Outcomes after thoracoabdominal aortic aneurysm repair with hypothermic circulatory arrest

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Objective: Recent advances in endovascular surgery have put into question the role of open operative treatment of thoracoabdominal aortic aneurysms. In this context we evaluated our experience with thoracoabdominal aortic aneurysm repair using cardiopulmonary bypass and hypothermic circulatory arrest.

Methods: From January 1986 to December 2008, 218 patients (mean age, 63 ± 14 years) underwent thoracoabdominal aortic aneurysm repair with cardiopulmonary bypass and hypothermic circulatory arrest. The degree of repair was as follows: Crawford extent I, 57 (26%) patients; Crawford extent II, 91 (41%) patients; and Crawford extent III, 70 (32%) patients. Degenerative aneurysms were present in 160 (73%) patients. Eighteen (8%) patients underwent emergency operations.

Results: The mean durations of cardiopulmonary bypass and hypothermic circulatory arrest were 160 ± 44 and 31 ± 12 minutes, respectively. Stroke occurred in 8 (3.7%) patients, and spinal cord ischemic injury occurred in 10 (4.6%) patients (8 with paraplegia and 2 with paraparesis). Temporary dialysis for new-onset renal failure was required in 3.6% of hospital survivors. Thirty-day and 1-year mortality rates were 7.3% and 24.5%, respectively. After emergency operations, the 30-day mortality rate was 33.3% compared with 5.0% after elective operations ($P = .001$). Five- and 10-year survivals were 55% and 23%, respectively. Twenty-five patients required reoperation on the graft or contiguous aorta at a mean of 5 ± 3 years after the initial procedure. Five- and 10-year rates of freedom from reoperation were 87% and 60%, respectively.

Conclusions: Cardiopulmonary bypass with hypothermic circulatory arrest can be safely used for thoracoabdominal aortic aneurysm repair, providing excellent protection against end-organ injury. Early mortality and morbidity rates do not exceed those reported for endovascular repair, with particularly favorable outcomes among patients undergoing elective operations. (J Thorac Cardiovasc Surg 2011;141:953-60)

Several approaches exist for the open surgical repair of thoracoabdominal aortic aneurysms (TAAAs), including the clamp-and-sew technique and the use of distal perfusion either with partial (left heart) or total cardiopulmonary bypass (CPB). These strategies can be supplemented with epidural cooling, cerebrospinal fluid (CSF) drainage, renal and visceral perfusion, and the monitoring of motor and somatosensory evoked potentials to prevent visceral, renal, and spinal cord ischemic injury (SCII).[1-5]

CPB with intervals of hypothermic circulatory arrest (HCA) is a widely used technique for the protection of the central nervous system during complex cardiac and proximal thoracic aortic procedures. It has been used less frequently for repair of TAAAs. Its advantages over other techniques in this setting include minimal aortic dissection and the elimination of the need for proximal and sequential aortic clamping. It also affords access to the distal aortic arch, a bloodless field, return of shed blood into the perfusion circuit, and reduced potential for atheromatous embolization, which can result from aortic clamping. HCA provides protection of the central nervous system, heart, and abdominal organs without the need for monitoring of evoked potentials or separate perfusion of the renal and visceral arteries.[6,7]

Endovascular stent graft technology has evolved since its first application for the exclusion of aneurysms involving the abdominal and descending thoracic aortas. Recent advances include the use of hybrid debranching strategies and multibranched stent grafts for TAAA repair. Small series have confirmed the feasibility of these novel endovascular approaches to TAAA repair,[8-16] with cited advantages, including the avoidance of a thoracoabdominal incision, less postoperative pain, limited pulmonary complications, and more rapid recoveries.[8,17,18] Advocates for these techniques have now brought into question the current role of conventional open surgical repair. Nevertheless, the risks of death and SCII persist with these new endovascular strategies,[8-16] as well as complications and concerns regarding the durability of TAAA stent graft repair.[17] In this context we reviewed our experience with open TAAA repair using CPB and HCA.

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Abbreviations and Acronyms

CPB = cardiopulmonary bypass
CI = confidence interval
CSF = cerebrospinal fluid
HCA = hypothermic circulatory arrest
OR = odds ratio
SCII = spinal cord ischemic injury
TAAA = thoracoabdominal aortic aneurysm

MATERIALS AND METHODS

Patients' Characteristics

Between January 1986 and December 2008, 218 patients underwent TAAA repair with CPB and HCA. No other operative technique was used during this interval. The clinical characteristics of the patients are shown in Table 1. The mean age at the time of the operation was 63 ± 14 years (range, 15–86 years), and 110 (50%) patients were female. Twenty-seven (12.4%) patients had clinical manifestations of Marfan syndrome. One hundred twelve (51%) patients had symptoms associated with their aortic disease. The remaining patients had aneurysms that were more than twice the size of adjacent normal aorta or had evidence of progressive aortic enlargement.

Degenerative aneurysms were present in 160 (73%) patients. Fifty-two (24%) patients had chronic type B dissections with aneurysms, and 3 (1.4%) patients presented with complicated acute type B dissections. Eighteen (8.3%) patients underwent emergency operations for rupture or acute dissection. Two (0.9%) patients had previous TAAA repair. The extent of the aortic disease requiring replacement is listed in Table 2. During the time period of the study, patients with extent IV TAAA were treated with techniques that differed from those described herein and were therefore not included. The study was reviewed by the Institutional Review Board of the Missouri Baptist Medical Center and was exempted from board approval.

Operative Technique

Our general technique has been previously described.[6] With increasing experience, several modifications have been introduced. Since September 2004, CSF drainage has been used routinely, if technically feasible (55 patients). After the insertion of monitoring catheters, the induction of anesthesia, and the placement of a double-lumen endotracheal tube, the patient is positioned in a right lateral decubitus position with the hips turned to the left at a 45° angle. A standard posterolateral thoracotomy incision is made either through the fifth or sixth intercostal space. If necessary, the incision is extended into the abdomen across the costochondral junction, and the diaphragm is incised circumferentially. Simultaneously, the left common femoral artery and vein are isolated through an oblique incision in the skin crease of the groin. Heparin is administered to achieve an activated clotting time of more than 400 seconds (3 mg/kg). A long cannula (28F–34F) is inserted through the left common femoral vein, and the tip is positioned in the right atrium under transesophageal echocardiographic guidance. The femoral artery is cannulated with an 18F to 22F short cannula. CPB is established, and cooling is initiated (target temperature, 15°C–17°C). A catheter is preferentially placed through the left inferior pulmonary vein or through the apex of the left ventricle for venting of the left heart.

This technique of femoral artery perfusion might lead to the retrograde embolization of thrombus or atheroma into the cerebral circulation. Therefore in 1997, we modified our cannulation technique for the 82 subsequently treated patients with atherosclerotic disease and degenerative aneurysms. In addition to standard femoral artery cannulation, a dispersion arterial cannula (Edwards Lifesciences, Inc, Midvale, Utah) is inserted directly into the descending thoracic aorta at a noncalcified, thrombus-free site identified by means of epiaortic ultrasonographic analysis. The aortic cannula and the femoral artery cannula are then connected to separate arterial lines from the pump oxygenator. In these patients CPB is established by using the arterial line attached to the aortic cannula, and the femoral line is clamped. Alternatively, a small left axillary incision can be performed, and an 8-mm collagen-impregnated graft anastomosed to the left axillary artery can be used for proximal perfusion. The femoral arterial line remains clamped while the patient is cooled during CPB. In all other cases, particularly in the setting of aortic dissection, the femoral artery is used to establish CPB.

During the period of cooling, methylprednisolone (7 mg/kg) and thiopental (10–15 mg/kg) are administered intravenously, and the head is packed in ice. Cardioplegia is not induced, and somatosensory and motor evoked potentials are not monitored. When electroencephalographic silence is achieved and the nasopharyngeal temperature is 22°C or less, circulatory arrest is established. The diseased segment of the aorta is incised, and if indicated, the incision is extended into the aortic arch. The left phrenic, left vagus, and left recurrent nerves are identified and protected. A collagen-impregnated, woven polyester aortic graft (26–32 mm) containing a 10-mm side arm (Hemashield Platinum single branch graft; MAQUET Cardiovascular LLC, San Jose, Calif) is sutured end-to-end to the transected descending thoracic aorta or aortic arch with a continuous 3–0 or 4–0 polypropylene suture buttressed with a strip of felt. As this suture line is completed, cold blood (10°C–12°C) from the pump oxygenator is infused retrogradely into the venous cannula to assist in the evacuation of air and debris from the upper circulation and from the graft (flow rate, up to 800 mL/min; maximum central venous pressure, 20 mm Hg). The side arm of the graft is attached to the proximal arterial line from the pump oxygenator. After evacuation of air, a clamp is placed on the graft just distal to the side arm, and flow to the upper body is re-established at a rate of 15 to 20 $mL \cdot kg^{-1} \cdot min^{-1}$ and a temperature of 20°C to 22°C (low-flow hypothermic bypass). Patent intercostal and lumbar arteries below the level of the sixth or seventh intercostal space are preserved when technically feasible by using a full-thickness cuff of aorta that is sutured to an opening in the graft. The proximal clamp is subsequently repositioned on the graft below the anastomosis between the graft and the intercostal patch to permit perfusion of the implanted arteries. Intercostal arteries were reimplanted in 122 (56.0%) patients.

When possible, a clamp is placed on the abdominal aorta below the level of the renal arteries to permit hypothermic perfusion of the lower body by using the femoral artery cannula (33 patients). Alternatively, the distal aorta (or a previously placed infrarenal aortic graft) is occluded with an intraluminal balloon-tipped catheter, and flow to the lower body is established. The distal thoracic and upper abdominal aorta is opened, and the orifices of the celiac, superior mesenteric, and left and right renal arteries are identified. If the aortic disease extends to the level of the iliac arteries, a bifurcation graft (Hemashield Platinum bifurcation graft, MAQUET Cardiovascular LLC) is implanted, with each common iliac artery anastomosis performed with a 5–0 polypropylene suture (19 patients).

Before 2003, the visceral and renal arteries were attached to the aortic graft as a Carrel patch by using a full-thickness cuff of native aortic tissue surrounding the orifices of these arteries. In the 52 most recent patients, the visceral and renal arteries were separately reimplanted with a collagen-impregnated, woven polyester branched aortic graft (Hemashield Platinum TAAA graft, MAQUET Cardiovascular LLC).[19] With this technique, these arteries are detached from the aorta with a small cuff of aortic tissue. If the origin of a vessel is severely stenotic, the vessel is transected beyond the stenosis. The body of the aortic graft is positioned so that the 3 adjacent branches lie opposite the origins of the celiac, superior mesenteric, and right renal arteries. The distal end of the aortic branched graft is cut to the appropriate length and sutured to the infrarenal aorta (or previously placed infrarenal aortic graft) with a continuous 3–0 or 4–0 polypropylene suture buttressed with a strip of felt. As the distal suture line is being completed, the clamp or balloon on the distal aorta is released to evacuate air and debris by using femoral arterial perfusion.

TABLE 1. Preoperative patients' characteristics

Characteristics	No. (%) of patients (n = 218)
Comorbidity	
Marfan syndrome	27 (12.4)
Hypertension	167 (76.6)
History of smoking	137 (62.8)
Hyperlipidemia	94 (43.1)
Chronic obstructive pulmonary disease	89 (40.8)
Coronary artery disease	87 (39.9)
Peripheral vascular disease	64 (29.4)
Creatinine ≥1.5 mg/dL	30 (13.8)
Previous transient ischemic attack or stroke	32 (14.7)
Diabetes mellitus	13 (6.0)
Previous operations	
Coronary artery bypass graft surgery or percutaneous coronary intervention	72 (33.0)
Aortic valve procedure	60 (27.5)
Ascending aortic repair	75 (34.4)
Aortic arch repair	40 (18.3)
Descending thoracic aortic repair	5 (2.3)
Thoracoabdominal aortic repair	2 (0.9)
Abdominal aortic aneurysm repair	38 (17.4)
Cause of aortic disease	
Degenerative aneurysm	160 (73.4)
Chronic type B dissection	52 (23.9)
Acute type B dissection	3 (1.4)
Other	3 (1.4)

A clamp is placed proximal to the distal suture line, and flow to the lower body is re-established. Thirty-five percent of the total arterial flow is directed through the proximal arterial line and 65% through the distal line. The temperature of the perfusate is adjusted to maintain the nasopharyngeal temperature between 20°C and 22°C, and total flow is maintained between 0.75 and 1.5 L $\cdot$ min^{-1} $\cdot$ m^{-2}. During this period of hypothermic low flow, the anastomoses between the branches of the graft and the visceral and renal arteries are completed by using 5–0 or 6–0 polypropylene sutures. Typically, the most distal branch is anastomosed to the right renal artery, followed by the middle limb to the superior mesenteric artery and the upper limb to the celiac artery. The left renal artery is either sewn to the fourth (perpendicular) branch or attached end-to-end to a separate 6- to 10-mm interposition graft. Direct perfusion of the intercostal, lumbar, renal, or visceral arteries is not used. After each anastomosis is completed, the distal clamp on the aortic graft is positioned more proximally to enable perfusion of each reimplanted artery. Rewarming is initiated after establishing perfusion to both the implanted intercostal and renal arteries. The final anastomosis between the proximal aortic graft (with the 10-mm side arm) and the distal branched aortic graft is performed with a 4–0 polypropylene suture. Air is evacuated from the graft through multiple puncture sites created with an 18-gauge needle. Flow from the femoral arterial line is discontinued, the proximal clamp is removed, and full antegrade flow is established from the proximal arterial line. Spontaneous defibrillation usually occurs when the nasopharyngeal temperature reaches 26°C to 28°C. When the bladder temperature reaches 35°C, the pulmonary vein venting catheter is removed, and CPB is discontinued.

TABLE 2. Extent of aortic repair

Crawford extent of repair	No. (%) of patients
Type I	57 (26.1)
Type II	91 (41.7)
Type III	70 (32.1)
Total	218

TABLE 3. Cardiopulmonary perfusion data

	Mean ± SD	Range
Time (min)		
Cardiopulmonary bypass	160.4 ± 43.9	62–296
Cooling	35.1 ± 11.0	18–75
Circulatory arrest	31.2 ± 11.9	8–76
Low-flow hypothermic bypass*	52.9 ± 27.9	8–160
Hypothermic ventricular fibrillation	104.3 ± 32.8	35–217
Spinal cord ischemia	65.3 ± 23.2	11–138
Left renal ischemia	94.0 ± 39.6	33–202
Right renal ischemia	81.6 ± 38.6	12–200
Celiac ischemia	102.5 ± 41.4	39–215
SMA ischemia	92.4 ± 41.6	31–230
Rewarming	70.9 ± 19.3	25–150
Temperature (°C)		
Lowest nasopharyngeal	15.3 ± 2.4	11–24
Lowest bladder/rectal	19.6 ± 3.0	12–28

SD, Standard deviation; *SMA*, superior mesenteric artery. *Two hundred one patients.

ACD

A membrane oxygenator (Optima XP; Cobe Cardiovascular, Arvada, Colo) was used in all cases. The mean durations of cooling, circulatory arrest, low-flow hypothermic bypass, hypothermic ventricular fibrillation, rewarming, and CPB are shown in Table 3. The mean duration of HCA was 31.2 ± 11.9 minutes. A period of hypothermic low flow to the upper and lower body was used in 201 patients (mean duration, 52.9 ± 27.9 minutes) after the interval of circulatory arrest.

Follow-up

After hospital discharge, patients are evaluated at 1 and 6 months postoperatively and subsequently at 12-month intervals. Computed tomographic scans are obtained before discharge, at 6 months, and yearly thereafter. Follow-up was 100% complete. Mean duration of follow-up was 3.3 years (range, 1 month to 14.9 years).

Statistical Analyses

Data were analyzed in Intercooled Stata 9.2 software (StataCorp, College Station, Tex). Standard descriptive statistical analyses were used. Continuous data are presented as a mean ± standard deviation and were compared between groups by using unpaired 2-sided Student *t* tests. Categorical data are presented as proportions and were compared between groups by using the Fisher exact test. Nonparametric estimates of freedom from all-cause death and reoperation were determined by using the Kaplan–Meier method and are reported as means. A composite outcome was developed that incorporated the adverse perioperative events of 30-day mortality, SCII, stroke, and the need for dialysis. Predictors of adverse perioperative events were determined with multivariate logistic regression. Multivariate models were developed by incorporating variables that had a *P* value of .20 or less on univariate testing. Stepwise forward selection and backward elimination techniques were used, with a *P* value of .20 for entry and removal criteria. Odds ratios (ORs) are reported with 95% confidence intervals (CIs).

RESULTS

Early Mortality

The 30-day mortality rate for the entire group was 7.3% (16 patients). Mortality according to the extent of aorta

TABLE 4. Extent of repair and associated morbidity and mortality

Crawford extent of repair	30-d Mortality	Stroke	Spinal cord ischemic injury	New dialysis
Type I (n = 57)	3 (5.3%)	1 (1.8%)	1 (1.8%)	4 (7.0%)
Type II (n = 91)	8 (8.8%)	2 (2.2%)	5 (5.5%)	6 (6.6%)
Type III (n = 70)	5 (7.1%)	5 (7.1%)	4 (5.7%)	5 (7.1%)
Total (n = 218)	16 (7.3%)	8 (3.7%)	10 (4.6%)	15 (6.9%)

replaced is summarized in Table 4. After emergency operations, the 30-day mortality rate was 33.3% (6 patients) compared with 5.0% (10 patients) after elective operations ($P = .001$). There was no association between 30-day mortality and the extent of aortic repair ($P = .72$) or whether the cause of aortic disease was degenerative (mortality, 7.4%) or nondegenerative (mortality, 7.3%) in cause ($P = 1.00$). Four patients undergoing emergency operations and 3 patients undergoing elective operations died in the operating room from myocardial infarction or ventricular failure. Eight other patients died within 30 days of the operation (disseminated intravascular coagulopathy, 2; right ventricular infarction, 1; stroke, 1; multiorgan system failure, 4). One patient died at home 12 days after the operation from an unknown cause. Six patients died during their hospital stay beyond the 30-day time period (respiratory failure, 1; multiorgan system failure, 5). In-hospital mortality was 9.6%. Twenty-four additional patients died after hospital discharge within 6 months of the operation. The 6- and 12-month mortality rates were 21.1% and 24.5%, respectively.

Neurologic Complications

Spinal cord ischemic injury occurred in 10 (4.6%) patients. SCII according to the extent of aorta replaced is summarized in Table 4. After emergency operations, SCII developed in 3 (16.7%) patients compared with 7 (3.5%) patients after elective operations ($P = .04$). Although patients with extent type I aneurysms had the lowest rate of SCII (1.8%), there was no significant relationship between SCII and the extent of aortic repair ($P = .51$). Neither the cause of aortic disease (4.9% vs 3.6%, degenerative vs nondegenerative; $P = 1.00$) nor the use of intercostal reimplantation (4.1% vs 5.2%, reimplantation vs no reimplantation; $P = .75$) had an effect on the rate of SCII. Spinal drains were inserted routinely since 2004. SCII developed in 1.8% (1/55) of patients who received CSF drainage compared with 5.5% (9/163) of those who did not receive CSF drainage ($P = .46$). SCII did not occur in any of the 21 patients who had both CSF drainage and intercostal artery reimplantation.

Seven patients had paraplegia immediately after the operation, all with minimal recovery. One patient had paraparesis immediately postoperatively. His motor strength improved slightly after the insertion of a spinal drain in the postoperative period. Another patient had paraparesis preoperatively at the time of presentation with a rupturing degenerative aneurysm. His lower extremity weakness did not improve with emergency surgery. However, with rehabilitation, he was ambulating with a cane 1 year later. The last patient had delayed paraplegia after an emergency laparotomy for bowel infarction 9 days after TAAA repair. She died from multiorgan failure 21 days later. Within 1 year postoperatively, 7 of the 10 patients with SCII had died.

Eight (3.7%) patients sustained a stroke. The incidence of stroke according to the extent of aorta replaced is summarized in Table 4. Patients with extent III aneurysms had the highest rate of stroke (7.1%). However, the relationship between stroke and the extent of aortic repair was not significant ($P = .27$). The cause of aortic disease also did not significantly affect the rate of stroke (3.7% vs 3.6%, degenerative vs nondegenerative; $P = 1.00$). Among the 163 patients with degenerative atherosclerotic aneurysms, the stroke rate was 6.3% (5/79) with the use of a cannula placed in the descending thoracic aorta compared with 1.2% (1/84) with the sole use of the femoral artery for perfusion ($P = .11$).

Other Morbidity

New-onset renal dysfunction requiring dialysis developed in 15 (6.9%) patients. Eight of these patients had acute renal failure associated with multiorgan dysfunction and died in the perioperative period. Temporary dialysis was required in 3.6% (7/197) of hospital survivors. Intraoperative transfusion of blood products was required in 209 (96%) patients. Average intraoperative transfusion requirements were 8.0 ± 5.3 units (median, 6 units) of packed red blood cells, 6.3 ± 3.8 units (median, 6 units) of fresh frozen plasma, 3.8 ± 2.6 units (median, 3 units) of platelets, and 1.2 ± 3.3 units (median, 0 units) of cryoprecipitate. Ventilatory support was required for a mean of 6 ± 14 days (median, 2 days), and 31% of patients required ventilation for longer than 72 hours. Twenty-five (11%) patients required tracheostomy. The duration of intensive care unit stay was 10 ± 15 days (median, 5 days). Hospital length of stay was 20 ± 17 days (median, 13 days). Injury to the left recurrent laryngeal nerve occurred in 17 (7.8%) patients. Additional morbidity data are listed in Table 5.

The composite outcome (incorporating 30-day mortality, SCII, stroke, and the need for dialysis) occurred in 40 (18.4%) patients. Predictors associated with the composite outcome on univariate analysis included increasing duration of CPB (OR, 1.1 per each additional 10 minutes; 95% CI, 1.0–1.2; $P = .04$), preoperative symptoms (OR, 1.7; 95%

TABLE 5. Postoperative morbidity

Type of morbidity	No. (%) of patients (n = 218)
Exploration for bleeding	19 (8.7)
Low cardiac output (inotropes >48 h)	19 (8.7)
Gastrointestinal bleeding or ischemia	19 (8.7)
Deep vein thrombosis or pulmonary embolus	10 (4.6)
Wound infections	10 (4.6)
Sepsis	10 (4.6)

CI, 0.8–3.5; $P = .16$), coronary artery disease (OR, 4.0; 95% CI, 1.9–8.4; $P < .0001$), peripheral vascular disease (OR, 1.8; 95% CI, 0.9–3.6; $P = .11$), hyperlipidemia (OR, 2.0; 95% CI, 1.0–4.0; $P = .05$), hypertension (OR, 2.4; 95% CI, 0.9–6.5; $P = .08$), preoperative creatinine level (OR, 4.7 per each 1 mg/dL increase in creatinine level; 95% CI, 2.2–10.4; $P < .0001$), emergency operation (OR, 5.5; 95% CI, 2.0–14.8; $P = .001$), male sex (OR, 1.7; 95% CI, 0.8–3.4; $P = .15$), and older age (OR, 1.1 per each additional year; 95% CI, 1.0–1.1; $P = .006$). On multivariate logistic regression, independent predictors of the composite outcome were preoperative creatinine level (OR, 3.5 per each 1 mg/dL increase in creatinine level; 95% CI, 1.5–8.1; $P = .004$), emergency operation (adjusted OR, 5.0; 95% CI, 1.5–16.6; $P = .008$), coronary artery disease (OR, 2.8; 95% CI, 1.1–6.9; $P = .03$), increasing duration of CPB (OR, 1.1 per each additional 10 minutes; 95% CI, 1.0–1.2; $P = .05$), and a trend toward older age (adjusted OR, 1.0 per each additional year; 95% CI, 0.9–1.1; $P = .13$).

Long-Term Follow-up

During the follow-up period that extended to 14 years, there were 62 late deaths. Five- and 10-year survivals were 55% and 22%, respectively (Figure 1). Patients who underwent elective operations had a 5-year survival of 57.6% compared with 31.8% for patients treated on an emergency basis ($P = .008$, Figure 2). Patients with degenerative aneurysms had 5- and 10-year survivals of 52% and 15% compared with 64% and 36% among all other patients ($P = .03$, Figure 3).

Twenty-five patients required reoperation on the graft or contiguous aorta at a mean of 5 ± 3 years after the initial procedure. Five- and 10-year actuarial rates of freedom from reoperation were 87% and 60%, respectively. Indications for reoperation included bleeding from an intercostal patch 5 weeks postoperatively (n = 1); repair of a false aneurysm at the proximal suture line (n = 2); progressive dilatation of the native aortic arch, proximal descending thoracic aorta, or both (n = 4); late aneurysmal dilatation of the intercostal patch (n = 9); late aneurysmal dilatation of the visceral patch (n = 2); concurrent late aneurysmal dilatation of the intercostal and visceral patches (n = 2); progressive dilatation of the distal native abdominal aorta after previous extent type I repair (n = 2); and progressive dilatation of iliac artery, femoral artery, or both aneurysms (n = 3). The remaining patients are free of symptoms and abnormal findings of the contiguous aorta on serial computed tomographic

FIGURE 1. Long-term survival after thoracoabdominal aortic aneurysm repair among the entire cohort.

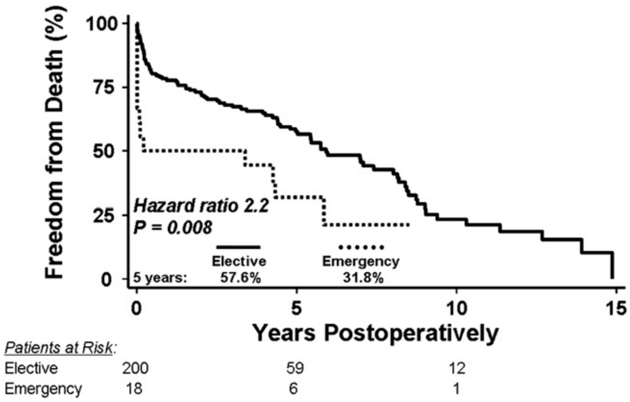

FIGURE 2. Long-term survival after thoracoabdominal aortic aneurysm repair among patients treated electively or on an emergency basis. Long-term survival was significantly better in patients treated electively.

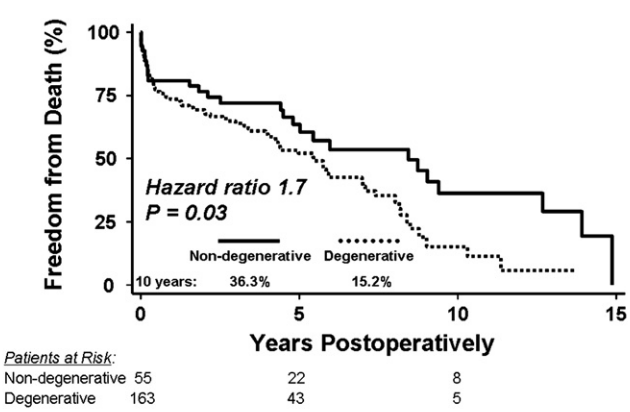

FIGURE 3. Long-term survival after thoracoabdominal aortic aneurysm repair among patients treated for degenerative or nondegenerative aneurysms. Long-term survival was significantly better in patients with nondegenerative aneurysms.

examinations from 1 month to 12 years postoperatively. No open reoperations have been required on the abdominal aorta or visceral branches for those patients who had implantation of a branched aortic graft.

DISCUSSION

Operative repair of TAAAs became feasible through the pioneering work of Svensson, Crawford, and colleagues.[20] Although early reports documented rates of mortality and SCII at 10% to 20%,[20] several techniques have since been developed to decrease the risk of neurologic, visceral, and renal ischemic injury. These include the use of left heart bypass, epidural cooling, CSF drainage, and selective organ perfusion.[1-5] In use for more than 50 years, hypothermic CPB with intervals of circulatory arrest is a safe and effective means for protection of the central nervous system during complex cardiac surgery. Applying this technique during TAAA repair provides a bloodless field and access to the aortic arch and eliminates the need for proximal and sequential aortic clamping and dissection of the aorta. The use of other adjunctive measures, such as separate perfusion of the renal and visceral arteries and epidural cooling, is not required. Furthermore, at hypothermic temperatures, monitoring of evoked potentials becomes unreliable and is no longer necessary. The majority of the shed blood is returned to the perfusion circuit, and excellent organ protection is provided by hypothermia of the central nervous system, heart, and viscera. Through the process of metabolic downregulation, HCA increases ischemic tolerance and allows for an unhurried aortic reconstruction in addition to visualization and reimplantation of the intercostal arteries.[6,7]

The goal of the current study was to present contemporary surgical outcomes using CPB and HCA during TAAA repair. Our results show that, when applied on a routine basis, TAAA repair with HCA is a safe operation. Adverse neurologic events occurred infrequently, with SCII and stroke occurring in 4.6% and 3.7%, respectively, and the 30-day mortality was 7.3%. Patients treated on an emergency basis clearly had worse outcomes, with a 30-day mortality rate of 33.3% and an SCII rate of 16.7%. Independent factors associated with adverse perioperative events in the cohort included the need for emergency surgical intervention, preoperative creatinine level, coronary artery disease, and increasing duration of CPB. We believe our results compare favorably with those currently reported for endovascular repair and confirm that CPB with HCA is a reliable and effective technique for use in TAAA repair.

Routine use of HCA for TAAA repair has been questioned in the past out of concern that it is associated with greater operative risk.[21,22] Morbidity and mortality rates of up to 50% have been reported from centers that have applied HCA selectively for TAAA repair to manage emergency conditions or situations in which the proximal aorta cannot be clamped.[21,22] However, when applied routinely, highly satisfactory results have been achieved. In a series of 39 consecutive patients treated with CPB and HCA for aneurysms of the descending and thoracoabdominal aorta, Soukiasian and associates[23] reported a 30-day mortality rate of 5.1% and an SCII rate of 2.6%. Fehrenbacher and coworkers[7] recently published their experience of TAAA repair using HCA in 110 consecutive patients. The hospital mortality rate was 4.5%, the stroke rate was 4.5%, and the SCII rate was 4.5%. Although concerns have been raised regarding excessive bleeding and respiratory complications with HCA, the results of this series compare favorably with those from centers using alternative surgical techniques.[1-5]

We believe that HCA provides excellent protection against ischemic injury of the viscera, kidneys, and spinal cord during TAAA repair. Since 2004, we have supplemented this operative strategy with the use of CSF drainage, and we have seen the SCII rate decrease to 1.8%. In the experience of Coselli and colleagues,[3] an SCII rate of 3.8% was reported after TAAA repair in 2286 patients treated with the selective use of CSF drainage, left heart bypass, and intercostal artery implantation. Safi and associates[5] reported a significant improvement in the SCII rate (6.8% to 2.4%, $P < .001$) when they applied the adjuncts of distal aortic perfusion and CSF drainage in the repair of descending thoracic aortic aneurysms and TAAAs. Although there is no consensus,[4] the majority of centers with expertise in aortic surgery support the reimplantation of patent intercostal arteries below the T7–T8 level at the time of TAAA repair. The intercostal arteries are believed to play a critical role in the maintenance of spinal cord perfusion, and studies have demonstrated improved SCII rates when they are implanted during TAAA repair.[2,24] Nevertheless, routine intercostal artery reimplantation might lead to a small number of patients ultimately requiring reintervention to manage intercostal patch dilatation.[25] We prefer to preserve the patency of all large intercostal arteries below the T5–T6 level using as small a patch as technically feasible, recognizing the possible risk of late dilatation.

Since its first application for the repair of aneurysms involving the abdominal and descending thoracic aorta, endovascular stent graft technology has evolved dramatically. Recently, hybrid debranching strategies[8-14] and multibranched stent grafts[15,16] have been developed to treat TAAA. Potential advantages of these techniques include a smaller incision, less postoperative pain, limited pulmonary complications, and more rapid recovery.[18] Citing high morbidity and mortality rates reported in surgical series published more than 15 years earlier, advocates for these novel therapies claim that endovascular TAAA repair reduces the risk of SCII and mortality.[8,17,18] In spite of these assertions, the reported series have documented morbidity and mortality rates that do not differ substantially from

TABLE 6. Risks associated with endovascular stent graft repair of thoracoabdominal aortic aneurysms

Authors	No. of patients	30-d Mortality (%)	Spinal cord ischemic injury (%)	Stroke (%)	Renal failure requiring dialysis (%)	6-mo Mortality (%)
Hybrid debranching						
Murphy and coworkers[8]	18	5.6	0	16.7	0	—
Black and coworkers[9]	29	20.7	0	3.4	6.9	—
Quinones-Baldrich and coworkers[10]	20	0	5	0	0	25
Zhou and coworkers[11]	31	3.2	0	0	6.4	3.2
Bockler and coworkers[12]	28	14.3	10.7	0	10.7	25
Siegenthaler and coworkers[13]	21	4.8	4.8	4.8	9.5	14.3
Patel and coworkers[14]	23	17.4	8.7	0	8.7	26.1
Mean	170	10.0 (17/170)	3.5 (6/170)	2.9 (5/170)	6.5 (11/170)	18.7 (23/123)
Multibranched stent grafts						
Chuter and coworkers[15]	22	9.1	13.6	9.1	9.1	13.6
Roselli and coworkwers[16]	73	5.5	2.7	1.4	1.4	15.1
Mean	95	6.3 (6/95)	5.3 (5/95)	3.2 (3/95)	2.8 (3/106)	14.7 (14/95)

those reported in the current study (Table 6). The risks of death and neurologic injury clearly persist with these new endovascular strategies,[8-16] and additional concerns exist regarding costs[8] and durability.[17] By using the strategy of debranching and aortic stent grafting, occlusion rates of grafts placed to the visceral and renal arteries have been as high as 11%, with follow-up generally limited to less than 2 years.[12,14] Moreover, complications, such as endoleak requiring reintervention and the need for long-term dialysis, have not been infrequent.[8-16] Because of differing techniques, potential selection bias, and the absence of a randomized controlled trial, it is impossible to fairly compare the published series of open and endovascular TAAA repair. However, at the present time, we remain unconvinced that stent graft repair appreciably improves the outcomes of patients with TAAAs compared with those after conventional open repair.

Limitations

This study is the largest to date to report outcomes associated with TAAA repair using CPB with HCA. However, the results presented must be interpreted within the context of the study design. Observational in nature, this study involved the retrospective review of prospectively collected data in a referral-based tertiary care center. Surgical techniques evolved during the 20-year time period of the study. Although many patients were referred from afar, every effort was made to obtain imaging surveillance at regular intervals, even if from a distance. Patients' outcomes were documented by means of direct examination or communication with family members or referring physicians.

CONCLUSIONS

In summary, we have documented that CPB with HCA can be safely used for TAAA repair. This technique provides excellent protection against ischemic injury that equals or exceeds that provided by other currently used techniques. Although more invasive than stent graft placement, this open surgical technique yields early mortality and morbidity rates that do not exceed those reported for endovascular repair.

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