Nifedipine attenuates the intraocular pressure response to intubation following succinylcholine

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Forty patients without eye disease, undergoing elective non-ophthalmic surgery, were studied to evaluate the efficacy of sublingual nifedipine in attenuating the intraocular pressure response to succinylcholine administration, laryngoscopy and intubation. The patients were randomly given either nifedipine 10 mg or placebo sublingually 20 minutes before induction of anaesthesia. Intraocular pressure (IOP) and systolic blood pressure (SBP) were recorded before and after induction of anaesthesia. The IOP response to succinylcholine administration, laryngoscopy and intubation was significantly less in patients receiving nifedipine ($P < 0.01$). The mean maximum rise in IOP above basal level at one minute post-intubation was 7.82 mmHg in the control group compared with 0.15 mmHg in the nifedipine pre-treated group. These results suggest that sublingual nifedipine is effective in attenuating the IOP response after succinylcholine administration, laryngoscopy and intubation.

Control of intraocular pressure (IOP) is crucial for open ophthalmic procedures.[1] Proper anaesthetic management contributes significantly to a successful surgical outcome. Succinylcholine administration causes a transient but significant increase in IOP,[2] which is further increased by tracheal intubation.[3] Various attempts have been made to attenuate the IOP response, but most of these methods have been only partially successful.[4–8]

A sudden increase in blood pressure may cause a transient but acute increase in IOP until the aqueous flow dynamics can accommodate.[9] Nifedipine, a calcium channel blocker, has been found to attenuate the reflex circulatory response to laryngoscopy and intubation.[10] Thus, the present investigation was planned to study the effects of nifedipine on IOP.

Key words
EYE: intraocular pressure; PHARMACOLOGY: calcium channel blocker, nifedipine; NEUROMUSCULAR RELAXANTS: succinylcholine.

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Methods

Forty patients of either sex, aged 30–40 years, ASA physical status I, without eye disease, scheduled for elective non-ophthalmic surgery were selected for study. They were randomly assigned to control (Group A, n = 20) or nifedipine groups (Group B, n = 20) with random number charts. Patients with preexisting elevated IOP (glaucoma), hypertension, obesity, neuromuscular or intracranial diseases were not included in the study. The procedure for measurement of IOP was explained in detail and informed consent obtained. All the patients were premedicated with meperidine 1 $mg \cdot kg^{-1}$ and promethazine 0.5 $mg \cdot kg^{-1}$ IM one hour before induction of anaesthesia. Twenty minutes before the induction of anaesthesia the patients were given either a 10 mg nifedipine gelatine capsule or a placebo capsule of similar physical characteristics sublingually by the third author who was not involved in the rest of the study.

Anaesthesia was induced in all patients with a sleep dose of thiopentone (4–5.5 $mg \cdot kg^{-1}$) given slowly over a period of 40–50 seconds to obtund the eye lash reflex, followed by succinylcholine 1.5 $mg \cdot kg^{-1}$. Laryngoscopy was attempted one minute after succinycholine administration and intubation completed within 30 seconds in all the patients. Any patient in whom tracheal intubation proved difficult or who strained or coughed during intubation was excluded from the study. Meanwhile, ventilation was assisted/controlled with a mask, using a Bain anaesthetic breathing circuit delivering 70 per cent nitrous oxide in oxygen at a total fresh gas flow of 100 $ml \cdot kg^{-1}$ body weight.[11]

After tracheal intubation, ventilation was controlled with 70 per cent nitrous oxide in oxygen with a Servo

TABLE Demographic data

	Age (year)	*Weight (kg)*	*Sex (M/F)*	*IOP (mmHg)*	*SBP (mmHg)*
Group A n = 20	34.6 ± 3.9	64.9 ± 4.2	12/8	18.53 ± 1.84	121.8 ± 10.32
Group B n=20	36.3 ± 3.6	65.9 ± 5.4	13/7	17.48 ± 1.67	119.4 ± 11.9

All values mean ± SD.

ventilator (Siemens Elema 900 B) at a respiratory rate of 12 breaths · min^{-1} with $\dot{V}E$ approximating 110 ml · kg^{-1}. Systolic blood pressure, heart rate and IOP were measured at the following times:

A Before nifedipine pretreatment (control value).
B Twenty minutes after pretreatment, i.e., just before induction of anaesthesia.
C After injection of thiopentone.
D After succinylcholine administration (immediately after fasciculations had ceased).
E,F,G At one, two and three minutes respectively following intubation.

Heart rate and arterial pressure were recorded using an automatic monitor (Nippon Colin Co., Japan). All measurements of IOP were made with a Schiotz tonometer[9,12] (technique accurate to within ±2 mmHg) by the first author who was unaware of the nature of the sublingual capsule. Readings were taken in both eyes following topical administration of four per cent lidocaine. The mean of the two readings was recorded. Two-way analysis of variance was used to analyse changes within the groups and unpaired t test for analysis between the groups. $P < 0.05$ was considered to indicate statistical significance. The correlation cofficient was found for SBP and IOP.

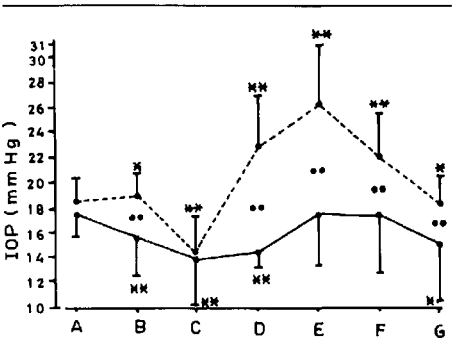

FIGURE 1 Intraocular pressure (IOP) in control (----) and nifedipine (———) treated patients at various time intervals. A – before nifedipine pre-treatment (control value). B – twenty minutes after pre-treatment, i.e., just before induction of anaesthesia. C – After injection of thiopentone. D – After succinylcholine administration. E,F and G – one, two and three minutes after tracheal intubation. Within group comparison *P < 0.05, **P < 0.01; Between group comparison 'P < 0.05, ''P < 0.01.

Results

The patients in both the groups were comparable with regard to age, body weight, resting IOP and systolic blood pressure (Table). The change in IOP and SBP in both the groups during the induction sequence are presented in Figure 1 and Figure 2 respectively. There was a significant decrease in IOP after pretreatment with nifedipine in Group B ($P < 0.05$). In both groups IOP decreased after administration of thiopentone. The increase in IOP following succinylcholine administration and tracheal intubation was significantly less in Group B than in Group A ($P < 0.01$). The mean maximum rise in IOP above basal level at one minute post-intubation was 7.82 mmHg in Group A compared with 0.15 mmHg in the nifedipine pretreated patients in Group B (Figure 1). The maximum increase in IOP at one minute post-intubation in Group A and B patients was 14.80 mmHg and 6.40 mmHg respectively. Only one patient in group B experienced an increase in IOP greater than 5 mmHg. The increase in SBP above the basal level in group B patients one minute after tracheal intubation was 8.55 mmHg compared with 21.7 mmHg in control patients. Although SBP and IOP values at each time interval were not constantly correlated, the combined correlation coefficient between all values of SBP and IOP for Group A as well as Group B showed significant correlation ($r = 0.49$, $P < 0.01$ for group A; $r = 0.52$, $P < 0.01$ for group B).

Discussion

Several studies have demonstrated that succinylcholine administration causes an increase in IOP. To avoid the use of succinylcholine, pancuronium and vecuronium have been used for rapid intubation in patients in whom an increase in IOP is undesirable.[13,14] However, the search for methods to allow the safe use of succinylcholine in

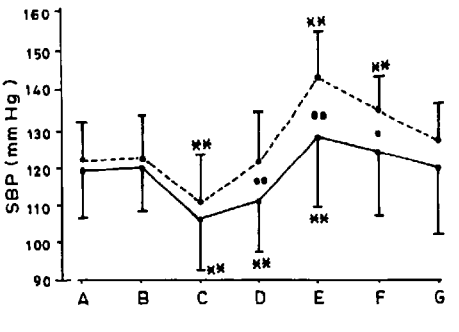

FIGURE 2 Systolic blood pressure (SBP) in control (----) and nifedipine (———) treated patients at various time intervals. For symbols see Figure 1.

patients with penetrating eye injuries continues because succinylcholine remains the drug of choice to provide good conditions for rapid intubation.[15,16] Pretreatment with a nondepolarizing neuromuscular relaxant,[4] diazepam[5] and lidocaine[8] have been suggested to prevent succinylcholine induced increase in IOP, although others have found these measures to be ineffective.[6–8,17]

Nifedipine, a dihydropyridine, is well absorbed by oral or sublingual routes. After a single SL administration of 10 mg nifedipine, an antihypertensive action is obvious at 20–30 min.[18,19] A significant decrease in IOP was found in nifedipine-pretreated patients before induction of anaesthesia. Nifedipine further prevented the increase in IOP after succinylcholine administration, laryngoscopy and intubation. This effect might be due to obtunded haemodynamic response as SBP in nifedipine-treated patients was significantly less following laryngoscopy and intubation and there was significant correlation between IOP and SBP values of each group. In addition there may be some direct effect of nifedipine on aqueous flow dynamics as IOP decreased below the basal level in patients pretreated with nifedipine without any significant fall in SBP before the induction of anaesthesia.

Other factors such as CVP or $PaCO_2$ could have affected the results.[20,21] However, we do not expect any change in CVP as all the patients were kept horizontal throughout the period of study. We did not monitor $PaCO_2$, but again we do not expect any significant change in $PaCO_2$ during this short period of study as all the patients were ventilated adequately before and after tracheal intubation with Bain circuit[11] and Servo ventilator respectively.

In conclusion sublingual nifedipine when employed as pretreatment significantly attenuates the IOP response that follows succinylcholine administration and tracheal intubation in young patients. However, it will be interesting to study the drug in older age groups and in patients with increased IOP coming for surgery.

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Résumé

Nous avons évalué le rôle de la nifédipine sub-linguale dans la prévention de l'augmentation de la pression intra-oculaire suivant l'injection de succinylcholine et l'intubation chez 40 patients aux yeux normaux. Le hasard déterminait s'ils allaient recevoir 10 mg de nifédipine ou un placebo 20 minutes avant l'induction de leur anesthésie pour chirurgie non-ophtalmique. Nous mesurions les pressions intra-oculaire (PIO) et artérielle systolique (PAS) avant et après cette induction. L'augmentation maximale moyenne de la PIO une minute après l'intubation a été de 7.82 mmHg dans le groupe placebo et de 0.15 mmHg dans le groupe nifédipine ($P < 0.01$). Donc, la nifédipine sublinguale semble capable de limiter l'augmentation de la PIO suivant la séquence succinylcholine-laryngoscopie-intubation trachéale.