



Changes in Lung Function Parameters after Total Intravenous Anaesthesia and Balanced Anaesthesia with Desflurane: A Prospective Randomised Study

Saurabh Sharma¹ , Anju Romina Bhalotra² , Shikha Awal³ 

¹Department of Neuroanaesthesia, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India

²Department of Anaesthesiology, Maulana Azad Medical College and associated Lok Nayak Hospital, New Delhi, India

³Department of Anaesthesiology, Kalinga Hospital, Bhubaneswar, Orissa, India

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Abstract

Objective: Following anaesthesia, there is a decrease in pulmonary function. Unlike volatile anaesthetics, propofol decreases the upper airway tone, and total intravenous anaesthesia (TIVA) with propofol may decrease coughing on emergence. Coughing may reduce postoperative atelectasis. Thus, TIVA may lead to greater decreases in lung function postoperatively as compared to balanced anaesthesia with desflurane.

Methods: Sixty patients of either sex, aged 18-60 years and American Society of Anaesthesiologists (ASA) status I/II, who were to undergo mastoid surgery, were randomly allocated to Group B and Group T. Anaesthesia was maintained with desflurane, nitrous oxide and oxygen in Group B, and with TIVA in Group T. Pulmonary function tests (PFT) were done preoperatively, and 1, 3 and 24 hours postoperatively.

Results: Demographic data and preoperative PFT were comparable in both groups. One hour after surgery, there was a greater decrease in FEV1 and peak expiratory flow rate (PEFR) in Group T ($p=0.044$ and 0.042 , respectively). Three hours postoperatively, the decrease in MEFR and PEFR was again greater in Group T ($p=0.005$ and 0.008 , respectively), while the MEFR recovered to preoperative values in Group B. By 24 hours, the forced vital capacity (FVC), MEFR and PEFR recovered to preoperative values in Group T, while FVC remained reduced in Group B ($p=0.006$).

Conclusion: Both anaesthetic techniques cause a postoperative impairment in the lung function, but while TIVA causes a greater reduction in PFT in the early postoperative period, recovery is also earlier. On the other hand, balanced anaesthesia with desflurane was associated with a greater reduction in PFT at 24 hours.

Keywords: Anaesthesia, anaesthetic techniques, general, inhalation, intravenous, monitoring, spirometry

Introduction

Postoperative respiratory complications are common, and they may increase morbidity and mortality. A decrease in respiratory parameters in the immediate postoperative period has been found after both general and regional anaesthesia (1). The choice of anaesthetic may influence the degree of postoperative lung dysfunction. Balanced anaesthesia with halogenated anaesthetics is perhaps the most popular general anaesthesia technique. Inhalational anaesthetic agents are effective, reliable, safe, easy to deliver, stable, and without major end organ sequelae. Recently, interest in total intravenous anaesthesia (TIVA) has increased due to an improved quality of emergence from anaesthesia, reduced postoperative nausea and vomiting, rapid onset of action independent of alveolar ventilation, easy usage in peripheral locations, and elimination of occupational exposure to inhalational agents. However, TIVA requires the use of comparatively expensive drugs and infusion pumps, and there is an increased risk of awareness during the procedure (2).

A significantly higher incidence of coughing has been reported in patients awakening from balanced anaesthesia with halogenated anaesthetics as compared to TIVA. While coughing, patients are required to take a vital capacity breath, and this may be beneficial in reducing postoperative atelectasis (3). On the other hand, unlike volatile anaesthetics, propofol decreases the tone of the upper airway (3). We thus hypothesised that the smoother recovery from TIVA may be associated with increased postoperative pulmonary dysfunction. Desflurane is a pungent airway irritant and may induce more coughing on emergence than the other inhalation agents. Also, desflurane and propofol have the most favourable pharmacokinetic profiles for rapid recovery from inhaled and intravenous anaesthesia, respectively (4). There is a paucity of studies evaluating the effects of desflurane on postoperative lung dysfunction. The aim of this study was to investigate whether balanced anaesthesia using desflurane and TIVA with propofol have differing effects on postoperative impairment of respiratory function.

Methods

Ethical approval was provided by the hospital ethical committee. This randomised comparative study was conducted from March 2013 to March 2014 on 60 adult patients of either sex, aged 18-60 years, with the ASA physical status I/II, were admitted for mastoid surgery under general anaesthesia and agreed to perform pulmonary function tests (PFTs). The change between pre- and postoperative forced expiratory volume in 1 second (FEV_1) was used to predict the sample size. In a previous investigation, Tiefenthaler et al. (2) found that a sample size of 27 in each group will have an 80% power to detect a difference in means of -0.820 in the two groups, assuming that the common standard deviation (SD) is 1.040 using a two-group t-test with 0.05 two-sided significance level. We recruited 60 patients, assuming a drop-out rate of 10%. Written informed consent was obtained from all patients. Patients who were obese and had a history of smoking, cardiopulmonary disease, obstructive sleep apnoea, kyphoscoliosis and neuromuscular disorders were excluded from the study.

A detailed pre-anaesthetic check-up with spirometry and pulse oximetry was done prior to surgery. No sedative pre-medication was given to the patients prior to performing baseline spirometry. On the morning of surgery, patients were randomly allocated to one of two study groups: Group B (balanced anaesthesia with oxygen, nitrous oxide and desflurane) or Group T (TIVA with propofol). Randomisation was by computer-generated numbers, and allocation into groups was conducted by means of opening a sealed opaque envelope immediately before surgery. Patients were blinded to their group allocation. On arrival in the operation theatre, intravenous

access and standard noninvasive monitoring was instituted consisting of electrocardiography, end tidal concentration of volatile anaesthetics, capnography, pulse oximetry, noninvasive blood pressure and neuromuscular junction monitoring. All patients were premedicated with midazolam 1 mg and fentanyl $2 \mu\text{g kg}^{-1}$ 5 minutes before induction of anaesthesia. In both the groups, general anaesthesia was induced with propofol $1.5-3 \text{ mg kg}^{-1}$, and muscle relaxation was achieved with rocuronium 0.6 mg kg^{-1} . In Group B, the lungs were ventilated with 50% nitrous oxide in oxygen and 4% desflurane for 3 minutes, after which endotracheal intubation was done. Anaesthesia was subsequently maintained with 50% nitrous oxide in oxygen and desflurane 3-6 vol %, intermittent rocuronium and fentanyl $1 \mu\text{g kg}^{-1}$ repeated every hour. At the completion of surgery, desflurane was discontinued. In Group T, intravenous induction was with propofol $1.5-3 \text{ mg kg}^{-1}$ and a propofol infusion was commenced at $150 \mu\text{g kg}^{-1} \text{ min}^{-1}$. After administration of rocuronium 0.6 mg kg^{-1} , the lungs were ventilated with an air/oxygen mixture, 2:1 for 3 minutes, after which tracheal intubation was done and anaesthesia maintained with intravenous infusions of propofol $100-150 \mu\text{g kg}^{-1} \text{ min}^{-1}$ and fentanyl $1 \mu\text{g kg}^{-1} \text{ hr}^{-1}$ and intermittent rocuronium. Propofol and fentanyl infusions were stopped 10 minutes before the end of surgery.

In both groups, endotracheal intubation was done under direct laryngoscopic vision using an endotracheal tube with a high-volume low-pressure cuff. A 7.0 mm and 8.0 mm internal diameter endotracheal tube was used for women and men, respectively. The cuff was inflated with air, and cuff pressure monitored and maintained at 20-25 mmHg. In both groups, fresh gas flow was 6.0 L min^{-1} during the first 10 minutes and then adjusted to 1.0 L min^{-1} with oxygen flow to maintain an FIO_2 of 0.5. Controlled ventilation was adjusted to maintain an end tidal carbon dioxide concentration ($EtCO_2$) of 35-45 mmHg. All patients received diclofenac 1.5 mg kg^{-1} and paracetamol 1 gm toward the end of surgery for postoperative analgesia and ondansetron 0.1 mg kg^{-1} . Neuromuscular blockade was reversed with neostigmine and glycopyrrolate, and the extubation of the trachea was done at an inspired oxygen concentration of 80% once the patient was awake and after ensuring a train-of-four ratio $\geq 90\%$. Any episode of bronchospasm, laryngospasm or oxygen desaturation, that is, peripheral oxygen saturation (SpO_2) $< 90\%$, was noted.

The patients were then transferred to the post anaesthesia care unit (PACU) breathing room air. Each patient was nursed in a 30° head-up tilt position in the PACU, and SpO_2 was measured continuously by pulse oximetry. Oxygen by face mask was supplemented if the SpO_2 fell under 92%. In the PACU, pain was treated with morphine boluses 1.5 mg given at 5-min intervals to achieve adequate analgesia (defined as a visual analogue score ≤ 3).

Pre- and postoperative spirometry was performed by the same anaesthesiologist trained to use the spirometer, who was not aware of the study hypothesis and was not otherwise involved in the study. All of the pulse oximetry and spirometry data were recorded by a blinded investigator after the patient was inhaling air for at least 5 minutes while in a 30° head-up position. Preoperative spirometry was done during the preoperative visit and values were recorded after demonstration of the manoeuvres to be performed. After surgery, postoperative spirometry was done 1 hour, 3 hours and 24 hours after shifting the patient to the PACU. The parameters recorded on spirometry included FEV₁, forced vital capacity (FVC), the FEV₁-to-FVC ratio, peak expiratory flow rate (PEFR) and maximal (mid) expiratory flow rate (MEFR) using a bedside portable spirometer Autospiro-AS 500 (Minato Medical). At each assessment time, spirometry was performed at least

three times, and the best measurement was recorded. Before each assessment, it was ensured that the patient was warm and pain free so that there was no pain, shivering etc., which could interfere with the patients ability to breathe deeply and the patient could perform spirometry in a reliable manner.

Statistical analysis

The data were collected and analysed using the Statistical Package for the Social Sciences and STATA (STATA 15, StataCorps) statistical software. The change between pre- and postoperative FEV₁ was used to predict the sample size. The secondary outcome variables include SpO₂, FEV₁, FEV₁/FVC ratio, PEFR and MEFR and were described as the mean±SD. For comparison of mean/median within the group, that is, preoperative and postoperative, a paired t-test or the Wilcoxon sign rank test was used. For comparison between both the groups, unpaired t-test or Mann-Whitney U test was used. A p-value <0.05 was considered statistically significant.

Results

A total of 60 patients were included in the study. There were no dropouts. Both groups were similar with regard to demographic data, that is, age, sex, height, weight, body mass index and duration of surgery (Table 1). The preoperative SpO₂ was similar in both the groups. There was a significant decrease in SpO₂ levels postoperatively at all times in both groups (p<0.001). However, there was no statistically significant difference in SpO₂ values between the two groups at any time in the postoperative period. No patient had a decrease in SpO₂ <92% requiring oxygen therapy. The values of SpO₂ remained significantly lower than preoperative values even at 24 hours after surgery with either anaesthesia technique (Table 2).

The preoperative FEV₁ and FVC were similar in Groups B and Group T. There was a statistically significant decrease in FEV₁ in both groups at 1, 3 and 24 hours after surgery as compared to the preoperative values (p<0.001). The decrease in FEV₁ as compared to preoperative values at 1 hr postoperatively was significantly greater in Group T when compared to Group B (p=0.044). However, there was no significant difference between the two groups at 3 and 24 hours postoperatively, and the values of FEV₁ remained significantly lower than preoperative values, even at 24 hours after surgery with either type of anaesthesia (Table 3).

There was a statistically significant decrease in FVC in both the groups 1 and 3 hours after surgery when compared to preoperative values (p<0.001). This decrease was similar in both Group B and Group T. However, the FVC recovered to near preoperative values after 24 hours in TIVA group, but not in the balanced anaesthesia group (Table 4). The decrease in FVC from the preoperative values remained significantly

Table 1. Patient characteristics

Patient characteristics	Group B	Group T	p
Age (years) mean±SD	22.13±6.77	25.17±9.05	0.192
Height (cm) mean±SD	153.93±7.33	156.73±7.88	0.570
Weight (kg) mean±SD	51.47±7.03	52.37±6.48	0.718
BMI (kg m ⁻²) mean±SD	21.65±1.78	21.26±1.85	0.825
Number of male patients	18 (51.4%)	17 (48.6%)	1.00
Duration of surgery (hrs) mean±SD	3.20±0.40	3.38±0.48	0.13

The patient characteristics were similar in both the groups. BMI: body mass index; SD: standard deviation

Table 2. Values of peripheral oxygen saturation pre- and postoperatively

Time	SpO ₂ %		p
	Group B (mean±SD)	Group T (mean±SD)	
Preoperative	98.3±1.05	98.0±0.983	0.259
After 1 hr	96.43±1.45	96.13±0.937	0.346
p	<0.001	<0.001	
Change at 1 hr from preoperative value	-1.87±1.67	-1.87±0.73	0.724
After 3 hrs	96.53±1.27	96.57±1.40	0.924
p	<0.001	<0.001	
Change at 3 hrs from preoperative value	-1.77±1.55	-1.43±1.10	0.510
After 24 hrs	97.40±0.72	97.10±0.923	0.167
p	<0.001	<0.001	
Change at 24 hrs from preoperative value	-0.90±0.76	-0.90±0.66	0.857

There was a significant difference in SpO₂ levels pre- and postoperatively at all times in both groups. There was no significant difference between the two groups at any time. SpO₂: peripheral oxygen saturation; SD: standard deviation

greater in Group B 24 hours after surgery (p=0.006). The FEV₁-to-FVC ratio preoperatively and at 1, 3 and 24 hours after surgery was similar in Group B and Group T.

The values of MEFR and PEFR preoperatively and at 1, 3 and 24 hours after surgery were comparable in both groups. There was a significant and similar decrease of MEFR at 1 hour post-

operatively in both the groups as compared to the preoperative values. However, while this recovered to near preoperative values after 3 hours in Group B, it remained significantly reduced in Group T (p<0.001). This drop from preoperative values at 3 hours was significantly higher in Group T when compared to Group B (p=0.005). The MEFR subsequently returned to preoperative values at 24 hours in Group T (Table 5).

Table 3. Values of FEV₁ pre- and postoperatively

Time	FEV ₁ (litres)		p
	Group B (mean±SD)	Group T (mean±SD)	
Preoperative	2.30±0.53	2.45±0.57	0.306
After 1 hr	1.98±0.56	2.04±0.81	0.941
p	<0.001	<0.001	
Change at 1 hr from preoperative value	-0.33±0.29	-0.41±0.70	0.044
After 3 hrs	2.03±0.59	2.01±0.55	0.890
p	<0.001	<0.001	
Change at 3 hrs from preoperative value	-0.27±0.34	-0.44±0.40	0.178
After 24 hrs	2.15±0.59	2.38±0.56	0.124
p	<0.001	<0.001	
Change at 24 hrs from preoperative value	-0.16±0.23	-0.07±0.16	0.122

There was a significant decrease in FEV₁ values postoperatively in both groups. The drop in preoperative values at 1 hr was significantly greater in Group T as compared to Group B. FEV₁: forced expiratory volume in the first second; SD: standard deviation

Table 4. Values of FVC pre- and postoperatively

Time	FVC (litres)		p
	Group B (mean±SD)	Group T (mean±SD)	
Preoperative	2.50±0.63	2.61±0.60	0.491
After 1 hr	2.07±0.58	2.05±0.52	0.880
p	<0.001	<0.001	
Change at 1 hr from preoperative value	-0.43±0.37	-0.56±0.37	0.114
After 3 hrs	2.11±0.62	2.09±0.57	0.886
p	<0.001	<0.001	
Change at 3 hrs from preoperative value	-0.39±0.38	-0.52±0.42	0.147
After 24 hrs	2.24±0.63	2.52±0.60	0.085
p	<0.001	0.055	
Change at 24 hrs from preoperative value	-0.26±0.35	-0.09±0.24	0.006

There was a significant decrease in FVC at 1 and 3 hours postoperatively which was similar in both groups. As compared to preoperative values, the drop in FVC at 24 hours after surgery was greater in Group B. FVC: forced vital capacity; SD: standard deviation

Table 5. Values of MEFR pre- and postoperatively

Time	MEFR (litres/sec)		p
	Group B (mean±SD)	Group T (mean±SD)	
Preoperative	3.28±0.99	3.27±0.88	0.767
After 1 hr	2.99±1.10	2.67±0.52	0.83
p	0.01	<0.001	
Change at 1 hr from preoperative value	-0.29±0.64	-0.59±0.72	0.087
After 3 hrs	3.28±1.24	2.68±0.84	0.085
p	0.74	<0.001	
Change at 3 hrs from preoperative value	-0.00±0.94	-0.58±0.70	0.005
After 24 hrs	3.23±1.08	3.20±0.93	0.929
p	0.54	0.058	
Change at 24 hrs from preoperative value	-0.06±0.62	-0.06±0.22	0.888

There was a significant decrease in the MEFR postoperatively in both the groups at 1 hour. The MEFR recovered to preoperative values by 3 hours in Group B and by 24 hours in Group T. MEFR: mid expiratory flow rate; SD: standard deviation

Table 6. Values of PEFR pre and postoperatively

Time	PEFR (litres/sec)		p
	Group B (mean±SD)	Group T (mean±SD)	
Preoperative	318.17±97.48	347.17±88.55	0.077
After 1 hr	267.80±102.81	256.53±83.93	0.819
p	<0.001	<0.001	
Change at 1 hr from preoperative value	-50.37±59.04	-90.63±71.34	0.042
After 3 hrs	284.20±105.29	255.60±73.58	0.438
p	0.01	<0.001	
Change at 3 hrs from preoperative value	-33.97±69.03	-91.57±68.70	0.008
After 24 hrs	302.73±97.52	340.97±92.19	0.078
p	0.04	0.056	
Change at 24 hrs from preoperative value	-15.43±57.87	-6.2±23.00	0.230

There was a significant decrease in PEFR in both groups at 1 and 3 hours postoperatively, and this decrease was significantly higher in Group T at both these times. The PEFR recovered to baseline values after 24 hours in Group T but remained reduced in Group B. PEFR: peak expiratory flow rate; SD: standard deviation

There was a significant drop in PEFR in both groups at 1 and 3 hours postoperatively, and this decrease was significantly higher in Group T at both the times ($p=0.042$ and $p=0.008$, respectively). However, the PEFR recovered to baseline values after 24 hours in Group T but remained reduced in Group B ($p=0.04$). The decrease from preoperative values was however similar in both groups at 24 hours (Table 6).

Discussion

In this study, there was a similar and consistent drop in the respiratory function postoperatively in all patients receiving either balanced anaesthesia with desflurane or TIVA with propofol. The SpO_2 and FEV_1 remained reduced in both groups at 1, 3 and 24 hours after surgery compared to preoperative values, and there was no difference between the two groups with respect to the drop in SpO_2 . In the early hours after surgery, the decrease in lung parameters was significantly greater in Group T compared to Group B. There was a significantly greater drop in FEV_1 and PEFR from preoperative values at 1 hour in Group T ($p=0.044$ and $p=0.042$ respectively). Similarly, at 3 hours after surgery the decrease in MEFR and PEFR from preoperative values was also greater in Group T ($p=0.005$ and $p=0.008$ respectively). However, the recovery of lung function tests was faster in Group T and by 24 hours, the values of FVC, MEFR and PEFR had returned to preoperative values in Group T. On the other hand, in Group B, a significant decrease in FVC as compared to preoperative values was recorded at 24 hours ($p=0.006$). Only the MEFR had returned to preoperative values by 24 hours in Group B, while all other parameters remained reduced.

The decrease in lung function with an unchanged FEV_1/FVC ratio observed in both groups indicates a restrictive disturbance in lung function, as already described in previous investigations (5, 6). The type of anaesthesia did not seem to have a significant effect on postoperative pulmonary dysfunction. The development of a restrictive lung dysfunction after general anaesthesia is a constant and reproducible finding which was observed as early as 1966 by Diamant and Palmer (7). Rothen et al. (8) demonstrated perioperative atelectasis using computed tomography and multiple inert gas elimination technique postoperatively and suggested that the development of atelectasis after induction of general anaesthesia is the cause of this restriction. The degree of atelectasis determines the reduction in values of FVC or FEV_1 (9). It is still not clear when the lung function after surgery returns to normal and may be influenced by several factors such as the type of anaesthesia, and the site and magnitude of the surgical intervention (10). In this study, the SpO_2 and FEV_1 remained significantly reduced even at 24 hours postoperatively in both groups.

Von Ungern-Sternberg et al. (1) reported a drop in pulmonary function parameters after both general and regional anaesthesia which was lesser after spinal anaesthesia when compared to general anaesthesia. Also, the extent of pulmonary dysfunction has been found to be greater after abdominal when compared to peripheral surgery (5). We chose patients undergoing mastoid surgery as the study population to eliminate any contribution of pain from an abdominal incision to the decrease in lung function parameters. Also, prior to performing postoperative spirometry, pain was ensured to be minimal by providing adequate analgesia.

It has been suggested that there is a significantly higher incidence of coughing in patients emerging from balanced anaesthesia with sevoflurane than after emerging from TIVA. Coughing has been likened to a vital capacity manoeuvre (inflating the lungs to 40 cm H_2O for 15 secs), and it has been shown to be effective in reducing the incidence of postoperative atelectasis (11). We did not notice any significant coughing in either group. This may be partially attributed to the use of adequate doses of opioids for analgesia. Also, in Group B, desflurane concentration was limited to ≤ 1.0 MAC. Jensen and colleagues, using computed tomography and arterial blood gas analysis postoperatively reported that there was no difference in the incidence of postoperative atelectasis or oxygenation when using propofol or isoflurane anaesthesia (12). They did not use spirometry to support their findings. The same findings were suggested using spirometry in our study on comparing propofol and desflurane. Dikmen et al. (13), investigating the effects of desflurane, isoflurane and sevoflurane on bronchial smooth muscle tone reported that desflurane, like isoflurane and sevoflurane, exhibits a bronchodilator effect at 1 MAC concentration. However, increasing the concentration to 2 MAC causes an increase in airway resistance with desflurane, while sevoflurane and isoflurane continue to have a bronchodilator effect. Evaluation of the effects of propofol on respiratory mechanics in rats revealed that propofol decreases respiratory and lung impedances as a result of central airway dilatation (14). Thus, it is not likely that either agent will cause obstructive lung dysfunction postoperatively if desflurane concentration is limited to less than 2 MAC as was done in our study. There was no drop in the FEV_1 -to-FVC ratio in our patients.

Numerous factors contribute to the restrictive lung defect, which is known to develop after general anaesthesia. These include anaesthesia-related reductions in pulmonary compliance, reduced functional residual capacity in the supine position and pain induced restriction of diaphragmatic movement as well as the choice of anaesthetic. Volatile agents are thought to be protective as they exert a bronchodilatory action through various mechanisms such as a decrease in the intracellular calcium concentration and a reduction in calcium

sensitivity. They also increase the baseline pulmonary dynamic compliance and attenuate increases in pulmonary airway resistance caused by chemical or mechanical stimuli (15).

Zoremba et al. (4) evaluated the influence of propofol versus desflurane anaesthesia in overweight patients undergoing minor peripheral surgery on postoperative lung function and pulse oximetry values. They found that within the first 2 hours after surgery, the propofol group displayed a lower oxyhaemoglobin saturation ($p < 0.007$) and lung function ($p < 0.001$) compared to the desflurane group, and even 24 hours after surgery, FEV₁, PEFR, MEFR, forced inspiratory VC and PIFR were reduced more in the propofol group (all $p < 0.01$). They found that increasing obesity decreases pulmonary function at 2 hours after propofol anaesthesia but not after desflurane anaesthesia (4). As the postoperative reduction in spirometric volumes has been found to be more significant in the obese patient, we confined our study population to patients with BMI ≤ 30 kg m⁻² (5).

Tiefenthaler and colleagues reported that in patients emerging from general anaesthesia, the postoperative reduction in FVC is greater after TIVA than after balanced anaesthesia with sevoflurane (2). In a recent meta-analysis, Uhliq and colleagues found that volatile anaesthetics were associated with less postoperative pulmonary complications as compared to TIVA in patients who underwent cardiac surgical procedures ($p = 0.038$). However, in noncardiac surgery, volatile anaesthetics were not associated with a lower incidence of postoperative pulmonary complications ($p = 0.081$), and when compared to TIVA, none of the volatile anaesthetics reduced pulmonary complications (15). In the present study, it was observed that both balanced general anaesthesia with desflurane and TIVA with propofol produced a decline in lung function and SpO₂ postoperatively. The recovery of FVC and PEFR was faster in the TIVA group, while the MEFR recovery was quicker in the balanced anaesthesia group. It appears that both anaesthesia techniques cause impairment of lung function postoperatively, but while TIVA seems to cause a greater reduction in PFT in the early postoperative period, the recovery is also earlier. Thus, there seems to be no reason to suggest that TIVA with propofol, if properly titrated, has detrimental effects on the postoperative lung function due to the suppression of upper airway reflexes and lack of coughing on termination of general anaesthesia.

However, our study has some limitations. It was conducted on non-obese patients and those without pre-existing pulmonary dysfunction so the results of this study cannot be extrapolated to obese patients and those with pre-existing pulmonary dysfunction. Postoperative ultrasonography or computed tomography was not done to study the extent of atelectasis in either group, and the SpO₂ analysis was used as a surrogate marker for the degree of postoperative atelectasis. Further studies are required

to be conducted on patients who are at higher risk of developing postoperative pulmonary complications where the choice of the anaesthetic technique may assume a greater importance.

Conclusion

Both general anaesthesia using desflurane and TIVA with propofol led to a decrease in the lung function and SpO₂ postoperatively. While lung function parameters like FEV₁, MEFR and PEFR were significantly reduced in the early postoperative period, the recovery of FVC and PEFR was also faster in the TIVA group. Only the MEFR recovery was quicker in the balanced anaesthesia group, while all other parameters remained significantly depressed. According to the present study, TIVA with propofol was not found to have detrimental effects on the lung function postoperatively as compared to a balanced anaesthesia technique with desflurane.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Maulana Azad Medical College (CTRI/2017/12/ 010910).

Informed Consent: Written informed consent was obtained from all patients who participated in this study.

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