

The treatment of facial palsy from the point of view of physical and rehabilitation medicine

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There are evidences to support recommending the early intake of prednisone (in its appropriate dose of 1 mg/kg body weight for up to 70 or 80 mg/day) or the combined use of prednisone and acyclovir (or valacyclovir) within 72 h following the onset of paralysis in order to improve the outcome of Bell's palsy (BP). Although there may be a controversy about the role of physiotherapy in BP or facial palsy, it seemed that local superficial heat therapy, massage, exercises, electrical stimulation and biofeedback training have a place in the treatment of lower motor facial palsy. However, each modality has its indications. Moreover, some rehabilitative surgical methods might be of benefit for some patients with traumatic facial injuries or long standing paralysis without recovery, but early surgery in BP is usually not recommended. However, few may recommend early surgery in BP when there is 90-100% facial nerve degeneration. The efficacy of acupuncture, magnetic pellets and other modalities of physiotherapy needs further investigation. The general principles and the different opinions in treating and rehabilitating facial palsy are discussed and the need for further research in this field is suggested.

Key words: **Rehabilitation - Bell palsy - Physical fitness.**

Bell's palsy (BP) is the most common cause of lower motor facial palsy. There is a relatively high incidence of BP in Alexandria, Egypt.¹ It is also common in many other countries including the Mediter-

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anean region. In BP, spontaneous complete recovery was found in about 69% of the patients.² Therefore, about 31% of BP patients who did not receive the appropriate treatment may suffer from incomplete recovery with residual facial muscle weakness with or without one or more of the commonly encountered complications *e.g.* synkinesis, hyperkinesis and/or contracture. The latter might cause secondary psychological sequels. A great concern has been made to improve the outcome; and to decrease the incidence of complications in BP. This article describes and discusses the different therapeutic options for treating and rehabilitating lower motor facial palsy.

Medical treatment

The available studies on the efficacy of corticosteroids (CS) in BP might reveal some controversy. In 1983, it was reported that, after analysing the available few properly controlled randomized prospective studies on the role of CS in BP (among 92 published articles on CS treatment in BP), it was found that there was no definite proof for the efficacy of CS in treating BP.³ Despite the lack of proven efficacy, CS in its appropriate dose (1 mg of prednisone/kg body weight) has remained the recommended medical treat-

TABLE I.—Recovery in the corticosteroid (CS) group (n=93) versus the control group (n=67) in relation to the start of corticosteroid intake following the onset of Bell's palsy. From Shafshak et al.^{2*}

Facial nerve recovery	CS group (Start of CS intake)				Control group n (%)
	Within 24 h** n (%)	24-48 h n (%)	2-3 days n (%)	3-5 days n (%)	
Excellent (%)	19 (83)	7 (35)	4 (23)	9 (27)	16 (24)
Good (%)	4 (17)	10 (50)	9 (53)	17 (52)	30 (45)
Fair (%)	0	3 (15)	3 (18)	5 (15)	17 (25)
Poor (%)	0	0	1 (6)	2 (6)	4 (6)
Total (%)	23 (100)	20 (100)	17 (100)	33 (100)	67 (100)

n=The number of patients in each group or subgroup.

*) This prospective study was done on patients with acute complete (or nearly complete) unilateral non recurrent facial paralysis (grade 5-6/6 according to the facial nerve grading system of House and Brackmann⁶) who were seen within the first few days following the onset of paralysis. The treated group received prednisolone in a dose of 1 mg/kg body weight/day (in 3 divided doses) for up to 70 mg/day for 6 days, and then the dose was tapered over the next 4 days.²

***) All patients in this subgroup (who started corticosteroid intake within the first 24 h following the onset of paralysis), unlike the other subgroups, had satisfactory results (excellent or good recovery). Also, patients of this subgroup had a significantly better recovery than those of the control group ($\chi^2=7.88$, $P<0.01$).

ment for BP.^{4, 5} Furthermore, it was found that the success of CS in improving the prognosis of BP depended on its early intake (preferably within the first 24 h from the onset of paralysis) in the appropriate dose (Table I).^{2, 6} The latter findings explained some reasons for the pre-existing controversy about the efficacy of CS in BP.²

Moreover, BP patients treated with acyclovir (2 g/day) and prednisone (40-60 mg/day) were found to have a statistically significant faster time of recovery and more complete recovery than patients treated with prednisone alone, especially when administered within the first 72 h following the onset of paralysis.^{7, 8} It was also reported that patients treated with acyclovir and prednisone had one half the incidence of synkinesis and facial contracture compared to patients treated with prednisone alone.⁷ The new antiviral drugs famciclovir and valacyclovir have better gastric absorption and less gastric irritation than acyclovir. Furthermore, prednisone (50 mg/day for 5 days) and valacyclovir (1 g of valacyclovir hydrochloride 3 times per day for 7 days) were found to be effective in managing BP, especially in the elderly.⁹

Although there is still some controversy about the role of CS in the recent literature, it has been con-

cluded that there is an evidence to support recommending prednisone intake alone or in combination with acyclovir in BP.^{10, 11} Therefore, it is recommended to administer prednisone (1 mg/kg/day, for up to 70-80 mg/day, in 2-3 divided doses) together with acyclovir (400 mg 5 times/day) in BP for 7-10 days, unless there is absolute contraindication for their use. Also, it may be recommended to taper the dose of prednisone gradually over 5-6 days, after its intake in full dose for 5-10 days.⁵ A high dose of acyclovir (800 mg/5-6 h) alone or in combination with prednisone (1 mg/kg body weight) has been recommended in Ramsay Hunt syndrome.⁵ It was also reported that the presence of severe postauricular pain might indicate poor prognosis; and might suggest *Herpes zoster* infection.⁵ According to our experience herpetic eruption on the external ear in some patients with Ramsay Hunt syndrome (or *Herpes zoster* infection) may not appear during the first few days following facial paralysis. Then, extensive herpetic eruption appears on the external ear few days after CS intake. Those patients usually suffer from severe facial nerve degeneration. Therefore, I strongly recommend the combined use of prednisone and antiviral drugs as early as possible in BP patients for the fear of having *Herpes zoster* infection, which might not be identifiable by laboratory means within the first few days following the onset of paralysis and prior to the vesicular eruption.

The intake of CS alone, or in combination with antiviral drugs, improves the prognosis of BP (*i.e.* induces rapid and complete recovery in most of the patients) through preventing (or minimizing) axonal degeneration of the facial nerve fibres. CS prevent or lessen nerve oedema and swelling in the facial bony canal; and antiviral drugs suppress viral replication in the neural tissue, thus they may protect the facial nerve from severe damage.

It was reported by one investigator that injection of 500 µg of vitamin B12 (in the form of methylcobalamin) given 3 times weekly for at least 8 weeks was of benefit in enhancing recovery in BP. In a comparative study, he found significantly faster recovery in the groups given B12 injections with or without CS, compared to those given CS alone.¹²

Early surgery in Bell's palsy

The role of early surgery in treating BP has been controversial. Surgical decompression of the facial

nerve may be recommended within 2 weeks following the onset of BP, if electroneurography (ENoG) revealed >90% degeneration of the facial nerve fibres.¹³ While, other authors do not recommend it unless there is 100% facial nerve degeneration.¹⁴ On the other hand, some may not recommend such surgical interference in BP.^{5, 11}

Principles of rehabilitation and physical treatment

Different physical modalities have been used in the treatment of facial palsy, although there may be a lack for a strong evidence for their recommendation. Furthermore, some authors did not recommend electrotherapy for BP or facial palsy.^{4, 5, 15-17} It seems better to discuss the principles of physical and rehabilitation medicine under the following items.

Clinical and electrophysiological assessment

Clinical evaluation for both the severity of paralysis and the presence of complication (*e.g.* synkinesis, hyperkinesis or contracture) is the first step before the start of treatment or rehabilitation. The most popular method for assessing the severity of paralysis is the facial nerve grading (FNG) system according to House and Brackmann.⁶ Clinical assessment should be repeated (approximately every month) to assess improvement.

Some electrodiagnostic tests have been used in selecting facial palsy patients for treatment and in predicting outcome. The commonly used tests include: the minimal nerve excitability test, the maximal stimulation test (MST) and ENoG. The MST and ENoG are the most reliable in predicting prognosis (and assessing the extent of facial nerve degeneration) if done 7-10 days after the onset of paralysis. Transcranial magnetic stimulation is still inferior to the above mentioned techniques. The facial nerve conduction velocity may be done when side to side comparison is not possible as in bilateral facial palsy. The blink reflex is done mainly to exclude lesion at the pons or medulla. Electromyography (EMG) of the facial muscles determines signs of denervation and/or reinnervation as well as the degree of recruitment of motor units. If EMG revealed signs for reinnervation, this may suggest that biofeedback training would be of help in functional restoration.¹⁸ If EMG revealed signs of com-

plete denervation within the first 2 weeks of paralysis, this may suggest the need for early facial nerve decompression according to the opinion of some authors.^{13, 14} In long standing denervation without signs for reinnervation, EMG might help in evaluating the facial muscle status *i.e.* whether there is complete muscle fibrosis or there is still viable contractile muscle fibres. This might help in determining the future line of physical or surgical treatment (*i.e.* muscle graft or nerve anastomosis).¹⁸

Physical treatment

It has been recommended to use local superficial heat therapy (*i.e.* hot pack or infrared rays) for 15 min/session for the facial muscles prior to electrical stimulation (ES), massage or exercises.¹⁷ Massage, which has frequently been prescribed for facial palsy,¹⁷ improves circulation and may prevent contracture. Active exercises (in front of a mirror) prevent muscle atrophy and improve muscle function. However, active facial muscle exercises can not be performed in complete palsy (*i.e.* when FNG is 6/6). Heat therapy improves local circulation and lower skin resistance to ES, thus the lowest current intensity could be used. ES of muscles aims at preserving muscle bulk especially in complete paralysis;¹⁹ and it has also a psychological benefit as the patient observes muscle contraction in his face that gives him hope for recovery from facial paralysis.^{5, 19}

From the physical medicine point of view, the type of ES should depend on the pathology of the facial nerve. If there is no electrophysiological signs of muscle denervation (*i.e.* the facial nerve lesion is focal demyelination or neurapraxia), faradic stimulation or ES using 0.1-1 ms duration pulses delivered at a frequency of 1-2 pulses/s or more (*e.g.* transcutaneous electric nerve stimulation, TENS) may be given for 50-200 contractions/session, 3 sessions/week until recovery. This protocol depends on the author's personal experience and is supported by the findings of other authors.^{20, 21}

If there is electrophysiological evidence for complete facial nerve degeneration, faradic stimulation or TENS (which delivers pulses of <1 ms duration) would not induce facial muscle contraction (unless possibly a very high intensity is used, which may be intolerable).^{19, 22} For stimulating muscles which is completely (or nearly completely) denervated, interrupted galvanic stimulation (IGS) of 100 ms rectangular pulses may be given at a rate of 1 pulse/s for 30-100

contraction/session.^{17, 22} During each session, ES should be stopped once muscle fatigue occurs (*i.e.* it is not recommended to increase current intensity once fatigue occurs). When IGS is used, it is usually given 3 sessions/week possibly for no more than 4 months. The lowest current intensity and only a few number of electrical pulses/session are recommended to minimize or prevent any possible complication. The IGS should not be used once contracture or synkinesis appears. Also, it may no longer be given once voluntary facial movement is regained even partially. Once active facial movement is regained (even partially), active exercises for the facial muscles should be practiced to enhance functional recovery. It should be noted that active exercises are not possible in complete palsy (*i.e.* when FNG is 6/6), hence the importance of ES.

Although many authors do not recommend ES for the fear of enhancing contracture, interfering with reinnervation or increasing cost of treatment,^{4, 5, 15-17} the findings of other authors may recommend its use. ES was found to enhance axonal regeneration in facial nerve lesion,²³ unlike the previous old observation that ES might interfere with reinnervation.²⁴ Furthermore, it was found that ES of skeletal muscles increased muscle neurotrophin-4 mRNA in a dose-dependent manner; and that the subsequent increased neurotrophin-4 stimulated axonal sprouting and skeletal muscle reinnervation.²⁵ Also, it was suggested that early initiation of ES after denervation injury might maintain normal motor unit characteristics; and might improve functional recovery.¹⁹ Therefore, ES seems to be of benefit, and the decision for using ES (including the type of the used current) in facial palsy may be left for the opinion of the treating physiatrist, especially because it has been practiced for several years.¹⁷

One should notice that some of the performed clinical studies^{20, 21} on the efficacy of ES (*i.e.* TENS) in facial palsy were done in a group of patients with variable degrees of facial muscle denervation. This makes difficulties in interpreting their results. If TENS induced improvement of function in partial denervation, this improvement would be most probably due to the improvement of function of the remaining innervated facial muscles as TENS would not induce contraction of the denervated muscles because it delivers pulses of <1 ms pulse duration.²² Furthermore, some²⁰ used TENS in low current intensity (sub-threshold for any motor response at the facial mus-

cles), rendering the underlying mechanisms of improvement unclear.

Biofeedback training for the facial muscles in front of a mirror was reported to prevent synkinesis after facial palsy.^{15, 26} EMG biofeedback training can also be used to improve (or enhance) functional recovery and facial symmetry in patients with electromyographic evidence of facial muscle reinnervation. Besides, facial retraining (or mime therapy) and EMG biofeedback retraining were successful in treating patients with synkinesis.^{27, 28} It has been reported to be an example for the plasticity of the central nervous system to reorganize even in long standing paralysis.²⁷

In the presence of facial contracture, local superficial heat and massage as well as stretching exercises are the appropriate methods. Also, ultrasound therapy (3MHz, for 5 min/session, 5 sessions/week for 3-6 weeks) may be given for the facial muscles of the lower face or frontalis for treating facial muscle contracture. It should not be given close to the eyes. It is recommended to cover the eyes with cotton and gauze during ultrasound session for further eye protection. These therapeutic modalities were suggested for treating contracture as they are known to loosen fibrous tissue adhesions.^{22, 29}

Short wave diathermy (SWD) has been suggested in the treatment of BP. However, some may not recommend its use in BP because there is acute viral inflammation of the facial nerve in its early stage;⁵ and heating of the inflamed nerve may be contraindicated.²⁹ It is possible that deep heating induced by SWD would increase facial nerve oedema in the facial bony canal. This might predispose to facial nerve degeneration. On the other hand, it was suggested that pulsed SWD (which has no thermal effect) might be of benefit in BP,³⁰ but there is little evidence to support the athermal theory and the efficacy of pulsed SWD.³¹ Therefore, the role of SWD in BP might be controversial. Also, heating of the facial muscles (which are superficial) prior to exercise, massage or ES can be attained by superficial heat therapy, which would have no effect on the inflamed nerve in the bony canal.

Acupuncture³² and magnets³³ have been used in combination with physiotherapy in the management of facial palsy, but their specific efficacy needs further investigation. Ultrasound therapy applied to the region of the mastoid process has been reported to be of some benefit in BP when applied early in the course

of the paralysis, before the onset of Wallerian degeneration.³⁴ However, this technique may have serious hazards on the inflamed nerve^{22, 29} or the parotid gland; and thus might not be recommended. Chiropractic manipulation with high-voltage electrotherapy were also tried in sporadic cases,^{35, 36} but the relation between BP and chiropractic manipulation of the cervical spine is unclear; and the real efficacy of these techniques in BP might be questionable.

Treatment with hyperbaric oxygen, through the inhalation of 100% oxygen under high pressure (at pressure 2.8 times greater than the normal atmospheric pressure), may possibly be considered as one of the physical modalities. It was reported that this modality has induced better recovery than prednisone treatment in BP patients.³⁷ Although, it seems that further research is needed before recommending it for general use in BP, one may try it in BP patients with contraindication to CS.

Eye protection

1) Early treatment: artificial tears, ophthalmic ointment especially before sleep and eyeglasses are usually used to protect from light, dust and wind.^{5, 38}

2) Long-term treatment: ophthalmologic consultation is necessary for possible surgical interference, if there is failure of spontaneous eye closure. The available options include tarsorrhaphy, lateral canthoplasty and gold weight implant to the upper lid.^{39, 40}

Facial muscle protection

Facial muscle protection from injury may be achieved by the use of porous adhesive tape (adherent to the skin and extending from the angle of the mouth to the tragus) to prevent deviation of the mouth to the healthy side during smiling.

Management of facial hyperkinesis

When surgery is not indicated, local injection with botulinum toxin A seems to be the most appropriate therapy.⁴¹

Rehabilitative surgical procedures

1) In traumatic facial injuries: microsurgical reanastomosis or nerve grafting including cross-facial nerve grafting may be recommended as early as possible.^{38, 42}

2) In BP: it is better to wait for 12 months for spontaneous return of facial function before any surgical intervention. Nerve substitution is possible e.g. hypoglossal-to-facial nerve crossover.⁴³

3) In long standing facial paralysis with muscle fibrosis: regional muscle transfer (temporalis or masseter muscles) or microneurovascular muscle transfer (from gracilis or latissimus dorsi) may be done to restore symmetrical facial movement.^{44, 45}

Discussion

It is evident from this review that there may be some controversy about the efficacy of the different physical modalities in facial palsy or BP. Besides, it was mentioned that several physical therapeutic modalities, including massage and facial exercises, were recommended to patients with facial palsy, but there are few controlled clinical trials to support their effectiveness in facial palsy.⁴⁶ The role of acupuncture, SWD or early ultrasound application at the mastoid process may be questionable, therefore these modalities need careful reassessment. On the other hand, chiropractic manipulation seemed to be unrelated to facial palsy.

Also, one should keep in mind the fact that spontaneous recovery can occur in many patients without any treatment; and that many factors can affect the prognosis of facial palsy. All these factors should be considered before designing any randomised controlled study for the efficacy of any physical modality.

Any study on the efficacy of physiotherapy in BP should take in account both the efficacy of any concomitant medical treatment (e.g. CS, antiviral drugs, vitamin B12...) and the presence of any factor that may affect the prognosis of BP (e.g. hypertension, diabetes mellitus, age, previous facial nerve lesion, the severity of paralysis, and possibly the type of the causative virus...). Also, we hope that any future animal or human experiment on the efficacy of ES should focus on studying the appropriate parameters of the used electrical current (*i.e.* intensity, frequency, pulse duration...) so that one can select the best parameters for facial palsy patients.

The intake of CS alone or in combination with antiviral drugs, including the dose and the start of their intake following the onset of paralysis, should be

known in all subjects prior to enrolling them in any study on the efficacy of physiotherapy in BP. It is not enough to say that all patients were on CS treatment; and some of them received the appropriate dose while others did not receive it in its appropriate dose. Also, some patients may take medical treatment early, while others may start taking medication after 24 h of the onset. All these factors should be eliminated before the start of any study.

I suggest developing a physiatrist facial nerve group (sponsored by the International Society of Physical and Rehabilitation Medicine, ISPRM) that includes many physiatrists from different countries for investigating and discussing the real efficacy of the different physical modalities in BP. Thus, we may overcome the contradictory views; and we may agree on a universal scientific protocol for managing facial palsy.

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