ORIGINAL ARTICLE

Systemic Temperature and Paralysis After Thoracoabdominal and Descending Aortic Operations

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Hypothesis: Systemic temperature influences the development of neurologic deficits after aortic surgery.

Design: Retrospective case-comparison study of prospectively collected data.

Setting: Tertiary referral center.

Patients and Interventions: We examined spinal cord injury according to mild passive hypothermia (mean temperature, 36.5°C; n=25), moderate active hypothermia (temperature range, 29°C-32°C; n=76), or profound hypothermia (temperature, <20°C; n=31) for complex repairs in 132 patients. Aortic dissection was present in 67 patients (51%), 41 (31%) had leaks or rupture, 39 (30%) were reoperations on the descending thoracic aorta, and 27 (20%) had concurrent arch and/or ascending thoracic aortic repairs.

Main Outcome Measure: Occurrence of permanent and transient deficits.

Results: Five patients (3.8%) had permanent deficits. One (4.0%) of the 25 patients underwent mild hypothermia, 3 (3.9%) of the 76 patients who underwent moderate hypothermia, and 1 (3.2%) of the 31 patients who underwent profound hypothermia ($P=.70$). Reversible deficits occurred in 7 patients (total 32%) who underwent mild hypothermia, 2 patients (total 6.6%) underwent moderate hypothermia, and 1 (total 6.5%) underwent profound hypothermia ($P=.004$). Six were delayed neurologic deficits. Independent predictors were intercostal ischemic time ($P=.02$), mild hypothermia ($P=.004$), and no cerebrospinal fluid drainage ($P=.05$). The total 30-day survival was 92.4% (122 of 132 patients). The only multivariable predictor of death was acuity of surgery (namely, emergent, urgent, or elective) ($P=.06$).

Conclusions: Moderate or profound hypothermia resulted in fewer transient neurologic deficits. Thus, we recommend active cooling and cerebrospinal fluid drainage for most patients, and profound hypothermia for patients undergoing complex repairs and reoperations.

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EVER SINCE the articles by Barnard and Schrire[1] and Pontius et al,[2] the influence of systemic temperature and methods of protecting the spinal cord from injury have been debated.[3-20] In a previous randomized study of Crawford types I (involves most of the descending thoracic and upper abdominal aorta above the renal arteries) and II (involves most of the descending thoracic aorta and most or all of the abdominal aorta) thoracoabdominal aneurysms,[12] we showed that active cooling with atriofemoral bypass to between 29°C and 32°C significantly reduced the occurrence of postoperative lower limb neurologic deficits. Kouchoukos et al[11] have reported excellent results using profound hypothermia with circulatory arrest for spinal cord protection during descending thoracic and thoracoabdominal operations. Others[6,9] have used deep hypothermia and circulatory arrest on a selective basis for high-risk complex repairs and have not reported good results. The role for profound hypothermia and circulatory arrest and its general application for these repairs are unclear based on the results of these studies. We have examined the influence of systemic temperature on the occurrence of neurologic deficits after thoracoabdominal and descending thoracic aortic surgery in our patients.

See Invited Critique at end of article

METHODS

One hundred thirty-two consecutive patients (78 men [59%] and 54 women [41%]) underwent thoracoabdominal and descending tho-

Table 1. Preoperative Comorbid Disease of 132 Patients

Variable	No. (%) of Patients
Chronic pulmonary disease	73 (55.3)
Aortic dissection	68 (51.5)
Peripheral vascular disease	61 (46.2)
Documented coronary artery disease	56 (42.4)
Ex-smoker	52 (39.4)
Cancer	28 (21.2)
Active smoker	26 (19.7)
Severe renal disease/dialysis	24 (18.2)
Stroke	12 (9.1)
Morbid obesity	12 (9.1)
Marfan syndrome	11 (8.3)
Trauma	9 (6.8)
Diabetes mellitus	7 (5.3)
Coarctation/congenital	6 (4.6)
Paraplegia/paraparesis	4 (3.0)
Polycystic kidney disease	2 (1.5)
Tuberculosis	2 (1.5)
Mycotic	1 (0.8)
Iatrogenic dissection from stent graft	1 (0.8)
Diaphragm hernia	1 (0.8)

racic aortic surgery using mild passive hypothermia (n=25; 11 with atriofemoral bypass), moderate active hypothermia to 29°C to 32°C with atriofemoral bypass and a heat exchanger (n=76), or profound hypothermia with full cardiopulmonary bypass (n=31) between November 9, 1990, and May 15, 2001. Subsequent to 1992, mild passive hypothermia was used less frequently. The selection of profound hypothermia in this series was based on extensive aortic arch involvement, reoperation, or the need for concurrent ascending aortic repair.

The mean (SD) age of the patients was 63 (13.3) years (age range, 27-85 years). Sixty-six patients (50%) underwent thoracoabdominal aortic repairs and 66 patients (50%) had descending thoracic aortic repairs. Preoperatively, aortic dissection was present in 67 cases (51%, 17 were acute); of these 41 patients (31%) had ruptured or had leaks outside the lumen of the aorta. Thirty-nine (30%) of the 132 were reoperations on the descending or the thoracoabdominal aorta. Ascending aorta, aortic arch, or abdominal aortic aneurysm repairs done previously were not counted as reoperations. Twenty-seven (20%) had concurrent aortic arch with or without ascending aortic repairs.

Aorta-related symptoms at presentation prior to surgery were graded 1 to 4 as previously described.[15-17,19] Patients with grade 1 were asymptomatic (9/34 [25%]), patients with grade 2 had occasional chest discomfort or minor signs or symptoms (10/37 [28%]), patients with grade 3 had constant pain (5/25 [19%]), and patients with grade 4 had either rupture, acute dissection, or shock (10/36 [27%]). Acuity of surgery was graded as emergent, namely, immediate surgery in 24 patients (18%); urgent (when the patient was scheduled for the next available operation time slot on the next day) in 34 patients (26%), or elective in 74 patients (56%). In addition, preoperative comorbid disease is summarized in **Table 1**.

The operative techniques have been described previously.[12,15,16,19,20] Briefly, this consisted of the following steps: transection of the proximal aorta whenever feasible and also the distal aorta; the technique we described of segmental sequential repair with perfusion of as much as possible of the intercostal and lumbar arteries while minimizing intercostal ischemic time[12,19,20]; the use of atriofemoral bypass or cardiopulmonary bypass as indicated earlier; the routine use of cerebrospinal fluid (CSF) drainage with intrathecal preservative-free papaverine hydrochloride administration whenever time allowed, except for patients for whom profound hypothermia was used; and reattachment of segmental arteries from T6 to L2 or selective mapping of the intercostal or lumbar arteries supplying the spinal cord with hydrogen.

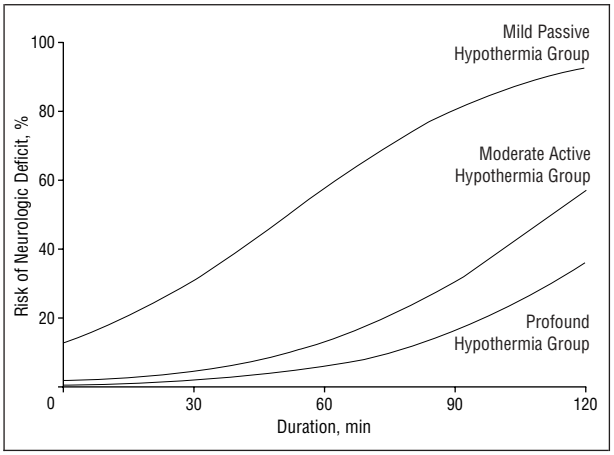
The patients were then divided into the following 3 groups: (1) Patients (n=25) whose temperature was allowed to drift down with mild passive hypothermia and no active cooling (mean [SD] bladder temperature, 34.5°C [1.0°C]). In this series, normothermia was not maintained with a heat exchanger during aortic cross clamping. (2) Those (n=76) who underwent active cooling to moderate hypothermia at 29°C to 32°C (mean bladder temperature, 30.3°C [4.0°C]). (3) Those (n=31) who underwent profound hypothermia with cooling to below 20°C (mean bladder temperature, 18.3°C [4.0°C]). Patients in the moderate active hypothermia group were significantly colder than the mild passive hypothermia group ($P<.001$) and significantly warmer than the profound hypothermia group ($P<.001$). The mean (SD) pump time was 50 [17] minutes for mild hypothermia (the pump was usually used for the proximal and intercostal anastomoses; the distal and visceral were preformed open), 92 [48] minutes for moderate hypothermia, and 166 [53] minutes for profound hypothermia ($P<.001$).

By univariate analysis for any variance, there was no statistically significant difference in the prevalence or distribution of the following variables: sex, age, acuity of surgery (namely, emergent, urgent, or elective), symptom grade, aortic valve regurgitation, blood conservation, peripheral vascular disease, Marfan syndrome (n=11), rupture, reoperation, extent (descending, Crawford type I, II, III [involves the distal descending aorta and most of the abdominal aorta], or IV [involves most or all of the abdominal aorta, including the visceral vessel segment]), number of intercostal arteries reattached or oversewn, 30-day death, stroke, perioperative myocardial infarction, or postoperative day of discharge. Of the profound hypothermia group, 15 had concurrent ascending and arch repairs and 4 had arch repairs only. Two patients who underwent mild passive hypothermia and 6 who underwent moderate active hypothermia had arch repairs. The surgical procedure for 1 patient who underwent mild passive hypothermia and who had a ruptured arch was done without using a pump. This patient also had a hemiascending arch and descending aorta repaired; however, this patient had a stroke.

Data were collected prospectively on a database; postoperative complications were cross-checked against a master database. The database was also cross-checked with data from morbidity and mortality meetings. Continuous variables were analyzed using the *t* test for univariate analysis; categorical data were analyzed using the χ^2 test. Multivariable analysis was done by stepwise logistic regression (SPSS Inc, Chicago, Ill) using the end point of all in-hospital deficits that were immediate, delayed, after CSF drainage catheter withdrawal, permanent, or transient (n=15). Permanent deficits, either immediate or delayed, were defined as those still present at the time of discharge from the hospital or at death. Transient deficits, either immediate or delayed, had resolved by the time of hospital discharge or death.

RESULTS

Permanent neurologic deficits occurred in 5 patients (3.8%). The percentage at different body temperatures was 4.0% (1/25) with mild passive cooling, 3.9% (3/76) with moderate active cooling, and 3.2% (1/31) with profound hypothermia ($P=.70$). A further 7 patients, all of whom had mild cooling, exhibited reversible neurologic deficits but had recovered by the time of hospital



Relationship by logistic regression analysis between intercostal ischemic time and risk of neurologic deficit (P=.003) according to systemic temperature. Profound hypothermia was significantly better than mild passive hypothermia (P=.008) but not moderate active hypothermia (P=.40).

Table 2. Complications

Variable	No. (%) of Patients
Atrial fibrillation*	26 (19.7)
Pulmonary	21 (15.9)
Renal disease	13 (9.5)
Renal dialysis	6 (4.6)
Vocal cord	9 (6.8)
Myocardial infarction	7 (5.3)
Reoperation	7 (5.3)
Stroke	3 (2.2)
Pulmonary embolus	2 (1.5)
Ventricular tachycardia	2 (1.5)
ECMO†	2 (1.5)
Deep vein thrombosis	2 (1.5)
Perforated esophagus	1 (0.7)

Abbreviation: ECMO, extracorporeal membrane oxygenation.
*By hypothermia group, 3 of the 25 patients in the mild passive group (ie, those with a mean temperature of 36.5°C), 16 of the 76 patients in the moderate active group (ie, those with a temperature range between 29°C and 32°C), and 7 of the 31 patients in the profound group (ie, temperature <20°C) had atrial fibrillation.
†Of those who received ECMO, 1 patient survived and 1 patient died.

discharge compared with 2 who underwent moderate active hypothermia and 1 who underwent profound hypothermia. By χ^2 analysis, this was a statistically significant difference in the occurrence of deficits (32%, 6.6%, and 6.5%, respectively, P=.004). Of all 15 deficits, either permanent or transient, 6 (40%) were delayed deficits. The cause and associated hypotension were supraventricular tachycardia; excessive diuresis; respiratory failure and reintubation; pulmonary embolus with hypotension and respiratory failure; delayed embolic shower to the kidneys, liver, gut, and legs; and cardiac tamponade. One patient with replacement of the entire aorta form aortic valve to bifurcation had slightly transient increased weakness but fully recovered. Two delayed deficits were permanent.

The multivariable independent predictors of neurologic deficits were intercostal ischemic time (P=.02), either moderate active or profound hypothermia (P=.004), and CSF drainage with intrathecal preservative-free papaverine administration (P=.05). Active cooling with moderate or profound hypothermia and CSF drainage with intrathecal preservative-free papaverine administration were significantly protective. Interestingly, the extent of the repair according to the Crawford classification of aneurysms, namely, type I, II, III, or IV, or descending thoracic repair did not significantly influence the neurologic deficit rate in this model (P=.10). The **Figure** shows the logistic regression (P=.003) relationship between the intercostal ischemic time and the risk of a neurologic deficit according to systemic temperature. Please note that there was no statistically significant difference between moderate active and profound hypothermia (P=.40).

The total 30-day survival was 92% (122/132). There were no intraoperative deaths. On multivariable analysis the only predictor of death was the acuity of surgery, namely, emergent (immediate) vs urgent (next operating day) vs elective (P=.06). The other complications in this series of patients are given in **Table 2**. Of note, 3 strokes occurred, 1 in each group.

COMMENT

The results of descending and thoracoabdominal aneurysm surgery, both survival and reduced risk of paralysis, have continued to improve.[7-17] Nevertheless, milder forms of paralysis, namely, transient or delayed deficits, are more often noted, although the occurrence of paraplegia is, fortunately, less frequent. There are 3 likely reasons. First, the shorter duration of ischemia as a result of sequential segmental repairs and better protection of the spinal cord during the period of cross clamping has prevented immediate severe injury but resulted in delayed deficits from secondary injury.[12] Second, segmental and intercostal arteries are aggressively reattached to replacement Dacron grafts.[12,17,19] Third, postoperative management (prolonged CSF drainage and induced hypertension)[12] has improved.

Clearly, as expected from previous research,[15,16] distal aortic perfusion was protective,[15,16] but in addition, actively cooling patients with atriofemoral[12,19] or cardiopulmonary[11] bypass offered more protection than mild passive hypothermia when all deficits were included. The logistic regression curves clearly show how active cooling moved the curve to the right, indicating greater protection according to the intercostal ischemia time. The benefits we observed were largely from reducing the occurrence of transient deficits, suggesting the latter deficits are a function of the spinal cord ischemia during a period of comparatively greater metabolism at a higher temperature when compared with the other 2 groups.

Based on the findings from this study and others,[1,2,11,12,16,18,19] reduction of spinal cord temperature is beneficial, although how this is achieved and what is the safest method remain a matter for debate. Certainly, a prolonged period of passive hypothermia prior to clamping works well, has associated good results, and is advocated by some authors.[7-10] Intrathecal spinal cooling[21] was used in the 1960s for traumatic spinal cord injury, and

subsequent studies in pigs by us[2] and in dogs by Berguer et al[22] showed this to be effective, although its general application in initial patient studies has been hampered by the increased CSF pressure and inadequate rate of CSF and effluent drainage from the intrathecal space. Others have advocated epidural cooling.[23] Direct perfusion of the spinal cord by a cold "spinoplegia" has also been effective in animal experiments.[20]

Initially, Svensson used the technique used by Crawford and colleagues[6,15,16] for descending or thoracoabdominal aneurysm repairs; however, despite similar cross-clamp times, the occurrence of neurologic deficits was higher than expected. At the time, review of data also clearly showed that despite distal aortic perfusion with atriofemoral bypass being protective,[15,19] if the patients were kept normothermic during bypass, the risk of a neurologic deficit was increased.[15,16] Concurrently, animal laboratory research[18,20] showed the strong protective effect of spinal cord cooling and segmental sequential reperfusion of the spinal cord. This led, in 1992, to the increasing use of active cooling and segmental sequential repairs by Svensson et al[16,20] and recommendation of the technique. The improved results were subsequently confirmed by others.[24]

In this series we were selective in the use of profound hypothermia in that we used it only for complex repairs when circulatory arrest was necessary. For example, we used hypothermic arrest when either the aortic arch was involved because of aneurysm formation, or dissection (in particular after previous ascending and arch repairs for aortic dissection), or atheroma, or if large segments of the ascending aorta and/or arch needed to be replaced. Clearly, the use of profound hypothermia and circulatory arrest is essential for replacement of the entire aorta or entire thoracic aorta as described previously using both a mediastinal and thoracoabdominal incision.

As paraplegia has receded in prevalence after these operations, probably because of better protection of the spinal cord during ischemia by cooling and sequential reattachment and reperfusion of segmental arteries, attention has focused more sharply on the complications of delayed neurologic deficits. In a series of traumatic ruptures of the aorta repairs reported in 1985, the only deficit we noted was a delayed deficit that occurred after a period of severe hypotension.[6] In a prospective study of types I and II thoracoabdominal aneurysms, including evaluating the problem of delayed deficits, we reported that 32% of the deficits in 98 patients were delayed and occurred between 3 and 22 days after surgery.[13] Postoperative hypotension was most markedly associated with delayed deficits ($P=.006$ univariate analysis; $P=.006$ multivariate analysis), and CSF drainage may have offered some protection (12% vs 40% control, $P=.08$).[13]

Whether profound hypothermia is more protective against delayed deficits, and also all deficits, when compared with moderate active hypothermia, is an interesting question, although we have seen delayed deficits occur after profound hypothermia as noted again recently. The logistic regression curve would suggest there is little difference based on this data set. Nevertheless, it may be that the lower temperatures protect the spinal cord better during ischemia with less risk of secondary biochemical injury, although at the disadvantage of more bleeding, respiratory complications, and possible death and in this series, prolonged intensive care unit stay (7.7 days for profound hypothermia vs 2.9 days for moderate active hypothermia vs 6.1 days for mild passive hypothermia, $P<.05$).

Clearly, the cause of delayed deficits remains to be fully elucidated before it can be prevented. We are aware of 2 animal studies that have examined the problem, one by us[20] and the other by Moore and Hollier.[5] In ours,[20] we found that those animals in which the spinal cord blood supply, as determined by hydrogen mapping, had been severed and, yet, did not develop paralysis, if they were taken back to the laboratory a few days later and hypotension was induced by withdrawing blood, they then had seizurelike activity of the legs and became paralyzed. This effect, however, was reversed by rapid reinfusion of blood and by raising the blood pressure. Thus, during hypotension, the collateral blood supply becomes inadequate and this certainly would parallel closely with human studies where postoperative hypotension of various causes, as in the present study, results in delayed deficits. Hypotension, however, does not explain all delayed deficits. For example, in 1 of our patients, it was related to an embolic shower. Respiratory failure is also a factor. In other patients there is no apparent cause, although we have noted on postoperative angiography it may be related to thrombosis of reattached segmental arteries. Delayed deficits are a consequence of intraaortic stent placement since segmental arteries are occluded by the stents. Other causes are possibly related to the complex biochemical cascade after spinal cord ischemia, such as apoptosis, delayed programmed death, and other secondary biochemical injuries resulting in secondary spinal cord injury.[19] In the experiments by Moore and Hollier in rabbits, intermediate periods of aortic occlusion with milder ischemic results were associated with delayed deficits, suggesting such a biochemical cause. Fortunately, delayed deficits are often less severe and transient and may be reversed by induced hypertension, improved oxygenation, and, sometimes, repeated CSF drainage.

CONCLUSIONS

To validate routine cooling based on these findings, we have operated on 57 patients since December 1, 2001, at the Cleveland Clinic Foundation, Cleveland, Ohio, using systemic cooling, which resulted in 2 in-hospital deaths (96.5% survival) and 1 permanent deficit (1.8%). One patient had a transient deficit after deep hypothermic arrest and replacement of the distal arch plus type II thoracoabdominal aorta repair. This occurred after the patient developed supraventricular tachycardia that was reversed by CSF drainage and induced hypertension. Furthermore, we no longer rewarm patients as much. Thus, we recommend active cooling with atriofemoral bypass and CSF drainage for most patients; we recommend profound hypothermia for complex concurrent arch with or without ascending repairs and reoperations.

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