Vitalis

Ropivacaina, ayer y hoy.

DR. JUAN SALVADOR VILCHIS RENTERIA

Si de todos los medicamentos con los que contamos para dar una anestesia solo pudiera elegir 4, independientemente del tipo de cirugía, ¿cuáles elegiría?

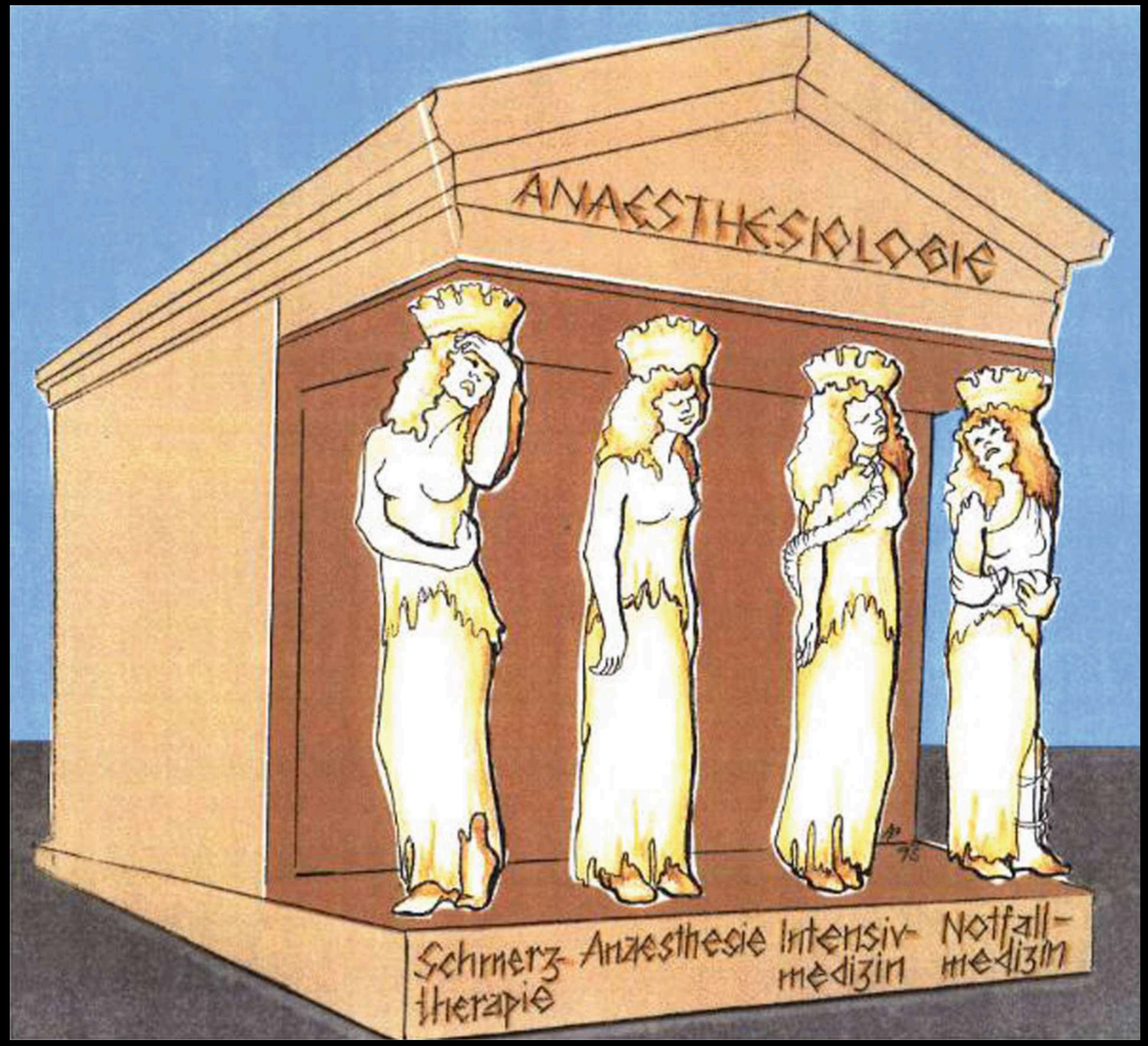
Vitalis

1. Propofol
2. Ropivacaina
3. Opioides (fentanil, remifentanil, sufentanil)
4. Bloqueadores neuromusculares (rocuronio, cisatracurio)
5. Dexmedetomidina
6. Lidocaina
7. Sulfato de Magnesio
8. Ketamina

De la narcosis a la homeostasis perioperatoria

Geschichte der Anästhesie
“Vom Narkotiseur zum perioperativen Homöostatiker“ Dr. Phil. H. Petermann, M. A.
Anaesthesist. DOI 10.1007/s00101-016-0223-y

Columnas de
La Anestesiología



Insensibilidad parte específica

SCIENCE

FRIDAY, MAY 21, 1920

CONTENTS

LOCAL ANESTHETICS[1]

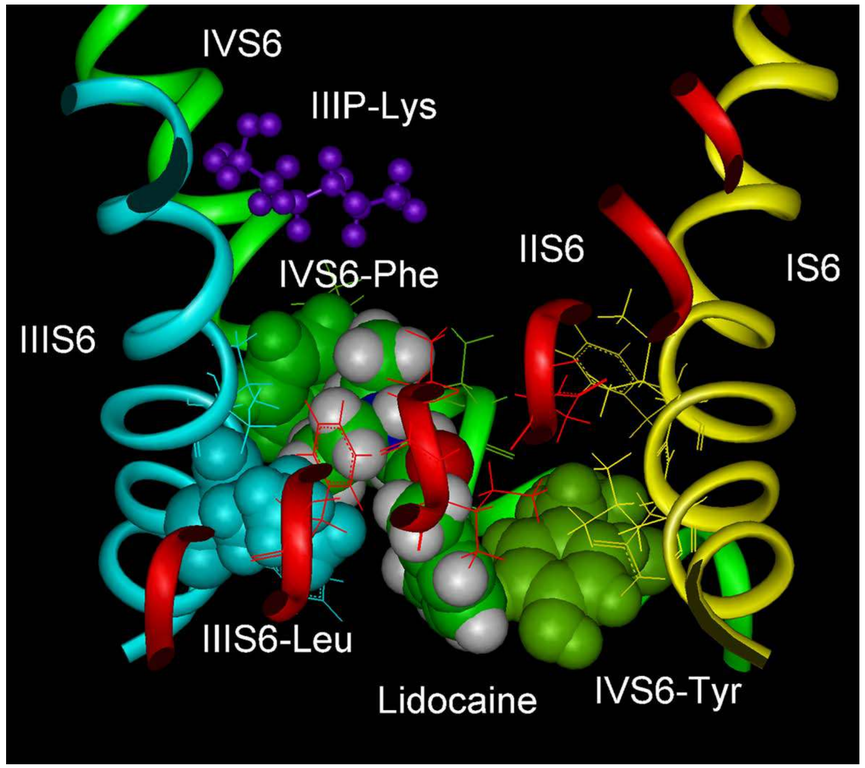
SINCE earliest times, those who have resorted to surgery for the relief of their fellow creatures, have desired to mitigate their procedures by the exclusion of pain. Generally

Exactly what happens to the nerve tissues when brought into contact with a local anesthetic drug has not been determined.

MECANISMO DE ACCION ANESTESICOS LOCALES

ANESTESICOS LOCALES

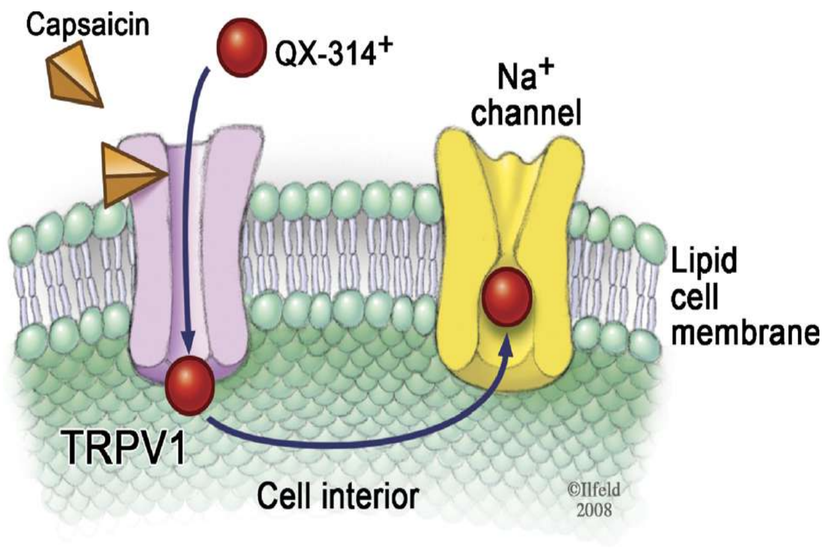
- Bloqueo de impulsos nerviosos para abolir las sensaciones.
- Amina terciaria unida a un anillo aromático por una cadena intermedia de éster o amida.



Briann M Ilfeld MD. The End of Postoperative Pain. A Fast Approaching Possibility? And, if So, Will We Be Ready? Regional Anesthesia and Pain Medicine & Volume 34, Number 2, March-April 2009 .

MECANISMO DE ACCION

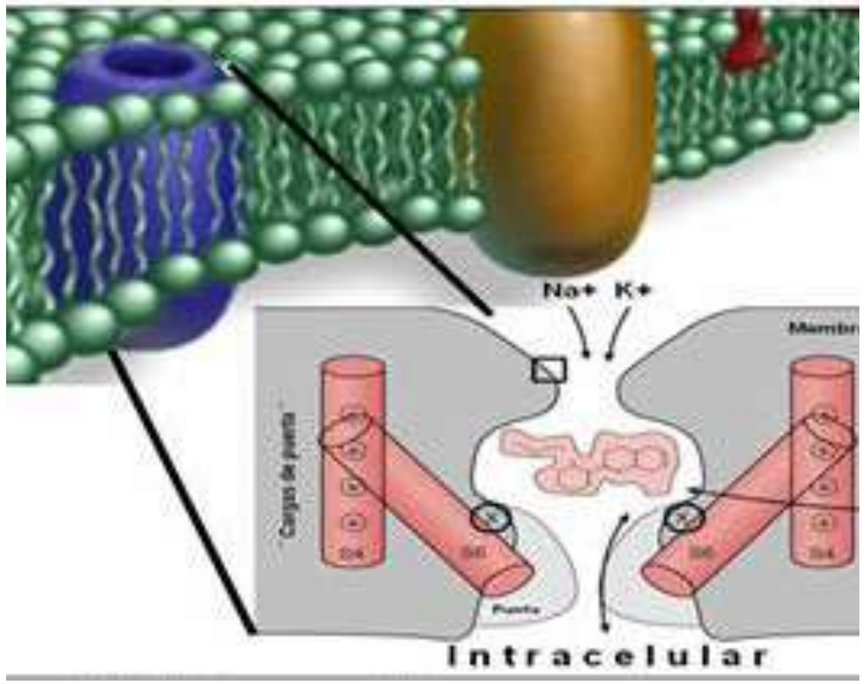
- Bloqueo de los canales de sodio.
- Proteínas integrales de la membrana que inician y propagan potenciales de acción.



Briann M Ilfeld MD. The End of Postoperative Pain. A Fast Approaching Possibility? And, if So, Will We Be Ready? Regional Anesthesia and Pain Medicine & Volume 34, Number 2, March-April 2009 .

ESTRUCTURA DE LA MEMBRANA

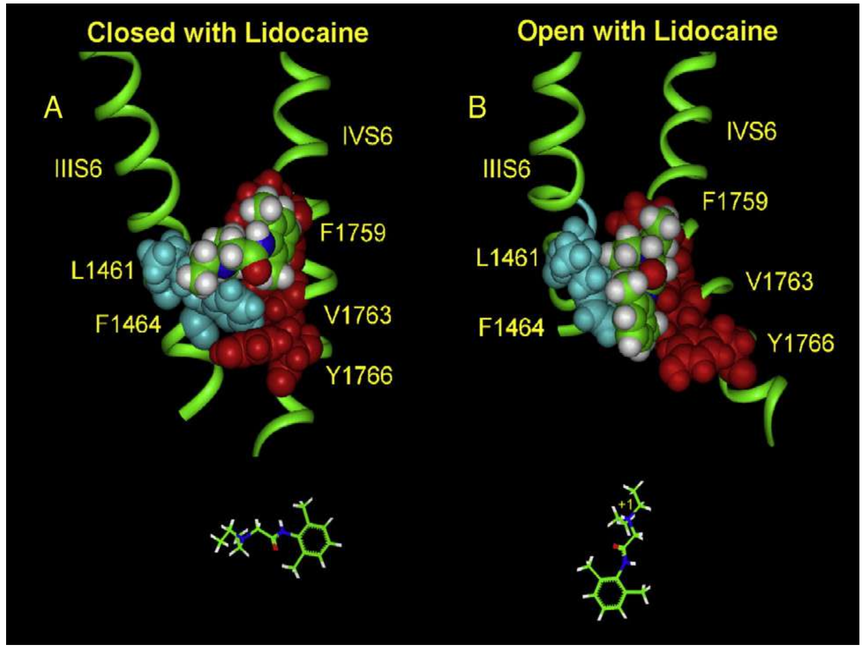
- El canal de sodio es una membrana intrínseca glicoproteica.
- Complejo molecular de 2,000 subunidades alfa de aminoácidos.



Patino, G. A., and Isom, L. L. (2010). Electrophysiology and beyond: multiple roles of Na channel β sub- units in development and disease. *Neurosci. Lett.* 486, 53–59.

SITIO DE APERTURA

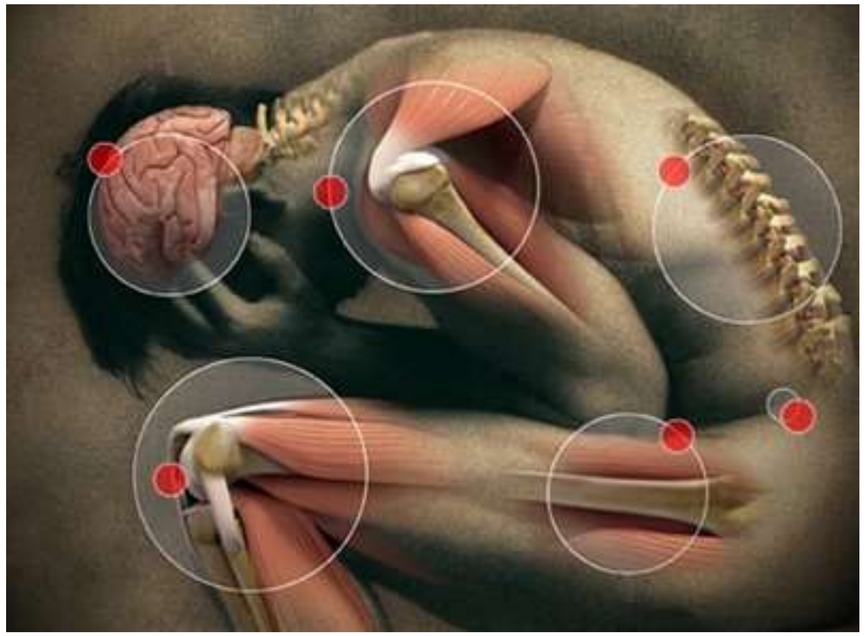
- Vinculación a medicamentos y toxinas en el poro.
- Abre y cierra en respuesta al potencial de membrana.
- Bloqueo de alta afinidad, interacción del AL con el poro.



Sheets, M. F., Fozzard, H. A., Lipkind, G. M., and Hanck, D. A. (2010). Sodium channel molecular conformations and antiarrhythmic drug affinity. *Trends Cardiovasc. Med.* 20, 16–21.

ESPECIFICIDAD DOLOR

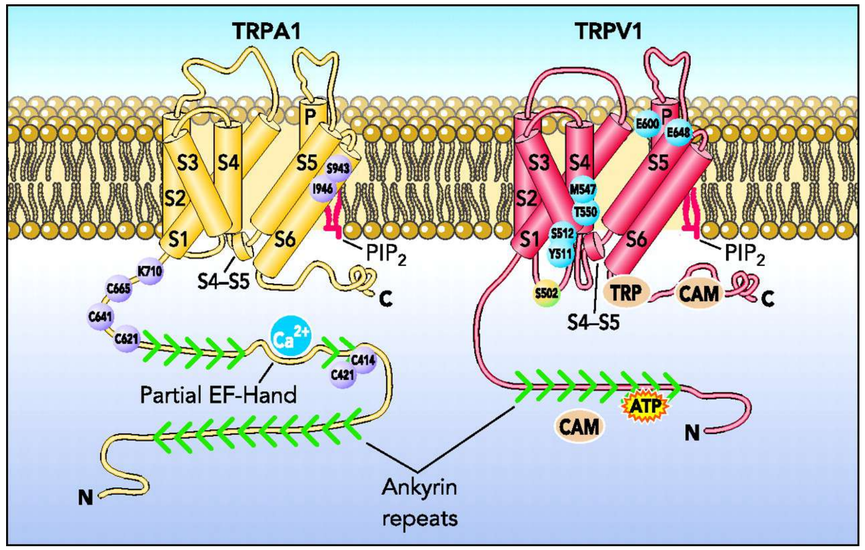
El dolor se transmite vía células nerviosas que dependen de isoformas específicas de canales de sodio para excitación y conducción.



Harry A.Fozzard, Michael F. Sheets , Dorothy A.Hanck. The sodium channel as a target for local anesthetic drugs. Review article. Frontiers in pharmacology. Nov. 2011 (2); 68: 1-6.

BIOLOGIA DEL DOLOR

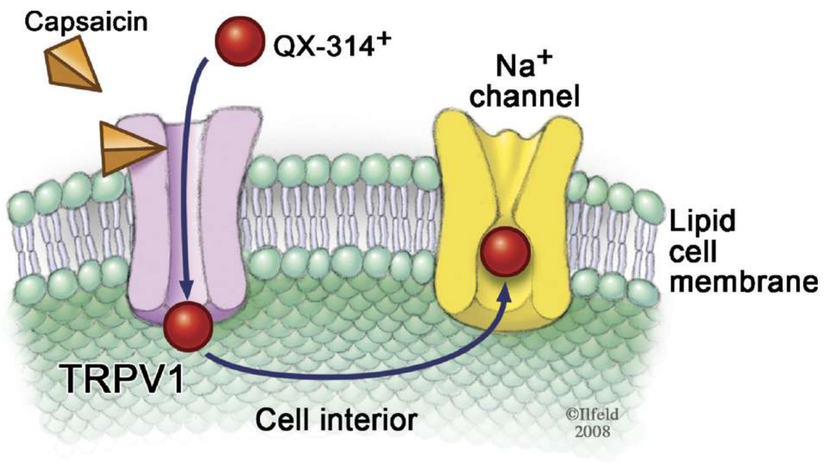
Selección de axones que permitan una conducción especifica de bloqueo en subpoblaciones de nervios.



Binshtok AM,Bean BP,Woolf CJ. Inhibition of nociceptors by TRPV1-mediated entry of impermeant sodium channel blockers. Nature. 2007;449:607-610.

ESPECIFICIDAD

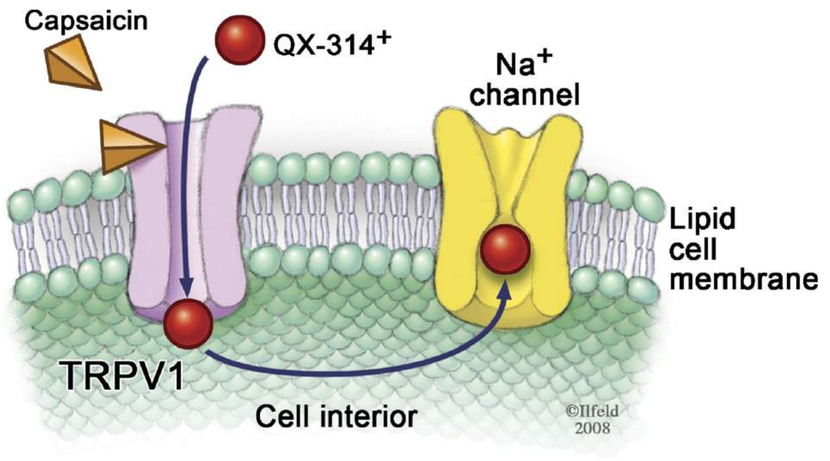
- 9 canales estructurales distintos Na_V 1.1 – 1.9.
- El bloqueo del canal de sodio subtipo Na_v 1.8 resulta en analgesia en modelos preclínicos.



Binshtok AM,Bean BP,Woolf CJ. Inhibition of nociceptors by TRPV1-mediated entry of impermeant sodium channel blockers. Nature. 2007;449:607-610.

Transient Receptor Potential Vanilloid

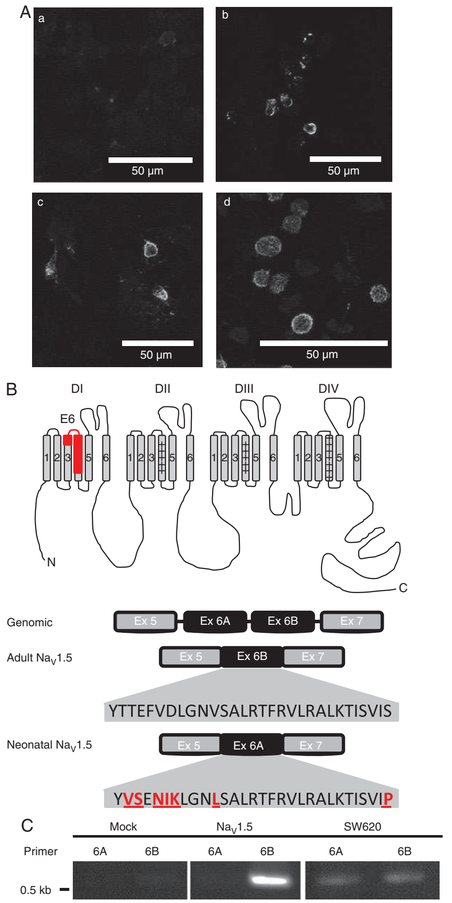
- Capsaicina abre el poro transmembrana.
- TRPV1 produce bloqueo potente de fibras.
- Con un análogo cuaternario de la lidocaína QX-314.



Binshtok AM,Bean BP,Woolf CJ. Inhibition of nociceptors by TRPV1-mediated entry of impermeant sodium channel blockers. Nature. 2007;449:607-610.

FUNCION ANTICANCERIGENA

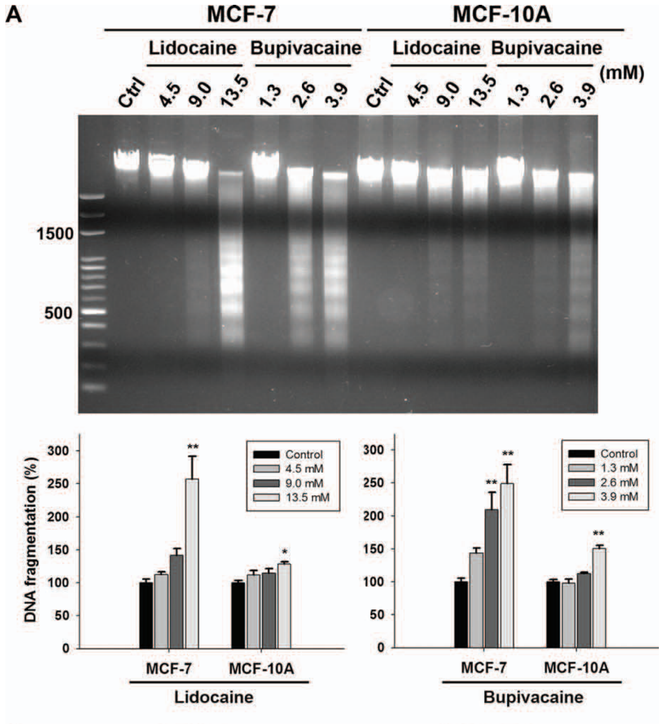
- Células cancerígenas de colon expresan voltajes de los canales de sodio que media su invasión.
- La ropivacaina inhibe este voltaje y por lo tanto la invasión.



D. T. Baptista-Hon. Potent inhibition by ropivacaine of metastatic colon cancer SW620 cell invasion and NaV1.5 channel function . British Journal of Anaesthesia 113 (S1): i39–i48 (2014)

FUNCION ANTICANCERIGENA

- Lidocaína y bupivacaina inhiben el crecimiento de líneas celulares de cáncer de mama MCF-7.
- 74% y 81% de las células manifestaron apoptosis respectivamente.



Yuan-Ching Chang

Local Anesthetics Induce Apoptosis in Human Breast Tumor Cells

Anesth Analg 2014;118:116–24

FUNCION ANTICANCERIGENA

- La apoptosis está controlada por proteasas de cisteina intracelular: caspasas.
- PARP enzima involucrada en el daño y reparación del DNA esta ligada a caspasa 3 y 7 durante la apoptosis.
- Lidocaina y bupivacaina inducen activación proteolítica de caspasa 7.

Yuan-Ching Chang
Local Anesthetics Induce Apoptosis in Human Breast Tumor Cells
Anesth Analg 2014;118:116–24

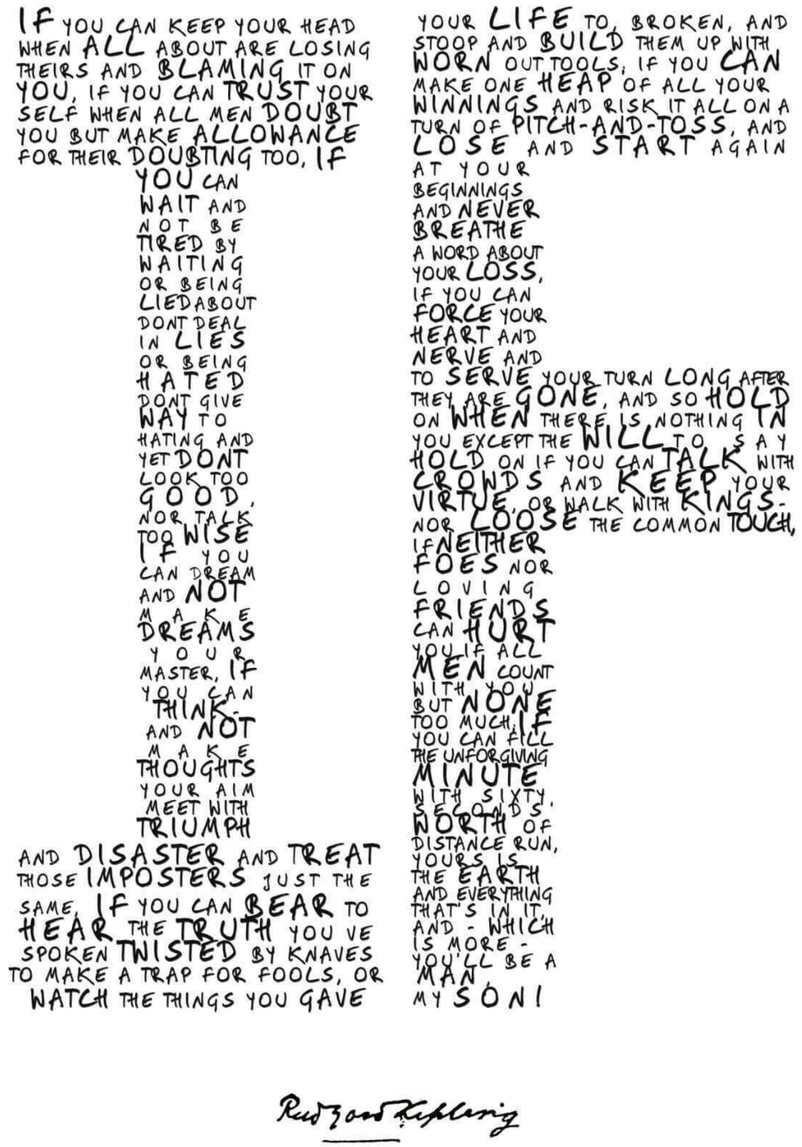
ROL CLINICO ACTUAL DE LOS ANESTESICOS LOCALES

- Principal uso bloqueo nervioso local o regional.
- Especificidad con altas concentraciones en el sitio de inyección minimizando efectos adversos.
- Características deseables rápido inicio del bloqueo nervioso, suficiente duración y disminución de reacciones.

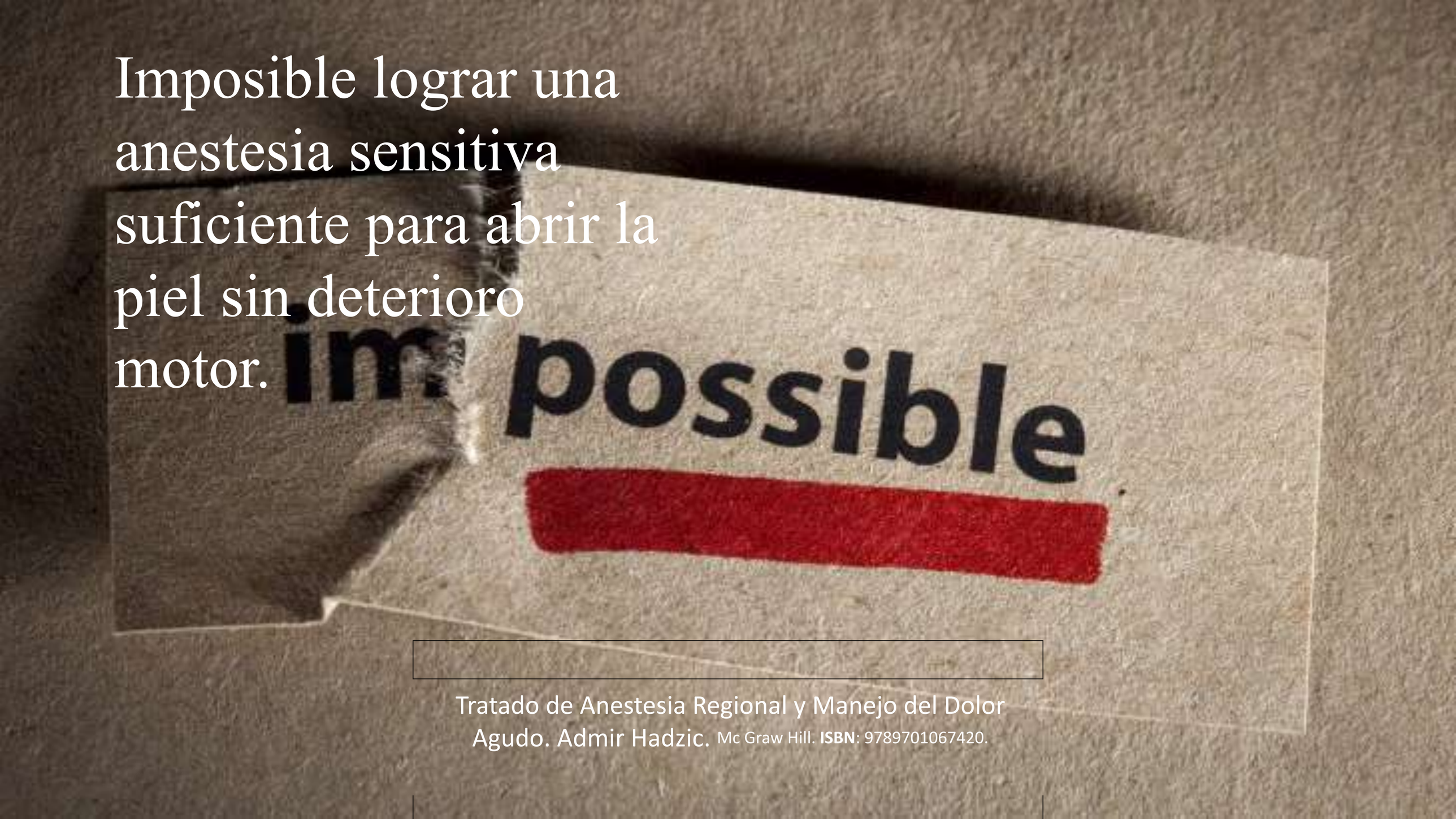
Harry A.Fozzard, Michael F. Sheets , Dorothy A.Hanck. The sodium channel as a target for local anesthetic drugs. Review article. Frontiers in pharmacology. Nov. 2011 (2); 68: 1-6.

¿ANESTESICO LOCAL IDEAL?

La anestesia regional y el manejo del dolor cambiarían con un AL que inhibiera selectivamente la transmisión del dolor y mantuviera intactas otras funciones.



Imposible lograr una anestesia sensitiva suficiente para abrir la piel sin deterioro motor.



Tratado de Anestesia Regional y Manejo del Dolor Agudo. Admir Hadzic. Mc Graw Hill. **ISBN**: 9789701067420.

La potencia del bloqueo nervioso de los AL se incrementa con el peso molecular y la liposolubilidad creciente.

La eficacia de un AL depende de la dosis, el sitio de administración, los aditivos, la temperatura y el embarazo (aumenta susceptibilidad neural).

Tratado de Anestesia Regional y Manejo del Dolor Agudo. Admir hadzic. Mc Graw Hill. **ISBN**: 9789701067420.

British Journal of Anaesthesia **87** (**1**): 27–35 (2001)

Developments in local anaesthetic drugs

J. B. Whiteside* and J. A. W. Wildsmith

University Department of Anaesthesia, Ninewells Hospital and Medical School, Dundee DD1 9SY, UK

**Corresponding author*

Thus, ropivacaine has other potential advantages besides that of reduced cardiotoxicity.

Further evaluation in both animal and volunteer human studies confirmed that ropivacaine is an effective local anaesthetic and showed that, unlike bupivacaine, it has a slight vasoconstrictor effect at lower concentrations.[28 36 66] Epinephrine was found to have little effect on the local action or the resultant systemic concentrations of ropivacaine in human studies.[28 56 81]

British Journal of Anaesthesia **87** (**1**): 27–35 (2001)

Developments in local anaesthetic drugs

J. B. Whiteside* and J. A. W. Wildsmith

University Department of Anaesthesia, Ninewells Hospital and Medical School, Dundee DD1 9SY, UK

**Corresponding author*

Clinical efficacy

Ropivacaine has been compared with bupivacaine in many clinical trials involving most forms of regional anaesthesia. Most studies have shown that the onset, potency and duration are very similar to those of bupivacaine. However, some studies, particularly those utilizing the concept of Minimum Local Analgesic Concentration (MLAC) in epidural analgesia, have questioned whether the difference in cardiotoxicity seen between the two agents is in fact a result of an absolute difference in potency.[27 84] The suggestion is that the therapeutic ratio of the two may be the same. Such concerns must be viewed against the important basic principle that the local, and subsequent systemic, dynamics of a particular local anaesthetic will depend on the site of injection.[8] Thus, each clinical application must be considered in turn.

The Cardiotoxicity of Local Anesthetics: The Place of Ropivacaine

Bernhard M. Graf*

Department of Anaesthesiology, University of Heidelberg, Im Neuenheimer Feld 110, D-69120 Heidelberg, Germany

Abstract: Central and regional block procedures have a well-defined role as safe and effective methods in modern anesthesia and analgesia with long-acting local anesthetics. Recent studies have shown that the incidence of intoxication by these drugs is a rare but catastrophic event. As classic neuronal sodium channel inhibitors, local anesthetics block peripheral fast voltage-gated sodium channels on neuronal axons, and these drugs have a particularly high level of activity in the CNS and the cardiovascular system. CNS-toxicity follows a two-stage process, whereby at lower concentrations inhibitory neurons are blocked first resulting in generalized convulsions, and at higher concentrations a global CNS depression can be seen. Although seizures are an impressive clinical syndrome, they can often be treated safely without permanent damage. More important is the cardiotoxicity of these drugs, which can be divided into indirect cerebrally mediated and a direct myocardial component. Like CNS-toxicity in general, indirect cardiotoxicity demonstrates an initial stimulating effect, followed by a depressive component at higher concentrations. Direct myocardial actions are comprised of negative chronotropic, dromotropic and inotropic effects. For dromotropy, stereoselectivity was found. The *S*-(-)-isomers of the long-acting local anesthetics were less delayed compared to racemic mixtures and the *R*-(+)-enantiomers. For inotropy, no stereospecific depression of this parameter was noted between isomers of ropivacaine or bupivacaine, but bupivacaine produced a significantly greater depression of LV pressure than ropivacaine, mepivacaine, or lidocaine. Pharmacokinetic differences in lipophilicity of local anesthetics correlate well with the depression of mitochondrial ATP-synthesis in fast metabolizing cells. Intracellular ATP-level may be involved in contractility

Current Topics in Medicinal Chemistry 2001, *1*, 207-214

The Cardiotoxicity of Local Anesthetics: The Place of Ropivacaine

Bernhard M. Graf*

Department of Anaesthesiology, University of Heidelberg, Im Neuenheimer Feld 110, D-69120 Heidelberg, Germany

CONCLUSION

Although a recent study [46] showed that incidence of cerebral intoxication and cardiovascular collapse caused by local anesthetic injection is low (1 in 40,010 epidural blocks), case reports of artificial intravasal injection or

anesthetics. Ropivacaine is presently the safest long-acting local anesthetic clinically used, since it shows a higher tolerated dose and unbound plasma concentration for CNS symptoms than bupivacaine and etidocaine. At ropivacaine doses producing CNS symptoms, cardiovascular changes, such as depression of conduction and diastolic and systolic function, were less pronounced when compared with bupivacaine.

Anaesthesist 2016 · 65:353–358 DOI 10.1007/s00101-016-0151-x ©
Springer-Verlag Berlin Heidelberg 2016
M.Chen·C.Chen·W.Fang

Determination of the median effective dose (ED50) of spinal plain ropivacaine for motor block in adults

Conclusion. the ED50 for motor block in spinal plain ropivacaine decreases with advancing age, indicating that age has an influence on the potency of spinal ropivacaine.

16.49–19.44 mg) in 51–60, 16.11 mg (95 % CI: 14.50–17.90 mg) in 61–70, and 15.75 mg (95 % CI: 13.98–17.73 mg) in 71–80 year-old patients. Maximum cephalic analgesic effects

ROPIVACAINE

Amino Amide group drug

- Ropivacaine injection is preservative-free and is available in single dose containers in 2 (0.2%), 5 (0.5%), 7.5 (0.75%) and 10 mg/mL (1%) concentrations
- Never exceed > 770 mg ropivacaine in 24 hrs. for postoperative management
- Solutions should be stored at 20° to 25°C
- Epidural administration of ropivacaine in some cases increases in temperature to > 38.5°C
- Allergic type reactions are rare with ropivacaine
- Avoid ropivacaine in patients treated with class III antiarrhythmic drugs because of additive cardiac effect

- Use of ropivacaine in the management of chronic pain is new advances
- Use of Ropivacaine in ophthalmic surgery is not recommended
- Dose is 2-3 mg/kg

Invented in 1980

- With ropivacaine clinically, the order of loss of nerve function is as follows: (1) pain, (2) temperature, (3) touch, (4) proprioception, and (5) skeletal muscle tone
- Addition of epinephrine to ropivacaine has no effect on limiting systemic absorption of ropivacaine
- In peripheral nerve block minimum duration of anesthesia is 4 hours and maximum is 9 hours
- In local infiltration the duration of effect varies from 2 to 6 hours
- Geriatric, & ASA III given reduced dose of ropivacaine

Pharmacokinetic

- Bioavailability : 87%–98% (epidural)
- Shelf life 36 hrs.
- Metabolism : Liver CYP1A2-
- Onset of action : 15 minutes
- Elimination half-life: 1.6–6 h
- Excretion : Kidney 86%
- Formula : $C_{17}H_{26}N_2O$
- Molar mass: 274.408 g·mol^{-1}
- Routes of administration: Parenteral

Adverse effects

Central Nervous System
Nervousness
Tingling around the mouth
Tinnitus
Tremor
Dizziness
Blurred vision
Seizures
Respiratory depression
Apnea

Cardiovascular Effects
Hypotension (37%)
Bradycardia (9%)
Arrhythmias
Cardiac arrest

Others
Nausea (25%)
Vomiting (12%)
Headache

These effects are more in geriatric patients
These effects are very low in paediatric patients

Dosage

- Lumbar epidural for Surgical anaesthesia and Caesarean section 0.75% 15-20 mL 113-150 mg
- Other surgery 1% 15-20 mL 150-200 mg
- Intrathecal administration 0.5% 3-4 mL 15-20 mg
- Peripheral nerve block 0.75% 10-40 mL 75-300 mg
- Local Infiltration 0.2% 20-25 ml 40-50 mg
- Postoperative pain (Continuous infusion)

-Lumbar epidural 0.2% 6-10 mL/h 12-20 mg/h
-Peripheral nerve block 0.2% 5-10 mL/h 10-20 mg/h
-Intra-articular injection 0.75% 20 mL 150 mg

- Labour pain (Lumbar epidural)

Bolus 0.2% 10-20 mL 20-40 mg
Intermittent top-ups 0.2% 10-15 mL 20-30 mg
Continuous infusion 0.2% 6-14 mL/h 12-28 mg/h

- It inhibit platelet aggregation in plasma
- Ropivacaine has antibacterial activity *in vitro*
- Ropivacaine is toxic to cartilage and their intra-articular infusions can lead to Postarthroscopic Glenohumeral Chondrolysis

Mechanism of Action

- Via reversible inhibition of sodium ion influx in nerve fibers, thereby blocks impulse conduction in nerve fibres
- Less lipophilic than bupivacaine and is less likely to penetrate large myelinated motor fibres, resulting in a relatively reduced motor blockade
- Has a greater degree of motor sensory differentiation, which could be useful when motor blockade is undesirable

Rapid recovery

Less motor block

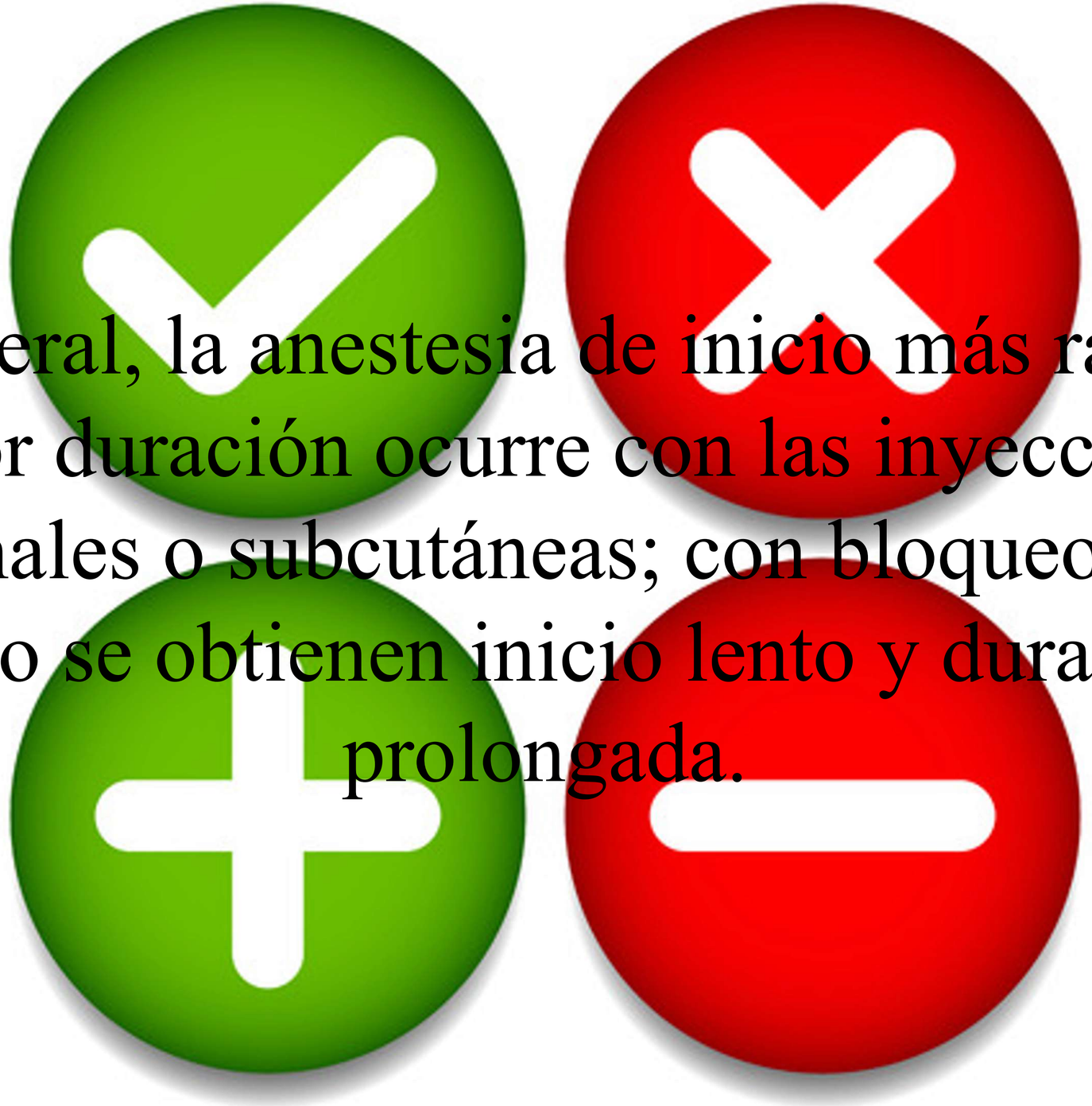
- Ropivacaine has less CNS & cardiotoxicity than Bupivacaine in high dose
- Treatment of overdose Is intravenous lipid emulsion
- Contraindicated for IV regional anaesthesia (IVRA)

Extra Shots

-Crosses the placenta during epidural administration for caesarean section but total plasma concentration of ropivacaine was lower in the foetal circulation than in the maternal circulation
- Toxicity as a result of inadvertent intravascular injection of ropivacaine is low (0.2 %)
-Caution in mixing any amide local ane. drugs with ropivacaine to avoid additive toxic effects

Long acting

Avoid in AV block



En general, la anestesia de inicio más rápido y menor duración ocurre con las inyecciones espinales o subcutáneas; con bloqueos del plexo se obtienen inicio lento y duración prolongada.

“Aun bajo AG se debe proteger con anestesia regional ya que pueden incurrir cambios persistentes en el SNC y favorecer el dolor postoperatorio”.

Crile G. Phylogenetic association in relation to certain medical problems. Boston Med J 1910;163:893-904.

General Articles

Section Editor: **Thomas R. Vetter**

SPECIAL ARTICLE

OPEN

CME

Multimodal General Anesthesia: Theory and Practice

Emery N. Brown, MD, PhD,*†‡§‖ Kara J. Pavone, BS, BSN, RN,* and Marusa Naranjo, MD¶

Because there are multiple different neurotransmitters and neural relays in the ascending and descending pathways, there are multiple targets at which antinociceptive agents can act to disrupt nociceptive information processing. Targeting simultaneously multiple targets in the nociceptive system is the key concept underlying the design of a multimodal strategy for nociceptive control, and hence, multimodal general anesthesia. We focus our discussion of antinociceptive agents on opi-

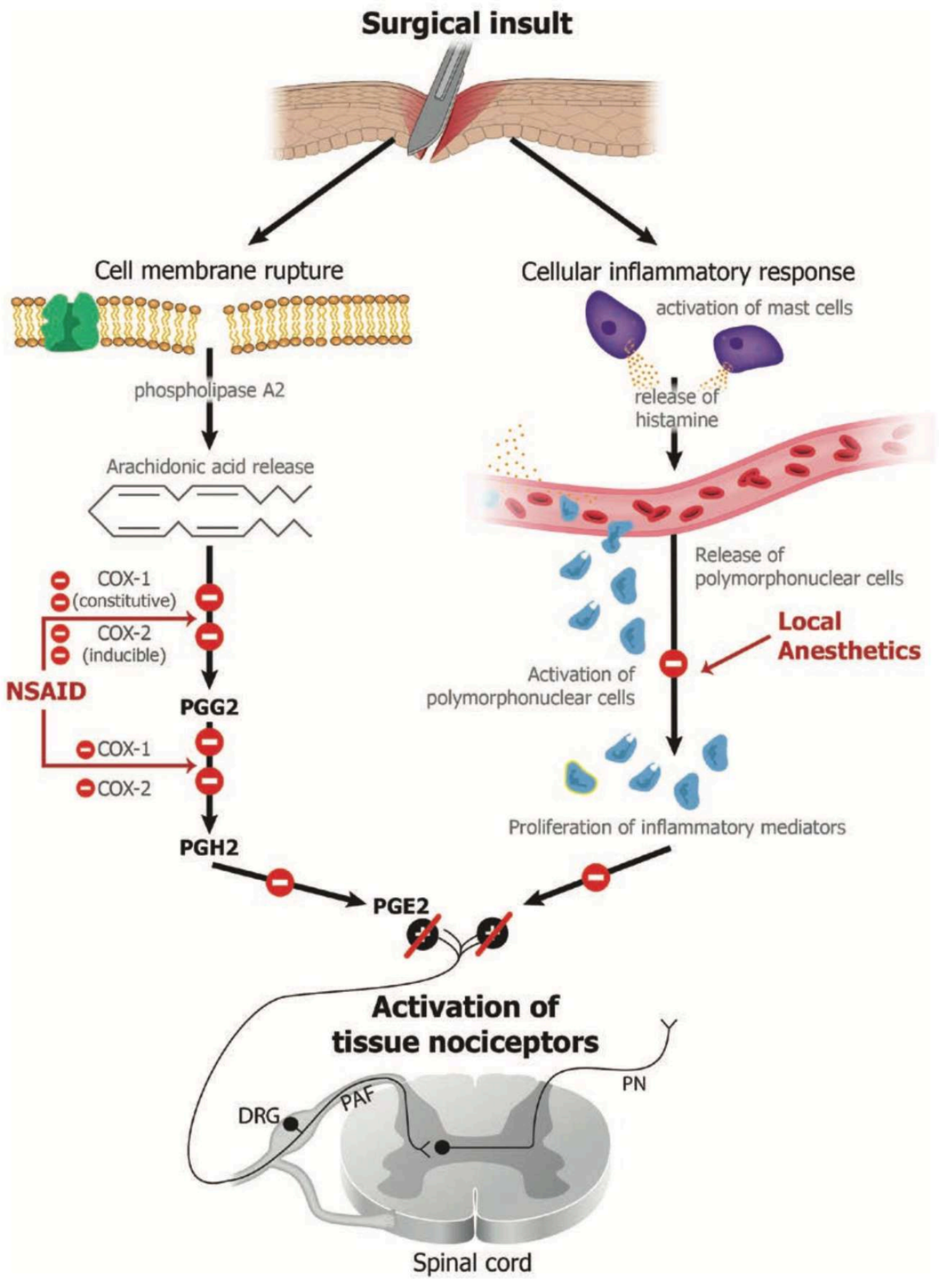


Figure 5. NSAIDs and lidocaine. Surgical insults induce rupture of cell membranes, leading to release of arachidonic acid, which, through the action of COX-1 and COX-2, is converted into prostaglandins, which are potent inflammatory and nociceptive mediators. NSAIDs modulate the nociceptive response by blocking the actions of COX-1 and COX-2, and lidocaine exerts their nociceptive effects by inactivating sodium channels, thus inhibiting excitation of nerve endings and blocking conduction of action potentials in peripheral nerves. Lidocaine also impedes neutrophil degranulation, thereby impeding the amplification of the inflammatory response. COX indicates cyclooxygenase; DRG, dorsal root ganglion; NSAID, nonsteroidal anti-inflammatory drug; PAF, peripheral afferent fiber; PGE2, prostaglandin E2; PGH2, prostaglandin H2; PGG2, prostaglandin G2; PN, projection neuron.

Table 1. Multimodal General Anesthesia: Explicit and Implicit Maintenance of the Anesthetic State

I. Anesthetic State	II. Explicit Maintenance	III. Implicit Maintenance
A. Antinociception	Ketamine (NMDA antagonist); remifentanil (opioid agonist); dexmedetomidine (α-2 agonist); magnesium (NMDA agonist); lidocaine (anti-inflammatory, sodium channel blockade); oral NSAID (anti-inflammatory)	Propofol (GABA agonist); sevoflurane (GABA agonist and other targets)
B. Unconsciousness (amnesia)	Propofol (induction and maintenance); sevoflurane	Ketamine (NMDA antagonist); remifentanil (opioid agonist); dexmedetomidine (α-2 adrenergic agonist); magnesium (NMDA receptor); cisatracurium/ rocuronium (decreased proprioception)
C. Immobility	Cisatracurium (induction and maintenance); rocuronium (induction and maintenance); succinylcholine (induction)	Magnesium (smooth muscle relaxant); propofol (central muscle relaxation); sevoflurane (central muscle relaxation)

Column I defines the behavioral component of general anesthesia. Column II gives examples of anesthetic agents that can be used to maintain the behavioral state of general anesthesia in column I. Column III shows the additional behavioral states to which the anesthetic contributes.

Abbreviations: GABA, γ-aminobutyric acid; NMDA, *N*-methyl-D-aspartate; NSAID, nonsteroidal anti-inflammatory drug.

Section Editor: **Thomas R. Vetter**

SPECIAL ARTICLE

OPEN

CME

Multimodal General Anesthesia: Theory and Practice

Emery N. Brown, MD, PhD,*†‡§‖ Kara J. Pavone, BS, BSN, RN,* and Marusa Naranjo, MD¶

medications other than opioids. In contrast, we believe that nociception should be maintained intraoperatively and postoperatively using multiple antinociceptive agents.

A recent report has summarized the modalities (non-anesthetic and anesthetic adjuncts, and regional techniques) that can be used to reduce opioid use perioperatively.[67] Our

Section Editor: **Thomas R. Vetter**

SPECIAL ARTICLE

OPEN

CME

Multimodal General Anesthesia: Theory and Practice

Emery N. Brown, MD, PhD,*†‡§‖ Kara J. Pavone, BS, BSN, RN,* and Marusa Naranjo, MD¶

ology practice. The fundamental feature of our strategy is administration of multiple antinociceptive agents simultaneously to suppress nociceptive trafficking during both general and regional anesthesia (Table 2). Each agent targets a different component of the nociceptive system. Our neural circuit analyses provide a neurophysiologically based approach for understanding the effects of each anesthetic and for choosing the anesthetic combinations (Figures 2–6). As

Table 2. Examples of Multimodal Anesthesia Care Practice

	Surgery			
	I. Bilateral L1–L4 Laminectomy With Instrumentation	**II. Right Total Knee Replacement**	**III. Scheduled Cesarean Delivery**	**IV. Exploratory Laparotomy: Abdominal Debulking**
Patient Description	78-y-old man with chronic back pain, a previous laminectomy, a rhinoplasty, a transurethral prostatectomy, and hypertension	71-y-old man with right knee osteoarthritis, a left femoral osteosynthesis, a subsequent left femur hardware removal, and hypertension	36-y-old woman gravida 3, para 1, cesarean delivery 1 at 38 wk gestation with preeclampsia with her previous pregnancy	58-y-old woman with a sarcoma, a previous hysterectomy and salpingo-oophorectomy, appendectomy, hernia repair, and sarcoma resection
Preoperative Management				
Antinociception	30 min before surgery infusions of[a] dexmedetomidine: 0.3 mcg·kg^{-1}·hour^{-1} magnesium: 15 mg·kg^{-1}·hour^{-1}	…	…	…
Sedation	…	Propofol sedation: 17–35 mcg/kg/min	…	Fentanyl 100 mcg
Intraoperative Management				
Antinociception	General anesthesia Ketamine Bolus: 0.5 mg·kg^{-1} Dexmedetomidine: 0.1–0.4 mcg·kg^{-1}·hour^{-1} Lidocaine: 0.8–1.3 mg·kg^{-1}·hour^{-1} Magnesium: 3–15 mg·kg^{-1}·hour^{-1}	Spinal anesthesia[b] Hyperbaric bupivacaine: 15 mg; clonidine: 15 mcg; morphine: 80 mcg	Spinal anesthesia[b] Hyperbaric bupivacaine: 9 mg; clonidine: 15 mcg; morphine: 60 mcg	General anesthesia Intravenous Ketamine Bolus: 0.5 mg·kg^{-1} Infusion: 0.5 mcg·kg^{-1}·minute^{-1} Dexmedetomidine: 0.2–0.3 mcg·kg^{-1}·hour^{-1} Remifentanil: 0.05–0.2 mcg·kg^{-1}·minute^{-1} Epidural Bupivacaine Bolus: 10 mg Infusion: 20 mg·hour^{-1}
Unconsciousness (amnesia)/Sedation	Propofol Induction: 150 mg; Maintenance: 17–50 mcg·kg^{-1}·minute^{-1} Sevoflurane Maintenance: 0.3–0.6 MAC	Propofol Sedation: 17–35 mcg/kg/min	Propofol Sedation: 25–50 mcg/kg/min until delivery, then discontinued	Propofol Induction: 200 mg Maintenance: 30–70 mcg·kg^{-1}·minute^{-1}
Immobility	Rocuronium Induction: 70 mg Sugammadex 140 mg for reversal before instrumentation	…	…	Succinylcholine Induction: 100 mg Rocuronium Maintenance: 40 mg (single dose)
Postoperative Management				
Pain (infiltration)	Field block[b] Mixed in 40 mL volume: Ropivacaine: 150 mg; dexmedetomidine: 40 mcg; ketorolac: 60 mg Administered as: 20 mL in the muscle layer 20 mL subcutaneous tissue	Field block[b] Mixed in 60 mL volume: Ropivacaine: 150 mg; dexmedetomidine: 60 mcg; ketorolac: 60 mg Administered as: 25 mL muscle layer 35 mL subcutaneous tissue	Field block[b] Mixed in 20 mL volume: Ropivacaine: 150 mg; dexmedetomidine: 35 mcg; ketorolac: 30 mg Administered as: 5 mL right rectus abdominis 5 mL left rectus abdominis 10 mL subcutaneous tissue	…

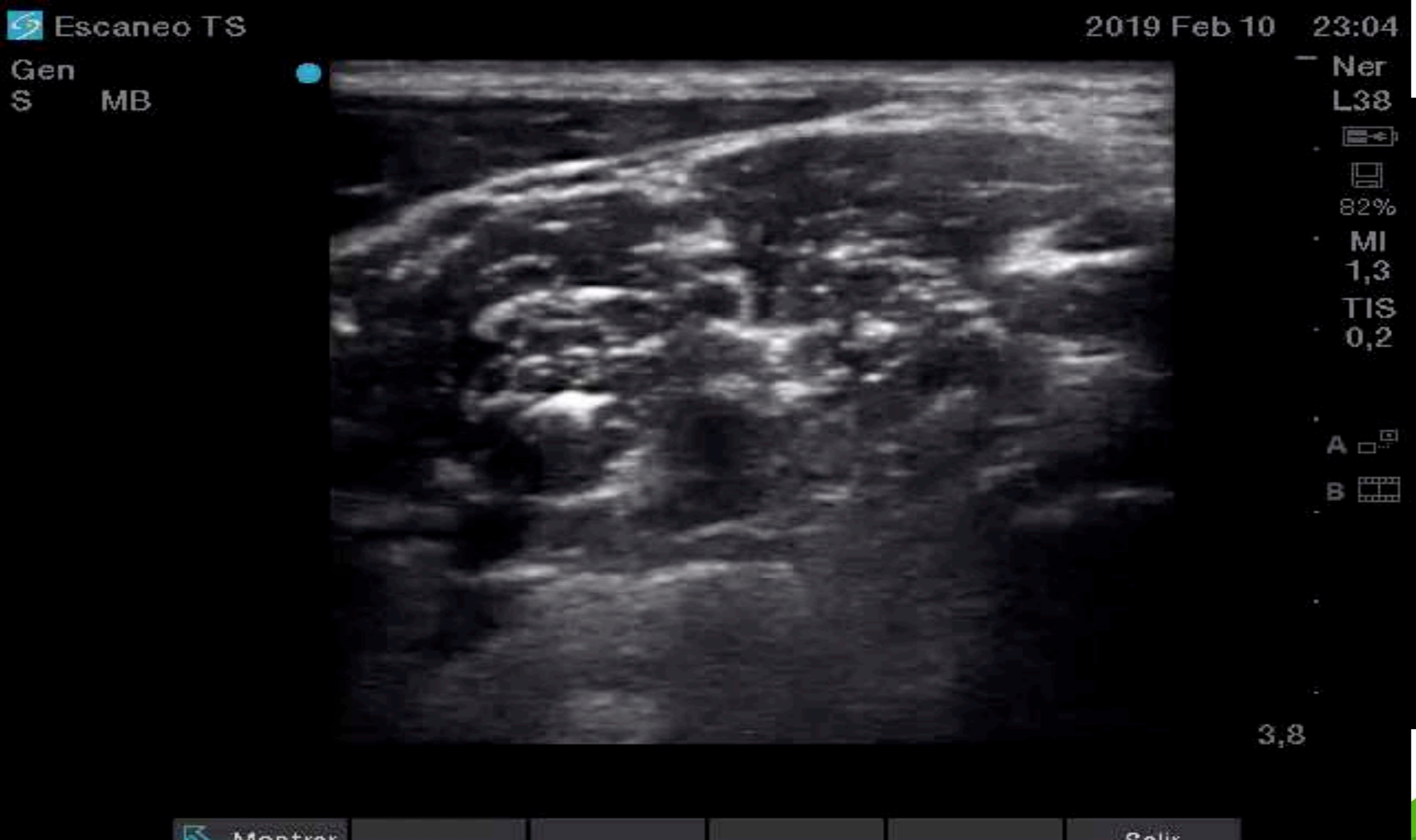
NARRATIVE REVIEW ARTICLE

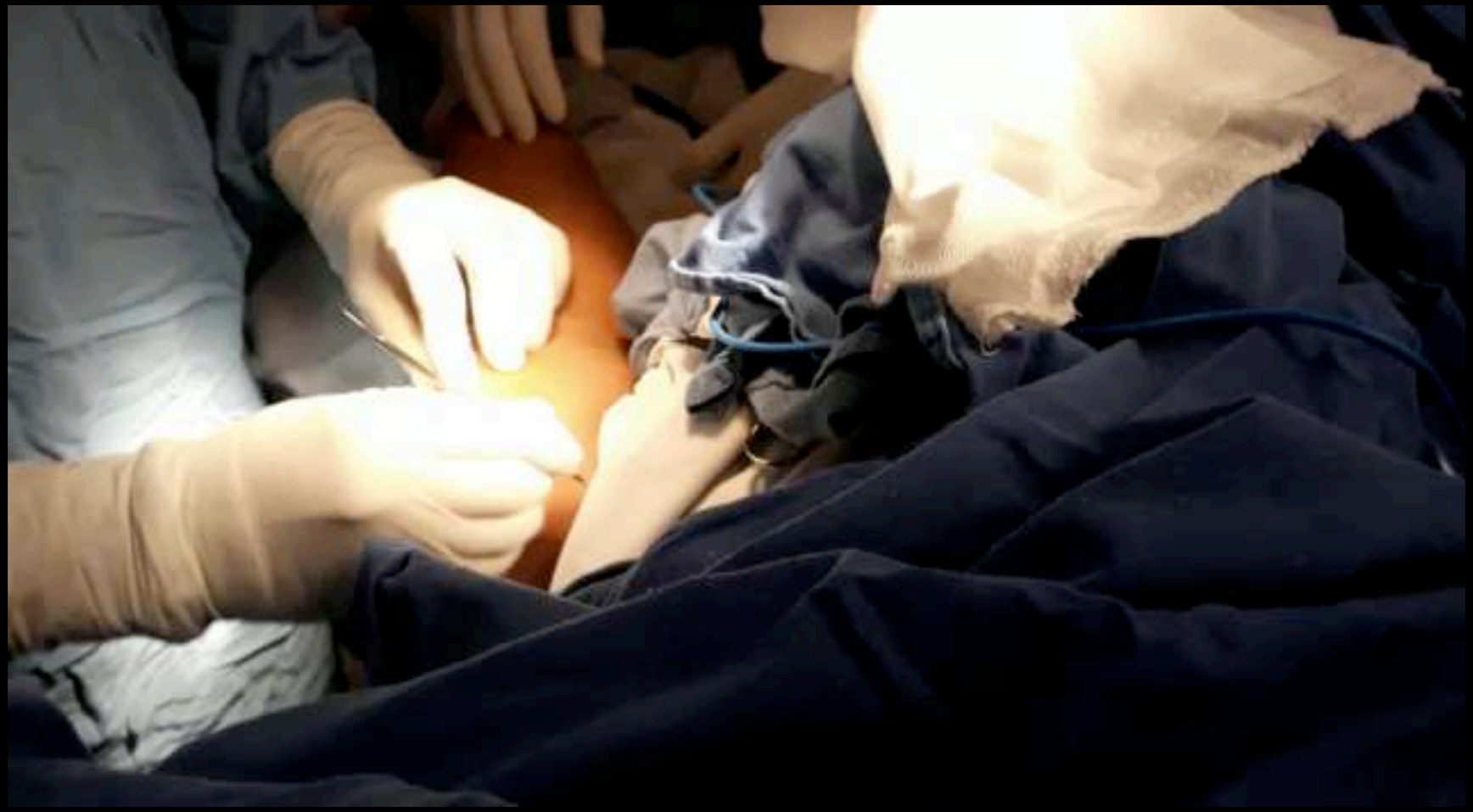
A Review of Opioid-Sparing Modalities in Perioperative Pain Management: Methods to Decrease Opioid Use Postoperatively

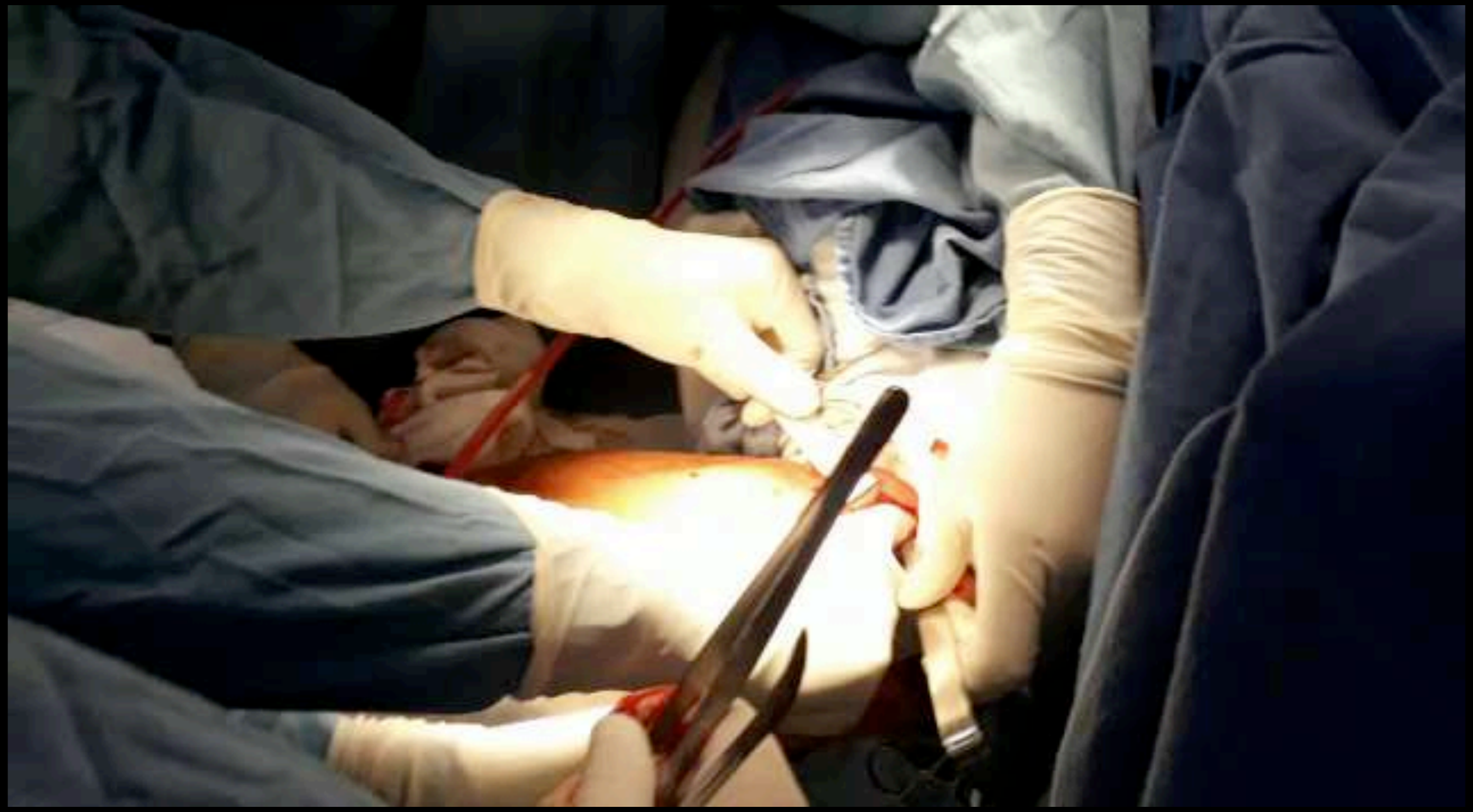
Kanupriya Kumar, MD,* Meghan A. Kirksey, MD, PhD,* Silvia Duong, BScPharm, PharmD,† and Christopher L. Wu, MD‡

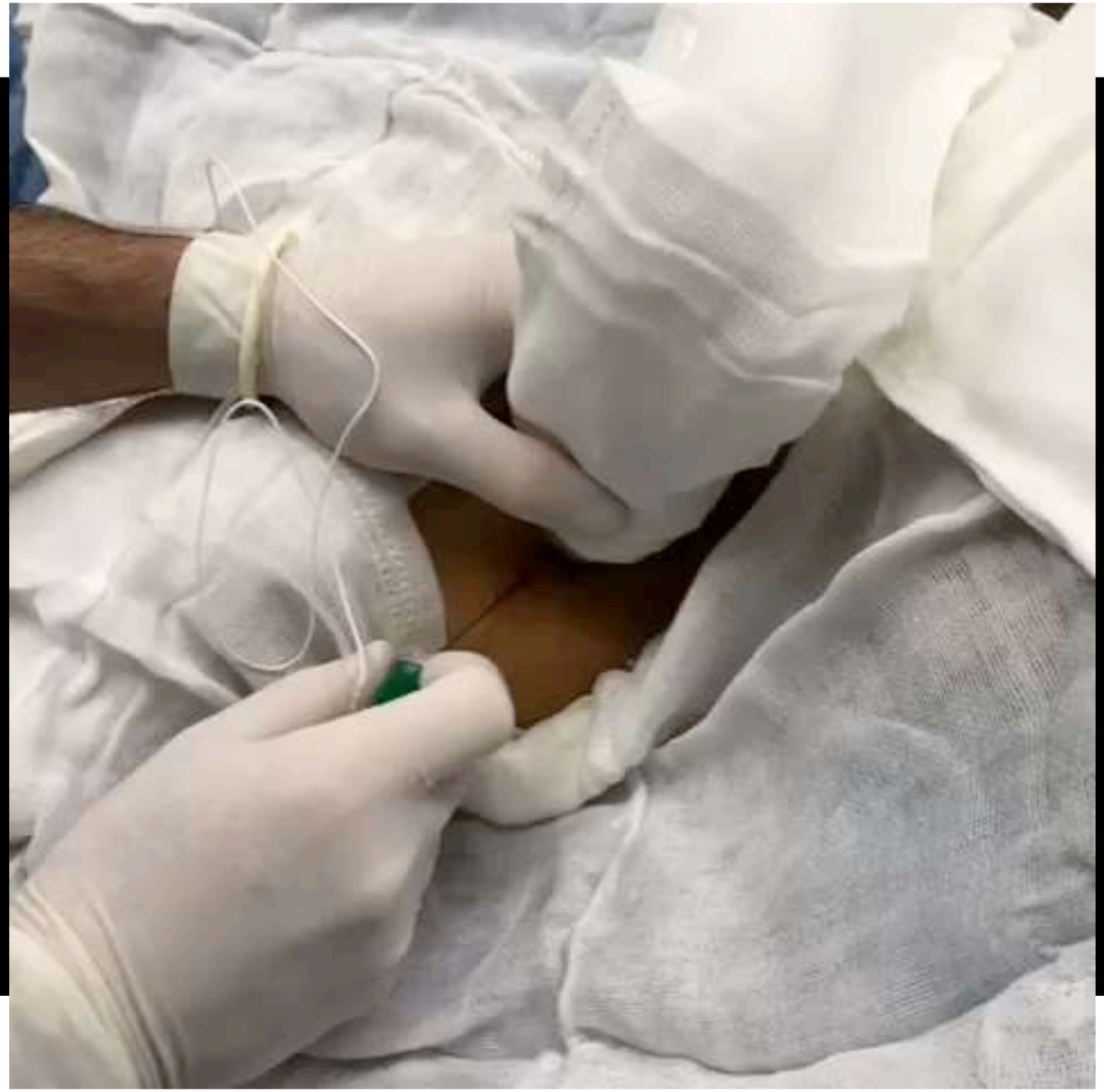
There is an epidemic of opioid use, abuse, and misuse in the United States, which results in significant morbidity and mortality. It may be difficult to reduce perioperative opioid use given known acute surgical trauma and resultant pain; however, the discrete and often limited nature of postoperative pain also may make management easier in part by utilizing nonopioid modalities, such as regional anesthesia/analgesia, and multimodal analgesia, which may decrease the need for powerful opioids. This article reviews the relevant literature describing the use of adjunct medications, regional anesthesia and analgesic techniques, and regional block additives in the context of providing adequate pain control while lessening opioid use. (Anesth Analg 2017;125:1749–60)

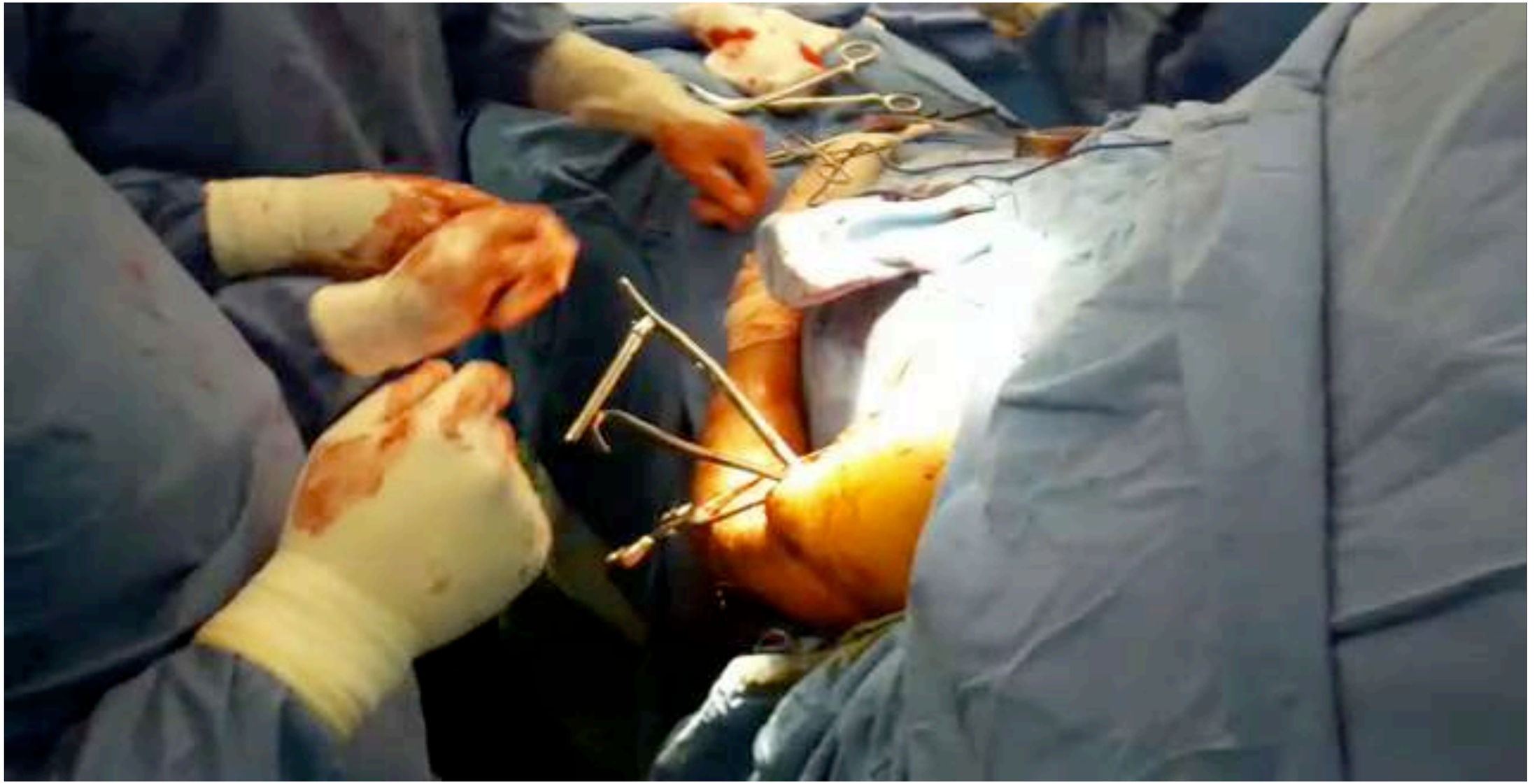
- **Revisión anatómica del área.**
- Estructuras nerviosas y circundantes.
- Guía la aguja hacia el objetivo.
- Visualiza la difusión del anestésico local.


Escaneo TS
2019 Feb 10 23:04
Gen
S MB
Ner
L38
82%
MI
1,3
TIS
0,2
A
B
3,8
Mostrar
Salir









RESULTADOS DE PREGUNTA

De la narcosis a la homeostasis perioperatoria

Geschichte der Anästhesie
“Vom Narkotiseur zum perioperativen Homöostatiker“ Dr. Phil. H. Petermann, M. A.
Anaesthesist. DOI 10.1007/s00101-016-0223-y

TOMA DE DECISIONES

Es el proceso de identificación y selección de la acción adecuada para la solución de un problema específico.

Identificación de Problemas y Oportunidades

Detección de problemas

Un problema es una situación que se produce cuando un estado de cosas real difiere del estado de cosas que se desea.

Identificación de oportunidades

¿Son oportunidades o problemas?

Una oportunidad es una situación que se produce cuando las circunstancias prevalecientes le ofrecen a la organización la posibilidad de ir más allá de las metas y los objetivos que se habían planteado.

Decisiones Programadas

Son aquellas tomadas como un hábito, regla o procedimiento; se aplican a problemas estructurados o rutinarios y en algunos casos son repetitivos y es posible definir, prever y analizar sus componentes.

En Condiciones De Certidumbre

Son aquellas que se toman con certeza acerca de lo que sucederá, ya que se cuenta con información confiable, exacta, medible y se conocen las relaciones de causa-efecto.


GOOD
DECISION
NEXT EXIT

¡Gracias!

dr.salvadorvilchis@gmail.com
cursos.armty.mx