• Basic Research •

Rebalancing translaminar pressure difference: a novel glaucoma surgery—a pilot trial in non-human primates

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Received: 2025-04-06 Accepted: 2025-07-04

Abstract

● **AIM:** To propose a novel glaucoma surgery for rebalancing translaminar pressure difference.

● **METHODS:** Three non-human primates with normal eyes and two with laser-induced glaucoma underwent the novel surgical procedure. Cannulation of the subarachnoid space was performed after completion of routine vitrectomy steps. An XEN 45 implant was inserted into the created puncture to communicate between the vitreous body and subarachnoid space. Intraocular pressure (IOP), fundus photography, and spectral-domain optical coherence tomography were assessed at baseline and regular intervals during follow-up.

● **RESULTS:** All operated eyes showed IOP reduction in the first postoperative month. Two (2/3) normal eyes and one (1/2) glaucomatous eye maintained lower IOP until 18mo after operation. The XEN 45 implant remained positioned through the lamina cribrosa in all normal eyes but was not detected in two glaucomatous eyes. Complications observed in this study included retinal vascular bleeding in 1/3 normal eyes and XEN implant dislocation in all 2 glaucomatous eyes.

● **CONCLUSION:** Subarachnoid space cannulation and mini-shunt implantation may contribute to IOP reduction, possibly by rebalancing translaminar pressure difference and enhancing aqueous humor drainage. The development of a suitable mini-shunt requires further investigation.

● **KEYWORDS:** translaminar pressure difference; glaucoma surgery; lamina cribrosa; intraocular pressure

DOI:10.18240/ijo.2025.09.02

Citation: Xin C, Sang Q, Wu S, Pang RQ, Lin DT, Wang HZ, Lu N, Wang NL. Rebalancing translaminar pressure difference: a novel glaucoma surgery—a pilot trial in non-human primates. *Int J Ophthalmol* 2025;18(9):1619-1625

INTRODUCTION

Glaucoma is the leading cause of irreversible blindness worldwide[1]. Primary open angle glaucoma (POAG) is the most common type characterized by structural changes in the optic nerve and corresponding visual field defects[2]. Raised intraocular pressure (IOP) is considered to be the most important risk factor for POAG. However, multiple studies have demonstrated that 51.4%-92.4% POAG present IOP in normal range[3].

An intriguing finding of clinical retrospective[4-5], cross-sectional[6] and prospective[7] studies is that intracranial pressure tends to be lower in patients with normal tension glaucoma. Recent clinic study showed that patients with normal pressure hydrocephalus undergoing ventriculoperitoneal shunt placement, exhibit lower intracranial pressure compared to normal individuals and demonstrate a dramatically increased risk of developing normal-tension glaucoma[8]. Glaucoma-like optic neuropathy was induced in rodent[9] and non-human primate[10] eyes by lowering intracranial pressure.

These findings suggested that abnormality of translaminar pressure difference, rather than transcorneal pressure difference, IOP itself, was a vital factor for the pathogenesis of normal tension glaucoma[11], leading to biomechaincal changes of optic head[12] and/or to deficient clearance of toxic substances in the subarachnoid space of the optic nerve[13-14] and/or in the ocular glymphatic system[15]. Thus, the search for methods to lower the translaminar pressure difference, in addition to the routine IOP control strategies represents an important and heated area for future research.

Lamina cribrosa is the tissue directly subjected to pressure difference across it. Assuming that direct communication was made between these two pressurized compartments, the pressure difference could achieve the maximum decline. In

late glaucoma, damaged region of the optic nerve, matching visual field defect, permit surgical access without deterioration of visual function. Thus, we proposed a new surgical technique targeting the lamina cribrosa and to rebalance translaminar pressure difference. In this pilot study, we practiced this new surgical technique in normal and glaucomatous non-human primate eyes and explained its potential feasibility, benefits, risks and complications in detail.

MATERIALS AND METHODS

Ethical Approval The work was conducted in accordance with the ARRIVE guidelines[16] and was approved by Animal Ethics Committees at Capital Medical University of China (No.AEEI-2019-112).

Five Rhesus macaques with an average age of 7.2y and body weight of approximately 6 kg were included. Three Rhesus macaques had no prior interventions or involved in any study. The remaining two monkeys had laser induced glaucoma in their right eye. The glaucoma model was induced two years prior to this study by green laser (532 nm) photocoagulation burns for uniform 360-degree irradiation around the trabecular meshwork. IOPs were measured intermittently and persisted high since the laser procedures were done.

Surgical Procedures Standard three-port pars plana vitrectomy was performed in all eyes by the same surgeon (Lu N). Core vitrectomy was performed, but the posterior hyaloid membrane was not specifically removed apart from the retina. A 27G needle with tip blended was inserted *via* the superiortemporal port. Then it was targeted to the peripheral region of the optic nerve and punctured into the laminai cribrosa. XEN 45 implant (Allergan Inc, USA) was prepared. The preload inserter needle was introduced from the same passage and XEN 45 implant was injected in front of the optic nerve. The XEN 45 implant was held by the forceps for peeling of inner limiting membrane and inserted into the premade puncture in the optic nerve until the length of 2 mm was visible anterior to the surface of the optic nerve (Figure 1). The scleral cuts were sealed by the 6-0 absorbable suture and water tightness was confirmed repeatedly. Finally, levofloxacin eye ointment was placed in the conjunctiva sac.

Ocular Examinations At baseline of the study and at regular intervals during the follow-up, all animals underwent an ocular examination in anesthesia state. The animals were anesthetized with an intramuscular injection of ketamine hydrochloride (20 mg/kg) and midazolam (0.2 mg/kg), with repeated injection of ketamine (10 mg/kg) as needed during the examinations and operations. The pupils were dilated by topical application of tropicamide (0.5%). Ocular examinations include slit-lamp biomicroscopy of anterior and posterior segment of the eye, rebound tonometry (Tonovet; iCare, Helsinki, Finland), photography of the optic nerve head fundus (Digital Retinal Camera, Canon CR-DGi with Canon EOS 40D; Canon, Inc., Tokyo, Japan), and spectral-domain optical coherence tomography (OCT; Spectarlis HRA+OCT; Heidelberg Engineering, Heidelberg, Germany) of the optic nerve head and retinal nerve fiber layer (RNFL).

RESULTS

Monkey No.1 Both eyes in this monkey are normal. During the operation, when the blended tip of the 27G needle was inserted deeply into the lamina cribrosa, the central retinal artery was blocked and the retina around the optic nerve was a bit swollen. The needle was retracted and the IOP was reduced immediately. The central retinal artery reperfused and the swollen retina became light gray. And no sign of blockage of central retinal artery was found during the follow-up period. No other complications happened. IOP reduced significantly until 180d after mini-shunt implantation comparing with the control eye, the IOP of which is stable. The mini shunt was located in the nasal peripheral region of optic disc, which is easily identified in the fundus photo and OCT image (Figure 2).

Monkey No.2 Both eyes in this monkey are normal. During the operation, hemorrhage was seen from deep tissue of the optic nerve after puncturing the lamina cribrosa. At the end of the operation, all the hemorrhage was cleared, and the mini shunt was located in the nasal periphery region of optic nerve. No other complications happened. However, it could not be seen during follow-up because of the vitreous hemorrhage. On 7d after operation, the IOP peaked to 12 mm Hg and then dramatically dropped to the level of 2-4 mm Hg until 180d after mini-shunt implantation (Figure 3).

Monkey No.3 No complications happened during and after surgical procedures. IOP reduced in first 14d after mini-shunt implantation and returned to the baseline since then. The mini shunt was located in the nasal periphery region of optic nerve, which is evidenced in the OCT image (Figure 4).

Monkey No.4 The right eye is laser-induced glaucomatous eye. No complications happened during and after surgical procedures. IOP reduced after the surgical procedures and remained until 180d. However, the mini shunt could not be found in the area of optic nerve after 7d postoperative (Figure 5).

Monkey No.5 The right eye is laser-induced glaucomatous eye. No complications happened during and after surgical procedures. IOP reduced for about two month and then returned to the baseline. However, the mini shunt could not be found in the area of optic nerve after 7d postoperative (Figure 6).

DISCUSSION

This pioneering study addresses in glaucoma management by proposing the world's first surgical approach targeting trans-lamina cribrosa pressure difference reduction, a pathophysiological mechanism not yet addressed by existing interventions. Historically, the lamina cribrosa was treated

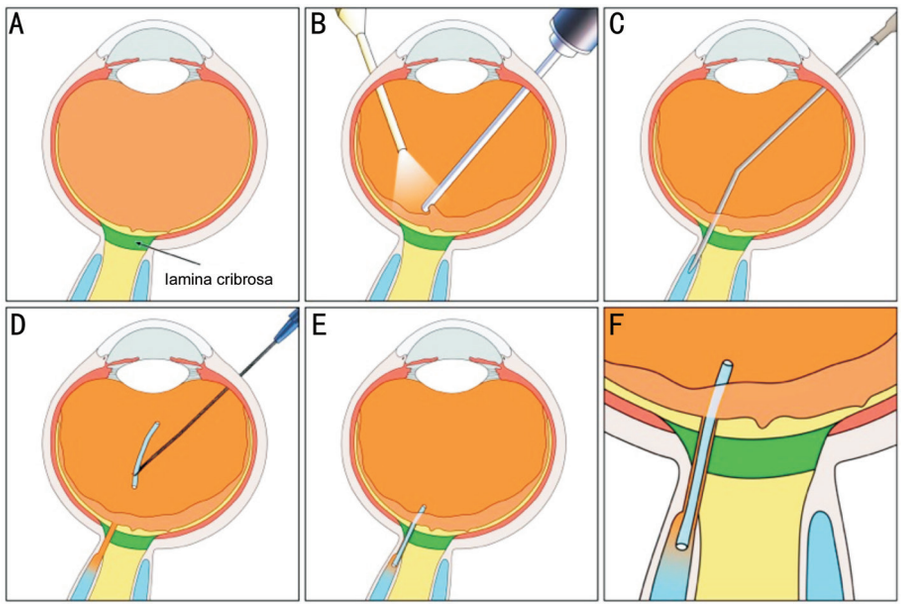

Figure 1 The diagram of surgical procedures of mini-shunt implantation in lamina cribrosa A: The sketch of ocular anatomy. The green region indicates the lamina cribrosa. B: A core vitrectomy was performed by routine three-port pars plana vitrectomy. C: Cannulation of the subarachnoidal space was made by 27G needle. D: XEN 45 implant was held and inserted to the premade puncture in the lamina cribrosa by the forceps for peeling of inner limiting membrane. E: The XEN 45 was inserted through lamina cribrosa with posterior port in the subarachnoid space. F: The enlarged view of the mini-shunt implantation in the lamina cribrosa, communicating the vitreous cavity and subarachnoid space.

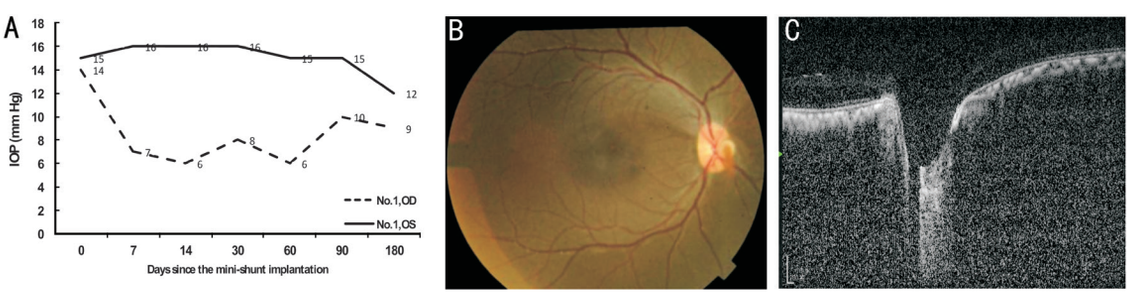

Figure 2 The operated eye after mini-shunt was implanted in Monkey No.1 A: IOP at baseline and after surgical procedure. IOP reduced significantly after operation. B, C: The mini shunt in the fundus photo and OCT imaging capture 1mo after operation. IOP: Intraocular pressure; OCT: Optical coherence tomography; OD: *Oculus Dexter* (operated eye); OS: *Oculus Sinister* (control eye).

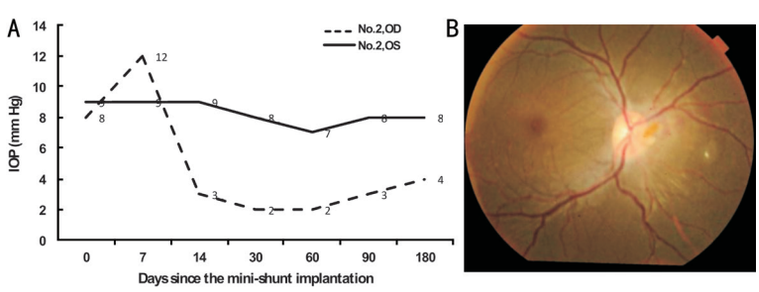

Figure 3 The operated eye after mini-shunt was implanted in Monkey No.2 A: IOP changes after mini-shunt implantation; B: Fundus image captured during operation. IOP: Intraocular pressure; OCT: Optical coherence tomography; OD: *Oculus Dexter* (operated eye); OS: *Oculus Sinister* (control eye).

as a blind and inaccessible region. Actually, it was reported that in eyes with optic pit, there is a direct passage between vitreous chamber and space behind the lamina cribrosa[17]. While no physiological communication exists between the vitreous chamber and the intracranial subarachnoid space, clinical and histopathological studies have revealed that, although rarely, SiO_2 can penetrate posterior to the lamina cribrosa into areas of the optic nerve and into the brain[18]. Moreover, our previous study revealed that glaucomatous eyes with nonprogressive optic neuropathy and visual field changes presented high prevalence of trans-lamina hole evidenced in OCT image[19]. These findings indicated that natural potential communication exists between pre-lamina vitreous and post-lamina subarachnoid space.

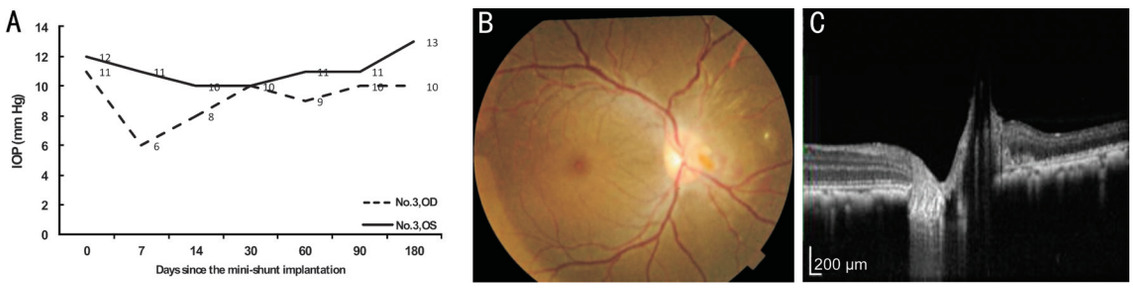

Figure 4 The operated eye after mini shunt was implanted in Monkey No.3 A: IOP at baseline and after surgical procedure; B, C: The mini shunt in the fundus photo and OCT imaging capture 1mo after operation. IOP: Intraocular pressure; OCT: Optical coherence tomography; OD: *Oculus Dexter* (operated eye); OS: *Oculus Sinister* (control eye).

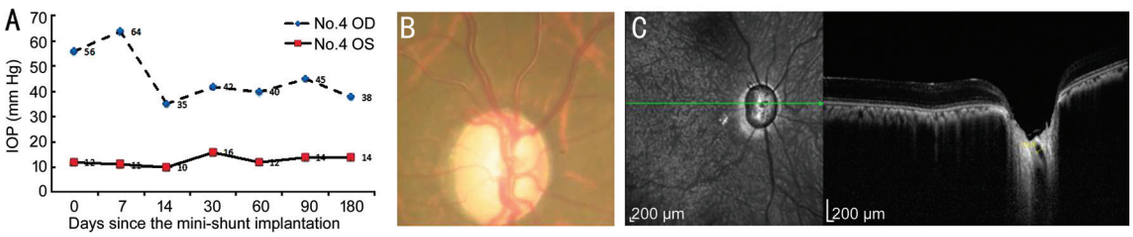

Figure 5 The operated eye after min-shunt was implanted in Monkey No.4 A: IOP at baseline and after surgical procedure; B, C: The mini shunt in the fundus photo and OCT imaging capture 1mo after operation. IOP: Intraocular pressure; OCT: Optical coherence tomography; OD: *Oculus Dexter* (operated eye); OS: *Oculus Sinister* (control eye).

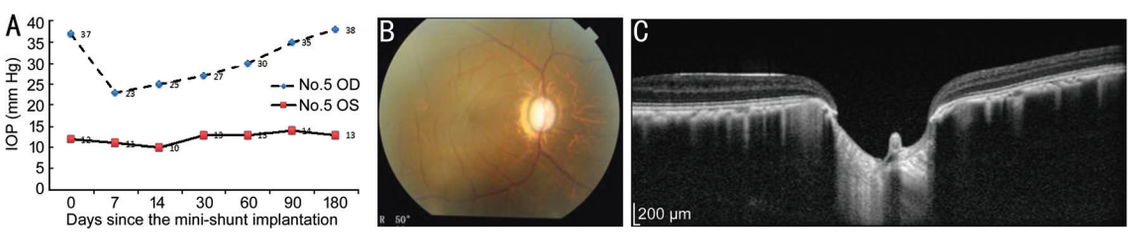

Figure 6 The operated eye after min-shunt was implanted in Monkey No.5 A: IOP at baseline and after surgical procedure; B, C: The mini shunt in the fundus photo and OCT imaging capture 1mo after operation. IOP: Intraocular pressure; OCT: Optical coherence tomography; OD: *Oculus Dexter* (operated eye); OS: *Oculus Sinister* (control eye).

A consensus suggests translaminar pressure difference contributes to the pathogenesis of glaucomatous optic neuropathy[20-22]. The gradient depends not only on the thickness but also the compliance of the collagen tissue[23] in the optic nerve. In eyes with advanced glaucoma, lamina cribrosa becomes thinner[24]. In normal tension glaucoma, the curvature of lamina cribrosa and the scleral flange properties are different from normal eyes, which increased susceptibility for glaucomatous optic nerve damage[25-26]. Furthermore, in late-stage glaucoma, the retinal nerve fiber layer attenuated, and pre-lamina tissue lost, which region is correspondence to the visual defect[27]. It provides chance to be manipulated without disturbance of the visual function. Thus, manipulating the lamina cribrosa to rebalance pressure difference across itself and to reorientation of the loaded force would be a potential therapy for glaucoma, whose IOP could not be controlled for nonprogressive optic neuropathy.

Furthermore, it was reported that aqueous humor shares key characteristics with the cerebral fluid in terms of production, circulation, absorption and composition[28]. Given that, IOP was higher than the pressure in the subarachnoid space. Thus, the circulating aqueous humor could be drained posteriorly in the eyes with the direct passage in the lamina cribrosa. There are no ocular and systemic complications were observed in these cases. Thus, we practice the mini shunt implantation in lamina cribrosa to rebalance the translaminar pressure different, which might be a promising strategy for glaucoma.

Previous, pars plana vitrectomy combined with radial optic neurotomy was designed to treat central retinal vein occlusion based on the hypothesis of alleviating the compartment syndrome[29-30]. Martínez-Jardón *et al*[31] accidentally found that IOP decreased after such surgical procedures. Then they tried pars plana vitrectomy and cannulation of the subarachnoidal space in two patients with refractory end-stage glaucoma[32]. Both patients presented significant IOP reduction in the early stage after surgery, however IOP returned to the baseline around 4mo postoperatively.

Similar as us, it was proposed that making a passage in lamina cribrosa might create an alternative method for drainage of aqueous humor in vitrectomized eyes. Moreover, considering

the similar pressure difference, the amount of the fluid drainage and good biocompatibility[33], we selected to insert XEN 45 in the cannulation of the subarachnoidal space. XEN 45 implant is 6.0 mm long with an internal diameter about 45 μm. The implant is hard when dry but is designed to be soft and flexible when hydrated, which could adapt to the tissue shape and maintain high flexibility. The high flexibility of XEN implants avoids migration and potential erosion and reduces the force between the tissue and itself. Considering higher pressure in the anterior chamber before the laminar cribrosa and the relative stagnant circulation of cerebrospinal fluid around optic nerve, the maximum possible flow rate is given by the aqueous production, which could be drained effectively by XEN 45 design.

It is ideal that the XEN 45 could be inserted into subarachnoid space around the end of the optic nerve, which directly connect the two pressurized space and a free diffusion between the cerebrospinal fluid in the optic nerve sheath and the optic nerve may be assumed. Besides the direct communication, cannulation of the subarachnoidal space could breakdown the hematic and fluid barriers in the optic nerve, which prevents the fluid communication between vitreous and intracranial fluid in physiological status. Additionally, fluid could also be eliminated by the vessels in the optic nerve head. Moreover, this fluid could drain into the orbit by the loose perivascular spaces in the optic nerve.

However, in our study, the diameter of the tip of 27G needle was relatively big for the normal optic nerve, which is fully occupied by the RNFL and glial tissue. Thus, when it was inserted deeply into the lamina cribrosa, the central retinal artery was blocked. The IOP should be appropriately lowered during this manipulation. Because the loss of the tissue content in the optic nerve, the blockage of the central retinal artery did not happen in glaucomatous eye even IOP maintained. Moreover, the dislocation of XEN 45 in glaucomatous eyes probably originated from the high-pressure difference across and severe deformation of the lamina cribrosa, which provides insufficient support and results in great mobility. A cicatrization process and partially obstruction in the lumen of XEN could be reasons for the IOP increase at the late follow-up examination.

This study's selection of non-human primates is necessary. The lamina cribrosa structure, collagen bundle arrangement, and microvascular distribution in non-human primates closely resemble those in humans, whereas rodents lack a true lamina cribrosa structure[34]. The optic nerve head size in non-human primates permits surgical precision approaching human levels[35], making microsurgical procedures feasible. The surgical operating space in monkey eyes allows direct application of standard human instruments. Complex operations cannot be performed in small animals like rabbit eyes. The intraocular healing response in non-human primates is highly comparable to humans[36], enabling risk prediction. Current alternative technologies have limitations—*ex vivo* eyes or other animal eyes lack the ocular conditions required to complete the study. This research has employed the minimum sample size and pain-minimizing techniques. Emergency pain response teams and contingency plans for ocular infections have been established.

Primary limitations of this study pertain to substantial knowledge gaps persist in surgical standardization, evidence-based treatment protocols, comprehensive risk management profiles, and refined patient selection criteria, necessitating granular optimization and further validation studies. At this exploratory stage, clinical translation remains contraindicated in human subjects. The limited cohort size (n=5) permits only preliminary assessment of procedural feasibility but lacks statistical power for robust efficacy or safety evaluation. But lasting IOP reduction in one normal eye and one glaucoma eye provide hope for us and new designed mini shunt may be helpful in the further study. Elevated surgical risks necessitate comprehensive eligibility screening algorithms and preemptive risk mitigation mechanisms. Lack of sham-operated animals hinders discrimination between IOP reduction attributable to the novel shunt versus vitrectomy-induced mechanisms, a critical confounding factor requiring future controlled studies. Direct quantification of translaminar pressure gradient changes was not performed due to technical constraints in simultaneous translaminar cribrosa pressure gradient monitoring, weakening mechanistic claims regarding pressure rebalancing. The follow-up time is relatively short. Six-month follow-up only captures short-term IOP trends but insufficiently evaluates delayed neuroinflammatory responses or optic nerve microstructure alterations *via* histopathology.

In conclusion, this study remains at a proof-of-concept stage for surgical methodology. The cannulation of the subarachnoidal space and mini shunt implantation might be related to the reduction of IOP. Clinical translation to human subjects is presently contraindicated given the unresolved technical and safety concerns. After further improvement and optimization of the surgical approach in the future, it may become a new surgical method. Nevertheless, this work represents the first-in-world exploration of translaminar cribrosa pressure gradient targeted surgical intervention. Its pioneering significance lies in establishing a novel therapeutic paradigm for glaucoma management. This may open up new ideas and directions for surgical treatment of glaucoma.

ACKNOWLEDGEMENTS

Conflicts of Interest: Xin C, None; **Sang Q,** None; **Wu S,** None; **Pang RQ,** None; **Lin DT,** None; **Wang HZ,** None; **Lu N,** None; **Wang NL,** None.

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