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

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 Mongraph
  - 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

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ARRHYTHMIAS (J. BUNCH, SECTION EDITOR)

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

Alexander G. Hajduczok[1] · Max Ruge[2] · Michael S. Emery[3]

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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

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

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

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✉ 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

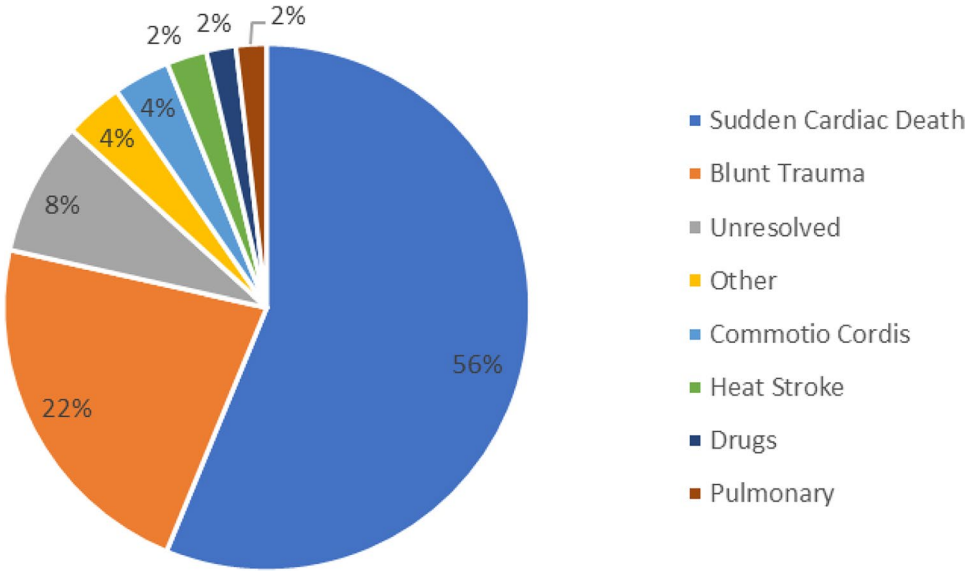

Fig. 1 General etiologies of sudden death in competitive athletes ≤ 39 years old (adapted from Maron et al., 2009) [2]

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* arrhythmogenic 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	- 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	- 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	- 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 primary 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

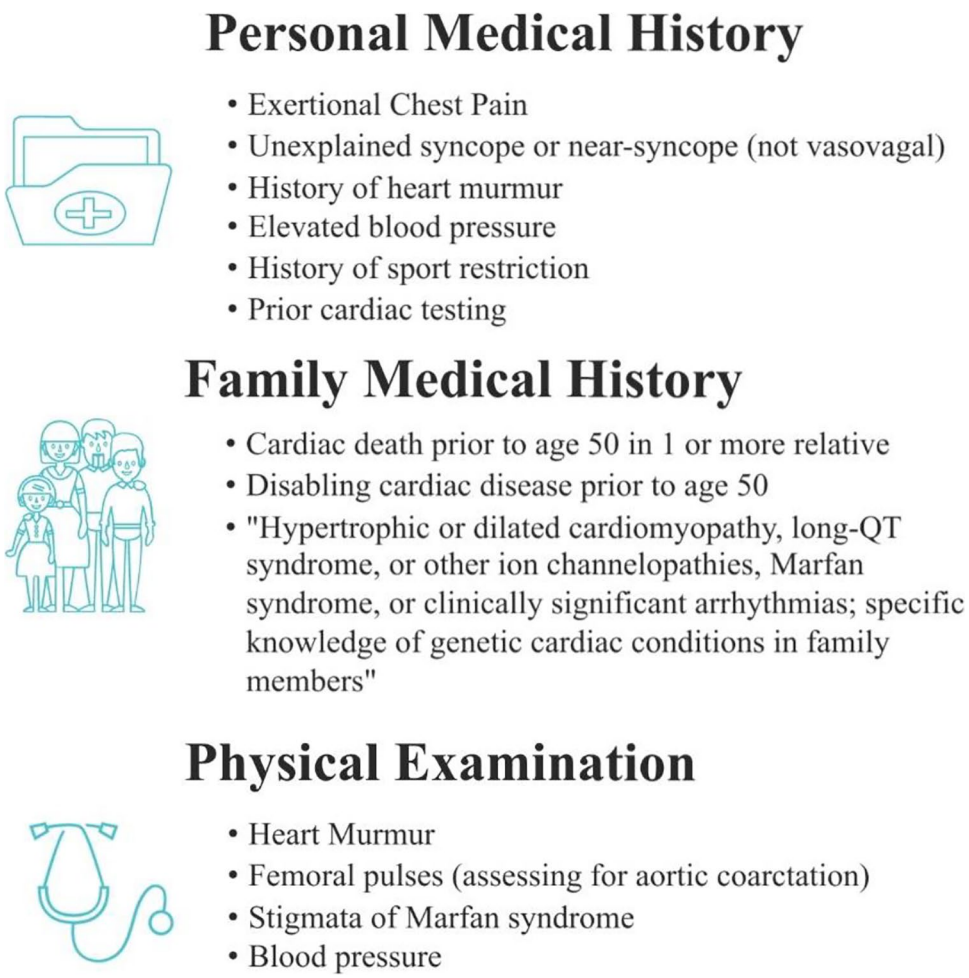

Fig. 2 Components of the AHA-recommended, 14-point pre-participation screening

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

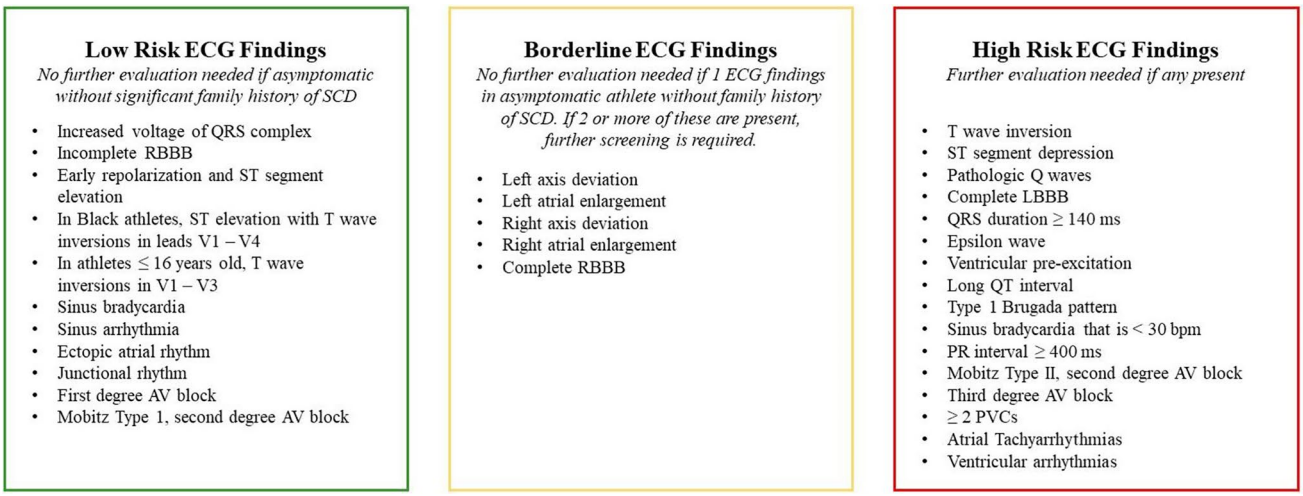

Fig. 3 The International Criteria for ECG interpretation in athletes detailing low, borderline, and high-risk ECG findings (adapted from Drezner et al., 2017) [61]. Abbreviations: ECG = electrocardiogram; SCD = sudden cardiac death; RBBB = right bundle branch block; AV = atrioventricular; PVC = premature 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.

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• Of importance
•• Of major importance

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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.

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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

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

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.

JACC Journals

From: Task Force 8: Classification of sports

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

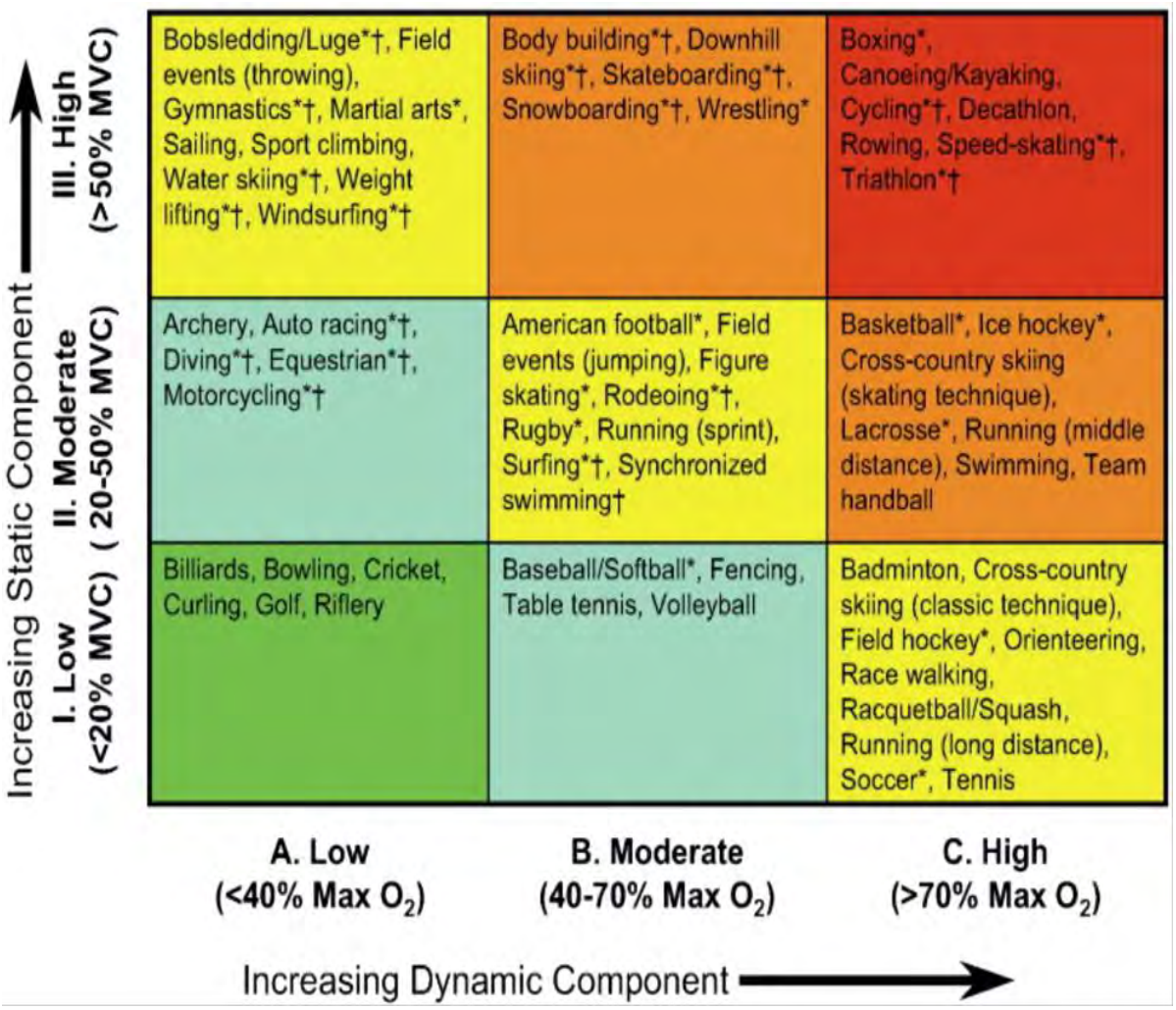

Figure Legend:

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 (MaxO_2) 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.

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 2005[2]. This tool also enables the calculation of the Standardized Assessment of Concussion (SAC)[3,4] score and the Maddocks questions[5] 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 distribtion 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.

[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

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] 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 a 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.

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":*

- Syncope or near-syncope on exertion
- Chest Pain/Discomfort on exertion
- Palpitations at rest
- Excessive SOB or fatigue with activities
- FHx of Marfan, cardiomyopathy, Long QT, or clinically significant arrhythmias
- FHx of premature, sudden death
- Irregular heart rhythm
- Weak or delayed femoral pulses
- Fixed, split S2
- Systolic murmur louder than 3/6
- Any diastolic murmur
- Stigmata of Marfan's
- Chest pain in Turners syndrome

2. The incidence of sudden death ranges from 1-2 per 100,000 athlete-years, with **75** percent due to cardiovascular disease. Complete the following table:

Condition	Mechanism of sudden death
Hypertrophic cardiomyopathy	Myofibrillary disarray → re-entrant ventricular arrythmias
Congenital CA anomalies	? Ischemia when myocardial demands ↑ w/exercise ?
Marfan Syndrome	Aortic root dilation → Dissection and rupture of the aorta
Long QT Syndrome	LQTS → Ventricular fibrillation
Commotio Cordis	Enhanced energy transfer to heart → arrythmias

3. Based on Bethesda Conference Sports Classifications, what sports are young athletes cleared for, who have the following heart conditions? (See Table 3 and Bethesda Classification and have residents list specific sports in each category)
    a. Marfan syndrome with normal aortic root diameter; currently cleared by cardiology for **class IA and IIA** competitive sports play.
    b. Recent dx of SVT with episodes causing breathlessness and dizziness, recently started on medication, currently cleared by cardiology for **class IA** sports.
    c. Moderate Mitral Regurgitation with mild LVH, currently cleared by cardiology for **IA-IB-IC, IIA-IIB-IIC** competitive sports play.

4. **CONCUSSION True or False:**
    A. Concussions result only from a direct blow to the head, face, or neck. False *(also from acceleration/deceleration or rotational injury)*
    B. Concussions result in structural injury to the brain. False *(usually no structural injury and standard neuroimaging is negative)*
    C. Loss of consciousness is a critical historical clue that determines concussion management. False *(LOC seen in <10% concussions, management is symptom based)*
    D. Concussion results in an impairment in neurologic function that usually resolves spontaneously. True
    E. The developing brain is more vulnerable to reinjury & may take longer to heal from TBI. True *(Second-impact syndrome has never been seen in >20yo athletes; high school students average 10-14 days to recover from concussion, vs 5-7 days for college athletes, 3-5 days for adults, based on computerized neuropsych testing).*

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
≥ 3 sxs at presentation	LOC or amnesia + HA, vomiting, short term memory deficits, seizures, GCS < 15, coagulopathy, focal neuro deficits
Specific symptoms (fatigue, fogginess)	
Amnesia	No LOC or amnesia, but focal neuro deficits, vomiting, severe headache, signs of basilar skull fracture, GCS <15, coagulopathy, significant mech of injury
HA > 60hrs	
LOC > 60sec	
h/o prior concussion	LOC > 60sec (AAP)
Age < 18 years	Focal neurologic findings/deficits (AAP)
Comorbid conditions; med use	Evidence of a skull fracture (AAP)
High-risk sport (contact, collision)	

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?
See Sports Physical I Case . . . It will be more important to pay extra special attention to prior injuries in this patient as he plays a high contact sport, especially joint and head injuries.

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?
Many adolescents/athletes do not consider an injury where they do not lose consciousness to be a concussion. You may need to do directed and specific questioning about any type of hit to the head, feeling "dazed" or "foggy," memory loss, headaches following a hit to the head, difficulty playing or practicing following a hit to the head, and any type of injury that resulted in LOC.

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!"
There are two potential concerns: Second-Impact Syndrome or Post-concussion Syndrome.

1. Second-Impact Syndrome: Athlete who has sustained an initial head injury sustains a second before the symptoms associated with the first have fully cleared. Results in cerebral vascular congestion, which can progress to diffuse cerebral swelling and death.

2. Post-concussion Syndrome: Presence of cognitive, physical, or emotional symptoms of a concussion lasting longer than expected, with a threshold of 1 to 6 weeks of persistent symptoms after a concussion to make the diagnosis *(from Extra-Credit CPG).*

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?

- No evidence that protective gear prevents concussions, but helmets and mouth guards reduce risk of skull and dental fractures, so should continue to be worn.
- Encourage concussion education for Michael & coaches through CDC's "Heads Up".
  - Symptoms of concussion: *See Table 2—physical, cognitive, emotional, sleep*
  - Initial evaluation: *See Figure 1 & Table 3—e.g. sideline evaluation w/SCAT2*
  - Management: *See Figure 1 & Table 6—rest, then graded return-to-play*

TABLE 5 Concussion Rehabilitation/Stepwise Return to Play

Rehabilitation Stage	Functional Exercise
1. No activity	Complete physical and cognitive rest
2. Light aerobic activity	Walking, swimming, stationary cycling at 70% maximum heart rate; no resistance exercises
3. Sport-specific exercise	Specific sport-related drills but no head impact
4. Noncontact training drills	More complex drills, may start light resistance training
5. Full-contact practice	After medical clearance, participate in normal training
6. Return to play	Normal game play

Each stage in concussion rehabilitation should last no less than 24 hours with a minimum of 5 days required to consider a full return to competition. If symptoms recur during the rehabilitation program, the athlete should stop immediately. Once asymptomatic after at least another 24 hours, the athlete should resume at the previous asymptomatic level and try to progress again. Athletes should contact their health care provider if symptoms recur. Any athlete with multiple concussions or prolonged symptoms may require a longer concussion-rehabilitation program, which is ideally created by a physician who is experienced in concussion management.

- Those with multiple concussions, like Michael, have increased risk of recurrent injury and have longer recovery periods. However, there are no guidelines regarding athletic disqualification or retirement.

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

The Zackery Lystedt Law has been enacted in all 3 states (in 34 states total). The law also mandates removal of athletes from activity if there is any suspicion of concussion, and return to play must be cleared by a licensed health-care professional.

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 (>95th %ile) Weight 52 kg (25th %ile) Height 184cm (95th %ile)

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

See Sports Physical I Case . . . Cardiac history is particularly important in this hypertensive patient. See 12-Element AHA Recommendations for Preparticipation Screening (*Module 1):*

***Personal History*:**

1. Exertional chest pain/discomfort,
2. Unexplained syncope/near-syncope,
3. Excessive exertional dyspnea
4. Prior recognition of a heart murmur
5. **Elevated systemic blood pressure**.

Family History:

6. Premature death (<50) d/t heart disease
7. Disability from heart disease in <50y rel.
8. Cardiac conditions in family: HCM, DCM, LQTS, Marfan, arrhythmias.

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?

Include standard health supervision evaluation, with focused orthopedic exam (e.g. 2min Orthopedic Exam). Cardiovascular exam should include 12-Element AHA recommendations:

Physical Examination:

9. Heart mumur (auscultate supine and standing, to pick up murmurs of dynamic LVOT obstruction)
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan (esp. considering Jay's height and history of joint dislocation)
12. Brachial artery blood pressure (sitting position)

Because of HTN noted on VS, would also obtain 4-extremity blood pressures.

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?

- Based on single reading, Jay would meet criteria for Stage I Hypertension (>95th%ile but < 99th%ile + 5mmHg for age, gender, height). ***Confirm with appropriately-sized cuff and 2 additional BP measurements at separate visits*** (see Hypertension Module)

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							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

- Does not appear to meet criteria for Marfan's Syndrome (see Ghent Nosology).

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?
Answers may vary; points to discuss include the following:

- According to "Medical Conditions & Sports Participation" (*see Module I*), patients with sustained HTN (>95th%ile) need evaluation, but should continue to participate in sports.
- Patients with severe HTN (>5mmHg + 99th%ile) should avoid high-static sports (*see Table 3—includes weight-lifting).*
- Consider EKG, if/when HTN is confirmed over 3 separate readings (e.g. LVH results from chronic HTN; one cause of HTN is aortic coarctation).
- *Differential diagnosis of HTN is beyond the scope of this module, but may need lab work-up (CBC, RFP, U/A, TFTs, lipids) and referral to Renal and/or Cardiology.*

What are the absolute contraindications to sports participation? Is HTN included?
See Table 2: pulmonary vascular disease with cyanosis; severe Pm HTN; severe AS, AR, MS, MR; cardiomyopathies; vascular EDS; coronary anomalies; acute pericarditis, myocarditis, Kawasaki disease; CPVT. (These Bethesda eligibility criteria do NOT apply to non-competitive, recreational activities. The AHA has published separate guidelines).

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?
Answers may vary; points to discuss include the following:

- The cost-to-benefit ratio of obtaining a screening EKG or echo continues to be debated in the US. The European Society of Cardiology, largely based on the Italian experience which showed an 89% relative risk reduction in sudden cardiac death, recommends mandatory EKG screening of all competitive athletes. The AHA does not. One analysis by the JACC found that EKG screenings of all young competitive athletes in the US would cost $69 billion over 20 years and save about 4,183 lives, making the cost per life saved over $10 million.
- In arguing against mandatory EKG screening, the AHA often notes the "human costs" of false positives, which can result in additional potentially unnecessary tests and removal from play of athletes who are not actually at risk.
- Screening considerations differ between Italy and the US, as 25% of Italian cases of sports related SCD are due to the same heritable arrhythmia (ARVD), and the total athlete population much smaller. This impacts the pre-test probability of an EKG screen and the feasibility of universal screening.

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

The young child or adolescent who experiences an episode of syncope must undergo 12-lead electrocardiography (ECG) as a part of his or her evaluation. Findings on ECG may indicate the possibility of a rhythm disturbance or conduction disorder. However, the corrected QT interval must be measured to assess for the possible diagnosis of long QT syndrome. Findings on ECG almost always are abnormal in the patient who has symptomatic long QT syndrome. In addition to a prolonged corrected QT interval, there may be bizarre or notched T waves and prominent U waves. Exercise testing may elicit abnormalities not seen on resting ECG.

Patients who have long QT syndrome are at risk for life-threatening ventricular tachycardia, torsades de pointes, and ventricular fibrillation. The syndrome may have an autosomal dominant or autosomal recessive inheritance pattern or may be a new mutation. Many of the mutations causing long QT syndrome demonstrate ion channel abnormalities. Clinical laboratory testing is available. Pharmacologic therapy and implantation of automatic cardiovertor-defibrillators are the currently employed treatment modalities. Affected patients may present with cardiac arrest, syncope, seizures, or palpitations. Any patient who presents with suspicious symptoms in whom ECG identifies a corrected QT interval greater than 450 msec warrants specialty evaluation.

Patients who have complete atrioventricular block also may present with syncope. However, the teenager who has complete atrioventricular block would have a resting heart rate dramatically lower than 70 beats/min (typically in the range of 40 to 60 beats/min), and the ECG would demonstrate a profound conduction disturbance characterized by a lack of relationship between the atrial and ventricular rates. It is atypical for first-degree atrioventricular block to result in syncope. In addition, first-degree block is identified easily on baseline ECG by the PR interval exceeding approximately 180 msec.

Hypertrophic cardiomyopathy can present with syncope due to either obstruction of left ventricular outflow and resultant hypotension or ventricular arrhythmias caused by the disturbance to repolarization. However, the corrected QT interval is not markedly prolonged. In addition, the findings of hypertrophic cardiomyopathy are readily discerned by echocardiography. Supraventricular tachycardia due to Wolff-Parkinson-White syndrome can result in syncope because patients are at risk for degeneration of their arrhythmia to atrial and subsequently ventricular fibrillation. However, baseline ECG should demonstrate the classic features of a short PR interval and a delta wave.

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

Marfan syndrome (MS) is an autosomal dominant connective tissue disorder that has a prevalence of 1 in 10,000 and usually is caused by alterations in the fibrillin 1 (FBN1) gene. MS primarily involves the skeletal, cardiovascular, and ocular systems. Skeletal features include pectus carinatum, pectus excavatum, reduced upper-to-lower segment ratio, scoliosis of greater than 20 degrees or spondylotlisthesis, reduced elbow extension, joint hypermobility, and others. Major cardiovascular features are dilation or dissection of the ascending aorta, and minor features include mitral valve prolapse, dilation of the main pulmonary artery, and calcification of the mitral annulus. Ocular features include ectopia lentis, flat cornea, hypoplastic iris, and increased axial length of the globe. Although high myopia; long, narrow face; mitral valve prolapse; and narrow palatal contour all are associated with MS, they are also relatively common findings in other syndromes and in the general population. Spontaneous pneumothorax occurring in a teenager, however, is unusual and is one of the minor criteria for the diagnosis of MS. Therefore, the history of spontaneous pneumothorax associated with this girl's physical features should increase the pediatrician's suspicion for MS.

Due to the risks for aortic root dilatation and dissection associated with MS, affected individuals are asked not to participate in contact or competitive sports or isometric exercise. In addition, they should avoid activities placing them at increased risk for joint injury or pain.

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

Chest pain is a common complaint in the general pediatric practice, and no significant cardiac abnormality is found in most cases. Musculoskeletal pain is the most common cause of chest pain in the child and adolescent and is the most likely source of the mild pain reported for the girl in the vignette.

Rarely, however, chest pain is a sign of cardiac pathology such as pericarditis, myocarditis, or myocardial infarction. The lack of pain during exercise (when the individual would be expected to be in a high myocardial demand state) for this girl is reassuring, suggesting that the pain is of a benign, noncardiac origin. In addition, the lack of associated symptoms such as radiation of the pain to the face, arms, or back; associated dizziness or syncope; and PE findings suggestive of CVD are all reassuring.

The low blood pressure and heart rate reported for this girl are not of particular concern and most likely reflect athlete's heart. The well-trained athlete exhibits findings on physical examination and laboratory assessment that differ from the general population. Specifically, the resting heart rate typically is 20% to 30% lower than the average for age in the population, and blood pressure is at the lower range of normal. Often, a physiologic flow murmur can be detected, which is caused by a high cardiac output state. Findings on electrocardiography can include left ventricular hypertrophy. Echocardiographic findings can include left ventricular wall thickening/hypertrophy associated with normal or hyperdynamic left ventricular contractility. Such physiologic left ventricular hypertrophy can be distinguished from pathologic hypertrophic cardiomyopathy based upon features of diastolic function, mitral valve disease, and assessment of left ventricular outflow tract obstruction. The patient who has hypertrophic cardiomyopathy exhibits mitral regurgitation, obstruction to blood flow due to the narrowed left ventricular outflow tract, and diastolic dysfunction.

The individual who has athlete's heart manifests none of these pathologic echocardiographic features. Novel imaging methods (including cardiac magnetic resonance imaging) can differentiate hypertrophic cardiomyopathy from left ventricular hypertrophy associated with athlete's heart. In addition, when the well-trained individual who has athlete's heart is deconditioned for several months, echocardiography demonstrates resolution of the hypertrophy.

Aortic valve stenosis of such severity to cause chest pain always is associated with a pronounced systolic ejection-type murmur that is heard most commonly at the left mid-sternal border, with radiation to the right infra-clavicular region. Cardiac conduction abnormalities can manifest as chest pain in the young child who has difficulty distinguishing pain from other unusual chest symptoms such as palpitations. However, the teenager is unlikely to describe palpitations or other rhythm disturbances as evoking chest pain. Coronary artery anomalies are rare and include acquired conditions such as Kawasaki disease and congenital lesions such as anomalous left coronary arising from the pulmonary artery (ALCAPA) and coronary fistulae. Coronary anomalies do not cause left ventricular hypertrophy and should be detected by detailed echocardiography. *As noted previously, hypertrophic cardiomyopathy can be distinguished from the left ventricular hypertrophy associated with athlete's heart by the presence of a pathologic murmur caused by either mitral regurgitation or left ventricular outflow tract obstruction, findings on echocardiography, and chest pain that is more likely to occur during exercise and have associated dizziness, syncope, and radiation of the pain.*

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

In recent years, an increasing body of literature has indicated that atherosclerotic disease and its effect on the cardiovascular system are progressive processes that begin during early childhood. Research has demonstrated that the complex process of acquired cardiovascular disease is the result of genetic predisposition, along with factors such as diet, physical activity, and other comorbidities.

In adults, the strongest risk factors for the development of cardiovascular disease include a high concentration of low-density lipoprotein, a low concentration of high-density lipoprotein, elevated blood pressure, type 1 or 2 diabetes mellitus, cigarette smoking, and obesity. Research in children and adolescents has shown that some of these risk factors may be present in early childhood. It is imperative, therefore, for pediatricians to take proactive roles in stressing the importance of healthy cardiovascular lifestyles and identifying children at risk for cardiovascular disease.

The importance of the history, especially the family history, cannot be overemphasized because the clinical manifestations of hypercholesterolemia are variable and may not be physically present until later in childhood, adolescence, or even adulthood. Some children who have homozygous familial hypercholesterolemia may demonstrate cutaneous or tendinous xanthomas, but often these findings are not apparent until early adulthood. As a result, some children who have significant hyper-cholesterolemia may have normal findings on physical examination.

The American Academy of Pediatrics has adopted the recommendation that all children undergo cholesterol screening between 9 and 11 years old. Accordingly, the boy in the vignette should undergo a screening test for lipoproteins that includes cholesterol, high-density lipoproteins, and low-density lipoproteins in the fasting state.

Random cholesterol screening may provide important information, but taken in isolation, will not offer as much information as a fasting lipoprotein panel. Referral to a specialized clinic such as cardiology or endocrinology may be indicated, but this should be considered only after more complete information is obtained from the diagnostic evaluation. Neither echocardiography nor electrocardiography is indicated for this patient, and neither is used as a screening test for cardiovascular risk factors in children.