What Is Pain? How Brains Make Nociceptive Sensations

Contents

Abstract

Brain transforms perceptual properties into patterns and motions of geometric-algebra vectors, making microscopic surface textures whose spatial and temporal properties are sensory experiences.

Keywords

acupuncture, anterior cingulate gyrus, aspirin, bradykinin peptide, calcitonin peptide, cingulate cortex, cytokine, dendritic cell, dorsal horn, dorsolateral funiculus, dynorphin, endomorphin, frontal lobe, gate control theory, glial activation, glutamate receptor, glycine receptor, histamine, interleukin, Lissauer's tract, locus coeruleus, microglia, myofascial nerve, nerve-associated lymphoid cell, nerve growth factor, neurokinin, N-type calcium channel, nitrous oxide, NMDA receptor, nociceptor, nucleus raphe magnus, nucleus tractus solitarius, opiate, paraganglia, prostaglandin, protein hormone, protopathic pathway, raphé nuclei, reticular formation, spinoreticular tract, spinothalamic tract, substance P, substantia gelatinosa, thalamus, transcutaneous electrical nerve stimulation, vagus nerve

1. Anatomy

Pain chemical receptors send to dorsal-horn neurons, which send to cortical regions.

1.1. Chemical receptors

Skin and body pain receptors {nociceptor} chemically bind endomorphins, prostaglandins, bradykinin peptides, and protein hormones, molecules released by inflammation and tissue damage [Woolf and Salter, 2000].

1.2. Neurons

All neurons that receive input from nociceptors have glutamate receptors {NMDA receptor}. Dorsal-horn neurons have NMDA receptors with the NR2B subunit.

Some dorsal-horn neurons have substance-P (neurokinin-1) receptors. Some dorsal-horn neurons have calcitonin peptide receptors.

Some connective-tissue dendritic cells {nerve-associated lymphoid cells} have interleukin-1 binding sites and send to sensory vagus-nerve paraganglia [Goehler et al., 1999].

Abdominal pain signals travel in subdiaphragmatic vagus nerve to nucleus tractus solitarius, nucleus raphe magnus, and spinal-cord dorsolateral funiculus [Ritter et al., 1992].

1.3. Brain

Spinothalamic tract, Lissauer's tract, and protopathic pathway carry pain information.

Cingulate cortex receives pain information [Chapman and Nakamura, 1999]. Reticular formation and spinoreticular tract regulate pain. Anterior cingulate gyrus, locus coeruleus, raphé nuclei, thalamus, and frontal lobe affect pain.

Throbbing pain, burning pain, and sharp pain use different brain regions.

Feeling pain and reacting to it involve separate brain pathways.

2. Physiology

Tissue damage, inflammation, and high-intensity stimuli release chemicals that excite nociceptors.

2.1. Nociceptors

Blows to body release bradykinins and prostaglandins, which excite nociceptors. High pressure, high temperature, harsh sound, intense light, sharp smells and tastes, and inflammation release chemicals that excite nociceptors.

Nociceptors can be for histamines, endomorphin prostaglandins, small bradykinin peptides produced by peripheral inflammation, and protein hormones, such as nerve growth factor. Pain-causing molecules vary in size, shape, chemical site, and vibration state.

2.2. Glial activation

Damaged tissue activates pain-activated microglia (immune cells), which release pro-inflammatory cytokines, which activate glia. (Other glia types do not release cytokines in response to pain.) Spinal glial activation affects nociceptive neurons at NMDA receptors and amplifies pain [Watkins et al., 2001].

2.3. Neurons

Pain involves too much small-nerve-fiber activity, uninhibited by large neurons.

2.3.1. Pain control

Brain pain control uses prostaglandins to block glycine receptors at first synapse, near spinal cord, and so excite dorsal-horn neurons.

Spinal-cord dorsal-horn substantia-gelatinosa neural circuits receive brain signals that inhibit nerve-impulse flow from spinal cord to brain [Melzack, 1973] [Melzack, 1996] {gate control theory of pain}, to close the gate. Large-fiber inputs, such as from gentle rubbing {counterstimulation}, also stimulate substantia-gelatinosa neurons to inhibit signal flow, to close the gate. Small-fiber inputs, such as from pinching {diffuse noxious inhibitory control} {counterirritation}, inhibit substantia-gelatinosa neurons and so release signal flow, to open the gate.

Endorphin and dynorphin inhibit pain nerve pathways. For example, flight-or-fight responses use endorphin neurotransmitters to suppress pain. Opiate drugs, such as morphine, are similar to endorphin and suppress pain. Stimulating the brain area that makes endorphin and dynorphin {transcutaneous electrical nerve stimulation} inhibits pain pathways. Acupuncture-needle insertion sites correspond to myofascial-nerve locations, and acupuncture-needle stimulation activates brain area that makes endorphin and dynorphin. Aspirin and nitrous oxide also alleviate pain.

Inhibiting N-type calcium channels lessens pain.

2.4. Brain

Pain perception uses thalamus and is not conscious. Pain sensation uses cerebral cortex and is always conscious. More and wider brain activation indicates more pain
[Chapman and Nakamura, 1999].

Randomly placed brainstem electrodes produce pain 5% of time. Direct cerebral-cortex stimulation can cause other sense qualities but never causes pain. Cortex stimulation does not decrease pain.

2.5. Effects

The fundamental pain characteristic is repulsion or withdrawal [Duncker, 1941]. Pain causes people to push painful object farther away or to move farther from pain source. Sharp pain causes withdrawal reflexes, writhing, jumping away, and wincing as people try to alleviate pain. Writhing typically escapes stimulus or pushes away stimulus.

To avoid reinjury and allow body to rebuild rather than use, dull and chronic pain reduces overall activity.

Painful skin stimuli cause flexion reflexes. Muscle contractions inhibit blood flow and squeeze out poisons.

To allow recovery from tissue damage, pain causes attention. To avoid future pain causes, pain triggers learning about possibly painful situations. People also learn pain responses.

Pain can cause anxiety, depression, increase breathing rate, increase blood pressure, dilate pupils, increase sweat, and make time appear to flow more slowly.

3. Perceptual properties

Pain can be acute or dull. People can distinguish 10 pain levels.

Pains are not concepts, observations, or judgments. Pain is not intentional but is only about itself.

3.1. Acuteness

Pains are sharp or dull. Sharp pains have small area and high intensity. Dull pains have large area and low intensity.

3.2. Variability

Pains are stable or variable. Stable pains are steady. Variable pains are throbbing.

3.3. Temperature

Pains can be hot and burning or without temperature.

3.4. Mixing

Pains are separate and independent and do not mix, so pain is an analytic sense.

3.5. Source location

People perceive pain at body locations. Lower back pains are the most common.

People can detect different pain sources from one location. People can detect different pain sources from many locations simultaneously, sometimes with interference.

4. Relations to other senses

High-intensity vision, hearing, touch, temperature, smell, and taste are painful.

Pain has a location and so has touch.

Pain and touch started from the same basic sensation. Pains can feel like signal overload or vibration-wave distortions.

5. Pain sensations

Pain detects inflammation intensity.

6. Pain descriptors

Pain perceives pain strength.

Pain perceives pain acuteness. Pain can feel sharp or dull.

Pain perceives pain variability. Pain can feel throbbing or steady.

Pain perceives pain temperature. Pain can feel hot or neutral.

Pain causes muscle contractions, to get away from pain cause. Pains cause repulsions.

7. Spatiotemporal properties and patterns

Pain perceptual-property spatiotemporal patterns are lines.

Lines can have contractions, for getting away from pain. Contractions can be fast or slow speed, for sharp or dull, respectively. Contractions can be high or low frequency, for throbbing or steady, respectively. Contractions can be directed or random, for neutral or hot, respectively.

8. Machines

Machines can simulate pain sensations using a microscopic-surface-texture array with elements that have random vibrations. More elements represent higher intensities.

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