

Steering Committee and Investigators. Dual antithrombotic therapy with dabigatran after PCI in atrial fibrillation. *N Engl J Med* 2017;377:1513–1524.

6. Vranckx P, Valgimigli M, Eckardt L, Tijssen J, Lewalter T, Gargiulo G, Batushkin V, Campo G, Lysak Z, Vakaliuk I, Milewski K, Laeis P, Reimitz P-E, Smolnik R, Zierhut W, Goette A. Edoxaban-based versus vitamin K antagonist-based antithrombotic regimen after successful coronary stenting in patients with atrial fibrillation (ENTRUST-AF PCI): a randomised, open-label, phase 3b trial. *Lancet* 2019;394:1335–1343.
7. Gargiulo G, Goette A, Tijssen J, Eckardt L, Lewalter T, Vranckx P, Valgimigli M. Safety and efficacy outcomes of double vs. triple antithrombotic therapy in patients with atrial fibrillation following percutaneous coronary intervention: a systematic review and meta-analysis of non-vitamin K antagonist oral anti-coagulant-based randomized clinical trials. *Eur Heart J* 2019;40:3757–3767.
8. Lopes RD, Hong H, Harskamp RE, Bhatt DL, Mehran R, Cannon CP, Granger CB, Verheugt FWA, Li J, Ten Berg JM, Sarafoff N, Vranckx P, Goette A, Gibson CM, Alexander JH. Optimal antithrombotic regimens for patients with atrial fibrillation undergoing percutaneous coronary intervention: an updated network meta-analysis. *JAMA Cardiol* 2020;5:1–8.

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Declines in Acute Cardiovascular Emergencies During the COVID-19 Pandemic



The coronavirus disease 2019 (COVID-19) pandemic (December 2019 – present) is a global public health crisis with no modern-day equivalent. Several lines of evidence suggest that it is reasonable to forecast a significant increase in cardiovascular (CV) related events and deaths immediately following, and for some time after, the current pandemic.^{1–3} We examined cardiac catheterization laboratory activations at Harford Hospital in Hartford, Connecticut, United States (US) for ST-segment elevation myocardial infarctions (STEMI) and non-ST elevation acute coronary syndromes (NSTEMI) by month for December–April 2020, compared to the monthly average for the previous 4 years (2015–2019). These months were selected to quantify monthly changes in acute cardiac events since first knowledge of COVID-19 (late December 2019). The first case of suspected local transmission in the United States was reported on February 26, 2020 and the first case

in Connecticut was confirmed on March 8, 2020. Local schools were closed March 17, 2020, and executive stay at home orders were put into effect on March 17, 2020.

Compared to historical averages (“expected”), there was a 38% increase in STEMI activations in February 2020, followed by 16% and 21% reductions in March and April, respectively (Figure 1). Compared with expected, there were 21%, 37%, and 80% reductions in NSTEMI-ACS activations for February, March, and April, respectively (Figure 2). Only 3 of 140 patients died February–April 2020; one delayed seeking medical attention for ~24 hours for fear of COVID-19 infection and died from cardiogenic shock. Patients with documented time to presentation (n=24) admitting March–April reported an average duration of 75 ± 196 hours from symptom onset to seeking medical attention. Patients had an average (±SD) age of 63 ± 14 years; 85% were white and 64% were men. Most were overweight (body mass index; mean ± SD: 30 ± 5, range: 21 to 42 kg/m²), current/former smokers (73%), and had chronic conditions (hypercholesterolemia, 63%; hypertension, 56%; diabetes mellitus, 22%). Most (85%) had no previous history of myocardial infarction.

These data, to our knowledge, are the first to confirm similar findings in the

United States⁴ reporting a 38% (95% confidence interval 26 to 49) reduction in STEMI activations from March 1 to March 31, 2020 compared with the preceding 14 months. These findings are consistent with data from Spain (40% reduction in STEMI in March)⁵; Austria (39% reduction in STEMI/NSTEMI-ACS in March)⁶; and poll results from US cardiologists (50% reduction in STEMI/NSTEMI-ACS in February–March).⁷ Our addition of data after March and including April highlights the magnitude and direction of the reduction in acute cardiac event presentation and can likely be extrapolated to a national level.

We observed a 38% increase in STEMI activations for February, which has not been described. Recent reports documenting reductions in post-COVID STEMI activations have evaluated January–February or February–March and have not evaluated February activations separately, and thus reductions in March activations may be greatly underestimated. This increase immediately prior to the epidemic needs verification, but may indicate an increase in events due to psychological stress since a 22% to 35% increase in STEMI/NSTEMI-ACS usually occurs immediately following the onset of a community crisis.^{1–3} It is unclear why there was not a similar increase in NSTEMI-ACS with COVID-19. Future

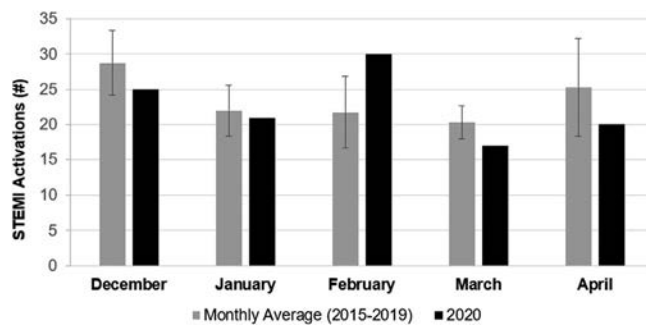


Figure 1. STEMI activations per month before and after COVID-19 compared to historical averages.

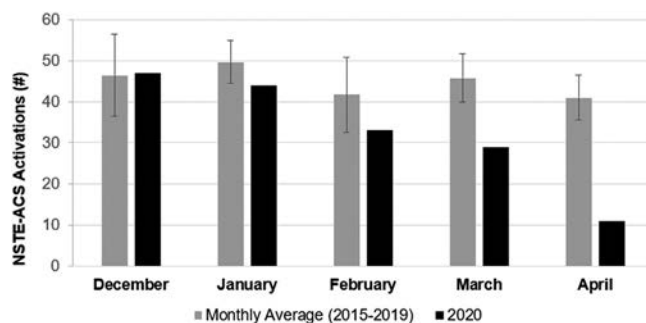


Figure 2. NSTEMI-ACS activations per month before and after COVID-19 compared to historical averages.

longitudinal studies will further elucidate the shape and magnitude of these early trends.

There are many possible explanations for the paradoxical *reduction* in acute CV events with COVID-19. Acute CV complications associated with COVID-19 could have increased morbidity and mortality in the most susceptible populations, a so-called harvesting effect. Out-of-hospital arrests could have increased, which will continue to be vastly underreported due to autopsy delay. Other hypotheses include reductions in behavioral triggers such as smoking and strenuous physical activity; reductions in environmental triggers; or increased volume of stable patients being temporarily medically managed at home. The most likely scenario is the possibility that symptomatic patients are not seeking care, as suggested by our observation that patients waited an average of 3 days after symptoms before presentation. Patient delay may be due to fear of infection, fear of overburdening the healthcare system, and/or loss of financial stability. Such delays are known to increase STEMI/NSTE-ACS complications and deaths. As the US reopens, there may be a surge in the number and severity of cardiac conditions worsened by delay in presentation. National media campaigns⁸ should be expanded to educate and reinforce the importance of recognizing and seeking immediate medical attention for cardiac-related prodromal symptoms so as not to delay timely, lifesaving intervention.

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1. Swerdel JN, Janevic TM, Cosgrove NM, Kostis JB. The effect of Hurricane Sandy on cardiovascular events in New Jersey. *J Am Heart Assoc* 2014;3:e001354.
2. Becquart NA, Naumova EN, Singh G, Chui KKH. Cardiovascular disease hospitalizations

in Louisiana parishes' elderly before, during and after Hurricane Katrina. *Int J Environ Res Public Health* 2018;16. pii: E74.

3. Shedd OL, Sears SF Jr, Harvill JL, Arshad A, Conti JB, Steinberg JS, Curtis AB. The World Trade Center attack: increased frequency of defibrillator shocks for ventricular arrhythmias in patients living remotely from New York City. *J Am Coll Cardiol* 2004;44:1265-1267.
4. Garcia S, Albaghdadi MS, Meraj PM, Schmidt C, Garberich R, Jaffer FA, Dixon S, Rade JJ, Tannenbaum M, Chambers J, Huang PP, Henry TD. Reduction in ST-segment elevation cardiac catheterization laboratory activations in the United States during COVID-19 pandemic. *J Am Coll Cardiol* 2020. pii: S0735-1097(20)34913-5. [Epub ahead of print].
5. Rodríguez-Leora O, Cid-Álvarez B, Ojedae S, Martín-Moreiras S, Rumorosog J, López-Paloh R, Serradori A, Cequier A, Romagueraj R, Cruz I, Pérez de Pradok A, Morenol A. Impacto de la pandemia de COVID-19 sobre la actividad asistencial en cardiología intervencionista en España. *REC Inter-Cardiol* 2020. [Epub ahead of print].
6. Metzler B, Siostrzonek P, Binder RK, Bauer A, Reinstadler SJ. Decline of acute coronary syndrome admissions in Austria since the outbreak of COVID-19: the pandemic response causes cardiac collateral damage. *Eur Heart J* 2020. pii: ehaa314. [Epub ahead of print].
7. Poll Results: COVID-19 and Interventional Cardiology [Internet]. Available from: <https://www.acc.org/latest-in-cardiology/articles/2020/04/27/09/38/poll-results-another-take-on-stemi-during-the-pandemic>.
8. American Heart Association. The new pandemic threat: People may die because they're not calling 911. 2020.

<https://doi.org/10.1016/j.amjcard.2020.05.029>

Racial and Gender Trends in Infective Endocarditis Related Deaths in United States (2004-2017)

Infective endocarditis (IE) is a deadly disease. Etiology is mostly a bacterial infection introduced through infected wounds or through use of Intravenous drugs. Currently, US has an aging population and is in midst of an opioid crisis that has led to increase in IE related deaths. The purpose of this analysis was to see racial, ethnic, and gender trends in deaths where IE was the primary cause of death.

We used the Centers for Disease Control and Prevention Wide-ranging Online Data for Epidemiologic Research database. Data of endocarditis related cause of mortality were extracted from year 2004 to 2017 using International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD

10 codes: I330 and I339). Data included age, ethnicity, race, and place of death. Trends were calculated across time using simple linear regression. Multiple logistic regression was for adjusted analysis between race, ethnicity and gender with other demographic characters. SPSS 26 was used for analyses.

Out of 21,210 records, 42.0% (8,912) were females, 81.3% (14,331) were white and 15.7% (2,772) were black. Over the years (2004 to 2017), deaths reported decreased in females (43.4% to 39.4%) and increased in males (56.6% to 60.6%), proportion of white in reported deaths has increased (80.0% to 83.8%), but the proportion of blacks has decreased (17.0% to 12.9%; [Figure 1](#)).

Among female decedents, 11.6% (1,032) belonged to age group <45 years, 18.8% (1,667) to 45-59 years, 29.9% (2,658) to 60-74 years and 39.7% (3,531) to 75+ years. Among males, 14.7% (1,806) were from <45 years, 26.0% (3,183) from 45 to 59 years, 28.7% (3,515) from 60 to 74 years and 30.6% (3,753) from 75+ years.

Among White decedents, 13.5% (1,927) belonged to age group <45 years, 20.6% (2,951) to 45-59 years, 28.8% (4,114) to 60-74 years and 37.1% (5,305) to 75 years or above. White decedents were less likely to be females (OR, 0.86 [0.78 to 0.95], $p < 0.01$) and were more likely to die at home (OR, 1.92 [1.54 to 2.40], $p < 0.01$). Among black decedents, 12.8% (1,790) belonged to age group <45 years, 35.4% (975) to 45-59 years, 32.6% (897) to 60-74 years and 19.2% (529) to 75 years or above. Black decedents were less likely to females (OR, 0.60 [0.59 to 0.74], $p < 0.01$), were less likely at home as compared with hospital (OR, 0.52 [0.40 to 0.66], $p < 0.01$).

IE incidence overall has been on an increase because of aging population and opioid epidemic.^{1,2} We found that the proportion of males and whites have been increasing over the years, possibly pointing towards the death related to opioid epidemic that is primarily affecting white males. This epidemic has led to 18 fold increase in overall deaths along with decrease in average age expectancy in white Americans.¹ Previous studies from other living databases have pointed towards similar demographic trends as well.³ We also noticed that whites were more likely to belong to the young (<45) and old age group (>75) in comparison to

