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An expert opinion: Facial rehabilitation: combining the science and the art

Key take home messages

1. Facial rehabilitation is a clinical specialism that aims to improve outcomes for people with facial weakness.
2. Optimal treatment requires an individualised package of care from a multidisciplinary team.
3. Clinically meaningful improvements are possible in persons with acute and chronic facial weakness.
4. Clinicians develop skills and knowledge of facial movement dysfunction after insult to the facial neuromotor system resulting in advanced competencies and skill sets.

Facial weakness resulting from damage to the corticobulbar tract, the facial nucleus or the facial nerve and its branches, causes resting and dynamic facial asymmetry. This can impact on eating, drinking, speech sound production and eye health, as well as psychosocial well-being and participation. Facial weakness is commonly associated with conditions such as Bell's palsy, Ramsay-Hunt Syndrome, Guillain-Barré Syndrome and its variant, Miller-Fisher Syndrome. Other causes include traumatic brain injury, skull base trauma, and cortical and subcortical strokes. Damage to the facial nerve may also result from direct injury or tumour resection. The resulting facial weakness can be unilateral or bilateral and can vary from a transient presentation to a more persistent and devastating weakness.

There is emerging evidence for the effectiveness of facial rehabilitation; a process that involves facilitating intended facial movement patterns as well as eliminating unwanted movements to advance recovery of the facial nerve[1,2,3,4,5,6] and the facial motor system.

A number of studies have shown statistically significant and long lasting improvements after facial rehabilitation in persons with Bell's palsy.[2,3,6] A Cochrane review[5] also reported evidence for tailored facial exercises to improve facial function for people with chronic and moderate facial weakness. The reviewers suggested that facial exercise could also reduce secondary sequelae in acute cases.

Facial rehabilitation aims to provide education on the cause of facial weakness, enhance the recovery of facial expression and function and improve social participation and well-being.[1] This is done through optimisation of facial symmetry and alignment as well as increased movement in facial expressions and function.

Frequent individualised goals include:

- Improved eye closure;
- Increased ability to smile/produce a more symmetrical smile;
- Increased self-confidence;
- Improved eating and drinking competence;
- Improved ease and clarity of speech sounds;
- Improved size of eye aperture;
- Ease of applying makeup;
- Return to playing wind instruments;
- Jaw/mouth opening.

In published studies, improvements in facial function are demonstrated using the Sunnybrook Facial Grading System (SFGS). The SFGS is a clinician-graded performance based measure of facial impairment which reflects improvement in both resting and dynamic symmetry and a reduction of mass movements. The SFGS assesses resting posture of the eye, nasolabial fold and corner of mouth; voluntary movement for five standard facial expressions in five regions of the face (forehead wrinkle, eye closure, open mouth smile, snarl and pucker) and synkinesis, associated with voluntary movement. Its psychometric properties have been defined including construct validity and responsiveness for clinically meaningful change and inter-rater and intra-rater reliability.[7] Measurement of synkinesis however has been found to be less reliable.[8]

Therapeutic management for people with facial weakness includes detailed assessment, incorporating observational analysis of both sides of the face, backed up by reliable, valid and sensitive evaluation measures, including patient-graded instruments such as the Facial Disability Index (FDI) which considers the impact of facial weakness on both physical and social/well-being function, the FaCE Scale which is a measure of facial impairment and disability and the EuroQol (EQ-5D-5L) which can identify low mood and/or pain associated with health conditions.

In addition to observational analysis, assessment employs palpation to identify specific areas of stiffness as well as significant areas of weakness. This allows for the

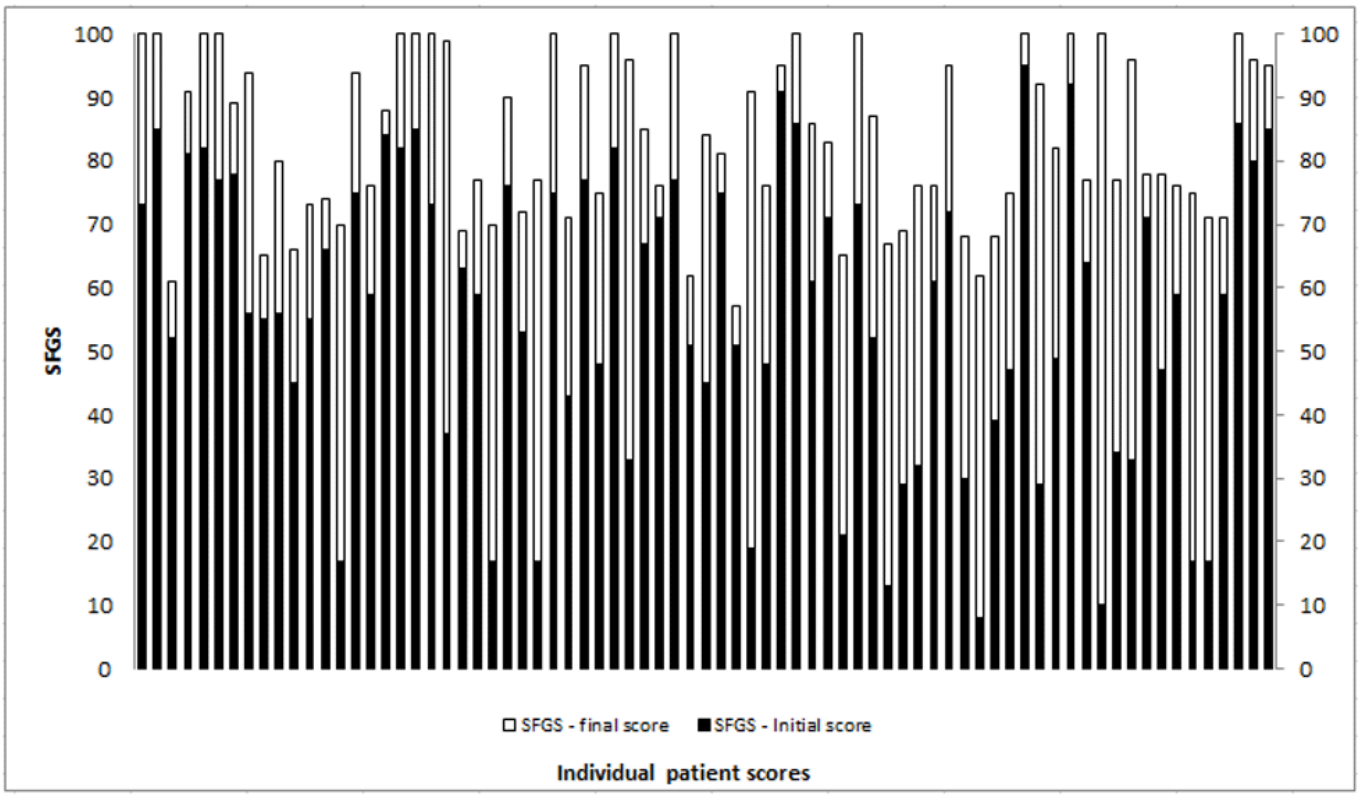

Figure 1. Bar graph representing SFGS score for 73 people with facial weakness. Initial score (dark grey) and final score (light grey) SFGS (Sunnybrook Facial Grading System). Scores range from 0 (worst) to 100 (best). The light grey/white demonstrates amount of change with facial rehabilitation intervention.

generation of an individually tailored neuromuscular facial programme, as indicated by the individual's clinical presentation. This is an important shift away from historically prescribing non-specific exercises, which often promote exaggerated facial movements and secondary complications. Treatment intervention focuses on centring the face by using specific self-stretches to improve muscle length and three to five facial exercises, with emphasis on the muscles being in appropriate alignment, with inhibition of contralateral over-activity and/or synkinesis.

Mirror feedback, due to a lack of facial muscle proprioceptors[9] and perfect practice are key elements in neuromuscular rehabilitation. Taping, either facilitatory or inhibitory, may also be used as an adjunct to an individual's facial programme. Additionally therapeutic mobilisation of the temporomandibular joint (TMJ) may facilitate improved mouth opening in both acute and chronic cases.[10]

When there has been damage to the facial nerve and/or its branches, recovery may be complicated by synkinesis. Synkinesis describes abnormal involuntary movement of one set of facial muscles that accompanies purposeful movement of a different muscle group[11] and may be part of the natural recovery. Common presentations include oculo-oral, involuntary mouth movement on eye closure and oral-oculo, involuntary eye closure on mouth movement, as well as synkinetic activation of the platysma muscle. Clinically, synkinesis is presumed to be due to aberrant axonal regeneration[12] but it has also been hypothesised to result from ineffective myelination leading to cross-talk between terminal facial nerve branches, or a centralised, post injury hypersensitisation of the facial nucleus.[13] The adjunctive use of low dose botulinum toxin type A (BoNT-A) injections in an individual's facial programme can selectively weaken synkinetic muscles and improve resting and dynamic symmetry,[6,11,14,15] especially when over-activity of the contralateral side of the face is also taken into account. BoNT-A is a potent neurotoxin produced by clostridium botulinum which inhibits the release of presynaptic acetylcholine from the neuromuscular junction when injected locally, causing temporary muscle weakness. The BoNT-A molecule is synthesised as a single inactive chain (150 kDa) and then cleaved to form the active di-chain molecule, made up of a heavy chain of ~100 kDa and a light chain of ~50 kDa, held together by a disulphide bridge. The light chain acts as a (zinc-dependent) metalloprotease with proteolytic activity located at the N-terminal end. After the heavy chain is injected, toxin binds to presynaptic receptors on the terminal ends of neurones, and the peptide enters the cytoplasm through endocytosis. Once in the cytoplasm, the light chain cleaves components of the SNARE (soluble N-ethylmaleimide-sensitive factor attachment protein receptor), a complex of proteins necessary for the exocytosis of acetylcholine. In the case of BoNT-A, this specific site is known as SNAP-25 (synaptosome-associated protein of 25 kD). As a result of this cleavage, acetylcholine remains in the neurone, and is unable to bind to receptors on muscle fibres and stimulate muscle contraction (chemodenervation). The effect is short-lasting (three to four months) and weakened muscle recovers over time. The duration of action and turnover of the metalloprotease within the nerve terminal cytoplasm appears to be the predominant, but not the only factor that contributes to the duration of paralysis. Other factors may include transient neural sprouting and re-innervation, although the role of this phenomenon is unclear in humans.[16,17,18,19,20,21]

The use of BoNT-A is well established for the treatment of hemifacial spasm and blepharospasm. There is accumulating evidence from prospective clinical studies for the use of low dose BoNT-A injections in conjunction with facial rehabilitation.[6,11,14] Lower doses of BoNT-A injections are used to treat synkinesis, compared to other facial dyskinesias, to avoid adverse reactions such as excessive weakness, ptosis and diplopia. Lower doses have been reported to be as effective as higher doses.[22]

Contralateral lower quadrant facial sensorimotor impairment is common after a stroke and there is an abundance of evidence for neuromuscular plasticity.[23] Facial rehabilitation in the stroke population aims to exploit this phenomenon to enhance recovery of the facial neuromotor system. Intervention frequently incorporates the emotional motor system to produce spontaneous facial expressions[9] with the face centred (reducing contralateral over-activity) as well selective strengthening in function (talking, eating and drinking). Low dose BoNT-A injections may also have a role in managing contralateral over-activity in conjunction with facial