Management of cardiotoxicant poisonings: indications of ECLS

Bruno Mégarbane, MD, PhD

Medical and Toxicological Critical Care Department
Lariboisière Hospital, Paris-Diderot University
Paris – France

bruno.megarbane@lrb.aphp.fr

Poisonings with cardiotoxicants

- In the USA: AAPCC 2012
  Cardiovascular agents: 8th cause of exposures (3.5%) but 2th cause of death (fatality rate: 0.27%)
- As usual no European data

January 1998 to October 2002
3,922 patients

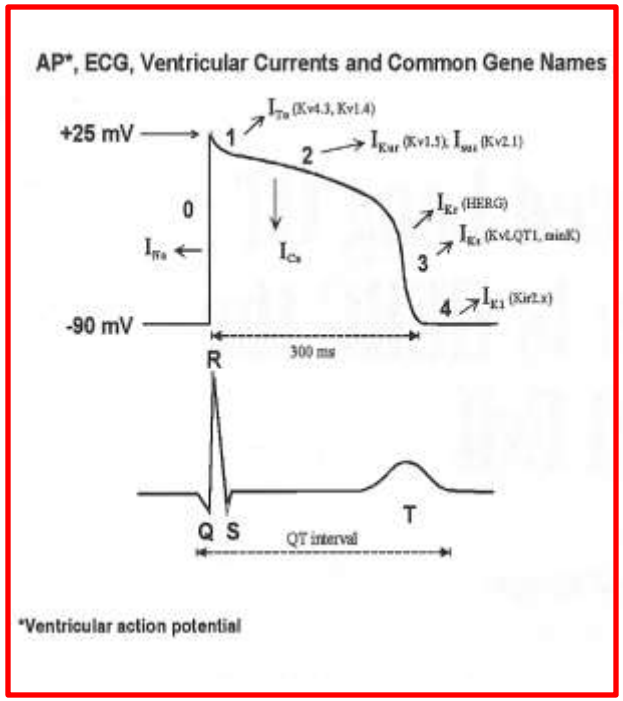
	N	Mortality rate
Poisoned patients	1,554	60 (4 %)
Cardiac complications (severe arrhythmias or failure)	164 (11 %)	37 (22 %)

Lariboisière Hospital ICU, Paris, France

Cardiotoxicants

A larger entity than cardiovascular drugs

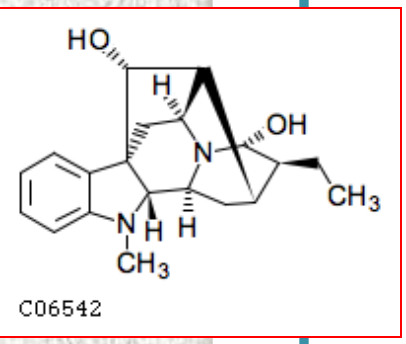
- **Cardiovascular pharmaceuticals**
  - Sodium-channel blockers (Class I)
  - Beta-blockers (class II)
  - Potassium channel blockers (sotalol) (class III)
  - Calcium-channel antagonists (class IV)
  - Cardioglycosides (class V)
- **Non-cardiovascular pharmaceuticals**: antipsychotics, antidepressants, 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

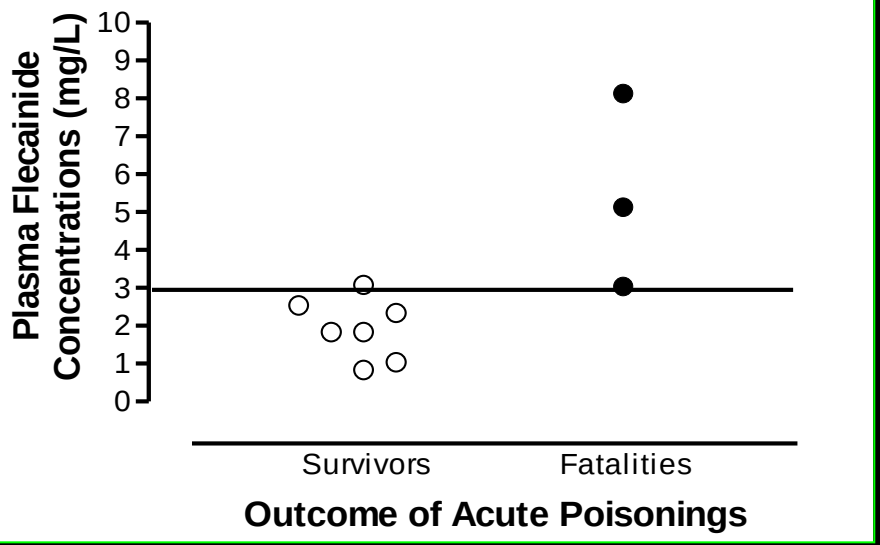
Ingested tablets	N	Cardiac arrest
1 g	7	0
2 g	13	1
3 g	16	8



Conso F. *Press Med* 1980

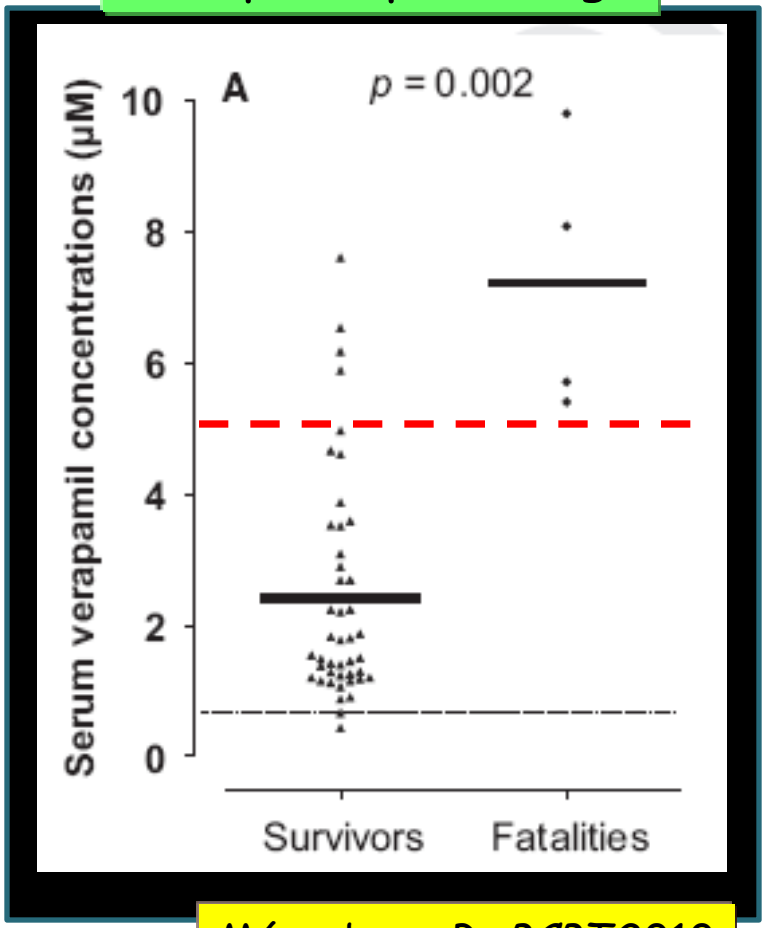
The prognostic value of plasma cardiotoxiant concentrations in acute poisonings

Flecainide poisonings



Mégarbane B. *Clin Tox* 2007

Verapamil poisonings



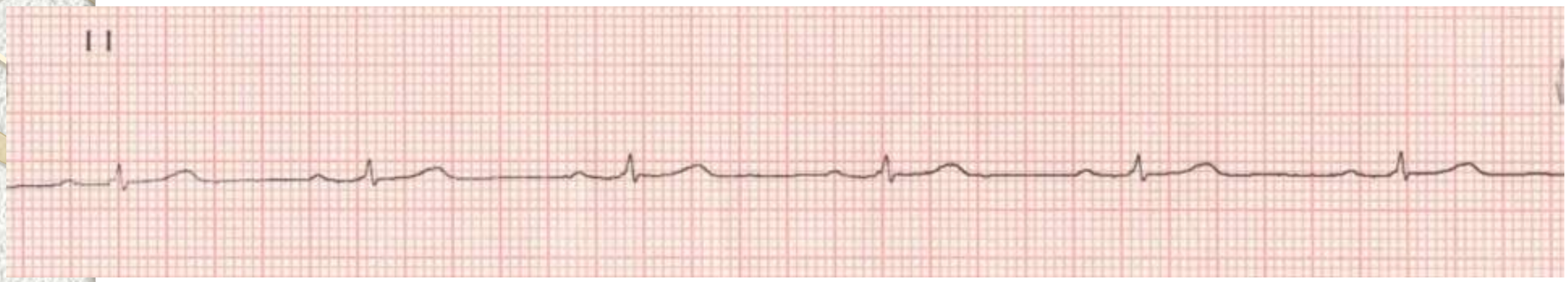
Mégarbane B. *BCPT* 2010

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

1

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

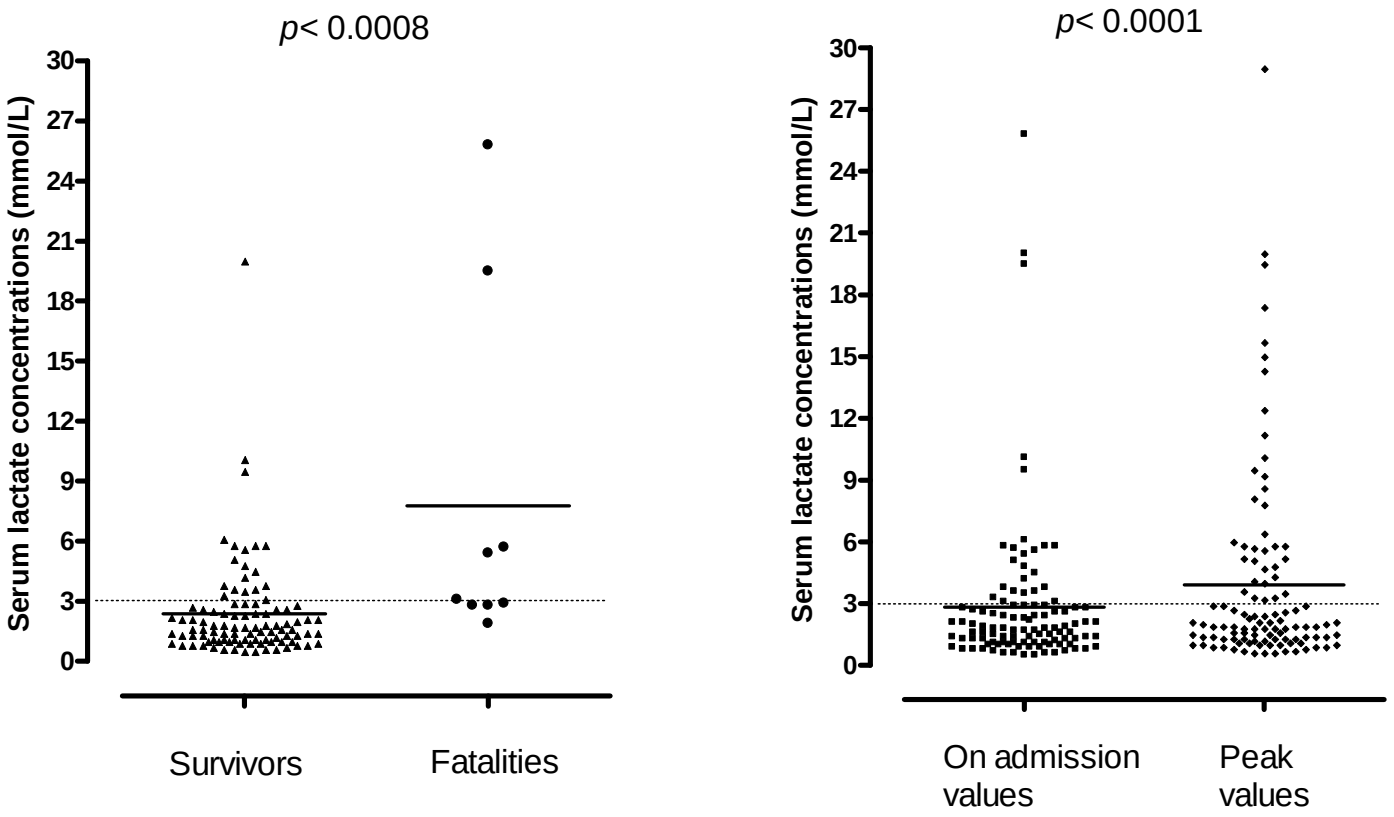
Beta Blocker	# Exposures	% Total Exposures	# Deaths	% Deaths
Propranolol*	22,334	43.9	27	71.1
Atenolol	13,587	26.7	6	15.8
Metoprolol	7,511	14.8	1	2.6
Nadolol	2,762	5.4	2	5.3
Labetalol*	1,907	3.7	0	0.0
Pindolol*	742	1.5	1	2.6
Timolol	686	1.4	0	0.0
Acebutolol*	584	1.1	3	7.9
Betaxolol	373	<1.0	0	0.0
Bisoprolol	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 (4)

Prgnostic value of lactate concentration on admission



The ROC-AUC of initial lactate for predicting mortality was 0.84 (0.74-0.94).

The cutoff point maximizing the sum of sensitivity and specificity was 2.7 mmol/L.

For the 3.0 mmol/L selected lactate cutoff point: 55% sensitivity, 80% specificity.

Mégarbane B. *Clin Tox* 2010

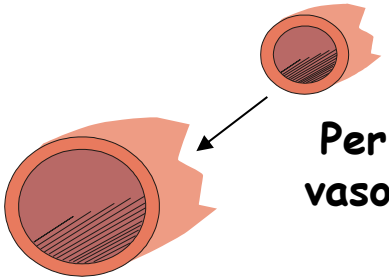
2

Calcium-channel antagonist poisonings (1)

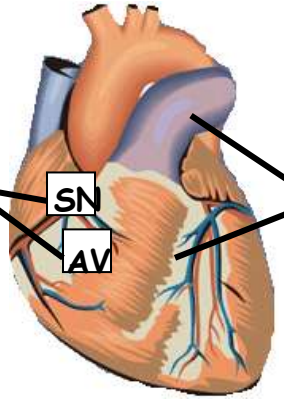
Toxicological consequences of pharmacological properties

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

Dihydropyridines: Selective vasodilators



Peripheral vasodilation

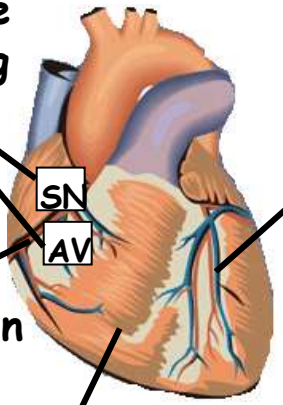


Potential reflex increase in HR, myocardial contractility and O_2 demand

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

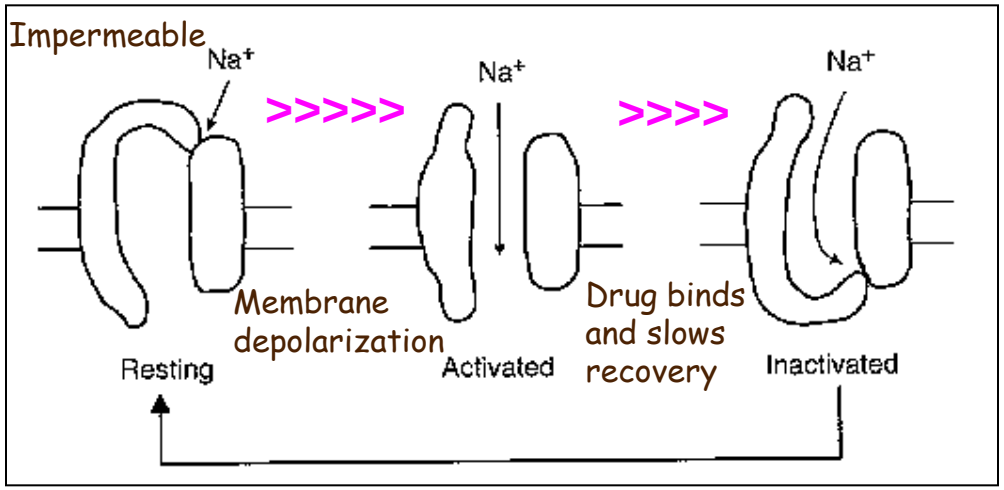
	Verapamil (N = 68)	Diltiazem (N = 27)	Nifedipine (N= 14)	Total (N = 109)
Hypotension	79%	89%	86%	84%
Bradycardia (< 60 /min)	56%	78%	43%	60%
Severe bradycardia (< 40 /min)	24%	26%	43%	60%
AV block	60%	63%	50%	60%
Complete AV block	53%	52%	21%	51%
Cardiac arrest	21%	22%	21%	21%
Death rate	25%	7%	7%	18%

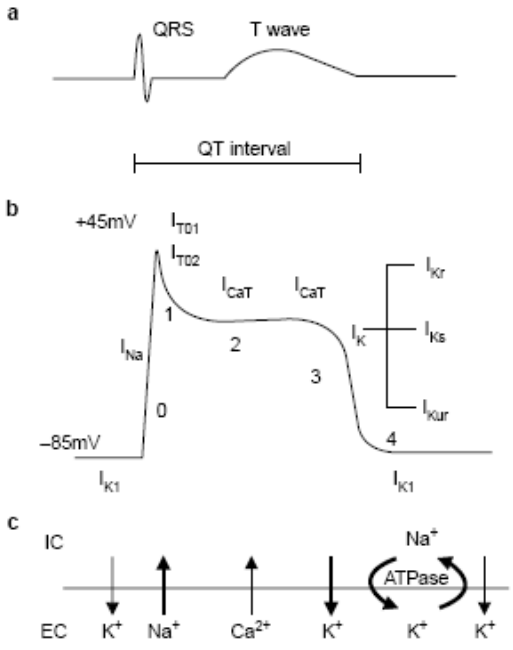
Sauder P. Intoxications aiguës. Elsevier, 1999

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**



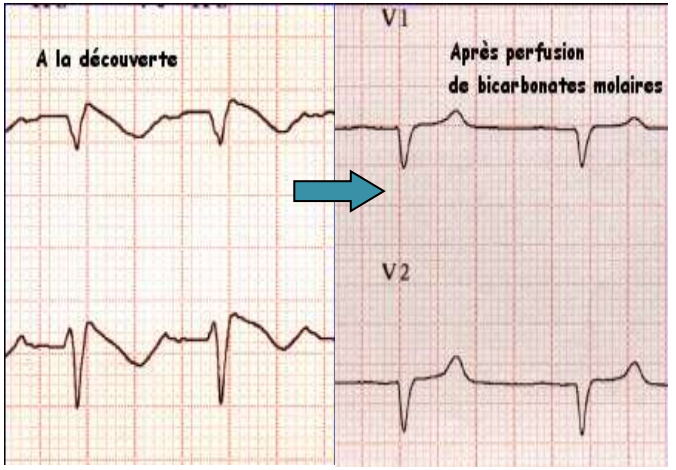


Poisonings with sodium channel blockers (2)
Clinical features

- **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

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

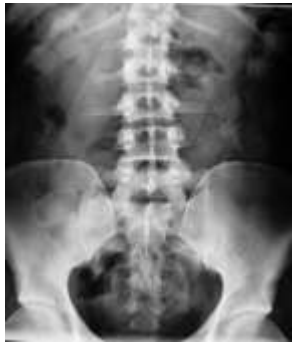
Boehnert MT. *N Engl J Med* 1985

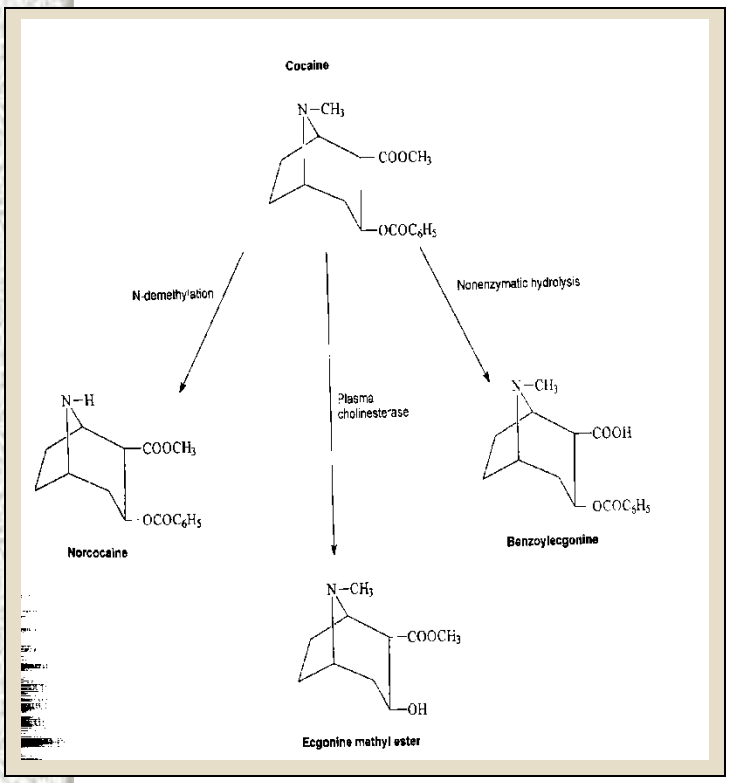


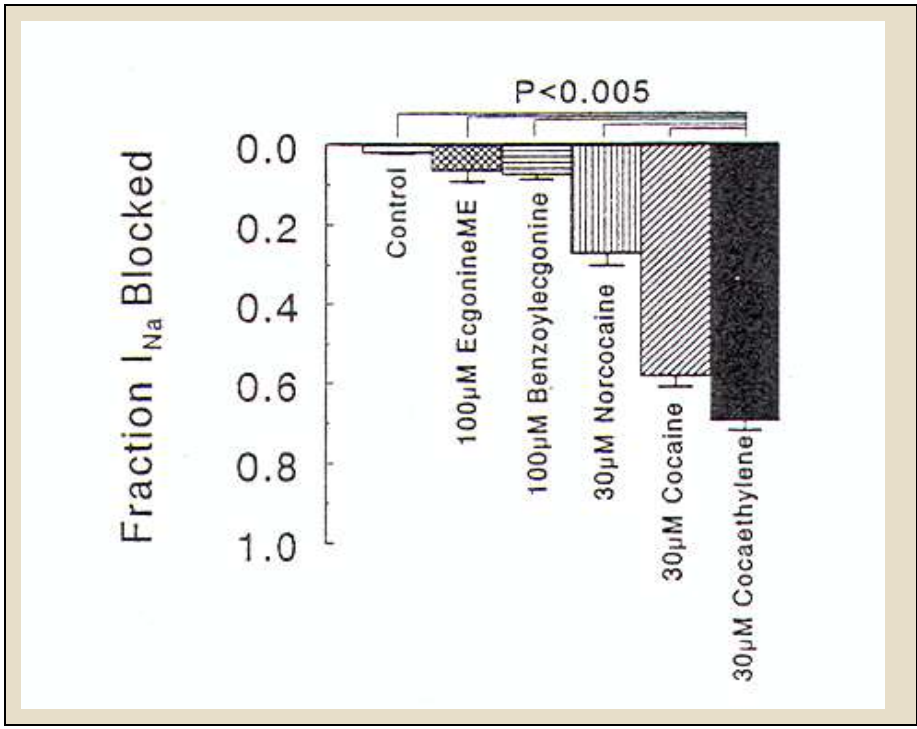
Brugada syndrome

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

4

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

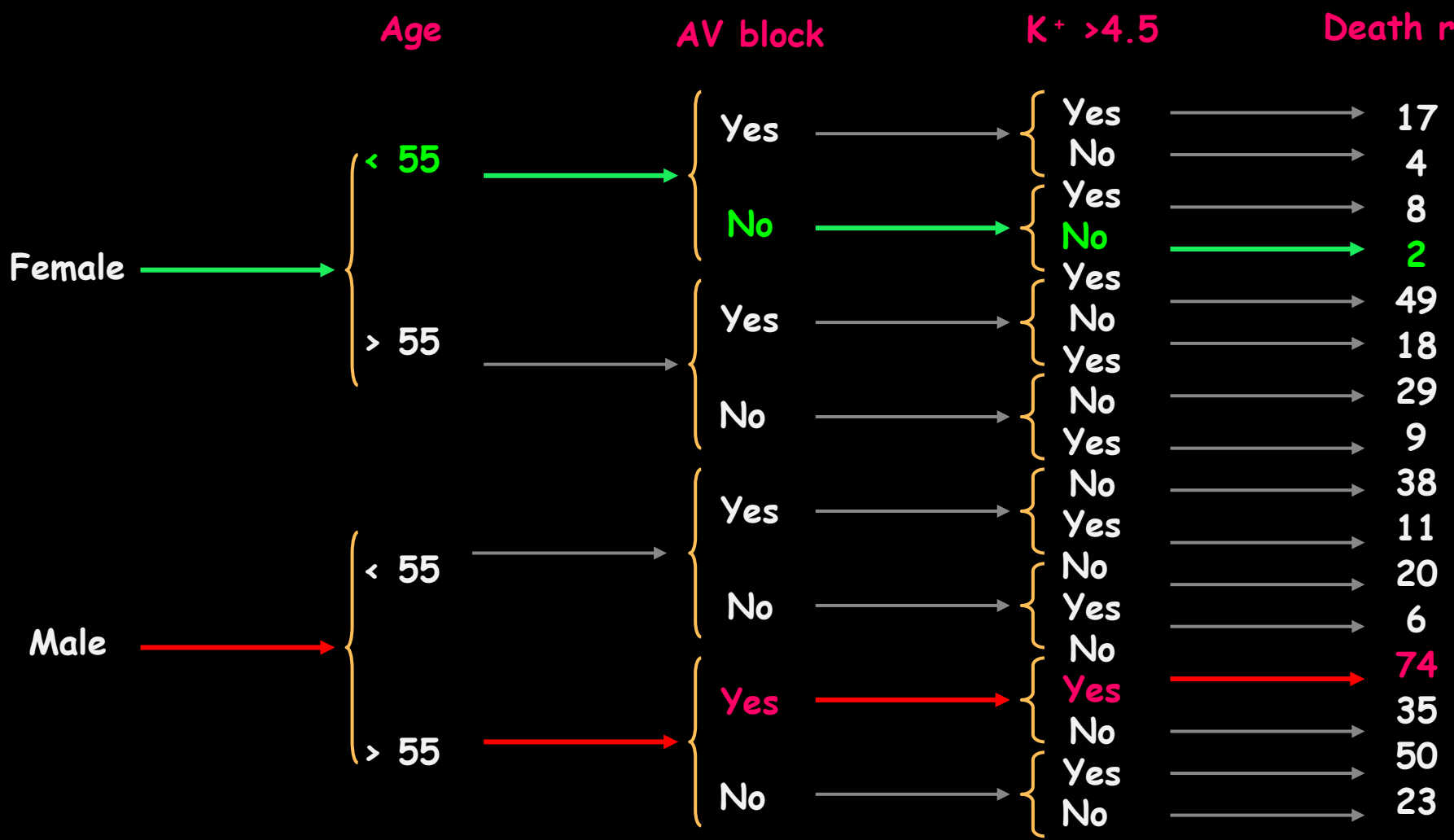
Headache, dizziness, confusion, delirium, and occasionally hallucinations

Rarely intestinal none occlusive infarction

- Blood pressure preserved while cardiac dysfunction possible

- ECG: Sinus bradycardia, ST-scoop, AVB
  Arrhythmias may are responsible for mortality

Main prognostic factors



Dally S. *Press Med* 1981

Management of drug-induced cardiac failure and arrhythmias

Strategy of management of toxic cardiovascular failure

Diagnosis of shock

↓

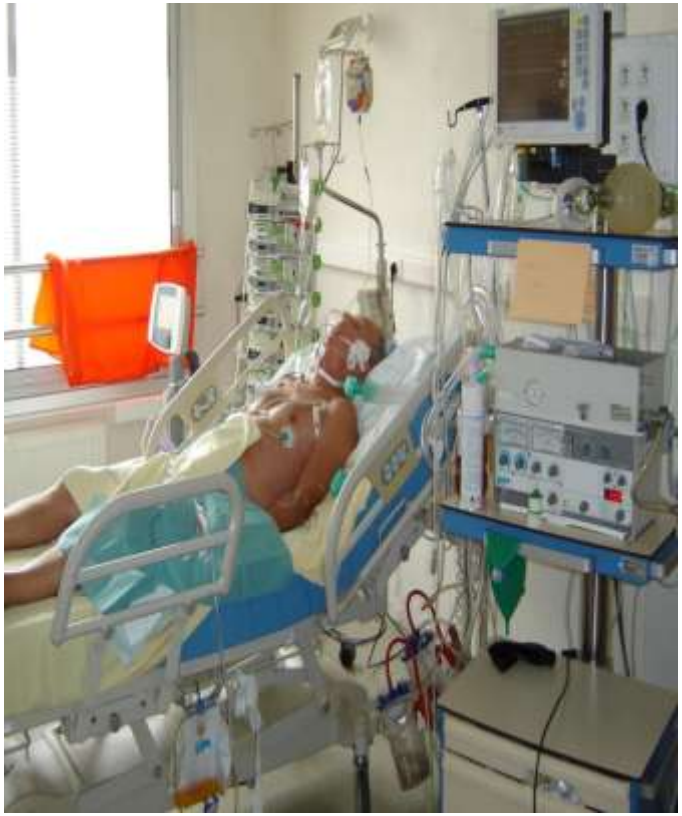
Determination of the mechanism of shock

↓

Definition of the optimal treatment

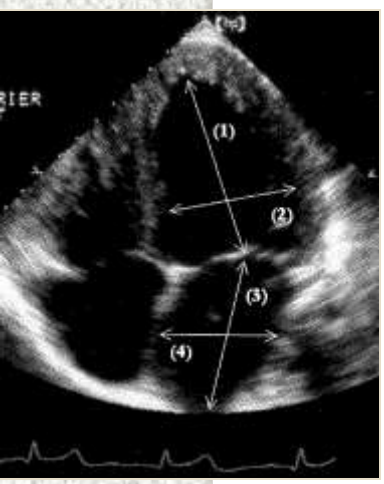
↓

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

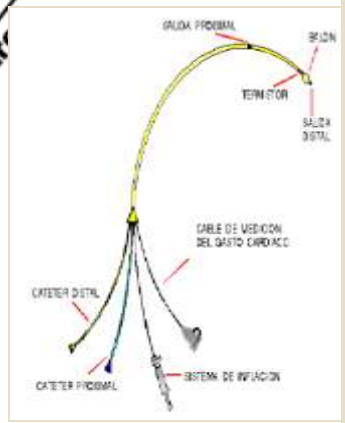
Low GEF (< 18%)

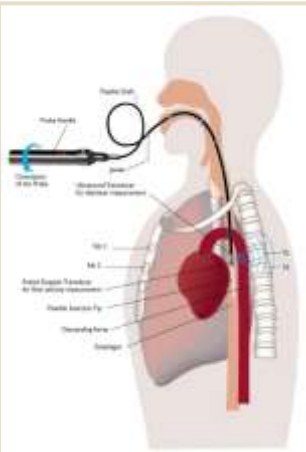
Hemodynamic evaluation:
CO, GEDV, GEF, PPV, SVV

Monitoring requir

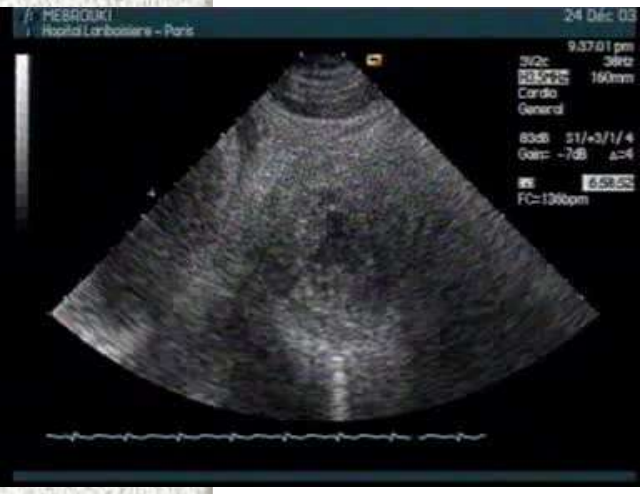
Specific ttt
(thrombolysis, surgery…)

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

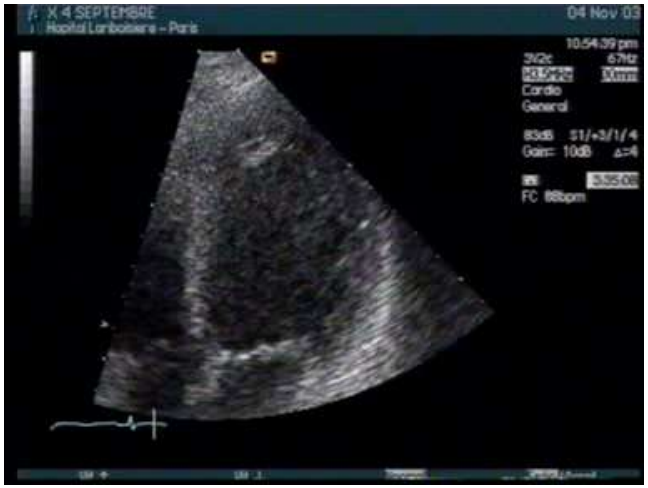




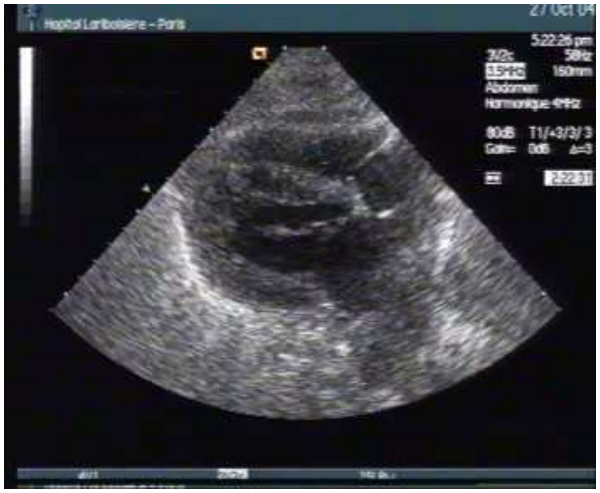
Echocardiography aspects



Hypovolemia
or vasoplegia



Cardiogenic shock



Severe
dysrhythmia

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

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

Chloroquine poisoning: prognosis assessment

	Supposed ingested dose		Systolic BP		QRS duration
Severe	≥ 4 g	or	< 100 mmHg	or	> 0.10 s
Non severe	< 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

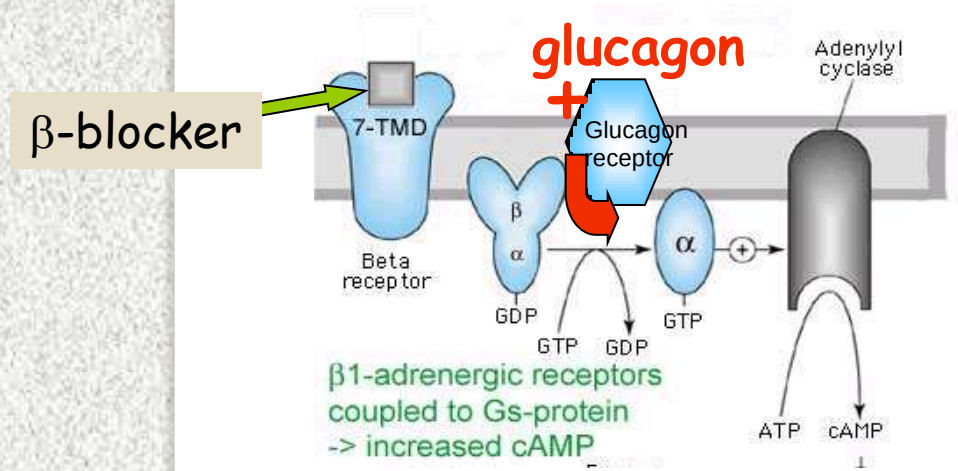
- **Dialysis:** limited interest as
  - Elevated protein binding
  - Elevated distribution volume
  - Liposolubility
  - Elevated endogenous clearance

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 1 UI/kg/h infusion + adequate glucose

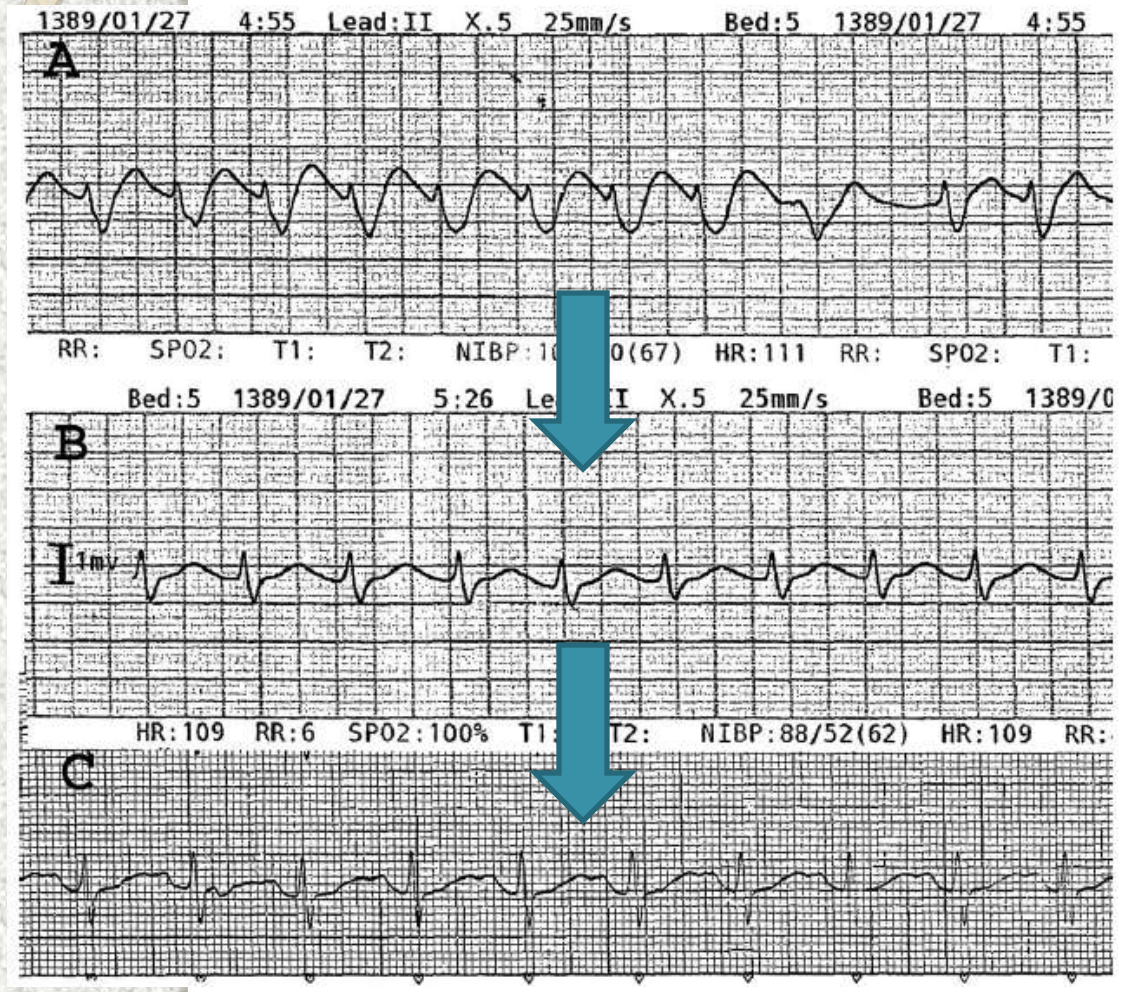
Yuan TH. *J Toxicol Clin Toicol* 1999
Boyer EW. *N Engl J Med* 2001

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

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

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%

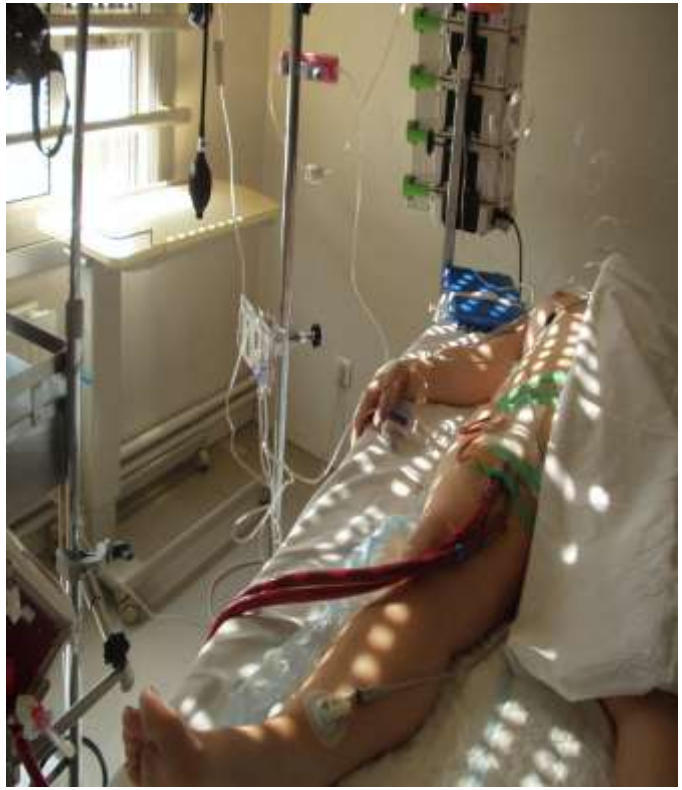
		Epinephrine 1.5 mg/h	Dobutamine 15 µg/kg/min	
BP	S	93	56	mmHg
	D	64	33	mmHg
	M	75	43	mmHg
P_{RA}		7	6	cmH_2O
P_{AP}	S	27	19	cmH_2O
	D	19	11	cmH_2O
	M	23	15	cmH_2O
P_{cw}		17	13	cmH_2O
Cardiac Index		1.4	1.8	$l/min/m^2$
Systemic resistances		50.3	20.3	UI

30 min later

Dramatic decrease in BP ...

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

Specialized team in ICU

Toxic cardiac arrest or failure is announced

Warning

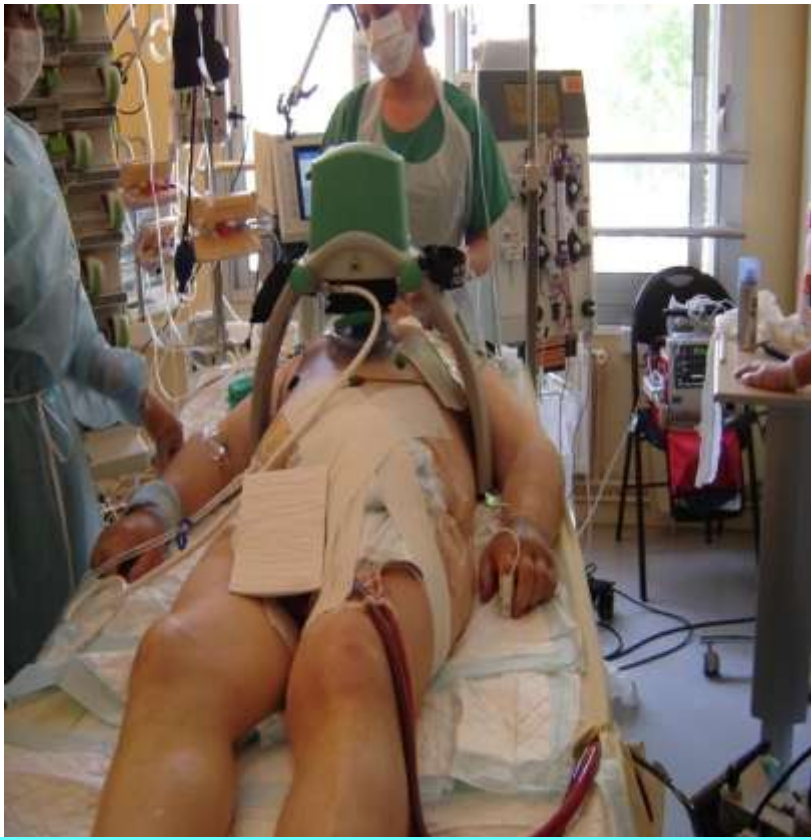
Perfusionist — Surgeons — ICU Physician — Cardiologists — Nurses

DISPONIBILITY

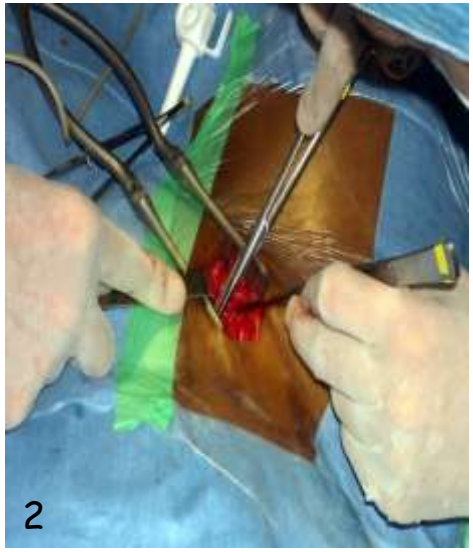
morning — night

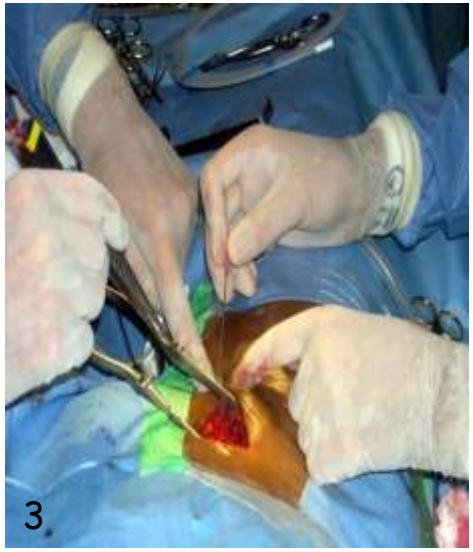
working days — week ends

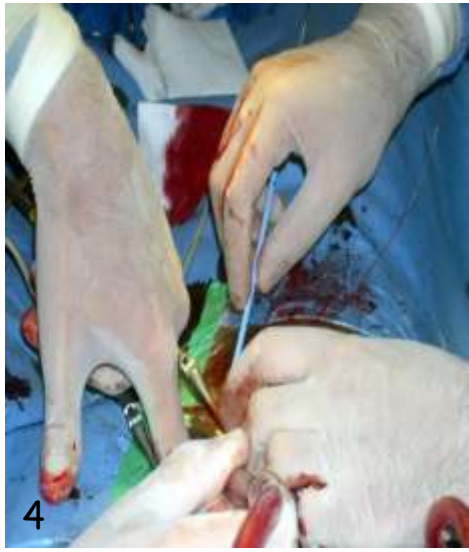
Adequate cardiac massage and ACLS are the keys for good prognosis in toxic cardiac arrest

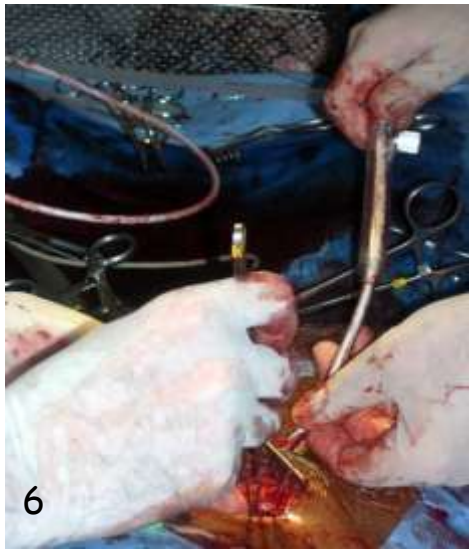


Cannulation of femoral vessels in medical ICU





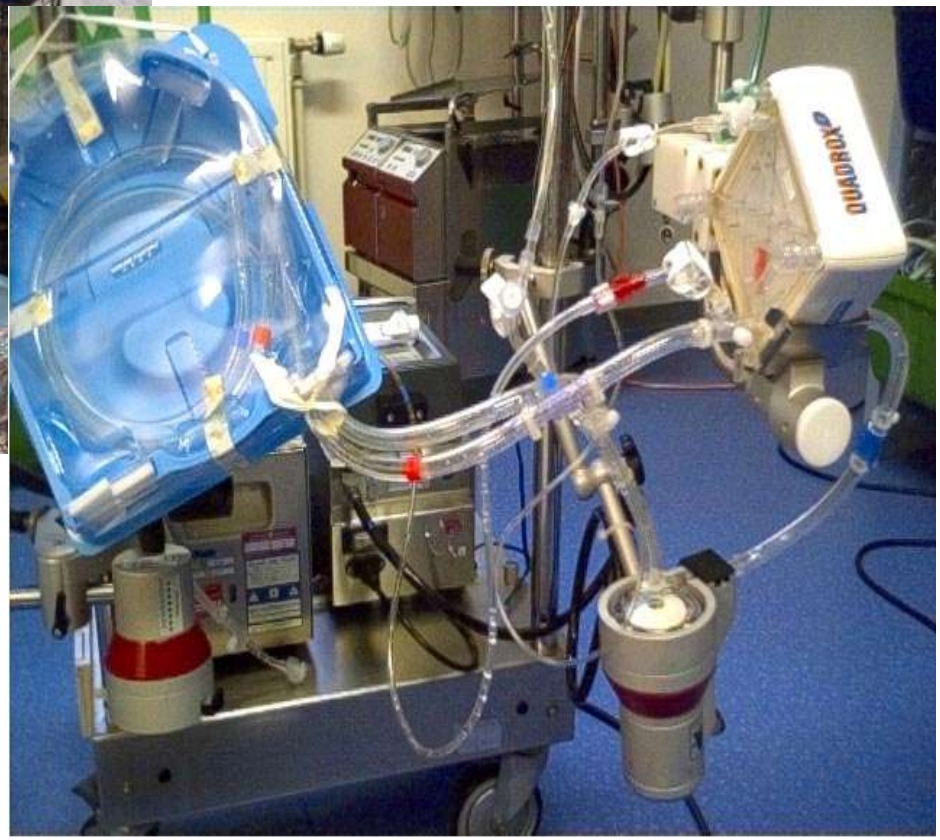




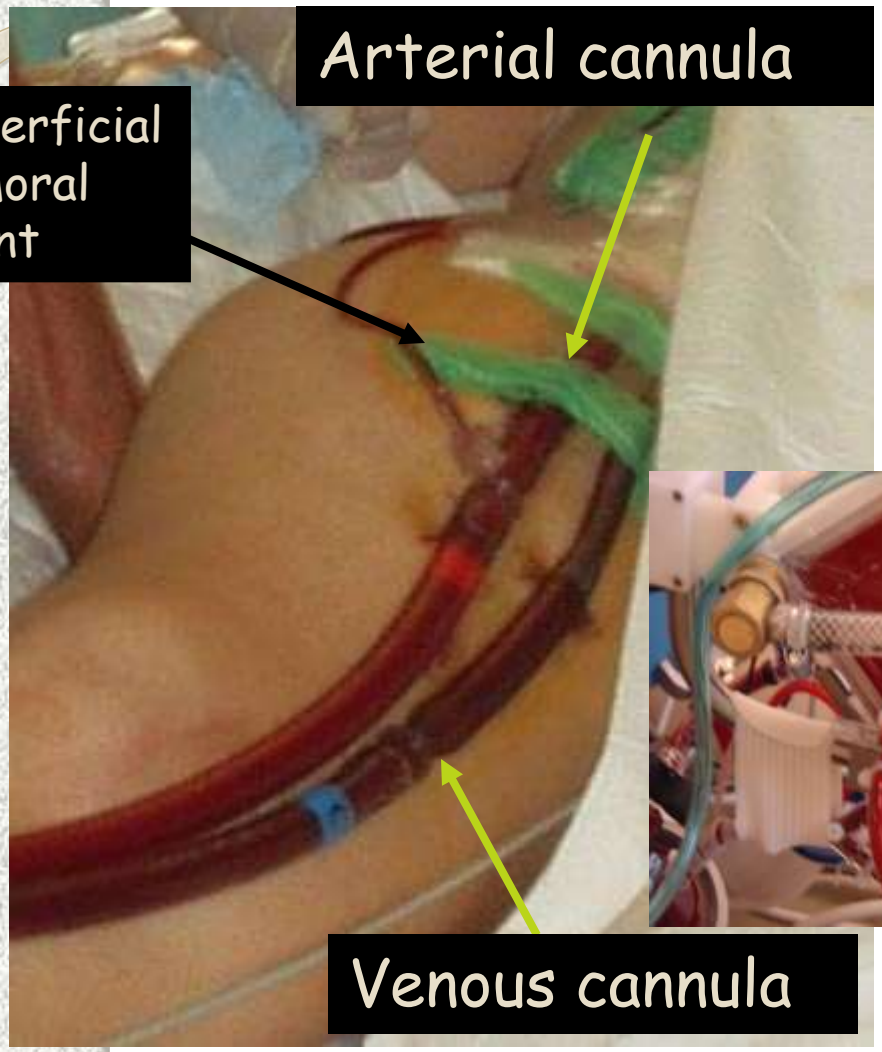
Mégarbane B. *Intensive Care Med* 2007

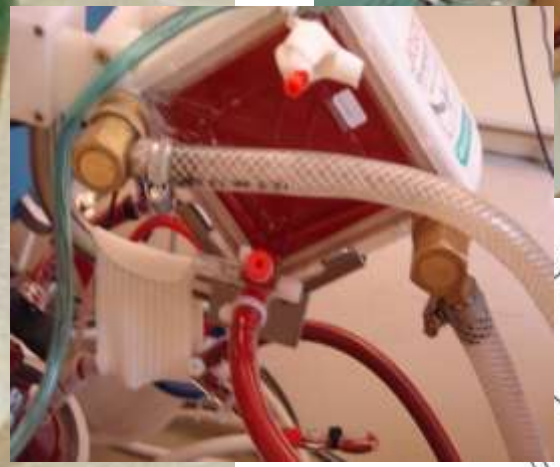
ECLS in the toxicological ICU

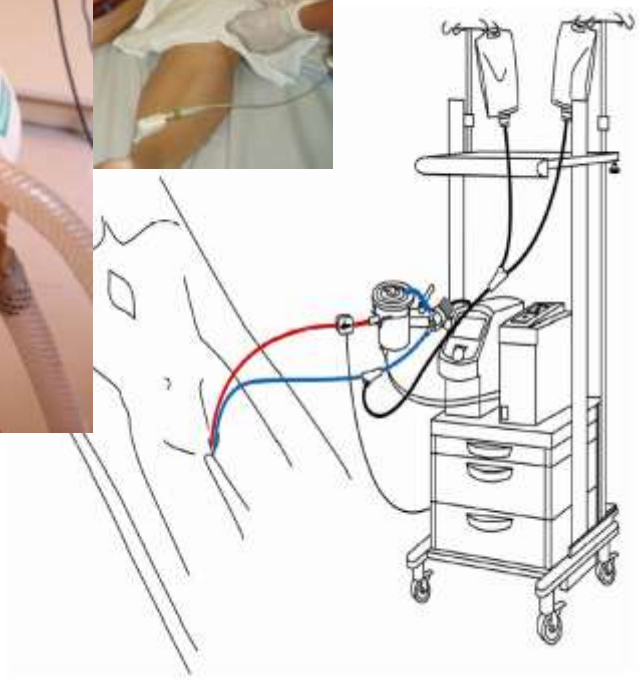
ECLS device preparation



ECLS in medical ICU



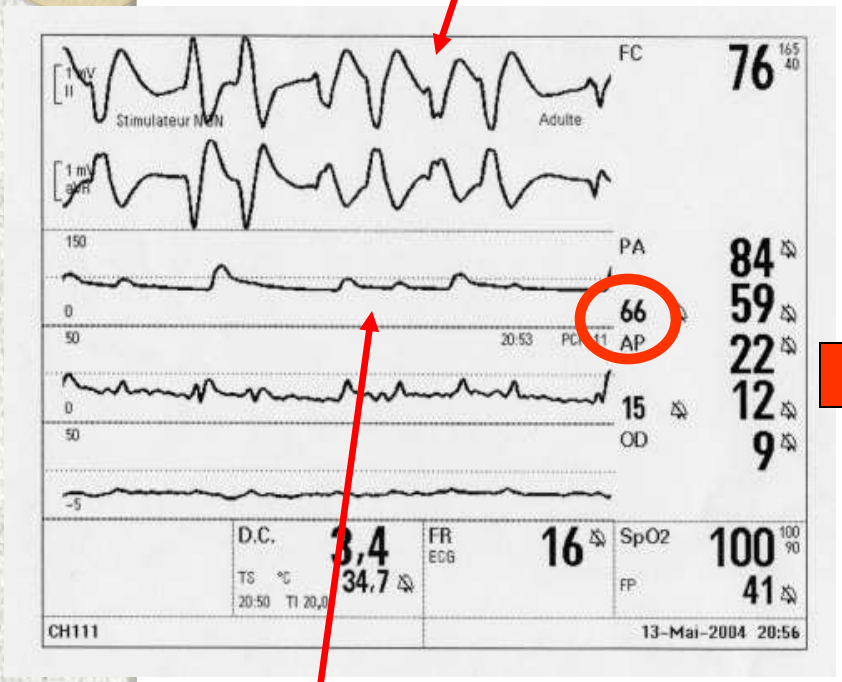




Babatasi G. *Arch Mal Cœur Vx* 2001
Mégarbane B. Intensive Care Med 2007

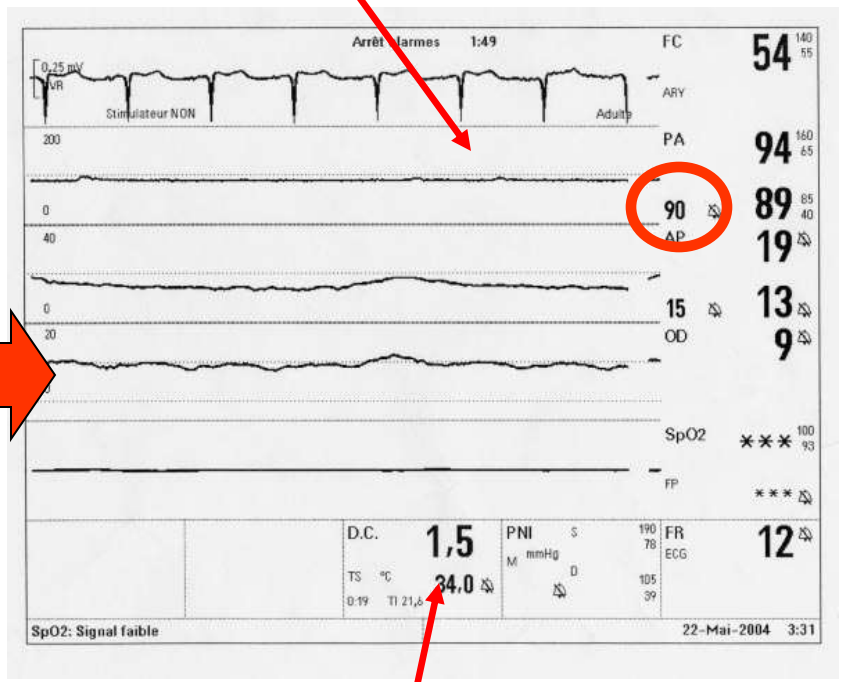
ECLS monitoring in ICU

Spontaneous cardiac rhythm



Severe hypotension despite high dose catecholamine

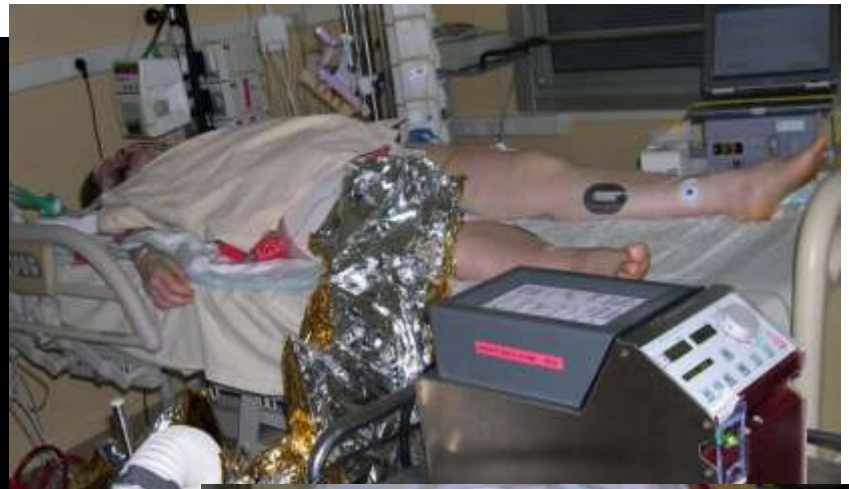
ECLS completely dependent cardiac flow (around 5-6 l/min)

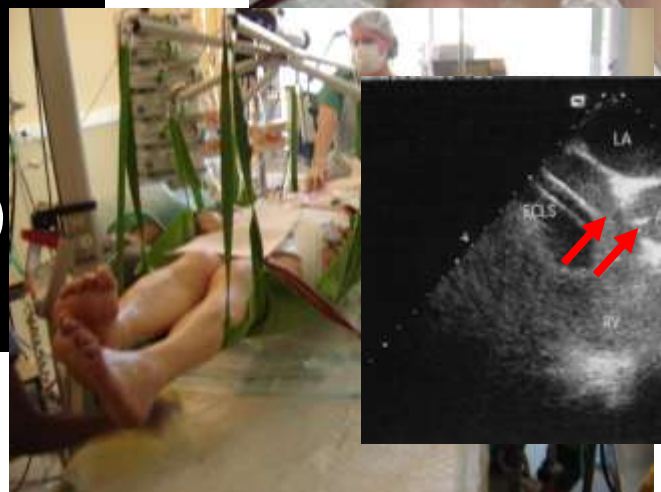


Spontaneous cardiac flow

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





Published cases of ECLS-treated acute poisonings:

- Beta-blockers
- CCB
- Sodium channel blockers

De Lange DW. *Clin Tox* 2013

Agent	References
Acebutalol	29,37
Amiodarone	38
Antidepressants (tricyclic)	15,29,39–41
Arsenic	42
Atenolol	29
Bisoprolol	29
Bupropion	43
Calcium Channel Blockers	1,44–49
Carbamazepine	29,50
Carbon monoxide	51
Chloroquine	15,52
Cibenzoline	29,53
Citalopram	29
Cocaine	54
Disopyramide	29,55
Diltiazem	29
Flecainide	29,56–58
Hydrocarbon products	59–63
Ibuprofen	64
Lidocaine	65
Mepivacaine	66
Methadone	67
Metoprolol	29
Opioids	67–69
Organophosphates	70
Paraquat	31,32
Paroxetine	29
Phosphine	71
Propafenone	15,29
Propranolol	29,72–74
Quetiapine	75
Quinidine	76
Radiocontrast material (intravenous)	77
Sotalol	29,78
Taxus	79
Venlafaxine	29
Verapamil	29
Zinc chloride	80
Zotepine	81

Case report (1)

Severe propafenone poisoning

F 50 years

H0 : ingestion of 9 g propafenone (RYTHMOL®, 30 pills)

H1 : GCS 4 + HR 50/min + non-measurable SBP + complete AV block

Intubation + isoprenaline + 11.2% lactate (250 ml) + 1.4% bicarbonates (1,000 ml)

In ICU :

Hypotonic coma then seizures (clonazepam + pentobarbital)

SBP 90/50 mmHg , HR 79 /min

ECG : AV block I, QRS 140 ms, RBBB, Brugada syndrome

Bio : Metabolic alcalosis (pH = 7.66 ; HCO_3^- = 42 mM)

PaO_2/FiO_2 : 246 mmHg, lactate : 3 mM, creatinine : 57 µM,

Propafenone concentration : 2.9 mg/l (N < 1 mg/l)

Cardiac failure (LVEF : 35%, cardiac output : 2.2 l/min)

despite epinephrine up to 5 mg/h and 8.4% bicarbonates

Case report (2)

Outcome in a severe propafenone poisoning

H3 : Renal failure : oliguria and creatinine of 107µM
Respiratory failure : PaO_2/FiO_2 ratio of 134 mmHg

H7 : ECLS with femoral cannulation
Anticoagulation with heparin
Assistance flow of 3.5 l/min with 2,800 turns/min.
Dobutamine : 10 µg/kg/min

H12 : Dissociation between electrical and mechanical activities
H48 : ECLS weaning

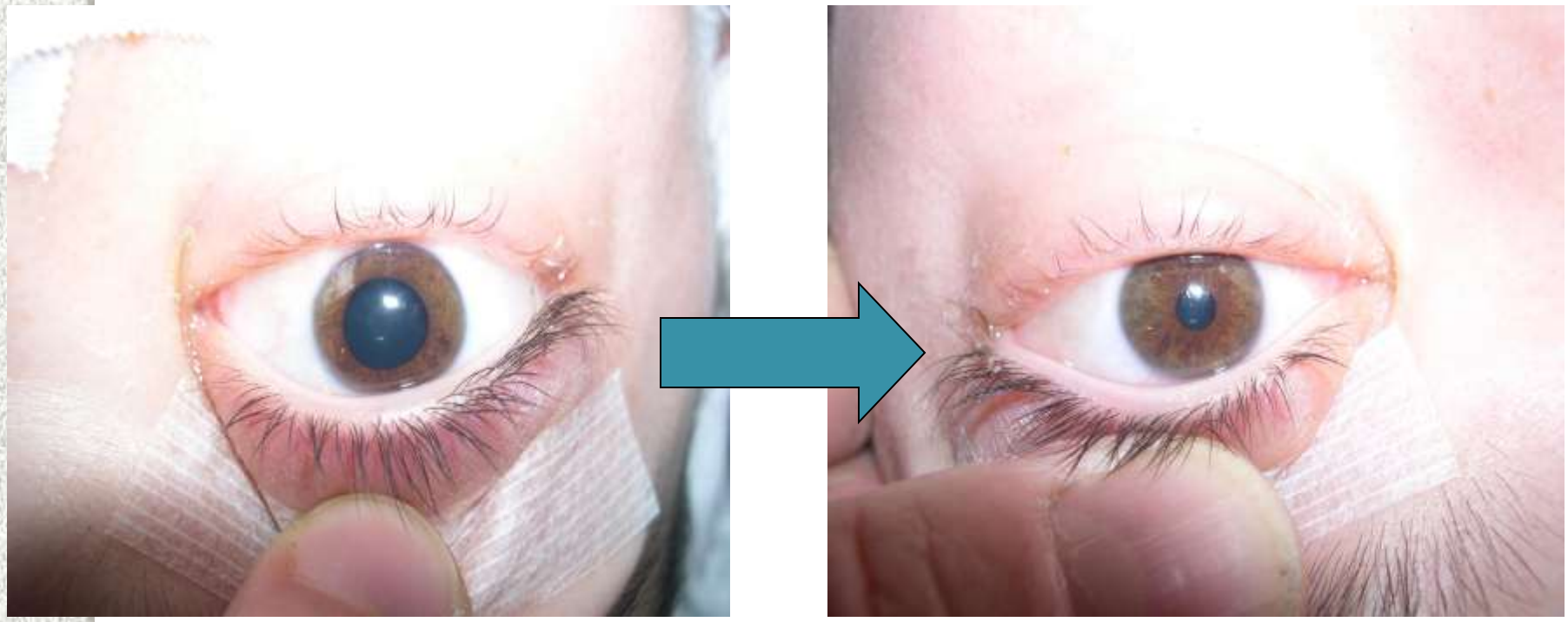
D4 : *P. aeruginosa* hospital-acquired pneumonia
D8 : Extubation
D22 : Return back home

M6: Normal life quality

Non-reactive mydriasis is not a sufficient reason to refuse ECLS

Initial non-reactive mydriasis

Photo-reactive pupils



On ICU admission

After ECLS

Case report (3)

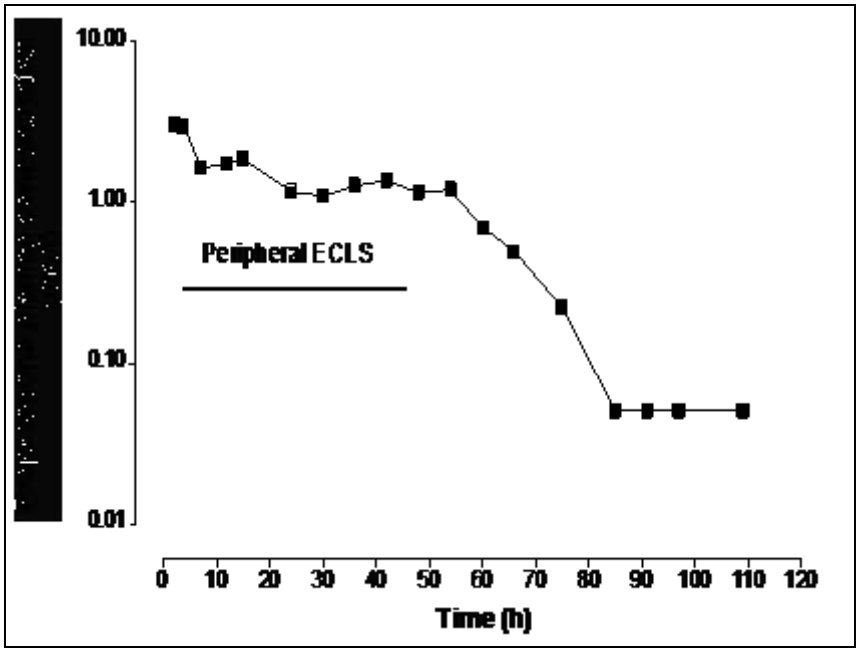
Assessment of ECLS interest in propafenone poisoning

	H3	H4	H12	H24	D2	D3
Spontaneous Q (l/min)	1.9	1.9	0	2.5	4.5	5.7
LVEF (%)	35	35	0	45	50	50
Assistance Q (l/min)	-	-	3.5	3.5	3	0
SvO_2 (%)	45	60	73	79	79	-
Plasma lactate (mmol/l)	8.3	-	4.6	1.8	0.9	0.9
Epinephrine (mg/h)	5	5	5	0.1	0	0
Dobutamine(µ/kg/min)	0	0	10	10	10	5

Case report (4)

Propafenone toxicokinetics in a severe poisoning requiring ECLS

- Elimination half-life: 30 h (pharmacology: 4 h)
- Volume of distribution: 151 l/kg
- Clearance: 262 l/h



Outcome of 57 poisoned patients treated with ECLS

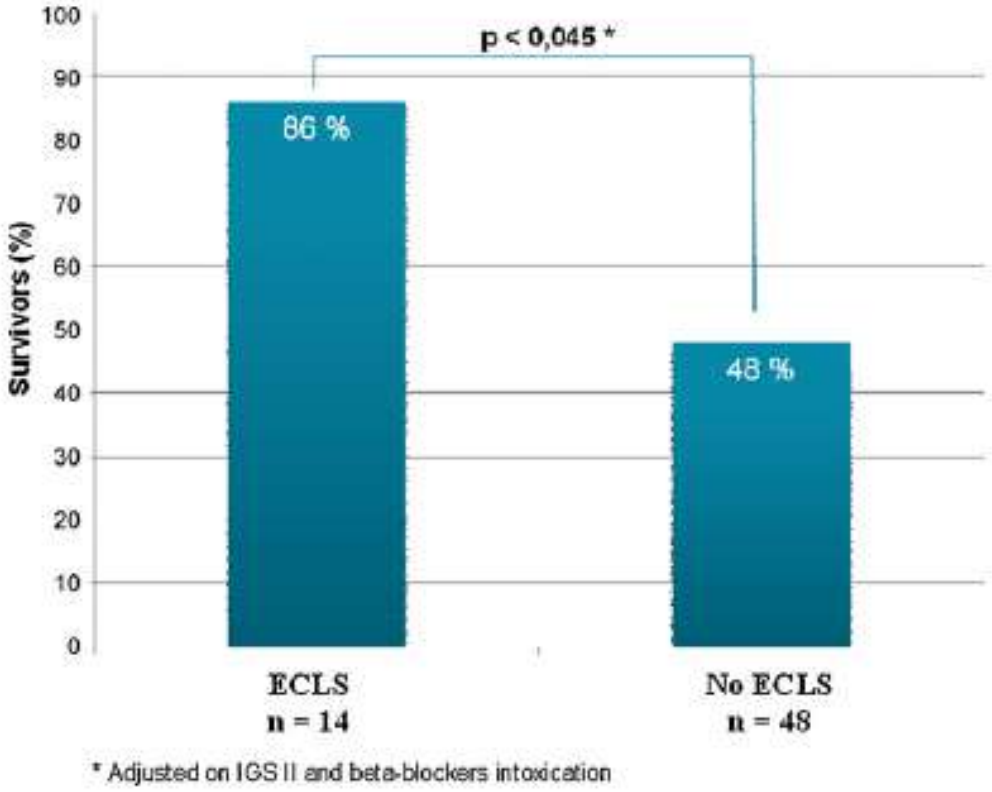
	Total (N = 57)	Cardiac failure (N = 26)	Refractory arrest (N = 31)
Survival	16 (28%)	12 (46%)	4 (13%)
Neurological sequellae	4	3	1
Hemorrhagic accidents	9	2	7
Thombo-embolic complications	3	2	1
Lower limb ischemia	4	3	1

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]

A comparison of survival with and without ECLS support treatment for severe poisoning due to drug intoxication



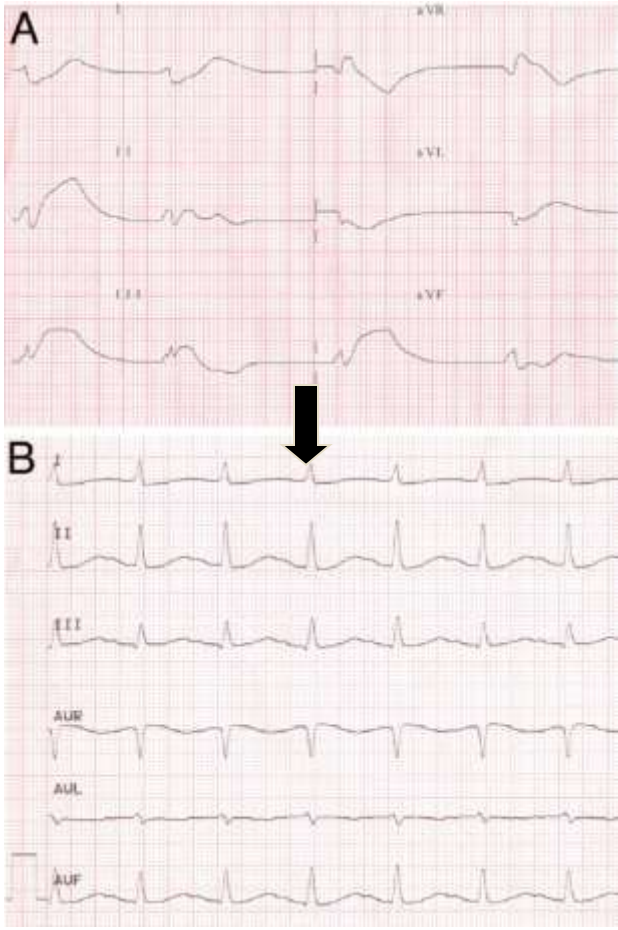
Masson R. *Resuscitation* 2012

Death of ECLS-treated poisoned patients

- Death resulted from multiorgan failure, anoxic encephalopathy or capillary leak syndrome if ECLS was performed under cardiac massage.

- Four patients presented documented brain death, allowing organ donation in 2 cases.

- The heart of one flecainide-poisoned patient was successfully transplanted, after normalization of ECG and myocardial function as well as toxicant elimination under ECLS.



Vivien B. Ann Emerg Med 2010

Capillary leak syndrome

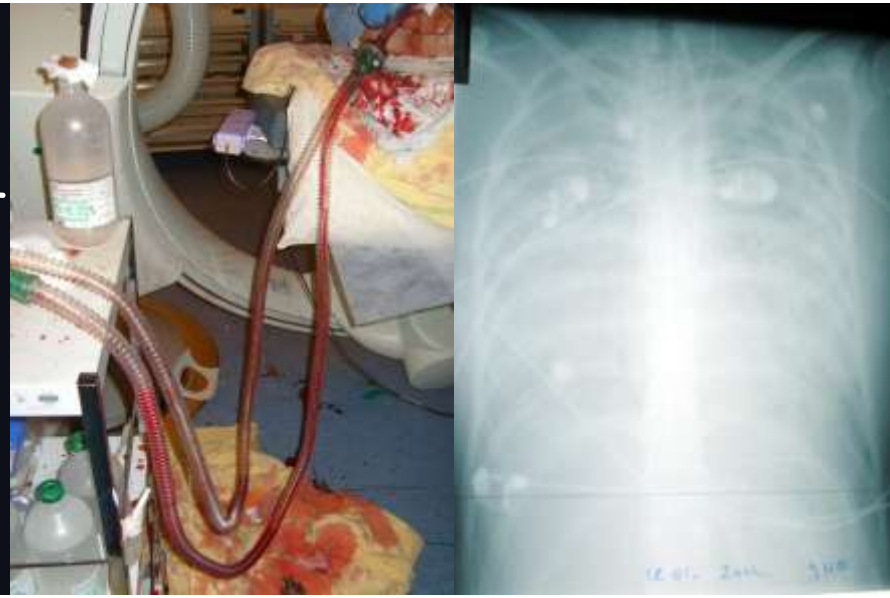
Fluid leak from the intravascular to the interstitial compartment
(increased capillary permeability due to endothelial injury)

Cause : prolonged cardiac arrest

Consequences :

- Generalized edema, increasing weight
- Alveolar hemorrhage
- ↘ ECLS efficiency
- Altered physical aspect

Final result : death



The thin lie between life and death …

Bracco D. Intensive Care Med 2007

Complications of ECLS

Adverse event	Reported range (%)
Related to ECMO	
Dysfunction of oxygenator	4–17.5
Pump malfunction	1–12
Rupture of the tubing	6–12
Air in circuit	1.6
Blood clots	
Oxygenator	2–12.2
Rest of the circuit	1–22
Cannula-related problems	8.4–12
Other mechanical problems	7.9
Not directly related to ECMO	
Bleeding	
Surgical site bleeding	13.6–36
Cannulation site bleeding	16.9–22
Pulmonary hemorrhage	6.5–10
Gastro-intestinal hemorrhage	3.8–10
Intracranial hemorrhage	1–7
Vaginal bleeding	9
Hemolysis	2.4–10
Disseminated intravascular coagulation	1.9–10
Culture-confirmed infection at any site (related or unrelated to ECMO)	10–21.3
Central Nervous system infections	2.8
Seizures	0.8–3
Leukopenia	1.2–3
Limb ischemia (in VA-ECMO)	19–21
Thrombocytopenia	51

De Lange DW. *Clin Tox* 2013

ECLS availability in France

- Poisonings are admitted to ERs and ICUs
- 2/3 of general and university hospitals are lacking from department of cardiac surgery
- Requirement of experience to perform ECLS for the whole team of care-givers

Development of mobile ECLS units

J. Théodore, 2008

Mobile ECLS unit

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.