

The Cardiovascular Side Effects during Carotid Artery Stenting under Ultrasound-Guided Carotid Sheath Block

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ABSTRACT

Background: The aim of this study was to determine if the implementation of a carotid sheath block will provide more hemodynamic stability during carotid artery stenting compared with no regional anesthesia procedure for this intervention.

Methods: Since 2014, we have been routinely conducting carotid artery stenting with carotid sheath block in our hospital. A quality-process control survey with before-after design has been performed. The period between January 2012 and December 2013 was before the introduction of regional anesthesia for carotid artery stenting and the period between January 2014 and December 2017 was after its introduction. During the observation, 142 consecutive elective carotid artery stenting interventions were analyzed. Blood pressure, heart rate and the administration of atropine sulphate, catecholamine and vasodilator respectively until 20 minutes after balloon dilatation were examined.

Results: At the beginning of the intervention, the block group showed enhanced hemodynamic parameters as blood pressure and heart rate with an increased demand of vasodilating medications (χ^2 7.15, df 1, $p=0.008$). After carotid artery stenting, we found a lower incidence of asystole and bradycardia (χ^2 4.27, df 1, $p=0.04$) as well as a lower incidence of atropine administration (χ^2 12.10, df 1, $p=0.001$). There was no difference of cardiovascular active medication between the two groups (χ^2 2.17, df 1, $p=0.14$).

Conclusion: Implementation of a carotid sheath block before carotid artery stenting shows a tendency towards more stable hemodynamic parameters during the intervention with significantly lower incidence of atropine administration due to bradycardia (< 25 beats/min) and asystole.

Keywords: Carotid artery stenting; Carotid sheath block; Hemodynamic influences

INTRODUCTION

Arteriosclerosis is a systemic disease, so that patients suffering from carotid artery stenosis typically possess numerous comorbidities, such as coronary heart disease, arterial hypertension, diabetes, etc. Carotid End Arterectomy (CEA) and Carotid Artery Stenting (CAS) are established treatments of symptomatic and asymptomatic, but considerable, carotid artery stenosis and they are effective in preventing a long term incidence of stroke [1,2]. In our hospital, the CEA has been conducted under carotid sheath block since 2007. The advantage of a regional technique is a decreased requirement of shunting

procedures due to continuous sensitive monitoring of neurological function when the patient remains responsive for the surgeons. Shunts should protect the brain from stroke during low cerebral blood flow in the carotid cross-clamping phase [3]. Unfortunately, the arterial wall can be damaged through the shunt which might result in cerebral embolism. Further advantage of Regional Anesthesia (RA) is an improved pain relief with higher patient satisfaction postoperatively and less perioperative hemodynamic instability due to General Anesthesia (GA) [4].

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Received: October 01, 2021; **Accepted:** October 15, 2021; **Published:** October 22, 2021

Citation: Hager C, Schurter D, Schuepfer G, Konrad C, Casutt M (2021) The Cardiovascular Side Effects during Carotid Artery Stenting under Ultrasound-Guided Carotid Sheath Block. J Anesth Clin Res. 12:10027.

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Some studies found an increased mortality due to Myocardial Infarction (MI) when CEA was performed under GA as compared to RA [5,6]. Others found no differences in the incidence of stroke, MI or death [7]. The anesthetic technique for CAS includes analgo-sedation with Local Anesthesia (LA). Performing CAS under GA increased cardiac complications [8]. Studies comparing CEA and CAS found no significant differences in long-term outcomes with respect to stroke, MI or death [1,2,9,10]. But the per procedural risk of stroke was higher after CAS and the risk of MI was higher after CEA [9]. Although in the last years, the risk of stroke seems to shift towards a higher rate of perioperative stroke after CEA compared with CAS [11]. Considering per procedural MI, CEA in RA and CAS in LA seem to carry similar risks [5].

Due to the advantage of RA for CEA with regard to perioperative hemodynamic stability, we decided to introduce the same RA for CAS. There is a pronounced parasympathetic reaction during therapeutic balloon dilatation of a stenosis in the internal carotid artery that regularly provokes reflex bradycardia or even asystole or other forms of arrhythmia, leading to hemodynamic instability. The prevailing medical therapy of such bradycardia consists of the intravenous administration of a parasympatholytic agent such as atropine. Due to the unpredictable dose-effect relation of atropine, it may result in tachycardia and up to 35% in dysrhythmia [12]. Persisting up to 30 minutes which can be deleterious for patients with coronary heart disease, a comorbidity often seen in such patients. After RA of the carotid artery bifurcation area, we anaesthetize the parasympathetic axonal fibers to increase hemodynamic stability during the intervention. For the purpose of a quality-process control survey, this study investigates whether bradycardia and the administration of atropine are decreased after introduction of RA for CAS and if this results in perioperative hemodynamic stability.

MATERIALS AND METHODS

After approval by the Ethics Committee of North-West and Central Switzerland (EKNZ 2014-176, provided by Chairperson Prof. A. P. Perruchoud on 22. June 2014), data were obtained between January 2012 and December 2017 in a single, non-university Swiss central hospital for interventional angiology. Data were analyzed retrospectively. The requirement for written informed consent for this quality-process control survey was waived by the EKNZ. In 2012 and 2013 CAS was conducted without RA (control group). Since January 2014, we have been routinely conducting CAS with RA. The interventional angiology team (two persons) and the CAS techniques remained unchanged during the entire observation period. The anesthesiology team consisted of 20 experienced anesthesiologists. Their anesthesiology strategies remained unchanged during the observation period.

After routine monitoring of five-lead electrocardiography, non-invasive blood pressure, pulse oximetry and peripheral insertion of a 20 or 18 gauge intravenous cannula, a 20 gauge cannula for continuous invasive blood pressure monitoring was placed in the radial artery contralateral to the site of CAS intervention. Oxygen (2 L/min) was administered nasally. The technique of performing the carotid sheath block has been described elsewhere by ourselves [13]. We administered a mixture of 10 ml

prilocaine 1% (100 mg) and 10 ml ropivacaine 0.75% (75 mg) with a total volume of 20 ml on the ventral side of the carotid artery bifurcation area. During the intervention, all patients in both groups were allowed a remifentanyl drip of 0.02-0.2 $\mu\text{g kg}^{-1} \text{min}^{-1}$ from the beginning and 1-2 mg of Midazolam for mild sedation and distress relief while permitting arousability.

Before dilatation of the stenosis, the systolic blood pressure range was requested to be between 160-180 mmHg for sufficient cerebral perfusion pressure. After the intervention, the systolic blood pressure target was requested to be lower than 140 mmHg. To achieve these values, we administered vasoactive medications like continuous administration of norepinephrine or bolus injection of ephedrine to elevate blood pressure.

To depress blood pressure, vasodilating medications such as bolus injection of urapidil or clonidine or continuous Glyceryl trinitrate were administered, respectively. The administration of the respective medication was not defined per protocol and was thus at the discretion of the responsible anesthetist.

From the demographic data, we included age, weight, body height and sex. We also collected blood pressures (systolic, diastolic and medium) and heart rate at the arrival of the patients in the intervention room, at the beginning of the intervention as well as minimal and maximal value during 20 minutes after the balloon dilatation of the stenosis in the internal carotid artery.

Also the application of atropine and the administration of catecholamine respectively vasodilators after balloon dilatation was documented. Neurological monitoring was provided by the interventional angiologist by spontaneous communication and periodic requests to squeeze a rubber duckling.

Data were analyzed using Excel (Microsoft Office 2010, (Microsoft Corporation, Redmond, WA, USA) and the statistic software R (Version 3.1.2). Results are depicted as the mean value and Standard Deviation (SD). A two-sample t test was used to compare the mean of continuous variables. Chi² test was applied to compare the relationship between categorical variables. A p-value of <0.05 was considered to be statistically significant.

RESULTS

From January 2012 to December 2017, we included 142 consecutive patients for elective CAS. There were 60 patients in the control group with a mean age of 72.1 years (9.3), 20% of patients (n=12) were women. Of the 82 patients in the block group, the mean age was 69.4 years (10.2), 20.7% of patients (n=17) were women. The body mass index was 26.1 kg m^{-2} (5.7) in the control group and 26.7 kg m^{-2} (4.2) in the block group. There was no difference in demographic data between the intervention and the control group.

Table 1. shows the hemodynamic comparisons between the two groups. Upon arrival of the patients at the intervention room, the block group showed a higher heart rate than the control group. At the beginning of the intervention, heart rate and blood pressure parameters were higher in the block group. After CAS, heart rate and blood pressure parameters were significantly lower

in the control group compared to the block group. The hemodynamic differences between the measurement time points showed a trend towards lower variability in the block group (results near zero in table 2). With the chi-square test, we found a significant lower incidence of asystolia and bradycardia (<25 beats min⁻¹) in the block group (Chi² 4.27, degrees of freedom df 1, p=0.04) as well as a lower administration of atropine in this group (Chi² 12.10, df 1, p=0.001). Furthermore, we found no differences between the two groups regarding administration of vasoactive medication (Chi² 2.17, df 1, p=0.14) but there was more administration of vasodilating medication in the block group. (Chi² 7.15, df 1, p=0.008).

Table 1: Comparison of hemodynamic parameters between groups.

	Control-group Mean(SD)	Block-group Mean (SD)	P-value
BPs arrival (mmHg)	170.5 (26.0)	167.2 (23.6)	ns
BPd arrival (mmHg)	74.8 (13.7)	78.4 (12.0)	ns
HR arrival (beats min ⁻¹)	70.4 (12.1)	74.6 (11.6)	0.04
BPs start (mmHg)	149.7 (23.9)	167.7 (25.5)	<0.001
BPd start (mmHg)	67.4 (11.4)	77.4 (11.2)	<0.001
HR start (beats min ⁻¹)	71.0 (10.7)	80.5 (13.7)	<0.001
BPs max (mmHg)	155.4 (25.2)	165.0 (23.1)	0.02
BPd max (mmHg)	71.9 (10.3)	76.6 (11.9)	ns
HR max (beats min ⁻¹)	87.1 (13.2)	86.6 (15.9)	ns
BPs min (mmHg)	114.2 (25.8)	139.2 (28.7)	<0.001
BPd min (mmHg)	57.0 (11.1)	66.1 (13.2)	<0.001
HR min (beats min ⁻¹)	65.3 (25.0)	75.7 (19.3)	0.04

Arrival, on arrival of the patient at the intervention room and before implementation of the carotid sheath block; BPs, invasive blood pressure systolic; BPd, invasive blood pressure diastolic; HR, Heart rate; ns, not significant; max, maximal value within 20 min after CAS; min, minimal value within 20 min after CAS; start, at the beginning of the intervention.

Table 2: Comparison of differences of the hemodynamic parameters between groups.

	Control-group Mean (SD)	Block-group Mean (SD)	P-value
Δ BDs arrival-start (mmHg)	20.8 ± 26.7	-0.5 ± 25.8	<0.001
Δ BDd arrival-start (mmHg)	7.3 (12.1)	1.0 (12.7)	0.004
Δ HR arrival-start (beats min ⁻¹)	-0.6 (12.1)	-5.9 (10.3)	0.006
Δ BDs start-max (mmHg)	-5.7 (22.0)	2.7 (23.2)	0.03
Δ BDd start-max (mmHg)	-4.5 (9.5)	0.7 (10.8)	0.003
Δ HR start-max (beats min ⁻¹)	-16.2 (13.9)	-6.1 (9.8)	<0.001
Δ BDs start-min (mmHg)	35.6 (29.1)	28.2 (30.6)	ns
Δ BDd start-min (mmHg)	10.5 (12.6)	11.1 (11.9)	ns
Δ HR start-min (beats min ⁻¹)	6.1 (26.2)	4.7 (15.8)	ns

Arrival, on arrival of the patient at the intervention room and before implementation of the carotid sheath block; BPs, Invasive blood pressure systolic; BPd, Invasive blood pressure diastolic; Δ, difference (delta); HR, Heart rate; ns, not significant; max, maximal value within 20 min after CAS; min, minimal value within 20 min after CAS; start, at the beginning of the intervention.

DISCUSSION

In the current quality-process control study, we considered in a before-after design the effect of carotid sheath block on patient hemodynamics during carotid artery stenting. There was a lower incidence of asystole and bradycardia of <25 beats min⁻¹ as well as a lower incidence of atropine administration in the block group compared to the control group during carotid artery stenting. The administration of vasoactive medication was similar but the administration of vasodilating medications was higher in the block group. Ultrasound-guided nerve blocks are easy to perform, very safe and have a low risk of complications such as vascular punctures [14]. This fact is important for CAS, as nearly all patients have anticoagulants and a hematoma in the stenosis area could further complicate the intervention. Ultrasound-guidance allows a precise injection of the local anesthetic into the carotid bifurcation area. In this area, referred to as sinus caroticus [15], the shell of the artery possesses sensible neuro-plexus of the fact which was stemming from vascular ramus of the glossopharyngeus nerve. In the medulla oblongata, afferent impulses induce inhibition of the sympathetic nervous system as well as amplification of the parasympathetic nervous system via efferent impulses in the vagus nerve [16]. This neuroplexus of the carotid sinus acts as baroreceptor and it controls blood pressure together with the carotid body (paraganglioma with chemoreceptors at the carotid artery bifurcation area). A pressure

sensation could provoke the so-called carotid sinus reflex, with the risk of bradycardia or even asystolia [15,16]. Due to these anatomical requirements, a nerve block in this area seems useful to prevent severe bradycardia and asystole after dilatation during CAS. Performing a carotid sheath block is associated with some risks as the needle can damage vulnerable structures such as arterial punctures (e.g. to the vertebral or carotid artery) resulting in hematoma or intravascular injection with associated anesthetic toxicity, phrenic-nerve paralysis and subsequent respiratory problems, and intrathecal injection resulting in total spinal anesthesia [17,18]. Ultrasound-guided visualization of the carotid artery may reduce the incidence of these complications [14]. We observed no clinically relevant side effects of the carotid sheath block technique and no patient required conversion to general anesthesia. We administered 20 ml of local anesthetic (a mixture of 10 ml prilocaine 1% (100 mg) and 10 cc ropivacaine 0.75% (75 mg)). With this amount of anesthetic, systemic toxicity is not expected in adults. This volume was chosen because we wanted to avoid moving the needle in this well vascularized area. Blockage of the artery bifurcation area as well as the stenosis area had to be performed using one puncture site only. In a former study, we could show with CT-scans and administered X-ray contrast medium, that in 27% of the carotid sheath blocks, carried out in the above described technique but only with 15 ml of local anesthetics, an enclosure of the full circumference of the carotid artery was achieved [13]. However, the clinical result is not dependent on a full circumference of the local anesthetics for CEA. At arrival in the intervention room, we observed the high tendency to slightly enhanced hemodynamic parameters, such as blood pressure and heart rate, in the patients of the block-group. As we did not prescribe any relaxant premedication, this may have been associated with increased activation of the sympathetic due to preintervention anxiety, possibly as a result of the prospect for a procedure perceived as risky. As bradycardia and asystole could be avoided in the block-group, the blood pressure values remained stable after dilatation of the carotid artery. In comparison to the control-group, where minimal blood pressure values were significantly lower, the consumption of vasodilating medications was higher in the block-group due to more enhanced blood pressure values since arrival at the intervention room. If we focus on the hemodynamic differences between the two groups, we see a tendency towards more stable hemodynamic values in the block-group (there is a lower distribution around zero).

To the best of our knowledge and in the regional of anesthesia as carotid sheath block for CAS has not yet been described. The current study does have limitations. Because of its retrospective character, patients were not randomized according to the type of anesthesia. Details regarding treatment of occurring arterial hypo- or hypertension and the management of blood pressure were not defined in a protocol. It was at the discretion of the treating anesthetists to decide on a required treatment. Further, we did not analyze hemodynamic data later than 20 minutes after dilatation of the carotid artery, neither did we analyze postoperative analgesic effects. Studies in the future have to assess whether a reduction in local anesthetic volume will have the same success as the currently used 20 ml. A prospective and randomized study design could record hemodynamic parameter and drug administration more accurately, such that a more detailed registration of adverse events,

impact of the cervical sheath block to perioperative mortality and neurological outcome could be projected.

CONCLUSION

In conclusion, patients undergoing CAS, benefit from a carotid sheath block. They have significantly less hemodynamic instability due to less frequent events of peri-procedural severe bradycardia or asystole. As a result of this, lower rates of atropine administration contribute even more to cardiovascular stability. An ultrasound-guided carotid sheath block is effective and has a low complication rate. Further knowledge of the role of bradycardia and asystole with consecutive drug treatment on neurological outcome and mortality is required to reduce more peri-procedural hemodynamic events of carotid revascularization.

FUNDING

Supported by the Department of Anesthesiology, Kantonsspital Lucerne

CONFLICT OF INTEREST

There is no conflict of interest

AUTHORSHIP CONTRIBUTION

Mattias Casutt designed the study, conducted the study, analyzed the data and wrote the manuscript. Chantal Hager and David Schurter helped to conduct the study, wrote the manuscript and collected the data. Guido Schüpfer conducted the statistical analysis and helped to write the manuscript. Christoph Konrad helped to conduct the study and wrote the manuscript.

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