Prior Authorization Review Panel  
MCO Policy Submission

A separate copy of this form must accompany each policy submitted for review. Policies submitted without this form will not be considered for review.

<table>
<thead>
<tr>
<th>Plan: Aetna Better Health</th>
<th>Submission Date: 09/04/2018</th>
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<tbody>
<tr>
<td>Policy Number: 0415</td>
<td>Effective Date:</td>
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<tr>
<td></td>
<td>Revision Date: 06/23/2017</td>
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<td>Policy Name: Optic Nerve Decompression Surgery</td>
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Type of Submission – Check all that apply:
- [ ] New Policy
- [x] Revised Policy*
- [ ] Annual Review – No Revisions

*All revisions to the policy must be highlighted using track changes throughout the document. Please provide any clarifying information for the policy below:

Clinical content was last revised on 06/23/2017. Additional non-clinical updates have been made by corporate since that time, as documented below.

08/22/2018 – This CPB was updated with additional background information and references.
04/25/2019 – Tentative next scheduled review date by corporate.

Name of Authorized Individual (Please type or print): Dr. Bernard Lewin, M.D.

Signature of Authorized Individual: 

[Signature]
Optic Nerve Decompression Surgery

Policy

*Please see amendment for Pennsylvania Medicaid at the end of this CPB.*

Aetna considers optic nerve decompression surgery medically necessary for the treatment of the following indications:

- Papilledema accompanying pseudotumor cerebri (idiopathic intracranial hypertension)
- Progressive visual loss associated with craniofacial fibrous dysplasia
- Traumatic optic neuropathy

Aetna considers optic nerve decompression surgery experimental and investigational for the following indications (not an all-inclusive list) because of insufficient evidence in the peer-reviewed literature:

- Intermittent paroxysmal unilateral phosphenes (i.e., light flashes) associated with worsening visual field defects
- Non-arteritic anterior ischemic optic neuropathy (NAION)

Policy History

Last Review 08/22/2018
Effective: 05/04/2000
Next Review: 04/25/2019

Review History

Definitions

Additional Information

Clinical Policy Bulletin
Notes
• Visual loss secondary to optic nerve drusen

Background

Optic nerve decompression surgery (also known as optic nerve sheath decompression surgery) involves cutting slits or a window in the optic nerve sheath to allow cerebrospinal fluid to escape, thereby reducing the pressure around the optic nerve.

Pseudotumor cerebri (also known as idiopathic intracranial hypertension) is a syndrome of increased intracranial pressure without a discernable cause. There is a distinct female preponderance from the teens through the fifth decade. If medical treatment has failed (i.e., Diamox, Lasix, corticosteroids), and disc swelling with visual field loss progresses, direct fenestration of the optic nerve sheaths via medial or lateral orbitotomy has been shown to be an effective and relatively simple procedure for relief of papilledema.

Non-arteritic anterior ischemic optic neuropathy (NAION) is a common cause of sudden loss of vision, especially in the elderly. It is caused by infarction of the short posterior ciliary arteries supplying the anterior optic nerve. There is no direct treatment for NAION, although corticosteroids are sometimes used to reduce optic nerve edema. Treatment goals are aimed at controlling the systemic vascular disease (i.e., hypertension, diabetes, and atherosclerosis) or collagen vascular disease that precipitated NAION in hopes of preventing or delaying bilateral involvement.

Initial results of uncontrolled studies suggested that optic nerve sheath decompression was a promising treatment of progressive visual loss in patients with NAION. Other investigators who evaluated this surgical procedure reported varying degrees of success. To resolve the controversy over
the effectiveness of optic nerve decompression for NAION, the National Eye Institute sponsored the Ischemic Optic Neuropathy Decompression Trial, a multicenter, randomized controlled clinical trial of optic nerve decompression surgery for patients with NAION. The study found no benefit from surgery in NAION patients with progressive visual loss; in fact, significantly more patients in the surgery group had progressive loss of vision than patients who received only careful follow-up. The investigators concluded that optic nerve decompression surgery is not an effective treatment for NAION, and in fact, may increase the risk of progressive visual loss in NAION patients.

A structured evidence review (Dickersin and Manheimer, 2002) concluded that ”[r]esults from the Ischemic Optic Neuropathy Decompression Trial indicate that optic nerve decompression surgery for nonarteritic ischemic optic neuropathy is not effective.”

A Cochrane review (Dickersin et al, 2012) concluded that results from the single trial indicate no evidence of a beneficial effect of optic nerve decompression surgery for NAION. Pletcher and associates (2006) stated that the indications and outcomes for endoscopic decompression of the optic nerve remain controversial.

Atkins et al (2010) stated that NAION is the most common clinical presentation of acute ischemic damage to the optic nerve. Most treatments proposed for NAION are empirical and include a wide range of agents presumed to act on thrombosis, on the blood vessels, or on the disk edema itself. Others are presumed to have a neuroprotective effect. Although there have been multiple therapies attempted, most have not been adequately studied, and animal models of NAION have only recently emerged. The Ischemic Optic Neuropathy Decompression Trial, the only class I large multi-center prospective treatment trial for NAION, found no benefit from surgical intervention. One recent large, non-randomized
controlled study suggested that oral steroids might be helpful for acute NAION. Others recently proposed interventions are intra-vitreal injections of steroids or anti-vascular endothelial growth factor (anti-VEGF) agents. There are no class I studies showing benefit from either medical or surgical treatments. Most of the literature on the treatment of NAION consists of retrospective or prospective case series and anecdotal case reports. Similarly, therapies aimed at secondary prevention of fellow eye involvement in NAION remain of unproven benefit.

Traumatic optic neuropathy (TON) is a complication of head trauma; and there is no uniformly accepted treatment protocol for this condition. Endoscopic, minimally invasive decompression of the optic nerve in its bony canal has been used as an alternative to conservative approach. Sieskiewicz et al (2008) performed endoscopic optic nerve decompression in 6 patients with blindness or severe impairment of vision caused by head trauma. In 5 of them, direct optic nerve injury might have been suspected due to presence of bony fractures in the region of the optic canal and the orbital apex. The time from the trauma to the surgical intervention varied from 8 hours to 30 days. All patients were treated with steroids before the attempted surgery, however the doses and time of this treatment varied significantly. There were no complications of the surgery; patients were mobilized on the day of operation and reported no problems with nasal breathing. Improvement in vision was observed in only 2 of 6 patients (33.3 %).

In a retrospective study, Wang and associates (2008) examined the effectiveness of endoscopic optic nerve decompression in patients with TON. Patients (n = 46) were first treated with methylprednisolone for 6 days. Forty-four patients (46 eyes) who did not improve with methylprednisolone treatment were offered endoscopic optic nerve decompression. In 38 eyes with no light perception vision pre-operatively, 21 eyes (45.6 %) had improvement in visual acuity. These patients had post-operative light
perception in 17 eyes, hand movement in 3 eyes and 60/200 in 1 eye. Four of 5 eyes with light perception pre-operatively had post-operative vision for hand movement in 2 eyes, finger counting in 1 eye and 20/200 in 1 eye. For 3 eyes with pre-operative visual acuity of hand movement, the post-operative visual acuities were 60/200, 60/200 and 120/200. Neither worsening of vision nor major complications was encountered in this series. The authors concluded that endoscopic optic nerve decompression in experienced surgeons’ hands can improve visual acuity in TON with minimal morbidity.

On the other hand, Li et al (2008) reported that there were no difference (statistically) between steroids and steroids plus optic nerve decompression in treating TON. A total of 237 patients were treated with steroids; 176 also consented to endoscopic optic nerve decompression. The total vision improvement rate was 55 % in the 176 patients treated with both steroids and endoscopic optic nerve decompression, compared with 51 % in the 61 patients treated with steroids alone; this difference was not statistically significant (p > 0.05).

Wu and co-workers (2008) stated that serious injury to the optic nerve, including direct and indirect events, induces significant visual loss and even blindness. For the past decade corticosteroids and/or optic canal decompression surgery have been widely embraced as therapeutic paradigms for the treatment of TON. However, the authors noted that there is little clinical evidence to support the effectiveness of these strategies, raising questions about the efficiency of current therapy for improving visual outcomes.

A Cochrane systematic evidence review (Yu Wai Man and Griffith, 2005; Yu Wai Man and Griffith, 2007) found no randomized controlled trials for either the use of corticosteroids or surgical treatment for traumatic optic neuropathy. Citing reports of visual recovery rates of 40 to 60 % with conservative management, the authors concluded that the decision to proceed with surgery or high-dose corticosteroids depends on
the clinical judgment and surgical skills of the surgeon as well as informed consent of the patient to appreciate the benefits and risks of both treatments. More recently, a randomized a placebo-controlled trial of the use of intravenous high-dose corticosteroids versus saline in 31 patients with traumatic optic neuropathy (Entezari et al, 2007) found no statistically significant improvement in visual acuity between the 2 groups.

The International Optic Nerve Trauma Study was organized to compare corticosteroids or surgery and corticosteroids, but after failure to enroll sufficient numbers of patients, the study was transformed into an observational study. Comparing no treatment (observation) versus corticosteroids or surgical decompression, the authors found no difference in the final visual acuity and commented that the decision to treat or not treat should be made on an individual patient basis (Levin et al, 1999).

Some authorities have recommended optic canal decompression if visual acuity does not improve to 20/400 or better despite 24 to 48 hours of steroid therapy or if visual acuity is 20/200 or better but deteriorates during or after completion of steroid therapy (Kountakis et al, 2000).

In a Cochrane review, Dickersin et al (2012) evaluated the safety and effectiveness of surgery compared with other treatment or no treatment in people with NAION. These investigators searched CENTRAL (which contains the Cochrane Eyes and Vision Group Trials Register) (The Cochrane Library 2011, Issue 11), MEDLINE (January 1950 to November 2011), EMBASE (January 1980 to November 2011), the metaRegister of Controlled Trials, ClinicalTrials.gov, and the WHO International Clinical Trials Registry Platform. There were no date or language restrictions in the electronic searches for trials. The electronic databases were last searched on November 19, 2011. All randomized trials of surgical treatment of NAION were eligible for inclusion in this review. These researchers obtained full copies of all

http://aetnet.aetna.com/mpa/cpb/400_499/0415.html 08/30/2018
potentially relevant articles. One author extracted data which was verified by another author. No data synthesis was required. The included trial randomized 258 participants and was stopped early for futility. At the time of the 24-month report the follow-up rate was 95.3% for 6 months and 67.4% for 24 months (174 participants; 89 careful follow-up and 85 surgery). There was no evidence of a benefit of surgery on visual acuity. Measurements of visual acuity and visual fields were performed by a technician masked to the treatment received. At 6 months 32.0% of the surgery group had improved visual acuity by 3 or more lines compared with 42.6% of the careful follow-up group (unadjusted relative risk (RR) 0.75, 95% confidence interval (CI): 0.54 to 1.04). At 24 months 29.4% of the surgery group had improved compared with 31.0% of the careful follow-up group (unadjusted RR 0.95, 95% CI: 0.60 to 1.49). Participants who underwent surgery had a greater risk of losing 3 or more lines of vision, although the increased risk was not statistically significant.

At 6 months 18.9% in the surgery group had worsened compared with 14.8% in the careful follow-up group (RR 1.28; 95% CI: 0.73 to 2.24). At 24 months 20.0% in the surgery group had worsened compared with 21.8% in the careful follow-up group (RR 0.92; 95% CI: 0.51 to 1.64). Participants who received surgery experienced both intra-operative and post-operative adverse events, including central retinal artery occlusion during surgery and light perception vision at 6 months (1 participant); and immediate loss of light perception following surgery and loss of vision that persisted to the 12-month visit (2 participants). In the careful follow-up group, 2 participants had no light perception at the 6-month follow-up visit; 1 of these had improved to light perception at 12 months.

Pain was the most common adverse event in the surgery group (17% in surgery group versus 3% in the careful follow-up group at 1 week). Diplopia (double-vision) was the next most common complication (8% in the surgery group versus 1% in the careful follow-up group at 1 week); at 3 months there was no statistically significant difference in proportion of participants with diplopia between the 2 groups. The authors
concluded that results from the single trial indicate no
evidence of a beneficial effect of optic nerve decompression
surgery for NAION. They stated that future research should
focus on increasing the understanding of the etiology and
prognosis of NAION; new treatment options should be
examined in the context of randomized clinical trials.

Moreau et al (2014) examined the safety and effectiveness of
optic nerve sheath decompression (ONSD) with a medial
trans-conjunctival approach for a variety of indications in a
larger population of patients than has previously been
reported. A retrospective chart review was performed on
consecutive patients who underwent ONSD between January
1992 and December 2010. Before ONSD, all patients had
documented evidence of progressive loss of visual acuity or
visual field, or both. Post-operative follow-up visits were
scheduled at 1 week, 1 month, and then every 3 to 6 months.
Main outcome measures were visual acuity, visual fields, and
surgical complications. A total of 578 eyes of 331 patients
underwent ONSD for progressive vision loss due to various
indications, which included but were not limited to idiopathic
intracranial hypertension (IIH), progressive NAION, and optic
nerve drusen (OND). During a mean follow-up of 18.7 months
(range of 1 week to 10 years), post-operative visual acuity
remained stable or improved in 536 of 568 eyes (94.4 %) and
progressively worsened in 32 of 568 eyes (5.6 %). Visual
fields remained stable or improved in 257 of 268 eyes (95.9 %)
and progressive visual field loss occurred in 11 of 268 eyes
(4.1 %). There were no reported intra-operative complications.
The most common post-operative complication was diplopia
(6.0 %). The authors concluded that this review represented
the largest series of patients who have undergone ONSD for
any indication. These data were consistent with current
literature supporting ONSD as a safe and effective procedure
for IIH. Other indications for ONSD, such as progressive
visual field loss associated with OND, warrant further study.
Regardless of the indication, complications following ONSD
with the technique described in this report were infrequent.
Intermittent Paroxysmal Unilateral Phosphenes

De Ridder et al (2016) stated that microvascular decompression surgery is standard neurosurgical practice for treating trigeminal neuralgia and hemi-facial spasm. Most other cranial nerves have been decompressed for paroxysmal intermittent hyperactivity of the affected cranial nerve or in very long-standing compressions to treat cranial nerve hypofunctioning. These investigators described a case of intermittent paroxysmal unilateral phosphenes (i.e., light flashes) associated with worsening visual field defects. Magnetic resonance imaging showed a sandwiched optic nerve/chiasm between an inferior compression of the internal carotid artery and a superior compression of the anterior communicating artery. The patient was successfully treated by microvascular decompression and anterior clinoidectomy plus optic canal un-roofing. The authors concluded that this case report added to the few previous case reports combining 2 previously described techniques (i.e., microvascular decompression and anterior clinoidectomy plus optic canal un-roofing). The major drawbacks of this study were that it was a single-case study and its findings were confounded by the combinational use of microvascular decompression and anterior clinoidectomy plus optic canal un-roofing.

Progressive Visual Loss Associated with Craniofacial Fibrous Dysplasia

Bhattacharya and Mishra (2015) stated that fibrous dysplasia (FD) is a non-malignant fibro-osseous bony lesion in which the involved bone/bones gradually get converted into expanding cystic and fibrous tissue. The underlying defect in FD is post-natal mutation of GNAS1 gene, which leads to the proliferation and activation of undifferentiated mesenchymal cells arresting the bone development in woven phase and ultimately converting them into fibro-osseous cystic tissue. Cherubism is a hereditary form of fibrous dysplasia in which the causative factor is transmission of autosomal dominant SH3BP2 gene
mutation. The disease may present in 2 distinct forms: (i) a less severe and limited monostotic form, and (ii) a more aggressive and more widespread polyostotic form. Polyostotic form may be associated with various endocrine abnormalities, which require active management apart from the management of FD. Management of FD is not free from controversies. While total surgical excision of the involved area and reconstruction using newer micro-vascular technique is the only definitive treatment available from the curative point of view, but this can be only offered to monostotic and very few polyostotic lesions. In polyostotic varieties on many occasions these radical surgeries are very deforming in these slow growing lesions and so their indication is highly debated. The treatment of craniofacial FD should be highly individualized, depending on the fact that the clinical behavior of lesion is variable at various ages and in individual patients. A more conservative approach in the form of aesthetic re-contouring of deformed bone, orthodontic occlusal correction, and watchful expectancy may be the more accepted form of treatment in young patients. Newer generation real-time imaging guidance during re-contouring surgery adds to accuracy and safety of these procedures. Regular clinical and radiological follow-up is needed to watch for quiescence, regression or reactivation of the disease process. Patients must be warned and watched for any sign of nerve compression, especially visual impairment due to optic nerve compression. Rather than going for prophylactic optic canal decompression (which does more harm than good), optic nerve decompression should be done in symptomatic patients only, and preferably be done via minimal invasive endoscopic neurosurgical approach than the conventional more morbid open craniotomy approach. The authors noted that there is growing research and possibilities that newer generation bisphosphonate medication may change the management scenario, as these medications show encouraging response in not only reducing the osteoclastic activity, but simultaneously also stimulating the osteoblastic and osteocytic activities. Belsuzarri and associates (2016)
noted that FD is a benign fibro-osseous lesion related to an abnormal bone development and replacement by fibrous tissue. Fibrous dysplasia has 3 clinical patterns: (i) monostotic, (ii) polyostotic, and (iii) the McCune-Albright syndrome (MAS). McCune-Albright syndrome is a rare genetic disorder (about 3% of all FDs) that comprises a triad of polyostotic FD, cafe-au-lait skin macules, and precocious puberty; MAS can involve the orbit region and cause stenosis in the optic canal, leading the patient to a progressive visual loss. These investigators reported a case of craniofacial FD in MAS in a 9-year old male with progressive visual loss, submitted to optic nerve decompression by fronto-orbitozygomatic approach, with total recovery. A research was made at Bireme, PubMed, Cochrane, LILACS, and Medline with the keywords: FD/craniofacial/McCune-Albright/Optic compression for the clinical review. A clinical review of the disease was made, the multiple, clinical, and surgical management options were presented, and the case report was reported. The authors concluded that MAS is a rare subtype of FD with endocrinopathy. The FD of the orbital region can lead to optic nerve compression and possibly to visual loss. They stated that there is no evidence to support the benefits of prophylactic surgery in children with normal visual fields and optic canal narrowing, shown by the CT or MRI, and there is no method to predict which child will stabilize or deteriorate the visual loss. The cystic degenerations can lead to sudden visual loss and is the only possible indication for prophylactic surgery, but the risk of nerve damage should be considered and well-explained. In all other cases of normal visual fields and CT/MRI optic canal narrowing, prophylactic surgery is not indicated, and follow-up should be done. On the other hand, early decompression of symptomatic children is a great standard for a better chance of visual loss reverse.

In a retrospective, chart-review study, DeKlotz and colleagues (2017) evaluated visual outcomes and potential complications for optic nerve decompression using an endoscopic endonasal...
approach (EEA) for FD. Patients with FD causing extrinsic compression of the canalicular segment of the optic nerve that underwent an endoscopic endonasal optic nerve decompression at the University of Pittsburgh Medical Center from 2010 to 2013 were included in this trial. The primary outcome measure assessed was best-corrected visual acuity (BCVA) with secondary outcomes, including visual field testing, color vision, and complications associated with the intervention. A total of 4 patients and 5 optic nerves were decompressed via an EEA. All patients were symptomatic pre-operatively and had objective findings compatible with compressive optic neuropathy: decreased VA was noted pre-operatively in 3 patients while the remaining patient demonstrated an afferent pupillary defect; BCVA improved in all patients post-operatively. No major complications were identified. The authors concluded that EEA for optic nerve decompression appeared to be a safe and effective treatment for patients with compressive optic neuropathy secondary to FD. Moreover, they stated that further studies are needed to identify selection criteria for an open versus an endoscopic approach.

Endoscopic Optic Nerve Decompression for the Treatment of Idiopathic Intracranial Hypertension

Tarrats and colleagues (2017) stated that the conventional treatment for IIH involves weight loss, steroids, diuretics, and/or serial lumbar punctures; however, if the symptoms persist or worsen, surgical intervention is recommended. Surgical options include cerebro-spinal fluid (CSF) diversion procedures, such as ventriculo-peritoneal and lumbo-peritoneal shunts, and optic nerve decompression with nerve sheath fenestration. The latter can be performed using an endoscopic approach, but the outcomes of this technique have not been firmly established. This systematic review examined the outcomes of performing endoscopic optic nerve decompression (EOND) in patients with IIH; 6 studies were included for a total of 34 patients. The patients presented with
visual field disturbances (32 of 32 [100 %]), VA disruptions (33 of 34 [97.1 %]), papilledema (26 of 34 [76.5 %]), and persistent headache (30 of 33 [90.1 %]). The mean duration of symptoms ranged from 7 to 32 months. Overall, the patients showed post-EOND improvement in signs and symptoms associated with IIH, specifically visual field deficits (93.8 %), VA (85.3 %), papilledema (81.4 %), and headaches (81.8 %). Interestingly, 11 cases showed post-operative improvement in their symptoms with bony decompression of the optic canal alone, without nerve sheath fenestration. There were no major AEs or complications reported with this approach. The authors concluded that EOND appeared to be a promising and safe surgical alternative for patients with IIH who failed to respond to medical treatment. Moreover, they stated that further studies are needed to ascertain the clinical validity of this procedure.

Endoscopic Endonasal Optic Nerve Decompression for the Treatment of Optic Neuropathy Secondary to Fibrous Dysplasia

DeKlotz and colleagues (2017) evaluated visual outcomes and potential complications for optic nerve decompression using an endoscopic endonasal approach (EEA) for fibrous dysplasia. These researchers carried out a retrospective chart review of patients with fibrous dysplasia causing extrinsic compression of the canalicular segment of the optic nerve that underwent an endoscopic endonasal optic nerve decompression from 2010 to 2013. The primary outcome measure assessed was BCVA with secondary outcomes, including visual field testing, color vision, and complications associated with the intervention. A total of 4 patients and 5 optic nerves were decompressed via an EEA. All patients were symptomatic pre-operatively and had objective findings compatible with compressive optic neuropathy: decreased VA was noted pre-operatively in 3 patients while the remaining patient demonstrated an afferent pupillary defect; BCVA improved in all patients post-operatively. No major
complications were identified. The authors concluded that fibrous dysplasia is a rare disease that can lead to compressive optic neuropathy and subsequent visual impairment. Only patients with signs and/or symptoms of compressive optic neuropathy are candidates for surgery. The EEA for optic nerve decompression appeared to be a safe and effective means of treating these patients. It is unknown if endoscopic decompression can be successfully applied to most or all patients with this disease component or rather if only a select subgroup is likely to benefit and requires further study with longer follow-up. They stated that while transcranial approaches are expected to continue to have a role in the management of these patients, the EEA shows promise and may become a preferred option for therapeutic intervention.

The authors stated that this study had several drawbacks. First, it was a small study population (n = 4). Benign pathology of the canalicular segment of the optic nerve that is amenable to surgical decompression is rare. The limited number of patients and interventions prevented making definitive statements about the overall efficacy and safety. The current study only evaluated patients affected by fibrous dysplasia and whether this can be extrapolated to those with other fibro-osseous lesions is unknown. The follow-up in the current study was limited (the mean follow-up was 14.8 months). As demonstrated by 1 patient, there was the possibility for recurrent compressive optic neuropathy following endoscopic endonasal decompression. Although there was no disease progression on imaging, his VA and color perception worsened on ophthalmologic testing and prompted a lateral decompression via craniotomy. The prevalence of the need for additional interventions as well as timing following an endoscopic approach is unknown. Additional work is needed in the future to fully define the role of endoscopic optic nerve decompression for fibrous dysplasia. More concrete indications need to be determined to enable more widespread acceptance. While randomized controlled trials (RCTs) are unrealistic given the rarity of these disorders, additional
reporting of outcomes is needed to critically analyze and
determine those likely to benefit from intervention. While
endoscopic optic nerve decompression appeared to have an
emerging role in the management of these lesions, this did not
mean that open cranial approach is now obsolete. The
potential benefits of improved visualization, preserved
olfaction, more rapid recovery, lack of scarring, decreased
operative stress, and lack of cerebral retraction would favor an
endoscopic approach in some settings, though it is not
universal. Location of the pathology (medial versus lateral) is
perhaps the most critical determinant of approach used as well
as surgeon experience and comfort with a particular technique.
While the use of endoscopic endonasal
decompression is expected to grow with further clarification of
its indications, it is not expected to completely supplant
traditional transcranial approaches.

CPT Codes / HCPCS Codes / ICD-10 Codes

Information in the [brackets] below has been added for
clarification purposes. Codes requiring a 7th character are
represented by "+":

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<td>Papilledema associated with increased intracranial pressure</td>
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<td>Injury of optic nerve</td>
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<td>H53.8</td>
<td>Other visual disturbances [Intermittent paroxysmal unilateral phosphenes (i.e., light flashes)]</td>
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The above policy is based on the following references:


8. The Ischemic Optic Neuropathy Decompression Trial Research Group. Optic nerve decompression surgery for nonarteritic anterior ischemic optic neuropathy (NAION) is not effective and may be harmful. JAMA. 1995;273(8):625-632.


AETNA BETTER HEALTH® OF PENNSYLVANIA

Amendment to
Aetna Clinical Policy Bulletin Number: 0415 Optic Nerve
Decompression Surgery

There are no amendments for Medicaid.

www.aetnabetterhealth.com/pennsylvania updated 08/22/2018