Title : NEURAL EFFECTS OF EPIDURAL ANESTHETICS

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Introduction. A paucity of information exists concerning the relative susceptibility of the various neural structures that have been implicated as sites of action of epidurally injected local anesthetic agents. To clarify this issue, alterations in averaged evoked potentials recorded from electrodes spaced along the conducting pathways of the monkey were evaluated during epidural blockade with a variety of local anesthetic agents. This method offers a means of assessing the functional integrity of peripheral nerve, nerve root and spinal components during the induction and maintenance of epidural analgesia.

Methods. Four monkeys (Macaca arctoides) had implantation through small incisions in the ligamentum flavum of bipolar electrodes consisting of three platinum iridium disks into the midline dorsal epidural space at the cauda equina and upper thoracic regions. At the level of the conus medullaris, three silver ball electrodes were inserted into the dorsolateral epidural space. Catheters, 19 gauge, were inserted into the epidural space with the tips positioned at the T8 and L2 levels, respectively. These small openings were sealed with gelfoam sponge and methacrylate. Additionally, platinum disk electrodes were implanted over sensorimotor cortices. Following a one to two day recovery period, recordings were carried out before and after injection of three different epidural anesthetic agents (Bupivacaine, Chloroprocaine, and Etidocaine). Stimulation was performed with rectangular pulses of 0.2 msec duration applied at 4 Hz with intensities at levels necessary to obtain maximum responses following stimulation of the sciatic nerve, spinal cord and sensorimotor cortex. Evoked potentials were retrieved with the CTC-2000 Evoked Potential Measurement System. Following control recordings, 3.0 cc of selected anesthetic agent was injected. Responses evoked by sciatic nerve (SN) stimulation were recorded from cauda equina (CE), conus medullaris (CM), upper thoracic cord and sensorimotor cortex (SMC). Responses evoked by CM stimulation were recorded from upper thoracic cord and SMC. Responses evoked by SMC stimulation were recorded from dorsolateral CM. The integrity of the epidural space was verified by an epidural metrizamide study.

Results. Bupivacaine (0.5% and 0.75%) caused its most prominent response alterations at the dorsal root region and spinal cord levels with mild changes at the CE region. Responses evoked by SMC stimulation and recorded at CM showed less sustained attenuation. These findings indicate that the major elements of epidural Bupivacaine blockade occurs at the dorsal root, root entry zone and spinal cord levels. The degree and occurrence of these changes were similar for these structures until the drug concentration was reduced (0.25%) at which the predominant alteration was at the dorsal root and entry zone regions. Chloroprocaine (3%), however, resulted in attenuation and distortion of the dorsal root region (SN→CM) responses without significant effects on the spinal cord as manifested by intact cord-to-cord and SMC-to-lower-cord responses. As compared to Bupivacaine, these changes at the dorsal root region were of lesser magnitude and duration. Etidocaine (1%), in contrast to previous agents, caused its major alterations at the spinal cord level with marked and sustained attenuation of the cord-to-cord and SMC-to-lower-cord responses. The dorsal root region (SN→CM) and cauda equina responses (SN→CE) remained relatively well preserved except for a delayed and mild attenuation of the dorsal root region responses (60→100 min).

The correlation of the varying neural susceptibilities and the different clinical properties of these local anesthetics indicate that this model is a valuable investigational tool. Clinically, this information may improve the application and manipulation of various local anesthetics for achieving the most appropriate type of blockade relative to the procedure being performed.