

Iatrogenic blindness after minor medical procedures in the face: critical review

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Cegueira iatrogênica após pequenos procedimentos médicos na face: revisão crítica

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RESUMO

Cegueira como consequência de um procedimento cirúrgico menor para uma enfermidade menor é uma experiência chocante e pavorosa tanto para o paciente como para o médico. O presente trabalho relata a maioria dos procedimentos que podem produzir tal complicação, assim como sua provável fisiopatologia, além de orientação para a prevenção e tratamento imediato e adequado deste evento catastrófico, tendo como base uma revisão da literatura sobre o assunto. Cegueira como resultado de injeções de várias substâncias na cabeça e pescoço surge com frequência alarmante na literatura. Estas substâncias variam de parafina, a álcool, agentes anestésicos, tintura de contraste angiográfico, penicilina e corticosteróides, como também combinações de drogas múltiplas. Estas drogas têm um potencial neurotóxico e embólico em comum. Em alguns exemplos, o local de injeção na cabeça e pescoço pode ser longe da artéria oftálmica, como injeções de couro cabeludo e injeções intranasais. Qualquer material injetado na face, nariz ou boca e até mesmo o pescoço superior representa um embolo potencial ao olho. **Objetivo:** Apresentar uma revisão da evidência relacionada aos eventos que conduzem a cegueira iatrogênica associada à manipulação cirúrgica facial em procedimentos secundários. **Material e Método:** Foram levantados documentos de 1906 a 2006, oriundos das principais bibliotecas eletrônicas. Um total de 58 documentos é apresentado. **Conclusão:** Embora raro os riscos de cegueira iatrogênica que segue procedimentos estéticos faciais secundários existem. O médico deve estar alerta para seus riscos e tomar todas as providências para evitá-los.

Descritores: cegueira, iatrogenia, procedimentos cirúrgicos menores, revisão.

ABSTRACT

Blindness as a complication of a minor operative procedure for a minor disease is a shocking and dreadful experience for both patient and doctor. The present paper reports most procedures which may produce such complication as well as its probable physiopathology and guidelines for the prevention and treatment of this catastrophic event, based upon literature review. Blindness as a result of injections of various substances in the head and neck are appearing with alarming frequency in the literature. These substances range from paraffin, to alcohol, anesthetic agents, angiographic contrast dye, procaine penicillin, and corticosteroids, as well as multiple drug combinations. These drugs have in common either a neurotoxic or embolic potential. In some instances, the site of injection in the head and neck may be quite remote from the ophthalmic artery and yet produce amaurosis, as with scalp injections and intranasal injections. Any material injected in the face, nose or mouth and even the upper neck represents a potential embolus to the eye or brain via the mechanism of retrograde arterial flow. **Objective:** To present a review of the evidence related to the events leading to iatrogenic blindness linked to facial surgical manipulation in minor procedures. **Material and Method:** Papers from 1906 till 2006, gathered from the main electronic libraries were collected. A total of 58 papers are presented. **Results: Conclusion:** Although rare the risks of iatrogenic blindness following minor facial procedures exist, it is real and even a very small percentual of blindness occurring they are certainly not negligible. If it is a occurrence from minor procedures for minor problems, not involving life preservation or the relief of chronic excruciating pain, it may be devastating for the patients and their relatives, and certainly catastrophic for the self-esteem and future of the practitioner himself.

Keywords: blindness, iatrogenic, minor surgical procedures, review.

INTRODUCTION

Blindness as a complication of a minor operative procedure for a minor disease is a shocking and dreadful experience for both patient and doctor.

The present paper reports most procedures which may produce such complication as well as its probable physiopathology and guidelines for the prevention and treatment of this catastrophic event, based upon literature review. Blindness as a result of injections of various substances in the head and neck are appearing with alarming frequency in the literature. These substances range from paraffin, to alcohol, anesthetic agents, angiographic contrast dye, procaine penicillin, and corticosteroids, as well as multiple drug combinations. These drugs have in common either a neurotoxic or embolic potential. In some instances, the site of injection in the head and neck may be quite remote from the ophthalmic artery and yet produce amaurosis, as with scalp injections and intranasal injections. Any material injected in the face, nose or mouth and even the upper neck represents a potential embolus to the eye or brain via the mechanism of retrograde arterial flow.

OBJECTIVE

To present a review of the evidence related to the events leading to iatrogenic blindness linked to facial surgical manipulation in minor procedures.

MATERIAL AND METHOD

Papers from 1906 till 2006, gathered from the main electronic libraries were collected. A total of 58 papers are presented.

REVIEW

Although extremely rare, cases of total or partial defects of the visual field have been reported after septal surgery on local infiltration anesthesia or corticosteroid injection in the mucosa of the septum or the inferior turbinate¹.

The first reported case of visual complications associated with intranasal corticosteroid injection was that of Hager², who, in 1962, described unilateral permanent blindness in a patient after an intranasal injection of hydrocortisone acetate. However, for Mabry³, "on the basis of reported clinical experience, the likelihood of visual complications following intranasal corticosteroid injection is quite small (six of 99.500 reported injections, or 0.006 %)." According to Baker⁴ "The risks for visual complications with intranasal steroid injections are minimal and are far outweighed by the therapeutic benefits. When the steroid is injected correctly intramucosally into the anterior tip of the inferior turbinate, the results have been gratifying and complications negligible". Shafir et al.⁵ described a case of permanent visual loss at the time of a long-acting steroid to the nasal dorsum for treatment of post rhinoplasty scarring. Ellis⁶ reported one case of partial but

permanent loss of visual acuity involving both eyes of a child who underwent tonsillectomy and injection of a concentrate suspension of penicillin, lidocaine, and methylprednisolone on both tonsillar fossae, in order to eliminate post tonsillectomy pain and infection. Transient or permanent loss of vision have been reported following local and nerve-blocking injections of anesthetic agents, such as procaine hydrochloride and mepivacaine hydrochloride in dental, oral, nasal and orbital procedures^{7,8}.

Makham⁹ reports sudden loss of vision following alcohol block of the infraorbital nerve. A case of paraffin injection into the nose followed immediately by blindness due to embolism of the central artery of the retina was reported by Hurd & Holden (1903) and a sudden unilateral visual loss after autologous fat injection into the glabellar area was described by Dreizen & Framm¹⁰. Temporary blindness and ophthalmoplegia from nasal packing was registered by Giammanco & Binns¹¹.

Savino et al¹² described four patients with retinal occlusion and optic nerve ischemia following nasal surgery. None of these patients had injections of particulate-containing solutions, and all appear to have suffered permanent visual defects secondary to vasospasm. Rettinger & Christ¹³ described a similar case stating that "The medical expertise concluded that the occlusion of the central retinal artery was not influenced by the pressure and the injected substance alone. A malpractice by the surgeon was denied. For legal purposes it is of importance that malpractice from intranasal injection is rarely evident. So expertises have to focus on the reactions of the medical staff following the onset of symptoms." Cheney & Blair (1987)¹⁴ presented a case of retinal artery ischemia following open rhinoplasty in a patient where no turbinate injection or manipulation was performed. There was no evidence of retinal emboli and CT scan showed no evidence of orbital violation., trauma or retrobulbar hematoma. A diagnosis of retinal artery occlusion and/or spasm was made. Kambic et al (1968)¹⁵ described a case presenting a vascular accident occluding the retinal central artery, during an anesthetic transnasal injection with Lidocaine 2% for corrective surgery of the nasal bone.

Cooley & Cottingham¹⁶ contend that eye complications after dental anesthesia are not rare. While some authors^{17,18} offer reassurance that recover will take place as soon as the anesthetic effect has dissipated, there have been cases of permanent eye damage from local anesthetics with or without epinephrine^{1,16}. There appears to be little question that vasospasm in itself can produce retinal damage, as for example when epinephrine alone or in combination with a local anesthetic have been responsible for blindness.

One of the complications of retrobulbar hemorrhage is the closure of central retinal artery. This event has been observed resulting from closed head injury, from ocular contusion, and as a complication of blepharoplasty, as well as retrobulbar injection¹⁹.

Although retrobulbar hemorrhage can follow the administration of retrobulbar anesthesia, it does not occur frequently.

Needless to say, the complication of central retinal artery occlusion following attempted retrobulbar anesthesia force us to regard this oft-performed procedure with added respect and caution. Ord (1981)²⁰ reported four cases of retrobulbar hemorrhage and blindness: two following malar elevation, one following malar elevation and orbital floor repair and one following malar and infraorbital floor bone grafting..

Blindness or decreased vision associated with cosmetic blepharoplasty is a rare occurrence. The term cosmetic blepharoplasty is a misnomer when the orbital septum is penetrated by a needle or is incised. The surgeon has entered the orbit and is doing an orbitotomy in addition to blepharoplasty. Traction by the surgeon on anterior orbital fat that has prolapsed into the eyelid may avulse the posterior ciliary blood vessels or even the central retinal artery, resulting in loss of vision²¹. About 50 cases of blindness after blepharoplasty have been reported and it is probable that more have occurred but not have been reported²².

“Practitioners in a number of medical and dental specialties have witnessed the frightening event of sudden visual loss following routine injections in these areas. While loss is often temporary, permanent visual loss has also occurred. Most of these injections have occurred during routine procedures in which such a serious complication is totally unforeseeing. A variety of materials have been responsible”²³.

An episode of blindness was reported by Von Bahr²⁴, in a patient who received subcutaneous intrascleral injections of hydrocortisone into the temporal and frontal-parietal region for the treatment of alopecia areata. Three similar cases were reported by Baran²⁵, in 1964, who explained these events on the basis of anastomoses between the superficial temporal artery and the branches of the ophthalmic artery.

DISCUSSION

Physiopathology

Selmanowitz & Orentreich²⁶ discussing the hazards of blindness from cutaneous corticosteroid injection, agreed that retrograde flow produced by forceful injection in the peripheral extraorbital branches of the ophthalmic artery to the face and scalp had probably been responsible for ocular accidents following injections in the scalp and nose. The authors cautioned against the use of large particle steroids, preparations which have been allow to stand, and steroids mixed with local anesthetics. They felt that further safety could be obtained by diluting the steroid with saline and by using a multiple puncture technique rather than the “fanning” technique.

Rowe²⁷ described the injection of 0,6 to 0.8 cc of triamcinolone suspension into the inferior turbinates and anterior septum of a patient, which was followed by loss of vision in the left eye, diplopia, and headache. Diplopia as well as the visual abnormalities cleared without specific therapy. The authors postulated a vasoconstrictive or an embolic phenomenon as a cause for these events.

Byers²⁸ reported two cases of inadvertent intra-arterial injection into the inferior turbinates for treatment of allergic rhinitis resulted in retrograde flow of methylprednisolone acetate in the ophthalmic artery. Both patients demonstrated embolization of the retinal circulation, and one of the choroidal circulation as well. Visual outcome was favorable, although one patient showed a permanent paracentral schotoma. Fundus photographs clearly showed intra-arterial obstruction of arterioles that cleared within several days.

McCleve & Goldstein²³ theorize that the mechanism of visual disturbance from corticosteroid injections of the turbinates is due to intravascular injection with a retrograde flow into the retinal artery and its branches. While it would logically seem that material injected into any artery would flow almost entirely in the normal direction of blood flow, one must keep in mind the steadily increasing resistance peripherally as an artery branches and re-branches into ever smaller units.

A rapidly injected bolus probably finds less resistance centrally due to the elasticity of the central arterial system. It would logically seem that arterial pressure would alert the practitioner that the needle had penetrated into the arterial circulation, and that it would be difficult to inject against pressure. Some authors affirm this is a danger sign for head and neck injections. But experience indicates otherwise. Physicians who routinely work with arterial lines find that arterial pressure is overcome surprisingly easily with a syringe. Most authors are now in agreement that the retrograde flow is most often responsible for the complication. Once this concept is accepted, the arterial avenues to the ophthalmic artery are clear and apparent. Multiple branches of the ophthalmic artery project outside the orbit into the nose and onto the forehead, external nose and face. These branches are particularly vulnerable to retrograde embolus, requiring only a small volume of material to reach the main ophthalmic artery. Moreover, these branches anastomoses freely with many branches of the external carotid artery, making injections as far away as the lower face, mouth, pharynx and possibly even the upper neck potentially hazardous. Anastomoses between external carotid branches and ophthalmic artery increases risk for injections. These anastomoses may actually provide an avenue for collateral circulation to the brain through the orbit when the internal carotid is narrowed.

Singh & Dass²⁹ report that in some patients the main arterial blood supply to the orbit is via the middle meningeal artery with a small underdeveloped ophthalmic artery trunk. In these situations, and possibly others, the external carotid circulation may flow into the orbit and ophthalmic artery than the reverse. Under these conditions the eye is obviously in greater risk as direct flow, rather than retrograde, could carry the embolic material to the ophthalmic artery. Might there have been some instances of retrograde injection reaching the internal carotid itself via branches of the external carotid? There are strong suggestions that this has happened, particularly in those cases in which bilateral blindness has occurred or when sensorium changes or other symptoms suggest that the material reached the brain.

Injections in the lower face or inside the mouth which have reached the eye may have done so via this route. An additional hazard exists with the not uncommon anomaly in which arteries such as the ascending pharyngeal arise directly from the internal carotid rather than from the external carotid²³.

For Castillo³⁰ five branches of the ophthalmic artery are particularly important for embolization: (1) the supratrochlear artery (a. frontalis) that supplies the integument, muscles and pericranium of the forehead, anastomosing with the supraorbital artery; (2) the supraorbital artery, which supplies the integument, the muscles and pericranium of the forehead, anastomosing with the supratrochlear and the frontal branch of the superficial temporal artery; (3) the dorsal nasal artery that anastomoses with the angular artery, the artery of the opposite side, and the lateral nasal branch of the facial artery; (4) the anterior and posterior ethmoidal arteries that are an important arterial supply for the nasal fossae probably being the main route for embolization from intratubular injections. Blindness due to retrobulbar haemorrhage has been reported in a number of situations: following ocular trauma³¹; following malar fracture reduction³², following orbital floor repair; and following orbital surgery.

Blepharoplasty procedures contribute most of the reported cases^{33,34}. Reported cases following malar reduction and orbital floor repair are rare and no cases following reduction of maxillary fractures have been reported²⁴.

"Retrobulbar injection of various drugs has been used for nearly eight decades. It is a commonly employed method for inducing ophthalmoplegia and anesthesia prior to intraocular surgery. Previously described complications include retrobulbar hemorrhage, perforation of the globe, optic atrophy, and central retinal artery obstruction³⁴.

Sullivan et al.³⁵ reported three cases that demonstrate combined obstruction of the central retinal artery and central retinal vein associated with intraneural sheath hemorrhage after retrobulbar injection."

Unilateral amaurosis has also followed carotid, aortic, and vertebral angiography, often in account of atheromatous emboli dislodged by the needle or catheter³⁶.

Transient cortical blindness is an uncommon but well known complication following cerebral angiography but has to be differentiated from embolic complications. One possible cause of this complication is an adverse reaction to contrast agent, resulting in an osmotic disruption of the blood-brain barrier that seems to be selective for the occipital cortex, plus direct neurotoxicity of the contrast agent itself³⁷.

For Heinze & Hueston²² the following appear to be the common features in the chain of events leading to the rare instances of blindness after blepharoplasty: 1 – retrobulbar hemorrhage with patient gross proptosis and echimosis within hours after the surgery; (2) raised intraorbital and intraocular tensions; (3) ocular fundoscopic findings ranging from pallor of the optic disc to collapse of the central retinal artery; (4) rapid onset of blindness, which is reversible if treated early.

Late or inadequate treatment may not prevent permanent

blindness. The initiating event is intraorbital hemorrhage, which may occur up to two or more hours after surgery. Beneath any firm bandage (or beneath lids which became rigid from postoperative edema) the orbit is converted into a "closed box", and continuing intraorbital hemorrhage combined with reactive edema can produce a rapid rise of intraorbital pressure. Gross proptosis ensues, contributing further to rigidity of the lids and orbital septa. Intraocular pressure now rises secondary to the increase in intraorbital pressure. As intraocular pressure reaches pathological levels, the blood circulation in the optic disc and retina is compromised and blindness may ensue.

In his extensive studies of anterior ischemic optic neuropathy, Hayreh^{38,39,40} has shown that the circulation in the intraocular blood vessels is dependent on the balance between the perfusion pressures in the posterior ciliary and central retinal arteries and the intraocular pressure. When the intraocular pressure rises, the vessels in the pre laminar part of the optic disc are the first to be obliterated, followed by those in the peripapillary choroid and retrolaminar optic nerve. Next in susceptibility are the vessels of the choroid proper and finally the central retinal artery will close. A fall of perfusion pressure in the posterior ciliary or central retinal arteries could produce the same sequelae. It is thus perfectly feasible for the intraocular pressure to rise just enough to compromise the circulation of the optic disc without affecting the central retinal circulation. If this process is acute, an infarction of the optic nerve head will ensue, with signs confined to pallor of the disc during the crisis and variable edema of the optic nerve head about the second or third day. Eventually, optic atrophy occurs. The central retinal vessels may appear normal throughout.

Obstruction of the central retinal vein alone did not produce loss of vision. Central retinal artery occlusion as an additional component was necessary before visual defect occurred.

Unilateral blindness and total ophthalmoplegia developed on the same side on which transantral ligation of the internal maxillary artery was performed for persistent posterior epistaxis. Evaluation with arteriograms and phlebograms showed no compromise of retinal vessels or cavernous sinus thrombosis but marked swelling of previously placed oxidized cellulose gauze was found. The possibility that the use of oxidized cellulose gauze was a cause of the complication was investigated in an animal experiment. Oxidized cellulose is a useful hemostatic agent, but its use in a closed, bony walled space can result in dangerous pressure from swelling of the oxidized cellulose.

By a mass effect, the swollen cellulose can then damage nearby structures. This mechanism is a likely explanation for the resulting blindness and total ophthalmoplegia reported in this paper⁴¹.

According to Cheney and Blair¹⁴, "blindness following elective cosmetic surgery can be a devastating complication, for the patient as well as the surgeon. A case of unilateral blindness secondary to central retinal artery occlusion following an

open septorhinoplasty is presented. The case appears to be the result of vascular disturbance leading to blindness. There was no evidence of retinal emboli. A diagnosis of retinal artery occlusion and/or spasm was made.”

Kronman & Kabani²⁰ reported a case of diplopia secondary to maxillary infiltration anesthesia. They think that the ocular changes observed after dental local anesthesia are related to sympathetic and parasympathetic effects, rather than to anesthetic solution circulating in the vascular tree. In any event, it is of extreme importance for the clinician to understand that the effects are transitory and are unavoidable. When such cases do occur, the patient should and must be reassured, comforted, and made to understand that the symptoms will generally disappear in an hour or less. However, there have been cases of permanent eye damage from local anesthetics with or without epinephrine^{1,16,20}. There appears to be little question that vasospasm itself can produce retinal damage, as for example when epinephrine alone or in combination with a local anesthetic have been responsible for blindness.

Magargal et al (1985)⁴² described a man in whom a branch retinal artery occlusion secondary to the chronic abuse of 0.5 % oxymetazoline hydrochloride long-acting spray developed. In the patient described by them, platelet emboli were reported in the retinal vessels, and they demonstrated enhanced platelet aggregation of their patient’s platelets when exposed to epinephrine in vitro. Such a hypersensitivity of coagulation may play a role in some instances of complications associated with submucosal injections.

From the analysis of the literature it seems clear that the basic mechanisms for the catastrophic complication of visual loss after the procedures generally considered routine and harmless are as below¹⁵: 1 – Direct or indirect traumatic injury to the optic nerve by surgical instruments, bone fragments or compression within the canal, due to increased intraorbital pressure from ruptured vessels.

2 – Direct or indirect damage to the intraorbital vascular system by vessel injury or compression and occlusion of the arterial system by spasm, embolism or thrombosis

Independent of the effects of the injected material the prerequisite is a connection between the side of injection and the ophthalmic artery. Three different ways are to be considered: (1) The substance reaches the ophthalmic artery via the venous system, but this possibility can be neglected because a corpuscular substance is filtered by the peripheral or pulmonary capillary system; (2) after intra-arterial injection into vessels of the external carotid artery system the internal carotid artery is reached via vessel anomalies; the ophthalmic artery can descend from the middle meningeal artery (external carotid artery system), a condition that is not rare and sometimes responsible for blindness following vidian neurectomy; (3) the substance reaches the ophthalmic artery via regular connections between the internal and external carotid artery system, and such “bridging arteries” are the supratrochlear, frontal and nasal dorsal arteries (within the facial skin) as well as the anterior, posterior and additional

ethmoidal artery (within the nasal mucosa).

The routes presume a reversion of the flow-direction induced by the injection pressure after unperceived arterial puncture. The direction of flow depends on small pressure gradients that are easily modified by intra-arterial injections. After invasion of the ophthalmic artery the substance can evoke thrombosis, embolism or vasospasm.

As McCleve & Goldstein²⁴ state “Any material injected in the face, nose or mouth and even the upper neck represents a potential embolus to the eye or brain via the mechanism of retrograde arterial flow.”

PREVENTION

For preventing ocular complications due to turbinate corticosteroid injections, McCleve & Goldstein²³ proposed the following guidelines: (1) The injection should be made very slowly, with minimal pressure, so that if the tip of the needle is intravascular, not enough pressure will be generated to push the solution retrograde in the artery; (2) the top of the needle should be moved during the course of the injection, depositing the medication at several points along a line rather than injecting a single bolus; (3) an attempt should be made to produce some ballooning or blanching of the mucosa, indicating that the injection is taking place extravascularly; (4) topical vasoconstrictor should be used to diminish the size and flow of the submucosal vessels; (5) a small diameter needle should be used for the injection, eg. 25 gauge 1 ½ inch; (6) aspiration prior to injection is advocated. Mabry⁴ advocates practically the same measures emphasizing that the use of topical cocaine prior to injection is important for topical anesthesia and for local vasoconstriction of the extremely vascular tissue. It is postulated that this will reduce the likelihood of inadvertent intravascular injection causing embolization of the retinal circulation. For the same reason the repository corticosteroid injected should contain the smallest possible particle size. Materials meeting this criterion are triamcinolone acetonide and prednisolone tertiary-butylacetate, all of which have an average particle size of less than 10 micra.

For head and neck injections McCleve & Goldstein²³ encourage the same precautions taken for intratubal injections, with only slight variation.

Summing up: (1) inject slowly with minimal force; (2) inject in small increments preferably moving the needle slightly between injections (The rationale here is that even if the needle happens to be intra-arterial, a smaller volume of material might not reach a critical artery); (3) aspirate prior to injection; (4) injections of viscous, particulate, or toxic materials and injections close to the orbit demand extreme caution and extra care.

According to Rettinger & Christ¹³ the general rules for intranasal injections are: 1 – The amount of 5cc, max 10 cc of solution should not be exceeded. The injection should be restricted to regions of planned incisions, multiple punctures

and infiltration of wider areas should be avoided. The dissection of the perichondrium layer from the septal cartilage by infiltration is not possible as is supposed very often. One has to be conscious that a negative blood aspiration (in the syringe) after puncture of the mucosa does not exclude an intra-arterial position of the needle tip, as the small artery may collapse. 2 – Because of the vascularity, injections into the turbinates should be avoided. Complications may also occur even when solutions are used that do not induce thrombosis or vasoconstriction. It is very likely that in most of these cases, small air bubbles are the only reason for occlusion. During funduscopy they are invisible and therefore not described in the literature. The total freeing of the contents of the syringe from air must be stressed particularly. The solution to be injected, in its marketed bottle and later in the syringe (just prior to injection), should be agitated thoroughly for re suspension of particles because sedimentation and agglomeration of particles may occur when the suspension is allowed to stand. Since local anesthetics solutions may induce vasospasm, it is generally advisable that they not be used as diluents for steroid suspensions.

Selmanowitz & Orentreich²⁶ on the other hand, say “strong electrostatic forces may occur, tending to clump particles when these drugs are combined with other drugs, such as lidocaine.”

TREATMENT

Although many ophthalmologists have advocated conservative treatment of retrobulbar haemorrhage, active treatment is called for in the presence of increasing proptosis, decreasing visual acuity or blindness. With complete retinal occlusion blindness may be irreversible after one or two hours. Treatment can be either surgical or medical or a combination of both. Decompression is the essential surgical measure²⁵.

This may be carried out through existing infraorbital or blepharoplasty incisions when present. A lateral canthotomy have also been used. Alternatively, decompression may be carried out transantrally via a Caldwell-Luc approach. Care should be taken not to push spicules of bone from the orbital floor into the orbit. Optic nerve damage by bony spicules causing blindness has been reported, following antral packing and balloon catheters. Medical measures may be used as an alternative treatment to decompression. Should medical measures fail to improve proptosis and visual acuity rapidly decrease, then surgical decompression must not be delayed.

If decompression and medical measures fail to restore vision in ten minutes, and disc is ischemic with the central retinal artery occluded, then further treatment is necessary. A simple treatment for central retinal artery occlusion is massage the eye and sudden release of pressure. If this fails to relieve the occlusion, then anterior chamber paracentesis should be undertaken by an experienced ophthalmologist. As early diagnosis is the most important factor in reversing blindness,

nursing staff should be aware of the importance of reporting orbital pain, swelling or visual loss immediately to the surgeon. It should be remembered that the appearance of the normal retina and blood vessels can be deceptive. Visualization of the disc is essential.

Although retrobulbar haemorrhage is rare and the mechanism of visual loss is still not completely understood, early diagnosis and decompression should prevent the serious complication of permanent blindness²³. Anterior chamber paracentesis must be regarded as an emergency measure with a potential for complications²⁷.

“Intraorbital hemorrhage as a complication of ethmoid surgery, blepharoplasty, and trauma occasionally results in blindness. Present methods of treatment directed at reducing intraocular pressure, such as acetazolamide, lateral canthotomy, and anterior chamber paracentesis, generally yield poor results. Orbital decompression can result in complete recovery of vision, after total blindness⁴³. The last important consideration deals with treatment of central retinal artery embolism once it has been established. The medical ophthalmic literature is replete with multiple, sometimes controversial forms of treatment. However, it is important to remember some facts. After embolization, functional deficit-and-dead occur before anatomic deficit-and-death and, while the former may be a reversible change, the later is not. Thus, one should not have a hopeless and helpless action in cases of central artery embolization, even in the presence of marked loss of vision. Every effort should be made to treat them. Retinal blood flow may be increased by reducing intraocular pressure, and intravenous injection of acetazolamide has received universal approval in this respect. This is an effective form of therapy and, if it is combined with ocular massage, the intraocular pressure can be reduced rapidly to about 5mmHg and maintained at this level. Because a rapid fall in intraocular pressure when ocular massage and intravenous acetazolamide are combined, firm massage with compression of the globe for up 15 seconds at a time, followed by a sudden release, would seem (at this time) to be a very effective method of improving retinal blood flow. It is simple to perform and can be started as soon as the diagnosis is suspected. It also has the advantage of mechanically aiding in the disintegration or is lodging of emboli into the more peripheral parts of circulation. Mannitol profoundly lowers intraocular pressure when given intravenously³⁵.”

CONCLUSION

Although rare the risks of iatrogenic blindness following minor facial procedures exists, it is real and even a very small percentage of blindness occurring they are certainly not negligible. If it is an occurrence from minor procedures for minor problems, not involving life preservation or the relief of chronic excruciating pain, it may be devastating for the

patients and their relatives, and certainly catastrophic for the self-esteem and future of the practitioner himself.

Therefore, the practitioner must be aware of the endangering vision procedures for the pertinent prevention and adequate measures for early diagnosis and prompt and adequate treatment of such a complication. "For legal purposes it is

of importance that malpractice from intranasal injection, for instance, is rarely evident. So, expertises have to focus on the reactions of the medical staff following the onset of symptoms. Early diagnosis and immediate and adequate treatment of the problem are essential for saving the patient eye and to face up to occasional malpractice suits.

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