Effects of prophylactic atropine in prevention of spinal anesthesia induced hypotension and bradycardia in geriatrics undergoing urological surgeries at a resource limited setting in Central Ethiopia, 2018; prospective cohort study.

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Research

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Abstract

Background: Spinal anesthesia induced hypotension and bradycardia are common and hazardous in elderly patients. Many techniques are being tried to prevent and treat these problems even if there is a controversy. The effects of prophylactic atropine on prevention of spinal anesthesia induced hypotension and bradycardia in geriatrics for urologic surgeries are not well-established.

Objective: To assess the effects of prophylactic atropine in prevention of spinal anesthesia induced hypotension and bradycardia in geriatrics undergoing urological surgeries at a resource limited setting in Central Ethiopia from December 1, 2017 to February 28, 2018 G.C.

Methods: This is a prospective cohort study that recruits 76 patients who underwent elective urological surgeries. Independent t-test and Manny Whitney tests were used for numeric data while Chi-Square or Fisher exact test was used for categorical variables. P-values < 0.05 were considered as statistically significant.

Results: There was no significant difference in baseline heart rate, mean arterial pressure, type & duration of surgery and total fluid administrations. There was a statistically significant difference in mean heart rate and mean arterial pressure at different times of measurement between the exposed and un-exposed groups. Total one hour vasopressor consumption was minimal in the exposed group (P = 0.038).

Conclusion: Prophylactic atropine with in one minute of induction of spinal anesthesia in elderly patients undergoing urological surgery might reduce the incidence of hypotension and bradycardia.

Background

Spinal anesthesia (SA) is associated with many complications among which the most common side effects are hypotension and bradycardia [1-3].

Spinal anesthesia induced hypotension is believed to occur due to two possible mechanisms. The first and widely accepted mechanism is systemic vasodilation induced by sympathetic blockade, resulting in venous pooling of blood and reduction in systemic vascular resistance [4]. This could be treated by administering peripheral vasoconstrictors thereby increasing the systemic vascular resistance and facilitating the venous return [5].

The second cause is believed to be the blunted reflex tachycardia. This may result from the blockade of cardioaccelerator sympathetic fibers at T1 to T4, and due to the “reverse” of the Bainbridge reflex. To prevent this mechanism, prophylactic use of intravenous (IV) atropine might have importance [6].

Currently, different techniques are being used for the prevention of hypotension and bradycardia which include pre or co-loading of IV fluid, vasopressors, and physical methods such as table tilt, leg binders, and compression devices [7]. However, none of these techniques alone are effective and there is a search
for a technique or combinations of techniques for the proper prevention of spinal anesthesia induced hypotension and bradycardia [8].

Elderly patients are prone to spinal anesthesia induced hypotension and bradycardia than young adults. This is because they may have coexisting degenerative cardiovascular diseases with deranged reflex compensatory mechanisms for hypotension and bradycardia. They are also sensitive for both fluid resuscitation and sympathomimetic drugs. So, aggressive fluid therapy may end up with pulmonary edema and congestive heart failure. Vasoactive agents may also increase myocardial workload and ischemic injuries in elderly [9-11].

Atropine is more easily available and cost effective than vasopressors and IV fluids. Therefore, the results of the current study could be easily applicable in any resource limited hospital setups. Researches showing the effect of atropine to prevent spinal anesthesia induced hypotension are limited. As per the researcher’s knowledge, there is no published study in Ethiopia that assessed the effect of atropine in preventing hypotension and bradycardia.

The aim of the current study was to assess the effects of prophylactic atropine in prevention of spinal anesthesia induced hypotension and bradycardia in geriatrics undergoing urological surgeries at a resource limited setting in Central Ethiopia.

**Methods**

**Study setting, design, period and population**

A Hospital-based prospective cohort study was conducted at Tikur Anbesa Specialized Hospital, in Central Ethiopia from December 1, 2017 to February 28, 2018. Patients with age 50 years and above, who have undergone elective urologic surgeries under spinal anesthesia at the study setting were the source population. While, selected patients with age 50 and above, American Society of Anesthesiologists Physical status I and II, who undergone elective urologic surgeries under spinal anesthesia during the study period were the study population.

Patients with Failed spinal, Heart block greater than first degree, left bundle branch block, hypertension (SBP > 140 mmHg or DBP > 90 mmHg), preoperative tachycardia (HR > 120 bpm), β-adrenergic blockers, combinations of spinal block with other type of anesthesia, prior anticholinergic use within 2 hours before entering the operating room, supplementation of strong opioids [6] were excluded in the study.

**Sample size determination**

By using Epiinfo-7 statistical calculator for independent cohort and considering one-to-one ratio of the exposed and un-exposed groups with the assumption of a P value <0.05 as statistically significant and a power of 80%, the sample size was calculated to be 68 (34 patients each). Adding 10% nonresponse rate the total sample size was 76 (38 subjects to each group). The sample size was calculated based on a
previous study in Nepal that showed the incidence of hypotension in atropine exposed and un-exposed groups to be 5% and 60% respectively [6].

**Sampling techniques and procedures**

Patients who underwent elective urological surgeries were recruited into the study by using a systematic random sampling technique. Within 105 patients estimated to undergo elective urological surgeries during the study period, 76 participants were included. The first participant was selected by the lottery method. Then 2 patients for every 3 consecutive patients were included until the required sample size was met. The patients were grouped based on their exposure to atropine.

**Operational definition**

**Spinal Anesthesia**: Administration of local anesthetics into the subarachnoid space.

**Elderly**: age greater than or equal to 50 years [6, 12,13].

**Hypotension**: systolic BP < 90mmHg or diastolic BP < 60mmHg [6].

**Hypertension**: greater than 20% increase from baseline BP [14].

**Bradycardia**: HR < 50 beats per minute [6].

**Tachycardia**: greater than 20% increase from baseline heart rate [6, 15]

**Data collection procedures and tools**

Data was collected using a prepared checklist starting from immediate preoperative to 1st intra/postoperative hour by four trained anesthetists. Mean arterial pressure and heart rate were recorded at 0 (baseline), 5th, 10th, 15th, 20th, 30th, 40th, 50th, and 60th minutes intraoperatively. Requirement for vasopressors was studied for the 1st one-hour intra/postoperatively.

**Data quality control**

Prior to the actual data collection pretest was done on 10% of the sample size to see the effectiveness of the data collection tool. Collected data were checked for completeness, accuracy, and clarity.

**Data processing and analysis**

Data were checked manually for completeness, coded and entered into SPSS version 23 computer program. Descriptive statistics were used to summarize data. Chi-square or Fisher exact test was used for discrete variables and student's t-test was used for comparing numerical variables of normally distributed data. Manny Whitney U test was used for skewed data. A P-value of less than 0.05 was considered as statistically significant.
Ethics approval and consent to participate

Ethical clearance was obtained from the Institutional Review Board of the College of health science, Addis Ababa University. Written consent was taken from each study participants. Confidentiality and the patients’ right to withdraw from the study were maintained throughout the study period.

Results

Comparison of Demographic data

A total of 76 (38 exposed and 38 un-exposed) patients were included in this study. There was no statistically significant difference in age between exposed and un-exposed groups with a P value of 0.656. The two groups were comparable in other demographic data (Table 1).

Table 1: Comparison of demographic data between the two groups: Tikur Anbesa Specialized Hospital, December 1, 2017 - February 28, 2018

|                      | Exposed (n=38) | Un-exposed(n=38) | P value |
|----------------------|---------------|------------------|---------|
| Age in years (Mean +SD) | 62.95 ± 7.392 | 62.21 ± 6.975    | .656    |
| BMI in Kg/m² (Mean +SD) | 21.08 ± 2.136 | 20.11 ± 2.275    | .058    |
| Sex                  |               |                  |         |
| Male [f (%)]         | 28 (73.7%)    | 23 (60.5%)       | .222    |
| Female [f (%)]       | 10 (26.3%)    | 15 (59.5%)       |
| ASA class            |               |                  |         |
| I [f (%)]            | 20 (52.6%)    | 21 (55.3%)       | .818    |
| II [f (%)]           | 17 (47.4%)    | 18 (44.7%)       |
| Type of surgery      |               |                  |         |
| TURP [f (%)]         | 15 (38.5%)    | 11 (28.9%)       | .626    |
| TURBT [f (%)]        | 11 (28.9%)    | 13 (34.3%)       |
| URS [f (%)]          | 12 (31.6%)    | 14 (36.8%)       |

ASA: American Society of Anesthesiologists, BMI: Body Mass Index, TURP/BT: Transurethral Resection of prostate/ Bladder Tumor; URS: Ureteroscopic Removal of stone; f: frequency
Comparison of baseline hemodynamics and fluid management

There was no significant difference in the type, baricity and volume of local anesthetic used, size of spinal needle and level of autonomic block between the two groups. Preload, total IV fluids in the first hour and baseline hemodynamics were also comparable (Table 2).

Table 2: Comparison of baseline hemodynamics and fluid management between the two groups: Tikur Anbesa Specialized Hospital, December 1, 2017 – February 28, 2018

|                         | Exposed (n=38) | Un-Exposed (n=38) | P value |
|-------------------------|----------------|-------------------|---------|
|                         | Mean | Std. Deviation | Mean | Std. Deviation |         |
| Preload (ml)*           | 444.74 | 148.319 | 455.26 | 169.972 | .774 |
| Total fluid in 1st hrs. (ml)* | 1039.47 | 440.847 | 1221.05 | 559.033 | .120 |
| Duration of surgery (minute)* | 64.21 | 20.253 | 65.53 | 24.489 | .799 |
| Baseline HR (bpm)*      | 76.16 | 11.988 | 78.18 | 11.359 | .452 |
| Baseline MAP (mmHg)*    | 94.66 | 7.528 | 94.16 | 6.310 | .755 |
| Volume of LA in ml [median (IQR)]** | 3.5 (3.0-3.5) | 3.0 (3.0-3.5) | .507 |

HR: Heart Rate, MAP: Mean Arterial Pressure. *: Independent samples t test, **: Mann Whitney U test.
The mean baseline heart rate was comparable between the two groups. There was a statistically significant difference in mean heart rate records between exposed and un-exposed groups at all measurements in the 1\textsuperscript{st} hour (Table 3).

As compared to the baseline, mean heart rate was significantly low in un-exposed group with the lowest mean HR of 65.84 ± 8.964 bpm at 15\textsuperscript{th} minute. While in exposed group; all the mean records of HR were higher than the baseline with the highest to be 93.63 ± 16.210 at the 10\textsuperscript{th} minute (Table 3).

**Table 3:** Comparison of mean heart rate within and between the two groups: Tikur Anbesa Specialized Hospital, December 1, 2017 - February 28, 2018

|          | Exposed (n=38) |                  | Un-exposed (n=38) |                  |         |
|----------|---------------|------------------|-------------------|------------------|---------|
|          | Mean +SD      | P value          | Mean +SD          | P value          | P value |
|          |               | (within group)*  |                   | (within group)*  | (between groups)** |
| Baseline HR | 76.16+11.9  | ...              | 78.18+11.3         | ...              | .452    |
|           | 88            |                  | 59                |                  |         |
| HR 5     | 89.18+10.2  | < .001           | 73.82+9.54        | .003             | < .001  |
|           | 21            |                  | 3                 |                  |         |
| HR 10    | 93.63+16.2  | < .001           | 66.53+10.8        | < .001           | < .001  |
|           | 10            |                  | 30                |                  |         |
| HR 15    | 85.95+11.1  | < .001           | 65.84+8.96        | < .001           | < .001  |
|           | 16            |                  | 4                 |                  |         |
| HR 20    | 85.18+11.9  | < .001           | 69.34+12.3        | .002             | < .001  |
|           | 82            |                  | 12                |                  |         |
| HR 30    | 84.29+12.2  | < .001           | 70.11+12.6        | .007             | < .001  |
|           | 56            |                  | 12                |                  |         |
| HR 40    | 81.82+12.6  | .003             | 69.89+12.0        | .004             | < .001  |
|           | 23            |                  | 78                |                  |         |
| HR 50    | 77.89+7.77  | .314             | 68.45+7.99        | < .001           | < .001  |
|           | 7             |                  | 9                 |                  |         |
| HR 60    | 76.58+9.10  | .806             | 69.92+8.26        | < .001           | .001    |
|           | 5             |                  | 4                 |                  |         |

HR: Heart Rate, SD: Standard Deviation. *: Paired sample t test, **: Independent samples t test
Comparison of Mean MAP

The mean baseline MAP was comparable between the two groups (94.66 ± 7.528 in exposed and 94.16 ± 6.310 in un-exposed). MAP in exposed group was significantly greater than the non-exposed group throughout the whole times of measurement with P values < 0.001 (Table 4).

**Table 4:** Comparison of Mean MAP within and between the two groups: Tikur Anbesa Specialized Hospital, December 1, 2017 – February 28, 2018

|                  | Exposed (n=38) | Un-exposed (n=38) | P value (between groups)** |
|------------------|----------------|-------------------|---------------------------|
|                  | Mean +SD       | P value (within group)* | Mean +SD       | P value (within group)* |
| Baseline MAP     | 94.66+7.528    | ...               | 94.16+6.310    | ...               | .755 |
| MAP 5            | 94.82+10.717   | .922              | 79.95+12.559   | < .001           | <.001 |
| MAP 10           | 94.97+9.048    | .843              | 74.32+8.690    | < .001           | <.001 |
| MAP 15           | 94.55+11.086   | .955              | 73.21+10.351   | < .001           | <.001 |
| MAP 20           | 94.21+11.630   | .830              | 74.55+8.186    | < .001           | <.001 |
| MAP 30           | 90.32+7.447    | .010              | 74.61+8.189    | < .001           | <.001 |
| MAP 40           | 90.47+9.443    | .015              | 77.53+8.320    | < .001           | <.001 |
| MAP 50           | 90.55+8.193    | .010              | 78.74+8.275    | < .001           | <.001 |
| MAP 60           | 91.68+8.650    | .110              | 80.58+8.314    | < .001           | <.001 |

MAP: Mean Arterial Pressure, SD: Standard Deviation. *: Paired sample t test, **: Independent samples t test

Comparison of Vasopressor consumption

The frequency of vasopressor consumption was significantly different between the exposed and un-exposed groups with a P value of 0.038 (Figure 3). The overall occurrence of hypotension (BP<90/60mmHg) was 3(7.9%) and 12(31.6%) in exposed and un-exposed groups respectively. There were four patients (10.5%) with heart rate <50 bpm in un-exposed group, but this was not statistically significant (Figure 3). A Chi-square test for independence (with Yates Continuity Correction) indicated that there is a significant association between atropine prophylaxis and intraoperative vasopressor consumption, $\chi^2 (1, n = 76) = 4.290$, P = 0.038, phi = 0.272.

Discussion
Hypotension and bradycardia are the two most common complications of spinal anesthesia [1-2]. There are various techniques to prevent these complications which may include fluid loading, vasopressors, leg up positioning, low dose local anesthetic combined with opioid additives and others [14,16-17]. However, none of these techniques are sufficient for proper prevention of hypotension and bradycardia after spinal anesthesia [3, 8, 16, 18].

Systemic vasodilation induced by sympathetic blockade, resulting in venous pooling of blood and reduction in systemic vascular resistance, has been regarded as the predominant mechanism for hypotension induced by SA [3]. However, the absence of reflex tachycardia after spinal anesthesia induced hypotension has been observed to contribute by Hwee H. Lim et al. [2]. We postulate that the absence of reflex tachycardia may be an important component in the pathogenesis of hypotension induced by SA in addition to effects of venous and arterial dilation.

The current study showed that there was a statistically significant difference in heart rate between the exposed and unexposed groups throughout all the measurements with P values less than 0.001 (Table 3). The increase in HR was statistically significant but no patient required treatment for tachycardia. Compared with the baseline, the heart rate was significantly decreased in the non-exposed group and significantly increased in exposed group (Figure 1). The possible justification for this difference could be due to the vagolytic effect of atropine which increased the heart rate in the exposed group.

These findings were in line with other studies. A Randomized Controlled Trial done in Nepal in 2015 compared mean heart rate between a group of 20 patients given a single dose of 0.6 mg atropine and a placebo group of equal number. This study showed that the mean heart rate was significantly different between the atropine premedicated and the control groups at all the times they measure [6].

Another RCT that compares the prophylactic effect of atropine and ephedrine with a placebo group also showed that as compared to the placebo, mean heart rate was significantly increased in atropine and ephedrine groups at all the times. This study also showed that maximum heart rate in atropine group was 89.30 ± 14.62 bpm at 5th minute [19].

A study done in Chai Wan, Hong Kong to assess the effect of different doses of atropine in prevention of spinal anesthesia induced hypotension showed that the heart rate in both small dose (5mcg/Kg) and large dose (10mcg/Kg) groups was significantly different from that of the placebo group [2].

In contrast to our findings of increased mean heart rate in the exposed group in all times, a study in Seoul Paik Hospital of Inje University, Republic of Korea showed that as compared to the baseline, there was a significant decrease in heart rate at the 5th, 10th, 15th, 20th, 25th, and 30th minutes in the atropine group [17]. This difference may be due to the administration of dexmedetomidine for sedation at a loading dose of 0.6 mcg/kg for 10 minutes followed by an infusion at 0.25 mcg/(kg h) in their study. Dexmedetomidine decreases sympathetic out flow from the central nervous system which may result in bradycardia.
The current study showed that the mean MAP was significantly different between the two groups throughout the whole measurements with \( P < 0.001 \) (Table 4). Compared to the baseline the mean MAP in un-exposed group showed a significant decrease. There was not a statistically significant difference in MAP from baseline until the 30\(^{th}\) minute in exposed group (Figure 2).

The possible explanation for the maintenance of MAP in the exposed group may be due to the increased heart rate which again increases the cardiac output and mean arterial pressure by the anticholinergic effect of atropine.

A study done in Nepal that compared patients premedicated with a fixed dose of 0.6mg atropine with a placebo showed a significant increase in MAP at the first minute in the atropine group and significant decrease at the 5\(^{th}\), 10\(^{th}\), 15\(^{th}\), 20\(^{th}\), 30\(^{th}\), 40\(^{th}\), 50\(^{th}\) and 60\(^{th}\) minute in placebo. To the contrary to the current study, they found that the mean MAP in the atropine group was not significantly different from the baseline throughout the whole times [6]. While our study showed a significant decrease in MAP at the 30\(^{th}\), 40\(^{th}\) and 50\(^{th}\) minute in exposed group. This difference may be due to variations in study designs.

Another RCT that compares atropine and ephedrine with a placebo showed MAP was comparable between atropine and ephedrine groups but in placebo group MAP was significantly low compared to other groups [19]. This is also in line with our findings showing a better maintenance of MAP after spinal anesthesia in the atropine exposed group than un-exposed group especially in the first 30 minutes.

A comparative study done by Jain and Kaushik in India that compared the effect of atropine and ephedrine in the prevention of spinal anesthesia induced hypotension in elderly patients who undergo surgery under spinal anesthesia showed that as compared to placebo group both atropine and ephedrine groups had a significantly different mean MAP from the placebo in all times they measured [20].

In the current study there was a significant difference in the occurrence of hypotension and frequency of vasopressor consumption between exposed and un-exposed groups (with \( P \) values of 0.021 and 0.038 respectively) (Figure 3). Adrenaline was required in 28.9\% of un-exposed and 7.9\% of exposed groups. This result was also in line with other studies.

Possible explanation for the reduced incidence of hypotension and decreased vasopressor consumption with the prophylactic use of atropine may be the help in preventing the blunted Bainbridge reflex thus increasing heart rate and cardiac output.

In a randomized controlled trial that compared occurrence of hypotension and vasopressor consumption in a group of patients given 0.6mg of atropine one minute after administration of spinal anesthesia with a placebo group the occurrence of hypotension and total vasopressor consumption was significantly different between the two groups. They showed that 60\% of placebo and 5\% of atropine groups developed hypotension that required mephentermine [6]. While in our study the occurrence of hypotension in un-exposed group was 31.6\% which is significant with a \( P \) value of 0.021. The variation in percentage may be due to difference in study design.
Another study done in India that compared vasopressor consumption between three groups of patients (atropine, ephedrine and placebo) showed that as compared to the atropine and ephedrine groups, there was a significant increase of vasopressor consumption in the placebo group ($P = 0.02$). Based on their findings mephentermine was required in more than 50 percent, 5 percent and 5 percent of the patients of the placebo, ephedrine and atropine groups respectively [20].

This result was also in line with a study done in Hong Kong that compared the incidence of hypotension between three groups of small dose atropine (5mcg/Kg), large dose atropine (10mcg/Kg) and placebo that showed the incidence of hypotension to be 76%, 50% and 40% in placebo, small dose and large dose atropine groups respectively [2].

In contrast to our findings, a study done at Seoul Paik Hospital of Inje University, Republic of Korea, in 2016 showed that the incidence of hypotension needing ephedrine treatment showed no significant difference between atropine pretreated and placebo groups. This variation could be due to the difference in study design and they used a fixed dose of 0.5mg atropine for all patients [17].

In the current study the occurrence of tachycardia had no significant difference between exposed and unexposed groups. This was also in line with other studies that showed the reflex tachycardia after atropine prophylaxis in patients undergoing urological procedures under spinal anesthesia is not statistically significant [2, 6, 19].

**Conclusion**

Prophylactic atropine with in one minute of induction of spinal anesthesia in elderly patients undergoing urological surgery might reduce the incidence of hypotension and bradycardia.

**Declarations**

**Funding:** Addis Ababa University

**Authors’ contributions**

Gelaw M. developed the proposal, analyzed the data and prepared the manuscript. Haddis L. Abrar M., Aregawi A, and Melese E. revised the proposal, involved in data analysis and manuscript preparation. All authors approved the final manuscript for publication.

**Ethics Declarations**

**Ethics approval and consent to participate**

See methods section.

**Consent for Publications**
Not applicable.

**Conflict of Interest**

The authors declare that there is no conflict of interests.

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**Availability of data and materials**

Data and materials will be shared upon reasonable request.

**Abbreviations**

BMI: Body mass index, BP: HR: Heart rate, IV: Intravenous, LAs: Local anesthetics, MAP: Mean arterial pressure, SA: Spinal Anesthesia, TURBT: Transurethral resection of bladder tumor, TURP: Transurethral resection of prostate, URS: Uretroscopic removal of stone.

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Figures

-Hypotension – Blood pressure < 90/60 mmHg. Bradycardia – HR < 50 bpm, Tachycardia >20% increase in baseline heart rate.

-P was 0.021 for hypotension and 0.038 for vasopressor consumption.

Figure 1

Occurrence of hypotension & bradycardia and total vasopressor consumption in the first hour after spinal anesthesia: Tikur Anbesa Specialized Hospital, December 1, 2017 – February 28, 2018