Editorial Review

Treatment of sequelae after facial paralysis: a global approach

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It was none other than Charles Darwin who stated more than 100 years ago that ‘the movements of expression in the face and body, whatever their origin may have been, are in themselves of major importance for our welfare’ (Darwin, 1872). This means that every time a patient suffers a facial paralysis his welfare may be at risk because we know that, when degeneration of the nerve develops, 100 per cent recovery is no longer possible. Unilateral associated movements (synkineses) will develop; these are evidence of denervation (Taverner et al., 1971). Slight degeneration resulting in some associated movements and some asymmetry in the face is usually not very disturbing, unless the face is very important in professional life, as it is for models, actors etc. More degeneration will produce a deficiency in the face amounting to a lasting handicap in facial expression and communication for the rest of the patient’s life. In a younger person this will have far greater consequences than in older people. Young people are more easily disturbed in this respect.

Extent of the problem

How often does degeneration and recovery with sequelae occur? Generally, the more severe the loss of function, the worse the sequelae will be. Degeneration depends to a great extent on the cause of the paralysis. In Figure 1 a survey is given of the different aetiologies of facial paralysis we have seen at our department since 1958. The most frequent cause of peripheral facial paralysis is Bell’s palsy, in which the herpes simplex virus type I most probably is a major aetiological agent (Murakami et al., 1996). In 1994 Peitersen reported on the Copenhagen Study he conducted for 25 years (Peitersen, 1994). His material included 1701 covered untreated Bell’s palsy patients. The spontaneous course and recovery were documented. None of the patients remained totally paretic; in 85 per cent function returned within three weeks and in the remaining 15 per cent between three and five months after onset. In the end 70 per cent recovered normal function of the face. 12 per cent had insignificant sequelae and 18 per cent had permanently more or less diminished function with contracture and associated movements. The age of the patients proved to be an important negative factor.

In our study of about 1000 treated and untreated Bell’s palsy patients 64.5 per cent recovered with slight asymmetry at rest and 1.7 per cent with distinct asymmetry, after one year; 29.9 per cent recovered with slight synkineses and 25.4 per cent with severe associated movements. The nerve excitability test in 1176 patients predicted moderate degeneration in 17.35 per cent and severe degeneration in 13.6 per cent. It was also obvious from this study that the age of the patient is an important factor in recovery (Devriese et al., 1990). It can safely be concluded from these studies that about 15 per cent of patients with Bell’s palsy will have unsatisfactory and disturbing sequelae. They will continue to have a handicap in their facial expression and therefore in their communication, for the rest of their lives.

In cases of herpes zoster, complete recovery is reached without treatment in about 10 per cent of the patients after complete loss of function (paralysis) and in about 66 per cent after incomplete loss of function (paresis) (Devriese and Moesker, 1988).
is reasonable to predict a better prognosis when a patient with facial paralysis with herpes zoster of the cephalic extremity is treated with valaciclovir (1000 mg three times a day, for seven days) from the beginning (Beutner et al., 1995).

For other cases of facial paralysis (about 30 per cent) it is difficult to give exact figures for degeneration because of the different aetiologies.

Operative trauma of the facial nerve, either deliberate in order to remove a tumour or accidental as a consequence of surgery, will often result in damage to the nerve. Damage by a fracture of the base of the skull often cannot be prevented by surgery. When the nerve has to be reconstructed by direct anastomosis or transplantation sequelae will always accompany the regeneration, probably because of disturbances in the facial nucleus and misdirection of the outgrowing nerve fibres. About one third of the patients we see in our department every year need treatment in the chronic phase.

In the past few decades there has been a shift in the concept of treatment of these patients. In the 1950s and 60s attention was directed almost exclusively to the medical, somatic problem. Gradually, it became obvious that the whole person suffering from a disfigured face needed more. The psychological and social aspects (communication, expression) received more and more attention. Counselling and rehabilitation of expression were added to the treatment modalities. Plastic surgical procedures were diversified.

The evolution of these ideas is reflected, in my opinion, in titles of the International Symposia on the facial nerve. Dr J. Conley, a pioneer in surgical treatment of facial paralysis, stated at the Symposium in Cologne in 1992: 'Surgery is nothing, our aim is facial expression!', thus strongly emphasising the personal and social problems of these patients.

A global approach

Today we should offer a patient with sequelae after facial paralysis three modalities: plastic surgery, psychological counselling and rehabilitation of expression. These treatments are directed towards the three main problems: the somatic (eye closure, asymmetry, loss of function, drooling, lacrimation, etc.), the psychological (loss of identity, fear, shame) and the social (impaired communication, social isolation, inability to work) (Figure 2).

Management of facial paralysis (chronic phase)

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<thead>
<tr>
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<th>Treatments</th>
</tr>
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<tr>
<td>Medical</td>
<td>Surgical</td>
</tr>
<tr>
<td>Psychological</td>
<td>Guidance</td>
</tr>
<tr>
<td>Social</td>
<td>Rehabilitation</td>
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There is a wide variety of primary and secondary surgical procedures available. Some are sophisticated (nerve reconstruction, nerve transplantation, nerve and muscle transplantation with microvascular anastomosis, anastomosis with other cranial nerves, etc.). The indication for surgery will depend on many factors: the age of the patient, his physical and mental condition and profession, the tolerance to undergo several operations, the condition of the eye, etc. Obviously, the decision to operate has to be taken after careful deliberation. It is impossible to discuss here the surgical possibilities in detail. A review of surgical re-animation techniques has been published by May (1986).

Relatively little attention has been given to psychological counselling of facial paralysis patients. At the Facial Nerve Symposium in Bordeaux (1984), the problem was introduced by Hoos and Devriese (1985). In his monograph on the facial nerve, May (1986) included two sections on this subject. Ekman (1986) concluded that further research in facial expression is needed to evaluate and rehabilitate the paralysed face. According to Twerski and Twerski (1986) these patients fall into three categories in their psychological reaction. Some adjust well and require little psychological intervention, the second category may have mild to moderate difficulties, which can be adequately managed within the primary physician’s practice, finally some patients must be referred to a psychiatric specialist.

Early in the seventies we started to look for a non-surgical approach to improve facial expression. We asked Jan Bronk, a professional mime actor and teacher with experience in the field of the rehabilitation of neurological patients (aphasia, etc.), to look for possibilities for rehabilitating patients with sequelae after facial paralysis. A mime, by virtue of his profession, is a master of his facial musculature and expression. He can even communicate without words! The so-called mime therapy was gradually developed, incorporating principles of mime acting. In this therapy a link is built between the body and the emotional state of the patient, between the movements of the body and emotional expression. Emphasis is laid on the whole person (Deviere and Bronk, 1977).

In retrospect another statement by Charles Darwin is striking: With man the respiratory organs are of especial importance in expression, not only in a direct, but to a still higher degree in an indirect manner. Moreover, Charles Bell (1774–1842) described the facial nerve as ‘the respiratory nerve of the face!’ (Bell, 1821). These two statements emphasize the close connection between respiration and facial expression.

The mime therapy was introduced at the Facial Nerve Symposium in Zürich, in 1976 (Deviere and Bronk, 1977). A meta-analysis of 70 publications on physical therapy methods was presented in Cologne, including mime therapy (Beurskens et al., 1994). Only one randomized clinical trial was found in the literature. They concluded that exercise therapy, massage and biofeedback tend to have a positive
Editorial Review

At the end of 1996 we had experience in the Netherlands with mime therapy with 1758 patients (13,427 treatment sessions) from 1974. At present about 30 therapists (mainly physiotherapists) are active in 20 centres in the country. The effect of mime therapy on the physical, emotional and social level is presently evaluated at the Department of Physiotherapy of the University of Nijmegen (Recurrent Institutional Cycle design).

Conclusion

A 'global approach' for treatment of patients with sequelae after facial paralysis by different disciplines – medicine, psychology and rehabilitation – should be offered in a coordinated fashion. The medical, psychological and rehabilitative aspects of sequelae are interrelated and have to be treated almost simultaneously because the treatment modalities reinforce each other. (In general we wait for the rehabilitation of expression until the definitive situation is reached). In most cases in which we have followed this policy the patients became able to cope with the situation and to resume their daily activities.

I am convinced that these possibilities should be available in all centres where patients with facial paralysis seek help. Close cooperation between the disciplines offering these is necessary.

References

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