Medical Evaluation of Athletes: Medical History and Physical Examination

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Learning Objectives

1. To recognize "red flags" in an athlete's personal, systemic and family history.
2. To know the cornerstones of an accurate physical examination of athletes.
3. To understand and be able to classify the implications of specific clinical findings.
4. To recognize the limitations of the assessment of medical history and clinical examination in athletic populations.

6.1 Personal History/Symptoms

The clinical importance of personal, systemic and family medical history in athletic populations is apparent, but its accuracy very much depends on the particular circumstances. The yield of a questionnaire during pre-participation screening is sparse, with about two-thirds of the athletes indicating symptoms but with finally less than two percent leading to a cardiac diagnosis [1] (Fig. 6.1). This highlights the pivotal role of an experienced physician in classifying and clearing these subjective symptoms in order to prevent unnecessary anxiety and further examinations. On the other hand, athletes referred from their general practitioner because of particular cardiac symptoms have a high yield of 44% for having a cardiac diagnosis [2].

- Independent of the setting, the athlete's symptoms should always be evaluated in the context of personal and family history, as well as physical examination and the 12-lead ECG.

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Physical Activity Readiness Questionnaire (PAR-Q)		
Question		Answer
1.	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?	☐ YES ☐ NO
2.	Do you feel pain in your chest when you do physical activity?	☐ YES ☐ NO
3.	In the past month, have you had chest pain when you were not doing physical activity?	☐ YES ☐ NO
4.	Do you lose your balance because of dizziness or do you ever lose consciousness?	☐ YES ☐ NO
5.	Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?	☐ YES ☐ NO
6.	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?	☐ YES ☐ NO
7.	Do you know of any other reason why you should not do physical activity?	☐ YES ☐ NO
YES to one or more questions: medical examination reasonable before becoming much more physically active		

Fig. 6.1 The Physical Activity Readiness Questionnaire (PAR-Q) as a classic example of a questionnaire designed to evaluate symptoms or conditions indicative of an increased risk during sports. This and similar, modified questionnaires are frequently recommended by various scientific or commercial sporting associations as a pre-participation screening tool (e.g. German Society of Sports Medicine, www.dgsp.de)

6.2 Syncope/Near-Syncope

The most alarming symptom among athletes is syncope. This is defined as

- "a sudden transient loss of consciousness and postural tone due to transient global cerebral hypoperfusion, characterized by rapid onset, short duration and spontaneous complete recovery" [3].

A syncope is not uncommon among athletes, as shown recently by Colivicchi and coworkers [4] on n = 7568 athletes undergoing pre-participation screening. A syncopal episode was reported in 6.2% within the preceding 5 years, but only 13.3% of these episodes were related to exercise (i.e. post-exertional in 12.0%, and during exercise in only 1.3%). At follow-up, those athletes with an exercise-unrelated syncope had a diagnosis of either vasovagal or situational syncope. Unlike post-exertional syncope, 50% of syncopal episodes occurring during exertion were cardiogenic, either due to structural heart disease or primary arrhythmia, and these causes are also associated with an increased risk of sudden cardiac death (SCD) [5].

- Exercise-related syncope is a red flag symptom that always warrants thorough and rapid evaluation.

Evaluation of an exercise-induced syncope should include obtaining a thorough history of circumstances related to its occurrence, including objective or subjective warning signs prior to the event (e.g. sweating, nausea, palpitations, chest pain), single or multiple appearance, duration, and state of mind at recovery. It should also

be assessed whether the syncope had occurred during long-distance exercise with possible electrolyte derangement, during specific activities (e.g. swimming or sudden and loud noises as typically being associated with the long QT syndrome), during fever or after exercise in high temperature as observed in the Brugada syndrome, or after a blunt chest trauma as seen in commotio cordis. A syncope leading to an injury suggests cardiac, arrhythmogenic etiology.

Further issues that need to be evaluated within the diagnostic pathway are:

1. Use of medication and stimulants.
2. Familiar predisposition to early sudden unexplained death or cardiac disease.
3. Clinical examination with focus on cardiac murmurs (see below).
4. 12-lead ECG and, at least, 24 h of continuous Holter monitoring.
5. Basic clinical chemistry: hemoglobin, electrolytes, D-dimer and cardiac biomarkers.
6. Echocardiography including the detection of coronary ostia.
7. Exercise test if still uncertain or if symptoms have clearly been exercise-related, important to encourage to maximum exercise, preferably applying cardiopulmonary exercise testing (CPET)
8. Coronary computed (CT) angiography if coronary anomaly or coronary artery disease is suspected.
9. Cardiac magnetic resonance imaging (MRI) if possible and if structural disease is suspected, especially e.g. right heart disease as Arrhythmogenic Cardiomyopathy or apical Hypertrophic Cardiomyopathy (HCM; see Chaps. 14 and 15).
10. If still unclear: Loop recorder implantation.
11. Electrophysiologic examination for risk assessment (debatable).

6.3 Exertional Chest Pain/Discomfort

During lifetime, thoracic pain is experienced in 40–60% of adults in the general population and possibly similarly frequent in athletes [2, 6]. Fortunately, chest pain is most often benign and of non-cardiac origin, but in the acute setting the initial assessment is to rule-out potentially life-threatening causes. The most common cardiac cause of chest pain is ischemic heart disease (IHD) therefore the initial assessment includes assessment of risk factors for IHD:

- Age
- Gender
- Smoking history
- Plasma lipids
- Familiar predisposition
- Further cardiovascular risk factors

Other diagnostic considerations are pulmonary embolism, cardiomyopathies, peri-myocarditis, coronary anomaly, aortic stenosis (in young athletes often caused

Table 6.1 Common causes of chest pain

Cardiac causes	
Structural heart disease	• Ischemic heart disease • Cardiomyopathy
Congenital	• Coronary artery anomaly • Bicuspid aortic valve leading to premature aortic stenosis
Drug-induced	• Cocaine • Other substances used for performance-enhancing purposes
Inherited	• Familial hypercholesterolemia leading to premature ischemic heart disease • Marfan syndrome with aortic dissection
Infectious	• Perimyocarditis
Valvular	• Aortic stenosis
Traumatic	• Commotio cordis
Autoimmune	• Kawasaki disease (coronary aneurysms)
Non-cardiac causes	
Muscular-skeletal	• Costochondritis • Intercostal/Serratus anterior strain • Rib stress fracture
Gastrointestinal	• Gastroesophagal reflux • Peptic ulcer • Hiatal hernia
Respiratory	• Asthma (including exercise-induced) • Pneumothorax (spontanously or traumatic) • Pulmonary embolism • Pleuritis • Pneumonia
Miscellaneous	• Malignancy • Herpes zoster • Psychogenic (e.g. anxiety)

prematurely by bicuspid aortic valve), mitral valve prolapse or aortic dissection (usually associated with systemic disease such as the Marfan syndrome). The main causes of acute chest pain in younger athletes origin from the muscular-skeletal system and the lungs [7], whereas in adults acute coronary syndrome, gastrointestinal disease, muscular-skeletal origin and (less common) pericarditis, pneumonia and pulmonary embolism should be suspected [8] (Table 6.1).

- In general, chest pain among athletes is often an unspecific symptom of non-cardiac origin such as gastroesophageal reflux, asthma or muscular-skeletal pain.
- Nonetheless, it should always be evaluated thoroughly to exclude any cardiac cause, given the significance of sudden cardiac arrest in this population.

6.4 Dyspnea and/or Reduced Exercise Capacity

Among athletes, exercise-induced shortness of breath is usually reported as reduced exercise capacity. If reported with concurrent symptoms as coughing and wheezing there is often a pulmonary cause such as exercise-induced asthma or vocal cord abnormalities [9].

- With a prevalence of 8%, asthma or airway hyperreagibility are the most common chronic medical conditions among Olympic athletes [10].

If a cardiac cause is suspected, cardiomyopathy should particularly be excluded in younger athletes whereas in older ones the prevalence of coronary artery disease poses the main risk factor for acute cardiac events during exercise (see Chap. 6). If a murmur appears, valvular heart disease (bicuspid aortic valve leading to premature aortic stenosis or regurgitation, or mitral valve prolapse), or innate cardiac disease (atrial or (less common) ventricular septal defects) should be suspected. Coarctatio aortae should particularly be considered if reduced exercise capacity is combined with blood pressure differences between upper and lower extremities and if additional symptoms such as abdominal angina and early fatigue of the legs during exercise are present. It also sometimes coincides with bicuspid aortic valve (see Chap. 24).

6.5 Palpitations

There is not a clear definition of palpitations but the symptom is often described by the athlete as an increased awareness of their own heartbeat [11]. Palpitations are common in athletes [2], and the symptom can represent the whole spectrum from benign ectopic beats to life-threatening ventricular tachycardia. To distinguish the different conditions it can often be helpful to ask the athlete to tap the rhythm of their palpitations or to choose from a range of rhythms tapped by the physician [12]. Palpitations followed by other cardiac symptoms such as lightheadedness or even syncope as well as shortness of breath most likely indicate a significant clinical arrhythmia. Correspondingly, it is important whether the palpitations are

- fast or slow
- regular or irregular
- start and terminate abruptly or gradually
- triggered by exercise or other external or internal factors.

Palpitations can be of *cardiac* or *non-cardiac* origin. The *cardiac* causes can either be secondary to structural heart disease or represent a primary cardiac arrhythmia. The structural conditions that can enhance cardiac irritability are most often due to cardiomyopathy (inherited as well as acquired), ischemic heart disease or valvular disease. The primary cardiac arrhythmias are of supraventricular and ventricular origin and include genetic channelopathies such as the Long QT syndrome, Brugada syndrome and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT; see Chap. 21). The *non-cardiac* causes of palpitations are often due to

- reactive sinus tachycardia caused by systemic disease (e.g. thyroid disease and anemia),
- psychosomatic disorders (e.g. anxiety, depression and panic attacks) and
- medication or drugs (e.g. alcohol, caffeine, performance-enhancing drugs/stimulants).

The most common cause of palpitations among athletes at rest are ectopic beats that decrease during exercise. Supraventricular arrhythmias are often influenced by vagal maneuvers, with a temporary reduction in heart rate or sudden interruption. In contrast, ventricular arrhythmias remain unaffected. Among middle-aged male master athletes, atrial fibrillation is up to five times more common than in age-matched non-athletes [13] (see Chap. 34). Athletes often suffer from irregular heart rhythm, associated weakness, fatigue and polyuria (as an effect of increased secretion of atrial natriuretic peptide).

Beyond medical history and physical examination, further clinical evaluation should always include ECG and echocardiography, and if no signs of structural heart disease are found, they should be followed by exercise testing and/or Holter monitoring. If the symptoms cannot be correlated to a specific finding during these tests, loop recorders or similar devices could be considered.

6.6 Family History

Obtaining a family history is of crucial importance as many of the causes of sudden cardiac arrest or death in young athletes are due to inherited cardiac disease. The goal of the investigation is to identify athletes at higher risk for inherited cardiovascular disorders. These include cardiomyopathies, valvular heart disease, channelopathies and other primary arrhythmic disturbances. Many of these diseases carry autosomal-dominant inheritance patterns; thus, a family history can specify the evaluation.

- If there is a positive family history of sudden unexplained death before the age of 50 years it is of utmost importance to obtain further information for risk assessment and stratification of the athlete.

This includes creating a pedigree to clarify the exact relation of the family member who died, getting an overview of who could be at risk and, if possible, clarifying the exact circumstances and cause of death from the deceased (e.g. autopsy). First degree relatives of SCD victims are at highest risk, as shown in a recent Danish study [14], and if genetic testing reveals positive findings it can be followed with family cascade screening.

6.7 Physical Examination

There is an ongoing debate about the ideal screening concept to prevent SCD in sport. The crux of the matter is the inclusion of an ECG, and most of the sports and cardiologic associations, like the European Society of Cardiology (ESC), the International Olympic Committee (IOC), the Federation Internationale de Football Association (FIFA), and various U.S. professional sporting organizations recommend a cardiac screening integrating an ECG.

However, the American Heart Association (AHA) still stands with a more traditional concept, using a 14-point personal and family history questionnaire (Fig. 6.2)

The 14-Element AHA Recommendations	
Medical history*	
Personal history	
1.	Chest pain/discomfort/tightness/pressure related to exertion
2.	Unexplained syncope/near-syncope†
3.	Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise
4.	Prior recognition of a heart murmur
5.	Elevated systemic blood pressure
6.	Prior restriction from participation in sports
7.	Prior testing for the heart, ordered by a physician
Family history	
8.	Premature death (sudden and unexpected, or otherwise) before 50 years of age attributable to heart disease in ≥ 1 relative
9.	Disability from heart disease in close relative < 50 years of age
10.	Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members
Physical examination	
11.	Heart murmur‡
12.	Femoral pulses to exclude aortic coarctation
13.	Physical stigmata of Marfan syndrome
14.	Brachial artery blood pressure (sitting position)§
•Parental verification is recommended for high school and middle school athletes. †Judged not to be of neurocardiogenic (vasovagal) origin: of particular concern when occurring during of after physical excertion. ‡Refers to heart murmurs judged likely to be organic and unlikely to be innocent: auscultation should be performed with the patient in both the supine and standing positions (or with valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction. §Preferably taken in both arms.	

Fig. 6.2 The 14-Element AHA recommendations for preparticipation cardiovascular screening of competitive athletes (adapted from [17])

and a physical examination [15–19]. The questions focusing on medical history are mainly based on consensus expert opinion, and there are only few data regarding feasibility of these questions among various ethnic and socio-economic subgroups. The reliability of the questions is unclear, and existing data is ambiguous. With respect to the AHA-based history questions, prior to physician review, athletes reported positive rates in up to 35–60%, while other studies report positive history responses in only 1% of athletes [20, 21]. However, this once again highlights the importance of an accurate clinical assessment by a dedicated physician. Moreover,

- It should be kept in mind that in many athletes SCD occurs without suggestive preceding symptoms [22].

In a recent meta-analysis [23] physical examination even had a lower sensitivity than the athlete's history, but nevertheless, a relatively high specificity and negative likelihood ratio. However, despite all debates and limitations, a personal clinical evaluation is still the cornerstone of every medical assessment, including a population of competitive athletes. It may provide important information that relies on intuition and experience, not only regarding rather subjective questionnaires but also clinical findings during physical evaluation. Primary findings which may prompt further assessment of an athlete's heart and cardiovascular system are heart murmurs, clinical stigmata of connective tissue disease (e.g. Marfan syndrome) and high blood pressure [23].

The physical examination of an athlete should be focussed and clearly structured. As such, a standard approach, as in the assessment of other organs, would be as following:

1. Inspection
2. Palpation and percussion (incl. pulse and blood pressure measurement)
3. Auscultation

1. *Inspection.* Physical assessment of an athlete starts with an initial clinical impression. Complexion or obvious stigmata of an underlying disease should be noted. Particularly, clinical stigmata suggesting connective tissue disease are important to recognize, as affected athletes (e.g. in Marfan Syndrome) may develop aortic aneurysm with consecutive dissection. Table 6.2 provides an overview of the current criteria to assess an athlete regarding possible Marfan syndrome (revised Ghent nosology [24]).
2. *Palpation and percussion.* The examination of the heart should start with the classification of the *heart rhythm* as
   (a) regular
   (b) partly irregular (due to sinus arrhythmia or premature beats) or
   (c) absolutely irregular (due to atrial fibrillation).

   An accurate assessment of the athlete's *blood pressure* (BP) is crucial as it may gain false positive ("white coat hypertension") or false negative ("masked hypertension") results. The athlete should ideally be examined in a supine or sitting position with the arm supported at the level of the heart

Table 6.2 Overview of the «Revised Ghent Nosology/Criteria» for the diagnosis of Marfan Syndrome (MFS) [24])

In the absence of family history	
1. Aortic root diameter (Z-score ≥ 2) and ectopia lentis	MFS[a]
2. Aortic root diameter (Z-score ≥ 2) and causal FBN1 mutation	MFS
3. Aortic root diameter (Z-score ≥ 2) and systemic score ≥ 7 points (see below)	MFS[a]
4. Ectopia lentis and causal FBN1mutation with known aortic root dilatation	MFS
In the presence of family history	
5. Ectopia lentis and family history of MFS (as defined above)	MFS
6. Systemic score ≥ 7 points (see below) and family history of MFS (as defined above)	MFS[a]
7. Aortic root diameter (Z-score ≥ 2 above 20 years old, ≥3 below 20 years) and family history of MFS (as defined above)	MFS[a]
Scoring of systemic features of MFS	*Score*
1. Wrist AND thumb sign (alt.: wrist OR thumb sign)	3 (1)
2. Pectus carinatum deformity (pectus excavatum or chest asymmetry)	2 (1)
3. Hindfoot deformity (plain pes planus)	2 (1)
4. Pneumothorax	2
5. Dural ectasia	2
6. Protrusio acetabuli	2
7. Reduced upper/lower body segment ratio AND increased arm/height AND no severe scoliosis	1
8. Scoliosis or thoracolumbar kyphosis	1
9. Reduced elbow extension	1
10. Facial features (at least 3 out of the following 5: dolichocephaly, enophtalmos, downslanting palpebral fissures, malar hypoplasia, retrognathia)	1
11. Skin striae	1
12. Myopia >3 diopters	1
13. Mitral valve prolapse (all types)	1
Maximum Score	20
Indicative of systemic involvement	≥7

The Systemic score (lower part) refers to signs that can be detected by physical examination
[a]Caveat: without discriminating features of other forms of connective tissue syndromes AND certain genetic profiles (see [24] for details)

and after, at least, five minutes rest. Systolic pressure tends to be 2–3 mmHg higher and the diastolic pressure a similar degree lower in supine position. Mercury sphygmomanometers provide more accurate measurements than aneroid sphygmomanometers. Oscillometric BP measuring devices give readings that are typically lower than BP values obtained with the auscultatory method. A properly sized cuff use is essential, as an inappropriately small cuff provokes higher than intra-arterial pressure. As a rule of thumb, the length of the cuff bladder should be 80%, and the width at least 40% of the circumference of the upper arm. The cuff should be inflated to a pressure approximately 30 mmHg higher than the estimated systolic pressure (by pulse palpitation) and deflated slowly, at a rate of 2–3 mmHg per heartbeat.

Systolic pressure is equal to the pressure at which the pulse can first be detected by auscultation ("Korotkoff phase I") and the brachial/radial pulse can first be palpated again. As the cuff is further deflated, a brief period may follow during which the sounds soften (phase II) and then return to sharper

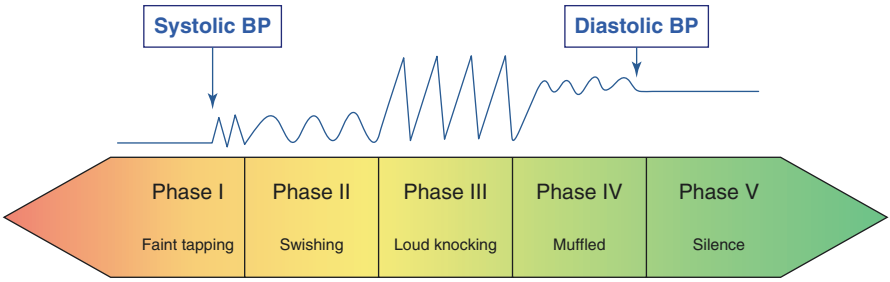

Fig. 6.3 The five so-called Korotkoff phases that can be discriminated when measuring blood pressure by auscultation of the brachial artery

sounds again (phase III) before the pulse is first abruptly muffled (phase IV) and finally disappears (phase V), which generally equals the diastolic blood pressure (with the possible exception of high cardiac output in well trained athletes, where the pulse is continuously detectable) (Fig. 6.3).

(d) BP should at least once be measured on both arms to detect possible vascular perfusion deficits.

3. *Auscultation.* For more than a century, cardiac auscultation was the mainstay of cardiac examination. Despite all technical advances and possibilities to use cardiac imaging as a more accurate method to detect structural heart disease, a doctor's stethoscope has still the right to exist and clinical situations in which it should be used primarily, although the accuracy has been questioned repetitively [25, 26]. However, this cost-effective and highly available technique should be provided adequately to increase diagnostic accuracy and forego false positive findings. Examiner variability is a matter of fact; however, auscultation has a reported sensitivity of 70% and a specificity of 98% for the detection of valvular heart disease.

Cardiac auscultation is commonly performed with the athlete lying in a supine position in the end-expiratory phase. However, by changing the body position (e.g. left lateral position or prone) and varying respiratory phases (e.g. end-inspiratory, Valsalva manoeuvre, etc.) specific cardiac conditions can be provoked in case of clinical suspicion. The findings in auscultation can roughly be separated into *heart sounds* and *heart murmurs.*

6.8 Heart Sounds

First heart sound (S1): The first heart sound (S1), normally best heard over the cardiac apex, consists of two high-frequency components: the first component is attributed to the dominant mitral valve closure while the second component refers to the closure of the tricuspid valve. The intensity of the valve closure sound is increased alongside an

- increased transvalvular gradient (e.g. in mitral stenosis)
- increased transvalvular flow (e.g. left-to-right shunt in ventricular septal defect or high output state)
- shortened diastole (tachycardia)
- short PR interval (pre-excitation syndrome).

Restricted valve mobility decreases the intensity of S1 (e.g. fibrosis or calcification of the mitral valve). Furthermore, S1 may also be diminished if the valve leaflets are already half-closed at the onset of systole, as it may occur in left ventricular systolic dysfunction. Abnormal splitting of S1 can result from delayed closure of the tricuspid valve (e.g. in patients with atrial septal defect). A widely split S1 can also occur in complete right bundle branch block (or after ectopic beats of LV origin).

Second heart sound (S2): The second heart sound consists of two components: the aortic (A2) and pulmonary valve (P2) closure. P2 is best heard over the upper left sternal border, whereas A2 is widely transmitted to the right second interspace, along the sternal border and to the cardiac apex. Separation of A2 and P2 particularly occurs during inspiration, which allows comparison of the relative intensities of the two components and differentiation to a third heart sound (S3). This can best be heard over the left second interspace. Increased intensity of S2 occurs in

- systemic hypertension
- coarctation of the aorta
- ascending aortic aneurysm (mainly due to a louder A2)
- pulmonary hypertension (mainly due to a louder P2).

Decreased intensity of S2 is a rare finding in athletic screening but may occur in very low arterial diastolic pressure (e.g. in severe aortic regurgitation), immobilisation of the aortic valve due to calcification and severe aortic stenosis. Increased separation of A2 and P2 during inspiration may result from complete right bundle branch block (or premature beats or idioventricular rhythm of LV origin), the Wolff-Parkinson-White syndrome with LV pre-excitation and haemodynamic causes like pulmonary arterial hypertension of any aetiology. Fixed splitting of S2 is a relatively specific finding in case of interatrial communication (e.g. large atrial septal defect) or due to any condition with severe right ventricular failure. Paradoxical splitting (A2 following P2) may also be provoked during expiration, and is mostly seen with left bundle branch block, premature beats of RV origin or pre-excitation of the RV in Wolff-Parkinson-White syndrome.

Third (S3) and fourth (S4) heart sounds: S3 occurs at the very beginning of the passive filling phase of the ventricles and is a normal and common finding in healthy young athletes. However, beyond the age of 35–40 it may also suggest increased volume load due to a pathologic underlying condition.

In contrast, S4, which coincides with atrial systole, suggests increased LV pressure and is an unusual and suspicious finding in athletes. S3 as well as S4 are both low-frequency diastolic sounds and are thus best heard if the stethoscope is placed

with slight pressure over the cardiac apex or over the lower left sternal border. Noise and murmurs of higher frequency will be eliminated like this and differentiation to components of S1 or S2, respectively, is easier.

So called aortic ejection sounds can occur in association with a geometrically deformed aortic valve (e.g. bicuspid aortic valve or aortic root dilatation). Another quite common finding is a mid-systolic click, suggesting mitral valve prolapse. The detection of a mitral-valve prolapse is important: Beside issues regarding valve function and training recommendations, this condition may be associated with concomitant cardiovascular anomalies such as cardiomyopathy, atrial septal defect, connective tissue disease (e.g. Marfan syndrome) or systemic lupus erythematosus.

Finally, a pericardial rub, that can be heard best during atrial systole, ventricular systole, and the rapid-filling phase of the ventricle ("three-component rub") is generated by friction of the two inflamed layers of the pericardium, suggesting peri(myo-)carditis.

6.9 Heart Murmurs

Heart murmurs are a common finding, particularly in young athletes, and are frequently of physiologic origin. However, an accurate characterisation of the murmur and thus detection of an eventual pathologic underlying condition is crucial. Heart murmurs can be characterised by five factors:

1. *Systolic versus diastolic murmurs* (including specification of whether the murmur is proto-, meso-, tele- or holosystolic or –diastolic, respectively).
2. *Localization* where the murmur can be heard loudest, according to the classical auscultation spots: Second intercostal space (ICS) parasternal right (aortic valve) and left (pulmonary valve), fifth ICS parasternal right (tricuspid valve), fifth ICS medio-clavicular left (mitral valve) and third ICS parasternal left (the so-called "Erb's point"). Transmission spots like the carotid arteries (aortic stenosis) or the left axilla (mitral valve pathology) should also be checked regularly (Fig. 6.4). Furthermore, the examiner should assess whether a murmur can be heard interscapular (aortic isthmus stenosis).
3. *Frequency* of a heart murmur (high or low frequency) can help differentiating between different conditions (e.g. the low frequent, harsh systolic murmur of an aortic stenosis versus the higher frequent systolic murmur of a mitral regurgitation).
4. The *shape* of a murmur is quite characteristic for certain pathologies and relies on the diagram of murmur intensity over time, as in a phonocardiogram:
   (a) Crescendo (increasing)
   (b) Decrescendo (diminishing)
   (c) Crescendo-decrescendo (increasing-decreasing or diamond/spindle shaped)
   (d) Plateau (unchanged in intensity)

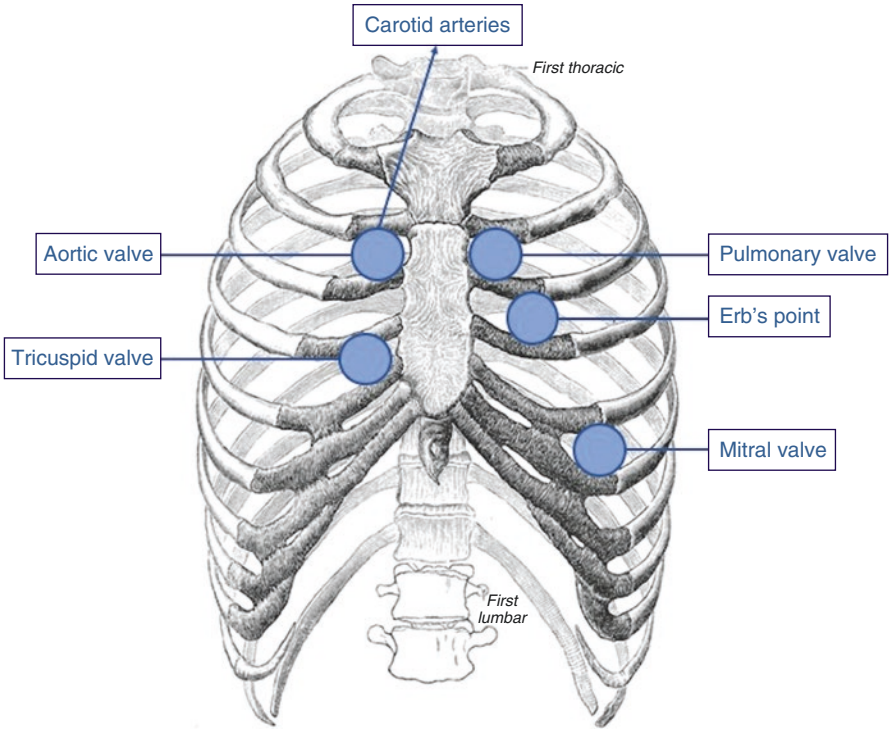

Fig. 6.4 Overview of the most important cardiac auscultation points. It is important to note that murmurs implicating disorders of the aortic valve (i.e. aortic stenosis) typically can also be heard at the level of the carotid arteries

Assessing the shape of a murmur can, for example, help differentiating between the classic decrescendo shape of an aortic regurgitation and the spindle shaped murmur of aortic stenosis.

5. The *intensity* of a murmur may allow assessing the severity of a valve pathology:
   (a) Grade I (1/6) is the faintest murmur that can be heard (with difficulty)
   (b) Grade II (2/6) murmur is also a faint murmur but can be identified immediately
   (c) Grade III (3/6) murmur is moderately loud
   (d) Grade IV (4/6) murmur is loud (possibly associated with a palpable thrill)
   (e) Grade V (5/6) murmur is very loud (cannot be heard without the stethoscope)
   (f) Grade VI (6/6) murmur is the loudest (can be heard without the stethoscope)
      - A *systolic murmur* generally starts with or after S1 and terminates before or at S2 and is therefore recognised by identifying S1 and S2.
      - A diastolic *murmur* starts with or after S2 and ends at or before S1.

- Continuous *murmurs* continue through the whole systolic and diastolic phase, without interruption.
- *Mid*-systolic *murmurs* are most commonly benign flow murmurs due to physiologic flow, increased flow rate across a normal semilunar valve or due to aortic valve sclerosis. This kind of systolic murmur is present in up to 60% of athletes, however, in around 90% of these it is associated with a normal echocardiogram. Benign "flow" murmurs also occur when the relative flow volume across the semilunar valve is increased, as it occurs in high-level endurance athletes, during pregnancy but also in individuals with anaemia or thyrotoxicosis.

To distinguish between fixed valvular aortic stenosis (AS) and dynamic LV outflow obstruction (obstructive HCM) some clinical tests are established. With the patient changing from a squatting into a changing position, the intensity of the murmur in HCM increases, whereas the murmur of valvular aortic stenosis will decrease. In the straining phase of Valsalva manoeuvre, the murmur of HCM increases in intensity; both the intensity of the murmur and the carotid pulse volume decline with Valsalva in AS. It can be difficult to distinguish between a long mid-systolic murmur and a holosystolic regurgitant murmur in certain situations. Handgrip manoeuvre may help to distinguish between a mitral regurgitation murmur, where the intensity of the murmur increases (increased afterload effect), and an AS murmur, where intensity usually decreases.

- *Early systolic murmurs* most often result from mitral regurgitation (MR) and generally have a "plateau-configuration".
- *Late systolic murmurs* are most commonly caused by mitral valve prolapse. They are best heard with the diaphragm of the stethoscope and are usually preceded by single or multiple clicks.
- There are three classical causes of *holosystolic murmurs*—MR, tricuspid regurgitation and ventricular septal defect (VSD). The holosystolic murmur of MR is high pitched and is therefore best heard with the diaphragm of the stethoscope and the patient in the left lateral decubitus position [27].
- *Early diastolic murmurs* are most often due to aortic or pulmonary regurgitation and appear in decrescendo-shape with, in addition, a slight systolic murmur due to increased ventricular stroke volume.
- *Mid-diastolic murmurs* classically result from turbulent flow across the atrioventricular valves during the rapid filling phase because of mitral ("opening snap") or tricuspid valve stenosis. In mitral valve stenosis (MS) it can be stated that the longer the duration of the murmur, the more severe the MS might be.
- *Late diastolic or pre-systolic murmurs* usually have a crescendo configuration and result from increased flow across the mitral or tricuspid valve (e.g. in mitral or tricuspid stenosis, atrial fibrillation or left-to-right shunts).

Clinical Pearls An athlete's personal, systemic and particularly family history frequently detects "red flags" which lead to the diagnosis of a relevant underlying cardiovascular disease.

- Physical examination mainly targets connective tissue disease (with the potential for aortic dilation and dissection), as well as hypertensive blood pressure and cardiac murmurs.
- Blood pressure assessment needs to be performed correctly to differentiate physiologic findings (including "white coat hypertension") and pathologic findings (including "masked hypertension").
- Cardiac auscultation is challenging but, performed correctly, it may reliably detect underlying cardiac disease—particularly, valvular heart disease. It should follow a strict and standardized procedure (sounds vs. murmurs, systolic vs. diastolic, localization, shape, frequency, intensity, etc.).

Review

Questions

1. A 26-year old male leisure endurance athlete reports recurrent lightheadedness after brisk changing from a sitting to a standing position (i.e. after a meal). Two days ago, he experienced a syncope during his regular training (jogging of moderate intensity). The syncope appeared unheralded and the patient does not remember any warning symptoms and denied amnesia. However, due to his syncope he suffered from bruises on the knees, elbows and the forehead. What is your suspicion after knowledge of the patient's history?
2. You see a 19-year old asymptomatic male basketball player for routine precompetition examination. During physical examination some clinical findings are suspicious. The skin on his back shows multiple slightly blue "scar-like" striae and during auscultation you can detect a mid-systolic click with a moderately loud tele-systolic murmur. What is your suspicion and what tests would you add?
3. A 54-year old marathon runner asks for consultation due to massive headaches, particularly during exercise, slowly decreasing vision and with a recent laboratory test which exhibited moderate renal failure. The patient's personal history is normal, and his family history only highlights systemic hypertension of his father. Physical examination shows normal findings with unsuspicious auscultation and normal blood pressure measurement (135/85 mmHg on both arms in supine position). In an additional exercise test the athlete performed well without abnormal ECG changes during exercise but headache and pronounced increase of blood pressure (up to 240/110 mmHg until exhaustion). What are your thoughts and what examination should be added to confirm your suspicion?

Answers

1. The patient's history of the syncopal event is highly suspicious for cardiac/arrhythmic etiology. This is due to four classical points: The syncope occurred during exercise and without any prodromal symptoms. Amnesia has been denied but he suffered from (minor) injury (bruises) due to the syncope.
2. Skin striae are highly suspicious for connective tissue disease and are part of the so-called "Ghent Criteria" characterizing Marfan Syndrome. Thus, the next clinical step would be to complete these clinical criteria. The auscultatory findings are classical for mitral valve prolapse which is also part of the Ghent criteria and frequently seen in patients with connective tissue disease. To confirm this suspicion transthoracic echocardiography should be performed. It is also crucial, particularly in contact sports, to assess the diameter of the ascending aorta as aortic aneurysm (and possible dissection) may occur as another clinical finding in these patients.
3. The patient's history and physical examination raises suspicion for "masked hypertension". Systemic hypertension is genetically linked (positive family history) and may clinically appear with headaches. Ocular and renal damage can also be explained by longstanding hypertension. Although "office" blood pressure measurements were normal "masked hypertension" is most likely as up to 40% of athletes with exercise hypertension show underlying (often undetected and masked) hypertension. Thus, in a next diagnostic step ambulatory blood pressure monitoring (24-h automatic device or self-measurement at home) should urgently be recommended.

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