Hôpitaux de Lyon

RESUVal
RESEAU DES URGENCES DE LA VALLÉE DU RHÔNE

CONTUSION MYOCARDIQUE

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INTRODUCTION

- **Contusion Myocardique =**
  - Dommage Tissulaire
  - Fait suite à un Trauma Thoracique non Pénétrant
- **Initialement association critères échographiques et biologiques**

Frazee et al. J. Trauma1986

- **Puis regroupement en une entité « Traumatisme Cardiaque Fermé »**

Rudusky et al. Angiology 2007

- **Incidence** : 8 à 76 %

Feghali NT et al. Chest 1995
Maenza et al. Am J Emerg Med 1996
Edouard AR et al. Anesthesiology 2004

- **Série Autopsique** : 14 %

Kaye P et al. 2002

Stade 0. Suspicion : pas d'argument clinique ou paraclinique franc
Pas de symptôme cardiaque
Pas d'arythmie cardiaque autre qu'une tachycardie sinusale
Pas d'anomalie échocardiographique
Pas d'élévation ou élévation faible des enzymes cardiaques (troponine T ou I)
Pas d'anomalie scintigraphique
Radiographie de thorax normale
Pas de séquelles résiduelles
Stade I. Faible : Diagnostique clinique non significatif ; pas de séquelles résiduelles
Symptômes cardiaques minimes, de durée et d'extension limitée (douleur pseudo angineuse, douleur thoracique atypique, palpitations)
Pas d'arythmies autres qu'une tachycardie sinusale ou des extrasystoles auriculaire ou ventriculaires
Anomalies du segment ST ou des ondes T minimes et transitoires (repolarisation, ischémie ou péricarde)
Élévation faible des enzymes cardiaques
Absence d'anomalie échocardiographique
Absence d'anomalie scintigraphique
Radiographie de thorax normale
Pas de séquelle résiduelle ou permanente
Stade II. Modérée : Diagnostique clinique significatif ; pas de séquelles permanentes
Douleur thoracique significative ou prolongée
Tachycardie sinusale marquée
Extrasystoles auriculaires ou ventriculaires fréquentes
Arythmie supra ventriculaire transitoire, limitée ou requérant une intervention minime
Anomalie du segment ST ou des ondes T significatives et persistant plusieurs jours ou plus (repolarisation, ischémie ou péricarde)
Élévation modérée des enzymes cardiaques
Anomalie échocardiographique : hypokinésie ou dyskinésie modéré, épanchement péricardique minime
Etude scintigraphique anormale
Radiographie de thorax anormale pouvant révéler des signes de traumatisme externes (fractures de côte ou du sternum)
Pas de séquelles permanente
Stade III. Sévère : Mise en jeu du pronostic vital; pouvant mener à des séquelles permanentes ou retardées
Douleur thoracique sévère et persistante
Tachycardie sinusale marquée et persistante
Arythmie supra ventriculaire nécessitant des traitements agressifs
Arythmie ventriculaire
Anomalies prolongées et significatives du segment ST ou des ondes T (repolarisation, ischémie, péricarde)
Élévation marquée des enzymes cardiaques, ou élévation modérée plus longue que prévue)
Anomalie échocardiographique majeure: hypokinésie marquée, dyskinésie, akinésie ou épanchement péricardique
Anomalie scintigraphique évidente
Certitude concernant un infarctus du myocarde résultant de la lésion myocardique ou secondaire à une lésion artérielle coronaire (thrombose, lacération)
Fistule artério-veineuse, lacération péricardique
Anomalie valvulaire ne requérant pas immédiatement un traitement médical ou chirurgical (anneau, feuillet, cordage)
Radiographie de thorax anormale révélant des signes de traumatisme externes et internes (épanchement pleural, contusion pulmonaire, congestion modérée des vaisseaux pulmonaires)
Il peut y avoir des séquelles permanente ou retardées
Stade IV : Catastrophique : séquelles permanentes ou décès
Signes et symptômes systémiques sévères (cardiaque, pulmonaires ou vasculaires)
Dysfonction valvulaire aigue nécessitant un traitement médical ou chirurgical (piliers, cordages ou désinsertion valvulaire)
Hernie cardiaque à travers une rupture péricardique avec des signes d'obstruction vasculaire nécessitant une intervention immédiate
Tamponnade
Insuffisance cardiaque aigue et sévère nécessitant une intervention immédiate et agressive
Rupture ventriculaire ou atrioseptale, rupture d'un gros vaisseau
Anévrysme myocardique, pseudo anévrysme myocardique, rupture myocardique
Séquelles permanentes ou mort hautement probable

Tableau 1: Classification de RuDusky.

Rudusky et al. Angiology 2007

PHYSIOPATHOLOGIE

- **Application d'une Pression sur le Myocarde**
  - Impact Direct (Sternum isolé, peu de risque)
  - Impact Indirect sur Structures Osseuses Adjacentes
  - Décélération Brutale (< 40 km/h)

Tenzer ML et al. J Trauma 1985

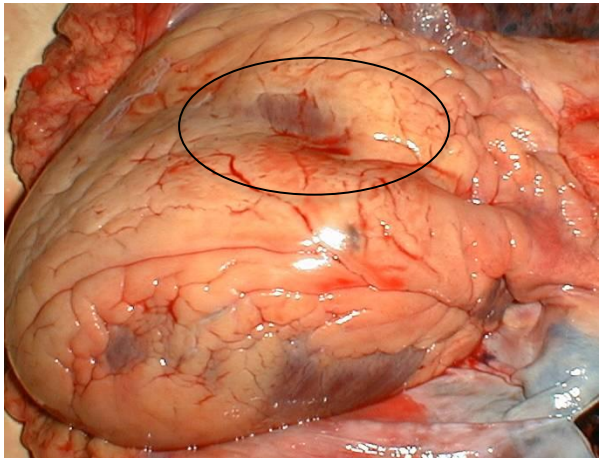
- **Energie pas Forcément très Elevée**
  - MCE
- **Le VD est le plus exposé**
- **Contusion** peut être aussi en relation avec un Blast
  - Thoracique : Faisant suite à une explosion ou du à un Effet arrière des gilets pare-balle
  - Myocardique : Libération d'énergie du projectile au cours de son passage intra-thoracique

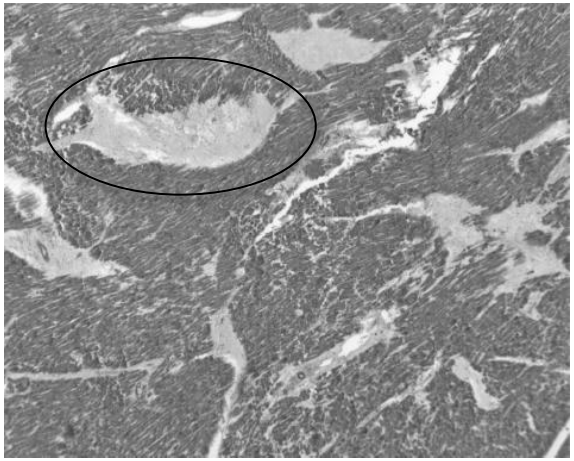
HISTOLOGIE

- **Gold standard pour le diagnostic**
- **Association de Zones de Nécrose et d'Hémorragie** réparties en tache (Patchy) sans Distribution Vasculaire **proche de l'ischémie**.
- **Différence avec ischémie** : Lésions progressives qui suivent la Distribution des Vaisseaux.

Tenzer ML et al. J Trauma 1985
Roxburgh JC. Injury 1996

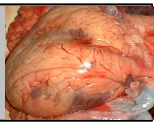
- **Cicatrisation** : Fibrose en Tache Irrégulière
- **Relation entre la Taille / Localisation** des Contusions et Clinique.





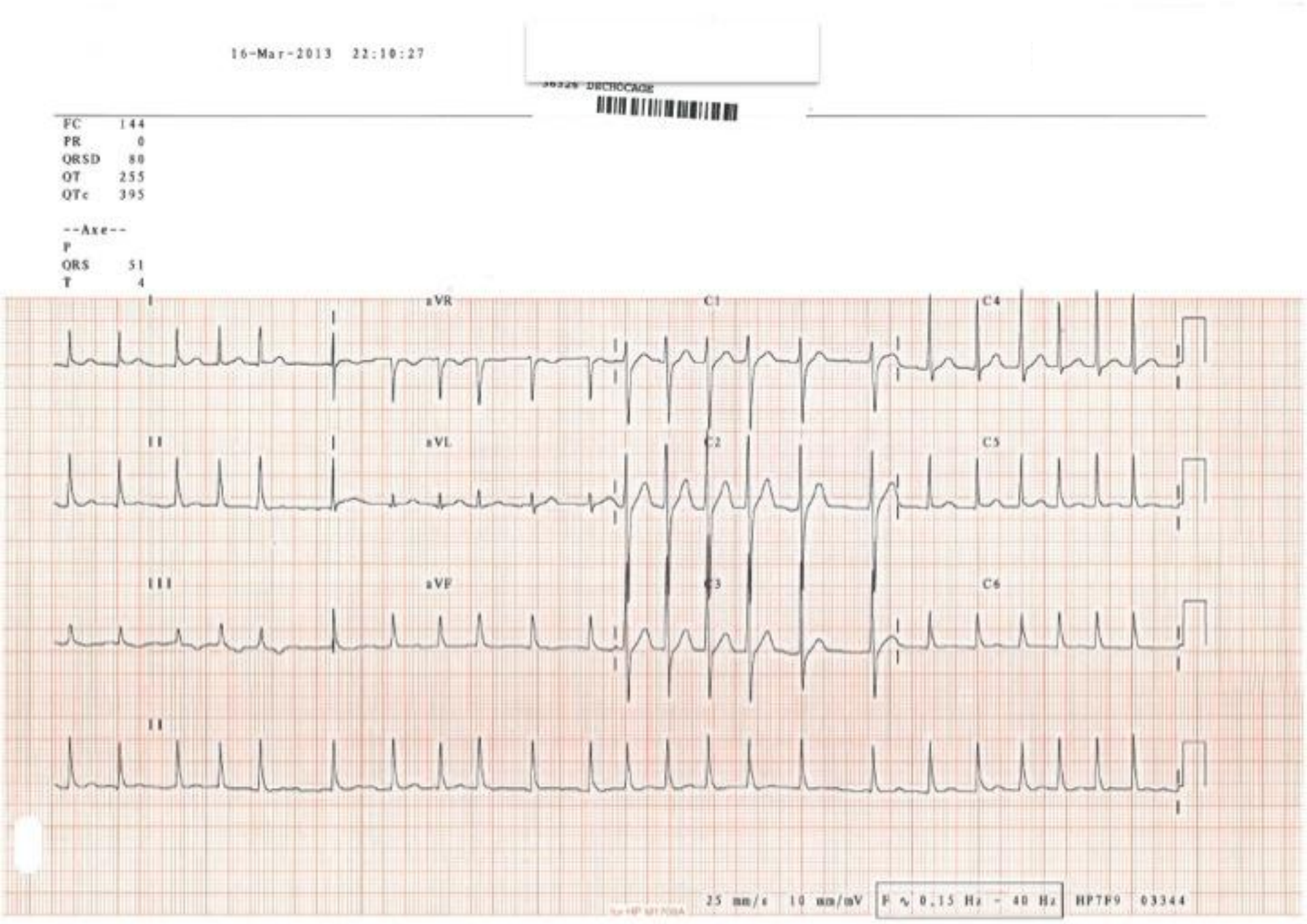
Gonon J et al. Am J Forensic Med Pathol 2009

PRÉSENTATION CLINIQUE

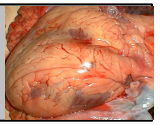


- Le plus souvent **Asymptomatique** Devitt et Al Can J Anaesth 197-200 (1993)
- **Peut être responsable de**
  - Trouble du Rythme (70%)
    - Auriculaire : ACFA …
    - Ventriculaire : TV, FV (Commotio Cordis)
  - Trouble de la Conduction (7%)
  - Anomalie de la Repolarisation
  - Insuffisance Cardiaque Aigue(8%)
  - Mort Subite

AVP Haute Cinétique, 30 ans - ACFA



ASSOCIATION LESIONNELLE

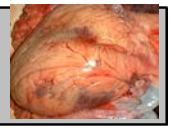


- **Lésion Extra-Thoracique** Fréquentes

Traumatisme Crânien++

- **Lésions Thoraciques**
  - Lésions Osseuses et/ou Parenchymateuses: doivent faire rechercher un traumatisme cardiaque, écarté si Tropo et ECG neg
  - 62% des lésions aortiques s'accompagnent d'un trauma Cardiaque

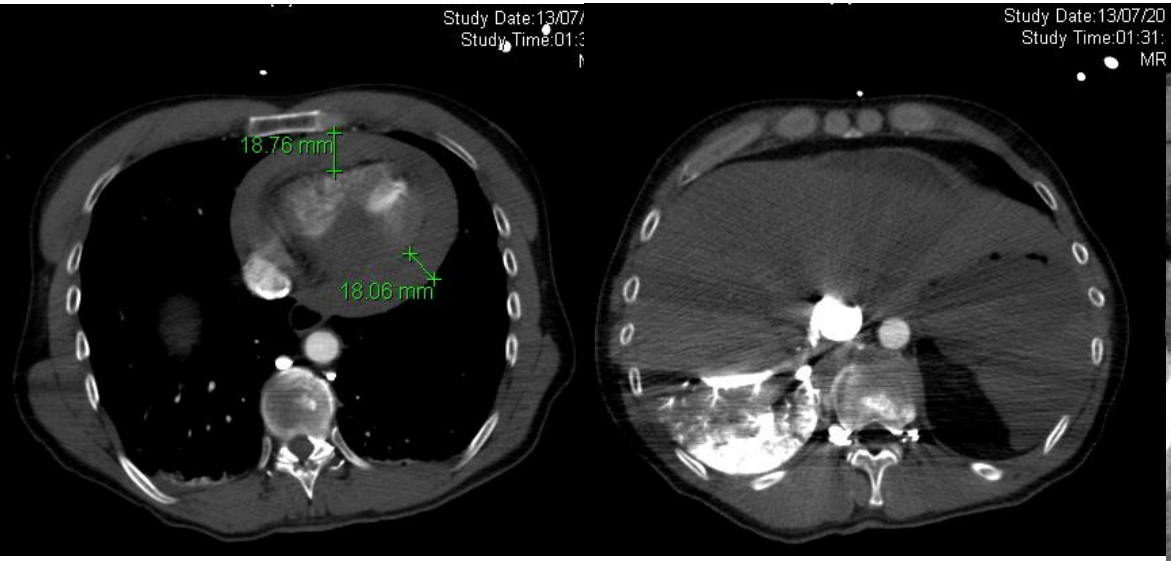
ASSOCIATION LESIONNELLE

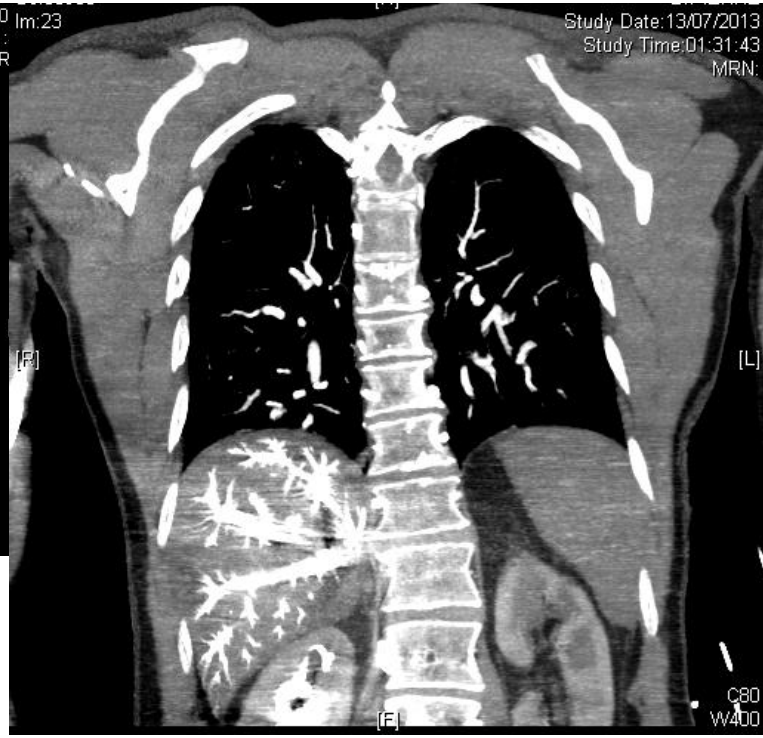


- Lésions Myocardiques
  - Valves : G > D
  - Paroi : Rupture Oreillette D > G, Anévrysme du VG
  - Péricarde (Tamponnade)
  - Coronaire : Très rare dissection coronaire associée
    - Tableau de SCA ST+

TAMPONNADE TRAUMA FERME

Homme 49 ans
Rixe avec coup de poing sternal
10-15 min plus tard, malaise au volant de son VL avec AVP basse cinétique
PEC SMUR : HypoTA 80 mmHg, FC ≈ 50 batt.min-1, GCS 15, SpO_2 90 en air
Déchocage : PAS ≈ 100-110 mmHg – FAST négative sauf Hémopéricarde



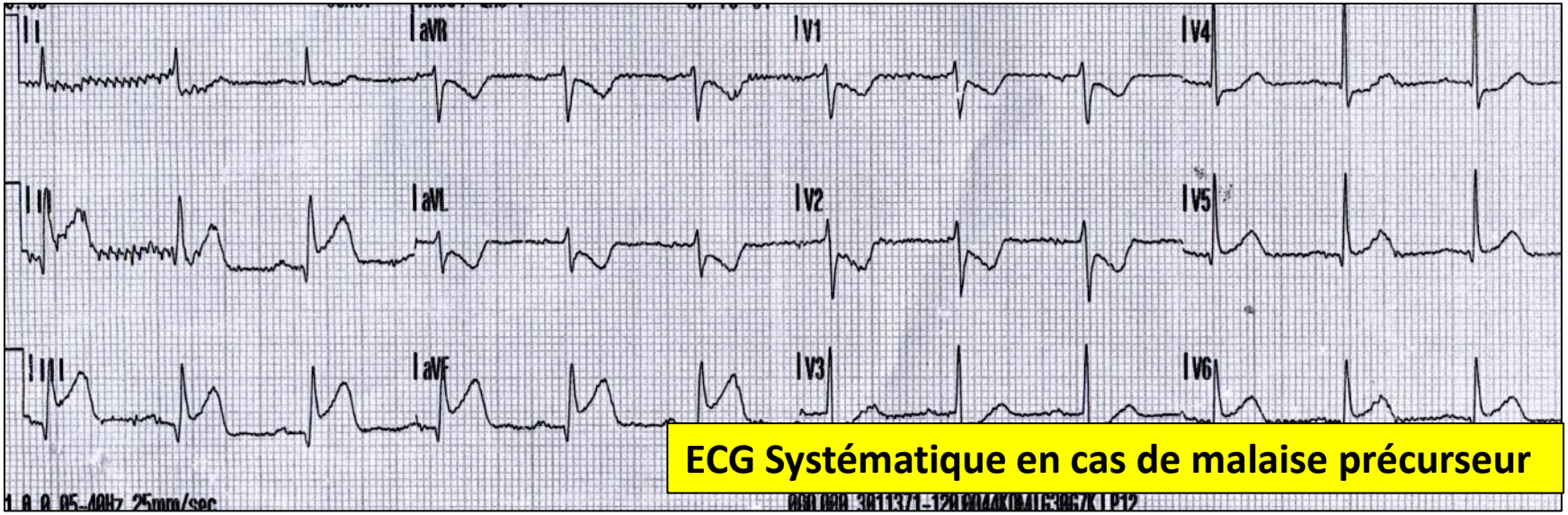


	01h36	08:40	17:00
Tropo	<0,04	1,74	2,32
Fibri	0,6		
Ac Lact	7,1		7,31

Hémopéricarde : 1,5 L …

ASSOCIATION LESIONNELLE

- **Lésions Non Traumatiques**
  - Trauma résulte ou cause d'une I Coronaire …
  - Séquence Malaise puis Accident
  - Ou Séquence Trauma (Epreuve d'Effort) puis I Coronaire …



ECG Systématique en cas de malaise précurseur

Ordre de la Séquence de Traitement : Dilatation/Stent par Rapport aux Lésions Trauma !

ASSOCIATION LESIONNELLE

- **Coronarographie en Urgence**
  - Stent nu sur l'IVA
  - AAP + HNF …
- **Dans les 2 heures …**
  - Etat de choc qui s'aggrave +++
  - DCD à H+3 post coronarographie / Hémorragie +++
- **Stratégie Combinée !?**
  - Coronarographie + Embolisation et/ou Chirurgie
  - AAP / HNF décalés ? Dilatation sans Stent ?
  - Centre Spécialisé …

PRONOSTIC

- **Bon à long terme** sauf lésions cardiaques associées

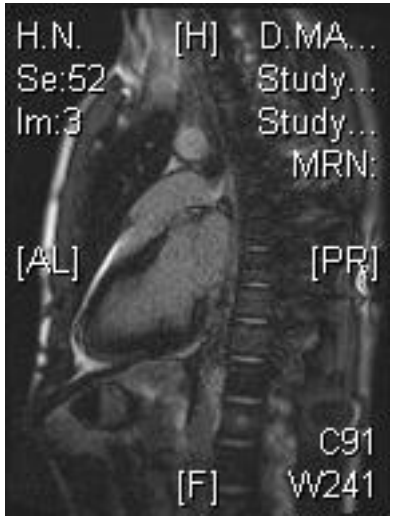
Lindstaedt M et al. J Trauma 2002
Bertinchant JP et al. J Trauma 2000

- **Séquelles Graves possibles (50 % anomalies persistantes à 10 semaines)**
  - I Cardiaque
  - Arythmie
  - Pathologies Valvulaires
  - Anévrysme du VG

Tenzer ML et al. J Trauma 1985
Maenza RL et al. Am J Emerg Med 1996

DIAGNOSTIC

- **ECG**
- **Dosage Biologique**
  - CPK MB
  - Troponine I / T
- **Echographie Cardiaque**
  - Tr de la Cinétique Segmentaire,
  - Epanchement Péricardique,
  - Valve
- **IRM**
- **Scintigraphie Myocardique**



Oui mais pas faisable à la phase initiale

ELECTROCARDIOGRAMME

- **ECG Systématique en cas**
  - Trauma Thoracique et/ou Troponine Positive
  - ATCD Cardiaque, malaise précédant accident
- **En SMUR si Contexte Coronaropathie Aigue**
- **Signes à Confronter au Contexte**
  - Tr Rythme ou Conduction
  - Tr de la Repolarisation / Sus ou Sous Décalage ST

ELECTROCARDIOGRAMME

- **Anomalies ECG dans 5 à 83 % des cas …**

Edouard AR et al. Anesthesiology 2004
Taenzer ML et al. J Trauma 1985
Bertinchant JP et al. J Trauma 2000

- **ECG non suffisant pour le Diagnostic car :**
  - Signes Non Spécifique
  - Peut être Normal avec une Contusion …
- **Néanmoins, Pour Certains VPN 80-90 % …**

Taenzer ML et al. J Trauma 1985
Bertinchant JP et al. J Trauma 2000
Velmahoc GC et al. J Trauma 2003

ECG EVOLUTIF !!

ELSEVIER

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International Journal of Cardiology

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Letter to the Editor

Cardiac contusion: Ending myocardial confusion in this capricious syndrome

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Abstract

Symptoms of cardiac contusion are very greatly and sometimes are non recognized or are masked by associated injury in severe chest trauma. Cardiac contusion clinically presents as a spectrum of signs and symptoms of varying severity, ranging from precordial pain, dyspnoea, and non specific ECG changes to increased serum activity of several enzymes, early severe rhythm abnormalities, severe conduction defects and death. We present a fatal case in which the definitive diagnosis of myocardial contusion has proved complex. All clinical data were suggestive of acute myocardial infarction, but the history of chest wall injury and gross and histological examination of the heart and coronary vessels led us to conclude for a cardiac contusion without myocardial infarction. In case of chest blunt trauma, the ECG should be interpreted within the context of the clinical situation, on history of chest wall injury, since a fatal myocardial contusion may occur after apparently mild injury.

Keywords: Cardiac contusion; Blunt chest trauma; Acute myocardial infarct; Diagnosis; Troponins

1. Introduction

Although the Cardiac Contusion (CC) is a well defined pathological entity, however the clinical diagnosis of this "capricious syndrome" is not easy. Symptoms of CC are very greatly and sometimes are non recognized or are masked by associated injury in severe chest trauma. Cardiac contusion clinically presents as a spectrum of signs and symptoms of varying severity, ranging from precordial pain, dyspnoea, and non specific ECG changes to increased serum activity of several enzymes, early severe rhythm abnormalities, severe conduction defects and death [1,2]. Therefore, frequently, the clinical signs of cardiac contusion are similar to those of an myocardial infarction.

We report the case of a young man, with blunt chest trauma after a motorcycle crash, who died 8 h since his hospitalization after four episode of VF. The moderate trauma, the clinical symptoms and the ECG alterations led physicians to diagnose an AMI (acute myocardial infarct) as cause of death. The autopsy, instead, revealed the typical findings of a cardiac contusion.

2. Case report

A 41 year old man after a motorcycle crash, was transported by emergency medical service to the Emergency Department. On admission (2:00 AM) the patient was awake and alert with GSC (Glasgow Coma Scale) 15/15, complaining of general and diffuse pain. The haemodynamic parameters were normal.

On physical examination he presented only a frontal laceration, a second-degree burn at right leg and generalized pain. Blood samples for laboratory tests were taken.

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A Répéter si Anomalies !

CPK MB

- **Mauvais Marqueur**
- **CPK-MB augmente** en cas de Trauma Musculaire Etendu
- **N’est pas** un critère prédictif de mortalité ou complication

TROPONINE I

- **Plus Sensible que la Troponine T**

Bertinchant JP et al. J Trauma 2000

- **Travail Prospectif, 44 Tr Thoracique**
  - Contusion Myocardique = ECG + Echo
  - Se 100 %, Sp 97 %

Adams JE et al. Am Heart J 1996

- **Troponine I ≥ 0,1** $\mu g.l^{-1}$:
  - Très spécifique (97 %)
  - Peu sensible (23 %)

Bertinchant JP et al. J Trauma 2000

TROPONINE I

- **Relation Troponine I et Trouble du Rythme**
  - **Troponine > 1,05** $\mu g.l^{-1}$: Risque de Trouble du Rythme
  - ↗ Troponine de 1 point =
    - ↗ Arythmie de 1,9 %
    - ↘ Baisse FE de 0,4 %
- **<u>Elimination d'une Contusion Myocardique si</u> :**
  - Asymptomatique et Troponine I < 1,05 $\mu g.l^{-1}$

    Rajan JP et al. J Trauma 2004
  - ECG Normale et Troponine I < 1,5 $\mu g.l^{-1}$

    Velmahos GC et al. J Trauma 2003

Anesthesiology 2004; 101:1262–8

Incidence and Significance of Cardiac Troponin I Release in Severe Trauma Patients

Alain R. Edouard, M.D., Ph.D., Marie-Louise Felten, M.D.,† Jean-Louis Hebert, M.D., Ph.D.,‡ Claudine Cosson, Ph.D.,§ Laurent Martin, M.D.,‖ Dan Benhamou, M.D.#*

Background: The incidence and significance of troponin I release and its mechanism are unknown in severe trauma patients. The characteristics of this release were prospectively studied in such patients and correlated with presence of shock, existence of myocardial contusion, and outcome.

Methods: During a 24-month period, serial electrocardiogram recordings and troponin I measurements were performed in all trauma patients admitted at a surgical intensive care unit. The diagnosis of a significant myocardial contusion was made on electrocardiographic criteria. According to the time course of troponin I, three groups of patients were defined *a priori*: very transient (≤ 12 h) and limited release (troponin I < 2 μg/l), transient (≤ 36 h) and significant release (troponin I ≥ 2 μg/l), and sustained (> 36 h) and significant release (troponin I > 2 μg/l). In the last group, coronary artery angiography was performed.

Results: The incidence of troponin I release was 12% (95% confidence interval [CI], 9.6–14.4%) in 728 patients. A significant myocardial contusion was found in 35 patients (5%; 95% CI, 3.4–6.6%) and may occur in the absence of chest trauma and without troponin I release. Sensitivity, specificity, and positive and negative predictive values of troponin I for the diagnosis of myocardial contusion were 63, 98, 40, and 98%, respectively. Troponin I release was observed in 54 early (> 48 h) survivors (7%; 95% CI, 5.6–9.6%) without preexisting coronary artery disease. A sustained and significant release of troponin I (17 patients) was frequently associated with chest trauma (82%) and constantly with electrocardiographic abnormalities. A coronary artery injury was found in 7 patients (2 major and 5 minor vascular injuries) (1% of the whole group; 95% CI, 0.4–2.0%). Mortality was similar in early survivors with (15%; 95% CI, 7–27%) or without (12%; 95% CI, 9–14%) troponin I release. The odds ratio for late mortality was 1.32 (95% CI, 0.61–2.85) in patients with troponin I release.

Conclusions: Serial electrocardiogram recordings and troponin I assessments may be proposed for initial screening in high-risk trauma patients to detect anatomical cardiac injuries through the time course of circulating protein. Troponin I release does not have a prognosis value in trauma patients.

THE routine repeated assessment of cardiac enzymes for detecting a blunt cardiac injury or myocardial injury is a controversial practice during the treatment of trauma patients.[1] The lack of cardiac specificity of creatine kinase MB isoform greatly contributes to explain the low diagnosis value of this marker for such an elusive pathology in patients who generally have peripheral muscle injuries. Conversely, immunoassays provide an accurate assessment of troponin I cardiac release, which gains popularity for the diagnosis of myocardial contusion even if the diagnosis time window is narrower than that reported for "medical" myocardial ischemia.[2,3] However, circulating troponin I has been observed in a large range of clinical settings, especially circulatory failure of septic or hemorrhagic origin in the absence of direct cardiac trauma.[4–7] Because studies about troponin I have generally been concerned with limited series of selected chest trauma patients, the aims of the current prospective observational study were therefore (1) to determine the incidence and the profile of troponin I release in consecutive severe blunt trauma patients admitted at a level I surgical and trauma intensive care unit and (2) to correlate the biologic results with the presence of shock, the existence of myocardial contusion, and the outcome of the patients.

Materials and Methods

The investigation was approved by the hospital review board (Le Kremlin Bicêtre, France). Because data were recorded without any specific intervention and according to a protocol used in routine in the unit, authorization was given to waive informed consent in most trauma patients. Nevertheless, informed consent was obtained from all subjects or from their relatives when coronary angiography was performed because this investigation was not previously performed on a routine basis. During a period of 24 months (January 2001 to December 2002), all patients consecutively admitted in the unit with significant blunt trauma were prospectively included in the study. All these patients were cared for by a mobile intensive care unit (French Service d'Aide Médicale Urgente system).[8] The severity of the trauma was considered high enough by the prehospital team to war-

This article is accompanied by an Editorial View. Please see: Riou B: Troponin: Important in severe trauma and a first step in the biological marker revolution. ANESTHESIOLOGY 2004; 101:1259–60.

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TROPONINE I

728 Trauma, Prospectif
Diagnostic Contusion = ECG
3 Groupes selon Troponine :

- < 2 μg.L^{-1} et < 12h
- > 2 μg.L^{-1} et < 36h
- > 2 μg.L^{-1} et > 36h

- **Relargage Troponine = 12 % des cas**
- **Contusion 5 %**
- **> 2 μg.L^{-1} et prolongé (> 36h) 17 patients = 7 Lésions Coronaires !!**

LESIONS CORONAIRES

CLINICAL INVESTIGATIONS

Anesthesiology 2004; 101:1262–8 © 2004 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

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Results: The incidence of troponin I release was 12% (95% confidence interval [CI], 9.6–14.4%) in 728 patients. A significant myocardial contusion was found in 35 patients (5%; 95% CI, 3.4–6.6%) and may occur in the absence of chest trauma and without troponin I release. Sensitivity, specificity, and positive and negative predictive values of troponin I for the diagnosis of myocardial contusion were 63, 98, 40, and 98%, respectively. Troponin I release was observed in 54 early (> 48 h) survivors (7%; 95% CI, 5.6–9.6%) without preexisting coronary artery disease. A sustained and significant release of troponin I (17 patients) was frequently associated with chest trauma (82%) and constantly with electrocardiographic abnormalities. A coronary artery injury was found in 7 patients (2 major and 5 minor vascular injuries) (1% of the whole group; 95% CI, 0.4–2.0%). Mortality was similar in early survivors with (15%; 95% CI, 7–27%) or without (12%; 95% CI, 9–14%) troponin I release. The odds ratio for late mortality was 1.32 (95% CI, 0.61–2.85) in patients with troponin I release.

Conclusions: Serial electrocardiogram recordings and troponin I assessments may be proposed for initial screening in high-risk trauma patients to detect anatomical cardiac injuries through the time course of circulating protein. Troponin I release does not have a prognosis value in trauma patients.

THE routine repeated assessment of cardiac enzymes for detecting a blunt cardiac injury or myocardial injury is a controversial practice during the treatment of trauma patients.[1] The lack of cardiac specificity of creatine kinase MB isoform greatly contributes to explain the low diagnosis value of this marker for such an elusive pathology in patients who generally have peripheral muscle injuries. Conversely, immunoassays provide an accurate assessment of troponin I cardiac release, which gains popularity for the diagnosis of myocardial contusion even if the diagnosis time window is narrower than that reported for "medical" myocardial ischemia.[2,3] However, circulating troponin I has been observed in a large range of clinical settings, especially circulatory failure of septic or hemorrhagic origin in the absence of direct cardiac trauma.[4–7] Because studies about troponin I have generally been concerned with limited series of selected chest trauma patients, the aims of the current prospective observational study were therefore (1) to determine the incidence and the profile of troponin I release in consecutive severe blunt trauma patients admitted at a level I surgical and trauma intensive care unit and (2) to correlate the biologic results with the presence of shock, the existence of myocardial contusion, and the outcome of the patients.

Materials and Methods

The investigation was approved by the hospital review board (Le Kremlin Bicêtre, France). Because data were recorded without any specific intervention and according to a protocol used in routine in the unit, authorization was given to waive informed consent in most trauma patients. Nevertheless, informed consent was obtained from all subjects or from their relatives when coronary angiography was performed because this investigation was not previously performed on a routine basis. During a period of 24 months (January 2001 to December 2002), all patients consecutively admitted in the unit with significant blunt trauma were prospectively included in the study. All these patients were cared for by a mobile intensive care unit (French Service d'Aide Médicale Urgente system).[8] The severity of the trauma was considered high enough by the prehospital team to war-

This article is accompanied by an Editorial View. Please see: Rivo B: Troponin: Important in severe trauma and a first step in the biological marker revolution. ANESTHESIOLOGY 2004; 101:1259–60.

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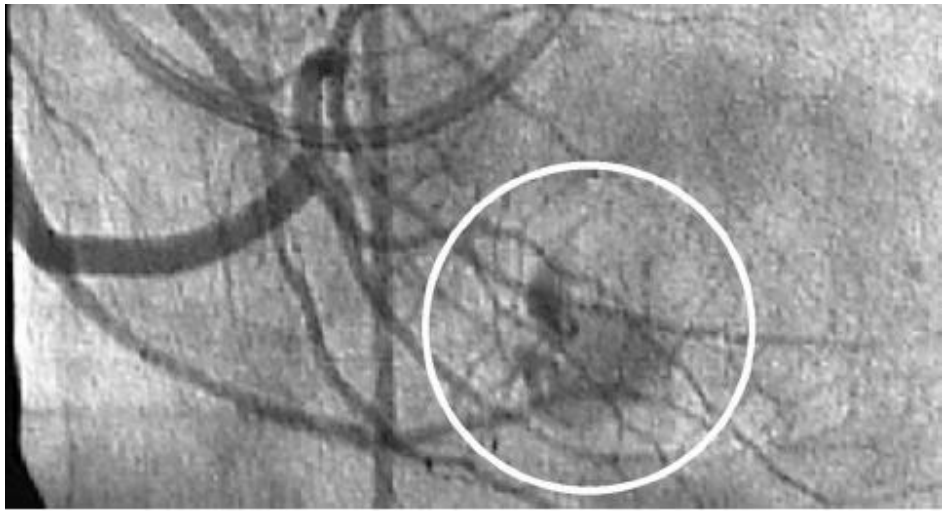
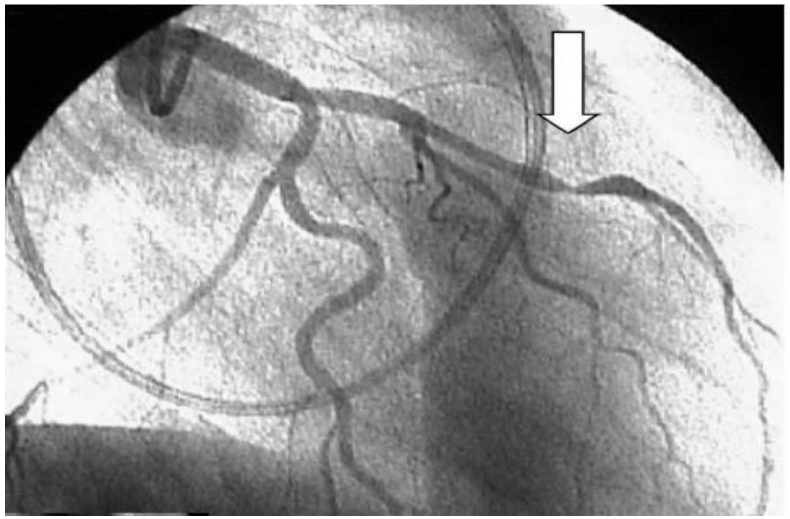
Incidence 1 %

Clinique

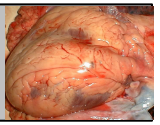
- Très Variable
- Patient Sédaté
- Etat de Choc

Coronarographie ou CoroScanner si Doute

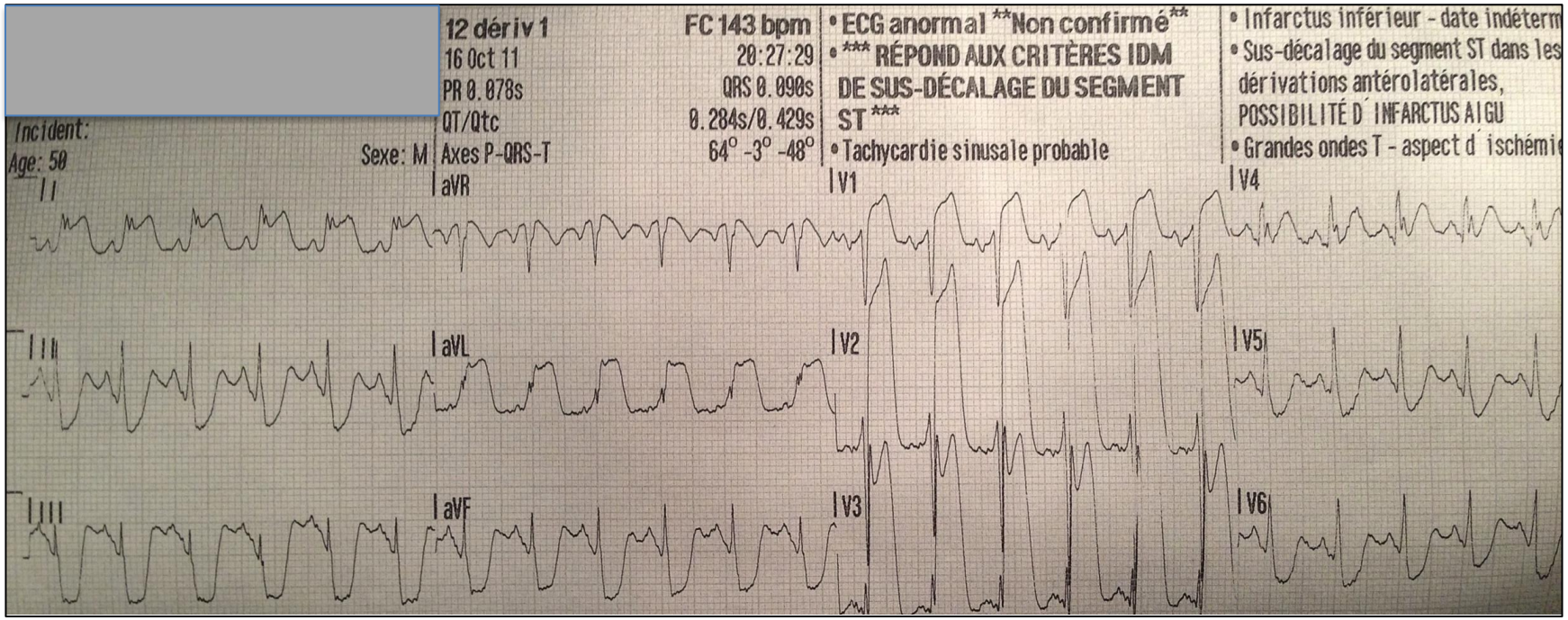




ASSOCIATION LESIONNELLE



- **TS Arme à Feu, H49 ans, 9 mm**
  - Plaie para-mamelonaire Gauche
  - SMUR : Fc 120 batt/min, PAS 80 mmHg



Ordre de la Séquence de Traitement : Dilatation/Stent par Rapport aux Lésions Trauma !

ASSOCIATION LESIONNELLE

Trajet balistique :

- Orifice d'entrée thoracique antérieur sous mamelonaire gauche
- **Fracture 12 ème côte** G et **arrêt en paravertébrale T12** gauche.
- **Hémopéritoine** intéressant la loge rénale gauche, les 2 gouttières pariéto- colique, péri hépatique et pelvien sans extravasation de produit de contraste, **dilacération pôle supérieur** rein gauche.

Et la Rate ????

The American Journal of Emergency Medicine
Volume 30, Issue 1, January 2012, Pages 266.e3–266.e5

Case Report

Acute myocardial infarction and multiple traumas: a dilemma of protocol

Loic Chimot, MD
Lionel Leroux, MD
Nicolas Morel, MD

TIME

Targeted cardiac examination included in the quick initial check list for a patient with multiple traumas

ECG	-Diagnose potentiality of ischemic complications -Evidence whether heart attack was responsible for the accident -Reference ECG for the following (troponin that subsequently increases is difficult to interpret)
Echocardiography	-Associat systematically with general echography -Heart lesion: contusion, tamponade, papillary muscle rupture, acute aortic, mitral or tricuspid regurgitation -Other complications guided by clinical examination or ECG

Prioritization of emergency treatment →

-Control blood loss
-Hemodynamic stabilization
-Respiratory stabilization
-Treat neurological emergency

Acute myocardial infarction-specific treatment

-Diagnostic coronarography

-If necessary, consider balloon-only dilatation

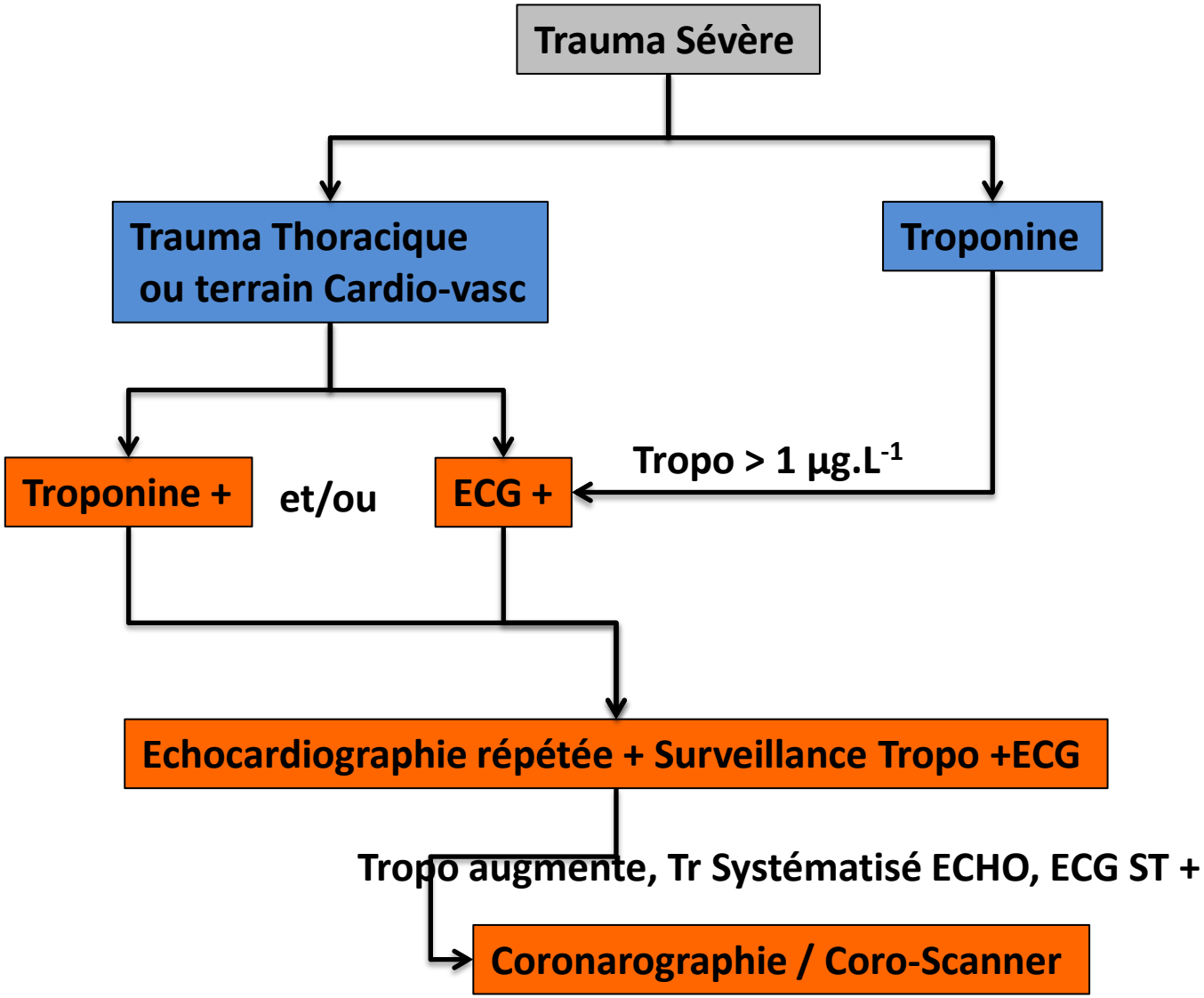
-Limit anti-aggregation treatment

-Non-fractioned heparin treatment (can be reversed quickly with protamine)

-Treat cardiac failure: inotropic agent, intra-aortic balloon

-If previous step fail, consider quick cardiac assistance

ALGORITHME


Trauma Sévère
Trauma Thoracique
ou terrain Cardio-vasc
Troponine
Troponine +
et/ou
ECG +
Tropo > 1 µg.L-1
Echocardiographie répétée + Surveillance Tropo +ECG
Tropo augmente, Tr Systématisé ECHO, ECG ST +
Coronarographie / Coro-Scanner

TRAITEMENT

- **Si Contusion (ECG + Tropo) :**
  - **USC 48 h+ETT**
  - **Cycle ECG + Tropo**
- **Traitement Symptomatique**
  - Tr Rythme
  - I Cardiaque (ECLS, CPDIA)
- **Traitement des Complications :**
  - Lésions Valvulaire
  - Hémopéricarde
- **Traitement Lésions Préexistantes Coronaires**
  - Gestion des AAP compliquées …

COMMOTIO CORDIS

Représente un exemple **de trouble du rythme létal**
Décrit initialement :

- **Jeunes athlètes**
- Victimes d'un traumatisme thoracique
- Intensité modérée voire faible

Pathologie avant tout **Sportive** / Ado – Jeune Adulte

1. **Sport avec :**
   - Percutions thoracique : Karaté, Box
   - Impact Thoracique : Base ball, Hockey
2. **Autres Circonstances avec Impact**
   - Accident
   - Arme non létale

Mise à jour 21:02

LE FIGARO.fr ACTUALITÉ ÉCONOMIE CULTU

Un homme dans un état grave après un tir de flash-ball

Mots clés : Flash-Ball, Tir, Blessé, FRANCE, MARSEILLE, Brice Hortefeux

Par lefigaro.fr

12/12/2010 | Mise à jour : 20:43 Réagir

S'ABONNER AU FIGARO.FR - 8 €/mois

Des pompiers stationnent dimanche devant la résidence de la Madrague, à Marseille, pour évacuer l'homme victime d'un arrêt cardiaque à la suite d'un tir de flash-ball. Crédits photo : ANNE-CHRISTINE POUJOULAT/AFP

L'homme, âgé d'une quarantaine d'années, a été victime d'un arrêt cardiaque après avoir reçu un tir de flash-ball d'un policier, qui intervenait pour mettre un terme à une rixe dans un foyer de travailleurs.

Un homme était dans un état critique dimanche à Marseille, victime d'un arrêt cardiaque après avoir reçu un tir de flash-ball d'un policier, ont indiqué la police et le parquet. Le policier intervenait pour mettre un terme à une rixe survenue dans un foyer de travailleurs.

L'homme, âgé d'une quarantaine d'années et résidant dans ce foyer situé dans le 15e arrondissement de la ville, avait

COMMOTIO CORDIS

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

MEDICAL PROGRESS

Commotio Cordis

Barry J. Maron, M.D., and N.A. Mark Estes III, M.D.

VENTRICULAR FIBRILLATION AND SUDDEN DEATH TRIGGERED BY A BLUNT, nonpenetrating, and often innocent-appearing unintentional blow to the chest without damage to the ribs, sternum, or heart (and in the absence of underlying cardiovascular disease) constitute an event known as commotio cordis, which translates from the Latin as agitation of the heart. This term was first used in the 19th century,[1-3] although the occurrence of commotio cordis was described earlier, in accounts of the ancient Chinese martial art of Dim Mak (or touch of death), in which blows to the left of the sternum caused sudden death in opponents.[3] An absence of structural cardiac injury distinguishes commotio cordis from cardiac contusion, in which high-impact blows result in traumatic damage to myocardial tissue and the overlying thorax.

Beginning in the mid-1700s, sporadic accounts of commotio cordis appeared in the medical literature, mostly in the context of workplace accidents,[4-10] and through the mid-1990s, the disorder was noted only occasionally in case reports, going largely unrecognized, except by the forensic pathology community and the Consumer Product Safety Commission.[6,11-14] Since then, however,[1] both the general public and the medical community have become increasingly aware of commotio cordis as an important cause of sudden cardiac death. It occurs primarily in children, adolescents, and young adults, most often during participation in certain recreational or competitive sports, with rare occurrences during normal, routine daily activities.[1,2,15-19]

Continued interest in commotio cordis and its tragic consequences is evident in epidemiologic studies[1,2,19] and a number of experimental laboratory investigations.[20-35] This review focuses on the available information regarding the clinical profile, proposed mechanisms, and prevention and treatment of commotio cordis.

From the Hypertrophic Cardiomyopathy Center Minneapolis Heart Institute Foundation, Minneapolis (B.J.M.); and the New England Cardiac Arrhythmia Center, Cardiology Division, Tufts University School of Medicine, Boston (N.A.M.E.). Address reprint requests to Dr. Maron at Minneapolis Heart Institute Foundation, 920 E. 28th St., Suite 620, Minneapolis, MN 55407, or at hcm.maron@mhif.org.

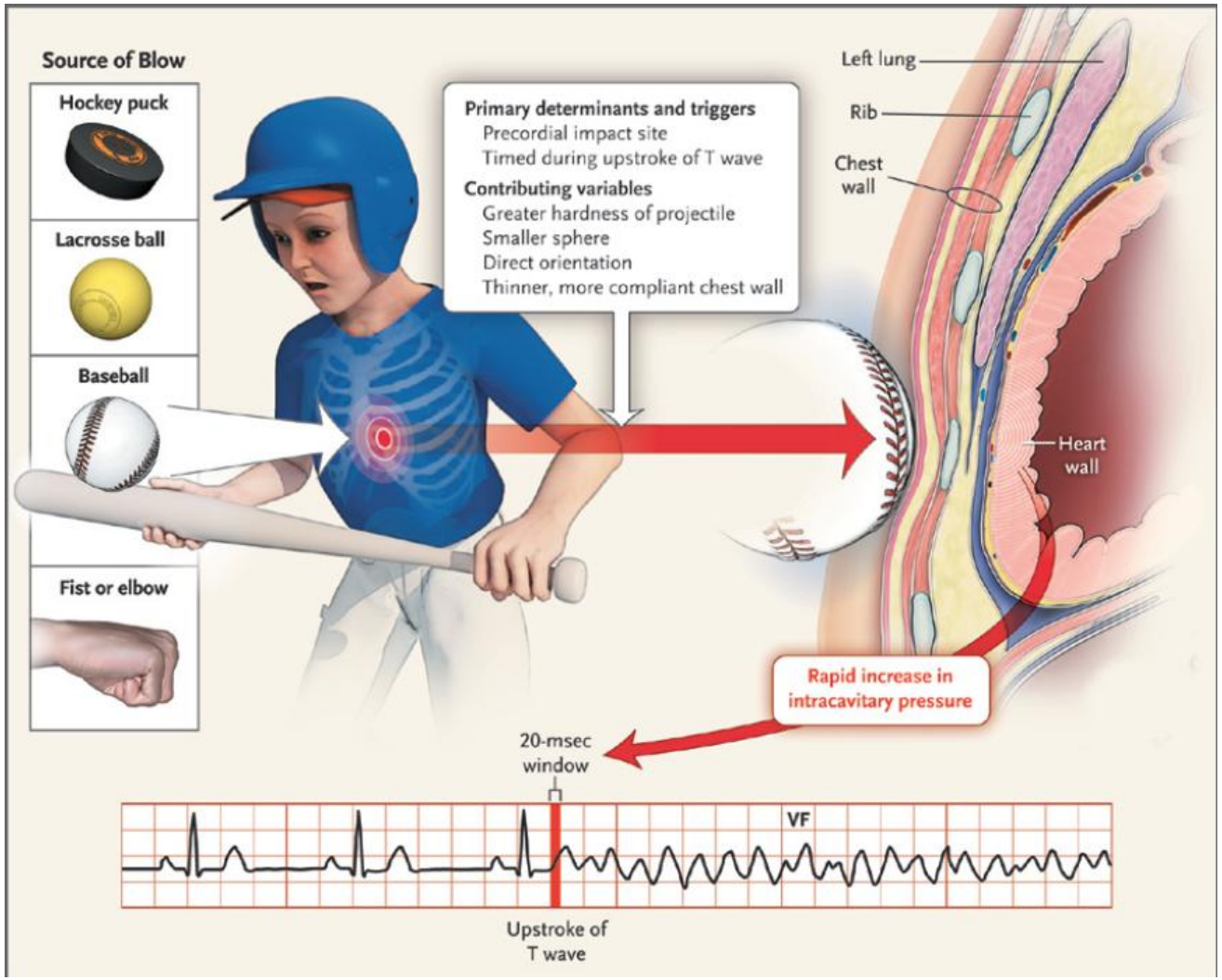
N Engl J Med 2010;362:917-27.

INCIDENCE

The precise incidence of commotio cordis is unknown because of the absence of systematic and mandatory reporting, but on the basis of data from the National Commotio Cordis Registry in Minneapolis,[1,2,17] it is among the most frequent cardiovascular causes of sudden death in young athletes, after hypertrophic cardiomyopathy and congenital coronary-artery anomalies.[17,18] Since commotio cordis occurs in a wide variety of circumstances, it has undoubtedly been underreported but it is being recognized with increasing frequency and is probably more common than it is believed to be.

EPIDEMIOLOGY

In addition to episodic case studies,[6,12-14,36-44] more specific information concerning the clinical profile of commotio cordis comes from the Minneapolis registry,

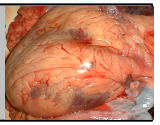


Maron BJ – NEJM 2010

TT COMMOTIO CORDIS

Celui d'un AC par **Trouble du Rythme Primaire**
Réanimation Cardio-Pulmonaire
MCE + Défibrillation Précoce
Si No-Flow < 3-5 min et CHU Proche : ECLS ?

CONCLUSION



- Le plus souvent Asymptomatique
- En dehors lésions associées, bon pronostic
- Diagnostic = ECG + Troponine / Echographie
- Contusion = USC 48h
- Difficultés = Différencier
    - Montée Troponine (choc …)
    - Contusion Myocardique
    - SCA