Original Article

Efficacy of Low-dose Suxamethonium in Reducing Induction Dose of Propofol for Laryngeal Mask Airway Insertion in Nigerian Adults

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Abstract

Background: Propofol is commonly used as a sole induction agent during laryngeal mask airway (LMA) insertion, and additional doses are often required with systemic consequences. **Objective:** Our study evaluated the effect of 0.1 mg/kg suxamethonium on the dose of induction of propofol when used during insertion of LMA. **Methodology:** Eighty patients aged between 18 and 60 years were included in this prospective study. Patients undergoing elective procedures under general anaesthesia with LMA and spontaneous ventilation were randomized into two groups. Patients in both groups were induced with an initial dose of 2.5 mg/kg of propofol; the control group (group P) and the second group (group S) received 5 ml of normal saline and 0.1 mg/kg of suxamethonium made up to 5 ml, respectively. The need for additional doses of propofol following insertion of LMA was then assessed based on insertion conditions (ease of insertion, severity of airway response in terms of coughing, gagging, laryngospasm, and patient movement). The total dose of propofol required before successful insertion was recorded, as well as the incidence and duration of apnea post-induction. The pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were assessed at 0, 1, 3 and 5 min after insertion of LMA for each group. **Results:** The total dose of propofol required for successful LMA insertion was significantly lower in group S (2.82 ± 0.39 mg/kg) compared to group P (3.13 ± 0.49 mg/kg), $P = 0.002$. Haemodynamic parameters (SBP, DBP, MAP, PR, and SpO_2) were well-controlled post-LMA insertion and were comparable between the two groups. Furthermore, no incidence of hypotension or bradycardia requiring intervention was observed at different time intervals, throughout the study period, in both groups. **Conclusion:** The dose of propofol is lesser when low-dose suxamethonium (0.1 mg/kg) precedes its use for induction during LMA insertion, and there is a reduction in the duration of apnea; however, it causes a higher apnea incidence. Vital signs were similar in the two study groups.

Keywords: Laryngeal mask airway insertion, low-dose suxamethonium, propofol induction

Introduction

The airway device laryngeal mask airway (LMA) is of importance during general anaesthesia as well as in emergency airway management. It came into clinical practice in 1988 though it was first designed by Archie Brain in 1981.[1] Although propofol is the drug of choice for induction during LMA insertion because of its depressant effect on airway reflexes, propofol alone at the recommended induction dose of 1.5–2.5 mg/kg does not always guarantee successful LMA insertion. The total amount of propofol required to ease insertion and prevent reflex airway response when used alone exceeds 3.0 mg/kg.[2] This increases the incidence of dose-related side effects such as hypotension, bradycardia, and apnea.[3] The insertion of LMA following induction with propofol in the absence of a muscle relaxant would require a depth of anaesthesia adequate enough to depress reflexes of the airway; the propofol dose to achieve that depth of anesthesia however, varies from one patient to another.[4]

Propofol is an alkylphenol derivative with a rapid onset of action, good recovery profile, and antiemetic effects. These

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qualities make it a popular induction agent for short procedures and day-case surgeries. It has a distribution half-life of 2–8 min[3] and induction of anaesthesia after a bolus dose occurs in 30–45 s, which is one-arm brain circulation time. The time to peak effect (as assessed by bispectral index) is 1.5 min.[3,5]

The loss of response to verbal command and loss of eyelash reflex is recognized as endpoints of propofol induction.[6] This endpoint, however, does not indicate an optimal anaesthetic state that yields a smooth LMA insertion, resulting in reflex airway responses. A lesser induction dose of propofol can be achieved by the addition of other agents such as midazolam, ketamine, low-dose muscle relaxants, and opioids, thus reducing undesired effects such as hypotension, respiratory depression, and reflex airway responses.[6-8]

Suxamethonium, a depolarizing muscle relaxant, at a low dose (0.1 mg/kg), has been found to suppress laryngeal reflexes by depolarization of motor end-plate,[9] thus lessening the amount of propofol required to achieve LMA insertion. It has a rapid onset and offset of action. It decreases the incidence of coughing and gagging without causing full muscle paralysis.[10] Low-dose suxamethonium has no significant effect on spontaneous ventilatory effort and repeated doses do not cause bradycardia.[11]

This research work was aimed at evaluating the effect of suxamethonium at a dose of 0.1 mg/kg on propofol dose used for LMA insertion, its effect, and related postoperative complications in adult patients undergoing general anaesthesia in our setting.

Methodology

This was a randomized single-blinded clinical study prospectively performed in a tertiary institution in North West Nigeria. With the hospital ethics committee approval, we obtained informed consent from 80 American Society of Anaesthesiologist status classification I and II patients aged between 18 and 60 years. In-patients eligible for the study were routinely reviewed a day before the scheduled surgery on the ward by the investigator and day-care patients were evaluated for inclusion in the study on the morning of surgery. Patients who are known to have hypersensitivity to propofol or suxamethonium, family history of plasma cholinesterase deficiency, neuromuscular disorders, family history of malignant hyperthermia, restricted mouth opening (inter-incisor gap <2.5 cm), cervical spine disease, at risk of aspiration, undergoing oral or nasal surgery, obese with body mass index >35 kg/m^2, taking sedative drugs, and with failed LMA insertion were excluded.

A formula used to determine the appropriate sample size for the study showed that a minimum of 72 patients were required.[12] Allowing for an estimated 10% nonconsent or dropout rate, the total sample size for the 2 groups was 80 patients.

Patients enrolled in the study were randomly allocated into one of two groups. Eighty pieces of uniformly sized sheets of paper were labelled P or S (40 each), representing groups P (placebo) and group S (suxamethonium), respectively. These papers were folded and shuffled in a large box. Each patient picked one folded sheet of paper from the box and handed it over to the research assistant. The patient's hospital file number was written on a sheet of paper and sealed in a separate envelope that was only opened after completion of the study. The investigator was blinded to the study drug.

The study drug (suxamethonium 0.1 mg/kg) and placebo (0.9% saline) were prepared in identical 5-ml syringes, by the research assistant according to the patient's group, while the investigator remained blinded. The study drug had the same appearance (colourless) as the placebo, made up to equal volumes of 5 ml, and the content of each syringe was not disclosed to the investigator. The initial dose of propofol based on body weight was prepared in a 20-ml syringe. Another preparation was made inside a 10-ml syringe for rescue doses. Lidocaine 1% was added to propofol in a 1:10 ratio to minimize injection pain.

On arrival in the operating room, the patient was positioned supine on the operating table and baseline vital signs which include noninvasive systolic and diastolic blood pressures (SBPs and DBPs), mean arterial pressure (MAP) blood, pulse rate (PR), respiratory rate, peripheral arterial oxygen saturation of haemoglobin (SpO2), and electrocardiograph were obtained. An intravenous access was secured using an 18G cannula and 0.9% saline infusion was commenced. The LMA classic TM (TeleflexR) was used in all patients of either group. The size of LMA was chosen based on patients' weight as recommended by the manufacturer.[1] These patients were scheduled for short elective surgeries (in-patients and day-surgery cases) under general anaesthesia with LMA as the airway device of choice.

Patients were preoxygenated with 100% oxygen at the rate of 5–6 L/min for 3–5 min using a Bain circuit with a tight-fitting facemask. The LMA was prepared by pressing the concave part of the mask against a hard surface and its cuff was deflated. The back of the mask was then lubricated using K-Y Jelly. All patients were induced with 2.5 mg/kg intravenous 1% propofol injected continuously over 30 s by the research assistant. The adequacy of induction was assessed 30 s later (i.e., 60 s after the start of propofol injection) by loss of response to verbal command (open your eyes) the amount of propofol injected was noted. The patients immediately then received either 5 ml of 0.9% saline in group P or 5 ml of low-dose suxamethonium, the investigator then assessed for adequacy of mouth opening 30 s after administration of the study drug or placebo. If there was an inability to open the mouth or inadequate mouth opening (jaw not relaxed), a bolus dose of 0.5 mg/kg propofol was given to deepen anesthesia and a reassessment was done 30 s later.

When mouth opening was adequate, the patient's head was extended and the neck was flexed on the chest (sniffing position). The LMA was grasped like a pen in the dominant hand, with the

tip of the index finger at the junction of the mask and tube. It was inserted into the mouth and advanced until resistance was felt. The cuff was then inflated with an appropriate volume of air until the LMA tube was seen to rise slightly out of the patient's mouth. If mouth opening was adequate but correct positioning after insertion was not achieved because of severe airway response, or head or body movement, the LMA was removed. A dose of 0.5 mg/kg of propofol was administered to deepen anaesthesia and reinsertion was attempted 30 s later. Not more than three attempts at insertion were permitted for the study. In between insertion attempts, the patients were ventilated via a facemask with 100% oxygen devoid of volatile agents at a flow rate of 5–6 L/min. If LMA insertion was to be unsuccessful after three attempts, the patient's airway would have been secured with an appropriate-sized endotracheal tube. This was termed as a failure of LMA insertion and the patient would have been excluded from the study.

Following successful insertion of the LMA, the patient was connected to the anaesthesia machine via the Bain circuit. Correct LMA placement was ascertained by auscultation for bilaterally equal air entry and square wave form on capnograph trace in spontaneously breathing patients or during assisted breaths in patients with apnea. If correctly placed, the LMA was then secured with adhesive tape. Anaesthesia was maintained on 1%–2% minimum alveolar concentration isoflurane in 50% oxygen in air at a total fresh gas flow rate of 5–6 L/min. The total dose of propofol injected was recorded. The haemodynamic changes (SBP, DBP, MAP, and PR) were recorded before induction of anaesthesia and 0, 1, 3 and 5 min after the insertion of LMA. Any incidence of hypotension, bradycardia, and apnea was noted and treated accordingly. Hypotension was defined as more than 30% decrease in baseline SBP, while bradycardia was defined as more than 30% decrease in baseline PR. Apnea was defined as cessation in breathing, as evidenced by absence of chest movement, from the end of propofol injection. The duration of apnea, defined as the time from successful LMA insertion till return of spontaneous breathing, was noted. Assisted ventilation was to be given to apneic patients (with SpO_2 below 90%) through the LMA to maintain SpO_2 above 95% and end-tidal carbon dioxide concentration between 35 and 45 mmHg till resumption of spontaneous breathing.

The end points were considered to be when the patient was able to make appropriate tidal volume, and when tidal volumes were stable enough and comparable to the baseline values. No further intraoperative anesthetic management was influenced by the study. Protocol for the management of anticipated adverse effects is as follows:

Hypotension: Intravenous ephedrine 3 mg

Bradycardia: Intravenous atropine 0.01 mg/kg

Desaturation: 100% oxygen via face mask at 5–6 L/min.

The data obtained were recorded on the preformed data collection form. Data entry was done by two separate clerical staff and was further cross-checked for consistency.

The data obtained were analyzed using Statistical Package for the Social Sciences (SPSS is a software package used for statistical analysis produced by SPSS Incorporated and acquired by IBM in 2009) version 21.0. Quantitative variables such as age, weight, height, total propofol requirement per kilogram body weight, and the duration of apnea were summarized using mean (± standard deviation) and compared using independent *t*-test. Qualitative variables such as the incidence of apnea were summarized using percentages and compared using Chi-squared test or Fisher's test.

Results

Table 1 shows the demographic profile of patients in the two groups, and the results were comparable. There was no statistically significant difference between the two groups with respect to age, weight, and sex distribution.

The mean total propofol consumed in group P, 186.15 ± 24.04 mg, was significantly higher than in group S, 174.00 ± 24.89 mg ($P = 0.029$). The mean dose of propofol per body weight used in group P (3.13 ± 0.49 mg/kg) was significantly higher than in group S (2.82 ± 0.39 mg/kg) ($P = 0.002$). The mean duration of apnea in group P (1.04 ± 1.26 min) was longer than in group S (0.65 ± 0.85 min), but there was no statistically significant difference ($P = 0.107$) [Table 2].

Both systolic and DBPs showed no significant statistical difference for both groups S and P at the different study timings [Tables 3 and 4]. Post-insertion of the LMA, the mean SBP of patients in both groups was comparable ($P = 0.709$, $P = 0.351$, $P = 0.707$, and $P = 0.617$ at 0, 1, 3 and 5 min post insertion, respectively). The mean DBP of patients in group P was higher than those in group S at 0, 1, 3 and 5 min post-insertion of the LMA, but the differences were not statistically significant ($P = 0.124$, $P = 0.471$, $P = 0.251$, and $P = 0.338$, respectively).

Table 5 shows the mean MAP changes at different time intervals in the two groups; the mean MAP of patients in group P was comparable with those in group S ($P = 0.927$, $P = 0.767$, $P = 0.645$, and $P = 0.612$ at 0, 1, 3 and 5 min post-insertion, respectively).

Table 6 shows the mean PR changes at different time intervals in the two groups. At all study timings post insertion of the LMA, the mean PR of patients in group P was lower than those in group S. However, the difference was not statistically significant ($P = 0.731$, $P = 0.138$, $P = 0.082$, and $P = 0.112$ at, 0, 1, 3 and 5 min post insertion, respectively).

Table 1: Patients' demographic data and clinical characteristics

	Group P (n=40)	Group S (n=40)	P
Age (years)	38.40±14.01	35.55±14.01	0.319
Weight	60.50±6.87	61.90±6.83	0.364
BMI (kg/m^2)	22.00±1.50	22.08±2.29	0.863
Gender (male: female)	18:22	26:14	0.072
ASA (I: II)	34:6	31:9	0.390
Interincisor gap (cm)	7.13±0.82	6.75±1.08	0.085

BMI: Body mass index

Table 2: Induction characteristics of patients

	Mean±SD		*P*
	Group P	Group S	
Initial bolus propofol (mg)	152.50±15.73	155.00±18.08	0.511
Additional propofol given (mg)	34.65±24.81	17.75±21.24	0.002*
Mean total propofol consumed (mg)	186.15±24.04	174.00±24.89	0.029*
Mean dose of propofol used (mg/kg)	3.13±0.49	2.82±0.39	0.002*
Mean duration of apnea (min)	1.04±1.26	0.65±0.85	0.107

SD: Standard deviation

Table 3: Changes in mean systolic blood pressure at different time intervals

SBP (mmHg)	Mean±SD		*P*
	Group P	Group S	
Baseline	131.00±4.22	131.90±6.83	0.481
After LMA insertion (min)			
0	113.60±9.61	114.40±9.49	0.709
1	108.70±9.69	106.60±10.32	0.351
3	108.70±12.49	107.00±11.18	0.707
5	110.70±11.98	109.40±11.17	0.617

SBP: Systolic blood pressure, LMA: Laryngeal mask airway, SD: Standard deviation

Table 4: Changes in mean diastolic blood pressure at different time intervals

DBP (mmHg)	Mean±SD		*P*
	Group P	Group P	
Baseline	78.50±6.44	78.20±9.32	0.867
After LMA insertion (min)			
0	66.50±7.19	65.40±8.69	0.124
1	63.20±8.14	61.60±11.35	0.471
3	63.60±10.47	60.60±12.65	0.251
5	65.80±7.74	64.00±8.91	0.338

DBP: Diastolic blood pressure, LMA: Laryngeal mask airway, SD: Standard deviation

Table 5: Changes in mean arterial pressure at different time intervals

MAP (mmHg)	Mean±SD		*P*
	Group P	Group S	
Baseline	96.40±4.60	96.50±7.51	0.943
After LMA insertion (min)			
0	82.60±6.71	82.40±12.12	0.927
1	78.60±8.33	78.00±9.68	0.767

MAP: Mean arterial pressure, LMA: Laryngeal mask airway, SD: Standard deviation

At all study timings post insertion of the LMA, the mean SpO_2 of patients in group P was comparable to those in group S and the difference was not statistically significant ($P = 0.245$, $P = 0.632$, $P = 0.129$, and $P = 0.386$ at 0, 1, 3 and 5 min post insertion, respectively) [Table 7].

Figure 1 shows the incidence of drug side effects (apnea, hypotension, and bradycardia) in both study groups. Apnea was observed in 20 patients (50.0%) in group P and 23 patients (57.5%) in group S. The difference was not statistically significant ($P = 0.501$). There was no incidence of hypotension or bradycardia (30% or more drop in MAP and PR respectively) in the two study groups.

Apnea was observed in 20 patients (50.0%) in group P and 23 patients (57.5%) in group S. The difference was not statistically significant ($P = 0.501$). The mean duration of apnea was 1.04 min in group P and 0.65 min in group S; the difference was not statistically significant ($P = 0.107$).

Discussion

In order to avoid complications such as laryngeal spasm, gaging and coughing during insertion of LMA, optimal conditions are required; these include a sufficient mouth opening with suppression of airway reflexes and a relaxed jaw.

The findings from this study showed that following propofol induction, suxamethonium at 0.1 mg/kg significantly reduced the required propofol dose appropriate for insertion of LMA. We found that the initial propofol requirement based on the standard induction dose of 2.5 mg/kg was similar in the two groups, $P = 0.511$. However, additional propofol requirement as seen in patients that received suxamethonium was significantly less than those that received the placebo, $P = 0.002$. Consequently, the mean dose of propofol required before successful LMA insertion was less in the suxamethonium group (2.82 mg/kg) compared to the placebo group (3.13 mg/kg), $P = 0.002$. This result is consistent with the findings from other studies.[9,13,14]

Aghamohammadi *et al*.[13] reported that the number of patients that required additional propofol before successful LMA insertion in their suxamethonium group (10%) was significantly lower compared to that (53%) in the placebo group, $P = 0.001$. Similarly, Jain and Parikh[14] found a significant reduction in the dose of propofol required for insertion of the LMA in the propofol plus suxamethonium group (2.66 mg/kg) compared to the propofol-only group (3.1 mg/kg), $P < 0.01$.

On the contrary, Salem[6] did not find any significant difference in the propofol requirement in the group that had suxamethonium compared to the placebo group, $P > 0.05$. This

Table 6: Changes in mean pulse rate at different time intervals

PR (beats/min)	Mean±SD		*P*
	Group P	Group S	
Baseline	82.40±8.54	81.30±8.24	0.559
After LMA insertion (min)			
0	97.30±10.80	98.20±12.43	0.731
1	94.30±11.90	98.20±11.37	0.138
3	94.80±13.74	99.70±10.95	0.082
5	92.70±12.33	96.60±9.16	0.112

LMA: Laryngeal mask airway, PR: Pulse rate, SD: Standard deviation

Table 7: Changes in mean oxygen saturation at different time intervals

SPO_2 (%)	Mean±SD		*P*
	Group P	Group S	
Baseline	99.80±0.41	99.65±0.48	0.136
After LMA insertion (min)			
0	99.50±0.51	99.58±0.76	0.245
1	98.90±0.96	99.00±0.91	0.632
3	97.40±2.41	98.10±1.60	0.129
5	98.00±1.92	98.30±1.02	0.386

SPO_2: Oxygen saturation, LMA: Laryngeal mask airway, SD: Standard deviation

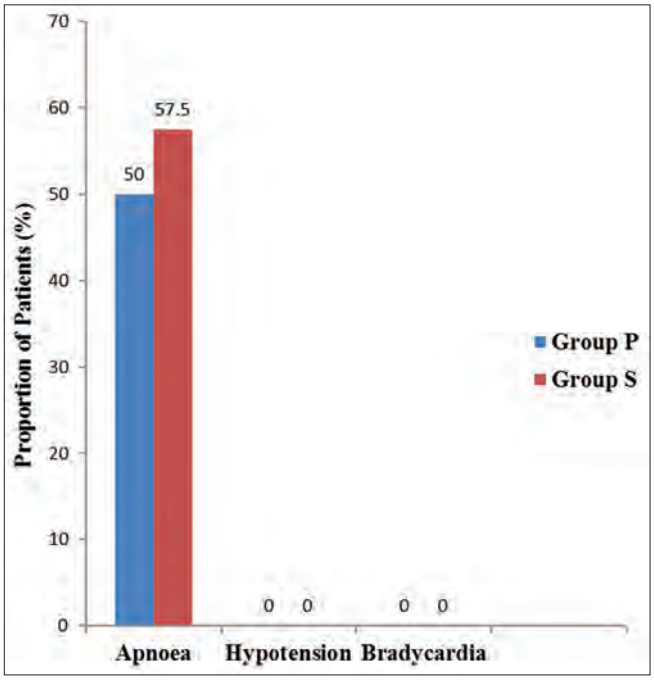

Figure 1: Incidence of drug side effects during laryngeal mask airway insertion

is likely because patients who required more than one attempt at insertion in his study were kept anaesthetized with isoflurane unlike the additional propofol doses used in this present study.

In this study, a reduction from baseline SBP, DBP, and MAP and a rise in PR were observed at all study timings post-LMA insertion in both study groups. This conforms to the report of Jamil *et al*.[9] who observed a drop in MAP and a rise in PR from baseline at all study timings in both the placebo and suxamethonium groups. Furthermore, despite the higher propofol consumption in the placebo group compared to the suxamethonium group ($P = 0.029$), the MAP at the, 0, 1, 3 and 5 min in the two groups was comparable, $P = 0.927$, 0.767, 0.645, and 0.612, respectively. This could be explained by the fact that the higher number of patients requiring extra doses of propofol in the placebo group might have had airway responses to initial LMA insertion resulting in a pressor effect that attenuated the hypotensive changes that comes with propofol induction.

Contrary to the finding in this study, Ho and Chui[15] reported that the mean reduction in the MAP within the first 5 min of insertion was more profound in the placebo group compared to the suxamethonium group. The explanation for this is likely due to the recruitment of the elderly patients in their placebo group compared to the suxamethonium group. Elderly subjects are more prone to the cardiodepressant effect of propofol than middle-aged or young adults.[16] Also, a rescue propofol dose of 1 mg/kg was used in their study rather than the 0.5 mg/kg in this present study, which might have been responsible for more drops in the MAP. However, there was no significant difference in the changes in heart rate between the two groups ($P > 0.05$).

George *et al*.[17] also found that haemodynamic changes after insertion of the LMA in patients who received either placebo, suxamethonium 0.1 mg/kg, or 0.25 mg/kg following propofol induction were similar.

In this study, none of the patients in both study groups developed severe hypotension or bradycardia. This is similar to the report of Jain and Parikh.[14] George *et al*.,[17] however, noted 9.2% of patients developed hypotension that required a vasopressor. This could be attributed to the fentanyl pretreatment given to their patients, which potentiates the cardiorespiratory depressant effect of propofol.[16]

The SpO_2 values in both study groups in the current study were found relatively unchanged at all study timings post-LMA insertion. This is consistent with the report of Jain and Parikh.[14] Salem, however, noted a trend toward reduction in SpO_2 from baseline values post LMA insertion. Although their patients were preoxygenated and their apnea periods were similar to those recorded in this study, they were kept oxygenated in between insertion attempts with oxygen from an isoflurane–nitrous oxide mixture rather than the 100% oxygen used in this study.

Duration of action of suxamethonium is dose-dependent; reducing the dose allows a more rapid return of spontaneous ventilation and airway reflexes.[18] In this study, a higher incidence of apnea occurred in the suxamethonium group compared to the placebo group, though the difference was not significant, $P = 0.501$. However, the mean duration of apnea was shorter in the suxamethonium group compared to

the placebo group, but the difference was also not significant, $P = 0.107$. Similarly, Ho and Chui[15] did not find any significant difference in the duration of apnea between the suxamethonium and placebo groups, $P = 0.46$. The incidence of apnea recorded by Jain and Parikh[14] in their propofol plus suxamethonium group (84%) and the propofol-only group (80%) is higher than those (57.5% and 50%, respectively) obtained in this study. This is likely due to the higher suxamethonium dose (0.25 mg/kg) used in their study.

Contrary to the finding in this study, Jamil *et al*.[9] reported a significantly higher incidence of apnea in the placebo group compared to suxamethonium group, $P < 0.05$. The difference in propofol consumption in their two study groups (0.7 mg) was wider than that observed in this study (0.31 mg). That difference might have accounted for the significantly higher incidence of apnea in their placebo group.

Conclusion

Suxamethonium (0.1 mg/kg) following induction reduced propofol consumption for LMA insertion. The haemodynamic responses following LMA insertion using propofol combined with suxamethonium (0.1 mg/kg) were well controlled and comparable to when propofol is used as a sole induction agent. The duration of apnea is reduced in patients that receive low-dose suxamethonium, but the incidence of apnea is similar between the two study groups.

Recommendation

Suxamethonium is a readily available drug and its role in the significant reduction of induction dose of propofol, while using LMA makes it desirable and is recommended for use.

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Conflicts of interest

There are no conflicts of interest.

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