Hypothermia and anoxic arrest

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Myocardial hypothermia, induced by topical cooling, selective cooling by perfusion of the coronary arteries, or by a combination of these methods, has proved to be an effective method for protection of the myocardium during cardiac surgical procedures which require interruption of normal coronary perfusion. For patients undergoing coronary bypass grafting procedures, selective myocardial hypothermia (15 to 18 C) resulting in cardiac arrest over a period of several minutes has been shown to be superior to ventricular fibrillation and moderate hypothermia (30 to 35 C) as assessed by rate of physiologic recovery of ventricular function, myocardial creatine phosphokinase (MB CK) isoenzyme release, and by study of myocardial ultrastructure.[1] Hypothermic cardioplegia, produced by a cold, potassium-containing solution, has also been shown to be an effective method of myocardial protection. The latter technique produces almost immediate and complete cessation of electromechanical activity, thereby further reducing metabolic demands during the period of aortic clamping. Potassium-induced cardioplegia has been shown experimentally to result in less depletion and more rapid restoration of myocardial energy stores than anoxic

arrest at similar temperatures.[2] Despite the theoretical advantages of hypothermic cardioplegia, few clinical studies, particularly in patients undergoing coronary artery grafting procedures, are available to document the beneficial effects noted in the experimental studies.

To evaluate these two methods of myocardial protection (hypothermic anoxic arrest, hypothermic cardioplegia), 50 consecutive patients undergoing multiple coronary bypass grafting procedures on one surgical service during a 4-month period were evaluated using serial MB CK determinations and electrocardiographic changes as indices of myocardial injury. Twenty-five patients had distal coronary anastomoses performed during a single period of aortic cross-clamping following cooling of the myocardium with the perfusate (12 C) and topically with iced Ringer's solution (4 C). During the period of clamping, the myocardial temperature in the anterior ventricular septum ranged from 20 to 28 C. The other 25 patients received an infusion of a hypothermic (4 C) cardioplegic solution containing 30 mEq/liter of potassium chloride, dextrose, mannitol, and albumin, and adjusted to a pH of 7.55 and an osmolality of 330, in conjunction with topical cooling using 4 C Ringer's solution. The average myocardial temperature in this group during the period of clamping ranged from 12 to 20 C. In both groups, all proximal anastomoses of the vein grafts to the aorta were performed during rewarming with the heart beating and at low left atrial pressures. Serial MB CK determinations were obtained 4, 24, 48, 72, and 168 hours following operation. To obtain an estimate of the total amount of MB CK released postoperatively, the area described by a line connecting each MB CK valve at each sampling time up to 48 hours was calculated and was expressed as International Units (I.U./L·hours).[3] Standard electrocardiograms were obtained on the day of operation and on the first, second, third, and seventh postoperative days. Using the criteria of Righetti et al,[3] new, persistent (>24 hours) Q waves in three contiguous electrocardiographic leads, one of which was at least 0.4 seconds in duration, in the absence of a conduction abnormality or marked shift in the QRS axis, were considered diagnostic of myocardial infarction. Horizontal S-T segment depression of 1 ml or more, associated with T wave inversion in two or more adjacent leads that persisted for at least 48 hours was considered a significant change suggestive of ischemia. Other ST-T wave changes were considered to be nonspecific.

The patients in the two groups were comparable in regard to age, sex, and severity of disease. The mean number of arteries bypassed was 3.2 in the hypothermic anoxic arrest group and 3.8 in the hypothermic cardioplegia group. The durations of cardiopulmonary bypass and aortic clamping were significantly greater in the patients with hypothermic cardioplegia (*Table 1*).

The results are summarized in *Table 2*. There were no hospital deaths in either group. Only one patient (hypothermic anoxic arrest) required support with catecholamines in the intraoperative or postoperative period. This patient had significant impairment of left ventricular function preoperatively, but did not demonstrate electrocardiographic or enzy-

Table 1. Durations of cardiopulmonary bypass and aortic clamping

	Hypothermic anoxic arrest (n = 25)	Hypothermic cardioplegia (n = 25)	p value
Mean age (years)	53 ± 1.9*	55 ± 1.3	N.S.†
Percent males	80	92	N.S.
Mean number of vessels diseased	2.7 ± 0.1	2.8 ± 0.1	N.S.
Mean number of grafts inserted	3.2 ± 0.2	3.8 ± 0.2	0.03
Mean duration of cardiopulmonary bypass (minutes)	63 ± 3.5	74 ± 2.5	0.01
Mean duration of aortic clamping (minutes)	29 ± 2.0	40 ± 2.0	0.0003
Myocardial temperature range	20 – 28 C	12 – 20 C	

* Standard error of the mean.
† Not statistically significant.

Table 2. Results: anoxic arrest and cardioplegia

	Hypothermic anoxic arrest (n = 25)	Hypothermic cardioplegia (n = 25)	p value
Postoperative catecholamine support	1	0	N.S.
New myocardial infarction	0	1	N.S.
MB CK detected postoperatively	19	19	N.S.
Mean integrated MB CK area (I.U./L·hours)	900 ± 340	530 ± 116	N.S.
Number of patients with integrated MB CK area			
465 I.U./L·hours	8	11	N.S.
1000 I.U./L·hours	5	4	N.S.

matic evidence of myocardial infarction postoperatively. No patient required preoperative or postoperative support with the intra-aortic balloon.

Using the electrocardiographic criteria outlined above, one myocardial infarction (inferior) developed in a single patient (hypothermic cardioplegia). No patient in either group developed ST-T changes suggestive of ischemia. MB CK was detected postoperatively in 19 (76%) of the patients with hypothermic ischemic arrest and in 19 of those with ischemic cardioplegia. The mean (±SEM) integrated area of MB CK was 900 ± 340 I.U./L·hours for the hypothermic ischemic arrest group, and 530 ± 116 I.U./L·hours for the hypothermic cardioplegia group. Eight patients (32%) with hypothermic ischemic arrest and 11 (44%) with hypothermic cardioplegia had mean MB CK levels above 465 I.U./L·hours (considered as the upper limit of normal in previous studies[3]), and five (20%) and four (16%) respectively had mean levels above 1000 I.U./L·hours. None of these differences was statistically significant.

The data indicate that these two methods of hypothermic myocardial protection for extensive myocardial revascularization procedures are comparable with regard to the extent of myocardial injury produced as assessed by electrocardiographic and enzymatic criteria. Impaired cardiac performance requiring inotropic support was present in only one of the 50 patients postoperatively. In previous studies using electrocardi-

ographic[3] and enzymatic[1] criteria to assess myocardial damage, a higher incidence of perioperative injury following coronary bypass grafting was observed with lesser degrees of hypothermia and spontaneously occurring ventricular fibrillation, suggesting that more profound hypothermia in the absence of ventricular fibrillation provides greater myocardial protection.

While cardioplegic techniques provide a flaccid, quiet heart which facilitates the technical aspects of coronary bypass grafting, hypothermic cardioplegia was not shown in the study to provide any greater protection of the myocardium than hypothermic ischemic arrest. Either of the techniques appears superior to more moderate hypothermia and ventricular fibrillation as a method of myocardial protection during coronary bypass grafting procedures.

References

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