International Journal of
Surgery & Surgical Procedures

Review Article Open Access

Carotid Stenosis and Anaesthesia

Escalona Belmonte JJ, Biteri Martínez De Iturrate A, Guerrero Orriach JL*, Ramirez Fernandez A, Ramirez Aliaga M, Rodriguez Capitán MJ, Bermudez L, Gallego M, Loras P, Raigon A, Baena M, Rubio Navarro M and Cruz Mañas J

Department of Anaesthesia, Hospital Virgen de la Victoria, Málaga, Spain

Abstract

Carotid stenosis is an issue of enormous clinical and social relevance, so the scientific literature about this subject is very large. The intent of this review is to make a brief review of this entity to subsequently address it from the point of view of the anaesthesiologist who meets her in the operating room, in order to provide some idea of the impact of this entity, the indication of intervention, the possibilities for them, monitoring during the process and the different plausible anaesthetic techniques.

Publication History:

Received: January 09, 2015
Accepted: May 08, 2016
Published: May 10, 2016

Keywords:

Liver transplantation, Older donors

Epidemiology

The importance of carotid stenosis approach lies in the relationship it has with ischemic stroke, since it is present in 10-20% of all of them. This is of magnitude when we know that in Western countries, stroke is the third leading cause of death, behind only heart disease and cancer, with a mortality ranging from 10 to 30%, having survivors risk of recurring ischemic cardiac or neurologic events, so much so that is the leading cause of disability. The prevalence of stenosis in the internal carotid artery (ICA) is high and furthermore increases with age, in fact more than 50% stenosis rises from 0.5% in people aged between 50 and 59 years to 10% in those over 80 years. The primary mechanism involved in stroke in patients with carotid stenosis is embolism of atherosclerotic or thrombotic waste material from the plate to the distal cerebral vasculature. Infiltration of inflammatory cells on the surface of carotid plaques can play a key role in causing plaque rupture and embolization or occlusion of the carotid and also hemodynamic factors are correlated with increased risk of stroke in patients with carotid stenosis [1,2,3]. Of all the factors studied, the degree of arterial stenosis has emerged as the most directly related to the increased risk of stroke. Any diagnostic strategy, whatever the technique used, should have as its primary objective to determine, in the most accurate way, the amount of this stenosis [4].

In the NASCET study the risk of ipsilateral recurrent stroke in patients with symptomatic carotid stenosis treated conservatively was 4.4% per year for patients with stenosis between 50 and 69% and 13% per year for those with 70% stenosis. The risk in patients with asymptomatic stenosis of 60% was around 1-2%, but could rise to 3-4% per year in elderly patients or in the presence of stenosis or occlusion of the contralateral artery, evidence of silent embolization in an imaging test, heterogeneity of the carotid plaque, poor collateral circulation, generalized inflammatory state or peripheral or coronary artery disease associated [5].

Treatment

Medical versus interventional treatment

Given the above discussion, we will assume that the main objective in the treatment of carotid stenosis is the prevention of ischemic stroke and its aftermath, regardless of the type of patient, the presence or absence of symptoms and the degree of stenosis. However, despite the clarity of the target it is unclear what therapeutic option to choose among only medical treatment or revascularization and, in the latter case, between endoluminal or surgical treatment. We will be guided in this review by the latest clinical practice guidelines of the Society for Vascular Surgery (Updated Society for Vascular Surgery (SVS) Guidelines) [6], considering that these guidelines place more emphasis on stroke prevention in the prevention of ischemic heart disease, on the grounds that according to the CREST study, stroke has shown greater impact on quality of life of the patient than nonfatal myocardial ischemia [7].

The major determinants of the clinical course are the presence or absence of neurological symptoms and the degree of stenosis. The threat of ischemic stroke in asymptomatic patients with stenosis less than 60% and with symptomatic stenosis of 50% is small and generally not justifies intervention. NASCET and ECTS studies concluded that carotid endarterectomy (CEA) in symptomatic patients with stenosis less than 50% did not improve the rate of ischemic stroke and even increased morbidity of patients against medical treatment. Stenosis below 60% in asymptomatic patients was excluded from studies because it is considered that such a scenario does not benefit from carotid reconstruction [6].

Medical versus interventional treatment

The decision between the two types of intervention is also controversial because, there are no published trials with sufficient power, although in general, and without exceeding the limits of this review, we can draw some recommendations [6].

1. Neurological symptoms and less than 50% stenosis or asymptomatic stenosis less than 60%: optimized medical treatment.
2. Any patient susceptible of intervention, CEA would be made better than SCA, with the intention of reducing the risk of stroke and death.
3. It is preferred CEA over CAS in patients over 70 years, with lesions larger than 15 mm, preocclusive stenosis or lipid-rich plaques.

***Corresponding Author:** Dr Jose Luis Guerrero Orriach, Hospital Virgen de la Victoria, Campus Universitario Teatinos C.P. 29010, Malaga, Spain; E-mail: guerreroorriach@terra.com

Citation: Escalona Belmonte JJ, Biteri Martínez De Iturrate A, Guerrero Orriach JL, Ramirez Fernandez A, Ramirez Aliaga M Rodriguez Capitán MJ, et al. (2016) Carotid Stenosis and Anaesthesia. Int J Surg Surgical Porced 1: 105. doi: http://dx.doi.org/10.15344/ijssp/2016/105

- It is preferred CAS over CEA in symptomatic patients with stenosis of 50% and tracheal stoma, situations where there are fibrosis or scarring in local tissues as a result of previous surgery or radiotherapy, in previous cranial nerve injuries and injuries that lower the level of the clavicle or rise above the level C2.
- It is preferred CAS over CEA in symptomatic patients with greater than or equal to 50% stenosis, and severe coronary and uncorrectable disease, congestive heart failure or chronic obstructive pulmonary disease (COPD).
- Patients without neurological symptoms considered high risk for CEA should be considered for medical treatment. They will be considered for CEA when evidence of perioperative morbidity and mortality is less than 3%. The CAS should not be performed in these patients.
- There are insufficient data to recommend CAS as the first therapy for neurologically asymptomatic patients with stenosis of 70 to 99%. SCA could be equivalent to CEA in well-selected cases in centres with sufficient experience, with a combined rate of ischemic stroke and death below 3%.

	CAS candidates	CEA candidates
Anatomical factors	Lesion above C2	lipid-rich soft plaque
	Below collarbone injury	further injury to 15 mm
	Reoperation after previous CEA	preocclusive injury
	Cervical stoma	severe circumferential Calcification
	Previous cervical radiotherapy	aorto-iliac tortuosity
	Previous ablative cervical surgery	Great angulation of the aortic arch and carotid artery
		heavily calcified aortic arch or with severe atherosclerosis
Pathophysiological factors	Heart disease	Patient over 80 years
	Contralateral vocal cord paralysis	

Table 1: Anatomical factors, Pathophysiological factors among CAS candidates and CEA candidates.

However it is noteworthy that the recommendations we have followed are indicative, based on this clinical practice guideline and that there are review papers comparing the different guides and finding in them some differences [8].

Monitoring cerebral ischaemia

Both carotid clamping during CEA as balloon inflation during the stenting can cause a cessation in blood flow, especially in an already diseased carotid artery. At such times, the ipsilateral to the technique cerebral perfusion depends on the contralateral flow, so if this is inappropriate, appears tissue ischemia. In addition, the rupture of atherosclerotic plaque or other surgical debris can lead to the appearance of embolic ischemic events, if they are accidentally dragged into the cerebral circulation. Thus, brain tissue is susceptible to ischemic injury during the development of this surgery.

However, not all episodes of cerebral ischemia conclude in ischemic stroke, the outcome depends on the severity, duration and location of the ischemic insult [9]. Thus, it is crucial to monitor intraoperative ischemia to reverse it immediately, with the aim of minimizing the severe ischemic lesions.

There are various monitoring methods with its advantages and disadvantages, and we will try to go over them.

Awake patient

The most reliable monitoring of cerebral ischemia is the continuous assessment of neurocognitive function in the awake patient, given the sensitivity of brain tissue even to very brief periods of ischemia and hypoxia [10,11]. This type of monitoring requires us to carry out the operation under local or regional anaesthesia. Monitoring will include neurological status, orientation, language, muscle strength, sensation and calculation. The main advantage of this monitor is its sensitivity and specificity, even resulting in a cohort study more sensitive and specific identifying candidates for shunting than the electroencephalography (EEG) and the stump pressure [12,13].

Electroencephalography

In an episode of ischemia, the waves are affected appearing as a slowdown, attenuation or loss of signal, however, they can also be affected by hypothermia or anaesthetic agents. Furthermore electroencephalography it detects only processes in the cerebral cortex and may be affected due to previous stroke. To this is added, the complexity of this technique, causing little acceptance in routine practice [16]. Despite this, it has been reported as reliable predicting the need for arterial shunt in certain studies [14].

The BIS as processed EEG for detection of ischemia has not shown sufficient evidence, generating even paradoxical values during carotid clamping [15,16].

Somatosensory and motor evoked potentials

Somatosensory evoked potentials (SSEP) measure the brain's response to peripheral stimulation through somatosensory pathways. Thus, improve over the prior technique the ability to monitor deeper structures than the cerebral cortex [17]. A reduction of over 50% amplitude and / or a prolongation of latency above 10% are considered significant [18].

Using transcranial motor evoked potentials (tcMEP) has also been postulated as a tool for detecting ischemia, considered clinically significant an increase greater than 50% in amplitude, or presence / absence of the same. Several studies conclude that they are useful as adjuncts to other monitoring methods [16].

Note that both types of evoked potentials study only the integrity of their own way, so that they would leave the other structures unmonitored.

Transcranial doppler

This tool measures the velocity of blood flow in cerebral arteries, such as the middle cerebral artery (MCA). Thus, a reduction of flow in the MCA suggests cerebral ischemia, considering that different ranges have been proposed to be considered clinically significant. Besides this, it is the only effective technology in detecting micro-emboli during CEA [19-22].

Carotid stump pressure

This technique involves puncturing the common carotid and pressure measurement at that level after the distal clamping of the external carotid and proximal of the common carotid. In this way, we can evaluate the perfusion pressure which provides the posterior circulation and circulation of the opposite side. The limits defined vary widely among different studies, thus extreme values may be the most useful indicators, this is less than 25 or greater than 50 mmHg. Its sensitivity and specificity are around 75 and 88% respectively. Despite being useful and simple, there is no clear evidence to support its use [16, 25-28].

Near infrared spectroscopy (NIRS)

Cerebral oximetry based on this tool measures oxygen saturation of haemoglobin in arterial, capillary and venous mixed blood (SctO2), which decays due to ischemia from the carotid clamping or balloon inflation in the case of CAS. It has also shown its correlation with EEG changes, transcranial Doppler, carotid stump pressure and postoperative neurological deficits [29,30-33]. The sensitivity of this test ranges from 30 to 100% according to various studies and its specificity between 77 and 98% [29,30-36]. The limit value is not well established, although several studies suggest a decrease of 11.7-25% above baseline value [29,30,34-36]. This method has the advantage of being non-invasive, continuous and easy to interpret but the fact of collecting information of the frontal lobe, behaves as a disadvantage as it leaves the parietal lobe unmonitored, being the most sensitive lobe to ischemia during carotid surgery [34].

Other techniques

Other techniques have been described, such as the lactate difference between artery and jugular vein, the oxygen saturation of venous jugular bulb or xenon-133 techniques with different sensitivities and specificities, but are expensive techniques and complicated to perform and interpret, so they are not accepted in daily clinical practice [37, 38].

Recent publications support the possibility of combining several monitoring systems in order to improve the sensitivity of these methods, such as the combination of EEG and SSEP where the possibility of occurrence of changes during ischemia is 17 times higher than using the same isolation methods [39,40]. Another example published, is the combination of MEP with NIRS [41].

Carotid Shunt

The use of shunt during carotid clamping has also been the subject of several studies. The shunt, on the one hand, ensures correct flow in the distal internal carotid artery during carotid clamping, but on the other hand increases the risk of complications, and difficult the endarterectomy. So there are several publications advocating selective or routine use or non-use. The Cochrane Library has conducted meta-analyses with different upgrades including quasi and randomized trials published until 2013 comparing the use of routine shunting with no shunting or selective shunting and have not found statistically significant results in favour of any of the options, keeping all of them low rates of perioperative ischemic stroke [42]. We should note that they have been described different complications during insertion of the shunt as the possibility of distal carotid dissection, pseudo aneurysm, air or atheromatous distal embolization, acute arterial occlusion, nerve damage, hematoma, infection and even long-term restenosis [43, 44, 45]. The guides belonging to international societies of Vascular Surgery, allow the surgical team to choose the placement or not of the shunt according to professional experience. Zogogiannis et al. [46] conducted a prospective, randomized controlled trial to evaluate the usefulness of NIRS for intra-operative decision of shunting or not, dividing the patients in three groups depending on NIRS's use. Group A for shunt placement or not depending on NIRS values, group B for algorithm development based on NIRS and group C, without NIRS. They found in this study a decrease in the use of shunt with the use of the algorithm, although they found no difference in neurological outcome of patients. We will perform an adaptation of this algorithm as a possible proposal for action.

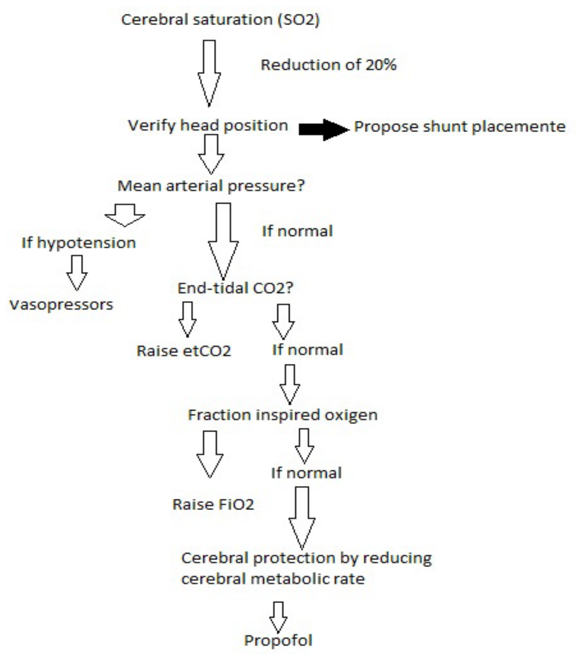

Figure 1: Algorithm for study, use of shunt during carotid clamping.

Anaesthetic technique

Both general anaesthesia (GA) and regional anaesthesia (RA) are equally valid for techniques of CEA and CAS. Ideally, the anaesthesiologist should be comfortable with both options because one of them could have advantages over the other in certain patients because of medical, surgical or patient specific reasons.

In either option, early and complete cooperation of the patient in the neurological examination is important for early diagnosis of ischemic events that have occurred during the development of the intervention. It is also important to maintain hemodynamic avoiding large changes in blood pressure and / or heart rate during the procedure [46].

General anaesthesia

General anaesthesia has advantages and disadvantages. Among the first we have control of ventilation and normocarbia, control of the airway and immobility and patient comfort. Among the latter we found a reduction in sympathetic and baroreceptor activity causing a dose-dependent drop in cardiac output and blood pressure [47], and also as disadvantage, we found a possible masking of neurological complications in the immediate postoperative period given the residual effects of general anesthesia [48].

Citation: Escalona Belmonte JJ, Biteri Martínez De Iturrate A, Guerrero Orriach JL, Ramirez Fernandez A, Ramirez Aliaga M Rodriguez Capitán MJ, et al. (2016) Carotid Stenosis and Anaesthesia. Int J Surg Surgical Porced 1: 105. doi: http://dx.doi.org/10.15344/ijssp/2016/105

During the procedure should be monitored electrocardiogram, heart rate, arterial oxygen saturation by pulse oximetry, exhaled carbon dioxide and invasive blood pressure. The channelling of central venous access is not necessary and may even lead to puncture of the contralateral carotid artery if you attempt to channel the internal jugular vein with the consequences that this would entail, in a patient with such dependence on cerebral blood flow contralateral to the operated side.

Endotracheal intubation is more convenient in patients undergoing general anesthesia TEA due to limited access to the airway during this procedure, even when ASC allows considering the convenience of using a laryngeal mask airway (LMA). The endotracheal tube secures the airway and allows a reliable controlled mechanical ventilation throughout the procedure. It can nevertheless cause sympathetic stimulation and tachycardia, hypertension and myocardial ischemia as a result of this in high risk patients. Moreover cough and hypertension may occur at extubation causing problems in the suture lines, carotid hematoma with compression of the airway in the TEA or a femoral hematoma after ASC [49]. Furthermore, the ipsilateral brain is no longer protected by carotid stenosis against perfusion pressure sudden increases that can occur at this stage. The ML has been associated with less hemodynamic lability during induction [50]. During TEA however there is greater chance of dislodging an LMA that an endotracheal tube, or puncturing the pneumo-tamponade given its closeness to the surgical field.

Normocapnia must be maintained during general anesthesia. Permissive hypercapnia has been proposed as a method for increasing cerebral blood flow and mitigate the possible cerebral ischemia caused by carotid clamping [51]. It were however detected adverse reactions in patients who experienced intracerebral vascular "steal" during hypercapnia [52,53].

The ideal anesthetic should not interfere with cerebral autoregulation during changes in cerebral perfusion pressure. Intravenous anesthetics are used both as volatile agents for maintenance of anesthesia, having like propofol theoretical neuroprotective effects [54]. Propofol and sevoflurane produce a similar reduction in brain metabolism, but the flow and cerebral blood volume are lower when using the first one, but we must clarify that certain studies indicate that in fact propofol and sevoflurane at a concentration up to 1 CAM produced similar reductions in cerebral blood flow and metabolic rate [55,56].

Since both carotid TEA and ASC are usually less than 90 min short procedures it is useful to use short-acting medication as remifentanil for a quick eduction and neurological examination. The use of large doses of long/half-life opioids may produce a slow awakening and impaired neurological evaluation after procedure. Ketamine should be avoided because of its ability to increase brain metabolism, stimulate the release of adrenergic substances and interact with the neurological monitoring [57]. Nitrous oxide (N2O) should be avoided for its ability to increase cerebral metabolic rate and oxygen consumption.

Both TEA and ASC, when AG is done and not depending from the used agent, it is detected a reduction in sympathetic and baroreceptor activity, thus resulting a dose dependent decrease in cardiac output and TA. When TEA is performed, and during carotid clamping, ipsilateral cerebral ischemia may be precipitated due to the reduced blood flow in the carotid and/or plaque rupture with distal embolization. Thus, during clamping, systolic blood pressure should be maintained 20% above the baseline, in order to optimize the collateral cerebral perfusion [58,59]. We must however keep in mind that increasing TA also entails a risk of myocardial ischemia, intracerebral hemorrhage or hematoma in the surgical wound. After unclamping and circulation restore blood pressure may return to baseline [47].

Other circumstances that we must consider is that during the performance of ASC, expansion of the balloon and stent placement in the artery, the endovascular pressure will increase, reducing sympathetic activity and increasing parasympathetic stimulation, which can cause bradycardia and hypotension [60].

Locoregional anesthesia

In recent times and with increased longevity carotid surgery is more frequently indicated in patients with greater comorbidity, and the use of ALR in these patients seems attractive. ALR options are the administration of local anesthetics to cervical epidural level and cervical plexus block (superficial and deep or shallow supplemented with local anesthesia by the surgeon).

The effectiveness of blocking may be increased by associating local anesthetics adjuvants as opioid or adrenergic agents. Opioid and alpha2 receptor agonists such as clonidine improve perioperative and postoperative anelgesia [61]. Clonidine, unlike epinephrine also used for this purpose, does not produce tachycardia and does not mask the adrenergic response during TEA, not damaging the clinical and neurological monitoring 62]. Fentanyl, used in this way, seems to improve the quality of the blockade and its duration in patients undergoing TEA [63].

General anaesthesia

General anaesthesia has advantages and disadvantages. Among the first we have control of ventilation and normocarbia, control of the airway and immobility and patient comfort. Among the latter we found a reduction in sympathetic and baroreceptor activity causing a dose-dependent drop in cardiac output and blood pressure [47], and also as disadvantage, we found a possible masking of neurological complications in the immediate postoperative period given the residual effects of general anesthesia [48].

During the procedure should be monitored electrocardiogram, heart rate, arterial oxygen saturation by pulse oximetry, exhaled carbon dioxide and invasive blood pressure. The channelling of central venous access is not necessary and may even lead to puncture of the contralateral carotid artery if you attempt to channel the internal jugular vein with the consequences that this would entail, in a patient with such dependence on cerebral blood flow contralateral to the operated side.

Endotracheal intubation is more convenient in patients undergoing general anesthesia TEA due to limited access to the airway during this procedure, even when ASC allows considering the convenience of using a laryngeal mask airway (LMA). The endotracheal tube secures the airway and allows a reliable controlled mechanical ventilation throughout the procedure. It can nevertheless cause sympathetic stimulation and tachycardia, hypertension and myocardial ischemia as a result of this in high risk patients. Moreover cough and hypertension may occur at extubation causing problems in the suture lines, carotid hematoma with compression of the airway in the TEA or a femoral hematoma after ASC [49]. Furthermore, the ipsilateral brain is no longer protected by carotid stenosis against perfusion

pressure sudden increases that can occur at this stage. The ML has been associated with less hemodynamic lability during induction [50]. During TEA however there is greater chance of dislodging an LMA that an endotracheal tube, or puncturing the pneumo-tamponade given its closeness to the surgical field.

Normocapnia must be maintained during general anesthesia. Permissive hypercapnia has been proposed as a method for increasing cerebral blood flow and mitigate the possible cerebral ischemia caused by carotid clamping [51]. It were however detected adverse reactions in patients who experienced intracerebral vascular "steal" during hypercapnia [52,53].

The ideal anesthetic should not interfere with cerebral autoregulation during changes in cerebral perfusion pressure. Intravenous anesthetics are used both as volatile agents for maintenance of anesthesia, having like propofol theoretical neuroprotective effects [54]. Propofol and sevoflurane produce a similar reduction in brain metabolism, but the flow and cerebral blood volume are lower when using the first one, but we must clarify that certain studies indicate that in fact propofol and sevoflurane at a concentration up to 1 CAM produced similar reductions in cerebral blood flow and metabolic rate [55,56].

Since both carotid TEA and ASC are usually less than 90 min short procedures it is useful to use short-acting medication as remifentanil for a quick eduction and neurological examination. The use of large doses of long/half-life opioids may produce a slow awakening and impaired neurological evaluation after procedure. Ketamine should be avoided because of its ability to increase brain metabolism, stimulate the release of adrenergic substances and interact with the neurological monitoring [57]. Nitrous oxide (N2O) should be avoided for its ability to increase cerebral metabolic rate and oxygen consumption.

Both TEA and ASC, when AG is done and not depending from the used agent, it is detected a reduction in sympathetic and baroreceptor activity, thus resulting a dose dependent decrease in cardiac output and TA. When TEA is performed, and during carotid clamping, ipsilateral cerebral ischemia may be precipitated due to the reduced blood flow in the carotid and/or plaque rupture with distal embolization. Thus, during clamping, systolic blood pressure should be maintained 20% above the baseline, in order to optimize the collateral cerebral perfusion [58,59]. We must however keep in mind that increasing TA also entails a risk of myocardial ischemia, intracerebral hemorrhage or hematoma in the surgical wound. After unclamping and circulation restore blood pressure may return to baseline [47].

Other circumstances that we must consider is that during the performance of ASC, expansion of the balloon and stent placement in the artery, the endovascular pressure will increase, reducing sympathetic activity and increasing parasympathetic stimulation, which can cause bradycardia and hypotension [60].

Locoregional anesthesia

In recent times and with increased longevity carotid surgery is more frequently indicated in patients with greater comorbidity, and the use of ALR in these patients seems attractive. ALR options are the administration of local anesthetics to cervical epidural level and cervical plexus block (superficial and deep or shallow supplemented with local anesthesia by the surgeon).

The effectiveness of blocking may be increased by associating local anesthetics adjuvants as opioid or adrenergic agents. Opioid and alpha2 receptor agonists such as clonidine improve perioperative and postoperative anelgesia [61]. Clonidine, unlike epinephrine also used for this purpose, does not produce tachycardia and does not mask the adrenergic response during TEA, not damaging the clinical and neurological monitoring 62]. Fentanyl, used in this way, seems to improve the quality of the blockade and its duration in patients undergoing TEA [63].

1. **Cervical epidural**

A widely described technique which provides good conditions for surgery but with significant risks (air embolism, hypotension, bradycardia, respiratory depression, dural puncture and vascular puncture), so it must be done by expert hands. It provides surface plexus and brachial plexus part sensory block.

The effectiveness of blocking may be increased by associating local anesthetics adjuvants as opioid or adrenergic agents. Opioid and alpha2 receptor agonists such as clonidine improve perioperative and postoperative anelgesia [61]. Clonidine, unlike epinephrine also used for this purpose, does not produce tachycardia and does not mask the adrenergic response during TEA, not damaging the clinical and neurological monitoring 62]. Fentanyl, used in this way, seems to improve the quality of the blockade and its duration in patients undergoing TEA [63].

2. Cervical plexus blockage

Cervical plexus covers areas from the back of the head to the collarbone, obtaining an adequate anesthesia for carotid surgery. There may however be not anesthetized areas in the surgical field by superimposing of dermatomes, which could demand surgeon infiltration. In addition, carotid sheath receives innervation of cranial nerves IX and X, and local infiltration is needed to avoid discomfort coming from artery section [59].

There are descriptions of superficial or deep cervical plexus blocking, deep or of a given combination of both. This last option is widely described in the literature but there is evidence that surface blocking with additional infiltration of the incision and sedation is equally effective [64].

Deep plexus blockade is achieved by injections at C2, C3 and C4 levels, or using a single injection at C3 level. Patient lies supine, slightly upright with head turned to the contralateral side, the skin being disinfected and cervical transverse processes palpated, usually 1 cm behind the posterior edge of the sternocleidomastoid muscle and 1-2 cm deep. Puncture is performed at C3 level, located at cricoid cartilage level, perpendicular to the skin with slight slope flow to prevent an intrathecal puncture. Patient may report paresthesia below his shoulder during the process.

Superficial plexus blockade is technically simpler and has fewer complications, even when theoretically it may be insufficient to relax the neck muscles, thus worsening dissection of the deep cervical musculature. It is achieved by infiltrating local anesthetic along the posterior edge of the sternocleidomastoid muscle. Local infiltration of the lower edge blocking afferent facial nerve branches can reduce the pain which is associated with a prolonged use of the cephalic retractor in the incision site.

It has been defined the "intermediate" blocking as the injection of local anesthetic between superficial and deep cervical fascias on the grounds that the last one is permeable [65]. It has the advantage of avoiding practical difficulties and complications of deep plexus blockade.

Potential complications associated with cervical plexus block result in a higher conversion rate to AG [66] and we shall briefly review them. The vertebral artery is very close to the injection target site (0.5 cm below the end of the transverse process in the spinal canal) and since it gives a direct supply to the brain, only a small amount of local anesthetic injected can cause effects on the CNS. Another possible complication is the unilateral phrenic nerve paralysis, because this is composed of branches of the III, IV and V cervical nerve roots. This paralysis is usually well tolerated unless the patient has a severe lung disease and is dependent of the diaphragmatic function for a correct breathing [67]. Other complications or side effects include local hematoma, dysphonia, dysphagia, stellate ganglion block and Horner syndrome.

Advantages include direct neurological monitoring with the subsequent option to establish appropriate measures in case of neurological impairment, a greater hemodynamic stability, maintenance of cerebral autoregulation and a lower requirement of shunt.

The most significant benefit is the constant neurological patient monitoring to detect cerebral ischemia during ACI clamping which may preview neurological complications. The appearance of new neurological symptoms such as changes in consciousness, speech or muscle function after clamping should alert us of a possible ipsilateral cerebral hypoperfusion which may require a shunt placement. In addition, while patient awake during clamping neurological monitoring, surgeon may place the shunt in a safer way than under general anesthesia indirect monitoring techniques. This way less shunts are used [68]. We must not forget that some patients under ALR don't show an immediate neurological deterioration after clamping, which results in a late shunt insertion which can lead to longer times of ischemia and shunt insertion under suboptimal conditions [69]. We must remember anyway that most LCAs are the result of thromboembolism, not hypoperfusion, occurring during the reperfusion phase. The placement of a shunt, which can occur with ALR or AG does not diminish the incidence of embolic stroke [70].

Another aspect to consider during ALR is the reduction of respiratory complications from the IOT and a lower incidence of postoperative hypertension, which can reduce the incidence of cerebral hyperflow syndrome [70,71]. In addition to this, another possible benefit of local anesthesia would be the increase in systemic blood pressure that occurs after clamping and its effect on the maintenance of cerebral perfusion. We must emphasize that it could also reduce the stress caused by surgery, although other factors (size of the incision, visceral damage and blood loss) affect this response rather than the type of anesthesia [68].

Main disadvantages are eliminating potential neuroprotective effect of general anesthesia, the need for immobility and patient cooperation, a limited access to the airway, possible complications of cervical block (subarachnoid injection, intravascular, epidural, phrenic nerve palsy) and the possibility of having to convert to AG that, particularly under emergency conditions and lack of control, involves a greater risk [47].

Regional techniques are rarely fully effective for carotid TEA. In some patients, carotid disease spreads so high that the incision required extends to tissues innervated by cranial nerves and others sympathetic afferents cause discomfort when handling the carotid sheath. Sometimes, you may find jaw, molars or ear pain, which can be relieved by removing the surgical retractor, supplementing with local anesthesia or performing a mandibular block 25. Other factors that must be considered are pain and anxiety during the procedure under ALR that may increase the risk of myocardial ischaemia, eventually needing short periods of deep sedation for restless or uncomfortable patients, especially at the end of the procedure, using for this different drugs such as midazolam, fentanyl, morphine, propofol or dexmedetomidine. It seems reasonable to use an infusion of remifentanil to manage a predictable and reversible sedoanalgesia during the blockade that allows patient's cooperation. It should be avoided an excessive sedation because it may complicate the assessment of neurological status by producing respiratory depression, hemodynamic instability or altered mental status with a loss of the patient cooperation [59].

General anesthesia with patient cooperation

It is a neurological AG allowing awake patient monitoring. Therefore, it has the advantages of the AG and the ALR, cooperation to take control of neurological functions during surgery and optimal control of the airway with tracheal intubation. TIVA AG is only reduced to the remifentanil necessary dose to allow cooperation and proper endotracheal tube tolerance during clamping. Local anesthesia may be associated to also provide a postoperatively pain relief [72].

New Horizons (dexmedetomidine, ultrasound)

Sonographic techniques can increase safety by reducing the complications of this technique and its importance is increasingly widespread. The advantages of this technique are clear, on the one hand the direct vision of the nerves and adjacent anatomical structures, the needle and the spread of local anesthetic (AL) during the injection, and on the other the ability to detect anatomical abnormalities and reducing the volume of used AL, also achieving a higher patient satisfaction as we managed to mitigate the pain caused by dissection around and inside the carotid sheath, the most common cause of discomfort during surgery [73].

Two-dimensional technology (2D) ultrasound captures a flat image, whereas the three-dimensional (3D) technology can provide information on the entire anatomical region including relationships of adjacent structures seeing the spread of local anesthetic along the nerve and identifying better the adjacent structures such as blood vessels and pleura. In addition, technology 2D ultrasound only provides the "sign of the donut" as a sign of the spread of local anesthesia in a plane, in contrast to 3D images that provide data of 960 degrees on the distribution of local anesthesia around nerve [73].

Dexmedetomidine

Dexmedetomidine, an agonist of α-2 receptors, is an ideal drug as sedation during TEA or ASC conducted under AG or ALR. Due to their sympatholytic effect and the hemodynamic stability which it provides, any adverse hemodynamic response can be controlled during the procedure. In addition there is less respiratory depression and the patient wakes up easily with verbal stimuli and may cooperate for neurological assessment. Although dexmedetomidine can theoretically decrease cerebral blood flow by inducing some degree of vasoconstriction in the brain, evidence suggests that is safe and effective in this context [74-76].

Discussion

Regional anesthesia has been claimed to have a greater advantage in the strict monitoring of patients which allows detecting cerebral ischemia during carotid clamplaje and prevent neurological complications.

GALA is a multicenter study comparing surgery with ALR TEA AG under the hypothesis that there is a faster diagnosis and better prevention of perioperative stroke with ALR. With all the biases that this study presents, it showed that in regard to major perioperative complications, this is stroke, myocardial infarction and death, there is no reason to prefer AG against ALR or vice versa. It also showed no definitive evidence that the choice of a type of anesthesia over another affects the length of hospital stay or quality of life [68]. There are also different studies which indicate no significant differences between AG and AL regarding local bleeding, cranial nerve injury or pulmonary complications. Data relating to the control of blood pressure are mixed, but in general terms it seems AL to be more prone to intraoperative and postoperative hypertension and hypotension, while AG presents a greater tendency to intraoperative hypotension and postoperative hypertension. There are not statistically significant differences between both regarding patient satisfaction [77].

The ideal solution would probably be that surgical and anesthetic teams were competent in both techniques, and that the type chosen anesthesia were based on the medical-surgical and standard conditions of patient anxiety, preference of surgeons and anesthesiologists and routine and availability center resources [68].

Concerning the neurological monitoring multiple techniques to monitor cerebral blood flow directly or indirectly have been used, but none of them has been particularly sensitive for detecting intraoperative cerebral hypoperfusion, so there is no consensus to advise an improvement [77]. Iv medication and volatile anesthetics can alter the interpretation of EEG, SSEPs or MEPs. TIVA anesthesia is preferred when PEs are monitored, because it has minimal impact on the evaluation of the neurological signal. It is preferable to avoid the use of neuromuscular relaxants when using MEPs.

Conclusions

There remains a controversy regarding the use of AG vs. ALR in carotid surgery. Existing studies suggest that there is not any evidence that any of both techniques is better than the other one for this type of surgery, and this demands more randomized controlled trials comparing local with general anesthesia. In addition, the introduction of new techniques such as 3D ultrasound or dexmedetomidine may provide new approaches to this type of intervention. And we also still need a reliable method for monitoring brain even in patients under direct monitoring with ALR.

Competing Interests

The authors declare that they have no competing interests.

References

1. Brott TG, Halperin JL, Abbara S, Bacharach JM, Barr JD, et al. (2011) ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American Stroke Association, American Association of Neuroscience Nurses, American Association of Neurological Surgeons, American College of Radiology, American Society of Neuroradiology, Congress of Neurological Surgeons, Society of Atherosclerosis Imaging and Prevention, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of NeuroInterventional Surgery, Society for Vascular Medicine, and Society for Vascular Surgery. Circulation 124: e54-130.
2. European Stroke Organisation, Tendera M, Aboyans V, Bartelink ML, Baumgartner I, et al. (2011) ESC Guidelines on the diagnosis and treatment of peripheral artery diseases: document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries: the Task Force on the Diagnosis and Treatment of Peripheral Artery Diseases of the European Society of Cardiology (ESC). Eur Heart J 32: 2851-906.
3. Mas JL (2007) Internal carotid artery revascularization. Rev Esp Cardiol 60: 861-871.
4. Liapis CD, Kakisis JD, Kostakis AG (2001) Carotid stenosis: factors affecting symptomatology. Stroke 32: 2782-2786.
5. Inzitari D, Eliasziw M, Gates P, Sharpe BL, Chan RK, et al. (2000) The causes and risk of stroke in patients with asymptomatic internal-carotid-artery stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 342: 1693-1700.
6. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, et al. (2011) Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease. J Vasc Surg 54: e1-e31.
7. Brott TG, Hobson RW, Howard G, Roubin GS, Clark WM, et al. (2010) Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med 363: 11-23.
8. Paraskevas K, Mikhailidis DP, Veith FJ (2012) Comparison of the five 2011 guidelines for the treatment of carotid stenosis. J Vasc Surg 55: 1504-1508.
9. Bramlett HM, Dietrich WD (2004) Pathophysiology of cerebral ischemia and brain trauma: similarities and differences. J Cereb Blood Flow Metab 24: 133-150.
10. Moritz S, Kasprzak P, Arlt M, Taeger K, Metz C, et al. (2007) Accuracy of cerebral monitoring in detecting cerebral ischemia during carotid endarterectomy: a comparison of transcranial Doppler sonography, near-infrared spectroscopy, stump pressure, and somatosensory evoked potentials. Anesthesiology 107: 563-569.
11. Ali AM, Green D, Zayed H, Halawa M, El-Sakka K, et al. (2011) Cerebral monitoring in patients undergoing carotid endarterectomy using a triple assesment technique. Interact cardiovasc Thorac Surg 12: 454-457.
12. Hans SS, Jareunpoon O (2007) Prospective evaluation of electroencephalography, carotid artery stump pressure, and neurologic changes during 314 consecutive carotid edarterectomies performed in awake patients. J Vasc Surg 45: 511-515.
13. Guay J, Kopp S (2013) Cerebral monitoris versus regional anesthesia to detect cerebral ischemia in patients undergoing carotid endarterectomy: a meta-analysis. Can J Anaesth 60: 266-279.
14. Ballotta E, Saladini M, Gruppo M, Mazzalai F, Da Giau G, et al. (2010) Predictors of electroencephalographic changes needing shunting during carotid endarterectomy. Ann Vasc Surg 24:1045-1052.
15. Bonhomme V, Desiron Q, Lemineur T, Brichant JF, Dewandre PY, et al. (2007) Bispectral index profile during carotid cross clamping. J Neurosurg Anesthesiol 19: 49-55.
16. Li J, Shalabi A, Fuhai J, Meng L (2016) Monitoring cerebral ischemia during carotid endarterectomy and stenting. J Biomed Res 3: 31.
17. MacDonald DB (2005) Re: Comparison of monitoring techniques for intraoperative cerebral ischemia. DW Rowed, DA Houlden, LM Burkholder, AB Taylor. Can J Neurol Sci 2004;31(3):347-356. Can J Neurol Sci 32: 131-132.
18. Nwachuku EL, Balzer JR, Yabes JG, Habeych ME, Crammond DJ, et al. (2015) Diagnostic value of somatosensory evoked potential changes during carotid endarterectomy: a systematic review and meta-analysis. JAMA Neurol 72: 73-80.

19. Wolf O, Heider P, Heinz M, Poppert H, Sander D, et al. (2004) Microembolic signals detected by transcranial doppler sonography during carotid endarterectomy and correlation with serial diffusion- weighted imaging. Stroke 34: e373.

20. McCarthy RJ, McCabe AE, Walker R, Horrocks M (2001) The value of transcranial Doppler in predicting cerebral ischaemia during carotid endarterectomy. Eur J Vasc Endovasc Surg 21: 408-412.

21. Skjelland M, Krohg-Sørensen K, Tennøe B, Bakke SJ, Brucher R, et al. (2009) Cerebral microemboli and brain injury during carotid artery endarterectomy and stenting. Stroke 40: 230-234.

22. Horn J, Naylor AR, Laman DM, Chambers BR, Stork JL, et al. (2005) Identification of patients at risk for ischaemic cerebral complications after carotid endarterectomy with TCD monitoring. Eur J Vasc Endovasc Surg 30: 270-274.

23. Moppett IK, Mahajan RP (2004) Transcranial Doppler ultrasonography in anaesthesia and intensive care. Br J Anaesth 93: 710-724.

24. Howell SJ (2007) Carotid endarterectomy. Br J Anaesth 99: 119-131.

25. Calligaro KD, Dougherty MJ (2005) Correlation of carotid artery stump pressure and neurologic changes during 474 carotid endarterectomies performed in awake patients. J Vasc Surg 42: 684-689.

26. Belardi P, Lucertini G, Ermirio D (2003) Stump pressure and transcranial Doppler for predicting shunting in carotid endarterectomy. Eur J Vasc Endovasc Surg 25: 164-167.

27. Aburahma AF, Stone PA, Hass SM, Dean LS, Habib J, et al. (2010) Prospective randomized trial of routine versus selective shunting in carotid endarterectomy based on stump pressure. J Vasc Surg 51: 1133-1138.

28. Jacob T, Hingorani A, Ascher E (2007) Carotid artery stump pressure (CASP) en 1135 consecutive endarterectomias under general anesthesia: an old method that survived the test of times. J Cardiovasc Surg (Torino) 48: 677-681.

29. Rigamonti A, Scandroglio M, Minicucci F, Magrin S, Carozzo A, et al. (2005) A clinical evaluation of near-infrared cerebral oximetry in the awake patient to monitor cerebral perfusion during carotid endarterectomy. J Clin Anesth 17: 426-430.

30. Pedrini L, Magnoni F, Sensi L, Pisano E, Ballestrazzi MS, et al. (2012) Is Near-Infrared Spectroscopy a Reliable Method to Evaluate Clamping Ischemia during Carotid Surgery? Stroke Res Treat 2012: 156975.

31. Shang Y, Cheng R, Dong L, Ryan SJ, Saha SP, et al. (2011) Cerebral monitoring during carotid endarterectomy using near-infrared diffuse Cerebral ischemia during carotid procedure 5 optical spectroscopies and electroencephalogram. Phys Med Biol 56: 3015-3032.

32. Pugliese F, Ruberto F, Tosi A, Martelli S, Bruno K, et al. (2009) Regional cerebral saturation versus transcranial Doppler during carotid endarterectomy under regional anaesthesia. Eur J Anaesthesiol 26: 643-647.

33. Giustiniano E, Alfano A, Battistini GM, Gavazzeni V, Spoto MR, et al. (2010) Cerebral oximetry during carotid clamping: is blood pressure raising necessary? J Cardiovasc Med (Hagerstown) 11: 522-528.

34. Samra SK, Dy EA, Welch K, Dorje P, Zelenock GB, et al. (2000) Evaluation of a cerebral oximeter as a monitor of cerebral ischemia during carotid endarterectomy. Anesthesiology 93: 964-970.

35. Mille T, Tachimiri ME, Klersy C, Ticozzellic G, Bellinzona G, et al. (2004) Near infrared spectroscopy monitoring during carotid endarterectomy: which threshold value is critical? Eur J Vasc Endovasc Surg 27: 646-650.

36. Ritter JC, Green D, Slim H, Tiwari A, Brown J, et al. (2011) The role of cerebral oximetry in combination with awake testing in patients undergoing carotid endarterectomy under local anaesthesia. Eur J Vasc Endovasc Surg 41: 599-605.

37. Moritz S, Kasprzak P, Woertgen C, Taeger K, Metz C, et al. (2008) The accuracy of jugular bulb venous monitoring in detecting cerebral ischemia in awake patients undergoing carotid endarterectomy. J neurosurg Anesthesiol 20: 8-14.

38. Latchaw RE, Yonas H, Hunter GJ (2003) Guidelines and recommendations for perfusión maging in cerebral ischemia: A scientific statement for healthcare professionals by the writing group of perfusión imaging, form the Council on Cardiovasc Radiology of the American Heart Association. Stroke 34: 1084-1104 .

39. Thiagarajan K, Cheng HL, Huang JE, Natarajan O, Crammond et al. (2015) Is Two Really Better Than One? Examining the Superiority of Dual Modality Neurophysiological Monitoring During Carotid Endarterectomy: A Meta-Analysis. World Neurosurg 84: 1941-1949.

40. Malek BN, Mohrhaus CA, Sheth AK (2011) Use of multi-modality intraoperative monitoring during carotid endarterectomy surgery: a case study. Am J Electroneurodiagnostic Technol 51: 42-53.

41. Uchino H, Nakamura T, Kuroda S, Houkin K, Murata J, et al. (2012) Intraoperative Dual Monitoring During Carotid Endarterectomy Using Motor Evoked Potentials and Near-Infrared Spectroscopy. World neurosurg 78: 651-657.

42. Rerkasem K, Rothwell PM (2009) Routine or selective carotid artery shunting for carotid endarterectomy (and different methods of monitoring in selective shunting). Cochrane Database Syst Rev 4: CD000190.

43. Katsuno M, Tanikawa R, Hashimoto M, Matsuno A (2015) Distal internal carotid artery dissection due to the carotid shunt during carotid endarterectomy. Br J Neurosurg 29 : 862-864.

44. Varetto G, Castagno C, Quaglino S, Garneri P, Benintende E, et al. (2015) Successful management with 2 overlapping bare stents for post-carotid endarterectomy carotid pseudoaneurysm secondary to carotid shunt. Ann Vasc Surg 29: 594.

45. Aburahma AF, Mousa AY, Stone PA (2011) Shunting during carotid endarterectomy. J Vasc Surg 54: 1502-1510.

46. Livingstone K, Raju I (2011) Anesthesia for carotid surgery. Anesthesia and Intesive Care Medicine 17: 249-252.

47. Raju I, Fraser K (2011) Anaesthesia for carotid surgery. Anaesthesia and Intensive Care Medicine 14: 208-211.

48. Unic-Stojanovic D, Babic S, Neskovic V (2013) General versus regional anesthesia for carotid endarterectomy. J Cardiothorac Vasc Anesth 27: 1379-1383.

49. Augoustides JG, Groff BE, Mann DG, Johansson JS (2007) Difficult airway management after carotid endarterectomy: utility and limitations of the Laryngeal Mask Airway. J Clin Anesth 19: 218-221.

50. Marietta DR, Lunn JK, Ruby EI, Hill GE (1998) Cardiovascular stability during carotid endarterectomy: endotracheal intubation versus laryngeal mask airway. J Clin Anesth 10: 54-57.

51. Thompson JE, Talkington CM (1976) Carotid endarterectomy. Ann Surg 184:1.

52. Waltz AG, Sundt TM Jr, Michenfelder JD (1972) Cerebral blood flow during carotid endarterectomy. Circulation 45: 1091-1096.

53. Sundt TM Jr, Sharbrough FW, Anderson RE, Michenfelder JD (1974) Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy. J Neurosurg 41: 310-320.

54. Trop D (1987) Con: carotid endarterectomy: general is safer than regional anesthesia. J Cardiothorac Anesth 1: 483-488.

55. Kaisti KK, Långsjö JW, Aalto S, Oikonen V, Sipilä H, et al. (2003) Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. Anesthesiology 99: 603-613.

56. Mielck F, Stephan H, Weyland A, Sonntag H (1999) Effects of one minimum alveolar anesthetic concentration sevoflurane on cerebral metabolism, blood flow, and CO2 reactivity in cardiac patients. Anesth Analg 89: 364-369.

57. Apinis A, Sehgal S, Leff J (2014) Intraoperative management of carotid endarterectomy. Anesthesiol Clin 32: 677-698.

58. Allain R, Marone LK, Meltzer J, Jeyabalan G (2005) Carotid endarterectomy. Int Anesthesiol Clin 43: 15-38.

59. Yastrebov K (2004) Intraoperative management: carotid endarterectomies. Anesthesiol Clin North America 22: 265-287, vi-vii.

60. Mangin L, Medigue C, Merle JC, Macquin-Mavier I, Duvaldestin P, et al. (2003) Cardiac autonomic control during balloon carotid angioplasty and stenting. Can J Physiol Pharmacol 81: 944-951.

61. Danelli G, Nuzzi M, Salcuni PF, Caberti L, Berti M, et al. (2006) Does clonidine 50 microg improve cervical plexus block obtained with ropivacaine 150 mg for carotid endarterectomy? A randomized, double-blinded study. J Clin Anesth 18: 585-588.
62. Schneemilch CE, Bachmann H, Ulrich A, Elwert R, Halloul Z, et al. (2006) Clonidine decreases stress response in patients undergoing carotid endarterectomy under regional anesthesia: a prospective, randomized, double-blinded, placebo-controlled study. Anesth Analg 103: 297-302.
63. Sindjelic RP, Vlajkovic GP, Davidovic LB, Markovic DZ, Markovic MD (2010) The addition of fentanyl to local anesthetics affects the quality and duration of cervical plexus block: a randomized, controlled trial. Anesth Analg 11: 234-237.
64. Guay J (2008) Regional anesthesia for carotid surgery. Curr Opin Anaesthesiol 21: 638-644.
65. Pandit JJ, Dutta D, Morris JF (2003) Spread of injectate with superficial cervical plexus block in humans: an anatomical study. Br J Anaesth 91: 733-735.
66. Pandit JJ, Satya-Krishna R, Gration P (2007) Superficial or deep cervical plexus block for carotid endarterectomy: a systematic review of complications. Br J Anaesth 99: 159-169.
67. Allain R, Marone LK, Meltzer J, Jeyabalan G (2005) Carotid endarterectomy. Int Anesthesiol Clin 43: 15-38.
68. GALA Trial Collaborative Group, Lewis SC, Warlow CP, Bodenham AR, Colam B, et al. (2008) General anaesthesia versus local anaesthesia for carotid surgery (GALA): a multicentre, randomised controlled trial. Lancet 372: 2132-2142.
69. McCleary AJ, Maritati G, Gough MJ (2001) Carotid endarterectomy; local or general anaesthesia? Eur J Vasc Endovasc Surg 22: 1-12.
70. Eibes TA, Gross WS (2000) The influence of anesthetic technique on perioperative blood pressure control after carotid endarterectomy. Am Surg 66: 641-647.
71. Eibes TA, Gross WS (2000) The influence of anesthetic technique on perioperative blood pressure control after carotid endarterectomy. Am Surg 66: 641-647.
72. Scuderi PE, Prough DS, Davis CH Jr, Balestrieri FJ, McWhorter JM, et al. 1989) The effects of regional and general anesthesia on blood pressure control after carotid endarterectomy. J Neurosurg Anesthesiol 1: 41-45.
73. Bevilacqua S, Romagnoli S, Ciappi F, Lazzeri C, Gelsomino S, et al. (2009) Anesthesia for carotid endarterectomy: the third option. Patient cooperation during general anesthesia. Anesth Analg 108: 1929-1936.
74. Ciccozzi A, Angeletti C, Guetti C, Pergolizzi J, Angeletti PM, et al. (2014) Regional anaesthesia techniques for carotid surgery: the state of art. J Ultrasound 17: 175-183.
75. Prielipp RC, Wall MH, Tobin JR, Groban L, Cannon MA, et al. (2002) Dexmedetomidine-induced sedation in volunteers decreases regional and global cerebral blood flow. Anesth Analg 95: 1052-1059.
76. Bekker AY, Basile J, Gold M, Riles T, Adelman M, et al. (2004) Dexmedetomidine for awake carotid endarterectomy: efficacy, hemodynamic profile, and side effects. J Neurosurg Anesthesiol 16: 126-135.
77. Huncke TK, Adelman M, Jacobowitz G, Maldonado T, Bekker A (2010) A prospective, randomized, placebo-controlled study evaluating the efficacy of dexmedetomidine for sedation during vascular procedures. Vasc Endovascular Surg. 44: 257-261.
78. Rerkasem K, Rothwell PM (2008) Local versus general anaesthesia for carotid endarterectomy. Cochrane Database Syst Rev 8: CD000126.