BASICS OF FIRST AID
Cardiac arrest and BLSD

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Slides

All the slides can be downloaded as pdf documents here (teacher webpage at unica.it)

Practical activities

Hands-on training IS MANDATORY BEFORE TAKING THE TEST.

Practical activities

The Secretariat of the Dean of the Faculty organizes the practical activities.

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Section 1

Physiology of the cardiovascular sytem

The Circulatory System: Essential Transport and Homeostatic Regulation

Transport and distribution of essential substances

The circulatory system transports and distributes essential substances like oxygen, nutrients, hormones, and waste products to tissues throughout the body.

Participation in homeostatic mechanisms

The circulatory system helps maintain homeostasis by regulating body temperature, fluid balance, and oxygen and nutrient supply according to physiological needs.

Blood as a transport medium

Blood, a heterogeneous fluid, is essential for the transport processes performed by the heart and blood vessels throughout the body.

Cardiovascular system components

The cardiovascular system is composed of the heart as a pump, blood vessels for distribution and collection, and an extensive network of capillaries for rapid exchange between tissues and the vascular system.

Koeppen BM. Berne and Levy Physiology. 8th ed. Elsevier; 2023.

The Functioning of the Heart

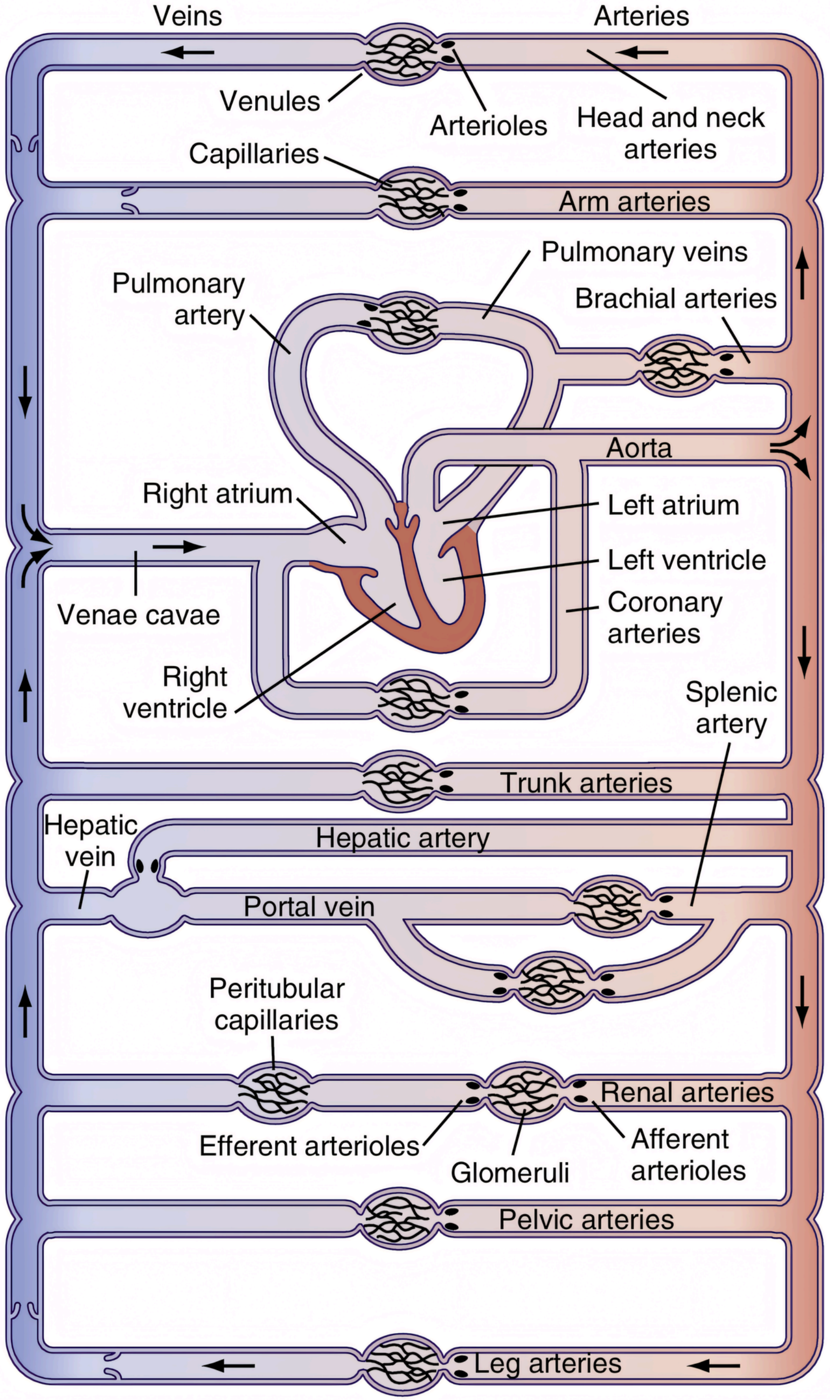
1. **Two Pumps in Series**
   The heart consists of two pumps in series: one pump propels blood through the lungs for exchange of O2 and CO2 (the pulmonary circulation), and the other pump propels blood to all other tissues of the body (the systemic circulation).

2. **Unidirectional Flow**
   Flow of blood through the heart is in one direction (unidirectional). Unidirectional flow through the heart is achieved by the appropriate arrangement of flap valves.

3. **Continuous Peripheral Flow**
   Although cardiac output is intermittent, continuous flow to body tissues (periphery) occurs by distention of the aorta and its branches during ventricular contraction (systole) and by elastic recoil of the walls of the large arteries with forward propulsion of the blood during ventricular relaxation (diastole).

The heart's efficient and unidirectional flow ensures continuous blood supply to the body's tissues, enabling vital gas exchange and nutrient delivery.

Koeppen BM. Berne and Levy Physiology. 8th ed. Elsevier; 2023.



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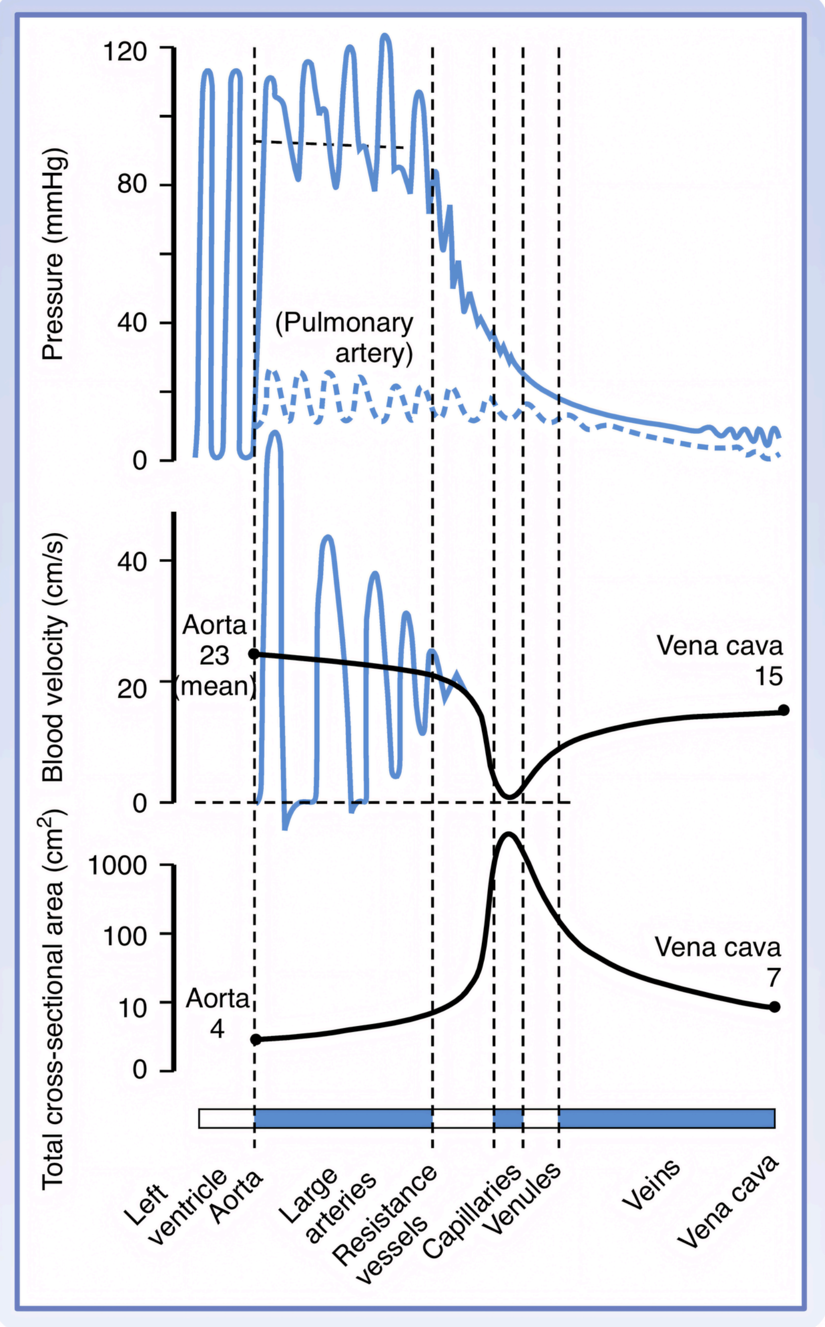
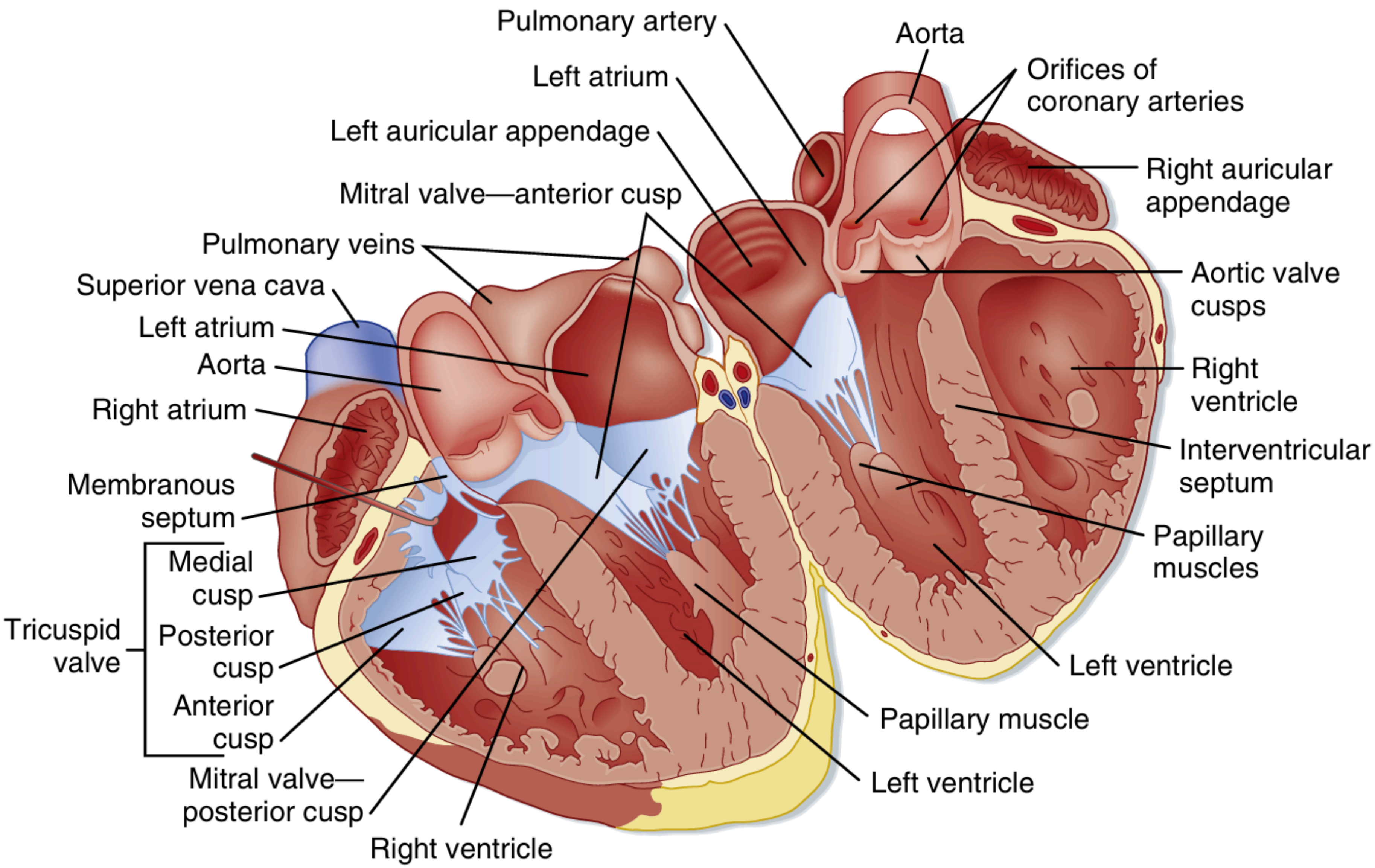


Blood moves rapidly through the aorta and its arterial branches. As these branches approach the periphery, they narrow, and their walls become thinner.

The proportions of elastic tissue, smooth muscle, and fibrous tissue (largely collagen) change in each type of blood vessel, leading to significantly different physical and physiological properties. The aorta is predominantly elastic, but the peripheral arteries become more muscular, with the muscular layer predominating in the arterioles. In the large arteries, frictional resistance is relatively small, and pressures are slightly less than those in the aorta.

Conversely, small arteries offer moderate resistance to blood flow, with maximal resistance found in the arterioles, which are often referred to as the “stopcocks” of the vascular system. Therefore, the pressure drop is greatest across the terminal segment of small arteries and arterioles. Adjustments in the contraction of the circular muscle in these small vessels regulate tissue blood flow and help control arterial blood pressure.

Koeppen BM. *Berne and Levy Physiology*. 8th ed. Elsevier; 2023.



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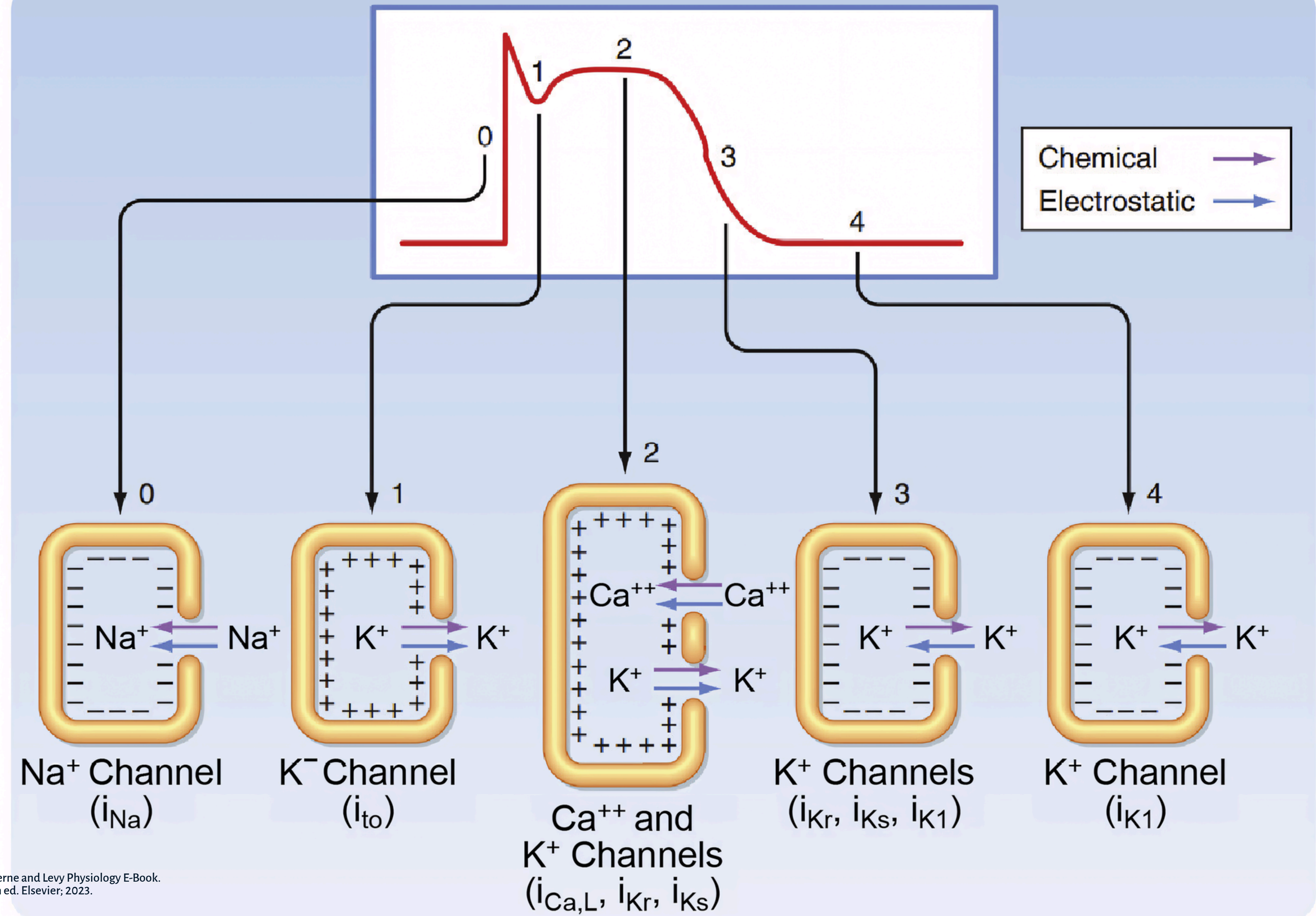

Koeppen BM. Berne and Levy Physiology E-Book.
8th ed. Elsevier; 2023.

WebView

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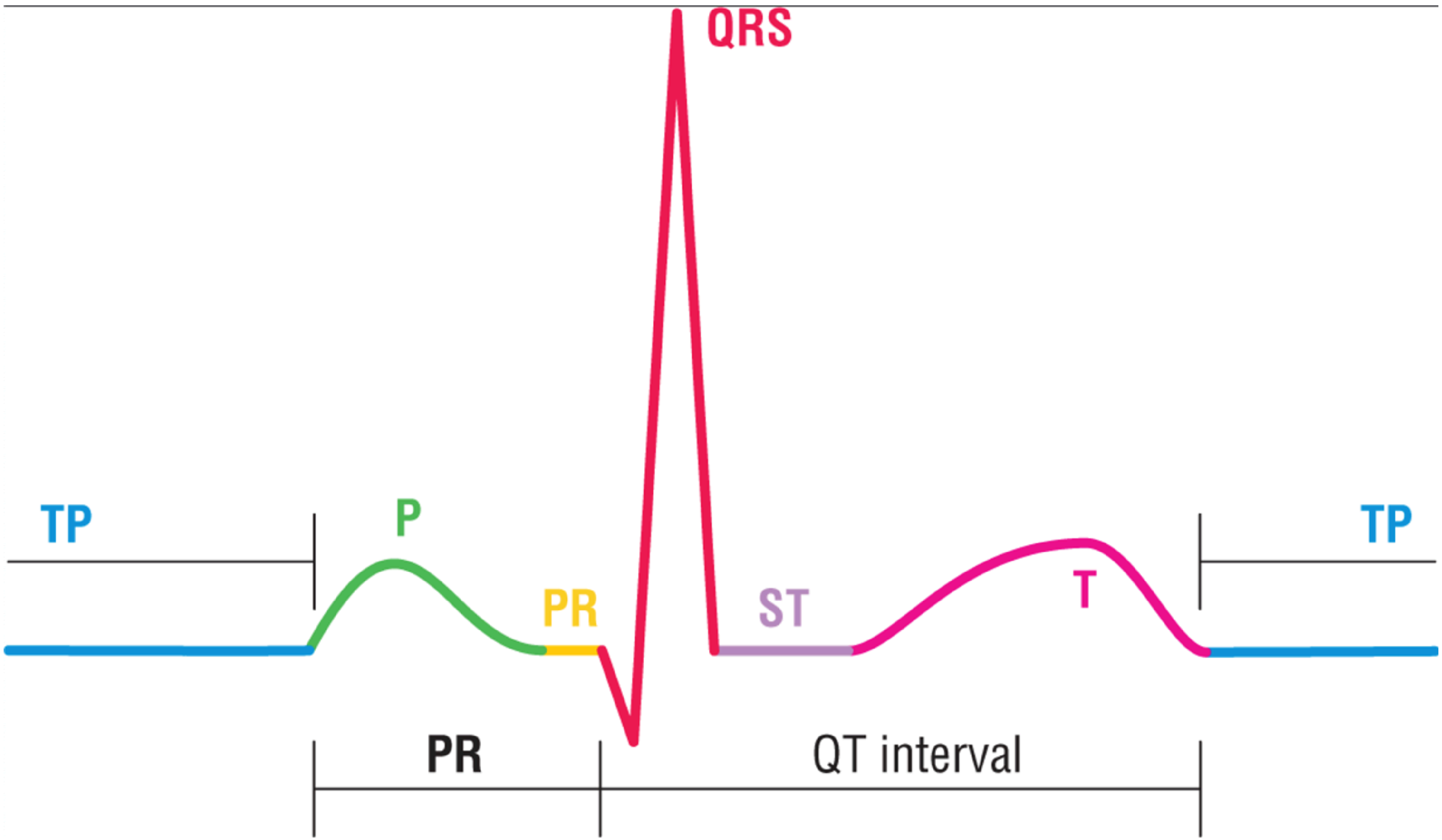
QRS
TP
P
TP
PR
ST
T
PR
QT interval

Section 2

Cardiocirculatory arrest definition and epidemiology

Cardiac arrest (CA) is a clinical syndrome defined as the "cessation of cardiac mechanical activity, as confirmed by the absence of signs of circulation"

Varvarousis D, Varvarousi G, Iacovidou N, D'Aloja E, Gulati A, Xanthos T. The pathophysiologies of asphyxial vs dysrhythmic cardiac arrest: implications for resuscitation and post-event management. *The American Journal of Emergency Medicine*. 2015;33(9):1297-1304. doi:10.1016/j.ajem.2015.06.066

More than just a setting?

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Cardiac Arrest Epidemiology and Outcomes in Europe

- **Cardiac arrest registries in Europe**

  Approximately 70% of European countries have out-of-hospital cardiac arrest (OHCA) registries, but the completeness of data capture varies widely.

- **OHCA incidence in Europe**

  The annual incidence of OHCA in Europe is between 67 to 170 per 100,000 inhabitants.

- **OHCA resuscitation attempts by EMS**

  Resuscitation is attempted or continued by emergency medical services (EMS) personnel in about 50-60% of OHCA cases (between 19 to 97 per 100,000 inhabitants).

- **Bystander CPR and AED use**

  The rate of bystander CPR varies between and within European countries (average 58%, range 13%-83%), and the use of automated external defibrillators (AEDs) remains low (average 28%, range 3.8%-59%).

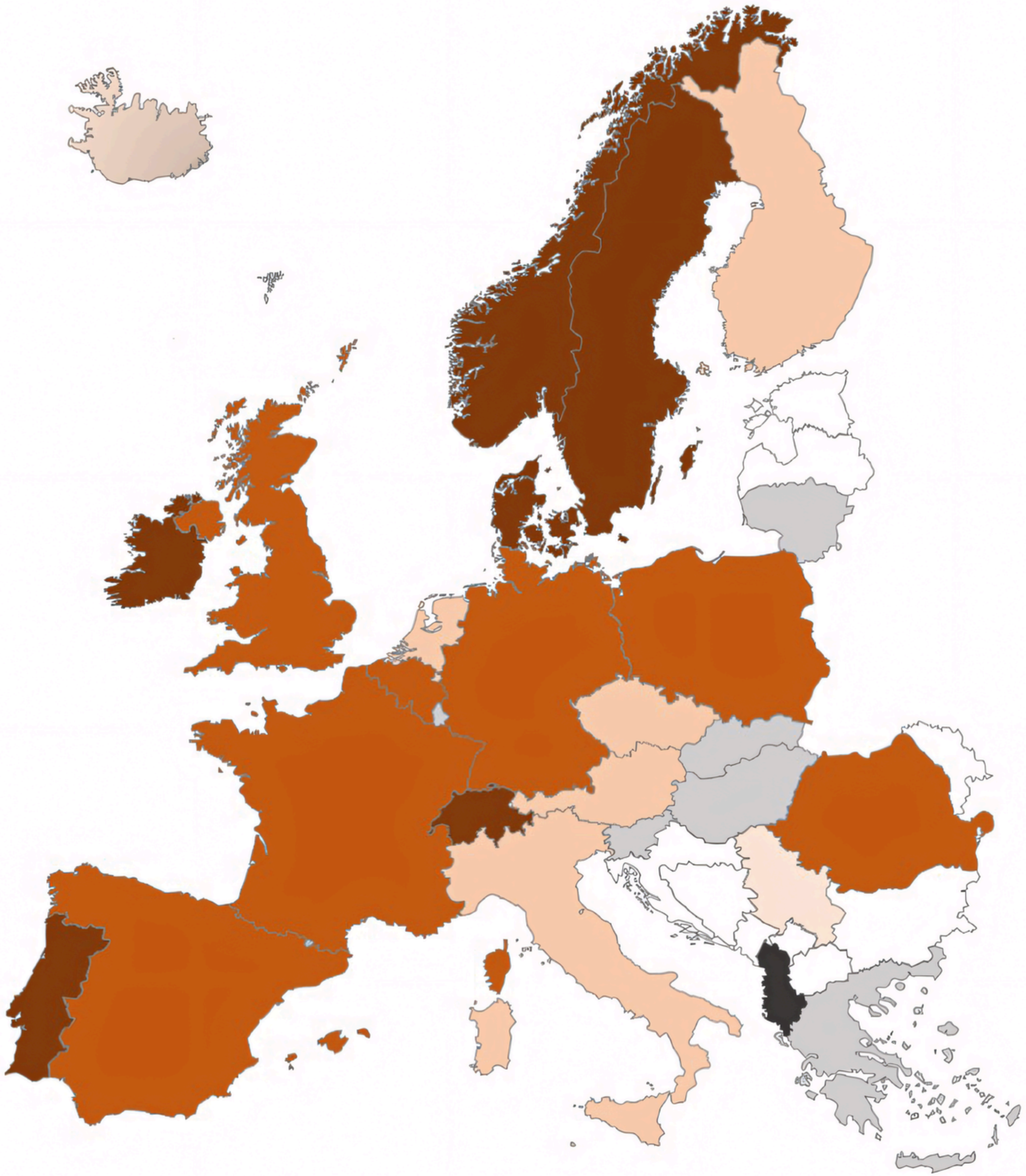
- **IHCA incidence and survival rates**

  The annual incidence of in-hospital cardiac arrest (IHCA) in Europe is between 1.5 and 2.8 per 1,000 hospital admissions, and survival rates at 30 days/hospital discharge range from 15% to 34%.

- **Long-term outcomes and rehabilitation**

  In countries where withdrawal of life-sustaining treatment is routinely practiced, good neurological outcomes are seen in > 90% of survivors, but post-cardiac arrest rehabilitation services are widely variable across Europe.

Gräsner JT, Herlitz J, Tjelmeland IBM, et al. European Resuscitation Council Guidelines 2021: Epidemiology of cardiac arrest in Europe. Resuscitation. 2021;161:61-79. doi:10.1016/j.resuscitation.2021.02.007



The European Resuscitation Council's (ERC) European Registry of Cardiac Arrest (EuReCa) provides comprehensive data on cardiac arrest epidemiology in Europe.

The reported incidence of cardiac arrest varies significantly between and within countries.

EuReCa ONE estimated the annual incidence of EMS-confirmed out-of-hospital cardiac arrest (OHCA) at 84 per 100,000 inhabitants, ranging from 28 to 160.

The incidence of OHCA with EMS-attempted resuscitation was 49 per 100,000, varying from 19 to 104.

The follow-up EuReCa TWO study reported an annual EMS-confirmed OHCA incidence of 89 per 100,000, ranging from 53 to 166, and an EMS-attempted resuscitation incidence of 56 per 100,000, varying from 27 to 91.1

Resuscitation is attempted in approximately 50-60% of EMS-attended cases considered for resuscitation, although substantial underreporting and considerable international variability exist.

The number of reported OHCAs in Europe has increased in recent years, likely due to improved case identification and increased registry coverage, rather than necessarily reflecting a true incidence rise.

National registries across Europe. The darkest colour indicates a national registry covering the whole country, the second darkest colour indicates a national registry covering parts of the country, medium orange indicates several local registries, light with grey indicates one local registry, grey indicates no local registries and black is unknown. White colour indicates the country did not participate in the survey.

Gräsner JT, Herlitz J, Tjelmeland IBM, et al. European Resuscitation Council Guidelines 2021: Epidemiology of cardiac arrest in Europe. *Resuscitation*. 2021;161:61-79. doi:10.1016/j.resuscitation.2021.02.007

Section 3

Cardiac arrest pathophysiology

Common Causes of Cardiac Arrest

- **Ventricular Fibrillation (VF) and Pulseless Ventricular Tachycardia**

  These arrhythmic events are the predominant cardiac causes of sudden cardiac arrest in adults, triggered by myocardial ischemia, cardiac channelopathies, and electrolyte disturbances.

- **Asphyxial Cardiac Arrest**

  Noncardiac causes, such as impaired alveolar ventilation due to pulmonary diseases, airway obstruction, or neuromuscular issues, are the leading causes of cardiac arrest in younger individuals, children, and neonates.

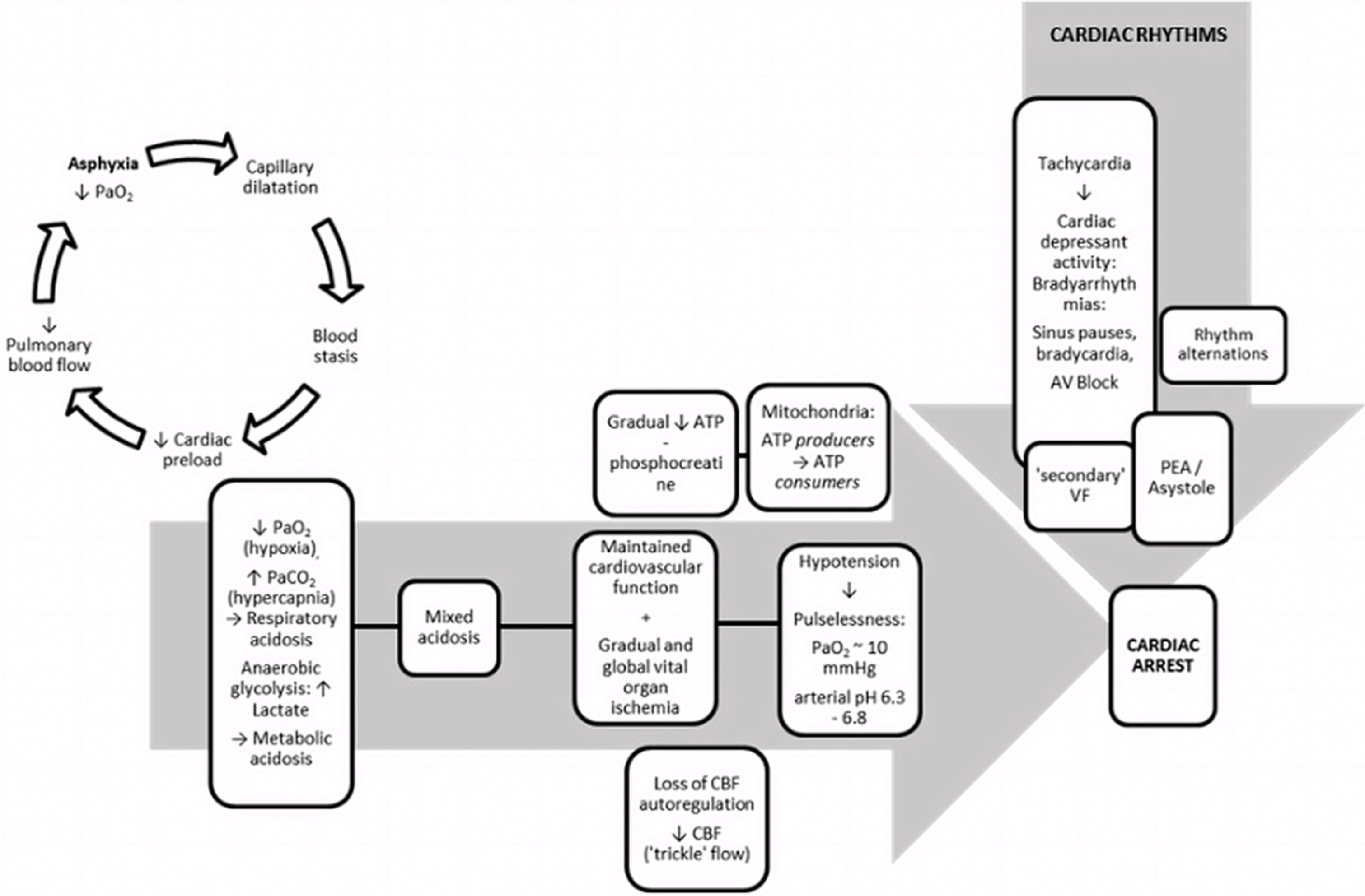
- **Cardiac vs. Respiratory Causes**

  While cardiac causes are predominant in adults, noncardiac or asphyxial causes far outnumber cardiac causes in younger populations, accounting for 20-40% of out-of-hospital cardiac arrests.

- **Hypovolemia and Shock**

  A third category of causes includes hypovolemia, hemorrhage, and circulatory shock, which can also lead to cardiac arrest, although with a lower incidence compared to cardiac and respiratory causes.

Varvarousis D, Varvarousi G, Iacovidou N, D'Aloja E, Gulati A, Xanthos T. The pathophysiologies of asphyxial vs dysrhythmic cardiac arrest: implications for resuscitation and post-event management. The American Journal of Emergency Medicine. 2015;33(9):1297-1304. doi:10.1016/j.ajem.2015.06.066



CARDIAC RHYTHMS
Asphyxia
↓ PaO_2
Capillary dilatation
Blood stasis
↓ Cardiac preload
↓ Pulmonary blood flow
↓ PaO_2 (hypoxia)
↑ $PaCO_2$ (hypercapnia) → Respiratory acidosis
Anaerobic glycolysis: ↑ Lactate
→ Metabolic acidosis
Mixed acidosis
Gradual ↓ ATP - phosphocreatine
Mitochondria: ATP *producers* → ATP *consumers*
Maintained cardiovascular function
+
Gradual and global vital organ ischemia
Loss of CBF autoregulation
↓ CBF ('trickle' flow)
Hypotension
↓
Pulselessness:
PaO_2 ~ 10 mmHg
arterial pH 6.3 - 6.8
Tachycardia
↓
Cardiac depressant activity: Bradyarrhythmias:
Sinus pauses, bradycardia,
AV Block
Rhythm alternations
'secondary' VF
PEA / Asystole
CARDIAC ARREST

Historical Insights into Ventricular Fibrillation Initiation

De Boer's Demonstration (1923)

De Boer showed that a single electric shock delivered late in systole to the frog's ventricle induced fibrillation.

Wiggers & Wegria's Findings (1940)

Wiggers & Wegria confirmed de Boer's results and demonstrated that the application of very short electrical shocks to a small area on the ventricle induced VF when the shocks fell during late systole, but never at any other phase of the heart cycle.

Wegria et al.'s Observation (1941)

Wegria et al. demonstrated that the vulnerable phase was of longer duration in premature ventricular complexes than in normal beats.

Moe et al.'s Electrographic Studies (1940s)

Moe et al. carried out detailed electrographic studies on the mechanism of reentry initiation by shocks applied during the vulnerable phase, indicating that repetitive impulses induced by the shock were accompanied by a progressive decrease in refractory period and an increase in conduction time.

Weiner & Rosenblueth's Theoretical Description (1946)

Weiner & Rosenblueth provided a theoretical description of the mechanisms of initiation of flutter and fibrillation, postulating that wave rotation around single or multiple obstacles was necessary to initiate and maintain both types of arrhythmia.

Mechanisms of Cardiac Vortex Induction

- ## Pinwheel Experiment

  Based on the protocol devised by Winfree, this involves establishing spatial gradients of stimulus intensity and refractoriness in a 2D excitable medium to induce mirror-image vortices around crossings of critical contours.

- ## Cross-Field Stimulation

  This protocol uses a conditioning stimulus S1 to initiate a plane wave, followed by a perpendicular stimulus S2 timed to interact with the S1 wave tail, forming a phase singularity and vortex.

- ## Vulnerable Domain and Phase

  The vulnerable domain is a specific temporal window just before complete recovery from a previous excitation, where a stimulus of the right size can induce vortices.

- ## Single Electrode Stimulation

  Reentry and fibrillation can also be induced by successive stimulation through a single unipolar or bipolar electrode, potentially due to the discrete and anisotropic nature of cardiac tissue.

Jalife J. Ventricular Fibrillation: Mechanisms of Initiation and Maintenance. *Annu Rev Physiol*. 2000;62(1):25-50. doi:10.1146/annurev.physiol.62.1.25

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Mechanisms of Spontaneous Reentrant Arrhythmias

- **Reentrant Activity**
  The most dangerous cardiac arrhythmias are the result of reentrant activity, where electrical waves rotate uninterruptedly and in a self-sustaining manner.
- **Circuit and Vortex Formation**
  Reentry may be initiated by the formation of circuits or vortices, brought about by the interaction of a propagating wave front with an obstacle in its path.
- **Triggered Activity**
  Reentry may also be initiated by a spontaneous premature beat, in the form of an early or delayed afterdepolarization, known as triggered activity.

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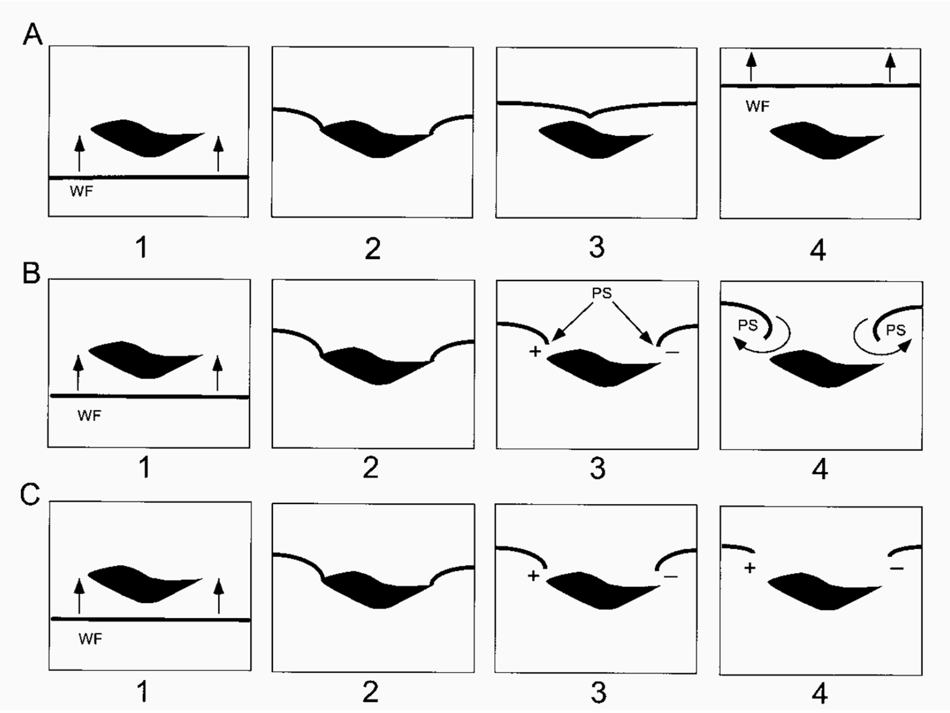

Formation of a wavebreak. In both panels, a wave front (WF) is seen moving upward to interact with an unexcitable obstacle, shown in black.

(A) Normal excitability, Upon reaching the obstacle, the wave front splits in two. The two newly formed wave fronts circumnavigate the obstacle without detaching from it and then merge into a com mon wave front that continues moving upward, apparently undisturbed by its interaction with the discontinuity.

(B) Lower excitability. The two broken wave fronts (wavelets) now detach from the obstacle, resulting in the formation of a singularity point (PS; also known as phase singularities) at the broken end of each wavelet. This leads to two counter rotating vortices.

(C) When excitability is too low, the two broken waves are unable to rotate and instead undergo decremental conduction

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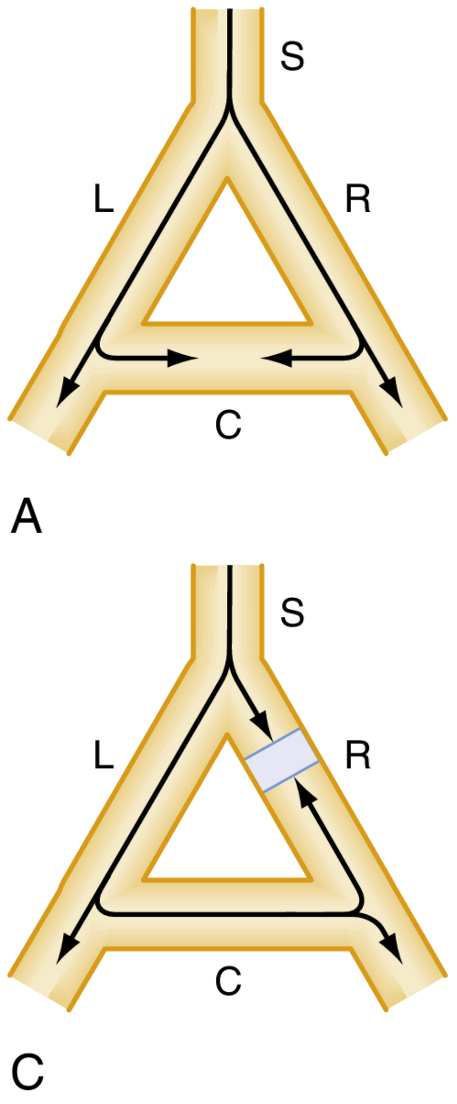

The role of unidirectional block in reentry.

A, An excitation wave traveling down a single bundle (S) of fibers continues down the left (L) and right (R) branches. The depolarization wave enters the connect ing branch (C) from both ends and the two wave fronts are extinguished when they collide.

B, The wave is blocked in the L and R branches.

C, A bidirectional block exists in the R branch.

D, A unidirectional block exists in the R branch. The antegrade impulse is blocked, but the retrograde impulse is conducted through and reenters the S bundle.

Koeppen BM. *Berne and Levy Physiology E-Book*. 8th ed. Elsevier; 2023.

Pathophysiology of Ischemic Ventricular Fibrillation

- **Coronary Vascular Events**

  Acute ischemia produces immediate electrical, mechanical, and biochemical dysfunction of cardiac muscle.

- **Myocardial Injury**

  Myocardial tissue that has healed after ischemic injury shows lasting electrophysiological abnormalities, including regional changes in transmembrane action potentials and refractory periods.

- **Autonomic Tone Changes**

  Changes in autonomic tone contribute to the pathophysiology of ischemic ventricular fibrillation.

- **Metabolic and Ionic Changes**

  Metabolic and ionic changes in the myocardium play a role in the development of ischemic ventricular fibrillation.

- **Reperfusion Arrhythmias**

  Reperfusion after transient ischemia can also lead to ventricular arrhythmias, including ventricular fibrillation. These reperfusion arrhythmias are related to the duration of ischemia to reperfusion.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Ventricular Fibrillation Types and Ischemic Arrhythmia Phases

Types of Ventricular Fibrillation

Two types of VF have been described in isolated perfused rabbit heart: Type I (Fast VF): Steep restitution of action potential duration, flat restitution of conduction velocity, and multiple wandering wavelengths. Type II (Slow VF): Flat restitution of action potential duration, broad restitution of conduction velocity, and spatiotemporal periodicity in the activation map.

Clinical Implications of VF Types

Type I VF induces acute global ischemia, which can flatten the restitution of action potential duration and decrease excitability, leading to conversion to Type II VF or coexistence of the two types. Type II VF is more likely to be lethal as it is unlikely to self-terminate. The window of opportunity to prevent Type I VF from converting to Type II is wider in patients with normal hearts.

Phases of Ischemic Arrhythmogenesis

The various phases of ventricular arrhythmia due to myocardial ischemia are: Phase Ia (2-10 min): Abundant ventricular arrhythmogenesis Phase Ib: Mechanism of arrhythmias differs from Phase Ia, and can be postponed by ischemic or pharmacological preconditioning

Phase II and III Ischemic Arrhythmias

Phase II (5-72 h): Arrhythmogenesis may be related to reperfusion of ischemic areas or the evolution of differing injury patterns. Phase III: Arrhythmogenesis is related to old healed myocardial infarcts and scars.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Mechanisms of Ventricular Fibrillation

Multiple Wavelet Hypothesis

New wavebreaks are themselves responsible for the continuation of ventricular fibrillation (VF).

Focal Source Hypothesis

Rapidly firing focal focus where the bulk of the myocardium cannot follow 1:1 conduction leading to wave breaks.

Role of Border Zone

The border zone of ischemia is the predominant site of wave fragmentation, while the central ischemic zone is depleted of wave breaks.

Effects of Acute Ischemia

Acute ischemia opens IKATP channels, causes acidosis and hypoxia, and increases extracellular potassium, leading to dispersion in repolarization and changes in excitability.

Calcium Transient Alternans

Calcium transient alternans in a localized ischemic area can create regional variations in action potential duration and refractoriness, contributing to arrhythmogenesis.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Theories of Ventricular Fibrillation

Focal Source Hypothesis

Most waves during VF do not complete full loops, lacking the appearance of complete reentry. Discrete stationary regions have been identified, with evidence of conduction block between them. However, the postulated mother rotor within the myocardium has never been demonstrated experimentally.

Multiple Wavelet Hypothesis

Wavebreak is a primary phenomenon that perpetuates fibrillation, rather than an epiphenomenon. Mapping the ventricle reveals multiple wavelets appearing in random fashion, with the wavefront corresponding to the upstroke and the waveback to the repolarization.

Two Types of VF

Fast type VF is associated with steep action potential duration restitution and wandering wavelets, while slow type VF is associated with flat restitution. Both excitability and action potential duration restitution are important factors in the maintenance of VF.

Factors Causing Wavebreak

Anatomic and electrophysiological obstacles, often aggravated by channelopathies, cardiomyopathies, myocardial ischemia, and other cardiac diseases, can lead to dispersion of refractoriness and cause wavebreak. Wavebreak can also occur due to spontaneous wavelength oscillations.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Focal Source Hypothesis in Ventricular Fibrillation

Waves during VF

Most waves during ventricular fibrillation (VF) do not complete full loops, lacking the appearance of complete reentry.

Discrete Stationary Regions

Discrete stationary regions have been identified within the myocardium during VF.

Conduction Block

There is evidence of conduction block between the stationary regions identified during VF.

Mother Rotor

The postulated mother rotor, presumed to reside within the myocardium, has never been demonstrated experimentally.

Multiple Wavelets and Wavebreak

The multiple wavelets and/or wavebreak become an epiphenomenon in the focal source hypothesis.

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The Multiple Wavelet Hypothesis

- **Wavebreak as a Primary Phenomenon**

  The multiple wavelet hypothesis proposes that the wavebreak is a primary phenomenon, rather than an epiphenomenon that perpetuates fibrillation. This has been directly demonstrated through experimental mapping of the ventricle with multi-electrode plaques.

- **Observing Multiple Wavelets**

  The experiments have observed the predicted multiple wavelets appearing in a random fashion, with the wavefront corresponding to the upstroke of the cardiac action potential and the waveback corresponding to the repolarization.

- **Wavelength and Its Components**

  The wavelength corresponds to the product of conduction velocity and action potential duration. In a normal beat, this wave travels through the atrium and the ventricle without hindrance.

- **Wavebreak and Phase Singularities**

  When the wave breaks, it leaves behind its broken ends called phase singularities, spiral wave tips, or scroll wave tips. These tend to form waves that are often called rotors or leading circles.

- **The Resulting Wavelets**

  The big wave breaks and produces smaller wavelets that collide and push each other, perpetuating the fibrillation process.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Mechanisms of Wavebreak and Arrhythmia

Factors Causing Wavebreak

Anatomical and electrophysiological obstacles, often aggravated by channelopathies, cardiomyopathies, myocardial ischemia, and other cardiac diseases.

Dispersion of Refractoriness

The major force behind causing wavebreak during fibrillation or in generating the focal source.

Spontaneous Wavelength Oscillations

Can lead to wavebreak in the absence of disease states, due to shortening of action potential duration in response to increased heart rate.

Reentry Models

Various models of reentry have been described, with factors causing wavebreak being entwined with the underlying anatomical and electrophysiological factors.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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Factors Influencing Ventricular Fibrillation

Myocardial Ischemia as a Trigger

Myocardial ischemia can initiate triggers in the form of premature ventricular contractions and affect parameters that influence the maintenance of ventricular fibrillation (VF).

Reentry Models

Various models of reentry have been described as a mechanism for the initiation and maintenance of VF.

Wavebreak Factors

Factors that cause wavebreak are often aggravated by channelopathies, cardiomyopathies, myocardial ischemia, and other cardiac diseases. Dispersion of refractoriness is a major force behind causing wavebreak during fibrillation or in generating the focal source.

Spontaneous Wavelength Oscillations

Wavebreak can also occur in the absence of disease states, due to spontaneous wavelength oscillations caused by shortening of action potential duration in response to an increase in heart rate.

Luqman N, Sung RJ, Wang CL, Kuo CT. Myocardial ischemia and ventricular fibrillation: pathophysiology and clinical implications. Int J Cardiol. 2007;119(3):283-290. doi:10.1016/j.ijcard.2006.09.016

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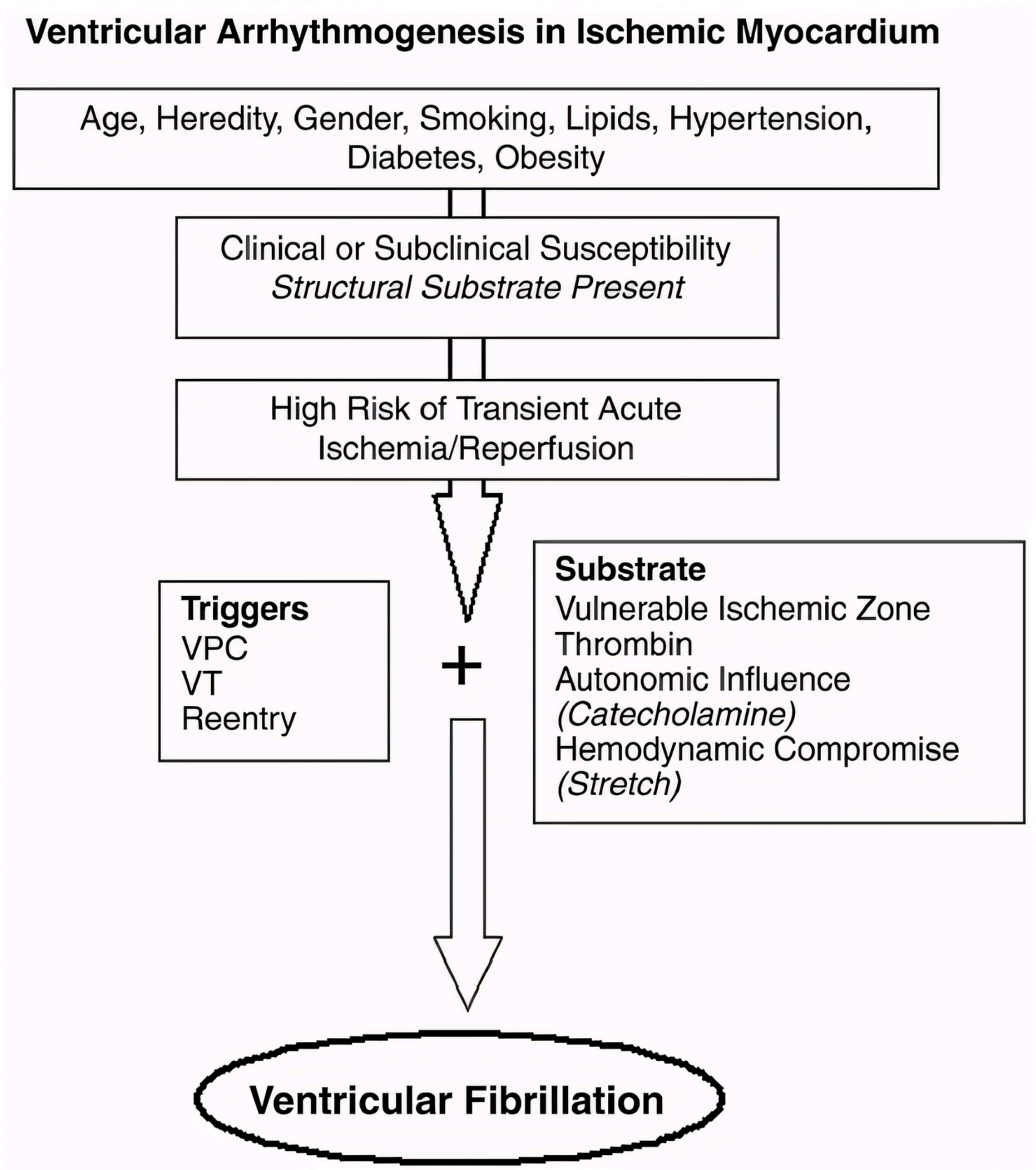

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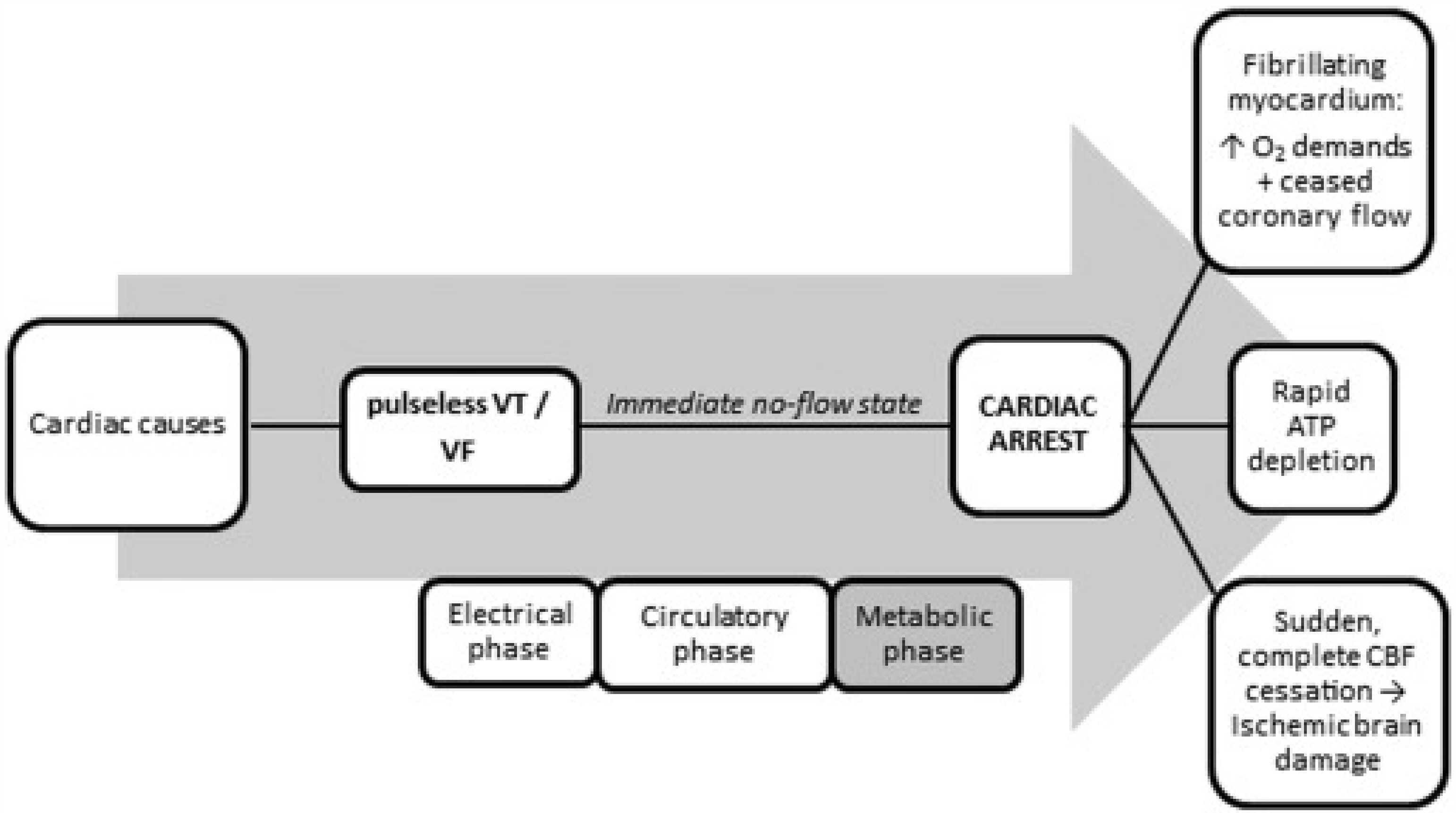
Cardiac causes
pulseless VT / VF
Immediate no-flow state
CARDIAC ARREST
Fibrillating myocardium: ↑ O_2 demands + ceased coronary flow
Rapid ATP depletion
Sudden, complete CBF cessation → Ischemic brain damage
Electrical phase
Circulatory phase
Metabolic phase

Section 3

Post-cardiac arrest syndrome

Hypoxic Ischemic Brain Injury after Cardiac Arrest

- **Dismal Outcomes**

  Only 10% of patients survive until hospital discharge, and 5% experience full neurologic recovery

- **Hypoxic Ischemic Brain Injury (HIBI)**

  Primary cause of death in 68% of inpatient cardiac arrest and 23% of out-of-hospital cardiac arrest, leading to significant neurological disability

- **Quality of Life and Psychiatric Comorbidities**

  HIBI survivors experience a high prevalence of depression, anxiety, and post-traumatic stress disorder, greatly impacting quality of life

- **Pathophysiology of HIBI**

  A 'two-hit' model: primary injury from immediate cessation of cerebral oxygen delivery during cardiac arrest and secondary injury after resuscitation

- **Cerebral Autoregulation**

  Optimizing the balance between cerebral oxygen delivery and use is critical, requiring a detailed understanding of cerebral physiologic perturbations

- **Hemoglobin, Carbon Dioxide, and Cerebral Edema**

  Advances in understanding the role of these factors in HIBI pathophysiology are essential for improving outcomes

- **Normobaric Hyperoxia and Targeted Temperature Management**

  Exploring the effects of these interventions on HIBI pathophysiology may lead to improved outcomes

Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a “two-hit” model. Critical Care. 2017;21(1):90. doi:10.1186/s13054-017-1670-9

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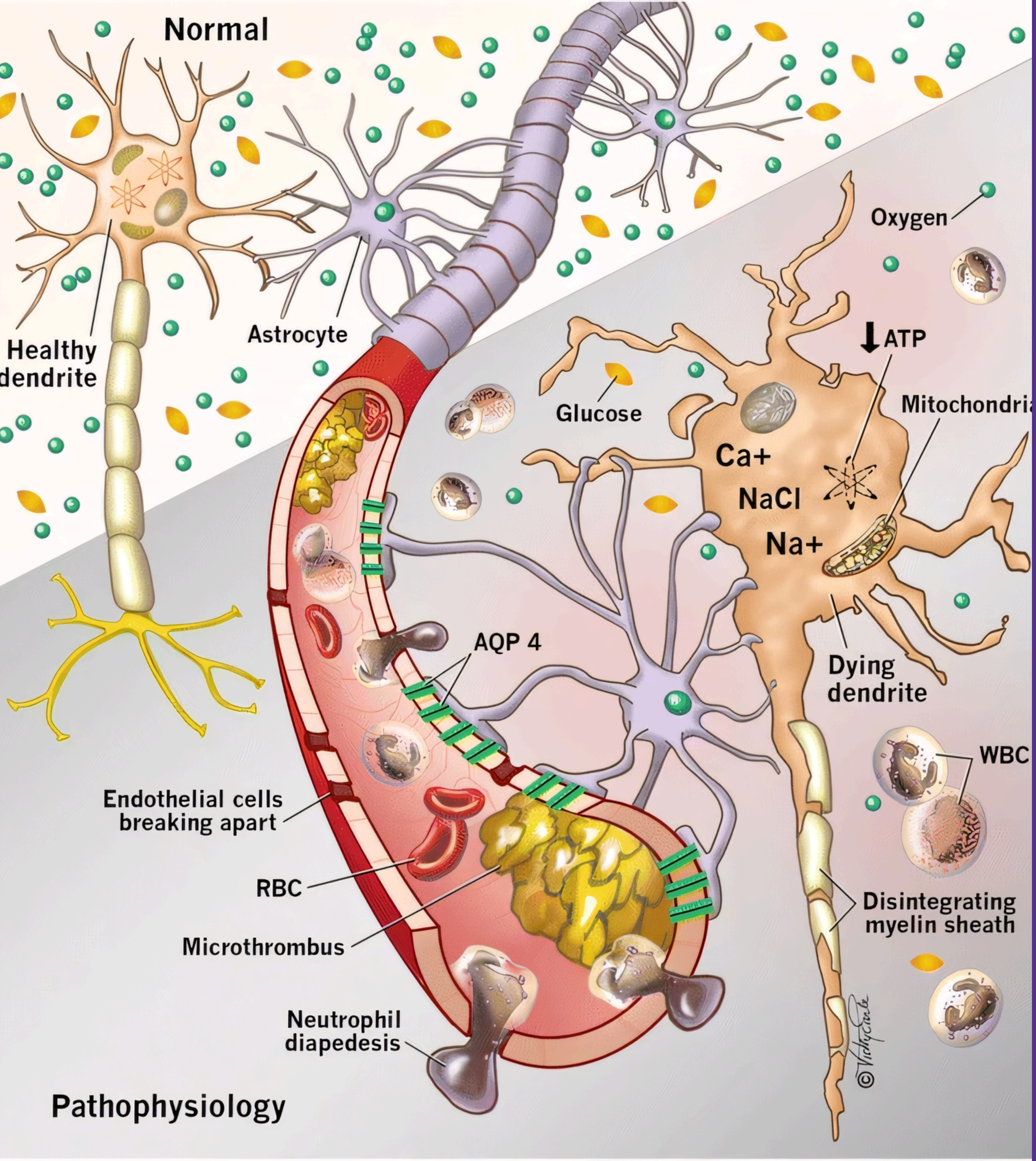

A schematic demonstrating the various microvascular and cellular pathophysiologic consequences which occur during the primary and secondary injury in hypoxic ischemic brain injury (HIBI).

Decreased cerebral oxygen delivery manifests as reduced neuronal aerobic metabolism, causing reduced cellular adenosine triphosphate (ATP) production. Intracellular calcium accumulation leads to mitochondrial toxicity and further reduced ATP production.

Inability to sustain cellular respiration results in cell death and apoptosis. Additionally, in the microvasculature, endothelial dysfunction leads to a porous blood-brain barrier, formation of cerebral edema, formation of microthrombi and limitation of cerebral blood flow with exacerbation of cellular ischemia. AQP 4 Aquaporin-4, RBC Red blood cells, WBC White blood cells

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Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a "two-hit" model. *Critical Care*. 2017;21(1):90. doi:10.1186/s13054-017-1670-9

Pathophysiology of Hypoxic-Ischemic Brain Injury

- **Cessation of Cerebral Oxygen Delivery**
  During cardiac arrest, cessation of cerebral oxygen delivery (CDO2) occurs, leading to neuron ischemia and cell death within minutes.

- **Cerebral Metabolic Demands**
  The cerebrum consumes 20% to 25% of cardiac output to maintain function. The brain is devoid of nutrient stores, leading to neuroglycopenia and metabolic crisis within minutes after cardiac arrest.

- **Cellular Cascade of Injury**
  Decreased CDO2 leads to cessation of energy-dependent ion channel function, intracellular Na+ accumulation, anaerobic metabolism, cerebral lactate accumulation, intracellular acidosis, and activation of lytic enzymes and apoptosis.

- **Early Loss of Neurologic Function**
  Loss of neurologic function is manifested by a decreased level of consciousness, which occurs within 20 seconds of the onset of ventricular fibrillation. Isoelectric electroencephalography has been observed within 15-30 seconds of the onset of cerebral ischemia.

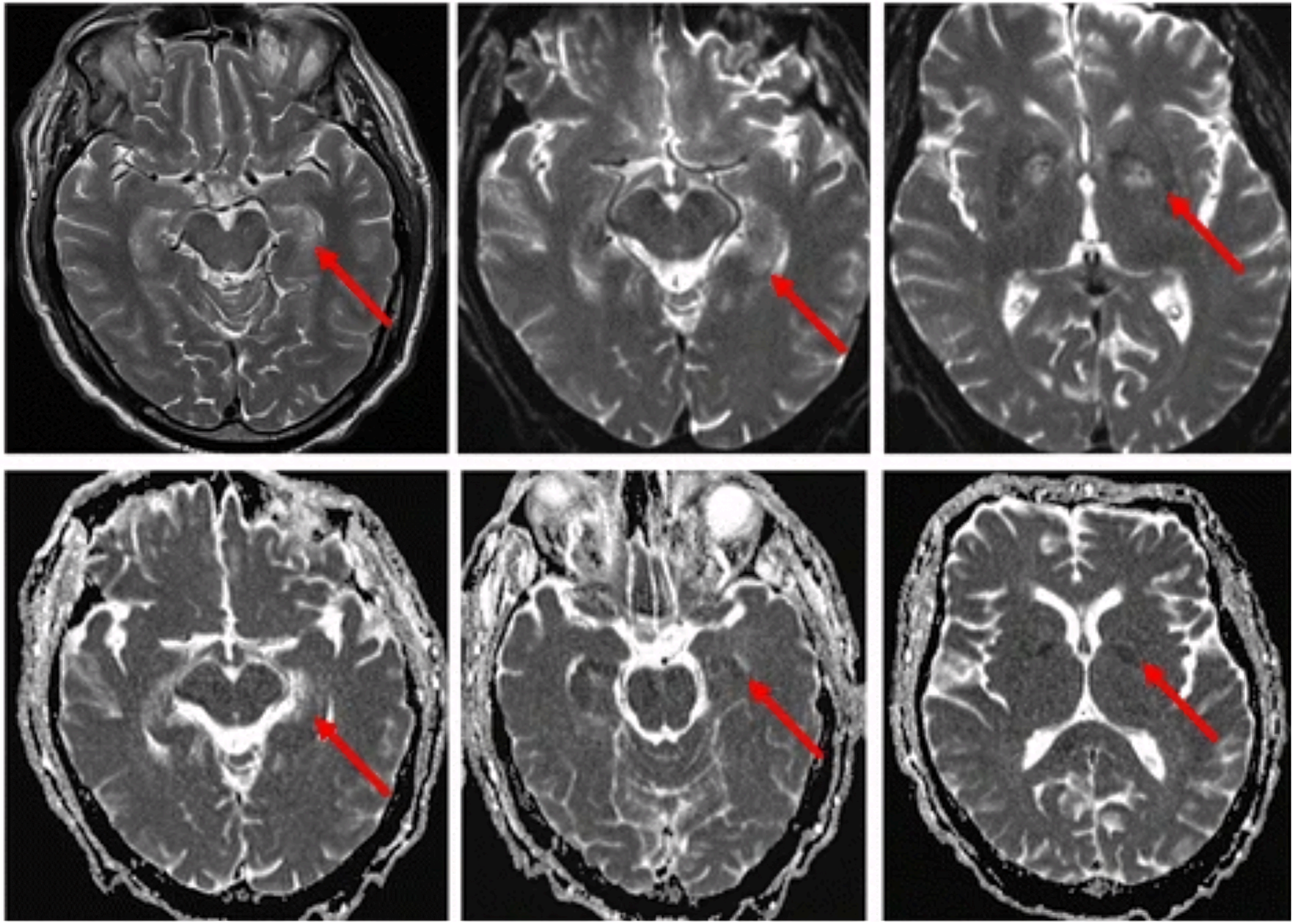
- **Primary and Secondary Injury**
  While primary injury causes substantial neuronal loss, the ensuing postresuscitation additive cerebral injury accounts for significant cerebral ischemia and cellular death. Secondary injury and physiologic determinants are targets of therapeutic interventions after hypoxic-ischemic brain injury.

Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a “two-hit” model. Critical Care. 2017;21(1):90. doi:10.1186/s13054-017-1670-9

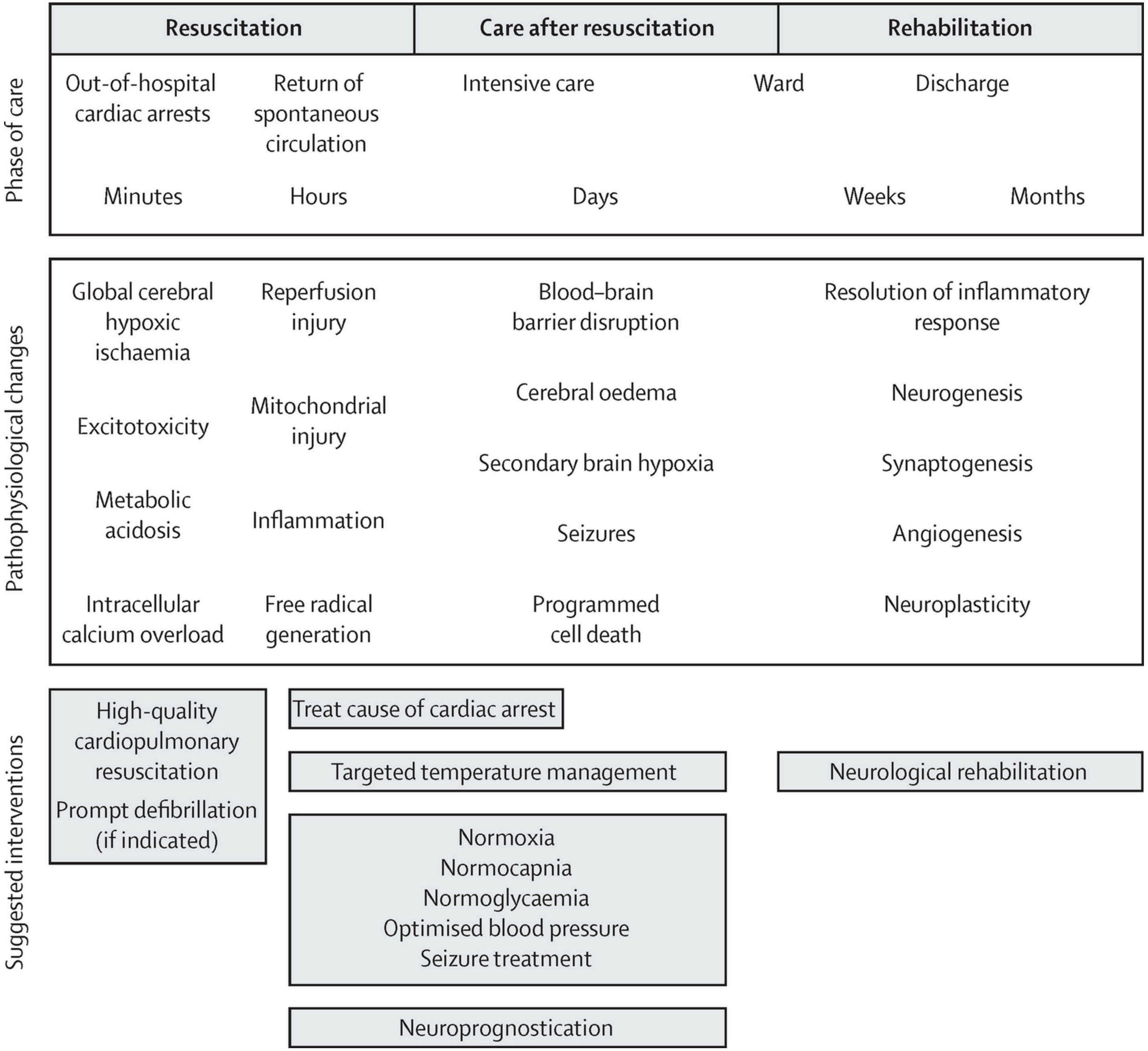
Secondary injury

Pathophysiology	Mechanisms	Consequences
Microvascular dysfunction	Microthrombi, cerebral vasoconstriction, blood-brain barrier disruption	Increased cerebrovascular resistance, decreased CBF, decreased cerebral O2 delivery, vasogenic cerebral edema
Cerebral edema	Vasogenic cerebral edema, cytotoxic cerebral edema	Increased ICP and decreased CPP, decreased CBF, herniation, brain death
Anemia	Decreased arterial oxygen content	Cerebral ischemia
Impaired autoregulation	Narrowed and right-shifted autoregulation	Pressure passive cerebral hemodynamics, cerebral ischemia and hyperemia
Carbon dioxide	Hypocapnia-induced vasoconstriction, hypercapnia-induced vasodilation	Decreased CBF, cerebral ischemia, increased ICP, decreased CPP, decreased CBF
Hyperoxia	Increased O2 free radicals	Neuronal cell dysfunction and cell death
Hyperthermia	Increased CMRO2, decreased seizure threshold, induction of apoptosis	Neuronal cell metabolic crisis, cell death, nonconvulsive seizures, increased CMRO2, neuronal cell death

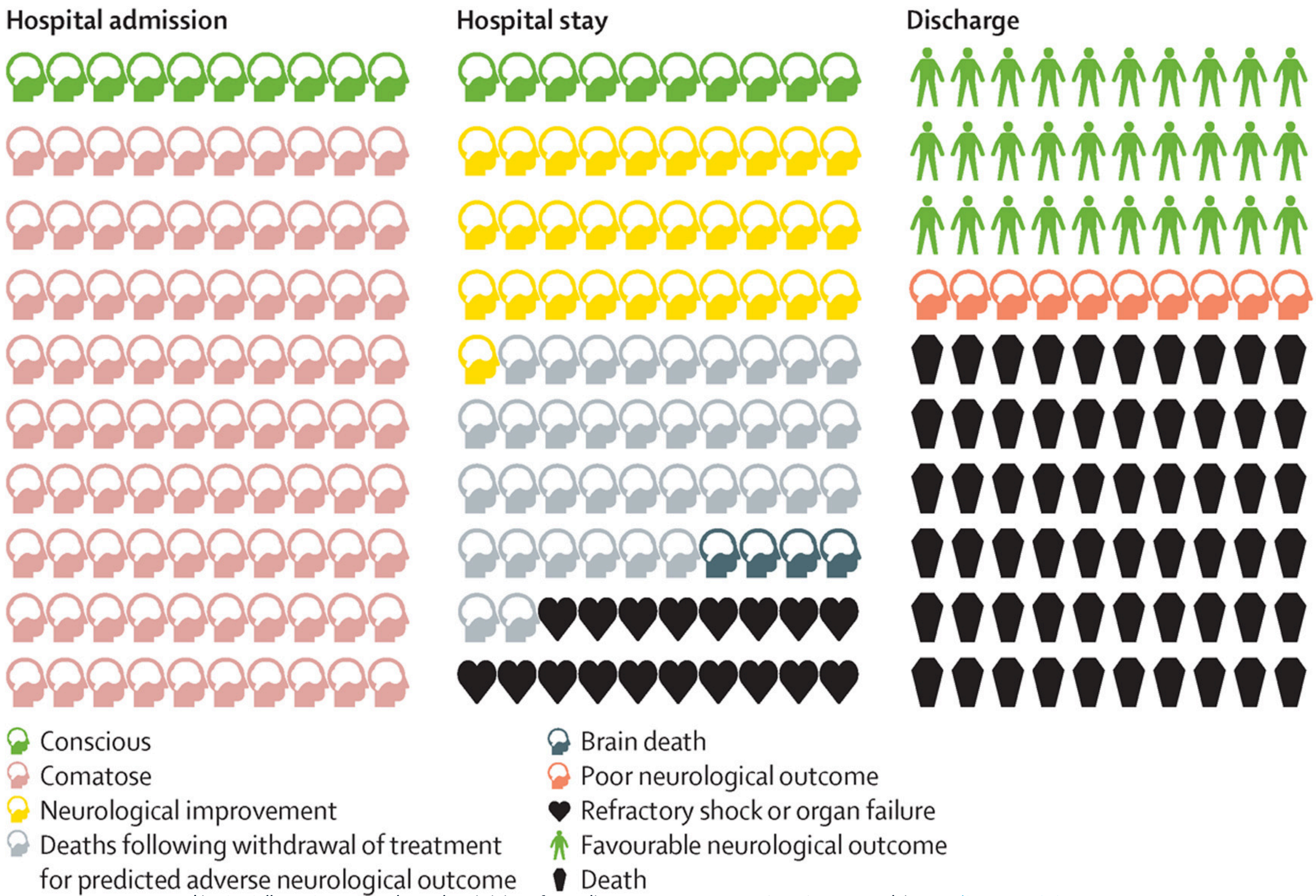
Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a "two-hit" model. Critical Care. 2017;21(1):90. doi:10.1186/s13054-017-1670-9



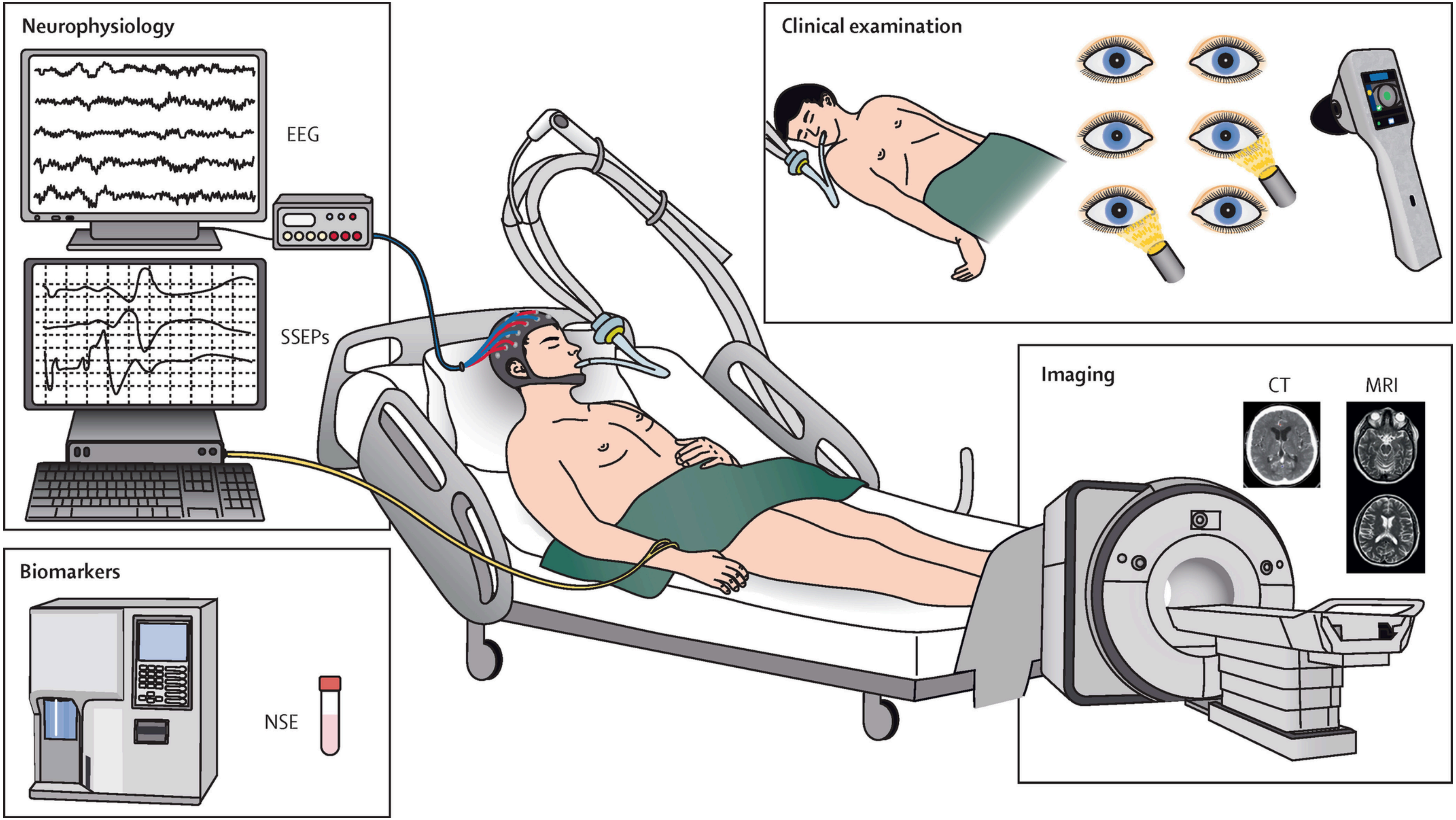
Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a "two-hit" model. Critical Care. 2017;21(1):90. doi:10.1186/s13054-017-1670-9



Perkins GD, Callaway CW, Haywood K, et al. Brain injury after cardiac arrest. Lancet. 2021;398(10307):1269-1278. doi:10.1016/S0140-6736(21)00953-3



Perkins GD, Callaway CW, Haywood K, et al. Brain injury after cardiac arrest. Lancet. 2021;398(10307):1269-1278. doi:10.1016/S0140-6736(21)00953-3



Perkins GD, Callaway CW, Haywood K, et al. Brain injury after cardiac arrest. Lancet. 2021;398(10307):1269-1278. doi:10.1016/S0140-6736(21)00953-3

Cerebral Performance Category (CPC)

Category	Description
CPC 1	Conscious, alert, able to work and lead a normal life. May have minor psychologic or neurologic deficits (mild dysphasia, non-incapacitating hemiparesis, or minor cranial nerve abnormalities).
CPC 2	Conscious. Sufficient cerebral function for part-time work in sheltered environment or independent activities of daily life (dress, travel by public transportation, food preparation). May have hemiplegia, seizures, ataxia, dysarthria, or permanent memory or mental changes.
CPC 3	Conscious. Dependent on others for daily support (in an institution or at home with exceptional family effort). Has at least limited cognition. This category includes a wide range of cerebral abnormalities, from patients who are ambulatory but have severe memory disturbances or dementia precluding independent existence, to those who are paralyzed and can communicate only with their eyes, as in the "locked in" syndrome.
CPC 4	Unconscious. Unaware of surroundings, no cognition. No verbal and/or psychologic interaction with environment.
CPC 5	Brain dead, circulation preserved.

Rittenberger JC, Raina K, Holm MB, Kim YJ, Callaway CW. Association between Cerebral Performance Category, Modified Rankin Scale, and discharge disposition after cardiac arrest. *Resuscitation*. 2011;82(8):1036-1040. doi:10.1016/j.resuscitation.2011.03.034

Section 4

BLSD overview

ADULT BASIC LIFE SUPPORT
KEY MESSAGES

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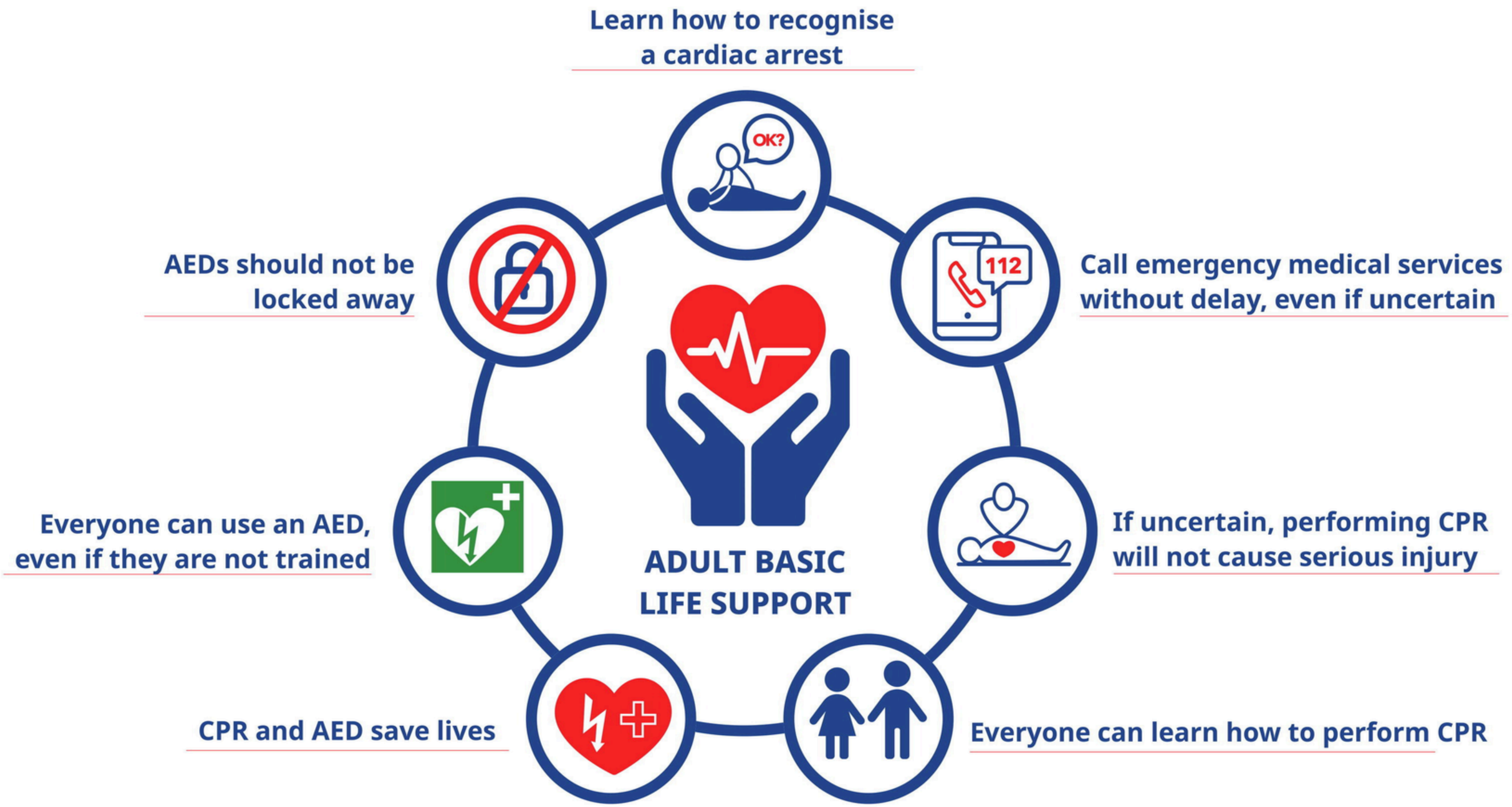

Smyth MA, Goor S van, Hansen CM, et al. European Resuscitation Council Guidelines 2025 Adult Basic Life Support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771

BLS with AED – 2 Rescuers

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SEQUENCE / ACTION	TECHNICAL DESCRIPTION
SAFETY	• Make sure that you, the victim and bystanders are safe
RESPONSE Check for a response	• Shake the victim gently by the shoulders and ask loudly: ***"Are you all right?"***
ALERT EMERGENCY SERVICES	• If victim is unresponsive, ask a helper to call the emergency medical services or call them yourself • Stay with the victim if possible • Activate the speaker function or hands-free option on the telephone so that you can start CPR whilst talking to the dispatcher
AIRWAY Open the airway	• If there is no response, position the victim on their back • With your hand on the forehead and your fingertips under the point of the chin, gently tilt the victim's head backwards, lifting the chin to open the airway
BREATHING Look, listen and feel for breathing	• Look, listen and feel for breathing for no more than 10 seconds • A victim who is barely breathing, or taking infrequent, slow and noisy gasps, is not breathing normally
SEND FOR AED Send someone to get an AED	• Send someone to find and bring back an AED, if available • If you are on your own, fetch an AED only if you can get and apply it within one minute; otherwise, start CPR immediately
CIRCULATION Start chest compressions	• Kneel by the side of the victim • Place the heel of one hand in the centre of the victim's chest - this is the lower half of the victim's breastbone (sternum) • Place the heel of your other hand on top of the first hand and interlock your fingers • Keep your arms straight • Position yourself vertically above the victim's chest and press down on the sternum at least 5 cm (but not more than 6 cm) • After each compression, release all the pressure on the chest without losing contact between your hand and the chest • Repeat at a rate of 100-120 min^{-1}
COMPRESSION-ONLY CPR	• **If you are untrained, or unable to give rescue breaths,** give chest-compression-only CPR (continuous compressions at a rate of 100-120 min^{-1})

COMBINE RESCUE BREATHING WITH CHEST COMPRESSIONS	• **If you are trained to do so**, after 30 compressions, open the airway again, using head tilt and chin lift • Pinch the soft part of the nose closed, using your index finger and thumb of your hand on the forehead • Allow the victim's mouth to open, but maintain chin lift • Take a normal breath and place your lips around the victim's mouth, making sure that you have an airtight seal • Blow steadily into the mouth whilst watching for the chest to rise, taking about 1 second as in normal breathing. This is an effective rescue breath • Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out • Take another normal breath and blow into the victim's mouth once more to achieve a total of two rescue breaths • Do not interrupt compressions by more than 10 seconds to deliver the two breaths, even if one or both are not effective • Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions • Continue with chest compressions and rescue breaths in a 30:2 ratio
WHEN AED ARRIVES Switch on the AED and attach the electrode pads	• As soon as the AED arrives, switch it on and attach the electrode pads to the victim's bare chest • If more than one rescuer is present, CPR should be continued whilst the electrode pads are being attached to the chest
FOLLOW THE SPOKEN/ VISUAL DIRECTIONS	• Follow the spoken and visual directions given by the AED • **If a shock is advised**, ensure that neither you nor anyone else is touching the victim • Push the shock button as directed • Then **immediately** resume CPR as directed by the AED
IF NO SHOCK IS ADVISED Continue CPR	• **If no shock is advised,** immediately resume CPR and continue as directed by the AED

IF NO AED IS AVAILABLE
Continue CPR
	• If no AED is available, **or** whilst waiting for one to arrive, continue CPR
	• Do not interrupt resuscitation until:
	  • A healthcare professional tells you to stop OR
	  • The victim is definitely waking up, moving, opening eyes, and breathing normally OR
	  • You become exhausted
	• It is rare for CPR alone to restart the heart. Unless you are certain that the victim has recovered, continue CPR
	• Signs that the victim has recovered
	  • Waking-up
	  • Moving
	  • Opening eyes
	  • Breathing normally

Smyth MA, Goor S van, Hansen CM, et al. European Resuscitation Council Guidelines 2025 Adult Basic Life Support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771

Provider Course CPR/AED

Cardiopulmonary Resuscitation with Automated External Defibrillator

Objectives

- At the end of this course participants should be able to demonstrate:
  - How to assess the collapsed victim
  - How to perform chest compression and rescue breathing (CPR)
  - How to operate an automated external defibrillator safely
  - How to place an unconscious breathing victim in the recovery position

Background

- In Europe every 45 seconds a cardiac arrest takes place
- Bystander CPR is a vital intervention before arrival of emergency services
- Early resuscitation and prompt defibrillation (within 1-2 minutes) can result in >60% survival

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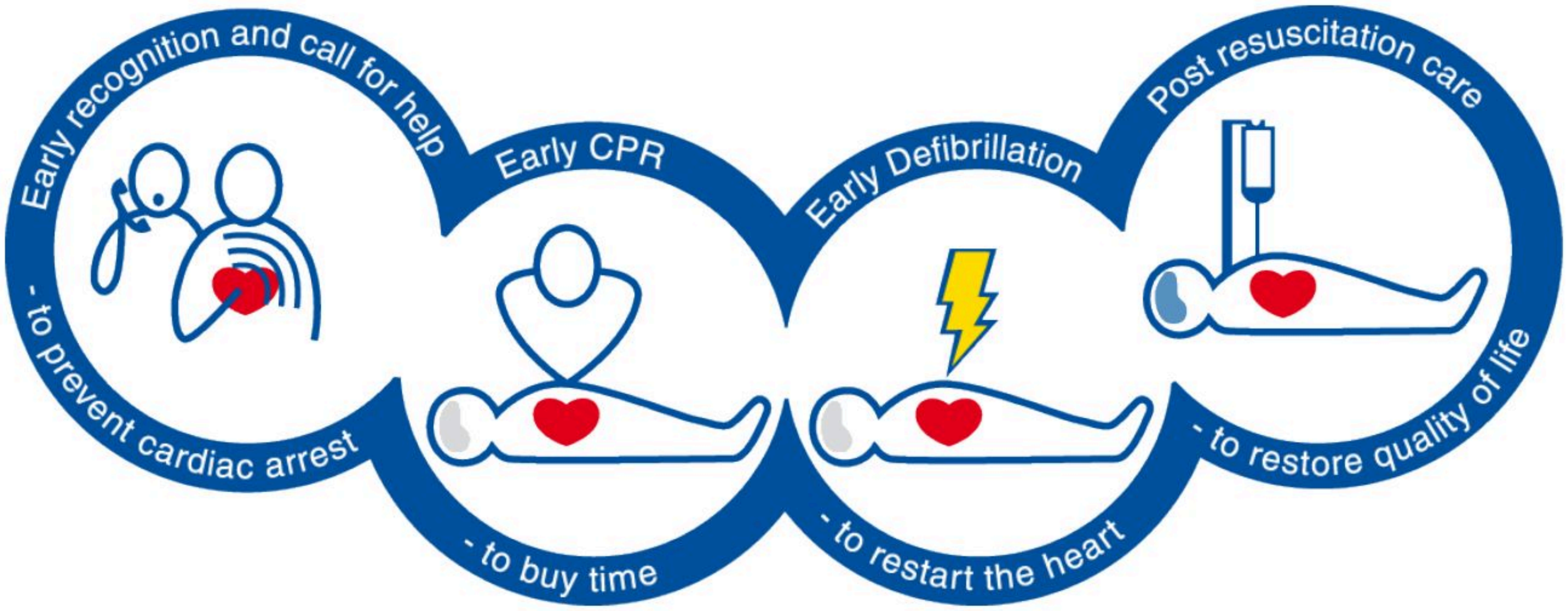
Chain of survival



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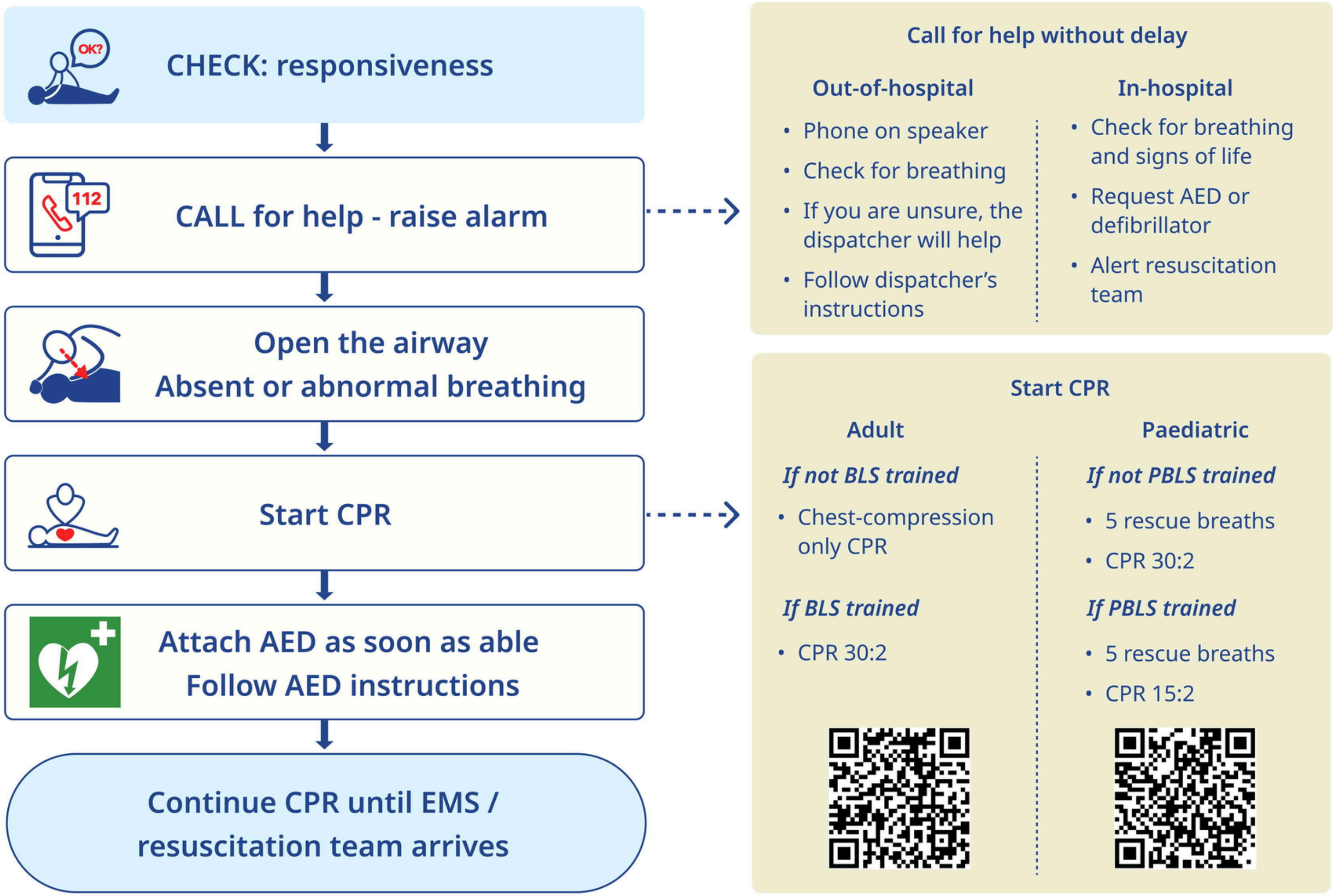

Smyth MA, Goor S van, Hansen CM, et al. European Resuscitation Council Guidelines 2025 Adult Basic Life Support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771

Approach safely

Check response

Call 112

Open airway

Check breathing

30 chest compressions

2 rescue breaths

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Approach safely

Approach safely

Check response

Call 112

Open airway

Check breathing

30 chest compressions

2 rescue breaths

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Check response

Shake shoulders gently

Ask “Are you all right?”

If he responds

- Leave as you find him.
- Find out what is wrong.
- Reassess regularly.

Check response

- Approach safely
- **Check response**
- Call 112
- Open airway
- Check breathing
- 30 chest compressions
- 2 rescue breaths

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Call 112

- Approach safely
- Check response
- **Call 112**
- Open airway
- Check breathing
- 30 chest compressions
- 2 rescue breaths

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Check

- Safety
- Responsiveness

Unresponsive

Call

- Call EMS without delay
- Use phone on speaker
- Check for breathing
- Follow the dispatcher's instructions

112

Absent or abnormal breathing

CPR & AED

- Start CPR
- Attach AED as soon as one is available
- Follow AED instructions
- If you are unsure, the dispatcher will help

3 steps to save a life

Smyth MA, Goor S van, Hansen CM, et al. European Resuscitation Council Guidelines 2025 Adult Basic Life Support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771

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Call 112

ABSENT OR ABNORMAL BREATHING
Alert emergency services

- If breathing is absent or abnormal, ask a helper to call the emergency services or call them yourself
- Stay with the victim if possible
- Activate the speaker function or hands-free option on the telephone so that you can start CPR whilst talking to the dispatcher

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Shout for help

SEND FOR AED
Send someone to get an AED

AED

- Send someone to find and bring back an AED if available
- If you are on your own, **DO NOT** leave the victim, but start CPR

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Open airway

- Approach safely
- Check response
- Call 112
- **Open airway**
- Check breathing
- 30 chest compressions
- 2 rescue breaths

EUROPEAN RESUSCITATION COUNCIL

Check breathing

- Approach safely
- Check response
- Call 112
- Open airway
- **Check breathing**
- 30 chest compressions
- 2 rescue breaths

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Check breathing

- Look, listen and feel for NORMAL breathing

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Check breathing

BREATHING
Look, listen and feel for breathing

- Look, listen and feel for breathing for **no more** than 10 seconds
- A victim who is barely breathing, or taking infrequent, slow and noisy gasps, is not breathing **normally**

Abnormal breathing

- Occurs shortly after the heart stops in up to 40% of cardiac arrests
- Described as barely, heavy, noisy or gasping breathing
- Recognise as a sign of cardiac arrest

Abnormal breathing

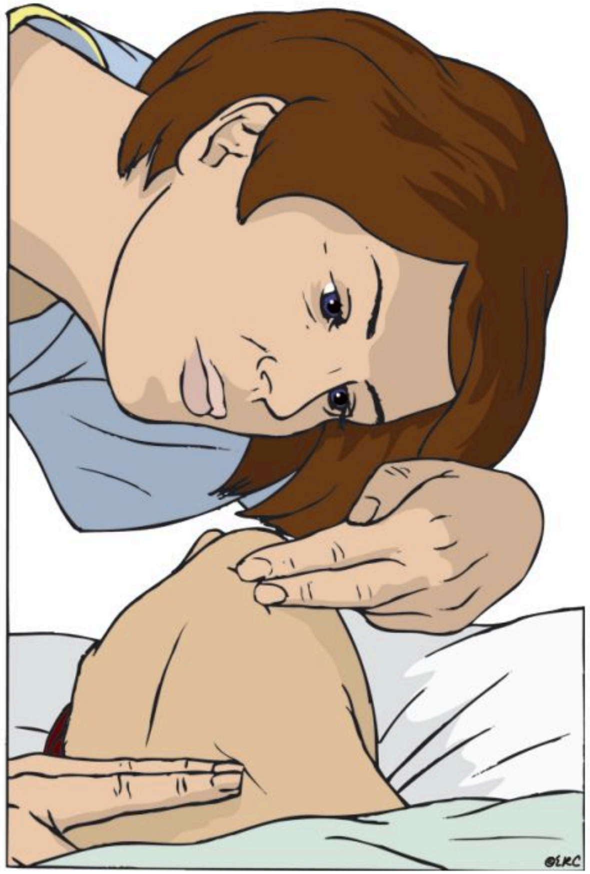
- Start CPR in any unresponsive person with absent or abnormal breathing.
- A short period of seizure-like movements can occur at the start of cardiac arrest. Assess the person after the seizure has stopped: if unresponsive and with absent or abnormal breathing, start CPR.

Recognising cardiac arrest

- Ensure it is safe to approach.
- Suspect cardiac arrest in any person who is unresponsive.
- Call your local emergency number without delay.
- Assess their breathing while you wait for the call to be answered.
- Slow, laboured breathing, as well as other abnormal patterns such as agonal gasping or panting, must be recognised as signs of cardiac arrest.
- A short period of seizure-like activity may occur at the onset of cardiac arrest. Once the seizure stops, assess breathing.
- If any person is unresponsive with abnormal breathing, cardiac arrest should be assumed.
- If you are uncertain, the call-taker will be able to assist you.
- If there is any doubt, assume cardiac arrest and start CPR.

Smyth MA, Goor S van, Hansen CM, et al. European Resuscitation Council Guidelines 2025 Adult Basic Life Support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771

CAROTID PULSE CHECK



- Not recommended for lay rescuers
- Use the index and middle fingers in the groove on *one side* of the neck *only*
- Check for breathing and carotid pulse at the same time
  - Extend neck
  - No more than 10 seconds

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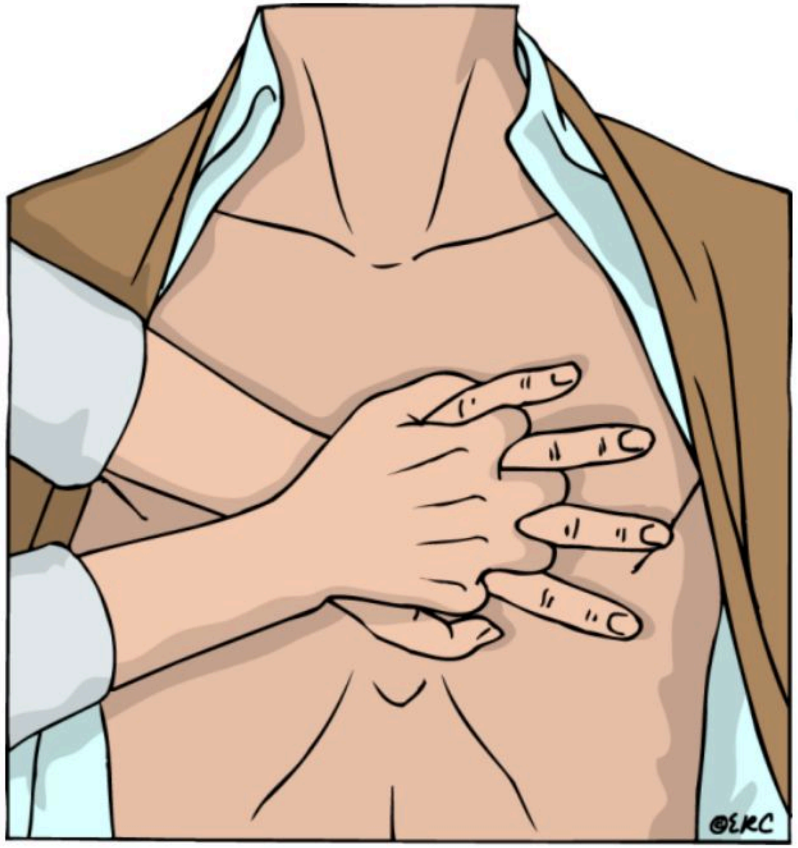
CAROTID PULSE CHECK

- **Pulse absent or <u>unsure</u>**
  - Start chest compressions
  - Continue 30 chest compressions : 2 rescue breaths
  - Do not re-check for carotid pulse or signs of life
- **Pulse definitely <u>present</u>**
  - Give rescue breaths on their own at a rate of 10 per minute
  - Re-check for carotid pulse or signs of life after 1 minute

30 Chest compressions

- Approach safely
- Check response
- Call 112
- Open airway
- Check breathing
- **30 chest compressions**
- 2 rescue breaths

Chest compressions



- Place the heel of one hand in the centre of the chest
- Place other hand on top
- Interlock fingers or avoid putting pressure on the side of the chest
- Compress the chest
  - Rate 100 – 120 min^{-1}
  - Depth 5 – 6 cm
  - Equal compression : relaxation
- When possible change CPR operator every 2 min

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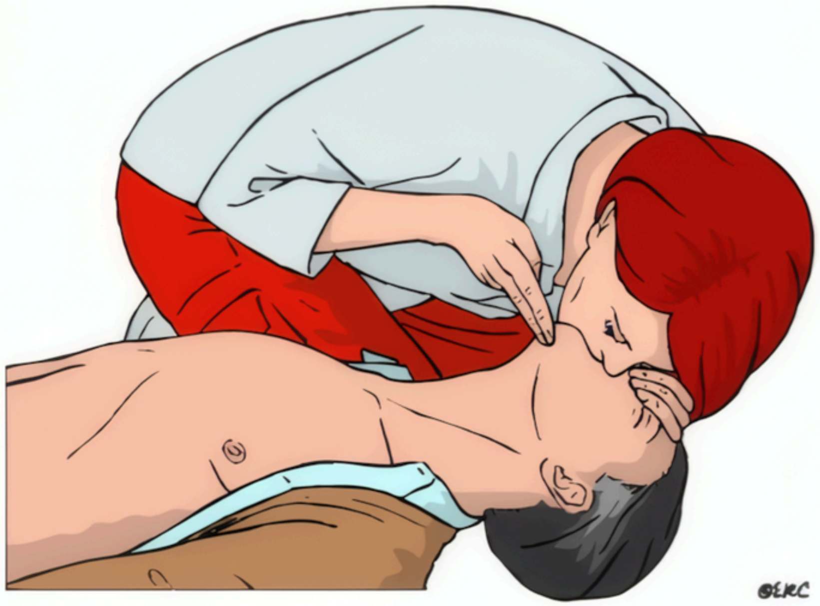
Chest compressions

CIRCULATION
Start chest compressions

- Kneel by the side of the victim
- Place the heel of one hand in the centre of the victim's chest - this is the lower half of the victim's breastbone (sternum)
- Place the heel of your other hand on top of the first hand and interlock your fingers
- Keep your arms straight
- Position yourself vertically above the victim's chest and press down on the sternum at least 5 cm (but not more than 6 cm)
- After each compression, release all the pressure on the chest without losing contact between your hands and the sternum
- Repeat at a rate of 100-120 min-1

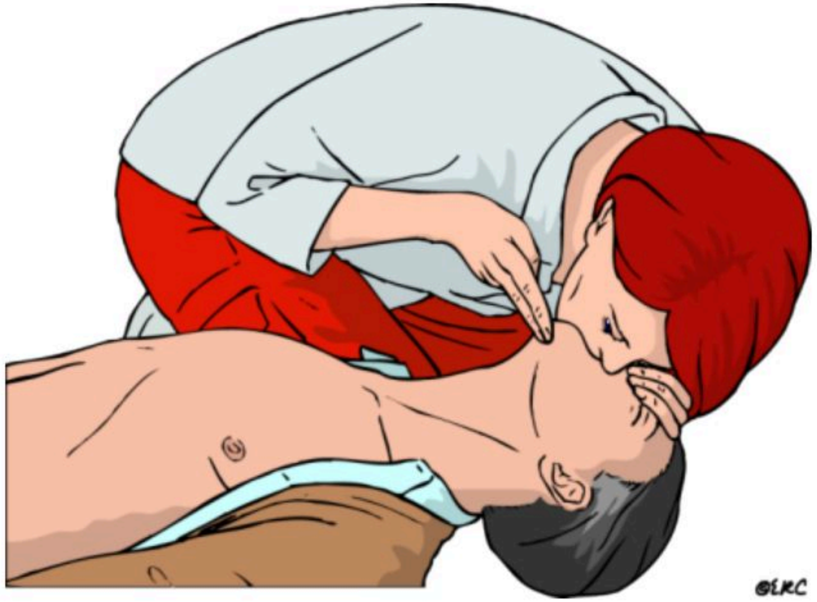
2 Rescue breaths



- Approach safely
- Check response
- Call 112
- Open airway
- Check breathing
- 30 chest compressions
- **2 rescue breaths**

Rescue breaths

- Pinch the nose
- Take a normal breath
- Place lips over mouth
- Blow until the chest rises
- Take about 1 second
- Allow chest to fall
- Repeat



GENERAL POINTS ON USING FACE-MASKS

- Extend neck fully
- Create a good seal between mask and victim’s mouth and nose
- Deliver each breath over 1 second
- Blow just enough to make chest rise and fall as in normal breathing
  - Do not over-ventilate
- Combine 30 chest compressions with 2 rescue breaths

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USE OF OXYGEN

- Supplemental oxygen can be used in cardiac arrest if available
- Given via a bag-valve mask or pocket-mask
- **Give as <u>much</u> as possible as <u>soon</u> as possible:** *Flow rates of 10-15L/min*

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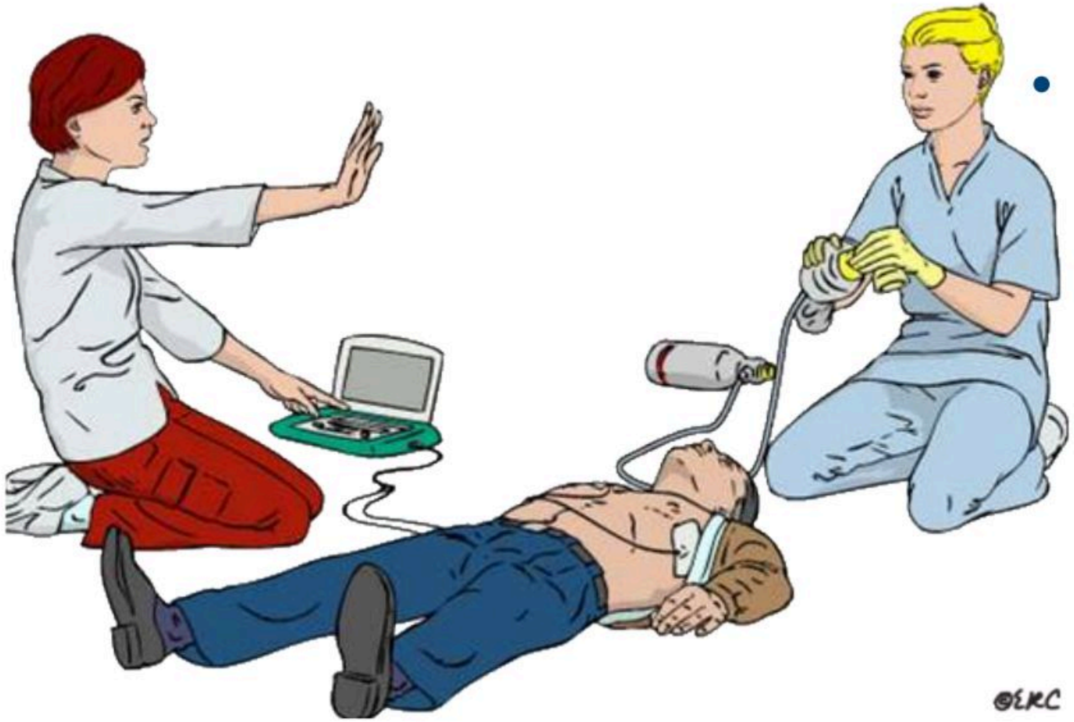
USE OF OXYGEN



Removal of oxygen from victim during defibrillation

- Take care with automated external defibrillators:
  - Stick pads down firmly
  - Remove oxygen source at least 1 metre from victim's chest during shocks

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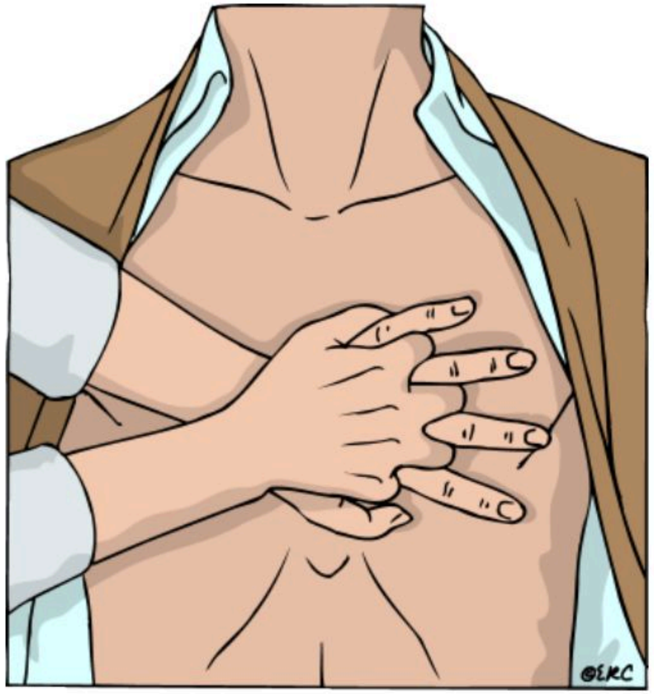
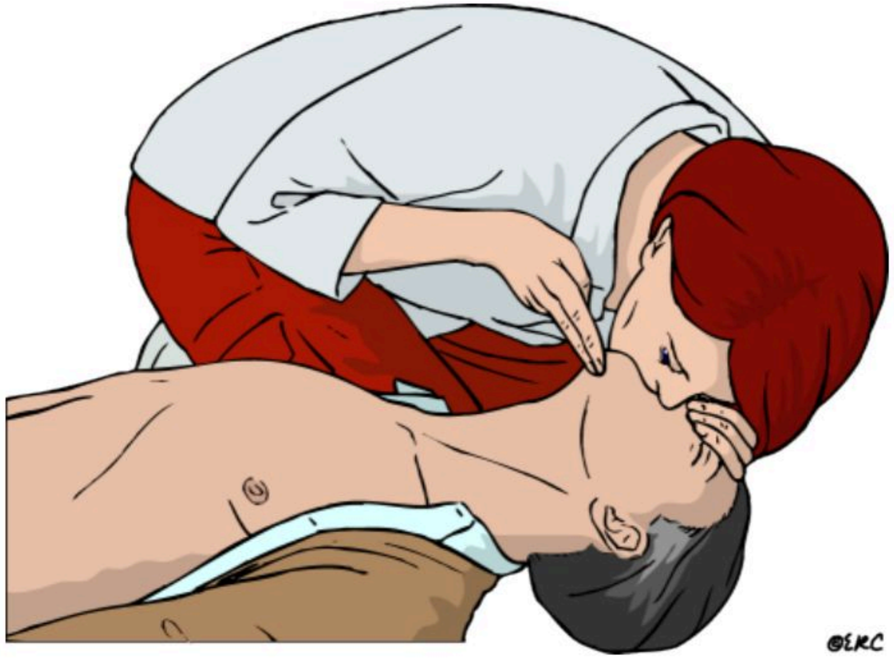
Continue CPR





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TWO-RESCUER CPR

Continue chest compressions whilst attaching AED pads

- **30 chest compressions : 2 rescue breaths**
  - One rescuer performs rescue breaths
  - The other performs chest compressions
- Use of the AED:
  - One rescuer operates AED and attaches pads
  - Other rescuer performs CPR
  - Only interrupt CPR to analyse and to deliver shock

EUROPEAN RESUSCITATION COUNCIL

- Approach safely
- Check response
- Call 112
- Open airway
- Check breathing
- 30 chest compressions
- 2 rescue breaths

Any questions?

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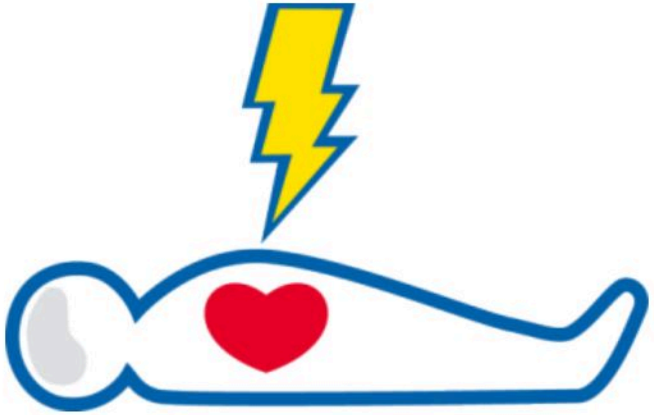
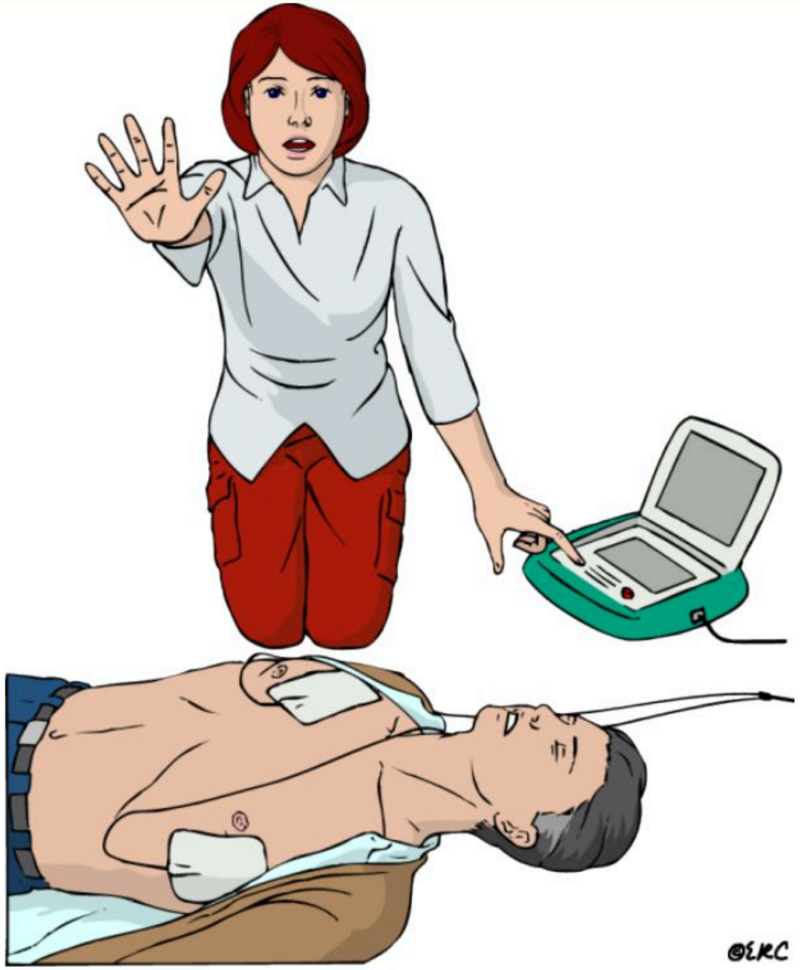
Defibrillation





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Approach safely

Check response

Call 112

Open airway

Check breathing

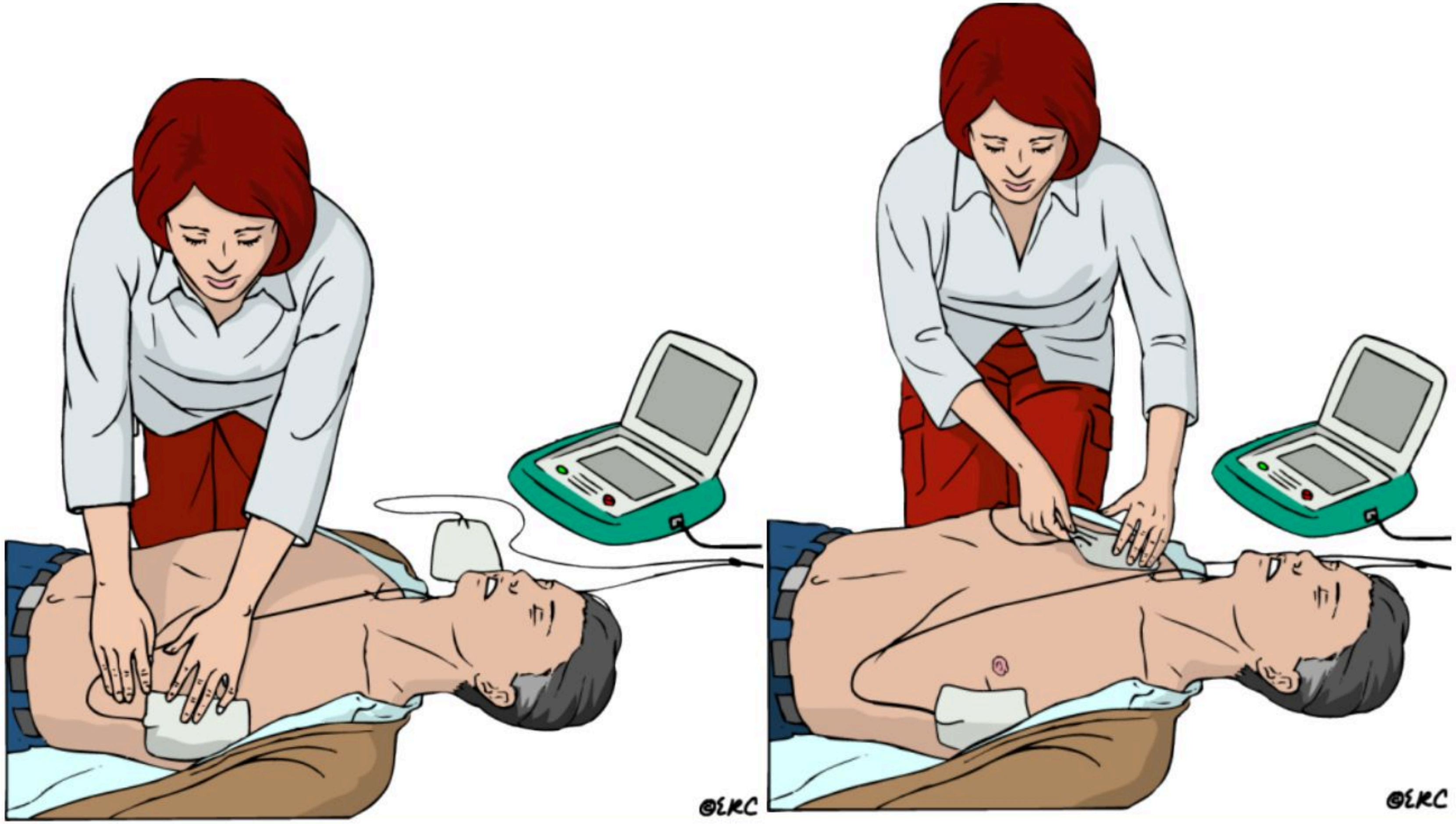
Attach AED

Follow voice

Switch on AED

- Some AEDs will automatically switch themselves on when the lid is opened

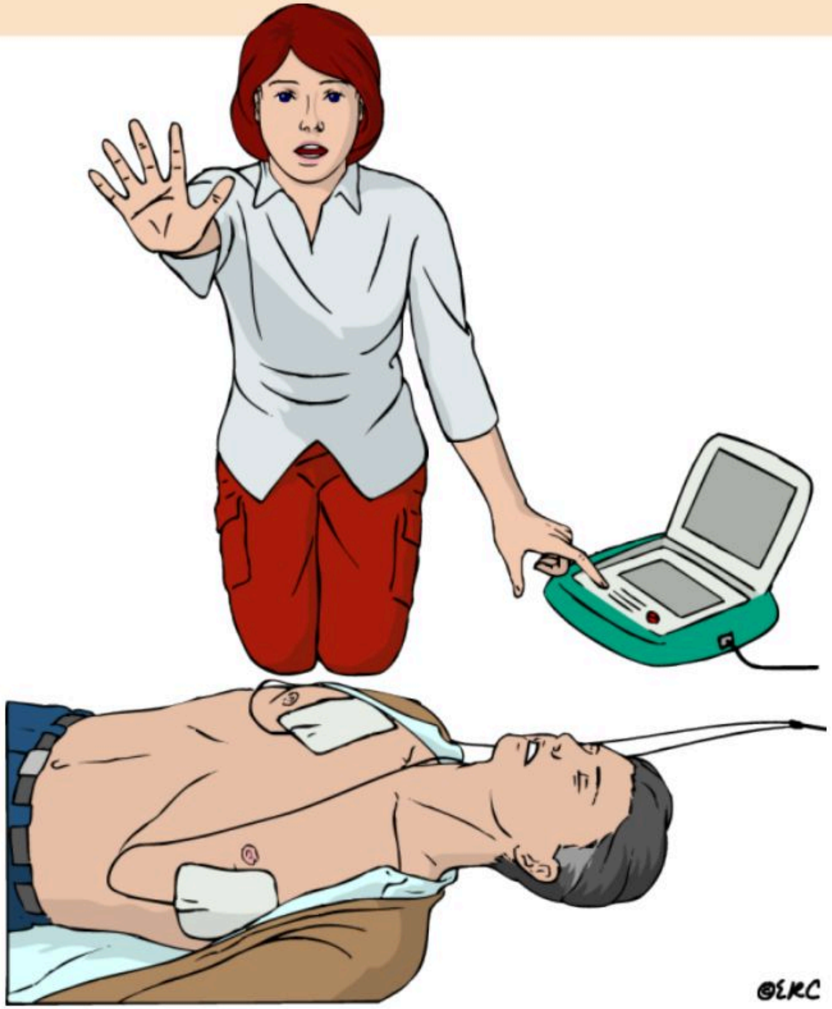
Attach pads to victims bare chest



Analysing Rhythm: Do not touch victim

Shock indicated

- Stand clear
- Deliver shock



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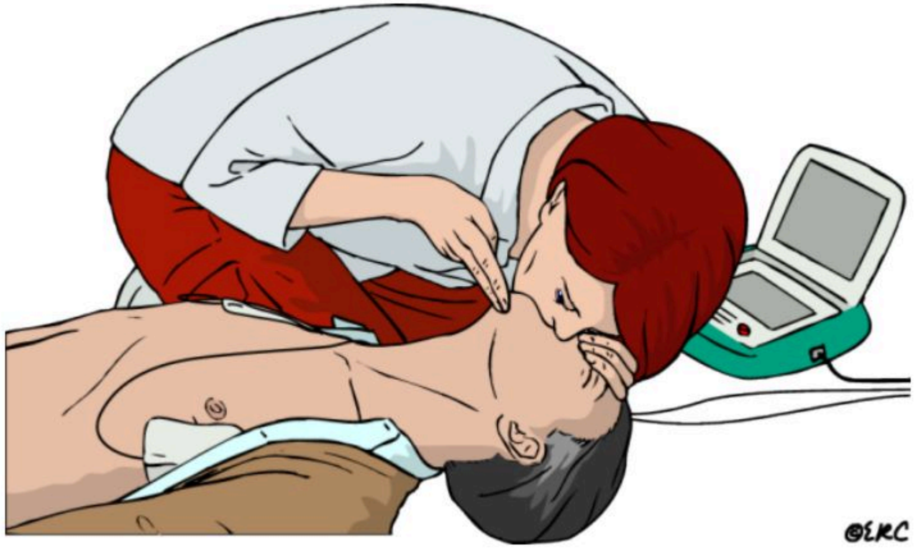
Shock delivered: Follow AED instructions

30 ©ERC



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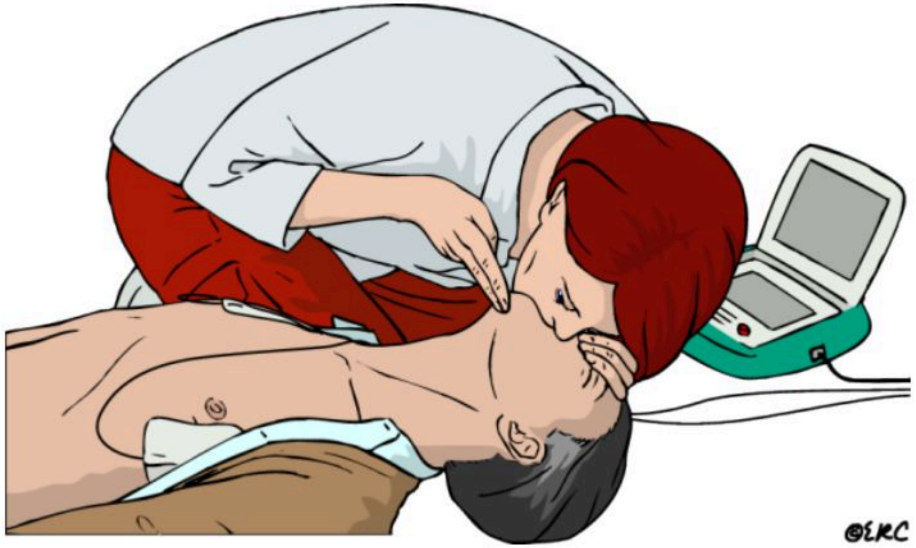
No shock advised: Follow AED instructions

30



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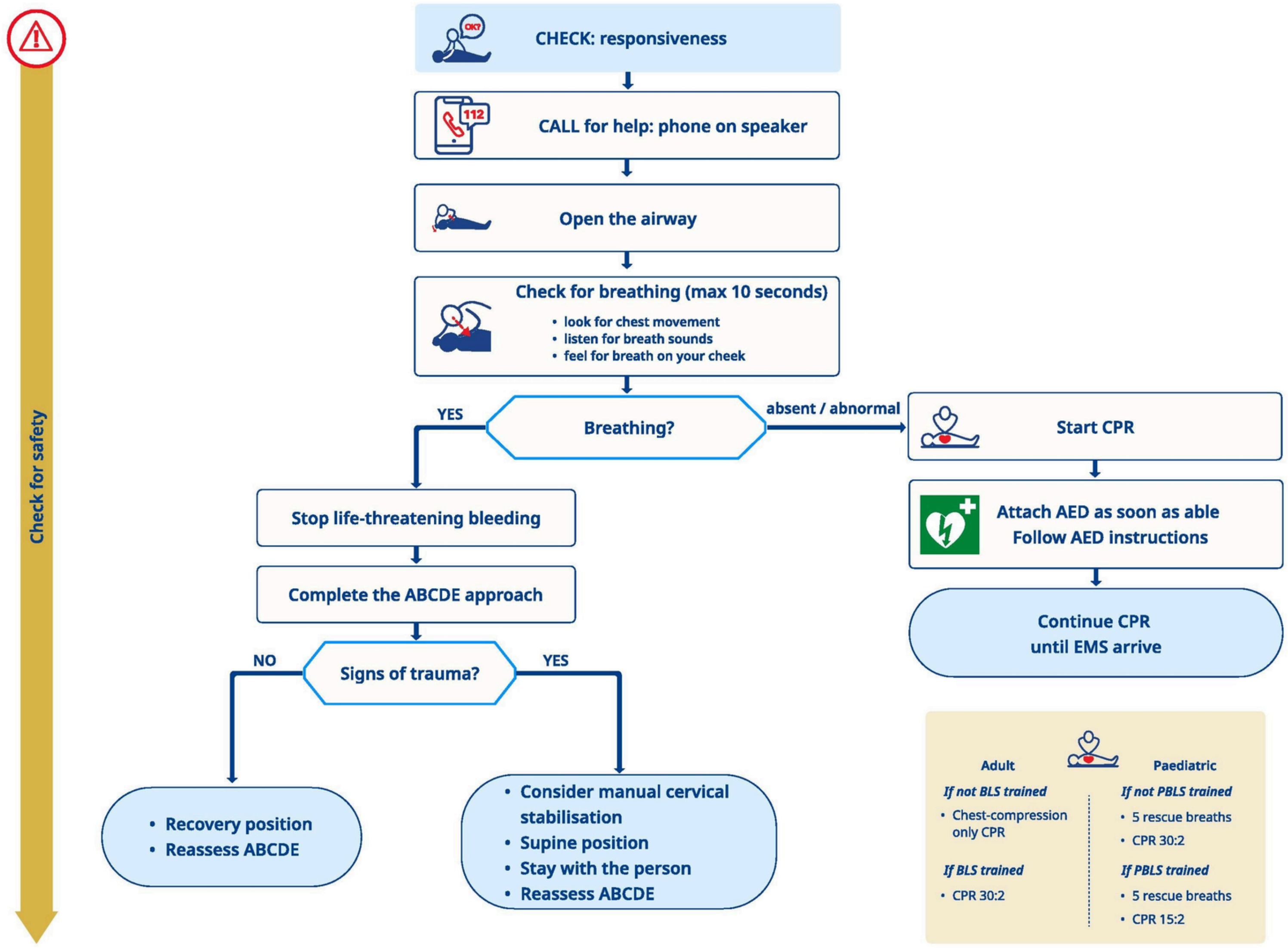
Any questions?



Djärv T, Rogers J, Semeraro F, et al. European resuscitation council guidelines 2025 first aid. *Resuscitation*. 2025;215:110752. doi:10.1016/j.resuscitation.2025.110752

If victim starts to breathe normally place in recovery position

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CPR in children

- Adult CPR techniques can be used on children

- Compressions at least 1/3 of the depth of the chest

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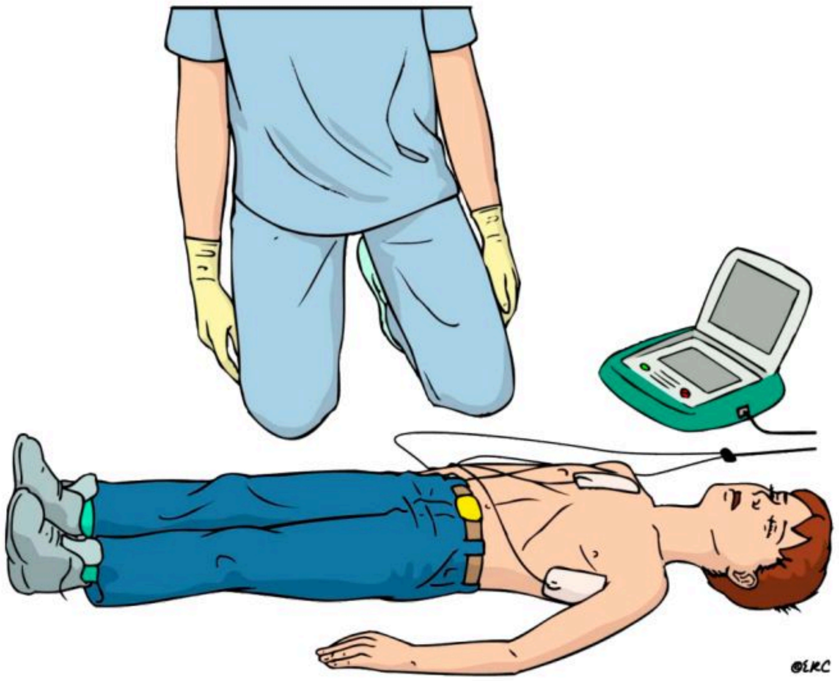
AED in children

- Age > 8 years
    - use adult AED
- Age 1-8 years
    - use paediatric pads / settings if available (otherwise use adult mode)
- Age < 1 year
    - use only if manufacturer instructions indicate it is safe



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Any questions?

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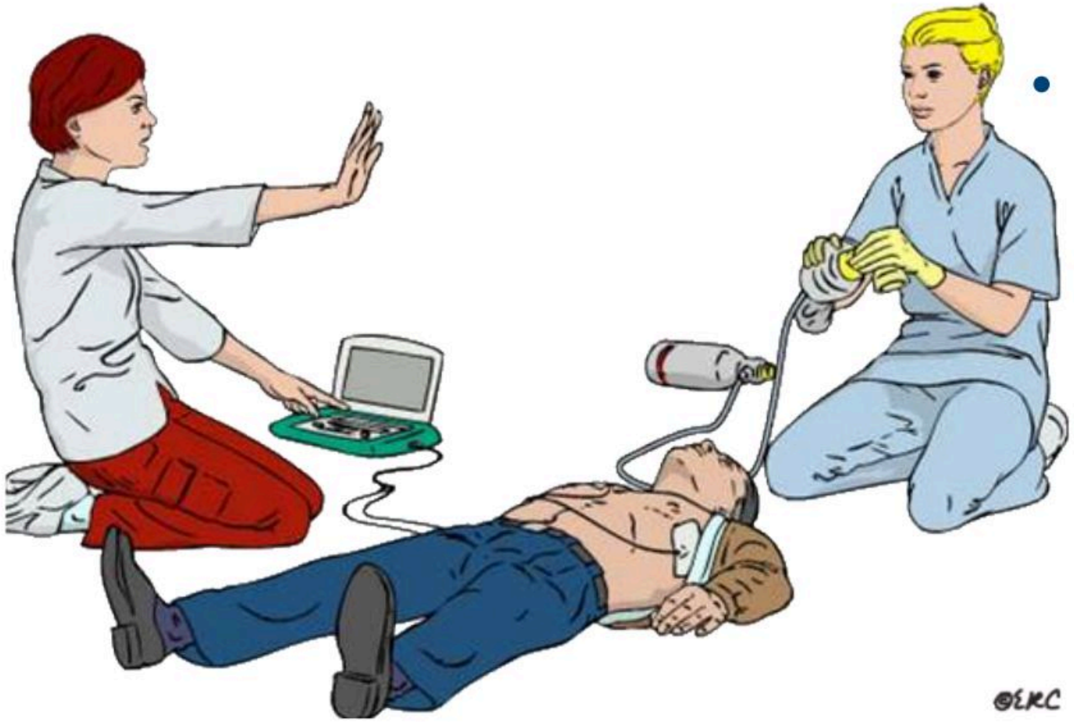
GENERAL POINTS ON USING FACE-MASKS

- Extend neck fully
- Create a good seal between mask and victim’s mouth and nose
- Deliver each breath over 1 second
- Blow just enough to make chest rise and fall as in normal breathing
  - Do not over-ventilate
- Combine 30 chest compressions with 2 rescue breaths

USE OF OXYGEN

- Supplemental oxygen can be used in cardiac arrest if available
- Given via a bag-valve mask or pocket-mask
- **Give as <u>much</u> as possible as <u>soon</u> as possible:** *Flow rates of 10-15L/min*

USE OF OXYGEN



Removal of oxygen from victim during defibrillation

- Take care with automated external defibrillators:
  - Stick pads down firmly
  - Remove oxygen source at least 1 metre from victim's chest during shocks

TWO-RESCUER CPR

Continue chest compressions whilst attaching AED pads

- **30 chest compressions : 2 rescue breaths**
  - One rescuer performs rescue breaths
  - The other performs chest compressions
- Use of the AED:
  - One rescuer operates AED and attaches pads
  - Other rescuer performs CPR
  - Only interrupt CPR to analyse and to deliver shock

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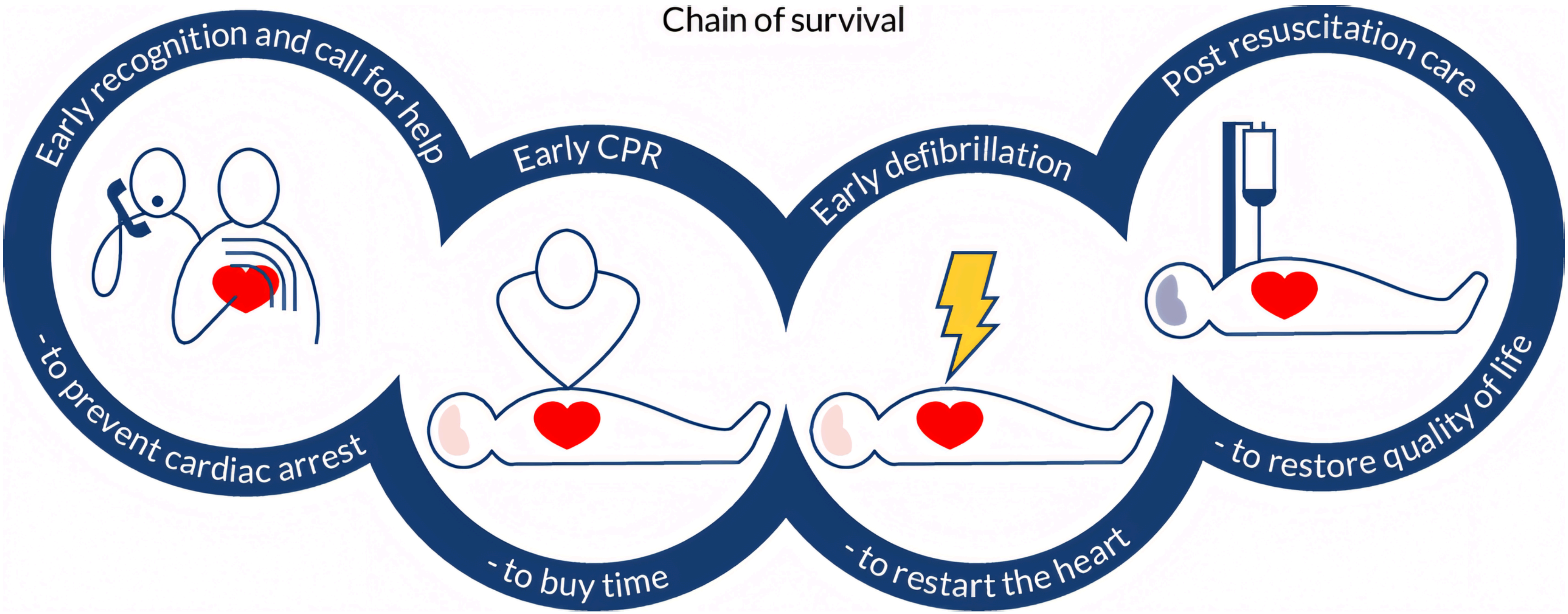
Any questions?

Section 5

Systems and the bystander


Chain of survival
Early recognition and call for help
- to prevent cardiac arrest
Early CPR
- to buy time
Early defibrillation
- to restart the heart
Post resuscitation care
- to restore quality of life

SYSTEMS SAVING LIVES GL 2021

5 TOP MESSAGES

EUROPEAN RESUSCITATION COUNCIL

1. RAISE AWARENESS ABOUT CPR AND DEFIBRILLATION

- Train as many citizens as possible
- Engage with World Restart a Heart Day
- Develop new and innovative systems and policies that will save more lives

2. USE TECHNOLOGY TO ENGAGE COMMUNITIES

- Implement technologies to alert first responders to cardiac arrests through smartphone apps / text messages
- Develop communities of first responders to help save lives
- Map and share the locations of public access defibrillators

3. KIDS SAVE LIVES

- Teach all school children to do CPR using "check, call and compress"
- Get children to teach their parents and relatives how to do CPR

4. CARDIAC ARREST CENTRES

- Where possible care for adult patients with OHCA in cardiac arrest centres

5. DISPATCH ASSISTANCE DURING CPR

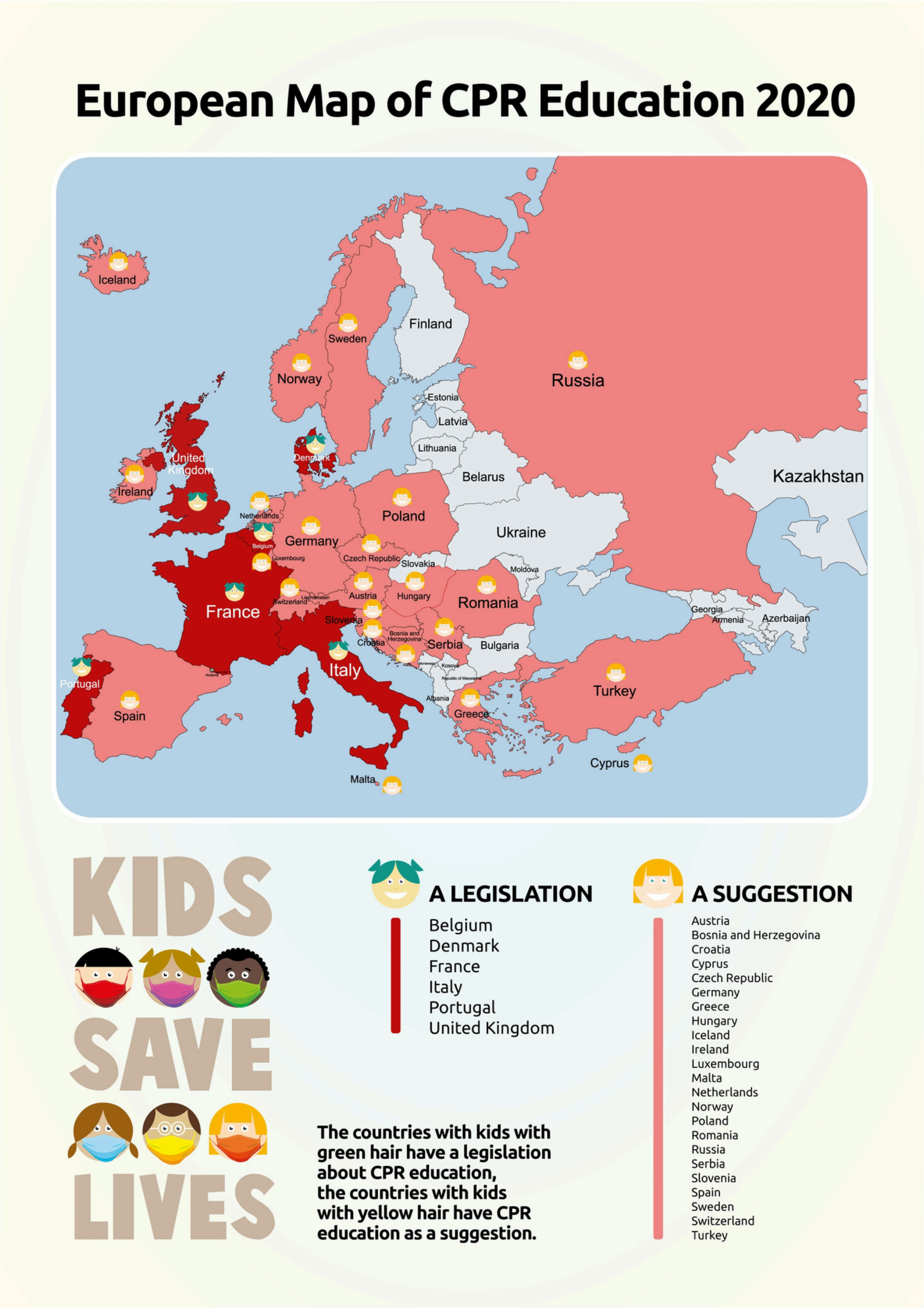
- Provide telephone assisted CPR for people who are unresponsive with absent or abnormal breathing
- Work with dispatch staff to continually monitor and improve telephone assisted CPR

The Systems Saving Lives concept emphasises the interconnection between community and EMS (e.g. KIDS SAVE LIVES) and should be implemented in each European community.

Systems Saving Lives ranges from the young student who learns CPR at school, to a citizen who receives a cardiac arrest alert through their mobile phone and is willing to start CPR and to use an automated external defibrillator (AED) on the scene, to the EMS team that continues advanced treatment to stabilise and transport the patient for post-resuscitation care in a high-performance hospital.

In Systems Saving Lives, everyone and everything is an important link to survival – we are moving from the classical four-link chain of survival to a multitude of links encompassed in the new System Saving Lives concept. Every single step in this complex system is important.

Semeraro F, Greif R, Böttiger BW, et al. European Resuscitation Council Guidelines 2021: Systems saving lives. *Resuscitation*. 2021;161:80-97. doi:10.1016/j.resuscitation.2021.02.008



Semeraro F, Greif R, Böttiger BW, et al. European Resuscitation Council Guidelines 2021: Systems saving lives. *Resuscitation*. 2021;161:80-97. doi:10.1016/j.resuscitation.2021.02.008

Effects of Bystander Interventions on Long-Term Outcomes in Out-of-Hospital Cardiac Arrests

- ## Study Objective

  To investigate the impact of bystander cardiopulmonary resuscitation (CPR) and defibrillation on 1-year risks of anoxic brain damage, nursing home admission, and all-cause mortality among survivors of out-of-hospital cardiac arrest.

- ## Methods

  Nationwide data on out-of-hospital cardiac arrests in Denmark was linked to functional outcome data. Risks were analyzed based on whether bystander CPR or defibrillation was performed, and temporal changes in bystander interventions and outcomes were evaluated.

- ## Key Findings

  During the study period (2001-2012), the rates of bystander CPR and defibrillation increased significantly, while the rates of brain damage/nursing home admission and all-cause mortality decreased.

- ## Bystander Intervention Impacts

  Bystander CPR was associated with significantly lower risks of brain damage/nursing home admission, all-cause mortality, and the composite end point of brain damage, nursing home admission, or death, compared to no bystander resuscitation.

- ## Bystander Defibrillation Impacts

  The risks of these outcomes were even lower among patients who received bystander defibrillation, compared to no bystander resuscitation.

- ## Conclusion

  Bystander CPR and defibrillation were associated with improved long-term functional outcomes and survival among survivors of out-of-hospital cardiac arrest.

Kragholm K, Wissenberg M, Mortensen RN, et al. Bystander Efforts and 1-Year Outcomes in Out-of-Hospital Cardiac Arrest. N Engl J Med. 2017;376(18):1737-1747. doi:10.1056/NEJMoa1601891

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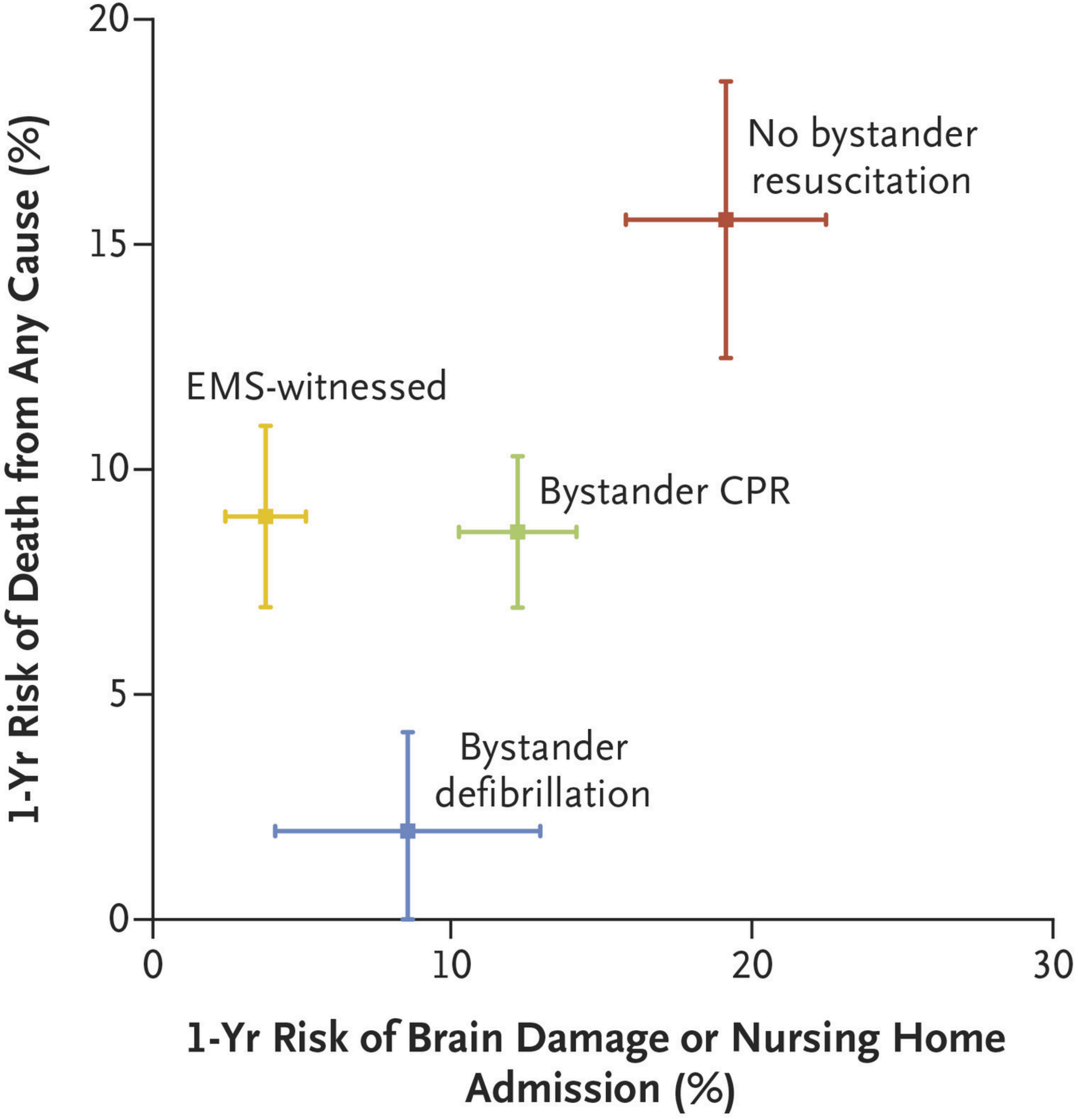
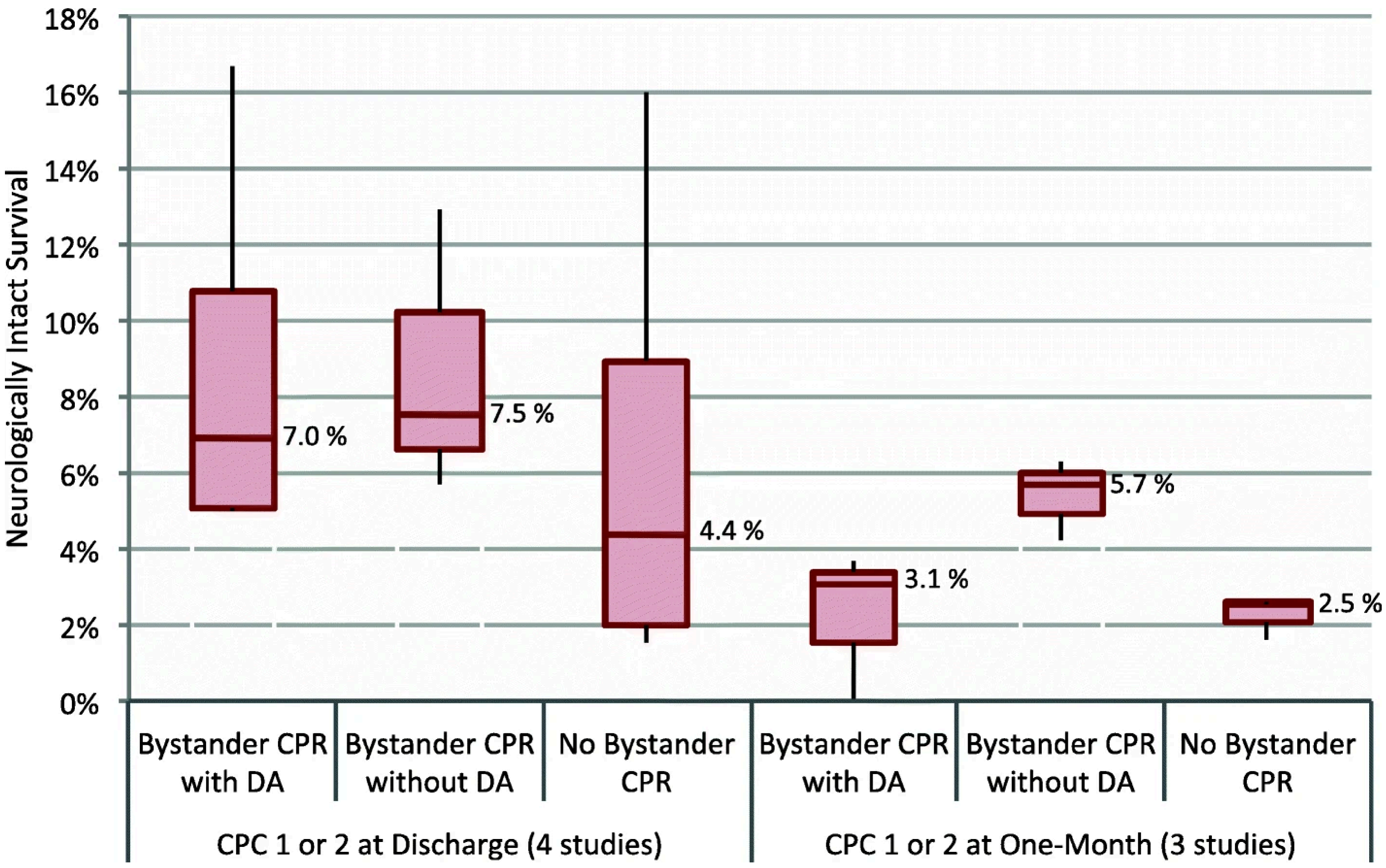


Absolute Risk of Anoxic Brain Damage or Nursing Home Admission and Death from Any Cause at 1 Year of Follow-up According to EMS-Witnessed and Bystander-Intervention Status.

Shown are the 1-year absolute risk of anoxic brain damage or nursing home admission and the 1-year absolute risk of death from any cause in relation to EMS-witnessed and bystander-intervention status. Data for 2527 of 2855 patients are included; those with missing status for bystander CPR or bystander defibrillation (328 patients) are not included in the analyses. Squares indicate point estimates (absolute risks), and I bars 95% confidence intervals.

Kragholm K, Wissenberg M, Mortensen RN, et al. Bystander Efforts and 1-Year Outcomes in Out-of-Hospital Cardiac Arrest. N Engl J Med. 2017;376(18):1737-1747. doi:10.1056/NEJMoa1601891



Eberhard KE, Linderoth G, Gregers MCT, Lippert F, Folke F. Impact of dispatcher-assisted cardiopulmonary resuscitation on neurologically intact survival in out-of-hospital cardiac arrest: a systematic review. *Scand J Trauma Resusc Emerg Med*. 2021;29(1):70. doi:10.1186/s13049-021-00875-5

Role of Emergency Dispatch Centers

- **Standardized Algorithms and Criteria**
  ILCOR recommends dispatch centers implement standardized algorithms and criteria to immediately determine if a patient is in cardiac arrest during emergency calls. This is to optimize sensitivity and minimize false negatives in recognizing cardiac arrest.

- **Dispatch-Assisted CPR**
  ILCOR and ERC recommend dispatch centers have systems in place to enable call handlers to provide CPR instructions for adult patients in cardiac arrest. This has been shown to improve survival with good neurological outcome compared to no bystander CPR.

- **Monitoring and Improving Diagnostic Capabilities**
  Dispatch centers should monitor and track their diagnostic capabilities, as studies show wide variation in recognition of cardiac arrest (46% to 98% sensitivity, 32% to 100% specificity), and continuously look for ways to improve their performance.

- **Comparing Dispatch-Assisted CPR Outcomes**
  Studies show dispatch-assisted CPR leads to better outcomes than no bystander CPR, and can be as good as unassisted bystander CPR in some cases. Adjusted analyses are important to account for prognostic differences between cohorts.

Semeraro F, Greif R, Böttiger BW, et al. European Resuscitation Council Guidelines 2021: Systems saving lives. *Resuscitation*. 2021;161:80-97. doi:10.1016/j.resuscitation.2021.02.008

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Dispatch-assisted Chest Compression-only CPR vs Standard CPR

Dispatch-assisted chest compression-only CPR

ILCOR recommends dispatchers provide instructions to perform compression-only CPR to callers for adults with suspected OHCA.

Survival with favorable neurological outcome

Only one study reported this outcome, and did not demonstrate any benefit of chest compression-only CPR over standard CPR.

Randomized controlled trials

Three randomized controlled trials involving 3728 adult OHCAs were considered in this recommendation.

Survival to hospital discharge

Similarly, survival to hospital discharge was not significantly different between the two CPR methods.

Despite low-certainty evidence, ILCOR and ERC recommend compression-only CPR instructions for dispatcher-assisted CPR, prioritizing the initiation of bystander compressions over possible harms of delayed ventilation.

Semeraro F, Greif R, Böttiger BW, et al. European Resuscitation Council Guidelines 2021: Systems saving lives. Resuscitation. 2021;161:80-97. doi:10.1016/j.resuscitation.2021.02.008

Sanità e affari sociali

Welfare

Utilizzo dei defibrillatori semiautomatici ed automatici

informazioni aggiornate a sabato, 14 agosto 2021

Il **testo unificato A.C. 181 ed abb.-B**, recante *Disposizioni in materia di utilizzo dei defibrillatori semiautomatici e automatici,* definitivamente approvato dalla Camera in seconda lettura il 28 luglio 2021, è diventato legge (con pubblicazione nella G.U, del 13 agosto 2021, L. n. 116 del 4 agosto 2021).

- Utilizzo dei defibrillatori semiautomatici ed automatici vigente.

L'articolo 3 apporta alcune **modifiche alla** legge n.120/2001 (*Utilizzo dei defibrillatori semiautomatici in ambiente extraospedaliero).* Più in particolare esso, modificando il comma 1 dell'articolo 1 della citata legge, **inserisce i defibrillatori automatici - accanto a quelli semi-automatici - nella previsione della disposizione** diretta a consentirne l'uso al **personale sanitario non medico** nonché al personale non sanitario che abbia ricevuto una specifica formazione nelle attività di rianimazione cardio-polmonare . Inoltre, con l'inserimento di un periodo aggiuntivo nel comma in esame, esso dispone che, in assenza di personale sanitario o non sanitario formato, **nei casi di sospetto arresto cardiaco è comunque consentito l'uso del defibrillatore semiautomatico od automatico anche ad una persona non in possesso dei requisiti citati**. Viene poi espressamente sancita, ai **sensi dell'articolo 54 del codice penale, la non punibilità delle azioni connesse all'uso del defibrillatore** nonché alla rianimazione cardiopolmonare intraprese dai soggetti che agiscano in stato di necessità nel tentativo di prestare soccorso ad una vittima di sospetto arresto cardiaco .

Viene poi modificato **il titolo della legge citata** inserendo anche il riferimento ai defibrillatori automatici.

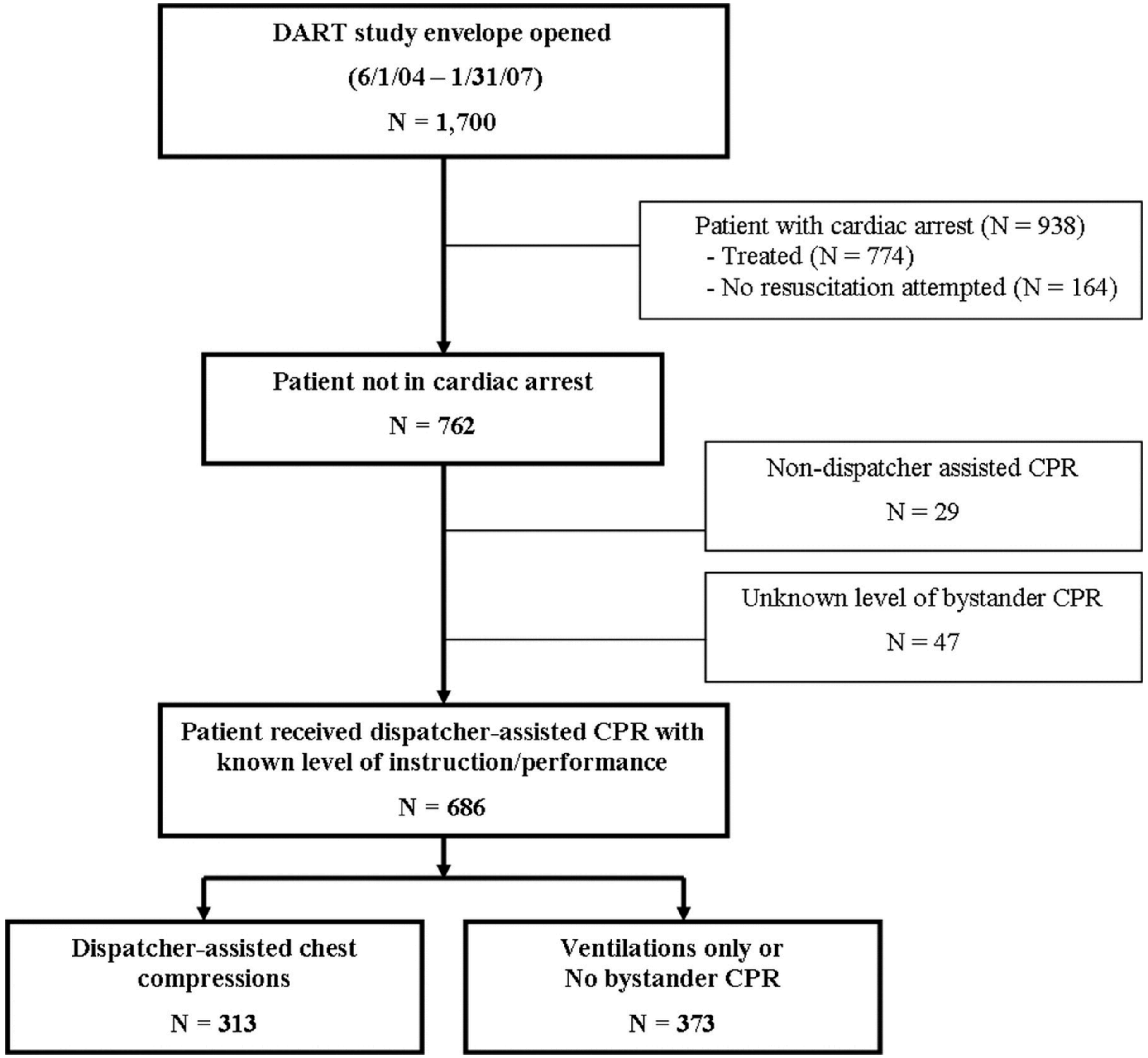
L'articolo 4 apporta alcune **modifiche all'articolo 7 del D.L 158/2012** , in tema di dotazione ed **utilizzo dei DAE** da parte delle **società sportive dilettantistiche e** professionistiche .

Con una **modifica al comma 11 del citato articolo 7**, viene specificato che **l'obbligo relativo alla dotazione ed all'impiego, da parte di società sportive sia professionistiche che dilettantistiche, di defibrillatori semiautomatici e automatici e di eventuali altri dispositivi salvavita**, sussiste nelle competizioni e durante gli allenamenti.

Ai sensi dell'**articolo 5**, inoltre, si prevede **l'introduzione alle tecniche di rianimazione cardiopolmonare di base e di utilizzo del DAE nelle scuole secondarie di primo e secondo grado**. A tale scopo viene integrato il contenuto del comma 10 dell'articolo 1 della legge 107/2015 (cd. *Buona Scuola*) che ha previsto i**niziative di formazione per gli studenti**, presso le medesime scuole, relative alle **tecniche di primo soccorso**, anche in collaborazione con il servizio di emergenza territoriale 118 del SSN. Con l'integrazione proposta si specifica che le iniziative di formazione citate devono comprendere anche **le tecniche di rianimazione cardiopolmonare di base e l'uso del DAE e la disostruzione delle vie aeree da corpo estraneo**. Nell'organizzazione di tali iniziative devono essere adottate speciali misure di attenzione nei confronti degli studenti delle scuole secondarie di primo e di secondo grado, in modo da tenere conto della sensibilità connessa all'età. Tali iniziative sono estese al personale docente e al personale amministrativo tecnico ed ausiliario.

L'**articolo 6** disciplina la **registrazione dei DAE presso le centrali operative del sistema di emergenza sanitaria 118**, disponendo che, al fine di consentire la tempestiva localizzazione del DAE più vicino in caso di evento di un arresto cardiaco, e di fornire indicazioni per il suo reperimento ai chiamanti o ad altri soccorritori, entro sessanta giorni dall'entrata in vigore della legge, i soggetti, siano essi pubblici o privati, già dotati di un DAE, sono obbligati a darne **comunicazione** alla **centrale operativa del sistema di emergenza sanitaria 118** territorialmente competente. Tale comunicazione deve specificare il numero di dispositivi, le caratteristiche e la loro ubicazione, gli orari di accessibilità al pubblico, le date di scadenza delle parti deteriorabili, nonché gli eventuali nominativi dei soggetti in possesso dell'attestato di formazione all'uso dei DAE. A tale fine, per i DAE acquistati successivamente alla data di entrata in vigore della presente legge, all'atto della vendita, il venditore deve comunicare, attraverso modulistica informatica, alla centrale operativa del sistema di emergenza sanitaria 118 territorialmente competente,sulla base dei dati forniti dall'acquirente, il luogo dove è prevista l'installazione dei DAE e il nominativo dell'acquirente, previa autorizzazione al trattamento dei dati personali. Inoltre, nei luoghi pubblici presso i quali è presente un DAE registrato, deve essere individuato un **soggetto responsabile del corretto funzionamento dell'apparecchio** e dell'adeguata informazione all'utenza sullo stesso. La Centrale operativa del sistema di emergenza sanitaria 118 territorialmente competente, sulla base dei dati forniti, presta un servizio di segnalazione periodica delle date di scadenza parti deteriorabili.

L'articolo 7 demanda ad un **Accordo da adottarsi in sede di Conferenza Stato-Regioni**, entro centoventi giorni dall'entrata in vigore della legge, la definizione delle modalità operative per la realizzazione e l'adozione di **un'applicazione mobile integrata con i servizi delle centrali operative del sistema di emergenza sanitaria "118"** per la rapida geolocalizzazione dei soccorritori e dei DAE più vicini al luogo in cui si sia verificata l'emergenza. I soccorritori, reclutabili attraverso l'applicazione del presente comma, sono individuati tra quelli registrati su base volontaria negli archivi informatici della Centrale operativa del 118 territorialmente competente .



The investigation was a prospective cohort study of adult patients not in cardiac arrest for whom dispatchers provided CPR instructions in King County, Washington, between June 1, 2004, and January 31, 2007.

The study focused on those who received chest compressions. Information was collected through review of the audio and written dispatch report, written emergency medical services report, hospital record, and telephone survey. Of the 1700 patients for whom dispatcher CPR instructions were initiated, 55% (938 of 1700) were in arrest, 45% (762 of 1700) were not in arrest, and 18% (313 of 1700) were not in arrest and received bystander chest compressions.

Of the 247 not in arrest who received chest compressions and had complete outcome ascertainment, 12% (29 of 247) experienced discomfort, and 2% (6 of 247) sustained injuries likely or possibly caused by bystander CPR. Only 2% (5 of 247) suffered a fracture, and no patients suffered visceral organ injury.

White L, Rogers J, Bloomingdale M, et al. Dispatcher-assisted cardiopulmonary resuscitation: risks for patients not in cardiac arrest. Circulation. 2010;121(1):91-97. doi:10.1161/CIRCULATIONAHA.109.872366

Table 1. Characteristics of Patients Not in Cardiac Arrest, Overall and According to Chest Compression Status

	All Patients Not in Cardiac Arrest (n=686)	Chest Compressions(n=313)	Ventilations Only or No Bystander CPR (n=373)	*P*
Age, mean±SD, y	56.8±22.4	53.9±22.0	59.2±22.6	0.002
Male sex, n (%)	367 (53.5)	175 (55.9)	192 (51.5)	0.35
EMS assessment, n (%)				<0.0001
Cerebrovascular event	78 (11.4)	42 (13.4)	36 (9.7)	
Hypoglycemia	62 (9.0)	35 (11.2)	27 (7.2)	
Overdose/intoxication	141 (20.6)	87 (27.8)	54 (14.5)	
Seizure	120 (17.5)	51 (16.3)	69 (18.5)	
Syncope	105 (15.3)	32 (10.2)	73 (19.6)	
Other illness*	180 (26.2)	66 (21.1)	114 (30.6)	
Transport, n (%)				<0.0001
Advanced life support	310 (45.2)	167 (53.4)	143 (38.3)	
Basic life support	275 (40.1)	114 (36.4)	161 (43.2)	
Private vehicle	4 (0.6)	1 (0.3)	3 (0.8)	
No transport	97 (14.1)	31 (9.9)	66 (17.7)	
Randomization, n (%)				<0.0001
Chest compressions alone	340 (49.6)	194 (62.0)	146 (39.1)	
Compressions plus ventilations	346 (50.4)	119 (38.0)	227 (60.9)	

*The other illness category comprised mostly respiratory conditions, cardiovascular emergencies, and psychiatric issues.

White L, Rogers J, Bloomingdale M, et al. Dispatcher-assisted cardiopulmonary resuscitation: risks for patients not in cardiac arrest. Circulation. 2010;121(1):91-97. doi:10.1161/CIRCULATIONAHA.109.872366

Table 2. Outcomes of Patients Not in Cardiac Arrest Who Received Dispatcher-Assisted Bystander Chest Compressions, Overall and According to Randomization Assignment

	Overall (n=247)	Compressions Alone (n=152)	Compressions Plus Ventilations (n=95)	*P*
Hospital admission status, n (%)				0.31
Admitted	157 (63.6)	97 (63.8)	60 (63.2)	
Treated in emergency department and released	83 (33.6)	50 (32.9)	33 (34.7)	
Died in emergency department	3 (1.2)	1 (0.7)	2 (2.1)	
Left emergency department against medical advice	4 (1.6)	4 (2.6)	0 (0.0)	
Discharge disposition (n=157), n (%)				0.89
Discharged alive	125 (79.6)	76 (78.3)	49 (81.7)	
Died in hospital	17 (10.8)	12 (12.4)	5 (8.3)	
Transferred, disposition unknown	15 (9.6)	9 (9.3)	6 (10.0)	
Chest imaging performed, n (%)				0.71
Yes	166 (67.2)	101 (66.4)	65 (68.4)	
No	81 (32.8)	51 (33.6)	30 (31.6)	
Any CPR pain or injury, n (%)				0.92
Yes	22 (8.9)	14 (9.2)	8 (8.4)	
Possible	9 (3.6)	6 (3.9)	3 (3.2)	
No	216 (87.4)	132 (86.8)	84 (88.4)	
Injury type, n (%)				
Pain				0.71
Yes	22 (8.9)	14 (9.2)	8 (8.4)	
Possible	7 (2.8)	4 (2.6)	3 (3.2)	
Rib fractures				0.46
Yes	2 (0.8)	2 (1.3)	0 (0.0)	
Possible	2 (0.8)	2 (1.3)	0 (0.0)	
Internal bleeding				0.71
Yes	0 (0.0)	0 (0.0)	0 (0.0)	
Possible	1 (0.4)	1 (0.7)	0 (0.0)	
Other injuries				0.71
Yes	1 (0.4)	1 (0.7)	0 (0.0)	
Possible	0 (0.0)	0 (0.0)	0 (0.0)	

White L, Rogers J, Bloomingdale M, et al. Dispatcher-assisted cardiopulmonary resuscitation: risks for patients not in cardiac arrest. Circulation. 2010;121(1):91-97. doi:10.1161/CIRCULATIONAHA.109.872366

Section 6

The safe scene


TOWING LYDENBURG


12
28

Section 7

The normal and agonal breathing


THIS ISN'T
BREATHING
YouTube

Understanding Gasping: Physiology, Definition, and Terminology

Gasping Physiology

Gasping is a physiologic entity observed in mammals who have sustained a global ischemic insult, such as sudden cardiac arrest or severe hemorrhagic shock.

Definition of Gasping

Gasping is an 'abrupt, sudden, transient inspiratory effort', which has been described in the literature since 1812.

Terminology: Agonal Breaths

The classic gasping that occurs after sudden cardiac arrest is sometimes referred to as 'agonal breaths' or 'agonal respirations', though the term 'agonal breathing' may also be used to describe a broader variety of abnormal respiratory efforts.

Characteristics of Gasping

Classic gasps are usually sudden, abrupt, and much brisker and larger than normal respiratory efforts, distinct from the slower and often shallow breaths observed in progressive respiratory failure.

Agonal Respiration in Out-of-Hospital Cardiac Arrest

Prevalence of Agonal Respiration

Only 40% of patients experiencing out-of-hospital cardiac arrest exhibited agonal respirations, as detected by dispatchers.

Witnessed Cardiac Arrests

In cases where the cardiac arrest was witnessed by bystanders, the rate of agonal respirations increased to 55%.

Unwitnessed Cardiac Arrests

The incidence of agonal breaths was much lower (16%) for unwitnessed cardiac arrests.

Ventricular Fibrillation Arrests

In a recent study, the presence of gasping was significantly higher in cases of witnessed ventricular fibrillation (18.4%) compared to other presenting EKG findings.

Implications

The detection of gasping in only about half of the witnessed cardiac arrest cases indicates that agonal respiration is not a universal event in sudden out-of-hospital cardiac arrest.

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Agonal Respirations in Cardiac Arrest: Physiologic and Resuscitation Implications

Agonal Respirations in Cardiac Arrest

Agonal respirations, also known as 'gasping' respirations, are commonly observed in animal models of cardiac arrest.

Physiologic Consequences of Agonal Respirations

Agonal respirations can produce clinically important ventilation, oxygenation, and circulation by generating substantial tidal volumes, higher arterial oxygenation, and lower arterial carbon dioxide levels.

Cardiovascular Effects of Agonal Respirations

Agonal respirations create favorable hemodynamic conditions, such as increased venous return and improved coronary perfusion, by modulating intrathoracic and aortic pressures.

Agonal Respirations and Resuscitation Outcomes

Animal studies have demonstrated a dose-effect relationship between agonal respirations and improved survival outcomes, suggesting a potential mechanistic and prognostic role of agonal respirations.

Roppolo LP, Pepe PE, Bobrow BJ. The Role of Gasping in Resuscitation. In: Vincent JL, ed. Yearbook of Intensive Care and Emergency Medicine 2010. Springer; 2010:83-95. doi:10.1007/978-3-642-10286-8_9

Abnormal Respiratory Patterns in Brainstem Lesions

Thomas Lumsden's Early Investigations

Thomas Lumsden first investigated patterns of abnormal respirations in the early 20th century through a series of animal studies.

Brainstem Transection and Respiratory Patterns

Transection of the brainstem at different levels produces distinct abnormal respiratory patterns, such as 'apneusis' (prolonged inspiration with inspiratory pause) at the midpons level, and 'gasping' respirations (more vigorous but shorter inspirations) at the pontomedullary junction.

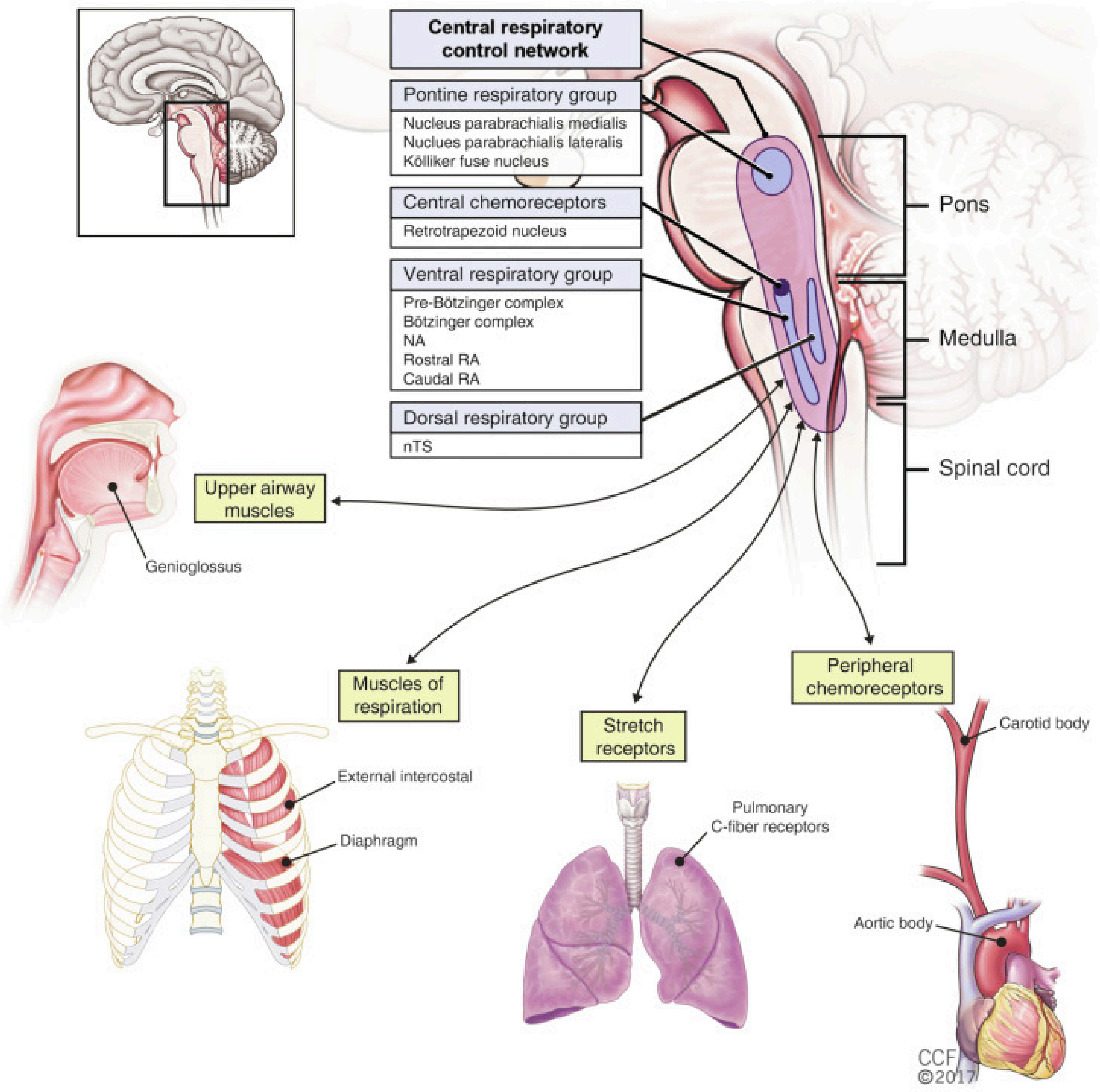
Abnormal Ventilatory Patterns

Other abnormal ventilatory patterns, such as 'ataxic' or irregular ventilations, appear to occur when the pons and midbrain retain varying levels of function.

Spectrum of Abnormal Breathing Patterns

Given the dynamic state of brain oxygenation during cardiac arrest, a spectrum of abnormal breathing patterns might be expected even within the individual patient with agonal respirations.

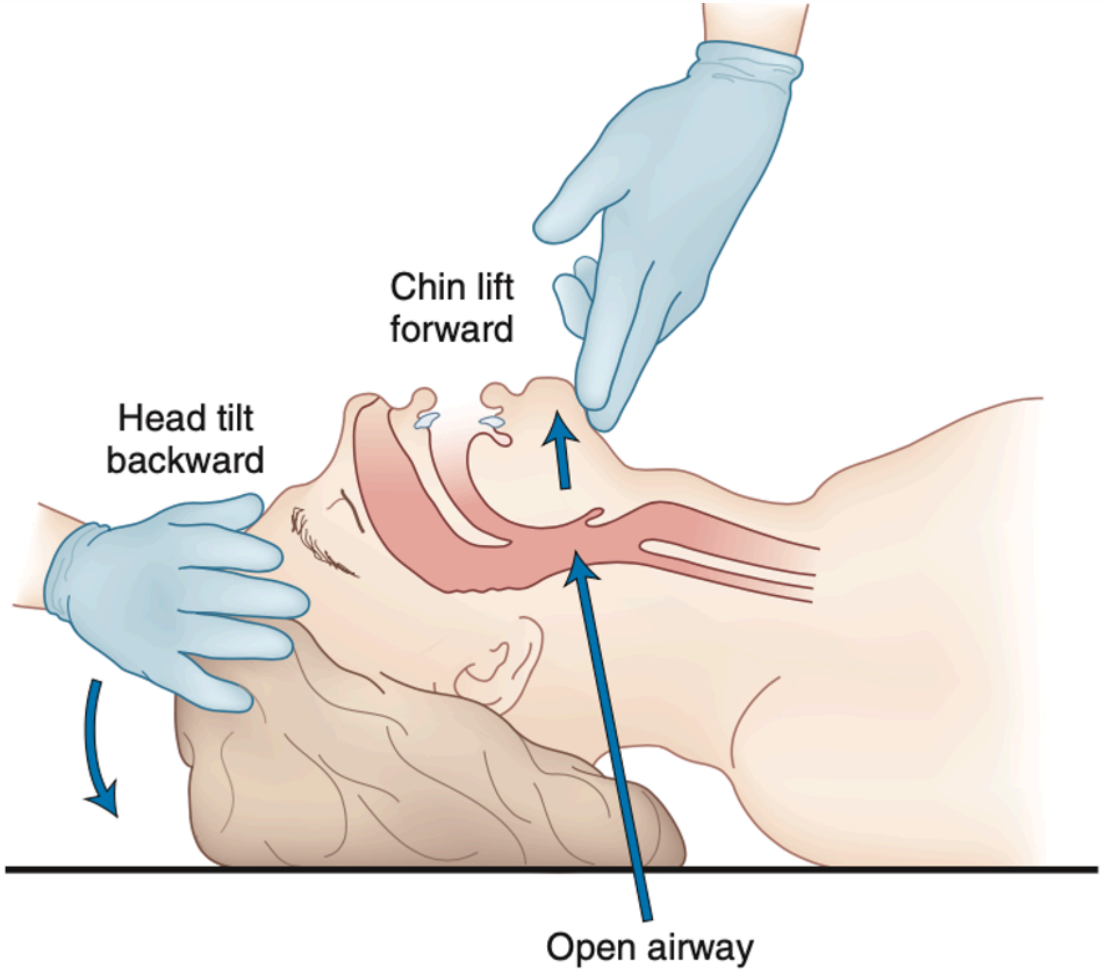
Rea TD. Agonal respirations during cardiac arrest. doi:10.1097/01.ccx.0000162095.08148.64



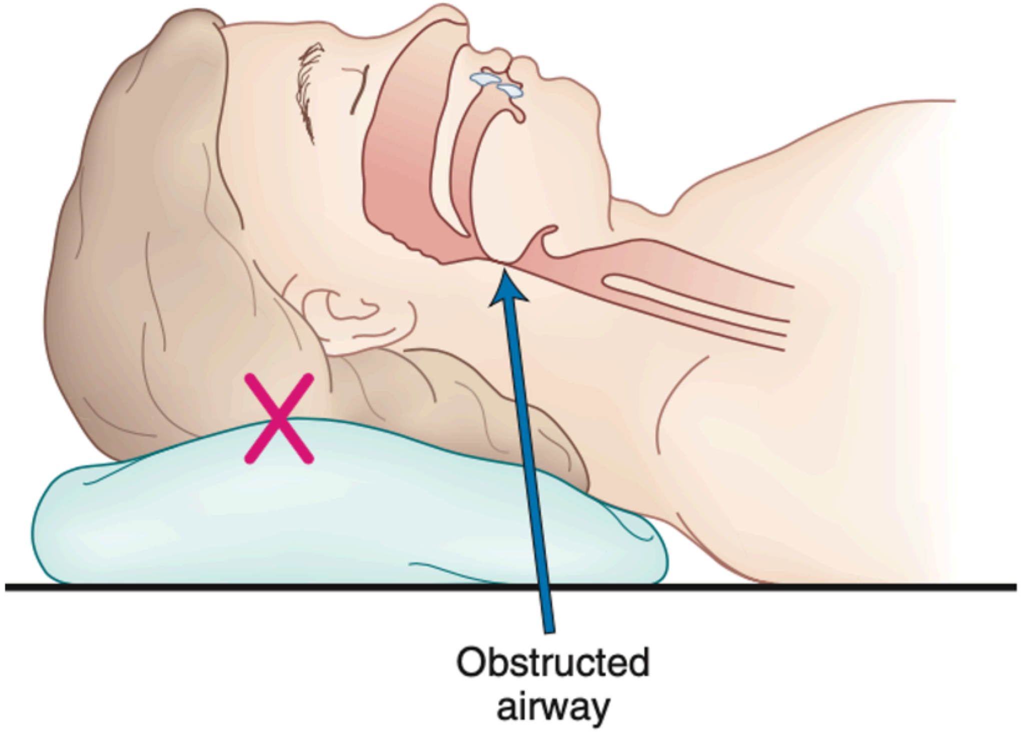
Waters T, Mehra R. Chapter 22 - Clinical neurophysiology of apnea. In: Levin KH, Chauvel P, eds. Handbook of Clinical Neurology. Vol 161. Clinical Neurophysiology: Diseases and Disorders. Elsevier; 2019:345-352. doi:10.1016/B978-0-444-64142-7.00059-X

Section 8

The airway



B **Head tilt and chin lift to obtain extended position**



Tongue in apposition to posterior pharyngeal wall

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.


a) Jaw thrust

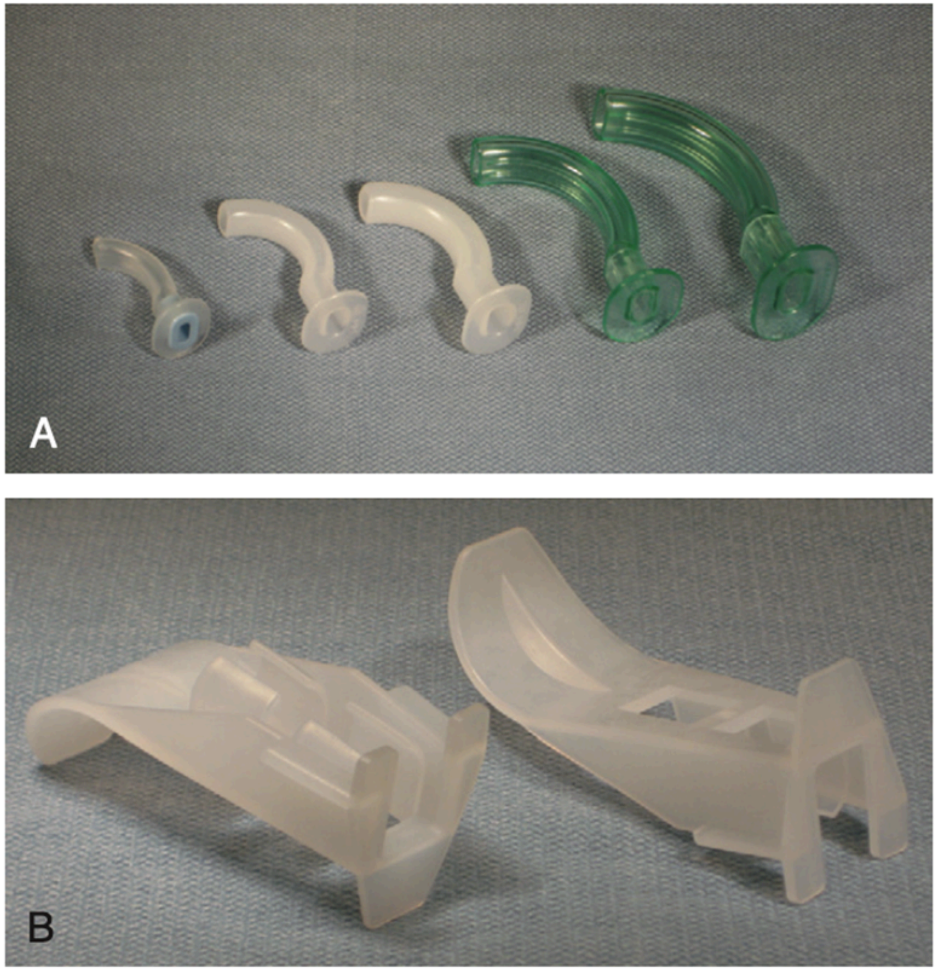
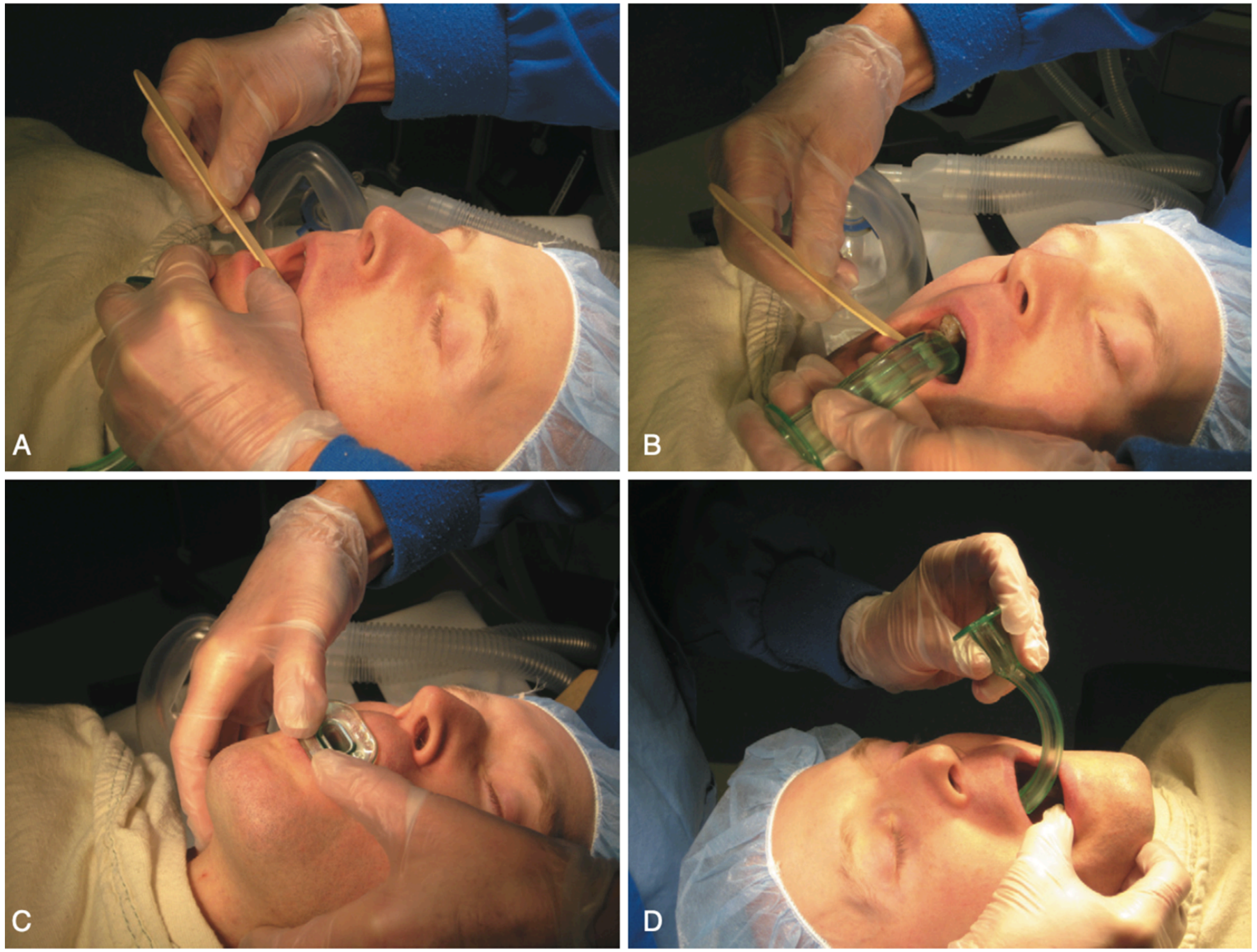


Figure 15-7 Oropharyngeal airways. **A,** Guedel Airways in sizes from neonatal to large adult. **B,** The Ovassapian Airway has a large anterior flange to control the tongue. The airway is open posteriorly (including no posterior flange) so that an endotracheal tube can be inserted with a flexible fiberoptic scope and the assembly later separated.



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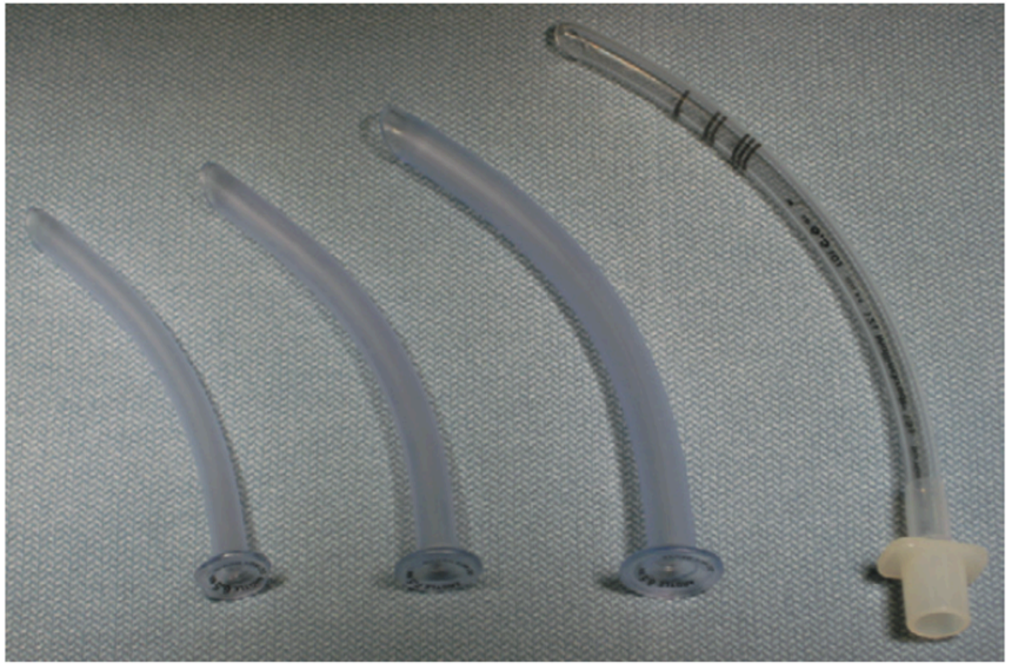


Figure 15-9 Nasopharyngeal airways. A flange prevents the outside end from passing beyond the nares, controlling the depth of insertion. Alternatively, an endotracheal tube may be cut down to provide a longer airway, with its 15-mm adapter reinserted in the cut end.

Figure 15-10 Insertion of a nasopharyngeal airway. The airway is oriented with its concave side toward the hard palate and inserted straight posteriorly. Gripping the airway near the top allows the tube to bend if there is resistance to passage. If it is gripped too close to the naris, the clinician can generate sufficient force to shear off a turbinate.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

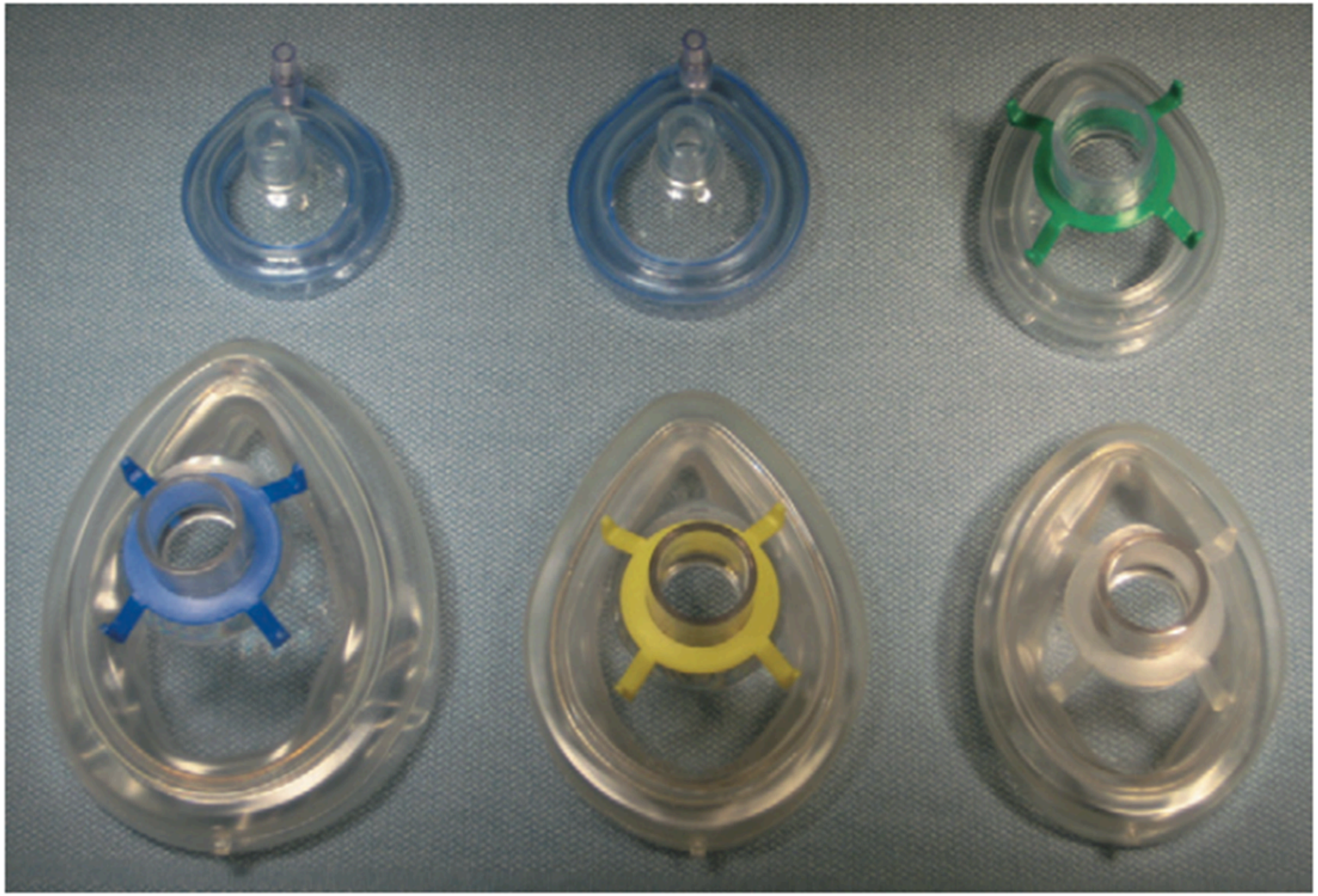


Figure 15-11 Assorted sizes of disposable, transparent face masks. The smallest masks have a 15-mm male adapter, and the larger sizes have a 22-mm female adapter to allow them to be connected to a standard breathing circuit or resuscitator bag.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

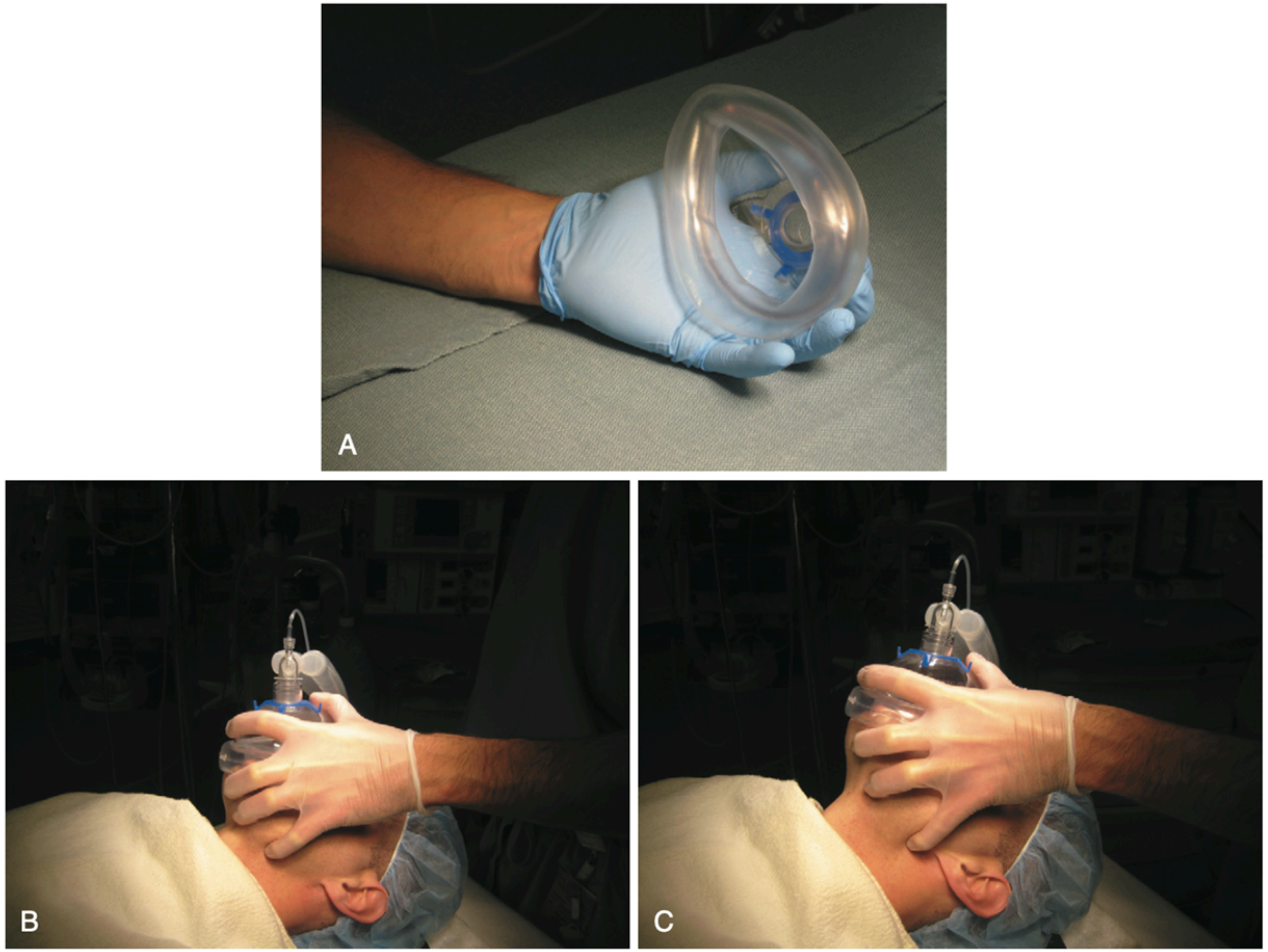


Figure 15-13 Suggested techniques for holding and supporting a face mask. **A,** In the proper hand grip of the face mask, the thumb and index finger encircle the collar while the hypothenar eminence extends below the left side of the mask. **B,** In the side view of the standard one-handed application of the face mask, the thumb and first finger (or first two) encircle the collar of the mask while the remaining fingers pull the mandible up into the mask while gently extending the head. **C,** During the one-handed mask grip with concurrent jaw thrust, notice how the little finger is located at the angle of the jaw, pulling backward and upward to maintain the jaw thrust (subluxation). Because of the increased span of the hand, only the first finger is on the mask while the middle and ring fingers pull the mandible up into the mask and extend the head.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

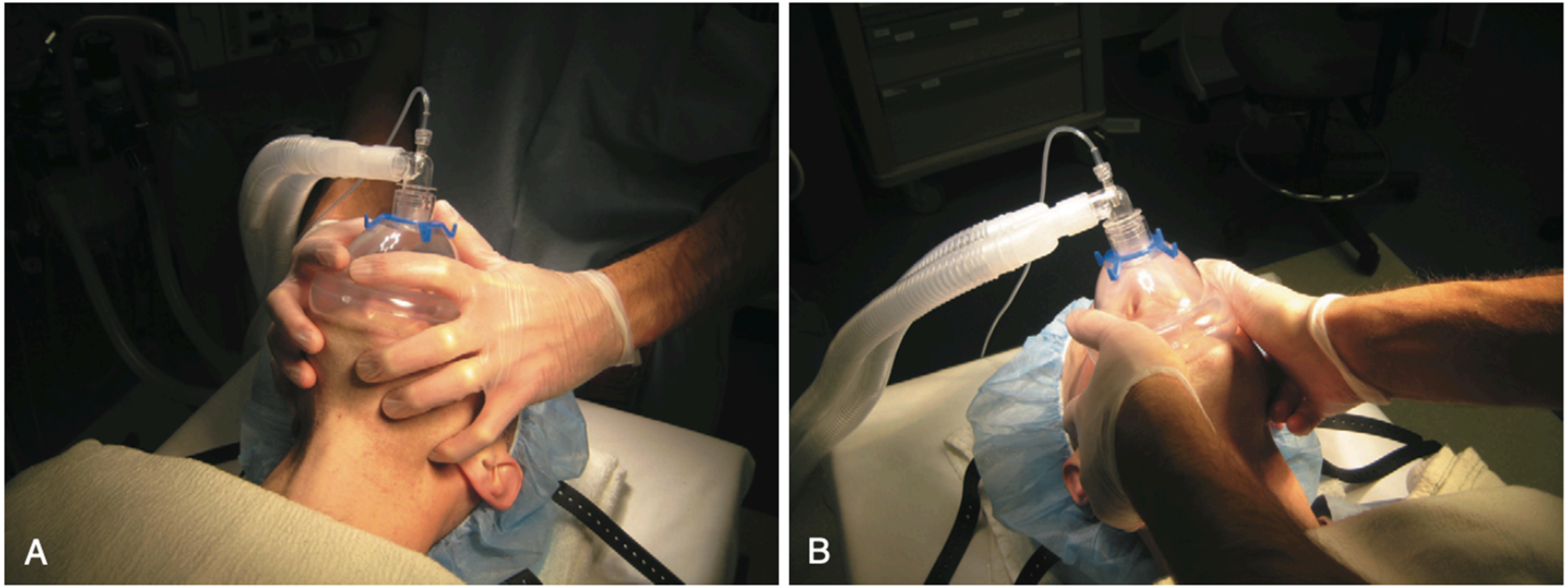


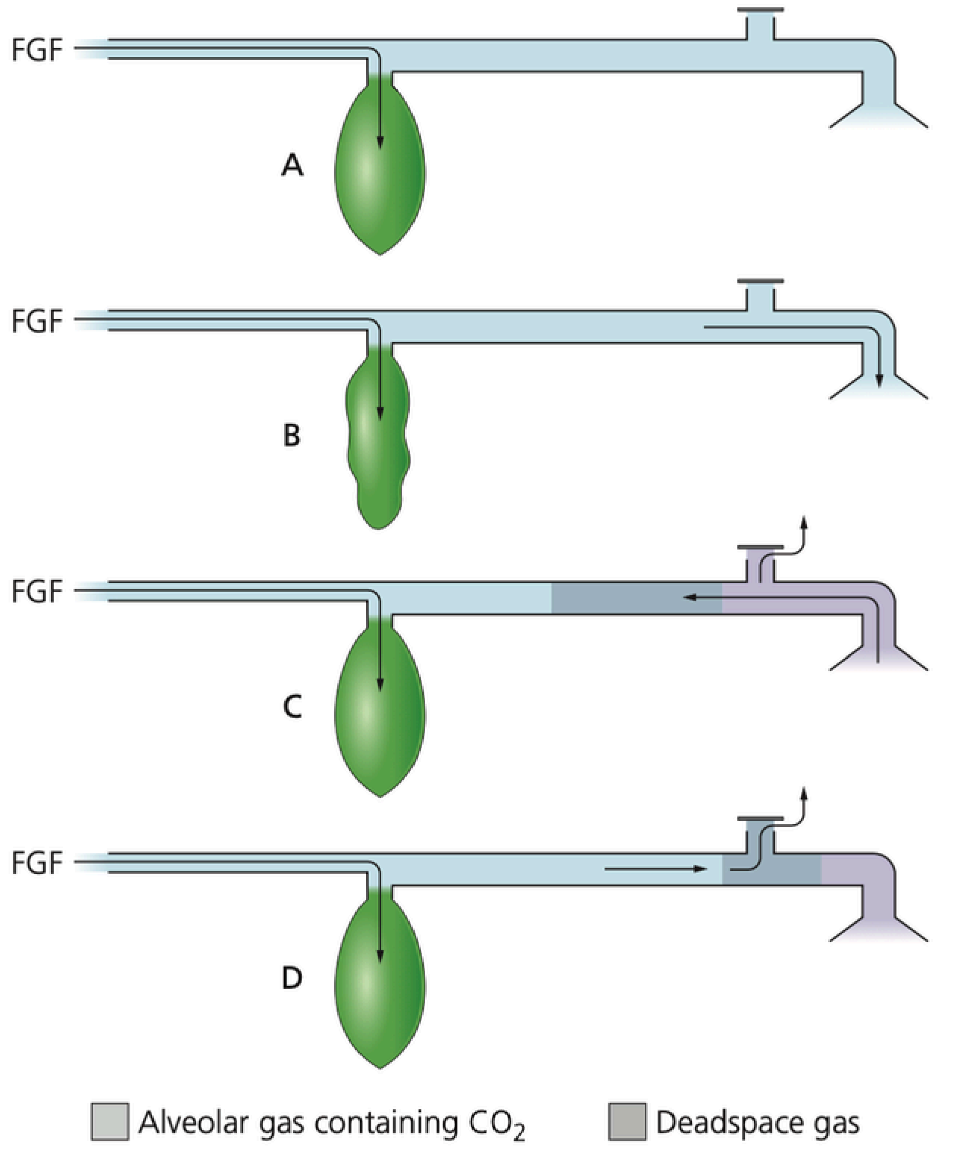
Figure 15-15 Two-hand control of a face mask. In both scenarios, a second provider must ventilate the patient. **A,** In the view of two-hand control of a face mask from above the patient, notice how the lower fingers on both hands apply a jaw thrust while the thumbs seal the mask to the face. **B,** In the view of two-hand control of a face mask from the side of the patient, the person ventilating the patient has improved access to the head as the airway is maintained from the patient's side. This arrangement is beneficial if the ventilating provider is preparing to perform laryngoscopy.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

Figure 15-14 Adult and pediatric sizes of air-mask-bag unit (AMBU). The AMBU is a portable, self-inflating, easy-to-use system for the delivery of positive-pressure ventilation. It can be used with a face mask, laryngeal mask airway, or endotracheal tube.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

Fig. 4.8 Intersurgical adult Mapleson C system.



Al-Shaikh B. *Essentials of Equipment in Anaesthesia, Critical Care, and Peri-Operative Medicine e-Book*. 5th ed. Elsevier; 2017.

Head and neck position and the axes of the head and neck upper airway

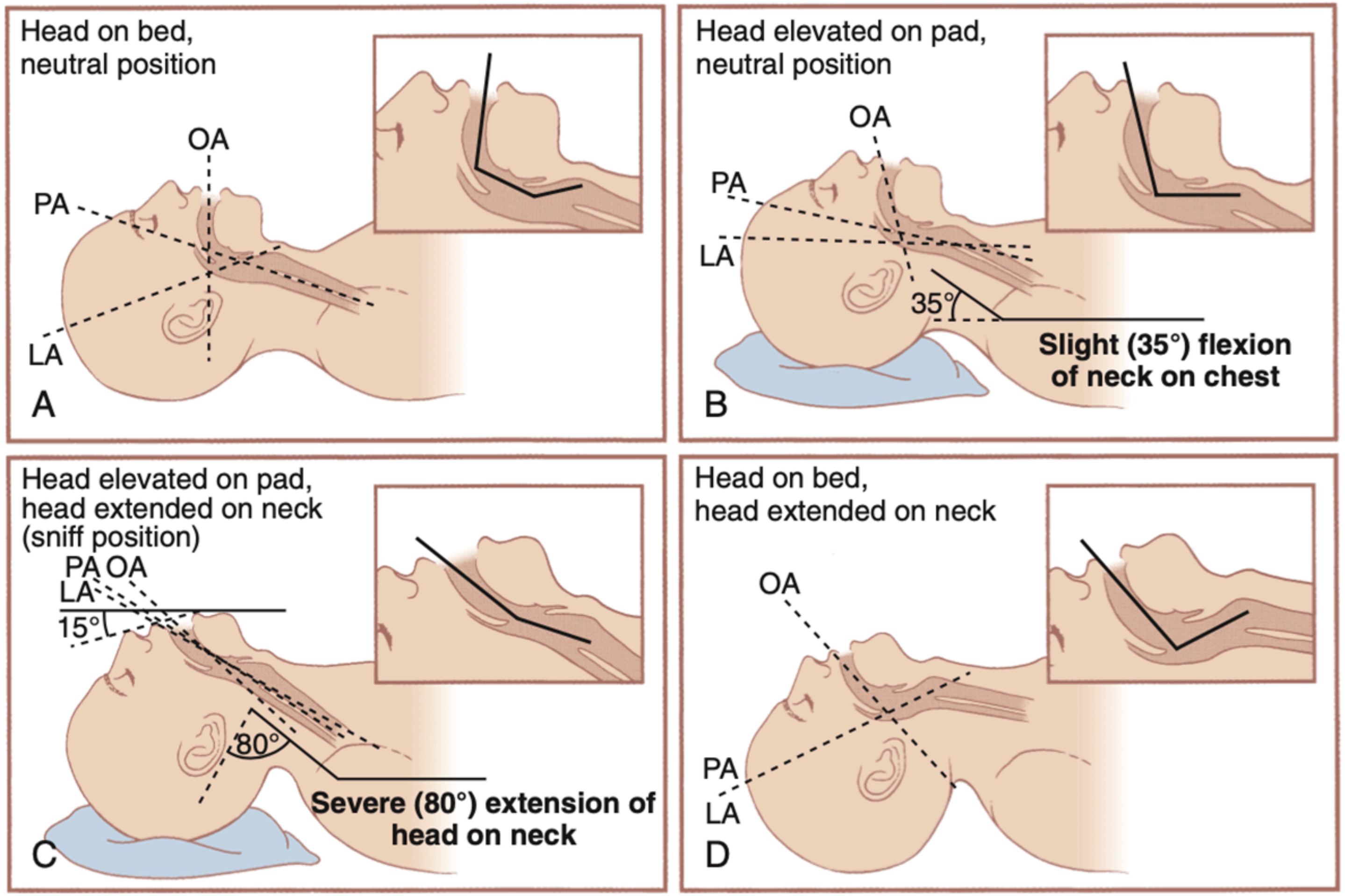


Figure 17-1 Schematic diagrams show the alignment of the oral axis *(OA)*, pharyngeal axis *(PA)*, and laryngeal axis *(LA)* in four different head positions. Each head position is accompanied by an *inset* that magnifies the upper airway (oral cavity, pharynx, and larynx) and superimposes *(bent bold line)* the continuity of these three axes within the upper airway. **A,** The head is in the neutral position with a marked degree of nonalignment of the LA, PA, and OA. **B,** The head is resting on a large pad that flexes the neck on the chest and aligns the LA with the PA. **C,** The head is resting on a pad (which flexes the neck on the chest). Concomitant extension of the head on the neck brings all three axes into alignment (sniffing position). **D,** Extension of the head on the neck without concomitant elevation of the head on a pad, which results in nonalignment of the PA and LA with the OA. (From Benumof JL, editor: *Airway management: principles and practice*, St. Louis, 1996, Mosby, p 263.)

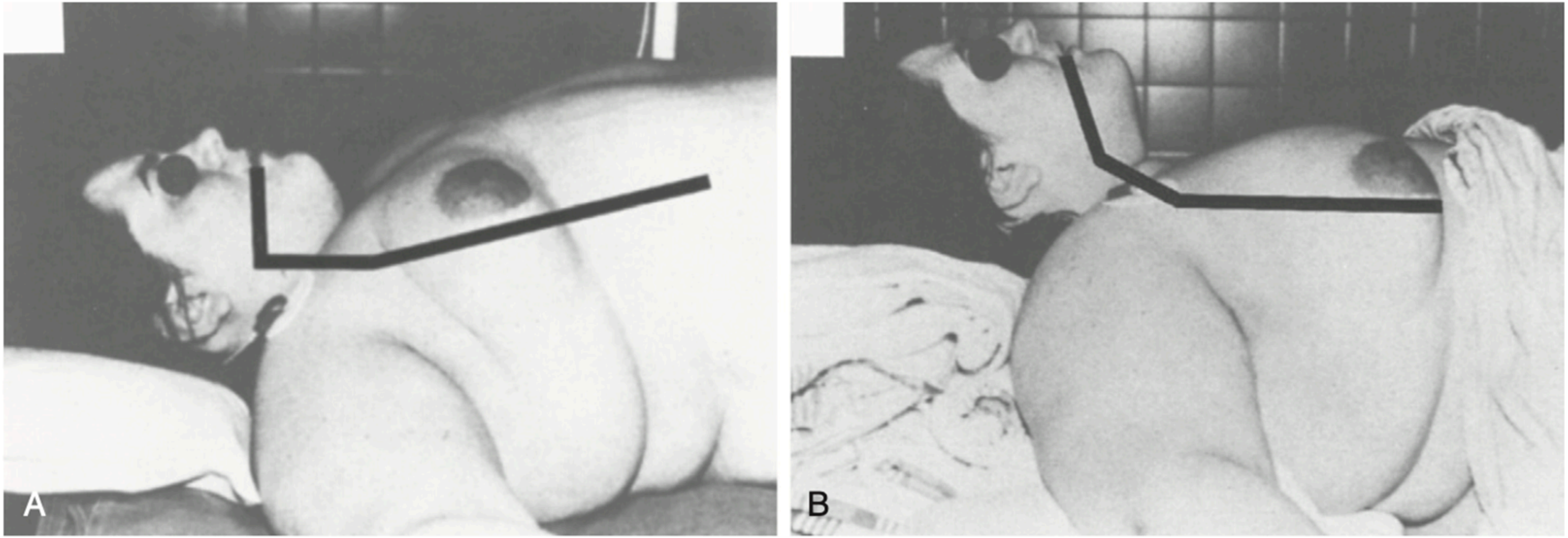
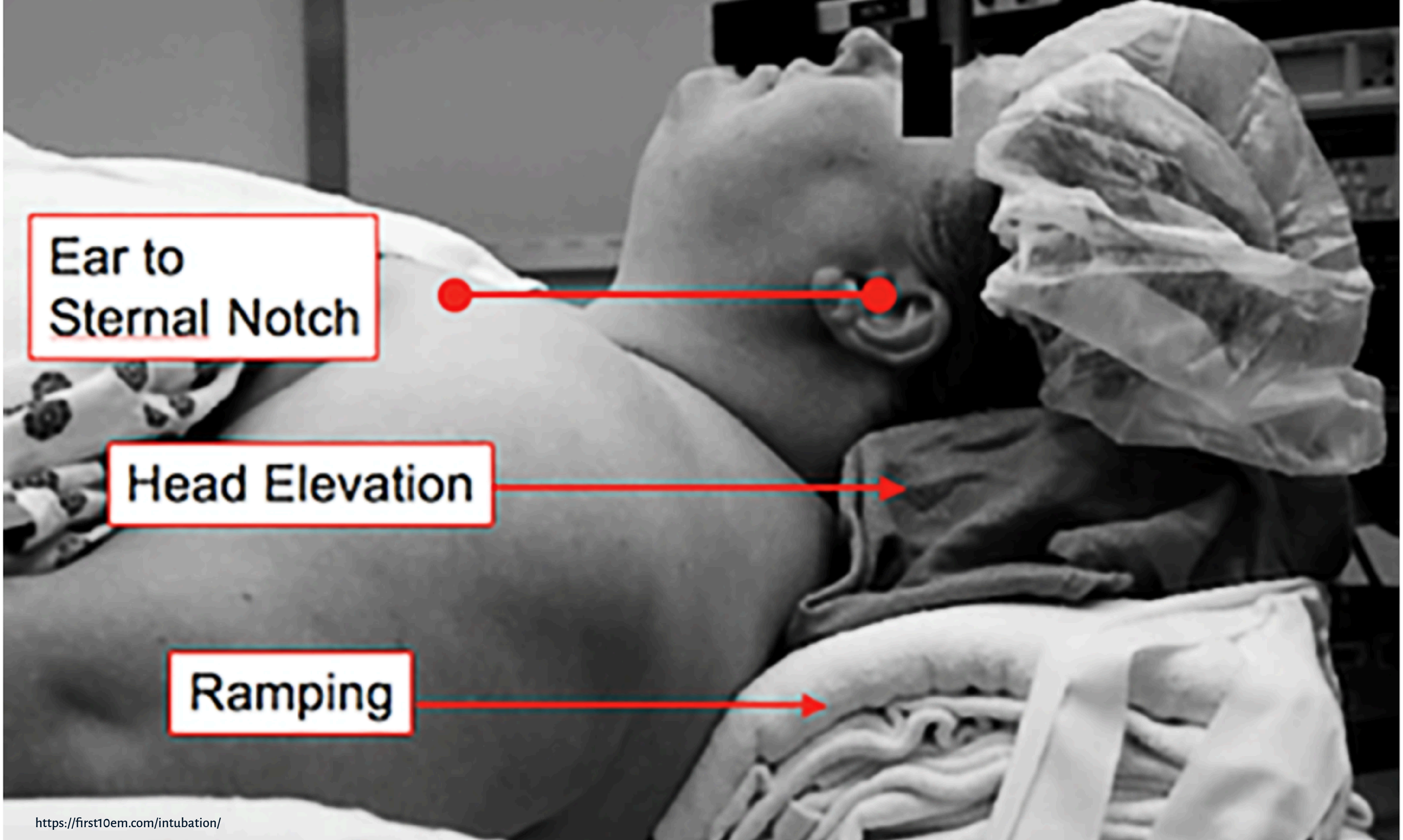


Figure 17-2 **A,** In some obese patients, placing the head on a pillow does not result in the sniffing position; in the obese patient shown and as illustrated by the overlying *bold black line*, the oral and laryngeal axes are perpendicular to one another, the neck is not flexed on the chest, and the head is not extended on the neck at the atlanto-occipital joint. **B,** In the same patient, placing support (e.g., blankets, towels) under the scapula, shoulders, nape of the neck, and head results in a much better sniffing position; the oral, pharyngeal, and laryngeal axes form only a slightly bent curve, the neck is flexed on the chest, and the head is extended on the neck at the atlanto-occipital joint. (From Benumof JL, editor: *Airway management: Principles and practice*, St. Louis, 1996, Mosby, p 264.)



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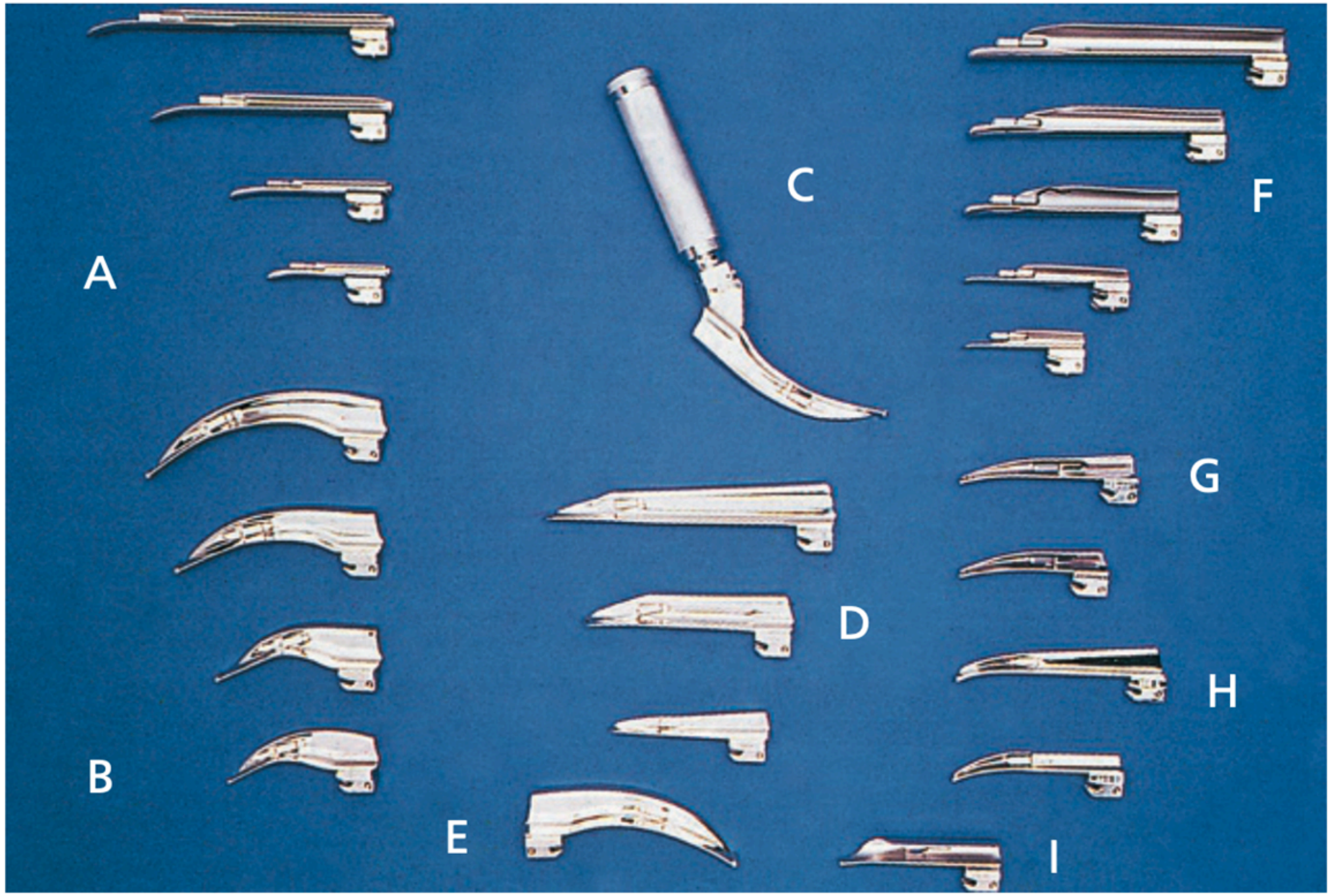
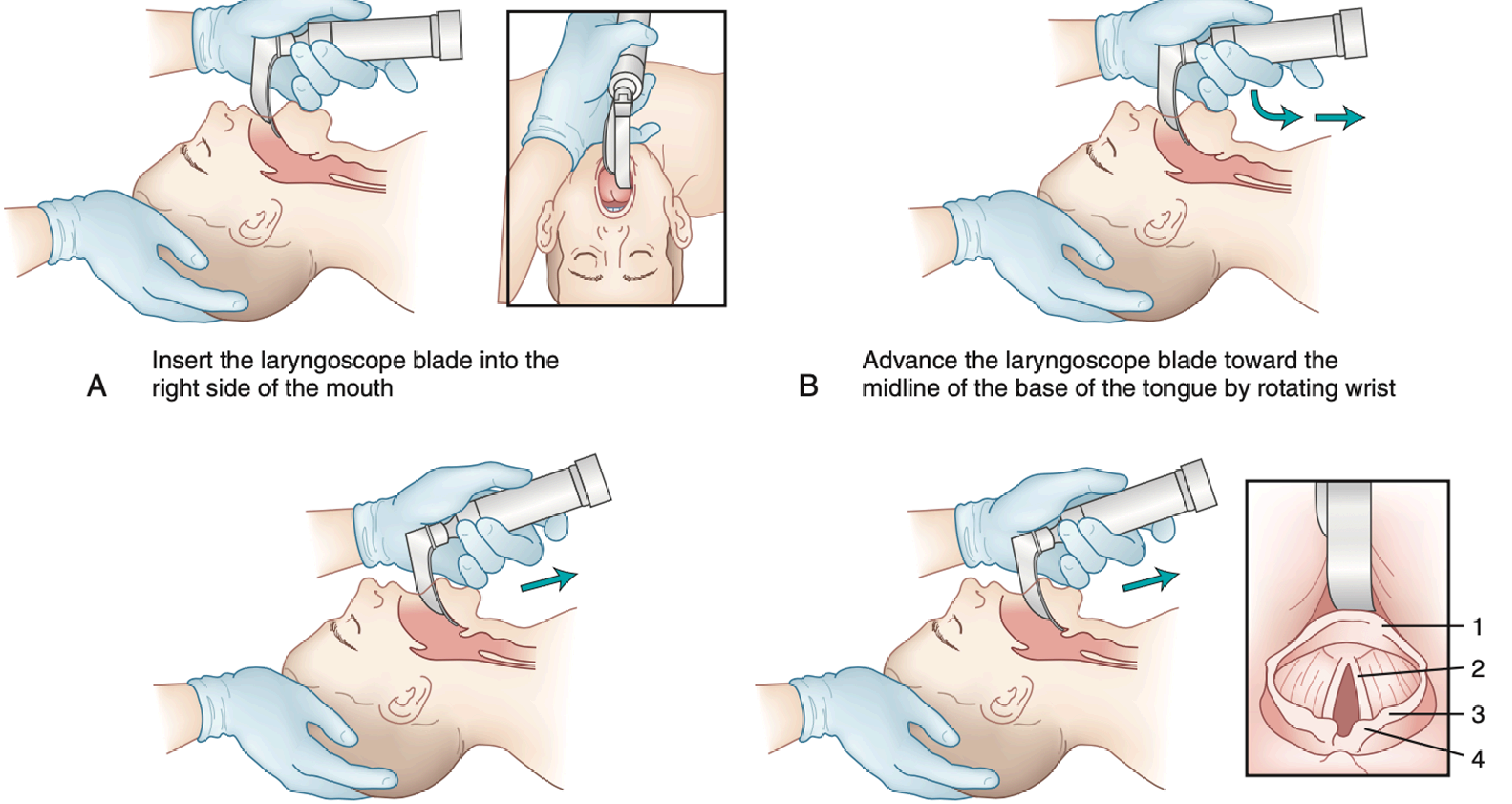


Fig. 7.2 A wide range of laryngoscope blades. (A) Miller blades (large, adult, infant, premature); (B) Macintosh blades (large, adult, child, baby); (C) Macintosh polio blade; (D) Soper blades (adult, child, baby); (E) left-handed Macintosh blade; (F) Wisconsin blades (large, adult, child, baby, neonate); (G) Robertshaw blades (infant, neonatal); (H) Seward blades (child, baby); (I) Oxford infant blade.



Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.


YouTube

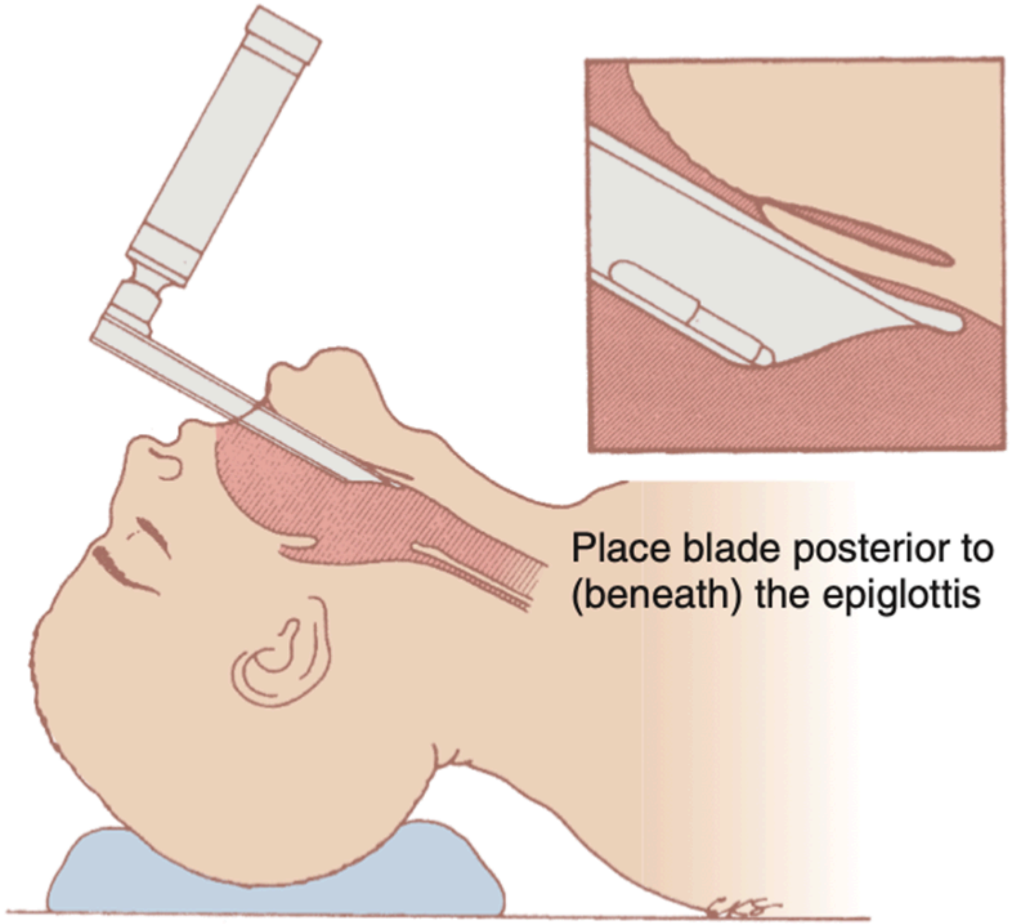


Figure 17-6 Conventional laryngoscopy with a straight blade. A straight laryngoscope blade (Miller blade) should be passed underneath the laryngeal surface of the epiglottis. The handle of the laryngoscope then should be elevated at a 45-degree angle, similar to the lifting that takes place with the use of a curved laryngoscope blade. (From Benumof JL, editor: *Airway management: Principles and practice*, St. Louis, 1996, Mosby, p 268.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

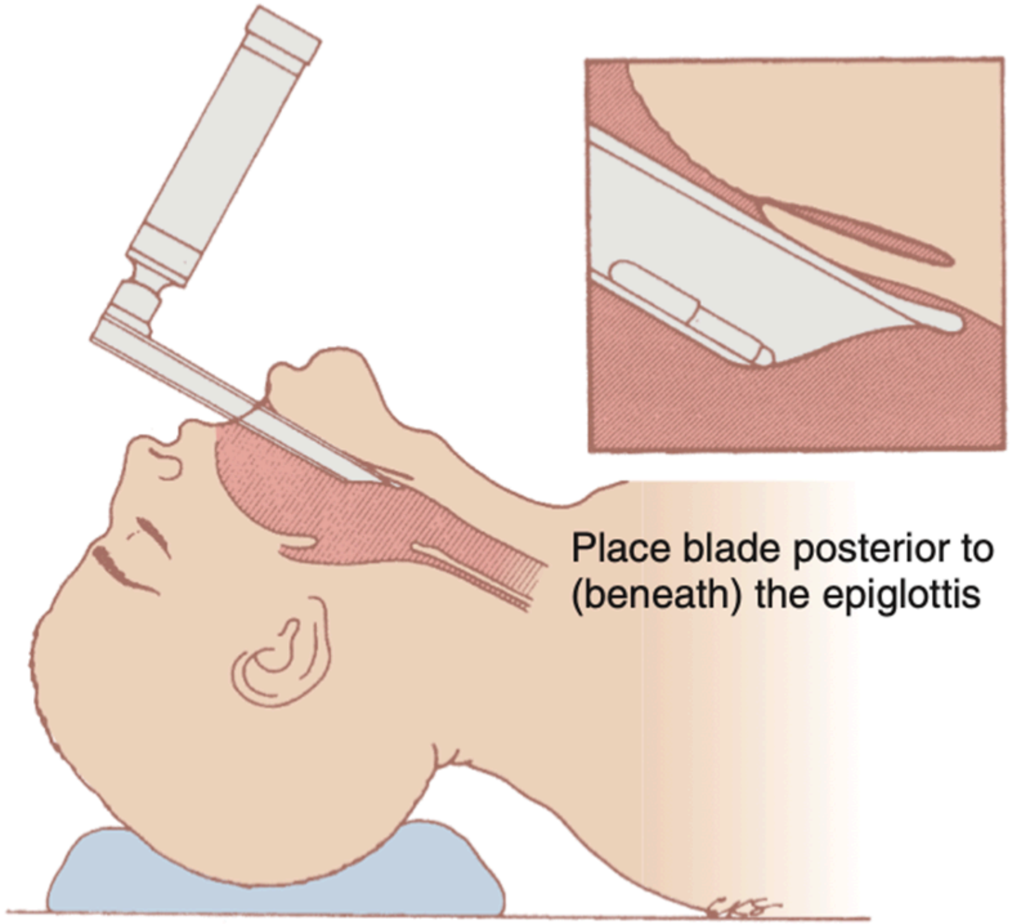


Figure 17-6 Conventional laryngoscopy with a straight blade. A straight laryngoscope blade (Miller blade) should be passed underneath the laryngeal surface of the epiglottis. The handle of the laryngoscope then should be elevated at a 45-degree angle, similar to the lifting that takes place with the use of a curved laryngoscope blade. (From Benumof JL, editor: *Airway management: Principles and practice*, St. Louis, 1996, Mosby, p 268.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

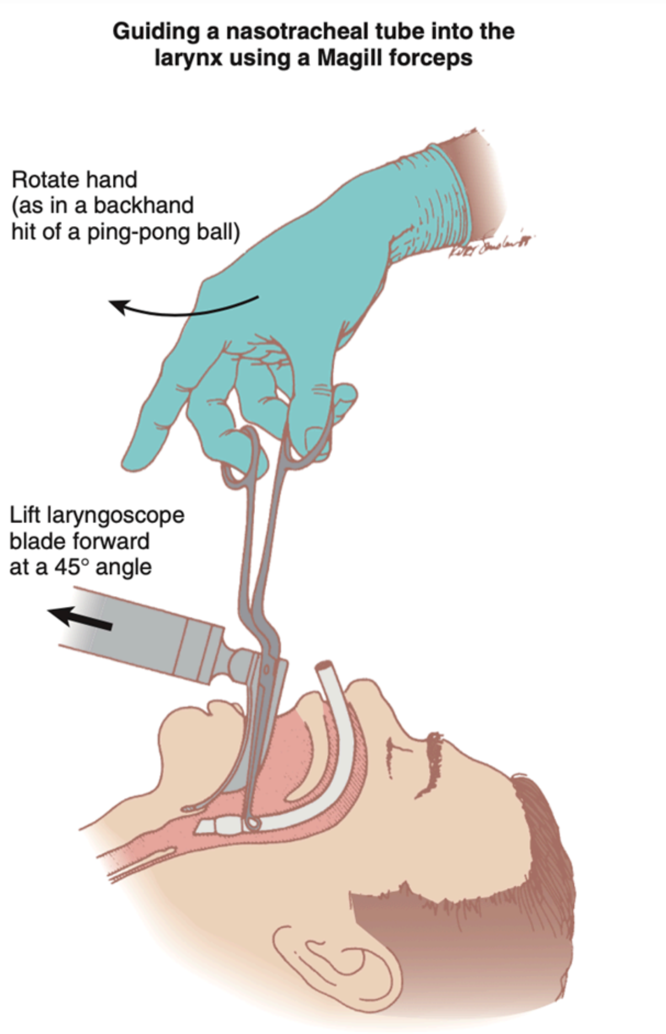


Figure 17-12 A nasotracheal tube can be guided under direct vision (laryngoscopic control) through the laryngeal aperture with a Magill forceps by rotating the hand as when using a backhand motion to hit a ping pong ball. The Magill forceps should grab the nasotracheal tube proximal to the cuff of the ETT. (From Benumof JL, editor: *Airway management: Principles and practice*, St. Louis, 1996, Mosby, p 275.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

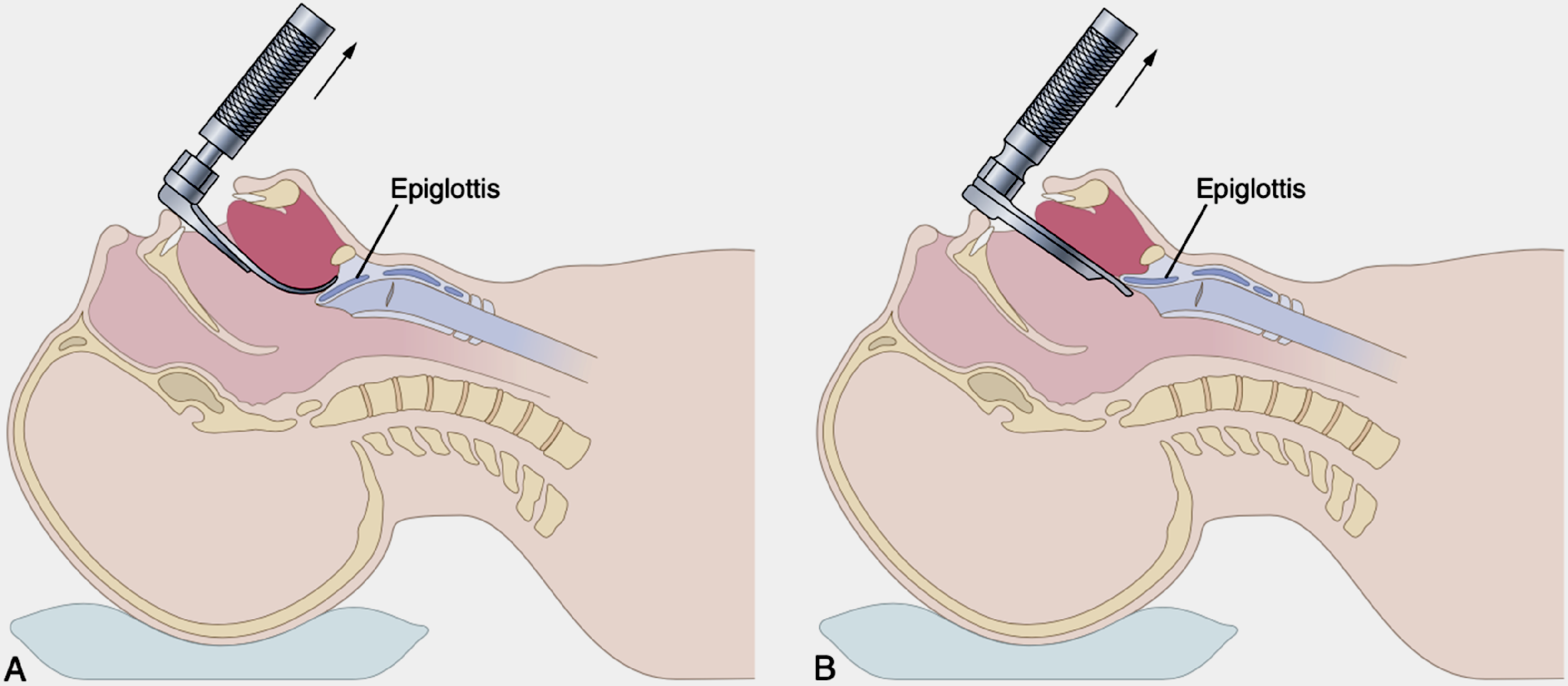


Fig. 16.13 Schematic diagram depicting the proper position of the laryngoscope blade for exposure of the glottic opening. (A) The distal end of the curved blade is advanced into the space between the base of the tongue and the pharyngeal surface of the epiglottis. (B) The distal end of the straight blade is advanced beneath the laryngeal surface of the epiglottis. Regardless of blade design, forward and upward movement exerted along the axis of the laryngoscope handle, as denoted by the arrows, serves to elevate the epiglottis and expose the glottic opening.

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.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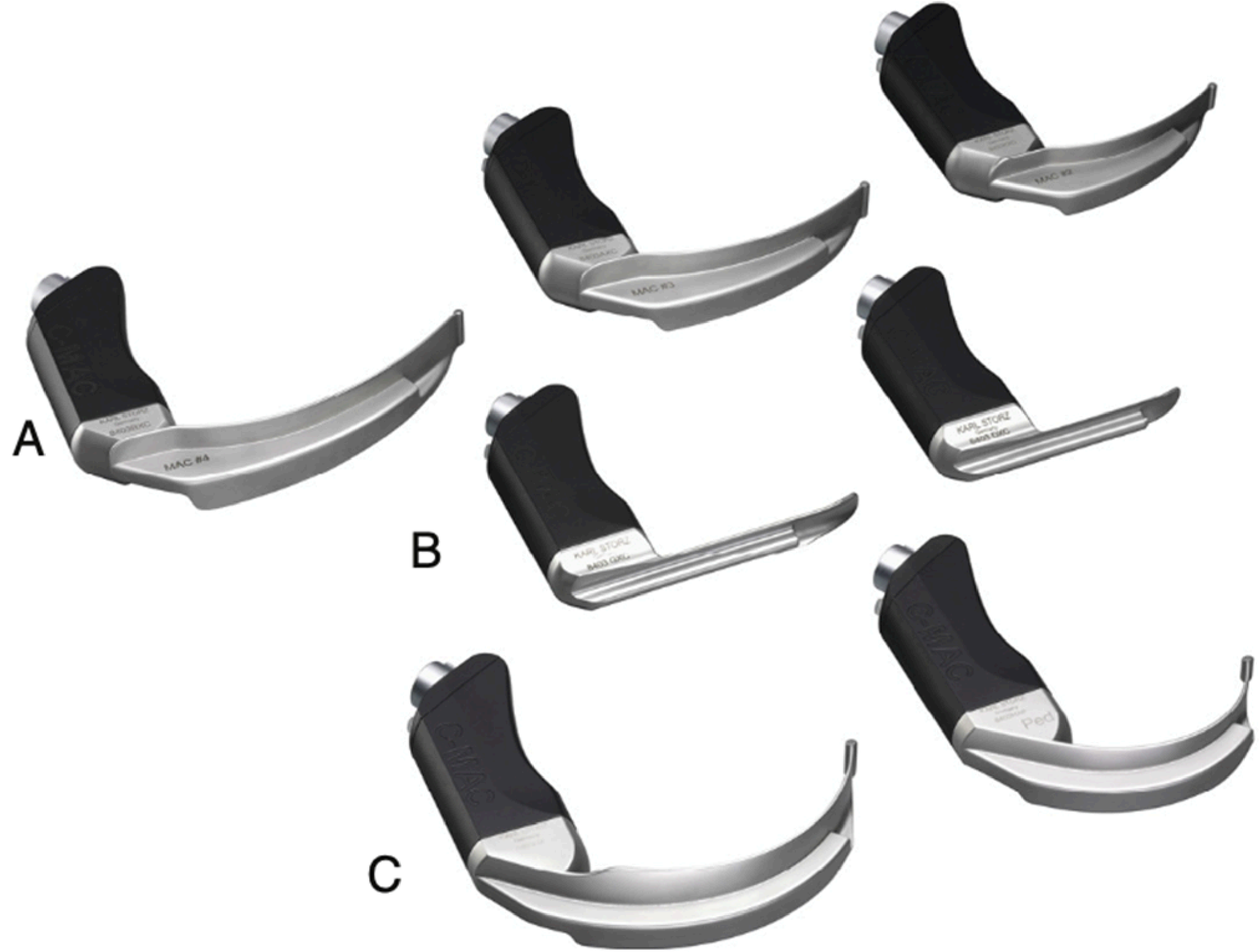


Fig. 16.16 Comparison of the different C-MAC blade types. (A) Macintosh style blade, (B) Miller style blade, and (C) D-blade. (Images courtesy of KARL STORZ Endoscopy, El Segundo, CA.)

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.

Box 16.2 Complications of Endotracheal Intubation

During Direct Laryngoscopy and Endotracheal Intubation

Dental and oral soft tissue trauma
Systemic hypertension and tachycardia
Cardiac dysrhythmias
Myocardial ischemia
Inhalation (aspiration) of gastric contents

While the Endotracheal Tube Is in Place

Endotracheal tube obstruction
Endobronchial intubation
Esophageal intubation
Endotracheal tube cuff leak
Pulmonary barotrauma
Nasogastric distention
Accidental disconnection from the anesthesia breathing circuit
Tracheal mucosa ischemia
Accidental extubation

Complications After Endotracheal Extubation

Laryngospasm
Inhalation (aspiration) of gastric contents
Pharyngitis (sore throat)
Laryngitis
Laryngeal or subglottic edema
Laryngeal ulceration with or without granuloma formation
Tracheitis
Tracheal stenosis
Vocal cord paralysis
Arytenoid cartilage dislocation

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.

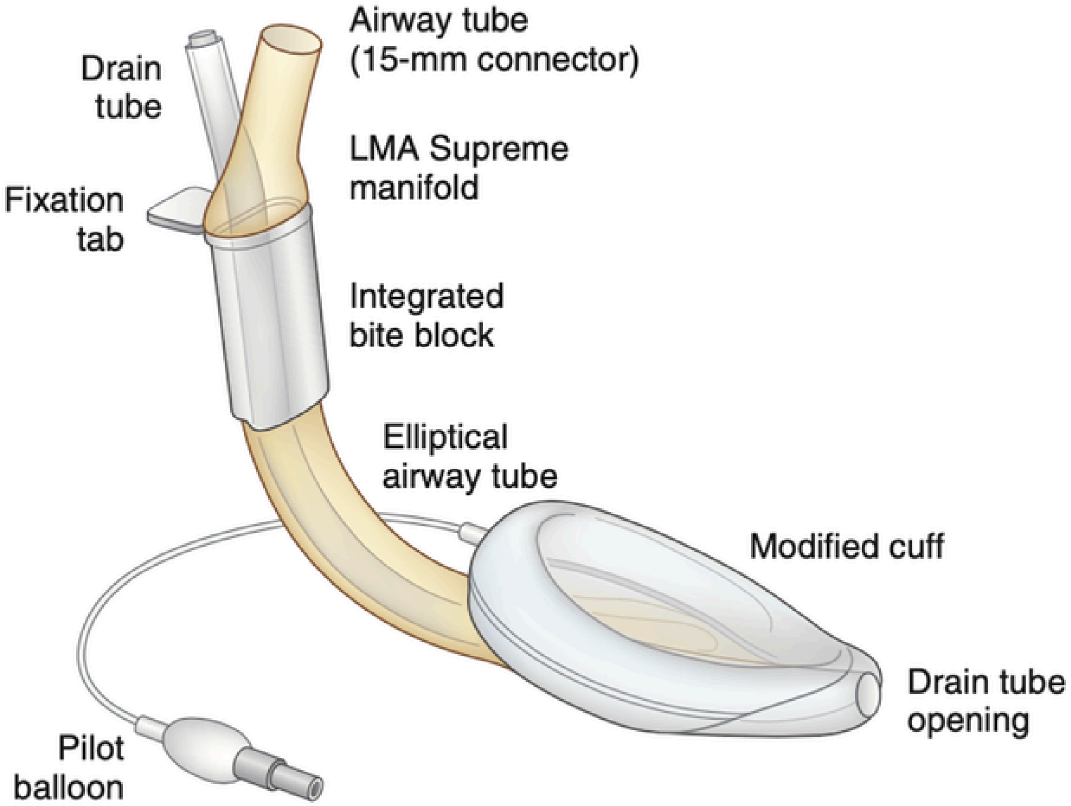


Figure 22-24 The LMA Supreme has a manifold with an integral bite block, an anatomically shaped airway tube enclosing a drain tube, a modified cuff through which the drain tube passes, and a cuff inflation line with pilot balloon.

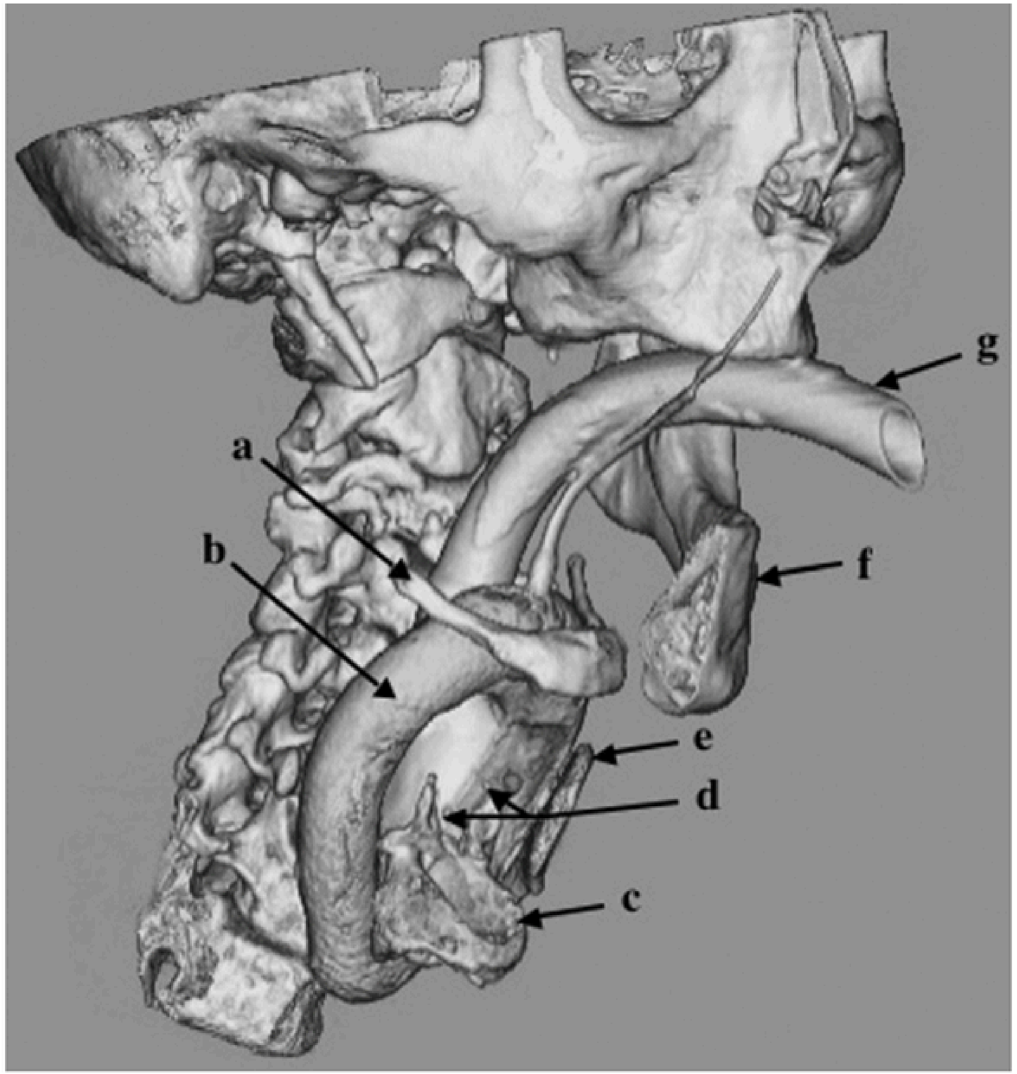


Figure 22-1 Three-dimensional radiologic reconstruction of the human airway with the laryngeal mask airway (LMA) in situ: hyoid bone *(a)*; LMA's cuff *(b)*; cricoid ring *(c)*; arytenoid cartilages *(d)*; thyroid cartilage *(e)*, which is digitally partially removed to demonstrate the position of the LMA; mandible *(f)*, which is digitally partially removed to demonstrate the position of the LMA; and the LMA's shaft *(g)*. The LMA's cuff forms a seal with the periglottic tissues and provides a continuous connection between the natural airway and the device.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

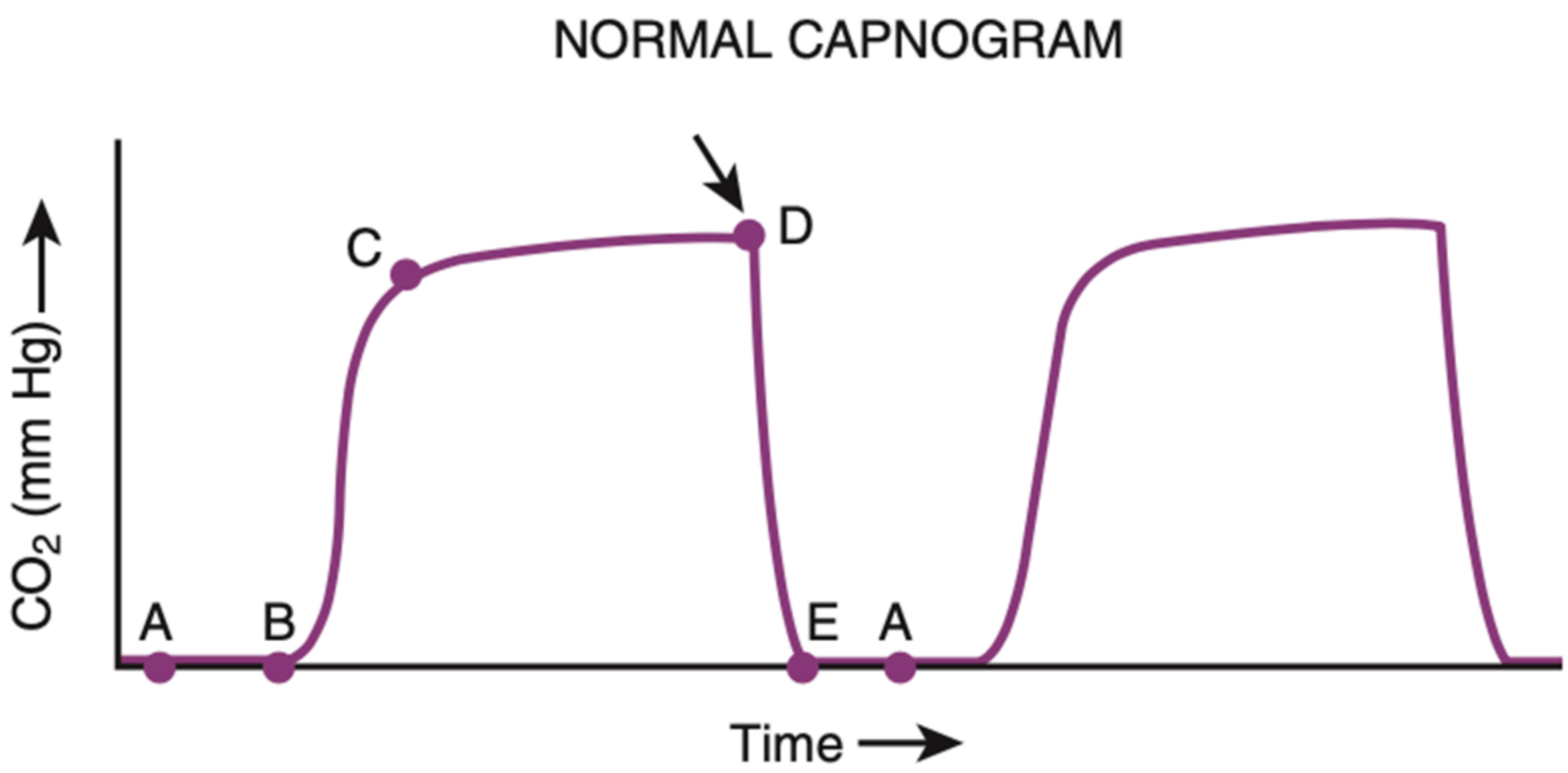
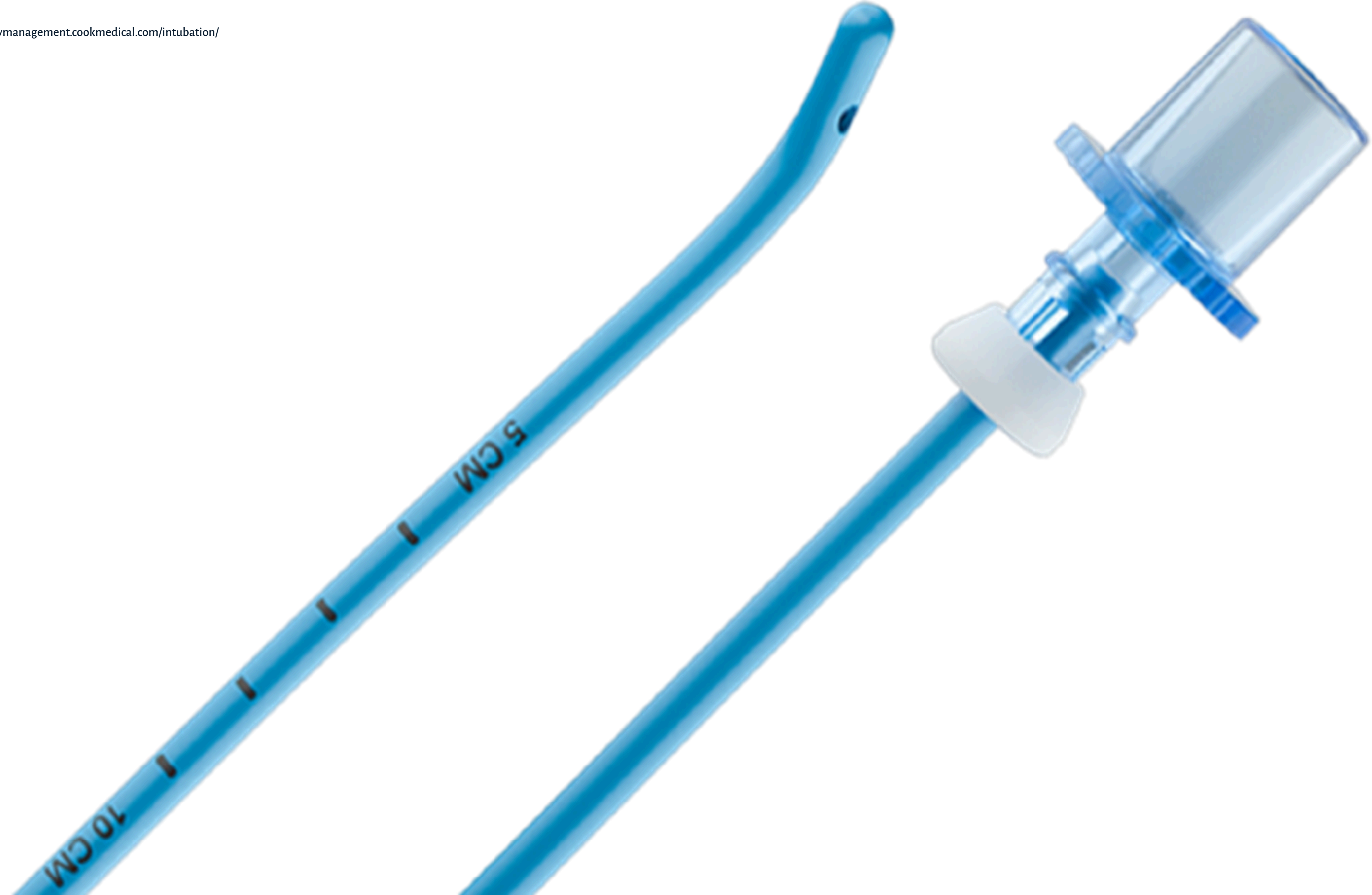


Figure 32-7 The CO_2 waveform. *A,* Expiratory pause begins. *A–B,* Clearance of anatomic dead space. *B–C,* Dead space air mixed with alveolar air. *C–D,* Alveolar plateau. *D,* End-tidal partial pressure of CO_2 registered by capnograph *(arrow)* and beginning of inspiratory phase. *D–E,* Clearance of dead space air. *E–A,* Inspiratory gas devoid of CO_2. (Modified from May WS, Heavner JE, McWorther D, Racz G: *Capnography in the operating room: An introductory directory,* New York, 1985, Raven Press, p 1.)

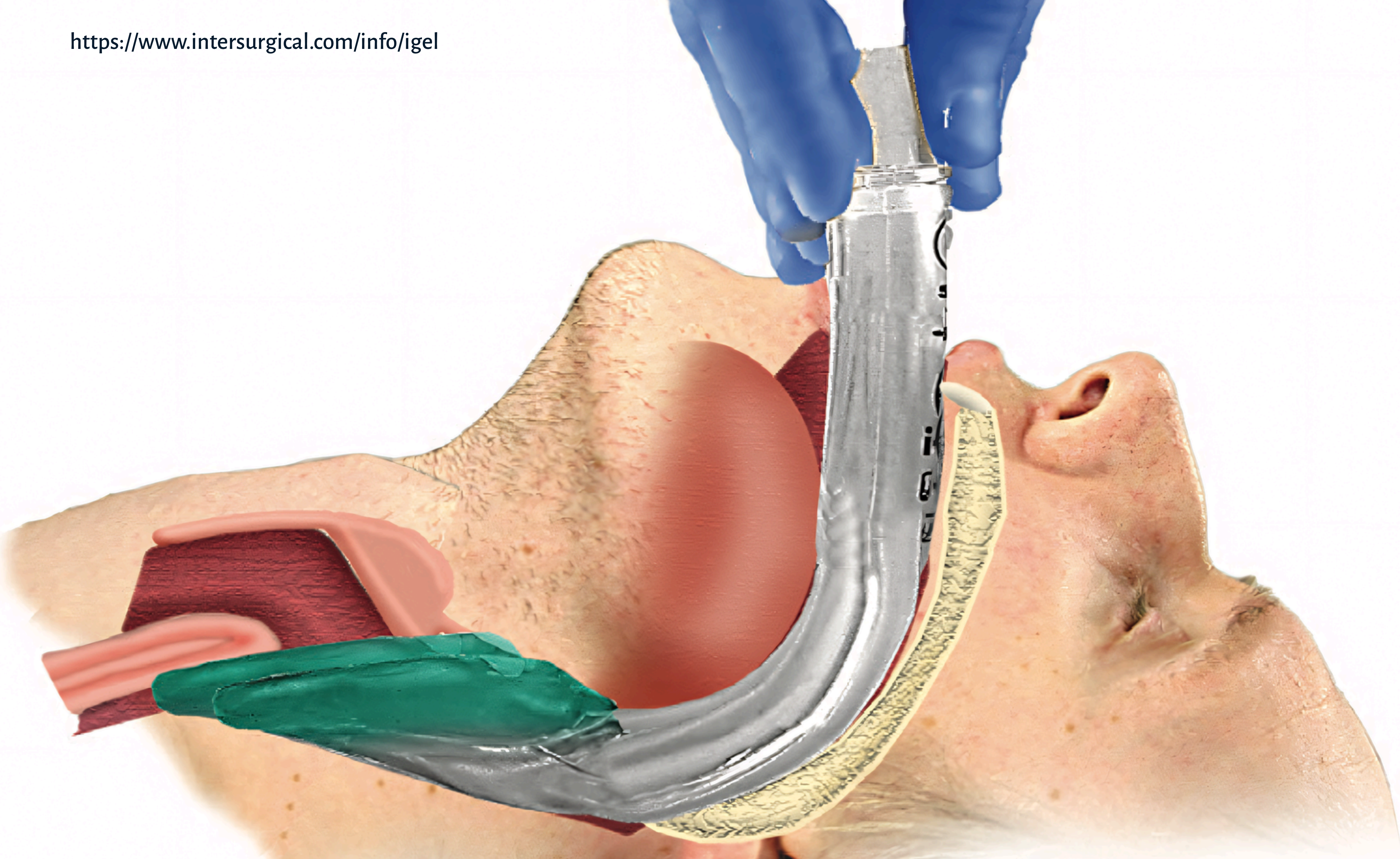
Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

https://airwaymanagement.cookmedical.com/intubation/




The operator manipulates the coude tip into the larynx.

https://www.intersurgical.com/info/igel



Preparations for use

Adult patient

1

Open the **i-gel** package, and on a flat surface take out the protective cradle containing the device.

2

Remove the **i-gel** and transfer it to the palm of the same hand that is holding the protective cradle, supporting the device between the thumb and index finger.

3

Place a small bolus of a water-based lubricant, such as K-Y Jelly, onto the middle of the smooth surface of the protective cradle in preparation for lubrication.

4

Grasp the **i-gel** with the opposite (free) hand along the integral bite block and lubricate the back, sides and front of the cuff with a thin layer of lubricant.

5

Place the **i-gel** back into the protective cradle in preparation for insertion.

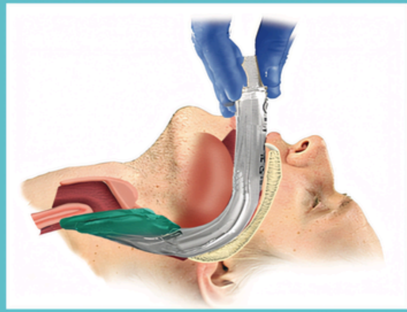
Step 6

Insertion technique

6

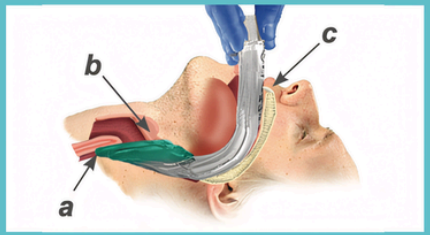
Remove the i-gel from the protective cradle. Grasp the lubricated i-gel firmly along the integral bite block. Position the device so that the **i-gel** cuff outlet is facing towards the chin of the patient. The patient should be in the 'sniffing the morning air' position with head extended and neck flexed. The chin should be gently pressed down before proceeding. Introduce the leading soft tip into the mouth of the patient in a direction towards the hard palate.

7



Glide the device downwards and backwards along the hard palate with a continuous but gentle push until a **definitive resistance** is felt .

8

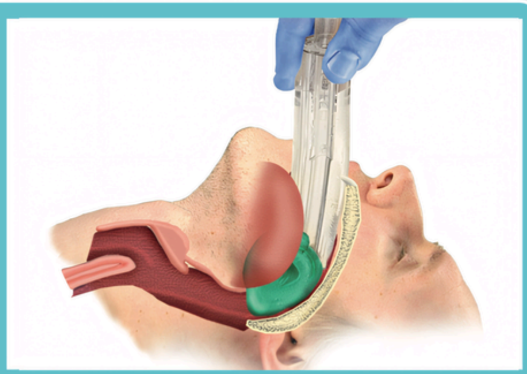


The tip of the airway should be located into the upper oesophageal opening (a) and the cuff should be located against the laryngeal framework (b). The incisors should be resting on the integral bite-block (c).

9

i-gel should be taped down from 'maxilla to maxilla'.

10



If there is early resistance during insertion a 'jaw thrust' (above) or 'Insertion with Deep Rotation' (right) is recommended.

AIRWAYS-2 Trial: Airway Management in Out-of-Hospital Cardiac Arrest

Trial Participants

2,041 paramedics from 4 NHS ambulance trusts expressed interest, and 1,523 were recruited and randomized (764 to tracheal intubation, 759 to i-gel)

Attended Cardiac Arrests

73,893 out-of-hospital cardiac arrests were attended, and 29,733 (40.2%) patients received resuscitation, of which 13,462 (45.3%) were attended by AIRWAYS-2 paramedics

Eligible Patients

9,296 eligible patients were attended by 1,382 trial paramedics, with 7 patients missing primary outcome data

Airway Management

7,580 patients received advanced airway management, with 2,840 receiving tracheal intubation first, 4,632 receiving i-gel first, and 108 receiving a non-i-gel supraglottic airway device first

Outcomes

Similar proportions of patients in the two treatment groups had a favorable functional outcome (modified Rankin Scale score) at 30 days/hospital discharge (tracheal intubation group, 6.8%; i-gel group, 6.4%)

Economic Evaluation

Mean quality-adjusted life-years to 6 months were 0.03 in both groups, with total costs of care from out-of-hospital cardiac arrest up to 6 months being £3,570 and £3,413 in the i-gel and tracheal intubation groups, respectively

Benger JR, Kirby K, Black S, et al. Supraglottic airway device versus tracheal intubation in the initial airway management of out-of-hospital cardiac arrest: the AIRWAYS-2 cluster RCT. Health Technology Assessment. 2022;26(21):1-158. doi:10.3310/VHOH9034

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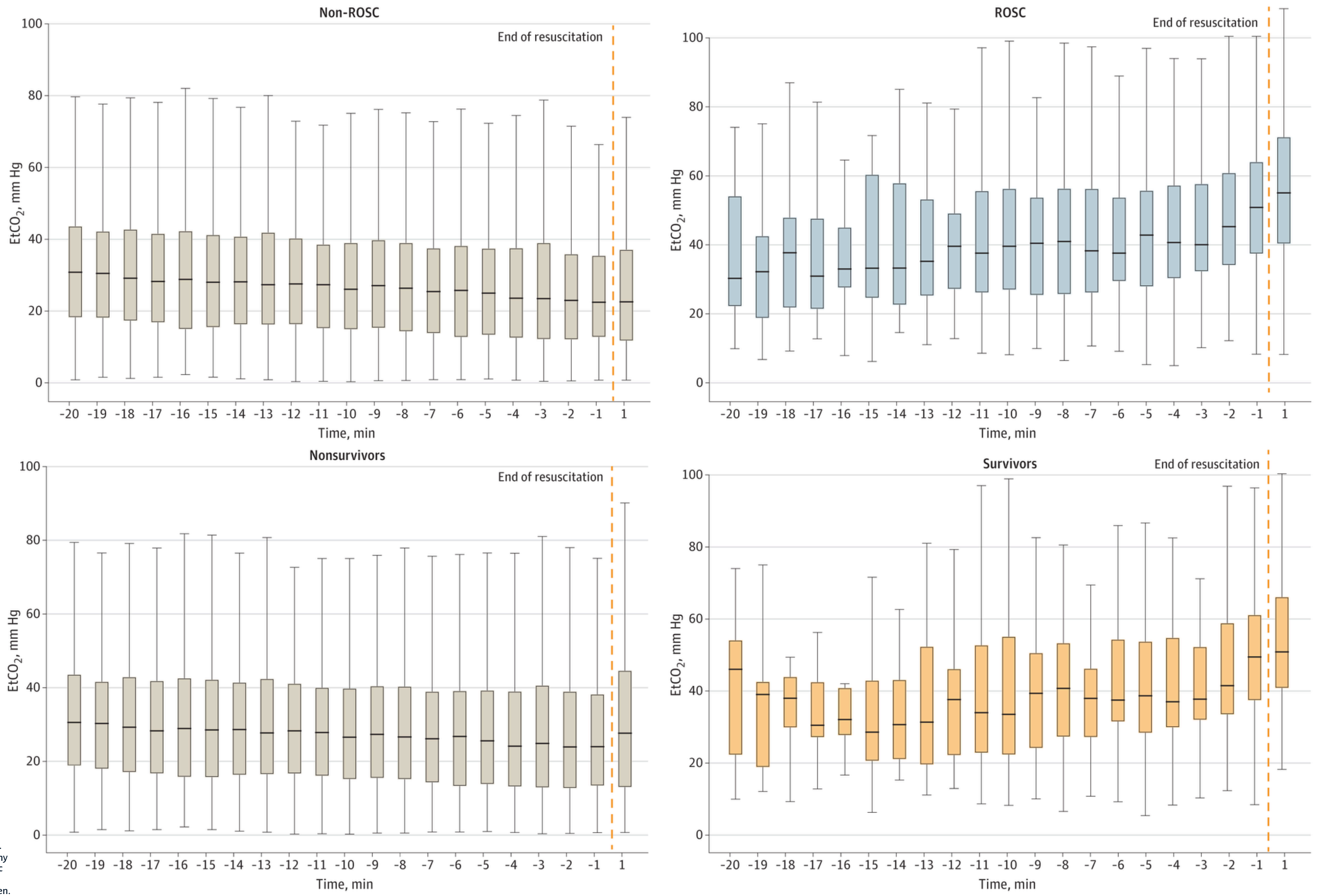

Nassal MMJ, Elola A, Aramendi E, et al. Temporal trends in end-tidal capnography and outcomes in out-of-hospital cardiac arrest: a secondary analysis of a randomized clinical trial. JAMA Netw Open. 2024;7(7):e2419274. doi:10.1001/jamanetworkopen.2024.19274

Leveraging Dynamic Capnography in OHCA Resuscitation

Limitations of Discrete EtCO2 Values

Prior studies relied on simple approaches like discrete EtCO2 values, which showed variable and challenging interpretation during resuscitation. Even patients without ROSC had unexpectedly higher EtCO2 values, and some ROSC cases had values below 10 mmHg.

Advantages of Continuous Capnography

Continuous capnography can account for waveform variability and monitor changes during resuscitation. Automated signal processing enables tracking of EtCO2 value change and rate of change, which can be correlated with outcomes.

Temporal Increases in EtCO2 Predict Outcomes

This study found that while early discrete EtCO2 values did not differentiate outcomes, temporal increases in EtCO2 over time were associated with both ROSC and survival. Dynamic changes in EtCO2 capnography may be a better predictor of OHCA outcome.

Clinical Implications

Using the change in capnography throughout resuscitation may be an advancement over using discrete EtCO2 cut-offs. Validation of this approach (EtCO2 trend monitoring) can be useful in resuscitation and merits independent validation prior to clinical application.

Nassal MMJ, Elola A, Aramendi E, et al. Temporal trends in end-tidal capnography and outcomes in out-of-hospital cardiac arrest: a secondary analysis of a randomized clinical trial. JAMA Netw Open. 2024;7(7):e2419274. doi:10.1001/jamanetworkopen.2024.19274

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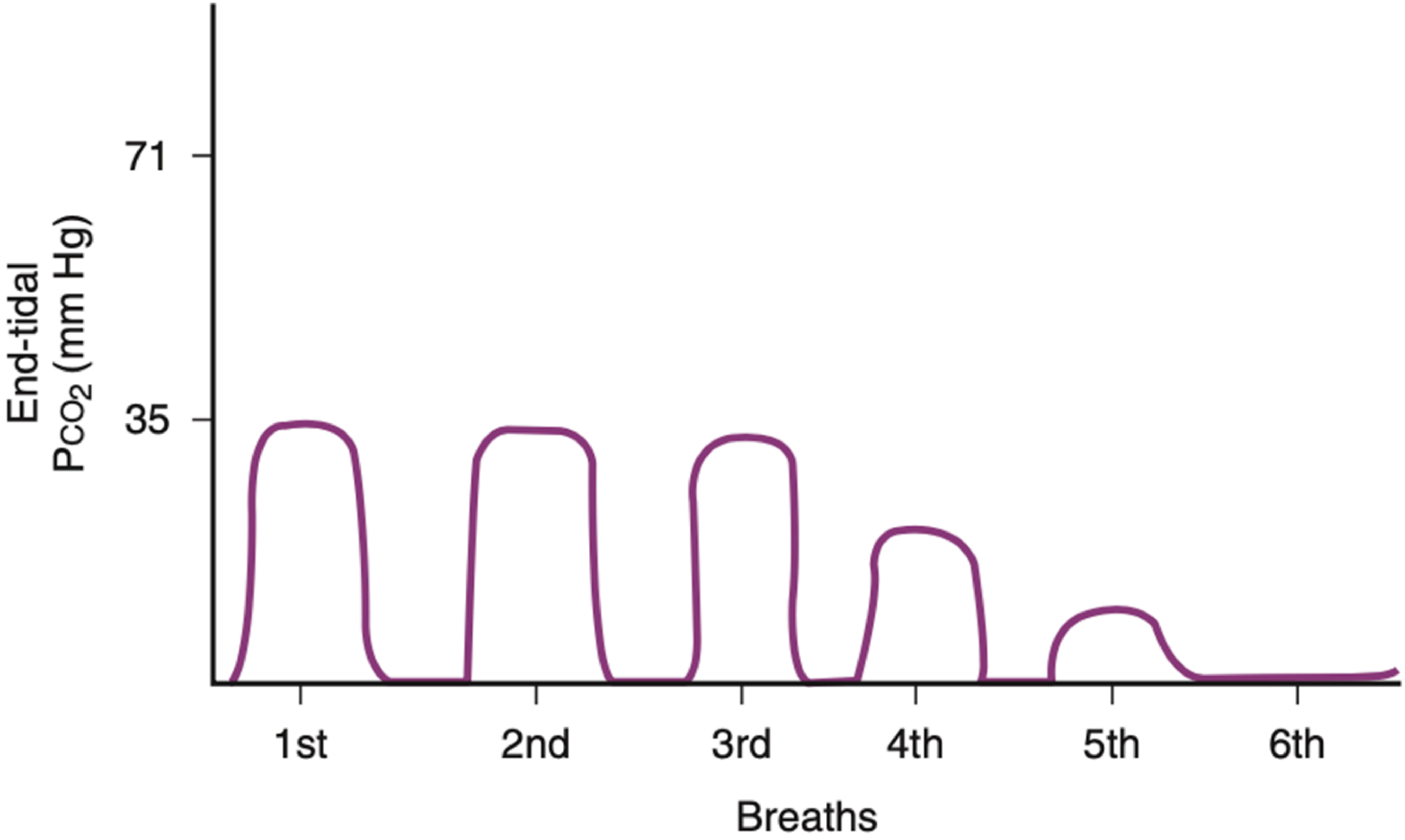

71
35
End-tidal
P_{CO_2} (mm Hg)
1st
2nd
3rd
4th
5th
6th
Breaths

Section 9

The external chest compressions

CLOSED-CHEST CARDIAC MASSAGE

W. B. Kouwenhoven, Dr. Ing., James R. Jude, M.D.
and
G. Guy Knickerbocker, M.S.E., Baltimore

Cardiac resuscitation after cardiac arrest or ventricular fibrillation has been limited by the need for open thoracotomy and direct cardiac massage. As a result of exhaustive animal experimentation a method of external transthoracic cardiac massage has been developed. Immediate resuscitative measures can now be initiated to give not only mouth-to-nose artificial respiration but also adequate cardiac massage without thoracotomy. The use of this technique on 20 patients has given an over-all permanent survival rate of 70%. Anyone, anywhere, can now initiate cardiac resuscitative procedures. All that is needed are two hands.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173(10):1064-1067. doi:10.1001/jama.1960.03020280004002

History of External Chest Compressions

1878

Boehm's closed-chest method: Rhythmic chest pressure in cats produced striking blood pressure changes.

1920s

Tournade and colleagues: Abrupt chest compression in dogs produced blood pressures of 60-100 mmHg during cardiac arrest.

1930s

Killick and Eve: Rocking technique of artificial respiration changed atrial blood pressure from 38 to 76 mmHg.

1947

Gurvich and Yuniev: Capacitor discharge through the chest resumed cardiac function in dogs, extended by rhythmic chest pressure.

1950s

Rainer and Bullough: Resuscitation of children by lowering the head, flexing the legs and buttocks against the chest.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173(10):1064-1067. doi:10.1001/jama.1960.03020280004002

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Closed-Chest Cardiac Massage

- **Experimental Trials**

  More than 100 dogs were used to test various methods of moving blood by massaging the intact chest. A safe and effective method of

- **Physiological Monitoring**

  Simultaneous recording of blood flow in the carotid artery, blood pressure in the femoral artery, and the electrocardiogram showed that closed-chest cardiac massage could maintain vigorous blood flow and blood pressure during ventricular fibrillation.

- **Defibrillation Effectiveness**

  A closed-chest defibrillating shock was found to immediately restore normal sinus rhythm in the dogs with ventricular fibrillation, demonstrating the effectiveness of this technique.

- **Applying the Technique in Humans**

  The method of closed-chest cardiac massage is simple to apply and requires only the human hand. The technique compresses the heart between the sternum and the vertebral column, forcing out blood during the compression and allowing the heart to refill during relaxation.

- **Positioning and Technique**

  The patient should be in a supine position on a rigid support. The heel of one hand, with the other hand on top, is placed on the sternum just above the xiphoid. Firm, vertical pressure is applied about 60 times per minute, moving the sternum 3-4 cm towards the spine.

- **Combined with Rescue Breathing**

  If there are two or more persons present, one should perform the closed-chest cardiac massage while the other provides mouth-to-nose respiration, ensuring both circulation and ventilation.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173(10):1064-1067. doi:10.1001/jama.1960.03020280004002

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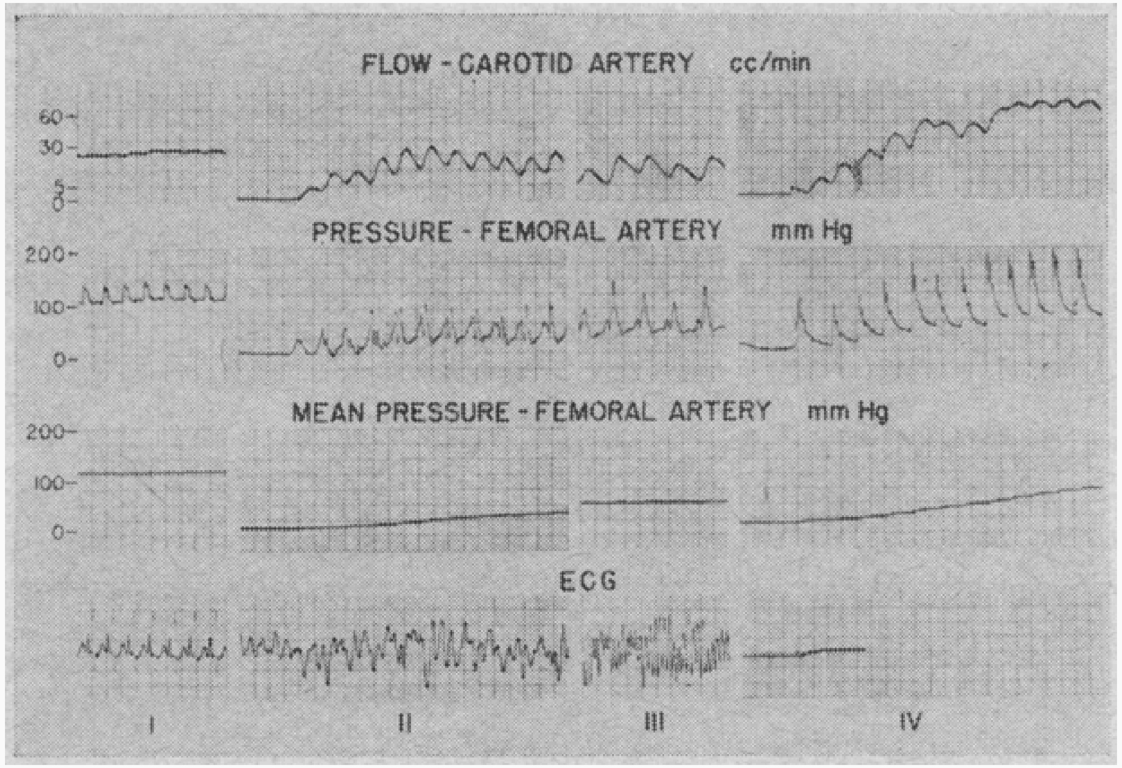


Fig. 1.—Record of blood flow, pressures, and electrocardiogram of dog whose heart was in ventricular fibrillation for eight minutes. I: normal initial values; II: start of closed-chest massage; III: seventh minute of massage; IV: closed-chest defibrillation.

Fig 2.—Position of hands during massage of adult.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA. 1960;173(10):1064-1067. doi:10.1001/jama.1960.03020280004002

About nine months prior to the time of writing, at Johns Hopkins Hospital, the clinical application of closed-chest cardiac massage was successfully illustrated in a case of cardiac arrest.

Initially, it was felt that the method might be useful in treating arrest in children, whose ribs are known to be flexible, but that it would not be effective in adults. This latter assumption was proved to be incorrect, since the chest of an unconscious adult was found to be remarkably flexible. During the 10 months prior to writing, this method alone has been applied on 20 patients aged from 2 months to 80 years. In 13 of these patients, artificial respiration was applied simultaneously with the massage; the duration of the massage varied from less than 1 minute to 65 minutes.

In seven cases, records were obtained of either the blood pressure or the electrical activity of the heart (by electrocardiogram) during the episode. Systolic pressure is shown in Figure 3, which displays the blood pressures recorded on an adult.

The hearts of 3 of the 20 patients treated were in ventricular fibrillation, and all were defibrillated by a closed-chest A.C. defibrillator shock. All 20 patients were resuscitated, and at the time of writing, 14 of them are alive without central nervous system damage and without undergoing thoracotomy.

Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. *JAMA*. 1960;173(10):1064-1067. doi:10.1001/jama.1960.03020280004002

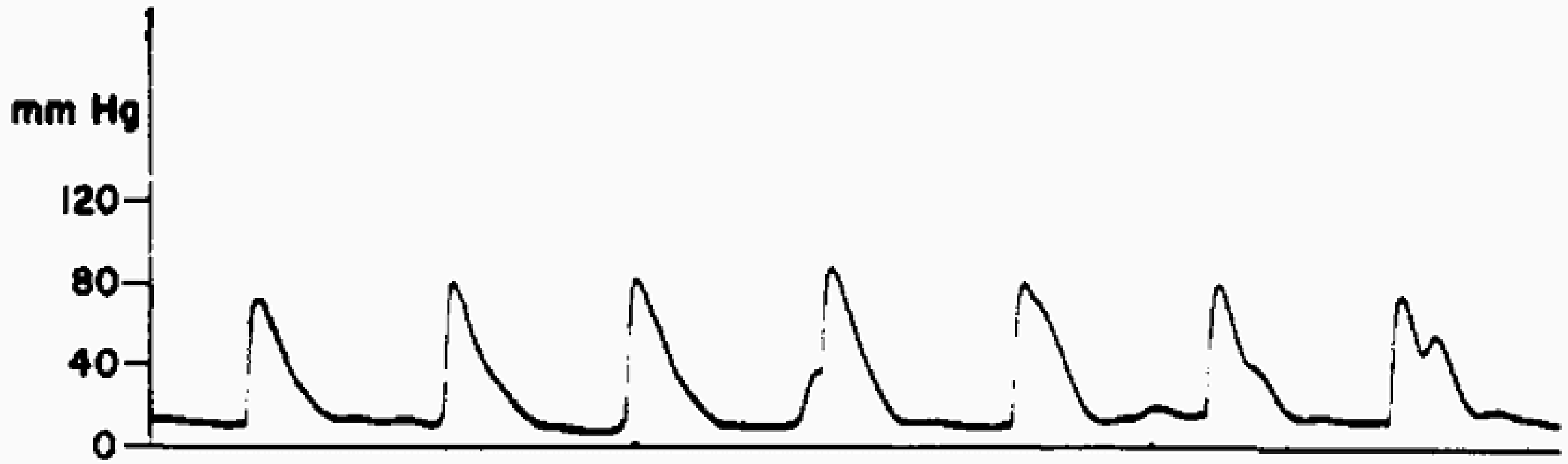
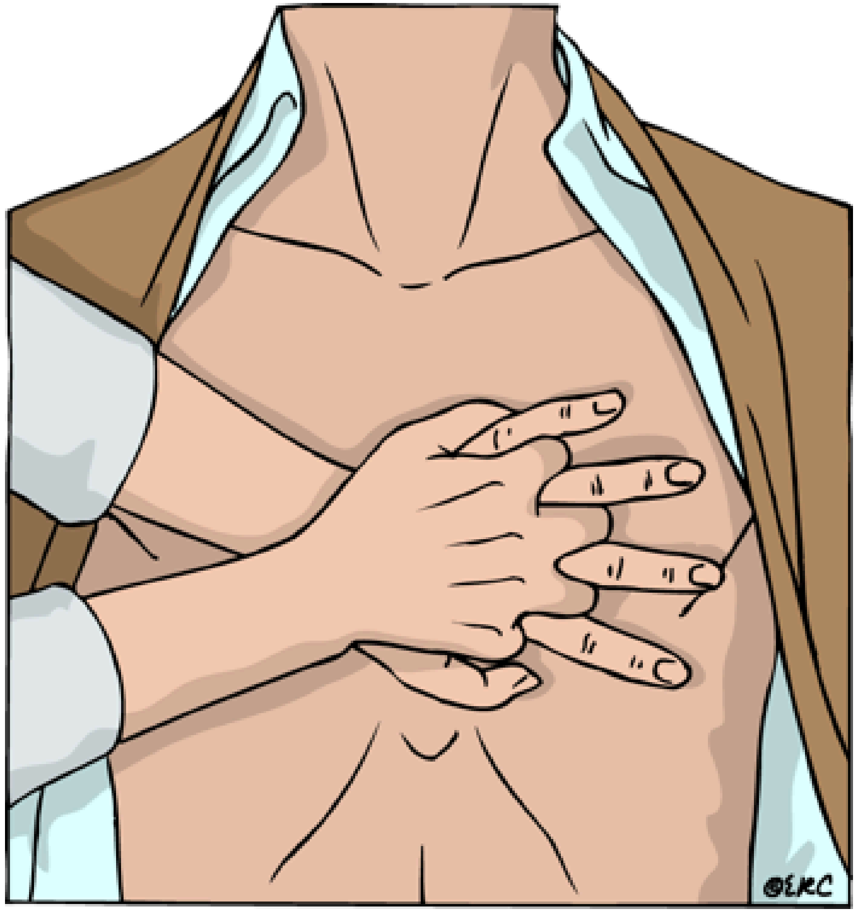


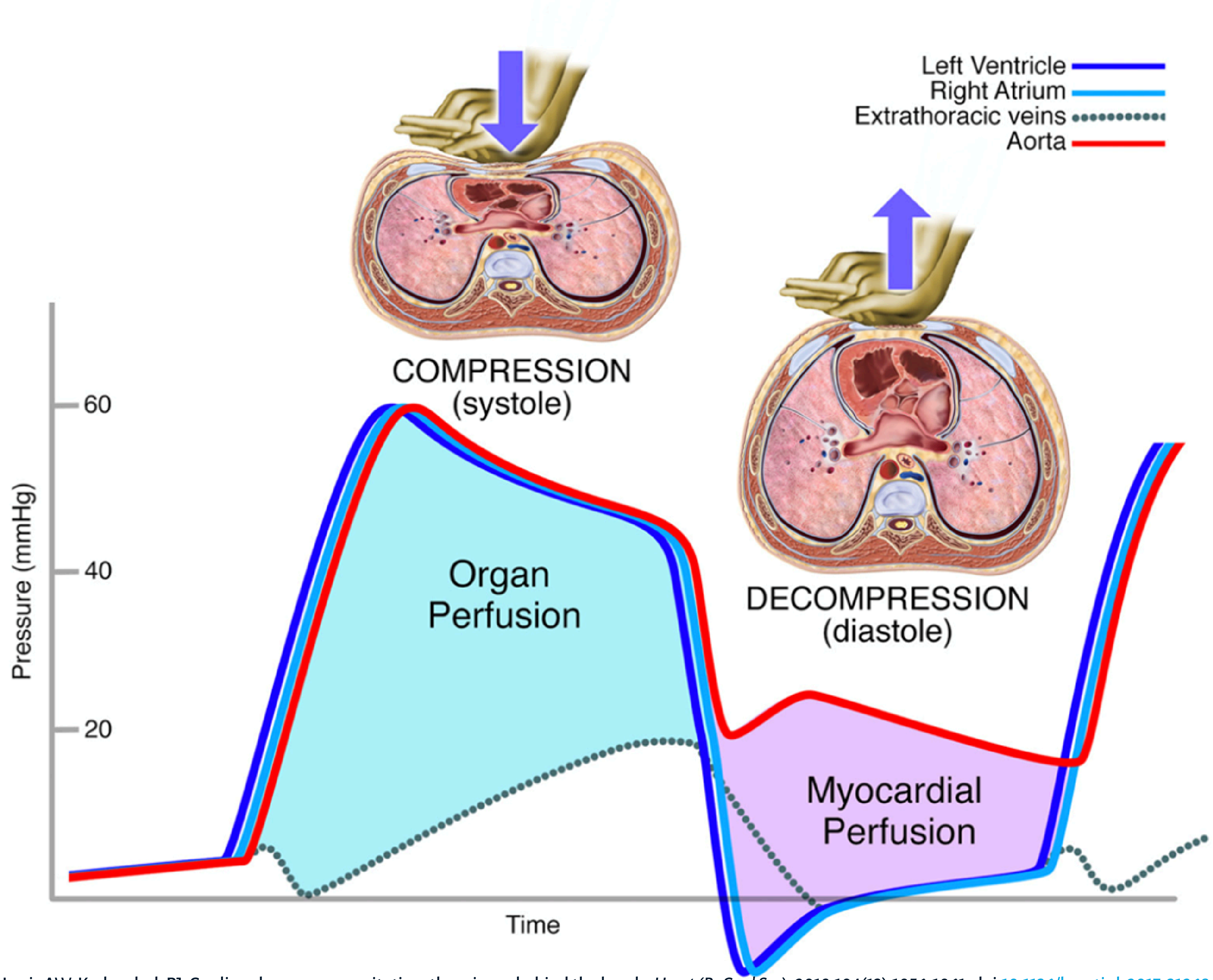
Fig. 3.—Blood pressure produced in an adult by closed-chest cardiac massage.

High quality chest compressions

- Start chest compressions as soon as possible.
- Deliver compressions on the lower half of the sternum ('in the centre of the chest').
  Compress to a depth of at least 5 cm but not more than 6 cm.
- Compress the chest at a rate of 100–120 min^{-1} with as few interruptions as possible.
- Allow the chest to recoil completely after each compression; do not lean on the chest.
- Perform chest compressions on a firm surface whenever feasible.



Olasveengen TM, Semeraro F, Ristagno G, et al. European Resuscitation Council Guidelines 2021: Basic Life Support. Resuscitation. 2021;161:98-114. doi:10.1016/j.resuscitation.2021.02.009



Harris AW, Kudenchuk PJ. Cardiopulmonary resuscitation: the science behind the hands. *Heart (Br Card Soc)*. 2018;104(13):1056-1061. doi:10.1136/heartjnl-2017-312696

Mechanics of Conventional CPR

- **Compression Phase**
  External chest compression results in arterial blood flow due to phasic changes in intrathoracic pressure produced by the force generated by the hands.

- **Chest as a Pump**
  In closed-chest CPR, the increased intrathoracic pressure and reduced thoracic volume created by chest compressions squeeze the heart itself and all blood-containing structures in the chest, including the lungs and great vessels.

- **Uniform Pressure Rise**
  Chest compression leads to a relatively uniform rise in the pressure of all intrathoracic vascular structures, and it is this pressure that ejects blood from the thorax during this phase of CPR.

- **Venous Valves**
  Thoracic pressure is transmitted to the body via the great arteries, but fortunately not via the great veins, where this is prevented by the closure of venous valves at the thoracic outlet.

- **Cardiac Output and Cerebral Blood Flow**
  The greater the vigor of these chest compressions, the greater the resulting cardiac (actually thoracic) output and magnitude of cerebral blood flow.

Harris AW, Kudenchuk PJ. Cardiopulmonary resuscitation: the science behind the hands. *Heart (Br Card Soc)*. 2018;104(13):1056-1061. doi:10.1136/heartjnl-2017-312696

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The Importance of Diastole during CPR

Diastole during CPR

Diastole occurs during the decompression phase of the CPR cycle, when the thorax is permitted to rebound to its normal fully expanded configuration.

Intrathoracic Vacuum

The intrathoracic vacuum created by the recoiling thoracic cage draws blood back into the chest from the periphery, filling the heart, lungs, and great vessels in preparation for the next chest compression.

Coronary Perfusion Pressure (CPP)

During the compression phase of CPR, all intrathoracic pressures equalize, resulting in no coronary blood flow. In CPR diastole, the higher aortic pressure above its closed valve compared to the falling intrathoracic pressure results in a positive CPP, perfusing the heart.

The diastole phase of CPR is critical for maintaining organ perfusion, including the heart itself, through the creation of an intrathoracic vacuum and the restoration of positive coronary perfusion pressure.

Harris AW, Kudenchuk PJ. Cardiopulmonary resuscitation: the science behind the hands. Heart. 2018;104(13):1056. doi:10.1136/heartjnl-2017-312696

Importance of Full Chest Recoil during CPR

- **Importance of Full Chest Recoil**

  Permitting complete chest recoil during the decompression phase of CPR is essential for refilling the chest and adequate myocardial perfusion.

- **Consequences of Incomplete Recoil**

  Incomplete chest recoil or 'leaning' results in an increase in intrathoracic pressure when it needs to be at its minimum. This can decrease coronary perfusion pressure by more than a third and cerebral perfusion by over 50%.

- **Causes of Leaning**

  The problem often arises due to fatigue, where the chest compressor unintentionally uses the patient's chest as a resting table during the decompression phase.

- **Prevalence of Leaning**

  An observational study found leaning, defined as 5 pounds of residual pressure left on the chest, in 91% of in-hospital cardiac arrest resuscitations.

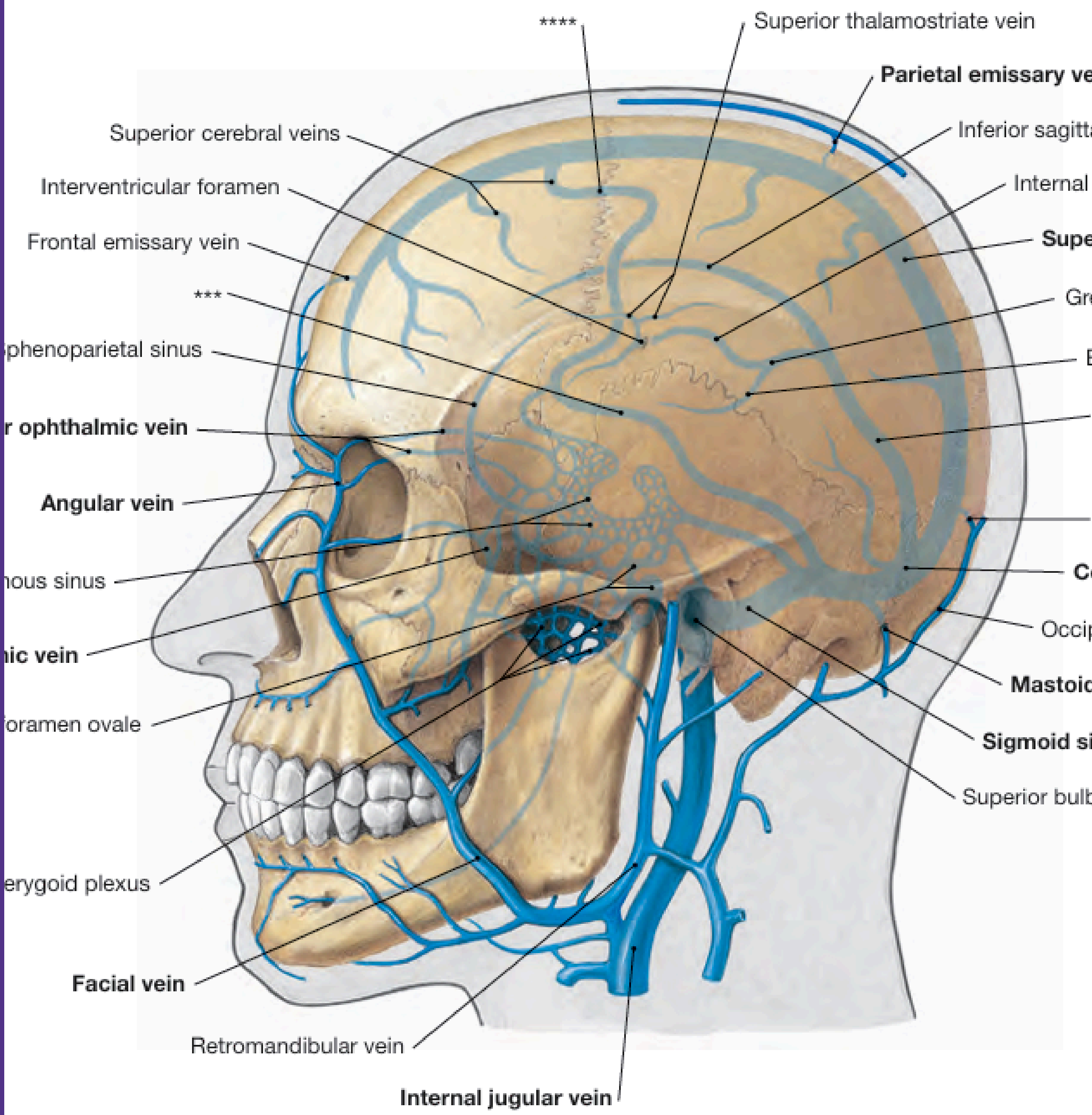
- **Importance of Monitoring Recoil**

  Inattention to full recoil is a common problem during resuscitation that needs to be actively monitored and addressed to ensure effective CPR.

Incomplete chest recoil or 'leaning' results in an **increase in intrathoracic pressure** when it needs to be at its minimum. **This can decrease coronary perfusion pressure by more than a third and cerebral perfusion by over 50%.**

Harris AW, Kudenchuk PJ. Cardiopulmonary resuscitation: the science behind the hands. Heart. 2018;104(13):1056. doi:10.1136/heartjnl-2017-312696

Hombach-Klonisch S, Klonisch TJ, Peeler J, eds. *Sobotta Clinical Atlas of Human Anatomy*. 1. edition. Elsevier; 2019.



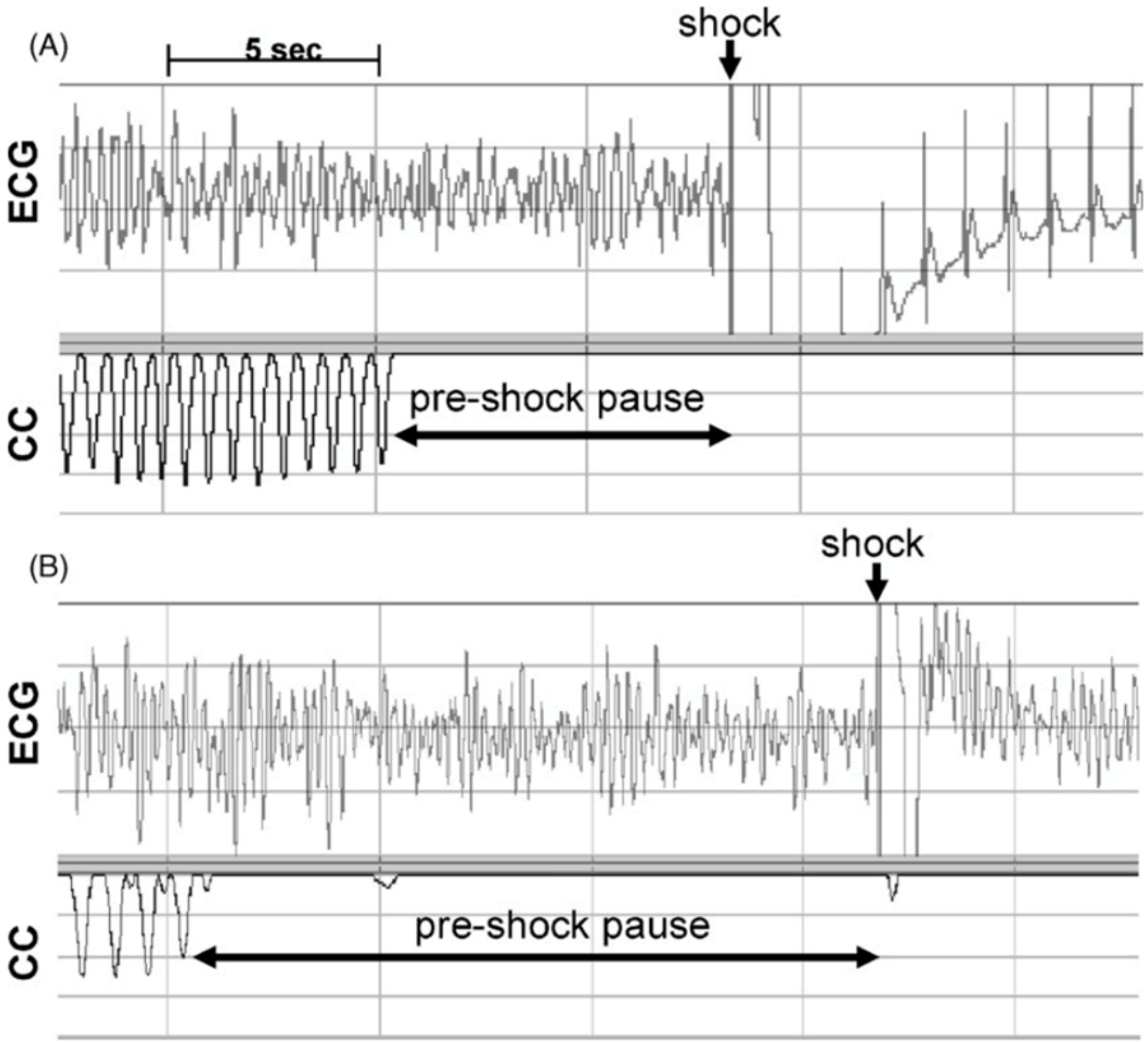


Figure 1 Examples of defibrillation attempts. (A) Successful shock preceded by an 8-s pre-shock pause and deep chest compressions. (B) Unsuccessful shock preceded by a 16 s pre-shock pause and shallower chest compressions. ECG, electrocardiogram; CC, chest compressions.

Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation. 2006;71(2):137-145. doi:10.1016/j.resuscitation.2006.04.008

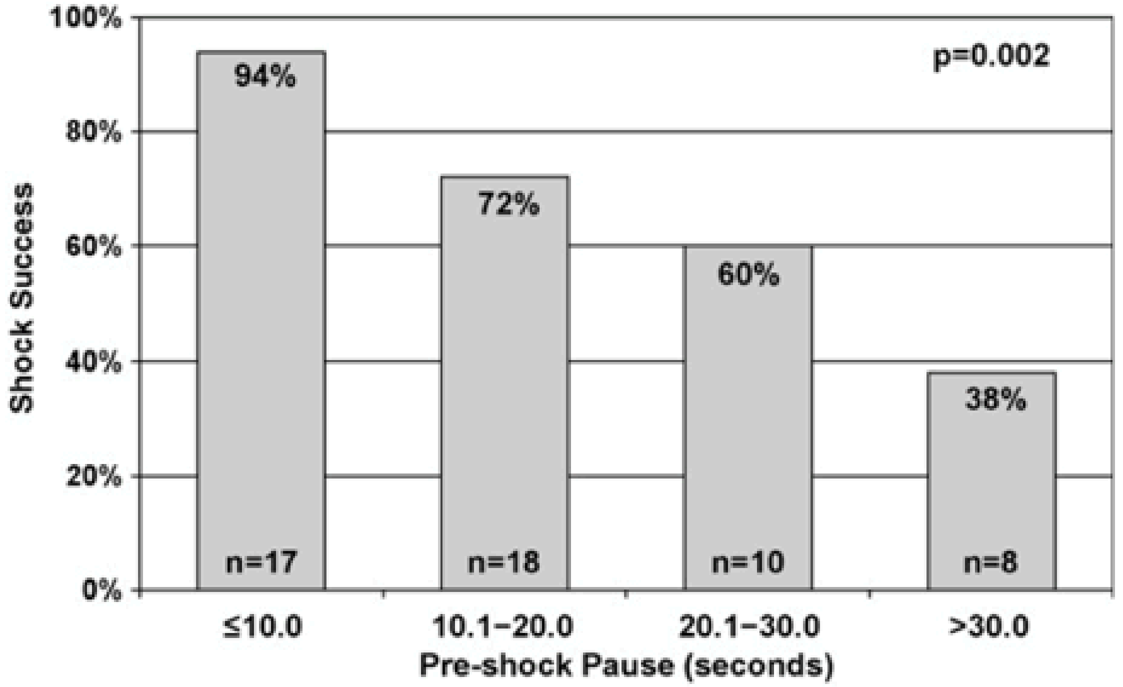


Figure 2 Association between pre-shock pause and shock success. Cases are grouped by pre-shock pause in 10 s intervals. Note that longer pre-shock pauses are significantly associated with a smaller probability of shock success.

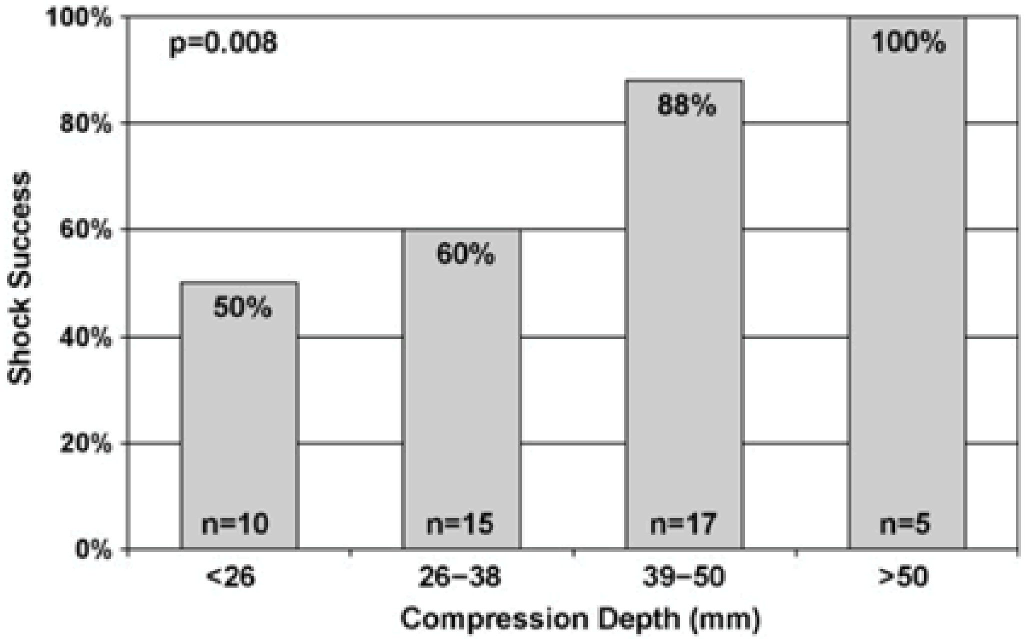


Figure 3 Association between chest compression depth and shock success. Cases are grouped by 30 s average compression depth in approximately 11 mm (0.5 in.) intervals. Chest compression depth of 38—50 mm (1.5—2 in.) represents current CPR guidelines recommendations. Deeper chest compressions are significantly associated with increased probability of shock success.

Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation. 2006;71(2):137-145. doi:10.1016/j.resuscitation.2006.04.008

Closed-Chest Cardiac Massage

Cardiac Arrest and Restoration of Circulation

When cardiac arrest occurs, it is crucial to restore circulation promptly to prevent irreversible damage from anoxia. Two techniques can be used: open-chest cardiac massage and closed-chest cardiac massage.

Closed-Chest Defibrillator

The closed-chest alternating current defibrillator developed in our laboratories has proven to be an effective and reliable means of terminating ventricular fibrillation. The counter-shock must be administered quickly, within 3 minutes of the onset of ventricular fibrillation.

Enhancing Circulation in Arrested Heart

Researchers have explored closed-chest methods to enhance circulation and maintain the tone of the heart and nourish the central nervous system, with the goal of extending the time limitation for defibrillation without opening the chest.

Closed-Chest Cardiac Massage Techniques

Techniques explored include rhythmic pressure on the chest, rocking the patient, and flexing the legs and buttocks against the chest to improve blood flow and blood pressure.

Time Extension for Defibrillation

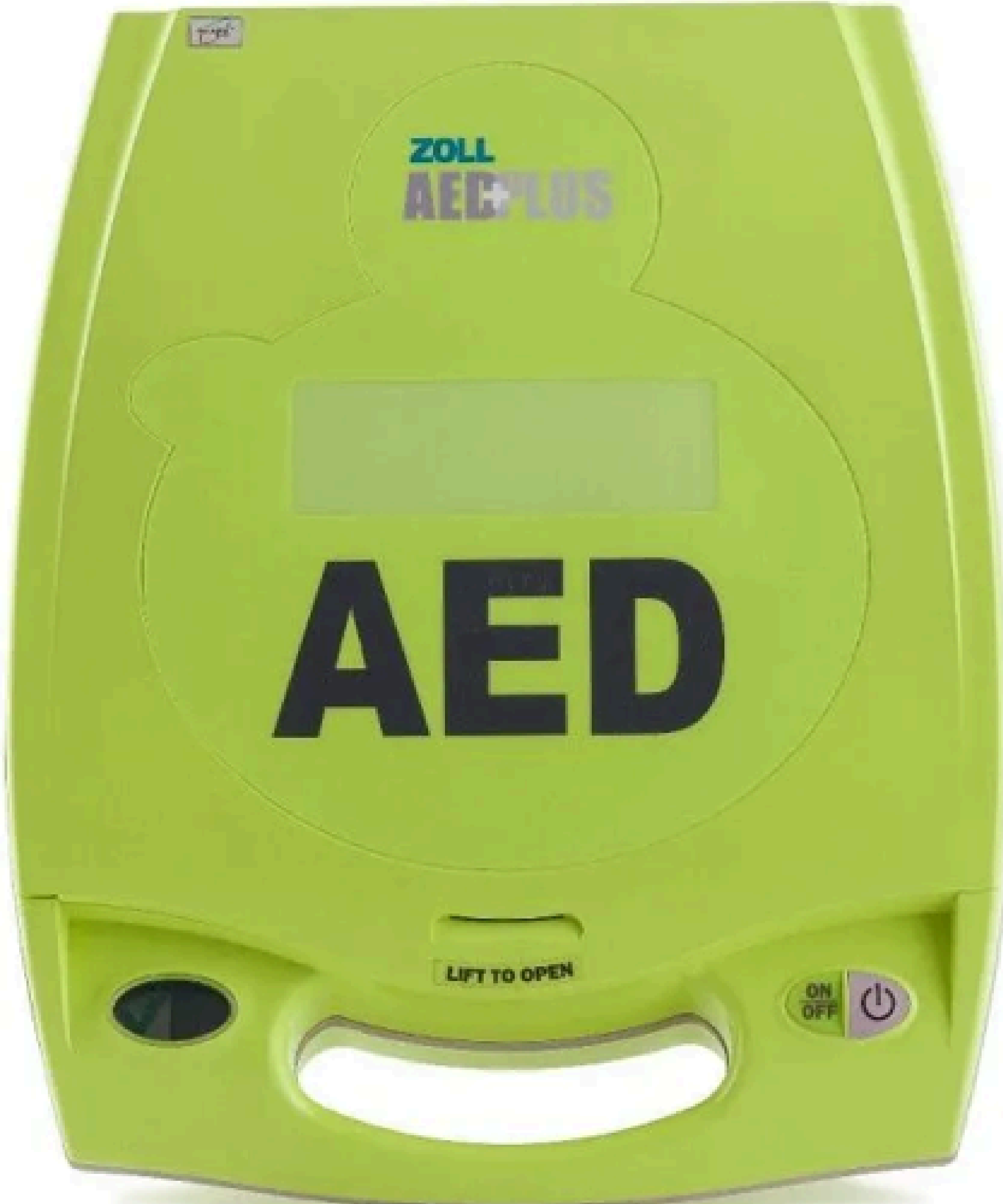
Studies have shown that rhythmic pressure on the thorax can extend the time limitation for defibrillation from 1-1.5 minutes to up to 8 minutes, improving survival rates.


YouTube

Section 10

Defibrillation


defibtech
Lifeline AED
ON
OFF
check pads
do not touch patient
analyzing
SHOCK


ZOLL
AED PLUS
AED
LIFT TO OPEN
ON
OFF

Finding an automated external defibrillator (AED)

Smyth MA, Van Goor S, Hansen CM, et al. European resuscitation council guidelines 2025 adult basic life support. Resuscitation. 2025;215:110771. doi:10.1016/j.resuscitation.2025.110771

AED locations indicated by clear signage

Signage should state that **AEDs can be used by anyone and no training is needed**

AED locations should be identified using electronic mapping systems

Local emergency service should direct callers to nearest available AED

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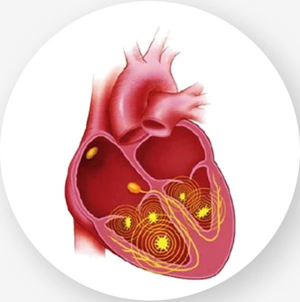
Ventricular Fibrillation and Defibrillation

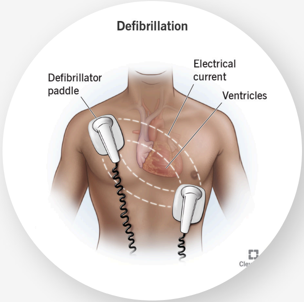


Causes of Ventricular Fibrillation

Increased automaticity of multiple ectopic ventricular pacemakers and multiple reentry circuits in the ventricles, leading to chaotic depolarization and contraction of the ventricles.

Consequences of Ventricular Fibrillation

Ventricular fibrillation results in a lack of organised ventricular contraction and cardiac output, which is incompatible with life. Spontaneous termination of ventricular fibrillation does not occur.



Immediate Treatment of Ventricular Fibrillation

The only effective treatment for ventricular fibrillation is immediate defibrillation, which delivers an electrical stimulus to synchronise the depolarisation of all ventricular myocytes.

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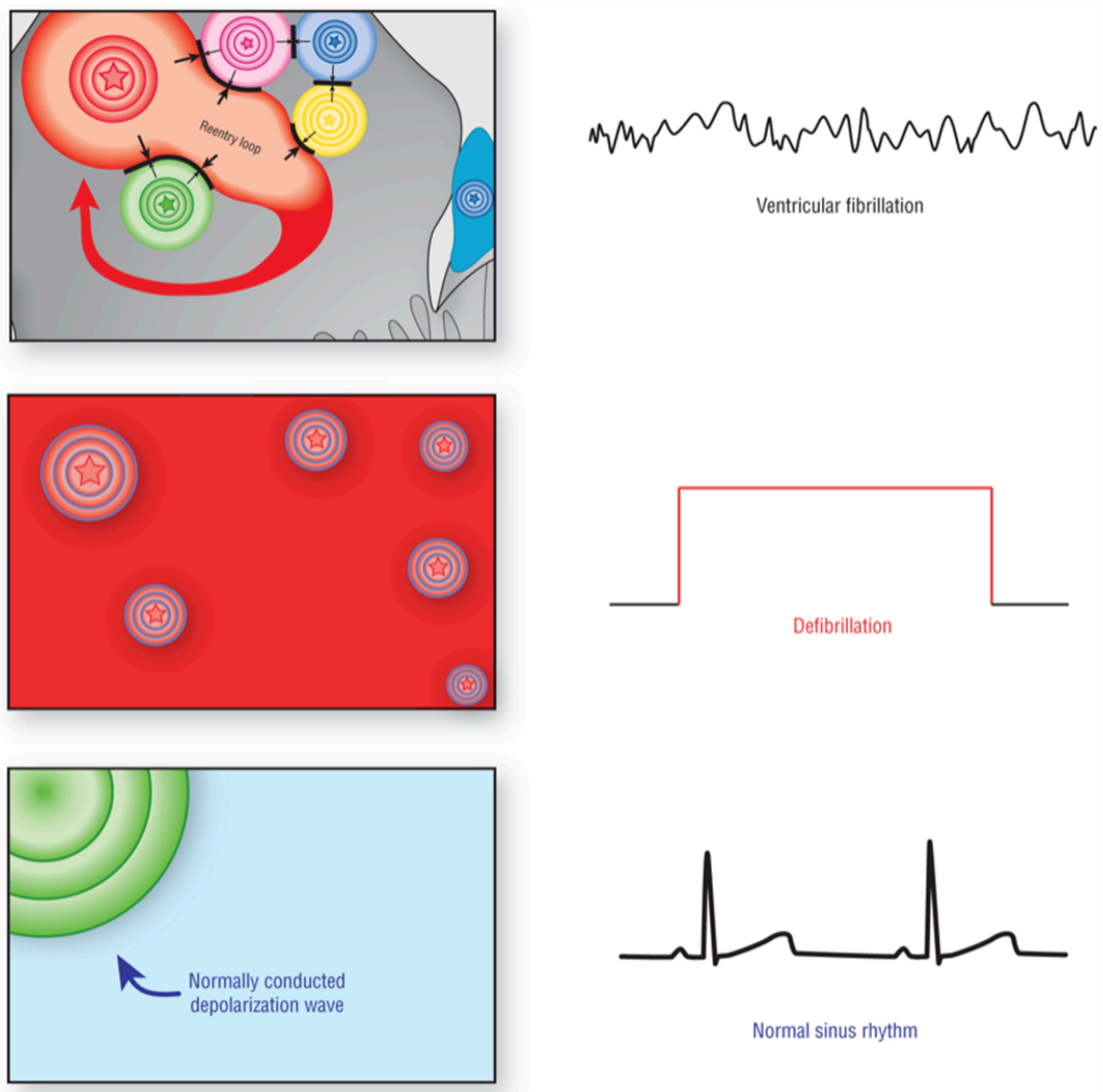

The electrical stimulus provided by emergent defibrillation causes an immediate and synchronized depolarization of all ventricular myocytes

Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation. Second edition. Jones & Bartlett Learning; 2020.

Transthoracic Impedance

- **Energy Dissipation**

  In animal studies, only 4% of the energy supplied reaches the heart.

- **Average Adult TTI**

  The average adult human TTI is around 70-80 Ω, determined by factors like energy level, electrode size, interelectrode distance, skin-electrode interface, electrode pressure, ventilation phase, myocardial tissue, and blood conductive properties.

- **High TTI Impact**

  When TTI is too high, a low-energy shock will not generate sufficient current to achieve defibrillation.

- **Reducing TTI**

  To reduce TTI, the defibrillator operator should use conductive materials like gel pads or electrode paste with paddles or self-adhesive pads.

Delgado H, Toquero J, Mitroi C, et al. Principles of External Defibrillators. In: Cardiac Defibrillation. IntechOpen; 2013. doi:10.5772/52512

Inadvertent Electric Shocks During CPR-D

Defibrillator Misuse

Seven reports of intentional or accidental defibrillator misuse, with one case resulting in a life-threatening arrhythmia and unsuccessful resuscitation efforts.

Faulty Equipment

Three incidents of electric shocks due to broken or failing equipment during resuscitation procedures.

Training/Maintenance Procedures

Four cases of electric shocks to personnel during training or device maintenance procedures, with one emergency medical technician requiring intubation, medication, and electrical therapy but eventually surviving without complications.

Regular Resuscitations

Fifteen accounts of accidental shocks during resuscitation efforts not related to device failure, with one unexplained electric shock to a nurse during resuscitation.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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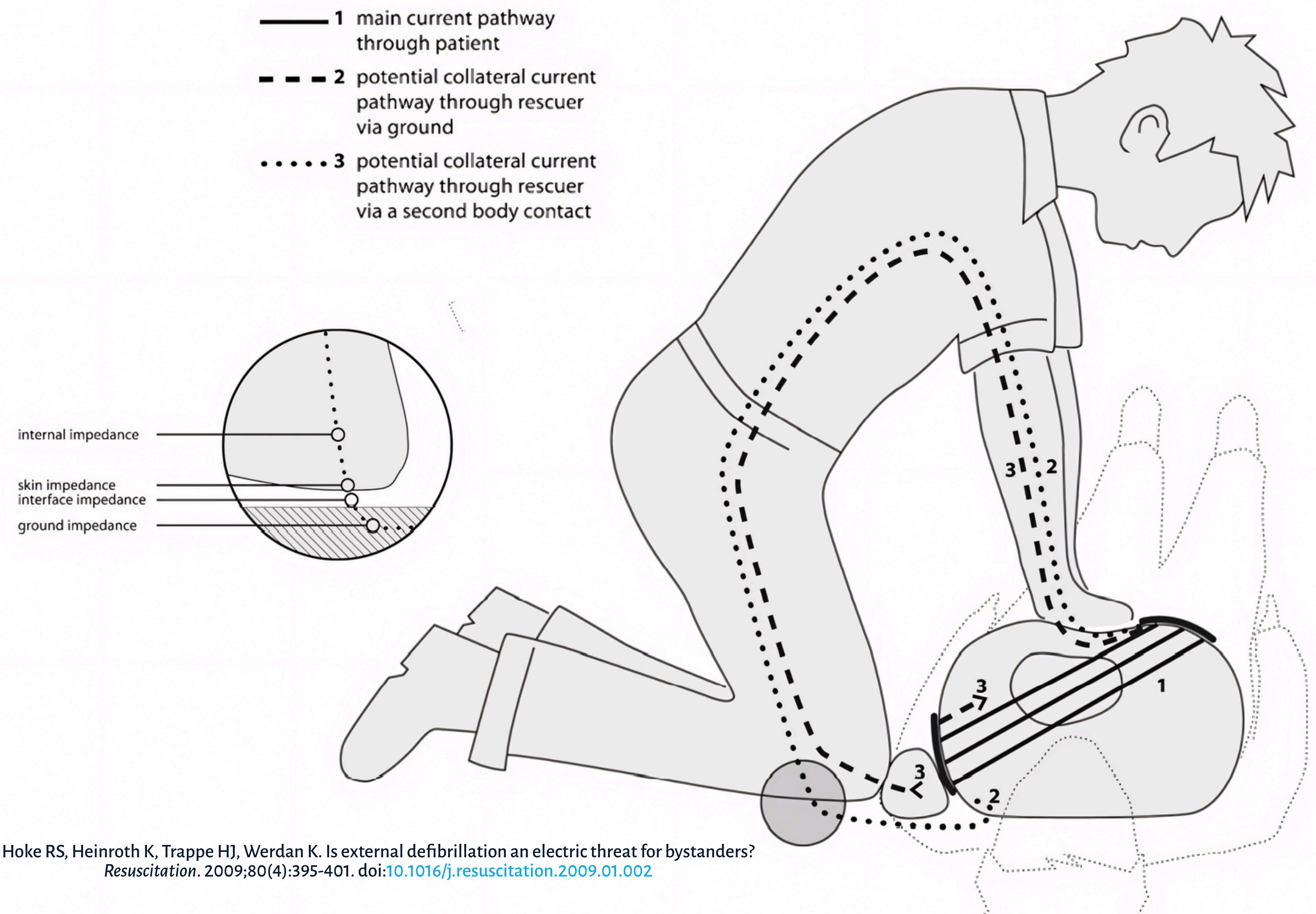

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

Collateral Circuits through Rescuers/Bystanders

- ## Collateral Circuit Formation

  Once a collateral circuit through a rescuer is established, the total current between the defibrillator electrodes divides into one portion through the patient's chest and another through the rescuer.

- ## Impedance Factors

  The total impedance of the collateral circuit comprises the impedance of interfaces (shoes, gloves, skin-to-bed interface, etc.), skin impedance, and the internal impedance of the rescuer's body.

- ## Skin Impedance Variability

  Skin impedance decreases with contact area, voltage, hydration, temperature, decreasing thickness, and fluctuates with contact time. Reported values range from less than 100Ω for thin, wet skin/large contact area, to above 1MΩ for callous, dry skin/small contact area.

- ## Current Division

  Given a certain discharge voltage of the defibrillator, the lower the total impedance of the collateral circuit, the higher the current through the rescuer.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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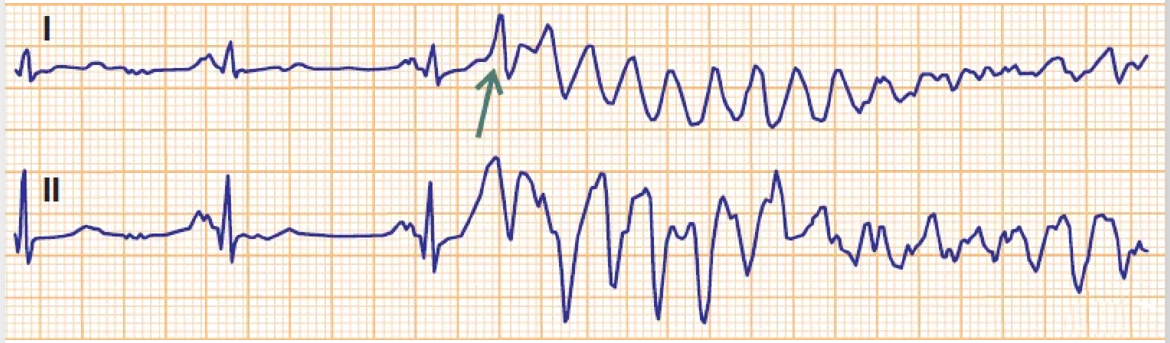
Skin Impedance Breakdown and Electrocution Risks

- **Skin Impedance Breakdown**
  Sudden and dramatic decrease in impedance observed above a certain voltage threshold, leading to larger current flow.
- **Voltage Threshold**
  Skin impedance breakdown has been described to occur above 200V for currents longer than a defibrillator impulse.
- **Body Impedance**
  Internal impedance of the human body is approximately 500Ω for high voltage capacitive discharges, and is similar for hand-to-hand and hand-to-foot pathways.
- **Rescuer Impedance**
  Total impedance of collateral circuits through rescuers is highly variable, with a mean of 22.7 kΩ and a range of 1.09 to 100 kΩ observed in a study simulating inadvertent contact during defibrillation.
- **Resuscitation Scenario**
  A study simulated inadvertent contact between a rescuer and a patient during cardioversion, with the rescuer's gloved hands pressed on the patient's sternum and their thigh electrically connected to the patient's shoulder.

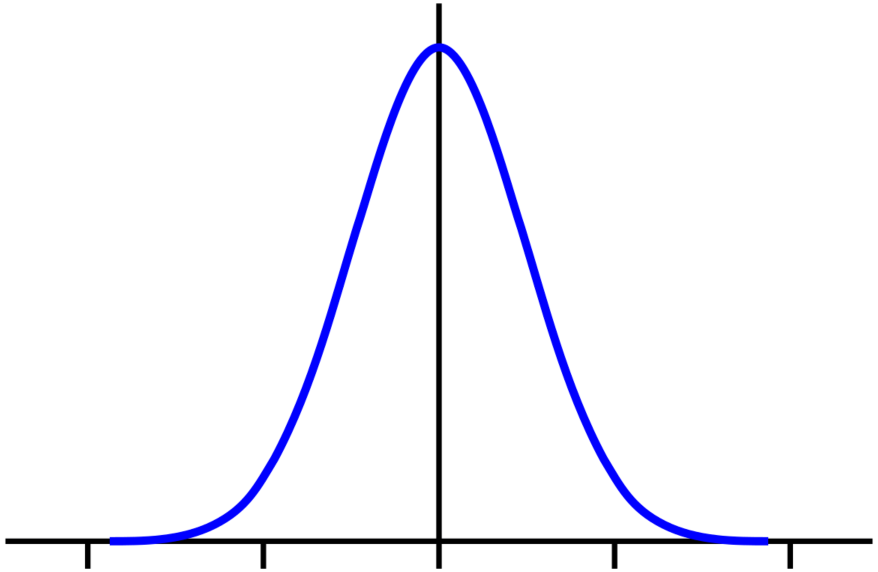
Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

Ventricular Fibrillation and Vulnerability



Ventricular Fibrillation Induction

VF can be induced by an electric stimulus during phases of myocardial non-uniform refractoriness, known as the vulnerable period, which extends from about 60 to 90% of the QT-phase on the surface ECG in the normal heart.



Excitation Susceptibility

The excitation susceptibility has a bell-shaped distribution, and VF has also been described as a result of appropriately R-wave triggered external cardioversion shocks and inadvertent external defibrillation of normal sinus rhythm.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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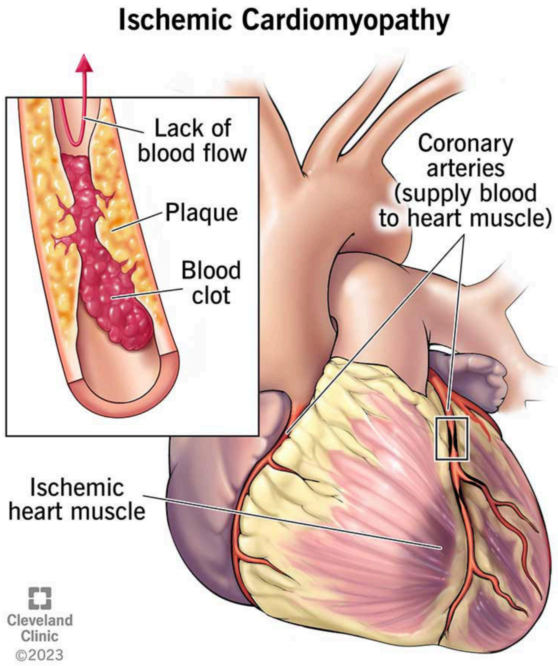
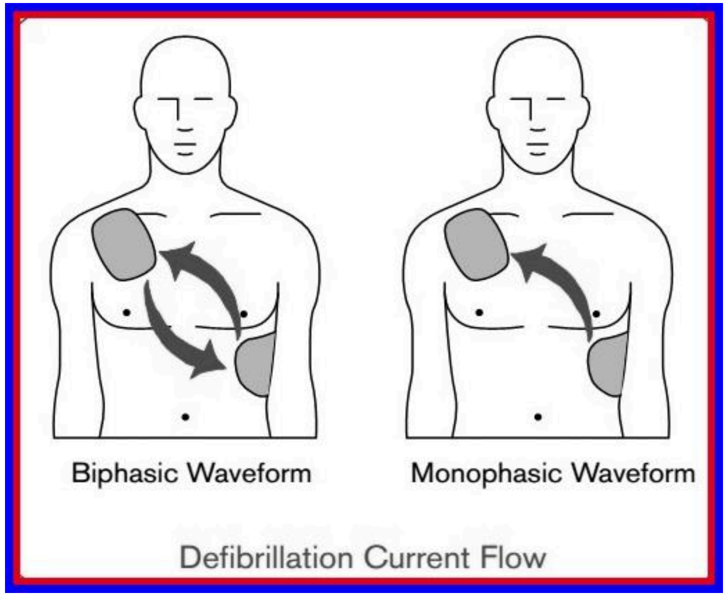
Ventricular Fibrillation and Vulnerability



Factors Increasing Vulnerability

Vulnerability of the heart is increased after premature ventricular beats, during hypothermia, ischaemia, acidosis, adrenergic stimulation, or rapid pacing and ventricular tachycardia.



Shock Waveform Efficacy

Biphasic impulses are generally less effective at inducing VF than monophasic shocks, and the least shock energy is required if the negative electrode is placed over the apex of the heart.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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Fibrillation Threshold for External Currents

Animal Experiments on 50-60 Hz AC

Experiments in animals have shown that fibrillation threshold increases with body weight and is lower for currents applied in ECG leads II and III compared to lead I. The more 60 Hz AC cycles applied, the higher the likelihood of ventricular fibrillation (VF) occurrence, with the threshold decreasing up to about 120 cycles.

Fibrillation Thresholds from Animal Studies

Based on extrapolation from animal experiments, the International Electrotechnical Commission (IEC) has set the fibrillation threshold for 50/60 Hz AC of 4-20 ms duration (one complete AC cycle or less) traveling from hand to feet at 500 mA RMS current. Underwriters Laboratory has set the threshold at 300 mA RMS current.

Fibrillation Thresholds in Humans

Experiments to determine fibrillation current for short-duration external shocks have never been undertaken in humans. However, insights can be derived from external cardiac pacing studies, where ventricular capture thresholds are typically around 75 mA, with the current that precipitates ventricular tachycardia or VF being at least five times greater.

Worst-Case Scenario for Defibrillator Shocks

In a worst-case scenario with a defibrillator shock directly applied over the apex of a rescuer's heart through contact impedance minimizing electrodes at the vulnerable period, currents up to 200 mA should be regarded as safe.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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Safety Considerations for Rescuers during Defibrillation

Defibrillator Waveforms

Modern external defibrillators use various waveforms, including damped sinusoidal, exponential, truncated exponential, biphasic truncated exponential, biphasic rectangular, and biphasic chopped truncated exponential. The duration of the complete impulse ranges between 8 and 30 ms.

Electrical Characteristics

During defibrillator discharge, 500–2000 V are impressed between the electrodes, resulting in peak transthoracic currents up to 70 A, depending on the patient's thoracic impedance. The potential gradients and resulting currents that rescuers may be exposed to while contacting the patient or adjacent conductive material are largely unknown.

Experimental Findings

Lyster et al. reported maximum potential gradients of 14 V and 30 V between two metal probes on the ground, 15 cm from a defibrillated turkey on a wet surface. Lloyd et al. reported currents between 18.9 and 907 A through a rescuer simulating chest compressions and potential gradients from 0.28 to 14.1 V behind an isolation with a polyethylene glove.

Guidelines and Recommendations

The AHA recommends recovering a patient when lying in water and wiping the chest dry before attempting defibrillation. The ERC has not made a specific statement. With the use of post-cardiac arrest mild therapeutic hypothermia and peri-arrest cooling, the need for safety data on electrical therapy under wet conditions has gained new attention.

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

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Electric shocks to healthcare professionals during resuscitation due to human error. *Abbreviations*: J, joules; EMT, emergency medical technician; N/R, not reported.

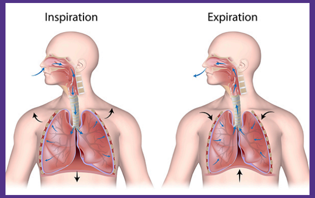
Reference	Circumstances	Consequences, outcome
Gibbs et al.[9]	Shock to paramedic in contact with side rail of stretcher during defibrillation	"Tingling in right arm for 30 min"
	Shock to defibrillating paramedic, hand in contact with conduction gel during defibrillation	"Mild soreness to right arm"
	Shock to paramedic checking the femoral pulse of the patient during defibrillation	"Knocked paramedic away from patient"
	Shock to paramedic, leg in contact with the patient during defibrillation	"Mild shock to leg"
	Shock to EMT holding a bag-valve mask during defibrillation	"Mild shock to finger tips"
	Shock to EMT, thumb in contact with chest of patient during defibrillation	"Shock to hand, lethargy for several minutes"
	Shock to EMT, leg in contact with stretcher during defibrillation	"Shock to leg"
	Shock to paramedic during defibrillation	N/R
	Shock to nurse during defibrillation	"Tingling sensation"
	Shock to paramedic during defibrillation	Arm discomfort
	Arching between electrode paddles and patient chest during defibrillation	"Ringing in ears"
	Arching between electrode paddles and patient chest during defibrillation	"Burn to hand"
	Shock to three EMTs due to accidental discharge during charging	"Mild shock"
	Shock to nurse due to accidental discharge during assessment of ECG	"Mild shock"
Dickinson et al.[74]	200 J through self-adhesive pads, shock to rescuer performing chest compressions due to inadvertence of device operator (no gloves worn – personal communication, Dr Jasmeet Soar, May 2008)	Electric charge felt in arms, no immediate or long-term dysfunction

Hoke RS, Heinroth K, Trappe HJ, Werdan K. Is external defibrillation an electric threat for bystanders? *Resuscitation*. 2009;80(4):395-401. doi:10.1016/j.resuscitation.2009.01.002

Section 11

Assessing the return of spontaneous circulation

Indicators of Return of Spontaneous Circulation (ROSC)



Breathing

The patient may begin to breathe spontaneously or show signs of respiratory effort, indicating the return of respiratory function.

Coughing

Reflexive coughing can indicate the return of neurological function and airway responsiveness, a positive sign of improved condition.

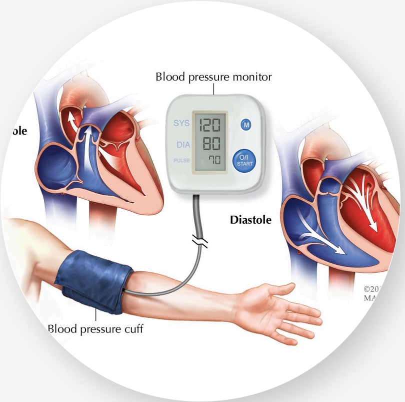
Movement

Any purposeful movement or reflexive responses can signify improved neurological status and returning brain function.

Indicators of Return of Spontaneous Circulation (ROSC)

Palpable Pulse

A detectable pulse indicates that the heart is effectively pumping blood, a crucial sign of circulation resumption.



Measurable Blood Pressure

Restoration of blood pressure is a crucial sign that circulation has resumed, showing the heart is effectively delivering blood throughout the body.


118
SpO2 %
95
SpO2
CO2 mmHg
11
18RR
CO2
NIBP mmHg
121
79
93
CPR
2
ANALYZE
3
LEAD
SIZE
SYNC
NIBP
ALARMS
OPTIONS
EVENT
CHARGE
PACER
RATE
CURRENT
PAUSE
SPEED DIAL
12 LEAD
TRANSMIT
YouTube

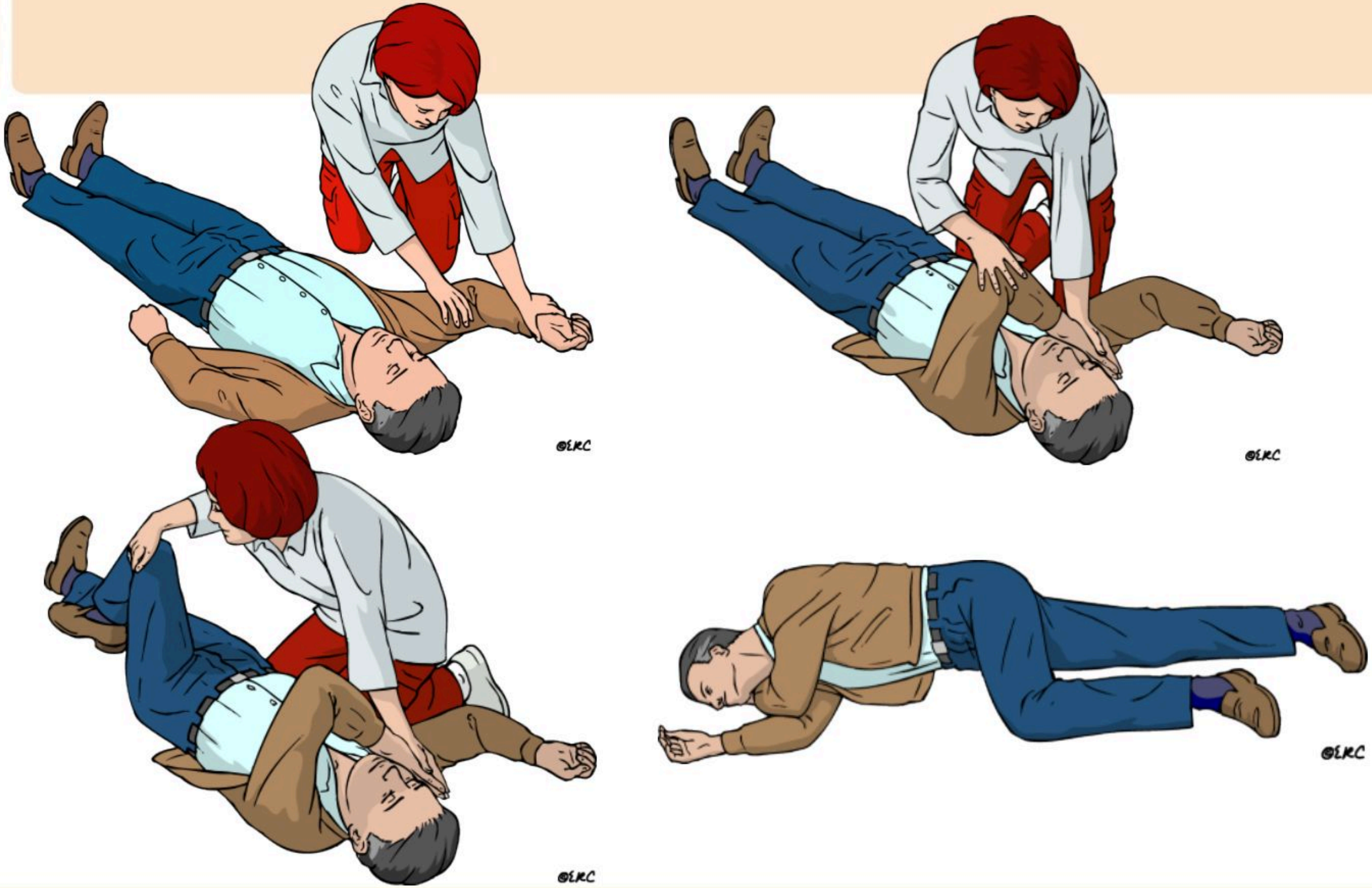
Section 12

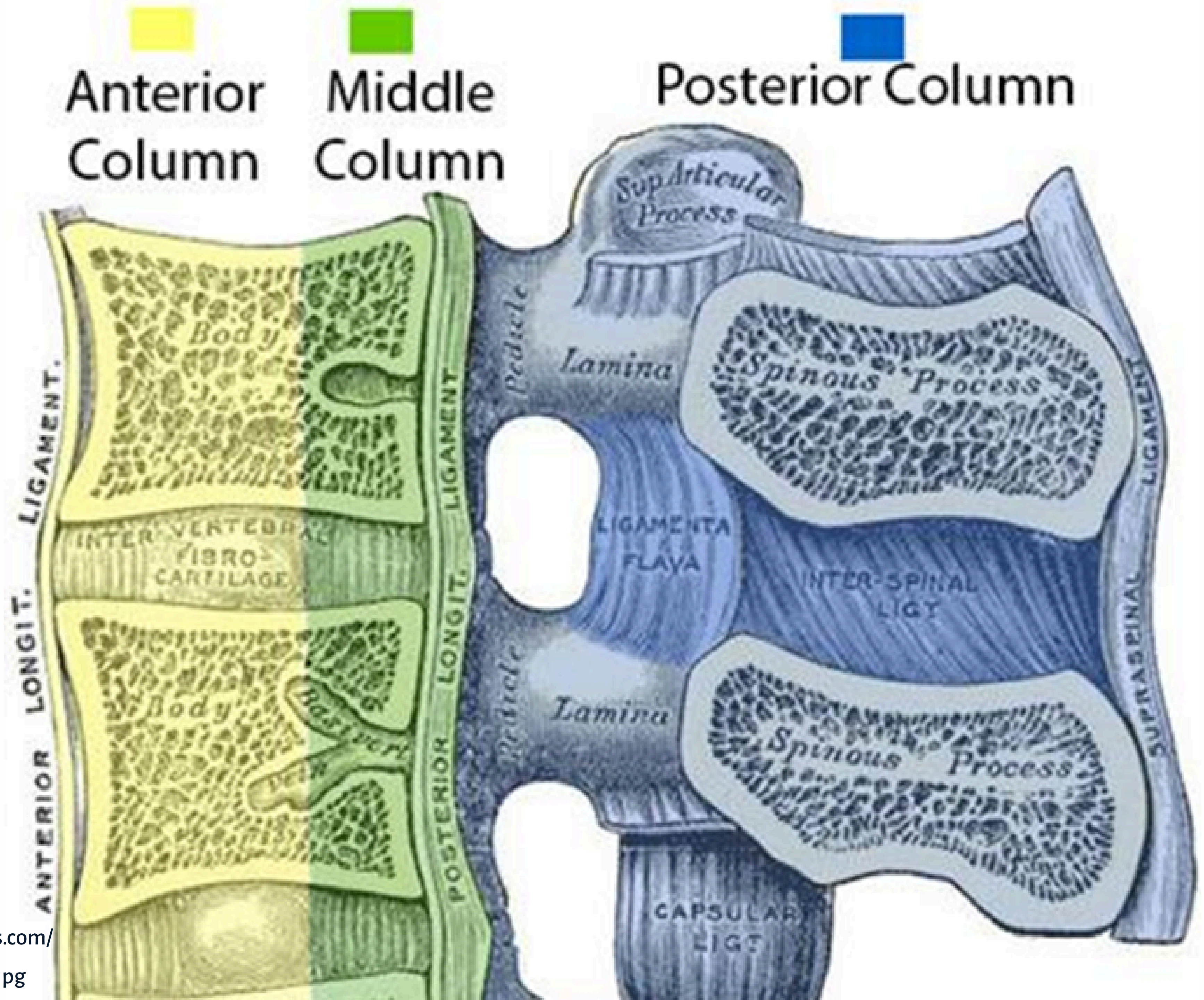
Recovery position

EUROPEAN RESUSCITATION COUNCIL

If victim starts to breathe normally place in recovery position

EUROPEAN
RESUSCITATION
COUNCIL





https://upload.orthobullets.com/topic/2022/images/denis.jpg

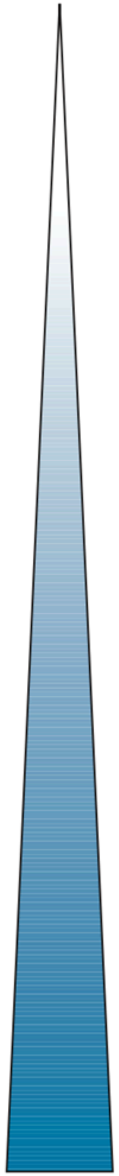
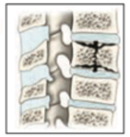
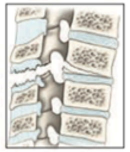
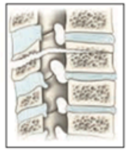
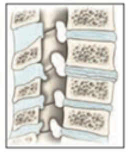
Fracture type (AO/OTA)	Stability	Risk of SCI	Management
A-type: Axial compression		0%	
 A1: Impaction/ compression	Stable		Nonoperative
 A2: Split	Stable		Nonoperative
 A3: Burst	Stable/unstable*		Nonoperative/operative*
B-type: Flexion/distraction or hyperextension			
 B1: Flexion/ distraction	Unstable		Operative
 B2: Chance fracture	Unstable		Operative
 B3: Hyperextension	Unstable		Operative
C-type: A or B type with rotation			
 C1: Rotational wedge	Unstable		Operative
C2: Rotational flexion/ extension	Unstable		Operative
C3: Rotational shear (Holdsworth slice fracture)	Unstable	100%	Operative

FIGURE 26-3 Spinal fractures, traumatic dislocations, and fracture–dislocations are best classified by the comprehensive AO Foundation/ Orthopaedic Trauma Association (AO/OTA) classification system, which is based on the alphanumeric classification published by Magerl et al[13] in 1994.

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw-Hill Education; 2020.

Table 11-2: Clues to Spinal-Cord Injury Revealed During Patient Assessment

Mechanism of Injury
Blunt trauma above the clavicle
Diving accident
Motor-vehicle or bicycle crash
Fall
Stabbing or impalement anywhere near the spinal column
Shooting or blast injury to the torso
Any violent injury with forces that could act on the spinal column or cord
Patient Complaints
Neck or back pain
Numbness or tingling
Loss of movement or weakness
Signs Revealed During Assessment
Pain on movement of back or spinal column
Obvious deformity of back or spinal column
Guarding against movement of back
Loss of sensation
Weak or flaccid muscles
Loss of bladder or bowel control
Erection of the penis (priapism)
Neurogenic shock

ITLS, Alson RL. International Trauma Life Support for Emergency Care Providers. Pearson Education; 2015.

Comparing Spinal Distortion in Recovery P

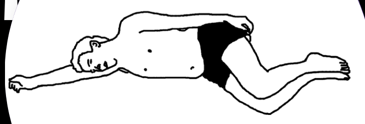


Fig. 2. The modified HAINES position.

- **Lateral Recovery Position**
  Widely used for unconscious patients, but concerns about spinal cord injury in trauma cases
- **Modified HAINES Position**
  A new recovery position described to reduce distortion of the spine compared to lateral recovery
- **Hypothesis**
  The modified HAINES position results in less distortion of the spine than the lateral recovery position
- **Findings**
  The modified HAINES position showed 13.08 degrees less lateral flexion and 12.68 degrees less extension of the cervical spine, while the thoraco-lumbar spine was similar in both positions
- **Subjective Experience**
  19 out of 28 subjects found the modified HAINES position more comfortable (not statistically significant)
- **Conclusion**
  The modified HAINES position is preferable to the lateral recovery position in the management of patients when trauma may have occurred, as it results in a more neutral position of the spine

Blake WED, Stillman BC, Eizenberg N, Briggs C, McMeeken JM. The position of the spine in the recovery position—an experimental comparison between the lateral recovery position and the modified HAINES position. Resuscitation. 2002;53(3):289-297. doi:10.1016/S0300-9572(02)00037-0

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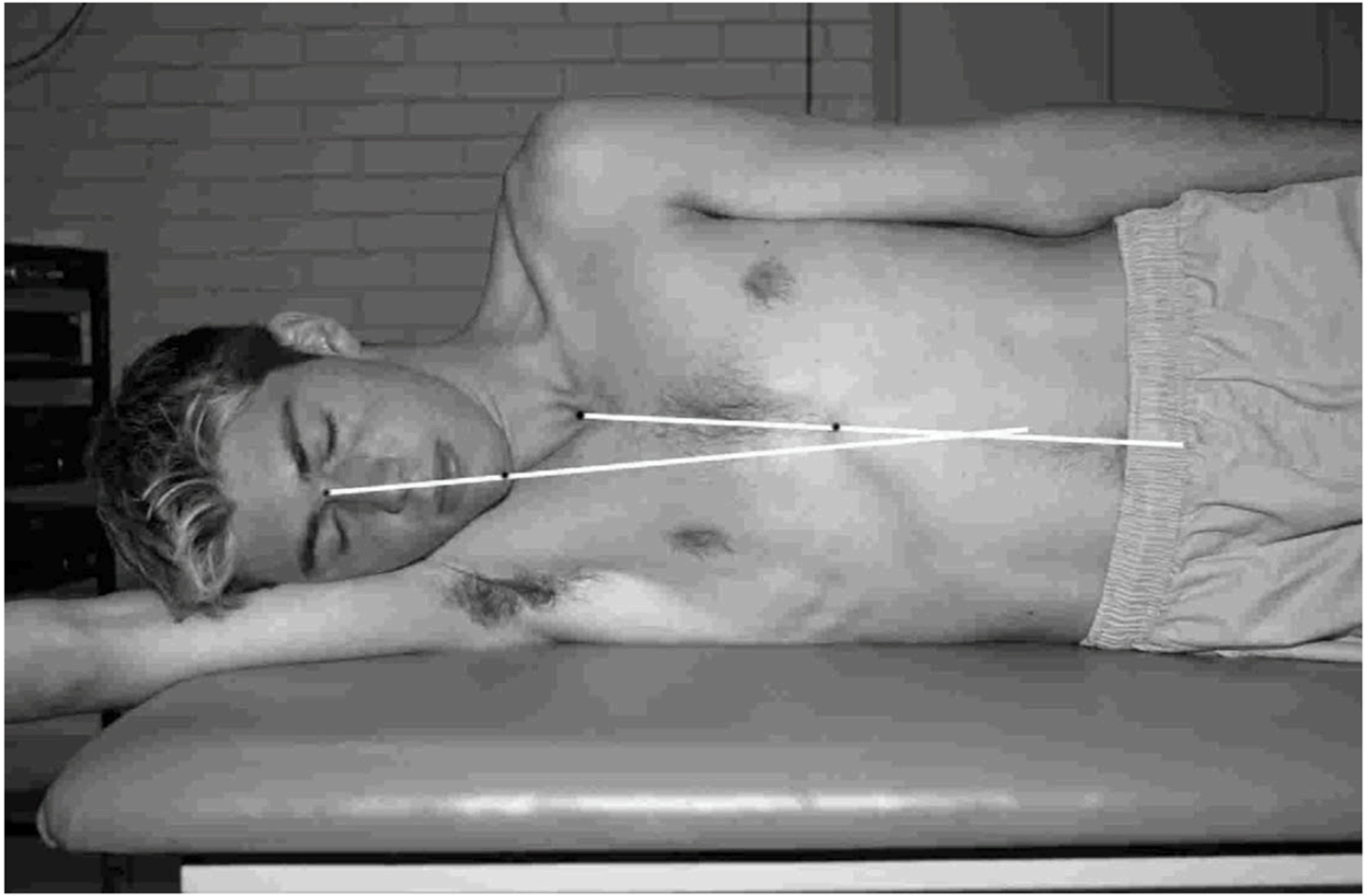

Fig. 3. Cervical lateral flexion was measured by comparing the midline of the face to the midline of the sternum. Here it is 6°.

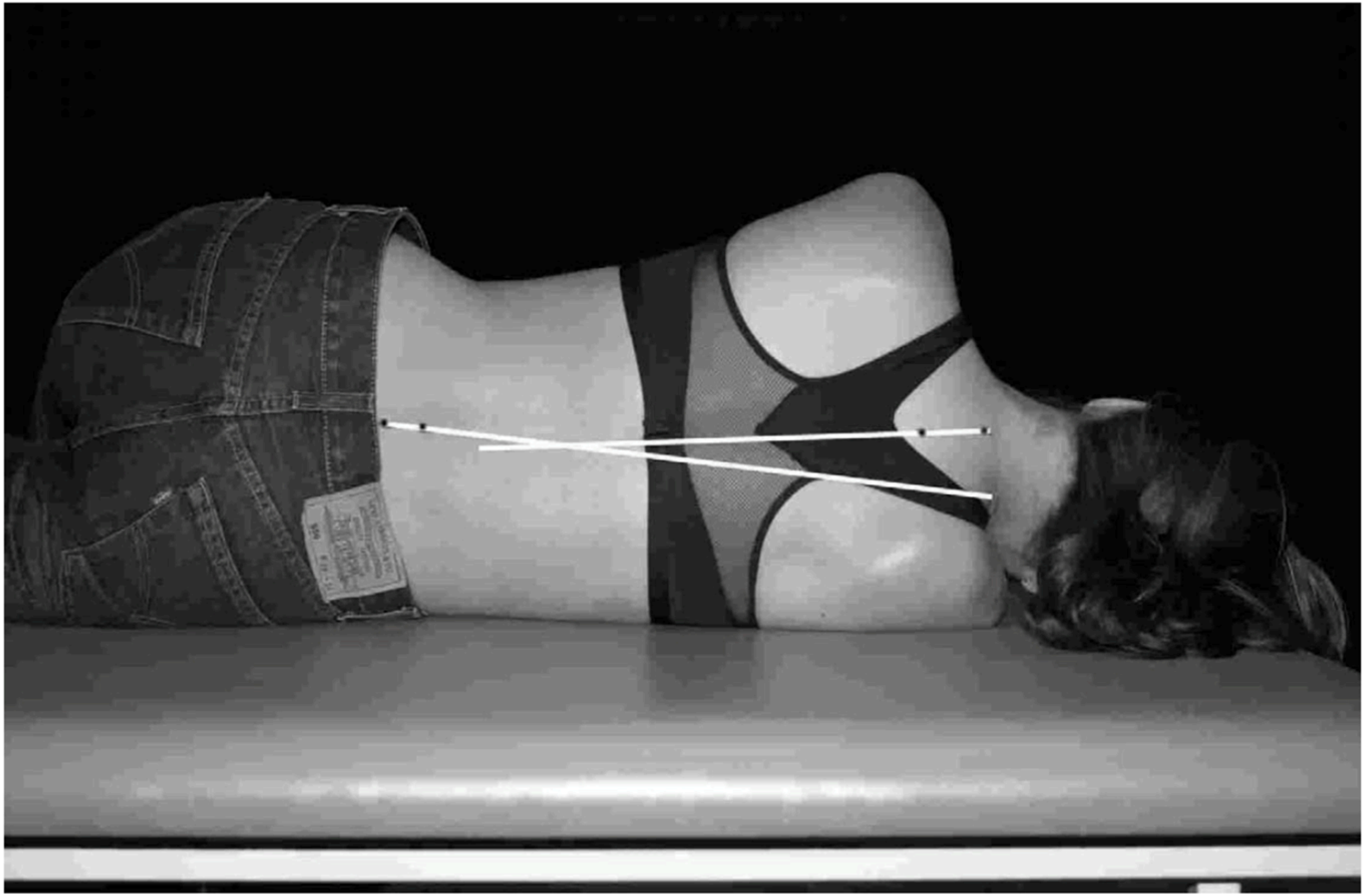

Fig. 4. Thoraco-lumbar lateral flexion was measured by marking the spinous processes of C7, T2, L2 and L4. Here it is 6°.

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Fig. 5. Flexion of the cervical and thoraco-lumbar spine was assessed from above. Here the angles are −25 and 22° respectively. See text for details.

Table 2
Summary of data

	Lateral recovery position	Modified HAINES position	Difference	99% CI of difference	*P* value
Cervical region					
Flexion	−36.89	−23.91	12.98	7.47−18.49	≪ 0.001
Rotation	1.85	2.05	0.20	−4.28−4.68	0.91 NS
Lateral flexion	22.35	9.74	12.61	9.37−15.85	≪ 0.001
Thoraco-lumbar region					
Flexion	23.90	23.44	0.46	−2.17−3.09	0.64 NS
Rotation	−4.64	5.22	9.86	6.41−13.31	≪ 0.001
Lateral flexion	−7.01	−10.72	3.71	0.28−7.14	< 0.01

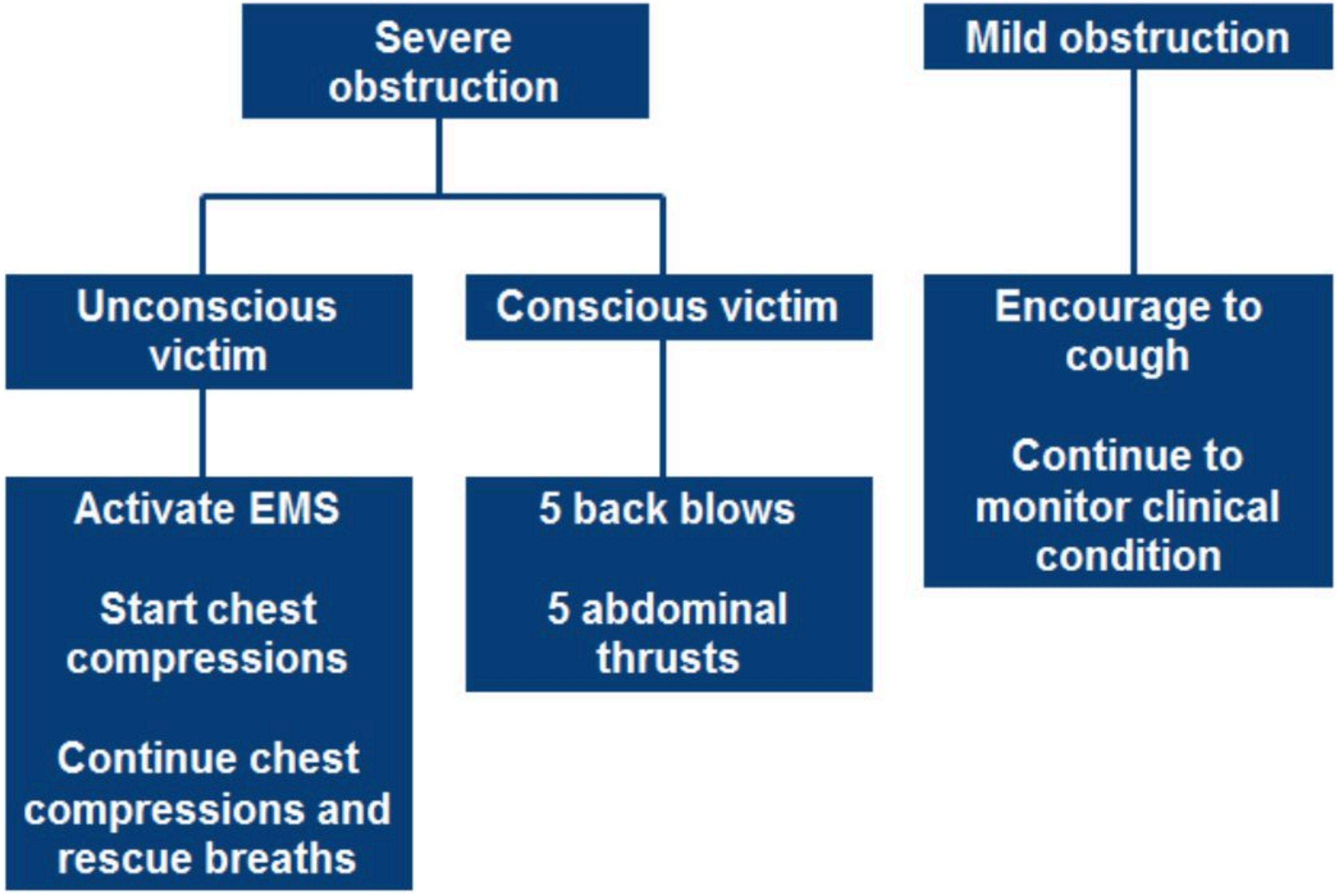
Expressed as mean of 38 observations, all angles in degrees, see text for sign convention.

Blake WED, Stillman BC, Eizenberg N, Briggs C, McMeeken JM. The position of the spine in the recovery position—an experimental comparison between the lateral recovery position and the modified HAINES position. *Resuscitation*. 2002;53(3):289-297. doi:10.1016/S0300-9572(02)00037-0

Section 13

Foreign Object Airway obstruction

CHOKING: ALGORITHM



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CHOKING

Back Blows

Abdominal thrusts: position of first hand

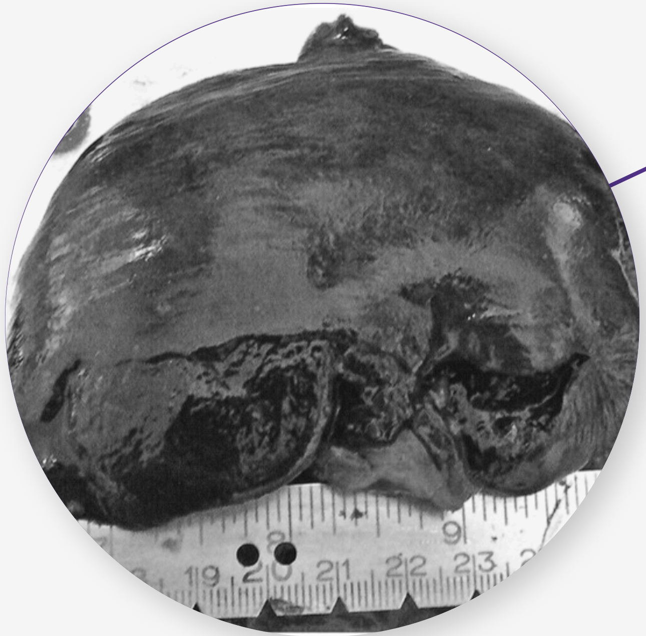
Abdominal thrusts: position of second hand

Rodriguez JAO, Ladd M, Brandis D. Abdominal thrust maneuver. In: *StatPearls [Internet]*. StatPearls Publishing; 2023. Accessed January 6, 2025. https://www.ncbi.nlm.nih.gov/sites/books/NBK531467/ Cecchetto G, Viel G, Cecchetto A, Kusstatscher S, Montisci M. Fatal splenic rupture following heimlich maneuver: case report and literature review. *Am J Forensic Med Pathol*. 2011;32(2):169. doi:10.1097/PAF.0b013e318219c878

Complications of the Heimlich Maneuver

- **Rib Fractures**

  The forceful displacement of the diaphragm and sudden increase in intrathoracic pressure can lead to rib fractures during the Heimlich maneuver.



- **Rare Traumatic Injuries**
  - Other rare traumatic injuries associated with the Heimlich maneuver include
  - splenic rupture,
  - pneumomediastinum,
  - aortic valve cusp rupture,
  - aortic dissection,
  - diaphragmatic herniation,
  - esophageal and jejunal perforation,
  - hepatic rupture,
  - cholesterol embolization,
  - and mesenteric laceration.

Rodriguez JAO, Ladd M, Brandis D. Abdominal thrust maneuver. In: *StatPearls [Internet]*. StatPearls Publishing; 2023. Accessed January 6, 2025. https://www.ncbi.nlm.nih.gov/sites/books/NBK531467/

Abdominal Thrust Maneuver Considerations

1

Contraindications

The abdominal thrust maneuver is not recommended for infants or unconscious patients.

2

Pregnant Subjects

Pregnant subjects should receive management with **sternal compressions**, instead of abdominal thrusts.

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Section 14

BLSD in the pregnant patient

OBSTETRIC LIFE SUPPORT

- Known pregnancy
- Fundus ≥ umbilicus
- In-hospital

Abbreviations

CPR - cardiopulmonary resuscitation
ECPR - extracorporeal cardiopulmonary resuscitation
IO - intraosseous
IV - intravenous
LUD - left uterine displacement
OBLS - Obstetric Life Support
PEA - pulseless electrical activity
pVT - ventricular tachycardia
RCD - resuscitative cesarean delivery
ROSC - return of spontaneous circulation
TTM - targeted temperature management
VF - ventricular fibrillation

B	Bleeding
A	Anesthesia
A	Amniotic fluid embolism
C	Cardiovascular/cardiomyopathy
C	Clot/cerebrovascular
T	Trauma
O	Overdose (magnesium sulfate/opioids/other)
L	Lung injury/Acute respiratory distress syndrome
I	Ions (glucose/K+)
F	Fever (sepsis)
E	Emergency hypertension/eclampsia

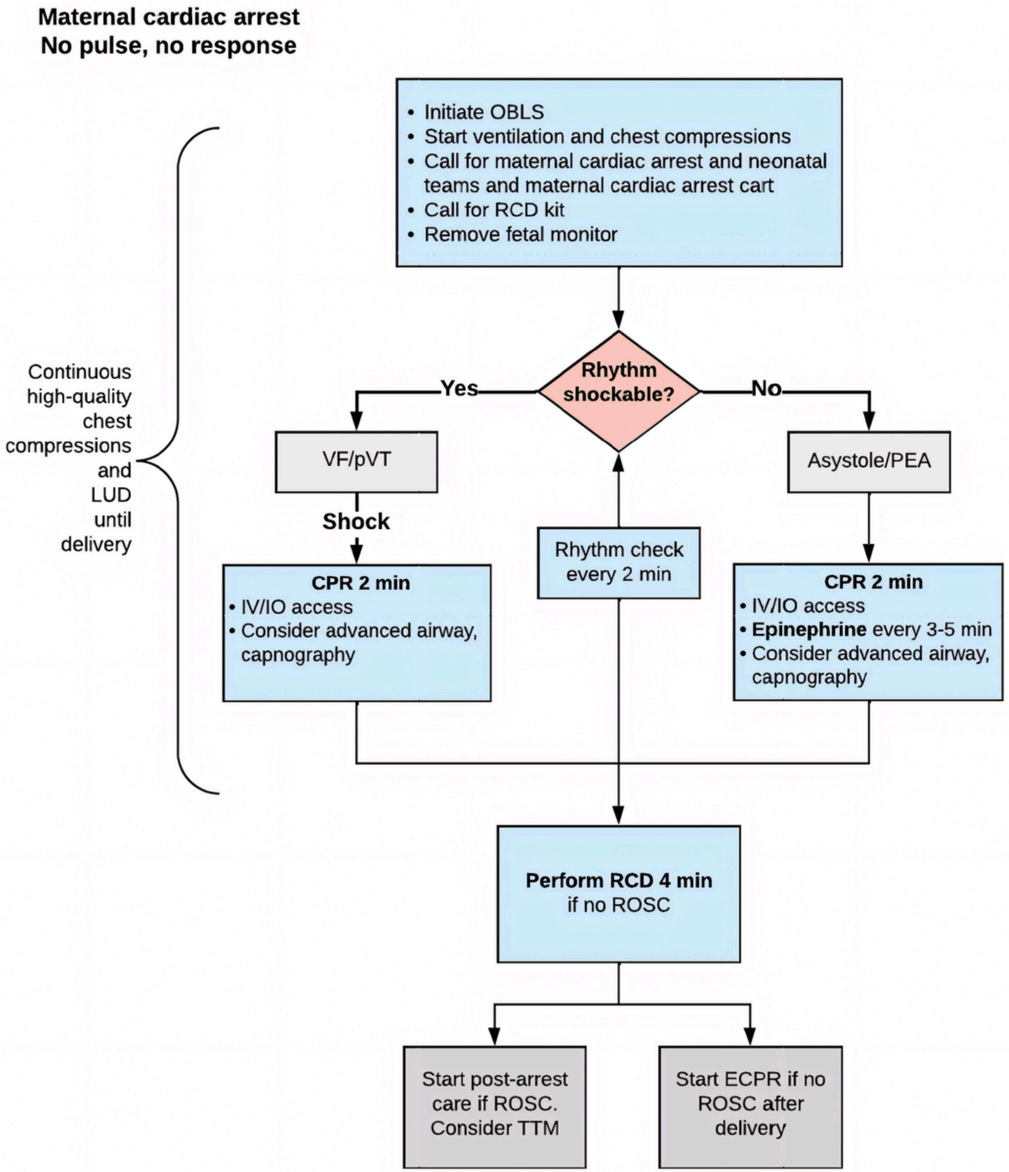


FIGURE 5.6 Obstetric Life Support algorithm with modifications of cardiopulmonary resuscitation (known pregnancy >20 weeks).

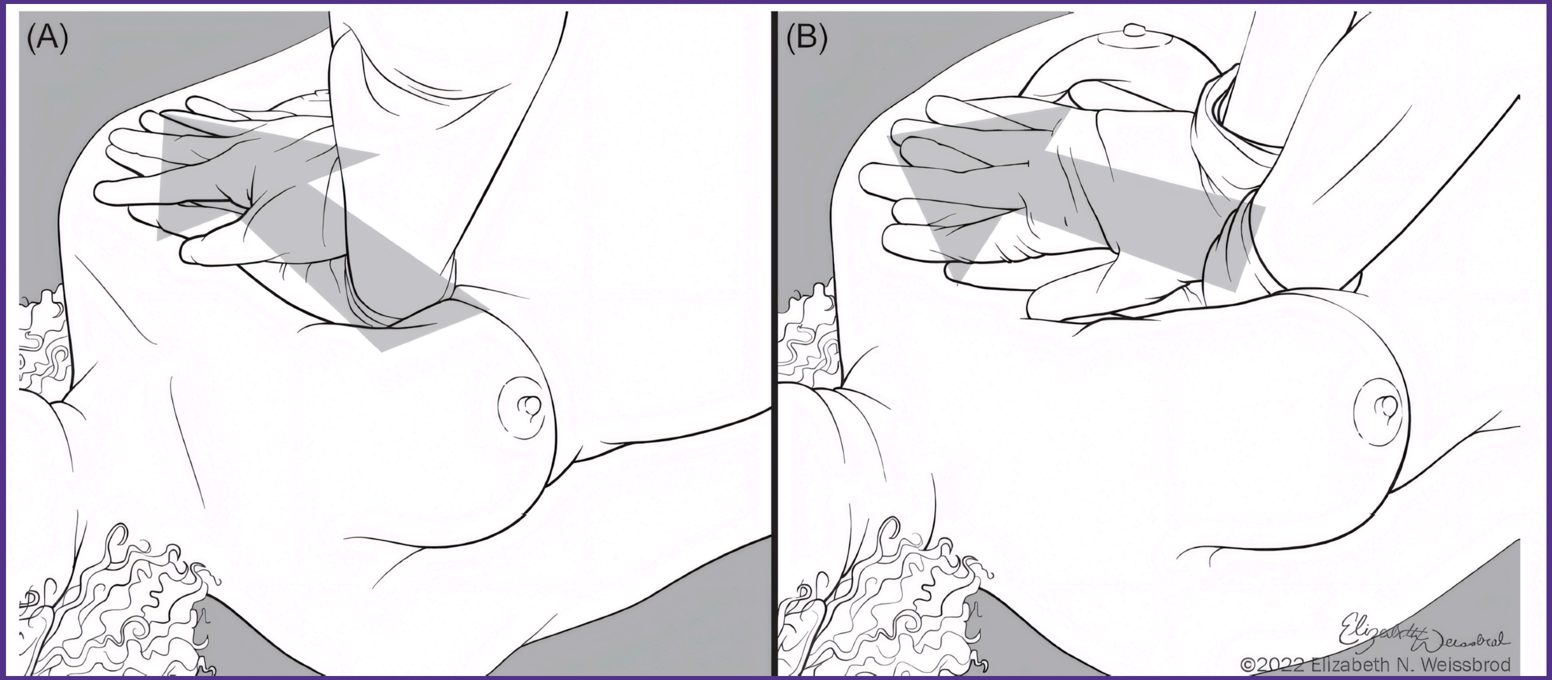
Shields A, Battistelli J, Kavanagh L, Nielsen P, Thomson B, eds. Obstetric Life Support Manual: Etiology, Prevention, and Treatment of Maternal Medical Emergencies and Cardiopulmonary Arrest in Pregnant and Postpartum Patients. First edition. CRC Press; 2024.

A pregnant patient's large, dense breasts may impede the ability to perform effective chest compressions as the fingers may rest atop the breast in an elevated position.

This position may not allow for the proper force to be delivered with each chest compression. If this is the case, keep the heel of the hand on the sternum in the same place and slightly rotate the hand so the fingers point toward the patient's shoulder.

This allows the hand to lay flat on the chest and allows maximum force to be applied to the sternum.

Shields A, Battistelli J, Kavanagh L, Nielsen P, Thomson B, eds. Obstetric Life Support Manual: Etiology, Prevention, and Treatment of Maternal Medical Emergencies and Cardiopulmonary Arrest in Pregnant and Postpartum Patients. First edition. CRC Press; 2024.

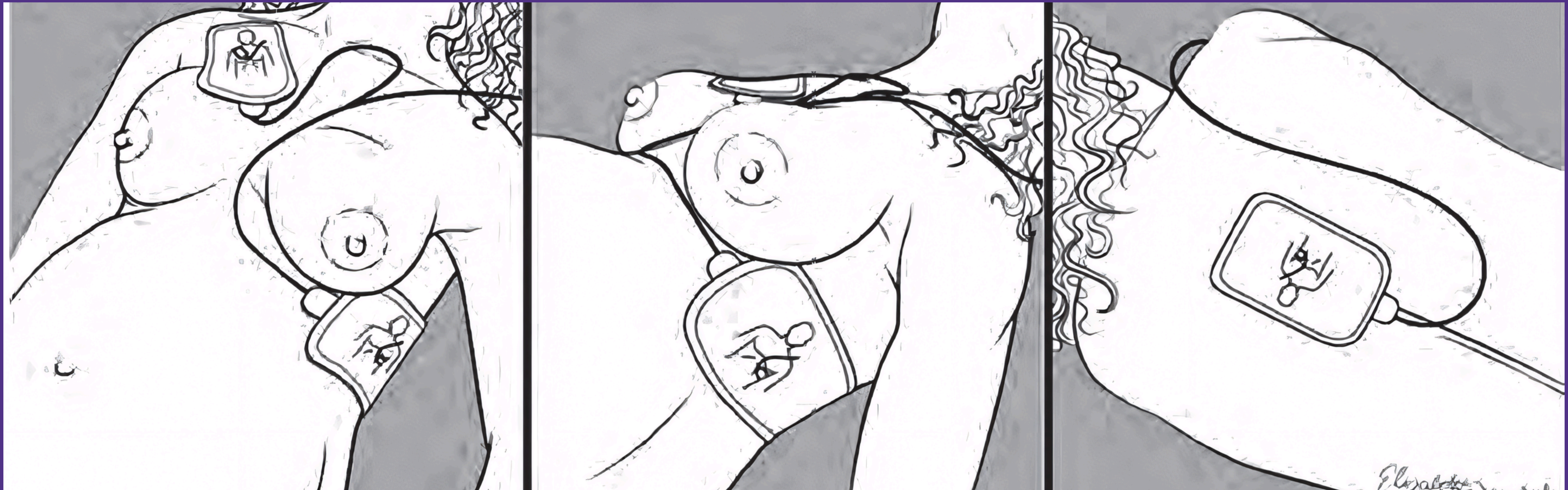


Defibrillation is a key component of resuscitation and should be administered without delay in a pregnant patient with a shockable rhythm. Deliver shocks via a manual defibrillator or an AED. Place pads either anterolateral or anteroposterior, depending on device manufacturer recommendations.

If the patient has a suspected spinal cord injury, place the pads anterolateral to limit movement and potential further injury. In the anterolateral position, elevate and move medially any pendulous left breast tissue, and place the lateral pad under and just lateral to the left breast tissue. The pad should not lay on the breast tissue. Energy doses do not change during pregnancy; use the manufacturers' recommendations for specific devices.

Remove any fetal monitors prior to defibrillation. Though not a significant safety concern if inadvertently left in place, removing fetal monitors focuses attention on maternal care and avoids the theoretical risk of arcing from the defibrillation pads to the fetal monitors.

Shields A, Battistelli J, Kavanagh L, Nielsen P, Thomson B, eds. Obstetric Life Support Manual: Etiology, Prevention, and Treatment of Maternal Medical Emergencies and Cardiopulmonary Arrest in Pregnant and Postpartum Patients. First edition. CRC Press; 2024.



The gravid uterus at term can decrease venous return to the maternal heart by 25%–30%.

In the setting of Maternal Cardiac Arrest, the gravid uterus will also impede return of blood to the maternal heart during CPR.

Thus, **LEFT UTERINE DISPLACEMENT is a critical adjunct in performing high-quality CPR when the uterus is at the level of the umbilicus or higher**.

If the pregnancy is clearly visible on abdominal exam, and the fundus (the top of the uterus) can be felt at or above the umbilicus, the uterus must be manually displaced upward and to the left to allow for improved blood return to the heart.

LUD may be accomplished in one of two ways:

1. Kneel on the **RIGHT** side of the patient, and **PUSH** the uterus leftward and slightly upward (Figure 5.4).

2. Kneel on the **LEFT** side of the patient, and using both hands, **PULL** the uterus leftward and slightly upward (Figure 5.5).

These steps are critical to ensure high-quality CPR in a pregnant person.

Shields A, Battistelli J, Kavanagh L, Nielsen P, Thomson B, eds. Obstetric Life Support Manual: Etiology, Prevention, and Treatment of Maternal Medical Emergencies and Cardiopulmonary Arrest in Pregnant and Postpartum Patients. First edition. CRC Press; 2024.

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Shields A, Battistelli J, Kavanagh L, Nielsen P, Thomson B, eds. Obstetric Life Support Manual: Etiology, Prevention, and Treatment of Maternal Medical Emergencies and Cardiopulmonary Arrest in Pregnant and Postpartum Patients. First edition. CRC Press; 2024. Ganti L, ed. Atlas of Emergency Medicine Procedures. Springer International Publishing; 2022. doi:10.1007/978-3-030-85047-0

Resuscitative Cesarean Delivery

RCD is indicated for any pregnant person with a uterus at or above the umbilicus or ≥20 weeks gestation and has not achieved ROSC in the first 4 minutes of the arrest.

RCD facilitates decompression of the inferior vena cava to improve blood flow back to the heart allowing for autotransfusion as the uterus contracts down after delivery. **RCD should occur either at the site of arrest (if already hospitalized) or upon admission to the ED (if transported by EMS personnel).**

Under certain circumstances, (such as intrapartum, nonshockable rhythms), OBLS recommends immediately performing an RCD.

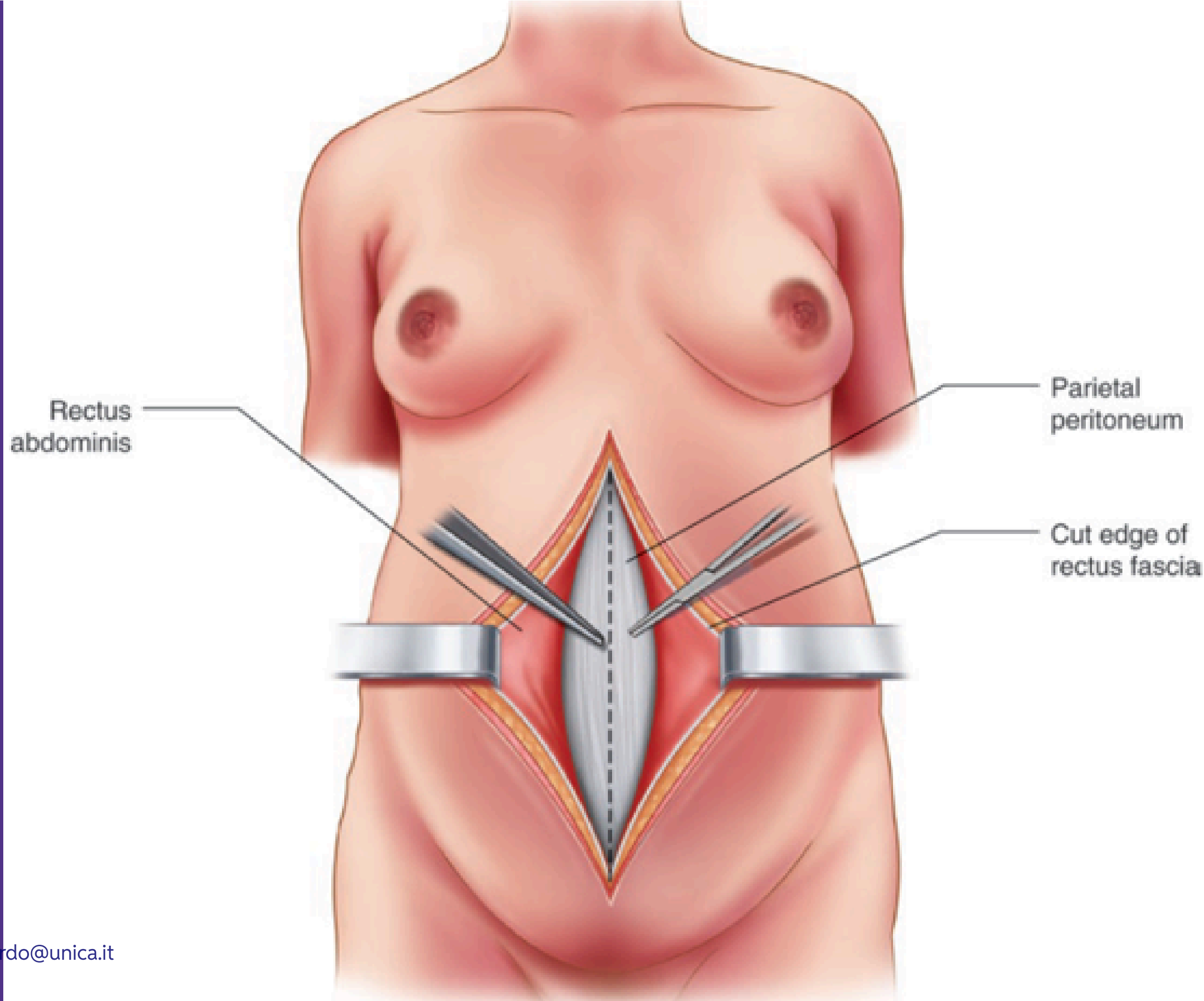

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TABLE 6.2 Factors That Decrease the Effectiveness of Obstetric Life Support

FACTOR	*REDUCED EFFECTIVENESS IN PREGNANT PATIENTS DUE TO*
Quality of CPR	• Dense/large breasts resulting in incorrect hand placement or shallow compressions • Incorrect technique of rolling or wedging the pregnant patient to reduce aortocaval compression (historical teaching) • Not performing LUD when indicated • Not performing RCD when indicated
Inaction	• Too focused on fetal status, resulting in not performing or delaying RCD • Fear that actions may adversely impact the pregnancy (such as withholding resuscitation medications) • Fear of performing RCD due to limited or no real-life experience

Other	• Resuscitation team:
	  • Does not know about or does not suspect pregnancy
	  • Has poor communication
	  • Lacks knowledge of modifications of CPR in pregnancy
	• No communication with the command center about pregnancy in an OH arrest
	• Crew transports patient to a hospital with inadequate resources to care for MCA

Beckett V, Knight M, Sharpe P. The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. BJOG: Int J Obstet Gy. 2017;124(9):1374-1381. doi:10.1111/1471-0528.14521

Maternal Cardiac Arrest in the UK

Surveillance Period

During the 3-year surveillance period, 125 cases were reported to UKOSS in an estimated 2,347,670 maternities.

Case Definition

Of the 125 reported cases, 66 met the case definition, which had to include cardiac compressions following maternal collapse.

Incidence of Maternal Cardiac Arrest

The incidence of maternal cardiac arrest was **2.8 per 100,000 maternities** (95% CI 2.2–3.6).

Final Year Incidence

In the final year of the surveillance period, the estimated incidence of maternal cardiac arrest during pregnancy or immediately postpartum was 6.3 per 100,000 maternities (95% CI 4.7–8.4).

Case Fatality Rate

Twenty-eight women died, representing a **case fatality rate of 42%** (95% CI 30–55%).

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Table 4. Suspected and confirmed (at post mortem) causes for women who died and women who survived

Cause	Women who survived (n = 37)	Women who died (n = 22)
Presumed premortem causes (n = 59)		
Cardiac tamponade	1	0
Hypoxia	4	0
Hypovolaemia	5	8
Venous thromboembolism	1	7
Toxic drug cause	1	0
Anaphylaxis	1	0
Sepsis	0	1
Anaesthetic cause	17	0
Amniotic fluid embolism	5	3
Cardiac cause	5	1
Intracerebral bleed	0	3
Aortic dissection	0	2
Asthma	0	1
Pulmonary artery rupture	0	1
Postmortem causes of collapse (n = 19)		
Amniotic fluid embolism		6
Vessel bleed/rupture		5
Thrombembolic		3
Cardiomyopathy		2
Other		3

Data were available for 59 women. Some women were suspected of having more than one cause, where this is the case both causes have been recorded.

Data were available for 59 women regarding the suspected cause of maternal arrest. In 19 women, the cause identified at post-mortem was also recorded. In the women who survived (n = 38; 58%), information about a presumed cause for maternal collapse was recorded for 37 women.

Of the 28 women who died, data were provided ascribing a cause of death in 21 (75%). **Sixteen women had cardiac arrest as a direct complication of obstetric anesthesia**. One additional woman was reported as having two events, both capable of causing a cardiac arrest.

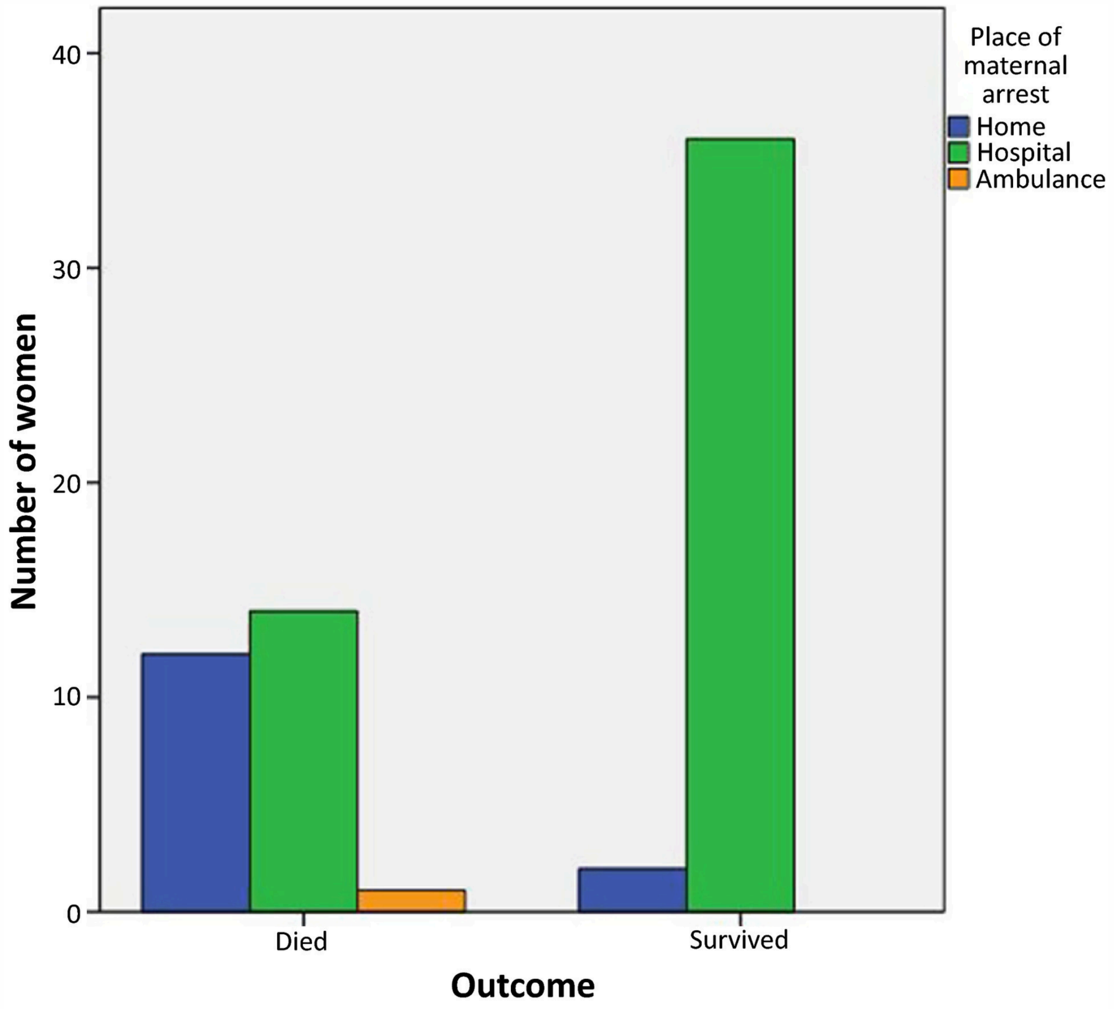
These anaesthetic causes were: problem with intubation (n = 3), cardiovascular collapse following epidural top-up (n = 3), total spinal anaesthetic (from de novo intrathecal block) (n = 10), and other anaesthetic-related reasons (n = 1).

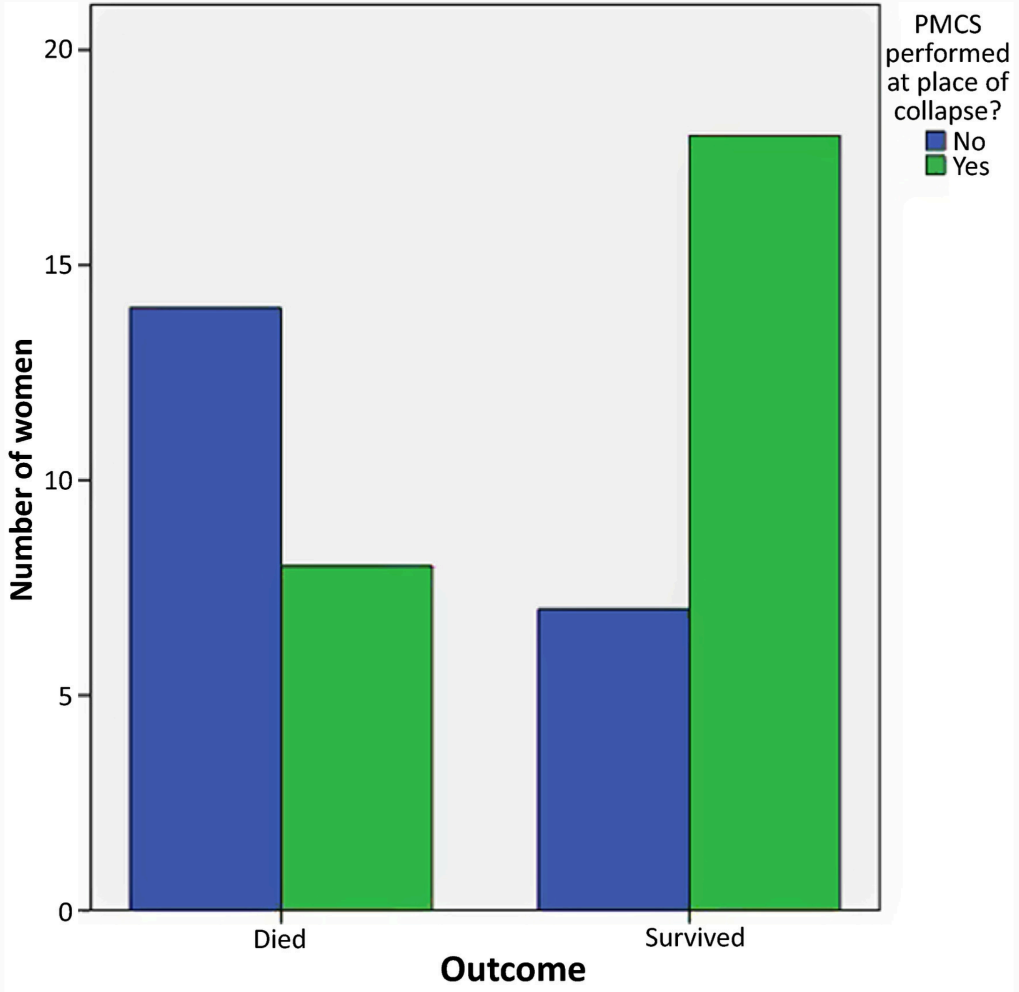
All of these women survived. **Note that 12 of the 16 women who had a cardiac arrest solely due to obstetric anaesthesia were obese, and this may explain the apparent association between maternal obesity and survival.**

Of the postnatal arrests reported in the final year (n = 26), the most common reasons for the collapse were hypovolaemia (n = 11), amniotic fluid embolism (n = 5), and thromboembolic events (n = 3). The longest delivery-to-arrest interval was 12 hours and 25 minutes, with 16 of the postnatal arrests occurring within an hour of delivery.

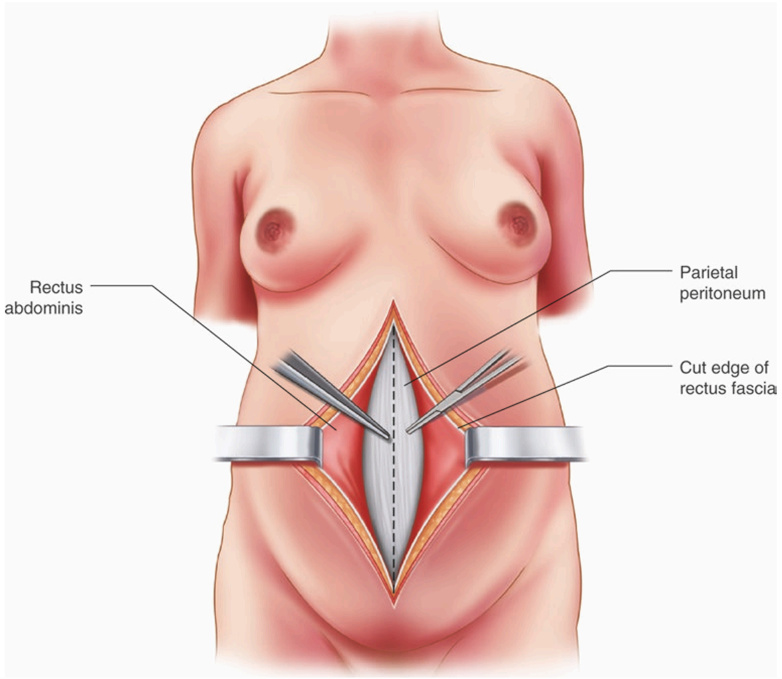
In total, seven of the 26 women died.

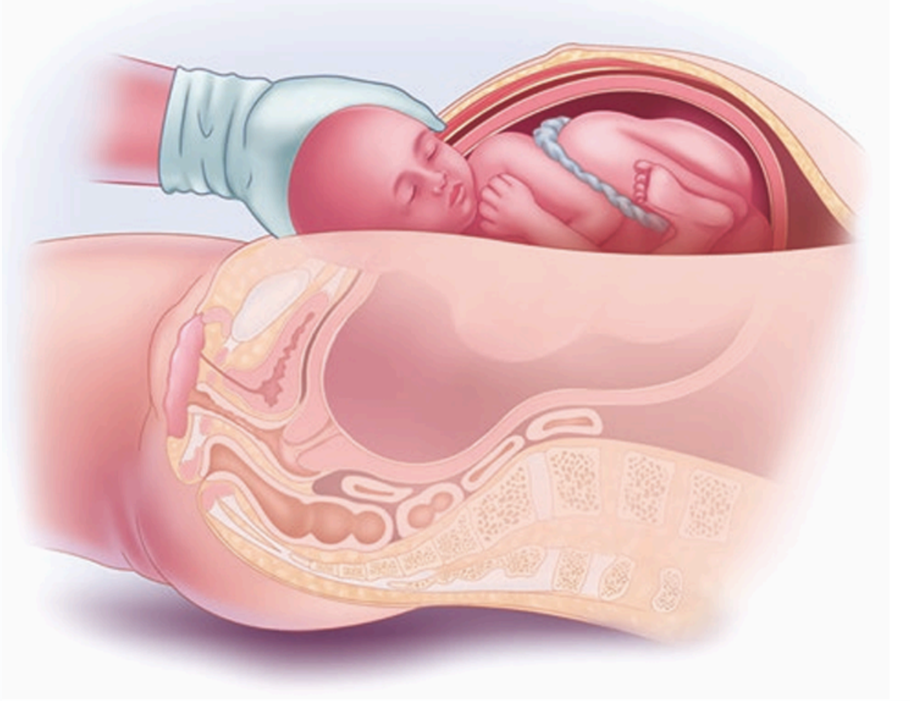
Beckett V, Knight M, Sharpe P. The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. BJOG: Int J Obstet Gy. 2017;124(9):1374-1381. doi:10.1111/1471-0528.14521





ckett V, Knight M, Sharpe P. The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. BJOG: Int J Obstet Gy. 2017;124(9):1374-1381. doi:10.1111/1471-0528.14521





Data were available for 58 babies, of whom 46 were born alive, 32 babies to surviving mothers, and 14 to women who died. Seven of the mothers of the 12 stillborn babies also died. Data were available for 35 of the 49 cases regarding the time of PMCS and survival.

Twenty-four of 25 babies survived (96%) when PMCS was performed within 5 minutes compared with seven of 10 babies (70%) when PMCS occurred >5 minutes after cardiac arrest (P = 0.059), noting the association between location of maternal collapse and delay in PMCS.

Birth weights ranged from 1766 to 2744 g in the babies of mothers who died compared with from 2489 to 3329 g in the babies of women who survived, reflecting a more advanced gestational age.

Some surviving babies had neurological and respiratory complications in both maternal groups, but the numbers of affected babies reported were too small to make any further comment. In total, seven babies had neurological complications, six had respiratory complications, and two had sepsis.

We did not collect data following discharge from the neonatal unit. Five babies, born alive, were subsequently recorded as dying in the early postnatal period.

Beckett V, Knight M, Sharpe P. The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. BJOG: Int J Obstet Gy. 2017;124(9):1374-1381. doi:10.1111/1471-0528.14521

Ganti L, ed. Atlas of Emergency Medicine Procedures. Springer International Publishing; 2022. doi:10.1007/978-3-030-85047-0

Section 15

BLSD in infants and children

PAEDIATRIC LIFE SUPPORT
KEY MESSAGES

GUIDELINES 2025
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Early recognition is crucial

Use quick-look tools like the BBB-tool of the Paediatric Assessment Triangle to recognise critical illness

Use three steps to save a child: Check - Call - CPR

Call for help as soon as you identify an unconscious child

Systems

Systems should aim to link all parts of the chain of survival and establish clear protocols for life-threatening conditions in children

ABCDE

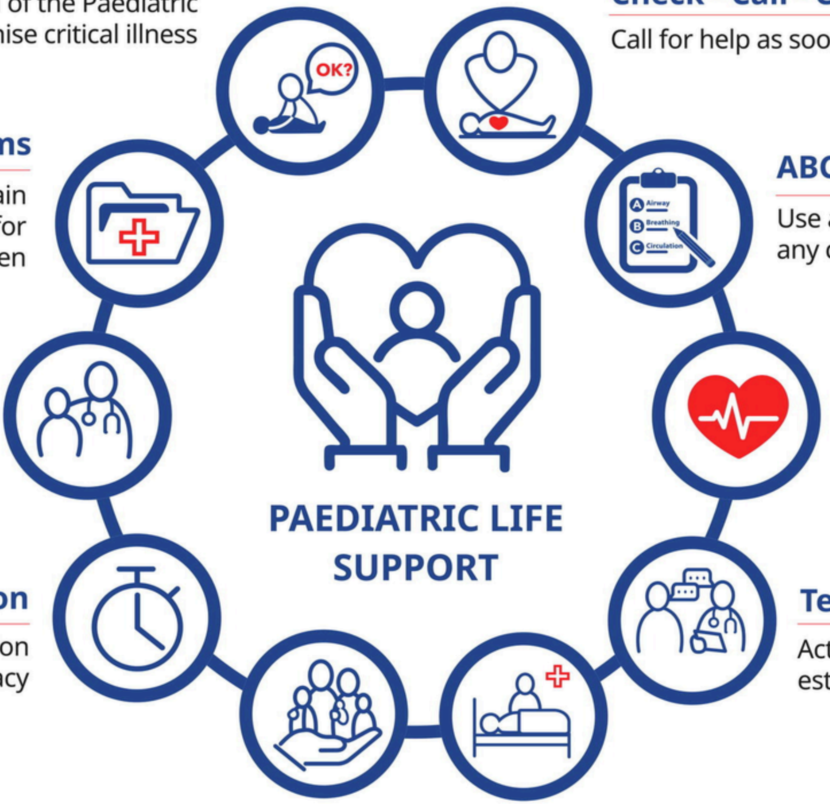
Use a structured ABCDE approach to treat any child who appears critically ill or injured

Post-discharge care

Use structured post-discharge care considering patient and family needs as standard of patient care

Paediatric advanced life support

Follow the PALS algorithm while considering and treating relevant reversible causes of cardiac arrest and modify approach in special circumstances



Prognostication

No single modality can be used in isolation for prognostication with high accuracy

Team approach

Activate additional resources early and establish a team with clearly defined roles

Family-centred approach

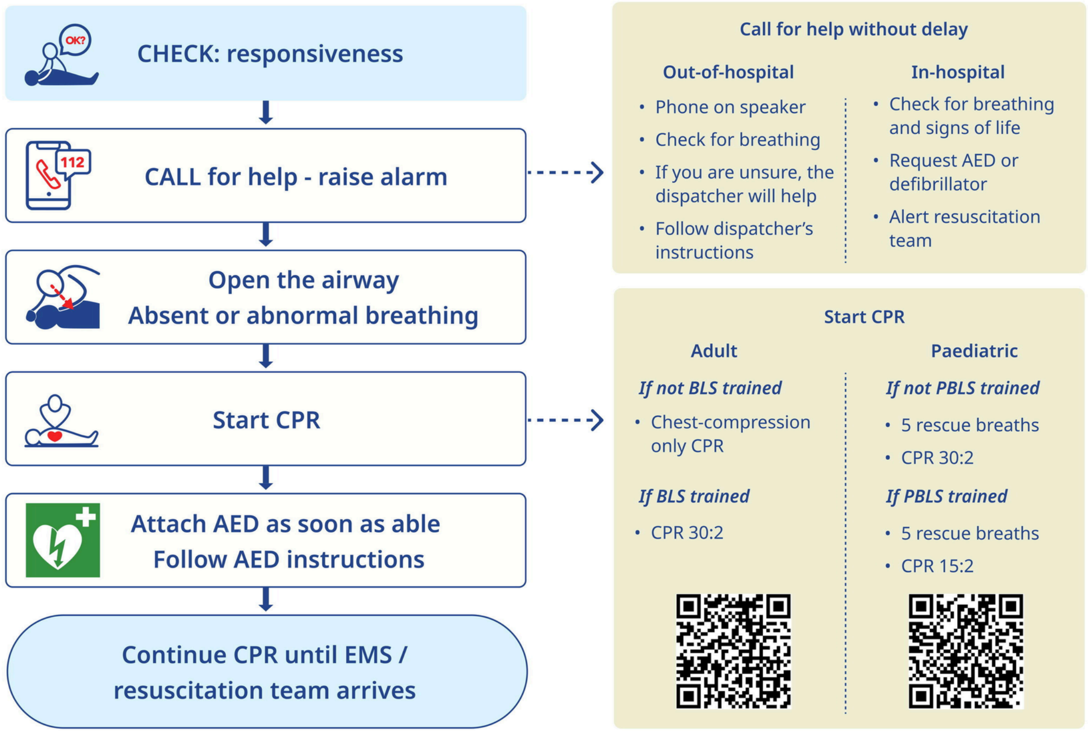
Involve parents/caregivers at all stages of care
Communicate with honesty and empathy while considering the needs of the family

Post-resuscitation care

Initiate post-resuscitation care immediately after ROSC
Implement individualised goals and care bundles

Djakow J, Turner NM, Skellett S, et al. European resuscitation council guidelines 2025 paediatric life support. *Resuscitation*. 2025;215:110767. doi:10.1016/j.resuscitation.2025.110767

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL©



Pediatric basic life support (BLS) is not simply a scaled-down version of the adult algorithm. The epidemiology, pathophysiology and common aetiologies of paediatric cardiorespiratory arrest are different from those in adults.

Cardiorespiratory arrest in infants and children is NOT usually the result of a primary cardiac cause but instead is the end result of progressive respiratory failure or shock.

To reflect these differences, the ideal sequence of BLS is different in the paediatric, newborn, and adult populations.

This must be balanced against the educational advantages of training using a general BLS algorithm so that, where possible, guidelines are standardized for all ages to aid teaching and retention.

Djakow J, Turner NM, Skellett S, et al. European resuscitation council guidelines 2025 paediatric life support. *Resuscitation*. 2025;215:110767. doi:10.1016/j.resuscitation.2025.110767

Smith S, Advanced Life Support Group (Manchester, England), editors. Advanced paediatric life support: a practical approach to emergencies. Seventh edition. Hoboken, NJ: Wiley-Blackwell; 2023.

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Paediatric basic life support

2025

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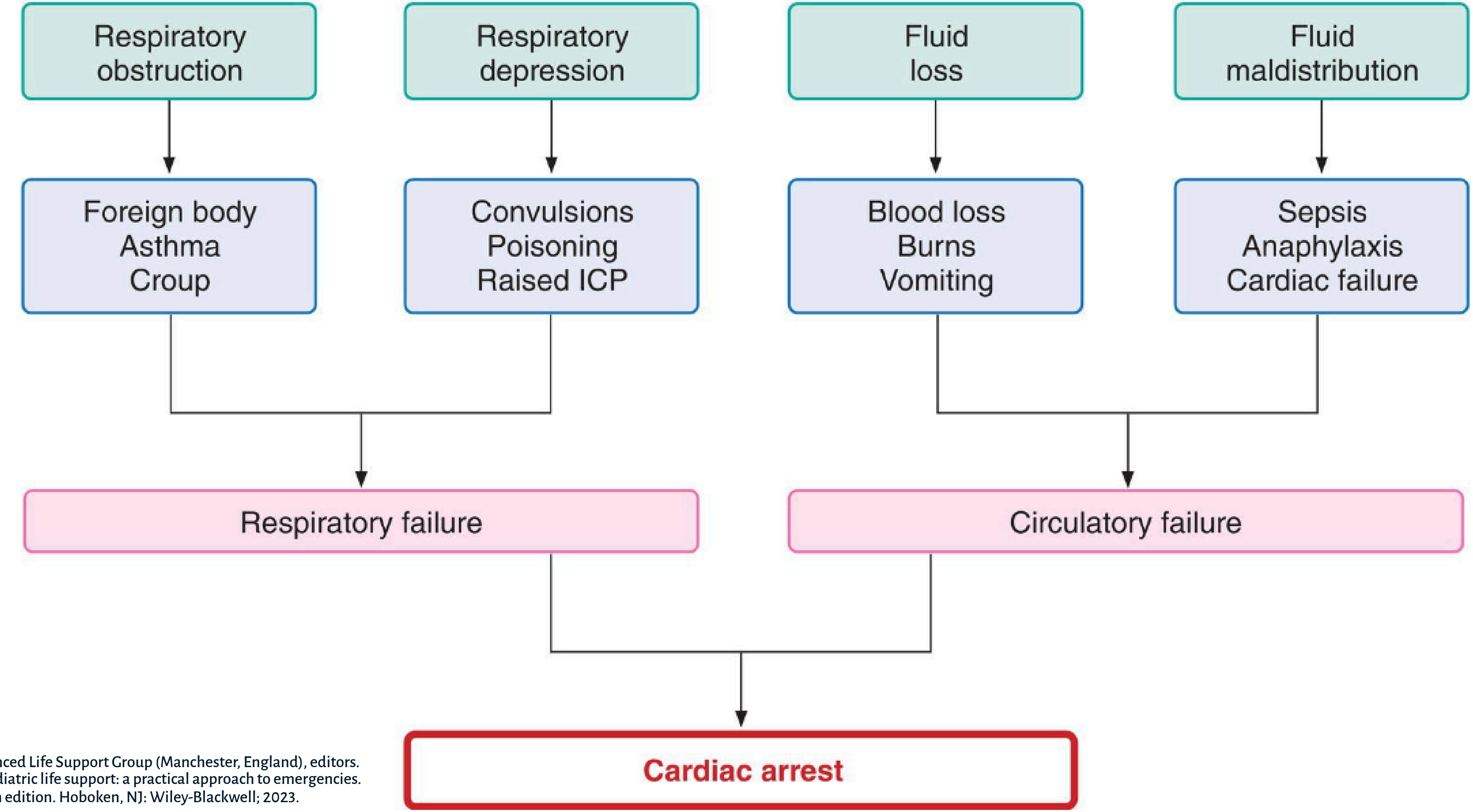
Pathways to cardiac arrest



Smith S, Advanced Life Support Group (Manchester, England), editors. Advanced paediatric life support: a practical approach to emergencies. Seventh edition. Hoboken, NJ: Wiley-Blackwell; 2023.

GUIDELINES 2025

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	A - Airway	B - Breathing	C - Circulation	D - Disability	E - Exposure
Recognition	• Partial or total airway obstruction • Look-listen-feel for air entry and chest rise	• Respiratory failure • Respiratory rate, work of breathing, tidal volume, oxygenation	• Shock and its type • Pulse rate, pulse volume, peripheral perfusion, blood pressure, preload, rhythm	• Neurological failure • Posture, pupils, AVPU, pGCS, lateralisation, tone, seizures	• Special circumstances • Exposure and physical examination
Monitoring & investigations		• SpO_2, $EtCO_2$, ABG, POCUS	• ECG-monitor, NIBP • 12-lead ECG, POCUS, POC blood samples incl. lactate, urine output	• Blood glucose	• Body temperature • AMPLE
Treatments & considerations	• Suction, foreign body removal, basic manoeuvres for positioning, oropharyngeal or nasopharyngeal airways, supraglottic devices (intubation, FONA)	• Oxygen + titration of FiO_2 • Bag-mask ventilation • Mechanical ventilation • ECMO	• IV/IO, isotonic crystalloids, blood products • Vasopressors, inotropes • Treatment of arrhythmias • Specific interventions according to the type of shock, incl. ECMO	• Neuroprotective strategies (treat seizures & hypoglycaemia, analgesia, sedation) • Signs of paediatric stroke or neuroinfection	• Antibiotics, antivirals • Treatments and interventions for special circumstances • Avoiding and treating hypothermia and hyperthermia • Child abuse & neglect
Goals	• Airway allowing for adequate oxygenation and ventilation	• Adequate oxygenation • Adequate ventilation	• Adequate organ perfusion, SBP & MAP above 5^{th} percentile	• Neuroprotection	• Identification of underlying disease
High risk of sudden CA	• Airway obstruction	• Unstable or silent chest, tension pneumothorax	• Exsanguinating haemorrhage, tamponade	• Intracranial hypertension, hypoglycaemia	• Hypothermia, severe trauma, thrombosis

Recognise emergency, support organs, treat underlying condition, reassess and include family

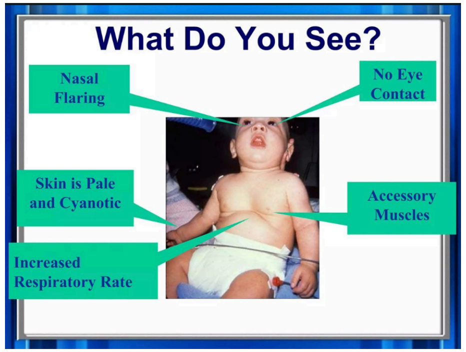
Djakow J, Turner NM, Skellett S, et al. European resuscitation council guidelines 2025 paediatric life support. *Resuscitation*. 2025;215:110767. doi:10.1016/j.resuscitation.2025.110767

Immediately call the emergency medical service (EMS) if

Djakow J, Turner NM, Skellett S, et al. European resuscitation council guidelines 2025 paediatric life support. Resuscitation. 2025;215:110767. doi:10.1016/j.resuscitation.2025.110767

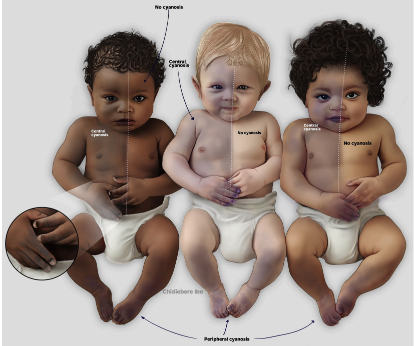
Behaviour

• Reduced consciousness or difficult to rouse, floppy or rigid posture. • Seizures. • Confusion, agitation, or abnormal interaction with caregivers. • Inconsolable crying. • Inability to move one or more limbs. • Severe pain or inability to speak or walk when previously able.



Breathing

• Difficulty breathing or inability to take a deep breath. • Increased work of breathing such as rapid breathing, grunting, nostril flaring, or chest indrawing. • Abnormal breathing sounds. • Breathing that is too fast, too slow, irregular, or absent. • Abnormal posture adopted to help breathing.



Body colour

• Cyanosis, mottled skin, marked pallor, or greyish colour. • Assess palms, soles, and mucous membranes, especially in children with darker skin tones.

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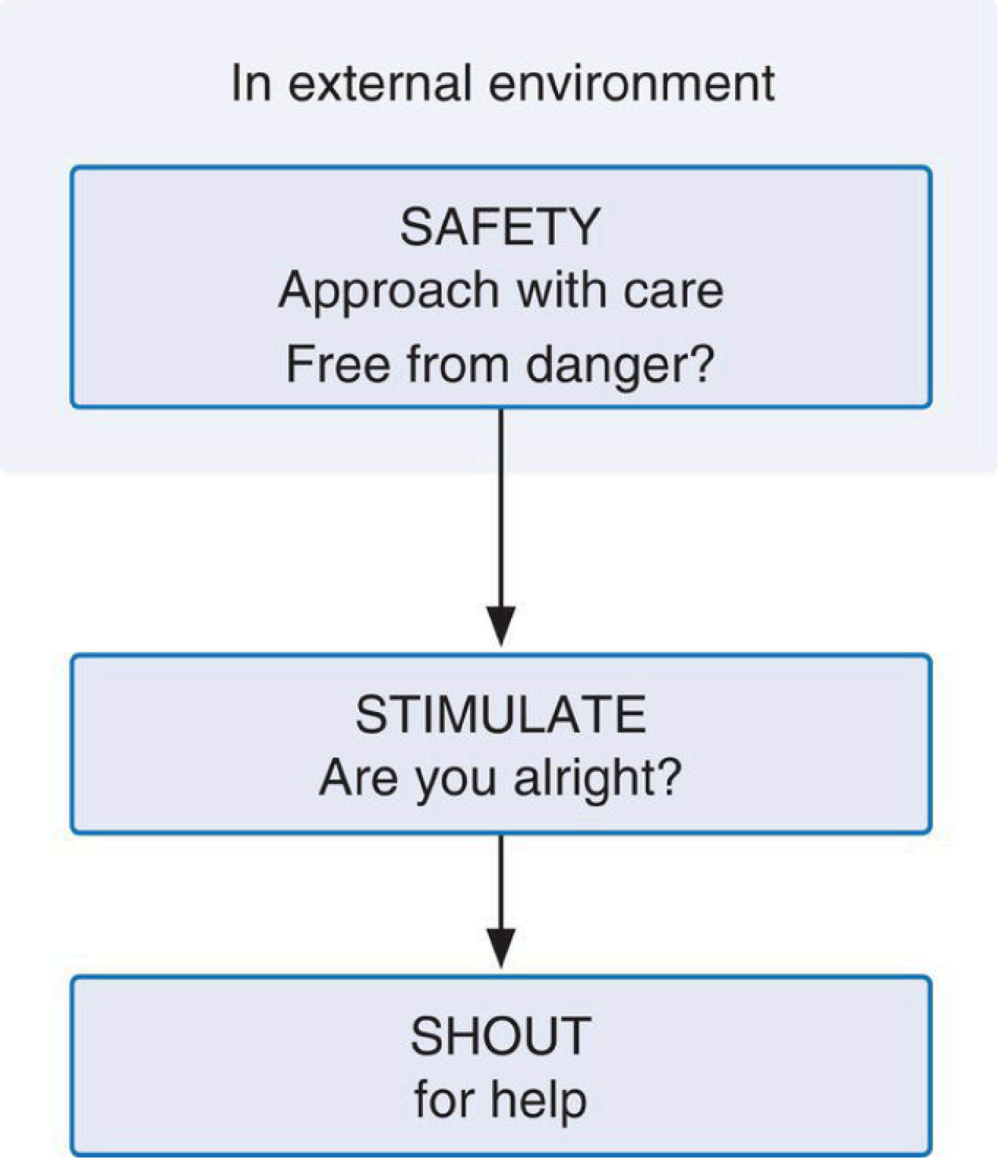
S – Safety In the external environment, it is essential that the rescuer does not become a second victim and that the child is removed from continuing danger as quickly as possible. These considerations should precede the initial airway assessment. Within a healthcare setting, the likelihood of risk is minimal, and help should be summoned as soon as the victim is found to be unresponsive.

S – Stimulate The initial simple assessment of responsiveness consists of asking the child loudly, 'Are you alright?' and gently applying a stimulus, such as holding the head and shaking the arm. This will avoid exacerbating a possible neck injury whilst still waking a sleeping child. Infants and very small children who cannot talk yet, and older children who are very scared, are unlikely to reply meaningfully, but may make some sound or open their eyes to the rescuer's voice or touch.

S – Shout for help When more than one rescuer is present, one should commence BLS while others activate the emergency medical services (EMS) system, collect the emergency equipment (e.g., manual defibrillator or automated external defibrillator (AED)), and then return to assist in the BLS effort (including applying defibrillator pads). Bystanders may be asked to help.

If there is only one rescuer, they should call for help immediately (preferably using a mobile phone with speaker function) and then commence cardiopulmonary resuscitation (CPR). If no phone is available, the single rescuer should perform 1 minute of CPR before leaving the child to activate the EMS system. If the patient is a baby or small child, the rescuer may be able to take the patient to a telephone while performing CPR on the way.



Smith S, Advanced Life Support Group (Manchester, England), editors. Advanced paediatric life support: a practical approach to emergencies. Seventh edition. Hoboken, NJ: Wiley-Blackwell; 2023.

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Head tilt and chin lift in infants: neutral position in an infant

If a child is not breathing, it may be due to their airway being blocked by the tongue falling back and obstructing the pharynx. Correcting this obstruction can lead to a quick recovery without further intervention.

To open the airway initially, the rescuer should use the head tilt/chin lift maneuver. Start by placing the hand closest to the child's head on their forehead, applying gentle pressure to tilt the head back. With the fingers of the other hand, lift the chin upwards to raise the base of the tongue away from the back of the throat, thereby improving airway patency. It's important to be gentle to avoid injuring the soft tissue by gripping too tightly. Since this maneuver may close the child's mouth, the rescuer may need to use their thumb from the same hand to slightly part the lips.

For infants, the airway is best optimized by tilting the head into a neutral position.

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Head tilt and chin lift 'sniffing' position in a child

If a child is not breathing, it may be due to their airway being blocked by the tongue falling back and obstructing the pharynx. Correcting this obstruction can lead to a quick recovery without further intervention.

To open the airway initially, the rescuer should use the head tilt/chin lift maneuver. Start by placing the hand closest to the child's head on their forehead, applying gentle pressure to tilt the head back. With the fingers of the other hand, lift the chin upwards to raise the base of the tongue away from the back of the throat, thereby improving airway patency. It's important to be gentle to avoid injuring the soft tissue by gripping too tightly. Since this maneuver may close the child's mouth, the rescuer may need to use their thumb from the same hand to slightly part the lips.

The older child's airway is better placed with the neck more extended in the 'sniffing' position.

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Jaw thrust maneuver

If the head tilt/chin lift maneuver is not possible or is contraindicated (e.g., suspected neck injury), then the jaw thrust maneuver can be performed. This is achieved by placing one or two fingers bilaterally under the angle of the mandible and lifting the jaw upwards (towards the sky). This technique may be easier if the rescuer's elbows are resting on the same surface as the child is lying on. A small degree of head tilt may also be applied if there is no concern about neck injury.

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It should be noted that if there is a history of trauma, then the head tilt/chin lift maneuver may exacerbate cervical spine injury.

In general, the safest airway intervention in these circumstances is the jaw thrust without head tilt. However, on rare occasions in trauma, it may not be possible to control the airway with a jaw thrust alone.

In these circumstances, an open airway takes priority over cervical spine risk, and a gradually increased degree of head tilt may be tried.

Cervical spine control should be achieved by a second rescuer maintaining manual in-line cervical stabilisation throughout.

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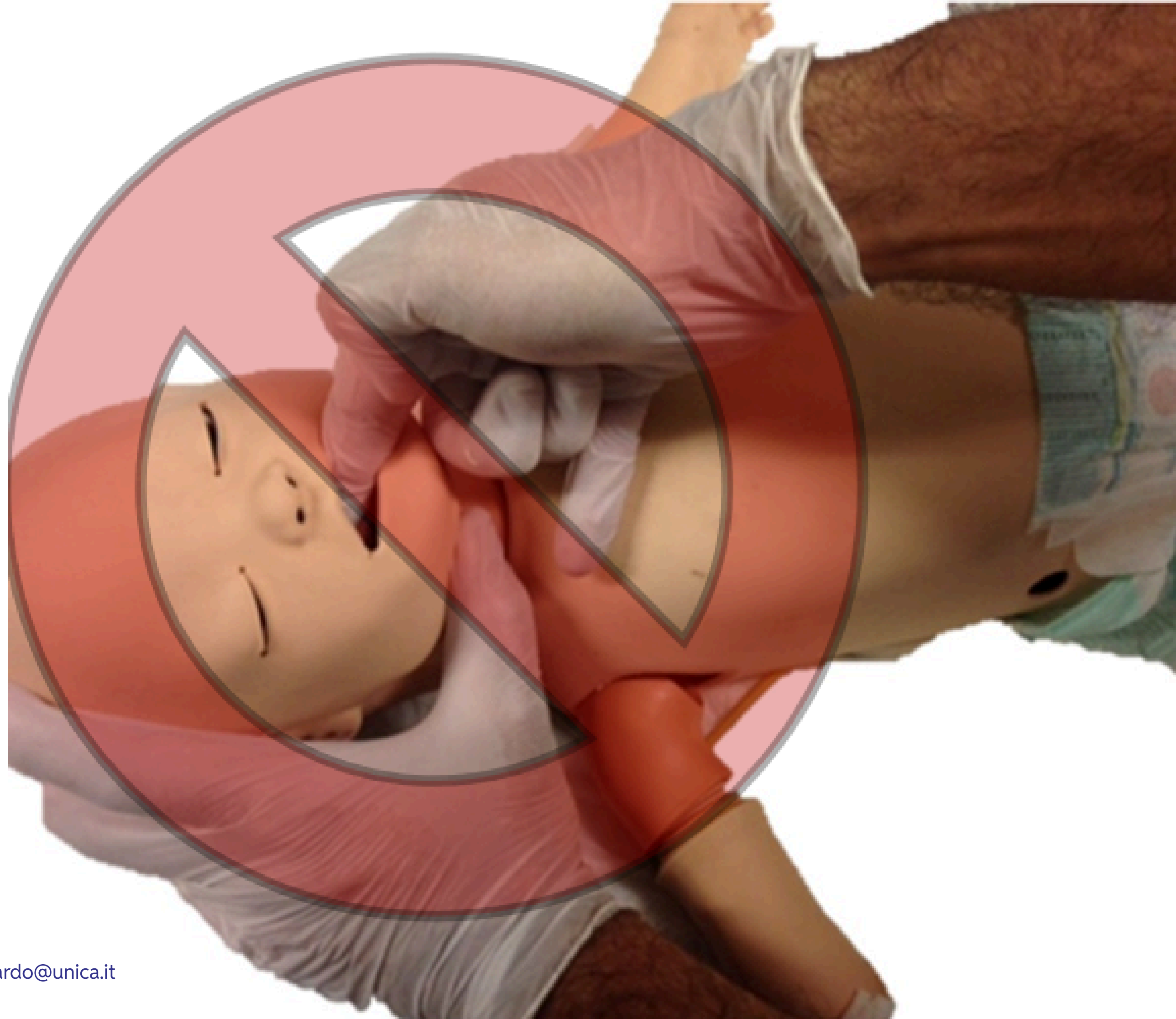
Blind finger sweep technique

CONTRAINDICATED

The blind finger sweep technique for the removal of a foreign body should not be used in children.

The child's soft palate is easily damaged, and bleeding from within the mouth can worsen the situation. Furthermore, foreign bodies may be forced further down the airway, becoming lodged below the vocal cords (vocal folds), making removal even more difficult.



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Tonson la Tour A, Sanchez O, Gervaix A, Vunda A (2017) Blind Finger Sweep Maneuver is Not Only Dangerous but Could Be Fatal. J Emerg Med Trauma Surg Care 1: 003.

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Ventilation

While the airway is kept open as described, the rescuer breathes in and seals their mouth around the victim's mouth (for a child) or mouth and nose. If the mouth alone is used, then the nose may be pinched closed using the thumb and index fingers of the hand to maintain the head tilt. Slow exhalation (1 second) by the rescuer should cause the patient's chest to rise visibly.

Too vigorous a breath will cause gastric inflation and increase the chance of regurgitation of stomach contents into the lungs. The rescuer should take a breath between rescue breaths to maximize the oxygenation of the victim.

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If the chest does not rise, then the airway is not clear.

The usual cause is failure to correctly apply the airway-opening techniques. Therefore, the first thing to do is to readjust the head tilt/ chin lift position and try again. If this does not work, a jaw thrust should be tried. It is not always feasible for a single rescuer to open the airway using this technique and perform exhaled air resuscitation; if two rescuers are present, one should maintain the airway whilst the other breathes for the child. Up to five attempts should be made to achieve effective breaths. If still unsuccessful, the rescuer should move on to chest compressions.

Failure of both head tilt/chin lift and jaw thrust should lead to the suspicion that a foreign body is causing the obstruction, and appropriate action should be taken.

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Chest compressions: infants

Infant chest compression is most effectively performed using the two- thumb tech nique: the infant is held with both the rescuer's hands encircling or partially encircling the chest. The thumbs are placed over the lower half of the sternum and compression performed.

The single rescuer may alternatively use the two- finger method, placing two fingers on the lower half of the sternum and employing the other hand to maintain the airway position.

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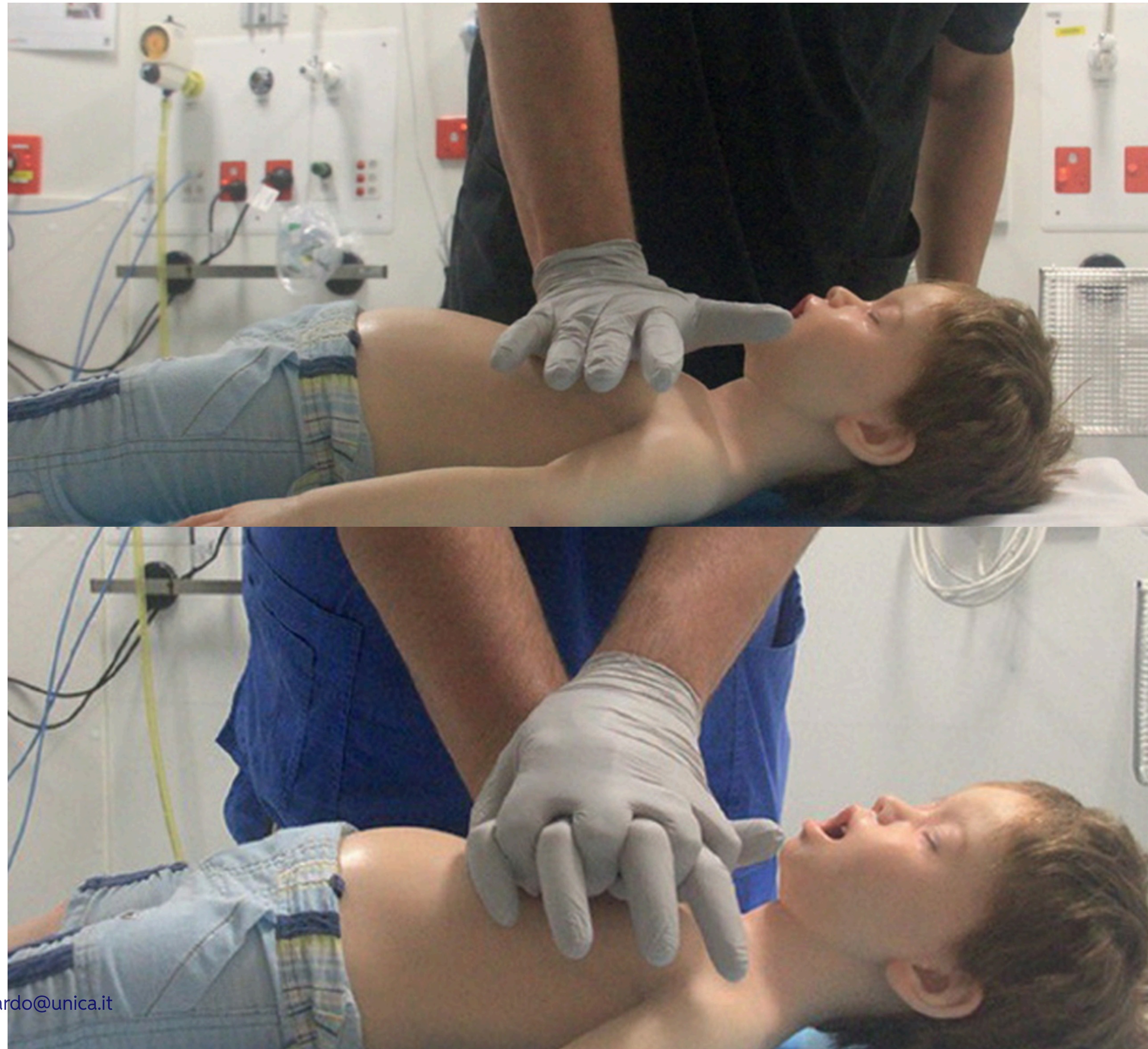
Chest compressions: children

The rescuer should place the heel of one hand over the lower half of the sternum. The fingers should be lifted to ensure that pressure is not applied over the child's ribs. The rescuer is best positioned vertically above the child's chest, and, with a straight arm (elbow extended), the sternum is compressed to depress it by at least **one-third of the depth of the chest** (at least 5 cm).

For larger children or for small rescuers, this may be achieved more reliably by using both hands with the fingers interlocked. The rescuer should choose one or two hands to achieve the desired compression of at least one-third of the depth of the chest. It is important to avoid leaning and to allow for complete chest recoil in between.



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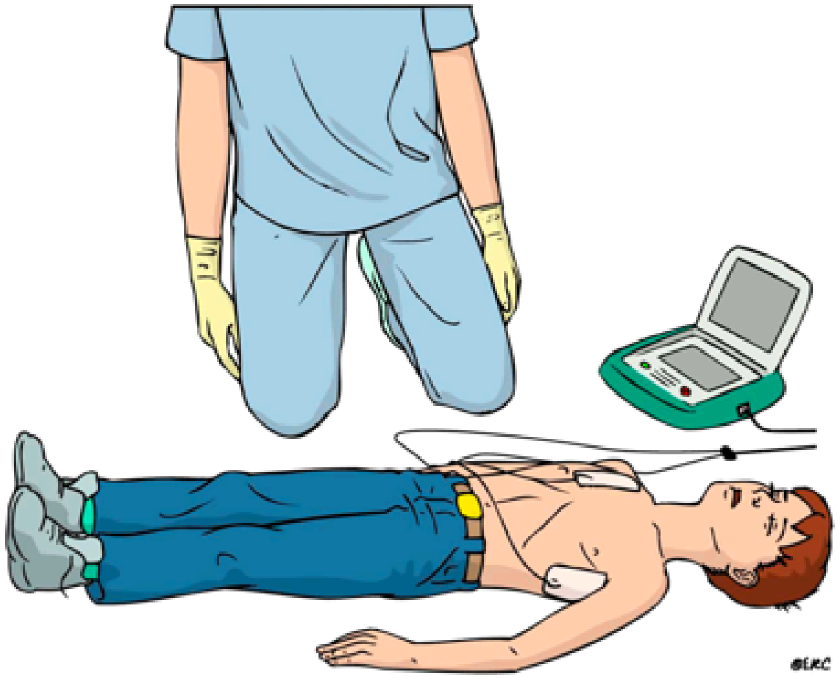
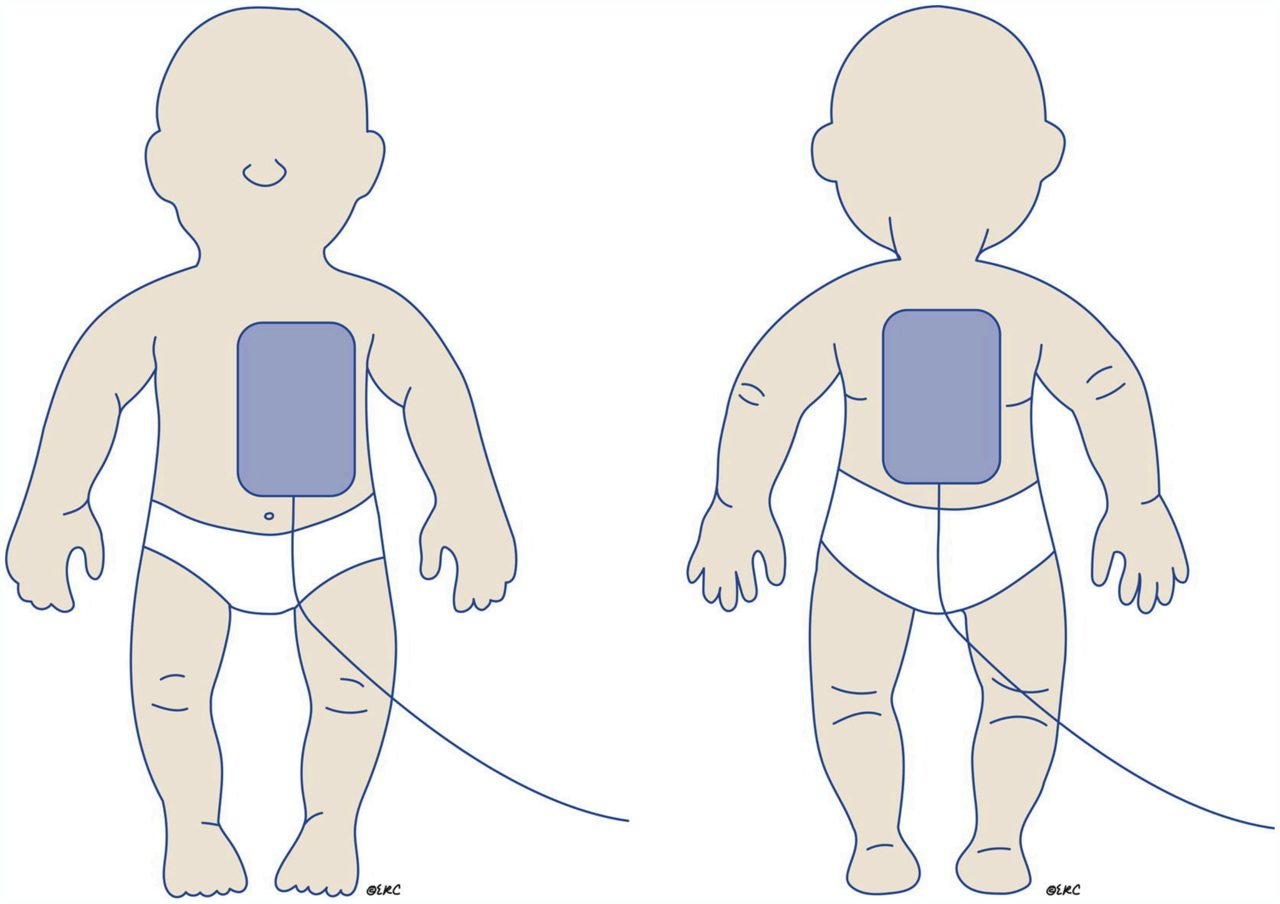
AED in children

- Age > 8 years
  - use adult AED
- Age 1-8 years
  - use paediatric pads / settings if available (otherwise use adult mode)
- Age < 1 year
  - use only if manufacturer instructions indicate it is safe





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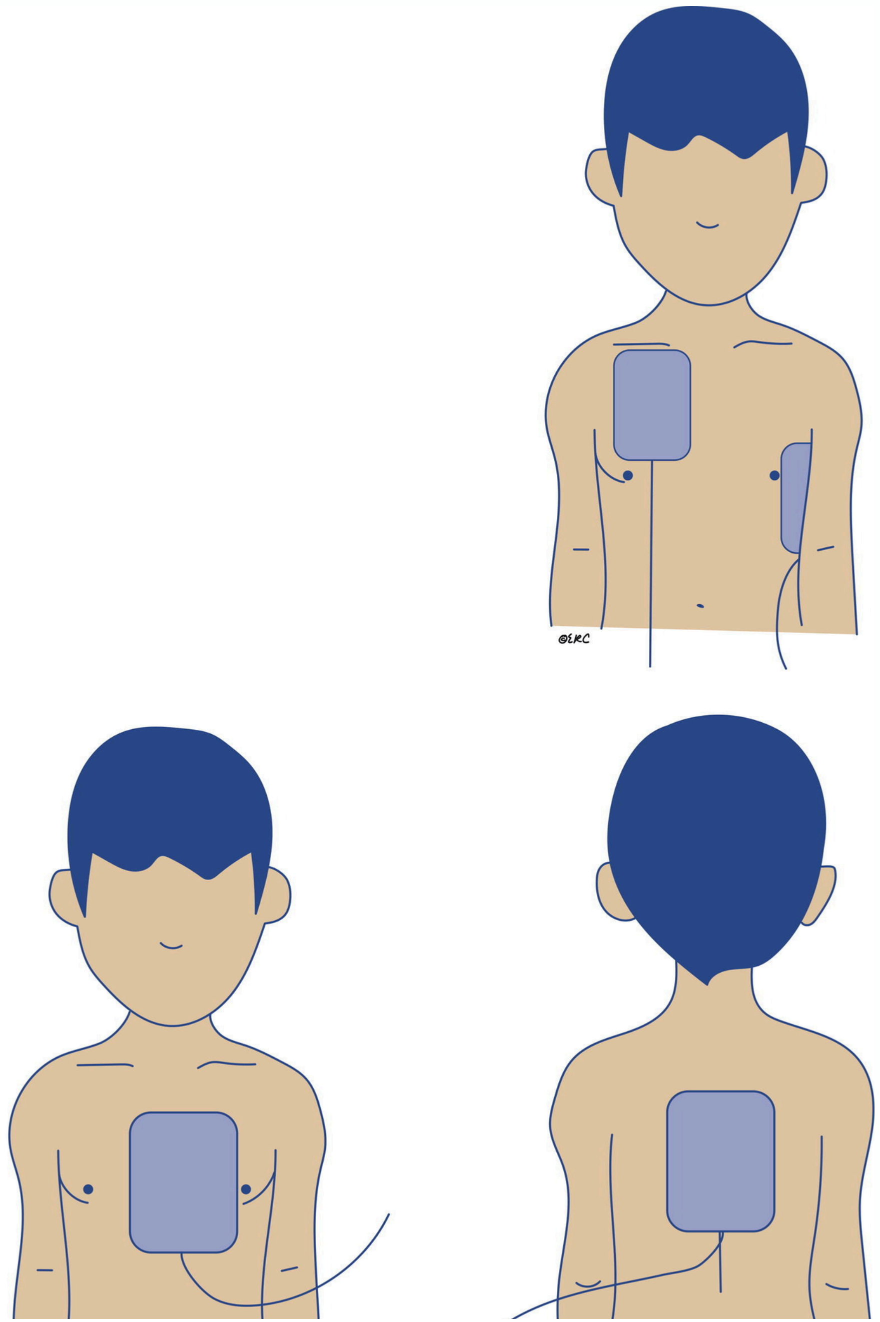

Djakow J, Turner NM, Skellett S, et al. European resuscitation council guidelines 2025 paediatric life support. *Resuscitation*. 2025;215:110767. doi:10.1016/j.resuscitation.2025.110767

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Defibrillation Pad Placement for Pediatric Patients

- ## Pad Size

  Appropriately sized adhesive defibrillation pads should be used. The recommended sizes are 4.5 cm for children weighing less than 10 kg and 8–12 cm for children more than 10 kg.

- ## Pad Placement 1

  One pad is placed over the apex of the heart in the mid-axillary line.

- ## Pad Placement 2

  The other pad is applied immediately below the clavicle just to the right of the sternum.

- ## Alternative Pad Placement

  If only adult pads are available or if the pads are too large for the infant/child (and they overlap on application), one pad should be placed on the upper back (below the left scapula) and the other on the front (below the clavicle and to the left of the sternum) in an anteroposterior configuration.

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Defibrillation in Children and Infants

AEDs for Children Over 8 Years

AEDs can be used on children over 8 years of age, using standard adult shock doses if a manual defibrillator is not available.

Pediatric Pads for Children Under 8 Years

For children under 8 years, attenuated pediatric pads should preferably be used with the AED.

Defibrillation for Infants Under 1 Year

In infants under 1 year, a manual defibrillator that can be adjusted to give the correct shock is recommended. If an AED is the only option, it should be used with pediatric attenuation pads or a pediatric setting.

Preference for Defibrillation in Infants

The order of decreasing preference for defibrillation in infants is: 1. Manual defibrillator, 2. AED with dose attenuator or pediatric switch, 3. AED without dose attenuator.

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Table 16.1 Summary of basic life support techniques in infants and children

	Infant (under 1 year)	**Child (1 year to puberty)**
Airway		
Head tilt position	Neutral	Sniffing
Breathing		
Initial slow breaths	Five	Five
Chest compressions		
Landmark	Lower half of sternum	Lower half of sternum
Technique	Two-thumb or two-finger	One-hand or two-hand
CPR ratio	15:2	15:2

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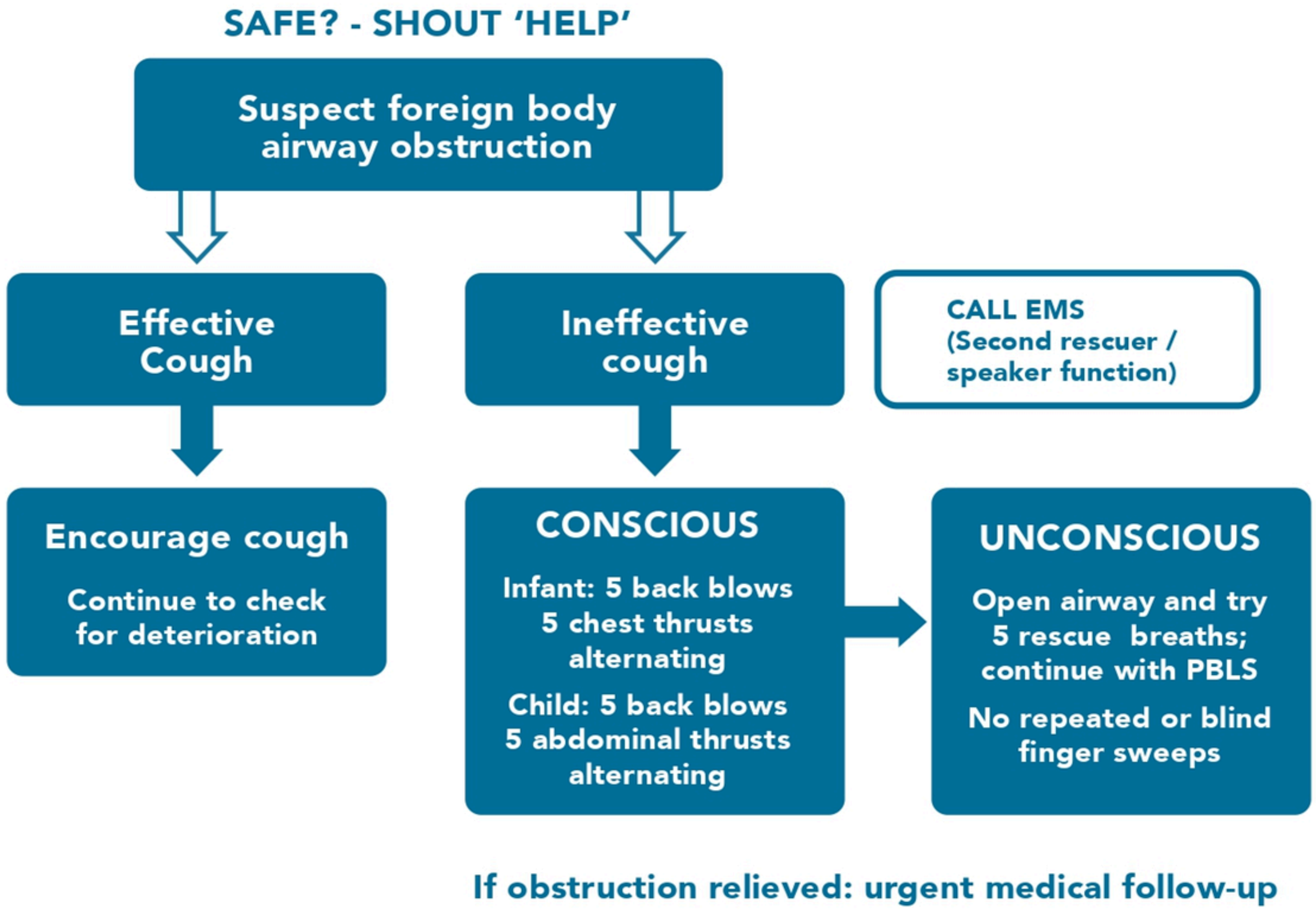
PAEDIATRIC FOREIGN BODY AIRWAY OBSTRUCTION

EUROPEAN RESUSCITATION COUNCIL
GUIDELINES 2021



Van De Voorde P, Turner NM, Djakow J, De Lucas N, Martinez-Mejias A, Biarent D, et al. European Resuscitation Council Guidelines 2021: Paediatric Life Support. Resuscitation 2021;161:327–87. https://doi.org/10.1016/j.resuscitation.2021.02.015.

Foreign body airway obstruction: infants

Abdominal thrusts may cause intra-abdominal injury in infants. A combination of back blows and chest thrusts is recommended for the relief of foreign body airway obstruction in this age group. The baby is placed along one of the rescuer's arms in a head-down position, with the rescuer's hand supporting the infant's jaw in such a way as to keep it open in the neutral position. The rescuer then rests their arm along their own thigh and delivers five back blows between the infant's shoulder blades with the heel of the free hand. If the obstruction is not relieved, the baby is turned over and laid along the rescuer's thigh, still in a head-down position.

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Foreign body airway obstruction: infants

Five chest thrusts are given – using the same landmarks as for cardiac compression **but at a slower rate of 1 per second and sharper than chest compressions.** If an infant is too large to allow the use of the single-arm technique described, then the same maneuvers can be performed by laying the baby across the rescuer's lap.

In a large infant, the heel of the hand can be used if the two-finger technique is not effective or deemed too difficult to do.

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Foreign body airway obstruction: children

In the child, the abdominal thrust (Heimlich maneuver) is recommended instead of chest thrusts. This can be performed with the victim either standing or lying on the ground, but the former is usually more appropriate. If abdominal thrusts are to be attempted with the child standing, the rescuer moves behind the victim and passes their arms around the victim's body.

Owing to the smaller stature of children, it may be necessary for an adult to raise the child or kneel behind them to carry out the standing Manoeuvre effectively. One hand is formed into a fist and placed against the child's abdomen above the umbilicus and below the xiphisternum. The other hand is placed over the fist, and both hands are thrust sharply upwards into the abdomen. This is repeated five times unless the object causing the obstruction is expelled before then.

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Foreign body airway obstruction: children

To carry out the abdominal thrust in a supine child, the rescuer kneels at the child's feet. If the child is large, it may be necessary to kneel astride the child. The heel of one hand is placed against the child's abdomen above the umbilicus and below the xiphisternum. The other hand is placed on top of the first, and both hands are thrust sharply upwards into the abdomen, with care being taken to direct the thrust in the midline.

This is repeated five times unless the object causing the obstruction is expelled before that. When performing the abdominal thrust, make sure that the pressure is not applied to the xiphisternum or the lower rib cage to avoid trauma.

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