NUEVAS Evidencias en la atención inicial del paciente intoxicado en urgencias.

Dr. Jordi Puiguriguer Ferrando

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OVIEDO, 20 de marzo de 2025

Sin conflictos de interés, ni empleo de la IA, en la elaboración y exposición de esta presentación

- ✓ **Sospechar la posibilidad de intoxicación.**
- ✓ Ante disyuntiva clínica y anamnesis**, debe prevalecer la clínica.**
- ✓ Precisa una **actuación y exploración sistemática,** evita errores.
- ✓ Las **medidas generales** (A,B,C…) serán casi siempre **prioritarias.**
- ✓ **Individualizar** cualquier **terapia específica** (indicadas sólo en situaciones muy concretas).
  → **Relación riesgo /beneficio**: **claramente beneficiosa.**
- ✓ **Registrar** todas las actuaciones de forma rigurosa.

“LA PRIORIDAD (casi) SIEMPRE ES EL PACIENTE, NO EL TÓXICO”

DIARREAS

CONVULSIONES

VIOLENCIA EXTREMA

DIAFORESIS

CRISIS HTA

COMA

DOLOR INTENSO

HIPERTERMIA

TOS

ASTENIA

VOMITOS

SHOCK - HIPOTENSIÓN

TEMBLORES

ASINTOMÁTICO

ARRITMIAS

SCA

ACIDOSIS SEVERA

TAQUICARDIA

INSUF RESPIRATORIA

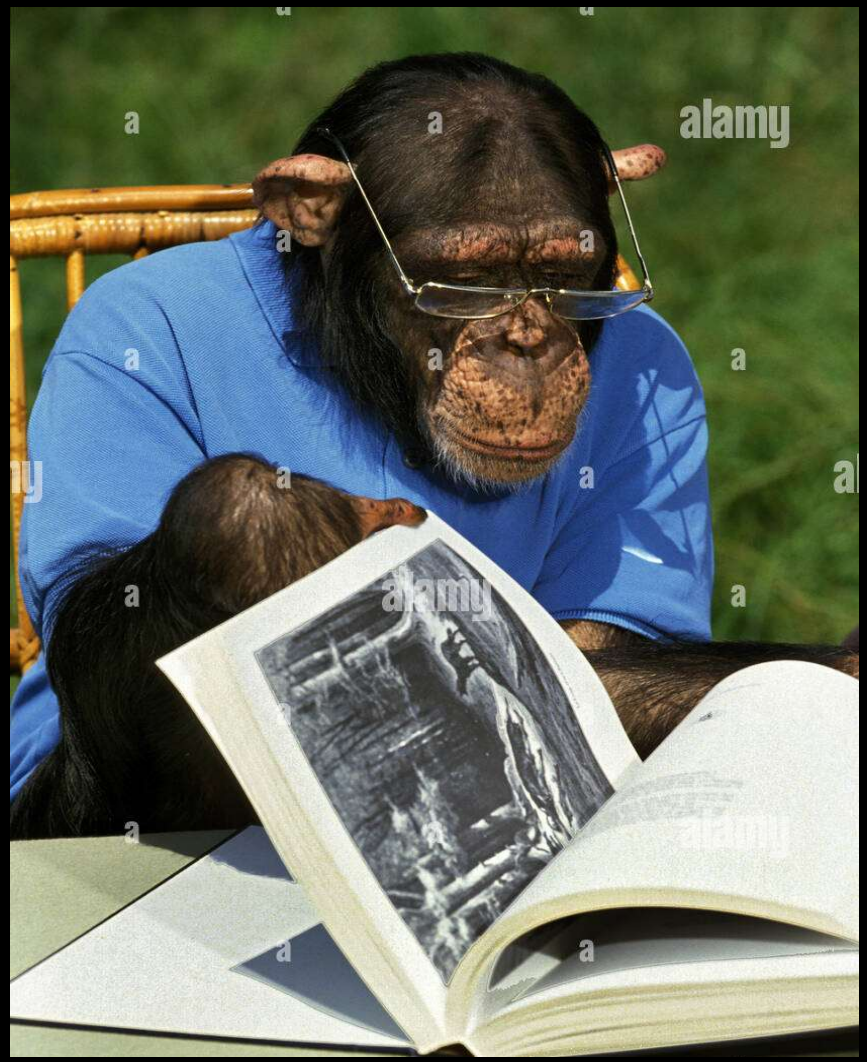
CARTAS CIENTÍFICAS

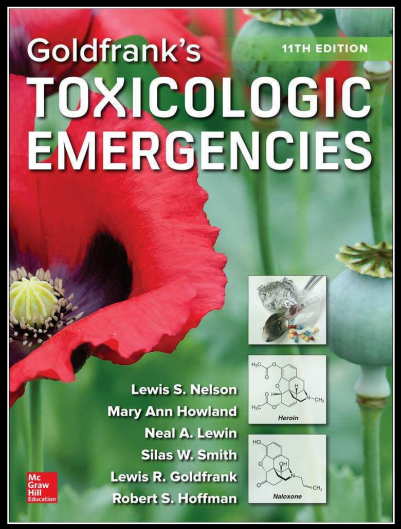
¿Puede la inteligencia artificial ayudar al urgenciólogo en el diagnóstico de las intoxicaciones?

Can artificial intelligence help the emergency physician diagnose poisoning?

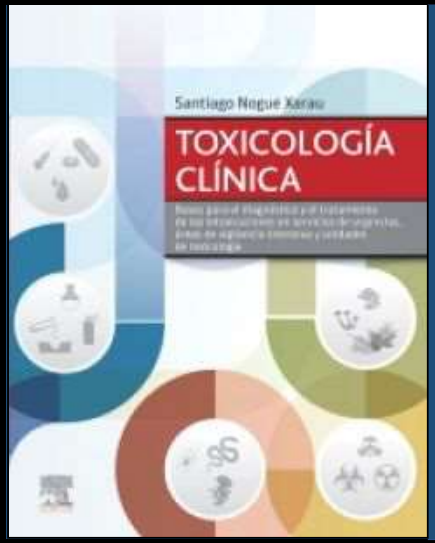
Santiago Nogué-Xarau[1], Montserrat Amigó-Tadin[2], José Ríos-Guillermo[3]

Asistencia inicial al intoxicado→ exige sistemática

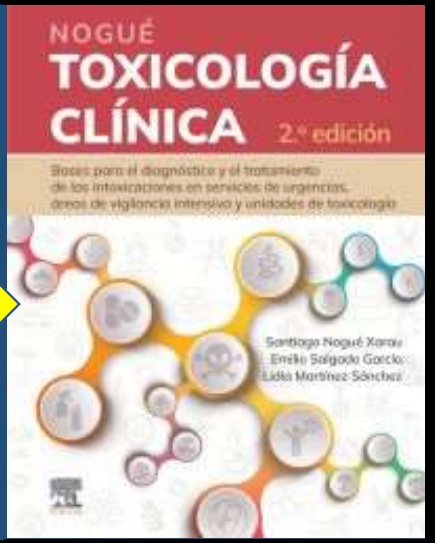




iTox
URGENCIAS POR INTOXICACIÓN
EN EL ADULTO Y NIÑO



2024



COMPROBACION DE UNA VIA AEREA PERMEABLE	VENTILACIÓN OXIGENACION	CIRCULACIÓN	SISTEMA NERVIOSO CENTRAL
			
- Liberar secreciones o restos de vómito - aspiración - cánula de Guedel - IOT + VM	- PLS y control Sat O2 - Mascarilla O2 - Antidotos (si los hubiera) - IOT + VM	- Control arritmias - Hipotensión - acceso venoso - cristaloides - drogas (NA)	-Coma - descartar hipoglucemia u otras causas. - PLS y control sat O2 -Convulsión - Diazepan / clonazepan Agitación violencia

Oct 2023

Professor of Emergency Medicine, Denver Health

Eric Labovas

Circulation

2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Lavonas EJ, Akpunonu PD, Arens AM, et al; American Heart Association. 2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2023 ;148(16):e149-e184.

1. Fallo cardio-respiratorio agudo grave **reversible.**

El tratamiento del paro cardíaco y de la toxicidad potencialmente mortal tras una intoxicación **requiere**, además de un soporte vital básico y avanzado eficaz, tratamientos especializados que la mayoría de los médicos no utilizan con frecuencia: antídotos, algunos medicamentos y ECMO.

Illum B,. Curr Treat Options Neurol. 2021;23(5):15.

En algunos casos, pueden ser necesarias terapias extracorpóreas para la eliminación de fármacos (p. ej., hemodiálisis) o soporte cardiovascular (p. ej., ECMO-VA) para la supervivencia y la recuperación.

- ECMO veno-venosa (VV ECMO): fracaso respiratorio refractario.
- ECMO veno-arterial (VA ECMO): shock cardiogénico refractario.

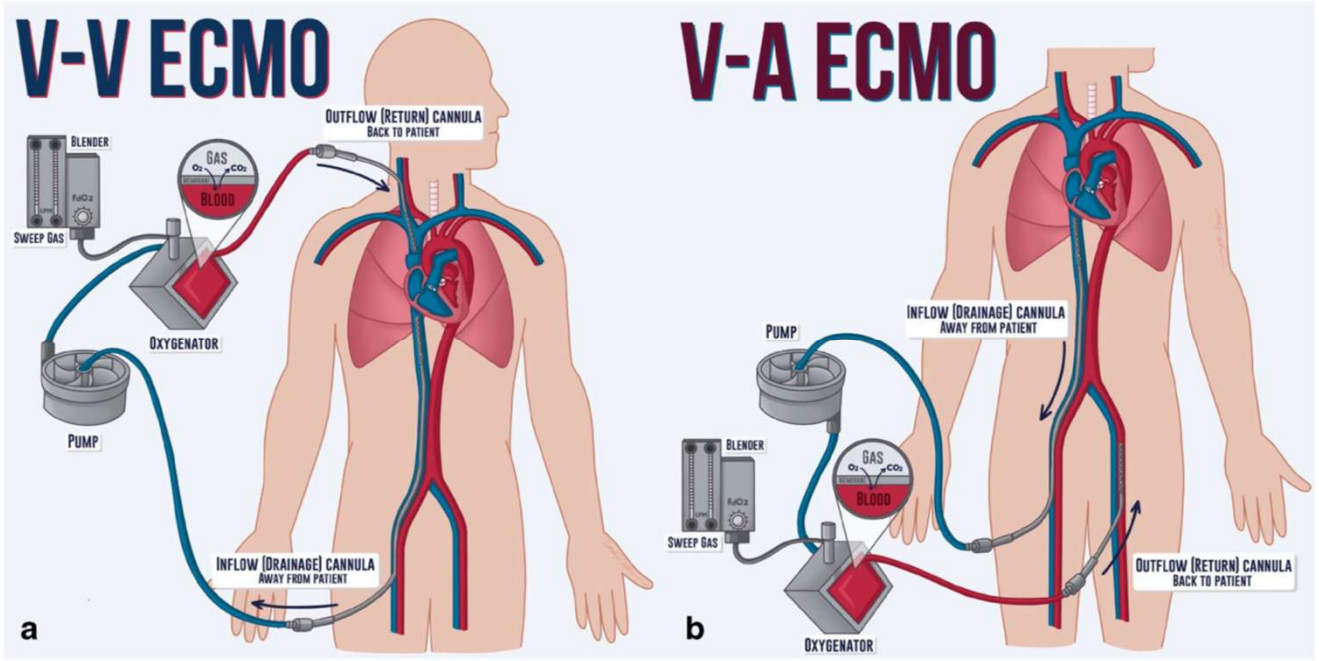


Fig. 2. (a) Veno-venous (V-V) ECMO with a duel site cannulation. Drainage cannula inserted into the right femoral vein and return

Review

Clinical review: Aggressive management and extracorporeal support for drug-induced cardiotoxicity

Frédéric J Baud[1], Bruno Megarbane[1], Nicolas Deye[1] and Pascal Leprince[2]

[1]Medical and Toxicological Intensive Care Unit, Assistance Publique-Hôpitaux de Paris, University Paris 7, Hôpital Lariboisière, 75010 Paris, France
[2]Department of Cardiovascular and Thoracic Surgery, Assistance Publique-Hôpitaux de Paris, University Paris 6, Hôpital Pitié Salpétrière, 75013 Paris, France

Clinical Toxicology (2013), **51**, 385–393

ISSN: 1556-3650 print / 1556-9519 online
DOI: 10.3109/15563650.2013.800876

REVIEW ARTICLE

Extracorporeal membrane oxygenation in the treatment of poisoned patients

D. W. DE LANGE[1,2,3], M. A. SIKMA[1,2], and J. MEULENBELT[1,2,4]

The organ support that can be applied with ECMO makes it especially useful in patients with severe poisoning as the clinical impact of the intoxication is often temporary; ECMO can be used as a **'bridge to recovery'.**

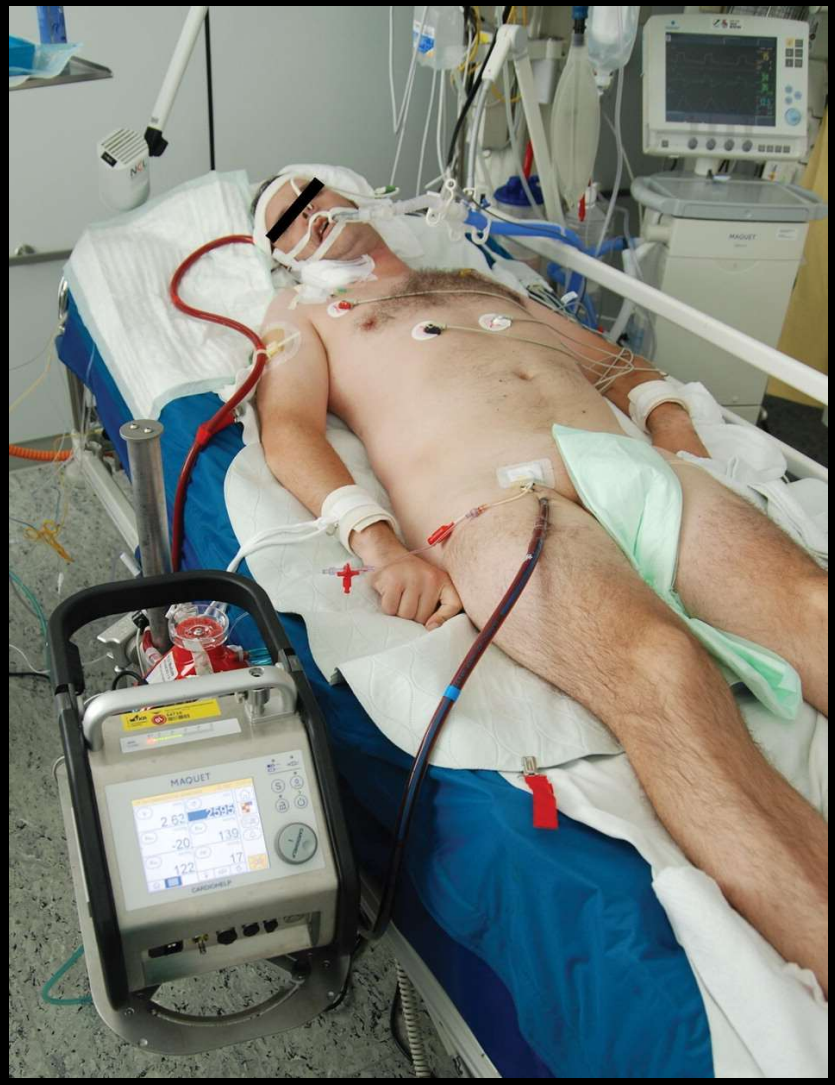
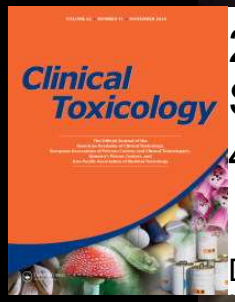


Table 15. Therapy Provided in Human Exposures by Age

Therapy	≤ 5 y	6-12 y	13-19 y	≥ 20 y	Unknown child	Unknown adult	Unknown age	Total
ECMO	7	7	48	122	0	0	1	185



2023 Annual Report of the National Poison Data System® (NPDS) from America's Poison Centers®: 41st Annual Report

David D. Gummin, James B. Mowry, Michael C. Beuhler, Daniel A. Spyker,

Hutin A, Loosli F, Lamhaut L, et al. How Physicians Perform Prehospital ECMO on the Streets of Paris. Journal of Emergency Medical Service. On line: 20/1/2017.

Extracorporeal membrane oxygenation use in poisoning: a narrative review with clinical recommendations

Table 2. Comparison of survival among poisoned patients admitted at an ECMO-capable hospital who received VA ECMO for refractory shock or refractory cardiac arrest (ECPR) and patients admitted to a hospital without ECMO capabilities who received conventional treatment.

	No ECMO ($n=48$)		Received VA ECMO ($n=14$)		
	Refractory cardiac arrest ($n=7$)	Refractory shock ($n=41$)	Refractory cardiac arrest ($n=3$)	Refractory shock ($n=11$)	p-Value
Survival, n (%)	0 (0%)	23 (56%)	3 (100%)	9 (82%)	
Overall survival, n (%)	23 (48%)		12 (86%)		$p=0.02$

VA ECMO: venoarterial extracorporeal membrane oxygenation; ECPR: extracorporeal cardiopulmonary resuscitation.

Upchurch C, Clin Toxicol (Phila). 2021 Oct;59(10): 877-887.

Table 4. Survival and neurologic outcomes at discharge of all poisoned patients at an institution placed on VA ECMO for refractory shock, cardiac arrest with subsequent ROSC prior to ECMO and refractory shock, or ECPR for refractory cardiac arrest due to poisonings.

	VA ECMO for refractory shock ($n=10$)		ECPR for refractory cardiac arrest ($n=7$)	
	Cardiac arrest with ROSC ($n=2$)	Refractory shock ($n=8$)	In-hospital cardiac arrest ($n=6$)	Out-of-hospital cardiac arrest ($n=1$)
Survived, n (%)	2 (100%)	6 (75%)	5 (83%)	0 (0%)
Overall survival, n (%)	8 (80%)		5 (71%)	
CPC score for survivors	CPC = 1 ($n=6$) CPC = 2 ($n=2$)		CPC = 1 ($n=5$)	

VA ECMO: venoarterial extracorporeal membrane oxygenation; ECPR: extracorporeal cardiopulmonary resuscitation; ROSC: return of spontaneous circulation; CPC: Cerebral Performance Category.

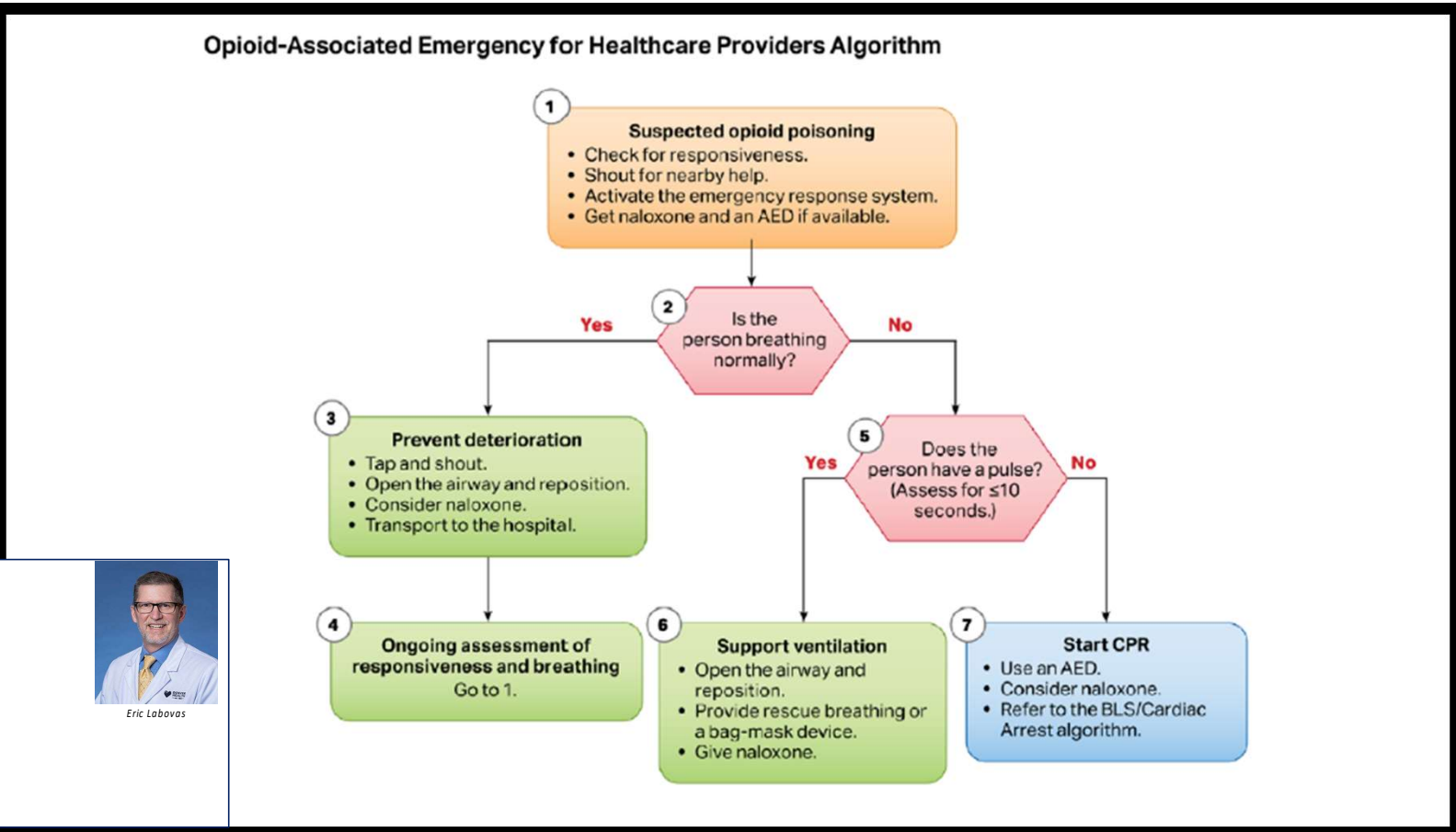
Positive Outcomes

Box. Cerebral Performance Category (CPC) Scale

CPC 1: Full recovery or mild disability

CPC 2: Moderate disability but independent in activities of daily living

2. La administración de naloxona puede revertir el paro respiratorio, previniendo la progresión a paro cardíaco



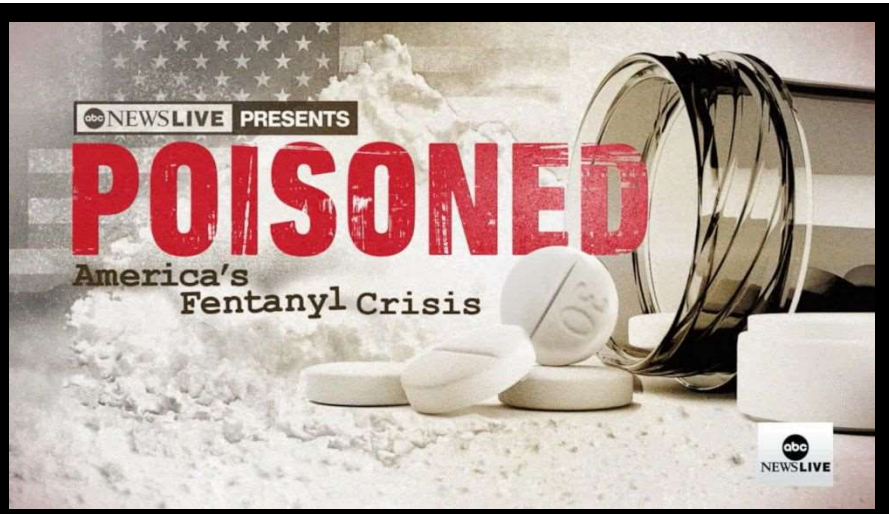
Circulation

2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

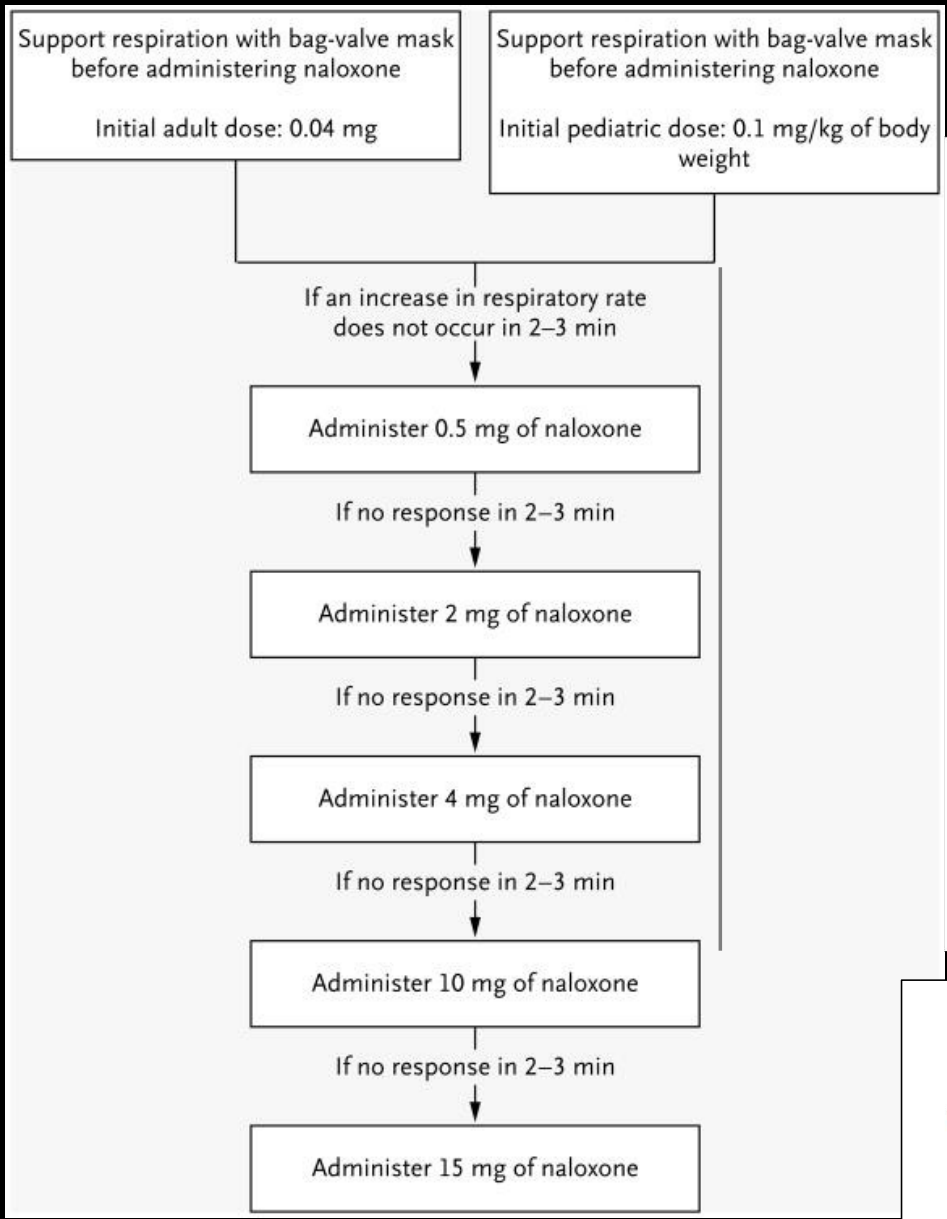
Eric Labovas

Table 15. Therapy Provided in Human Exposures by Age

Therapy	≤5 y	6-12 y	13-19 y	≥20 y	Unknown child	Unknown adult	Unknown age	Total
Naloxone	1,740	279	2,842	22,224	1	153	57	27,296


abc NEWSLIVE PRESENTS
POISONED
America's Fentanyl Crisis
abc NEWSLIVE


Overdose Rescue Kit
Equipo de Rescate para Sobredosis
NALOXONE HYDROCHLORIDE
INJ, USP
(1 mg/mL)



Publicado: 12/06/2023 | 18:38 | Actualizado 18:42 Notas Mundo

Compartir Nota

Escrito por: Marissa Espinosa Gutiérrez

Una máquina expendedora de salud pública con kits gratuitos de naloxona, tiras reactivas de fentanilo y xilazina, y paquetes de anticonceptivos de emergencia nusca hacerle frente a la crisis de opiáceos en Nueva York. | REUTERS

2005 No. 1507

MEDICINES

The Medicines for Human Use (Prescribing) (Miscellaneous Amendments) Order 2005

Made - - - - 6th June 2005

viernes, febrero 21, 2025

Trump declara la guerra a los cárteles: Busca designarlos como terroristas para combatir el fentanilo

PALCO NOTICIAS

3. Se recomienda tratamiento precoz con **dosis altas de insulina** en el tratamiento de intoxicación grave por **bloqueadores β y calcioantagonistas.**

Recommendations for the Management of Patients With Life-Threatening Beta Blocker Poisoning

COR	LOE	Recommendations
1	B-NR	1. We recommend that high-dose insulin be administered for hypotension due to β-blocker poisoning refractory to or in conjunction with vasopressor therapy.
1	C-LD	2. We recommend that vasopressors be administered for hypotension due to β-blocker poisoning.
2a	C-LD	3. It is reasonable to use a bolus of glucagon, followed by a continuous infusion, for bradycardia or hypotension due to β-blocker poisoning.
2a	C-LD	4. It is reasonable to utilize extracorporeal life support techniques such as VA-ECMO for life-threatening β-blocker poisoning with cardiogenic shock refractory to pharmacological interventions.
2b	C-LD	5. It may be reasonable to administer atropine for β-blocker–induced bradycardia.
2b	C-LD	6. It may be reasonable to attempt electrical pacing for β-blocker–induced bradycardia.
2b	C-LD	7. It may be reasonable to use hemodialysis for life-threatening atenolol or sotalol poisoning.
3: No Benefit	C-LD	8. Intravenous lipid emulsion therapy is not likely to be beneficial for life-threatening β-blocker poisoning.

Recommendations for the Management of Patients With Life-Threatening Calcium Channel Blocker Poisoning

COR	LOE	Recommendations
1	B-NR	1. We recommend administering vasopressors for hypotension from calcium channel blocker (CCB) poisoning.
1	B-NR	2. We recommend administering high-dose insulin for hypotension due to CCB poisoning.
2a	C-LD	3. It is reasonable to administer calcium for CCB poisoning.
2a	C-LD	4. It is reasonable to administer atropine for hemodynamically significant bradycardia from CCB poisoning.
2a	C-LD	5. It is reasonable to utilize extracorporeal life support techniques such as VA-ECMO for cardiogenic shock due to CCB poisoning that is refractory to pharmacological interventions.
2b	C-LD	6. It might be reasonable to attempt electrical pacing for CCB poisoning with refractory bradycardia.
2b	C-LD	7. The usefulness of a glucagon bolus and infusion for CCB poisoning is uncertain.
2b	C-LD	8. The usefulness of administering methylene blue for refractory vasodilatory shock due to CCB poisoning is uncertain.
3: No Benefit	C-LD	9. The routine use of intravenous lipid emulsion (ILE) therapy for CCB poisoning is not recommended.

INTOXICACIÓN POR CCA:

En intoxicaciones significativas la SELECTIVIDAD se pierde

Corazón: miocitos y nodos SA –VA: ↓↓ Inotropía y cronotropía → Bloqueo completo: **shock cardiogénico**

Musculatura lisa vascular: Vasodilatación periférica → **HIPOTENSIÓN / BRADICARDIAS / ARRITMIAS**

Páncreas: células beta: ↓↓ Liberación de insulina → **HIPERGLICEMIAS**

Grado hiperglicemia factor pronóstico de gravedad

About the Creator

Dr. Lewis Goldfrank

ADVICE

Give an initial insulin bolus to rapidly saturate insulin receptors to speed the physiological response.

- 1 unit/kg bolus of regular human insulin along with 0.5 g/kg bolus of dextrose.
- An infusion of regular insulin should immediately follow the bolus starting at 1 unit/kg/hr.

If blood glucose is greater than 250 mg/dL (16.7 mmol/L), then the dextrose bolus is not necessary.

MANAGEMENT

- Assess cardiac function every 10 to 15 minutes after starting HIET.
- If cardiac function remains depressed, then the insulin dose should be increased by 0.5-1 unit/kg increments.
- Dosing recommendations typically range up to 10 units/kg/hr; however, doses up to 22 units/kg/hr have been used, and the maximum dose is not established.
- Typical duration of therapy has been 1 to 2 days, although HIET has been used for up to 4 days.

BB dosis terapéuticas: **BB dosis tóxicas:**

ADRENALINA Y NORADRENALINA

Corazón (b1): Contracción y fuerza cardíaca → Bradicardia, ↓↓ demanda O2 → **HIPOTENSIÓN / BRADICARDIAS** **Shock cardiogénico**

Musculatura lisa vascular y vías respiratorias (b2): Vasoconstricción → Vasodilatación periférica → **Depresión respiratoria** **Broncoespasmo**

Hipoglicemia / Inhibición glucogenólisis y gluconeogénesis

3. Se recomienda tratamiento precoz con **dosis altas de insulina** en el tratamiento de intoxicación grave por **bloqueadores β y calcioantagonistas**.

Recommendations for the Management of Patients With Life-Threatening Beta Blocker Poisoning

COR	LOE	Recommendations
1	B-NR	1. We recommend that high-dose insulin be administered for hypotension due to β-blocker poisoning refractory to or in conjunction with vasopressor therapy.
1	C-LD	2. We recommend that vasopressors be administered for hypotension due to β-blocker poisoning.
2a	C-LD	3. It is reasonable to use a bolus of glucagon, followed by a continuous infusion, for bradycardia or hypotension due to β-blocker poisoning.
2a	C-LD	4. It is reasonable to utilize extracorporeal life support techniques such as VA-ECMO for life-threatening β-blocker poisoning with cardiogenic shock refractory to pharmacological interventions.
2b	C-LD	5. It may be reasonable to administer atropine for β-blocker–induced bradycardia.
2b	C-LD	6. It may be reasonable to attempt electrical pacing for β-blocker–induced bradycardia.
2b	C-LD	7. It may be reasonable to use hemodialysis for life-threatening atenolol or sotalol poisoning.
3: No Benefit	C-LD	8. Intravenous lipid emulsion therapy is not likely to be beneficial for life-threatening β-blocker poisoning.

Recommendations for the Management of Patients With Life-Threatening Calcium Channel Blocker Poisoning

COR	LOE	Recommendations
1	B-NR	1. We recommend administering vasopressors for hypotension from calcium channel blocker (CCB) poisoning.
1	B-NR	2. We recommend administering high-dose insulin for hypotension due to CCB poisoning.
2a	C-LD	3. It is reasonable to administer calcium for CCB poisoning.
2a	C-LD	4. It is reasonable to administer atropine for hemodynamically significant bradycardia from CCB poisoning.
2a	C-LD	5. It is reasonable to utilize extracorporeal life support techniques such as VA-ECMO for cardiogenic shock due to CCB poisoning that is refractory to pharmacological interventions.
2b	C-LD	6. It might be reasonable to attempt electrical pacing for CCB poisoning with refractory bradycardia.
2b	C-LD	7. The usefulness of a glucagon bolus and infusion for CCB poisoning is uncertain.
2b	C-LD	8. The usefulness of administering methylene blue for refractory vasodilatory shock due to CCB poisoning is uncertain.
3: No Benefit	C-LD	9. The routine use of intravenous lipid emulsion (ILE) therapy for CCB poisoning is not recommended.

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Original Contribution

High dose insulin for beta-blocker and calcium channel-blocker poisoning☆

Jon B. Cole, M.D. [a,*], Ann M. Arens, M.D. [a], JoAn R. Laes, M.D. [b], Lauren R. Klein, M.D. [c], Stacey A. Bangh, PharmD [d], Travis D. Olives, M.D., M.P.H., M.Ed. [a]

199 patients

Median age was **48 years** (range 14–89); 50% were male.

Eighty-eight patients **(44%)** were poisoned by **BBs, 66 (33%) by CCBs, and 45 (23%) by both.**

Fortyone patients (21%) experienced cardiac arrest; 31 (16%) died.

Median insulin bolus was 1 U/kg (range, 0.5–10). median peak infusion was 8 U/kg/h (range 0.5–18).

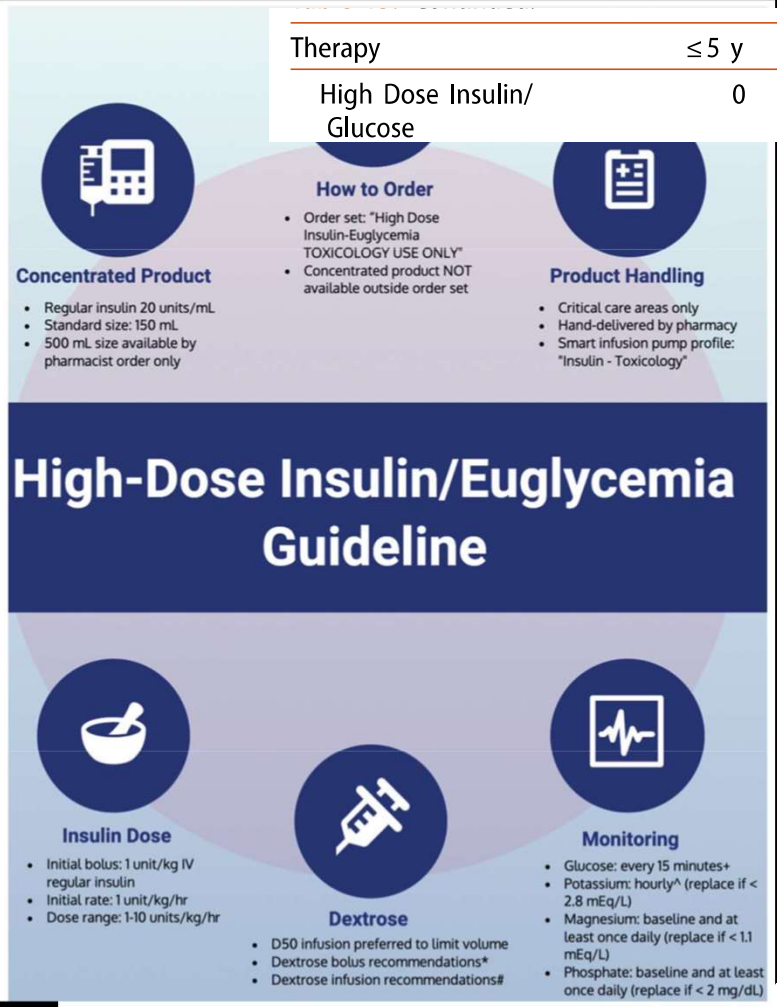
Hypokalemia occurred in 29% of patients.

Hypoglycemia occurred in 31% of patients; 50% (29/50) experienced hypoglycemia when dextrose infusion concentration ≤10%, and 30% (31/105) experienced hypoglycemia when dextrose **infusion concentration ≥20%.**

- Check baseline creatinine, glucose, and potassium. Replace potassium if hypokalemia is present.
- Administer 25-50 g of dextrose 50% (D50) IV if blood glucose < 200 mg/dL.
- Administer 1 U/kg of regular insulin IV push.
- Begin infusion of 1 U/kg/hr of regular insulin along with an infusion of dextrose; use D20 via a peripheral IV line until central access has been obtained, then start D50 at 25 g/hr. Initial effects of HDI take at least 5-10 minutes to manifest.
- Obtain central venous access for safe infusion of concentrated glucose.
- Concentrate all fluids to avoid pulmonary edema. Consider using stock D70 as the glucose infusion. The insulin infusion should be concentrated to 10 U/mL.
- Increase insulin infusion 1-2 U/kg/hr every 10-15 minutes to a maximum of 10 U/kg/hr until shock improves. Doses up to 22 U/kg/hr have been safely used.
- Monitor blood glucose every 10-15 minutes until stable, then every 1-2 hour after steady state is reached. For every hypoglycemic reading, give 25 g of D50 as a bolus and increase the dextrose infusion by 25 g/hr.
- Monitor serum potassium every hour during titration, then every 4-6 hours once steady state is reached.
- Regarding electrolytes, maintain serum potassium ≥ 2.8 mEq/L and calcium 1.5-2 times the upper limit of normal. Monitor magnesium and phosphorus every 4-6 hours and replace as needed. We recommend standard ICU magnesium and phosphorus replacement protocols.
- Once stable, wean vasopressors before HDI. Contact the Poison Center regarding weaning of HDI once stability has been reached.

Therapy	≤ 5 y	6-12 y	13-19 y	≥ 20 y	Unknown child	Unknown adult	Unknown age	Total
High Dose Insulin/ Glucose	0	6	44	635	0	2	1	688

2023 Annual Report of the National Poison Data System® (NPDS) from America's Poison Centers®: 41st Annual Report



CLINICAL TOXICOLOGY, 2016
http://dx.doi.org/10.1080/15563650.2016.1209766

Taylor & Francis
Taylor & Francis Group

BASIC RESEARCH

Stability of high-dose insulin in normal saline bags for treatment of calcium channel blocker and beta blocker overdose

Dayne Laskey[a], Rajesh Vadlapatla[b] and Katherine Hart[c,d]

[a]University of Saint Joseph School of Pharmacy, Hartford, CT, USA; [b]Marshall B. Ketchum University College of Pharmacy, Fullerton, CA, USA; [c]Hartford Hospital, Hartford, CT, USA; [d]Connecticut Poison Control Center, Farmington, CT, USA

Objective: To determine the stability of insulin at 16 units/mL in 0.9% saline solution.
Materials and methods: Eight-hundred units of regular insulin (8 mL from a stock vial containing 100 units/mL) were added to 42 mL of 0.9% saline solution in a polyvinyl chloride bag to make a final concentration of 16 units/mL. Two bags were stored at 4 °C (refrigerated) and two at 25 °C (room temperature). Samples were withdrawn and tested for insulin concentration periodically over 14 days.
Results: Concentrated regular insulin in a polyvinyl chloride bag remained within 90% of equilibrium concentration at all time points, indicating the 16 units/mL concentration was sufficiently stable both refrigerated and at room temperature for 14 days.
Discussion: Administration of high-dose insulin can cause fluid volume overload when using traditional insulin formulations. The 16 units/mL concentration allows for the treatment of a patient with severe calcium channel blocker or beta blocker toxicity for a reasonable period of time without administering excessive fluid.
Conclusion: ...nits/mL is stable for 14 days, the maximum timeframe currently allo... for compounding of sterile preparations. This stability data will allo... dating for intravenous fluids containing concentrated insulin and u... alcium channel blocker toxicity.

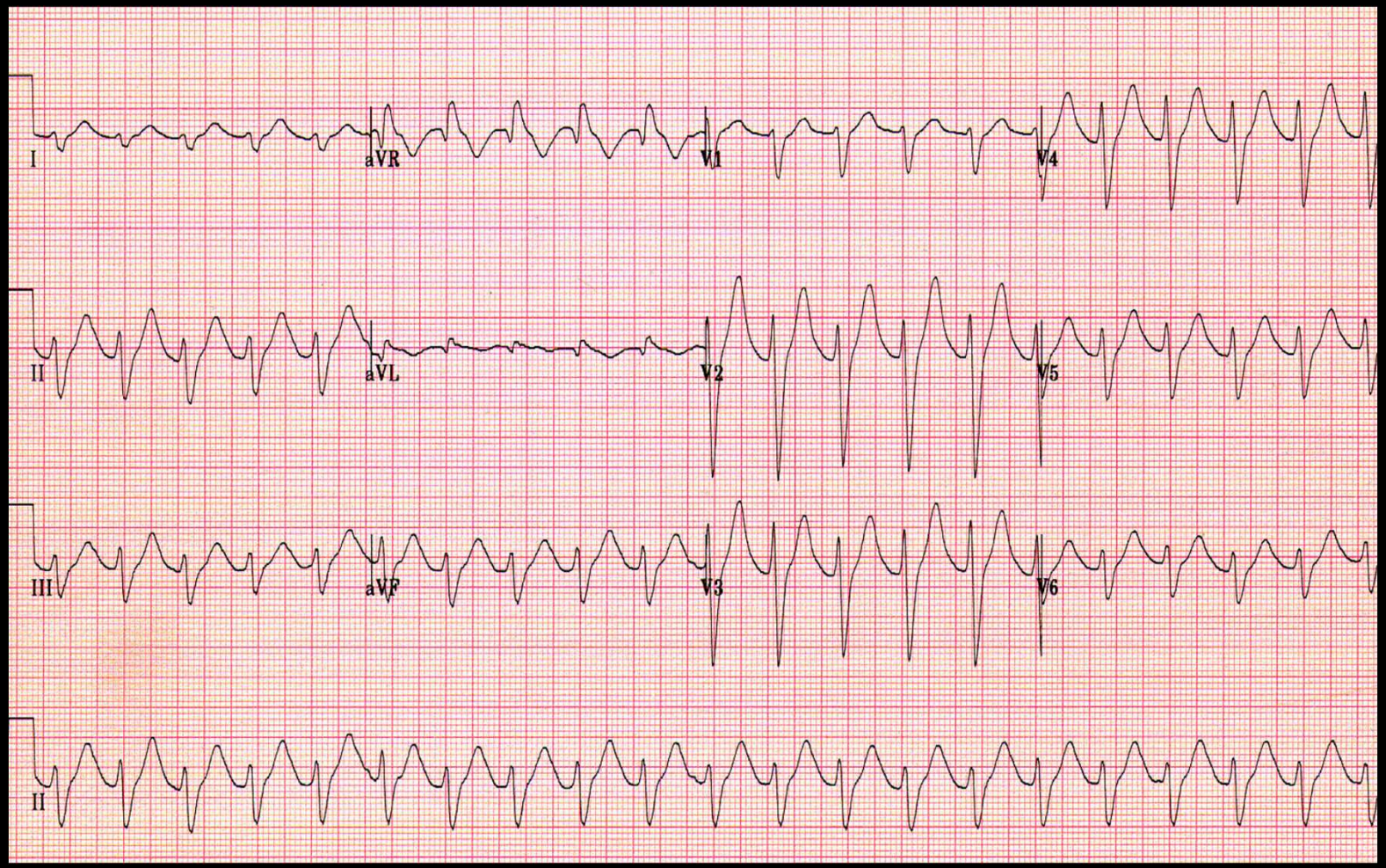
Concentraciones:
10 - 16 – 20 UI/ml

Preparación:
800 UI (8 mL) insulina regular + 42 ml SF

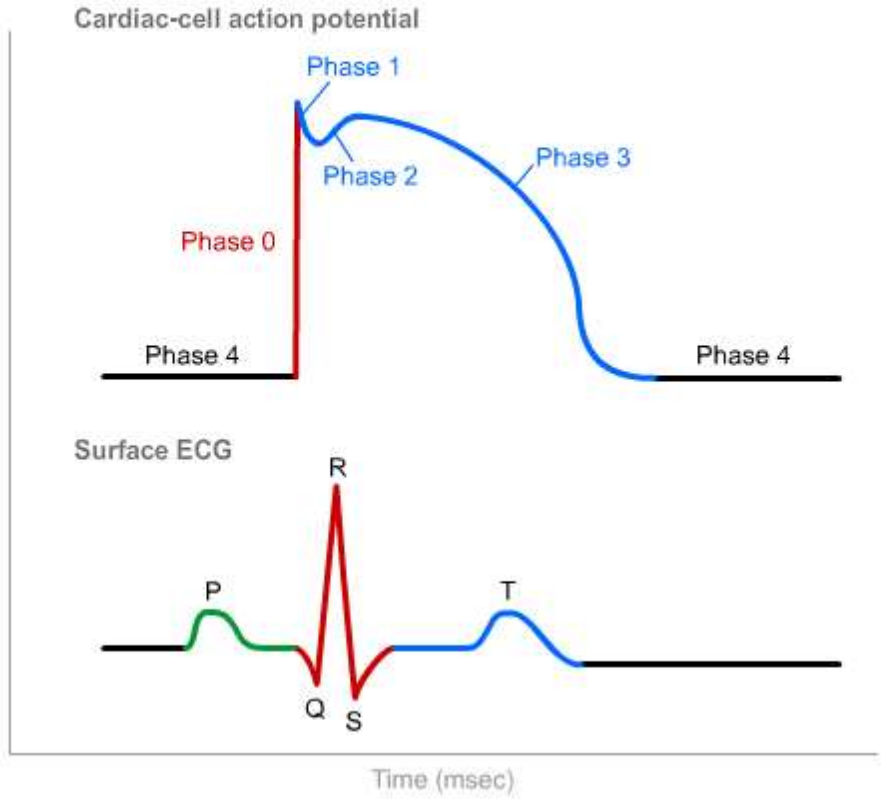
Concentración: 16 UI/ml

Estabilidad:
14 días en nevera o temperatura ambiente

4. El soporte vital avanzado debe contemplar la administración de bicarbonato sódico en el tratamiento de arritmias graves y mortales causadas por bloqueadores de los canales de sodio.



◉ **Normal conduction** ○ Sodium-channel blockade ○ Potassium-channel blockade ○ Nodal blockade



Normal conduction

Phases of ventricular action potential

Phase 4: The sodium–potassium (Na^+/K^+–ATPase) pump maintains the resting membrane potential.

Phase 0: Fast sodium channels open to allow an influx; depolarization of the cell membrane occurs and is represented by the QRS complex on ECG.

Phase 1: Sodium channels close and potassium efflux channels open, polarizing the cell and allowing voltage-gated calcium channels to open.

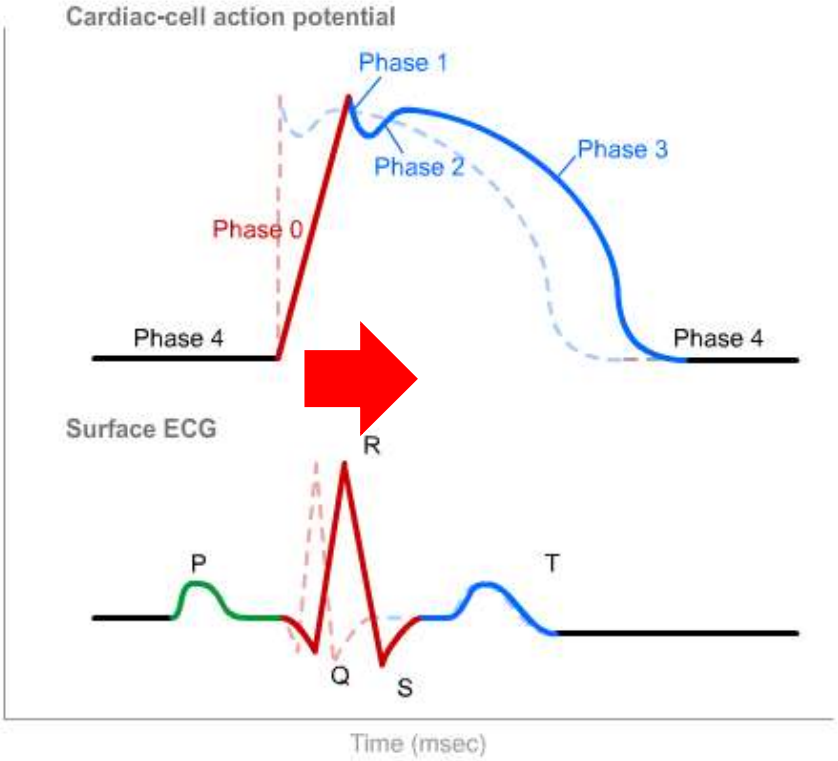
Phase 2: Calcium-channel activation permits calcium influx and a sustained myocardial contraction.

Phase 3: With persistent potassium efflux, calcium channels close to facilitate the repolarization of myocardial cells, which restores their resting potential, an action that corresponds with the QT interval on ECG.

The changes in conduction shown have been exaggerated for the purposes of instruction.

The NEW ENGLAND JOURNAL of MEDICINE

○ Normal conduction ◉ **Sodium-channel blockade** ○ Potassium-channel blockade ○ Nodal blockade



Sodium-channel blockade

Representative agents
Class IA antiarrhythmics (e.g., quinidine, procainamide)
Class IC antiarrhythmics (e.g., flecainide, propafenone)
Tricyclic antidepressants

Effect
Delayed influx of sodium ions and slowing of depolarization, which slows conduction

Action potential
Decreased slope in phase 0

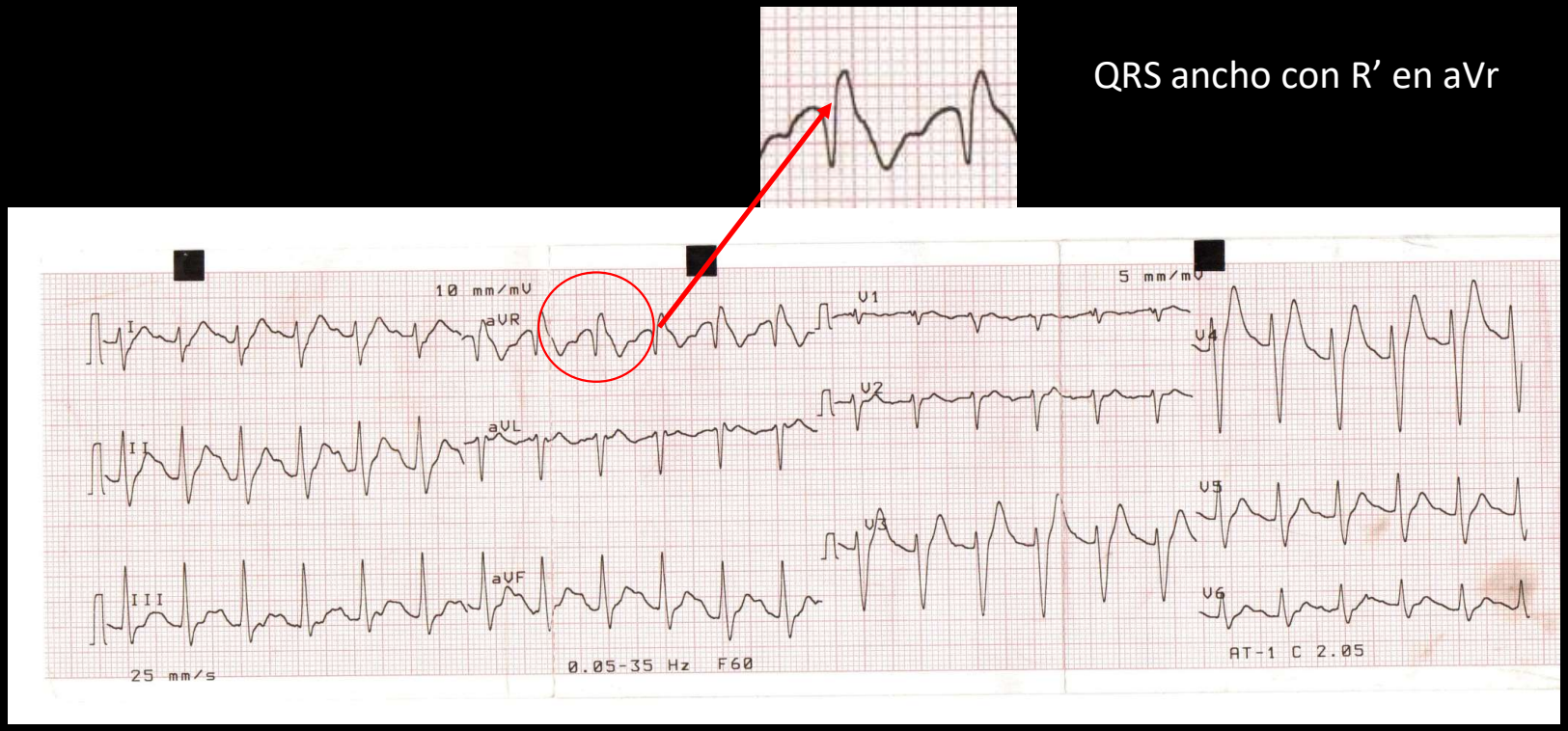
Pattern on ECG
Widening of the QRS complex

Complications
Bundle-branch block, heart block, and degeneration into ventricular tachycardia and fibrillation, especially in patients with concurrent heart disease

The changes in conduction shown have been exaggerated for the purposes of instruction.

The NEW ENGLAND JOURNAL of MEDICINE

- ✓ **ANTIDEPRESIVOS CICLICOS.**
- ✓ **CARBAMAZEPINA Y DERIVADOS.**
- ✓ **LAMOTRIGINA.**
- ✓ **FENITOINA.**
- ✓ **GABAPENTINA.**
- ✓ **LIDOCAINA.**
- ✓ **ANESTESICOS LOCALES**



Ingesta de 2 blisters de amitriptilina con síntomas anticolinérgicos

Taquicardia

QRS 120 msg

BRD

onda r’ en aVR “prominente”

Caravati EM. The electrocardiograma as a diagnostic discriminator for acute trcyclic antidepressant poisoning.
J Toxicol Clin Toxicol 1999;37:113-5.

R_{AVR} >3mm ES

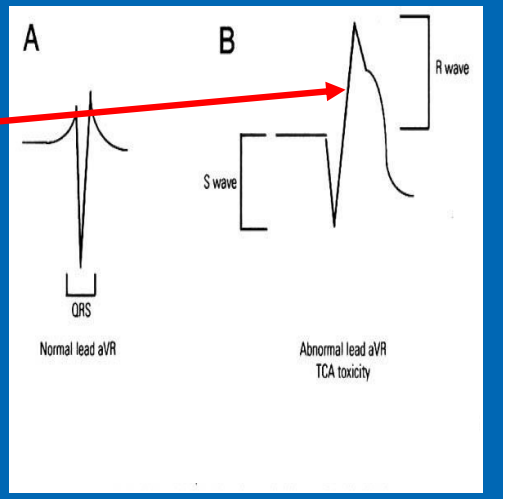
PREDI CTOR DE

ARRI TMI AS Y

CONVULSI ONES (VP+

46%, Sensibilidad 81%).

Niemann J T. Am J Cardiol 1986
Liebelt EL. Ann Emerg Med 1995.



Recommendations for the Treatment of Patients With Life-Threatening Sodium Channel Blocker Poisoning

COR	LOE	Recommendations
1	B-NR	1. We recommend using sodium bicarbonate to treat life-threatening cardiotoxicity from tricyclic and/or tetracyclic antidepressant poisoning.
2a	C-LD	2. It is reasonable to use sodium bicarbonate to treat life-threatening cardiotoxicity caused by poisoning from sodium channel blockers other than tricyclic or tetracyclic antidepressants.
2a	C-LD	3. It is reasonable to use extracorporeal life support, such as VA-ECMO, to treat refractory cardiogenic shock from sodium channel blocker poisoning.
2b	C-LD	4. It may be reasonable to use Vaughan-Williams class Ib antidysrhythmics (eg, lidocaine) to treat life-threatening cardiotoxicity from class Ia or Ic sodium channel blockers.

Bloqueantes de canales de Na^+ voltaje dependiente:

- Bicarbonato 1 M.
  - Dosis inicial: 1 mEq/Kg en 3 min.
  - Mantenimiento: 150 mEq en solución de 1-3 mL/Kg/h.
  - Objetivo: pH 7,50-7,55.
  - **OJO**: hipernatremia, alcalosis, hipokalemia e hipocloremia.

5. Si sospecha envenenamiento por cianuro, no espere las pruebas de confirmación. Trate inmediatamente con hidroxocobalamina (preferido) o nitrito de sodio más tiosulfato de sodio.

asphyxia, may impede the victims' escape.[7] So may

From Réanimation Toxicologique (F.J.B., C.B.), the Département de Biophysique (E.V.), and the Laboratoire de Toxicologie (R.B.), Hôpital Fernand Widal, Université Paris 7, Paris; the Service Médical de la Brigade des Sapeurs-Pompiers de Paris, Paris (P.B.); the Laboratoire de Toxicologie, Hôpital Henri Mondor, Créteil (V.T., A.A.); the Département d'Anesthésie-Réanimation, Hôpital Pitié-

men, ranging in age from 2 to 87 years (mean, 33). Blood specimen were collected in dry heparin by the first medical squad to reach th scene after the start of isobaric oxygen therapy in all patients wh were still alive and the start of mechanical ventilation in some, bu before the administration of hydroxocobalamin (an antidote to cya nide) and any hyperbaric oxygen therapy. Thirty-six of these pa tients had already died at the scene of the fire when the bloo sample was collected. Two or three blood samples were collected

Recommendations for the Management of Patients With Life-Threatening Cyanide Poisoning

COR	LOE	Recommendations
1	C-LD	1. We recommend that hydroxocobalamin be administered for cyanide poisoning.
1	C-LD	2. We recommend that sodium nitrite be administered for cyanide poisoning when hydroxocobalamin is unavailable.
2a	C-LD	3. In addition to administering hydroxocobalamin or sodium nitrite, it is reasonable to administer sodium thiosulfate for cyanide poisoning.
2a	C-EO	4. It is reasonable to administer 100% oxygen for cyanide poisoning.

Los **criterios de Baud**, que sugieren la existencia de CNH son:

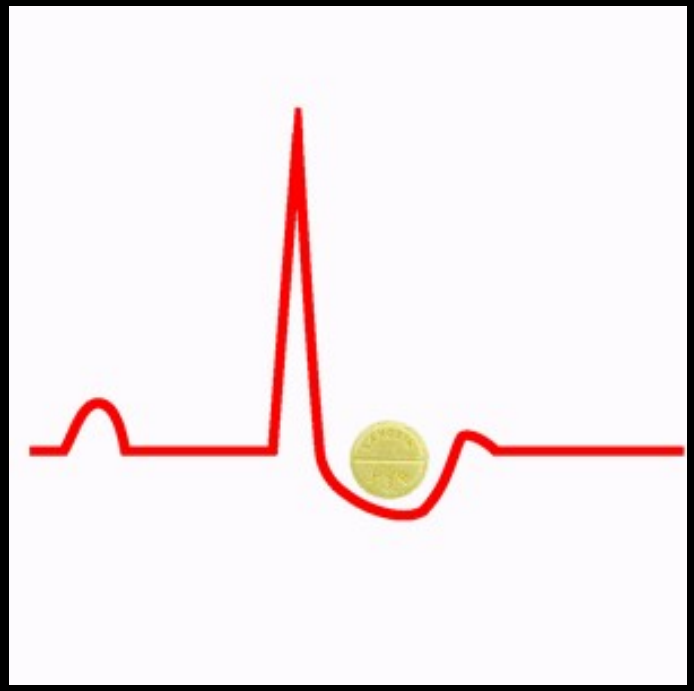
1 Inhalación de humo de incendio en un lugar cerrado, con alta temperatura y combustión de productos sintéticos nitrogenados (indican la presencia de CNH)

y

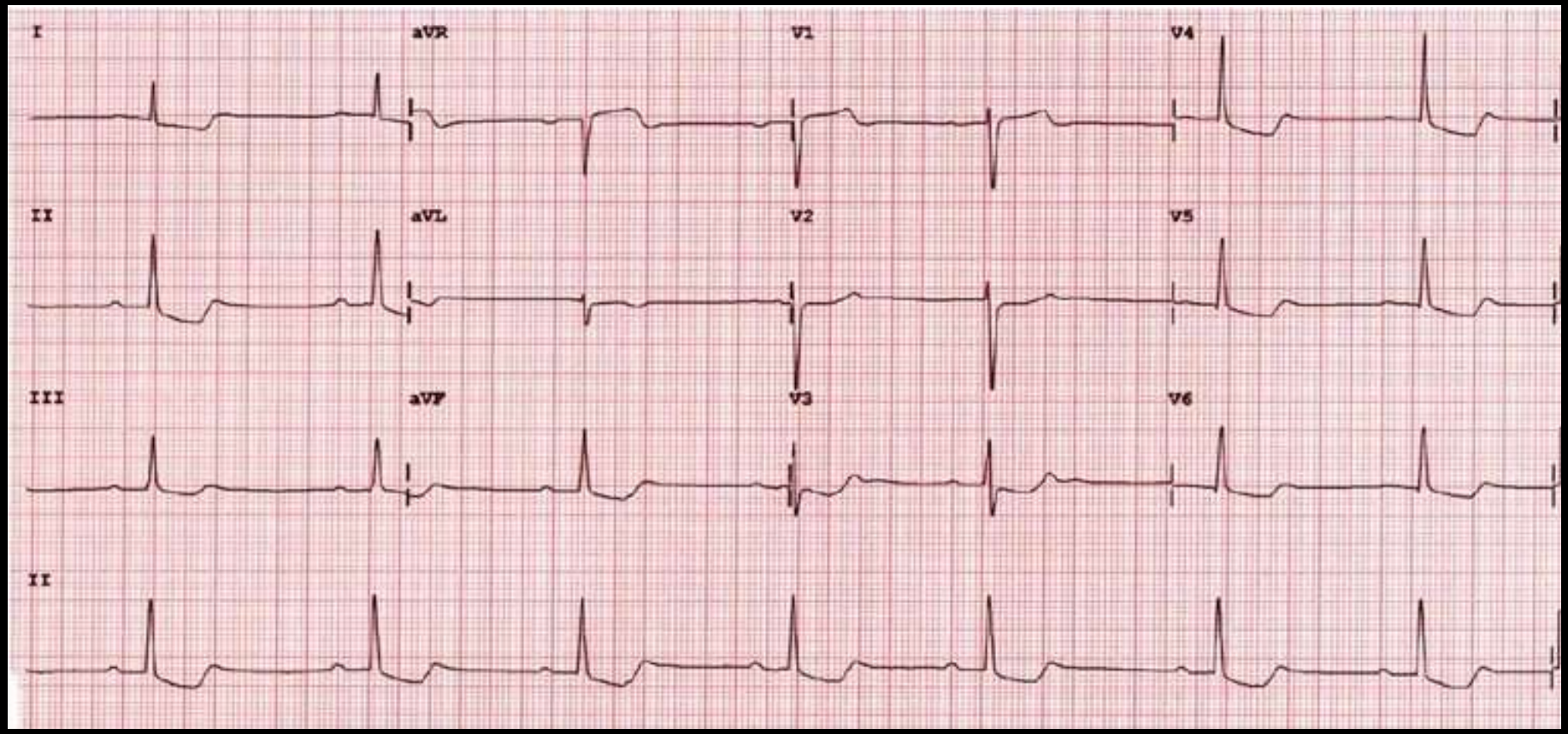
2. disminución del nivel de conciencia, hipotensión arterial, acidosis metabólica, presencia de hollín en boca y fosas nasales

3. Además, elevación del **lactato ≥ 8 mmol/L** sugestiva de intoxicación por CNH.

6. La administración de **FabAD** puede revertir arritmias potencialmente mortales por envenenamiento por digoxina.



Recommendations for the Management of Patients With Life-Threatening Poisoning From Digoxin and Related Cardiac Glycosides		
COR	**LOE**	**Recommendations**
1	**B-NR**	1. We recommend administration of digoxin-specific antibody fragments (digoxin-Fab) for digoxin or digitoxin poisoning.
2a	**C-LD**	2. It is reasonable to administer digoxin-Fab for poisoning due to *Bufo* toad venom and yellow oleander.
2b	**C-LD**	3. It may be reasonable to administer digoxin-Fab to treat poisoning from cardiac glycosides other than digoxin, digitoxin, *Bufo* toad venom, and yellow oleander.
2b	**C-LD**	4. It may be reasonable to administer atropine for bradydysrhythmias caused by digoxin and other cardiac glycoside poisoning.
2b	**C-LD**	5. It may be reasonable to attempt electrical pacing to treat bradydysrhythmias from digoxin and other cardiac glycoside poisoning.
2b	**C-LD**	6. It may be reasonable to administer lidocaine, phenytoin, or bretylium to treat ventricular dysrhythmias caused by digitalis and other cardiac glycoside poisoning until digoxin-Fab can be administered.
3: No Benefit	**B-NR**	7. We do not recommend the use of hemodialysis, hemofiltration, hemoperfusion, or plasmapheresis to treat digoxin poisoning.



Impregnación digitálica **no equivale** a intoxicación digitálica

Ix digitálica

ORIGINAL

Características de las intoxicaciones por digoxina atendidas en diversos servicios de urgencias españoles en función del tipo de intoxicación y de la administración de anticuerpos antidigoxina: estudio DIGITOX

August Supervía[1-4], Andrea Martínez Baladrón[5], Francisca Córdoba[3,4,6], Francisco Callado[7], Victoria Lobo Antuña[8], Jordi Puiguriguer[4,9], Elena Fuentes[3,10], Valle Molina Samper[11], Antonio F. Caballero-Bermejo[12], Susana Vert[3,13], Francisco Ruíz-Ruíz[14], F. Javier Guijarro Eguinoa[15], Beatriz Martín-Pérez[16], Samuel Olmos[3,17], Guillermo Burillo-Putze[4,18], María Teresa Maza Vera[5], Oriol Pallàs[1,2], Benjamín Climent[8], Maider Igartua Astibia[11], Edith Gutiérrez[12], Santiago Nogué[3,4], Ana Ferrer Dufol[4]

Emergencias 2023;35:328-334

Tabla 1. Manifestaciones electrocardiográficas en las intoxicaciones por digoxina según el tipo

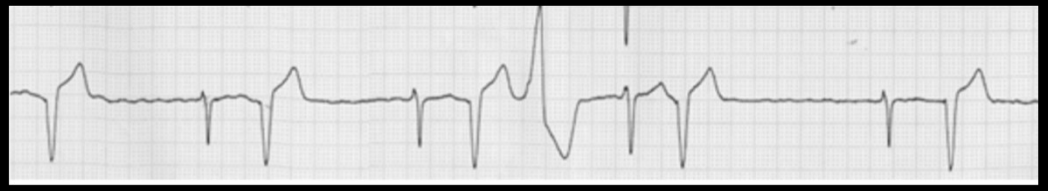
	Aguda N = 27 n (%)	Crónica N = 631 n (%)	p
Auriculares rápidas	1 (3,7)	13 (2,1)	ns
Fibrilación auricular	1 (3,7)	10 (1,6)	
Flutter auricular	0	2 (0,3)	
Taquicardia auricular	0	1 (0,2)	
Auriculares lentas	12 (44,4)	348 (55,2)	ns
Bradicardia sinusal	0	16 (2,5)	
BAV primer grado	1 (3,7)	6 (0,9)	
BAV segundo grado	0	5 (0,8)	
BAV completo	1 (3,7)	7 (1,1)	
FA lenta	6 (22,2)	248 (39,3)	
FA bloqueada	0	4 (0,6)	
Ritmo nodal	2 (7,4)	17 (2,7)	
Ventriculares	2 (7,4)	16 (2,5)	**0,009**
Bigeminismo	0	2 (0,3)	
Extrasistolia	0	9 (1,4)	
Taquicardia ventricular	1 (3,7)	2 (0,3)	
Fibrilación ventricular	1 (3,7)	2 (0,3)	
Asistolia	2 (7,4%)	3 (0,5%)	**< 0,001**

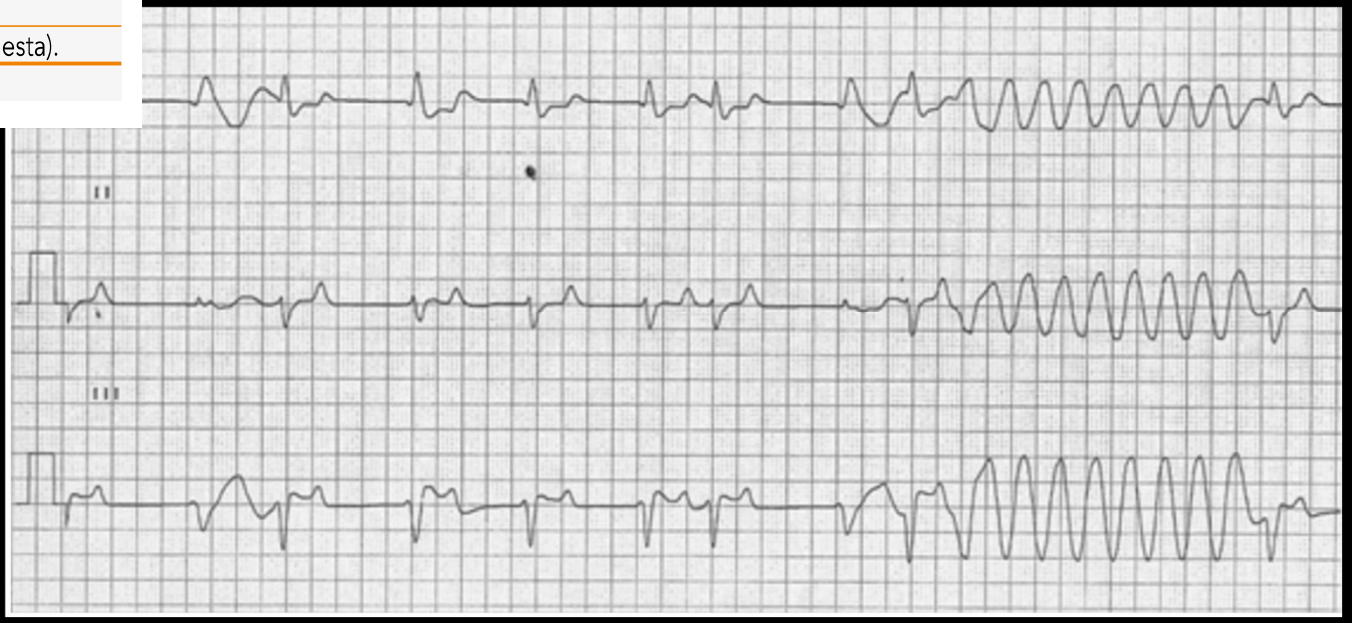
BAV: bloqueo auriculoventricular; FA: fibrilación auricular; ns: no significativo. Los valores en negrita denotan significación estadística ($p < 0,05$).

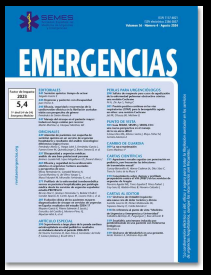
Tabla 1. Situaciones de riesgo vital asociadas a toxicidad digitálica y con indicación de anticuerpos antidigital

– Bradiarritmia con frecuencia ventricular < 40 lat/min y que no responde (mantiene frecuencia ventricular < 60 lat/min) a dosis repetidas de 0,5 mg/iv de atropina (hasta un máximo de 2 mg).
– Extrasistolia ventricular con riesgo de taquicardia o fibrilación ventricular (extrasistolia ventricular frecuente, dupletes, tripletes, multifocales o con fenómeno de R sobre T).
– Taquicardia ventricular.
– Fibrilación ventricular.
– Asistolia.
– *Shock* cardiogénico.
– Kaliemia > 5 meq/L con presencia de otros signos de toxicidad digitálica, en intoxicación aguda.
– Concentración plasmática de digoxina > 6 ng/ml (> 6 horas postingesta).

Reproducida de Nogué S, et al. Emergencias. 2012;24:462-75.







Emergencias 2012; 24: 462-475

Tratamiento de la intoxicación digitálica. Bases para el uso de los anticuerpos antidigital

SANTIAGO NOGUÉ[1], JUAN CINO[2], EMILIA CIVEIRA[3], JORDI PUIGURIGUER[4], GUILLERMO BURILLO-PUTZE[5], ANTONIO DUEÑAS[6], DOLORS SOY[7], RAQUEL AGUILAR[8], NÚRIA COROMINAS[7]

Tabla 5. Estimación de la carga corporal de digoxina (CCTD) en base a la concentración plasmática y una vez alcanzado el estado de equilibrio estacionario

Ejemplo de edad y peso	Fórmula para el cálculo de la CCTD	Ejemplo de concentración plasmática	Ejemplo de CCTD	Ejemplo de necesidad de AcAD*
Adulto (67 Kg)	[Concentración plasmática de digoxina en ng/mL] x [Volumen de distribución aparente en L/Kg] x [Peso en Kg]	6 ng/mL	6 x 5 x 67 = 2 mg	160 mg (4 viales)
Niño (16,7 Kg)	[Concentración plasmática de digoxina en ng/mL] x [Volumen de distribución aparente en L/Kg] x [Peso en Kg]	6 ng/mL	6 x 5 x 16,7 = 0,5 mg	40 mg (1 vial)

*La necesidad teórica de AcAD (anticuerpos antidigital) se calcula en base a que 40 mg de AcAD neutralizan 0,5 mg de digoxina.

Tabla 4. Estimación de la carga corporal total de digoxina (CCTD) en base a la dosis ingerida en la intoxicación aguda por digoxina

Fórmula para el cálculo de la CCTD	Ejemplo de dosis ingerida	Ejemplo de CCTD
[Nº comp] x [mg/comp] x [Biodisponibilidad]*	25 comp de 0,25 mg	25 x 0,25 x 0,8 = 5 mg

*La biodisponibilidad de la digoxina (80% de la dosis ingerida) es independiente del peso y edad del paciente.

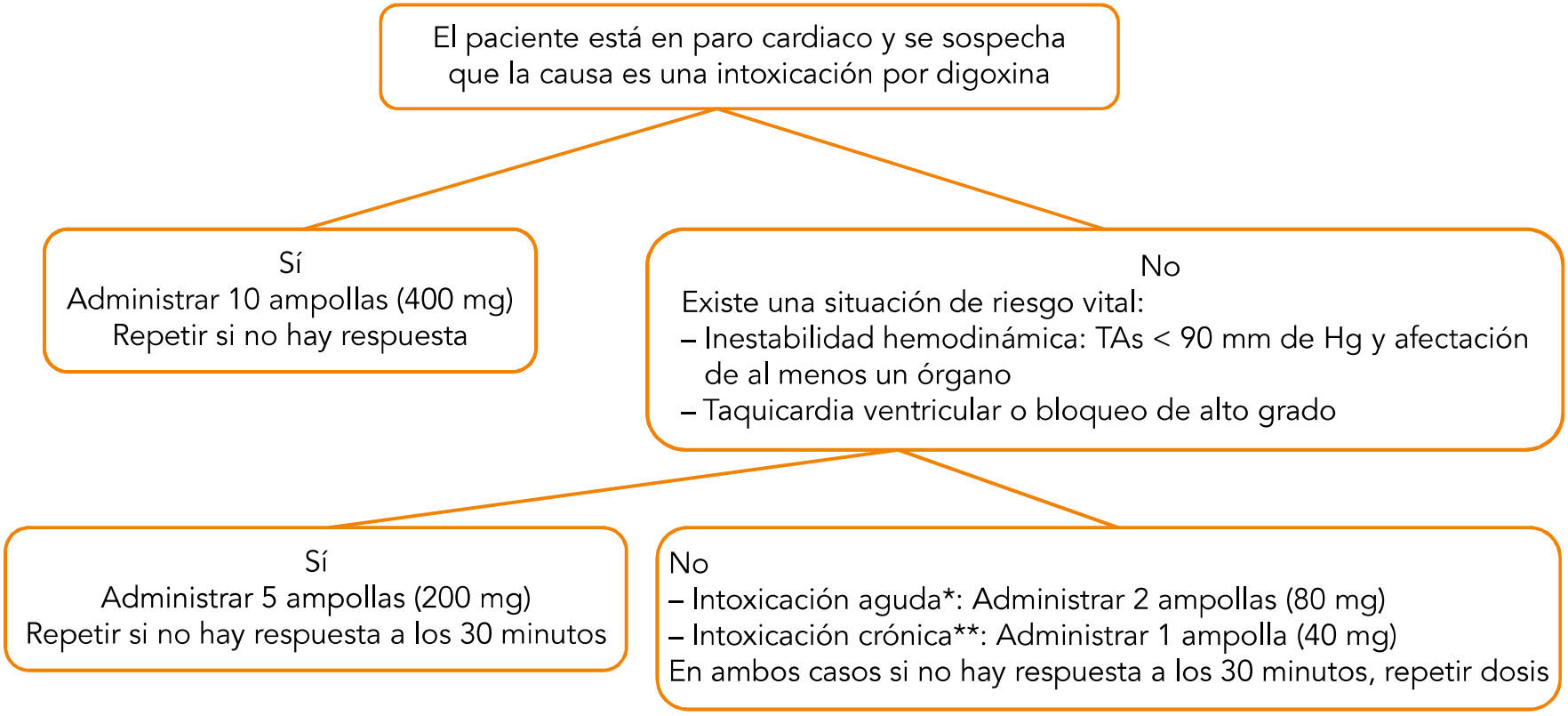


Figura 3. Propuesta de dosificación de anticuerpos antidigoxina en pacientes con intoxicación por digoxina y que reúnen criterios para indicar este antídoto.

Supervía A, et al. Rev Esp Urg Emerg. 2024;3:90-95

7. El uso de **ELI al 20%** puede ser eficaz en la reanimación de la toxicidad de los anestésicos locales.

Recommendations for the Management of Patients With Life-Threatening Local Anesthetic Poisoning

COR	LOE	Recommendations
1	C-LD	1. We recommend the administration of intravenous lipid emulsion for local anesthetic poisoning.
1	C-LD	2. We recommend the use of benzodiazepines to treat seizures associated with local anesthetic systemic toxicity.
2a	C-LD	3. It is reasonable to administer sodium bicarbonate for life-threatening wide-complex tachycardia associated with local anesthetic toxicity.
2a	C-EO	4. It is reasonable to administer atropine for life-threatening bradycardia associated with local anesthetic systemic toxicity.
2a	C-EO	5. It is reasonable to utilize extracorporeal life support techniques such as VA-ECMO in local anesthetic toxicity with refractory cardiogenic shock.

LipidRescue™ Resuscitation

20% lipid emulsion for rescue from drug toxicity

Initial Focus

- Airway management: ventilate with 100% oxygen
- Seizure suppression: benzodiazepines are preferred
- Basic and Advanced Cardiac Life Support (BLS/ACLS) may require prolonged effort

Infuse 20% Lipid Emulsion (values in parenthesis are for a 70 kg patient)

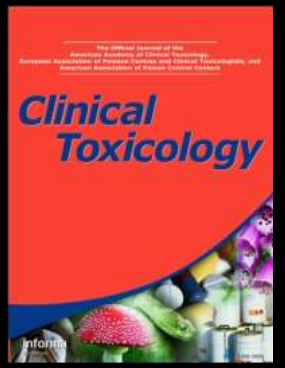
- Bolus 1.5 mL/kg (lean body mass) intravenously over 1 min (~100 mL)
- Continuous infusion at 0.25 mL/kg/min (~18 mL/min; adjust by roller clamp)
- Repeat bolus once or twice for persistent cardiovascular collapse
- Double the infusion rate to 0.5 mL/kg per minute if blood pressure remains low
- Continue infusion for at least 10 mins after attaining circulatory stability
- Recommended upper limit: approximately 10-12 mL/kg lipid emulsion over the first 30 mins

Avoid vasopressin, calcium channel blockers, β-blockers, or local anesthetic

Evidence-based recommendations on the use of intravenous lipid emulsion therapy in poisoning

Sophie Gosselin, Lotte C. G. Hoegberg, Robert. S. Hoffman, Andis Graudins,

CLINICAL TOXICOLOGY, 2016
VOL. 54, NO. 10, 899–923
http://dx.doi.org/10.1080/15563650.2016.1214275



- **For the management of life-threatening toxicity**, (1) as first line therapy**, we suggest not to use ILE** with toxicity from amitriptyline, non-lipid soluble beta receptor antagonists, bupropion, calcium channel blockers, cocaine, lamotrigine, diphenhydramine, malathion but are neutral for other toxins,

Conclusion: Clinical recommendations regarding the use of ILE in poisoning were only possible in a small number of scenarios and were based mainly on very low quality of evidence, balance of expected risks and benefits, adverse effects, laboratory interferences as well as related costs and resources. The workgroup emphasizes that dose-finding and controlled studies reflecting human poisoning scenarios are required to advance knowledge of limitations, indications, adverse effects, effectiveness, and best regimen for ILE treatment.

LA ÚLTIMA BALA

Red de Antídotos

redantidotos.org

ACCESO PRIVADO

RED DE ANTÍDOTOS

Quiénes somos | La Red | Centros | Guía | Fuentes información | Consultas | Formación | Noticias

EMULSIÓN LIPÍDICA INTRAVENOSA (ELI)

DESCARGAR PDF

PRESENTACIONES HABITUALES

Solución al 20% envase de 100 mL, 250 mL y 500 mL (FFT)

INDICACIÓN TOXICOLÓGICA

Cardiotoxicidad o neurotoxicidad por anestésicos locales.

Intoxicaciones muy graves por fármacos altamente liposolubles (antagonistas de los canales de calcio, antidepresivos tricíclicos, lamotrigina, quetiapina, bupropion y otros) y que no responden al tratamiento convencional.

POSOLOGÍA ADULTOS

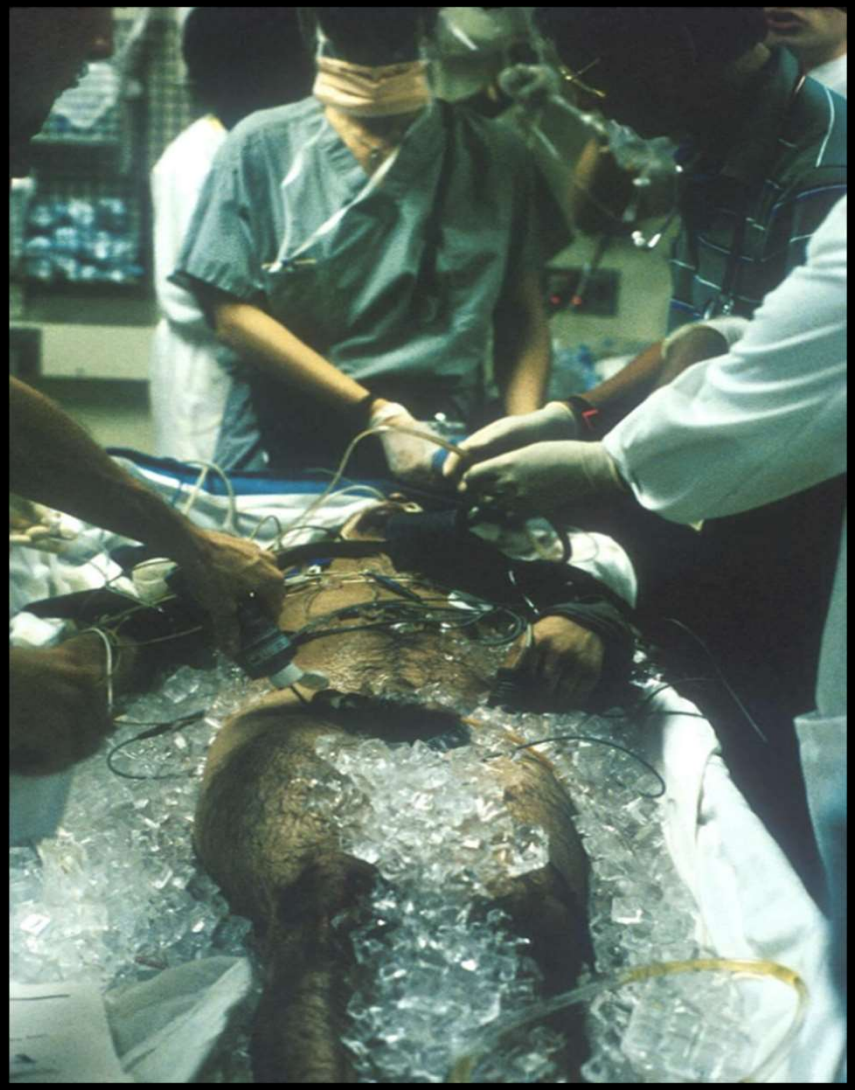
La posología óptima no está definitivamente establecida. A continuación, se muestran las recomendaciones más actualizadas en la bibliografía reciente.

En casos de intoxicación grave que no responde a otras medidas terapéuticas, iniciar con una dosis de ELI al 20% de 1,5 mL/kg en bolus endovenoso durante 2-3 min. Iniciar a continuación una perfusión continua a 15 mL/kg/h (0,25 mL/kg/min) durante 3 minutos. Si transcurrido este tiempo el paciente presenta mejoría, disminuir la velocidad a 1,5 mL/kg/h (0,025 mL/kg/min). Si a los 3 minutos el paciente sigue respondiendo, continuar la infusión durante 10 minutos sin superar la dosis acumulada de 10 mL/kg y finalizar el tratamiento. Si el paciente se inestabiliza, incrementar de nuevo la velocidad de la perfusión a 15 mL/kg/h (0,25 mL/kg/min) durante 30 minutos pudiéndose repetir el bolus inicial en los casos más graves. No superar la dosis máxima de 10 mL/kg.

En caso de parada cardiaca refractaria a las medidas convencionales, la dosis más recomendada es en forma de bolus de 1,5 mL/Kg de ELI al 20% (100 mL para un individuo de 60-70 Kg), repetible cada 3 minutos en caso de persistir la parada hasta un máximo de 5 veces.

LA ÚLTIMA BALA

8. Los pacientes con agitación grave por intoxicación por simpaticomiméticos requieren sedación para controlar la hipertermia y la acidosis, prevenir la rabdomiolisis y las lesiones, y permitir su evaluación.

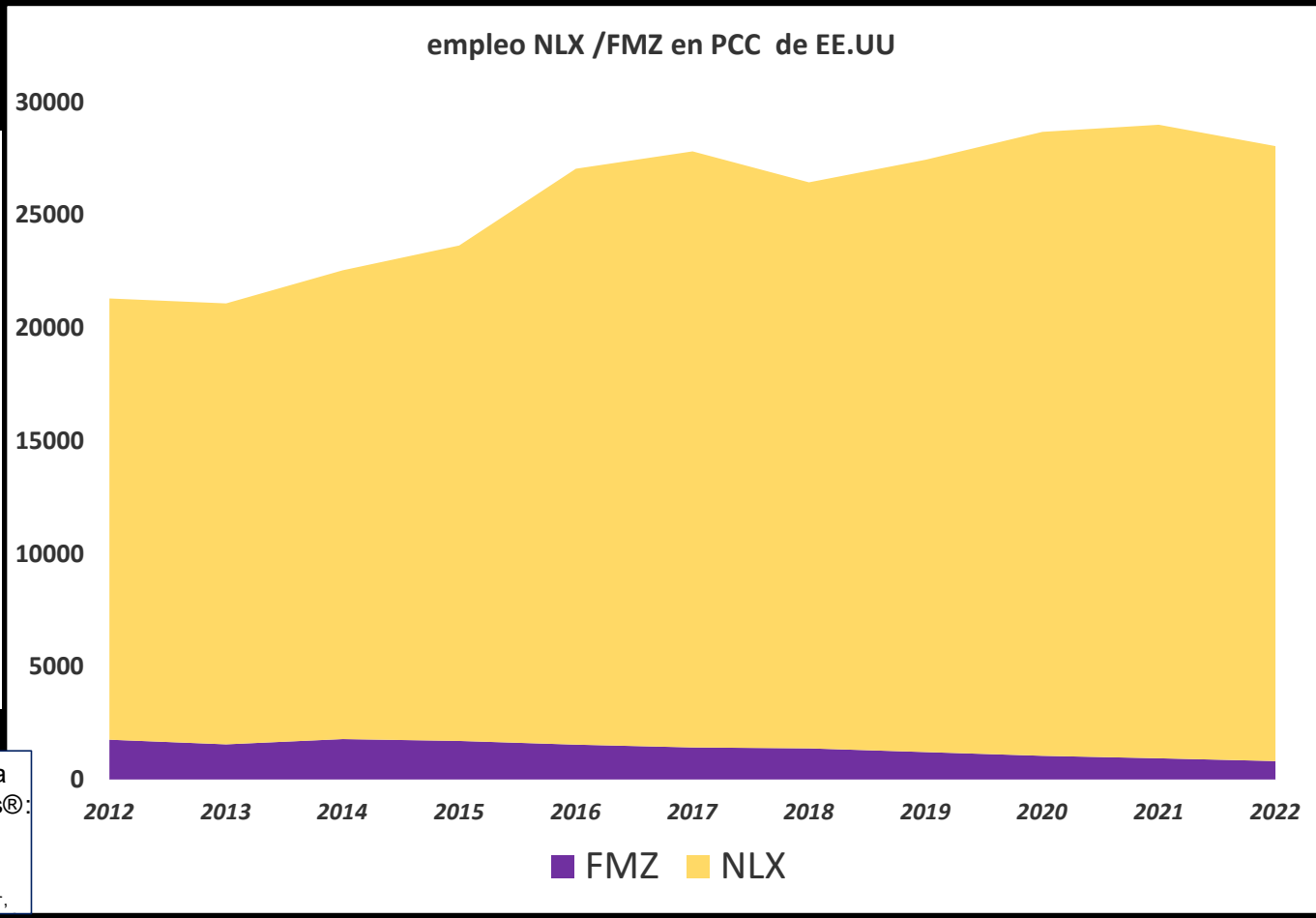


Cedida por Dr. M Galicia

9. El **flumazenilo** revierte la depresión respiratoria y del sistema nervioso central causada por las **BZD pero existen riesgos y contraindicaciones** importantes que limitan su uso.

Recommendations for the Management of Patients With Life-Threatening Benzodiazepine Poisoning

COR	LOE	Recommendations
2a	B-NR	1. If combined opioid and benzodiazepine poisoning is suspected, it is reasonable to administer naloxone first (before other antidotes) for respiratory depression/respiratory arrest.
2a	B-NR	2. Flumazenil can be effective in select patients with respiratory depression/respiratory arrest caused by pure benzodiazepine poisoning who do not have contraindications to flumazenil.
3: No Benefit	C-EO	3. Flumazenil has no role in cardiac arrest related to benzodiazepine poisoning.
3: Harm	B-R	4. Flumazenil administration is associated with harm in patients who are at increased risk for seizures or dysrhythmias.



2023 Annual Report of the National Poison Data System® (NPDS) from America's Poison Centers®: 41st Annual Report

David D. Gummin, James B. Mowry, Michael C. Beuhler, Daniel A. Spyker,

Criterios para el **uso seguro de FLUMAZENILO**

1) COMA (S Glasgow < 12, AV**DN**)

+

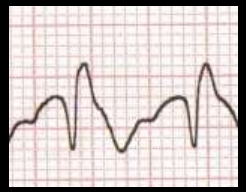
2) Criterio de **gravedad** asociado

SATURACIÓN BAJA o INSUFICIENCIA RESPIRATORIA

HIPOTENSIÓN – BRADICARDIA MANIFIESTA

+

3) **Garantía** de **NO coingestas convulsivantes** o epilepsia previa

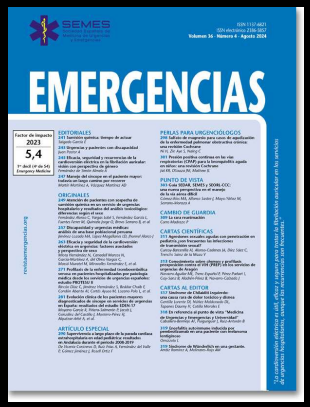


para evitar una IOT…….. aunque no la evitará

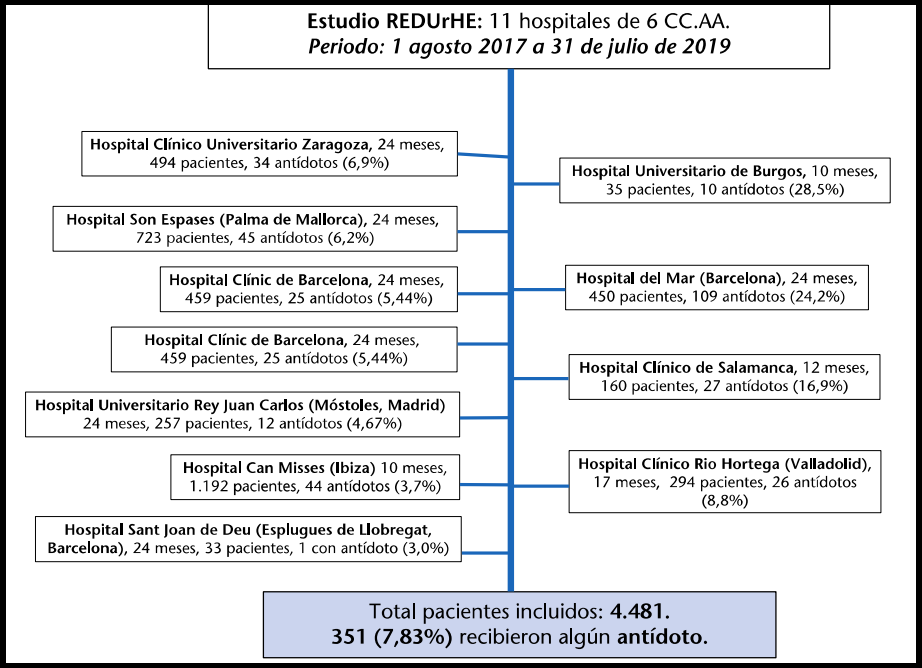
ORIGINAL

Análisis del uso de antídotos en intoxicaciones por drogas de abuso en servicios de urgencias españoles

M.ª Carmen Rodríguez-Ocejo[1], Blanca Rodríguez-Gamella[2], Miguel Galicia Paredes[3], Francisco Pagán[3], August Supervía Caparrós[4], Dima Ibrahim-Achi[5], Guillermo Burillo-Putze[5], Jordi Puiguriguer Ferrando[1], en representación de la Red de estudio de drogas en Urgencias Hospitalarias en España (REDUrHE)

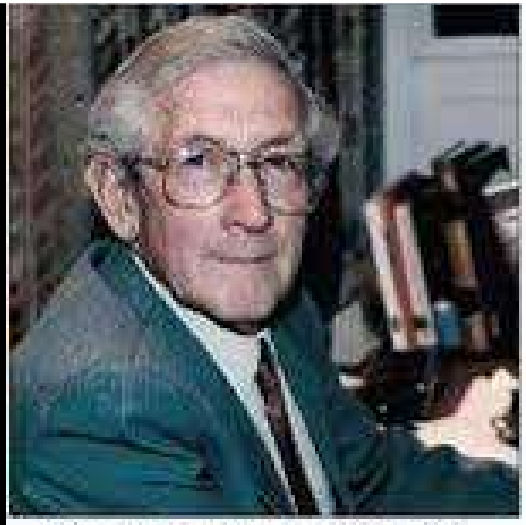


- **Se administró naloxona a 243 y flumazenilo a 225 pacientes. El 34,5% recibieron ambos antídotos.**
- **Urgencias extrahospitalarias (79,3%).**
- **administración inadecuada de flumazenilo fue del 81,3% y del 70,7% para la naloxona.**
  - **El flumazenilo se administró en pacientes con SCG > 12 en 49,7% de ocasiones.**
  - **4,8% de los tratados con antídotos habían convulsionado o estaban en tratamiento antiepiléptico,**
  - **el 40,8% habían consumido cocaína, el 26,8% algún anfetamínico**
- **un 25,9% precisó tratamiento con ansiolítico o medidas de sedación.**
- **En 5 casos (1,6%) complicaciones neurológicas graves tras su empleo.**



Assessment of coma and impaired consciousness. A practical scale

Lancet . 1974 Jul 13;2(7872):81-4. G Teasdale, B Jennett



Bryan Jennett (1926–2009)

Graham Teasdale (n. 1940)

es fácil de usar y tiene poca variabilidad.

1) **decidir** ciertos tratamientos
2) **compara**r diferentes series de lesiones,
3) **predecir** el grado de recuperación final esperada
4) Permite **seguir** la evolución

ASSESSMENT OF COMA AND IMPAIRED CONSCIOUSNESS

A Practical Scale

GRAHAM TEASDALE BRYAN JENNETT

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Summary A clinical scale has been evolved for assessing the depth and duration of impaired consciousness and coma. Three aspects of behaviour are independently measured—motor responsiveness, verbal performance, and eye opening. These can be evaluated consistently by doctors and nurses and recorded on a simple chart which has proved practical both in a neurosurgical unit and in a general hospital. The scale facilitates consultations between general and special units in cases of recent

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especially their
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risks of Japan
such as those

brain damage, and is useful also in defining the duration of prolonged coma.

Introduction

A WIDE range of conditions may be associated with coma or impaired consciousness. Apart from acute brain damage due to traumatic, vascular, or infective lesions, there are metabolic disorders such as hepatic or renal failure, hypoglycæmia or diabetic ketosis, and also drug overdose. In gauging deterioration or improvement in the acute stage of such conditions, as well as in predicting the ultimate outcome, the degree and duration of altered consciousness usually overshadow all other clinical features in importance. It is therefore vital to be able to assess and to record changing states of altered consciousness reliably.

Need for a Clinical Scale

Impaired consciousness is an expression of dysfunction in the brain as a whole. This may be due to agents acting diffusely, such as drugs or metabolic imbalance; or to the combination of remote and local effects produced by brain damage which was initially focal. Such focal brain damage may affect some of the responses which are used to assess the level of consciousness, and any scale devised for general use must allow for this possibility. A simpler scale might suffice for metabolic or drug coma, when the likelihood of structural brain damage is small, but in an emergency there may be insufficient information to assign patients confidently to a particular diagnostic category. Moreover, coma of mixed origin is not uncommon, as when head injury is suspected of being associated with ingestion of drugs or alcohol, or with a vascular accident. These seem good reasons for devising a generally applicable scheme of assessment.

Existing Systems

The development of equipment for monitoring various functions in critically ill patients has not altered the need for doctors and nurses to assess the level of consciousness. There is an abundance of alternative terms by which levels of coma or impaired consciousness are described and recorded. Systems for describing patients with impaired consciousness are not consistent.[1–14] Indeed, many clinicians retreat from any formal scheme in favour of a general description of the patient's state, without clear guidelines as to what to describe and how to describe it.

In practice, such unstructured observations commonly result in ambiguities and misunderstandings when information about patients is exchanged and when groups of patients treated by alternative methods are compared, or reported from different centres. There is no general agreement about what terms to use, nor are those in common use interpreted similarly by different workers. Almost every report of patients in coma offers yet another classification. Most divide the spectrum of altered consciousness into a series of steps, which in the reports we reviewed ranged from 3 to 17 and were often described in terms which defied clear definition. Many assume the existence of constellations of clinical features which are unique to each "level", whilst

The Glasgow Coma Scale at 40 years: standing the test of time

Graham Teasdale, Andrew Maas, Fiona Lecky, Geoffrey Manley, Nino Stocchetti, Gordon Murray

	Patients	Deaths (N)	Proportion that died (95% CI)
3	53246	13823	26·1% (25·7–26·4)
4	3076	818	26·8% (25·2–28·3)
5	3093	717	23·2% (21·7–24·7)
6	5948	1019	17·2% (16·2–18·2)
7	5787	669	11·6% (10·8–12·4)
8	5367	547	10·2% (9·4–11·0)
9	5613	536	10·0% (8·8–10·4)
10	7127	567	8·0% (7·4–8·6)
11	8233	551	6·7% (6·2–7·3)
12	11312	557	5·0% (4·5–5·3)
13	21517	872	4·0% (3·8–4·3)
14	86791	2381	2·8% (2·6–2·7)
15	801025	8280	1·04% (1·01–1·06)

Data for 1018135 individuals in the National Trauma Data Bank and a known score on Glasgow Coma Scale and outcome.

***Table 3*: Relation of Glasgow Coma Score to mortality in injured people with or without traumatic brain injury**

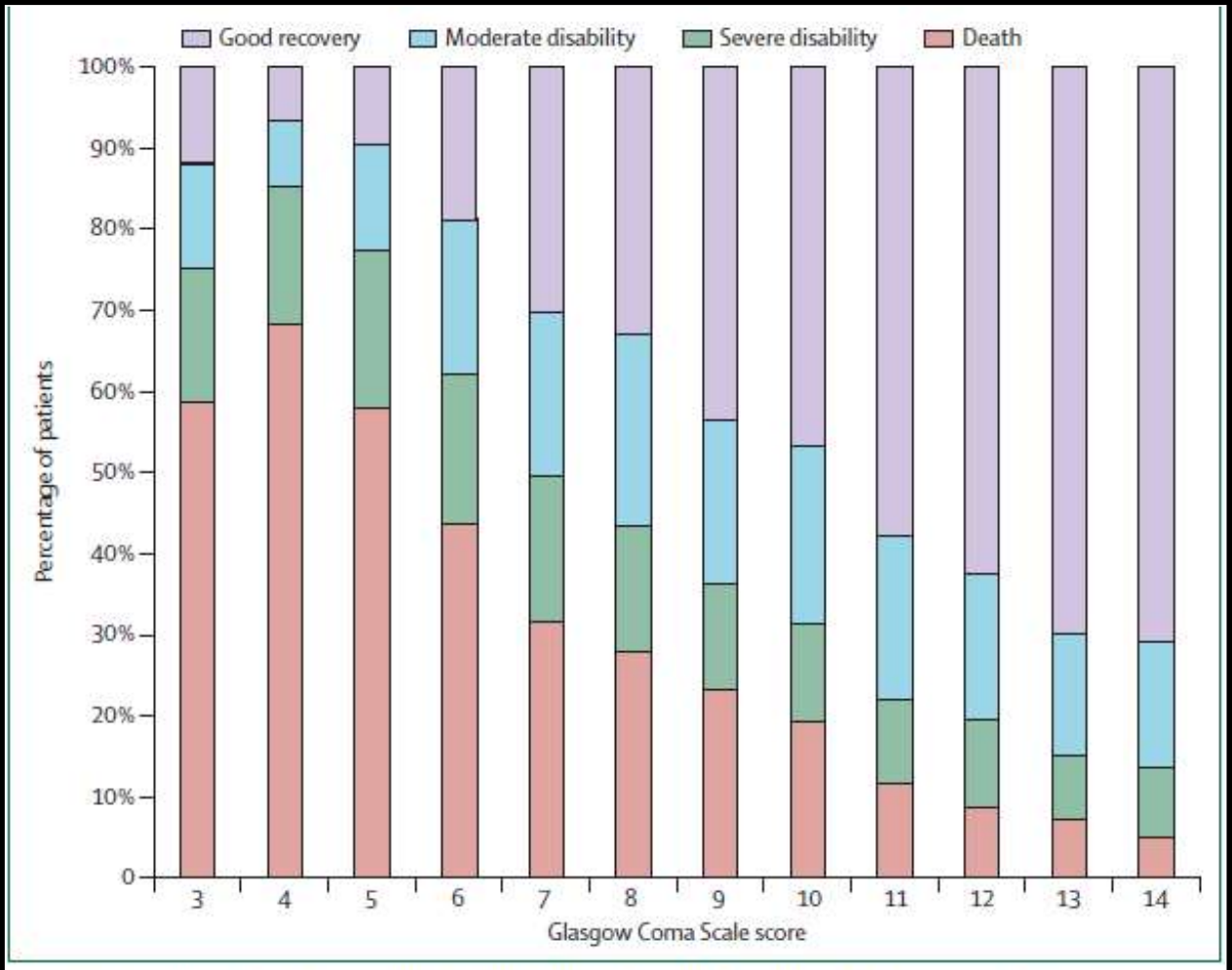


***Figure 3*: Mortality and outcome 6 months after injury in relation to Glasgow Coma Score recorded at the time of recruitment onto the MRC CRASH trial[24]**

***Lancet Neurol* 2014; 13: 844–54**

Nanah A, **Outcomes and Practices of Endotracheal Intubation Using the Glasgow Coma Scale in Acute Non-Traumatic Poisoning: A Systematic Review and Meta-Analysis of Proportions**. **J Intensive Care Med. 2024** Aug 16:8850666241275041.

The conventional **"less than 8, intubate"** approach may **not be directly applicable to acute poisoning patients** due to heterogeneity in patient presentation, intubation practices, and low mortality.

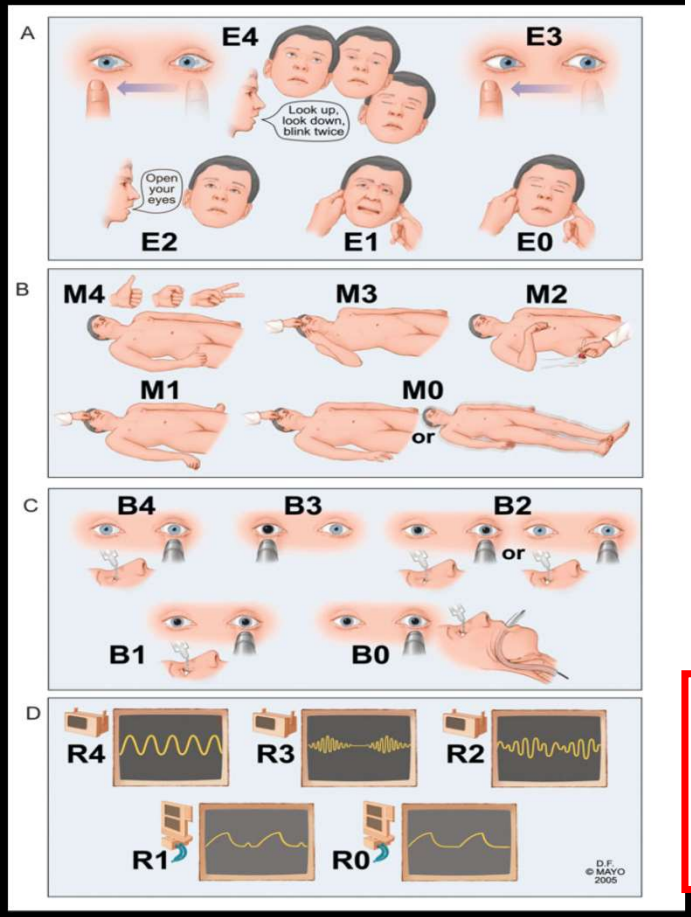
Therefore, a nuanced approach is warranted to optimize airway management strategies tailored to individual patient needs.

FOUR (Full Outline of UnResponsiveness) (2005)

Ann Neurol 2005;58:585–593

→ **PARA EL TCE.**

NO VALIDADO PARA INTOXICADOS



Validation of a New Coma Scale: The FOUR Score

Eelco F. M. Wijdicks, MD,[1] William R. Bamlet, MS,[2] Boby V. Maramattom, MD,[1] Edward M. Manno, MD,[1] and Robyn L. McClelland, PhD[2]

FOUR Score

Eye response
- 4 = eyelids open or opened, tracking, or blinking to command
- 3 = eyelids open but not tracking
- 2 = eyelids closed but open to loud voice
- 1 = eyelids closed but open to pain
- 0 = eyelids remain closed with pain

Motor response
- 4 = thumbs-up, fist, or peace sign
- 3 = localizing to pain
- 2 = flexion response to pain
- 1 = extension response to pain
- 0 = no response to pain or generalized myoclonus status

Brainstem reflexes
- 4 = pupil and corneal reflexes present
- 3 = one pupil wide and fixed
- 2 = pupil or corneal reflexes absent
- 1 = pupil and corneal reflexes absent
- 0 = absent pupil, corneal, and cough reflex

Respiration
- 4 = not intubated, regular breathing pattern
- 3 = not intubated, Cheyne–Stokes breathing pattern
- 2 = not intubated, irregular breathing
- 1 = breathes above ventilator rate
- 0 = breathes at ventilator rate or apnea

https://www.youtube.com/watch?v=QitWrm93Kzg

PASS Poisoning Agitation-Sedation Score

- ✓ **Intoxicados agudos** con alteración del nivel de conciencia
- ✓ 187 pacientes.

Minimum-maximum	18–70	
Median (IQR)	25 (20–33)	
Male	99	52.9%
Female	88	47.1%
Antipsychotics	55	29.4%
Aluminum phosphide	26	13.9%
Benzodiazepines	18	9.6%
Antiepileptics	18	9.6%
Heroin	17	9.1%
Organophosphates	10	5.3%
Antidepressants	16	8.6%
Carbon monoxide	4	2.1%
Methanol	3	1.6%
Baclofen	6	3.2%
Tramadol	3	1.6%
Ethanol	3	1.6%
Co-ingestion	4	2.1%
Carbamate	3	1.6%
Dormex	1	0.5%

Table 5. Components of the proposed Poisoning Agitation-Sedation Score (PASS) for predicting the need for endotracheal intubation and mechanical ventilation in acutely poisoned patients with disturbed consciousness with or without agitation.

Parameters	Response	Grade
Reflexes	Pupil and corneal reflexes present	4
	One pupil wide and fixed	3
	Pupil or corneal reflexes absent	2
	Pupil and corneal reflexes absent	1
	Absent pupil, corneal, and cough reflex	0
Respiration	Not intubated, regular breathing pattern	4
	Not intubated, Cheyne–Stokes breathing pattern	3
	Not intubated, irregular breathing	2
	Breathes above ventilator rate	1
	Apnea or breathes at ventilator rate	0
Motor	Obeys commands	6
	Localizes pain	5
	Flexion withdrawal	4
	Decerebrate flexion	3
	Decerebrate extension	2
	No response	1
Agitation	Overtly combative or violent; immediate danger to staff	4
	Pulls on or removes tube(s) or catheter(s) or has aggressive behaviour toward staff	3
	Frequent non purposeful movement or patient–ventilator dyssynchrony	2
	Anxious or apprehensive but movements not aggressive or vigorous	1

Development and validation of a novel poisoning agitation-sedation score for predicting the need for endotracheal intubation and mechanical ventilation in acutely poisoned patients with disturbed consciousness

A. Abd Elghany et al Human and Experimental Toxicology Volume 42: 1–13. 2023

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Valor entre 1-14
SEDACIÓN

RIESGO SI <9

2 y 18
AGITACION

RIESGO SI < 14

Development and validation of a novel poisoning agitation-sedation score for predicting the need for endotracheal intubation and mechanical ventilation in acutely poisoned patients with disturbed consciousness

Volume 42: 1–13
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DOI: 10.1177/09603271231222253
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Human and Experimental Toxicology

PASS Poisoning Agitation-Sedation Score

Valor entre 1 o 2 y 14 o 18

- 1 a 14 sedado

- 2 a 18 agitado

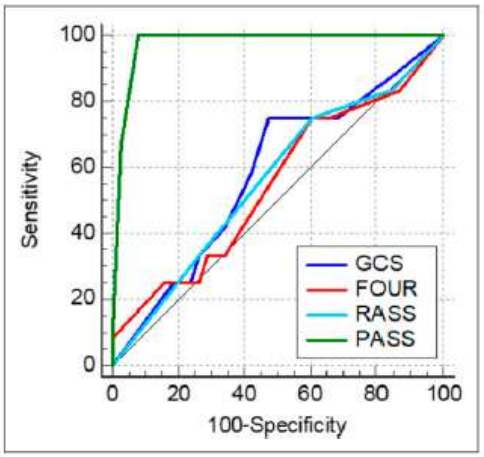


Figure 6. ROC curves of GCS, FOUR, RASS, and PASS (sedation) for predicting the need for endotracheal intubation and mechanical ventilation in the validation cohort.

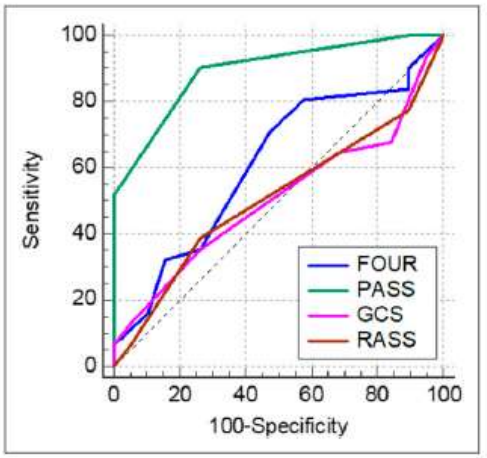


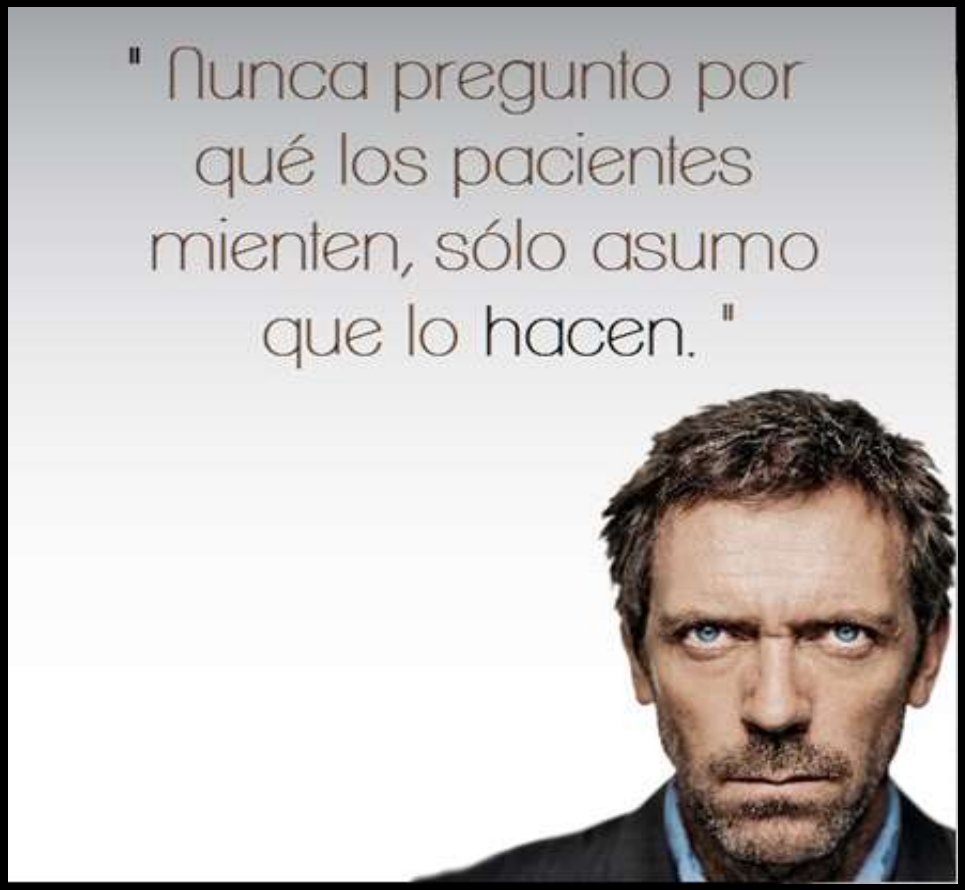
Figure 7. ROC curves of GCS, FOUR, RASS, and PASS (agitation) for predicting the need for endotracheal intubation and mechanical ventilation in the validation cohort.

- ✓ **cutoff ≤9** with 100% sensitivity and 92.11% specificity for predicting the need for ETI and MV **in sedated**
- ✓ 90.32% sensitivity, and 73.68% specificity at a **cutoff ≤14** for predicting the need for ETI and MV in disturbed consciousness patients with **agitation**.

A. Abd Elghany et al Human and Experimental Toxicology Volume 42: 1–13. 2023

Escasa fiabilidad de la anamnesis

→ Hasta **el 40% de las anamnesis no aportan información** relevante o pueden llegar a confundir al médico que la realiza*



*INTOXICACIONES AGUDAS EN MEDICINA DE URGENCIA Y CUIDADOS CRITICOS. A Dueñas

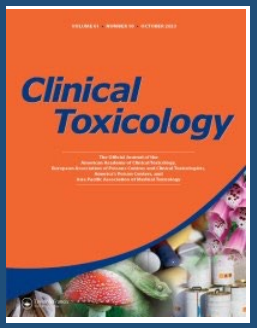
Table 2. Calculated negative predictive values and positive predictive values for self-report that a substance either was, or was not, taken. Note the sum of the prevalence of that category in laboratory sampling is greater than 100% due to polysubstance identification.

Substance	Negative predictive value	Positive predictive value	Prevalence in laboratory samples *n* (%)
Alcohol	0.90	0.37	202 (21.5)
Benzodiazepine	0.50	0.86	496 (52.8)
Cannabis	0.89	0.48	147 (15.7)
Cathinone	0.99	0.40	13 (1.4)
Gamma hydroxybutyrate	0.97	0.53	136 (14.5)
Hallucinogen	1.00	0.19	5 (0.5)
Ketamine	0.91	0.64	110 (11.7)
Opioid	0.51	0.91	534 (56.9)
Other	0.10	0.93	847 (90.2)
Psychiatric or pain medications	0.64	0.86	358 (38.1)
Synthetic cannabinoid receptor agonist	0.93	0.72	149 (15.9)
Stimulant	0.54	0.80	599 (63.8)

Reported recreational drug and new psychoactive substance use versus laboratory detection of substances by high-resolution mass spectrometry in patients presenting to an emergency department in London with acute drug toxicity

Caitlin E. Wolfe, Ashley Rowe, Simon Hudson, John RH. Archer, Paul I. Dargan & David M. Wood

CLINICAL TOXICOLOGY
2024, VOL. 62, NO. 11, 693–697
https://doi.org/10.1080/15563650.2024.2402070

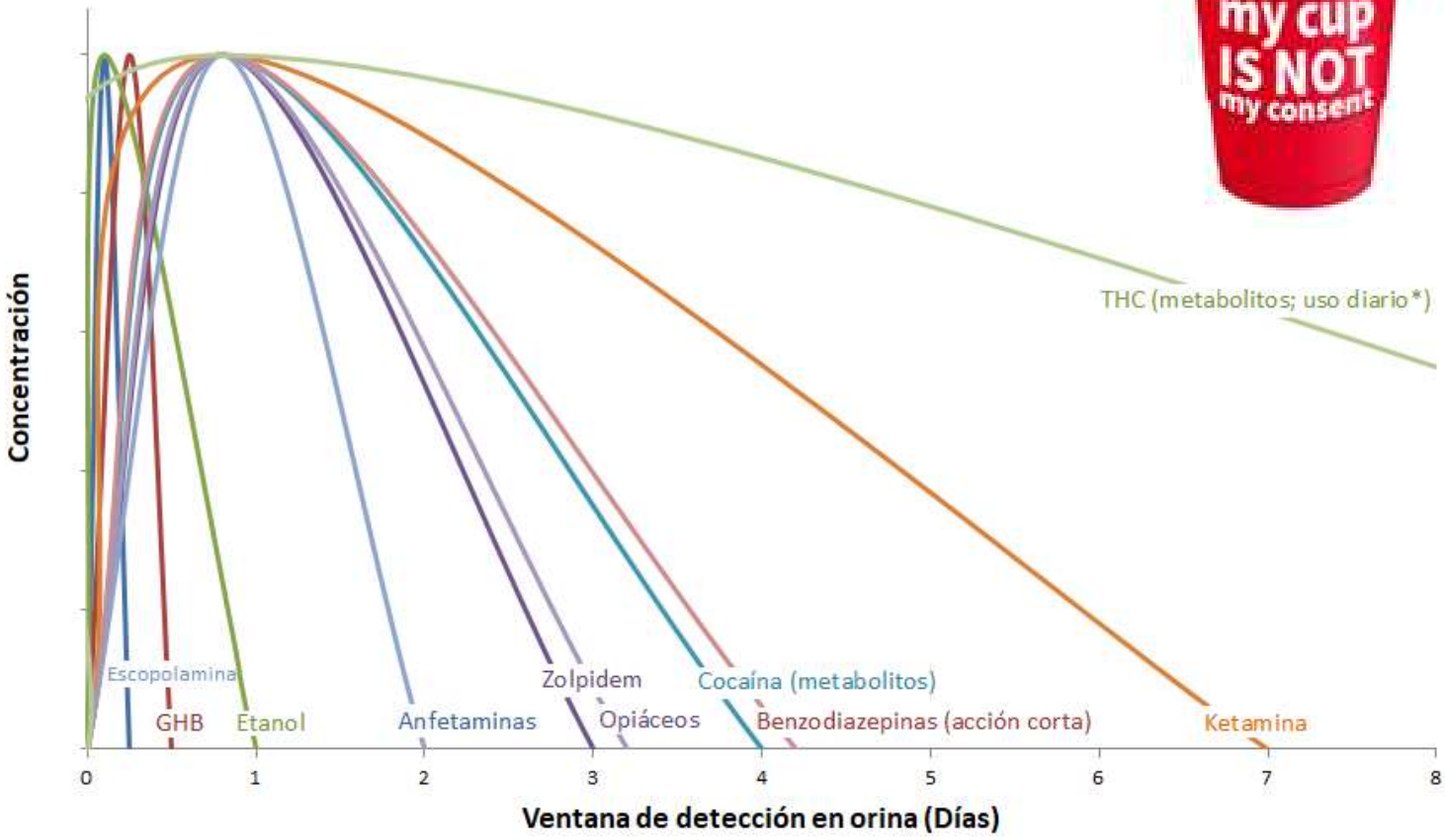


"Los individuos fueron aún más precisos al saber a qué no habían estado expuestos, lo que sugiere que si se niega una sustancia o clase como posible ingesta, es muy probable que eso sea cierto."

https://twitter.com/i/status/982254419721961472

Estudio analítico-toxicológico

Ventana de detección en orina



Fuente: I Gomila (HSLL). B Barceló (HUSE)

Mayo Clin Proc. 2017
Drug Test. Analysis 2

Nuevas evidencias en la atención inicial del paciente intoxicado en urgencias.

¡¡¡ MUCHAS GRACIAS !!!

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