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Efficacy of Physiologic Temperature on the Spasm of Harvested Radial Artery

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ABSTRACT

Background. The potential disadvantage of using the radial artery for coronary artery bypass grafting is its increased tendency to vasospasm. Therefore, different antispastic agents are being used in the perioperative and postoperative period. During the preparation of the radial artery, normal local and systemic temperatures are lost.

Methods. We investigated the effects of topical normal saline solution at 20°C (group SI), normal saline solution at 36°C (group SII), diltiazem at 20°C (group DI), and diltiazem at 36°C (group DII) on radial artery free flow. Each group contained 10 patients undergoing coronary bypass surgery. Free flow and local temperature were measured at 3 stages: after the exploration and preparation of the distal 3 cm of the radial artery, after total preparation of the radial artery, and a median of 12 minutes after the pedicle had been sprayed with one of the agents.

Results. Parallel to the significant decrease of the second local temperatures ($P < .001$), the second flow of the 4 groups decreased significantly ($P < .001$).

Conclusion. Hypothermia plays an important role in radial artery vasospasm, and normothermia may be the best perioperative vasodilating agent since the normal radial artery flows were reached with normothermia.

INTRODUCTION

Since its reintroduction by Acar and colleagues [Acar 1992], the use of the radial artery (RA) as an alternative coronary artery bypass conduit has gained increased popularity. The increased patient population undergoing a coronary reoperation [Shapira 1999] and the increasing interest in the arterial revascularization concept [Weinschelbaum 1997; Muneretto 2003] led surgeons to attempt to find the best alternative arterial conduit to the internal thoracic artery (ITA), which is the gold standard as the first choice conduit in most institutes [Okies 1984; Loop 1986; Sones 1986].

Aside from reports of high patency rates [Brodman 1996; Manasse 1996; Weinschelbaum 1997; Tatoulis 2002], its sufficient length and its easy simultaneous harvestability with the ITA make the RA an attractive choice among the other alternative arterial grafts to the ITA.

Since the RA was first used as a coronary artery bypass graft by Carpentier [Carpentier 1973], surgeons started facing vasospasm in the perioperative period. In recent years most of the research on the RA has attempted to find the best method to prevent vasospasm. Various in vitro studies suggest the use of different vasodilating agents for the prevention of vasospasm [He 1996; He 2000; Chanda 2000; Dipp 2001]. Studies to find the best agent and method still continue.

During the preparation of the RA, normal local and systemic temperatures are lost. This study was designed on the hypothesis that hypothermia occurrs because of the decrease of the local temperature at the RA harvesting surgical field while the RA is being harvested, which causes a decrease in the RA flow by vasoconstriction. We believed that the preservation of the physiological state of normothermia during RA harvesting would decrease the degree of vasospasm. To prove our hypothesis, we investigated the local temperature changes and their effects on RA flow and compared them with the effects of topical diltiazem treatment at room temperature and at 36°C.

METHODS

In 40 patients whose RA were used as a conduit for myocardial revascularization, the effects of topically administered normal saline solution at the operating room temperature (20°C, group SI), normal saline solution at 36°C (group SII), diltiazem at the operating room temperature (20°C, group DI) and diltiazem at 36°C (group DII) were investigated.

Patients who met one of the following criteria were excluded: age over 70 years, chronic renal failure, diabetes

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Table 1. Clinical Characteristics and Hemodynamic Data of the Patients*

Characteristic	Group SI, n = 10	Group SII, n = 10	Group DI, n = 10	Group DII, n = 10
Male/female	9/1	8/2	8/2	9/1
Age, y	57.9 ± 7.3	53.4 ± 8.5	55.8 ± 7.7	57.4 ± 7.0
Range	45-67	42-69	42-67	47-69
Body surface area, m^2	1.94 ± 0.13	1.88 ± 0.10	1.86 ± 0.16	1.86 ± 0.08
Range	1.74-2.09	1.74-2.02	1.68-2.22	1.75-2.00
Heart rate 1, beats/min	85.3 ± 12.0	80.8 ± 8.0	79.1 ± 8.7	82.5 ± 12.7
Heart rate 2, beats/min	88.5 ± 8.6	86.2 ± 6.9	84.0 ± 9.2	90.2 ± 11.2
Heart rate 3, beats/min	83.1 ± 6.5	79.7 ± 8.0	79.1 ± 7.0	81.3 ± 7.2
Mean arterial pressure 1, mmHg	81.8 ± 10.4	81.9 ± 11.2	82 ± 12.8	81.6 ± 10.5
Mean arterial pressure 2, mmHg	85 ± 9.1	84.1 ± 8.6	83.5 ± 8.0	82.8 ± 7.9
Mean arterial pressure 3, mmHg	79.7 ± 9.5	79.2 ± 9.2	78.4 ± 9.0	78.1 ± 2.9
Central venous pressure 1, mmHg	6.2 ± 2.4	5.9 ± 2.3	6.2 ± 1.8	5.6 ± 2.1
Central venous pressure 2, mmHg	7.4 ± 1.6	6.8 ± 1.6	6.9 ± 2.1	6.3 ± 2.0
Central venous pressure 3, mmHg	5.7 ± 3.0	5.1 ± 3.1	4.7 ± 3.1	4.6 ± 2.5
t1, min	16.4 ± 2.8	16.7 ± 2.9	16 ± 2.7	16.2 ± 2.5
t2, min	12.4 ± 2.1	12.7 ± 2.2	12 ± 2.1	12.6 ± 2.3

*Data are presented as mean ± standard deviation except gender ratios. t1 indicates time period between the first and second measures; t2, time period between the second and third measures.

mellitus, prior trauma or surgery to the relevant upper limb, Raynaud's phenomenon, or low ejection fraction (<30%).

RA grafts were prepared in 40 patients undergoing elective coronary artery bypass surgery. Each patient was randomly placed in 1 of the 4 equally sized groups (Table 1).

Preoperative Management

All patients underwent a modified Allen's test preoperatively. Oxygen saturation was measured by connecting the patient's thumb or index finger to the oxygen saturation monitor. The radial and ulnar arteries were compressed until the saturation value decreased to zero. The ulnar artery was then released and the period it took for the oxygen saturation to reach the prior value was noted. The test was not considered acceptable if it lasted longer than 10 seconds. The test was performed on both of the patient's hands. Peripheral intravenous and arterial ways were set on the dominant arm before anesthesia induction.

Surgical Technique

In all of the patients the RA was harvested from the nondominant left arm. The left arm was placed on a separate table. Arm and thorax were prepared simultaneously. A 3-cm long horizontal incision starting from 1 cm medial and proximal of the radial styloid was made. The RA was explored all around with sharp dissection along the incision. The distal end was clamped and the RA was divided proximally from the clamp. The superficial palmary artery was left on the distal part of the division. The distal part of the clamp was tied with 2/0 silk. Flow 1 was measured. The distal end of the radial artery was clipped. The incision was extended to 1 cm medial and distal of the biceps tendon. Subcutaneous tissues were dissected with low diathermia. The fascia covering the superficial flexors of the forearm and brachioradialis, extensor carpi radialis longus, and extensor carpi radialis brevis muscles was divided between brachioradialis and flexor carpi radialis muscles. The RA was harvested with 2 satellite veins and loose areolar tissue. The RA was mobilized from the muscle bed at the medial portion. The perforating branches that could be easily seen by a light upward traction were clipped and divided between the clips with scissors. Proximally the RA was grafted to the radial recurrent artery. Flow 2 was noted. The graft was sprayed with one of the solutions of normal saline solution at the operating room temperature (20°C, hypothermic), normal saline solution at 36°C (normothermic), diltiazem at the operating room temperature (20°C, hypothermic), and diltiazem at 36°C (normothermic). The graft was wrapped in a gauze soaked with normal saline solution at 36°C in normothermic groups or with the same solution at room temperature in hypothermic groups. The gauze was sprayed with the same solution, and it was soaked every 2 minutes. After a waiting period no less than 10 minutes, flow 3 was noted. Flows were determined by measuring the volume of blood expelled from the end of the bleeding artery in a 30-second period. After each measuring, the bleeding end of the artery was occluded with a bulldog clamp.

The topical solutions consisted of the following: group SI received 10 mL normal saline solution at room temperature (20°C); group SII, 10 mL normal saline solution at 36°C; group DI, 25 mg diltiazem in 10 mL normal saline solution at room temperature (20°C); group DII, 25 mg diltiazem in 10 mL normal saline solution at 36°C.

Patients who needed vasopressing or vasodilating agents during the operation were excluded. At the 3 stages the free flows were measured; time, mean arterial pressure, esophageal temperature, local temperature, heart rate, and central venous pressure values were also recorded. Local temperature was measured from the areolar tissue around the RA using a myocardial temperature probe (De Royal, Powell, TN, USA). A general-purpose temperature probe (De Royal) was used for measuring esophageal temperature.

All statistical procedures were performed using the program GraphPad InStat Version 2.02 (GraphPad Software,

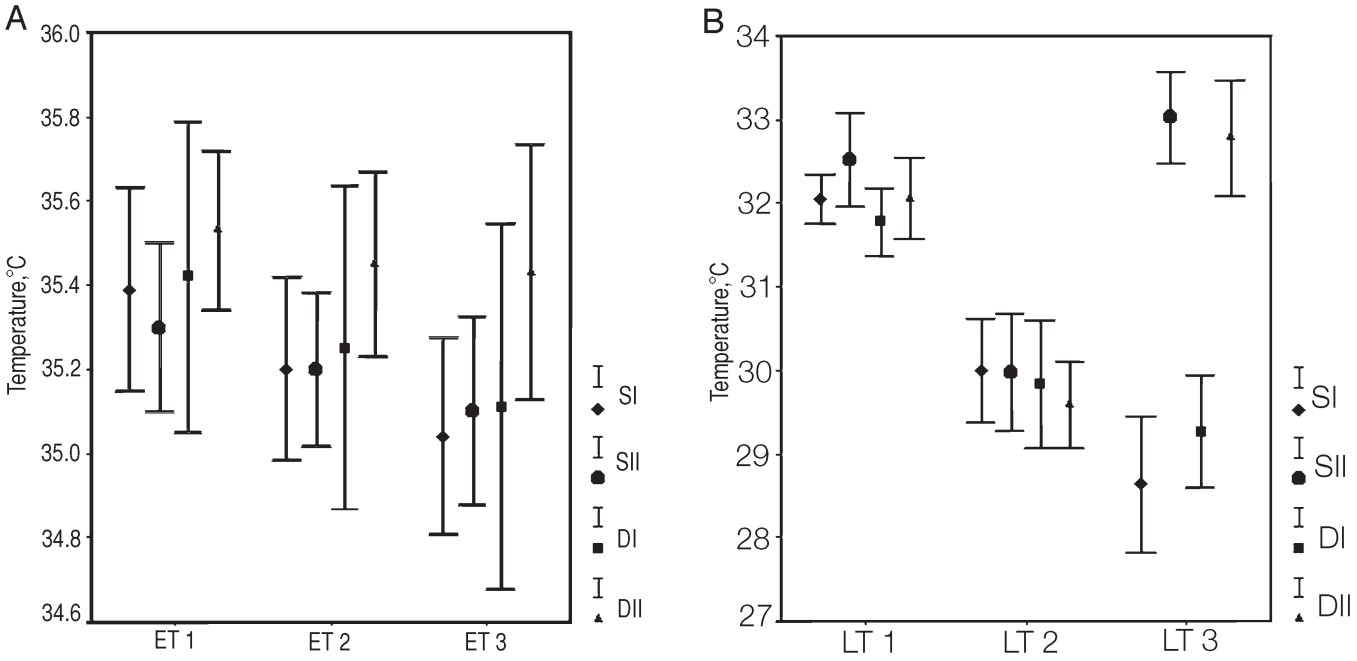

Figure 1. A, Esophageal temperature data are shown with error bars. ET indicates esophageal temperature. B, Local temperature data are shown with error bars. LT indicates local temperature.

San Diego, CA, USA) for DOS. All values are expressed as mean ± standard deviation. For comparing the repeated flow, mean arterial pressure, esophageal temperature, local temperature, heart rate, and central venous pressure measures in each group, repeated measures analysis of variance and Tukey's multicomparison tests were used. The comparison of measurements between the groups was made by one-way analysis of variance and Tukey's multicomparison tests. A *P* value less than .05 was considered significant.

RESULTS

Sex ratios and ages of the patients in each of the 4 groups are shown in Table 1. There is no significant difference between body surface areas and ages of the patients in the 4 groups. Since low cardiac output and hypovolemia could affect the free flow measures, we recorded heart rate, mean arterial pressure, and central venous pressures as the signs of low cardiac output and hypovolemia. When these parameters were compared there was no statistically significant difference within or between the groups. Medians of the temperature and flow measurements are shown in Tables 2 (and Figure 1) and 3 (and Figure 2), respectively. Esophageal temperature measurements at the same stages were not significantly different, just as with the local temperatures.

There was no significant difference between the first flows of the groups that were measured after the exploration and preparation of the distal 3 cm of RA with minimal surgical trauma and without using diathermia. A significant decrease occurred in the second flows that were measured after the preparation of the RA graft (from 68.1 ± 28.1 mL/min to 37.2 ± 11 mL/min in group SI, from 69.8 ± 29.2 mL/min to 41 ± 15.4 mL/min in group SII, from 61.9 ± 28.6 mL/min to 37 ± 14.2 mL/min in group DI, and from 65.5 ± 23.9 mL/min to 35 ± 10.1 mL/min in group DII; $P < .001$ for all). Similarly within all the groups there were significant differences between the first and second local temperatures of the RA graft ($P < .001$). This significant decrease occurred as follows: from 32.05 ± 0.4°C to 30 ± 0.9°C in group SI, from 32.52 ± 0.8°C to 29.98 ± 1°C in group SII, from 31.78 ± 0.6°C to 29.83 ± 1°C in group DI, and from 32.06 ± 0.7°C to 29.59 ± 0.7°C in group DII.

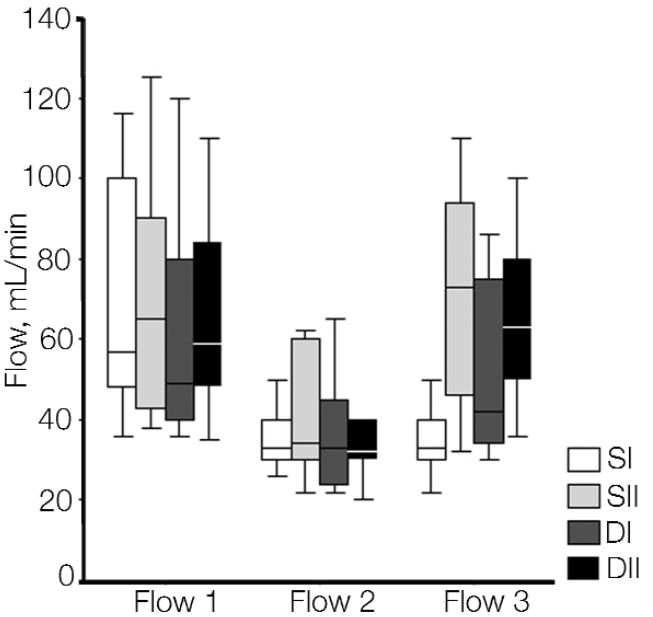

Figure 2. Flow measurements are shown in a box-plot graph.

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Table 2. Esophageal and Local Temperature Measurements*

Measurement	Group SI	Group SII	Group DI	Group DII
Esophageal temperature 1, °C	35.39 ± 0.3	35.3 ± 0.3	35.42 ± 0.5	35.53 ± 0.3
Esophageal temperature 2, °C	35.2 ± 0.3	35.2 ± 0.2	35.25 ± 0.5	35.45 ± 0.3
Esophageal temperature 3, °C	35.04 ± 0.3†	35.1 ± 0.3‡	35.11 ± 0.6	35.43 ± 0.4
Local temperature 1, °C	32.05 ± 0.4	32.52 ± 0.8	31.78 ± 0.6	32.06 ± 0.7
Local temperature 2, °C	30 ± 0.9§	29.98 ± 1§	29.83 ± 1§	29.59 ± 0.7§
Local temperature 3, °C	28.64 ± 0.4§‖#	33.03 ± 0.8‖	29.26 ± 0.9§¶#	32.78 ± 0.9‖

*Data are presented as mean ± standard deviation.

†$P < .001$ versus esophageal temperature 1 of group SI.

‡$P < .01$ versus esophageal temperature 1 of group SII.

§$P < .01$ versus local temperature 1 within each group.

‖$P < .001$ versus local temperature 2 in groups SI, SII, DII.

¶$P < .05$ versus local temperature 2 in group DI.

#$P < .001$ versus local temperature 3 of groups SII and DII.

In group SI, the significant decrease in the local graft temperature continued (28.64 ± 0.4°C, $P < .001$ when compared with the first and second temperatures). The decrease in the third free flow was significant when compared with the first flow ($P < .001$), but not significant when compared with the second.

In group SII, a significant increase in the third free flow from 41 ± 15.4 mL/min to 70.6 ± 28.4 mL/min ($P < .001$) occurred with a significant increase in the local graft temperature (from 29.98 ± 1°C to 33.03 ± 0.8°C, $P < .001$). The third local graft temperature was nearly similar to the second temperature in group DI, but the increase in the third flow was significant (from 37 ± 14.2 mL/min to 50.4 ± 21.4 mL/min, $P < .05$). In group DII, normothermic diltiazem produced a significant increase in the third flow (from 35 ± 10.1 mL/min to 66.1± 22 mL/min, $P < .001$) and the local graft temperature increased significantly from 29.59 ± 0.7°C to 32.78 ± 0.9°C.

DISCUSSION

The potential disadvantage of using the RA for coronary artery bypass grafting is its increased tendency to vasospasm. Spasm and intimal hyperplasia, which were thought to be due to the harvesting technique, were the reasons for the abandonment of Carpentier's initial advocation of using the RA for coronary artery bypass grafting [Acar 1992].

Vasospasm occurs in all kinds of vessels but especially in arterial conduits. Despite many studies, our knowledge of how it occurs is quite limited. Isolated tissue studies that form the majority of these studies are not expected to identify the cause of in vivo spasm. As Rosenfeldt et al say, they can determine what factors have the potential to contract the tissue in vivo [Rosenfeldt 1999]. It is important to determine the major factors that cause abnormal vasoconstrictor activation in the vessel. Physical factors such as surgical trauma and temperature changes, and pharmacological factors such as neural factors, locally released vasoconstrictors, and circulating hormones are included in these vasoconstricting factors.

Although temperature changes are thought to be one of the factors leading to vasospasm, our study is first to study the effect of temperature changes on the RA flow. Since Acar's reintroduction of the RA graft in 1992 [Acar 1992], aside from a meticulous harvesting technique, a systemic or topical vasodilating treatment has been generally used in the perioperative period. Calcium (Ca) antagonists [Acar 1992; Reyes 1995], papaverine [Dietl 1995], phosphodiestherase inhibitor milrinone [Buxton 1997], phenoxybenzamine [Dipp 2001; Velez 2001] and verapamil-nitroglycerine [He 1996], and verapamil-papaverine [Fremes 1995] solutions are among these vasodilators.

Diltiazem, the agent we used in our study, is a Ca antagonist used as a vasodilating agent since Acar's initial surgical experience to release vasospasm. He et al have compared the spasmolytic effects of Ca antagonists for relief of RA spasm. Among 4 Ca antagonists, potency is in the following order: nifedipine > nicardipine > verapamil > diltiazem. For topical use, however, any of the Ca antagonists would provide an effective antispastic effect [He 2000].

In moderate environments, peripheral compartment temperature is usually 2 to 4°C less than the core temperature. This difference increases in extreme thermal and physiological conditions [Sessler 2000]. In our study, the second local radial temperatures significantly decreased in all groups. The significant decrease continued in the third measurements in hypothermic groups (group SI and group DI). Esophageal temperatures, despite a small decrease, were determined to be above 35°C (Figure 1, Table 2). The high gradients between esophageal and local radial temperatures in the second and third measurements of hypothermic groups are indicators of the constraining of metabolic heat in the core. Vasoconstriction that occurs to store the metabolic heat in the core increases the temperature gradient between the core and periphery. The decrease in the second and third flows of the hypothermic groups reflects the effect of vasospasm.

Although in group DI a significant increase from 37 ± 14.2 mL/min to 50.4 ± 21.4 mL/min ($P < .05$) occurred in the third flow, it showed no significant difference with the third flow of the control group, group SI (35.3 ± 10.5 mL/min). In the normothermic diltiazem–administered group, group DII, the increase in the third flow was significant (from 35 ±

Table 3. Flow Measurements*

Measurement	Group SI	Group SII	Group DI	Group DII
Flow 1, mL/min	68.1 ± 28.1	69.8 ± 29.2	61.9 ± 28.6	65.5 ± 23.9
Flow 2, mL/min	37.2 ± 11†	41 ± 15.4†	37 ± 14.2†	35 ± 10.1†
Flow 3, mL/min	35.3 ± 10.5†	70.6 ± 28.4‡‖	50.4 ± 21.4§	66.1 ± 22‡¶

*Data are presented as mean ± standard deviation.
†$P < .001$ versus flow 1 within each group.
‡$P < .001$ versus flow 2 of groups SII and DII.
§$P < .05$ versus flows 1 and 2 of group DI.
‖$P < .01$ versus flow 3 of group SI.
¶$P < .05$ versus flow 3 of group SI.

10.1 mL/min to 66.1 ± 22 mL/min, $P < .001$) (Figure 2, Table 3). We believe that the significant increase in the third local graft temperature (from 29.59 ± 0.7 mL/min to 32.78 ± 0.9 mL/min, $P < .001$) has played a greater role than the vasodilator agent diltiazem in this significant free flow increase. That there was no significant difference between the third flows of the normothermic groups (groups SII and DII) supports our opinion.

CONCLUSION

Topical diltiazem had no superiority to normothermia as a vasodilator. Since all Ca antagonists have the same topical effect on arterial tissue, we suggest that Ca antagonists do not have any superiority to normothermia. We believe hypothermia plays an important role in RA vasospasm and regaining the physiological state normothermia may be the best perioperative vasodilating treatment as the normal RA flows were reached with normothermia.

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