III. Other Topics on Pain

One Possible Mechanism of Central Pain. Autokindling Phenomenon on the Phantom Limb or Sensory Loss Oriented Patients

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With 6 Figures

Summary

The authors compare the electrical activity of the specific and non-specific thalamic nuclei in cases of central pain (phantom pain, thalamus pain and anesthesia dolorosa) and in cases of other disturbances. They found that in central pain from these structures chronic paroxysmal activity can be recorded. This process evolves by the patients own abnormal sensory stimuli and is probably an autokindling phenomenon. The authors' findings satisfy the necessary conditions of central pain evolvement namely, that the pain appears gradually after the sensory lesion, the pain is progressive in nature and that the evolvement needs the patients personality. In the management of central pain chronic stimulation needs reconsidering because of these results.

Keywords: Central pain; thalamus; irritative mechanism; autokindling; chronic stimulation.

Introduction

The underlying mechanism of central pain (phantom pain, anesthesia dolorosa, thalamus pain) still remains enigmatic despite the considerable effort to solve it. Melzack⁶ and later Melzack and Loeser⁷ postulated that a lesion of the sensory system results in

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hyperactivity in cells lying central from the lesion. According to the
gate control theory the pain results from the unbalanced stimuli
between C and A fibers. Others emphasize the absence of central
inhibitory mechanisms. Sano postulates reverberating
circuits between the specific and non-specific thalamic nuclei
originating from the hyperirritability of the sensory nuclei.

The basic mechanism is still a puzzle and considerable
pessimism has attended the therapy of central pain because it is very
difficult to control either by conservative or by surgical means. One
thing is certain; central pain takes time to appear after the lesion in
the sensory system, appears gradually and is not consistent with the
lesion, sometimes it appears, sometimes not, with the same lesion. It
is also probable that the normally evolved body scheme which is in
constant need of reinforcement because of the significantly altered
sensory experience, undergoes certain changes which play a role in
the originating pain. The chronic multielectrode stereoxial method used by us made it possible to observe the spontaneous and evoked activity of
specific and non-specific sensory structures for a long time in
central pain patients and in patients with other disorders. From an
incidental observation it was supposed that in patients suffering
from central pain the activity of the subcortical sensory structures
differs strongly from that observed in other patients.

Materials and Method

The examinations were carried out on 14 patients. Four patients suffered from
phantom pain, 3 from anesthesia dolorosa, 3 from thalamus pain and these results
were compared with 2 patients suffering from parkinsonism, 1 from hyperkinetic
motor disturbance and 1 from obsessive-compulsive symptoms. The activity of the
same central structures were compared in these patients.

The following central structures were examined: the ventral posterolateral and
posteromedial thalamic nuclei, the centrum medianum, pulvinar and the
mesencephalic reticular formation. Chronic gold or platina-irridium electrode
bundles were inserted in these structures by the aid of a stereotactic
instrument. The spontaneous electrical activity was registered, then during
the stimulation of one structure we registered the evoked potentials and the evoked
electrical activity in the other not stimulated structures.

Registration was carried out with a 16 channel ELEMA-Mingograph and with
a MEDICOR MG-42 four channel amplifier. The stimulations were carried out
with a DISA Multistim or with a MEDICOR ST-21 stimulator.

Stimulation parameters were 0.5–1 Hz; 0.05 ms; 10–30 V single square pulses
and 100 Hz; 0.05 ms, 5–30 V, 500 ms trains.