

Gate Control Theory of pain stands the test of time

**British Journal of Anaesthesia,
Vol. 88, No. 6, June 2002, Pgs. 755-757**

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THIS AUTHOR NOTES:

"In 1965, Pat Wall (who died August 8, 2001) and Ron Melzack published their paper in Science, entitled a 'New Theory of Pain'."

The GATE THEORY "has stood the test of time."

The theory stated "that the transmission of pain from the peripheral nerve through the spinal cord was subject to modulation by both intrinsic neurones and controls emanating from the brain."

Because of the GATE THEORY, the present emphasis in pain control is that "pain could be controlled by modulation—reduce excitation or increase inhibition."

[IMPORTANT]

The Gate Theory proposed:

- (1) that small "C" fiber nociceptors activated excitatory pain systems,
- (2) and that this pain system excitation is "controlled by the balance of large fiber [mechanoreceptors]" which are "under the control of descending systems."

[This is the neurological basis for chiropractic adjustments helping with pain]

"The Gate Theory did not emphasize peripheral processes since the aim was to propose how the central nervous system dealt with sensory inputs."

"The main clinical pains arise from damage to tissue (inflammatory pain), whereas neuropathic pain results from changes in damaged nerves." **[IMPORTANT]**

Both inflammatory tissue damage pain and neuropathic pain "cause profound changes in the spinal cord and the brain."

"We now believe that all persistent pains exhibit plasticity in that the peripheral and central signalling mechanisms can alter."

[Synaptogenesis/Neuroplasticity]

"When tissue is damaged, peripheral [inflammatory] chemicals sensitize the sensory endings." **[Inflammatory Altered Thresholds]**

In neuropathic pain, "excitability changes occur within the nerve itself."

Nerve damage would be expected to cause sensory loss, not increased pain. "It is possible that increased central hyperexcitability is a maladaptive compensation for the marked loss of peripheral input that occurs after nerve injury."

The Gate Theory looks at the balance "between local and distant excitatory and inhibitory systems in the dorsal horn." **[Again, chiropractic Adjustment]**

"Inflammation will produce peripheral sensitization in the system that will be driven harder for a given stimulus." **[Inflammatory Altered Thresholds]**

"Ongoing ectopic activity in damaged peripheral nerves will continually produce transmitter release into the spinal cord, and this will cause subsequent neuronal activity."

Tissue and nerve injury increases the activity of calcium channels in the spinal cord, increasing glutamate transmitter release and increasing neuronal excitability. **[Glutamate is the pain neurotransmitter. Is it in your food?]**

Now pain is evoked by low-threshold peripheral stimuli. **[Altered Thresholds]**

During inflammation, calcium channels release glutamate into the spinal dorsal horn. **[Glutamate Again]**

Thus, as there is augmented transmitter release, an increased release of glutamate, the major transmitter in afferent A- and C-fibers, has been shown to occur in the human spinal cord of patients after nerve injury. **[Have you notice how many processed and packaged foods include glutamate?]**

"Increased glutamate then leads to enhanced activation of the receptors for glutamate, especially the N-methyl-D-aspartate (NMDA) receptor implicated in wind-up and central sensitization." **[Glutamate Again]**

"Central sensitization occurs when peripheral sensory neurone activity drives central spinal systems that amplify and prolong the incoming sensory messages."

"One manifestation of central sensitization is wind-up where repeated constant C-fibre stimulation elicits increased spinal neuronal responses and pain." **[Wind-Up]**

"As spinal neurones become more excitable, their receptive fields expand and this is thought to be a major factor in secondary hyperalgesia." **[Receptive Field Enlargement]**

"Glutamate is believed to be a key transmitter in central sensitization," and in the spinal cord plays a pivotal role in determining the level of pain transmission. **[Glutamate Again]**

In the dorsal horn, the NMDA receptor then causes wind-up. **[Wind-Up, again]**

“This enhances and prolongs transmission and so has been implicated in many states of central hypersensitivity, including hyperalgesia and allodynia seen in postoperative, inflammatory and neuropathic pains.”

Inflammatory prostaglandins are produced by NMDA receptor activation and further enhance pain signaling. **[Like the omega-6 derived PGE2]**

The inhibitory systems use opioids, but opioid receptor sensitivity can be reduced by chronic pain.

Morphine is an opioid pain inhibitor.

Gamma aminobutyric acid (GABA) is an inhibitory neurotransmitter pathway descending from the brain to the spinal cord.

KEY POINTS FROM DAN MURPHY

- (1) This article reviews our model of inflammatory altered thresholds, receptive field enlargement, synaptogenesis/neuroplasticity, and wind-up.
- (2) This article documents the adverseness of glutamate in both acute and chronic pain syndromes. **[Don't ingest glutamate, i.e. MSG]**
- (3) This article supports chiropractic spinal adjustments by noting that pain perception is a balance between small diameter nociceptors and large diameter mechanoreceptors [which we fire with spinal adjustments, tissue work, and exercise].