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Effect of peribulbar anesthesia on intraocular pressure and ocular pulse amplitude

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Abstract

• AIM: To investigate the effect of peribulbar anesthesia on intraocular pressure (IOP) and ocular amplitude pulse (OPA).

• METHODS: Thirty-two consecutive adult patients with monocular cataract enrolled in this study. IOP and OPA were measured with dynamic contour tonometer (DCT) before and 3, 10 minutes after administration of lidocaine anesthesia. Data were analyzed with software SPSS 11.5.

• RESULTS: The IOP remained stable in the injected eyes and the non-injected eyes after administration of lidocaine anesthesia. The OPA was significantly decreased after injection of anesthesia agent in the injected eyes. The OPA in the non-injected eyes increased significantly 3 minutes after injection of the anesthesia agent, returning to preinjection level 10 minutes after the injection.

• CONCLUSION: Peribulbar anesthesia leads to decrease of OPA and shows no effect on IOP in the injected eyes.

• KEYWORDS: peribulbar anesthesia; intraocular pressure; ocular pulse amplitude
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INTRODUCTION

Most routine ophthalmologic surgeries are now performed using local anesthesia, including retrobulbar, peribulbar, and sub-Tenon's, which is less dependent on patient health status and entails fewer risks. In current view, the optimal mode for obtaining local anesthesia has yet to be determined.

The decreasing use of retrobulbar anesthesia can be partly attributed to the risk of serious complications such as retrobulbar hemorrhage, globe perforation, optic nerve injury and so on[1-3]. However, many of these complications are not eliminated by peribulbar anesthesia. A further drawback is that peribulbar anesthesia tends to produce a reduction in ocular blood flow that may lead to sight-threatening complication such as retinal and choroidal vascular occlusion and optic atrophy.

In this study, we analyzed our clinical patient database to evaluate the effect of peribulbar anesthesia on IOP and OPA.

SUBJECTS AND METHODS

Subjects Thirty-two patients who admitted to our cataract center participated in this study. Measurements were performed in the operated eye and the contralateral eye as control. The mean age of the twenty men (62.5%) and twelve women(37.5%) was 64.48 ± 5.72(ranged from 45 to 78) years. All patients received peribulbar anesthesia when operated. The eligible criteria used in this study were no significant cardiovascular disease, uncontrolled intraocular disease, arterial hypertension, primary or secondary glaucoma, carotid artery stenosis and those treated with drugs that influence ocular blood flow.

Peribulbar anesthesia The injection was performed at the inferotemporal site using a 5.0mm 25 gauge sharp needle with the eye in primary gaze. The needle was inserted at the junction of the lateral and medial third of the inferior orbital rim parallel to the floor of the orbital and tangential to the globe. After negative aspiration for blood, 2mL of lidocaine 2% without epinephrine was injected at the depth of 16.0mm. The same surgeon performed all injections.

Measurements of IOP and OPA The IOP and OPA measurements were made immediately with a dynamic contour tonometer before and 3 and 10 minutes after administration of the anesthesia agent. One observer did all measurements.

Statistical Analysis Results are presented as mean and standard deviation. Comparisons between groups and between variables were made using paired and unpaired t test. A P value less than 0.05 was considered statistically significant.

RESULTS

As shown in Table 1, there was no significant difference in IOP at baseline in both the injected and the control eyes($P > 0.05$). The IOP did not change significantly from baseline to 3 and 10 minutes after injection in the injected or the control eyes($P > 0.05$).

Table 1 Effect of anesthesia on IOP and OPA

Group	Time	IOP(mmHg)	OPA
Injected	Baseline	17.6±1.5	2.6±0.2
	3 minutes postinjection	18.2±1.1	2.0±0.1
	10 minutes postinjection	18.2±1.0	1.9±0.2
Control	Baseline	17.2±1.3	2.6±0.2
	3 minutes postinjection	16.8±1.1	2.8±0.2
	10 minutes postinjection	18.2±1.0	2.6±0.2

The peribulbar injection induced a significant decrease in OPA in the injected eyes 3 and 10 minutes after the administration of the anesthesia agent. The OPA in the control eyes showed a increase 3 minutes after injection; the OPA returned to preinjection levels after 10 minutes.

DISCUSSION

The total blood flow to the eye ball consists of pulsatile and nonpulsatile components as shown by Doppler ultrasound studies of the ophthalmic artery[4]. It is not known what contributions they make to the total ocular blood flow. Animal studies show that pulsatile perfusion maintain better capillary and venous perfusion than nonpulsatile blood flow[5]. This suggests that the pulsatile flow has an important role in ocular perfusion and that any decrease in this component might lead to functional and morphological damage to the eye. The ocular pulse amplitude (OPA) is determined by net intraocular volume changes produced by the inflow of a bolus of blood entering the eye during cardiac systole and the simultaneous out flow through the venous channels. It equals to the difference value of IOP between cardiac systole and cardiac diastole. The OPA mirrors the choroid perfusion indirectly and the ocular blood flow corresponding to a single cardiac impulse. Decrease of blood flow may lead to hypoxia and apoptosis resulting in glaucoma and other disease with circulatory disturbance. It is believed that the OPA plays an important role in the process of glaucoma[6,7].

Given that local anesthesia is thought to reduce ocular blood flow, leading to sight-threatening complications, the OPA may provide valuable information on the vascular component in the pathogenesis of theses complications. Our study demonstrates the changes in ocular hemodynamic after peribulbar anesthesia. The OPA was significantly reduced in the eyes injected with lidocaine, while in the fellow eyes, peribulbar anesthesia induced an increase in OPA as early as 3 minutes after anesthesia; the OPA returned to preinjection levels 10 minutes later. Our study confirms earlier observations in animal and humans that retrobulbar anesthesia and peribulbar anesthesia with lidocaine reduce the ocular pulse volume[8,9].

The mechanism of postinjection ocular hemodynamic changes is unknown. The decline in OPA after peribulbar anesthesia might be caused by an increase in IOP resulting from mechanical compression of the eye and blood vessels by the large volume of solution injected in the small orbital space. However, this does not explain the findings in our study. We found no significant change in IOP in either the injected or fellow eyes. These results contradict the findings of other studies, which observed a greater rise in IOP with peribulbar anesthesia[10-12]. We believe the difference in results is because we used a smaller injection volume(2mL) than other studies used 7 to 12mL).

The changes we observed in OPA in both the injected eyes and fellow eyes suggest that the anesthetic drug rather than the anesthetic technique was the trigger for hemodynamic changes; we ascribe the hemodynamic changes to the drug's pharmacological action. The effect of lidocaine is unexpected because the drug is known to cause vasodilatation and thus should have provoked an increase in local blood circulation. A possible explanation for the decrease in blood flow after lidocaine injection could be paradoxical vasoconstriction caused by the high local concentration of the drug. High concentrations of lidocaine produced pulmonary vasoconstriction[13] and a reduction in renal and hepatic blood flow[14] in animal models. The variability of the hemodynamic changes in the injected and the fellow eyes can be attributed to the difference in the local concentration of the medication. With peribulbar anesthesia, the influence of lidocaine is noticed as soon as 3 minutes after injection. Because only a small amount of lidocaine was absorbed this time, the low concentration that reached the fellow eye caused vasodilatation and increased OPA. The OPA returned to the preinjection levels 10 minutes after injection because of the dissipation of lidocaine.

Our results indicate that the OPA decreases after the administration of peribulbar anesthesia. We presume that this effect is related to the use of lidocaine as the anesthetic medication. Our results are based on the use of an injection volume of 2mL, because this is a relatively small amount, it can be assumed that large volumes induce a greater postinjection decrease in ocular blood flow. It indicates that we should be cautious to choose other modes of anesthesia when considering surgery in patients whose ocular circulation may be compromised.

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球周麻醉对眼压及眼脉动振幅的影响

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摘要

目的:探讨球周麻醉对眼压(IOP)及眼脉动振幅(OPA)的影响。

方法:随机选择单眼白内障患者 32 例,患眼施行球周麻醉,另眼设为对照,分别于麻醉前及麻醉后 3,10min 采用动态轮廓眼压计测量注射眼和对照眼的 IOP 及 OPA 值,对数据进行统计学分析。

结果:注射利多卡因后注射眼和对照眼的眼压均无明显变化。注射眼 OPA 值显著降低,而对照眼在注射后 3min OPA 值升高,10min 后恢复至注射前水平。

结论:球周麻醉可导致眼脉动振幅降低,而对眼压没有影响。

关键词:环周麻醉;眼压;眼脉动振幅