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ORIGINAL RESEARCH

Evaluating the efficacy of propofol in attenuating the cardiorespiratory response to extubation: single-blinded randomised placebo-controlled trial

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Background: Extubation at the end of general anaesthesia (GA) should be performed in a way that ensures patient comfort and minimises cardiorespiratory changes to prevent harm. Several drugs have been shown to attenuate these changes during emergence. This study aimed to investigate if a sub-hypnotic dose of propofol can produce such favourable peri-extubation conditions.

Methods: A total of 50 American Society of Anesthesia (ASA) physical status I–II patients (aged 18–70) undergoing elective abdominal or pelvic surgery under GA with a volatile agent were randomly assigned to a propofol group (n = 28) or a control group (n = 22). At the end of the surgery, once the minimal alveolar concentration reached 0.6, patients received either propofol 0.5 mg kg^{-1} or an equivalent volume of 0.9% normal saline intravenously (IV). The primary outcome was the incidence and severity of bucking and coughing observed during emergence, with the assessment performed by a blinded anaesthetist. Haemodynamic parameters, airway responses, extubation complications, and time to extubation were evaluated during the emergence period at predetermined intervals.

Results: The demographic and clinical characteristics of the two groups were comparable before surgery. Results indicated the incidence and severity of bucking at extubation were significantly lower in the propofol group (21.4%) compared to the control group (68.2%, $p < 0.001$). Similarly, patients in the propofol group had significantly fewer heart rate (HR) ($p = 0.031$) and systolic blood pressure (BP) ($p = 0.031$) changes at extubation.

Conclusion: The addition of propofol 0.5 mg kg^{-1} before extubation successfully attenuated cardiorespiratory responses following GA in ASA Grade I–II adult patients undergoing elective abdominal or pelvic surgery, but did not reduce the overall incidence of cough at extubation.

Keywords: low-dose propofol, blunt extubation, extubation cough, extubation response

Introduction

Tracheal extubation represents a critical point at the end of general anaesthesia (GA). The process is associated with transient physiological changes that trigger a range of cardiovascular and respiratory responses.[1,2] While the physiological changes are well tolerated by most patients, they can be harmful. Adverse cardiovascular outcomes include cardiac arrhythmias, tachycardia, hypertensive as well as hypotensive periods, myocardial ischaemia, and a prolonged increase in myocardial oxygen consumption.[1,2] Adverse respiratory sequelae include coughing, bucking, laryngospasm, bronchospasm, apnoea, and desaturation.[2,3] Avoiding adverse cardiorespiratory responses at extubation is a key concern for anaesthetists. Furthermore, procedures involving microsurgery require a smooth emergence without coughing or straining.[3] Antitussive strategies at extubation have become particularly relevant in the era of the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) due to its aerosol generation and the associated risk to healthcare professionals.[4]

A variety of technical methods and pharmacological agents have been shown to facilitate smooth extubation with minimal cardiovascular and respiratory responses. Some of the more frequently studied intravenous (IV) agents that have demonstrated effectiveness in attenuating cardiorespiratory responses at extubation include lignocaine, alpha-2 adrenergic agonists, calcium channel blockers, beta blockers, and opioids.[5,6] Non-pharmacological strategies include removing the tracheal tube (TT) while the patient is in a deep plane of anaesthesia, performing Bailey's manoeuvre, and performing a "no-touch" technique.[7]

More recently, the use of propofol (2,6-diisopropylphenol) to minimise cardiorespiratory responses and facilitate smooth emergence following extubation has been studied.[8-17] Propofol is listed on the World Health Organization (WHO) essential drugs list and is readily available in operating theatres in many countries; relatively inexpensive compared to newer anaesthetic drugs, and there are few contraindications to its use.[18] Sub-anaesthetic doses of propofol are known to induce sedative and anxiolytic effects in a dose-dependent manner, while its anti-emetic properties reduce the incidence of nausea and vomiting after GA.[18] Its administration is associated with the blunting of airway reflexes, allowing for its use as an effective treatment for laryngospasm at extubation.[10-12] Results from several studies have shown that a sub-hypnotic dose of propofol significantly

reduced cardiorespiratory changes in patients following extubation compared to control groups.[9-13]

However, these findings have not been universal, with other studies using similar protocols finding either no significant effect or showing non-superior effects when compared with other interventions.[8,14,16]

Considering the high incidence of coughing at extubation with potentially serious consequences at the time of TT manipulation, this study aimed to investigate the efficacy of a sub-hypnotic dose of propofol in attenuating the cardiorespiratory response to extubation.[1-3] We hypothesised that the incidence and severity of bucking and coughing would be significantly lower in adult patients who received a low dose of propofol IV (0.5 mg kg^{-1}) compared to those who received a placebo during emergence from TT extubation following GA. Haemodynamic parameters, airway complications, and time to extubation were also evaluated during emergence.

Methods

Ethical approval

Ethical approval was obtained from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand prior to commencement (ethics number: M210232 obtained on 22 April 2021; National Health Research Database study number: GP_202101_034). Informed written consent was obtained from each participant before participating in the study. Patient confidentiality was maintained throughout the study.

Study design and participants

A prospective, randomised, placebo-controlled, parallel-group trial with single blinding was conducted. Adult patients (aged 18–70) who presented for elective abdominal or pelvic surgery under GA at the Chris Hani Baragwanath Academic Hospital in South Africa from April to October 2021 were invited to participate in the study. Patients were required to have an ASA physical status I–II. Patients with contraindications to receiving propofol, hypotension at the end of surgery, or for whom different extubation plans (e.g. deep extubation, extubation in ICU) were required were excluded from the study. Patients were randomly allocated to either the intervention group (propofol) or the control group (placebo) with a 1:1 allocation ratio. Simple randomisation was performed once the patient was in theatre by the study's principal investigator, K Wakabayashi (KW), using the "RandomIZE" application program. KW generated all allocation sequences, enrolled patients, and assigned them to either group. Concealment of group allocation was achieved by restricting access to the enrolment results of the application program to all other assessors. Patients and outcome assessors were kept blinded to their group allocation.

Sample size calculations, based on the incidence of post-extubation cough from previous studies, indicated a sample of 22 patients per group would sufficiently power the study to detect a 33% difference in cough between the two groups, assuming an α-level of 0.05 and power (β) of 80%.[13,19] Once both groups achieved their enrolment targets of a minimum of 22 patients using simple randomisation, the study was stopped.

All patients were nil per os for eight hours preoperatively, and no anxiolytic premedication was administered. Once in the operating room, non-invasive BP, pulse oximetry, temperature, end-tidal carbon dioxide ($EtCO_2$), and electrocardiogram (ECG) were monitored at 1–3-minute intervals. Anaesthesia was induced with 1–2 mcg kg^{-1} fentanyl and 2.5 mg kg^{-1} propofol IV. Once the patient became unconscious, 0.6 mg kg^{-1} rocuronium was administered IV and tracheal intubation was performed. Anaesthesia was maintained with a low-flow mixture of oxygen, medical air, and sevoflurane to maintain a minimum alveolar concentration (MAC) of 1.0, with controlled ventilation to achieve an $EtCO_2$ of 35–45 mmHg. Vital signs were maintained within 20% of their baseline values intraoperatively until emergence, and analgesia was provided with 0.1 mg kg^{-1} morphine IV and 1 g paracetamol IV. Supplemental analgesia with 1 mcg kg^{-1} fentanyl boluses IV was provided as necessary. At the end of the surgery, a standardised extubation was performed, which included gentle oropharyngeal suction under direct vision, discontinuation of sevoflurane and manual ventilation with 95% oxygen. The level of neuromuscular paralysis was assessed using train-of-four (TOF) stimulation monitoring. Once a TOF count of three or four was measured, neostigmine 2.5 mg and glycopyrrolate 0.4 mg IV were administered. Two anaesthetists were involved in the extubation process. At a MAC value of 0.6, the second anaesthetist was asked to leave the operating theatre momentarily, while the first anaesthetist (KW) administered either propofol (0.5 mg kg^{-1}) or placebo (equivalent volume of 0.9% normal saline) to the patient. Once the intervention was completed, the second anaesthetist returned, and KW continued with the standardised extubation, by allowing the patient to emerge without further stimulation.

The second anaesthetist, who was blinded to the intervention, recorded a set of data during the emergence and extubation process.

The primary endpoint was measured by separately recording the amount and severity of bucking and coughing against TT using a modified Minogue scale:[20]

i. Grade 1 indicated no coughing or bucking.
ii. Grade 2 indicated one to two coughs or bucking.
iii. Grade 3 indicated sustained coughs or bucking less than five seconds.
iv. Grade 4 indicated severe or repetitive coughing or bucking lasting longer than five seconds.

Cardiorespiratory parameters, including systolic, diastolic, and mean arterial BP, HR, and pulse oximetry, as well as the time to emergence, were recorded throughout the extubation process. Once the patient opened their eyes with purposeful movement, with a TOF ratio > 0.9 and adequate spontaneous ventilation, the TT was removed. Oxygen supplementation was continued for 1–5 minutes. Cardiorespiratory complications including

laryngospasm, bronchospasm, tachycardia, and hypertension were recorded during extubation and treated if necessary. The patient was then transferred to the post-anaesthesia care unit (PACU). During the PACU stay, a final set of vitals was recorded.

Data analysis

Completed assessment forms were entered into a Microsoft Excel database once data collection was complete and exported into Stata 14.2 (StataCorp. 2015, College Station, TX, USA) for analysis. The demographic and clinical profiles of enrolled patients included in the analysis were described using descriptive statistics (frequencies and percentages for categorical data) as well as means and standard deviations for continuous variables. These descriptive analyses were compared to those in the intervention and control groups and assessed the adequacy of randomisation.

The effect of low-dose propofol was determined using a generalised linear model appropriate for the frequency of cough and poor (> 20% change from baseline) haemodynamic response. Univariate and multivariate correlation analyses were used to identify factors (independently and in combination) associated with cough, bucking, and poor haemodynamic responses as a composite outcome, adjusting for the intervention arm and other covariates.

Differences between the two groups were assessed using the Student's unpaired t-test for continuous data, the Mann–Whitney U test for non-normally distributed continuous data, and the chi-squared test for categorical data. A value of $p < 0.05$ was considered statistically significant.

The report was prepared in line with the CONSORT guidelines for reporting randomised control trials, as stipulated in the EQUATOR Network.

Results

A total of 62 patients aged 18 years and older were screened and 50 patients enrolled during the study period. Of these, 28 (56%) were randomised into the propofol group and 22 (44%) into the control group (Figure 1). All patients completed the study, and assessor blinding was maintained throughout. Key demographic and clinical characteristics of participants are summarised in Table I, and the two groups were comparable, with no significant differences in age, sex, ASA physical status, or time from intervention to extubation.

Frequency and severity of bucking and coughing at extubation

Overall, 21 participants experienced severe bucking, representing an incidence of 42% (95% confidence interval [CI] 28.8–56.4%), while nine participants had a severe cough at extubation equating to an incidence of 18% (95% CI 9.4–31.7%). The coughing severity scores were significantly lower in the propofol versus the control group (1.43% vs 2.09%; $p = 0.003$) (Figure 2A) and there were fewer complications at extubation (21.4% vs 50.0%; $p = 0.034$). Participants in the propofol group also experienced significantly less bucking and had significantly lower bucking severity scores compared to the control group (21.4% vs 68.2%; $p = 0.001$; and 1.93% vs 2.95%; $p < 0.001$) (Figure 2B).

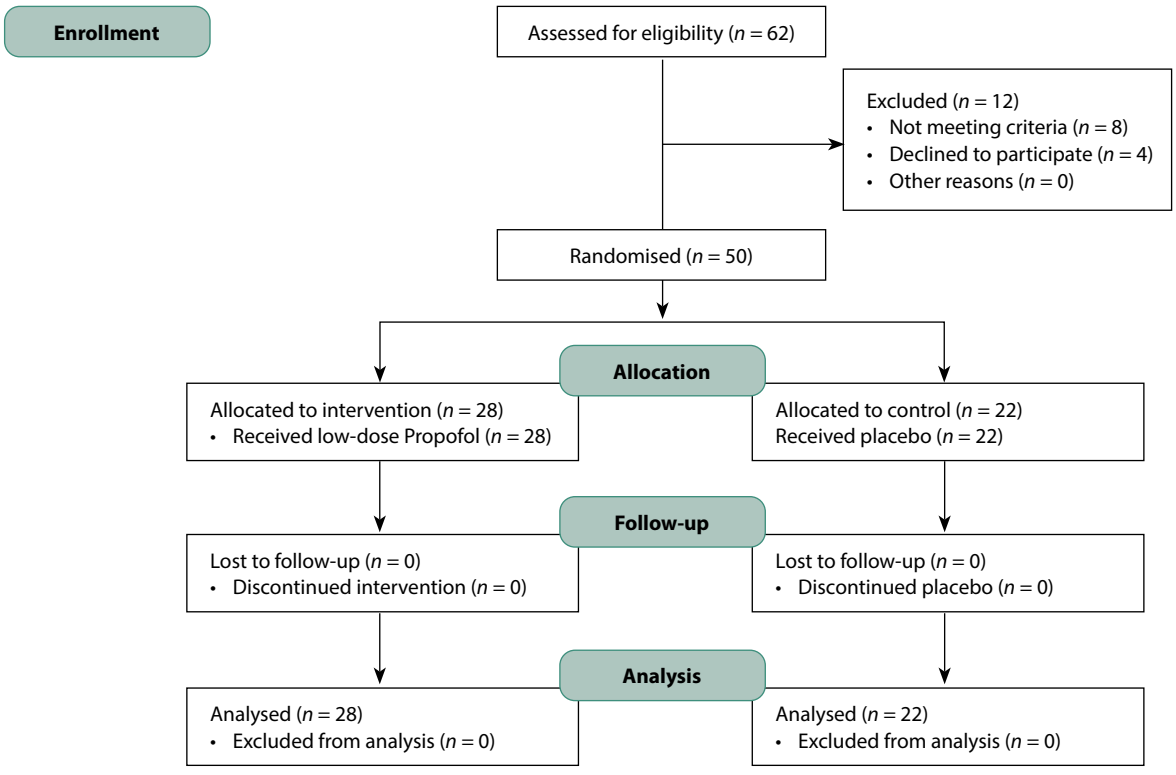
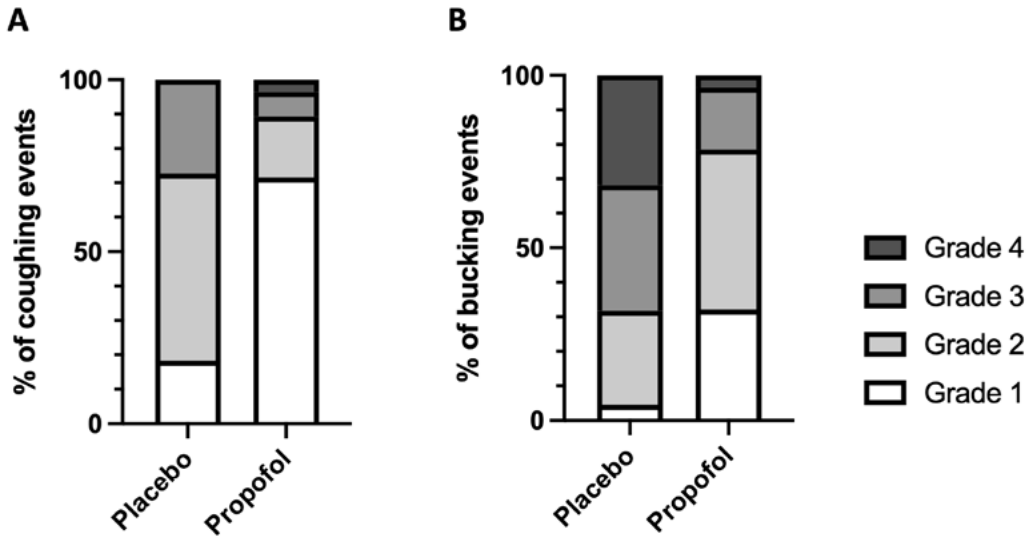


Figure 1: Consolidated standards of reporting trials flow diagram of patient selection

Table I: Patient demographics and clinical characteristics of the primary analysis (presented as either frequency, number and percentage of the total, or mean, standard deviation)

	Propofol (*n* = 28)	Placebo (*n* = 22)	*p*-value
Age (years) (mean ± SD)	49.0 ± 16.9	48.5 ± 12.7	0.917
Sex, male (*n*, %)	14 (50)	11 (50)	1.000
ASA status			
ASA I (*n*, %)	7 (25)	7 (31.8)	0.594
ASA II (*n*, %)	21 (75)	15 (68.2)	
One or more comorbidities (*n*, %)			
No	9 (32.1)	11 (50	0.201
Yes*	19 (67.9)	11 (50)	
One or more risk factors at extubation (*n*, %)			
No	17 (60.7)	15 (68.2)	0.585
Yes**	11 (39.3)	7 (31.8)	
Time from intervention to extubation in minutes (mean ± SD)	11 ± 3.5	13 ± 5.2	0.175

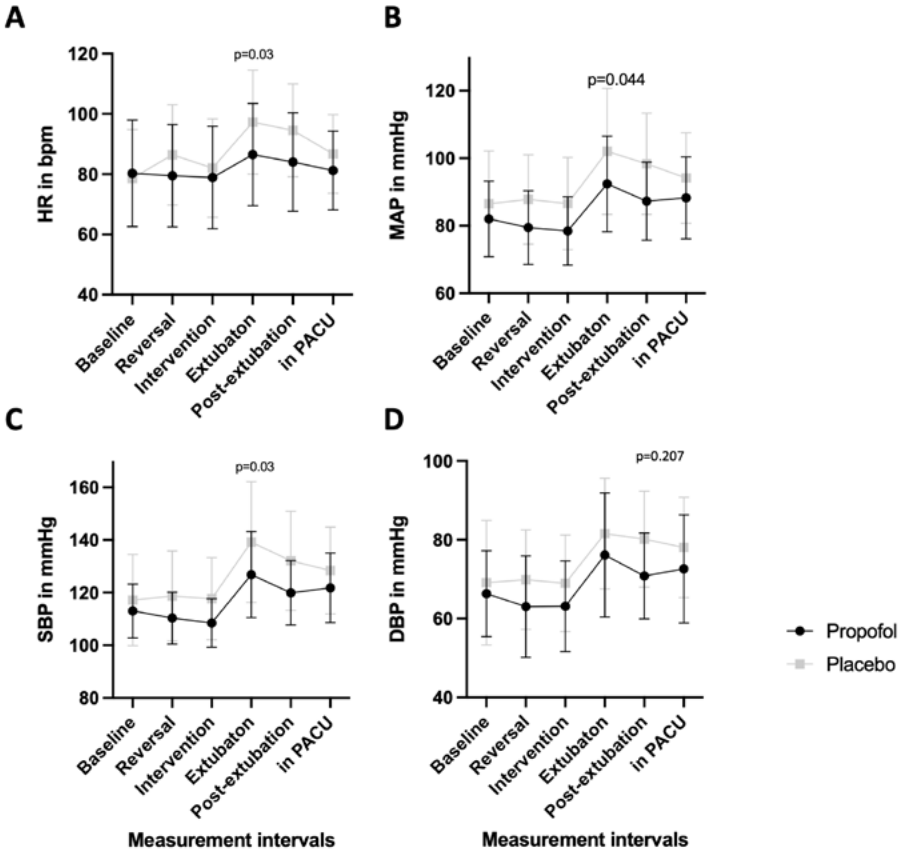
SD – standard deviation
*HIV, hypertension, diabetes, obesity, anaemia
**Includes morbid obesity, recent URTI, smoking



C

	Propofol (*n* = 28)	Placebo (*n* = 22)	*p*-value
Bucking severity score (mean ± SD)	1.93 ± 0.81	2.95 ± 0.90	< 0.001***
Severe bucking (*n*, %)			0.001**
No	22 (78.6)	7 (31.8)	
Yes	6 (21.4)	15 (68.2)	
Coughing severity score (mean ± SD)	1.43 ± 0.79	2.09 ± 0.68	0.003**
Severe coughing (*n*, %)			0.180
No	25 (82.3)	16 (72.7)	
Yes	3 (10.7)	6 (27.3)	
Extubation complications (*n*, %)			0.034*
No	22 (78.6)	11 (50.0)	
Yes	6 (21.4)	11 (50.0)	

Figure 2: Incidence and severity scores of coughing and bucking at extubation
(A) Comparison of frequency and severity grade of coughing between the placebo and propofol groups at extubation
(B) Comparison of frequency and severity grade of bucking between the placebo and propofol groups during emergence
(C) Representation of composite outcomes of bucking and coughing severity scores (Grade 3–4), the incidence of severe bucking and coughing, and incidence of extubation complications (tachycardia, hypertension, hypotension, laryngospasm, bronchospasm, desaturation, apnoea, vomiting, sore throat)
*$p < 0.05$
**$p < 0.01$
***$p < 0.005$
Grade 1 – no coughing or bucking, Grade 2 – one to two coughs or bucking, Grade 3 – sustained coughs or bucking less than five seconds, Grade 4 – severe or repetitive coughing or bucking lasting longer than five seconds, SD – standard deviation



E

	Propofol (*n* = 28)	Placebo (*n* = 22)	*p*-value
Heart rate bpm, (mean ± SD)	86.5 ± 17.0	97.3 ± 17.3	0.031*
Systolic blood pressure in mmHg (mean ± SD)	126.8 ± 16.4	139.2 ± 23.0	0.031*
Diastolic blood pressure in mmHg (mean ± SD)	76.1 ± 15.8	81.6 ± 14.1	0.207
Mean arterial pressure in mmHg (mean ± SD)	92.4 ± 14.2	102.0 ± 18.7	0.044*
Oxygen saturation in % (mean ± SD)	97.4 ± 2.2	97.8 ± 2.6	0.570

F

	Propofol (*n* = 28)	Placebo (*n* = 22)	X^2 test *p*-value
Significant HR response to extubation (*n*, %)			
No	21 (75.0)	9 (40.9)	0.015*
Yes	7 (25.0)	13 (59.1)	
Significant SBP response to extubation (*n*, %)			
No	20 (71.4)	14 (63.6)	0.058
Yes	8 (28.6)	8 (36.4)	
Significant DBP response to extubation (*n*, %)			
No	15 (53.6)	12 (54.6)	0.945
Yes	13 (46.4)	10 (45.4)	
Significant MAP response to extubation (*n*, %)			
No	16 (57.1)	15 (68.2)	0.425
Yes	12 (42.9)	7 (31.8)	

Figure 3: Haemodynamic changes from baseline measurement through emergence, extubation, and in PACU stay

(A) Comparison of HR changes between the placebo and propofol groups during emergence up until PACU ($p = 0.03$ at extubation)

(B) Comparison of mean arterial pressure changes between the placebo and propofol groups during emergence up until PACU ($p = 0.044$ at extubation)

(C) Comparison of systolic pressure changes between the placebo and propofol groups during emergence up until PACU ($p = 0.03$)

(D) Comparison of diastolic pressure changes between the placebo and propofol groups during emergence up until PACU

(E) Representation of mean differences of haemodynamic measures between the placebo and propofol groups at extubation

(F) Representation of composite haemodynamic changes from baseline to extubation between the placebo and propofol groups; a significant response was defined as a change of > 20% from the baseline

*$p < 0.05$

HR – heart rate, MAP – mean arterial pressure, SBP – systolic blood pressure, DBP – diastolic blood pressure, PACU – post-anaesthesia care unit, SD – standard deviation

There were, however, no differences between the two groups in the incidence of severe coughing (83.3% vs 72.7%; $p = 0.18$). The incidence and severity scores of coughing, bucking, and complication rates for both groups at extubation are summarised in Figure 2C. No differences in apnoea, desaturation, aspiration, vomiting, hypotension, and sore throat were observed between both groups during the PACU stay (78.6% vs 90.9%; $p = 0.238$).

Haemodynamic changes at extubation

Compared to the control group, patients in the propofol group had significantly lower HR at extubation (86.5 bpm vs 97.3 bpm; $p = 0.031$) (Figure 3A). They also exhibited significantly less rise in systolic BP compared to the control group (126.8 mmHg vs 139.2 mmHg; $p = 0.031$) (Figure 3C) and had lower mean arterial pressure readings (92.4 mmHg vs 102.0 mmHg; $p = 0.044$) at extubation (Figure 3B). Diastolic BP changes did not significantly differ between the two groups (Figure 3D). More patients in the control group exhibited an increase in HR at extubation compared to the propofol group (59.1% vs 25.0%; $p = 0.015$) (Figure 3F), although no other significant differences between the two groups were observed. The haemodynamic measures at extubation are summarised in Figure 3E.

Correlation between cardiac and respiratory changes at extubation

Analysis within both groups of bucking and cough severity scores and haemodynamic changes around extubation showed a positive correlation relationship. There was a moderate correlation between higher bucking severity scores and HR increases at emergence (Pearson's r = 0.478; 95% CI 0.2232, 0.6728; $p = 0.004$) (Figure 4A). Increased HR at extubation was also significantly correlated with higher bucking severity scores, specifically when comparing bucking severity Grades 1–3 ($p = 0.0326$), and 1–4 ($p = 0.0056$) (Figure 4B). There was also a weak positive correlation relationship between higher cough severity scores and HR increases at emergence (r = 0.2559, 95% CI -0.03259, 0.5051; $p = 0.07$) (Figure 4C). However, increases in HR at extubation did not significantly correlate with higher cough severity grades (Figure 4D).

Discussion

The results of this study show that a sub-hypnotic dose of propofol at 0.5 mg kg^{-1} significantly reduced the incidence and severity of bucking as well as the severity of coughing at extubation when compared to a placebo. Furthermore, administering low-dose propofol during emergence from GA significantly attenuated HR

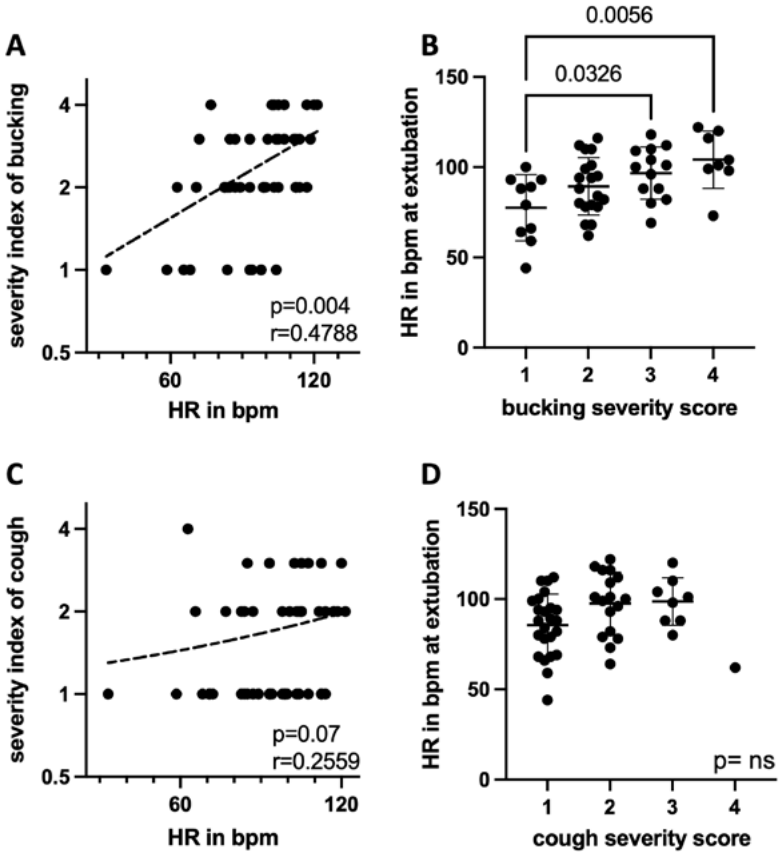


Figure 4: Subgroup analysis of bucking and coughing severity scores with HR changes at emergence and extubation using linear regression models
(A) Positive correlation relationship between higher bucking severity scores and HR increases at emergence (Pearson's r = 0.4788, $p = 0.004$)
(B) HR changes at extubation based on bucking severity scores with mean and standard deviations (1–3, $p = 0.0326$; 1– 4, $p = 0.0056$)
(C) Positive correlation relationship between higher coughing severity scores and HR increases at extubation (Pearson's r = 0.2559, $p = 0.07$)
(D) HR changes at extubation based on cough severity scores with mean and standard deviations
HR – heart rate, Grade 1 – no coughing or bucking, Grade 2 – one to two coughs or bucking, Grade 3 – sustained coughs or bucking less than five seconds, Grade 4 – severe or repetitive coughing or bucking lasting longer than five seconds

and systolic and mean BP changes. Fewer complications were encountered at extubation in the propofol group. However, the incidence of coughing at extubation was not attenuated by low-dose propofol. There were no significant differences in extubation time between the two groups. Higher bucking severity scores correlated with higher HR at extubation in both groups.

Previous studies have reported mixed results with low-dose propofol administration at emergence from anaesthesia. Jung and colleagues found that a 0.3 mg kg^{-1} dose of propofol reduced the incidence and severity of coughing in adults undergoing nasal surgery.[13] Similar studies in paediatric populations found that propofol at 0.25–0.5 mg kg^{-1} was able to reduce coughing in children at emergence from GA.[12,21] At a dose of 0.8 mg kg^{-1}, propofol reduced the incidence and severity of cough and laryngospasm in adults undergoing oropharyngeal procedures.[10] Safavi and colleagues used 0.25 mg kg^{-1} propofol and also found a reduced incidence of cough at emergence.[16] However, Vaziri et al. found that propofol at 0.5 mg kg^{-1} did not result in significant haemodynamic and respiratory outcomes at extubation.[8] Similarly, a study by Ozturk et al. showed that propofol at 0.5 mg kg^{-1} was not able to reduce coughing in children at extubation, but reported a reduction in emergence delirium when compared to a placebo.[9] Furthermore, when comparing low-dose propofol with other interventions, Nagrale and colleagues found that although propofol 0.5 mg kg^{-1} blunted the coughing and bucking response to extubation, esmolol was superior in effect.[14] Overall, the time to extubation was not prolonged with propofol administration in any of the above studies.

Tracheal intubation remains the focus of research and guidelines when it comes to airway management. However, the Royal College of Anaesthetists' 4th National Audit Project (NAP4) found that more cardiorespiratory complications occurred during emergence and extubation than at any other time during GA.[22] Many complications at emergence may appear transitory and minor, but the NAP4 results noted that all complications were both preventable and had the potential to result in long-term morbidity and mortality. Hence, it is important to recognise that airway management extends into the postoperative period. In our study, propofol was shown to reduce the incidence of complications at this critical point.

Following the findings of NAP4, the Difficult Airway Society published general guidelines on the management of tracheal extubation, and included recommendations for improving physiological conditions at emergence.[23] However, there is still a lack of large-scale trials on optimal attenuation strategies peri-extubation, but it is clear from reviews on the topic that any intervention is better than a placebo or no intervention.[5] The rapid onset of action and the short duration of propofol makes it an ideal drug for attenuating the cardiovascular responses to tracheal extubation, as shown in several studies.[10,12,13,21] Airway protection afforded by the cough reflex is not adversely affected by residual sedation after propofol administration, meaning that patients are not at increased risk of uncleared secretions or aspiration after extubation.[24]

Blunting the extubation response has been shown to reduce sympathetic nervous system activation during emergence and improve patient recovery.[1] However, although extubation is often associated with bucking, previous studies investigating propofol and other agents did not assess their effects on bucking. Our study found a positive correlation between higher bucking severity scores and increased HR at extubation, indicating that the presence of one of these findings would likely predict the presence of the other.

Bucking against the TT was shown to pre-empt bronchospasm and prevention of bucking was described as a successful method in preventing bronchospasm.[25] Furthermore, perioperative cardiorespiratory aberrations, apart from coughing, have been found to affect outcomes in terms of myocardial injury, morbidity and postoperative mortality.[26,27]

Intraoperative tachycardia and hypertension were independently found to predict morbidity in the postoperative period.[27] Tachycardia was also associated with worse outcomes, leading to increased morbidity, mortality, as well as a prolonged length of hospital stay. Hence, blunting these cardiorespiratory changes at emergence may improve patient recovery and postoperative outcomes. In our study, low-dose propofol was shown to significantly blunt these responses at extubation.

The mechanism of action of propofol includes peripheral and central activity, with potentiation of γ-aminobutyric acid Type A receptor activation and inhibition of further synaptic transmission, as well as inhibition of release of the excitatory neurotransmitters.[28] The cardiovascular effects of propofol include a reduction in the mean arterial pressure and blunting of the baroreceptor reflex, thereby preventing an increase in HR in response to a drop in BP.[28,29] Its central effects include an overall inhibition of excitatory signal transduction, which leads to a smooth, rapid emergence after propofol administration.[29] This may explain why our findings of low-dose propofol attenuate the cardiorespiratory changes at extubation.

Study limitations

The primary limitation of this study was our patient sample size which failed to provide sufficient power to detect a difference in our primary outcome – the incidence of cough – and confirm our hypothesis that propofol would reduce the incidence of cough at extubation compared to a placebo. We had estimated that a sample size of 22 patients per group would provide sufficient power (1-β = 80%) to detect a 33% difference in the incidence of cough between the two groups, based on previous studies.[13,19] However, the observed difference in cough between the two groups was considerably less (16.6%), meaning the sample size provided only 32% power to detect a difference. This result means that the sample size should have been approximately double (47 patients per group = a total of 94), to sufficiently power the study (1-β = 80%).

Other limitations include the fact that the optimal sub-hypnotic dose of propofol has not been determined, meaning that the results we collected by administering 0.5 mg kg^{-1} propofol are not reflective of its ideal potential to blunt the extubation response. The optimal timing of when to administer propofol is also unknown. The lack of double-blinding and assessor inter-rater variability may have contributed to cognitive and measurement biases. A standardised extubation protocol was implemented to reduce the risk of experimenter bias since the principal investigator was performing the extubation himself.

Recommendations

In line with previous studies, our small, randomised control trial has shown that the administration of a low dose of propofol significantly reduced cardiorespiratory responses at extubation. However, replication with larger sample sizes is required to confirm its efficacy in reducing bucking, coughing, and haemodynamic aberrations.

Additional, larger studies with patient populations undergoing different types of surgery, including those with ASA physical status III and above are required before any recommendation regarding the use of low-dose propofol to promote smooth emergence in these populations can be made. In addition to establishing efficacy, studies to determine the optimal dose of the drug are also required, particularly as previous studies used different doses, resulting in the safest, most effective dose being unknown. Another important area for research is to determine the relative efficacy of propofol in comparison to other drugs that have also been shown to effectively facilitate smooth extubation and minimise cardiovascular and respiratory responses to extubation, including lignocaine, dexmedetomidine, calcium channel blockers, and beta blockers.[5]

Conclusion

Administering propofol 0.5 mg kg^{-1} attenuates respiratory responses, particularly bucking, at extubation after GA in healthy adults undergoing elective abdominal or pelvic surgery without increasing the time to extubation. Low-dose propofol blunts aberrations in HR, as well as systolic and mean BP at extubation, with fewer complications.

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Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Ethical approval was obtained from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand before commencement (ethics number: M210232).

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