CONTROLLED LIMB REPERFUSION IN PATIENTS HAVING CARDIAC OPERATIONS

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Hypothesis: Severe limb ischemia in patients having cardiac operations may occur after intraaortic balloon pump insertion, prolonged femoral vessel cannulation, percutaneous cardiopulmonary bypass, dissecting aneurysms, or emboli. Normal blood reperfusion can cause a postischemic syndrome that increases morbidity and mortality. This clinical study is based on an experimental infrastructure patterned after controlled cardiac reperfusion. (1) It tests the hypothesis that controlled limb reperfusion (i.e., modifying the composition of the initial reperfusate and the conditions of reperfusion) reduces the local and systemic complications seen after normal blood reperfusion. (2) It reports initial clinical application of this strategy in three cardiac surgery centers. *Methods:* Controlled limb reperfusion was applied to 19 patients with signs of severe prolonged unilateral or bilateral ischemia (including paralysis, anesthesia, and muscle contracture); six patients (32%) were in cardiogenic shock. The mean ischemic duration was 26 ± 6 hours. The reperfusion method includes a 30-minute infusion into the distal vessels of a normothermic reperfusate solution mixed with the patient's arterial blood (obtained proximal to the obstruction) in a 6:1 blood/reperfusate ratio. Data are mean ± standard error of the mean. *Results:* Sixteen patients (84%) survived with salvaged and functional limbs at the time of discharge. No renal, cardiac, pulmonary, cerebral, or hemodynamic complications developed in the survivors. The three deaths occurred in patients undergoing controlled limb reperfusion while in profound postoperative cardiogenic shock; neither postischemic edema nor contracture developed in any of them. *Conclusions:* These findings show that controlled limb reperfusion can be applied readily with standard equipment that is used for cardiac surgery and may salvage limbs while reducing postreperfusion morbidity and mortality. (J THORAC CARDIOVASC SURG 1996;111:873-81)

Severe limb ischemia in patients having cardiac operations may occur after (1) transfemoral insertion of an intraaortic balloon pump (IABP), (2) prolonged femoral vessel cannulation (i.e., thoracic aortic aneurysms, reoperations), (3) thoracoabdominal acute aortic dissections, (4) prolonged percutaneous cardiopulmonary bypass, (5) thrombosis during or after removal of coronary angiography catheters into atherosclerotic vessels, or (6) thromboembolization.

Reperfusion of these severely ischemic limbs with normal blood may result in a postischemic syndrome that is associated with high mortality and morbidity rates.[1] The massive edema can increase fluid requirements and cause shock. Additionally, the washout of myoglobin, potassium, lactate, and microthrombi from the damaged skeletal muscle into the systemic circulation may cause renal failure, arrhythmias, shock, and eventually death,[1-3] inasmuch as mortality rates vary from 7.5% to 41%.[1, 2] A postreperfusion syndrome may result in amputation or severe dysfunction in the salvaged limbs.[1, 2] Our

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Table I. *Composition of the asanguineous solution for the controlled limb reperfusate* (to be mixed with blood at a ratio of 6 to 1†)*

Substance	*Amount (ml)*	*Principle*
Glucose 5%	500	Hyperosmolarity Substrate
Citrate-phosphate-dextrose	150	Reduce Ca^{++}
Tromethamine 0.3 mol/L	200	Buffer
Glutamate/aspartate	150	Substrate
Allopurinol	2.5	Free radical scavenger

*Manufactured by Dr. F. Köhler Chemie GmbH-Hähnlein, Germany.
†Six parts blood and one part asanguineous solution.

recent experimental studies[4-10] and those of others[11-14] show that this injury can be reduced substantially by modifying the initial reperfusion phase during surgical revascularization of severely ischemic skeletal muscle. The concept of controlled limb reperfusion[4-10] is based on principles described for cardiac reperfusion in patients with acute myocardial infarctions[15-18] or after aortic clamping.[19-21] This new application to ischemic skeletal muscle incorporates modifications of the conditions of reperfusion (temperature, pressure, and flow) and the composition of the reperfusate (oxygen tension, hematocrit value, free radical scavengers, osmolarity, calcium, pH, substrates, and glucose) during the first 30 minutes of regional revascularization. It also limits the local and systemic adverse effects of reperfusion after prolonged limb ischemia.

The initial experience with clinical application of this method in 19 patients at three university cardiac surgical centers is summarized, and the results suggest that limb salvage is possible in extremities thought previously to be damaged irreversibly because of ischemic contracture.

Material and methods

Patient population. Controlled limb reperfusion was performed in 19 patients with completely ischemic limbs at the Johann Wolfgang Goethe-University Frankfurt/M. (n = 15), Johannes Gutenberg-University Mainz (n = 3), and German Heart Center Berlin (n = 1). The design of the study was approved by the ethics committee. The onset of acute limb ischemia was defined as the time of pain as reported by the patient or the diagnosis made by the referring physician. Acute lower limb ischemia was confirmed by history, physical examination, and Doppler sonographic examination. Angiograms were not performed routinely in the preoperative period.

Initial signs and symptoms, length of time from sudden occlusion to start of reperfusion, severity of ischemia, associated disease, source and location of thrombi or emboli, type of surgical intervention, and outcome of surgery were registered and are described in the *Resu* section.

Surgical technique for controlled limb reperfusio Fluid, electrolytes, and cardiovascular abnormalities we corrected as much as possible before the operation. A patients received a standard heparin dose (300 IU/k after induction of general anesthesia. Central venous an urinary catheters were introduced, and blood pressur heart rate, and electrocardiogram were monitored contir uously. Fluid replacement was restricted to account fc the crystalloid load infused with the controlled reperfu sate, and furosemide (10 mg) was given intravenously, a needed.

The common, superficial, and deep femoral arterie were exposed and isolated by a standard groin incision. A common femoral longitudinal arteriotomy was made jus above the bifurcation, and all thromboembolic materia was removed from the iliac, superficial, and femora arteries with a Fogarty catheter. The distal vessel was not irrigated. A wire-reinforced 22F cannula was inserted into the iliac artery via the same arteriotomy to aspirate autogenous oxygenated blood for subsequent admixture with the reperfusate solution (Table I). This cannula was connected to the blood line (Fig. 1) of the reperfusion set (HP Medica, Augsburg, Germany), which also contained a smaller caliber tubing for delivery of asanguineous solution. Both tubing lines were inserted into the head of a single roller pump, thereby permitting delivery of oxygenated autogenous blood and reperfusate solution (Dr. F. Köhler Chemie GmbH, Alsbach-Hähnlein, Germany) at a ratio of 6 to 1 (6 parts blood and 1 part asanguineous solution) to achieve the composition described in Table II.

The lines were connected with a Y connector beyond the pump, and the modified blood reperfusate solution was channeled through a heat exchanger and an arterial filter. An additional Y connector was added to the delivery line for connection of two reperfusion cannulas to be inserted into the superficial and deep femoral arteries. These vessels were each cannulated with a 10F dual-lumen catheter with self-inflating balloons (Research Medical Inc., Salt Lake City, Utah) to allow individual pressure measurement during reperfusion. The system was deaired before cannulation of the superficial and deep femoral arteries. Controlled reperfusion flow rate was adjusted by the roller pump at 150 to 250 ml/min. A tubing clamp on each perfusion line was used to change resistance, as needed, to ensure that intravascular pressure in each vessel never exceeded 60 mm Hg.

Suturing of the venous patch with 5-0 monofilament suture to close the longitudinal arteriotomy was started during the 30-minute limb reperfusion interval. The cannulas were then removed and the patch anastomosis completed to restore normal blood supply. Arterial systolic pressure was kept below 120 mm Hg with systemic vasodilators within the first 24 hours after the operation. Systemic heparin was continued for 2 days to keep activated clotting time at 150 seconds, and conversion to warfarin sodium was undertaken whenever a source of embolism (i.e., cardiac) was documented.

Statistical analysis. Statistical analysis was done with the EPISTAT and the BIAS computer package provided by the Johann Wolfgang Goethe-University Frankfurt/

Table II. *Final composition of the controlled limb reperfusate (after mixing with blood in a ratio of 6 to 1)*

K^+ (mmol/L)	4.1 ± 0.3 (3.4–5.1)
Na^+ (mmol/L)	132 ± 1 (130–135)
Ca^{++} (mmol/L)	0.32 ± 0.04 (0.26–0.40)
Glucose (mg/dL)	439 ± 44 (335–560)
Po_2 (mm Hg)	147 ± 18 (85–195)
Pco_2 (mm Hg)	31.1 ± 2.5 (24.6–37.6)
pH	7.47 ± 0.03 (7.37–7.55)
Hemoglobin (gm/L)	8.4 ± 0.3 (7.8–8.9)
Osmolarity (mOsm/L)	335 ± 12 (320–350)

Data are mean ± standard error of the mean (range). Po_2, Oxygen tension; Pco_2, carbon dioxide tension.

Table III. *Preoperative data (n = 19)*

Age (yr)	56 ± 4
Male/female	14/5
Site of occlusion	
Infrarenal aorta	3/19
Iliac artery	4/19
Femoral artery	10/19
Popliteal artery	2/19
Complete/incomplete ischemia	18/1
Cardiogenic shock	32% (6/19)
Muscle contracture (thigh or shank)	53% (10/19)
Duration of ischemia (hr)	26 ± 6

Data are mean ± standard error of the mean.

M., Germany, in conjunction with Dr. H. Ackermann, a biomathematician (Johann Wolfgang Goethe–University Frankfurt/M., Germany). Comparisons between groups were done with the one-way analysis of variance and ominal data by Fisher's exact test. Data are expressed as mean ± standard error of the mean. Differences were onsidered significant at a p value less than 0.05.

Results

Preoperative data. Patients' ages ranged from 19 to 83 years (Table III). Fourteen patients were male and five female. Of these, six patients (32%) were in cardiogenic shock requiring both inotropic support and intraaortic balloon counterpulsation. The femoral artery was the site of vascular occlusion in 16 patients; three patients had acute infrarenal aortic occlusion with subsequent bilateral femoral artery occlusion. The mean ischemic interval averaged 26 hours (range 6 to 39 hours). All limbs were pulseless, pale, and paralyzed, and patients who were not intubated complained of pain and paresthesia. Muscle contracture of the thigh or shank occurred in 10 of 19 patients (53%). The decision to offer controlled limb reperfusion was based on (1) clinical concern that severe postischemic syndrome would

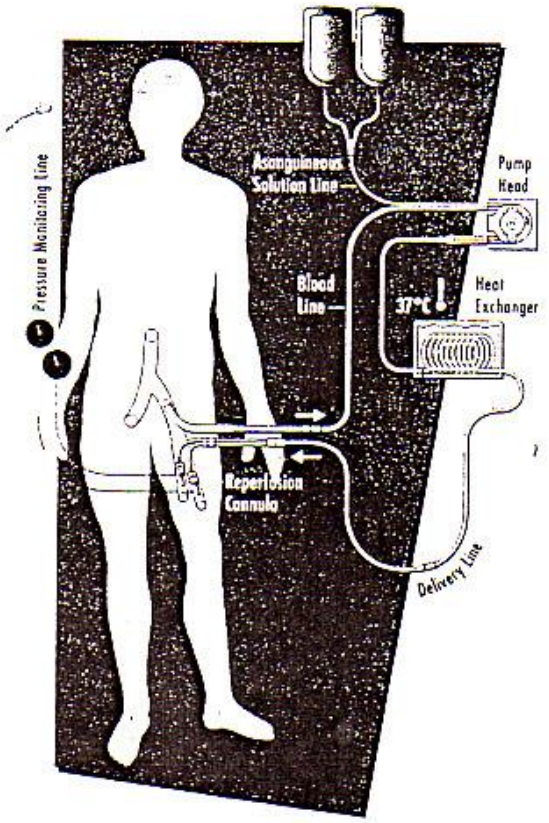

Fig. 1. Schematic representation of the clinical reperfusion set for controlled limb reperfusion. The iliac artery is cannulated to withdraw autogenous oxygenated blood from the patient. This blood is mixed with an asanguineous solution at a ratio of 6:1, administered at normothermia (37° C) through the heat exchanger, and delivered into the superficial and deep femoral arteries while intraarterial pressures are being monitored.

follow normal blood reperfusion or (2) the fear that primary amputation would be necessary because of contracture.

Intraoperative data. The external iliac artery and the superficial and deep femoral arteries could be cannulated via the longitudinal arteriotomy after thrombectomy in 18 of 19 patients. Iliac artery thromboendarterectomy was needed in one patient to allow proximal cannulation. Systolic blood pressure fell slightly while the reperfusion system was primed with the patient's autologous oxygenated blood, and this was reversed after controlled reperfusion was initiated. Systemic blood pressure, central venous pressure, systemic vascular resistance,

Table IV. *Arteriovenous difference of electrolytes, glucose, Po_2, Pco_2, and pH during controlled limb reperfusion in patients*

	Duration of controlled limb reperfusion			
	1 min	*5 min*	*15 min*	*30 min*
Electrolytes				
K^+ (mmol/L)	0.2 ± 0.8	−0.6 ± 0.1	−1.2 ± 0.1	−0.9 ± 0.3
Na^+ (mmol/L)	−2 ± 2	−3 ± 1	−3 ± 1	−3 ± 1
Ca^{++} (mmol/L)	−0.5 ± 0.3	−0.4 ± 0.2	−0.4 ± 0.2	−0.4 ± 0.2
Glucose (mg/dl)	74 ± 37	143 ± 66	165 ± 59	186 ± 53
Blood gases				
Po_2 (mm Hg)	113 ± 22	127 ± 26	99 ± 23	94 ± 19
Pco_2 (mm Hg)	−16 ± 4	−13 ± 2	−18 ± 3	−13 ± 3
pH	0.12 ± 0.02	0.06 ± 0.05	0.14 ± 0.02	0.11 ± 0.02

Data are mean ± standard error of the mean.

Table V. *Maximal postoperative serum values for enzymes, creatinine, blood urea nitrogen, and potassium*

Enzymes	
CK (U/L)	20,414 ± 7,710
AST (U/L)	455 ± 184
LDH (U/L)	1,356 ± 540
Renal data	
creatinine (mg/dl)	2.4 ± 0.7
BUN (mg/dl)	65 ± 8
Electrolytes	
K^+ (mmol/L)	5.3 ± 0.5

Data are mean ± standard error of the mean. *CK*, Creatine kinase; *AST*, aspartate aminotransferase; *LDH*, lactic dehydrogenase; *BUN*, blood urea nitrogen; K^+, potassium.

heart rate, and urine output did not change, and each patient received approximately 800 ml of crystalloid solution (range 214 to 1285 ml) as the 6:1 blood/reperfusate solution was delivered. Reperfusion rates averaged 150 to 250 ml/min (range 50 to 300 ml/min) to keep intravascular pressure between 50 and 60 mm Hg. Normal arterial inflow was restored by vein patch closure of the arteriotomy after decannulation.

Twelve of 19 patients underwent fasciotomy, which was performed prophylactically in the initial patients because severe limb edema was expected. Our early experience showed that most fasciotomies were unnecessary; therefore this was done subsequently only if severe limb swelling followed revascularization.

Intraoperative metabolic data (Table IV). Minimum and significant changes in serum, potassium, sodium, and calcium concentrations occurred during the 30-minute period of controlled limb reperfusion, and severe hyperkalemia (>2 mEq/L increase in serum K^+) never occurred. In contrast, glucose uptake rose progressively during the period of regional limb reperfusion, as arteriovenous glucose difference exceeded 140 mg/dl after 5 minutes. Substantial oxygen extraction occurred throughout the entire controlled reperfusion period, as arteriovenous oxygen difference ranged between 94 and 127 mm Hg. Effluent femoral vein pH remained in the normal range of 7.3 to 7.4, even though carbon dioxide is released into the venous circulation.

Postoperative systemic alterations. Table V shows maximum values for serum, potassium, creatinine, and blood urea nitrogen. Massive creatine kinase (CK) release occurred in all patients, but the increase in K^+, creatinine, and blood urea nitrogen was negligible. The average postoperative fluid requirements were 30 ml/kg for the first 24 hours.

Postoperative clinical data. Sixteen of 19 patients survived (84%), with deaths occurring in three of six patients undergoing controlled limb reperfusion during cardiogenic shock. No systemic postoperative complications occurred in survivors, inasmuch as none required hemodialysis, had rhythm disturbances, or had lung changes. All fasciotomies but one were closed between the fourth and eighth postoperative days. Each of the 16 survivors had full sensory and motor functional recovery of both limbs at the time of discharge as assessed by the discharging surgeon.

Only one death was associated with the reperfusion process. Progressive congestive heart failure and multisystem organ failure developed in a patient with cardiogenic shock who had acute distal aortic occlusion. A second death was due to irreversible low output syndrome, and the third death was due to cerebral hypoxia and sepsis in a patient who underwent acute coronary revascularization after arriving in the operating room while undergoing cardiopulmonary resuscitation.

Table VI. *Clinical results after thromboembolectomy for acute arterial occlusion in 2392 patients*

Reference	No. of patients	Ischemic time	Mortality (%)	Amputation (%)	Nonfunctional (%)	Mean age
1	17	1 hr–7 days	11.8	23.5	17.6	59 yr
53	94	1–21 days	13.7	5.4	No data	72.8 yr
54	246	1 hr–42 days	29.7	5.3	9.8	62 yr
55	142	1 hr–30 days	28	9	No data	No data
56	224	No data	23.1	9	No data	63 yr
57	118	No data	19.5	31	No data	No data
58	91	1 hr–8 days	34	11	No data	77 yr
2	85	1 hr–7 days	41	40	No data	No data
59	124	1 hr–?	30	22	No data	No data
60	100	1 hr–30 days	26	10	No data	67 yr
61	128	No data	34	5	No data	68 yr
62	125	1 hr–24 hr	25.6		No data	No data
63	119	1 hr–>8 days	27	18	No data	No data
64	142	1 hr–>24 hr	27	8.5	No data	65.5 yr
65	66	1 hr–>24 hr	34.8	6.1	No data	No data
66	65	1 hr–>12 hr	48	12	No data	No data
67	122	1 hr–48 hr	10.6	11.5	8	No data
68	221	No data	37	27	No data	77 yr
69	163	No data	14.7	8	16	62.5 yr

Discussion

Limb ischemia in patients who have had cardiac operations is an infrequent but life-threatening complication that occurs most commonly after prolonged transfemoral intraaortic balloon counterpulsation. The reported prevalence varies between 9% and 12%,[22-27] and amputation is needed in 1% of patients.[28] Limb ischemia after insertion of an IABP is due to either flow obstruction by the cannula or mechanical vessel wall trauma during insertion and accentuated by the intense vasoconstriction that accompanies the low cardiac output in these patients.[29-31] Ischemic limb complications are more common in patients with preexisting arterial occlusive disease,[32] in women[33, 34] because of their smaller caliber vessels, and in patients in whom larger IABP catheters are used.[35] Recommended prophylactic measures include transaortic IABP insertion,[36, 37] use of shunt sheaths with side holes,[38] sheathless IABP insertion,[39, 40] iliac artery balloon angioplasty if the vessel is stenotic,[41] and papaverine treatment.[42] Unfortunately, limb ischemia remains the major complication after transfemoral IABP insertion despite these efforts. Additional causes of limb ischemia include acute dissecting aneurysms, presence of aortoiliac disease in patients who undergo percutaneous femoral arterial cannulation for instituting cardiopulmonary bypass for emergency cardiac operations,[43, 44] or circulatory support during angioplasty.[45]

The mortality rate after primary amputation for acute limb ischemia is approximately 1%, whereas mortality increases to approximately 20% to 30% (range 10.6% to 48%) after revascularization (Table VI). Reperfusion injury of tissue jeopardized by the preceding ischemic period is the principal cause for increased morbidity and mortality after acute limb revascularization.[3, 4] Skeletal muscle has been shown to be relatively intact biochemically and structurally even after prolonged periods of ischemia,[6, 46] yet several experimental and clinical reports indicate that sudden restoration of normal blood flow supply may cause severe edema, rhabdomyolysis, and leakage of myocyte content into the plasma.[46-48] The resultant massive washout of lactate, potassium, and myoglobin may produce life-threatening myoglobinuric renal failure, hyperkalemia, disseminated intravascular coagulation, and acute cardiomyopathy.[1, 2, 49]

We[4-10] have demonstrated experimentally that local and systemic complications after normal blood reperfusion can be reduced significantly if the vulnerable skeletal muscle is managed during the first 30 minutes of restoration of its blood supply by the controlled limb reperfusion strategy. Using a model of acute infrarenal aortic occlusion for 6 hours, we[6, 10] developed evidence that uncontrolled normal blood reperfusion produced by suddenly releasing the aortic occlusion reduced oxygen consumption, produced marked edema and massive CK and potassium release, and increased vascular resistance severely, causing low reflow. The result was decreased recovery of range of motion of

the knee joint. Biopsy of the jeopardized muscle before reperfusion and evaluation of the tissue with an electron microscope showed structural integrity,[6] and this observation emphasized the importance of developing a reperfusion method that could limit injury to skeletal muscle jeopardized by ischemia otherwise destined for necrosis. These adverse effects of normal blood reperfusion were relatively comparable with those after normal blood reperfusion in ischemic cardiac myocytes. Our management strategy is based on the salutary effects shown when controlled reperfusion was provided to cardiac muscle after regional and global ischemia.[15-18]

The severe damage produced by normal blood reperfusion could be reduced substantially when the conditions of reperfusion and the composition of the reperfusate were controlled for 30 minutes in a fashion somewhat similar to that applied to regionally ischemic cardiac muscle.[15-18] The controlled limb reperfusion phase restored oxygen consumption to control levels, avoided tissue edema, limited CK and potassium release significantly, decreased vascular resistance, restored limb flow to greater than control values, and restored a normal range of motion to the knee joint.[4, 7, 8]

The findings of the safety and superiority of controlled limb reperfusion led to the current application of these principles in 19 patients with prolonged severe ischemia averaging 26 ± 6 hours. The treatment protocol was used despite the presence of ischemic muscle contracture in 53% of patients. The reversal of ischemic contracture is, to our knowledge, a new finding that irreversible damage has not yet occurred despite the clinical sign of extensive ischemic damage. The only deaths occurred in three of six patients who were in profound cardiogenic shock before the operation (16% mortality). Sixteen of 19 patients (84%) were discharged with salvaged limbs, and renal, pulmonary, and cardiac complications did not occur.

No randomization was applied to the patient cohort undergoing controlled limb reperfusion (1) because the experimental data in both crystalloid[4-9] and blood-perfused models[10] showed significant improvement of skeletal muscle viability as compared with that obtained with unmodified reperfusion and (2) because of our successful preliminary clinical experience with this technique.[50]

Reperfused skeletal muscle exhibited active oxygen and glucose uptake (see Table IV). We interpret these findings as evidence of active cellular repair during the initial controlled reperfusion phase, which restores some of the cellular hemostatic mechanisms that render jeopardized skeletal muscle able to cope with the subsequent normal blood reperfusion. The marked washout of CK (see Table V) reflects the severe ischemic insult to the skeletal muscle, yet the recovery of function indicates that extensive necrosis had not yet occurred. However, because the sensory and motor functional recovery was not assessed by neurologists, it is possible that some slight alterations might not have been recognized by the discharging physician. Our finding of high CK values and functional recovery of the skeletal muscle is paralleled by reports in cardiac muscle where the mode of reperfusion had no effect on the CK-MB values, whereas a significantly better recovery of regional wall motion could be achieved by an initial treatment of the previously ischemic myocardium during the initial reperfusion phase.[16-18] Nevertheless, it is likely that the massive release of the large-molecule CK will indicate some cellular death. Additionally, no renal impairment was detected in surviving patients, and slight hyperkalemia did not cause cardiac rhythm disturbances.

The surgical technique applied technology used routinely for delivery of cardioplegic solutions during operations—a heat exchanger, tubing of different caliber to fix the blood/reperfusate ratio, and method of monitoring reperfusion pressure. The 6:1 ratio (blood/crystalloid solution) was used to limit fluid overload attendant with the crystalloid component of the reperfusate solution, especially in patients with congestive heart failure or cardiogenic shock. Priming of the current system included withdrawal of 400 ml of autogenous blood from the patient, causing slight hypotension. This was reversed readily after controlled limb reperfusion was initiated, and this volume was returned to the patient together with the crystalloid solution. The total volume of reperfusate was approximately 6 L (200 ml/min in 30 minutes), but the crystalloid component was limited to 800 ml by the 6:1 blood/reperfusate ratio. This crystalloid infusion did not produce signs of hypervolemia. Future reperfusates will likely include higher ratios of blood to crystalloid solution to reduce the volume of crystalloid solution further.

The reperfusate composition differs slightly from the cardiac reperfusate on which it is based, and which is diluted by the volume of the extracorporeal circuit during cardiac surgery. Modifications were made to limit potential adverse systemic effects, because the effluent returns to the patient by way of the femoral vein. For these reasons, we reduce the volume of tromethamine buffer to decrease the tendency for

systemic alkalosis, decrease the amount of citrate-phosphate-dextrose to limit the propensity for systemic hypocalcemia, and lower the concentration of glutamate and aspartate, because amino acids may be associated with systemic vasodilatation.[51]

The selected antioxidant was allopurinol because of its availability and reported use in patients with cardiac disease.[52] Other antioxidants, especially those having those site-directed effects, may be used subsequently (i.e., superoxide dismutase, catalase, and deferoxamine). We suspect also that subsequent protocols will include interventions that alter other aspects of reperfusion injury. These protocols may incorporate mechanisms of decreasing leukocytes or their adherence and modifications of endothelial function (i.e., L-arginine or nitric oxide) as more is learned about reperfusion injury.

Controlled reperfusate strategy was straightforward and simple to establish in patients requiring cardiac operations, inasmuch as similar principles are used for blood cardioplegic delivery during myocardial protection. Cardiac surgeons and perfusionists are familiar with these principles of organ protection. This study was restricted to patients with impending limb loss as a result of prolonged ischemia. We suspect that future applications may include the prophylactic use of controlled limb reperfusion with prolonged ischemia (>3 hours after femoral vessel cannulation), as well as for extremities exhibiting signs of ischemia during prolonged use of an IABP.

We conclude that clinical application of an experimentally based strategy of controlled limb reperfusion is feasible and useful in the setting of acute limb ischemia in patients having cardiac operations. Its feasibility is enhanced by the fact that the tools for its application are present during most cardiac procedures in which blood cardioplegia is used (i.e., heat exchanger, differential tubing sets, reperfusion cannulas). The encouraging early results in patients with profound ischemia (averaging 26 hours, including contracture) may lead to the prophylactic use of revascularization after prolonged femoral vessel cannulation and limit postreperfusion syndrome further. We hope this report stimulates others to test the applicability of controlled reperfusion in limbs otherwise destined for severe damage. Reproduction of our results by others should decrease the prevalence of postischemic syndrome after acute limb ischemia in patients having cardiac operations, as well as in other patients at risk for this devastating complication of revascularization.

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