# "No es pot administrar KETAMINA al malalt amb TCE"

to mL Mum-Dose NDC MISHE Ketaming USA 500 mg per 10 mL\* (50 mg/mL) France Ket Lake Forest, IL 600 100

Benito Pérez, Infermer d'Emergències, USVAI B-406 SEM



El repte és l'excel·lència



So MUE

 $\mathbf{D}\mathbf{O}$ 

Societat Catalana de Medicina d'Urgències i Emergències

## Què és la KETAMINA?

- Derivat liposoluble de la fenciclidina (PCP), anestèsic dissociatiu amb efectes al·lucinògens i neurotòxics que es va comercialitzar als anys 50 a EUA.
- Potent efecte hipnòtic i analgèsic degut a la seva acció als receptors GABA i opiacis.
- Inhibició NMDA al SNC provocant anestesia dissociativa, caracteritzada per catalèpsia, amnèsia i potent analgèsia. Pacient DESPERT però allunyat del dolor o qualsevol estímul extern
- Acció a nivell receptors colinèrgics. Allibera catecolamines.





## Què és la KETAMINA?













## Història









- Va aparèixer al 1962 buscant alternatives a la PCP. Aprovat per la FDA al 1970 com anestèsic segur i eficaç en persones i animals.
- Als 80, davant l'ús "alternatiu" i a la por a efectes adversos en humans (TCE?), n'hi ha una marcada reducció en la utilització en humans.
- És al final dels 90 i principis del 2000, quan retorna el seu interès en EUA en els SEM i SU hospitalaris

### Punts forts



- Efecte 4/1. Anestèsic-sedant-amnèsic-analgèsic.
- Via IV, IM, IN, IR, VO
- Ràpid inici acció (30 seg), corta duració (10 min- 70 min)
- Augmenta FC, PA, PAM. (Ideal pacient amb hipoTA). Millora contractilitat cardíaca i gasto cardíac
- Reflexos i capacitat per respirar: INTACTES



## Punts "dèbils"

- "Duració"...
- Despertar "difícil"
- Alliberació de catecolamines
- Consum d'oxigen miocàrdic
- No antídot









## EL MITE



Study	Study type	Ketamine dosage	Study population	ICP	MAP	Calculated CPP
Garner et al.⁵	Case-	2 mg/kg IV	11 healthy males for simple surgery	CSFP ↑ by mean 18 mm Hg	î by mean 28 mm Hg	î
Wyte et al.7	Case report	2 mg/kg (route unknown)	2 patients (aged 8 and 17 yr) with VP shunts, obstructive hydrocephalus (secondary to aqueductal stenosis and astrocytoma)	ICP 1 to 75 mm Hg in only 1 patient; no change in other patient	_	_
Gibbs <sup>®</sup>	Case– control	1–1.3 mg/kg IV	11 healthy patients for lumbar discectomy; second group of 9 patients with intracranial space occupying lesions	No change in CSFP in healthy patients; in group 2, CSFP 1 by ~ 12 mm Hg in 6/9	↑ by 24 mm Hg	î
Gardner et al. <sup>9</sup>	Case report	2 mg/kg IV	13-year-old boy with glioma, midline shift	CSFP ↑ by ~ 8 mm Hg	1 by ~ 16 mm Hg	Ŷ
Shapiro et al. <sup>10</sup>	Case– control	2 mg/kg IV or 4 mg/kg IM	7 patients (5 with external shunts and ↑ ICP)	No change in patients without shunts; ICP 1 up to 60 mm Hg in certain patients	↑ up to 22 mm Hg	Variable
List et al. <sup>11</sup>	Case– control	2 mg/kg IV	7 patients with hydrocephalus	1 patient had 1 CSFP to ~ 25 mm Hg; others had mild 1 CSFP within normal range	_	_

VP = ventriculoperitoneal.

- Sis estudis realitzats a la dècada de 1970, "discrets" en mètodes qualitat, petits informes de casos "control".
- Estudis realitzats en pacients amb lesions intracranials NO traumàtiques.
- Els canvis en la PIC es mesuraven quan s'anaven produint canvis en la pressió del LCR a la columna lumbar i ventricles laterals. Aquests canvis els extrapolaven a canvis en el FSC.
- L'associació de ketamina i augment de la PIC la feien amb pacients amb vies de LCR anormals (com les causades per estenosi aqüeducte Silvio, hidrocefàlia i altres patologies amb efecte massa.
- En pacients sans, la PIC es mantenia en nivells normals <10mm Hg. N'hi havia l'augment de PAM i això augmentava FSC.



### EL MITE





## **EVIDÈNCIA**



Study	Study type	Study population	ICP	CPP
Mayberg et al. <sup>12</sup>	Prospective trial	<ul> <li>20 neurosurgical patients (10 with supratentorial tumours, the rest with intracranial aneurysms)</li> <li>ICP measured before and after administration of ketamine 1 mg/kg IV</li> </ul>	Small but statistically significant decrease in ICP after ketamine administration	No significant change over 10 min
Kolenda et al. <sup>13</sup>	Prospective RCT	<ul> <li>35 patients with moderate or severe head injury</li> <li>Ketamine + midazolam sedation v. fentanyl + midazolam sedation</li> </ul>	Slightly higher ICP values in the ketamine group (~ 2 mm Hg difference)	Higher in the ketamine group than the control group by average of 8 mm Hg
Bourgoin et al.14	Prospective double-blind RCT	<ul> <li>25 patients with severe head injury</li> <li>Continuous infusion ketamine-midazolam v. sufentanil-midazolam infusion</li> </ul>	No significant difference between groups	No significant difference between groups
Bourgoin et al.¹⁵	Prospective double-blind RCT	<ul> <li>30 patients with TBI receiving sufentanil- midazolam or ketamine- midazolam using target controlled infusion</li> </ul>	No significant difference between groups	No significant difference between groups
Schmittner et al. <sup>16</sup>	Randomized prospective trial	<ul> <li>24 patients with TBI</li> <li>Group 1: methohexitone + ketamine sedation</li> <li>Group 2: methohexitone + fentanyl sedation</li> </ul>	No significant difference between groups	No significant difference between groups

CPP = cerebral perfusion pressure; ICP = intracranial pressure; IV = intravenously; RCT = randomized controlled trial; TBI = traumatic brain injury.



#### CONCLUSION:

Patients with intracranial hypertension should not be excluded from receiving Ketamine during intubation out of concern for worsening outcomes.

We conducted a retrospective chart review of patients transported to our facility with evidence of intracranial hypertension that were intubated before trauma center arrival. Patients were identified during a 22-month period from January 2014 to October 2015. Goals were to evaluate the impact of sedative agent selection on morbidity, mortality, and length of stay.

#### RESULTS:

Med

World

During the review 148 patients were identified as meeting inclusion criteria, 52 were excluded due to incomplete data. Of those the patients primarily received; Etomidate, Ketamine, and Midazolam. Patients in the Ketamine group were found to have a lower mortality rate after injury stratification.

#### CONCLUSION:

Patients with intracranial hypertension should not be excluded from receiving Ketamine during intubation out of concern for worsening outcomes.

Keywords: Ketamine, Intracranial hypertension, Craniocerebral trauma, Head injury, Intubation

J Anesth. 2014 Dec;28(6):821-7. doi: 10.1007/s00540-014-1845-3. Epub 2014 May 24.

#### Ketamine does not increase intracranial pressure compared with opioids: meta-analysis of randomized controlled trials.

Wang X<sup>1</sup>, Ding X, Tong Y, Zong J, Zhao X, Ren H, Li Q.

Author information

#### Abstract

so**r**<sup>2</sup>MUE

**BACKGROUND:** Ketamine is traditionally avoided in sedation management of patients with risk of intracranial hypertension. However, results from many clinical trials contradict this concern. We critically analyzed the published data of the effects of ketamine on intracranial

**CONCLUSIONS:** The results of this study suggest that ketamine does not increase ICP compared with opioids. Ketamine provides good maintenance of hemodynamic status. Clinical application of ketamine should not be discouraged on the basis of ICP-related concerns.

Central (last search performed on January 15, 2014). That characteristics and outcomes were independently extracted by two assessors (Xin Wang, Xibing Ding). For continuous data, mean differences (MD) were formulated. If the P value of the chi-square test was >0.10 or I(2) <50%, a fixed-effects model was used; otherwise, the random effects model was adopted.

**RESULTS:** Five trials (n = 198) met the inclusion criteria. Using ICP levels within the first 24 h of ketamine administration as the main outcome, the use of ketamine leads to the same ICP levels as opioids [MD = 1.94; 95% confidence interval (95% CI), -2.35, 6.23; P = 0.38]. There were no significant differences in mean arterial pressure values between the two groups (MD = 0.99; 95% CI, -2.24, 4.22; P = 0.55). Ketamine administration was also comparable with opioids in the maintenance of cerebral perfusion pressure (MD = -1.07; 95% CI, -7.95, 5.8; P = 0.76).

**CONCLUSIONS:** The results of this study suggest that ketamine does not increase ICP compared with opioids. Ketamine provides good maintenance of hemodynamic status. Clinical application of ketamine should not be discouraged on the basis of ICP-related concerns.

J Neurosurg Pediatr. 2009 Jul;4(1):40-6. doi: 10.3171/2009.1.PEDS08319.

#### Effectiveness of ketamine in decreasing intracranial pressure in children with intracranial hypertension.

Bar-Joseph G<sup>1</sup>, Guilburd Y, Tamir A, Guilburd JN.

Author information

#### Abstract



**OBJECT:** Deepening sedation is often needed in patients with intracranial hypertension. All widely used sedative and anesthetic agents (opioids, benzodiazepines, propofol, and barbiturates) decrease blood pressure and may therefore decrease cerebral perfusion pressure (CPP). Ketamine is a potent, safe, rapid-onset anesthetic agent that does not decrease blood pressure. However, ketamine's use in patients with traumatic brain injury and intracranial hypertension is precluded because it is widely stated that it increases intracranial pressure (ICP). Based on anecdotal clinical experience, the authors hypothesized that ketamine does not

**CONCLUSIONS:** In ventilation-treated patients with intracranial hypertension, ketamine effectively decreased ICP and prevented untoward ICP elevations during potentially distressing interventions, without lowering blood pressure and CPP. These results refute the notion that ketamine increases ICP. Ketamine is a safe and effective drug for patients with traumatic brain injury and intracranial hypertension, and it can possibly be used safely in trauma emergency situations.

compare these values with those recorded every minute for 10 minutes following ketamine administration.

**RESULTS:** The results of 82 ketamine administrations in 30 patients were analyzed. Overall, following ketamine administration, ICP decreased by 30% (from 25.8 +/- 8.4 to 18.0 +/- 8.5 mm Hg) (p < 0.001) and CPP increased from 54.4 +/- 11.7 to 58.3 +/- 13.4 mm Hg (p < 0.005). In Group 1, ICP decreased significantly following ketamine administration and increased by > 2 mm Hg during the distressing intervention in only 1 of 17 events. In Group 2, when ketamine was administered to lower persistent intracranial hypertension, ICP decreased by 33% (from 26.0 +/- 9.1 to 17.5 +/- 9.1 mm Hg) (p < 0.0001) following ketamine administration.

**CONCLUSIONS:** In ventilation-treated patients with intracranial hypertension, ketamine effectively decreased ICP and prevented untoward ICP elevations during potentially distressing interventions, without lowering blood pressure and CPP. These results refute the notion that ketamine increases ICP. Ketamine is a safe and effective drug for patients with traumatic brain injury and intracranial hypertension, and it can possibly be used safely in trauma emergency situations.

Ann Emerg Med. 2015 Jan;65(1):43-51.e2. doi: 10.1016/j.annemergmed.2014.06.018. Epub 2014 Jul 23.

### The effect of ketamine on intracranial and cerebral perfusion pressure and health outcomes: a systematic review.

Cohen L<sup>1</sup>, Athaide V<sup>1</sup>, Wickham ME<sup>2</sup>, Doyle-Waters MM<sup>3</sup>, Rose NG<sup>4</sup>, Hohl CM<sup>5</sup>.

Author information

#### Abstract

**STUDY OBJECTIVE:** We synthesize the available evidence on the effect of ketamine on intracranial and cerebral perfusion pressures, neurologic outcomes, ICU length of stay, and mortality.

METHODS: We developed a systematic search strategy and applied it to 6 electronic reference databases. We completed a gray literature

CONCLUSION: According to the available literature, the use of ketamine in critically ill patients does not appear to adversely affect patient outcomes.

standardized forms. Data from randomized controlled trials and prospective studies were synthesized in a qualitative manner because the study designs, patient populations, reported outcomes, and follow-up periods were heterogeneous. We used the Jadad score and Cochrane Risk of Bias tool to assess study quality.

**RESULTS:** We retrieved 4,896 titles, of which 10 studies met our inclusion criteria, reporting data on 953 patients. One study was deemed at low risk of bias in all quality assessment domains. All others were at high risk in at least 1 domain. Two of 8 studies reported small reductions in intracranial pressure within 10 minutes of ketamine administration, and 2 studies reported an increase. None of the studies reported significant differences in cerebral perfusion pressure, neurologic outcomes, ICU length of stay, or mortality.

CONCLUSION: According to the available literature, the use of ketamine in critically ill patients does not appear to adversely affect patient outcomes.

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#### Comment in

Ketamine and intracranial pressure: no contraindication except hydrocephalus. [Ann Emerg Med. 2015]



Ann Emerg Med. 2015 Jan;65(1):43-51.e2. doi: 10.1016/j.annemergmed.2014.06.018. Epub 2014 Jul 23.

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METHODS: We developed a systematic search strategy and applied it to 6 electronic reference databases. We completed a gray literature search and searched medical journals as well as the bibliographies of relevant articles. We included randomized and poprandomized

CONCLUSION: According to the available literature, the use of ketamine in critically ill patients does not appear to adversely affect patient outcomes.

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#### Comment in

Ketamine and intracranial pressure: no contraindication except hydrocephalus. [Ann Emerg Med. 2015]



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Neurocrit Care. 2014 Aug;21(1):163-73. doi: 10.1007/s12028-013-9950-y.

#### The ketamine effect on ICP in traumatic brain injury.

Zeiler FA<sup>1</sup>, Teitelbaum J, West M, Gillman LM.

Author information

#### Abstract

Our goal was to perform a systematic review of the literature on the use of ketamine in traumatic brain injury (TBI) and its effects on intracranial pressure (ICP). All articles from MEDLINE, BIOSIS, EMBASE, Global Health, HealthStar, Scopus, Cochrane Library, the

#### There currently exists Oxford level 2b, GRADE C evidence to support that ketamine does not increase ICP in severe TBI patients

#### that are sedated and ventilated, and in fact may lower it in selected cases.

treatment characteristics. The strength of evidence was adjudicated using both the Oxford and GRADE methodology. Our search strategy produced a total 371 citations. Seven articles, six manuscripts and one meeting proceeding, were considered for the review with all utilizing ketamine, while documenting ICP in severe TBI patients. All studies were prospective studies. Five and two studies pertained to adults and pediatrics, respectively. Across all studies, of the 101 adult and 55 pediatric patients described, ICP did not increase in any of the studies during ketamine administration. Three studies reported a significant decrease in ICP with ketamine bolus. Cerebral perfusion pressure and mean blood pressure increased in two studies, leading to a decrease in vasopressors in one. No significant adverse events related to ketamine were recorded in any of the studies. Outcome data were poorly documented. There currently exists Oxford level 2b, GRADE C evidence to support that ketamine does not increase ICP in severe TBI patients that are sedated and ventilated, and in fact may lower it in selected cases.

## Conclusions Evidència Científica



- La evidència que conclou que la ketamina augmenta la PIC és molt dèbil i questionable.
- <u>No</u> n'hi ha evidencia que la ketamina provoqui <u>danys</u> en el TCE. El que podem veure és la manca de dany demostrable que la ketamina té en els paràmetres neurològics d'interès quan es compara amb el grup control en cadascun dels papers.
- L'estabilitat hemodinàmica de la ketamina podria ser <u>beneficiosa</u> en el pacient amb TCE que requereix SIR

#### TABLE 5

#### Relative Contraindications to Ketamine Use for Acute Pain





### Fins aquí el MITE....







i DOLOR





Onset is in 1 min if IV, 5 min if IM or IN.

Care (TCCC), and the Army's 75th Ranger Regiment, have now been incorporated into the Triple-Option Analgesia approach. This novel strategy has gained wide acceptance in the US military. It calls for battlefield



IN ketamine resulted in a below-threshold increase in side effects over fentanyl with similar levels of pain reduction, supporting the feasibility of a large-scale, multi-center pediatric trial to assess efficacy and safety of IN ketamine for acute pain management in peds extremity injuries.

Fenta Diff (9	nyl 15% (1)	- 35 mm [-5 to 23]		80% [-20 to 15]		(60 min)
		•••		××		
	Any s	side effects	Bad Taste	Dizzy	Sleepy	Itchy Nose
Ketami	ine	100% ★	90% 🗡	73%	46%	24%
Fentan	yl	61%	22%	15%	37% ★	22%
<b>CONCLUSION</b> IN ketamine resulted in a below-threshold increase in side effects over fentanyl with similar levels of pain reduction, supporting the feasibility of a large-scale, multi-center pediatric trial to assess efficacy and safety of IN ketamine for acute pain management in peds extremity injuries.						
	@PainProfiles					

#### Intravenous Subdissociative-Dose Ketamine Versus Morphine for Analgesia in the Emergency Department: A Randomized Controlled Trial

### Anal

Sergey Motov, MD\*; Bradley Rockoff, MD; Victor Cohen, PharmD; Illya Pushkar, MPH; Antonios Likourezos, MA, MPH; Courtney McKay, PharmD; Emil Soleyman-Zomalan, MD; Peter Homel, PhD; Victoria Terentiev, BA; Christian Fromm, MD

\*Corresponding Author. E-mail: smotov@maimonidesmed.org, Twitter: @smotovmd.



**Study objective:** We assess and compare the analgesic efficacy and safety of subdissociative intravenous-dose ketamine with morphine in emergency department (ED) patients.

**Conclusion:** Subdissociative intravenous ketamine administered at 0.3 mg/kg provides analgesic effectiveness and apparent safety comparable to that of intravenous morphine for short-term treatment of acute pain in the ED. [Ann Emerg Med. 2015; **1**-8.]

at 30 and 60 minutes.

**Results:** Forty-five patients per group were enrolled in the study. The primary change in mean pain scores was not significantly different in the ketamine and morphine groups: 8.6 versus 8.5 at baseline (mean difference 0.1; 95% confidence interval -0.46 to 0.77) and 3.2 versus 4.2 at 30 minutes (mean difference 0.2; 95% confidence interval -1.19 to 1.46; *P*=.97). There was no difference in the incidence of rescue fentanyl analgesia at 30 or 60 minutes. No statistically significant or clinically concerning changes in vital signs were observed. No serious adverse events occurred in either group. Patients in the ketamine group reported increased minor adverse effects at 15 minutes post-drug administration.

**Conclusion:** Subdissociative intravenous ketamine administered at 0.3 mg/kg provides analgesic effectiveness and apparent safety comparable to that of intravenous morphine for short-term treatment of acute pain in the ED. [Ann Emerg Med. 2015; **1**-8.]

Please see page XX for the Editor's Capsule Summary of this article.

### Com l'administrem?

Reg Anesth Pain Med. 2018 Jul;43(5):456-466. doi: 10.1097/AAP.0000000000000806.

Consensus Guidelines on the Use of Intravenous Ketamine Infusions for Acute Pain Management From the American Society of Regional Anesthesia and Pain Medicine, the American Academy of Pain Medicine, and the American Society of Anesthesiologists.

Schwenk ES, Viscusi ER, Buvanendran A<sup>1</sup>, Hurley RW<sup>2</sup>, Wasan AD<sup>3</sup>, Narouze S<sup>4</sup>, Bhatia A<sup>5</sup>, Davis FN<sup>6</sup>, Hooten WM<sup>7</sup>, Cohen SP<sup>8</sup>.

Author information

#### Abstract

BACKGROUND: Ketamine infusions have been used for decades to treat acute pain, but a recent surge in usage has made the infusions a mainstay of treatment in emergency departments, in the perioperative period in individuals with refractory pain, and in opioid-tolerant patients. The widespread variability in patient selection, treatment parameters, and monitoring indicates a need for the creation of consensus guidelines.

**METHODS:** The development of acute pain ketamine guidelines grew as a corollary from the genesis of chronic pain ketamine guidelines. The charge for the development of acute pain ketamine guidelines was provided by the Boards of Directors of both the American Society of Regional Anesthesia and Pain Medicine and the American Academy of Pain Medicine, who approved the document along with the American Society of Anesthesiologists' Committees on Pain Medicine and Standards and Practice Parameters. The committee chair developed questions based on input from the committee during conference calls, which the committee then refined. Groups of 3 to 5 panel members and the committee chair were responsible for answering individual questions. After preliminary consensus was achieved, the entire committee made further revisions via e-mail and conference calls.

**RESULTS:** Consensus guidelines were prepared in the following areas: indications, contraindications for acute pain and whether they differ from those for chronic pain, the evidence for the use of ketamine as an adjunct to opioid-based therapy, the evidence supporting patient-controlled ketamine analgesia, the use of nonparenteral forms of ketamine, and the subanesthetic dosage range and whether the evidence supports those dosages for acute pain. The group was able to reach consensus on the answers to all questions.

**CONCLUSIONS:** Evidence supports the use of ketamine for acute pain in a variety of contexts, including as a stand-alone treatment, as an adjunct to opioids, and, to a lesser extent, as an intranasal formulation. Contraindications for acute pain are similar to those for chronic pain, partly based on the observation that the dosage ranges are similar. Larger studies evaluating different acute pain conditions are needed to enhance patient selection, determine the effectiveness of nonparenteral ketamine alternatives, define optimal treatment parameters, and develop protocols optimizing safety and access to care.



Clinical Practice Guideline for Emergency Department Ketamine Dissociative Sedation: 2011 Update

Steven M. Green, MD<sup>a,\*</sup> 1, Mark G. Roback, MD<sup>b</sup>, Robert M. Kennedy, MD<sup>c</sup>, Baruch Krauss, MD, EdM<sup>d</sup>

#### Retrics

DOI: https://doi.org/10.1016/j.annemergmed.2010.11.030

E Article Info

Abstract Full Text Images References

#### Article Outline

- I. Introduction
- II. Why a Separate Clinical Practice Guideline for Ketamine?
- III. Materials and Methods
- IV. Explanation of Clinical Practice Guideline Content
- A. <u>Objective</u> B. Definition of Dissociative Sedation
- C. Characteristics of the Ketamine "Dissociative State"
- D. Indications
- E. Contraindications: Absolute (Risks Essentially Always Outweigh Benefits)
- Age
   Mental state
- F. Contraindications: Relative (Risks May Outweigh Benefits)
- 1. Age
- 2. Laryngeal stimulation
- 3. Anatomy
- Upper respiratory infections
   Asthma
- 5. <u>Astrima</u>
- 6. Laryngospasm
- <u>Cardiac disease</u>
   Increased intracranial pressure
- S. Increased Intracranial pro

## Com l'administrem?

Adult: Dosi dissociativa 1-2 mg/kg IV en bolus durant 1' Dosi analgèsica 0.3-0.5 mg/kg IV en bolus o intermitent 10' Midazolam 0.03 mg/kg IV Antiemètic IV Dosi IM 4-5 mg/kg Dosi IN??

Pediatria: Dosi dissociativa >1.5 mg/kg

No es recomana administració d'anticolinèrgics de forma rutinària No es recomana administració d'antiemètics de forma rutinària



KETAMINA INTRANASAL -dosis màx Dosis analgésico-sedativa (0,75 - 1 mg/kg)					
	(kg)		ml dosis IN		
	5		0,1		
	10		0,2		
	15		0,3		
	20		0,4		
	25		0,5		
	30		0,6		
	35		0,7		
	40		0,8		
	50		1		
	60		1,2		
	70		1,4		
	80		1,6		
	90		1,8		
	100		2		
	110		2,2		
	120		2,4		
	Dosis 1 mg/Kg IN				

Guia Farmacologica de emergencias EPES-061. Any 2011 X. Basurto. (Congrés SoCMUE'17)

## Possibles efectes secundaris



Analgèsia i efectes en NENS:

- Sialorrea (infreqüent i poc perillós)
- Laringoespasme 0.3%



- Apnea (0.8% només IV i associada a velocitat infusió)
- Agitació al despertar (lleu 6.3%, important 1.4%)
- Nàusees i Vòmits (5-15%)
- Reaccions al "despertar" (10-30%)







### Exemple 1



Comença agitació...

### Analgèsia?? Sedació??











• . S'ha posat en oxigen i s'estableix una línia intraòssia en l'húmer proximal. Després de l'anestèsia de l'húmer canulat amb lidocaïna, s'administra ketamina. En pocs minuts, el pacient informa una reducció significativa del dolor, però no té cap canvi en l'estat mental, el comportament, l'esforç respiratori o l'estatus hemodinàmic.



## Exemple 2



- Home de 62 a amb dx de pneumònia i xoc sèptic
- Malgrat l'oxigen amb venturi al 50%, el seu SpO2 es manté sobre 70%.
- Hipotens, taquicàrdic, diaforètic ....
- Està assegut amb treball respiratori important.
- A causa de l'ansietat i falta de cooperació del pacient, el manteniment d'un segell de màscara és gairebé impossible





## Exemple 3



- Home 28 a, AP asma, ara presenta dispnea amb broncoespasme greu
- TA 146/55, FC 120, FR 14, SP02 80%
- Disminució nivell de consciència
- Silenci auscultatori

SIR + IOT





### Conclusions



- La ketamina és una de les drogues més versàtils però simultàniament, més incompresa encara al 2019.
- Els seus usos abasten des de l'analgèsia fins a la broncodilatació i té un perfil de sedació que cap altre agent pot igualar.
- És una droga **SEGURA**. La ketamina preserva els reflexos de les vies respiratories i l'impuls respiratori d'una manera que cap altre sedant pot.
- La ketamina ha mostrat efectes *sinèrgics* amb altres opiacis i sedants usats habitualment.
- Droga ideal en el malalt *INESTABLE*

## Conclusions



Esperem amb interès el dia en què la nostra pregunta ja no sigui,

#### "A quins pacients podem donar ketamina?" sinó

"En quins pacients hem de considerar donar qualsevol altra cosa?"

### Presentacions







Spravato™ (esketamine) ©

## Now Approved

SPRAVATO<sup>™</sup> is a prescription medicine, used along with an antidepressant taken by mouth, for treatment-resistant depression (TRD) in adults.

## Moltes GRÀCIES!!!!!



# I recordeu....

