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Facial palsy after dental treatment

Abstract: Facial nerve palsy may occur in rare cases in connection with dental treatment. Its causes have not been entirely clarified so far. If facial nerve palsy sets in immediately after the administration of a local anesthetic, it is likely that the unintended, accidental anesthesia of the branches of the facial nerve has occurred. Once the effect of the anesthetic subsides, the palsy disappears and is completely reversible. Direct damage to the facial nerve with the injection needle seems improbable. Delayed facial nerve palsy, which initially sets in hours or days after dental treatment, must be considered separately. In the past, various causes have been discussed in literature. Presently, it is thought that the most likely cause is the reactivation of viruses (herpes simplex virus type 1 or varicella zoster virus) from nerve ganglia of the facial nerve by dental treatment. This can also occur in cases where no local anesthesia has been used. In addition, this does not have to result in the formation of blisters on the skin (zoster sine herpete), which is typical of herpes labialis or herpes zoster. In order to treat delayed onset facial palsy after dental treatment, drug therapy with glucocorticoids (prednisolone) and antivirals (acyclovir) is the current recommendation. For successful therapy, the drugs should be administered in less than 72 hours after the onset of the first symptoms. Dentists should thus promptly refer patients to a neurologist and otherwise be aware of the potential (irreversible) damage to the facial nerve by the viruses.

Keywords: Treatment complications; Bell's Palsy; facial nerve palsy; local anesthesia; viral infection; dental treatment

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Translation from German: Cristian Miron
Citation: Dammaschke T: Facial palsy after dental treatment. Dtsch Zahnärztl Z Int 2021; 3: 225–230
Peer-reviewed article: submitted: 21.07.2020, revised version accepted: 22.09.2020
DOI.org/10.3238/dzz-int.2021.0027

The facial nerve

The facial nerve is the 7th cranial nerve and it emerges from the skull via the stylomastoid foramen. Before the facial nerve emerges from the skull, the chorda tympani branches off from it at the level of the petrous bone. It supplies preganglionic secretomotor fibers to the submandibular as well as sublingual salivary glands and carries efferent taste fibers from the front two thirds of the tongue with the exception of the lingual papillae. After emerging out of the skull, the facial nerve divides into 2 main branches – the temporal and cervical – before it enters the parotid gland, where it continues to divide into the temporal, zygomatic, buccal, mandibular and cervical branches, which eventually supply the mimic muscles [6, 31].

In literature, a localized, but very rarely reported neurological complication after routine dental procedures under local anesthesia is the loss of function of the facial nerve (facial palsy). Consequently, no numbers can be derived from literature to determine the frequency of facial palsy after dental interventions. In comparison, the so-called idiopathic facial nerve palsy, in which there is no recognizable cause or triggering moment, is more frequently described. Idiopathic facial palsy affects approximately 7–53 patients per 100,000 persons and year across all population groups [17, 45]. The idiopathic facial palsy is called Bell's Palsy and it is basically defined as a peripheral partial or total loss of function of the facial musculature without an obvious cause. The exact mechanism is still not clear [5, 11, 17, 42, 45]. Five etiological factors are most frequently associated with idiopathic facial nerve palsy: special anatomical structures deviating from the norm, viral infections, ischemia, inflammation and cold stimulation (weather, cold and draft exposure) [17, 45].

Peripheral facial nerve palsy can also occur during dental treatment, either immediately or delayed, depending on the time elapsed from the moment the anesthetic is injected until the onset of symptoms [42]. The occurrence of facial nerve palsy after dental treatment requires a correct diagnosis and immediate, appropriate therapy.

Patients with peripheral facial palsy have characteristic clinical features, which include: generalized muscular weakness of the ipsilateral (same) side of the face, absence of frowning, eyebrow drooping (brow ptosis), incomplete closure of the eyelids, disappearance of the nasolabial fold, drooping of the corner of the mouth and deviation of the mouth towards the unaffected side. Additionally, patients may also complain of pain in the retroauricular region and a reduced sense of taste [6, 17]. Clinically, peripheral facial palsy can be distinguished from central palsy (e.g. stroke) through the involvement of the forehead. If the function of the forehead is intact and the middle and lower parts of the face affected, this indicates a central ("supranuclear") lesion, since the forehead muscles are supplied by nerve fibers from both hemispheres and therefore maintain their function in central lesions. In contrast, peripheral nerve palsy is a lesion of the lower motor neurons and therefore it affects all muscles of the face. The lower nucleus of the facial nerve only receives a one-sided contralateral cortical projection and it supplies the lower facial muscles [6, 17, 22].

Direct nerve injury through injection cannula

In principle, the touching of a nerve with an injection needle during dental anesthesia seems sufficient to cause trauma, which can lead to paresthesia (loss of sensation up to a feeling of numbness) in the area supplied by the sensory nerve. Fortunately, most paresthesias disappear without treatment within a period of 8 weeks. The paresthesia can be permanent only when the damage to the nerve is severe, but this is a very rare occurrence [4]. It has been described in literature that an incorrectly placed injection needle can also lead to facial nerve palsy, e.g. motor neuron failure [2]. However, a direct trauma of the facial nerve due to the injection needle seems very improbable. Nerves have a diameter of about 2–3 mm and consist of a multitude of fascicles. In comparison, an injection needle that has an outer diameter of < 0.5 mm would cause little direct trauma. Additionally, the injection needle can run into the nerve so that it slides between the individual fascicles without cutting the entire nerve. It is unlikely that a needle with a diameter of < 0.5 mm can cause such extensive damage to the entire nerve, so as to result in facial nerve palsy [27]. During the injection, many patients immediately feel an "electric shock" when the needle touches the branch of a sensory nerv. When patients perceive this "electric shock", it does not necessarily mean that it will result in irreversible nerve damage. In the case of 200 dental nerve block anesthesia, 7 patients reported having sensed an "electric shock". Of the 7 patients, only one suffered from a sensory disturbance which completely subsided within 2 weeks [14]. The symptom of an "electric shock" is usually completely reversible without any nerve damage. Therefore, a direct trauma via an injection needle is probably not responsible for facial nerve palsy [27].

However, the injection may lead to the formation of an intraneural hematoma. The needle can increase the tonicity of the small blood vessels that run within the epineurium, producing bleeding in the nerve, which can then lead to the compression and fibrosis of the nerve. This compression may occur relatively quickly (within 20–30 minutes) so that nerve damage arises before the local anesthesia subsides. The patient would not be aware of the increasing pressure on the nerve and the resulting damage [27, 28], but would perceive the nerve damage, and the resulting consequences, immediately after the anesthesia subsides. Moreover, these explanations do not explain the delayed onset of facial palsy hours after the local anesthesia has subsided and/or the involvement of chorda tympani together with the associated impaired taste sensation, described in facial palsy after dental treatment [6, 43].

Immediate palsy of the facial nerve after dental treatment

Acute facial palsy, which occurs during or immediately after a dental pro-

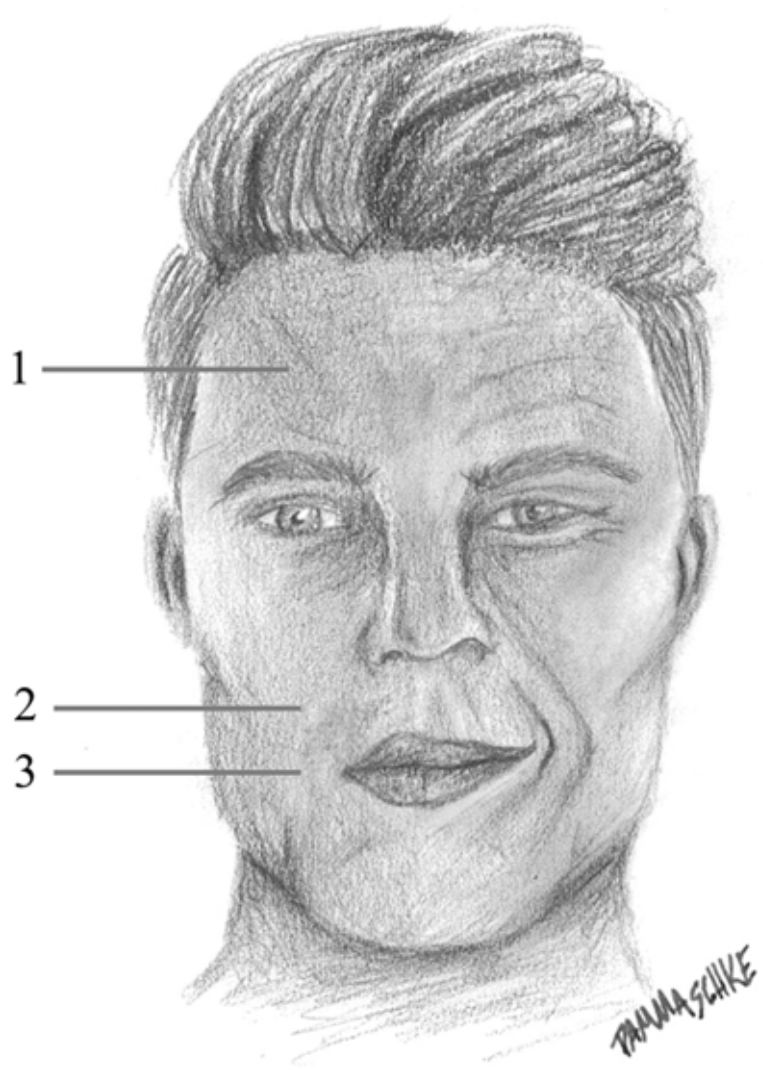

Fig. 1: Sophie Dammaschke

Figure 1 Clinical signs of right-sided peripheral facial palsy: 1. lack of frowning; 2. disappearance of the nasolabial fold; 3. drooping of the corner of the mouth and deviation of the mouth towards the unaffected side

cedure, is usually the direct result of a complication during local anesthesia. It can start immediately after the injection of a local anesthetic and it occurs on the ipsilateral side of the dental procedure. An immediate, direct palsy of the facial musculature thus results from the direct accidental anesthesia of one or more branches of the facial nerve. Normally, in the immediate type, this palsy occurs within minutes of the injection, with a recovery time of 3 hours or less; however, in exceptional cases, it can take 12–24 hours [3, 5, 6, 11, 13, 21, 42, 43]. The visible palsy of the mimic musculature is always temporary, and as the anesthetic subsides, this disappears completely without leaving any permanent damage [20].

Acute facial palsy can occur if the injection is accidentally injected too far posteriorly, thus causing the anesthetic solution to be injected close to or even into the parotid gland. The deep lobe of the parotid gland extends around the posterior ramus of the facial nerve and projects forward on the medial surface of the ramus. In most cases, the parotid gland surrounds the facial nerve and an injection in the vicinity of the parotid gland consequently results in the direct anesthesia of the facial nerve [4, 20, 34].

However, there are also anatomical variations in which the parotid gland does not envelop the facial nerve and its branches [6], or the facial nerve's branches in the retromandibular space appear abnormal [5, 21]. Such deviations from the normal anatomy increase the probability of direct exposure to an anesthetic, even if the anesthesia is correctly performed.

It must be emphasized that some authors believe that it is difficult to anesthetize the facial nerve in this manner through the oral cavity [5, 43]. Moreover, even with these explanations, it cannot be elucidated how a delayed onset of facial palsy occurs hours after the local anesthesia has subsided and/or the involvement of the chorda tympani with the associated taste disturbance [6, 43].

Delayed palsy after dental treatment

In the delayed type of facial nerve palsy, symptoms appear within a few hours to several days after dental treatment, while the recovery period can extend from 24 hours to several months [6, 21, 42]. The pathogenesis of the delayed type is more complex than that of the immediate type and it has not been ultimately clarified. Consequently, the exact cause can only be speculated [27, 42], but in literature, the following theories have been discussed:

Compression edema due to the sympathetic vascular reflex

Facial nerve palsy can possibly be the result of a sympathetic vascular reflex that leads to ischemia in the area of the stylomastoid foramen. The anesthetic solution, its respective decomposition products, or the mechanical action of the injection needle itself stimulates the sympathetic nerve plexus connected to the external carotid artery (vegetative nerve plexus). From the external carotid artery, the fibers of this plexus continue to remain in contact with the stylomastoid artery (a branch of the occipital artery in 66 % of cases and a branch of the auricular artery in the remaining cases) until they enter the parotid gland. Stimulation of the sympathetic stylomastoid plexus leads to a delayed reflex spasm of the vascular nerves of the blood vessels that supply the facial nerve. This leads to ischemic neuritis and secondary edema. These sympathetic

nerve fibers originate in the upper cervical ganglion, from which the lateral, medial and anterior branches arise. From these, it is the anterior branches that follow the common and external carotid arteries and form plexuses that accompany the blood vessels [6, 21, 40]. In short, the anesthesia indirectly leads to a reflex-induced vasospasm of the stylomastoid artery and a retrograde epidural compression edema of the facial nerve in the bone canal of the stylomastoid foramen, and consequently, to ischemia of the nerve [32, 44].

Allergic reaction

The possibility of a localized allergic reaction was also considered. The delayed onset of the reaction and the rather generalized peripheral nerve dysfunction [12] would argue for this. However, in literature, there are no further references to this theory.

Aromatic alcohols

Furthermore, there have been considerations as to whether an alternative reaction path in the chemical decomposition of the local anesthetic could lead to the formation of aromatic alcohols (phenols) around the nerves. These aromatic alcohols could give rise to delayed chemical nerve damage [27, 29, 43].

Prolonged mouth opening

Moreover, prolonged mouth opening during treatment, which results in an overstretching of the facial nerve, has been associated with facial palsy [3].

There is no scientific evidence for any of the theories mentioned above so far. Therefore, it must be emphasized that these are merely attempts at an explanation.

Viruses as the cause of delayed facial palsy

Presently, the most accepted theory that explains why delayed facial palsy can occur after dental treatment is a latent virus infection; dental treatment and/or anesthesia can reactivate viruses such as the herpes simplex virus type 1 or varicella zoster virus [11, 18, 25, 42].

The mechanism leading to viral reactivation in nerve ganglia is known to be caused by varicella zoster viruses in the case of shingles (herpes zoster) for instance. Primary infection with the varicella zoster virus leads to chickenpox in childhood. Even after complete recovery, varicella zoster viruses persist for life in the brain and spinal ganglia. Reactivation of the varicella zoster virus (usually by stress or immunodeficiency for example) leads to shingles that is accompanied by characteristic painful unilateral and dermatome-related skin redness with blistering. The same applies to infection with herpes simplex virus type 1; although the primary infection often goes unnoticed in childhood, reactivation leads to a typical blistering of the lips (herpes simplex labialis) subsequently. However, these skin changes do not necessarily have to occur (zoster sine herpete) or they can be delayed in up to half of patients [18]. Similarly, this applies to facial palsy too. Some patients show no herpetic skin changes neither before nor after the onset of facial palsy [9]. Reactivation of the varicella zoster virus was demonstrated in 29 % of patients with facial palsy without causing skin changes (zoster sine herpete) [10]. For this reason, it is currently believed that zoster sine herpete is the cause of nearly one third of facial palsies that were previously diagnosed as being idiopathic [37].

When facial palsy occurs, the viruses from the geniculate ganglion, which is part of the facial nerve, are probably reactivated. After their reactivation, the viruses destroy ganglionic cells and spread into the endoneurial fluid along the motor branches of the facial nerve. The viruses infect Schwann cells, which leads to demyelination and inflammation of the facial nerve [1, 5, 6, 30, 32, 41]. Inflammation of the nerve initially leads to reversible neurapraxia (dysfunction of the nerve), but ultimately to Wallerian degeneration. Wallerian degeneration refers to a complex molecular process that occurs after damage to a nerve in the peripheral nervous system (PNS) or to fiber tracts in the central nervous system (CNS) and, viewed from the site of damage, it results in the destruction of the nerve component located distally of the perikaryon. The varicella zoster virus shows a more aggressive biological behavior than the herpes simplex virus type 1 because it spreads via satellite cells (= mantle cells, which coat the cell body of the neurons) across the nerve [18].

This theory of the viral etiology of facial palsy was published almost 50 years ago [1, 23] and has been confirmed in various studies [26, 33]. In particular, the herpes simplex virus is suspected to be the causative agent of facial palsy based on investigations in molecular biological [8]. Herpes simplex virus genomes could be detected in facial nerve cell fluid in 79 % of patients with acute facial palsy using polymerase chain reaction (PCR) techniques [25]. Moreover, it has been shown in animal experiments that herpes simplex viruses have the ability to induce facial palsy [7, 15, 35, 38].

Various mechanisms can trigger the reactivation of viruses, despite the fact that the exact mechanism is not always known. At present, though, it is assumed that this can also happen through dental treatment. In cases where viruses are reactivated by dental treatment, this is not necessarily related to the administration of a local anesthetic. It is important to note that in 1/3 [8] to 1/2 [11] of cases, where delayed facial palsy occurred after dental treatment, no local anesthesia was used. Even comparatively less invasive dental treatment measures that do not require local anesthesia, such as professional tooth cleaning, caries excavation, filling therapy and the fitting of dentures, have been shown to reactivate viruses, and consequently, lead to facial palsy [8, 11].

Differential diagnoses

Besides the already mentioned central facial palsy that results from a stroke for instance, peripheral facial palsy should be distinguished from a number of diseases that have similar clinical features, but which are clearly not related to dental treatment. The list of differential diagnoses includes, for example, anatomical variations in the path of the nerve, ischemia (inadequate blood flow), trauma (fractures of the petrous bone), surgical in-

juries, acoustic neuroma, otitis media, malignant parotid tumors, Lyme disease, various infectious diseases (Rickettsia-, HIV, mumps, cytomegaly, rubella infections), syphilitic or tuberculous basilar meningitis, sarcoidosis (Heerfordt's syndrome), Ramsay-Hunt's syndrome, Guillain-Barré's syndrome, Sjögren's syndrome, Melkersson-Rosenthal's syndrome, Miller-Fisher's syndrome, meningeosis carcinomatosa [17, 24, 40, 42, 45].

Therapy and healing

There is hardly any data on the prognosis of delayed peripheral facial palsy in connection with dental treatment in literature. In principle, it can be assumed that the prognosis of idiopathic facial palsy is good even without therapy. A complete recovery can be expected in circa 70 % to 94 % of patients after 6–9 months [17–19, 45]. Full recovery can take up to 24 months (or longer) [11]. Yet, in a few patients, facial palsy does not heal if left untreated [18].

The evidence-based therapy of choice for idiopathic facial palsy is the oral administration of glucocorticoids (prednisolone). The therapy should begin as soon as possible after the onset of symptoms (< 72 hours) [17, 36]. The hypothesis that dental treatment can reactivate viruses, which then result in the development of facial palsy, suggests that glucocorticoid administration should be supplemented by virostatic therapy (acyclovir). Thus, prednisolone in combination with acyclovir should improve the probability of a complete recovery. In this manner, the healing rate of patients treated with prednisolone combined with acyclovir was 95.7 %, which is higher than that of patients treated with prednisolone alone (88.6 %) [16]. Nonetheless, the available studies on idiopathic facial palsy are relatively heterogeneous. In particular cases, a treatment that combines glucocorticoids and antivirals can be considered after discussing the findings with the patient [17]. Especially if there is suspicion that the dental treatment could have reactivated viruses, additional antiviral therapy is absolutely necessary. Prophylactic antiviral therapy prior to dental surgery can be helpful for preventing recurrent facial palsy in patients who have had prior facial palsy or who complain of recurrent herpes infections [11].

In addition to oral drug therapy, symptomatic therapy can be recommended if necessary; for example, in cases of insufficient eyelid closure, artificial tears, dexpanthenol eye ointment and nocturnal watch glass dressing can be used [17, 44]. Conversely, a recent meta-analysis on physical therapy (physiotherapeutic exercises, mimic exercises, electrotherapy) showed neither a significant benefit nor a significant harm [39].

Principally, the exact diagnosis and therapy of delayed facial palsy is the responsibility of a specialist physician. A referral to a neurologist for further therapy should be made as soon as possible after the first signs of facial palsy appear. The physician should be informed about the possible reactivation of viruses through dental treatment.

Conclusion

A delayed onset of facial palsy after dental treatment can possibly be related to the reactivation of viruses (herpes simplex virus type 1 or varicella zoster virus) from nerve ganglia. Dentists should be aware of the potential for irreversible damage to the facial nerve by the viruses and the need for early drug therapy, which is the neurologist's responsibility. An immediate referral to a specialist is therefore necessary.

Acknowledgements

I would especially like to thank Sophie Dammaschke for creating the illustration.

Conflict of interest

The author declares that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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