

How to recognise sudden cardiac arrest on the pitch

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Sudden cardiac arrest (SCA), which occurs at a rate of about 1 in ~50 000 athlete years, is the most common cause of death in exercising young athletes.¹ SCA is most frequently caused by lethal cardiac arrhythmias—that is, ventricular fibrillation (VF). If clinicians recognise key features of SCA early and accurately, they can immediately begin cardiopulmonary resuscitation (CPR) and use an automated external defibrillator (AED) as needed.² This review aims to (1) assist health professionals recognise the signs (and avoid common pitfalls) of SCA and (2) emphasise best practice responder strategies for SCA on the pitch.

CASE EXAMPLE OF SCA

Miklos Feher, a striker for Benfica, suffered a SCA on 25 January 2004. The event can be viewed at: https://youtu.be/T7-kKy_XDQU. The underlying cause of death was later reported to be VF in a patient with hypertrophic cardiomyopathy.

CLINICAL QUESTION

How can clinicians and sports professionals rapidly recognise SCA on the field of play? Recognising SCA on the pitch can be challenging due to the sports setting, other mimicking causes and the rapid onset of signs following cerebral hypoperfusion. To prevent death or serious sequelae, it is key that clinicians recognise SCA immediately and start adequate management (ie, CPR and defibrillation).

CLINICAL FEATURES OF SCA

Figure 1 is a schematic of SCA from time T=0–60 s with corresponding signs, ECG, blood pressure tracing and breathing pattern. Prolonged, sudden cerebral hypoperfusion causes a typical set of events.³ The onset of VF leads to a rapid fall in blood pressure. Symptoms of cerebral hypoperfusion start about ~5–8 s after circulatory arrest; this delay is explained by the cerebral ischaemic anoxia reserve time.³

Prolonged prodromal period

Most athletes with SCA have no warning symptoms prior to their collapse. However, after the onset of VF, some athletes may experience symptoms that occur rapidly and are of short duration. The athlete may experience light-headedness or seeing black for a few seconds, after which consciousness is lost. If the initial rhythm is ventricular tachycardia which later deteriorates to VF, the fall in blood pressure may be less steep, resulting in a prolonged prodromal phase. The patient will then not ‘drop

dead’ but instead bend over or sit/lay down. This more gradual presentation is an important pitfall and may delay clinicians recognising the SCA. When a collapse or failure to respond are not preceded by body or head contact with other competitors—think SCA.

Myoclonic jerks

At the onset of SCA, a striking pallor can be observed, along with sweating, pupillary dilatation and changes in breathing. The eyes are wide open and turn upwards—the ‘face’ of SCA. Over 50% of athletes with SCA will display seizure-like activity or myoclonic jerks such as shaking, quivering or twitching.⁴ Avoid confusing SCA with an epileptic seizure. The jerks that accompany SCA are different from those seen in a seizure: fewer than 10 jerks in SCA versus more than 20 jerks in a seizure.⁵ Posturing (unilateral or bilateral flexion or extension of the arms) may also occur in the context of SCA and is not a specific sign for a seizure. On the contrary, such seizure-like phenomena should be seen as highly suspicious of SCA in a collapsed and unresponsive athlete, especially one who had no apparent head trauma.

The presentation of myoclonic jerks can be viewed at: <https://youtu.be/SOsNeUg1iGA>.

Abnormal breathing

A common misconception is that breathing stops immediately after SCA. Instead, the depth and rate of breathing increases initially, but after about 30 s gasping (‘agonal’ breathing) starts. Gasping can be recognised as low frequency (~4–6/min) deep but fast in and out respirations and occurs in ~35% of athletes with SCA.⁴ Gasping is sometimes confused with the so-called phenomena of ‘swallowing one’s tongue’.⁶ It is a serious misconception that SCA can be caused by tongue swallowing, and manoeuvres such as a ‘tongue sweep’ should never delay starting CPR and defibrillation. The duration of agonal gasping is variable, lasting from <30 s to about 2 min and is followed by terminal apnoea. The observation of gasping during SCA means rescuers reached the athlete rapidly and is associated with higher survival in patients with SCA in the general population.⁷ When apnoea sets in the patient is pale, with open eyes, wide pupils and completely flaccid.

HOW TO MANAGE SCA

Management of patients with SCA on the pitch starts with early recognition, activation of the emergency medical response system, immediate chest compressions and defibrillation as soon



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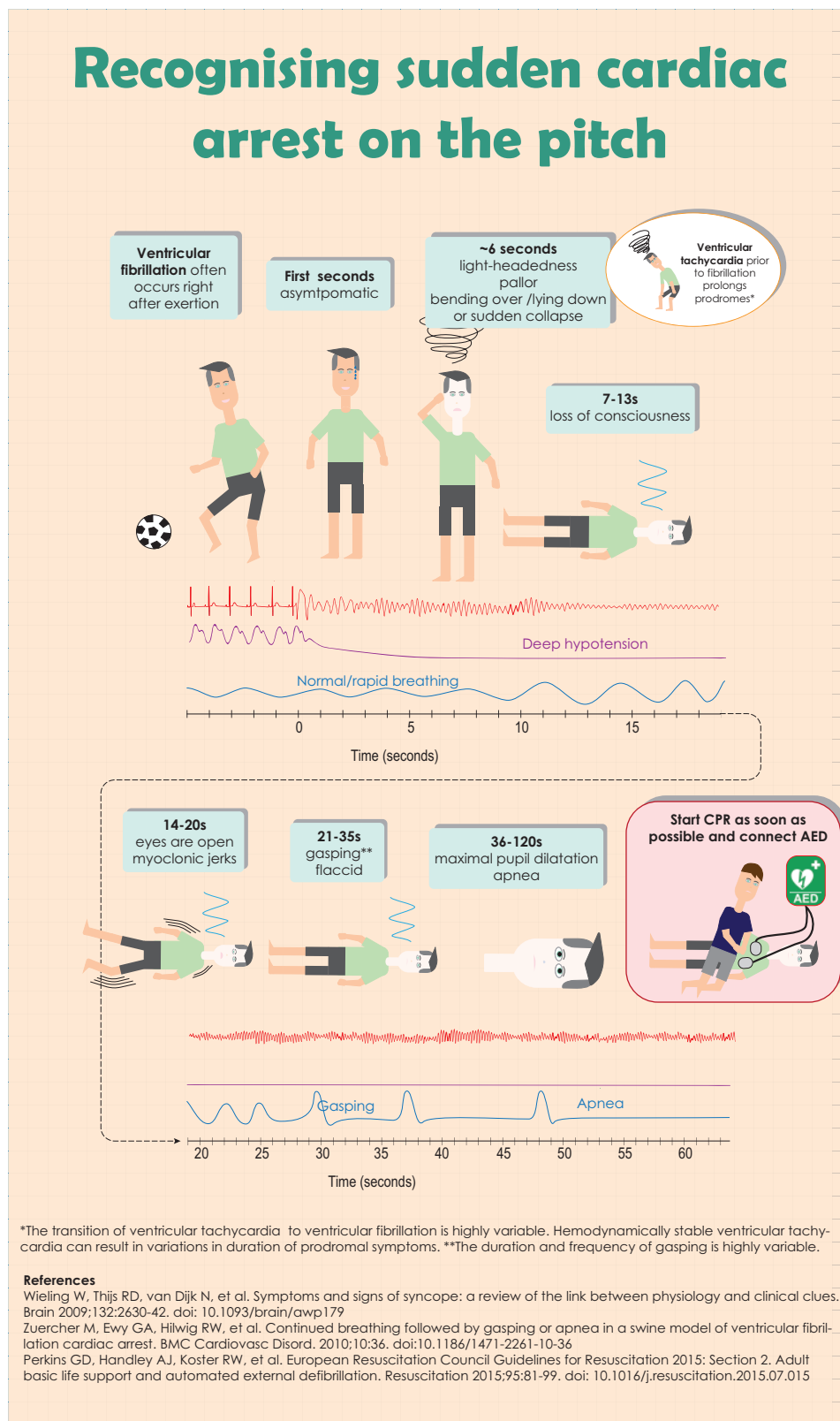


Figure 1 Recognising sudden cardiac arrest on the pitch. From top to bottom: symptoms and signs of sudden cardiac arrest, schematic ECG, schematic continuous blood pressure tracing, schematic tracing of pulmonary volume.

as possible. After 30 chest compressions, give two rescue breaths and continue with this CPR ratio. As soon as an AED arrives, the pads should be placed for rhythm analysis and defibrillation as indicated.⁸ All efforts should be made to limit interruptions in CPR, and chest compressions should

be started immediately after defibrillation and continued until the victim becomes responsive or the AED reanalyses the rhythm (every 2 min). Survival after SCA in young athletes is >80% when CPR is provided and an AED closely accessible.⁹

PRACTICAL TIPS

1. Rapid recognition of SCA is key to survival.
2. Collapse and failure to respond not associated with body or head contact with competitors is an SCA until proven otherwise.
3. Normal/rapid breathing can continue for up to 30s after SCA.
4. Abnormal breathing followed by agonal (periodic) gasping and apnoea point strongly to the diagnosis of SCA.
5. Myoclonic jerks (usually <10) and posturing often occur in SCA.
6. Bending over, sitting or laying down may precede loss of consciousness.
7. Immediately begin chest compressions and apply an AED when SCA is suspected.

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