Management of severe poisonings with cardiotoxicants

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Poisonings with cardiotoxicants

- In the USA: AAPCC 2008
  Cardiovascular agents: 10th cause of exposures (3.7%) but 4th cause of death (fatality rate: 0.27%)
- As usual no European data

January 1998 to October 2002
3,922 patients

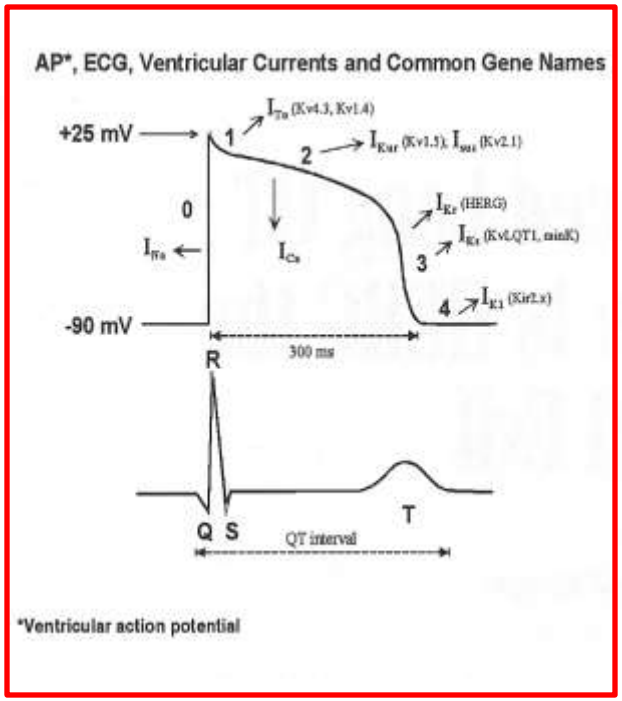
	N	Mortality rate
Poisoned patients	1,554	60 (4 %)
Cardiac complications (severe arrhythmias or failure)	164 (11 %)	37 (22 %)

Lariboisière Hospital ICU, Paris, France

Cardiotoxicants

A larger entity than cardiovascular drugs

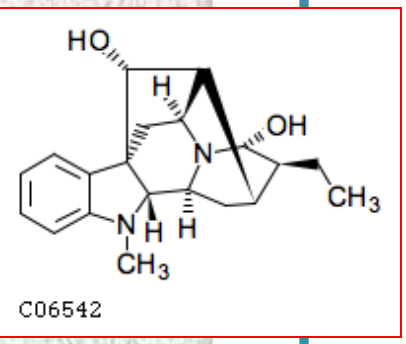
- **Cardiovascular pharmaceuticals**
  - Sodium-channel blockers (Class I)
  - Beta-blockers (class II)
  - Potassium channel blockers (sotalol) (class III)
  - Calcium-channel antagonists (class IV)
  - Cardioglycosides (class V)
- **Non-cardiovascular pharmaceuticals**: antipsychotics, antidepressants, antihistamines, …
- **Drugs**: cocaine, amphetamines, …
- **Rural toxicants**: organophosphates, pesticides, …
- **Industrial toxicants**: alumine phosphide, …
- **Household toxicants**: trichloroethylene, …
- **Plants**: digitalis, aconit, colchicine, yew, Taxus baccata…
- **Over-the-counter**: « Best life » (sibutramine)



The prognostic value of the ingested dose: The example of ajmaline poisoning

Delay for symptom occurrence: 1 - 3 h
All patients in cardiac arrest died

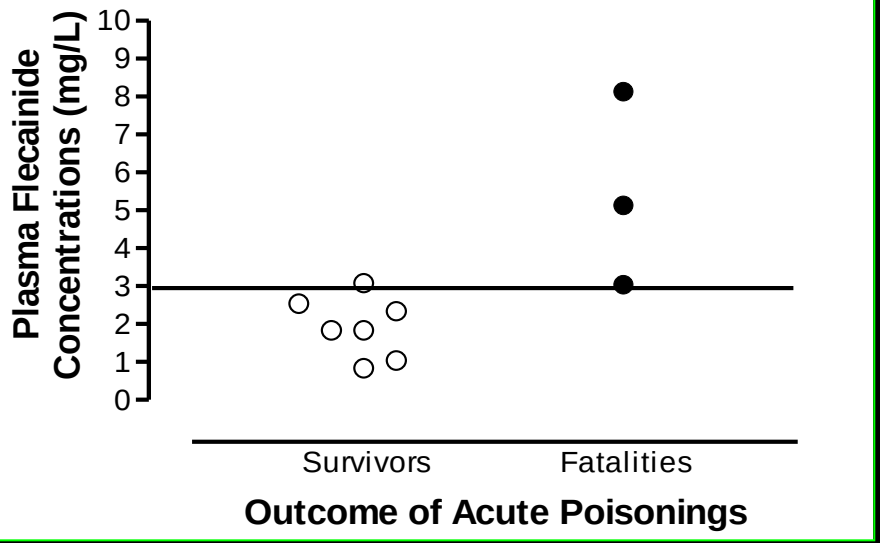
Ingested tablets	N	Cardiac arrest
1 g	7	0
2 g	13	1
3 g	16	8



Conso F. *Press Med* 1980

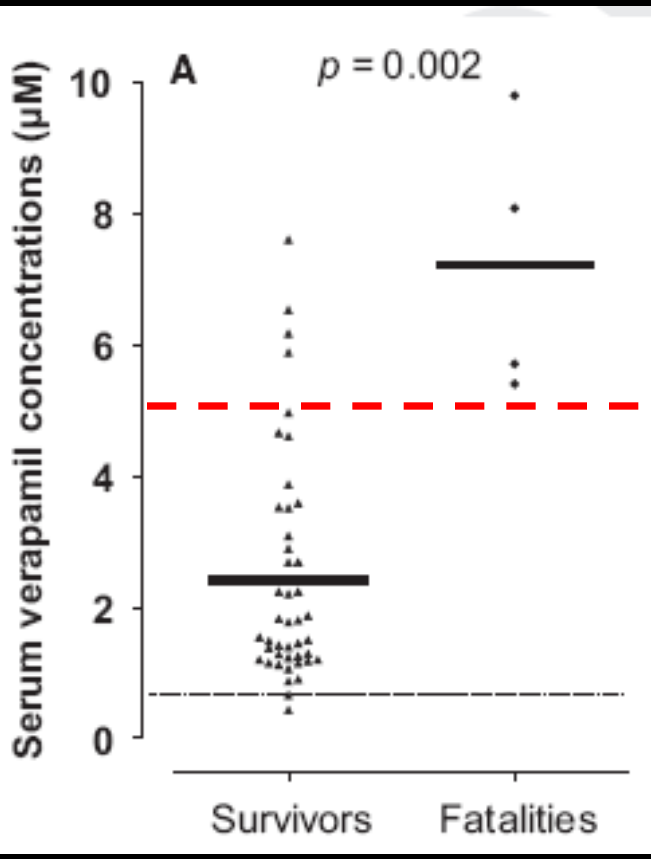
The prognostic value of plasma cardiotoxiant concentrations in acute poisonings

Flecainide poisonings



Mégarbane B. *Clin Tox* 2007

Verapamil poisonings



Mégarbane B. *BCPT* 2010

Specific drug-dependent considerations to assess the risk and features of the intoxicated heart

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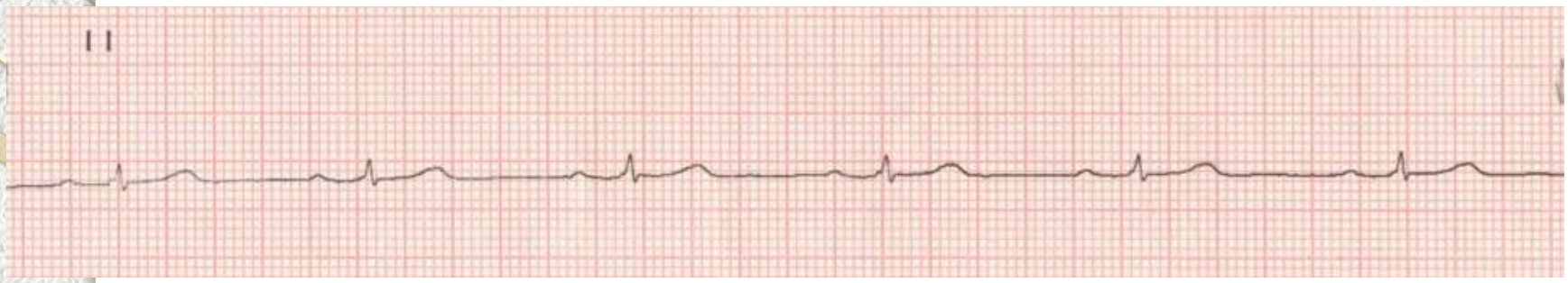
Beta-blocker poisonings (1)

Clinical features



Sinus bradycardia or AV blocks

Other signs:

- Hypotension, collapse
- Bronchospasm
- Respiratory depression
- Drowsiness, seizures, coma
- Hypoglycemia, hyperkaliemia

Dysrhythmias Reported in 23 Beta Blocker Fatalities

Rhythm	Incidence
Bradycardia	15
Asystole	10
Electrical-mechanical dissociation	4
Ventricular fibrillation	4
Junctional rhythm	3
Idioventricular rhythm	3
Ventricular tachycardia	2
Third degree heart block	1

Multiple dysrhythmias were reported in some patients.

Love JN. *J Toxicol Clin Toxicol* 1997

Beta-blocker poisonings (2)

Excess mortality in case of membrane stabilizing activity

Beta Blocker	# Exposures	% Total Exposures	# Deaths	% Deaths
Propranolol*	22,334	43.9	27	71.1
Atenolol	13,587	26.7	6	15.8
Metoprolol	7,511	14.8	1	2.6
Nadolol	2,762	5.4	2	5.3
Labetalol*	1,907	3.7	0	0.0
Pindolol*	742	1.5	1	2.6
Timolol	686	1.4	0	0.0
Acebutolol*	584	1.1	3	7.9
Betaxolol	373	<1.0	0	0.0
Bisoprolol	226	<1.0	0	0.0
Penbutolol*	72	<1.0	0	0.0
Sotalol	48	<1.0	0	0.0
Others	29	<1.0	0	0.0
Unspecified	1,295	2.5	0	0.0
Total	52,156		40	

Two cases involved mixed ingestions of propranolol and atenolol. *Nonspecific membrane activity.

Love JN. *J Toxicol Clin Toxicol* 1997

Beta-blocker poisonings (3)

Excess morbidity in case of cardioactive coingestants (calcium channel blocker, cyclic antidepressant, neuroleptics)

Beta Blocker	All Exposures		Exposures Without Cardioactive Coingestants	
	Number of Exposures	Cardiovascular Morbidity	Number of Exposures	Cardiovascular Morbidity
*Propranolol	121 (43%)	19 (46%)	85 (44%)	8 (50%)
*Metoprolol	36 (13%)	7 (17%)	23 (12%)	4 (25%)
*Labetalol	12 (4%)	3 (7%)	10 (5%)	2 (13%)
*Acebutolol	4 (1%)	1 (2%)	3 (2%)	1 (6%)
*Pindolol	1 (<1%)	0 (0%)	0 (0%)	0 (0%)
Atenolol	87 (31%)	8 (20%)	61 (32%)	1 (6%)
Nadolol	8 (3%)	2 (5%)	4 (2%)	0 (0%)
Bisoprolol	5 (2%)	0 (0%)	3 (2%)	0 (0%)
Timolol	3 (1%)	0 (0%)	3 (2%)	0 (0%)
Sotalol	2 (<1%)	1 (2%)	0 (0%)	0 (0%)
Betaxolol	1 (<1%)	0 (0%)	1 (<1%)	0 (0%)
Total	280	41	193	16

Love JN. *J Toxicol Clin Toxicol* 2000

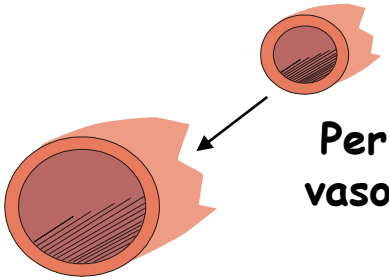
2

Calcium-channel antagonist poisonings (1)

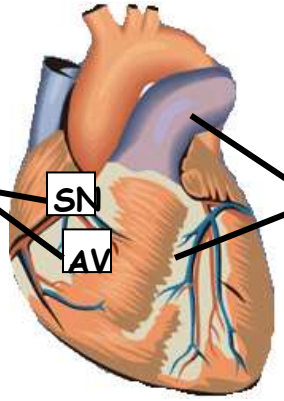
Toxicological consequences of pharmacological properties

Five different CCB classes, including dihydropyridines (nefidipine and amlodipine), phenylalkylamine (verapamil), benzothiazepine (diltiazem), diphenylpiperazine (mibefradil), and diarylaminopropylamine (bepridil).

Dihydropyridines: Selective vasodilators



Peripheral vasodilation

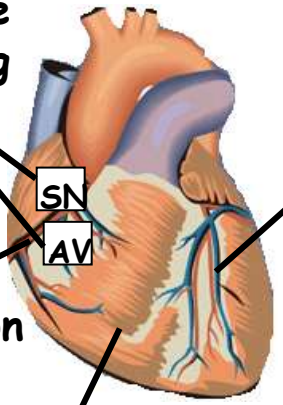


Potential reflex increase in HR, myocardial contractility and O_2 demand

Coronary VD

Non-dihydropyridines: equipotent for cardiac tissue and vasculature

Heart rate moderating



Peripheral and coronary vasodilation

AV conduction dysfunction

Reduced inotropism

Harris NS. *N Eng J Med* 2006

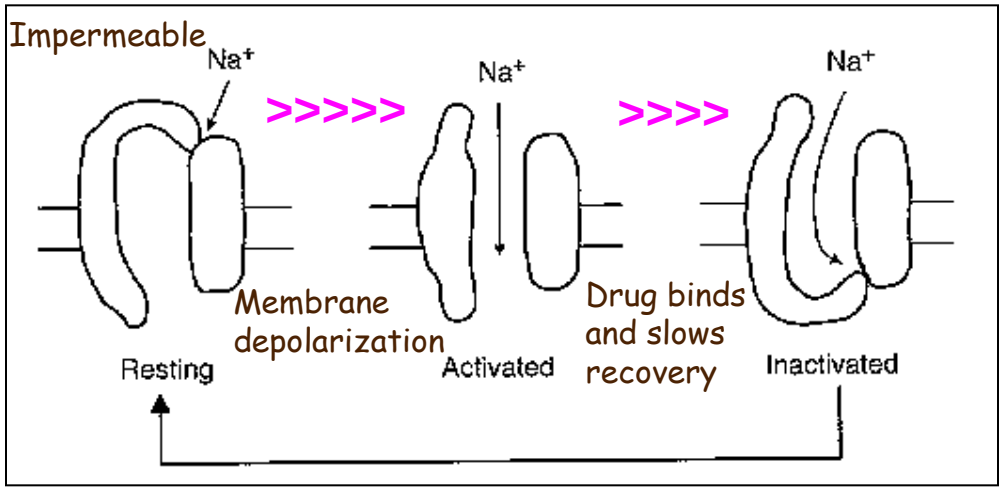
Calcium-channel antagonist poisonings (2)
Features and severity

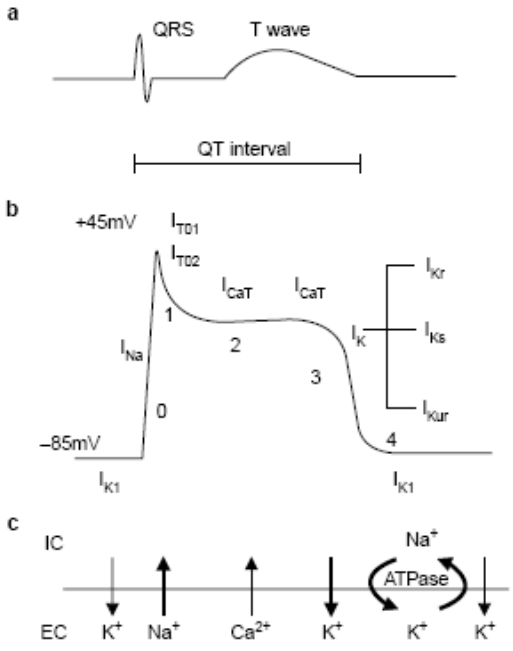
	Verapamil (N = 68)	Diltiazem (N = 27)	Nifedipine (N= 14)	Total (N = 109)
Hypotension	79%	89%	86%	84%
Bradycardia (< 60 /min)	56%	78%	43%	60%
Severe bradycardia (< 40 /min)	24%	26%	43%	60%
AV block	60%	63%	50%	60%
Complete AV block	53%	52%	21%	51%
Cardiac arrest	21%	22%	21%	21%
Death rate	25%	7%	7%	18%

Sauder P. Intoxications aiguës. Elsevier, 1999

Poisonings with sodium channel blockers (1)
Molecules

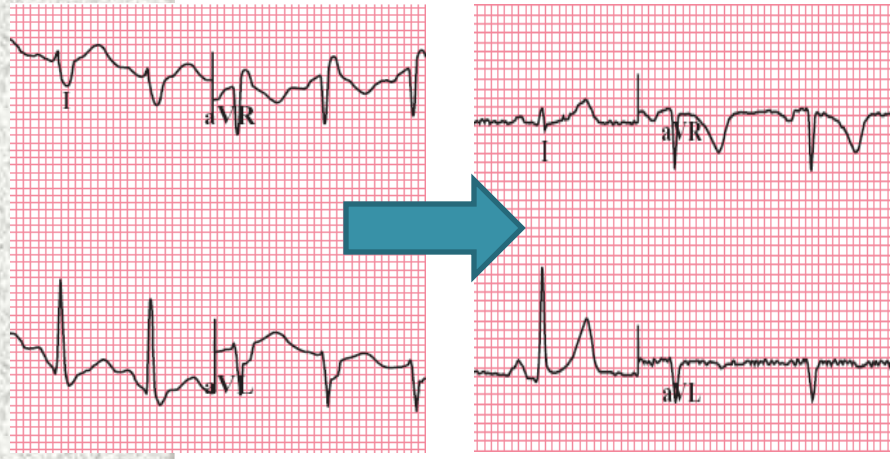
- Polycyclic antidepressants, citalopram and venlafaxin
- Quinine and chloroquine
- Class I anti-arrhytmics (quinidine, cibenzoline, flecainide, propafenone)
- Some β-blockers like propranolol and acebutolol
- Carbamazepine
- Propoxyphene
- Cocaine

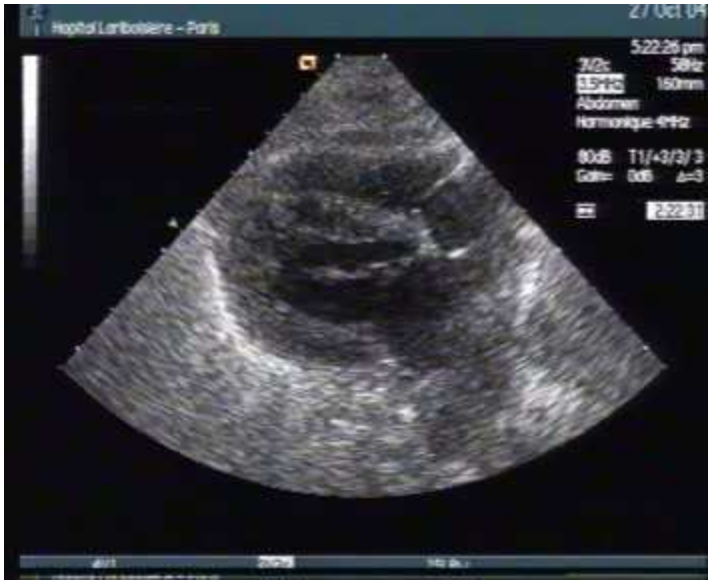




Poisonings with sodium channel blockers (2)
Clinical features

- **Cardiovascular syndrome:**
  - **ECG** : QRS enlargement, QT prolongation, AV blocks
  - **Circulation** : Cardiogenic and vasoplegic shock
- **Metabolic syndrome** : Hypokaliemia, lactic acidosis
- **Neurological syndrome** : Convulsive coma
- **Respiratory syndrome** : Delayed ARDS with alveolar hemorrhage





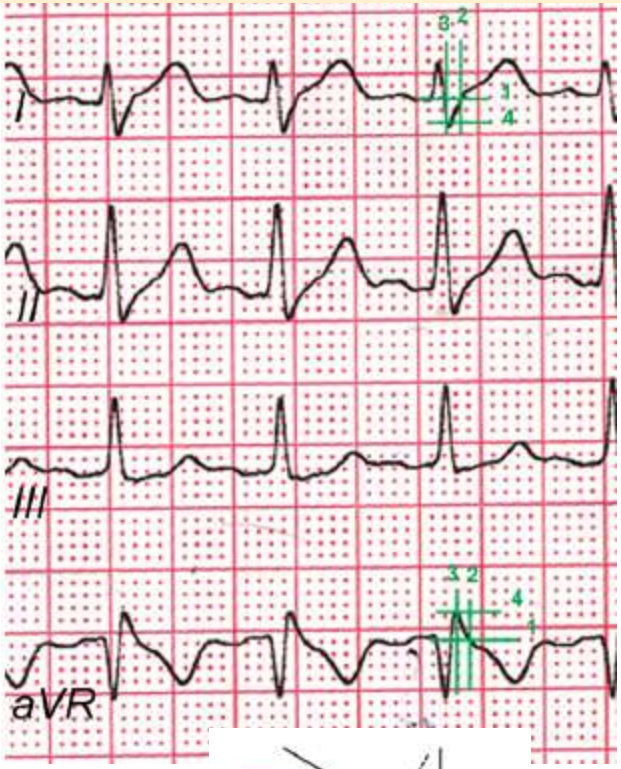
Poisonings with sodium channel blockers (3)

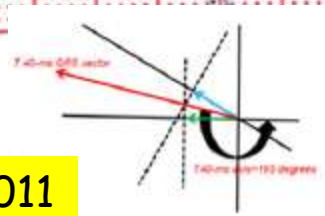
Value of QRS to predict arrhythmias in tricyclic antidepressant poisonings

QRS duration (msec)	Seizure risk	Ventricular dysrhythmia risk
< 100	mild	mild
100 - 160	moderate	mild
> 160	elevated	elevated

Boehnert MT. *N Engl J Med* 1985

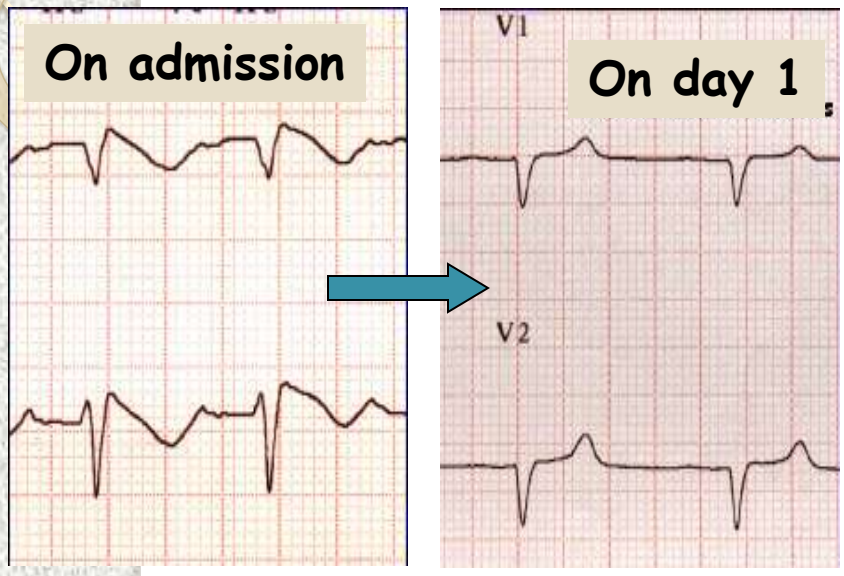
Measurement of the terminal 40-millisecond frontal plane axis





Sanaei-Zadeh H. *Resuscitation* 2011

Poisonings with sodium channel blockers (4)
Brugada syndrome



Prevalence: 15% tricyclic AD poisonings

Disappears if < 1 µmol/l

Response to $NaHCO_3$ controversial

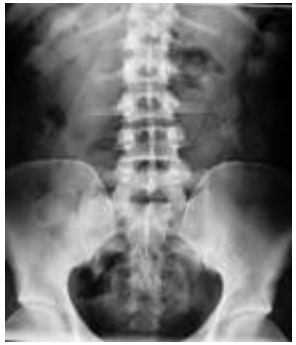
Associated to genetic polymorphism (cocaine)

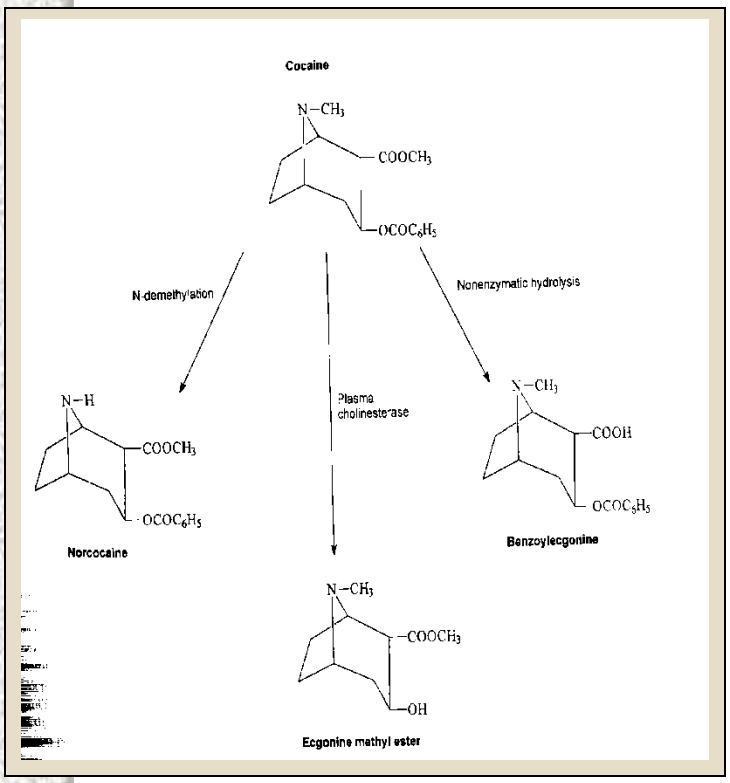
	Type 1	Type 2	Type 3
J wave amplitude	≥ 2 mm	≥ 2 mm	≥ 2 mm
T wave	Negative	Positive or biphasic	Positive
ST-T configuration	Coved type	Saddleback	Saddleback
ST segment (terminal portion)	Gradually descending	Elevated ≥ 1 mm	Elevated < 1 mm

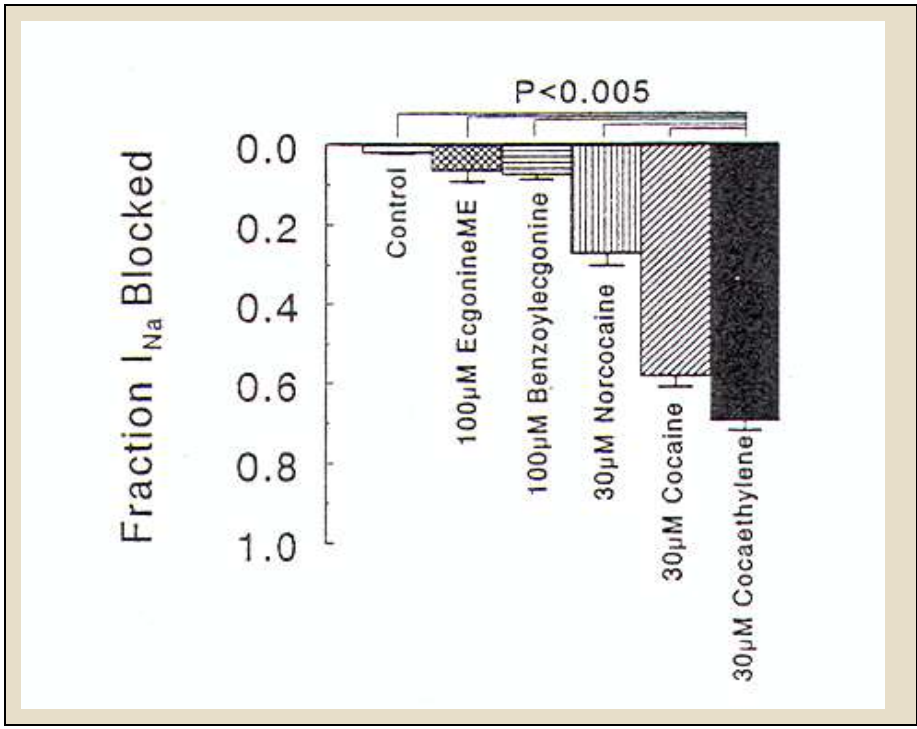
Goldgran-Tolédano D. *NEJM* 2002
Monteban-KooistraWE. *Intensive Care Med* 2005

Cocaine poisoning:

Mechanisms of arrhythmia genesis:
- Sodium channel blockade
- Potassium channel blockade
- Catecholamine excess and SNC agitation
- Myocardial ischemia and infarction







J Pharm Exp Ther 1994

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Cardioglycoside poisonings (1)

Clinical features of digitalis poisoning

Na/K - ATPase blockade
Circumstances: therapeutic overdose > suicide

Multiple and mostly nonspecific manifestations

Fatigue, blurred vision, disturbed color perception

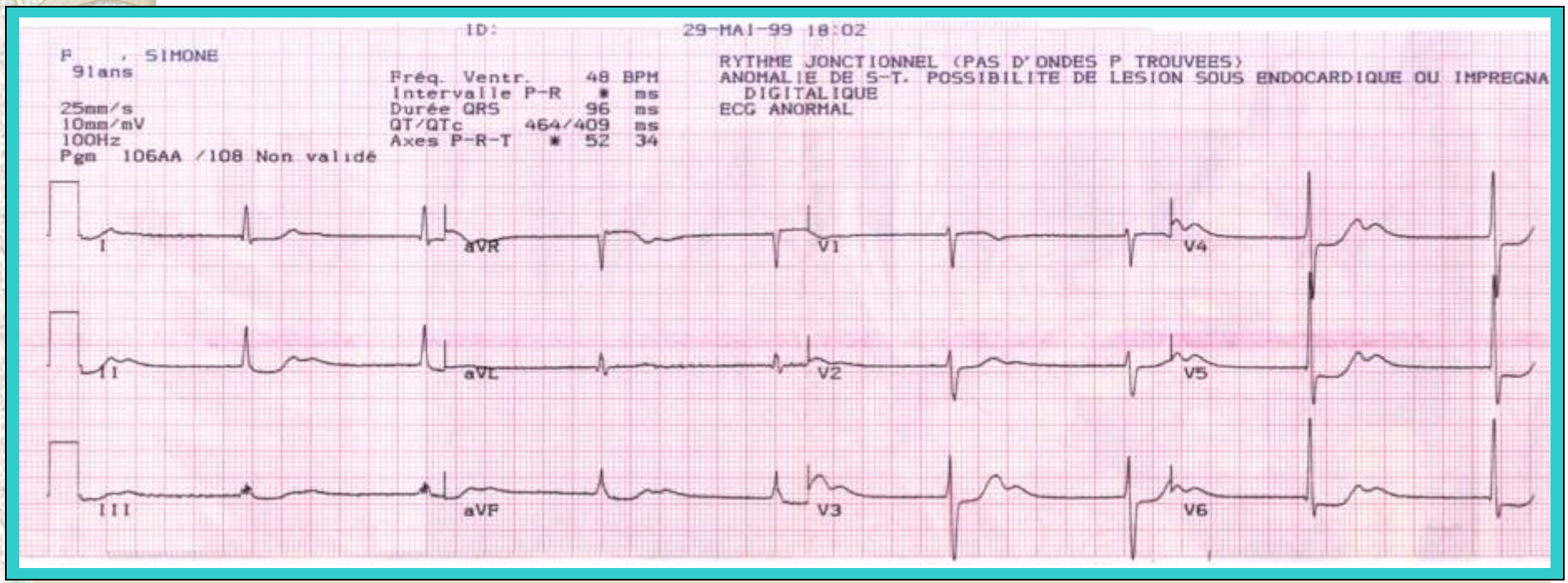
Anorexia, nausea, vomiting, diarrhea, abdominal pain

Headache, dizziness, confusion, delirium, and occasionally hallucinations

Rarely intestinal none occlusive infarction

- Blood pressure is usually preserved (sympathic tone), while cardiac dysfunction possible
- Cardiac arrhythmias may take almost any form and are responsible for mortality

Cardioglycoside poisonings (2)
Typical ECG in digitalis poisoning



Combination of SVT + AV block is highly suggestive of digitalis toxicity

Harmless	Sinus-bradycardia, ST-scoop, AVB I
Less harmless	AVB II, bigeminus, PAT
Dangerous	AVB III, Polytope ventricular extrasystolia, VT
Near death	VF, asystolia

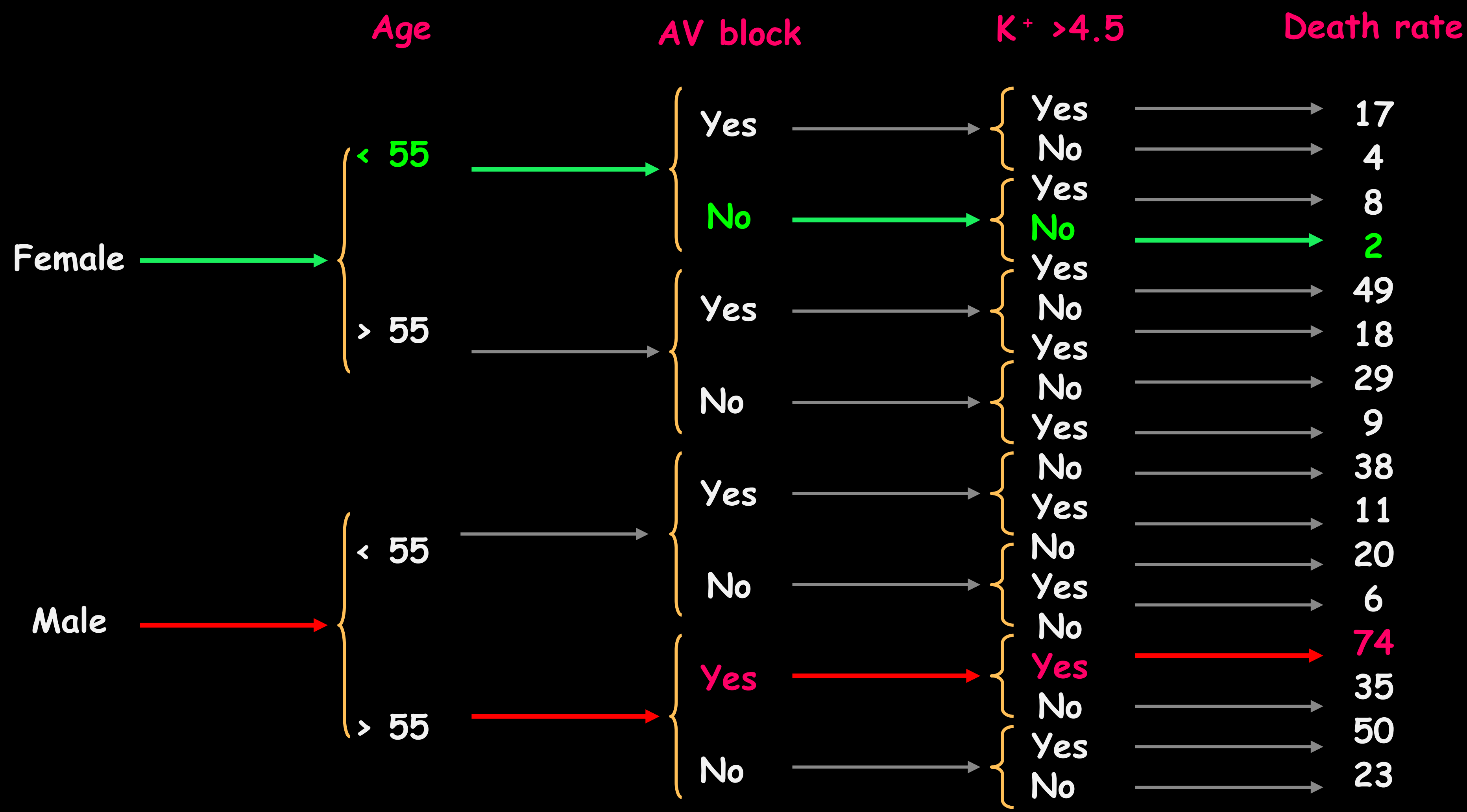
Cardioglycoside poisonings (3)
ECG features

	AF	SAB	AVB 1	AVB 2	AVB 3	VT/VF
Taboulet, % N=141	17	26.8	12.2	9.8	14.6	9.7
Lapostolle, % N=141	25.7	24.2	10.6	14.3	19.7	9.1

AF: Atrial fibrillation with ventricular response <50/min
BSA III : Sinoatrial block of third degree
AVB I / II / III : Atrioventricular block of first, second or third degree

Taboulet P. Clin Toxicol 1993
Lapostolle F. Crit Care Med 2009

Main prognostic factors



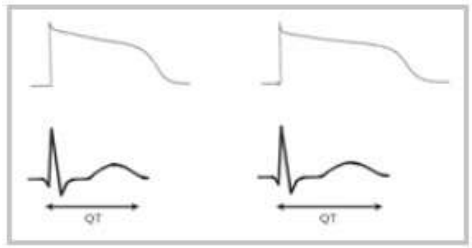
Dally S. *Press Med* 1981

5

Drugs associated with QT-prolongation and torsade-de-pointes

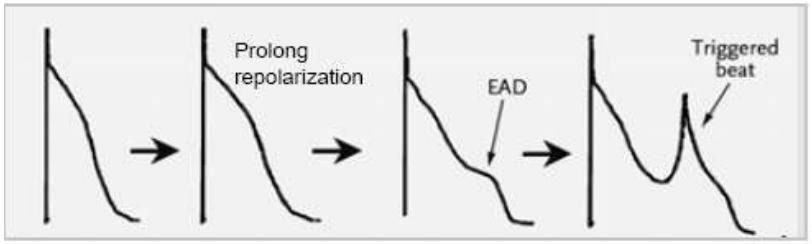
Drug-drug interactions (P-450 inhibitors) > overdoses
+/- genetic vulnerability (hereditary long QT) or cardiac disease

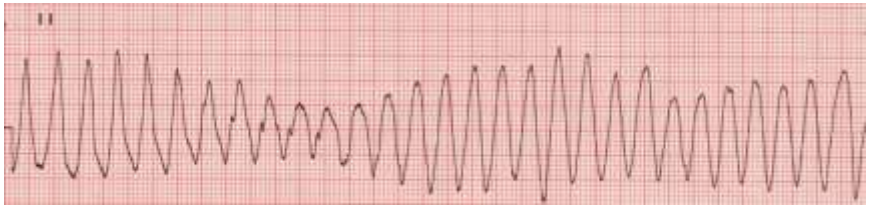
-**Antiarrhythmics** (class Ia, Ic, III, V)
- **Sympathomimetics**
- **Methadone**
- **Antipsychotics & Antidepressants**
(phenothiazines, atypical antipsychotics tricyclics, tetracyclics, SSRIs)
- **Antihistamines**
(terfenadine, astemizole, loratadine)
- **Gastrointestinal agents**
(cisapride, domperidone, dolasetron)
- **Antiinfectives & antifungals**
(macrolides, fluoroquinolones, azoles)



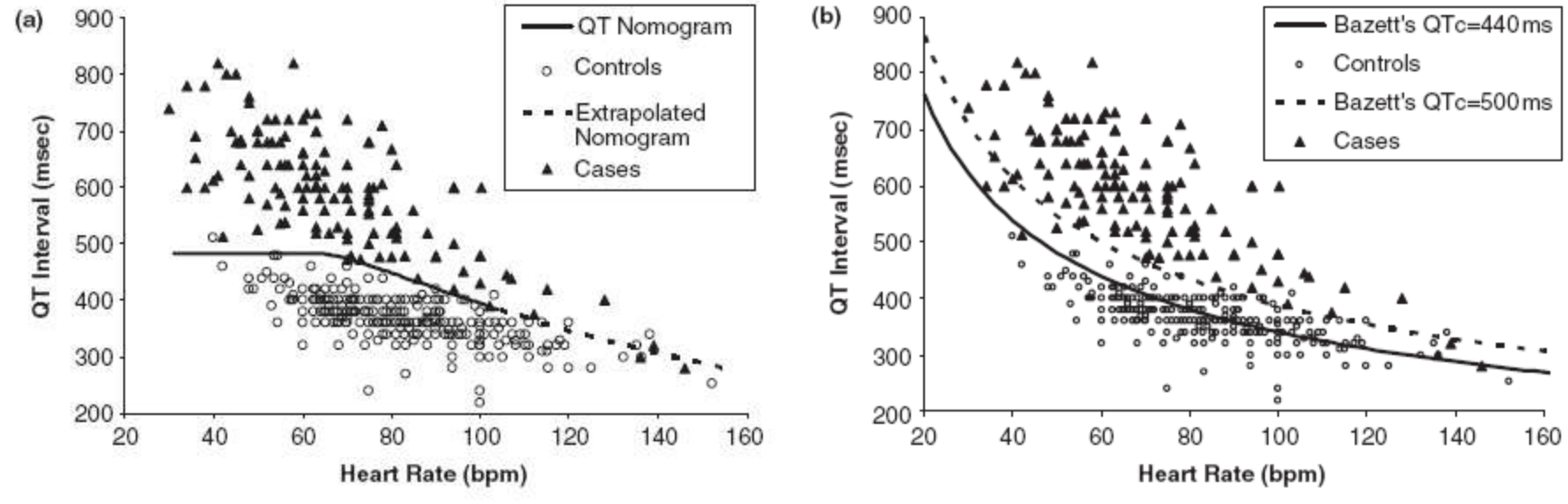
Prolong Repolarization
(QT-Prolongation)

Early After Depolarization
Triggered Activity





Estimation of TdP risk using a QT nomogram



The QT nomogram is a clinically relevant risk assessment tool that accurately predicts arrhythmogenic risk for drug-induced QT prolongation

Chan A. *QJM* 2007

Management of drug-induced cardiac failure and arrhythmias

Strategy of management of toxic cardiovascular failure

Diagnosis of shock

↓

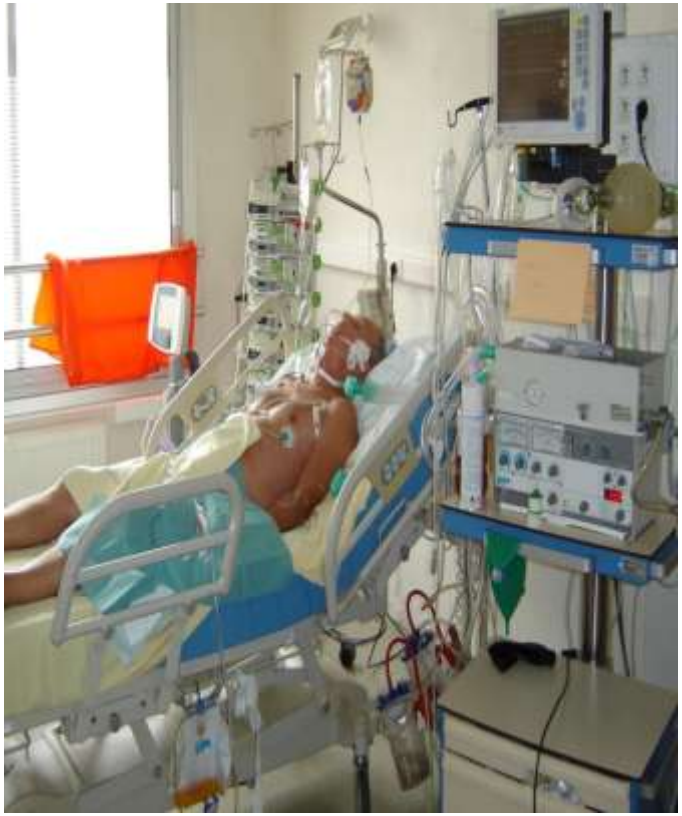
Determination of the mechanism of shock

↓

Definition of the optimal treatment

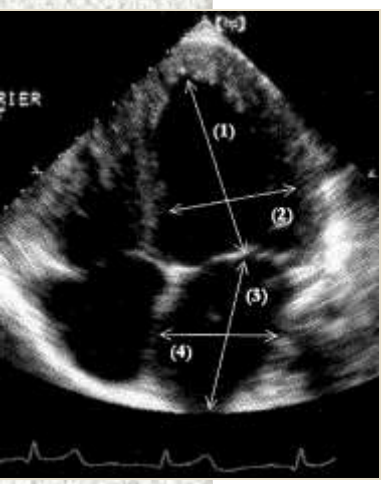
↓

Diagnosis of the refractoriness of shock



Assessment of the mechanism of the toxic shock

1- Hypotension: systolic BP < 90 mm Hg
or systolic BP decrease > 40 mmHg
or mean BP < 65 mmHg
2- Unresponsive to fluids
3- At least one sign of organ hypoperfusion



ECHO immediately available

Yes

No

ECHO

PiCCO

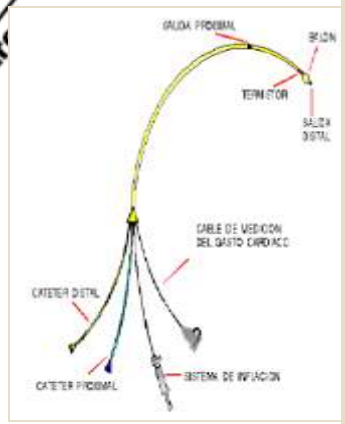
Low GEF (< 18%)

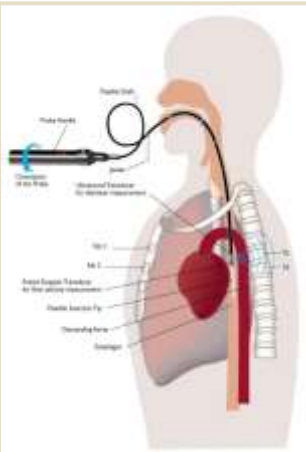
Hemodynamic evaluation:
CO, GEDV, GEF, PPV, SVV

Monitoring requir

Specific ttt
(thrombolysis, surgery…)

Hemodynamic evaluation:
CO, LVEDA, LVEF, ΔVpeak, ΔDivc





Consequences of convulsion-induced hypoxemia and acidosis on cardiac toxicity

	Before	Just after	3h later
Arterial pH	7.39	7.19	7.46
Lactate concentration (mmol/l)	1.7	6.5	3.1
PaO_2 (mmHg)	95	55	90
Systolic BP (mmHg)	120	80	120
QRS width (s)	0.08	0.13	0.08

Taboulet P. *Réan Urg* 1993

Conventional supportive treatments in ICU

- **Intubation and mechanical ventilation :**
  - Severe arrhythmias and associated collapse
  - Coma, convulsions, respiratory failure
- **Treatment of collapse/shock**
  - Fluids + adequate catecholamines
- **Treatment of torsade-de-pointes**
  - Defibrillation, $MgSO_4$, titrated isoproterenol, cardiac pacing
  - Correction of electrolyte imbalance (K^+, Mg^{2+})
- **Treatment of monomorphic ventricular tachycardia**
  - Defibrillation, $MgSO_4$, lidocaine infusion
- **Cardiac pacing**
  - High degree AV block with preserved inotropism

Chloroquine poisoning: prognosis assessment

	Supposed ingested dose		Systolic BP		QRS duration
Severe	≥ 4 g	or	< 100 mmHg	or	> 0.10 s
Moderate	2 - 4 g	and	≥ 100 mmHg	and	≤ 0.10 s
Mild	< 2 g	and	≥ 100 mmHg	and	≤ 0.10 s

Clemessy JL, et al. Crit Care Med 1996

Severe poisoning :

- Epinephrine 0,25 µg/kg/min with increasing 0.25 µg/kg/min steps to obtain SBP ≥ 100 mmHg
- Intubation and mechanical ventilation
- Diazepam 2 mg/kg in 30 min followed with 2-4 mg/kg/24h

Riou B. *N Engl J Med* 1988

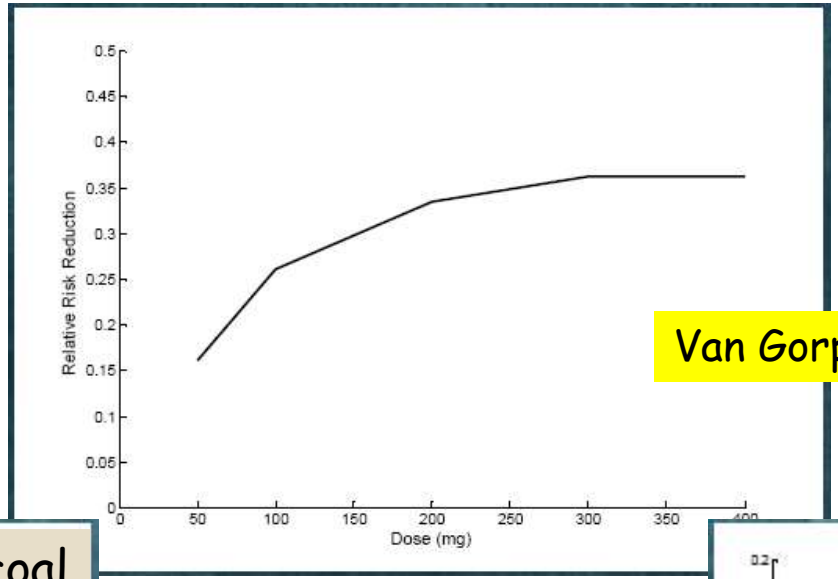
Place of GI decontamination and elimination enhancement

- Activated charcoal: within 2 h following the ingestion

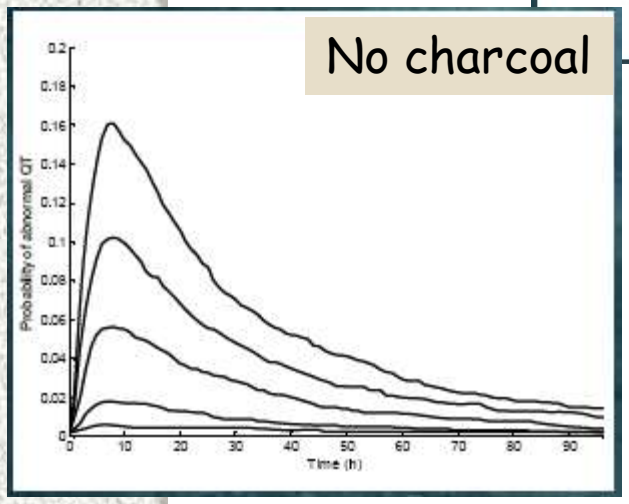
- Repeated doses of charcoal: Low-sustained forms

- Dialysis: limited interest as
  - Elevated protein binding
  - Elevated distribution volume
  - Liposolubility
  - Elevated endogenous clearance

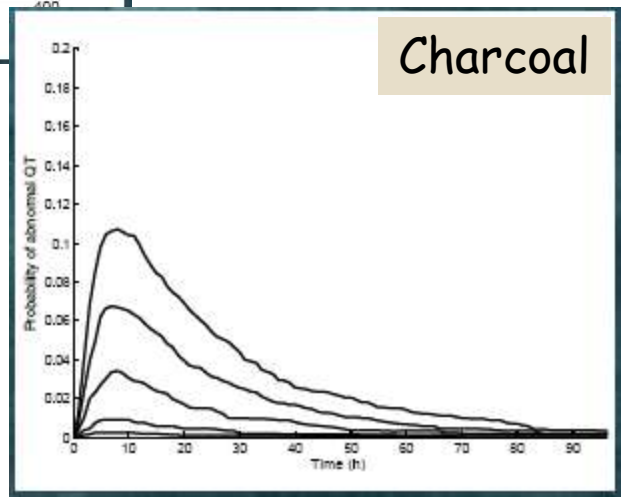
Risk reduction in escitalopram-related QT prolongtation with charcoal



Van Gorp F. *Br J Clin Pharm* 2011



Dose-related lengthening of the QT interval lagging the increase in drug concentration.



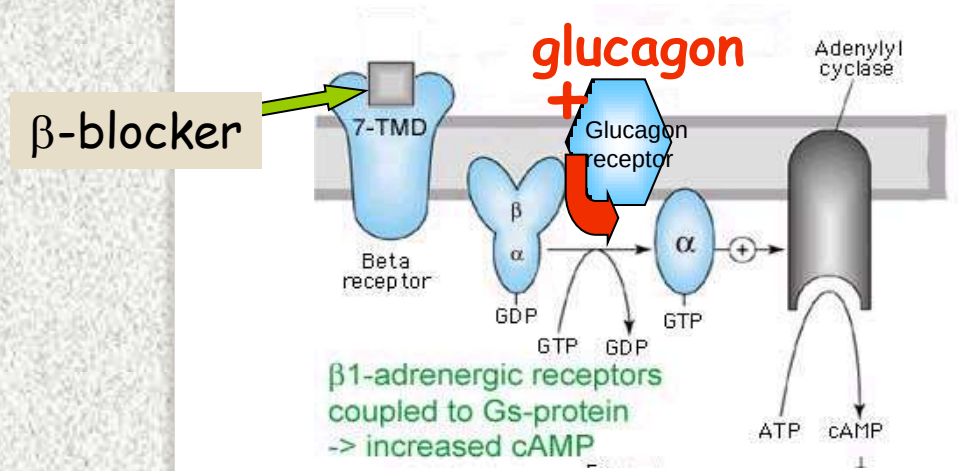
Moderate reduction in the risk of abnormal QT interval with SDAC

Antidotes for beta-blocker poisonings

Specific treatments

We recommend if supportive measures (adequate fluids and atropine) are ineffective, the administration of antidotes in the following order: dobutamine (or isoprenaline, especially in sotalol intoxication), glucagon, and epinephrine.

Taboulet P. *Clin Toxicol* 1993



Suspicion of beta-blocker poisoning
(HR <60 /min and/or SBP <100 mmHg)

↓

Atropine 0.5 mg IV bolus
(if HR <60 /min)
Fluid loading 500-1,000 ml
(if SPB <100 mmHg

↓

Failure of symptomatic therapies

↓

Dobutamine 5-20 µg/kg/min
Isoprenaline 1-5 mg/h (Sotalol)

↓

Glucagon 2-5 mg IV bolus
2-10 mg/h continuous infusion

↓

Epinephrine 0.5-10 mg/h

↓

Ventricular pacing
Exceptional therapies (ECLS)

Antidotes for the calcium-channel blocker poisonings

- Calcium salts: 1 g IV bolus /15-20 min, 4 doses followed with 20-50 mg/kg/h infusion

- Glucose - insulin: 1 UI/kg IV bolus followed with 0.5-1 UI/kg/h infusion + adequate glucose

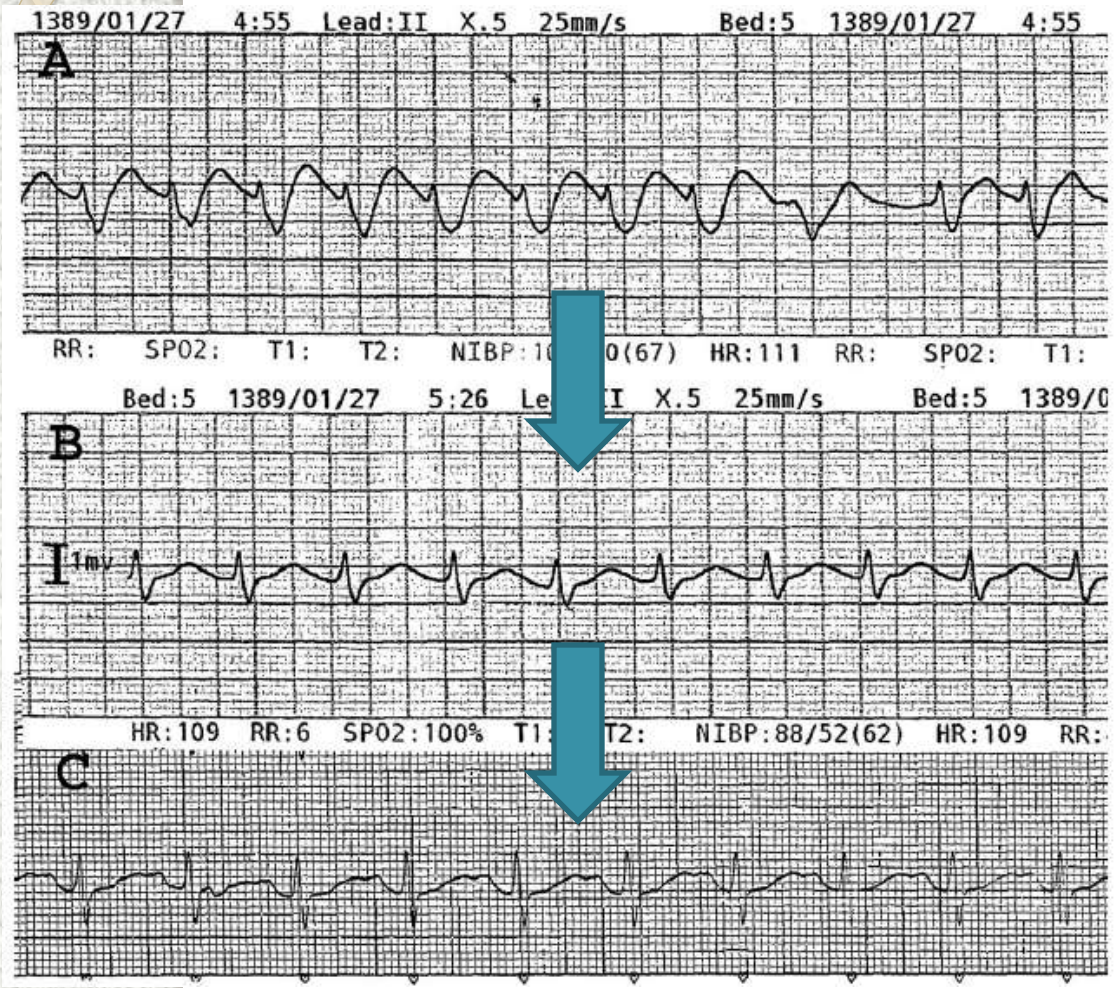
Yuan TH. *J Toxicol Clin Toicol* 1999
Boyer EW. *N Engl J Med* 2001

Metabolic basis for myocardial beneficial effect :

- Increase of insulin pancreas secretion
- Decrease of insulin resistance
- Decrease of free fatty acid uptake and switch to carbohydrates
- Increase of cytoplasmic calcium concentration
- Increase of myocardial "oxygen delivery / work" ratio

Kline JA. *Toxicol Appl Pharmacol* 1997

8.4% Sodium bicarbonate for poisonings with sodium channel blocker agents



The exact mechanism, optimal dosing, and mode of infusion are not well defined.

The most common approach: 1mEq/kg IV bolus if widened QRS or dysrhythmia.

Repeat boluses /3–5 min or place continuous infusion to achieve resolution of the dysrhythmia or QRS narrowing.

Serum pH should not exceed 7.55.

Goldfrank's toxicologic emergencies, McGraw-Hill, 2007

Fat emulsion for local anesthetic toxicity

To treat severe anesthetics side-effects in the OR as well as membrane-stabilizing agent or calcium-channel blocker poisonings.

Dose regimen: 1.5 ml/kg IV bolus then 0.25 ml/kg/min infusion

Mechanisms:

- Lipid sink / sponge: alteration of tissue distribution
- Modulator of myocardial energy, overcoming the inhibition of fatty acid-dependent metabolism
- Activator of myocardial Ca^{2+} channel increasing Ca^{2+} current
- Other toxin-specific mechanisms?

Sirianni AJ. *Ann Emerg Med* 2008
Finn SD. *Anesthesia* 2009
Weinberg GL. *Anesthesiology* 2009

Is pacing still appropriate in digitalis poisonings?

92 acute digitalis poisoning (1983-1990)

51 treated with cardiac pacing ± Fab fragments

(14 digoxin /36 digitoxin /1 mixed; no significant differences)

	Number	Mortality rate
Pacing alone	23	17 %
Fab alone	12	25 %
Pacing + Fab	16	31 %

Taboulet P. *Clin Toxicol* 1993

Indication & dosage regimen of Fab fragments

Life-threatening conditions

- Ventricular arrhythmia : VF or VT
- Bradycardia with HR ≤ 40 /min despite atropine infusion (1 mg)
- Hyperkalemia > 5 mmol /L
- Cardiogenic shock
- Mesenteric infarction

Molar neutralization for curative treatment

Poor prognosticators

- Male
- Age over 55 years
- Underlying heart disease
- Atrioventricular block
- Bradycardia with HR < 60 /min despite atropine infusion (1 mg)
- Hyperkalemia > 4.5 mmol /L

Half-molar neutralization for prophylactic treatment

Curative/prophylactic strategy of Fab fragments administration

(N = 141)

First-line therapy with Fab fragments in patients with digitalis poisoning was associated with a low mortality rate (7.5%) without increase in cost, vial number, and duration of ICU stay

	Age (yrs) (gender)	History	Other Toxins	Overdose	Glycoside	Serum Concentration (ng/mL)	K (mmol/L)	ECG	Fab Dose (vials)	Time Before Fab (hrs)	Time Fab to Death (hrs)	Cause of Death
1	61 (M)	Cardiac failure	Verapamil	Voluntary	Digoxin	23.4	4.6	VF	12	?	<1	Cardiac failure
2	90 (F)	Cardiac failure, diabetes	None	Treatment	Digoxin	7.5	4.6	AVB I	2	NA	72	MOF
3	82 (M)	Parkinson's disease, cardiac failure	Meprobamate, TCA	Voluntary	Digitoxin	230.0	4.7	VF	12	10	60	MOF
4	71 (M)	Cancer	None	Treatment	Digoxin	7.3	5.7	VF	3	NA	36	MOF
5	82 (F)	Cardiac failure, AF, and HTA	Betablocker	Treatment	Digoxin	4.6	4.7	AF	2	3	19	MOF

Death: sepsis, co-ingestion, post-cardiac arrest anoxia

Lapostolle F. *Crit Care Med* 2009

Non-responsiveness to conventional supportive treatments and antidotes

Difficulty to manage catecholamines
- epinephrine versus dobutamine -

F, 17 years, severe propranolol poisoning
Sedation + mechanical ventilation + FiO_2 100%

		Epinephrine 1.5 mg/h	Dobutamine 15 µg/kg/min	
BP	S	93	56	mmHg
	D	64	33	mmHg
	M	75	43	mmHg
P_{RA}		7	6	cmH_2O
P_{AP}	S	27	19	cmH_2O
	D	19	11	cmH_2O
	M	23	15	cmH_2O
P_{cw}		17	13	cmH_2O
Cardiac Index		1.4	1.8	$l/min/m^2$
Systemic resistances		50.3	20.3	UI

30 min later

Dramatic decrease in BP ...

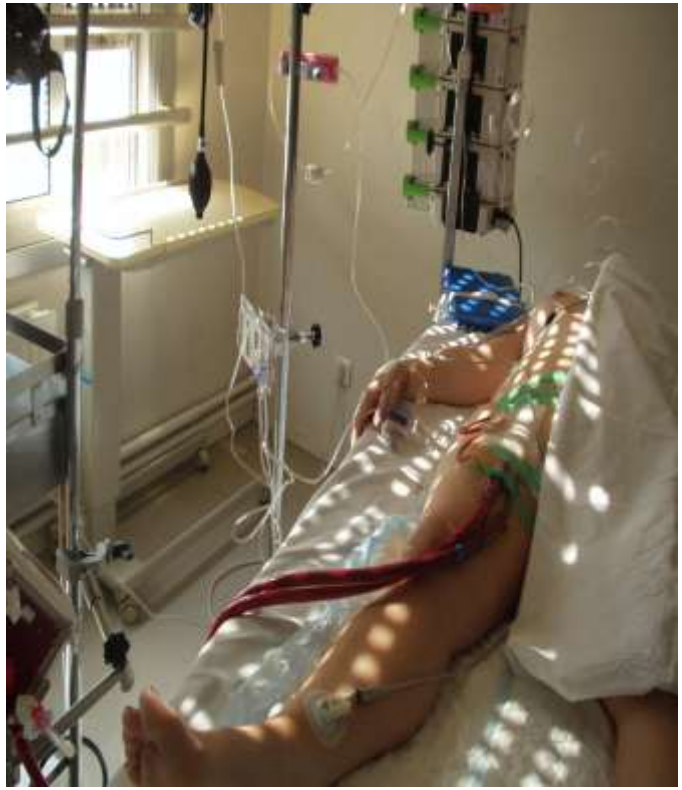
Fatal poisonings with cardiotoxic agents despite optimal pharmacological management in ICU

Toxicant	N	Mortality
Chloroquine	63	27%
Antidepressants	40	28%
Beta-blockers	23	22%
Flecainide	8	50%
Cocaine	3	33%
Total	137	28%

Cause of death : Refractory ventricular fibrillaton
Refractory asystole
Refractory cardiogenic shock
Brain anoxia
ICU-acquired complications

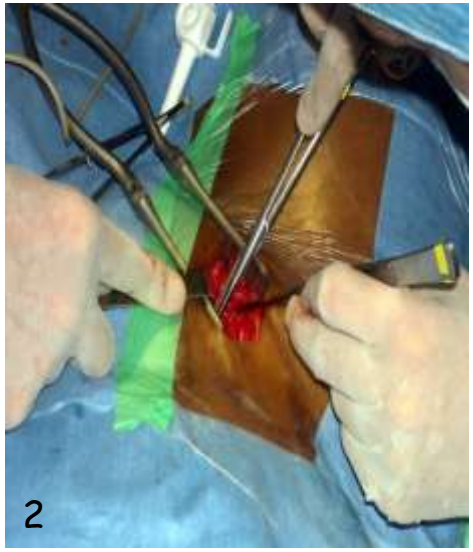
ECLS in cardiogenic schock

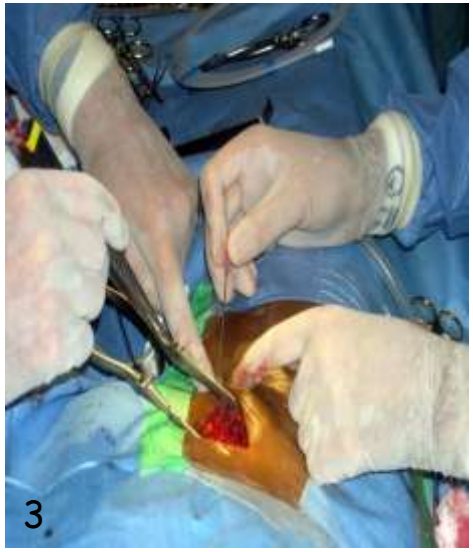
The purpose of ECLS is to take over heart function until recovery can occur, minimizing myocardial work, improving organ perfusion, and maintaining the renal and biliary elimination of the toxicant.

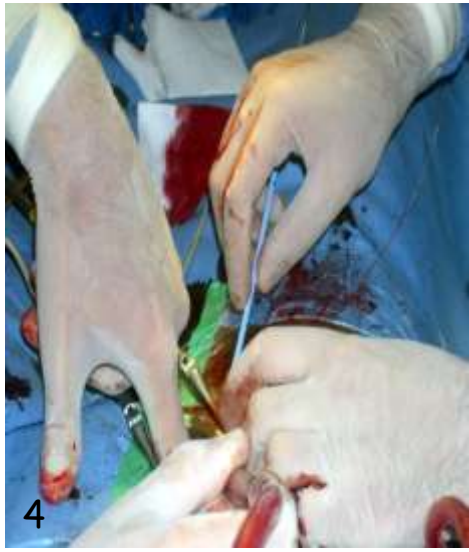


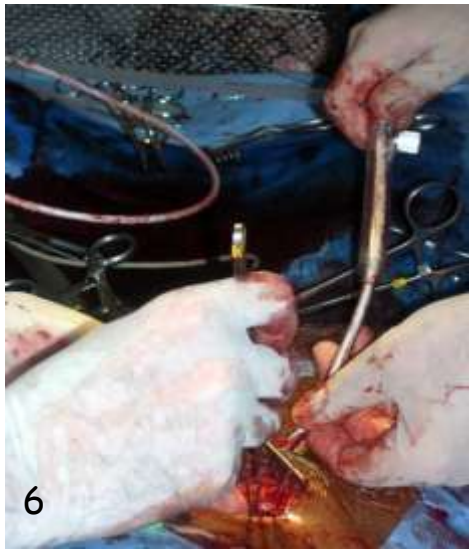
Baud FJ. *Crit Care* 2007

Cannulation of femoral vessels in medical ICU





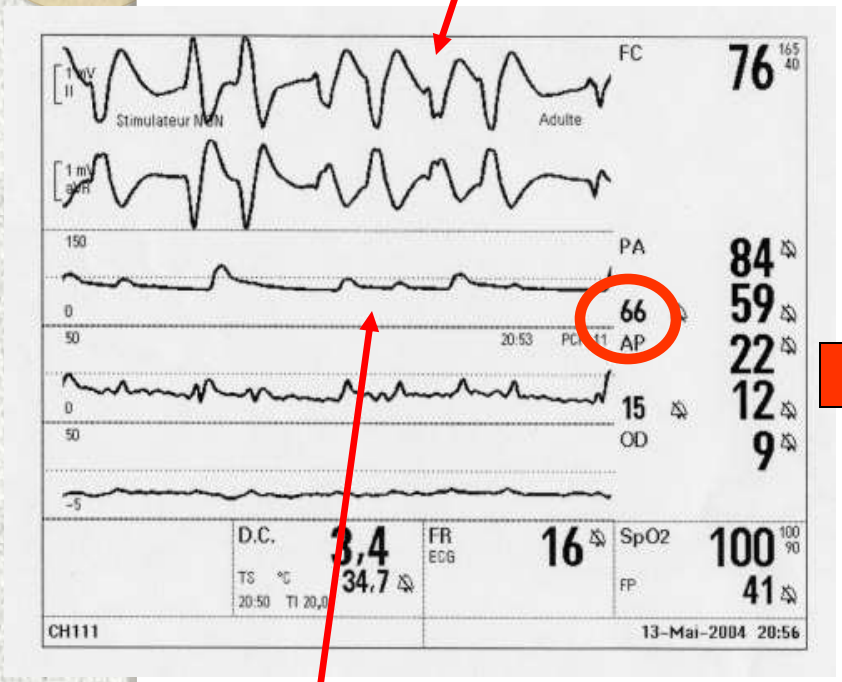




Mégarbane B. *Intensive Care Med* 2007

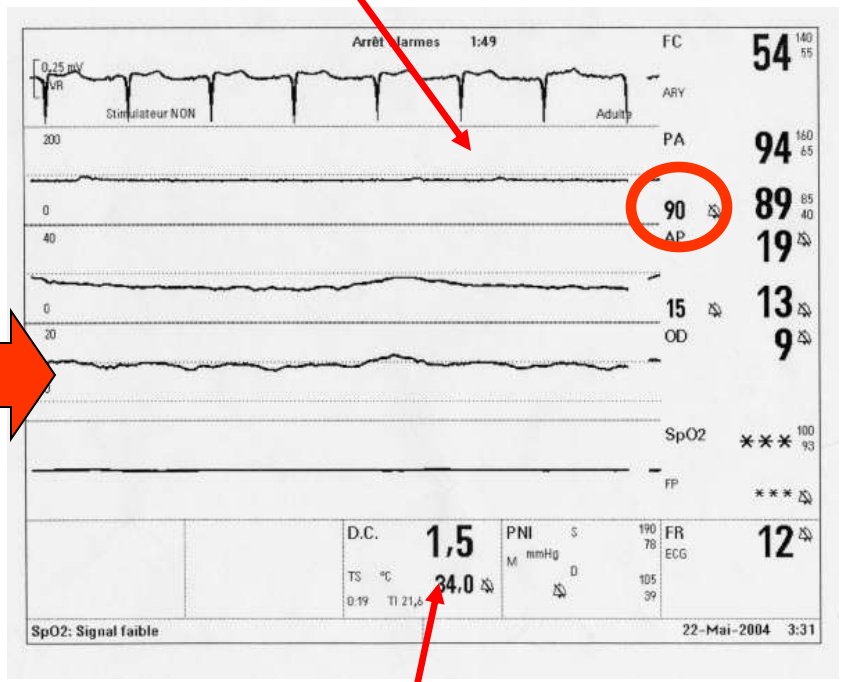
ECLS monitoring in ICU

Spontaneous cardiac rhythm



Severe hypotension despite high dose catecholamine

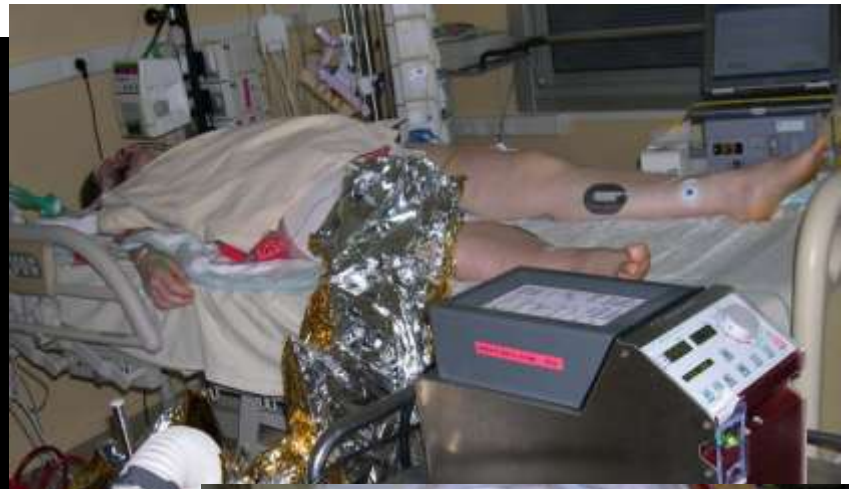
ECLS completely dependent cardiac flow (around 5-6 l/min)

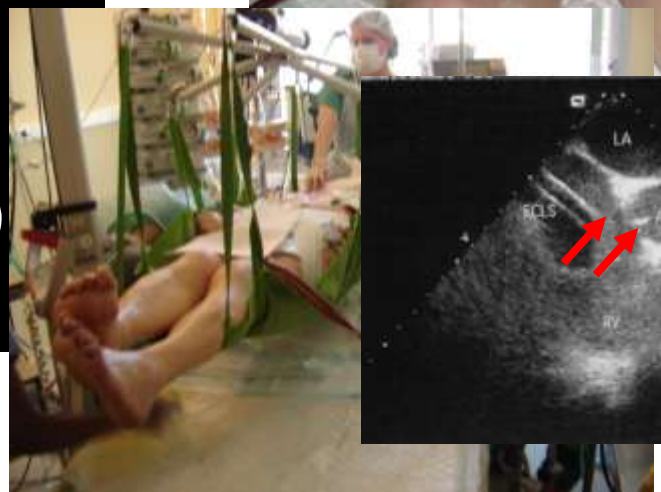


Spontaneous cardiac flow

Monitoring of an ECLS-treated poisoned patient in ICU

- Efficient anticoagulation:
  heparin to obtain ACT = 2N
- Catecholamines
  for mean BP = 60-70 mmHg +
  dobutamine to facilitate LV discharge
- Adequate transfusions
- Adapted Mechanical ventilation
- Temperature control
- Canulated lower limb monitoring (NIRS)
- Echocardiography:
  weaning criteria
- Neurological evaluation (EEG, clinical)
- Care, nursing





Outcome of 57 poisoned patients treated with ECLS

	Total (N = 57)	Cardiac failure (N = 26)	Refractory arrest (N = 31)
Survival	16 (28%)	12 (46%)	4 (13%)
Neurological sequellae	4	3	1
Hemorrhagic accidents	9	2	7
Thombo-embolic complications	3	2	1
Lower limb ischemia	4	3	1

Multivariate analysis of the prognostic factors of death in 57 poisonings treated with ECLS

ECLS indication for refractory cardiac arrest, plasma AST level, and plasma bicarbonate concentration were the 3 independent predictive factors of death ($p < 0.0001$)

	Odds Ratio	95% Confidence interval
Refractory cardiac arrest	5.8	[1.6 - 21.3]
AST > 750 IU//l	9.0	[1.1 - 75.2]
Plasma bicarbonate concentration < 16.0 mmol/l	11.8	[1.4 - 97.4]

ICU management of severe poisonings with medications or illicit substances

- Cardiovascular collapse or shock is a life-threatening complication. Determination of the underlying mechanism (hypovolemia, vasodilatation, contractility disorders) is essential to guide the treatment. In severe poisonings, invasive or noninvasive hemodynamic investigations are warranted.

- When conventional treatments fail in patients with persistent circulatory arrest or refractory shock, ECLS should be considered.

SRLF expert recommandations. *Réanimation* 2006

Conclusions :

- Shock and arrhythmias following poisonings with cardiotoxicants (especially with digitalis, sodium-channel, and calcium channel blockers) are frequent and may lead to life-threatening symptoms and death.
- Adequate monitoring of severity and assessment of prognostic criteria are mandatory to improve patient management.
- Treatment is mainly supportive. Despite the absence of high-level of evidence, administration of antidotes is life-saving.
- Peripheral ECLS may represent the unique solution in patients admitted for severe poisonings with non-responding arrhythmias or cardiac arrest. Its definitive benefit should be prospectively evaluated on a larger cohort.