QUẢN LÝ ĐAU TRONG ĐIỀU TRỊ THOÁI HÓA KHỚP

Dưới góc nhìn ngoại khoa

THS. BS Lê Đình Khoa

Trưởng khoa tái tạo khớp bệnh viện đa khoa Tâm Anh TPHCM

SĐT: 0775009969

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Content

- Introduction
- Pathophysiology
- Prevention strategies
- Experience


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20 %
- Hawker G et al, Health-related quality of life after knee replacement. J Bone Joint Surg. 1998
- Kahlenberg CA et al, Patient satisfaction after total knee replacement: a systematic review. HSS J. 2018
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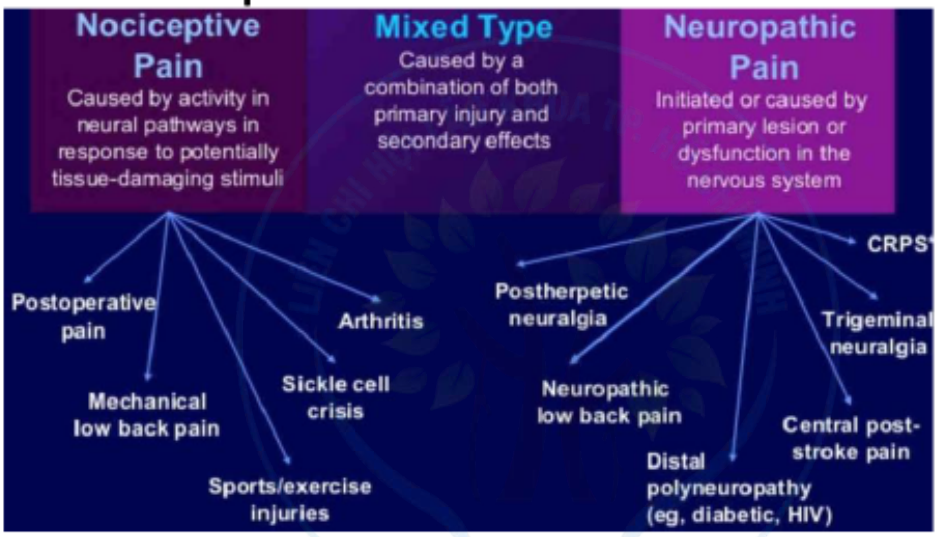
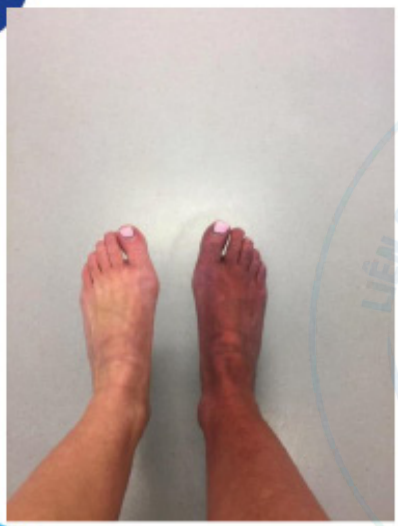
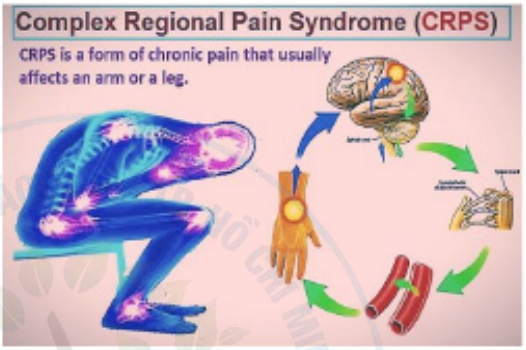
Chronic pain



Kela I, Kakarala C L, Hassan M, et al. Chronic Pain: A Complex Condition With a Multi-Tangential Approach. Cureus 13 (2021)

CRPS





- Increased pain sensitivity
- Change skin color and temperature
- Function ↓
- Swelling
- Depression

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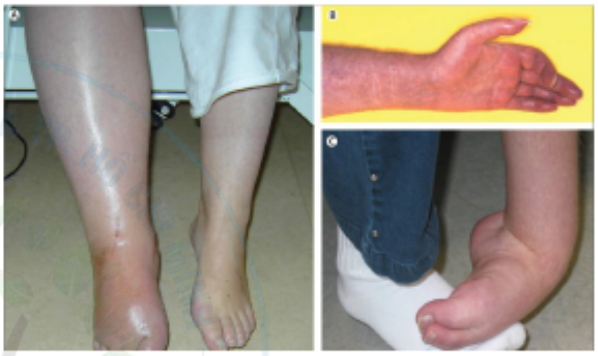
■ TRAUMA

Amputation in patients with complex regional pain syndrome

A COMPARATIVE STUDY BETWEEN AMPUTEES AND NON-AMPUTEES WITH INTRACTABLE DISEASE



Some are intractable like Complex Regional Pain Syndrome, until AMPUTATION…

Results

The amputation group showed consistently better results compared to the non-amputation group in the following parameters: median pain intensity (VAS): 80 (inter-quartile range (IQR) 13 to 92) *vs* 91 (IQR 85 to 100); p = 0.007; median SF-MPQ score 28 (IQR 9 to 35) *vs* 35 (IQR 31 to 38), p = 0.025; median PDI: 42 (IQR 11 to 64) *vs* 58 (IQR 50 to 62), p = 0.031; median BDI: 19 (IQR 5 to 28) *vs* 27 (IQR 21 to 32), p = 0.061 (borderline significant) and in six of the eight SF-36 domains.

Midbari A, et al. *Bone Joint J* 2016;98-B:548–54.

Take home message: Amputation should be considered as a form of treatment for patients with intractable CRPS.

Prevalence of chronic post-surgical pain

Review > Lancet. 2019 Apr 13;393(10180):1537-1546. doi: 10.1016/S0140-6736(19)30352-6.

Transition from acute to chronic pain after surgery

Paul Glare [1], Karin R Aubrey [2], Paul S Myles [3]

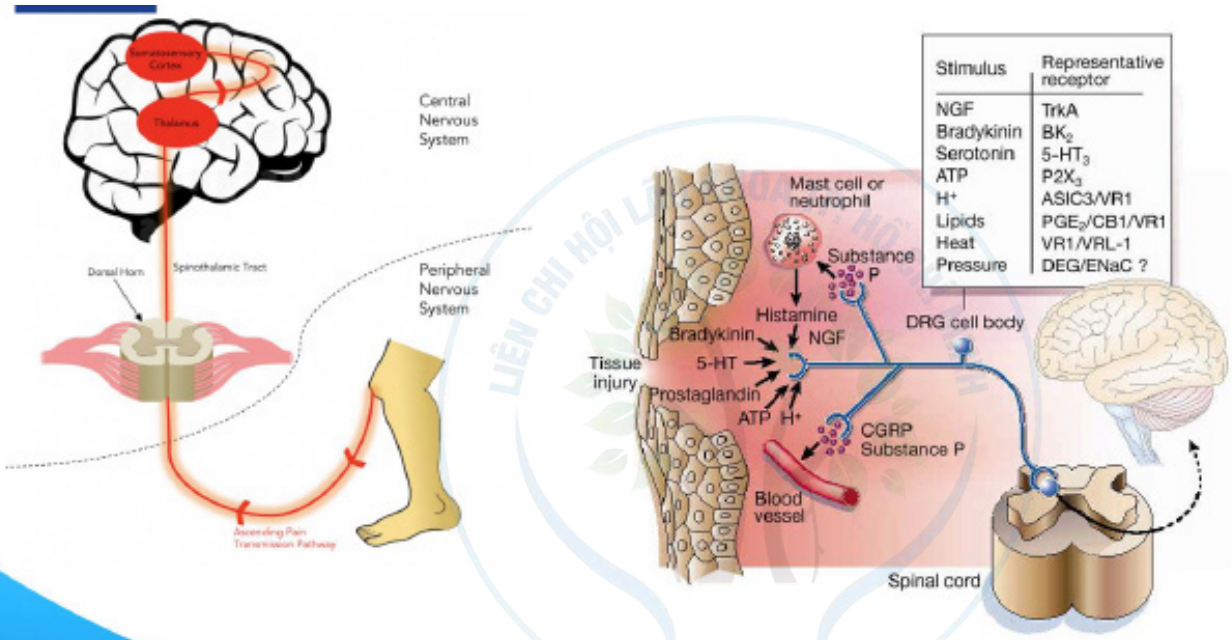
	Any intensity (%)	Moderate-severe intensity (%)	Prevalence (%); prevalence if restricted to a severe pain rating	Number of operations in US non-federal community hospitals* in 2014[7]
Amputation of limb	30–85%	5–10%	Up to 85%[8]	Not available
Arthroplasty, knee	13–44%	15%	44% (15%)[9]	723 086
Caesarean section	6–55%	5–10%	Up to 12%[10]	1 142 680
Cholecystectomy	3–50%[11]	Not reported	Not reported	300 245
Craniotomy	0–65%[12]	25%	12–16%[13]	Not available
Hip replacement	27%	6%	27% (15%)[9]	487 625
Inguinal hernia repair	5–63%	2–4%	6–29%[14]	Not available
Laminectomy and spinal fusion	10–40%	4–6[15]	5–36%[16,17]	564 911
Mastectomy	11–57%	5–10%	22%[18]	Not available
Coronary artery bypass graft	30–50%	5–10%	28% (4%)[19]	160 240
Thoracotomy	5–65%	10%	48%[20,21]	Not available

*Non-federal community hospitals account for 786 874 (87%) of 902 202 hospital beds in the USA.

Table 1: Prevalence of chronic postsurgical pain in common surgeries in the USA[11,22–24]

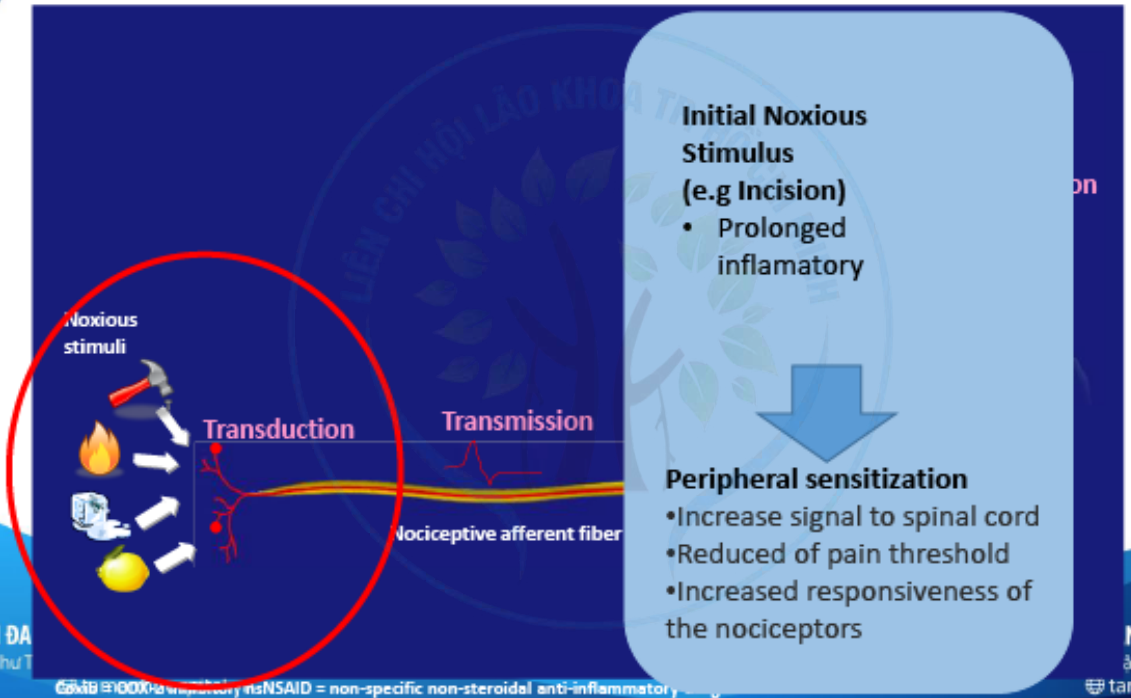
Pain is also the stubborn enemy with Orthopedic doctors and patients!

Glare P, et al. *Lancet* 2019;393(10180):1537-1546.

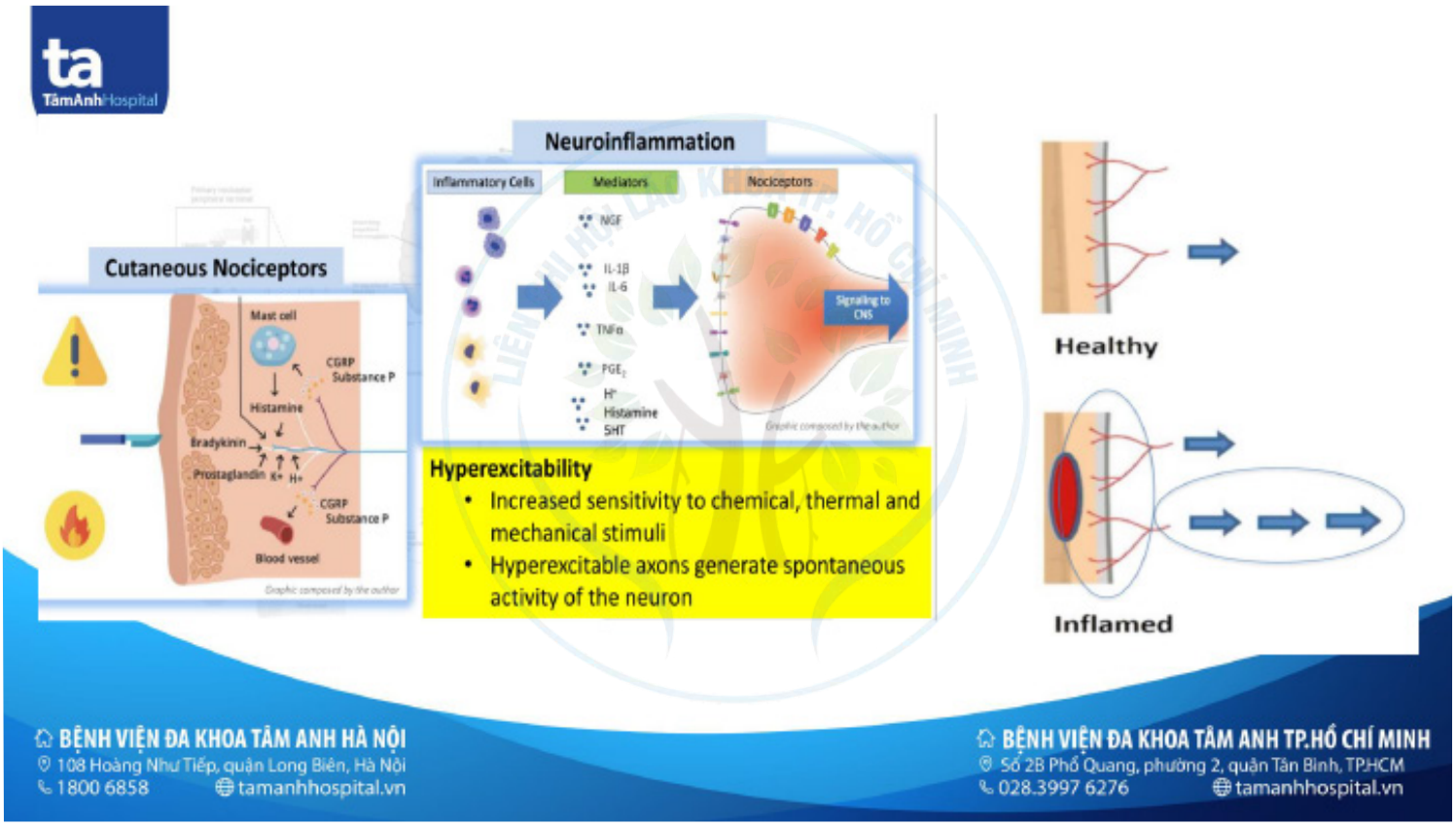


Julius D. & Basbaum A.I; Molecular mechanism of nociception; *Nature* 2001, Sept 13; 413 (6852): 203-10.

Mechanism of acute to chronic pain



Scholz J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7.


Cutaneous Nociceptors
Mast cell
CGRP
Substance P
Histamine
Bradykinin
Prostaglandin
K+
H+
CGRP
Substance P
Blood vessel
Neuroinflammation
Inflammatory Cells
Mediators
Nociceptors
NGF
IL-1β
IL-6
TNFα
PGE2
H+
Histamine
5HT
Signaling to CNS
Hyperexcitability
• Increased sensitivity to chemical, thermal and mechanical stimuli
• Hyperexcitable axons generate spontaneous activity of the neuron
Healthy
Inflamed
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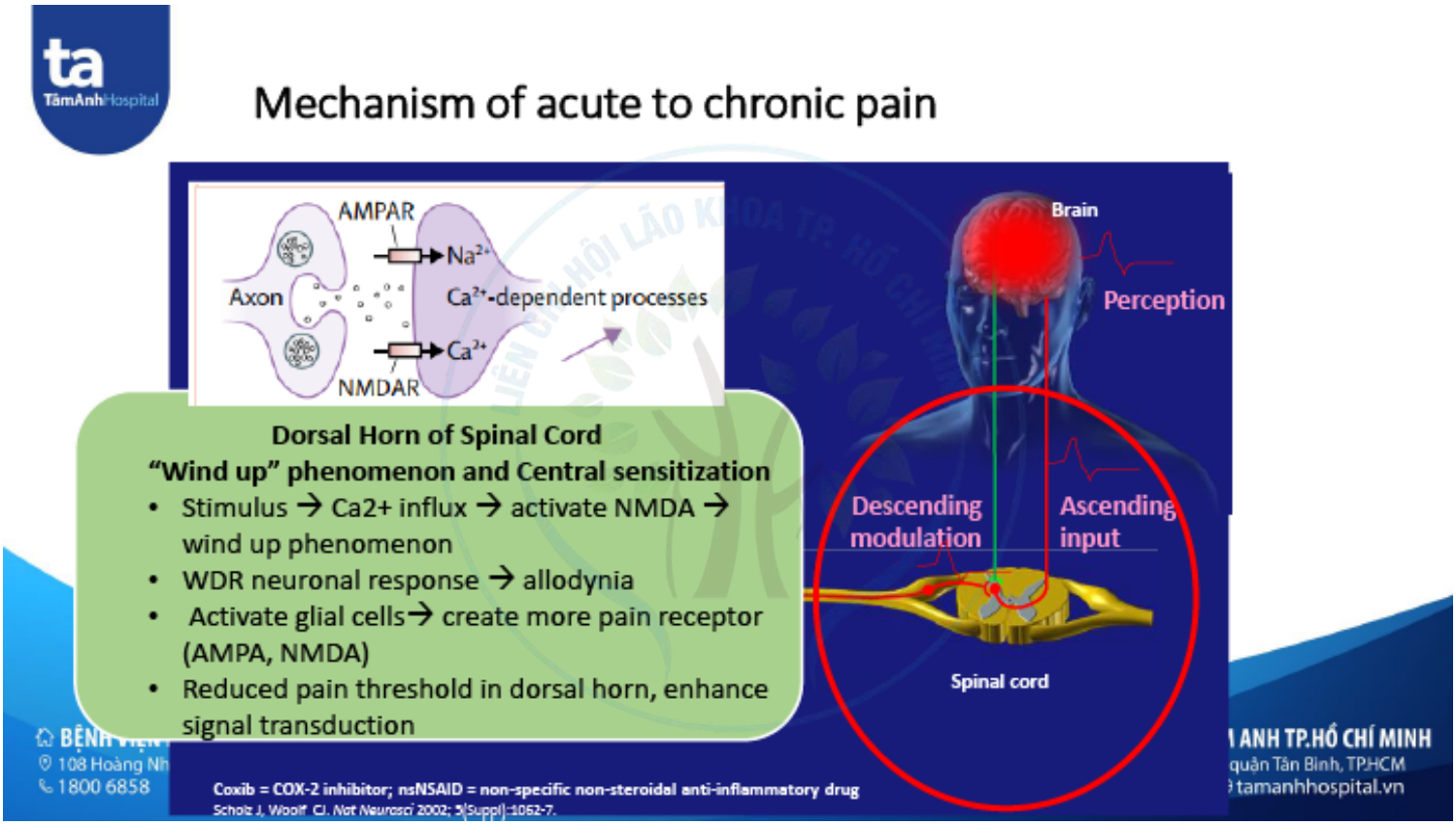
Mechanism of acute to chronic pain
AMPAR
Na2+
Axon
Ca2+-dependent processes
Ca2+
NMDAR
Brain
Perception
Descending modulation
Ascending input
Spinal cord
Dorsal Horn of Spinal Cord
"Wind up" phenomenon and Central sensitization
• Stimulus → Ca2+ influx → activate NMDA → wind up phenomenon
• WDR neuronal response → allodynia
• Activate glial cells→ create more pain receptor (AMPA, NMDA)
• Reduced pain threshold in dorsal horn, enhance signal transduction
Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug
Scholz J, Woolf CJ. Nat Neurosci 2002; 5(Suppl):1062-7.
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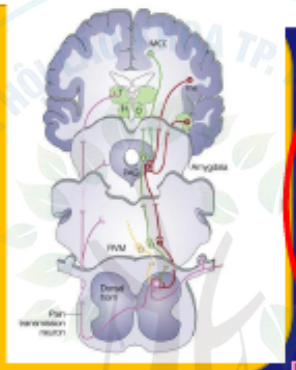
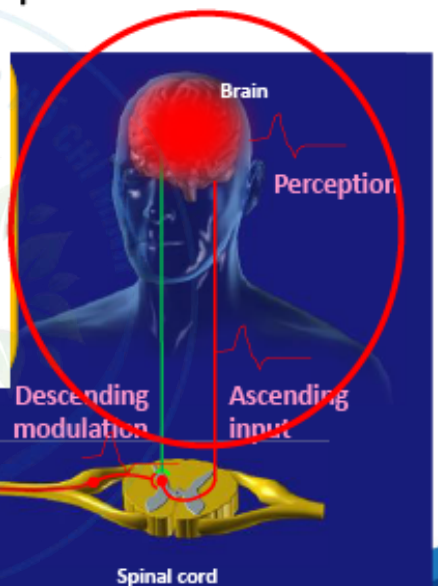
Mechanism of acute to chronic pain

Brain

Biopsychological contribute Central sensitization: Pain matrix

- Pain experience
- Mental status





Preexisting stress have a higher risk of developing CPSP.

Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug
Scholz J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7.

Central sensitization produces pain hypersensitivity by changing the sensory response elicited by normal inputs, including inputs that usually evoke innocuous sensations.

Chronic pain:
more about sensitivity than about injury

Peripheral sensitization
- Reduction threshold for nociceptor activation
- Increase in membrane excitability
- Primary allodynia and hyperalgesia

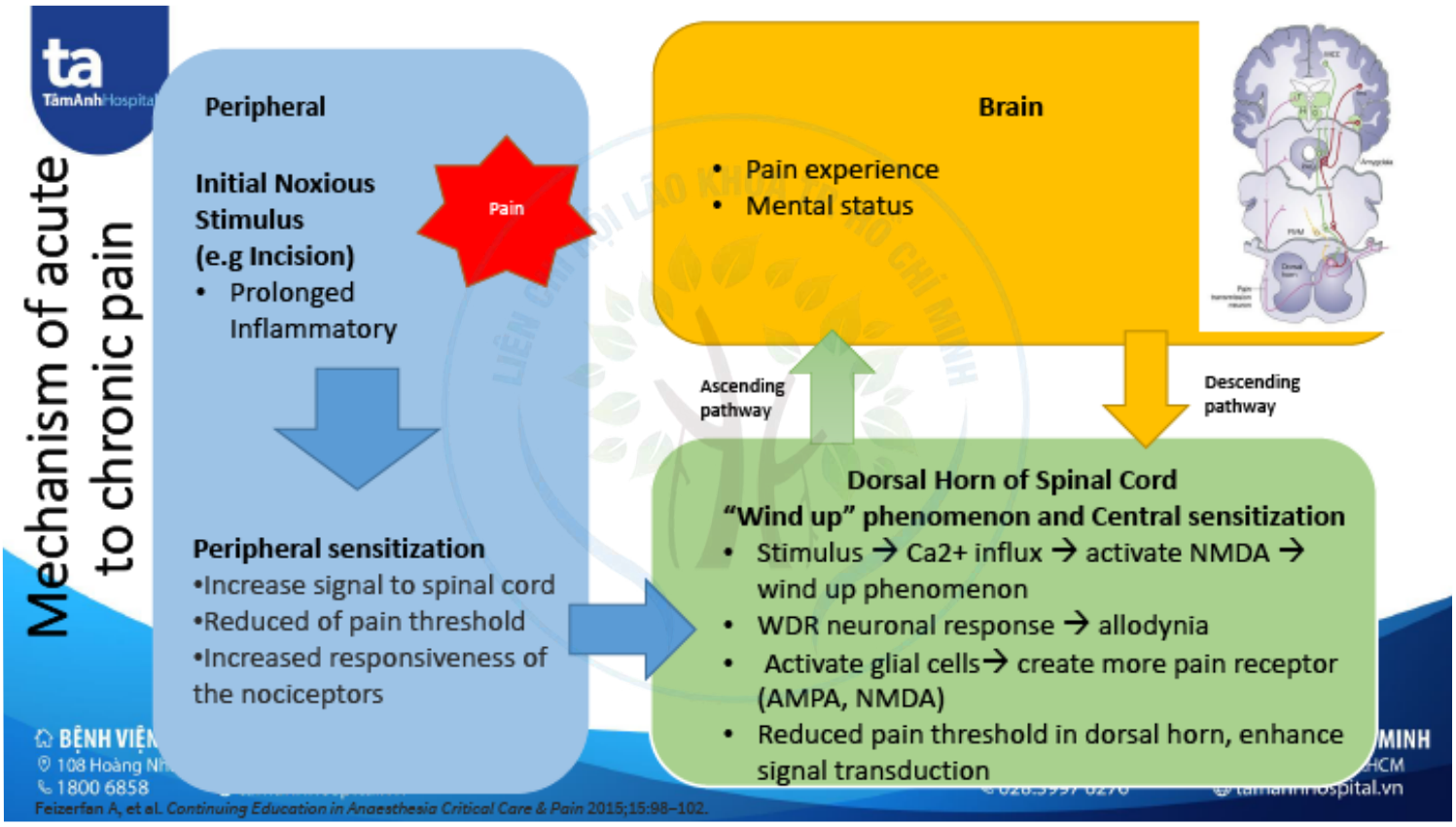
Central sensitization
- Amplification of synaptic strenghts in nociceptive circuits
- Secondary hyperalgesia

Failing descending inhibition
Signs of central sensitization

- Spreading of signs: more widespread referred pain
- Higher pain intensity
- Pain hypersensitivity
- Spontaneous pain
- Sleep disturbance
- Cognitive disturbance

Morlion B, et al. *Curr Med Res Opin*. 2018 Jul;34(7):1169-1178. Giron I, et al. *Lancet Neurol*. 2013;12:1084–1095.
Arendt-Nielsen L, Morlion B, Perrot S, et al. *Eur J Pain* 2018;22:216–41 Graphic composed by the author.

Main factors influencing the pain chronification process


Mechanism of acute to chronic pain
Peripheral
Initial Noxious Stimulus (e.g Incision)
• Prolonged Inflammatory
Pain
Peripheral sensitization
•Increase signal to spinal cord
•Reduced of pain threshold
•Increased responsiveness of the nociceptors
Brain
• Pain experience
• Mental status
Ascending pathway
Descending pathway
Dorsal Horn of Spinal Cord
"Wind up" phenomenon and Central sensitization
• Stimulus → Ca2+ influx → activate NMDA → wind up phenomenon
• WDR neuronal response → allodynia
• Activate glial cells→ create more pain receptor (AMPA, NMDA)
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Feizerfan A, et al. Continuing Education in Anaesthesia Critical Care & Pain 2015;15:98–102.


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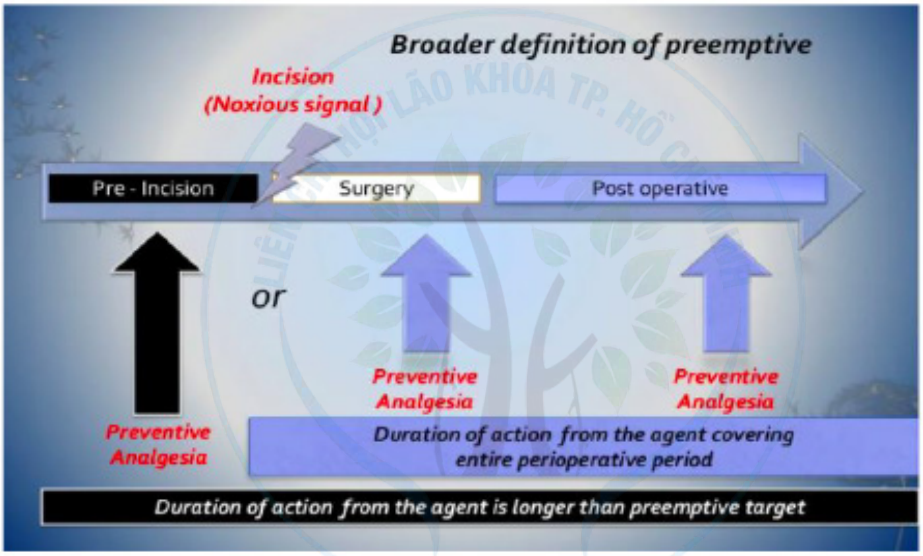
Preventing Analgesia



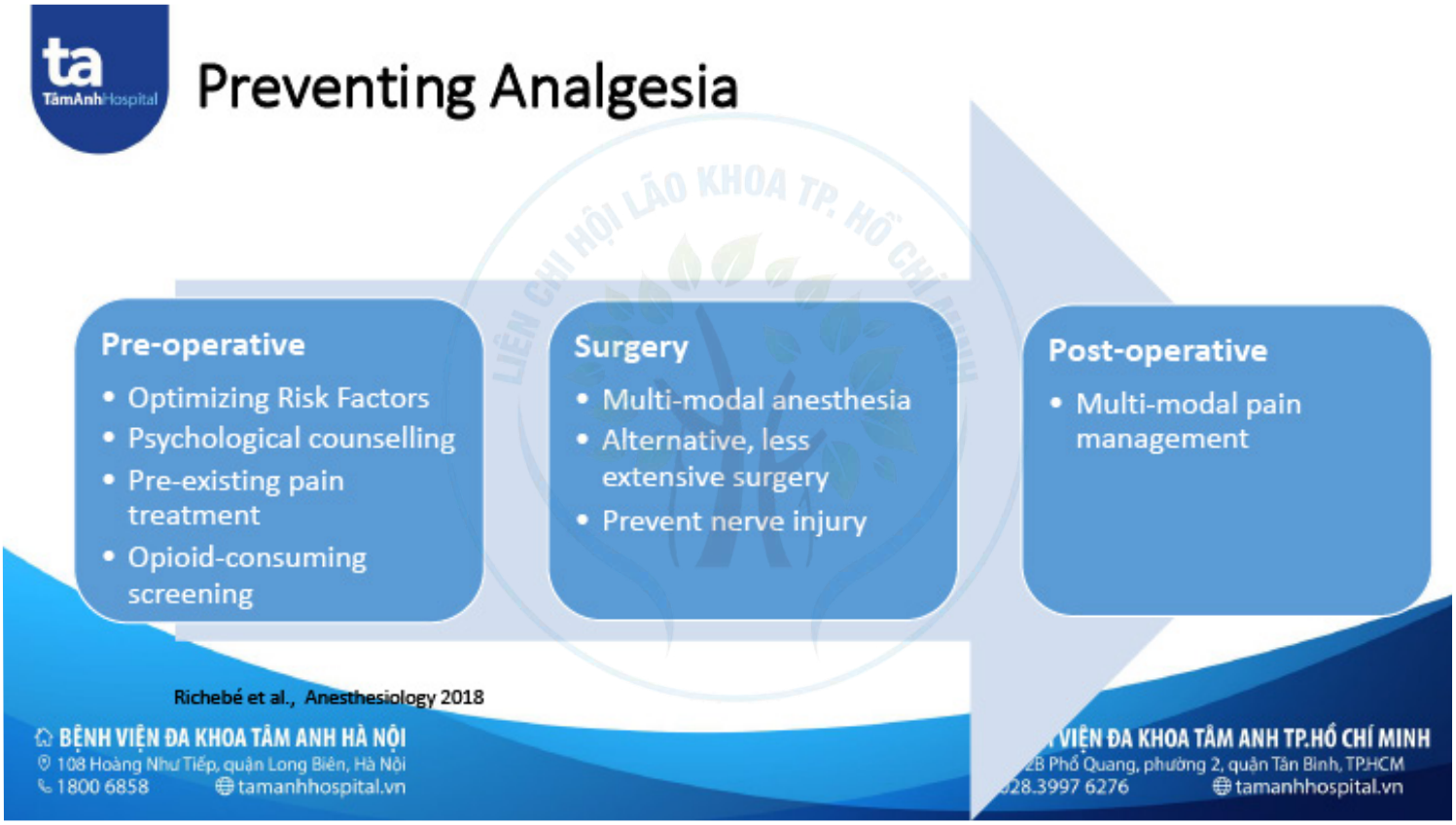
Kissin, Igor (2005). Preemptive Analgesia at the Crossroad. Anesthesia & Analgesia,

Pre-emptive vs. Preventing Analgesia

- Proportion of significant preventive effects (0.72) is significantly greater than the proportion of negative effects (0.28) across the different classes of drugs studied (p = 0.03)

Agents	No. of studies	Preemptive effects(%)		Preventive effects(%)		Opposite effects(%)	Total No. effects(%)
		Positive	Negative	Positive	Negative		
Local anesthetics	65	8(10.7)	16(21.3)	27(36.0)	18(24.0)	6(8.0)	75(100)
Opioids	25	7(25.0)	5(17.9)	10(35.7)	3(10.7)	3(10.7)	28(100)
NSAIDs	25	3(11.5)	12(46.2)	1(3.8)	8(30.8)	2(7.7)	26(100)
NMDA anatgonists	31	5(13.2)	6(15.8)	19(50.0)	7(18.4)	1(2.6)	38(100)
Clonidine	2	0(0.0)	0(0.0)	2(100.0)	0(0.0)	0(0.0)	2(100.0)
Local anesthetics and opioids	21	4(17.4)	5(21.7)	7(30.4)	6(26.1)	1(4.3)	23(100)
Multimodal	6	2(25.0)	0(0.0)	2(25.0)	3(37.5)	1(12.5)	8(100)
Total	175	29(14.5)	44(22.0)	68(34.0)	45(22.5)	14(7.0)	200(100)

Katz et al, 2008, Clinical Pain Management Second Edition: 154-198.


Preventing Analgesia
Pre-operative
• Optimizing Risk Factors
• Psychological counselling
• Pre-existing pain treatment
• Opioid-consuming screening
Surgery
• Multi-modal anesthesia
• Alternative, less extensive surgery
• Prevent nerve injury
Post-operative
• Multi-modal pain management
Richebé et al., Anesthesiology 2018
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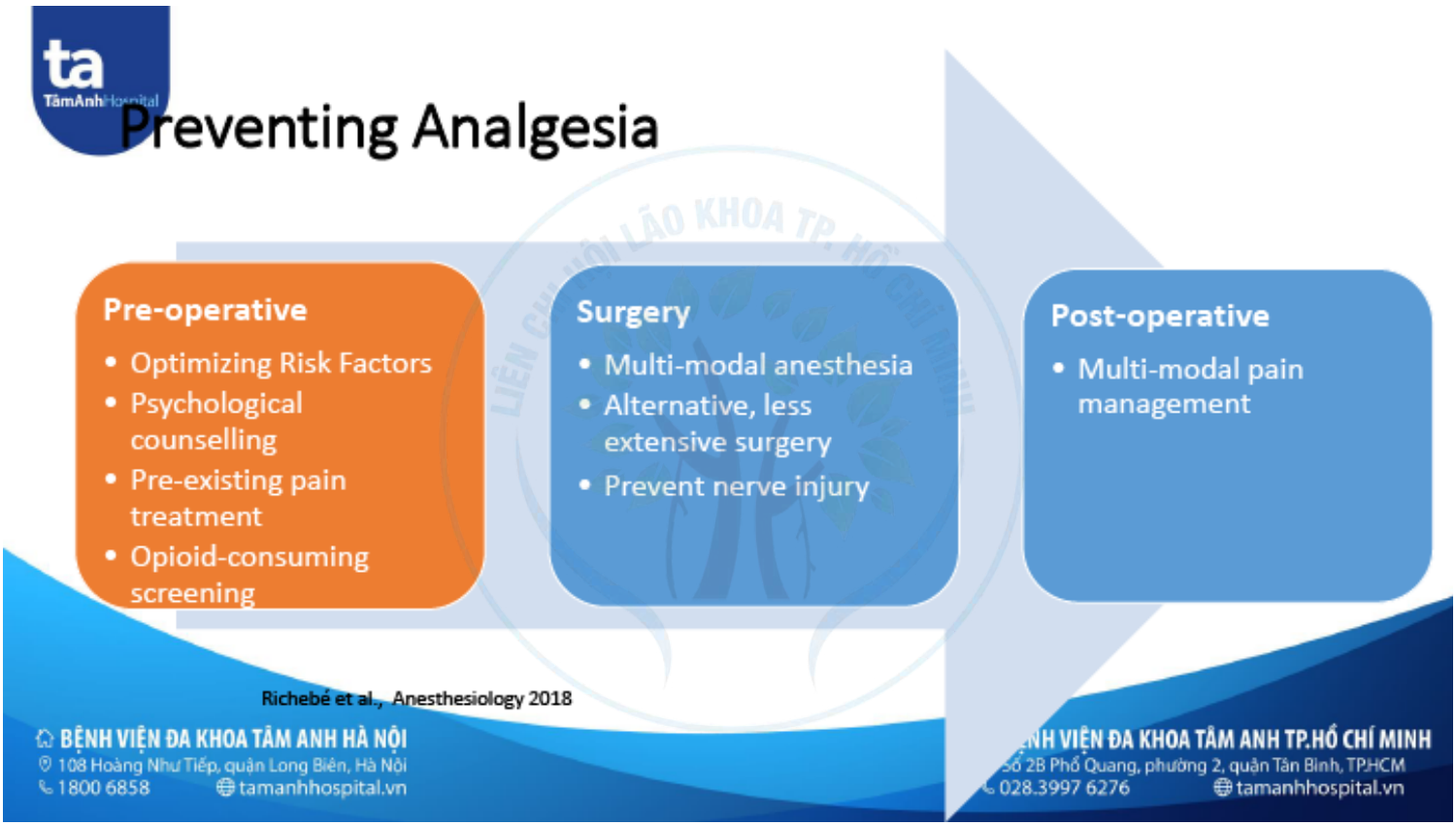
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Preoperative Phase:

- Education and Expectations
- Optimize pain control, anxiety/depression
  Involve family members
- Premedication:
  - Nsaid/Coxib; Acetaminophen;
  - Gabapentinoid

Preventing Analgesia

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- Opioid-consuming screening

Surgery
- Multi-modal anesthesia
- Alternative, less extensive surgery
- Prevent nerve injury

Post-operative
- Multi-modal pain management

Richebé et al., Anesthesiology 2018

Neuraxial and general anesthesia for outpatient total joint arthroplasty result in similarly low rates of major perioperative complications: a multicentered cohort study

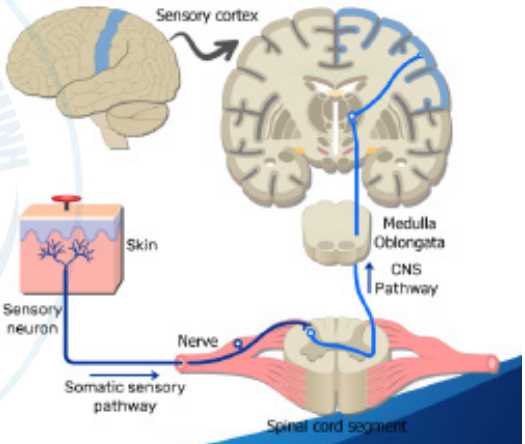
Edward Yap ,[1,2] Julia Wei,[3] Christopher Webb,[1,2] Kevin Ng,[4] Matthias Behrends[2]

Original research

Table 3 Secondary outcomes for outpatient total knee and hip arthroplasty† §

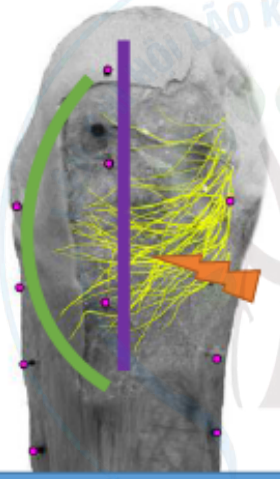
Postoperative outcome	Anesthesia type: Neuraxial n=10 003	General n=1520	P value
Pain and PONV outcomes			
Intraoperative opioid (MME), median (Q1–Q3)	0 (0–22.5)	40 (19–65)	<0.01
PACU opioid usage (MME), median (Q1–Q3)	15 (7.5–37.5)	36 (15–60)	<0.01
PACU average pain scores, median (Q1–Q3)	1.5 (1.1–2.3)	2.5 (1.8–3.2)	<0.01
PACU maximum pain scores, median (Q1–Q3)	5 (2–7)	7 (5–8)	<0.01
PACU PONV, n (%)	297 (3.0)	69 (4.5)	0.01*
Blood loss and transfusion outcomes			
Intraoperative blood loss (mL), median (Q1–Q3)	50 (25–100)	75 (45–100)	<0.01
Transfusion (intraoperative and postoperative), n (%)	13 (0.1)	9 (0.6)	<0.01†
Tranexamic acid administered, n (%)	9646 (96.4)	1492 (98.2)	<0.01*
Duration and admission outcomes			
Surgical duration (min), median (Q1–Q3)	76 (66–87)	86 (74–101)	<0.01
Length of PACU stay (min), Median (Q1–Q3)	188 (111–278)	136 (89–225)	<0.01
Admitted after surgery, n (%)	2336 (23.4)	502 (33.0)	<0.01*

*P value calculated using χ^2 test.
†P value calculated using Fisher exact test.
‡Pain scores reported by Numeric Rating Scale, scale from 0 to 10.
§All p values are calculated using Wilcoxon rank-sum test unless indicated otherwise.
MME, morphine milligram equivalents; PACU, postanesthesia care unit; PONV, postoperative nausea and vomiting; Q, quartile.



Knee surgery: Saphenous nerve and Incision

Using Lateral Parapatellar Incision with Normal Medial Parapatellar Arthrotomy may reduce IBSN injury[1]

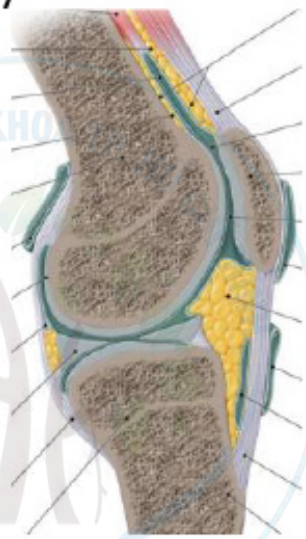


- Symptomatic IBSN injury has 55–100% longitudinal incision[2]
- Mean area of Medial Parapatellar vs. Midline incision is 28.9 cm^2 and 23.8 cm^2 respectively[3]

Surgical factors: vary from this surgeon to another one

1. Kenney RA, et al. *J Bone Joint Surg Am* 2013;95:2119-25; 2. Regev GJ, et al. *J Orthop Surg Res* 2014;9:464-5; 3. Sundaram RO, et al. *The Knee* 2007;14(5):375–378.

Less invasive surgery



Ye C, et al. Influence of the Infrapatellar Fat Pad Resection during Total Knee Arthroplasty: A Systematic Review and Meta-Analysis. 2016

Regional Blockade for Joint Replacement

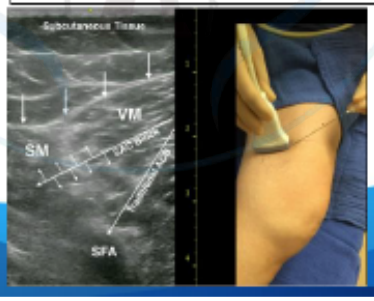
- ACB vs. FNB: equivalent effect, not affect QUADS → earlier ambulation and recovery.[1]
- ACB + iPACK improves anesthesia.[2]

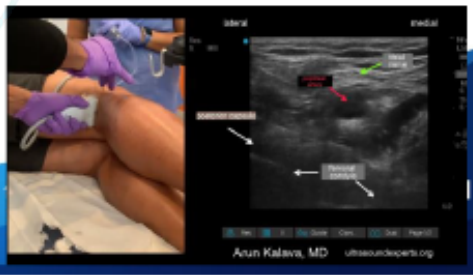
1. Karkhur Y, et al. *J Anaesthesiol Clin Pharmacol.* 2018;34(4):433-438; 2. Kandarian B, et al. *Korean J Anesthesiol* 2019;72(3):238-244; 3. Kim DH, et al. *Anesth Analg* 2019;129(2):526-535.

Table 2. Suggestions for Anesthesia and Analgesia to Optimize Outpatient Total Knee and Total Hip Arthroplasty

Medication or Technique	Comments
Total Knee Arthroplasty	
Adductor canal block (single shot or continuous)	Preserves quadriceps function better than femoral nerve block
iPACK block	Improves analgesia to posterior knee and overall; appears to not affect motor function
Local infiltration	Combination of adductor canal block, iPACK block and local infiltration may be better than individual techniques alone; total local anesthetic dose must be accounted for
Multimodal analgesia	Good evidence that each nonopioid component added improves analgesia after TJA; the "ideal" combination is not known
Mepivacaine spinal anesthesia	Evidence limited but may reduce time to ambulation and increase likelihood of same-day discharge; could decrease urinary retention vs. bupivacaine

iPACK, infiltration in the interspace between the popliteal artery and the capsule of the knee; **TJA,** total joint arthroplasty





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Richebé et al., Anesthesiology 2018

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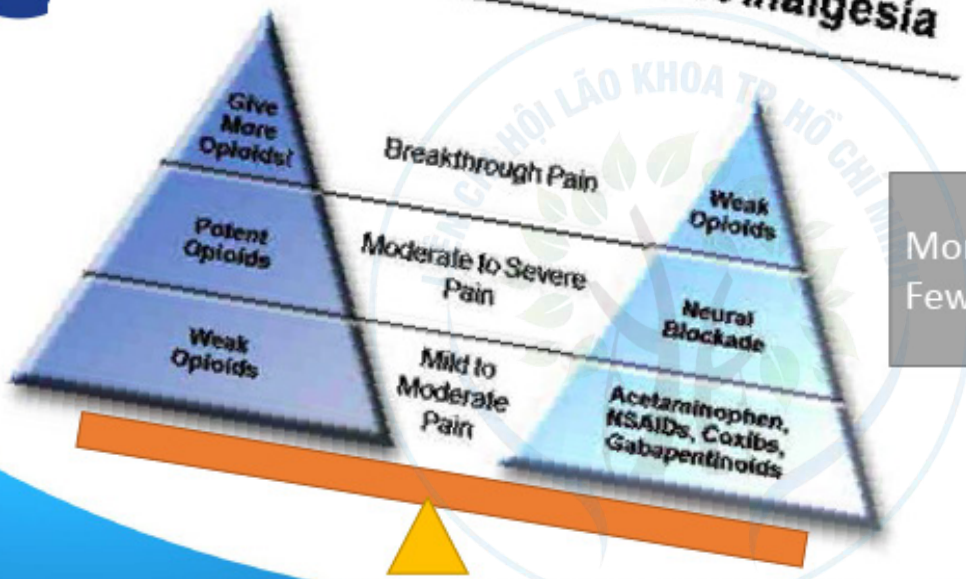
Opioids: Two-faced one

- Used to be primary modality for post-op. pain treatment
- Risk of opioid addiction, death.
- Not merely prevent central sensitization.
- Not effective at movement-evoked pain.
- Intra-operative development of tolerance may reduce the postoperative analgesic efficacy of opioids.
- Increasing rates of opioid-induced hyperalgesia and chronic post-op. pain.

Strassels SA,et al. Am J Health Syst Pharm.2005;62:2019–25.

Monotherapy vs Multimodal Analgesia



More effective
Fewer side effect

1. Gilron I, et al. *N Engl J Med* 2005;352:1324–34; 2. Moiniche S, et al. *Acta Anaesth Scand* 1997;41:785-9.

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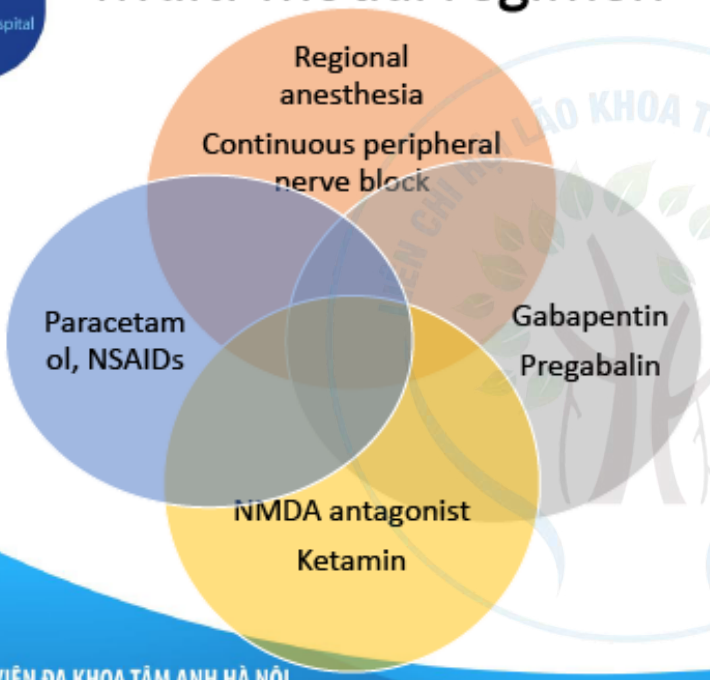
Multi-modal regimen



Purpose: To treat moderate-severe pain

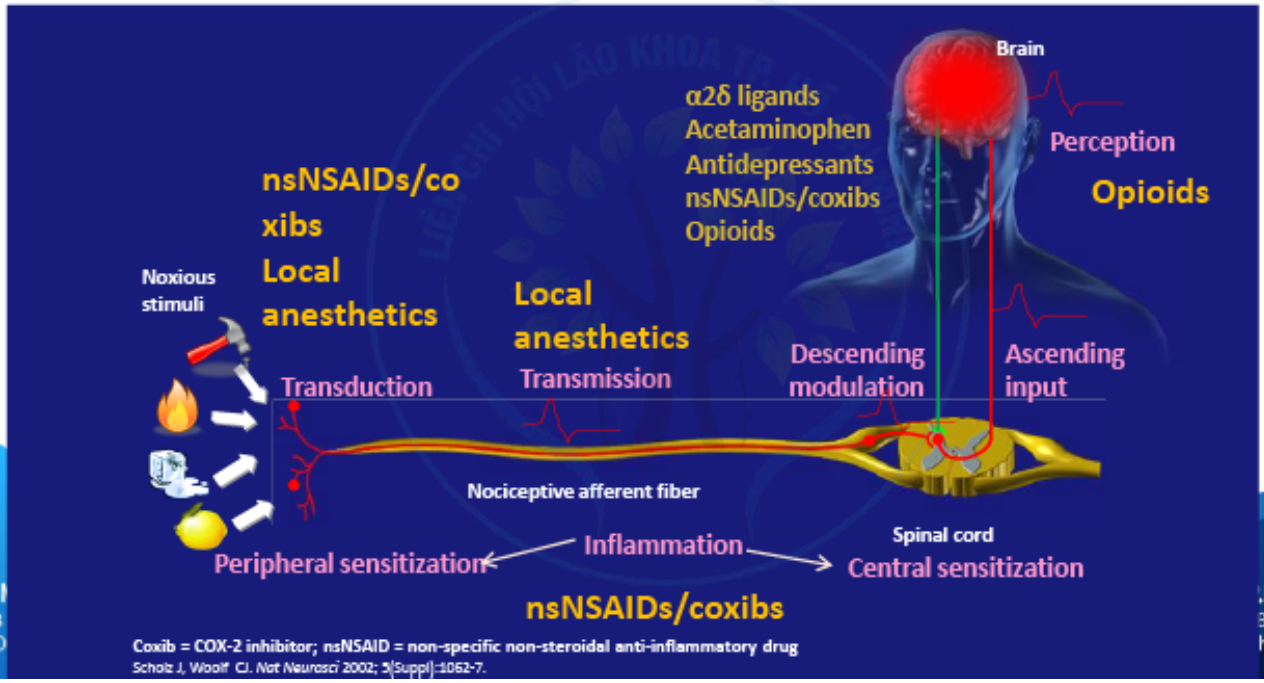
(1) provide superior pain relief at rest and with movement

(2) reduce opioid consumption

(3) reduce analgesic related adverse effects

Joshi G. *Anesthesiol Clin North Am* 2005;23(1):185–202

Multi-modal: More than two mechanisms for managing pain.



Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Schaible J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7.

Acetaminophen

Efficacy	Safety	Mechanism of Action
• Effective • Efficacy improved by addition of nsNSAIDs or coxibs	• Favorable safety profile and low cost • May cause liver damage at doses higher than 4 g/day	• Unclear

Acetaminophen is the first-line option in acute and chronic pain.

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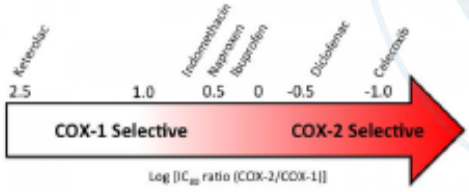
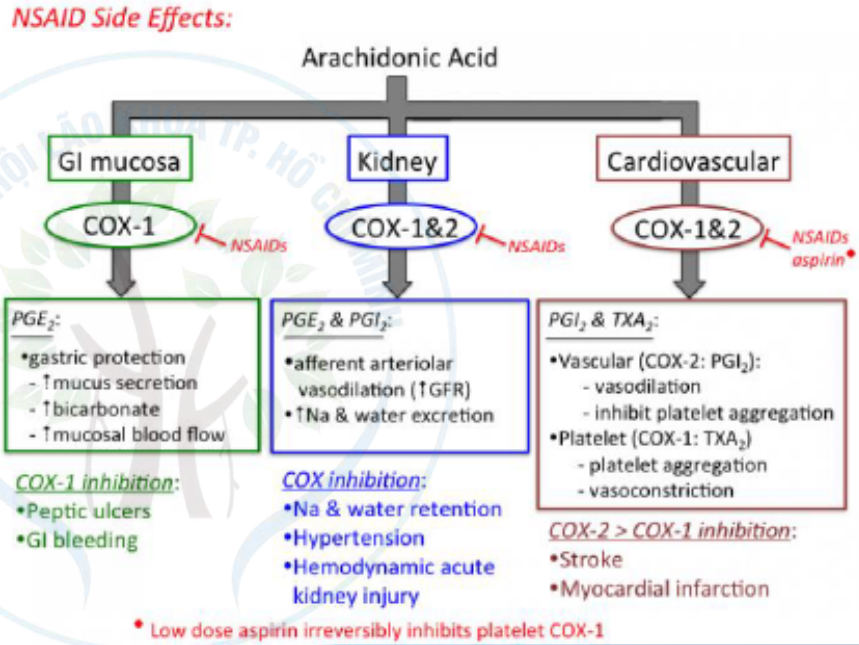
NSAIDS

- Play important part of multi-modal regimen
- Effective and resulted in improved patient outcomes including decreased opioid consumption





Memtsoudis SG, et al. *Anesthesiology* 2018;128(5):891–902.

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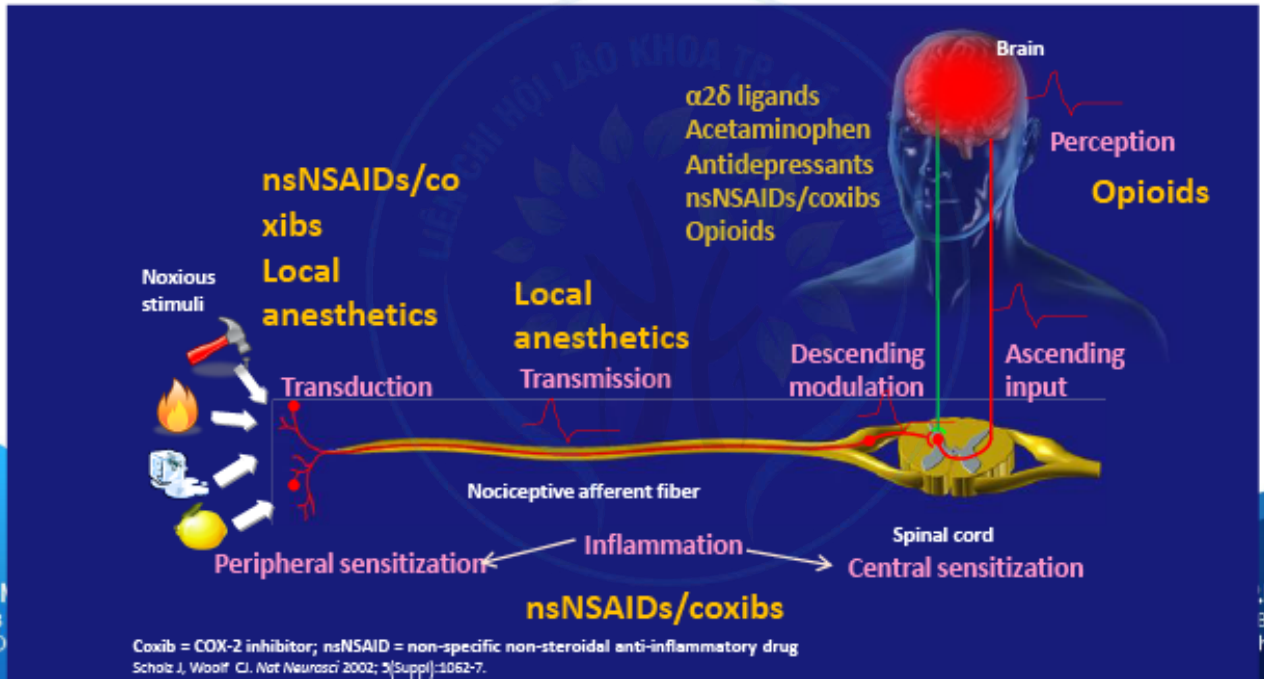
nsNSAIDs/Coxibs

Efficacy	Safety	Mechanism of Action
• Effective • More effective than acetaminophen alone • Improved efficacy in combination with acetaminophen	• Gastrointestinal risk • Cardiovascular risk • Renal risk	• Block action of COX-2 enzyme, which is induced by inflammatory stimuli and results in increased production of prostaglandins • Coxibs specifically inhibit COX-2, while nsNSAIDs block action of COX-2 and COX-1 enzyme, which is involved in gastrointestinal cytoprotection and platelet activity

First-line option in acute and chronic pain

Multi-modal: More than two mechanisms for managing pain.



Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug
Scholz J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7.

α2δ Ligands

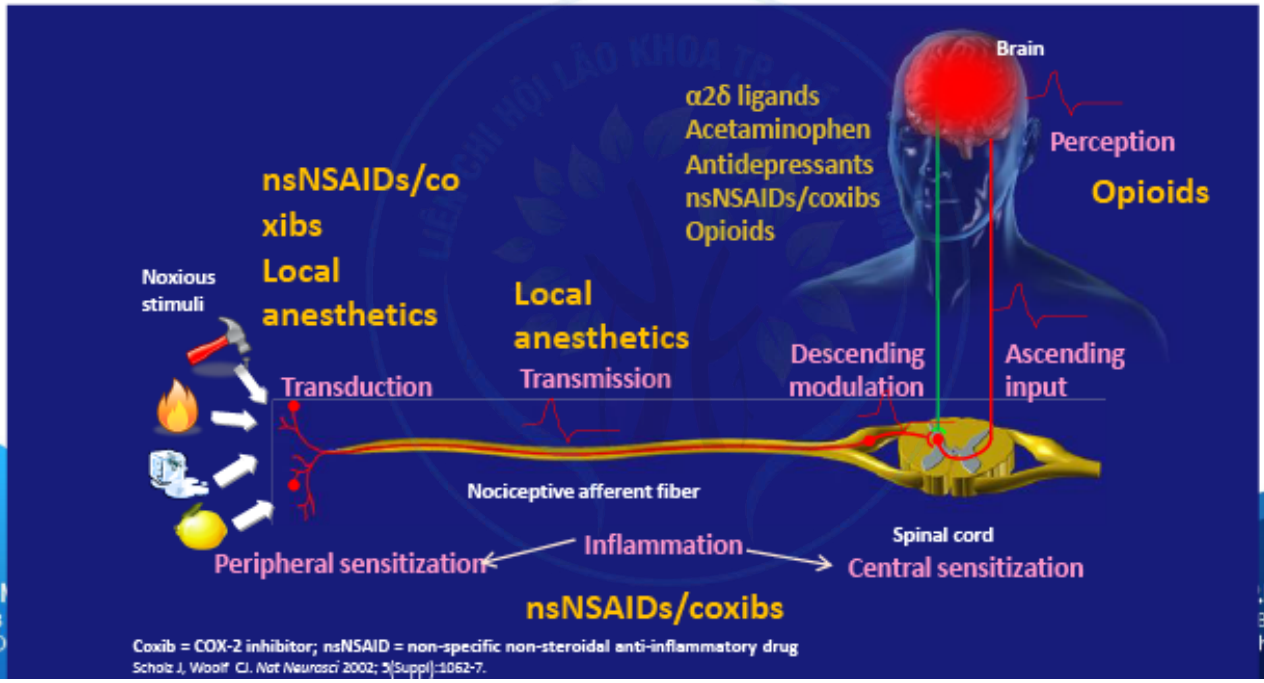
Useful in combination with other treatments with a neuropathic component

Efficacy	Safety	Mechanism of Action
• Pregabalin + coxib combination is more effective than each drug used alone	• Most common side effects are dizziness and somnolence	• Bind to $\alpha_2\delta$ subunit of calcium channel, which is upregulated in neuropathic pain • Binding reduces neurotransmitter release and pain sensitization

*Gabapentin and pregabalin are $\alpha_2\delta$ ligands

Attal N, Finnerup NB. *Pain Clinical Updates* 2010; 18(9):1-8; Bauer CS *et al. J Neurosci* 2009; 29(13):4076-88; Chou R *et al. Ann Intern Med* 2007; 147(7):505-14; Lee J *et al. Br J Anaesth* 2013; 111(1):112-20; Romanò C *et al. J Orthop Traumatol* 2009; 10(4):185

Multi-modal: More than two mechanisms for managing pain.



Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Scholz J, Woolf CJ. *Nat Neurosci* 2002; 5(Suppl):1062-7.

Antidepressants

Useful in combination with other treatments for pain with a neuropathic component

Efficacy	Safety	Mechanism of Action
• Not recommended for non-specific acute pain • May be considered for chronic pain with a neuropathic component	• TCAs can cause cognitive disorders, confusion, gait disturbance and falls • SNRIs are contraindicated in severe hepatic dysfunction or unstable arterial hypertension	• Inhibit reuptake of serotonin and norepinephrine, enhancing descending modulation

TCA = tricyclic antidepressant; SNRI = serotonin norepinephrine reuptake inhibitor

Attal N, Finnerup NB. *Pain Clinical Updates* 2010; 18(9):1-8; Lee J *et al. Br J Anaesth* 2013; 111(1):112-2; Skljarevski V *et al. Eur J Neurol* 2009; 16(9):1041-8; Verdu B *et al. Drugs* 2008; 68(18):2611-32.

	Drug class	Examples
FIRST LINE	Tricyclic Antidepressants	Nortriptyline, desipramine
	Anticonvulsants	Gabapentin, pregabalin (carbamazepine: tic douloreux)
	SNRI	Duloxetine, venlafaxine
SECOND LINE	Tramadol Opioids	
THIRD LINE	Cannabinoids	Sativex buccal spray, dronabinol
FOURTH LINE	SSRI	
	Topical lidocaine	
	Methadone	
	Other anticonvulsants	Lamotrigine, topiramate, valproic acid

Moulin DE, et al. Pharmacological management of chronic neuropathic pain: Revised consensus statement from the Canadian Pain Society. Pain Res Manage 19(6) Nov/Dec 2014

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KJA
Korean Journal of Anesthesiology

Review Article
pISSN 2005-6419 · eISSN 2005-7563

Designing the ideal perioperative pain management plan starts with multimodal analgesia

Eric S. Schwenk[1] and Edward R. Mariano[2,3]

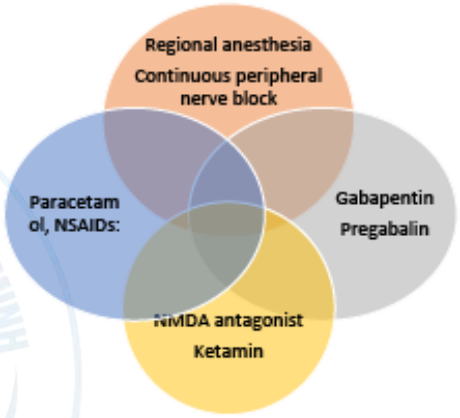


Table 1. Commonly Used Perioperative Systemic Non-opioid Multimodal Analgesics in Adults

Drug	Route (s)	Preoperative dose	Intraoperative dose	Postoperative dose
Acetaminophen	IV/PO	1000 mg (> 50 kg)	1000 mg	1000 mg q6h
Celecoxib	PO	400 mg	N/A	200 mg q12h
Gabapentin	PO	300–1200 mg	N/A	300–800 mg q8h
Ketamine	IV	N/A	0.25–0.5 mg/kg bolus	0.25 mg/kg/h infusion
Ibuprofen	IV/PO	600–800 mg	N/A	600 g q6h
Pregabalin	PO	75–150 mg	N/A	75 mg q12h

IV: intravenous, N/A: not applicable, PO: by mouth.

Schwenk EC and Mariano ER. *Korean J Anesthesiol. 2018;71(5):345–352.*

- Psychological consultation
- Pre-operative regimens: (2 hour prior surgery) Pregabalin 75mg BD PO, Celecoxib 400mg , Paracetamol 1g

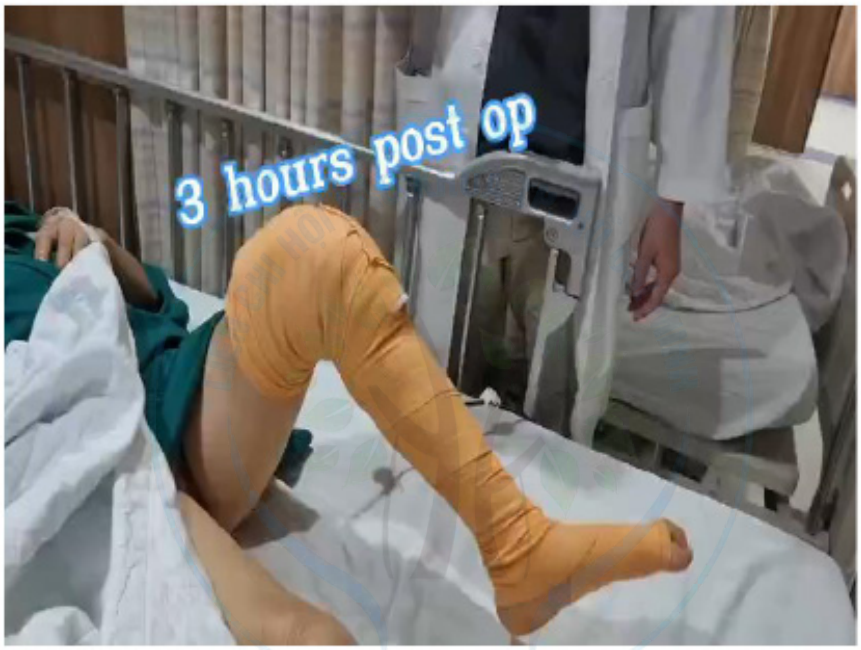
Surgery

- Spinal anesthesia,
- No tourniquet, mini-invasive
- Abductor canal bock + iPack

- Pregabalin 75mg BD PO, Celecoxib 200mg BD PO, Paracetamol 1g/100mL TD IV, Neopam 20mg BD IV, Tramadol 0.1g BD IM
- Physical therapy asap
- Cryptotherapy
- (Amitryptiline 25mg)

Pregabalin 75mg x2, Celecoxib 200mg x2 Paracetamol 0.5g x 3, Tramadol 50mg x 2 if needed

(2 – 6 weeks)



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Take home messages

- Chronic post-surgical pain is problematic to 15% orthopedic surgery.
- Pain control before surgery is important – do not let the patient suffer worse pain experience
- Multi-modal is the state of the art which is balancing risk and benefit. .