Brain protection in thoracic aortic surgery – An interdisciplinary challenge

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In thoracic aortic disease, diagnostic imaging, prognostic information, surgical outcomes and follow-up have improved substantially within recent years [1, 2]. As a consequence, cardiovascular anesthesia and surgery are faced with an increasing caseload of thoracic aortic procedures [3]. With decreasing overall and perioperative mortality, permanent or transient neurological deficits (ND) become the major cause of morbidity, low quality of life and cost in this group [4-6].

Etiologies and risks for perioperative ND include
a) the natural course of disease (e.g., cerebrovascular involvement [7], embolization, malperfusion [8,9], dissection, rupture);
b) emergency and complexity of repair [2,10]
c) use of hypothermic circulatory arrest (HCA) [2,11-13].
d) CPB-related damage (e.g., embolization of debris, gas, fat; malperfusion, dissection, hypoperfusion, reperfusion injury)

Clinical strategies of all specialties involved in thoracic aortic surgery must focus on CNS protection:

A) diagnosis and preparation
a) early diagnosis, close control, elective repair, close follow-up
b) surgical indication must balance individual disease risks against institutional perioperative risks.
c) elective cases (ascending/arch/thoracoabdominal aneurysms/chronic dissections) require
   - comprehensive diagnostic workup (angioCT, MRI; coronaries, carotids; echo, neurological assessment etc.) and
   - interdisciplinary planning with a focus on CNS protection
d) emergencies (Type A aortic dissection, rupture) require swift, comprehensive, standardized management.

B) surgical strategies
e) usually aim for definitive repair; however, staged or hybrid procedures may be indicated to reduce ischemic risks [14,15]
f) include careful selection of cannulation/crossclamp sites; axillary/subclavian cannulation is nowadays preferred over femoral artery cannulation for fear of retrograde embolization, false lumen perfusion or dissection [5, 16,17]
g) cannulation injury/embolization can be reduced by TEE and epiaortic scanning [18-20]
h) periods of hypothermic circulatory arrest (HCA) without any perfusion support for the brain should be limited to < 30 min at < 20°C nasopharyngeal temperature [21,22]
i) de-airing must be meticulous, using positioning, CO₂-insufflation, flushing, venting or even retrograde perfusion.

C) perfusion management
j) must ensure homogeneous cerebral cooling with temperature gradients of < 10°C, should last for a sufficient amount of time; EEG silence should always precede HCA.
k) must minimize periods of cerebral no-flow/low flow by
   - antegrade cerebral perfusion (ACP) [23,24] and variants [9, 25-29] using right axillary artery cannulation [5] and/or only moderate body hypothermia during arrest [30-33]
   - HCA may be avoided altogether by appropriately combining ACP with distal body perfusion [28,34-36]
- retrograde CP [6, 10, 37,38] is still used by some
  groups despite unproven neuroprotective advan-
  tage [39], with the main goals of maintaining
  brain hypothermia and removing embolic load
l) optimizing venous drainage [40]
m) rewarming must be controlled to levels strictly be-
low 37°C [41]

D) anesthesia management
n) Opioid-based regimes are supplemented with
volatile agents or i.v. sedatives. Both for propofol
and current volatiles, neuroprotective and/or neu-
ronal ischemic preconditioning effects have been
demonstrated experimentally. However, evidence
of clinical superiority of any such regime in terms
of neurological outcome is lacking in this patient
population [42,43]. Relaxants are empirically indi-
cated to reduce oxygen demand from subclinical
shivering during cooling/rewarming.
o) Neuromonitoring:
  - Cerebral perfusion pressure must be reliably mon-
tored by appropriate arterial line placement, ex-
actly adapted to surgical plan and individual
anatomy. This should be combined with continu-
ous proximal internal jugular pressure monitoring
[40]
  - Processed EEG helps to monitor for adequate
depth of anesthesia, for episodes of EEG silence
due to hypothermia, hypnotics or ischemia, and
EEG recovery thereafter.
  - Multi-site temperature monitoring (nasopharyn-
geal, bilateral tympanic, bladder, CPB inflow etc
[44]) is required to control cooling and rewarming
of brain, core and shell.
  - TEE and epiaortic ultrasound have evidence-
  based indications in this field [18,45], e.g., assess-
ment of aortic pathology, atheromatosis, valvular
and myocardial function; guidance of cannulation
and de-airing procedures etc.
  - Cerebral oximetry by near-infrared spectroscopy
(NIRS) or invasive jugular bulb oximetry is used
to detect critical cerebrovascular Hb desaturation
due to malperfusion, hypotension, hypocapnia, ins-
sufficient cooling or ACP flow, brisk rewarming
or other causes of regional or global ischemia. Ev-
idence towards reduction of postoperative ND in
cardiothoracic patients is accruing slowly for non-
invasive continuous NIRS [40,46], but less so for
invasive jugular bulb oximetry [47-49].
  - Transcranial Doppler has been found useful for
monitoring adequacy of antegrade or retrograde
cerebral perfusion setups [50,51] and for assessing
supraaortic malperfusion or embolic load.
p) Guidance of surgical cannulation
  - During arterial and venous cannulation, TEE, mu-
lisite arterial lines, and CVP readings from the
lumen most proximal to the jugular bulb help to
avoid cannulation disasters (atheroma dislodge-
ment, malposition, dissection, venous obstruction
etc.)
q) Guidance of brain cooling and rewarming
  - multi-site temperature monitoring assesses homo-
geney and bilateral synchrony of head tempera-
ture changes.
  - Head cooling should be accompanied by appro-
riate EEG suppression and recovery, as well as by
reversible mydriasis. Mydriasis should rather be
induced by hypothermia than by arrest.
  - Topical head cooling prevents external rewar-
mimg; despite wide empirical use, optimal technique
and neuro-outcome benefits remain unclear [52-
54]

r) Monitoring and guidance of ante- or retrograde
cerebral perfusion
  - flow meter, tympanic T, data from NIRS [46] and
  TCD [50,51,55] all give some indication about ad-
edquacy of selective cerebral perfusion.
  - The Circle of Willis is incomplete in about 15% of
  patients [56]: unilateral (RA-) ACP may not suf-
fice, and may need to be supported by selective L
carotid ACP.
  - Run-off of ACP flow (into IMA or L subclavian),
malperfusion or embolism do occur and may be
detected by appropriate monitoring.
s) Guidance of de-airing
  - By TEE, head-down positioning, intermittent
carotid compression etc. anesthesia contributes to
  – largely empirical – efforts to de-air the left heart
and arterial tree (like field flooding with CO2
[57], short RCP, slow reperfusion, agitation, nee-
dle venting etc).
  - “Pharmacologic neuroprotection” [42,43 ]
  - A variety of agents is in wide use, but still without
  – even empirical – evidence of benefit [58]
  - Steroids: there is no evidence for neuroprotective
efficacy in HCA [59], but hyperglycemic risk is
promoted.
  - Thiopental, Propofol: since decades, clinical evi-
dence for benefit prior to HCA remains insuffi-
cient [21]; bolus doses interfere with EEG moni-
toring and are cerebral vasoconstrictors. Both may
be useful to normalize cerebral O₂ balance at rewarming [40].
- Volatiles: evidence for clinically useful neuronal protection and preconditioning is insufficient [42]; agents may reduce CPP but promote cooling and improve post-CPB myocardial function.
- Aprotinin: despite some neuroprotective evidence [60], the substance has been withdrawn in 2007.

u) Blood gas management
- There is wide agreement to use a-stat monitoring of blood gases
- Inadvertent hypocapnia may impair CBF and cerebral oxygenation and is to be avoided.
- Many institutions employ mild hypercapnia during cooling for HCA, and α-stat normocapnia during rewarming.

v) Monitoring/guidance of glycemia
- Hyperglycemia is known to worsen ischemic CNS damage. Postoperatively, “tight” normoglycemic control (4.4 – 6.1 mmol/L) has been shown to improve survival after high-risk cardiac surgery [61], but at a substantial risk of hypoglycemia. Intraoperatively, a RCT in cardiac surgery failed to show any benefits of intraoperative “tight” glycemic control but undesirable trends in death and stroke rate [62]. Therefore, intraoperative glycemic control (e.g., to 4.5 - 9 mmol/L) appears preferable to “tight” schedules with hypoglycemic risks.

w) Management of coagulation
- Large transfusion requirement is an independent predictor of perioperative stroke risk in cardiac surgery [63]. Tranexamic acid has been shown to reduce transfusion requirement in thoracic aortic surgery [64]. Reduction of homologous transfusion (and in particular, platelets) by appropriate point-of-care testing and transfusion algorithms may open new approaches to reduce ND in thoracic aortic surgery.

x) Temperature management
- Posts ischemic hyperthermia worsens neurologic outcome [41]. After thoracic aortic surgery, T > 37° in perfusate, core, nasopharynx or tympanon of patients must be avoided at all times, by early start of rewarming, by keeping patient-perfusate gradients < 10 °C, by stopping rewarming at nasopharyngeal T ≤ 36.5°C (even at the cost of some afterdrop), and slow correction by external warming devices [65].


