Original Article

Optic nerve sheath decompression for visual loss in intracranial hypertension: Report from a tertiary care center in South India

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Aim: Severe visual loss is the only serious complication of intracranial hypertension secondary to idiopathic intracranial hypertension (IIH) and some cases of cerebral venous thrombosis (CVT). Optic nerve sheath decompression (ONSD) has been shown to improve or stabilize visual function in patients with IIH, while its role in CVT is yet to be established. We report our experience with optic nerve sheath decompression for visual loss in IIH and CVT.

Materials and Methods: In this prospective noncomparative, interventional study, 41 eyes of 21 patients with IIH and CVT and visual loss underwent ONSD. The main outcome measures included best-corrected visual acuity (BCVA), visual fields, pupillary light reflex, optic nerve sheath diameter on B-scan and resolution of papilledema which were evaluated preoperatively and at follow-up at four days, two weeks, one month, three months and final follow-up. In 7/41 eyes with absent light perception preoperatively, the functional outcome was analyzed separately.

Results: Following ONSD BCVA and visual fields stabilized or improved in 32/34 (94%) eyes. Statistically significant improvement in BCVA, visual fields and pupillary light reflex occurred over the three month follow-up period. Surgical success was indicated by reduction in optic nerve diameter and papilledema resolution occurred in all patients. The outcome in the IIH and CVT groups was comparable. Four eyes with absent light perception showed marginal improvement in visual acuity. Four eyes had transient benign complications.

Conclusion: Optic nerve sheath decompression is an effective and safe procedure to improve or stabilize vision in patients with visual loss caused by IIH and CVT.

Key words: Cerebral venous thrombosis, idiopathic intracranial hypertension, optic nerve sheath decompression

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Introduction

Intracranial hypertension (IH) is a multifactorial syndrome characterized by severe headache, nausea, vomiting, transient visual obscuration and diplopia. Idiopathic intracranial hypertension (IIH) is the terminology used when no underlying etiology is detected. It is termed secondary IH when an underlying cause is detected like cerebral venous thrombosis (CVT) or a space-occupying lesion.[1]

Severe visual loss is the only significant complication of IIH.[1,2] The natural history of visual loss due to chronic papilledema is of early visual field loss with loss of acuity occurring later. Intractable headaches and visual loss are indications for treatment. Headache is usually treated with oral acetazolamide or frusemide to reduce intracranial pressure (ICP).[1,2] Failure of conservative management to prevent progressive visual loss is an indication for surgery which includes optic nerve sheath decompression (ONSD) or cerebrospinal fluid (CSF) shunting procedures.[1,2] Progressive visual loss in the presence of a functioning shunt is documented.[3] Therefore, a direct approach to the distal optic nerve by ONSD is an attractive option in these cases.

Forty per cent of patients with CVT clinically manifest as isolated mimicking IIH.[4,5] The management of such patients is similar to the management of IIH including ONSD for visual loss. There are a few reports of the successful use of ONSD to treat visual loss in CVT.[4,6,7] The aim of the study was to evaluate the outcome of ONSD in relation to visual loss due to IIH and CVT of any cause and the complications of the procedure.

Materials and Methods

In this prospective noncomparative interventional study, patients undergoing ONSD for visual loss due to IH caused by IIH and CVT in our department from January 2000 to December 2005 were included for analysis. The inclusion criteria were demonstrated raised ICP (CSF opening pressure >250 mm H_2O), visual loss as evidenced by deteriorating visual fields or visual acuity and ultrasonologically demonstrable increased subarachnoid fluid in the retrobulbar optic nerve. All patients were on maximum tolerable medical therapy of oral acetazolamide up to 2000 mg/day in four divided doses, oral glycerol 30 ml three times/day (TID) and in acute cases IV mannitol 100 ml TID. The CVT patients were on oral anticoagulation with nicoumalin to maintain a prothrombin time INR of 2 to 3. Exclusion criteria included absence of visual

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loss in the presence of papilledema and raised ICP, enlarged blind spot being the only visual field change, disc edema due to other causes (such as optic neuritis)and patients not available for follow-up for at least three months.

A detailed history regarding patient demographics, presenting complaints including visual and non-visual symptoms of papilledema and raised ICP and details of medical or surgical treatment received, was taken. All patients underwent a detailed neurological evaluation including history, clinical examination, neuroimaging studies with magnetic resonance imaging (MRI) and CSF studies including opening pressure recording and CSF composition. Relevant investigations were done to detect any metabolic, endocrine or hematologic cause for the raised ICP or CVT. CSF opening pressures were recorded high with normal CSF composition in all patients. Ocular parameters (assessed at baseline and follow-up at four days, two weeks, one month, three months and subsequent follow-up) included best-corrected visual acuity (BCVA), perimetry, stereoscopic funduscopy, pupillary light response, stereoscopic fundus photography and measurement of optic nerve sheath diameter on B-scan ultrasonography (USG).

Best-corrected visual acuity was recorded using Snellen's chart. Snellen visual acuity was converted to the logarithm of the minimum angle of resolution (logMAR) visual acuity for the purpose of analysis. Ishihara's pseudoisochromatic chart was used to assess color vision in all patients. Visual field assessment was performed using a Humphrey field analyzer and either a full field suprathreshold program or a SITA standard 30-2 threshold test. The same algorithm was used for follow-up in each individual patient. Visual field defects were graded from Grade 1 to 5 as per the criteria described by Wall and George for automated perimetry[8] and scored as such so that a lower number indicated a better field.

Direct and consensual pupillary reactions were assessed and scored as numbers 0 to 3 (0-non-reactive, 1-sluggishly reactive, 2-ill sustained and 3-brisk) for analysis. Ocular motility evaluation was performed. Indirect ophthalmoscopy, slit-lamp biomicroscopy with 90D lens and stereoscopic fundus photography was performed in all eyes. The degree of papilledema of each eye was graded using the Frisen's staging scheme[9] from Stage 0 to 5, for the purpose of analysis. All eyes underwent orbital echography to assess the optic nerve sheath diameter and it was found increased in all eyes.

Optic nerve sheath decompression was performed using a medial transconjunctival approach under general anesthesia. After a 270 degree medial peritomy, the medial rectus was tagged with 6-0 polygalactin and the muscle disinserted. The globe was fully abducted with the help of a 'base-ball' traction suture placed on the sclera at the site of the medial rectus disinsertion. The optic nerve was exposed under the operating microscope using malleable retractors and tailed cotton applicators to maneuver the orbital fat. The short posterior ciliary vessels were gently reflected with a nerve hook or a cotton-tipped applicator and an avascular area of anterior optic nerve sheath was incised using a side-port knife used in cataract surgery or MVR Unitome 5560. An incision about 4 to 5 mm in length was made about 2 mm posterior to the junction of the globe and the nerve. Adhesions between the nerve and dura were gently lysed by movement of a Fisher tenotomy hook within the subarachnoid space. Cerebrospinal fluid was drained on incision of the sheath, confirming the fenestration. Base-ball traction sutures were released, the medial rectus reinserted and the conjunctiva closed with running suture. Bilateral ONSD was done in all cases except in two patients who did not have significant visual loss in one eye. Peroperatively a five-day course of intravenous ceftriaxone 2 g/day in two divided doses was started. Patients were discharged on the fifth postoperative day. Complications if any were recorded during each postoperative follow-up visit.

To obtain a picture of clinical relevance, functional outcome was categorized into "qualified success" and "success". Qualified success or stabilization of visual function was defined as improvement or worsening of less than two lines on the Snellen acuity or maintenance of existing Snellen acuity, visual field improvement or worsening less than one grade of staging scheme[8] or maintenance of existing visual field at three months follow-up and subsequent follow-up visits. Success was defined as improvement in visual acuity by two or more lines of Snellen's acuity or an increase in the visual field grading by at least one grade or more as per the grading criteria for automated perimetry in papilledema.[8] Surgical 'success' was considered in terms of resolution of papilledema on funduscopy and echographic evidence of optic nerve sheath diameter reduction. Resolution of papilledema was considered 'successful' if the disc appearance improved by at least one stage or more of Frisen's staging scheme.[9] "Worsening" in visual function was defined as decrease in visual acuity by two lines or more of Snellen's acuity and a decrease in visual field grading by at least one grade or more.[8]

The data were analyzed as follows. Descriptive statistics were computed for the range, mean and standard deviation for quantitative variable and category frequency counts and percentages for qualitative variable. In the inferential statistical analysis, one-way repeated measures analysis of variance (RMANOVA), with Pillai's trace as the statistical criterion was used to compare the means of variables obtained from the same group at several different points in time. It was also used to compare outcome between the groups across time. Subsequently the paired t test was used to make comparisons between means at two points in time.

Results

Forty-one eyes of 21 patients who fulfilled the inclusion criteria underwent ONSD. There was a female preponderance with 15 females and six males. The age range was 18 to 48 years with mean (SD) of 29.5 (±8.2) years. The patients were divided into three groups based on the cause of IH: this included Group I with IIH in five patients (10 eyes), Group II with postpartum CVT in seven patients (14 eyes) and Group III with other causes of CVT in nine patients (17 eyes). Seven eyes (7/41) with visual acuity of absent light perception at presentation were not included for analysis of functional outcome and their functional outcome is given separately.

Headache and vomiting were present in 18/21 patients preceding complaints of visual loss. Three patients, however, presented with advanced visual loss only. Sixth nerve palsy accounted for the far majority of non-visual neurological deficits in 14/21 patients. Only two patients had other focal neurological deficits.

Thirty-two of 34 eyes (94%) showed either improvement (50%) or maintenance (44%) of preoperative BCVA at three months follow-up with only 2/34 eyes of one patient showing postoperative worsening [Table 1]. Statistically significant improvement in BCVA over time occurred in all three groups ($P < 0.001$) [Table 2]. Further analysis showed significant improvement at Day 4, two weeks and at one month follow-up [Table 2]. Final visual acuity computed for eyes which were available for follow-up at six months and more showed further improvement in BCVA. The BCVA outcome between the three diagnostic groups was comparable [Table 3].

Thirty-two of 34 eyes (94%) showed either improvement (64%) or maintenance (29%) of preoperative visual field at three months follow-up [Table 1]. Higher per cent of eyes showed improvement in visual fields than BCVA. Overall visual field progression over three months showed a statistically significant improvement ($P < 0.001$) [Table 4]. All three groups showed a significant improvement in visual fields at Day 4 postoperatively which remained stable till three months postoperatively [Table 4]. Further improvement in fields occurred in patients who were available for follow-up at six months and more, which was statistically significant ($P = 0.03$).

Table 1: Functional outcome at three months postoperatively of the three groups

	Best-corrected visual acuity			Fields		
	Group 1 n = 10 (%)	Group 2 n = 9 (%)	Group 3 n = 15 (%)	Group 1 n = 10 (%)	Group 2 n = 9 (%)	Group 3 n = 15 (%)
Success* (improved)	4 (40)	7 (78)	6 (40)	9 (90)	4 (45)	9 (60)
Qualified* success (maintained)	6 (60)	2 (22)	7 (47)	1 (10)	5 (56)	4 (27)
Failure* (worsened)	0 (0)	0 (0)	2 (13)	0 (0)	0 (0)	2 (13.3)

*For definition please refer to results section

Table 2: Changes in best-corrected visual acuity over the follow-up period for all patients together

	N	Mean (SD)	Paired t test	Df	*P*
BCVA day 0	34	1.08 (1.19)			
BCVA day 4	34	0.81 (0.92)	4.05	33	$P \leq 0.001$
BCVA 2 weeks	34	0.70 (0.86)	2.39	33	$P = 0.02$
BCVA 1 month	34	0.60 (0.79)	2.03	33	$P = 0.05$
BCVA 3 months	34	0.66 (0.83)	1.05	33	$P = 0.29$
BCVA >6 months	27	0.51 (0.71)	0.48	26	$P = 0.63$

Pillai's trace = 0.57; F = 7.65; df = 5; $P < 0.001$; BCVA - Best-corrected visual acuity given in logMAR equivalent is the outcome variable

Table 3: Comparison of progression of best-corrected visual acuity over follow-up period in the three groups

Group*	Best-corrected visual acuity					
	Pre-op mean (SD)	4 days mean (SD)	2 weeks mean (SD)	1 month mean (SD)	3 months mean (SD)	6 months or more mean (SD)
1 (n = 10)	0.75 (0.83)	0.60 (0.52)	0.54 (0.55)	0.45 (0.62)	0.44 (0.62)	0.37 (0.68)
2 (n = 9)	1.07 (1.09)	0.81 (0.92)	0.70 (0.86)	0.60 (0.79)	0.66 (0.83)	0.51 (0.71)
3 (n = 15)	1.0 (1.14)	0.79 (1.03)	0.61 (0.94)	0.51 (0.72)	0.67 (0.82)	0.50 (0.70)

Effect between groups for visual acuity: F = 2.44; df = 10; $P = 0.06$; BCVA - Best-corrected visual acuity given in logMAR equivalent is the outcome variable.
*For definition of groups please refer to results section

Table 4: Progression of visual fields over the follow-up period for all patients together

	n	Mean (SD)	Paired t test	df	*P*
Field day 0	34	4.3 (1.1)			
Field day 4	34	3.9 (1.1)	4.24	33	$P < 0.001$
Field 2 weeks	34	3.6 (1.3)	4.04	33	$P < 0.001$
Field 1 month	34	3.5 (1.4)	0.37	33	$P = 0.71$
Field 3 months	34	3.4 (1.6)	1.75	33	$P = 0.09$
Field final	27	3.0 (1.9)	2.18	26	$P = 0.03$

Pillai's trace = 0.5; F = 9.14; df = 4.37; $P < 0.001$; Visual field given as grades is the outcome variable

Table 5: Comparison of progression of visual fields over follow-up period in the three groups

Groups*	Visual field					
	Pre-op mean (SD)	4 days mean (SD)	2 weeks mean (SD)	1 month mean (SD)	3 months mean (SD)	6 months or more mean (SD)
1 (n = 10)	4.7 (0.48)	4.1 (0.73)	3.7 (0.82)	3.4 (0.96)	3.2 (0.91)	2.8 (1.5)
2 (n = 9)	4.2 (1.03)	4.0 (1.32)	3.6 (1.66)	3.6 (1.73)	3.4 (2.12)	2.8 (2.40)
3 (n = 15)	4.1 (1.16)	3.8 (1.22)	3.5 (1.45)	3.5 (1.59)	3.4 (1.65)	3.07 (1.71)

Effect between groups for visual fields: F = 1.59; df = 10; *P* = 0.14; Visual field given as grades is the outcome variable. *For definition of groups please refer to results section

Table 6: Demographics and functional outcome of patients with visual acuity of absent light perception at presentation

Age/sex	Cause of IH	*BCVA0 (logMAR equivalent)	BCVA 3m (logMAR equivalent)	Field Grade 0	Fields Grade 3m	Follow-up (months)
24/F	PP-CVT	No PL	No PL	5	5	3
23/F	PP-CVT	No PL	2	5	5	29
23/F	PP-CVT	No PL	2	5	5	29
35/M	CVT	No PL	No PL	5	5	3
35/M	CVT	No PL	No PL	5	5	3
18/F	PP-CVT	No PL	1.4	5	5	12
18/F	PP-CVT	No PL	1.4	5	5	12

PP-CVT- Postpartum cerebral venous thrombosis, CVT - Cerebral venous thrombosis of any cause, *As visual acuity of absent light perception has no equivalent notation in logMAR units, in the table "absent light perception" has been given as "No PL" itself, BCVA - Best corrected visual acuity

The visual field outcome between the three diagnostic groups was comparable [Table 5].

Pupillary light reflex showed a statistically significant improvement at three months ($P < 0.001$). Statistically significant papilledema resolution occurred by Day 4 postoperatively ($P < 0.001$) with no further significant change over three months and six months. Statistically significant change in echographic optic nerve sheath diameter was seen at three months ($P < 0.001$) and further reduction was seen in patients available for follow-up after six months ($P = 0.023$).

Seven eyes of four patients had a visual acuity of absent light perception preoperatively. The details of these patients are given in Table 6. Intracranial hypertension was secondary to CVT in all four patients. Four eyes showed a mild improvement in visual acuity which remained stable at 12 to 24 months follow-up.

The most common complication in this study was transient pupillary atony (13.4%) followed by transient diplopia (3.4%) and early orbital cellulitis in one patient which resolved completely with the addition of intravenous vancomycin 1 g twice/day and amikacin 500 mg TID for seven days. Deterioration of vision was seen in two eyes starting one month postoperatively, which did not improve despite repeat ONSD.

Discussion

The pathophysiologic mechanism by which ONSD improves visual function is unsettled. The two main suggested mechanisms include obliteration of the subarachnoid space surrounding the optic nerve by fibroblast proliferation, preventing CSF pressure transmission distal to the operative site[10] and the second being the creation of a dural fistula which allows egress of CSF from the operative site.[11] Findings which favor the second hypothesis include relief of headache following ONSD and bilateral improvement in visual function following unilateral surgery.[12,13] Optic nerve sheath decompression has been performed using different approaches to the optic nerve like medial transconjunctival orbitotomy,[11,14-17] lateral orbitotomy[18] and a lateral canthotomy approach.[19] Overall, the results are similar with both medial and lateral approaches.[14,18,20]

There is increasing evidence on the efficacy of ONSD in improving or preserving vision in patients with IH, especially IIH.[3,6,7,14-16,20] The benefits of ONSD in stabilizing vision in IIH, have also been shown to be applicable in IH due to other causes like CVT, intracranial tumors and infections.[4-7,21-23] Previous reports[4-7] have shown that ONSD in CVT-related IH was visually beneficial. Visual loss in CVT is multifactorial and includes pressure effect of transmitted ICP on the optic nerve and vascular compromise to the optic nerve and rest of the optic pathway due to thrombosis.[4] One could expect poorer outcomes after the procedure in this group of patients, where vascular factors compromised recovery. However, 22/24 eyes with CVT of any cause showed stabilization or improvement during the study period. Hence ONSD should be considered in all patients with visual loss due to CVT. Anecdotally, it was noted that dramatic improvements in visual acuity and fields were seen more frequently in patients with IIH than those with CVT. We recommend caution in prognosticating outcome in patients with CVT and advanced visual loss for this reason.

The overall visual acuity and visual field outcome at three months follow-up showed significant improvement and the benefits of the procedure accrued to all the three etiological groups. The improvement in visual function occurred in the early postoperative period which was maintained at three

months follow-up. Follow-up at six months and beyond showed a favorable trend in visual function in 27/34 eyes. Reduction in optic nerve sheath diameter and resolution of papilledema, indicators of surgical success of ONSD showed statistically significant change following ONSD.[15,18,24] Periodic ultrasonic evaluation of retrobulbar optic nerve sheath diameter is advisable, as partially atrophic discs would not show a significant swelling to alert the physician about possible long-term surgical closure of the shunt.

Presence of optic disc pallor in association with chronic papilledema and advanced visual loss is not a contraindication for surgery. Sergott *et al.*,[14] showed that 3/23 eyes with optic disc pallor regained 20/20 vision. However, the experience of other studies was not encouraging for similar cases.[17,20] Ten eyes with disc swelling and pallor showed no dramatic functional improvement in our study. However, they all showed stabilization and six had improved vision. We advocate ONSD for all patients with some retained visual acuity and optic disc pallor in an edematous disc, to preserve residual vision. Four eyes of two patients with absent light perception regained ambulatory vision almost one month after surgery. Both these patients had rapid loss of vision due to postpartum CVT and both received intravenous methylprednisolone prior to surgery. The remaining three eyes with absent light perception had gradual loss of vision and did not benefit from surgery. The probable explanation is that in acute loss of vision there exist some fibers which remain hypofunctional or nonfunctional and can regain functional viability after a pressure-relieving operation. However, it is unlikely that if the condition persists for several weeks, these axons would remain viable.

The complications were benign and transient. The reported incidence of complications ranges from 2 to 35%[16,25,26] and these include pupillary atony, transient diplopia, lost medial rectus muscle, sudden loss of vision due to traumatic optic neuropathy and central retinal artery occlusion. The occurrence of orbital cellulitis as a complication of ONSD has not been reported in the literature. The reported incidence of late visual loss ranges from 10 to 40%.[7,19,27,28] Rapid loss of vision one month postoperatively following an initial improvement was seen in two eyes (10%) and this was similar to that experienced by five patients reported by Corbett *et al*.[20] The patient in our study and one patient in the study by Corbett *et al.*,[20] had CVT as the underlying cause of raised ICP. Ischemia of the optic nerve due to thrombotic disease may have been the precipitating factor.

One of the limitations of this study is that it is a prospective nonrandomized noncomparative study where ONSD was the primary surgery done rather than a neurosurgical shunt, which classically, is the conventional paradigm. A randomized study would include primary shunt treatment as the other treatment alternative for a short and long-term follow-up study. More number of patients would be necessary if the power of such a study is to be relevant. The second limitation being the short follow-up period. Long-term follow-up of these patients including ultrasonic evaluation of the optic nerve sheath diameter is mandatory as there is always the risk of closure of filtration site and a repeat procedure may be required.

In conclusion ONSD is effective in improving or maintaining vision in patients with IIH and visual loss. This study has also established the effectiveness of ONSD in cases of visual loss in CVT. With proper attention to detail, it is a safe procedure with minimal complications. Early surgical intervention is associated with better visual outcome. However, the study cannot make specific recommendations at this time, of the ideal stage at which to intervene and what the most sensitive parameters are, of early decompensation, which could be used as guidelines to decompress.

The benefit of ONSD in preserving residual vision extends to eyes with chronic papilledema, disc pallor and advanced visual loss.

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References

1. Friedman DI. Pseudotumour cerebri. Neurosurg Clin North Am 1999;10:609-21.
2. Radhakrishnan K, Ahlskog JE, Garrity JA, Kurland LT. Idiopathic intracranial hypertension. Mayo Clin Proc 1994;69:169-80.
3. Kelman SE, Sergott RC, Cioffi GA, Savino PJ, Bosley TM, Elman MJ. Modified optic nerve decompression in patients with functioning lumbooperitoneal shunts and progressive visual loss. Ophthalmology 1991;98:1449-51.
4. Biousse V, Ameri A, Bousse MG. Isolated intracranial hypertension as the only sign of cerebral venous thrombosis. Neurology 2000;54:2030-6.
5. Purvin VA, Trobe JD, Kosmovsky G. Neuroophthalmic manifestation of ceberbral venous thrombosis. Arch Neurol 1995;52:880-5.
6. Lam BL, Schatz NJ, Glaser JS, Bowen BC. Pseudotumour cerebri from cranial venous obstruction. Ophthalmology 1992;99:706-12.
7. Banta JT, Farris BK. Pseudotumour cerebri and optic nerve sheath decompression. Ophthalmology 2000;107:1907-12.
8. Wall M, George D. Idiopathic Intracranial hypertension: A prospective study of 50 patients. Brain 1991;114:155-80.
9. Frisen L. Swelling of the optic nerve head:a staging scheme. J Neurol Neurosurg Psychiatry 1982;45:13-8.
10. Davidson SI. A surgical approach to pleurocephalic disc oedema. Trans Ophthalmol Soc UK 1969;89:669-90.
11. Keltner JL. Optic nerve sheath decompression. Arch Ophthalmol 1988;106:1365-9.
12. Hupp SL, Buckley EG. Post traumatic venous obstructive retinopathy associated with enlarged optic nerve sheath. Arch Ophthalmol 1984;102:254-8.
13. Burde RM, Karp JS, Miller RN. Reversal of visual deficit with optic nerve decompression in long standing pseudotumour cerebri. Am J Ophthalmol 1974;77:770-2.
14. Sergott RC, Savino PJ, Bosley TM. Modified optic nerve sheath decompression provides long-term visual improvement for pseudotumour cerebri. Arch Ophthalmol 1988;106:1384-90.
15. Acheson JF, Green WT, Sanders HD. Optic nerve sheath decompression for the treatment of visual failure in chronic raised intracranial pressure. J Neurol Neurosurg Psychiatry 1994;57:1426-9.
16. Brourman ND, Spoor TC, Ramocki JM. Optic nerve sheath decompression for pseudotumour cerebri. Arch Ophthalmol 1988;106:1378-83.
17. Spoor TC, McHenry JG. Long term effectiveness of optic nerve sheath decompression for pseudotumour cerebri. Arch Ophthalmol 1993;11:632-5.
18. Tse DT, Nerad JA anderson RL, Corbett JJ. Optic nerve sheath

fenestration in pseudotumour cerebri: A lateral orbitotomy approach. Arch Ophthalmol 1988;106:1458-62.

19. Kersten RC, Kulwin DR. Optic nerve sheath fenestration through a lateral canthotomy incision. Arch Ophthalmol 1993;111:870-4.
20. Corbett JJ, Nerad JA, Tse DT, Anderson RL. Results of optic nerve fenestration for pseudotumour cerebri. Arch Ophthalmol 1988;106:1391-7.
21. Johnston I, Hawke S, Halmagyi M, Teo C. The Pseudotumour syndrome-disorders of cerebrospinal fluid circulation causing intracranial hypertension without ventriculomegaly. Arch Neurol 1991;48:740-7.
22. Hupp SL. Optic nerve sheath decompression: The emerging indications. Ophthalmol Clin North Am 1991;4:575-83.
23. Galbraith JE, Sullivan JH. Decompression of the perioptic meninges for relief of papilloedema. Am J Ophthalmol 1973;76:687-92.
24. Galetta S, Byrne SF, Smith JL. Echographic co-relation of optic nerve sheath diameter and cerebrospinal fluid pressure. J Clin Neuro Ophthal 1989;9:79-82.
25. Spoor TC, Ramocki JM, Madion MP, Wilkinson MJ. Treatment of pseudotumour cerebri by primary and secondary optic nerve sheath decompression. Am J Ophthalmol 1991;112:177-85.
26. Subburam P, Loganathan VM. Decompression of the perioptic meninges for relief of papilloedema in benign intracranial hypertension. Indian J Ophthalmol 1984;32:9-12.
27. Brodsky MC, Rettele GA. Protracted postsurgical blindness with visual recovery following optic nerve sheath decompression. Arch Ophthalmol 1997;115:1473-4.
28. Mauriello JA Jr, Shaderowfsky P, Gizzi M, Frohman L. Management of visual loss after optic nerve sheath decompression in patients with pseudotumour cerebri. Ophthalmology 1995;102:441-5.

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