Original Article

Comparison of Renal Function during Sevoflurane versus Total Intravenous Propofol Anaesthesia in Patients undergoing Abdominal Surgeries: A Randomised Controlled Trial

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ABSTRACT

Introduction: For practically all surgeries, inhalational anaesthesia has been the most widely used way to maintain general anaesthesia. Inhalational anaesthesia has a more severe effect on the oxygenation and circulation of the kidneys than propofol does. Over the past thirty years, many studies have examined the impact of sevoflurane on kidney function following surgery, even if the amount of evidence remains incompletely studied. Compared to propofol, we hypothesized sevoflurane to cause a decrease in the excretion of salt and water in the urine.

Aims/ objective: To compare the effect of sevoflurane and propofol total intravenous anaesthesia on renal function peri-operatively in patients undergoing abdominal surgeries in tertiary care centre of eastern India.

Materials and Method: 33 patients in Group S planned for elective abdominal surgery were given 1-2% sevoflurane in a mixture of oxygen and air to maintain anaesthesia following the delivery of pre-anaesthetic medications. To maintain anaesthesia, 32 patients in group P received intravenous rote propofol at a dosage of 2-4 mg/kg/h. The primary outcome indicator during surgery was the amount of urine produced. Secondary outcome measures were electrolyte excretion, plasma osmolality, serum creatinine, and serum electrolytes.

Results: Patients under sevoflurane anaesthesia had a significantly greater fall in urine output during surgery (1.14 to 0.56 ml/kg/hr) than those under propofol anaesthesia (1.09 to 0.946 ml/kg/hr) ($p<0.05$). Additionally, patients under sevoflurane anaesthesia excreted less sodium fractionally than those under propofol anaesthesia ($p<0.05$). Compared to patients under propofol anaesthesia, those under sevoflurane anaesthesia had a considerably higher serum NGAL level (110.38 to 239.21 ng/ml), indicating renal damage ($p<0.0001$). Patients under sevoflurane anaesthesia had slightly elevated serum levels of potassium and plasma osmolality.

Conclusion: When propofol anaesthesia was used instead of sevoflurane, patients' renal function was more intact. The reduced increase in NGAL level in the propofol group after surgery served as evidence for this.

Keywords: Sevoflurane, Propofol, Anaesthesia, Renal Function, Urine Output, Serum Creatinine, Acute Kidney Injury.

INTRODUCTION

For practically all surgeries, inhalational anaesthesia has been the most widely used way to maintain general anaesthesia. Sevoflurane and isoflurane are two of the most commonly used strong inhalation anaesthetics during adult surgery. Isoflurane is a halogenated anaesthetic with modest biological transformation and few deleterious effects. Due to its limited metabolism and relative stability, it is believed to be less harmful to tissue.[1]

When inhaled, sevoflurane, a non-irritating fluid halogen-based ether anaesthetic, is less likely to induce coughing and breathing difficulties. In the same way, restricted solubility promotes quick and effective brain concentration.[1] Another common inhalational anaesthetic is isoflurane, a derivative of ether. It undergoes minimal biotransformation, thus those with established liver or renal illness can safely use it.

Propofol is an intravenous anaesthetic used to maintain general anaesthesia in patients. It is largely attached to plasma proteins and is metabolized via conjugation in hepatocytes. Its therapeutic benefits are not as long-lasting since it is swiftly re-distributed into the perivascular tissues.[1, 2]

Strong base carbon dioxide absorbents convert sevoflurane to a compound called compound A (CpA).[2] The use of baralyme as an absorbent, decreased fresh gas flow rates, raised temperatures, a drop in the water content of the absorber, and increased sevoflurane levels have all been linked to CpA, which can cause kidney damage.[3]

An increased risk of mortality and developing chronic renal illness is associated with acute kidney injury (AKI), which occurs in 6–8% of surgical patients. [4-6] Acute kidney injury is primarily diagnosed by reduced urine production, with indirect evaluations of reduced glomerular filtration rate (GFR) also being useful.[7] Inhalational anaesthesia causes irreversible impairment to the kidneys' excretory function, which directs the use of urine output in the peri-operative evaluation and staging of acute kidney injury. [8, 9] Although oliguria during surgery is a warning indication for the development of acute renal damage later, most patients

with urine production below 0.5 ml/kg/h during surgery are not initially diagnosed with acute kidney injury. [10]

Hypovolaemia, which typically necessitates IV fluids to boost blood volume, is commonly linked to oliguria. [11] Nevertheless, intravenous fluid therapy for oliguria does not reduce the risk of acute kidney injury after surgery or enhance urine output during the procedure. [12, 13]

Recent research has shown that sevoflurane after surgery for hypospadias repair can have negative consequences on hemodynamically stable children. These effects include elevated plasma renin, decreased excretion of salt and urine, and little impact on arginine-vasopressin.[14] Furthermore, in an animal model, the induction of renal sympathetic nerve function by sevoflurane results in renal vasoconstriction, which lowers the kidneys' excretion of salt and water.

Inhalational anesthesia has a more severe effect on the oxygenation and circulation of the kidneys than propofol does.[15] A recent thorough analysis of six research papers on sevoflurane and isoflurane in people with normal kidney function was unable to find any differences in kidney function between the two medications after surgery.[16] Over the past thirty years, many studies have examined the impact of sevoflurane on renal function following surgery, even if the body of evidence is still incompletely studied. Any relationship between sevoflurane administration and post-operative kidney function should take pre-existing renal disease, variable fresh gas fluxes, and absorbents into consideration.

Compared to propofol, we hypothesized sevoflurane to cause a decrease in the excretion of salt and water in the urine. This study was done to compare the effect of sevoflurane and propofol total intravenous anaesthesia on renal function peri-operatively in patients undergoing abdominal surgeries in tertiary care centre of eastern India.

MATERIALS AND METHODS

From January 2023 to December 2023, an open label randomised controlled study with parallel 1:1 allocation was carried out in the anaesthesia department of a tertiary care centre located in eastern India. With approval from the institutional ethics committee and in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines, the study was carried out on patients who were scheduled for elective abdominal procedures. All eligible participants scheduled for elective abdominal surgery received a participant information sheet, which was explained to them, and their written informed permission was obtained.

Inclusion Criteria: Patients with physical statuses 1 to 2 according to the American Society of Anaesthesiologists (ASA) who have been scheduled for elective abdominal surgeries, of either gender and age between 18-65 years.

Exclusion Criteria: Women who are pregnant or nursing, individuals in NYHA class 3–4, individuals allergic to sevoflurane or propofol, individuals with pre-existing renal disease, individuals with hepatic disease, and individuals with a genetic malignant hyperthermia were excluded from study.

According to earlier studies, a minimum sample size of 70 patients, with 35 patients in each group, was needed for detecting a difference of a minimum of 20% across two groups given 95% power and a 0.05 alpha value.

The random numbers generated by the web were used for randomization. Patients were assigned to one of two groups: Group P (propofol) or Group S (sevoflurane).

The same anaesthesia induction protocol was used for each participant, consisting of intravenous fentanyl (5–10 mg per kg), midazolam (0.025 mg per kg), and rocuronium (1 mg per kg) to facilitate endotracheal intubation. Every subject received an intravenous injection of atracurium (7.5 mg/kg/min) and fentanyl (1 mg/kg/h) during the procedure. To maintain anaesthesia, Group S was given a mixture of air and oxygen containing 1-2% sevoflurane. The group P patients were ventilated with a combination of oxygen and air, and propofol was given intravenously at a rate of 2-4 mg per kg per hour to sustain anaesthesia. The recommended dosages of investigational medications were modified to maintain the BIS value between 40 and 50 after the bispectral index (BIS) was measured.

The primary outcome indicator during surgery was the amount of urine produced. Secondary outcome measures were potassium excretion, fractional sodium, plasma osmolality, serum creatinine, and serum electrolytes.

Neutrophil gelatinase-associated lipocalin (NGAL) is a novel biological biomarker of renal injury that was investigated for the goal of identifying acute kidney injury (AKI). Blood samples from the implanted artery cannula were obtained for test NGAL measurement after four hours of operation, as well as for standard NGAL assessment prior to the induction of anaesthesia. Blood levels of NGAL have been found to be early markers of AKI under the right circumstances. After stress, NGAL is released from the kidney's tubular cells.

A Foley catheter was inserted into the urethra to facilitate urine drainage after anaesthesia was induced, and each subject received a controlled dosage of acetate-containing Ringer's fluid at a rate of 5 millilitres per kilogram per hour. Prior to being transferred to the surgery ward, patients were transported from the operating theatre to the post-surgical rehabilitation facility for a minimum of two hours. Post-operative discomfort was managed with acetaminophen or Intravenous opioid (morphine or oxycodone) following the treating surgeon's advice. Blood samples were taken before the procedure began, 30 minutes after the anaesthesia began, an hour after the surgical care began, and the next day in the surgical ward.

Urine was continuously collected before surgery under anaesthesia, in the initial two hours after surgery, and in the last two hours of the surgery ward the day after

surgery. A combined urine sample was taken and quantified for each time period.

Statistical Analysis

SPSS version 24 was used to analyse the data that was gathered from patients in groups S and P. The data was tabulated and presented using Microsoft Excel 365. Fisher's exact test was used to determine the statistical significance of the difference between groups S and P. Categorical data, such as sex and ASA status, were expressed as numbers and percentages. Age, urine output, serum creatinine level, eGFR, serum electrolytes, and urinary NGAL levels were among the continuous data that were represented as mean ± standard deviation (SD). The statistical significance of the difference between groups S and P was assessed using the unpaired t-test. When the p-value was less than 0.05, the difference between groups S and P was deemed statistically significant.

RESULTS

Between January 2023 and December 2023, 70 patients meeting our inclusion and exclusion criteria were enrolled and randomised to each group with 35 patients in each group.

Table 1: Comparison of baseline demographic and clinical characteristics between group P and group S

Variables	Group P n = 35	Group S n = 35	P-Value
Age in years (mean ± SD)	53.38 ± 7.20	52.53 ± 6.87	0.60
Gender, n (%)			
Male	19	20	0.78
Female	16	15	
Weight in kg (mean ± SD)	69.83 ± 8.60	70.20 ± 8.95	
BMI in kg/m^2 (mean ± SD)	25.56 ± 4.44	25.28 ± 4.32	0.71
Duration of Surgery in minutes (mean ± SD)	97.65 ± 10.33	103.57 ± 9.46	0.02
ASA physical status, n (%)			
1	24	25	0.76
2	11	10	
Intraoperative Bleeding in ml (mean ± SD)	233.77 ± 37.61	242.02 ± 38.29	0.35

Age, gender, body weight, body mass index, ASA grade, and intra-operative blood loss were similar in both patient groups receiving either inhalational anaesthesia with sevoflurane or total iv anaesthesia with propofol (p>0.05). However, individuals who received sevoflurane anaesthesia had a longer intra-operative time. Of the total patients, about 70% had ASA grade 1.

Table 2: Comparison of primary and secondary outcome of urinalysis between group P and group S

Variables	Group P n = 32	Group S n = 33	P-Value (Unpaired t test)
Urine Output in ml/kg/hour (mean ± SD)			
Before Surgery	1.09 ± 0.16	1.14 ± 0.14	0.17
During Surgery	0.96 ± 0.11	0.56 ± 0.05	<0.0001
After Surgery	0.99 ± 0.10	0.68 ± 0.06	<0.0001
24 hours after surgery	1.02 ± 0.17	0.90 ± 0.14	0.001
Fractional Sodium Excretion in % (mean ± SD)			
During Surgery	0.95 ± 0.09	0.56 ± 0.04	<0.0001
After Surgery	1.34 ± 0.19	0.96 ± 0.11	<0.0001
24 hours after surgery	0.65 ± 0.07	0.79 ± 0.09	<0.0001
Fractional Potassium Excretion in % (mean ± SD)			
During Surgery	12.93 ± 1.69	10.81 ± 1.48	<0.0001
After Surgery	14.28 ± 1.98	16.71 ± 2.04	<0.0001
24 hours after surgery	7.66 ± 1.21	8.23 ± 1.26	0.057

Patients under sevoflurane anaesthesia had a significantly greater fall in urine output during surgery (1.14 to 0.56 ml/kg/hr) than those under propofol anaesthesia (1.09 to 0.946 ml/kg/hr) (p<0.05). Additionally, patients under sevoflurane anaesthesia excreted less sodium fractionally than those under propofol anaesthesia (p<0.05).

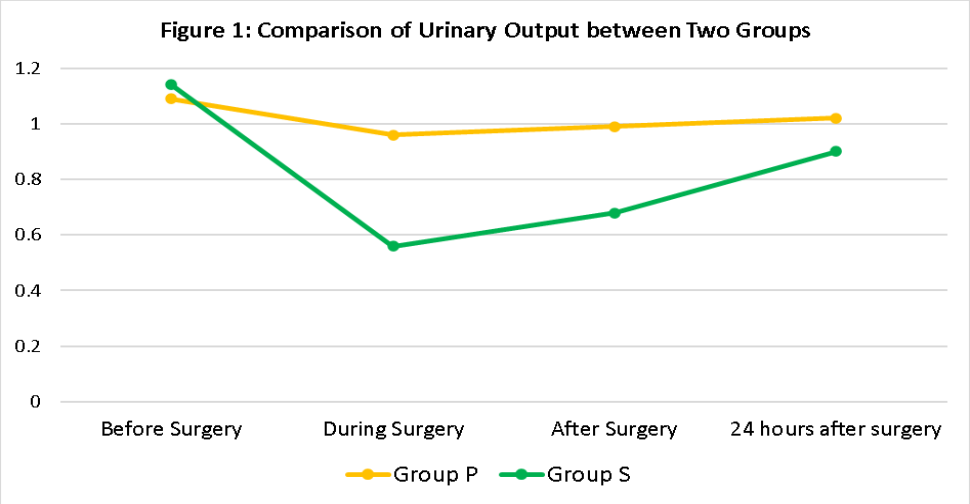

Table 3: Comparison of Renal Injury based on NGAL level and Serum Creatinine between Two Groups

Variables	Group P n = 32	Group S n = 33	P-Value
NGAL level in ng/ml (mean ± SD)			
Before Anaesthesia	107.67 ± 10.48	110.38 ± 11.24	0.30
After Anaesthesia	131.64 ± 12.75	239.21 ± 32.27	<0.0001
Number of patients with serum creatinine >0.3 mg/dl over baseline (%)	3	11	0.03

Compared to patients under propofol anaesthesia, those under sevoflurane anaesthesia had a considerably higher serum NGAL level (110.38 to 239.21 ng/ml), indicating renal damage (p<0.0001). There were also substantially more participants in the sevoflurane group who experienced a rise in blood creatinine levels of more than 0.3 mg/dl.

Table 4: Comparison of Serum Electrolyte and Plasma Osmolality between Two Groups

Variables	Group P n = 32	Group S n = 33	P-Value (Unpaired t test)
Serum sodium in mmol/L (mean ± SD)			
Before Anaesthesia	142.63 ± 6.36	141.51 ± 5.78	0.44
After Anaesthesia	140.21 ± 4.19	137.98 ± 5.17	0.051
Serum potassium in mmol/L (mean ± SD)			
Before Anaesthesia	4.12 ± 0.49	4.21 ± 0.58	0.49
After Anaesthesia	3.89 ± 0.51	4.25 ± 0.62	0.009
Plasma osmolality (mean ± SD)			
Before Anaesthesia	292.48 ± 5.12	287.19 ± 4.79	<0.0001
After Anaesthesia	293.35 ± 4.95	298.24 ± 5.18	0.0001

Patients under sevoflurane anaesthesia had slightly elevated serum levels of potassium and plasma osmolality.

DISCUSSION

The effects of propofol or sevoflurane anaesthesia on renal function and hormones regulating water balance were investigated in a sample without substantial concurrent medical problems. As hypothesized, we found that sevoflurane anaesthesia resulted in less excretion of salt and urine than propofol anaesthesia. Serum electrolytes were only little impacted during surgery, regardless of the kind of anaesthesia utilized. After surgery, sevoflurane resulted in a little but significant increase in plasma creatinine compared to propofol.

There has been evidence of decreased urine output during general anaesthesia since the turn of the 20th century.[17] Early studies found that inhalational anaesthesia decreased eGFR, urine output, and sodium elimination, and that ether decreased renal excretory activity.[18] Studies conducted more recently have shown that isoflurane anaesthesia results in lower urine output and higher plasma levels of aldosterone and renin than before anesthesia.[6] IV crystalloid fluid removal is hampered by laparoscopic surgery and inhalational anesthesia.[19]

It has been observed that sevoflurane reduces the amount of urine produced by patients following colorectal

surgery.[20] Unlike propofol anaesthesia, sevoflurane anaesthesia caused the loop diuretics to be used for a longer period of time after the procedure in cases of elective abdominal surgery. This shows that the anaesthetic medications themselves, not the harm from the surgery, are to blame for the initial renal excretion impairment induced by sevoflurane as compared to propofol.

A decrease in urine production following inhalational anaesthesia may be a sign of reduced blood flow to the kidneys. Reductions in renal circulation have been documented by patients under isoflurane anaesthesia. Research on animals has demonstrated that inhalational anaesthetics cause increased sympathetic nerve activity in the kidneys and decreased kidney blood flow and delivery of oxygen when compared to propofol.[15, 21, 22]

While our subjects did not experience acute kidney injury as a result of sevoflurane-induced putative renal hypoperfusion, patients with other risk factors such as as overweight, type 2 diabetes mellitus, chronic kidney disease, or cardiovascular sickness may be more susceptible to compromised renal function. Given that the eGFR and fractional excretion of sodium were lower under anaesthesia than they were during the post-operative period, this may indicate that there is a need for additional research to determine whether decreased renal circulation is related to decreased urine output, a lower concentration of filtered sodium, and increased sodium reabsorption. It has been shown that isoflurane reduces sodium excretion; in our study, the reduction was more pronounced when sevoflurane anaesthesia was used rather than propofol anesthesia.[23]

Julier et al. previously looked at the influence of propofol on decreasing biochemical markers for myocardial and kidney failure in CABG patients. [24]

Furthermore, Yoo et al.'s study shown that patients receiving propofol anaesthesia for valvular heart surgery had a decreased incidence and severity of acute renal injury compared to those receiving sevoflurane anaesthesia. This has been linked to propofol's enhanced ability to lessen peri-operative elevations in inflammatory mediators. [25] The effect of sevoflurane and propofol on renal damage following laparoscopic bariatric surgery was investigated by Fernando et al. They observed that the anaesthetic medications utilized had no effect on the serum levels of NGAL. [26]

Hypovolemia and lowered blood pressure during anaesthesia and surgery are frequent causes of oliguria.[27] Even though there isn't much of a correlation between the amount of fluids taken under anaesthesia and urine production, it's standard clinical practice to increase fluid supply when intraoperative oliguria can indicate hypovolemia. [28] An assumption from the current data suggests that using the urine output to direct the fluid delivery may be even more difficult under sevoflurane anaesthesia than under propofol anaesthesia.

One major drawback of the current study is the inclusion of patients who were comparatively less likely to experience post-operative renal impairment since they were not included in the group of patients with pre-operative serum creatinine levels higher than 2 mg/dL. Furthermore, the post-operative NGAL level was measured at a single time point in order to determine the incidence of acute renal injury.

CONCLUSION

When propofol anaesthesia was used instead of sevoflurane, patients' renal function was more intact. The diminished increase in NGAL level in the propofol group after surgery served as evidence for this. Sevoflurane anaesthesia caused more impairment in urine output, salt and potassium excretion, and post-operative serum creatinine levels than propofol anaesthesia did. Given earlier research showing a higher risk of acute kidney damage following surgery when sevoflurane anaesthesia is used, the decreased renal excretory activities during the anaesthesia may be linked to decreased renal blood flow, hypotension, and hypervolemia. Larger trials with more varied patient populations are required to confirm these findings.

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REFERENCES

1. Eger EI, Eisenkraft JB, Weiskopf RB. The Pharmacology of Inhaled Anesthetics. USA: Springer-Verlag; 2003.
2. Conzen PF, Kharasch ED, Czerner SF, et al. Low-flow sevoflurane compared with low-flow isoflurane anesthesia in patients with stable renal insufficiency. Anesthesiology. 2002;97:578–584.
3. Morio M, Fujii K, Satoh N, et al. Reaction of sevoflurane and its degradation products with soda lime. Toxicity of the byproducts. Anesthesiology. 1992;77:1155–1164.
4. Biteker M, Dayan A, Tekkes,in A, et al. Incidence, risk factors, and outcomes of perioperative acute kidney injury in noncardiac and nonvascular surgery. Am J Surg 2014; 207: 53-9.
5. Mizota T, Yamamoto Y, Hamada M, Matsukawa S, Shimizu S, Kai S. Intraoperative oliguria predicts acute kidney injury after major abdominal surgery. Br J Anaesth 2017; 119: 1127-34.
6. Heung M, Steffick DE, Zivin K, et al. Acute kidney injury recovery pattern and subsequent risk of CKD: an analysis of

Veterans Health Administration data. Am J Kidney Dis 2016; 67: 742-52.

7. Kidney Disease Improving Global Outcome, Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Intern Suppl 2012; 2: 19-23.
8. Kataja J, Viinamaki O, Punnonen R, Kaukinen S. Renin- € angiotensin-aldosterone system and plasma vasopressin in surgical patients anaesthetized with halothane or isoflurane. Eur J Anaesthesiol 1988; 5: 121-9
9. Norberg A, Hahn RG, Li H, et al. Population volume kinetics predicts retention of 0.9% saline infused in awake and isoflurane-anesthetized volunteers. Anesthesiology 2007; 107: 24-32.
10. Myles PS, McIlroy DR, Bellomo R, Wallace S. Importance of intraoperative oliguria during major abdominal surgery: findings of the restrictive versus liberal fluid therapy in major abdominal surgery trial. Br J Anaesth 2019; 122: 726-33.
11. Cecconi M, Hofer C, Teboul JL, et al. Fluid challenges in intensive care: the FENICE study: a global inception cohort study. Intensive Care Med 2015; 41: 1529-37.
12. Egal M, Erler NS, de Geus HR, van Bommel J, Groeneveld AB. Targeting oliguria reversal in goaldirected hemodynamic management does not reduce renal dysfunction in perioperative and critically ill patients: a systematic review and meta-analysis. Anesth Analg 2016; 122: 173-85.
13. Shiba A, Uchino S, Fujii T, Takinami M, Uezono S. Association between intraoperative oliguria and acute kidney injury after major noncardiac surgery. Anesth Analg 2018; 127: 1229-35.
14. Taavo M, Rundgren M, Frykholm P, et al. Role of renal sympathetic nerve activity in volatile anesthesia's effect on renal excretory function. Function 2021; 2: 042-6.
15. Iguchi N, Kosaka J, Booth LC, et al. Renal perfusion, oxygenation, and sympathetic nerve activity during volatile or intravenous general anaesthesia in sheep. Br J Anaesth 2019; 122: 342-9.
16. Ong Sio LC, Dela Cruz RG, Bautista AF. Sevoflurane and renal function: a meta-analysis of randomized trials. Med Gas Res 2017; DOI: https://doi.org/10.4103/2045-9912.215748.
17. Lhoest L. Study of the renal function during anesthesia with halothane and enflurane. Acta Anaesthesiol Belg 1976; 27: 272-82.
18. Miller RH, Cabot H. The effect of anesthesia and operation on the kidney function, as shown by the phenolsulphonephtalein test. Arch Intern Med 1915; XV: 369-91.
19. Olsson J, Svensen CH, Hahn RG. The volume kinetics of acetated Ringer's solution during laparoscopic cholecystectomy. Anesth Analg 2004; 99: 1854-60
20. Bang JY, Lee J, Oh J, Song JG, Hwang GS. The influence of propofol and sevoflurane on acute kidney injury after colorectal surgery: a retrospective cohort study. Anesth Analg 2016; 123: 363-70.
21. Groves ND, Leach KG, Rosen M. Effects of halothane, enflurane and isoflurane anaesthesia on renal plasma flow. Br J Anaesth 1990; 65: 796-800.
22. Iguchi N, Kosaka J, Iguchi Y, et al. Systemic haemodynamic, renal perfusion and renal oxygenation responses to changes in inspired oxygen fraction during total intravenous or volatile anaesthesia. Br J Anaesth 2020; 125: 192-200.
23. Frithiof R, Soehnlein O, Eriksson S, et al. The effects of isoflurane anesthesia and mechanical ventilation on renal function during endotoxemia. Acta Anaesthesiol Scand 2011; 55: 401-10
24. Julier K, da Silva R, Garcia C, Bestmann L, Frascarolo P, Zollinger A, et al. Preconditioning by sevoflurane decreases biochemical markers for myocardial and renal dysfunction in coronary artery bypass graft surgery: A double-blinded, placebo-controlled, multicenter study. Anesthesiology. 2003;98:1315–27.
25. Yoo YC, Shim JK, Song Y, Yang SY, Kwak YL. Anesthetics influence the incidence of acute kidney injury following valvular heart surgery. Kidney Int. 2014;86:414–22.
26. Fernandes A, Ettinger J, Amaral F, Ramalho MJ, Alves R, Módolo NS. General anesthesia type does not influence serum levels of neutrophil gelatinase-associated lipocalin during the perioperative period in video laparoscopic bariatric surgery. Clinics. 2014;69:655–9.
27. Goren O, Matot I. Perioperative acute kidney injury. Br J Anaesth 2015; 115: 113-14.
28. Matot I, Dery E, Bulgov Y, Cohen B, Paz J, Nesher N. Fluid management during video-assisted thoracoscopic surgery for lung resection: a randomized, controlled trial of effects on urinary output and postoperative renal function. J Thorac Cardiovasc Surg 2013; 146: 461-6.