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The effect of remifentanil and sufentanil in TCI mode on airway pressures during laparascopic gastric banding procedures in patients with adiposity based chronic disease

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Abstract

Introduction: **Wooden chest syndrome or opioid induced thorax wall rigidity, presents a serious challenge during anesthesia in patients with adiposity-based chronic disease. This study aims to investigate the impact of remifentanil TCI and sufentanil TCI on airway pressures during laparoscopic gastric banding in obese patients.**
Methods: **During the first analysis of the original data De Baerdemaeker L. et al 1 , a statistical significant difference in mean peak airway pressure between remifentanil TCI and sufentanil TCI was observed. This study was a post-hoc analysis with detailed analysis of airway pressures at defined timepoints. Thirty-six patients were randomly allocated to receive remifentanil TCI (Minto Model) or sufentanil TCI (Gepts model) during laparoscopic gastric banding surgery. 18 patients were allocated to each group. Two patients in each group were excluded due to missing data. All patients received propofol, rocuronium 0.9 mg.kg-1 ideal body weight during induction, anesthesia was maintained using BIS guided desflurane. Airway pressures were measured before (T1), during (T2), and after pneumoperitoneum (T3). Independent samples t-test was used for statistical analysis between groups and paired t-test for within group analysis.**
Results: **No statistically significant difference in peak - and plateau ventilation pressures was observed between the remifentanil and sufentanil groups throughout the surgical procedure. Both opioids exhibited similar effects on airway pressures before (T1), during (T2) and after pneumoperitoneum (T3).**
Discussion and Conclusion: There is no significant advantage in selecting remifentanil over sufentanil in terms of ventilation pressures during laparoscopic gastric banding. The concurrent administration of opioids with muscle relaxants may mitigate the risk of opioid-induced rigidity, aligning with previous findings. Further research is warranted to elucidate optimal strategies for airway management in obese patients undergoing laparoscopic procedures.

Keywords: **Sufentanil, remifentanil, respiratory failure, wooden chest syndrome, fentanyl-induced chest wall rigidity, opioids.**

Introduction

Thorax wall rigidity, commonly known as "wooden chest syndrome," is a rare but serious complication associated with opioid use. This condition can occur even at low doses of opioids and has been reported with various opioids including fentanyl, sufentanil, and remifentanil, among others. Typically, it manifests within 1-2 minutes following opioid administration and may persist for 8-15 minutes. The onset of wooden chest syndrome is characterized by severe rigidity in the respiratory muscles of the chest wall, diaphragm, and upper airway, often leading to laryngospasm. This rigidity poses challenges for bag-mask ventilation and intubation, thereby compromising airway management. If not promptly and effectively managed, it can result in severe hypoxia and hypercarbia, ultimately leading to fatal outcomes. Notably, wooden chest syndrome has been documented in both clinical settings and instances of illicit opioid abuse. When occurring outside of a hospital environment, it is particularly perilous and frequently fatal. Thus, heightened

awareness and effective management strategies are imperative for mitigating the risks associated with this syndrome[1-4].

In various settings, diagnosing this syndrome can be challenging, as its presentation often resembles that of acute respiratory distress syndrome (ARDS) or laryngospasm. Treatment typically involves swift airway management, including intubation and ventilation, along with muscle relaxation. Additionally, discontinuation of opioids and administration of naloxone are essential components of therapy[5,6].

However, there is a documented case report where wooden chest syndrome occurred following administration of naloxone. This counter intuitive finding might be explained by the possible involvement of α1-adrenergic in the pathophysiology of wooden chest syndrome[7,8].

Several studies indicate that the respiratory muscle rigidity induced by fentanyl is mediated by the activation of mu-opioid receptors in the locus coeruleus. This activation leads to increased noradrenergic outflow from the locus coeruleus by stimulating α1-adrenergic receptors in both the locus coeruleus and spinal cord[8-10].

Various risk factors predispose individuals to the development of wooden chest syndrome. These include extremes of age (such as newborns or the elderly), critical illness, underlying neurologic or metabolic disorders, and the use of dopaminergic medications[11]. Notably, infants and neonates are particularly vulnerable, as evidenced by several documented case reports of wooden chest syndrome in this population[6,12-14]. Remarkably, there is even a case report detailing neonatal wooden chest syndrome following the administration of remifentanil to the mother during a cesarean section[12].

The true incidence of wooden chest syndrome remains elusive due to underreporting and misdiagnosis of this complication. Furthermore, there exists a significant disparity in the literature owing to variations in dosing regimens and the use of different opioids.

A study by Streisand et al. previously reported a noteworthy incidence of peripheral muscle rigidity, occurring in 50% of patients administered high-dose fentanyl monoanesthesia at 15 mcg/kg without the use of muscle relaxation. However, it's important to note that this study focused specifically on peripheral muscle rigidity and did not specifically address wooden chest syndrome. Importantly, none of the 12 patients in this study developed thorax wall rigidity necessitating ventilation[15].

Most reports detailing wooden chest syndrome occurrences often do not involve the use of muscle relaxation. A study conducted by Nakada J. et al. demonstrated that administering a muscle relaxant during anesthesia induction effectively prevents the onset of wooden chest syndrome[16].

It's worth noting that Choong et al. reported a higher incidence of chest rigidity with the use of remifentanil (3 mcg/kg) without curare compared to fentanyl (2 mcg/kg) combined with succinylcholine (20 mcg/kg)[13]. However, their findings did not reach statistical significance. These observations suggest a potential protective effect of curare administration against chest wall rigidity.

Additionally, a recent study by Oh YJ et al. also suggests a protective effect of administering a hypnotic agent before an opioid during anesthesia[17].

Wooden chest syndrome seems to manifest even with low doses of fentanyl, sufentanil, and remifentanil[2]. Evidence suggests a dose-response relationship in the incidence of wooden chest syndrome[18-20].

Very few studies tried to compare the occurrence of wooden chest syndrome between opioids.

Zhao et al. compared propofol, propofol + sufentanil and propofol + remifentanil. Significantly more patients developed thorax wall rigidity in the propofol + remifentanil group compared to the sufentanil + propofol group or propofol alone[9]. This suggests that remifentanil could be more prone to produce chest wall rigidity when compared to sufentanil[9].

The aim of this RCT is to look for differences in airway pressures between equipotent doses sufentanil and remifentanil in patients with obesity undergoing laparoscopic gastric banding.

Methods

After Institutional Ethics Committee (Ghent University Hospital, Ghent, Belgium) approval, written informed consent was obtained from morbidly obese patients (BMI 35 kg/m²), aged 18 – 70 years of age (ASA I – II status), under- going laparoscopic gastric banding. Exclusion criteria included diagnosed obstructive sleep apnoea syndrome, re-do surgery, history of drug abuse, use of β-blockers, significant cardiopulmonary disease, renal failure (serum creatinine 120 mmol/L), abnormal liver enzymes (transaminases 1.5 times normal values), or history of allergy to anaesthetics. All patients were operated on by the same team of surgeons, using the same surgical technique (Swedish Adjustable Gastric Band, Obtech Medical, Baar, Switzerland). Four 10 mm trochars and one 5 mm trochar were placed on a line 10 cm parallel to the costal border bilaterally

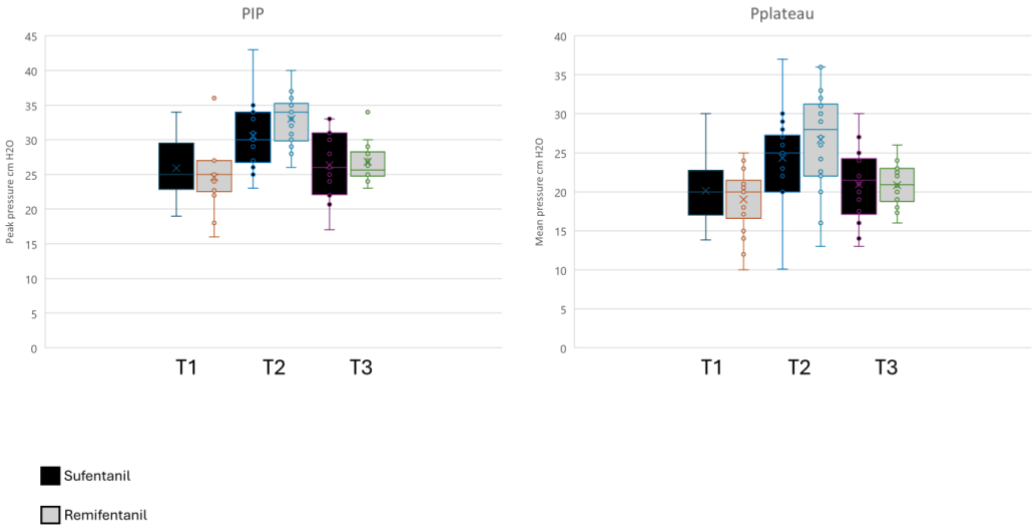

Fig. 1 — Boxplot of Peak inspiratory pressure (PIP) and Plateau pressure (Pplateau) in cmH2O. T1 = Pressure before insufflation, T2 = Pressure after insufflation, T3= Pressure after desufflation.

on the mid-clavicular line and anterior axillary line and paramedian of the umbilicus. None of the surgical sites was infiltrated with local anaesthetics. In all patients, CO2 insufflation was initiated at 20 cmH2O and afterwards decreased to 15– 17 cmH2O. Spirometry was standardized with each patient in a 30° head-up position.

One hour before surgery, all patients received ranitidine 150 mg p.o. Midazolam (2 mg) was given i.v. before placement of a catheter in the left radial artery, approximately 10 min before induction. Patients were pre-oxygenated by mask for 5 min in the supine position with oxygen 10 L/min.

Heart rate (HR), invasive arterial pressure, SpO2 , capnography, spirometry, inspiratory, and end-tidal anaesthetic drug concentrations were measured continuously using an S5 monitor (Datex-Ohmeda, Helsinki, Finland). BIS index(version 4.0) was derived from the frontal EEG (At-Fpzt) and calculated by the A-2000 BIS Monitor using a BIS-XP Sensor (Aspect Medical Systems, Inc., Newton, MA, USA). The smoothening time of the BIS monitor was set at 15 s. All data were continuously recorded, using the RUGLOOP data manager.

Patients were randomly allocated to one of the two groups. In the remifentanil group (Group R), the remifentanil infusion was started 2.5 min before induction via a computer-assisted continuous infusion device (RUGLOOP II, Demed, Temse, Belgium) to an initial target plasma concentration of 4 ng/ml using a three-compartment model based on the Minto model[21].

In the sufentanil group (Group S), the sufentanil infusion was started 2.5 min before induction via a computer-assisted continuous infusion device (RUGLOOPII, Demed) to an initial target effect-site concentration of 0.2 ng/ml using a three-compartment model based on the Gepts model[22].

Anaesthesia was induced with a bolus of propofol, administered at 300 ml/h until loss of consciousness (LOC). At LOC, rocuronium 0.9 mg/kg of ideal body weight (IBW) was administered while applying cricoid pressure. The trachea was intubated 60 s later. The lungs were ventilated with a mixture of oxygen/air (FiO2= 50%) using an ADU ventilator (GE Healthcare, Helsinki, Finland).

Tidal volume was set at 10 ml/kg IBW for volume controlled ventilation with 5 – 8 cmH2O PEEP. Respiratory frequency was adjusted to achieve an end-tidal CO 2 pressure of 4.0– 4.6 kPa. If required, FI O2 was adjusted to maintain oxygen saturation above 95%. After tracheal intubation, all patients received a prophylactic antibiotic dose of cefazoline 2 g i.v., propacetamol 4g, and diclofenac 150 mg.

All patients received desflurane. Initial fresh gas flow (FGF) was 6 litre.min-1 with the vaporizer set at 6 vol% (desflurane). After 2.5 min, the FGF was lowered to 2 litre min-1 and the FD desflurane was targeted to maintain a BIS value between 45 and 55. If the BIS value was <45 for >30 s, the FD desflurane was decreased by 25%. If BIS values exceeded 55 for >30 s, an 'inhalation bolus of desflurane' was administered[3]. The remifentanil and sufentanil administration was adjusted according to haemodynamic measurements. A baseline arterial pressure and HR were taken 5 min after tracheal intubation. Inadequate analgesia was defined as: rise in systolic arterial pressure (SAP) >15 mm Hg above baseline, HR > 90 beats/min in the absence of hypovolaemia, autonomic signs (e.g. sweating, salivation, and flushing) and somatic signs (e.g. movement, swallowing). If any of the above

were present, the opioid target concentration was increased by 25%. A level of excessive analgesia was defined as: mean arterial pressure (MAP) below 60 mm Hg or HR below 50 beats/min. In this case, the opioid target concentration was decreased by 25%. After each change in infusion rate, there was a lockout period of 2.5 min. If requested by the surgeon, an additional bolus of rocuronium (25% of the initial dose) was given. If more than three consecutive adjustments were needed to bring arterial pressure or heartbeat within limits, i.v. rescue medication was used: urapidil 12.5 mg, phenylephrine 0.1 mg, or atropine 0.5 mg as appropriate. For surgery, all patients were positioned in the semi- recumbent position after having received a crystalloid loading dose 10 ml.kg-1 IBW. In case of persistent hypotension in the sitting position, a bolus of phenylephrine, 0.1 mg i.v., was given rather than changing the opioid dosage. Sufentanil administration was stopped at the moment of exsufflation of the pneumoperitoneum. Remifentanil in Group R and desflurane in both groups were stopped at completion of dressing. Residual muscle relaxation was assessed by double burst stimulation and reversed with atropine 10 mcg/kg and neostigmine 35 mcg/kg. After stopping all drug delivery, FGF was set at 6 L/ min with an FIO2 of 50%. Two minutes after the drug discontinuation, ventilation was stopped and manual breathing support was installed (one breath every 15 s until return of spontaneous ventilation. If EtCO2 became higher than 60 mm Hg, manual-breathing support was increased until EtCO2 was below 50 mm Hg). The anaesthesia time was defined as the time period between LOC and the moment of drug discontinuation.

We measured airway pressures before pneumoperitoneum (T1), during pneumoperitoneum (T2) and after pneumoperitoneum (T3).

Power analysis was based on the previous work done by Cadi P. and al[23]. We considered a mean difference of a plateau pressure of 3,0 cmH2O to be clinically relevant, with a standard deviation of 3,1 cmH2O. To achieve a power of 80% and an alpha risk of 5% and the power of the study at 80%; at least 18 patients were required in each group to detect a difference .

This study was performed as an extra analysis on the dataset of the study performed by De Baerdemaeker L. et al.[24]. In the original study 20 patients were allocated to the sufentanil group (S) and 20 patients were allocated to the remifentanil group (R). However due to faulty airway registration 4 patients, 2 in the sufentanil and 2 in the remifentanil group, had to be excluded from the dataset. For the statistical analysis we used SPSS v. 29 software from IBM.

For all data sets, Gaussian distribution was tested using the Kolomogorov – Smirnov test. Between groups, continuous data were analysed using independent samples t-test or Mann– Whitney test, where appropriate. Categorical data were analysed using Fisher's exact test.

Within groups, statistics were done using repeated measures ANOVA statistics. Significance level was set at 0,05 unless otherwise reported. Significance is reached when zero is not included in the 95% CI.

Results

We enrolled 18 patients in each group, both receiving either sufentanil or remifentanil.

Table I shows comparable patient characteristics, anesthesia depth, and rocuronium dosage between the remifentanil and sufentanil group.

Our main findings revealed no statistically significant difference in peak and mean ventilation pressures (in cmH2O) between the two groups. Specifically, the mean peak inspiratory pressures (PIP) before insufflation were similar in both groups and did not reach statistical significance. The mean PIP before insufflation was 25.9 cmH2O (95% CI:

Table I. — Patient characteristics: Mean, range and standard deviation (SD).

	Sufentanil (n=18)	Remifentanil (n=18)	P value
Height (cm)	169 (156-190) SD: 3,4,	166 (153-183) SD: 8,0	0,28
Weight (kg)	115 (96-164) SD: 16,2,	117 (92 – 180) SD: 24	0,51
IBW (kg)	61 (40-97) SD: 9,5,	62 (48-80) SD: 7,5	0,29
BMI	39,9 (35 - 48) SD: 3,4,	40,3 (35 - 47) SD: 4,1	0,75
Age (years)	37,7 (21- 56) SD: 9,7,	37,5 (20 - 56) SD: 7,5	0,28
Men/woman ratio	2/16	3/15	0,65
Number of smokers	6/18	5/18	1
Number of ASA II patients	5/18	4/18	1
Rocuronium dose (mg)	66 (50 - 95) SD:11	60 (54 - 80) SD:7	0,06
Rocuronium dose (mg/kg IBW)	1,05 (0,87 -1,53) SD: 0,16	0,98 (0,88 – 1,24) SD: 0,1	0,18
Desflurane highest Et concentration (%)	6,29 (4,7 – 7,8) SD: 0,86	6,25 (4,0 – 8,3) SD: 1,1	0,9
Highest MAC	1,05 (0,78 – 1,3) SD: 0,14	1,01 (0,6 – 1,4) SD: 0,2	0,57
IBW: Ideal body weight, BMI: body mass index, MAC: minimum alveolar concentration.			

Table II. — Ventilation pressures in cmH2O before, during and after pneumoperitoneum.

		Min	Max	Mean + 95% CI	Std. Deviation
Sufenta	PIP T1	19,0	34,0	25,9 (23,7 - 28,1)	4,4200
	Pplat T1	13,8	30,0	20,1 (18,0 - 22,1)	4,1496
	PIP T2	23,0	43,0	30,5 (28,1 - 32,8)	4,7231
	Pplat T2	10,0	37,0	24,3 (21,5 - 27,1)	5,6256
	PIP T3	17,0	33,0	26,3 (23,9 - 28,7)	4,7671
	Pplat T3	13,0	30,0	20,9 (18,7 - 23,3)	4,5454
Remi	PIP T1	16,0	36,0	24,6 (22,3 - 26,8)	4,1854
	Pplat T1	10,0	25,0	19,0 (16,8 - 21,2)	4,1594
	PIP T2	26,0	40,0	33,0 (31,1 – 34,0)	3,5629
	Pplat T2	13,0	36,0	26,6 (24,1 – 30,4)	6,4810
	PIP T3	23,0	34,0	26,8 (25,2 – 28,6)	3,2184
	Pplat T3	16,0	26,0	20,8 (19,6 – 22,3)	2,5649

T1 = Pressure before insufflation, T2 = Pressure after insufflation, T3= Pressure after desufflation. PIP = Peak inspiratory pressure, Pplateau = plateau pressure. Min = minimum, Max = maximum.

23.7 - 28.1) in the sufentanil group vs. 24.6 cmH2O (95% CI: 22.3 - 26.8) in the remifentanil group, with a difference of 1.3 cmH2O (P = 0.37). The plateau pressures (Pplat) before insufflation were also similar between both groups. The mean Pplat before insufflation was 20.1 cmH2O (95% CI: 18.0 - 22.1) in the sufentanil group vs. 19.0 cmH2O (95% CI: 16.8 - 21.2) in the remifentanil group, with a difference of 1.1 cmH2O (P = 0.42).

During insufflation to achieve pneumoperitoneum, both PIP and Pplat increased in both groups. However, the choice of opioid did not significantly impact the rise in ventilatory pressures. Specifically, the mean PIP after insufflation was 30.5 cmH2O (95% CI: 28.1 - 32.8) in the sufentanil group vs. 33.0 cmH2O (95% CI: 31.1 – 34.0) in the remifentanil group, with a difference of -2.48 cmH2O (P = 0.08). The mean Pplat after insufflation was 24.3 cmH2O (95% CI: 21.5 - 27.1) in the sufentanil group vs. 26.6 (95% CI: 24.1 – 30.4) in the remifentanil group, with a difference of -2.31 cmH2O (P= 0.26).

After desufflation to end the pneumoperitoneum, both PIP and Pplat decreased to similar levels as before the pneumoperitoneum. Once again, the choice of drug did not significantly affect ventilation pressures. Specifically, the mean PIP after desufflation was 26.3 (95% CI: 23.9 - 28.7) in the sufentanil group vs. 26.8 (95% CI: 25.2 – 28.6) in the remifentanil group, with a difference of -0.51 cmH2O (P = 0.91). The mean Pplat after desufflation was 20.9 (95% CI: 18.7 - 23.3) in the sufentanil group vs. 20.8 (95% CI: 19.6 – 22.3) in the remifentanil group, with a difference of 0.13 cmH2O (P = 0.45).

Within-group analysis showed that pneumoperitoneum (T2) significantly raised PIP and Pplat compared to before (T1) and after (T3) pneumoperitoneum in both the sufentanil and remifentanil groups, with specific changes detailed in Table III.

The average peak inspiratory pressure (PIP) in the sufentanil group exhibited a notable increase of 4.6 cmH2O ($P < 0.01$) during pneumoperitoneum (T2) in comparison to pre-pneumoperitoneum levels (T1), followed by a significant decrease of 4.1 cmH2O ($P < 0.01$) post-desufflation (T3). Similarly, the mean plateau pressure (Pplat) in the sufentanil group showed a significant rise of 4.2 cmH2O ($P < 0.03$) during pneumoperitoneum (T2) compared to pre-insufflation (T1), followed by a decrease of 3.3 cmH2O ($P < 0.05$) post-desufflation (T3).

The average PIP in the remifentanil group saw a more marked increase of 8.3 cmH2O ($P < 0.01$) during pneumoperitoneum (T2) compared to pre-insufflation (T1), followed by a significant decrease of 6.1 cmH2O ($P < 0.01$) post-desufflation (T3). Similarly, the mean Pplat in the remifentanil group exhibited a substantial rise of 7.6 cmH2O ($P < 0.01$) during pneumoperitoneum (T2) in comparison to pre-pneumoperitoneum levels (T1), followed by a decrease of 5.8 cmH2O ($P < 0.01$) post-desufflation (T3).

Furthermore, in the entire population, both the absolute weight of the patient and BMI showed a positive but weak correlation with ventilation pressures, with means and confidence intervals presented in Tables IV and V.

Discussion and conclusion

In conclusion, our study did not find any statistically significant difference in ventilation pressures between the groups receiving sufentanil and

Table III. — Ventilation pressures in cmH2O before, during and after pneumoperitoneum.

	Opioid	Mean pressure in cmH20 + 95% CI	Mean difference	P-value
PIP T1	Sufentanil	25,9 (23,7 - 28,1)	- 1,3	N.S. (0,37)
	Remifentanil	24,6 (22,3 - 26,8)		
Pplat T1	Sufentanil	20,1 (18,0 - 22,1)	- 1,1	N.S. (0,42)
	Remifentanil	19,0 (16,8 - 21,2)		
PIP T2	Sufentanil	30,5 (28,1 - 32,8)	+ 2,5	N.S. (0,08)
	Remifentanil	33,0 (31,1 – 34,0)		
Pplat T2	Sufentanil	24,3 (21,5 - 27,1)	+ 2,3	N.S. (0,26)
	Remifentanil	26,6 (24,1 – 30,4)		
PIP T3	Sufentanil	26,3 (23,9 - 28,7)	+ 0,5	N.S. (0,91)
	Remifentanil	26,8 (25,2 – 28,6)		
Pplat T3	Sufentanil	20,9 (18,7 - 23,3)	- 0,1	N.S. (0,45)
	Remifentanil	20,8 (19,6 – 22,3)		

T1 = Pressure before insufflation, T2 = Pressure after insufflation, T3= Pressure after desufflation. PIP = Peak inspiratory pressure, Pplateau = plateau pressure. N.S. = not significant.

Table IV. — Pearson correlation of BMI and respiratory pressures.

BMI	PIP before insufflation	Pplat before insufflation	PIP after insufflation	Pplat after insufflation	PIP after desufflation	Pplat after dessuflation
Pearson correlation	0,360	0,228	0,338	0,299	0,281	0,390
Significance 2-tailed	0,034	0,093	0,047	0,082	0,102	0,021

Table V. — Pearson correlation of weight and respiratory pressures.

Weight	PIP before insufflation	Pplat before insufflation	PIP after insufflation	Pplat after insufflation	PIP after desufflation	Pplat after dessuflation
Pearson correlation	0,528	0,370	0,378	0,372	0,2	0,289
Significance 2-tailed	<,001	0,026	0,023	0,026	0,242	0,088

remifentanil during laparoscopic surgery. This lack of significant difference may be attributed to the use of rocuronium as a muscle relaxant, potentially masking the appearance of opioid-induced muscle rigidity. Additionally, the equipotent doses of opioids administered at the effect site may have resulted in similar levels of airway rigidity between the groups. Further research could be done using opioid free anesthesia to confirm if using opioids or not has any effect while using muscle relaxation. Since we performed a rapid sequence induction in all patients with a dose of 1mg/kg IBW, we have no spirometric data of the spontaneous breathing patient or mask ventilation before the administration of the rocuronium. Wooden chest syndrome mostly occurs in patients not receiving NMBA. All patients received midazolam, which is known to reduce the incidence of wooden chest syndrome[25]. Since we did not use neuromuscular transmission monitoring, we have no data on the presence and the quality of the neuromuscular blockade at the moments of registration of the airway pressures.

Desflurane is recognized for its potential to induce bronchoconstriction, particularly at higher minimum alveolar concentration (MAC) values and especially in smokers[26-27]. However, in our study, we did not observe bronchoconstriction, likely attributable to the utilization of lower MAC values of desflurane.

Comparisons with previous studies suggest that the ventilation pressures recorded in our study are consistent with those observed in obese patients undergoing laparoscopic gastric banding, regardless of ventilation mode[23,28]. One of these has been performed in our center to look at differences between volume - (VCV) and pressure controlled ventilation (PCV) and found no difference between the ventilation modes on ventilatory mechanics. The Ppeak and Pplateau in that study were similar to our study during all the phases of surgery: Ppeak of 22 cmH2O and Pplateau of 18 cmH2O before initiation of pneumoperitoneum and a Ppeak of 29 cmH2O with a Pplateau of 26 cmH2O after initiating pneumoperitoneum for both volume and pressure controlled ventilation[28].

The study of Cadi. P. Et al in also had similar results on ventilatory mechanics in patients undergoing laparoscopic gastric banding in both PCV and VCV ventilation. During pneumoperitoneum their study showed a Pplateau in VCV of 27 cmH2O and of 26 cmH2O in the PCV group[23].

Lastly we did not offer a dose response effect of opioids, because the timing of the highest dosage of both remifentanil and sufentanil always

coincided with the start of the pneumoperitoneum which created a significant confounding factor for analyzing ventilatory mechanics during pneumoperitoneum[23,28].

Despite the limitations of our study, our findings indicate that there is no definitive advantage in selecting one opioid over another concerning airway pressures. Additionally, our observation that the concurrent administration of opioids with rocuronium used for rapid sequence induction seemed to mitigate the occurrence of wooden chest syndrome is noteworthy. This finding aligns with the conclusions drawn by Nakada et al.[16], further bolstering the evidence in support of this phenomenon.

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