Effect of Superficial Cervical Plexus Block on Baroreceptor Sensitivity in Patients Undergoing Carotid Endarterectomy

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Objectives: Regional anesthesia for patients undergoing carotid endarterectomy is associated with improved intraoperative hemodynamic stability compared with general anesthesia. The authors hypothesized that the reported advantages might be related to attenuated ipsilateral baroreflex control of blood pressure, caused by chemical denervation of the carotid bulb baroreceptor nerve fibers.

Design: A prospective cohort study.

Setting: Single-center university hospital.

Participants: The study included 46 patients undergoing carotid endarterectomy using superficial cervical block.

Interventions: A noninvasive computational periprocedural measurement of baroreceptor sensitivity was performed in all patients. Two groups were formed, depending on the patients' subjective response to surgical stimulation regarding the necessity of additional intraoperative local anesthesia (LA) administration on the carotid bulb. Group A (block alone) included 23 patients who required no additional anesthesia, and group B (block + LA) consisted of 23 patients who required additional anesthesia.

Measurements and Main Results: Baroreceptor sensitivity showed no significant change after application of the block in both groups (group A: median [IQR], 5.19 [3.07-8.54] *v* 4.96 [3.1-9.07]; p = 0.20) (group B: median [IQR], 4.47 [3.36-8.09] *v* 4.53 [3.29-8.01]; p = 0.55). There was a significant decrease in baroreceptor sensitivity in group B after intraoperative LA administration (median [IQR], 4.53 [3.29-8.01] *v* 3.31 [2.26-7.31]; p = 0.04).

Conclusions: Standard superficial cervical plexus block did not impair local baroreceptor function, and, therefore, it was not related to improved cerebral perfusion in awake patients undergoing carotid endarterectomy. However, direct infiltration of the carotid bulb was associated with the expected attenuation of baroreflex sensitivity.

KEY WORDS: superficial cervical plexus block, baroreceptor sensitivity, carotid endarterectomy

CAROTID ENDARTERECTOMY (CEA) is well recognized as secondary prophylaxis of stroke in symptomatic and asymptomatic atherosclerotic carotid disease.[1] Controversy remains over which anesthetic technique should be applied during CEA because results of the multicenter, randomized, controlled GALA (General Anaesthesia versus Local Anaesthesia for Carotid surgery) trial were inconclusive and showed no significant difference between regional and general anesthesia in terms of the primary endpoints of stroke and death.[2] However, there is evidence that, apart from the more readily neurologic assessment,[3] regional anesthesia is beneficial in terms of cerebral perfusion due to improved hemodynamic stability,[4,5] which can be understood as fluctuations in blood pressure (BP) and heart rate (HR) that stay in range without requiring additional catecholamine use. The authors, therefore, hypothesized that a temporary chemical denervation of the ipsilateral local baroreceptor nerve fibers after superficial cervical plexus block (SCB) might exist because the injected anesthetic may diffuse to the carotid bulb. A prior study showed that mechanical denervation during CEA decreased baroreceptor sensitivity (BRS), consequently leading to increased postoperative sympathetic activity.[6] To the authors' knowledge, there are no published studies showing the impact of SCB on BRS in patients undergoing CEA.

The baroreceptors located in the carotid bulb are a key component in the baroreflex loop, transmitting a registered increase in wall tension due to elevated BP to the central nervous system, which generates a negative chronotropic and dromotropic effect on the heart and causes vasodilation of peripheral and cardiac blood vessels.[7] The resulting reductions in HR and total peripheral resistance (TPR) counteract the rise in BP.[8] An impairment of the baroreflex function would correlate with a decrease in BRS, which is associated with an increase of the autonomic cerebrovascular regulation, resulting in an increase in BP.[9] The aim of this study was to assess the influence of SCB on BRS in patients undergoing CEA.

METHODS

The study protocol was approved by the appropriate Institutional Review Board (Heidelberg, Germany, reference number: S-507/2012; approval date: January 30, 2013), and written informed consent was obtained from all patients. From November 11, 2013, until December 31, 2014, fifty patients with symptomatic or asymptomatic internal carotid artery stenosis (>70% stenosis grade on carotid duplex ultrasound)

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were scheduled to undergo CEA using SCB. Symptomatic stenosis was defined as transient ischemic attack or minor stroke (modified Rankin scale [mRS] of 0-2). To prevent patient selection bias, study participants were recruited consecutively from the authors' institution's patient contingent.

Anesthetic Procedures

In the surgical ward, all patients took their daily antihypertensive medication, if indicated, and 150 µg of clonidine was administered orally for sedation and analgesia and for its stabilizing hemodynamic effect with fewer postoperative hypertensive episodes[10] before the patients were transferred to the induction room, where arterial BP monitoring, peripheral venous access, pulse oximetry, and electrocardiography were initiated. Recording of BRS was started 10 minutes after patients were acclimatized in the supine position. Within this period, oxygen via nasal cannula (6 L/min) was begun and administered continuously throughout the surgical procedure, and remifentanil perfusion (0.05 µg/kg body weight/min) was started for anxiolytic purposes. The method of SCB application was standardized, and all blocks were performed or assisted by an experienced anesthesiologist who used ultrasonographic guidance. A standard mixture of 30 mL of prilocaine 1%, 20 mL of ropivacaine 0.5%, and 150 µg of clonidine was used for all blocks. A single injection site was chosen on the posterior border of the sternocleidomastoid muscle, approximately at the level of the sixth cervical vertebra. An initial 5 mL were deposited directly under the platysma, and after bending of the needle, the remaining 45 mL were distributed in a fan fashion with the intention of reaching in the cranial, caudal, medial, and lateral directions of the subcutaneous, subplatysmal, and subfascial compartments, staying deeper than the investing fascia. The analgesic efficacy was controlled through loss of pinprick or altered temperature sensation in the dermatomes corresponding to the targeted nerves. After effective analgesia was achieved, the patient was taken to the operating room. Intraoperatively pain was managed with a standard 10-mL dilution of 1% articaine on the carotid bulb.

Data Acquisition

Noninvasive estimates of spontaneous BRS: BP (systolic pressure [SP, mmHg]; mean arterial pressure [MAP]; diastolic pressure [DP, mmHg]); HR (beats/min); and total peripheral resistance (TPR, dyn.s/cm[5]) records were obtained using the Finometer MIDI device (Finapres Medical Systems BV, Amsterdam, The Netherlands). The Finometer MIDI system is based on the volume clamp of the finger artery obtained using a servo-controlled pneumatic micro-cuff placed around a finger of the hand. It computes the cross-correlation in time domain between beat-to-beat systolic BP and R-R interval, resampled at 1 Hz, in a sliding 10-second window, with delays of 0 to 5 seconds for interval. The delay with the greatest positive correlation is selected and, when significant at $p = 0.01$, the slope and delay are recorded as $1 \times$ BRS value. Each 1 second of the recording is the start of a new computation.[11] A number of studies have shown a satisfactory agreement between Finometer MIDI and intra-arterial BP values measured over short periods at rest and during laboratory tests.[12-20]

The Finometer MIDI reconstructs the brachial artery pressure wave in waveform and level. Compared with intrabrachial artery pressure, the mean difference (bias) of systolic pressure is +1 mmHg; of diastolic pressure, -8 mmHg; and of mean arterial pressure, -10 mmHg compared with corresponding standard deviations (precision) of 11 mmHg, 8 mmHg, and 7 mmHg, respectively.[13] After waveform reconstruction and Riva-Rocci return-to-flow level calibration, a reconstructed brachial artery pressure is produced with reduced errors. The results are within the American national standard specification (AAMI: American National Standard ANSI/AAMI SP10-1992: electronic or automated sphygmomanometers. Association for the Advancement of Medical Instrumentation, 1993, Arlington, VA), which requires bias to be less than 5 mmHg and precision to be better than 8 mmHg. Clearly, waveform reconstruction reduces finger pressure uncertainty.[21] Therefore, application of Finometer MIDI in clinical practice seems to not be limited by inaccuracies or by risk to the patient. The TPR is measured by using the model-flow method of Wesseling et al.[18] This parameter is a quantity that cannot be measured directly, but is derived from the simultaneous measurement of pressure and flow by division. Thus, the medical unit for TPR is mmHg.s/mL. However, TPR often is expressed in centimeter gram-second system (CGS) units. In this system, pressure is expressed in dyn/cm^2 and flow in cm^3/s, and, consequently, resistance in dyn.s/cm[5].

Intraoperative local anesthesia (LA) was administered as a response to pain during surgical incision of the periadventitial layer of the carotid bulb during preparation. A 5-mL syringe containing the anesthetic solution was given to the surgeon, who in turn applied the LA to the adventitial segment of the carotid bulb and on the surrounding tissue. After a brief period to allow for analgesia, surgical preparation was continued. None of the patients requiring additional LA exhibited hypotension or bradycardia.

Depending on whether administration of additional LA was performed, the measurement period was subdivided in different phases. If the patient did not require additional intraoperative LA (group A: block alone), the measurement consisted of 2 sections: the first, from start of measurement until completion of cervical block; and the second, from completion of cervical block until cross-clamping. If the patient required additional LA (group B: block plus LA), the measurement consisted of 3 sections: the first, from start of measurement until completion of cervical block; the second, from completion of cervical block until administration of LA by the surgeon; and the third, from administration of LA until cross-clamping. This subdivision permitted differentiation of eventual effects of SCB on BRS from the intraoperatively administered LA. Restricting the measurement period until cross-clamping was necessary to prevent any confounding effect of additional surgical impairment of the baroreceptor function.

Artificial increase of BP was not performed until the end of the measurement (cross-clamping) to prevent any bias regarding interpretation of potential BP increase due to cervical plexus block and/or intraoperative LA administration. All data were collected again in a preconfigured Apache OpenOffice database (version 4.1.1; Apache Software Foundation, Los Angeles, CA) for later statistical analysis.