MINISTRY OF PUBLIC HEALTH OF UKRAINE

ZAPOROZHYE STATE MEDICAL UNIVERSITY

DEPARTMENT OF PHYSICAL REHABILITATION, SPORTS MEDICINE, PHYSICAL TRAINING AND HEALTH

URGENT STATES IN SPORTS MEDICINE

Study book

Zaporozhye, 2014

Mikhalyuk E.L., Cherepok A.A., Malakhova S.N. Urgent states in sports medicine. Study book. - Zaporozhye: ZSMU, 2014. - 73 p.

Authors:

Mikhalyuk E.L., Doctor of Medicine, professor, the head of the department of physical rehabilitation, sports medicine, physical training and health, Zaporozhye State Medical University.

Cherepok A.A., Candidate of Sciences, assistant of the department of physical rehabilitation, sports medicine, physical training and health, Zaporozhye State Medical University.

Malakhova S.N., Candidate of Sciences, assistant of the department of physical rehabilitation, sports medicine, physical training and health, Zaporozhye State Medical University.

Reviewers:

Syvolap V.V. - Doctor of Medicine, professor, the head of the department of propedeutics of internal deseases with care of patients, Zaporozhye State Medical University.

Pertsov V.I. - Doctor of Medicine, professor, the head of the department of medical catastrophes and military medicine, Zaporozhye State Medical University.

Study book compiled based on the current curriculum and programs for educational discipline "Physical Rehabilitation and Sports Medicine" for medical students in higher educational institutions III-IV levels of accreditation for training areas "Medicine" in 1201, for specialties 7.12010001 "Medicine" 7.12010002 "Pediatrics" according to educational qualification characteristics and educational and vocational training programs approved by the Ministry of Health of Ukraine orders from 16.04.03 № 239 and from 28.07.03 № 504, and experimental curriculum of Ministry of Health of Ukraine, developed on the principles of the European credit transfer system and approved Order of the Ministry of Health of Ukraine from 31.01.2005, № 52.

Study book is intended for independent work of students of medical faculties IV course in preparation for the practical training in the discipline "Physical rehabilitation and sports medicine."

Study book discussed and approved at a meeting of the Cyclic Methodical Committee from Therapeutic Disciplines of Zaporozhye State Medical University and recommended for publication by the Central Methodical Council of Zaporozhye State Medical University (the record No 3 as of 13.02.2014).

INTRODUCTION

While most sports injuries are nonemergent and musculoskeletal in nature, there are certain life- and limb-threatening injuries that the *field-side physician* (FP) must be prepared to handle immediately. The most important step in the management of field-side emergencies is preparation, and depending on the setting of the event and the level of competition, resources may be limited. The FP must at a minimum have ready access to appropriate health care personnel to assist in an emergency, appropriate medical supplies and emergency equipment, immediate access to a telephone, and the ability to transport an athlete to a medical facility. It would also be advisable to be certified in *basic life support* (BLS), *advanced cardiac life support* (ACLS), and *advanced trauma life support* (ATLS) and to have a working knowledge of the common and uncommon injuries specific to the event being covered.

GENERAL APPROACH TO THE FALLEN ATHLETE

When approaching the fallen athlete, the field-side evaluation should be both rapid and focused. The “primary survey” should follow the “ABCDE” approach taught by ATLS (Committee on Trauma, 1997) and should occur where the athlete is found. They should initially be left in that position unless they are prone and unconscious or there is a problem performing the “ABCs” (Luke and Micheli, 1999; Blue and Pecci, 2002), in which case they should be logrolled to a supine position.

The logroll should ideally be a four person technique in which the team leader is at the victim’s head maintaining in-line immobilization of the head and neck, while the other three members of the team are controlling the torso, hips, and legs. The athlete should be turned in the direction of the three assistants according to the count of the leader and then onto a spine board placed under the athlete.

If an athlete is wearing an appropriately fitted helmet, neither the helmet nor its chin strap should be removed. Padding or sandbags should be placed around the helmet and the shoulders; hips and legs immobilized. The face-guard can easily be removed by prying or cutting it off for access to the airway. The helmet and shoulder pads should be considered a single unit the removal of either one necessitates the removal of the other, as leaving only one of them in place forces the neck out of a neutral position (Haight and Shiple, 2001; Gastel et al, 1998). If the athlete is not wearing a helmet, a rigid cervical collar should be applied with in-line immobilization of the spine.

After the primary survey is complete and the patient stabilized, a more detailed secondary survey should be performed either on the field or on the sideline, depending on the status of the athlete and the environmental conditions.

The factors to be considered while evaluating the fallen athlete include whether or not the injury was witnessed / unwitnessed and / or traumatic / atraumatic. The age, general conditioning, and specific medical conditions of the athlete should be considered, as well as the general characteristics of the sport, such as the amount of contact (i.e., collision, limited contact, and noncontact), the degree of speed involved, and the duration of the event. Finally, the environmental conditions must be considered as both a potential causative and / or exacerbating factor in the injury.

After the initial examination of the patient is completed, the FP should identify any problem areas and categorize them as being of either an immediate or potential life threatening / disabling nature and treat accordingly. Frequent reevaluation of the injured athlete is a must.

IMMEDIATE LIFE THREATENING INJURIES

RESPIRATORY COMPROMISE

Upper airway obstruction

Although rare in organized sports, respiratory arrest can result from *upper airway obstruction* (UAO). Signs include respiratory distress with little or no air movement, significant accessory muscle use, and stridorous, wheezing, or snoring breath sounds. If the athlete is unconscious, the airway should be opened with a jaw-thrust maneuver to keep the tongue from occluding the airway and an oral/nasal airway inserted as necessary. In-line repositioning of the head/neck may be necessary to establish airway patency if the neck is significantly contorted. The oropharynx should be inspected for foreign bodies and removed if visualized; however, blind finger sweeps are not recommended in either children or adults. Significant facial / mandibular trauma with resultant loss of sup port of the tongue or with blood, secretions, and loose teeth in the pharynx can produce UAO, particularly in the unconscious athlete who has lost protective airway reflexes. Other causes of UAO, such as airway edema from anaphylaxis, inhalation burn injuries, or an expanding neck or retropharyngeal hematoma from neck trauma should be considered, with early intubation a priority. Surgical airway capability is a necessity as well.

Laryngeal fracture

This rare injury occurs after direct trauma to the ante rior neck. Signs include stridor, hoarseness, subcuta neous emphysema, and perhaps bony crepitus and a palpable fracture. Although airway obstruction may not be immediate, it can rapidly progress to this stage because of resultant edema and as with other causes of obstruction, early intubation is a priority; surgical airway capability is again a

necessity.

Pneumotorax

A simple pneumothorax may be spontaneous (i.e., rupture of a bleb) or traumatic, with spontaneous pneumothoraces occurring more often in sports that involve changes in intrathoracic pressure (i.e., scuba diving and weightlifting) (Partridge et al, 1997) and traumatic pneumothoraces occurring secondary to rib fractures. Symptoms may include unilateral chest pain, dyspnea, and cough. Immediate treatment is rarely needed unless the patient is severely dyspneic or the pnuemothorax is open or under tension. Those with a stable simple pneumothorax should be given oxygen and transported to a medical facility for further evaluation and management.

Open pneumotorax

This is defined as a pneumothorax accompanied by an open wound to the chest (sucking chest wound). Treatment consists of placing an occlusive dressing over the open wound and taping it down on three sides to create a one-way valve that allows air to exit without reentering till a definitive thoracostomy tube can be placed.

Tension pneumotorax

This occurs when a pneumothorax is accompanied by progressive accumulation of air in the pleural space with the resultant increase in intrathoracic pressure causing a shift of mediastinal structures away from the pneumothorax as well as a decrease in venous return and cardiac output. In addition to the previously listed symptoms, these athletes may have tracheal deviation away from the affected side with jugular venous distention and hypotension. This is a true medical emergency that requires immediate treatment by needle decompression of the chest

with a large (14–16 gauge) needle or catheter inserted in the anterior chest wall in the second intercostal space at the midclavicular line, followed by placement of a thoracostomy tube.

CARDIAC ARREST

Although devastating when it occurs in a young athlete, a traumatic sudden death is extremely rare with incidence varying depending on the age of the athlete and the sporting event (O'Connor et al, 1998). The most common cause of sudden cardiac death in young athletes is congenital cardiovascular structural abnormalities with hypertrophic cardiomyopathy leading the list, followed by coronary artery anomalies and myocarditis (McCaffrey et al, 1991). The most common cause in older athletes (age > 30–35) is atherosclerotic heart disease causing acute ischemic events.

The field-side treatment of any cause of cardiac arrest should follow *advanced cardiac life support* (ACLS) guidelines with attention to early *cardiopulmonary resuscitation* (CPR) and defibrillation as indicated. An equally important task for the FP is to identify those athletes who are having warning signs of cardiac disease and dysrhythmias, such as sudden unexplained syncope or collapse, exertional syncope, early fatigue, or anginal chest pain during or immediately following exertion (O'Connor et al, 1998). Strong consideration should be given to withholding these athletes from further competition until a thorough evaluation is performed.

ANAPHYLAXIS

Anaphylactic reactions are acute systemic hypersensitivity reactions that can be idiopathic, exercise-induced, or allergen-induced, and although rare, they can progress very rapidly and prove fatal if unrecognized. Insect stings (esp. hymenoptera) may be a cause of sports related anaphylaxis.

The symptoms of anaphylaxis may include urticaria / angioedema, upper airway edema, dyspnea, wheezing, flushing of skin, dizziness / hypotension / syncope, gastrointestinal symptoms, rhinitis, and headache (Winbery and Lieberman, 1995). Symptom onset is typically rapid (within 5–30 min of exposure), and in its most severe form can progress to severe bronchospasm, airway edema, and fatal cardiovascular collapse.

Treatment consists of prompt attention to the ABCs, followed by treatment with 100% oxygen, epinephrine (1:1000) 0.3–0.5 mL in adults or 0.01 mg/kg in children given subcutaneously or intramuscularly and repeated every 10–15 minutes as needed, IV (intravenous) fluids if hypotensive, beta-agonists by nebulizer if bronchospasm is present, antihistamines (H1 and H2 blockers), and glucocorticoids if available. The athlete must be rapidly transported to a medical facility as continued observation will be required.

SEVERE HEMORRHAGE

Hemorrhage in the athlete may be the result of lacerations, fractures, vascular disruptions, or visceral organ or muscle disruptions. It can manifest as either massive external bleeding or insidious and occult internal bleeding. Control of external bleeding should follow the basic principles of hemostasis, which include steady direct pressure over the bleeding site and over larger arteries proximal to the site of injury,

as well as elevation of the affected body part. Blind clamping of bleeding vessels and tourniquet application (with the possible exception of a traumatic amputation) are not recommended.

Scalp lacerations can cause significant hemorrhage and often go unnoticed if the athlete is lying on his back or is strapped to a spine board.

Occult bleeding may produce delayed signs and symptoms, and what may at first appear to be an atraumatic incident may actually have been caused by recent unnoticed or unwitnessed trauma (Blue and Pecci, 2002).

Potential injuries, which may be major sources of occult blood loss, include hemorrhage into the thoracic and abdominal cavities, the soft tissues surrounding major long bone fractures, the retroperitoneal space secondary to a pelvic fracture, and as a result of penetrating torso injury (Committee on Trauma, 1997).

Signs and symptoms of hypovolemic shock include altered sensorium, pale and cool extremities with a decreased capillary refill, weak, thready, and rapid pulses, hypotension, tachycardia, and tachypnea.

Treatment should follow ATLS protocol and at a minimum two large bore peripheral IVs should be started and oxygen administered. Consideration should be given to starting crystalloid fluids, although there is some debate as to whether or not aggressive fluid resuscitation may actually be more detrimental to patients with certain types of injuries, and one should consider the concept of permissive hypotension when managing hypovolemic shock in the alert patient (Fowler and Pepe, 2002).

POTENTIAL LIFE THREATENING/DISABLING INJURIES

HEAD INJURY

Head injuries in sports are quite common and often provoke anxiety and uncertainty. Fortunately, the most common head injury in sports is a concussion and 90% or more of concussions do not involve a *loss of consciousness* (LOC) (McAlindon, 2002; Harmon, 1999). The FP must learn not only how to recognize them (which is not always easy) and become familiar with a system to grade them, but must also search for clues to more serious underlying injury, and finally determine if and when an athlete may return to play.

When approaching the fallen athlete with a suspected head injury, the FP should rapidly assess the ABCs and determine the level of consciousness as well as note any spontaneous movement and speech. Assessment for potential spine injury should be done, and once on the sidelines, a full neurologic examination performed, including a full sensory, motor, and cranial nerve examination as well as cognitive functioning and memory testing.

Obvious signs of skull fracture or intracerebral bleeding such as pupillary asymmetry, postauricular or periorbital ecchymosis, clear otorrhea, rhinorrhea, or hemotympanum, and any depression in the skull should be searched for. It must be emphasized that even if the initial examination is completely normal, frequent reassessment is mandatory as victims of head injury will often rapidly deteriorate and many of the above listed findings may not appear until later.

A concussion is by far the most common head injury in sports and is defined by the American Academy of Neurology (AAN) as “a trauma induced alteration in mental status that may or may not involve loss of consciousness” (Quality Standards Subcommittee, 1997). Several grading systems for concussions exist (Quality

Standards Subcommittee, 1997; Cantu, 1986; Colorado Medical Society School and Sports Medicine Committee, 1990) and cannot be adequately discussed in this chapter alone, but broadly speaking, the three most commonly used systems assess severity based on the presence or absence of an LOC and / or posttraumatic amnesia, as well as the duration of *post-concussive symptoms* (PCS).

Despite the multiple differences amongst the recognized guidelines, most authorities would agree with the following statements:

1. No athlete should return to play while *any* symptoms are still present either at rest or with exertion.

2. No athlete should return to play on the same day if the concussion involved an LOC (even if brief) or if postconcussive symptoms are still present 15–20 min after the injury.

3. An athlete with a mild concussion (Grade 1) with no LOC and resolution of PCS within 15–20 min both at rest and with provocative exertional maneuvers may safely return to play that same day, provided this was the first concussion.

4. Regardless of whether an athlete returns to play or is disqualified from play for that day, frequent reevaluation and serial examinations are *absolutely mandatory*.

Two specific head injuries deserve mention because of the rapidity with which they present and their associated morbidity and mortality.

Epidural hematoma

This most commonly results from a tear of the middle meningeal artery after high-velocity impact to the temporoparietal region and is associated with a skull fracture 80% of the time. Athletes will often experience a brief LOC followed by a lucid interval which may last up to several hours, and then progress to rapid neurologic deterioration and eventually coma and brainstem herniation. Treatment is surgical and immediate transfer to a medical facility is required.

Second impact syndrome

This is defined as a second head injury occurring before the symptoms of a first head injury have resolved.

A controversial topic, it is a catastrophic injury that may occur because of a loss of cerebral autoregulation caused by the initial injury (Harmon, 1999; Crump, 2001; Graber, 2001). When the second injury occurs, and it is often a very mild injury, cerebral edema rapidly develops with subsequent brainstem herniation within a matter of seconds to minutes. Treatment consists of immediate intubation and hyperventilation, administration of an osmotic diuretic (i.e., mannitol), and transport to a medical facility. Despite aggressive treatment, mortality and morbidity are around 50% and 100% respectively (Cantu, 1998; 1992).

NECK INJURY

Neck injuries, although relatively uncommon and usually self-limited (McAlindon, 2002), represent one of the most feared and potentially catastrophic injuries in sports. The FP must promptly recognize the potential for spine injury, adhere strictly to spinal precautions (discussed previously in this chapter), and finally determine whether an athlete requires immobilization and transfer to a medical facility, can return to play, or simply requires further sideline observation.

Indications for spinal immobilization include a post-traumatic LOC, subjective neck pain or bony tenderness on examination, significant neck / upper back trauma, significant head injury, mental status changes, neurologic abnormalities, or significant mechanism of injury (Luke and Micheli, 1999; McAlindon, 2002).

One of the more daunting tasks as an FP is distinguishing the minor from the more serious spinal injuries thus determining which athletes may safely return to play

after a neck injury. Usually minor, “burners” or “stingers” are nerve injuries resulting from trauma to the neck and/or shoulder that causes either a compressive or a traction injury to the 5th or 6th cervical nerve roots or the brachial plexus itself (Haight and Shiple, 2001; McAlindon, 2002; Kuhlman and McKeag, 1999). It consists of an immediate onset of burning pain radiating down the arm and is usually unilateral in distribution and often associated with other symptoms such as numbness, paresthesias, and muscle weakness or paresis. It is typically self-limiting with most cases resolving in a matter of minutes, although some symptoms may persist for weeks to months.

A “burner” should *not* be considered as an initial diagnosis if an athlete has any of the following:

a. Bilateral upper extremity involvement
b. Any lower extremity involvement
c. Neck pain or tenderness

Although there are no definitive guidelines as to which athletes with neck injuries are safe to return to play, it is generally agreed on that only those players with absolutely no neck pain or neurologic symptoms and with completely normal examinations may return to play safely, with repeated evaluation being absolutely necessary (Haight and Shiple, 2001; McAlindon, 2002).

OPHTHALMOLOGIC INJURY

Any injury to the eye warrants an immediate and thorough ocular examination, as seemingly minor injuries can be potentially vision-threatening. Examination of the eyes should include an assessment of visual acuity, visual fields, the eyelids and periorbital bony structures, the surface of the globe (conjunctiva, sclera, cornea), the pupils (size, shape, reactivity), extraocular movements, and fundoscopic examination

and possibly intraocular pressure measurement as indicated (Cuculino and DiMarco, 2002).

Potential injuries to the eye and/or ocular structures include (Cuculino and DiMarco, 2002) the following:

Eyelid lacerations

Any lacerations involving the lid margin or lacrimal system or those with significant tissue loss should be repaired by an ophthalmologist.

Corneal abrasion

A superficial defect in the cornea presenting with pain, photophobia, tearing, and a foreign body sensa tion. Diagnosis is by fluoroscein examination and treatment consists of topical antibiotics, analgesia, and tetanus prophylaxis.

Corneal foreign body

The presentation is similar to corneal abrasion, and corneal perforation must be ruled out if there is a his tory of high-velocity objects involved. Removal can usually be accomplished with slit-lamp assistance under topical anesthesia.

Corneal laceration

Many of these are self-sealing and difficult to visualize, thereby requiring a high index of suspicion. Examination may show a teardrop pupil, hyphema, or flat anterior chamber. The eye should be covered with a hard shield and the athlete told not to move the eye. Intraocular pressures should *not* be measured and immediate ophthalmology consult is required.

Hyphema

Blood within the anterior chamber, usually owing to trauma, although atraumatic hyphemas may occur in the presence of coagulopathies. Presenting symptoms include decreased vision, pain, and a history of trauma. The size of the hyphema should be noted, the eye shielded, and immediate ophthalmology consult obtained.

Intraocular foreign body

Presenting symptoms include pain, irritation, and injection, and suspicion should be based on a history of any high-velocity projectile or metal striking metal. Fluoroscein staining may reveal a positive Seidel sign, a washing away and streaking of fluoroscein as aque ous humor leaks out of the globe. The eye should be shielded, intraocular pressure measurements avoided, and ophthalmology consultation obtained.

Globe rupture

Usually occurs from direct blunt trauma to the eye because of a sudden increase in intraocular pressure. Examination may reveal a total subconjunctival hem orrhage, enophthalmos, teardrop pupil, or a flat ante rior chamber. Treatment is the same as that of an intraocular foreign body or corneal laceration.

Retrobulbar hemorrhage

Usually occurs after trauma and presents with acute proptosis, pain, swelling, and limitation of *extraocular muscle* (EOM) movement. It is essentially an “orbital compartment syndrome” and irreversible vision loss can occur within 1 h. Immediate referral to an ophthalmologist is required.

Orbital rim fracture

Usually a result of blunt trauma with examination revealing periorbital bony tenderness, crepitus, or paresthesias in the distribution of the infraorbital nerve, as well as limitation of EOM movement if there is entrapment. Athletes should be sent for radi- ographic evaluation, with treatment depending on the extent of injury.

NASAL INJURY

The field-side care of problematic nasal injuries generally involves identification of nasal fractures, control of epistaxis, or treatment of septal hematomas.

Isolated nasal fractures are usually not corrected acutely unless associated with significant deformity or other soft tissue injury. Treatment includes ice, analgesics, nasal decongestants, and avoidance of further injury.

Given that 95% of nosebleeds are anterior in origin, most can be controlled with either direct pressure and, if necessary, cauterization of an identified bleeding site or packing with a nasal tampon.

A potential complication of nasal injuries that must be carefully looked for is a septal hematoma, which is a red-blue, bulging mass on the nasal septum. These should be drained promptly by incision or aspiration followed by packing to prevent reaccumulation, as avascular necrosis and / or an abscess of the nasal septum may develop within a few days if left untreated.

EAR INJURY

An auricular hematoma is a subperichondral accumulation of blood following blunt trauma. If large enough and left untreated it can cause avascular necrosis as well as asymmetrical regrowth of new cartilage with a resultant cosmetic deformity of the ear known as a *cauliflower ear*. Treatment involves drainage of the hematoma followed by a pressure dressing to prevent reaccumulation.

Tympanic membrane perforation, although not an acute emergency, must be recognized so that proper follow up care is obtained to ensure proper healing and avoidance of hearing loss. Most will be caused by either blunt or noise induced trauma and greater than 90% will heal spontaneously. Antibiotics (either systemic or topical) are typically not necessary for uncomplicated perforations. Those that are caused by penetrating trauma should be promptly referred to an otolaryngologist.

ABDOMINAL / PELVIC INJURY

Although potentially serious and even life-threatening, most abdominal injuries can be managed nonoperatively with close observation. These injuries generally result from either rapid deceleration, direct blunt trauma to the abdomen, or indirect trauma from a displaced lower rib fracture (Amaral, 1997).

Injuries to the abdominal wall include simple contusions and rectus sheath hematomas, both of which are benign and usually managed conservatively, although the latter can occasionally require surgical intervention. The importance of these injuries to the FP lies in excluding associated intra-abdominal injuries, with mechanism of injury being perhaps the most important clue since a single field-side

abdominal examination, even if benign, is often misleading and inadequate in excluding significant intra-abdominal injury (Amaral, 1997).

SPLENIC / HEPATIC INJURY

The spleen and liver comprise the two most common organs injured in blunt abdominal trauma. There may be left or right upper quadrant and/or shoulder pain respectively, as well as signs of hypotension if bleeding is significant. All athletes with significant pain and/or appropriate mechanism of injury should be sent for *computed tomography imaging* (CT scan) and/or observation.

GENITOURINARY INJURY

Injuries to the renal system seldom require immediate intervention and suspicion should be based on the mechanism of injury as well as the presence and degree of hematuria (Amaral, 1997). One must keep in mind that injury to the kidney may be present with out hematuria and that hematuria does not always sig nify significant renal injury. In terms of evaluating hematuria, usually only those athletes with gross hematuria or with persistent microscopic hematuria accompanied by hypotension or associated nonrenal injuries require radiographic evaluation of the genitourinary system (Amaral, 1997).

URETHRAL/GENITAL INJURY

Gross blood at the urethral meatus, a scrotal or perineal hematoma, and an absent or high-riding prostate on rectal examination are all signs of urethral trauma and require consideration of a pelvic CT with contrast to look for bladder or urethral extravasation or hematomas followed by a retrograde urethrogram. Blunt trauma to the scrotal area may result in displacement of the testicle into the perineum or inguinal canal or may rupture the testicular capsule, both of which may require surgical intervention. Examination is often difficult because of pain and swelling; however, severe scrotal or testicular swelling or a nonpalpable testicle warrants further evaluation. In the absence of direct trauma, testicular torsion must be ruled out in the athlete presenting with acute onset of testicular pain. In either case, color flow doppler ultrasound studies may define the nature or extent of the problem.

MUSCULOSKELETAL INJURY

Musculoskeletal injuries are the most commonly encountered injuries in sports. Most are minor and self-limited and it is certainly beyond the scope of this chapter to discuss various specific fractures; however, a few general statements about fracture care can be made and a handful of limb-threatening injuries discussed.

In terms of fracture care, the FP must always ascertain the mechanism of injury and never assume that the obvious deformity is the only injury. Always check the neurovascular status of the affected body part distal to the fracture site. If there is vascular compromise, reduction of dislocations and / or fractures should be attempted in the field with gentle traction. Otherwise, fractures should be splinted in the

position in which they are found, unless some degree of reduction is required because of neurovascular compromise. Finally, no athlete should return to play if there is a question of a fracture, no matter how minor the injury may seem, as this may transform a nondis-placed or a closed fracture into a displaced or open one.

The following injuries represent a potential threat to a limb:

Open fracture

Previously known as a compound fracture, this is a fracture associated with overlying soft tissue injury with communication between the fracture site and the skin. These are at high risk for subsequent infection and osteomyelitis and require washout in the operat ing room. On the field, the open wound should be covered with moist sterile gauze and the extremity splinted with no attempts made to push extruding bone or soft tissue back into the wound or reduce the fracture, unless neurovascular compromise is present.

Traumatic amputations

This is a very rare and dramatic injury which is easy to recognize. The proximal stump should be irrigated with a sterile solution and a sterile pressure dressing applied, with a tourniquet used only for severe, uncontrolled bleeding. The amputated portion should be irrigated, wrapped in a sterile fashion, placed in a bag, and put on ice with rapid transport to an appropriate medical facility.

COMPARTMENT SYNDROME

This is a state of increased pressure within a closed tissue compartment that compromises blood flow through nutrient capillaries supplying muscles and nerves within that compartment. The potential causes of compartment syndrome are numerous, although in terms of athletes, this is typically an injury with the most

common site being the anterior compartment of the leg. Presentation typically occurs within a few hours after injury and will consist of severe and constant pain over the involved compartment, with an increase in pain with both active contraction and passive stretching of the involved muscles. There may also be significant dysesthesias as well as an absent or diminished pulse, pallor, and/or paralysis of the affected neuromuscular group, although these are considered to be late findings which indicate that significant myoneural ischemia has already occurred. Treatment is an emergent fasciotomy and requires rapid transport of the athlete to a medical facility.

KNEE DISLOCATION

Although extremely rare and usually associated with a high-velocity/high-energy mechanism of injury, this is a very serious injury which may require a high index of suspicion as many dislocations will have spontaneously reduced prior to evaluation. The knee will typically be very swollen and painful and will often demonstrate severe instability in multiple directions on examination. The seriousness of the injury lies in the high rate of associated complications, specifically popliteal artery injury and peroneal nerve injury (which may occur despite spontaneous reduction and normal pulses). Early reduction of a visible dislocation is important. Rapid transport of the patient with a known or suspected dislocation to a medical facility for orthopedic and/or vascular consultation is essential.

HIP DISLOCATION

Like the knee, this dislocation is rare in sports and usually involves a high-velocity/high-energy mechanism of injury. Posterior dislocations are by far the most common type, and the seriousness of this injury lies in the risk for *avascular necrosis* (AVN) of the femoral head as circulation is disrupted. This occurs in a matter of hours with 6 h being the danger zone—as approximately 60% of reductions beyond 6

h develop AVN, while only 5% of reductions occurring under 6 h develop this complication (Scopp and Moorman, 2002).

ENVIRONMENTAL INJURY

Hypotermia

Defined as core body temperature <95°F, this usually occurs as a result of prolonged exposure to cold environmental conditions. When approaching the hypothermic athlete, the FP must keep the following points in mind:

1. Treatment should routinely start with passive external rewarming (i.e., moving the athlete from a cold to a warm environment, removing all wet clothing, and covering with dry blankets). Active external rewarming and core rewarming should usually be deferred until the hospital environment because patients with moderate to severe hypothermia are at high risk of having significant electrolyte, acid-base, and cardiovascular changes associated with rewarming.

2. Significantly hypothermic patients are at very high risk of fatal cardiac arrhythmias and should be moved and handled very gently to avoid triggering ventricular fibrillation (Jacobsen et al, 1997).

3. Pulses are often difficult to detect in significantly hypothermic patients, so CPR should not be started prematurely as it may actually trigger a cardiac dysrhythmia. And if CPR is started, it should continue until warming has been completed; “they’re not dead until they’re warm and dead.”

Hypertermia

Heat related illnesses represent a spectrum of disease ranging from heat cramps and edema all the way to heat stroke and death. Heat stroke is a true medical

emergency with high mortality rates if unrecognized. It typically presents in warm, humid conditions with elements of overexertion and dehydration on the part of the athlete. Signs of dehydration (tachycardia, hypotension, and oliguria) are often present, as well as a temperature >105°F and prominent *central nervous system* (CNS) and autoregulatory changes. The FP must keep the following in mind when approaching the hyperthermic athlete:

a. Active and passive cooling measures (i.e., removing from the heat, removing clothing, placing ice packs around the groin, neck, and axillae) should be instituted immediately with the goal of therapy being to lower the core temperature to ≤102°F as quickly as possible (Jacobsen et al, 1997).

b. Intravenous fluids should be started early; how ever, caution must be used as overaggressive rehydration may put the victim at an increased risk of pulmonary edema and *adult respiratory distress syndrome* (ARDS). All victims of heatstroke should be transported to a medical facility for further care.

LIGHTNING INJURY

Although rare, lightning injury is one of the more frequent injuries by a natural phenomenon with the largest number of sports injuries occurring in water sports and most injuries occurring during the months of June–September (Jacobsen et al, 1997). Although it is by definition an *electrical injury*, it differs significantly from high-voltage electrical injuries in both the pattern and severity of injuries as well as the immediate treatment. Although the voltage of lightning is extraordinarily high, it is usually an instantaneous contact that tends to flash over the outside of a victim’s body, often creating superficial burns, but sparing extensive damage to internal organs and structures. Lightning may injure a person by striking either the person directly or something they are holding, or by splashing over from a nearby person or object that has been struck. It may also strike the ground and spread circumferentially, often creating multiple victims. Although it can potentially affect any organ system, injuries to the cardiovascular and neurologic systems tend to be the

most common, with the immediate cause of death most commonly being cardiopulmonary arrest (Jacobsen et al, 1997). Minor injuries include dysesthesias, minor burns, temporary LOC, confusion, amnesia, tympanic membrane perforation, and ocular injury. More serious injuries usually result from sequelae of the blunt trauma of the electrical blast and from cardiac arrest. The FP should keep the following points in mind when approaching a victim of lightning injury:

1. Standard ACLS protocols should be followed.
2. Victims do not “retain charge” and are not dangerous to touch, so CPR should not be delayed for this reason.
3. Contrary to popular belief, lightning can and often does strike the same place twice, so personal safety must be taken into consideration.
4. Hypotension in a lightning victim should prompt a search for occult hemorrhage or fractures as a result of blunt trauma. Spinal precautions are required.
5. Pupils may become “fixed and dilated” because of the nature of lightning injuries and this should not preclude resuscitation attempts as these changes do not necessarily indicate brain death in lightning victims.
6. In lightning victims with cardiopulmonary arrest, cardiac automaticity and contractions will often resume spontaneously and in a short period of time, while respiratory arrest from paralysis of the medullary respiratory center may be prolonged. Therefore, unless the victim is ventilated quickly they will progress to a secondary hypoxic cardiac arrest despite normal cardiac activity. If promptly resuscitated and supported, full recovery may ensue.
7. In consideration of the previous two points, in a multicasualty situation from a lightning strike, the FP should always resuscitate the dead first, a reversal of the standard rule of triage where the obvious moribund are left to the last.

References

1. Amaral JF: Thoracoabdominal injuries in the athlete. Clin Sports Med 16(4):739–753, 1997.

2. Blue JG, Pecci MA: The collapsed athlete. Orthop Clin North Am 33(3):471–478, 2002.

3. Cantu RC: Guidelines for return to contact sports after a cerebral concussion. Phys Sportsmed 14(10):75, 76, 79, 83, 1986.

4. Cantu RC: Second-impact syndrome. Clin Sports Med 1: 37–44, 1998.

5. Cantu RC: Second impact syndrome: Immediate management. Phys Sportsmed 20(9):55–66, 1992.

6. Colorado Medical Society School and Sports Medicine Committee: Guidelines for the management of concussion in sports. Colo Med, 87:4, 1990.

7. Committee on Trauma: Advanced Trauma Life Support for Doctors: Student Course Manual. American College of Surgeons, 1997.

8. Crump WJ: Managing adolescent sports head injuries: A case-based report. Fam Prac Recert 23(4):27–32, 2001.

9. Cuculino GP, DiMarco CJ: Common ophthalmologic emergencies: A systematic approach to evaluation and management. Em Med Rep 23(13):163–178, 2002.

10. Fowler R, Pepe PE: Prehospital care of the patient with major trauma. Emerg Med Clin North Am 20(4):953–974, 2002.

11. Gastel JA, Palumbo MA, Hulstyn MJ, et al: Emergency removal of football equipment: a cadaveric cervical spine injury model. Ann Emerg Med 32(4):411–417, 1998.

12. Graber M: Minor head trauma in children and athletes. Emerg Med 14, 17, 18, 20, Oct. 2001.

13. Haight RR, Shiple BJ: Sideline evaluation of neck pain. Phys Sportsmed 29(3):45–62, 2001.

14. Harmon KG: Assessment and management of concussion in sports. Am

Fam Physician 60(3):887–892, 1999.

15. Jacobsen TD, Krenzelok EP, Shicker L, et al: Environmental injuries. Dis Mon 814–912, 1997.

16. Kuhlman GS, McKeag DB: The “burner”: A common nerve injury in contact sports. Am Fam Physician 60(7):2035–2040, 1999.

17. Luke A, Micheli L: Sports injuries: Emergency assessment and field-side care. Pediatr Rev 20(9):291–302, 1999.

18. McAlindon RJ: On field evaluation and management of head and neck injured athletes. Clin Sports Med 21(1):1–14, 2002.

19. McCaffrey FM, Braden DS, Strong WB. Sudden cardiac death in young athletes. Am J Dis Child 145:177–83, 1991.

20. O’Connor FG, Kugler JP, Oriscello RG: Sudden death in young athletes: Screening for the needle in a haystack. Am Fam Physician 57(11):2763–2770, 1998.

21. Partridge RA, Coley A, Bowie R, et al: Sports-related pneumoth-orax. Ann Emerg Med 30(4):539–541, 1997.

22. Quality Standards Subcommittee: Practice parameter: The management of concussion in sports (summary statement). Neurology 48:581–585, 1997.

23. Scopp JM, Moorman CT: Acute athletic trauma to the hip and pelvis. Orthop Clin North Am 33(3):555–563, 2002.

24. Winbery SL, Lieberman PL: Anaphylaxis. Immunol Allergy Clin North Am 15(3):447–475, 1995.

SUDDEN DEATH IN ATHLETES

Death is a natural and inevitable event; however, when death occurs suddenly and unexpectedly in a trained athlete who personifies health, strength, and invulnerability, it cannot be accepted at all. The sudden death of an athlete in training or competition is an infrequent but devastating event to all involved (patient, family, friends, team, and caregivers) and to the public as a whole. The mass media's interest in these tragic events, especially when young or famous athletes are involved, serves to increase their impact on public perception.

Over the past 2 decades, a great deal of medical literature has been generated on the causes and mechanisms of sport-related sudden death and on screening strategies and disqualification criteria for competitive athletes. Nevertheless, such tragedies continue to occur: while this article was being written, Mohamed Abdelwahab, a 22-year-old member of the Egyptian national team that won the previous year's African Cup, collapsed and died during a training session.

Sudden death is defined as a witnessed or unwitnessed natural death occur-ring unexpectedly within 6 hours of a previously normal state of health [1]. Although nontraumatic athletic-field deaths may be attributed to noncardiac causes (cerebral aneurysm, heat stroke, sickle cell trait, bronchial asthma, drug abuse), more than 90% of these events occur in subjects who have pre-existing and usually clinically silent cardiac abnormalities [2]. For this reason, sport-related sudden death should be distinguished in sudden cardiac death (SCD) and in death due to noncardiac causes.

SCD is defined as nontraumatic, nonviolent, unexpected natural death of cardiac origin occurring within 1 hour of the onset of symptoms in a person who does not have a previously recognized cardiovascular condition that would appear fatal [1]. According to this definition, SCD may occur during or immediately after the exertions involved in competition or training.

EPIDEMIOLOGY

The extensive media attention that greets cases of sudden death in athletes might give the impression that these events occur frequently; however, the overall incidence of SCD during athletic activity is low.

There have been few medical studies investigating the prevalence of SCD in athletes. Among Minnesota high school athletes, the annual incidence of SCD has been reported to be about 1 in 200,000 [3]. More recently, Corrado and colleagues [4] reported an incidence of 2.3 in 100,000 young athletes per year in the Veneto region of Italy, about twice the incidence previously reported by Maron and colleagues [3].

The risk of SCD in athletes, though low, significantly increases with age. Available data suggest that prevalence of SCD among athletes older than 35 years ranges from 1 in 15,000 joggers [5] to 1 in 50,000 marathon runners [6]. SCD appears to be much more common in male than in female athletes (with the male-to-female ratio as high as 10:1) [7]. To explain this sex-related discrepancy in death rate, various speculative explanations have been suggested: women have a lower participation level and training intensity, a lower incidence of structural cardiovascular disease (ie, hypertrophic cardio-myopathy [HCM]), and the tendency compared with men to not deny or ignore prodromal symptoms [8,9].

Nevertheless, Corrado and colleagues [10] reported a similar risk of SCD in adolescents and young athletes of both sexes in a 21-year prospective cohort study of the Veneto region of Italy. It is remarkable that in this study, the relative risk of SCD in athletes was twice the risk of nonathletes. The investi-gators interpreted their results by concluding that sport itself is not a cause of the enhanced mortality but rather a potential trigger of sudden death in athletes affected by a predisposing cardiovascular condition.

CAUSES OF SUDDEN CARDIAC DEATH IN ATHLETES

A broad spectrum of congenital and acquired cardiac diseases has been identified as causing SCD in athletes, although a relatively small number of diseases are responsible for most cases.

The most significant conclusion that can be drawn from the medical literature on this subject is that the specific causes of athletic-field deaths differ considerably with regard to an athlete's age [1]. By differentiating according to the causes of SCD, two groups of athletes are generally identified: the ''young'' athletes (<35 years) and the ''older'' athletes (>35 years).

THE YOUNG ATHLETE (<35 YEARS)

Causes of Sudden Cardiac Death and Demographic Features

Autopsy-based studies of young athletes who died suddenly demonstrated that in most cases (95% or more), a structural cardiac abnormality is detectable [1,9,10]. The most frequent abnormalities identified were inheritable cardiomy-opathies and congenital coronary artery anomalies (CCAAs), followed by a number of diseases including myocarditis, Marfan syndrome, valvular heart diseases, dilated cardiomyopathy (DCM), premature coronary artery disease (CAD), and myocardial bridge [2,9]. Further, in a small but significant group of young athletes who died suddenly (2%–5% of all cases), no structural cardiac abnormalities could be detected, even after gross and microscopic examination of the heart [2,9,10]. Most of these cases, once unexplained, are probably due to ionic-channel disorders such as the long QT syndrome (LQTS), cathechola-minergic polymorphic ventricular tachycardia (CPVT), or Brugada syndrome [11], although it may not be possible to exclude with certainty noncardiac factors such as drug abuse.

In United States series, the single most common cause of SCD in young

athletes is HCM, especially among African American athletes, accounting for more than one third of cases. This proportion would further increase if an increased left ventricular (LV) mass consistent with, but not diagnostic of, HCM were considered a variant morphologic expression of HCM [2,12]. The second most frequent cause of death reported by different investi-gators are CCAAs, particularly anomalous origin of the left main coronary artery from the right sinus of Valsalva [1,2,10].

Important demographic differences in the causes of SCD in young athletes emerge when comparing the American and the Italian series. HCM accounted for only a small proportion (about 2%) of SCD in young athletes in the Veneto re-gion in northeastern Italy over a long period of observation. In this region, ar-rhythmogenic right ventricular cardiomyopathy (ARVC) accounted for most SCD (22%), followed by premature CAD (18%) and CCAAs (12%) [10]. This demographic difference has been related to a possible genetic predisposition of Ital-ian people to ARVC [13]; however, the efficacy of the unique Italian preparticipa-tion screening national program, including 12-lead ECG, which identifies and disqualifies athletes who have HCM much more readily than those who have ARVC or CCAAs [10,14], appears to be the most reliable explanation.

SCD in young athletes may occur in every kind of sports activity, although it has been reported most commonly among subjects engaged in organized team sports, particularly basketball and ‘‘American football’’ players in the United States [2] and soccer players in Europe [10,15]. These data, rather than showing an increased risk associated with these specific activities, more likely reflect the high level of participation in these sports on the two different continents. More-over, athletic-field deaths have been reported to occur more frequently in the late afternoon and early evening, corresponding largely to the time of training and competition [2].

Although most of the young athletes who died suddenly had never experi-enced cardiovascular symptoms before exitus, prodromal complaints (chest pain, palpitation, exertional dyspnea, dizziness, weakness) have been described in some cases [2,10,16].

Specific Causes of Sudden Cardiac Death

Hypertrophic cardiomyopathy

HCM is the most common genetic cardiac disease, with a prevalence of 0.2% (1/500) in the general population [17], and represents the most common cause of SCD among young athletes in the United States [2,12]. A large amount of information about clinical, pathologic, pathophysiologic, and genetic aspects of this disease is present in the medical literature; however, the authors include only the information that would be useful for the present discussion.

HCM is inherited as an autosomal dominant trait with a variable expression. Although ‘‘de novo’’ mutations are possible [18], most people who have HCM have at least one first-degree relative affected. HCM is characterized by a wide genetic heterogeneity: a number of different mutations in genes encoding for the myocardial sarcomere proteins have been identified as being responsible for the HCM phenotype [11].

Clinically, HCM is defined as a primary disease of cardiac muscle that usu-ally is genetically transmitted and is characterized by a hypertrophied but non-dilated left ventricle, in the absence of another cardiac or systemic disease that may produce LV hypertrophy [1,17].

Although different patterns of hypertrophy have been described [19], the most common one involves the interventricular septum, which results in it being disproportionately thicker (>15 mm to 30 mm or more) than most of the LV free walls [20].

The main microscopic features are a bizarre arrangement of muscle cells with diffuse interstitial fibrosis referred to as ‘‘myocardial disarray,’’ and changes in intramural coronary vessels that show thickened walls and narrowed lumen [1].

Patients who have clear HCM are symptomatic and functionally limited, whereas young affected subjects may be completely free of symptoms [17]. Most sudden death cases occur in the latter subset of patients [21]. As reported by Maron

and colleagues [22], a clinical profile of 78 patients who died suddenly of HCM before the age of 30 years showed that 55% had never ex-perienced functional limitation and about 40% were used to training vigorously before death. Maron and colleagues [2] also reported that of 134 young athletes who died suddenly, just 10 (21%) of the 48 who collapsed due to HCM had experienced cardiovascular symptoms such as exertional dyspnea, syncope, dizziness, and chest pain.

More than 90% of patients who have HCM show ECG abnormalities [23] including

- Left axial deviation
- Diffuse, symmetric, and marked T-wave inversion
- ST-segment flattening, depression, or both
- Increased R or S voltages in precordial leads
- Deep Q wave.

Two-dimensional echocardiography is the most reliable diagnostic tool for HCM, except in prepubertal subjects in whom false-negative results may occur because the phenotypic expression of HCM may not be evident or complete until pubertal spurt [23].

At echocardiographic examination, a small subset of highly trained athletes may present a marked cardiac hypertrophy with the anterior ventricular septum ranging from 13 to 15 mm [24,25]. In athletes who fall in a morphologic ‘‘gray zone,’’ it may be difficult to distinguish a mild expression of HCM from the physiologic, training-induced LV hypertrophy (‘‘athlete’s heart’’). Obvi-ously, this differential diagnosis has crucial medical and medicolegal implica-tions because the identification of the disease would lead to disqualification of the athlete from training and competition to minimize his or her risk of SCD. Clinical, ECG, and echocardiographic criteria for the differential diagnosis between HCM and athlete’s heart when a morphologic gray zone is observed have been assessed by Maron and colleagues [26]:

1. Pattern of LV hypertrophy: typically symmetric with similar thickness of all segments in athlete's heart; in contrast, asymmetry is the hallmark of HCM

2. LV internal cavity dimension: in athlete's heart, thickening of free walls is always associated with an enlargement of end-diastolic cavity dimensions (>55 mm), whereas HCM is characterized by a hypertrophied but nondi-lated left ventricle (<45 mm)

3. Left atrial size: the left atrium is typically dilated in HCM but usually not in ath-lete's heart (<40 mm), even though in highly trained athletes who have the most marked LV hypertrophy, left atrial size greater than 40 mm has been ob-served, including a small subset (2%) of athletes who have a left atrium greater than 45 mm. Nevertheless, in these cases, the other differential criteria gener-ally permit assessment of the final diagnosis with certainty [27]

4. LV diastolic filling: typically abnormal in HCM patients due to the reduced compliance of the left ventricle as a result of the diffuse interstitial fibrosis; in contrast, typically normal in athlete's heart

5. Sex: female athletes rarely show LV wall thickness of 12 mm or greater [28]; therefore, female athletes who have such echocardiographic measures are most likely to be affected by HCM

6. Effect of detraining: short-term (3 months) deconditioning is generally suffi-cient to induce a regression of physiologic cardiac hypertrophy of trained athletes, whereas it has no effect on pathologic hypertrophy [29,30]

7. Family history and genetics: HCM is a genetic disease inherited as an auto-somal dominant trait. Therefore, most patients have at least one first-degree relative affected so that family screening, when other criteria are not conclu-sive, could be determinant for the diagnosis. If certain diagnosis is not possible but the suspect of disease is high, the most definitive evidence for the presence of HCM comes from DNA analysis, although the genetic het-erogeneity of HCM and the expensiveness of the required methodologies limit this diagnostic tool to a very few cases [31]

Arrhythmogenic right ventricular cardiomyopathy

ARVC is a very rare cause of SCD in young athletes in North America [2,12], whereas it has been reported to be the most common cause among young Italian athletes of northern Italy [4,10] where mandatory preparticipation screening for competitive athletes has significantly reduced other causes of death in the athletic population, in particular HCM [14].

ARVC is a genetic cardiac disease inherited as an autosomal dominant trait with incomplete penetrance [11]. Mutations in genes encoding desmosomal proteins have been identified as the major cause of the disease [32,33]. The overall prevalence of ARVC has not been well defined but is estimated to be between 1 in 1000 and 1 in 10,000 [21]. ARVC is defined as a primary disorder of the myocardium (a ‘‘cardiomyopathy’’) whose main pathologic features are a progressive diffuse or segmental myocyte atrophy accompanied by fatty or fibrofatty tissue replacement that typically involves the right ventricle free wall, with only occasional additional involvement of the left ventricle. This pathologic process results in thinning and dilatation of the right ventricle with consequent dysfunction and creates a highly arrhythmogenic substrate [34,35]. Therefore, the affected patients have a high risk of SCD due to a fatal ventricular arrhythmia that is not only related to [36] but may also be precip-itated by intense physical effort [37]. It is worth noting that in the series by Cor-rado and colleagues [10], ARVC and CCAA were the only cardiovascular conditions associated with sudden death significantly more often in athletes than in nonathletes.

Early diagnosis of ARVC is usually difficult and is rare until the patient develops symptoms. Indeed, the initial presentation of ARVC is syncope in almost 30% of patients, due to monomorphic ventricular tachycardia with left bundle-branch block morphology [21,34,38]. In young athletes, as in the general population, suspicion of ARVC may arise from the rest 12-lead ECG that, in most patients (50%–90%), shows a broad spectrum of abnormal-ities. The most common abnormalities include

- T-wave inversion in the right precordial leads (V_1 through V_3) in subjects older than12 years associated with slight T-segment elevation (<0.1 mV)
- Complete or incomplete right bundle-branch block
- Prolongation of QRS duration in the right precordial leads

Epsilon wave in leads V_1 through V_3 has small amplitude potentials occurring after the QRS complex at the beginning of the ST segment. This finding is un-common on standard ECG but may be revealed in about 30% of patients with high-resolution ECG and signal averaging techniques.

Frequent premature ventricular beats with left bundle-branch block morphol-ogy may be present; more frequently, ventricular arrhythmias (isolated or re-petitive premature ventricular contraction, sustained or nonsustained ventricular tachycardia) are found on 24-hour ECG monitoring in symptom-atic and asymptomatic patients [34,38].

Recently, a small subset of ARVC patients showing a Brugada-like ECG pattern with complete right bundle-branch block and right precordial ST-segment elevation has been described. In contrast with the typical form of ARVC, these patients show higher risk of sudden death at rest than during effort, as do those affected by Brugada syndrome [35].

In ARVC patients, echocardiography may demonstrate a dilated, poorly contractile right ventricle with thin walls [34,39]. MRI provides noninvasive localization of structural changes and regional dysfunction, although identifica-tion of intramyocardial fibrofatty infiltration is difficult [34,40]. Although en-domyocardial biopsy may reveal the hystopathologic feature of disease, it lacks sufficient sensitivity because of the segmental nature of disease process and because the samples are usually taken from the septum for safety reasons, a region uncommonly involved by the disease [34]. Although recognized as a familial disease [41], to date, no specific genetic testing for ARVC is available [42].

In 1994, the study group on ARVC of the European Society of Cardiology and

the International Society and Federation of Cardiology implemented a scoring system for the diagnosis of ARVC based on major and minor criteria [37]. Because SCD is frequently the first manifestation of the disease, the diag-nosis of ARVC is only made post mortem. In the series reported by Corrado and colleagues [10], of the 49 young Italian athletes who died suddenly on the athletic field over a period of 20 years, 11 (22.4%) died as a result of ARVC. All these athletes had undergone the Italian national preparticipation screening, which demonstrated family history of premature SCD in 2 cases, exertional palpitation in 6, history of syncope in 5, ECG abnormalities in 9, and ventric-ular arrhythmias in 6, with 9 athletes presenting one or more associated find-ings. In 1998, ARVC was a still poorly recognized disease. Data presented by Corrado and colleagues [10] raised awareness of this disease among cardiolo-gists and sports physicians and taught that the finding at preparticipation screening of even isolated premature ventricular contraction with left bun-dle-branch block morphology associated with T-wave inversion in right pre-cordial leads (eventually associated with other ECG abnormalities), with or without family history of premature SCD or personal history of exertional pal-pitation or syncope, should raise the suspicion of ARVC and lead to further diagnostic tests.

Congenital coronary artery anomalies

In most published series, CCAAs are a relatively frequent cause of SCD in young athletes (about 20% of all cases) [2,9,10] and the single most common cause of SCD in female athletes [2,9]. In particular, the anomalous origin of the left main coronary artery from the right (anterior) sinus of Valsalva, followed by its mirror image (ie, the right coronary artery arising from the left sinus of Valsalva) [43], represent the most common abnormalities found at autopsy [16,44].

Congenital anomalies of coronary arteries, in all their variations, are overall an infrequent condition. Previous studies suggest an overall prevalence of 0.1% to 0.2% in the general population [45,46], which is similar to the prevalence es-timated in athletes. In an echocardiographic study performed on 1360 compet-itive athletes, no

CCAAs were found, suggesting a prevalence of less than 0.1% in the athletic population [47]. Correct and timely diagnosis of CCAAs is extremely important for prevention of SCD because surgical correction is feasible. Nevertheless, such coronary anomalies are rarely identified in living persons because of insufficient clinical suspicion and the poor sensitivity of first-level cardiovascular testing. Rest ECG is generally normal, as is the exer-cise ECG [16], because the myocardial ischemia occurring in CCAA is typically episodic. Thus, careful attention should be paid to referred symptoms, with particular regard to exercise-induced chest discomfort/pain or syncope, even though these symptoms have been reported in less than 30% of the young athletes who suddenly collapsed due to CCAA [10,16].

When clinical suspicion of CCAAs arises, diagnosis may be confirmed with echocardiography. Using this noninvasive and relatively low-cost method, a good anatomic definition of the ostium and proximal epicardial course of coronary arteries can be ascertained in most cases. Indeed, among the 1360 athletes studied by Pelliccia and colleagues [47], the left main artery was visualized in 97% of subjects and the right coronary in 80%. In cases of poor acoustic windows or difficulty interpreting images at the transthoracic examina-tion, diagnosis of CCAAs may better be achieved by transesophageal echocar-diography, MRI [48], or coronary angiography [45].

Other Causes of Sudden Cardiac Death in Young Athletes

A number of other pathologic entities have been identified as causes of SCD in young athletes. Each of them is usually considered a minor cause, accounting for 5% or less of athletic-field deaths [2,9,10,12]. Taken together, however, they cover about 40% of all cases.

Aortic rupture and Marfan syndrome

Abrupt aortic rupture with pericardial tamponade is an infrequent cause of

SCD in young athletes in the United States and European series [2,10,12]. Most of these cases occur in patients who have Marfan syndrome, a genetic disorder of connective tissue caused by mutations in the fibrillin-1 gene, with a sex-neutral prevalence of about 1 in 10,000 in the general population [49].ZO

Marfan syndrome is characterized by multisystemic involvement, including musculoskeletal (scoliosis and pectus excavatum) and ocular abnormalities (dislocated lens and myopia). Tall stature, arachnodactyly, and hyperextensible joints represent other characteristic features. Nevertheless, the cardiovascular manifestations that occur in most patients, particularly mitral valve prolapse (MVP) and severe aortic dilatation, may put young athletes at risk of life-threat-ening events. Indeed, cardiovascular complications are the cause of death in more than 80% of patients [50,51].

Many of us remember the tragic death of Flo Hyman, a 31-year-old member of the US Olympic volleyball team who collapsed during a game in Japan in 1984. Her autopsy revealed that she was affected by Marfan syndrome and that her cause of death had been the rupture of an aortic aneurysm with peri-cardial tamponade.

Aortic rupture in Marfan syndrome patients is the consequence of an intrin-sic weakness of aortic media due to specific histologic alterations, including ap-optotic cell death, elastic fragmentation, and changes in smooth muscle cell orientation that characterize a pathologic lesion known as ‘‘cystic medial necro-sis’’ [52]. The cardiovascular complications of Marfan syndrome are most effec-tively detected by two-dimensional echocardiography. Aortic root enlargement, evaluated according the standardized criteria and after normalization of the ab-solute values for age and body size [53,54], is the most common finding and takes the characteristic form of an ‘‘onion bulb’’ malformation, with dilatation of the aortic annulus, sinuses of Valsalva, and proximal ascending aorta [55].

Vigorous physical activity and competitive sports are widely restricted in Mar-fan syndrome patients. The increase in heart rate, blood pressure, and LV con-tractility associated with strenuous exercise might increment the mechanical stress imposed on the weakened aorta, thus potentially triggering lethal compli-cations such

as aortic rupture or dissection [56,57], with the greatest risk occur-ring among patients who have an aortic dimension beyond 6.0 cm [58].

Therefore, identifying athletes who have Marfan syndrome should be a primary goal of preparticipation screening of all competitive athletes [59].

Because of their characteristic tall stature, subjects who have Marfan syndrome gravitate toward sports in which this anthropometric feature is an advantage, such as basketball or volleyball. However, because of the clinical heterogeneity of the disease, tall stature should be associated with only mild or no other clin-ical Marfan signs, as in the case of Flo Hyman [57]. Although it is not feasible, some investigators have proposed mandatory inclusion of echocardiography in the preparticipation screening protocols of certain sports [60]. Despite the pre-viously discussed clinical variability, suspicion of Marfan syndrome might, in most cases, arise from an accurate physical examination and careful family in-vestigation [61,62], and the echocardiographic study should be limited to the cases of well-grounded clinical suspicion.

Myocarditis

Myocarditis is an inflammatory process of the myocardium usually caused by an acute viral infection (most often Coxsackie B) [63], and less frequently by cocaine abuse [64]. Myocarditis is an infrequent cause of SCD in most athletic series (5%) [2,9,10], although it represented the most common cause of sport-related death among healthy, young US Air Force recruits (42%) over a 20-year period of observation [65]. A possible explanation for this unique finding is increased spread of infectious disease in crowed living conditions, which is con-sistent with the high incidence of asymptomatic viral myocarditis (documented by ECG changes or echocardiography) in barracks-residing recruits reported in a Finnish study [66].

The myocardium inflammation leads to contractile dysfunction and creates a highly arrhythmogenic substrate that in turn exposes patients to an increased risk of life-threatening ventricular arrhythmias [67].

In most cases, myocarditis remains asymptomatic, thus increasing the risk of sudden death that appears not to be related to the stage or clinical manifestation of disease. The suspect of myocarditis should arise when an athlete develops, particularly close to a systemic viral illness, fatigue, exercise intolerance, and palpitation associated with signs of heart failure at physical examination [8,68]. Diagnosis is confirmed by endomyocardial biopsy, although patchy involvement of disease reduces the overall sensitivity of this method. It is sig-nificant that focal myocarditis was identified at macroscopic autopsy examina-tion as the most frequent cause of SCD (35%) in a group of 79 young subjects who had apparently normal hearts [69].

Athletes who have active myocarditys should be withdrawn from training and rested for up to 6 months after clinical recovery before returning to sport [67]. This precaution is also necessary because healed myocarditis has arrhyth-mogenic potential [70].

Dilated cardiomyopathy

DCM is an uncommon cardiac disease that has a prevalence of 1 in 2500 in the general population [71]. It is a similarly infrequent cause of SCD in young athletes, responsible for 0% to 4% of all cases in most published series [2,9,10]. Of interest, in a French forensic series, DCM was identified as the cause of 11% of the exercise-related deaths in a group of 80 subjects. Those subjects, however, were not competitive athletes but poorly trained individuals who only occasionally participated in various sports activities [72].

DCM is defined as a primary disease of the myocardium, characterized by LV dilatation and impaired systolic function; the natural history includes heart failure, arrhythmias, and SCD [73]. Ventricular arrhythmia is the commonest cause of SCD in DCM, and resulting deaths account for most cases in patients who have less severe disease [21].

Athlete’s heart is physiologic exercise-induced cardiac hypertrophy and is

principally characterized by LV cavity enlargement and free-wall thickening. These changes are mild in most cases so that the differences between athletes and nonathletes are generally small, although statistically significant [24]. Nev-ertheless, suspicion of DCM may sometimes arise in highly trained endurance athletes who show markedly enlarged LV cavity [26]. In 1999, Pelliccia and colleagues [74] assessed the upper limit of physiologic LV enlargement in a large cohort of elite athletes of both sexes from 38 different sports, ranging in age from 13 to 59 years. LV end-diastolic cavity dimension varied markedly, from 38 mm to 66 mm in women (mean, 55 mm) and from 43 to 70 mm in men (mean, 55 mm). LV dimension was within the accepted normal limits (54 mm) in 55% of the athletes, whereas it exceeded this limit in the remain-ing 45%, including the 14% of athletes who showed markedly enlarged cavity (60 mm) consistent with DCM but who had global LV systolic function within normal limits and no regional wall-motion abnormalities. The major determinants of LV cavity dimension were male sex, high body surface area, and participation in certain endurance sports (such as rowing, cycling, and cross-country skiing).

Therefore, a morphologic gray zone has also been described between ath-lete's heart and DCM, raising the crucial issue of differential diagnosis. It should be noted that DCM may present with an enlarged LV cavity before clinical expression develops with onset of symptoms and hemodynamic deteri-oration, and such eventuality may not be excluded in athletes. The differential diagnosis, however, is generally easily assessed because the absence of LV systolic dysfunction in athlete's heart is usually sufficient to distinguish the physiologic enlargement induced by training versus that due to DCM. Other important elements indicating athlete's heart are the absence of familial history of DCM and absence of arrhythmias [26].

Mitral valve prolapse

MVP is likely the most common cardiac abnormality in the general population, but because it remains asymptomatic in many cases, the exact prevalence is not well

known but estimated to be between 1% and 3% [75]. Although it generally represents a mild, benign valvular abnormality, MVP has been associated with arrhythmias and sudden death in the general population and in athletes [10,76,77]. MVP is reported to be a cause of SCD in young athletes of different series, although with highly variable rates ranging from 0.7% to 10% [2,9,10]. Among 163 young subjects who died suddenly, MVP was the only cardiac abnormality found at autopsy in 10% of cases [77].

Nevertheless, the relation between MVP and SCD has not been completely assessed. The risk of SCD appears to be related to the morphologic changes of the mitral valve. Indeed, MVP characterized by redundant and mixomatous leaflets has frequently been observed in subjects who died suddenly [21]. In these subjects, a fatal ventricular arrhythmia was the presumed mechanism of SCD. The electrical pathogenesis of SCD in MVP has been confirmed by different studies demonstrating an increased risk of malignant arrhythmias in patients who show prolongation of QT interval and increased QT dispersion [78,79].

Exertional syncope, family history of SCD, and severe mitral regurgitation are believed to further increase the athlete's risk and justify the restriction of sports participation [67].

Suspicion of MVP should arise from cardiac auscultation that, in most cases, reveals the characteristic midsystolic click and the late systolic murmur, and the diagnosis may be further confirmed by transthoracic echocardiography accord-ing to the assessed criteria [80].

Sudden Cardiac Death in Athletes Who Have Apparently Normal Hearts

In a small subset of young athletes who died suddenly (2%–5%), no structural cardiac abnormalities could be detected, even after careful examination of the heart at necropsy, with the cause of death remaining unexplained [2,12].

Corrado and colleagues [69] reported a large series (76 cases) of sudden deaths in apparently normal hearts occurring in the Veneto region of Italy over a 20-year

period. In 79% of these cases, a cardiac disease was revealed through an accurate hystologic examination of the heart: focal myocarditis (27 cases), regional ARVD (9 cases, most involving the right ventricle outflow tract), and conduction system abnormalities (18 cases leading to ventricular pre-excita-tion and 6 cases leading to heart block). In fact, a broad spectrum of abnormalities of the conduction system had been previously reported as a cause of SCD in ath-letes, including sclerosis of small arteries [12], dysplasia of the atrioventricular nodal artery [81], and Wolff-Parkinson-White syndrome [82].

In the study by Corrado and colleagues [69], no evidence of structural cardiac disease was found in 16 cases (6%), even after histologic study of the heart.

When extra cardiac causes were excluded, most of these cases were likely related to so-called ‘‘channelopathies,’’ a group of primary electrical heart diseases caused by mutations in genes encoding for cardiac ion channel pro-teins. These diseases include long and short QT syndromes (caused by sodium or potassium ion channel gene mutations), Brugada syndrome (related to a de-fective sodium channel gene), and CPVT (resulting from an abnormal ryano-dine receptor regulating calcium release from the sarcoplasmic reticulum) [83].

These diseases have been recognized as causes of premature SCD [21,83,84]. Their overall prevalence in athletes is still unknown but likely not different from that in the general population. Sports-related SCD occurs more frequently in patients who have LQTS and CPVT, in which ventricular arrhythmias are adrenergically induced. By contrast, in patients who have short QT and Bru-gada syndromes in which ventricular arrhythmias are vagal induced, these tragic events usually occur at rest, and even during sleep [21,35].

Channelopathies occur without physical findings, although most patients have a family history of premature SCD, cardiovascular symptoms, or both. Furthermore, diagnosis would be assessed or suspected in many cases with an accurate ECG evaluation [85].

Congenital long QT syndrome

LQTS refers to a group of inherited disorders of ventricular repolarization phase transmitted as autosomal dominant or recessive traits with low pene-trance [11] and having an estimated prevalence of 1 in 10,000 in the general population [86]. LQTS is characterized by ECG abnormalities including pro-longation of QT interval corrected for heart rate (QTc), T-wave alterations, and relative bradycardia. LQTS becomes symptomatic early in life, usually be-tween age 5 and 15 years [21]. Most patients present with seizure, palpitation, or syncope (all generally related to physical effort), and in about a third of asymptomatic patients, SCD due to episodic ventricular tachyarrhythmias, particularly torsades de pointes, is the initial presenting feature [87].

The strong correlation between SCD and exercise in LQTS [21] and the possibility of identification by family/personal history and ECG make this disorder of particular interest to sports cardiologists/physicians [88].

The following are the main ECG findings in more detail:

- QTc prolongation: QTc is generally greater than 480 milliseconds in LQTS. The upper limits of normality are 460 milliseconds in women and 440 millisec-onds in men and in prepubertal subjects of both sexes [89]. The risk of ma-lignant arrhythmias is highest for QTc >500 milliseconds [90].
- T-wave alterations: two-phased or notched T waves might be observed in precor-dial leads V_2 through V_5 (particularly pronounced in V_3 and V_4). These anomalies are observed more frequently in patients who have experienced cardiac events (81% versus 19%) [90].
- Relative bradycardia: patients who have LQTS have a lower average heart rate than their healthy counterparts and have chronotrophic response to exercise [91]. In contrast, trained athletes who generally present even marked rest bradycardia have a normal chronotrophic response to exercise [92].

- Catecholaminergic polymorphic ventricular tachycardia

CPVT is a primary electrical cardiac disorder clinically characterized by adrenergically induced ventricular tachycardia that typically presents polymorphi-cally and with a bidirectional pattern of QRS complex.

The standard ECG is normal, as is the echocardiographic examination; however, typical arrhythmia may be reproduced during an exercise test at a heart rate higher than 120 beats per minute [21].

At present, prevalence of CPVT and its role in SCD of athletes has not been assessed.

THE OLDER ATHLETE (>35 YEARS)

Causes of Sudden Death and Demographic Features

In contrast with their younger counterparts, the main cause of SCD in older athletes is atherosclerotic CAD, which accounts for more than 80% of cases [93,94]. Acquired valvular disease, MVP, and HCM have been recognized as much less frequent causes of death in this age group [95].

In all the published series, older athletes who died suddenly were generally men who were engaged in various vigorous individual sports such as marathon running and jogging. Most of those who died of CAD had underlying coronary risk factors (hypertension, hypercholesterolemia, smoking, diabetes) or previ-ous diagnosed CAD [95,96]. About half had experienced prodromal symptoms before the fatal event and, at necropsy, all presented severe disease (>75% ste-nosis) of one or more major coronary vessels (the left descending artery, the circumflex or the right coronary artery) [1,93,95].

Independent from the underlying cause, in most cases the final common pathway of SCD is the development of an electrical instability resulting in a fatal arrhythmia (ventricular fibrillation in more than 90% of cases) and cardiac ar-rest [97]. The ventricular arrhythmia may be primary or preceded by supra-ventricular tachycardia or bradyarrhythmia [1].

Most of the cardiac diseases described previously result in increased risk of SCD in athletes and nonathletes. Because the hallmark of all these diseases is arrhythmogenicity (the propensity for potentially lethal arrhythmias), however, the stress of intense training and competition (with increased sympathetic drive) and associated transient alterations in blood volume and hydroelectro-lytes balance undoubtedly further increases the risk [98]. Therefore, withdraw-ing affected subjects from sports activity may significantly reduce their overall risk. In the report by Corrado and colleagues [10], during a mean follow-up period of about 8 years, none of the 22 athletes who were disqualified for HCM died.

In HCM, the arrhythmogenic substrate is determined by the fibrotic and disorganized myocardium and by the areas of scarred myocardium consequent to episodes of myocardial ischemia [17,99].

In ARVD, the areas of fatty or fibrofatty replacement narrowed by normal myocardium determine the structural arrhythmogenic substrate, although induction of arrhythmias can also be explained by the dispersal of electrically conducting myocytes [42].

LQTS may be caused by potassium or sodium ion channel genes. Indepen-dent of the specific gene mutation, the final common event results in prolonged action potentials and delayed repolarization, augmenting myocardial excitabil-ity and resulting in re-entrant arrhythmias and predisposing to torsades de pointes and ventricular fibrillation [100].

Although the electric pathogenesis occurs most frequently in determination of SCD, other mechanisms should be considered for different pathologic entities.

In patients who have CCAA, SCD is due to myocardial ischemia due to anatomic malformations, particularly the acute angle takeoff of the anomalous vessel, with a narrowed slitlike orifice that collapses in a valvelike manner, thereby limiting the blood flow. These events generally occur during exercise, when dilatation of the ascending aorta due to the increased stroke volume exacerbates this anatomic condition, dramatically reducing coronary ostium and blood flow through the coronary vessel [101,102]. It has been observed, however, that patients who died suddenly during exercise usually participated in exercise of equal or even greater intensity multiple times without any symp-toms. Therefore, a sporadic spasm of the anomalous coronary artery has been proposed as the most likely mechanism of ischemia [102]. Furthermore, it may not exclude the occurrence of ventricular tachyarrhythmias related to electri-cally unstable myocardium. In fact, in several patients who had been monitored by ECG at the time of sudden death, ventricular fibrillation was documented as the final event [103].

Nonelectrical mechanism is also the basis of SCD in Marfan syndrome patients in whom abrupt aortic rupture with pericardial tamponade is the most frequent cause of exitus. Nevertheless, it has recently been suggested that a fatal ventricular arrhythmia may be the cause of death in Marfan syndrome patients, particularly in those who have MVP, LV dilatation, and abnormalities of ventricular repolarization on the ECG [104].

Finally, acute myocardial infarction usually precipitates death in older athletes who have CAD [93–96].

Given that the final common pathway in nearly all cases of athletic-field sudden deaths is cardiac arrest due to ventricular fibrillation, all athletic staff (including team physicians, trainers, and coaches) should receive specific train-ing in cardiopulmonary resuscitation (CPR) and use of external defibrillators. Because public-access defibrillation programs have demonstrated a survival benefit in persons who have out-of-hospital cardiac arrest [105], the availability of external automatic defibrillators on athletic fields has been evaluated in an attempt to minimize the occurrence of tragic events [20]. Available data suggest, however, that chances for

successful resuscitation are remote, even when CPR is started immediately and defibrillation equipment is readily available [106]. In a series of nine sudden collapses occurring in young athletes, CPR was initiated within 30 seconds after cardiac arrest in six cases and within 1 minute in two cases. An automated external defibrillator was provided by an athletic trainer in five cases and by arriving emergency medical service in four cases. In seven cases, a shock was deployed, with an average time from cardiac arrest to defibrillation of 3.1 minutes. Nevertheless, eight of the nine athletes died [107].

IS IT POSSIBLE TO PREVENT SUDDEN CARDIAC DEATH IN ATHLETES?

State-of-the-Art Preparticipation Cardiovascular Screening of Competitive Athletes

As discussed earlier, most cases of sudden death in young and older athletes are related to pre-existing cardiac diseases of which the subjects are not aware.

Recognition of these causes has raised the crucial issue of prevention all over the world. It seems clear that to prevent or minimize the risk of these tragic events, preparticipation cardiovascular evaluation of all competitive athletes is necessary; however, the exceptionally large number of athletes required to be evaulated compared with the relative rarity of SCD is a major obstacle for the implementation of preparticipation screening programs in most coun-tries. Furthermore, the most appropriate and cost-effective screening strategy has yet to be determined and there is a great disparity in the overall medical supervision of competitive athletes. At present, there exist two principal models of preparticipation screening: the American and the Italian.

The American experience

In 1996, the Sudden Death Committee and the Congenital Cardiac Defects Committee provided a consensus panel statement for health professionals with guidelines for preparticipation cardiovascular screening of young compet-itive athletes [108]. The recommended protocol included a complete medical history and physical examination, including brachial artery blood pressure measurement. Although such a protocol has the advantages of being low cost and easily implemented in a large population, it fails to identify most cardiovascular diseases that may lead to sudden death. Indeed, although most persons who have inheritable cardiac diseases have a family history of premature SCD, cardiac symptoms are infrequently referred. In fact, most young athletes who died as a result of HCM, ARVD, and CCAA in different published series had been free of symptoms before the tragic event (less than 30% had experienced premonitory symptoms such as syncope, palpitation, and chest pain) [2,10,16]. Furthermore, physical examination is generally completely normal in athletes who have CCAA and ARVD [10,16] and in most athletes affected by HCM (ie, the nonobstructive form—characterized by the absence of murmurs—the most frequently observed in young deceased athletes) [98].

In the series reported by Maron and colleagues [2] of 134 young athletes who died suddenly from cardiac causes, 115 had previously been evaluated by history and physical examination; however, a suspicion of cardiac disease had arisen in only 4 (3%) and in only 1 athlete was a correct diagnosis of Marfan syndrome assessed.

The Italian experience

A 1982 Italian law mandated that all competitive athletes of all ages (‘‘citizens engaged in official competitive sports activities’’) must undergo a periodic med-ical examination to evaluate their eligibility for sports participation. Athletes who are judged disease-free obtain a certificate of eligibility for competitive sports that must be annually renewed; conversely, athletes who have any detected or suspected

conditions that would make sports participation unsafe are referred to specific clinical centers for a definitive diagnosis and determina-tion of sports participation according to the Italian guidelines [109]. The main features of Italian preparticipation screening protocol include a general physical examination, including artery blood pressure measurement, spirometry, 12-lead ECG at rest, and a submaximal exercise test (step-test). When suspicion of a cardiac disease arises from this first-level evaluation, additional tests are required for a confirming diagnosis. This evaluation is only performed by sports physicians who have attended a postgraduate program for 4 years, who are responsible for the accuracy of their assessment, and are the final judges of eligibility in sports [110,111]. The cost of this evaluation is about 70 euro (about $90) and is charged to the athlete. It has been estimated that about 6 million athletes are screened every year in Italy.

Implementation of the screening with 12-lead ECG appears to significantly increase the diagnostic power of the screening itself. Indeed, as discussed previously, ECG abnormalities are commonly found in patients who have potentially lethal congenital cardiac diseases, with the exception of those who have CCAA. Such abnormalities are detected in more than 90% of HCM patients, in 50% to 90% of ARVD patients, and in almost all of those affected by channelopathies and Wolff-Parkinson-White syndrome. It is unfortunate that in healthy, highly trained athletes, abnormal ECG patterns mimicking structural cardiac diseases may occasionally be observed [112,113]. The most common of these ECG abnormalities include marked increase in R- and S-wave voltages in precordial leads, ST-segment elevation, T-wave changes (markedly tall, flattened, or frankly inverted), and deep Q waves. Although such alterations would be a consequence of the training-induced cardiac remod-eling and therefore a marker of athlete’s heart, their observation imposes a differ-ential diagnosis that may be obtained by echocardiography in most cases [14,23,113]. Although these false-positive cases actually reduce the overall diag-nostic power of ECG in athletes, Pelliccia and colleagues [113] demonstrated that ECG maintains a high predictive negative value (about 96%), which is to say, if ECG is normal, then the chance for presence of cardiac disease is remote.

The effectiveness of the Italian screening protocol is suggested by the reports of Corrado and colleagues [10] and Pelliccia and colleagues [14,111]. Corrado and colleagues [10] reported the results of screening 33,735 nonselected com-petitive athletes in the Veneto region of Italy over a period of 17 years. The total number of disqualified athletes was 1058, 60% of whom were withdrawn from competitive sports because of cardiovascular conditions, including 22 ath-letes who had HCM (suspected by medical history, physical findings, or ECG abnormalities and confirmed by echocardiography). All disqualified athletes underwent a mean follow-up period of 8 years during which only 1 died sud-denly of natural causes. Regarding athletes judged eligible for competitive sports, 49 died suddenly during athletic activity of cardiovascular causes who had not been diagnosed by the screening protocol. Taken together, ARVD and CCAA accounted for about two thirds of these deaths, whereas HCM was found at autopsy in just 1 athlete. These data, further strengthened by a recent report of Pelliccia and colleagues [14], suggest that the Italian national screening protocol is effective in detecting most of the potentially lethal cardiac diseases in athletes, with particular regard to HCM, which accounts for a small number of deaths in Italy. Regarding ARVD, as discussed earlier, in 1998 it was a poorly recognized disease and the criteria for diagnosis were not yet formulated. To date, detection of ARVD is one of the main targets of the Italian screening, and therefore, the number of deaths related to this cardiomyopathy will likely reduce in time.

Pelliccia and colleagues [14] reported the unique experience of the National Institute of Sports Medicine of Rome, a medical division of the National Olym-pic Committee that is responsible for the physiologic and medical evaluation of competitive athletes who form the Italian national teams from which Olympic athletes are selected every 4 years. Of the 22,000 elite competitive athletes who underwent a complete cardiovascular evaluation over a period of about 30 years, 480 (2.2%) were disqualified from competition after detection of cardiac abnormalities and only 3 (who continued to train vigorously, disregarding medical advice) experienced SCD. Such a population is highly elite and probably not representative of

the entire athletic population that annually un-dergoes Italian national screening, but it is sufficiently large for judging the effectiveness of the screening itself.

The Italian screening model has inspired the Sport Cardiology Study Group of the European Society of Cardiology, a group of cardiovascular specialists and other physicians from different European countries who have extensive clinical experience dealing with competitive athletes and which has recently provided a consensus panel with recommendations for systematic preparticipation cardiovascular evaluation of young competitive athletes [7]. The panel proposed a European standard based on 12-lead ECG in addition to history and physical examination. Their recommendations were based on the Italian model, which is the only model proven effective in detecting the largest number of potentially fatal cardiac diseases, particularly HCM, and therefore the most effective model in preventing athletic-field catastrophes.

According to this model, all young athletes should receive a basic evaluation with medical history, physical examination, and 12-lead rest ECG. Those who have positive personal history, family history of potentially inherited cardiac disease, or positive physical or ECG findings would require further evaluation, which may include echocardiography, maximal exercise testing, 24-hour ECG monitoring, and other noninvasive or invasive testing, if required. Finally, athletes affected by a cardiovascular condition associated with exercise-related sudden death would be disqualified according to the European guidelines provided by the same working group [114].

Preparticipation screening of older athletes

The preparticipation screening of older athletes is an even more complex issue. As discussed previously, the leading cause of exercise-related death in those older than 35 years is CAD and, as suggested by all the published series, these deaths usually occur in individuals who have one or more risk factors or previously rec-ognized disease. Therefore, the cardiovascular screening of older athletes should target the identification of CAD, although the screening modalities are still a con-

troversial issue. In 2001, the American Heart Association (AHA) published a con-sensus document with guidelines for preparticipation screening of master athletes that emphasizes the detection of unknown CAD [115]. This document recom-mends a basic evaluation with history and physical examination of all older ath-letes. Further, subjects who show a cardiovascular risk profile for CAD should be evaluated with symptom-limited maximal ECG exercise testing. This last recom-mendation is based on the evidence that, in adults who have coronary risk factors, an exercise test positive for myocardial ischemia is associated with an increased risk of future coronary events [116]. In contrast, the AHA does not recommend maximal exercise testing in asymptomatic older athletes who have no major cor-onary risk factors due to the poor positive predictive accuracy of the test in this subset of individuals and the high-frequency occurrence of false-negative tests that can have negative psychologic implications and result in unnecessary and costly further medical testing [117,118].

Finally, the AHA recommends, with regard to prevention of sudden death, that personnel trained in CPR and automatic external defibrillators be available at all sanctioned masters sports events [115].

NONTRAUMATIC DEATH IN ATHLETES WHO DO NOT HAVE PREEXISTING CARDIOVASCULAR DISEASES:

Commotio Cordis

Commotio cordis (or cardiac concussion) is a form of SCD in subjects free of cardiac disease that occurs with circulatory arrest due to a nonpenetrating blow to the chest that produces ventricular fibrillation without structural injury to the ribs, sternum, or heart [119].

The overall incidence of commotio cordis on athletic fields has not been established with certainty, although it has been reported as the second leading cause

of sport-related death among children and adolescents who do not have pre-existing heart disease. Up to the 70% of SCD due to commotio cordis oc-curs in subjects younger than 16 years, and such events are very uncommon after age 21 years [21,120].

The relative high frequency of commotio cordis among young athletes is likely related to the high compliance of the chest wall that might facilitate the transmission of mechanical energy from the chest blow to the myocardium, which in turn induces an electrical impulse sufficient to trigger a fatal ventric-ular arrhythmia [121].

The sports in which these incidents are most frequently seen are those such as baseball or hockey in which a small ball or puck assumes the characteristics of a projectile whereby acceleration may reach 64 km/h or 144 km/h, respec-tively [122].

Death from commotio cordis is so tragic because ventricular fibrillation is induced only when the projectile strikes the cardiac silhouette within 15 to 30 milliseconds before T-wave peak (which represents no more than 1% of the cardiac cycle) during the vulnerable phase of repolarization [123].

Because commotio cordis has a very low rate of survival even when CPR and external defibrillation are begun without delay (15%), chest protectors have been introduced in high-risk disciplines in an effort to minimize the occur-rence of these tragic events [13,119,122]. A recent study performed on animal models, however, demonstrated that commercially available baseball and lacrosse chest wall protectors were ineffective in protecting against ventricular fibrillation triggered by chest blows. Therefore, the investigators concluded that improvements in materials and design of chest wall barriers are necessary to reduce the occurrence of these tragic events and to make the athletic field safer for youths [120].

Exertional Heat Illness

Exertional heat illness is an important cause of morbidity and mortality in athletes. Heat stroke is ranked third behind head and neck trauma and cardiac diseases as a cause of death among United States high school athletes [124]. Severe

heat illness (particularly heat stroke) occurring during training and mil-itary operations has also frequently been reported in soldiers and represents the most common cause of hospitalization and nonviolent death in this population [125].

Epidemiologic data suggest that exercise-related heat illness occurs more frequently in athletes who begin training in the late summer (football, soccer, cross-country athletes) than in those who begin training in the winter or spring [126]. Significant risk factors for exertional heat illness in athletes include dehy-dration, hot and humid climate, lack of acclimatization, sleep deprivation, medications (especially diuretics or antidepressants), and dressing in full uni-form (such as football players who have an additional risk related to their clothes) [124,127]. Dehydration, with fluid loss occasionally as high as 6% to 10% of body weight, appears to be the most important risk factor in athletes exercising in the heat [124].

The American Trainer's Association provided a position statement with guidelines for prevention, recognition, and timely treatment of heat-related illness in athletes. Environmental monitoring, acclimatization, conditioning, maintaining adequate hydration, and early recognition and intervention appear to be the most important factors to reduce athletes' morbidity and mortality due to severe heat illness [126].

Athletic-field Deaths Related to Substance Abuse

It is unfortunate that doping should be included in the causes of death in ''healthy'' athletes, even if the number of athletic-field deaths attributable to substance abuse is still unknown. Doping is not only an unfair practice but also a high-risk practice for the athlete's health. In a recent position paper, the Sports Cardiology Study Group of the European Society of Cardiology [128] reported the possible induction of cardiovascular side effects (including SCD) by almost all the prohibited substances (anabolic steroids, peptide hor-mones, beta-2 agonists, diuretics, and stimulants) or methods (blood doping) included in the list provided by the World Antidoping Agency. Thus, most doping-related deaths would be likely due

to fatal cardiac arrhythmias or acute myocardial infarction. Occasionally, severe cardiovascular side effects have been associated with abuse of some dietary supplements containing stimulant substances, which might induce ventricular arrhythmias [129].

Athletic-field deaths are rare but dramatic events. A broad spectrum of diseases has been recognized as the basis of these tragic events, most of which are cardiac abnormalities. Knowledge of the causes and mechanisms underlying exercise-related sudden death and the assessment and implementation of an effective screening strategy for all competitive athletes are the tools necessary to prevent premature loss of life.

References

1. Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. J Am Coll Cardiol 1986;7:204–14.
2. Maron BJ, Shirani J, Polia LC, et al. Sudden death in young competitive athletes. Clinical, demographic and pathological profiles. JAMA 1996;276:199–204.
3. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. J Am Coll Cardiol 1998;32:1881–4.
4. Corrado D, Basso C, Rizzoli G, et al. Does sports activity enhance the risk of sudden death in adolescents and young adults? J Am Coll Cardiol 2003;42(11):1956–63.
5. Thompson PD, Stern MP, Williams P, et al. Incidence of death during jogging in Rhode Island from 1975 to 1980. JAMA 1982;274:2535–8.
6. Maron BJ, Poliac LC, Roberts WC. Risk for sudden cardiac death associated with marathon running. J Am Coll Cardiol 1996;28:428–31.
7. Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening for young competitive athletes for prevention of sudden death: proposal for a common Euro-pean protocol. Consensus Statement of the Study Group

of Sport Cardiology of the Work-ing Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Eur Heart J 2005;26:516–24.

8. Futterman LG, Myerburg R. Sudden death in athletes. An update. Sports Med 1998;26(5): 335–50.

9. Van Camp SP, Bloor CM, Mueller FO, et al. Nontraumatic sports deaths in high school and college athletes. Med Sci Sports Exerc 1995;27:641–7.

10. Corrado D, Basso C, Schiavon M, et al. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med 1998;339:364–9.

11. Towbin JA. Molecular basis of sudden cardiac death. Cardiovasc pathol 2001;10:283–95.

12. Maron BJ, Roberts WC, McAllister HA, et al. Sudden death in young athletes. Circulation 1980;62:218–29.

13. Maron BJ. Sudden death in young athletes. N Engl J Med 2003;349:1064–75.

14. Pelliccia A, Di Paolo FM, Corrado D, et al. Evidence for efficacy of the Italian national pre-participation screening programme for identification of hypertrophic cardiomyopathy in competitive athletes. Eur Heart J 2006;27(18):2196–200.

15. Corrado D, Thiene G, Nava A, et al. Sudden death in young competitive athletes: clinico-pathologic correlations in 22 cases. Am J Med 1990;89:588–968.

16. Basso C, Maron BJ, Corrado D, et al. Clinical profile of congenital coronary artery anom-alies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. J Am Coll Cardiol 2000;35(6):1493–501.

17. Maron BJ. Hypertrophic cardiomyopathy: a systematic review. JAMA 2002;287: 1308–20.

18. Watkins H, Thierfelder L, Hwang DS, et al. Sporadic hypertrophy cardiomyopathy due to de novo myosin mutations. J Clin Invest 1992;90:1666–71.

19. Klues HG, Schiffers A, Maron BJ. Phenotypic spectrum and patterns of left ventricular hypertrophy in hypertrophic cardiomyopathy: morphologic

observations and significance as assessed by two-dimensional echocardiography in 600 patients. J Am Coll Cardiol 1995;26:1699–708.

20. Basilico FC. Cardiovascular disease in athletes. Am J Sports Med 1999;27:108–21.

21. Priori SG, Aliot E, Blomstrom-Lundquist L, et al. Task force on Sudden Cardiac Death of the European Society of Cardiology. Europace 2002;4(1):3–18.

22. Maron BJ, Roberts WC, Epstein SE. Sudden death in hypertrophic cardiomyopathy: a profile of 78 patients. Circulation 1982;65:1188–94.

23. Maron BJ, Wolfoson JK, Ciro` E, et al. Relation of electrocardiographic abnormalities and patterns of left ventricular hypertrophy identified by 2-dimensional echocardiography in patients with hypertrophic cardiomyopathy. Am J Cardiol 1983;51:189–94.

24. Spirito P, Pelliccia A, Proschan MA, et al. Morphology of ‘‘athlete’s heart’’ assessed by echocardiography in 947 elite athletes, representing 27 sports. J Am Coll Cardiol 1994;74:802–69.

25. Pelliccia A, Maron BJ, Spataro A. The upper limit of physiologic cardiac hypertrophy in highly trained athletes. N Engl J Med 1991;324:295–301.

26. Maron BJ, Pelliccia A, Spirito P, et al. Cardiac disease in young trained athletes: insights into methods for distinguishing athlete’s heart from structural heart disease with particular emphasis on hypertrophic cardiomyopathy. Circulation 1995;91: 1956–16019.

27. Pelliccia A, Maron BJ, Di Paolo F, et al. Prevalence and clinical significance of left atrial remodelling in competitive athletes. J Am Coll Cardiol 2005;46(4):690–6.

28. Pelliccia A, Maron BJ, Culasso F, et al. Athlete’s heart in women. Echocardiographic characterization of highly trained and elite female athletes. JAMA 1996;276:211–5.

29. Pelliccia A, Maron BJ, Spataro A, et al. Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes. Br Heart J 1993;69:125–8.

30. Pelliccia A, Maron BJ, De Luca R, et al. Remodelling of left ventricular hypertrophy in elite athletes after long-term deconditioning. Circulation 2002;105:944–9.

31. Marian AJ, Roberts R. Recent advances in the molecular genetics of hypertrophic cardio-myopathy. Circulation 1995;92:1336–47.

32. Yang Z, Bowles NE, Scherer SE, et al. Desmosomal dysfunction due to mutations in desmo-plakin causes arrhythmogenic right ventricular dysplasia/cardiomyopathy. Circ Res 2006.

33. MacRae CA, Birchmeier W, Thierfelder L. Arrhythmogenic right ventricular cardiomyopa-thy: moving toward mechanism. J Clin Invest 2006;116(7):1825–8.

34. Corrado D, Basso C, Thiene G. Cardiomyopathy: arrhythmogenic ventricular cardiomy-opathy: diagnosis, prognosis and treatment. Heart 2000;83:588–95.

35. Cho Y, Park T, Yang DH, et al. Arrhythmogenic right ventricular cardiomyopathy and sudden cardiac death in young Koreans. Circ J 2003;67:925–8.

36. Thiene G, Nava A, Corrado D, et al. Right ventricular cardiomyopathy and sudden death in young people. N Engl J Med 1988;318:129–33.

37. McKenna WJ, Thiene G, Nava A. Diagnosis of arrhythmogenic right ventricular dysplai-sa/cardiomyopathy. Br Heart J 1994;71:215–8.

38. Corrado D, Basso C, Buja G, et al. Right bundle branch block, right precordial ST-segment elevation, and sudden death in young people. Circulation 2001;103:710–1.

39. Baran A, Nanda NC, Falkoff M, et al. Two-dimensional echocardiography detection of arrhythmogenic ventricular dysplasia. Am Heart J 1982;123:711–24.

40. Tandri H, Bomma C, Calkins H, et al. Magnetic resonance and computed tomography imaging of arrhythmogenic right ventricular dysplasia. J Magn Reson Imaging 2004;19:848–58.

41. Ahmad F. The molecular genetics of arrhythmogenic ventricular

dysplasia-cardiomyopathy. Clin Invest Med 2003;26:167–78.

42. Anderson EL. Arrhythmogenic right ventricular cardiomyopathy/dysplasia. Am Fam Physician 2006;73:1391–8, 1401.

43. Roberts WC, Siegel RJ, Zipes DP. Origin of the right coronary artery from the left sinus of Valsalva and its functional consequences: analysis of 10 necropsy patients. Am J Cardiol 1982;49:863–8.

44. Maron BJ, Liberthson RR. Sudden death from cardiac causes in children and young adults. N Engl J Med 1996;334:1039–44.

45. Yamanaka O, Hobbs RE. Coronary artery anomalies in 12595 patients undergoing coronary arteriography. Cathet Cardiovasc Diagn 1990;21:28–40.

46. Davis JA, Cecchin F, Jones TK, et al. Major coronary anomalies in a pediatric population: incidence and clinical importance. J Am Coll Cardiol 2001;37:593–7.

47. Pelliccia A, Spataro A, Maron BJ. Prospective echocardiographic screening for coronary artery anomalies in 1360 elite competitive athletes. Am J Cardiol 1993;72:978–9.

48. McConnell MV, Ganz P, Selwyn AP, et al. Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. Circulation 1995;92:3158–62.

49. Tilstra DJ, Byers PH. Molecular basis of hereditary disorders of connective tissue. Annu Rev Med 1994;45:149–63.

50. Roman MJ, Rosen SS, Kramer-Fox R, et al. The prognostic significance of the pattern of aortic root dilatation in the Marfan syndrome. J Am Coll Cardiol 1993;22:1470–6.

51. Marsalese DL, Moodie DS, Vacante M, et al. Marfan’s syndrome: natural history and long-term follow-up of cardiovascular involvement. J Am Coll Cardiol 1989;14:422–8.

52. Dingemans KP, Teeling P, Van der Wal AC, et al. Ultrastructural pathology of aortic dissec-tions in patients with Marfan syndrome: comparison with dissections in patients without Marfan syndrome. Cardiovasc Pathol 2006;15(4):203–

12.

53. Pan CW, Chen CC, Wang SP, et al. Echocardiographic study of cardiac abnormalities in families of patients with Marfan's syndrome. J Am Coll Cardiol 1985;6:1016–20.

54. Roman MJ, Devereux RB, Kramer-Fox R, et al. Two-dimensional echocardiographic aortic root dimensions in normal children and adults. Am J Cardiol 1989;64:507–12.

55. Asher CR, Lever HM. Echocardiographic profiles of diseases associated with sudden cardiac death in young athletes. In: Williams RA, editor. The athlete and heart disease: diagnosis and management. Philadelphia: Lippincott Williams & Wilkins; 1999.p. 155–72.

56. Graham TP Jr, Bricker JT, James FW, et al. Task force 1: congenital heart disease. 26th Be-thesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task force 1: congenital heart disease. J Am Coll Car-diol 1994;24(4):867–73.

57. Saeed IM, Braverman AC. Approach to the athlete with thoracic aortic disease. Curr Sports Med Rep 2007;6(2):101–7.

58. McDonald GR, Schaff HV, Pyeritz RE, et al. Surgical management of patients with the Marfan syndrome and dilatation of the ascending aorta. J Thorac Cardiovasc Surg 1981;81:180–6.

59. Glorioso J Jr, Reeves M. Marfan syndrome: screening for sudden death in athletes. Curr Sports Med Rep 2002;1(2):67–74.

60. Kinoshita N, Mimura J, Obayashi C, et al. Aortic root dilatation among young competitive athletes: echocardiographic screening of 1929 athletes between 15 and 34 years of age. Am Heart J 2000;139(4):723–8.

61. Kasikcioglu E. Aortic elastic properties with Marfanoid habitus: the need for early and accurate diagnostic methods. Eur Heart J 2005;26(1):100.

62. Glover DW, Maron BJ. Profile of preparticipation cardiovascular screening for high school athletes. JAMA 1998;279:1817–9.

63. Bresler MJ. Acute pericarditis and myocarditis. Emerg Med 1992;24:35–

41.

64. Warner WA. Cocaine abuse. Ann Intern Med 1993;119:226–35.

65. Philips M, Robinowitz M, Higgins JR, et al. Sudden cardiac death in Air Force recruits. JAMA 1986;256:2696–9.

66. Karalainen J, Nieminen MS, Heikkila J. Influenza A1 myocarditis in conscripts. Acta Med Scand 1980;207:27–30.

67. Maron BJ, Isner JM, McKenna WJ. Task force 3: hypertrophic cardiomyopathy, myocar-ditis and other myopericardial diseases and mitral valve prolapse. In: Maron BJ, Mitchell JH, editors: 26th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. J Am Coll Cardiol 1994; 24:880–885.

68. Chimenti C, Pieroni M, Frustaci A. Myocarditis: when to suspect and how to diagnose it in athletes. J Cardiovasc Med (Hagerstown) 2006;7(4):301–6.

69. Corrado D, Basso C, Thiene G. Sudden cardiac death in young people with apparently normal heart. Cardiovasc Res 2001;50(2):399–408.

70. Koester MC. A review of sudden cardiac death in young athletes and strategies for prepar-ticipation screening. J Athl Train 2001;36(2):197–204.

71. Manolio TA, Baughman KL, Rodeheffer R, et al. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung and Blood Institute Work-shop). Am J Cardiol 1992;69:1458–66.

72. Tabib A, Miras A, Taniere P, et al. Undetected cardiac lesions cause unexpected sudden cardiac death during occasional sport activity. Eur Heart J 1999;20:900–3.

73. Richardson P, McKenna W, Briston M, et al. Report of the 1995 World Health Organiza-tion/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies. Circulation 1996;93:841–2.

74. Pelliccia A, Culasso F, Di Paolo F, et al. Physiologic left ventricular cavity dilatation in elite athletes. Ann Intern Med 1999;130:23–31.

75. Freed LA, Levy D, Levine RA, et al. Prevalence and clinical outcome of

mitral valve prolapse. N Engl J Med 1999;34:1–7.

76. Zuppiroli A, Rinaldi M, Kramer-Fox R, et al. Natural history of mitral valve prolapse. Am J Cardiol 1995;75:1028–32.

77. Corrado D, Basso C, Nava A, et al. Sudden death in young people with apparently isolated MVP. G Ital Cardiol 1997;27:1097–105.

78. Kulan K, Komsuoglu B, Tunler C, et al. Significance of QT dispersion on ventricular arrhyth-mias in mitral valve prolapse. Int J Cardiol 1996;54:251–7.

79. Digeos-Hasnier S, Copie X, Paziaud O, et al. Abnormalities of ventricular repolarization in mitral valve prolapse. Ann Noninvasive Electrocardiol 2005;10(3):297–304.

80. Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical applica-tion of echocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography) developed in collaboration with the American Society of Echocardi-ography. Circulation 1997;95:1686–744.

81. Burke A, Subramanian R, Virmani R, et al. Nonatherosclerotic narrowing of atrioventricu-lar nodal artery and sudden death. J Am Coll Cardiol 1993;21:117–22.

82. Thiene G, Pennelli N, Rossi L. Cardiac conduction system abnormalities as a possible cause of sudden death in young athletes. Hum Pathol 1983;14:704–9.

83. Corrado D, Basso C, Thiene G. Is it time to include ion channel diseases among cardiomy-opathies? J Electrocardiol 2005;38(4 Suppl):81–7.

84. Arias MA, Fernandez-Guerrero JC, Herrador J, et al. Catecholaminergic polymorphic ventricular tachycardia as a cause of sudden death in athletes. Am J Emerg Med 2006;24(2):253–4.

85. Seidman C. Cardiovascular genetic disorders that cause sudden death in athletes. In: William RA, editor. The athlete and heart disease: diagnosis and management. Philadel-phia: Lippincott Williams & Wilkins; 1999. p. 41–52.

86. Ackeman MJ. The long QT syndrome. Pediatr Rev 1998;19:232–8.

87. Moss AJ, Robinson J. Clinical features of the idiopathic long QT

syndrome. Circulation 1992;85(I):140–4.

88. Napolitano C, Bloise R, Priori SG. Long QT syndrome and short QT syndrome: how to make correct diagnosis and what about eligibility for sports activity. J Cardiovasc Med 2006;7(4):250–6.

89. Merri M, Benhorin J, Alberti M, et al. Electrocardiographic quantification of ventricular repolarization. Circulation 1989;80:1301–8.

90. Priori SG, Schwartz PJ, Napolitano C, et al. Risk stratification in the long QT syndrome. N Engl J Med 2003;348:1866–74.

91. Schwartz PJ, Priori SG, Napolitano C. The Long QTsyndrome. In: Zipes DP, Jalife J, editors. Cardiac electrophysiology: from cell to bedside. Third edition. Philadelphia: WB Saun-ders; 2000. p. 788–811.

92. Franklin BA, Roitman L. Cardiorespiratory adaptations to exercise. In: ACSM's resource manual for guidelines for exercise testing and prescription. Third edition. Philadelphia: Lippincott Williams & Wilkins. p. 156–63.

93. Waller BF, Roberts WC. Sudden death while running in conditioned runners aged 40 years or over. Am J Cardiol 1980;45:1292–300.

94. Northcote RJ, Evans ADB, Ballantyne D. Sudden death in squash players. Lancet 1984;1: 148–51.

95. Noakes T. Heart disease in marathon runners: a review. Med Sci Sports Exerc 1987;19: 187–94.

96. Jackson RT, Beaglehole NR, Sharpe N. Sudden death in runners. N Z Med J 1983;96: 289–92.

97. Myerburg RJ, Kessler KM, Castellanos A. Sudden cardiac death: structure, function and time-dependence of risk. Circulation 1992;85(I):2–10.

98. Maron BJ. Sudden cardiac death due to hypertrophic cardiomyopathy in young athletes. In: Williams RA, editor. The athlete and heart disease: diagnosis and management. Phila-delphia: Lippincott Williams & Wilkins; 1999.

99. Basso C, Thiene G, Corrado D, et al. Hypertrophic cardiomyopathy and sudden death in the young: pathologic evidence of myocardial ischemia. Hum Pathol 2000;31(8):893–4.

100. Bennet PB, Yazawa K, Makita N, et al. Molecular mechanisms for an inherited cardiac arrhythmia. Nature 1995;376:683–5.

101. Taylor AJ, Rogan KM, Virmani R. Sudden cardiac death associated with congenital coro-nary anomalies. J Am Coll Cardiol 1992;20:640–7.

102. Pelliccia A. Congenital coronary artery anomalies in young patients. New perspectives for timely identification. J Am Coll Cardiol 2001;37:598–600.

103. Liberthson RR, Dinsmore RE, Fallon JT. Aberrant coronary artery origin from the aorta. Report of 18 patients, review of the literature and delineation of the natural history and management. Circulation 1979;59:748–54.

104. Yetman AT, Bornemeier RA, McCrindle BW. Long-term outcome in patients with Marfan syndrome: is aortic dissection the only cause of sudden death? J Am Coll Cardiol 2003;41(2):329–32.

105. Kerber RC, Becker LB, Bourland JD, et al. Automatic external defibrillators for public access defibrillation: recommendations for specifying and reporting arrhythmia analysis algo-rithm performance, incorporating new waveforms, and enhancing safety. Circulation 1997;95:177–82.

106. Maron BJ. Cardiovascular risks to young persons on the athletic field. Ann Intern Med 1998;129:379–86.

107. Drezner JA, Rogers KJ. Sudden cardiac arrest in intercollegiate athletes: detailed analysis and outcomes of resuscitation in nine cases. Heart Rhythm 2006;3(7):755–9 [Epub 2006 Mar 28].

108. Maron BJ, Thompson PD, Puffer JC, et al. Cardiovascular preparticipation screening of competitive athletes. A statement for health professionals from the Sudden Death Commit-tee (clinical cardiology) and Congenital Cardiac Defects Committee (cardiovascular disease in the young), American Heart Association. Circulation 1996;94:850–6.

109. Cardiologic protocols for competitive sport’s qualification of the Italian Committee for the competitive sport’s qualification. Comitato organizzativo per l’idoneita` allo sport (FMSI, SIC-Sport, SIC, ANCE, AMCO), et al. Protocolli cardiologici per il giudizio di idoneita` allo sport agonistico [in Italian]. G Ital Cardiol

1989;19:250–72.

110. Pigozzi F, Spataro A, Fagnani F, et al. Preparticipation screening for the detection of cardiovascular abnormalities that may cause sudden death in competitive athletes. Br J Sports Med 2003;37(1):4–5.

111. Pelliccia A, Maron BJ. Preparticipation cardiovascular evaluation of the competitive athlete: perspectives from the 30-year Italian experience. Am J Cardiol 1995;75:827–9.

112. Lictman J, O'Rourke RA, Klein A, et al. Electrocardiogram of the athlete: alterations simu-lating those of organic heart disease. Arch Intern Med 1973;132:763–70.

113. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardio-graphic patterns in trained athletes. Circulation 2000;102:278–84.

114. Pelliccia A, Fagard R, Bjornstad HH, et al. Recommendations for competitive sports partic-ipation in athletes with cardiovascular disease. A consensus document from the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Eur Heart J 2005;26(14):1422–45.

115. Maron BJ, Arau`jo CG, Thompson PD, et al. Recommendations for preparticipation screen-ing and the assessment of cardiovascular disease in master athletes. An advisory for healthcare professionals from the working groups of the World Heart Federation, the Inter-national Federation of Sports Medicine, and the American Heart Association Committee on Exercise, Cardiac Rehabilitation and Prevention. Circulation 2001;103:327–34.

116. Rautaharju PM, Prineas RJ, Eifler WJ, et al. Prognostic value of exercise electrocardiogram in men at high risk of future coronary heart disease: multiple risk factor intervention trial experience. J Am Coll Cardiol 1986;8:1–10.

117. Gibbons RJ, Balady GJ, Beasley JW, etal. ACC/AHA guidelines for exercise testing: a report of the American College of Cardiology/American Heart

Association Task Force on Practice Guidelines (Committee on Exercise Testing). J Am Coll Cardiol 1997;30:260–311.

118. Pigozzi F, Spataro A, Alabisio A, et al. Role of exercise stress test in master athletes. Br J Sports Med 2005;39:527–31.

119. Maron BJ, Poliac LC, Kaplan JA, et al. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. N Engl J Med 1995;333:337–42.

120. Weinstock J, Maron BJ, Song C, et al. Failure of commercially available chest wall protec-tors to prevent sudden cardiac death induced by chest wall blows in an experimental model of commotio cordis. Pediatrics 2006;117(4):656–62.

121. Stasburger JF, Maron BJ. Commotio cordis. N Engl J Med 2002;347:1248.

122. Maron BJ, Gohman TE, Kyle SB, et al. Clinical profile and spectrum of commotio cordis. JAMA 2002;287:1142–6.

123. Link MS, Maron BJ, VanderBrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. J Am Coll Cardiol 2001;37:649–54.

124. Coris EE, Ramirez AM, Van Durme DJ. Heat illness in athletes: the dangerous combination of heat, humidity and exercise. Sports Med 2004;34(1):9–16.

125. Carter R III, Cheuvront SN, Williams JO, et al. Epidemiology of hospitalizations and deaths from heat illness in soldiers. Med Sci Sports Exerc 2005;37(8):1338–44.

126. Binkley HM, Beckett J, Casa DJ, et al. National Athletic Trainer's Association position statement: exertional heat illness. J Athl Train 2002;37(3):329–43.

127. Francis K, Feinstein R, Brasher J. Heat illness in football players in Alabama. Ala Med 1991;60(9):10–4.

128. Deligiannis A, Bjornstad H, Carre F, et al. ESC Study Group of Sports Cardiology position paper on cardiovascular adverse effects of doping in athletes. Eur J of Card Prev and Rehab 2006;13(5):687–94.

129. Samenuk D, Link MS, Homoud MK, et al. Adverse cardiovascular events temporally asso-ciated with ma huang, an herbal source of ephedrine. Mayo Clin Proc 2002;77:12–6.

TESTS

1. Anaphylactic shock - this is a common systemic reaction that develops:

a) irrespective of the route of administration of the allergen into the body;

b) only when parenterally administered drug;

c) only if a radiopaque material.

2. When fulminant anaphylactic shock states:

a) sharp drop in blood pressure, consciousness is kept, sometimes there are convulsions;

b) sharp drop in blood pressure, depression of consciousness, convulsions appear;

c) sharp drop or increase blood pressure, depression of consciousness, convulsions appear.

3. With the development of anaphylactic shock during intramuscular and subcutaneous injections should:

a) to continue administering the drug, optionally enter antihistamines;

b) immediately discontinue the drug, not to remove the needle from the injection site;

c) immediately discontinue the drug and remove the needle from the injection site.

4. With the development of anaphylactic shock by the intravenous injection should:

a) discontinue the drug, but do not go out of the vein;

b) discontinue the drug, get out of the vein;

c) administering the drug to terminate.

5. All drugs in anaphylactic shock is preferable to administer:

a) intravenously;

b) intramuscularly;

b) subcutaneously.

6. Allocate the following mechanisms of sudden cessation of circulation:

a) ventricular fibrillation, asystolia;

b) ventricular tachycardia, electromechanical dissociation;

c) all of the above.

7. Causes sudden cessation of circulation can be:

a) pulmonary embolism, cerebrovascular accident;

b) an overdose of drugs, drowning in fresh water, electric shock;

c) There are no right answers.

8. Sudden cessation of circulation is diagnosed when the following symptoms:

a) Lack of awareness in translation in the horizontal position, the absence of a pulse on the main arteries, lack of breath, dilated pupils;

b) lack of consciousness when translated into a horizontal position, the absence of the radial pulse, no breathing, dilated pupils;

c) the absence of consciousness, no pulse, no breathing, dilated pupils.

9. Incipient hypoglycemic states are:

a) acute hunger, fatigue, anxiety, mental irritation, impaired speech;

b) retardation, dezorintatsiya, anxiety, impaired speech;

c) expressed unmotivated weakness, irritation, brief loss of consciousness, disorientation in space.

10. At a young age is more common:

a) anterior myocardial infarction;

b) back myocardial infarction;

c) infarction of the right ventricle.

11. Fainting - it is:

a) sudden prolonged unconsciousness resulting from acute stroke or acute metabolic disturbances in the brain tissue ;

b) short-term reversible sudden loss of consciousness resulting from acute stroke or acute metabolic disturbances in the brain tissue ;

c) sudden irreversible loss of consciousness resulting from acute stroke or acute metabolic disorders in the brain tissues.

12. Development of orthostatic syncope is associated with:

a) deposition in the blood vessels of the lower limbs;

b) deposition of blood vessels in the brain;

c) deposition of blood in the liver.

13. Causes of acute left ventricular failure can be:

a) valvular heart disease, hypertension, myocardial infarction, myocarditis, the deterioration of the coronary circulation;

b) a significant increase in the load in excess of reserve possibilities infarction;

c) all the answers are correct.

14. First aid for acute left ventricular failure includes the following activities:

a) give the patient semi-sitting position, to impose on the upper and lower limbs harnesses provide breathing oxygen defoaming agent, every 10 minutes, until medical help arrives, give the patient under the tongue 1 tablet of nitroglycerin to force diuresis - 2-3 tablets furosemide;

b) give the patient a horizontal position on a hard surface to provide breathing oxygen defoaming agent, every 10 minutes, until medical help arrives, give the patient under the tongue 1 tablet of nitroglycerin to force diuresis - 2-3 tablets

furosemide;

c) provide access to fresh air, give the patient semi-sitting position, a tourniquet on the upper limbs, provide breathing oxygen defoaming agent, every 10 minutes, until medical help arrives, give the patient under the tongue 1 tablet of nitroglycerin to force diuresis - 2-3 tablets furosemide.

15. Spontaneous pneumothorax - this is:

a) the accumulation of air in the pleural cavity associated with mechanical damage lung or chest cavity;

b) the accumulation of air in the pleural cavity that is not associated with mechanical damage lung or chest cavity;

c) both statements are true.

16. The most frequent causes of brain compression are:

a) intracranial hematoma , bone fragments ;

b) foreign bodies , edema and brain swelling ;

c) all the answers are correct.

17. Brain injury is the result of:

a) shock wave propagates in brain structures locally in shock;

b) the propagation of the shock wave through the brain structures from the point of impact to the opposite pole of the skull with the rapid pressure drop in the ground;

c) there are no right answers.

18. Epidural hematoma occurs in the spot kick with a head injury of varying intensity, but more often:

a) mild;

b) moderate severity;

c) severe.

CONTENTS

Оригінал-макет підготовлено на кафедрі фізичної реабілітації, спортивної медицини, фізвиховання і здоров`я ЗДМУ
Тиражування - кафедра фізичної реабілітації, спортивної медицини, фізвиховання і здоров`я ЗДМУ
69035, м. Запоріжжя, пр. Маяковського, 26