SHOCK…SHOCK…SHOCK

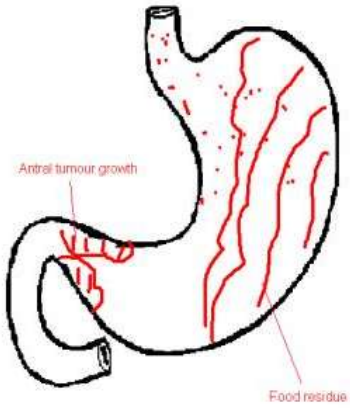
Dr KK CHAN
PMH/YCH ICU
24 May 2011
CCM inter-hospital grand-round

Case

- M/60
- Newly diagnosed Cancer of stomach
  - Presented with gastric outlet obstruction
  - Epigastric pain, vomiting, haematemesis

OGD

Exam. Date/Time: 10/12/2010 09:50
Service Location: Endoscopy Room
Condition: Elective
Team: General Surgery Team 3
Endoscopist / Nurse:
[C]
[N]
([C] = Chief; [S] = Supervisor; [E] = Endoscopist; [N] = Nurse)
Endoscopes / Equip:
(G13) GIF160/ 2203920
(G14) 1T140 / 2111667
Medication: X 10% Xylocaine Spray LA



Indication / History
ca stomach with gastric outlet obstruction

Finding and Endoscopic Procedure
Severe esophagitis and gastritis with contact bleeding
Food residue and drug tablets filled up the whole stomach
Change to therapeutic scope. Gastric lavage and removal part of food residue done
Change back to OGD scope with outer diameter 8.6 mm.
Antral tumour seen extending into pylorus
Scope can pass through into 2nd part of duodenum
Entriflex tube was inserted under endoscopy guidance

Endoscopic Diagnosis: *(*Modifier: ?=Provisional; C=Complications)*
Oesophagitis (530.10)
Gastritis with haemorrhage (535.51)
Cancer of stomach - pyloric antrum (gastric outlet obstruction) (151.2)

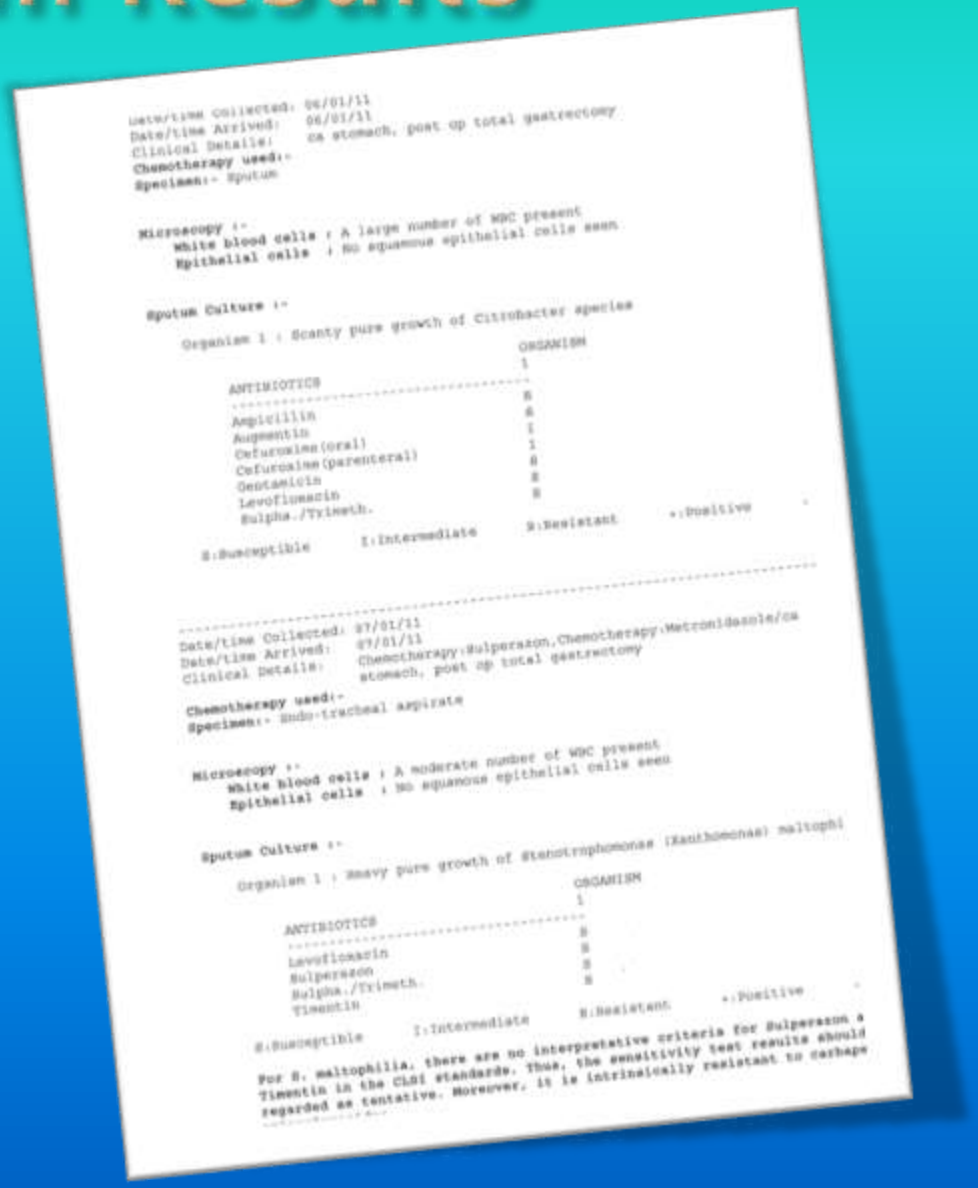
Procedure:
Therapeutic oesophagogastroduodenoscopy - insertion of feeding tube - site : duodenum (45.13, 96.35)

Hospital course

- Optimise nutrition by enteral feeding via entriflex
- Total gastrectomy performed on 31/12/2010
- Post operation managed in surgical HDU
  - TPN
  - Sputum retention requiring bronchoscopic clearance
  - Developed nosocomial pneumonia
    - Empirical Sulperazon

Sputum Results

- Antibiotics changed according to culture sensitivity of organisms
- Organisms
  - Citrobacter species
  - Stenotrophomonas maltophilia
- Antibiotics
  - Levofloxacin
  - Timentin



Date/time Collected: 06/01/11
Date/time Arrived: 06/01/11
Clinical Details: ca stomach, post op total gastrectomy
Chemotherapy used:-
Specimen:- Sputum

Microscopy :-
White blood cells : A large number of WBC present
Epithelial cells : No squamous epithelial cells seen

Sputum Culture :-

Organism 1 : Scanty pure growth of Citrobacter species

ANTIBIOTICS	ORGANISM 1
Ampicillin	R
Augmentin	R
Cefuroxime(oral)	I
Cefuroxime(parenteral)	I
Gentamicin	S
Levofloxacin	S
Sulpha./Trimeth.	S

S:Susceptible I:Intermediate R:Resistant +:Positive

Date/time Collected: 27/01/11
Date/time Arrived: 27/01/11
Clinical Details: Chemotherapy:Sulperazon,Chemotherapy:Metronidazole/ca stomach, post op total gastrectomy

Chemotherapy used:-
Specimen:- Endo-tracheal aspirate

Microscopy :-
White blood cells : A moderate number of WBC present
Epithelial cells : No squamous epithelial cells seen

Sputum Culture :-

Organism 1 : Heavy pure growth of Stenotrophomonas (Xanthomonas) maltophi

ANTIBIOTICS	ORGANISM 1
Levofloxacin	S
Sulperazon	S
Sulpha./Trimeth.	S
Timentin	R

S:Susceptible I:Intermediate R:Resistant +:Positive

For S. maltophilia, there are no interpretative criteria for Sulperazon & Timentin in the CLSI standards. Thus, the sensitivity test results should regarded as tentative. Moreover, it is is intrinsically resistant to carbape

ICU admission

ICU admission for nosocomial pneumonia

~2week post gastrectomy

- Respiratory failure requiring mechanical ventilation
- Septic shock with dopamine commenced in HDU
- Brief arrhythmia episode with sponatanous termination

Initial few hours

Resolving electrolytes disturbance with replacement

- K 2.4 → 3.6
- Mg 0.67 → 0.93
- iCa 1.01
- QTc 670→ 467 with normal sinus rhythum
- TnI not raised

General NURSING ASSESSMENT Created by RN [redacted] 13-Jan-2011 23:15

ICU DAY: 1

GC / CNS: GC critical. GCS 4T6, limbs power 3. pupils PEARL 3mm. temp 36C borderline, blankets offered, a bit cool limbs, patient denied coldness.

Respiratory: breathe via OETT PSV mode low support. keep monitor ABG. suction with minimal sputum, good coughing effort. chest: AE satisfactory and clear. keep prop up.

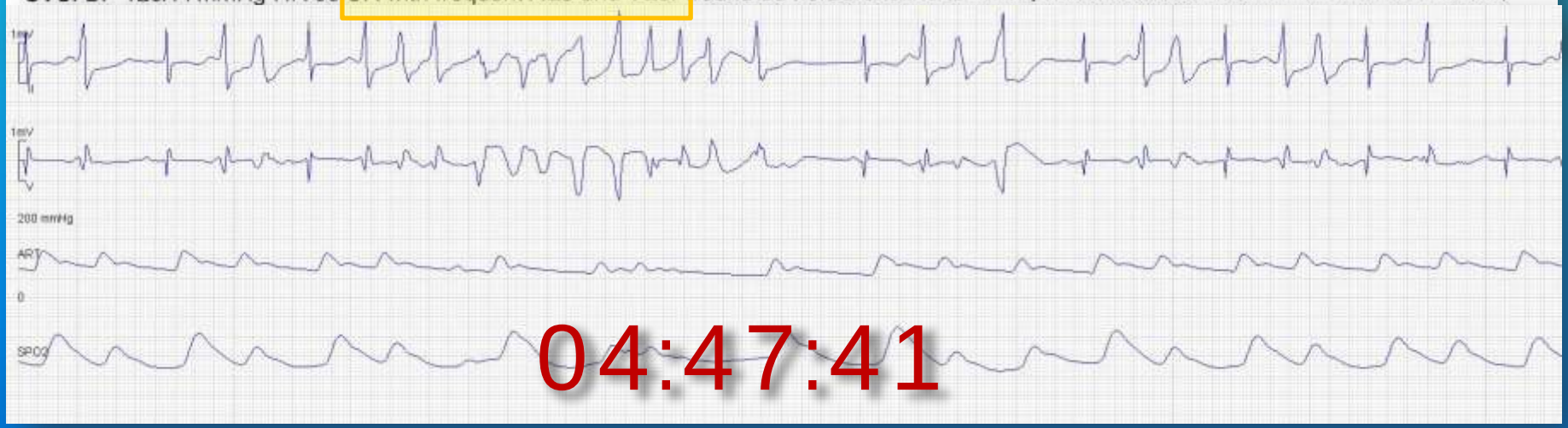
CVS: BP 125/75mmHg HR 81 SR with AEs and occassional VEs, with noradrenaline 6ml/h, limbs warm and pink. all lines mild blood-stained

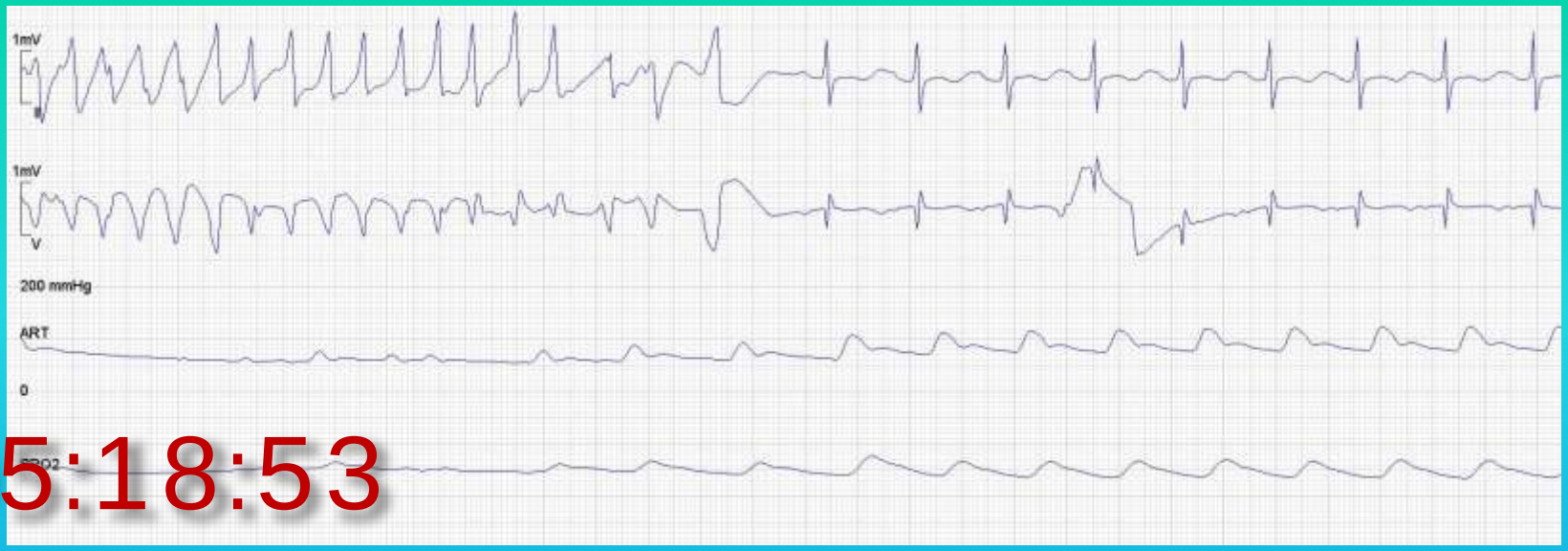
General EVALUATION OF CARE Created by RN [redacted] 14-Jan-2011 6:13

GC / CNS: GC critical. neurololgically same. temp normal.

Respiratory: continue PSV mode low support. ABG satisfactory, SpO2 100% RR 25/min. minimal sputum.

CVS: BP 128/77mmHg HR 93 SR with frequent AEs and VEs. weand\ed noradrenaline to 4ml/h, limbs circulation same with mild edematous,





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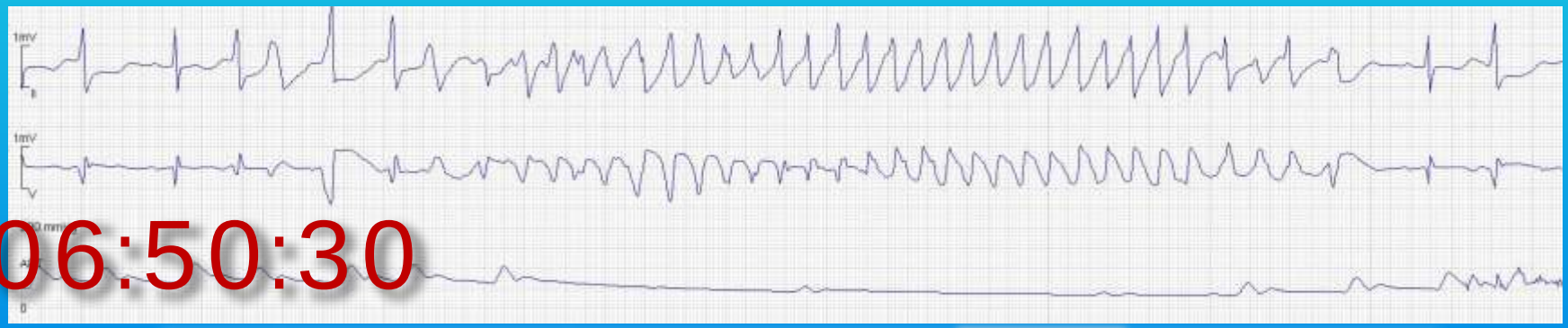


06:50:30

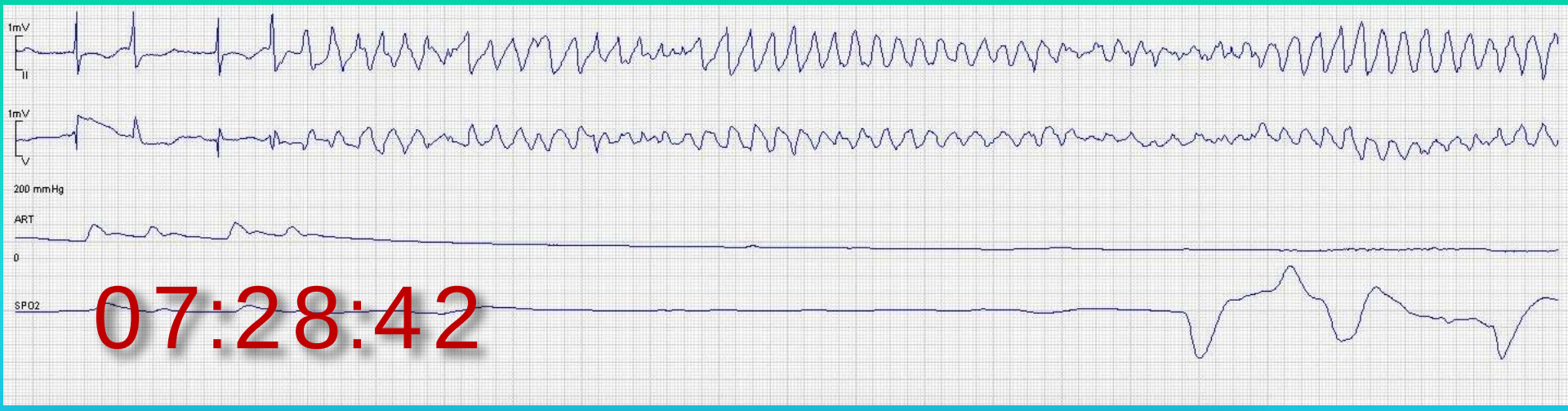
General NURSING ASSESSMENT Created by APN L 14-Jan-2011 8:13

ICU DAY:2(AM)

GC / CNS:
General condition is critical.Conscious and obeys commands.Expressed no discomfort.Upper limbs power~4 and lower limbs power~3.

Respiratory:
On PSV,FiO2 0.4,RR~23/min,Vt~300ml,SpO2~97%,ETCO2~4.7.Fair air entry on both sides.

CVS:
Frequent arrthymia with atrial/ventricular ectopics and wide complex tachycardia,Polymorphic VT. Amiodarone IV infusion in progress. Full Lead ECG done, QTc 479.BP borderline on Nor-Adrenlaine infusion.Warm extremities.Arterial/CVC site no oozing and no s/s infection.


1mV
II
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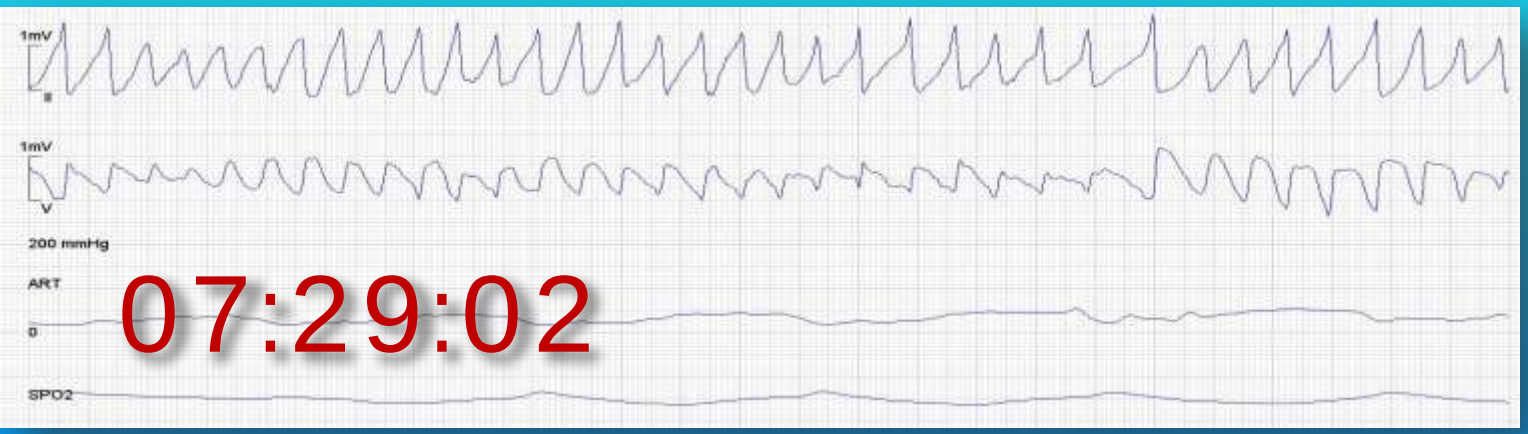
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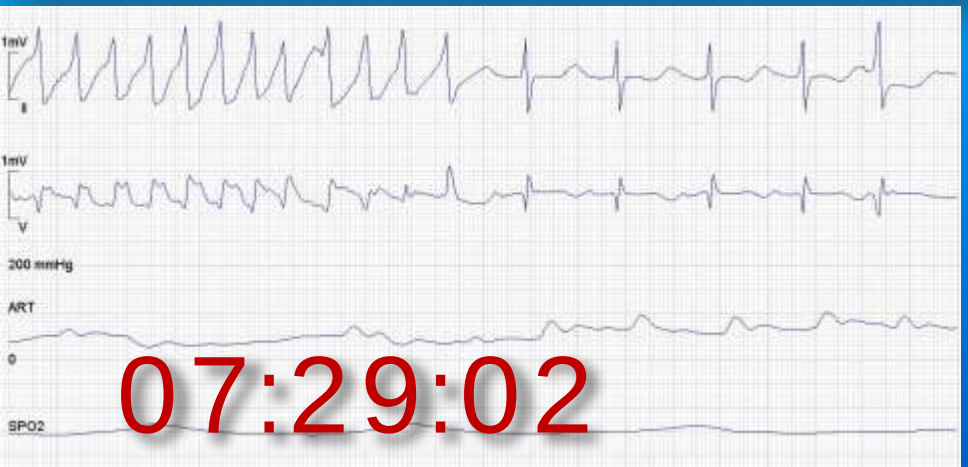
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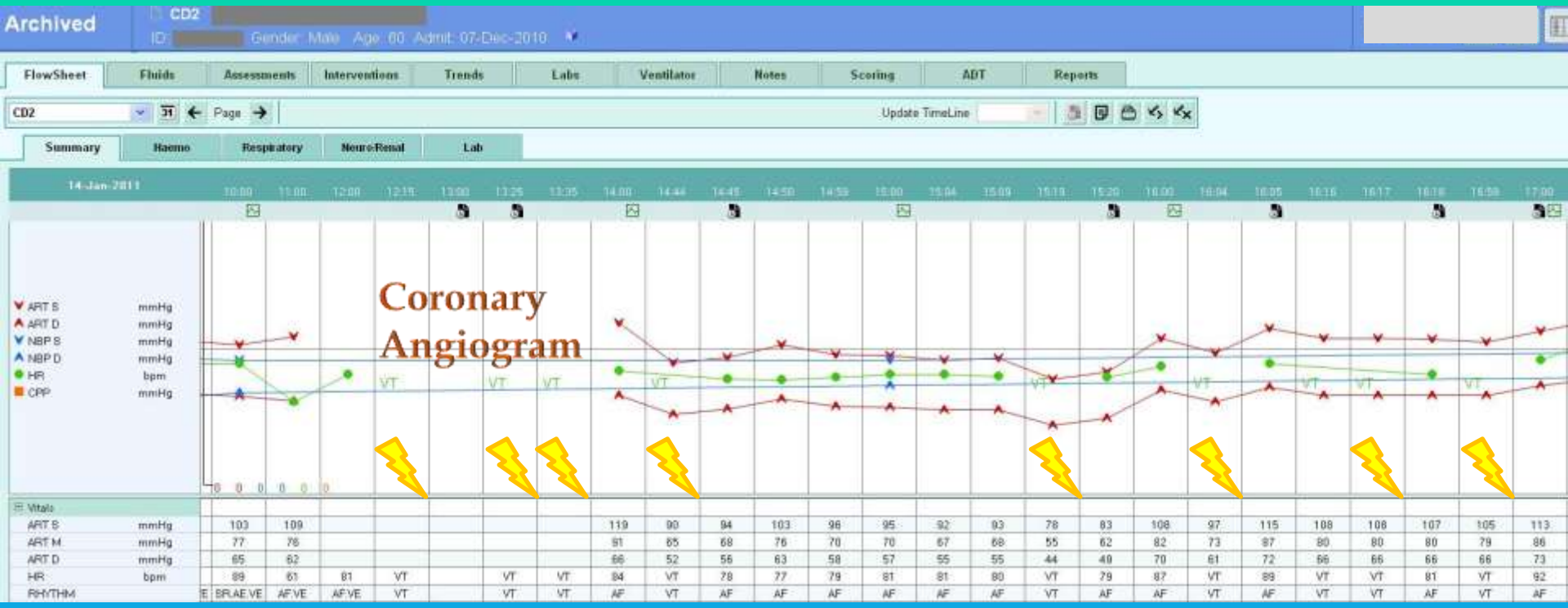
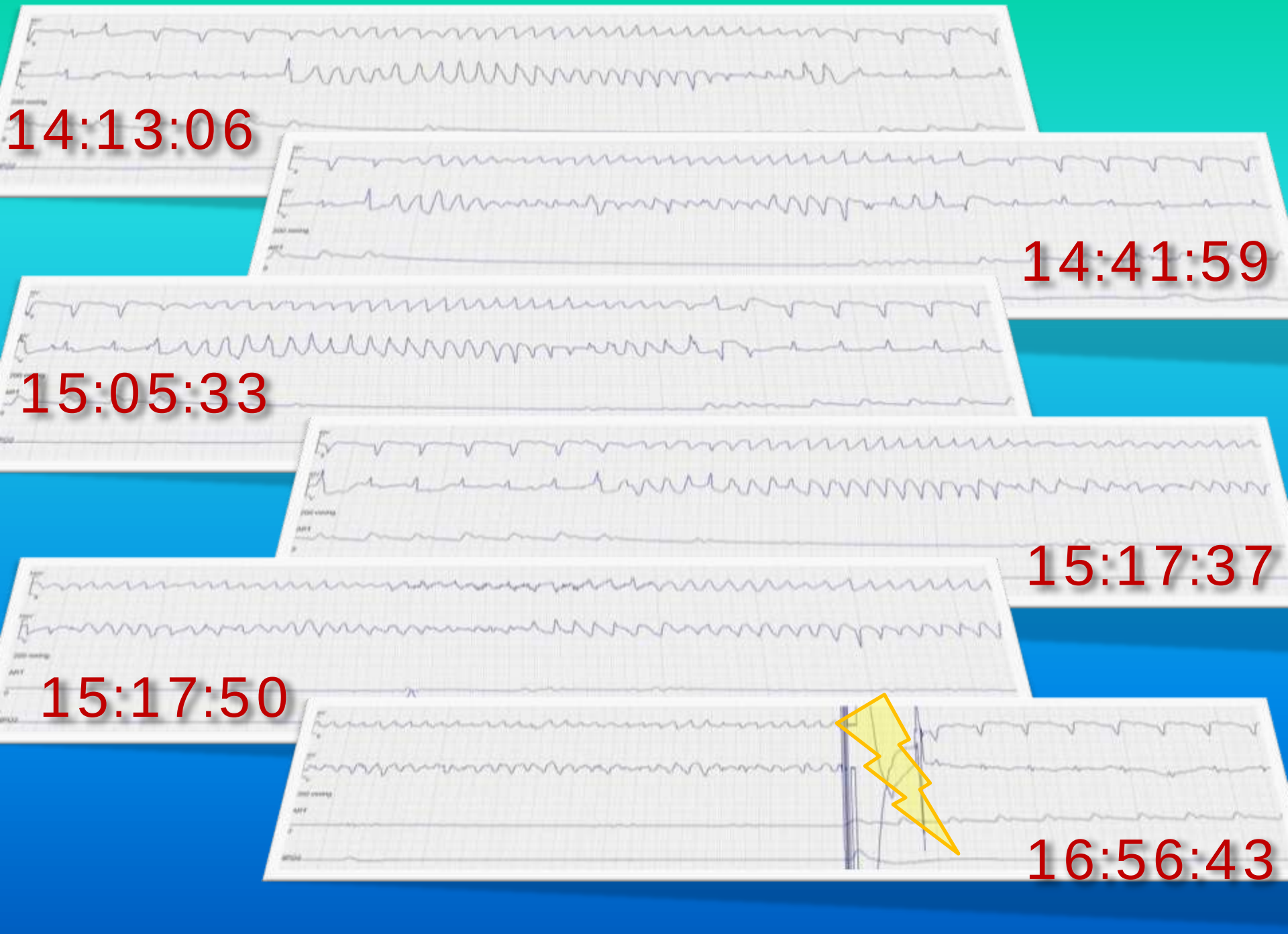


- Non-sustained polymorphic VT
  - Treated with Amiodarone and MgSO4 infusion
- Cardiologist assessment
  - Echo: global LV dysfunction
  - Coronary angiogram unremarkable
  - Over-drive pacing inserted with rate 80/min


14:13:06
14:41:59
15:05:33
15:17:37
15:17:50
16:56:43

Progress

- TdP eventually suppressed with overdrive pacing rate of 110/min
- Stayed in ICU for 2 weeks
- Hospital discharge ~4weeks after ICU admission
- Cardiac assessment
  - QTc 324 after stopping the offending drugs and correcting electrolytes

Levofloxacin

Electrolytes were approaching normal ,
Levofloxacin 500mg iv (4th daily dose)
was administered at 20:00.

PVC→ NSVT→ Sustained Polymorphic VT TdP

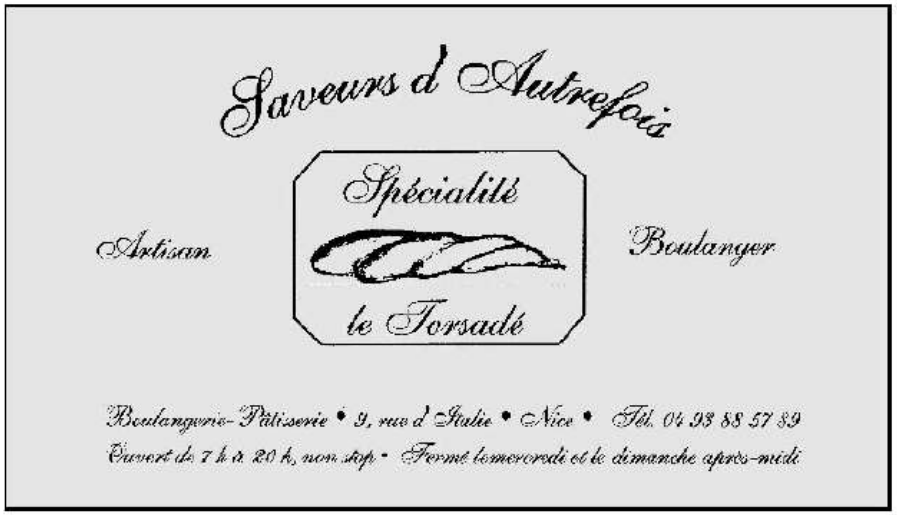
Drug Induced TdP
Torsades de pointes

Classification by Electrocardiography		
Nonsustained VT		Three or more beats in duration, terminating spontaneously in less than 30 s. VT is a cardiac arrhythmia of three or more consecutive complexes in duration emanating from the ventricles at a rate of greater than 100 bpm (cycle length less than 600 ms)
	Monomorphic	Nonsustained VT with a single QRS morphology.
	Polymorphic	Nonsustained VT with a changing QRS morphology at cycle length between 600 and 180 ms.
Sustained VT		VT greater than 30 s in duration and/or requiring termination due to hemodynamic compromise in less than 30 s.
	Monomorphic	Sustained VT with a stable single QRS morphology.
	Polymorphic	Sustained VT with a changing or multiform QRS morphology at cycle length between 600 and 180 ms.
Bundle-branch re-entrant tachycardia		VT due to re-entry involving the His-Purkinje system, usually with LBBB morphology; this usually occurs in the setting of cardiomyopathy.
Bidirectional VT		VT with a beat-to-beat alternans in the QRS frontal plane axis, often associated with digitalis toxicity.
Torsades de pointes		Characterized by VT associated with a long QT or QTc, and electrocardiographically characterized by twisting of the peaks of the QRS complexes around the isoelectric line during the arrhythmia: ■ "Typical," initiated following "short-long-short" coupling intervals. ■ Short coupled variant initiated by normal-short coupling.
Ventricular flutter		A regular (cycle length variability 30 ms or less) ventricular arrhythmia approximately 300 bpm (cycle length—200 ms) with a monomorphic appearance; no isoelectric interval between successive QRS complexes.
Ventricular fibrillation		Rapid, usually more than 300 bpm/200 ms (cycle length 180 ms or less), grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.

Torsade de Pointes

Figure 1. Meaning of *torsade*

Daily utterance of the term *torsade* in French refers to different bakery items, including bread loaves with a characteristic twisting configuration. This card displays one advertisement for such products. Reprinted with permission [2].



Risk Factors for Drug-Induced Torsade de Pointes.

Table 2. Risk Factors for Drug-Induced Torsade de Pointes.*

Female sex[10]
Hypokalemia[11,12]
Bradycardia[11,12]
Recent conversion from atrial fibrillation, especially with a QT-prolonging drug[13,14]
Congestive heart failure[15]
Digitalis therapy[16]
High drug concentrations (with the exception of quinidine)
Rapid rate of intravenous infusion with a QT-prolonging drug[17]
Base-line QT prolongation[16]
Subclinical long-QT syndrome[18,19]
Ion-channel polymorphisms[20-22]
Severe hypomagnesemia

* Studies providing evidence of the effects are cited in the table.

Roden DM. N Engl J Med 2004;350:1013-1022.

Some reported causes and potentiators of the long QT syndrome

Congenital	**Antihistamines**
Jervell and Lange-Nielsen syndrome (including "channelopathies")	Terfenadine
Romano-Ward syndrome	Astemizole
Idiopathic	**Psychotropic drugs**
Acquired	Thioridazine
Metabolic disorders	Phenothiazines
Hypokalemia	Tricyclic or tetracyclic antidepressants
Hypomagnesemia	Haloperidol and other butyrophenones
Hypocalcemia	**Other drugs**
Starvation	Selective serotonin reuptake inhibitors
Anorexia nervosa	Risperidone
Liquid protein diets	Methadone
Hypothyroidism	Vasodilators - Prenylamine, bepridil, mibefradil
Bradyarrhythmias	Diuretics - Via electrolyte changes (esp. hypokalemia or hypomagnesemia)
Sinus node dysfunction	Serotonin antagonist - Ketanserin
AV block - second or third degree	Motility drugs - Cisapride, domperidone
Antiarrhythmic drugs	Droperidol - may be safe at the low doses used by anesthesiologists (0.625 to 1.25 mg)
Quinidine	Ranolazine
Procainamide or N-acetylprocainamide	HIV protease inhibitors
Disopyramide	Miscellaneous - Organophosphate insecticides, probucol, cocaine, terodiline, papaverine, certain Chinese herbs, chloral hydrate, arsenic trioxide, cesium chloride, levomethadyl
Amiodarone and dronedarone	**Other factors**
Sotalol	Myocardial ischemia or infarction, esp. with prominent T wave inversions
Dofetilide, ibutilide, azimilide, sematilide	Intracranial disease
Antimicrobial drugs	HIV infection
Erythromycin, clarithromycin, telithromycin, azithromycin (minor)	Hypothermia
Pentamidine	Connective tissue diseases with anti-Ro/SSA antibodies
Some fluoroquinolones (eg, sparfloxacin, gatifloxacin, levofloxacin, moxifloxacin)	
Other - Spiramycin, chloroquine, halofantrine mefloquine	

Indicate removed from market

UpToDate

Drugs That May Cause Torsade de Pointes.

Table 1. Drugs That May Cause Torsade de Pointes.*

Drugs commonly involved

Disopyramide
Dofetilide
Ibutilide
Procainamide
Quinidine
Sotalol
Bepridil

Other drugs†

Amiodarone
Arsenic trioxide
Cisapride
Calcium-channel blockers: lidoflazine (not marketed in the United States)
Antiinfective agents: clarithromycin, erythromycin, halofantrine, pentamidine, sparfloxacin
Antiemetic agents: domperidone, droperidol
Antipsychotic agents: chlorpromazine, haloperidol, mesoridazine, thioridazine, pimozide
Methadone

* Further information on the strength of the evidence linking various drugs to torsade de pointes may be found at http://www.torsades.org.

† The level of risk associated with these drugs depends on the dose and the population being treated; in general, the risk is probably less than 1 percent.

Roden DM. N Engl J Med 2004;350:1013-1022.

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ECG signs of TdP

- Premonitoring
  - QTc>500ms
  - T wave:

    Flattened/Biphasic or notched T wave/
    T waves with superimposed U waves
    (T-U distortion)/T wave Alternans
  - Short-long-short sequence of RR interval

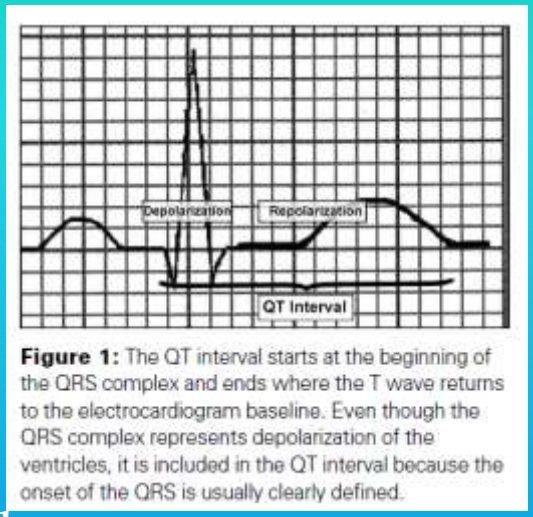


Figure 1: The QT interval starts at the beginning of the QRS complex and ends where the T wave returns to the electrocardiogram baseline. Even though the QRS complex represents depolarization of the ventricles, it is included in the QT interval because the onset of the QRS is usually clearly defined.

- TdP (Polymorphic VT)
  - Twisting of QRS around isoelectric line
  - Cycling of QRS axis through 180 degree every 5 to 20 beats
  - Ventricular rate 160 to 250/min
  - Frequent terminate spontaneously
  - May degenerates into VF

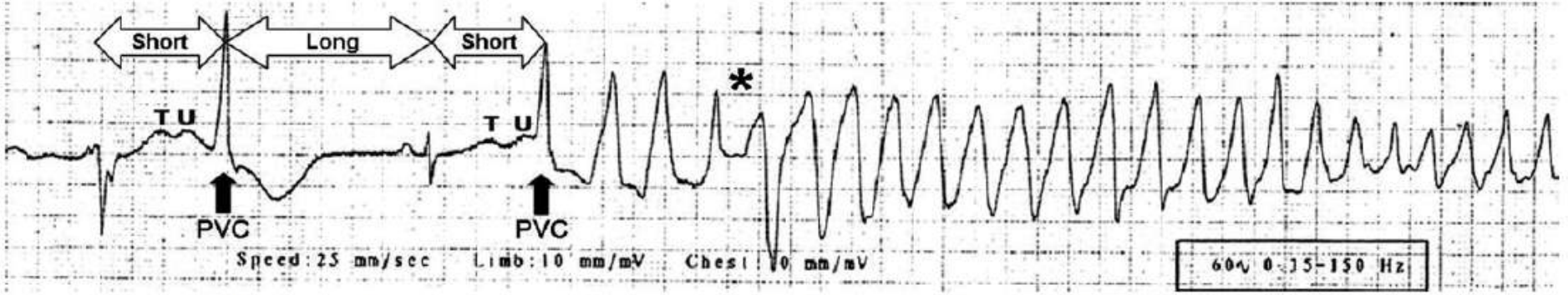
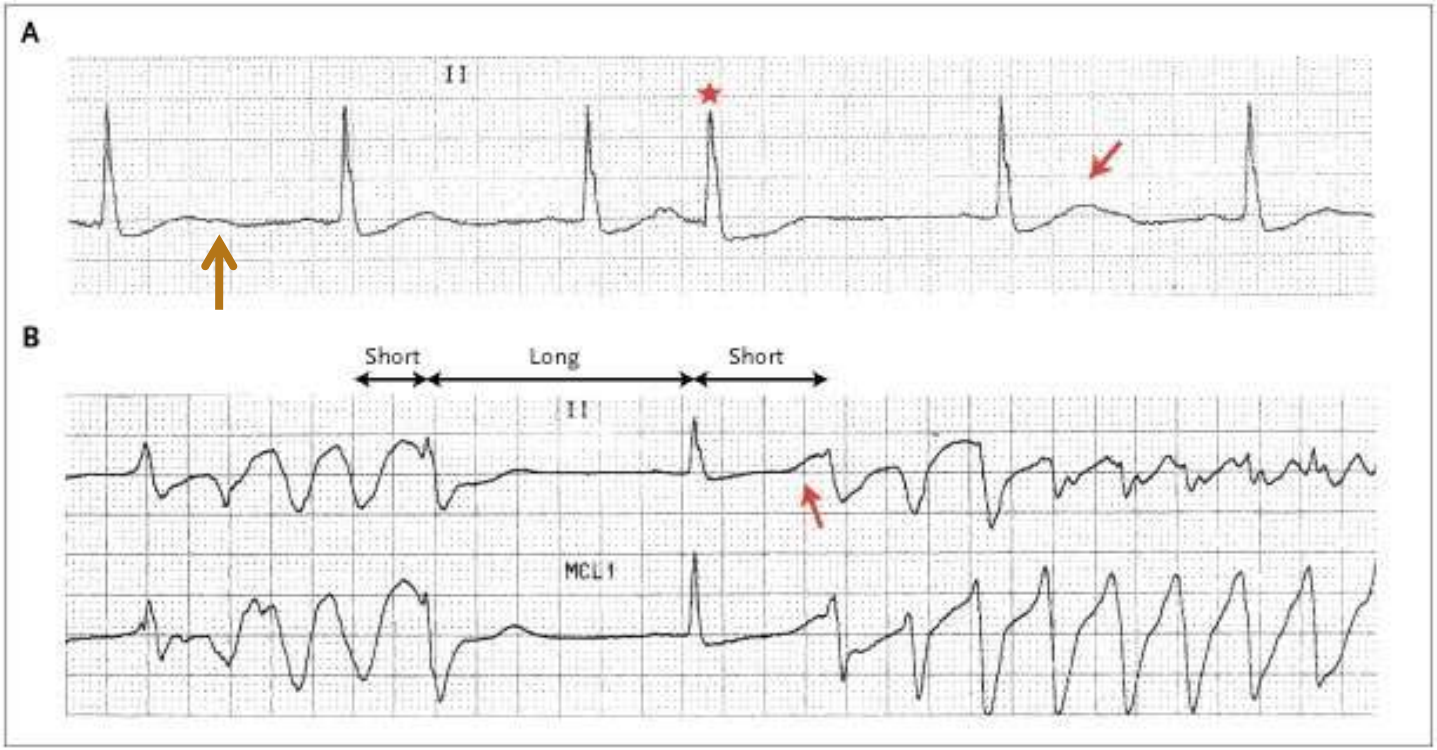
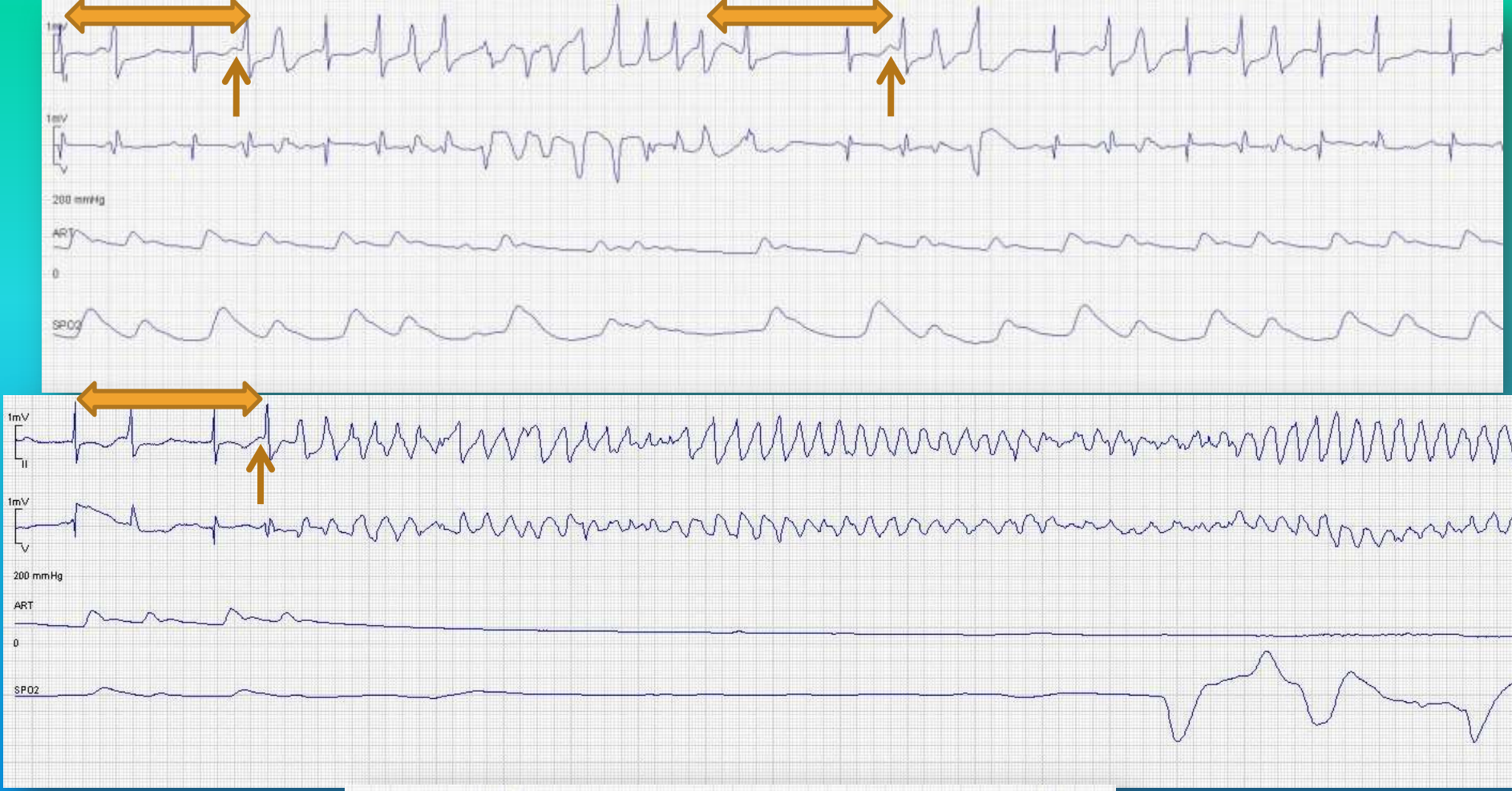


Figure 1. Onset of TdP during the recording of a standard 12-lead ECG in a young male with a history of drug addiction treated with chronic methadone therapy who presented to a hospital emergency department after ingesting an overdose of prescription and over-the-counter drugs from his parent's drug cabinet. Classic ECG features evident in this rhythm strip include a prolonged QT interval with distorted T-U complex, initiation of the arrhythmia after a short-long-short cycle sequence by a PVC that falls near the peak of the distorted T-U complex, "warm-up" phenomenon with initial R-R cycles longer than subsequent cycles, and abrupt switching of QRS morphology from predominately positive to predominately negative complexes (asterisk).

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1mV
V
200 mmHg
ART
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SPO2

1mV
II
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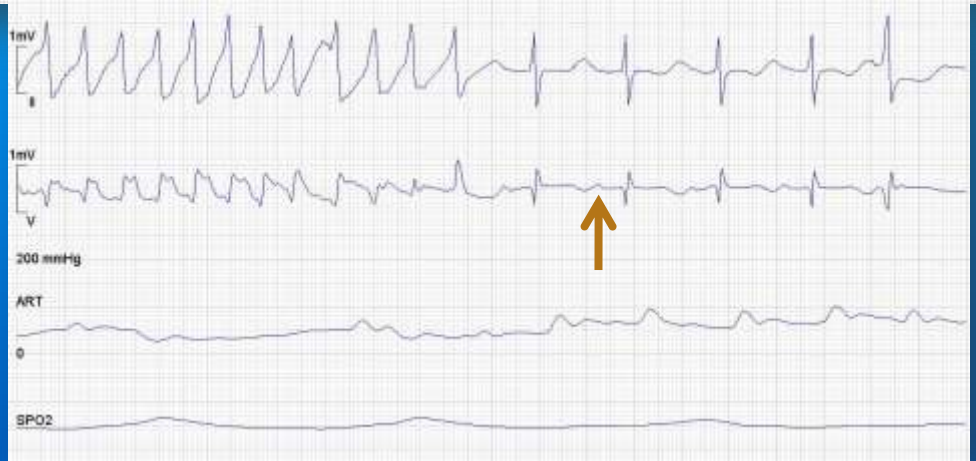
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ECG & Membrane Potential of Ventricular Cell

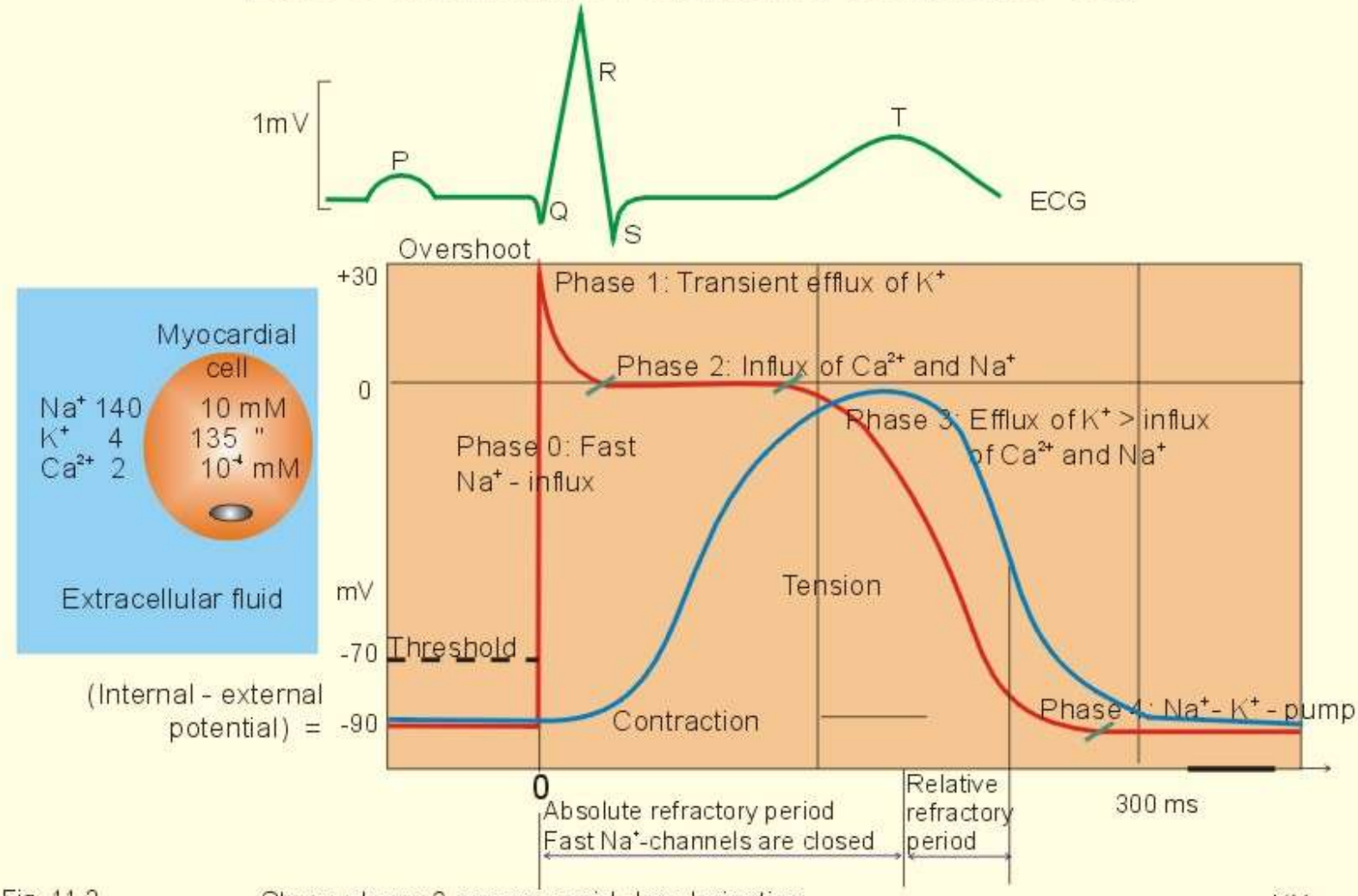
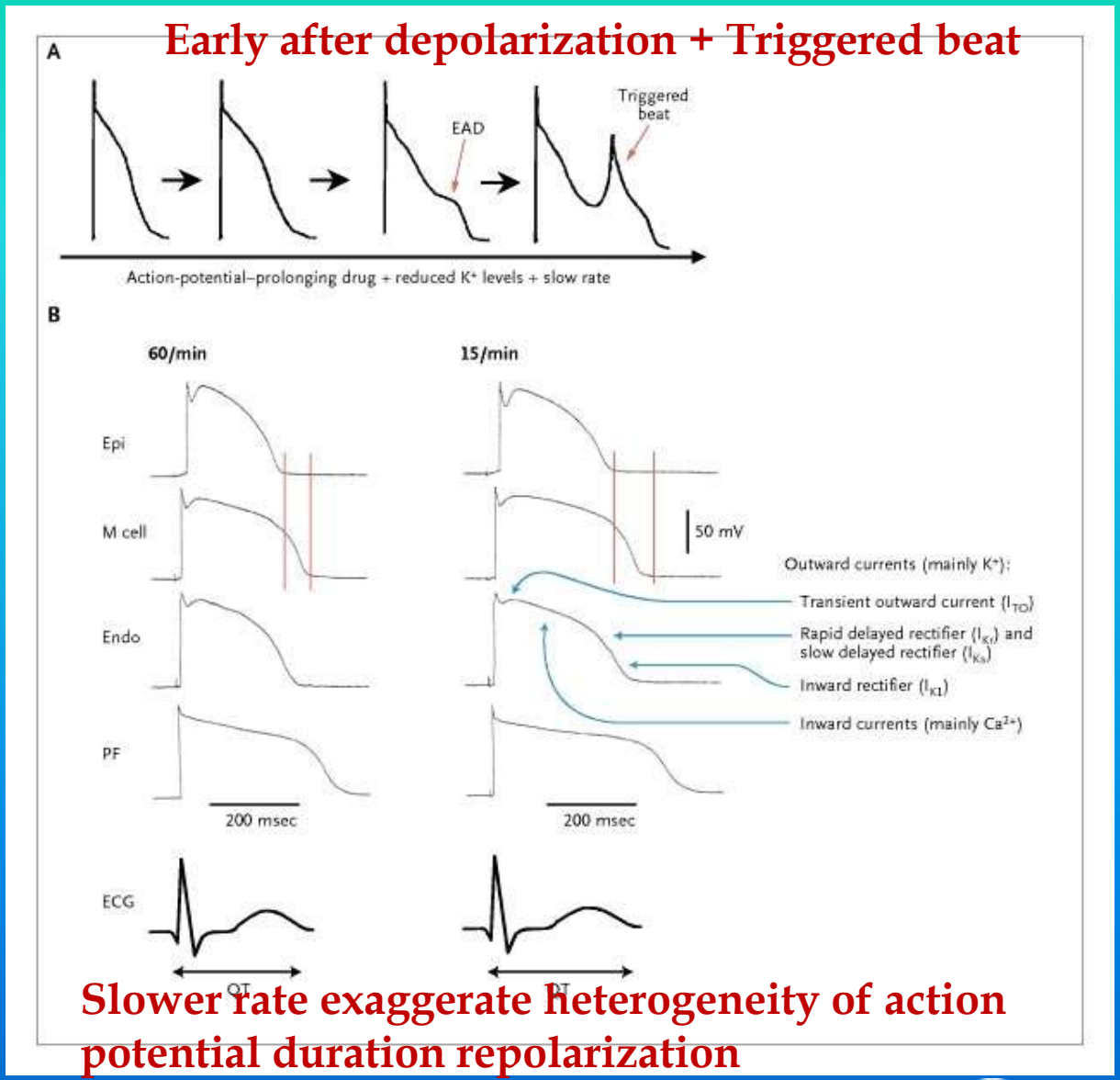


Fig. 11-2

Steep phase 0 means rapid depolarisation

KMc

Postulated Basic Mechanisms in Arrhythmias Related to Long-QT Intervals.



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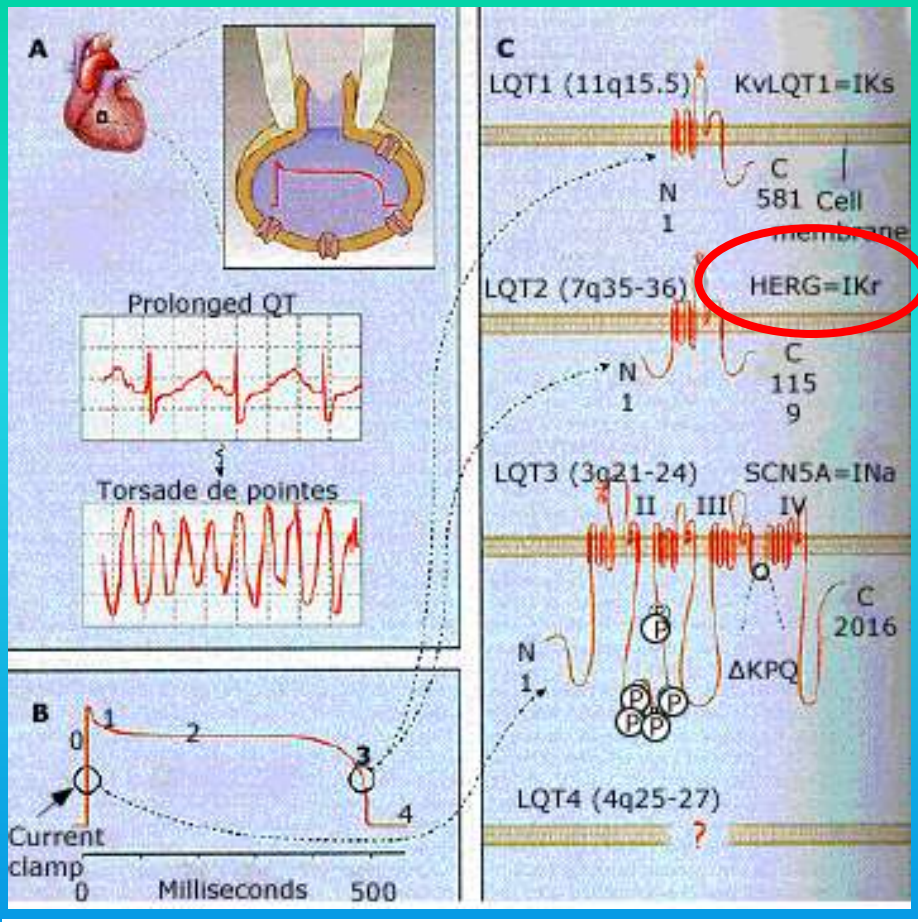
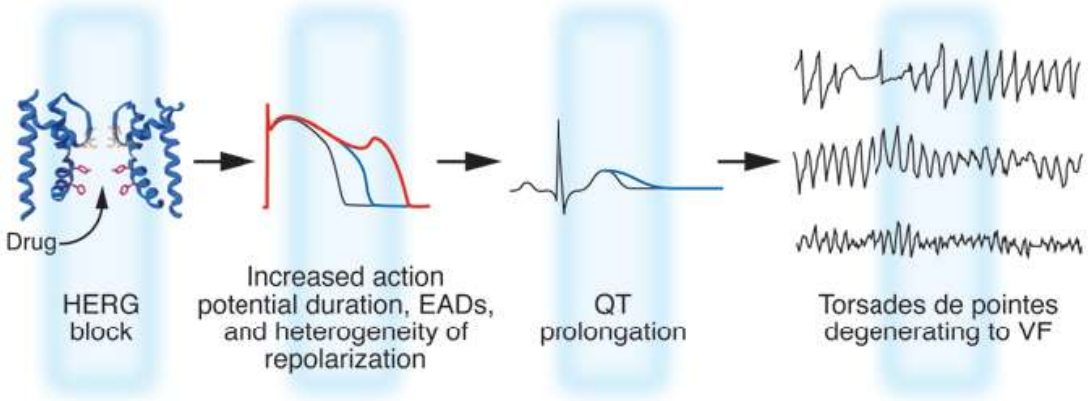


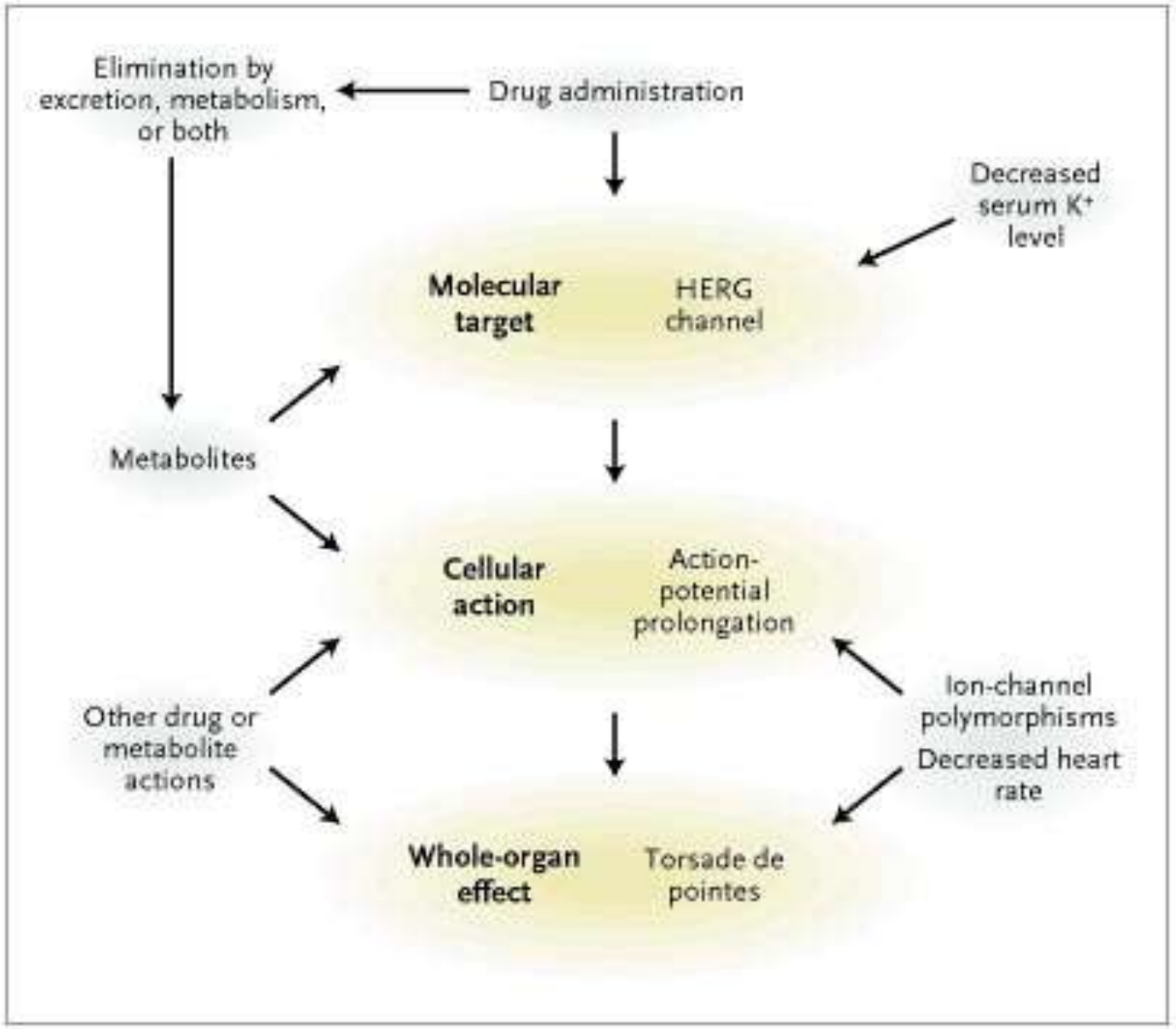
HERG – human ether-a-go-go (Now termed KCNH2)

I_{Kr} - rapid component of delayed rectifier K current

Figure 5
Mechanisms of sudden death with HERG blockade. Drug blockade of the HERG channel (left) produces prolongation (blue) and an EAD (red) in the cardiac action potential. These changes, which are heterogeneous across the ventricular wall, generate QT interval prolongation and, through mechanisms described further in the text, torsade de pointes (right; upper panel). In this example, the arrhythmia degenerates to ventricular fibrillation (VF).



Effects of a Drug on Whole-Organ Function.



Roden DM. N Engl J Med 2004;350:1013-1022.

ELSEVIER

International Journal of Antimicrobial Agents 29 (2007) 374–379

International Journal of Antimicrobial Agents

www.ischemo.org

Review

Arrhythmias associated with fluoroquinolone therapy

Matthew E. Falagas[a,b,*], Petros I. Rafailidis[a], Evangelos S. Rosmarakis[a,c]

Table 2
Pharmacological

Fluoroquinolones
Sparfloxacin[b]
Grepafloxacin[b]
Gatifloxacin[c]
Gemifloxacin[c]
Levofloxacin[c]
Moxifloxacin[c]
Ofloxacin [c]
Ciprofloxacin[e]

[a] Data taken from [26].
[b] Drugs that are generally accepted to have a risk of causing Tdp.
[c] Drugs that in some reports may be associated with Tdp but at this time lack substantial evidence for causing Tdp.
[d] Drugs to be avoided for use in patients with diagnosed or suspected congenital long QT syndrome. (Drugs on lists b, c and e should also be avoided in patients with QT syndrome.)
[e] Drugs that, in some reports, have been weakly associated with Tdp and/or QT prolongation but that are unlikely to be a risk for Tdp when used in usual recommended dosages and in patients without other risk factors.

Table 1
Prospective clinical trials examining the effect of fluoroquinolones on QT interval prolongation and on triggering episodes of torsades de pointes (Tdp)

Reference	Study design	No. of patients	Inclusion criteria	Medication/treatment course	Effect on QTc	Mean increase in QTc	Episode of Tdp
Tsikouris et al. [12]	Randomised, open-label crossover study	13	(1) Healthy volunteers; (2) mean age 34 years; (3) patients without cardiovascular disease or renal dysfunction who were not on medication that prolongs the QT interval	Ciprofloxacin, 500 mg twice daily for 7 days	No effect	N.S.	None
				Levofloxacin, 500 mg once daily for 7 days	No effect	N.S.	None
				Moxifloxacin, 400 mg once daily for 7 days	Prolongation of QTc at 7-day measurements	6 ms (from baseline, $P=0.022$)	None
Makaryus et al. [16]	Prospective clinical trial	38	(1) Patients treated for respiratory tract infection or urogenital infection; (2) mean age 65 years; (3) patients without arrhythmia or not receiving medication that prolongs the QT interval	Ciprofloxacin, 250 mg twice daily	No effect	N.S.	None
				Levofloxacin, 500 mg once daily or 250 mg once daily for patients with UTI	No effect	N.S.	None
Noel et al. [15]	Double-blind, randomised, placebo-controlled, four-period, four-sequence, crossover trial	47	(1) Healthy volunteers; (2) mean age 47.1 years; (3) volunteers who had normal 12-lead ECG, heart rate 50–100 beats/min, with no past medical history of cardiac disease, who had creatinine clearance >50 mL/min and without receiving concomitant medications	Levofloxacin, single dose of 500 mg	No effect	N.S.	None
				Levofloxacin, single dose of 1000 mg	Prolongation of QTc 24 h after single doses of 100 mg	From 1.5 ms to 3.9 ms ($P \leq 0.05$)	None
				Levofloxacin, single dose of 1500 mg	Prolongation of QTc 24 h after single doses of 1500 mg	From 6.4 ms to 7.7 ms ($P \leq 0.001$)	None
Noel et al. [14]	Double-blind randomised, placebo-controlled, four-period, four-sequence, crossover trial	47	(1) Healthy volunteers; (2) mean age 47.6 years; (3) volunteers who had normal 12-lead ECG, heart rate 50–100 beats/min, with no past medical history of cardiac disease, who had creatinine clearance >50 mL/min and without receiving concomitant medications	Placebo	No effect	N.R.	None
				Moxifloxacin, 800 mg once daily	Prolongation of QTc 24 h after administration	From 16.3 ms to 17.8 ms ($P<0.001$)	None
				Levofloxacin, 1000 mg once daily	Prolongation of QTc 24 h after administration	From 3.5 ms to 4.9 ms ($P<0.05$)	None
				Ciprofloxacin, 1500 mg once daily	Prolongation of QTc 24 h after administration	From 2.3 ms to 4.9 ms ($P<0.05$)	None
Demolis et al. [13]	Double-blind, randomised, placebo-controlled, crossover trial	18	(1) Healthy volunteers; (2) mean age 23.8 years	Placebo	No effect	N.R.	None
				Moxifloxacin, 400 mg once daily	Prolongation of QTc 2 h after oral administration	$4.0 \pm 5.1\%$ (after 400 mg per os administration; $P<0.05$)	None
				Moxifloxacin, 800 mg once daily	Prolongation of QTc 2 h after oral administration	$4.5 \pm 3.8\%$ (after 800 mg per os administration; $P<0.05$)	None

QTc, corrected QT interval; N.S., not significant; UTI, urinary tract infection; ECG, electrocardiogram; N.R., not reported.

Case Reports

Levofloxacin-Induced Torsades de Pointes

Parag D. Patel, MD
Hamid Afshar, MD
Yochai Birnbaum, MD, FACC

A 91-year-old woman, presenting with flu-like symptoms, developed a brief episode of polymorphic ventricular tachycardia in the emergency department. The arrhythmia resolved spontaneously, and a subsequent electrocardiogram revealed Q waves and ST-segment elevation in the anterior precordial leads, along with a prolonged QT interval. The presumed diagnosis was ST-segment–elevation myocardial infarction with ischemia-induced ventricular tachycardia. Emergent coronary artery angiography revealed only minimal luminal irregularities. It was discovered that the patient had been taking levofloxacin and, apparently as a result, developed drug-induced torsades de pointes. The case of this patient is an example of the difficulties that are occasionally encountered in differentiating ST-segment–elevation myocardial infarction from nonischemic ST elevation. ***(Tex Heart Inst J 2010;37(2):216-7)***

QTC INTERVAL PROLONGATION AND POLYMORPHIC VENTRICULAR TACHYCARDIA IN ASSOCIATION WITH LEVOFLOXACIN

Case Report

Ciprofloxacin-induced torsade de pointes

Abstract

A 65-year-old man with recently diagnosed urinary tract infection treated with ciprofloxacin (Cipro) presented to our institution with recurrent seizure-like activity. His rhythm revealed torsade de pointes, which required defibrillation. Subsequent electrocardiogram revealed severely prolonged QT interval, which near-completely resolved 7 days later off Cipro. This case highlights a rare but potentially fatal side effect of quinolone antibiotics, especially in combination with other QT-prolonging medications. Review of the literature with regard to prevalence, mechanism, and assessment and treatment of this potentially fatal incidence is provided.

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ACC/AHA/ESC PRACTICE GUIDELINES—EXECUTIVE SUMMARY

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death—Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death)
Developed in Collaboration With the European Heart Rhythm Association and the Heart Rhythm Society

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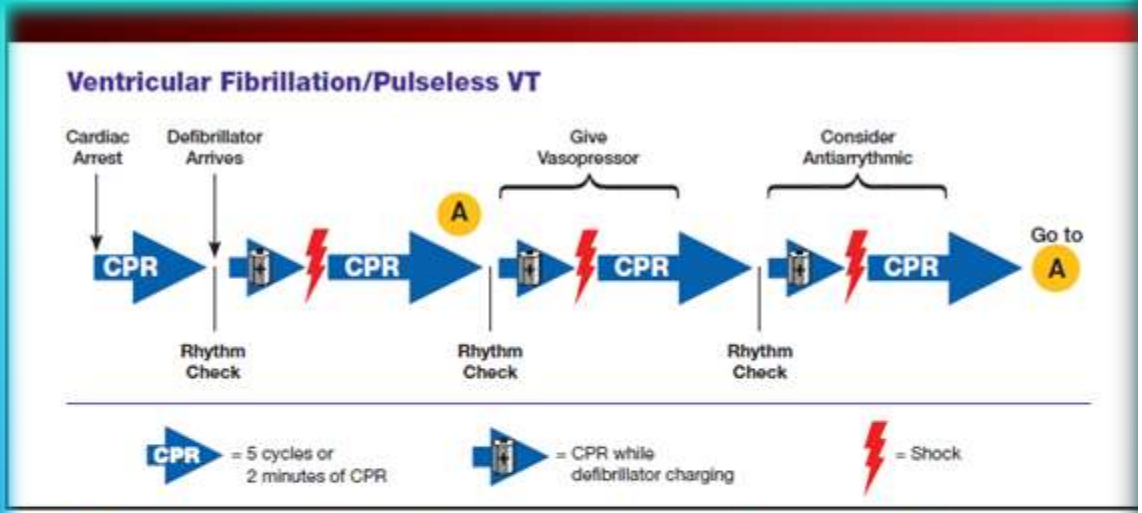

Figure 2: Ventricular Fibrillation and Pulseless VT: Treatment Sequences for ACLS and PALS. This illustrates suggested timing of CPR, rhythm checks, attempted defibrillation (shock delivery), and drug delivery for persistent VF/pulseless VT. Drug doses should be prepared *prior* to rhythm check. Drugs should be administered during CPR, as soon after a rhythm check as possible. Ideally CPR (particularly chest compressions) is interrupted only for rhythm check and shock delivery. If possible, rescuers should perform chest compressions while the defibrillator is charging. Rescuers should resume chest compressions immediately after a shock is delivered. In in-hospital settings with continuous (eg, electrocardiographic, hemodynamic) monitoring in place, this sequence may be modified by the physician. If PEA or asystole develops after a shock (and CPR), rescuers should follow the Asystole/PEA branch of the ACLS or PALS Pulseless Arrest Algorithms.

7.1.1.1. Pulseless Ventricular Tachycardia/Ventricular Fibrillation

In the event of pulseless VT or VF in ACS, the standard ACLS protocol is initiated including unsynchronized electric shock following basic assessment of airway and initiation of CPR. Energy delivery consists of 1 or more monophasic shocks at 360 J or biphasic shocks at a dose range demonstrated by manufacturer to be effective. If not available, a dose of 200 J is recommended for the first shock and an equal or higher dose for subsequent shocks. The optimal dose for biphasic shocks has not been determined, and no waveform or escalating energy levels recommendations can be made for biphasic defibrillators at this time. If return to normal rhythm is not accomplished by defibrillation, the ACLS protocol for pulseless VT or VF is followed. This includes epinephrine (1 mg intravenously every 3 to 5 min) or vasopressin (40 U intravenously once only; 1 dose of vasopressin intravenously/intraosseously may replace either the first or second dose of epinephrine), and amiodarone (300-mg or 5-mg/kg intravenous push, with a possible repeat 150-mg intravenous push once only), or as a second tier, lidocaine (1.0 to 1.5 mg/kg with repeat dose of 0.5 to 0.75 mg intravenously/intraosseously up to a total dose of 3 mg/kg). Additional second-tier therapy includes intravenous magnesium (1 to 2 g) or procainamide (30 mg/min up to 17 mg/kg). The latter is considered acceptable but is no longer recommended (334).

Following resuscitated VF, prophylactic drug infusion, typically with amiodarone plus a beta blocker, may be continued. The antiarrhythmic should be withdrawn as appropriate to assess the presence of ongoing arrhythmias.

G. Torsades de Pointes

Recommendations

Class I

1. Withdrawal of any offending drugs and correction of electrolyte abnormalities are recommended in patients presenting with torsades de pointes. *(Level of Evidence: A)*
2. Acute and long-term pacing is recommended for patients presenting with torsades de pointes due to heart block and symptomatic bradycardia. *(Level of Evidence: A)*

Class IIa

1. Management with intravenous magnesium sulfate is reasonable for patients who present with LQTS and few episodes of torsades de pointes. Magnesium is not likely to be effective in patients with a normal QT interval. *(Level of Evidence: B)*
2. Acute and long-term pacing is reasonable for patients who present with recurrent pause-dependent torsades de pointes. *(Level of Evidence: B)*
3. Beta blockade combined with pacing is reasonable acute therapy for patients who present with torsades de pointes and sinus bradycardia. *(Level of Evidence: C)*
4. Isoproterenol is reasonable as temporary treatment in acute patients who present with recurrent pause-dependent torsades de pointes who do not have congenital LQTS. *(Level of Evidence: B)*

Class IIb

1. Potassium repletion to 4.5 to 5 mM/L may be considered for patients who present with torsades de pointes. *(Level of Evidence: B)*
2. Intravenous lidocaine or oral mexiletine may be considered in patients who present LQT3 and torsades de pointes. *(Level of Evidence: C)*

Summary of the treatment of torsade de pointes and long QT syndrome

	Pharmacological	Nonpharmacological
Congenital	Magnesium sulfate	Permanent cardiac pacemaker
	β-Blockers	Cardiothoracic sympathectomy
	Mexiletine[a]	Implantable cardioverter defibrillator
Acquired	Magnesium sulfate	Removal of the cause
	Isoproterenol	Temporary cardiac pacing
	Atropine	
	Lidocaine	
	Phenytoin	
	Sodium bicarbonate[b]	

Classification of Recommendations

- **Class I:** Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.
- **Class II:** Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure or treatment.
  - **Class IIa:** Weight of evidence/opinion is in favor of usefulness/efficacy.
  - **Class IIb:** Usefulness/efficacy is less well established by evidence/opinion.
- **Class III:** Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

Level of Evidence

- **Level of Evidence A:** Data derived from multiple randomized clinical trials or meta-analyses.
- **Level of Evidence B:** Data derived from a single randomized trial or nonrandomized studies.
- **Level of Evidence C:** Only consensus opinion of experts, case studies, or standard-of-care.

Amiodarone

- Class III
- Rarely associated with TdP
- Uniformly delays repolarization in all layers of the myocardium
- Only prolong QT but no transmural heterogeneity of repolarization
- Risk increase with concurrent use of Class IA agent or hypokalemia

F. Polymorphic Ventricular Tachycardia

Recommendations

Class I

1. Direct current cardioversion with appropriate sedation as necessary is recommended for patients with sustained polymorphic VT with hemodynamic compromise and is reasonable at any point in the treatment cascade. *(Level of Evidence: B)*
2. Intravenous beta blockers are useful for patients with recurrent polymorphic VT especially if ischemia is suspected or cannot be excluded. *(Level of Evidence: B)*
3. Intravenous loading with amiodarone is useful for patients with recurrent polymorphic VT in the absence of abnormal repolarization related to congenital or acquired LQTS. *(Level of Evidence: C)*
4. Urgent angiography with a view to revascularization should be considered for patients with polymorphic VT when myocardial ischemia cannot be excluded. *(Level of Evidence: C)*

AHA Scientific Statement

Practice Standards for Electrocardiographic Monitoring in Hospital Settings

An American Heart Association Scientific Statement From the Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young

- QT Interval and ECG monitoring
  - Class I
  - Patients Administered the drug known to cause TdP
  - Patients who overdose from a potentially proarrhythmic agent
  - New-onset Bradyarrhythmias
  - Patients with severe hypokalemia or hypomagnesemia
- Baseline strip then QTc measurement
  - every 8-12 hour for 48 to 72 hour

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SCIENTIFIC STATEMENT

Prevention of Torsade de Pointes in Hospital Settings

A Scientific Statement From the American Heart Association and the American College of Cardiology Foundation

THANK YOU

Table 3. Summary of Key Points

1. Drugs associated with TdP vary greatly in their risk for arrhythmia; an updated list can be found at www.qtdrugs.org.
2. Risk factors for drug-induced TdP include older age, female sex, heart disease, electrolyte disorders (especially hypokalemia and hypomagnesemia), renal or hepatic dysfunction, bradycardia or rhythms with long pauses, treatment with more than 1 QT-prolonging drug, and genetic predisposition.
3. The risk-benefit ratio should be assessed for each individual to determine whether the potential therapeutic benefit of a drug outweighs the risk for TdP.
4. After initiation of a drug associated with TdP ECG signs indicative of risk for arrhythmia include an increase in QT_c from predrug baseline of 60 ms, marked QT_c interval prolongation >500 ms, T-U wave distortion that becomes more exaggerated in the beat after a pause, visible (macroscopic) T-wave alternans, new-onset ventricular ectopy, couplets and nonsustained polymorphic ventricular tachycardia initiated in the beat after a pause.
5. In monitoring QT intervals in an individual before and after drug administration, a consistent method should be used (i.e., same recording device, ECG lead, measurement tool [automated or manual], and heart rate–correction formula).
6. Recommended actions when ECG signs of impending TdP develop are to discontinue the offending drug, replace potassium, administer magnesium, consider temporary pacing to prevent bradycardia and long pauses, and transfer the patient to a hospital unit with the highest level of ECG monitoring surveillance where immediate defibrillation is available.