R-R interval of 480 ms. At an upper rate setting of 150 beats per minute, the AV delay will maintain an R-R interval of 400 ms. This mechanism is clearly illustrated in Figure 2. When the upper rate was set at 150, the AV delay remained at its original setting of 175 ms as the ELT cycle length was 400 ms. (The calculated retrograde conduction time was 240 ms, Fig 1). At this cycle length, retrograde conduction was blocked in this patient after only one conducted retrograde beat (second degree retrograde block). VA conduction was refractory to a second retrograde beat at the rate of 150 beats per minute (400 ms interval) and the ELT terminated after one beat. When the upper rate limit was set at 125 beats per minute, the AV delay automatically extended to 240 ms during the ELT to maintain an R-R interval of 480 ms. At this cycle length, VA conduction successfully transmitted a few consecutive (in this case 7) retrograde beats until it became fatigued and block occurred (retrograde block). When the pacemaker was programmed to an upper rate of 100 beats per minute, the AV delay extended to 300 ms during the ELT to maintain a cycle length of 600 ms. At such a prolonged AV delay, VA conduction recovered and was capable of stimulating the atrium following each paced QRS complex (1:1 retrograde conduction), thus maintaining the tachycardia. This case illustrates the mechanism by which increasing the upper-rate setting of a pacemaker in which AV delay varies, may, in some patients, prevent or restrain the ELT.

CONCLUSION

In a dual chamber pacemaker using the Wenckebach mode to limit the upper rate, the prolongation of the AV delay at high rates may permit recovery of the VA conduction pathway. As the AV delay prolongs maximally at the lowest upper rate limit, this setting is more likely to sustain an endless loop tachycardia. Setting a higher upper rate limit permits a shorter AV delay, may block conduction pathway recovery and paradoxically limit or eliminate a tachycardia based on retrograde conduction.

REFERENCES

1 Tolentino AO, Javier RP, Byrd C, Samet P. Pacer-induced tachycardia associated with an atrial synchronous ventricular inhibited (ASVIP) pulse generator. Pace 1982; 5:251-58
4 Furman S, Fisher JD. Endless loop tachycardia in an AV universal (DDD) pacemaker. Pace 1982; 5:486
5 Hayes DL, Furman S. Atroventricular and ventriculio-atrial conduction times in patients undergoing pacemaker implant. Pace 1983; 6:38

Use of Profound Hypothermia and Circulatory Arrest for the Calcified Aorta*

Israel J. Jacobowitz, M.D.; Daniel M. Rose, M.D.; Ketan Shroeder, M.D.; and Joseph N. Cunningham, Jr., M.D., F.C.C.P.

*From the Department of Surgery, Maimonides Medical Center, Brooklyn. Reprint requests: Dr. Jacobowitz, 4902 Tenth Avenue, Brooklyn 11219

This case report discusses one method of dealing with the extensively calcified aorta in patients undergoing open heart surgery. Profound hypothermia and circulatory arrest was used in a patient undergoing aortic valve replacement with severe calcification of the ascending aorta and transverse arch. This patient recovered from surgery and was discharged from the hospital with no neurologic deficits.

The severely calcified aorta presents a serious problem when crossclamping or cannulation of the ascending aorta is carried out during cardiac surgery.12 Maneuvers such as isolation of innominate and carotid arteries and temporary occlusion of these vessels, prior to aortic crossclamping has, in our experience, decreased the incidence of serious neurologic complications secondary to cerebral embolism of either calcium or atheromatous debris following crossclamping of the diseased calcified aorta.4 This report describes a patient with severe aortic stenosis in whom the entire ascending aorta, transverse arch, innominate and carotid arteries were totally calcified. Use of profound hypothermia with total circulatory arrest is herein described as a valuable adjunct for valve replacement in such complex patients.

CASE REPORT

A 71-year-old man presented with a one-year history of angina, exertional dyspnea and syncope. The ECG revealed left ventricular hypertrophy, and the chest x-ray film demonstrated an enlarged left ventricle with heavy calcification in the aortic valve and ascending and transverse aorta. Cardiac catheterization revealed severe aortic stenosis with normal coronary arteries, as well as extensive calcification of the ascending aorta and arch vessels. The patient was referred for surgery.

At operation, cannulation of the ascending aorta and aortic crossclamping were not safely possible because of calcification of the entire ascending aorta, aortic arch, and great vessels. A decision was made to cannulate the femoral artery and right atrium, and to employ profound hypothermia and circulatory arrest during replacement of the stenotic aortic valve. The head was packed in ice and the esophageal temperature constantly monitored. Bypass was initiated at a flow rate of 2.5 L/min/m² and systemic blood temperatures lowered to 10°C over a 45-minute period. Cardiac fibrillation occurred soon after cooling began and a left ventricular vent was inserted for cardiac decompression. When esophageal temperature was 13°C and rectal temperature was less than 20°C, the head was lowered 20° below the horizontal, and circulatory arrest was instituted. Snare was placed around the left carotid and innominate arteries in soft noncalcified areas about 1.5 cm beyond their origin from the transverse arch. An aortotomy was made and valve replacement carried out in the routine fashion.

Thirty minutes prior to total circulatory arrest, pre-treatment with intravenously administered sodium thiopental (2 g) and methylprednisolone (2 g) was achieved. Myocardial protection during valve replacement was carried out utilizing our previously described technique of cold blood cardioplegia* with direct instillation of the arrest solution into the right and left coronary ostia. Following valve replacement and aortotomy closure, systemic flow was gradually increased from 500 ml to 4 L/min over a two-three minute period. During this time, blood was allowed to fill the transverse and ascending aorta and origin of the innominate and carotid arteries. Venting of air through the aortotomy suture line was achieved with the patient in reverse Trendelenburg position and snare around the innominate and left carotid arteries subsequently released. Systemic rewarming to 37°C was carried out, the heart spontaneously defibrillated, and bypass was terminated uneventfully. Total pump time was
192 minutes. Total circulatory arrest time was 56 minutes.
Postoperation, the patient recovered from anesthesia within eight-
nine hours and was evaluated neurologically. No evidence of focal
neurologic signs or symptoms were noted, and the patient’s neu-
rologic status remained completely normal throughout his hospital

**Discussion**

Dealing with the calcified aorta can certainly be problematic.
A number of approaches have been used successfully to
perform open cardiac procedures under this circumstance.
Mills has used systemic and topical hypothermia with
ventricular fibrillation and femoral artery cannulation for
performance of bypass grafts in patients with calcified aortas.
The internal mammary artery is generally used as the conduit,
and when saphenous vein grafts are required, they are
anastomosed to the innominate artery or in an end-to-
side fashion to the internal mammary artery itself.

Our usual method of addressing the problem of the
calculated aorta involves encircling the innominate and left
carotid artery and clamping these vessels each time the aorta
is manipulated (cannulation, crossclamping and unclamping).
The clamps are left in position for approximately 90
seconds and are applied and released at flow rates of 500 ml/
minute. These maneuvers have successfully prevented neu-
rologic injury.

Profound hypothermia and circulatory arrest as described
in this report have been reserved for situations where
calcification is so extensive that clamping of the carotid and
innominate arteries is unsafe. Based on our previous experi-
ence and that of others with this methodology in arch
aneurysm replacement, we extended its use to the situation
presented here. Profound hypothermia and circulatory arrest
have proved to be efficacious methods for preservation of
central nervous system function where it is either necessary
to occlude the carotid vessels or for repair of complex
congenital heart defects.

A satisfactory result was obtained in this patient using an
alternative surgical approach. Particular attention was
directed towards: 1) maintaining a circulatory arrest time of
less than 60 minutes at 20ºC rectal temperature, and 2)
prevention of cerebral air emboli by placing the patient's
head in reverse Trendelenburg position on declamping; and 3)
pertreatment with methylprednisolone (Solu-Medrol),
thiopental (Pentothal), and propranolol (Inderal) as ad-
juncts to protection of the brain and heart.

Utilization of these guidelines along with employment of
total circulatory arrest and profound systemic hypothermia
may permit an alternative approach to safe valve replacement
or coronary bypass without crossclamping in even the most
calcified aorta.

**References**

1 Callaghan JC, Despres JP, Benvenuto R. A study of the causes of
60 deaths following total cardiopulmonary bypass. J Thorac
Cardiovasc Surg 1961; 42:489
2 Magner JB. Complications of aortic cannulation for open heart
surgery. Thorax 1971; 26:172
3 Parker R. Aortic cannulation. Thorax 1969; 24:742
4 Landmore R, Spencer FC, Colvin S, Culliford A, Trehan N,
Cartier P, et al. Management of the calcified aorta during
84:455
5 Cunningham JN, Adams PX, Knopp EA, Baumann FG, Snively
SL, Gross RI, et al. Preservation of ATP, ultrastructure, and
ventricular function after aortic crossclamping and reperfusion.
J Thorac Cardiovasc Surg 1979; 78:708
6 Catinella FP, Cunningham JN, Sturgaram RK, Nathan IM,
7 Catinella FP, Nathan IM, Paone G, Adams PX, Cunningham JN,
et al. An on-line system for delivery of blood cardiopedia. J
Cardiovasc Surg 1982; 23:252
8 Mills NL. Physiologic and technical aspects of internal mammary
artery and coronary artery bypass grafts. In: Cohn LH (ed),
Modern technics in surgery: Cardiac/thoracic surgery, (VII).
9 Rittenhouse EA, Mohri H, Dillard DH, Merendino KA. Deep
17:63
10 Connolly JE, Roy A, Guernsey JM, Stemmer EA. Bloodless
surgery by means of profound hypothermia and circulatory
11 Subramanian S, Wagner H, Vlad P, Lambert EC. Surface-
induced deep hypothermia in cardiac surgery. J Pediatr Surg
1971; 6:612
12 Dumanian AV, Hoehsema TD, Sautsch DR, Greenwald JH,
Frakin CJ. Profound hypothermia and circulatory arrest in the
surgical treatment of traumatic aneurysm of the thoracic aorta.
J Thorac Cardiovasc Surg 1970; 59:541
13 Michenfelder JD. Brain hypoxia: Current status of experimental

**Spontaneous Regression of Lung Metastases of Adenoid Cystic
Carcinoma**

B. Grillet, M.D.; J. Demedts, M.D., F.C.C.P.†; J. Roelens, M.D.;‡
P. Goddeeris, M.D., A. Fossom, M.D. §

Two patients with spontaneous regression of histologically
confirmed lung metastases from a classic cribriform ade-
noi cystic carcinoma are presented. The first case was
moribund when multiple small lung metastases were de-
tected, but after a very strict diet, he presented progressive
improvement in his general condition and regression of the
metastases. In the second case, three large lung metastases
and a subcutaneous metastasis regressed after several local
recurrences of the primary tumor had been removed.

Regression of primary tumors has been reported on rare
occasions; carcinoma of the kidney, neuroblastoma, malignant
melanoma and choriocarcinoma are those reported. Immunologic action, elimination of carcinogens,
hormones, trauma, irradiation, drugs, diet and meditation
have been mentioned as possible etiologic factors.

Regression of metastases has been described both with and

*From the Catholic University of Leuven, Leuven, Belgium.
†Department of Internal Medicine.
‡Department of Pathology.
§Department of Dentistry, Oral Medicine and Maxillo-facial Sur-
gery.
Reprint requests: Dr. Demedts, Academisch Ziekenhuis, Wellingroer-
d 1, B-3041 Pellenberg, Belgium.