Abstract
Background and objective: To compare the effect of prophylactic administration of normal saline against the hypotensive effect of propofol in female patients booked for gynecological procedures.
Method: Sixty nine ASA (I, II) patients were randomly allocated into two groups, group 1 received 5 mL.kg\(^{-1}\) of 0.9% normal saline 10 minutes before induction, whereas group 2 received nothing (control). Anesthesia was induced with propofol and fentanyl then maintained with propofol and remifentanil. We measured hemodynamic variables pre and post general anesthesia induction.
Results: Both groups had significant drops in post induction mean arterial blood pressure (P < 0.001). Also both groups had significant drops in post induction heart rate ((P < 0.02 in sample group and P < 0.001 in control group), and 35% of patients in the control group had more than 25% drop in the pre induction mean arterial blood pressure, compared with only 17% of patients in the saline group (P < 0.04).
Conclusion: The prophylactic administration of normal saline could decrease the percentage of patients who had a significant drop in their blood pressure after propofol induction of general anesthesia.

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Normal saline is a cheap and safe crystalloid fluid that could decrease this undesirable side effect of propofol usage. The study aimed to determine the efficacy of normal saline administration before GA induction against the anticipated hypotensive effect of propofol.

**Method**

After obtaining approval from the hospital ethics committee and informed consent from the patients, 69 female ASA (I, II) patients undergoing elective gynecological procedures were enrolled in this study. Patients did not receive any premedication and were randomized into two groups based on computer generated numbers: group 1 received 5 mL.kg⁻¹ 0.9% normal saline 10 minutes before induction and group 2 did not receive any fluid. The investigators who collected vital signs from the patients were blinded to the randomization process. Exclusion criteria were age below 18 years and pregnancy.

Blood pressure was measured pre induction and post induction (3 minutes post airway manipulation) using a non-invasive automated method with appropriate-sized cuff at admission area. A 25% drop in mean arterial blood pressure was considered to be significant.

All patients were anesthetized in a standard protocol by a senior anesthesiologist. GA was induced with propofol 1% 2-2.5 mg.kg⁻¹ over 30 seconds, fentanyl 2 µg.kg⁻¹, and rocuronium 0.6 mg.kg⁻¹ to facilitate tracheal intubation. Total intravenous anesthesia (TIVA) started with propofol and remifentanil immediately upon loss of verbal contact with the patients.

The number of patients required for the study was calculated from previous studies. Using Altman’s nomogram for a study with a power of 0.90, 65 patients would be needed to demonstrate a difference in the mean arterial blood pressure of 15 mm Hg, when the level of statistical significance was set to 5%.

Parametric data were analyzed with t-test. Non-parametric data were analyzed with Mann-Whitney U test. Categorical data were analyzed by chi square test or Fisher’s exact test, as appropriate. A P value of less than 0.05 was considered significant.

**Results**

There were 35 patients in group 1 and 34 patients in group 2. The two groups were comparable with respect to age, weight and the physical classification system. Table 1 showed patient characteristic data. There were no statistical differences in baseline blood pressures between the two groups (Table 2).

The mean pre induction arterial blood pressure in the study group was 90 mm Hg ± 8.8; and dropped to 76 mm Hg ± 10 after induction. In the control group it was 95 mm Hg ± 15 and fell to 73 mm Hg ± 13. These changes were significant (P < 0.001). Furthermore, both groups had significant drops in post induction heart rate (P < 0.02 in sample group and P < 0.001 in control group). Figure 1 summarized these changes.

<table>
<thead>
<tr>
<th>Table 1 Patients’ demographic features.</th>
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<td>Sample group (35 patients)</td>
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<tr>
<td>Age in years (mean ± SD)</td>
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<tr>
<td>Weight in kg (mean ± SD)</td>
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<tr>
<td>ASA I</td>
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<td>ASA II</td>
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<th>Table 2 - Patients’ vital signs at admission.</th>
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<td>Sample group</td>
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<td>Systolic blood pressure, mm Hg (mean ± SD)</td>
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<td>Mean Arterial pressure, mm Hg (mean ± SD)</td>
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<td>Heart rate (mean ± SD)</td>
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Figure 2 showed the percentage of patients who had more than a 25% drop in mean arterial blood pressure after propofol induction. Twelve patients in the control group had a significant drop, compared with only 5 patients in the saline group (P < 0.04).
The use of crystalloid fluid preload to prevent hypotension after propofol induction of anesthesia hasn’t been thoroughly evaluated. In fasting patients, fluid administration is more likely to remain intravascular than to be distributed into the interstitial and the intracellular spaces, thus, raising venous return and increasing cardiac output that may attenuate hypotensive effect.

The hypotensive property of propofol is dose dependent and more obvious in elderly patients. There is some evidence that the use of fentanyl with propofol induction may potentiate the hypotensive effect of propofol. These factors are unlikely to bias our results.

Both groups in our study had anesthesia according to standard protocol by one anesthesiologist. There were no significant differences in age as well as propofol, fentanyl and ramifentanil dose between the sample and control group.

Although propofol-induced hypotension is usually well tolerated in patients with stenotic coronary artery disease, it may occasionally cause cardiovascular collapse and death. Propofol anesthesia is associated with a significant decrease in cardiac oxygen consumption, and the global myocardial oxygen supply-demand ratio is therefore well-preserved. However, despite an increase in the global oxygen supply-demand ratio, arterial hypotension may occasionally lead to local myocardial ischemia in areas supplied by a stenotic artery. Arterial hypotension may also jeopardize cerebral perfusion in patients with a significant stenosis in carotid or intracerebral arteries. These serious complications are definitely more important when the mean arterial blood pressure drop significantly, especially in such medically compromised patients. Vital organs’ auto-regulation is often maintained within mean arterial blood pressure of 60-160 mm Hg; therefore, any critical drop below this level will be hazardous.

Different authors reported variable results on the effect of propofol on the heart rate. Our data was consistent with Michelsen et al. study where induction of anesthesia with propofol was followed by a decrease in heart rate. In contrast, Gamlin et al. found no effect of propofol on heart rate, whereas Turner et al. found increase in heart rate. These conflicting results can be explained by the different methods of anesthesia maintenance performed in these studies.

In summary, we did not find administration of 0.9% normal saline before propofol induction of GA in gynecological procedures to be valuable in preventing a drop in post induction mean arterial blood pressure. However, it was effective in decreasing the percentage of patients with a significant drop as defined by 25% decrease in pre induction mean arterial pressure.

References