Predictors of electrocerebral inactivity with deep hypothermia

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Objective: Cooling to electrocerebral inactivity (ECI) by electroencephalography (EEG) remains the gold standard to maximize cerebral and systemic organ protection during deep hypothermic circulatory arrest (DHCA). We sought to determine predictors of ECI to help guide cooling protocols when EEG monitoring is unavailable.

Methods: Between July 2005 and July 2011, 396 patients underwent thoracic aortic operation with DHCA; EEG monitoring was used in 325 (82%) of these patients to guide the cooling strategy, and constituted the study cohort. Electroencephalographic monitoring was used for all elective cases and, when available, for nonelective cases. Multivariable linear regression was used to assess predictors of the nasopharyngeal temperature and cooling time required to achieve ECI.

Results: Cooling to a nasopharyngeal temperature of 12.7°C or for a duration of 97 minutes was required to achieve ECI in >95% of patients. Only 7% and 11% of patients achieved ECI by 18°C or 50 minutes of cooling, respectively. No independent predictors of nasopharyngeal temperature at ECI were identified. Independent predictors of cooling time included body surface area (18 minutes/m^2), white race (7 minutes), and starting nasopharyngeal temperature (3 minutes/°C). Low complication rates were observed (ischemic stroke, 1.5%; permanent paraparesis/paraplegia, 1.5%; new-onset dialysis, 2.2%; and 30-day/in-hospital mortality, 4.3%).

Conclusions: Cooling to a nasopharyngeal temperature of 12.7°C or for a duration of 97 minutes achieved ECI in >95% of patients in our study population. However, patient-specific factors were poorly predictive of the temperature or cooling time required to achieve ECI, necessitating EEG monitoring for precise ECI detection. (J Thorac Cardiovasc Surg 2014;147:1002-7)

ACD

Deep hypothermic circulatory arrest (DHCA) is commonly used to provide a bloodless surgical field and to facilitate replacement of the aortic arch or descending/thoracoabdominal aorta. Although all end organs are at risk of ischemic injury during circulatory arrest, neuronal tissues may be most sensitive to oxygen deprivation. Neurologic injury during ischemia can be minimized by using hypothermia to lower the cerebral metabolic rate of oxygen consumption. Preclinical studies have shown that maximal suppression of the cerebral metabolic rate of oxygen consumption occurs at electroencephalographic (EEG) isoelectricity or electrocerebral inactivity (ECI),[1,2] and cooling to deep hypothermia (≤18°C) has evolved as the preferred technique for cerebral and systemic organ protection during DHCA.[3,4] However, the cooling time and temperature required to achieve ECI are highly variable among patients.[5] As a result, intraoperative EEG monitoring has become a valuable method for guiding temperature selection with DHCA to allow for confirmation of ECI without over- or undercooling.

Intraoperative EEG monitoring requires an advanced team of specially trained neurologists, anesthesiologists, and EEG technologists, and these services are not available at all times or at all institutions. Thus, strategies for predicting ECI in the absence of EEG monitoring are needed. In 2001, Stecker and colleagues[5] reviewed their monitored DHCA experience and found that cooling to a nasopharyngeal (NP) temperature of 12.5°C or for a duration of 50 minutes achieved ECI in 100% of patients (n = 47). However, these data have not been validated independently in the decade since they were published. In the current study, we sought to report the NP temperatures and cooling times required to achieve ECI in a larger modern cohort of patients

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Abbreviations and Acronyms
BSA = body surface area
CPB = cardiopulmonary bypass
DHCA = deep hypothermic circulatory arrest
ECI = electrocerebral inactivity
EEG = electroencephalographic
NP = nasopharyngeal
$PaCO_2$ = arterial carbon dioxide tension

undergoing thoracic aortic operation with DHCA and EEG monitoring. In addition, we hypothesized that patient-specific factors would be predictive of the temperature or cooling time required to achieve ECI and could be used to help guide cooling protocols when EEG monitoring services are unavailable.

METHODS

Patient Population and Data Collection

This study was approved by the institutional review board of Duke University; the need for individual patient consent was waived. The Duke Thoracic Aortic Surgery Database is a prospectively maintained electronic clinical registry of all patients who have undergone a thoracic aortic procedure at Duke University Medical Center (Durham, NC) since July 2005.[3,6] A query of the database identified 396 consecutive thoracic aortic operations with DHCA performed between July 2005 and July 2011; EEG monitoring was used in 325 (82%) of these patients to guide the cooling strategy, and constituted the study cohort. Electroencephalography was used for all elective cases and, when available, for nonelective cases (urgent, n = 31; emergency, n = 12). Data on intraoperative cooling parameters were ascertained from anesthesia and perfusion records. For data collection purposes, the time point of ECI was considered to be equivalent to the time of initiation of DHCA, consistent with institutional practice protocols. Cooling time was defined as the duration between the start of cooling and the time of ECI. The NP temperature was recorded at the start of cooling and at ECI. The lowest NP temperature achieved prior to rewarming was also recorded. The baseline hemoglobin concentration was recorded prior to surgery and the first arterial carbon dioxide tension ($PaCO_2$) concentration was recorded after cooling onset. Comorbid conditions and postoperative complications were defined using Society of Thoracic Surgeons definitions (www.sts.org).

Electroencephalographic Monitoring

Electroencephalographic monitoring was performed with gold disc electrodes that were applied to the scalp according to the International 10-20 electrode application system. All electrodes except Fp1 and Fp2 were applied; these electrodes were omitted because of placement of other probes on the frontal scalp region. The baseline electroencephalogram was recorded after anesthetic induction but before initiation of cooling. Continuous EEG monitoring was performed from the onset of cardiopulmonary bypass (CPB) and continued during rewarming until the termination of CPB. The EEG sensitivity was typically 5 to 7 μV/mm at the onset of CPB and was increased gradually as the EEG activity diminished in amplitude. After anesthesia was discontinued, the EEG sensitivity was increased to 2 μV/mm. Electrocerebral inactivity was not assessed until 15 minutes after cessation of anesthetics to remove any confounding effects of anesthetics on the electroencephalogram. Electrocerebral inactivity was then defined as the absence of EEG activity >2 μV/mm for a period of at least 3 minutes. When this criterion was met, the surgeon was alerted and circulatory arrest was initiated. The presence of artifact did not prevent the determination of ECI if the underlying electroencephalogram could still be interpreted. No quantitative EEG analysis was used to determine ECI. The surgeon was alerted if the electroencephalogram had asymmetry that had not been noted in the baseline recording.

Anesthetic and Perfusion Technique

Cerebral oximetry was initiated prior to induction of anesthesia. Temperature was monitored at the NP and bladder. Anesthesia was induced using a combination of intravenous fentanyl and propofol. Anesthesia was maintained using a combination of propofol and remifentanil/fentanyl infusion. Neuromuscular blockade was achieved using standard nondepolarizing agents at the anesthesiologist's discretion. Propofol and remifentanil/fentanyl infusion rates were reduced by 50% at the onset of CPB and discontinued at 28°C NP. Infusions were restarted during rewarming on resumption of electrocerebral activity. Body temperature was maintained during the rewarming and post-CPB phases with surface warming pads (Arctic Sun; Medivance Inc, Louisville, Colo). Pharmacologic neuroprotection was provided with 1 g methylprednisolone given intravenously prior to induction of anesthesia, and 4 g magnesium and 200 mg lidocaine given intravenously at the start of CPB.[7] Lidocaine (200 mg) administration was repeated at the start of rewarming.

Nonpulsatile CPB was conducted using a membrane oxygenator following a crystalloid and mannitol prime, and using an arterial line filter. Porcine heparin was administered as a bolus of 300 U/kg and supplemented to maintain an activated clotting time of >480 seconds. Cooling and rewarming were performed using a Stockert 3T (Sorin Group USA Inc, Arvada, Colo) or Hemotherm 400MR (Cincinnati Sub-Zero, Cincinnati, Ohio) heater cooler, with a 10°C gradient maintained between the blood inflow temperature and the NP temperature. A 5000-U bolus of heparin was given prior to circulatory arrest. During CPB, temperature-adjusted flow rates of 2.5 L/min/m^2 were used, and mean arterial pressure was maintained between 50 mm Hg and 70 mm Hg. Alpha stat management for maintenance of normal pH, arterial oxygen tension, and $PaCO_2$ values was used. Transfusion of blood and blood products was directed by point-of-care tests.[6,8]

Operative Technique

Prior to the portion of the aortic reconstruction requiring circulatory arrest, the patient was cooled on CPB until ECI was detected on the electroencephalogram. Concomitant procedures, such as coronary artery bypass grafting or valve procedures, were typically performed at 26°C, with full cooling resumed near completion of the concomitant procedure. After ECI was achieved, the circulation was stopped and the open aortic reconstruction portion of the case was carried out. For proximal aortic repairs including hemi- or total arch replacement, antegrade cerebral perfusion via the right axillary artery was typically used for adjunctive cerebral perfusion during the period of systemic DHCA; retrograde cerebral perfusion was used in lieu of antegrade cerebral perfusion in select hemiarch cases in which the right axillary artery was not suitable for cannulation, generally because of a diameter <6 mm on preoperative imaging or dissection of the artery.[3] For distal arch, descending, and thoracoabdominal repairs using DHCA via the left chest, no adjunctive cerebral perfusion was undertaken given the short, usually <20 minutes, cerebral DHCA times required.[9] Cannulation for left chest repairs typically involved use of a percutaneous multistage venous cannula placed via the right common femoral vein; arterial cannulation was most commonly in the descending aorta, with the femoral artery being used much less often. The arterial cannulation site was then moved to the Dacron graft, usually via an integral graft sidearm, after completion of the open proximal aortic anastomosis. After the portion of the aortic reconstruction requiring DHCA, CPB was reinstituted and the patient was rewarmed gradually after a 5-minute period of cold reperfusion for free radical washout.[10,11] The remainder of the aortic repair was completed during the rewarming phase.

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