Regional Myocardial Temperature Variations with Asanguineous Potassium Cardioplegia

Jeffrey C. Crowley and William B. Pelley

PSICOR, Inc.
San Diego, CA
and
Alden H. Harken, Jr.

University of Colorado Health and Science Center
Denver, CO

Keywords: cardioplegia, administration; cardioplegia, crystalloid; cardioplegia, hypothermic; monitoring, temperature; potassium; myocardial preservation

Abstract

***(J. Extra-Corpor. Technol.* 19[4] pp. 415-419 Winter 1987,) The temperature of the myocardium during cardioplegic arrest is an indication of the extent to which that area has received both hypothermic and chemical protection. Regional myocardial temperatures were examined during asanguineous cardioplegic arrest to assess differences in temperatures, examine regional rates of rewarming, and to identify maximum temperature gradients. Myocardial temperatures were measured at the left ventricular apex, the right ventricular free wall and the interventricular septum in twenty patients undergoing coronary artery bypass using multidose asanguineous cardioplegic arrest. Aortic root temperatures were measured to identify the temperature of the cardioplegia and left heart blood temperatures during venting. Temperatures in the left ventricular apex ranged from 13.9° to 21.3°, right ventricular free wall from 15.9° to 21.9°, and the interventricular septum from 13° to 23.6°. Temperature variations in the three areas were as great as 15° and as little as 2°. Myocardial temperatures are 1) not predictable due to the nature of coronary artery disease, 2) variable despite optimal delivery technique, and 3) may vary due to cannulation and systemic blood temperature.**

This work conducted at the Hospital of the University of Pennsylvania, Philadelphia, PA

Direct communications to: William B. Pelley, PSICOR, Inc., 16818 Via del Campo Court, San Diego, CA 92127

Abstract submitted December 15, 1984
Manuscript submitted March 15, 1986
Accepted for publication August 20, 1987

Introduction

Delivery of hypothermic asanguineous cardioplegic solution (CPS) into the aortic root and subsequent aortic root venting has become an accepted method of myocardial protection and decompression during cardiac operations.[1-6] The method of cardioplegic delivery, the degree of hypothermia, the content of the CPS and the extent of myocardial protection have all been examined by previous investigators.[7-11] Presumably, the temperature of the myocardium during cardioplegic arrest is an indication of the extent to which that area has received both hypothermic and chemical protection. By the nature of the coronary artery disease being treated, however, delivery of cardioplegic solution may not be uniform throughout the myocardium.[12] Heterogenous myocardial protection might lead to heterogeneous myocardial injury. Suboptimal regional protection might predispose to local contractile injury and even post-operative arrhythmias.[13-19] Personal experience has shown that most surgical groups do not assess the degree of myocardial protection, some measure septal

temperature, while a few groups monitor multiple sites.

The purpose of this study was: 1) to examine regional myocardial temperature during asanguineous multidose cardioplegic arrest; 2) to assess differences in post-arrest temperature that might predispose to non-uniform injury; 3) to examine regional rates of warming that might identify the zones at greatest risk; and 4) to identify maximum temperature gradients that might predispose to heterogeneous injury promoting post-operative arrhythmias.

Materials and Methods

Twenty patients undergoing elective coronary artery bypass procedures were studied. Following standard anesthesia induction, the patients were cannulated with an ascending aortic arch cannula and a single venous cannula in the right atrium. CPS was delivered through a roller pump. Aortic root pressures were maintained at 80 mm of mercury during CPS administration. The initial CPS dose was given for 2 minutes and subsequent CPS doses were administered into the aortic root for 1 minute each at the conclusion of each distal anastomosis. The left ventricle was decompressed via the cardioplegic cannula with gravity drainage to the venous line of the cardiopulmonary bypass circuit. Topical cooling was utilized during aortic crossclamping.

Myocardial temperatures were measured at the aortic root, the left ventricular apex, the right ventricular free wall, and the interventricular septum. The aortic root temperature was measured to give an indication of CPS delivery temperature and left heart blood temperature during venting. Multiple myocardial temperature probes were used which had been checked against each other for accuracy.[a] Nasopharygeal and arterial blood temperatures were also measured. Immediate pre-cardiopulmonary bypass baseline temperatures were recorded. With initiation of cardiopulmonary bypass, the patient was cooled to 26° C. When the heart fibrillated, temperatures were again recorded and the aorta was cross-clamped and cardioplegic solution delivered. Temperatures were recorded at the completion of CPS delivery and every two minutes thereafter for the duration of the cross-clamp time.

Statistical analysis was performed using a statistical package for IBM computers.[b]

Results

During CPS administration, aortic root pressures were maintained at 80 mm of mercury. Cardioplegic solution flows varied from 200 ml to 400 ml per minute. Initial cardioplegic doses ranged from 400 ml to 700 ml, and subsequent doses generally delivered at the conclusion of each distal anastomosis ranged from 100 ml to 450 ml. Table 1 illustrates the temperature results at the conclusion of the initial dose of cardioplegia. There were no statistically significant differences in the three areas. Patients with multiple, stenotic lesions received less CPS than others due to the nature of their disease.

Crystalloid CPS temperature ranged from 1.4° to 15.4° during infusion. Higher CPS temperatures occurred in the latter stages of aortic cross clamping, particularly if rewarming had begun. During the period of aortic cross clamping, the temperature in the aortic root ranged from 16.6 to 26.9°. These temperature ranges are probably the result of not using bi-caval cannulation and total cardiopulmonary bypass. The temperatures in the left ventricular apex ranged from 13.9° to 21.3° while the right ventricular free wall ranged from 15.9° to 21.9°. The temperatures in the interventricular septum ranged from 13° to 23.6°. The arterial blood temperatures ranged from 24.2° to 28.4° and the nasopharyngeal temperatures were 26.4° to 28.4°. Individual temperature differences between any of the four areas measured in the heart were as great as 15° and as little as 2°. The mean temperature at fibrillation, in the aortic root was 25°, the left ventricular apex was 15°, the right ventricular free wall was 18°, and the septum was 10°. The mean arterial blood temperature was 20° and the nasopharyngeal was 28° (Figure 1).

Discussion

Measurement of temperature on the myocardium in three locations, the left ventricular apex, right ventricular free wall, and septum, and measurement of aortic root blood temperature have indicated a heterogeneity of temperatures across all four regions. Although regional myocardial temperature differences are well described in the literature, the effects of these independent temperature differences have not been well examined. Differences in cardioplegic delivery systems is one explanation for the variability in their temperatures across the myocardium. The method of cardioplegia administration, the pressure and/or flow during cardioplegia delivery, the volume of CPS used, the type of solution used (blood or crystalloid), the temperature of the CPS, and the duration of the CPS administration will ultimately affect the degree of hypothermic cardioplegia arrest. Grover and associates described the differences in administration of CPS via roller pump and that of the pressurized bag administration of cardioplegic solution.[20] Results of their study indicated that the

a TM, TMPN, Shiley Laboratories, Santa Ana, CA 92714
b Stats-2, Statsoft, 2832 East 10th Street, Tulsa, OK 74104

Table 1
Temperatures at End of First Infusion (Arrest)

Pt. #	Qb ml/min	AO C	LV C	RV C	SP C	LV-RV	LV-SP	RV-SP
1	325	7.8	14.4	18.8	8.1	-4.4	6.3	10.7
2	350	3.2	11.6	17.5	8.6	-5.9	3.0	8.9
3	300	3.4	14.1	24.6	14.7	-10.5	-0.6	9.9
4	325	4.9	8.4	3.5	10.9	4.9	-2.5	-7.4
5	300	8.0	18.0	17.2	11.7	0.8	6.3	5.5
6	325	5.6	11.4	19.8	8.8	-8.4	2.6	11.0
7	270	7.9	13.4	17.2	14.1	-3.8	-0.7	3.1
8	325	5.7	14.6	16.8	12.2	-2.2	2.4	4.6
9	300	5.9	21.6	14.7	16.3	6.9	5.3	-1.6
10	300	4.4	13.3	10.7	15.3	2.6	-2.0	-4.6
11	300	4.4	12.2	10.4	14.9	1.8	-2.7	-4.5
12	325	6.5	10.8	16.1	9.5	-5.3	1.3	6.6
13	325	2.0	17.8	7.5	12.0	10.3	5.8	-4.5
14	260	6.7	13.1	12.9	8.1	0.2	5.0	4.8
15	300	6.1	15.3	10.1	14.7	5.2	0.6	-4.6
16	200	4.2	17.0	13.3	14.6	3.7	2.4	-1.3
17	300	3.5	15.5	13.3	14.5	2.2	1.0	-1.2
18	300	2.9	11.2	14.8	10.5	-3.6	0.7	4.3
19	300	1.4	10.2	5.2	2.8	5.0	7.4	2.4
20	300	7.4	18.0	18.0	20.1	0.0	-2.1	-2.1
Mean	301.50	5.0950	14.0950	14.1200	12.1200	-0.0250	1.9750	2.0000
SD	31.3763	1.9819	3.2346	5.1404	3.8513	5.4011	3.2157	5.7686
T Value						-0.018	1.756	1.393
P Value						0.9336	0.0835	0.1686

LV = Left Ventricular Apex, RV = Right Ventricular Free Wall,
SP = Interventricular Septum, Qb = Cardioplegia delivery flow,
AO = Aortic Root

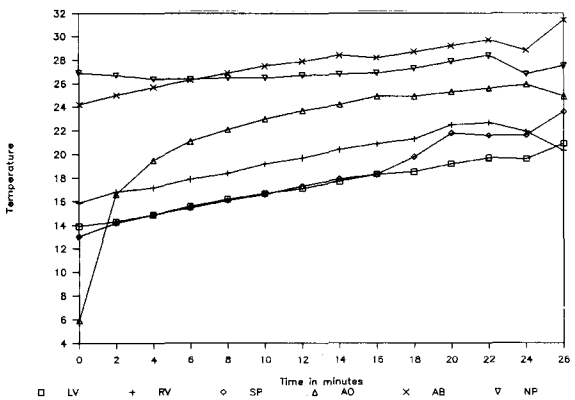

Figure 1: Regional Myocardial Temperature Variations
LV = Left Ventricular Apex, RV = Right Ventricular Free Wall, SP = Interventricular Septum, AO = Aortic Root, AB = Arterial Blood, and NP = Nasopharyngeal

pressurized bag method of administering CPS might limit the pressure delivered at the aortic root. Engleman and coworkers described a high volume crystalloid CPS technique purporting to provide additional hypothermic protection. Additionally, the high volume cardioplegic system appears to provide a more rapid metabolic recovery.[21]

The protective effects of systemic hypothermia have also been studied by numerous authors. Recently Rosenfeldt and associates found that deep systemic hypothermia had additional protective effects on the myocardium; however, the protection offered by systemic hypothermia may vary with venous cannulation techniques.[22] Warming of the right side of the heart by venous blood return has been implicated as the cause of suboptimal right heart preservation. The addition of

cold topical solutions around the myocardium during CPS infusion, continuous drip during the operation, and intermittently during the operation have led to additional hypothermic protection of the myocardium. Chiu indicates that topical hypothermia alone is inadequate protection, but in conjunction with infused cardioplegic solution provides more homogeneous hypothermia.[23] Hearse and Harlan have indicated that regions subject to inadequate preservation can be predicted from the pre-operative angiogram.[24-25] Areas distal to significant lesions tend to remain warmer. Intraaortic pressure of approximately 80 mm of mercury is necessary to deliver cardioplegic solution past the coronary stenosis.

Aortic root perfusion pressures of approximately 80 mm of mercury and as high as 85 to 100 mm of mercury as measured by Bretschneider in his myocardial temperature mapping studies are accepted as adequate for cardioplegic solution infusion.[26] Despite the monitoring of myocardial temperature, CPS temperature, and aortic root perfusion pressure, the results are conflicting. Fisk and associates have reported that intramyocardial temperatures are not predictable and may differ up to 17° C. between the anterior and posterior left ventricular sites.[27] This lack of predictability and variability in temperature may be due to such factors as collateral circulation or the ability of various cardioplegic vehicles to bypass stenosed areas.

Hilton and Ekroth have demonstrated the significance of critical coronary stenosis on the maldistribution of hypothermia across the myocardium.[28-29] Summarizing Rosenfeldt and Hartz in their work on the safety limits of solution pressure and temperature, CPS infusion pressures as high as 150 mm of mercury were used safely and solution temperature as low as 0° C. were infused into the myocardium via the aortic root with preservation of myocardial function.[30-31] They report that the use of colder cardioplegic solutions and higher infusion pressures may have an additional protective effect on areas not normally perfused.

In conclusion, there are many variables which may affect adequate myocardial protection during cardioplegic arrest. Temperature, flow, pressure, stenotic lesions, and hypertrophied muscle may all affect preservation techniques, as well as cannulation and time between infusions. The only effective method of ensuring adequate protection seems to be continuous temperature mapping of the entire heart. However, this is an impractical solution to the problem. While there may be some controversy over the effects of these variables, it is important to recognize that they exist, in order to be more adequately prepared to deal with complications.

References

1. Gay, W.A. Jr.: Potassium Induced Cardioplegia. *Ann. Thorac. Surg.* 20:95, 1975.
2. Jynge, P., Hearse, D.J., and Deheiris, J.: Protection of the Ischemic Myocardium. Ultrastructural, Enzymatic and Functional Assessment of the Efficacy of Various Cardioplegic Infusates. *J. Thorac. Cardiovasc. Surg.* 76:2, 1978.
3. Rousou, J.H., Engleman, R.M., and Dobbs, W.A.: The Optimal Potassium Concentration in Cardioplegic Solutions. *Ann. Thorac. Surg.* 32:75, 1981.
4. Engleman, R.M., Rousou, J.H., and Auvil, J.: The Safety of Prolonged Ischemic Arrest Using Hypothermic Cardioplegia. *J. Thorac. Cardiovasc. Surg.* 79:705, 1980.
5. Conti, V.R., Bertroman, E.G., and Blackstone, E.H.: Cold Cardioplegia Versus Hypothermia for Myocardial Protection. *J. Thorac. Cardiovasc. Surg.* 76:577, 1978.
6. Engleman, R.M., Rousou, J.H.: A New Approach to Left Ventricular Venting During Cardioplegic Arrest. *Ann. Thorac. Surg.* 28:603, 1979.
7. Hearse, D.J., Stewart, D.A., and Braimbridge, M.D.: Hypothermic Arrest and Potassium Arrest: Metabolic and Myocardial Protection During Elective Cardiac Arrest. *Circ. Res.* 36:481, 1975.
8. Rosenfeldt, F.L., Watson, D.A.: I. Development of an In Vitro Model of Myocardial Cooling: A Study of the Effect of Cardiac Size on Cooling Rate. *Ann. Thorac. Surg.* 27:7, 1979.
9. Buckberg, G.D.: A Proposed "Solution" to the Cardioplegic Controversy. *J. Thorac. Cardiovasc. Surg.* 77:803, 1979.
10. Follette, D.M., Mulder, D.G., Maloney, J.V., and Buckberg, G.D.: Advantages of Blood Cardioplegia Over Continuous Coronary Perfusion or Intermittent Ischemia: Experimental and Clinical Study. *J. Thorac. Cardiovasc. Surg.* 76:604, 1978.
11. Conti, V.R., Bertranau, E.G., Blackstone, E.H., Kirklin, J.W, and Digerness, S.B.: Cold Cardioplegia Versus Hypothermia for Myocardial Protection: Randomized Clinical Study. *J. Thorac. Cardiovasc. Surg.* 76:577, 1978.
12. Becker, H., Vinten-Johansen, J., Buckberg, G.D., Follette, D.M., and Robertson, J.M.: Critical Importance of Ensuring Cardioplegic Delivery with Coronary Stenoses. *J. Thorac. Cardiovasc. Surg.* 81:507, 1981.
13. Schwartz, A.B.: Potassium Related Cardiac Arrhythmias and their Treatment. *Angiology* 29:194, 1978.
14. Urschel, H.C., Greenberg, T.J.: Differential Hypothermic Cardioplegia. *Surg. Forum* 10:507, 1960.
15. Holland, C.E. Jr., Olsen, R.E.: Prevention of Hypothermia by Prardoxical Calcium Necrosis in Cardiac Muscle. *J. Mol. Cell. Cardiol.* 7:917, 1975.
16. Brown, A.H., Braimbridge, M.V., and Darraeott, S.: An Experimental Evaluation of Continuous Normothermic, Intermittent Hypothermic and Hypothermic and Intermittent Normothermic Coronary Perfusion. *Thorax* 29:38, 1974.
17. Follette, D.W., Steed, D.L. and Foglia, R.P.: Reduction of Post Ischemic Myocardial Damage by Maintaining Arrest During Initial Reperfusion. *Surg. Forum* 28:281, 1977.
18. Ellis, R.J., Manguano, D.T., Van Dyke, D.C., and Ebert, P.A.: Protection of Myocardial Function not Enhanced by High Concentrations of Potassium During Cardioplegic Arrest. *J. Thorac. Cardiovasc. Surg.* 78:698, 1979.
19. Engleman, R.M., Auvil, J., O'Donoghue, M.J., and Levitsky, S.: The Significance of Multidose Cardioplegia and Hypothermia in Myocardial Preservation During Ischemic Arrest. *J. Thorac. Cardiovasc. Surg.* 75:555, 1978.
20. Grover, F.L., Fewel, J.G., Ghidoni, J.J., Bennett, E.V., and Trinkle, J.K.: Comparison of Roller Pump Versus Pressurized Bag Administration of Potassium Cardioplegic Solution. *Ann Thorac. Surg.* 34:278, 1982.
21. Engleman, R.M., O'Donoghue, M.J., and Auvil, J.: Techniques for Myocardial Preservation During Ischemic Arrest. *Surg. Forum* 28:229, 1977.

22. Rosenfeldt, F.L., and Watson, D.A.: III. Local Cardiac Hypothermia; Experimental Comparison of Shumway's Technique and Perfusion Cooling. *Ann. Thorac. Surg.* 27:17, 1979.
23. Chiu, R.C.J., Blundell, P.E., Scott, H.J., and Cain, S.: The Importance of Monitoring Intramyocardial Temperature During Hypothermic Myocardial Protection. *Ann. Thorac. Surg.* 28:317, 1979.
24. Hearse, D.J., Stewart, D.A., Braimbridge, M.V.: Cellular Protection During Myocardial Ischemia; The Development and Characterization of a Procedure for the Induction of Reversible Ischemic Arrest. *Circulation* 54:193, 1976.
25. Harlan, B.J., Ross, D., Macmanus, Q., Knight, R., Luber, J., and Starr, A.: Cardioplegic Solutions for Myocardial Preservation: Analysis of Hypothermic Arrest, Potassium Arrest and Procaine Arrest. *Circulation* 58:114, 1978.
26. Bretschneider, H.J., Hubner, K.D., Lohr, B., Nordbeck, H. and Spieckermann, P.G.: Myocardial Resistance and Tolerance to Ischemia. *J. Thorac. Cardiovasc. Surg.* 16:241, 1975.
27. Fisk, R.L., Guilbeau, E.J., Edgar, S.J., Switzer, A.J., and Moore, L.K.: Delayed Recovery Following Hypothermic Arrest in Rabbit Myocardium. *Adv. Exp. Med. Biol.* 159:211, 1983.
28. Hilton, C.J., Teubl, W., Acker, M., Levinson, H.J., Millard, R.V., Riddle, R., and McEnany, M.T.: Inadequate Cardioplegic Protection with Obstructed Coronary Arteries. *Ann. Thorac. Surg.* 28:323, 1979.
29. Ekroth, R., Berggren, H., Sudow, G., Wojciechowski, J., Zackrisson, BoF., and William-Olsson, G.: Thermographic Demonstration of Uneven Myocardial Cooling in Patients with Coronary Lesions. *Ann. Thorac. Surg.* 29:341, 1980.
30. Rosenfeldt, F.L., and Arnold, M.: Absence of Myocardial Damage during Profound Local Cardiac Hypothermia. In *Cardioplegia: The First Quarter Century. An International Symposium*, London, Royal Society, June 1980. Proceedings p. 158.
31. Hartz, R.S., and Michaelis, L.L.: Intraoperative Protection of the Myocardium. In *Perioperative Cardiac Dysfunction Volume III, Cardiothoracic Surgery Series*. Utley, J.R. (ed), Baltimore: Williams and Wilkins, 1985 p. 97.