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(CASE REPORT)

Extreme intraoperative bradycardia during total thyroidectomy: A case report

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Abstract

Severe bradycardia in the operating room is uncommon during thyroid surgery but may occur during deep cervical maneuvers due to vagal or carotid sinus stimulation. We report the case of a 52-year-old female, ASA II, operated on for a compressive multinodular euthyroid goiter, who developed extreme bradycardia at 24 beats/min with hypotension at 68/35 mmHg at the time of right lateral-tracheal traction during dissection of the superior pole. Immediate cessation of the surgical maneuver, 100% oxygenation, administration of atropine 1 mg IV followed by ephedrine 6 mg IV led to rapid hemodynamic recovery. There was no evidence of hypoxia, hypercapnia, electrolyte disturbance, anaphylaxis, or acute coronary event. The most likely mechanism was a cardioinhibitory reflex due to carotid stimulation or increased vagal tone. This case highlights the need for early recognition, immediate communication between surgeon and anesthetist, and management in accordance with recommendations for symptomatic bradycardia.

Keywords: Total thyroidectomy; Bradycardia; Cervical maneuver; Vagal reflex.

1. Introduction

Thyroid surgery is a common procedure whose classic complications include cervical hematoma, hypocalcemia, and recurrent laryngeal nerve injury. Severe rhythm disturbances are rare but potentially serious. The NAP7 report reminds us that profound perioperative bradycardias occur in routine anesthetic practice and can, in some cases, progress to a peri-arrest cardiac state. During cervical procedures, neck hyperextension, lateral traction, and dissection near the neurovascular bundle can cause excessive stimulation of the carotid baroreceptors, producing a cardioinhibitory response with bradycardia, hypotension, or even asystole

2. Clinical case

A 52-year-old female, 74 kg, ASA II, with well-controlled hypertension on amlodipine and no history of arrhythmia or coronary disease, was admitted for scheduled total thyroidectomy for a compressive multinodular euthyroid goiter. Preoperative ECG was normal; electrolytes, high-sensitivity TSH, and FT4 were within normal limits. After induction with fentanyl 150 MG, propofol 140 mg, and rocuronium 50 mg, orotracheal intubation was uneventful. Anesthesia was maintained with sevoflurane at 1 MAC and mechanical ventilation ensuring an EtCO2 between 34 and 38 mmHg. Thirty-five minutes after incision, during right lateral-tracheal traction while dissecting the superior pole, there was a sudden drop-in heart rate from 71/min to 24/min and blood pressure from 124/72 to 68/35 mmHg. SpO2 remained 99% and EtCO2 was 35 mmHg. The absence of rash, bronchospasm, or increased airway pressures excluded anaphylaxis. The surgical maneuver was immediately stopped. FiO2 was increased to 100%; atropine 1 mg IV and ephedrine 6 mg IV produced, in less than one minute, an increase in heart rate to 62/min and blood pressure correction to 102/61 mmHg. Surgery was cautiously resumed with reduced traction, without recurrence. Postoperative troponins, blood gas analysis, electrolytes, and echocardiography were normal. The course was favorable and the patient was discharged on day three. Table 1.

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Table 1 Summary of clinical event and management of intraoperative bradycardia

Parameter	Findings
Patient characteristics	52-year-old female, ASA II, 74 kg
Indication for surgery	Compressive multinodular euthyroid goiter
Preoperative assessment	Normal ECG, normal electrolytes, normal TSH and FT4
Timing of event	35 minutes after incision
Triggering factor	Right lateral-tracheal traction during superior pole dissection
Hemodynamic changes	Heart rate: 71 → 24 bpm; Blood pressure: 124/72 → 68/35 mmHg
Respiratory parameters	SpO_2: 99%, $EtCO_2$: 35 mmHg (stable)
Differential diagnoses excluded	Hypoxia, hypercapnia, anaphylaxis, metabolic disturbance, myocardial ischemia
Immediate management	Surgical maneuver stopped; 100% oxygen administered; Atropine 1 mg IV; Ephedrine 6 mg IV
Outcome	Rapid recovery: HR 62 bpm, BP 102/61 mmHg within <1 min
Surgical course	Procedure resumed cautiously, no recurrence
Final diagnosis	Vagal reflex / carotid sinus stimulation

3. Discussion

The abrupt onset of the event, its simultaneity with a deep cervical maneuver, and reversibility after stopping the stimulus and giving atropine strongly point to a vagal reflex or carotid sinus stimulation. Lilit is et al. reported asystole during cervical hyperextension for thyroidectomy positioning [1]. Chung et al. described transient cardiac arrest during robot-assisted thyroidectomy attributed to carotid sinus hypersensitivity [2]. Similar observations have also been published during neck dissections. The differential diagnosis must first exclude hypoxia, hypercapnia, excessive anesthetic depth, opioid overdose, metabolic causes, anaphylaxis, or myocardial ischemia [3,4, 5]. In our case, these causes were unlikely given stable saturation, EtCO2, and laboratory workup. Management recommends rapid identification of the trigger, immediate cessation of the stimulus, oxygen, close monitoring, and IV atropine as first-line therapy; if ineffective, external pacing and/or dopamine or epinephrine infusion should be considered [3].

4. Conclusion

Conclusion Extreme bradycardia during total thyroidectomy is rare but potentially dramatic. Carotid sinus stimulation or an exaggerated vagal reflex should be considered for any sudden drop-in heart rate during deep cervical maneuvers. Stopping the surgical stimulus, anticholinergic treatment, and hemodynamic support usually result in a favorable outcome. Such events warrant constant vigilance and close coordination between the anesthetist–resuscitator and the surgeon.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict-of-interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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