Everybody has experienced pain. Pain by definition is an unpleasant sensory and emotional experience associated with actual or potential tissue damage. In the peripheral tissues acute painful stimuli activate specialized endings of afferent neurons called nociceptors. The information about tissue damage is then transmitted to the cell bodies of these dorsal root ganglion neurons by unmyelinated or thinly myelinated axons (C and A fibers, respectively). The central branches of these neurons form synapses with superficial dorsal horn neurons in the spinal cord. The information is conveyed at the synaptic connections by neurotransmitters such as glutamate and many others neuromodulators. Important is the subsequent activation of projection neurons that transmit the information to supraspinal brain areas. Activity of excitatory and inhibitory interneurons, glial cells and descending pathways from the CNS are also important for the modulation of nociceptive information at the spinal cord level. After peripheral tissue damage and in other pathological states, increased sensitivity to peripheral stimuli may develop. As results of this change innocuous stimuli are perceived as painful (alodynia) and increased pain is perceived after noxious stimuli (hyperalgesia). The underlying mechanisms of these changes may be both peripheral and central. This paper reviews the basic information about processing of nociceptive inputs at the spinal cord level. Understanding and study of these mechanisms is important for the development of new effective analgesics.