



## Paradigm shift in rehabilitation of long-standing Bell's palsy during later stages of recovery

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Bell's palsy (idiopathic peripheral neuropathy of the facial nerve) is an acute, usually unilateral paralysis of facial muscles. This disorder had been described and studied by Sir Charles Bell in 1836. In majority of Bell's palsy cases the patient achieves full spontaneous recovery within 3-5 weeks even without treatment. In about 15-30% of cases the recovery takes considerably longer time (3 to 6 months, sometimes longer) and is usually incomplete. After the incomplete recovery of Bell's palsy some residuals and complications usually develop. Those can include weakness of facial muscles, facial asymmetry in both neutral expression and with facial movements, pathological synkinesis, contractures of facial muscles, crocodile tears, facial pains etc.

The above-mentioned complications may result in disfigured face with various degrees of severity, involuntary facial movements during speech and emotions, facial pains that are difficult to relieve with pain-killers, inability to adequately express emotions. All the named manifestations can have a dramatic impact on individual's self-esteem, social contacts and general quality of life. They may lead to stress, depression, self-isolation and in some extreme cases – to suicidal behavior.

Customary treatment for acute Bell's palsy include prednisone, antiviral medications, eye care to prevent drying of the cornea, mime-therapy (neuromuscular retraining). Sometimes acupuncture, chiropractors, laser therapy or high doses of B-vitamins are used. In some countries such controversial interventions as electrical stimulation (shocks) and massage with ice-cubes are being used as treatment modalities for acute Bell's palsy. If the facial condition does not considerably improve within the first year, then the choice of treatments for residuals and complications of long-standing Bell's palsy is rather limited. It includes continuation of mime-therapy, periodic injections of botulinum toxin, and in some exceptional cases surgical interventions (nerve graft, muscle transposition etc.) Facial nerve decompression has not been proven to be effective as Bell's palsy treatment and therefore is nowadays seldom used. Long-standing Bell's palsy patients are left with very limited choice of treatments for their residuals and complications. In the medical world and among rehabilitation specialists it is considered that if Bell's palsy had not recovered within first 1-2 years and if complications had formed, then the patient will retain the developed synkinesis, contractures, related facial pains and other manifestations for the rest of his or her life.

In the recent years there has not been much research done of the underlying causes for the complications of long-standing Bell's palsy. Without thorough understanding of such causes it is rather challenging to compose proper rehabilitation programs that deliver the desired improvements in facial symmetry and respectively, in patient's quality of life.

In this article we discuss the traditional approach to possible causes, available treatments of Bell's palsy complications and share our concept of a new paradigm in rehabilitation of long-standing Bell's palsy during later stages of the recovery (2 years and more). According to this new paradigm, synkinesis, contractures and mass movements of facial muscles, as well as related chronic facial pains are the result of newly-formed, pathological mimetic patterns in the CNS, which can be reduced and then reversed by applying the new approach to rehabilitation, based on other than

traditional principles. The results achieved by our patients can serve as an evidence that this new approach may indicate a beginning of a paradigm shift in rehabilitation of long-standing Bell's palsy.

*Keywords: Bell's palsy; facial palsy; synkinesis; contractures; facial pain; facial asymmetry; crocodile tears.*

## Introduction

Peripheral neuropathy of facial nerve is the second most common disorder of peripheral nerve system. On average, each year Bell's palsy affects 25 persons for every 100.000 of population. This disorder was first described and studied by Sir Charles Bell, and therefore had received his name. There is no consensus about the actual causes of Bell's palsy. During the last decades the authors of publications often name *herpes simplex* virus as the most probable cause. Quite often Bell's palsy occurs as a result of local over-cooling (sleeping under the air-condition, driving a car with both windows open, riding a bicycle with wet hair in a cold and windy weather etc.)

Other risk factors for Bell's palsy are hypertension, diabetes mellitus and last weeks of pregnancy. It is unclear why last weeks of pregnancy increase the risk of Bell's palsy occurrence. Perhaps it can be attributed to focal changes of blood circulation in the body of a pregnant woman that may lead to disruption in blood supply to the facial nerve.

According to various sources, full spontaneous recovery after idiopathic facial palsy (Bell's palsy) occurs in 70-85% of cases. Such recovery normally takes place within 2 to 5-6 weeks after the onset, often without any treatment.

Customary treatments for acute Bell's palsy include prednisone, antiviral medications, eye care to prevent drying of the cornea, mime-therapy (neuromuscular retraining). Sometimes acupuncture, massages, chiropractors, laser therapy or high doses of B-vitamins are being used at patients' own initiative in an attempt to facilitate the recovery. In some countries, such controversial interventions as electrical stimulation (shocks) and massage with ice-cubes are being used as treatment modalities for acute Bell's palsy.

In the remaining 15-30% of Bell's palsy cases the severity of nerve damage results in degeneration of axons in the acute phase. The regeneration of the nerve in most of these cases is incomplete. Such patients can experience complete immobility of one side of the face from 3 weeks to 3-4 months, and in some rare cases even longer. Recovery in such cases happens slowly and can take from 6 months to 1 year or more. Usually during the long recovery period various residuals and complications may be formed.

Residuals and complications of long-standing Bell's palsy can include:

- Weakness of facial muscles
- Facial asymmetry at rest and/or during facial movements
- Incomplete closure of the eye
- Bell's phenomenon
- Increased tone of mid- and lower face muscles on the healthy side
- Contractures (hyper-tone) of facial muscles on affected side

- Synkinesis (involuntary contractions of facial muscles that accompany voluntary movements)
- Rigidity of facial muscles, pulling sensations in the face and neck
- Mass movements during speech, emotions, eating etc.
- Crocodile tears (excessive tearing during eating or speech)
- Related chronic facial pains in certain points or areas of the face, head and neck– either spontaneous or from touch
- Headaches on the affected side that often cannot be relieved by regular pain-killers

The existing approach to rehabilitation of the patients with long-standing Bell's palsy offers a rather limited choice of treatments aimed at reduction of common residuals and complications of this disorder during later stages of recovery (1 year and later). It includes mime-therapy (neuromuscular retraining), phono-audio therapy (logopedic sessions), periodic injections of botulinum toxin and in extreme cases – surgical interventions (nerve graft and muscle transposition).

At the moment there is no clear understanding and no consensus among the specialists about what are the causes for forming of complications of Bell's palsy, what are the mechanisms of their manifestations and which fundamental issues must be particularly addressed in order to reduce and reverse synkinesis and contractures, to eliminate facial pains and excessive tearing, to improve facial symmetry in dynamics, increase self-esteem of long-standing Bell's palsy patients and to improve their quality of life.

## Background

During the recovery process after Bell's palsy, the tone of facial muscles usually returns first. It is related to the less complex structure of *tonic nerve fibers*. The axons of tonic neurons are thinner and less myelinated. Therefore, their regeneration and function recovery takes less time. As the tonic muscles regenerate, the following changes gradually occur: nasolabial fold becomes more pronounced than during the acute phase, the sagging of the mouth corner reduces, the hanging of the eyebrow reduces, the mouth line becomes straighter, the "numb" sensation on the affected side of the face reduces.

The recovery of voluntary facial movements occurs slower than the return of tone, because the axons of phasic (dynamic) motor neurons are thicker and more myelinated than their tonic counterparts, which requires more prolonged regeneration time.

Usually the trunk of the facial nerve becomes damaged along the narrow Fallopian channel inside the pyramid of temporal bone. This channel is long, narrow and curved. It can reach up to 33mm in length – the longest bone channel

of the human body. Along this channel the facial nerve is vulnerable to mechanical compression from inflammation and swelling of surrounding tissue resulting from infections, disruption of blood supply and from mechanical impact of traumatic damages.

There are two possible scenarios for the recovery of mimetic movements after Bell's palsy.

*First scenario (neuropraxia).* If the impact of the damaging factor on the nerve trunk remains for a relatively short time (3-4 days or less), then only Schwann cells become damaged. Schwann cells form the myelin sheath of the nerve fiber and make possible the propagation of the action potential (conduction of the nerve signal). When Schwann cells in the point of lesion die, this results in a conduction block. However, the integrity of the axons remains intact. This means that there is still a physical connection between the facial nerve nucleus and facial muscles. Within this scenario, the new Schwann cells proliferate on the scene and form new myelin sheath around the axons, thus reinstating the conductivity of the nerve fibers. As a result, facial movements return within 2-5 weeks, without any residuals and complications.

*Second scenario (axonotmesis).* If the damaging factor maintains its impact on the nerve trunk longer than 3-4 days, then beside the damage to the Schwann cells, also the axons of the motor neuron become damaged and degenerate. As a result, there is no more physical connection between the facial nerve nucleus and facial muscles. As soon as damaging factor is finally removed or subsides, the regeneration process begins within the endoneurial tubes, which work as "guiding rails". Each regenerating axon re-grows within his tube until it reaches "his own" muscle fibers and re-creates the previously lost motor unit.

The regenerating axon grows about 1mm per day. If we assume that the nerve lesion happens within the pyramid of the temporal bone, then the approximate distance from there to the facial muscles will be 90-100 mm. This corresponds to 90-100 days before re-connection of the muscles to the nerve happens, and gradual return of the facial movements begins.

Our description of the regeneration process describes the picture that is at some points different from the concept of aberrant regeneration (mis-wiring) of the facial nerve. The concept of aberrant regeneration is nowadays the most widely accepted version of the cause of facial synkinesis. From our point of view, after Bell's palsy, when there was no physical interruption of the continuity of endoneurium and perineurium inside the nerve trunk, there is no physical possibility for the regenerating axons to penetrate its own endo- and perineurium and then to penetrate from outside into the other nerve branches' peri- and endoneurium tubes in order to re-innervate fibers of the "wrong" facial muscles.

The regeneration speed for different axons of facial nerve may vary. That is the reason why the recovery of functions of facial muscles does not happen all at the same time but follows the re-innervation of muscle fibers and re-creation of respective motor units.

By the second scenario (axonotmesis) we can observe residuals and forming complications of various grades of severity. Among the residuals of Bell's palsy, we can name weakness of facial muscles, lack of expression on affected side, incomplete closing of the eye-fissure, remaining Bell's

phenomenon, less pronounced lines and wrinkles on the affected side, unclear articulation during speech, difficulty to hold food and liquids inside the mouth. If during the acute phase the patient was experiencing hyperacusis and taste distortions, then usually those symptoms disappear within first 4-6 weeks of recovery. However, in some rare cases hyperacusis can remain much longer – up to 6 months or more. In our experience we only had two of cases when the patients complained about the distorted taste sensation more than 6 months after the onset of Bell's palsy.

Complications after long-standing Bell's palsy manifest themselves as contractures of facial muscles, pathological synkinesis, excessive tearing during speech and eating (crocodile tears). Some patients complain about pains – either spontaneous or from touching certain points of the face, neck and head on the affected side. Not infrequent are complaints of headaches that are localized mainly on the affected side, as well as of the rigidity and painfulness of the muscles on the back of the head and neck.

Contractures of facial muscles can manifest themselves in narrowing of the eye fissure at rest (hyper-tone of the circular muscle of the eye), more pronounced naso-labial fold (hyper-tone of *m.zygomaticus minor* and *m.levator labii superioris*), mouth corner pulled sideward hyper-tone of *m.zygomaticus major*), multiple dimples in the chin and in involuntary contraction of *m.platysma* – both at rest and during facial movements.

Manifestations of synkinesis are numerous. Synkinesis appear during speech and facial expressions (both emotional and volitional) and are experienced as involuntary contractions of facial muscles that normally do not participate in a given facial movement. The most common are ocular-oral and oral-ocular synkinesis

*Ocular-oral synkinesis* manifests itself in involuntary contractions of zygomatic muscles when patient blinks or squints his eyes. Sometimes by ocular-oral synkinesis the *m.platysma* et *m.depressor anguli oris* may contract too, which results in a noticeable deformity of the lower part of the face. In some rare cases of ocular-oral synkinesis the patient may experience a clicking sound in the ear when he or she blinks the eyes. Most probably this phenomenon is related to synkinetic contractions of *m.stapedius*.

By *oral-ocular synkinesis* involuntary contractions of palpebral parts (upper, or lower, or both) of the circular muscle of the eye happen during speech, eating, smiling, puckering the lips or blowing the cheeks. This synkinesis manifests in the involuntary narrowing of the eye fissure on the affected side.

## Discussion

Human face is one of the most sophisticated communication tools that have ever existed. It can produce thousands of various facial expressions that reflect the full range of human emotions. It is well-known that about 75% of all interpersonal communications happens on a non-verbal level. If after Bell's palsy the patient's face is not able to adequately reflect emotions and cannot convey non-verbal messages, then such an individual experiences him- or herself as communicatively crippled, emotionally deprived and socially inadequate. He or she loses self-esteem, self-confidence, limits social contacts and often

resorts to self-isolation. In especially complex cases the patient may develop suicidal thoughts and behavior.

Not only our emotions form our facial expressions. The opposite is also true: our facial expressions inevitably influence our emotional state. This is a two-directional highway.

During millions of years, the evolution had created steady relations between our emotions and our facial expressions. Certain mimetic patterns (highly coordinated simultaneous contractions of some and relaxation of other facial muscles) are firmly connected to emotions that we experience. And those connections are universal. Emotions are absolutely equally reflected on the face of all representatives of *homo sapiens* species, irrespective of the race, gender, age or cultural differences.

When our brain forms a particular facial expression, the proprioceptive signals from facial muscles, skin and tissue provide feedback that is being compared to the "standard, intended" values in the brain centers. If the brain notices differences, it corrects the pattern and intensity of contraction and relaxation of respective facial muscles so that the character and intensity of the experienced emotions are fully and exactly reflected by the facial expression.

If after long-standing Bell's palsy the patient is unable to adequately reflect his or her emotions with proper mimetic movements, it results in distortion of internal balance, leads to emotional tension, feeling of one's social and aesthetical inadequacy.

It is well-known that contractions and relaxations of our facial muscles and thus our facial expressions are managed independently from two distinctly different "control centers". One is our limbic system, the "factory of emotions". The patterns of mimetic signals that are produced under the management of this center are perfected by the evolution, are well-balanced and equal for all humans. They are "imprinted" in our brain and their structure cannot be changed.

The other matter is our volitional or "rational" mimetic center, that allows us to contract and relax our facial muscles voluntarily, at will. It helps us to suppress our real emotional mimetic reflections if the situation so dictates. For example, we may "construct" a polite smile on our face during an official meeting, even if at that moment we are totally exhausted, angry, depressed or worried. To the contrary, we may pull up a neutral or even a sad face if the situation does not allow us to show our overjoy from winning, for example a million dollars in the lottery.

Respectively, at any given moment the "screen" of a human face reflects an "overlap" from two projections – patterns from the emotional and from volitional center. The one that has higher intensity, takes the upper hand on our face, and its projection is more clearly visible.

Under normal circumstances the facial movements of a healthy individual reflect natural, spontaneous facial expressions that are produced by our limbic system. Only in exceptional situations we may force our face to reflect volitionally managed facial expressions and thus suppress our natural mimetic reflections.

In most cases, during long recovery after Bell's palsy, the natural functioning of facial muscles becomes distorted by constant volitional amplification of mimetic signals. Content-rich and highly fine-tuned signals originating from the

emotional mimetic center are being constantly overridden by much stronger and more coarse volitional signals, that come from our brain's voluntary attempts to contract facial muscles. The resulting picture on the affected side of the face looks very different from the one on the healthy side.

As the time passes and regenerating axons reach their target muscles, the changes in volitional mimetic patterns firm up and the signals that reach patient's face, carry a very strong voluntary component, which by definition is unable to produce natural facial movements. For example, in most cases during long recovery after Bell's palsy, the natural smile pattern on affected side becomes distorted. This manifests itself in an involuntary contraction of the *m.depressor anguli oris* simultaneously with contraction of zygomatic muscles when the patients wants to smile. The former and the latter are muscles-antagonists. This simultaneous contraction results in a considerable tension in both *mm.zygomatici* and *m.depressor anguli oris*. This counteraction of antagonists leads to immobility of the mouth corner despite all the applied efforts, and results in visible asymmetry of the smile. In our opinion, these changes in the dominant mimetic stereotype of the smile are caused by disruption of reciprocal inhibition of the antagonist of zygomatic muscles – *m.depressor anguli oris*. How exactly these changes in CNS happen is at this moment unclear and needs further research. Under these circumstances strong contraction signals arriving from volitional mimetic center, lead to high levels of excitation of the areas of motor cortex where zygomatic muscles and *m.depressor anguli oris* project. This results in excitation of adjacent areas that are "responsible" for managing other facial muscles, namely *m.orbicularis oculi* and *m.platysma*. Resulting excitation of those areas of motor cortex produces contraction signals and involuntary contractions of these muscles, which produce synkinesis and mass movements.

In contrast to the theory of aberrant regeneration, our point of view on possible causes of certain complications of long-standing Bell's palsy allows the possibility to reduce and reverse synkinesis, contractures and mass movements by application of techniques that will facilitate the non-use of volitional mimetic center and allow the natural, emotional patterns of facial movements to re-surface and to find their way to the patient's face.

## Conclusions

The present paradigm for rehabilitation of the long-standing Bell's palsy leaves both the rehabilitation specialist and the patient with very few options for treatment, and the patient - with very little hope for recovery. After 1-2 years since the onset the patients very often hear the message that nothing can be done any more and that they should learn to live with their residuals and complications.

Our considerations explained in this article suggest that if a new paradigm receives support and finds its confirmation in subsequent research, it can completely change the picture for future of rehabilitation for long-standing Bell's palsy patients. It can bring new techniques, new approaches, and a new, well-substantiated hope for the patients to reverse their synkinesis and contractures, reduce or eliminate facial pains and excessive tearing, improve the

facial symmetry and the most important – dramatically improve their quality of life.

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