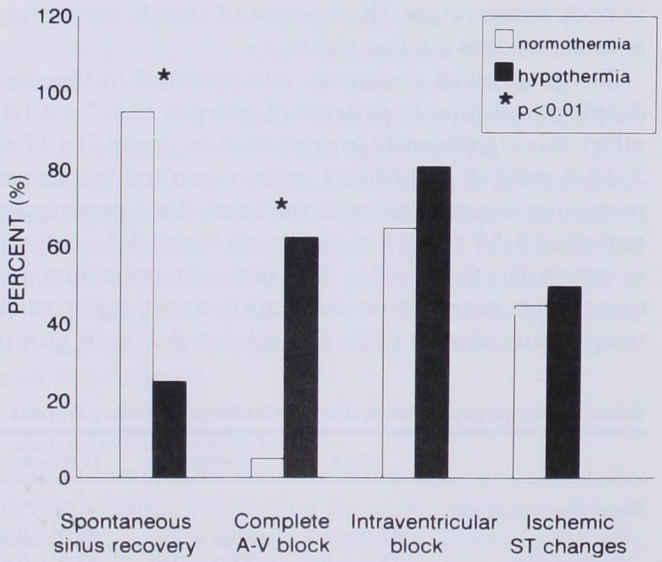

Fig. 1. Electrocardiographic characteristics in the postcardiopulmonary bypass period as obtained by continuous electrocardiography (Holter) recordings. A comparison between myocardial protection by normothermic blood cardioplegia (n = 20) and hypothermic blood cardioplegia (n = 16). *Significant difference between the two groups (P < 0.01).

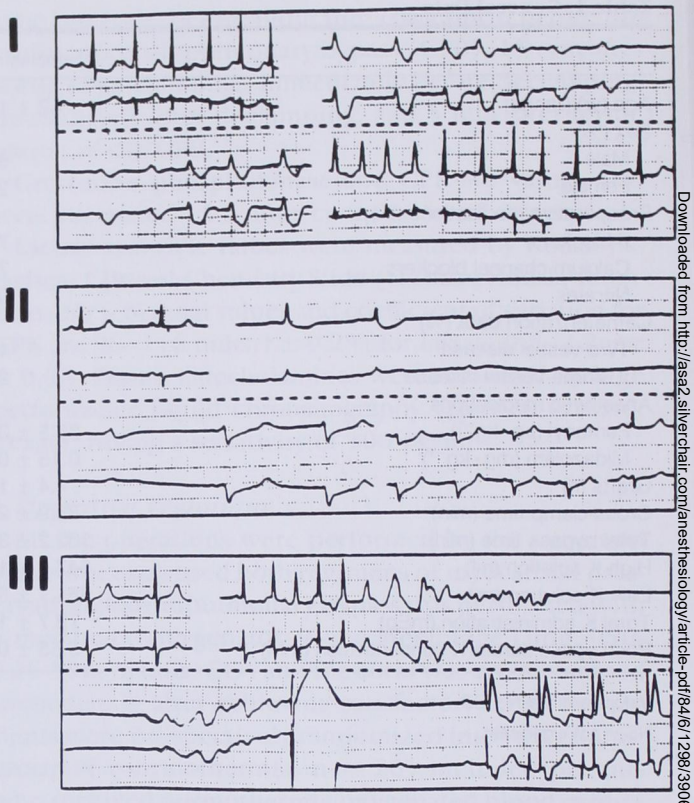

Fig. 2. Patterns of electrocardiographic changes during induction of cardioplegic arrest of the heart (upper part of each strip chart) and during recovery after release of the aortic cross-clamping (lower part of each strip chart). Pattern 1: Sinus rhythm to atrial fibrillation to idioventricular rhythm to sinus arrest. Recovery in reverse order. Pattern 2: Sinus rhythm to idioventricular rhythm to sinus arrest. Recovery in reverse order. Pattern 3: Sinus rhythm to atrial fibrillation to ventricular fibrillation to sinus arrest. On recovery: sinus arrest to ventricular fibrillation to electric countershock to complete atrioventricular block to atrioventricular pacing.

In group N, high K^+ normothermic cardioplegia solution was administered while body temperature was not actively decreased. Pattern 1 of cardioplegic arrest occurred in 13 patients (65%) and pattern 2 in 6 patients (30%). However, in group H in which active systemic cooling was started and hypothermic cardioplegia was given, pattern 3 with early ventricular fibrillation occurred in 12 patients (75%), while patterns 1 and 2 occurred in 4 patients (25%). At the time of aortic declamping, myocardial temperature had reached 37°C in both groups, by normothermic blood perfusion of the heart (table 2). In only 25% of the patients of group H did the heart spontaneously regain

sinus rhythm (fig. 1), compared to 95% in group N ($P < 0.0003$). In 10 group H patients (62.5%) a complete AV block occurred and electrical pacing was needed for a mean duration of 168 ± 2 min. In group N, a temporary complete AV block occurred in only one patient (5%) and lasted 380 min. There was no significant difference between the two groups in the incidence of intraventricular block (fig. 1). Analysis of the demographic characteristics of the patients who developed complete AV block in group H, compared to those in whom sinus rhythm recovered immediately, revealed no difference in age (65 ± 5 and 66 ± 6 yr), preoperative heart rate, administration of verapamil or nifedipine calcium channel blockers, beta blockers, cross-clamp time, and number of grafts performed. Mean blood K^+ in the AV block patients was 4.8 ± 0.8 mEq/l in group N, compared to 4.2 ± 0.8 (P = NS) in group H.

Another phenomenon that was limited to 11 patients (55%) of group N only, was persistent atrial electrical activity detected during the cross-clamping time despite large doses of cardioplegic solution, and that lasted for approximately one third of the aortic cross-clamping time. In group H, patients had more ($P < 0.01$) ventricular fibrillation after aortic declamping, thus requiring electric countershock and pacing to a greater extent.

The incidence of ventricular premature beats in the prebypass period was similar in both study groups: mean of 1.5 ± 0.3 ventricular premature beats/h in group H and 1 ± 0.4 in group N. Group H had a high incidence of ventricular premature beats in the first 2 h post-CPB (fig. 3). The mean number of ventricular premature beats monitored was 20 ± 26.3/h (range = 0–128) in this time period compared to 3 ± 5.1/h (range = 0–26) in group N ($P = 0.03$). In the next 16 h postoperative follow-up, a mean number of ventricular premature beats of 6.2 ± 7.9/h in group H was found as opposed to 2.8 ± 4.2/h in group N ($P < 0.05$). One patient in group N and five patients in group H developed verified episodes of ventricular tachycardia in the postbypass period, none of which progressed to ventricular fibrillation. *$P < 0.05$ by analysis of variance.

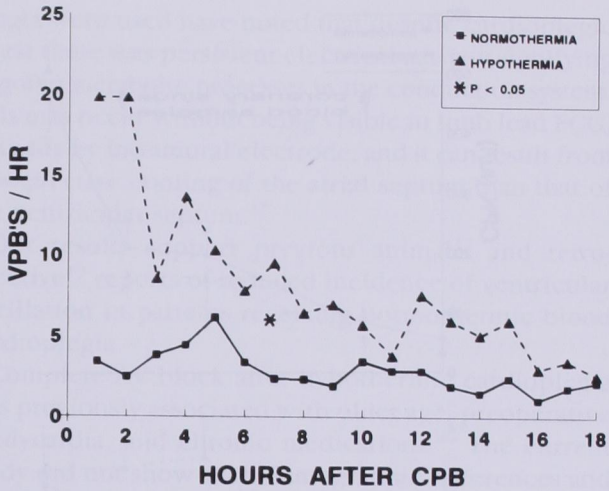

Fig. 3. Incidence of ventricular premature beats in the postbypass period, in group N as compared to group H. *Denotes significant difference between the mean ventricular premature beats number in the two groups ($P < 0.05$ by analysis of variance).

Biochemical and Enzymatic Parameters

A gradual increase in CPK values was observed in both groups during the CPB period (fig. 4). Immediately after termination of CPB, an increase in the CPK concentration was noted to a mean peak of 510 ± 401.1 IU/l and 460 ± 317 (an increase of 307% and 218%) in groups H and N, respectively (P = NS). The MB fractions at that time point were in H − 5.4% and in N − 5.3%. Twelve hours after surgery mean CPK was 451 ± 201 IU/l (MB = 7.7%) in group H and 662 ± 253 (MB = 3.1%) in group N (P = NS). Two of the patients in group H (12.5%) and three in group N (15%) had electrocardiographic and biochemical evidence for acute myocardial infarction with mean CPK-MB values in H − 110 ± 26 IU/l and in N − 82 ± 17, 12 h postoperatively (P = NS).

Although a gradual increase in the lactate concentrations was noted during the operation, there was no difference between the two groups (P = NS; fig. 4). The mean increase in lactate concentration from baseline at time 0 to maximal value at the end of CPB was 252% in group N and 212% in group H (P = NS compared to baseline).

The increase in the blood epinephrine concentrations in group N had a biphasic pattern. Immediately after initiation of CPB, a nonsignificant change from baseline value of 106 ± 72 to 423 ± 338 pg/ml was observed. After aortic declamping, in both groups an increase in epinephrine concentration was noted, the difference of which again was nonsignificant as compared to baseline or between the two groups.

Norepinephrine concentrations increased gradually in both groups during the CPB period to peak values

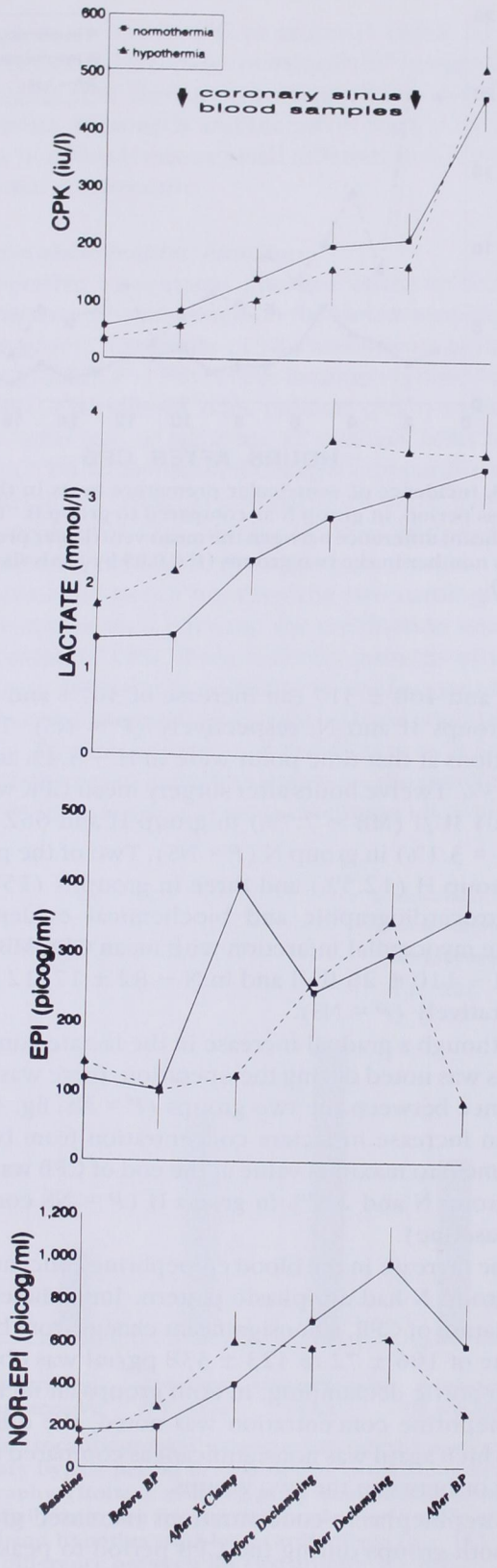

Fig. 4. Biochemical indicators. Mean blood concentrations of creatinint phosphokinase, lactate, epinephrine, and norepinephrine aspirated throughout the study from central venous blood or from the coronary sinus during the cardiopulmonary bypass period. Each data point represents the mean value in the normothermic blood patients or the hypothermic blood patients. The changes in blood concentrations were neither significant between the two groups, nor when compared to baseline.

of 964 ± 576 pg/ml in group N and 615 ± 546 in group H (P = NS).

Discussion

The current study prospectively evaluates the effect of temperature of the cardioplegic perfusate on the quality of myocardial protection during CPB and on the ECG characteristics of the heart during the cardioplegic arrest.

Owing to residual metabolic consumption during prolonged aortic cross-clamp time, neither electrical arrest nor hypothermia is sufficient to avoid the development of ischemia. Continual delivery of oxygenated blood is thus recommended. Recently, improvement in myocardial protection has been suggested when retrograde normothermic blood cardioplegias were used, thus avoiding the adverse effects of hypothermia on subcellular constituents and metabolic transport mechanisms.[8,15–16]

Our results indicate that a normothermic cardioplegic solution, as compared to a hypothermic solution, may provide greater cardiac protection, particularly to the conduction system. Recovery of sinus rhythm was significantly enhanced and the development of ventricular dysrhythmias was significantly diminished in the normothermic group. These differences suggest that normothermic arrest of the heart may better preserve oxygen homeostasis of the myocardium, avoiding the development of electrophysiologic abnormalities in the myocardial conduction system.

Using Holter recording analysis we were able to characterize three patterns of transition from the original rhythm to cardioplegia-induced arrest. These patterns were dependent on the temperature of CPB and the cardioplegic solution. In the hypothermic group, at the beginning of CPB, the frequent occurrence of ventricular fibrillation was presumably a question of timing, *i.e.*, the result of a short period of systemic cooling

of the heart with cold blood from the CPB, which preceded the administration of high potassium cardioplegia. In contrast, in the normothermic group the arrest of the heart was solely a consequence of the high potassium levels.

The phenomenon of persistent atrial activity in the arrested heart during aortic cross-clamping in the normothermic group is not completely understood. It might be related to inadequate cardioplegia delivery to the right atrium, or to excessive flushing of the right atrium by noncoronary blood flow. This phenomenon does not necessarily represent inadequate myocardial protection, because right-sided ventricular failure was not observed. On the contrary, the current data confirm better recovery of the conduction system when normothermic oxygenated blood cardioplegia was used throughout the ischemic period.

The clear group differences in spontaneous recovery of sinus rhythm after aortic declamping may particularly represent better protection of the AV node by the normothermic technique. Only one patient (5%) in this group required pacing after CPB compared to 62.5% of the patients with complete AV block in the hypothermic group, reflecting sinus node and AV junction protection. These results were noted despite a 67% greater total K^+ dose administered in group N and higher serum K^+ concentration immediately after CPB. The difference between groups in the amount of K^+ administered is explained by the natural concern of the surgical team for the persistence of electrical activity in the warm heart during aortic cross-clamp time. This limitation of the study would not, however, account for the lower incidence of complete heart block in the normothermic cardioplegia group.

Before aortic clamp release, in both groups of patients, the heart was reperfused with warm blood. Thus, myocardial temperature at the time of declamping was similar, 37°C. Nevertheless, the electrophysiologic recovery of the heart was different in the hypothermic compared to the normothermic cardioplegia group. Are the observed differences related just to a slow cellular recovery from the effect of hypothermia, or to hypothermia-induced localized ischemia in the conduction system? This issue is still controversial.

Conduction disturbances after coronary artery bypass grafting have been attributed to localized ischemia in the region of the first perforating branch of the left anterior descending artery, which supplies the conduction system (proximal bundle branches).[16] Previous studies[17–18] in which cold crystalloid or blood cardioplegia were used have noted that despite cardioplegic arrest there was persistent electrical activity signifying ongoing metabolic processes in the conduction system. This may occur without being visible in limb lead ECG, but only by intramural electrode, and it can result from less effective cooling of the atrial septum than that of the ventricular septum.[17]

Our results support previous animal[12] and retrospective[19] reports of reduced incidence of ventricular fibrillation in patients receiving normothermic blood cardioplegia.

Complete AV block after hypothermic cardioplegia was previously associated with older age, preoperative bradycardia, and chronic medications.[20] The current study did not show such demographic differences and the gradual dissolution of complete AV block might be explained by slow recovery of the conduction system from the insult of the ischemic period and hypothermia.

The overall degree of myocardial protection provided by both types of cardioplegic solutions appeared to be adequate. There was no difference in the coronary sinus CPK blood levels between the two groups. We suggest that the temporary cellular dysfunction after hypothermic cardioplegia did not produce cellular damage, and thus was not reflected in significant CPK release or excessive production of lactate or catecholamines.

The current study suggests that although both normothermic and hypothermic blood cardioplegia provide adequate myocardial protection, they have different effects on the rate of functional recovery of the cardiac conduction system. Administration of normothermic blood cardioplegia offers better preservation of sinus rhythm, reduces the incidence of AV block, and decreases the rate of occurrence of ventricular arrhythmias. The advantage of this type of cardioplegia is limited to the immediate post-CPB period and to the first postoperative hours. Further clinical studies are recommended to define the best temperature for myocardial preservation that will provide rapid recovery of heart function together with a low rate of complications to other body organs.

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