

Efficacy of Transcutaneous Transseptal Orbital Decompression in Treating Acute Retrobulbar Hemorrhage and a Literature Review

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Abstract

Decreasing visual acuity secondary to orbital trauma may be caused by sudden space-occupying or expanding intraorbital lesions, including retrobulbar hemorrhage (RBH), herniation, or swelling. RBH must be diagnosed and treated immediately. This article addresses the efficacy of transcutaneous transseptal orbital decompression in a combination with a systematic review of the literature for a comparison of this method with existing treatment options. For this study the department's database was retrospectively screened for patients with acute RBH who were treated between 2009 and 2011 using the authors' approach. Patients presenting with RBH were classified into RBH classes I to III according to three different clinical and radiological manifestations of acute RBH. The efficacy of transcutaneous transseptal orbital decompression was assessed by postoperative visual acuities. The literature review was performed by using the MEDLINE database. The time period for the study was between 2009 and 2011 during which 10 patients were diagnosed with suspected RBH and 9 were treated with the authors' technique. Visual acuities were reconstituted or maintained in almost 86% of patients who were diagnosed and treated according to the authors approach and who survived initial trauma. It was concluded that transcutaneous transseptal orbital decompression provides an efficient and rapid approach for treating patients with acute RBH. By distinguishing three different manifestations of acute RBH, the authors present a diagnostic tool that may facilitate classification of RBH and determination of treatment options.

Keywords

- ▶ retrobulbar hemorrhage
- ▶ retrobulbar hematoma
- ▶ orbital trauma
- ▶ VEP
- ▶ amaurosis
- ▶ optic nerve trauma

Etiology

Retrobulbar hemorrhage (RBH) is an uncommon but potentially blinding orbital emergency in which immediate diagnosis and treatment are necessary to maintain or reconstitute

visual acuity. The literature indicates that two main causes of RBH can be distinguished as: traumatic and nontraumatic. Traumatic causes of RBH can be further classified into non-penetrating and penetrating trauma. Penetrating orbital trauma is essentially iatrogenic. The most common causes

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are surgical and anesthesiological procedures performed in the periorbital region, whereas impalement injuries are less frequent.¹ RBH has been reported to be the most common risk of peri- and retrobulbar anesthesia²⁻⁵ and the main cause of loss of vision after blepharoplasty.^{6,7} In addition, several cases of RBH following sub-Tenon block have been reported.⁸⁻¹¹ However, orbital surgery and procedures performed in adjacent anatomical sites, including the sinus and oral cavity, may give rise to intraorbital hemorrhage.¹² The rate of orbital complications during sinus surgery is approximately 0.5 to 5%.^{13,14} In nonpenetrating orbital trauma, fractures of the orbit following blunt facial trauma may be associated with RBH, and loss of vision occurs in 0.3% of these patients.^{15,16} However, drug therapy¹⁷ or hemorrhage due to congenital or systemic conditions¹⁸⁻²⁰ may lead to nontraumatic RBH. Drug-related nontraumatic RBH occurs in hemorrhagic diatheses due to circulating anticoagulation medication,^{21,22} whereas vessel malformations are congenital causes of RBH.²³ RBH can occur spontaneously in drug-related pharmacological or congenital conditions.²⁴ Ischemia of the optic nerve is the main underlying pathophysiological cause²⁵ of loss of vision in RBH. Hemorrhage or swelling caused by an expanding intraorbital lesion²⁶ results in elevated pressure in the orbital compartment; this condition occludes the central retinal artery, leading to mechanical compression of both the optical nerve and the afferent blood supply. Both pathways result in optic nerve ischemia and optic nerve neuropathy.²⁷ Indirect damage to the optic nerve, which occurs in 0.5 to 5% of closed head injuries, is thought to be the most common form of traumatic optic neuropathy.²⁸ However, in the context of traumatic optic neuropathy, total rupture (avulsion) of the optic nerve is reported to be an independent entity.²⁹

Diagnosis, Signs, and Symptoms

The most significant signs and symptoms of acute RBH include decreasing visual acuity, axial proptosis, and a fixed and dilated pupil (→ **Fig. 1A, B**) or a relative afferent pupillary defect (RAPD). Severe pain, monocular hematoma, swelling of the periorbital region, ophthalmoplegia, chemosis, and subconjunctival hemorrhage³⁰ are common but nonspecific signs of RBH. The basic elements of primary clinical examination are testing visual acuity and pupillary light reflexes, especially the swinging flashlight test.^{31,32} However, these elements are not applicable in noncooperative patients or when clinical tests are compromised because of unconsciousness, morphine medication, or extensive periorbital swelling. In these cases, electrophysiological testing of the visual pathway, such as flash-evoked visual potentials (VEPs) and electroretinograms (ERGs), can be important diagnostic tools for distinguishing between reversible and irreversible optic nerve damage and for selecting either surgical decompression or conservative therapy.³³

Existing Treatment Options

Lateral canthotomy and cantholysis are the standard emergency surgical treatments for patients with acute RBH.³⁴

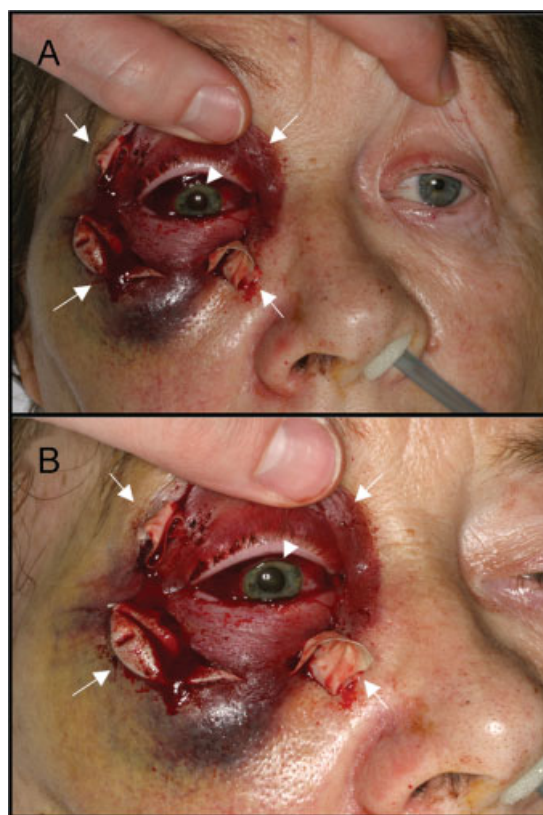


Figure 1 Clinical image of a patient with acute retrobulbar hemorrhage. (A) and (B) Patient's right eye postoperatively. Arrows indicate the four incisions with elastic Penrose drainages and the arrowheads indicate the dilated pupil, nonreacting, or with relative afferent pupillary defect.

Conservative therapy is considered as an adjuvant option and includes steroids, mannitol, and acetazolamide.³⁵ Although lateral canthotomy and other medical options may facilitate the release of intraorbital pressure, they are limited by the lack of drainage of the hemorrhage from the orbital compartment. In addition, the transseptal approach does not affect the lateral canthal tendon, a structure with fundamental roles in eyelid attachment, function, and esthetics.

Aims

The aim of this study was to provide both an efficient alternative to existing treatment options and to present a diagnostic and therapeutic algorithm for simplifying the treatment decisions in patients who present with acute RBH. In this study, we evaluated the efficacy and potential complications of transcutaneous transseptal orbital decompression, described the procedure in a stepwise fashion, and performed a statistical analysis of the patient group. Furthermore, we also performed a systematic review to address the differences with existing diagnostic and therapeutic options.

Methods

Patients and Literature Review

The database of the Department of Oral and Maxillofacial Surgery of Hannover Medical School was retrospectively

Table 1 Signs, symptoms, and radiological features of acute retrobulbar hemorrhage

	Clinical signs and symptoms (►Fig. 1)	Radiological features (►Fig. 2)	
Specific	<ul style="list-style-type: none"> – Decreasing visual acuity – Axial proptosis – Fixed and dilated pupil/RAPD 	<ul style="list-style-type: none"> – Retrobulbar hyperdensity – Stretched optical nerve – Tulip-shaped bulb 	Space-occupying lesion
Nonspecific	<ul style="list-style-type: none"> – Hematoma – Diplopia – Ophthalmoplegia – Pain and swelling 	<ul style="list-style-type: none"> – Orbital fractures – Intraorbital hemorrhage – Subperiosteal hemorrhage – Palpebral hematoma 	Nonspace-occupying lesion

Abbreviation: RAPD, relative afferent pupillary reflex.

screened for records of patients with orbital trauma and acute RBH who visited the department between January 2009 and December 2011. After obtaining informed consent, patient's charts and surgical records were reviewed for diagnosis and treatment, including the duration of the surgical procedure, outcome, complications, and follow-up. The mean patient age is expressed as mean years \pm standard deviation.

Patients presenting with RBH were classified into RBH classes I to III according to three different clinical and radiological manifestations of acute RBH and the efficacy of transseptal orbital decompression was measured by assessing postoperative visual acuity of the affected eye.

A literature review was undertaken using the MEDLINE database (1950–2011), supplemented by further review of any additional relevant publications quoted in the articles. The initial search was based on the keywords “RBH/hemorrhage,” “retrobulbar haematoma/hematoma” and “lateral canthotomy/cantholysis.”

Manifestation of Acute RBH, Classification and Diagnostic Algorithm

We distinguished three different clinical and radiological manifestations of acute RBH and used these cases for the classification of RBH into classes I to III. The most significant clinical and radiological findings were divided into specific and nonspecific clinical signs and symptoms and radiological findings, respectively. Treatment was determined according to the following classification (►Tables 1, 2): *RBH class I* was defined as a patient with a history of orbital trauma presenting with either nonspecific clinical signs or nonspecific radiological features (►Table 1) of a possible RBH, while *class II RBH* was based on either typical clinical signs or typical radiological features (►Table 1). The typical clinical signs of RBH (►Table 1, ►Fig. 1A,B) were defined as decreasing visual acuity, axial proptosis, and a fixed and dilated right pupil.³⁶ Typical radiological features in native computed tomography (CT) scans were defined as space-occupying or expanding retrobulbar hyperdense lesions consistent with hemorrhage, axial proptosis, a stretched optic nerve, and a tulip-shaped bulb (►Fig. 2A). These features result from elevated intra-orbital pressure and volume shift.³⁷ RBH class III was defined as a combination of both typical clinical signs and typical radiological features. Patients diagnosed with RBH class I were not treated but were monitored closely while RBH class

II or above were immediately treated. It has to be noted that fractures of the orbit are frequently associated with nonspecific orbital hemorrhage (►Table 1; RBH class I) and RBH classes II and III need to be excluded.

Flash-Evoked Visual Potentials and Electroretinograms

During craniomaxillofacial reconstructions and postoperative care of patients with RBH resulting from head injuries, flash-VEPs, and ERGs can be used to estimate and monitor the functionality and integrity of the visual pathway.^{26,33} In contrast to pattern stimulation, flash-VEPs can be used in uncooperative patients or when optical factors complicate detection.³⁸ Furthermore, VEP and ERG can be recorded simultaneously.³⁹ In our study, two recordings of each VEP condition were obtained (blue dotted curve in ►Fig. 3) to verify reproducibility. A normal flash-VEP and ERG is shown in ►Fig. 3, in which the VEP consists of a series of negative (N) and positive (P) peaks. Abnormal VEPs and ERGs differ in terms of the amplitude ($> 50\%$ interocular variability) and the peak latency (> 150 ms) of the electric signal. The flash-VEP consists of three main components (N75, P100, and N140), which can be distinguished in ►Fig. 3. Of these three components, the P100 peak is the most reliable.

Transcutaneous Transseptal Orbital Decompression Technique

Emergent surgical decompression of the orbit was performed in the following stepwise fashion (►Fig. 4A–E).

Table 2 Classification and therapy of acute retrobulbar hemorrhage

Classification	Description	Therapy
RBH class I	Nonspecific signs and/or radiological features	Vision Monitoring > 24 h
RBH class II	Specific signs or specific radiological features	Surgical decompression + Megadose regimen + Vision monitoring > 48 h
RBH class III	Specific signs and specific radiological features	

Abbreviations: h, hour; RBH, retrobulbar hemorrhage.

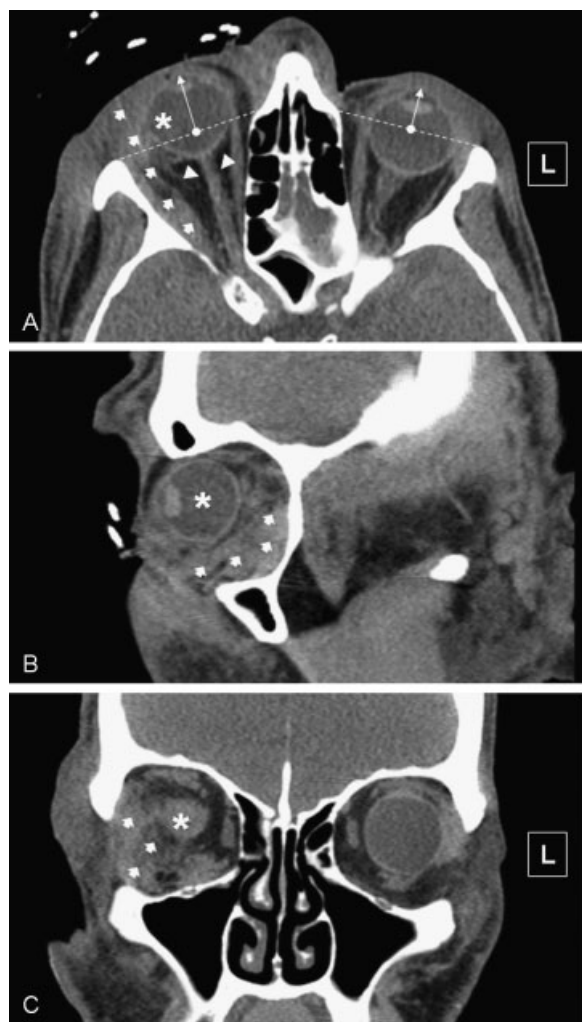


Figure 2 Native computerized tomography of a patient with acute retrobulbar hemorrhage (RBH). (A) axial; (B) sagittal; and (C) coronal view of typical radiological features of RBH. Asterisks indicate the tulip-shaped bulb and arrows show axial proptosis. Fat arrows show retrobulbar hemorrhage and the arrow heads show stretched and elongated optical nerve.

- (A) The surgical area of the affected side was prepared in a sterile manner by cleaning with topical antiseptics, if possible. Care was taken not to rinse the eye itself.
- (B) Local anesthesia and hemostasis were achieved by blocking the infra- and supraorbital nerve, and the paranasal and lateral orbital areas were infiltrated with an epinephrine-containing local anesthetic, with the needle directed away from the eye. Care was taken not to inject into the eye or the orbit itself.
- (C) Four 5-mm incisions were made in the natural crease located 2 to 3 mm lateral and medial from the eyelid margins of the upper and lower eyelids. The four incisions were oriented accurately while following the natural skin folds of the eyelids.
- (D) After dissecting the orbicularis muscle and the orbital septum (palpebral ligament), the four incisions were elongated in a strictly subperiosteally manner into the orbit behind the globe by using scissors.

(E) Elastic Penrose drainage tubes were inserted to release extraconal and intraconal hematomas from the orbit. Penrose drainage tubes must be fixated via suture. Blood flow from the drainages was observed immediately after insertion. The drainage tubes were removed 3 to 5 days after decompression.

Conservative and Adjuvant Medical Management

All patients received the methylprednisolone megadose regimen, starting with a single shot of 30-mg Urbason (Sanofi Aventis, Frankfurt, Germany)/kg body weight intravenously and 5.4 mg/kg/h for 47 hour to treat or prevent trauma to the optical nerve and swelling of orbital tissues.⁴⁰ Blood sugar levels were monitored closely. Postoperative pain relief was achieved by intravenous administration of analgesics (The World Health Organization Stage I).

Results

Retrospective Cohort Analysis

Between January 2009 and December 2011, 484 patients with orbital trauma were diagnosed and treated in our department. The patient group consisted of 126 female patients and 358 male patients with ages ranging from 4 to 97 years (mean age, 42.1 ± 18.8 y).

Patients with RBH class I and nonspecific clinical and radiological findings were not treated and were therefore not part of the study. Their visual acuities were monitored closely for 24 hours and remained normal.

In total, 10 patients (2.1%) were diagnosed with acute RBH class II or above (►Table 3).

Class-III-RBH was found in only 0.83% of patients with orbital trauma. The mean age of patients treated for acute RBH (classes II + III) was 59.9 years (range, 20–89 y) with a female to male (2.33) ratio of 7:3. Although the mean duration of surgical decompression ranged from 10 to 20 minute, the procedure was occasionally extended because of accompanying soft tissue lacerations. The outcomes of patients with acute RBH are shown in ►Table 4. Of the 10 patients, 9 were treated with a combination therapy of transcutaneous transseptal orbital decompression and the methylprednisolone megadose regimen, and 1 patient refused to undergo surgical treatment. The patient who refused surgery received conservative treatment. Among the patients who received combination therapy, two intubated and ventilated polytraumatized patients underwent full body CT scans that revealed typical radiological findings of RBH. Both received prophylactic transcutaneous transseptal orbital decompression followed by the methylprednisolone megadose regimen since visual acuities were not assessable. Unfortunately, the two polytraumatized patients included in this study did not survive initial trauma and could not be followed postoperatively. After orbital decompression, visual acuity improved in all but one case (patient 8). This outcome in patient 8 may be explained by the fact that this patient was not diagnosed with acute RBH in a regional hospital and was transferred to our department with a 24-hour delay after trauma.

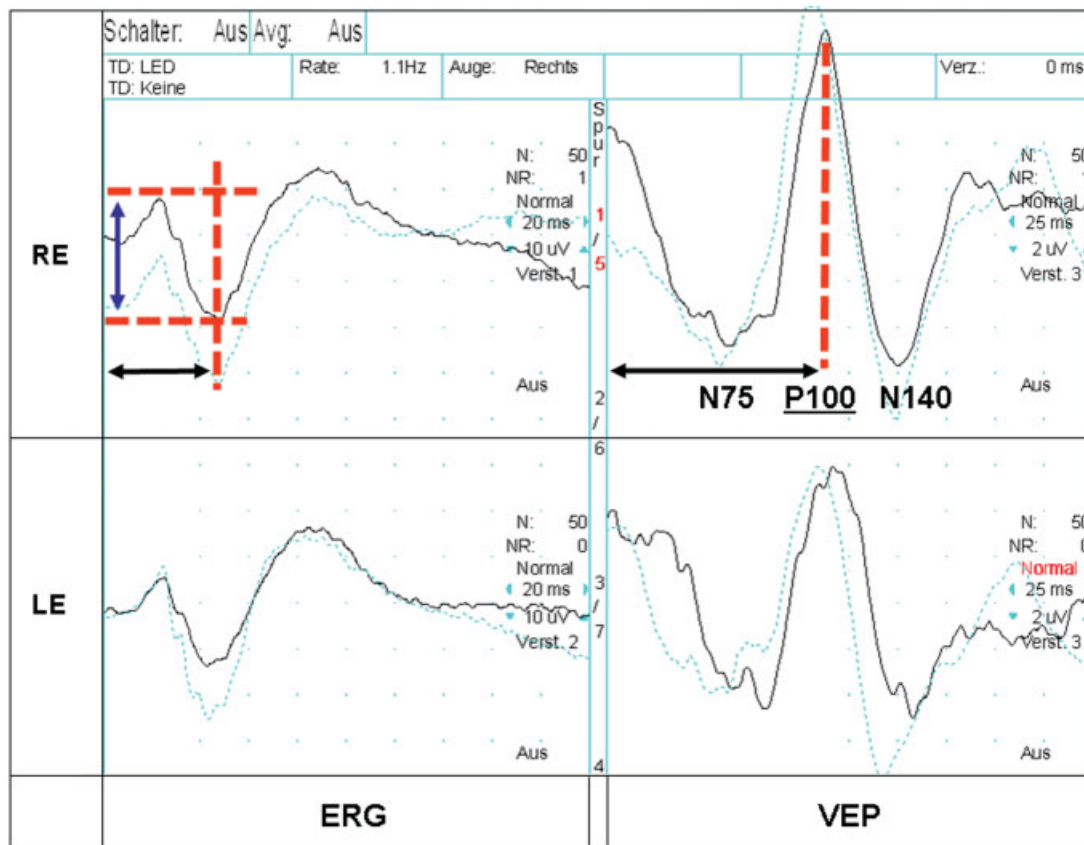


Figure 3 Normal flash evoked visual potential (right) and electroretinogram (left). RE, right eye; LE, left eye; blue arrow, amplitude (μV = microvolts); black arrow, peak latency; N75, negative peak wave at 75 milliseconds poststimuli; P100, positive peak wave at 100 milliseconds poststimuli; and N140, negative peak wave at 140 milliseconds poststimuli.

According to the authors' diagnostic and therapeutic algorithm, visual acuities were either reconstituted or maintained in almost 86% of patients (6/7) who presented with any stage of RBH, who survived initial trauma, and whose vision could be monitored postoperatively.

Taken together, the results indicate that 2% of patients with orbital trauma present with suspected RBH, and the diagnosis of acute RBH can be confirmed in approximately 0.83% of patients. Elderly women and severely injured patients are at a higher risk of developing acute RBH.

Adverse Events

As with any minor surgical procedure, there is a risk of infection, hemorrhage, scarring, and mechanical injury of the globe (**►Fig. 5**). Major risks include palsy of the infra- and supraorbital nerve and injury of the ocular muscles. Any palpebral approach to the orbit involves risks and complications such as the development of entropion or ectropion. Only one case (patient 8) developed infraorbital palsy, but this was due to a preexisting eyelid laceration and did not result from our approach. Furthermore, patient 5 required treatment for postoperative wound infection (**►Table 4**). None of the patients developed ocular motility disorder, entropion, ectropion, or other severe side effects. It should be noted that minor complications respond to therapy, but optical nerve ischemia does not.

Discussion

Recommendations for RBH diagnosis and treatment vary in the literature.^{25,34,35,41–44} Evaluation of visual acuity and pupillary light reflexes are fundamental components of any clinical examination of patients presenting with craniomaxillofacial injuries. Although pupil morphology and diameter should be assessed, anisocoria is not a sign of defects in the afferent nervous pathway. However, the swinging flashlight test is a reliable clinical test to exclude primary damage to the optical nerve. For patients presenting to the emergency department (ED) with changes in vision or even loss of vision, the underlying causes can be attributed to trauma, systemic disorders, or a combination of both. Potentially sight-threatening lesions must be recognized and immediately differentiated from other causes. Thus, it is essential to follow simple diagnostic algorithms and take the patient's history to identify these rare patients among those with simple eye complaints. Deterioration or loss of vision is considered the pivotal clinical sign of RBH and should be immediately treated. Further diagnosis and imaging must not delay therapy in patients with a history of blunt orbital trauma or orbital surgery and deterioration or loss of vision. However, changes in vision might also be caused by nontraumatic occlusion of the central retinal artery or vein, acute angle-closure glaucoma, temporal arteritis, or detachment of the retina.⁴⁵ In addition to changes in vision, the most significant symptoms of RBH are axial

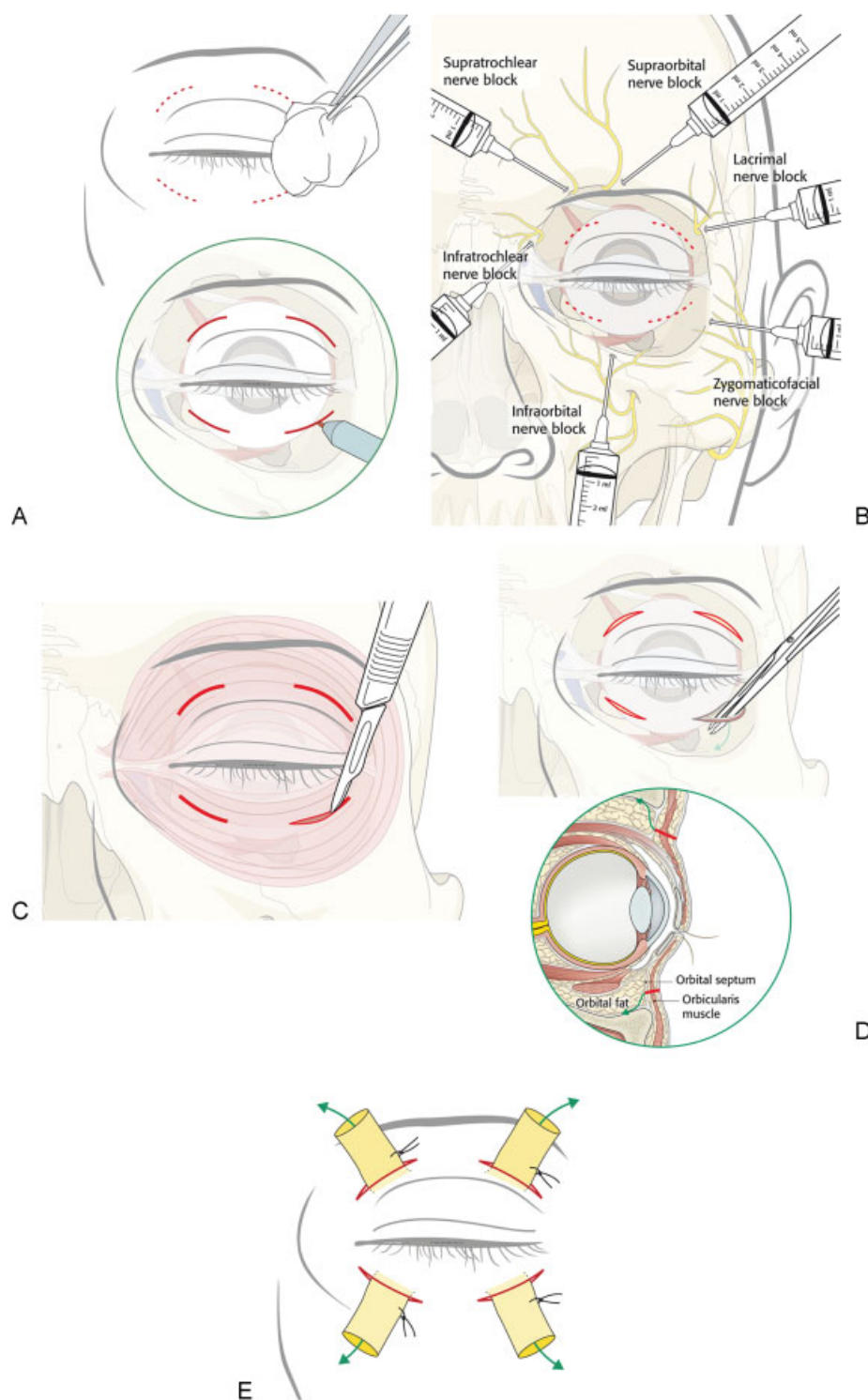


Figure 4 Transcutaneous transseptal orbital decompression technique. (A–E) Diagrammatic representation of a transcutaneous transseptal orbital decompression in a stepwise fashion. Image courtesy AO Foundation, Davos, Switzerland.

proptosis, a fixed and dilated pupil, or RAPD. Although non-specific clinical signs vary, the most common signs are listed in ►Table 1. Patients should immediately undergo surgery if RBH is suspected, but visual acuity is not assessable because of the state of awareness, general anesthesia, or distinct peri-orbital swelling. VEP and ERG can help physicians decide whether surgery is appropriate in these cases.

Imaging

Suspected RBH should be managed surgically without performing further diagnostic procedures because rapid diagnosis is required for successful treatment.^{26,46} Otherwise, any patient with suspected midface injuries, especially of the orbit, usually undergoes CT scan because skull base, orbital apex, and ocular injuries require rapid identification. In

Table 3 Summary of patients with acute retrobulbar hemorrhage classes II/III

Patient	Sex/Age	Etiology	CT	VA preop	RBH class	Therapy
1	♂61	iatrogenic blepharoplasty	yes	LP	III	TTOD MPMR
2	♀75	iatrogenic reconstruction	no	LOV	II	TTOD MPMR
3	♀76	blunt trauma collapse	yes	n.a.	II	TTOD MPMR
4	♂20	blunt trauma orbital fracture	yes	4/20	III	MPMR
5	♂42	iatrogenic reconstruction	yes	02/20	III	TTOD MPMR
6	♀53	severely injured orbital fracture	yes	n.a.	II	TTOD MPMR
7	♀49	severely injured orbital fracture	yes	n.a.	II	TTOD MPMR
8	♀83	blunt trauma orbital fracture	yes	LOV	III	TTOD MPMR
9	♀81	blunt trauma orbital fracture	yes	LP	II	TTOD MPMR
10	♀56	severely injured orbital fracture	yes	n.a.	II	TTOD MPMR

Abbreviations: CT, computed tomography; LOV, loss of vision; LP, light perception; MPMR, methylprednisolone megadose regimen; n.a., not assessable; preop, preoperative; RBH, retrobulbar hemorrhage; TTOD, transcutaneous transseptal orbital decompression; VA, visual acuity; VEP, evoked visual potential.

Table 4 Results of transseptal decompression

Patient	Anesthesia	OP duration	VA preop	VA postop	Adverse event
1	GA	15 min	LP	16/20	
2	LA	10 min	LOV	20/20	
3	GA	70 min	n.a.	6/20	
4		No surgery	4/20	16/20	
5	GA	20 min	2/20	20/20	Wound infection
6	GA	45 min	n.a.	n.a.	
7	GA	20 min	n.a.	n.a.	
8	LA	15 min	LOV	LOV	IO nerve palsy
9	GA	30 min	LP	18/20	
10	GA	30 min	n.a.	20/20	

Abbreviations: GA, general anesthesia; IO, infraorbital; LA, local anesthesia; LOV, loss of vision; LP, light perception; min, minute; n.a., not assessable; postop, postoperative; preop, preoperative; VA, visual acuity.

addition, most severely injured, polytraumatized, and unconscious patients with suspected head injuries undergo trauma body CT scans. Thus, RBH diagnosis is usually image guided. However, a patient's visual acuity should be monitored hourly for the first 24 hours after any type of orbital trauma⁴⁷ to detect delayed RBH. Clinically assessed visual acuity remains the critical parameter for legitimate intervention because RBH and trauma to the optic nerve or swelling of the orbital tissues may affect a patient's vision. Patients with typical radiological findings of RBH should be treated with surgical

intervention. Conscious trauma patients with stable vision and nonspecific radiological findings should be monitored closely for at least 24 hour.

Flash-Evoked Visual Potentials and Electroretinograms

The significance of neuro-ophthalmological examinations is limited by the severity of injuries, the degree of vigilance, and pharmacological influences (e.g., administration of morphine derivatives and barbiturates). Furthermore, it is not possible to distinguish reversible (edema) from irreversible optical

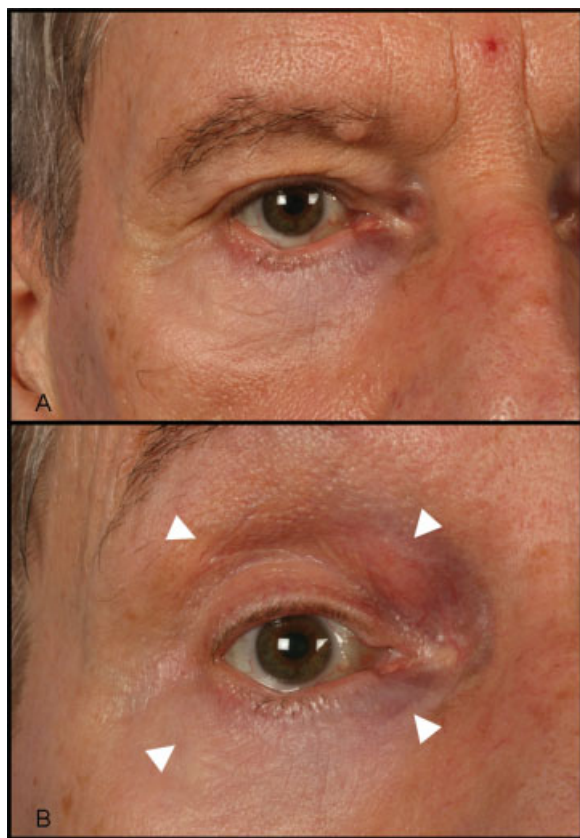


Figure 5 (A) and (B) Images of patient 1-year follow-up after transcutaneous transseptal orbital decompression. Arrowheads, scars of surgical approach.

nerve damage in a clinical setting.⁴⁸ In this case, intraindividual electrophysiological evaluation of the visual pathway (VEP or ERG) might provide a diagnostic tool that accelerates the process of selecting surgical intervention or conservative therapy.

Surgical Approach

Transcutaneous transseptal orbital decompression has evolved from the basic treatment requirements of acute RBH, namely, rapid pressure release and drainage of hemorrhage. Drainage of hematoma from the orbital compartment without osteotomy of orbital walls can only be performed through the orbital septum. Thus, by avoiding time-consuming orbital osteotomies and by combining rapid pressure release with drainage of hemorrhage, the transcutaneous transseptal approach fulfills these requirements. In particular, immediate treatment is mandatory to prevent pressure-related damage to intraorbital neurovascular structures, which may lead to changes in or even loss of vision. The time window for releasing intraorbital pressure is limited to approximately 1 hour from the onset of blindness.^{26,49,50} Lateral canthotomy and inferior cantholysis are the standard procedures for releasing intraorbital pressure but not for evacuation of hematoma. Although the recent literature indicates that this is the safest approach, the risks include hemorrhage, scarring, and mechanical injury to the globe and

ocular muscles. These procedures involve dissection or lysis of the lateral canthal tendon, which might affect eyelid attachment, esthetics, lacrimation flow, and ocular motility.^{27,36,45,51} In contrast, the major risks of the transcutaneous transseptal approach include injuries of the infra- and supraorbital nerve and ocular muscles and direct mechanical injury of the globe. Unlike lateral canthotomy with inferior cantholysis, the transcutaneous approach does not affect the lateral canthal tendon or its important functions. In addition, infraorbital and supraorbital nerve palsy are almost negligible if the transcutaneous incisions are performed correctly. Most importantly, lateral canthotomy fails to evacuate the hemorrhage from the orbital compartment. An extraconal hemorrhage can collect anywhere in the orbit and may be difficult to access through an inappropriately sited incision.³⁰ In some case reports, lateral canthotomy with inferior cantholysis failed to sufficiently decompress the orbit because of severe tension of the globe and periorbital swelling; thus, visual acuity could not be maintained or reconstituted in these cases.²⁰

Taken together, these findings indicate that transcutaneous transseptal orbital decompression provides a rapid intervention which safely releases intraorbital pressure and efficiently drains hemorrhage from the orbital compartment, particularly, in extensive orbital hemorrhage, periorbital swelling or when imaging (CT or magnetic resonance imaging) is not available. Pulsating exophthalmos, which may be a sign of carotid-cavernous sinus fistula, is the only contraindication for surgical treatment. This condition requires appropriate preoperative imaging. Because anesthesiological contraindications do not exist, our surgical approach can be performed in EDs and under local or general anesthesia.

Conservative and Adjuvant Management

Steroids are part of any published conservative or adjuvant medical therapy for acute RBH.^{20–54} In most published cases, conservative treatment was considered an adjuvant option. In addition to steroids, the use of mannitol and acetazolamide is included in almost all published protocols,^{20,35,52} although the administration protocols vary. In contrast, our conservative therapy of choice for the treatment of acute RBH or other sudden expanding retrobulbar lesions or traumatic optic nerve lesions is the methylprednisolone megadose regimen (see Methods).

Conclusions

Acute RBH can be classified into RBH classes I to III, providing a simple tool for rapid identification and treatment of this rare condition. Elderly women and severely injured patients are at a higher risk of developing acute RBH. Visual acuity should be monitored for 24 hour in RBH class I, orbital trauma, or after surgical procedures. RBH class II and class III with either decreasing visual acuity or/and typical radiological features should be treated immediately via surgical decompression. Electrophysiological evaluation (VEP or ERG) can provide diagnostic information for a therapeutic decision in severely injured patients or in those with compromised

clinical tests. Conservative therapy should be considered an absolute exception (patient's refusal). Since objective anesthesiological and surgical contraindications exist only in cases of pulsating exophthalmos, transcutaneous transseptal orbital decompression in combination with the methylprednisolone megadose regimen provides a fast and efficient approach for treating acute RBH. Furthermore, this approach can be used in EDs or intensive care units under local anesthesia. Most importantly, it has to be noted that most procedure-related complications respond to therapy, whereas prolonged ischemia of the optical nerve does not.

Competing Interests

The authors declare that they have no competing interests.

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