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

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IMPORTANCE Cardiac injury with attendant negative prognostic implications is common among patients hospitalized with coronavirus disease 2019 (COVID-19) infection. Whether cardiac injury, including myocarditis, also occurs with asymptomatic or mild-severity COVID-19 infection is uncertain. There is an ongoing concern about COVID-19–associated cardiac pathology among athletes because myocarditis is an important cause of sudden cardiac death during exercise.

OBSERVATIONS Prior to relaxation of stay-at-home orders in the US, the American College of Cardiology's Sports and Exercise Cardiology Section endorsed empirical consensus recommendations advising a conservative return-to-play approach, including cardiac risk stratification, for athletes in competitive sports who have recovered from COVID-19. Emerging observational data coupled with widely publicized reports of athletes in competitive sports with reported COVID-19–associated cardiac pathology suggest that myocardial injury may occur in cases of COVID-19 that are asymptomatic and of mild severity. In the absence of definitive data, there is ongoing uncertainty about the optimal approach to cardiovascular risk stratification of athletes in competitive sports following COVID-19 infection.

CONCLUSIONS AND RELEVANCE This report was designed to address the most common questions regarding COVID-19 and cardiac pathology in athletes in competitive sports, including the extension of return-to-play considerations to discrete populations of athletes not addressed in prior recommendations. Multicenter registry data documenting cardiovascular outcomes among athletes in competitive sports who have recovered from COVID-19 are currently being collected to determine the prevalence, severity, and clinical relevance of COVID-19–associated cardiac pathology and efficacy of targeted cardiovascular risk stratification. While we await these critical data, early experiences in the clinical oversight of athletes following COVID-19 infection provide an opportunity to address key areas of uncertainty relevant to cardiology and sports medicine practitioners.

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Editorial
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In May 2020, the American College of Cardiology's Sports and Exercise Cardiology Section generated recommendations to promote safe return to play (RTP) for athletes involved with competitive sports after severe acute respiratory syndrome coronavirus 2 infection.[1] Motivated by observations of cardiac injury in patients hospitalized with coronavirus disease 2019 (COVID-19),[2] that document[1] and others[3-5] proposed algorithms for preparticipation screening, geared toward the detection of COVID-19–associated cardiac complications. At present, the prevalence and clinical implications of COVID-19 cardiac pathology in athletes are unknown. However, publicized media reports of athletes with suspected COVID-19–induced myocarditis,[6,7] coupled with emerging data documenting cardiac injury among community-based cohorts,[8,9] have fueled concerns about the safety of athletics. The definition of an athlete can also be arbitrary. From enthusiasts of youth sports to masters-level exercise (in those aged >35 years), athletes are generally considered individuals who place a high premium on training, competition, and sports achievement.[10] As such, targeted RTP risk stratification for all athletes deserves careful consideration.

Relaxation of stay-at-home orders across the US enabled many athletes to return to training and competition. In collaboration with the sports medicine community and despite the current lack of supportive data, we have since accrued considerable experience overseeing RTP testing among athletes with prior COVID-19 infection at all levels of sport. This document was written to address the most common questions posed by the media and in clinics, athletic training rooms, and ongoing discussions among cardiologists who participate in the care of athletes. The authors were selected by the Leadership Council of the Sports and Exercise Cardiology Section of the American College of Cardiology to provide this narrative reassessment of the previous consensus statement.

Part 1. COVID-19 Pathology and the Athletic Heart

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 COVID-19 cardiac injury is variable, with inconsistent evidence of direct myocyte viral invasion.[14-16] In cases of mild or asymptomatic COVID-19, the prevalence and clinical implications of pathologic CV sequelae remain unknown.

Reports of presumptive myocarditis among several athletes with high profiles have magnified concerns about COVID-19 CV sequelae in athletes.[6,7] Our combined experience suggests that most athletes with COVID-19 are asymptomatic to mildly ill, and to date, RTP risk stratification has yielded few cases of relevant cardiac pathology. However, we underscore that these observations may not reflect the true prevalence and attendant prognosis of COVID-19 CV involvement in athletes.

Challenges in Determining If COVID-19 Myocardial Injury Is Present in Athletes

At present, there is no widely accepted definition of what constitutes clinically relevant myocardial injury secondary to COVID-19 infection among athletes in competitive sports. Observations made early during the pandemic, stemming from patients hospitalized with COVID-19, suggest that a considerable percentage manifest myocardial involvement, as evidenced by elevated levels of cardiac troponin and abnormalities on noninvasive imaging.[2,13] The presence of cardiac injury among patients hospitalized with COVID-19, largely an older population with preexisting comorbidities, has emerged as an important determinant of prognosis.[17]

These observations have not been replicated in athletes of younger ages in competitive sports. Further, abnormalities that have emerged as markers of COVID-19 cardiac injury may overlap with normal physiology and the established attributes of the athlete's heart.[18] For example, bouts of exercise in individuals in good health often lead to a transient elevation in troponin level and short-term imaging findings suggestive of cardiac fatigue, including myocardial inflammation and tissue edema.[19,20] Similarly, benign consequences of longer-term exercise training, including mild reductions in left ventricular ejection fraction[21] and nonischemic myocardial fibrosis, may further complicate the diagnosis of cardiac injury secondary to COVID-19.[22]

Appropriate Response to Isolated, Mildly Elevated Troponin Levels With a Normal 12-Lead Electrocardiogram and Echocardiogram

Several limitations regarding cardiac troponin testing for RTP risk stratification are noteworthy.[23] First, while high-sensitivity cardiac troponin (hs-cTn) levels allow detection of myocardial injury and are associated with outcomes in patients hospitalized with COVID-19,[24] implications among healthy athletes with asymptomatic or mild disease have yet to be established. Second, 99th-percentile assay values were not derived from athletes and should be used as a rule-in or rule-out criterion with caution.[25] Third, normal hs-cTn reference ranges for athletes, stratified by age and sex, do not exist, which can lead to clinical dilemmas in interpreting borderline or mildly elevated values. Finally, most medical centers in the US still rely on the less sensitive cardiac troponin assays, thus limiting standardization for hs-cTn test results.[26]

Troponin testing to screen for COVID-19 cardiac injury must be performed at least 24 to 48 hours after exercise and should be repeated after a similar period of rest following an isolated abnormality. Persistently elevated troponin levels should prompt detailed characterization of the myocardium with cardiac magnetic resonance imaging (CMR). In the absence of imaging findings consistent with active myocardial inflammation,[27] other systemic causes of elevated troponin must also be considered. Among athletes with persistently elevated troponin levels but normal CMR, there are several considerations that should dictate clinical decision-making. Clinical presentations characterized by systemic and/or CV symptoms indicate a higher pretest probability of clinically relevant myocarditis, thus favoring exercise restrictions for at least 3 months.[28] In contrast, athletes with isolated hs-cTn level elevation but lower pretest cardiac injury probability (because of asymptomatic or mild COVID-19 symptoms) may be considered for more rapid RTP, with close monitoring and gradual escalation of training intensification. The increased sensitivity of hs-cTn testing comes at the cost of reduced specificity for clinically relevant myocardial injury, which may lead to unnecessarily prolonged exercise restrictions. In this scenario, after hs-cTn level normalization, maximal effort–limited exercise testing[29] and extended-duration ambulatory rhythm monitoring are indicated prior to RTP consideration.

Insights From Recent COVID-19 CMR Data

Recent observational CMR data from Puntmann and colleagues[8] suggest cardiac pathology may develop among people with cases of COVID-19 of mild to moderate severity. In a small cohort of 100 patients in Germany who had recovered from COVID-19, most of whom had mild or no symptoms (49% and 18%, respectively), 32% demonstrated late gadolinium enhancement (LGE).[8] However, participants had a mean (SD) age of 49 (14) years, had a clinically significant burden of preexisting comorbidities (hypertension, 22%; diabetes, 18%; intrinsic lung disease, 21%), and in at least 36% of patients, reported ongoing symptoms at the time of CMR.[8] Thus, the study cohort was not an equivalent comparison with athletes who are younger and healthier. A more recent observational data set included only young athletes in competitive sports. In this single-center, cross-sectional case series of athletes in US collegiate sports who were asymptomatic or mildly ill (N = 26) with normal electrocardiogram (ECG) results, hs-cTn levels, and echocardiography results, the authors reported a 15% prevalence (all men; 2 with no symptoms) of CMR findings meeting criteria for myocardial inflammation[27] and 46% prevalence of mild LGE without evidence of active inflammation.[9] However, the absence of an appropriate control population, normative CMR data among young athletes, and details pertaining to study methods render the clinical implemen-

Box. Adapted Myocarditis Summary Taken From "Eligibility and Disqualification Recommendations for Competitive Athletes with Cardiovascular Abnormalities: Task Force 3"[28]

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.

Sports Eligibility Myocarditis Recommendations

1. Before returning to sports, athletes diagnosed with a clinical syndrome consistent with myocarditis should undergo a resting echocardiogram, 24-h Holter monitoring, and an exercise 12-lead electrocardiogram no less than 3 to 6 mo after the illness (class I; level of evidence C).
2. It is reasonable that athletes can resume training and/or competition if all of the following criteria are met (class IIa; level of evidence C):
   A. Ventricular systolic function has normalized.
   B. Serum markers of myocardial injury, heart failure, and inflammation have returned to normal levels.
   C. Clinically relevant arrhythmias on Holter monitor and graded exercise 12-lead electrocardiogram are absent.

tation of these findings uncertain.[9] In addition, the degree to which similar, perhaps completely benign, postinfectious CMR findings develop with common respiratory viral pathogens remains unknown. In aggregate, these data underscore the need for carefully standardized, CMR-based phenotypic studies of athletes with COVID-19 that include appropriate control participants and clinically relevant outcomes.

The Role of CMR in the Diagnosis of Myocarditis

Myocarditis preceded by a viral infection[30,31] is a common causative mechanism of sudden cardiac death in athletes[32] and military personnel[33] and should be considered in the differential diagnosis of athletes with persistent symptoms and prior COVID-19 infection. Symptoms including chest pain, exercise intolerance, ventricular arrhythmias, and abnormal findings on basic testing (ie, cardiac biomarker level elevation, focal or global ventricular systolic function impairment) determine pretest probability and justify the use of CMR to confirm disease.[28,30,34,35] As emphasized in the most recent sports eligibility (**Box**) and CMR myocarditis guidelines,[27,28] CMR is a valuable confirmatory tool in the diagnostic evaluation of athletes with symptoms and moderate to high pretest probability of myocarditis.[34] Cardiac magnetic resonance may confirm the diagnosis based on the specific criteria of nonischemic myocardial injury (an increase on T1-weighted imaging, elevated extracellular volume, or presence of LGE) and edema (on T2-weighted imaging).[27]

Determining the clinical significance of CMR abnormalities (ie, nonspecific LGE or altered T2-weighted or T1-weighted images) that in isolation fail to meet criteria for myocardial inflammation can be challenging. Accordingly, increased CMR use during the COVID-19 pandemic may lead to an increase in clinical referrals for isolated abnormal CMR findings and putative COVID-19 myocarditis. Athletes with low clinical pretest probability of myocarditis and isolated abnormal CMR findings should not be presumed to have myocardial injury attributable to COVID-19. In this clinical scenario, we recommend further risk stratification for inducible ventricular arrhythmias with maximal-effort exercise testing and extended ambulatory rhythm monitoring. If this additional testing is normal, RTP with close clinical monitoring is reasonable.

Whether All Athletes With Positive Test Results for COVID-19 Need CMR Imaging

At present, there are insufficient data to support CMR-based screening of all athletes with suspected or confirmed prior COVID-19 infection. Cardiac magnetic resonance based solely on documented or suspected infection in the absence of symptoms suggestive of myocarditis is not recommended, because it has not been shown to prognosticate clinical outcomes among athletes with COVID-19. We caution against broad and premature application of CMR screening until data justify this approach, based on the following rationale. First, inconsistency in CMR parametric mapping techniques requires proficient studies, which are only performed at experienced centers. Second, there is a dearth of normative CMR data derived from cohorts of young athletes. Third, current observational data lack comparison with appropriate control groups.[9] Finally, the specificity of current CMR myocarditis criteria in an asymptomatic and otherwise healthy population appears to be unknown.[27] Historical lessons learned from the introduction of ECG screening for athletes[36] indicate that application of CMR in the absence of standardized measurements and normative data may yield unacceptable false-positive rates, leading to unnecessary downstream testing and unwarranted medical disqualifications. In accordance with the guidelines,[27,28] CMR is appropriate when pretest probability is high based on a clinical syndrome suggestive of myocarditis coupled with isolated or combined objective pathologic criteria (Box) or recurrence of CV symptoms or new exercise intolerance during a graded resumption in exercise training.

Whether the Risks of Cardiac Injury to Athletes With COVID-19 Warrant Cancellation of a Sport or Sports Season

While concerns about the implications of cardiac injury attributable to COVID-19 infection deserve further study, they should not constitute a primary justification for the cancellation or postponement of sports. Screening for RTP, as delineated in the original American College of Cardiology Section report,[1] was designed to identify athletes at high risk after COVID-19 infection for subsequent clinical management, including potential sports restriction. Rather than canceling sports because of unsubstantiated concerns about cardiac safety based on limited data of unestablished clinical relevance,[8,9] this decision should be driven by the need to limit viral spread. With uncontrolled community transmission, we share concerns with public health officials about risks of increased disease transmission attributable to the resumption of organized sports. Accordingly, the decision to proceed with or delay organized sports should be based on community disease prevalence, coupled with the availability of resources that can be responsibly allocated to identify and prevent new infections among athletes.

Part 2. Updates on Expert Consensus COVID-19–Associated CV Considerations for RTP for Athletes

How Health Care Resources Might Be Considered in a Balanced Approach to RTP

The implementation of screening algorithms requires consideration of available health care resources and acknowledgment that health care disparities are affected by social determinants.[37] Among athletes, barriers to accessing appropriate care are driven in part by geography, social background, and demographics. The sheer numbers of athletes in recreational and competitive sports in the US appropriately call into question the logistic feasibility and financial expenditures of COVID-19 cardiac screening, particularly in the absence of clinical outcomes data. Early experiences within the sports cardiology community suggest that nearly all athletes, particularly those who have completely recovered from mild COVID-19 infection, do not develop clinically significant COVID-19 CV pathology. Prevalence and outcomes data will enable the refinement of RTP risk stratification protocols in a manner that balances clinical necessity with the realities of finite resource availability.

Recommendations for Athletes in Competitive Sports Who Have COVID-19 But Remain Asymptomatic

We do not advocate for CV risk stratification among athletes who remain completely asymptomatic with prior COVID-19 infection, following completion of US Centers for Disease Control and Prevention (CDC) guided self-isolation.[38] Given the current lack of published data, consideration of comprehensive screening for this population could be reasonable if it is based on research and data collection.

Appropriate RTP Approach for High School Athletes Infected With COVID-19

Risks of COVID-19 infection differ between pediatric and adult populations.[39,40] Most children of high school age who are infected with COVID-19 experience only mild symptoms or remain asymptomatic, and the overall risk of myocarditis prior to the COVID-19 pandemic among children is low.[41] However, an exceedingly small number of individuals younger than 21 years may develop a multisystem inflammatory syndrome (MIS-C) several weeks after infection.[39,40] While the pathogenesis of MIS-C remains unclear, a multicenter analysis of 186 patients revealed a median age of 8.3 years and that 73% were healthy prior to COVID-19 infection.[40] Among athletes hospitalized with MIS-C who recover, RTP risk stratification as delineated by the original American College of Cardiology algorithm is warranted.[1]

Members of this writing group routinely evaluate athletes in high school who have completed or nearly completed puberty (generally ≥15 years old). For those younger than 15 years recovering from moderate to severe COVID-19 infection, we recommend formal evaluation with general pediatrics (or pediatric cardiology) prior to RTP to determine the need for CV risk stratification. To date, for high school athletes 15 years or older, there has been no compelling evidence of clinically relevant CV pathology following asymptomatic to mild COVID-19 infection. Thus, we recommend, in the absence of systemic symptoms or persistent CV complaints, CV risk stratification is unnecessary after CDC-recommended self-isolation.[38] However, for athletes in this age group with systemic or CV symptoms during or after infection,[42] we recommend a similar approach to athletes of older ages in competitive sports and close observation for MIS-C (**Figure 1**). In our opinion, isolated ECG screening after COVID-19 infection is of limited value because of the limited sensitivity for the detection of myocarditis (47%).[43]

The Appropriate RTP Approach for Masters-Level Athletes With COVID-19

Aging athletes harbor traditional CV risk factors and established forms of disease,[44,45] and athletes at the masters level constitute most patients seen in sports cardiology clinics. At present, CV sequelae and outcomes among athletes in masters-level endurance sports with prior COVID-19 infection remain unknown. Taking into account the logistics required for widespread CV screening of athletes at the masters level, coupled with the anticipated low risk of clinically significant cardiac injury in the context of mild infection, routine RTP CV assessment in this population is not recommended. However, masters athletes older than 65 years, particularly those with preexisting CV conditions (eg, hypertension, coronary artery disease, atrial fibrillation, diabetes) and those with persistent symptoms may benefit from risk stratification.[11] Similarly, those with moderate to severe prior COVID-19 infection should be evaluated by a cardiologist for consideration of RTP risk stratification. An approach to individualized RTP risk stratification for athletes at the masters level is presented in **Figure 2**.

Reconciling Focal LGE of Right Ventricular Septal Insertion Points in Masters Athletes With COVID-19 Infection

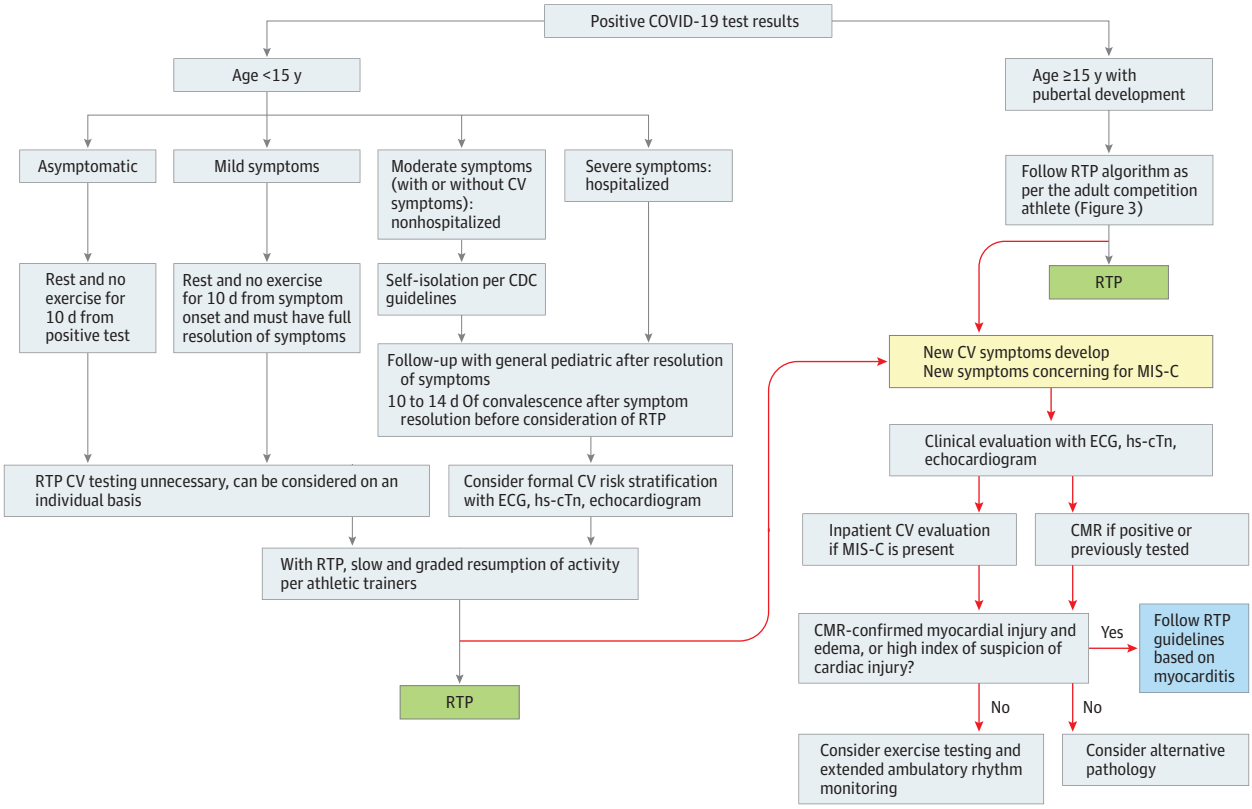
Ultra-endurance exercise imparts a substantial hemodynamic load on the right ventricle (RV) because of high cardiac output and proportionately greater increase in RV afterload compared with the left ventricle.[46] It has been hypothesized that this physiology may lead to maladaptive structural RV remodeling based on the finding of incidental fibrosis, defined by CMR LGE of the interventricular septum and RV septal insertion points, among a minority of athletes in ultra-endurance sports.[20,47] However, the clinical significance of this observation remains uncertain, and to date (and to our knowledge), there are no data indicating that incidentally detected LGE in this pattern is associated with adverse outcomes in athletes at the masters level.

As use of CMR increases during the pandemic, this LGE narrative provides another cautionary tale for screening in the absence of diagnostic specificity and established clinical prognosis. We anticipate that this nonspecific LGE pattern will be scrutinized as suggestive evidence of myocarditis. In the absence of a clinical presentation suggestive of myocarditis and CMR findings suggestive of active injury and edema, we recommend not ascribing this finding to COVID-19 and allowing gradual escalation in endurance training.

Adjustments to the Original Consensus RTP Algorithm for the Competitive Athlete

Self-isolation has been reduced from 14 to 10 days from the time of documented infection per CDC guidelines.[38] As such, we believe it is reasonable to reduce complete exercise abstinence in cases of asymptomatic COVID-19 infection to 10 days from the date of the positive test result (**Figure 3**). Following this restriction, a slow and carefully monitored resumption of activity, ideally under the direction of a certified athletic trainer, is appropriate.

Figure 1. Proposed Coronavirus Disease 2019 (COVID-19) Return-to-Play Algorithm for Athletes in Competitive High School Sports



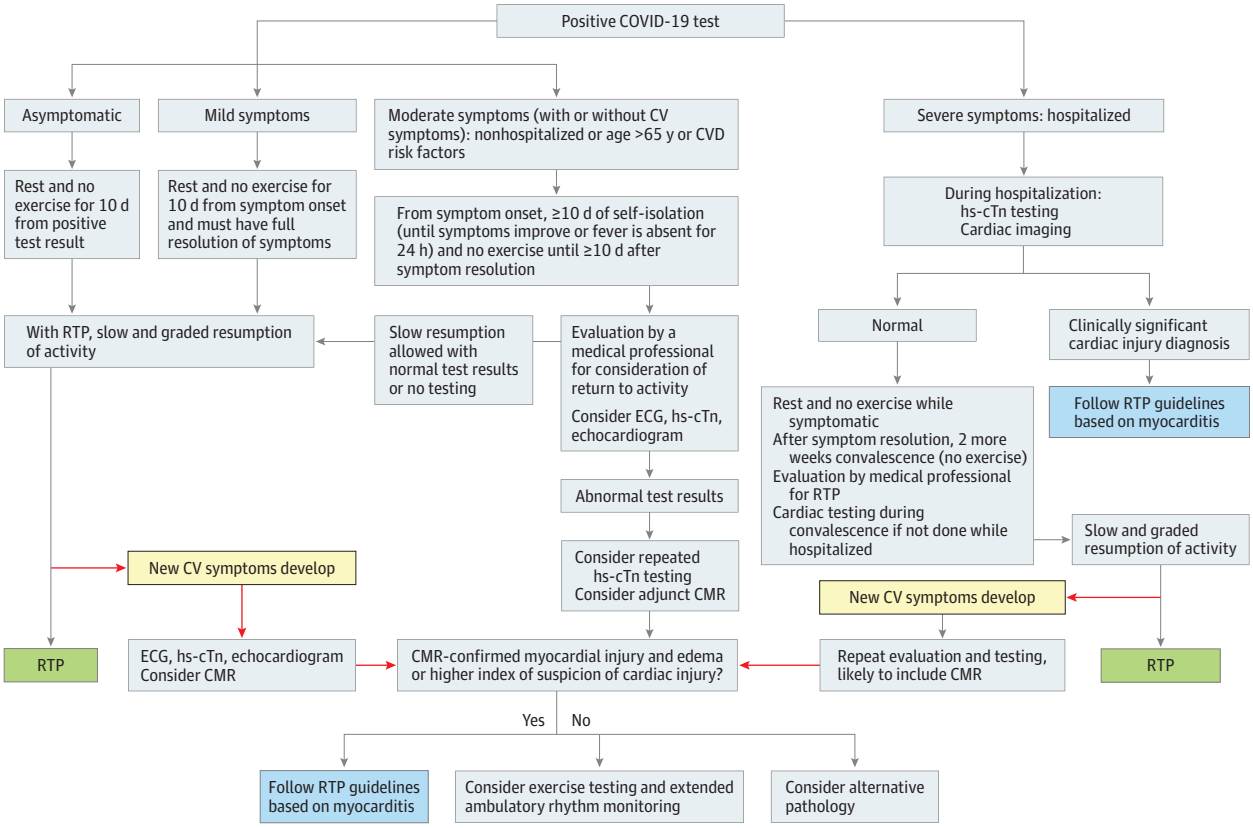
All high school athletes who are asymptomatic with no history of COVID-19 and negative test results for COVID-19 are cleared for athletic participation. The proposed algorithm is for those with confirmed COVID-19. Note that among the cardiovascular (CV) symptoms, syncope of unclear causative mechanism (ie, presumptive cardiogenic syncope) identifies individuals who definitely require advanced CV testing, including cardiac magnetic resonance (CMR) imaging, exercise testing, and extended rhythm monitoring. Typical initial testing is obtained via a nasopharyngeal swab and polymerase chain assay for conserved regions of severe acute respiratory syndrome coronavirus-2 RNA. Mild symptoms include anosmia, ageusia, headache, mild fatigue, mild upper respiratory tract illness, and mild gastrointestinal illness; moderate symptoms include persistent fever, chills, myalgias, lethargy, dyspnea, and chest tightness; CV symptoms include dyspnea, exercise intolerance, chest tightness, dizziness, syncope, and palpitations; multisystem inflammatory syndrome in children (MIS-C) involves fever, rash, abdominal pain, vomiting, diarrhea, lethargy, and conjunctivitis, possibly developing weeks after infection. Although full understanding of COVID-19 cardiac pathophysiology remains uncertain, where following RTP guidelines based on myocarditis is indicated, follow American College of Cardiology/American Heart Association athlete myocarditis guidelines (Box). CDC indicates the US Centers for Disease Control and Prevention; ECG, 12-lead electrocardiography; echo, echocardiogram; hs-cTn, high-sensitivity cardiac troponin-I; RTP, return to play.

For positive antibody test results in athletes without symptoms, formal CV risk stratification is likely to be low yield and thus not recommended. In athletes with positive antibody test results and a prior history of COVID-19 symptoms, consideration of CV risk stratification can be similar to positively identified cases of COVID-19.

In athletes infected with COVID-19 with mild symptoms that completely resolve during 10 days of self-isolation after a positive test result or symptom onset,[38] RTP CV risk stratification appears to be low yield. Accordingly, we do not advocate CV RTP risk stratification among athletes in competitive sports with mild, self-limited disease. However, CV testing should be considered on an individualized basis for athletes with protracted symptoms (≥10 days). Among all athletes with COVID-19, regardless of symptom severity, a gradually escalating approach to training is recommended. The optimal duration of this process remains to be defined and will likely vary. Severity and duration of infection coupled with baseline fitness levels and short-term athletic goals may be used to develop individualized plans. If symptoms develop, comprehensive CV evaluation, as recommended following moderate or severe COVID-19 infection, is recommended.

For athletes with prior moderate or severe COVID-19 infection, we recommend comprehensive CV risk stratification. The association between severe COVID-19 infection and cardiac injury[2,12] justifies the rationale to continue with this empirical strategy while we await forthcoming prevalence and clinical outcomes data. Testing should include a clinical evaluation, ECG, hs-cTn (or available cTn) level test, and echocardiography. Further testing may include CMR, exercise testing, and extended-duration ambulatory rhythm monitoring, if baseline test results are abnormal or inconsistent with normal athletic findings or symptoms persist or recur. Importantly, the presence of cardiogenic syncope particularly identifies individuals at high risk who require more advanced testing beyond the initial screening test results. If clear cardiac involvement is diagnosed, despite the lack of well-defined myocarditis pathophysiology after COVID-19 infection, RTP should be based on current myocarditis guidelines (Figure 3).[28,35]

Figure 2. Proposed Coronavirus Disease 2019 (COVID-19) Return-to-Play Algorithm for Recreational Masters Athletes



All athletes at the masters level who are asymptomatic, have no history of COVID-19, and have negative test results for COVID-19 are cleared for athletic participation. The proposed algorithm is for those with confirmed COVID-19. Note that among the cardiovascular (CV) symptoms, syncope of unclear causative mechanism (ie, presumptive cardiogenic syncope) identifies individuals who definitely require advanced CV testing, including cardiac magnetic resonance (CMR) imaging, exercise testing, and extended rhythm monitoring. Typical testing is obtained via a nasopharyngeal swab and polymerase chain reaction assay for conserved regions of severe acute respiratory syndrome coronavirus 2 RNA. Symptom differentiation is as in Figure 1. Cardiovascular disease (CVD) risk factors include hypertension, coronary artery disease, atrial fibrillation, and diabetes. Although full understanding of COVID-19 cardiac pathophysiology remains uncertain, where following RTP guidelines based on myocarditis is indicated, follow American College of Cardiology/American Heart Association athlete myocarditis guidelines (Box). ECG indicates 12-lead electrocardiography; echo, echocardiogram; hs-cTn, high-sensitivity cardiac troponin-I; RTP, return to play.

Part 3. Future Directions and the Path Forward

Pressing Gaps in Knowledge and Areas of Research

At present, there is a shared focus in the sports medicine and cardiology communities to define the prevalence of clinically significant cardiac injury in athletes infected with COVID-19 and determine the efficacy of current consensus-based CV risk stratification practices. Prospective acquisition of comprehensive large scale registry data at the collegiate and professional levels, coupled with surveillance for adverse clinical outcomes, will be required to address these areas of uncertainty. Carefully standardized phenotyping studies, using advanced imaging modalities, will enhance our understanding of COVID-19 disease pathogenesis and facilitate a data-driven approach to CMR in the risk stratification of athletes with COVID-19.

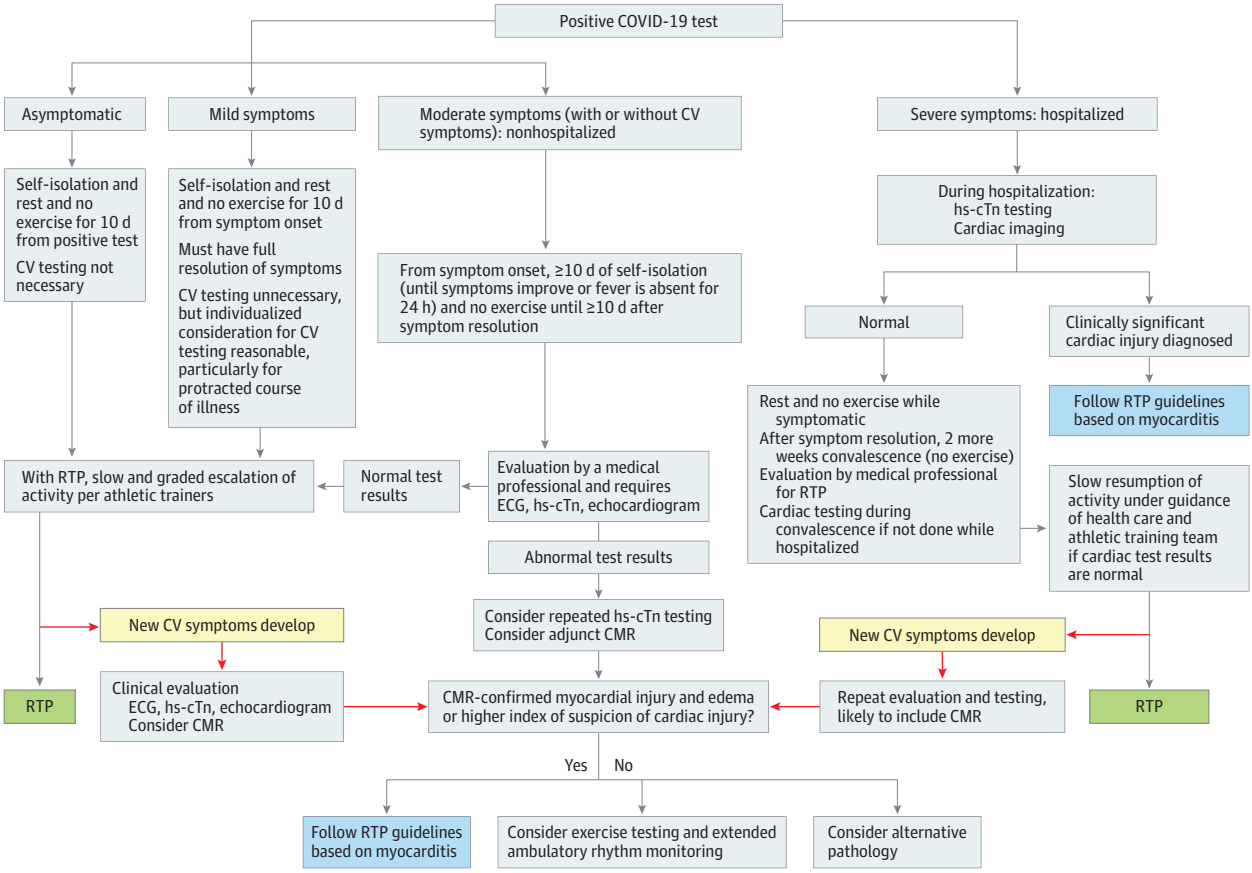
Shared Decision-Making as Applied to COVID-19 and RTP

Increases in COVID-19 CV screening is leading to more athletes with gray-zone findings of unclear clinical relevance, which translates into a growing number of athletes being considered for medical disqualifications. Shared decision-making has emerged as the foundational framework of the contemporary sports eligibility discussion.[48,49] This patient-centered approach is predicated on the notion that sports eligibility decisions should not be driven solely by clinicians in the absence of quantifiable risk. In the wake of COVID-19, isolated abnormal findings, such as mildly elevated hs-cTn levels or nonspecific imaging findings, will require careful clinical consideration involving shared decision-making to strike balance between the clinician's estimation of risk and the patient's tolerance for risk assumption, with input from relevant stakeholders. It is our opinion that athletes with gray-zone cardiac findings following COVID-19 infection deserve comprehensive clinical risk stratification coupled with transparent shared decision-making as a means to establish sports eligibility.

Conclusions

The dynamic nature of the COVID-19 pandemic has placed considerable stress on sports medicine and sports cardiology practitioners. As we collectively await critical data to advance our under-

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



All adult athletes in competitive sports who are asymptomatic with no history of COVID-19 and negative test results for COVID-19 are cleared for athletic participation. The proposed algorithm is for those with confirmed COVID-19. Note that among the cardiovascular (CV) symptoms, syncope of unclear causative mechanism (ie, presumptive cardiogenic syncope) identifies individuals who definitely require advanced CV testing, including cardiac magnetic resonance (CMR) imaging, exercise testing, and extended rhythm monitoring. Typical COVID-19 testing was obtained via a nasopharyngeal swab and polymerase chain reaction assay for conserved regions of severe acute respiratory syndrome coronavirus 2 RNA. Symptom differentiation is as in Figure 1. Although full understanding of COVID-19 cardiac pathophysiology remains uncertain, where following RTP guidelines based on myocarditis is indicated, follow American College of Cardiology/American Heart Association athlete myocarditis guidelines (Box). ECG indicates 12-lead electrocardiography; echo, echocardiogram; hs-cTn, high-sensitivity cardiac troponin-I; RTP, return to play.

standing of the outcomes of COVID-19 in athletes, it is of paramount importance that we do not lose sight of the value of thoroughly rehearsed emergency action planning that ensures timely response to the athlete who has collapsed with immediately available automated external defibrillation therapy. Through all stages of the COVID-19 pandemic and beyond, risk of adverse CV outcomes during athletics will persist despite best CV screening practices, and emergency action planning initiatives represent our best strategy to save lives. To proceed safely with sports during the COVID-19 pandemic, the critical pieces on which we must focus have not changed. An emphasis on public health, suppression of viral spread, increased access to testing, and ultimately vaccination should all be prioritized. These foundational public health mandates, coupled with dedication to the emergency action planning and collaborative science, are all required to protect the athletic heart.

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Concept and design: All authors.
Acquisition, analysis, or interpretation of data: Kim, Thompson.
Drafting of the manuscript: Kim, Phelan, Martinez.
Critical revision of the manuscript for important intellectual content: Kim, Levine, Emery, Martinez, Chung, Thompson, Baggish.
Administrative, technical, or material support: Kim, Emery.
Supervision: Kim, Levine, Emery, Chung, Baggish.

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