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NEUROPHYSIOLOGICAL AND CLINICAL LONG TERM FOLLOW UP IN SACRAL NEUROMODULATION: A HYPOTHESIS OF NEUROPLASTICITY.

Hypothesis / aims of study

Neurophysiological (NPhys) evaluation of patients addressed to Sacral Neuromodulation (SNM) revealed an undisclosed neurogenic alteration in neurocontrol afferent system in idiopathic situations as a possible cause of "dysbalancement" in afferent imput along the pathway from lower urinary tract (LUT) to cortical site.

Furthermore there are evidence that SNM acts with a specific modulating effect on the afferent pathway to the central nervous system depending upon parameters of stimulation (e.g. frequency of stimulation).

A modification in impulse pulse rate of IPG (e.g. from 21Hz to 40Hz) leads to a adjustment (decrease in this case) of cortical latency of Pudendal Somatosensory Evoked Potentials (SEPs) making in turn a sort of "facilitation" of the afferent impulse transmission which suggests a "reset" of the mechanism of processing the afferent pathway.

Therefore the modification of Pudendal SEPs induced by SNM seems to be a prognostic factor of clinical outcomes.

We hypothesized that the alteration in neurocontrol afferent system in pts implanted for LUTs can be modify by chronic modulation of afferent pathway resulting in a sort of neuroplastic effect on LUT neurocontrol.

Aim of study was to evaluate by means of Pudendal SEPs the effect on afferent pathway cortical area after a mean period of 24 months of chronic SNM and to compare with clinical outcome.

Study design, materials and methods

From November 2001 to March 2005, 160 patients underwent NPhys diagnostic evaluation to define possibile neurogenic alteration in LUT neurocontrol.

In 100 pts implanted with SNM, Pudendal SEPs, (cortical latency) were performed studying the influence of modification in pulse rate on afferent pathway.

After a 24 months mean follow up, Pudendal SEPs were again performed in every patients and a comparison with pre-implant data were done.

Results

Here we report 2 opposite situations:

A) in 4 pts implanted for idiopathic detrusor overactivity, for whom the clinical efficacy never was fully achieved and with a slow decline in time, under stimulation at 40 HZ of the IPG, a return of Pudendal SEPs at level prior to the implant.
B) in 4 pts implanted for dysfunctional voiding in whom for some reason the IPG was switched off with a persistent clinical efficacy, the PSEPs had the same latency recorded during stimulation with IPG set on.

Interpretation of results

For group A we hypothesized that for some pts, SNM through a chronic modulation of afferent pathway can modify the plasticity of neurocontrol mechanism, but need to be reinforced, perhaps in correlation with the underlined pathophysiology of the symptom.

For group B we conclude that if a physiological restoration is achieved with SNM, the effect on neurocontrol mechanism persist in a normal fashion.

In our view, neuroplasticity, that is the capacity of the nervous system to modify its organization, to form new connections in order to compensate for injury or changes in one's environment, may be the mechanism by which chronic SNM acts modulating the afferent pathway to the central nervous system and resetting the dysbalancement in LUT neurocontrol.

Concluding message

We normally use NPhys evaluation to select patients candidates to neuromodulation and to define the functional residual potential, that means not only what is left (what works) but what can change, showing the ability to modify under certain circumstances of specific modulation.

NPhys is also important to study the best parameters of stimulation and to monitor the success rate after implant.

In a speculative view NPhys can help to understand the mechanism by which central nervous system modify its organization under neuromodulation therapies.

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