CHAPTER 13.

FACIAL NERVE PARALYSIS

Introduction

Facial nerve paralysis, whilst not a disease of the ear itself, commonly arises within the ear due to its anatomical course, and often as a result of ear disease. These paralyses therefore require ontological input. It is important to note that facial paralysis is an otological emergency. Ear disease is a leading cause of these problems. Origins in the ear often require same day management for optimal outcomes; this may require experience, specialised equipment and ear cleaning expertise for diagnosis. Conversely, few non-otological origins are of similar urgency.

Anatomy

The facial nerve emerges from the brain as a motor root and the nervus intermedius, entering the internal ear canal with the nerves of hearing and balance. The roots fuse at the geniculate ganglion. From here it zig-zags through the middle ear to emerge from the stylomastoid foramen, deep to the tip of the mastoid, then passes into the parotid, as an interconnecting plexus, between the superficial and deep divisions of this gland, fanning out to the facial muscles. For the most part the nerve is encased within a bony canal, but this is often lacking superior to the stapes. The nerve may therefore be damaged by problems within the skull, in the ear, or externally.

Functions

a) Motor
b) Secreto-motor, lacrimation
c) Taste
d) Tactile
Functional plan of the facial nerve in the temporal bone. iam: internal auditory meatus; gspn: greater superficial petrosal nerve; gg: geniculate ganglion; st nerve to stapedius; ct: chorda tympani; smf: stylomastoid foramen.

The facial nerve activates the facial muscles during expression, speech and ingestion. It also supplies the “muffler” and reflex actions of the Stapedius and the Tensor Tympani within the middle ear itself.

In addition to this muscular action, the nerve supplies taste, tear and saliva production via the greater superficial petrosal nerve (to the lacrimal gland, nose and palate), and the chorda tympani (anterior two-thirds of the tongue, submandibular and sublingual glands). It supplies tactile sensation to part of the outer ear canal and conchal bowl.

Presentation

Strokes not uncommonly affect the nerve, but the results differ from other damage in that only the mid and lower facial muscles are weakened as a result.

Figure 2: Congenital partial facial palsy, mandibular branch, history of forceps delivery, CT normal.

Figure 3: Facial palsy. Severe left loss. Bell’s palsy, spontaneous resolution over several weeks.

Other cases of paralysis may arise from viral infections within the nerve itself.

Bell’s Palsy is a paralysis of uncertain origins, probably viral (herpes simplex), but without known activation causes. The paralysis is frequently of rapid onset without other major symptoms, although pain may be noted. The patient is free of findings related to the problem, and especially the ear appears normal. Prompt treatment with steroid and antiviral agents may be beneficial, but must be started early after onset. Generally, most Bell’s palsy cases resolve to a substantial degree, or totally. Severe cases, especially in the older age group, may do less well. Some require complex re-innervation or other surgical methods.
Infection of the nerve due to the herpes zoster virus is often severe. “Shingles” in the ear (Ramsay Hunt syndrome, herpes zoster oticus) may cause severe irreversible paralysis, with nerve deafness and vertigo. The outlook for these is poorer.

**Figure 4: Herpes zoster oticus. Healing vesicles, facial palsy and viral labyrinthitis. Older patient, poor recovery of facial function and lingering disequilibrium as a result of the inner ear damage.**

Other forms of facial palsy are more related to its anatomical position in the ear and outer soft tissues.

Whilst in the internal ear canal, the nerve may be troubled by growth of an acoustic schwannoma. The tumour itself rarely paralyses the nerve, but surgery to remove these is a common cause of a damaged nerve, perhaps totally divided, as the nerve is often splayed into fine strands due to the growth of the tumour.

**Figure 5: Acoustic schwannoma, impacting on the brainstem. Removal, whilst sparing a very splayed facial nerve, will be difficult.**

Infection in the middle ear is a major cause of problems. Acute bacterial middle ear infections may invade the nerve causing swelling then paralysis of the nerve fibres in their bony passage as the blood supply is compressed. Prompt treatment with steroids and antibiotics is optimal, plus surgical drainage of the infection to accelerate recovery.

Cholesteatoma requires more aggressive action. These sacs of infected debris gradually erode overlying bone, exposing the nerve to chronic infection. Relief, and prevention of longer term paralysis, will require rapid surgical removal of the cause, preferably within 24 hours.

**Figure 6: Attic cholesteatoma. Prompt surgery is required to salvage a recent facial palsy.**
Paralysis outside the skull frequently has more sinister overtones, arising from tumours of the salivary gland in the cheek, e.g. squamous cell carcinoma, or other sites nearby. The nerve may be cut during removal of these problems. Also, the branch of the nerve to the lower lip muscle is at risk during surgery beneath the line of the jaw.

Figure 7: Facial palsy due to a squamous cell carcinoma of the parotid. Excision required sacrifice of the nerve.

Investigation
Detection of the level of the paralysis may be judged by testing the other functions of the nerve; in turn, middle ear reflexes, taste, or lacrimation, which are sequentially affected depending on the site, as one moves from outside inwards along the nerve.

Schirmer’s test places the site of paralysis at the geniculate ganglion, or medially. This reduces greater superficial petrosal stimulus of ipsilateral lacrimation.

Figure 8: Schirmer’s test. Litmus paper is hooked onto the lower eyelids and lacrimation is stimulated. If a facial nerve paresis is located at the geniculate ganglion or above, lacrimation on the affected side will be significantly depressed.

Acoustic reflex testing may show hypofunction of the stapedial reflex if a nerve block is medial to the stapedius.

Electrogustomery, using an electrode on the tongue, may indicate paresis that originates medial to the chorda.

Tests of motor function are useful to gauge severity and progress:

a) Nerve excitability testing utilises an electrode over the stylomastoid foramen to stimulate facial action, compared to the contralateral (normal) function.

b) Electromyography records muscle function from a needle electrode placed into facial muscles:
   i. Volitional motor potentials: if these are present on stimulation, degeneration has not occurred.
   ii. Denervation potentials/fibrillations appear about ten days after the onset of palsies as a result of axon degeneration. Their absence is a positive sign.
iii. Polyphasic motor unit potentials appear during reinnervation of muscle whose motor supply has degenerated, appearing 2-3 months after the onset of a palsy.

c) Electroneuronography (ENoG) provides the best assessment of neuronal function and is valuable in assessing clinical progress of degeneration after a complete palsy. A bipolar electrode stimulates the facial trunk just anterior to the mastoid and action potentials are read by an electrode over the nasolabial fold. The affected and the normal sides are compared; degeneration is detected by diminishing potentials on successive tests.

Prognosis

Four features help to assess the likelihood of recovery.

a) Age: As a rule, the elderly fare worse than the young.

b) Speed on onset: rapid progression seems a poorer outlook

c) Severity of the palsy: Complete loss of function is ominous.

d) Pain at the onset may indicate a worse prospect.

Management

The treatment of the paralysis for which there is no overt cause (Bell’s Palsy) is controversial. Steroid and antiviral treatment evidently must be prompt for optimal effect. In a minority of pressing circumstances, the surgeon may consider relieving pressure on the nerve by removal of overlying bone (decompression). If a nerve has been completely severed, a sensory nerve from elsewhere may be used to repair the site, but with varying results. Alternatively, redirection of other muscle (motor) nerves may be possible.

Failure to close the eye is a frequent concern in palsy cases, causing drying, irritation and potential corneal ulceration. Partial closure of the eye (tarsorrhaphy) or a weight implant in the upper eyelid may be considered.