Intraoperative baroreflex failure following lignocaine infiltration during unilateral radical neck dissection

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The baroreceptor reflex serves to prevent excessive fluctuations of arterial blood pressure (BP). In baroreflex failure, there is loss of buffering ability and wide excursions of pressure and heart rate (HR) occur.[1] We report a 58-year-old patient who developed intraoperative baroreflex failure manifesting as severe hypotension necessitating vasopressor support following lignocaine infiltration in the carotid sinus area during unilateral radical neck dissection (RND).

A 58-year-old male, with neck secondaries, was scheduled for left RND. Baseline hemodynamic parameters were normal. Anesthesia was induced with morphine 4.5 mg and propofol 90 mg. Intubation was facilitated with atracurium 30 mg. Marked hemodynamic lability was observed at induction and intubation [Table 1]. Anesthesia was maintained with 0.6% isoflurane in N_2O and O_2. The initial intraoperative period was associated with bradycardia, followed by tachycardia and hypertension [Table 2]. Anesthesia was deepened (isoflurane 1-2%, fentanyl 50 μg) and esmolol 20 mg intravenous (IV) was administered. As tachycardia persisted, the operative site near carotid bifurcation was infiltrated with 5 ml lignocaine 2% (without preservative). The hemodynamic perturbations that followed lignocaine infiltration and its management are detailed in Table 2. The patient received IV boluses of mephentermine and adrenaline. Dopamine infusion was initiated (15 μg/kg/min) followed by norepinephrine infusion (5 μg/min). Within 2 min of norepinephrine infusion, BP stabilized between 110/87/91 and 117/74/91 mmHg; however, tachycardia persisted (HR 155-172/min). Isoflurane was re-started, FiO_2 was 0.5 and esmolol 20 mg IV was administered. Arterial blood gas (ABG) analysis revealed metabolic acidosis. Surgery recommenced. Dopamine and norepinephrine infusion rates were reduced. At 75 min of lignocaine infiltration, norepinephrine was discontinued, and dopamine continued at 5 μg/kg/min. HR was 100/min, BP 103/65/79 mmHg, SpO_2 100% with FiO_2 0.5. Left RND was completed. Patient received 2.5 L Ringer's lactate solution. The patient was transferred to the Intensive Care Unit on controlled ventilation. Echocardiogram was normal. Two hours after lignocaine infiltration, the patient was hemodynamically stable, ABG was normal, dopamine infusion was discontinued and trachea was extubated. The patient had a normal recovery.

During RND, surgical manipulation around the carotid artery has the potential to cause hemodynamic perturbations and dysrhythmias. Our 58-year-old, normotensive patient developed tachycardia and hypertension intraoperatively during unilateral RND. The hemodynamic changes did not respond to conventional methods. Local anesthetic (LA) infiltration near the carotid bifurcation was performed with the intention of mitigating tachycardia and hypertension. We had not anticipated the occurrence of severe hypotension that followed LA infiltration. The response to norepinephrine suggests that the patient experienced severe vasomotor failure. A search of the literature revealed some interesting data.

Baroreflex failure is likely to develop in patients after RND,[2] carotid artery surgery, throat irradiation and neck injury.[1] The baroreceptors are located in the carotid sinus at the bifurcation of the common carotid artery and in the aortic arch. For clinical baroreflex failure to manifest, severe interruption of afferent input must be present. We believe that the occurrence

Table 1: HR and BP changes during induction of anesthesia and intubation

Time	HR (beats/min)	SBP/DBP (MAP) (mmHg)	Intervention
Preoperative	86	134/82 (99)	
Postinduction	93	60/46 (84)	
Postintubation 1 min	117	172/99 (123)	Isoflurane 1-2%
Postintubation 2 min	103	192/116 (166)	Propofol 20 mg
Postintubation 5 min	96	176/104 (128)	Isoflurane 1-2%
Postintubation 10 min	87	153/89 (114)	Isoflurane 1%

BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, MAP = Mean arterial pressure, HR = Heart rate

Table 2: Summary of intraoperative events during left radical neck dissection

Patient exhibited hemodynamic lability at induction and intubation
Bradycardia (42/min) during neck dissection, glycopyrrolate 0.2 mg administered IV
Tachycardia with hypertension (HR 115-120/min, BP 138/97/118 mmHg)
Anesthesia deepened (isoflurane, fentanyl, morphine); esmolol boluses (20+20 mg)
0 min: Lignocaine infiltration in operative site near carotid bifurcation
3 min later: Sinus tachycardia, severe hypotension (minimum BP 40/26 (32) mmHg, HR 151/min); surgery stayed
6 min: Mephentermine (6+6 mg; HR 139/min, BP 60/39/49 mmHg)
18 min: Adrenaline boluses (50+50 μg; HR 151/min, BP 40/26/32 mmHg)
24 min: Dopamine infusion (15 μg/kg/min)
27 min: Norepinephrine infusion (5.0 μg/min)
29 min: BP 110/87/91-117/74/91 mmHg; HR 155-172/min. Isoflurane re-started; FiO_2 0.5; dopamine and norepinephrine infusions decreased
Surgery recommenced and radical neck dissection completed
75 min: Noradrenaline infusion discontinued, dopamine infusion (5 μg/kg/min)
110 min: Transferred to ICU (HR 100/min, BP 102/64/77 mmHg; on dopamine 5 μg/kg/min)
Echocardiogram: Normal study
Restoration of normal hemodynamic parameters within 2 h of lignocaine infiltration

HR = Heart rate, BP = Blood pressure, ICU = Intensive Care Unit, IV = Intravenous

of severe hypotension following LA infiltration in the carotid sinus area was a consequence of baroreflex failure.

Manipulation of these baroreceptors during neck surgery may result in bradycardia, hypotension or cardiac dysrhythmia.[3] Many surgeons advocate anesthetizing the carotid bulb prophylactically; however, this practice is not universally accepted. LA infiltration of the carotid sinus may result in hypertension,[4] hypotension[5,6] or no effect.[7] Barman *et al.*[4] reported possibly harmful intraoperative increases in systolic BPs and mean pressures with lidocaine infiltration of the carotid sinus. Tang *et al.*[5] found that LA blockade of the carotid sinus was associated with a pooled odds ratio of hypotension −1.25 (95% confidence interval [*CI*] 0.496-3.15; $P = 0.216$) and hypertension −1.28 (95% *CI* 0.699-2.33; $P = 0.428$) for development of postoperative hypotension and hypertension, respectively. Although none reached significance, there was a trend toward increased risk of developing a complication in those patients who received LA. Similarly, stripping the aortic arch and the carotid sinus areas (sino-aortic denervation) in dogs resulted in extreme lability of arterial BP with normal, hypertensive and hypotensive values being recorded.[6]

Carotid sinus hypersensitivity (CSH), an exaggerated response of the carotid sinus baroreceptor to local stimulation, is a possibility in our patient that manifested intraoperatively as baroreflex failure following LA infiltration. The exhibition of BP lability during induction of anesthesia and intubation, intraoperative occurrence of severe bradycardia initially and later tachycardia with hypertension during RND suggests the presence of CSH in our patient. The incidence of intraoperative CSH is 28% in elderly patients undergoing RND.[8] Local factors that exert pressure on the carotid sinus such as tumor, lymph nodes, and scars may lead to CSH.[3] The majority of reports of carotids sinus syndrome associated with head and neck malignancy relate to extensive nodal involvement in the neck;[9] our patient had nodal neck secondaries. The temporal sequence of hemodynamic events following LA infiltration around the carotid bifurcation (in a bid to control the hemodynamic response during RND), the response to norepinephrine and the subsequent return of hemodynamics to ≈ 90-100 mmHg at 75 min is in keeping with duration of action of lignocaine and suggests that baroreflex failure was a consequence of lignocaine infiltration. Clinicians should be aware of the possibility of occurrence of hemodynamic lability should they consider LA infiltration in the carotid sinus area during RND.

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References

1. Ketch T, Biaggioni I, Robertson R, Robertson D. Four faces of baroreflex failure: Hypertensive crisis, volatile hypertension, orthostatic tachycardia, and malignant vagotonia. Circulation 2002;105:2518-23.
2. Prakash S, Rapsang A, Kumar SS, Bhatia PS, Gogia AR. Postoperative hypertension following radical neck dissection. J Anaesthesiol Clin Pharmacol 2012;28:121-3.
3. Babin RW, Panje WR. The incidence of vasovagal reflex activity

during radical neck ddissection. Laryngoscope 1980;90:1321-3.
4. Barman AA, Moideen AS, Chaudhry SS, Ardell J, Barman SA, Cantu R. Nerve-sparing dissection as alternative to anesthesia of carotid sinus nerve in preparation for carotid endarterectomy. Tex Heart Inst J 1989;16:107-9.
5. Tang TY, Walsh SR, Gillard JH, Varty K, Boyle JR, Gaunt ME. Carotid sinus nerve blockade to reduce blood pressure instability following carotid endarterectomy: A systematic review and meta-analysis. Eur J Vasc Endovasc Surg 2007;34:304-11.
6. Cowley AW Jr, Liard JF, Guyton AC. Role of baroreceptor reflex in daily control of arterial blood pressure and other variables in dogs. Circ Res 1973;32:564-76.
7. Ajduk M, Tudoric I, Sarlija M, Pavic P, Oremuš Z, Held R, *et al.* Effect of carotid sinus nerve blockade on hemodynamic stability during carotid endarterectomy under local anesthesia. J Vasc Surg 2011;54:386-93.
8. Kim TI, Lim HJ, Chang SH, Kim NS. Intraoperative carotid sinus hypersensitivity and postoperative complication of radical neck dissection retrospective study. Korean J Crit Care Med 1998;13:49-54.
9. Papay FA, Roberts JK, Wegryn TL, Gordon T, Levine HL. Evaluation of syncope from head and neck cancer. Laryngoscope 1989;99:382-8.

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