« SCA chez les patients COVID »

P.Commeau, Ollioules

H.Benamer, Aubervilliers, modérateur

ESC European Society of Cardiology

European Heart Journal (2020) **0**, 1–13
doi:10.1093/eurheartj/ehaa381

FASTTRACK CLINICAL RESEARCH
Interventional cardiology

EAPCI Position Statement on Invasive Management of Acute Coronary Syndromes during the COVID-19 pandemic

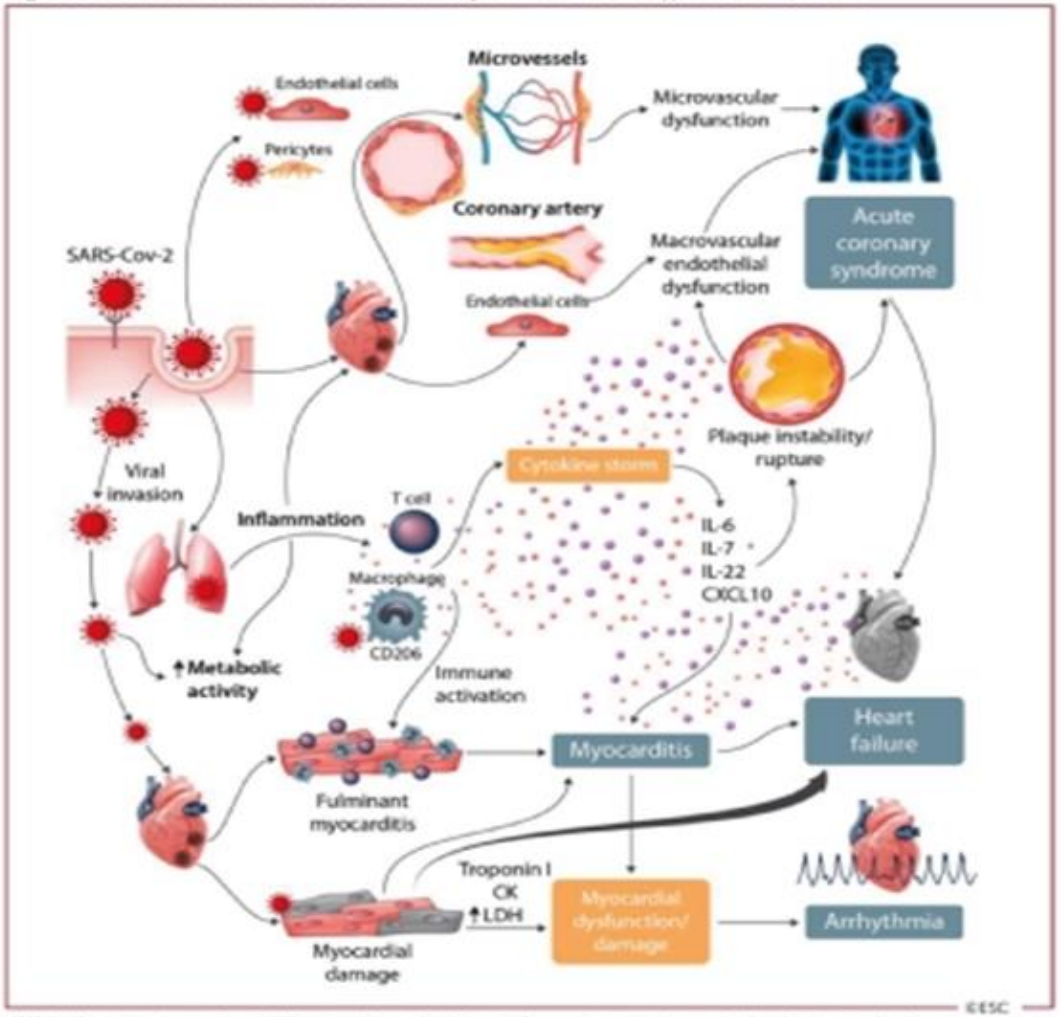
Alaide Chieffo (Chair) [1], Giulio G. Stefanini [2], Susanna Price[3], Emanuele Barbato[4], Giuseppe Tarantini[5], Nicole Karam [6], Raul Moreno[7], Gill Louise Buchanan[8], Martine Gilard[9], Sigrun Halvorsen [10], Kurt Huber[11], Stefan James[12], Franz-Josef Neumann [13], Helge Möllmann[14], Marco Roffi[15], Guido Tavazzi [16], Josepa Mauri Ferré[17], Stephan Windecker [18], Dariusz Dudek[19†], and Andreas Baumbach (co-Chair) [20*†]

[1]Interventional Cardiology Unit, IRCCS San Raffaele Hospital, Milan, Italy; [2]Humanitas Clinical and Research Hospital IRCCS, Rozzano, Milan, Italy; [3]Royal Brompton Hospital, London, UK; [4]Department of Advanced Biomedical Sciences University Federico II, Naples, Italy; [5]Interventional Cardiology Unit, Department of Cardiac, Thoracic, Vascular Sciences and Public Health, University of Padova, Padua, Italy; [6]European Hospital Georges Pompidou (Cardiology Department) and University of Paris, Paris, France; [7]Cardiology Department, Hospital La Paz and IDIPAZ, Madrid, Spain; [8]Department of Cardiology, North Cumbria Integrated Care NHS Foundation Trust, Cumbria, UK; [9]Service de Cardiologie, Brest University Hospital, Brest, France; [10]Department of Cardiology, Oslo University Hospital Ulleval, and University of Oslo, Oslo, Norway; [11]3rd Department of Medicine, Cardiology and Intensive Care Medicine, Wilhelminen Hospital, and Sigmund Freud University, Medical School, Vienna, Austria; [12]Department of Medical Sciences, Cardiology and Uppsala Clinical Research Center, Uppsala University, Sweden; [13]Division of Cardiology and Angiology II University Heart Centre Freiburg, Bad Krozingen, Germany; [14]Department of Cardiology St.-Johannes-Hospital Klinik für Innere Medizin I, Dortmund, Germany; [15]Division of Cardiology, University Hospitals, Geneva, Switzerland; [16]Department of clinical-surgical, diagnostic and pediatric sciences, Unit of anaesthesia and intensive care, Fondazione Policlinico San Matteo Hospital IRCCS, Pavia, Italy; [17]Hospital Universitari Germans Trias i Pujol, Badalona, Spain; [18]Department of Cardiology, Swiss Cardiovascular Center, University Hospital Bern, Bern, Switzerland; [19]Institute of Cardiology, Jagiellonian University Medical College, Krakow, Poland and Maria Cecilia Hospital GVM, Cotignola, Ravenna, Italy; and [20]Centre for Cardiovascular Medicine and Devices, William Harvey Research Institute, Queen Mary University of London, Barts Heart Centre, London, UK and Yale University School of Medicine, New Haven, CT, USA

Received 30 March 2020; revised 15 April 2020; editorial decision 23 April 2020; accepted 6 May 2020; online publish-ahead-of-print 10 May 2020

Atteinte cardiaque et COVID 19

Figure 3 Cardiovascular involvement in COVID-19 – key manifestations and hypothetical mechanisms



SARS-CoV-2 anchors on trans-membrane ACE2 to enter the host cells including type-2 pneumocytes, macrophages, endothelial cells, pericytes and cardiac myocytes leading to inflammation and multi-organ failure. Infection of endothelial cells or pericytes is of particular importance because this could lead to severe microvascular and macrovascular dysfunction. In addition, immune over-reactivity can potentially destabilise atherosclerotic plaques and explain the development of acute coronary syndromes. Infection of the respiratory tract, particularly type-2 pneumocytes, by SARS-CoV-2 is manifested by the progression of systemic inflammation and immune cell over-activation leading to "cytokine storm", resulting in increased levels of cytokines such as IL-6, IL-7, IL-22 and CXCL10. Subsequently, it is possible that activated T cell and macrophages may infiltrate infected myocardium resulting in the development of fulminant myocarditis and severe cardiac damage. This process may be further intensified by a cytokine storm. Similarly, the viral invasion may cause cardiac myocyte damage directly leading to myocardial dysfunction and contribute to the development of arrhythmias. Frank Gaźik et al., COVID-19 and the cardiovascular system – implications for risk assessment, diagnosis and treatment options. Cardiovasc Res., 2020, doi: 10.1093/cvr/cvaa086.[4]

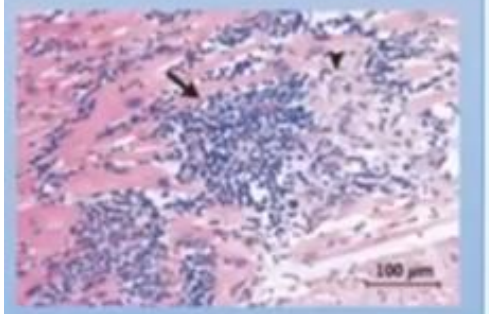
Mécanisme de la souffrance myocardique au cours de l'infection COVID-19 ?

Souffrance myocardique

Atteinte myocardique directe

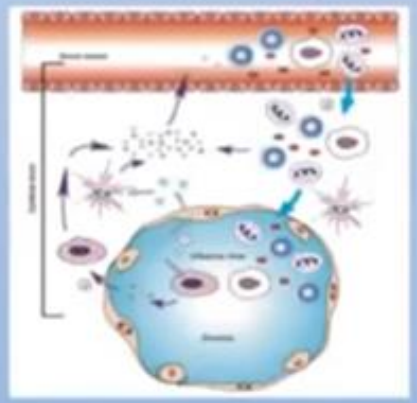
Atteinte myocardique indirecte

Myocardite



Cooper, N Eng J Med 2009

Orage cytokinique

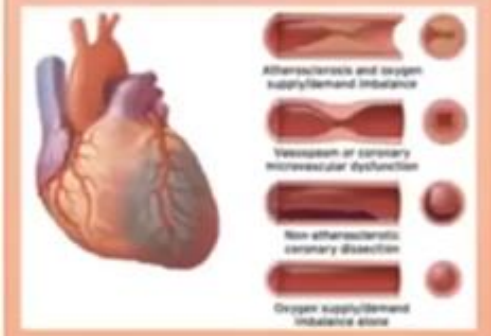


Liu, Cell Mol Immunol 2016

IDM type 1

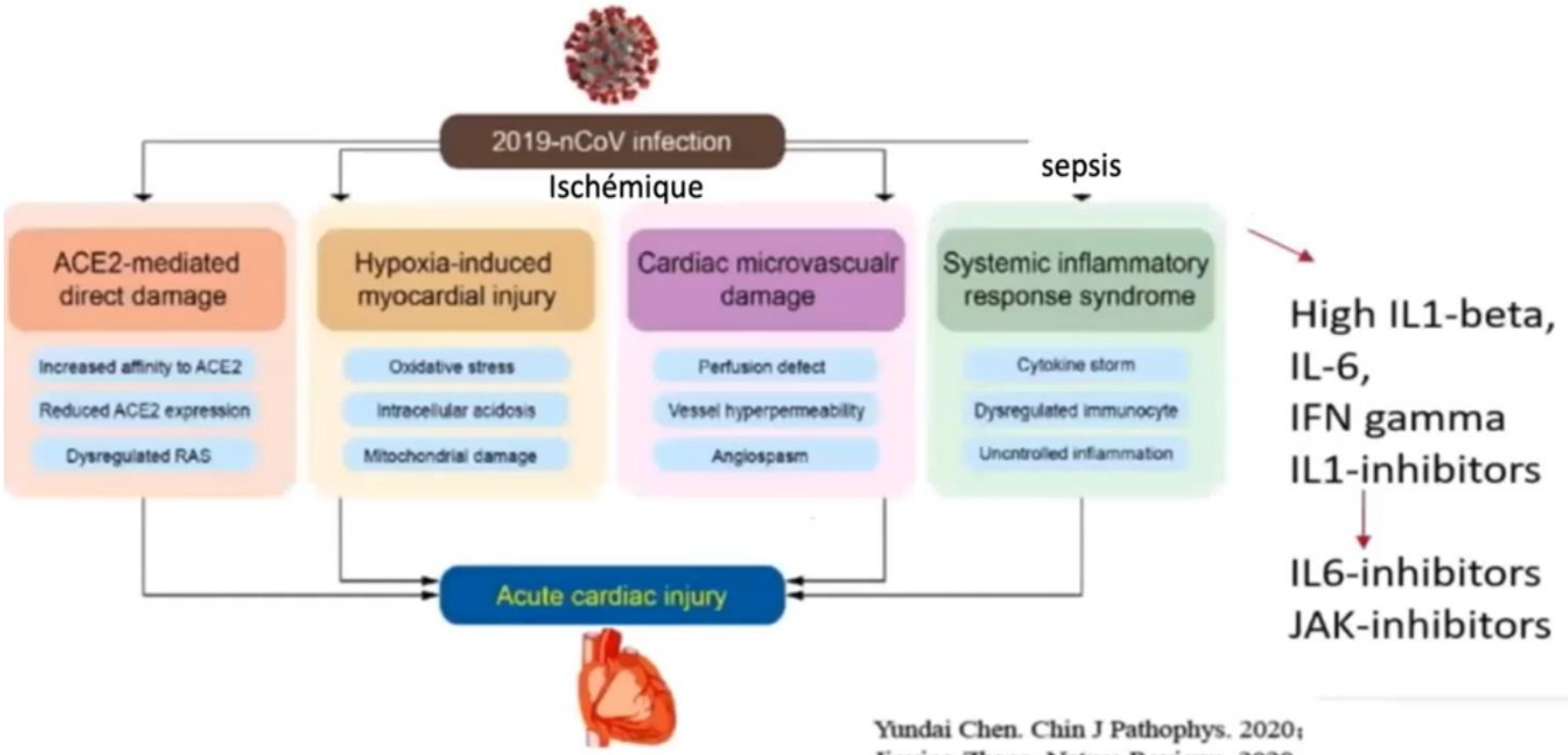
Musher, N Eng J Med 2019

IDM type 2



Thygesen, Eur Heart J 2018

Mécanismes de la lesion cardiaque



Augmentation de la Troponine

Yundai Chen. Chin J Pathophys. 2020;
Jinying Zhang. Nature Reviews. 2020

SCA et Pandémie COVID

3 situations à considérer

1. SCA chez le patient non COVID
2. SCA chez le patient COVID
3. Pseudo-SCA chez le patient COVID

situations artificiellement distinguées car la plupart du temps le statut COVID était inconnu et donc tout patient devait être considéré comme tel…

SCA chez le patient non COVID

Où sont passés les SCA….?

1. Diminution drastique des admissions pour SCA, dont une partie a été vue très tardivement
2. Raisons connues : peur du patient, débordement des centres SAMU, USIC réarmés, mise en œuvre des protections

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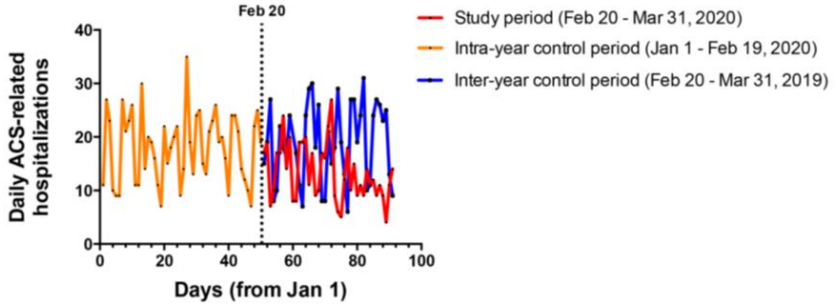
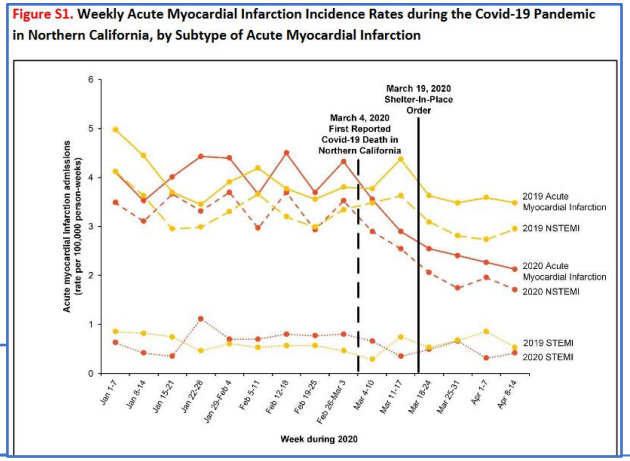
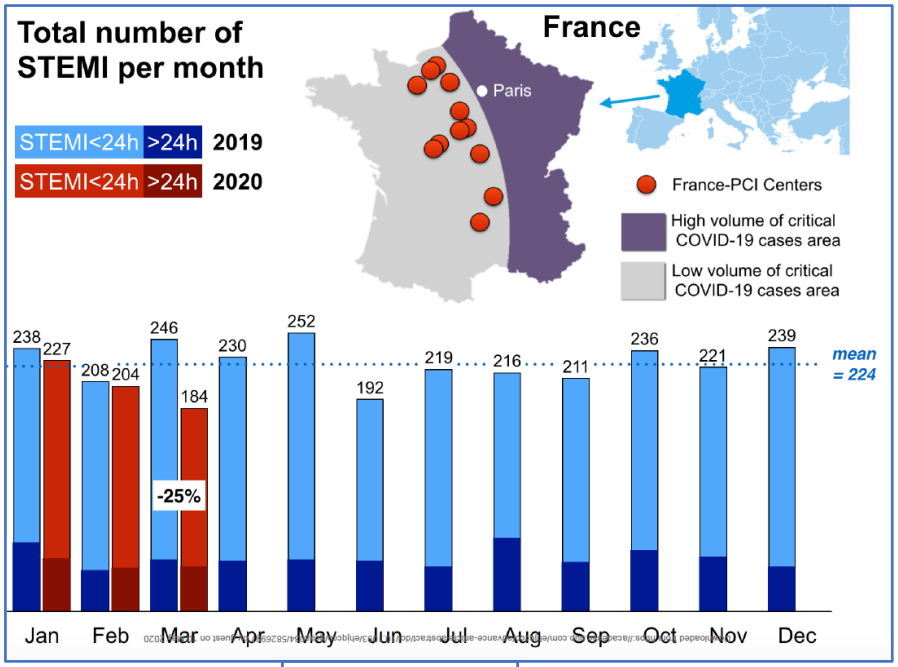


Italie du Nord



Californie du Nord Kayser



France PCI

SCA chez le patient non COVID

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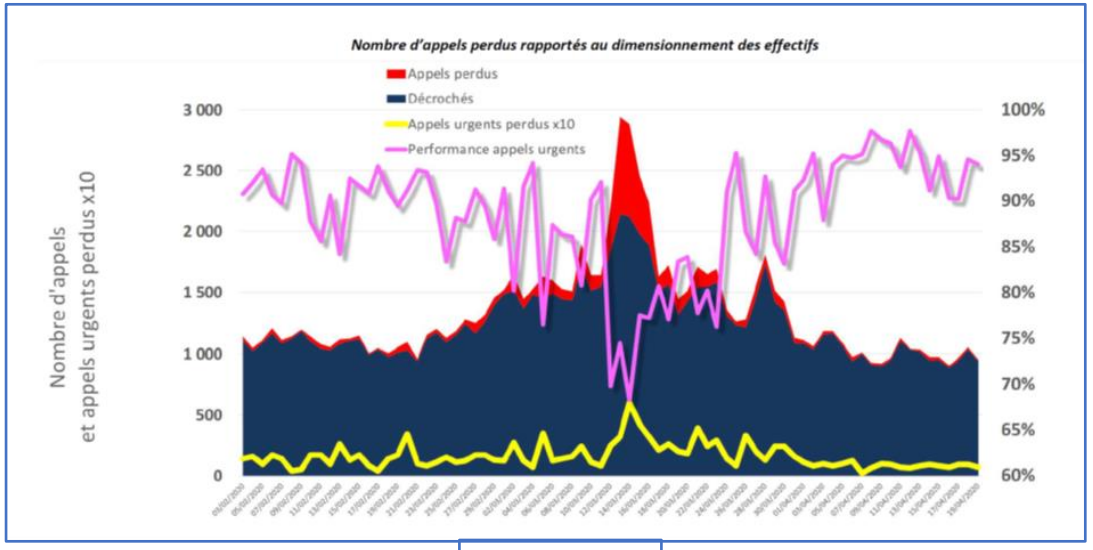
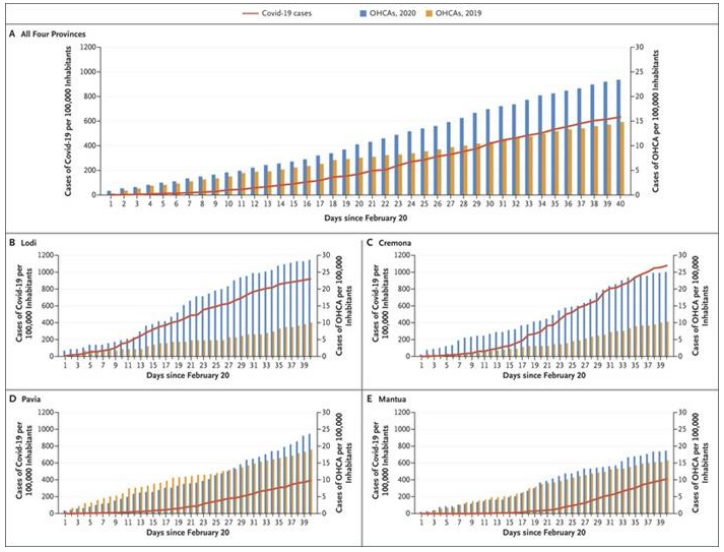
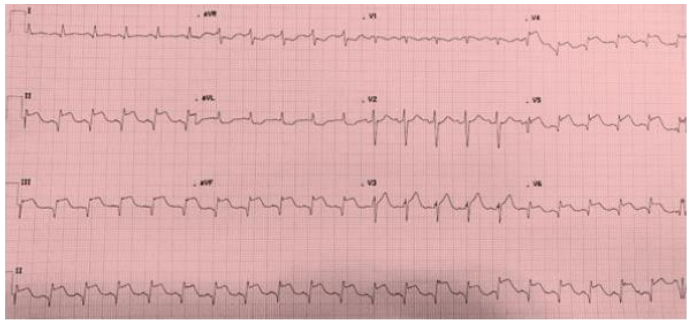
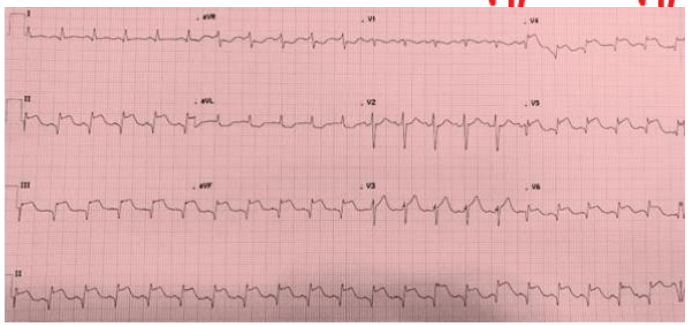
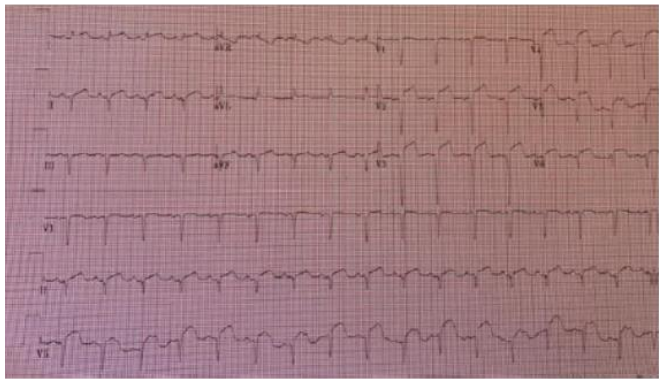
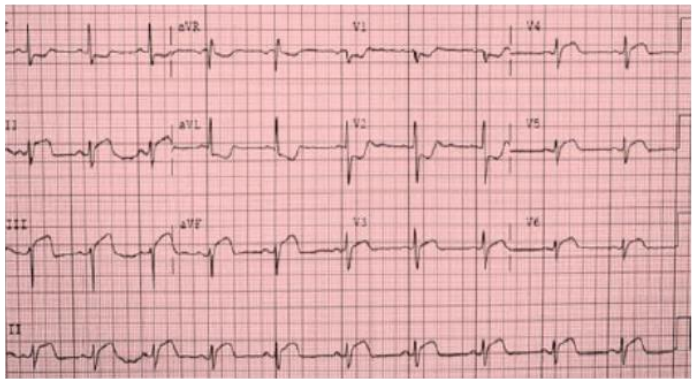
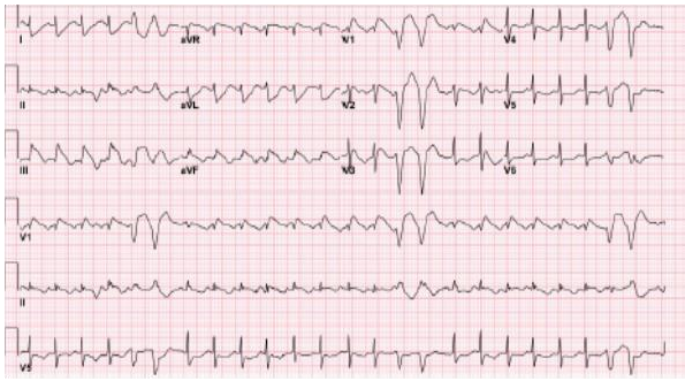
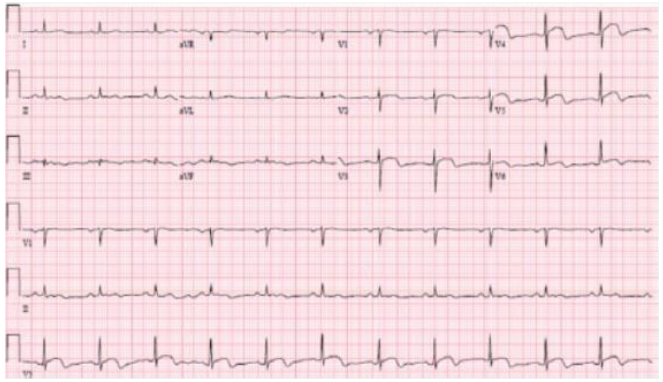
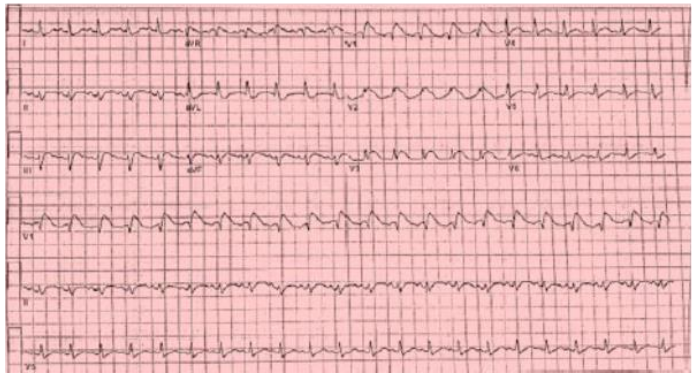


SAMU 83

1. Patients plus graves car hors délais
2. Complications mécaniques (CIV, rupture pariétale, rupture de cordage, choc cardiogénique avec besoin d'assistance..)
3. Augmentation des arrêts cardiaques non hospitaliers



« SCA » chez le patient COVID


Bangalore S, Sharma A, Slotwiner A, et al. ST-segment elevation in patients with Covid-19: a case series. N Engl J Med. DOI: 10.1056/NEJMc2009020

Figure S2A. Timing of ST-segment elevation, length of stay and outcome

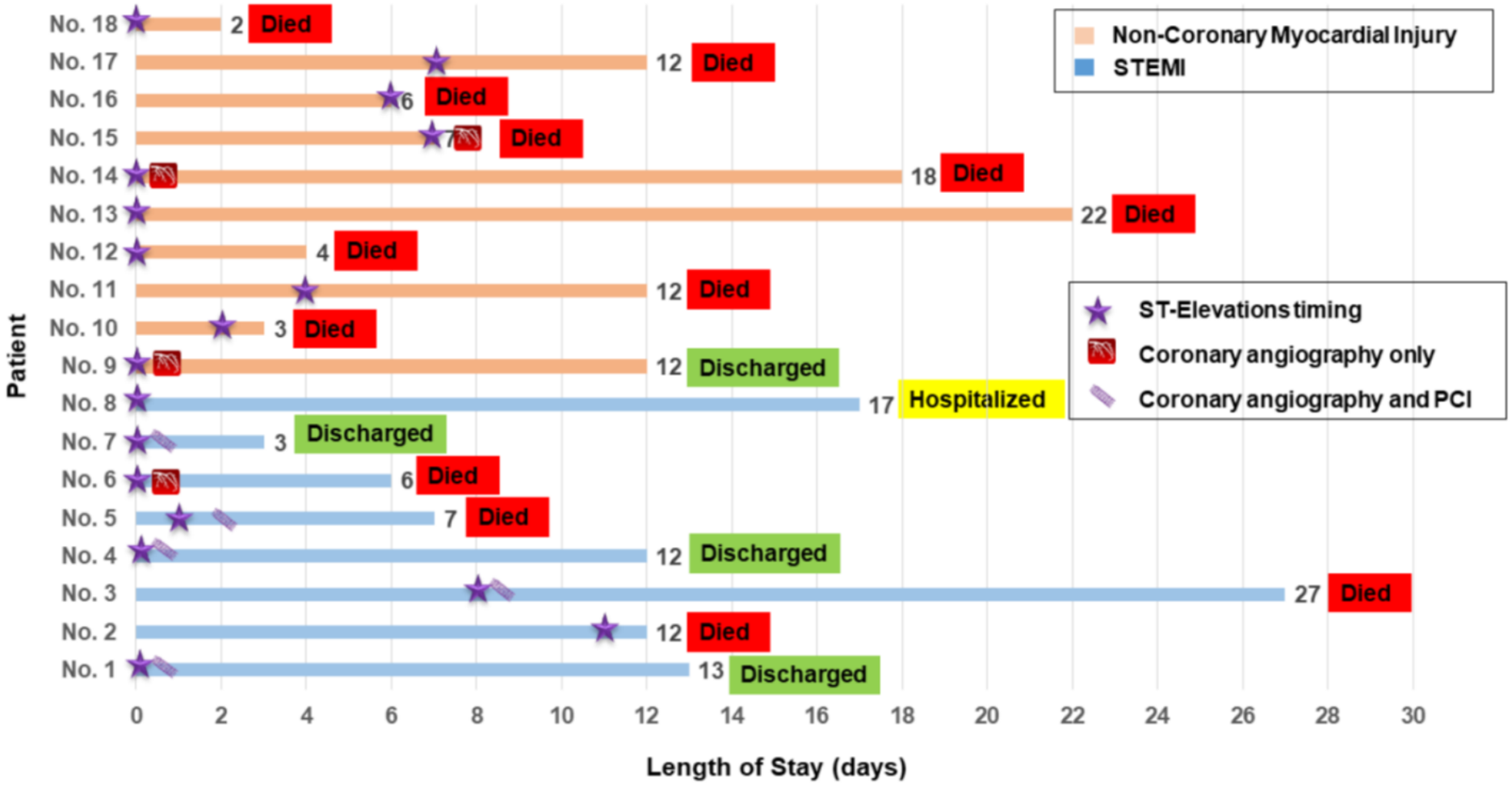
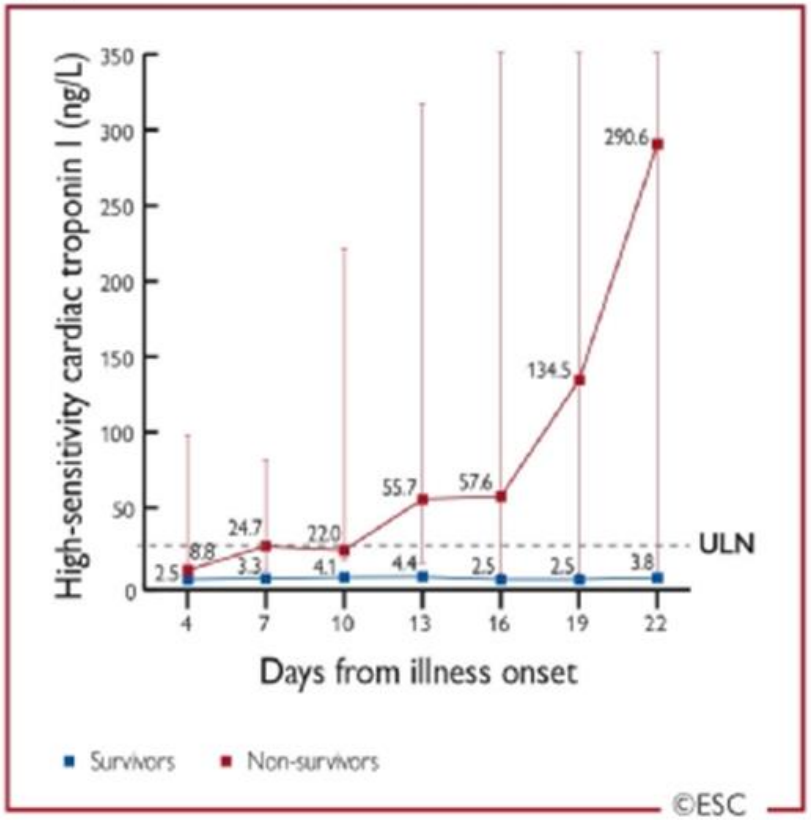
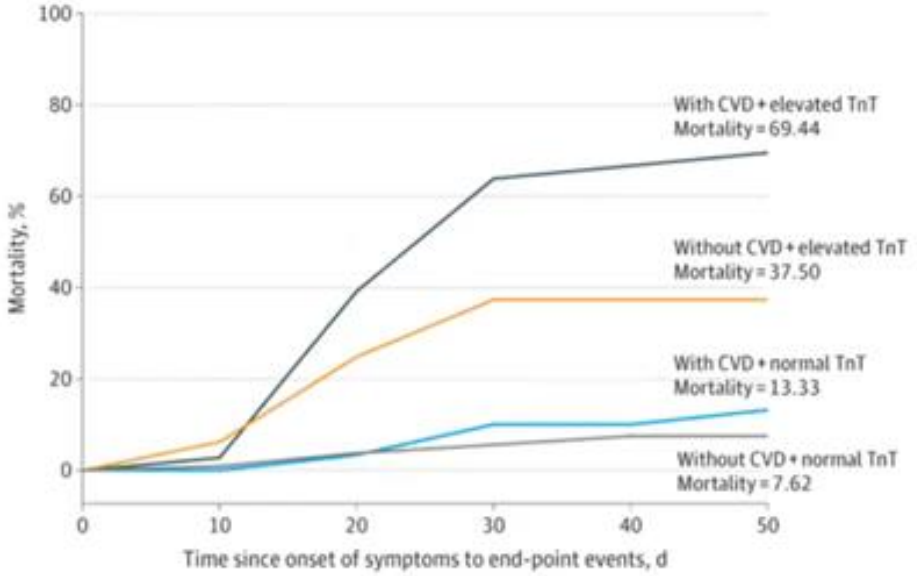


Figure 10 Temporal changes in high-sensitivity cardiac troponin I concentrations from illness onset in patients hospitalised with COVID-19

Differences between survivors and non-survivors were significant for all time points shown. ULN denotes upper limit of normal (adapted from Zhou et al.[23])





No. at risk

Without CVD + normal TnT (n = 105)	102	86	41	10	0
Without CVD + elevated TnT (n = 16)	15	12	7	1	0
With CVD + normal TnT (n = 30)	29	25	10	4	0
With CVD + elevated TnT (n = 36)	34	20	8	2	0

CARDIOVASCULAR FLASHLIGHT

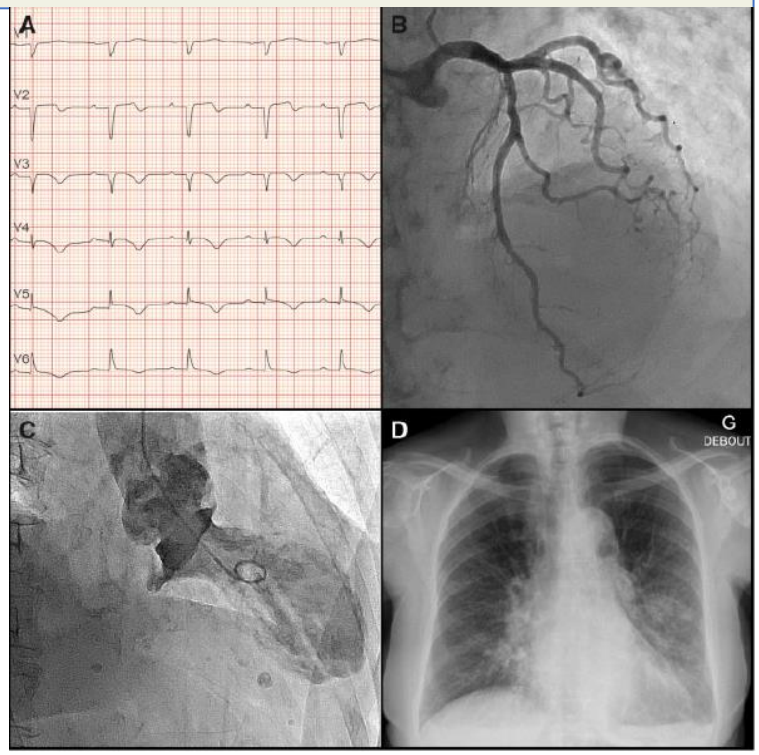
doi:10.1093/eurheartj/ehaa306

Typical takotsubo syndrome triggered by SARS-CoV-2 infection

Philippe Meyer [1]*, Sophie Degrauwe [1], Christian Van Delden[2], Jelena-Rima Ghadri[3], and Christian Templin [3]

[1]Cardiology Service, Geneva University Hospitals, Geneva, Switzerland;; [2]Infectious diseases Service, Geneva University Hospitals, Geneva, Switzerland; and [3]Department of Cardiology, University Hospital of Zurich, Zurich, Switzerland

*Corresponding author. Cardiology Service, Geneva University Hospitals, Rue Gabrielle-Perret-Gentil 4, 1205 Geneva, Switzerland. Tel: +41 22 372 95 97, Fax: +41 22 372 37 45, Email: philippe.meyer@hcuge.ch



Myocardial localization of coronavirus in COVID-19 cardiogenic shock

Guido Tavazzi[1,2], Carlo Pellegrini[1,3], Marco Maurelli[4], Mirko Belliato[2], Fabio Sciutti[2], Andrea Bottazzi[2], Paola Alessandra Sepe[5], Tullia Resasco[5], Rita Camporotondo[6], Raffaele Bruno[1,7], Fausto Baldanti[1,8], Stefania Paolucci[8], Stefano Pelenghi[3], Giorgio Antonio Iotti[1,2], Francesco Mojoli[1,2]*, and Eloisa Arbustini[9]*

[1]Department of Clinical-Surgical, Diagnostic and Pediatric Sciences, University of Pavia, Pavia, Italy; [2]ICU1 Department of Intensive Medicine, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; [3]Cardiac Surgery, Department of Intensive Medicine, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; [4]ICU2, Department of Intensive Medicine, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; [5]Cardiology Unit, Ospedale Maggiore, ASST, Lodi, Italy; [6]Coronary Care Unit and Laboratory of Clinical and Experimental Cardiology, Department of Medical Sciences and Infectious Diseases, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; [7]Infectious Diseases I, Department of Medical Sciences and Infectious Diseases, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; [8]Molecular Virology Unit, Department of Diagnostic Medicine, IRCCS Policlinico San Matteo Foundation, Pavia, Italy; and [9]Transplant Research Area and Centre for Inherited Cardiovascular Diseases, Department of Medical Sciences and Infectious Diseases, IRCCS Policlinico San Matteo Foundation, Pavia, Italy

Received 6 April 2020; revised 7 April 2020; accepted 7 April 2020

We describe the first case of acute cardiac injury directly linked to myocardial localization of severe acute respiratory syndrome coronavirus (SARS-CoV-2) in a 69-year-old patient with flu-like symptoms rapidly degenerating into respiratory distress, hypotension, and cardiogenic shock. The patient was successfully treated with venous-arterial extracorporeal membrane oxygenation (ECMO) and mechanical ventilation. Cardiac function fully recovered in 5 days and ECMO was removed. Endomyocardial biopsy demonstrated low-grade myocardial inflammation and viral particles in the myocardium suggesting either a viraemic phase or, alternatively, infected macrophage migration from the lung.

Keywords SARS-CoV-2 • Coronavirus • Myocardial inflammation • Cardiac injury • Cardiogenic shock • Extracorporeal membrane oxygenation

Vrai SCA chez le patient COVID

1. Rupture de plaque classique en rapport avec une déstabilisation de l'atteinte par l'infection, avec thrombus, traitée classiquement par stenting ..mais nbx cas de thrombose itérative du fait de l'effet procoagulant
2. Thrombose distale souvent difficilement visible (micro-thrombi classiques de le maladie)

Traitement adopté pendant cette période

SCA ST-

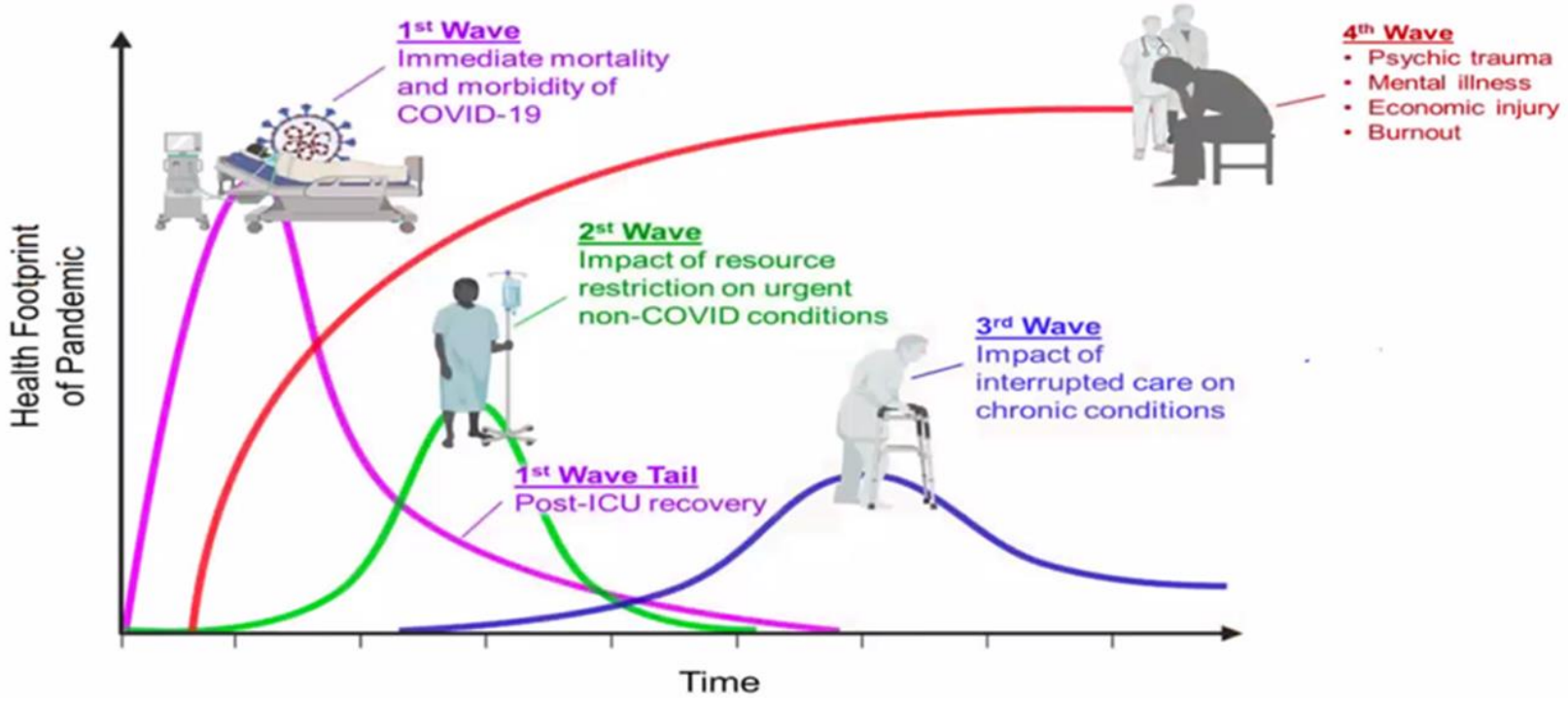
1. Traitement médical de façon prioritaire et sortie la plus précoce possible
2. Chez les patients à haut risque (Grace), stratégie invasive de la lésion coupable

SCA ST+

1. Stratégie invasive de façon prioritaire (Fibrinolyse au cas par cas)
2. Traitement de la lésion coupable et traitement des lésions associées reporté dans une hospitalisation ultérieure

PERSPECTIVES….

The peak , the wave, the tsunami? Which one?



Répliques??

Evolution d'une épidémie émergente avec interventions

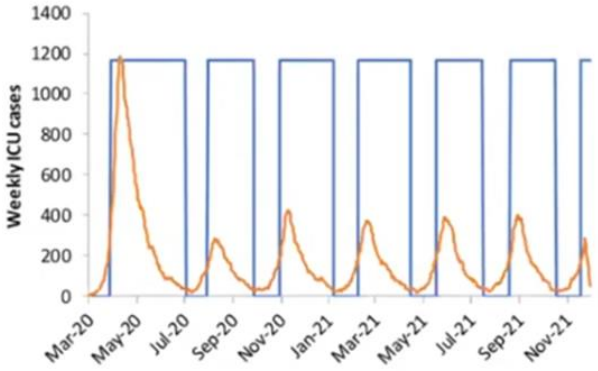
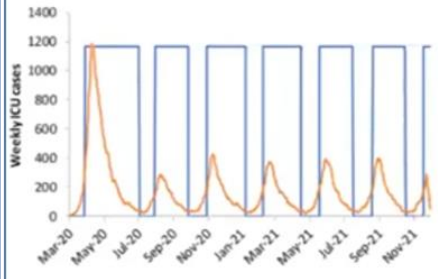


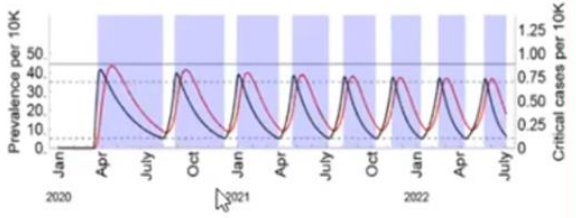
Figure 4: Illustration of adaptive triggering of suppression strategies in GB, for R_0=2.2, a policy of all four interventions considered, an "on" trigger of 100 ICU cases in a week and an "off" trigger of 50 ICU cases. The policy is in force approximate 2/3 of the time. Only social distancing and school/university closure are triggered; other policies remain in force throughout. Weekly ICU incidence is shown in orange, policy triggering in blue.

Ferguson et al., Imperial College, 16 mars 2020

"Stop and go"

- Alternance de confinements et de retours à la "normale".
- Le confinement est remis en place à chaque poussée de l'épidémie, pour limiter la saturation des hôpitaux.
- En attente d'un vaccin ou d'une "immunité de groupe".





Ferguson et al., Imperial College, 16 mars 2020

Kissler et al., Science 2020

Registre rétrospectif STEMI-COVID

CCF-GACI

1. Étude comparative des périodes Mars-Avril-Mai 2019 et 2020
2. Tous les STEMIs de cette période seront analysés
3. Registre national auquel participent plus de 50 centres
4. Implication des jeunes cardiologues en formation sous la direction de Guillaume Bonnet et Guillaume Cayla

Mail : Guillaume.bonnetccf@gmail.com

Conclusions

- La pandémie Covid a confiné non seulement la population mais également les SCA. Les conséquences de ce retard ou de l'absence de revascularisation sont difficiles à apprécier aujourd'hui mais incontestablement on peut craindre une recrudescence de la mortalité à court et moyen terme.
- La physiopathologie complexe de l'atteinte par le SRAS Cov2, encore mal élucidée , risque de nous réserver des surprises (séquelles?)
- Tout cela a entrainé et probablement pour longtemps le mode de prise en charge des patients (circuit, ambulatoire etc..)