REVIEW ARTICLE

Benefits of using dexmedetomidine during carotid endarterectomy: A review

Abhijit S. Nair

Department of Anesthesia, Yashoda Hospitals, Somajiguda, Hyderabad, Andhra Pradesh, India

Address for correspondence:
Dr. Abhijit S. Nair
Department of Anesthesia,
Fifth Floor, Yashoda Hospitals,
Raj Bhavan Road, Somajiguda,
Hyderabad - 500 082,
Andhra Pradesh, India.
E-mail: abhijitnair95@gmail.com

ABSTRACT

As per current recommendation, patients with acute ischemic stroke should be offered carotid endarterectomy (CEA) within 24-72 hours. The same applies to patients with recurrent transient ischemic attacks (TIA). This time is usually less for hemodynamic optimization of patients who've suffered acute ischemic stroke. Hence' they are hemodynamically labile and can have accelerated hypertension on induction/extubation. This can have disastrous outcomes. It is a common practice among anesthesiologists to avoid angiotensin converting enzyme(ACE) inhibitors or angiotensin receptor blockers on the day of surgery. This also adds to hypertensive issues perioperatively. Dexmedetomidine is a wonderful drug which can be used during CEA. Due to its centrally mediated sympatholytic effect, it confers good hemodynamic control during induction, intraoperatively, and during extubation. We did a search on PubMed and Google for carotid endarterectomies done under general and locoregional anesthesia during which dexmedetomidine was used. The keywords used by us during the search were as follows: anesthesia, carotid endarterectomy, anesthesia. We also searched for use of dexmedetomidine infusion to attenuate hypertensive response to intubation and for providing stability in major surgeries like CABG, craniotomies, bariatric surgeries, and valve replacements.

Key words: *Anesthesia, carotid endarterectomy, dexmedetomidine, stroke*

INTRODUCTION

Carotid endarterectomy (CEA) is a non-cardiac vascular surgery considered in patients with symptomatic carotid artery disease to prevent or decrease the risk of disabling or life-threatening strokes. These patients usually have significant co-morbidities like coronary artery disease, long standing hypertension and diabetes, chronic obstructive pulmonary disease (COPD), peripheral vascular disease, renal dysfunction, dyslipidemias, and extremes of age. CEA is done under general anesthesia or locoregional anesthesia. General anesthesia offers good surgical conditions and the anesthesiologist has control over ventilation. However, in this method, the hemodynamic response during induction, intubation, and extubation can be disastrous and can lead to significant morbidity. The advantage of the locoregional technique is that one can monitor neurological status intraoperatively and stressful situations during anesthesia induction, intubation, and extubation can be avoided.

Access this article online	
Quick Response Code:	Website: www.saudija.org
	DOI: 10.4103/1658-354X.130744

GA vs locoregional!

The GALA trial by Gough *et al.*[1] in 2008 was a multicenter, randomized controlled trial which enrolled 3500 patients from 90 centers between 2001 and 2007. They compared general anesthetic versus local anesthesia technique for patients scheduled for CEA. They concluded that there was no major difference in 30 day mortality in either groups and that the choice of anesthetic doesn't affect the perioperative outcome. Rerkasem *et al.*[2] also concluded from their review published in Cochrane Database Systematic Review that there is insufficient evidence of comparing local versus general anesthesia for carotid endarterectomy from the randomized trials.

Goals of the anesthetic technique for CEA should be to maintain optimal hemodynamics, avoid precipitous hypotension, reduced perioperative stress, to use short-acting anesthesia drugs agents to facilitate postoperative cerebral function assessment, and to optimize cerebral perfusion. The anesthetic agent should have no or minimal respiratory

depression, shouldn't cause hemodynamic instability i.e. no tachycardia, hypertension/hypotension, short acting and without active metabolites and residual effects.

Hypertension in CEA

In patients with carotid artery disease, the baroreceptor in carotid sinus, which is one of the important factor responsible for physiological control of blood pressure (BP) in humans, gets included in the disease process. This leads to alteration in the baroreceptor function which leads to perioperative hypertensive issues.[3] Badly controlled preoperative BP leads to possible end organ damage. Uncontrolled preoperative BP is independently responsible for postoperative hypertension. If BP is not judiciously controlled in the postoperative period, the possible serious consequences are: Myocardial infarction, arrhythmias, left ventricular failure, prolonged stay in ICU, intracerebral hemorrhage (ICH), and cerebral hyperperfusion syndrome (CHS).[4] ICH and CHS can have disastrous outcomes if BP is not managed appropriately in the postoperative period. CHS is usually seen between 2 and 7 days after surgery and leads to severe headache, seizures, and neurodeficits. Clinically, it mimics hypertensive encephalopathy. Earlier it was thought to be due to increased perfusion after CEA but later it was proved to be due to ischemia — reperfusion injury which leads to cerebral edema.[5] Causes of ICH after CEA are uncontrolled postoperative hypertension, severe contralateral carotid artery disease, and increased cerebral blood flow in middle cerebral artery territory due to CEA.[6] Postoperative hypertension after CEA is usually managed with IV medications like Labetalol, Esmolol, Enalaprilat, hydralazine, etc. Agents like nitroglycerin, sodium nitroprusside, nicardipine are usually not used as it leads to cerebral vasodilation which can increase ICP.[7]

Advantages of dexmedetomidine

Dexmedetomidine is a centrally acting α2 agonist, approved by FDA for sedation in ICU for not more than 24 hours. It provides sedation, anxiolysis, analgesia, causes sympatholysis, blunts hemodynamic response to intubation/extubation/surgical stress. It reduces the requirement of intravenous anesthetic, inhalational anesthetic during induction, and maintenance of anesthesia.[8] Dexmedetomidine potentiates the action of regional anesthesia, reduces opioid requirement significantly, has anti-sialogogue effect, and is a very good anti-shivering agent. The recommended dose of dexmedetomidine is 1 μg/kg as a loading dose over 10-15 min, followed by a maintenance infusion @ 0.2-0.7 μg/kg/hour. After the initial loading dose due to reduction in CNS sympathetic activity, bradycardia and a fall in BP is observed but it can be managed by small doses of anticholinergics (Atropine, Glycopyrollate).[9] However, a dose reduction is recommended in patients with significant renal and hepatic impairment. It should be used judiciously in geriatric patients and in patients with significant biventricular dysfunction. Bajwa *et al.*[10] and Yildiz *et al.*[11] described how effectively dexmedetomidine attenuated the pressor response to laryngoscopy, intubation, extubation and how there was a dose sparing of narcotics and anesthetics when given as a loading dose preoperatively and continued perioperatively.

Review of literature

Recent guidelines suggests that patients with acute ischemic stroke or recurrent TIA should be offered CEA in 24-72 hours contrary to earlier recommendation of after 4-6 weeks after the event.[12,13] Post stroke, patients have an elevated BP and for a surgery which is scheduled in 24-72 hours, the time for optimization of BP is less. Adding to this, patients for CEA usually have associated comorbidities like hypertension, coronary artery disease, peripheral vascular disease, COPD, diabetes mellitus, etc. Stoneham *et al.*[14] in their review article discussed in detail about hemodynamic instability during CEA. They discussed the patient factors, surgical factors, and anesthetic factors and suggested practical aspects of arterial pressure management. They stressed on the point that aggressive management can be harmful to the patient and that closed monitoring of patient is important rather than specific choice of agents. McCutcheon *et al.*[15] recruited 54 patients for CEA under regional anesthesia and enrolled 25 patients in thedexmedetomidine group and 29 patients in the standard group. Patients in the dexmedetomidine group received 0.5 μg/kg bolus over 10 min followed by infusion @ 0.2 μg/kg/hour. The supplementation was with 2 ml of placebo. In the standard group, patients received 40 μg fentanyl and 1 mg midazolam bolus (they prepared a 10 ml syringe with fentanyl 20 μg/ml and 0.5 mg/ml of midazolam). After bolus, the infusion for maintenance was started from the 10 ml syringe at the same rate as that of dexmedetomidine infusion. Supplementation was done whenever required with 2 ml placebo. They concluded that in the dexmedetomidine group, there was less intraoperative and postoperative hypertension and tachycardia. But the incidence of bradycardia and hypotension was also more in the dexmedetomidine group. Bekker *et al.*[16] randomized 66 patients undergoing awake CEA to receive dexmedetomidine and normal saline. The surgeries were done under locoregional anesthesia. They used midazolam, fentanyl, and propofol for supplementation. They found that patients receiving dexmedetomidine required less antihypertensive therapy in the post-operative period as compared to the second group which received more supplementation. Sidorowicz *et al.*[17] randomized 64 patients into two groups, first received 1 μg/kg of dexmedetomidine over 10 min followed by 0.2 μg/kg/hour infusion. The second group received placebo with fentanyl and urapidil for supplementation.

The patients received infiltration anesthesia. They concluded that patients in the dexmedetomidine group were hemodynamically stable and didn't require additional analgesia. However, a significant decrease in heart rate was seen in the dexmedetomidine group after 12 minutes.

DISCUSSION

Dexmedetomidine is a very safe drug which when used as an adjunct to general anesthesia provides excellent hemodynamics due to its central sympatholytic effects. But *et al.*[18] used dexmedetomidine infusion along with routine anesthesia agent for patients with pulmonary hypertension posted for mitral valve replacement surgery and found that there was attenuation of sympathetic response during intubation and during sternotomy in patients who received dexmedetomidine. Kabukçu *et al.*[19] used dexmedetomidine as an adjunct to general anesthesia for 20 patients posted for coronary artery bypass grafting and concluded that it provided stable hemodynamics in the perioperative period.

Bakhamees *et al.*[20] used dexmedetomidine infusion in morbidly obese patients posted for laparoscopic gastric bypass surgery and found that it provided better recovery profile, stable hemodynamics, reduced narcotic consumption, and better analgesia as compared to placebo. Similarly, Tufanogullari *et al.*[21] after using dexmedetomidine infusion for laparoscopic bariatric surgery recommended its use to minimize risks of perioperative cardiovascular events. Talke *et al.*[22] measured plasma norepinephrine levels in patients posted for vascular surgery in patients who received dexmedetomidine as an adjunct to routine general anesthesia as well as in patients who didn't receive dexmedetomidine.

They found out that plasma norepinephrine levels were significantly low in the patients who received dexmedetomidine. Bekker *et al.*[23] used dexmedetomidine infusion in 72 recruited patients for craniotomy and concluded that there was global hemodynamic stability with its use perioperatively.

Similarly, if dexmedetomidine infusion is used for CEA under general or locoregioanal anesthesia in recommended doses, there is central sympatholysis which provides great hemodynamic stability perioperatively. The infusion can be continued postoperatively till the patient starts taking oral antihypertensives.

CONCLUSION

Perioperative management of hypertension is very important in patients who undergo CEA. If not managed appropriately the patients can have intracerebral bleed, LVF, MI, arrhythmias, etc. The patients are on antiplatelets and heparin is also used intraoperatively. Uncontrolled hypertension postoperatively can lead to bleeding, hematoma, and in unfortunate patients can lead to respiratory compromise due to airway obstruction. Dexmedetomidine is an ideal drug which can be used as an adjunct during CEA either with general or locoregional anesthesia. Due to its sympatholytic effect and the hemodynamic stability that it confers when it is used judiciously, adverse hemodynamic response during induction and extubation can be avoided.

REFERENCES

1. Gough MJ, Bodenham A, Horrocks M, Colam B, Lewis SC, Rothwell PM *et al*. GALA: An international multicentre randomised trial comparing general anesthesia versus local anesthesia for carotid surgery. Trials 2008;9:28.
2. Rerkasem K, Rothwell PM. Local versus general anesthesia for carotid endarterectomy. Cochrane Database Syst Rev 2008;4:CD000126.
3. Sykora M, Diedler J, Poli S, Rupp A, Turcani P, Steiner T. Blood pressure course in acute stroke relates to baroreflex dysfunction. Cerebrovasc Dis 2010;30:172-9.
4. Jain AR, Bellolio MF, Stead LG. Treatment of hypertension in acute ischemic stroke. Curr Treat Options Neurol 2009;11:120-5.
5. Adhiyaman V, Alexander S. Cerebral hyperperfusion syndrome following carotid endarterectomy. QJM 2007;100:239-44.
6. Russell DA, Gough MJ. Intracerebral haemorrhage following carotid endarterectomy. Eur J Vasc Endovasc Surg 2004;28:115-23.
7. Aiyagari V, Gorelick PB. Management of blood pressure for acute and recurrent stroke. Stroke 2009;40:2251-6.
8. Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. Drugs 2000;59:263-8.
9. Chrysostomou C, Schmitt CG. Dexmedetomidine: sedation, analgesia and beyond. Expert Opin Drug Metab Toxicol 2008;4:619-27.
10. Bajwa SJ, Kaur J, Singh A, Parmar S, Singh G, Kulshrestha A, *et al*. Attenuation of pressor response and dose sparing of opioids and anesthetic with pre-operative dexmedetomidine. Indian J Anaesth 2012;56:123-8.
11. Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. Effect of dexmedetomidine on haemodynamic responses to laryngoscopy and intubation: Perioperative haemodynamics and anesthetic requirements. Drugs R D 2006;7:43-52.
12. Bruls S, Van Damme H, Defraigne JO. Timing of carotid endarterectomy: A comprehensive review. Acta Chir Belg 2012;112:3-7.
13. Baron EM, Baty DE, Loftus CM. The timing of carotid endarterectomy post stroke. Neurosurg Clin N Am 2008;19:425-32.
14. Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. Br J Anaesth 2009;102:442-52.
15. McCutcheon CA, Orme RM, Scott DA, Davies MJ, McGlade DP. A comparison of dexmedetomidine versus conventional therapy for sedation and hemodynamic control during carotid endarterectomy performed under regional anesthesia. Anesth Analg 2006;102:668-75.
16. Bekker AY, Basile J, Gold M, Riles T, Adelman M, Cuff G, *et al*. Dexmedetomidine for awake carotid endarterectomy: Efficacy, hemodynamic profile, and side effects. J Neurosurg Anesthesiol 2004;16:126-35.

17. Sidorowicz M, Owczuk R, Kwiecińska B, Wujtewicz MA, Wojciechowski J, Wujtewicz M. Dexmedetomidine sedation for carotid endarterectomy. Anestezjol Intens Ter 2009;41:78-83.
18. But AK, Ozgul U, Erdil F, Gulhas N, Toprak HI, Durmus M *et al*. The effects of pre-operative dexmedetomidine infusion on hemodynamics in patients with pulmonary hypertension undergoing mitral valve replacement surgery. Acta Anaesthesiol Scand 2006;50:1207-12.
19. Kabukçu HK, Sahin N, Temel Y, Titiz TA. Hemodynamics in coronary artery bypass surgery: Effects of intraoperative dexmedetomidine administration. Anaesthesist 2011;60: 427-31.
20. Bakhamees HS, El-Halafawy YM, El-Kerdawy HM, Gouda NM, Altemyatt S. Effects of dexmedetomidine in morbidly obese patients undergoing laparoscopic gastric bypass. Middle East J Anesthesiol 2007;19:537-51.
21. Tufanogullari B, White PF, Peixoto MP, Kianpour D, Lacour T, Griffin J, *et al*. Dexmedetomidine infusion during laparoscopic bariatric surgery: The effect on recovery outcome variables. Anesth Analg 2008;106:1741-8.
22. Talke P, Chen R, Thomas B, Aggarwall A, Gottlieb A, Thorborg P *et al*. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. Anesth Analg 2000;90:834-9.
23. Bekker A, Sturaitis M, Bloom M, Moric M, Golfinos J, Parker E, *et al*. The effect of dexmedetomidine on perioperative hemodynamics in patients undergoing craniotomy. Anesth Analg. 2008;107:1340-7.

How to cite this article: Nair AS. Benefits of using dexmedetomidine during carotid endarterectomy: A review. Saudi J Anaesth 2014; 8:264-7.

Source of Support: Nil, **Conflict of Interest:** None declared.