But if with this term we state that any stenotic vessel has to be grafted, regardless of its importance and size, without any demonstration of ischemia in the territory, this definition of "complete revascularization" has to be forgotten. Today we have at our disposition many tools to properly investigate the importance of a stenosis, and complete revascularization (interventional, surgical, or combined) of all ischemic territories is always our goal. However, the timing can change and the strategy can differ from patient to patient.

In our article I thought that the real problem was focused not on the surgical indication (single LAD disease or multiple-vessel disease), which can be debatable, but on the future of the graft. (Is this approach able to guarantee satisfying permeability of the graft? Can this strategy be considered safe and reproducible?) For this reason I mixed all my patients, but the patients with "multiple-vessel disease" in this group, with only a few exceptions (hybrid procedures), are not the same patients we operate on every day in our daily practice.

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The use of the calcium antagonist nicardipine in arterial coronary bypass surgery

To the Editor:

In the December 1998 issue of the Journal, Acar,[1] Possati,[2] and their associates independently provided important data on the 5-year patency rate of the radial artery graft.

Both groups acknowledge the impressive spastic characteristics of this arterial conduit and advocate perioperative release of spasm and routine administration of the calcium antagonist diltiazem to the patients for 1 year or more. Interruption of this treatment after 1 year or later during follow-up did not seem to adversely influence either clinical outcome or 5-year patency. This observation is related to the apparent loss of reactivity of the radial graft over time.

The use of antispasmodic drugs for patients receiving arterial grafts is a common practice, although so far no study has demonstrated its value on the surgical outcome, keeping in mind that "spasm" may be confounded by other factors such as surgical technique, familiarity with arterial grafts, and perioperative conduit preparation. Since 1994, we have been using nicardipine hydrochloride, the first intravenously administered dihydropyridine calcium channel antagonist, for sequential or bilateral internal thoracic artery grafting. The patients are started perioperatively on intravenous nicardipine hydrochloride (0.25 μg/kg per minute) after a 1 mg intravenous bolus. The drug is titrated according to the systemic vascular resistance and discontinued on the second postoperative day. For patients receiving a radial artery graft, we added to this protocol gentle hydrostatic dilatation of the conduit with 1% papaverine and oral administration of nicardipine 20 to 30 mg three times a day after discontinuation of intravenous perfusion on day 2. The first 50 patients undergoing myocardial revascularization with a radial graft using that protocol were observed. A total of 150 anastomoses were performed with a mean of 3.0 anastomoses per patient, and 108 arterial grafts were used for the completion of 111 coronary distal anastomoses. Fifty radial arteries were used for 52 distal anastomoses, and 36 venous grafts were used for 39 distal anastomoses. The radial artery was placed on the obtuse marginal branch in 58% of cases, diagonal branches in 13%, and the right coronary artery in 29%. Proximal anastomoses were done directly on the aorta in 80% of cases.

The operative mortality was 4% because of 2 cases of fulminant pulmonary sepsis. There was no evidence of perioperative myocardial infarction or arterial graft hypoperfusion syndrome. Mean creatine kinase MB level at 18 hours was 36 μg/L. Neither ischemic anomalies of the electrocardiogram nor wall motion abnormalities on discharge transthoracic echocardiography were detected. Angiography performed in the last 20 patients showed a 98% (51/52) permeability rate for all grafts. Nineteen of 20 radial grafts were patent. A moderate spasm (40%) developed in the middle part of the conduit in one radial artery.

Our experience with the radial artery is part of a larger experience with the perioperative use of nicardipine in more than 550 cases of bilateral or sequential internal thoracic artery grafting. This protocol has virtually eliminated internal thoracic artery spasm or hypoperfusion in our practice. Nicardipine has several potential advantages over diltiazem. Nicardipine is a more potent and selective arteriolar vasodilatory agent, which also has the capability to inhibit endothelin-induced vasoconstriction.[3] This drug has neither chronotropic, dromotropic, nor inotropic negative effects. Its short action duration makes it convenient for perioperative management, and it is well tolerated in association with β blockers in the routine management of patients with coronary artery disease. Nicardipine also has a documented cardioprotective effect on ischemic myocardium, and some data suggest cardioprotective effects during cardioplegic cardiac arrest.[4] We therefore suggest that the time has come to assess the real need for antispasmodic drugs in arterial coronary bypass grafting and to determine the most adequate pharmacologic management for patients receiving arterial grafts such as the radial artery.

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Regional cerebral blood flow and regional glucose use during rewarming after hypothermic cardiopulmonary bypass

To the Editor:

We read with great interest the article titled "Regional Cerebral Blood Flow During Rewarming of Cardiopulmonary Bypass Correlates With Posthypothermic Regional Glucose Use" (J Thorac Cardiovasc Surg 1998;116:503-10). The authors are to be congratulated on the timely demonstration of the potentially deleterious derangements caused by a period of hypothermic perfusion followed by rewarming.

Because cerebral blood flow and metabolism remained coupled during hypothermic (20°C) perfusion for 60 minutes, but apparently not during or after rewarming, particularly in the parietal cortex, rewarming was considered to be the culprit. However, the 60-minute period of hypothermic perfusion used in their protocol 2 might not have been long enough to assess the effects of hypothermia per se, since reducing the temperature to only 29.5°C can provide protection for 60 minutes of total blood flow cessation in a rabbit model of spinal cord ischemia with pH-stat ventilation.[1] Metabolic derangements may result from inadequate oxygen delivery caused by the leftward shift of the oxyhemoglobin curve and alkalosis induced by hypothermia. Given the relatively short hypothermic period with continuous perfusion, these derangements may have been present before rewarming, but may have been too subtle even for the sophisticated methods used by the authors. Those metabolic derangements could not recover fast enough during the rewarming time and indeed were exaggerated by the increased metabolic rate. Thus they became apparent only after body temperature was returned to normal. If metabolism was impaired and oxygen could not be consumed, there was no need for flow; consequently, decreased flows were observed.

Unfortunately, the authors studied only animals with alpha-stat strategies for pH management during hypothermia. Alpha-stat strategies have been considered as the standard for many years to avoid the so-called "luxury perfusion" of pH-stat management and consequent increased chances of microembolization. Such "luxury perfusion" may actually not be a "luxury" but a necessary compensatory mechanism for the leftward shift of the oxyhemoglobin dissociation curve induced by hypothermia. A recent report[2] indicated better neurologic outcome of pH-stat over alpha-stat strategies for deep hypothermic cardiopulmonary bypass in infants, an observation that could have been anticipated and should not be surprising. Whether this improved outcome was due to better blood flow[3] or better oxygen delivery and use is irrelevant. However, it is in line with the physiologic principles of the remarkable constancy of the expiratory carbon dioxide concentration remaining close to that observed in normothermic conditions in hibernating animals, regardless of the body temperature, by ventilatory rate adjustment, which is the nature-provided pH-stat management. This is most obvious in poikilothermic animals,

The authors have the modern methods to resolve the controversial issue of whether alpha-stat is better than pH-stat management or vice versa. We believe the neurologic injury coincidental to hypothermic perfusion is the result of the algebraic sum of both factors, positive metabolic factors and negative microembolization. The relative role of each one is variable for each individual depending on various factors—the duration of the hypothermic perfusion period, the rate of cooling and rewarming, the hemodynamic status before and after cardiopulmonary bypass, the type of oxygenator, and the lack of pulsatility. The metabolic component is maximized by pH-stat strategy of the ventilation, which restores oxygen delivery to the tissues. However, if the supposedly positive metabolic influences of hypothermia are less than ideal because of the impaired oxygen delivery caused by alpha-stat management, which would be equivalent to hypoxic perfusion, the negative influence of microembolization will become relatively more significant. If sustained for long enough, it would be detrimental by itself, thus resulting in overt neurologic impairment.

Hemodynamic superiority of pH-stat over alpha-stat strategies in dogs during surface-induced hypothermia was reported years before the term of pH-stat management was coined.[3] It would not be surprising if the reported hypotension during the hypothermic state and the flow-metabolism derangements would be at least abated, if not completely prevented, provided that expiratory carbon dioxide concentrations of the oxygenator are maintained constant close to 5% to 6%. pH-Stat strategies are used by controlling the oxygen/blood flow ratio of cardiopulmonary bypass (or gasses, by adding carbon dioxide, if necessary), not only during cooling but during rewarming as well.

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