

Cerebral oxygen saturation is improved by xenon anaesthesia during carotid clamping

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ABSTRACT

Introduction: The cerebral protective effect of xenon anesthesia could be of interest during carotid surgery. The purpose of this study was to compare the effects of xenon on cerebral oxygen saturation with those of propofol during carotid clamping.

Methods: After approval of Research Ethics Board and patient informed consent, 74 patients scheduled for carotid endarterectomy were enrolled. Patients were not randomized but were well matched by preoperative characteristics. Patients in the Xenon group were the ones scheduled for surgery in the operative theater equipped with the xenon anesthesia system. Anesthesia was started with a target control infusion of propofol and remifentanyl. Patients were then divided into the control group (37 patients) with anesthesia maintained with target control infusion propofol and remifentanyl and the Xenon group with anesthesia maintained with xenon (target inspired concentration of 60%) and target control infusion remifentanyl. Remifentanyl and xenon or propofol were stopped at the end of skin closure.

Results: A cerebral oxygen saturation decrease below 55% was less frequently observed in the Xenon group during carotid cross-clamping (7/37 patients vs 15/37; $p = 0.01$). Compared with values observed before clamping, the decrease in cerebral oxygen saturation during clamping was significantly less important in the Xenon group ($12 \pm 11\%$ vs $17 \pm 14\%$, $p = 0.04$). Blood pressure and heart rate were not different between groups during carotid clamping.

Conclusions: This pilot study suggests that xenon anesthesia may be associated to higher cerebral oxygen saturation values when compared to propofol anesthesia during cross-clamping for carotid endarterectomy.

Keywords: carotid surgery, general anesthesia, TCI, propofol, remifentanyl, xenon, near-infrared spectrometry.

INTRODUCTION

Providing an optimal cerebral perfusion is a major objective during carotid surgery. Xenon's effects on cerebral perfusion and metabolism are quite complex. Its neuroprotective effect is caused by enhanced ischemic tolerance.

A second advantage theoretically brought by xenon is cardio protection. Xenon anesthesia allows a fast recovery of cognitive functions (1) and provides cerebral protective effects in experimental models (2, 3). Its use might be particularly interesting in patients with poor cerebral perfusion (4). These advantages suggest that xenon may be an attractive alternative to the various techniques of general or regional anesthesia (5, 6) usually proposed during carotid surgery.

Measurement of frontal cortex cerebral oxygen saturation (SrcO₂) can allow and

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indirect evaluation of the cerebral perfusion quality. All elements that modify cerebral perfusion can explain a decrease of SrcO_2 . Murkin et al. reported a good correlation between the modifications of SrcO_2 during the clamping and the modifications of other techniques of monitoring such as the transcranial Doppler (7). A decline of more than 12% of SrcO_2 or an absolute value lower than 55%, is predictive of cerebral ischemia (detected by electroencephalogram (EEG) or somesthetic evoked potentials) or of modifications of the transcranial Doppler or the stump pressure which can incite the use of a shunt (7-9).

METHODS

This cohort study was conducted in a single center. Both anesthetic regimens studied are administered on a daily basis to patients undergoing carotid surgery in our hospital. No therapeutic intervention other than those already used was necessary for the study. This protocol was approved by our Research Ethics Board and patients were enrolled after their informed consent was obtained.

A preliminary study estimated at 60% the number of operated patients presenting a decrease below 55% of SrcO_2 during carotid clamping under conventional general anesthesia. In order to observe a decrease of two third in the number of patients developing a decrease below 55%, with risks $\alpha = 5\%$ and $\beta = 10\%$, 68 patients had to be included in the study.

All patients were anesthetized by the same anesthetist (GG). Seventy-four consecutive patients scheduled for surgery of carotid stenosis were included. Patients with uncontrolled hypertension, and/or severe chronic obstructive pulmonary disease (COPD), and/or hypoxemia at rest, were excluded.

Treatments taken chronically by the patients, were continued or not, according to the latest French guidelines (10): continuation of cardiovascular treatments except angiotensin-converting-enzyme inhibitor and/or angiotensin II receptor antagonist (AI-IR1A) which were discontinued 48 hours before surgery, continuation of antidiabetic agents except metformin. Treatment by clopidogrel was stopped 8 days before surgery and replaced by aspirin if necessary and except for specific indication. For all patients, monitoring included an invasive measure of blood pressure, monitoring of pulsed pressure index (VPP®, Philips Medical Systems, Eindhoven, The Netherlands), bispectral analysis of the EEG (BIS Aspect®, Covidien, Elancourt, France) and SrcO_2 (NIRS®, Covidien, Elancourt, France). BIS sensor was positioned on the opposite side of the operated carotid, and NIRS sensor was positioned on operated carotid side.

All patients initially received propofol (Schnider model) and remifentanyl (Minto model) via a target controlled infusion (TCI) (11-13) (Primea®, Fresenius-Vial, Brezins, France) and cisatracurium. Ventilation parameters included a positive end-expiratory pressure (Peep) at 5 mmHg and a FIO_2 at 35% with a closed circuit. Patients were then divided into 2 groups according to the drug administered for the maintenance of anesthesia:

- 1) in Control group, anesthesia was maintained with TCI propofol and remifentanyl;
- 2) in Xenon group, anesthesia was maintained with xenon (target inspired concentration of 60%) and TCI remifentanyl.

Propofol was discontinued when inspired concentration of xenon reached 40%. Patients were not randomized. Patients in the Xenon group were the ones scheduled for surgery in the operative theater equipped with the xenon anesthesia system (Taema

Félix Dual[®], Air Liquide Santé International, Paris, France). Postoperative analgesia was initiated 30 minutes before skin closure and included paracetamol 1g and morphine 0.05 mg/kg. Maintenance of anesthesia was stopped at the end of skin closure.

In both groups of patients, anesthesia was conducted with the following objectives:

- a) hemodynamic stability with the smallest variation possible in systolic blood pressure compared with preoperative value. In case of hypotension (decreased of more than 20% in comparison with preoperative value lasting more than 3 minutes) associated with a high VPP index, patients received a volume expansion (hydroxyethylstarch (HES) 130/0.4 Voluven[®], Fresenius-Kabi, Brezins, France). In case of low VPP index, patients received ephedrine using titration;
- b) BIS was maintained between 40 and 50 (14);
- c) trachea was extubated at the end of surgery in patients free of neurological defect.

Cerebral oxymetry was continuously recorded, but was not taken into account in a decision-making algorithm. SrcO₂ data were analyzed after the end of patients' enrollment. Per routine, and at the discretion of the surgeon, a shunt was performed prior to the carotid clamping in case of a controlateral carotid occlusion and/or a non functional circle of Willis. An angiogram was performed to assess the success of the carotid endarterectomy after the release of a cross-clamp.

In patients with a blood pressure higher than 180 mmHg after the carotid artery has been declamped, remifentanil concentration was increased and/or nicardipine was administered.

Patients were hospitalized in Intensive Care Unit until day 1 after surgery. Systolic

blood pressure was maintained below 160 mmHg, heart rate below 80 b/min, using nicardipine and/or atenolol and/or administration of pre existing chronic treatments as needed. An electrocardiography (ECG) was performed at recovery and on postoperative day 1 and day 2. A measurement of cardiac troponin I (cTnI) was systematically done 6 hours after the end of surgery, and on postoperative day 1 and day 3.

The purpose of this study was to compare the effects of xenon on SrcO₂ with those of propofol using TCI. The secondary endpoints were awaking delay, postoperative adverse events, and cardiovascular follow-up until the patient was discharged from the hospital.

Statistical analysis between SrcO₂, automatically recorded during procedures, was retrospectively studied for each patient. Statistical analysis included a Student's t test for continuous variables, or a chi square test for ordinal variables.

RESULTS

Demographics data, American Society of Anesthesiologists score (ASA) physical status, chronic treatments and intraoperative data were not different between the 2 groups (*Table 1*).

At clamping, a decrease of SrcO₂ below 55% was less frequently observed in the Xenon group (7/37 patients vs 15/37; $p=0.01$) (*Table 2*). Decrease in SrcO₂ at clamping was significantly less important in Xenon group (average variation: $12\pm 11\%$ vs $17\pm 14\%$, $p=0.04$).

After anesthesia induction, no hypotension was noted in patients treated with xenon, while this observation was almost constant with propofol and remifentanil TCI. Systolic blood pressure (143 ± 13 vs 143 ± 15 mmHg), PetCO₂ (32 ± 2 vs 31 ± 3 mmHg) and BIS values (39 ± 5 vs 43 ± 10)

Table 1 - Demographic and perioperative characteristics.

	Control group N = 37	Xenon group N = 37	p value
Age (years) (mean \pm SD)	72 \pm 11	74 \pm 8	> 0.05
Sex ratio M/F (n)	26/11	31/6	> 0.05
ASA I-II / ASA III (n)	27/10	19/18	> 0.05
Modified Lee' score* (mean \pm SD)	1.3 \pm 0.5	1.5 \pm 0.7	> 0.05
Hypertension (n (%))	27 (74)	28 (76)	> 0.05
Coronary artery disease (n (%))	12 (32)	17 (46)	> 0.05
Left cardiac failure (n (%))	0	1 (4)	> 0.05
COPD (n (%))	4 (12)	6 (16)	> 0.05
Estimated GFR < 60 ml/min (n (%))*	7 (18)	13 (35)	> 0.05
Diabetes mellitus (n (%))	16 (44)	12 (32)	> 0.05
Antiplatelet agents (n (%))	35 (94)	37 (100)	> 0.05
β -blockers (n (%))	12 (33)	13 (35)	> 0.05
Statins (n (%))	27 (74)	30 (81)	> 0.05
ACEI or A2A (n (%))	17 (47)	20 (54)	> 0.05
Previous stroke or TIA (n (%))	38	49	> 0.05
Stroke with neurologic sequel (n (%))	6 (15)	5 (14)	> 0.05
Contralateral thrombosis (n (%))	4 (12)	2 (5)	> 0.05
2 nd side surgery (n (%))	3 (9)	3 (9)	> 0.05
Surgery duration (min) (mean \pm SD)	79 \pm 42	89 \pm 30	> 0.05
Clamping duration (min) (mean \pm SD)	27 \pm 12	30 \pm 15	> 0.05
Shunt (n (%))	3 (9)	6 (16)	> 0.05

*Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, Sugarbaker DJ, Donaldson MC, Poss R, Ho KK, Ludwig LE, Pedan A, Goldman L. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999; 100: 1043-9.
 ASA = American Society of Anesthesiologists score; GFR = glomerular filtration rate; COPD = chronic obstructive pulmonary disease; ACEI = converting enzyme inhibitor; A2A = angiotensin II antagonist; SD = standard deviation; TIA = transient ischemic attack.

were identical in the two groups during carotid clamping. Xenon use was associated with an increase in blood pressure after carotid clamping.

The same level of blood pressure during cross-clamping was obtained with less volume expansion (850 \pm 240 vs 1020 \pm 315 ml, $p < 0.01$) and a smaller dose of ephedrine (9 \pm 11 mg vs 16 \pm 18 mg, $p = 0.04$) in Xenon group (Tables 2-4). Heart rate was significantly lower during all the procedure in the Xenon group (55 \pm 10 vs 65 \pm 14 at clamping, $p < 0.01$) (Table 3). All the patients in the Xenon group were awakened

and trachea was extubated in the operating room (Table 5). In comparison, eleven patients in the Control group were extubated after a 10 to 50 minutes delay (timing started after remifentanyl administration was stopped, and when the effect-site concentration reached 0.5 ng/ml). Three patients in the Control group developed excitement or disorders of consciousness, all of which resolved within the first postoperative hour. Four patients presented a neurological transient stroke (contralateral hemiparesis), all regressive within one hour (3 in the Xenon group, 1 in the

Table 2 - Arterial blood pressure, heart rate and cerebral oxygen saturation in the Xenon and in the Control group.

	Before anesthesia induction	Before skin incision	Carotid cross-clamping	Carotid declamping	End of surgery
Systolic blood pressure					
Control group	150 ± 16	112 ± 20	143 ± 13	117 ± 15	124 ± 20
Xenon group	157 ± 19	111 ± 21	143 ± 15	132 ± 16*	135 ± 19**
Mean arterial pressure					
Control group	99 ± 11	78 ± 14	95 ± 11	78 ± 11	82 ± 12
Xenon group	104 ± 13	76 ± 15	93 ± 12	86 ± 13***	90 ± 16**
Heart rate					
Control group	64 ± 10	61 ± 10	65 ± 14	62 ± 12	61 ± 12
Xenon group	64 ± 12	60 ± 10	55 ± 10***	55 ± 9***	57 ± 10
Cerebral oxygen saturation					
Control group	68 ± 8	68 ± 9	60 ± 13	66 ± 10	66 ± 10
Xenon group	68 ± 7	67 ± 7	62 ± 8	67 ± 10	68 ± 8
Data are expressed as mean ± SD if not otherwise stated. *p < 0.01; **p = 0.02; SD = standard deviation.					

Control group). One patient in the Control group presented a worsening of a previous cognitive disorder (Table 5). As shown in the Table 6, no correlation seems to exist between these neurological abnormalities

and the symptomatic characteristics of the operated lesion, the use of a shunt, and SrcO₂. No patient developed significant postoperative cardiovascular and/or hemorrhagic complications, and/or required

Table 3 - Reduction in cerebral oxygen saturation, hemodynamic data, drugs and volume requirements in the Xenon and Control group.

	Control group N = 37	Xenon group N = 37	p value
SrcO ₂ <55% (n patients) at XC	15	7	P = 0.01
Decrease of SrcO ₂ more than 12% at XC			
- compared to basal value (n/%)	17 (46)	9 (24)	> 0.05
- compared to preXC value (n/%)	20 (54)	14 (38)	> 0.05
Variation of SrcO ₂ at XC			
- compared to basal value (%) (mean ± SD)	13 ± 17	8 ± 11	> 0.05
- compared to preXC value (%) (mean ± SD)	17 ± 14	12 ± 11	0.042
Delay between XC and max SBP (min)(mean ± SD)	4 ± 7	18 ± 22	< 0.01
Ephedrine (mg) (mean ± SD)	16 ± 18	9 ± 11	0.040
Max remifentanyl concentration (ng/ml) (mean ± SD)	3.2 ± 1.2	4.5 ± 2	< 0.01
Intraoperative nicardipin (n (%))	1 (3)	8 (22)	= 0.02
Volume expansion (ml) (mean ± SD)	1020 ± 315	850 ± 240	< 0.01
SrcO ₂ = cerebral oxygen saturation; XC = carotid cross-clamping; SBP = systolic blood pressure; SD = standard deviation.			

Table 4 - Cerebral oxygen saturation values over time.

	Control group N = 37	Xenon group N = 37	p value
Before anesthesia	68 ± 8	68 ± 7	> 0.05
Before skin incision	68 ± 9	67 ± 7	> 0.05
XC + 2 minutes	60 ± 13	62 ± 8	> 0.05
XC + 5 minutes	59 ± 11	62 ± 8	0.02
XC + 10 minutes	59 ± 12	64 ± 6	> 0.05
UC	66 ± 9	67 ± 6	> 0.05
End of surgery	66 ± 10	68 ± 8	> 0.05

Data are expressed as mean ± SD; XC = carotid cross-clamping; UC = carotid declamping; SD = standard deviation.

Table 5 - Postoperative data.

	Control group N = 37	Xenon group N = 37	p value
Patients extubated at the end of surgery (n (%))	26 (70)	37 (100)	< 0.01
Extubation delay (min)* (mean ± SD)	25 ± 13	0	-
Nausea or vomiting (n (%))	1 (3)	4 (11)	> 0.05
Shivering n (%)	0	3 (8)	< 0.05
Hypertension (n (%))	23 (63)	25 (68)	> 0.05
Nicardipin (mg) (mean ± SD)	1.9 ± 2.3	1.6 ± 1.4	> 0.05
Atenolol (mg) (mean ± SD)	0.7 ± 2.0	0.6 ± 1.4	> 0.05
Transient ischemic attack (n)	1	3	> 0.05
Stroke (n)	0	0	> 0.05
ECG abnormalities (n)	0	0	> 0.05
Cardiac troponin I peak > 0.05 mcg/L (n)	1	2	> 0.05
Discharge (days) (mean ± SD)	4 ± 2	4 ± 1	> 0.05
Mortality	0	0	0.9

*from remifentanyl discontinuation *(site-effect concentration < 0.5 ng/ml) in 11 patients who were not extubated at the end of surgery. SD = standard deviation.

Table 6 - Clinical characteristics of patients with postoperative transient ischemic attack (N = 4) or other postoperative neurologic abnormalities (N = 4).

	Group	Σ	CCT	SP	SP pulsatility	Min SrcO2	SrcO2 decrease	Shunt	Stroke	Recovery
1	Xenon	Yes	No	21	No	73	6	yes	½ paresis	30 min
2	Xenon	Yes	No	10	Yes	23	65	yes	½ paresis	60 min
3	Xenon	Yes	No	32	Yes	49	14	no	½ paresis	180 min
4	Control	Yes	No	Nd	-	53	18	no	Stupor	60 min
5	Control	No	No	71	Yes	38	42	no	Excitement	15 min
6	Control	Yes	No	Nd	-	75	4	yes	½ paresis	50 min
7	Control	No	No	67	Yes	58	17	no	Memory troubles	No
8	Control	yes	No	Nd	-	73	1	no	Stupor	60 min

Σ= Transient or permanent preoperative stroke; CCT = Controlateral carotid thrombosis; SP = Stump pressure; SrcO2 = cerebral oxygen saturation; nd = not done.

a reoperation. All the patients were discharged free of neurologic abnormalities from the vascular unit.

DISCUSSION

The main result of the study is that xenon anesthesia is associated to a smaller decrease in ScvO_2 during carotid clamping and that a decrease below 55% was less frequently observed in the Xenon group. These results add to the experimental literature showing a cerebral protective effect of xenon anesthesia (15).

Patients in the Xenon group had more hemodynamic stability. After induction of general anesthesia, the usual hypotension is related to the well described vasodilator effect of the conventional agents such as propofol. In patients undergoing vascular surgery, the hypotension is exacerbated by chronic hypertension and use of chronic antihypertensive treatments. This hypotension is readily managed by volume expansion and administration of vasoactive agents. Xenon use was associated with higher systolic and mean blood pressures after carotid declamping and at the end of surgery (Table 2). Even if the hypertension is partially artificial and partially related to the administration of vasoactive agents, it is necessary to remember that this increase is quasi-constant, appears in the first 2 or 3 minutes following carotid cross-clamping, and is due to a baroreceptor reflex stimulation (16, 17). With xenon, the pressure profile is quite different, with a later and more important rise of blood pressure, requiring in some patients an increase of remifentanyl concentration, or administration of nicardipine. This hypertension with xenon was previously described as frequent, and its mechanism needs to be clarified. A sympathetic activation cannot be a definitive explanation because a bradycardia (50 to 60

b/min) was always associated to the operation. Xenon-induced hypertension may confound the clinician's understanding of the depth of anesthesia and therefore some sort of cerebral function monitoring indicative of the depth of anesthesia should be employed. Another hemodynamic observation was a stable bradycardia observed even in patients not treated with a β -blocking agent. This phenomenon cannot be related to the increase in remifentanyl concentrations as it appeared previously. It does not receive a definitive physiopathological explanation at the moment. An activation of the parasympathetic system could be hypothesized. We did not consider as an objective of this study the incidence of postoperative neurological events because they are rare (near 1%) in patients scheduled for carotid surgery. Moreover, mortality and morbidity are both extremely low within all skilled teams (6), and, so far, no agent or technique of anesthesia has demonstrated its superiority in decreasing them.

Although cerebral neurological accidents occurring during carotid surgery are largely related to thromboembolisms (atheromatous, clot or gaseous) which arise before the clamping or after the removal of an arterial clamp, a postoperative stroke can also arise in a situation of poor cerebral perfusion during clamping, more frequently observed in case of occlusion of the contra lateral carotid, or of discontinuity of the circle of Willis. The main measurement of this study was the variation of ScvO_2 , a non-invasive but relevant parameter of the cerebral perfusion. Cerebral perfusion monitoring has been shown of interest under general anesthesia during endarterectomy. Amongst all the monitoring systems available, most of them used for several years, none is perfect. They all present limits that could be logistic or concerning their reliability. Transcranial Doppler is unfeasible for anatomical reasons in up to 20% of the patients, EEG

and monitoring of the somaesthetic potential can have a poor sensitivity and/or a poor specificity, while they are perturbed by agents of anesthesia, measurement of stump pressure is poorly sensitive in indicating the need of a shunt, while furthermore, it is only a punctual measure during a test of clamping, influenced by numerous factors such as arterial pressure, PaCO₂ and agents of anesthesia (18). Measurement of SrcO₂ in regard of the frontal cortex is one of elements that can allow and indirect evaluation of the quality of the cerebral perfusion. It is a technique which is neither invasive, nor observer dependent (8, 19-22). This parameter is in fact a mixture of SaO₂, SvO₂ and tissue saturation in O₂. The measure is moderately polluted by an extra cranial signal. All elements which contribute to modify one of its constituents can explain a decrease of SrcO₂. Several studies concerning the monitoring of SrcO₂ in carotid surgery, in particular those of Murkin et al. (7), reported a good correlation between the modifications of SrcO₂ during the clamping and the modifications of the other techniques of monitoring such as the transcranial Doppler. For Murkin et al. (7), a decline of more than 12% of SrcO₂ or an absolute value lower than 55%, would be predictive of a cerebral ischemia (detected by EEG or somaesthetic evoked potentials) or of modifications of the transcranial Doppler or the stump pressure which can incite the use of a shunt (8, 9). Several criticisms can be made concerning these studies (23-25) since a high incidence of neurological complications was recorded and no study focused on patient operated under general anesthesia. Nevertheless, monitoring of SrcO₂, can allow improvement of hemodynamic status in patients with high cerebral risk. Under xenon, we noted that, compared with a conventional general anesthesia, decline of SrcO₂ at clamping is less important and is observed in fewer patients. This

result is in agreement with the hypothesis of a neuroprotection conferred by this molecule. The neuroprotection may be related in part to a better respect of cerebral perfusion and/or its regulation. Xenon's effects on cerebral perfusion and metabolism are quite complex. Its neuroprotective effect is caused by enhanced ischemic tolerance. Moreover, the connection between neuroprotection and increased perfusion is quite hypothetical. The neuroprotective effects of xenon have been demonstrated in several experimental studies (3). These neuroprotective properties imply complex mechanisms involving: inhibition on the *N*-Methyl-D-aspartic acid (NMDA) receptors, decrease of the glutamate, opening of potassium channels (TREK), comparable to a preconditioning against the ischemic effects. Many cell targets are evoked, at the origin of this preconditioning: mitochondrial, K-ATP canals, PI3-Akt kinases, serine threonine and tyrosine kinases, NO-synthetase, inhibition of free radical scavengers.

The second advantage theoretically brought by xenon is cardioprotection (26). A modification of the activity of the enzyme cyclooxygenase-2 could be involved in this cardioprotective effect. Clinical study has shown a better left ventricular function with xenon compared with propofol (20).

The limits of this study are mostly methodological since this study was not randomized and the patients were included in one or the other group depending on which operative room had been scheduled. Furthermore the use of xenon can have several limits such as the necessity of specific equipment, a phase of learning for its manipulation, an important cost, and the existence of contraindications. A strength of the study was that the cerebral oxymetry was continuously recorded, and was not taken into account in a decision-making algorithm.

CONCLUSION

In this pilot, non-randomized study Xenon was associated with higher cerebral oxygen saturation values when compared to propofol anesthesia during cross-clamping for carotid endarterectomy.

Other studies are needed to confirm these results, to further understand the mechanisms of action and to suggest its use in patients at high-risk of cerebral adverse events.

REFERENCES

1. Bronco A, Ingelmo PM, Aprigliano M, et al. Xenon anaesthesia produces better early postoperative cognitive recovery than sevoflurane anaesthesia. *Eur J Anaesthesiol*. 2010; 27: 912-6.
2. Schifilliti D, Grasso G, Conti A, Fodale V. Anaesthetic-related neuroprotection: intravenous or inhalational agents? *CNS Drugs*. 2010; 24: 893-907.
3. Brücken A, Coburn M, Rex S, et al. Current developments in xenon research. Importance for anesthesia and intensive care medicine. *Anaesthesist*. 2010; 59: 883-95.
4. Chaer RA, Shen J, Rao A, et al. Cerebral reserve is decreased in elderly patients with carotid stenosis. *J Vasc Surg*. 2010; 52: 569-74.
5. Rossaint R, Reyle-Hahn M, Schulte Am Esch J, et al. Xenon Study Group. Multicenter randomized comparison of the efficacy and safety of xenon and isoflurane in patients undergoing elective surgery. *Anesthesiology*. 2003; 98: 6-13.
6. Lewis SC, Warlow CP, Bodenham AR, et al. GALA Trial Collaborative Group. General anaesthesia versus local anaesthesia for carotid surgery (GALA): a multicentre, randomised controlled trial. *Lancet*. 2008; 372: 2132-42.
7. Murkin JM, Arango M. Near-infrared spectroscopy as an index of brain and tissue oxygenation. *Br J Anaesth* 2009; 103: 3-13.
8. Hirofumi O, Otone E, Hiroshi I, et al. The effectiveness of regional cerebral oxygen saturation monitoring using near-infrared spectroscopy in carotid endarterectomy. *J Clin Neurosci* 2003; 10: 79-83.
9. Samra SK, Dy EA, Welch K, et al. Evaluation of a cerebral oximeter as a monitor of cerebral ischemia during carotid endarterectomy. *Anesthesiology* 2000; 93: 964-70.
10. Comité des référentiels de la SFAR. Recommandations formalisées d'experts Gestion périopératoire des traitements chroniques et dispositifs médicaux, 2009.
11. Godet G, Watremez C, El Kettani C, et al. A comparison of sevoflurane, target-controlled infusion propofol, and propofol/isoflurane anesthesia in patients undergoing carotid surgery: a quality of anesthesia and recovery profile. *Anesth Analg* 2001; 93: 560-5.
12. De Castro V, Godet G, Mencia G, et al. Target-controlled infusion for remifentanyl in vascular patients improves hemodynamics and decreases remifentanyl requirement. *Anesth Analg* 2003; 96: 33-8.
13. Godet G, Reina M, Raux M, et al. Anaesthesia for carotid endarterectomy: comparison of hypnotic and opioid-based techniques. *Br J Anaesth* 2004; 92: 329-34.
14. Fahlenkamp AV, Krebber F, Rex S, et al. Bispectral index monitoring during balanced xenon or sevoflurane anaesthesia in elderly patients. *Eur J Anaesthesiol*. 2010; 27: 906-11.
15. Schmidt M, Marx T, Glöggel E, et al. Xenon attenuates cerebral damage after ischemia in pigs. *Anesthesiology*. 2005; 102: 929-36.
16. Godet G, Bernard JM, Bertrand M, et al. [Hemodynamic effect of the clamping of the carotid in the surgically treated coronary patient]. *Ann Fr Anesth Réanim* 1986; 5: 473-8.
17. Godet G, Bernard JM, Bertrand M, et al. [Baroreflex activity in carotid endarterectomy during general anaesthesia]. *Ann Fr Anesth Réanim* 1989; 8: 93-97.
18. Howell SJ. Carotid endarterectomy. *Br J Anaesth* 2007; 99: 119-31.
19. Moritz S, Kasprzak P, Arlt M, et al. 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* 2007; 107: 563-9.
20. Carlin RE, McGraw DJ, Calimlim JR, et al. The use of nearinfrared cerebral oximetry in awake carotid endarterectomy. *J Clin Anesth* 1998; 10: 109-13.
21. Vets P, ten Broecke P, Adriaensen H, et al. Cerebral oximetry in patients undergoing carotid endarterectomy: preliminary results. *Acta Anaesthesiol Belg* 2004; 55: 215-20.
22. Yamamoto K, Komiyama T, Miyata T, et al. Contralateral stenosis as a risk factor for carotid endarterectomy measured by near infrared spectroscopy. *Int Angiol* 2004; 23: 388-93.
23. Kirkpatrick PJ, Lam J, Al-Rawi P, et al. Defining thresholds for critical ischemia by using near-infrared spectroscopy in the adult brain. *J Neurosurg* 1998; 89: 389-94.
24. Mille T, Tachimiri ME, Klersy C, et al. Near infrared spectroscopy monitoring during carotid endarterectomy: which threshold value is critical? *Eur J Vasc Endovasc Surg* 2004; 27: 646-50.
25. Rigamonti A, Scandroglio M, Minicucci F, et al. A clinical evaluation of near-infrared cerebral oximetry in the awake patient to monitor cerebral perfusion during carotid endarterectomy. *J Clin Anesth* 2005; 17: 426-30.
26. Baumert JH, Hein M, Hecker KE, et al. Xenon or propofol anaesthesia for patients at cardiovascular risk in non-cardiac surgery. *Br J Anaesth*. 2008; 100: 605-11.

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