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Covid and the Athlete's Heart: Lessons Learned

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MGH 1811 Cardiovascular Performance Program

Financial Disclosures

Affiliations:

Funding Sources:

- National Institutes of Health
- American Heart Association
- American Society of Echocardiography
- Department of Defense
- National Football League Player's Association
- American Medical Society for Sports Medicine

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COVID-19 Cardiac

Cardiovascular Performance Program

MGH 1811

Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China

Chaolin Huang*, Yeming Wang*, Xingwang Li*, Lili Ren*, Jianping Zhao*, Yi Hu*, Li Zhang, Guohui Fan, Jiuyang Xu, Xiaoying Gu, Zhenshun Cheng, Ting Yu, Jiaan Xia, Yuan Wei, Wenjuan Wu, Xuelei Xie, Wen Yin, Hui Li, Min Liu, Yan Xiao, Hong Gao, Li Guo, Jungang Xie, Guangfa Wang, Rongmeng Jiang, Zhancheng Gao, Qi Jin, Jianwei Wang†, Bin Cao†

Findings By Jan 2, 2020, 41 admitted hospital patients had been identified as having laboratory-confirmed 2019-nCoV infection. Most of the infected patients were men (30 [73%] of 41); less than half had underlying diseases (13 [32%]), including diabetes (eight [20%]), hypertension (six [15%]), and cardiovascular disease (six [15%]). Median age was 49·0 years (IQR 41·0–58·0). 27 (66%) of 41 patients had been exposed to Huanan seafood market. One family cluster was found. Common symptoms at onset of illness were fever (40 [98%] of 41 patients), cough (31 [76%]), and myalgia or fatigue (18 [44%]); less common symptoms were sputum production (11 [28%] of 39), headache (three [8%] of 38), haemoptysis (two [5%] of 39), and diarrhoea (one [3%] of 38). Dyspnoea developed in 22 (55%) of 40 patients (median time from illness onset to dyspnoea 8·0 days [IQR 5·0–13·0]). 26 (63%) of 41 patients had lymphopenia. All 41 patients had pneumonia with abnormal findings on chest CT. Complications included acute respiratory distress syndrome (12 [29%]), RNAaemia (six [15%]), acute cardiac injury (five [12%]) and secondary infection (four [10%]). 13 (32%) patients were admitted to an ICU and six (15%) died. Compared with non-ICU patients, ICU patients had higher plasma levels of IL2, IL7, IL10, GSCF, IP10, MCP1, MIP1A, and TNFα.

of IL2, IL7, IL10, GSCF, IP10, MCP1, MIP1A, and TNFα.

Interpretation The 2019-nCoV infection caused clusters of severe respiratory illness similar to severe acute respiratory syndrome coronavirus and was associated with ICU admission and high mortality. Major gaps in our knowledge of the origin, epidemiology, duration of human transmission, and clinical spectrum of disease need fulfilment by future studies.

Funding Ministry of Science and Technology, Chinese Academy of Medical Sciences, National Natural Science Foundation of China, and Beijing Municipal Science and Technology Commission.

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Introduction

Coronaviruses are enveloped non-segmented positive-sense RNA viruses belonging to the family Coronaviridae and the order Nidovirales and broadly distributed in humans and other mammals.[1] Although most human coronavirus infections are mild, the epidemics of the two betacoronaviruses, severe acute respiratory syndrome coronavirus (SARS-CoV)[2-4] and Middle East respiratory syndrome coronavirus (MERS-CoV),[5,6] have caused more than 10 000 cumulative cases in the past two decades, with mortality rates of 10% for SARS-CoV and 37% for MERS-CoV.[7,8] The coronaviruses already identified might only be the tip of the iceberg, with potentially more novel and severe zoonotic events to be revealed.

In December, 2019, a series of pneumonia cases of unknown cause emerged in Wuhan, Hubei, China, with clinical presentations greatly resembling viral pneumonia.[9] Deep sequencing analysis from lower respiratory tract samples indicated a novel coronavirus, which was named 2019 novel coronavirus (2019-nCoV). Thus far, more than 800 confirmed cases, including in health-care workers, have been identified in Wuhan, and several exported cases have been confirmed in other provinces in China, and in Thailand, Japan, South Korea, and the USA.[10-13]

Sciences (G Fan, X Gu), and Department of Radiology (M Liu MD), China-Japan Friendship Hospital, Beijing, China; Institute of Respiratory Medicine, Chinese Academy of Medical Sciences, Peking Union Medical College, Beijing, China (Y Wang, G Fan, X Gu, H Li, Prof B Cao); Department of Respiratory Medicine, Capital Medical University, Beijing, China (Y Wang, H Li, Prof B Cao); Clinical and Research Center of Infectious Diseases, Beijing Ditan Hospital, Capital Medical University, Beijing, China (Prof X Li MD, Prof R Jiang MD); NHC Key Laboratory of Systems Biology of Pathogens and Christophe Merieux Laboratory, Institute of Pathogen Biology (Prof L Ren PhD, Y Xiao MS, Prof L Guo PhD, Q Jin PhD, Prof J Wang PhD), and Institute of Laboratory Animal Science (Prof H Gao PhD), Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China; Tongji Hospital (Prof J Zhao MD, Prof J Xie MD), and Department

	All patients (n=41)	ICU care (n=13)	No ICU care (n=28)	p value
Duration from illness onset to first admission	7·0 (4·0–8·0)	7·0 (4·0–8·0)	7·0 (4·0–8·5)	0·87
Complications				
Acute respiratory distress syndrome	12 (29%)	11 (85%)	1 (4%)	<0·0001
RNAaemia	6 (15%)	2 (15%)	4 (14%)	0·93
Cycle threshold of RNAaemia	35·1 (34·7–35·1)	35·1 (35·1–35·1)	34·8 (34·1–35·4)	0·35
Acute cardiac injury*	5 (12%)	4 (31%)	1 (4%)	0·017
Acute kidney injury	3 (7%)	3 (23%)	0	0·027
Secondary infection	4 (10%)	4 (31%)	0	0·0014
Shock	3 (7%)	3 (23%)	0	0·027

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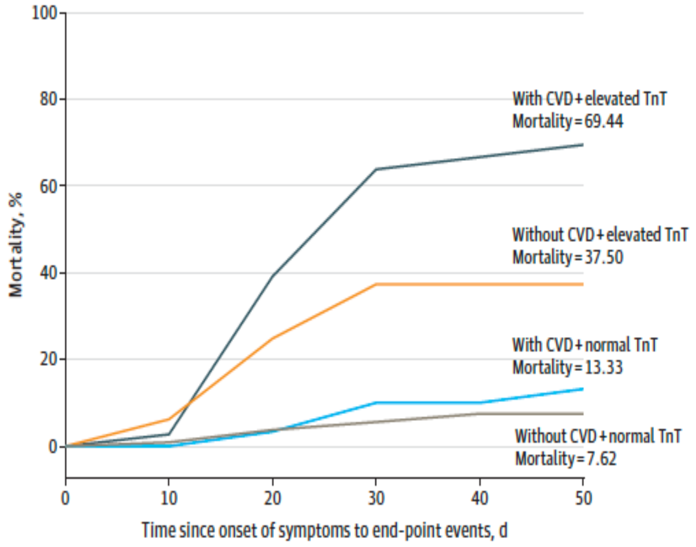
JAMA Cardiology | Original Investigation

Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19)

Tao Guo, MD; Yongzhen Fan, MD; Ming Chen, MD; Xiaoyan Wu, MD; Lin Zhang, MD; Tao He, MD; Hairong Wang, MD; Jing Wan, MD; Xinghuan Wang, MD; Zhibing Lu, MD

RESULTS Among 187 patients with confirmed COVID-19, 144 patients (77%) were discharged and 43 patients (23%) died. The mean (SD) age was 58.50 (14.66) years. Overall, 66 (35.3%) had underlying CVD including hypertension, coronary heart disease, and cardiomyopathy, and 52 (27.8%) exhibited myocardial injury as indicated by elevated TnT levels. The mortality during hospitalization was 7.62% (8 of 105) for patients without underlying CVD and normal TnT levels, 13.33% (4 of 30) for those with underlying CVD and normal TnT levels, 37.50% (6 of 16) for those without underlying CVD but elevated TnT levels, and 69.44% (25 of 36) for those with underlying CVD and elevated TnTs. Patients with underlying CVD were more likely to exhibit elevation of TnT levels compared with the patients without CVD (36 [54.5%] vs 16 [13.2%]). Plasma TnT levels demonstrated a high and significantly positive linear correlation with plasma high-sensitivity C-reactive protein levels (β = 0.530, *P* < .001) and N-terminal pro–brain natriuretic peptide (NT-proBNP) levels (β = 0.613, *P* < .001). Plasma TnT and NT-proBNP levels during hospitalization (median [interquartile range (IQR)], 0.307 [0.094-0.600]; 1902.00 [728.35-8100.00]) and impending death (median [IQR], 0.141 [0.058-0.860]; 5375 [1179.50-25695.25]) increased significantly compared with admission values (median [IQR], 0.0355 [0.015-0.102]; 796.90 [401.93-1742.25]) in patients who died (*P* = .001; *P* < .001), while no significant dynamic changes of TnT (median [IQR], 0.010 [0.007-0.019]; 0.013 [0.007-0.022]; 0.011 [0.007-0.016]) and NT-proBNP (median [IQR], 352.20 [174.70-636.70]; 433.80 [155.80-1272.60]; 145.40 [63.4-526.50]) was observed in survivors (*P* = .96; *P* = .16). During hospitalization, patients with elevated TnT levels had more frequent malignant arrhythmias, and the use of glucocorticoid therapy (37 [71.2%] vs 69 [51.1%]) and mechanical ventilation (31 [59.6%] vs 14 [10.4%]) were higher compared with patients with normal TnT levels. The mortality rates of patients with and without use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers was 36.8% (7 of 19) and 21.4% (36 of 168) (*P* = .13).

Figure 2. Mortality of Patients With Coronavirus Disease 2019 (COVID-19) With/Without Cardiovascular Disease (CVD) and With/Without Elevated Troponin T (TnT) Levels



No. at risk	0	10	20	30	40	50
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

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THE WALL STREET JOURNAL.

WORLD

The Soccer Match that Kicked Off Italy's Coronavirus Disaster

Decision to hold Atalanta-Valencia Champions League match in February accelerated spread of pandemic

February 19th, Milan ITALY

Atalanta vs. Valencia Champions League Match

45,732 Fans
(~40K Bergamaschi)

4 to 1 Victory for Italy

35% of team & staff infected within 7 days

1 month Bergamo death toll 10k, base pop. of 120K

Valencia becomes Spanish Epicenter by early March

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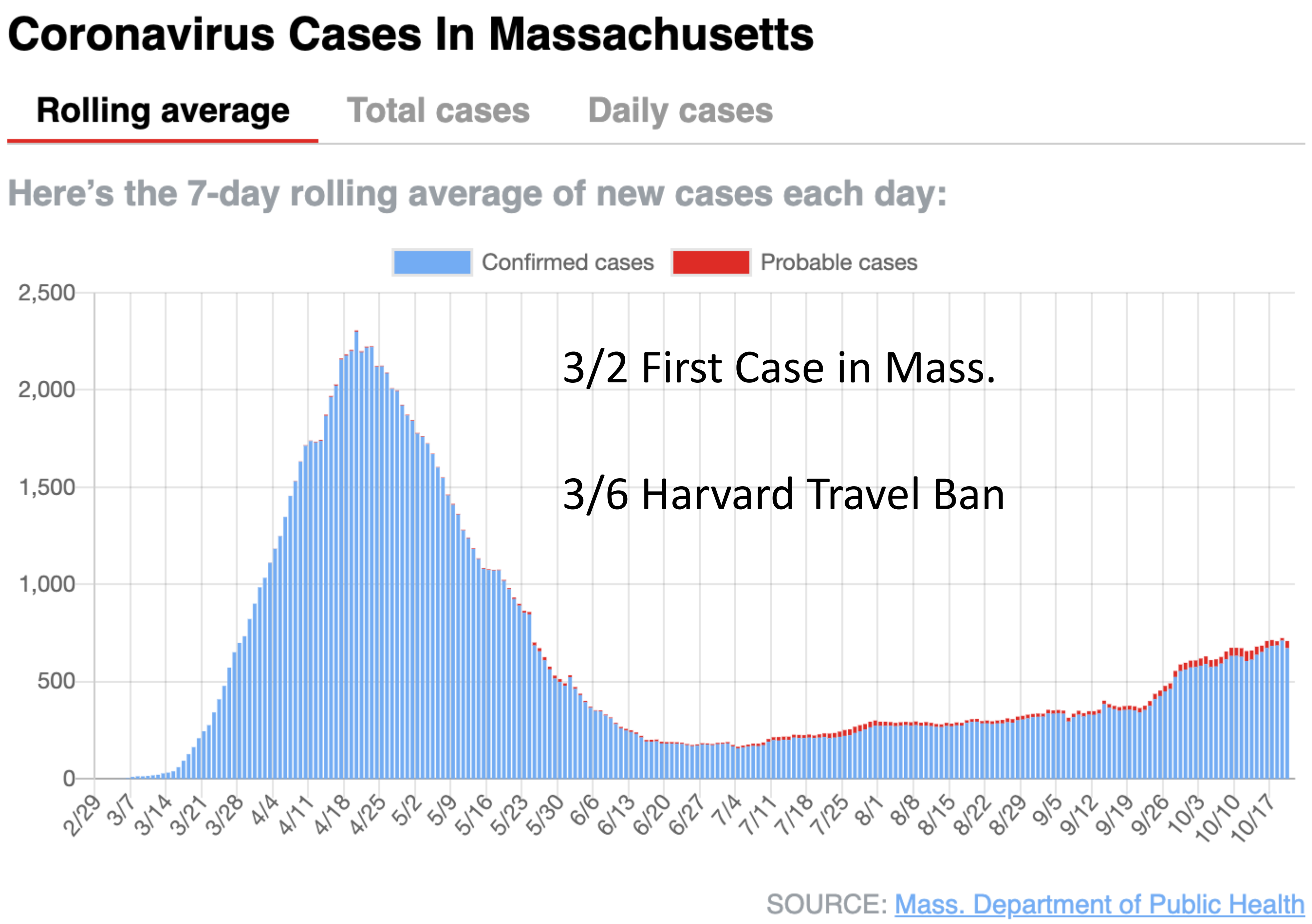

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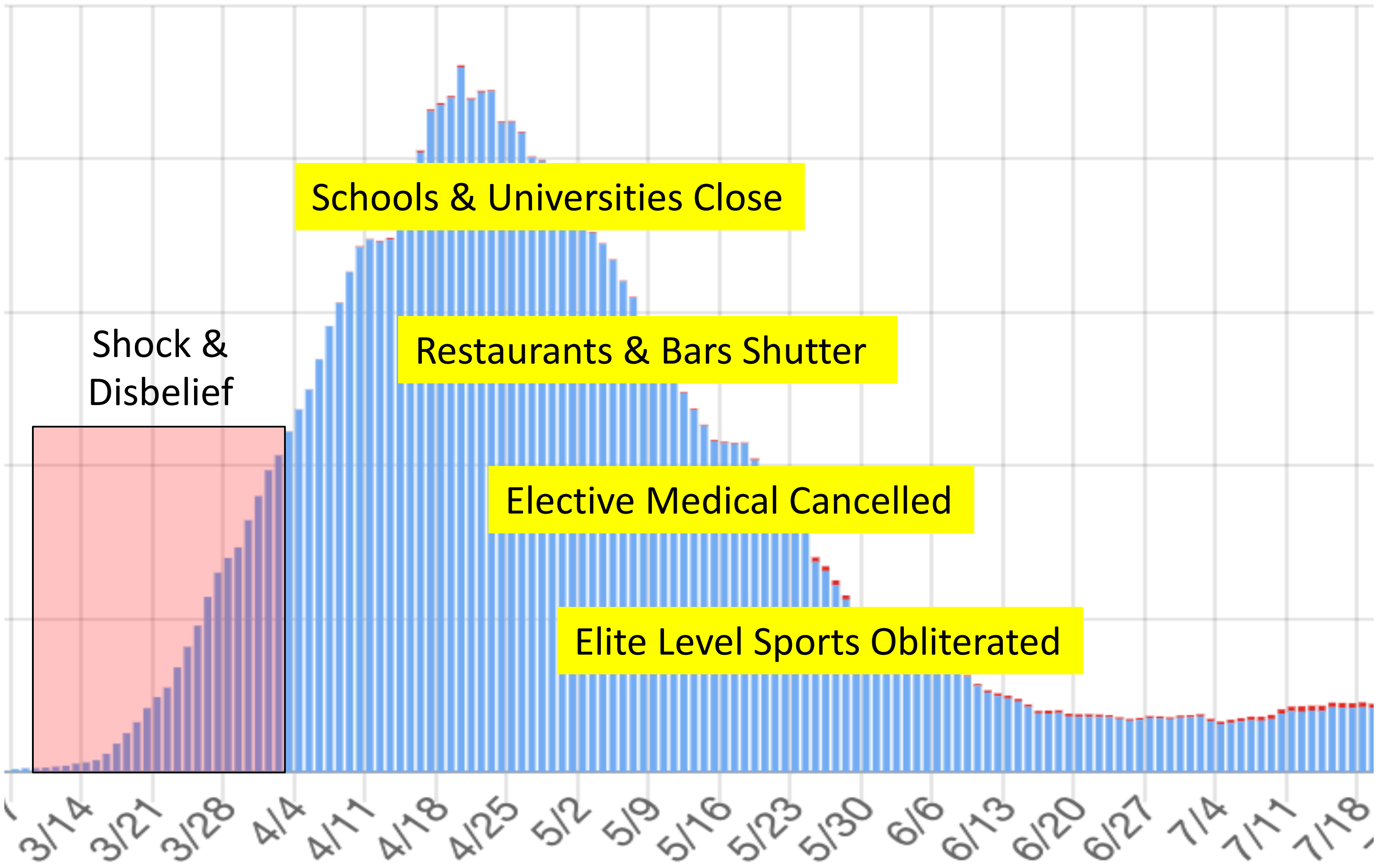

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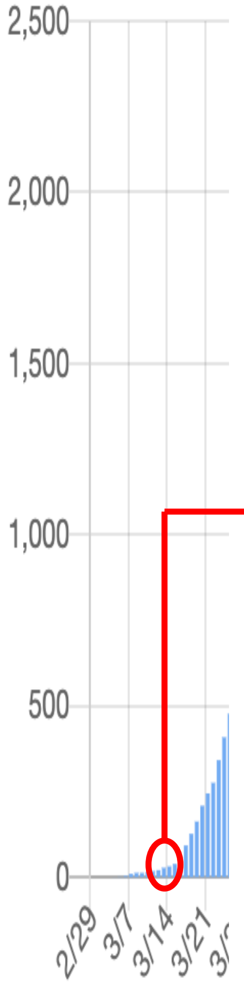
The 5 Day Demise of Elite US Sport



3/11: Rudy Gobert (Utah Jazz) COVID+ >> NBA Suspends Season

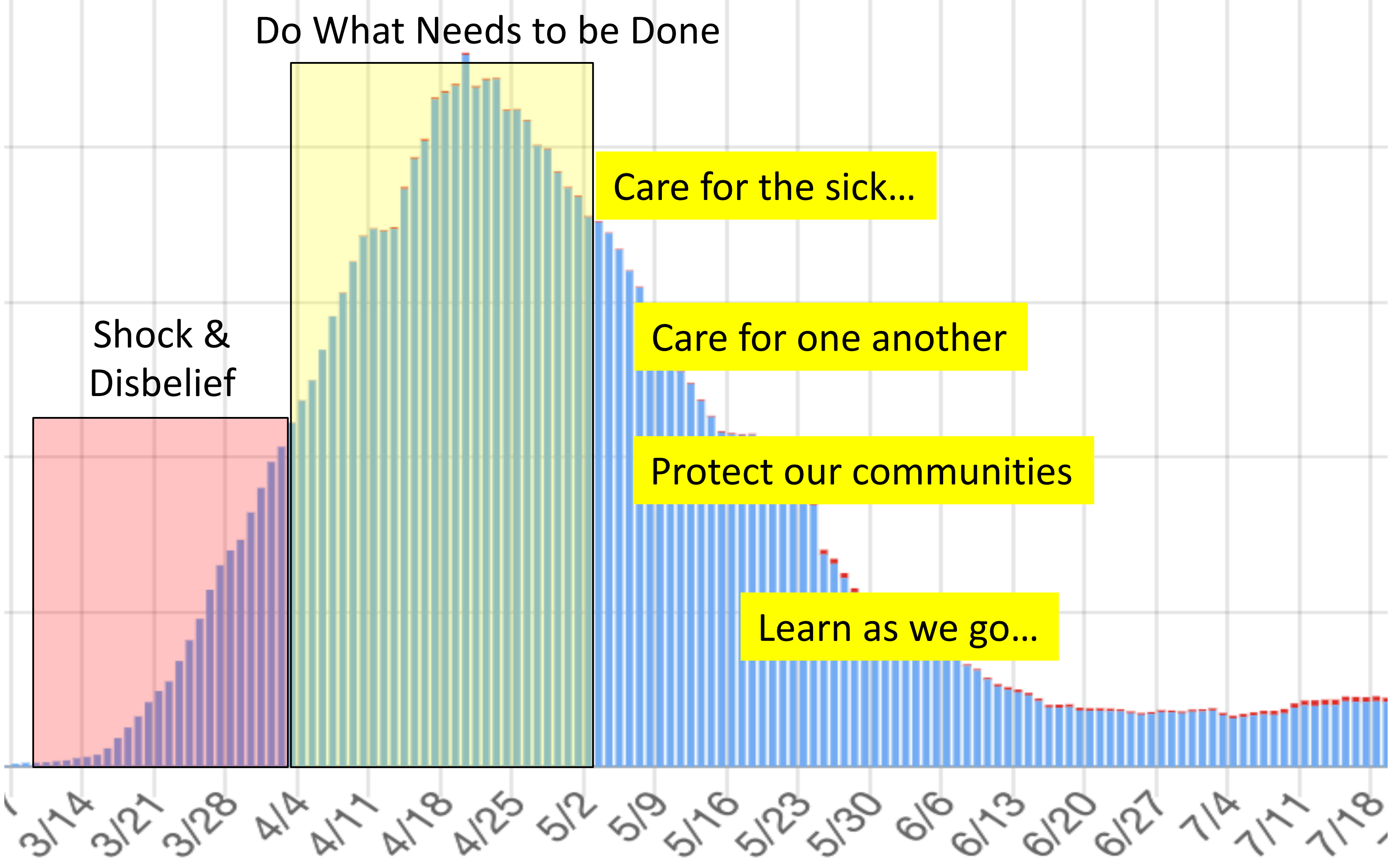
3/12: Major League Soccer (MLS) suspends season
NCAA Basketball Cancelled (ACC, SEC, Big 12, Big 12, etc.)
Big East (Creighton- St John's at Halftime)
NHL Suspends season
MLB cancels spring training

3/13: Boston Marathon cancelled
Master's Golf and all PGA events cancelled
NASCAR cancels series of major races

3/14: Nevada gaming commission suspends all contact sport
Christian Woods (Detroit Pistons) COVID+ after game with Jazz

3/15: CDC orders cancellation events > 50 people for 8 weeks….

COVID-19 Cardiac



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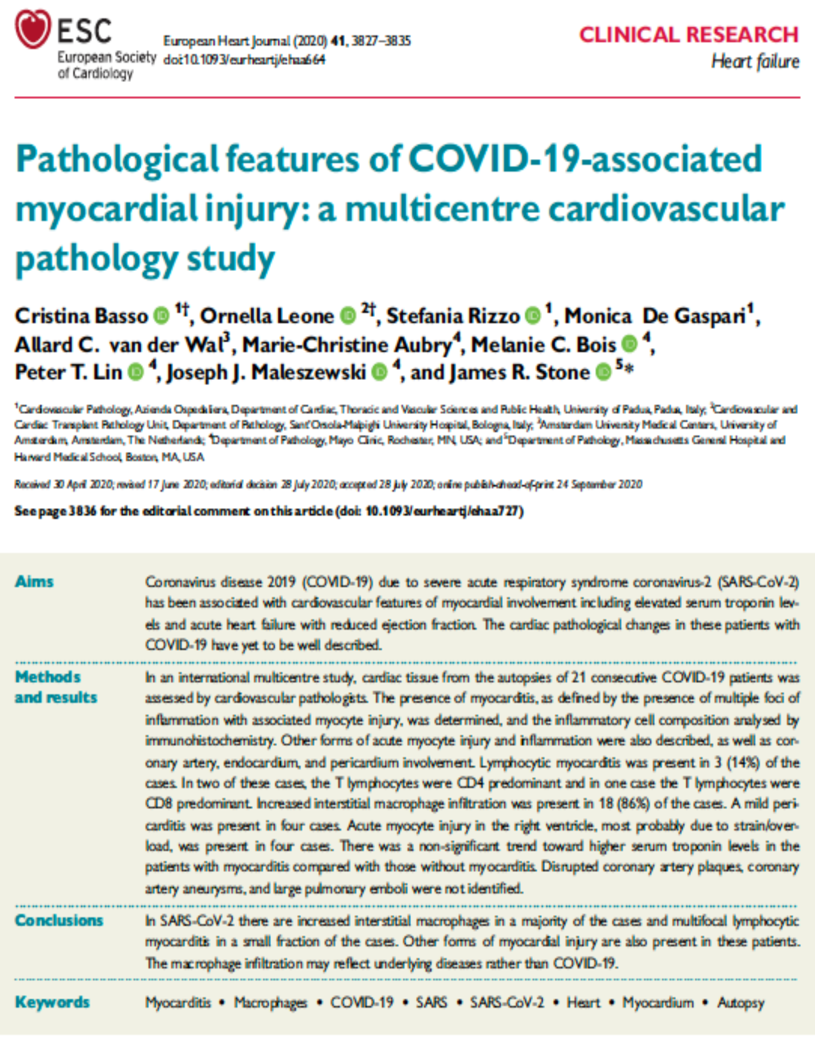
Hemodynamic injury

Cytokine-mediated injury

Myocardial viral invasion

Underlying CV disease



ESC European Society of Cardiology

European Heart Journal (2020) 41, 3827–3835
doi:10.1093/eurheartj/ehaa664

CLINICAL RESEARCH
Heart failure

Pathological features of COVID-19-associated myocardial injury: a multicentre cardiovascular pathology study

Cristina Basso [1†], Ornella Leone [2†], Stefania Rizzo [1], Monica De Gaspari[1], Allard C. van der Wal[3], Marie-Christine Aubry[4], Melanie C. Bois [4], Peter T. Lin [4], Joseph J. Maleszewski [4], and James R. Stone [5*]

[1]Cardiovascular Pathology, Azienda Ospedaliera, Department of Cardiac, Thoracic and Vascular Sciences and Public Health, University of Padua, Padua, Italy; [2]Cardiovascular and Cardiac Transplant Pathology Unit, Department of Pathology, Sant'Orsola-Malpighi University Hospital, Bologna, Italy; [3]Amsterdam University Medical Centers, University of Amsterdam, Amsterdam, The Netherlands; [4]Department of Pathology, Mayo Clinic, Rochester, MN, USA; and [5]Department of Pathology, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA

Received 30 April 2020; revised 17 June 2020; editorial decision 28 July 2020; accepted 28 July 2020; online publish-ahead-of-print 24 September 2020

See page 3836 for the editorial comment on this article (doi: 10.1093/eurheartj/ehaa727)

Aims Coronavirus disease 2019 (COVID-19) due to severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) has been associated with cardiovascular features of myocardial involvement including elevated serum troponin levels and acute heart failure with reduced ejection fraction. The cardiac pathological changes in these patients with COVID-19 have yet to be well described.

Methods and results In an international multicentre study, cardiac tissue from the autopsies of 21 consecutive COVID-19 patients was assessed by cardiovascular pathologists. The presence of myocarditis, as defined by the presence of multiple foci of inflammation with associated myocyte injury, was determined, and the inflammatory cell composition analysed by immunohistochemistry. Other forms of acute myocyte injury and inflammation were also described, as well as coronary artery, endocardium, and pericardium involvement. Lymphocytic myocarditis was present in 3 (14%) of the cases. In two of these cases, the T lymphocytes were CD4 predominant and in one case the T lymphocytes were CD8 predominant. Increased interstitial macrophage infiltration was present in 18 (86%) of the cases. A mild pericarditis was present in four cases. Acute myocyte injury in the right ventricle, most probably due to strain/overload, was present in four cases. There was a non-significant trend toward higher serum troponin levels in the patients with myocarditis compared with those without myocarditis. Disrupted coronary artery plaques, coronary artery aneurysms, and large pulmonary emboli were not identified.

Conclusions In SARS-CoV-2 there are increased interstitial macrophages in a majority of the cases and multifocal lymphocytic myocarditis in a small fraction of the cases. Other forms of myocardial injury are also present in these patients. The macrophage infiltration may reflect underlying diseases rather than COVID-19.

Keywords Myocarditis • Macrophages • COVID-19 • SARS • SARS-CoV-2 • Heart • Myocardium • Autopsy

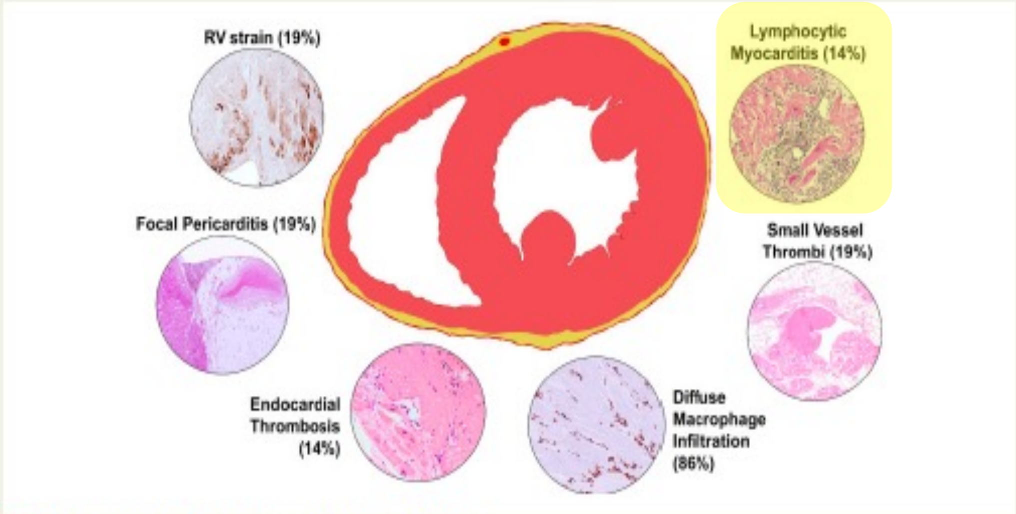


Figure 7 Cardiac pathological changes associated with COVID-19.

COVID-19 Cardiac

Heart Rhythm Disorders

Sudden Death in Young Adults

An Autopsy-Based Series of a Population Undergoing Active Surveillance

Robert E. Eckart, DO,* Eric A. Shry, MD,† Allen P. Burke, MD,‡ Jennifer A. McNear, MD,* David A. Appel, MD,* Laudino M. Castillo-Rojas, MD,* Lena Avedissian, MD,§ Lisa A. Pearse, MD,‡ Robert N. Potter, MPH,‡ Ladd Tremaine, MD,‡ Philip J. Gentlesk, MD,* Linda Huffer, MD,§ Stephen S. Reich, MD,* William G. Stevenson, MD,‖ for the Department of Defense Cardiovascular Death Registry Group

San Antonio, Texas; Landstuhl, Germany; Washington, DC; and Boston, Massachusetts

Objectives The purpose of this study was to define the incidence and characterization of cardiovascular cause of sudden death in the young.

Background The epidemiology of sudden cardiac death (SCD) in young adults is based on small studies and uncontrolled observations. Identifying causes of sudden death in this population is important for guiding approaches to prevention.

Methods We performed a retrospective cohort study using demographic and autopsy data from the Department of Defense Cardiovascular Death Registry over a 10-year period comprising 15.2 million person-years of active surveillance.

Results We reviewed all nontraumatic sudden deaths in persons 18 years of age and over. We identified 902 subjects in whom the adjudicated cause of death was of potential cardiac etiology, with a mean age of 38 ± 11 years. The mortality rate for SCD per 100,000 person-years for the study period was 6.7 for males and 1.4 for females ($p < 0.0001$). Sudden death was attributed to a cardiac condition in 715 (79.3%) and was unexplained in 187 (20.7%). The incidence of sudden unexplained death (SUD) was 1.2 per 100,000 person-years for persons <35 years of age, and 2.0 per 100,000 person-years for those ≥35 years of age ($p < 0.001$). The incidence of fatal atherosclerotic coronary artery disease was 0.7 per 100,000 person-years for those <35 years of age, and 13.7 per 100,000 person-years for those ≥35 years of age ($p < 0.001$).

Conclusions Prevention of sudden death in the young adult should focus on evaluation for causes known to be associated with SUD (e.g., primary arrhythmia) among persons <35 years of age, with an emphasis on atherosclerotic coronary disease in those ≥35 years of age. (J Am Coll Cardiol 2011;58:1254–61) © 2011 by the American College of Cardiology Foundation

Sudden death of the healthy young adult is uncommon, but receives substantial attention from the media and raises issues of accountability for screening programs (1). The relative importance of different etiologies of sudden death varies among studies. Among cohorts collected using passive surveillance (e.g., newspaper accounts and Internet queries), hypertrophic cardiomyopathy was the most commonly identified abnormality in sudden death of young adults and young athletes (2–4). Passive surveillance methods are, however, subject to ascertainment and referral bias. Studies utilizing active surveillance to collect all deaths in a defined population, found by administrative diagnostic coding or death certificate review, have found either no identifiable structural abnormality or coronary artery disease (CAD) in the majority of cases of sudden death (5–11).

Despite advances in defining the causes of sudden death and dramatic developments in the ability to screen for genetic diseases and premature atherosclerosis (12–24), recommendations for screening the young, apparently healthy adult have not changed over the past 4 decades.

From the *Brooke Army Medical Center, San Antonio, Texas; †Landstuhl Regional Medical Center, Landstuhl, Germany; ‡Armed Forces Institute of Pathology, Washington, DC; §Walter Reed Army Medical Center, Washington, DC; and the ‖Brigham & Women's Hospital, Boston, Massachusetts. Dr. Appel is currently affiliated with Northwestern University, Chicago, Illinois. Dr. Avedissian is currently affiliated with Madigan Army Medical Center, Seattle, Washington. Dr. Pearse is currently affiliated with the Uniformed Services University, Bethesda, Maryland. Dr. Tremaine is currently affiliated with the Landstuhl Regional Medical Center, Landstuhl, Germany. Dr. Gentlesk is currently affiliated with Cardiology Consultants Ltd., Norfolk, Virginia. The opinions and research contained herein are the private ones of the authors and are not to be considered as official or reflecting the views of the Department of the Army or the Department of Defense. All authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Manuscript received November 22, 2010; revised manuscript received December 22, 2010, accepted January 31, 2011.

Table 3 Cause-Specific Findings in 902 Cases of Adjudicated Unanticipated Sudden Cardiac Death Stratified by Age <35 Years and ≥35 Years in a Cohort Undergoing Active Surveillance

Findings	<35 Yrs of Age (n = 298)	≥35 Yrs of Age (n = 604)	p Value
Sudden unexplained death	123 (41.3%)	64 (10.6%)	<0.001
Atherosclerotic disease	69 (23.2%)	442 (73.2%)	<0.001
Hypertrophic cardiomyopathy	38 (12.8%)	19 (3.1%)	<0.001
Myocarditis	17 (5.7%)	13 (2.2%)	0.009
Idiopathic dilated cardiomyopathy	14 (4.7%)	21 (3.5%)	0.478
Anomalous coronary artery	12 (4.0%)	1 (0.2%)	<0.001
Hypertensive cardiomyopathy	11 (3.7%)	15 (2.5%)	0.419
Arrhythmogenic RV dysplasia	4 (1.3%)	6 (1.0%)	0.737
Ischemic cardiomyopathy	2 (0.7%)	14 (2.3%)	0.135
Other*	8 (2.7%)	9 (1.5%)	—

Data presented as raw (columnar percent [incidence]). *Other cases (n = cases <35 years of age, cases ≥35 years of age, respectively): additional causes of death associated with coronary artery disease included coronary artery bridge (n = 6, 1), spontaneous coronary thrombosis (n = 1, 2%) and spontaneous coronary dissection (n = 0, 1); causes of death associated with valvular heart disease included aortic valve disease (n = 0, 3), mitral valve disease (n = 1, 1), and endocarditis (n = 0, 1).

RV = right ventricle.

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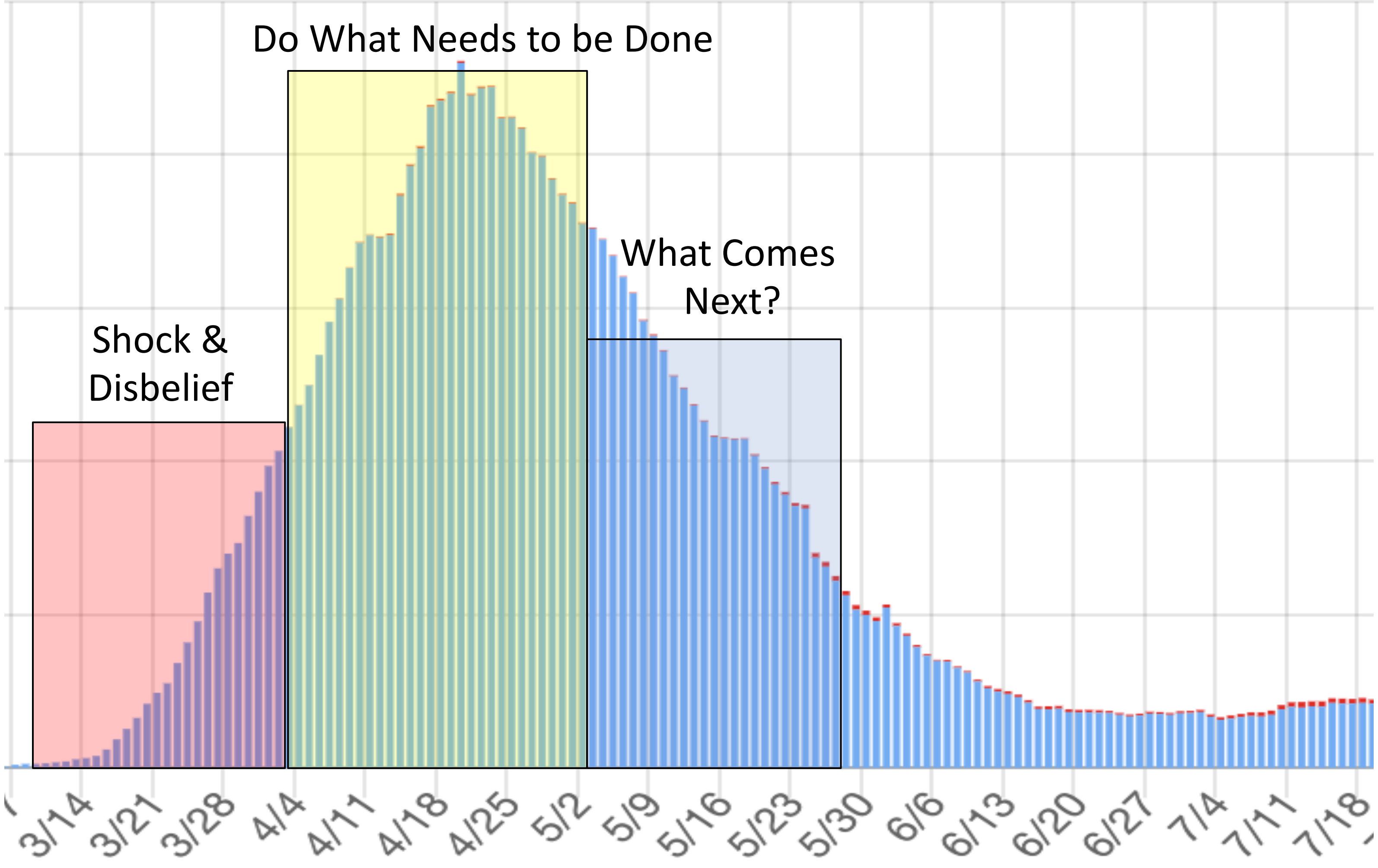

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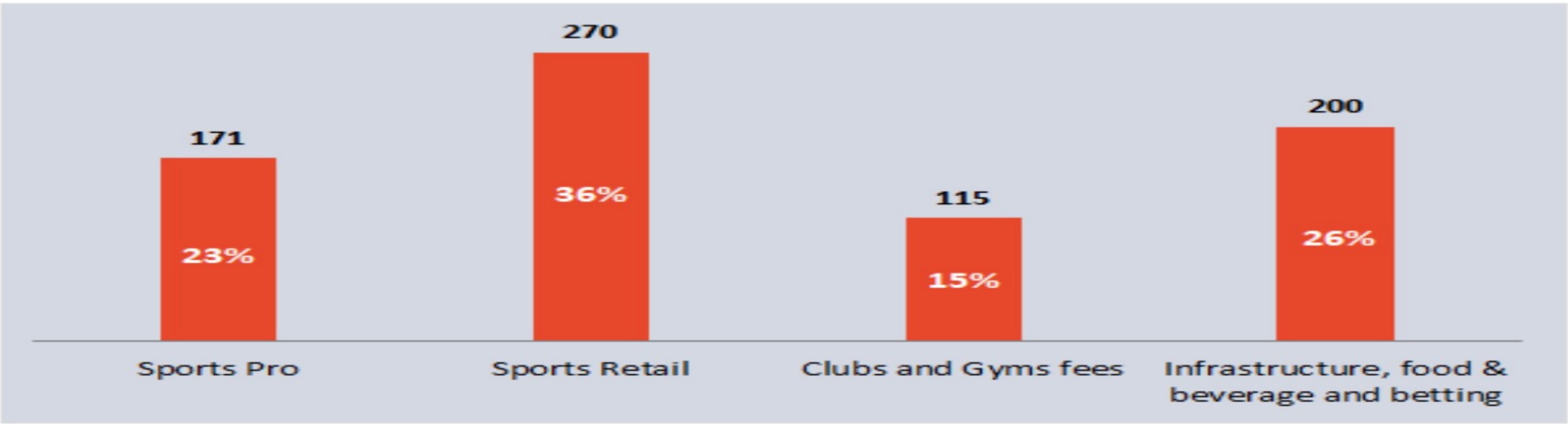
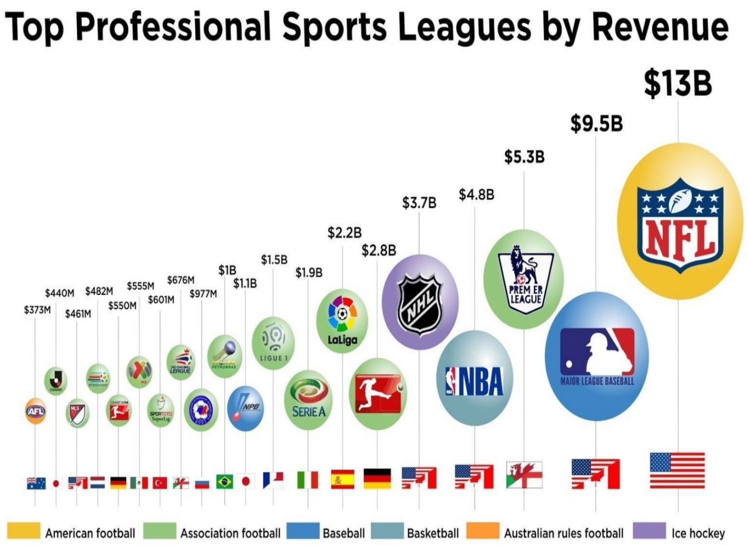
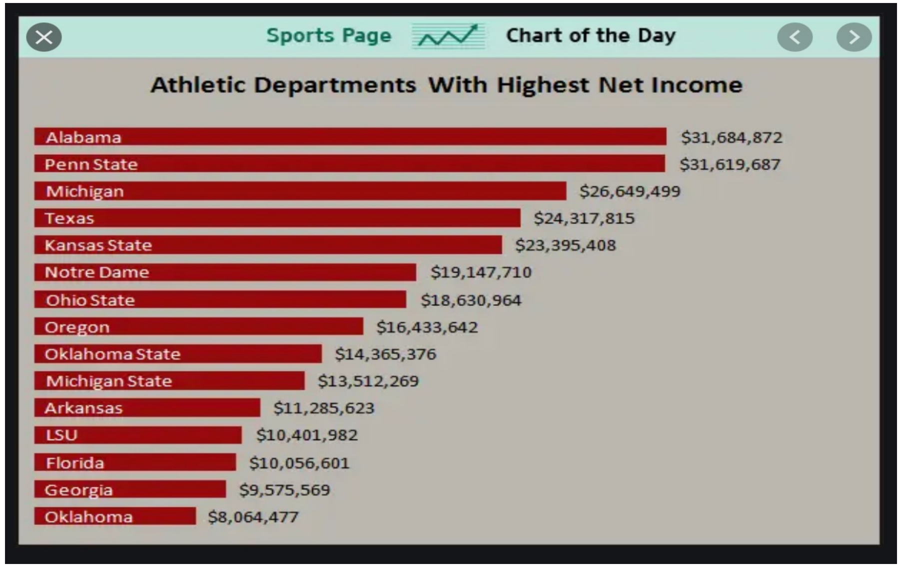
Global Revenues- Sports Industry- US$ billion







COVID-19 Cardiac

Morbidity and Mortality Weekly Report

An Outbreak of COVID-19 Associated with a Recreational Hockey Game — Florida, June 2020

David Atrubin[1]; Michael Wiese[2]; Becky Bohinc[3]

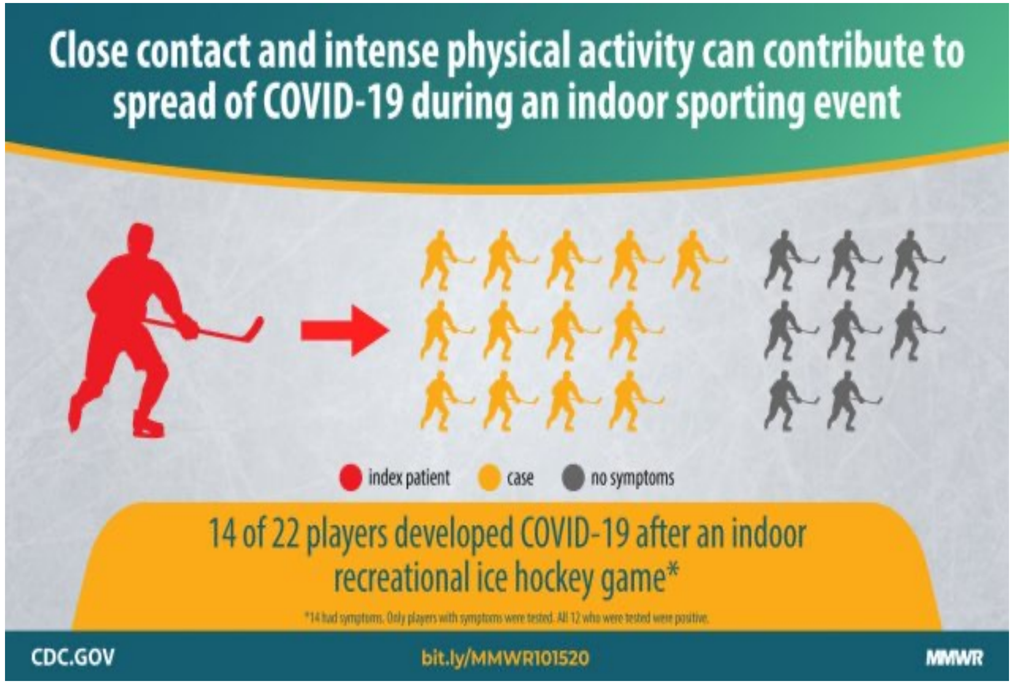
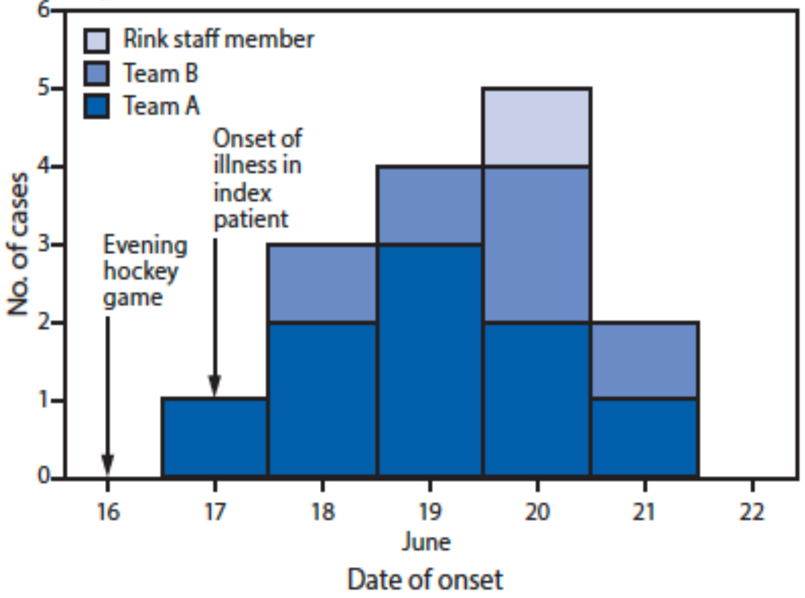


FIGURE. COVID-19 cases associated with a recreational ice hockey game, by date of onset (N = 15) — Florida, June 2020



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Resurgence of sport in the wake of COVID-19: cardiac considerations in competitive athletes

Aaron Baggish,[1] Jonathan A Drezner ,[2] Jonathan Kim,[3] Matthew Martinez,[4] Jordan M Prutkin[5]

Blog | British Journal of Sports Medicine

Posted on April 24, 2020 by BMJ

Table 1 Cardiac evaluation in athletes with prior COVID-19 infection

Clinical scenario	
Athletes with *prior asymptomat* confirmed antibody to severe a syndrome coronavirus 2	o COVID-19 should be ith a history of new-onset n in the absence of fever s), palpitations or exercise valuation, regardless of d in athletes with new-onset s or exercise intolerance.
Athletes with a *history of mild i hospitalised)* related to confirm COVID-19	ndicate viral-induced myocardial al Q waves, ST segment e ST segment elevation and valuation, regardless of d in athletes with new-onset s or exercise intolerance.
Athletes with a history of mode illness (hospitalised) related to suspected COVID-19	*ore likely in patients with a* rse, and normal cardiac function hould be established prior to a onsidered based on clinical njury.†
Athletes with a *history of COVI (regardless of severity) and doc myocardial injury* as indicated b of the following: in-hospital EC or NP elevation, arrhythmia or i function	uld be gradual and under the gist. o, including serial cardiac d in athletes with initially n.

*ECG as a screening test to exc be warranted based on clinical †Cardiac MRI should be perform events.
es. Additional evaluation may major adverse cardiovascular
hs-Tn, high-sensitivity cardiac troponin; LGE, late gadolinium enhancement; NP, natriuretic peptide.

safety. There are fundamental questions about how COVID-19 will leave its mark on the millions of athletes world-wide and what steps should be taken to prevent further unnecessary loss of life. While these questions will be asked by the men and women on the front lines of athlete care, they will only be answered by the sharing of experiences and the pooling of rigorously collected data.

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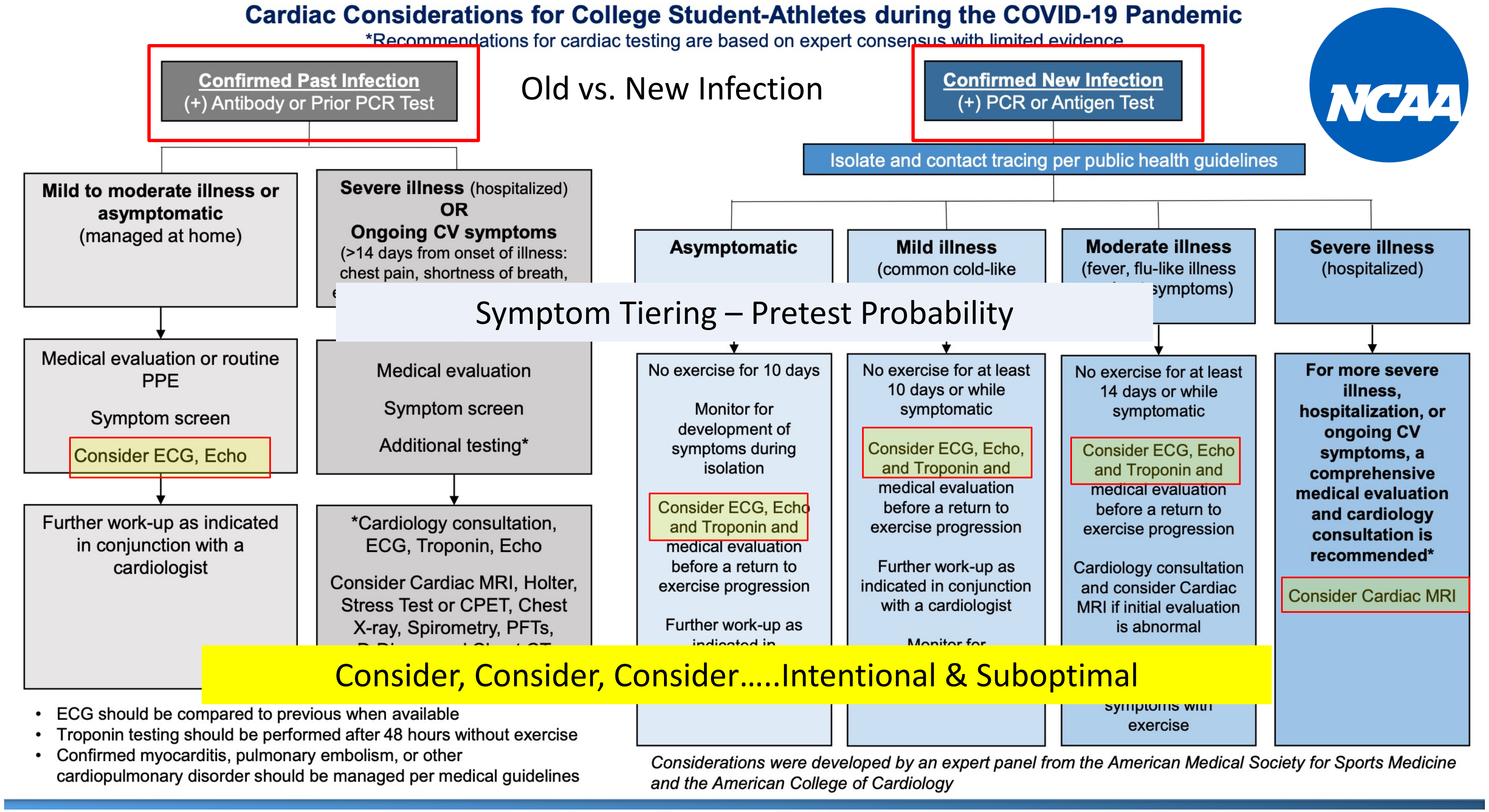

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VIEWPOINT

A Game Plan for the Resumption of Sport and Exercise After Coronavirus Disease 2019 (COVID-19) Infection

JAMA Cardiology

Circulation

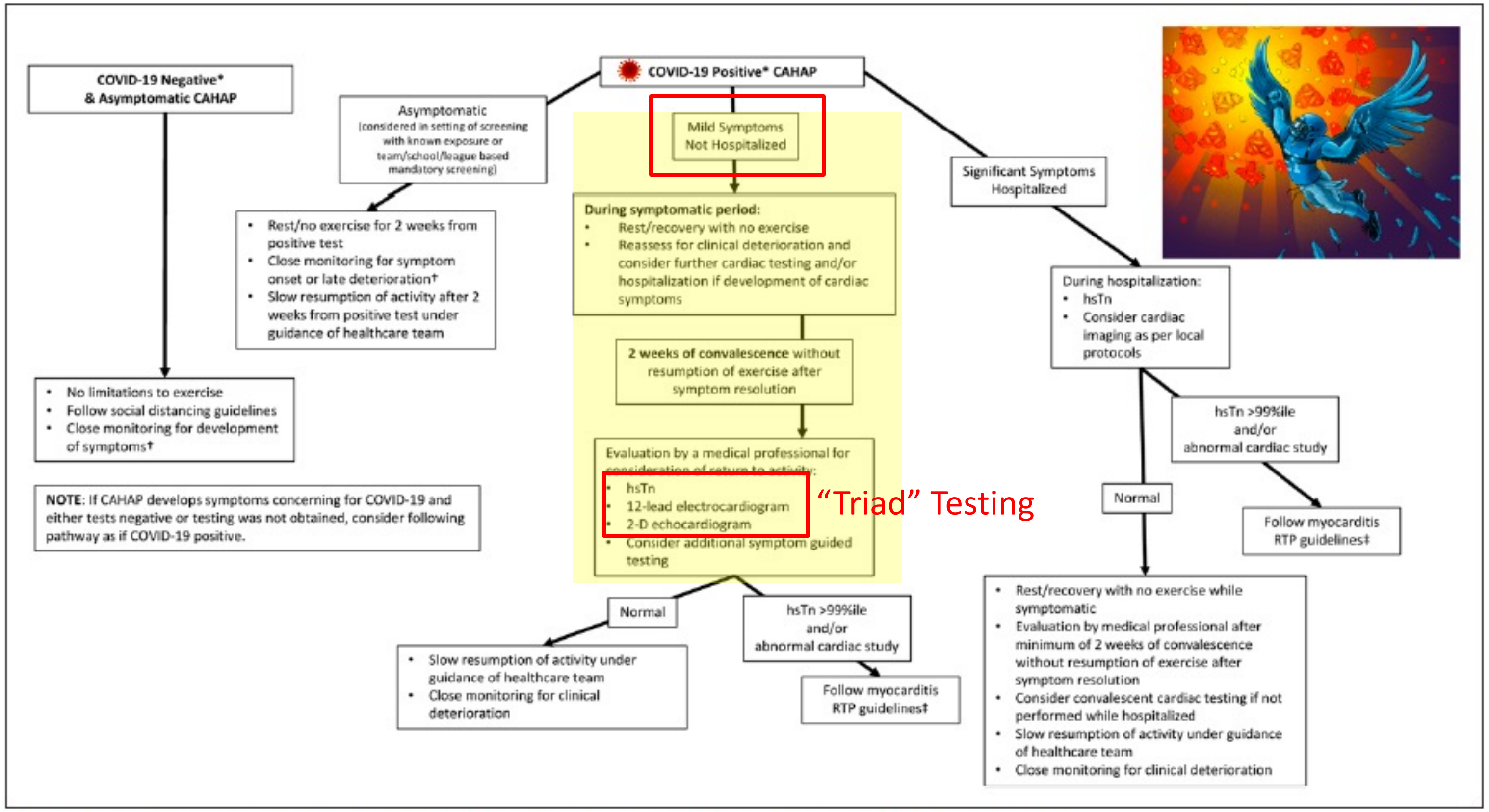

Figure. Coronavirus disease 2019 return-to-play algorithm for competitive athlete and highly active people.
*Typical testing obtained via a nasopharyngeal swab. All athletes with positive testing should be isolated for 2 weeks regardless of symptoms. †If clinical or cardiac symptoms develop, follow appropriate clinical pathway. ‡Given lack of clear pathophysiology, we recommend the American College of Cardiology/American Heart Association Athlete myocarditis guidelines. CAHAP indicates competitive athlete or highly-active person; COVID-19, coronavirus disease 2019; hsTn, high-sensitivity troponin-I; and RTP, return-to-play. Reproduced with Permission from *JAMA Cardiology*[5] Copyright©2020. American Medical Association. All rights reserved.

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“Triad” Testing: Early Experience

Asymptomatic & Mild COVID-19

Lots of Testing, Very Little Pathology

Jamain Stephens, a football player at California University of Pennsylvania and the son of a former Steelers first round-draft pick, has died at the age of 20 of what was initially described as COVID-19 complications but was later said to be unconfirmed.

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JAMA Cardiology | Original Investigation

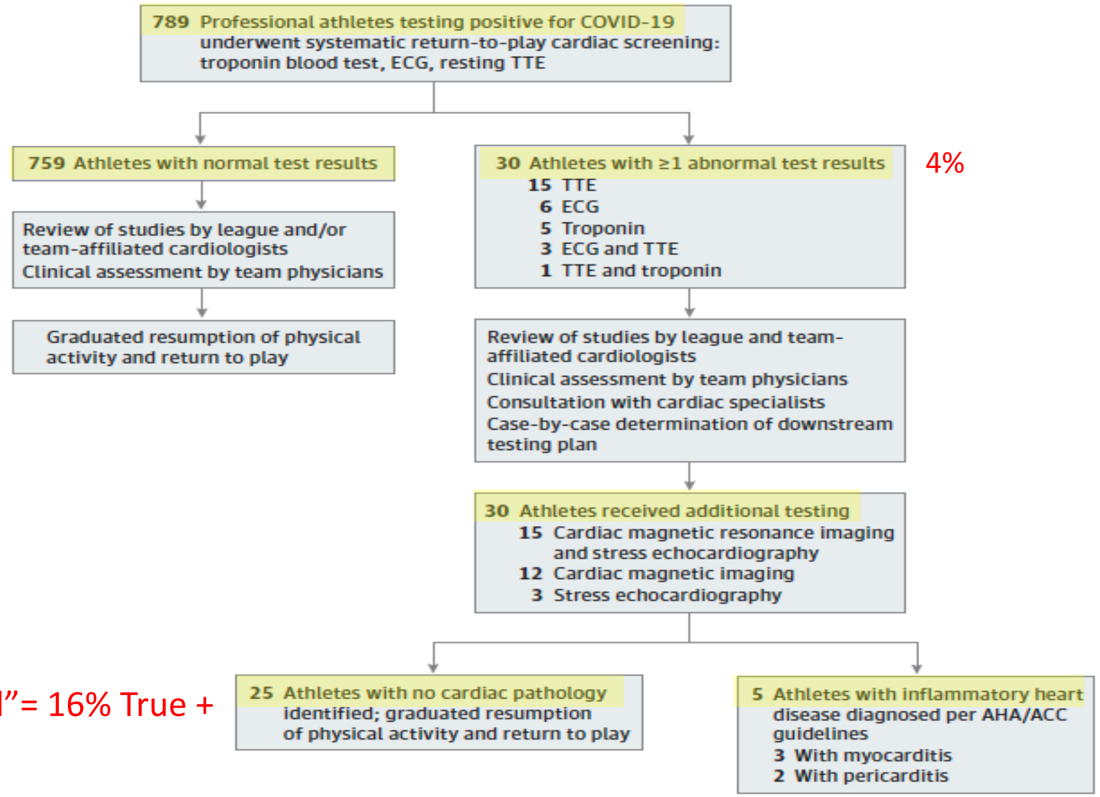
Prevalence of Inflammatory Heart Disease Among Professional Athletes With Prior COVID-19 Infection Who Received Systematic Return-to-Play Cardiac Screening

Matthew W. Martinez, MD; Andrew M. Tucker, MD; O. Josh Bloom, MD, MPH; Gary Green, MD;
John P. DiFiori, MD; Gary Solomon, PhD; Dermot Phelan, MD, PhD; Jonathan H. Kim, MD, MSc;
Willem Meeuwisse, MD, PhD; Allen K. Sills, MD; Dana Rowe, BA; Isaac I. Bogoch, MD; Paul T. Smith, MD;
Aaron L. Baggish, MD; Margot Putukian, MD; David J. Engel, MD

Figure. Flow Diagram of the Systematic Return-to-Play Cardiac Screening Process Used for Professional Athletes Testing Positive for Coronavirus Disease 2019 (COVID-19)

96%

4%



+"Triad"= 16% True +

0.6%

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COVID-19 Cardiac

Resumption of Sport at the United States Olympic and Paralympic Training Facilities During the COVID-19 Pandemic

Ankit B. Shah, MD, MPH,*† Dustin Nabhan, DC,‡ Robert Chapman, PhD,§
George Chiampas, DO,‖ Jonathan Drezner, MD,¶ J. Tod Olin, MD, MSCS,#
David Taylor, BPhys (Hons), MHPS (Hons),‡ Jonathan T. Finnoff, DO,‡ and Aaron L. Baggish, MD**

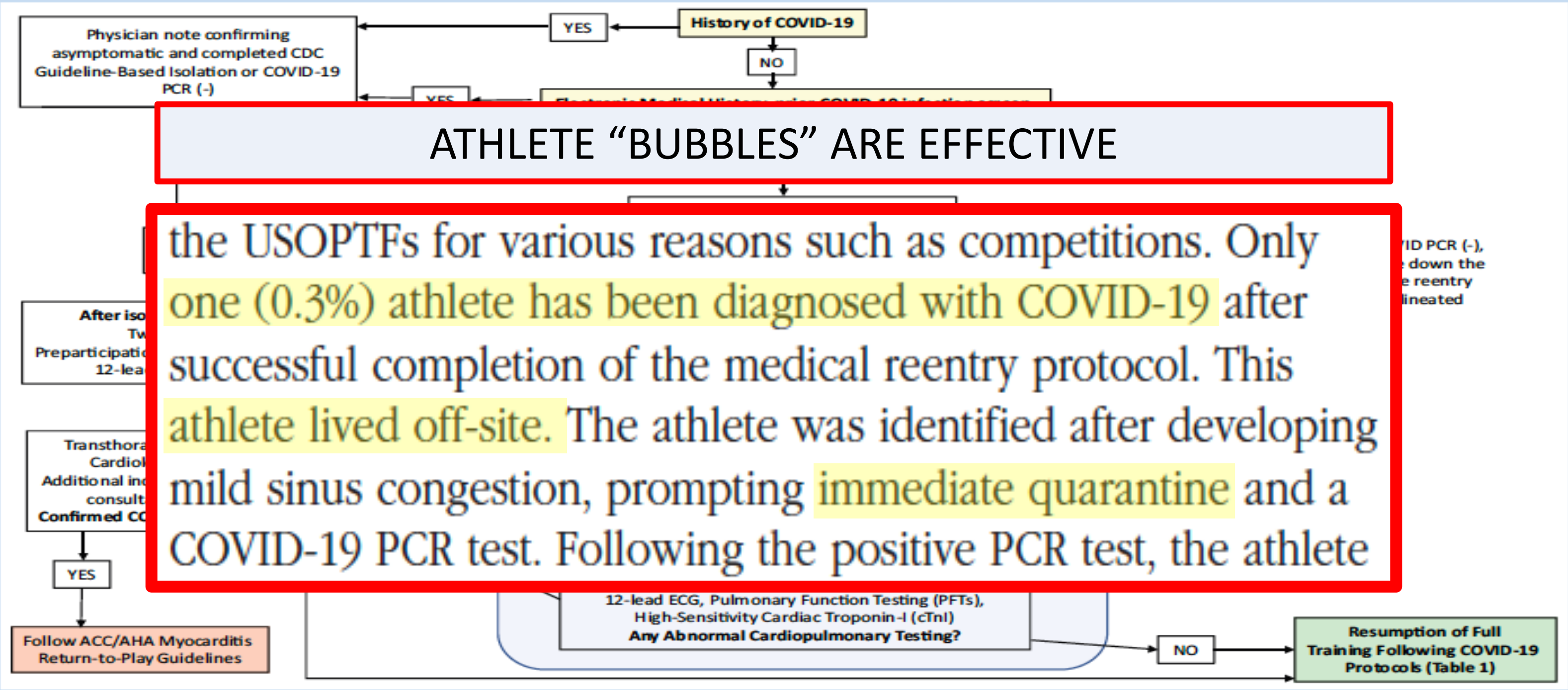

Figure 1. The United States Olympic and Paralympic Training facilities' (USOPTFs) COVID-19 pandemic "return-to-training" protocol. Ab, antibody; COVID-19, coronavirus disease 2019; cTnI, cardiac specific troponin I assay; ECG, electrocardiogram; PCR, polymerase chain reaction; PFT, pulmonary function testing; SS, signs and symptom survey.

COVID-19 Cardiac

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JAMA Cardiology | Special Communication

Coronavirus Disease 2019 and the Athletic Heart
Emerging Perspectives on Pathology, Risks, and Return to Play

Jonathan H. Kim, MD, MSc; Benjamin D. Levine, MD; Dermot Phelan, MD, PhD; Michael S. Emery, MD, MS; Mathew W. Martinez, MD; Eugene H. Chung, MD, MSc; Paul D. Thompson, MD; Aaron L. Baggish, MD

What Is Known About the Effects of COVID-19 Infection on the Heart For Individuals With Mild or No Symptoms

Symptoms defining mild COVID-19 include nonspecific and self-limited fatigue; anosmia or ageusia; nausea, vomiting, and/or diarrhea; headache; cough; sore throat; and nasopharyngeal congestion.[11] Progression to moderate or severe disease and the potential need for hospitalization are characterized by the onset of systemic symptoms (persistent fever [temperature ≥100.4 °F] or chills, myalgias, severe lethargy, and hypoxia or pneumonia) and/or cardiovascular (CV) symptoms (dyspnea and chest pain, tightness, or pressure at rest or during exertion).[11] In patients hospitalized with moderate or severe COVID-19, particularly among those with underlying CV conditions, cardiac injury is common (>20% of cases).[2,12,13] However, the pathogenesis of

Myocarditis (Probable Acute Myocarditis With Both of the Following Criteria)

1. Clinical syndrome, including acute heart failure, angina-type chest pain, or known myopericarditis of less than 3 months' duration.
2. Otherwise unexplained increase in serum troponin levels, ischemic 12-lead electrocardiogram changes, arrhythmias or high-grade atrioventricular block, regional wall-motion abnormalities, or pericardial effusion. Additional cardiac magnetic resonance imaging findings that suggest myocarditis in the short-term clinical setting include altered tissue signals on T2-weighted or T1-weighted images and late gadolinium enhancement.

October 2020

COVID-19 Cardiac

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MGH 1811 Cardiovascular Performance Program

JAMA Cardiology | Special Communication

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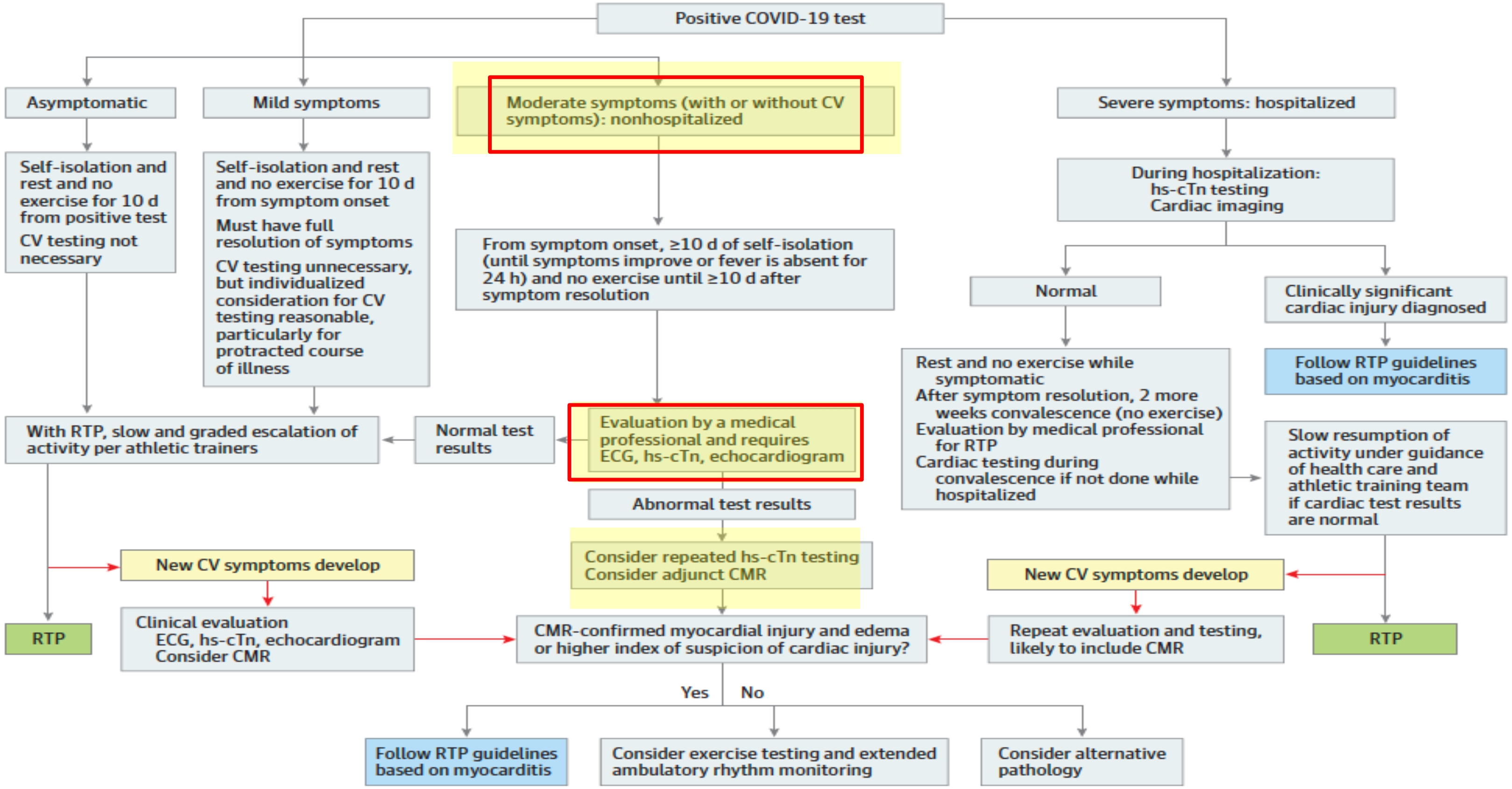
Figure 3. Proposed Coronavirus Disease 2019 (COVID-19) Return-to-Play Algorithm for Adult Athletes in Competitive Sports



COVID-19 Cardiac

ESPN

B1G

9/16/2020 9:12:11 AM

The Big Ten Conference Adopts Stringent Medical Protocols; Football Season to Resume October 23-24, 2020

COLLEGE SPORTS NOW
A podcast for college sports fans, by college sports fans.
New episodes weekly!

All COVID-19 positive student-athletes will have to undergo comprehensive cardiac testing to include labs and biomarkers, ECG, Echocardiogram and a Cardiac MRI. Following cardiac evaluation, student-athletes must receive clearance from a cardiologist designated by the university for the primary purpose of cardiac clearance for COVID-19 positive student-athletes. The earliest a student-athlete can return to game competition is 21 days following a COVID-19 positive diagnosis.

MANDATORY SCREENING MRI!!!

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LLC+ Cardiac Injury = 2 to 15%

Pericardial Injury = 0 to 40%

Isolated LGE = 0 to 46%

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COVID-19 Cardiac

Cardiovascular Performance Program

The "ORCCA" Registry

Outcomes Registry for Cardiac Conditions in Athletes

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Bradley Petek

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MGH 1811

Circulation

ORIGINAL RESEARCH ARTICLE

SARS-CoV-2 Cardiac Involvement in Young Competitive Athletes

Nathaniel Moulson, MD*; Bradley J. Petek, MD*; Jonathan A. Drezner, MD; Kimberly G. Harmon, MD; Stephanie A. Kliethermes, PhD; Manesh R. Patel, MD; Aaron L. Baggish, MD; for the Outcomes Registry for Cardiac Conditions in Athletes Investigators†

BACKGROUND: Cardiac involvement among hospitalized patients with severe coronavirus disease 2019 (COVID-19) is common and associated with adverse outcomes. This study aimed to determine the prevalence and clinical implications of COVID-19 cardiac involvement in young competitive athletes.

METHODS: In this prospective, multicenter, observational cohort study with data from 42 colleges and universities, we assessed the prevalence, clinical characteristics, and outcomes of COVID-19 cardiac involvement among collegiate athletes in the United States. Data were collected from September 1, 2020, to December 31, 2020. The primary outcome was the prevalence of definite, probable, or possible COVID-19 cardiac involvement based on imaging definitions adapted from the Updated Lake Louise Imaging Criteria. Secondary outcomes included the diagnostic yield of cardiac testing, predictors for cardiac involvement, and adverse cardiovascular events or hospitalizations.

RESULTS: Among 19 378 athletes tested for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, 3018 (mean age, 20 years [SD, 1 year]; 32% female) tested positive and underwent cardiac evaluation. A total of 2820 athletes underwent at least 1 element of cardiac triad testing (12-lead ECG, troponin, transthoracic echocardiography) followed by cardiac magnetic resonance imaging (CMR) if clinically indicated. In contrast, primary screening CMR was performed in 198 athletes. Abnormal findings suggestive of SARS-CoV-2 cardiac involvement were detected by ECG (21 of 2999 [0.7%]), cardiac troponin (24 of 2719 [0.9%]), and transthoracic echocardiography (24 of 2556 [0.9%]). Definite, probable, or possible SARS-CoV-2 cardiac involvement was identified in 21 of 3018 (0.7%) athletes, including 15 of 2820 (0.5%) who underwent clinically indicated CMR (n=119) and 6 of 198 (3.0%) who underwent primary screening CMR. Accordingly, the diagnostic yield of CMR for SARS-CoV-2 cardiac involvement was 4.2 times higher for a clinically indicated CMR (15 of 119 [12.6%]) versus a primary screening CMR (6 of 198 [3.0%]). After adjustment for race and sex, predictors of SARS-CoV-2 cardiac involvement included cardiopulmonary symptoms (odds ratio, 3.1 [95% CI, 1.2, 7.7]) or at least 1 abnormal triad test result (odds ratio, 37.4 [95% CI, 13.3, 105.3]). Five (0.2%) athletes required hospitalization for noncardiac complications of COVID-19. During clinical surveillance (median follow-up, 113 days [interquartile range=90 146]), there was 1 (0.03%) adverse cardiac event, likely unrelated to SARS-CoV-2 infection.

CONCLUSIONS: SARS-CoV-2 infection among young competitive athletes is associated with a low prevalence of cardiac involvement and a low risk of clinical events in short-term follow-up.

Key Words: athletes ■ COVID-19 ■ myocarditis ■ return to sport ■ SARS-CoV-2

Editorial, see p 267

Cardiac involvement associated with adverse outcomes is common among hospitalized patients with severe coronavirus disease 2019 (COVID-19).[1] Limited data exist on the prevalence and clinical relevance of cardiac involvement in nonhospitalized–and otherwise healthy–populations including young

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*N. Moulson and B.J. Petek contributed equally.

†A complete list of the members of the Outcomes Registry for Cardiac Conditions in Athletes Study Group is provided in the Data Supplement.

Continuing medical education (CME) credit is available for this article. Go to http://cme.ahajournals.org to take the quiz.

The Data Supplement, podcast, and transcript are available with this article at www.ahajournals.org/doi/suppl/10.1161/circulationaha.121.054824.

For Sources of Funding and Disclosures, see page 265.

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CONCLUSIONS: SARS-CoV-2 infection among young competitive athletes is associated with a low prevalence of cardiac involvement and a low risk of clinical events in short-term follow-up.

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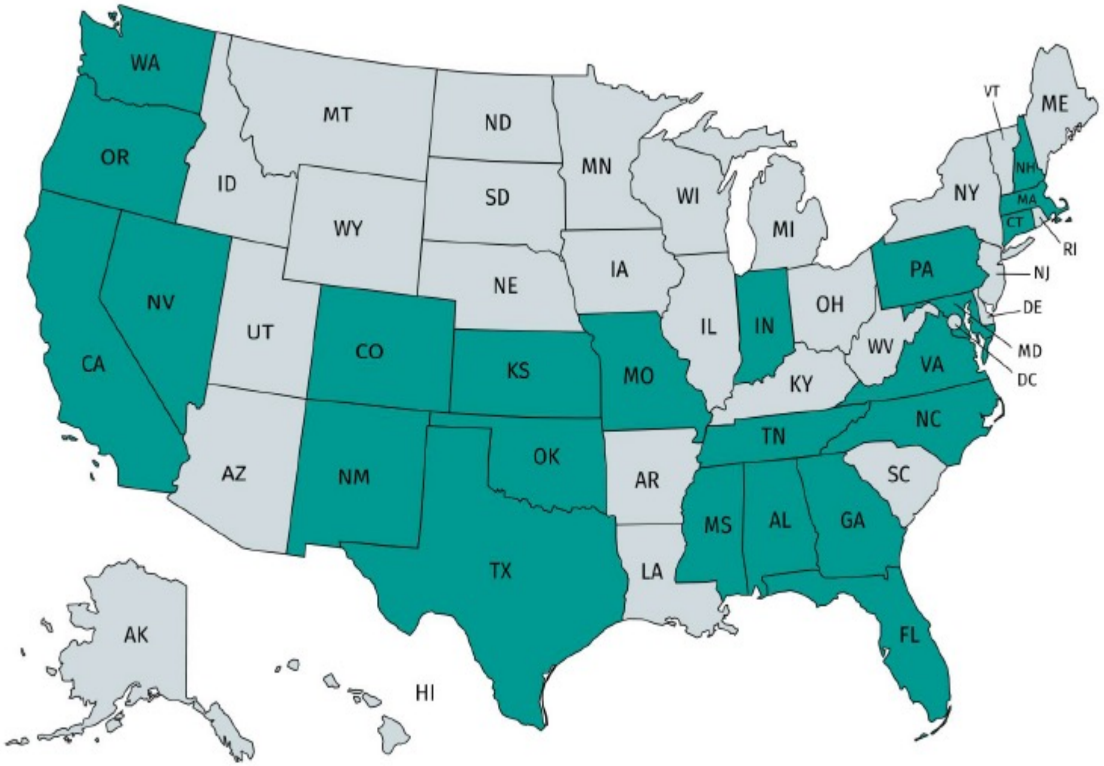
COVID-19 Cardiac

Cardiovascular Performance Program (MGH 1811)

42 Colleges/Universities • 23 States • 14 Athletic Conferences



Median Length of Follow-up = 113 days IQR [90,146]

Total Athletes Tested for SARS-CoV-2 (n=19,378)

↓

SARS-CoV-2 Positive Athletes (n=3384) — **17.5%**

↓ → No cardiac evaluation per school preference (n=74)

Planned Cardiac Evaluation (n=3310) — **17.1%**

↓ → Incomplete Evaluation
- Athlete quit/cut (n=10)
- Testing not started (n=98)
- Missing data from incomplete school submission (n=184)

Final Cohort with Cardiac Evaluation (n=3018) 15.6% — **15.6%**

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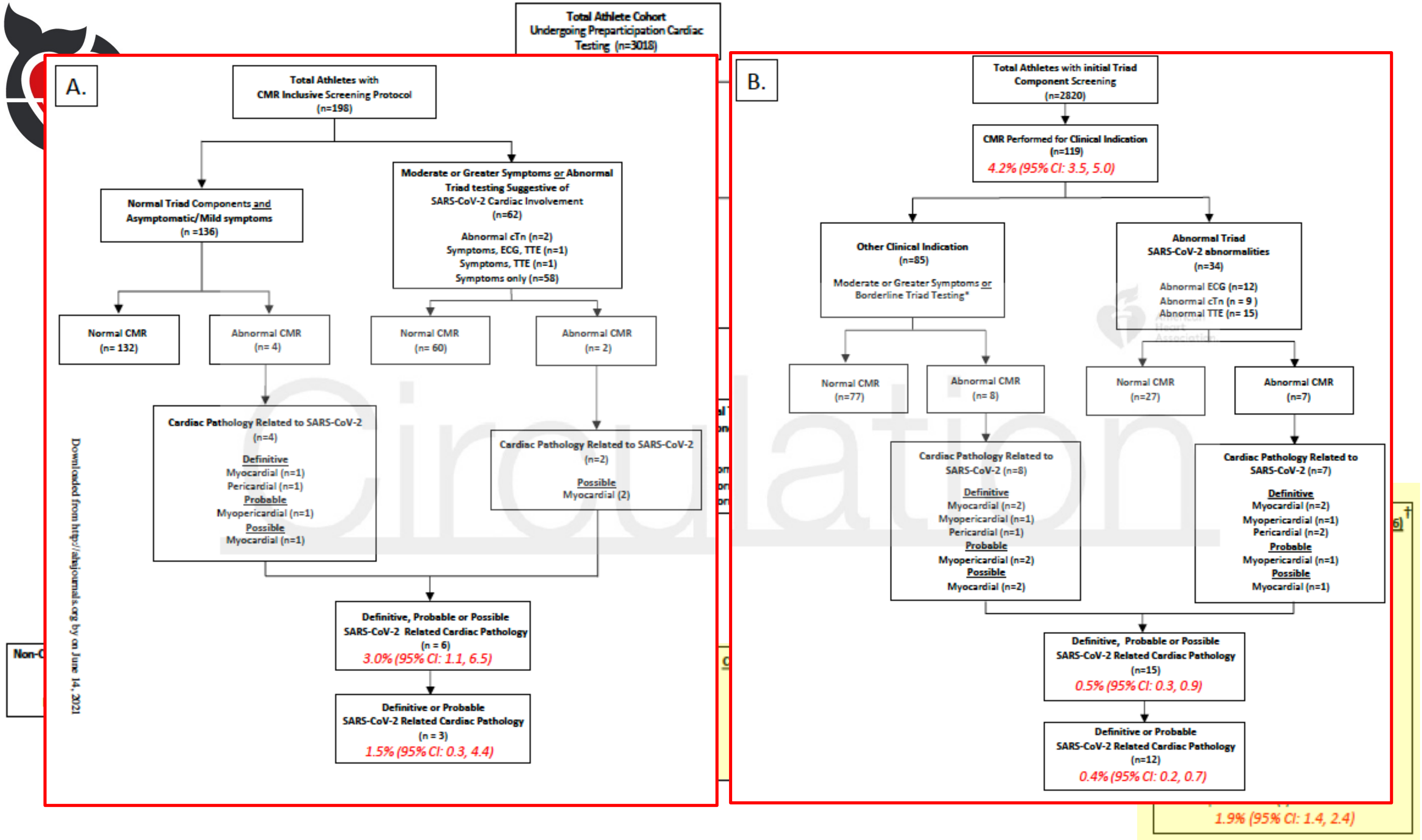
COVID-19 Cardiac

Cardiovascular Performance Program



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COVID-19 Cardiac

MGH 1811 Cardiovascular Performance Program

The "ORCCA" Registry

Initial Conclusions

Cardiac screening unnecessary for all young competitive athletes
-focus on moderate disease and symptoms on return to exercise-
(Moulson et al. Circulation 2021)

MRI is not recommended as a primary screening modality
-reserve for use in patients with intermediate or greater pretest probability of disease
(Petek et al. *BJSM* 2022)

Data suggest a benign clinical course for young competitive athletes with COVID-19 cardiac inflammation, long term f/u needed (Petek et al, *Circulation 2022*)

Screening will detect non-COVID cardiac issues more commonly (3:1) than true COVID cardiac involvement (Klein et al., *Heart, 2023)*

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COVID-19 Cardiac

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JAMA Cardiology | Original Investigation

Prevalence of Clinical and Subclinical Myocarditis in Competitive Athletes With Recent SARS-CoV-2 Infection

Results From the Big Ten COVID-19 Cardiac Registry

Curt J. Daniels, MD; Saurabh Rajpal, MBBS, MD; Joel T. Greenshields, MS; Geoffrey L. Rosenthal, MD; Eugene H. Chung, MD; Michael Terrin, MD; Jean Jeudy, MD; Scott E. Mattson, DO; Ian H. Law, MD; James Borchers, MD; Richard Kovacs, MD; Jeffrey Kovan, DO; Sami F. Rifat, MD; Jennifer Albrecht, PhD; Ana I. Bento, PhD; Lonnie Albers, MD; David Bernhardt, MD; Carly Day, MD; Suzanne Hecht, MD; Andrew Hipskind, MD; Jeffrey Mjaanes, MD; David Olson, MD; Yvette L. Rooks, MD; Emily C. Somers, PhD; Matthew S. Tong, DO; Jeffrey Wisinski, DO; Jason Womack, MD; Carrie Esopenko, PhD; Christopher J. Kratochvil, MD; Lawrence D. Rink, MD; for the Big Ten COVID-19 Cardiac Registry Investigators

Key Points

Question What is the prevalence of myocarditis in competitive athletes after COVID-19 infection, and how would different approaches to screening affect detection?

Findings In this cohort study of 1597 US competitive collegiate athletes undergoing comprehensive cardiovascular testing, the prevalence of clinical myocarditis based on a symptom-based screening strategy was only 0.31%. Screening with cardiovascular magnetic resonance imaging increased the prevalence of clinical and subclinical myocarditis by a factor of 7.4 to 2.3%.

Meaning These cardiac magnetic resonance imaging findings provide important data on the prevalence of clinical and subclinical myocarditis in college athletes recovering from symptomatic and asymptomatic COVID-19 infections.

IMPORTANCE Myocarditis is a leading cause of sudden death in competitive athletes. Myocardial inflammation is known to occur with SARS-CoV-2. Different screening approaches for detection of myocarditis have been reported. The Big Ten Conference requires comprehensive cardiac testing including cardiac magnetic resonance (CMR) imaging for all athletes with COVID-19, allowing comparison of screening approaches.

OBJECTIVE To determine the prevalence of myocarditis in athletes with COVID-19 and compare screening strategies for safe return to play.

DESIGN, SETTING, AND PARTICIPANTS Big Ten COVID-19 Cardiac Registry principal investigators were surveyed for aggregate observational data from March 1, 2020, through December 15, 2020, on athletes with COVID-19. For athletes with myocarditis, presence of cardiac symptoms and details of cardiac testing were recorded. Myocarditis was categorized as clinical or subclinical based on the presence of cardiac symptoms and CMR findings. Subclinical myocarditis classified as probable or possible myocarditis based on other testing abnormalities. Myocarditis prevalence across universities was determined. The utility of different screening strategies was evaluated.

EXPOSURES SARS-CoV-2 by polymerase chain reaction testing.

MAIN OUTCOME AND MEASURE Myocarditis via cardiovascular diagnostic testing.

RESULTS Representing 13 universities, cardiovascular testing was performed in 1597 athletes (964 men [60.4%]). Thirty-seven (including 27 men) were diagnosed with COVID-19 myocarditis (overall 2.3%; range per program, 0%-7.6%); 9 had clinical myocarditis and 28 had subclinical myocarditis. If cardiac testing was based on cardiac symptoms alone, only 5 athletes would have been detected (detected prevalence, 0.31%). Cardiac magnetic resonance imaging for all athletes yielded a 7.4-fold increase in detection of myocarditis (clinical and subclinical). Follow-up CMR imaging performed in 27 (73.0%) demonstrated resolution of T2 elevation in all (100%) and late gadolinium enhancement in 11 (40.7%).

CONCLUSIONS AND RELEVANCE In this cohort study of 1597 US competitive athletes with CMR screening after COVID-19 infection, 37 athletes (2.3%) were diagnosed with clinical and subclinical myocarditis. Variability was observed in prevalence across universities, and testing protocols were closely tied to the detection of myocarditis. Variable ascertainment and unknown implications of CMR findings underscore the need for standardized timing and interpretation of cardiac testing. These unique CMR imaging data provide a more complete understanding of the prevalence of clinical and subclinical myocarditis in college athletes after COVID-19 infection. The role of CMR in routine screening for athletes safe return to play should be explored further.

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COVID-19 Cardiac

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Indiana University

Michigan State University

Northwestern University

Ohio State University

Penn State University

Purdue University

Rutgers University

University of Iowa

University of Maryland

University of Michigan

University of Minnesota

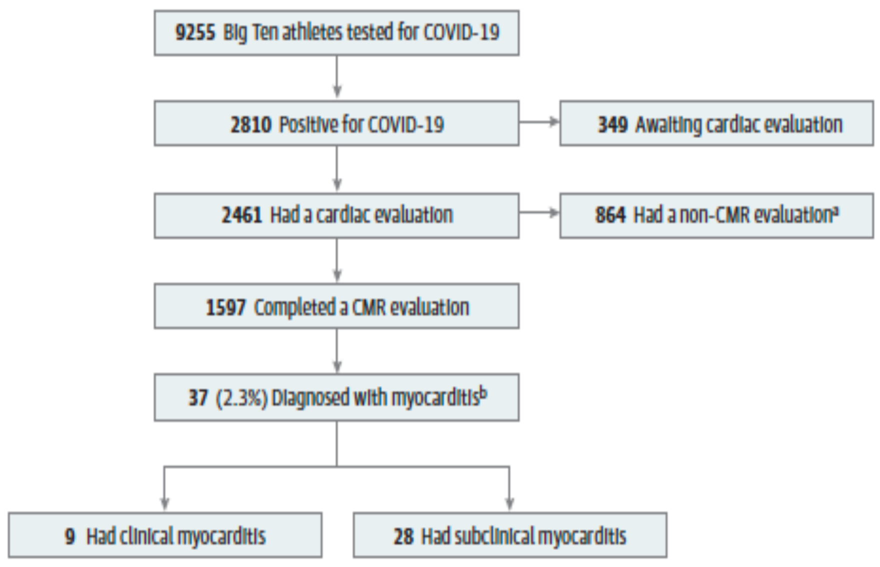
University of Nebraska

University of Wisconsin

Myocarditis Diagnosis Definitions

Myocarditis diagnoses were divided into 3 categories: (1) clinical myocarditis (cardiac symptoms present before or at the time of cardiac testing), (2) subclinical probable myocarditis (no cardiac symptoms) with abnormal ECG, echocardiogram, or troponin findings consistent with myocarditis, and (3) subclinical possible myocarditis (no cardiac symptoms) without abnormal ECG, echocardiogram, or troponin findings and only abnormal CMR imaging findings.

Figure 1. Cohort of Big Ten Athletes



[a] Athletes were excluded from analysis for not completing cardiac magnetic resonance (CMR) imaging as part of cardiac evaluation and described in more detail in eAppendix 2 in Supplement 1.

[b] Athletes diagnosed with myocarditis were categorized as clinical or subclinical based on presence or absence of cardiac symptoms.

COVID-19 Cardiac

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Table. Demographic, Imaging, and Biomarker Data for Athletes Diagnosed With Myocarditis[a]

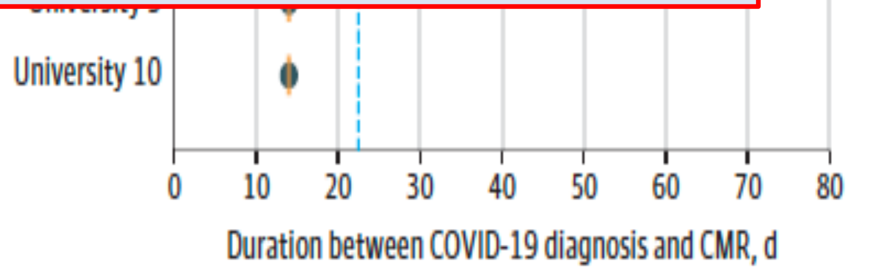
Athlete	Cardiac symptoms	Troponin level	ECG findings	ECHO findings	Time from COVID-19 diagnosis, d	CMR imaging findings	Follow-up CMR imaging time and findings
Clinical myocarditis							
1	Chest pain, palpitations	Elevated	Abnormal	Abnormal	46	↑T2, LGE	12 wk; Residual LGE
Subclinical probable myocarditis							
10	None	Elevated	NCM	NCM	30	↑T1,↑ T2, LGE	Pending[c]
11	None	Elevated	NCM	NCM	14	↑ T2, LGE	Pending[c]
12	None	Elevated	NCM	NCM	14	↑T2, LGE	12 wk; Residual LGE
13	None	Elevated	NCM	NCM	11	↑T2, LGE	4 wk; Residual LGE
14	None	Normal	Abnormal	NCM	13	↑T1,↑ T2, LGE	Pending[c]
15	None	Normal	NCM	Abnormal	42	↓LVEF, LGE	13 wk; Residual LGE
16	None	Normal	NCM	Abnormal	12	↓LVEF, LGE	4 wk; Resolved[b]
17	None	Normal	NCM	Abnormal	25	↑T1, ↑T2, LGE	Pending[c]
32	None	Normal	NCM	NCM	25	↑T2, LGE	Pending[c]
33	None	Normal	NCM	NCM	20	↑T2, LGE	11 wk; Resolved[b]
34	None	Normal	NCM	NCM	48	↑T2, LGE	Pending[c]
35	None	Normal	NCM	NCM	14	↑T1, ↑T2, LGE	Pending[c]
36	None	Normal	NCM	NCM	11	↑T2, LGE	12 wk; Residual LGE
37	None	Normal	NCM	NCM	19	↑T2, LGE	10 wk; Resolved[b]

Abbreviations: CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; ECHO, echocardiogram; LGE, late gadolinium …

… asymptomatic (subclinical myocarditis). Further breakdown of the subclinical myocarditis group with those demonstrating abnormal cardiac testing outside of CMR imaging (subclinical probable myocarditis) and those with only CMR … has not been performed.

"Triad+ Myocarditis" Prevalence = 1.0% (17/1597)

Overall CMR+ Prevalence = 2-3%



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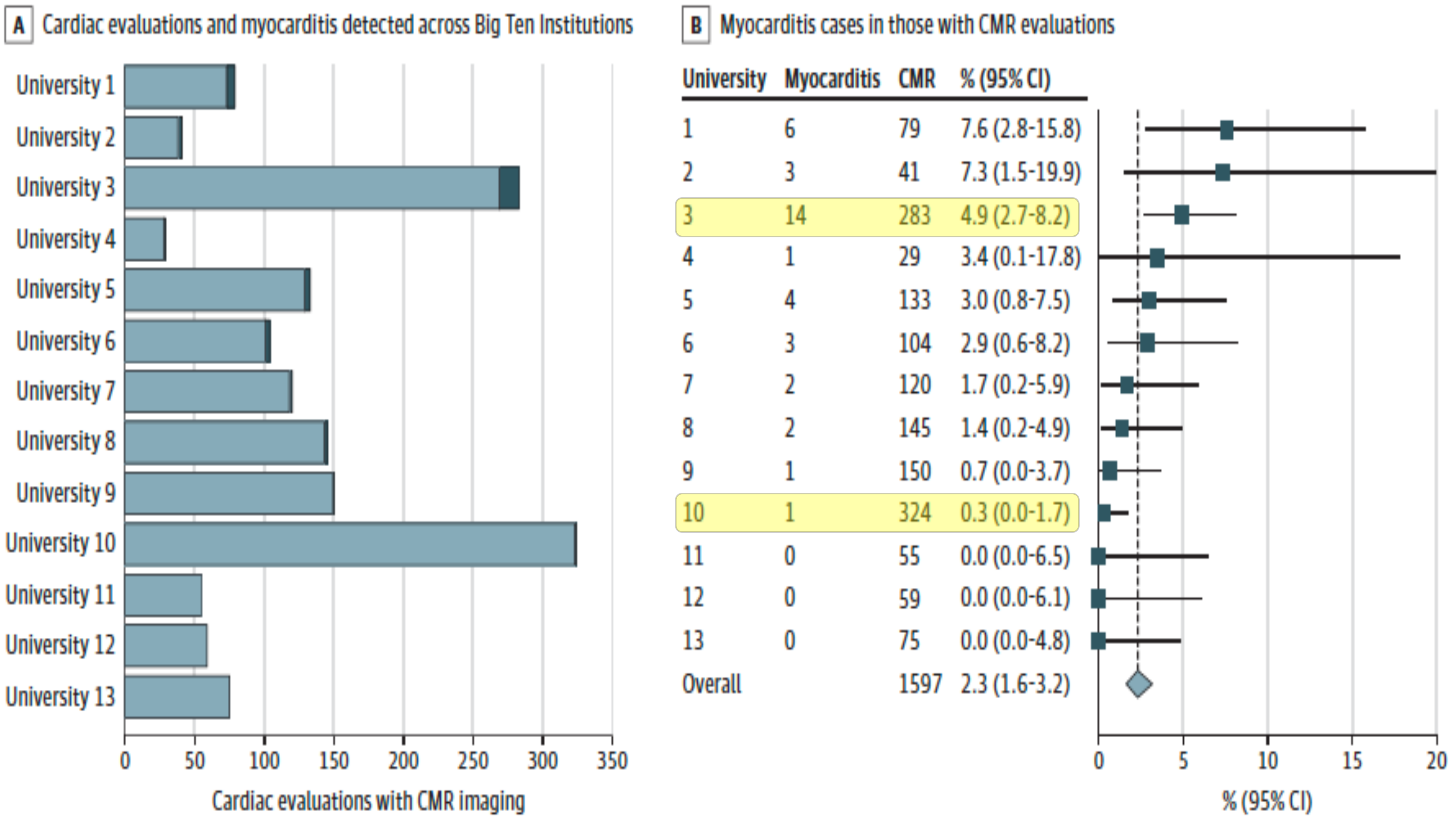

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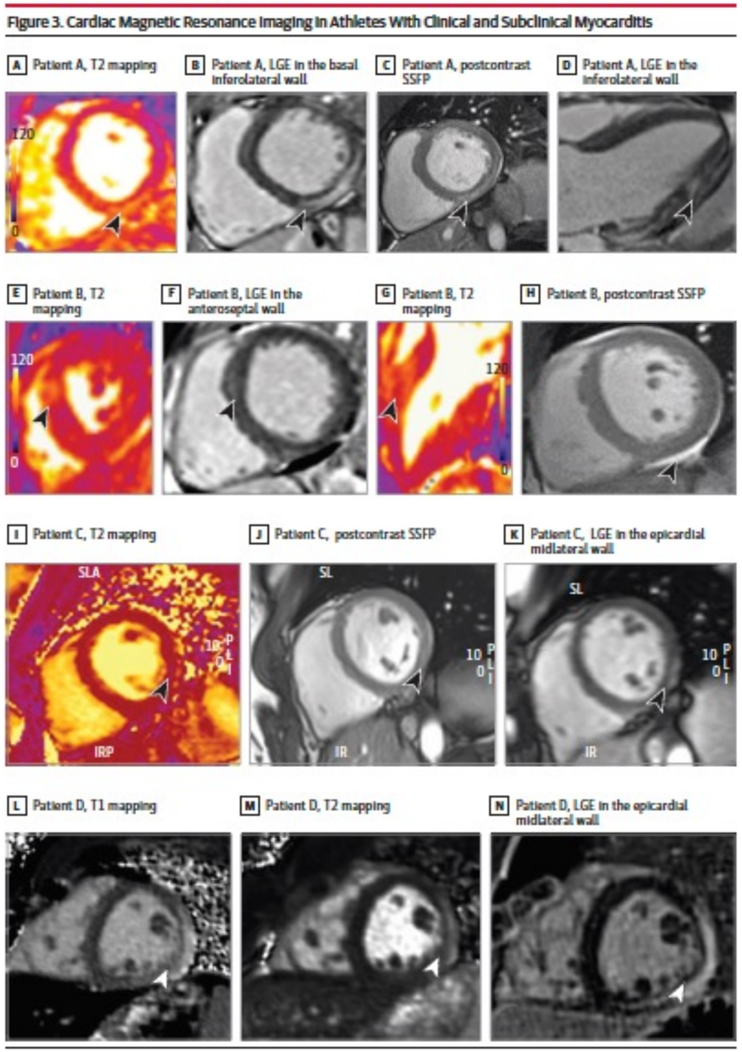


Is CMR Screening Ready for Prime Time?

Characteristics of a ”Good” Screening Test

~~Inexpensive~~

~~Easy to administer~~

~~Minimal discomfort~~

~~Reliable (consistent)~~

Valid (distinguishes dx from non-dx) ?

Predictive of Outcomes ?

Sources of Unreliability

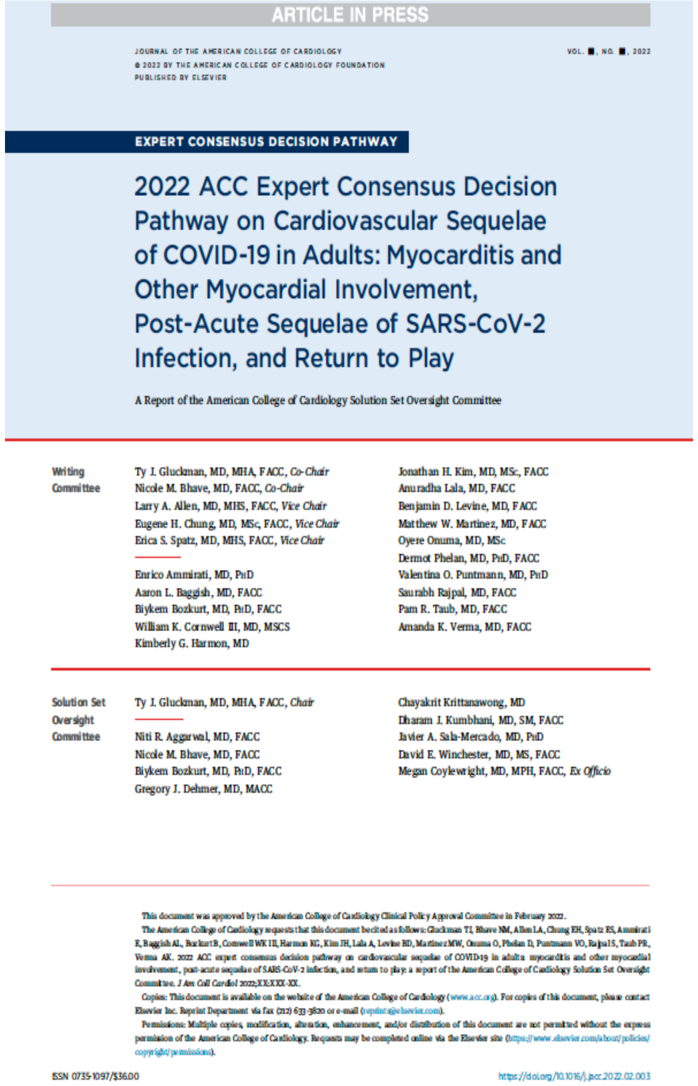
Biological variability ?

~~Instrument variability~~

Intra-observer variability ?

~~Inter-observer variability~~

COVID-19 Cardiac



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JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
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PUBLISHED BY ELSEVIER
VOL. ■, NO. ■, 2022

EXPERT CONSENSUS DECISION PATHWAY

2022 ACC Expert Consensus Decision Pathway on Cardiovascular Sequelae of COVID-19 in Adults: Myocarditis and Other Myocardial Involvement, Post-Acute Sequelae of SARS-CoV-2 Infection, and Return to Play

A Report of the American College of Cardiology Solution Set Oversight Committee

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This document was approved by the American College of Cardiology Clinical Policy Approval Committee in February 2022.

The American College of Cardiology requests that this document be cited as follows: Gluckman TJ, Bhave NM, Allen LA, Chung EH, Spatz ES, Ammirati E, Baggish AL, Bozkurt B, Cornwell WK III, Harmon KG, Kim JH, Lala A, Levine BD, Martinez MW, Onuma O, Phelan D, Puntmann VO, Rajpal S, Taub PR, Verma AK. 2022 ACC expert consensus decision pathway on cardiovascular sequelae of COVID-19 in adults: myocarditis and other myocardial involvement, post-acute sequelae of SARS-CoV-2 infection, and return to play: a report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol* 2022;XX:XXX-XX.

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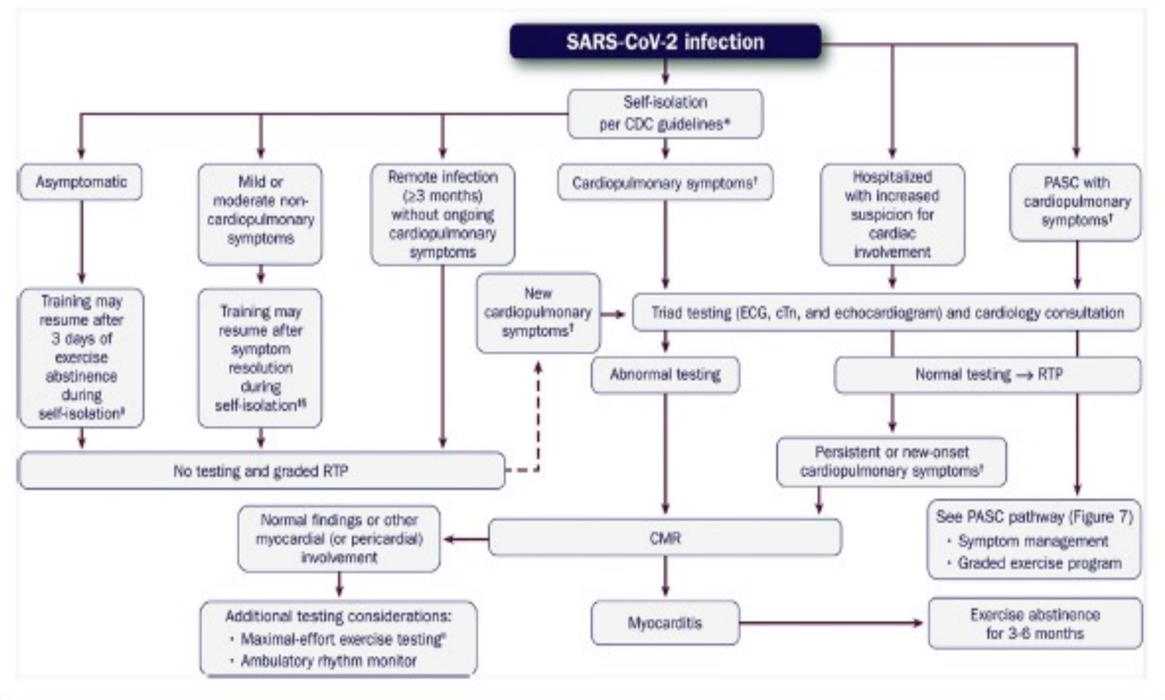
ISSN 0735-1097/$36.00
https://doi.org/10.1016/j.jacc.2022.02.003

FIGURE 9 Evaluation of the Athletic Patient Convalesced From COVID-19 and Guidance on RTP and/or Intense Training



*CDC Guidelines: COVID-19 Quarantine and Isolation.[240]

†Cardiopulmonary symptoms include chest pain/tightness, dyspnea, palpitations, and lightheadedness/syncope; this also includes symptoms occurring ≤1 week following COVID-19 mRNA vaccination.

‡Strategies to minimize transmission of SARS-CoV-2 to other athletes 3-10 days following a positive COVID-19 test include 1) training in isolation, 2) participating in socially-distanced outdoor training, 3) training with a face mask in a well-ventilated facility with appropriate social distancing, and 4) participating in group training after a single negative NAAT (eg, RT-PCR test) or 2 negative rapid antigen tests 24-48 hours apart.

§Excludes prolonged, isolated anosmia/ageusia, which should not delay return to training.

‖Maximal-effort exercise testing should be deferred until myocarditis has been excluded.

CDC = Centers for Disease Control and Prevention; CMR = cardiac magnetic resonance imaging; COVID-19 = novel coronavirus disease 2019, cTn = cardiac troponin; ECG = electrocardiogram; NAAT = nucleic acid amplification test, PASC = post-acute sequelae of SARS-CoV-2 infection; RTP = return to play; RT-PCR = reverse transcription polymerase chain reaction, SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2.

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PROTOCOL

Rationale and Design of the ORCCA (Outcomes Registry for Cardiac Conditions in Athletes) Study

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BACKGROUND: Clinical practice recommendations for participation in sports and exercise among young competitive athletes with cardiovascular conditions at risk for sudden death are based largely on expert consensus with a paucity of prospective outcomes data. Recent guidelines have taken a more permissive approach, using a shared decision-making model. However, the impact and outcomes of this strategy remain unknown.

METHODS: The ORCCA (Outcomes Registry for Cardiac Conditions in Athletes) study is a prospective, multicenter, longitudinal, observational cohort study designed to monitor clinical outcomes in athletes with potentially life-threatening cardiovascular conditions. The study will assess sports eligibility decision-making, exercise habits, psychosocial well-being, and long-term cardiovascular outcomes among young competitive athletes with cardiovascular conditions. Competitive athletes aged 18 to <35 years diagnosed with a confirmed cardiovascular condition or borderline finding with potential increased risk of major adverse cardiovascular events are eligible. Outcomes will be monitored for an initial 5-year follow-up period or until age 35, and metrics of psychosocial well-being and composite adverse cardiovascular events including arrhythmias, sudden cardiac arrest/sudden cardiac death, and evidence of disease progression will be compared among athletes who continue versus discontinue competitive sports participation.

CONCLUSIONS: The ORCCA study aims to assess the process and results of return to sport decision-making and to monitor major adverse cardiovascular events, exercise habits, and the psychosocial well-being among young competitive athletes diagnosed with confirmed cardiovascular conditions or borderline findings with potential increased risk of major adverse cardiovascular events. The results of this work will generate an evidence base to inform future guidelines.

Key Words: athletes ■ cardiovascular disease ■ shared decision making ■ sudden cardiac arrest

The field of sports cardiology developed to provide care for athletic individuals with confirmed or suspected cardiovascular conditions across the age spectrum and at all levels of performance.[1–3] Clinical practice recommendations for sports and exercise eligibility in athletes with cardiovascular conditions have been developed by major societies, including the American Heart Association, American College of Cardiology, and the European Society of Cardiology.[2,3] Although these recommendations, and subsequently clinical practice, have evolved with time to reflect more contemporary management approaches in situations of clinical uncertainty (ie, shared decision-making), the evidence base underpinning these recommendations

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This article was sent to Mahasin S. Mujahid, PhD, MS, FAHA, Associate Editor, for review by expert referees, editorial decision, and final disposition.
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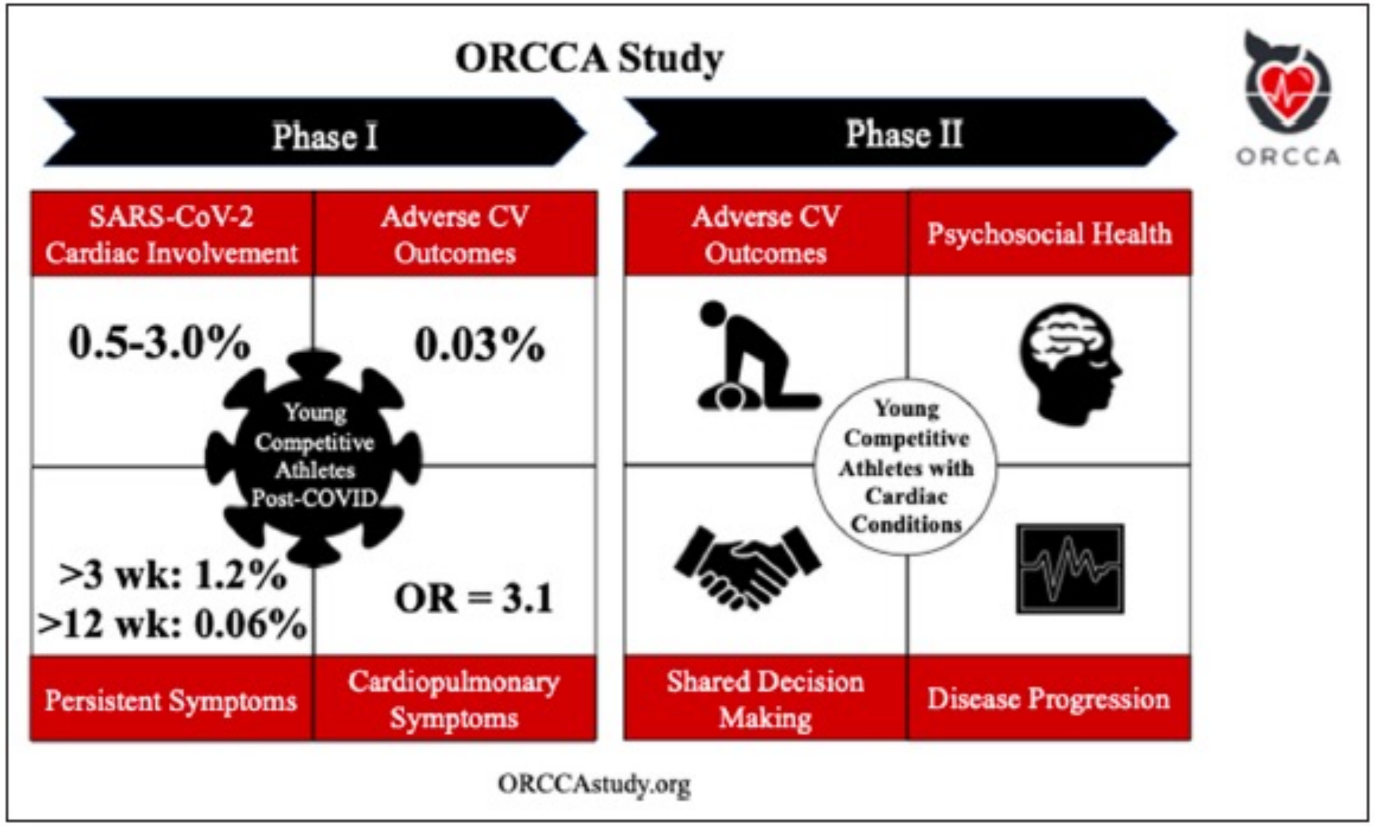

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