In clinical practice, aesthetic intervention for platysma bands is one of the most common demands from patients wishing to improve the appearance of their aging neck. Concerns are attributable to overprominent platysma muscles that can be seen as two vertical, bulky cords from the lower border of the mandible to the suprasternal region. Platysma bands may become visible at a younger age in patients with thin skin. The muscular bands can remain hidden in patients with more fat.

Some authors have stated that laxity of the skin, loss of tone of the platysma muscle, and detachment from the deeper planes are responsible for these vertical muscular bands. According to this theory, the loss of muscle tone follows the skin sagging. Therefore, treatment of the platysma bands should aim to tighten the skin and muscle.

In our opinion, platysma bands are caused by aging of the anterior portion of the platysma muscle that makes it more visible. The prominent platysma bands reflect hyperkinetic platysma activity. Skin laxity follows the anterior part of the platysma and not the other way around. In our clinical experience, the only exception is in very old patients, where compromised skin elasticity can make a minor contribution to the platysma bands.

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bands. We demonstrated this concept in a clinical study that involved patients with total, unilateral, facial palsy who were followed for up to 10 years.

**PATIENTS AND METHODS**

Between June of 2000 and June of 2015 at Sainte Anne’s Hospital of Paris, France, we performed a prospective, descriptive study in patients who had undergone otoneurosurgical treatment and subsequently presented with definitive, unilateral, facial palsy confirmed by electromyography. This study was carried out according to the principles of the Declaration of Helsinki.

Patients were seeking a solution to their facial palsy and initially attended our Plastic and Reconstructive Surgery Department immediately after surgery during their hospitalization in the Neurosurgery Department of the same hospital. They returned to our Department for follow-up at 3 months, 6 months, and 1 year after surgery and then once per year to complete 10 years of follow-up.

All patients received surgical treatment that included canthoplasty and a gold plate for the paralyzed eye, plus a passive or a dynamic suspension (temporalis muscle transfer) for the labial paralyzed commissure. On the contralateral side, they were treated with botulinum toxin injections on the upper and middle thirds of the face. The mandibular line and neck were not treated on the healthy side.

At each visit, we observed the anterior neck appearance through a meticulous clinical examination of the muscles responsible for facial expressions. While the patients were sitting, we assessed the platysma muscle and the skin overlying it at rest and at maximum frown. Two surgeons independently recorded the aesthetic consequences of the platysma muscle denervation and differences in appearance compared with the healthy side.

During each consultation, we took standardized patient photographs: one front view at rest, one front view of the platysma muscle at maximum frown, two profile views, and two three-quarters views. [See Figure, Supplemental Digital Content 1, which shows a 55-year-old woman with left facial palsy at rest and at maximum frown (left). Prominent right muscular bands are visible. The same patient, 10 years later, continues to receive treatment with botulinum toxin (right). The right anterior muscular band is still present, whereas lateral muscular bands are considerably less prominent because of treatment. No changes were observed on the neck skin of the paralyzed side. Reprinted with permission from Expert2Expert, http://links.lww.com/PRS/B949.]

**RESULTS**

**Patients**

A total of 25 patients were enrolled in the study. Fifteen patients were women (60 percent) and 10 were men (40 percent). The age range was 35 to 80 years. The average age was 55 years. Twenty-two patients (88 percent) were followed for 10 years. Two patients (8 percent) were lost to follow-up after 8 years, and one patient (4 percent) was lost to follow-up after 5 years. The average follow-up for the full study population time was 9 years 6 months.

**Observations**

Of the 25 patients with definitive, unilateral, facial palsy included in this study, 19 (76 percent) had visible platysma bands on the healthy side but not on the paralyzed side of their face. Three patients (12 percent) had a spastic form of facial paralysis. In these patients, the platysma bands were apparent on both the paralyzed and the non-paralyzed sides (Fig. 1). Three patients (12 percent) did not show visible platysma bands. These patients were the youngest of the study cohort (aged 35, 37, and 40 years), and all had fatty necks (Fig. 2).

Based on the photographs that were taken at different times during patient follow-up, there appeared to be no modifications of the platysma
band appearance in any patient. No ptosis of the neck skin on the paralyzed side was observed. [See Figure, Supplemental Digital Content 2, which shows a 65-year-old man with right facial palsy (left). A prominent left anterior platysma band is visible, and there is no muscular band on the paralyzed side. Frontal view of the same patient at age 75 years (right). Ten years later, no changes were observed either on the left muscular band or on the skin of the neck on the paralyzed side. Reprinted with permission from Expert2Expert, http://links.lww.com/PRS/B950. See Figure, Supplemental Digital Content 3, which shows the frontal view of a 58-year-old patient with right facial palsy (left). A prominent left anterior platysma band is visible. Ten years later, the neck appearance has not changed (right). Reprinted with permission from Expert2Expert, http://links.lww.com/PRS/B952.

DISCUSSION

Our study found an absence of platysma bands on the paralyzed side of the face of patients with nonspastic, unilateral, facial palsy. In three patients, bands were not present. However, these patients were young with good skin elasticity and had fatty necks, features that delay the appearance of platysma bands. The heightened visibility of the submaxillary gland and submental fat on the paralyzed side in 80 percent of patients probably arises because the platysma muscle on the affected side does not push these anatomical structures inward.

Platysma bands are one of the first signs of the neck aging process that starts at approximately the age of 55 years and increases exponentially throughout the rest of life. Age of onset in the periorbital area differs and starts earlier, typically at approximately 45 years of age. Age-related neck changes may also include submental fat accumulation, laxity of the skin with loss of the cervicomental angle, bone reabsorption, and compromised neck contour caused by jowl formation.

The platysma is a flat, broad, shallow muscle that runs under the subcutaneous tissue on each side of the neck. The fibers arise obliquely upward and inward from the fascia superficialis of the upper portion of the thorax, clavicle, acromial region, pectoralis major, and deltoideus muscles and insert into oral commissures, perioral muscles, the dermis of the cheek and chin, and into the anterior third of the linea obliqua mandibulae posterior to the depressor anguli oris bone origin (Fig. 3).

The platysma muscle is innervated by the cervical branch of the cervicofacial trunk of the facial nerve, and it anastomoses with the transverse superficial cervical plexus. The cervical branch divides into an ascending branch to innervate the anterosuperior part of the platysma and into a branch that descends to the lower muscle. The upper part of the platysma muscle may be co-innervated by the mandibular branch of the cervicofacial trunk of the facial nerve.

The platysma muscle partially covers the sternocleidomastoidus muscle. Between these muscles, there is a superficial cervical fascia that...
corresponds to a fibroareolar connective tissue layer\textsuperscript{15} and, immediately underneath, a deep cervical fascia.\textsuperscript{16,17} The main connections of the platysma muscle are, superiorly and medially, to the lateral part of the orbicularis oris muscle; and medially in the mandibular region, to the posterior border of the depressor anguli oris muscle. The platysma, together with the depressor anguli oris, pulls the oral commissures laterally downward, depresses the inferior part of the cheek, and tenses the skin over the neck.

Although many anatomical variations have been described, the platysma muscle usually has one of the three patterns.\textsuperscript{5,6,15,18} In 75 percent of cases, the medial fibers in the submental region interdigitate with the contralateral platysma muscle 1 to 2 cm below the chin. In 15 percent of cases, the medial fibers interdigitate with the contralateral platysma muscle in the submental region down to the thyroid cartilage. In 10 percent of cases, the platysma medial fibers do not interdigitate with the contralateral platysma muscle. The remaining vertical fibers of the medial border do not interdigitate with those of the contralateral platysma.

It is commonly thought that the appearance of the platysma muscle is dictated by sagging of the neck skin. However, in our study, we have found that the skin of the neck follows the location and action of the platysma muscle. Platysma bands are related to the platysma muscle and its anterior border. At rest, platysma bands appear because of muscle activity and are not caused by skin sagging.\textsuperscript{9} It is thought that the contraction of the free anterior fibers in the submental and neck regions creates the platysma bands.\textsuperscript{9,9} The platysma is also responsible for horizontal neck lines. In our clinical experience, the only exception is in very old patients, where compromised skin elasticity can make a minor contribution to the platysma bands.

Many surgical strategies for platysma band treatment have been proposed to improve or restore the definition of the neck. Most of them are based on the concept that muscle relaxation and skin sagging cause the muscular bands and therefore aim to tighten the drooping structures. These surgical options may treat superficial structures (i.e., skin, subcutaneous fat, and platysma muscle), deep elements (e.g., subplatysmal fat and digastric muscles), or both.\textsuperscript{15} Different techniques have been proposed, including lateral tightening of the superficial musculoaponeurotic system–platysma complex through facial lifting,\textsuperscript{19,20} submental midline and/or the lateral approaches,\textsuperscript{2,21} platysma muscle flaps,\textsuperscript{22} corset platysmaplasty,\textsuperscript{21,25} cervical lipectomy,\textsuperscript{24} submental lipectomy and platysmaplasty,\textsuperscript{8} platysma suspension procedures,\textsuperscript{4,25} and/or an isolated neck lift.\textsuperscript{3,21} Other suggested approaches include direct myotomy of the platysma bands\textsuperscript{1,26} or myectomy.\textsuperscript{14} Many procedures aiming to treat the deep structures have also been published in the literature; these include, subplatysmal fat approaches,\textsuperscript{27,28} digastic corset,\textsuperscript{29} and partial resection of the digastic muscle.\textsuperscript{30}
Despite the diverse surgical techniques, persistence or recurrence of platysma bands is frequent. Some authors report that bands can appear within 1 year postoperatively, and in most cases, recurrent banding appears when only a lateral skin pull is performed. The refinement of certain surgical techniques has yielded longer lasting outcomes, but reappearance of bands seems inevitable.

In our study, we observed that platysma bands are related to the activity of the platysma muscle. Therefore, we believe that the surgical paradigm focused on tightening of the neck’s structures should be changed. In our opinion, treatment should target the cause of platysma bands (i.e., muscular activity) rather than just its consequences, through denervation of this muscle. Platysma denervation may be performed by surgical and nonsurgical procedures. Skin excess will decrease because, as we noticed in our study, the skin follows the platysma bands and not the contrary (Fig. 4).

Chemical Denervation

Botulinum toxin injection is the main nonsurgical treatment for platysma bands and also has a role postoperatively to reduce the risk of recurrent bands. A detailed review of the extensive evidence supporting use of botulinum toxin in the management of platysma bands is outside the scope of this article. In general, botulinum toxin injection achieves good results. However, efficacy may be limited in cases of redundant cutaneous laxity, excessive lipodystrophy, or hypertrophic submandibular glands. Thus, to have satisfactory outcomes, it is important to remember that botulinum toxin may not be appropriate in all patients.

Moreover, the dose of botulinum toxin is controversial. Some authors inject a maximum of 20 U per platysma band. Others affirm that the dose must be correlated with the degree of the platysma deformity and can be increased up to a total of 250 U per case. Such pharmacologic denervation has a temporary clinical effect.

Surgical Denervation

Hyperkinetic movement disorders of the platysma constitute an aesthetic, functional, and psychological burden for patients and frequently induce the formation of platysma bands. In these cases, neurectomy of the cervical branch of the facial nerve—the motor branch of the platysma—has been successful. Surgical denervation of the platysma muscle by resection of the cervical branch of the facial nerve has been proposed as a treatment for muscular banding, with no recurrent neck longitudinal bands if not co-innervated by the mandibular branch. Our study clearly shows that in most of our patients, denervation of the platysma muscle prevented the formation of muscular bands. Therefore, in our opinion, the most effective and durable treatment of platysma bands is by targeted muscle denervation. There is a theoretical concern that dividing the main upper branch of the cervical nerve could produce undesirable and permanent lower lip depressor weakness. However, we did not see this in our patients.

Some authors have described the association of a few anatomical landmarks and measurements to predict the location of the bifurcation point when the cervicofacial trunk of the facial nerve divides into the mandibular and the cervical branches. This branching point can be placed approximately 2 cm below a perpendicular line running from the angle of the mandible to a mastoid-mentum.
The section of the cervical branch of the facial nerve led to no deficits (Fig. 5). The cervical branch of the facial nerve has been used with success for reconstructing the brachial plexus and has become a useful donor nerve. We use perioperative nerve stimulation to locate the right motor branch when performing surgical platysma denervation during neck plastic surgery.

Further studies need to fully characterize the efficacy and safety of denervating the platysma muscle to improve the neck appearance. In addition, our observations could have implications for other indications. For example, in patients with established banding, platysmaplasty to correct platysma descent would be appropriate. In these patients, neurotomy to prevent postoperative reanimation could reduce the risk of recurrent bands. Additional studies need to assess this possibility.

CONCLUSIONS

In this clinical study, the evaluation of the neck appearance in cases of nonspastic, unilateral, facial palsy revealed the absence of platysma bands on the paralyzed side in most patients. Thus, we conclude that platysma bands appear because of platysma muscle activity during the aging process and not secondary to skin sagging. Therefore, platysma denervation could result in total and permanent success in the treatment of neck muscular bands. Further studies need to fully characterize the efficacy and safety of denervating the platysma muscle to improve the neck’s cosmetic appearance.

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