PREDICTORS OF SUCCESSFUL OPTIC NERVE SHEATH DECOMPRESSION IN CHRONIC INTRACRANIAL HYPERTENSION

by

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CERTIFICATE OF APPROVAL

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Table of Contents

List of Tables

List of Figures

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Abstract

Purpose. Chronic intracranial hypertension (IH) can lead to visual impairment in an estimated 25% to 50% of those afflicted. Optic nerve sheath decompression (ONSD) or fenestration has been shown to be an effective form of vision preservation with improvement in visual acuity (VA) and visual fields (VF). The purpose of this study was to examine the role of underlying etiology in IH, examine factors related to IH treatment paths, and preoperative factors leading to a successful outcome in VA and VF after ONSD intervention.

Methods. This study was an observational retrospective cohort of 1028 eyes in 514 of chronic intracranial hypertension cases from the Intracranial Hypertension Registry, Portland, Oregon. Main outcome measures included visual acuity and visual fields.

Results. The signs and symptoms of intracranial hypertension did not differ according to etiology. Those that underwent an ONSD procedure as the first procedure had worse visual symptoms at diagnosis than those that remained medically treated. Stabilization/Improvement post-ONSD over a short-term and long-term follow-up was associated with worse visual acuity pre-ONSD surgery (odds ratio = 0.86, confidence interval 0.77, 0.96, $p = 0.01$) adjusting for time to surgery.

Conclusion. Diagnostic signs and symptoms of IH are poor predictors of successful VF and VA outcomes after ONSD surgery. ONSD appears to be an

effective stop gap measure for preserving abnormal vision in chronic intracranial hypertension but should not be considered as a long-term treatment intervention.

Introduction

Intracranial hypertension (IH) is a condition of raised intracranial pressure that can present with symptoms of headache, pulsatile tinnitus, stiff neck/shoulders, transient visual obscurations, visual gray-outs/black-outs/or white-outs. Signs of the condition are usually manifested by papilledema (swelling of the optic nerve disc), loss of visual field, and documented raised intracranial pressure on lumbar puncture. Intracranial hypertension has been classically divided into two categories, idiopathic and secondary. A diagnosis of idiopathic IH (IIH) is made when no underlying pathophysiologic mechanism is found to account for the increased intracranial pressure (ICP). When ICP can be attributed to an underlying cause, such as a drug toxicity or tumor, the diagnosis of secondary IH (SIH) is applied.

Various factors such as gender, obesity, and age have been found to influence the incidence rates of idiopathic IH. Men have a much lower incidence (0.3 – 1.5/100,000)[12] than women who have an incidence rate of 1-3/100,000[29, 30]. Women of childbearing age (age 15 to 44) that are obese have a much higher incidence rate (20/100,000)[13] than those of either much older age groups or much younger age groups. It has been suggested that hormonal changes in and around puberty play a role in the incidence of IH with pediatric cases occurring at approximately the same rate until puberty[3, 20]. The true incidence of secondary IH has never been extensively examined epidemiologically.

The main medical treatment for intracranial hypertension involves a class of diuretics, carbonic anhydrase inhibitors (CAIs), in particular acetazolamide

(Diamox). The use of acetazolamide in IH is off-label and therefore, there have been no randomized trials examining the efficacy of acetazolamide on the reduction, production, or re-absorption of cerebrospinal fluid (CSF) in IH patients, nor has there been an examination of the most effective dose to date. Friedman and Jacobson[15] state that 1 to 2 grams are generally prescribed and tolerated but as little as 250 mg are generally tried. For those IH patients with intolerance to acetazolamide or an allergy to sulfas, other medications may be used alone or in combination with acetazolamide. These other medications, topiramate (Topamax), furosemide (Lasix), and/or methazolamide (Neptazane), are also off-label uses for the drugs and have not been rigorously investigated with respect to IH[15].

Once maximal medical treatment has failed, there are two main forms of surgical treatment for IH: CSF diversion (shunting) and optic nerve sheath decompression. CSF diversion procedures are generally performed for severe intractable headaches and include lumboperitoneal shunts (LPS) and ventriculoperitoneal shunts (VPS). The long-term efficacy of LPS appears to be limited. In a retrospective study of LPS, 67% of those initially receiving a shunt underwent 6.35 subsequent revisions over a follow-up period of 6 to 143 months[7]. The average durability (length of a functioning shunt) was approximately 9 months[7, 31]. Furthermore, shunt failure can occur from 1 day postoperative to 15 years and may not reduce visual loss[31]. In a recent literature review, Feldon[13] found that vision improved in 38.7% of VPS procedures and

44.6% of LPS procedures compared to 80% of non-chronic optic nerve sheath decompression cases.

As opposed to shunt operations for on-going symptoms of intractable headaches, optic nerve sheath decompressions (ONSD) are performed for the sign of visual acuity and/or visual field loss. Visual impairment in IH is estimated to be 25 to 50% of those afflicted[10, 13] although the rate of ONSD surgeries for IH is unknown. The loss of visual acuity and/or visual function is caused by papilledema due to ICP. The pathophysiology of papilledema due to ICP remains unclear[34] and several mechanisms of ONSD action have been proposed to account for the unilateral and bilateral effect of the surgery[24,35, 43].

ONSD has been shown to be an effective form of vision preservation with improvement in visual acuity and visual fields [1, 2, 4, 19, 27, 28, 39, 42] with a 32% failure rate in 6 to 60 months of follow-up[39]. Furthermore, visual improvement can be seen in as little as 4 days postoperatively[27]. Table 1 summarizes the efficacy of ONSD over various follow-up time frames. Complications of ONSD can lead to adverse vascular events such as central retinal artery occlusion and branch artery occlusion, motility disorders (adduction deficits), tonic pupils, diplopia (double vision), retrobulbar or orbital hemorrhage, protracted blindness, orbital globe perforation, extensive scarring, re-operation, and even death[4, 8, 9, 15, 22, 28, 36, 37, 39, 40, 43, 44]. Complication rates depend upon the approach (medial or lateral) to the optic nerve used during surgery[9, 23, 36, 44].

Although many retrospective studies appeared in the late 1980s and early 1990s examining efficacy of ONSD, examination of preoperative factors have

yielded no conclusive results[9] and the severity of papilledema preoperatively has not been associated with postoperative visual function[9, 37]. Only one study to date specifically examined preoperative factors predicting the long-term success of the procedure[8]. In examining postoperative visual field function, Chandrasekaran et al. found that a visual field defect outside of 10 degrees of fixation was strongly associated with improvement (OR = 9.7, $CI_{95\%}$ 1.1 – 85.9) and that surgery after six months of diagnosis significantly associated with a poorer visual field outcome (OR = 0.06, $CI_{95\%}$ 0.01 – 0.70).

As the incidence, and subsequently the number of surgical interventions for chronic IH is increasing[11], the establishment of a preoperative rubric for effective and efficient treatment of chronic IH is necessary, especially given that in many IH cases medical and/or surgical care is an urgent matter. For example, of the 3518 discharges for adult IH in 2005, almost half (48.4%) were admitted from an emergency department (NIS, 2005). Furthermore, many IH patients may be admitted on more than one occassion[16] indicating that known interventions for IH are not effective in either the long-term or the short-term.

A majority of past studies have not specifically examined whether or not an underlying etiology could contribute to the overall success and/or failure of ONSD for IH. Therefore, the preoperative factors as well as underlying etiology were examined in a well-defined population of patients to establish what potentially influences the success and/or failure of the procedure.

Methods

Participants.

This retrospective cohort study examined 514 chronic intracranial hypertension cases from the Intracranial Hypertension Registry in Portland, Oregon. The Intracranial Hypertension Registry (IHR or the Registry) is a worldwide database of IH patients that is a joint project of the Intracranial Hypertension Research Foundation and the Casey Eye Institute at Oregon Health & Science University. The IHR was founded in 2003. A majority of the patients self-refer to the Registry; although physician referral has occurred in approximately 30%. Patients in the Registry represent individuals with both idiopathic IH and secondary IH (IH due to an underlying cause). After the initial patient questionnaire has been returned to the IHR, the patient's pertinent medical records are requested and abstracted by Registry staff. Each patient's medical history is reviewed by a staff ophthalmologist and either confirmed to be an idiopathic or secondary case. The review is based on the modified Dandy criteria[12] for idiopathic cases (See Table 2 for the modified Dandy criteria). Secondary IH is determined by the medical record review in which the treating or diagnosing physician stated that there was an underlying cause of the increased intracranial pressure. The IHR follows patients long-term with yearly patient follow-up questionnaires and annual to biennial medical record updates.

All confirmed cases at the time of this study of chronic intracranial hypertension from the IHR were evaluated for the completeness of the ophthalmologic data. If there were no ophthalmology exams present in the patient's medical chart they were excluded from this analysis.

Measurements.

Preoperative and Postoperative Measurements.

Papilledema, the swelling of the optic nerve head, was measured in two ways – overall assessment as present/absent and by classification. Classification generally included the terms contained within the medical chart referring to papilledema and, when necessary, Frisén Grades[17] were converted into this classification scheme (See Table 3). Duration of papilledema was computed as the time from the first positive notation of papilledema to the time of ONSD surgery. Snellen lines of visual acuity were coded from -1 (< 20/20) to 19 (no light perception; NLP). Rate of visual acuity decline was measured by visual acuity at diagnosis minus visual acuity preoperatively divided by the time between diagnosis and the ONSD operation. Visual field measures utilized the mean deviation score on automated perimetry. The Wall & George[45] rating system (WaGRS) which combines visual field deficits and visual acuity scores was used as a composite score for visual symptoms (See Table 4). The WaGRS was developed for Humphrey Visual Fields (HVF) and Goldmann Perimetry (GVF) and when necessary, this rating system was extrapolated to classify Tangent Screen Perimetry and HVF frequency doubling. All visual field and visual acuity reviews were blinded to the surgical status of the participant.

Success Measurements.

There were two main outcomes of ONSD surgery examined in this study. The first outcome was measured by change of visual acuity preoperatively to at the time of last follow-up. Visual acuity was considered stabilized if the change

was one Snellen line or less, and improved if visual acuity increased by two or more Snellen lines. The second outcome measure was visual field defects at time of last follow-up. Our first attempt to examine visual field defects classified the Wall and George rating system into minimal defect (0 – 2) and marked defect (3 – 5). The second attempt to examine visual field defects classified the mean deviation score of the visual field into non-severe (≤ 10 Db) and severe (> 10 Db).

Statistical Analysis.

All univariate and multivariate analyses used the mixed effects models which can account for the potential intra-person correlations (eyes)[33]. All analyses used Statistical Package for the Social Sciences (SPSS) version 16.0 (SPSS Inc., 2007) and SAS version 9.1 (SAS, 2008). The Institutional Review Board at Oregon Health & Science University approved this study.

Results

447 (87.0%) of the cohort were diagnosed with idiopathic intracranial hypertension. Table 5. shows the demographics of this cohort by etiology of intracranial hypertension. A majority of the cohort were female (86.6%), white (91.2%), non-Hispanic (97.1%), and had more than a high school degree/diploma (61.6%). 92.2 % were residents of the United States and 10.7% (N = 55) were disabled at the time of IH Registry enrollment. Average age at diagnosis of intracranial hypertension was 29.0 (*SD* = 12.0) and most diagnoses occurred after the year 2000 (72.2%). The average duration of disease as defined by the length of time in months from diagnosis to the last date of follow-up where

symptoms were still reported by either the physician or the patient was 49.2 months (*SD* = 70.8, Min = 0, Max = 542.0).

Headache was the most common symptom of intracranial hypertension and was present in 93.0% (n = 478) of cases. Blurred vision was reported in 88.7% of cases as was pulse synchronous tinnitus. Table 5. shows the signs and symptoms of intracranial hypertension according to etiology. Papilledema was reported in a majority of the cohort 89.7% (n = 461). The mean highest opening pressure measurement for the idiopathic group was 386 (*SD* = 109.2) and for the secondary group was 361.7 (*SD* = 122.7). At the time of this study, 38.1% had undergone a surgical procedure for intracranial hypertension including shunting, ONSD, subtemporal decompression, gastric bypass, and others.

Etiological Factors.

Since there has been no report of empirical evidence regarding the performance of surgery with regard specifically to the etiology of the chronic intracranial hypertension, we examined the differences and similarities between those with idiopathic and secondary intracranial hypertension. For those with secondary intracranial hypertension (SIH), most had an underlying medical process identified as the etiology (68%). Some examples of these underlying medical conditions were: cerebral venous thrombosis, meningitis, sarcoidosis, subdural hematoma, hyperthyroidism, lupus, and arachnoid cysts. Drug-induced SIH (26%) included use of doxycycline, minocycline, tetracycline, and amiodarone. In 6 % of the SIH group, the disorder was caused by head trauma.

Idiopathic and secondary intracranial hypertension cases did not differ in the rate of surgical intervention. Furthermore, approximately 21% of both the IIH and the SIH groups received shunts and approximately 16% received an ONSD surgery as the first surgical intervention. More women were diagnosed with idiopathic than men, and secondary IH was evenly split between the genders. Moreover, the idiopathic group was more obese than that of the secondary group. With regard to symptomology (headache, TVOs, blurred and double vision, and neck stiffness) , reports were similar between the two etiological groups. Pulse synchronous tinnitus was reported more frequently in the idiopathic group compared to the secondary group (See Table 6.).

Furthermore, the idiopathic and secondary groups did not differ on a variety of visual field measurements and visual acuity either at diagnosis or at equivalent time periods prior to the first surgical intervention (See Table 7.). Since the etiological factors underlying intracranial hypertension did not differ, further analysis examining the preoperative factors leading to surgical intervention will be collapsed.

Predictors of Surgery.

In order to examine the post-operative success of any surgical intervention, it is necessary to examine the factors that lead to the intervention in the first place. In order to establish a preoperative rubric for optic nerve sheath decompression (ONSD), we first compared the preoperative clinical factors between those that remained medically treated (N = 305) to those that underwent either a shunt surgery (N = 121) and those that underwent an ONSD (N = 82) as

the primary (first) surgical procedure. Those that underwent another type of surgical intervention, such as gastric bypass, as the primary procedure were excluded from this portion of the analysis (N = 10). In comparing the signs and symptoms of IH at diagnosis, the two surgical groups (ONSD and shunt) did not differ in IH symptoms and demographics at the time of diagnosis (See Table 8.).

Notably, those that remained medically treated and those that underwent a shunt surgery reported headache at diagnosis more often than those that underwent an ONSD procedure. Furthermore, those that underwent a surgical procedure had worse visual acuity at diagnosis than those that remained medically treated (See Table 9.). The average visual acuity at diagnosis for the medical group was 20/25 in the both eyes, for the shunt group was 20/40 in the right eye and 20/30 in the left eye, and for the ONSD group 20/50 in both eyes. Although gross visual field readings did not differ between the groups, examination of the WaGRS and mean deviation showed that the ONSD and shunt groups had worse visual fields than those that remained medically treated. Although the differences between the shunt and ONSD group were not significant, there was a trend for the ONSD group to have worse visual field findings as measured by WaGRS and mean deviation at diagnosis than the shunt group.

Moreover, when comparing the visual acuity and visual field ratings prior to the first surgery and/or an equivalent time period for the medical only group, the ONSD group had worse visual acuity than the shunt and the medical only group. On average, the medical group and the shunt group remained stable

while the ONSD group declined at least one snellen line (20/50 to 20/60 in the right eye only; See Figure 1). Furthermore the ONSD group had worse visual field defects (more abnormal and worse WaGRS), worse papilledema, less SVPs than the shunt or the medical groups,indicating that the ONSD group had worse manifestation of the visual symptoms of this disorder prior to the first surgical intervention than those of the shunt or those that remained medically treated.

Preoperative Factors.

Of the 1028 eyes in the study sample, 171 eyes of 102 participants underwent an ONSD procedure with 85% (N = 140) as the first surgical intervention for their IH. 37 (22%) had prior shunt surgeries ranging from 1 to 7 surgeries (*M* = 2.4, *SD* = 1.8). 63% of the 171 eyes had no other surgical intervention, 4% had a repeat ONSD, and 41% (N = 70) had 1 to 22 post ONSD surgeries (*M* = 2.8, *SD* = 3.2). Average duration of disease as defined by the date of diagnosis to the time of the first ONSD surgery was 21.2 months (*SD* = 39.2; range 0 – 203 months; median 3 months). 70% of the study sample underwent a bilateral simultaneous optic nerve sheath decompression followed by 18.7% undergoing a bilateral sequential surgery and 19.3% undergoing a unilateral surgery. For those that underwent a bilateral sequential surgery, there was an average of 204 days between the surgeries (Median = 27, *SD* = 490, Min = 3, Max = 2036) and an average of 0.18 surgeries between the first ONSD procedure and the second ONSD procedure (Median = 0, *SD* = 0.54, Max = 2).

Visual Acuity.

A total of 118 of the 171 eyes had complete visual acuity data for the time prior to ONSD surgery and at last follow-up. For the 53 eyes (21 participants) that did not have visual acuity measures post ONSD surgery, ophthalmology records were not received for 5 and 16 were lost to follow-up post ONSD surgery.

The outcome of visual acuity was defined by stabilization and/or improvement of at least 2 Snellen lines within the time just prior to ONSD surgery to the end of the follow-up period. A majority of the study sample experienced stabilization (53 eyes; 44%) of their visual acuity at follow-up with only 35 eyes (29%) showing an improvement. As can be seen in Table 10. , there were no differences between those that stabilized/improved in visual acuity and those that declined for a variety of baseline characteristics such as age at diagnosis, body mass index, and lumbar puncture.

Visual acuity at diagnosis was equivalent between those that stabilized/improved and those that declined after ONSD surgery. In addition, visual acuity was worse for those that stabilized/improved than those that declined preoperatively. Moreover, those that declined after the surgery had a slower rate of visual acuity change preoperatively (See Figure 2). This slower rate of decline in visual acuity preoperatively indicates that those that stabilized/improved were experiencing a more precipitous decline in their visual acuity symptoms prior to surgical intervention. It is important to note that those that stabilized/improved had worse visual acuity (mean 20/70; median 20/40) at last follow-up than those that declined post-ONSD surgery (mean 20/30; median

20/25). Although those that stabilized/improved in visual acuity post-ONSD surgery had worse visual field findings pre-operatively than those that declined post-ONSD, the precipitous rate of decline in visual acuity was not seen with the visual field measurements (WaGRS and mean deviation, See Table 10.).

Based on the univariate analysis of each predictor and visual acuity outcome (Table 10.), the full model included pre-ONSD surgeries, rate of visual acuity change pre-ONSD surgery, papilledema duration in months, visual acuity pre-ONSD, WaGRS pre-ONSD, and months of follow-up. Based on Chandrasekaran et al.[8], duration of disease prior to ONSD surgery was also included. As Table 11. demonstrates, the final model included duration of disease prior to ONSD surgery in months and visual acuity pre-ONSD surgery. The final model indicates that those with worse visual acuity prior to ONSD surgery have a better outcome as measured by visual acuity at last follow-up when adjusting for duration of disease prior to surgery (OR = 0.86, $CI_{95\%}$ 0.77 – 0.96). The final model indicates that ONSD as a surgical intervention may be a good stop gap measure in preserving abnormal vision but that it is not a long-term treatment option for intracranial hypertension.

Visual Fields.

A second examination of visual field outcome was attempted using visual field mean deviation scores at the time of last follow-up. Visual field outcome was measured by severity of visual field defects based on the mean deviation score. A mean deviation score of greater than -10 Db was considered a severe visual field outcome. For those that had defects less than -10 Db, 9 (42.8%)

were considered to have severe defects in their visual fields and 12 (57.2%) were considered to have non-severe defects.

A total of 35 eyes (24 participants) had mean deviation visual field scores at the time of the last follow-up. Examination of those missing mean deviation scores and those with mean deviations scores after ONSD surgery revealed that those missing the mean deviation scores were diagnosed more frequently after 2000 and also, similar to the Wall and George ratings system findings, were more obese at diagnosis.

Univariate analysis of visual field loss with regard to preoperative symptoms did not reveal any significant findings either at diagnosis nor pre-operatively (See

Table **14.**). Multivariate analysis was conducted using a theoretical model based on Chandrasekaran et al.[8]. There were no significant pre-operative factors entered into the model that predicted visual field outcome (See Table 15.).

Wall and George rating system.

Composite scores of visual symptoms was measured by minimal or marked Wall and George rating system (WaGRS) grades at last follow-up. For the 46 eyes (31 participants) that had WaGRS at the time of last follow-up, 50% had none to minimal visual field loss (WaGRS 0 – 2) and 50% had marked visual field loss (WaGRS 3 – 5). Comparison of those without a WaGRS rating at the time of last follow-up found that those without a WaGRS rating were diagnosed after 2000 more often and had a higher body mass index at diagnosis. There

were no differences in visual acuity and visual field symptoms either at diagnosis or at the pre-operative time.

Those with marked visual field loss were more likely to have greater visual field loss as measured by mean deviation and WaGRS at the time of diagnosis than those that had minimal WaGRS field loss (See Table 16). Visual acuity was worse for those with marked visual field loss at the pre-operative time. On average, those with minimal loss had a visual acuity of 20/25 and those with marked visual field loss had a visual acuity of 20/80 to 20/100. Furthermore, those with marked visual loss had worse visual acuity at the time of last follow-up (average 20/60) when compared with those with minimal visual field loss (average 20/20).

Univariate analysis of visual field loss with regard to preoperative symptoms did not reveal any significant findings either at diagnosis nor pre-operatively (See Table 17). Multivariate analysis was conducted using a theoretical model based on Chandrasekaran et al.[8]. There were no significant pre-operative factors entered into the model that predicted visual field outcome (See Table 18).

Discussion

We examined various preoperative factors that lead to surgical intervention for chronic intracranial hypertension from an international registry. In this large sample of intracranial hypertension sufferers, the underlying etiology of the disorder did not contribute to the rate of surgical intervention nor to the choice of surgical intervention. In addition, we compared the symptoms of IH at the time

of diagnosis between the idiopathic and secondary IH groups and found that the secondary group were less obese at diagnosis and had less frequently reported pulsatile tinnitus.

Although there is no explanation as to why the secondary group had lower rates of obesity at diagnosis in the IH literature, it is possible that this result was due to the higher proportion of men in the secondary group. The link between obesity in IH men has not been documented as consistently as the link between obesity and IH women. Moreover, Kesler et al.[25] found that in a retrospective chart review of 123 cases of intracranial hypertension during the time span 1982 to 1999, only 25% of the men were obese while 78% of the women were obese.

Moreover, we found that the secondary group reported tinnitus less frequently than the idiopathic group. This finding has also not been substantiated in the IH literature to date. For idiopathic cases, pulsatile tinnitus was not reported as a symptom of IH as frequently as is found in the literature, although the reported range varies widely among studies. For example, Giuseffi et al.[18] found pulsatile tinnitus present in 58% (of 50), Round et al.[32] found that in 27% (of 101), and Friedman[14] states that 60% of all IIH cases experience pulsatile tinnitus. 30% of our sample of IIH participants reported pulsatile tinnitus.

Similar to Sylaja et al.[41] we did not find any differences in vision between those diagnosed with idiopathic and secondary intracranial hypertension. Since there were no differences found in visual signs, visual acuity, and/or visual fields at the time of diagnosis for the etiology of intracranial hypertension, subsequent analysis was collapsed over the etiological groups.

In examination of the treatment modalities for IH, we found that there were three paths of treatment for the study sample leading to the first surgical intervention. Those that were treated medically had less severe disorder states than those that underwent either a CSF diversion procedure and/or an optic nerve sheath decompression procedure when adjusting for length of time after diagnosis to surgical intervention. Although not statistically significant, those that underwent an ONSD had more severe visual acuity and visual field loss than those that underwent a shunting procedure.

Our findings support the treatment schema put forth by Friedman[14] in 2006, which states that those that fail best medical treatment (diamox up to 4 grams/day) should receive a surgical intervention (Figure 3). Friedman makes no differentiation on when to surgically intervene with an optic nerve sheath decompression or a CSF diversion technique. Our findings suggest that those with worse visual acuity findings preoperatively (20/70) and a larger rate of decline should undergo an optic nerve sheath decompression for preservation of visual acuity.

When examining the preoperative factors leading to long-term visual acuity stabilization, we found that those that stabilized and/or improved at the time of last follow-up had a steeper decline in visual acuity before diagnosis when adjusted for the amount of follow-up. We found that regardless of rate of visual acuity decline preoperatively, those with worse preoperative visual acuity had better visual acuity outcomes when adjusting for the time to surgery. Degree of visual field defect was not related to visual acuity stabilization/improvement.

Unlike Chandrasekaran et al.[8], we found no preoperative factors that predicted visual field outcomes.

Limitations

This study was based on a large international population of intracranial hypertension cases. Similar to the previous preoperative study by Chandrasekaran et al.[8] this study contains referral bias. Participants that enroll in the Intracranial Hypertension Registry are largely self-referred and, therefore, may be those with long-standing difficulties with the disorder. The majority of the participants discover the Registry through on-line searches for information related to intracranial hypertension which may make them more socioeconomically advantaged than those that do not enroll in the Registry and, therefore, could influence their ability to obtain on-going and more expensive medical care.

In addition, the retrospective nature of this study resulted in incomplete data at all three time points in this study. Since the Registry's data is based on medical chart review and the enrolled participants are from around the world, a majority of the work of the Registry is obtaining medical records. Through the medical management of the disorder, an IH patient may see multiple physicians and have various tests performed at numerous hospitals and centers (such as MRIs, CTs, and visual fields). Although the Registry attempts to obtain all medical records pertaining to this disorder, the feasibility of obtaining all records on all participants is slim. The nature of the missing follow-up data in this study was examined against those that had complete data sets. For the outcome of

visual acuity, missing data was due to either the participant being lost to follow-up post ONSD surgery or lack of ophthalmology medical records. These participants were evaluated against those that had complete follow-up data and did not differ in visual symptoms prior to surgery.

For the outcome of visual fields, a small sub-sample of the study population was used. This small sample was largely due to incomplete data at last follow-up (74%) of those undergoing ONSD surgery. Moreover, data was not available to examine the change pre- versus post- surgical intervention. Therefore, the visual field results in this study should be viewed with caution.

For the purposes of this initial examination of preoperative factors affecting the success rates of ONSD surgery, no attempt was made to examine either the type of the surgical incision (windows versus slits), the approach to the optic nerve (medial versus lateral), or the manner of ONSD (unilateral, bilateral, or bilateral simultaneous). To date, there have been no controlled studies examining the outcomes of the type of surgical incision (windows versus slits) and it has been suggested that making several slits in the optic nerve sheath increases the chances of the opening staying patent after surgery[37] and thus, would increase the postoperative success rates. Moreover, in approximately 1990, the lateral approach to the optic nerve was replaced with the medial approach based on a number of factors. These factors included the need for general anesthesia, duration of surgery, difficulty of accessing the optic nerve, and number of complications post lateral ONSD[44]. Similar to the surgical incision, there have been no controlled studies examining the approach to the

optic nerve[36]. Furthermore there is an indication that within 5 years of follow-up, the type of ONSD does influence the initial postoperative success as measured by need for subsequent CSF diversion procedures[5].

Future Studies

Future studies need to explore the surgical factors that may predict postoperative success such as type of surgical incision, type of approach, type of ONSD, and duration of surgery. Few studies in the medical literature on ONSD discuss the type of surgical incision made to the optic nerve to alleviate the swelling caused by ICP. At the current time, the type of the surgical incision made depends upon the preference of the operating physician. Although Sergott et al.[37] suggest the use of slits to increase the chance of the opening staying patent, the pathophysiologic explanation for this suggestion remains nebulous and depends upon the surgeon's preference. Table 19 shows the reported improvement and stabilization success rates for the type of surgical approach used during surgery. Although discussion in the literature leans towards supporting the medial approach to the optic nerve, there have been no controlled studies comparing the outcome of this type of surgical intervention[36, 44].

Additionally, the type of ONSD operation needs further exploration. Benes et al.[5] suggest that a bilateral simultaneous approach may reduce the need for additional surgeries when ONSD is performed as the first surgical intervention for IH. This initial examination of the type of ONSD did not consider preoperative factors that may influence the postoperative success and future investigation should evaluate the effect of these factors. Finally, it has been suggested that

the length of the surgery may impact the success rates of ONSD[21]. Thus far no controlled studies have examined the length of surgery on the long-term efficacy of ONSD for IH.

Summary and Conclusions

In this study of a large retrospective cohort of chronic intracranial hypertension cases, we found that the signs and symptoms of chronic intracranial hypertension are not influenced by etiology. Those that remain medically treated have less visual symptoms than those that underwent a surgical procedure and those that received a shunt had slightly better visual symptoms than those that underwent a optic nerve sheath decompression. Similar to Chandrasekaran et al.[8], the preoperative factors that define intracranial hypertension were poor predictors of visual outcomes (both visual acuity and visual fields) post-ONSD surgery. Moreover, those with poorer visual acuity pre-ONSD had better outcomes in visual acuity when examining stabilization/improvement at the time of last follow-up.

References

1. Acheson, J. F., Green, W. T., & Sanders, M. D. (1994). Optic nerve sheath decompression for treatment of visual failure in chronic raised intracranial pressure. Journal of Neurology, Neurosurgery, and Psychiatry, 57, 1426 - 1429.
2. Agarwal, M. R., & Yoo, J. H. (2007). Optic nerve sheath fenestration for vision preservation in idiopathic intracranial hypertension. Neurosurgical Focus, 23, E7.
3. Balcer, L. J., Liu, G. t., Forman, s., Pun, K., Volpe, N. J., Galetta, S. L., et al. (1999). Idiopathic intracranial hypertension: relation of age and obesity in children. *Neurology, 52,* 870 – 872.
4. Banta, J. T., & Farris, B. K. (2000). Pseudotumor cerebri and optic nerve sheath decompression. Ophthalmology, 107, 1907 - 1912.
5. Benes, S. (2004, February). *Does the choice of primary surgical procedure correlate with a significant difference in total number of surgeries needed in medical failure patients with intracranial hypertension (pseudotumor cerebri)? Comparing those undergoing optic nerve sheath decompression versus CSF shunting as their primary surgery.* Poster session presented at the annual meeting of the North American Neuro-Ophthalmology Society, Copper Mountain, CO.
6. Brourman, N. D., Spoor, T. C., & Ramocki, J. M. (1988). Optic nerve sheath decompression for pseudotumor cerebri [case report]. Archives of Ophthalmology, 106, 1378 - 1383.

7. Burgett, R. A., Purvin, V. A., & Kawasaki, A (1997). Lumboperitoneal shunting for pseudotumor cerebri. Neurology, 49, 734 - 739.

8. Chandrasekaran, S., McClusky, P., Minassian, D., & Assaad, N. (2006). Visual outcomes for optic nerve sheath fenestration in pseudotumour cerebri and related conditions. Clinical and Experimental Ophthalmology, 34, 661 - 665.

9. Corbett, J. J., Nerad, J. A., Tse, D. T., & Anderson, R. L. (1988). Results of optic nerve sheath fenestration for pseudotumor cerebri: the lateral orbitotomy approach. Archives of Ophthalmology, 106, 1391 - 1397.

10. Corbett, J. J., Savino, P. J., Thompson, S., Kansu, T., Schatz, N. J., Orr, L. S., et al. (1982). Visual loss in pseudotumor cerebri: Follow-up of 57 patients from five to 41 years and a profile of 14 patients with permanent severe visual loss. *Archives of Neurology, 39,* 461 – 474.

11. Curry, Jr, W., Butler, W., Barker, F. (2005). Rapidly rising incidence of cerebrospinal fluid shunting procedures for idiopathic intracranial hypertension in the United States, 1988-2002. *Neurosurgery, 57*, 97 – 107.

12. Digre, K. B., & Corbett, J. J. (2001). Idiopathic intracranial hypertension (pseudotumor cerebri): A reappraisal. *The Neurologist, 7,* 2 – 67.

13. Durcan, F. J., Corbett, J. J., & Wall, M. (1988). The incidence of pseudotumor cerebri: Population studies in Iowa and Louisiana. *Archives of Neurology, 45,* 875 – 877.

14. Friedman, D. I. (2004). Pseudotumor cerebri. *Neurological Clinics of North America, 22,* 99-131.

15. Friedman, D. I., & Jacobson, D. M. (2004). Idiopathic intracranial hypertension. Journal of Neuro-Ophthalmology, 24, 138 - 145.

16. Friesner, D., Lobb, B. M., Rosenman, R., & Tanne, E. (in preparation). The costs of a quiet disorder: Estimates of direct and indirect costs of idiopathic intracranial hypertension.

17. Frisén, L. (1982). Swelling of the optic nerve head: A staging scheme. *Journal of Neurology, Neurosurgery, and Psychiatry, 45,* 13 – 18.

18. Giuseffi, V., Wall, M., Siegel, P. Z., & Rojas, P. B. (1991). Symptoms and disease associations in idiopathic intracranial hypertension (pseudotumor cerebri): A case-control study. *Neurology, 41,* 239.

19. Goh, K. Y., Schatz, N. J., & Galser, J. S. (1997). Optic nerve sheath fenestration for pseudotumor cerebri. Journal of Neuro-Ophthalmology, 17, 86 - 91.

20. Gordon, K. (1997). Pediatric pseudotumor cerebri: Descriptive epidemiology. *Canadian Journal of Neurological Sciences, 24,* 219 – 221.

21. Katz, S. (2008, June). *Saving sight: Optic nerve sheath decompression one surgeon's personal experience with 300 procedures.* Paper presented at the 2008 Intracranial Hypertension Research Foundation Patient Conference, Houston, TX.

22. Kelman, S. E., Heaps, R., Wolf, A., & Elman, M. J. (1992). Optic nerve decompression surgery improves visual function in patients with psudotumor cerebri. Neurosurgery, 30, 391 - 395.

23. Kelman, S. E., Sergott, R. C., Cioffi, G. A., Savino, P. J., Bosley, T. M., & Elman, M. J. (1991). Modified optic nerve sheath decompression in patients with functioning lumboperitoneal shunts and progressive visual loss. Ophthalmology, 98, 1449 - 1453.

24. Keltner, J. (1988). Optic nerve sheath decompression. How does it work? Has its time come?. Archives of Ophthalmology, 106, 1365 - 1369.

25. Kesler, A., Goldhammer, Y., & Gadoth, N. (2001). Do men with pseudotumor cerebri share the same characteristics as women? A retrospective review of 141 cases. *Journal of Neuro-Ophthalmology, 21,* 15 – 17.

26. NCSS. (2008). Power analysis and sample size. Retrieved August 10, 2008 from www.ncss.org

27. Nithyanandam, S., Manayath, G. J., & Battu, R. R. (2008). Optic nerve sheath decompression for visual loss in intracranial hypertension: Report from a tertiary care center in south India. Indian Journal of Ophthalmology, 56, 115 - 120.

28. Plotnik, J. L., & Kosmorsky, G. S. (1993). Operative complications of optic nerve sheath decompression. Ophthalmology, 100, 683 - 690.

29. Radhakrishanan, K., Ahlskog, J. E., Cross, S. A., Kurland, L. T., O'Fallon, W. M. (1993). Idiopathic intracranial hypertension (pseudotumor cerebri):

Descriptive epidemiology in Rochester, Minn, 1976 to 1990. *Archives of Neurology, 50,* 78 – 80.

30. Radhakrishanan, K., Sridharan, R., Ashok, P. P., & Mousa, M. E. (1986). Pseudotumor cerebri: Incidence and patter in North-Eastern Libya. *European Journal of Neurology, 25,* 117 – 124.
31. Rosenburg, M. L., Corbett, J. J., Smith, C., Goodwin, j., Sergott, R., Savino, P., & Schatz, N. (1993). Cerebrospinal fluid diversion procedures in pseudotumor cerebri. Neurology, 42, 1071 - 1072.
32. Round, R., & Keane, J. R. (1988). The minor symptoms of increased intracranial pressure: 101 patients with benign intracranial hypertension. *Neurology, 38,* 1461 – 1464.
33. SAS. (nd). Production GLIMMIX Procedure. Retrieved February 28, 2009 from www.support.sas.com/rnd/app/da/glimmix.html
34. Schirmer, C. M., & Hedges, III, T. R. (2007). Mechanisms of visual loss in papilledema. *Neurosurgical Focus, 23,* 1 – 7.
35. Seiff, S. R., & Shah, L. (1990). A model for the mechanism of optic nerve sheath fenestration. *Archives of Ophthalmology, 108,* 1326 – 1329.
36. Sergott, R. C. (1991). Optic nerve sheath decompression: History, techniques, and indications. International Ophthalmology Clinical, 31, 71 - 81.
37. Sergott, R. C., Savino, P. J., & Bosley, T. M. (1988). Modified optic nerve sheath decompression provides long-term visual improvement for psudotumor cerebri. Archives of Ophthalmology, 106, 1384 - 1390.

38. Smith, J. K., Hoyt, W. F., & Newton, T. H. (1969). Optic nerve sheath decompression for relief of chronic monocular choked disc. American Journal of Ophthalmology, 68, 633 - 639.

39. Spoor, T. C., & McHenry, J. G. (1993). Long-term effectiveness of optic nerve sheath decompression fro psudotumor cerebri. Archives of Ophthalmology, 111, 632 - 635.

40. Spoor, T. C., Ramocki, J. M., Madion, M. P., & Wilkinson, M. J. (1991). Treatment of pseudotumor cerebri and secondary optic nerve sheath decompression. American Journal of Ophthalmology, 112, 177 - 185.

41. Sylaja, P. N., Ahsan Moosa, N. V., RAdhakrishanan, K., Sankara Sarma, P., & Pradeep Kumar, S. (2003). Differential diagnosis of patients with intracranial sinus venous thrombosis related isolated intracranial hypertension from the with idiopathic intracranial hypertension. *Journal of the Neurological Sciences, 215,* 9 – 12.

42. Thuente, D. D., & Buckley, E. G. (2005). Pediatric optic nerve sheath decompression. Ophthalmology, 112, 724 - 727.

43. Tsai, J. C., Petrovich, M. S., & Sadun, A. A. (1995). Histopathological and ultrastructural examination of optic nerve sheath decompression. British Journal of Ophthalmology, 79, 182 - 185.

44. Tse, D. T., Nerad, J. A., Anderson, R. L., & Corbett, J. J. (1988). Optic nerve sheath fenestration in pseudotumor cerebri. A lateral orbitotomy approach. Archives of Ophthalmology, 106, 1458 - 1462.

45. Wall, M., & George, D. (1991). Idiopathic intracranial hypertension: A prospective study of 50 patients. *Brain, 114,* 155 – 180.

Table 1. The efficacy of ONSD as reported by the IH literature.

Authors (Date)	Improvement /Stabilization Reported				
	Visual Acuity	Visual Fields	Total Number of Patients	Total Number of Eyes	Mean Follow-Up[Ψ]
Banta & Farris (2000)[4]	94%	88%	86	158	20
Chandrasekaran et al. (2006)[8]	42%	42%	32	51	6
Thuente & Buckley (2005)[42]	64%	55%[e]	12	17	39.6
Agarwal & Yoo (2007)[2]	100%	--	10	--	--
Goh et al. (1997)[19]	52%[e]	82%	19	29	15.7
Spoor & McHenry (1993)[39]	68%	--	54	75	6 – 60
Acheson et al. (1994)[1]	73%	85%	14	20	12
Tsai et al. (1995)[43]	100%	95%	1	2	10
Tse et al. (1988)[44]	95%[e]	95%[e]	--	--	17
Sergott et al. (1988)[37]	100%	100%	23	--	22
Brourman et al (1988)[6]	100%[e]	83%	6	10	4
Corbett et al. (1988)[9]	85%	82%	28	40	1.2
Kelman et al. (1991)[23]	50%	88%[e]	12	16	31
Rosenburg et al. (1993)[31]	66%[e]	--	--	--	17.9
Lui et al. (1996)[3]	31%[e]	--	1	--	14.6
Average:	75%	81%	298	418	20.9

[Ψ]Months
[e]Eyes

Table 2. The modified Dandy criteria used by the IHR for a diagnosis of idiopathic intracranial hypertension.

The modified Dandy criteria
Signs and symptoms of increased intracranial pressure
No localizing findings on neurologic exam
Normal MRI/CT with not evidence of venous obstructive disease
Opening CSF pressure : > 25 cm H_2O with normal CSF constituents
Awake and Alert patient
No known cause for increased pressure found

Table 3. Papilledema classification and Frisén Grade categories.

Papilledema[17]		
Classification	**Frisén Grade**	**Description**
None	0	Normal optic disc
Early	1	Mild disc blurring a. blurring nasal border b. temporal bored intact c. subtle grayish halo along circumference with a temporal gap
Early	2	Mild disc blurring a. elevation off all nasal circumference b. blurring of all temporal margin c. grayish halo all around optic disc
Fully Developed	3	Moderate disc elevation a. elevation of entire circumference b. increased diameter of optic nerve head
Fully Developed	4	Severe disc elevation a. Elevation of entire nerve head b. Obliteration/compression of optic cup
Chronic	5	Severe disc elevation a. Optic nerve head assumes smooth dome-shape protrusion b. smoothly demarcated halo c. Vessels may or may not be obscured

Table 4. Wall and George visual field grading system.

Visual Fields[45]	
Goldmann Perimetry	
Grading	**Description / Criteria**
0	Normal VF
1	Minimal VF Loss 1. Isopter constriction: a. step defects < 10° and > 5° 2. Defects not involving fixation: a. relative scotomas that do not involve fixation (1). Up to 20° x 20° in area outside 30° (2) up to 10° in area inside 30° b. blind spot enlargement – involves central 10° but not fixation 3. Defects involving fixation: none – visual acuity is 20/20 or better
2	Mild VF Loss 1. Isopter constriction a. Up to 20° in area b. $I4_e$ isopter inside 30° nasally, 50° temporally c. $I2_e$ isopter inside 20° 2. Defects not involving fixation: a. relative: less than on quadrant in size, defect greater than criteria of 1.B.2.a. b. absolute: less than 20° x 20° in area 3. Defects involving fixation: acuity 20/30 or better
3	Moderate VF Loss 1. Isopter constriction a. greater than 20° to any isopter but more than 50° of field to the $V4_e$ stimulus b. $I3_e$ isopter inside blind spot c. $I2_e$ isopter inside 10° 2. Defects not involving fixation a. relative: greater than on quadrant but less than on hemifield b. absolute: greater than 20° x 20 ° in diameter but less than one quandrant 3. Defects involving fixation – acuity greater than 20/30 but less than 20/100
4	Marked VF Loss 1. Constriction - < 50° but greater than 20° in diameter to $V4_e$ 2. Defects not involving fixation a. relative: one hemifield or greater with more than 20° of field left to $V4_e$ b. absolute: greater than one quadrant with more than 20° to $V4_e$ stimulus 3. Defects involving fixation – acuity 20/100 to 20/200
5	Blinding VF Loss 1. Constriction – less than 20° to $V4_e$ 2. Acuity worse than 20/200

Table 4. Wall and George visual field grading system (continued).

Automated Perimetry Grading	**Description / Criteria**
0	Normal VF
1	Minimal VF Loss 1. Deficit no greater than 3 contiguous points abnormal, no point with loss greater than 10 dB 2. Enlarged blind spot defined by presence of 5 or more disturbed points adjacent to the blind spot with no encroachment into the central 10° 3. Defects involving fixation – none, acuity 20/20 or better
2	Mild VF Loss 1. Defect greater than criteria for deficit 1 2. Enlarged blind spot defined by presence of 5 or more disturbed points adjacent to the blind spot with encroachment into the central 10° 3. Defects involving fixation – less than 10dB loss; acuity 20/20 or better
3	Moderate VF Loss 1. Isopter constriction – all points abnormal in one isopter 2. Defects not involving fixation: a. relative: greater than on quadrant (all points involved) but less than one hemifield b. absolute: greater than 20° x 20° in diameter but less than one quadrant 3. Deficits involving fixation – acuity worse than 20/30 but less than 20/100; blind spot encroaching on fixation (relative defect) greater than 10dB loss
4	Marked VF Loss 1. Constriction – less than 50° but more than 20° to brightest stimulus 2. Defects not involving fixation: a. relative: on hemifield or greater with more than 20° of field left to brightest stimulus b. absolute: greater than one quadrant with more than 20° to brightest stimulus 3. Defects involving fixation a. acuity worse than 20/100 to 20/200 b. blind spot encroaching on fixation (relative defect) greater than 2 log units of loss
5	Blinding VF Loss 1. Constriction – Less than 20° to brightest stimulus 2. Acuity worse than 20/200 or greater than 3 log unit loss at fixation

Table 5. Demographic characteristics of chronic intracranial hypertension retrospective cohort.

	Etiology				
	Idiopathic		**Secondary**		
	N	**%**	**N**	**%**	***P****
Gender					
Female	405	90.6	40	59.7	<.0001
Male	42	9.4	27	40.3	
Race					
White	405	90.6	64	95.5	
African American	24	5.4	2	3.0	
Other	10	2..2	1	1.5	
Unknown	8	1.8	0	0	
Ethnicity					
Non-Hispanic	432	96.6	67	100	
Hispanic	15	3.4	0	0	
Education					
High School or Less	136	36.4	29	52.8	
College/Trade School	207	55.3	18	34.0	
Graduate School	31	8.3	7	13.2	
Age at Diagnosis					
≤ 15	59	13.2	19	28.4	
16 – 23	82	18.3	9	13.4	
24 – 44	268	60.0	28	41.8	
45 +	38	8.5	11	16.4	0.82
IH Treatment					
Medical Only	536	60.0	74	55.2	
Medical & Surgical	358	40.0	60	44.8	

*Chi-square tests

Table 6. Etiological differences in chronic intracranial hypertension at diagnosis.

	Etiology				
	Idiopathic		Secondary		
	N	%	N	%	P*
Diagnosis Year					
< 2000	126	24.5	17	3.3	
>= 2000	321	86.5	50	9.7	0.63
Headache					
None	30	5.8	6	1.2	
Present	417	81.1	61	11.9	0.50
TVOs					
None	78	15.2	17	3.3	
Present	369	71.8	50	9.7	0.12
Blurred vision					
None	50	9.7	8	1.6	
Present	397	77.2	59	11.5	0.86
Pulsatile Tinnitus					
None	292	56.8	57	11.1	
Present	155	30.2	10	1.9	.001
Diplopia					
None	96	18.7	13	2.5	
Present	351	68.3	54	10.5	0.69
Neck Stiffness					
None	59	11.5	9	1.8	
Present	388	75.5	58	11.3	0.96

	M	SD	M	SD	P**
Body Mass Index	35.3	9.2	31.4	8.0	0.007
Highest lumbar puncture	370.2	106.8	367.1	115.0	0.84
Lumbar puncture at diagnosis	337.3	97.7	369.2	110.0	0.11
Age at diagnosis	29.0	11.4	28.6	15.4	0.77
Follow-up (mo)	48.1	67.5	56.2	81.5	0.39

TVOs = Transient Visual Obscurations; BMI = Body Mass Index; LP = Lumbar Puncture
*Chi-square test
**T-test

Table 7. Differences at diagnosis in visual symptoms by intracranial hypertension etiology.

	Right					Left				
	IIH		SIH			IIH		SIH		
	N	%	N	%	P	N	%	N	%	P*
Visual Fields										
Normal	10	10.9	4	26.7		8	8.9	2	13.3	
Abnormal	82	89.1	11	73.3	0.11	82	91.1	13	86.7	0.43
Fundus										
Normal	17	16.2	5	38.5		16	15.0	5	35.7	
Abnormal	88	83.8	8	61.5	0.07	91	85.0	9	64.3	0.07
Papilledema										
Normal	19	17.9	5	38.5		20	18.5	5	35.7	
Abnormal	87	82.1	8	61.5	0.09	88	81.5	9	64.3	0.13
Papilledema Grade										
Early	75	69.8	11	84.6		76	69.4	12	85.7	
Fully Developed	9	8.5	1	7.7		10	9.3	1	7.1	
Chronic	22	21.7	1	7.7	0.68	22	21.3	1	7.1	0.62
SVP										
Present	91	85.8	12	92.3		91	84.3	13	92.9	
Absent	15	14.2	1	7.7	0.45	17	15.7	1	7.1	0.35
	M	SD	M	SD	P	M	SD	M	SD	P**
Visual Acuity	1.8	3.5	1.8	3.7	0.97	1.5	2.7	3.3	5.6	0.04
WaGRS	1.8	1.5	1.7	1.4	0.68	1.7	1.4	2.3	1.6	0.18
Mean Deviation	-6.5	8.2	-3.1	3.8	0.18	-5.9	7.3	-5.7	7.3	0.93

IIH = Idiopathic; SIH = Secondary; SVP = Spontaneous Venous Pulsations; WaGRS = Wall & George Rating System

*****Chi-square test

**T-Test

Table 8. Differences at diagnosis for those receiving a surgical intervention from those who remained medically treated.

	Medically Managed		ONSD		Shunt		
	N	**%**	**N**	**%**	**N**	**%**	***P****
	306	60.1	82	16.1	121	23.8	
Gender							
Female	263	51.7	74	14.5	105	20.6	
Male	43	8.5	8	1.6	16	3.1	0.59
Etiology							
Idiopathic	269	52.9	71	13.9	104	20.4	
Secondary	37	7.3	11	2.2	17	3.3	0.85
Diagnosis Year							
< 2000	54	10.6	30	5.9	57	11.2	
>= 2000	252	49.5	52	10.2	64	12.6	<.0001
Headaches							
None	23	4.5	10	1.9	3	0.6	
Present	283	55.6	72	14.2	118	23.2	0.03
TVOs							
None	51	10.0	19	3.7	25	4.9	
Present	255	50.1	63	12.4	96	18.9	0.33
Blurred vision							
None	39	7.7	5	1.0	13	2.6	
Present	267	52.5	77	15.1	108	21.2	0.24
Pusatile Tinnitus							
None	205	40.3	58	11.4	82	16.1	
Present	101	19.8	24	4.7	39	7.7	0.81
Diplopia							
None	67	13.2	19	3.7	22	4.3	
Present	239	46.9	63	12.4	99	19.5	0.63
Neck Stiffness							
None	40	7.9	15	3.0	14	2.8	
Present	266	52.3	67	13.2	107	21.0	0.36

	M	**SD**	**M**	**SD**	**M**	**SD**	***P****
BMI at Diagnosis	35.0	8.6	35.7	10.5	33.6	9.3	0.32
High LP	350.6	90.6	368.4	113.0	402.6	136.6	<.0001
LP at Diagnosis	337.6	92.9	333.7	103.1	343.6	108.5	0.85
Age at Diagnosis	28.6	12.7	30.0	11.8	28.4	11.4	0.62
Follow-up (mo)	36.0	61.7	67.6	81.7	68.9	72.5	<.0001

TVOs = Transient Visual Obscurations; BMI = Body Mass Index; LP = Lumbar puncture

*****Chi-square test

**T-Test

Table 9. Visual Acuity and Visual Field findings by eye for those remaining medically treated and undergoing a surgical intervention at the time of diagnosis.

	Right Eye							Left Eye						
	Medical		ONSD		Shunt			Medical		ONSD		Shunt		
	N	%	N	%	N	%	P*	N	%	N	%	N	%	P*
Visual Fields														
Normal	26	11.6	2	0.9	7	3.1		27	11.9	1	0.4	7	3.1	
Abnormal	127	56.7	28	12.5	28	12.5	0.35	126	55.5	31	13.7	35	15.4	0.11
Fundus														
Normal	48	16.6	3	1.0	18	6.2		47	16.0	4	1.4	18	6.1	
Abnormal	159	54.8	21	7.2	41	14.1	0.20	161	54.8	22	7.5	42	14.3	0.29
Papilledema														
Normal	52	17.8	5	1.7	21	7.2		54	18.2	5	1.7	22	7.4	
Abnormal	156	53.2	21	7.2	38	12.9	0.18	155	52.2	23	7.7	38	12.8	0.13
Papilledema Grade														
Early	142	48.5	19	6.5	41	14.0		145	48.8	18	6.1	41	13.8	
Fully Developed	29	9.9	2	0.7	5	1.7		28	9.4	4	1.4	7	2.4	
Chronic	37	12.6	5	1.7	13	4.4	0.71	36	12.1	6	2.0	12	4.0	0.96
SVP														
Present	191	65.2	23	7.9	52	17.8		191	64.3	25	8.4	53	17.9	
Absent	1	75.8	3	1.0	7	2.4	0.63	18	6.1	3	1.0	7	2.4	0.75
	M	SD	M	SD	M	SD	P**	M	SD	M	SD	M	SD	P**
Visual Acuity	1.1	2.0	4.0	5.8	2.8	4.5	<.0001	1.4	2.8	3.7	5.9	2.3	3.8	<.0001
Wall & George	1.1	1.2	1.8	1.4	1.6	1.4	0.012	1.1	1.3	2.0	1.4	1.2	1.3	0.007
Mean Deviation	-4.0	5.5	-9.5	9.6	-6.5	7.6	0.001	-4.3	5.1	-9.8	10.2	-5.5	7.5	0.001

SVP = Spontaneous Venous Pulsations
*****Chi-square test
**T-Test

Table 10. Preoperative and postoperative factors by those that declined and stabilized/improved in visual acuity after surgery.

Panel A. Preoperative factors at time of diagnosis.

	Visual Acuity				
	Decline		Stable/Improve		
	N	%	N	%	P*
VA	53	44.9	65	55.1	
Etiology					
Idiopathic	46	39.0	55	46.6	
Secondary	7	5.9	10	8.5	0.74
Gender					
Female	48	40.7	57	48.3	
Male	5	4.2	8	6.8	0.77
ONSD type					
Unilateral[reference]	11	9.3	16	13.6	
Bilateral Simultaneous	32	27.1	31	26.3	0.38
Bilateral Sequential	10	8.5	18	15.3	0.70
Diagnosis Year					
Before 2000	24	20.3	29	24.6	
After 2000	29	24.6	36	30.5	0.94
Pre ONSD Surgeries					
No	46	39.0	46	39.0	
Yes	7	5.9	19	16.1	0.04
Post ONSD Surgeries					
No	35	29.7	35	29.7	
Yes	18	15.3	30	25.4	0.18
VF Mean Deviation					
> -20 Db	50	42.4	50	42.4	
<= - 20 Db	3	2.5	15	2.7	0.001

	N	M	SD	M	SD	P**
LP	74	339.7	108.4	345.6	112.1	0.82
BMI at diagnosis	97	33.9	10.3	35.6	11.9	0.49
Age	114	29.3	11.7	30.1	12.1	0.71
Highest LP	114	398.5	126.1	374.1	108.6	0.27
Visual Acuity	95	2.2	4.6	3.7	5.5	0.15
Mean Deviation	30	-4.7	7.4	-15.4	10.2	0.003
WaGRS	43	1.3	1.0	2.6	1.3	<.0001
Number of Pre ONSD surgeries	118	0.4	0.3	0.5	0.2	0.66
Number of Post ONSD surgeries	118	1.2	3.4	1.3	2.1	0.88
Pap Duration (mo)	117	6.0	13.6	15.6	26.6	0.02

Panel B. Preoperative factors at time of ONSD surgery				
	Visual Acuity			
	Decline		**Stable/Improve**	
	N	**%**	**N**	**%**	***P****
Papilledema*					
Absent	6	5.9	12	11.9	
Present	38	37.6	45	44.6	0.33
Visual Fields*					
Normal	1	1.2	1	1.2	
Abnormal	36	42.9	46	54.8	0.99
Disease Duration					
<= 6 mo	31	26.3	32	27.1	
> 6 mo	22	18.6	33	27.9	0.32

	N	**M**	**SD**	**M**	**SD**	**P****
Age	118	31.4	11.4	32.0	12.2	0.77
Visual Acuity	118	2.0	4.2	6.0	6.0	<.0001
Visual Acuity Change	95	0.2	-0.6	-2.7	-3.9	0.01
Rate Visual Acuity Change	95	-0.1	1.9	-2.5	5.1	0.003
WaGRS	79	2.1	1.5	3.2	1.3	0.002
WaGRS Change	40	-1.1	1.1	-0.9	1.4	0.62
Rate of WaGRS Change	32	0.4	0.4	0.3	0.8	0.65
Mean Deviation	74	-8.6	8.0	-15.1	10.2	0.004
Mean Deviation Change	27	3.7	4.1	4.4	7.5	0.74
Rate of Mean Deviation Change	22	-1.4	3.3	-0.9	3.6	0.73
Duration of Disorder (mo)	118	25.1	46.2	25.2	43.6	0.99

Panel C. Post-operative factors at time of last follow-up					
	Visual Acuity				
	Decline		**Stable/Improve**		
	N	**%**	**N**	**%**	**P***
Papilledema					
Absent	22	19.0	33	28.5	
Present	29	25.0	32	27.6	0.41
Visual Fields					
Normal	4	10.8	3	8.1	
Abnormal	11	29.7	19	51.4	0.32
VF Mean Deviation					
Decline	33	28.0	39	33.0	
Stable/Improve	20	17.0	26	22.0	0.80
Time of Follow-up (mo)					
<= 2 yr	28	23.7	30	25.4	
> 2 yr	25	21.2	35	29.7	0.47

	N	M	SD	M	SD	P**
Time of Follow-Up (mo)	118	54.4	62.8	54.0	60.9	0.98
Visual Acuity	118	2.0	4.1	5.6	6.1	0.0002
Change in Visual Acuity	118	0.1	0.7	0.4	7.6	0.71
WaGRS	35	2.1	1.8	2.8	1.9	0.30

*****Chi-square test
****T-Test**

Table 11. Univariate mixed-effects logistic regression analysis of visual acuity outcome for optic nerve sheath decompression cases.

Panel A. Preoperative factors at diagnosis.							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Etiology	0.14	0.72	33	0.83	1.17	0.27	5.01
Gender	0.22	0.84	33	0.79	1.25	0.23	6.82
Body Mass Index	-0.01	0.03	27	0.66	0.99	0.94	1.04
Age	-0.01	0.02	32	0.84	0.99	0.94	1.04
Diagnosis Year	0.00	0.52	33	0.99	1.00	0.35	2.88
Highest Lumbar Puncture	0.00	0.00	31	0.44	1.00	0.99	1.01
Lumbar Puncture	-0.01	0.00	21	0.75	0.99	0.99	1.01
Papilledema Present	0.86	1.12	14	0.74	2.36	0.20	28.32
ONSDtype *							
Bilateral Simultaneous	0.43	0.62	25	0.49	1.54	0.43	5.55
Bilateral Sequential	-0.19	0.65	8	0.78	0.83	0.18	3.76
Visual Acuity	-0.06	0.06	26	0.32	0.94	0.84	1.06
iVFmd (-20 Db)	1.45	0.79	31	0.08	4.27	0.84	21.63
Mean Deviation	0.13	0.07	10	0.09	1.13	0.98	1.32
WaGRS	-0.97	0.44	13	0.05	0.38	0.15	0.98
Panel B. Preoperative factors at the time of ONSD surgery.							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Age	-0.04	0.02	32	0.87	0.99	0.95	1.04
Duration of Disease (≤ 6 mo)	0.37	0.52	33	0.48	1.45	0.51	4.15
Duration of Disease (mo)	0.001	0.00	32	0.99	1.00	0.99	1.01
Pre Surgeries	1.01	0.63	32	0.12	2.75	0.77	9.83
Number of pre surgeries	-0.07	0.19	32	0.73	0.94	0.63	1.38
Post surgeries	0.51	0.52	32	0.33	1.66	0.58	4.79
Number of post surgeries	-0.01	0.09	32	0.99	0.99	0.83	1.20
Papilledema Present	0.06	0.04	14	0.17	1.06	0.97	1.16
Duration of Papilledema (mo)	-0.02	0.01	30	0.13	0.98	0.95	1.01
Visual Field (gross reading)	0.04	1.89	26	0.99	1.04	0.02	50.87
Visual Acuity	-0.15	0.05	32	0.01	0.86	0.78	0.96
Visual Acuity Change	0.12	0.29	26	0.10	1.12	0.98	1.29
Rate of Visual Acuity Change	0.19	0.09	26	0.05	1.21	0.99	1.48
Mean deviation	0.07	0.04	22	0.06	1.08	1.00	1.16
WaGRS	-0.53	0.24	25	0.04	0.59	0.36	0.96
Panel C. Postoperative factors at time of last follow-up							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Follow-up time (mo)	-0.01	0.00	32	0.99	1.00	0.99	1.01
Follow-up Time Group	00.28	0.52	33	0.59	0.76	0.26	2.16

WaGRS = Wall and George rating system

*reference group unilateral.

Table 12. Multivariate mixed-effects logistic regression of preoperative factors leading to stabilization/improvement in visual acuity at the end of the follow-up period.

				CI (95%)	
Model	**Preoperative Factors**	**P**	**OR**	**L**	**U**
1	Duration of Disease (≤ 6mo)	0.23	0.16	0.01	3.56
	Pre-ONSD surgeries	0.53	1.04	0.67	1.60
	Rate Visual Acuity Change (pre surgery)	0.86	1.01	0.94	1.07
	Papilledema Duration (mos)	0.88	0.56	0.35	0.90
	Visual Acuity at ONSD	0.02	0.91	0.46	1.83
	WaGRS at ONSD	0.79	2.14	0.20	22.36
	Time of Follow-up (mos)	0.50	2.76	0.10	77.61
2	Duration of Disease (≤ 6 mo)	0.14	0.19	0.02	1.87
	Pre-ONSD surgeries	0.52	1.04	0.68	1.58
	Rate Visual Acuity Change (pre Surgery)	0.85	0.57	0.36	0.89
	Visual Acuity at ONSD	0.02	0.91	0.46	1.78
	WaGRS at ONSD	0.77	1.97	0.25	15.41
	Time of Follow-up (mos)	0.50	2.44	0.14	43.38
3	Duration of Disease (≤ 6 mo)	0.24	0.32	0.05	2.26
	Pre-ONSD surgeries	0.37	0.66	0.48	0.90
	Visual Acuity at ONSD	0.01	0.72	0.41	1.27
	WaGRS at ONSD	0.24	2.31	0.36	14.76
	Time of Follow-up (mos)	0.36	2.69	0.28	25.43
4	Duration of Disease (≤ 6 mo)	0.35	0.46	0.08	2.49
	Pre-ONSD surgeries	0.38	0.68	0.51	0.92
	Visual Acuity at ONSD	0.01	0.71	0.41	1.23
	WaGRS at ONSD	0.21	2.57	0.29	23.20
5	Duration of Disease (≤ 6 mo)	0.59	0.74	0.24	2.29
	Pre-ONSD surgeries	0.13	0.86	0.77	0.96
	Visual Acuity at ONSD	0.01	2.81	0.72	10.93
6	Duration of Disease (≤ 6 mo)	0.28	0.57	0.20	1.63
	Visual Acuity at ONSD	0.01	0.86	0.77	0.96

Table 13. Descriptive analysis of visual field mean deviation for those undergoing optic nerve sheath decompression surgery.

Panel A. Preoperative facros at time of diagnosis.

	Visual Fields				
	Non-Severe		Severe		
	N	%	N	%	P*
Visual Fields	21	60.0	14	40.0	
Etiology					
Idiopathic	20	57.1	12	34.3	
Secondary	1	2.9	2	5.7	0.55
Gender					
Female	14	40.0	14	40.0	
Male	7	20.0	0	0.0	0.03
ONSD type					
Unilateral[reference]	6	17.1	4	11.4	
Bilateral Simultaneous	12	34.3	6	17.1	0.99
Unilateral Sequential	3	8.6	4	11.4	0.64
Diagnosis Year					
Before 2000	14	40.0	8	22.9	
After 2000	7	20.0	6	17.1	0.72
Pre ONSD Surgeries					
No	17	48.6	11	31.4	
Yes	4	11.4	3	8.6	0.99
Post ONSD Surgeries					
No	13	37.1	8	23.0	
Yes	8	22.9	6	17.1	0.78
VF Mean Deviation					
> -20 Db	21	60.0	10	28.6	
≤ - 20 Db	0	0.0	4	11.4	0.02

	N	M	SD	M	SD	P**
LP	16	313.4	68.5	428.0	111.7	0.02
BMI at diagnosis	26	29.9	9.5	28.2	7.7	0.64
Age	35	30.9	13.1	27.1	12.7	0.41
Highest LP	33	362.3	104.8	410.0	112.2	0.23
Visual Acuity	16	0.9	0.8	4.6	8.1	0.37
Mean Deviation	9	-1.8	1.8	-24.8	5.7	0.003
WaGRS	9	0.4	0.5	4.8	0.5	<.0001
Number of Pre ONSD surgeries	35	0.6	1.4	0.3	0.6	0.35
Number of Post ONSD surgeries	35	1.2	2.3	1.1	2.1	0.90
Pap Duration (mo)	27	2.2	3.5	6.4	23.2	0.51

Panel B. Preoperative factors at time of ONSD surgery

	Visual Fields				
	Non-Severe		Severe		
	N	%	N	%	*P**
Papilledema*					
Absent	5	26.3	0	0.0	
Present	8	42.1	6	31.6	0.13
Missing	16				
Disease Duration					
<= 6 mo	13	37.1	6	17.1	
> 6 mo	8	22.9	8	22.9	0.27

	N	M	SD	M	SD	P**
Age	35	33.0	13.1	30.4	10.0	0.55
Visual Acuity	25	2.1	4.2	5.6	7.3	0.14
Visual Acuity Change	16	-1.6	5.1	-2.8	13.6	0.86
Visual Acuity Change Rate	16	-1.6	5.1	-6.3	8.1	0.29
WaGRS	4	2.0	2.0	4.0	0.0	0.48
Mean Deviation	3	-2.9	2.4	-33.5	0.0	0.06
Duration of Disorder (mo)	35	25.4	50.5	39.5	49.8	0.42

Panel C. Post-operative factors at time of last follow-up

	Visual Fields				
	Non-Severe		Severe		
	N	%	N	%	P*
Papilledema					
Absent	14	41.2	8	23.5	
Present	6	17.7	6	17.7	0.44
Visual Fields					
Normal	5	14.3	0	0.0	
Abnormal	16	45.7	14	40.0	0.07
VF Mean Deviation					
Decline	1	2.9	0	0.0	
Stable/Improve	20	47.1	14	40.0	0.99
Time of Follow-up (mo)					
<= 2 yr	9	25.7	8	22.9	
> 2 yr	12	34.3	6	17.1	0.41

	N	M	SD	M	SD	P**
Time of Follow-Up (mo)	35	1.1	1.0	3.9	1.4	<.0001
Visual Acuity	35	0.5	1.0	2.9	4.8	0.08
Change in Visual Acuity	24	1.5	4.7	4.0	7.5	0.33
WaGRS	4	2.0	2.0	4.0	0.0	0.48
Change in WaGRS	4	1.3	2.1	0.0	0.0	0.63

*****Chi-square test; **T-Test

Table 14. Univariate mixed-effects logistic regression analysis of visual field outcome as measured by mean deviation for those undergoing optic nerve sheath decompression surgery for intracranial hypertension

						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Etiology	1.25	1.58	8	0.45	3.48	0.09	134.3
Body Mass Index	0.03	0.07	7	0.67	1.03	0.87	1.22
Age	0.02	0.04	8	0.63	1.02	0.94	1.11
Diagnosis Year	-0.43	0.95	8	0.66	0.65	0.07	5.84
Highest lumbar puncture	-0.01	0.01	8	0.27	0.99	0.98	1.01
Lumbar puncture	-0.02	0.02	5	0.23	0.98	0.94	1.02
ONSDtype *							
Bilateral Simultaneous	0.14	0.98	5	0.89	1.14	0.09	14.28
Bilateral Sequential	-0.67	1.43	3	0.67	0.51	0.01	48.44
Visual Acuity	-0.30	0.43	3	0.54	0.74	0.19	2.94
Panel B. Preoperative factors at the time of ONSD surgery							
Age	0.01	0.04	7	0.74	1.01	0.92	1.11
Duration of Disease (≤ 6 mo)	0.75	0.92	8	0.44	2.12	0.25	17.61
Duration of Disease (mo)	-0.01	0.01	7	0.63	0.99	0.97	1.02
Pre Surgeries	0.07	1.08	7	0.95	1.08	0.08	13.91
Number of pre surgeries	0.33	0.47	7	0.50	1.39	0.46	4.21
Post Surgeries	0.13	0.91	7	0.89	1.14	0.13	9.74
Number of post surgeries	0.07	0.22	7	0.75	1.08	0.64	1.82
Duration of Papilledema	-0.02	0.04	5	0.61	0.98	0.88	1.09
Visual Acuity	-0.12	0.11	6	0.33	0.89	0.68	1.17
Visual Acuity Change	-0.01	0.10	3	0.92	0.99	0.73	1.34
Rate of Visual Acuity Change	0.10	0.12	3	0.48	1.11	0.75	1.64
Panel C. Postoperative factors at the time of last follow-up							
Visual Acuity (Stabilization)	0.63	1.29	6	0.64	1.88	0.08	44.21
Follow-up time (mo)	0.01	0.01	7	0.28	1.01	0.99	1.03
Follow-up time Group (≤ 2 yr)	0.86	0.93	8	0.39	2.36	0.27	20.28

WaGRS = Wall and George rating system

Table 15. Multivariate mixed-effects logistic regression analysis of preoperative factors leading to stabilization/improvement in visual fields as measured by mean deviations scores at the end of the follow-up period.

				CI (95%)	
Model	**Preoperative Factors**	**P**	**OR**	**L**	**U**
1	Duration of Disease (≤ 6 mo)	0.98	0.94	<0.001	>999.9
	Age at ONSD surgery	0.95	0.99	0.71	1.38
	Rate of Visual Acuity change	0.79	1.07	0.42	2.18
	Time of Follow-up (≤ 2 years)	0.63	3.87	<0.01	>999.9
2	Age at ONSD surgery	0.96	1.00	0.77	1.28
	Rate of Visual Acuity change	0.75	1.06	0.61	1.87
	Time of Follow-up (≤ 2 years)	0.61	3.63	<0.01	>999.9
3	Duration of Disease (≤ 6 mo)	0.72	1.06	0.66	1.72
	Time of Follow-up (≤ 2 years)	0.58	3.48	<0.01	>999.9

Table 16. Descriptive analysis of visual field Wall and George rating system for those undergoing optic nerve sheath decompression surgery.

Panel A. Preoperative factors at time of diagnosis.

	Visual Fields				
	Minimal		Marked		
	N	%	N	%	P*
Visual Fields	23	50	23	50	
Etiology					
Idiopathic	19	41.3	21	45.7	
Secondary	4	8.7	2	4.4	0.38
Gender					
Female	16	34.8	23	50.0	
Male	7	15.2	0	0.0	0.01
ONSD type					
Unilateral[reference]	4	8.7	7	15.2	
Bilateral Simultaneous	15	32.6	9	19.6	
Unilateral Sequential	4	8.7	7	15.2	
Diagnosis Year					
Before 2000	1	8.3	0.	0.0	
After 2000	4	33.3	7	58.3	0.42
Pre ONSD Surgeries					
No	18	39.1	18	39.1	
Yes	5	10.9	5	10.9	0.99
Post ONSD Surgeries					
No	17	37.0	11	23.9	
Yes	6	13.0	12	26.1	0.07
VF Mean Deviation					
> -20 Db	23	50.0	18	39.1	
≤ - 20 Db	0	0.0	5	10.9	0.02

	N	M	SD	M	SD	P**
LP	20	320.9	69.3	364.0	104.8	0.29
BMI at diagnosis	33	30.5	10.2	30.5	8.4	0.99
Age	46	30.6	14.6	29.0	11.3	0.68
Highest LP	44	384.1	116.0	388.6	90.9	0.89
Visual Acuity	24	3.1	5.5	6.7	7.6	0.20
Mean Deviation	9	-1.8	1.8	-24.8	5.6	0.003
WaGRS	13	0.4	0.5	4.4	0.8	<.0001
Number of Pre ONSD surgeries	46	0.5	1.2	0.3	0.9	0.58
Number of Post ONSD surgeries	46	0.5	1.2	1.7	2.5	0.05
Pap Duration (mo)	33	8.2	22.9	5.2	16.3	0.66

Panel B. Pre-operative factors at time of ONSD surgery

	Visual Fields				
	Minimal		Marked		
	N	%	N	%	*P**
Papilledema*					
Absent	4	17.4	2	8.7	
Present	9	39.1	8	34.8	0.56
Missing	23				
Disease Duration					
<= 6 mo	12	26.1	11	23.9	
> 6 mo	11	23.9	12	26.1	0.77

	N	M	SD	M	SD	P**
Age	46	32.5	13.6	32.1	11.5	0.92
Visual Acuity	35	1.7	4.1	7.3	6.9	0.006
Visual Acuity Change	24	0	7.5	-2.3	6.5	0.44
Visual Acuity Change Rate	24	-1.4	4.9	-2.3	6.5	0.74
WaGRS	8	1.7	1.5	4.4	0.5	0.009
Mean Deviation	5	-5.9	5.4	-29.4	5.8	0.02
Duration of Disorder (mo)	35	40.8	44.1	24.5	49.7	0.32

Panel C. Post-operative factors at time of last follow-up

	Visual Fields				
	Minimal		Marked		
	N	%	N	%	P*
Papilledema					
Absent	17	38.6	10	22.7	
Present	5	11.4	12	27.3	0.03
Visual Fields					
Normal	1	2.2	2	4.4	
Abnormal	22	47.8	21	45.7	0.55
VF Mean Deviation					
Decline	8	17.4	0	0.0	
Stable/Improve	15	32.6	23	50.0	0.004
Time of Follow-up (mo)					
<= 2 yr	9	19.6	12	26.1	
> 2 yr	14	30.4	11	23.9	0.37

	N	M	SD	M	SD	P**
Time of Follow-Up (mo)	46	72.0	58.6	61.5	68.1	0.58
Visual Acuity	45	0.7	1.0	5.3	6.3	0.003
Change in Visual Acuity	35	1.8	4.5	1.9	6.9	0.92
WaGRS	46	0.7	0.6	4.1	0.9	<.0001
Change in WaGRS	8	1.3	2.1	0.2	1.3	0.37

*****Chi-square test; **T-Test

Table 17. Univariate mixed-effects logistic regression analysis of visual field outcome as measured by Wall and George rating system for those undergoing optic nerve sheath decompression surgery for intracranial hypertension.

Panel A. Preoperative factors at diagnosis.							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Etiology	-0.98	1.09	33	0.49	0.38	0.03	4.99
Body Mass Index	-0.01	0.05	9	0.99	0.99	0.88	1.13
Age	-0.01	0.03	11	0.90	0.99	0.93	1.07
Diagnosis Year	0.06	0.84	11	0.49	1.82	0.28	11.60
Highest Lumbar Puncture	0.01	0.01	11	0.94	1.00	0.99	1.01
Lumbar Puncture	0.01	0.01	6	0.51	1.01	0.98	1.03
ONSDtype *							
Bilateral Simultaneous	1.02	0.93	7	0.31	2.76	0.31	25.0
Bilateral Sequential	0.07	1.17	4	0.95	1.07	0.04	27.38
Visual Acuity	0.08	0.09	5	0.43	1.08	0.86	1.37
Panel B. Preoperative factors at the time of ONSD surgery.							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Age	0.01	0.03	10	0.89	1.01	0.93	1.08
Duration of Disease (≤ 6 mo)	0.06	0.81	11	0.94	1.07	0.18	6.34
Duration of Disease (mo)	0.01	0.01	10	0.45	1.01	0.99	1.03
Pre Surgeries	-0.08	0.92	10	0.93	0.92	0.12	7.10
Number of pre surgeries	-0.17	0.40	10	0.68	0.84	0.35	2.05
Post surgeries	1.25	0.84	10	0.17	3.50	0.54	22.72
Number of post surgeries	0.42	0.27	10	0.16	1.52	0.83	2.80
Duration of Papilledema	-0.01	0.02	7	0.68	0.99	0.94	1.05
Visual Acuity Change	-0.05	0.09	5	0.59	0.95	0.76	1.19
Rate of Visual Acuity Change	-0.02	0.11	5	0.89	0.99	0.75	1.30
WaGRS	1.67	1.55	1	0.48	5.30	<.001	>999
Panel C. Postoperative factors at the time of last follow-up							
						CI (95 %)	
	B	**SE**	**DF**	**P**	**OR**	**L**	**U**
Visual Acuity (Stabilization)	-0.59	0.94	8	0.55	0.56	0.06	4.92
Follow-up time (mo)	-0.01	0.01	10	0.77	1.00	0.98	1.01
Follow-up time Group (≤ 2 yr)	0.96	0.83	11	0.32	2.35	0.38	14.44

WaGRS = Wall and George rating system
*reference group unilateral

Table 18. Multivariate mixed-effects logistic regression analysis of preoperative factors leading to in visual fields as measured by Wall and George rating system at the end of the follow-up period.

				CI (95%)	
Model	**Preoperative Factors**	**P**	**OR**	**L**	**U**
1	Duration of Disease (≤ 6 mo)	0.53	0.29	0.01	41.96
	Age at ONSD surgery	0.95	1.00	0.85	1.18
	Rate of Visual Acuity change	0.79	1.05	0.68	1.60
	Time of Follow-up (≤ 2 years)	0.62	0.38	0.01	55.69
2	Duration of Disease (≤ 6 mo)	0.52	0.32	0.01	22.72
	Rate of Visual Acuity change	0.78	1.04	0.72	1.50
	Time of Follow-up (≤ 2 years)	0.60	0.39	0.01	26.99
3	Duration of Disease (≤ 6 mo)	0.77	1.30	0.19	8.81
	Time of Follow-up (≤ 2 years)	0.31	0.40	0.06	2.69

Table 19. Type of surgical approach and success rates after optic nerve sheath decompression as reported in the intracranial hypertension literature.

		Improvement/ Stabilization		
Authors (Year)	Surgical Incision	Visual Acuity	Visual Fields	Follow-up Period (months)
Banta & Farris (2000)[4]	Windows	94%	88%	20
Goh et al. (1997)[19]	Windows	52%[e]	82%	15.7
Acheson et al. (1994)[1]	Windows	73%	85%	12
Windows Average:	**N = 3**	**73%**	**85%**	**15.9**
Nithyanandam et al. (2008)[27]	Slits	100%	100%	--
Tsai et al. (1995) [43]	Slits	100%	95%	10
Kelman et al. (1992)[22]	Slits	95%[e]	95%[e]	17
Spoor & McHenry (1993)[39]	Slits	68%	--	6-60
Slits Average:	**N = 4**	**91%**	**97%**	**13.5**

Figure 1. Visual acuity across time by surgical intervention.

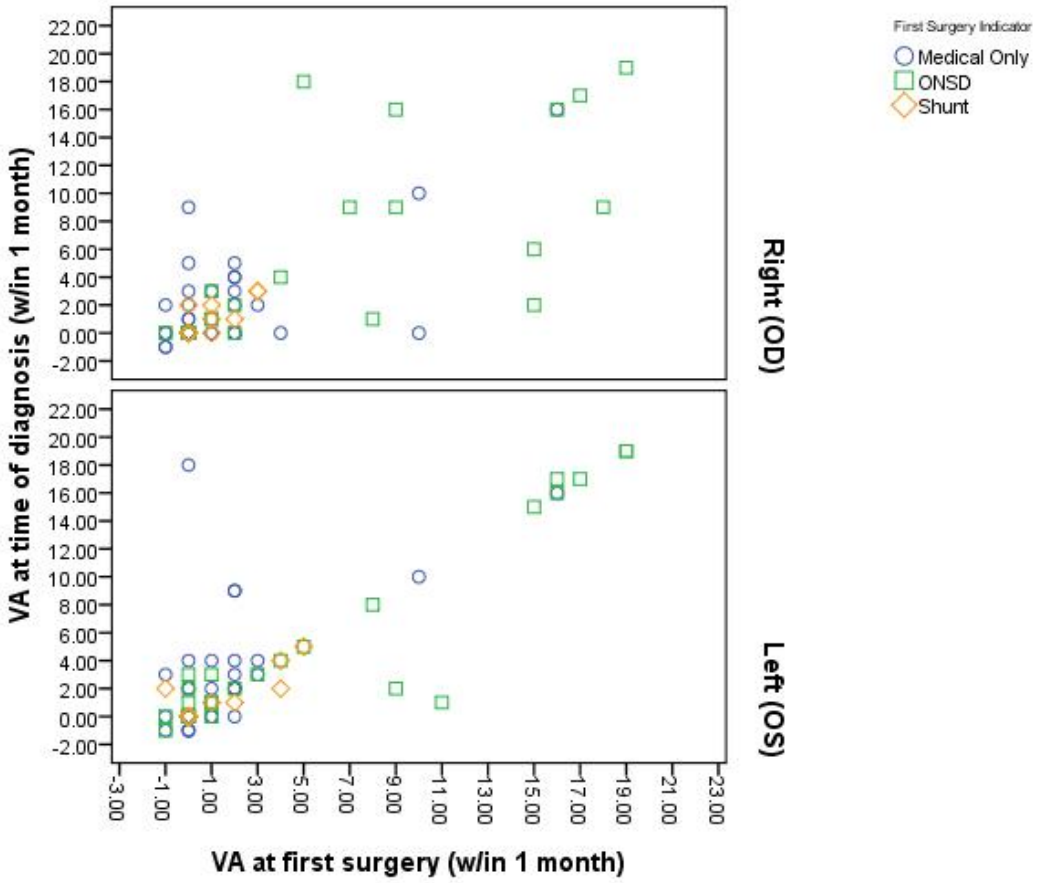

Figure 2. Rate of Visual Acuity decline from diagnosis to pre optic nerve sheath decompression surgery for visual acuity outcome in intracranial hypertension.

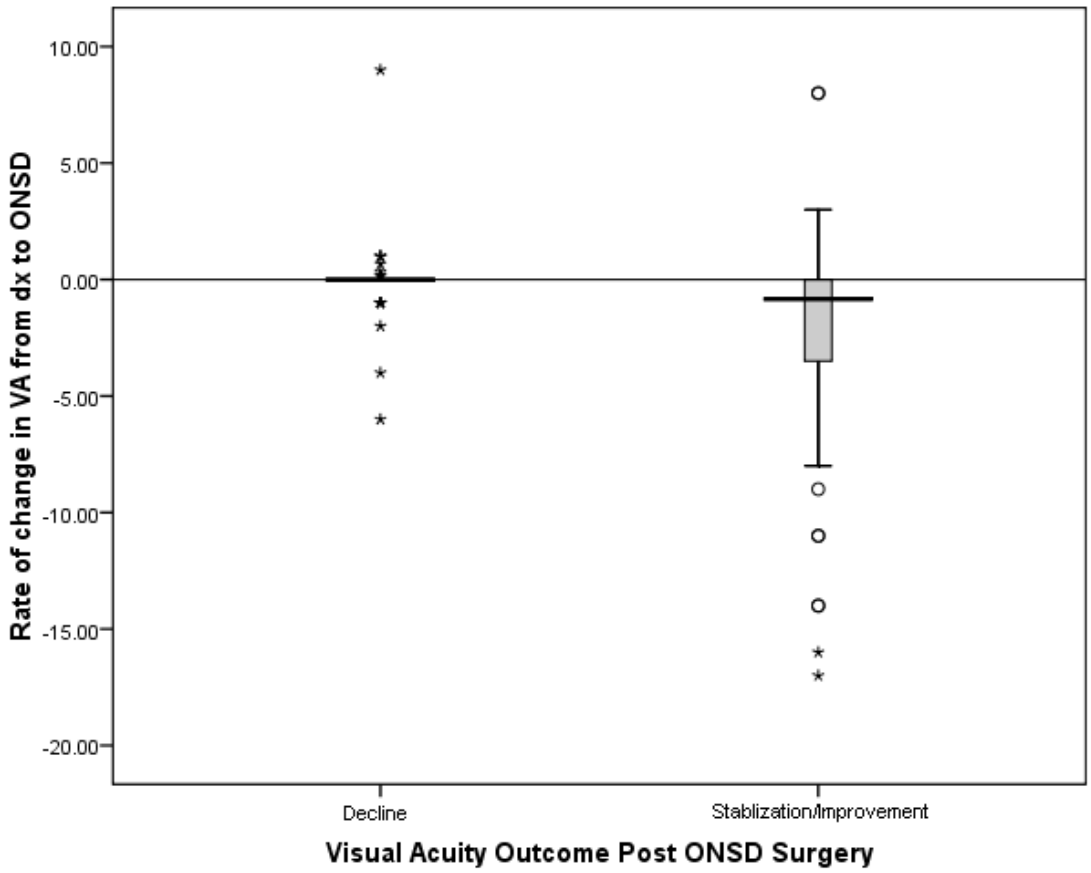

Figure 3.. Excerpt from Friedman's[14] 2006 intracranial hypertension treatment rubric.