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Prevalence and Risk Factors for Intraoperative Hypotension during Craniotomy for Traumatic Brain Injury

Deepak Sharma, MD, DM^{1,2}, Michelle J Brown, BS¹, Parichat Curry, MD¹, Sakura Noda, BA¹, Randall M. Chesnut, MD^{2,3}, and Monica S. Vavilala, MD^{1,2,4,5}

¹Department of Anesthesiology & Pain Medicine, University of Washington, Seattle, WA

²Department of Neurological Surgery, University of Washington, Seattle, WA

³Department of Orthopedic & Sports Medicine, University of Washington, Seattle, WA

⁴Department of Pediatrics, University of Washington, Seattle, WA

⁵Department of Radiology, University of Washington, Seattle, WA

Abstract

Background—Hypotension after traumatic brain injury (TBI) is associated with poor outcomes. However, data on intraoperative hypotension (IH) are scarce and the effect of anesthetic agents on IH is unknown. We examined the prevalence and risk factors for IH, including the effect of anesthetic agents during emergent craniotomy for isolated TBI.

Methods—A retrospective cohort study of patients 18 years who underwent emergent craniotomy for TBI at Harborview Medical Center (level-1 trauma center) between October 2007 and January 2010. Demographic, clinical and radiographic characteristics, hemodynamic and anesthetic data were abstracted from medical and electronic anesthesia records. Hypotension was defined as systolic blood pressure (SBP) < 90 mmHg. Univariate analyses were performed to compare the clinical characteristics of patients with and without IH and multiple logistic regression analysis was used to determine independent risk factors for IH.

Results—Data abstracted from 113 eligible patients aged 48±19 years was analyzed. Intraoperative hypotension was common (n=73, 65%) but not affected by the choice of anesthetic agent. *Independent risk factors* for IH were multiple Computed Tomographic (CT) lesions (AOR 19.1 [95% CI: 2.08–175.99]; p=0.009), SDH (AOR 17.9 [95% CI: 2.97–108.10]; p=0.002), maximum CT lesion thickness (AOR 1.1 [95% CI: 1.01–1.13]; p=0.016), and anesthesia duration (AOR 1.1 [95% CI: 1.01–1.30]; p=0.009).

Conclusion—Intraoperative hypotension was common in adult patients with isolated TBI undergoing emergent craniotomy. The presence of multiple CT lesions, subdural hematoma, maximum thickness of CT lesion and longer duration of anesthesia increase the risk for IH.

Keywords

Intraoperative Hypotension; Traumatic Brain Injury; Craniotomy

Address correspondence to, Deepak Sharma MD, DM, Department of Anesthesiology & Pain Medicine, Box # 359724, 325 9th Ave, Seattle, WA. dsharma@uw.edu.

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Introduction

Traumatic brain injury (TBI) is a leading cause of death and disability and a major public health concern.¹ While the severity of brain damage due to the traumatic impact accounts for the majority of early mortality after TBI, secondary damage to the injured brain can occur as a result of hypoxia, hypotension, hyper and hypocarbia, increased intracranial pressure and hyperglycemia, all leading to poor outcome.²⁻⁹ The intraoperative period involving anesthesia for craniotomy is a crucial phase of TBI management where the patients may be exposed to further risk of a variety of secondary insults.^{5-7,9-14} The prevention and rapid correction of these secondary insults can result in improved outcomes after TBI. The current Brain Trauma Foundation guidelines recommend prevention and early correction of hypotension (defined as systolic blood pressure [SBP] < 90 mmHg) after severe TBI.¹⁵

While hypotension in TBI patients has been studied in a variety of settings, including pre-hospital phase, emergency department and intensive care units,²⁻⁴ intraoperative data on hypotension are scarce. In the earliest study published about 20 years ago, Pietropaoli et al⁹ demonstrated the deleterious effect of IH (systolic blood pressure < 90 mmHg) on the outcomes of severe TBI in a small series of 53 patients. However, they did not specifically examine craniotomies and did not elucidate any risk factors for IH.⁹ Four years later, in another small series of 56 patients, Kawaguchi et al¹⁰ reported that greater than 20% reduction in the mean arterial pressure after dural opening in patients with acute subdural hematoma could be predicted by low Glasgow Coma Scale (GCS) score, absence of mesencephalic cisterns on computed tomographic (CT) scan and bilateral dilated pupils. However, they used a definition of hypotension that is not commonly used.¹⁰ More recently, Kinoshita et al,⁵ in a series of 67 patients, found that low admission GCS, preoperative tachycardia and hypertension and delayed surgery were risk factors for IH but they included patients with only subdural hematoma and cerebral contusion and did not examine radiographic characteristics of TBI.⁵ Importantly, none of these studies described the anesthetic details which are important determinants of hemodynamic stability during these procedures.

Patients with TBI who suffer intraoperative hypotension (IH) following decompressive surgery may have significantly high incidence of death, persistent vegetative state or disability.^{5,9} Hence, it is imperative to anticipate and avoid IH during craniotomy for TBI. Therefore, the aim of this study was to investigate the prevalence and risk factors for IH in adult patients undergoing emergent craniotomy for isolated TBI, considering the effects of anesthetic agents.

Materials and Methods

Study Design

Following approval by the institutional review board, we conducted a retrospective cohort study of adult patients who underwent emergent craniotomy for TBI at Harborview Medical Center, a level 1 adult and pediatric trauma center.

Subjects and Setting

Patients aged 18 years who were brought to the operating room directly from the emergency department for emergent craniotomy for TBI between October 2007 and January 2010 at Harborview Medical Center were included. We excluded patients undergoing repeat craniotomy, non-emergent craniotomy, burr hole evacuation procedures and patients with polytrauma.

Institutional Anesthetic Management of Craniotomy for TBI

During the study period, there was no standardized protocol for the anesthetic management of patients undergoing craniotomy for TBI and the attending anesthesiologist determined the anesthetic regime for each case. Typically, anesthesia was induced with intravenous bolus of propofol / etomidate in patients that arrived in the operating room without a tracheal tube or inhalation of an inhalational anesthetic agent (isoflurane / sevoflurane / desflurane) via indwelling tracheal tube in those who arrived intubated. For maintenance, the patients typically received general anesthesia with < 1.0 minimum alveolar concentration isoflurane / sevoflurane / desflurane without nitrous oxide, or intravenous anesthesia with propofol infusion and intermittent boluses of rocuronium / vecuronium and opioid at the discretion of attending anesthesiologist. In addition to the standard ASA (American Society of Anesthesiologists) monitors, arterial catheters were used for continuous blood pressure monitoring and for sampling blood gases and glucose values. Mannitol (0.5–1 g/kg) and mild hyperventilation were used to induce brain relaxation during surgery, if required. Isotonic non-glucose containing intravenous fluids were used intraoperatively and colloids use was infrequent. Transfusion of blood products and treatment of IH including choice of vasopressor agents was at the discretion of the attending anesthesiologist. Normothermia was maintained by using forced warm air blankets and infusion of warm intravenous fluids. Intraoperative hypothermia was not used in any patient.

Data Sources

Data sources were Harborview Medical Center medical records, electronic anesthesia records and emergency department records. ICD-9 (International Classification of Diseases, ninth revision) codes reflecting diagnosis of subdural hematoma (SDH), extradural hematoma (EDH), subarachnoid hemorrhage (SAH) and intraparenchymal hemorrhage were matched with anesthesia records for craniotomy during the study period. Admission Glasgow Coma Scale (GCS) score, pupillary asymmetry, medical history and current medications were retrieved from the emergency department records. Emergency department records were also used to abstract preoperative heart rate and blood pressure data and administration of mannitol. Imaging characteristics of TBI lesions were abstracted from the official reports of the preoperative CT scans by attending radiologists. Electronic anesthesia records were used to abstract intraoperative data including heart rate, blood pressure, fluid balance, mannitol administration, blood transfusion, vasopressor use and anesthetic agents. The heart rate and blood pressure data are recorded for every minute in the electronic anesthesia records but displayed once every five minutes. For analyses, we used the measurements that were displayed every 5 minutes.

Mortality information was abstracted from discharge summaries.

Definitions

Intraoperative hypotension was defined as any SBP < 90 mmHg (irrespective of duration of the episode) and hypertension was defined as SBP > 140 mmHg. The electronic anesthesia records displayed the hemodynamic parameters once every five minutes and every displayed reading of SBP < 90 mmHg was counted as an “episode” of IH. Bradycardia was defined as heart rate < 60 beats per minute and tachycardia was defined as heart rate > 100 beats per minute. Maximum thickness of CT lesion was defined as the largest dimension of the hemorrhagic lesion reported on immediate preoperative CT scan by clinical radiologists.

Outcomes

The outcome measures were the prevalence and risk factors for IH. We also examined the effect of anesthetic agents on IH and the relationship between IH and all cause in-hospital mortality.

Statistical Analysis

Descriptive statistics were used to characterize baseline clinical characteristics and IH data. Kolmogorov-Smirnov tests were used to test for normal distribution and guided subsequent analyses. To examine differences of variables between patients with IH and no IH, Student's T test was used for continuous variables and Chi-square test or Fisher's exact test for categorical variables. Multiple logistic regression analysis was used to determine independent risk factors for IH. Data are reported as mean $\pm$ SD, median (range), n (%) for categorical variables and adjusted odds ratios (AOR; 95% confidence intervals [CI]). A p value < 0.05 reflects significance.

Results

Patients

During the three-year study period, 211 adult patients underwent craniotomy for TBI of which 117 met inclusion criteria while 94 patients were excluded for repeat surgery or polytrauma. Four additional patients were excluded for missing anesthetic records and/or misclassification of procedures. The remaining 113 patients with isolated TBI who underwent craniotomy were included in the final analysis.

Preoperative Characteristics (Table 1)

The mean age of patients in this cohort was 48 ± 19 years, most were males and most had severe TBI (GCS ≤ 8 , 53%). Few (4%) patients had documented hypotension on arrival to the emergency department. The majority (66%) of patients had a midline shift > 5 mm on admission head CT scan and the median maximum thickness of the CT lesion was 18 mm (range 2–91 mm). Relevant to hemodynamic stability, one patient had history of diabetes mellitus, 5 patients had ischemic heart disease, 5 patients had hypertension and 13 had some combination of the three. Twenty-six patients (23%) did not have diabetes mellitus, ischemic heart disease or hypertension while the medical history was unknown for the remaining patients. The current medications were unknown for the majority of patients ($n=64$, 57%). Four patients were taking ACE inhibitors, 2 were taking beta-blockers, 1 was taking furosemide and 5 were taking some combination of the three. Thirty-one patients were not taking any of these 3 medications while 9 patients were on anticoagulants (aspirin / warfarin).

Intraoperative Characteristics (Table 2)

Propofol and etomidate were used for induction in 64 and 4 patients, respectively in this series while inhalation of a volatile anesthetic through indwelling tracheal tube was used in 42 patients. Only 3 patients received propofol for maintenance anesthetic and the remaining patients received volatile anesthesia. While 89 patients received intermittent 25–100 mcg boluses of fentanyl, 2 patients received morphine and remifentanyl each, one patient received hydromorphone and 8 patients received some combination of various opioids.

Seventy-three patients (65%) had at least one episode of IH; 40 patients (35%) did not have any IH. Thirty-four (30%) patients had 1–4 episodes and 39 (35%) patients had > 5 episodes of IH (Figure 1). The mean lowest intraoperative SBP was 68 ± 11 mmHg (range 50–85 mmHg) for the IH group and 97 ± 8 mmHg (range 90–120 mmHg) for non-IH group. The

median number of episodes of IH was 5 (range 0–40). Intraoperative hypotension was treated with fluid replacement, blood products transfusion and vasopressors at the discretion on the attending anesthesiologist. The most common vasopressor used to treat IH was phenylephrine. Of the 73 patients who had IH, 38 (52%) were treated by intermittent boluses: phenylephrine 100–200 mcg alone (n=27, 37%), ephedrine 5–10 mg alone (n=4, 6%) or both (n=7, 10%). Twelve (16%) patients received infusion of phenylephrine (10–100 mcg/min) alone and 17 (23%) patients received phenylephrine infusion (10–100 mcg/min) in addition to boluses of phenylephrine and ephedrine.

The clinical characteristics of patients with and without IH are described in Table 3. Of the 40 patients with pupillary asymmetry, 73% had IH. Sixty-three percent patients with hypertension and / or tachycardia and 60% patients with bradycardia in the emergency department had IH. Among those who received mannitol in the emergency department, 69% suffered from IH while 67% of those who received mannitol during surgery suffered from IH. Intraoperative hypotension occurred in 71% patients with SDH and 72% with multiple lesions but in only 32% patients with EDH. Two-thirds of the patients with midline shift > 5 mm also had IH. There was no difference in the choice of induction or maintenance anesthetic agent between patients with IH and no-IH.

Risk Factors for Intraoperative Hypotension (Table 3,4)

The clinical characteristics of patients with and without IH are detailed in Table 3. The TBI severity of the groups was similar (p=0.62). There was no difference in the distribution of documented medical history of ischemic heart disease, diabetes mellitus, hypertension between patients with IH and patients with no-IH (p=0.54). There was also no difference in the documented use of ACE inhibitors, beta-blockers, furosemide or any combination of these medications between the two groups (p=0.17).

There was no difference in the prevalence of IH in the entire cohort based on induction agents (p=0.87), maintenance anesthetic agents (p= 0.37) or opioid administration (p=0.45). There was also no difference in the prevalence of IH based on anesthetic agents including opioids when we analyzed the patients separately according to TBI severity. *Univariate* risk factors for IH were SDH, multiple CT lesions, immediate preoperative SBP, and anesthesia duration (Table 3). *Independent risk factors* for IH were multiple CT lesions (AOR 19.1 [95% CI: 2.08–175.99]; p=0.009), SDH (AOR 17.9 [95% CI: 2.97–108.10]; p=0.002), maximum CT lesion thickness (AOR 1.1 [95% CI: 1.01–1.13]; p=0.016), and anesthesia duration (AOR 1.1 [95% CI: 1.01–1.30]; p=0.009). All-cause in-hospital mortality for the entire cohort was 16% (n=18). There was no difference in the in-hospital mortality between patients with IH and no IH (p=0.28).

Discussion

The main findings of this study are that in anesthetized adult TBI patients, (1) the burden of hypotension is high in the intraoperative period and (2) multiple lesions on preoperative CT scan, SDH, maximum thickness of lesion on CT scan and duration of general anesthesia were independent risk factors for IH. Choice of anesthetic agent was not associated with IH. These findings indicate that during emergent / urgent craniotomy for TBI, the burden of IH is high but can be predicted by select characteristics of TBI on head CT.

Importance of General Anesthesia and the Intraoperative period

Hypotension in TBI has been well described in published literature in prehospital, emergency department and intensive care settings. However, the intraoperative period is physiologically very different due to ongoing surgery, fluid shifts and exposure to anesthetic

agents. Traumatic brain injury has intracranial as well as extracranial effects including myocardial and pulmonary dysfunction^{16,17} and the commonly used anesthetic agents also affect cerebral hemodynamics and physiology^{18–21} as well as myocardial depression and peripheral vasodilation, leading to hypotension.^{22–24} Additionally, surgical intervention with dural opening results in decompression hypotension which is thought to result from sudden loss of the Cushing's response.^{5,10} Thus, the hemodynamic effects of TBI in anesthetized patients during craniotomy may be complex.

The intraoperative period may be particularly important in the course of TBI management for numerous reasons.¹⁴ *First*, despite interventions to correct secondary insults preoperatively, one or more secondary insults may persist or remain undetected as the patient is emergently transported to the operating room.^{6,7} Hence, the intraoperative period may provide an opportunity for the anesthesiologists to either continue ongoing resuscitation or to aggressively correct uncorrected secondary insults, such as hypotension. *Second*, surgery and anesthesia predispose the TBI patient to new secondary insults and injuries.¹¹ We, and others have previously reported new onset hypotension during craniotomy for TBI in children^{12,13} as well as adults.^{5,9,10} Our previous work in adults as well as children with TBI receiving similar anesthetic agents has shown that new onset intraoperative hyperglycemia is common during craniotomy for emergent / urgent TBI and is associated with poor neurological outcomes.^{6,7} Since secondary injury is largely preventable and/or treatable, the intraoperative period may be an important potential window to initiate interventions to aggressively treat intraoperative hypotension and improve TBI outcomes.

Prevalence of Intraoperative Hypotension

Our results document a large IH burden during craniotomy for TBI (Figure 1). The prevalence of IH in this study was higher than previously reported in children (range 41–52%)^{12,13} as well as adults (32–46%).^{5,9,10} This difference may be due to a greater accuracy of automated recording of hemodynamic data in the electronic anesthesia records in our study. While on average 5 hypotensive episodes occurred per patient and as many as 40 episodes in some, we were not able to depict the timing of occurrence of these episodes in the course of surgery, given the retrospective nature of this study. Kinoshita et al reported that all episodes of IH occurred immediately after dural opening.⁵ However, hypotension may occur following induction and maintenance of general anesthesia and may also be related to intravascular volume shifts. Yet, in this cohort, we did not find any difference in intraoperative blood loss, mannitol use or fluid balance between patients with and without IH indicating that IH may occur despite appropriate fluid management during craniotomy for TBI. Although we do not have data on intraoperative brain swelling in our series, others have shown that IH during craniotomy can cause intraoperative brain swelling.⁵ Irrespective, our results indicate that goals to preserve cerebral perfusion may not be currently met in significant number of patients undergoing emergent craniotomy for TBI and resuscitation may pose substantial challenges for the anesthesiologist.

Kinoshita et al⁵ reported 46% incidence of IH but their definition of hypotension was stricter requiring SBP < 90 mmHg to persist for 10 minutes. The minimum duration of hypotension that is detrimental to the injured brain is unknown and some have reported increased mortality in patients with TBI with SBP below 110 or 120 mmHg.^{2,25} However, since the clinical goal is to avoid any episode of SBP < 90 mmHg and blood pressure below this threshold frequently triggers vasopressor treatment in accordance with current guidelines,¹⁵ we defined IH as any SBP < 90 mmHg. Unlike others,^{5,9} we did not find an association of IH with mortality but this may be because the overall mortality in our series was only 16% and approximately 30% of our patients had mild TBI. Moreover, low prevalence of pre-hospital hypotension may have contributed to reduction of overall ischemic burden. However, we do not have Glasgow Outcome Score (GOS) data and long-

term outcomes that may have been affected by IH. Alternatively, it is possible that SBP < 90 mmHg is not necessarily detrimental during general anesthesia given the potential neuroprotective effects of anesthetic agents and reduced cerebral metabolic rate and current TBI guidelines do not address and may not apply in the intraoperative period. This lack of information from the intraoperative period and the equivocal impact of blood pressure control suggests the need for TBI research focusing on the intraoperative period in patients with TBI. However, assuming that SBP < 90 mmHg leads to cerebral hypoperfusion, the occurrence of IH in 65% is alarming and underlines the need for interventions to avoid IH.

Predictors of Intraoperative Hypotension

We found that while the duration of general anesthesia determines the risk of IH, anesthesiologists can predict IH based on the presence of multiple lesions, SDH and maximum thickness of hemorrhage on preoperative CT scan. The association of multiple lesions and maximum thickness of hemorrhage on CT scan with IH may be due to higher blood loss within cranial cavity. Pediatric data suggests that the frequency of IH is higher in patients with hematoma larger than 8% of intravascular volume.¹³ The thickness of lesion on CT may also indicate higher intracranial pressure and mass effect, the release of which by decompression and hematoma evacuation may lead to sudden loss of the sympathetic tone causing peripheral vasodilatation resulting in hypotension. One reason we did not observe an effect of presence or absence of midline shift may be because we had very few patients with no midline shift. While the mechanism by which SDH leads to IH may be unclear, SDH may predict more than one secondary insult during craniotomy,^{6,7,10} indicating the importance of the anesthesiologist's evaluation of preoperative CT scans in patients with TBI.

We did not find any relationship between specific anesthetic agents and IH. Since very few patients in this series received total intravenous anesthesia, we cannot comment on the relative effect of inhalational versus intravenous anesthetic agents on IH. However, there was no difference in IH between patients who received isoflurane or sevoflurane. Our results indicate that the anesthesiologist can select between isoflurane and sevoflurane without concern for hypotension in patients with TBI, provided they are used at low dose and provided that the goals for fluid and intravascular volume status are met. Nevertheless, we found that longer anesthesia time increases the risk of IH, which may be explained by the increased probability of occurrence of an event leading to hypotension over a longer period of time.

Limitations

The most important limitation in this study is the retrospective study design and reliance on medical records not designed for data collection specific to this study. It is also a single center study and there was no uniform care practice. We have since instituted a clinical care pathway. We are unable to precisely report the duration or timing of hypotension. Few patients received intravenous anesthesia limiting our ability to compare anesthetic agents. We also do not have long-term outcome data. Despite these limitations, this study provides new insight into the high prevalence of IH and suggests a new perspective to future considerations of the role of intraoperative care in TBI patients.

In conclusion, IH can be expected in a significant proportion of patients undergoing emergent / urgent craniotomy for TBI despite the absence of preoperative hypotension and should anticipate the need to treat IH in patients with multiple CT lesions or SDH requiring longer duration of anesthesia.

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Figure 1.

Number of episodes of Intraoperative Hypotension (IH) during Craniotomy for Traumatic Brain Injury. Note that majority of patients with IH had < 5 episodes and very few patients had > 20 episodes of IH.

Table 1**Preoperative Characteristics (n=113)**

Age (yr)	48 ± 19
Gender	80 M: 33 F
GCS (n=111)	9 ± 4 (3–15)
Mild (13–15)	33 (30%)
Moderate (9–12)	19 (17%)
Severe (8)	59 (53%)
ED Hypotension (SBP<90 mmHg)	5 (4%)
ED Hypertension (SBP>140 mmHg)	92 (82%)
ED Tachycardia (HR>100 bpm)	43 (38%)
ED Bradycardia (HR<60 bpm)	37 (33%)
Pupil Asymmetry	40 (35%)
ED Mannitol	55 (46%)
ED Furosemide	2 (2%)
ED Hematocrit (%)	39 ± 5 (21–48)
Midline shift (mm) (n=97)	8 ± 5 (0–24)
Midline shift >5mm	64 (66%)
Maximum thickness of CT lesion (mm) (n=88)	23 ± 19 (2–91)

Data as mean ± SD (range), n(%)

GCS=Glasgow Coma Scale score; ED=emergency department; CT= Computed Topography; bpm=beats per minute

Table 2

Intraoperative Hypotension Data (Hypotension defined as Systolic Blood Pressure < 90 mmHg)

Patients with Intraoperative Hypotension	73 (65%)
Number of episodes of hypotension	5 ± 6 (0–40)
Lowest systolic blood pressure (mmHg)	78 ± 17 (50–120)
Intravenous Fluids (mL)	3400 ± 1950 (400–12400)
Blood products transfused (mL)	455 ± 779 (0–3600)
Urine output (mL)	1400 ± 1260 (0–7200)
Fluid Balance (mL) (n=112)	2165 ± 1770 (–600–9150)
Mannitol	68 (60%)

Data as mean ± SD (range).n (%)

Table 3

Clinical Characteristics of Patients With and Without Intraoperative Hypotension (Systolic Blood Pressure < 90 mmHg)

	IH (n= 73)	No IH (n= 40)	p value
Glasgow coma scale score (n=111)	8 ± 4 (3–15)	9 ± 4 (3–15)	0.61
TBI severity			0.62
Mild (13–15)	21	12	
Moderate (9–12)	11	8	
Severe (8)	41	18	
Pupil asymmetry (n=40)	29 (40%)	11 (28%)	0.22
ED Hypotension (n=5)	3 (4%)	2 (5%)	1
ED Hypertension (SBP >140 mmHg) (n=93)	59 (81%)	34 (85%)	0.8
ED Tachycardia (heart rate > 100 bpm) (n=43)	27 (37%)	16 (40%)	0.45
ED Bradycardia (heart rate < 60 bpm) (n=37)	22 (30%)	15 (38%)	0.53
ED mannitol (n=58)	40 (55%)	18 (45%)	0.33
CT lesion thickness (mm) (n=88)	25 ± 21 (2–91)	19 ± 13 (2–56)	0.13
CT lesion type			0.02
Subdural hemorrhage (n=70)	50 (68%)	20 (50%)	
Extradural hemorrhage (n=19)	6 (8%)	13 (33%)	
Multiple hemorrhages (n=18)	13 (18%)	5 (13%)	
Intracerebral hemorrhage (n=5)	3 (4%)	2 (5%)	
CT midline shift >5mm	42 (58%)	21 (53%)	0.74
Anesthetic induction agent (n=110)			0.875
Propofol (n=64)	40 (55%)	24 (60%)	
Etomidate (n=4)	3 (4%)	1 (3%)	
Inhalational induction (through tracheal tube) (n=42)	27 (37%)	15 (38%)	
Maintenance Anesthetic agent (n=109)			0.37
Isoflurane (n=37)	22 (30%)	15 (38%)	
Sevoflurane (n=72)	49 (67%)	23 (58%)	
Fentanyl (n=89)	53 (73%)	36 (90%)	0.45
OR Baseline SBP (mm Hg)	129 ± 38 (60–270)	143 ± 28 (90–200)	0.05
OR Baseline Hypertension (SBP>140 mmHg) (n=55)	31 (42%)	24 (60%)	0.08
Intraoperative blood loss (mL) (n=112)	406 ± 346 (75–1800)	553 ± 630 (50–2500)	0.18
Intraoperative mannitol (n=45)	30 (41%)	15 (38%)	0.841
Intraoperative fluid balance (mL) (n=112)	2035 ± 1818 (–600–9150)	2410 ± 1678 (122–6300)	0.2
Anesthesia time (min)	228 ± 70 (128–437)	189 ± 60(93–317)	0.003
In-hospital mortality (n=18, 16%)	14 (19%)	4 (10%)	0.28

Data as mean ± SD (range); n (%) with % indicating column %

OR= Operating Room; CT= Computed Topography; bpm= beats per minute

Table 4

Independent Risk Factors of Intraoperative Hypotension

	Odds Ratio	95.0% C.I.		Sig.
		Lower	Upper	
Multiple CT Lesions	19.1	2.08	175.99	0.009
Subdural Hemorrhage	17.9	2.97	108.1	0.002
Maximum Thickness of Lesion on CT	1.1	1.01	1.13	0.016
Anesthesia Duration	1.1	1.01	1.30	0.009