Postparotidectomy facial nerve paralysis: peripheral versus proximal identification

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Abstract. Postparotidectomy facial nerve paralysis: peripheral versus proximal identification.

Objectives: Facial nerve palsy is a distressing complication of parotid surgery. We determined to analyse parotid surgery outcomes in a district general hospital in order to identify our own risk factors leading to increased incidence of facial nerve palsy. Primarily, we aimed to determine the efficacy of peripheral versus proximal facial nerve identification in preventing facial nerve damage.

Methods: This was a retrospective study of hospital records. The records of sixty-four patients aged between 32 and 84 years who had attended our Otolaryngology department over a six-year period were analysed, with notes made of operative procedure, identification of the facial nerve and post-operative facial nerve palsy. The Neurosign400 facial nerve monitor was used and the degree of facial nerve paralysis was assessed using the House-Brackmann (HB) classification of facial nerve paralysis.

Results: Sixty-four patients underwent parotidectomy. Thirty-two patients had the peripheral branch of the facial nerve identified while another thirty-two patients had proximal nerve identification. Six patients had HB 2 facial nerve palsy, but all completely recovered within six months. Four out of the six patients had peripheral identification of the facial nerve.

Conclusions: Although peripheral versus proximal identification of the facial nerve was associated with greater incidence of temporary facial nerve paralysis, all the patients recovered within six months. This study may indicate that exposing a peripheral branch of the facial nerve with the help of Neurosign400 is a useful way of performing parotidectomy where proximal identification of a nerve is not possible.

Introduction

Since Thomas Carwardine first reported facial nerve preservation during parotidectomy in 1907 it has become the standard rather than the exception. During early twentieth-century parotidectomies, surgeons identified a peripheral facial nerve branch that was then followed to the main trunk, but it remained for Janes to describe the routine identification of the facial nerve trunk at the beginning of the procedure before proceeding with an anterograde dissection of facial nerve branches.

The risk factors associated with facial nerve paralysis include more extensive surgery, with total parotidectomy associated with a significantly greater incidence. Other significant factors include previous parotid surgery, malignant tumours and lesion size. The method of identifying the facial nerve has not however been extensively studied although in practice some otolaryngologists assume peripheral versus proximal identification to be associated with greater risks, even though no evidence exists to support this. We aimed to determine through our retrospective analysis whether peripheral versus proximal nerve identification was indeed a significant risk factor in its own right.

Methods

This is a retrospective study. The study period includes parotid surgery on benign lesions performed from April 2000 to June 2006. Beside the pre-operative explanation and consent, all patients underwent a pre-operative evaluation of facial nerve function. Parotidectomy was performed using standard surgical techniques and intraoperative facial nerve monitoring. An EMG-based facial nerve monitor, the Neurosign400, was employed in all cases. The Neurosign400 facial nerve monitor produces a differential electromyography recording on four channels. The facial nerve was handled as atraumatically as possible during the surgical procedure, with the number of facial nerve stimulations being limited to the fewest necessary,
and the minimum amount of current possible, as per accepted surgical technique.

The parotidectomy operations were classified into superficial and partial superficial. If the facial nerve was not immediately apparent proximally, efforts were then made to locate the nerve peripherally and a retrograde dissection was performed.

Facial nerve function was evaluated before surgery, at 1 week and approximately 6 months after surgery. Facial nerve function was graded according to House-Brackmann (HB) scale.

**Results**

During the six-year study period, 64 patients underwent a parotidectomy for a benign parotid lesion. The population was composed of 31 males and 33 females, producing a female-male ratio of 0.51. The average age was 58 years, with a range of 32-84 years. Thirty-one patients had right-sided involvement, with 33 suffering left-sided disease.

Sixty patients underwent superficial parotidectomy and four patients underwent partial superficial parotidectomy. The facial nerve was easily identified at the tympanomastoid groove in 32 patients. In a further 32 cases the facial nerve was not immediately apparent and so a retrograde dissection from a peripheral branch was employed (Table 1).

Routine clinical evaluation using the HB scale showed that all 64 patients had normal preoperative facial nerve function. Postoperatively the facial nerve function was normal (HB1) in 58 patients with six patients suffering HB 2 palsy (Table 3). All six patients achieved complete recovery after 6 months. Overall, normal facial function was present in 90.6% of our patients on postoperative day 7 and 100% on prolonged follow-up of 6 months or more.

Retrospective studies by their very nature are not as accurate as some other means of studying this phenomenon, but we believe our results are in keeping with previous work on the subject and are strong enough to stimulate a debate about the issue and to assist the planning of further studies into facial nerve identification techniques.

**Discussion**

In recent publications the incidence of temporary facial nerve deficits has been variously quoted. The incidence was 18% for O’Brien et al., with most others arriving at similar figures. The incidence was somewhat higher at 37% for Bron et al. and 46% for Mehle and colleagues. In these same studies, the long-term deficits range from 0% to 19%. We demonstrated lower overall rates, with 9.4% suffering temporary facial nerve palsy and 0% suffering longer-term deficits.

Two retrospective, non-randomised studies have compared complications with and without EMG monitoring during parotidectomy. Wolf et al. found HB scores greater than 1 in 69% of monitored versus 75% of unmonitored patients. Terrell et al. reported on abnormal facial function in 44% of monitored and 62% of unmonitored patients (p = 0.04). In view of this evidence, we advocate the routine use of EMG-based facial nerve monitoring, but only a prospective, randomised study, after stratification for the risk factors discussed below, could settle once and for all the role of routine facial nerve monitoring in parotid surgery. However, with

<table>
<thead>
<tr>
<th>Table 1</th>
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<tbody>
<tr>
<td>Peripheral versus Proximal identification of facial nerve</td>
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<tr>
<td>Peripheral</td>
</tr>
<tr>
<td>Buccal branch 2</td>
</tr>
<tr>
<td>Mandibular branch 30</td>
</tr>
<tr>
<td>Total 32</td>
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</tbody>
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this evidence, including our own, we are duty bound to optimise our technique as much as possible in order to produce the best clinical outcome.

The factors associated with a high incidence of temporary facial nerve deficit are traditionally thought to include the extent of surgery (superficial vs. total parotidectomy), the histopathology, the size of the lesion and the duration of the operation. Our own study suggests that the method of identification and dissection of the facial nerve may also be important in the consideration of potential complications.

Although the role of these factors deserves further study, the surgeon cannot directly control most of them. More interestingly, the exact physiopathological mechanisms of post-surgical nerve paralysis are still poorly understood. Mechanical trauma is associated with nerve paralysis. Trauma can be divided into compression, crushing, and stretching. Lundborg et al.11 described how peripheral nerves can withstand compressions of approximately 100 mm Hg (13 kpa) before microcirculation becomes impaired. This can result in a metabolic conduction block at higher and sustained pressures with focal demyelination taking place that requires 6 to 12 weeks for complete recovery.12 It seems unlikely that, during a careful parotidectomy, the facial nerve will encounter such high pressures. It has, however, also been found that crushing peripheral nerves with surgical forceps reliably produces a mechanical deformation of the myelin sheaths resulting in a segmental demyelination11 that also requires 6 to 12 months to recover. While the crushing of facial nerve branches could occur, it seems to be a rare phenomenon in careful parotidectomy. The most probable mechanical factor involved could be nerve stretching. Peripheral nerves have been found to follow a peculiar stress-strain relationship, with zones of straightening and elastic elongation, followed by mechanical rupture (see Grewal et al.13). Earlier data showed that rupture occurs at 38% elongation, but more recent studies have demonstrated perineurium tears with disturbances of the intrafascicular homeostasis at elongations of only 6%. The resulting oedema further impedes the microcirculation of the nerve and results in the irrecoverable loss of compound action potential. During such trauma, the nerve remains grossly normal. It is easy to imagine how such nerve stretching could happen during parotidectomy.

Other possible aetiologies of nerve damage include heat damage from electro-coagulators, which is unlikely without massive nerve twitching, and damage from overzealous nerve stimulation, which is also unlikely. Experimental animal data therefore point to nerve elongation as the most probable factor involved in anatomically intact facial nerves associated with post-parotidectomy facial paralysis.

The direct stimulation of nerve should be kept to a minimum in order to avoid fatigue. The

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**Table 2**

<table>
<thead>
<tr>
<th>Histopathology</th>
<th>Number</th>
<th>%</th>
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<tbody>
<tr>
<td>Total</td>
<td>64</td>
<td>100</td>
</tr>
<tr>
<td>ADENOMAS</td>
<td>53</td>
<td>82.8</td>
</tr>
<tr>
<td>Pleomorphic adenoma</td>
<td>37</td>
<td>57.8</td>
</tr>
<tr>
<td>Monomorphic adenoma</td>
<td>1</td>
<td>1.6</td>
</tr>
<tr>
<td>Adenolymphoma (warthin’s tumour)</td>
<td>15</td>
<td>23.4</td>
</tr>
<tr>
<td>Sialadenitis</td>
<td>3</td>
<td>4.7</td>
</tr>
<tr>
<td>Lymphadenitis</td>
<td>2</td>
<td>3.1</td>
</tr>
<tr>
<td>Granuloma</td>
<td>1</td>
<td>1.6</td>
</tr>
<tr>
<td>Cyst 2</td>
<td>3.1</td>
<td></td>
</tr>
<tr>
<td>Lipoma 3</td>
<td>4.7</td>
<td></td>
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**Table 3**

<table>
<thead>
<tr>
<th>Temporary facial nerve palsy</th>
<th>Number</th>
<th>%</th>
</tr>
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<tbody>
<tr>
<td>(A) Peripheral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Sialectasis</td>
<td>1</td>
<td>1.6</td>
</tr>
<tr>
<td>2 Pleomorphic adenoma</td>
<td>2</td>
<td>3.1</td>
</tr>
<tr>
<td>3 Adenolymphoma</td>
<td>1</td>
<td>1.6</td>
</tr>
<tr>
<td>(B) Tympanomastoid groove</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pleomorphic adenoma</td>
<td>2</td>
<td>3.1</td>
</tr>
<tr>
<td>(C) Permanent facial nerve palsy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>0</td>
<td>0.0</td>
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Histopathology of parotidectomy specimens

Peripheral versus proximal facial nerve palsy

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monitor will detect the spontaneous activity caused by the manipulation of the nerve by the surgery, and this is the most useful information for the surgeon.

The stimulation should be set to the 50 µA to 50 mA ranges for all head and neck surgeries. In normal use, the maximum current that should be used to stimulate a nerve directly is 1 mA. We set the stimulation current at 1 mA for identification of proximity to the nerve and 0.25 mA for direct identification of nerves.

We believe that following this protocol increases the chance of finding the facial nerve, which has been demonstrated to be a significant factor in preventing post-operative facial nerve palsy due to the mechanism identified above. Peripheral nerve identification did result in a slightly higher incidence of temporary nerve palsy, but a comparison of these figures with those in which no monitoring was used clearly demonstrates the usefulness of finding the nerve in this manner. It must be remembered that, although malignant cases were removed from our study, pleomorphic adenomas themselves can become malignant and metastasise, underlining the importance of clearance during the initial operation and the balance that must thus be struck between this and facial nerve injury.14

It is also interesting to hypothesise that, in view of the increased incidence of temporary facial nerve paralysis with peripheral identification, residents learning how to perform parotidectomies should perhaps start with anterograde dissections and only move on to retrograde dissection once they are comfortable with the technique. There is a further case to be made that centralisation of expertise may further improve complications such as temporary and permanent facial nerve palsy and this is certainly an issue we hope will generate a lively debate.

Conclusion

In this study, the overall incidence of facial paralysis is 9.4% for temporary deficits and 0% for long-term deficits. The factors associated with an increased incidence of temporary facial paralysis include the extent of parotidectomy, the histopathology and the size of lesion, and the duration of the operation. Our own study suggests that the method of identification and dissection of the facial nerve could also be important in the consideration of potential complications. We found routine use of EMG-based facial monitoring to be helpful during routine parotid surgery. It is particularly helpful in those cases where it was not possible to find the nerve proximally. The mandibular branch of facial nerve was identified peripherally and followed proximally. Although the incidence of temporary facial nerve paralysis was high in the group of patients where the nerve was followed from peripheral to proximal, the paralysis did not last for more than a month. For this reason, it may be pertinent for trainee otolaryngologists to commence surgical training on parotids where anterograde dissection is possible before attempting retrograde procedures. A review of the pathophysiological factors possibly responsible for facial nerve deficits points to nerve stretching as the most probable aetiology.

References

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