Editorial

Do nociceptors and nociception solely imply noxious stimuli?

Volume 1 Issue 1 - 2014

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Received: April 30, 2014 | **Published:** May 01, 2014

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In the anesthesiology, the term, nociception is often used to describe transmission and perception of sensory information induced by noxious stimulation from nociceptive receptors (nociceptors) at the nerve endings to neurons in the central nervous system (CNS). However, in the anesthesiological field, the term, nociceptor is also used to describe nociceptive neurons which perceive noxious stimuli. Under normal physiological conditions, it is right, in general, that nociceptors sense nociception induced by noxious stimuli, such as pricking, pinching, acid, alkali, heat, and cold. Nevertheless, transmission and interpretation of sensory information are complicated. For example, a person may not feel any pain when he/she is wounded under immense stress. Under pathological conditions or certain conditions, nociception or nociceptor may represent a manifestation of a clinical pain state, underscoring the need to differentiate the various mechanisms involved in nociception in order to devise strategic approaches to treating pain.

Nociception caused by cross-talking between A-beta fibers and A-delta or C-fibers

Neurosensory pain signals produced by noxious stimuli are conducted via C-and A-delta-fibers to reach the dorsal root ganglia and the dorsal horn of spinal cord. In parallel sensory signals produced by innocuous stimuli, such as touch, warm, vibration, are conducted in A-beta fiber to the dorsal root ganglia, then to the dorsal horn. The sensory signals are coded in a regular and special mode so that our sensory center in the spinal cord and brain can perceive and interpret which kind of senses received. After nerve or tissue injury, or diseases, pathological changes in the peripheral nerve include Wallerian degeneration and demyelination. As a result of this disruption of the nerve fiber integrity, A-beta fibers can cross-talk with A-delta or C-fibers, which will convert innocuous sensory signals (coded in A-beta fibers), into noxious signals (coded in A-delta and C-fibers). Innocuous sensations such as touch, warmth, or vibration can then be perceived as painful stimuli. Allodynia, induced by touch or warm stimulus, is an example for this cross-talk. The cross-talk can further take place between nerve fibers, in dorsal root ganglia, and spinal cord. Cross-talk from C and A-delta fibers to A-beta fibers is also possible in that noxious signals are interpreted as innocuous signals. Unlike noxious sensory signals, the misinterpreted innocuous sensory signals may be neglected by our sensory centers.

Nociception induced by altered nature of a neuron

In the dorsal root ganglia, neurons can be classified into small, medium, and large neurons according to their size. The small and medium-size neurons receive noxious stimuli-induced signals via C and A-delta-fibers, while the large-size neurons receive innocuous stimuli-induced signals via A-beta-fibers. Further, the small- and medium-size neurons in the dorsal root ganglia extend their axons into lamina I and II neurons of spinal dorsal horn, where the spinal center processes noxious signals. The large-size neurons send their axons into lamina III and IV neurons, where the innocuous signals are processed and interpreted. The features of a cell are determined by protein expression in this cell. For example, proteins, such as cholecystokinin (CCK), substance P, and tyrosine kinase A, are expressed in the small-size neurons of dorsal root ganglia under normal physiological conditions. In nerve injury or neuropathic disease states, some of these proteins can be expressed in the large and medium-size neurons. As a result of this protein expression, large and medium size neurons will have new functions, implying that the large-size neurons can sense or interpret noxious stimuli-like signals. A similar alteration for neuron's function may also occur in the dorsal horn. When the nature of large-size neurons is changed, these neurons could interpret innocuous signals as noxious signals, in which a light touch stimuli could be interpreted as a pain signal.

Both nociception and non-nociception perceived by wide dynamic range neurons

During electrophysiological experiments, neurons located in deep lamina III and lamina IV were found to respond to both noxious (pinch, heat) and innocuous (brush, touch) stimuli. These neurons are called wide dynamic range neurons. The experimental results suggest that these neurons are linked to several types of nerve endings which sense different sensations. Currently, our understanding on how a neuron differentiates nociceptive or non-nociceptive signaling from sensory receptors at the nerve endings is not well established. It is not clear that a neuron receives information from only one type or multiple types of sensory receptor at the nerve endings. Wide dynamic range neurons can sense both noxious and innocuous stimuli. But during nerve injury or neuropathic disease states, the functions of wide dynamic neurons become more complicated such that non-nociceptive signaling appears to become nociceptive signaling. Could we say the non-nociceptive receptors at the nerve endings became nociceptors? This question remains unclear. When using the word,

nociceptor or nociception, in relation to a pain state, we need to consider if the painful, noxious sensations are induced by noxious stimuli or innocuous stimuli that converted to painful stimuli. In this way, an enhanced understanding of our mechanisms of nociception would aid the clinician in providing a more thorough assessment in the management of patients with chronic pain syndromes.

Acknowledgments

None.

Conflicts of Interset

None.

Citation: Jian-Guo C, Eugene F. Do nociceptors and nociception solely imply noxious stimuli? *J Anesth Crit Care Open Access*. 2014;1(1):1–2.
DOI: 10.15406/jaccoa.2014.01.00001