Using a controlled trauma technique when injecting hyaluronic acid dermal fillers

Abstract

The use of dermal fillers, as well as the effects of dermal needling on the skin, have been studied and confirmed to stimulate neocollagenesis. In this article, the author discusses the combination of these two concepts in one technique to offer superior outcomes. The article supports the notion that hyaluronic acid (HA) stimulates collagen in the skin and uses evidence to suggest that specific techniques and instruments can greatly enhance the durability of results. Based on the controlled injury theory in which type I and type III collagen production occur at accelerated rates post trauma, the technique uses a sharp needle to create a modest subcuticular undermining effect. This controlled trauma disrupts fragmented collagen strands and replaces them with purposeful, elongated strands of promoted neocollagenesis. Controlled injury begins the wound healing cascade and creates new, non-fragmented collagen, providing improvement in the appearance and health of ageing skin. When neocollagenesis is increased, the duration of the desired outcome also increases. Type I and III collagen have been shown to last 4–7 years, creating an aesthetic result lasting long after the duration of the HA filler itself.

Key words

► Controlled trauma ► Dermal fillers ► Injection technique ► Neocollagenesis

For the first decade after the inception of hyaluronic acid (HA) dermal fillers, most aesthetic practitioners attributed treatment outcomes to the product injected, rather than any effect it may have had on the skin. However, in more recent years, multiple studies have demonstrated that injecting HA can stimulate the body to create its own collagen, producing results that outlast the duration of the dermal filler itself (Carruthers et al, 2014; Paliwal et al, 2014).

To date, nothing has been studied regarding the possibility of neocollagenesis resulting from the instrument being used to deliver an HA filler, nor the technique used in its delivery. This article will both support the notion that HA stimulates collagen in the skin and use evidence to suggest that specific techniques and instruments can greatly enhance the durability of results.

Dermal collagen matrix network

To understand the mechanism of action underlying the creation of long-lasting results, aesthetic practitioners need to consider the workings of the dermal collagen matrix network. In youth, the collagen matrix is filled with abundant, intact, coiled, supportive collagen fibrils (Fisher et al, 2009). These intact bundles act as a stimulant to their neighbouring fibroblasts—the primary collagen-producing cells in the dermis. On binding to intact collagen coils, the fibroblasts are stretched, causing them to produce more collagen (Fisher et al, 2009). However, during the ageing process, dermal collagen begins a process of fragmentation, leaving behind a series of unsupportive, deflated collagen fragments. These fragments are not able to reach the fibroblasts and cannot send messages to them to stretch. Therefore, in aged skin, collagen fragmentation contributes to the loss of structural integrity and impairment of fibroblast function. This unfortunate imbalance represents a self-perpetuating cycle resulting in thin, collagen-deflated and aged skin (Fisher et al, 2008).

The importance of this lattice-like dermal collagen matrix network is appreciated when it is realised that type I collagen is the most abundant protein in human skin, making up more than 90% of its dry weight (Fisher et al, 2008; Makpol et al, 2011). The loss of collagen manifests as ageing skin because type I collagen is the primary component of the reticular dermis, and type III collagen is the main component of the papillary dermis (Kim et al, 2011).

Skin architecture

The skin is the largest organ of the body and the first thing people see when they look at another person. It is easy to determine a person’s age by the quality of their skin—even though babies have wrinkles and folds, one can tell by looking at them that they are not old. The visual appearance of ageing occurs with abnormal texture of the dermis, as well as skin atrophy, elastosis, deterioration and laxity (Makpol et al, 2011). The thickening of the stratum corneum, and thinning, not only of the viable epidermal layer, but loss or flattening of the undulating epidermal–dermal border, are clear indicators of both intrinsic and extrinsic ageing (Quan et al, 2011).

Modifying the architecture and losing the armature of the skin’s framework contributes to the laxity and descent of the underlying tissue. It has been reported in the literature that collagen content in the dermis rapidly decreases...
by approximately 1% per year after the age of 30 years (Spock et al, 2012). This collagen loss contributes to some of the primary markers of age, such as skin deterioration, skin laxity and volume depletion (Makpol et al, 2011). Unless the cycle of ageing (Figure 1) is interrupted, the loss of collagen will quickly result in these visible signs of ageing.

Fragmentation
Fragmented, disorganised collagen in the skin is detrimental to the process of creating new collagen and contributes significantly to the ageing process (Fisher et al, 2008; Setterfield, 2013). Collagen becomes fragmented as a result of the release of collagen-degrading enzymes. Although there are four degrading enzymes, or matrix metalloproteinases (MMPs), capable of breaking down type I collagen, only one enzyme is able to interrupt the healthy collagen turnover in the skin. This enzyme is called interstitial collagenase and labelled MMP-1 (Fisher et al, 2008). Thankfully, the body has some natural inhibitors to the release of MMPs; however, due to the slow rate of collagen reproduction, the degradation of existing collagen usually takes over, leaving aged skin full of fragmented, unusable collagen. Consequently, the accumulation of fragmented collagen is a significant contributor to age-related changes in the skin.

In addition, fragmented collagen cannot be repaired or incorporated into new collagen fibrils and therefore causes visible defects in the collagen matrix. Fibroblasts that produce and organise new collagen cannot attach to fragmented collagen. Loss of this attachment prevents fibroblasts from receiving mechanical information to stretch and therefore they collapse, rendering them not just useless, but also detrimental (Fisher et al, 2008; Setterfield, 2013). Moreover, collapsed fibroblasts have been found to produce higher levels of MMPs (Fisher et al, 2008).

Interrupting the cycle
Some interruption is necessary to prevent this destructive cycle from continuing unchecked. When treating patients with dermal fillers, aesthetic practitioners should aim to initiate a process that will diminish the amount of fragmented collagen present, since fragmented collagen renders the healthy collagen incapable of performing; and instead promote healthy collagen to begin forming once again. This can reverse degradation and initiate a new cycle of healthy collagen formation (Figure 1).

Inducing controlled trauma
Treatments that stimulate production of new, non-fragmented collagen should provide substantial improvement in the appearance and health of ageing skin (Fisher et al, 2008). Recent studies have shown that collagen production after injection of HA occurs with enhanced structural support within the extracellular matrix (ECM), which in-
dues fibroblast elongation and activation, and initiates the synthesis and deposition of type I collagen (Carruthers et al, 2014). HA is a colourless injectable gel composed of naturally occurring sugar (polysaccharide). Polysaccharides are found in the highest concentrations in the skin and connective tissues; they are an important element for skin hydration due to the attraction of sugar molecules to water (Wang et al, 2007). There are three primary ways to instigate an interruption to this process (Kim et al, 2011; Collier, 2013) and induce ‘controlled trauma’:

- Chemical injury: treatment of depressed acne scars with trichloroacetic acid increases collagen fibre density and elastic fibre fragmentation
- Thermal injury: the principle of laser therapy is to stimulate collagen production by delivering controlled heat that contracts and remodels existing collagen fibres, as well as promoting neocollagenesis
- Mechanical injury: microneedles induce micro-injuries, which increases collagen fibres and elastin deposition in acne scars.

For the purpose of this article, the author will be looking primarily at trauma created by mechanical stimulation, namely by the introduction of HA into the dermis using a sharp needle, with some controlled undermining.

**Blanketing**

There are various ways of delivering dermal fillers and all techniques have their own benefits. In the patented ArqueDerma technique, ‘blanketing’ (Figure 2), the injection of thin strands of HA filler, placed in strategic multidirectional vectors using a sharp needle, allows controlled trauma benefits to cover more surface areas and address volume loss, skin laxity and skin deterioration (Fulton et al, 2012). HA filler is administered applying an intentional, controlled force on the tissues, stimulating the fibroblasts, causing them to stretch.

Stretching of the fibroblasts is essential to the formation of collagen (Kim et al, 2011). Counterbalancing inward pull and outward push, both originating from the attachment of fibroblasts to intact collagen, establishes dynamic tension within the fibroblasts and collagen matrix (Fisher et al, 2008). This dynamic mechanical tension controls fibroblasts shape (stretch) and function (Fisher et al, 2008). It could also be argued that there is an additional element of keratinocyte involvement, as keratinocytes are responsible for releasing dermal growth factors that orchestrate underlying cellular turnover and communicate with fibroblasts to facilitate collagen production (Setterfield, 2013).

**Injecting with a sharp needle**

The advantage of using a sharp needle in injection zones, within an area requiring additional collagen stimulation, is that through inducing controlled trauma one is not only injecting HA into the area, but also mechanical trauma is being introduced, thereby setting off the wound healing cascade (Figure 3). Injuries initiate the wound healing
cascade, and intentionally ‘wake up’ the fibroblasts to start producing collagen (Setterfield, 2013). If the fanning technique was used with a blunt-tipped cannula rather than a sharp needle, one may be reducing the amount of controlled injury, thus potentially creating less swelling and producing less neocollagenesis (Fulton et al, 2012). It is important to note that anytime neocollagenesis can be increased, the duration of the outcome will also increase. In general, new type I and type III collagen will last 4–7 years before the natural cycle of degradation occurs (Aust et al, 2008).

Undermining the tissue
The patented ArqueDerma technique using the blanket- ing pattern described above comprises an element of undermining, or ‘subcising’ the tissue before injecting it with a HA filler. US dermatologist Orentreich trademarked the term ‘Subcision’ to describe the technique of non-surgical separation of the skin to its adhesions (Orentreich and Orentreich, 1995). He discussed the process of undermining scars, wrinkles or cutaneous depressions that work to break up and release the attachments of contour abnormalities from the surface of the deeper structures (Orentreich and Orentreich, 1995). Blood accumulates under the defect and subsequently brings about collagen formation, which leads to long-term correction of the defect (Goodman, 2001). Subcision is simple, effective and appears to result in the long-term correction of contour defects (Goodman, 2001).

Figure 2: Neocollagenesis as a result of the ArqueDerma technique where a hyaluronic acid dermal filler is ‘blanketed’ throughout the dermis

Figure 3: Wound healing cascade
Skin texture
In addition to the blood being an important space holder, the hyaluronic acid injected is seen as crucially important, acting as a ‘liquid glue’ and keeping the tissues from early reattachment. Using the needle as a tool to create a modest subcuticular undermining effect results in a controlled trauma that disrupts fragmented collagen strands and replaces them with purposeful, vectored strands. Treatment with skin needling breaks old collagen strands in scars and wrinkles, which may promote the removal of damaged collagen and induce healthier collagen growth immediately under the epidermis (Fisher et al, 2002; Kim et al, 2011).

The broader the area over which HA is delivered into various areas of the reticular dermis, the greater is the surface area that can implement the wound healing cascade process illustrated in Figure 3. Using a combination of the above described technique, ArqueDerma translates into longer-lasting results, with the additional effect of hydrating and smoothing the skin’s texture in the treated areas (Figure 4), lasting several years (Figure 5).

Cross-linked dermal fillers
Cross-linked HA fillers have been shown to stimulate the production of several ECM components through space occupancy in the dermis and swelling through absorption of interstitial fluid during the first few weeks post injection (Paliwal et al, 2014). Therefore, it can be postulated that the more HA that is in contact with areas of the dermis, the more swelling that will occur after a treatment, consequently creating more neocollagenesis than with an atraumatic bolusing technique (Fulton et al, 2012).

Inducing a wound healing response over a broader surface area
Inflammatory phase, days 1–3
Scar (or wrinkle) improvement requires collagen remodelling, including breaking old collagen strands and inducing collagen growth (Kim et al, 2011). Using a needle during the treatment over scars, or wrinkles, causes a mechanical breakdown of existing collagen structures that connect the scar (or wrinkle) with the surface (Aust et al, 2008; Setterfield, 2013). Injecting with a needle also creates more intentional space for the HA to be placed, enabling the wound healing cascade to produce an instant wash of MMPs during the initial inflammatory phase, resulting in the initial degradation and clearance of the fragmented collagen matrix (Fisher et al, 2002; 2008; Kim, 2011). This initial degradation plays an important role in remodelling the scar tissue, by allowing healthy, coiled collagen to form, uninterrupted (Fisher et al, 2002; 2008; Kim, 2011). The associated trauma induces the normal wound healing inflammatory cascade, and the fragmented collagen is broken down further and reabsorbed, through that initial wash of the MMPs. It is after this phase that new, healthy collagen can be replaced (Aust et al, 2008).
Inducing a wound proliferative response along with a HA filler over a broader surface area
Proliferation and fibroplastic, days 3–28
This phase allows more existing fibroblasts to respond (stretch) to the stimulus (HA). According to Setterfield (2013), ‘a direct physicochemical effect on collagen organisation by HA is possible, so that an elevated content of HA favours a dispersed, hydrated conformation of collagen’. Fibroblast stretching is critical for normal, balanced production of collagen (Fisher et al, 2008). Activated, stretched fibroblasts begin depositing glycosaminoglycans (GAGs), type I and III collagen, and fibronectin—all stimulating collagen production (Wang et al, 2007; Fisher et al, 2008).

Inducing an increased wound remodelling response
Remodelling and maturation, 28 days–2 years
MMPs break down disorganised collagen, leaving new collagen to become oriented along the lines where mechanical trauma took place. This process lasts from 20 days up to 2 years (Setterfield, 2013).

Conclusion
It has been proven that the injection of HA filler into the dermis leads to significant neocollagenesis (Wang et al, 2007). It has also been shown that inducing controlled trauma using a needle in the dermis can cause notable neocollagenesis (Setterfield, 2013). By combining these two strategies, aesthetic practitioners can increase the amount of collagen that is produced by increasing both the level of HA distribution and the controlled trauma created with the strategic use of the injecting needles.

As technology and science progresses, there will be more answers to the question of how to best stimulate enough collagen and achieve a longer-lasting effect on the skin’s appearance. In the meantime, aesthetic practitioners are able to combine various techniques of proven effectiveness to achieve collagen stimulation.

Acknowledgements: The author would like to acknowledge Dr Allison Divers (US) and Dr Sarah Boxley (Australia) for their help during the original systematic review process.

References