Sensitization in the spinal dorsal horn is believed to be a cellular mechanism for enhanced pain sensitivity. In this study, by using optical imaging with voltage-sensitive dye, we measured the neuronal excitation in spinal cord slices from normal rats and Complete Freund's Adjuvant (CFA)-induced arthritis rats. The amplitude of evoked-neuronal excitation in the spinal dorsal horn in slices of arthritis rats was bigger than normal rats. The evoked-neuronal excitation in the dorsal horn of arthritis rats was suppressed in the presence of a glial metabolism inhibitor, monofluoro-acetic acid, a NMDA-receptor antagonist, D-AP5, and P2X-receptor antagonists, PPADS and TNP-ATP. In contrast, these inhibitor and antagonists did not show any affect on the evoked-neuronal excitation in the dorsal horn of normal rats. These results suggest that facilitation of neuronal excitation mediated by glial cells via NMDA-receptor and P2X-receptor in the spinal dorsal horn is underling mechanism for induction of mechanical hyperalgesia in arthritis rats.