

Bell's Palsy

ABSTRACTS

Anon., 1998. Methylcobalamin.

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Jalaludin MA., 1995. Methylcobalamin treatment of Bell's palsy.

Roob G., 1991. Peripheral facial palsy: etiology, diagnosis and treatment.

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Kuwabara S., 1999. Intravenous methylcobalamin treatment for uremic and diabetic neuropathy in chronic hemodialysis patients.

Matsumoto Y., 1981. Administration of methylcobalamin after surgical repair of the facial nerve

Ohtani F., 2000. Rapid strip assay for detection of anti-herpes simplex virus antibodies: application to prediction of varicella-zoster virus reactivation in patients with acute peripheral facial palsy.

Takahashi H., 2001. Mouse model of Bell's palsy induced by reactivation of herpes simplex virus type 1.

Fernandez E., 1997. Motoneuronal changes after cranial nerve injury and regeneration.

Fernandez E., 1995. Effects of levo-acetylcarnitine on second motoneuron survival after axotomy.

Ishikawa M., 1999. F-waves of the facial muscles in healthy control subjects and in patients with peripheral facial nerve disturbance.

Accomando J., 1999. An unusual manifestation of diabetes mellitus.

Syed NA., 1999. Blink reflex recovery in facial weakness: an electrophysiologic study of adaptive changes.

De Diego JL., 1999. Seasonal patterns of idiopathic facial paralysis: a 16-year study.

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Pisonero P., 1991. Treatment for facial neuronitis: a new approach to Bell's palsy.

Kinishi M., 1989. [Conservative treatment of Bell's palsy--high dose steroid infusion with low-molecular dextran]

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Hurtado Garcia JF., 1997. Early corticoid treatment of idiopathic facial palsy (Bell)

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Methylcobalamin.

Anon. No authors listed

Altern Med Rev Dec 1998, 3 (6) p461-3

Methylcobalamin is one of the two coenzyme forms of vitamin B12. Evidence indicates this form of vitamin B12, in addition to having a theoretical advantage over cyanocobalamin, actually has some metabolic and therapeutic applications not shared by the other forms of vitamin B12. This monograph provides an overview of the pharmacokinetics of methylcobalamin, and will highlight the potential therapeutic relevance for Bell's palsy, cancer, diabetic neuropathy, eye function, heart rate variability, HIV, homocysteinemia, male impotence, and sleep disorders.

Bell's palsy: an update on idiopathic facial paralysis.

Billue JS Department of Community Nursing, School of Nursing, Medical College of Georgia, Augusta, USA.

Nurse Pract (United States) Aug 1997, 22 (8) p88, 97-100, 102-5; quiz 106-7

Patients with Bell's palsy, or idiopathic facial paralysis, present sporadically in the primary care setting. New evidence implicates

reactivated herpes simplex virus (HSV) as the etiologic agent in greater than 70% of cases diagnosed as Bell's palsy. Careful evaluation of the patient with facial paralysis, including history, physical examination, and diagnostic assessment, may mandate the expeditious treatment of facial paralysis to prevent faulty nerve regeneration during the recovery period. Using the results of an objective tool for grading resting facial symmetry, symmetry of voluntary movement, and synkinesis can provide a quantitative measurement for decision making. These data are also useful in documenting progression or regression of the patient's facial paralysis. Administration of acyclovir with prednisone improves the recovery of complete facial functioning following an episode of Bell's palsy. During the acute and convalescent stages, the eye on the affected side must be protected until function is restored to the facial nerve. Residual effects of Bell's palsy lasting more than 6 months may indicate another diagnosis and the need to refer the patient to a specialist. (13 Refs.)

Methylcobalamin treatment of Bell's palsy.

Jalaludin MA Department of Otorhinolaryngology, Faculty of Medicine, Univeristy of Malaya, Kuala Lumpur, Malaysia.

Methods Find Exp Clin Pharmacol (Spain) Oct 1995, 17 (8) p539-44

Sixty patients with Bell's palsy were included in an open randomized trial. Patients were assigned into three treatment groups: steroid (group 1), methylcobalamin (group 2) and methylcobalamin + steroid (group 3). Comparison between the three groups was based on the number of days needed to attain full recovery, facial nerve scores, and improvement of concomitant symptoms. The time required for complete recovery of facial nerve function was significantly shorter (< 0.001) in the methylcobalamin (mean of 1.95 +/- 0.51 weeks) and methylcobalamin plus steroid groups (mean of 2.05 +/- 1.23 weeks) than in the steroid group (mean of 9.60 +/- 7.79 weeks). The facial nerve score after 1-3 weeks of treatment was significantly more severe (< 0.001) in the steroid group compared to the methylcobalamin and methylcobalamin plus steroid groups. The improvement of concomitant symptoms was better in the methylcobalamin treated groups than the group treated with steroid alone.

Peripheral facial palsy: etiology, diagnosis and treatment.

Roob G; Fazekas F; Hartung HP Department of Neurology, Karl Franzens University, Graz, Austria.

Eur Neurol (Switzerland) Jan 1999, 41 (1) p3-9

Treatment options for peripheral facial palsy (PFP) are an often discussed problem in neurologic practice. Following a short description of the complex anatomy of the seventh cranial nerve we therefore review possible etiologies in the context of leading clinical signs, with idiopathic PFP or Bell's palsy (BP) being most frequent. A rather typical clinical course of BP allows to focus differential diagnostic workup predominantly on the rapid identification of treatable infections such as with Herpes zoster or Borrelia burgdorferi. Neuroimaging studies are needed only in case of trauma, with slowly developing PFP or in the presence of associated signs and symptoms. As BP is characterized by an overall high rate of spontaneous recovery, major emphasis has to be put on avoiding complications by protecting the eye. Meta-analysis of four randomized controlled studies suggests a marginal benefit of steroids concerning eventual achievement of complete recovery. Beneficial effects of a combination of acyclovir and prednisone have also been claimed. While such therapies may be considered in patients with a presumptive bad prognosis, more general recommendations on medical treatment of BP will have to await further trials. (43 Refs.)

Viral infections of the CNS with special emphasis on herpes simplex infections.

Schmutzhard E.

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J Neurol 2001 Jun;248(6):469-477

Within the past decade the management of acute HSV I encephalitis has been improved dramatically by the advent of the polymerase chain reaction (PCR), a method which has become the gold standard of diagnosis of HSV I encephalitis, replacing diagnostic uncertainties and, avoiding, in particular, invasive brain biopsy. Early detection of HSV II in the neonate is mandatory; however, prevention by Caesarean section and/or prenatal therapy of the mother are for this the best option. Very recently the causative agent of Mollaret's meningitis has proved to be, at least in part, HSV I or II. So far prospective randomized therapeutic trials are awaited for the treatment of Mollaret's meningitis using intravenous acyclovir or the more modern oral forms of virostatics (famciclovir, valaciclovir). For decades the causative agent of facial palsy (Bell's palsy) has been sought; only with the advent of PCR has this question been answered. Although one single study indicates the superiority of a combination of acyclovir plus prednisone, this finding has to be confirmed by a large scale prospective randomised double blind study. Nevertheless, if other causes for the clinical/neurological syndrome of peripheral facial palsy have been excluded, a combination therapy with acyclovir plus prednisone seems to be indicated in a patient with Bell's palsy.

SUGGESTED READING

Intravenous methylcobalamin treatment for uremic and diabetic neuropathy in chronic hemodialysis patients.

Kuwabara S, Nakazawa R, Azuma N, Suzuki M, Miyajima K, Fukutake T, Hattori T. Department of Neurology, Chiba University School of Medicine.

Intern Med 1999 Jun;38(6):472-5

OBJECT: To study the effects of the intravenous administration of methylcobalamin, an analogue of vitamin B12, for uremic or uremic-diabetic polyneuropathy in patients who are receiving maintenance hemodialysis. An ultra-high dose of vitamin B12 has been reported to promote peripheral nerve regeneration in experimental neuropathy.

METHODS: Nine patients received a 500 microg methylcobalamin injection 3 times a week for 6 months. The effects were evaluated using neuropathic pain grading and a nerve conduction study.

RESULTS: Serum concentrations of vitamin B12 were ultra-high during treatment due to the lack of urinary excretion. After 6 months of treatment, the patients' pain or paresthesia had lessened, and the ulnar motor and median sensory nerve conduction velocities showed significant improvement. There were no side effects.

CONCLUSION: Intravenous methylcobalamin treatment is a safe and potentially beneficial therapy for neuropathy in chronic hemodialysis patients.

Administration of mecobalamin after surgical repair of the facial nerve

Matsumoto Y.; Yanagihara N.; Okamura H. Ehime Univ., Matsuyama Japan

Practica Otologica (Japan) 1981, 74/10 (2301-2307)

Mecobalamin, 1500 mug/day, was administered to the 11 patients on whom surgical repair of the facial nerve had been done. On nine patients, eight with Bell's palsy and one with Ramsay Hunt's syndrome, an intratemporal decompression operation was carried out one month or more after the onset. Their palsies were complete and the nerves were proved to be seriously degenerated. On two patients, extratemporal facial nerve grafting was performed immediately after removal of the parotid tumor. Administration of mecobalamin continued for more than three months, an average of 27 weeks, after the operation. Comparing with our previous result of operation, we have noted favourable effects on the restoration of the facial nerve function. Although further clinical and experimental verification is needed, this preliminary study has suggested that the drug potentially promotes regeneration of the facial nerve.

Rapid strip assay for detection of anti-herpes simplex virus antibodies: application to prediction of varicella-zoster virus reactivation in patients with acute peripheral facial palsy.

Ohtani F, Furuta Y, Horal P, Bergstrom T. Department of Otolaryngology, Hokkaido University School of Medicine, Sapporo, Japan.

J Med Virol 2000 Sep;62(1):37-41

Varicella-zoster virus (VZV) reactivation causes acute peripheral facial palsy in the majority (88%) of patients who lack anti-herpes simplex virus (HSV) antibodies, suggesting that an absence of anti-HSV antibodies is a reliable serological marker for the diagnosis of VZV reactivation in patients who are diagnosed initially as idiopathic peripheral facial palsy (Bell's palsy) [Furuta et al., 2000] Clinical Infectious Diseases]. A simple and rapid immunoassay for detection of anti-HSV antibodies based on HSV type 1 glycoprotein D was developed by modifying the conventional Western blot technique. The assay was evaluated by comparing the results with those of conventional Western blot. In total, 100 sera obtained from patients with acute peripheral facial palsy were tested and judged blindly by two investigators. Twenty-four of 26 HSV-seronegative sera were obtained from patients with VZV reactivation (Ramsay Hunt syndrome or zoster sine herpette). The sensitivity of the assay was over 95% and the specificity was 100%. The two investigators agreed on the diagnosis in 99 of the 100 sera. These results indicate that the rapid strip assay is applicable to prediction of VZV reactivation in patients diagnosed clinically with Bell's palsy before zoster lesions appear or PCR using saliva samples indicates VZV reactivation. Copyright 2000 Wiley-Liss, Inc.

Mouse model of Bell's palsy induced by reactivation of herpes simplex virus type 1.

Takahashi H, Hitsumoto Y, Honda N, Hato N, Mizobuchi M, Murakami S, Kisaki H, Wakisaka H, Gyo K. Department of

J Neuropathol Exp Neurol 2001 Jun;60(6):621-627

In order to investigate the mechanism of Bell's palsy, we developed an animal model of facial nerve paralysis induced by the reactivation of herpes simplex virus type 1 (HSV-1). Eight weeks after recovery from facial nerve paralysis caused by inoculation with HSV-1, the mice were treated with auricular skin scratch at the site of the previous inoculation, or with intraperitoneal injection of anti-CD3 monoclonal antibody (mAb), or combination of both procedures. No mice developed facial nerve paralysis when they were treated with either auricular scratch or mAb injection alone. In contrast, 20% of mice developed facial nerve paralysis with the combined treatment. With one exception, no mouse treated with either auricular scratch or mAb injection showed HSV-1 DNA in their facial nerve tissue, whereas 4 out of 6 mice receiving both treatments showed HSV-1 DNA on day 10 after treatment. Histopathological findings showed neuronal degeneration in the geniculate ganglion and demyelination of the facial motor nerve in paralyzed mice. These findings suggest that a combination of stimuli, local skin irritation, and general immunosuppression is essential for successfully inducing facial nerve paralysis in mice with latent HSV-1 infection.

Motonuclear changes after cranial nerve injury and regeneration.

Fernandez E, Pallini R, Lauretti L, La Marca F, Scogna A, Rossi GF. Center for Research in Regeneration of the Nervous System, Catholic University Medical School, Rome, Italy.

Arch Ital Biol 1997 Sep;135(4):343-351

Little is known about the mechanisms at play in nerve regeneration after nerve injury. Personal studies are reported regarding motonuclear changes after regeneration of injured cranial nerves, in particular of the facial and oculomotor nerves, as well as the influence that the natural molecule acetyl-L-carnitine (ALC) has on post-axotomy cranial nerve motoneuron degeneration after facial and vagus nerve lesions. Adult and newborn animal models were used. Massive motoneuron response after nerve section and reconstruction was observed in the motonuclei of all nerves studied. ALC showed to have significant neuroprotective effects on the degeneration of axotomized motoneurons. Complex quantitative, morphological and somatotopic nuclear changes occurred that sustain new hypotheses regarding the capacities of motoneurons to regenerate and the possibilities of new neuron proliferation. The particularities of such observations are described and discussed.

Effects of levo-acetylcarnitine on second motoneuron survival after axotomy.

Fernandez E, Pallini R, Tamburrini G, Lauretti L, Tancredi A, La Marca F. Department of Neurosurgery, Catholic University Medical School, Rome, Italy.

Neurol Res 1995 Oct;17(5):373-376

Little is known about factors that regulate the survival of cranial motoneurons which project to peripheral targets. Various neurotrophic factors of central and peripheral origin have been isolated. In this study, we examined thirteen newborn Wistar rats to determine the effects of acetyl-L-carnitine treatment on the survival of motoneurons within the facial nucleus after transection of the facial nerve. Acetyl-L-carnitine was administered for 7 days in seven rats after nerve transection, while saline solution was injected in 6 rats used as controls. Both the motoneuron number and the motoneuron diameter were significantly higher in the facial nucleus of the rats treated with acetyl-L-carnitine than in the facial nucleus of the control rats. The results obtained suggest that acetyl-L-carnitine can rescue a substantial number of facial motoneurons from axotomy-induced cell death. Compared to neurotrophic factors, because of its simple molecular structure, acetyl-L-carnitine permits a safe oral and parenteral administration. It is suggested that acetyl-L-carnitine could be considered for use as a therapeutic agent in neurodegenerative disorders.

F-waves of the facial muscles in healthy control subjects and in patients with peripheral facial nerve disturbance.

Ishikawa M; Namiki J; Takase M; Kojima A; Kawase T Department of Neurosurgery, Saitama National Hospital, Japan.

Electromyogr Clin Neurophysiol (Belgium) Apr-May 1999, 39 (3) p167-74

F-waves were recorded from the mentalis muscles with surface electrodes following stimulation of the marginal mandibular branch of the facial nerve in healthy control subjects during wakefulness, non-REM (rapid eye movement) sleep and voluntary contraction and in patients with Bell's palsy and acoustic neurinoma. The F-wave of the facial muscles results from the backfiring of antidromically activated alpha motoneurons in the facial motonucleus. Therefore, first, the F-waves were not easily elicited in patients with any disturbance in the proximal segment of the facial nerve (Bell's palsy and acoustic neurinoma). Second, the F-waves were affected by excitability of the facial motonucleus; the F-waves were inhibited significantly during sleep and enhanced significantly during voluntary contraction compared with those at rest during wakefulness. When the stimulation strength was set submaximum for M-

waves, F-waves were elicited but H-waves, which have lower threshold than M-waves, were not elicited in the facial muscles, unlike the case of the extremities. Measurement of the F-waves of facial muscles is a new method for estimating excitability of the facial motonucleus unless there is any disturbance of the proximal segment. Fundamental characteristics of the facial F-waves were shown in the present study and measuring facial F-waves is clinically applicable for investigation of both excitability of the facial motonucleus and facial peripheral nerve disturbance.

An unusual manifestation of diabetes mellitus.

Accomando J; D'Agostino A; Adelman HM Department of Medicine, University of South Florida College of Medicine, Tampa, USA.

Hosp Pract (Off Ed) May 15 1999, 34 (5) p39-40

MEDICAL HISTORY: Type 2 diabetes mellitus for five years; unexplained 35-lb weight loss three years ago; Bell's palsy on right side many years ago.

MEDICATIONS: Glipizide, 10 mg/day.

FAMILY HISTORY: Father died of leukemia at age 65; mother has kidney stones; no diabetes or neuromuscular disease.

SOCIAL HISTORY: Insurance salesman; heterosexual, promiscuous, uses condoms; smokes (25 pack years); does not drink.

PHYSICAL EXAMINATION: Well-nourished, well developed, not in acute distress; had difficulty rising from a sitting position because of right lower extremity weakness. Blood pressure, 154/74; pulse, 88; temperature, 36.6 degrees C; respiratory rate, 16. Head, eyes, ears, nose, and throat: normal. Neck: normal. Heart: S4. Lungs: clear. Abdomen: mildly obese. Extremities: no cyanosis, clubbing, or edema; atrophy and weakness of right thigh and both calves; wide-based gait; able to walk on toes but not heels. Neurologic responses: cranial nerves intact; deep tendon reflexes, 1 + symmetrically; plantar reflexes, flexor bilaterally. Skin: macular rash in sun-exposed areas.

LABORATORY FINDINGS: Hemoglobin, 13.2 gm/dL; mean corpuscular volume, 80 micron³; white blood cell count, 7,200/mm³ (normal differential); platelet count, 137,000/mm³. Serum: electrolytes, normal; blood urea nitrogen, 18 mg/dL; creatinine, 0.8 mg/dL; glucose, 308 mg/dL; total protein, albumin, liver enzymes, and creatine kinase, normal. Urine: 1 + glucose. Venereal disease test: nonreactive; HIV test: negative.

DIFFERENTIAL DIAGNOSIS: Dermatomyositis; heavy-metal poisoning; diabetic amyotrophy.

HOSPITAL COURSE: The patient was given 50 mg/day of oral amitriptyline to alleviate the painful paresthesias and was switched to 20 U/day of subcutaneously injected neutral protamine Hagedorn (NPH) insulin to normalize the blood glucose level. Histologic studies of skin and muscle showed sun damage and neuropathic changes, respectively. There was no evidence of vasculitis. Screening for heavy-metal toxins produced negative results.

Blink reflex recovery in facial weakness: an electrophysiologic study of adaptive changes.

Syed NA; Delgado A; Sandbrink F; Schulman AE; Hallett M; Floeter MK Neurology Section, The Aga Khan University, Karachi, Pakistan.

Neurology (United States) Mar 10 1999, 52 (4) p834-8

OBJECTIVE: To study the electrophysiologic effects of unilateral facial weakness on the excitability of the neuronal circuitry underlying blink reflex, and to localize the site of changes in blink reflex excitability that occur after facial weakness.

BACKGROUND: Eyelid kinematic studies suggest that adaptive modification of the blink reflex occurs after facial weakness. Such adaptations generally optimize eye closure. A report of blepharospasm following Bell's palsy suggests that dysfunctional adaptive changes can also occur.

METHODS: Blink reflex recovery was evaluated with paired stimulation of the supraorbital nerve at different interstimulus intervals. Comparisons were made between normal control subjects and patients with Bell's palsy who either recovered facial strength or who had persistent weakness.

RESULTS: Blink reflex recovery was enhanced in patients with residual weakness but not in patients who recovered facial strength. Facial muscles on weak and unaffected sides showed enhancement. In patients with residual weakness, earlier blink reflex recovery occurred when stimulating the supraorbital nerve on the weak side. Sensory thresholds were symmetric.

CONCLUSION: Enhancement of blink reflex recovery is dependent on ongoing facial weakness. Faster recovery when stimulating the supraorbital nerve on the paretic side suggests that sensitization may be lateralized, and suggests a role for abnormal afferent input in maintaining sensitization. Interneurons in the blink reflex pathway are the best candidates for the locus of this plasticity.

Seasonal patterns of idiopathic facial paralysis: a 16-year study.

De Diego JI; Prim MP; Madero R; Gavilan J Department of Otorhinolaryngology, La Paz Hospital, Autonomous University, Madrid, Spain.

Otolaryngol Head Neck Surg Feb 1999, 120 (2) p269-71

The annual frequency of Bell's palsy in 16 consecutive years was investigated in a 465,000-person health area in Madrid, Spain. The annual incidence of Bell's palsy per 100,000 population was found to be 24.1. The male-to-female ratio was 46:54. Seasonal difference in the incidence was noted in our series with fewer cases during summer. According to these results, the illness in Spain seems to have a similar incidence to that in Western countries. In addition to this, Bell's palsy does not have an infectious epidemiologic pattern in our country, but its frequency decreases in warm weather. (29 Refs.)

[Bell's palsy: diagnostic and therapeutical trial in childhood]

Micheli R; Telesca C; Gitti F; Giordano L; Perini A Divisione di Neuropsichiatria Infantile, Azienda USSL 18, Spedali Civili, Brescia.

Minerva Pediatr (Italy) Jun 1996, 48 (6) p245-50

Bell's palsy is caused by a nuclear and/or infranuclear lesion of the facial nerve, producing an ipsilateral deficit of the facial muscles. The etiology is unknown. Bell's palsy has a frequency of 20:100000 individuals/year, a familiarity of 1-2% of cases, and a recurrence rate of 9% of cases. We studied 33 children (mean age 9.1 years) admitted to our Division during the period 1991-1994 because of Bell's palsy. We propose our personal diagnostic and therapeutical trial: in every patient a full neurological and otoscopic examination, and an audiometric test. Treatment is then commenced with prednisone (1 mg/kg/day p.o. for 5-10 days and gradual reduction in 5 days), vitamins B1, B6 and B12 p.o. for 30 days, and local treatment with artificial tears, and occlusive bandage at nighttime until the lagophthalmus is resolved. Electromyography-electroneurography, and brain CT-scan are carried out after 15 days and 21 days respectively of treatment, if neurological deficit is unchanged. 80 to 90% of patients are reported to recover spontaneously from Bell's palsy. With our approach we achieved a 100% recovery rate within an average of 3 weeks (range: 1 week to 5 months).

Treatment for facial neuronitis: a new approach to Bell's palsy.

Pisonero P; Vallejo L; Menendez E; Evangelista CR; Alonso A Servicio de Otorrinolaringologia, Hospital del Rio Hortega, Valladolid, Espana.

An Otorrinolaringol Ibero Am (Spain) 1991, 18 (4) p361-74

The presence of sensitive symptoms, symptoms of parasympathetic ganglion's affection, as well as signs of affection of other cranial nerves accompanying to Bell's palsy, led us to consider and treat this disorder as a symptom of motor lesion of a possible primary neuron inflammation. Although we have not found valid statistical correlations between the lesion and location patterns, we have found, however, that continues electromyographic studies, and the early treatment carried out, may be of great interest to avoid sequels. The treatment was focussed to eliminate both the possible cause and the inflammatory process, as to improve neuron regeneration. From 60 patients selected to enter this study, 91,66 percent recovered the motor function.

[Conservative treatment of Bell's palsy--high dose steroid infusion with low-molecular dextran]

Kinishi M; Hosomi H; Amatsu M

Nippon Jibiinkoka Gakkai Kaiho (Japan) May 1989, 92 (5) p694-702

The etiology of Bell's palsy has not been as yet completely elucidated and the treatment is empirical and controversial. The two most common forms of treatment are steroid therapy and surgery. On the basis of the pathophysiology of Bell's palsy that edema as well as primary or secondary ischemia lead to both compression and hypoxia, Stennert employed high doses of cortisone for a strong antiphlogistic and anti-edematous effect, and dextran in combination with pentoxifylline to increase peripheral nerve perfusion and reported high recovery rate. Since the past 3 years, we have been treating patients with Bell's palsy with a high dose of steroid plus low-molecular dextran (SD therapy). Hydrocortisone was added directly to 500 ml of dextran solution with ATP and vitamins,

starting with 500 mg and finally down with 100mg during 7 days. Before we had adopted this regimen, the patients with Bell's palsy were treated with orally-administrated steroid. A half dose of steroid was administrated in the latter regimen. SD therapy was employed in 120 cases of Bell's palsy, and its results were compared with those of 82 cases with orally-administrated steroid. In a total of 67 cases with incomplete palsy, all cases obtained complete recovery within one month after the onset regardless of the mode of treatment. Each patients with complete palsy was examined with a nerve excitability test (NET) at the first visit and one week later. According to the response of NET, the patients with complete palsy were divided into the following three groups; "good", "poor" and "absent". In "good" group, all cases with SD therapy had complete recovery, while the recovery rate of 31 cases with orally-administrated steroid therapy was 90%. This difference was statistically significant (p less than 0.05).(ABSTRACT TRUNCATED AT 250 WORDS)

Medical treatment of Bell's palsy. Oral vs. intravenous administration.

Tani M; Kinishi M; Takahara T; Hosomi H; Amatsu M Department of Otolaryngology, Kobe University School of Medicine, Japan.

Acta Otolaryngol Suppl (Stockh) (Sweden) 1988, 446 p114-8

Infusion therapy using low-molecular dextran in combination with high-dose cortisone was modified from Stennert's original protocol and indicated in 50 cases of Bell's palsy. The effects of infusion were compared with the outcome in 36 cases treated by orally-administered steroids and vasodilators. In the case of incomplete palsy, the recovery rate was excellent regardless of the mode of treatment. If the palsy is not progressive, it is not necessary for patients with this condition to have infusion therapy. In the case of complete palsy, 95% of those with normal nerve excitability (NE) experienced complete recovery when treated by infusion. However, only 71% of this group experienced complete recovery when treated with oral administration. In the group with diminished or absent NE, complete recovery was obtained in 58% of the patients treated with infusion, whereas only 18% recovered completely when given oral administration. Thus, the recovery rate increased sharply in the case of infusion therapy. Therefore, the above-mentioned method of infusion therapy is indicated in cases of complete or progressively incomplete Bell's palsy except in those cases where its use is contra-indicated for some other reason.

Susceptibility of isolated rat facial nerve to anaerobic stress.

Jund R; Kastenbauer E Department of Otorhinolaryngology, University of Munich, Klinikum Grosshadern,Germany.

Eur Arch Otorhinolaryngol Suppl (Germany) 1997, 1 pS64-7

Ischemic lesions are presumed to be part of many facial nerve pathologies, such as Bell's palsy. The response of facial nerve to hypoxia has not been evaluated previously in an in vitro model. In the present study, the effects of transient anaerobic stress on functional parameters and their recovery were assessed. Extratemporal rat facial nerves were desheathed and incubated in an experimental chamber using solutions containing either low (5 mM) or high (25 mM) D-glucose. In some of the experiments, 40 microM phenytoin or lidocaine was added to observe the effects of membrane stabilizing drugs. Peak height of compound nerve action potential (CNAP), extracellular direct current (DC) potential and latency were measured simultaneously during and after a 40-min period of hypoxia, induced by bubbling the solutions with N₂ or application of 3 mM cyanide. This resulted in a rapid decrease of CNAP and a depolarization of the DC potential with a fast and complete post hypoxic recovery. Elevated glucose concentrations led to a slower decline in CNAP and a smaller rise of membrane potential depolarization. This was accompanied by a slower change of latency. However, post-anaerobic recovery was always diminished in the high glucose solutions. In experiments with phenytoin or lidocaine longer impulse conduction during hypoxia was observed. These findings indicate that the availability of energy-rich components underlies the complex array of physiological derangements seen in ischemia. Membrane-stabilizing drugs show an effect on signal conduction during hypoxia and need further exploration.

Prognosis in Bell's palsy. Influence of early treatment

Ramamurthi B. Inst. Neurol., Madras India

Journal of the Indian Medical Association 1974, 62/8 (281-282)

92 Cases of Bell's palsy, treated over a period of 15 years, have been analysed with reference to the time of starting treatment after the onset of illness and the influence of early treatment with 'medical decompression' of the nerve by corticosteroids and deep X-ray therapy. It is found that 85% of patients, treated within the first 5 days, recovered with the return of almost total facial function. In the group treated between the first 5 days and 6 weeks, only 40% recovered fully. In the group seen after 6 weeks, there was no complete recovery and the maximum recovery of facial function was only partial. These results indicate the value of early treatment of Bell's palsy.

[Idiopathic facial paralysis]

HNO (Germany) Sep 1998, 46 (9) p786-98

Although acute idiopathic facial paresis is often labelled "Bell's palsy", historical studies show that Nicolaus Anton Friedreich (1761-1836) from Würzburg was the first physician to describe the typical symptoms of the disorder in 1797, approximately 24 years prior to the paper published by Sir Charles Bell. Diagnostics has now improved to the extent that acute idiopathic facial palsy can more frequently be assigned to etiologies caused by inflammatory disorders. Herpes simplex virus type I and *Borrelia burgdorferi* are particularly relevant. Underestimation of the degree of paresis is, particularly in children, a drawback of the clinical examination. "Incomplete eyelid closure" is not a reliable indicator of remaining nerve function. For this reason complete electromyography (EMG) is recommended in all cases of severe facial paresis. Since electroneurography does not reliably reflect the degree of denervation present, needle EMG is preferred. The therapy of the facial palsy of unclear etiology is still not well defined. Nevertheless, we recommend that a combined treatment should be used early, at least in patients with disfiguring pareses. Combinations may consist of cortisone, virostatic agents and hemorrheologic substances and possibly antibiotics. Surgical decompression of the facial nerve remains controversial, since positive surgical results lack statistical support. Individual instructions for facial exercises, massage and muscle relaxation can support rehabilitation and possibly reduce the production of pathological synkinesia. Electrical stimulation should not be used. There are a number of possibilities available to reduce the effects of misdirected reinnervation, especially the use of botulinum-A-toxin. However, intensive diagnosis and therapy in the early phase of paresis are decisive in obtaining a favorable outcome. Further refinements in rehabilitation and comparative multicenter controlled studies are still required for future improvements in affected patients. (42 Refs.)

[Diagnosis and treatment of facial palsy]

Noya M; Pardo J Servicio de Neurología, Hospital General de Galicia-Clinico Universitario, Santiago de Compostela.

Neurologia (Spain) Jan 1997, 12 (1) p23-30

The topographic diagnosis of facial nerve lesions is based on the symptoms that accompany paralysis, allowing lesions to be located in the protuberance, pontocerebellar angle, facial channel or trajectory distal to the stylomastoid foramen. Most cases of peripheral facial palsy have no apparent cause (idiopathic, or Bell's, peripheral facial palsy). However, facial palsy can sometimes be a manifestation of neuroborreliosis, multiple sclerosis, diabetes, HIV infection or neurinoma. Neurophysiologic studies complement physical examination to establish a prognosis; after the fifth day axonal degeneration related to incomplete recovery can be recognized. Magnetic resonance identifies nerve lesions but is useful only in atypical cases. Prednisone 1 mg/kg over 5 days, with gradual weaning, is the most widely accepted treatment for Bell's palsy. Acyclovir is indicated in Ramsay-Hunt syndrome. Early surgical decompression in cases with poor prognosis is not generally considered beneficial. Cases of permanent facial palsy have serious consequences, particularly because facial expression is altered.

[6 years experience with reversible and surgical upper eyelid weighting in lagophthalmos]

Müller-Jensen K; Jansen M Augenklinik, Städtisches Klinikum, Karlsruhe.

Ophthalmologie (Germany) Apr 1997, 94 (4) p295-9

BACKGROUND: The procedures for prophylaxis and treatment of keratopathy following facial palsy with lagophthalmos are unsatisfying from the functional point of view.

PATIENTS AND METHODS: Three years ago we created a "lid-dynamic" procedure and applied it to 46 patients with Bell's palsy or before implantation of a gold lid weight. Fixation of lead weights of 0.8-2.0 g to the upper lid by a foil adhesive on both sides (Tesafix), can lead to restoration of lid closure. Within 6 years we implanted 24-carat gold weights into the upper lid in 72 patients.

RESULTS: In all cases lid function was markedly improved; all patients appreciated the procedure. The lead weights were well tolerated. In 27% of the operative cases we observed a slight underdosage, in 10% a slight overdosage.

CONCLUSION: Lid loading is a simple and effective method for functional and cosmetic rehabilitation of patients with lagophthalmos. Despite the dependence upon gravity, the procedure can be recommended for all cases of facial palsy.

[Early corticoid treatment of idiopathic facial palsy (Bell)]

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Idiopathic facial palsy (IFP) (Bell's palsy) is the commonest cause of acute facial paralysis. Perhaps because of its unknown origin, a wide range of treatments are used. Controversy exists as to whether the disorder should be left to run its natural course or treated with steroids. The effect of early steroid treatment on the evolution of IFP was evaluated in the Ear, Nose, and Throat Service of the University Hospital of Alicante (Spain) with a prospective protocol from September 1991 to January 1992. The therapeutic protocol for all patients (47 patients) was an intramuscular injection of 60 mg prednisone in the Emergency Room followed by a course of oral steroids (deflazacort) that was gradually tapered-off. The average duration of IFP before presentation in the Emergency Department was 130.9 days. Clinical improvement was observed on day 149 and a complete cure by day 3026. Full recovery of facial motor function without sequelae occurred in 95.6% of patients. Age, the intensity of paralysis, and a history of hypertension and diabetes had a negative influence on the course of IFP. These results support early steroid treatment for IFP.

The use of low-dose histamine therapy in otolaryngology

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Ear, Nose and Throat Journal 1999, 78/5 (366-370)

Low-dose histamine therapy has been prescribed by otolaryngologists primarily to treat Bell's palsy, vestibular disorders, vascular headache, Miniere's attacks, and urticaria vasculitis. The scientific explanations for the usefulness of this once-empiric treatment are becoming more apparent. Two methods of establishing the appropriate dosages have emerged: the empiric, optimum-dosage approach and the objective, endpoint-titration approach. In this article, the author describes and recommends the latter approach. The author also reports on a 100-patient retrospective clinical study that revealed that the objective, endpoint-titration approach was effective in treating 80% of patients. In light of such success and the ease and economy of this treatment, low-dose histamine therapy appears to be a valuable clinical tool.

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