INJECTION PROCEDURES ENCOUNTERED IN OPTOMETRY

by

Brian D. Dornbos
and
Courtney D. Moliterno

This paper is submitted in partial fulfillment of the requirements for the degree of

Doctor of Optometry

Ferris State University
Michigan College of Optometry

May, 2012
INJECTION PROCEDURES ENCOUNTERED IN OPTOMETRY

by

Brian D. Dombos
and
Courtney D. Moliterno

Has been approved
May, 2012
We, Brain Dornbos and Courtney Moliterno, hereby release this paper as described above to Ferris State University with the understanding that it will be accessible to the general public. This release is required under the provisions of the Federal Privacy Act.
Abstract

At present, twenty-three states permit doctors of optometry to perform epinephrine injections for anaphylaxis, and only eight states permit the use of injections for management of conditions other than anaphylaxis. The National Board of Examiners in Optometry (NBEO) now requires that all students applying for a doctor of optometry license be competent in and capable of performing basic injection procedures, including preparation of medications, intravenous fluorescein angiography injections, and intramuscular epinephrine injections. The purpose of this guide is to provide a practical step-by-step outline of common injection procedures to optometry students preparing for the injections skill portion of the NBEO Clinical Skills Examination (Part III), as well as to review common clinical conditions requiring the use of injections for either treatment or diagnostic purposes. This guide reviews the proper techniques required to perform subcutaneous, intradermal, intramuscular, and intravenous injections, as well as aseptic techniques to prepare for an injection procedure. The guide will be supplemented by an injections video, which was recorded in the fall of 2010. Like any other procedure utilized in the practice of optometry, these injection techniques require practice and repetition to become proficient.
TABLE OF CONTENTS

INTRAVENOUS ................................................................. 1
INTRAMUSCULAR ............................................................ 4
SUBCUTANEOUS .............................................................. 7
INTRADERMAL ................................................................. 9
INTRAVITREAL ............................................................... 11
SUB-TENON ................................................................. 14
INTRAVENTOUS INJECTION

Clinical Uses
Intravenous (IV) injection consists of injecting a pharmaceutical agent directly into the bloodstream. This allows almost immediate systemic absorption and transportation from the site of injection to the rest of the body.

There are three primary uses of IV injection in the ophthalmic setting: fluorescein angiography (or indocyanine green angiography) for detection of retinal or sub-retinal neovascularization, Visudyne injection with photodynamic therapy (PDT) for treatment of leaking retinal vessels, and the administration of systemic antibiotics or antifungals. Because the primary use of IV injection in the ophthalmic setting is performing a fluorescein angiogram to evaluate the retinal vasculature, the following procedural component will focus on this technique.

Clinical Note: IV Injection and Eye Disease
When certain systemic conditions present in the ophthalmic setting, patients may need to be hospitalized for intravenous injection of steroids. Two such diseases are giant cell arteritis and optic neuritis. Rarely IV injection of a 1:1,000 concentration of epinephrine to treat anaphylaxis or IV edrophonium to test for myasthenia gravis is required in the clinical setting. See the clinical note at the end of the section for more information on giant cell arteritis and optic neuritis.

Procedure
For this procedure, the following items are needed:
- Protective gloves
- Alcohol swabs or Betadine
- Cotton gauze or balls
- Tape, precut to 3-4"
- Butterfly needle with plastic tubing (The plastic cap should be loosened slightly.)
- Syringe, pre-loaded with sodium fluorescein
- Tourniquet
- Emesis container

The primary site of injection for fluorescein angiography is the antecubital vein, on the inside of the elbow. If a suitable vein cannot be located, a vein on the back of the hand can be used. Instruct the patient to relax. Don protective gloves, then select and palpate a vein. Apply the tourniquet four to five inches above the site of injection. To properly apply a tourniquet, have the patient’s arm outstretched on the table, with the tourniquet lying on the table. Stretch the tourniquet out, form a cross with the tourniquet, and then tuck a small portion underneath the crossed section, forming a loop; this allows for easy removal. (Please see the...
"Proper Tourniquet Application" section at the end of the article). Clean and disinfect the area. To promote vasodilation, gently tap the vein, or have the patient open and close his or her fist.

Apply counter-tension to the skin with the non-dominant hand. Pinch the wings of the butterfly needle together, and pierce the skin with the bevel facing upward and the needle at 30-45°. Steadily continue to push into the vein and move the needle parallel to the forearm. A small amount of blood should slowly enter the tubing. Tape the plastic wings to the patient’s arm, and with the syringe containing sodium fluorescein in hand, remove the plastic cap of the tubing and allow air to completely leave the IV system to avoid injecting an air embolus. Quickly attach the syringe to the tubing as blood reaches the end of the tubing. The patient must now be positioned for photography.

Inject a small amount of blood/medication, and evaluate the tissue for extravasation of fluorescein. Extravasation represents leakage of fluorescein into the tissue around the site of injection. This can be painful for the patient and increases the risk of adverse reaction and complication. If present, abort the procedure and immediately apply a cold compress to the affected area (2). Over a course of five seconds, steadily inject the rest of the fluorescein. (For the NBEO examination, the injection set must now be removed and properly disposed of.) The photography session can progress with the IV set still in the patient’s arm.(1,3)

Gently remove the infusion set and place the cotton gauze or cotton ball over the injection site and apply tape. Discard the needle and infusion set properly.(1,3)

Complications of Fluorescein Angiography
A study published in 2007 reviewed the complications associated with fluorescein angiography. Of the 1,500 patients to receive a fluorescein angiogram, 9.72% experienced some form of adverse reaction. The most commonly encountered adverse reactions were nausea, vomiting, and urticaria. Bronchospasm and laryngeal edema were very rare complications (<.04%). The exact mechanism for adverse reaction to fluorescein is not clear, but a histamine-mediated response and anxiety about the procedure may induce an adverse reaction. Other studies have shown similar reactions. Additional information on allergic and anaphylactic reactions is included following the intramuscular injection section.(4)

If the patient undergoes a severe allergic reaction, immediate intervention is necessary. Intravenous or intramuscular epinephrine must be administered. Oxygen and airway manipulation may be necessary. A history of a mild allergy to fluorescein is not an absolute contraindication to fluorescein angiography. These patients should be given oral diphenhydramine (Benadryl) at least 30 minutes prior to injection to minimize the risk of a severe allergic response.(2)
Overall, fluorescein angiography is a safe procedure. As with all medical procedures requiring the use of pharmaceuticals, a thorough history of any drug or medical allergies must be taken.

**Proper Tourniquet Application**

![Tourniquet Application Diagram](image)

Stretch the tourniquet out so that the rubber is taught. Cross the ends, one over the other and form an X. Form a small loop with a section of the tourniquet nearest the X and tuck it under the taught band to facilitate easy removal. The final result is imaged below.

---

**Clinical Note: Giant Cell Arteritis and Optic Neuritis**

**Giant cell arteritis**: Giant cell arteritis (GCA), or temporal arteritis, is the most common cause of arteritic anterior ischemic optic neuropathy (AAION). In GCA, the posterior ciliary artery is occluded, which prevents adequate perfusion of the anterior optic nerve head. The disc becomes swollen and pale, often with flame-shaped hemorrhages near the disc margin. This condition presents as sudden, unilateral, painless vision loss. Most patients are over the age of fifty-five and many experience jaw claudication and/or scalp tenderness in the temple region.

If GCA is confirmed by elevated erythrocyte sedimentation and C-reactive protein rates, systemic steroids are administered immediately. Methylprednisolone (250 mg) is administered intravenously every six hours for twelve doses; then, oral prednisone (80 to 100 mg) is taken daily. Treatment should be continued for at least six to twelve months. If treatment is delayed, the biggest ocular threat is vision loss in the fellow eye; the patient could be blind within twenty-four hours.

Systemic steroids can cause side effects, such as increased blood sugar, increased intraocular pressure, posterior subcapsular cataracts, poor wound healing, and an increased risk of secondary infection.5

**Optic neuritis**: Optic neuritis also presents as sudden, unilateral vision loss. Young, female patients are most commonly affected. Many patients experience pain on eye movement (92%), and an afferent pupillary defect is usually present. Two-thirds of cases of optic neuritis are retrobulbar. There are many causes of optic neuritis, including multiple sclerosis (most common), viral infections or vaccinations (mononucleosis, herpes zoster, measles, chickenpox), and granulomatous inflammations (tuberculosis, sarcoidosis, syphilis); some cases of optic neuritis are idiopathic.

Based on the Optic Neuritis Treatment Trial, the main treatment for the demyelinating form is intravenous methylprednisolone (1 g/day) for three days, followed by oral prednisone (1 mg/kg/day) for eleven days, which is then tapered over four days (20 mg on day 1; 10 mg on days 2 and 4). This treatment regimen has been shown to speed up the visual recovery but does not improve final visual acuity. Without treatment, vision usually returns to near normal within two to three months.5
INTRAMUSCULAR INJECTION

Clinical Uses
Injection of a substance directly into muscle tissue will allow the substance to be rapidly absorbed and distributed, due to the high amount of vascularization present in muscle tissue. Intramuscular (IM) injections have two main uses in the ophthalmic setting: treatment of severe bacterial infection, typically gonococcal conjunctivitis, and treatment of anaphylaxis.(1,6)

Gonococcal conjunctivitis typically has a hyper-acute onset with severe mucopurulent discharge. Treatment must be initiated quickly, as the causative organism, Neisseriae gonorrhea, is one of several microbes that can perforate the cornea. Classic treatment of gonococcal conjunctivitis is a single dose of 1g IM ceftriaxone and appropriate management of associated corneal complications.(5)

Clinical Note: Gonococcal Conjunctivitis
Gonococcal conjunctivitis is one of only a handful of cases of acute red-eye that requires immediate systemic treatment. Gonococcal conjunctivitis presents as a hyperacute red eye with excessive mucopurulent discharge. The majority of patients are sexually active and have a concomitant, often asymptomatic genital infection. The organism responsible, Neisseria gonorrhoeae, is capable of eroding and perforating the cornea within days. Intramuscular ceftriaxone (Rocephin), 1 g, along with continued flushing with sterile saline is the treatment of choice. N. gonorrhoeae rarely causes neonatal conjunctivitis, as prophylactic antibiotics are routinely administered to newborns' eyes during the birthing process.(7)

Procedure
For this procedure, the following items are needed:
- Protective gloves
- Alcohol swabs or Betadine
- Cotton gauze or balls
- Tape, precut to 3-4’’
- Syringe with proper amount of medication to be administered (0.5 to 1.0 mL for deltoid IM injection)
- 22 to 25 gauge, 5/8 inch needle

The four primary sites of injection for intramuscular administration of epinephrine are the deltoid muscle, the dorsogluteal region (upper, outer quadrant of the buttock), the vastus lateralis (anterior, lateral portion of the thigh), and the ventrogluteal region (lateral aspect of the pelvis). A review of literature found that the deltoid muscle site requires the smallest amount of medication to be injected; however, this is the only IM injection site which causes post-injection discomfort to the patient. The discomfort is most likely due to the reduced amount of muscle mass at this site compared to other injection sites. The dorsogluteal site carries a heightened risk of damage to the sciatic nerve and is typically a last resort for IM injection. The vastus lateralis site is easily accessible and carries a low risk of damage to internal structures. The ventrogluteal site, however, has the lowest of all risks for complications following injection.(9)
The needle used for an IM injection depends on the muscle and location of the injection, as well as the amount of subcutaneous fat present over the injection site. Typical needle lengths are between 32 and 38 mm. The amount of medication to be injected also varies with injection site (5). The EpiPen auto-injector is pre-loaded with 0.3 mL epinephrine (0.15 mL for EpiPen Jr) and is made to be injected into the vastus lateralis site (6,8).

The National Board of Examiners in Optometry (NBEO) requires students to be proficient in performing an IM injection of 0.4 mL of epinephrine into the deltoid muscle. The remainder of this section will focus on the injection procedure at this site (3).

As with all injections, the injection site must first be inspected for any signs of inflammation or infection and palpated to further examine the site. Clean and disinfect the injection site in a circular motion, beginning in the center and extending peripherally, and instruct the patient to relax. If a small amount of subcutaneous fatty tissue is present, the clinician must bunch the skin around the injection site with the non-dominant hand. Conversely, if a large amount of subcutaneous fatty tissue is present, the skin should be stretched with the non-dominant hand.

With the needle held firmly in the dominant hand, quickly insert the needle at a 90° angle. Release the skin held with the non-dominant hand and aspirate the syringe slightly to ensure that the end of the needle is not in a blood vessel. If blood is aspirated, the needle tip should be changed and a new injection area should be chosen.

Inject the medication at a smooth and steady pace, then remove the needle quickly, maintaining the 90° orientation to the deltoid. Discard the needle and syringe in a sharps container.

Place the cotton ball or gauze over the injection site and apply tape to secure the bandage (1,3).

**Complications of Intramuscular Injections**

The most common complication of an IM injection is post-injection site tenderness. Other common complications include erythema, pain, bleeding, and abscess formation. Complications may be due in part to improper injection technique, which can lead to injection of medication into the subcutaneous tissue rather than directly into the muscle (9).

Contraindications to IM injection include known hypersensitivity to the medication to be administered, infection and/or inflammation at the injection site, and prominent nerves, bone tissue, or blood vessels at the injection site (1).
Clinical Note: Anaphylaxis

Anaphylaxis is an acute reaction whereby the body responds to an allergen it was previously exposed to. Anaphylaxis begins with an immediate immunoglobulin E antibody response, which in turn triggers mast cells and basophils to release chemical mediators, the most prominent mediator being histamine. Histamine has an array of systemic effects, including bronchospasm, vasodilation and vascular leakage, rapid or weak pulse, and itching. Severe anaphylactic reactions can lead to loss of consciousness, cardiac arrest, and death. More common than complete anaphylaxis are pseudoanaphylactic, or anaphylactoid, reactions, which are less severe.(10)

In primary eye care, the majority of anaphylactic reactions occur after systemic (intravenous) administration of sodium fluorescein for evaluation of the retinal and choroidal vasculature during a fluorescein angiogram. Fortunately, the risk of severe anaphylactic reaction from sodium fluorescein is less than 1%. There have been isolated reports of anaphylactic reactions from other medications that are administered intravenously, such as verteporfin, the pharmaceutical agent used during photodynamic therapy.(4,10) Therapeutics used topically in eye care typically elicit a mild and localized allergic response; however, isolated incidences have been reported of anaphylactoid reactions to eye drops.(11) In any instance, following administration of any medication, by either injection, oral, or topical route, a patient should be monitored. Any patient showing signs of dyspnea, pallor, abnormal pulse, itching, or urticaria requires immediate treatment.(4,10)

Treatment consists of intramuscular injection of a 1:1,000 solution of epinephrine in the limb opposite the exposure site. In cases of respiratory depression, oxygen may need to be administered. Oral antihistamines, such as diphenhydramine (Benadryl), may be appropriate for mild cases of anaphylaxis.(10)
SUBCUTANEOUS/LESIONAL INJECTION

Clinical Uses
Subcutaneous injection is used for eyelid anesthesia prior to procedures such as excision of chalazia, wound repair, suture placement, and blepharoplasty, entropion or ectropion repair. The eyelids are innervated by the supraorbital, infraorbital, and zygomaticotemporal nerves. Lesional injection is a type of subcutaneous injection that is used to treat chalazia; both injections will be described below. (1)

Clinical Note: Chalazia
A chalazion is a painless, well-defined area of inflammation in the subcutaneous layer of the eyelid; the nodule is caused by a clogged meibomian gland. Preliminary treatment involves warm compresses. At times, a topical antibiotic ointment is used (bacitracin or erythromycin two times per day). In other cases, doxycycline (100 mg two times per day) is recommended. If the above treatments do not work adequately, a subcutaneous/intralesional steroid injection can be performed (0.2 to 1.0 mL of triamcinolone 40 mg/mL, mixed 1:1 with 2% lidocaine with epinephrine). Another treatment option is removal of the chalazion via incision and curettage. (5)

For anesthesia, 1% or 2% lidocaine HCl (Xylocaine) is typically used, with or without epinephrine, which helps to decrease bleeding by stimulating vasoconstriction (1). For injection of a chalazion, the steroid of choice is triamcinolone acetonide 40 mg/mL (Kenalog-40). This steroid is preferred over dexamethasone because triamcinolone is a viscous suspension that is better stored at the injection site, making it more effective for the treatment of long-standing chalazia. If administered properly, treatment of a chalazion with triamcinolone is effective in 80% to 90% of cases. (1)

Procedure
For this procedure, the following items are needed:
- Protective gloves
- Alcohol swabs
- 2 X 2 gauze pads or cotton balls
- 1-mL or 3-mL syringe
- 23 to 25 gauge, 1/2 to 5/8 inch needle
  - Needle length is dependent upon the site of injection, the size of the patient, and the amount of subcutaneous fat overlying the muscle.

In the ophthalmologic setting, the primary site of subcutaneous injection is the eyelid. In other fields of study, common injection sites include the abdomen, the lateral and anterior aspects of the upper arm or thigh, the scapular area of the back, and the upper ventrodorsal gluteal area.

To begin the lesional injection procedure, partially recline the patient. To control the blink reflex, put a drop of topical anesthetic in each eye. Anesthetic is rarely used during this procedure. To prepare for injection, pull the skin taut to the temporal side. Insert the needle at a 30-degree angle, 2 to 3 mm on the proximal side of the chalazion. Once the needle is inserted into the subcutaneous tissue, decrease the angle to 10 degrees. Push the needle further until it goes
through both the proximal and distal walls of the chalazion; continue for another 2 to 3 mm past the chalazion. Next, begin to inject the medication as the needle is slowly withdrawn. This will allow the triamcinolone to be administered on both sides of the chalazion as well as within it. During the entire procedure, the skin should be held taut. Generally, only 0.2 mL to 0.8 mL of medication is necessary, with the dosage dependent on the size, hardness, and duration of the chalazion (1).

For subcutaneous injections at a site other than the eyelid, the procedure is slightly different. Instead of holding the skin taut, the skin should be pinched to create a 1-inch fold of fat tissue. The insertion angle depends on the amount of fat at the injection site. The angle can vary between 45 and 90 degrees; the more fat tissue present, the larger the insertion angle. The needle should be inserted with a dart-like motion. Aspiration is not necessary during a subcutaneous injection.

Complications of Subcutaneous Injections
Complications associated with subcutaneous injection are rare. The most common complication of subcutaneous injection occurs with steroid injection. In darkly pigmented individuals, subcutaneous corticosteroids can cause depigmentation of the skin around the injection site. Other possible complications include infection and nerve damage. Needle breakage in the skin is also possible at certain injection sites (12). Subcutaneous injections involving the eyelid are very safe, with the main complication being ecchymoses, or small subcutaneous bleeds along the path of the needle (1). These bleeds usually clear without intervention within a week.
INTRADERMAL INJECTION

Clinical Uses
Intradermal injection is used to deliver medication into the dermis layer of the skin. In the ophthalmic setting, intradermal injection is not commonly used. There are two main skin tests an optometrist or ophthalmologist may order which require intradermal administration: the purified protein derivative (PPD) test for tuberculosis and the histoplasmin skin test for histoplasmosis (13). See the clinical note at the end of the section for more information on the PPD and histoplasmin skin tests.

Procedure
For this procedure, the following items are needed:
- Protective gloves
- Alcohol swabs
- 2 X 2 gauze pads or cotton balls
- 1-mL syringe
- 25 to 27 gauge, 3/8 to 5/8 inch needle

The primary site of injection for the above-mentioned skin tests is the forearm, 2 to 4 inches below the elbow with the arm in the supinated position. Before the procedure, wash hands and put on gloves. Clean the injection site with an alcohol swab, starting in the center and continuing outward in a circular motion. Allow the area to dry completely before proceeding.

For the tuberculin skin test, the syringe must be filled immediately before administration (14). Follow the proper steps for drawing medication from a vial. Prior to injection, there should be 0.1mL of solution in the syringe.

Next, stretch the skin taut over the injection site with the thumb and index finger of the non-dominant hand. With the dominant hand, hold the syringe between the thumb and index finger, making sure the bevel of the needle is facing upward. Keep the syringe nearly parallel to the forearm, and slowly insert the needle at a 5 to 15 degree angle just below the surface of the skin (14). The bevel of the needle should be visible through the skin. To get the proper depth, it helps to have the tip of the thumb and index finger that are holding the syringe to just barely touch the forearm. Do not place the index finger between the forearm and the syringe; this will create an insertion angle greater than 15 degrees. After the bevel is completely inserted, release the tension on the skin. Continue to hold the syringe with the dominant hand, while slowly administering the medication with the non-dominant hand.
Continue to inject the solution until a 5 to 6 mm wheal is formed. If a wheal is not created, perform the procedure at least 2 inches from the original site or on the other arm.

After removing the needle, discard the syringe in a sharps container. Do not massage or press on the area after injection. If any bleeding occurs, lightly dab the area with a gauze pad or cotton ball. Do not place an adhesive bandage over the injection site, as it may cause irritation.

**Complications of Intradermal Injection**

Mild itching, swelling, and irritation are common with intradermal injection; this may last for up to 1 week. Patients should not scratch the site, as this could lead to infections. Patients should also avoid using lotions and adhesive bandages to prevent irritation of the injection site.\(^{(14)}\)

---

**Clinical Note: PPD and Tuberculosis**

Tuberculosis is caused by *Mycobacterium tuberculosis*. The main characteristic of this chronic infection is necrotizing granulomas in the lungs. When the eye is involved, a recurrent, granulomatous anterior uveitis is often the presentation. The resultant anterior uveitis may include keratic precipitates, posterior synechiae, and iris granulomas. A retinal vasculitis may also occur. Ocular tuberculosis is usually presumed as the infection in the eye when there is a concurrent systemic tuberculosis infection is present.\(^{(15)}\)

Systemic infection is confirmed with a positive PPD test, also known as the Mantoux test. The test results are dependent upon certain characteristics of the person being tested. In patients with no known risk for tuberculosis, an area of firm swelling greater than or equal to 15mm in diameter indicates a positive test result. Reactions 10mm or larger is a positive reaction in people with an increased risk of injection, such as health care workers, injection drug users, people with diabetes or kidney failure, and people living in prisons, nursing homes, or homeless shelters. A small reaction, considered to be 5mm or greater, is positive in those with a suppressed immune system, organ transplant recipients, and people who are HIV positive.\(^{(16)}\)

---

**Clinical Note: Histoplasmin Skin Test**

The Histoplasmin skin test is not used often to diagnose ocular histoplasmosis syndrome (OHS) for two main reasons:

1) Funduscopic examination is sufficient to correctly diagnose OHS

2) The Histoplasma capsulatum antigen that is used during the skin test may re-activate the organism that is dormant in longstanding chorioretinal scars.\(^{(15)}\)

The test result becomes positive after the fungal infection has been present for two to four weeks and remains positive throughout life. People who have been previously infected often have partial protection from reinfection.
INTRA VITREAL INJECTION

Clinical Uses
The vitreous humor makes up nearly 80% of the total volume of the globe. The vitreous serves as both a shock absorber for the eye, as well as a storage area for nutrients (17). Inflammatory cells or red blood cells can fill the vitreous if a posterior uveitis or vitreous hemorrhage occurs. This reservoir-like property of the vitreous can be utilized by injecting medication directly into the vitreous body. Intravitreal injection of triamcinolone acetonide (Kenalog) is commonly used to treat numerous causes of posterior uveitis and macular edema. Antibiotics can also be introduced into the vitreous to treat endophthalmitis (18).

Ocular inflammatory processes which cause intermediate, posterior, or panuveitis commonly require intravitreal steroid injections to quiet the inflammatory response. Infection, which can be caused by viruses, fungi, bacteria, or protozoa, stimulates an immune response (19). Systemic inflammatory conditions can localize to the eye or affect the eye along with the rest of the body. Also, neoplastic tumors can enter the eye and mimic an inflammatory condition.

Intravitreal injections of anti-vascular endothelial growth factor (VEGF) pharmaceutical agents, such as pegaptanib (Macugen) and ranibizumab (Lucentis) are used to halt the progression of choroidal neovascular membranes and/or retinal neovascularization.

Choroidal neovascular membranes are often treated by intravitreal injection of anti-vascular endothelial growth factor (VEGF) pharmaceuticals. Anti-VEGF treatment inhibits the growth of immature vessels by inhibiting tyrosine kinase receptors on the cell’s surface.

Although this procedure is reserved for ophthalmologists, optometrists should be familiar with the procedure for intravitreal injections. See the clinical note at the end of the section for more information on common conditions requiring intravitreal injections.

Procedure
For this procedure, the following items are needed:
- Protective gloves
- Betadine
- Gauze pads
- Eyelid speculum
- Topical anesthetic
- Topical antibiotic
- 27, 30, or 31 gauge needle

The injection site is typically 3 to 4 mm from the limbus.
www.peo-imagestock.com
Topical anesthetic is used to numb the eye. Some practitioners also use a subconjunctival injection of anesthetic to make the procedure less painful for the patient. The ocular adnexa is then cleansed with an iodine preparation (typically betadine), and the injection site is also cleansed with iodine.(20,21)

The lid speculum is put in place to retract the eyelids. The globe is stabilized and the needle is inserted into the site. The site of injection is typically between 3 and 4mm from the limbus. Following injection, a topical antibiotic is instilled to reduce the risk of endophthalmitis. Immediately following injection, the patient is checked to ensure that light perception is still possible in the injected eye.(20,21)

**Complications of Intravitreal Injections**

Fortunately, intravitreal injections have a relatively low complication rate. The most severe complication is bacterial endophthalmitis; however, endophthalmitis following intravitreal injection is reported in only 1 per 1,000 cases of intravitreal injection. Other complications following intravitreal injection include vitreous hemorrhage, retinal breaks, and retinal detachment.

Patients report variable amounts of discomfort or pain following the procedure. In rare instances, improper removal of the eyelid speculum can cause a corneal abrasion. Blepharitis must be controlled prior to intravitreal injection, and uncontrolled, active blepharitis is a contraindication to injection (18).

---

**Clinical Note: Inflammatory Retinal Inflammatory Diseases**

**Toxoplasmosis**: Toxoplasmosis is a common cause of posterior uveitis. The causative organism is *Toxoplasma gondii*, an intracellular parasitic protozoan that is capable of existence in three different forms: oocyst, tachyzoite (active), or cystic (latent). The primary host of *T. gondii* is the cat. The organism can be shed in fecal matter and transmitted to intermediate hosts. Human infection is usually from consumption of undercooked meat that contains the organism or from congenital infection from a mother exposed to the organism during pregnancy. *T. gondii* can cause a necrotizing retinochoroiditis, which presents clinically with a classic "headlights in fog" appearance to the fundus on ophthalmoscopy—the optic nerve and the active retinochoroiditis infiltrate appear yellow in a haze of inflammatory vitreous debris. Acuity can be decreased if the infiltrate involves the macula.

**Toxocariasis**: Much less common than toxoplasmosis, toxocariasis is caused by the nematode *Toxocara canis*. The natural reservoir for *T. canis* is young dogs. The organism is excreted via fecal matter and persists in soil in a dormant form. If contaminated soil is ingested (typically by young children that ingest dirt (i.e. pica)), the organism can migrate through the intestinal wall, enter the blood and lymphatic systems, and possibly migrate to the eye. *T. canis* can cause leukocoria, retinal traction bands, and endophthalmitis. The condition is usually unilateral.

**Ocular Histoplasmosis Syndrome**: Ocular histoplasmosis syndrome (OHS) is caused by *Histoplasma capsulatum*, a fungus native to the Ohio and Mississippi River valleys. The organism has also been identified in bird and bat feces. Primary infection most often occurs by inhalation of spores. Systemically, *H. capsulatum* can damage the lungs, liver, and spleen. If the eye is involved, OHS follows a classic triad appearance of mid-peripheral punched-out lesions, which represent local areas of chorioretinitis, peripapillary atrophy, and maculopathy. Two of the three criteria are needed to clinically diagnose OHS. Acuity is typically unaffected unless a choroidal neovascular membrane develops in the macula.

**Macular Edema**: Macular edema can occur by multiple mechanisms including inflammation, vascular leakage, or retinal ischemia. Physiologically, the blood-retinal barrier becomes compromised, which allows fluid to accumulate in Henle's nerve fiber layer or the outer plexiform layer of the macula. Common disease processes that induce macular edema include diabetic retinopathy, retinitis pigmentosa, branch and central retinal vein occlusion, and posterior segment inflammation. Macular edema can be induced after ocular surgery as well. Macular edema secondary to inflammatory processes is typically treated by intravitreal steroid injection if the inflammation is severe. Mild cases may be treated by a combination of topical steroidal and non-steroidal therapeutics. Intravitreal administration of anti-VEGF agents has become an increasingly popular method of managing diabetic macular edema and macular edema caused by other ischemic events, such as a branch or central retinal vein occlusion.(22)
Clinical Note: Choroidal Neovascular Membrane

Choroidal neovascular membranes (CNVM) are associated with numerous ocular morbidities. Age-related macular degeneration, pathological myopia, ocular histoplasmosis, angioid streaks, and trauma are the most common disease processes that lead to the development of CNVM. Idiopathic formation of CNVM is also possible. A choroidal neovascular membrane usually develops in the posterior pole, but peripheral CNVM is possible with chronic inflammatory diseases, such as pars planitis and retinitis pigmentosa.

Though not completely understood, the primary stimulus to CNVM formation seems to be hypoxia. Damage to the retinal pigment epithelium (RPE) and, more importantly, Bruch's membrane in a hypoxic environment stimulates the release of vasculogenic molecules, specifically vascular endothelial growth factor (VEGF), from the RPE. The new vessels that form lack endothelial tight junctions. The result can be seen clinically as hemorrhaging, serous RPE or retinal detachment, and eventual scarring and atrophy.

Age-Related Macular Degeneration: In AMD, pathological changes to Bruch's membrane deprive the RPE and outer sensory retina of oxygen and nutrients. Bruch's membrane naturally thickens with age. Patients present with a wide spectrum of pathological changes to the macula. Early-stage AMD presents as hard or soft drusen in the macula. Soft drusen may coalesce to form a pigment epithelial detachment, which further deprives the outer retina of nutrients that pass through the RPE. The RPE and sensory retina may become atrophic from chronic hypoxia. If a break in Bruch's membrane occurs, a CNVM may develop. Formation of a CNVM is the primary cause of severe vision loss in patients with AMD.

Ocular Histoplasmosis Syndrome: (Described in detail in earlier sections.) The macula can be involved in ocular histoplasmosis syndrome. Damage to Bruch's membrane in the area of the macula stimulates CNVM formation. Maculopathy is the primary cause of reduced vision in OHS patients.

Pathological Myopia (Myopic Macular Degeneration): Pathological myopia refers to a disease process involving progressive elongation of the globe with degenerative changes to the sclera, Bruch's membrane, RPE, and retina. Pathological myopia is the leading cause of CNVM formation in young patients. (23)

Angioid Streaks: Angioid streaks are pathological changes to Bruch's membrane. Although angioid streaks can be idiopathic, they are usually associated with certain diseases, such as pseudoxanthoma elasticum, Ehlers-Danlos syndrome, Paget's disease, and sickle cell anemia. Angioid streaks represent focal weakening of Bruch's membrane, which can lead to CNVM formation.

Trauma: Trauma to the globe can cause damage by either a coup (at the site of injury) or contrecoup (opposite the site of injury) mechanism. Eighty percent of choroidal damage, specifically choroidal rupture, occurs due to contrecoup injury (24). Choroidal rupture refers to breaks in the choroid, RPE, and Bruch's membrane. Ament et al found that CNVM formation was highest in older eyes with concomitant choroidal rupture involving the macula.
SUB-TENON INJECTION

Clinical Uses
Sub-Tenon’s injections have been used in eye care to treat recalcitrant uveitis, vitritis, and macular edema. In this procedure, the pharmaceutical agent is injected into the sub-Tenon’s capsule space, the area between the sclera and Tenon’s capsule (25). The use of sub-Tenon’s injection of triamcinolone acetonide (Kenalog) has decreased, as intravitreal injections have taken over as the standard of care for the previously mentioned procedures.

Sub-Tenon injection of an anesthetic can also be used to anesthetize the eye during cataract surgery, although less invasive techniques of anesthesia are more common. Although this is not performed by Michigan optometrists, it is important for us to understand the use and procedure of this injection, as well as the possible complications.(1,26)

Procedure
For this procedure, the following items are needed:

- Protective gloves
- Betadine (povidone iodine)
- Local anesthetic eye drops
- Eyelid speculum
- Forceps
- Westcott scissors
- Curved cannula
- Syringe

Sub-Tenon’s injections have decreased in popularity, as intravitreal injections have increased in primary eye care. Photo: www.revoptom.com

Complications of Sub-Tenon Injections
Subconjunctival hemorrhaging is common, and a topical epinephrine drop may be added after injection to promote vasoconstriction. The most devastating ocular complications associated with sub-Tenon’s injection are retrobulbar hemorrhaging and globe perforation. Trauma to the extraocular muscles is also possible. As with all steroid injections, use of triamcinolone acetonide can cause steroid-induced glaucoma and cataract formation (25).

Active inflammation (conjunctivitis, scleritis) and active infection are contraindications to sub-Tenon’s injection (1).
REFERENCES


14. Guidelines for Administering the Mantoux Tuberculin Skin Test. 


21. Moshfeghi A. Intravitreal injections: how you and your patient can sleep well. 
Ophthalmology Web. 


