Graeme E Glass and Kallirroi Tzafetta

Optimising treatment of Bell's Palsy in primary care:

the need for early appropriate referral

Graeme E Glass, BSc, PhD, MRCS, clinical academic lecturer in plastic surgery, Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK. **Kallirroi Tzafetta**, MPhil, FRCS(plast), consultant plastic surgeon, St Andrews Centre for Plastic Surgery, Broomfield Hospital, Broomfield, Chelmsford, UK.

Address for correspondence

Graeme E Glass, Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Kennedy Institute of Rheumatology, Roosevelt Drive, Oxford, OX3 7FY, UK.

E-mail: graeme.glass@ndorms.ox.ac.uk

Submitted: 10 June 2014; **Editor's response:** 26 June 2014; **final acceptance:** 19 August 2014.

 Cite this article as: **Br J Gen Pract 2014; DOI: 10.3399/bjgp14X683041**

INTRODUCTION

Bell's palsy remains the most common cause of lower motor neurone facial nerve paralysis. While 71% experience complete spontaneous resolution, including 61% who demonstrate a complete palsy and 94% who demonstrate a partial palsy, the remaining 29% exhibit lifelong residual hemifacial weakness.[1,2] In 55% of these cases, the deficit is moderate to severe. In addition to stigmatising facial asymmetry, other distressing and socially embarrassing sequelae include: facial spasms and mass-movement contractions (synkinesis); loss of oral competence; facial pain and paraesthesia; nocturnal corneal exposure; dry mouth and eyes caused by loss of parasympathetic innervation to the submandibular/sublingual salivary glands and lacrimal glands respectively; and intolerance to loud noise caused by involvement of the nerve branch to stapedius, resulting in loss of acoustic dampening. Unsurprisingly, anxiety and depression are common.[3] Therapeutic targets for acute Bell's palsy seek to reach these 29%, accepting that prognostic indicators of an incomplete or minimal recovery may be of some value but require further validation.[4] As GPs are likely to see, on average, one acute case every 2 years, it is highly improbable that they will have sufficient expertise to know what to do in the event that acute medical management fails. Worse still, referral to a facial reanimation service is critically time dependent, with the reconstructive options dwindling as the months progress.[5] This article explores this referral pathway, making a case for early referral in every patient failing to demonstrate evidence of resolution by around 6 weeks.

CURRENT BELL'S PALSY MANAGEMENT: GOOD, BAD, AND INDIFFERENT

Bell's palsy is thought to occur as a result of ischaemic compression following inflammation of the nerve in the region of the geniculate ganglion as it traverses the narrow meatal segment of the facial canal. The mainstay of acute management of Bell's palsy is a short course of high-dose (for example, 1 mg/kg) oral steroids. This approach has been validated by a number of systematic reviews[5,6] and constitutes level-1 evidence. The inciting factor (or factors) is unknown. Antiviral therapy has not been demonstrated to be clinically effective.[6,7] Surgical decompression of the meatal segment has been the subject of several studies, but a recent systematic review concluded that evidence to support this approach was lacking, owing to the small numbers reported, methodological flaws, reporting bias, and safety concerns.[8] Electrotherapy, physical therapy, and acupuncture have also been evaluated systematically. The evidence does not support their use.[9,10] While the evidence, summarised in a recent practice guideline by Baugh *et al*,[11] points to a clear way forward, it is evident that this is not yet reflected in clinical practice. A recent UK-wide study of management and referral practices revealed that, in 2012, 44% of all Bell's palsy diagnoses were untreated, while only 6.6% were referred to secondary care, usually otolaryngology, ophthalmology, or neurology.[12] Fewer patients were actually referred in 2012 than in 2001. Clearly, large numbers of patients are not being treated effectively in the acute setting and most patients with incomplete symptom resolution are not referred on to secondary services.

NON-RESPONDERS TO STEROID THERAPY

Corticosteroid therapy is beneficial in three ways. It reduces the number of patients who fail to recover completely, it reduces the severity of residual deficit in those who do fail to recover completely, and it hastens resolution in those who do recover completely.[11] Those who are not given the correct treatment and/or those who do not experience complete resolution face an uncertain future, with no clear idea of where they may find help and support. Referrals for facial reanimation occur after a mean of 2 years via (mainly) oculoplastic surgeons, neurologists, and otolaryngologists. Typically,

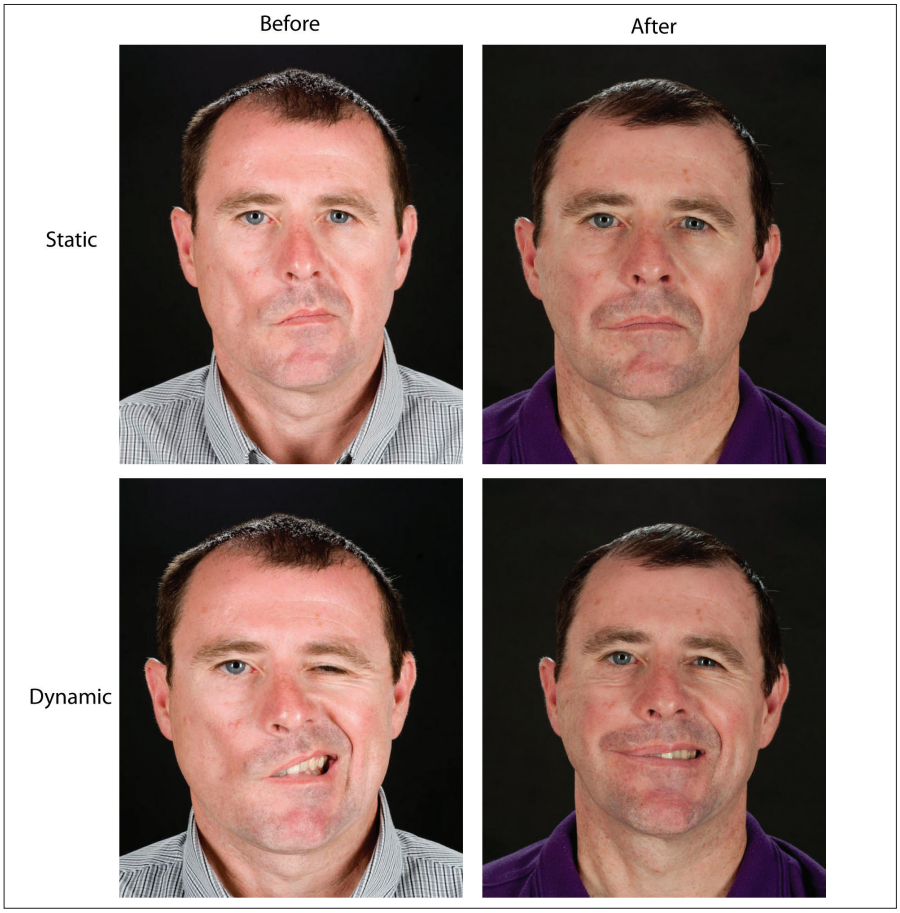

Figure 1. A case of longstanding idiopathic facial palsy treated with a free gracilis flap to the right side. Note: while satisfactory dynamic facial reanimation was achieved, the imported muscle was unable to match the refined animation achievable using autologous muscle targets. These were not available in this case owing to the prolonged duration of the palsy.

patients have given up hope of a solution to their facial disfigurement, especially those with longstanding palsy who had been told there were no further options available. Hence facial reanimation referrals are traditionally considered 'tertiary'. It is our view that a more dynamic referral approach is required.

CONSEQUENCES OF DELAYED REFERRAL

Surgical management of established facial nerve paralysis among healthy, motivated, and compliant patients aspires to achieve symmetry at rest and during both voluntary and emotional expression. In the absence of spontaneous resolution, primary repair of the facial nerve represents the ideal solution, but this is not feasible in an intratemporal nerve lesion of a non-traumatic nature. The alternative solution is to use nerve grafts, joining ('coapting') peripheral motor branches of the contralateral facial nerve to the corresponding branches on the affected side. Through a process known as neurotisation, the affected side may be spontaneously and symmetrically animated on excitation by the contralateral (normal) side. This is known as cross-facial nerve grafting (CFNG) and typically uses the expendable sural nerve as a donor. Importantly, when the denervation time is less than about 6–12 months, CFNG alone is usually sufficient.[5] However, prolonged hemifacial paralysis results in irreversible atrophy of the facial musculature and the loss of these autologous motor targets. When referral is delayed beyond 6–12 months, the ipsilateral hypoglossal nerve can be partially coapted to the facial nerve trunk to preserve the ipsilateral facial musculature pending neurotisation of the cross-facial nerve grafts.[13] In addition to requiring a further procedure, the hypoglossal nerve produces volitional (conscious thought), not spontaneous, movement. When referral is delayed beyond 18–24 months, irreversible atrophy of the facial musculature renders CFNG ineffective owing to the lack of a muscle target. A new muscle target must be imported, requiring yet another operation, in this case a microvascular free tissue transfer.[5] This approach, typically using pectoralis minor, gracilis, or latissimus dorsi, is functionally effective but not as elegant a solution as using the facial musculature. It is also associated with additional surgical morbidity.[5,14] An example of this approach is shown in Figure 1.

THE CHANGING FACE OF REANIMATION SURGERY

The use of CFNG directly to neurotise the affected side represents the ideal surgical option among healthy, motivated patients with residual deficits. That free tissue transfer and, indeed, hypoglossal co-aptation are commonly performed reflects a failure of the referral process. One of the reasons for this is the failure of our specialty to engage with primary care and with patients directly to ensure that the referral process is understood to be critically time dependent. When we consider that the facial nerve is given every chance to demonstrate recovery prior to surgical intervention (usually 3–6 months), then the window of opportunity for achieving the best outcome with the fewest operations is a small one. In most cases it is missed altogether. We must strive to deliver a better service than this. All acute cases of Bell's palsy exhibiting residual symptoms after a few weeks should be considered for referral to a plastic surgical facial reanimation service. The decision when to operate may then be based on the evolving picture. We recommend referral to an otolaryngologist or neurologist when the diagnosis is in doubt (which, admittedly, may often be the case). However, regional facial reanimation services may include otolaryngology and neurology input, and it is worth clarifying this. This should be considered as the default position when managing these infrequent cases in primary care.

Patient consent

The patient has consented to the publication of this article and the photograph.

Provenance

Freely submitted; externally peer reviewed.

Competing interests

The authors have declared no competing interests.

Acknowledgements

Our thanks to our patient for consenting to the use of these images for publication.

Discuss this article

Contribute and read comments about this article: **bjgp.org/letters**

REFERENCES

1. Gilden DH. Clinical practice. Bell's palsy. *N Engl J Med* 2004; **351(13):** 1323–1331.
2. Holland NJ, Weiner GM. Recent developments in Bell's palsy. *BMJ* 2004; **329(7465):** 553–557.
3. Walker DT, Hallam MJ, Ni Mhurchadha S, *et al.* The psychosocial impact of facial palsy: our experience in one hundred and twenty six patients. *Clin Otolaryngol* 2012; **37(6):** 474–477.
4. Fisch U. Prognostic value of electrical tests in acute facial paralysis. *Am J Otol* 1984; **5(6):** 494–498.
5. Terzis JK, Konofaos P. Nerve transfers in facial palsy. *Facial Plast Surg* 2008; **24(2):** 177–193.
6. Sullivan FM, Swan IRC, Donnan PT, *et al.* Early treatment with prednisolone or acyclovir in Bell's palsy. *N Engl J Med* 2007: **357(16):** 1598–1607.
7. de Almeida JR, Al Khabori M, Guyatt GH, *et al.* Combined corticosteroid and antiviral treatment for Bell palsy. *JAMA* 2009; **302(9):** 985–993.
8. McAllister K, Walker D, Donnan PT, Swan I. Surgical interventions for the early management of Bell's palsy. *Cochrane Database Syst Rev* 2011; **2:** CD007468.
9. Cardoso J, Teixeira E, Moreira MD, *et al.* Effects of exercises on Bell's palsy: systematic review of randomized controlled trials. *Otol Neurotol* 2008; **29(4):** 557–560.
10. He L, Zhou M, Zhou D, *et al.* Acupuncture for Bell's palsy. *Cochrane Database Syst Rev* 2007; **4:** CD002914.
11. Baugh RF, Basura GJ, Ishii LE, *et al.* Clinical practice guideline: Bell's palsy. *Otolaryngol Head Neck Surg* 2013; **149(3 Suppl):** S1–S27.
12. Morales DR, Donnan PT, Daly F, *et al.* Impact of clinical trial findings on Bell's palsy management in general practice in the UK 2001–2012: interrupted time series regression analysis. *BMJ Open* 2013; DOI: 10.1136/bmjopen-2013-003121.
13. Terzis JK, Tzafetta K. The 'babysitter' procedure: minihypoglossal to facial nerve transfer and cross-facial nerve grafting. *Plast Reconstr Surg* 2009; **123(3):** 865–876.
14. Fattah A, Borschel GH, Manktelow RT, *et al.* Facial palsy and reconstruction. *Plast Reconstr Surg* 2012;**129(2):** 340e-352e.