ISSN 2515-8260 Volume 08, Issue 04, 2021

Effect of Dexmedetomidine Infusion on Haemodynamic Responses to CapnoperitoneuminPatients Undergoing Elective Laparoscopic Cholecystectomy under General Anaesthesia

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Abstract
Dexmedetomidine (Dexmed) belongs to the imidazole subclass of α_2 receptor agonists, similar to clonidine. It is a potent and highly selective α_2 adrenoreceptor agonist,approved by FDA in 1999 for use in humans for analgesia and sedation. It has a greater affinity for α_2 receptors over α_1 (1620:1). Its α_2/α_1 selectivity ratio is eight times higher than that of clonidine. All patients underwent a thorough preanaesthetic check-up. Written informed consent was obtained from each patient after explaining the procedure in a language that the patient understood. Patients were kept NPO from midnight and advised Tab. Diazepam (0.1-0.15 mg/kg) and Tab. Rantac 150 mg night before and at 6 AM on the morning of surgery with sips of water. The mean E_{tsevo} in Group D was 0.29±0.02% and in Group C, it was 0.31±0.01% upto 5 mins after intubation. Thereafter, it showed an increasing trend in both the groups.From 5 minutes post insufflation, mean E_{tsevo} (0.48±0.20%) was higher in Group C as compared to Group D (0.33±0.11%). The difference was very highly significant upto about 30 minutes of CO_2 insufflation. There after mean E_{tsevo} (0.32±0.01%) was comparable in both the groups till the end of surgery.

Keywords: Dexmedetomidine, haemodynamic responses, elective laparoscopic cholecystectomy

Introduction

Laparoscopic Cholecystectomy (LC) is considered the gold standard for therapy of symptomatic gall stone disease.General anaesthesia (GA) is the usual technique under which the operation is carried out.Benefits of laparoscopy are minimal invasion, reduced acute phase responses, lesser postoperative pain and respiratory function alterations, early recovery and ambulation and shorter hospital stay[1].

Carbon dioxide (CO_2) is the commonest gas used for creating pneumoperitoneum (Pnp) during laparoscopic surgeries. CO_2 gets absorbed from the peritoneal surface into the

systemic circulation and gets eliminated from the lungs. In case, CO_2 retention occurs, it leads to hypercarbia. Excess of CO_2 produces systemic effects in two ways. By direct action, it causes peripheral vasodilation, myocardial depression, hypotension and bradycardia. Due to its indirect action, it leads to increased sympathoadrenal responses, tachycardia, tachyarrhythmia and hypertension.Net effect is variable, depending on patient, anaesthesia and surgical factors. Stretching of peritoneum and irritation by CO_2 stimulates a vagal

ISSN 2515-8260 Volume 08, Issue 04, 2021

response, further adding to bradycardia responses[2, 3].

High intra-abdominal pressure (IAP) created by CO_2 insufflation causes compression of Inferior Vena Cava (IVC), decrease in venous return, cardiac preload, cardiac output and cardiac index. This is compensated by sympathoadrenal response leading to vasoconstriction and increase in systemic vascular resistance. Elevation of diaphragm from increased IAP, leads to decrease in tidal volume, vital capacity and functional residual capacity, which also expose the patient to the risk of developing hypoxemia, besides hypercarbia[4].

Various drugs have been tried to obtund adverse CVS effects of CO_2 and Pnp e.g.; β-blockers, nitroglycerine, ephedrine, high dose opioids and inhalational agents, with variable success. Epidural analgesia has also been tried with variable success.

Dexmedetomidine (Dexmed) belongs to the imidazole subclass of α_2 receptor agonists, similar to clonidine. It is a potent and highly selective α_2 adrenoreceptor agonist, approved by FDA in 1999 for use in humans for analgesia and sedation.It has a greater affinity for α_2 receptors over α_1 (1620:1). Its α_2/α_1 selectivity ratio is eight times higher than that of clonidine. It possesses hypnotic, sedative, anxiolytic, sympatholytic and analgesic properties without significant respiratory depression. Due to these properties, it is used as a premedication to attenuate pressor response during intubation, supplement to GA/RA, and for sedation in ICU.It has rapid redistribution half-life of 6 minutes. Hence, Dexmed is given as a continuous infusion and its effect weans off within minutes after stopping. It undergoes biotransformation by conjugation (41%), n-methylation (21%) or hydroxylation in liver. Elimination half-life is approximately 2 hrs. Major problem with Dexmedetomidine are its significant haemodynamic effects such as severe bradycardia and hypotension. Other side effects are respiratory (depression, hypoxia, pleural effusion, pulmonary edema), GIT (nausea, vomiting, dryness of mouth), hematological (anemia, leukocytosis) and general (pain, thirst, fever).

Dexmed is not used routinely in anaesthesia practice because of its significant CVS effects like hypotension and bradycardia. But, during laparoscopic surgeries this effect itself may be beneficial in providing CVS stability following CO_2 Pnp. Anaesthetists have used Dexmed for laparoscopic surgeries in various dose regimens. There is no established dose and there are only a few reports quoting use and effects of Dexmed in laparoscopic cholecystectomy[5, 6].

Keeping this fact in mind, we decided to study the effect of Dexmed infusion on haemodynamic responses to CO_2 pneumoperitoneum in patients undergoing elective laparoscopic cholecystectomy under General Anaesthesia.

Methodology

All patients underwent a thorough preanaesthetic check-up. Written informed consent was obtained from each patient after explaining the procedure in a language that the patient understood. Patients were kept NPO from midnight and advised Tab. Diazepam (0.1-0.15 mg/kg) and Tab. Rantac 150 mg night before and at 6 AM on the morning of surgery with sips of water. In the OT, SBP, DBP, MAP (using NIBP), heart rate (HR), Oxygen saturation (SpO_2), electrocardiography (ECG) and End tidal CO_2 (E_tCO_2) were monitored, and baseline parameters recorded. Two intravenous (IV) lines were established with 18G (for IV fluids) and 20G (for drug infusion) cannulae on the dorsum of left and right hands, respectively. Patients were randomly allocated into 2 groups of 40 each.

Study group (Group D) received IV Dexmed infusion.
Control group (Group C) received NS infusion.

Technique of GA

General Anaesthesia (GA) technique was similar in all patients. All patients received Fentanyl (1 µg/kg) and Glycopyrolate (0.004µg/kg) IV, as premedication on OT table. Induction of anaesthesia was achieved with sleep dose of thiopentone (3-5 mg/kg) using

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100% O_2 via mask and Bains circuit. Tracheal intubation was done after paralysing with Inj. Rocuronium (0.5-0.6 mg/kg) and IPPV for 3 minutes. Anaesthesia was maintained with sevoflurane (0.4%) in O_2:N_2O mixture (1:2). Ventilation was controlled with a tidal volume of 6-8 ml/kg and at a rate of 12-14/min. (Respiration was adjusted to maintain $EtCO_2$ of 36-40 mmHg). Further doses of rocuronium, fentanyl and sevoflurane were given as per requirement to maintain BP and HR within 25% of baseline. At the end of surgery, muscle relaxant effect was reversed with Inj. neostigmine (0.05 mg/kg) and Inj. glycopyrolate (0.01 mg/kg) and trachea extubated. 100% O_2 was provided with mask for 2 mins after extubation. All patients received IV Ondansetron (0.1 mg/kg) and Diclofenac (1.5 mg/kg) at the end of surgery.

Group D

Dexmed infusion was started along with IV premedication in 20 G line at 0.4 µg/kg/hr (1 ml Inj. Dexmed (100 µg/ml) to 49 ml of NS in paediatric burette infusion set or infusion pump, to get 2µg/ml Dexmed e.g. 50 kg patient = 30 µg Dexmed/hr =15 ml/hr).

Group C

Normal saline was infused at similar rates (15 ml/hr for 50 kg patient).

IV fluids was given according to the calculated requirement and losses and continued throughout the surgery. Sevoflurane and Dexmed infusion were stopped at the end of Pnp.

Any change of >±25% in hemodynamics was treated with increasing or decreasing the concentration of Sevoflurane, additional muscle relaxants or Fentanyl. IV atropine (0.3 mg)was given if HR fell <60 beats/min.All patients were shifted to the postoperative ward for monitoring. Study period ended at the first requirement of analgesia in the post-operative period.

Results

Table 1: Systolic Blood Pressure (mmHg)

SBP (mmHg)	Group D		Group C		Intergroup (p value)
	Mean ± SD	p-value	Mean ± SD	p-value	
Baseline	125.43 ±10.88	-	119.50±9.37	-	0.005
Intubation	126.30±11.71	0.232	132.28±11.12	0.000	0.011
5 min Post Int.	118.63±13.29	0.004	118.15±7.52	0.143	0.422
Skin incision-0 min	122.75±16.12	0.147	124.00 ±9.34	0.020	0.336
CO_2 insufflation 5 min	124.08±18.60	0.319	144.60±14.57	0.000	0.000
10 min	123.58±18.61	0.269	143.23±16.64	0.000	0.000
15 min	123.60±18.14	0.281	142.90±12.27	0.000	0.000
20 min	125.68±14.88	0.460	139.68±10.47	0.000	0.000
25 min	125.03±15.39	0.434	134.60±7.28	0.000	0.000
30 min	123.28±12.81	0.165	131.90±5.11	0.000	0.000
40min	122.45±12.78	0.076	130.65±5.70	0.000	0.000
50 min	123.93±10.96	0.202	130.08±5.30	0.000	0.001
60 min	123.59±11.26	0.198	129.74±4.41	0.000	0.002
70 min	121.87±15.80	0.069	129.88±3.65	0.005	0.026
Post-Extubation 0 min	121.35±13.49	0.049	126.08± 8.49	0.001	0.032
15 min	121.98±13.90	0.068	119.55±8.09	0.490	0.172
30 min	124.05±12.94	0.270	118.45±8.75	0.275	0.013

The baseline mean SBP was 125.43±10.88 mmHg in Group D and 119.50 ± 9.37 mmHg in Group C which was comparable.

In Group D, there was no change in mean SBP at intubation. 5 minute post intubation a slight fall to 118.63 ± 13.29 mmHg was noticed which though was statistically significant as

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compared to baseline was not clinically significant. Thereafter the mean SBP remained between 118-125 mmHg throughout the surgery.

In Group C there was an increase in mean SBP at intubation to 132.28 ± 11.12 mmHg which was statistically significant. By 5 minutes of intubation, mean SBP returned to baseline value. At 5 mins after CO_2 insufflation mean SBP increased to 144.60 ± 14.57mmHgand it continued to remain so for about 20 minutes. Thereafter, there was a declining trend but it continued to remain high till the end of surgery. All these changes were statistically highly significant, but were not clinically significant.

On comparing the two groups, mean SBP was higher in Group C than in Group D all throughout the surgery and during the period of CO_2 pneumoperitoneum. This rise in Group C was statistically highly significant though clinically it was not significant.

Table 2: Diastolic Blood Pressure (mmHg)

DBP (mmHg)	Group D		Group C		Intergroup(p value)
	Mean SD	p-value	Mean SD	p-value	
Baseline	82.15±8.96	-	80.48 ± 6.44	-	0.170
Intubation	82.65± 10.07	0.308	85.25 ± 8.09	0.002	0.103
5 min Post Int	80.30 ± 12.99	0.154	78.30 ± 5.31	0.071	0.185
Skin incision-0 min	82.90 ± 11.03	0.339	82.85 ± 6.71	0.077	0.490
CO_2 insufflation-5 min	84.03 ± 13.66	0.183	93.13 ± 10.19	0.000	0.001
10 min	81.93 ± 12.18	0.460	91.78 ± 9.33	0.000	0.000
15 min	80.93 ± 11.40	0.292	92.80 ± 6.55	0.000	0.000
20 min	81.33 ± 10.99	0.342	90.18 ± 5.72	0.000	0.000
25 min	82.20 ± 10.92	0.491	88.43 ± 3.97	0.000	0.001
30 min	79.75 ± 10.08	0.118	89.48 ± 5.07	0.000	0.000
40min	80.55 ± 10.71	0.215	87.93 ± 6.22	0.000	0.000
50 min	80.00 ± 9.86	0.138	87.43 ± 4.63	0.000	0.000
60 min	80.46 ± 10.30	0.213	88.06 ± 5.41	0.000	0.000
70 min	84.00 ± 13.84	0.457	88.50 ± 4.44	0.000	0.107
Post extubation0 min	81.25 ± 10.29	0.320	85.50 ± 7.19	0.001	0.018
15 min	80.63 ± 11.08	0.221	80.55 ± 5.49	0.481	0.485
30 min	82.98 ± 10.00	0.340	78.95 ± 4.62	0.038	0.012

The mean baseline DBP was 82.15 ± 8.96 mmHg in Group D and 80.48 ± 6.44 mmHg in Group C respectively.

In Group D, mean DBP remained between 79.75 ± 10.08-84.03 ± 13.66 mmHg all throughout surgery, in all patients.

In Group C, there was a slight increase in mean DBP at intubation to 85.25 ± 8.09mmHg which came back to near baseline values by 5 mins. Another increase was noted at 5 mins of CO_2 insufflation to 93.13 ± 10.19 mmHg which continued for about 20 minutes. Thereafter, though there was a declining trend, it continued to remain high till the end of surgery. All

these changes though statistically significant were not clinically significant when compared to baseline.

On comparing, mean DBP was comparable in the two group's upto the time of skin incision. Rise in mean DBP at 5 minutes post CO_2 insufflation was significant in Group C than Group D. Though the difference was highly significant it was clinically not significant till the end.

Table 3: Mean Arterial Pressure (mmHg)

MAP (mmHg)	Group D		Group C		Intergroup (p value)
	Mean SD	p-value	Mean SD	p-value	
Baseline	96.58 ± 8.77	-	93.48 ± 6.80	-	0.041
Intubation	97.20 ± 9.99	0.230	100.93 ± 7.45	0.000	0.031
5 min Post Intubation	93.08 ± 12.53	0.039	91.58 ± 4.35	0.055	0.240
Skin incision- 0 min	96.18 ± 11.53	0.415	96.57 ± 6.91	0.032	0.429
CO_2 insufflation5 min	97.38 ± 14.65	0.355	110.28 ± 11.41	0.000	0.000
10 min	95.81 ± 13.65	0.372	108.93 ± 11.43	0.000	0.000
15 min	95.15 ± 13.04	0.279	109.50 ± 8.14	0.000	0.000
20 min	96.11 ± 11.71	0.411	106.68 ± 6.54	0.000	0.000
25 min	96.48 ± 11.64	0.481	103.82 ± 3.85	0.000	0.000
30 min	94.26 ± 10.34	0.110	103.62 ± 4.28	0.000	0.000
40min	94.52 ± 10.70	0.139	102.17 ± 5.12	0.000	0.000
50 min	94.64 ± 9.68	0.140	101.64 ± 4.07	0.000	0.000
60 min	94.84 ± 9.97	0.187	101.95 ± 4.46	0.000	0.000
70 min	96.62 ± 13.85	0.311	102.29 ± 3.75	0.000	0.059
Post Extubation0 min	94.62 ± 10.79	0.158	112.23 ± 7.50	0.000	0.000
15 min	94.41 ± 11.24	0.136	93.33 ± 5.81	0.460	0.295
30 min	96.67 ± 10.39	0.481	91.83 ± 4.57	0.034	0.004

The mean MAP in Group D and Group C was 96.58 ± 08.77mmHg and 93.48 ± 6.80 mmHg respectively.

In Group D, the mean MAP remained between 93.08 ± 12.53-97.20 ± 9.99 mmHg all throughout the surgery.

In Group C there was an increase in mean MAP at intubation to 100.93 ± 7.45 mmHg which returned to base line values by 5 minutes. At 5 minutes after CO_2 insufflation mean MAP was 110.28 ± 11.41 mmHg, thereafter the mean MAP continue to remain high till 70 minutes of surgery. Another peak in mean MAP at extubation to 112.23 ± 7.50 mmHg which returned to baseline by 15 minutes. All these changes in Group C though statistically significant from baseline were not clinically significant.

On comparing, at intubation the mean MAP in Group C was higher than Group D which was statistically significant but within normal clinical range. Thereafter mean MAP was comparable upto the time of skin incision. Rise in mean MAP at 5 mins post CO_2 insufflation was significant in Group C than Group D. Thereafter, all throughout the surgery mean MAP was higher in Group C than in Group D which was statistically significant but clinically not significant.

The trends in mean DBP and mean MAP were similar to mean SBP changes.

Table 4: Mean Heart Rate (beats per minute)

Heart rate (bpm)	Group D		Group C		Intergroup (p value)
	Mean SD	p-value	Mean SD	p-value	
Baseline	89.38 ± 16.81	-	86.65 ± 7.18	-	0.174
Intubation	83.83 ± 13.35	0.002	92.65 ± 8.87	0.000	0.000
5 min Post Int.	84.15 ± 15.40	0.027	89.63 ± 7.83	0.026	0.024

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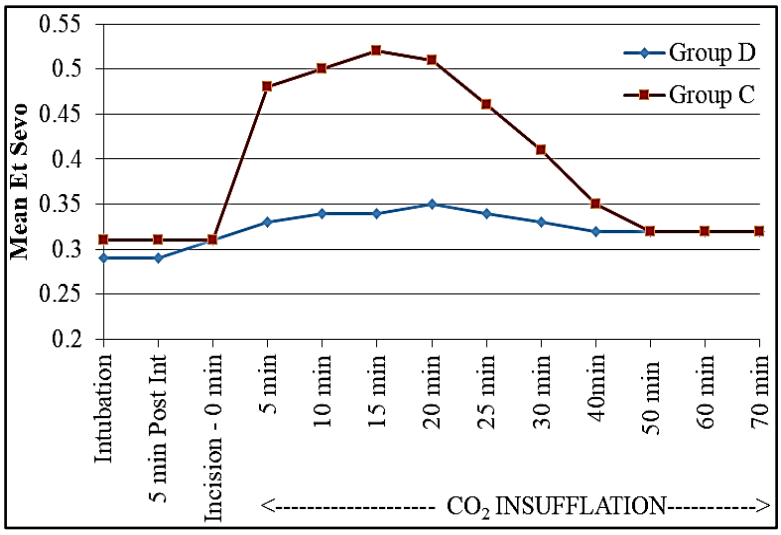
Skin incision-0 min	82.50 ± 12.97	0.004	92.05 ± 10.71	0.004	0.000
CO_2 insufflation 5 min	86.03 ± 18.89	0.128	104.28 ± 9.52	0.000	0.000
10 min	89.00 ± 23.47	0.459	102.85 ± 13.02	0.000	0.001
15 min	86.05 ± 20.10	0.155	101.60 ± 12.50	0.000	0.000
20 min	85.00 ± 16.95	0.063	98.55 ± 11.54	0.000	0.000
25 min	85.08 ± 13.38	0.054	93.05 ± 10.98	0.001	0.002
30 min	85.00 ± 12.83	0.052	92.30 ± 9.36	0.000	0.002
40min	83.83 ± 12.32	0.019	89.25 ± 8.84	0.043	0.013
50 min	82.85 ± 10.17	0.007	89.60 ± 8.97	0.026	0.001
60 min	82.67 ± 10.30	0.006	88.97 ± 10.59	0.188	0.006
70 min	82.97 ± 11.89	0.017	88.38 ± 8.24	0.288	0.056
Post extubation 0 min	84.10 ± 12.78	0.032	90.80 ± 9.36	0.003	0.005
15 min	84.75 ± 12.26	0.044	86.03 ± 6.21	0.328	0.280
30 min	82.85 ± 12.83	0.009	85.35 ± 4.36	0.130	0.123

The mean baseline heart rate in Group D and Group C were 89.38 ± 16.81 bpm and 86.65 ± 7.18 bpm respectively.

In Group D, there was a decrease in mean HR at intubation and skin incision. Though the decrease in HR was statistically significant, it was not clinically significant when compared to baseline value. Thereafter the mean HR remained between 82.50 ± 12.97-89.38 ± 16.81 bpm throughout the surgery.

In Group C there was increase in mean HR at intubation and another rise at 5 minutes post CO_2 insufflation and remained high till 30 minutes of surgery. Though all these changes were highly significant statistically clinically not significant. Thereafter the mean HR remained between 86.03 ± 6.21-90.80 ± 9.36 bpm throughout surgery.

On comparing, the mean baseline HR was comparable in both groups. At intubation there was slight increase in HR in Group C which returned to baseline after 5 minutes. Another peak in mean HR was noted 5 minutes post CO_2 insufflation in Group C which remained high till end of surgery. Though the difference was significant statistically, it was clinically insignificant.



Graph 1: E_{tsevo}

The mean E_{tsevo} in Group D was 0.29 ± 0.02% and in Group C, it was 0.31 ± 0.01% upto 5 mins after intubation. Thereafter, it showed an increasing trend in both the groups.From 5 minutes post insufflation, mean E_{tsevo} (0.48 ± 0.20%) was higher in Group C as compared to Group D (0.33 ± 0.11%). The difference was very highly significant upto about 30 minutes of CO_2 insufflation. There after mean E_{tsevo} (0.32 ± 0.01%) was comparable in both the groups till the end of surgery.

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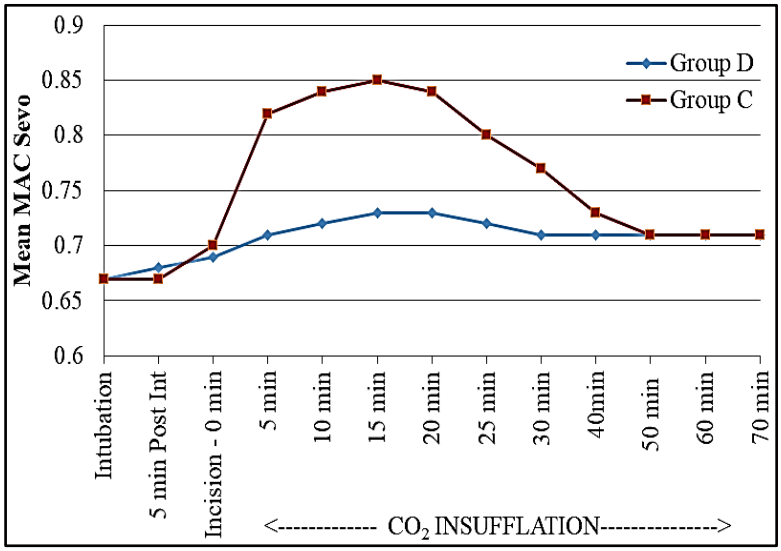

The mean MAC_{sevo} in Group D was 0.67 ± 0.02% and in Group C, it was 0.67 ± 0.01% was similar and comparable till 5 mins post intubation.Post CO_2 insufflation, mean MAC_{sevo} (0.82 ± 0.16%) was higher in Group C as compared to Group D (0.21 ± 0.08%).The difference was very highly significant upto about 40 minutes of CO_2 insufflation. Thereafter mean MAC_{sevo} (0.71 ± 0.01%) was comparable in both the groups.

Discussion

The mean baseline heart rate in Group D was (89.38 ± 16.81 bpm) and in Group C (86.65 ± 7.18 bpm) and was comparable. Baseline HR was >100/min in 11 patients in Group D and in 2 patients in Group C.

In our study, in Group D, there was decrease in mean HR at intubation and at the time of skin incision. Thereafter mean HR returned to near baseline values and maintained throughout the intraoperative period. One patient in group D, had a HR of 52/min at the time of intubation. No intervention was required and HR returned to baseline value within minutes. In Group C, there was an increase in mean HR at intubation which continued to be high till 30 minutes of CO_2 insufflation. Thereafter mean HR returned to near baseline value. Another increase in mean HR was noted at the time of extubation in this group. Though all these changes were statistically significant, mean HR remained within normal clinical values all throughout in Group D whereas the changes in control group were highly significant as compared to the baseline.

During laparoscopic surgeries patients are prone for cardiac rate and rhythm abnormalities. All types of arrhythmias have been reported. Patients may develop tachycardia and tachyarrhythmias, due to the indirect effects of hypercarbia and sympathoadrenal stimulation. Bradycardia and bradyarrhythmias secondary to vagal stimulation have been reported in literature in patients undergoing laparoscopic cholecystectomy. There have been reports of even sudden cardiovascular collapse and death after the creation of pneumoperitoneum.

In our study, 8 patients in the control group required additional fentanyl, in 3 patients at the time of skin incision, in 1 patient at 5 min after Pnp and in 4 patients at 10 mins after creation of Pnp.

The mean baseline SBP in Group D (125 ± 10.88 mmHg) was higher when compared to Group C (119.50 ± 9.37 mmHg), but was within clinical range.There were 5 patients who were hypertensives (ASA II) on medication and well controlled. This may be the reason for high mean baseline SBP in group D.

In our study, there were no significant changes in mean SBP in Dexmed Group (D) at

intubation, skin incision, all throughout intra operative period, and at extubation. Whereas in the Control Group (C), there was an increase in mean SBP at intubation which was highly significant, statistically.Another rise in mean SBP was seen 5 minutes post CO_2 insufflation, which continued to remain so, throughout the intraoperative period till extubation. These changes in mean SBP in Group C were highly significant when compared to Group D.In the recovery room, mean SBP was comparable in the two groups.
Mean DBP in Group D (82.15 ± 8.96 mmHg) and in Group C (80.48 ± 6.44 mmHg) was comparable and within normal range in the two groups.
Mean MAP in Group D (96.58 ± 8.77 mmHg) and in Group C (93.48 ± 6.80 mmHg) was also comparable.

The trends in mean DBP and mean MAP were similar to mean SBP trends

In 2007, Bakhameese*t al.*, studiedthe effects of dexmedetomidine onanaesthetic requirements during surgery on haemodynamics, recovery profile and morphine requirement in the post-operative period, in 80 morbidly obese (ASA II and ASAII)patients undergoing laparoscopic gastric by pass surgery. They hypothesised that obese patients are more sensitive to respiratory depressant effect of anaesthetics and narcotics. They were randomly assigned to either of the two groups. All patients were premedicated with IV midazolam (0.3 mg) and glycopyrolate (0.2 mg),and dexmedetomidine (0.8 µg/kg) IV over 10 mins as loading dose followed by 0.4 µg/kg/hr as maintainance in the study group.Other group received normal saline.Anaesthesia was induced with fentanyl (0.5 µg/kg), lidocaine (100 mg), Propofol 1-2 mg/kg, Cisatracurium (0.2 mg/kg) for intubation, and ventilation with mixture of 50% air in oxygen. PEEP of 5-10 cmH_2O to maintain normocapnia ($EtCO_2$ 35-45 mmHg) was applied. Intraoperatively propofol infusion (10 mg/kg/hr) was started and adjusted to maintain BIS level between 40-60. Additional doses of fentanyl (0.5 µg/kg) were given for changes in blood pressure or heart rate. They noted significant decreases in MAP and HR in the dexmedetomidine group as compared to the placebo group, and decrease in the requirement of propofol in the Dexmed Group.They concluded that, dexmedetomidine offered better control of intraoperative and postoperative haemodynamic parameters[7].
Basar H *et al.*, in 2008 carried out a study in 40 patients undergoing elective LC to evaluate the effects of single dose of dexmedetomedine on induction, haemodynamic, and other cardiovascular parameters (EDI, CI, SVI, EF) using noninvasive thoracic electrical bio-impedence. All patients were premedicated with atropine IM 45 mins before anaesthesia. Study group received dexmedetomidine (0.5 µg/kg) over 10 mins and other group received normal saline. Induction was with sleep dose of thiopentone (BIS monitoring) and intubation with vecuronium. Anaesthesia was maintained with $O2:N_2O$ (50:50) and Desflurane. No opioids were used. They found significant decrease in HR in Dexmed group (90/min to 70/min) after intubation, and significant increase in mean MAP in the control group. They also observed significant decrease in MAP and CI at the end of infusion in dexmedetomidine group. Recovery profile was assessed using Modified Aldrete scores. They concluded that a single dose of dexmedetomidine (0.5 µg/kg),given before induction over a period of 10 minutes,did not cause any major haemodynamic changes at intubation or during the intraoperative periods andrather blunted the haemodynamic responses to intubation, with no change in recovery profile[8].
In 2008 itself, Tufanogullari*et al.*, carried out a study on 80 morbidly obese adult patients, undergoing laparoscopic bariatric surgery. All patients received oral celecoxib 400 mg orally,30-60 min prior to induction.They studied the effect of different doses of dexmedetomidine on early and late recovery profiles using VRS scores.Patients were divided into 4 groups according to the infusion they receieved (NS, Dexmed 0.2, Dexmed 0.4 and Dexmed 0.8 µg/kg hr). All patients recieved premedication with IV midazolam 20 µg/kg.Induction of anaesthesia was done with Propofol and intubation using Rocuronium.

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Intraoperative monitoring consisted of NIBP, ECG, E_tCO_2, S_pO_2, cerebral state monitor. They found significantly low BP and HR in Dexmed group intraoperativelyand in the postoperative period.Bradycardia (HR<45/min) was treated with glycopyrolate 0.2 mg IV bolus. Tachycardia (HR> 25% of baseline values) was treated with IV boluses of 5 mg labetolol.In their study, 5 patients in the control group and 3 patients in dexmed 0.2 group required rescue beta blocker for tachycardia. There was no incidence of bradycardia in any patient. Persistent hypotension was more with higher doses of Dexmed (0.8 µg/kg/hr). Persistent and significant hypotension was treated with IV phenylephrine 100 µg. 10 patients in dexmed 0.8 group required rescue phenylephrine. They recommended an infusion rate of 0.2 µg/kg/hr of Dexmed, as anaesthetic adjuvant to facilitate early recovery while minimizing adverse perioperative cardiovascular side effects[9].

In 2010, Dhurjoti Prasad *et al*., conducted a study in 60 patients undergoing LC to evaluate the efficacy of dexmedetomidine to provide haemodynamic stability. They were randomly assigned to two groups, either Dexmed (0.2 µg/kg/hr infusion) or 0.9% saline groups. After 5 minutes of infusion, anaesthesia was induced with fentanyl 1 µg/kg, Propofol 2 mg/kg (20 mg every 5 sec) and Rocuronium (0.6 mg/kg) for intubation. Anesthesia was maintained with O_2 in N_2O, intermittent boluses of fentanyl (0.5 µg/kg) and Rocuronium. IAP was maintained at 14 mmHg. Patients were ventilated to maintain E_tCO_2 between 35-40 mmHg. For introperative hypertension, NTG infusion was started.HR and MAP were noted at regular intervals. They found thatHR and MAP weresignificantly low in Dexmed group after induction (67.3 ± 9.6 vs 79.4 ± 11.5/min, 60 ± 7.8 vs 94 ± 12.4 mmHg),after intubation (75.1 ± 11 vs 97.2 ± 12.7/min, 87.5 ± 10.1 vs 119.2 ± 16.23 mmHg), and after PnP (73.8 ± 9.3 vs 96 ± 14.8/min, 94 ± 11.6 vs 130 ± 18.39 mmHg) as compared to saline group. HR and MAP remained on lower side throughout Pnp and even in the postoperative period. In their study, in 3 patients in the Dexmed group HR was < 50/ min, but none of the patients required any intervention. None of the patients in their study required NTG infusion. They concluded that dexmedetomidine reduces the elevation of MAP and HR during and after Pnp and thereby helps to maintain perioperative haemodynamic stability during laparoscopic surgery even in low doses[10].

Conclusion

Thus, we conclude that General Anaesthesia with low dose dexmeditomidine infusionis a better technique for laparoscopic cholecystectomy than General Anaesthesia alone in normal healthy patients (ASA I and II). From our study, we can extrapolate that General Anaesthesia with dexmedetomidine may be a better anaesthetic technique than General Anaesthesia alone, in high risk patients (ASA III and IV) undergoing laparoscopic cholecystectomy, where haemodynamic stability and early recovery are prime concerns, though more work needs to be done.

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