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Commentary

A Short Note on Pain Pathway

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DESCRIPTION

Primary afferent nociceptors conduct impulses to the spinal cord (or, if they originate from the head, to the medulla oblongata of the brain stem). In the spinal cord, primary afferent nociceptors terminate near secondary neurons in the dorsal horn of gray matter. Primary afferent nociceptors release chemical messengers at the ends of the spinal cord. These transmitters activate secondary pain transmitter cells. The identity of these transmitters has not been established, but candidates include small polypeptides such as substance P and somatostatin, and amino acids such as glutamic acid and aspartic acid. Some axons of these secondary cells cross the opposite side of the spinal cord and extend long distances to the brainstem and thalamus. The path of pain transmission is in the anterolateral quadrant of the spinal cord. Most of our information on the anatomy and physiology of central nervous system pain transmission pathways comes from animal studies. However, in humans, lesions in this anterolateral pathway are known to permanently impair pain perception and its electrical stimulation causes pain. Elevation of nociceptive axons in the anterolateral quadrant of the spinal cord has two major targets: the thalamus and the medial reticular formation of the brainstem. Our knowledge is most extensive about spinal cord cells, where axons project directly into the thalamus, that is, cells of the spinothalamic tract. The spinothalamic tract is involved in human pain perception, as lesions at all levels cause permanent impairment of pain perception. Studies on the cellular properties of the spinothalamic tract have been conducted in several species. In all these types, the majority of spinothalamic tract neurons respond maximally to harmful stimuli. In addition, in cells of the spinothalamic tract, there is a direct relationship between firing frequency and a range of stimulus intensities that are detrimental to human subjects. These observations, coupled with decades of painstaking clinical studies, strongly suggest that the spinothalamic tract is the leading pathway for human pain. Another major ascending nociceptive pathway in the anterolateral quadrant is the spinal reticular tract. The medulla oblongata reticular formation receives a large direct projection from the spinal cord and branches from several spinal neurons that project onto the thalamus. At the thalamic level, the pain pathway has two major terminators, the ventral and medial sides. The ventral thalamus receives nociceptive inputs directly from the projecting spinal cord neurons. Neurons in the ventral thalamus project directly into the somatosensory cortex. The medial dorsal nucleus of the thalamus receives indirect input from the spinal cord, but in addition it receives large input from the reticular region of the brainstem projected by nociceptive spinal reticular neurons. The medial thalamus projects to a large area of the forebrain, including the somatosensory cortex.

Common pain pathways

Within the pain pathway are third-order neurons that carry action potentials that signal pain.

Primary neurons: These are pseudounipolar neurons with perikaryons within the dorsal root ganglion. They have axons that divide into two branches: the peripheral branch (extending peripherally) and the central branch (extending centrally to the spinal cord/brainstem).

Secondary neurons: The cell bodies of these neurons are found in the Rexed laminae of the spinal cord or in the nucleus of the cranial nerves of the brain stem. These neurons then intersect at the anterior white commissure of the spinal cord and ascend the skull in the spinothalamic tract to the posterolateral ventral (VPL) nucleus of the thalamus.

Tertiary neurons: The cell bodies of tertiary and tertiary neurons are located within the VPL of the thalamus. They project across the posterior lower leg of the internal capsule and end in the ipsilateral postcentral gyrus (primary somatosensory cortex). The postcentral gyrus is somatically organized. Therefore, the hand-initiated pain signal ends up in an area of cortex dedicated to the sensation of the hand.

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