Original Article

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A Comparative Study between IV Clonidine 1.5Mcg/Kg and IV Lignocaine 1.5 Mg/Kg as Premedication for Attenuation of Hemodynamic Responses to Pneumoperitoneum in Laparoscopic Surgeries

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Abstract

Laparoscopy, has emerged as the main stay for most of the commonly performed surgeries. Pneumoperitoneum and different positions of the patient during laparoscopy contribute to adverse cardiovascular effects like elevated arterial pressure, increased systemic and pulmonary vascular resistance and reduced cardiac output. This randomised controlled double blind prospective study was conducted to compare the efficacy of intravenous(I.V) Lidocaine and I.V. Clonidine in providing hemodynamic stability in patients undergoing laparoscopic surgeries.

Methods: Seventy five adult patients of ASA grade 1 and 2, scheduled for elective laparoscopic surgery with a mean duration of 30- 90 minutes, were randomly allocated to one of the three groups.

Group A: Clonidine group (n=25) - received injection Clonidine 1.5 mcg/kg 15 minutes prior to induction of anaesthesia.

Group B: Lidocaine group (n=25) - injection Lidocaine 1.5 mg/kg 90 seconds prior to induction of anaesthesia.

Group C: Control group (n=25) - received 10 ml normal saline intravenously.

Results: There is no statistically significant difference between lidocaine and control group regarding mean HR, SBP, DBP, MAP during the entire period of pneumoperitoneum. Whereas, there is significant increase of blood pressures (SBP, DBP, MAP) in the lidocaine and control groups compared to the clonidine group from intubation, throughout the period of pneumoperitoneum. Clonidine also decreases the requirement of propofol for induction.

Conclusion: I.V Clonidine 1.5mcg/kg premedication is more effective than Lidocaine 1.5mg/kg at preventing changes in hemodynamic parameters induced by carbondioxide pneumoperitoneum.

Keywords: Pneumoperitoneum, I.V Clonidine, I.V Lignocaine, Hemodynamic parameters.

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Introduction

Laparoscopy, being a minimally invasive surgery is associated with better homeostasis, reduced trauma to the patient, decrease morbidity and mortality. The pneumoperitoneum and different positions of the patient, Trendelenberg to reverse trendelenberg contribute to adverse cardiovascular effects like elevated arterial pressure, increased systemic and pulmonary vascular resistance and reduced cardiac output.[10] It also results in stress hormone responses (norepinephrine, epinephrine, cortisol and rennin.[4,6] Clonidine is an α2 adrenoreceptor agonist, exerts central sympatholytic effect and blunts the stress response. Lidocaine, a membrane stabilising agent and peripheral vasodilator, provides hemodynamic stability during laryngoscopy and intubation and also during laparoscopic surgeries. Hence, the study to compare the efficacy of both the drugs in providing hemodynamic stability in patients undergoing laparoscopic surgeries.

Methods

This prospective double blind randomised controlled study was conducted in 75 adult patients of ASA grade 1 and 2, scheduled for elective laparoscopic cholecystectomy and appendicectomy. This study was approved by the institutional ethical committee and written informed consent was obtained from all the patients before being included in the study. Patients with hypertension, ischaemic heart disease, cerebrovascular diseases, diabetes mellitus, endocrine disorders, body mass index >28 were excluded from the study. Patients on drugs which alter the autonomic nervous system activity were also excluded.

Patients were randomly allocated into one of the three groups (A, B and C) according to computer generated tables of random numbers.Baseline investigations such as haemoglobin, blood sugars, blood urea, serum creatinine and electrolytes of all the groups were recorded and comparable.

On arrival in the operation theatre, monitors were attached and baseline parameters like heart rate, oxygen saturation, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were noted. Two 10 ml syringes were taken. The first syringe was loaded with either clonidine or lidocaine or plain saline. The second syringe loaded with 10 ml plain saline. The drug solution was prepared according to a random number table. The anaesthesiologist giving the drug was blinded to the treatment group.

Group A received I.V.Clonidine 1.5 mcg/kg body weight diluted to 10ml with normal saline, 15 min prior to induction and 10 ml normal saline 90 seconds prior to induction.

Group B received normal saline at 15 minutes prior to induction and I.V. Lidocaine 1.5 mg/kg body weight diluted with normal saline to 10 ml, 90 seconds prior to induction.

Group C received saline at both 15 minutes and 90 seconds prior to induction.

All the patients were preoxygenated and received Inj. Fentanyl 2mcg/kg. Induction done with I.V Propofol and Inj. Rocuronium 0.9 mg/kg. Dose of Inj. Propofol was titrated with the loss of verbal response to oral commands (end point of induction) and was noted down. Endotracheal intubation done and End tidal carbondioxide ($EtCO_2$) and oxygen saturation were monitored throughout the surgery. Maintenance of anaesthesia with nitrousoxide 60 % and oxygen 40 % and sevoflurane.

The heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressure (MAP) at different time points at baseline, before induction, after induction, immediately after intubation, subsequently at 1 min, 3min, 5min after intubation, before pneumoperitoneum and subsequently 1min, 3 min, 5 min, 15min, 30 min and 45 min after pneumoperitoneum were noted .

After completion of surgery, residual neuromuscular blockade was reversed by neostigmine and glycopyrrolate and were extubated once the patients were awake and met the extubation criteria.

Time for recovery was recorded - which was defined in the present study as, time from reversal to extubation. The sedation score was done immediately after extubation as per Ramsay sedation scoring. Patients were monitored for any adverse event like desaturation, excessive sedation and post operative nausea and vomiting in the post anesthesia care unit (PACU) for 24 hours.

Statistical Methods Employed

Frequencies, Crosstabs, Descriptive statistics, One-way ANOVA, Scheffe's Post hoc test, Repeated measure ANOVA, Contingency table analysis. P value <0.05 was considered as significant and $p<0.01$ was considered as highy significant.

Results

All three groups were comparable with respect to gender (Figure 1), age and weight.

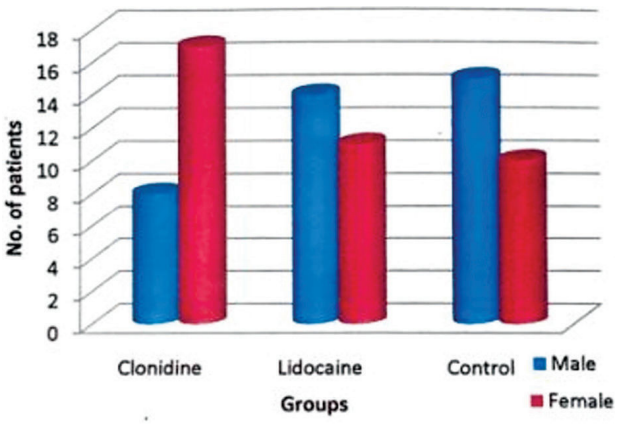

Fig. 1: Gender distribution in all the three groups.

Heart rate response

Table 1: Showing the comparision of mean heart rate (bpm) changes between all the three groups.

Group	Clonidine	Lidocaine	Control	P value
Baseline	86.8 ±16.8	88.6 ±9.2	86.4 ±15.3	0.848
Before induction of anesthesia	83.1 ±16.9	86.7 ±8.9	86.8 ±16.0	0.597
After induction of anaesthesia	79.8 ±15.8	84.2 ±9.4	88.8 ±15.4	0.079
Immediately after intubation	83.6 ±13.8	96.6 ±16.4	97.3 ±16.5	0.004
1 minute after intubation	86.8 ±16.7	98.3 ±13.6	101.7±15.9	0.003
3 minutes after intubation	80.5 ±15.5	93.2 ±12.8	100.3±15.9	0.000
5 minutes after intubation	78.2 ±15.9	88.7 ±13.7	96.1±15.4	0.000
Before pneumop-eritoneum	76.2 ±16.9	84.3 ±11.9	91.5±16.3	0.003
1 minute after pneumop-eritoneum	77.7 ±17.2	83.4 ±12.9	89.9±17.8	0.034
3 minutes after pneumo-peritoneum	77.4 ±15.0	85.5 ±11.3	89.6±15.3	0.011
5 minutes after pneumo-peritoneum	78.9 ±13.5	85.0 ±10.5	89.2 ±13.4	0.017
15 minutes after pneumo-peritoneum	77.5 ±13.5	86.0 ±11.3	88.0 ±14.8	0.016
30 minutes after pneumo peritoneum	76.4 ±14.9	84.4 ±11.5	89.1 ±14.0	0.006
45 minutes after pneumo peritoneum	75.0 ±14.3	84.1 ±10.9	89.7 ±13.5	0.001

Mean heart rate response during intubation, in the clonidine group was low compared to the lidocaine and the control group. It was statistically highly significant. The mean heart rate response was comparable between the lidocaine and the control groups. The mean heart rate in clonidine group remained significantly low throughout the pneumoperitoneum compared to the control group. The lidocaine group had the mean heart rate almost comparable to the control group and the clonidine group throughout the pneumoperitoneum (Fig. 2).

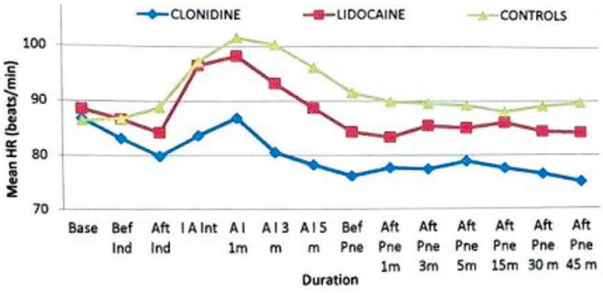

Fig. 2: Mean heart rate (bpm) changes between all three groups.

Systolic Blood Pressure Response

Table 2: Showing the comparision of mean systolic blood pressure (mm of Hg) changes between all the three groups.

Group	Clonidine	Lidocaine	Control	P value
Baseline	126.0±14.4	128.8±18.4	126.8 ±11.9	0.797
Before induction of anesthesia	110.4±12.0	121.4±15.1	125.0 ±13.6	0.001
After induction of anaesthesia	97.1 ±10.5	116.2±13.2	117.2±15.7	0.000
Immediately after intubation	104.3±10.7	120.5±15.1	127.9±24.5	0.000
1 minute after intubation	109.4±19.5	114.5±11.6	135.9±20.7	0.000
3 minutes after intubation	99.1 ±16.5	108.6±13.2	122.5±19.4	0.000
5 minutes after intubation	94.9 ±10.5	105.8±13.0	118.8±17.2	0.000
Before pneumo-peritoneum	99.6 ±13.7	114.9±14.0	117.6±15.9	0.000
1 minute after pneumo-peritoneum	114.2±19.9	120.9±16.6	123.6±14.9	0.145
3 minutes after pneumo-peritoneum	127.3±11.9	127.8±18.3	134.6±16.3	0.192
5 minutes after pneumo-peritoneum	126.7±17.9	132.5±20.8	140.3±18.2	0.045
15 minutes after pneumo-peritoneum	124.4±17.8	134.0±19.8	142.8±18.7	0.004
30 minutes after pneumo-peritoneum	123.1 ±16.2	136.2 ±18.1	141.3 ±17.6	0.001
45 minutes after pneumo-peritoneum	118.9 ±16.3	134.0 ±17.6	140.6 ±15.7	0.000

Statistical analysis between the groups showed that increase in the mean systolic pressure observed in control group was statistically highly significant compared to the clonidine group immediately after intubation and at 1, 3, 5 min after intubation and at 1, 3, 5 min after intubation when compared to lidocaine group. The increase in mean systolic pressures in lidocaine group was statistically significant only at immediately after intubation and after 5 min after intubation compared to the clonidine group.

The mean systolic pressures in all the three groups were comparable at 1 and 3 min after pneumoperitoneum. Thereafter, clonidine group had statistically significant lower mean systolic pressures compared to the control group till the end of pneumoperitoneum.

Whereas clonidine group when compared to lidocaine group, the mean systolic pressures were comparable between the two groups till 15 min after pneumoperitoneum and thereafter clonidine group significantly had lower mean systolic pressures compared to the lidocaine group till the end of pneumoperitoneum. Comparision between the lidocaine and control group showed no significant difference throughout the pneumoperitoneum (Figure 3).

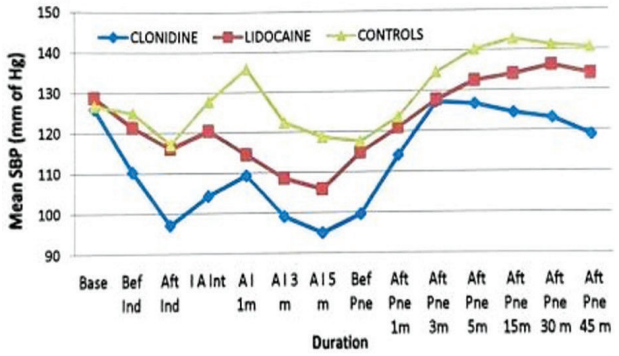

Fig. 3: Mean systolic blood pressure (mm of Hg) changes between all the three groups.

Diastolic Blood Pressure Response

The mean diastolic pressures in the clonidine group at 1 and 3 min after intubation were comparable with lidocaine group and significantly low at 5 min after intubation. The mean diastolic pressures in the control group remained significantly (P=0.00) high compared to the clonidine and the lidocaine group at 1, 3, and 5 min after intubation.

Table 3: Showing the comparision of mean diastolic blood pressure (mm of Hg) changes between all the three groups.

Group	Clonidine	Lidocaine	Control	P value
Baseline	82.9 ±10.0	83.7 ±9.8	85.6 ±8.5	0.609
Before induction of anesthesia	74.1 ±8.4	81.4 ±9.6	85.0 ±9.0	0.000
After induction of anaesthesia	65.8 ±8.9	78.5 ±10.2	79.4 ±11.8	0.000
Immediately after intubation	72.6 ±10.7	84.2 ±9.3	89.6 ±18.5	0.000
1 minute after intubation	74.9 ±12.9	80.0 ±9.9	94.2 ±17.3	0.000
3 minutes after intubation	66.4 ±12.7	74.4 ±9.8	86.6 ±15.6	0.000
5 minutes after intubation	63.6 ±8.7	72.4 ±10.0	81.9 ±14.0	0.000
Before pneumo-peritoneum	67.9 ±9.9	79.8 ±8.4	81.8 ±12.5	0.000
1 minute after pneumo-peritoneum	79.6 ±14.3	85.2 ±10.3	86.5 ±14.2	0.144
3 minutes after pneumo-peritoneum	91.2 ±10.7	91.3 ±11.9	98.2 ±13.2	0.065
5 minutes after pneumo-peritoneum	88.4 ±11.8	92.8 ±10.9	100.5 ±12.3	0.002
15 minutes after pneumo-peritoneum	85.4 ±12.5	93.5 ±11.4	101.4 ±12.9	0.000
30 minutes after pneumo-peritoneum	86.0 ±10.5	94.5 ±10.8	100.7 ±11.0	0.000
45 minutes after pneumo-peritoneum	84.1 ±9.9	93.8 ±11.4	101.5 ±10.5	0.000

During pneumoperitoneum, the mean diastolic pressures in all the three groups were comparable at 1 and 3 min after pneumoperitoneum. Thereafter clonidine group had statistically significant lower mean diastolic pressures compared to the control group till the end of pneumoperitoneum (P=0.00). Whereas, clonidine group when compared to lidocaine group, the mean diastolic pressures were comparable between the two groups till 15 min after pneumoperitoneum and thereafter clonidine group significantly had lower mean diastolic pressures compared to the lidocaine group till the end of pneumoperitoneum (P=0.00). Comparision between the lidocaine and control group showed no significant difference throughout the pneumoperitoneum except at the end of the pneumoperitoneum (Figure 4).

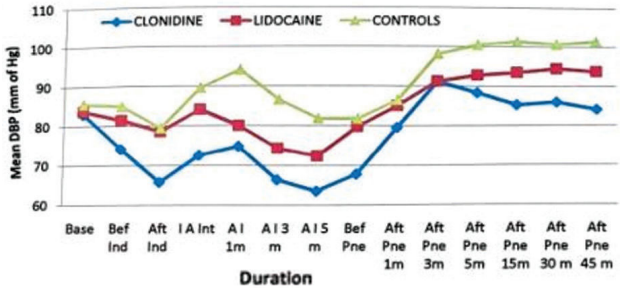

Fig. 4: Mean diastolic blood pressure (mm of Hg) changes between all the three groups.

Mean Arterial Blood Pressure Response

Table 4: Showing the comparision of mean arterial blood pressure (mm of Hg) changes between all the three groups.

Group	Clonidine	Lidocaine	Control	P value
Baseline	97.3 ±10.8	98.8 ±12.1	99.3 ±8.5	0.790
Before induction of anesthesia	86.2 ±9.0	94.8 ±10.3	98.3 ±10.0	0.000
After induction of anaesthesia	76.2 ±8.7	91.1 ±10.6	92.0 ±12.4	0.000
Immediately after intubation	83.1 ±9.9	96.3 ±10.8	102.4 ±20.1	0.000
1 minute after intubation	86.4 ±14.7	91.5 ±10.2	108.1 ±17.8	0.000
3 minutes after intubation	77.3 ±13.6	85.8 ±10.5	98.6 ±16.6	0.000
5 minutes after intubation	74.0 ±8.9	83.6 ±10.6	94.2 ±14.6	0.000
Before pneumo-peritoneum	78.4 ±10.9	91.5 ±9.9	93.7 ±13.1	0.000
1 minute after pneumo-peritoneum	91.1 ±15.8	97.1 ±12.2	98.9 ±13.9	0.128
3 minutes after pneumo-peritoneum	103.2 ±10.5	103.5 ±13.6	110.3 ±13.9	0.088
5 minutes after pneumo-peritoneum	101.1 ±13.3	106.1 ±13.8	113.8 ±13.9	0.007
15 minutes after pneumo-peritoneum	98.4 ±13.4	107.0 ±13.9	115.2 ±14.4	0.000
30 minutes after pneumo-peritoneum	98.4 ±11.8	108.4 ±12.9	114.0 ±13.0	0.000
45 minutes after pneumo-peritoneum	95.7 ±11.6	107.2 ±13.2	114.3 ±11.9	0.000

The mean arterial pressures in the control group remained significantly high (p=0.00) at 1, 3 and 5 min after intubation compared to the lidocaine and the clonidine groups. The mean arterial pressures of clonidine and the lidocaine group were comparable at 1 and 3 min after intubation but significantly low at 5 min after intubation in clonidine group.

The mean arterial pressures in all the three groups were comparable at 1 and 3 min after pneumoperitoneum. Thereafter clonidine group had statistically significant lower mean arterial pressures compared to the control group till the end of pneumoperitoneum (P=0.00). Whereas, clonidine group when compared to lidocaine group, the mean arterial pressures were comparable between the two groups till 15 min after pneumoperitoneum and thereafter clonidine group significantly had lower mean arterial pressures compared to the lidocaine group till the end of pneumoperitoneum (P=0.00). Comparision between the lidocaine and control group showed no significant difference throughout the pneumoperitoneum (Figure 5).

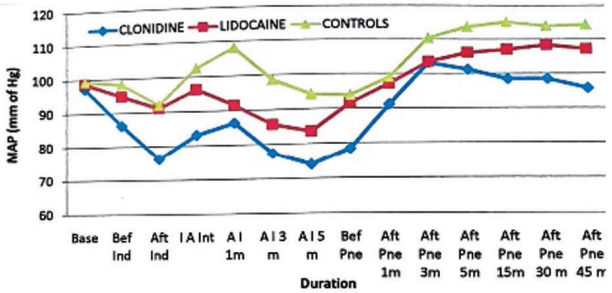

Fig. 5: Mean arterial blood pressure (mm of Hg) changes between all the three groups.

Table 5: Mean dose of propofol required for induction of anaesthesia in all the three groups.

Group	Clonidine	Lidocaine	Control	P value
Propofol	74.8 ±14.2	87.4 ±24.3	93.6 ±9.9	0.001

The reqiurement of propofol in the clonidine group was significantly low compared to the lidocaine group and the control group. The requirement of propofol in lidocaine group was significantly low compared to the control group (Figure 6).

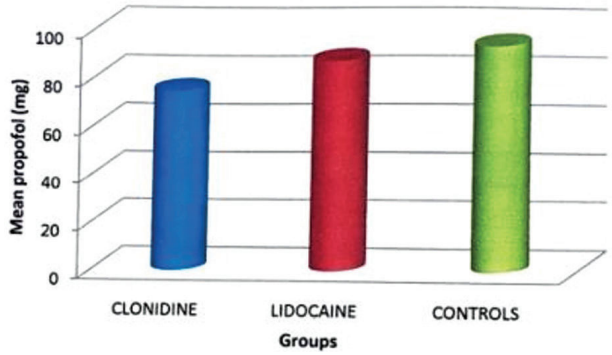

Fig. 6: Mean dose of propofol required for induction of anaesthesia in all the three groups.

Table 6: Showing mean time for recovery (seconds) in all the three groups.

Groups	Clonidine	Lidocaine	Control	p value
Time for recovery(sec)	42.54±21.88	39.08±13.53	27.84±10.23	0.006

The above table shows that the time for recovery is prolonged significantly in both clonidine (+14.7 sec) and the lidocaine(11.24 sec) group compared to control group (Figure 7).

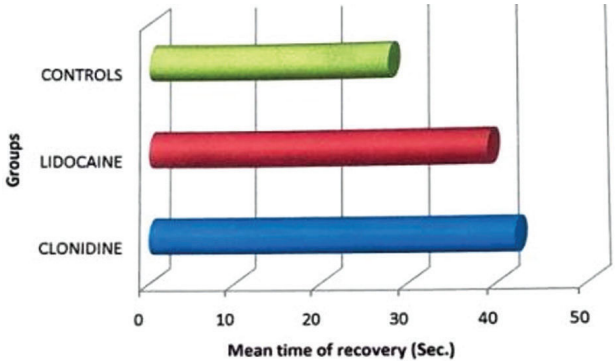

Fig. 7: Mean time for recovery (seconds) in all the three groups.

Table 7: Showing the percentage of patients in each group with respect to Sedation score based on ramsay sedation score in all the three groups.

Ramsay Sedation Score	Clonidine	Lidocaine	Control
Anxious/ restless/both	0%	0%	0%
Cooperative, oriented and tranquil	84%	80%	80%
Responding to commands	8%	12%	20%
Brisk response to stimulus	4%	4%	0%
Sluggish response to stimulus	4%	4%	0%
No response to stimulus	0%	0%	0%

Majority of the patients in all the three groups belong to cooperative, oriented and tranquil (score 2). There was no statistically significant difference in the sedation scoring among the three groups (p =0.753) (Figure 8).

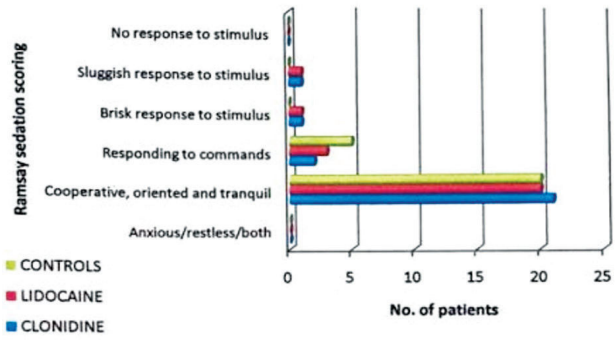

Fig. 8: Showing the percentage of patients in each group with respect to sedation score based on ramsay sedation score in all the three groups.

Side effects attributed to study drugs like nausea, vomiting, hypotension, bradycardia were not noticed in either of the three groups.

Discussion

Laparoscopy has been promoted aggressively as "gentle" surgery with minimal tissue trauma, reduced postoperative complications, and early recovery to normal level of activity. This, indeed, is generally true, but the procedure is not risk free. The patients undergoing laparoscopic procedures pose the problem of labile hemodynamics to the anaesthesiologist. In laparoscopic surgery, pneumoperitoneum is routinely created, which elevates intra abdominal pressure and produces some adverse effects on the cardiovascular system.

Cunningham et al[2] assessed the ejection fraction (EF) of left ventricle by transesophageal echocardiography during pneumoperitoneum. No significant change in ejection fraction was reported up to 15 mmHg of intra-abdominal pressure. Considering all these facts intra abdominal pressure was kept below 14 mm Hg.

Increased catecholamine level activates renin-angiotensin aldosterone system (RAAS) leading to some characteristic hemodynamic alterations which include decreased cardiac output (25-35%), elevated arterial pressure and increased systemic/pulmonary vascular resistance.[6] Induction of pneumoperitoneum and changes in position appear to be associated with a biphasic hemodynamic response, with an early reduction followed by partial recovery of cardiac index. Normal heart can cope with the increase in afterload under physiologic conditions. But patients with compromised cardiac function may not be able to tolerate the changes in afterload produced by pneumoperitoneum and it may have deleterious effects on their hemodynamics.

Carbondioxide pneumoperitoneum leads to carbondioxide absorption from the peritoneal surface and may lead to clinically relevant hypercapnia and acidosis. In the event of any impaired alveolar ventilation by altered ventilatory mechanics and/or due to compromised cardiopulmonary status, the additional carbondioxide is not cleared, resulting in hypercarbia and acidosis. Hypercapnia may also contribute to changes in MAP and plasma concentrations of adrenaline.[8] Clonidine an α2 agonist, has been found by various authors to blunt the hemodynamic response to laparoscopic pneumoperitoneum.[4,5,9] Clonidine is a highly lipid soluble drug and readily distributes into extravascular sites including the central nervous system. The onset of action of intravenous clonidine is within 11±9 min. Hence, in our study intravenous clonidine was given 15 min prior to the induction of anaesthesia.

Lidocaine a local anaesthetic, has been found by many authors to blunt the hemodynamic response to laryngoscopy and intubation.[12] Lidocaine is known to maintain the hemodynamic stability possibly by the membrane stabilising action and peripheral vasodilating effect. Onset of action of lidocaine is between 60-90s. Hence, in our study i.v. lidocaine was given 90 seconds prior to induction of anaesthesia.

Dose of clonidine employed and administered

Many authors[5,9] have found oral clonidine 150 mcg (as a premedicant 90 min prior to induction of anaesthesia) effective in attenuating the hemodynamic reponse to laparoscopic pneumoperitoneum. In the present study we have used clonidine 1.5 mcg/kg i.v. which is concurring with the effective dose available after oral clonidine 150 mcg, the bioavailability of clonidine being 70% .

Dose of lidocaine employed and administered

Authors have used lidocaine 0.5 mg/kg[3] and 1.5 mg/kg[7] i.v to attenuate the stress response to laparoscopic pneumoperitoneum. Many studies have used lidocaine 1.5 mg/kg i.v[12] to attenuate the stress response to laryngoscopy and intubation. Hence in our study we have chosen lidocaine 1.5 mg/kg i.v. as a single bolus premedicant to attenuate the hemodynamic response to laparoscopic pneumoperitoneum.

The results of the present study with regards to increase in HR are similar to studies by Jean L Joris et al[4], and Shivinder singh et al.[9] The results of the present study with regards to increase in mean arterial pressure are similar to studies by Mrinmoy et al[5] and Shivinder singh et al.[9]

There is no increase in the heart rate in clonidine group in the present study concurring with the studies by Mrinmoy et al[5] and Shivinder singh et al.[9] There is very less increase of MAP in the clonidine group similar to studies by Jean L Joris et al[4] and Shivinder singh et al.[5]

The change in the mean heart rate in lidocaine group, which has decreased slightly following pneumoperitoneum is similar to the studies by Boccara et al[3] and Qazi ehsan ali et al[7] The change in the MAP in the present study group is similar to the study by Boccara et al.[3]

Comparision of lidocaine and clonidine group

Heart rate response: Both the clonidine and lidocaine groups after premedication, throughout pneumoperitoneum had very stable HR. There was no statistically significant change in HR between the two groups.

Blood pressure response: There is a statistically significant difference from the period after administration of study drugs till 45 min after pneumoperitoneum (except at 1st and 3rd minute after pneumoperitoneum).

There is significant increase of blood pressures (SBP, DBP, MAP) in the lidocaine group compared to the clonidine group from intubation, throughout the period of pneumoperitoneum.

This shows that clonidine effectively attenuates the hemodynamic response of laryngoscopy and intubation and laparoscopic pneumoperitoneum compared to lidocaine.

This is due to the decrease in catecholamine levels by clonidine[4] unlike lidocaine. The other reason is i.v. clonidine has a duration of action of 90 min and i.v. lidocaine has a shorter duration of action of 20 min.

Comparision between lidocaine and control group

There is no statistically significant difference between lidocaine and control group regarding mean HR, SBP, DBP, MAP which does not concur with the study done by Qazi ehsan ali[7], wherein similar dose (1.5mg/kg) as our study has been used. Yet there's a statistically significant difference between control and lidocaine which does not concur with our study. This is probably due to not using any opioid premedication in their study. In our study, for both the lidocaine and control group inj. Fentanyl 2mcg/kg i.v has been used as a premedication. It has been found by studies done by Helfman et al[11] that there is no difference in the hemodynamic responses comparing lidocaine and fentanyl. The finding of this study concurs with our study.

Dose of propofol required for induction is clinically and statistically significantly low in clonidine group which concurs with the study done by Altan A et al.[1]

References

1. Altan A, Turgut N, Yildiz F, Turkmen A, Ustin H. Effects of magnesium sulphate and clonidine on propofol consumption, hemodynamics and post operative recovery. British Journal of Anaesthesia 2005; 93(4):438-41.
2. Cunningham AJ, Turner J, Rosenbaum S.

Transoesophageal echocardiographic assessment of haemodynamic function during laparoscopic cholecystectomy. Br J Anaesth 1993; 70: 621-5.

3. G.Boccara, J.Eliet, Y.Pouzeratte, C.Mann, P.Colson. Pre-emptive lidocaine inhibits arterial vasoconstriction but not vasopressin release induced by a carbondioxide pneumoperitoneum in pigs. Br. J. Anaesth 2003; 90 (3):343-8.
4. Joris JL,Jean Daniel Chiche, Jean-Luc M.Canivet. Hemodynamic changes induced by laparoscopy and their endocrine correlates: Effects of clonidine. JACC1998; 32:1389-96.
5. Mrinmoy Das, Manjushree Ray, Gauri Mukherjee: Effect of clonidine premedication on Haemodynamic changes during laparoscopic cholecystectomy. Indian Journal Anaesthesia 2007; 51: 205-10.
6. O' leary E, Hubbard K, Tormey W. Laproscopic cholecystectomy: haemodynamic and neuroendocrine responses after pneumoperitonium and changes in position.British Journal of Anaesthesia 1996; 76: 640-4
7. Qazi Ehsan Ali, Obaid A Siddiqui, Yasir A. Khan. Effect of Xylocard pretreatment on hemodynamics in patients undergoing laparoscopic cholecystectomy. Rawal medical Journal2010; 35(2):188-91.
8. Rasmussen JP, Dauchot PJ,DePalma RG. Cardiac function and hypercarbia. Archieves of surgery 1978; 113:1196-200.
9. Shivinder singh and Kapil arora. Effect of premedication of Clonidine on perioperative haemodynamics and requirement of post operative analgesia in laparoscopic cholecystectomy. Indian Journal of Anaesthesia 2011; 55: 26-30.
10. Siamack Alishahi, Nadir Francis, Sally Crofts. Central and peripheral adverse hemodynamic changes during laparoscopic surgery and their reversal with novel intermittent sequential pneumatic compression device. Ann Surg 2001; 233(2):176-82.
11. Steven M.Helfman, Martin I. Gold, Everard A. DeLisser, Claire A. Herrington. Which drug prevents Tachycardia and hypertension associated with tracheal intubation: Lidocaine, Fentanyl, or Esmolol. Anesth Analg1991; 72:82-6.
12. Yoshiro Hamaya, Shuji Dohi. Differences in cardiovascular response to airway stimulation at different sites and blockade of the responses by lidocaine. Anesthesiology2000; 93:95-103.