Cardiac Considerations:

Cardiac screening, Cardiac Conditions and COVID RTP
Sports Cardiology Clinic: Our team

CAMDEN HEBSON, MD
Department of Cardiology

SARA GOULD, MD
Department of Orthopedics

JENNIFER WALLACE, ATC
Clinic Coordinator
Objectives

1. Understand the sports cardiology components of a pre-participation physical

2. Describe medical conditions that can result in sudden cardiac death

3. Know which tests should be ordered for further cardiac work up, including post-COVID
Pre-participation Sports Screening in Pediatrics

“How to Properly Dig Up Landmines”
Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.

2. Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes

3. Pre-participation Screening in the Time of COVID
Background

◆ Sudden cardiac death (SCD) in a young, previously “healthy” athlete is rare but devastating – family, school, community, etc.

◆ Some events are unexplained, however many harbor undiagnosed cardiovascular disease, with the end result being VT/VF → SCD
Background

In patients with certain CV conditions, athletics increases the risk of VT/VF in 2 theoretical ways:

1. In those with abnormal cardiac substrate, physical training can lead to maladaptive changes in cardiac structure (intracardiac fibrosis, dilation of RV/LV) → ↑arrhythmia risk

2. The demands of intense athletics (hemodynamic stress, catecholamine release, electrolyte imbalance) can trigger arrhythmias in susceptible individuals
How Should We Screen?
U.S.A. Guidelines

Emphasis on 14 point H&P screening

Routine EKG NOT recommended

- Limitations due to false (+)s – time lost, $, family angst, etc
- U.S. : approximately 4 million high school athletes
- Diverse population: “We ain’t Italy”
- Even AFTER many diagnoses, uncertainty re: restricting sports/physical activity


Personal History

1. Exertional chest pain/discomfort
2. Exertional syncope or near-syncope
3. Excessive exertional and unexplained fatigue/fatigue associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing of the heart ordered by a physician

Family history

8. Premature death-sudden/unexpected < 50 yr due to heart disease, in a relative
9. Disability from heart disease in a close relative < 50 yr
10. Specific knowledge of certain cardiac conditions in family members: HCM, DCM, LQTS, other channelopathies, Marfan, other arrhythmias

Physical exam

11. Heart Murmur-exam supine and standing or with yalsalva, specifically to identify murmurs of dynamic L ventricular outflow tract obstruction
12. Femoral pulses to exclude aortic stenosis
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting, preferably taken in both arms)
European Guidelines
◆ Biggest difference from U.S. guidelines is that additional EKG screening is RECOMMENDED
◆ Why different? Chiefly data from Italy
  ◆ National EKG screening program since 1982 (data to be discussed)

So, How Effective is Screening Anyway?

◆ Limited data on efficacy

◆ Prevalence of abnormal EKG findings?
  ◆ N = 32,652 Italians who underwent routine pre-participation screening that included an ECG
  ◆ The prevalence of an abnormal EKG was 11%, and of markedly abnormal ECG pattern < 5%

◆ Most frequent abnormal findings: prolonged PR interval, incomplete right bundle branch block (RBBB) and early repolarization pattern

◆ More likely abnormal findings: deep inverted T-waves (2.3%), LVH (0.8%), RBBB (1.0%), left anterior fascicular block (0.5%), LBBB (0.1%), pre-excitation pattern (0.1%) and prolonged QTc interval (0.03%)

So, How Effective is Screening Anyway?

Other European Perspectives*

N = 11,168 adolescent English athletes (mean age 16.4 years)
Underwent EXTENSIVE cardiac screening (including H&P, ECG, and echo)

N = 42 athletes (0.4 percent) were found to have disorders associated with sudden cardiac death

N = 8 athletes with a negative screen STILL suffered SCD (5 from a cardiomyopathy)
  • SCD at a mean of 7 years after initial screen
  • Six of the 8 had a normal initial screen, but no follow-up → need for serial assessment

What is the Impact of Screening?

◆ Data from Italy, again

Annual incidence of SCD in athletes was 3.6/100,000 person-years from 1979 to 1980

◆ H&P + EKG screening instituted nationwide in 1982

◆ SCD incidence dropped to 0.4/100,000 person-years in 2003 to 2004 (89 percent reduction)

Presumably due to H&P + EKG screening

Notably, there was no change in the incidence of SCD among non-athletes over the same time period.

Incidence of SCD in U.S. Athletes

Best overall estimate: 1:50,000 to 1:100,000 per year

Similar odds to dying from a lightning strike

Males higher risk than females, college > high school, African-American > others

---


---

**Figure 6** Incidence rate ratios (IRR) of sudden cardiac arrest and death in select athlete populations compared with all high school male athletes. DI, division I; NCAA, National Collegiate Athletic Association.
Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.

2. Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes

3. Pre-participation Screening in the Time of COVID
<table>
<thead>
<tr>
<th>Structural/Functional</th>
<th>Electrical</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Hypertrophic Cardiomyopathy (HCM)</td>
<td>11) Long QT Syndrome (LQTS)</td>
<td>17) Drugs and Stimulants</td>
</tr>
<tr>
<td>2) Coronary Artery Anomalies</td>
<td>12) Wolff-Parkinson-White Syndrome (WPW)</td>
<td>18) Primary Pulmonary Hypertension (PPH)</td>
</tr>
<tr>
<td>3) Aortic Rupture/Marfan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4) Dilated Cardiomyopathy (DCM)</td>
<td>13) Brugada Syndrome</td>
<td>19) Commotio Cordis</td>
</tr>
<tr>
<td>5) Myocarditis</td>
<td>14) Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)</td>
<td></td>
</tr>
<tr>
<td>6) Left Ventricular Outflow Tract Obstruction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) Mitral Valve Prolapse (MVP)</td>
<td>15) Short QT Syndrome</td>
<td></td>
</tr>
<tr>
<td>8) Coronary Artery Atherosclerotic Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9) Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Etiology of SCD in Young Athletes

The most common structural heart diseases leading to SCD:

- Hypertrophic cardiomyopathy (HCM)
- Anomalous origin of a coronary artery

Study*: Among 1435 young U.S. athletes in a SCD registry, on post-mortem exam, the most common diagnoses were HCM (36%) and anomalous origin of a coronary artery (20%)

Six million U.S. military recruits (mean age 19) 126 nontraumatic sudden deaths occurred

N = 108 [86%] deaths were related to exercise
- 33 % Anomalous origin of a coronary artery
- 20% myocarditis
- 16% coronary atherosclerosis
- 13% HCM

Etiology of SCD in H.S. and College U.S. Athletes, 2014-2018

Figure 2  Aetiology of sudden cardiac arrest and death (n=209).
Hypertrophic Cardiomyopathy

- Relatively common! 1:500 individuals in the general population
- The most common structural heart cause of SCD in athletes
  - Figure: 2007 AHA statement, showing results from the Minneapolis Heart Institute registry of causes of SCD in athletes from 1980-2005
  - HCM or POSSIBLE HCM = 44% of all causes
- In most athletes with SCD due to HCM, the diagnosis was not previously established
Hypertrophic Cardiomyopathy

- **Pathology:** gene mutation(s) in different proteins involved in the cardiac sarcomere (50% with identifiable gene)
  - Disorganized, fibrotic, and hypertrophied myocardium
  - Prone to arrhythmias
  - **Additional problems:** LV outflow obstruction, diastolic dysfunction, MR, heart failure

- **Autosomal dominant** inheritance (vs. de novo mutation) → Family history!!

- **Typical onset of clinically significant findings** (EKG, echo) → during puberty

- SCD can be initial presentation
<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Mechanism</th>
<th>Effect on gradient and murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valsalva (strain)</td>
<td>Decreased LV cavity, pre and after load</td>
<td>Increased</td>
</tr>
<tr>
<td>Standing</td>
<td>Decreased LV cavity, preload</td>
<td>Increased</td>
</tr>
<tr>
<td>Squatting</td>
<td>Increased LV cavity, pre and after load</td>
<td>Decreased</td>
</tr>
<tr>
<td>Isometric hand grip</td>
<td>Increased afterload</td>
<td>Decreased</td>
</tr>
</tbody>
</table>
# Effect of selected maneuvers

<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Mechanism</th>
<th>Effect on gradient and murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valsalva (strain)</td>
<td>Decreased LV cavity, pre and after load</td>
<td>Increased</td>
</tr>
<tr>
<td>Standing</td>
<td>Decreased LV cavity, preload</td>
<td>Increased</td>
</tr>
<tr>
<td>Squatting</td>
<td>Increased LV cavity, pre and after load</td>
<td>Decreased</td>
</tr>
<tr>
<td>Isometric hand grip</td>
<td>Increased afterload</td>
<td>Decreased</td>
</tr>
</tbody>
</table>
Hypertrophic Cardiomyopathy and Sports Clearance

**Old school**: not cleared for sports, recreational/low static/dynamic sports only

**Trend toward clearance of more HCM patients for more types of exercise** → don’t turn ALL of these patients into couch potatoes!

**Highest Risk Patients**: proceed with caution!

1. Family history of SCD
2. Super severe LVH – IVS ≥ 30 mm
3. Sustained VT on Holter monitor
4. Non-vasovagal syncope
5. Decrease BP with exercise, or failure to augment
Coronary Anomalies

182 Sudden deaths in NCAA athletes from 2002-2011

- HCM (21)
- Coronary artery anomaly (8)
- CAD (6)
- Aortic rupture (3)
- ARVC (3)
- Dilated CM (2)
- Myocarditis (2)
- LQTS (1)
- MVP (1)
- AMI [Kawasaki Syndrome] (1)

Coronary Anomalies

◆ Incidence: 0.64% of live births*

◆ Anomalous Left Coronary Artery from the Opposite Sinus is most common and when found is an indication for surgery!

◆ Anomalous Right Coronary Artery from the Opposite Sinus is less common and controversial in terms of management

◆ Other variations:
  • Single coronaries (often left alone)
  • Anomalous origin from the pulmonary artery (typically symptomatic as infants)
  • Ectopic origin from the aorta
  • Myocardial bridges

Coronary Anomalies

◆ **Presentation**: asymptomatic until it is not!

◆ **Chest pain with exercise** – can’t rule out w/o imaging

◆ **Treatment**: Surgery
Marfan Syndrome
Arrhythmogenic RV Dysplasia (ARVD)

◆ Generalized cardiomyopathy; principally RV fibrofatty replacement and progressive dysfunction
  ▪ Prevalence: 1:3000 adults
  ▪ Cause for up to 11% of SCD in young adults

◆ ARVC mutational genes most commonly encode desmosomal proteins

**Arrhythmogenic RV Dysplasia (ARVD)**

**Symptoms:** exercise-associated syncope/palpitations/VT/SCD
- Family history – AD form is most common
- But, 25-40% of patients are asymptomatic at time of diagnosis

Rarely diagnosed before puberty; mean age at diagnosis ~ 30 years

EKG changes

Diagnosis

**Long QT syndrome**

- Problem with the way electricity travels through the heart, which can result in disorganized electrical conduction (ventricular tachycardia)
- Diagnosed by a long interval between the Q and T segments on an EKG
- A “normal” Q on a single resting EKG does **not** exclude the diagnosis
Long QT syndrome

◆ Normal QTc < 440

◆ Borderline QTc
  • 440 - 460 ms men
  • 440 - 470 ms women
Long QT syndrome

- EKG
  - Prolonged QTc
  - T-wave changes
  - Torsades de Pointes
- Other things that can cause long QT interval – drugs, electrolytes (K, Mg, Ca), acidosis
Brugada syndrome

- **Autosomal dominant** arrhythmia syndrome characterized by risk of SCD and an abnormal EKG
- **EKG** – “pseudo-right bundle branch” pattern with ST segment elevation in leads V1/V2 (coved pattern). TWI V1-3
- Most common gene mutation is in **SCN5A** (Na channel, loss of function)
Type 1: Coved type
ST-segment elevation

Type 2: Saddle-back type
ST-segment elevation

Type 3: Saddle-back type
“ST-segment elevation”
Brugada syndrome

Typical presentation at older patient age (third to fourth decade)

Rare pediatric presentation
  • Syncope
  • Febrile seizure
  • SCD can be the first presentation
  • Family history of Brugada syndrome, syncope, SCD

Precipitating events → fever and sleep

Treatment
  • Fever treatment
  • Med avoidance - www.brugadadrugs.org
  • ICD
Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Cause
Inherited genetic code for abnormal protein which allows calcium to leak in the heart. This interferes with electrical conduction

Clinical
Young patient age
**Exercise/emotion-induced** seizure/syncope (can be misdiagnosed as epilepsy)
Drowning/near drowning
Family history of CPVT, seizure/syncope/SCD

EKG
- Normal resting EKG
- Exercise-induced bidirectional VT
CPVT

Treatment

• Avoiding competitive sports
• Beta blockade
• ICD

Heart Rhythm. 2018
Wolff-Parkinson-White Syndrome

Cause

• Extra electrical pathway through the heart
• Can result in a very rapid heart rate
• Short PR <120 ms, delta wave, prolonged QRS >120 ms
Wolff-Parkinson-White Syndrome

Evaluation
• Exercise EKG
• EP study
• Echocardiogram (Ebstein’s anomaly and cardiomyopathy)
Delta wave
Commotio Cordis

International Criteria

The “normal” athlete’s heart

- Guidelines to identify EKG changes that occur with physical conditioning
- Eliminate unnecessary further testing
Physiologic Cardiac Adaptation: ‘Athlete’s Heart’

- Type of Sport
- Age
- Gender
- Size
- Race/Genetics

- Increased Vagal Tone

- Enlarged Chamber Size
  - Wall thickness
  - Cavity dimension

- Sinus bradycardia
- Sinus arrhythmia
- Early repolarization
- 1° AVB
- Mobitz Type 1 2° AVB

- LVH voltage criteria
- Incomplete RBBB
International criteria for electrocardiographic interpretation in athletes


This statement has been endorsed by the following societies: American Medical Society for Sports Medicine (AMSSM), Austrian Society of Sports Medicine and Prevention, Brazilian Society of Cardiology – Department of Exercise and Rehabilitation (SBC – DER), British Association for Sports and Exercise Medicine (BASEM), Canadian Academy of Sport and Exercise Medicine (CASEM), European College of Sports Physicians (ECOSEP), European Society of Cardiology (ESC) Section of Sports Cardiology, Fédération Internationale de Football Association (FIFA), German Society of Sports Medicine and Prevention, International Olympic Committee (IOC), Norwegian Association of Sports Medicine and Physical Activity (NIMF), South African Sports Medicine Association (SASMA), Spanish Society of Cardiology (SEC) Sports Cardiology Group, Sports Doctors Australia, and the Swedish Society of Exercise and Sports Medicine (SFAIM). The American College of Cardiology (ACC) affirms the value of this document. ACC supports the general principles in the document and believes it is of general benefit to its membership.
International Criteria for ECG Interpretation in Athletes

Normal ECG Findings
- Increased QRS voltage for LVH or RVH
- Incomplete RBBB
- Early repolarization/ST segment elevation
- ST elevation followed by T wave inversion V1-V4 in black athletes
- T wave inversion V1-V3 ≤ age 16 years old
- Sinus bradycardia or arrhythmia
- Ectopic atrial or junctional rhythm
- 1° AV block
- Mobitz Type I 2° AV block

Abnormal ECG Findings
- T wave inversion
- ST segment depression
- Pathologic Q waves
- Complete LBBB
- QRS ≥ 140 ms duration
- Epsilon wave
- Ventricular pre-excitation
- Prolonged QT interval
- Brugada Type 1 pattern
- Profound sinus bradycardia < 30 bpm
- PR interval ≥ 400 ms
- Mobitz Type II 2° AV block
- 3° AV block
- ≥ 2 PVCs
- Atrial tachyarrhythmias
- Ventricular arrhythmias

Borderline ECG Findings
- Left axis deviation
- Left atrial enlargement
- Right axis deviation
- Right atrial enlargement
- Complete RBBB

No further evaluation required in asymptomatic athletes with no family history of inherited cardiac disease or SCD

Further evaluation required to investigate for pathologic cardiovascular disorders associated with SCD in athletes

In isolation
2 or more
Normal Variant: Repolarization Changes in Black/African Athletes

ECG from a 24 year old asymptomatic black/African soccer player demonstrating J-point elevation, convex ('domed') ST elevation followed by T-wave inversion in leads V1-V4 (circles). This is a normal repolarization pattern in black/African athletes.
Brugada – abnormal
Excavation Plan

1. “Lay of the Land” – Background, Incidence, Guidelines, etc.

2. Particularly Bad Landmines – Causes of Sudden Cardiac Death in Young Athletes

3. Pre-participation Screening in the Time of COVID
COVID Guidelines: A Journey

Histrionic initial reactions

Data gathering

Current state
What is the worry about COVID and the heart?
Myocarditis – inflammatory disease of the cardiac muscle, often viral in etiology

- Presentation is variable – from fulminant to subclinical
- In pediatrics, fulminant patients can often make a quick and full recovery

Definitive diagnosis requires biopsy (Dallas Criteria) showing inflammatory cell infiltration with myocyte damage

- Cardiac MRI has become an accepted surrogate marker, with specific criteria being more accepted in adults > children
- Lake Louise criteria (involving T1/T2 mapping and LGE)

Clinical myocarditis – signs of heart failure associated with abnormalities on EKG/Echo/MRI and laboratory signs of inflammation and myocardial injury (elevated CRP/troponin/BNP)
How do COVID-19, myocardial injury, and MIS-C relate?
Outcomes of Cardiovascular Magnetic Resonance Imaging in Patients Recently Recovered From Coronavirus Disease 2019 (COVID-19)

Valentina O. Puntmann, MD, PhD; M. Ludovica Carerj, MD; Imke Wieters, MD; Masia Fahim; Christophe Arendt, MD; Jedrzej Hoffmann, MD; Anastasia Shchendrygina, MD, PhD; Felicitas Escher, MD; Mariuca Vasa-Nicotera, MD; Andreas M. Zeiher, MD; Maria Vehreschild, MD; Eike Nagel, MD

• Prospective observational cohort study
• April – June 2020
• N = 100 adults, ~ 2 months after COVID-19
• Compared to risk-factor matched/non-COVID control group
• All “symptomatic” - 67% recovered at home, 33% hospitalized
Table 1. Patient Characteristics, Cardiac Magnetic Resonance (CMR) Imaging Findings, and Blood Test Results on the Day of CMR Examination

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>COVID-19 (n = 100)</th>
<th>Risk factor-matched controls (n = 57)</th>
<th>P value&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>49 (14)</td>
<td>49 (13)</td>
<td>.91</td>
</tr>
<tr>
<td>Male, No. (%)</td>
<td>53 (53)</td>
<td>28 (49)</td>
<td>.88</td>
</tr>
<tr>
<td>BMI, median (IQR)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>25 (23-28)</td>
<td>27 (23-29)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypertension, No. (%)</td>
<td>22 (22)</td>
<td>14 (25)</td>
<td>.003</td>
</tr>
<tr>
<td>Diabetes, No. (%)</td>
<td>18 (18)</td>
<td>12 (22)</td>
<td>.002</td>
</tr>
<tr>
<td>Hypercholesterolemia, No. (%)</td>
<td>22 (22)</td>
<td>13 (23)</td>
<td>.02</td>
</tr>
<tr>
<td>Known CAD, No. (%)</td>
<td>13 (13)</td>
<td>9 (16)</td>
<td>.02</td>
</tr>
<tr>
<td>Smoking, No. (%)</td>
<td>22 (22)</td>
<td>11 (19)</td>
<td>.54</td>
</tr>
<tr>
<td>COPD or asthma, No. (%)</td>
<td>21 (21)</td>
<td>13 (23)</td>
<td>.002</td>
</tr>
</tbody>
</table>
Conclusions and Caveats

Cardiac “involvement” in 73% of adult patients recovering from COVID-19, independent of severity of presentation

Yikes

Caveats!

• The “risk-factor matched controls” had some MRI changes as well
• This study is not applicable to pediatric patients
• Also not applicable to asymptomatic patients
RESEARCH LETTER

Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From COVID-19 Infection

Myocarditis is a significant cause of sudden cardiac death in competitive athletes and can occur with normal ventricular function. Recent studies have raised concerns of myocardial inflammation after recovery from coronavirus disease 2019 (COVID-19), even in asymptomatic or mildly symptomatic patients. Our objective was to investigate the use of cardiac magnetic resonance (CMR) imaging in competitive athletes recovered from COVID-19 to detect myocardial inflammation that would identify high-risk athletes for return to competitive play.

Methods | We performed a comprehensive CMR examination including cine, T1 and T2 mapping, extracellular volume fraction, and late gadolinium enhancement (LGE), on a 1.5-T scanner (Magnetom Sola; Siemens Healthineers) using standardized protocols, in all competitive athletes referred to the sports medicine clinic after testing positive for COVID-19 (reverse transcriptase-polymerase chain reaction) between June and August 2020. The Ohio State University institutional review board approved the study, and informed consent in writing was obtained from participating athletes. Cardiac magnetic resonance imaging was performed after recommended quarantine (11-53 days). Electrocardiogram, serum troponin I, and transthoracic echocardiogram were performed on day of CMR imaging.

Figure. Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From Coronavirus Disease 2019 Infection

| A | Steady-state free precession cine, patient 1 |
| B | T2 map, patient 1 |
| C | Phase-sensitive inversion recovery with late gadolinium enhancement, patient 1 |
Study
• In May 2020, U.S. professional sports leagues adopted “Return to Play” rules in line with ACC Guidelines at the time
• Cross-sectional study of this cardiac testing on n = 789 professional athletes s/p COVID-19

Testing
• Troponin, EKG, echocardiogram on all patients
• Abnormal screening → stress testing, cMRI
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>National Basketball Association</th>
<th>Major League Soccer</th>
<th>Major League Baseball</th>
<th>National Hockey League</th>
<th>National Football League</th>
<th>Total professional athlete cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td></td>
</tr>
<tr>
<td>Total athletes positive for COVID-19, No.</td>
<td>109 (12%)</td>
<td></td>
<td>70 (181)</td>
<td>68 (349)</td>
<td>349 (1)</td>
<td>789 (58.3%)</td>
</tr>
<tr>
<td>Age, mean (range), y</td>
<td>25 (19-35)</td>
<td>27 (21-33)</td>
<td>25 (18-31)</td>
<td>25 (19-38)</td>
<td>25 (19-41)</td>
<td>25 (19-41)</td>
</tr>
<tr>
<td>COVID-19 symptom burden</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preceding viral symptoms</td>
<td>71 (65.1%)</td>
<td>8 (67)</td>
<td>33 (47)</td>
<td>109 (60.2)</td>
<td>51 (75)</td>
<td>188 (53.9%)</td>
</tr>
<tr>
<td>Asymptomatic or paucisymptomatic</td>
<td>38 (34.9%)</td>
<td>4 (33)</td>
<td>37 (53)</td>
<td>72 (39.8)</td>
<td>17 (25)</td>
<td>161 (46.1%)</td>
</tr>
<tr>
<td>COVID-19 test</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polymerase chain reaction</td>
<td>75 (68.8%)</td>
<td>9 (75)</td>
<td>51 (73)</td>
<td>172 (95.0)</td>
<td>54 (79)</td>
<td>226 (64.8%)</td>
</tr>
<tr>
<td>Antibody</td>
<td>34 (31.2%)</td>
<td>3 (25)</td>
<td>19 (27)</td>
<td>9 (5.0)</td>
<td>14 (21)</td>
<td>123 (35.2%)</td>
</tr>
<tr>
<td>Days between COVID-19 polymerase chain reaction test and cardiac screen, mean (range)</td>
<td>32 (9-124)</td>
<td>23 (14-60)</td>
<td>14 (13-16)</td>
<td>21 (3-90)</td>
<td>18 (9-97)</td>
<td>17 (3-156)</td>
</tr>
<tr>
<td>Abnormal cardiac testing results</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Troponin</td>
<td>3 (2.8%)</td>
<td>1 (8)</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
<td>1 (0.3%)</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5 (2.8)</td>
<td>0</td>
<td>5 (1.4%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>2 (1.8%)</td>
<td>3 (25)</td>
<td>3 (4)</td>
<td>6 (3.3)</td>
<td>1 (1)</td>
<td>5 (1.4%)</td>
</tr>
</tbody>
</table>

a An abnormal troponin level was defined as a level greater than the 99th percentile of the reference laboratory value.

b An abnormal electrocardiogram was defined as meeting international recommendations and demonstrating findings raising concern for potential acute cardiac injury.

c An abnormal echocardiogram was defined by ventricular dysfunction or another finding raising concern for potential acute cardiac injury.
Thirty of 789 athletes (3.8%) had abnormal cardiac screening test results necessitating additional evaluation and downstream testing; 5 athletes (0.6%) were detected to have findings raising concern for COVID-19-associated inflammatory heart disease that resulted in restriction from sport participation per American Heart Association (AHA)/American College of Cardiology (ACC) guidelines. ECG indicates electrocardiogram; TTE, transthoracic echocardiogram.
CONCLUSIONS AND RELEVANCE This study provides large-scale data assessing the prevalence of relevant COVID-19-associated cardiac pathology with implementation of current RTP screening recommendations. While long-term follow-up is ongoing, few cases of inflammatory heart disease have been detected, and a safe return to professional sports activity has thus far been achieved.


5 out of 789 players = 0.6%
Cardiac Screening and Return-to-Play following COVID-19 Infection

◆ All student-athletes diagnosed with a COVID-19 (SARS-CoV-2) will require isolation for 10 days with day 0 starting at the onset of symptoms or the day of testing, if asymptomatic.
◆ No exercise during the isolation period.
  • After the isolation period is completed, each student-athlete will undergo a medical evaluation by a team physician.
◆ Cardiac testing and a period of re-acclimation to exercise required prior to returning to full participation in sport.
The required cardiac testing will include:
1. Electrocardiogram (EKG)
2. Serum Troponin level
3. Echocardiogram (ECHO)

The results of these tests, medical evaluation findings, or the clinical course of the student-athlete (i.e. moderate to severe infections requiring hospitalization) may warrant further testing (such as cardiac MRI) based on the discretion of the team physician.

In addition to cardiac testing, a minimum of a 4-day period of re-acclimation to exercise will be required to monitor for any signs or symptoms of cardiac complications (i.e. chest pain, shortness of breath, presyncope, syncope). Day 1 of re-acclimation should be approximately 25% of a normal practice or conditioning session, with Day 2 being 50%, Day 3 being 75% and Day 4 being full participation.
UAB Sports and COVID-19

N = 184 UAB athletes screened to date

Football, basketball, baseball, volleyball, soccer, track, cross-country, tennis, golf, and yes . . . bowling

Patients

• S/p COVID-19, now asymptomatic, at least 10 days after infection
• Everyone receives COVID-19 IgG testing prior to clearance regardless

Clearance to play

• Normal troponin, EKG, and echocardiogram
• Cleared by team physician

No disqualifications to date
<table>
<thead>
<tr>
<th></th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rates of infection</strong></td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Evidence</strong></td>
<td>Chinese Center for Disease Control and Prevention reported only 2% of patients younger than 20 years of age among 44,672 cases [20]</td>
<td>United States Center for Disease Control and Prevention (CDC) reported only 1.7% children less than 18 years of age among 149,082 reported cases [7]</td>
</tr>
<tr>
<td><strong>Severity of illness</strong></td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Evidence</strong></td>
<td>Chinese data: 14% of adult patients classified as ‘severe’ and 5% as ‘critical’ disease [20]</td>
<td>Chinese data: 5% of pediatric patients classified as ‘severe’ and &lt;1% as ‘critical’ disease [36]</td>
</tr>
<tr>
<td></td>
<td>Italian data: 25% of adult patients classified as ‘severe’ and 5% as ‘critical’ [6]</td>
<td>Italian data: 2% of pediatric patients classified as ‘severe’ or ‘critical’ disease [5]</td>
</tr>
<tr>
<td><strong>Complications and mortality</strong></td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Evidence</strong></td>
<td>United States data: Among 2634 adults hospitalized in 12 hospitals in New York City, 14% required intensive care and 12% received mechanical ventilation with a case fatality rate of 21% [4]</td>
<td>United States data: Among 46 North American Pediatric ICUs, only 48 children were admitted to intensive care with a case fatality rate of 4.2% [3]</td>
</tr>
<tr>
<td></td>
<td>Chinese data: Case fatality 2.3% of overall COVID-19 affected population [20]</td>
<td>Chinese data: 1 death [36]</td>
</tr>
<tr>
<td></td>
<td>Italian data: Case fatality of overall COVID-19 affected population 7% [6]</td>
<td>Italian data: No deaths [5]</td>
</tr>
<tr>
<td><strong>Potential risk factors leading to severe disease</strong></td>
<td>Hypertension, diabetes, and obesity [4]</td>
<td>Infants &lt; 1 years of age, medical complexity, immune suppression, obesity [3, 5]</td>
</tr>
</tbody>
</table>

*aDefined as dependence on technological support (tracheostomy with or without ventilator dependence) in association with developmental delay and/or genetic anomalies*
WHO case definition Multisystem Inflammatory Syndrome Children – need all 5 criteria

1. Fever for ≥3 days

2. Clinical signs of multisystem involvement (at least 2 of the following):
   - Rash, bilateral nonpurulent conjunctivitis, or mucocutaneous inflammation signs
   - Hypotension or shock
   - CV dysfxn, pericard-/valvul-/myocarditis, coronary changes, abnormal trop/BNP
   - Evidence of coagulopathy
   - Acute gastrointestinal symptoms

3. Elevated markers of inflammation (eg, ESR, CRP, or procalcitonin)

4. No other obvious microbial cause of inflammation

5. Evidence of SARS-CoV-2 infection (any of the following):
   - Positive SARS-CoV-2 RT-PCR
   - Positive serology
   - Positive antigen test
   - Contact with an individual with COVID-19
COVID(+), either asymptomatic or mildly symptomatic (< 4 days fever, short duration of mild sx)

*See PCP who performs H&P
- If possible CV symptoms or exam findings, refer to cardiology
- If screening normal, can return to gradual play after 10 days from infection
- No mandatory EKG

COVID(+), with moderate initial sx (>4 days of sx or non-ICU hospital stay. No MIS-C

*Cardiology consult is recommended at minimum 10 days s/p (+) test result. No exercise until cleared
-Cardiologist will consider testing including labs, cMRI, stress testing
-If CV testing negative, can be allowed to return to play on similar schedule as the asymptomatic/mild sx patients

COVID-19 Interim Guidance: Return to Sports

*Cardiology consult is recommended and should be arranged prior to hospital discharge
-Recommend 3-6 months of exercise restriction
-Cardiologist will coordinate testing needed over time

COVID(+), with severe symptoms (ICU, MIS-C)
