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Surgery for traumatic optic neuropathy (Review)

Yu-Wai-Man P, Griffiths PG

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[Intervention Review]

Surgery for traumatic optic neuropathy

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ABSTRACT

Background

Traumatic optic neuropathy (TON) is an important cause of severe visual loss following blunt or penetrating head trauma. Following the initial insult optic nerve swelling within the optic nerve canal or compression by bone fragments are thought to result in secondary retinal ganglion cell loss. Optic nerve decompression with steroids or surgical interventions or both have therefore been advocated to improve visual prognosis in TON.

Objectives

To examine the effects and safety of surgical interventions in the management of TON.

Search methods

We searched CENTRAL (which contains the Cochrane Eyes and Vision Group Trials Register) (*The Cochrane Library* 2013, Issue 4), Ovid MEDLINE, Ovid MEDLINE In-Process and Other Non-Indexed Citations, Ovid MEDLINE Daily, Ovid OLDMEDLINE, (January 1950 to May 2013), EMBASE (January 1980 to May 2013), Latin American and Caribbean Literature on Health Sciences (LILACS) (January 1982 to May 2013), the *meta*Register of Controlled Trials (*m*RCT) (www.controlled-trials.com), ClinicalTrials.gov (http://clinicaltrials.gov) and the WHO International Clinical Trials Registry Platform (ICTRP) (www.who.int/ictrp/search/en). We did not use any date or language restrictions in the electronic searches for trials. We last searched the electronic databases on 28 May 2013. We also searched the reference lists of other reviews and book chapters on TON. We also contacted researchers in the field.

Selection criteria

We planned to include only randomised controlled trials (RCTs) of TON in which any form of surgical intervention either on its own or in combination with steroids was compared to steroids alone or no treatment.

Data collection and analysis

Two authors independently assessed the titles and abstracts identified from the search strategy. No studies were found that met our inclusion criteria and therefore none were included for analysis.

Main results

No studies were found that met our inclusion criteria.

Authors' conclusions

The current body of evidence consists mostly of small, retrospective case series. Given the wide range of surgical interventions used in TON it is very difficult to compare these studies, even qualitatively. However, there is a relatively high rate of spontaneous visual recovery and

no evidence that surgical decompression of the optic nerve provides any additional benefit. On the other hand, surgery carries a definite risk of complications such as postoperative cerebrospinal fluid leak and meningitis. The decision to proceed with surgery in TON therefore remains controversial and each case needs to be assessed on its own merits. Although there is an urgent need for an adequately powered, RCT of surgical intervention in TON, this will prove a difficult endeavour.

PLAIN LANGUAGE SUMMARY

Surgery for the treatment of traumatic optic neuropathy

The optic nerve transmits visual information from the eye to the brain and traumatic optic neuropathy (TON) refers to any injury to the optic nerve secondary to trauma. After the optic nerve has been injured, it becomes more swollen and this can lead to further damage. Traumatic optic neuropathy often results in severe visual loss and the vast majority of affected patients are young males in their thirties. Surgery has been used in TON to try and reduce this abnormal swelling or remove bone fragments. There are currently no good quality studies that show greater visual improvement following surgery compared to no treatment. Surgery carries a definite risk of complications which must be considered.

BACKGROUND

Introduction

Traumatic optic neuropathy (TON) refers to any insult to the optic nerve secondary to trauma. It can be classified depending on the site of injury (optic nerve head, intraorbital, intracanalicular, intracranial) or according to the mode of injury (direct or indirect).

Direct TON results from anatomical disruption of the optic nerve, for example a projectile penetrating the orbit and impinging on the optic nerve. Indirect TON is caused by the transmission of forces to the optic nerve from a distant site without disruption of normal tissue structures. The deformative stress transmitted to the skull from blunt trauma is concentrated in the region of the optic canal. Since the optic nerve's dural sheath is tightly adherent to the periosteum the intracanalicular segment is particularly susceptible to injury (Anderson 1982; Gross 1981). The intracranial portion of the optic nerve in close proximity to the falciform dural fold is the next most common site of injury (Crompton 1970). In one report, using computed tomography (CT) scans, about half of all TON cases were found to have an associated sphenoidal bone fracture, an indirect measure of the significant compressive forces involved (Seiff 1990). However, both direct and indirect mechanisms can contribute to optic nerve damage and a clear distinction is not always possible.

Pathophysiology

The pathophysiology of indirect TON is likely to be multifactorial and the concept of primary and secondary injury has been proposed (Steinsapir 1994). Retinal ganglion cells (RGC) are specialised cells within the optic nerve and form part of an intricate chain responsible for transmitting information from the eye to the vision centres within the brain. Following trauma there is an immediate mechanical shearing of a proportion of RGC axons, an irreversible process with subsequent RGC degeneration. There is then postulated optic nerve swelling within the limited confines of the optic canal secondary to direct mechanical trauma or vascular ischaemia or both. This further impairs the already compromised blood supply to surviving RGCs, setting up a downward spiral towards apoptotic cell death. It is therefore plausible that visual prognosis could be improved by limiting these secondary mechanisms and preserving RGCs that survived the initial insult. This model forms the current rationale for optic nerve decompression in TON whether by medical or surgical means.

Epidemiology

Traumatic optic neuropathy is an uncommon cause of visual loss following blunt or penetrating head trauma with a reported incidence of 0.7% to 2.5% in published case series (al-Qurainy 1991; Edmund 1963; Nau 1987). A recent national epidemiological survey of TON in the UK found a minimum prevalence in the general population of 1 in 1,000,000 (Lee 2010). The vast majority of affected patients are young males (79% to 85%) in their early thirties males; one study quoting 85% as being male with a mean age of 34 years (Lee 2010; Levin 1999). The most common causes of TON in adults are motor vehicle and bicycle accidents (49%), falls (27%) and assaults (13%) (Steinsapir 1998). In a paediatric case series, TON was the result of a fall in 50% and a road traffic accident in 40% of cases (Mahapatra 1993).

Clinical features

Traumatic optic neuropathy is a clinical diagnosis supported by a history of direct or indirect trauma to the head or face. The injury can sometimes be trivial and a careful history must be elicited from the patient. Although usually straightforward the assessment of TON can sometimes prove difficult in the setting of severe trauma when the patient's level of consciousness is impaired. It is also important to exclude possible reversible causes of visual loss that require immediate attention, for example a retrobulbar haemorrhage.

The features of TON are:
1. unilateral or bilateral ocular involvement;
2. relative afferent papillary defect except in cases of symmetrical, bilateral TON;
3. variable loss of visual acuity ranging from normal to no light perception. At first presentation, 40% to 60% of patients have been reported as having severe visual loss of light perception or worse (Lee 2010; Lessell 1989; Mauriello 1992; Spoor 1990). Direct TON causes severe and immediate loss of vision with little prospect of recovery. The prognosis is better for indirect TON but it can be associated with delayed visual loss secondary to the development of an optic nerve sheath haematoma;
4. impairment of colour vision;
5. variable visual field defects;
6. optic disc appearance will depend on the anatomical site of injury. With injuries to the optic nerve anterior to the entry point of the central retinal vessels there is optic disc swelling with associated retinal haemorrhages. With more posterior injuries, which are more common, the fundus looks normal;
7. development of optic atrophy which usually becomes evident about six weeks following the injury.

Computed tomography (CT) is the best imaging modality for delineating optic canal fractures. However, there is wide variation in practice worldwide regarding the use of neuroimaging in TON. Some clinicians request CT or magnetic resonance imaging (MRI) or both for all cases, whereas others limit them to those patients with progressive visual deterioration or when therapeutic interventions are being considered (Raji 1982; Seiff 1984; Takehara 1994).

Treatment options

The main treatment options in current use for TON are:
1. systemic steroids in various doses, duration and modes of administration;
2. surgical decompression of the optic canal;
3. a combination of steroids and surgery (Steinsapir 1994).

Rationale for a systematic review

There is persisting controversy among clinicians regarding the indications for surgery in TON. Given the wide variety of surgical approaches advocated and the potential risks that surgery entails for the patient, there is a need for a systematic review of the literature to make recommendations for best clinical practice. The role of steroids in TON is the subject of another Cochrane review written by the same authors (Yu-Wai-Man 2013).

OBJECTIVES

The aim of this review was to examine the effects and safety of surgical interventions in the management of TON.

METHODS

Criteria for considering studies for this review

Types of studies

This review included only randomised controlled trials (RCTs).

Types of participants

We included trials in which participants were people diagnosed clinically as having either direct or indirect TON. Bilateral cases were excluded.

Types of interventions

We included studies in which any form of surgical treatment either on its own or in combination with steroids was compared to steroids alone or no treatment.

Types of outcome measures

Primary outcomes

The primary outcome measure was the number of Snellen lines of visual acuity gained or lost at three and six months follow up.

Secondary outcomes

Secondary outcomes that were considered included:
1. any other validated measures of visual function, for example contrast sensitivity and visual fields;
2. any adverse outcome reported in the trials;
3. any validated quality of life scale assessing participants' views of their treatment and visual disability resulting from TON.

Follow up

We included trials in which participants were followed for at least one month.

Search methods for identification of studies

Electronic searches

We searched the Cochrane Central Register of Controlled Trials (CENTRAL) 2013, Issue 4, part of *The Cochrane Library*. www.thecochranelibrary.com (accessed 28 May 2013), Ovid MEDLINE, Ovid MEDLINE In-Process and Other Non-Indexed Citations, Ovid MEDLINE Daily, Ovid OLDMEDLINE, (January 1950 to May 2013), EMBASE (January 1980 to May 2013), Latin American and Caribbean Literature on Health Sciences (LILACS) (January 1982 to May 2013), the *meta*Register of Controlled Trials (*m*RCT) (www.controlled-trials.com), ClinicalTrials.gov (http://clinicaltrials.gov) and the WHO International Clinical Trials Registry Platform (ICTRP) (www.who.int/ictrp/search/en). We did not use any date or language restrictions in the electronic searches for trials. We last searched the electronic databases on 28 May 2013.

See: Appendices for details of search strategies for CENTRAL (Appendix 1), MEDLINE (Appendix 2), EMBASE (Appendix 3), LILACS (Appendix 4), *m*RCT (Appendix 5), ClinicalTrials.gov (Appendix 6) and the ICTRP (Appendix 7).

Searching other resources

We searched the reference lists of other reviews and book chapters on TON to find references to additional trials. We did not manually search any journals or conference proceedings. We contacted trial investigators and experts in the field to identify additional published and unpublished studies.

Data collection and analysis

No studies were identified that met our inclusion criteria.

Methods to be used in updates to the review

If trials become available in the future they will be included in this review using the following methods.

Selection of studies

Two authors will independently assess the titles and abstracts of newly identified reports. We will obtain full copies of studies that appear to meet our inclusion criteria. Both authors will then assess the report to ensure that they meet the inclusion criteria detailed above. Any disagreement will be discussed and a consensus opinion reached. Should the need arise we will contact the main study authors to clarify any data necessary to make a comprehensive assessment of the relevance of the study. Reports that do not completely fulfil our inclusion criteria will be excluded.

Data extraction and management

This will be done independently by two review authors and a proforma sheet will be developed to record the data extracted from the studies. This will include the following.

- study design: method of randomisation, exclusion after randomisation, masking (blinding) of outcome measures, loss to follow up;
- participants: setting; numbers enrolled, numbers randomised, demographics, inclusion and exclusion criteria;
- interventions: timing; duration; details of surgical interventions; information on operating surgeons if available, for example level of expertise.
- outcomes: primary and secondary outcomes as detailed above;
- other: additional details thought relevant, for example funding sources.

One review author (PYWM) will enter the data into RevMan 5 and a second author (PGG) will check the data once it has been entered for errors and inconsistencies.

Assessment of risk of bias in included studies

Two authors independently will appraise studies that meet the inclusion criteria for methodological quality. We will not be masked to publication details or trial results during this process. The following parameters will be used as detailed further in Chapter 8 of the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011):
1. method used for generating the randomisation sequence;
2. allocation concealment;
3. masking (blinding) of clinicians assessing outcome measures;
4. extent of follow up in the various intervention groups.

Given that the comparisons will be surgery with or without steroids versus steroids versus no treatment, masking of participants and clinicians to assigned intervention is difficult. This criterion will therefore not be considered in assessing methodological quality for the review.

Each parameter will be assessed and subsequently graded as: Yes (low risk of bias); No (high risk of bias) or Unclear. If any parameter is graded as Unclear we will attempt to contact the study authors for further details before reclassifying to either Yes or No. If clarification is not available, or if any disagreement arises between the two review authors, a consensus decision will be reached.

Trials with inadequate methods for generating the randomisation sequence and allocation concealment will be excluded. Trials assessed as No for the other two parameters will be included and subjected to sensitivity analyses (see below).

Measures of treatment effect

We will analyse our dichotomised visual outcome data using odds ratios.

Assessment of reporting biases

We will investigate the potential influence of publication bias by examining funnel plots.

Data synthesis

For the primary outcome measures, we will collect the data as LogMAR visual acuity gained or lost at three and six months follow-up. If BCVA was measured using the Snellen chart, for the purpose of statistical analysis, Snellen ratios will be converted to LogMAR decimal values. A LogMAR value of 0 is equivalent to 6/6 Snellen vision and a value of 1.0 is equivalent to 6/60 Snellen vision, the largest optotype on standard Snellen charts. Patients with visual acuities reduced to counting fingers (CF) will be assigned a LogMAR value of 2.0, and those with only hand movement (HM) perception will be given a LogMAR value of 2.3 (Lange 2009; Schulze-Bonsel 2006).

Our goal will be to extract similar outcome data from each study in our review to achieve consistency of results. If data are missing or difficult to interpret from a paper we will contact the authors for more information.

Before carrying out a meta-analysis we will assess heterogeneity by examining the characteristics of the study, the forest plot of results in the studies, the chi squared statistic and I^2 value for statistical heterogeneity. If heterogeneity is not detected a random-effects model will be used unless there are fewer than three trials in which case we may use a fixed-effect model.

Sensitivity analysis

We will perform sensitivity analyses to assess how robust the results are to changes in the methods for the review such as:
1. different inclusion criteria, for example timing and type of surgical interventions;
2. excluding studies of lower methodological quality, that is those graded as No (high risk of bias) on any parameter of quality;
3. excluding unpublished studies.

RESULTS

Description of studies

Results of the search

The initial electronic searches identified 516 reports of studies. It was clear from the abstracts that there were no RCTs of surgical interventions in TON. The following experts were contacted and no relevant trials were identified: Professor Stuart R Seiff; Dr Kenneth D Steinsapir; Professor Roy W Beck; Dr Leonard A Levin; Professor Alfredo A Sadun; and Professor Andrew G Lee.

Updated searches

An updated search was done in June 2007 which yielded a further 362 reports of studies. The Trials Search Co-ordinator (TSC) scanned the search results and removed any references which were not relevant to the scope of the review. Two reports were identified for potential inclusion in the review. One report was excluded by looking at the abstract but the full copy of Gupta 2007 was obtained as it met the inclusion criteria. On inspecting the full copy, it was excluded as it was an observational study.

A further update search was done in December 2010. After deduplication the search identified a total of 718 references. The TSC scanned the search results and removed any references which were not relevant to the scope of the review. No new studies were identified.

The update search in May 2013 identified a further 643 references. The TSC removed 74 duplicates, scanned 569 references and removed 562 records which were not relevant to the scope of the review. We screened the remaining seven references but did not find any trials that met the inclusion criteria for the review.

Risk of bias in included studies

Since no studies were found that met our inclusion criteria no studies were assessed for quality.

Effects of interventions

No RCTs were identified and therefore no data were collected for analysis.

DISCUSSION

We did not find any RCTs examining the role of surgery for traumatic optic neuropathy (TON). There are several published case series in the literature looking at surgical interventions in TON (*see* references in Chou 1996; Cook 1996; Joseph 1990; Levin 1999; Li 2008; Steinsapir 1998) and these are discussed below.

Methodological flaws

These mostly small, retrospective studies all suffer from several methodological flaws that render interpretation difficult. Due to the lack of adequate randomisation there was an inevitable trend towards offering surgery to patients with the worse baseline visual acuities or those who failed to improve with steroids. The evidence in the literature strongly suggests that the final visual outcome in TON is largely dictated by the visual acuity at first presentation (Levin 1999; Mine 1999; Wang 2001; Yang 2004). Recruitment bias towards a subgroup with worse baseline visual acuities could, therefore, have resulted in either a failure to detect any potential benefit or an underestimation. We do not know whether patients who fail to respond to steroids initially represent a group less likely to benefit from subsequent surgical optic canal decompression. This is another potential confounding factor.

The actual assessment and definition of visual improvement is also open to criticism. In the acute setting the baseline visual status was often assessed at the bedside by basic methods but the final

visual outcome was always based upon subsequent clinic reviews. Presumably, most of these patients will then have had a formal refraction to determine best corrected visual acuity. The time interval between injury and recruitment is also not detailed in most published case series. This information is relevant as vision can improve spontaneously in TON (*see* below). If there is earlier active recruitment of TON cases to the surgical arm as opposed to the control arm of the study the former is more likely to demonstrate a better visual outcome, skewing the results spuriously in favour of intervention. These inherent biases in patient selection and methods of outcome assessment could therefore account for the improved visual benefit reported by studies supporting surgical decompression over observation. There is also no evidence of any additional functional visual benefit for patients following surgery.

Timing of surgery

There is conflicting evidence relating to whether the length of time between the initial insult and surgical intervention impacts on visual recovery in TON. This is surprising given that intuitively one would expect that the longer the delay, the less likely would optic canal decompression salvage compromised retinal ganglion cells from irreversible cell death. This is an important point, especially in the context of trauma where other life-threatening injuries often lead to unavoidable delay in ophthalmological assessment.

Surgical intervention

It is very difficult to compare studies, even qualitatively, because of the wide range of intra and extracranial surgical techniques used in TON. The favoured intervention was largely influenced by the expertise available locally and surgeon's preference. However, the current trend is towards an extracranial surgical approach, for example transethmoidal, endonasal and sublabial.

Spontaneous visual recovery

A visual recovery rate of 40% to 60% has been reported for indirect TON cases managed conservatively (see references in Chou 1996; Cook 1996; Levin 1999; Seiff 1990; Steinsapir 2005). Most of these studies also consistently showed a significant correlation between initial and final visual acuities, patients with no light perception at presentation invariably having a poor prognosis. Direct TON is a distinct category that results in severe, irreversible visual loss, and surgical intervention is of no proven benefit.

International Optic Nerve Trauma Study (IONTS)

The International Optic Nerve Trauma Study (IONTS) is the largest, prospective, multi-centre study of TON published to date (Levin 1999). It was intended to be an RCT but it had to be converted to an observational study after two years due to recruitment failure. The study analysed a total of 133 people with indirect TON treated within seven days of injury and categorised into three groups: untreated (n = 9), steroids (n = 85), or optic canal decompression surgery (n = 33). All the participants in the surgical group, except for one, also received steroids. After adjustment for baseline visual acuity, there were no significant differences between the three treatment groups; visual acuity increased by three lines or more in 57% of the untreated group, 52% of the steroid group and 32% of the surgery group, (P = 0.22).

Surgical complications

In the IONTS, three of the 33 patients (10%) who underwent external decompression suffered postoperative cerebrospinal fluid leak, with one developing meningitis (Levin 1999). Another case series reported accidental dural exposure in 5% of patients who underwent endoscopic optic nerve decompression (Jiang 2001). In the light of the relatively high rate of spontaneous visual improvement in indirect TON, the decision to subject the patient to a surgical intervention with potentially serious complications must be even more circumspect.

Optic canal fracture

Some authorities make an argument for imaging the optic canal in all TON cases. Their rationale is that if a canalicular fracture is found, with a bone fragment impinging on the optic nerve, this is an indication for prompt surgical intervention (Lee 2000; Levin 2003). However, there is no evidence to support the assertion that these cases actually benefit more from surgery. On the contrary some, although not all, studies actually identify the presence of an optic canal fracture as a poor visual prognostic factor whatever the intervention, be it steroids or surgery (Rajiniganth 2003; Tandon 1994; Wang 2001). This makes sense biologically since bone fragments presumably are more likely to transect retinal ganglion cell axons resulting in irreversible injury and decompressing the optic canal in this situation would not restore function. The clinical usefulness of universal neuroimaging in TON therefore remains debatable since there is no consistent correlation between the finding of a fracture, severity of visual loss and prognosis for visual recovery, with or without surgical intervention.

AUTHORS' CONCLUSIONS

Implications for practice

There is no conclusive evidence that any particular form of surgical decompression improves the visual outcome in TON. The decision to proceed with surgery in TON remains controversial and each case needs to be assessed on its own merits. The final decision will inevitably reflect a combination of clinical judgement, the availability of local surgical expertise and the patient's perception of the possible risks and benefits.

Implications for research

Setting up an adequately-powered, randomised controlled trial of surgery in TON poses several difficulties, crucial ones being the ability to recruit enough patients and standardising the surgical intervention across several centres. It is debatable as to whether the resources will ever be available for such a major undertaking. Given the current lack of evidence regarding treatment benefit there also needs to be more emphasis on what primary prevention measures would be effective in reducing the incidence of TON. Some groups, as detailed in the epidemiology section of this review, are clearly at a higher risk and should be targeted. The role of neuroprotective strategies in TON is currently experimental and further animal and human studies are required regarding the usefulness of these agents.

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REFERENCES

References to studies excluded from this review

Gupta 2007 *{published data only}*

* Gupta AK, Gupta AK, Gupta A, Malhotra SK. Traumatic optic neuropathy in pediatric population: early intervention or delayed intervention?. *International Journal of Pediatric Otorhinolaryngology* 2007;**71**(4):559-62.

Additional references

al-Qurainy 1991

al-Qurainy IA, Stassen LF, Dutton GN, Moos KF, el-Attar A. The characteristics of midfacial fractures and the association with ocular injury: a prospective study. *British Journal of Oral and Maxillofacial Surgery* 1991;**29**(5):291-301.

Anderson 1982

Anderson RL, Panje WR, Gross CE. Optic nerve blindness following blunt forehead trauma. *Ophthalmology* 1982;**89**(5):445-55.

Chou 1996

Chou PI, Sadun AA, Chen YC, Su WY, Lin SZ, Lee CC. Clinical experiences in the management of traumatic optic neuropathy. *Neuro-Ophthalmology* 1996;**16**(6):325-36.

Cook 1996

Cook MW, Levin LA, Joseph MP, Pinczower EF. Traumatic optic neuropathy. A meta analysis. *Archives of Otolaryngology - Head and Neck Surgery* 1996;**122**(4):389-92.

Crompton 1970

Crompton MR. Visual lesions in closed head injury. *Brain* 1970;**93**(4):785-92.

Edmund 1963

Edmund J, Gotfredsen E. Unilateral optic atrophy following head injury. *Acta Ophthalmologica* 1963;**41**(6):693-7.

Glanville 2006

Glanville JM, Lefebvre C, Miles JN, Camosso-Stefinovic J. How to identify randomized controlled trials in MEDLINE: ten years on. *Journal of the Medical Library Association* 2006;**94**(2):130-6.

Gross 1981

Gross CE, DeKock JR, Panje WR, Hershkowitz N, Newman J. Evidence for orbital deformation that may contribute to monocular blindness following minor frontal head trauma. *Journal of Neurosurgery* 1981;**55**(6):963-6.

Higgins 2011

Higgins JPT, Altman DG, Sterne JAC (editors). Chapter 8: Assessing risk of bias in included studies. In: Higgins JPT, Green S (editors). Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0 (updated March 2011). The Cochrane Collaboration, 2011. Available from www.cochrane-handbook.org.

Jiang 2001

Jiang RS, Hsu CY, Shen BH. Endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy. *Rhinology* 2001;**39**(2):71-4.

Joseph 1990

Joseph MP, Lessell S, Rizzo J, Momose KJ. Extracranial optic nerve decompression for traumatic optic neuropathy. *Archives of Ophthalmology* 1990;**108**(8):1091-3.

Lange 2009

Lange C, Feltgen N, Junker B, Schulze-Bonsel K, Bach M. Resolving the clinical acuity categories "hand motion" and "counting fingers" using the Freiburg Visual Acuity Test (FrACT). *Graefe's Archive for Clinical and Experimental Ophthalmology* 2009;**241**(1):137-42.

Lee 2000

Lee AG. Traumatic optic neuropathy. *Ophthalmology* 2000;**107**(5):814.

Lee 2010

Lee V, Ford RL, Xing W, Bunce C, Foot B. Surveillance of traumatic optic neuropathy in the UK. *Eye* 2010;**24**(2):240-50.

Lessell 1989

Lessell S. Indirect optic nerve trauma. *Archives of Ophthalmology* 1989;**107**(3):382-6.

Levin 1999

Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. *Ophthalmology* 1999;**106**(7):1268-77.

Levin 2003

Levin LA, Baker RS. Management of traumatic optic neuropathy. *Journal of Neuro-Ophthalmology* 2003;**23**(1):72-5.

Li 2008

Li H, Zhou B, Shi J, Cheng L, Wen W, Xu G. Treatment of traumatic optic neuropathy: our experience of endoscopic optic nerve decompression. *Journal of Laryngology and Otology* 2008;**122**(12):1325-9.

Mahapatra 1993

Mahapatra AK, Tandon DA. Traumatic optic neuropathy in children: A prospective study. *Paediatric Neurosurgery* 1993;**19**(1):34-9.

Mauriello 1992

Mauriello JA, DeLuca J, Krieger A, Schulder M, Frohman L. Management of traumatic optic neuropathy - a study of 23 patients. *British Journal of Ophthalmology* 1992;**76**(6):349-52.

Mine 1999

Mine S, Yamakami I, Yamamura A, Hanawa K, Ikejiri M, Mizota A, et al. Outcome of traumatic optic neuropathy. *Acta Neurochirurgica* 1999;**141**(1):27-30.

Nau 1987

Nau HE, Gerhard L, Foerster M, Nahser HC, Reinhardt V, Joka T. Optic nerve trauma: clinical, electrophysiological and histological remarks. *Acta Neurochirurgica* 1987;**89**(1-2):16-27.

Raji 1982

Raji MR, Manfredi SJ, Gabriele OF, Sprinkle PM, Weinstein GW. Computerized tomography-evaluation in facial trauma associated with blindness. *American Journal of Neuroradiology* 1982;**3**(1):92.

Rajiniganth 2003

Rajiniganth MG, Gupta AK, Gupta A, Bapuraj JR. Traumatic optic neuropathy: visual outcome following combined therapy protocol. *Archives of Otolaryngology - Head and Neck Surgery* 2003;**129**(11):1203-6.

Schulze-Bonsel 2006

Schulze-Bonsel K, Feltgen N, Burau H, Hansen L, Bach M. Visual acuities "hand motion" and "counting fingers" can be quantified with the Freiburg visual acuity test. *Investigative Ophthalmology and Visual Science* 2006;**47**(3):1236-40.

Seiff 1984

Seiff SR, Berger MS, Guyon J, Pitts LH. Computed tomographic evaluation of the optic canal in sudden traumatic blindness. *American Journal of Ophthalmology* 1984;**98**(6):751-5.

Seiff 1990

Seiff SR. High dose corticosteroids for treatment of vision loss due to indirect injury to the optic nerve. *Ophthalmic Surgery* 1990;**21**(6):389-95.

Spoor 1990

Spoor TC, Hartel WC, Lensink DB, Wilkinson MJ. Treatment of traumatic optic neuropathy with corticosteroids. *American Journal of Ophthalmology* 1990;**110**(6):665-9.

Steinsapir 1994

Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. *Survey of Ophthalmology* 1994;**38**(6):487-518.

Steinsapir 1998

Steinsapir KD, Goldberg RA. Traumatic optic neuropathies. In: Miller NR, Newman NJ editor(s). Walsh and Hoyt's Clinical Neuro-Ophthalmology. Baltimore, Maryland: Williams and Wilkins, 1998:715-39.

Steinsapir 2005

Steinsapir KD, Goldberg RA. Traumatic optic neuropathy: A critical update. *Comprehensive Ophthalmology Update* 2005;**6**(1):11-21.

Takehara 1994

Takehara S, Tanaka T, Uemura K, Shinohara Y, Yamamoto T, Tokuyama T, et al. Optic nerve injury demonstrated by MRI with STIR sequences. *Neuroradiology* 1994;**36**(7):512-4.

Tandon 1994

Tandon D, Thakar A, Mahapatra AK, Ghosh P. Trans-ethmoidal optic nerve decompression. *Clinical Otolaryngology and Allied Sciences* 1994;**19**(2):98-104.

Wang 2001

Wang BH, Robertson BC, Girotto JA, Liem A, Miller NR, Iliff N, et al. Traumatic optic neuropathy: a review of 61 patients. *Plastic and Reconstructive Surgery* 2001;**107**(7):1655-64.

Yang 2004

Yang WG, Chen CT, Tsay PK, De Villa GH, Tsai YJ, Chen YR. Outcome of traumatic optic neuropathy - surgical versus nonsurgical treatment. *Annals of Plastic Surgery* 2004;**52**(1):36-42.

Yu-Wai-Man 2013

Yu-Wai-Man P, Griffith P. Steroids for traumatic optic neuropathy. *Cochrane Database of Systematic Reviews* 2013, Issue 6. [DOI: 10.1002/14651858.CD006032.pub4]

References to other published versions of this review

Yu-Wai-Man 2005

Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. *Cochrane Database of Systematic Reviews* 2005, Issue 4. [DOI: 10.1002/14651858.CD005024.pub2]

* Indicates the major publication for the study

CHARACTERISTICS OF STUDIES

Characteristics of excluded studies *[ordered by study ID]*

Study	Reason for exclusion
Gupta 2007	Observational study.

APPENDICES

Appendix 1. CENTRAL search strategy

#1 MeSH descriptor Optic Nerve
#2 MeSH descriptor Optic Nerve Diseases
#3 MeSH descriptor Optic Nerve Injuries
#4 optic near nerve*
#5 optic near neuropath*
#6 optic near injur*
#7 optic near trauma*
#8 optic near contusion*
#9 optic near compress*
#10 optic near avulsion*
#11 optic near transection*
#12 optic near damage*
#13 (#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10 OR #11 OR #12)
#14 MeSH descriptor Surgery
#15 MeSH descriptor Surgical Procedures, Operative
#16 surg* or operat*
#17 (#14 OR #15 OR #16)
#18 (#13 AND #17)

Appendix 2. MEDLINE (OvidSP) search strategy

1 randomized controlled trial.pt.
2 (randomized or randomised).ab,ti.
3 placebo.ab,ti.
4 dt.fs.
5 randomly.ab,ti.
6 trial.ab,ti.
7 groups.ab,ti.
8 or/1-7
9 exp animals/
10 exp humans/
11 9 not (9 and 10)
12 8 not 11
13 exp optic nerve/
14 exp optic nerve diseases/
15 exp optic nerve injuries/
16 (optic adj2 nerve$).tw.
17 (optic adj2 neuropath$).tw.
18 (optic adj3 injur$).tw.
19 (optic adj3 trauma$).tw.
20 (optic adj3 contusion$).tw.
21 (optic adj3 compress$).tw.
22 (optic adj3 avulsion$).tw.
23 (optic adj3 transection$).tw.
24 (optic adj3 damage$).tw.
25 or/13-24
26 exp surgery/
27 exp surgical procedures operative/
28 (surg$ or operat$).tw.
29 or/26-28
30 25 and 29
31 12 and 30

The search filter for trials at the beginning of the MEDLINE strategy is from the published paper by Glanville (Glanville 2006).

Appendix 3. EMBASE (OvidSP) search strategy

1. exp randomized controlled trial/
2. exp randomization/
3. exp double blind procedure/

4. exp single blind procedure/
5. random$.tw.
6. or/1-5
7. (animal or animal experiment).sh.
8. human.sh.
9. 7 and 8
10. 7 not 9
11. 6 not 10
12. exp clinical trial/
13. (clin$ adj3 trial$).tw.
14. ((singl$ or doubl$ or trebl$ or tripl$) adj3 (blind$ or mask$)).tw.
15. exp placebo/
16. placebo$.tw.
17. random$.tw.
18. exp experimental design/
19. exp crossover procedure/
20. exp control group/
21. exp latin square design/
22. or/12-21
23. 22 not 10
24. 23 not 11
25. exp comparative study/
26. exp evaluation/
27. exp prospective study/
28. (control$ or prospectiv$ or volunteer$).tw.
29. or/25-28
30. 29 not 10
31. 30 not (11 or 23)
32. 11 or 24 or 31
33. exp optic nerve/
34. exp optic nerve disease/
35. exp optic nerve injury/
36. (optic adj2 nerve$).tw.
37. (optic adj2 neuropath$).tw.
38. (optic adj3 injur$).tw.
39. (optic adj3 trauma$).tw.
40. (optic adj3 contusion$).tw.
41. (optic adj3 compress$).tw.
42. (optic adj3 avulsion$).tw.
43. (optic adj3 transection$).tw.
44. (optic adj3 damage$).tw.
45. or/33-44
46. exp surgery/
47. exp surgical technique/
48. (surg$ or operat$).tw.
49. or/46-48
50. 45 and 49
51. 32 and 50

Appendix 4. LILACS search strategy

optic neuropath$ and surg$

Appendix 5. *meta*Register of Controlled Trials search strategy

optic neuropathy AND surgery

Appendix 6. ClinicalTrials.gov search strategy

Optic Neuropathy AND Surgery

Appendix 7. ICTRP search strategy

Optic Neuropathy = Condition AND Surgery = Intervention

WHAT'S NEW

Date	Event	Description
17 June 2013	New search has been performed	Issue 6, 2013: Electronic searches updated.
17 June 2013	New citation required but conclusions have not changed	Issue 6, 2013: No new trials met the inclusion criteria.

HISTORY

Protocol first published: Issue 4, 2004
Review first published: Issue 4, 2005

Date	Event	Description
6 December 2010	New search has been performed	Issue 1, 2011: Updated searches yielded no new trials.
12 October 2008	Amended	Converted to new review format.
4 August 2005	New citation required and conclusions have changed	Substantive amendment

CONTRIBUTIONS OF AUTHORS

Conceiving the review: PYWM
Designing the review: PYWM
Coordinating the review: PYWM
Screening search results: PYWM, PGG
Obtaining and screening data on unpublished studies: PYWM, PGG
Writing the review: PYWM

DECLARATIONS OF INTEREST

None known.

SOURCES OF SUPPORT

Internal sources

- Department of Ophthalmology, Royal Victoria Infirmary, Newcastle-upon-Tyne, UK.

External sources

- No sources of support supplied

INDEX TERMS

Medical Subject Headings (MeSH)

Optic Nerve Injuries [*surgery]

MeSH check words

Humans