



DERMAL FILLER COMPLICATIONS HANDBOOK

BY DR TIM PEARCE

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NON-INFLAMMATORY LESIONS

MODULE LINK

<https://drtimpearce.com/modules/non-inflammatory-lesions/>



Skin Lesions in the face can be divided at the highest level into either inflammatory or non-inflammatory.

Here we will look closely at non-inflammatory lesions.

Non-inflammatory lesions are very common, perhaps the most common follow up from dermal fillers but thankfully are also the simplest because there is fundamentally no biochemical reaction between the product and the body.

The diagnosis is based on the presence of a soft spongy lump, fullness or oedema in an area previously injected with dermal filler, but the complete absence of any inflammatory signs or symptoms. On examination by the clinician, there should be no erythema, itching pain or any significant tenderness on palpation.

The only inflammatory lesions that are difficult to differentiate from non-inflammatory nodules are chronic inflammatory nodules. The main difference with these is that they are not always inflamed enough to be red or tender. The nodule is very different to filler, it forms very slowly over some time a hard lump on palpation, more like a smooth slightly rubbery pebble. It can happen with hyaluronic acid dermal filler but happens more often with products designed to trigger a foreign body response such as poly-L-lactic acid, or Sculptra. All other lesions are most likely non-inflammatory and can be managed in a similar way.

Non-inflammatory lesions consist of isolated pockets of sterile filler or

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fluid which cause an aesthetic problem or mild symptoms in a rather passive way. Nonetheless, they are still highly anxiety-provoking for both the clinician and the patient.



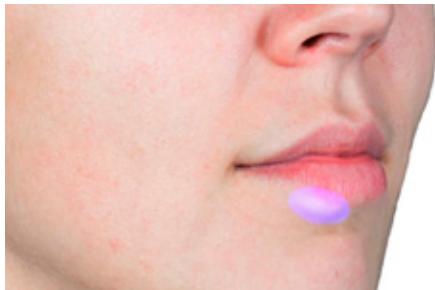
Dr Tim

These non-inflammatory lesions can be caused by 3 different factors, I call the 3 ps.

P{
the Product
the Patient
or the Procedure.

The ideal procedure would involve perfect placement of the perfect product, which would stay exactly as the clinician placed it, and never change shape, size or position. Of course, no product or procedure is ideal, so for various reasons filler is placed poorly in the wrong position or volume, or changes shape, size or position which causes a problem for the patient.

PRODUCT



Looking at the product causes, a common cause of problems is the ability of many fillers to attract moisture. Though sometimes this can be a benefit, as extra volume was needed to get a better result, it is often a problem when placed underneath thin tissues such as the superficial layers of skin anywhere, the tear trough in particular, or in any intricate structures such as lips, cheeks or noses.

The result may be perfect initially, but over the course of a few days to weeks or occasionally months or years, the filler attracts increasing amounts of water until it causes an aesthetic problem. This can be a lump or a loss of shape and detail. You see this most commonly around the vermillion border and this occurs in the days and weeks after a procedure with hydrophilic slow integrating products.

Aside from lost structure or detail, the same problem can cause lumps or bumps to form. This often happens if during the treatment you place boluses, use a hydrophilic dermal filler, and do not massage to aid tissue integration you may find a non-inflammatory, non-tender, softish lumps develop in the weeks after the procedure.

These problems are all as a result of products which integrate poorly and migrate, or attract moisture over time and become too voluminous or shapeless.

The next factor is the patient. Client selection is as always vitally important. Find out from your patient before doing certain treatments if they tend towards fluid retention. This tends to show up particularly in the morning, where they may have puffiness around their eyes and cheeks that may last for several hours.

Sometimes there is a malar mound that is present throughout the first part of the day which indicates fluid retention in that localised anatomical space.

They have anatomy that could result in fluid compartments swelling at different rates causing the formation of a bad. If this occurs without filler and you treat the area it can often get a lot worse.

Product type is of course also key, as even a low volume of the wrong filler in a sensitive place such as lips or tear trough can cause lumps bumps puffiness or nodules which physically annoy the patient and cause poor aesthetic outcomes.

Typically this is using a high viscosity product in an area of soft tissue in the hypodermis or lower dermis. For example, a filler designed to treat folds in a crease can cause a line to develop under the skin weeks later. Similarly, a filler designed to create volume when used in a place like the tear trough will invariably cause a puffy swelling a few weeks or months later.

PLACEMENT

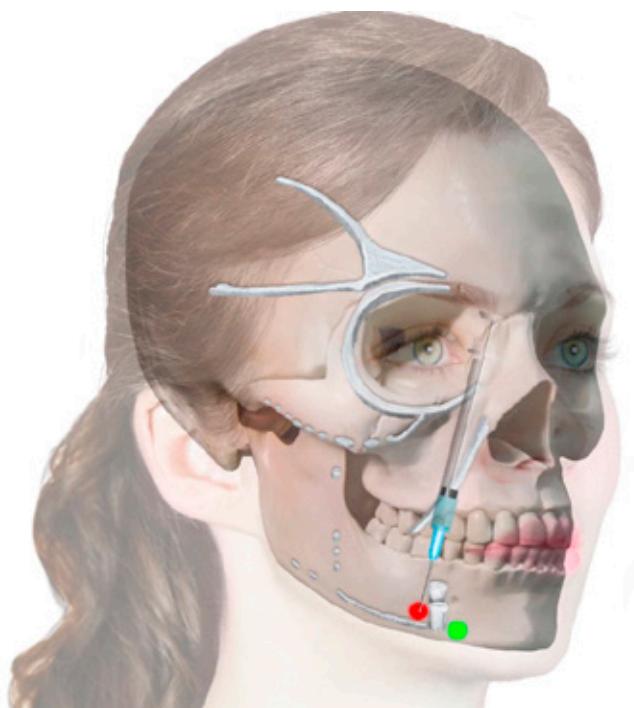
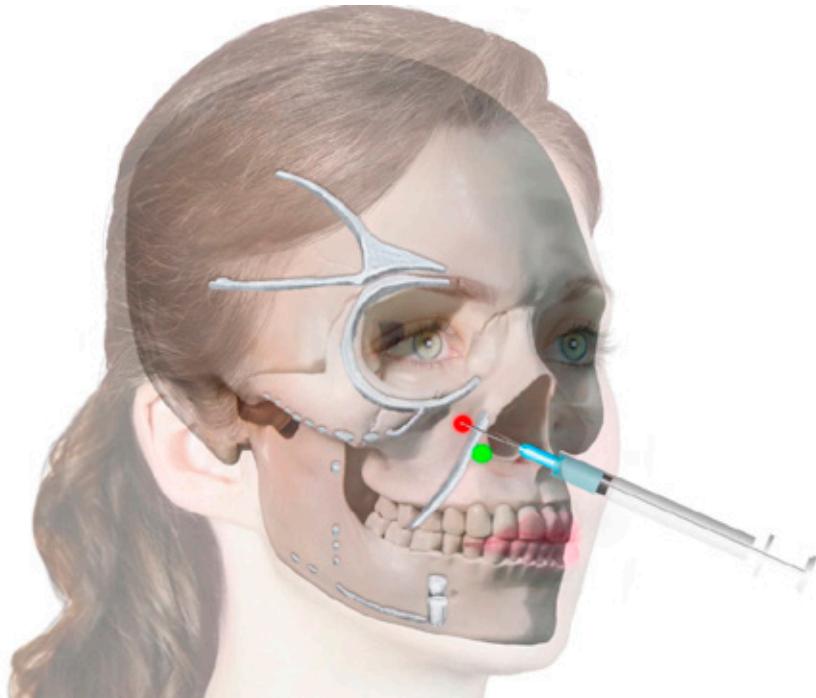
In some cases, a good quality product placed in the correct volume could end up in the wrong compartment of the face or the wrong depth. Soft bulges occurring in tissue such as the lips or in delicate structures around the modiolus, the tear trough or oral commissures. These problems are nearly always caused by being too superficial for the product, or by increasing volume on the wrong side of a ligament.

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For example, injecting the nasolabial fold, but enhancing the fat pad above by mistake, or increasing the jowl through placement in the jowl fat instead of the melolabial fold distal to the mandibular ligament. Mid-face ligaments if crossed can also cause aesthetic problems as bulges can appear where they should not if the filler is placed poorly and enhances instead of reduces bulges.



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For example, in this case, if filler was to be placed above the zygomatic ligament it would have made the problem significantly worse where as just beneath the ligament resolved the issue quite well. Accurate placement by a knowledgeable injector is vital to avoid these non-inflammatory complications.



Excessive amounts of filler is a rather self-explanatory problem that occurs either in novice injectors or competent injectors who get a little bit too overconfident before they reach proficiency.

Problem areas are tear trough and lips, especially the vermillion border, or anywhere superficial. It can, of course, occur anywhere but most places are fairly forgiving.

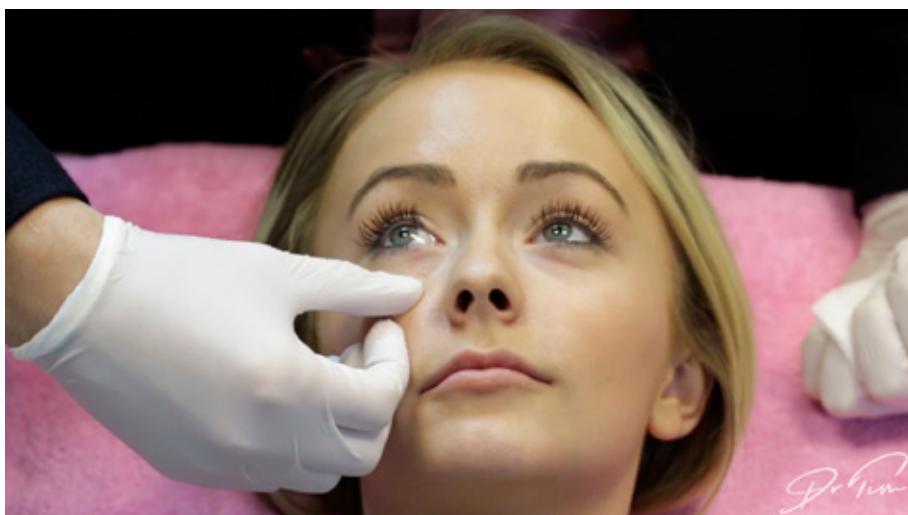
MANAGEMENT

Management of non-inflammatory nodules are all to varying degrees the same.

Invariably the first step is to massage the area of fullness or bumpiness to try to blend the dermal filler in or if its fluid to move it away from the site where it is causing an aesthetic problem.

Often new practitioners are too afraid to do this with the right amount of pressure, and then have to resort to more risky measures.

You shouldn't need to hurt your patient to get a result, but many practitioners can push significantly harder to get an improvement from these lesions and not make your patient significantly uncomfortable. A good tip is to apply the same pressure to your own face or lip and see how much pressure it takes before you feel it is too uncomfortable. You will find it's much more than you expect in many cases.



After you have tried to blend in or massage away the lesion for 5 to 10 minutes you should see a significant improvement in many cases, however, a significant proportion will remain only slightly improved, so a second option to explore with your patient is that they try a similar exercise at home for a period of 1 to 2 weeks before they return to consider more invasive options.

It is also reasonable in some cases to advise just watch for waiting if the lesion is not that distressing for the patient as many will resolve without intervention whatsoever in a period of weeks. This may be particularly appropriate where the lesion is not visible but only palpable.

It has been noted that in some patients injecting normal saline can reduce the size of a lump if it is followed with a massage. This technique is likely making hyaluronic acid dermal filler which is soluble more able to move and redistribute itself in a way that does not cause

a lump. The advantage of this is that there is no risk of allergic reaction from hyalase.

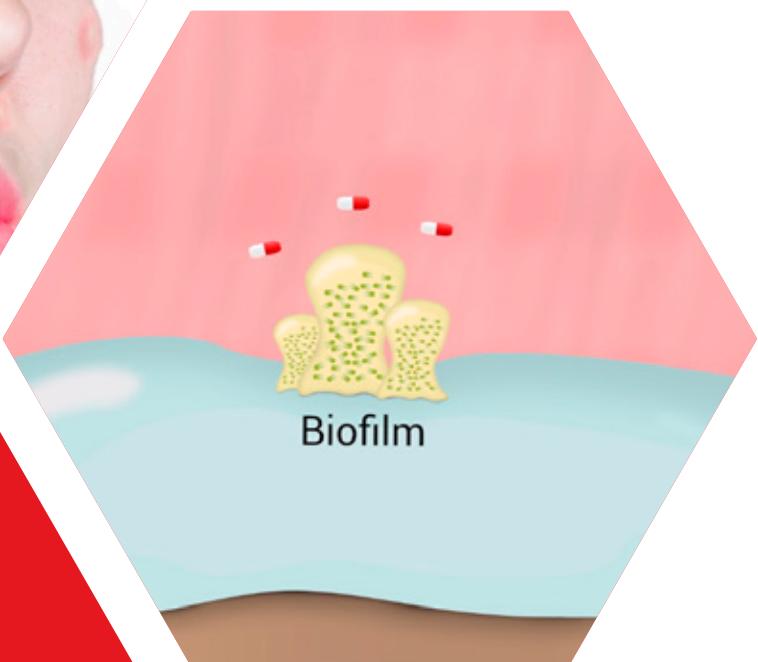
In some cases, it may be reasonable to attempt to mechanically extrude lumps. Inserting a green needle into the tissue and then squeezing the area fullness until the filler is extruded. The advantage of this technique is that you may be able to reduce the size of a lump and still get a good aesthetic result without reversing the whole procedure, and there is no risk of an allergic reaction to hyaluronidase. The disadvantage is that it often fails or can be quite traumatic compared with a chemical reversal.

Of course with hyaluronic acid, the final solution is always to inject a lesion with hyaluronidase to break down hyaluronic acid and return the patient to the untreated state.



Each region is unique and you should work through the options carefully considering the benefits and risks and patient preferences to the best outcome for the individual patient.

In cases where you failed to get resolution by following this non-inflammatory pathway you should consider treating as an inflammatory nodule as your initial diagnosis may have been incorrect, please refer to learning material on inflammatory nodules.



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INFLAMMATORY LESIONS

MODULE LINK

<https://drtimpearce.com/modules/inflammatory-lesions/>



As previously discussed inflammatory responses after dermal filler procedures can be caused by several different mechanisms. Trauma as a result of post-procedural inflammation, infection, viral reactivation, or a hypersensitivity reaction.

The literature has used multiple different terms to describe the problem. Nodules, biofilms, granulomas and type iv reactions are often discussed as if they are easily distinguished and entirely separate entities. It is becoming recognised that clinically it is often very hard if not impossible to separate these concepts. Reactions and infections, in particular, biofilm infections and delayed hypersensitivity reactions are often indistinguishable. As a result, these are often treated as the same thing, and called delayed onset nodules.

There are of course publications supporting treating most of these nodules as reactive, and others which propose mainly a bacterial cause. only and so the debate continues.

<http://www.ncbi.nlm.nih.gov/pubmed/26166260/>

To give you a complete understanding of the possible mechanisms behind nodule formation in this section we focus on understanding reactive causes even though in practice you may treat them similarly to biofilm infections.

Broadly speaking there are two different types of hyperinflammatory reaction to dermal filler, Type I and Type IV hypersensitivity. Post procedural swelling can also vary considerably and there can be an overlap of symptoms clinically with Type I hypersensitivity.

Type I hypersensitivity reaction is an antibody-mediated response to dermal filler or its constituents which classically occurs immediately, causing a bright red erythematous reaction and may cause angioedema or an anaphylactic reaction. This is exceedingly rare but does occur as a result of aesthetic treatments.

In this section, our focus will be primarily on inflammatory nodule formation, which are the most common inflammatory complication requiring treatment. This problem is caused by a Type IV immune system reaction to the dermal filler or impurities from the manufacturing process or introduced with the procedure, for example, makeup. They may be diagnosed histologically as granulomas, though we cannot make this diagnosis clinically.

Inflammatory nodules appear of course with the characteristics of inflammation: pain, tenderness and erythema or redness, but the main difference aside from the delay, is that Type IV causes a change to the texture of the filler which becomes harder and nodular.

Infection can cause a similar pattern of response, but though there are clinical differences between reactive nodules and infective nodules, they are not always clear. As a result, there is much debate as to which is which, and whether or not many are the same entity. There is some empirical evidence from treatment regimes that there is an overlap between infective and delayed onset nodules.

Many nodules thought to be reactive seemed to do worse when treated with steroids alone, rather than with antibiotics. Another study also found DNA evidence of bacteria in culture-negative lesions. These factors deny us the certainty that would enable us to treat them in a discrete manner as either infected or reacting lesions.

DIAGNOSIS

Aside from the medical diagnosis, it is important to take into account the psychosocial impact of any lesions. A small lesion that is not developing and is not visible may be left untreated, while similar lesion in a visible place may be completely intolerable to the patient and justify more aggressive treatment.

As with any intervention, it's important to make sure your patient understands all the options and the side effects and risks of any interventions and makes the decision with you to proceed and to achieve that you will need to explain your rationale.

Although we are focusing on inflammation here, remember to consider Non-inflammatory nodules caused by the filler which are the most common cause of lumps, bumps and nodules.

Inflammatory nodules are caused by infection or reaction. Complete diagnostic certainty is seldom possible in clinical practice, but here we will consider some of the factors that have classically been used to differentiate infection from hypersensitivity reactions.



Hypersensitivity occurs in two forms, Type I reaction usually occurs rapidly after the procedure often within a few minutes. This antibody-mediated reaction can be localised to the tissue or as angioedema, or systemic causing anaphylaxis. It involves rapid inflammation of the tissues, swelling and edema and can be life-threatening and classically occurs in minutes.

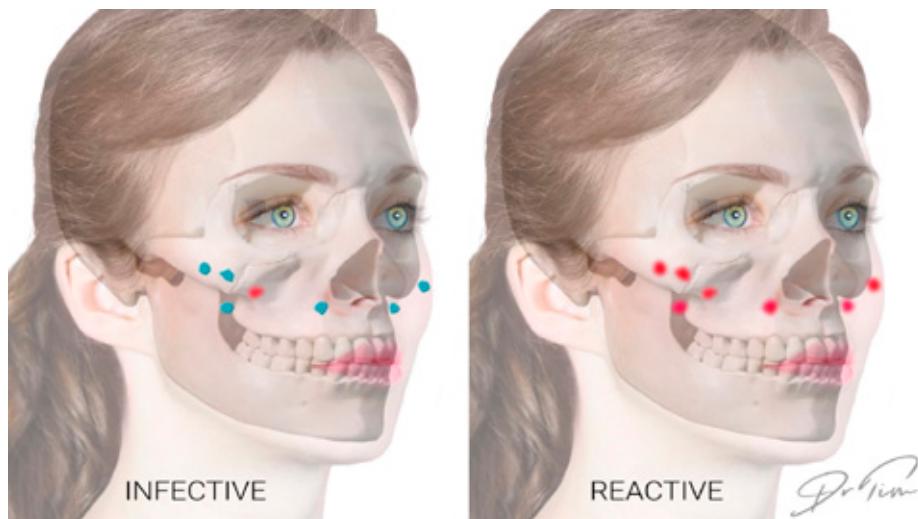
Type IV reactions, on the other hand, are always delayed in onset. They classically form multiple red tender lumps or nodules a minimum of 48 to 72 hours after the procedure, and often occur many months after the procedure.



Though they may appear very similar to infective nodules, there are some differences that may be helpful.

For example, sterile reactions are theoretically more likely when the lumps occur wherever the product was injected, whereas an infection may be more likely if the lumps are isolated to one injection site and not all. They occur a minimum of 48 to 72 hours after exposure.

Nodules which form during periods of high immune activity during viral infections such as flu, or flare-up of an autoimmune disease such as rheumatoid arthritis are likely to be reactive rather than infective, especially if all the filler reacts at simultaneously.



An isolated area of nodule formation and inflammation which occurs without any obvious immune trigger would fit better with an infective cause and may seem more likely to be a biofilm reaction.

Firm nodules which develop very slowly without redness or pain all over the areas treated are also likely to be foreign body reactions or granulomas. These are the hardest to treat as they tend to present late and the only clue to their reactive nature is that all the areas treated show nodule formation.

What about bacterial causes?

When can you be wrong about inflammatory causes? Some of the literature points out that any product that has been mixed in the clinic rather than in a factory, injected with a different device to the one supplied, or injected through generally unclean skin or through thick makeup may still appear to become inflamed at multiple different sites but due to infection. Therefore, widespread inflammation does not rule out the chance of infection completely. Within the context of a procedure

you carried to a high standard yourself, there is more utility in noting the spread of nodules as you will know that the risk of infection in multiple sites is lower.

Clinical diagnosis can be aided by any improvements seen after antibiotics are used in isolation. In these cases, it may seem to be that the inflammatory response is due to the presence of bacteria. Similarly, if you see a generalised reaction soon after the procedure, a Type I allergy may be quelled by steroids and antihistamines alone.

In the case of lumps and bumps, the use of steroids alone with an initial response does not necessarily indicate the absence of bacteria, as the clinical signs of infection are also diminished by steroids which obviously decrease the inflammatory response to the infection.

Ideally, along with clinical examination and history, one should attempt to send a sample to try and Culture any bacteria that may be present.

PATHOGENESIS

Angioedema, redness, itching occurring immediately after an injection means a Type I hypersensitivity to lidocaine, anesthetic cream or possibly but rarely, hyaluronic acid.

Type I reactions occur as a result of immunoglobulin mediated allergic response. In theory, these reactions only occur after the first exposure to an allergen at some previous point in time. The subsequent inflammation on secondary exposure to an allergen is far worse. This type of allergic reaction is much more common when the patient has a history of other allergic reactions. To prime, the immune system first antigen presenting cells need to pick up the allergen and take it to the lymph nodes. This happens regardless of allergy. If the patient is allergic, here the antigen triggers T helper cells to change. They first pick up the new allergen and then trigger B cells to start making IGE antibodies to the new allergen. These antibodies stimulate eosinophils, and

activate mast cells. The mast cells then become ready for action if the antigen reappears.

This is when the anaphylactic reaction may occur. The primed mast cells if it sees another allergen will release a load of pro-inflammatory mediators which trigger the allergic response. Of course one of the main mediators of this reaction is histamine. This can trigger the allergic response, including blood vessel dilation and increase in permeability as well as bronchospasm in hyper-allergic individuals in the early phase reaction. There is also a late phase that continues an allergic response 9-12 hours later even after the allergen is gone.

Of course, as we know if the scale of inflammation is large enough and the vessels become permeable enough, the loss of fluid into the tissues can make it impossible for the heart to supply enough oxygen-rich blood to the brain. The airways of course also become inflamed and the bronchus may constrict, decreasing air entry and adding to the perilous situation of an anaphylactic reaction which requires immediate life support and emergency assistance.

http://www.youtube.com/watch?v=2tmw9x20t_Q

TYPE IV HYPERSENSITIVITY

Reactions to the product that cause delayed lumps or nodules are thought to be a result of type iv hypersensitivity reactions, the same response that causes granulomas. This type of hypersensitivity is still rare but much more common than Type I hypersensitivity with respect to hyaluronic acid dermal fillers.

Granuloma formation could be considered the final stage of inflammation and wound healing following reaction to foreign material, so not all reactions progress all the way to granuloma formation. Granulomas are composed mainly of macrophages and foreign body giant cells. Technically they can only be diagnosed through a histological analysis, not through clinical examination.

Granulomatous reactions to sterile products are type IV hypersensitivity reactions and are by definition systemic. This means all the areas where the offending product was injected would, in theory, become granulomatous.

Type IV hypersensitivity is a cell rather than antibody-mediated, reaction that damages tissues in the process of trying to destroy a foreign compound.

HOW DOES THIS REACTION OCCUR?

This type of immune response is usually triggered in its first step by a Langerhans cell, these dendritic cells are present in all layers of the skin. They are the bodies detection mechanism for infection in the skin. When they pick up a foreign antigen they present it at the nearest draining lymph node to CD4+ T cells.

If the protein is recognised by CD4 cells the cell matures into Type I helper T cell. This cell can release interleukin 2, and interferon gamma, which trigger the proliferation of T cells and activation of phagocytes in the area.

The activated macrophages release many powerful inflammatory cytokines, which dilate blood vessels and make the endothelium more permeable, allowing more immune cells into the area. This all triggers what we see as inflammation. Oedema, redness, tenderness, and sometimes systemic side effects like a fever. The activation of macrophages also releases many damaging compounds, though intended of course to damage bacteria, they also unfortunately damage healthy tissue in the process.

This entire process doesn't happen immediately in Type IV hypersensitivity reactions. Clinically, it is useful to know that it takes a minimum of 48 to 72 hours for inflammation to really take hold and this can be useful in terms of diagnosis.

It's also useful to know that the same type IV hypersensitivity reaction is involved in the autoimmune diseases that we worry about, such as rheumatoid arthritis, Inflammatory bowel disease or multiple sclerosis.

This is one of the reasons autoimmune disease can be a risk factor for having filler procedures. The same arm of the immune system causes a reaction to dermal filler is already active in patients with active autoimmune disease. This is also why an intradermal test for people with autoimmune conditions are not sufficiently reassuring unless you wait for at least 72 hours.

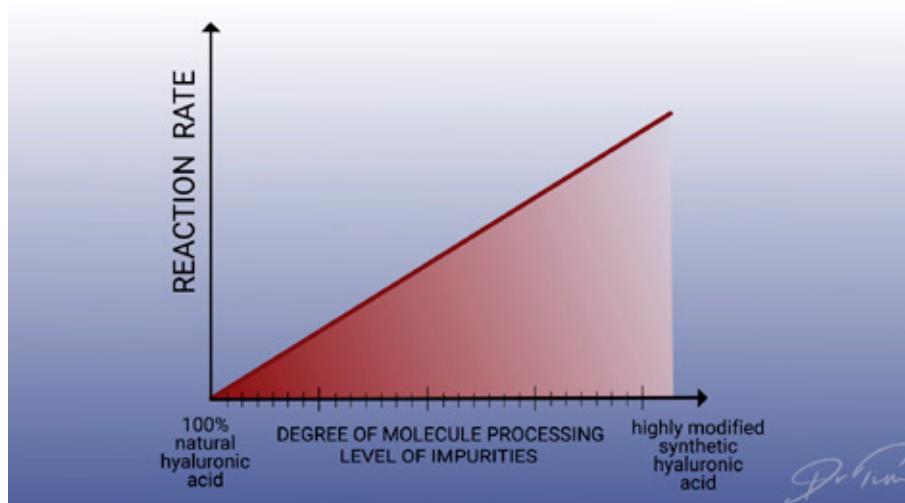
What are the theoretical risk factors Type IV hypersensitivity reaction from dermal fillers?

Hyaluronic acid is in theory immunologically fairly inert, as it is widespread in the body already and shouldn't usually stimulate an immune response.

There is some evidence of a difference in reaction rates between different products, implying that the way different products are manufactured may alter the risk of reactions. It would fit with the foundation principles of the immune system that the more altered the molecules get from their natural form, the more likely they would cause a reaction.

There may be some indication that different products have different reaction rates, supporting the hypothesis that the more engineered a product, the more chance of hypersensitivity reactions. In addition, impurities from the cross-linking or fermentation process as well as particle characteristics like size, surface and charge may affect the reaction rates. Unfortunately, a lot of this information is of commercial value and not shared. In the UK and Europe there is no requirement to test the reaction rates as they must to get FDA approval in the US, which is why there is most certainly to be gained by using products

approved by the FDA, as at the very least the risk is known, and it creates a pressure on manufacturers to create the safest possible formulas.



Of course, most hyaluronic acids are actually produced by bacteria, usually, *Streptococcus zooepidemicus* and it does say on product literature that Fragments of that bacteria, particularly the cell wall may be present in trace amounts. This has been hypothesized as a cause of reactions in some cases, though it is also argued that these fragments would be too scant to generate a significant sustained response.

There have been cases of dermal fillers manufactured poorly and causing frequent reactions at every injection point. Even well-known brands such as Restylane, according to a claim in one review article, they had an unusually high reaction rates due to impurities left in them prior to reformulation and subsequent FDA approval.

This is one reason why FDA approval is still the gold standard for product selection as it involves data on the outcomes on humans treated unlike CE marking which does not require this data. As a result CE-marked fillers are an unknown quantity when it comes to reaction rates. We can only rely on the class of molecule rather than the specifics of each manufacturer to reassure our patients.

It's also worth remembering that many non-hyaluronic acid products are also used around the world and are well known to cause reactions, some with catastrophic consequences.

We must also consider the black market, where even inappropriate substances have been used in human beings including oils, and silicone sealant intended for plumbing purposes. Most patients are still not aware of what they were injected with, and the attending clinician must be aware of this if it is not their patient.

MANAGEMENT

Management of delayed onset nodules can vary depending according to the clinical picture.

Conservative management may be appropriate if the clinical picture shows the nodules are gradually improving or small, stable and of no aesthetic consequence. Often lesions will recover without intervention and the risks associated with those interventions are therefore minimised.

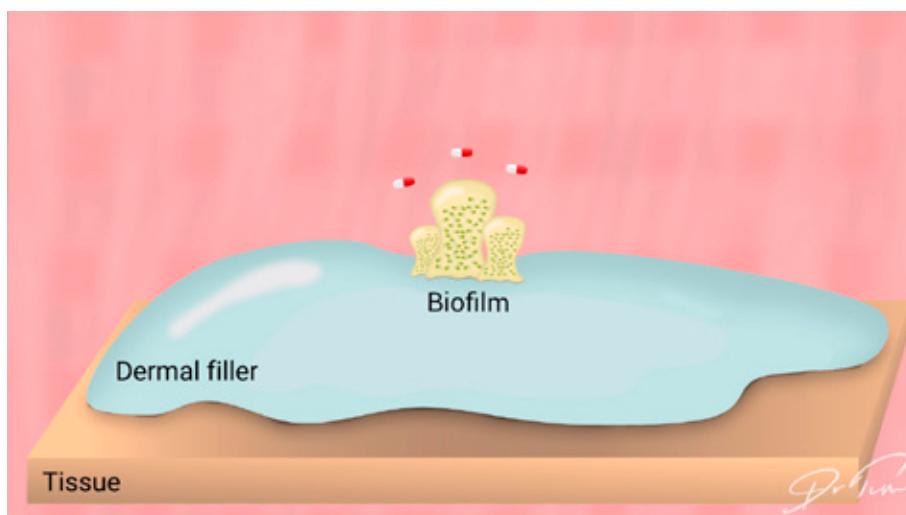
It is sometimes appropriate if there is a clear primary cause for a reaction such as a flu-like illness a few days before the flare-up, to either monitor closely with watchful waiting or to prescribe antihistamines and steroids once the patient is over the primary infection.

If there is any uncertainty it may be wise to prescribe an antibiotic along with steroids and this is a common regime where inflammation appears quite significant and the clinician is concerned about making a hidden infection more severe by suppressing the immune system with steroids.

I do not advise any immunosuppression in a patient who is systematically unwell with the primary infection of any sort. They should be seen and treated if appropriate by their GP first.

If treatment is slow to yield results, continuing antibiotics under a watchful eye is a reasonable course of action, however, you can consider the patient's emotional state and decide to be more aggressive if this seems to be in their best interests.

We know from some small studies that hyaluronidase works to resolve reactive nodules and in theory that it may act to deprive bacteria in a biofilm of a surface to hide on.



For this reason, In my practice, I have used hyaluronidase at the end of week 1 if no significant improvement has occurred and then to continue the antibiotics for one more week before a review.

If you are considering a biofilm reaction as the primary cause there is some rationale in being more aggressive early on since biofilms theoretically become harder to treat the more developed they get, and while there is filler in the area the antibiotics may be ineffective.

The pathway suggested by the ACE group in the UK is to continue antibiotics alone for 2 weeks, and then to add an additional antibiotic for another 2 weeks before using hyaluronidase.

BIO FILM- First Line
Mono Therapy

Macrolide

Clarithromycin 500mg twice daily for 14 days

Tetracyclin

Minocycline 100mg twice daily for 14 days

Doxycycline 100mg twice daily for 14 days

Continue for another 14 days if improving but.

HYALURONIDASE (See Elective Reversal Protocol)

SUSPECTED BIOFILM- Second Line
Dual antibiotic therapy

Macrolide AND Tetracycline or Quinilone

Clarithromycin 500mg twice daily for 14 days

Doxycycline 100mg twice daily for 14 days

Ciprofloxacin 500mg twice daily 14 days

Steroid Regime *See BNF

Prednisolone

Initially 10–20 mg daily for 7 days
Up to 60mg daily in severe cases

Alternative: Dexamethasone 0.5–10 mg daily for 5–7 days.



Hyaluronidase can be repeated 2- 4 weekly to try to break down the product and any biofilms.

If deletion fails to respond initiate dual therapy second-line antibiotics. It's suggested a combination of a macrolide and a tetracycline such as clarithromycin and doxycycline are used.

PREVENTION

Reducing the risk of biofilm reaction and nodule formation requires controlling variables in the following 5 domains:

- Patient selection
- Product selection
- Pre-procedure and Injection technique
- After-care and safety netting
- Early diagnosis and treatment

PATIENT RISK FACTORS

Patients at higher risk of inflammatory nodules include anyone who is immunosuppressed, including anyone with a systemic illness that may cause immunosuppression such as uncontrolled diabetes liver or kidney failure or blood disorder including any disease affecting the cellular immune system.

Course many patients have iatrogenic immunosuppression caused by the drugs we used to suppress autoimmune diseases. These patients may be at both increased risk of infection and a sterile nodule formation due to the risks associated with under and over activity they may experience.

Avoid treating anyone with an active infection, especially oral, throat or skin infections which may seed bacteria into the filler triggering a biofilm reaction.

Remember also that infection anywhere including viral infection may be a risk factor for sterile reactions to dermal filler and nodule formation due to the extra activity of the immune system when fighting an infection.

Patients with active autoimmune disease may be at risk for nodule formation Especially when their disease is active. currently, manufacturers recommend a dual allergy test before any treatment.

This involves an intradermal small 0.025 bolus of hyaluronic acid in the forearm before waiting two weeks for a review and checking the area is free of lumps bumps nodules or erythema and then repeating the test again, before bringing the patient back two weeks later and treating only if that test is negative

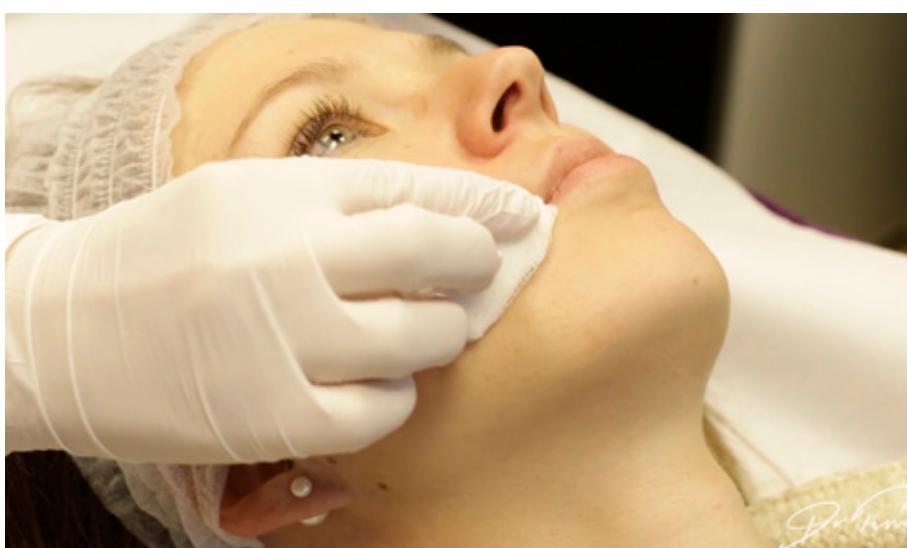
Product

When it comes to reactions it's my opinion that the best guide to product risk factors comes from the FDA approval process. unfortunately in the UK dermal fillers are not required to be tested on humans before being sold. As a result, the data is unknown about rates of reactions to different dermal filler products. Using a product that has at least been approved in the US gives some reassurance that the risk will be in the acceptable range.

It is also my opinion based on how awful it is to deal with even reversible complications from dermal filler that non-reversible dermal fillers should be avoided wherever possible, and used only when they are the only option for the patient and the benefits outweigh the risks.

PROCEDURE

Reducing the risk of nodule formation during the procedure really is entirely about clean working practices. it is essential to remove all makeup which is often high in bacterial count, and clean the skin thoroughly. remember that bacterial count will be higher and harder to reduce near the oral comitia or anywhere close to the wet dry border, as well as the complex curvatures around the alar base nostrils hand hairline.



Aftercare advice and safety netting

Aftercare is primarily about reducing the risk of introducing bacteria or makeup into needle holes. advise your patients of the importance of keeping the area clean and not introducing makeup until holes have healed over. most people are constantly touching their face many times per day, so it may be helpful to advise your patient to wash their hands and not to touch their face to give an extra layer of protection.

We often say to wait 24 hours before applying makeup, there is some evidence that holes actually reduce size quite quickly and become bacterial resistant within a few hours which is reassuring

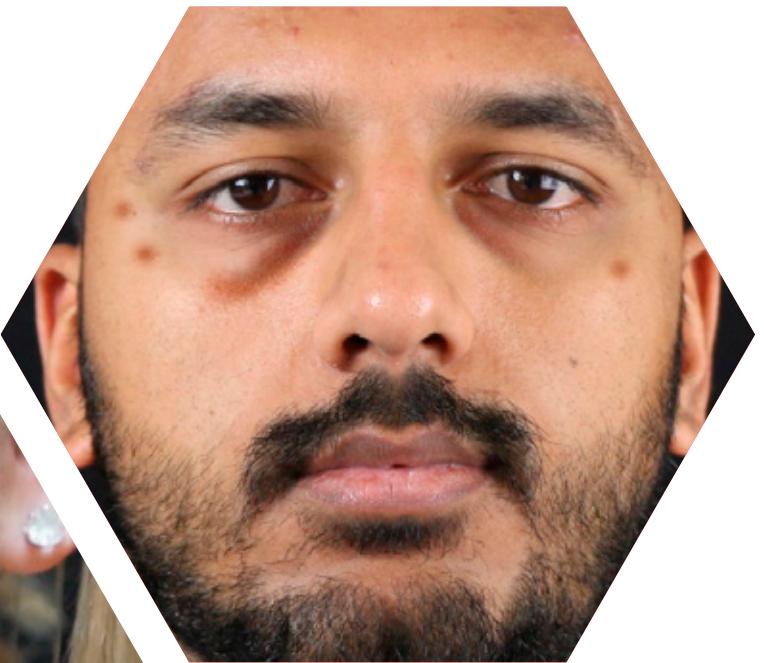
Safety netting is an important part of aftercare advice. make sure your patient knows what to look for, what is normal and what requires you to review them as soon as possible.

Early diagnosis makes most problems easier to solve, and affords some medicolegal protection to the clinician if written down in an aftercare leaflet.

Make sure your patients know exactly why they should contact you how they should contact you and what to do if you are not available so that they get the treatment they need as soon as possible.

In summary

Reactive and infected nodules are difficult to differentiate and can be stressful to manage as recovery is quite often slow and tortuous, requiring different treatment regimes and eventually sometimes referral. One of the most helpful things you can do for your patient is be clear what the road ahead looks like as far as possible to give them certainty about what will happen in each event reality. ultimately that is the primary purpose of this video series and supporting materials, so put them into practice when required.



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HAEMATOMA

MODULE LINK

<https://drtimpearce.com/modules/inflammatory-lesions/>



Bruising, Haematoma, ecchymosis, petechiae, purpura.... These are all terms to describe different types of bruising. Bruising is a major issue for patients, and often seen as a minor issues for clinicians. I have reduced my rate of bruising substantially over the years, and it is vital for improving the client experience, client retention rate, after-care workload and reputation. Bruises are so much more to a patient than just blood beneath the skin. For many, they represent a story but other people made tell about them. Is it an injury from a drunken fall or abusive husband, or is it that you have been having cosmetic treatments!? They will need to explain the bruise to many concerned friends, and they may see strangers wondering about the cause. For many it simply represents an injury, when the patients says to you 'you bruised me last time' they're really saying 'you injured me!'. This is why it's so important to reduce the chances of bruising and to know how to explain it to your patients and help them through it. It's possible to reduce bruising although it may seem self-evident to you what causes a bruise, there is much to be gained by breaking this problem down into detail so that you have a greater ability to reduce bruising, to explain it to your patients (who frequently do not truly understand it), and to manage it more effectively.

SO WHAT MAKES A BRUISE?

Bruising of any type is blood which has escaped the vascular system, and spilled into the tissues. It can be caused in various ways during different procedures. The most common would be needles puncturing vessels, which may be veins, venules, arteries or arterioles, or capillaries, or by tearing vessels often with a cannula.

- Petechiae are the smallest pinpoint bruises 1-2 mm across you may see at needle entry points.
- Ecchymosis is simply skin discolouration, the medical term for a common bruise.
- Smaller bruises up to 1 cm may be called purpura while bleeding under the skin that forms a palpable lump of clotted blood is called a haematoma, the most severe form of bruising.

Each one of these scenarios will cause a different outcome, and we should be aiming to limit trauma to all these vessels as much as possible. Bruises are highly variable, and you may find yourself occasionally getting severe bruises from minor procedures, or doing huge procedures and getting no bruising. There are many factors at work. Understanding bruising variables in detail will also increase the number of ways you can reduce this event.

You could break down bruising into three main factors. Firstly there is the degree of trauma, that is the number of injections or the size of holes or tears in the tissue. Second, the volume of blood flowing through vessels in the area being treated, and finally the rate of blood clotting once trauma has ensued.

The first factor, trauma is the factor most easily controlled by the clinician. By focusing on technique it is possible to dramatically decrease bruising, but it takes a relentless intolerance to bruising, precise needle control, and anatomical knowledge to reduce trauma to a minimum.

The second factor, the volume of blood flowing through the skin is affected by the cardiac output, and states which increase blood diversion to the skin. A patient who rushed to get to your surgery may be flushed and have a higher rate of blood flow. Most commonly the cause is raised body temperature, as thermoregulation mechanisms automatically divert blood to the skin's surface. The ability to cool the body down comes from our ability to divert blood into more superficial tissues and then to perspire to lose heat as sweat evaporates.

Veins and arteries become dilated, and the quantity of bleeding and bruising increases proportionally.

In addition to temperature variations, hormones can also affect the blood in the skin. Blood flow prior to ovulation and diversion to the skin's surface increase and perimenopausal flushing may also affect the amount of blood in the area being injected.

Recent alcohol ingestion can also dilate vessels, as both alcohol and the breakdown products of alcohol, especially acetaldehyde cause vasodilation which is a risk for bruising.

In these scenarios, the increased volume of blood in the skin makes bleeding more likely and increases the amount of blood which may escape from the vessel. Take note the next clinic you run on a hot summers day and you will observe an obvious change to the number of injection points which bleed, and the total quantity of bleeding.

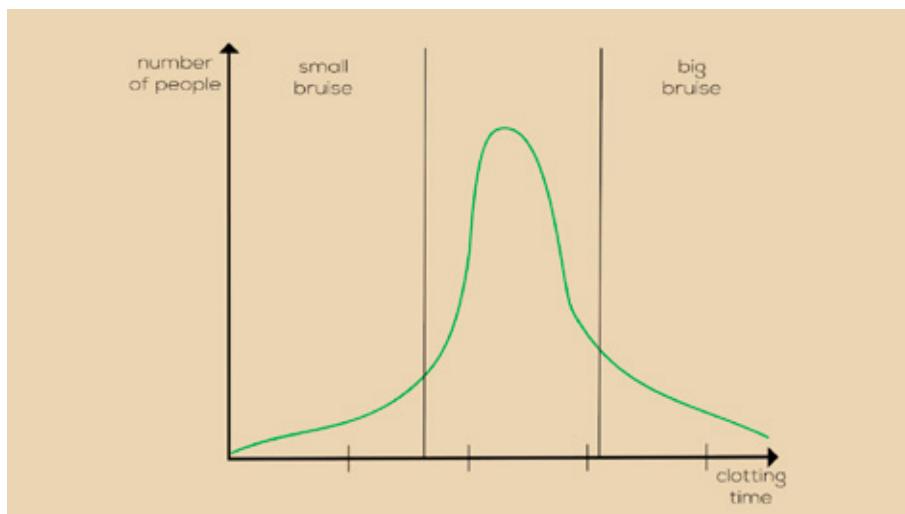


It's also worth noting that very heavy exercise post-procedure is a risk for severe bruising. I personally have witnessed cases of patients ignoring advice and returning from a long run with two black eyes after a tear trough procedure.

Thirdly, the rate of clotting is a significant risk factor for bruising.

There are those patients who seem to stop bleeding rapidly, while others may slowly bleed under the skin for hours after the procedure.

Clotting, like so many natural phenomena, fits to a degree on a bell curve. though most people clot within the average time there are those who will take longer for no specific reason, and bruise more easily.



There are of course many external factors that may affect clotting time too. The most common will be the use of blood thinners.

These include anticoagulants like warfarin, low molecular weight heparins, and factor Xa inhibitors like rivaroxaban as well as anti platelets like dipyridamole and aspirin.

None of these drugs are a complete contraindication to having injectable procedures, but they do decrease the benefit to risk ratio, and this should be carefully discussed with your patient during the consent process Weigh up the impact of a bigger bruise, especially if it lasts for a month and the benefit a treatment which may only last three months and make sure they accept that ratio.

Alcohol intake can both decrease and increase clotting times depending on the level of intake and individual variation.

TREATMENT OF BRUISES

The most effective time to start bruise management is as soon as it appears. Compress a developing bruise firmly for 5 minutes to massively reduce the size it reaches.



It may aid to compress a haematoma to spread it out and increase the surface area which will both reduce the risk of visible lumps and also increase the rate of breakdown as the blood is not lumped together. An established area of ecchymosis is not easily treated, but many people do use vitamin K and arnica creams. The evidence for these remedies working is absent, but belief in them is widespread and it may help people feel in more control which is valuable. Finally, there are very rare occasions when a haematoma may be so large that it is worth treating, and hyalase is sometimes used to reduce the size of haematomas. It is possible to aspirate blood from a haematoma after you have injected hyaluronidase, because it can help the blood to liquefy. Because the process of injecting can also cause for the bleeding so this should be considered carefully depending on the context.

SO HOW CAN WE REDUCE BRUISING FROM INJECTIONS?

Seek out risk factors and limit them: alcohol, heat, exercise, and medication. Do not stop medication prescribed for secondary prevention of cardiovascular disease. The decision to stop any medication should be taken in the wider context of the patient's health, not with a narrow focus of reducing bruising.

Consider what you can do to avoid the major vessels? Make sure you know the most likely three-dimensional position of the arteries and veins so that they may be avoided in each part of the face.

You can, of course, see veins, so you can spend some time finding the veins before a procedure and marking out the area so that they can be avoided. This is most simply done by lying the patient flat so that blood becomes distributed to their head and makes veins easier to see. Use good light, and mark the safe areas to inject or the veins themselves with a pencil.



Because blood is pooled when lying down, sit the patient up for injection so the blood drains from their head and the veins empty again prior to the injection.

Reduce trauma

Control of your needle tip is one of the most significant ways to reduce bleeding and bruising. The vast majority of the improvements I have made have been as a result of these injection techniques. Next time you see a really experienced injector, observe their whole body, not just the needle tip. Reducing trauma all starts with getting perfect control of your needle and perfect control starts from your feet up.

By following these tips I think you can reduce bruising by 90% compared with a novice injector. Increase needle control in the following ways:

- Control starts with your feet. You must be stabilised. Feet shoulder distance apart and with equal weight distribution across your feet.
- Then stabilise your upper body by leaning against your bed with your upper body.
- Stabilise your arm below the elbow. You can stabilise your hand at the wrist or below usually on the top of the bed or the patient's shoulder, forehead or cheek.

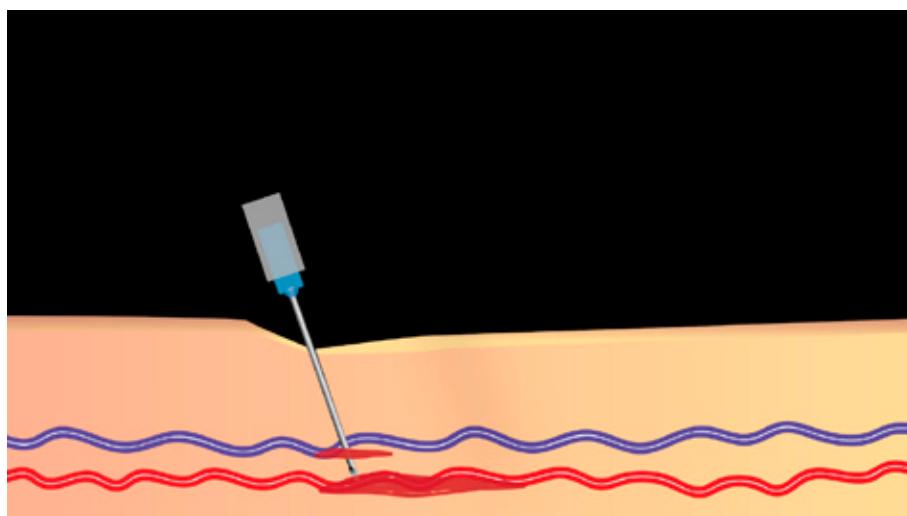


Make sure the patient's head is resting against the head of the bed and not being held up by their neck muscles which is not a position able to completely resist the forces of an injecting hand.

With your supporting hand control, the patient's head position and the surface of the skin so that it becomes a fixed, rather than easily moved by the injecting hand. Injecting unstable skin decreases control and will increase the depth of penetration

Make sure needles are always sharp

After a needle has been used a few times on the patient it becomes dull, this increases the force required to penetrate the skin. The problem is skin is tough on the outside and tissues are much easier to penetrate deeper in, so at the moment it breaks through the epidermis, the skin snaps forward and the needle penetrates much deeper than intended and is more likely to traumatise arteries and veins.



Inject as superficially as possible, taking the anatomy into account. Injecting around the eyes, in particular, is much less likely to bruise if you only just penetrate the dermis - 1-2 mm is usually enough. Beneath this depth is a venous plexus and bruising is very likely.

Withdraw needles slowly, especially when deep which may give layers of tissue time to close up and decrease blood pooling.

Immediately compress any bleeders, and hold the area for 1 minute if bleeding seems notable.

It's also useful to reduce the number of injections wherever possible, this can be done by fanning when you are injecting dermal fillers- in certain places you may be able to deliver product in two distinct areas using the same entry point by partially withdrawing, changing the angle and strategically placing injections to reduce the total number of penetration required.

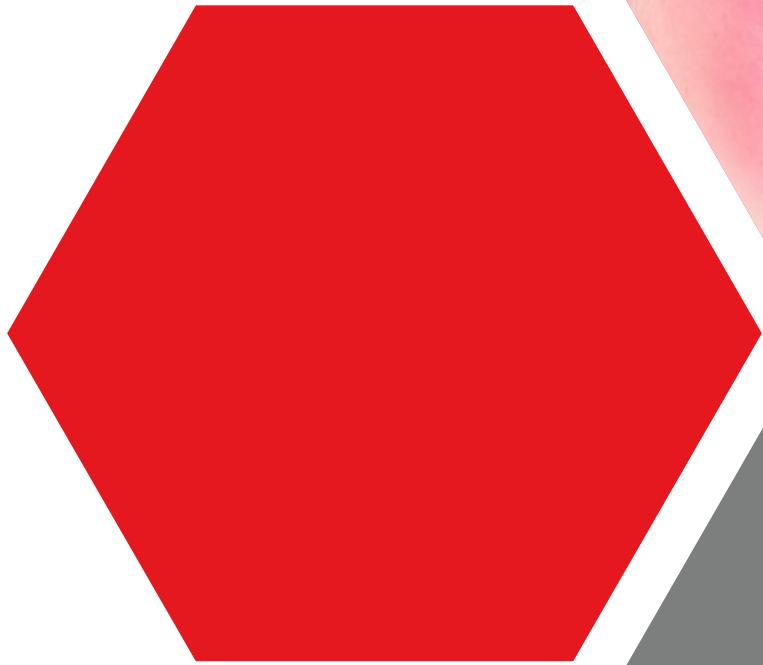
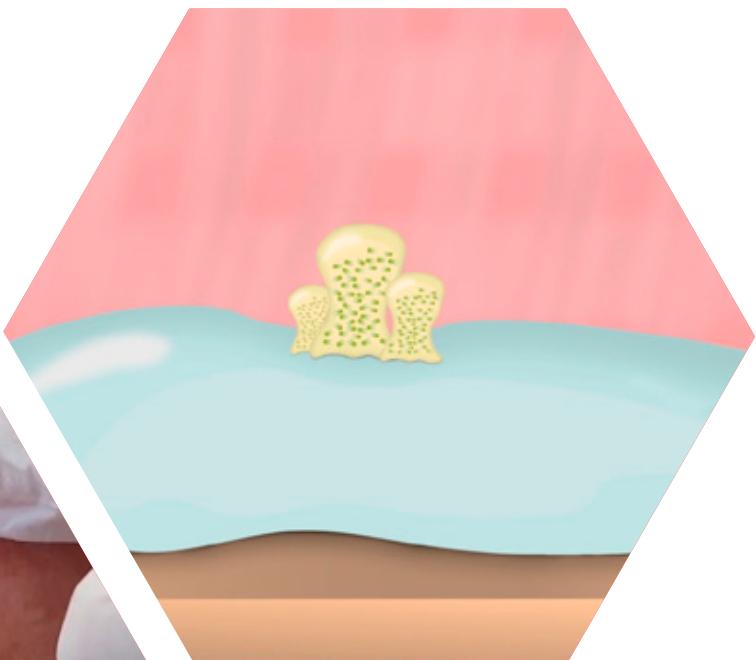
Use a cannula for dermal fillers where possible.

In summary

Remember it's more than just a bruise to your patient, it's an injury and a story which others may tell about them. When you do get a bruise, explain to your patient what has happened and why. Take time to hear how it has affected them and show that you care. Give them clear expectations of what will occur next. small bruises recover in 5 to 10 days. large bruises will take a month. In the most extreme cases bruises may still show some signs of yellow in the skin at 6 weeks after injection.

Reflect on the injection that caused it and discuss with your patient ways that you may reduce the risk if they were to have the procedure again. This may restore trust if you feel they have lost it.





4

BACTERIAL INFECTION

MODULE LINK

<https://drtimpearce.com/modules/bacterial-infection/>

**INTRODUCTION**

The effect of bacteria on dermal filler procedural outcomes is rare but varied. The reported incidence ranges between 1 in 500 to 1 in 2500 depending on the data set.

Any clinician needs to be able to recognise signs of infection across a spectrum of presentations.

On one end of the spectrum a very mild period of transient inflammation may be all that is noticed before the body kills off the offending bacteria, while on the other end of the spectrum are biofilm reactions, abscess formation and even the chance of life threatening septicaemia.

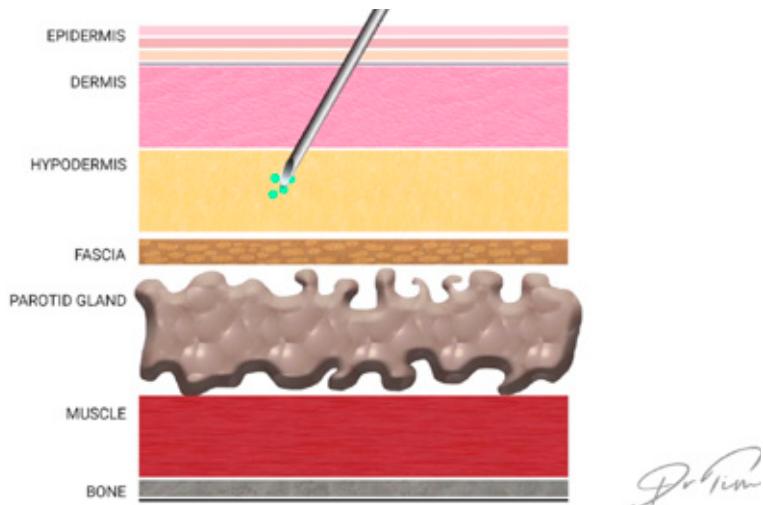
The competent physician must understand how to diagnose, manage and prevent infections in order to minimise risk to their patients.

PATHOGENESIS

So how does infection arise as a result of dermal filler procedure?

There are two ways that bacteria can reach dermal filler and trigger an infection. The least spoken about is hematogenous spread from nearby sources of bacteria, such as infected tonsils, teeth or gums. It is well known that even after brushing healthy teeth, there is an influx of bacteria into the bloodstream and this bacteraemia is a risk for spreading infection to nearby implants. Bacteria can enter the bloodstream and then in rare circumstances make their way to other parts of the body, settle and then start a local infection. The more obvious cause of infection that clinicians can control occurs as the bacteria is introduced along with the product when the procedure is carried out.

This is most likely a bacteria that is carried directly from the surface of the patient's skin with a needle before starting to grow beneath the skin near the filler.



The origin of the bacteria from the surface of the skin makes the most likely organisms skin based pathogens. You can divide the body terms of its skin flora roughly at the level of the waste. Typical organisms above the waste include gram-positive species such as *staphylococcus epidermidis*, *corynebacterium* species, *staphylococcus aureus* and *streptococcus pyogenes*. Though less relevant to us in facial aesthetic procedures, below the waist we see gram positive and gram-negative species, such as *enterobacteriaceae* and *enterococcus* species.

<https://www.ncbi.nlm.nih.gov/books/NBK333408/>

Other sources of bacteria importantly include bacteria that reside in the mouth, and procedures that involve the dermis or mucosa near the wet-dry border are highly likely to risk introducing oral bacteria into the tissue and causing a wide range of infections. The oral cavity is home to anaerobic bacteria which include *actinomyces*, *bacteroides*, *eubacterium lactobacillus* and many fungi. Importantly, bacteria tend to accumulate in both the hard and soft oral tissues in biofilms because bacterial adhesion is particularly important for oral bacteria.

It may also be an increased risk to inject near hair which is difficult to sterilise. Men with beards or entry points near the hairline could be considered a risk for introducing bacteria at a higher rate than standard.

BIOFILMS

Biofilm infections are a special class of infection that need extra explanation. The formation of a biofilm is associated with persistent tissue and foreign body infections which tend to be highly resistant to antibiotics. It's thought that up to 80% of human bacterial infections are associated with these films, and they are difficult to diagnose and treat appropriately. Biofilms tend towards chronic infection and can have a significant impact on the function of the immune system. They are also particularly associated with some of the bacteria commonly found on the skin including staphylococcus epidermidis and staphylococcus aureus.

Biofilms form part of a sophisticated defence mechanism generated by bacteria. They occur in response to challenge from the immune system, antibiotics and mechanical forces. They dramatically improve the bacteria chance of survival.

They occur when the bacteria binds to the surface of a foreign body like dermal filler and then secretes a protective gel which shields the bacteria. what is more, is that over time the collection of bacterial cells Start to behave more like a multicellular organism. They share genes to aid resistance, and may develop specialised metabolic roles that enable the collective to survive better in the challenging environment. It may be that overtime biofilms become increasingly difficult to eradicate as they get bigger and more complex. To make matters more complex biofilms also challenge the belief that in most cases one disease is caused by one organism, as biofilms often form multi-species infections.

TYPES OF SKIN INFECTION

The different layers of skin are also associated with different types of skin infection and different managements. There is an anatomical relationship between the position of the infection and the nature of the infection even if it is the same bacteria causing the problem. Naturally there is significant overlap and a discrete diagnosis is not always possible as infections may have multiple different characteristics.

On the top layer of skin the stratum corneum, the infection appears as impetigo, resulting in crusty lesions with a golden colour. The superficiality of this infection makes it best treatable with topical antibiotics, oral antibiotics are less effective, indicating the need for precise diagnosis.



Erysipelas Affects the next layer down, the superficial epidermis, which results in three key features:

- Very well demarcated borders of infection,
- Raised bumps,
- and a bright 'salmon' red skin colour.



It is usually caused by a beta hemolytic streptococcus, and Lesions can spread quite rapidly and need urgent treatment including IV antibiotics Depending on the presence of systemic effects and severity of infection.

Cellulitis is a deeper subcutaneous infection and has a pink hue with less defined edges. It is very hard to culture but usually arises from *Staphylococcus aureus* or *streptococcus pyogenes*.



Necrotizing fasciitis is an infection that involves the superficial or deep fascia in association with necrosis of tissue, but in practice, this infection tends to involve all layers of tissue including muscle.

Muscle infection can cause Myositis or myonecrosis so this is usually in larger traumatic injuries to limbs. These infections are relatively unheard of in aesthetics and would require urgent hospital treatment with IV antibiotics and surgical involvement.

Overall the most common cause of skin infection is staphylococcus aureus, this bacteria also carries with it a high risk of resistance.

Risk Factors

The overall risk of infection is very low but is increased with patient-specific risk factors which predispose to infection. This includes elderly patients and those in an immunocompromised state, most often due to medication they are taking such as steroids or immune modulators for diseases like Crohn's disease, and those with chronic liver and kidney disease.

SPECTRUM OF INFECTION



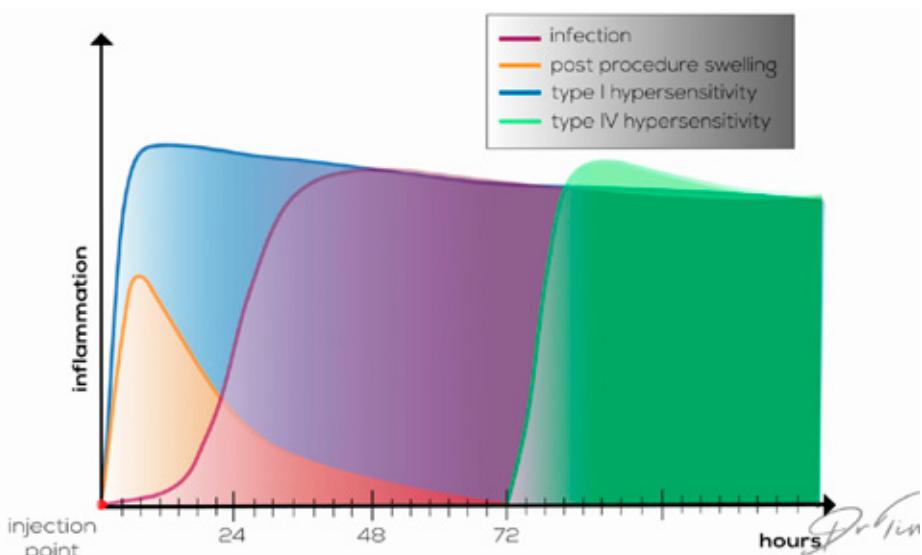
Dr Tim

DIAGNOSIS

The minimum diagnostic criteria are erythema, oedema, warmth, and pain or tenderness. Unfortunately, these symptoms are the same as for any cause of inflammation after a procedure, and so can look similar to both allergic reaction and post-procedural inflammation, but there are timing and qualitative differences that can help you diagnose with sufficient accuracy.

If possible it is valuable to attempt whenever possible to get a culture of the infected area. This becomes increasingly important if first-line treatment does not resolve the problem. You may be able to achieve this through working with the patient's GP and requesting culture is sent via their normal family practitioner, but the reality is most cases do not allow for easy sampling.

The biggest challenge for the clinicians in day to day practice is understanding the differences between immune reaction, infection and normal post-procedure swelling.



Let's take a look at how you can increase the diagnostic accuracy.

The earliest part of the inflammatory response from infection may occur within 2 to 24 hours but it is usually mild and isolated to one specific point on the face. For example, I have experience of several cases where itching and redness at the oral commissure was a clear first sign of an infection.

The first more obvious infected symptoms including pain along with tenderness and redness which may occur within the first week, from

Day 2 to 6, while the formation of abscesses occurs from day 6 to 14.

Normal Post-procedural inflammation has some overlap but tends to peak at 6 hours and then start to reduce. Allergy is more likely to react all over the face in the first 24 hrs and swell wherever the product resides. Infection tends to swell a little later, more gradually and only in one localised place rather than in all sites of injection.

You can see looking at the overlap of these symptoms why it is common to treat for both allergy and infection.

MANAGEMENT

If you suspect an infection it's important to treat early to reduce the size and complexity of the infected site. Different antibiotics are used internationally, but there is considerable overlap. You should follow local prescribing guidelines for acute skin infections.

The most common type of infection would be infection around the dermal filler and the early stages of abscess formation and this would be treated first with antibiotics alone.

In the UK skin infections are treated first with flucloxacillin 500mg QDS for 7 days or if the patient is penicillin-allergic, with clarithromycin 500 mg twice daily.

Second-line treatment includes the addition of penicillin amoxicillin or co-amoxiclav, or if allergic to penicillin, clindamycin 300 mg 4 times daily (Ae group reference).

It is also possible that the infection is more superficial and causes an impetigo type infection.

This is best treated with a topical antibiotic such as fusidic acid.

Erysipelas if mild, could be treated with oral antibiotics but there should be a low threshold for referral to hospital if the borders of infection spread while on antibiotics or there are systemic symptoms such as fever, rigors or signs of sepsis which require aggressive treatment in a hospital setting.

Fluctuant abscesses often require surgical interventions for incision and drainage, as well as antibiotics.



What about Reversal?

If there is an infection triggered by a dermal filler treatment We must always consider the risk that a biofilm will form on the implant and make the infection a lot harder to cure. There is very little data To give certainty about what to do with dermal filler during an acute infection.

The advantage of hyaluronidase is that it will breakdown the dermal filler Removing the foreign body surface the bacteria might hide on, and allow antibiotics to get to the infection more easily.

However, it is also the case that if the infection is severe there may be a small increased risk of spreading the infection into nearby tissues due to the effect that hyaluronidase has on tissue permeability.

To mitigate this risk I do not believe we should ever use hyaluronidase on an inflammatory nodule without antibiotic cover. similarly, however it does not make sense to leave the dermal filler in place if the antibiotics on their own are not able to cure it in the first 7 days. It is my belief that we should use hyaluronidase and the antibiotic cover to reduce the risk of biofilms forming chronic infections that are difficult to treat.

I believe it's reasonable to reverse the dermal filler if antibiotics have not completely cured the infection but continue antibiotics for 1 week further after any reversal to make sure bacteria are fully cleared.

In Summary

If you make a diagnosis of inflammatory or non-inflammatory nodule. If it's inflammatory, Decide if the lesion is fluctuant or not. If there is a small collection you should where possible try to sample it for culture and sensitivities. Larger abscesses may require incision and drainage in a hospital setting.

Start antibiotics and then plan to review the patient on day 7. If the lesion has not fully recovered consider reversal. If reversal is carried out continue antibiotic cover. If the lesion deteriorates it may be wise to add a second line antibiotic to stabilise and then reverse or refer for IV antibiotics.

https://www.researchgate.net/publication/312300131_Overview_and_management_of_fillers_complications

PREVENTION

Prevention of skin infections is first and foremost about client selection.

It's important to acknowledge that medical risk factors are an important factor. Chronic disease particularly liver and kidney disease or poorly controlled diabetes, along with anyone requiring immunosuppression for inflammatory bowel disease and connective tissue disease or chemotherapy. Though not an absolute contraindication, these patients are at an increased risk of infection. Similarly, anyone suffering from an acute infection on the day of anticipated treatment should be declined, Partly because the risk of immune reaction may be higher due to higher immune activity, and partly because there is a potential risk of haematogenous spread of bacteria to the filler, particularly if it's a bacterial throat or dental infection.

Technique

You can reduce the risk of infection further by making sure you assiduously remove makeup and clean the skin thoroughly with antibacterial wipes. you can use chlorhexidine or more recently cleanisept wipes.

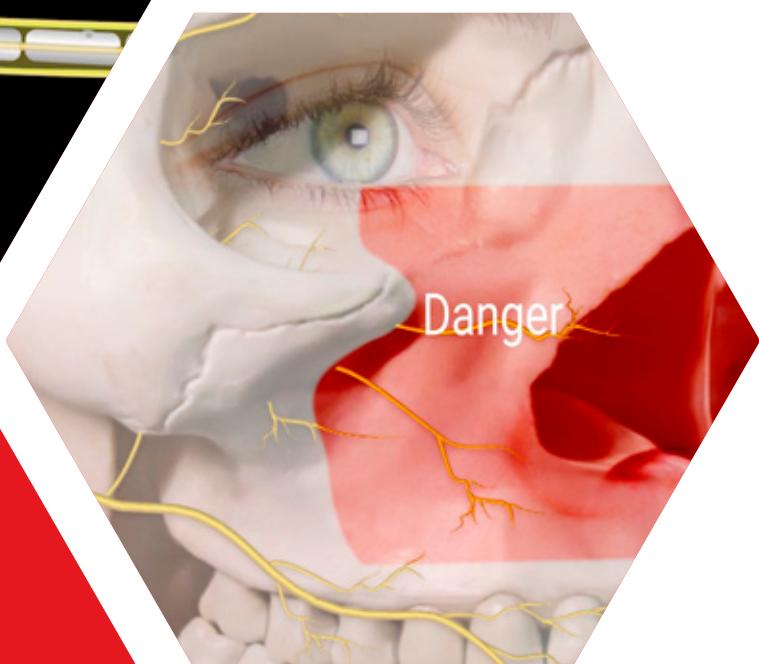
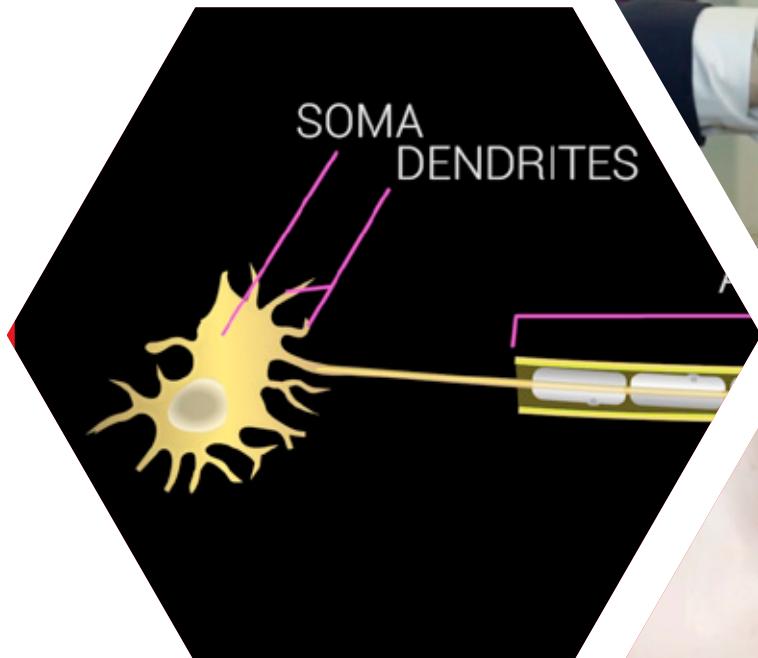
Ensure you clean well around any complex curvatures such as the alar base the nostrils the oral commissures nasolabial folds gonial angle, jawline, lateral canthus, eyebrows and any other hair follicles near the site of injection.

It is my view that the biggest risk for infection are injections around the lips, particularly at the oral commissures where the entry point is often closest to the wet-dry border, especially if there is a deep melolabial fold it may be normal for small amounts of saliva to reach that point immediately post-procedure which would be a significant risk for infection.

After the procedure, it's important to clean the skin again and remove blood spots so that the patient doesn't feel the need to clean the skin again. Ask the patient to wash their hands and advise them not to touch the skin at all for at least 2 hours. Most patients will completely ignore this advice and touch the skin almost immediately unconsciously hence the importance of cleaning hands.

Finally, advise your patient of the signs and symptoms of complications and side effects so that they seek help immediately, Which should minimise the scale of any infection and make resolution easier to achieve.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4450319/>



5

NERVE INJURY

MODULE LINK

<https://drtimpearce.com/modules/nerve-injury/>



Nerve injury in facial aesthetics is thankfully a rare complication, but it is also severely disruptive and upsetting for patients and clinicians alike.

It's vital to understand what can cause this problem, how it manifests and how to diagnose when this is caused by a dermal filler injection and when it is as a result of a medical problem that may need completely different management.

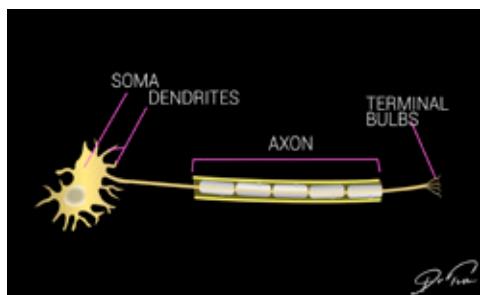
Nerve injury can occur by piercing compressing or tearing and injecting nerves during a procedure. The types of injury that you can cause have been classified in two main ways, and this classification will help you explain to a patient what has happened and what the time course to recovery will be.

Let's first have a look at the anatomy of nerves, the sort you would see in a facial nerve bundle.

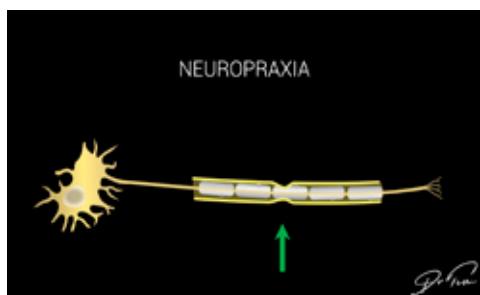
Spinal nerves are surrounded by a sheath called the epineurium, within that sheath there are bundles of nerves called fascicles and each of these is surrounded by a sheath called the perineurium and within each fascicle, we find individual neurons, which are surrounded by a sheath called endoneurium which are often myelinated.

Each nerve consists of an axon a cell body or soma with its associated dendrites and on the other end the terminal bulbs where the neuro-muscular junctions lie.

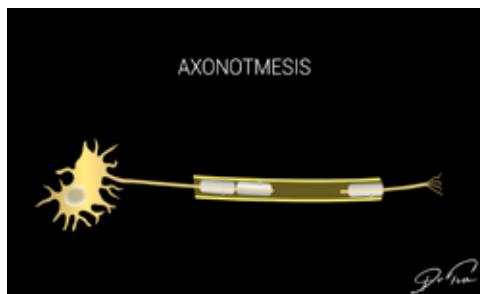
ANATOMY



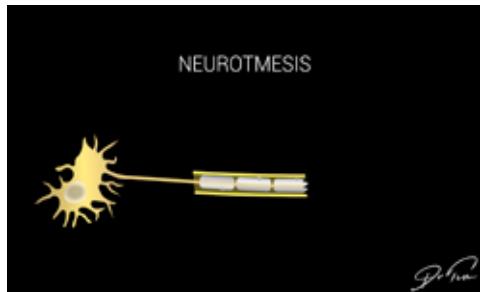
NEUROPRAXIA



AXONOTMESIS



NEUROTMESIS



The Seddon classification describes three types of nerve injury which may occur.

<https://youtu.be/OKr-9WJTHME>

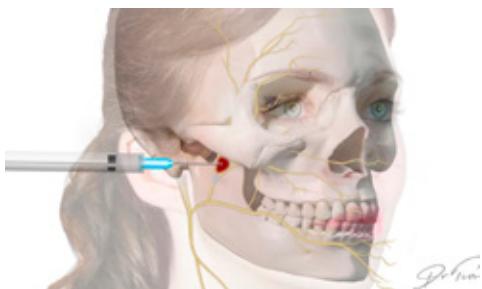
The first type is called neuropraxia This is the mildest form of nerve damage, probably the most common in medical aesthetics and results from compression of the nerve that leaves the axon and the endoneurium intact. This type of injury usually results from dysfunction of the myelin layer, but nerves make a full recovery in a matter of weeks. You could think of this as an electrical wire that has its sheath damaged halfway along and the current is leaking out to earth instead of carrying on to the end of the wire but the wire itself is intact.

The second type of injury is called axonotmesis. In this type of injury, there is not only demyelination but also loss of a section of the axon. Crucially the endoneurium is preserved, which means the neuron can regenerate, the distal end of the nerve will degenerate and recovery takes many months as axon must regrow from the break, all the way to the nerve terminals and this occurs at a rate of roughly 1 mm per day. You could think of this situation as being a bit like an electrical wire with copper inside that has snapped, and the loose end pulled out of the rubber casing, but the casing is broadly intact.

The third and most severe type of nerve injury is called neurotmesis.

In this type of injury we have myelin sheath damage plus axonal loss and then one of three other variations of injury

COMPRESSION INJURY



NEUROTMESIS INJURY



- Damage to the endoneurium, which means growth is possible but slower.
- Damage to the perineurium, which means growth is possible but poor.
- And finally damage to the epineurium, which means no regrowth

Let's look now at the mechanical means by which injury could occur during anaesthetic treatment.

The mildest type is probably a compression injury caused by localised bleeding, dermal filler or inflammation around the nerve and the sustained pressure causing a demyelination injury and neuropraxia. This is probably the most common, as most case reports report recovery in weeks, which would only be consistent with neuropraxia.

Next, you could have an injury caused by the needle passing through the nerve, or a direct injection into the nerve. In most cases, these would likely cause damage to the myelin sheath and probably a section of the axon, therefore causing axonotmesis.

Finally, tearing or completely cutting the nerve with a needle or cannula would cause neurotmesis, to varying degrees severing either the epineurium, perineurium or endoneurium and leaving the patient with a long-term or permanent disability. In my opinion, this would only be likely using a forceful cannula technique or large needle in dangerous areas nearer the origins of nerves.

The resulting symptoms for a patient vary from numbness or paraesthesia, to a distressing loss of muscle control, sometimes affecting all the branches of the facial nerve.

INFECTIVE CAUSES

Aside from mechanical causes of nerve injury, it's also been hypothesized that trauma from the needle could trigger a varicella zoster breakout. Similar to the way injections can trigger herpes simplex activity in the lips. If the varicella zoster virus were to become active in the facial nerve it would cause Ramsay Hunt syndrome, a shingles affecting the facial nerve.

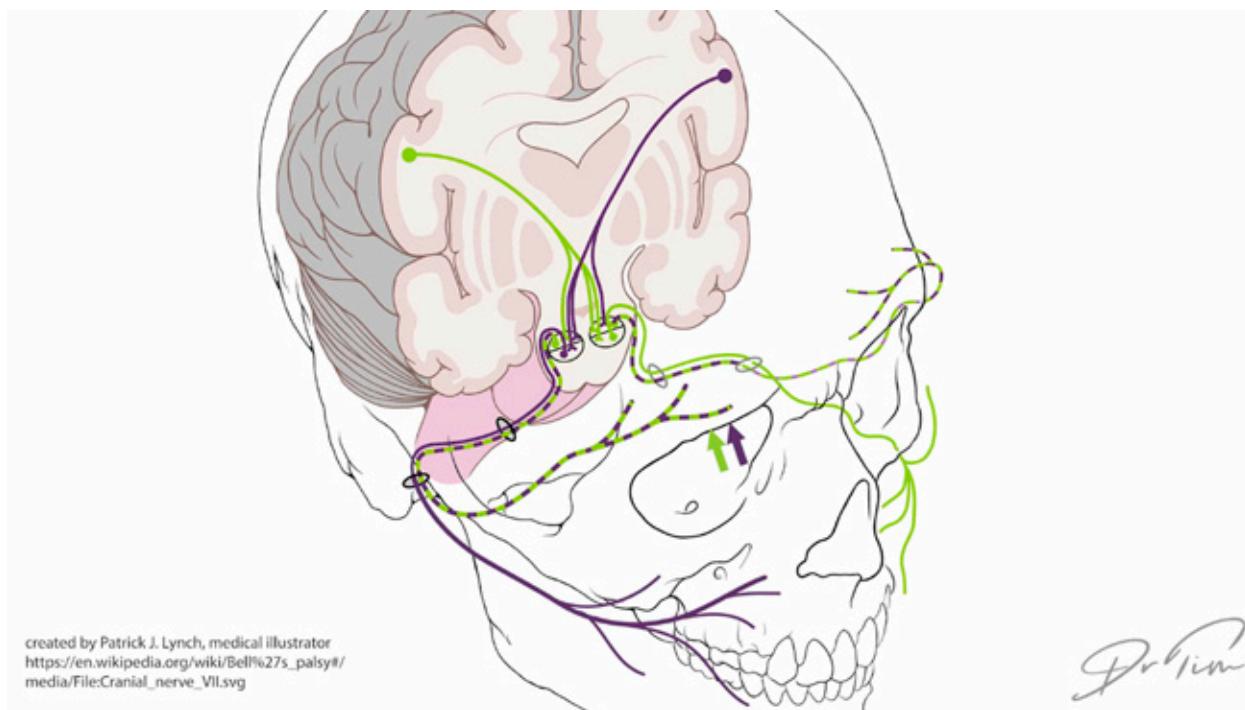
DIAGNOSIS

The patient's presentation will likely involve either weakness or paralysis of groups of muscles in the face or areas of numbness or tingling. If it was caused by the procedure you would expect this to occur either immediately, or within 24 to 48 hours on average during which time inflammation peaks.

When the patient attends it's important to first rule out the most severe cause of facial asymmetry which would be a stroke. In your initial assessment, you should rapidly check for other symptoms so that you can rule out this cause. In the UK we use the FAST algorithm and your patient may already have the first sign, which is face drooping. Next, ask if they have arm weakness or speech difficulty. If you conclude stroke is a likely cause it's time to call an ambulance.

The simplest way to differentiate a stroke from a facial nerve palsy is to look at the forehead. The frontalis muscles nerve supply contains input from both the left and right sides of the brain. Therefore if the cause is a stroke, the forehead will still move on both sides, as the unaffected side of the brain can still cause muscle contraction bilaterally in the forehead but cannot achieve this in the lower face. The result is facial paralysis from the forehead down on one side. In lower motor neuron problems similar to Bell's palsy, the entire side of the face may be affected. Of course, some of our patients may have had botulinum toxin in their forehead which may complicate assessment. If in any doubt it's always best to initiate treatment for the most threatening of the likely differential diagnosis.

Once you have ruled out a stroke, compare their presentation if it involves muscle movement with the pictures you took beforehand, and carefully document muscle function. The use of video is invaluable in the situation. by carefully documenting facial movement you can monitor progress in much more detail.



It's important to examine muscular function across the whole face including the ocular and periocular muscles.

The facial nerve also includes sensory and parasympathetic functions in its terminal branches. If it is traumatized there may be associated loss of sensory function near the ear at the concha and the auricle, lost taste sensation on the anterior two-thirds of the tongue as well as a decrease of activity in the salivary glands, mucous glands in the nose and importantly lacrimal glands supplying the eye on the affected side.

Once you have carefully explored the pattern of signs and symptoms you should attempt to relate them back to the procedure that you carried out by considering the anatomy.

It can be very difficult to be certain that the nerve symptoms your patient's report are due to the procedure that you carried out. The two most obvious connection is the time at which symptoms occurred relative to the procedure and the area treated.

- In the case of a neuropraxia or mechanical cause, you would expect symptoms to arise in a matter of hours but not immediately.
- In the case of axonotmesis you would expect a much more rapid onset of symptoms as the axon itself has been damaged.

Similarly, you would expect neurotmesis to occur immediately, as much of the nerve has been severed.

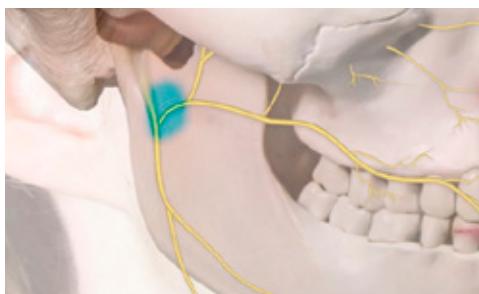
If the cause was the procedure triggering an infection, you would expect a delay in symptoms as the infection became more active, hypothetically 24 to 48 hours after the procedure at the soonest and up to one week after the procedure.

Beyond this time it's reasonable to consider that any signs and symptoms of nerve injury are unrelated to the procedure and you should be on the lookout for signs of viral infection more consistent with classical Bell's palsy.

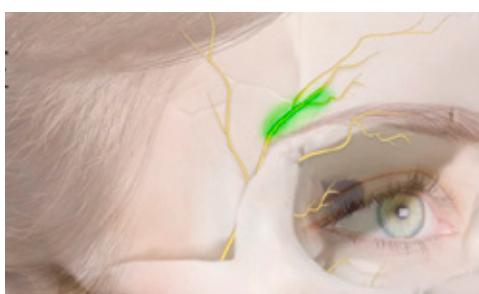
PREAURICULAR AREA



BUCCAL BRANCHES



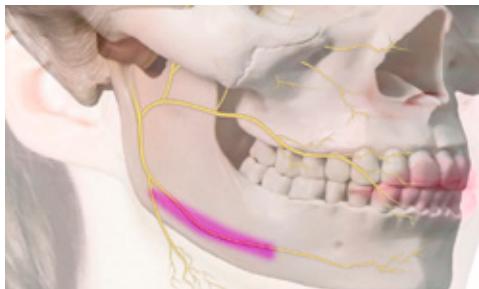
TEMPLE BRANCHES



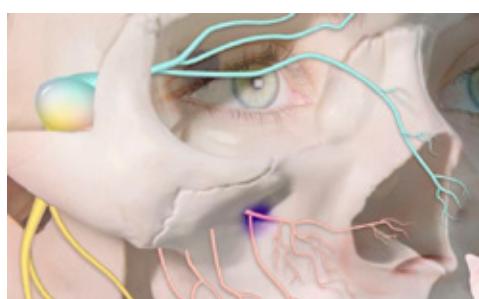
Consider the anatomy and the places on the face that are most vulnerable to nerve injury. I have identified the following areas to be highly cautious.

- 1.** The preauricular area where the facial nerve emerges under the zygoma. This area is closest to the root of the whole facial nerve and in one case report was seemingly traumatised by cheek injection the past inferior to the zygoma and triggered a Bell's palsy 8 hours later that lasted for 6 weeks
- 2.** Masseter injections of botulinum toxin have also been shown to be a risk for traumatising the buccal branches of the facial nerve which can cause right-sided paralysis of the zygomatic and buccinator muscles causing an obvious upsetting asymmetry during smiling.
- 3.** Temple and lateral forehead injections with a cannula are a potential risk for trauma to the temple branches which innervate the frontalis and orbicularis oculi. I have seen no recorded cases there is a machine that relies on this method to cause anaesthetic effect. sometimes known as frotex a machine that cools the nerve down causing a controlled neuropraxia for 3 to 4 months has been marketed, and to get it that affect you place the probe superior lateral to the orbital rim and cool that area for a few minutes. This is an area that is sometimes associated with cannula treatments to revolumise the lateral part of the forehead, and trauma to this nerve may cause neuropraxia affecting the muscles innervated.

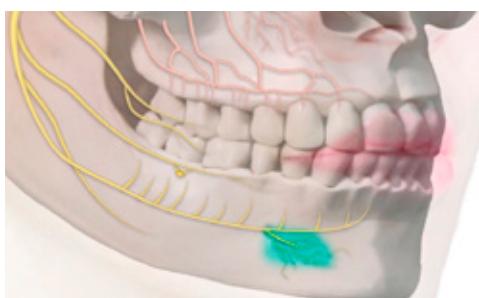
MARGINAL MANDIBULAR BRANCH



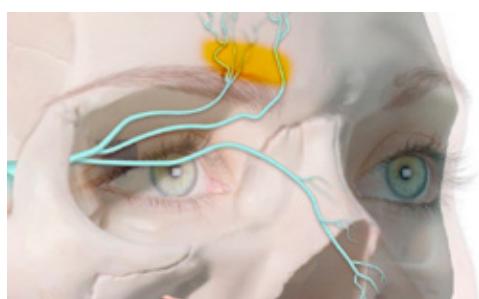
INFRAORBITAL NERVE



MENTAL NERVE



SUPRATROCHLEAR AND SUPRAORBITAL NERVES



4. Inferiorly, treatments of the jawline and chin could be a risk for trauma to the marginal mandibular branch of the facial nerve, though the muscles supplied are relatively small including the mentalis muscle.
5. In the literature, the infraorbital nerve is most commonly reported as a site of injury. I wonder if this is most likely because it is a target of infraorbital nerve blocks. Aesthetic injections should always be avoiding this area as it is also a risk for arterial occlusion. Trauma to the nerve would cause a sensory deficit affecting the lower eyelid, upper lip and part of the nose
6. The mental nerve may also be vulnerable to either injection or cannula around the chin and injury to this nerve would disrupt sensation to the chin lower lip labial gingiva of the mandible and the anterior teeth and premolars.
7. The supratrochlear and supraorbital nerves may be vulnerable to trauma in rare instances from botox injection or cannula used around the orbit. Injury would result in paraesthesia to the forehead but not paralysis.

MANAGEMENT OF NERVE INJURIES

Sensory injuries are beyond our influence in terms of recovery. Advice could be given to protect the face if the areas of paraesthesia are at greater risk. Advise your patient to be careful of hot foods and drinks in particular. thankfully most injuries will be unilateral and the practical risk is probably not that high of inadvertently burning yourself because of half your lip being anaesthetised. it is mainly an irritation, and reassurance that recovery is most likely in a few weeks or months is all that is required.

MANAGEMENT OF MOTOR NERVE PALSY

A lot can be learnt for the management of Bell's palsy, and we will look in detail at a case I saw in 2018 that resulted in a Bell's palsy from a dermal and filler injection of the cheek.

It is true that in most cases Bell's palsy resolves without treatment. our goal, of course, is to minimise the length of time the patient suffers the distressing symptoms of hemifacial paralysis.

The primary goal of active management is to decrease pressure on the nerve.

Pressure may be due to inflammation, or the product itself.

In the case of a dermal filler injection, it may be reasonable to reverse the procedure if you are concerned the dermal filler may be compressing the nerve. Hyalase in the area of question using a low trauma technique is reasonable. It may be that you rely on the ability of hyaluronidase to perfuse through to the area of the nerve rather than place and instrument in the area again, risking further trauma.

It is possible to decrease inflammation most effectively using a steroid such as prednisolone. this is a strategy borrowed from classical Bell's palsy management, where the dose is high, usually around 60 mg of prednisolone daily for a week followed by a reducing dose over the next week.

It is reasonable to include an antiviral at the same time just in case you were dealing with a viral-induced Bell's palsy that happened to occur at the time of your procedure. If you are treating with prednisolone you may make this problem worse if you do not also add acyclovir and it transpires that the symptoms were in fact and early stage of a classical Bell's palsy, made worse through immunosuppression alone without antiviral prophylaxis.

The second element of acute management particularly when treating a Bell's palsy or any nerve damage that affects the function of the eye

is to protect the eye from complications caused by not being able to blink or close the eye, or by the associated decrease in tear production.

Regular eye drops should be used throughout the day and at night and an eye patch should be applied so that the eye can be kept closed while sleeping. referral for specialist eye care is also a possibility if there is an increase in ocular symptoms such as pain or inflammation caused by dehydration.

Beyond the acute stage if you have a patient with Bell's palsy I would recommend referral to a specialist as rehab is a speciality in itself and you want to maximize the chances of your patient making a full recovery.

I include for your information useful video links below on how to manage Bell's palsy in the various stages that it goes through. If your patient has a new ataxia that is more isolated all of these principles would still apply and you may find this extremely helpful to manage them in the medium term. I still believe that even if you refer your patient on to a specialist, this knowledge can be useful to share with them so that they feel fully supported and in safe hands.

Below are several useful links that will give you all the information you need to understand the management of facial palsies and advise your patient while they await or undergo specialist care.

There are different stages of recovery, a 'flacid' phase and 'tight' phase with different exercises.

Below are links to the specialist exercises that may improve recovery

There are two stages to a recovering in Bell's palsy, a 'Flaccid' Stage' and a 'tight' phase, with different exercises required at each phase.

<https://www.youtube.com/watch?v=DI1vg-yYozM&list=PLxSoy1PlqBcZKdLXyBtJ6xZB80zWXB4rP>

Tight Phase- for three months later if required in conjunction with ENT specialist.

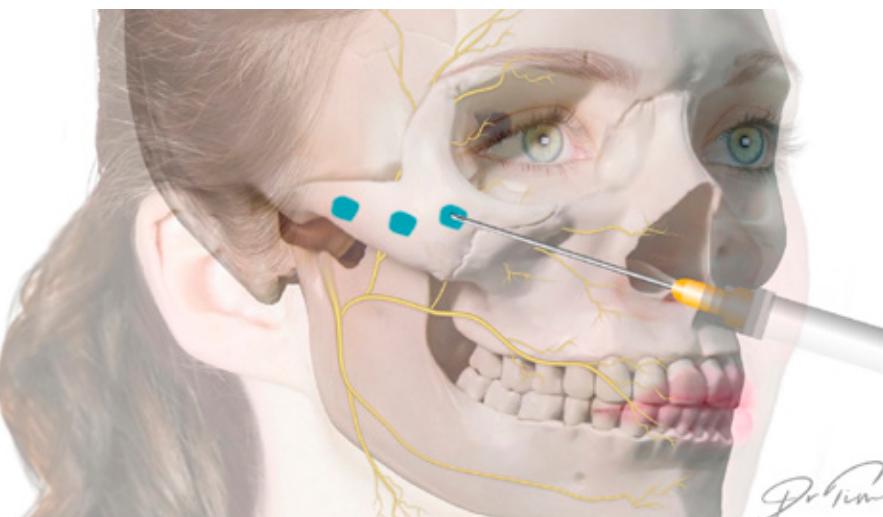
<https://www.youtube.com/watch?v=wFYFXgQHgcA&list=PLxSoy1PlqBcadbB0mr-C3YTIXNLppBzJd>

AVOIDANCE

To break down how to avoid nerve injuries will look at each part of the face and where they are a potential risk and discuss injection technique.

The highest risk area is the preauricular area with a facial nerve emerges into the face.

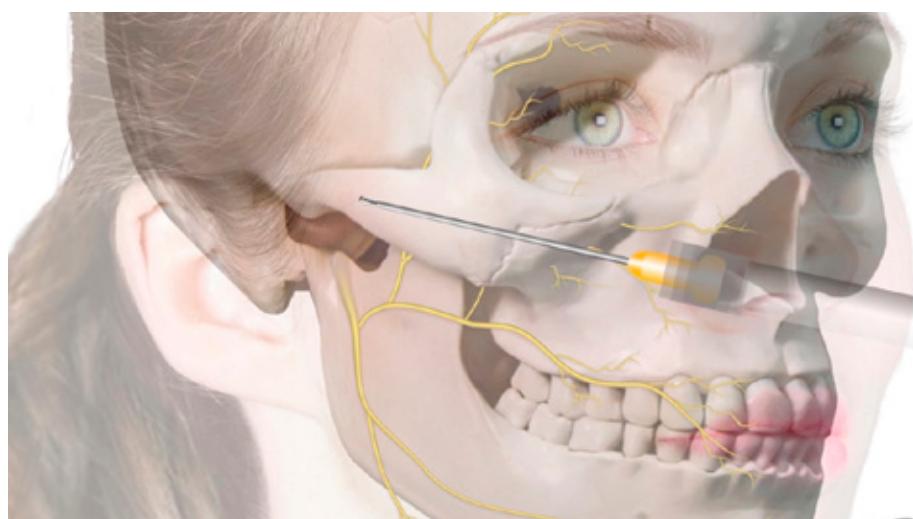
This area could easily be damaged while injecting cheeks. If you use a cannula, you're likely in a more superficial plane in layers of fat and risk the nerve is much less likely.



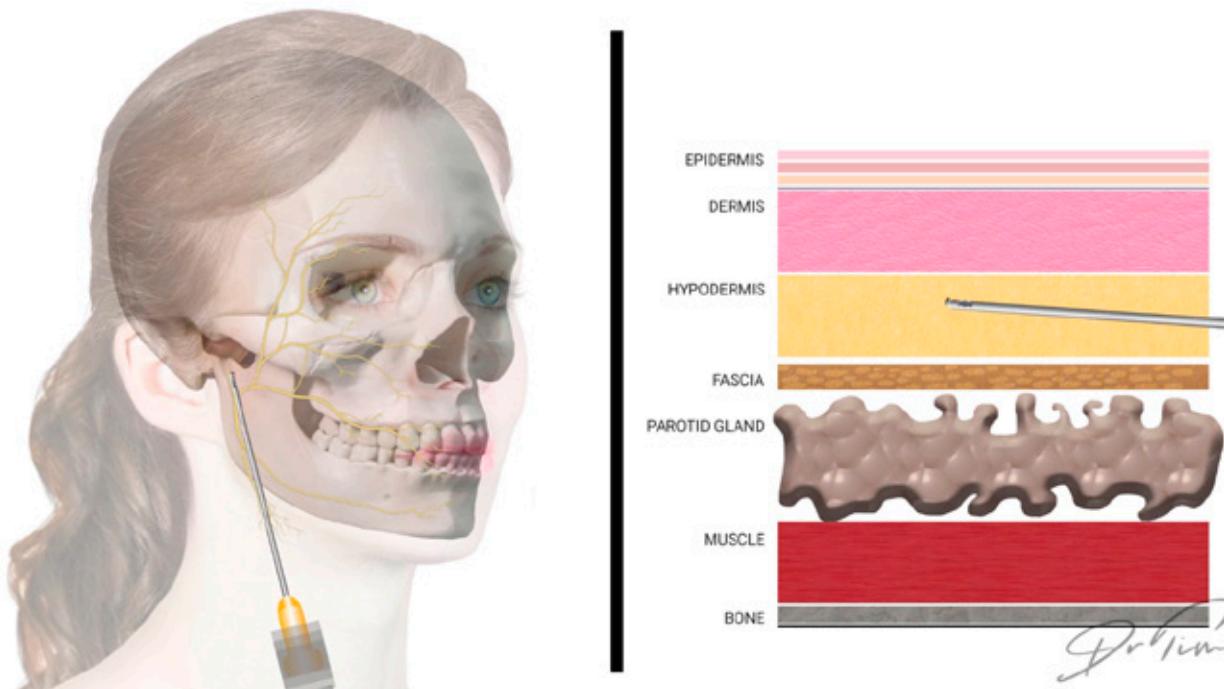
However this technique may not produce as stable a result as using a needle to place dermal filler on the periosteum of the zygoma. This commonly taught technique is a well established way of augmenting cheeks, however it is quite common while inserting the needle aiming to touch periosteum to find that you either pass underneath or above the zygomatic arch and are then placing your needle directly where facial nerves are likely to reside.

To reduce the risk we must build this knowledge into a technique and start first of all by becoming better at identifying the art of the zygoma.

I find it very helpful to palpate carefully the superior and inferior border of the zygoma with my thumb and forefinger before choosing where to inject. I also find that holding the needle parallel with the transverse plane as the arch can make it easier to guide the needle to its desired destination on the periosteum. If you were to inject with a slightly angled needle pointing inferior or superior to the transverse plane it is easier to miss the bone and pass into the danger zone where the transverse facial artery maxillary artery and facial nerves reside.

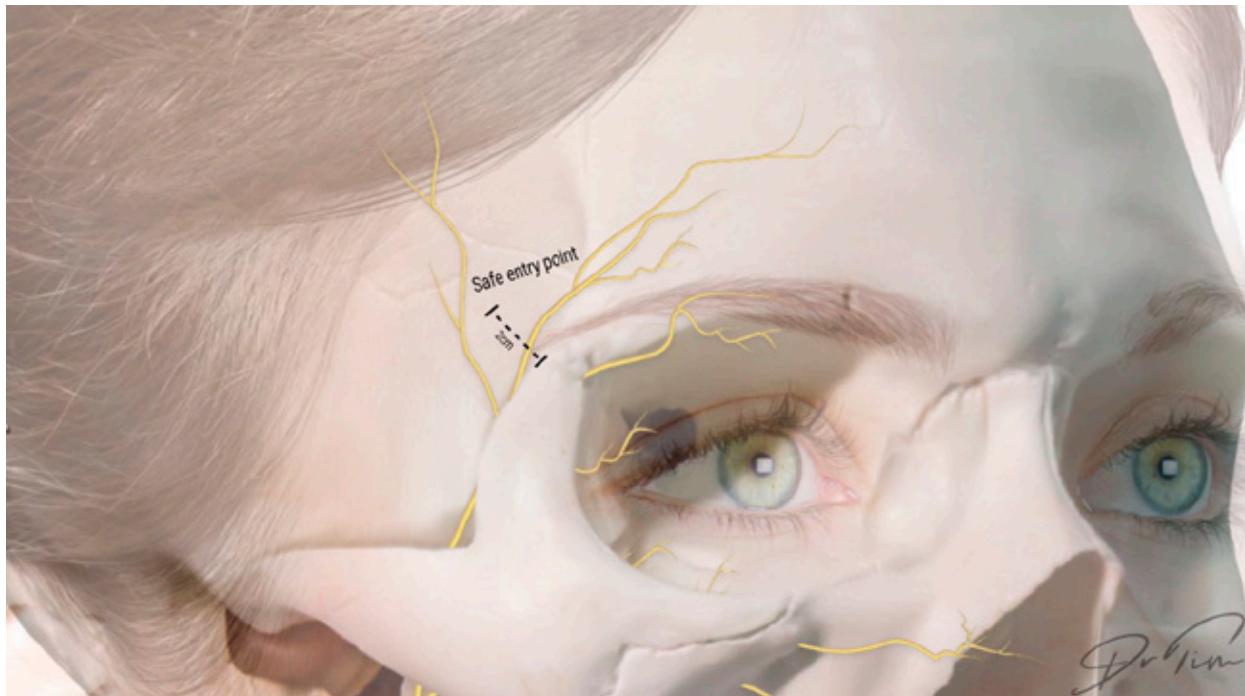


When injecting inferior to the zygomatic arch, for example when replacing the lateral fat pad as part of a jawline restoration, it is imperative to use a cannula and stay in the hypodermic fat carefully angling your cannula so that it does not go too deep and risk damaging structures under the zygoma.



When treating the midface avoid the infraorbital nerve by always staying lateral to the mid pupillary line when using a needle, and stay in the fat more superficially when using a cannula and crossing the mid pupillary line.

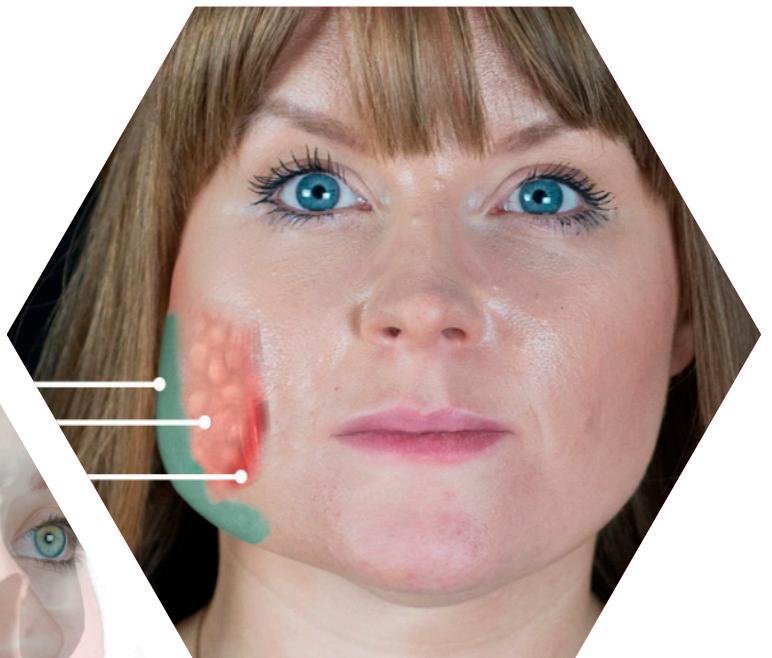
When treating the temple and forehead minimise the number of injections in the temple preferably to 1, and when using a cannula in the forehead do not make your entry point Too close to the periorbital ridge where the nerve off and lies. enter 2 cm above this zone, and always be gentle and careful with cannulas as you pass them through the structures.



The mental nerve can be avoided by palpating for the foramen and never injecting deep at this point, and by careful gentle placement of cannulas whenever treating the chin or jawline.

In summary

Nerve injuries are a rare but important side effect. Consenting your patients is vital, but an awareness of the anatomy and your injection technique is really the key to limiting the chances of being confronted with this problem. If it ever does occur you should from the history be able to initiate the correct management and appropriately reassure your patients.



6

SALIVARY GLAND INJURY

MODULE LINK

<https://drtimpearce.com/modules/salivary-gland-injury/>



With the recent increase in frequency and popularity of chin and jawline restoration and augmentation, there has been an increase in salivary gland injuries which occur during the procedure.

When passing a cannula along the jawline, particularly over the masseter muscle and near the inferior border of the mandible, the instrument may become involved with the surface of the parotid and submandibular glands, placing it at risk of injury from tearing and penetrating forces.

The outcome from these injuries can be perplexing to the uninformed injector. So in this module will look in detail at what causes this injury what the symptoms and management are and how you can avoid it.

PATHOGENESIS

Let's consider how these injuries may occur.

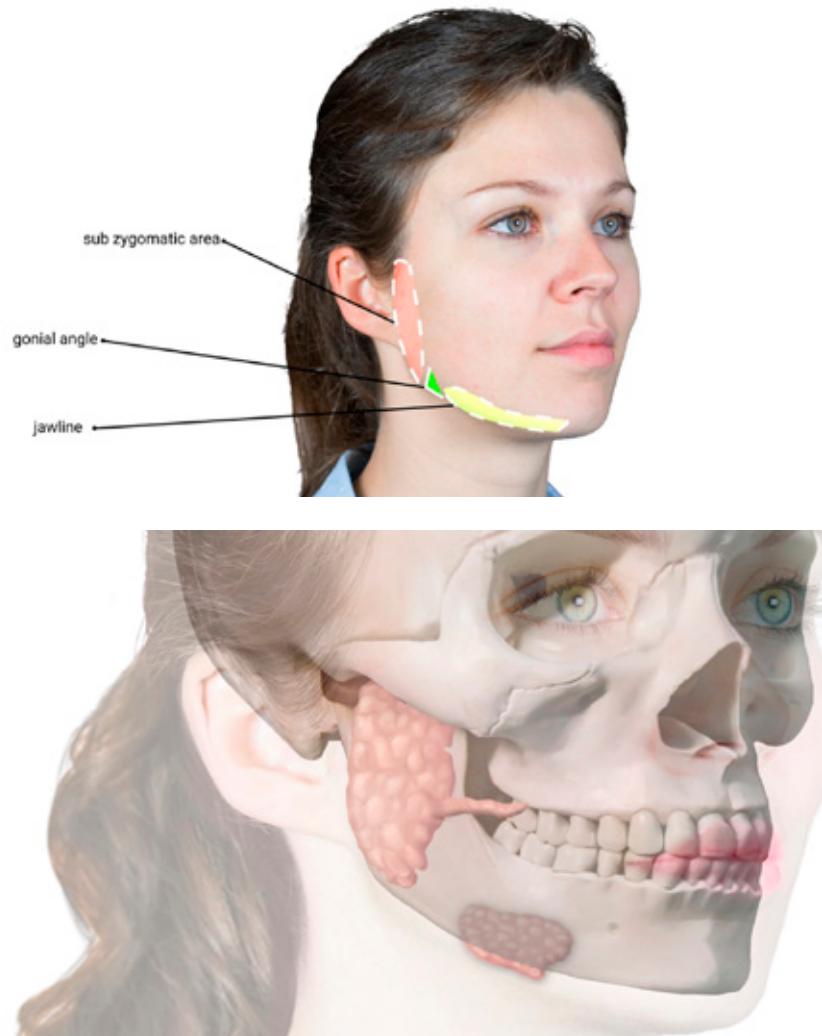
I believe this injury occurs more often using a cannula because the areas treated tend to be cannula only areas, and cannulas often expose structures to shearing forces as they are pushed through tissues.

The glands are vulnerable when treating the subzygomatic area, the gonial angle, and jawline.

Let's consider the anatomy

The parotid is the largest of the salivary glands. 75 % lies over the masseter muscle and 25% behind the ramus of the mandible.

The parotid fascia covers the glands and extends into the neck over the platysma surface, as an extending layer of the deep cervical fascia in the neck.

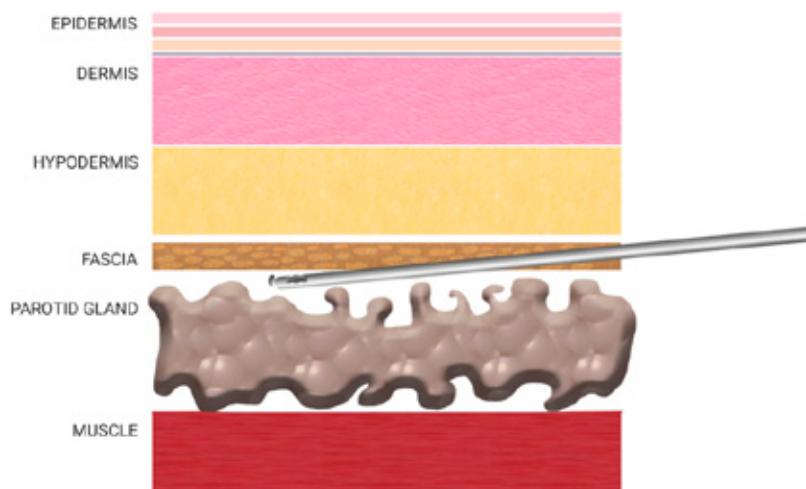


Above the fascia is the hypodermic fat.

The passage of the instrument should be in the hypodermic fat, but this lies directly adjacent to the layer of fascia covering the surface of the gland.

You can see therefore that the parotid and submandibular glands would be particularly vulnerable if the instrument was to penetrate a little too deep and then lie in the plane between the fascia and the gland. During its movement, the instrument may snag on the surface of the glands causing trauma to its surface. In more severe injuries, it may be possible to damage the parotid duct and the branches of the facial

nerve which passes through the gland. Fortunately, these structures are deeper within the gland and an experienced injector should know they are in the wrong place before they went that deep. What we are left with are traumas that occur because we are only a few millimetres too deep, and behind this fascia instead of in front of it.



Tears to the surface of the glands may cause leakage of saliva into the tissues causing painless swelling that peaks and resolves a few hours after eating. Salivary gland injury, in general, may be associated with cysts which form, called Sialoceles. These occur as a result of the accumulation of saliva that is not drained intraorally. More rarely still, Sialoceles can result in the formation of fistulas which leak out of the skin. Though this is a known complication of trauma to the salivary glands, it is not a reported complication of dermal filler as yet.

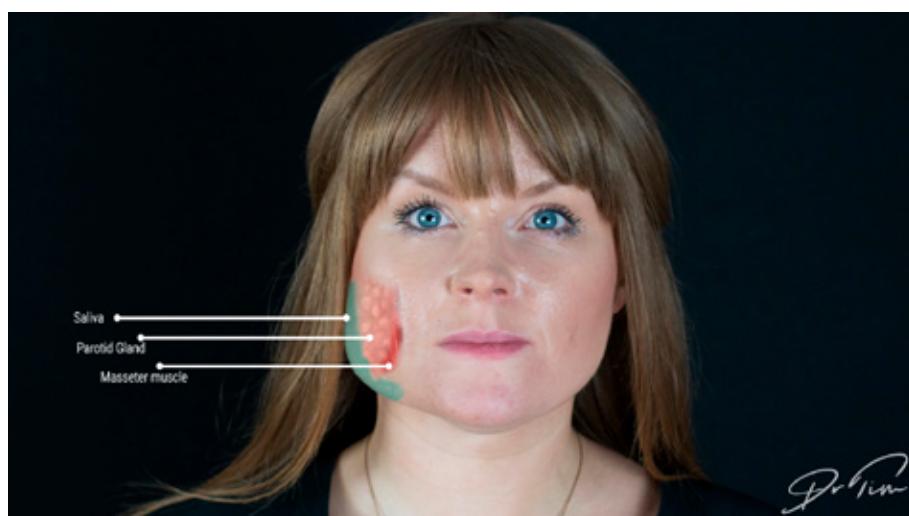
DIAGNOSIS

The diagnosis of salivary gland trauma is often difficult the first time you come across it.

The patient usually presents with swelling of various degrees over the parotid gland, or the angle of the jaw or neck. This is often consistent with a normal post-procedure inflammatory side effect.

- There is usually minimal pain but maybe a general achy feeling around the area.
- The swelling sometimes feels fluctuant, or if under a layer of fat can feel diffuse.
- Differential diagnoses are actually the most common side effects we see: swelling and bruising.
- There are several subtle differences which illuminate the true cause of the problem.

On examination, you should notice the area is painless, non-tender and there is an absence of bruising. These are the key aspects which clinically differentiate the pathology from a haematoma or inflammation.



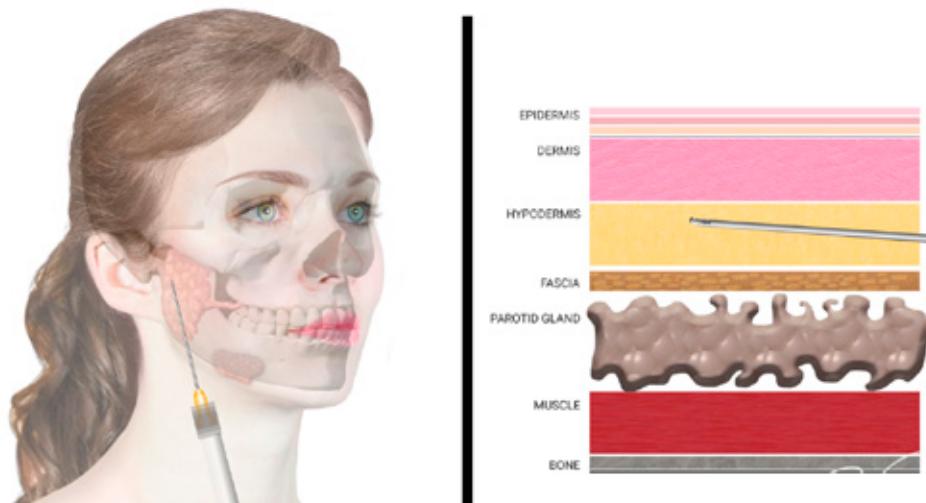
In some cases, the diagnosis becomes more obvious when the history reveals that the swelling noticeable worsens before and during eating, and then subsides between meals. Swelling usually subsides fairly quickly after eating and returns to a baseline level within a few hours as the saliva is reabsorbed or drains away.

We can now tell what has likely occurred. The instrument has penetrated beyond the subcutaneous fat through the parotid fascia. While passing along this space it has created tears in the surface of the gland. During salivary gland stimulation, saliva will spill out of the

gland to fill the potential space between the gland and the fascia. The layers of fascia cause it to collect in key areas, before being slowly reabsorbed.

MANAGEMENT

For the vast majority of people, the situation can be managed conservatively. It may be wise to avoid foods that cause excessive saliva production, salty or very sweet food and not to graze but to eat in discreet meals.

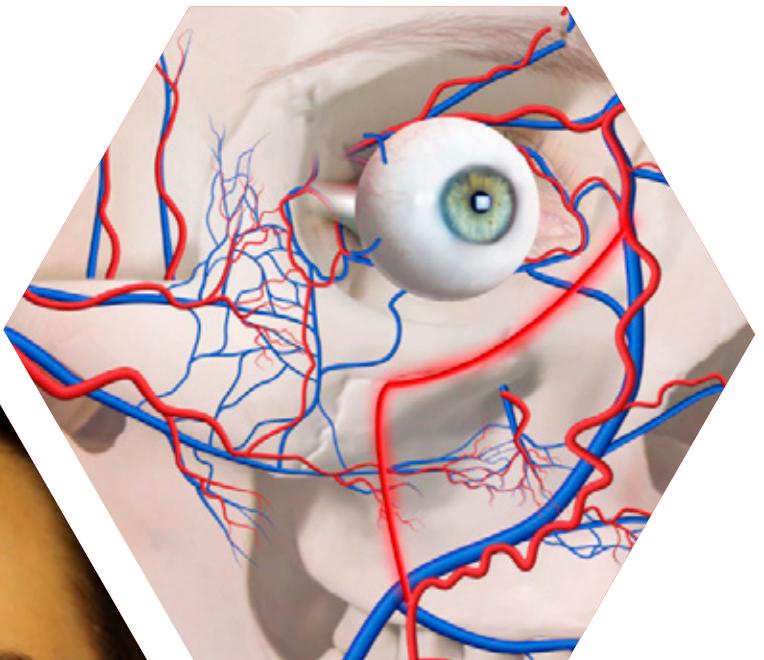


In cases of trauma to the gland, surgeons recommend a pressure dressing for 48 hours to minimize the risk of sialocele formation. This is probably unlikely for non-surgical trauma, but it could be advisable in certain cases. Certainly, if there is only mild swelling during eating it may be beneficial to apply pressure while salivary gland production is increased to reduce leakage of saliva into the tissues. This may reduce the continuous reformation of tracts and facilitate the closing of perforations more quickly.

Recovery occurs naturally in a period of a few days but can take longer. If the problem gets worse or does not recover it may be appropriate to refer for a specialist opinion, though this should be exceedingly rare.

You can prevent this injury from occurring through careful and gentle placement of the cannula when doing procedures near the parotid and submandibular gland.

Cannula in the fat should move fairly easily and be mobile at the tip. Once under the parotid fascia, it may be less easy to move, with a sensation that more force and more associated trauma is occurring. In these cases, it is better to pull the cannula out and attempt to re-enter in a more superficial plane. It is also useful to angle the cannula in such a way that it is skirting underneath the skin rather than naturally diving deeper into the underlying gland.



7

BLINDNESS

MODULE LINK

<https://drtimpearce.com/modules/blindness/>



These guidelines are only intended to be interpreted by degree qualified healthcare professionals. They are not intended to replace clinical judgement and it is important that the practitioner makes the correct diagnosis and works within their scope of competency. Some complications may require prescription medicines to help in their management and if the practitioner is not familiar with the medication, the patient should be appropriately referred. Informing the patient's General Practitioner is considered good medical practice and patient consent should be sought. It may be appropriate to involve the General Practitioner or other Specialist for shared care management when the treating practitioner is not able or lacks the experience to manage the complications themselves. Practitioners have a duty of care and are accountable to their professional bodies and must act honestly, ethically and professionally.

INTRODUCTION

Blindness from dermal filler injections is a rare but highly significant event, first documented in 1963 when a patient suffered visual loss after injections of steroid into the scalp while treating alopecia. This situation is obviously one of the most terrifying complications any injector could face, and so we must understand it in as much detail as possible, in order to develop the safest possible injection strategies, give the best advice to our patients, and the best response if the worst does occur.

PATHOGENESIS

To understand how blindness can occur after a dermal filler injection we must first look closely at the structure of the eye. We must consider the arterial blood supply and how it connects with the blood supply of the face. As you will see as we progress and look at particular cases, visual loss is theoretically possible in nearly a dozen different ways.

ARTERIAL ANATOMY

The central retinal artery is the primary blood supply to the retina. It branches off the ophthalmic artery and runs inferior to the optic nerve within the dural sheath, towards the eyeball.

It pierces the eyeball just near the optic nerve and then sends branches onto the internal surface of the retina, supplying all of the retina except for the fovea, which gets its blood supply from the posterior ciliary arteries via the choroid layer.



Both the central retinal artery and the posterior ciliary arteries Originate from the ophthalmic artery.

The ophthalmic artery is also connected with 3 other major vessels of the orbit- the supratrochlear, supraorbital and the lacrimal artery, and posteriorly to the Internal carotid artery.

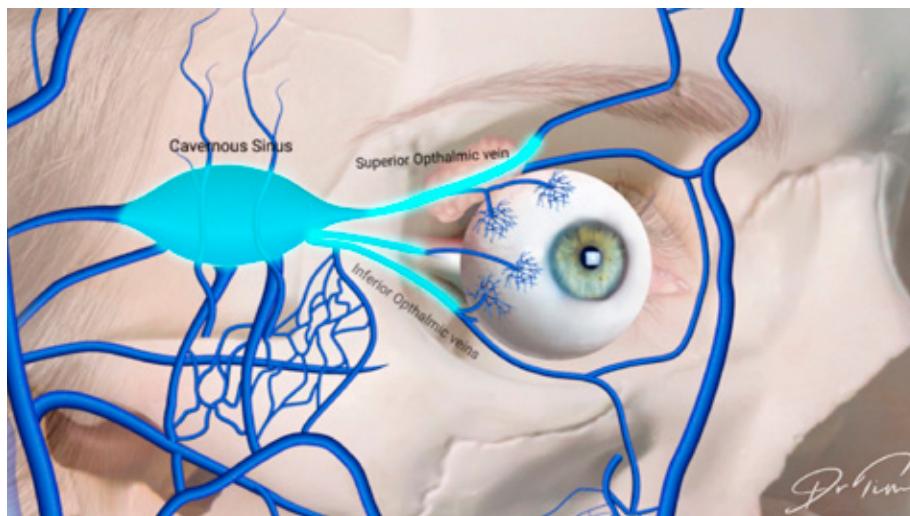
Hypothetically, dermal filler entering any of these vessels could affect the blood supply to the eye, but as we will see the frequency of such events is dictated by the frequency and proximity of dermal filler injections to this network.

VENOUS ANATOMY

It's also helpful to understand the venous drainage of the eye as this can also be relevant to the function of the retina.

Venous drainage into the cavernous sinus is via the superior and inferior ophthalmic veins. The ophthalmic veins, in turn, drains usually 4 vorticos veins which drain the posterior quadrants of the retina.

It's possible that blockage to any of these vessels may have a detrimental effect on the function of the retina.



ARTERIAL PATHOLOGY

Most of the literature on this subject comes from studying atherosclerotic disease in the eye which causes infarctions and embolisms as a result of plaques forming in the vessels.

There are 6 established subtypes of injury that may occur to the arterial supply and several variants of venous occlusion in the eye that are also known to cause blindness.

Let's look in detail at the types of injury that may cause visual loss.

The first is ophthalmic artery occlusion.

This is the most severe occlusion not affecting the central nervous system, as this vessel supplies all the other vessels in the orbit, and therefore also the muscles of the eye and entire blood supply to the globe and all parts of the retina.

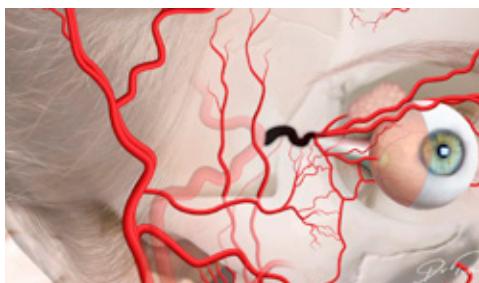
The second variant is generalized posterior ciliary artery occlusion with relative central retinal artery sparing.

The macula is the only part of the retina not supplied by the central retinal artery but by the posterior ciliary vessels, so this type of occlusion may affect the Macula, causing loss of the most central and acute aspects of vision but sparing of peripheral vision.

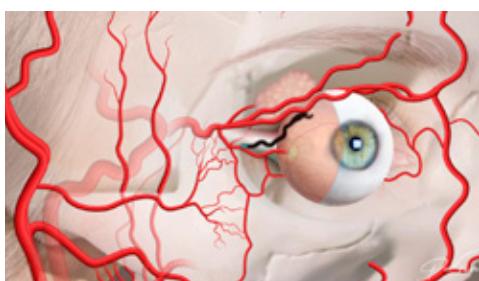
The third is the type of pattern we all expect: Central retinal artery occlusion (CRAO)

Central retinal artery occlusion affects the blood supply of the entire retina except for the small area at the macula supplied by the posterior ciliary artery.

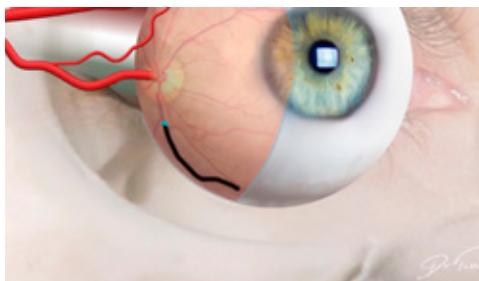
OPHTHALMIC ARTERY OCCLUSION



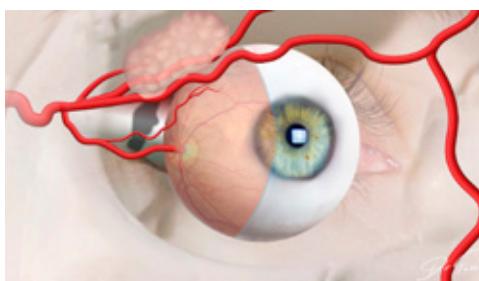
POSTERIOR CILIARY ARTERY



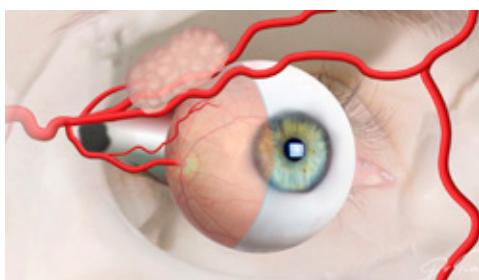
BRANCH RETINAL ARTERY OCCLUSION (BRAO)



ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AION)



POSTERIOR ISCHEMIC OPTIC NEUROPATHY (PION)



Next is a branch retinal artery occlusion (BRAO) is caused by emboli affecting only one of the branches of the central retinal artery and causing partial peripheral visual loss.

Anterior ischemic optic neuropathy (AION) is caused when the blood supply damaged is to the anterior part of the optic nerve, at the optic disc, causing variable degrees of visual loss depending on the amount of damage.

Finally, posterior ischemic optic neuropathy (PION) is caused by damage of the blood supply to the posterior aspect of the optic nerve.

VENOUS PATHOLOGY

Retinal damage from blockage or compression of the vorticose veins or their branches are also a potential cause, as these vessels drain the posterior quadrants of the eye.

These can be divided into branch retinal vein occlusions which occur due to blockage of one of the four retinal veins which each drain a quadrant, or Central retinal vein occlusion is due to blockage of the main retinal vein, and cavernous sinus occlusion which drains blood from the whole retina. In general, visual loss is more severe if the central retinal vein is blocked.

In 2012, a case was reported of blindness and orbital infarction after injection of poly-L-lactic acid into the temporal vein. It has connections to the cavernous sinus through the periorbital veins. This vein lies between the deep and superficial temporal fascia in the temporal fat pad in the temporal fossa just above the zygoma.

The different types of occlusion cause different patterns of injury and these have been classified as Type I, II, III and IV. The classification is a shorthand for blindness that occurs in isolation which is a Type I injury, blindness with corresponding damage to the levator muscles of the eyelid which is Type II, blindness with injury to the ocular muscles causing ophthalmoplegia which is Type III, and finally, Type IV which with both eyelid ptosis and ophthalmoplegia.

Let's look now at the reported cases and what we can learn from them about the risk factors.

In a 2015 review, 98 cases were analysed.

It's apparent that there are many variables which correlate with the risk of blindness.

- The anatomy of the face and how it relates to the eye.
- The area injected
- The type of product injected
- The volume of product injected
- The technique used to place the product.

The only one which we are not in complete control of is the anatomy, so we must adjust all the other variables around the anatomy.

Let's consider first how and why the anatomy correlates with the risk of blindness.

39% of cases were as a result of injections in the glabella

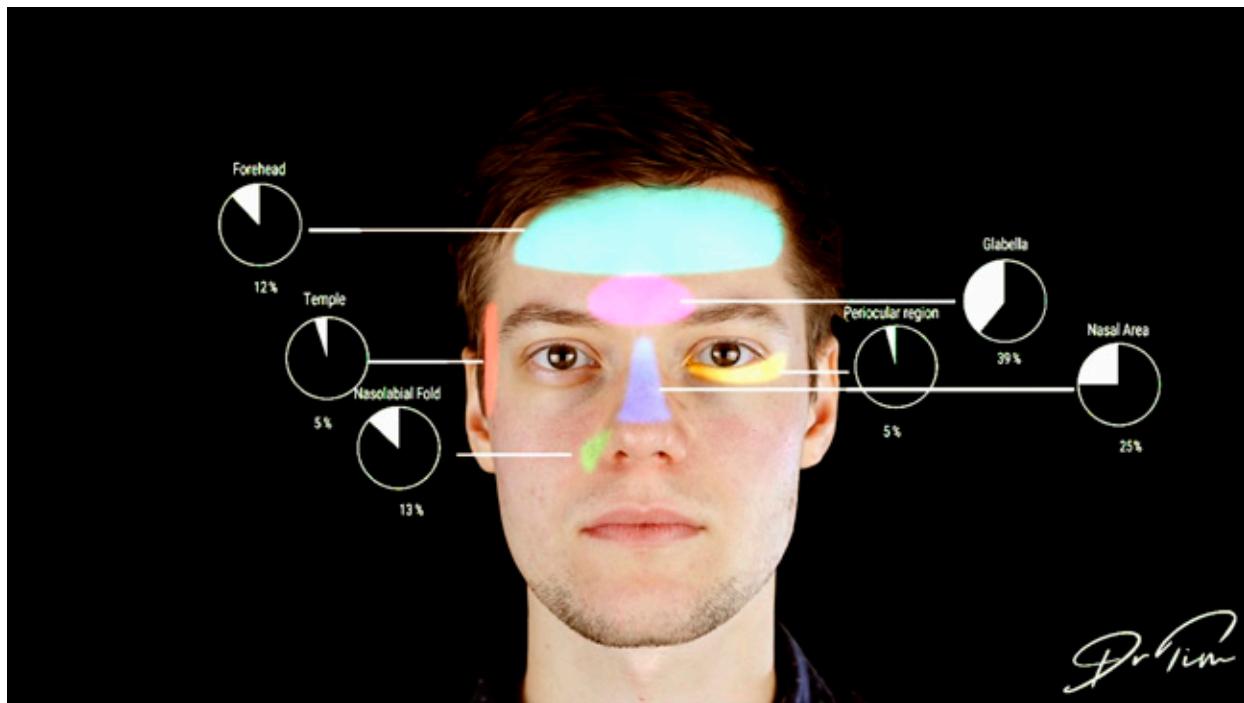
25% in the nasal region

13% in the nasolabial fold and

12% in the forehead.

5% in the temple

6% in the periocular region



It's important to remember that these percentages are not necessarily related to the relative risk of injecting in each area. It is not possible to tell from this subset of data whether or not differences in percentages represent a difference in the frequency that each area is treated or the vulnerability ocular blood supply at each area. We can only assume that there is a risk in all of these areas of the face.

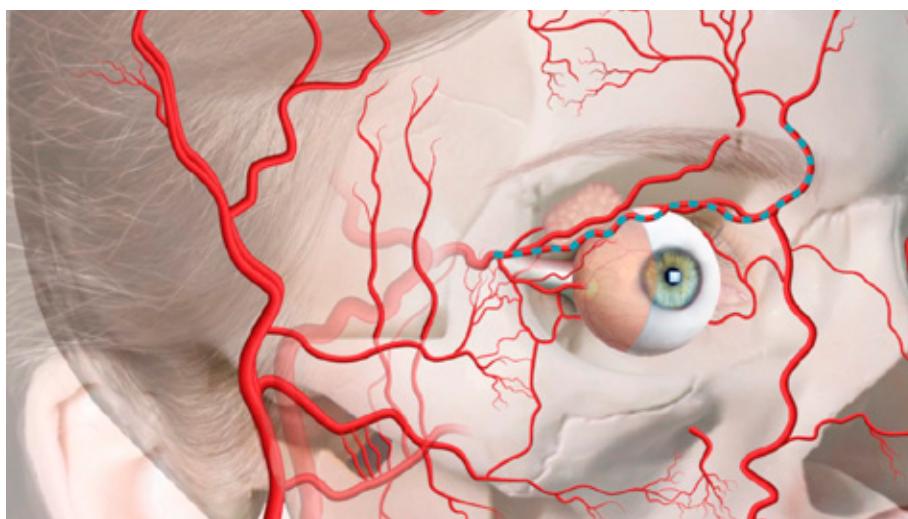
Let's consider how blindness occurs as a result of treating each location.

THE GABELLA

This area is treated with dermal fillers to reduce creases caused by frowning and sometimes to create a more prominent supraorbital ridge.

Studies have conclusively shown that the crease that forms along with the glabella is almost always closely associated with the position of the supratrochlear archery.

Just a few millimetres of hypodermis and muscle separates the dermis from the supratrochlear artery. The supratrochlear artery branches off from the ophthalmic artery, so occlusion of this artery with a sufficient volume of dermal filler will eventually also occlude the ophthalmic artery. Branching from the ophthalmic artery are all the vessels of the orbit including the central retinal artery, posterior ciliary, the lacrimal and supraorbital arteries, potentially causing a catastrophic injury to all the structures in the orbit.



As we see in the most severe cases the vessel is also attached to the internal carotid artery and cases of stroke and death have occurred from injecting 3 to 4 ml of fat into the supratrochlear artery.

THE FOREHEAD

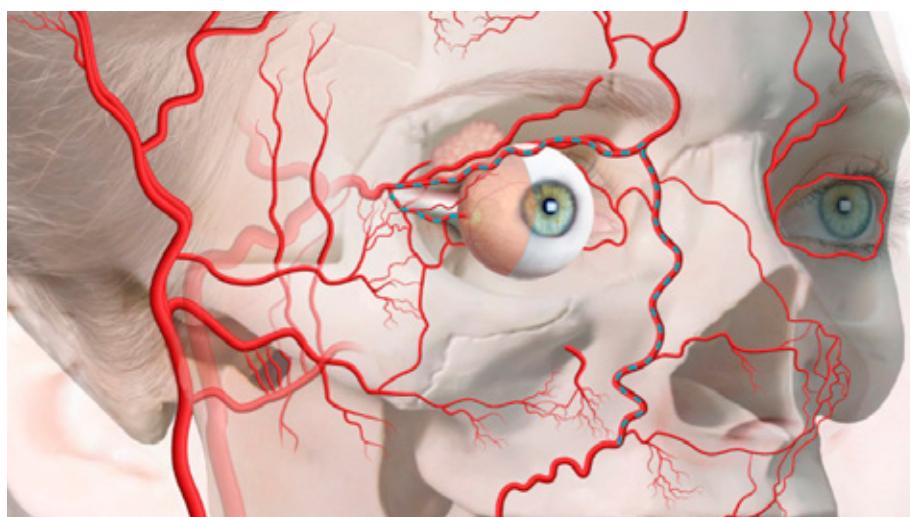
Treating lines, wrinkles or volume loss in the forehead is a risk with respect to blindness due to the supratrochlear and the supraorbital artery, which branch off the ophthalmic artery run in the forehead and are a likely source of ophthalmic injury. The superficial temporal artery often has connections with these vessels, and in particular, low-viscosity filler may pass through the temple and into the ocular circulation via these vessels.



THE NASAL REGION

The dorsal nasal artery is a branch of the supratrochlear artery. Injections along the dorsum of the nose near the glabella are particularly associated with blindness as a result of occlusion.

The superior blood vessels of the nose are closely connected with the blood supply of the orbit, moreover, injections in this area particularly in Asian countries can be fairly high-volume which may increase the risk of filling the supratrochlear artery which on average holds only 0.08 ml, with a range of between 0.04 and 0.12ml in one study.



Cases are recorded of blindness being caused by cannula and needle, and at the tip and the dorsum of the nose, as well as the lateral aspect of the nose presumably through the lateral nasal artery.

THE NASOLABIAL FOLD

The facial artery usually passes within 5mm to the nasolabial fold and can reside both in the cheek and in the fold. It tends to reside in the fatty layer, under the dermis and has a direct connection via the angular artery to the supratrochlear artery.



Emboli are at risk of making their way into the ophthalmic circulation via injections intended for the nasolabial fold. The artery lies at the level of the hypodermic fat in this region, and some more superficial patterns of treating the nasolabial fold are possibly riskier due to the needle tip becoming close to the artery, particularly if a fanning technique is used at the ala base. It is believed the periosteal injections and cannula techniques are hypothetically safer, which will cover in more detail later.

THE TEMPLE

There are several possible connections between temple injections and blindness. Through the arterial system, it has been established in cadaver studies that fluid may pass from the superficial temporal artery into the ophthalmic system, likely through superficial and anastomosis which occur along the forehead, and connect the supratrochlear artery, supraorbital artery and lacrimal artery to the superficial temporal artery.



There is also a hypothetical link between the deep temporal artery and the accessory branch of the middle meningeal artery which occasionally anastomoses with the ophthalmic circulation.

This is a rather indirect route as filler would have to pass inferiorly from a deep temporal artery into the maxillary artery then up into the accessory meningeal artery and then into the ophthalmic blood supply. If this was to occur you would assume significant other side effects from blockage of the deep temple and maxillary artery and all its branches.

It has also been hypothesised that intravenous injection of dermal filler into the deep temporal vein may allow filler to enter the cavernous sinus which in theory could cause blindness via reducing venous drainage in the same way that a cavernous sinus thrombosis can.

PERIOCULAR INJECTIONS

Periocular injections are responsible for several cases of blindness.

Lateral canthal lines and tear trough are commonly treated, but in China and Korea, it's not uncommon to inject filler into the eyelid to augment the pretarsal role. This treatment seems very strange to us, often seen as purposely creating an eye bag. The intent is to make the face look more positive and happy, as the bulge creates a vague sense of a smile. Aegyo sal actually means 'eye smiles' and is sometimes described as 'the happy fat'.

<http://www.youtube.com/watch?v=45l3bgSXsX8>

Unfortunately, the procedure is associated with cases of blindness and this is unsurprising given the proximity of the vessels that supply the eyelid to the injection and that these palpebral vessels branch off from the angular and supratrochlear arteries, as well as the angular artery, infraorbital and transverse facial.

Injections of low viscosity filler may flood the branches of the ophthalmic vessels.

THE EYELID

The venous drainage of the eye also has potential connections to the cavernous sinus or by directly blocking the vorticos veins drainage through occluding the inferior ophthalmic, or inferior palpebral vein

THE TEAR TROUGH

The Classical anatomy of the angular artery depicts the angular artery running just lateral to the nose and medial to the tear trough, In 2018 a systematic review of all the studies which looked at variants of the facial blood supply showed common and more dangerous variants to the classic view.



32% of cases had detoured variants of the angular artery, placing it in a position more vulnerable to tear trough injections. In these people, the artery ascends in the cheek more vertically instead of heading to the lateral nasal artery, and it then turns medially just lateral to the mid pupillary line in the mid-cheek, before following a path towards the orbicularis oculi just inferior to the orbicularis oculi retaining ligament, extremely close to where injections are sometimes placed.

THE CHEEK

The cheek is a relatively low-risk site and the systemic review of literature only found one case of Blindness occurring in cheek alone with autologous fat being used as the filler.

The most likely cause without details of the case would either be a medial cheek injection affecting the angular artery either directly or through an anastomosis of the transverse facial artery.

The risk is presumably higher in a patient with the detoured version of the facial artery which courses more laterally in the cheek, where more cheek injections are given, or possibly a complication caused by cannulation of a very lateral vessel, such as the superficial temporal artery or deep temple vein.

Let's now consider how the different materials may affect the chances of blindness from dermal filler.

Autologous fat was the most likely cause of blindness, though this is more likely in my opinion to be associated with the variables that go with injecting fat rather than the material itself. The biggest difference between fat and other products is the amount that is used. It is not uncommon to inject 2 to 20 ml of fat into an area at a time, and this factor is also no doubt responsible for the degree of damage that has occurred and the reduced chances of recovery of vision. In the biggest review, the chance of recovery was 21% when fat was injected versus 61% with an HA. The most severe cases of including one case of death after a stroke were caused by fat. The volumes are significantly higher, for example, the injection of 5 ml of fat into the supratrochlear artery during a procedure on the glabella was the cause of acute stroke and death in one tragic case.

I also observe that cases of blindness in temples both occurred with very low viscosity fillers (silicon oil and sculptra).

In the UK the first recorded case in the UK was in 2012 and occurred from a temple injection using poly-L-lactic acid, otherwise known as sculptra.

It may be that the injuries that occur from areas like the temple which are more indirectly connected with the eye are more likely with lower viscosity products which flow more easily along smaller vessels and may pass through narrower anastomoses into the ocular circulation.

In a cadaver study in 2011 researchers were able to reliably demonstrate the presence of a low viscosity pigment into the ocular circulation after injection into the superficial temporal artery in a clear majority of 9 cases. This effect is likely more powerful with lower viscosity products.

There is some interesting evidence that short-chain hydraulic acids are slightly pro-inflammatory and may irritate the tunica intima of blood vessels, hypothetically making blood clots more likely which

maybe particularly relevant in cases of venous occlusion. (Rayahin et al, 2015).

I am aware of one unpublished case report from Dr Patrick Treacy where volbella caused an occlusion Cavernous sinus after a non-surgical nose job.

Let's look in summary at all hypothetical routes a filler could result in blindness.

1. Direct injection into the supratrochlear artery and then in turn into the ophthalmic circulation.
2. Direct injection into the supraorbital artery and then into the ophthalmic circulation
3. Direct injection into the dorsal nasal artery and then into the supratrochlear artery and in turn into the ophthalmic circulation.
4. Direct injection into the angular artery and then into the supratrochlear artery and on to the ophthalmic artery
5. Injection into the palpebral arteries which branch from the supratrochlear and ophthalmic arteries.
6. Direct injection into the superficial temporal artery and then through anastomoses into the supratrochlear and supraorbital arteries and then into the ophthalmic circulation
7. Direct injection into the Deep temporal artery and then through the maxillary artery and the accessory branch of the middle meningeal artery into the ophthalmic circulation.
8. Direct injection into the middle temporal vein and then through the veins that drain the eye into the Cavernous sinus
9. Blockage of the ophthalmic vein superior and inferior through injections of the periorbital area.

10. Compression or blockage of the vorticosc veins which drain the eye. The lateral veins are most vulnerable in rare circumstances (see case study).
11. Stroke causing damage to the optic tract or visual cortex.
12. The pressure of a haematoma onto the eye
13. Blocked venous blood supply
14. Blood arterial blood supply.
15. Stroke or damage to the optic tract.

DIAGNOSIS

Classically the symptoms of blindness occur almost immediately certainly within a few minutes.

Blockage of the central retinal artery would stop the retina working within a few seconds. Many people have experienced this during a drop in blood pressure when standing up too quickly, the sense of your vision decreasing rapidly until you bend or sit down demonstrates how rapidly the retina stops functioning without blood flow.

In the example where dermal filler enters an artery and makes its way directly to the central retinal artery or blocks the whole ophthalmic artery, blindness in seconds would be the expected outcome. This is the most common and terrifying pattern scene in the literature.

There are cases in the literature where patients only appear to be diagnosed at the 3-week point several hours or days later. There is unfortunately little detail in the reports from the patients perspective about how their symptoms developed to make these cases more real to us. This is why I will be including more detailed case-studies in this course.

It's important to remember that there are acetylsalicylic at least 11 different ways that blindness can result from a treatment and not all of

them will evolve in exactly the same way. We must consider complete occlusions, emboli, compression injuries, venous and arterial injuries and stroke as potential causes of visual symptoms and investigate and refer appropriately according to the pattern that we see.

For example, if damage to one of the veins which drains the eye was to occur it may take longer for the build-up of blood to affect the function of the retina, particularly if the damage is only a compression injury rather than a complete occlusion.

Emboli may also take a period of time to arrive at the final site of blockage, be there in a vein or artery supplying the I all the visual cortex or optical tracks or nerves, all would have different natural histories and likely take longer to reveal themselves than the classic picture. Later in this course, we will discuss the case that may have been caused by the scenario and the visual changes were gradual over a few hours.

Partial occlusions which allow small amounts of blood flow, but insufficient to keep the retina alive may also cause blindness slightly more slowly, in hours rather than minutes.

Hours after the visual symptoms occur the effect on the muscles and the skin around the eye may occur. at this stage, patients develop ptosis or ophthalmoplegia and the injury becomes visually obvious to the clinician. These signs and symptoms are important because they give a clue as to where the potential injury is and may aid further treatment.

TREATMENT

So what should you do if you're ever in the awful situation of diagnosing blindness in one of your patients?

Blindness from dermal filler is an extremely rare event and the number of people with practical experience of dealing with it are extremely small. The number of cases available to study, especially where treatment was delivered expediently is also extremely small.

It is therefore extremely difficult to give evidence-based guidelines on how to deal with this rare event. We are left with only a 1st principle approach, knowledge gleaned from disease process affecting blood flow to the eye and a few case reports and partially relevant studies to guide us.

In the UK even specialist centres are inexperienced, and more so the chance of even a specialist centre being able to treat a patient within the short time a retina can survive without blood is incredibly small.

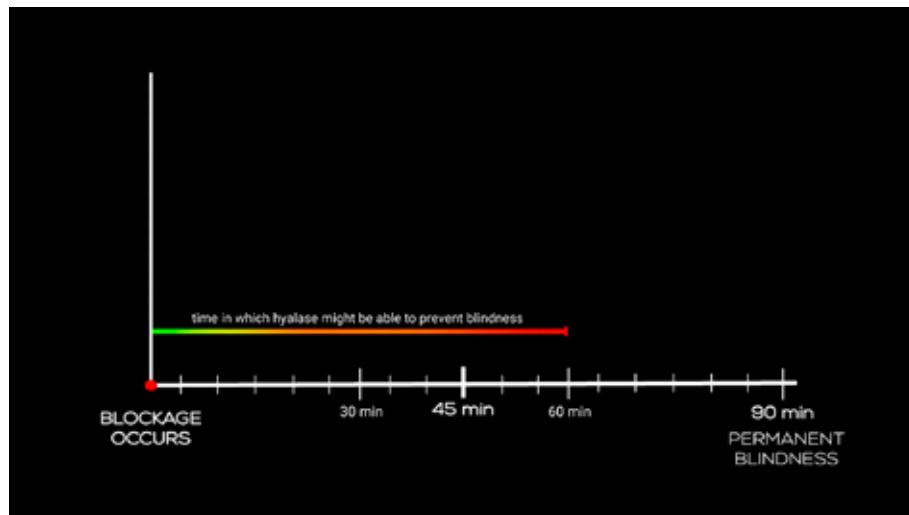
There is often a significant degree of inertia as clinicians are faced with what is invariably a new and unknown situation. To make the decision to aggressively treat would require time to assess the literature and do the necessary thinking to draw a plan of action from first principles.

Unfortunately, when you have a maximum of 60 to 90 minutes to restore blood flow to save the retina, way too much time passes as they attempt to navigate according to maps that do not apply to this situation and research the literature and assess risk.

It's my opinion that almost no patient referred to the hospital setting is likely to receive treatment in time. There are a few case reports of rapid diagnosis and treatment, but most of the detailed reports reveal a long delay between diagnosis and treatment.

Unfortunately, I believe it is up to the treating clinician to decide based on their perception of the risks, of their own ability to safely perform any rescue procedures, and the chances of their local ophthalmology department being able to help any better in the early stages.

Considering the length of time it takes to dissolve filler in any situation (45-90 minutes), it's likely that if hyalase is of any use at all, if it will be useful within the first 30 to 60 minutes of the diagnosis. Later injections may still be effective for preserving ocular muscles and skin.



As a result of this problem being so difficult to solve once it occurred the emphasis in your practice should be hugely on prevention. If you look at Industries which bear down on risk reduction incredible progress is possible through adding small improvements to processes systemically consistently. Will look closely at ways to prevent this in detail but first let's look at the suggested options for managing a case of blindness that has already occurred.

The rationale for treatment options all relate to the following strategic goals.

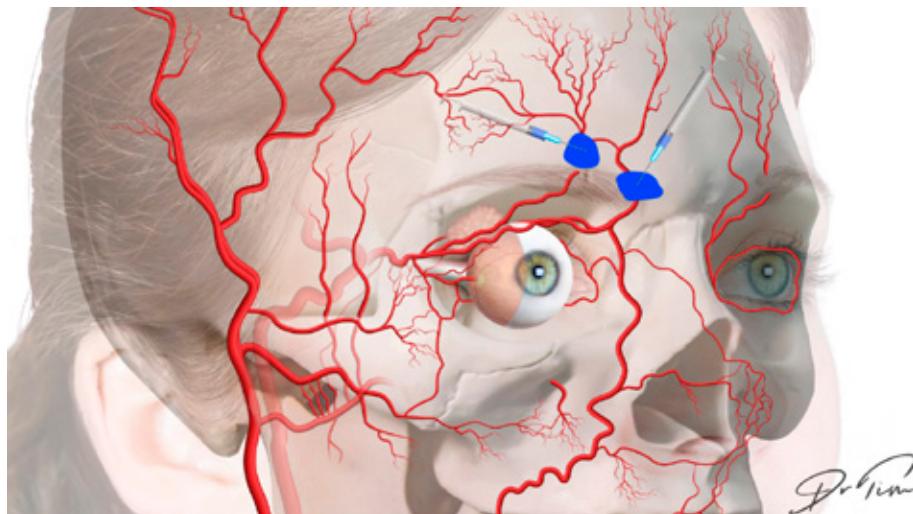
There are 7 ways that clinicians have identified that may improve blood flow to the retina. It's useful to remember precisely what the goals of each suggested are. Remembering the goal will help you critically analyse whether the intervention is likely to achieve the outcome.

Goal 1: To dissolve the Embolus to allow better perfusion of the retina.

There are 6 ways clinicians have suggested achieving this:

The first strategy to consider is supraorbital hyalase-perivascular injections into the superior orbit. Animal studies have shown that in

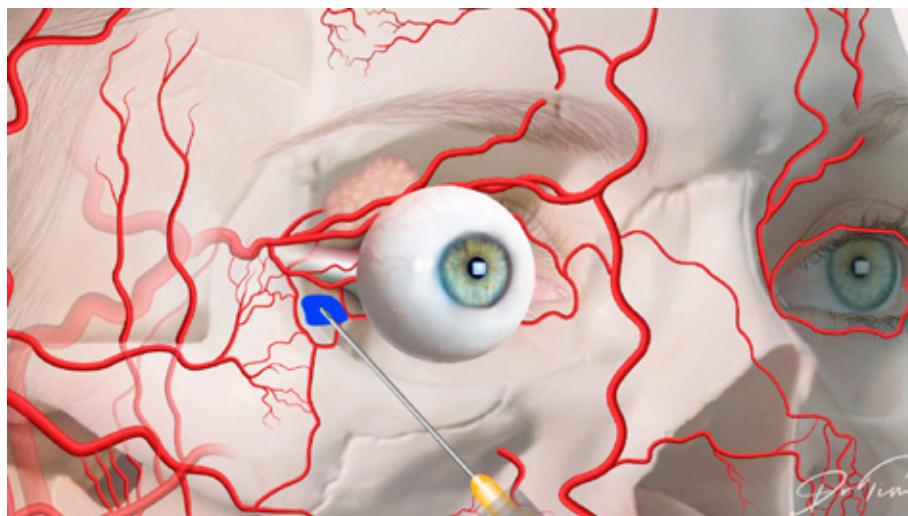
order for this to save the retina, blood flow must be restored within 97 minutes. Since the time to dissolve filler is likely 30 to 90 minutes, immediate treatment is required whichever method is used.



In this strategy, we aim to dissolve the filler that is blocking the supratrochlear or supraorbital artery by injecting the area around it as the artery emerges from the orbit at the notch. Without penetrating the orbital membrane it is less technically difficult and less likely to traumatise the bulb than a retrobulbar approach. This technique relies on the products defusing along the arteries and through the arterial wall to degrade the blockage.

This may be effective if the blockage resides directly underneath where the reversing agent is placed but, If the visual disturbance is caused by an emboli residing in the central retinal artery or a branch coming from this vessel, product that infiltrates these vessels will simply be washed into the forehead, away from the site of the blockage.

Retro Orbital hyalase is talked about most often and is often tentatively suggested as the best way to get hyaluronidase in contact with the central retinal artery and the ophthalmic artery since Hyaluronidase is placed behind the eyeball very close to these vessels. (Jean D. A. Carruthers et al 2014, 2015, Loh et al 2016, Beleznay et al 2015).



Unfortunately, it is technically more difficult, and so is often given too late for us to know if it would have worked. There are many more cases where this technique has been used unsuccessfully, but almost invariably 4 to 30 hours after the blockage, and therefore far too late for it to have any hope of preserving the retina.

Animal studies have demonstrated the ability to re-perfuse the area and restore vision if the injection is given within 30 minutes, but these studies included urokinase which is not usually part of the aesthetic practitioners toolkit (Chiang et al 2016).

Chestnut C. reported in 2018 successfully restoring vision when treating a patient with monocular blindness within 20 minutes of visual loss with the retrobulbar technique.

A study from Bangkok by Thanasarnaksorn et al reported in 2018 a case of retrobulbar hyalase that dramatically improved function of the extraocular muscles when given the day after the injury.

INTRAVASCULAR HYALASE: INTO THE OCCLUDED ARTERIES

Thanascaran reports a successful restoration of a quadrant of the left visual field. This would be consistent with an embolus rather than full occlusion.

Intravitreal hyaluronidase has been suggested but there are no recorded cases of it being tried. 55 IU to 75 IU of Hyalase in 0.1-0.2 ml has been used to treat intravitreal haemorrhage, but there are also established risks. It may reduce blood flow by causing a 30 mmhg pressure spike known to occur in 30% of patients who have this type of injection, and there is a risk this increase would decrease blood flow at a critical time. Higher doses of hyalase in animal models have been shown to cause retina damage in animal models.

Urokinase and hyaluronidase infusion combined. Shown to be effective in rabbit models if given in 30 minutes by (Chiang et al, 2016), the use of urokinase in medicine is usually for blood clots or embolisms, particularly pulmonary embolisms and thromboembolic peripheral vascular disease. There is one case report of these enzymes being used in combination when treating a supratrochlear artery occlusion with success (Demiri 2016). The use of urokinase stems from the hypothesis that after an occlusion blood clot within the vessel is common. It's hypothesized that this is more often the case with low viscosity short-chain acid fillers which are pro-inflammatory and may irritate blood vessel linings. The addition of urokinase allows for the breakdown of the clot alongside a breakdown of the dermal filler by hyaluronidase.

4 ways to reduce the intraocular pressure to increase blood perfusion

1. Timolol causes a reduction of the pressure within the eye by reducing Aqueous humour production.
2. Acetazolamide inhibits carbonic anhydrase and the sodium pump, decreasing aqueous humor production and lowering intraocular pressure.
3. IV Mannitol reduces the concentration of free radicals that promote cell membrane lipid peroxidation and ultimately destruction of cellular tissue. It also promotes cerebral blood flow by reducing blood viscosity and microcirculatory resistance.
4. Anterior chamber paracentesis. It's possible to reduce the intraocular pressure through this method of controlled drainage of the aqueous humour from the anterior chamber.

Goal 2: Reduce vessel resistance of blood flow into the eye

- Nitrates which dilate veins and arteries.
- Sildenafil: shown to induce vasodilation in the eye (Pache et al 2002)

Goal 3: Dislodge the embolus into a less damaging position

This technique involves rapid changes in Intraocular pressure over a period of 1 to 3 hours. The evidence is for embolic emboli occurring usually from blood clots or atherosclerotic plaque breakdown.

Apply pressure to the closed eye over the sclera. The eye is indented with one finger for only a few millimetres, pressing in and then releasing repetitively at a frequency of two to three times a second.

Goal 4: Reduce the chance of blood clot making the problem worse

- Aspirin
- Anticoagulants
- Reduce the chance of inflammation making blood perfusion worse
- Steroids
- Increase oxygen delivery to the retina

Hyperbaric oxygen increases the partial pressure of oxygen in the Plasma and can increase oxygen transfer to tissues by up to 16 fold (fleg byrn 2010)

RECOMMENDED MANAGEMENT PLAN

Vascular occlusions are well established to be best treated by dissolving the blockage with hyaluronidase. the problem with retinal artery occlusion is that it's very difficult to access. internationally these procedures are done by a complete cross-section of people from oculoplastic surgeons through to non-professionals. It's therefore vital that advice is given with the caveat that this is within the context of your own realm of competence and expertise. It is certainly possible to make matters worse through train far out of your competency in a moment of desperation.

Currently, there is no conclusive evidence of what is likely to help a patient with minimal risk of making matters worse, and so you must make an individual judgement on how you would handle this complication giving your experience, knowledge and competence.

Guidelines are unable to replace a clinicians careful individual analysis of this topic.

My thoughts are as follows: The key factor in reversing any occlusion is the administration of hyaluronidase. in the case of retinal artery occlusion, there is a window of only 90 minutes to fully unblock an artery.

My experience of vascular occlusions in other parts of the face has

taught me that it can take between 45 and 90 minutes to dissolve. This means in order for any procedure to have a chance of saving vision in a patient it must be given immediately. Reviews of the retrobulbar technique have never included using the technique before 4 hours have already passed and most cases occur many hours after the offending procedure, far too late to expect vision to be improved.

Guidelines often suggest that no intervention should delay referral to hospital, but neither should referral to hospital delay the most important intervention. The choice to opt for hospital-based treatment alone unfortunately, will likely condemn the patient to permanent visual loss.

The injecting clinician must, therefore, weigh up the potential risk of the different approaches and make an individual decision on whether or not the chance of saving sight through an immediate intervention warrants the potential risks associated with doing a procedure like this for potentially the first time on a real patient.

This is currently extremely difficult as we only have one case of a retrobulbar injection improving vision, and so a first-principles approach must be taken.

I believe the primary reason this is not recommended in all guidelines is due to a fear that the attending clinicians may be incapable of doing the procedure safely, and may end up making matters worse. While there is still little certainty that the intervention will make things better according to the literature.

Injection at the supratrochlear and supraorbital notches also seems like a low-risk approach that may help dissolve blockages in the nearby vessels. It would seem reasonable to do these as standard.

Retrobulbar injection is routine and represents a relatively low-risk intervention for many anaesthetists and ophthalmologists. For these and previously skilled individuals, I would have no hesitation in recommending retrobulbar hyaluronidase as soon as possible after verifying retinal artery occlusion.

This technique is now often covered on cadaver courses, which probably the most accessible form of experience an alone practitioner working in aesthetics can get, and forms part of a series of steps I would recommend any condition who carries out risky procedures in the nose, forehead, temple or glabella should do.

Carruthers et al 2015 recommend at least 500 units of hyaluronidase is injected in a bolus fashion n 4-8 ml. I would feel comfortable injecting 1500 units in this scenario.

Although I recommend intradermal allergy testing for vascular occlusions affecting the skin, I believe the chances of restoring site rely on the immediate infusion of the reversing agent and so in cases of retinal artery occlusion I would not do an allergy test on a patient with no history of allergic reactions.

At this point, I would recommend urgent transfer to a specialist Eye Centre. It's worth remembering the high incidence of cerebral vascular accident in patients suffering from ocular injury. It is reasonable to blue light the patient to the nearest emergency Centre.

While awaiting transfer may be worth carrying out a persistent ocular massage.

Once the patient has been transferred they should consider treating medically by lowering intraocular pressure. Topical timolol 0.5% (1 drop), IV Mannitol 20% 100 mL over 30 min, or IV acetazolamide or 500 mg tablet.

AVOIDING BLINDNESS FROM DERMAL FILLERS

The first thing to know about avoiding blindness is you have to change your attitude to risk. the truth is mostly humans assess risk through experience. if you've done it 10 times or 100 times or 1000 times and not cause blindness it starts to feel like familiar territory. I'll never forget the consultant who told me on my first day as a junior doctor

that first I would feel scared then, then I would gradually feel cautiously confident, then you'll feel like you have mastered things, and then you'll really fuck things up, and then you will become safe.

What I hope to achieve with this course is to give you a real sense of the reality of these complications so that you don't have to wait for it to happen to you before you completely transform your practice.

I don't believe you should ever inject without a sense of trepidation somewhere in the back of your mind. This uncertainty reflects the reality of what we do. We tread the line between order and complete chaos in our lives and the lives of our patients every time we inject dermal fillers. It's easy to forget this when you completed hundreds or thousands of treatments without severe complications.

The perfect balance comes when you are constantly thinking about the worst-case scenario so that you routinely do every safety step, and continuously add small improvements to chip away at the risk well not letting the fear of complication paralysed you and prevent you from building that makes a real difference to the people that you serve.

By far the biggest success story that you will ever come across about how this attitude really does reduce risk is the airline industry. 2017 Was the safest year for commercial airlines ever. Despite there being 10 x as many flights as we had in the 70s, we had only 0.1% the number of fatalities. This is an incredible achievement.

So what can we learn from the airline model?

The key is to continuously try to preempt potential accidents and then also to make system-wide changes in response to individual events. If you can learn what would prevent a catastrophe in a niche scenario and apply that to your systems and processes routinely on a daily basis forever after, you can mitigate that particular cause of catastrophe.

Remember to apply all the general advice for avoiding all vascular occlusions.

REDUCING THE RISK OF BLINDNESS

- Be Prepared
- Have a stocked emergency kit
- Patient Selection and consultation
- Seek a certain benefit/risk ratio
- Consent in detail
- Know your anatomy to select:
 - Appropriate injection depths
 - Safe angles of insertion
- Safer direction of insertion
- Select the appropriate instrument for the area, generally cannula
- Products, select according to the risk/benefit ratio
- Know the properties and risks profiles of products and select according to risk-benefit ratios
- Use reversible products wherever possible.
- Modify the volume of injection given at each site.
- Safety Steps Prior to injecting
- Do not prime first injection
- Aspirate
- Check pulses and mark out the anatomy
- Safety steps during the procedure
- Inject slowly
- Monitor patients for disproportionate pain and cease injecting.
- Safety steps after the injection

GLABELLA AREA

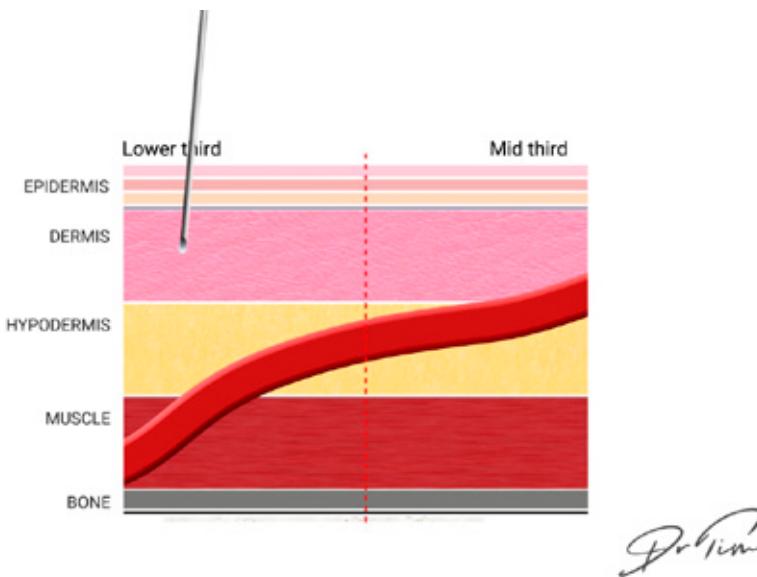
The most important way of avoiding the supratrochlear artery when injecting at the glabella crease is to be superficial. The artery starts deep on the bone and becomes superficial 2 to 3 cm up, as it travels into the forehead. Your needle should remain in the dermis at all times and not cross into the hypodermis or through the muscle close to the periosteum where the highest risk of occlusion lies.

Many clinicians advocate microdroplet technique instead of a linear thread in the glabellar area.

A linear thread could also be performed with a needle pointing upwards towards the forehead compressing the supraorbital notches firmly and aspirating multiple times along the line so before placing beads of product under the crease.

FOREHEAD

My preference for treating the forehead is to use botox in combination with dermal filler, where lines are deep and to use filler to revolmise under the muscle using a cannula angled in at 90 degrees to the direction of the primary arteries. At this depth, in theory, most arteries will be above the cannula and running at 90 degrees.



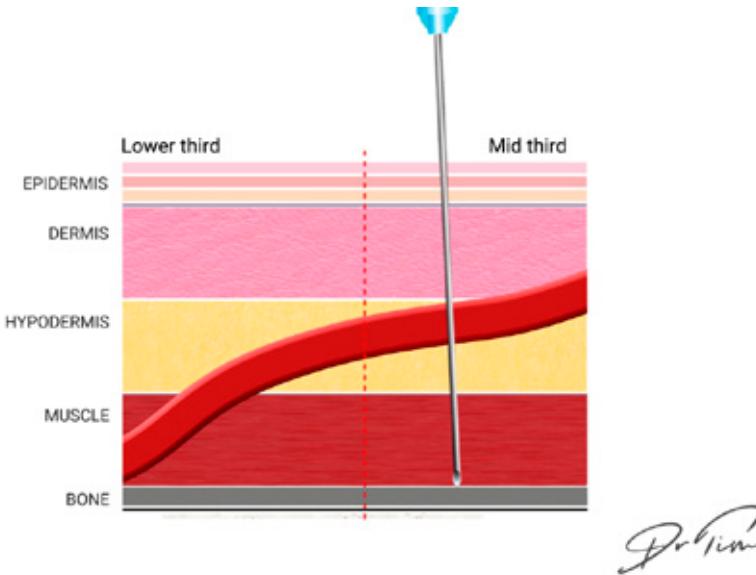
Above the midpoint of the forehead superficial lines have been treated with a dermal filler but the risk is significantly higher of getting filler into a blood vessel and causing local tissue necrosis. the small blood vessels here are branches of the orbital vessels and low viscosity filler could in theory flow back down the vessel into the orbital circulation, so this style of injection should be avoided in my opinion. It is helpful

if they ever are done to remain always at 90 degrees to the direction of most vessels.

Before injecting make sure of the cannula bevel is highly mobile to help reassure that it is not embedded in a transverse section of the superficial temporal artery where it may anastomose with the supraorbital or supratrochlear. If you do not prime the candy that you can aspirate with some sensitivity to reduce the chance of your first injection filling an artery,

Compression of the supraorbital and supratrochlear notches is also reasonable to do while injecting the forehead.

If you do choose to use a needle which is common practice it is advised to inject deep into the periosteum from Midway up the forehead, and very superficially in the dermis when injecting below this point.



THE NOSE

The majority of the blood vessels in the nose run rather superficially to the college or the bone. the most dangerous area is at the radius or the bridge of the nose and then at the tip. with respect to blindness, the bridge of the nose is by far the most serious risk as the dorsal nasal artery often branches off from the orbit.

Cannulas seem to feature disproportionately often. this could be because there is limited space for the vessel to move out of the way while sliding a cannula up the dorsum of the nose, and cannulas naturally gravitate towards medium depth in the hypodermis which is where blood vessels are more likely to run. If there is scar tissue from previous surgery these vessels will be less mobile and more likely to be penetrated even by a blunt cannula.



If using a needle it may be safer to angle the bevelled down and touch periosteum before placing small bonuses after aspirating. The total volume of the supratrochlear artery back to the ophthalmic artery averages is 0.085 mils. It may, therefore, be safer never to inject more than 0.05 ml per injection after aspiration.

When using a cannula do not prime the canular if you aim to reach the radix from the tip, and then aspirate which may rarely help you know you have penetrated a vessel. it can be also useful to move the tip of the cannula to check how mobile it is as a possible way of reassuring yourself you're not intraluminal.

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This month's guideline: visual loss secondary to cosmetic filler injection

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Blindness After Facial Injection

Askari Townshend, MD

An Anatomical Analysis of the Supratrochlear Artery: Considerations in Facial Filler Injections and Preventing Vision Loss

Article (PDF Available) in Aesthetic Surgery Journal37(2):sjw132 · August 2016

The Anatomical Origin and Course of the Angular Artery Regarding Its Clinical Implications

Kim, Yi-Suk MD, PhD*; Choi, Da-Yae BSDH, PhD†; Gil, Young-Chun PhD‡; Hu, Kyung-Seok DDS, PhD†; Tansatit, Tanvaa MD, MSc§; Kim, Hee-Jin DDS, PhD†

Dermatologic Surgery: October 2014 - Volume 40 - Issue 10 - p 1070–1076

The Journal of Anatomy

Variations in Branching Pattern of Facial Artery: An Anatomical Study

in 50 Indian Adult Cadavers

Ashish S. Kulkarni, Geetha K.N.

Department of Anatomy, MGM Medical College, Navi Mumbai, India

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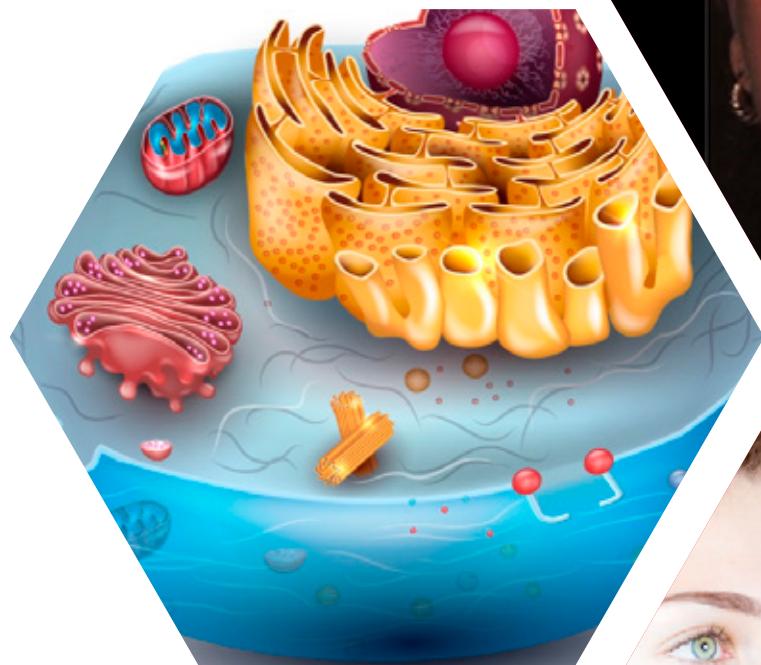
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8

IMPENDING NECROSIS

MODULE LINK

<https://drtimpearce.com/modules/impending-necrosis/>

**PATHOGENESIS: UNDERSTANDING THE CAUSE**

Tissue necrosis is arguably the most severe complications from dermal fillers. It is caused by oxygen supply not meeting cell demands, which over a short period of time leads to the dysfunction and eventual destruction of the cell and cell death. Many cells dying in an uncontrolled way in the tissue will lead to scarring and is a risk for infection when this occurs on the face it causes life-changing scarring and this in the context of aesthetic procedure is tragic.

We must therefore as clinicians spend a great deal of time understanding exactly how this problem can occur, how to diagnose it rapidly, how to treat it effectively, and how to reduce the risk of it occurring in future.

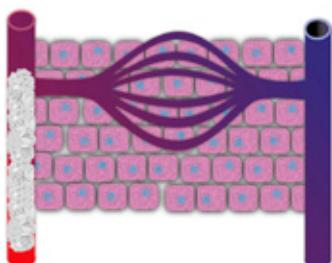
Let's look first at the pathogenesis of necrosis.

Understanding this in detail will help you immensely to judge the clinical picture at various different time points after the procedure.

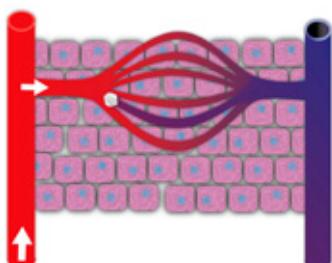
We can start by looking at impending necrosis. In this situation blood supply is compromised in one of several ways and the cells become distressed because the oxygen supply is insufficient to meet their needs. A lack of oxygen or hypoxia is the fundamental cause of necrosis from dermal filler injections.

Let's consider how dermal filler may reduce blood flow to an area of skin. There are actually several different mechanisms possible.

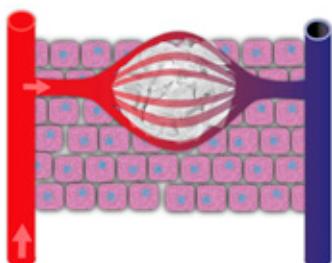
BLOCKED ARTERY



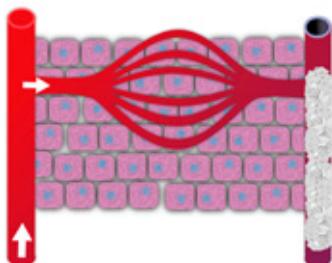
EMBOLUS



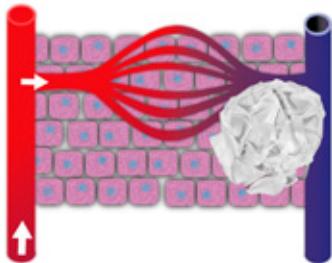
PRESSURE ON AN ARTERY



PRESSURE ON THE CAPILLARIES



VENOUS OCCLUSION



Cause 1 is a blocked artery. Locally injected filler completely occludes blood flow through an artery where there is no collateral blood supply to nearby tissue.

Cause 2 is an embolus. A small amount of dermal filler from the site of injection travels down the artery into smaller vessels causing distal areas of necrosis.

Cause 3 is pressure on an artery. Sufficient pressure on an artery and a key position, in theory, could decrease blood flow, although there have been no proven cases.

Cause 4 is pressure on the capillaries. This is similar to a pressure sore or ulcer, and it's caused by continuous pressure on the capillary bed reducing blood flow to cells supplied by those capillaries.

Cause 5 is venous occlusion. It is theoretically possible as we know from the impact of retinal vein thrombosis on the eye. In medicine, venous thrombosis of the retinal vein is a cause of blindness. This demonstrates the potential risk of blocking venous outflow from an area of tissue. If the exit of blood from a capillary bed is prevented by blockage of venous outflow, the entry of fresh blood may also be stopped, thereby reducing the options supply and risking necrotic injury. It is likely that most places in the skin have multiple collaterals which prevent this being a problem, but there may be rare instances that it could occur similar to the eye and should be a consideration.

Ok, so far we have a lack of fresh blood supply causing a lack of oxygen supply to the tissue. So let's look now at what is happening at a cellular level.

Oxygen is vital for extracting energy for the cell. Oxygen is required for the final stage of ATP production which occurs in the cells energy producing organelles, the mitochondria, and ATP is the fundamental energy currency of all living cells.

It's often called the energy currency because of the way that it powers molecules in a similar way as currency could be said to power an economy.

If you think of a cell as a factory, ATP could be understood as the currency that pays workers to do their jobs. Without payment, the workers would eventually stop doing their jobs and the factory would not function.

Without ATP the cell has no ability to do work of any sort and energy is required to power the work that keeps cells alive.

As the levels of ATP drop, many of the cells functions slow down or stop. But why doesn't the cell simply just take a break and rest and wait for oxygen to return at some other time?



The problem is many of the cells functions are involved in supporting the environment of the cell. In particular, the sodium ion pump which pumps sodium back out of the cell to balance the amount of water on either side of the membrane reduces its activity. This then causes an uncontrolled influx of both sodium and water causing the cell to swell.

The cell also starts to bleb and expand, causing a destruction of the cell's cytoskeleton.

Similarly the cell's protein production factory, the rough endoplasmic reticulum also swells uncontrollably.

There is a backup energy supply for the cell which is anaerobic respiration. The problem is it's very inefficient compared with aerobic respiration producing only 5% of the energy from a given supply of glucose, so time is still limited. What is more, there is also a toxic by-product of this process called lactic acid. Lactic acid eventually builds up and changes the pH of the cell which has a knock-on effect on many of the cell's other molecules systems and processes. New acidic environment that's created denatures and destroys many of the enzymes and proteins and stops them from functioning.

Up to this point, it may still be possible for the cell to survive if oxygen supply is restored even though it may be in a dysfunctional and damaged state.

Where things become irreversible is when more physical damage to the proteins that make up the cell occur and this occurs when loss of control of calcium balance occurs. Calcium is also controlled by an ATP-dependent pump and an influx of calcium into the cell can inadvertently activate many of the destructive enzymes usually used in a controlled way. The cell zone proteases, for example, can start to become active and destroy the cell's own proteins. Endonucleases can also be activated and these actually destroy the cell's own DNA. The damaging effect of an increasingly acidic pH, the influx of calcium and activation of certain enzymes can also destroy the membrane surrounding lysosomes. This results in a massive release of highly destructive digestive enzymes usually used to break down large molecules and make them useful for the cell. Eventually, even phospholipase is activated and this enzyme destroys the phospholipid bilayer. The result is a complete and irreversible destruction of the cell.

To make matters worse the destructive effect of these enzymes remain active once the cell is dead and will continue causing damage to neighbouring cells furthering the destruction of tissue in the area.

Cells with relatively low metabolism may be able to survive for many hours like this, while cells like neurons have only minutes.

Once cell death is complete the body will attempt to resorb the dead tissue but often cannot at a rate quickly enough to prevent secondary bacterial infection becoming part of the picture. With a lack of fresh blood supply, there is also a lack of immune system effectiveness and so any bacteria nearby will begin to multiply to make the most of the opportunity to grow on the dead tissue. This secondary element of the problem is important to understand and recognise as it affects diagnosis during a late presentation, as well as management.

In summary

A decrease in oxygen supply caused by a drop in blood flow through an area of cells will cause uncontrolled cell death which has a knock-on effect of causing destruction to nearby tissues even if they are perfused by a neighbouring blood supply.

DIAGNOSIS

It's vital to make the correct diagnosis as soon as possible in cases of vascular compromise. Most tissues will suffer irreversible necrosis within hours of losing their blood supply and so it matters to diagnose and treat as soon as possible.

The most common cause of vascular compromise is occlusion of a major artery in the face.

Let's look at signs and symptoms as they occur in most cases chronologically

The first sign that something may have gone wrong is usually while the filler is being injected. In many cases, the patient reports a particularly painful experience during the injection either as the needle enters the artery or as filler expands within it and travels down the lumen.

Sometimes particularly in lips, the injection is associated with spasm of the artery and an area of pallor that follows the path of the vessel. This is easier to see in lips because they are usually red and fill with blood, and change to pale during the injection never normally occurs.

After the injection, the area may feel a sense of pressure and pain different to other areas of the face in some situations.

This picture is not always present however and many injections seem innocuous at this stage.

In many parts of the face, there may be very little to see at this stage to the casual observer.

Many patients will be sent home at the stage without any particular reason to be concerned. However, if you take time to examine patients there is often a change that you can pick up in the early stages.

I believe it's vital to make it part of your routine to examine for capillary refill after every procedure. Lightly compress the skin while you are cleaning at the end of each injection and observe for a capillary refill. This should enable you to pick up some cases of occlusion before sending the patient home.

If in doubt you can also check for capillary blood flow by pricking the skin with a small needle to see if there is any bleeding.

Once the patient leaves your office presentations of vascular occlusions tend to be quite different on return.

Typically the patient will call the office back 6 to 24 hours later and

Report significant pain in the area of injection, and sometimes bruising. It's important to examine them in the flesh



Examining patients later on the same day is often much more of an obvious presentation. The skin may be a dusky grey and mottled, it may be swelling, and it's usually clearly in the path of one of the major arteries.

Capillary refill will be very slow or absent, and the area is often very tender to touch.

Beyond the first 24hrs pain can continue to increase as inflammatory response to dying tissue occurs. by 48 to 72 hours some signs of full-blown necrosis will start with tissue looking dark or black in areas and sometimes the skin becoming covered in small pustules.

The vast majority of occlusions will happen near large vessels in the face. There are particular danger zones that you should know, The area injected is an important part of what should sensitise you to signs and symptoms of necrosis.

Clearly, the first few hours are critical for accurate diagnosis, so I suggest the five steps to systematically diagnose or exclude vascular occlusion.

1. Capillary refill is King: Many inexperienced clinicians simply send

photographs of a lesion when requesting help from experienced clinicians revealing they do not understand the importance of capillary refill. Always check and document the capillary refill time. If you do this on patients routinely it becomes incredibly easy to see when refill time is compromised. Capillary refill is extremely reassuring you simply don't get necrotic lesions occurring with good pink CR occurring in less than 2 seconds, so take note of what you see and diagnose accordingly.

2. Always use a control area when checking for capillary refill it is much easier to see the difference in refill time in a compromised area when you have compressed a nearby area at the same time so that you can see the return of blood flow at different rates.
3. Always compress the area firmly for at least 5 seconds The sensitivity of capillary refill time is massively decreased when low-pressure short compressions are used, as not enough blood is squeezed out of the capillary network.



4. If you believe you two have diagnosed a vascular occlusion map out the area of blood flow compromised carefully using your anatomical knowledge to check the path of the artery. This could, for example, be checking the superior labial artery and the columella artery as it runs to the nose or perhaps the superficial temporal artery and its path into the forehead and scalp. Whatever the artery consider, where it leads and check all the places that may be occluded, remember to include areas that are harder to see, like the columella of the nose. It may be helpful in some areas to use a cotton bud to increase the resolution of your test and isolated small areas that may be showing blood flow compromise.
5. If in doubt and capillary refill is difficult to ascertain, for example in pigmented skin, you can use a pinprick test to identify areas of capillary bleeding which will reassure you the blood flow present.
6. Take into context how long ago the procedure was and if there is any apparent bruising. Question of blood flow soon after a procedure is more concerning than question will blood flow several days later without any signs of necrosis.

Venous occlusion may present in a different way, do I have yet to see one documented or met someone who has seen a necrotic wound from one.

You would expect puffiness in the area that is not being drained associated with some hypoxia what discolouration due to the build-up of venous blood.

Capillary refill may be present as there is still arterial blood flow but it would look purple due to the blood becoming stagnant and hypoxic due to a lack of outflow.

Anastomosis are such that this situation would be extremely rare.

MANAGEMENT

Once you've diagnosed vascular occlusion it is time to take action to restore blood flow if there is tissue left to save.

Evidence suggests it takes several hours, probably between 2 and 12 in total for the skin to die without blood flow.

If your examination in history fits with a diagnosis of occlusion and there is tissue still alive with signs of impending necrosis then it is important to take action to restore blood flow.

The rationale 4 suggested management is based largely on first principles with some evidence supporting it.

It is believed that we may be able to improve blood flow in the following ways.

● **Goal 1:** To dissolve the filler to allow reperfusion

Hyaluronidase applied to

- Site of injection
- Area of delayed refill
- Area supplied by the artery
- Rarely practitioners discuss intravascular hyalase but this is not generally recommended.

One application or pulsed high dose protocol.

● **Goal 2:** To Reduce vessel resistance of blood flow

- Nitrates which may dilate veins and arteries.
- Sildenafil: Shown to induce vasodilation of arterioles and could improve blood flow.
- Warm compress

- **Goal 3:** Reduce the chance of blood clots making things worse.

300mg Aspirin for it's antiplatelet properties.

- **Goal 4:** Reduce the Chance of inflammation making blood perfusion worse

Steroids

- **Goal 5:** Increase oxygen delivery to tissues

Hyperbaric oxygen increases the partial pressure of oxygen in the blood

- **Goal 6:** Reduce the risk of secondary infection

Antibiotics (Flucloxacillin or Clarithromycin)

Let's review these options as a protocol:

<https://vimeo.com/manage/282288138/general>

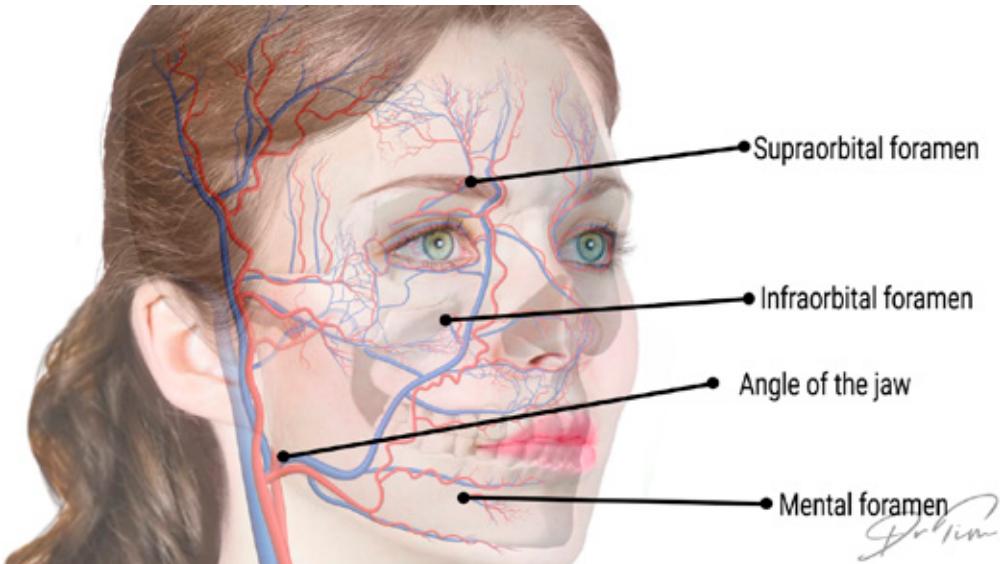
HOW TO REDUCE THE RISK OF VASCULAR NECROSIS

Let's consider some key principles

Inject with careful consideration of the 3-dimensional anatomy. Most people can easily read a diagram and learn to know where the key arteries are on the face. To inject safely you need to understand the 3-dimensional position of the arteries, and which layer of the face they will most likely reside.

General anatomical principles for artery avoidance are:

- Arteries usually are not at the depth of the bone except where arteries enter the face at the foramen, or the notch in the mandible.
- Arteries are not usually superficial, at the level of the dermis, but beneath in the hypodermis or lower.



- Try to pass arteries at 90 degrees instead of in parallel, which makes cannulation into a vessel less likely.
- Aspirate habitually. The more you aspirate the easier it gets- like wearing a seatbelt, do it all the time and there when you need it.
- Be stable when you aspirate to make it count when you inject in the same place.
- Aspirate for 3-10 seconds, particularly in high-risk areas.
- In high-risk areas aspirate along linear threads to increase safety.
- In most places, use a cannula if the result would be the same as with a needle.
- Be gentle with cannulas, they only increase the pressure required to penetrate a vessel, they do not make penetration impossible.
- Remember In tight spaces (like noses) it is easier to penetrate an artery with a cannula.
- Always check capillary refill times after every procedure, partly so your eye becomes attuned to what is normal, to reassure yourself and your patient, or in the worst case make sure diagnosis occurs as early as possible.
- Always Explain to your patient the risks and what the signs and symptoms of an occlusion are so that they seek help properly. The worst cases are invariably the ones diagnosed the latest.
- Give your patients an advice leaflet detailing

- Always be prepared, carrying emergency reversal kit at all times if you're injecting.
- Keep an emergency reversal protocol handy- You may only have to do this once in 10 years.

In Summary

- Most occlusions you have hours before scarring is likely. You should still act swiftly and decisively.
- Prevention is where your daily efforts should be, and this is really something you integrate into all of your processes.
- It takes years to develop the concepts and the mental model of what's going on under the skin.



9

STROKE

MODULE LINK

<https://drtimpearce.com/modules/stroke/>



The incidence of CVA from stroke is extremely low. It is hugely correlated with the incidence of blindness, hunting at the fact that the causative injections are very similar. Likewise, the way to reduce the risk of stroke is the same as what we have covered in blindness.

PATHOGENESIS

To cause a stroke from dermal filler, the product must enter the intra-cranial circulation and then come to rest in an artery supplying part of the brain. For this to occur dermal filler must make its way from the external carotid system into the internal carotid supply.

Blockage of a vessel will cause hypoxic damage to the brain in a matter of 3 minutes.

HOW WOULD A STROKE OCCUR?

There are only two routes by which the filler may enter the cerebral circulation from a facial injection.

One is by retrograde flow of filler from the facial artery into the common carotid artery and then into the internal carotid. It would take a relatively large volume of product to cause this.

The other is through the ophthalmic artery via one of the three vessels in the orbit, the supratrochlear, supraorbital or lacrimal arteries.

Once into the internal carotid supply, there is a close connection to the middle cerebral artery.



The middle cerebral artery supplies the frontal, temporal and parietal lobes. Small amounts of filler would travel into the lobes and block small but vitally important sections of the vessels causing symptoms of a stroke. You can see also how large volumes of fat have tragically caused life-threatening injury when greater portions of blood flow in this area were disrupted.

There have been several cases of fillers causing stroke and death, but mostly as a result of fat transfer.

DIAGNOSIS

Diagnosis of stroke should be made if the patient suffers sudden onset of the following symptoms

Their face may have dropped on 1 side, they may not be able to smile, or their mouth or eye may have drooped.

Arms: The patient may not be able to lift both arms and keep them there because of weakness or numbness in 1 arm.

Speech: Speech may be slurred or garbled, or the person may not be able to talk at all despite appearing to be awake; they may also have problems understanding what you're saying to them.

MANAGEMENT

In a case where the symptoms appeared immediately after the procedure, it is vital to call an ambulance immediately. Advise them your patient has had a stroke and they will attend urgently.

While you wait for the ambulance supportive measures should be started. Lie the patient down and start oxygen. Give them 300 mg of aspirin and consider hyalase in all of the areas treated, although it is implausible that this would make a significant difference in the short term to the brain, it may reduce the complication of local tissue necrosis in the areas occluded, which may go overlooked in the hospital.

There is little else that can be done in the short term to help. Hence prevention should be the primary concern.

AVOIDANCE

As you can see the key as always is prevention. Although the injections that cause blindness are all the same that may cause stroke, proximity to the connection to the internal carotid system is obviously a significant risk factor when we are using products of 1 ml or less.

In other words, the highest risk injection would be a high volume injection close to the supratrochlear artery or the common carotid artery it is therefore vital that we employ excellent injection technique in these areas as covered in the blindness module.

As a brief recap of the key areas, when injecting the forehead or glabella always inject superficially, aspirate well with an unprimed needle, and compress the supratrochlear artery as you press the plunger. To prevent intracranial injury always use small amounts at a time, less than 0.085 ml.

There has been a case of a surgeon using fat as a dermal filler around the jawline causing a stroke. In this case, fat was injected with a cannula and was high volume, filling the facial artery back to its origin at the common carotid, with fat then flowing into the internal carotid causing a complete hemiplegia. This is likely to be easily avoided simply by the fact that most hyaluronic acid fillers are not injected that quickly or in volumes of sufficient magnitude. hopefully understanding these principles will at least make you more aware as you inject and also worry less unnecessarily.

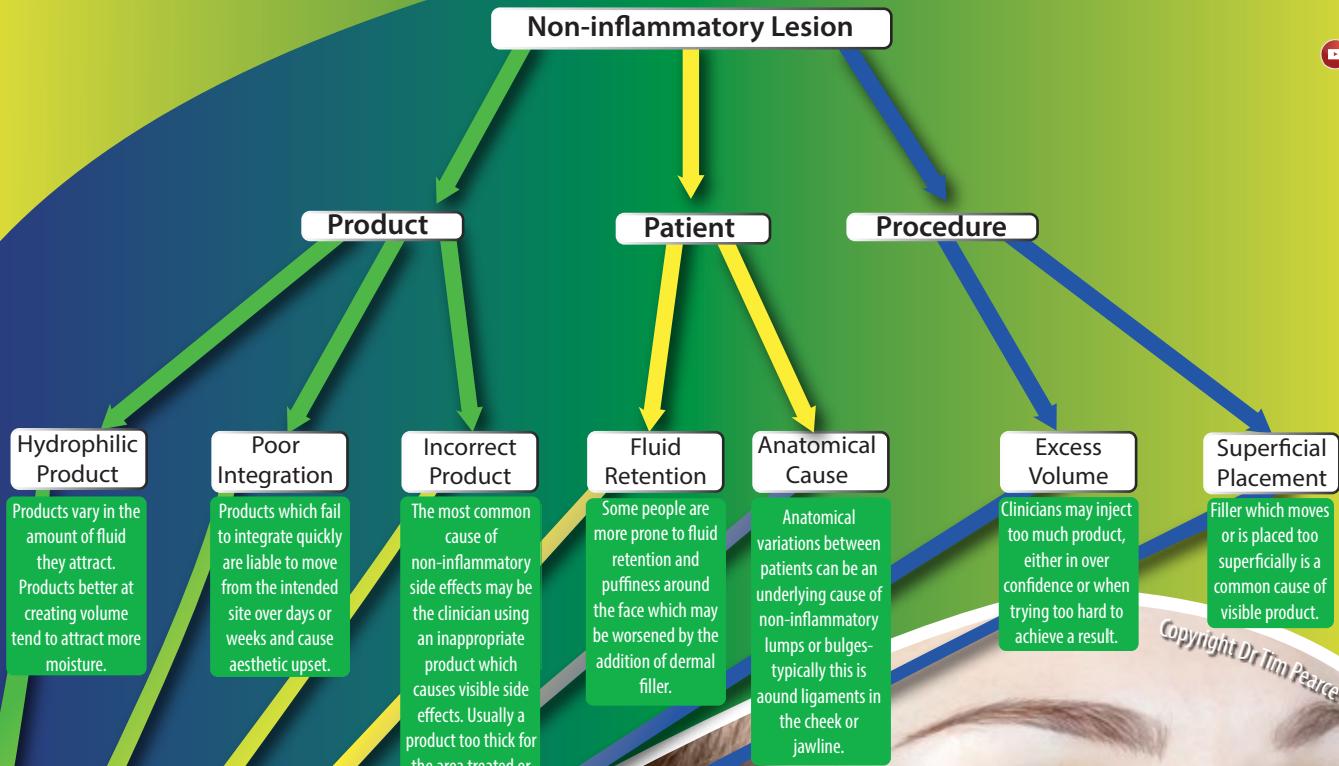


10

PROTOCOLS

Non-inflammatory Lesions

Lumps, bumps & bulges after dermal filler



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Thin skin and delicate aesthetically defining structures like lips, jawline, tear trough, cheek & nose are particularly sensitive to non-inflammatory lumps, bumps and fullnesses. Ligaments in the cheek, tear trough and jawline may highlight problems. Overly superficial injections may cause problems anywhere in the face.

Firmly compress areas of fullness and massage away from the area affected. Use a simple cream as a lubricant to aid

Massage
By Clinician

To increase the chance of a minimal intervention good outcome consider asking the patient to massage at home for a limited time.

Regular Massage
by the Patient

Time can allow natural improvement. Consider if the problem is mild or there is evidence of

Watch & Wait

Hyaluronic acid is water soluble, and lumps may be dispersed more easily if saline is injected here no risk of allergy.

Inject
Saline

Penetrate the lump with a green needle. Extrude filler by squeezing. This reduces allergy risk and allows for partial instead of complete removal.

Mechanical
Extrusion

Follow the elective reversal protocol to dissolve all product in the area of concern. This may result in more complete removal of the product but includes risk or allergy.

Hyaluronidase

Consider
Inflammatory
Causes

Each stage in isolation may lead to resolution on its own, or the next step should be considered. The goal is to achieve the best outcome for the patient with the minimum of risk. The sequence of steps may be covered in one appointment or over a period of weeks depending on the severity of the side effects, the judgment of the clinician, and the impact of the problem on the psychosocial well-being of the patient.

Resolved

Inflammatory Lesions

After Dermal Filler Procedures

Dr Tim Pearce
www.drtimpearce.com

Guidance is based on expert opinion. It is recommended you keep updating your knowledge as evidence emerges & seek senior support as

CAUSE

Infective



Viral Candida Bacterial Biofilm

Reactive



Often Treated as the Same Entity

Traumatic



Type IV Reaction

Traumatic



Type 1 Reaction Induration Bruising Haematoma

SYMPTOMATOLOGY

MANAGEMENT

Localised Inflammation

Inflammation at all injection points

May occur within days of the procedure

May occur months after the procedure

Purulent

Non-Purulent

Delayed onset lesions appear >48-72 hours from the procedure.

Vesicular Rash

Inflamed Itchy Skin

fluctuant Mass

Non-fluctuant Mass

Inflammation waxes & wanes

Inflammation peaks & fades or is chronic

+ve Culture more likely

Lab culture likely negative

Incision & Drainage

Treat Infection

Local Reaction

Angiodema, Urticaria, Anaphylaxis.

Occurs in weeks after haematoma

Occurs Immediately

Mildly Tender Lump or fullness

> 1cm Lump

Fibrous area of tissue

Skin Discolouration (Purple)

Faint pink or normal skin surface

Scale of Allergy

Anaphylaxis

Antivirals Anti-fungal

First Line Antibiotics 7days

Steroids

Persistent Infection

Improving

Persistent Nodules

Consider Hyaluronidase (Continue Antibiotic Cover)

Continue for 14 Days

Steroid Injections [Specialist]

Second Line Antibiotics

Consider Hyaluronidase (repeat 2-4 weekly if improving until resolution)

Anaphylaxis Pathway & hospital admission

Conservative

Hyaluronidase (Rarely Done)

Aspirate Blood

Resolution or Referral

SUSPECTED BIO FILM- First Line

Macrolide
Clarithromycin 500mg twice daily for 14 days
Tetracycline
Minocycline 100mg twice daily for 14 days
Doxycycline 100mg twice daily for 14 days

Steroid Regime *See BNF

Prednisolone
Initially 10–20 mg daily for 7 days
Up to 60mg daily in severe cases
Alternative: Dexamethasone
0.5–10 mg daily for 5–7 days.

INJECTABLE STEROIDS

Triamcinolone acetonide:
40mg/mL diluted as required.
Graduated injections of triamcinolone acetonide of 0.1mL starting with a 10mg/mL concentration and then increasing concentration to 20mg/mL and 40mg/mL at four weekly intervals.

SUSPECTED BIOFILM: Second Line

Dual antibiotic therapy
Clarithromycin 500mg twice daily for 14 days
Doxycycline 100mg twice daily for 14 days

It's important to stay in close contact with your patient and monitor the clinical signs and symptoms which occur as the treatments you have prescribed start to have an effect so that you can make adjustments if there is deterioration, improvement or failure to improve.

Elective Reversal Protocol

Hyaluronic Acid Dermal Fillers

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Advice is based on expert opinion. It is recommended you keep updating your knowledge as evidence emerges and seek colleague support as required.

1 Define the indication for reversal



Indications:

- Water attraction causing increased volume and lost definition
- Asymmetry
- Patient dissatisfaction despite technically good result
- Lumpiness due to poor injection technique
- Reactions, infections, inflammatory lumps.

2 Explore the other options and their risks

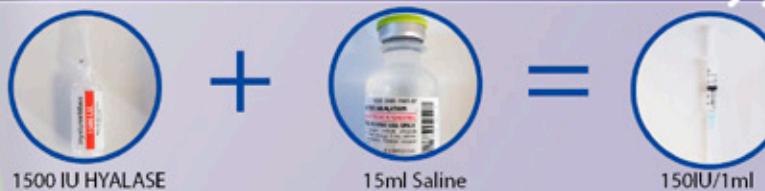
Firm massaging by practitioner, regular massaging at home and waiting are all options that may degrade filler lumps and asymmetries and have no additional risk which should be contrasted with the risks and benefits of immediate reversal.

3 Seek informed consent.

The risk of reversal is a 1/2000 risk of allergic reaction which is relatively high. This risk is higher if they are allergic to bees or wasp stings which contain hyaluronidases. All dermal filler in the area should be considered vulnerable as hyaluronidase permeates very easily. Reversing very specific amounts is not possible. Endogenous hyaluronic acid is also going to be degraded which causes a temporary loss of pre-existing volume that may be apparent for up to a week.

4 Prepare hyaluronidase solution.

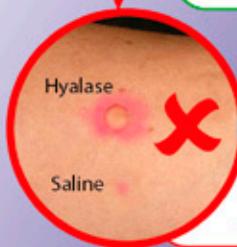
Create a solution of 1500 IU of Hyalase in 15mls of normal or bacteriostatic saline. This solution will dissolve approximately the same volume of hyaluronic acid- so inject 1ml of hyalase solution for each millilitre of dermal filler you wish to dissolve.



5 Inject a tiny intradermal bleb of the hyalase, 0.01mls using a BD/Insulin syringe commonly used for botulinum toxin treatments. Place a similar amount of saline as a control, so that you can see if redness is due to the needle or due to an allergic reaction.



Risk of anaphylaxis is low, proceed with injecting the solution.



HIGH RISK!
Raised pale itchy papule surrounded by erythema

6 Inject hyalase into each area affected. Use an equal volume of hyalase solution as the volume of HA you would like to dissolve.



Then you want a total reversal, inject more than is theoretically required to reduce the risk of needing multiple exposures to hyalase.

Place the reversal agent at the same depth and position as the filler. It takes ~24hrs to dissolve.

Do not retreat for 14 days

24hrs



Photos Courtesy of Sarah Murphy RNCP
antiageingaesthetics

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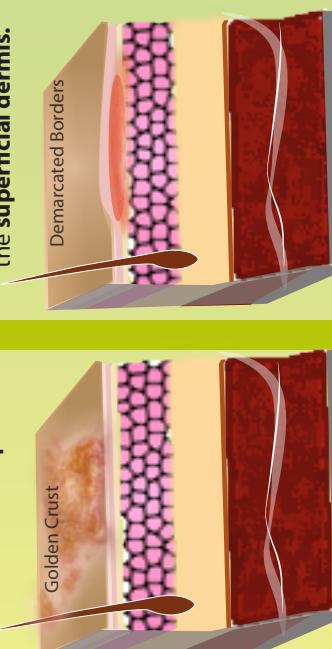
Infection Quick Reference Guidelines

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Impetigo



Impetigo appears first as red and then Golden Crusty lesions over a few days. Often itchy, painful, and spreads easily. It is caused by *staphylococcus aureus* infection of the **epidermis**.



Management

Localised infection:

-Treated with topical fucidic acid 3 times/day 5 days.

Wide spread or bullous:

Combine with oral antibiotics as per local guidelines:
-Flucloxacillin 500mg QDS for 7 days or
-Clarithromycin 500mg bd 7 days.

Wide spread or systemic symptoms-

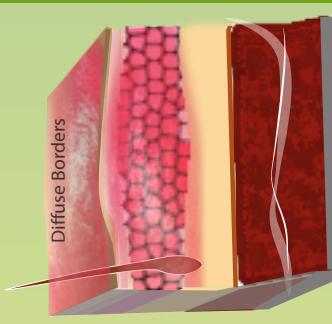
Refer for urgent IV antibiotics

Systemic Signs/Symptoms signs of sepsis should be documented and appropriate urgent referrals made if present

Erysipelas



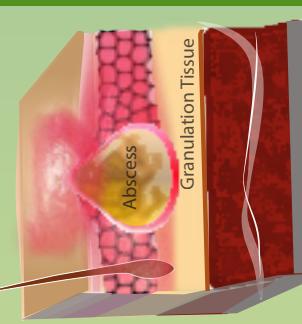
Cellulitis is an infection in the **lower dermis** and hypodermis. It is characterised by a tender pink lesion with poorly defined borders.



Cellulitis



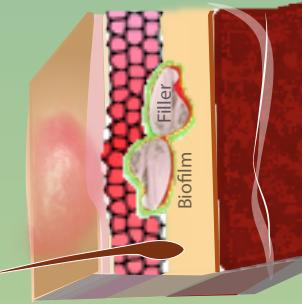
Abscesses form in the **lower dermis and hypodermis**, and are localised, very tender fluid filled masses which may produce a purulent discharge eventually.



Abscess



Biofilm reactions often form inflamed areas of lumpy or nodular filler. The classically fluctuate in severity or form chronic hard to treat infections. Clinically similar to type IV reactive nodules.



(See Inflammatory Lesions Protocol)

Biofilm Reaction



Herpes Simplex infection is often reactivated by procedures. Small vesicles on the **mucosa** of lips, or sometimes **superficial dermis**. Beware mixed bacterial infection.



Mild to moderate- topical aciclovir OTC.

Oral Aciclovir if diagnosed with blisters still present and severe. 200mg 5 times a day usually for 5 days.

Consider prophylactic aciclovir for treatments in the future if reactivation occurs post procedure. 400mg twice daily.

Herpes Simplex



Immediate Blindness

Emergency Treatment Protocol

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Signs & Symptoms

Guidance is based on expert opinion. It is recommended you keep updating your knowledge as evidence emerges stay within your competencies & seek senior support as required.

If you see these symptoms after a high risk procedure

Think Filler Blindness!

1 Diagnosis

- Severe Pain on injection
- Visual Disturbance
- Blindness
- Relative Afferent Papillary Defect
- Ptosis
- Ophthalmoplegia
- Severe Pain
- Vomiting
- Monitor for stroke symptoms

2 Call for Help



Immediately call for practical assistance from someone nearby to help you if possible.



High Risk Injection Areas

FOREHEAD
Supratrochlear
↳ supraorbital artery

GLABELLA
Supratrochlear artery

NOSE
Dorsal Nasal & Supratrochlear artery

TEMPLE
Deep & Superficial Temple artery & veins

NASOLABIAL FOLD
Facial Artery

3 Initiate Treatment

HYALASE RESCUE REGIME

SUPPORTIVE MEASURES

4 Prepare hyaluronidase solution.



Create a solution of 2 mls of saline with 1500 iu of Hyaluronidase in the 5ml syringe.



300mg Aspirin



Apply Timolol 0.5% Drops



Continuously Perform Ocular Massage



Provide Re-breath Bag

Low Risk Supportive Measures should be carried out with the help of an assistant or inbetween stages of the reversal process until the hand over to a specialist

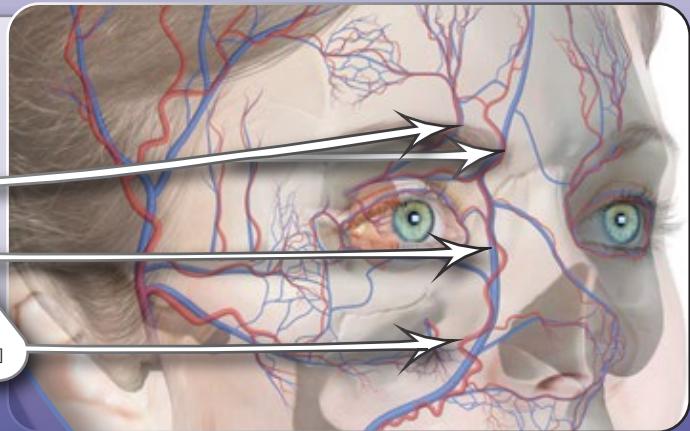
5 Consent Patient for Intervention

6 Flood the likely path of filler to the eye.

Flood emerging vessels of the orbit
[Inject the supraorbital and supratrochlear exit points]

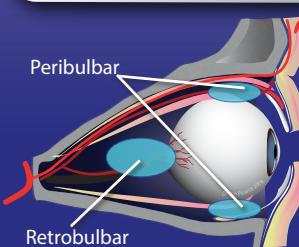
Flood path of connecting arteries
[Inject from the site of causative injection to the emerging orbital vessels
In the example illustrated, the angular artery but may be Superficial temple or forehead in different cases]

Flood original Site of HA placement
[e.g. Nasolabial fold, glabella, forehead, temple, cheek or other.]



HIGH RISK INTERVENTIONS:

It is possible to place hyaluronidase closer to the blood supply as it enters the orbit with retrobulbar or peribulbar placement. This placement is closer to some key vessels but the risks of damaging sight is substantially higher.



Are you competent to perform a peribulbar or retrobulbar injection, and able to justify the increase in risk of eye trauma?

YES
Inject 500 to 1500IU of hyaluronidase by retro/peribulbar technique.

7 Transfer to hospital

Causative Material/Brand: _____

Product Placement: _____

Diagnosis: _____

Brief Management Summary:

Clinician Name: _____

Phone: _____

Emergency Reversal Protocol

Advice is based on expert opinion and first principles rather than robust evidence. It is recommended you keep updating your knowledge as evidence emerges.



Artery Affected	Causative treatment	Area affected*
Supratrochlear	Frown lines	Glabella, forehead
Angular	Tear trough, nose, nasolabial fold.	Nose, forehead.
Lateral Nasal	Nasolabial fold, Nose, Alar (Lateral nostril), lateral lip.	
Transverse Facial	Cheek	Middle cheek
Sup/Inf labial.	Lips, oral commissures	Lips

Indications:

There is an area of skin that is persistently pale.

Capillary refill is absent or very sluggish

The offending injection may have triggered arterial spasm

Area of pallor may be distal to injection site.

Pricking the skin yields no blood

Usually painful immediately, gets more painful if left untreated.



1 Diagnose blood supply compromise

⌚ Remain calm, and communicate calm. You have 2 hours or more before necrosis could occur.

2 Explain the diagnosis & treatment options.

Unfortunately some filler has entered and blocked one of the blood vessels near the injection site. The safest course of action is to inject a dissolving agent which will break down the filler.

3 Seek informed consent.

The risk of not reversing the treatment is a scar, formed as a result of a lack of blood supply to an area of skin resulting in tissue death. The risk of reversing it is allergy, including anaphylaxis, temporary loss of native hyaluronic acid and treatment failure. [Document on a consent form].

4 Prepare hyaluronidase solution.

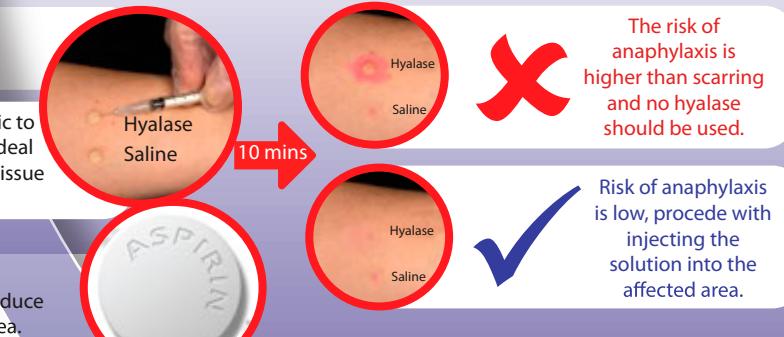
With a 5ml syringe and blue needle draw up 2mls of saline. Snap the glass vial of Hyaluronidase and squirt 1ml of the saline into the vial to dissolve the product. Then withdraw the solution back into the syringe to leave 2 mls of saline with 1500 iu of Hyaluronidase in the 5ml syringe. Change the needle to 27 guage or smaller- this is now ready to use.



5 Allergy test on forearm.

Inject a tiny intradermal bleb of the hyalase, ~5units using a BD/Insulin syringe. Inject saline as a control.

Is Allergy Testing Necessary? Consider the risk of anaphylaxis (are they allergic to bees or wasps or have had anaphylaxis to anything else) and how would you deal with anaphylaxis in the context of your working environment? Consider that tissue necrosis occurs in hours not minutes and make a judgement in each case.



6 300mg of Soluble Aspirin - optional.

While the allergy test is in progress, 300mg of Aspirin can be used to reduce the risk of a blood clot further reducing blood supply to the affected area. (Evidence of effectiveness is scant but benefits likely outweigh harms).

7 Inject Hyalase Solution

Place the solution exactly where the injection that caused the problem was placed and near any areas exhibiting palor. Aspirating is not necessary. The enzyme does permeate through vessel walls and recanulating is not required for effectiveness.

8 Massage, Warm Compress, Check Capillary Refill Cycle

Apply a warm compress to dilate vessels and promote enzyme activity for 3 minutes. Next give the area a firm massage for 2 minutes. Repeat this cycle 4 times. Check Capillary refill.

9 Repeat: Inject 0.5 to 1ml of hyaluronidase every 15-20 minutes, as can diffuse away.

10 Additional Options Post Reversal

Sildanifil- 50mg/day - may cause arterial dilation improving blood flow.

GTN paste- Still used, but some say detrimental blood flow due to disproportionate venous dilatation.

Prednisolone- 30mg/day for 3- 5 days- decreasing inflammation may improve arterial flow

Antibiotics- Prevents the spread of bacteria that may over grow due to reduced blood flow.

11 If you fail to achieve blood flow after 2 hours consider referral for wound management. If unsure, review daily and repeat the process.



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