MECHANISMS OF MOTOR CONTROL AUGMENTATION USING CONTINUOUS EPIDURAL SPINAL CORD STIMULATION

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ABSTRACT

Spinal cord stimulation resulted in decreased spasticity and ataxia, and moderate improvement of motor control and endurance in a group of 8 multiple sclerosis, 5 spinal cord injury, and 2 degenerative disorder patients. While a number of possible mechanisms may be involved in producing these effects, we evaluated whether they could be described in terms of depolarization of the dorsal columns. This would cause afferent volleys to be sent to the brain with their consequent influence on segmental reflexes through descending pathways. We developed a working hypothesis that the stimulation effects are indirect, primarily mediated via the dorsal columns. The afferent volleys are presumed to augment suprasegmental functions so as to at least partially restore brain influence on segmental reflexes.

INTRODUCTION

In clinical observations of a group of 15 patients with upper motor neurone disorders (8 multiple sclerosis, 5 spinal cord injury, and 2 degenerative disorder patients), we have found that chronic, continuous epidural Spinal Cord Stimulation (SCS) produced the following effects: 1) increased threshold for spinal reflex spasms, 2) decreased excitatory level for repetitive stretch reflexes (clonus), 3) improvement in reciprocal inhibition and coordination of spinal reflex activity, and 4) diminished ataxia. Location of the stimulating electrodes, clinical observations and our neurophysiological analysis suggest that SCS influences the posterior portion of the spinal cord, including the posterior columns of white and gray matter, and spinocerebellar and propriospinal pathways (1-5).

The dorsal column or posterior funiculi consists of two groups of ascending tracts, the fasciculus gracilis and the fasciculus cuneatus. According to Williams and Warwick, these two tracts, which occupy nearly the whole of the posterior white matter, convey proprioceptive sensations, including vibration and pressure and some elements of exteroceptive tactile sensibility (6). On the other hand, according to Wall and Noordenbos (7), the function of the dorsal columns is to carry out tasks of simultaneous analysis of spatial and temporal characteristics of the stimulus. Moreover, there is evidence for the projection of descending fibers from the pre- and post-central cerebral cortex to the corticospinal tracts. These fibers exert facilitory and inhibitory influences on the interneurones of the gracilis and cuneatus nucleii (8). Bilateral section of the dorsal columns in the high cervical region in monkeys led Gillman and Denny-Brown to conclude that

> "the dorsal columns constitute an essential part of the mechanism for projected movement into space, and in particular, for the fine contactual orienting reactions

of fore-limbs. The disturbances in behaviour and motor function after transection result from loss of an important afferent input to parietal lobe, leading not only to defective palpatory exploration, but also to aberrations in the visuospatial behavior of the entire organism" (9).

Patrick Wall, in reviewing the sensory and motor functions of the dorsal columns, suggested that the dorsal columns are essential for stimulus discrimination in which activity is required for the brain's analysis, and that the dorsal columns "initiate and convey the sensory information produced by exploration of the stimulus" (10).

These descriptions were based on positive findings after dorsal column sections. However, it is unclear what effect dorsal column stimulation has on brain structures and motor behavior. Functioning of the dorsal columns may or may not be impaired in patients who receive SCS. In discussing the effects of SCS, we can assume that stimulation induces ascending impulses which travel to the brain-stem, thalamus, cerebellum and cerebral cortex, since the dorsal columns have projections to these structures. These induced volleys travel over pathways and to structures which may be impaired in varying degrees , due to the effects of multiple sclerosis, spinal cord injury or degenerative disorders which necessitated the SCS. Therefore, the degree to which stimulation - induced afferent volleys affects each of these brain structures cannot be generalized.

Application of SCS in different patients will not necessarily produce identical results, due to the complexities of the stimulation pattern and parameters, and to the abnormalities of not only the affferent pathways, but also the motor control pathways. These factors make generalization of the mechanims involved in modification of motor control in these patients difficult if not impossible. Even though we discuss the action of SCS on the basis of current knowledge of motor control mechanisms, we recognize that this is a simplified view of what is in fact an extremely complex clinical epxperiment.

DISCUSSION

Considering the functional and anatomical features of SCS as shown in Figure 1, it can be hypothesized that the effect of SCS is predominantly indirect. In other words, the stimulation activates the posterior columns, thereby functionally integrating them with brain and brain-stem structures. This results in modifyication of the descending motor control pathways and their influence on segmental reflexes as described by Lundberg (11). This hypothesis can account for the large degree of plasticity observed in the effects of SCS on muscle tone regulation, cutaneo-muscular reflexes, and improvement of motor coordination of reflex activity in a large variety of motor disorders.

Two implications of this hypothesis of indirect action via the dorsal columns can be supported in our observations of the patients. First the indirect influence is widely distributed through the central nervous system. This assumption can be

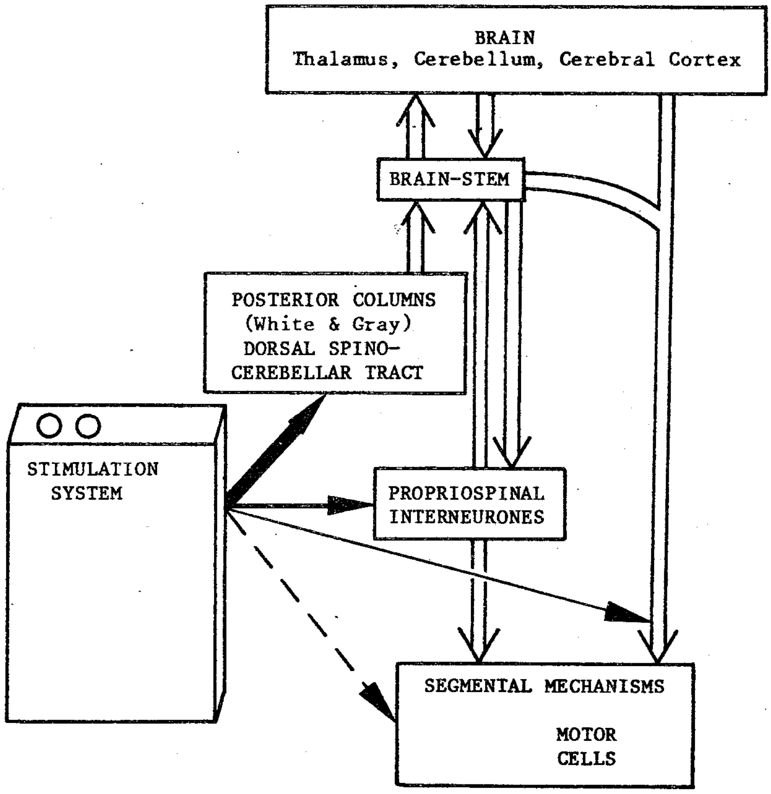

Figure 1. Structural Model of Spinal Cord Stimulation. The relative influence of the stimulation is indicated by the thickness of the line, with the dashed being the least influenced.

justified by observations of the effects in multiple sclerosis patients, who exhibit different kinds and degrees of motor disorders, and different degrees of recovery. Second, the stimulation does not generate any motor responses, but rather modifies ongoing activity. This indirect nature is indicated in the modification of spasticity in spinal cord injury patients. No completely absent functions can be generated, although hidden functions may become apparent following facilitation of the function or suppression of spasticity.

The proposed model assumes that the integrity of the brain and the posterior, lateral and anterior spinal columns is preserved, at least to some degree. Even though the functions of the pathways are impaired due to the demyelination process in M.S., it is evident that, in most instances, there are preserved pathways available to transmit information generated by the stimulation. This is particularly true in the not-too-advanced cases considered to be the best candidates for epidural stimulation. While the damage to tracts in the various pathways may be more severe in the degenerative disorders, some will be preserved, providing paths for the modification of motor control.

In paralyzed spinal cord injury patients, it might be assumed that there are no paths remaining to convey the indirect stimulation effects, and, in turn, to effect any improvement in motor control. Even in spinal cord injury patients with no clinical evidence of functioning spinal pathways, however, neurophysiological evidence of brain influence on segmental reflexes can frequently be found (12). The preserved pathways conveying this influence may also convey the indirect effects of the SCS. The criteria for the application of SCS in spinal cord injury patients which we have used is the evidence of such preserved pathways. Under the assumptions of this model, SCS could not work in the same way in patients without these pathways.

It may be asked what effects could be expected if the posterior tracts are damaged or absent due to the nature of the lesion. When the nature of the lesion is such that posterior column ascending volleys cannot reach brain structures which are relevant in the modification of motor control, then another, simpler system could at least partially play the same role, namely the propriospinal interneurone system (13). This system is much simpler and cannot reach ascending structures further than the medulla oblongata. It diffusely extends through the spinal cord and can mediate impulses within the spinal cord without traversing supraspinal structures.

The postulate that continuous SCS can effectively suppress spasticity in otherwise paralyzed spinal cord injury patients can be questioned since the relationship of the dorsal columns to spasticity has not been clearly defined. When lateral columns are damaged in addition top the dorsal columns, the phenomena of spasticity will appear (9). On the other hand, results of experiments involving lesions of the dorsal columns do not give any indication as to the nature of the effects SCS will have on spasticity. Based on our knowledge of the effects of regular, repetitive stimulation of the spinal cord and the resulting habituation (14), we have hypothesized that imposition of the regular, 30 to 70 Hz, pattern of stimulation on afferent flow in the dorsal columns may augment suprasegmental functions in such a manner as to at least partially restore descending influence on white and gray matter of the posterior portion of the spinal cord. In particular, such influence could induce presynaptic inhibition, thus suppressing the hyperactive segmental motor system.

Evidence in support of this assumption can be seen in the case of one patient with familial spastic paraparesis (CC). In this instance, the extent of the lesion is well known. In this disorder, there is a selective attack on the lateral and dorsal columns. The only missing sensory capability was the ability to integrate both position and time of a stimulus applied to the skin, such as in the recognition of letters or numbers traced on the Motor system impairment was evident in the spastic gait and neurological signs of pyramidal lesion. With the addition of SCS, this patient experienced improved motor coordination and endurance, presumably due to the activation of the posterior column fibers and elicitation of the newly established functional relationship between the spinal cord and brain structures. Further support for this hypothesis is seen in the case of (S.L.), with Fredriech's ataxia. In this case also, damage to the CNS presumably resulted in inadequate afferent volleys via the dorsal column, for which SCS can substitute, with consequent motor improvement.

Finally, the clinical findings, that the effects of SCS take some time to be fully developed and that there is a carry-over effect after stimulation, suggest that the effects are not due to stimulation of just one structure. This is further supported in the observation that the effects are diffuse. Spasticity of both flexor and extensor muscle groups can be reduced, and the functioning of spastic bladders can be improved as well. All these observations point to the development of a new sensory motor integration of the brain and spinal cord structures.

CONCLUSIONS

In conclusion, SCS can be seen to be another example of the utilization of external control of afferent volleys for the restoration of motor control (15). The proposed model is simplified to emphasize the major features of SCS, namely, that its primary effects are mediated through the dorsal columns and are indirectly effective at wide-spread levels of the CNS in modifying motor control. In reality, stimulation of additional ascending tracts, the propriospinal interneurone system, and the gray matter of the spinal cord, may contribute to the overall effects of SCS, but do not change the specific nature of the motor effects. Further clinical and neurophysiological studies will be needed to establish the relative contribution of stimulation of the dorsal column versus stimulation of these other structures. Such knowledge will facilitate the expansion of the application of this approach to other patient categories and degrees of disability.

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