



NCC Pediatrics Continuity Clinic Curriculum: Sports Physical II: Special Topics



Goals & Objectives:

To understand the importance of the pre-participation exam and to gain the skills necessary to perform an adequate exam and recognize common problems.

- Gain a better understanding of the importance of the cardiovascular risk factors elicited in the history and a greater understanding of the importance of the cardiovascular exam.
- Become familiar with classification of sports by contact vs. noncontact and by levels of dynamic components in order to adequately counsel potential athletes.
- Understand presentation and management of concussion, including baseline neuro-psychological testing and a strict return-to-play protocol with cognitive and physical rest.

Pre-Meeting Preparation:

Please read the following enclosures:

- "Risk Factors for Sudden Death in Athletes, Is There a Role for Screening?" (*Current Cardiovascular Risk Reports, 2022*)
- "Pediatric Head Trauma: A Review and Update" (*excerpt, PIR, 2019*)
- Review Tools
 - [36th Bethesda Conference Sports Classifications](#)
 - AHA 14 and PPE-4 Monograph
 - SCAT5: Sports Concussion Assessment Tool (2017)
 - [Heads Up](#) (CDC Concussion Program) and ACE Tool

Conference Agenda:

- Review Sports Physical II Quiz
- Complete Sports Physical II Cases
- **Exercise: Perform SCAT5 w/partner.**

Post-Conference: Board Review Q&A

Extra-Credit:

- ["Demystifying the Pediatric Electrocardiogram: Tools for the Practicing Pediatrician"](#) (*PIR, 2021*)
- ["Diagnosis and Management of Mild Traumatic Brain Injury in Children: A Systematic Review"](#) (*JAMA Peds, 2018*)
- ["Sudden Death in the Young: Information for the Primary Care Provider"](#) (*AAP Policy Statement, 2021*)
- ["Association Between Early Return to School Following Acute Concussion and Symptom Burden at 2Weeks Postinjury"](#) (*JAMA Pediatrics, 2022*)
- ["Characteristics and Outcomes of Athletes With Slow Recovery From Sports-Related Concussion"](#) (*Neurology, 2023*)
- **Local Programs:** [S.C.O.R.E. @ CNMC](#); [Kennedy Krieger Neurorehab Clinic](#)



Risk Factors for Sudden Death in Athletes, Is There a Role for Screening?

Alexander G. Hajduczuk¹ · Max Ruge² · Michael S. Emery³

Accepted: 13 June 2022 / Published online: 4 July 2022

© The Author(s), under exclusive licence to Springer Science+Business Media, LLC, part of Springer Nature 2022

Abstract

Purpose of Review Sudden cardiac death (SCD) in a young athlete is an infrequent yet devastating event often associated with substantial media attention. Screening athletes for conditions associated with SCD is a controversial topic with debate surrounding virtually each component including the ideal subject, method, and performer/interpreter of such screens. In fact, major medical societies such as the American College of Cardiology/American Heart Association and the European Society of Cardiology have discrepant recommendations on the matter, and major sporting associations have enacted a wide range of screening policies, highlighting the confusion on this subject. This review seeks to summarize the literature in this area to address the complex and disputed subject of screening young athletes for SCD.

Recent Findings The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can cause myocarditis, which is one acquired cardiac disease associated with SCD. The coronavirus 2019 (COVID-19) pandemic has therefore resulted in an increased incidence of an otherwise less common condition, providing an expanded dataset for further study of this condition. Recent findings indicate that cardiac complications of athletes with myocardial involvement of SARS-CoV-2 infection are rare. Other contemporary work in SCD screening has been focused on the implementation of various screening protocols and measuring their effectiveness.

Summary No universal consensus exists for athlete screening for conditions associated with SCD with varying guidelines and protocols across cardiology and sport-specific organizations. No screening program will prevent all SCD; however, small programs managed by physicians familiar with the examination of an athlete that carefully personalize screening to the individual may maximize detection of dangerous cardiac conditions while minimizing false positives.

Keywords Athlete · Pre-participation screening · Sudden cardiac death · Electrocardiogram · Emergency action plan

Alexander G. Hajduczuk and Max Ruge are co-first authors.

This article is part of the Topical Collection on *Arrhythmias*.

✉ Michael S. Emery
emerym2@ccf.org

¹ Division of Cardiology, Jefferson Heart Institute, Thomas Jefferson University Hospital, Philadelphia, PA, USA

² Department of Internal Medicine, Thomas Jefferson University Hospital, Philadelphia, PA, USA

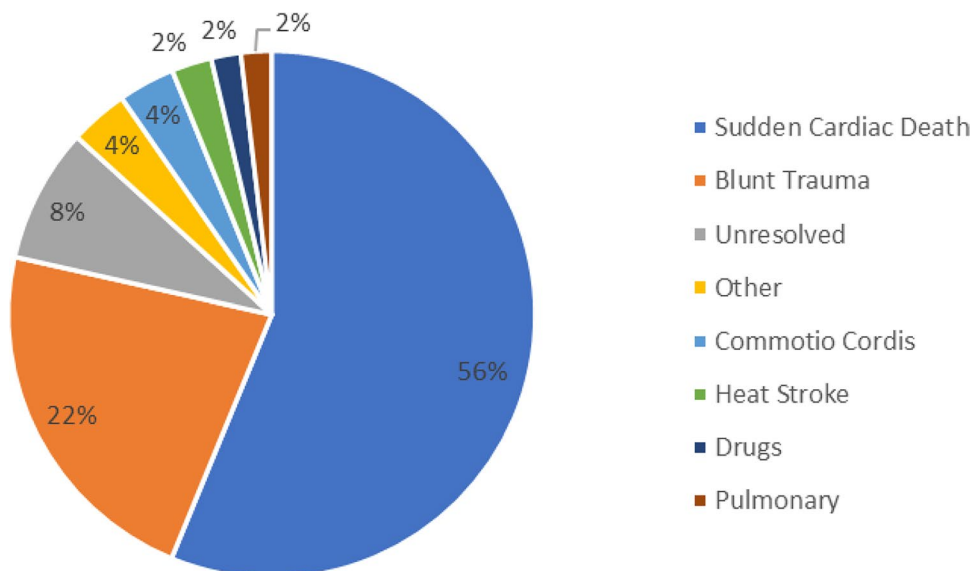
³ Sports Cardiology Center, Department of Cardiovascular Medicine, Heart, Vascular and Thoracic Institute; Cleveland Clinic, 9500 Euclid Ave, Cleveland, OH Desk J2-4, USA

Introduction

Sudden death of a child or young adult during exercise is an infrequent yet devastating event that can have substantial downstream effects on the community and loved ones. These events often receive substantial media attention, in part due to the paradox of athletes, often presumed to be some of the healthier members of society, being struck by a condition often associated with a sedentary and unhealthy lifestyle. Most cases of sudden death are from sudden cardiac death (SCD), which is the focus of this review (Fig. 1). Conversely, the minority of causes are non-cardiac, which include cerebral aneurysms, heat stroke, pulmonary diseases such as an asthma exacerbation, and even remained unexplained in a significant number of cases [1, 2].

The particularly devastating nature of these have prompted screening efforts in an attempt to prevent future

Fig. 1 General etiologies of sudden death in competitive athletes ≤ 39 years old (adapted from Maron et al., 2009) [2]



cases. While many major societies and organizations recommend various forms of primary prevention, more questions than answers exist to optimize the screening process. Who exactly should be screened and at what interval? What is the optimal screening method—history and physical alone or additional testing such as electrocardiography? Who should be performing and interpreting any form of cardiovascular screening?

The goal of this review is to summarize the extensive body of literature of screening for the prevention of SCD in children and young adults (≤ 40 years old).

Incidence

SCD is defined as a sudden unexpected death due to cardiac causes or sudden death in a structurally normal heart with no other explanation and a history consistent with cardiac related death [3]. Sudden cardiac arrest (SCA) is defined as “death from an unexpected circulatory arrest, usually due to a cardiac arrhythmia occurring within an hour of the onset of symptoms, in whom medical intervention (i.e., defibrillation) reverses the event” [4].

The incidence of SCD in athletes of all ages has been estimated to range from 1/39,000 [5] to 1/281,000 [6], while the incidence in young athletes is approximately 1–2 per 100,000 athletes per year [7]. While participation in sports or sport training may increase risk of SCD/SCA by 2.4 to 4.5-fold compared to non-athletes or recreational athletes, the majority of SCD cases occur in the non-athlete population [8–10]. In the general population, Kong et al. estimated the annual incidence of SCD to range between 180,000 and 450,000, corresponding to between 7 and 18% of all total deaths in a 2011 systematic review [4]. In the general

population of the USA, Stecker et al. (2014) provided an estimate of around 183,000 cases of SCD and 201,000 cases of SCA based upon a population-based surveillance study from 2002 and 2004 [11]. From this data, they posited that the age-adjusted national incidence of SCD was 60 per 100,000 individuals (95% confidence interval of 54–66 SCDs per 100,000).

A multitude of studies, both prospective and retrospective, have tried to determine the incidence of SCD over the years but have been limited by lack of a mandatory universal reporting structure with most studies gathering cases from media reports and/or insurance claims [2, 5, 6, 8, 9, 12–17]. The fundamental complexity of the term “sudden cardiac death” is a major obstacle, including what constitutes “cardiac,” “sudden,” and whether “resuscitated arrest” counts as SCD. One reason for the large variability in findings is due to differences in inclusion criteria for these studies. This leads to substantial discrepancies in the number of athletes who are reported to experience SCD; some include only events that result in death (SCD) versus others that include those that survive cardiac arrest (SCA) as well. The differences in data sources (spanning from the 1980s to the present day) and variability in case ascertainment criteria add to the inconsistencies in SCD incidence estimates.

Reporting and data collection methodology also differs between media databases, insurance claims, and National Collegiate Athletic Association (NCAA) databases. For example, in one study, there was nearly a 60% difference in cases reported by media database reports versus insurance claims (70% versus 11%) [16].

Ultimately, it may be difficult to obtain a true estimate of SCD incidence due to its infrequent nature and need for a stable population measured over a long study period, which may not be feasible. Despite the differences in reported

incidence, there is consistency in the finding that male athletes have a 3–5 × greater incidence of SCD than women [18]. In addition, from NCAA data, black athletes have over a threefold increase in the rate of SCD as compared to white athletes, and this is even more pronounced in black NCAA Division I basketball players [19]. Understanding this heterogeneity may help direct future studies and enhanced preventative strategies in more vulnerable populations.

Are Athletes at Higher Risk of Sudden Cardiac Death?

SCD in athletes receives significant attention from the media and the community, potentially skewing opinion to associate these events with sport. The paradox of SCD occurring during an activity otherwise associated with health likely drives this increased attention. In reality, SCD often occurs off the field as well which receives substantially less media attention. Many prior studies of SCD have primarily focused on competitive athletes further solidifying this association. From a physiological perspective, vigorous exercise generates a burst of sympathetic activation, which can precipitate arrhythmias particularly in genetically predisposed individuals. It is therefore important to acknowledge that sport itself does not cause the cardiac abnormalities but represents a trigger that can precipitate SCD in those with certain pre-existing cardiac conditions [1]. Therefore, the finding that athletes are at higher risk for SCD than non-athletes (relative risk 2.5–4.5) could be result of more frequent exposure to the trigger of vigorous exercise [1, 9].

What Causes Sudden Cardiac Death?

The majority of cardiac diseases that have been implicated in SCD are otherwise quiescent genetic abnormalities that can become unmasked by the sympathetic surge associated with vigorous exercise with potentially lethal consequences. Many diseases have been implicated in SCD, and prior reviews have broadly grouped these diseases into sub-classifications of structural, acquired, and electrical abnormalities [18]. The incidence of each varies significantly across studies (Table 1) [1, 2, 9, 15, 19–23].

Determining the etiology of a case of SCD is often challenging. First, no standardized criteria exist for autopsy diagnoses of many conditions associated with SCD, so pathology lab variation likely exists in diagnosis. A 2014 study found that a pathologist specialized in cardiovascular histopathology and the original referring pathologist differed on final diagnosis in 41% of cases of SCD highlighting both inter-provider variation and the need for specialists in these cases [24]. Some have suggested a more protocolized autopsy

Table 1 Summary of major studies that have examined the etiology of sudden cardiac death in children or young adult athletes

| Authors | Country | Age | Number of SCD | HCM | ARVC | AAOCA | DCM | CAD* | MVP | Myocarditis | LQTS or BrS | WPW |
|-------------------------|---------|----------|---------------|-----|------|-------|-----|------|-----|-------------|-------------|-----|
| Corrado et al. [1] | Italy | 12 to 35 | 55 | 2% | 22% | 13% | 2% | 18% | 11% | 9% | 2% | - |
| de Noronha et al. [20] | UK | ≤35 | 89 | 12% | 10% | 7% | - | 0% | 2% | 3% | - | - |
| Finocchiaro et al. [21] | UK | ≤35 | 258 | 7% | 12% | 6% | 3% | 2% | - | 2% | - | - |
| Marijon et al. [9] | France | 10 to 35 | 50 | 10% | 4% | - | 4% | 6% | 4% | 4% | 2% | 2% |
| Maron et al. [2] | USA | ≤39 | 1049 | 24% | 3% | 11% | 1% | 2% | 2% | 4% | 2% | 1% |
| Harmon et al. [19] | USA | 17 to 24 | 79 | 6% | 4% | 9% | - | 8% | - | 8% | 1% | 3% |
| Maron et al. [15] | USA | 17 to 26 | 64† | 33% | 5% | 13% | 3% | 8% | 2% | 3% | 2% | 0% |
| Maron et al. [22] | USA | 13 to 25 | 842 | 36% | 5% | 19% | 2% | 5% | 4% | 7% | 2% | 1% |
| Peterson et al. [23] | USA | 11 to 29 | 331 | 13% | 4% | 8% | 2% | 2% | - | 3% | 3% | 3% |

SCD sudden cardiac death, HCM hypertrophic cardiomyopathy, ARVC arrhythmic right ventricular cardiomyopathy, AAOCA anomalous origin of coronary artery, DCM dilated cardiomyopathy, CAD coronary artery disease, MVP mitral valve prolapse, LQTS long QT syndrome, BrS Brugada syndrome, WPW Wolff–Parkinson–White syndrome

*Atherosclerotic or not specified

†Including those with both confirmed and suspected CV death

could reduce variability, but even with this intervention, it is likely that those without a precise etiology of the SCD will make up a sizeable portion [25]. Second, post-mortem diagnoses may be biased towards structural heart disease simply by the nature of autopsy. Conversely, electrical abnormalities may be under-reported as they often require an ECG prior to the SCD, which may or may not be present, or even post-mortem genetic testing. Even after autopsy, no etiology of the SCD is found in a large proportion of victims, ranging from 7 to 44% [12, 19–21, 26]. Finally, autopsy is not always performed or the results are unavailable, so the etiology of death is often determined by review of medical history, death certificates, or even discussions with family, which have substantial limitations and bias. Since it is a rare event, identifying a case of SCD by retrospective review can be difficult with commonly used but somewhat superficial strategies such as media reports or insurance claims being biased and often incomplete [19].

Structural Cardiac Disease

The most common cited etiology of SCD is structural heart disease but is potentially biased by the nature of the autopsy studies, which are best suited to find such disorders [1, 2, 9, 15, 19–23]. Three structural cardiac abnormalities are most commonly associated with SCD: hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and coronary artery abnormalities (CAA) [2, 15, 20–23].

HCM is a category of genetic cardiomyopathies with several subtypes that subsequently can produce a range of hemodynamic changes and symptoms [27, 28]. ARVC is an inherited cardiomyopathy caused by fibrofatty replacement of the free RV wall muscle and can predispose to arrhythmias that can result in SCD [29]. ARVC is particularly difficult to detect prior to SCD because life-threatening arrhythmias are often the initial presentation [30]. CAA is a broad term that can refer to abnormal number or size of the coronary arteries, origin off the aorta, or vessel course [31, 32]. The CAA most associated with SCD occurs when the left coronary artery originates from the right coronary cusp, particularly when the vessel has an early intramural segment that takes an inter-arterial course between the pulmonary artery and the aorta [32]. While the mechanism for ischemia was traditionally thought to be direct compression of the anomalous artery, the hemodynamics are likely more complex and an area of ongoing research [33–35].

Significant geographic variation in some structural cardiac disease appears to be present in studies that examine the etiologies of SCD (Table 1). For example, HCM has been implicated in up to 36% of cases of SCD in the USA [2, 15, 19, 22, 23] compared to 2–12% of cases in Italy, the UK, and

France [1, 9, 20, 21]. Conversely, ARVC is highest reported in Italy (22%) [1] followed by the UK (10–12%) [20, 21], and then the USA and France (3–5%) [2, 9, 15, 19, 22, 23]. Since a genetic component exists for many of these conditions, these findings could reflect the regional prevalence of the abnormality [29]. These data therefore suggest that geographic region of the world should be a factor to consider when creating screening protocols.

Acquired Abnormalities

Acquired cardiac abnormalities, such as myocarditis, have also been identified in registries as causes of SCD in athletes. Myocarditis can be caused by both infectious and non-infectious pathologies [36]. The initial acute phase causes direct cardiac inflammation that can trigger electrical instability of the myocyte, while the arrhythmias in the post-acute phase of myocarditis are typically due to injury resulting in myocardial scar [37]. This group also includes commotio cordis (blunt trauma to the chest resulting in SCD), environmental factors such as heat stroke, and illicit substances including performing enhancing drugs [18].

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which is responsible for the coronavirus disease 2019 (COVID-19) pandemic, is known to cause myocardial injury and has reinvigorated interest in studying post-viral myocarditis and provided an abundance of objective data for the study of myocarditis after a viral illness [38••, 39–41]. The prevalence of myocardial involvement of COVID-19 is highly dependent on the screening modality used. In two multicenter studies of NCAA athletes with COVID-19, primary screening for myocardial involvement with cardiac magnetic resonance imaging (CMR) yielded a prevalence of 2.3–3.0% though many of these athletes had no clinical symptoms and as such a low pre-test probability making interpretation of the imaging findings more difficult [39, 40]. When a step-wise protocol was used in NCAA and professional athletes that initially screened via cardiac troponin, ECG, and transthoracic echocardiogram (TTE) followed by CMR if any abnormalities were found, the prevalence was estimated to be 0.6–0.8% [40, 41]. Despite the known association of viral myocarditis with SCD, a 2022 study that followed over 3500 athletes with COVID-19 for a median duration of approximately one year found only one cardiovascular adverse event, a case of atrial fibrillation, that was possibly related to COVID-19 [42••]. These data are reassuring and suggest that undeclared myocardial inflammation during COVID-19 infection resulting in cardiac complications is a rare event.

The current American College of Cardiology (ACC) return to play guidelines after COVID-19 infection recommend a modified step-wise approach that incorporates risk

stratifying the athlete for the likelihood of cardiac involvement first by symptoms [38••]. In athletes who had COVID-19 with no cardiac symptoms such as chest pain, palpitations, dyspnea, or syncope, no activity restriction is needed. If any of these symptoms are present, the ACC guidelines recommend further screening with cardiac troponin, ECG, and a TTE. Abnormal findings from these studies should be further investigated with CMR. If myocarditis is diagnosed, the athlete should avoid physical activity for 3–6 months and have repeat cardiac testing before being allowed to return to play.

Electrical Abnormalities

The last major category of causes of SCD is electrical abnormalities, which primarily consists of pre-excitation syndromes such as Wolf–Parkinson–White syndrome, channelopathies such as Brugada syndrome and long QT syndrome, and catecholamine polymorphic ventricular tachycardia [18, 43–45]. This category is consistently the least frequently cited cause of SCD [2, 9, 15, 19, 22, 23] though this underreporting could be due to detection bias as many of these cannot be diagnosed using a typical autopsy [43]. Some have posited that these conditions could make up a much larger proportion of otherwise unexplained deaths after autopsy [46]. Studies of patients with unexplained SCD and SCA have found that genetic testing is able to identify a clinically significant variant in 22–27% of patients, indicating a possible etiology for these otherwise unsolved cases [46, 47].

Primary Prevention

A version of pre-participation screening dates back to the 1890s in Britain and subsequently came to the USA after a large proportion of military-aged males that were screened during World War II were found to be unfit for service [48••]. In 1966, the American Medical Association formally supported the screening of athletes, which launched the process of the pre-participation examination (PPE) becoming routine [48••]. In the present day, the USA (American Heart Association; AHA/ACC) endorses, but does not mandate, routine PPE consisting of history and physical examination. ECG screening with a history and physical is recommended by the European Society of Cardiology (ESC) and mandated in Italy and Israel (Table 2). However, since there are no prospective randomized control trials, these recommendations are primarily based on observational data.

History and Physical Examination

History and physical examination for PPE is recommended by most major screening bodies, which can serve as a screen for potentially lethal cardiac disorders in addition to a touch point for an adolescent patient into the medical system. In fact, retrospective studies have found that 18–19% of athletes who suffered from SCD had antecedent symptoms such as chest pain, palpitations, syncope, or dyspnea that could have identified them at high risk for SCD [20, 21]. Approximately one in five victims of SCD also had significant personal past medical history including presence of a heart murmur,

Table 2 Summary of the AHA, ACC, ESC, and AMSSM guidelines for cardiovascular screening in athletes

| | |
|---------|--|
| AHA/ACC | <ul style="list-style-type: none"> - On 3 occasions (1996, 2007, and 2014), AHA consensus expert panels evaluated and decided not to support mandatory national athlete screening in the USA, particularly with routine use of ECGs [49–51] - The AHA has not opposed ECG-based screening in smaller venues (non-universal screening) - For such screening initiatives, the AHA has prudently advised adequate quality control with due consideration for the prominent limitations of the process (including false-negative and false-positive test results), so that the risks and benefits can be understood and are acceptable to all participants, communities, and organizations |
| ESC | <ul style="list-style-type: none"> - This panel suggests a European standard for medical evaluation of competitive athletes. The recommended protocol includes 12-lead ECG in addition to history and physical examination, which is the only screening modality proved to be effective in identifying athletes with HCM, and preventing sudden death - The addition of 12-lead ECG has the potential to enhance the sensitivity of the screening process for detection of cardiovascular diseases with risk of sudden death |
| AMSSM | <ul style="list-style-type: none"> - The electrocardiogram (ECG) increases early detection of some cardiac disorders associated with SCA/SCD - ECG interpretation accuracy and reliability are challenges with the principal concern of adding false-positive results to the PPE screening process - Results from centers with considerable experience in athlete ECG screening have demonstrated improved detection of cardiac conditions with potential risk for SCA/D and decreased false-positive rates - Physicians incorporating ECG in the cardiovascular screening process should optimize strategies to assure accurate ECG interpretation and adequate cardiology resources to conduct the secondary evaluation of ECG abnormalities |

AHA American Heart Association, ACC American College of Cardiology, ECG electrocardiography, ESC European Society of Cardiology, AMSSM American Medical Society for Sports Medicine, PPE pre-participation examination

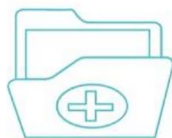
diabetes mellitus, congenital heart disease, myocarditis, or even previous cardiac arrest [20]. These retrospective studies also found that 6.9% of young SCD victims had a family history of SCD [20] and 8% had a family history of death of a first degree relative prior to the age of 50 years [21].

The most commonly accepted screening methodology is the AHA 14-point PPE, which includes inquiry about patient symptoms, medical history, and family history in addition to hallmark physical exam findings associated with potentially lethal cardiac abnormalities and is a class I recommendation by the AHA (Fig. 2) [49, 52]. The American Academy of Pediatrics (AAP), in collaboration with multiple other societies with an interest in athletic care including American Academy of Family Physicians, American College of Sports Medicine, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and the American Osteopathic Academy of Sports Medicine, also released the Preparticipation Physical Evaluation, 5th edition in 2019 (PPE-5). The PPE-5 incorporates the AHA 14-element history and physical with some changes in language and wording that may elicit more specific responses from young athletes to identify potential concerning cardiac

issues [48••]. The PPE-5 also contains a comprehensive non-cardiac screening inquiring about musculoskeletal pain, rashes, hernias, vision, eating disorders, and prior head injury [48••]. Others have developed web-based multimedia platforms to utilize as part of a PPE with the intent to reduce the false positive rate associated with the standard paper-based PPE [53•]. The recommended cardiac physical examination is primarily focused on identifying stigmata of Marfan's syndrome, cardiac murmurs, and delayed or absent femoral pulses indicative of coarctation of the aorta in both of these guidelines [48••, 49].

History and physical examination alone have several key limitations. Only about one in five patients who suffer SCD have antecedent symptoms [20, 21], which means the vast majority will have negative symptomatic screenings. The individual symptoms asked about in AHA 14-point PPE and PPE-5 are based off expert opinion and have never been systematically testing with a prospective, randomized controlled trial. These limitations significantly impact the sensitivity that can be obtained with history and physical examination alone. A 2015 meta-analysis of 15 publications with a total of 47,137 patients found a sensitivity/specificity

Fig. 2 Components of the AHA-recommended, 14-point pre-participation screening



Personal Medical History

- Exertional Chest Pain
- Unexplained syncope or near-syncope (not vasovagal)
- History of heart murmur
- Elevated blood pressure
- History of sport restriction
- Prior cardiac testing



Family Medical History

- Cardiac death prior to age 50 in 1 or more relative
- Disabling cardiac disease prior to age 50
- "Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members"



Physical Examination

- Heart Murmur
- Femoral pulses (assessing for aortic coarctation)
- Stigmata of Marfan syndrome
- Blood pressure

of 20%/94% for history and 9%/97% for physical examination using either the 14-element AHA or similar questionnaire [16].

Electrocardiography

One of the most controversial elements of screening in athletes is the potential addition of electrocardiography (ECG). It has been postulated that adding an ECG might be able to identify abnormalities not found with history and physical examination alone that could predispose a patient to potentially life-threatening arrhythmias. The AHA, ACC, AAP, and other co-developers of the PPE-5 recommend against widespread ECG screening for pre-participation physicals [48••, 52, 54, 55], while the ESC endorses its use in screening [56]. Many sporting organizations either recommend (e.g., International Olympic Committee, National Basketball Association (NBA), World Boxing Federation, and World Rugby) or mandate (e.g., Union of European Football Associations (UEFA), Fédération Internationale de Football Association (FIFA), Union Cycliste Internationale, and Fédération Internationale de l'Automobile) ECG screening [57].

Data from 47,137 athletes across 15 studies showed that ECG screening had a much higher sensitivity and specificity (94%/93%) compared to 20%/94% of screening with history and 9%/97% with physical examination [16]. This meta-analysis also found a positive predictive value of ECG, history, and physical to be 14.8, 3.22, 2.93, respectively, and the negative predictive value to be 0.055, 0.85, and 0.93, respectively. The authors argued that the significantly higher sensitivity of ECG was likely because only 20% of patients have symptoms prior to SCD, and these symptoms are often very nonspecific. A prospective study of 814 athletes found ECG screening superior to the AHA 14-point questionnaire in identifying CV conditions with the potential to cause SCA/SCD [58•]. Another study of 510 collegiate athletes found that the addition of an ECG to history and physical examination screening increased sensitivity from 45.5 to 90.9% at the expense of an increased in false positive rates from 5.5 to 16.9% [59]. Each of these studies examined the ECG's accuracy in identifying conditions associated with SCD, which is related though distinct from the more clinically relevant question of whether ECG utilization decreases the incidence of SCD. To date, no randomized controlled trial has been performed to assess the efficacy of screening with ECG or even history and physical examination.

The evidence supporting use of widespread screening with ECGs is primarily derived from a study of the Veneto region of Italy (~9% of the Italian population), which found an 84% reduction in the annual incidence of SCD with the implementation of a 1982 ECG screening program in 12 to

35 year olds [8]. The authors believed that much of the benefit of the program came from identification of those with a cardiomyopathy as the percentage of athletes who died from cardiomyopathy decreased from 36% to 17% while the proportion of those disqualified due to cardiomyopathy increased from 4.4% to 9.4%. This study has drawn a number of criticisms including the high rates of SCD immediately prior to initiating the screening program, the inclusion of only 2 years of data pre-screening compared to over 20 years after screening, and the overall low event rate of 320 events during an estimated 36,144,100 person-years [49]. The results of this study, while impressive, have not been replicated to date. Conversely, other studies have failed to find benefit in ECG screening. In 1997, Israel mandated the National Sport Law, which required pre-participation screening that included an ECG of all athletes by a physician specifically certified in the exam. However, a 2011 study found no difference in the annual incidence of SCD in the 12 years before versus after the screening program [5]. Interesting, the study authors found that limiting the pre-screening period to the two years prior to the implementation of the screening program yielded similar results to the Italian study. It is therefore possible that a relatively higher incidence of SCD yet with still low absolute numbers in a given year could skew or bias the data. Another study comparing screening with history and physical alone of athletes in Minnesota versus athletes who received the comprehensive ECG screening in Italy found similar mortality rates [60]. A study in Denmark, a country that does not require any screening, found no difference in its SCD incidence when compared to the Italian post-screening group or the Minnesota populations screened with history and physical alone [12].

ECG as a screening tool does has limitations. Interpretation of athlete ECG differs from the general population due to physiologic adaptations associated with routine vigorous exercise [61]. Most physicians are not trained to read the ECGs of athletes, and most computer interpretation algorithms used in common systems do not incorporate athlete ECG interpretation criteria. Interpretation of an athlete's ECG without consideration of these physiological differences significantly limits the ECG's specificity and can lead to unnecessary and potentially extensive downstream testing [50]. Physician experience and treatment specialty can also affect accuracy, thus multiple iterations ECG criteria for athletes have been created and refined, each of which have progressively reduced false positive rates [62]. The first attempt at creating an athlete-specific criteria was in 1998 and focused solely on the screening for HCM [63]. Seven years later in 2005, the ESC produced the first guideline document on ECG criteria specific to athletes. This was modified in 2010 in order to define criteria to distinguish normal physiologic versus pathologic findings on an athlete's ECG [56, 64]. Since the ESC criteria were formed

with a predominantly white population, efforts were made to incorporate ECG findings that were normal in non-white populations. The “Seattle Criteria” was published in 2013, which included normal ECG findings in Black athletes followed by the “Refined Criteria” in 2014 with identified a group of “borderline” ECG findings that should be considered a normal variant in isolation but abnormal if two or more are present on the ECG [65, 66]. The most current guidelines are the “International Criteria” that were published in 2017 that further refined the normal, borderline, and abnormal ECG findings in athletes (Fig. 3) [61]. A large study of 11,168 soccer players found that each iteration of ECG criteria improved specificity with decreased false positive rates while maintaining a sensitivity [67]. This study found a specificity/false positive rate of 87%/12.9% for the ESC 2010 guidelines compared to 98%/1.9% for the International Criteria [67].

The ECG is not able to detect all abnormalities associated with SCD, so it will never be a 100% sensitivity test for conditions at high risk for SCD [61]. A 2014 retrospective study of the US National Registry of Sudden Death found that 60% of the diagnoses responsible likely could have been identified if an ECG had been obtained, such as hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and long QT syndrome [15]. In a prospective cardiac screening program that included ECG of 11,168 adolescent soccer players in the United Kingdom over 20 years, 6 sudden cardiac deaths still occurred in the group of 10,625 who had normal screening, underscoring the imperfect nature of the ECG as a SCD screening tool [68]. Interpretation of an ECG tracing is also not an entirely objective exercise, which introduces inter-reader variability into the screening process, further limiting its accuracy. However, others have

found that ECG is significantly better in identifying conditions associated with SCD when compared to history or physical exam [16].

Transthoracic Echocardiogram

Given that many SCDs are from structural cardiac disease, a modality specifically aimed at assessing the structure of the heart, such as TTE, sounds promising. For example, one of the strongest predictors of SCD in HCM is extreme left ventricular hypertrophy, which can be rapidly assessed on TTE by measuring the ventricular wall thickness in the parasternal short axis plane [69]. A TTE is also able to screen for cardiac diseases associated with SCD that do not cause ECG abnormalities such as coronary abnormalities and aortopathies. It is noninvasive, safe, and widely available giving it many characteristics of an ideal screening test.

While promising in theory, the precise role of TTE in PPE screening has yet to be established. Currently, most major medical societies recommend against its use in primary screening though some professional sports organizations, such as UEFA, FIFA, Union Cycliste Internationale, and Fédération Internationale de l'Automobile, require TTE in addition to an ECG during PPE [70]. Studies that have assessed efficacy of TTE as a widespread screening tool of children and young adults have generally failed to demonstrate its effectiveness. In a screening study of 11,168 athletes that utilized TTE, 6 of the 8 adolescents who died of SCD had a normal TTE despite 7 of the 8 deaths being attributed to structural heart disease [68]. In another study of 595 professional athletes that screened using a TTE, none of the 6 patients who had severe cardiovascular incidents had

| Low Risk ECG Findings | Borderline ECG Findings | High Risk ECG Findings |
|---|---|--|
| <p><i>No further evaluation needed if asymptomatic without significant family history of SCD</i></p> <ul style="list-style-type: none"> • Increased voltage of QRS complex • Incomplete RBBB • Early repolarization and ST segment elevation • In Black athletes, ST elevation with T wave inversions in leads V1 – V4 • In athletes ≤ 16 years old, T wave inversions in V1 – V3 • Sinus bradycardia • Sinus arrhythmia • Ectopic atrial rhythm • Junctional rhythm • First degree AV block • Mobitz Type 1, second degree AV block | <p><i>No further evaluation needed if 1 ECG findings in asymptomatic athlete without family history of SCD. If 2 or more of these are present, further screening is required.</i></p> <ul style="list-style-type: none"> • Left axis deviation • Left atrial enlargement • Right axis deviation • Right atrial enlargement • Complete RBBB | <p><i>Further evaluation needed if any present</i></p> <ul style="list-style-type: none"> • T wave inversion • ST segment depression • Pathologic Q waves • Complete LBBB • QRS duration ≥ 140 ms • Epsilon wave • Ventricular pre-excitation • Long QT interval • Type 1 Brugada pattern • Sinus bradycardia that is < 30 bpm • PR interval ≥ 400 ms • Mobitz Type II, second degree AV block • Third degree AV block • ≥ 2 PVCs • Atrial Tachyarrhythmias • Ventricular arrhythmias |

Fig. 3 The International Criteria for ECG interpretation in athletes detailing low, borderline, and high-risk ECG findings (adapted from Drezner et al., 2017) [61]. Abbreviations: ECG=electrocardiogram;

SCD=sudden cardiac death; RBBB=right bundle branch block; AV=atrioventricular; PVC=premature ventricular contraction

an abnormal screening TTE [71]. A study of 1628 athletes in West Asia that screened using both TTE and ECG found that TTE screening was ineffective from either a clinical or economic standpoint [72]. Despite this data, a 2021 survey of 603 healthcare professionals across 97 counties, 68% of respondents use TTE “always” or “often” in the routine pre-participation screening of asymptomatic athletes [73]. There is a clear disconnect between this data, the multiple societies recommending against routine TTE screening, and the practice found among real-world practitioners in survey data [73].

While TTE is a beneficial secondary screening test to further evaluate abnormalities on primary screening, it has limitations that preclude it from being an effective primary screening tool. First, TTE is only able to assess for certain structural cardiac diseases that represent a small fraction of cardiac abnormalities associated with SCD. It is unable to detect most non-structural cardiac diseases and has only limited ability to detect some structural diseases such as ARVD, which is a major contributor to SCD. Second, despite screening for a limited number of pathologies, it carries significant cost though some have recommended a limited TTE screening to decrease cost but at the expense of decreased sensitivity. Third, those who routinely engage in vigorous exercise have cardiac adaptations that can closely mimic cardiovascular pathology, often termed “athlete’s heart” [74, 75]. For example, RV dilation can be seen as both a physiologic adaptation of athletes and as a marker of ARVC, and distinguishing the two often requires multi-modality imaging beyond standard TTE [76]. Similar overlap with “athlete’s heart” can also be seen in TTE findings of HCM and dilated cardiomyopathies [70].

Future Directions

While ACC and AHA guidelines recommend against mass, universal, mandated screening programs, they do allow for consideration of small screening programs for children and adolescents that are led by a team familiar with the inherent limitations of screening. This is an important distinction from the misconception that these organizations have a blanket guideline against screening [52]. Limiting screening programs to a smaller size allows for closer monitoring by a physician leader who is familiar with PPE and poses less logistic challenge in initiating the program. Even within the ESC recommendations for widespread screening, they acknowledge that “the proposed screening protocol is at present difficult to implement in all European countries” underscoring

the immense resources that would be required for execution [56]. Careful consideration should be given prior to starting a screening program as a poorly implemented screening program is likely less helpful, and possibly harmful, than not screening at all.

The ideal screening program maximizes the likelihood of detecting cardiac conditions associated with SCD while attempting to minimize burden on the overall healthcare system. While the ideal method for screening PPEs has yet to be determined, we believe a widespread, one-size-fits-all screening paradigm for all athletes is likely not the solution to this challenge. Just as other routine screening tests are only recommended for certain populations (e.g., mammography for women or abdominal aortic aneurysm screening in high-risk tobacco users), we advocate for a more personalized approach that caters the depth of screening to the patient’s existing risk factors for SCD as well as local resources and expertise. While further research is needed to determine the exact screening paradigm, an example could consist of the lowest risk patients being screened with history and physical alone and additional cardiac testing being added in those with increasing risk for SCD.

No screening program will be capable of preventing 100% of SCD, so the development and rehearsal of an emergency action plan (EAP), often between multiple stakeholders such as coaches and emergency medical services, is crucial to preventing mortality if an arrest were to occur [77]. A key component of EAPs is close access to automated external defibrillators (AEDs), which have been shown to almost double survival in out-of-hospital arrests (odds ratio 1.75, $p < 0.002$) [78]. The effective implementation and performance of an EAP can be a matter of life or death for an athlete who unexpectedly suffers arrest.

Christian Eriksen is a professional soccer player from Denmark who had been screened for cardiac conditions associated with SCD several times during his career. While competing in the 2020 European Football Championship, Eriksen suffered SCA and collapsed mid-match. Stadium medical staff promptly began resuscitation efforts with cardiopulmonary resuscitation, and an AED shocked him out of the malignant arrhythmia [79]. Eriksen was carted off the field conscious and was transported directly to the hospital [80]. He later underwent placement of an implantable cardioverter defibrillator [79]. This success story underscores the inherent limitations of SCD screening given that Eriksen had been screened multiple times in the decade preceding his arrest. It also stresses the importance of close access to AEDs and preparedness with EAPs. While controversy exists in many elements of screening for SCD, no debate exists for EAPs, which are responsible for saving the life of Eriksen.

Conclusion

Sudden cardiac death (SCD) in a young athlete is an infrequent yet devastating event often associated with substantial media attention. Efforts to screen athletes for cardiac conditions commonly associated with SCD is a controversial topic with debate surrounding virtually each component including the ideal subject, method, and performer/interpreter of such screens, resulting in disparate recommendations among major medical organizations and screening policies between sporting associations. While no screening program will be able to prevent all SCD, future efforts should be focused on personalizing screening recommendations to the individual athlete and developing small screening programs run by physicians familiar with the intricacies of the examination of athletes.

Declarations

Conflict of Interest The authors do not have existing conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol.* 2003;42(11):1959–63.
2. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation.* 2009;119(8):1085–92.
3. Hayashi M, Shimizu W, Albert CM. The spectrum of epidemiology underlying sudden cardiac death. *Circ Res.* 2015;116(12):1887–906.
4. Kong MH, Fonarow GC, Peterson ED, Curtis AB, Hernandez AF, Sanders GD, Thomas KL, Hayes DL, Al-Khatib SM. Systematic review of the incidence of sudden cardiac death in the United States. *J Am Coll Cardiol.* 2011;57(7):794–801.
5. Steinvil A, Chundadze T, Zeltser D, Rogowski O, Halkin A, Galily Y, Perluk H, Viskin S. Mandatory electrocardiographic screening of athletes to reduce their risk for sudden death proven fact or wishful thinking? *J Am Coll Cardiol.* 2011;57(11):1291–6.
6. Van Camp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc.* 1995;27(5):641–7.

7. Harmon KG, Drezner JA, Wilson MG, Sharma S. Incidence of sudden cardiac death in athletes: a state-of-the-art review. *Heart.* 2014;100(16):1227–34.
8. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA.* 2006;296(13):1593–601.
9. Marijon E, Tafflet M, Celermajer DS, Dumas F, Perier MC, Mustafic H, Toussaint JF, Desnos M, Rieu M, Benamer N, Le Heuzey JY, Empana JP, Jouven X. Sports-related sudden death in the general population. *Circulation.* 2011;124(6):672–81.
10. Marijon E, Uy-Evanado A, Reinier K, Teodorescu C, Narayanan K, Jouven X, Gunson K, Jui J, Chugh SS. Sudden cardiac arrest during sports activity in middle age. *Circulation.* 2015;131(16):1384–91.
11. Stecker EC, Reinier K, Marijon E, Narayanan K, Teodorescu C, Uy-Evanado A, Gunson K, Jui J, Chugh SS. Public health burden of sudden cardiac death in the United States. *Circ Arrhythm Electrophysiol.* 2014;7(2):212–7.
12. Holst AG, Winkel BG, Theilade J, Kristensen IB, Thomsen JL, Ottesen GL, Svendsen JH, Haunsø S, Prescott E, Tfelt-Hansen J. Incidence and etiology of sports-related sudden cardiac death in Denmark—implications for preparticipation screening. *Heart Rhythm.* 2010;7(10):1365–71.
13. Solberg EE, Gjertsen F, Haugstad E, Kolsrud L. Sudden death in sports among young adults in Norway. *Eur J Cardiovasc Prev Rehabil.* 2010;17(3):337–41.
14. Roberts WO, Stovitz SD. Incidence of sudden cardiac death in Minnesota high school athletes 1993–2012 screened with a standardized pre-participation evaluation. *J Am Coll Cardiol.* 2013;62(14):1298–301.
15. Maron BJ, Haas TS, Murphy CJ, Ahluwalia A, Rutten-Ramos S. Incidence and causes of sudden death in U.S. college athletes. *J Am Coll Cardiol.* 2014;63(16):1636–43.
16. Harmon KG, Zigman M, Drezner JA. The effectiveness of screening history, physical exam, and ECG to detect potentially lethal cardiac disorders in athletes: a systematic review/meta-analysis. *J Electrocardiol.* 2015;48(3):329–38.
17. Drezner JA, Harmon KG, Marek JC. Incidence of sudden cardiac arrest in Minnesota high school student athletes: the limitations of catastrophic insurance claims. *J Am Coll Cardiol.* 2014;63(14):1455–6.
18. Emery MS, Kovacs RJ. Sudden cardiac death in athletes. *JACC Heart Fail.* 2018;6(1):30–40.
19. Harmon KG, Asif IM, Maleszewski JJ, Owens DS, Prutkin JM, Salerno JC, Zigman ML, Ellenbogen R, Rao AL, Ackerman MJ, Drezner JA. Incidence, cause, and comparative frequency of sudden cardiac death in National Collegiate Athletic Association athletes: a decade in review. *Circulation.* 2015;132(1):10–9.
20. de Noronha SV, Sharma S, Papadakis M, Desai S, Whyte G, Sheppard MN. Aetiology of sudden cardiac death in athletes in the United Kingdom: a pathological study. *Heart.* 2009;95(17):1409–14.
21. Finocchiaro G, Papadakis M, Robertus JL, Dhutia H, Steriottis AK, Tome M, Mellor G, Merghani A, Malhotra A, Behr E, Sharma S, Sheppard MN. Etiology of sudden death in sports: insights from a United Kingdom regional registry. *J Am Coll Cardiol.* 2016;67(18):2108–15.
22. Maron BJ, Haas TS, Ahluwalia A, Murphy CJ, Garberich RF. Demographics and epidemiology of sudden deaths in young competitive athletes: from the United States National Registry. *Am J Med.* 2016;129(11):1170–7.
23. Peterson DF, Kucera K, Thomas LC, Maleszewski J, Siebert D, Lopez-Anderson M, Zigman M, Schattenkerk J, Harmon KG, Drezner JA. Aetiology and incidence of sudden cardiac arrest

- and death in young competitive athletes in the USA: a 4-year prospective study. *Br J Sports Med.* 2021;55(21):1196–203.
24. de Noronha SV, Behr ER, Papadakis M, Ohta-Ogo K, Banya W, Wells J, Cox S, Cox A, Sharma S, Sheppard MN. The importance of specialist cardiac histopathological examination in the investigation of young sudden cardiac deaths. *Europace.* 2014;16(6):899–907.
 25. Harmon KG, Drezner JA, Maleszewski JJ, Lopez-Anderson M, Owens D, Prutkin JM, Asif IM, Klossner D, Ackerman MJ. Pathogenesis of sudden cardiac death in national collegiate athletic association athletes. *Circ Arrhythm Electrophysiol.* 2014;7(2):198–204.
 26. Corrado D, Basso C, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden cardiac death? *J Cardiovasc Med (Hagerstown).* 2006;7(4):228–33.
 27. Maron BJ. Clinical course and management of hypertrophic cardiomyopathy. *N Engl J Med.* 2018;379(7):655–68.
 28. Neubauer S, Kolm P, Ho CY, Kwong RY, Desai MY, Dolman SF, Appelbaum E, Desvigne-Nickens P, DiMarco JP, Friedrich MG, Geller N, Harper AR, Jarolim P, Jerosch-Herold M, Kim DY, Maron MS, Schulz-Menger J, Piechnik SK, Thomson K, Zhang C, Watkins H, Weintraub WS, Kramer CM. Distinct subgroups in hypertrophic cardiomyopathy in the NHLBI HCM Registry. *J Am Coll Cardiol.* 2019;74(19):2333–45.
 29. Gandjbakhch E, Redheuil A, Pousset F, Charron P, Frank R. Clinical diagnosis, imaging, and genetics of arrhythmogenic right ventricular cardiomyopathy/dysplasia: JACC State-of-the-Art Review. *J Am Coll Cardiol.* 2018;72(7):784–804.
 30. Basso C, Corrado D, Bauce B, Thiene G. Arrhythmogenic right ventricular cardiomyopathy. *Circ Arrhythm Electrophysiol.* 2012;5(6):1233–46.
 31. Gentile F, Castiglione V, De Caterina R. Coronary artery anomalies. *Circulation.* 2021;144(12):983–96.
 32. Cheezum MK, Liberthson RR, Shah NR, Villines TC, O’Gara PT, Landzberg MJ, Blankstein R. anomalous aortic origin of a coronary artery from the inappropriate sinus of Valsalva. *J Am Coll Cardiol.* 2017;69(12):1592–608.
 33. Angelini P. Coronary artery anomalies: an entity in search of an identity. *Circulation.* 2007;115(10):1296–305.
 34. Bigler MR, Ashraf A, Seiler C, Praz F, Ueki Y, Windecker S, Kadner A, Räber L, Gräni C. Hemodynamic relevance of anomalous coronary arteries originating from the opposite sinus of Valsalva-in search of the evidence. *Front Cardiovasc Med.* 2020;7: 591326.
 35. Grollman JH Jr, Mao SS, Weinstein SR. Arteriographic demonstration of both kinking at the origin and compression between the great vessels of an anomalous right coronary artery arising in common with a left coronary artery from above the left sinus of Valsalva. *Cathet Cardiovasc Diagn.* 1992;25(1):46–51.
 36. Kindermann I, Barth C, Mahfoud F, Ukena C, Lenski M, Yilmaz A, Klingel K, Kandolf R, Sechtem U, Cooper LT, Böhm M. Update on myocarditis. *J Am Coll Cardiol.* 2012;59(9):779–92.
 37. Peretto G, Sala S, Rizzo S, De Luca G, Campochiaro C, Sartorelli S, Benedetti G, Palmisano A, Esposito A, Tresoldi M, Thiene G, Basso C, Della Bella P. Arrhythmias in myocarditis: State of the art. *Heart Rhythm.* 2019;16(5):793–801.
 - 38.●● Gluckman TJ, Bhavane NM, Allen LA, Chung EH, Spatz ES, Ammirati E, Baggish AL, Bozkurt B, Cornwell WK 3rd, 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;79(17):1717–56. **Importance:**
 39. Moulson N, Petek BJ, Drezner JA, Harmon KG, Kliethermes SA, Patel MR, Baggish AL. SARS-CoV-2 cardiac involvement in young competitive athletes. *Circulation.* 2021;144(4):256–66.
 40. Daniels CJ, Rajpal S, Greenshields JT, Rosenthal GL, Chung EH, Terrin M, Jeudy J, Mattson SE, Law IH, Borchers J, Kovacs R, Kovan J, Rifat SF, Albrecht J, Bento AI, Albers L, Bernhardt D, Day C, Hecht S, Hipskind A, Mjaanes J, Olson D, Rooks YL, Somers EC, Tong MS, Wisinski J, Womack J, Esopenko C, Kratochvil CJ, Rink LD. Prevalence of clinical and subclinical myocarditis in competitive athletes with recent SARS-CoV-2 infection: results from the big ten COVID-19 cardiac registry. *JAMA Cardiol.* 2021;6(9):1078–87.
 41. Martinez MW, Tucker AM, Bloom OJ, Green G, DiFiori JP, Solomon G, Phelan D, Kim JH, Meeuwisse W, Sills AK, Rowe D, Bogoch II, Smith PT, Baggish AL, Putukian M, Engel DJ. Prevalence of inflammatory heart disease among professional athletes with prior COVID-19 infection who received systematic return-to-play cardiac screening. *JAMA Cardiol.* 2021;6(7):745–52.
 - 42.●● Petek BJ, Moulson N, Drezner JA, Harmon KG, Kliethermes SA, Churchill TW, Patel MR, Baggish AL. Cardiovascular outcomes in collegiate athletes following SARS-CoV-2 infection: 1-year follow-up from the outcomes registry for cardiac conditions in athletes. *Circulation.* 2022. **Importance: Demonstrated that the incidence of cardiac involvement of those who had SARS-CoV-2 infection and showed that cardiac complications were rare for those with myocardial involvement.**
 43. Estes NA 3rd. Sudden cardiac arrest from primary electrical diseases: provoking concealed arrhythmogenic syndromes. *Circulation.* 2005;112(15):2220–1.
 44. Priori SG, Napolitano C, Grillo M. Concealed arrhythmogenic syndromes: the hidden substrate of idiopathic ventricular fibrillation? *Cardiovasc Res.* 2001;50(2):218–23.
 45. Wever EF, Robles de Medina EO. Sudden death in patients without structural heart disease. *J Am Coll Cardiol.* 2004;43(7):1137–44.
 46. Bagnall RD, Weintraub RG, Ingles J, Dufflou J, Yeates L, Lam L, Davis AM, Thompson T, Connell V, Wallace J, Naylor C, Crawford J, Love DR, Hallam L, White J, Lawrence C, Lynch M, Morgan N, James P, du Sart D, Puranik R, Langlois N, Vohra J, Winship I, Atherton J, McGaughran J, Skinner JR, Semsarian C. A prospective study of sudden cardiac death among children and young adults. *N Engl J Med.* 2016;374(25):2441–52.
 47. Isbister JC, Nowak N, Butters A, Yeates L, Gray B, Sy RW, Ingles J, Bagnall RD, Semsarian C. “Concealed cardiomyopathy” as a cause of previously unexplained sudden cardiac arrest. *Int J Cardiol.* 2021;324:96–101.
 - 48.●● A.A. American Academy of Family Physicians, P. American Academy of, M. American College of Sports, A.M. American Medical Society for Sports Medicine. PPE: Preparticipation Physical Evaluation, American Academy of Pediatrics, Elk Grove Village, UNITED STATES, 2019. **Importance: A widely utilized resource detailing the preparticipation physical examination that is endorsed by many relevant medical associations.**
 49. Maron BJ, Friedman RA, Kligfield P, Levine BD, Viskin S, Chaitman BR, Okin PM, Saul JP, Salberg L, Van Hare GF, Soliman EZ, Chen J, Matherne GP, Bolling SF, Mitten MJ, Caplan A, Balady GJ, Thompson PD. Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12–25 Years of Age): a scientific statement from the American Heart Association and the American College of Cardiology. *Circulation.* 2014;130(15):1303–34.

50. Maron BJ, Thompson PD, Puffer JC, McGrew CA, Strong WB, Douglas PS, Clark LT, Mitten MJ, Crawford MH, Atkins DL, Driscoll DJ, Epstein AE. Cardiovascular preparticipation screening of competitive athletes. A statement for health professionals from the Sudden Death Committee (clinical cardiology) and Congenital Cardiac Defects Committee (cardiovascular disease in the young), American Heart Association. *Circulation*. 1996;94(4):850–6.
51. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, Dimeff R, Douglas PS, Glover DW, Hutter AM Jr, Krauss MD, Maron MS, Mitten MJ, Roberts WO, Puffer JC. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation*. 2007;115(12):1643–2455.
52. Maron BJ, Levine BD, Washington RL, Baggish AL, Kovacs RJ, Maron MS. Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 2: Preparticipation Screening for Cardiovascular Disease in Competitive Athletes: A Scientific Statement From the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66(21):2356–61.
53. Parizher G, Putzke JD, Lampert R, Emery MS, Baggish A, Martinez M, Levine A, Levine BD. Web-based multimedia athlete preparticipation questionnaire: introducing the video-PPE (v-PPE). *Br J Sports Med*. 2020;54(1):67–8. **Importance: A study demonstrating the effectiveness of a novel, web-based preparticipation examination format that could expand access to screening examinations in this comporary era that is increasingly utilizing telemedicine.**
54. Maron BJ, Levine BD, Washington RL, Baggish AL, Kovacs RJ, Maron MS. Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 2: Preparticipation Screening for Cardiovascular Disease in Competitive Athletes: A Scientific Statement From the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e267–72.
55. Drezner JA, O'Connor FG, Harmon KG, Fields KB, Asplund CA, Asif IM, Price DE, Dimeff RJ, Bernhardt DT, Roberts WO. AMSSM position statement on cardiovascular preparticipation screening in athletes: current evidence, knowledge gaps, recommendations and future directions. *Br J Sports Med*. 2017;51(3):153–67.
56. Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, Panhuyzen-Goedkoop N, Deligiannis A, Solberg E, Dugmore D, Mellwig KP, Assanelli D, Delise P, van-Buuren F, Anastasakis A, Heidbuchel H, Hoffmann E, Fagard R, Priori SG, Basso C, Arbustini E, Blomstrom-Lundqvist C, McKenna WJ, Thiene G. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26(5):516–24.
57. Mont L, Pelliccia A, Sharma S, Biffi A, Borjesson M, Brugada-Terradellas J, Carré F, Guasch E, Heidbuchel H, La Gerche A, Lampert R, McKenna W, Papadakis M, Priori SG, Scanavacca M, Thompson P, Sticherling C, Viskin S, Wilson M, Corrado D, Lip GY, Gorenek B, Blomström-Lundqvist C, Merkely B, Hindricks G, Hernández-Madrid A, Lane D, Boriani G, Narasimhan C, Marquez MF, Haines D, Mackall J, Manuel Marques-Vidal P, Corra U, Halle M, Tiberi M, Niebauer J, Piepoli M. Pre-participation cardiovascular evaluation for athletic participants to prevent sudden death: Position paper from the EHRA and the EACPR, branches of the ESC. Endorsed by APHRS, HRS, and SOLAECE. *Eur J Prev Cardiol*. 2017;24(1):41–69.
58. Williams EA, Pelto HF, Toresdahl BG, Prutkin JM, Owens DS, Salerno JC, Harmon KG, Drezner JA. Performance of the American Heart Association (AHA) 14-Point Evaluation Versus Electrocardiography for the Cardiovascular Screening of High School Athletes: A Prospective Study. *J Am Heart Assoc*. 2019;8(14):e012235. **Importance: Recent study providing evidence that screening ECG is more effective than the standard AHA 14-point preparticipation examination.**
59. Baggish AL, Hutter AM Jr, Wang F, Yared K, Weiner RB, Kupperman E, Picard MH, Wood MJ. Cardiovascular screening in college athletes with and without electrocardiography: A cross-sectional study. *Ann Intern Med*. 2010;152(5):269–75.
60. Maron BJ, Haas TS, Doerer JJ, Thompson PD, Hodges JS. Comparison of U.S. and Italian experiences with sudden cardiac deaths in young competitive athletes and implications for preparticipation screening strategies. *Am J Cardiol*. 2009;104(2):276–80.
61. Drezner JA, Sharma S, Baggish A, Papadakis M, Wilson MG, Prutkin JM, Gerche A, Ackerman MJ, Borjesson M, Salerno JC, Asif IM, Owens DS, Chung EH, Emery MS, Froelicher VF, Heidbuchel H, Adamuz C, Asplund CA, Cohen G, Harmon KG, Marek JC, Molossi S, Niebauer J, Pelto HF, Perez MV, Riding NR, Saarel T, Schmied CM, Shipon DM, Stein R, Vetter VL, Pelliccia A, Corrado D. International criteria for electrocardiographic interpretation in athletes: Consensus statement. *Br J Sports Med*. 2017;51(9):704–31.
62. Magee C, Kazman J, Haigney M, Oriscello R, DeZee KJ, Deuster P, Depenbrock P, O'Connor FG. Reliability and validity of clinician ECG interpretation for athletes. *Ann Noninvasive Electrocardiol*. 2014;19(4):319–29.
63. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. *N Engl J Med*. 1998;339(6):364–9.
64. Corrado D, Pelliccia A, Heidbuchel H, Sharma S, Link M, Basso C, Biffi A, Buja G, Delise P, Gussac I, Anastasakis A, Borjesson M, Bjørnstad HH, Carré F, Deligiannis A, Dugmore D, Fagard R, Hoogsteen J, Mellwig KP, Panhuyzen-Goedkoop N, Solberg E, Vanhees L, Drezner J, Estes NA 3rd, Illiceto S, Maron BJ, Peidro R, Schwartz PJ, Stein R, Thiene G, Zeppilli P, McKenna WJ. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *Eur Heart J*. 2010;31(2):243–59.
65. Drezner JA, Ackerman MJ, Anderson J, Ashley E, Asplund CA, Baggish AL, Börjesson M, Cannon BC, Corrado D, DiFiori JP, Fischbach P, Froelicher V, Harmon KG, Heidbuchel H, Marek J, Owens DS, Paul S, Pelliccia A, Prutkin JM, Salerno JC, Schmied CM, Sharma S, Stein R, Vetter VL, Wilson MG. Electrocardiographic interpretation in athletes: the “Seattle criteria.” *Br J Sports Med*. 2013;47(3):122–4.
66. Sheikh N, Papadakis M, Ghani S, Zaidi A, Gati S, Adami PE, Carré F, Schnell F, Wilson M, Avila P, McKenna W, Sharma S. Comparison of electrocardiographic criteria for the detection of cardiac abnormalities in elite black and white athletes. *Circulation*. 2014;129(16):1637–49.
67. Malhotra A, Dhutia H, Yeo TJ, Finocchiaro G, Gati S, Bulleros P, Fanton Z, Papatheodorou E, Miles C, Ketepee-Arachi T, Basu J, Parry-Williams G, Prakash K, Gray B, D'Silva A, Ensam B, Behr E, Tome M, Papadakis M, Sharma S. Accuracy of the 2017 international recommendations for clinicians who interpret adolescent athletes' ECGs: a cohort study of 11 168 British white and black soccer players. *Br J Sports Med*. 2020;54(12):739–45. **Importance: Large study demonstrating the improved accuracy of the International Criteria for ECG screening.**

68. ●● Malhotra A, Dhutia H, Finocchiaro G, Gati S, Beasley I, Clift P, Cowie C, Kenny A, Mayet J, Oxborough D, Patel K, Pieles G, Rakhit D, Ramsdale D, Shapiro L, Somauroo J, Stuart G, Varnava A, Walsh J, Yousef Z, Tome M, Papadakis M, Sharma S. Outcomes of cardiac screening in adolescent soccer players. *N Engl J Med*. 2018;379(6):524–34. **Importance: In addition to other significant findings, this study highlighted the imperfect nature of screening and showed that no screening program will be able to fully prevent SCD.**
69. Christiaans I, van Engelen K, van Langen IM, Birnie E, Bonsel GJ, Elliott PM, Wilde AA. Risk stratification for sudden cardiac death in hypertrophic cardiomyopathy: systematic review of clinical risk markers. *Europace*. 2010;12(3):313–21.
70. Niederseer D, Rossi VA, Kissel C, Scherr J, Caselli S, Tanner FC, Bohm P, Schmied C. Role of echocardiography in screening and evaluation of athletes. *Heart*. 2020.
71. Berge HM, Andersen TE, Bahr R. Cardiovascular incidents in male professional football players with negative preparticipation cardiac screening results: an 8-year follow-up. *Br J Sports Med*. 2019;53(20):1279–84.
72. Riding NR, Sharma S, Salah O, Khalil N, Carré F, George KP, Hamilton B, Chalabi H, Whyte GP, Wilson MG. Systematic echocardiography is not efficacious when screening an ethnically diverse cohort of athletes in West Asia. *Eur J Prev Cardiol*. 2015;22(2):263–70.
73. D'Ascenzi F, Anselmi F, Mondillo S, Finocchiaro G, Caselli S, Garza MS, Schmied C, Adami PE, Galderisi M, Adler Y, Pantazis A, Niebauer J, Heidebuchel H, Papadakis M, Dendale P. The use of cardiac imaging in the evaluation of athletes in the clinical practice: A survey by the Sports Cardiology and Exercise Section of the European Association of Preventive Cardiology and University of Siena, in collaboration with the European Association of Cardiovascular Imaging, the European Heart Rhythm Association and the ESC Working Group on Myocardial and Pericardial Diseases. *Eur J Prev Cardiol*. 2021;28(10):1071–7.
74. Kovacs R, Baggish AL. Cardiovascular adaptation in athletes. *Trends Cardiovasc Med*. 2016;26(1):46–52.
75. Maron BJ, Pelliccia A. The heart of trained athletes: cardiac remodeling and the risks of sports, including sudden death. *Circulation*. 2006;114(15):1633–44.
76. Baucé B, Frigo G, Benini G, Michieli P, Basso C, Folino AF, Rigato I, Mazzotti E, Daliento L, Thiene G, Nava A. Differences and similarities between arrhythmogenic right ventricular cardiomyopathy and athlete's heart adaptations. *Br J Sports Med*. 2010;44(2):148–54.
77. Link MS, Myerburg RJ, Estes NA 3rd. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task Force 12: emergency action plans, resuscitation, cardiopulmonary resuscitation, and automated external defibrillators: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e334–8.
78. Weisfeldt ML, Sitlani CM, Ornato JP, Rea T, Aufderheide TP, Davis D, Dreyer J, Hess EP, Jui J, Maloney J, Sopko G, Powell J, Nichol G, Morrison LJ. Survival after application of automatic external defibrillators before arrival of the emergency medical system: evaluation in the resuscitation outcomes consortium population of 21 million. *J Am Coll Cardiol*. 2010;55(16):1713–20.
79. Professor Sanjay Sharma explains Christian Eriksen's collapse at Euro 2020, St. George's University of London Website; 2021.
80. Sharland P. Christian Eriksen: Euro 2020 Match Between Denmark v Finland Restarted after Midfielder Collapses on Pitch; 2021.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Pediatric Head Trauma: A Review and Update

Rose N. Gelineau-Morel, MD,* Timothy P. Zinkus, MD,[†] Jean-Baptiste Le Pichon, MD, PhD*

*Division of Neurology and [†]Division of Radiology, Children's Mercy Hospital, Kansas City, MO

Practice Gaps

There is still a considerable amount of confusion when it comes to managing concussions. An excessive number of head computed tomographic scans are being obtained for concussions, resulting in unnecessary exposure to ionizing radiation. Clinicians should be aware of the most recent guidelines for the management of concussion, including the need for imaging, and should be able to differentiate mild from moderate and severe traumatic brain injury.

Objectives After completing this article, readers should be able to:

1. Differentiate a mild from a moderate or severe traumatic brain injury (TBI).
2. Acutely manage a child with a TBI, including deciding when further imaging is necessary.
3. Manage a child with a postconcussion syndrome and identify when referral to a specialist is necessary.

Traumatic brain injury (TBI) is the leading cause of death or severe disability in children older than 1 year. (1)(2) In a report to Congress published by the Centers for Disease Control and Prevention (CDC) in 2018, (3) the CDC reported the public health burden of TBIs. They noted that 640,000 emergency department visits and 18,000 hospital stays were directly related to TBI. The etiology of TBI varies among age groups. In the 0- to 4-year-old age group, the most common cause of TBI is falls. On the other hand, in the 15- to 24-year-old age group the distribution of injuries caused by falls, assault, and motor vehicle events are nearly equal. Epidemiologic studies have found that rates of TBI seen in the emergency department have increased in all age groups since 2001, with children 0 to 24 years old having the highest rates of TBI of all age groups. Children 0 to 4 years old have almost twice the rate of TBI compared with the next highest age group (15–24 years old), making pediatric traumatic brain injury an especially salient topic for the modern-day pediatrician. (4) Moreover, 61% of children with moderate to severe TBI experienced a disability. Estimates conclude that at least 145,000 children aged 0 to 19 years are currently living with long-term symptoms due to a TBI (likely an underestimate with underreporting of mild TBI [mTBI]), with

AUTHOR DISCLOSURE Drs Gelineau-Morel and Zinkus have disclosed no financial relationships relevant to this article. Dr Le Pichon has disclosed that he has served as a consultant for an AADC Deficiency Advisors Forum and as a medical legal expert and that he currently serves as the chair of the Subcommittee on Education for the American Academy of Pediatrics Section on Neurology Committee. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

ABBREVIATIONS

| | |
|-------------|--|
| AAP | American Academy of Pediatrics |
| AHT | abusive head trauma |
| CDC | Centers for Disease Control and Prevention |
| Child SCAT5 | Child Sport Concussion Assessment Tool |
| CISG | Concussion in Sport Group |
| CT | computed tomography |
| DAI | diffuse axonal injury |
| FLAIR | fluid-attenuated inversion recovery |
| GCS | Glasgow Coma Scale |
| MRI | magnetic resonance imaging |
| mTBI | mild traumatic brain injury |
| TBI | traumatic brain injury |

symptoms extending far beyond their initial hospital visit into the following months and years. (3) Even children without overt neurologic deficits resulting from their TBI can demonstrate impairment in academic performance, attention and concentration, memory, and executive function, some of which only become apparent months or years after the initial injury. (4)(5)(6) The economic impact of TBI is substantial, with estimates ranging from \$77.9 million per year in direct costs to more than \$1 billion per year for TBI-associated hospitalizations. (5)

With all the associated morbidity and mortality, it is vital that pediatricians are educated in recognizing and treating TBIs and their sequelae. In this article, we aim to provide current evidence on the recognition, treatment, and rehabilitation of TBIs. We begin by discussing mTBI, typically manifesting as a concussion, and then discuss moderate and severe TBIs that are more often encountered in the emergency department or hospital setting.

MILD TBI

mTBI commonly manifests as concussion, and this is the focus of our discussion. However, it is also worth noting that even patients with more severe brain injury can exhibit signs and symptoms of concussion, and these should not be ignored. Concussion is a broad clinical diagnosis defined by the American Academy of Neurology as “a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness.” (6) Due to various mechanisms of action for concussion, and the multiple disciplines involved, including neurology, sports medicine, rehabilitation medicine, and military medicine, there are multiple diagnostic criteria and treatment recommendations in place, which can make it challenging for the primary care provider evaluating a patient with a concussion. (7) Yet, concussion is a common complaint in children, occurring in approximately 692 of 100,000 children younger than 15 years, (8) indicating that an evidence-based, comprehensive plan for concussion diagnosis and management is imperative. (7)

In this section, we aim to summarize current recommendations on concussion evaluation and management for the general pediatrician based on guidelines for concussion management developed by the American Academy of Neurology, (6) the CDC, (9)(10) and the Concussion in Sport Group (CISG), an international multidisciplinary group of clinicians and researchers focused on concussion diagnosis and management. (11) The CDC recently published new guidelines on concussion management, and these recommendations are reflected herein. (10) Although concussion

can be caused by any mechanical force on the brain, we focus on sports-related concussion because this is the focus of most research and clinical guidelines and is the most common presentation of concussion for the general pediatrician. (7) Note that although sports-related concussions have been studied most extensively, it is probable that most of the recommendations made thereafter apply equally well to concussions related to other accidental and nonaccidental injuries. In fact, in their most recent recommendations, the CDC (10) does not differentiate between these types of concussions as it relates to diagnosis and management.

Recognizing Concussion

The first step to treating concussion is recognition. The clinical phenotype of concussion can vary between patients, and it can be a challenge for the evaluating clinician to consider all the possible manifestations of concussion. One useful acronym is COACH CV, which was developed by Craton et al (12) and is based on the CISG guidelines. This acronym includes the most common clinical phenotypes of concussion: Cognitive dysfunction, Oculomotor dysfunction, Affective disturbances, Cervical spine disorders, Headaches, and Cardiovascular and Vestibular anomalies (Table 1).

Symptoms of concussion in these domains are broad and include impairment of memory or attention, blurred vision or abnormal extraocular movements, fatigue, mood changes, poor sleep, headaches, vestibular dysfunction, or heart rate variability (see Table 1 for a more extensive list of potential concussion symptoms). Of note, a patient with a concussion may have 1 or more of these symptoms. There is no loss of consciousness required for a diagnosis of concussion.

In their recent recommendations for the diagnosis and management of concussion, the CDC recommends using a validated symptom rating scale in the evaluation of concussion. (10) The most commonly used tools include the Child Sport Concussion Assessment Tool (Child SCAT5) developed by the CISG, (13) the Acute Concussion Evaluation developed by the CDC, (9) the Postconcussion Symptom Scale, (14) and the Graded Symptom Checklist. (15)(16) These tools use a Likert scale completed by the patient and/or parent to assess symptom severity, with a higher score indicating more severe symptoms. Although some of the scales, specifically the Child SCAT5, were developed for sports-related concussion, the symptoms of concussion are generalizable to other concussion etiologies as well. Studies have further analyzed these scales, attempting to identify the underlying symptom groups contributing to higher scores in concussed patients (eg, neurocognitive, somatic, emotional), with mixed conclusions. (14)(15)(17) In practice, it is best to choose a scale and get baseline testing, followed by

TABLE 1. Clinical Phenotypes of Concussion (COACH CV)

| CLINICAL PHENOTYPE | SYMPTOMS | SPECIFIC TESTING |
|----------------------------|---|---|
| C Cognitive function | Memory impairment, decreased attention and concentration, slowed processing speed | Neuropsychological testing (in person or computer-based, such as ImPACT testing) |
| O Oculomotor dysfunction | Convergence insufficiency, blurred vision, abnormal saccades and/or smooth pursuit, photophobia | Visual acuity testing King-Devick test (assess saccadic eye movements) |
| A Affective disturbances | Fatigue, sadness, irritability, sleep disturbance, poor concentration, emotionality | Depression screen |
| C Cervical spine disorders | Neck pain, headaches, dizziness, balance difficulty | Neck range of motion Palpation of bones and muscles of the neck |
| H Headaches | Migrainous, tension-type, or cervicogenic headaches | – |
| C Cardiovascular anomaly | Exercise intolerance, heart rate variability or elevation, postural orthostatic tachycardia syndrome, autonomic dysfunction | Orthostatic vital signs Exercise stress test Tilt table testing |
| V Vestibular dysfunction | Dizziness, vertigo, balance difficulties | Romberg test Tandem gait Vestibulo-ocular reflex Balance Error Scoring System (see the Child Sport Concussion Assessment Tool) |

This table includes the common clinical phenotypes of concussion that patients may endorse on a symptom scale. Listed are the corresponding symptoms of each phenotype, as well as further testing that can be considered to assess each symptom. See Craton et al. (12)

testing at the time of injury and repeated testing throughout recovery to track changes in individual and total symptom scores. If symptoms in any 1 or more of the tested clinical domains are present, this suggests a diagnosis of concussion. (13)

If further delineation of symptoms is required, additional testing, either in the office or via a referral to the appropriate provider, can be considered. Some toolkits, such as the Child SCAT5, include further testing that can be performed by the provider to screen for certain concussion phenotypes. These include the Balance Error Scoring System, which assesses postural stability, and the Sensory Organization Test, which assesses the patient's equilibrium with altering visual and somatosensory inputs. However, these methods are not as sensitive at concussion diagnosis as the previously mentioned Likert rating scales. (6) Other targeted testing could include visual acuity for oculomotor dysfunction, orthostatic vital signs of cardiovascular dysfunction, or a detailed spinal neuromuscular examination to evaluate for cervical spine abnormalities (Table 1). (7)

Of note, computed tomography (CT) cannot be used to diagnose concussion and should generally be avoided to prevent unnecessary ionizing radiation exposure, although it may be used to rule out a more severe TBI, especially in patients with loss of consciousness, posttraumatic amnesia, persistent altered mental status, focal neurologic deficit,

evidence of skull fracture, or signs of clinical deterioration. (6) The Pediatric Emergency Care Applied Research Network (PECARN) has established criteria that can be used in decision making for children presenting after a TBI. The clinical criteria for children 2 years and older include normal mental status, no loss of consciousness, no vomiting, nonsevere injury mechanism, no signs of basilar skull fracture, and no severe headache. If all of these criteria are met, they demonstrate a negative predictive value of 99.95% for clinically important TBI. Conversely, the presence of any 1 of these predictors has sensitivity of 96.8% in identifying clinically important TBI and indicates that further assessment with head CT is required. Criteria for children younger than 2 years are also included in the study. (18) Unfortunately, unnecessary head CT in children remains a common concern, and further education of community providers can help reduce this unneeded radiation exposure. (19)

Although clinical discretion is still required to make a concussion diagnosis, the previously mentioned tools can help identify symptoms and track recovery, aiding the clinician in decisions regarding return to play and when to pursue referral or further testing. With the typical natural history of concussion, an athlete's symptoms should return to baseline in 2 weeks for adults and in 4 weeks for children. (20)

Rest and Return to Play

Once a concussion is diagnosed it is imperative that the patient is given the appropriate guidelines for rest and return to play/school. Previous recommendations for complete rest until symptom resolution are now outdated and likely stemmed from sports medicine literature in which there was a concern for second impact syndrome. Although this remains a concern, strict rest is not required for this entire period but rather guidelines now recommend complete rest for 24 to 48 hours, after which patients have a gradual return to full activity. In fact, a recent study comparing 2-day and 5-day strict rest periods in children with concussion demonstrated a slower resolution of symptoms in the group with a more prolonged rest. (21) After the initial rest period, children can follow a gradual return-to-activity protocol, which is outlined further in available toolkits, including the CDC Heads Up guidelines (9) and the Child SCAT5. The general strategy includes gradually increasing physical activity, beginning with nonaerobic daily activities

and progressing through graduated steps until full return to sport (Table 2). The child should take at least 24 hours for each step of the plan, with return to the previous step for any worsening of symptoms. A similar progression can be used for return to school activities for children whose symptoms are exacerbated by mental activities, beginning with a few days of rest at home, followed by a gradual return to school full time. (13) Nonessential cognitive activities, such as playing video games, should be introduced as tolerated once a child is back to normal or near-normal physical routine.

There is some evidence to suggest that the period for full physiologic and metabolic recovery from concussion may extend beyond that for clinical symptom recovery and that a repeat concussion during this period could further prolong recovery. The NCAA Concussion Study on collegiate football players found that 11 of the 12 players with a repeat concussion in one season experienced their second concussion within 10 days of their initial concussion, indicating that athletes are especially prone to recurrent concussion during this period.

TABLE 2. Return to Play Progression

| EXERCISE STEP | EXAMPLE ACTIVITIES | ACTIVITY TIME | GOAL OF EACH STEP |
|----------------------------|---|--|---|
| No activity | | Complete physical and cognitive rest for 24–48 h | |
| Nonaerobic activity | Normal daily activities that do not provoke symptoms | – | Reintegrate into work and school activities |
| Light aerobic activity | Exercise bike, walking, light jogging at a slow pace (no weight lifting, jumping, or running) | 5–10 min | Light activities leading to a mild increase in heart rate |
| Moderate activity | Jogging, brief running, moderate-intensity stationary biking, light resistance activities | Reduced from normal routine | Limited body and head movement |
| Heavy, noncontact activity | Running, noncontact drills, weight lifting, stationary biking | At or near normal routine | Intense activity without contact Cognitive activity during exercise can be added |
| Full contact | Normal full-contact physical activities | Normal routine | Return to full-contact activities |
| Competitive activities | Return to full competitive activities | Normal routine | No further restrictions in activity |

The table is adapted from the Centers for Disease Control and Prevention Heads Up guidelines and the Child Sport Concussion Assessment Tool for returning to play. Each step should take a minimum of 24 hours. During the above progression, the child, family, and health-care provider should pay special attention to any new or worsening symptoms. If any symptoms worsen while exercising, the child should return to the previous step.

(22) However, further details of time to complete physiologic recovery are not known and, at this time, it is only recommended that children have a “buffer” period of gradual return to full activity after complete symptom resolution. (23)

Although pharmacologic treatment has not been shown to facilitate recovery from concussion, it may be considered in patients who have longer recovery periods or whose quality of life is significantly affected by their symptoms. In these cases, treatment should focus on symptom management, including medications such as melatonin for sleep disturbance, nonopioid analgesics for acute headaches and amitriptyline or topiramate for headache prevention, and selective serotonin reuptake inhibitors or amantadine for emotional or cognitive effects, respectively. (24) Of note, in patients using pharmacotherapy, this could mask the symptoms of concussion, and these medications should be weaned or careful consideration should be given before returning to full play. (11)

Referral

Occasionally, the situation will still arise in which children do not have a complete recovery with the previously described strategies. Children with symptoms persisting beyond 4 weeks and adults with symptoms persisting beyond 2 weeks should be referred to a health-care provider specializing in concussion. (11) Studies have shown that higher symptom scores on immediate postconcussive testing can indicate more severe or prolonged postcognitive effects with a longer time for return to play. (25)

Certain preexisting conditions may delay concussion recovery, including history of postural orthostatic tachycardia syndrome, motion sickness, strabismus or ocular abnormalities, attention-deficit/hyperactivity disorder and learning disabilities, and mood disorders. These children may require additional school accommodations to facilitate their return. (26) Other factors such as history of previous concussions, more severe presenting or postconcussive symptoms, memory problems, fatigue/fogginess, and disorientation may also contribute to a more prolonged recovery. (6)(27) Some intrinsic factors, such as low socioeconomic status, Hispanic race, and high school age (especially in girls), also place children at risk for more prolonged symptoms compared with other patient populations. (10)(11)

Residual Effects

Unfortunately, approximately 10% to 15% of concussion patients have persistent symptoms beyond the first few weeks. (28) As recently as 2014, the *Diagnostic and Statistical Manual of Mental Disorders* criteria included a diagnosis of postconcussion syndrome, although this has now been

renamed “major or mild neurocognitive disorder due to TBI.” It is up to the clinician to consider the severity and functional disability of the patient when assigning a diagnosis. Postconcussive symptoms can be widely variable and depend on preexisting comorbidities, including neurocognitive disorders, vestibular dysfunction, affective symptoms, and medication/substance use. (27)

Additional evaluation and therapy should be considered for children with persistent postconcussive symptoms. Although most concussion patients will have normal cognitive function by 3 months after injury, some children could have cognitive deficits persisting up to 1 year, especially in the presence of a severe original injury, a history of previous concussions, and psychological risk factors. (29) These children should be referred for formal neuropsychological testing. Magnetic resonance imaging (MRI) can be more sensitive at detecting certain types of brain injury, such as diffuse axonal injury (DAI) or petechial hemorrhages, which are not detected in 25% to 30% of CT scans (see the “Management” subsection later herein). (30) Patients with vestibular or oculomotor dysfunction should be referred to the appropriate therapy for rehabilitation. Finally, the possibility of depression after concussion should not be ignored because it may often masquerade as cognitive or neurosensory dysfunction. (28)

Risk Reduction

General risk reduction for concussion centers around preventive safety measures to decrease risk of head injury. This includes the use of appropriate car seats, booster seats, or seat belts in automobiles; helmets while on bicycles or scooters; stair gates; and soft surfaces in play areas. (9)

However, the use of equipment in sports to protect against concussions is a controversial topic. The use of mouth guards does not seem to provide protection from concussion. (6)(31) Helmets and headgear have been shown to reduce the risk of concussion in skiing and snowboarding, but the effect in full-contact sports such as hockey and football has not been as conclusive. (31) This, in part, has led to the new rules for helmet contact implemented recently by the National Football League to reduce unnecessary risk of head injury in its players. (32)

Research on risk reduction for sports-related concussion has also focused on analyzing age, level of competition, sex, and type of sport to determine whether any individual factor can affect concussion symptoms and risk. A recent study found that females with concussion are more likely to report a higher level of symptoms and to experience postconcussive headaches, whereas males are more likely to experience loss of consciousness, confusion, and amnesia with a

concussion. (33) Although rates of concussion in males are greater due to larger total numbers of athletes, concussion risk seems to be greater in female athletes playing soccer or basketball. In addition, for all athletes there seems to be a higher risk of concussion with American football and rugby compared with other sports, with baseball, softball, volleyball, and gymnastics having the lowest risk. (6)

There is also evidence that children experiencing a first concussion before age 10 years have approximately twice the risk of sustaining a subsequent concussion before age 18 years compared with patients experiencing their first concussion in adolescence. It is not known whether this is due to the early concussion itself, the duration of participation in contact sports, or intrinsic factors affecting an individual's concussion risk (eg, risk-taking behavior). (34)

Ultimately, health-care providers caring for amateur athletes with a history of an early concussion, recurrent concussions, or persistent concussive symptoms may need to discuss the previously mentioned evidence as well as implement formal neurologic and neuropsychiatric assessments to aid in the discussion of concussion risk management or possible retirement from play. (6)(35)

Second Impact Syndrome

One of the greatest concerns of parents and clinicians surrounding concussion is the threat of second impact syndrome. Second impact syndrome is described as a clinical syndrome of catastrophic cerebral edema that results when a second concussion occurs before resolution of symptoms from the initial concussion. (35) The second impact may be much less severe and not even a consequence of a direct impact to the head. (35) It is thought to be due to a failure of cerebral autoregulation coupled with a stress-induced catecholamine surge, leading to cerebral edema and consequent herniation, resulting in severe disability or death. (36) Although it seems to be extremely rare, a recent review of 17 cases reported in the literature from 1946 through 2015 noted an age range of 13 to 23 years, indicating that this is a syndrome that is particularly impactful in the pediatric population. (37) Although cases seem to be most common with repeated concussions within the first 2 weeks of the initial injury, children are considered to be at risk as long as they continue to be symptomatic from their initial concussion. This diagnosis has gained a lot of attention in the medical literature owing to its devastating consequences, yet there remains some controversy around its existence. It is known that diffuse cerebral swelling can occur after a single head injury, so the occurrence of a second impact to create the clinical syndrome may not be required. (38) Nonetheless, all concussion practitioners

agree on the importance of complete resolution of symptoms before return to play because this will decrease the risk of prolonged postconcussive symptoms and the possibility of second impact syndrome. (31)

STOP HERE

MODERATE AND SEVERE TBI

In this section we focus on moderate and severe TBI. Although there are several scales that have been used to differentiate mTBI from moderate and severe TBI, the most commonly accepted classification relies on the Glasgow Coma Scale (GCS), with moderate defined as a GCS score of 9 to 13 and severe as a GCS score less than 8 (39) (Table 3). These more severe injuries are differentiated from mTBI by the fact that they have clear imaging findings; mTBIs typically do not have any MRI or CT findings. Although TBI remains one of the leading causes of mortality and morbidity in children in the United States, the management of this entity continues to be applied quite unevenly despite the existence of American Academy of Pediatrics (AAP) recommendations. (40) The epidemiology of TBI was reviewed in the Introduction.

Pathophysiology of Pediatric Head Trauma

It is important to recognize that there are major differences between the pediatric brain and the adult brain in the pathophysiology of head trauma. Although it is generally true that the pediatric brain tends to be more resilient to focal lesions (stroke, surgical excision) as a result of plasticity, the opposite seems to be true when it comes to TBI. There is strong evidence that the younger a child is when experiencing a severe TBI, the longer he or she takes to recover. (41) Furthermore, the morbidity from TBI seems to be significantly higher in children than in adults. (42) This may be related to several factors, including incomplete myelination, the higher water content of the pediatric brain, and a critical period during development when synaptic pruning depends on complex physiologic mechanisms.

Space-Occupying Traumatic Injuries

The most urgent clinical factor associated with TBI is the rapid expansion of space-occupying lesions, including bleeds and progressing edema. Interestingly, posttraumatic hydrocephalus is much less common in children than in adults and can often be managed conservatively, obviating the need for decompression or evacuation. (43)

Space-Occupying Lesions: Bleeds

Bleeds caused by TBI can occur in several locations, including potential or anatomical spaces formed by the meninges and within the substance of the brain itself.

From: Task Force 8: Classification of sports

J Am Coll Cardiol. 2005;45(8):1364-1367. doi:10.1016/j.jacc.2005.02.015

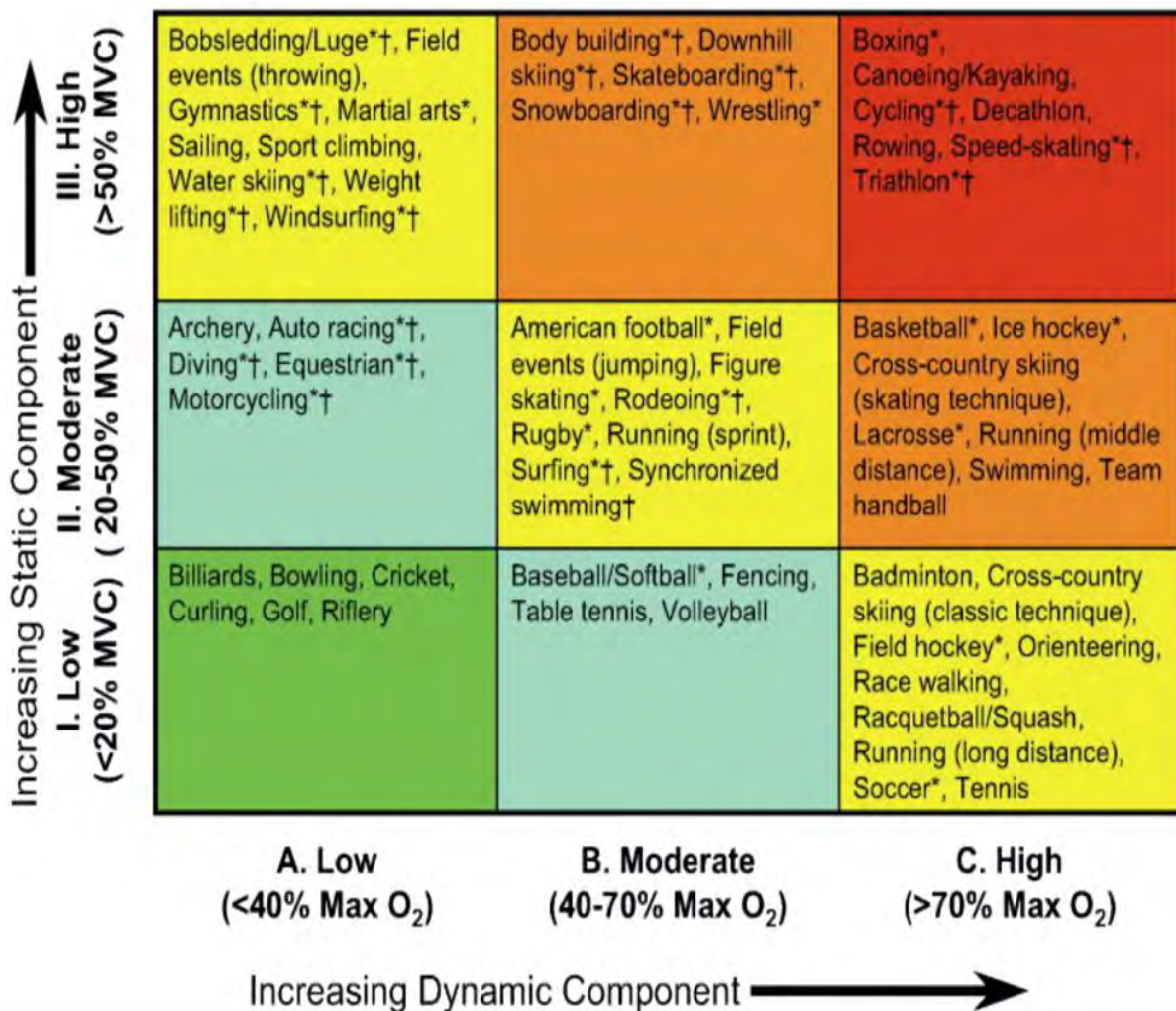


Figure Legend:

Classification of sports. This classification is based on peak static and dynamic components achieved during competition. It should be noted, however, that higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percent of maximal oxygen uptake (MaxO₂) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percent of maximal voluntary contraction (MVC) reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in green and the highest in red. Blue, yellow, and orange depict low moderate, moderate, and high moderate total cardiovascular demands. *Danger of bodily collision. †Increased risk if syncope occurs.

Table 1.

Comparison of AHA-14 Questionnaire and PPE-4 Monograph

| AHA-14 Questionnaire | PPE-4 Monograph |
|---|--|
| <p>Personal History</p> <p>1. Chest pain/discomfort/tightness/pressure related to exertion 2. Unexplained syncope/near syncope 3. Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise 4. Prior recognition of a heart murmur 5. Elevated systemic blood pressure 6. Prior restriction from sports 7. Prior testing for heart disease, ordered by a physician</p> | <p>Heart Health Questions About You</p> <p>6. Have you ever had discomfort, pain, tightness, or pressure in your chest during exercise? 5. Have you ever passed out or nearly passed out during or after exercise? 12. Do you get more tired or short of breath more quickly than your friends during exercise? 10. Do you get lightheaded or feel more short of breath than expected during exercise? 7. Does your heart ever race or skip beats (irregular beats) during exercise? 8. Has a doctor ever told you that you have any heart problems? If so, check all that apply: <input type="checkbox"/> High blood pressure <input type="checkbox"/> A heart murmur <input type="checkbox"/> High cholesterol <input type="checkbox"/> A heart infection <input type="checkbox"/> Kawasaki disease Other: _____ 1. Has a doctor ever denied or restricted your participation in sports for any reason? 9. Has a doctor ever ordered a test for your heart? (For example, ECG/EKG, echocardiogram) 11. Have you ever had an unexplained seizure?</p> |
| <p>Family History</p> <p>8. Premature death (sudden and unexpected or otherwise) before 50 yrs of age attributable to heart disease in >1 relative 9. Disability from heart disease in a close relative <50 yrs of age 10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac condition in family member</p> | <p>Heart Health Questions About Your Family</p> <p>13. Has any family member or relative died of heart problems or had an unexpected death before age 50 yrs (including drowning, unexplained car accident, or sudden infant death syndrome)? 14. Does anyone in your family have hypertrophic cardiomyopathy, Marfan syndrome, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, short QT syndrome, Brugada syndrome, or catecholaminergic polymorphic ventricular tachycardia? 15. Does anyone in your family have a heart problem, pacemaker, or implanted defibrillator? 16. Has anyone in your family had unexplained fainting, unexplained seizures, or near drowning?</p> |
| <p>Physical Examination</p> <p>11. Heart murmur</p> <p>12. Femoral pulses to exclude coarctation</p> <p>13. Physical stigmata of Marfan syndrome</p> <p>14. Brachial artery blood pressure (sitting position)</p> | <p>Physical Examination</p> <p>a. Heart</p> <ul style="list-style-type: none"> • Murmurs (auscultation standing, supine, +/- Valsalva) • Location of point of maximal impulse <p>b. Pulses</p> <ul style="list-style-type: none"> • Simultaneous femoral and radial pulses <p>c. Appearance</p> <ul style="list-style-type: none"> • Marfan stigmata (kyphoscoliosis, high-arched palate, pectus excavatum, arachnodactyly, ami span > height, hyperlaxity, myopia, MVP, aortic insufficiency) <p>d. Blood pressure</p> |

Differences between the two questionnaires are bolded.

SCAT2

Sport Concussion Assessment Tool 2



Name

Sport/team

Date/time of injury

Date/time of assessment

Age Gender M F

Years of education completed

Examiner

What is the SCAT2?¹

This tool represents a standardized method of evaluating injured athletes for concussion and can be used in athletes aged from 10 years and older. It supersedes the original SCAT published in 2005². This tool also enables the calculation of the Standardized Assessment of Concussion (SAC)³,⁴ score and the Maddocks questions⁵ for sideline concussion assessment.

Instructions for using the SCAT2

The SCAT2 is designed for the use of medical and health professionals. Preseason baseline testing with the SCAT2 can be helpful for interpreting post-injury test scores. Words in italics throughout the SCAT2 are the instructions given to the athlete by the tester.

This tool may be freely copied for distribution to individuals, teams, groups and organizations.

What is a concussion?

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms (like those listed below) and often does not involve loss of consciousness. Concussion should be suspected in the presence of **any one or more** of the following:

- Symptoms (such as headache), or
- Physical signs (such as unsteadiness), or
- Impaired brain function (e.g. confusion) or
- Abnormal behaviour.

Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle.

Symptom Evaluation

How do you feel?

You should score yourself on the following symptoms, based on how you feel now.

| | none | mild | moderate | severe | | | |
|--|------|------|----------|--------|---|---|---|
| Headache | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| "Pressure in head" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Neck Pain | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nausea or vomiting | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Dizziness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Blurred vision | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Balance problems | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sensitivity to light | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sensitivity to noise | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Feeling slowed down | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Feeling like "in a fog" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| "Don't feel right" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Difficulty concentrating | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Difficulty remembering | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Fatigue or low energy | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Confusion | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Drowsiness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Trouble falling asleep (if applicable) | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| More emotional | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Irritability | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sadness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nervous or Anxious | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

Total number of symptoms (Maximum possible 22)

Symptom severity score

(Add all scores in table, maximum possible: 22 x 6 = 132)

Do the symptoms get worse with physical activity? Y N

Do the symptoms get worse with mental activity? Y N

Overall rating

If you know the athlete well prior to the injury, how different is the athlete acting compared to his / her usual self? Please circle one response.

no different very different unsure

APPENDIX 1 THE SCAT2.

Cognitive & Physical Evaluation

1 Symptom score (from page 1)
 22 minus number of symptoms of 22

2 Physical signs score
 Was there loss of consciousness or unresponsiveness? Y N
 If yes, how long? _____ minutes
 Was there a balance problem/unsteadiness? Y N
Physical signs score (1 point for each negative response) of 2

3 Glasgow coma scale (GCS)

Best eye response (E)

| | |
|---------------------------------|---|
| No eye opening | 1 |
| Eye opening in response to pain | 2 |
| Eye opening to speech | 3 |
| Eyes opening spontaneously | 4 |

Best verbal response (V)

| | |
|-------------------------|---|
| No verbal response | 1 |
| Incomprehensible sounds | 2 |
| Inappropriate words | 3 |
| Confused | 4 |
| Oriented | 5 |

Best motor response (M)

| | |
|----------------------------|---|
| No motor response | 1 |
| Extension to pain | 2 |
| Abnormal flexion to pain | 3 |
| Flexion/Withdrawal to pain | 4 |
| Localizes to pain | 5 |
| Obeys commands | 6 |

Glasgow Coma score (E + V + M) of 15
 GCS should be recorded for all athletes in case of subsequent deterioration.

4 Sideline Assessment – Maddocks Score
"I am going to ask you a few questions, please listen carefully and give your best effort."

Modified Maddocks questions (1 point for each correct answer)

| | | |
|--|---|---|
| At what venue are we at today? | 0 | 1 |
| Which half is it now? | 0 | 1 |
| Who scored last in this match? | 0 | 1 |
| What team did you play last week/game? | 0 | 1 |
| Did your team win the last game? | 0 | 1 |

Maddocks score of 5
 Maddocks score is validated for sideline diagnosis of concussion only and is not included in SCAT 2 summary score for serial testing.

5 Cognitive assessment
Standardized Assessment of Concussion (SAC)

Orientation (1 point for each correct answer)

| | | |
|--|---|---|
| What month is it? | 0 | 1 |
| What is the date today? | 0 | 1 |
| What is the day of the week? | 0 | 1 |
| What year is it? | 0 | 1 |
| What time is it right now? (within 1 hour) | 0 | 1 |

Orientation score of 5

Immediate memory
"I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order."

Trials 2 & 3:
"I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before."

Complete all 3 trials regardless of score on trial 1 & 2. Read the words at a rate of one per second. Score 1 pt. for each correct response. Total score equals sum across all 3 trials. Do not inform the athlete that delayed recall will be tested.

| List | Trial 1 | Trial 2 | Trial 3 | Alternative word list |
|--------------|---------|---------|---------|-----------------------|
| elbow | 0 1 | 0 1 | 0 1 | candle baby finger |
| apple | 0 1 | 0 1 | 0 1 | paper monkey penny |
| carpet | 0 1 | 0 1 | 0 1 | sugar perfume blanket |
| saddle | 0 1 | 0 1 | 0 1 | sandwich sunset lemon |
| bubble | 0 1 | 0 1 | 0 1 | wagon iron insect |
| Total | | | | |

Immediate memory score of 15

Concentration
Digits Backward:
"I am going to read you a string of numbers and when I am done, you repeat them back to me backwards, in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7."

If correct, go to next string length. If incorrect, read trial 2. One point possible for each string length. Stop after incorrect on both trials. The digits should be read at the rate of one per second.

| | Alternative digit lists |
|-------------|--|
| 4-9-3 | 0 1 6-2-9 5-2-6 4-1-5 |
| 3-8-1-4 | 0 1 3-2-7-9 1-7-9-5 4-9-6-8 |
| 6-2-9-7-1 | 0 1 1-5-2-8-6 3-8-5-2-7 6-1-8-4-3 |
| 7-1-8-4-6-2 | 0 1 5-3-9-1-4-8 8-3-1-9-6-4 7-2-4-8-5-6 |

Months in Reverse Order:
"Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November ... Go ahead"

1 pt. for entire sequence correct

| | | |
|--|---|---|
| Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan | 0 | 1 |
|--|---|---|

Concentration score of 5

¹ This tool has been developed by a group of international experts at the 3rd International Consensus meeting on Concussion in Sport held in Zurich, Switzerland in November 2008. The full details of the conference outcomes and the authors of the tool are published in British Journal of Sports Medicine, 2009, volume 43, supplement 1. The outcome paper will also be simultaneously co-published in the May 2009 issues of Clinical Journal of Sports Medicine, Physical Medicine & Rehabilitation, Journal of Athletic Training, Journal of Clinical Neuroscience, Journal of Science & Medicine in Sport, Neurosurgery, Scandinavian Journal of Science & Medicine in Sport and the Journal of Clinical Sports Medicine.

² McCrory P et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. British Journal of Sports Medicine. 2005; 39: 196-204

³ McCrea M. Standardized mental status testing of acute concussion. Clinical Journal of Sports Medicine. 2001; 11: 176-181

⁴ McCrea M, Randolph C, Kelly J. Standardized Assessment of Concussion: Manual for administration, scoring and interpretation. Waukesha, Wisconsin, USA.

⁵ Maddocks, DL, Dicker, GD; Saling, MM. The assessment of orientation following concussion in athletes. Clin J Sport Med. 1995;5(1):32-3

⁶ Guskiewicz KM. Assessment of postural stability following sport-related concussion. Current Sports Medicine Reports. 2003; 2: 24-30

APPENDIX 1
 Continued.

6

Balance examination

This balance testing is based on a modified version of the Balance Error Scoring System (BESS)⁶. A stopwatch or watch with a second hand is required for this testing.

Balance testing

"I am now going to test your balance. Please take your shoes off, roll up your pant legs above ankle (if applicable), and remove any ankle taping (if applicable). This test will consist of three twenty second tests with different stances."

(a) Double leg stance:

"The first stance is standing with your feet together with your hands on your hips and with your eyes closed. You should try to maintain stability in that position for 20 seconds. I will be counting the number of times you move out of this position. I will start timing when you are set and have closed your eyes."

(b) Single leg stance:

"If you were to kick a ball, which foot would you use? [This will be the dominant foot] Now stand on your non-dominant foot. The dominant leg should be held in approximately 30 degrees of hip flexion and 45 degrees of knee flexion. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."

(c) Tandem stance:

"Now stand heel-to-toe with your non-dominant foot in back. Your weight should be evenly distributed across both feet. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."

Balance testing – types of errors

1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into > 30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position > 5 sec

Each of the 20-second trials is scored by counting the errors, or deviations from the proper stance, accumulated by the athlete. The examiner will begin counting errors only after the individual has assumed the proper start position. **The modified BESS is calculated by adding one error point for each error during the three 20-second tests. The maximum total number of errors for any single condition is 10.** If an athlete commits multiple errors simultaneously, only one error is recorded but the athlete should quickly return to the testing position, and counting should resume once subject is set. Subjects that are unable to maintain the testing procedure for a minimum of **five seconds** at the start are assigned the highest possible score, ten, for that testing condition.

Which foot was tested: Left Right
(i.e. which is the non-dominant foot)

| Condition | Total errors |
|--|--------------|
| Double Leg Stance (feet together) | of 10 |
| Single leg stance (non-dominant foot) | of 10 |
| Tandem stance (non-dominant foot at back) | of 10 |
| Balance examination score (30 minus total errors) | of 30 |

7

Coordination examination

Upper limb coordination

Finger-to-nose (FTN) task: *"I am going to test your coordination now. Please sit comfortably on the chair with your eyes open and your arm (either right or left) outstretched (shoulder flexed to 90 degrees and elbow and fingers extended). When I give a start signal, I would like you to perform five successive finger to nose repetitions using your index finger to touch the tip of the nose as quickly and as accurately as possible."*

Which arm was tested: Left Right

Scoring: 5 correct repetitions in < 4 seconds = 1

Note for testers: Athletes fail the test if they do not touch their nose, do not fully extend their elbow or do not perform five repetitions. Failure should be scored as 0.

Coordination score

of 1

8

Cognitive assessment

Standardized Assessment of Concussion (SAC)

Delayed recall

"Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order."

Circle each word correctly recalled. Total score equals number of words recalled.

| List | Alternative word list | | |
|--------|-----------------------|---------|---------|
| elbow | candle | baby | finger |
| apple | paper | monkey | penny |
| carpet | sugar | perfume | blanket |
| saddle | sandwich | sunset | lemon |
| bubble | wagon | iron | insect |

Delayed recall score

of 5

Overall score

| Test domain | Score |
|--------------------------------|---------------|
| Symptom score | of 22 |
| Physical signs score | of 2 |
| Glasgow Coma score (E + V + M) | of 15 |
| Balance examination score | of 30 |
| Coordination score | of 1 |
| Subtotal | of 70 |
| Orientation score | of 5 |
| Immediate memory score | of 5 |
| Concentration score | of 15 |
| Delayed recall score | of 5 |
| SAC subtotal | of 30 |
| SCAT2 total | of 100 |
| Maddocks Score | of 5 |

Definitive normative data for a SCAT2 "cut-off" score is not available at this time and will be developed in prospective studies. Embedded within the SCAT2 is the SAC score that can be utilized separately in concussion management. The scoring system also takes on particular clinical significance during serial assessment where it can be used to document either a decline or an improvement in neurological functioning.

Scoring data from the SCAT2 or SAC should not be used as a stand alone method to diagnose concussion, measure recovery or make decisions about an athlete's readiness to return to competition after concussion.

APPENDIX 1

Continued.



ACUTE CONCUSSION EVALUATION (ACE)

PHYSICIAN/CLINICIAN OFFICE VERSION

Gerard Gioia, PhD¹ & Micky Collins, PhD²

¹Children's National Medical Center
²University of Pittsburgh Medical Center

Patient Name: _____

DOB: _____ Age: _____

Date: _____ ID/MR# _____

A. Injury Characteristics Date/Time of Injury _____ Reporter: Patient Parent Spouse Other _____

1. Injury Description _____

- 1a. Is there evidence of a forcible blow to the head (direct or indirect)? Yes No Unknown
- 1b. Is there evidence of intracranial injury or skull fracture? Yes No Unknown
- 1c. Location of Impact: Frontal Lt Temporal Rt Temporal Lt Parietal Rt Parietal Occipital Neck Indirect Force
- 2. **Cause:** MVC Pedestrian-MVC Fall Assault Sports (*specify*) _____ Other _____
- 3. **Amnesia Before (Retrograde)** Are there any events just BEFORE the injury that you/ person has no memory of (even brief)? Yes No Duration _____
- 4. **Amnesia After (Anterograde)** Are there any events just AFTER the injury that you/ person has no memory of (even brief)? Yes No Duration _____
- 5. **Loss of Consciousness:** Did you/ person lose consciousness? Yes No Duration _____
- 6. **EARLY SIGNS:** Appears dazed or stunned Is confused about events Answers questions slowly Repeats Questions Forgetful (recent info)
- 7. **Seizures:** Were seizures observed? No Yes Detail _____

B. Symptom Check List* Since the injury, has the person experienced any of these symptoms any more than usual today or in the past day?

Indicate presence of each symptom (0=No, 1=Yes).

**Lovell & Collins, 1998 JHTR*

| PHYSICAL (10) | | COGNITIVE (4) | | SLEEP (4) | |
|---|-----|------------------------------------|-----|---|---------|
| Headache | 0 1 | Feeling mentally foggy | 0 1 | Drowsiness | 0 1 |
| Nausea | 0 1 | Feeling slowed down | 0 1 | Sleeping less than usual | 0 1 N/A |
| Vomiting | 0 1 | Difficulty concentrating | 0 1 | Sleeping more than usual | 0 1 N/A |
| Balance problems | 0 1 | Difficulty remembering | 0 1 | Trouble falling asleep | 0 1 N/A |
| Dizziness | 0 1 | COGNITIVE Total (0-4) _____ | | SLEEP Total (0-4) _____ | |
| Visual problems | 0 1 | EMOTIONAL (4) | | Exertion: Do these symptoms <u>worsen</u> with: Physical Activity <input type="checkbox"/> Yes <input type="checkbox"/> No <input type="checkbox"/> N/A Cognitive Activity <input type="checkbox"/> Yes <input type="checkbox"/> No <input type="checkbox"/> N/A Overall Rating: How <u>different</u> is the person acting compared to his/her usual self? (circle) Normal 0 1 2 3 4 5 6 Very Different | |
| Fatigue | 0 1 | Irritability | 0 1 | | |
| Sensitivity to light | 0 1 | Sadness | 0 1 | | |
| Sensitivity to noise | 0 1 | More emotional | 0 1 | | |
| Numbness/Tingling | 0 1 | Nervousness | 0 1 | | |
| PHYSICAL Total (0-10) _____ | | EMOTIONAL Total (0-4) _____ | | | |
| (Add Physical, Cognitive, Emotion, Sleep totals) | | | | | |
| Total Symptom Score (0-22) | | | | _____ | |

C. Risk Factors for Protracted Recovery (*check all that apply*)

| Concussion History? Y ___ N ___ | ✓ | Headache History? Y ___ N ___ | ✓ | Developmental History | ✓ | Psychiatric History |
|--|---|--|---|--|---|-------------------------------------|
| Previous # 1 2 3 4 5 6+ | | Prior treatment for headache | | Learning disabilities | | Anxiety |
| Longest symptom duration Days ___ Weeks ___ Months ___ Years ___ | | History of migraine headache ___ Personal ___ Family _____ | | Attention-Deficit/ Hyperactivity Disorder | | Depression |
| If multiple concussions, less force caused reinjury? Yes ___ No ___ | | | | Other developmental disorder _____ | | Other psychiatric disorder _____ |

List other comorbid medical disorders or medication usage (e.g., hypothyroid, seizures) _____

D. RED FLAGS for acute emergency management: Refer to the emergency department with sudden onset of any of the following:

- * Headaches that worsen
- * Looks very drowsy/ can't be awakened
- * Can't recognize people or places
- * Neck pain
- * Seizures
- * Repeated vomiting
- * Increasing confusion or irritability
- * Unusual behavioral change
- * Focal neurologic signs
- * Slurred speech
- * Weakness or numbness in arms/legs
- * Change in state of consciousness

E. Diagnosis (ICD): Concussion w/o LOC 850.0 Concussion w/ LOC 850.1 Concussion (Unspecified) 850.9 Other (854) _____
 No diagnosis

F. Follow-Up Action Plan Complete **ACE Care Plan** and provide copy to patient/family.

- No Follow-Up Needed
- Physician/Clinician Office Monitoring: Date of next follow-up _____
- Referral:
 - Neuropsychological Testing
 - Physician: Neurosurgery ___ Neurology ___ Sports Medicine ___ Psychiatrist ___ Psychologist ___ Other _____
 - Emergency Department

ACE Completed by: _____

A concussion (or mild traumatic brain injury (MTBI)) is a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head. Disturbance of brain function is related to neurometabolic dysfunction, rather than structural injury, and is typically associated with normal structural neuroimaging findings (i.e., CT scan, MRI). Concussion may or may not involve a loss of consciousness (LOC). Concussion results in a constellation of physical, cognitive, emotional, and sleep-related symptoms. Symptoms may last from several minutes to days, weeks, months or even longer in some cases.

ACE Instructions

The ACE is intended to provide an evidence-based clinical protocol to conduct an initial evaluation and diagnosis of patients (both children and adults) with known or suspected MTBI. The research evidence documenting the importance of these components in the evaluation of an MTBI is provided in the reference list.

A. Injury Characteristics:

1. Obtain **description of the injury** – how injury occurred, type of force, location on the head or body (if force transmitted to head). Different biomechanics of injury may result in differential symptom patterns (e.g., occipital blow may result in visual changes, balance difficulties).
2. Indicate the **cause of injury**. Greater forces associated with the trauma are likely to result in more severe presentation of symptoms.
- 3/4. **Amnesia:** Amnesia is defined as the failure to form new memories. Determine whether amnesia has occurred and attempt to determine length of time of memory dysfunction – **before** (retrograde) and **after** (anterograde) injury. Even seconds to minutes of memory loss can be predictive of outcome. Recent research has indicated that amnesia may be up to 4-10 times more predictive of symptoms and cognitive deficits following concussion than is LOC (less than 1 minute).¹
5. **Loss of consciousness (LOC)** – If occurs, determine length of LOC.
6. **Early signs.** If present, ask the individuals who know the patient (parent, spouse, friend, etc) about specific signs of the concussion that may have been observed. These signs are typically observed early after the injury.
7. Inquire whether **seizures** were observed or not.

B. Symptom Checklist:²

1. Ask patient (and/or parent, if child) to report presence of the four categories of symptoms since injury. It is important to assess all listed symptoms as different parts of the brain control different functions. One or all symptoms may be present depending upon mechanisms of injury.³ Record “1” for Yes or “0” for No for their presence or absence, respectively.
2. For all symptoms, indicate presence of symptoms as experienced within the past 24 hours. Since symptoms can be present pre-morbidly/at baseline (e.g., inattention, headaches, sleep, sadness), it is important to assess **change** from their usual presentation.
3. **Scoring:** Sum total **number** of symptoms present per area, and sum all four areas into Total Symptom Score (score range 0-22). (Note: most sleep symptoms are only applicable after a night has passed since the injury. Drowsiness may be present on the day of injury.) If symptoms are new and present, there is no lower limit symptom score. Any **score > 0** indicates **positive symptom** history.
4. **Exertion:** Inquire whether any symptoms worsen with physical (e.g., running, climbing stairs, bike riding) and/or cognitive (e.g., academic studies, multi-tasking at work, reading or other tasks requiring focused concentration) exertion. Clinicians should be aware that symptoms will typically worsen or re-emerge with exertion, indicating incomplete recovery. Over-exertion may protract recovery.
5. **Overall Rating:** Determine how different the person is acting from their usual self. Circle “0” (Normal) to “6” (Very Different).

C. Risk Factors for Protracted Recovery:

Assess the following risk factors as possible complicating factors in the recovery process.

1. **Concussion history:** Assess the number and date(s) of prior concussions, the duration of symptoms for each injury, and whether less biomechanical force resulted in re-injury. Research indicates that cognitive and symptom effects of concussion may be cumulative, especially if there is minimal duration of time between injuries and less biomechanical force results in subsequent concussion (which may indicate incomplete recovery from initial trauma).⁴⁻⁸
2. **Headache history:** Assess personal and/or family history of diagnosis/treatment for headaches. Research indicates headache (migraine in particular) can result in protracted recovery from concussion.⁸⁻¹¹
3. **Developmental history:** Assess history of learning disabilities, Attention-Deficit/Hyperactivity Disorder or other developmental disorders. Research indicates that there is the possibility of a longer period of recovery with these conditions.¹²
4. **Psychiatric history:** Assess for history of depression/mood disorder, anxiety, and/or sleep disorder.¹³⁻¹⁶

D. Red Flags:

The patient should be carefully observed over the first 24-48 hours for these serious signs. Red flags are to be assessed as **possible signs of deteriorating neurological functioning**. Any positive report should prompt strong consideration of referral for emergency medical evaluation (e.g. CT Scan to rule out intracranial bleed or other structural pathology).¹⁷

E. Diagnosis:

The following ICD diagnostic codes may be applicable.

850.0 (Concussion, with no loss of consciousness) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); no evidence of LOC (A5), skull fracture or intracranial injury (A1b).

850.1 (Concussion, with brief loss of consciousness < 1 hour) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); positive evidence of LOC (A5), skull fracture or intracranial injury (A1b).

850.9 (Concussion, unspecified) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); unclear/unknown injury details; unclear evidence of LOC (A5), no skull fracture or intracranial injury.

Other Diagnoses – If the patient presents with a positive injury description and associated symptoms, but additional evidence of intracranial injury (A 1b) such as from neuroimaging, a moderate TBI and the diagnostic category of 854 (Intracranial injury) should be considered.

F. Follow-Up Action Plan:

Develop a follow-up plan of action for symptomatic patients. The physician/clinician may decide to (1) monitor the patient in the office or (2) refer them to a specialist. Serial evaluation of the concussion is critical as symptoms may resolve, worsen, or ebb and flow depending upon many factors (e.g., cognitive/physical exertion, comorbidities). Referral to a specialist can be particularly valuable to help manage certain aspects of the patient's condition. (Physician/Clinician should also complete the ACE Care Plan included in this tool kit.)

1. **Physician/Clinician serial monitoring** – Particularly appropriate if number and severity of symptoms are steadily decreasing over time and/or fully resolve within 3-5 days. If steady reduction is not evident, referral to a specialist is warranted.
2. **Referral to a specialist** – Appropriate if symptom reduction is not evident in 3-5 days, or sooner if symptom profile is concerning in type/severity.
 - **Neuropsychological Testing** can provide valuable information to help assess a patient's brain function and impairment and assist with treatment planning, such as return to play decisions.
 - **Physician Evaluation** is particularly relevant for medical evaluation and management of concussion. It is also critical for evaluating and managing focal neurologic, sensory, vestibular, and motor concerns. It may be useful for medication management (e.g., headaches, sleep disturbance, depression) if post-concussive problems persist.

Sports Physical II Quiz

1. What are the “Red Flags” of cardiovascular history/physical that should prompt further evaluation prior to clearance? *Go around the table and list one “Red Flag”:*

2. The incidence of sudden death ranges from 1-2 per 100,00 athlete-years, with__ percent due to cardiovascular disease. Complete the following table:

| Condition | Mechanism of sudden death |
|-----------|---------------------------|
| | |
| | |
| | |
| | |
| | |

3. Based on Bethesda Conference Sports Classifications, what sports are young athletes cleared for, who have the following heart conditions?
 - a. Marfan syndrome with normal aortic root diameter; currently cleared by cardiology for **class** ___ **and** ___ competitive sports play.

 - b. Recent dx of SVT with episodes causing breathlessness and dizziness, recently started on medication, currently cleared by cardiology for **class** ___ sports.

 - c. Moderate Mitral Regurgitation with mild LVH, currently cleared by cardiology for **class** _____ competitive sports play.

4. **CONCUSSION True or False:**
 - A. Concussions result only from a direct blow to the head, face, or neck.

 - B. Concussions result in structural injury to the brain.

 - C. Loss of consciousness is a critical historical clue that determines concussion management.

 - D. Concussion results in an impairment in neurologic function that usually resolves spontaneously.

 - E. The developing brain is more vulnerable to reinjury & may take longer to heal from TBI-

5. What are the “Red Flags” of concussion history/physical that suggest prolonged recovery or caution for return-to-play? What clinical features would *also* indicate neuro-imaging?

| Red Flags for Prolonged Recovery | Indications for Neuroimaging |
|---|-------------------------------------|
| | |
| | |
| | |
| | |
| | |
| | |
| | |
| | |
| | |

Sports Physical II Cases

Case 1:

Michael is a 17 year-old male who comes to see you with his mom. He is on the varsity football team and the regional championship series is about to start this weekend. The first game is against his school's biggest rival, the Honey Badgers. He forgot to come in before the season started, but because he is one of his team's star players, his coach let him slide. Now the school administration has become aware and he cannot play in this weekend's big game until you sign his forms. He thinks a scout will be there. "Help me, Doc!"

What do you need to know in order to complete his sports clearance paperwork?

His past medical history is benign, no major illness, no hospitalizations. He takes no medications or supplements except a protein powder after practice. Family history unremarkable: no cardiac history or sudden death. When you ask specifically about injuries, Mom does report that he broke his ankle as a freshman and had it pinned by orthopedics, but "they said he was ok to play now." He denies any pain or instability when playing football. You ask him if he has ever had a concussion. He says, "No way, doc. I'm pretty tough. I've never passed out from a hit."

Do you want to know anything else about his injury history?

On further questioning, with mom's prompting, Michael admits that he has had a few hits where he felt dazed for a few minutes afterwards, but he says he felt fine after a few minutes and was able to still play, so he never told his coach. The most recent of these was less than 1 month ago. He denies any memory loss before or after any of his hits. He does say that he occasionally gets headaches "just like everybody" but they are "no big deal" and respond to Motrin.

What are you concerned about? Will you clear Michael to participate in his big game against the Honey Badgers this weekend? "Com'on Doc!"

You discuss some of these concerns with Michael and his mom, and she is surprised that none of his football coaches have ever called his "head dings" concussions and educated them about potential consequences. "What should we do if he gets another concussion? Are there any precautions we can take?"

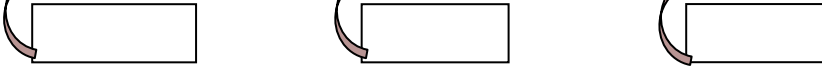
What sort of anticipatory guidance will you give Michael and his mother?

Bonus: What is the law which requires concussion education for coaches, athletes, and parents? Has it been enacted in Maryland, D.C., and Virginia?

Case 2:

Jay is a 15 year-old male who presents to clinic on “Sports Physical Day”. You have churned through 4 physicals so far. The corpsmen bring you his vitals sheet and you note the following:

HR 90 BP 145/95 Weight 52 kg Height 184cm



What history is most important to obtain when Jay is brought back to your room?

Jay tells you that he is center for his school’s basketball team, but also wants to start weight-lifting to “bulk up”. He denies exertional chest pain or dyspnea, syncope, or history of heart murmur. PMHx is positive for history of “shoulder dislocation” after a collision with another player during a game. Jay’s athletic trainer relocated his shoulder, and he has had no other joint issues. His HEADSS exam is unremarkable, and he denies use of alcohol, tobacco, or other recreational drugs, including supplements. His father reports history of HTN in multiple family members, but no other cardiac disease or premature deaths.

What will you focus on during your physical exam?

On your exam, you note that he has a thin body habitus. HR is regular. There are no murmurs or extra heart sounds, and femoral pulses are 2+ bilaterally. Lungs are clear. There is no organomegaly. 2-min orthopedic exam is normal, and there is no kyphoscoliosis, pectus deformity, joint hypermobility, or arachnodactyly. He is Tanner 5 and has no hernias.

What is your assessment of Jay?

| Age (Year) | BP Percentile ↓ | Systolic BP (mmHg) | | | | | | | Diastolic BP (mmHg) | | | | | | |
|------------|-----------------|--------------------------|------|------|------|------|------|------|---------------------|------|------|------|------|------|------|
| | | ← Percentile of Height → | | | | | | | | | | | | | |
| | | 5th | 10th | 25th | 50th | 75th | 90th | 95th | 5th | 10th | 25th | 50th | 75th | 90th | 95th |
| 15 | 50th | 109 | 110 | 112 | 113 | 115 | 117 | 117 | 61 | 62 | 63 | 64 | 65 | 66 | 66 |
| | 90th | 122 | 124 | 125 | 127 | 129 | 130 | 131 | 76 | 77 | 78 | 79 | 80 | 80 | 81 |
| | 95th | 126 | 127 | 129 | 131 | 133 | 134 | 135 | 81 | 81 | 82 | 83 | 84 | 85 | 85 |
| | 99th | 134 | 135 | 136 | 138 | 140 | 142 | 142 | 88 | 89 | 90 | 91 | 92 | 93 | 93 |

What will you write on his Pre-participation Evaluation Form (*Cleared, Cleared with further evaluation, Not cleared*)? Can Jay still participate in basketball & weight-lifting?

What are the absolute contraindications to sports participation? Is HTN included?

Before Jay leaves with his signed forms, his father asked whether you will do a “screening EKG” for Jay, as he has read in the news that this can prevent sudden death. *Imagine that Jay’s exam was completely normal*, how will you respond?

Sports Physical II Board Review

1. A 14-year-old boy loses consciousness while playing basketball. He regains consciousness in 30 seconds and is transported to a pediatric emergency department. Results of head computed tomography scan, electroencephalography, and echocardiography are within normal limits. Electrocardiography results are interpreted as abnormal, with a heart rate of 90 beats/min, PR interval of 150 msec, and QTc interval of 550.

Of the following, the MOST likely explanation for this patient's syncopal episode is

- A. complete atrioventricular block
- B. first-degree atrioventricular block
- C. hypertrophic cardiomyopathy
- D. long QT syndrome
- E. supraventricular tachycardia due to Wolff-Parkinson-White syndrome

2. A 16-year-old girl who is new to your practice comes to the clinic for a physical examination prior to enrollment in a summer volleyball camp. She is generally healthy, and she does well academically. On physical examination, you note that she is unusually tall and slender, and she appears to have long fingers and toes. You are concerned that she could have Marfan syndrome, and you refer her for a clinical genetics evaluation.

Of the following, the additional finding that would MOST strongly suggest the diagnosis of Marfan syndrome for this girl is

- A. high myopia
- B. long, narrow face
- C. mitral valve prolapsed
- D. narrow palatal contour
- E. spontaneous pneumothorax

3. An 18-year-old girl presents with a history of occasional mild chest pain of 1 week's duration. The episodes occur at rest and have not affected her performance as a competitive long-distance swimmer. On physical examination, her heart rate is 48 beats/min and blood pressure is 105/65 mm Hg. Electrocardiography demonstrates left ventricular hypertrophy, which is confirmed by echocardiography.

Of the following, the MOST likely cause of these findings is

- A. aortic stenosis
- B. athlete's heart
- C. cardiac conduction disturbance
- D. coronary artery anomaly
- E. hypertrophic cardiomyopathy

4. A family has just relocated to your community, and you are evaluating their 12-year-old son for the first time this afternoon. Family history reveals that the boy's father and grandmother had premature cardiovascular disease. The boy's parents are concerned about risk of heart disease.

Of the following, the MOST important next step in this child's evaluation is

- A. echocardiography
- B. electrocardiography
- C. fasting lipoprotein analysis
- D. random cholesterol measurement
- E. referral to the cardiology clinic