The hyperexcitability after facial palsy in the asymptomatic side is a frequent situation in the early evolution of these patients related with compensatory mechanism.\(^{[1,2]}\) The cranial reflexology is a valuable instrument to know it, specially the blink reflex. The hyperexcitability produced in the facial motoneuron pools has been documented in several publication, frequently transitory, and only sometime producing movement disorders.\(^{[3,4]}\) The postparalytic blepharospasm is probably the most interesting related situation and several authors have studied them. The increased ipsilateral sensorial inputs provoke a decompensation and subsequently a contralateral trigeminal hyperfunction. These are the more important hypothesis proposed, nevertheless the physiopathology is probably complex and multifactorial (sustantia nigra, cortical influences, peripheral and central synchronization of blink, etc.).

The reinnervation after facial axonotmesis and secondarily the central remodelation, are influential factors, undoubtedly more vigorous in youngs because your regeneration mechanisms are more effectives. This condition have a good correlation between the number of blinking motoneurons recruited and the R2 area-amplitude, and this has been confirmed by the paper.\(^{[5]}\)

The neurophysiological studies and the animal models are useful instruments to know why the hyperexcitability is produced and how to treat them, but other reflex responses and some electromiographical studies in different situations and lesional periods, are possibilities in the future.

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References