Exploring Hemodynamic Alterations: The Impact of Post-Induction and Pre-Intubation Urinary Catheterization in General Anesthesia: A Randomized Controlled Trial

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Abstract- General anesthesia with endotracheal intubation is essential for major surgical procedures; however, the associated laryngoscopy and intubation elicit significant hemodynamic responses, posing risks particularly in susceptible patients. Although noxious stimuli are traditionally deferred until after securing the airway, modern anesthetic techniques and depth monitoring may allow for safe pre-intubation interventions that improve operating room efficiency. This study aimed to evaluate whether urinary catheterization, a minor but potentially painful procedure, performed after anesthetic induction and prior to intubation, induces significant hemodynamic alterations. In this prospective, single-blind, randomized controlled trial, 60 adult patients undergoing elective open abdominal surgery were randomly assigned to either an intervention group (catheterization after induction and before intubation) or a control group (no catheterization). Hemodynamic variables—systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR)—and bispectral index (BIS) were recorded at five predefined time points: before induction, after induction, before and after catheterization, and post-intubation. No significant intergroup differences were observed in HR, DBP, MAP, or BIS at any time point except after intubation. Post-intubation, the control group demonstrated significantly higher SBP and MAP compared to the intervention group (SBP: 130.9±11.8 vs. 122.5±10.4 mmHg, *P*=0.027; MAP: 99.2±8.3 vs. 91.7±8.1 mmHg, *P*=0.032). BIS values remained within the target range (40-60) in both groups, indicating consistent anesthetic depth. Urinary catheterization performed after induction and before intubation does not cause significant hemodynamic instability or alter the depth of consciousness. This finding supports the safe incorporation of minor procedural steps prior to airway instrumentation, potentially enhancing intraoperative workflow without compromising patient safety. Validation in broader patient populations is warranted.

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Introduction

General anesthesia with endotracheal intubation is indispensable for ensuring adequate ventilation and optimal surgical conditions during major surgeries (1). However, laryngoscopy and endotracheal intubation provoke intense stimulation and marked hemodynamic changes, posing risks, especially in vulnerable patient

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populations (2-4). It is imperative to recognize that some hemodynamic alterations may significantly affect patient outcome in both the intraoperative and postoperative periods (5). Traditionally, anesthesiologists have avoided applying potentially painful stimuli until after securing the airway with endotracheal intubation to minimize patient stress (6). Advances in intravenous and volatile anesthetics now allow faster transitions through the stages of anesthesia compared to earlier agents. With effective monitoring of anesthesia depth and a pre-emptive analgesia plan, it is feasible to consider applying minor painful stimuli before intubation without adverse cardiovascular effects (7-9).

Reducing anesthesia duration can enhance operating room efficiency and minimize the risks associated with prolonged exposure to anesthetics (10,11). Additionally, essential pre-operative procedures such as urinary catheter placement, which can be discomforting when performed while the patient is awake, are preferably conducted after induction of anesthesia. This approach not only avoids prolonging the anesthesia duration but also helps maintain optimal turnover rates in the operating theatre, thereby potentially impacting costs, surgeon satisfaction, and staff morale. Surgical efficiency in this context refers to improved operating room turnover times, reduced idle periods between cases, and streamlined workflow.

Although this practice is widely adopted, the timing of such interventions during the induction period has not been thoroughly evaluated. It has been hypothesized that minor noxious stimuli applied after induction but before airway manipulation could influence hemodynamic responses; however, the effects of catheterization timing in this context remain unclear. To our knowledge, no prior studies have specifically examined the hemodynamic consequences of urinary catheterization timing relative to endotracheal intubation under general anesthesia. Thus, we aimed to investigate whether benign but unpleasant stimuli could be applied after anesthetic induction but before endotracheal intubation, provided adequate depth of anesthesia is documented.

We hypothesized that minor painful stimuli would not induce significant hemodynamic instability when administered under adequate general anesthesia depth, as monitored by the bispectral index (BIS). BIS values between 40 and 60 are associated with a low likelihood of consciousness during general anesthesia, which helps attenuate responses to stimuli (12). Our goal was to assess whether benign painful stimuli could be safely administered before airway intubation, guided by anesthesia depth monitoring, without provoking hemodynamic instability.

In this randomized clinical trial, we examined hemodynamic changes induced by standardized Foley catheter insertion before endotracheal intubation, compared with the control group without urinary catheterization, among patients undergoing elective open abdominal procedures requiring general anesthesia.

Materials and Methods

Study design and participants

This study was designed as a prospective, single-blind, randomized controlled trial registered on the Iranian Registry of Clinical Trials (IRCT) under the ID (IRCT20170709034978N3). Written informed consent was obtained from all participants. We enrolled 60 ASA physical status I-II patients aged 18-65 years undergoing elective Laparotomy surgery requiring general anesthesia with endotracheal intubation from January 2020 to December 2020. Exclusion criteria were: emergency surgery, pregnancy, predicted difficult intubation, obesity (BMI> 35 kg/m2), cardiovascular disease, respiratory disease, renal or hepatic dysfunction, taking medications known to alter hemodynamic parameters, and history of alcohol/substance abuse.

Sample size calculation

The primary outcome of this study was the mean arterial pressure (MAP) measured one minute after endotracheal intubation. The sample size was calculated based on a pilot study in which the lowest MAP after induction was 75±12 mmHg in the control group and 85±10 mmHg in the intervention group. A 10 mmHg difference in MAP was considered clinically meaningful based on prior evidence that even moderate intraoperative hypotension is associated with increased risk of adverse outcomes, including renal and cardiac complications, particularly in surgical patients (13). With a significance level (alpha) of 0.05 and a power of 90%, the minimum required sample size was 25 patients per group to detect this difference. To accommodate potential dropouts, we enrolled 30 participants per group.

Intervention

Participants were randomly allocated into either an intervention group or a control group (30 per group) using a computer-generated randomization sequence. The randomization sequence was generated independently by a biostatistician who was not involved in any part of the clinical trial. To ensure allocation concealment, sequentially numbered, opaque, sealed envelopes

(SNOSE) were prepared by an independent research assistant. After obtaining written informed consent, the envelopes were opened in sequence by an anesthesiologist immediately before anesthesia induction, who was not involved in patient recruitment or outcome assessment.

All patients were continuously monitored using standard ASA-recommended modalities, including 5-lead electrocardiography, noninvasive blood pressure measurement, pulse oximetry, capnography, and bispectral index monitoring (BIS Vista, Covidien). Hemodynamic parameters (SBP, DBP, MAP, HR) and BIS values were recorded by an anesthesia nurse who was blinded to group allocation. This ensured an unbiased outcome assessment.

Anesthesia was uniformly induced in both groups using intravenous administration of midazolam 0.03 mg/kg, fentanyl 3 µg/kg, sodium thiopental 5 mg/kg, and atracurium 0.5 mg/kg after 3 minutes of preoxygenation with 100% oxygen. Subsequently, participants were manually ventilated with 100% oxygen during the period between anesthetic induction and securing the airway with endotracheal intubation.

In the intervention group, Foley catheter insertion was performed under sterile conditions once BIS values reached the 40-60 range, indicating adequate depth of anesthesia. A standard-sized Foley catheter (16 Fr for males, 14 Fr for females) was used uniformly based on gender to minimize variability in urethral stimulation. No adjustments were made based on individual body size. Additionally, sterile 2% lidocaine gel (10 mL) was applied to the catheter tip prior to insertion in all cases to provide local lubrication and minimize urethral discomfort, consistent with standard anesthetic protocols. The catheter used in this study was the *Bardex® Lubricath® Foley Catheter* (manufactured by Bard Medical). Also ,catheterization for the control group was performed after intubation.

Endotracheal intubation via direct laryngoscopy using an appropriately sized cuffed tube was performed and confirmed with capnography 4 minutes after the administration of atracurium in both groups.

Hemodynamic parameters, including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), and depth of anesthesia, were measured at five time points:

1. Immediately before induction (baseline)
2. Immediately after induction
3. Immediately before catheter insertion (intervention group) or at the equivalent time point in the control group
4. Immediately after catheter insertion (intervention group) or at the equivalent time point in the control group
5. One minute after endotracheal intubation

Statistical analysis

Statistical analysis was performed using SPSS Version 25. Normality was assessed using the Shapiro-Wilk test. Between-group comparisons were conducted using the independent-samples t-test or Mann-Whitney U test, as appropriate. Within-group changes over time were analyzed using repeated measures ANOVA. Pairwise comparisons between specific time points were conducted to identify significant differences. A $P<0.05$ was considered statistically significant.

Results

A total of 60 patients scheduled for elective open abdominal surgery were enrolled between January 2020 and December 2020. Thirty patients were randomized to the intervention group, and thirty to the control group.

Participant demographics

The gender distribution was 18 males (50.0%) and 18 females (50.0%) in each study arm. There was no difference in gender frequency between the groups. The mean age was 51.3±13.6 years in the control group versus 50.9±14.2 years in the intervention group. There was no significant difference in mean age between the groups, as determined by an independent-samples t-test ($P=0.899$).

Hemodynamic changes

A comprehensive comparison of multiple physiological parameters, HR, SBP, DBP, MAP, and BIS, between the control and intervention groups is presented in Table 1. The analysis revealed variations in mean values and standard deviations for each parameter, along with significant differences within each group. Our analysis showed no significant differences in heart rate between the intervention and control groups during induction, stimulation, or intubation phases ($P>0.05$ at all intervals). Similarly, diastolic blood pressure did not exhibit significant variability between groups across all measurement intervals ($P>0.05$ at all time points).

However, when examining systolic blood pressure and mean arterial pressure, we observed no significant differences between the groups at baseline, after anesthetic induction, before Foley catheter insertion, or after Foley catheterization ($P>0.05$ at all time points). Notably, after endotracheal intubation, the control group showed statistically significant increases in mean SBP

(*P*=0.027) and MAP (*P*=0.032) compared with the intervention group (see Figures 1 and 2). The effect sizes for post-intubation differences in SBP and MAP between groups were moderate (Cohen's d=0.53 and 0.52, respectively). Moreover, the 95% confidence intervals for these differences did not cross zero, reinforcing the robustness and clinical relevance of the observed attenuation in the intervention group.

Regarding the depth of anesthesia, as quantified through mean bispectral index values, we found no significant differences between the study arms from baseline through post-intubation (*P*>0.05 at all time points).

However, when examining hemodynamic changes and levels of consciousness at the designated time intervals within each group and evaluating the significance of these changes between the two groups, the results are as follows (Table 1): Changes in HR were significant in both groups only after tracheal intubation. For the other indices examined—SBP, DBP, MAP, and BIS—the significant changes at the designated times were consistent across both groups. These indices showed substantial changes in both groups only after the induction of anesthesia and following tracheal intubation. No significant changes were observed during urinary catheterization in either group.

Table 1. Comparison of hemodynamic and anesthesia depth parameters between control and intervention groups across different perioperative time points. Data are expressed as mean±standard deviation.

		Intergroups comparison	Control group (n=30)	Intervention group (n=30)
Variable	**Measurement Time**	***P***	**Mean±SD**	**Mean±SD**
HR	**T1**	0.406	83.64 ± 11.58	86.22 ± 14.49
	T2	0.523	86.19 ± 16.06	88.58 ± 15.52
	T3	0.455	83.56 ± 11.52	85.89 ± 14.64
	T4	0.243	83.14 ± 11.7	87.22 ± 17.23
	T5	0.979	94.69 ± 12.17	94.78 ± 14.67
SBP	**T1**	0.895	131.97 ± 19.71	132.53 ± 15.7
	T2	0.734	107.14 ± 23.51	105.33 ± 21.26
	T3	0.071	107.31 ± 19.42	98.92 ± 19.37
	T4	0.089	105.92 ± 18.94	98.56 ± 17.22
	T5	0.027	140.97 ± 30.54	125.94 ± 25.55
DBP	**T1**	0.193	82.5 ± 9.76	85.61 ± 10.31
	T2	0.798	69.58 ± 15.34	68.64 ± 15.81
	T3	0.072	71.81 ± 14.42	65.58 ± 14.44
	T4	0.116	71.22 ± 14.51	65.78 ± 14.5
	T5	0.181	98.06 ± 20.85	91.86 ± 17.95
MAP	**T1**	0.958	104.47 ± 13.71	104.31 ± 12.79
	T2	0.890	84.86 ± 19.2	84.28 ± 16.5
	T3	0.071	86.36 ± 16.6	79.22 ± 16.4
	T4	0.083	85.56 ± 16.58	78.78 ± 16.08
	T5	0.032	117.83 ± 24.79	106.22 ± 20
BIS	**T1**	0.933	97.06 ± 1.09	97.03 ± 1.65
	T2	0.863	45.17 ± 15.85	44.5 ± 16.82
	T3	0.172	40.72 ± 17.27	46.53 ± 18.45
	T4	0.113	40.53 ± 17.48	47.08 ± 17.2
	T5	0.266	53.67 ± 19.95	58.83 ± 19.15

T1: baseline (before induction); T2: after induction; T3: pre-catheterization or equivalent; T4: post-catheterization or equivalent; T5: post-intubation. *P* indicates between-group differences based on independent-samples t-tests. Statistically significant differences were found only for SBP and MAP at T5, with moderate effect sizes (Cohen's d = 0.53 and 0.52, respectively); their 95% confidence intervals excluded zero

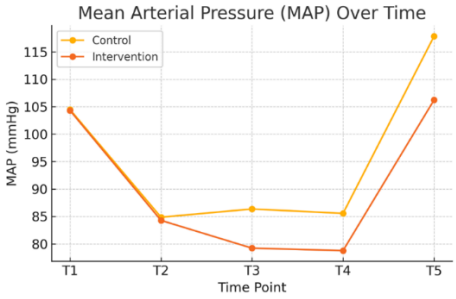

Figure1. Mean arterial pressure (MAP) across perioperative time points

MAP values were measured at the same five time points. While values were comparable between groups at T1–T4 (P>0.05), the control group demonstrated a statistically significant increase in MAP after intubation (T5) compared to the intervention group (P=0.032). Error bars represent standard deviation

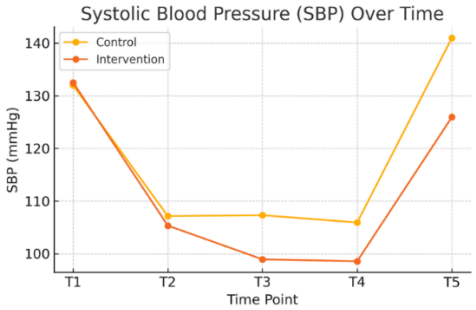

Figure2. Systolic blood pressure (SBP) trends across five time points in control and intervention groups

SBP measurements were recorded at five perioperative time points: baseline (T1), post-induction (T2), pre-catheterization (T3), post-catheterization (T4), and post-intubation (T5). The figure demonstrates a marked post-intubation increase in SBP, with significantly higher values in the control group. Data points represent group means. Error bars indicate standard deviation

Further, our repeated-measures ANOVA, evaluating changes in the mean line graph over procedural intervals, found no significant differences between the intervention and control groups over time for any of the measured parameters (P>0.05 for all). This was consistent with the mixed ANOVA results, which also showed no statistically significant differences in mean slope changes across distinct time points (P=0.747), as depicted in Chart 1.

In both groups, there were statistically significant increases in HR, SBP, DBP, MAP, and BIS from time point 3 (pre-intubation) to time point 5 (post-intubation) (P<0.001 for all), consistent with the expected hemodynamic response to laryngoscopy and tracheal intubation (see Figure 3 for HR trends). No significant differences were observed between time points 3 and 4 (before and after catheterization) in either group for any measured parameter (P> 0.05).

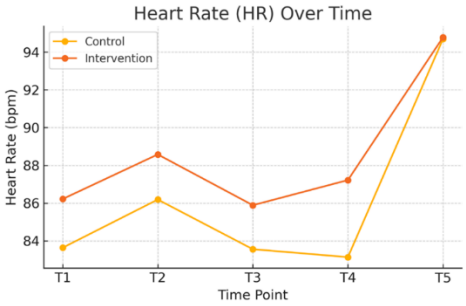

Figure 3. Heart rate (HR) changes over time

Heart rate increased progressively toward intubation, peaking at T5 in both groups. No significant differences were observed between groups at any time point, although the increase from T3 to T5 was significant within each group. Error bars show standard deviation

Discussion

In this randomized controlled trial, we aimed to explore the hemodynamic impact of pre-intubation urinary catheterization performed after induction of general anesthesia but before endotracheal intubation. Our findings suggest that introducing this benign but potentially discomforting stimulus did not result in significant hemodynamic instability or altered levels of consciousness, as monitored by the BIS.

When examining HR, SBP, DBP, and MAP, we observed no statistically significant differences between the intervention and control groups at most time points, particularly after anesthesia induction, before catheter insertion, and immediately after Foley catheterization (P>0.05). However, post-intubation, the control group exhibited a statistically significant increase in both SBP (P=0.027) and MAP (P=0.032) compared to the intervention group, suggesting that catheter insertion did not exacerbate the hemodynamic response to intubation. These observations are consistent with the broader understanding that endotracheal intubation elicits a significant sympathetic response, regardless of prior minor stimuli such as catheterization.

Although this finding may initially seem counterintuitive, it does not imply that catheterization directly suppressed hemodynamic responses. The lower SBP and MAP observed post-intubation in the intervention group may reflect a subtle blunting of the sympathetic surge induced by laryngoscopy, possibly due to the brief nociceptive stimulus that preceded it. However, this remains speculative. Since catheter insertion itself did not produce significant hemodynamic changes at the time of the intervention, it is more appropriate to interpret this as a secondary observation rather than a primary effect. Further investigation is warranted to explore whether pre-intubation interventions can modulate stress responses during airway manipulation.

A key aspect of our analysis was the intra-group comparisons at three critical transitions: from time point 1 to time point 2 (before and after the induction of anesthesia), from time point 3 to time point 4 (before and immediately after catheter insertion), and from time point 3 to time point 5 (before and after tracheal intubation). Among these, the comparison between time points 3 and 4—before and immediately after catheter insertion—was particularly important. Our results showed no significant changes in HR, SBP, DBP, MAP, or BIS in either group during this period, indicating that catheterization did not cause measurable hemodynamic alterations. This consistency across both groups reinforces the safety of performing this procedure during anesthesia induction.

In contrast, significant changes were observed in both groups from time point 1 to time point 2, corresponding to the period before and after anesthesia induction. This was expected as the induction process typically results in hemodynamic stabilization as the anesthesia takes effect. Similarly, from time point 3 to time point 5—before and after tracheal intubation—there were marked hemodynamic changes in both groups, reflecting the well-known hemodynamic response to intubation. Importantly, the pattern of these changes was consistent between the intervention and control groups, further confirming that the urinary catheterization did not introduce additional variability or instability in these parameters.

Urinary catheterization was employed in this study as a painful stimulus, in line with previous research by Wilson (12) and Pinar (14), which highlighted that discomfort associated with catheterization is influenced by factors such as the use of antiseptics and the passage of the catheter through the urethra, with variations depending on catheter size and gender. Despite this, our study suggests that, at sufficient anesthetic depth, as indicated by BIS values within the target range, discomfort from catheterization did not translate into significant hemodynamic changes.

On the other hand, our study found that MAP, SBP, DBP, HR, and BIS all increased during endotracheal intubation. Yakaitis' study (15) supports this finding, showing that higher doses of anesthetics are required to suppress movement and coughing during intubation than to prevent movement during skin incision. Additionally, studies by Yamashita (16) and Roizen (17) demonstrated that the MAC-BAR of inhaled anesthetics—the doses required to eliminate neuroendocrine responses to surgical pain—is much higher than the doses needed for immobility. These findings align with our results. However, repeated-measures ANOVA and mixed ANOVA analyses revealed similar trends in these parameters across time between the control and intervention groups. This indicates that the extent of increase in these values does not differ significantly,

regardless of whether noxious stimulation is present before tracheal intubation.

The absence of significant hemodynamic disturbances during urinary catheterization suggests that, under adequate anesthesia depth (BIS 40-60), minor painful stimuli can be administered without increasing the risk of cardiovascular instability. This aligns with our hypothesis that appropriate anesthesia management can mitigate the physiological response to such stimuli, potentially improving procedural efficiency in the operating room.

Few studies have directly examined the hemodynamic consequences of urinary catheterization under general anesthesia. Most of the literature has focused on major noxious stimuli, such as laryngoscopy, intubation, or surgical incision, while catheterization is typically considered a minor, routine intervention. Therefore, there is a lack of randomized controlled trials addressing its timing and cardiovascular impact. Isolated case reports describe conflicting autonomic responses (e.g., vasovagal reactions versus mild sympathetic activation), but these are context-dependent and often linked to extreme bladder conditions or insufficient anesthesia depth. Our findings align with the limited data available, indicating that, under adequate anesthesia depth, catheterization induces only subtle hemodynamic effects (18,19).

There were no major limitations in this study, aside from occasional inaccuracies in blood pressure monitoring and prolonged urinary catheterization due to anatomical difficulties, which necessitated the exclusion of some participants. Our study focused exclusively on hemodynamic parameters and the depth of anesthesia in the control and intervention groups. To gain a more comprehensive understanding of the neuroendocrine responses, we recommend that future studies measure epinephrine and norepinephrine levels at specified time points as surrogates of sympathetic outflow. This would provide deeper insights into the physiological effects of urinary catheterization and other stimuli during anesthesia induction.

This randomized controlled trial demonstrated that pre-intubation urinary catheterization, when performed under adequate general anesthesia as monitored by BIS values between 40 and 60, does not induce significant hemodynamic changes or alter levels of consciousness. Intra-group and inter-group comparisons confirmed the absence of notable cardiovascular responses to catheterization. Interestingly, the intervention group exhibited significantly lower SBP and MAP after intubation, though the exact mechanism remains to be investigated. These findings support the safe incorporation of minor procedural interventions during the anesthetic induction phase to enhance operating room workflow without compromising patient stability. However, broader application of this approach should be interpreted cautiously until it is validated across diverse surgical settings and patient populations.

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