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Original Research Article

Cardiovascular Changes Associated with Nasotracheal Intubation in Patients under General Anaesthesia

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Abstract:

Background: Nasotracheal intubation is frequently employed in maxillofacial, dental, and certain head-and-neck procedures to optimize surgical access. Unlike orotracheal techniques, the nasal route may evoke differing sympathetic and nociceptive stimuli, potentially leading to distinct haemodynamic perturbations. Acute elevations in heart rate and blood pressure during intubation can increase myocardial oxygen demand and precipitate complications in vulnerable patients. Despite its common use, detailed characterization of the timing, magnitude, and duration of cardiovascular responses to nasotracheal intubation remains limited. This study aimed to quantify the haemodynamic changes occurring immediately before, during, and after nasotracheal intubation under standardized general anaesthesia.

Methods: In this prospective observational study, 60 adult patients (ASA I–II), aged 18–65 years, scheduled for elective maxillofacial or dental surgery requiring nasotracheal intubation were enrolled. All received a uniform anaesthetic regimen: premedication with midazolam 0.05 mg/kg and fentanyl 2 µg/kg, induction with propofol 2 mg/kg and vecuronium 0.1 mg/kg, and maintenance with isoflurane in oxygen/air. A well-lubricated, appropriately sized nasotracheal tube was passed under direct laryngoscopy. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline (pre-induction), pre-intubation (post-muscle relaxation), immediately after tube placement (T0), and at 1 (T1), 3 (T3), and 5 minutes (T5) following intubation. Data were analysed using repeated-measures ANOVA with Bonferroni correction; $p < 0.05$ was deemed significant.

Results: All 60 patients completed the protocol. Compared with baseline, HR increased by 18 ± 6 bpm at T0 ($p < 0.001$), peaked at T1 with a rise of 22 ± 7 bpm ($p < 0.001$), and gradually returned toward baseline by T5 (increase 6 ± 4 bpm; $p = 0.04$). SBP rose from 122 ± 10 mm Hg at baseline to 150 ± 12 mm Hg at T0 ($p < 0.001$), peaked at 155 ± 14 mm Hg at T1 ($p < 0.001$), then declined to 130 ± 11 mm Hg by T5 ($p = 0.02$). DBP and MAP exhibited similar patterns, with maximal increases of 20 ± 5 mm Hg and 25 ± 6 mm Hg at T1, respectively (both $p < 0.001$). No patient experienced arrhythmias or required pharmacologic intervention beyond the planned protocol.

Conclusions: Nasotracheal intubation under general anaesthesia elicits pronounced sympathetic responses, with heart rate and blood pressure peaking at approximately one minute post-intubation and returning toward baseline by five minutes. These transient haemodynamic surges should be anticipated and, when necessary, attenuated with tailored anaesthetic depth or short-acting vasoactive agents, especially in patients with cardiovascular comorbidities.

Introduction

Nasotracheal intubation is a commonly employed technique in maxillofacial, dental, and otolaryngologic procedures to provide an unobstructed oral field while securing the airway. Unlike orotracheal intubation, advancing the endotracheal tube through the nasal passages stimulates richly innervated mucosal and pharyngeal receptors, potentially provoking a more pronounced sympathetic discharge [1]. This reflex sympathetic activation manifests as acute increases in heart rate and arterial blood pressure, which may elevate myocardial oxygen demand and precipitate ischemic events in patients with limited cardiac reserve. Although orotracheal intubation has been extensively studied, the haemodynamic consequences of the nasal route under contemporary anaesthetic regimens remain less well characterized [2].

Early reports suggested that nasal passage of the tube, combined with direct laryngoscopy, produces dual noxious stimuli at different levels of the upper airway, potentially resulting in a biphasic haemodynamic response [3]. Furthermore, the smaller internal diameter and longer length of nasotracheal tubes may necessitate greater force or repeated maneuvers, potentially accentuating cardiovascular perturbations. Standardizing premedication, induction agents, and muscle relaxation is therefore critical to isolate the effect of the intubation stimulus itself [4]. Quantifying the timing, magnitude, and duration of the heart rate and blood pressure surges associated with nasotracheal intubation can inform targeted interventions such as titrated analgesic dosing, deeper volatile anaesthetic depth, or prophylactic administration of short-acting β-blockers—to attenuate these responses [5].

Given the increasing prevalence of outpatient and ambulatory maxillofacial procedures, understanding and mitigating transient but potentially hazardous haemodynamic swings is of growing importance. This study seeks to provide a comprehensive temporal profile of cardiovascular changes around the moment of nasotracheal intubation, measured at baseline, immediately post-tube placement, and at defined intervals thereafter. By doing so, it aims to guide clinicians in optimizing anaesthetic management for patients at varying levels of cardiovascular risk undergoing nasal intubation.

Materials and Methods

This prospective observational study was conducted in the Department of Anesthesiology, Government Medical College, and Hospital, Purnea, Bihar, India, from January 2023 to December 2023.

Patient Selection

Sixty adult patients aged 18–65 years, ASA I–II, scheduled for elective maxillofacial or dental procedures requiring nasotracheal intubation under general anaesthesia were enrolled. Exclusion criteria included anticipated difficult nasal anatomy, coagulopathy, uncontrolled hypertension, ischaemic heart disease, chronic β-blocker therapy, allergy to study drugs, and active upper respiratory infection.

Anaesthetic Protocol

All patients fasted six hours and received oral alprazolam 0.25 mg the night before surgery. In the OR, standard monitors (ECG, NIBP, SpO_2) were applied. Baseline HR and blood pressures were recorded. An IV line was placed with lactated Ringer's at 10 mL/kg over 15 min pre-induction. Premedication: midazolam 0.05 mg/kg and fentanyl 2 μg/kg IV. Induction: propofol 2 mg/kg IV; muscle relaxation: vecuronium 0.1 mg/kg IV; mask ventilation with 100% O_2 for 3 min.

Intubation Technique and Measurements

A lubricated, appropriately sized cuffed nasotracheal tube (7.0 mm ID for women, 8.0 mm ID for men) was advanced under direct laryngoscopy, with no more than two attempts. HR, SBP, DBP, and MAP were recorded at:

- Baseline (pre-induction)
- Pre-intubation (post-relaxation)
- T0 (immediately after intubation)
- T1 (1 min post)
- T3 (3 min post)
- T5 (5 min post)

Management of Haemodynamic Changes

Hypotension (>20% drop in SBP or <90 mm Hg) was treated with 250 mL crystalloid and ephedrine 6 mg IV. Bradycardia (HR <50 bpm) was treated with atropine 0.6 mg IV. Any additional vasoactive use was recorded.

Statistical Analysis

A sample of 60 provided 80% power to detect a 15 bpm HR change (α=0.05). Data are mean ± SD and analysed with repeated-measures ANOVA and Bonferroni correction. Categorical data use χ^2 or Fisher's exact test. A p-value <0.05 was significant.

Results

Sixty patients were included in the analysis. Nasotracheal intubation was successful on the first attempt in 92 percent of cases, with a mean intubation time of 14.8 ± 2.9 seconds. Sympathetic responses peaked at one minute post-intubation and gradually resolved by five minutes. Ten patients (17 percent) required ephedrine for hypotension, and two (3 percent) received atropine for bradycardia. No arrhythmias occurred. Minor nasal mucosal bleeding was noted in three patients (5 percent) and minor nasal trauma in two (3 percent).

Table 1: Baseline demographic characteristics

Parameter	Value (n = 60)
Age (years, mean ± SD)	42.5 ± 11.2
Sex (M/F)	34/26
BMI (kg/m², mean ± SD)	23.9 ± 3.1

Table 2: Baseline hemodynamic parameters (n = 60)

Parameter	Mean ± SD
HR (bpm)	75 ± 8
SBP (mm Hg)	122 ± 10
DBP (mm Hg)	76 ± 8
MAP (mm Hg)	91 ± 7

Table 3: Intubation characteristics

Characteristic	Value
Mean intubation time (s)	14.8 ± 2.9
First-attempt success	55 (92 %)
Second-attempts	5 (8 %)

Table 4: Heart rate at key time points

Time point	HR (bpm, mean ± SD)	p-value
Baseline	75 ± 8	–
T0 (immed.)	93 ± 10	< 0.001
T1 (1 min)	97 ± 12	< 0.001
T3 (3 min)	85 ± 9	< 0.01
T5 (5 min)	81 ± 8	0.04

Table 5: Systolic blood pressure over time

Time point	SBP (mm Hg, mean ± SD)	p-value
Baseline	122 ± 10	–
T0 (immed.)	150 ± 12	< 0.001
T1 (1 min)	155 ± 14	< 0.001
T3 (3 min)	135 ± 11	< 0.01
T5 (5 min)	130 ± 11	0.02

Table 6: Diastolic blood pressure over time

Time point	DBP (mm Hg, mean ± SD)	p-value
Baseline	76 ± 8	–
T0 (immed.)	95 ± 9	< 0.001
T1 (1 min)	96 ± 10	< 0.001
T3 (3 min)	86 ± 7	< 0.01
T5 (5 min)	80 ± 8	0.03

Table 7: Mean arterial pressure over time

Time point	MAP (mm Hg, mean ± SD)	p-value
Baseline	91 ± 7	–
T0 (immed.)	113 ± 8	< 0.001
T1 (1 min)	116 ± 9	< 0.001
T3 (3 min)	102 ± 8	< 0.01
T5 (5 min)	97 ± 7	0.02

Table 8: Ephedrine requirement

Intervention	Patients (n, %)
Ephedrine	10 (17 %)

Table 9: Atropine requirement

Intervention	Patients (n, %)
Atropine	2 (3 %)

Table 10: Incidence of nasal mucosal bleeding

Event	Patients (n, %)
Nasal mucosal bleeding	3 (5 %)

Table 11: Incidence of minor nasal trauma

Event	Patients (n, %)
Minor nasal trauma	2 (3 %)

Table 12: Arrhythmias observed

Event	Patients (n, %)
Arrhythmias	0 (0 %)

Table 1 showed a balanced cohort with mean age 42.5 years, slight male predominance (34/26), and normal mean BMI of 23.9 kg/m². Table 2 indicated stable baseline hemodynamics with heart rate 75 bpm, SBP 122 mm Hg, DBP 76 mm Hg, and MAP 91 mm Hg. Table 3 confirmed rapid nasotracheal intubation, with mean time 14.8 s and 92 percent first-attempt success. Table 4 documented a significant heart rate rise to 93 bpm immediately post-intubation ($p < 0.001$). Table 5 showed heart rate peaking at 97 bpm one minute after intubation ($p < 0.001$). Table 6 revealed partial recovery to 85 bpm at three minutes ($p < 0.01$). Table 7 demonstrated near-baseline heart rate of 81 bpm at five minutes ($p = 0.04$). Table 8 depicted systolic blood pressure climbing to 155 mm Hg at one minute and then falling toward baseline by five minutes. Table 9 mirrored this pattern for diastolic blood pressure, with maximal elevation to 96 mm Hg at one minute. Table 10 confirmed mean arterial pressure peaked at 116 mm Hg one minute post-intubation and declined thereafter. Table 11 showed that 17 percent of patients required ephedrine and 3 percent atropine per protocol. Table 12 indicated minimal adverse events, with 5 percent experiencing nasal mucosal bleeding, 3 percent minor trauma, and no arrhythmias.

Discussion

The present study demonstrates that nasotracheal intubation under standardized general anaesthesia provokes a pronounced but transient sympathetic response. Heart rate increased by an average of 18 beats per minute immediately after intubation and peaked at 22 beats per minute one minute later [6]. Systolic, diastolic, and mean arterial pressures followed a similar trajectory, rising by approximately 28, 20 and 25 mm Hg respectively at their maximum. These findings confirm that nasal tube passage and laryngoscopy together stimulate both mucosal stretch receptors and pharyngeal mechanoreceptors, resulting in a rapid catecholamine surge [7]. By three minutes post-intubation, vital signs had begun to return toward baseline and by five minutes were within 10 percent of pre-induction values. This temporal pattern underscores the importance of vigilant monitoring in the first minutes after intubation [8].

Our observed magnitude of haemodynamic change aligns with earlier reports of orotracheal intubation, yet the nasal route may introduce additional stimulus from narrower nasal passages and increased manipulation [9]. The mean intubation time of 14.8 seconds and high first-attempt success rate suggest that operator proficiency was not a major contributor to exaggerated responses [10]. Ten patients required ephedrine for hypotension and two required atropines for bradycardia, indicating that even protocolized management may not fully prevent blood pressure and heart rate swings [11]. No arrhythmias or serious adverse events occurred, and minor nasal bleeding or superficial trauma were limited to under 5 percent of patients, attesting to the overall safety of the procedure when performed by experienced practitioners [12,13].

Clinically, these results highlight a window of heightened cardiovascular risk in the first one to three minutes after nasotracheal tube placement. Patients with coronary artery disease, left ventricular dysfunction or uncontrolled hypertension may benefit from preventive measures such as deeper anaesthetic depth, small boluses of short-acting beta blockers or opioids immediately prior to intubation, and readiness to administer vasoactive agents. For low-risk patients, routine monitoring and fluid optimization appear adequate to manage the transient changes without additional pharmacologic intervention.

Limitations of our study include its single-center design and exclusion of patients with significant cardiopulmonary comorbidities. The relatively young and healthy study population may have blunted the severity of haemodynamic responses compared with a more diverse cohort. Future research should examine different premedication regimens, alternative tube lubrication techniques or anesthetic adjuncts to further attenuate the intubation response. Studies in high-risk populations will also be valuable to guide tailored airway management strategies.

Conclusion

Nasotracheal intubation under general anaesthesia induces a rapid rise in heart rate and blood pressure that peaks at one minute and largely resolves by five minutes. While generally well tolerated in healthy adults, these transient cardiovascular surges warrant proactive monitoring and management, especially in patients with underlying cardiac disease. Employing targeted preventive measures and maintaining readiness to treat hypotension or bradycardia can enhance the safety of nasotracheal intubation in clinical practice.

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