

Effects of acepromazine on intraocular pressure, anterior chamber depth in horses: A prospective randomized controlled study

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Abstract

This study aimed to evaluate the effects of acepromazine on intraocular pressure (IOP), anterior chamber depth (ACD), cardiovascular and respiratory rates in horses. In a prospective, randomized, controlled trial, twelve horses were equally allocated randomly into two groups: treatment and control (n = 6 each). Horses of the treatment group received 0.1 mg/kg body weight (bwt) of acepromazine intravenously, while those in the control group received 0.4 mL/100 kg bwt of 0.9% sodium chloride intravenously. IOP, ACD, heart rate, and respiratory rate were recorded before (T0) and then at T10, T20, T30, T40, T50, T60, T70, T80, and T120 min post-treatment. Acepromazine induced a significant drop in IOP and ACD ($P < 0.05$) between T10 and T40 in comparison with the saline group. Regarding heart rate, the acepromazine group's heart rate noticeably increased ($P < 0.05$) from T10 to T150, while their respiratory rate significantly decreased when compared to the saline group ($P < 0.05$). In conclusion, with proper monitoring of the horse's respiratory and cardiac functions, acepromazine can be used as a sedative drug for intraocular surgery. The findings of this study on healthy horses may not be extrapolatable to findings in those with ophthalmic lesions. Therefore, further studies with larger populations, different dosage regimens, and longer monitoring intervals are recommended for horses with ophthalmic diseases to better understand the safety profile of the drug.

Keywords: acepromazine, cardiorespiratory functions, equine, IOP, normal saline

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Introduction

Intraocular pressure (IOP) refers to the pressure exerted by the aqueous humor on the inner surface of the anterior portion of the eye (Jang *et al.*, 2021). Aqueous humor is produced by the ciliary body, flows through the pupil, and is ultimately drained from the eye via the uveoscleral or iridocorneal outflow pathways (Tolar and Labelle, 2013). The autonomic nerve systems of the sympathetic and parasympathetic branches maintain a balanced production of aqueous fluid and control intraocular pressure by activating the trabecular meshwork and ciliary body receptors with a variety of neurotransmitters, including serotonin, dopamine, acetylcholine, adrenaline, and noradrenaline (Souza *et al.*, 2008; Glare *et al.*, 2011; Jang *et al.*, 2021). An imbalance between the production and flow of aqueous humor can lead to increased IOP, damaging retinal ganglion cells and the optic nerve head, ultimately resulting in blindness (Brath *et al.*, 2000). Both rebound and applanation tonometers are used for measuring IOP in horses (Knollinger *et al.*, 2005; Andrade *et al.*, 2011). A magnetic probe is employed by the rebound tonometer to gently touch the cornea and measure its deceleration at the point of contact. This portable, battery-operated device does not require daily calibration. In contrast, applanation tonometers assess the pressure required to smooth the corneal surface (Tolar and Labelle, 2013).

In human medicine, the ocular geometric parameters, including central corneal thickness, corneal curvature, anterior chamber depth (ACD), lens thickness, and axial length, are utilized to diagnose a variety of ocular diseases (Hoffmann *et al.*, 2022; Bian *et al.*, 2024). The effect of intraocular pressure on eye geometry is the scope of many studies in humans (Tatewaki *et al.*, 2019) and in poultry (Genest *et al.*, 2012). ACD represents the distance between the corneal endothelium and the anterior capsule of the crystalline lens (Hoffer, 2011). Several methods can measure ACD, including ultrasonography in both humans (Hassen *et al.*, 2014), and horses (Badial *et al.*, 2015), while partial coherence interferometry (Findl *et al.*, 1998), and ocular magnetic resonance imaging (Klaassen *et al.*, 2024) are used in humans.

Understanding sedation, several ocular surgeries, and comprehensive ophthalmic examinations can be performed in horses (Robertson, 2004). Nonetheless, certain sedative medications may have a negative impact on IOP (Trim *et al.*, 1985). The choice of sedative medication for the intraocular surgery is therefore crucial to the success of the procedure (Marzok *et al.*, 2014b). In some animal species, apnea and circulatory changes, such as arrhythmia and either hypertension or hypotension, have also been linked to several sedative drugs (Edwards and Brown, 1996; Lumb *et al.*, 2007; Hubbell, 2009; Peterson and Kutzler, 2010; Bian *et al.*, 2024;). The management of these side effects presents a significant challenge, emphasizing the necessity for sedatives that have little effect on the vital systems to ensure the animals' safety.

Acepromazine is a neuroleptic from the phenothiazine family that is widely used to induce sedation (Peterson and Kutzler, 2010). Acepromazine's sedative action is mediated by dopamine D2 receptor

blocking in the brain's dopaminergic pathways, which results in central depression (Edwards and Brown, 1996). Furthermore, acepromazine inhibits D₂ receptors in the medulla oblongata's chemoreceptor trigger zone, producing antiemetic effects (Hendren *et al.*, 2015). It also antagonizes dopamine's action at peripheral D₁ and D₂ receptors (Edwards and Brown, 1996). Additionally, acepromazine inhibits several other receptors, including cholinergic, histaminergic, and adrenergic receptors, causing vasodilation by preventing norepinephrine from binding to α -adrenergic receptors (Peterson and Kutzler, 2010).

However, acepromazine does not have analgesic properties; it can improve the pain-relieving effects of opioids and α_2 agonists (Hubbell, 2009). In addition to its sedative effects, acepromazine also possesses anticonvulsant, antispasmodic, and hypothermic properties (Lumb *et al.*, 2007). Acepromazine's maximal effect is typically attained rapidly after intravenous injection and can persist for several hours (Seddighi and Doherty, 2016). The effect of acepromazine on IOP remains uncertain due to conflicting results from different studies (Micieli *et al.*, 2018), which found that acepromazine sedation in dogs increased IOP. In contrast, Mrazova *et al.* (2018) observed no significant changes in IOP in dogs. Before administering any medication therapeutically, it is crucial to thoroughly assess its effects on each specific species, as extrapolating data from one species to another can be unreliable and potentially hazardous. Thus, the current investigation aims to evaluate the impact of acepromazine on intraocular pressure and cardiorespiratory variables in horses in a prospective randomized controlled study.

Materials and Methods

Horses: A total of twelve clinically healthy horses were enrolled for this study. The age of horses ranged from 6 to 9 years, and the weight of 350-420 kg. Horses were proven to be healthy based on the clinical examination and hematological examination (Constable *et al.*, 2016). Two weeks before starting the experiment, each horse was kept in a stable with straw-bedded boxes and fed on standard diets twice daily with ad libitum water access. This study was carried out at Police Heights Equine Hospital Club, Cairo, Egypt. The study was approved by the Animal Welfare and Ethics Committee, Faculty of Veterinary Medicine, Benha University (BUFVTM 04-04-23).

Study design: A randomized controlled trial (RCT) incorporated two treatment groups (6 each): the placebo (normal saline, NaCl 0.9%; El-Nasr Pharmaceutical Chemicals Co, Egypt) and the acepromazine at a dose rate of 0.1 mg/kg (Prozyl fort). Acepromazine was used as recommended by the manufacturer before treatment. Random allocation of groups was done using block randomization, which ensures balance between groups. In the block randomization, participants were divided into "blocks" and then randomly assigned to treatments within each block. In the present study, prior power analysis was not performed to assess the sample size. Therefore, post hoc power analysis was performed.

Post hoc power analysis was conducted using the observed effect size, sample size, and $\alpha = 0.05$ to estimate the achieved statistical power (O'Keefe, 2007). Upper eyelid lashes were clipped with scissors. Intraocular pressure was measured at T0 time just prior to medication administration as well as at 10, 20, 30, 40, 50, 60, 70, 80, and 120 min after administration. Evidence of clinical adverse effects was observed and documented simultaneously. For all the following investigations, the investigators remained blinded to treatment allocation throughout the trial to minimize observer bias and ensure objective outcome assessment.

Measurement of IOP: IOP was measured using rebound tonometry (Tono Vet-I care), with the equine's head held in a neutral position at heart level for each tonometry reading (Marzok *et al.*, 2014a). A light touch was applied to the cornea's center portion to take three measurements at 5-second intervals. The data were then averaged. Each animal received a new probe for the tonometer. Measurement of IOP was obtained by the same investigator in a quick and stress-free manner.

Ultrasonographic examination: Ultrasonographic examination was carried out via an Esaote ultrasound system with a multi-frequency linear transducer (3-10MHz). Transcorneal ultrasonography was performed, and images of the anterior chamber depth were obtained. The probe was oriented perpendicular to the limbus and placed perpendicular to the globe such that the corneal epithelium, Descemet's membrane, and anterior lens capsule were of similar echogenicity, ensuring a non-oblique image. Gel was applied to the probe, and the tip was touched to the surface of the central cornea, ensuring optimal contact and minimal pressure to the globe. The intraglobular changes were assessed at 0, 10, 20, 30, 40, 50, 60, 70, 80, and 120 min for all animals in both groups.

Heart and respiratory rates: Heart rate and respiratory rate were measured for each horse before treatment, at 10, 20, 30, 40, 50, 60, 70, 80, and 120 min post-treatment (Love *et al.*, 2012).

Statistical analysis: Data were analyzed using a commercial software program (GraphPad Prism Ver 8.0.2, USA). Firstly, Homogeneity between the two groups was assessed by using the Kruskal-Wallis test, and the normality distribution of the data was assessed by using the Shapiro-Wilk test. The two groups of horses were found homogeneous, and the data were found normally distributed. Secondly, data of IOP, ACD, heart rate, and respiratory rate were subjected to the general linear model with repeated measures ANOVA. Wilks' Lambda test was used to explore the effect of treatment, time, and treatment x time interaction. Whenever Wilks' Lambda test was found significant, the t-test at each time point was conducted to explore which group was varying. For exploration of within-subject (time) variation, ANOVA with post hoc Tukey multiple comparison test was used. Differences at ($P < 0.05$) were considered significant in all statistical analyses.

Result

This study investigated the IOP, ACD, and cardiorespiratory parameters in horses following acepromazine and 0.9% sodium chloride injections intravenously. The baseline IOP, ACD, respiratory rate, and heart rate showed non-significant differences between the two groups at T0. Whereas, compared with the control group, the acepromazine-treated group demonstrated a significant reduction in IOP at T10 to T40. The maximum decrease in IOP was observed at T20 min after treatment (Fig. 1).

There was a significant mild effect of acepromazine on ACD in comparison with placebo. ACD decreased significantly at T10, T20, T30, and T40 post-treatment (Fig. 2). The results also showed non-significant differences between the right and left eyes of the examined horses.

Regarding the respiratory rate, the study revealed a significant decrease in the acepromazine group compared to the control group from T10 to T50 post-treatment. The most substantial decrease in the respiratory rate was observed at T40 min post-acepromazine treatment (Fig. 3). In contrast, the heart rate was significantly increased in the acepromazine group compared to the control group from T10 to T50. The most significant increase in heart rate was observed at T40 post-treatment (Fig. 4).

Discussion

Many diagnostic and minor surgical operations were performed under standing sedation due to the considerable mortality and morbidity risks associated with general anesthesia in horses (Johnston *et al.*, 2004). In case of ophthalmic procedures, achieving favorable outcomes may depend on the management of IOP prior to, during, and following the procedure (Aarnes and Muir, 2011). High IOP can induce retinal cell death, optic nerve compression, and eventually blindness because it restricts blood flow and partially obstructs axoplasmic transport in the optic nerve (Brooks *et al.*, 1995). Several animal species can be sedated and tranquilized using acepromazine, with different intravenous dose recommendations.

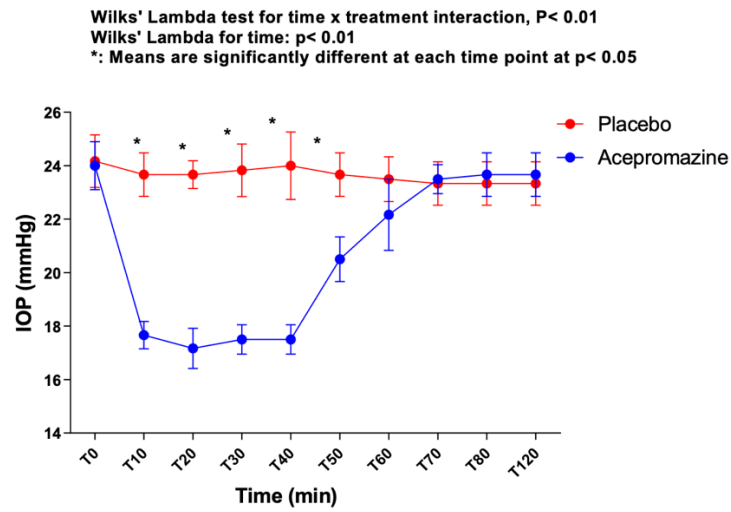


Figure 1 Mean intraocular pressure (IOP) in horses following intravenous administration of acepromazine (0.1 mg/kg) or saline (0.9% NaCl). Acepromazine induced a significant reduction in IOP between 10 and 40 min, with the maximum decrease at 20 min.

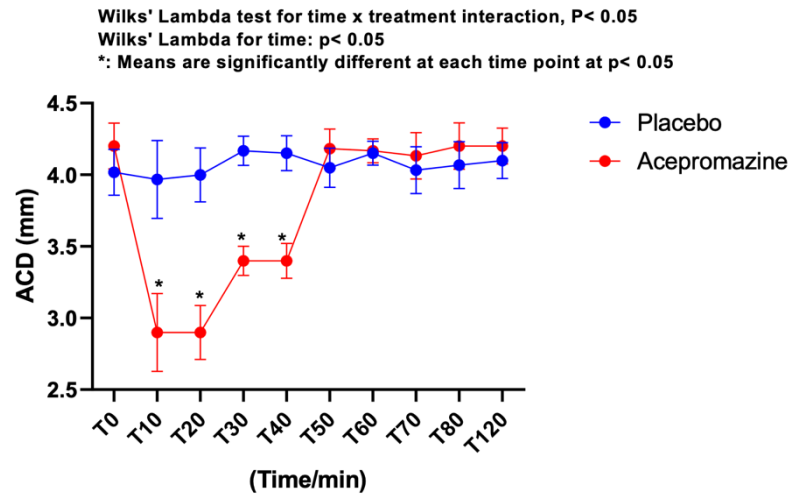


Figure 2 Anterior chamber depth (ACD) in horses after intravenous administration of acepromazine (0.1 mg/kg) or saline (0.9% NaCl). Acepromazine induced a significant reduction in ACD from 10 to 40 min post-administration compared with controls.

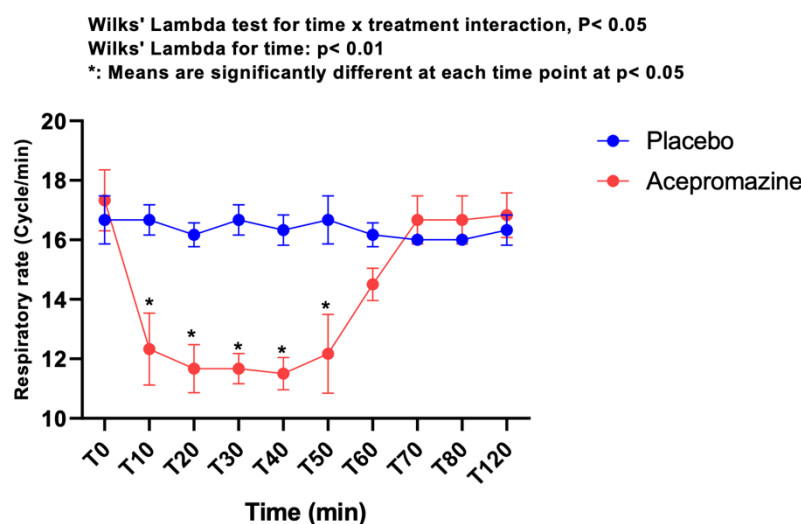


Figure 3 Respiratory rate in horses following intravenous administration of acepromazine (0.1 mg/kg) or saline (0.9% NaCl). Acepromazine significantly decreased respiratory rate between 10 and 50 min, with the lowest values observed at 40 min post-administration.

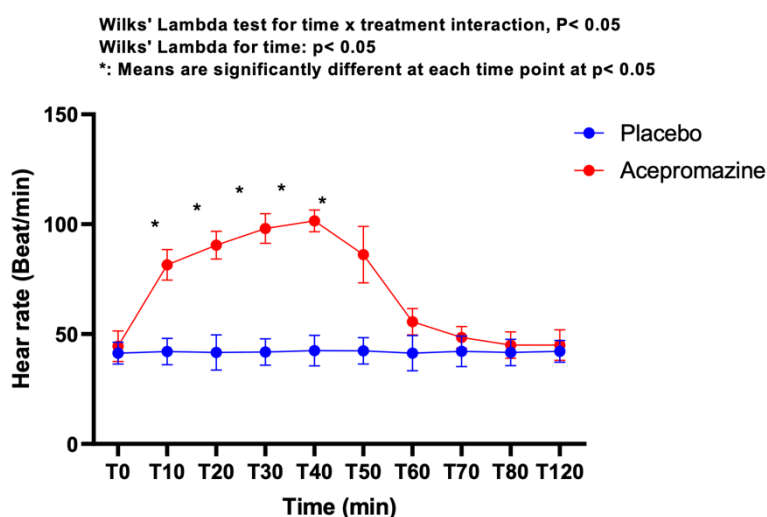


Figure 4 Heart rate in horses after intravenous administration of acepromazine (0.1 mg/kg) or saline (0.9% NaCl). Acepromazine significantly increased heart rate between 10 and 50 min, peaking at 40 min post-treatment.

In the present experiment, the significant decrease in IOP after the intravenous administration of acepromazine coincides with the results of a previous study on acepromazine or a combination of acepromazine with other anesthetics (McClure *et al.*, 1976; Kanay *et al.*, 2025). Similarly, in dogs, administering 0.01 mg/kg of acepromazine intramuscularly lowers IOP during the first 25 min and then again within the next 35 to 60 min post-administration (Kovačička and Birgele, 2009). On the contrary, minimal changes in IOP among healthy dogs receiving intravenous acepromazine at a dose of 0.02 mg/kg were recorded (Mrazova *et al.*, 2018). The exact mechanism by which acepromazine decreases IOP isn't fully understood; acepromazine's IOP-lowering effect in horses may involve its action as a dopamine antagonist, leading to reduced aqueous humor production (Green and Elijah, 1981; Magacho *et al.*, 2004). In addition, acepromazine is an $\alpha 1$ -adrenergic antagonist causing miosis, which improves aqueous humor outflow and lowers IOP, as reported in dogs (Green and Elijah, 1981; Kovačička and Birgele, 2009; Santos *et al.*, 2013).

The considerable decrease in ACD after acepromazine injection seen in this research might be ascribed to lower IOP. The relationship between IOP and ACD is controversial. In human medicine, a significant increase in ACD was reported in patients suffering from increased IOP (Hassen *et al.*, 2014). Additionally, in a study carried out on 3060 participants in Japan, a positive correlation was reported between increased IOP and ACD (Inooka *et al.*, 2024). However, other studies reported a non-significant relationship between the IOP and ACD. Among these investigations, a non-significant association was noted between IOP and ACD in patients with primary open-angle glaucoma (Adewara *et al.*, 2018). Furthermore, Hoffmann *et al.* indicated that ACD did not correlate with increased IOP in the human phakic eyes (Hoffmann *et al.*, 2022). Therefore, the considerable relationship between ACD and IOP after acepromazine injection observed in the present study remains hypothetical, as previous reports in human medicine have shown conflicting results.

The significant decrease in the respiratory rate observed in the acepromazine-treated group in this

study is in accordance with that previously reported in horses (Ballard *et al.*, 1982), camels (Kandeel *et al.*, 2021), and dogs (Santos *et al.*, 2013). Contradictory, insignificant changes in the respiratory rate in buffalo and calves following the administration of acepromazine via intramuscular injection (Bian *et al.*, 2024). This decrease in respiratory rate may be attributed to the diminished sensitivity of chemoreceptors to carbon dioxide induced by acepromazine (Coulter *et al.*, 1981b; Kovaččuka and Birgele, 2009).

The acepromazine-induced tachycardia reported in this study aligns with the findings of a previous report in horses after oral acepromazine administration (Hashem and Keller, 1993). In a study in horses, it was reported that the changes in heart rate after the intravenous, oral, or sublingual administration of acepromazine were a minimal significant increase relative to baseline that occurred about 2.5 h after drug administration (Knych *et al.*, 2018). They attributed this increase to external factors other than acepromazine. Also, a non-significant increase in the heart rate was recorded after administration of acepromazine at a dose rate of 0.1, 0.2, and 0.3 mg/kg in horses (Pequito, 2015). On the other hand, it was found that dogs' and horses' heart rates barely altered when acepromazine was administered intravenously at a dose rate of 0.1 mg/kg (Muir, 2000). Additional study on horses revealed that acepromazine at 0.1 mg/kg induced varying effects on the heart rate (Buhl *et al.*, 2007). However, a large intravenous dose of acepromazine (0.3mg/kg) could induce significant echocardiographic changes in the same species (Menzies-Gow, 2008). The mechanisms by which acepromazine could reduce blood pressure include blocking peripheral α_1 -adrenergic receptors (Santos *et al.*, 2013; Monteiro *et al.*, 2016) or direct action on the myocardium, or by suppressing the central nervous system (Knych *et al.*, 2018).

The limitations of the present study should be explored. First, the study evaluated only one intravenous dose of acepromazine (0.1 mg/kg). Different dosages or alternative routes of administration may produce varying effects on intraocular pressure (IOP), anterior chamber depth (ACD), and cardiorespiratory parameters (Knych *et al.*, 2018). However, the selected dose is the most commonly used dose of acepromazine in horses. Second, the study has a limited clinical context, where healthy horses were only included. Therefore, the result may not be consistent with clinical ophthalmology, and hence further study, including horses with ocular disease and systemic illness, should be conducted. Third, the relatively small sample size ($n = 12$, 6 per group) may restrict the generalizability of the findings. This constraint was largely due to ethical and logistical considerations in equine experimentation. Despite this, the randomized controlled design, homogeneity of groups at baseline, and repeated measures ANOVA revealed statistically significant differences in intraocular pressure (IOP), anterior chamber depth (ACD), heart rate, and respiratory rate between acepromazine and saline groups ($P < 0.05$). Post hoc power analysis based on the observed effect sizes demonstrated that the reduction

in IOP at T20 achieved a power exceeding 80%, suggesting adequate sensitivity to detect clinically relevant differences in this primary outcome. In addition, previous research on acepromazine could obtain reliable results by using a small sample size (Ghaffari and Moghaddassi, 2010; Micieli *et al.*, 2018). Nevertheless, smaller effects on cardiorespiratory parameters may require larger cohorts to confirm reliability. Fourth, we assessed only heart rate, which may not prove changes in cardiac function after administration of acepromazine. Nonetheless, we used a dose rate of 0.1 mg/kg B.W., which was confirmed to cause non-significant echocardiographic alterations (Pequito, 2015).

In conclusion, this prospective randomized controlled study demonstrated that intravenous administration of acepromazine at 0.1 mg/kg significantly reduced intraocular pressure and anterior chamber depth in horses, while also inducing a decrease in respiratory rate and an increase in heart rate compared with saline controls. These findings suggest that acepromazine can be a suitable sedative for ocular examinations and surgeries in horses, particularly when lowering intraocular pressure is desirable.

The findings of this study on healthy horses may not be extrapolatable to findings in those with ophthalmic lesions. Therefore, further studies with larger populations, different dosage regimens, and longer monitoring intervals are recommended for horses with ophthalmic diseases to better understand the safety profile of the drug.

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Data Availability: Data are available on request

Statement of author contributions: **Abdelhaleem Elkasapy:** Conceptualization, Methodology, Visualization, Data curation, Writing—original draft. **Magdy Elgiouhy:** Methodology, Formal analysis, Investigation, Writing—original draft. **Mohamed Marzok:** Conceptualization, Methodology, Supervision, Funding acquisition, Writing—review & editing. **Sayed Elhawari:** Software, Data curation, Investigation, Visualization. **Adel Almubarak:** Software, Formal analysis, Visualization, Supervision. **Mohamed Rizk:** Software, Data curation, Investigation, Visualization. **Sabry El-khodery:** Formal analysis, Writing—review & editing, Supervision. **Adel Al-Akraa:** Writing—review & editing. All authors have read and agreed to the published version of the manuscript.

Declaration of artificial intelligence use: During the preparation of this work, the author(s) used the

Microsoft Copilot AI agent for rephrasing some sentences.

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