Original Research Article

Indian Journal of Anesthesia and Analgesia
2019; 6(1): 9-18
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.6119.1

Sevoflurane Induction of Anaesthesia in Critically ill Patients Undergoing Emergency Laparotomy

Satyendra Yadav[1], Ajai Vikram Singh[2], Harun Ali[3]

[1]Assistant Professor [2]Associate Professor, Department of Anaesthesiology & Critical Care [3]Assistant Professor, Department of SPM, Veer Chandra Singh Garhwali Government Institute of Medical Science and Research (VCSGGMSRI) Srinagar, Garhwal, Uttarakhand 246174, India.

Abstract

Sevoflurane has characteristics of rapid inhalational induction of anaesthesia in 60 secs while maintaining spontaneous respiration, bronchodilatation, haemodynamic stability with rapid recovery in elective scenario. This retrospective study analysed 40 (n=40) critically ill patients undergoing induction of anaesthesia with sevoflurane in emergency laparotomy. Standard methods and techniques were used in patient induction and management. Patients were optimized prior to sevoflurane induction were in ASA-III E (72.5%) and ASA-IV E (27.5%) status. Clinical outcome of sevoflurane anaesthesia induction analysed in terms of any airway managment complication and haemodynamic stability. Statistical analysis of haemodynamics done by ANOVA (analysis of variance). Incidence of airway complication such as laryngospasm, aspiration were nil and none of patient required abandoning or modification in induction process. Patients Spo2 improved postinduction (only 2.5% SpO_2 between 85%-90%) even in comorbid COPD and smoker patients showing beneficial effects of sevoflurane induction. Induction of anaesthesia with sevoflurane is associated with significant changes ($p < .05$) in MAP and pulse peaking at 3 minutes postinduction as sevoflurane induction unable to attenuate intubation response on haemodynamics. None of the patient had bradycardia (HR < 60 bpm), hypotension (MAP < 60 mmHg) and arryythemias or required additional vasopressor support post induction and throughout perioperative period. None of patient required postoperative ventilation due to delayed recovery. Study concludes sevoflurane induction is without airway complications and haemodynamic unstability is suitable induction agent in emergency laparotomy in optimized critically ill patients while taking measure to prevent aspiration though unable to inhibit adrenergic endotracheal intubation responses.

Keywords: Sevoflurane; Induction of Anaesthesia; Emergency Laparotomy; Haemodynamic stability; Airway complications; safety; Critical patients.

How to cite this article:

Satyendra Yadav, Ajai Vikram Singh, Harun Ali. Sevoflurane Induction of Anaesthesia in Critically ill Patients Undergoing Emergency Laparotomy. Indian J Anesth Analg. 2019;6(1):9-18.

Introduction

Management of induction of anaesthesia in critically ill is challenge to any anaesthesiologist. Apart from time tested anaesthetic agent, more recently introduced sevoflurane (1990) has established itself as induction agent in elective patients [1]. Sevoflurane is a potent, non-pungent,

Corresponding Author: Ajai Vikram Singh, Associate Professor, Department of Anaesthesiology & Critical Care, Veer Chandra Singh Garhwali Government Institute of Medical Science and Research (VCSGGMSRI) Srinagar, Garhwal, Uttarakhand 246174, India.

E-mail: ajaivikramsingh872@gmail.com

Received on 10.10.2018, **Accepted on** 31.10.2018

haemodynamically stable volatile anaesthetic agent. In high concentration sevoflurane is haemodynamically stable with rapid uptake and elimination (blood gas partition coeffcient 0.62) without awareness while maintaining spontaneous respiration. This is of value in unanticipated difficult airway and haemodynamically unstable emergency critical patients [2]. Increasing the concentration of sevoflurane [3] relatively slow passage of sevoflurane through the excitatory stage 2 of anaesthesia will be passed more rapidly. Induction time is less than 60s have been demonstrated and reported without awareness. Incidents of increased airway secretions or laryngospasm rarely occur with minimal coughing or breath-holding during induction with sevoflurane [4]. 8% sevoflurane has been shown to offer good haemodynamic stability even in patients with poor cardiovascular reserve and arrythymia [5]. There is no biochemical evidence of associated hepatotoxicity and concerns of potentially nephrotoxic have been largely discounted [6].

Aims and Objective

1. To examine safety and efficacy of induction of general anaesthesia with sevoflurane in emergency laparotomy in critically ill patients.
2. To evaluate any airway complication and haemodynamic stability during sevoflurane induction and perioperative period in critically ill patient undergoing emergency laparotomy.

Material and Methods

This retrospective study was undertaken after institutional ethics committee approval. Standard technique material and methods were used for patient management. Patients managed with sevoflurane induction only included in this retrospective analysis were 40 (n=40). Informed consents were obtained as routine [7]. All patients were investigated and evaluated to know their physiological metabolic status and clinical diagnosis. This includes haemoglobin, Complete blood count (CBC), serum electrolytes and blood sugar, liver function test, kidney function test, x-ray chest and abdomen, Ultrasonography (USG) and arterial blood gas analysis (ABG). Haemodynamic management based on clinical and noninvasive vital parameter and on ABG. Table 2 showing distributution of clinical diagnosis of patient. After gaining intravenous access with wide bore cannula of 16 G and 18 G patients were optimized haemodynamically with intravenous fluids, colloids, blood product, oxygen with or without requirement of dopamine/noradrenaline (15% Table 3). Patients optimized prior to sevoflurane induction were in optimized American society of Anesthesiology ASA-III E (72.5%) and ASA-IV E (27.5%) status (Table 4). After optimization patient were taken for surgical intervention under general anaesthesia. Non invasive perioperative monitoring including noninvasive blood pressure (NIBP), electrocardiography (ECG), oxygen saturation (SpO_2) applied. Patients were premedicated with inj tramadol, inj pantoprazole, inj metoclopramide, inj ondansetron, inj glycopyrolate. Inj midazolam. 03 to .05 mg/kg given with preoxygenation with 100 percent oxygen for 5 minutes. All measures were taken to prevent aspiration including ryles tube aspiration of gastric content, intravenous pantropazole and sellick's maneuver as appropriate and preparation to deal with if any regurgitation of gastric content occurs.

Sevoflurane Induction Technique [Sevoflurane vaporizer Intermed Penlon Sigma Delta Vaporize]- Patients were explained and visually shown to breath gently after a full expiratory breath to residual volume comfortably and regularly till loss of conscious and eyelid reflex. Anaesthesia machine (oracle S505) closed breathing circuit primed with 100 percent oxygen and 8 percent sevoflurane for 3 to 5 minutes. Patients were asked to take forced exhalation to residual volume breath as explained followed by breath gently regular breath with oxygen flow at 80 to 100ml/kg/ minutes on Mapalson D circuit. Sevoflurane vaporizer set to 1% and increased after every 2 breaths, in sequence 1,2,4 and 8% [8]. Concentration of sevoflurane limited to 6% in patients above 65 years (12.50% Table 1).

Time to induction of anaesthesia taken from when the vaporizer was at 8% to the time when the eyelid reflex lost to central pupil [9]. In late stages 50% nitrous oxide mixed to enhance the induction to minimize agitation. Analgesic properties of nitrous oxide may enhance quality of induction of anaesthesia [9]. Time to loss of conscious defined as the interval from induction time to loss of eyelid reflex. It usually takes 60 to 70 sec to loss of consciousness and eyelid reflex after 3 to 6 breath [10]. With lose of eyelid reflex and consciousness in 50 to 70 secs, patient were given inj. succinylecholine 1 mg/kg and intubated in 60 secs. Patients maintained on intermittent positive pressure ventilation (IPPV) with sevoflurane 1.6 to 3% with oxygen - nitrous oxide and closed circuit sodalime

with minimum positive end expiratory pressure (PEEP) of 2-4 mmHg [12]. Perioperative monitoring included pulse rate, NIBP, ECG, endtidal carbondioxide ($EtCO_2$), tidal volume, Inspiratory :expiratory (I:E) ratio, repiratory rate, temperature and urine output. Neuromuscular relaxation ensured with inj atracurium and vecuronium as appropriate. Vitals were recorded non invasively at 3 to 5 minutes interval. NIBP, pulse rate and SpO_2 were taken at 1, 3, 5, 10, 15, 30, 60 minutes and 10 minutes post extubation and statistically analysed. Surgical intervention performed lasted 90 minutes to 180 minutes (Table 5) and patients extubated after neuromuscular reversal on table as haemodynamics were stable and assessment was possible. Appropriate analgesics were ensured and patient put on oxygen and 24 hrs intensive care unit (ICU) monitoring. Routine investigations were checked postoperatively were within normal limits Patients were given intensive and routine care and discharged from hospital without any increase in morbidity.

Statistical analysis

Statstical analysis was done with the help of statistical package for social sciences (SPSS) version 21. Demographic data and distribution were analysed. Haemodynamic variable analyzed using a Analysis of Variance method (ANOVA) for intergroup analysis. $p<0.05$ considered statistically significant.

Result

This retrospective study analysed 40 (n=40) patients undergoing emergency laparotomy management by standard methods and techniques of sevoflurane induction only. Patient distribution analysed according to age and gender (Table 1), disease (Table 2), cormorbid and physiological status (Table 3), ASA-E status (Table 4), duration of surgical anaesthesia (Table 5). Haemodynamic variable of this patient population analysed by ANOVA depicted in Table 6, 7, 8 & 9 and graphics representation in Graph 1, 2, 3. Table 10 showing incidence of complication during perioperative period.

Majority of patient were male (67.50%). Of all patients 45% were smoker and 45% anaemic. Age varied between 18 to 76 yrs, 77.50% in 18-65 years (Table 1). All patients were referred from distant areas or bought as first medical contact to our medical college hospital in late ASA 4 E (65%) status (Table 4). More than 50% of patients presented with duodenal perforation peritonitis (Table 2) and 90% were in comorbid state of sepsis (Table 3). 10% were in acute haemorrhagic shock require 2 or more units of blood transfusion. Patient were optimized before induction to improve ASA status (Table 4). Despite preinduction optimization, 5% patients remained in Spo2 between 85% to 90%, 5.12% remained in mean arterial pressure (MAP) range of 50-60 mmHg (Table 3). 6% of patients had required dopamine/noradrenaline support to maintain

Table 1: Age with gender distribution of Patients

Group	Male	Female	Age (Years)		
			>65	18-65	8-18
n	27	13	5	31	4
%	67.50%	32.50%	12.50%	77.50%	10%

Table 2: Disease wise distribution of Patients

S No.	Disease/Surgical condition	n=40	%	Surgical intervention
1	Duodenal ulcer perforation peritonitis	23	57.50%	Graham's Omentopexy
2	Tubercular perforation peritonitis	2	5%	Ileostomy/Resection & Anastomosis
3	Appendicle perforation	3	7.50%	Appendectomy
4	Idiopathic peritonitis perforation	1	2.50%	Exploratory Laparotomy & Lavage
5	Traumatic Intestinal perforation	3	7.50%	Ileostomy
6	Perforated Hepatic abscess with peritonitis	1	2.50%	Lavage and Drain of abscess
7	Multiple bleeding polyp jejunum	1	2.50%	Resection Anastomosis
8	Inguinal hernia gangrene	1	2.50%	Herniorrhaphy with Resection & Anastomosis
9	Intestinal obstruction	3	7.50%	Resection Anasttommosis
10	Caecum perforation peritonitis	1	2.50%	Limited Right Hemicolectomy
11	Ruptured ectopic pregnancy	1	2.50%	Right Salpigiectomy Oophrectomy

MAP >60 mmHg (Table 3) Resuscitation lead to optimization of patients with 72.50% patients in ASA-3 E status and 27.50% patients remain in ASA 4 E status (Table 4) with tachycardia, tachypneoa, decreased urine output, requiring repeated ABG correction despite optimization. These patients were induced with sevoflurane. None of the patient required abandoning/modification of the induction due to coughing, aponea <20 secs (5%), agitation or dystonia or had hypotension, vomiting or aspiration, bronchospasm or arrythemias (Table 10). Only 2.5% had SpO_2 between 85%-90%

Table 3: Co morbid and Critical Physiological Status of Patient

S. No.		N=40	%
1	COPD	4	10%
2	SEPSIS	36	90%
3	Haemorrhagic shock	4	10%
4	Ischaemic Heart Disease	0	0%
5	Smokers	18	45%
6	Anaemia(Hb 10 or<10gm%)	18	45%
7	Dopamine?Noradrenaline	6	15%
8	Spo2 Opti PreIND (85-90%)	2	5%
9	Mean BP Opti. PreIND (51-60 mm)	3	5%

Table 4: American Society of Anesthesiology (ASA) Distribution of Patients

ASA – E Status	I-1	II- 2	III-3	IV-4	V-5	E
Pre Optimized	0	0	14	26	0	40
%	0	0	35%	65%	0	100%
Optimized	0	0	29	11	0	40
%	0	0	72.50%	27.50%	0	100%

Table 5: Duration of Surgical Anesthesia

Time- Minutes	80-120	121-150	151-180
N=40	32	6	2
%	80%	15%	5%

Table 6: Descriptive analysis of Preinduction with Mean Arterial Pressure, Pulseand Spo2 values.

Variables	Mean	S.D.	Minimum	Maximum
Optimized Preinduction MAP	*80.84*	*6.41*	*68.67*	*91.33*
1 min.PIND MAP	78.71	5.97	66	88
3 min.PIND MAP	85.17	4.85	74.33	93.33
5 min.PIND MAP	88.16	4.19	72.33	91.33
10 min.PIND MAP	82.11	4.42	70	90.67
15 min.PIND MAP	82.46	4.35	69.33	90
30 min.PIND MAP	82.54	3.81	77.33	90.67
60 min.PINDMAP	82.15	3.48	72	88
10 min.PETBMAP	84.9	4.09	76	94
Optimized Preinduction Pulse	*107.28*	*6.62*	*90*	*120*
1 min.PIND Pulse	105.05	6.21	90	122
3 min.PIND Pulse	109.75	5.99	96	125
5 min.PIND PULSE	104.92	4.62	93	114
10 min.PINDPulse	102.88	4.4	92	110
15 min.PIND Pulse	101.2	3.85	93	108
30 min.PINDPulse	101.5	3.55	93	108
60 min.PIND Pulse	97.5	5.76	90	112
10 min.PETB Pulse	102.42	3.43	96	108
Optimized Preinduction SpO_2	*96.05*	*2.54*	*90*	*99*
1 min.PIND SpO_2	94.52	14.07	90	99
3 min.PIND SpO_2	97.4	3.54	92	95
5 min.PIND SpO_2	97.2	1.8	92	99
10 min.PIND SpO_2	96.75	4.63	96	99
15 min.PIND SpO_2	97.95	1.43	94	100
30 min.PIND SpO_2	97.68	1.47	94	99
60 min.PIND SpO_2	97.5	1.37	94	99
10 min.PETB. SpO_2	96.18	1.8	91	99

PIND-Postinduction, PETB-Postextubation

Showing preinduction Mean BP of 80.84 mmHg. None of post induction values was less than this except at 1 minute 78.71 mmHg. None of values was below 66 mmHg.

postinduction as compare to 5% before induction. Duration of surgical anaesthesia management lasted 80 to 120 minutes in 80% of patients and 5% lasted for between 150-180 minutes (Table 5).

Preinduction optimize MAP changed significantly at 1, 3, 5, 10, 15 min. postinduction (Table 7) peaking at 3 minute postinduction (Table 6) remained above preinduction optimized basal level all the time. At preinduction 5.12% of patients had optimize MAP in range of 50-60 mmHg (Graph 1). Minimum mean MAP observed was 66 at 1 min. postinduction (Table 6).

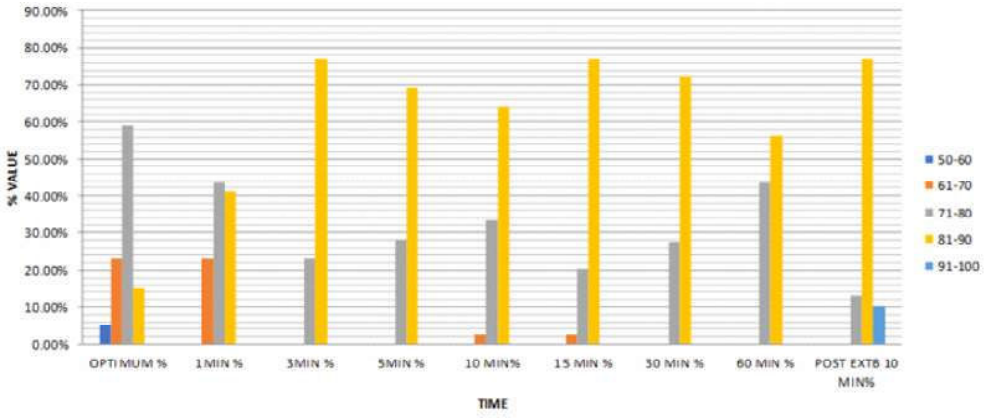
Graphic colour representation (Graph 1)showing % distribution of preinduction optimize and post induction peak MAP changes.

Changes in mean pulse values were significant at 1, 3, 5, 10 & 30 minutes postinduction as compare

Table 7: Analysis of variance between reference preinduction value of Mean Arterial Pressure (MAP) with different timing

Timing	Group	Sum of Squares	d.f	Mean Square	F	Sig.
1 min PIND MAP	Between Groups	1360.023	20	68.00	41.09	0.000
	Within Groups	31.442	19	1.66		
	Total	1391.465	39			
3 min. PIND MAP	Between Groups	835.139	20	41.76	9.63	0.000
	Within Groups	82.399	19	4.34		
	Total	917.538	39			
5 min PIND MAP	Between Groups	568.273	20	28.41	4.52	0.001
	Within Groups	119.373	19	6.28		
	Total	687.646	39			
10 min PIND MAP	Between Groups	660.929	20	33.05	6.15	0.000
	Within Groups	102.159	19	5.38		
	Total	763.088	39			
15 min PIND MAP	Between Groups	544.567	20	27.23	2.66	0.019
	Within Groups	194.804	19	10.25		
	Total	739.37	39			
30 min PIND MAP	Between Groups	367.758	20	18.39	1.74	0.116
	Within Groups	200.482	19	10.55		
	Total	568.24	39			
60 min PINDMAP	Between Groups	226.011	20	11.30	0.86	0.626
	Within Groups	248.402	19	13.07		
	Total	474.414	39			
10 min PINDMAP	Between Groups	451.143	20	22.56	2.13	0.053
	Within Groups	201.698	19	10.62		
	Total	652.841	39			

Table 7 showing significant changes in mean arterial pressure observed at 1,3,5,10 and 15 minutes post induction (PIND) as compare to preinduction. Changes were insignificatent at 30,60 postinduction and 10 min post extubation(PETB)



Graph 1: % distribution of Patients MAP in time variable
EXTB-Extubation, Optimum-Optimized or Basal preinduction

to optimize values (Table 8). None of values are less than 60 at any time. Optimize preinduction pulse depicted in (Table 6) showing peaked post induction rise at 3 minute postinduction (Table 6).

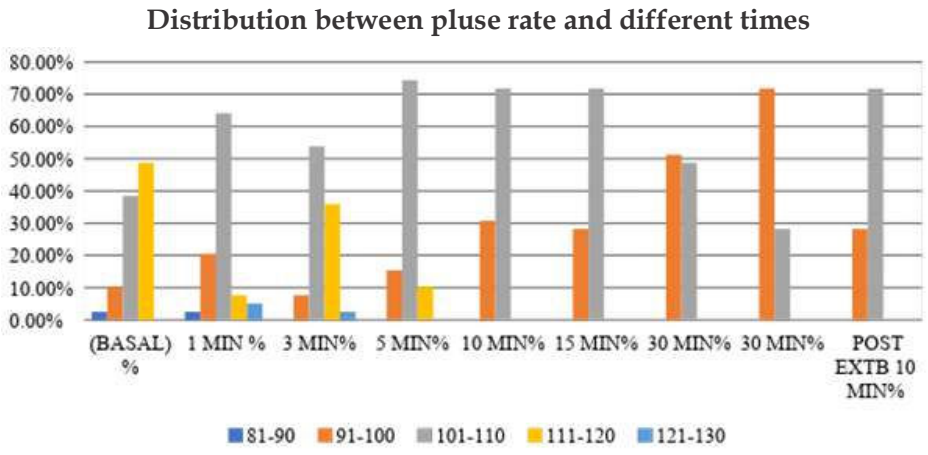
Except at 1 amd 15 min. changes in mean SpO_2 were significant at all times (Table 9) As compare to 5% preinduction only 2.50% of the patient had SpO_2 between 85%-90% range postinduction (Graph 3).

No airway complication reported (Table 10) except cough and apnonic episodes of <20 sec in 5% of patients without any modification or abandoning of induction process.

Table 8: Analysis of variance between preinduction pulse with different timing

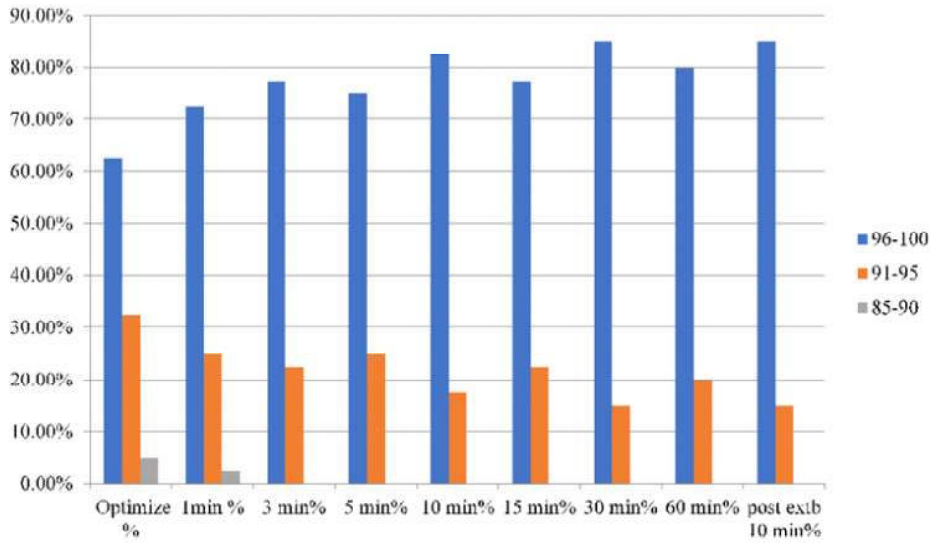
Timing	Variable	Sum of Squares	df	Mean Square	F	Sig.
1 min.PIND Pulse	Between Groups	1402.67	15	93.51	21.33	0.000
	Within Groups	105.23	24	4.39		
	Total	1507.90	39			
3 min.PIND Pulse	Between Groups	1228.82	15	81.92	11.52	0.000
	Within Groups	170.68	24	7.11		
	Total	1399.50	39			
5 min.PIND Pulse	Between Groups	716.54	15	47.77	9.86	0.000
	Within Groups	116.23	24	4.84		
	Total	832.78	39			
10 min.PIND Pulse	Between Groups	599.63	15	39.98	6.12	0.000
	Within Groups	156.75	24	6.53		
	Total	756.38	39			
15 min.PIND pulse	Between Groups	323.23	15	21.55	2.03	0.059
	Within Groups	255.17	24	10.63		
	Total	578.40	39			
30 min.PIND Pulse	Between Groups	321.23	15	21.42	2.98	0.008
	Within Groups	172.77	24	7.20		
	Total	494.00	39			
60 min.PIND Pulse	Between Groups	425.27	15	28.35	0.78	0.685
	Within Groups	870.73	24	36.28		
	Total	1296.00	39			
1 0 min.PETB Pulse	Between Groups	191.59	15	12.77	1.14	0.374
	Within Groups	268.18	24	11.17		
	Total	459.78	39			

Table 9 showing significant changes in pulse at 1, 3, 5, 10, 15 and 30 minutes as compare to preinduction optimize values. None of the time pulse rate was less than 60bpm.
PIND-Post Induction, PETB-Postextubation, min-minute



Graph 2: % Distribution of patient pulse rate in time variables

BASAL-Optimized Preinduction, Min-Minutes, EXTB-Extubation



Graph 3 % Distribution of patients spo2 in time variables

Min-Minutes, Extb.-Extubation, Optimize-Optimize Preinduction or Basal

Table 9: Analysis of variance between preinduction SPO2 with different timing

Timing	Variable	Sum of Squares	df	Mean Square	F	Sig.
1 min.PIND Spo2	Between Groups	1097.876	8	137.24	0.64	0.737
	Within Groups	6628.099	31	213.81		
	Total	7725.975	39			
3 min.PIND Spo2	Between Groups	239.419	8	29.93	3.71	0.004
	Within Groups	250.181	31	8.07		
	Total	489.6	39			
5 min.PIND Spo2	Between Groups	109.388	8	13.67	24.92	0.000
	Within Groups	17.012	31	0.55		
	Total	126.4	39			
10 min.PIND Spo2	Between Groups	160.508	8	20.06	0.92	0.514
	Within Groups	676.992	31	21.84		
	Total	837.5	39			
15 min.PIND Spo2	Between Groups	59.938	8	7.49	11.64	0.000
	Within Groups	19.962	31	0.64		
	Total	79.9	39			
30 min.PIND Spo2	Between Groups	58.454	8	7.31	8.61	0.000
	Within Groups	26.321	31	0.85		
	Total	84.775	39			
60 min.PIND Spo2	Between Groups	44.555	8	5.57	5.86	0.000
	Within Groups	29.445	31	0.95		
	Total	74	39			
10 min.PETB Spo2	Between Groups	102.797	8	12.85	16.48	0.000
	Within Groups	24.178	31	0.78		
	Total	126.975	39			

Significant changes in SpO_2 at all time except 1 and 10 minute postinduction.
PIND-Postinduction, PETB-Postextubation, SpO_2-Oxygen Saturation

Table 10: Postinduction -Perioperative Complication/Critical Incidence

Complication/ Critical incidence	Coughing/ Apnea<20s	Hypotension MAP<60 mmHg	Spo2<90%	Dystonia /Vomiting	Additional intervention	Abandoning/Modify Induction
Patients N=40	2	NIL	1	NIL	NIL	NIL
%	5%	0	2.5%	0	0	0

Discussion

Rationale of study is to utilize inhalational induction charateristices of sevoflurane i.e. non pungency and low blood gas solubility [1] and haemodynamic stability. Within 60 secs loss of eyelid reflex and conscious occurs with sevoflurane standard techniques of induction [10]. This retrospective study analysed sevoflurane induction of anaesthesia in critically ill patient undergoing emergency laparotomy. Most of the patients were in ASA 4 E status (65%). As table-1 showing majority (67.50%) were male, 77.50% patients were in age group of 18 to 65 years. Age affects MAC of sevoflurane and in critically ill patient require adjustment of % MAC is desirable particularly above 60 years. 6% sevoflurane instead of 8% above 65 years (15%) in critically ill at induction. 57.50% patients were of duodenal perforation peritonitis (Table 2) may be chornic smoking (45%) contribution to high incidence. Associated comorbid state sepsis (90%), haemorrhagic shock (10%), chronic smoker (45%) COPD (10%) and anaemia (45%) (Table 3). None had coronary artey disease or arrythemias clinically.

These patients were resuscitated to optimize their physiological and comorbid status from ASA4 E (65%) before induction to ASA 3E (72.50%) post optimization (Table 4) with fluid, colloids, blood and ionotrops like dopamine and noradrenaline. 5% patient had Spo2 85%-90% before induction (Table 9) Dopamine or noradrenaline required before induction in 6% of patients to maintain MAP 60 or above though 5.12% had MAP between 50-60 mmHg preinduction despite optimization before induction (Table 10).

Sevoflurane induction done using Mapelson D circuit with flow rate 80 to 100 ml/kg. This low flow compare to 100 to 200 ml/Kg allowed rebreathing to occur to maintain spontaneous respiration for faster induction [10]. This has the advantage that it allows ventilation to be sustained when consciousness is lost and specific maneuvre such as breath - holding or vital capacity breaths are not required to facilitate induction of anaesthesia as patients were critically ill [8]. After preoxygenation with 100% oxygen, stepwise increase addition of sevoflurane in increment of 1%, 2%, 4% and 8% every 2 breath allows an anaesthetist to assess airway patency and assist ventilation [2]. In such high concentration in emergency patients [14] inhalational induction of anaesthesia may parallel the speed of intravenous induction [10]. Induction with an initial concentration of 8% sevoflurane produces more rapid passage through the excitatory stage possibly resulting in fewer adverse events [15]. Maintainence of spontaneous ventilation is definitive advantage of sevoflurane particularly with potentially difficult airway in such critically ill patients [2,15]. Patient that had airway complication (Table 9) as coughing, laryngospasm, apnoea <20 sec (5%) resulting in abandoning or modifying induction were nil in our study indicating smooth induction charateristics of sevoflurane (Table 10) Nitrous oxide may add little to the efficacy of inhalational induction [16] though we have added 50% nitrous oxide to induction after achieving 8% sevoflurane concentration. Sevoflurane may be delivered in high inspired concentration without much airway complication may be attributed to a more rapid passage to a deeper plane of anaesthesia not associated with awareness [10]. Bronchodilator properties of sevoflurane have additional advantages in patients with COPD (10%) and chronic smoker (45%) and not associated with increased secretions, allergy and anaphylaxis. Only 2.5% had SpO_2 between 85%-90% postinduction as compare to 5% before induction Post induction only 2.5% of patient had SpO_2 in between 85 to 90% that to improved after 1 minute post induction showing beneficial effects of sevofluration induction (Graph 3). This improvement in SpO_2 observed due to its bronchodilatation properties, loss of pain, improving ventilation, application of PEEP and or positive pressure ventilation with improve oxygenation in basal atelectic lungs suggesting sevoflurane induction does not influence SpO_2 adversly if oxygenation ventilation is maintained during induction. Changes in SpO_2 were significant at perioperative period except 1 minutes and 15 min. though none of patient had SpO_2 less than 90% post induction anytime after one minute during perioperative period. Sevoflurane do affect ventilation perfusion significantly in hypoxic conditions [17]. Whether this result of SpO_2 fluctuation during perioperative period after 1 minute though within normal limits in patients is due to peripheral vasoconstriction or dilatation or changes in patient temperature reason is largely speculative critical ill.

As SpO_2 never below 90% after 1 min post induction. No active intervention required and positive pressure ventilation maintained with low PEEP of 2-4 mmHg with 35%-50% oxygen with nitrous oxide during perioperative management of patients.

Most intravenous anaesthetic agents as thiopental, propofol, midazolam may associated with haemodynamic instability in critically ill

patients. 90% patient were in sepsis and 10% in haemorrhagic shock in our study. In heart of septic rats maximal cardiac work dysfunction occurred in the order ketamine -6%, < etomidate -< 17%, midazolam 38% < profofol -50% as reported by Zausig et al. [13]. Etomidate has good haemodynamic induction profile but associated with adrenal suppression [13].

Ketamine is associated with undesirable psychotomimetic effects like illusion, delirium and disturbing dreams making assessment difficult after extubation and in ICU which is not observed with sevoflurane due to faster recovery from anaesthesia without any residual sedation or psychomimatic effects.

Postinduction mean MAP remain above preinduction mean values (except 66 mmHg at 1 min.) throughout peri-operative period. 5.12% of patients were in MAP 50-60 mmHg range before induction though none had MAP <60 mmHg (Table-9) post induction (Graph 2). This study found that changes in Mean Arterial Pressure (MAP) response were significant (Table 7) at 1 to 15 minutes post induction of anaesthesia. MAP peaked at 3 and 5 minutes post induction suggesting sevoflurane unable to inhibit adrenergic response of intubation though none of patient had MAP below 66 mmHg at any time showing haemodynamic stability with sevoflurane. Changes in pulse from optimize preinduction value to postinduction at 1 to 30 minutes post induction were significant (Table 8) peaking at 3 min postinduction. None of pateint had bradycardia or arrythemias (Table 10).

Sevoflurane induction result in increase in MAP and heart rate (Table 6,7,8) due to uninhibited sympathoadrenal responses of endotracheal intubation as though sevoflurane itself does not affect haemodynamics. At 1.5 MAC sevoflurane do not prevent haemodynamic response of incision. None of patient had bradycardia or MAP <60 mmHg. There is no increase in requirement of dopamine or noradrenaline support after induction of anaesthesia with sevoflurane. Its non arrythemogenic properties is particular value in critically ill patient with high catecholamine levels levels. Haemodynamic effects of sevoflurane voatile anaesthetic with N_2O are minimal compared with those of equi-MAC alone [12]. A CVP guided haemodynamic management, more potent short acting analgesic like fentanyle [18,19] may have more optimize anaesthetic management with sevoflurane induction attenuating haemodynamic responses in such patients though we did not find any hypotension or bradycardia or arrythemias or increased requirement of vasopressors with sevoflurne induction in such critically ill. Incidence of postoperative ventilator requirement or metabolic derangements which may likely to occur due to haemodynamic instability and residual sedation may be reduced. Sevoflurane has faster recovery characteristic as compare to ketamine, opiates and no adrenal suppression as compared to etomidate. Thus rational of sevoflurane has advantage of faster induction, haemodynamic stability [5], no adrenal suppression and quick recovery in emergent situations minimizing use or additional vasopressors to maintain haemodynamices in critically ill [13].

Conclusions

Sevoflurane has advantages of rapid inhalational induction, bronchodilation and relative haemodynamic stability and faster recovery [20,21]. This study concludes that sevoflurane likely to be agent of choice for anaesthetic induction and management of critically ill patients in emergency with rapid induction without airway complications, minimum haemodynamic disturbances, least requirement of vasopressors, early recovery in emergency laparotomy with all prophylaxsis to prevent aspiration. Sevoflurane unable to attenuate adrenergic responses of laryngoscopy and endotracheal intubation.

References

1. Peter K, Conzen P. Inhalational Anaesthesia. Clinical Anaesthesiology. 1993;7:898-913.
2. Mostafa SM, Atherton AMJ. Sevoflurane for difficult tracheal intubation. British Journal of Anaesthesia. 1997;79:392-3.
3. Hall JE, Ebert TJ, Harmer M. Induction characteristics with 3% and 8% sevoflurane in adults. An evaluation of second stage of anaesthesia and its haemodynamic consequences. Anaesthesia. 2000;55:545-50.
4. Yurimo M, Kimura H. A comparison of vital capacity breath and tidal breathing technique for induction of Eanaesthesia with high sevoflurane concentration in nitrous oxide and oxygen. Anaesthesia. 1995;50:308-11.
5. Djaiani GN, Hall JF, Pugh S, Peaston RT. Vital capacity inhalational induction with sevoflurane: An alternative to standard intravenous induction for patients undergoing cardiac surgery. Journal of Cardiothorasic vascular Anesthesia. 2001;15:169-74.
6. Ebert TJ, Frink Jr. EJ, Karasch ED. Absence of biochemical evidence for renal and hepatic

dysfunction after 8 hours of 1.25 minimum alveolar concentration sevoflurane anaesthesia and its haemodynamic consequences. Anaesthesia . 2000;55:545-50.

7. Kumar A, Mullick P, Prakash S, Bharadwaj A. Consent and the Indian medical practioner. Indian J. Anaesthesia 2015;59:695-700.
8. Knaggs CL, Drummond GB. Randomized comparison of three methods of induction of anaesthesia with sevoflurane. British J Anaesth. 2005;95(2):178-82.
9. Goodwin N, Campbell AE, Hall JE, Plummer S, Harmer M. A comparison of 8% and 12% sevoflurane for inhalational induction in adults. Anaesthesia. 2004;59:15-19.
10. Muzi M, Robinson BJ, Ebert TJ, O Brian TJ. Induction of anaesthesia and tracheal intubation with sevoflurane in adult. Anesthesiology. 1996;85(3):536-543.
11. OShea H, Moultries S, Drummond GB. Influence of nitrous oxide on induction of anaesthesia with sevoflurane. British J Anaesth. 2001;87:286-8.
12. Inada T. Haemodynamic comparison of sevoflurane and isoflurane anaesthesia in surgical patients. Canadian Journal of Anaesthesia. Feb 1997.
13. Seok HW. Concerns of anaesthesiologist: Anaesthesia induction in severe sepsis or septic shocked patients. Korean J Anaesthesiol. 2012;83(1):3-10.
14. Moore EW, Davis MW. Inhalational versus Intravenous induction. A survey of emergency anaesthetic practice in the United Kingdom. European Journal of Anaesthesiology. 2000;17:33-37.
15. Joo HS, Perks WJ. Sevoflurane versus propofol for anesthetic induction: a metaanalysis. Anaesthesia and Analgesia. 2000;91(1):213-219.
16. Thwaites A, Smith I. Sevoflurane for difficult tracheal intubation. British Journal of Anaesthesia. British Journal of Anaesthesia. 1998;81:103-4.
17. Beck DH, Doepfmer UR, Sinemus C, Bloch A, Schenk MR, Kox WJ. British Journal of Anaesthesia 2001 Jan;86(1):38-43.
18. Katoh T, Nakajima Y, Moriwaki G, Kbayashi S, Suzuki A, Iwamoto T, Bito H, Ikeda K. Sevoflurane requirement for tracheal intubation with and without fentanyl. British Journal of Anaesthesia 1999;82(4):561-5.
19. Hoda A, Khan FA. Effect of one minimum alveolar concentration of sevoflurane with or without fentanyl on hemodynamic response to laryngoscopy and tracheal intubation. J Anaesthesiol clin Pharmacol. 2011 Oct-Dec;27(4):522-526.
20. Sevoflurane-FDA, USA prescribing information side effects and uses.
21. Herrera LD, Ostroff KD, Rogers SA. Sevoflurane: Approaching the ideal inhalational anesthetic. A pharmacologic, pharmacoeconomic and clinical review. CNS Drug Review. 2001 Spring;7(1),48-120.