치과진료시의 통증관리

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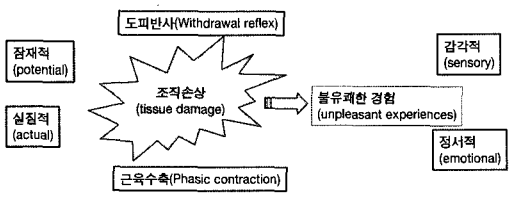
통증의 정의

IASP, 1994

An unpleasant sensory and emotional experiences associated with actual and potential tissue damage, or *described in terms of such damage*

실질적인 또는 잠재적인 조직손상과 그와 관련된 감각적이고 정서적인 불유쾌한 경험



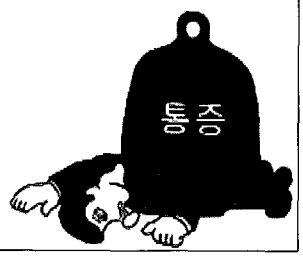
"Pain is the fifth vital sign"

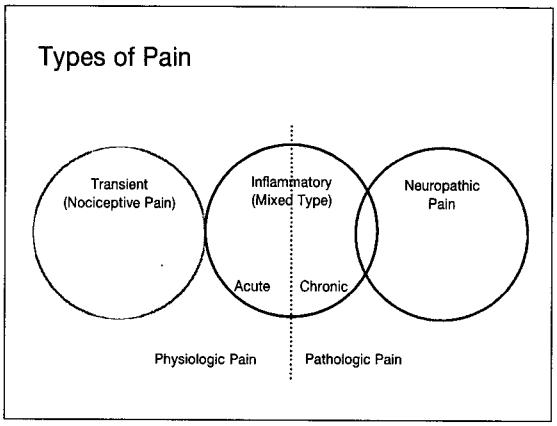
생체의 이상을 신속히 알리고 경고하는 중요한 방어기전

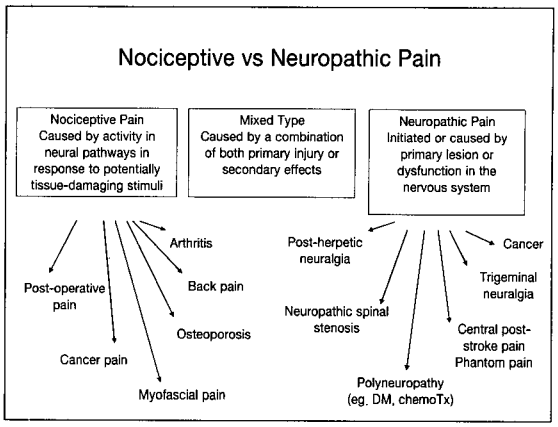
신체에 발생한 이상상태를 정상상태로 회복시키고자 하는 생체방어기전의 일환으로 경고의 뜻을 가짐

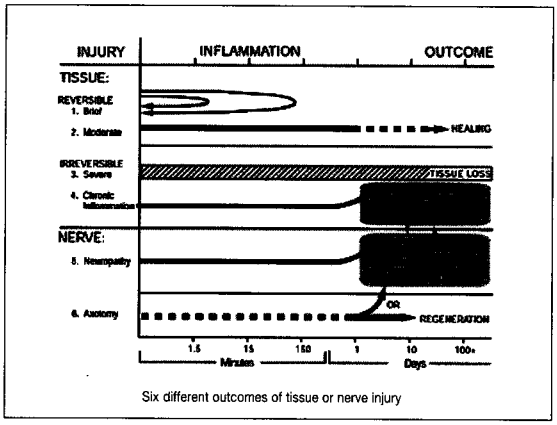
통증의 정의

- 본래의 목적과는 달리 통증이 격심해지거나 만성화 될 때 병적통증
  - 불안
  - 절망감
  - 식욕부진
  - 영양장애
  - 전신상태 악화
  - 수명단축

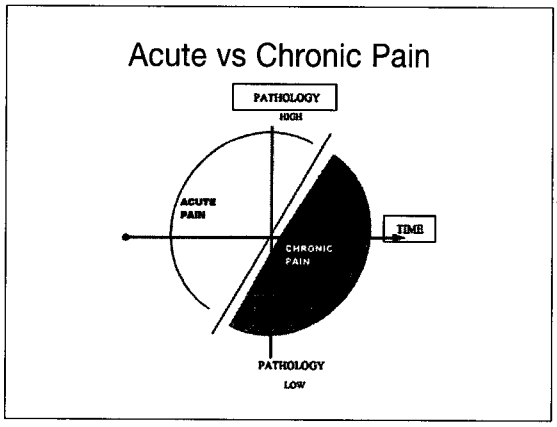








Six different outcomes of tissue or nerve injury



Acute vs Chronic Pain

Characteristic	Acute Pain	Chronic Pain
Cause	Generally known	Often unknown
Duration of pain	Short, well-characterized	Persists after healing, ≥3 mo
Treatment approach	Underlying disease	Underlying disease and pain disorder

급성통증의 유해한 생리적 영향

기관		임상효과
호흡기계	골격근 긴장, 폐탄성 감소 폐활량 및 기능적 잔기량 감소	저산소혈증 무기폐, 폐렴
심혈관계	심근운동 증가 심근산소소모량 증가 (교감신경계 항진으로)	혈압상승, 빈맥, 전신혈관저항 증가 부정맥, 협심증, 심근경색 울혈성 심부전
내분비계	Aldosteron, ADH, cortisol 분비 증가 Glucagon 및 epinephrine 분비 증가 Insulin 분비 감소	염기 및 수분 저류 지방분해 고혈당증
소화기계 및 비뇨기계	괄약근 수축 평활근 이완	장운동 지연, 장폐쇄증 뇨저류
혈액응고계	혈소판 응집력 증가 섬유소용해 감소 응고체계 활성화	혈전색전증 발생빈도 증가; 심부정맥 혈전증, 폐혈전증
면역계	면역기능 억제	상처 치유 지연, 감염

급성통증(Acute Pain)

조직손상 ⇨ 염증반응 ⇨ 병적통증유발

1. 외부자극과 무관한 통증
2. 골격근 반사성 수축, 교감신경항진: 근육통
3. 통각과민; 손상부위 및 주변부
4. 이질통; 무해한 자극에 통증

"통증은 참는 것" "아파야 낫는다" ⇨ "통증은 조기에 적절히 치료되어야 한다"

Rationale

통증은 일차적인 방어기능이지만 염증성 반응이 초래될 정도의 손상으로 유발된 통증은 여러 종류의 병태생리학적 변화를 초래한다

1. 신경호르몬 반응: 내분비 반응, 교감신경-부신피질 반응
2. 통증 전달과정의 변화: 말초감작, 중추감작

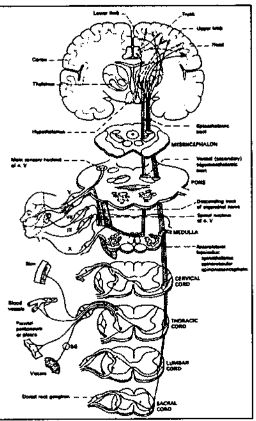
통증의 전달

"If for example fire (*A*) comes near the foot (*B*), the minute particles of this fire, which as you know move with great velocity, have the power to set in motion the spot of the skin of the foot which they touch, and by this means pulling upon the delicate thread (*c*) which is attached to the spot of the skin, they open up at the same instant the pore (*d*,*e*) against which the delicate thread ends, just as by pulling at one end of a rope makes to strike at the same instant a bell which hangs at the other end."

Descartes' (1664) concept of the pain pathway

Pain Pathways

- Transduction
- Transmission
- Modulation
- Perception
- Interpretation
- Behavior



통증의 전달

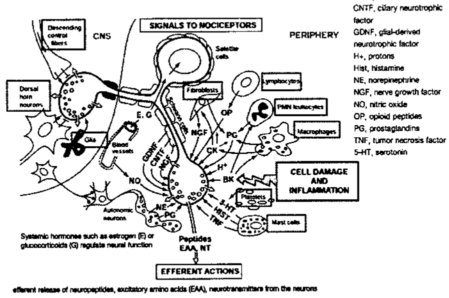
침해수용체 (nociceptor) ⇨ 척수 (spinal cord) ⇨ 뇌 (brain)

일차구심로 섬유

Lloyd/Hunt system	Diameter (µm)	Letter system	Conduction velocity (m/sec)	Myelin	Receptor/ending types
I-a*	12–20	—	70–120	+	Muscle spindle primary endings
I-b*	12–20	—	70–120	+	Golgi tendon organs
—	12–20	A-α	70–120	+	Muscle efferents (extrafusal)
II	6–12+	A-β†	30–70	+	Encapsulated endings (Meissner, Ruffini, Pacinian; Merkel intraepithelial; muscle spindle secondary endings
—	2–10	A-γ	10–50	+	Muscle efferents (intrafusal)
III	1–6	A-δ	5–30	+	A-δ specific nociceptors; A-δ polymodal receptors; cold receptors; most hair receptors; some visceral receptors
—	<3	B	3–15	+	Preganglionic autonomic
IV	<1.5	C	0.5–2.0	No	C-nociceptors; C-polymodal receptors; some visceral receptors; warmth receptors; some mechanoreceptors; postganglionic autonomic; enteric nerve fibers

*Data mostly from cats. Velocity in primates and humans has been measured up to 80 m/sec.
†Some afferents such as joint afferents can conduct up to 17 m/sec. These are sometimes classified as A-α, but sometimes all nonmuscle afferents are all considered to be A-β.
Adapted from Nolte J. The human brain. St. Louis: Mosby, 1993:213.

Summary in peripheral nociception



BK, bradykinin
CK, cytokines
CNTF, ciliary neurotrophic factor
GDNF, glial-derived neurotrophic factor
H+, protons
Hist, histamine
NE, norepinephrine
NGF, nerve growth factor
NO, nitric oxide
OP, opioid peptides
PG, prostaglandins
TNF, tumor necrosis factor
5-HT, serotonin

Type	Ligands	Source(s)	Receptors[a]
Amino acids	Glutamate	PAF[c], serum, macrophages	NMDA; AMPA, mGlu
	GABA	PAF, plasma	$GABA_A$, $GABA_B$
Cholinergic	Acetylcholine	Keratinocytes, PAF	Nicotinic: nAchR, muscarinic: mAchR
Biogenic amines	Norepinephrine	Sympathetic	Adrenergic α_2[d]
	Serotonin (5-HT)	Mast cells, platelets	$5\text{-}HT_{1,2,3}$
	Histamine	Mast cells	$Histamine_{H1,H2}$
Nucleotides	Adenosine	Damaged cells, PAF	Adenosine-R
Purines	Adenosine triphosphate	PAF, cell damage	$P2X_{2,3}$
Vanilloids	Capsaicin	Exogenous	VR-1
Cannabinoids	Anandamide	Serum	CB1,2
Prostanoids	Prostaglandin E_2	Sympathetic macrophages	PGE_2-R
Ions	Hydrogen (H+)	Tissue damage	DRASIC
Cytokines	Interleukin-1β	Macrophages	IL1-R
	Interleukin-6, leukocyte inhibitory factor, ciliary neurotropic factor	Fibroblasts, macro, Schwann	gp130, LIF-R, CNTF-R
Corticosteroids	Glucocorticoids,	Serum	CR
	estrogen	Serum	ER
Neuroactive peptides	Substance P	PAF	NK1
	Neurokinin A	PAF	NK1, 2
	Bradykinin	Plasma	BK1, 2
	Cholecystokinin	PAF	CCK-R
	Somatostatin	PAF	SOM-R
	Bombesin	PAF	Bomb-R
	Angiotensin II	Serum	ANG-R
	Neuropeptide Y	PAF	Y1
	Corticotropin releasing factor	Hypothalamus	CRF-R
Opioid peptides	Endorphins	PAF, immunocytes	MOR
	Enkephalin	PAF, immunocytes	KOR
	Dynorphin	PAF, immunocytes	DOR
Growth factors	Nerve growth factor	Fibroblasts, PAF, Schwann	p75NTR, trkA
	Brain-derived neurotrophic factor	PAF	p75NTR, trkB
	Glial-derived neurotrophic factor	Schwann	Ret, GFR
	Fibroblast growth factor	Fibroblasts, Schwann[f]	FGF-R

AMPA, 3-amino-3-hydroxyl-5-methyl-4-isoxazole-propionic acid; CB, cannabinoid; DOR, delta opioid receptor; DRASIC, dorsal root acid-sensing ion channel; GABA, gamma aminobutyric acid; HT, hydroxytryptamine; KOR, kappa opioid receptor; mGlu, metabotropic glutamate; MOR, mu opioid receptor; NK, neurokinin; NMDA, N-methyl-d-aspartate; [illegible], purinergic; VR-1, vanilloid; Y1, neuropeptide Y.
[a]Factors are inhibitory or excitatory depending on concentration and on target cell pharmacology.
[b]Receptor abbreviations refer to ligand molecules. Other abbreviations are listed above.
[c]PAF, primary afferent fiber.
[d]α_2-Adrenergic receptors are expressed by A-δ neurons after nerve injury [illegible].
Adapted from Coggeshall and Carlton [illegible]; Rothwell and Hopkins [illegible]; Sorkin and Kadaba [illegible]; Dray [illegible]; Carlton and Coggeshall [illegible]; Zigmond et al. [illegible], and Millan [illegible].

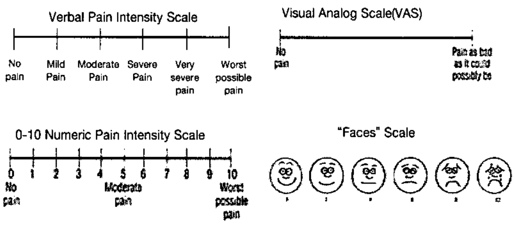
Some factors that affect nociceptors via membrane receptors

Peptide in dorsal root ganglion neurons	Axotomy	Inflammation
Substance P	− − −	+
Calcitonin gene-related peptide	− −	+
Somatostatin	− − −	?
Vasoactive intestinal polypeptide/peptide histidine-isoleucine	+++	. . .
Galanin	+++	−
Neuropeptide Y	+++	. . .
Pituitary adenylate cyclase activating peptide	+++	?
Cholecystokinin	− − +*	. . .
Y1-receptor	+	+
δ-Receptor	− −	−
μ-Receptor	−	− −
κ-Receptor	?	++
Neurotensin receptor	− −	?
Cholecystokinin-B receptor	+++	. . .
Nitric oxide synthase	++	. . .

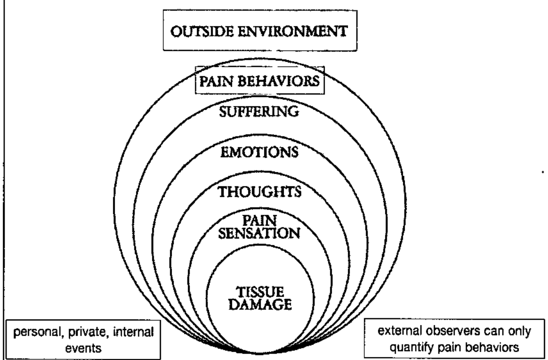
Explanation of symbols: No change (. . .); degree of increase: weak (+), moderate (++), large (+++); degree of decrease: weak (−), moderate (− −), large (− − −).
*Downregulation in small and upregulation in large neurons.
Modified from Hökfelt T, Zhang X, Xu ZQ, et al. Cellular and synaptic mechanisms in transition of pain from acute to chronic. In: Jensen TS, Turner JA, Wiesenfeld-Hallin Z, eds. *Proceedings of the 8th World Congress on Pain. Progress in pain research and management*. Vol 8. Seattle: IASP Press, 1997:133–153, with permission.

Sensory cytochemical changes with inflammation versus axotomy

Pain Assessment Scales



1. Portenoy RK, Kanner RM, eds. Pain Management: Theory and Practice. 1996;8-10.
2. Wong DL, Waley and Wong's Essentials of Pediatric Nursing 5th ed. 1997;1215-6.
3. McCaffery M, Pasero C. Pain: Clinical Manual. Mosby, Inc. 1999:16.



Loeser's model for components of pain

Assessing the Patients Who Has Pain

- Onset and Duration
- Location/Distribution
- Quality
- Intensity
- Aggravating/Relieving factors
- Associated features or secondary signs/symptoms
  - Mood/Emotional distress
  - Functional activities
- Treatment response

조직손상 억제 및 통증관리를 위한 다양한 방법들

1. 말초 침해수용체 활성 억제
   - 비스테로이드성 소염제 (NSAIDs)
   - 항히스타민제 (anti-histamine)
   - 항세로토닌제 (anti-serotonin)
2. 구심성 신경전도 차단
   - 말초신경 차단
   - 경막외 국소마취제 투여
3. 척수후각에서 전도 조정
   - 척수 아편양제제
   - 척수 α2 촉진제
4. 중추감작 억제
   - NMDA 길항제 (ketamine)
5. 중추신경 억제
   - 아편양제제
   - 항불안제
6. 급성 손상에 동반되는 이화반응 억제
   - Glucose, amino acid
   - Insulin
   - glucocorticoid
7. 신경반사 차단
   - 교감신경 차단제
   - 근육이완제

Step 3 Severe Postoperative Pain
Step 1 and Step 2 Strategies
AND
Local Anesthetic Peripheral Neural Blockade
(with or Without Catheter)
AND
Use of Sustained Release Opioid Analgesics

Step 2 Moderate Postoperative Pain
Step 1 Strategy
AND
Intermittent Doses of Opioid Analgesics

Step 1 Mild Postoperative Pain
Nonopioid Analgesic
Acetaminophen, NSAIDs, or COX-2 Selective Inhibitors
AND
Local Anesthetic Infiltration

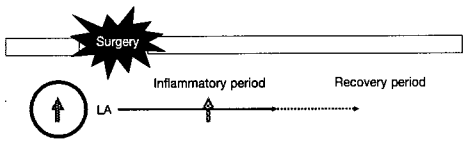
James C Crews JAMA 2002

Principles of Treatment

- Preemptive analgesia
  - The extent and duration of trauma and inflammation
  - Pre- to intra- and postoperative period
- Multimodal analgesia
  - More than two analgesics
  - Controlling the peripheral sensitization and conduction of primary afferent neuron
  - Local anesthetics, NSAIDs, opioid
- Mechanism-based therapy

Preemptive Analgesia

: Preoperative given before surgical injury



Peripheral sensitization
Inflammatory mediators lower the threshold of the nociceptors and render them responsive to less intensive stimuli.

Central sensitization
Nociceptor firing increases the receptivity of the dorsal horn to the A fiber stimulation.
Once initiated, can be quite prolonged.

The Changing Role of Non-Opioid Analgesic Techniques in the Management of Postoperative Pain

Paul F. White, PhD, MD, FANZCA
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center at Dallas, Dallas, Texas

the optimal non-opioid analgesic technique for postoperative pain management would not only reduce pain scores and enhance patient satisfaction but also facilitate earlier mobilization and rehabilitation by reducing pain-related complications after surgery

Recent evidence suggests that this goal can be best achieved by using a combination of preemptive techniques involving both central and peripheral-acting analgesic drugs and devices

(Anesth Analg 2005;101:S5–S22)

Clinical application

- Local anesthetic blockade
- Epidural opioid
- NSAIDs
- Ketamine
- Combination regimens

Controversial

1. Careful attention to preoperative induction
2. Prolongation of use well into the postoperative period
3. Use of multiple agent regimens

Relative efficacy of oral analgesics after third molar extraction

J. Barden,[1] J. E. Edwards,[2] H. J. McQuay,[3] P. J. Wiffen[4] and R. A. Moore[5]

BRITISH DENTAL JOURNAL VOLUME 197 NO. 7 OCTOBER 9 2004

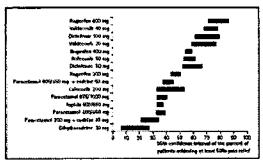


Fig. 1 The 95% confidence interval of the proportion of patients with at least half pain relief over 4-6 hours compared with placebo in third molar extraction trials

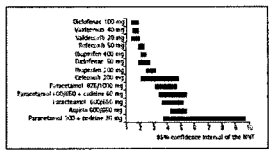
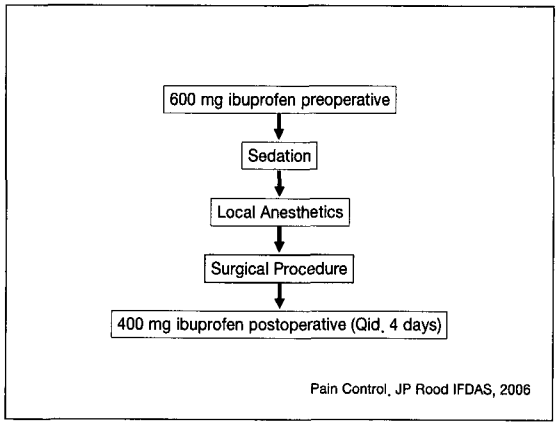


Fig. 2 The 95% confidence interval of the number needed to treat (NNT) for at least half pain relief over 4-6 hours compared with placebo in third molar extraction trials

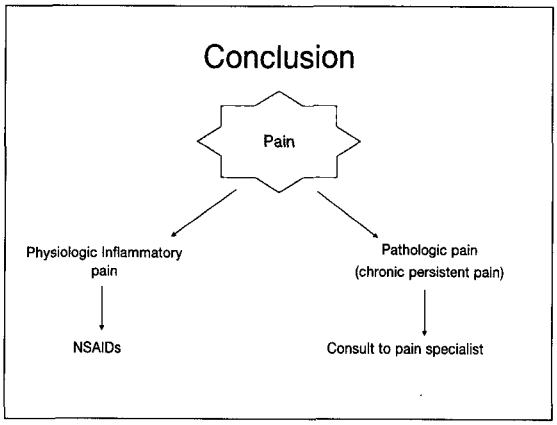
NSAIDs and COX-2 inhibitors have the lowest (best) NNTs. They may also have fewer adverse effects after third molar surgery, though conclusive evidence is lacking. At least 80% of analgesic prescribing by UK dentists is in line with the best available evidence on efficacy and safety.



The Key to Sedation

"Local Anesthetic Technique"

If a poor local anesthetic block has been given, the patient will continue to feel pain throughout the procedure



The art of life, is to live it without pain.

Thomas Jefferson 1790