A Danger Zone for Facial Expansion? 3 Cases of Facial Synkinesis Found after Buccal Expanding Therapy

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Abstract

Background: Facial tissue expansion is a routine method used to repair large areas of facial defects. However, complication rates are higher in the face than in the other parts of the body due to its unique neuromuscular distribution and dynamic nature. These include facial nerve injury manifested as transient facial nerve palsy as priorly reported.

Case Summary: Herein, we report another type of facial nerve injury. Three cases of facial synkinesis were observed after buccal tissue expansion and skin flap transplantation. During the follow-up period, there were no signs of improvement, suggesting that this may be an irreversible injury. Some hypotheses were made to discuss the probable factors driving this phenomenon, followed by strategies recommended to prevent this complication during the expansion procedure.

Conclusion: Facial tissue expansion in the cheek may cause facial nerve injury and lead to facial synkinesis, by the persistent non-physiological strong pressure originating from the expander. Therefore, special attention should be given when surgeons consider placing the expander in the cheek.

Keywords: Facial synkinesis; Facial tissue expansion; Facial nerve injury; Complications

Introduction

Since it was first reported by Neumann in 1957, tissue expansion has served as a creative solution for multiple reconstructive problems [1]. Compared to traditional operative procedures, tissue expansion has an overwhelming advantage as it allows for large area defects to be reconstructed with tissue of a similar texture and color with minimal morbidity in the donor site [2,3]. This technique is also commonly used on the face to resurface large areas of facial scars after severe burns or other kinds of traumas [3]. Nevertheless, complication rates in the face are higher than the other parts of the body due to its unique neuromuscular distribution and dynamic characters, ranging from 16% to 50% according to prior reports [4,5]. The observed complications include implant exposure or rupture, infection, hematoma, etc. occurring in the early stage of the expansion, and ectropion, flap necrosis, brow ptosis, etc. occurring in the late stage of the treatment procedure and usually requiring a planned and delayed revision surgery [4,5]. Notably, transient facial nerve palsy was reported in a patient receiving expander implantation in the cheek [6]. The symptom was developed at one week following the commencement of expansion. Although it recovered after removal of 10 cc of fluid from the expander, this phenomenon reminds us that buccal expanding therapy has a risk of causing facial nerve injury.

Facial synkinesis, which refers to abnormal involuntary facial movements that accompany volitional facial movements [7], has never been reported before as a complication of facial tissue expansion. The symptom of facial synkinesis varies including involuntary movements of the eyes, midface, or mouth corner when patients do volitional facial movements like eating, smiling, or closing their eyes. This phenomenon mostly appears in patients with enduring facial paralysis, as a sequela of facial nerve injury [8]. Apart from the functional limitations and esthetic affection, facial synkinesis can lead to social phobia and decreased quality of life [8]. So far, its pathogenesis has not been conclusively elucidated and complete relief from this annoying symptom is still difficult to achieve [7].

This paper, for the first time, reported the facial synkinesis symptom observed after buccal tissue expansion, sought to identify factors in expansion procedure that may contribute to the development
of facial synkinesis and also, discussed possible strategies for the prevention of this complication during the reconstructive process.

Case Presentation

The first patient was a 33-year-old woman suffering from burns on her right face and right upper arm due to a car accident 1 year ago. She received skin grafting right after the burns but gradually showed massive atrophic scars in the skin-grafting area. The patient asked for help in our hospital to improve the scar's appearance. She underwent right facial tissue expander implantation under general anesthesia. Regular water injection was performed twice a week during the expansion procedure (Figure 1A). Seven months later, the expander was removed followed by facial scar excisions and flap transplantations. The course of anesthesia and operation was smooth, and the patient was discharged 3 days after the surgery. During routine follow-up half a year after the surgery, the doctor observed facial synkinesis on the surgical side, that’s when she closes her eyes, and her right cheek is lifted up simultaneously and unconsciously (Video 1).

The second patient was a 25-year-old female who presented with scar contracture deformity in her left face due to a burn accident 23 years ago. The contracted scar led to patient’s crooked left mouth corner and restricted mouth opening. She sought help in our hospital intending to alleviate the deformity. The patient received expander implantation in the left cheek under general anesthesia. After four months of regular water injection and tissue expansion (Figure 1B), the expander was taken out and skin flap transplantation was performed with the patient under general anesthesia. The whole procedure of the operation was smooth, and the patient was discharged 3 days after the surgery. Similar to the first case, the doctor observed symptoms of facial synkinesis in her right face half a year after the expander was removed (Video 2).

The third patient was a 21-year-old female who presented with scar contracture deformity in her left face and neck due to a burn accident 19 years ago. The patient came to our hospital with the aim of improving her neck and facial deformity. She underwent expander implantation in the left cheek and the left shoulder under general anesthesia (Figure 1C). The expansion procedure lasts for seven months and then she received expander removal and skin flap transferring under general anesthesia. The whole procedure of the operation went smoothly, and the patient was discharged 3 days after the surgery. During a routine follow-up half, a year after the reconstructive surgery, the doctor found that she showed symptoms of facial synkinesis on her left face (Video 3).

For all three patients, the symptom of facial synkinesis still exists at the latest follow-up (12 to 18 months after the expanders were removed) (Table 1). Other basic information about patients was recorded in Table 1. Synkinesis Assessment Questionnaire (SAQ)
was used to evaluate the frequency/severity of symptoms, on a scale from 1 to 5 [9].

**Discussion**

**Possible etiology and pathogenesis of facial synkinesis**

Although the underlying pathophysiology has not been fully elucidated, facial synkinesis is postulated to result from anomalous structures or functions at multiple levels along the neural pathway [7]. Pathological changes including (1) peripheral ephaptic transmission of impulses between axons, (2) aberrant regeneration of facial nerve fibers, and (3) synaptic reorganization within the facial nerve nucleus have all been presented to be involved in its pathogenesis [7]. Currently, the most widely accepted theory is the “crosstalk” between distal nerve fibers which may result from the ineffective myelination...
and the nonspecific aberrant reorganization of neural networks [10,11].

Over-expansion in some cases shows the advantages of increasing the flap size and flap survival rate [12], hence it is commonly adopted by surgeons to promise the efficiency of the following skin flap transplantation. Considering that all three cases in this report experienced over-expansion during the treatment procedure, we speculate that pressure may be the dominant factor driving facial nerve injury in these cases. The facial nerve may suffer from non-physiological supramaximal pressure in the over-expansion state. The ischemic state caused by the chronic supramaximal pressure compressing important blood vessels nourishing the nerve fibers is an important mechanism resulting in nerve injury. Chronic ischemia can cause considerable morphological abnormalities in peripheral nerves. The severity of nerve injury caused by ischemic differs and is determined by the degree and duration of ischemia, which includes one or more morphological changes of follows: Demyelination and remyelination, endoneurial edema, and various but infrequent axonal changes [14]. Chronic ischemia can cause considerable morphological abnormalities in peripheral nerves. The severity of nerve injury caused by ischemic differs and is determined by the degree and duration of ischemia, which includes one or more morphological changes of follows: Demyelination and remyelination, endoneurial edema, and various but infrequent axonal changes [14]. During remyelination, the axon becomes remyelinated by the resident Schwann cells. However, this myelination is generally much thinner than normal [15], with predictable electrical consequences which may have a relationship with the development of facial synkinesis (Figure 2). If axonal degeneration happens, regeneration follows and this will be discussed later.

Pressure can also cause nerve injury by direct mechanical effects independent of ischemia [16,17]. The majority of peripheral nerve injury caused by compression fall under the class of neurapraxia, or Grade I nerve injuries (Table 1) [15]. In experiments on baboons, researchers found that the applying of pneumatic tourniquets to the thigh caused mechanical nerve injury in myelinated fibers characterized by a partial invagination (intrusion) of one side of the node of Ranvier into the other (Figure 2) [17], which is distinguished from ischemic injuries characterized by axonal degeneration of both myelinated and unmyelinated nerve fibers [14]. Damage to the myelin sheath may lead to the leaking of nerve impulses and subsequently the stimulation of the neighboring nerve (which may also suffer from myelin sheath damage), resulting in anomalous transmission of impulses between axons [18-21]. Of note, compression injuries can also be captured by Grade II to Grade IV of the commonly used classification schemes (Table 2) and nerve regeneration will be involved during recovery, which will be discussed later.

Pressure from the expander may also change the distribution of facial nerve branches. A study on tourniquet-related nerve injury demonstrated that the axoplasm and myelin under the point of the greatest compression of the cuff were pushed away and toward the edges of the cuff [22]. The shift of nerves caused by the direct mechanical effects of pressure may enhance “miscommunication” between nerves with damaged myelin sheath (Figure 2).

Another possible mechanism involved in the facial synkinesis in these patients is aberrant nerve regeneration after nerve injury. Many investigators believe that the facial synkinesis occurring after peripheral facial palsy is due primarily to the misdirection of regenerated axons that innervate incorrect muscle groups. According to Sunderland’s classification scheme, nerve regeneration only exists in moderate to severe nerve injuries (Grade II to Grade V) (Table 2), in which situation patients usually show sensory and/or motor

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
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<tbody>
<tr>
<td>History of facial paralysis</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Anomalous movement in the buccal area before the surgery</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Anomalous feeling in the buccal area before the surgery</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Facial area of expander placed</td>
<td>Right cheek</td>
<td>Left cheek</td>
<td>Left cheek</td>
</tr>
<tr>
<td>Duration of expansion (months)</td>
<td>7</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Anatomical layer of expander placed</td>
<td>Subcutaneous</td>
<td>Subcutaneous</td>
<td>Subcutaneous</td>
</tr>
<tr>
<td>Frequency of water injection (times per week)</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Specifications of expander (mL)</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Amount of water injection (mL)</td>
<td>260</td>
<td>180</td>
<td>260</td>
</tr>
<tr>
<td>SAQ</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Area of synkinesis</td>
<td>Ocular-buccal-oral</td>
<td>Ocular-buccal</td>
<td>Ocular-buccal</td>
</tr>
<tr>
<td>Anomalous feeling in the buccal area after the surgery</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>The latest follow-up (months after discharge)</td>
<td>18</td>
<td>13</td>
<td>12</td>
</tr>
</tbody>
</table>

SAQ: Synkinesis Assessment Questionnaire

Table 2: Classification of nerve injury.

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Demyelination</td>
<td>Grade I</td>
<td>Neurapraxia</td>
</tr>
<tr>
<td>Axon damaged with maintained endoneurial tubes</td>
<td>Grade II</td>
<td>Axonotmesis</td>
</tr>
<tr>
<td>Axon and endoneurium damaged with maintained perineurium</td>
<td>Grade III</td>
<td>Axonotmesis</td>
</tr>
<tr>
<td>Axon, endoneurium, and perineurium damaged with intact epineurium</td>
<td>Grade IV</td>
<td>Axonotmesis</td>
</tr>
<tr>
<td>Complete transection of nerve</td>
<td>Grade V</td>
<td>Neurotmesis</td>
</tr>
</tbody>
</table>
abnormalities in the controlled area before synkinesis occurs [15], just like the pathogenetic progress of facial paralysis. Although we didn’t observe any symptoms of Grade II to Grade V nerve injuries in our patients, the symptom may be ignored during the expansion, in which process the patients carried large expanders on one side of the cheeks, making it hard to distinguish abnormal facial movements in a state of extreme asymmetry. The majority of Grade V injuries are transection injuries, which is unusually seen in nerve injuries caused by compression [15]. The most probable situation in these three patients, if nerve degeneration had happen, is injuries of Grade II to Grade IV, corresponding with axonotmesis classified by Seddon (Table 2). Axonotmesis may result from the direct mechanical effects of pressure and/or the chronic ischemia state caused by the high pressure. After axonotmesis, nerve regeneration can occur in two mechanisms: Collateral branching or axonal regeneration. In injuries where greater than 90% of the axon population within a nerve were affected, axonal regeneration is the primary means for recovery, while when 20% to 30% of the axons are damaged, collateral branching is the primary mechanism of recovery [23]. During the regeneration process, more axonal branches will sprout than the actual number of nerves that eventually innervating a target-organ [15]. Although theoretically, those branches that can’t receive neurotrophic factors from the target-end organ are destined to degenerate, there is a possibility that some of the branches ending in the wrong muscles survive and innervate these muscles, leading to synkinesis (Figure 2) [15]. In sum, facial synkinesis caused by non-specific axons regeneration cannot be excluded from these three patients.

Although the causal link between the above pathological changes and facial synkinesis has not been fully revealed yet, pressure is certainly a key factor that surgeons must take into consideration for the prevention of nerve injury during buccal tissue expansion. A priori study using a rat sciatic nerve model has demonstrated that pressure of 30 mmHg leads to myelin damage and 80 mmHg applied over 2 h resulting in axonal loss [24]. Despite that these values may not be a guide for buccal tissue expansion, it reminds us to control the pressure when using expanders in those areas with abundant nerve distribution, and for that purpose, a new generation of expanders with a pressure monitoring system will make sense.

**Strategies to prevent facial synkinesis during facial tissue expansion**

The self-healing of facial synkinesis is rarely reported. Although nowadays several approaches including chemodenervation [25], physiotherapy [26,27], and selective neurectomy show some effects [28], the complete relief of the symptom is still difficult to achieve. Therefore, prevention of this complication during facial tissue expansion is of particular importance.

It is well-known that the types and rates of complications of facial tissue expansion are tightly related to the location of the expander placed [29]. These three cases underwent the expansion procedure in the same facial area-the cheek, where closely distributed the zygomatic branches and the (superior and inferior) buccal branches of the facial nerve [30]. Some terminal branches of these nerve fibers intermesh (Figure 3) and that makes the buccal area a danger zone for facial expansion. Compared to the cheek, other areas, including the neck and forehead with simpler distributions of mimetic muscles and nerves (Figure 3), are more unlikely to show symptoms of synkinesis even if suffering damage.

Our first advice is to avoid the use of expanders in the buccal area if alternatives exist. If inevitably, over-expansion is not recommended, and if possible, monitoring the pressure of the expander may guide the expansion procedure. Duration of the expansion is another factor that surgeons should consider. The length of time may be flexible, determined by how much the pressure is. Unfortunately, thresholds of these factors (pressure value and duration time) have not been established yet, hence, further research seeking a cut-off value is necessary for the establishment of the guideline.

**Conclusion**

Overall, facial tissue expansion should be given more attention compared to the other parts of the body, especially the expansion in the buccal area. In addition to the routine precautions, preventing facial nerve injury may be the primary consideration when the surgeons are planning to place the expander in this area. For this purpose, pressure and the duration time of the expansion must be controlled strictly if the operation is inevitable.

**Limitation**

The limitations of this report include the first, despite some probable explanations for facial synkinesis being discussed, this retrospective review cannot identify its etiology and pathogenesis, and further animal research and *in vitro* studies are necessary to elucidate the underlying mechanism. Secondly, we cannot tell the specific time of facial synkinesis onset. Patients might have developed synkinesis at any time during the expansion process, but doctors may ignore it due to the carried expanders making these symptoms unobservable. Nevertheless, this report can still provide some practical guides for the facial reconstructive process.

**Acknowledgment**

We thank all three patients for their support of this report.

**Ethical approval**

This report was approved by the Ethics committee of West China Hospital, Sichuan University, and written informed consent was obtained from each patient before the use of their information.

**Patient Consent Statement**

Written consents were obtained from all participants for the photos and other medical records.

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**References**


