Anesthetic management for resection of ascending/arch aortic aneurysm: CNS protection

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Hypotheses/Preliminary Recommendations:

1. A neuroprotection strategy geared towards stroke prevention and preservation of cognitive function should be a key element of the surgical, anesthetic, and perfusion techniques to accomplish repairs of the ascending aorta and transverse aortic arch. [1, 2, 3, 4, 5, 6, 7]
2. Deep hypothermic circulatory arrest, selective brain perfusion, and retrograde brain perfusion are each techniques that alone or in combination are reasonable to facilitate interruptions of aortic continuity during surgical repairs of the ascending aorta and transverse aortic arch. Institutional experience is an important factor in selecting these techniques. [8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31]
3. Perioperative brain hyperthermia is not recommended in repairs of the ascending aortic and transverse aortic arch as it is probably injurious to the brain. *(Level of Evidence: C)* [32, 33, 34].

Summary of Evidence

A brain protection strategy for open surgical repairs of the ascending aorta and/or the aortic arch is an essential component of the operative technique. Moderate or profound hypothermia with periods of circulatory arrest and/or selective anterograde brain perfusion and/or retrograde brain perfusion are the common strategies for achieving brain protection. The experience and outcomes of the operating surgeon and the institution are important considerations in selecting a brain protection strategy.

Achieving brain hypothermia is nearly universally performed using extracorporeal circulation, and the reported temperatures range from 12 to 30 degrees C. Retrograde brain perfusion is less commonly used, and is usually performed at a perfusion pressure of 20 to 40 mm Hg at a mildly or profoundly hypothermic temperature. Anterograde (selective) brain perfusion is usually performed so as to achieve a perfusion pressure of 50 to 80 mm Hg, and may be instituted by direct cannulation of the brachiocephalic vessels, side-graft anastomosis to the axillary artery, or direct cannulation of a portion of graft material that was anastomosed to the brachiocephalic vessels during a period of hypothermic circulatory arrest. The rewarming of a patient following completion of the repair of the thoracic aorta is usually performed at a measured rate so as not to induce brain hyperthermia following the period when the neuroprotective strategy was employed.

The reviewed literature describes an evolution of brain protection techniques over the last 2 to 3 decades. Deep hypothermic circulatory arrest emerged as the first technique associated with morbidity and mortality rates that represented vast improvements over the natural history of thoracic aneurysms and dissections that were managed conservatively.

Hypothermic circulatory arrest as a sole method of brain protection, however, was limited by increasing rates of neurological morbidity, other adverse outcomes, and mortality as the period of arrest exceeded 25-45 minutes ([35], [13]). These findings led clinicians to investigate adjuncts, such as various combinations of retrograde brain perfusion and selective anterograde brain perfusion in attempts to extend the “safe period” of interruption of full extracorporeal circulation. One area of variability in the literature is the degree of hypothermia that was induced with the addition of retrograde and anterograde brain perfusion. Theoretically, the lesser degree of hypothermia should require decreased cooling and rewarming intervals of extracorporeal circulation, which could decrease total time of extracorporeal circulation, as well as any adverse effects of more profound degrees of hypothermia (e.g., coagulopathy). Areas of variability among practices include the degree of hypothermia that is sufficient to ensure a safe period of arrest, and means of monitoring to ascertain that sufficient cooling has been achieved. The most common modalities utilized are cooling protocols that specify temperature targets at specific body sites and/or set periods of cooling at specific perfusate temperatures. Monitoring of brain function and metabolic suppression as assessed by electroencephalography, evoked potentials, bispectral index, non-invasive cerebral oximetry,

and jugular bulb oxyhemoglobin saturation are additional means used according to institutional protocol to guide the onset of extracorporeal circulation interruption for repairing the distal ascending aorta and/or aortic arch.

Retrograde brain perfusion remains in use in several centers. The literature is controversial regarding the ability of retrograde brain perfusion to support brain metabolic function, and to improve neurological outcomes, including transient postoperative neurological dysfunction, stroke rates, and mortality ([3, 10, **13**, **15**, **16**, **17**, 36, 37, 38, 39, 40, 41]. The experimental literature supports the ability of retrograde brain perfusion to maintain brain hypothermia[42], and this may explain how this modality has been associated with improved outcomes in the centers where it has assumed a role in the neuroprotection strategy.

Selective anterograde brain perfusion is implemented in a variety of ways. There may be direct cannulation of one or more of the brachiocephalic vessels with complete obviation of the need for a period of interruption of cerebral blood flow. The patency of the Circle of Willis will influence this approach if unilateral cannulation is considered. Alternatively, unilateral direct or side-graft cannulation of the (usually right) axillary or brachial artery is commonly used as a means of instituting extracorporeal circulation and cooling (without manipulation of the diseased thoracic aorta). This same cannula can then be used for delivering anterograde brain perfusion once continuity with the brain circulation is restored. For example, this may occur immediately after the section of aorta from which the brachiocephalic arteries originate is sutured to the graft or immediately after the brachiocephalic vessels are individually anastomosed to a trifurcated graft. The time required to complete these maneuvers to restore continuity of the brain circulation requires a relatively shorter period of hypothermic circulatory arrest compared with compete reconstruction of the aortic arch. ([**23**, **24**, **25**, 43, 44, 45, 46]). Alternatively, bilateral brachiocephalic vessel cannulation has been reported ([47, 48]). The literature is insufficient to determine whether unilateral or bilateral perfusion, or complete avoidance of circulatory arrest is associated with improved outcomes. A retrospective analysis by Svensson et al suggested that axillary artery perfusion via a side graft of prosthetic material was associated with improved outcomes, whereas femoral arterial cannulation was associated with adverse outcomes ([49]). Finally, direct cannulation of the graft material may be used to institute anterograde brain perfusion following a period of circulatory arrest. The variability among surgical centers complicates the interpretation of the literature, however the great majority of studies report outcomes that are comparable or better than hypothermic circulatory arrest alone or retrograde brain perfusion ([**1**, **2**, **7**, **27**, **28**, 50, 51]). Furthermore, there is ample literature to suggest that in the face of the diminished period of obligatory brain ischemia with selective anterograde brain perfusion, use of less profound hypothermia is associated with good clinical outcomes ([**20**, **29**, 52, 53, 54, 55, 56]).

There are several problems with the clinical literature regarding neuroprotection during reconstruction of the distal ascending aorta and aortic arch. The most important limitation is that most centers have evolved their procedural approaches over time. The typical pattern is a progression from the original technique where there was a prolonged period of hypothermic circulatory arrest through period(s) where retrograde brain perfusion and/or anterograde brain perfusion have been used. Therefore, the literature is almost universally composed of nonrandomized reports of clinical cohorts where the control groups were non-contemporaneous. The major limitation of that approach is that concurrent changes in surgical technique, perfusion technology, anesthetic and intensive care management, coagulation management, prosthetic graft materials, and the experience of the centers are confounding factors. It is arguable that randomized trials are impractical or potentially unethical in this clinical scenario, considering the gradual improvements in elective surgical results that have occurred over time. Therefore, the ability to draw evidence-based guidelines from the literature is particularly difficult in the case of brain protection.

References

1. Akashi H, et al. Jpn J Thorac Cardiovasc Surg 2000;48:782-8.
2. Di Eusanio M, et al. J Thorac Cardiovasc Surg 2003;125:849-54.
3. Ehrlich MP, et al. J Thorac Cardiovasc Surg 1999;118:1026-32.
4. Hagl C, et al. J Thorac Cardiovasc Surg 2001;121:1107-21.
5. Reich DL, et al. J Thorac Cardiovasc Surg 1999;117:156-63.
6. Reich DL, et al. Eur J Cardiothorac Surg 2001;19:594-600.
7. Usui A, et al. Eur J Cardiothorac Surg 1999;15:571-8.

8. Kunihara T, et al. J Thorac Cardiovasc Surg 2005;130:712-8.
9. Schepens, et al. Eur J Cardiothorac Surg 2002;21:276-8.
10. Bavaria JE, et al. Ann Surg 2001;234:336-43.
11. Deeb GM, et al. Ann Thorac Surg 1999;67:1883-6.
12. Ehrlich M, et al. Circulation 2003;108:318-23.
13. Fleck TM, et al. Ann Thorac Surg 2003;76:1198-202.
14. Moshkovitz Y, et al. Ann Thorac Surg 1998;66:1179-84.
15. Okita Y, et al. J Thorac Cardiovasc Surg 1998;115:129-38.
16. Ueda Y, et al. Ann Thorac Surg 1999;67:1879-82.
17. Wong CH, et al. Ann Thorac Surg 1999;67:1900-3.
18. Di Eusanio M, et al. J Thorac Cardiovasc Surg 2002;124:1080-6.
19. Di Eusanio M, et al. Ann Thorac Surg 2003;75:514-9.
20. Di Eusanio M, et al. Ann Thorac Surg 2003;76:1181-9.
21. Kazui T, et al. Ann Thorac Surg 2002;74:S1806-9.
22. Kazui T, et al. Ann Thorac Surg 2007;83:S796-8.
23. Numata S, et al. Eur J Cardiothorac Surg 2003;23:771-5.
24. Sasaki H, et al. Ann Thorac Surg 2007;83:S805-10.
25. Strauch JT, et al. Ann Thorac Surg 2004;78:103-8.
26. Kamiya H, et al. Ann Thorac Surg 2007;83:1055-8.
27. Matalanis G, et al. Ann Thorac Cardiovasc Surg 2003;9:174-9.
28. Okita Y, et al. Ann Thorac Surg 2001;72:72-9.
29. Zierer A, et al. Thorac Cardiovasc Surg 2005;53:74-9.
30. Okita Y, et al. Ann Thorac Surg 1999;67:72-8.
31. Svensson LG, et al. Ann Thorac Surg 2002;74:2040-6.
32. Grigore AM, et al. Anesth Analg 2002;94:4-10.
33. Grocott HP, et al. Stroke 2002;33:537-541.
34. Bar-Yosef S, et al. Anesth Analg 2004;99:641-646.
35. Chong SY et al. Ann Acad Med Singapore 2004;33:289-93
36. Bonser RS et al. J Thorac Cardiovasc Surg 2002;123:943-50.
37. Cheung AT et al. Anesth Analg 1999;88:8-15.
38. Ehrlich M et al. Circulation 1998;98:II-294-II-298.
39. Estrera AL et al. Ann Thorac Surg 2002;74:1058-65.
40. LeMaire SA et al. Ann Thorac Surg 2001;71:1913-9.
41. Moon MR et al. Ann Thorac Surg 2002;74:426-31.
42. Ehrlich MP et al. J Thorac Cardiovasc Surg 2001;122:331-8.
43. Karadeniz U et al. Ann Thorac Surg 2005;79:139-46.
44. Neri E et al. J Thorac Cardiovasc Surg 2002;123:901-10.
45. Wozniak G et al. Int J Angiol 1999;8:50-6.
46. Yilik L et al, Tex Heart Inst J 2006;33:310-5.
47. Olsson C et al. Ann Thorac Surg 2006;81:868-74.
48. Veeragandham RS et al. Ann Thorac Surg 1998;66:493-9.
49. Svensson LG et al. Ann Thorac Surg 2004;78:1274-84.
50. Harrington DK et al. Circulation 2004;110[suppl II];II-231:11-236.
51. Neri E, et al. Ann Thorac Surg 2004;77:72-80.
52. Bahktiary F et al. J Thorac Cardiovasc Surg 2006;132:153-4.
53. Cook RC et al. J Card Surg 2006;21:158-164.
54. Kamiya H et al. J Thorac Cardiovasc Surg 2007;133:501-9.
55. Kaneda T et al. Scand Cardiovasc J 2005;39:87-90.
56. Testolin L et al. Cardiovasc Surg 1998;6:398-405.