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EVIDENCE TO SUGGEST THAT OPIATE AND OPIOID ANALGESIA MAY BE DIFFERENTIALLY MEDIATED.

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Whilst several lines of evidence suggest that corticosteroids are critically involved in the mediation of opiate and opioid analgesia this has not, as yet, been unequivocally demonstrated.

Therefore, in order to examine this hypothesis in more detail intact morphine (5mg/kg) treated DBA/2 mice were administered with metyrapone, which blocks the synthesis of endogenous corticosteroids or dexamethasone, which in the acute phase may be regarded as having corticosteroid-like effects. Parallel studies involving the exposure of mice to opioid-activating attack parameters within a resident-intruder paradigm were also conducted.

Data revealed that 0.1-1 mg/kg metyrapone and 0.05-80mg/kg dexamethasone partially blocked morphine analgesia. By contrast, whilst 1-10mg/kg metyrapone also partially blocked analgesia resulting from exposure to 35 attack bites, dexamethasone (0-80mg/kg) failed to influence this response. Higher doses could not be used as these were found to have intrinsic analgesic properties.

These data together suggest that inhibition of corticosteroid synthesis attenuates the expression of opiate and opioid analgesia, whilst treatment with an exogenous synthetic corticosteroid only has influence on the expression of opiate analgesia, suggesting opioid and opiate analgesia to be mediated by different mechanisms under these circumstances.

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ACUTE INTRATHECAL BACLOFE
IN SUBJECTS WITH SUPRASA

RICHARD M. HERMAN, M.D., S
McDowell Road, Phoenix,
University of Arizona He

Baclofen, the prototypi
orally, has been used wid
When spasticity is suffic
al performance, oral ba
clinical interest in co
baclofen by an implantab
effect of a bolus inject
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pain, caused by pinch a
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ticity was markedly atte
pronounced suppression o
muscle stretch (without
voluntary motor contract
The effect on reflexes
stimulation by acute i.t
alpha$_2$-adrenergic agonist
opiate agonist) are compa
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1. Penn, D., et al. N
2. Herman, R.M. et al.
3. Herman, R.M. et al.

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FACILITATION OF THE H-REFLEX DURING A REACTION TIME TASK AND SOMATOSENSORY EVOKED POTENTIALS TO THE H-REFLEX STIMULI.

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If an H-reflex of the soleus muscle is elicited by posterial tibial nerve stimulation during a visual reaction time task (VRTT), the H-reflex begins to increase 60 msec before the onset of a conditioned plantar fexion. This facilitation is due to reduced presynaptic inhibition of the Ia afferent endings contacting the motoneurons. Ia afferents project also to the cortex via relais stations at the origin of the dorsal spinocerebellar tract, in the nucleus Z and the thalamus. The following two experiments were aimed to attain somatosensory evoked potentials (SEPs) eclusivly due to Ia afferent volleys and to study SEP gating before movement in a VRTT.

Cervical and cortical SEPs exclusivly to Ia afferent volleys of different sizes were computed by subtracting the SEPs due to exclusive skin stimulation from those of transcutanous nerve stimulation at the same stimulus intensity. Stimulation strength ranged from 80% of H-reflex threshold up to a current sufficient to elicit maximal H-reflexes. The cortical P30 and P50 of the ensuing Ia SEPs increased linearly with stimulation intensity in the range tested, thus Ia volleys subthreshold for H-reflexes evoked typical Ia SEPs. These findings are masked in most subjects before subtraction of the SEP to skin stimulation alone.

SEPs to H-reflex stimulation (H-reflex size 25% of the maximum at rest) applied at different intervals before movement onset were recorded during a VRTT. The contamination by the visual evoked potentials (VEPs) was eliminated by subtracting the VEPs from each test recording. The recordings were then grouped according to their occurance before movement onset , averaged and the SEPs to skin stimulation were subtracted. The first Ia SEPs

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STIMULATION-INDUCED
IN THE RAT SPINAL CO
J. SVOBODA AND E. SY
Regulations CAS, Bul

We studied the dy
volume (ECSV) change
by repetitive electr
nerves or by chemica
injury. Using the co
(1981) we used modif
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from the recording K
calculated the absol
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stimulation. We also
calculated from the
TMA^+, since the conc
proportional to the
$\alpha = 24 \pm 1\%$, $\lambda = 1.5$
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increase in ECSV by