
LATERAL CORD STIMULATION (LCS) TO RELIEVE SPASTICITY. EXPERIMENTAL PROTOCOL AND RESULTS

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Abstract

This experiment demonstrates lateral cord electrical stimulation producing increases in threshold to declenche abnormally propagated electromyographic evoked responses induced by electrical stimulation of 4th lumbar root in pigs with experimental cortical and sub-cortical lesion, its results, its physiopathogenical and therapeutical relations with spasticity. Cerebellar Electrical Stimulation sometimes has limited results as a treatment for spasticity accompanying Cerebral Palsy, because this stimulation can produce a mixture of excitatory and inhibitory impulses on Spinal Cord neural circuits of uncertain effects. The rationale of this research is to produce a more inhibitory and safer action by indirect cerebellar stimulation through Spino-cerebellar tracts on lateral cord surface avoiding collateral action produced by current electrical spreading on cerebellar surface. To demonstrate this hypothesis pigs with surgical unilateral cortical and sub-cortical brain damage were used. A double laminectomy, cervical (C3-C4) and lumbar (L3-L6) was made and thresholds for spasmogenic abnormal electromyographic responses, disseminated on adjacent segments, facilitated by spinal liberation, and produced by extradural electrical stimulation of 4th lumbar root, were measured before and after a lateral cord cervical stimulation. So, our studied variable is the minimal amount of current declenching electromyographic responses in far placed myotomes (L7). Results in ten animals show significant increase of threshold after Lateral Cord Stimulation (LCS). These findings allow extrapolate the transfer of results and propose LCS as a new tool to treat human spasticity.

1. INTRODUCTION

The use of cerebellar electrical stimulation by CES surgical implant of neurostimulators to improve motor performance in spastic cerebral palsy and refractory epilepsy, was described by Cooper in 1972 (7), and is based on classical works of Moruzzi showing decrease of hypertony in decerebrate cats after feline cerebellar cortex

stimulation at high frequency (17,18). Spinocerebellum is the main source of this inhibitory effect produced through deep nuclei and rubro-spinal tracts synapsing interneurons in the spinal cord (2,11,17,18,21, 22), through spinocerebellar tracts entering the cerebellum through superior and inferior cerebellar peduncle (9). Decrease of spinal extensor motoneuron reflexes following paravermal cerebellar stimulation at high frequencies, was demonstrated (5). This system works as a constant feed-back signaling level of descending motor activity (3). The main part of this system starts in vermal surface and parasagittal cortex, so stimulating electrodes are placed in those regions (8,10). Inhibitory action of cerebellar cortex is mediated by Purkinje cells (25) and also voluntary motor activity is shaped by Purkinje cells modulation (12,16,25). A good percentage of success has been reported by CES (8), but it has seldom limited usefulness because of the mixture of inhibitory and excitatory influences in reason of the intricate cerebellar physiology and because of activation of excitatory cortex and sub-cortex (23). Good results and shortcomings have been shown in many centers, (8,14). Our proposal is to try to avoid shortcomings and improve results by stimulating another point of the Spinocerebellar circuits by indirect way, via lateral spinal cord, by means of spino-cerebellar tracts. LCS could enhance the modulatory action of this system.

1.1. Investigation design

Our aim is to determine if mean thresholds necessary to declenche abnormally propagated electromyographic responses, by 4th root stimulation, increases after LCS in relation to identical previous measures without LCS in surgically brain damaged pigs. We have employed a new animal model in pigs (see Andreani et al, poster, this meeting), with cortical and sub-cortical brain damage. This propagated response isn't normally present under those parameters of stimulation (1,4,15), and they appear after an upper motor neuron lesion. Electrical parameters for radicular stimulation were adapted and both clinical (1) and experimental (20,24) methods were taken as models. Pulses of 25Hz, with a duration of 100 microseg. p.p., employing increasing units of 0,5 mA each one, were used. The rationale of such an adaptation was to

measure the threshold to declenche it in progressive measures. Our investigational design is a prospective, longitudinal, with related samples, and comparison experiment

2. MATERIALS AND METHODS

EMG records and radicular stimulation were done with an equipment MEDELEC- model SINERGY, range 2,5 mvolts, sew EEP 50mseg, low frequency filter 50 hz, high frequency filter 1kj, monitor sensibility 200 microvolts, two chnnels, setup adquisition. Electrical parameters for lateral cord stimulation were those used for Dosal Column Stimulation: 100microsec pp, 45 Hz, 8 volts (MEDTRONIC– Neuroestimador ITREL II – Mn Minnessota USA) Ten pigs were studied, weighting 30-40 kg , anesthesia with ketamine clorhidrate was used by intravenous perfusion 50 mg per kilo. Twenty days after brain lesion , a double exposure cervical (C3-C4) and lumbar (L3-L7) was preformed to explore contralateral stroke side. Cervical spinal cord was exposed to place an electrode on its lateral surface. Two needle electrodes were placed in cuadriceps muscle supplied by 4th lumbar root, and in semitendinous muscle innervated by 7th lumbar root for EMG record. left 4th lumbar root was localized by EMG recording on cuadriceps muscle. Once this root individualized an electode was extra durally placed on it . Then, L4 root was stimulated with an increasing electrical amplitude of equal amount, lasting 0,5 sec each, 3 seconds, till a maximal EMG response was obtained in rectus anterioris mucle (cuádriceps). This value was coded X0. A an identical set of electrical activation was again started from the value obtained in X0 with equal parameters, till an interferencial EMG response (also visible) was obtained in semitendinous muscle. This value was coded X1. This proceeding was repeated another 3 times, leaving a delay of 2,5 minutes among measures , and results were coded X2; X3, y X4. Next step was to perform a similar sequence of meas ures , but intermingling two minutes of lateral cord stimulation (LCS) in free-time among them, using an electrode Resumée TL cuadripolar – MR MEDTRONIC, adapted to deliver its charge on the top electrode disk for better discharge on the reduced surface of the lateral cord, and already described electrical parameters were applied. These values were coded X'0, X'1, X'2, X'3 and X'4. Sessions of LCS were delivered between measures X'0 – X'1; X'1 – X'2; and X'2 –X'3 ,but not between X'3 – X'4 to evaluate post-effect.

3. RESULTS

Mean values of X'0 compared to X0 showed no stadistical differences. Measures of current spreading X'1 in relation to X1, X2 toX'2, X3 to

X'3 and X4 to X'4 were sinificant (see table of results in figure1).

FIGURE 1

TABLE OF RESULTS

	Mean	Standart Deviation	P. Value		Mean	Standart Deviation	P. Value
X0	1,2	0,58	0,4062	X1	2	0,53	0,02
X'0	1,1	0,51		X'1	2,6	0,73	
	Mean	Standart Deviation	P. Value		Mean	Standart Deviation	P. Value
X2	2	0,43	0,001	X3	2,2	0,59	0,006
X'2	3,2	0,71		X'3	3,9	0,56	
	Mean	Standart Deviation	P. Value		Mean	Standart Deviation	P. Value
X4	2,15	0,66	0,006				
X'4	4,25	0,88					

TABLES SHOW STATISTICAL COMPARISONS BETWEEN SYMMETRICAL MEASURES (POTENTIALS ARE MEASURED IN mA) SIGNIFICANT DIFFERENCES (P < 0,05) ARE SEEN IN X1-X'1; X2-X'2; X3-X'3; X4-X'4 THERE ARE NOT DIFFERENCES BETWEEN X0-X'0 (WILCOXON TEST WAS EMPLOYED)

4. DISCUSSION AND CONCLUSIONS

Increases of mean electrical thresholds necessary to produce a maximal EMG response in our animal model, is the main fact in the present experiment. A post-effect in the last series of measures (X'4) was also noted confirming the validity of the method. In the whole measures we had observed a lesser amount of variation than observed for other authors (15, 19, 24). Increases of thresholds for propagated discharges to others spinal cord segments, seen after LCS, are, in our opinion, the result of electrical activation of the spinocerebellum by means of volleys of impulses travelling in the lateral cord and via spino – cerebellar tracts, wich are superficially placed. Neither movements nor EMG responses on forelimbs during LCS were observed, then inhibitory action through pyramidal tract stimulation should be considered absent or accessory. Dorsal column stimulation by current spreading could also play an accessory roll on this inhibitory action, but it couldn't be so marked on hindlimbs. LCS acting whatever way produces inhibition of those abnormal reflexes at spinal cord level, that were abnormally liberated by surgical upper motor experimental lesion. When CES was used as a treatment, by radiofrequency linked systems, atrophy of cerebellar cortex and lost of efficacy for chronic use were seldom seen. Electrical current spread during direct cerebellar stimulation and tingling on the occiput was felt by patients when charge was about 25 – 30 micro C/sq.cm./ph (8). There was also a “window effect”, that meant lost of effects below 0,8 and over 5 microC/sq.cm./ph.(8) and potential tisular damage when current intensity rised(6). This current spreading can occur in absence of tingling sensation by Coulomb's doctrine, producing undesirable neocerebellar activation. These shortcomings had been avoided with the use of new implantable stimulators and with correction of electrical charges delivered (8). LCS could be a different way to solve those problems. Direct stimulation of superior cerebellar peduncle by

stereotaxic method (SCP) was tried (13), requiring trained personal, high technology, and has a high cost. Dorsal cord stimulation shows 53% of improvement with an electrical stimulation of 500 HZ, and better results with quadripolar systems are described (8), and better effect on arms and neck are seen. In these experiments increases in threshold necessary to produce abnormal activity, via LCS, has the sense of inhibitory action on abnormal motor system. It is more physiologic than direct stimulation of the cerebellum, the volley of afferent impulses on it, comes of a far-placed zone, so current spreading cannot affect whole cerebellar cortex, increasing modulatory efficiency of the stimulation. Finally. The above exposed reasons let the author to postulate a new therapeutical method, by an implantable system, to produce LCS, to joint other therapeutical resources for Spasticity.

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