

Approccio al Primo Soccorso

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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

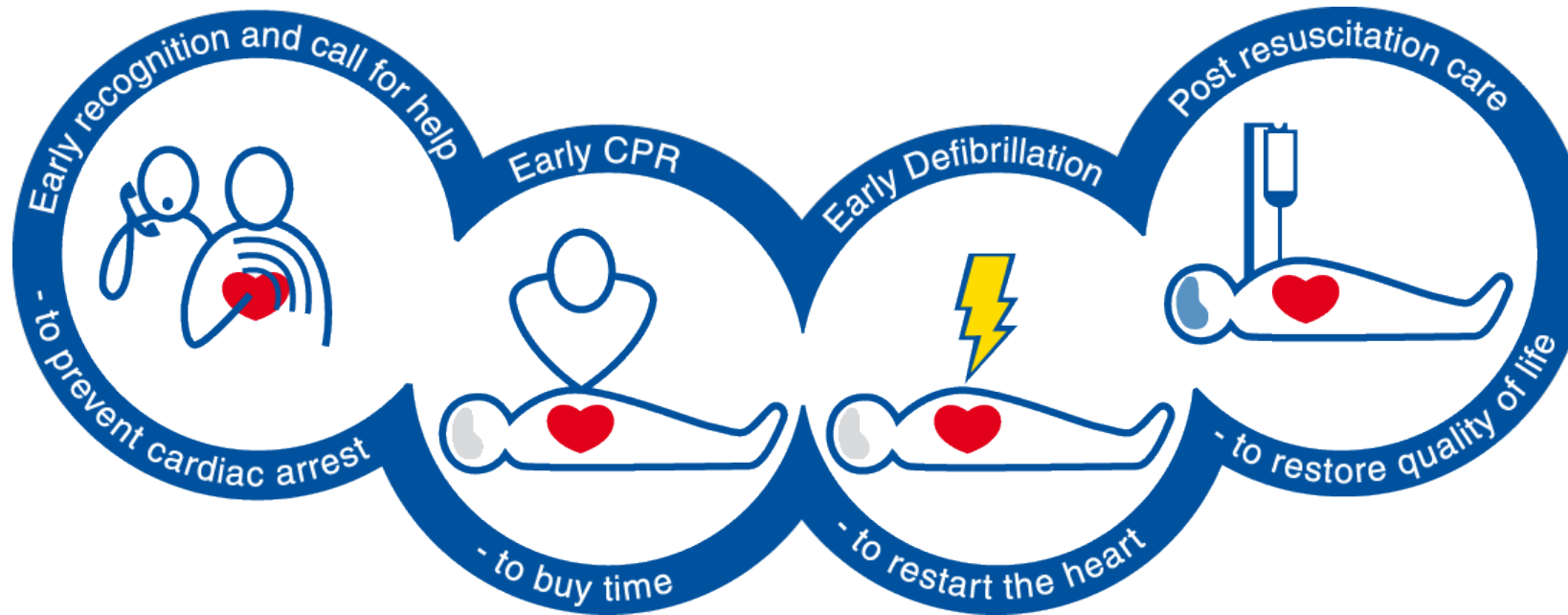


Background

- 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



Chain of survival





Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths





Approach safely

Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths



Check response



©ERC

Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

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.



Shout for help



Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths



Shout for help

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, **DO NOT** leave the victim, but start CPR



©ERC



Open airway



©ERC

Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths

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Check breathing



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Approach safely

Check response

Shout for help

Open airway

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Call 112

30 chest compressions

2 rescue breaths

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Check breathing

- Look, listen and feel for NORMAL breathing





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.



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



CAROTID PULSE CHECK

- Pulse absent or **unsure**
- Start chest compressions
- Continue 30 chest compressions : 2 rescue breaths
- Do not re-check for carotid pulse or signs of life
- Pulse definitely **present**
- 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



Call 112



Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths



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





30 Chest compressions



Approach safely

Check response

Shout for help

Open airway

Check breathing

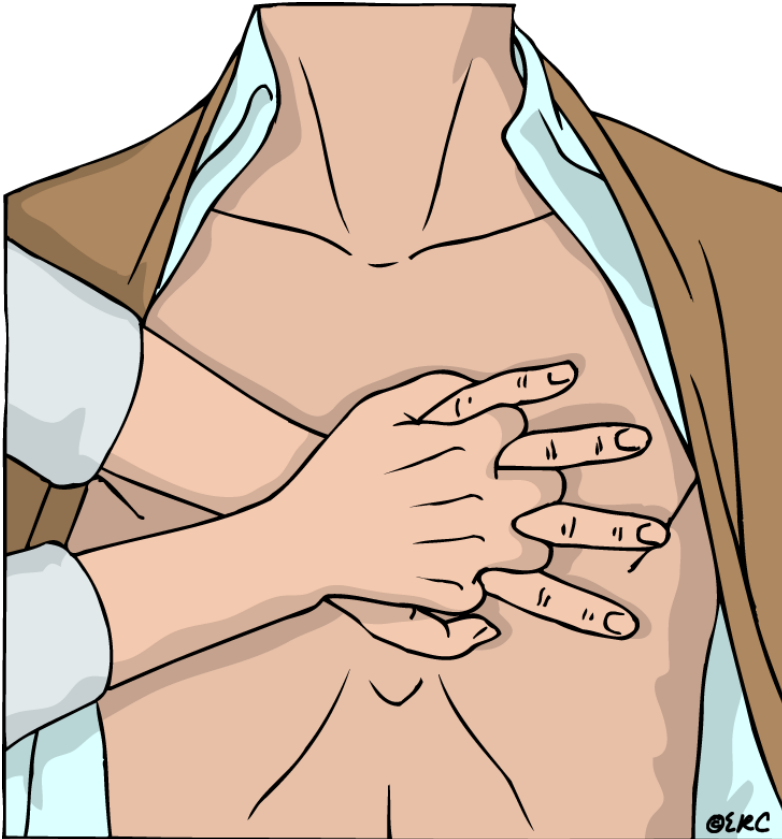
Call 112

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

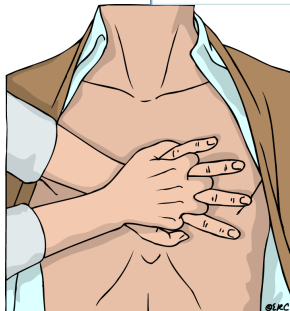


Chest compressions

CIRCULATION
Start chest compressions

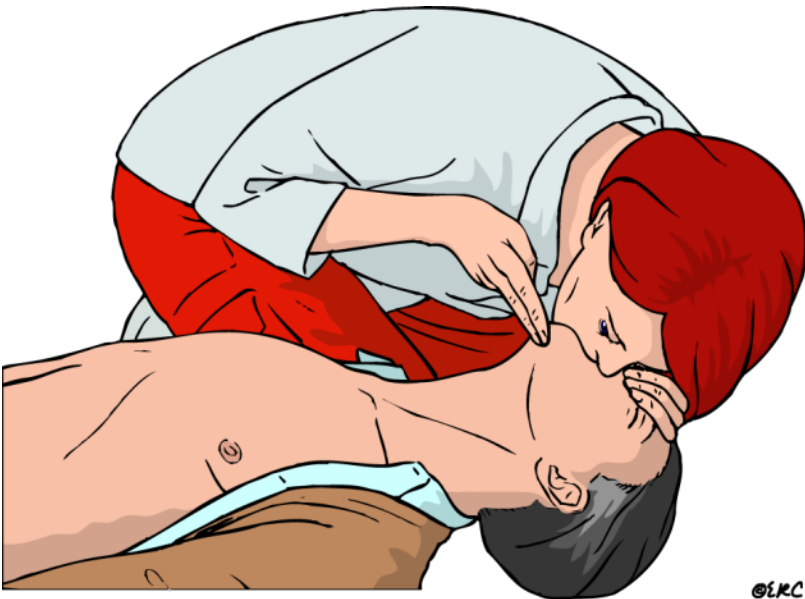


- Kneel by the side of the victim
- Place the heel of one hand in the centre of the victim's chest - this is the lower half of the victim's breastbone (sternum)
- Place the heel of your other hand on top of the first hand and interlock your fingers
- Keep your arms straight
- Position yourself vertically 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



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Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

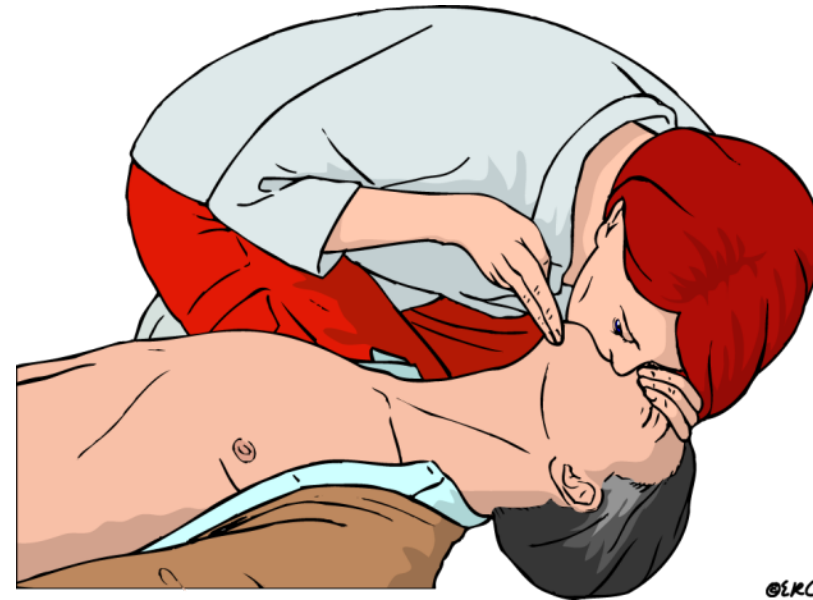
2 rescue breaths

©ERC



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



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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



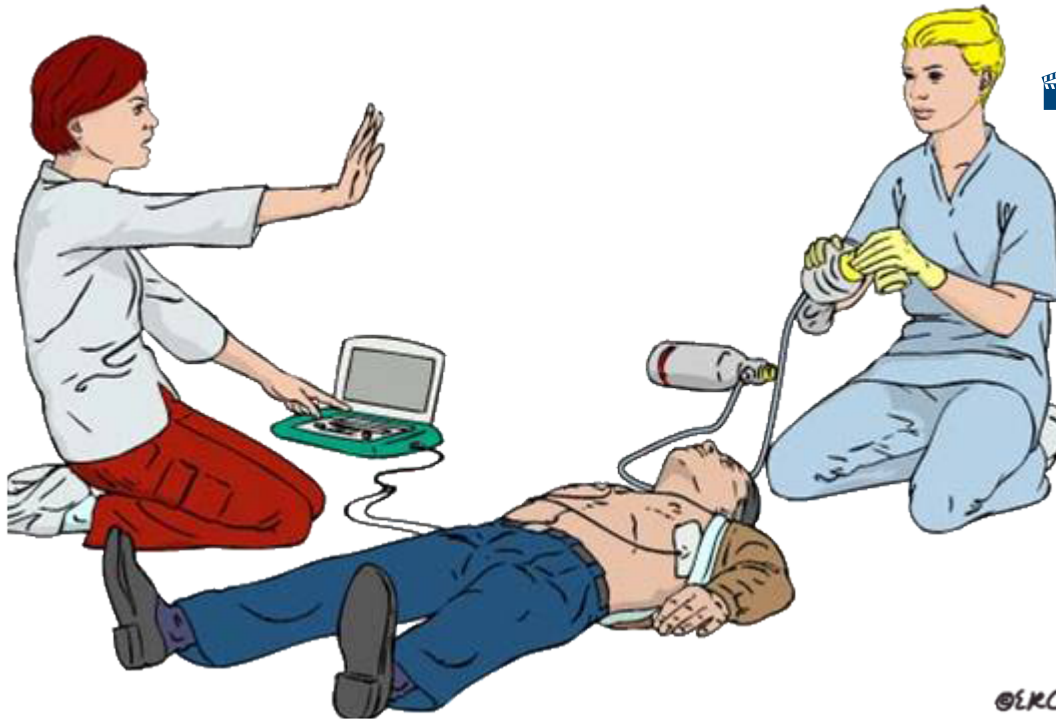
USE OF OXYGEN



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



USE OF OXYGEN



**Removal of oxygen from victim during
defibrillation**

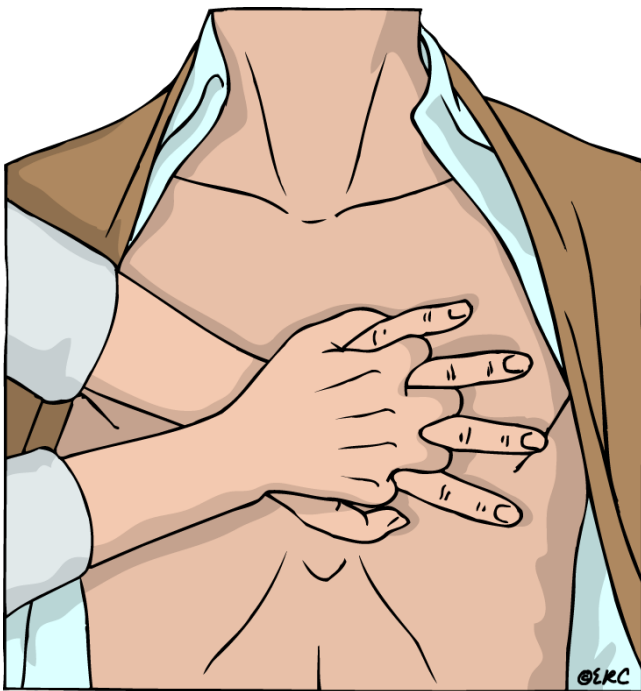


Take care with
automated external
defibrillators:

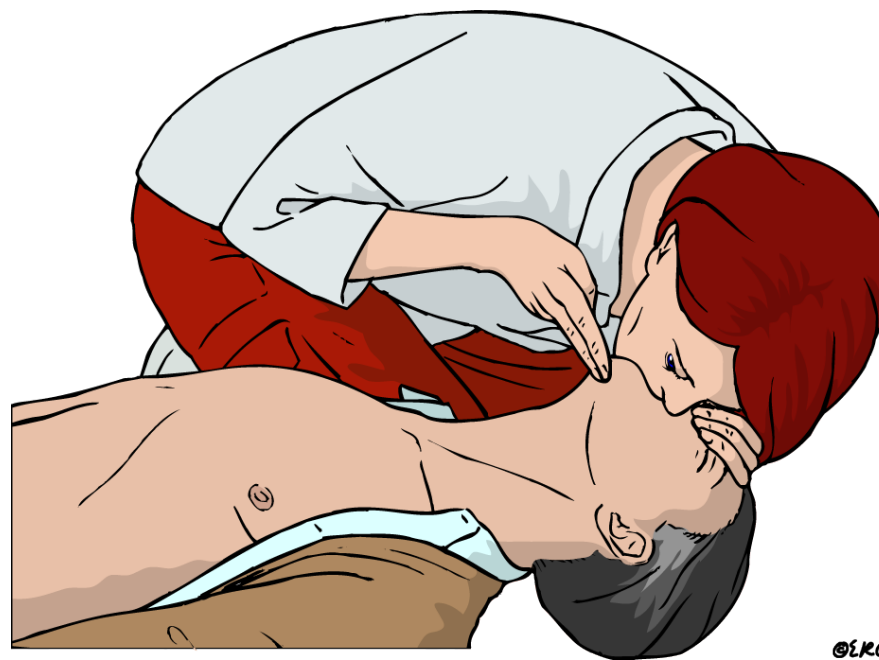
- ~ Stick pads down firmly
- ~ Remove oxygen
source at least 1 metre
from victim's chest
during shocks



Continue CPR



30



2



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



Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

30 chest compressions

2 rescue breaths

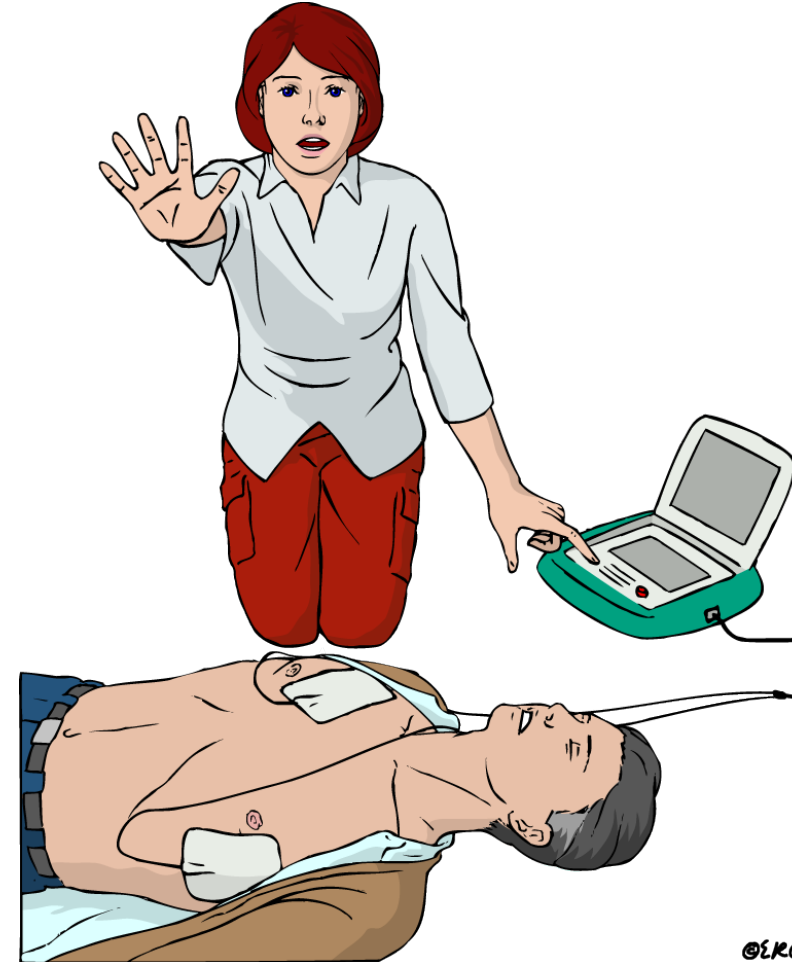
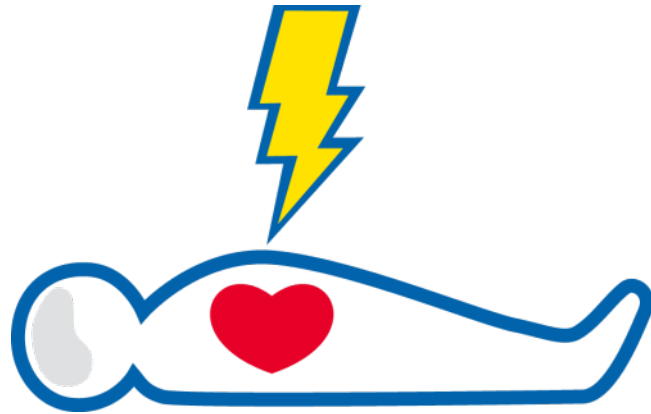




Any
questions?



Defibrillation





Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

Attach AED

**Follow voice
prompts**



Switch on AED

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

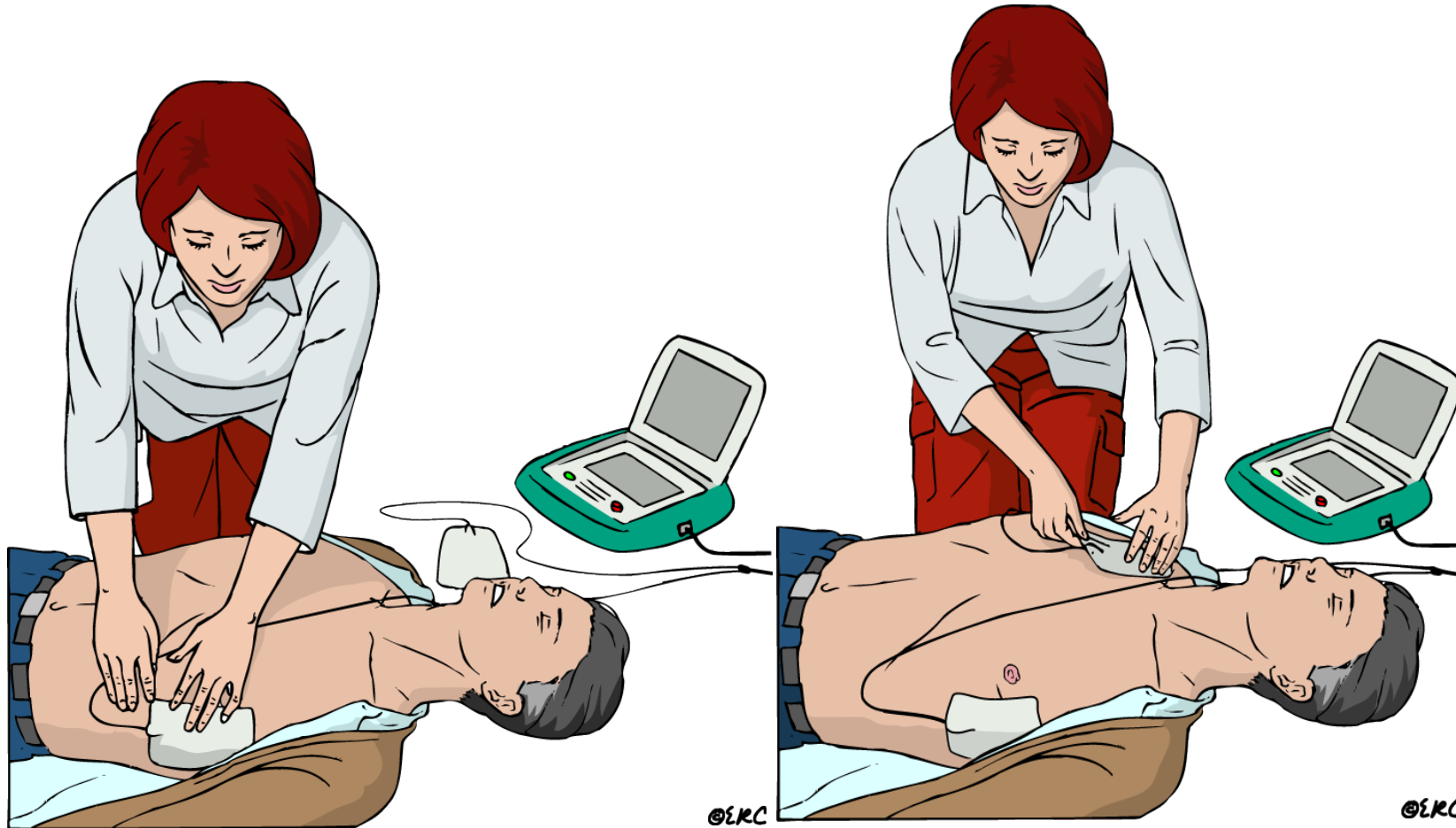


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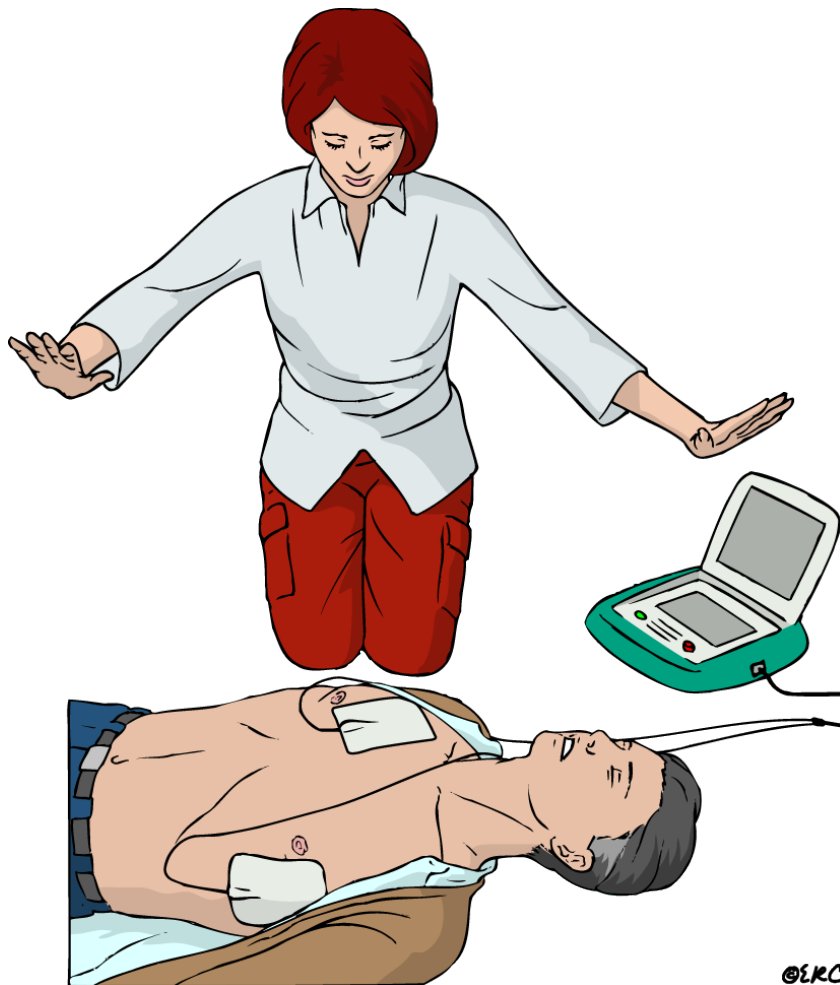


Attach pads to victims bare chest





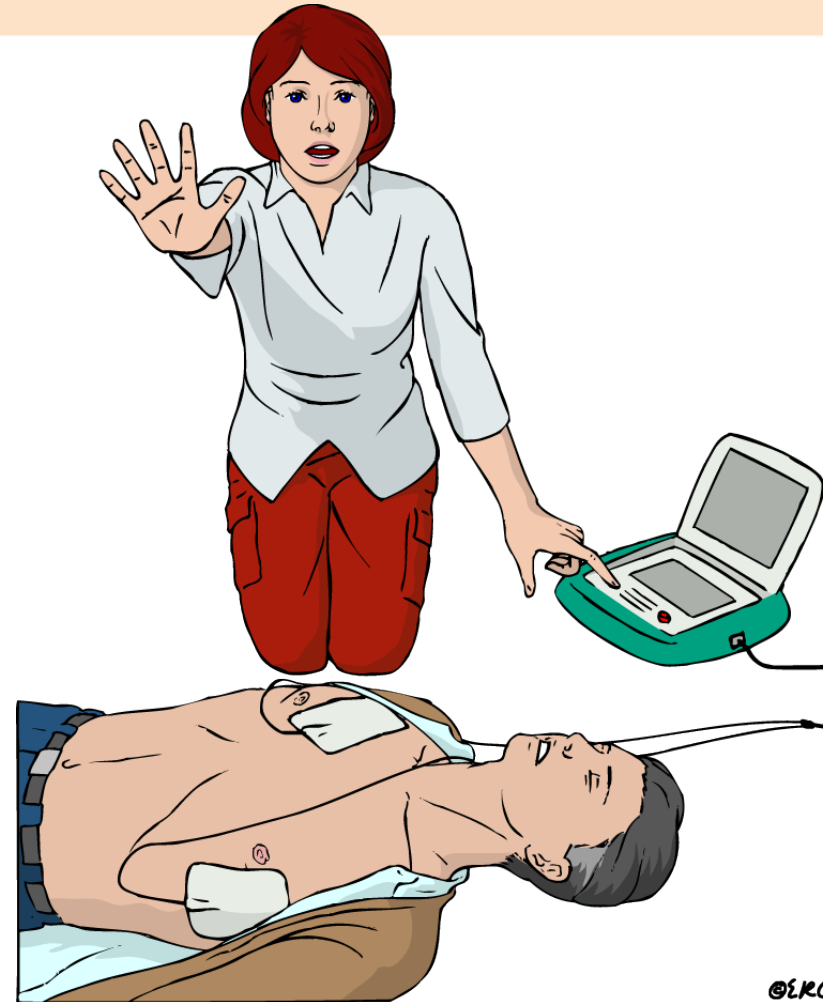
Analysing Rhythm: Do not touch victim





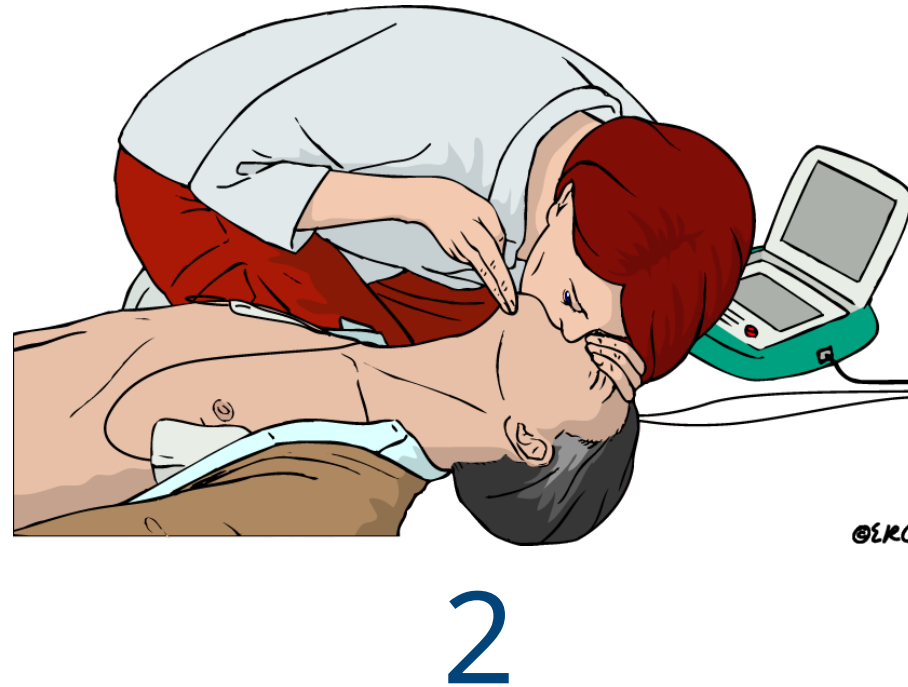
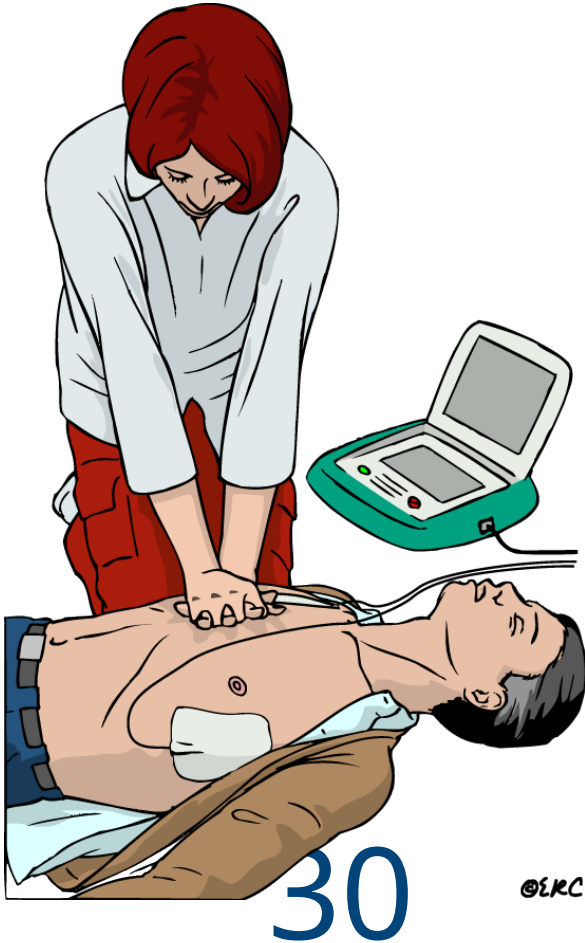
Shock indicated

- Stand clear
- Deliver shock



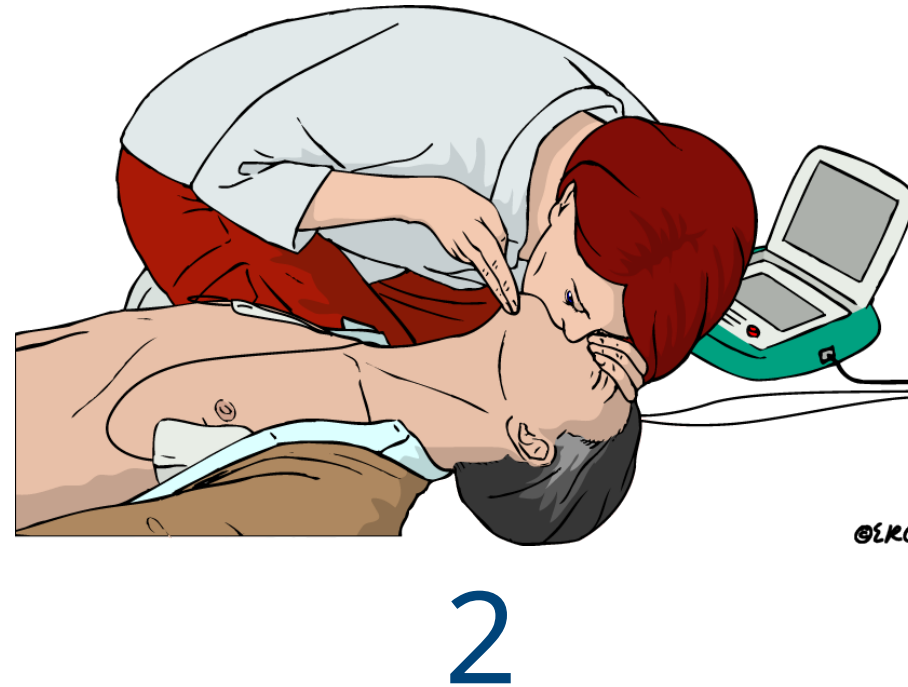
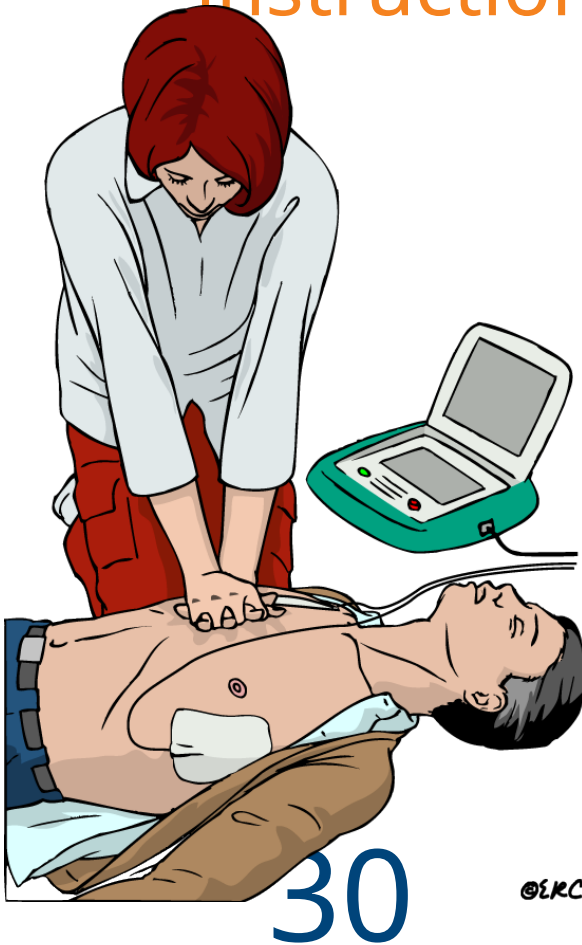


Shock delivered: Follow AED instructions





No shock advised: Follow AED instructions

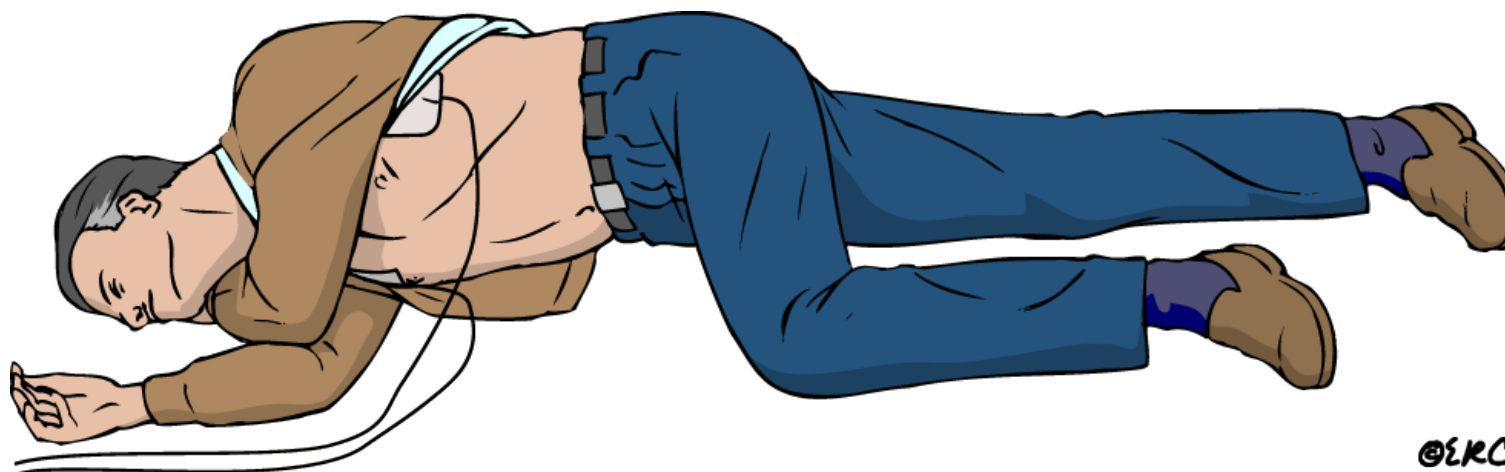




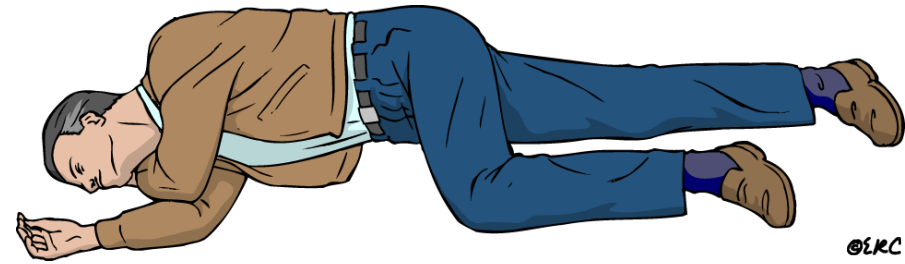
Any
questions?



If victim starts to breathe normally place
in recovery position



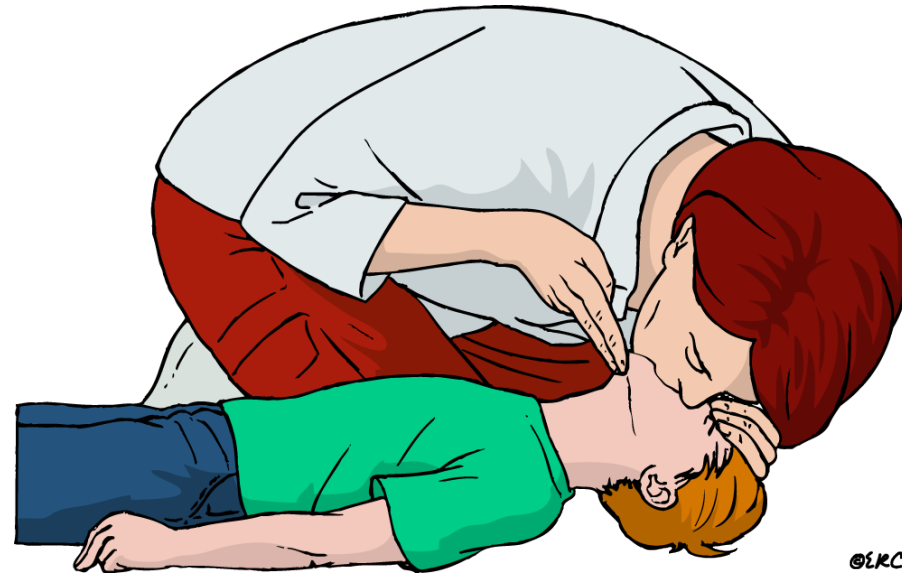
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CPR in children

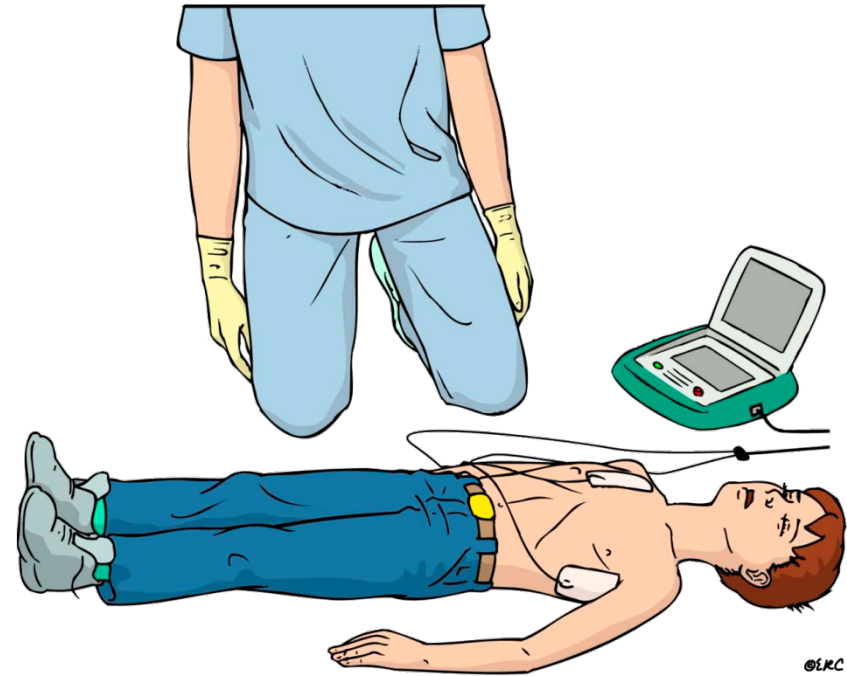
- Adult CPR techniques can be used on children
- Compressions at least $\frac{1}{3}$ of the depth of the chest





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





Any
questions?



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



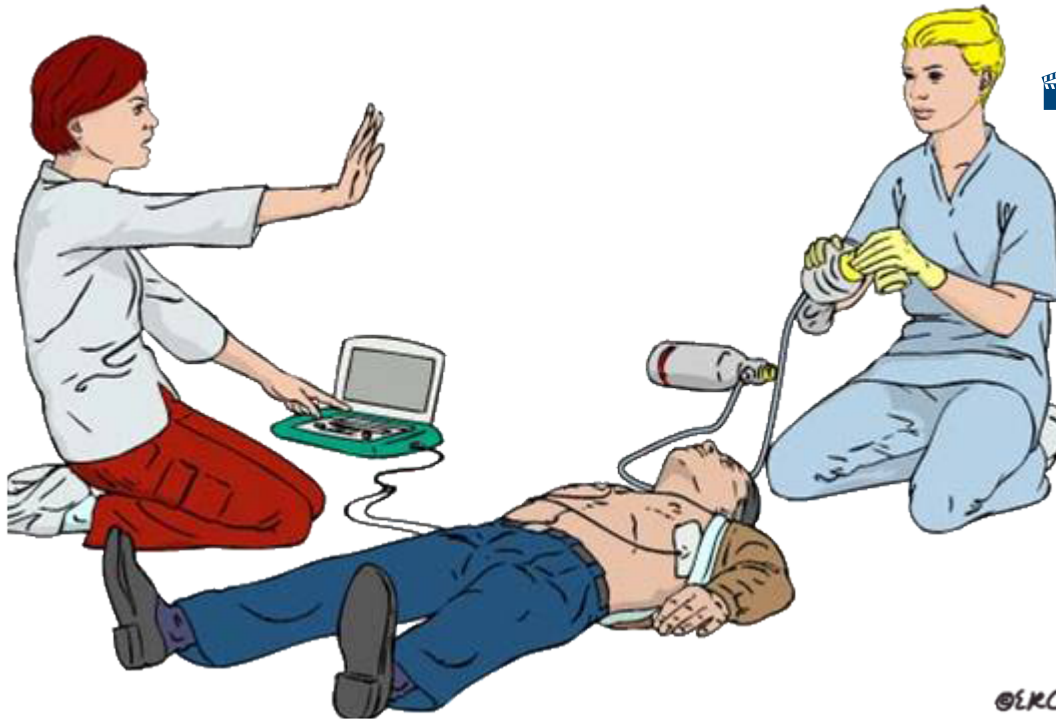
USE OF OXYGEN



- Supplemental oxygen can be used in cardiac arrest if available
- Given via a bag-valve mask or pocket-mask
- Give as much as possible as soon 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:

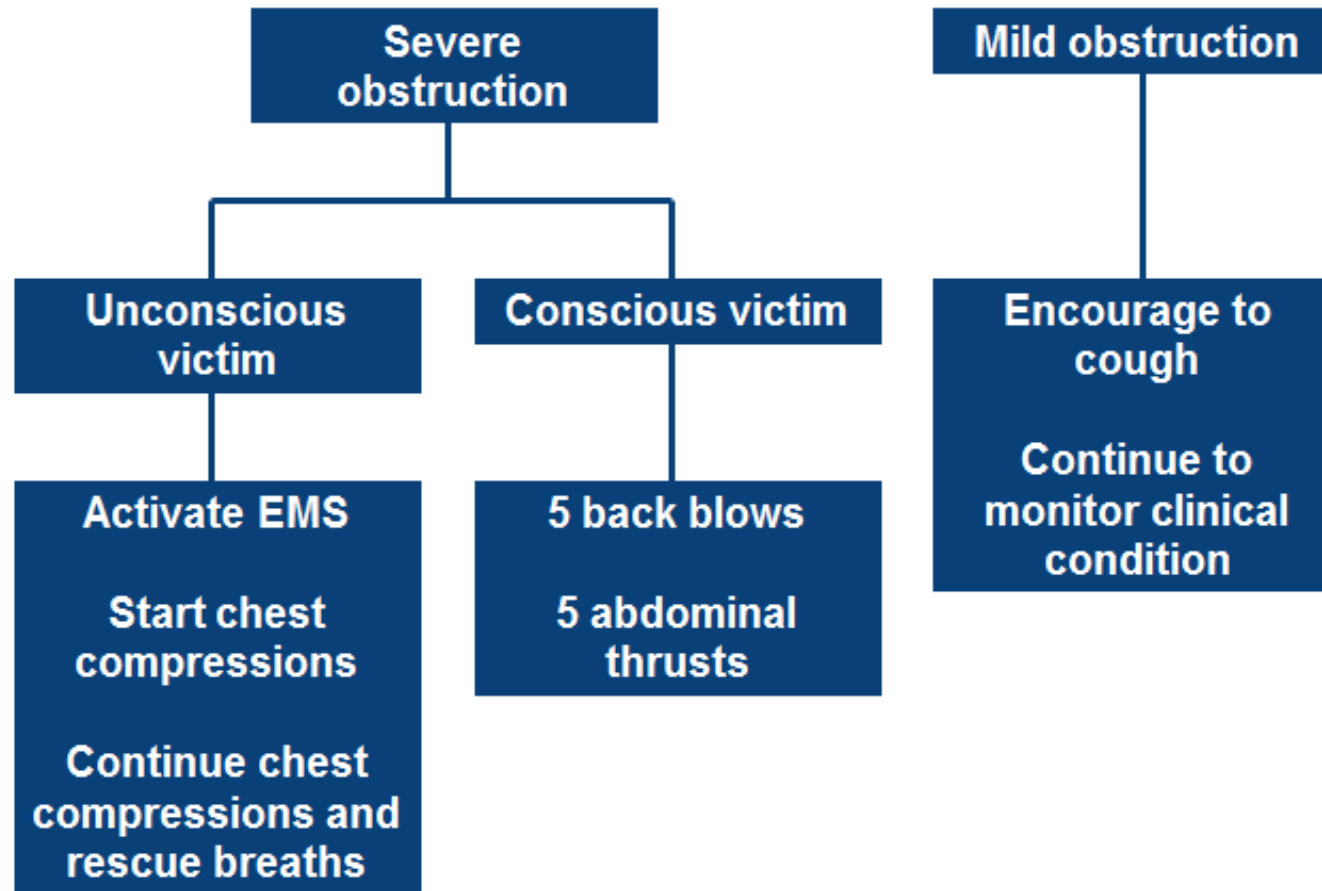
- ~ One rescuer operates AED and attaches pads
- ~ Other rescuer performs CPR
- ~ Only interrupt CPR to analyse and to deliver shock



Any
questions?



CHOKING: ALGORITHM





CHOKING

Back Blows



Abdominal thrusts: position of first hand



Abdominal thrusts: position of second hand





Any
questions?



Approach safely

Check response

Shout for help

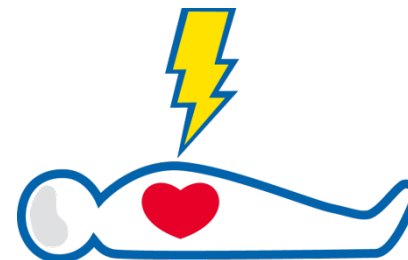
Open airway

Check breathing

Call 112

30 chest

**compressions
2 rescue breaths**



Approach safely

Check response

Shout for help

Open airway

Check breathing

Call 112

Attach AED

**Follow voice
prompts**

Arresto cardiocircolatorio (CA)

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

Cenni di emodinamica

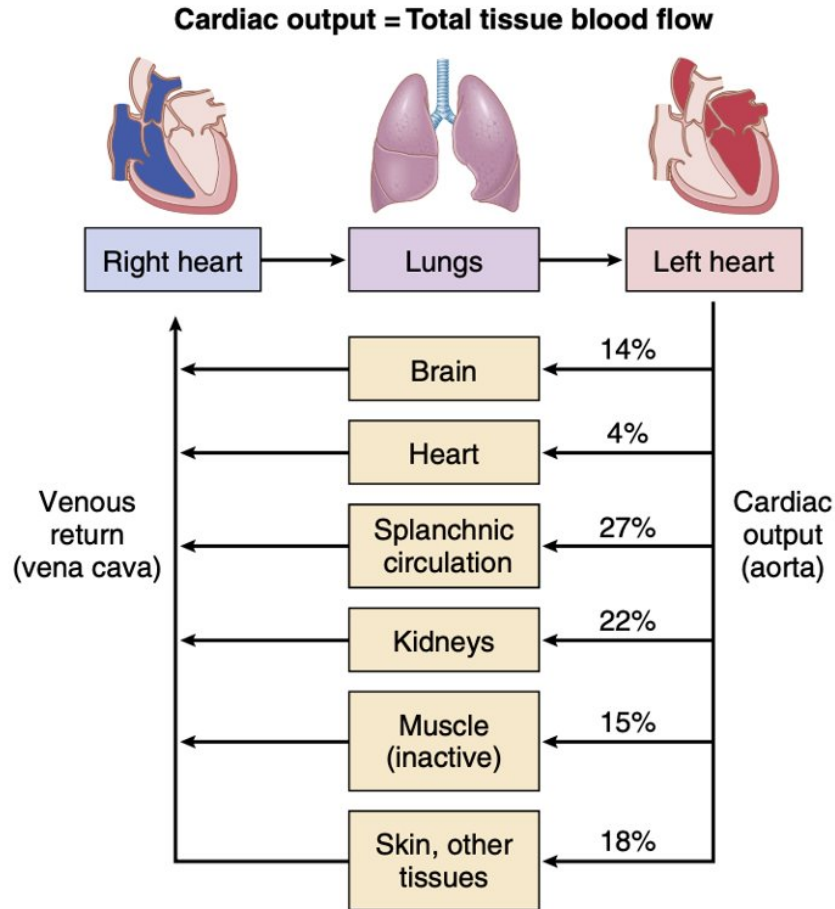


Figure 20-2. Cardiac output is equal to venous return and is the sum of tissue and organ blood flows. Except when the heart is severely weakened and unable to pump the venous return adequately, cardiac output (total tissue blood flow) is determined mainly by the metabolic needs of the tissues and organs of the body.

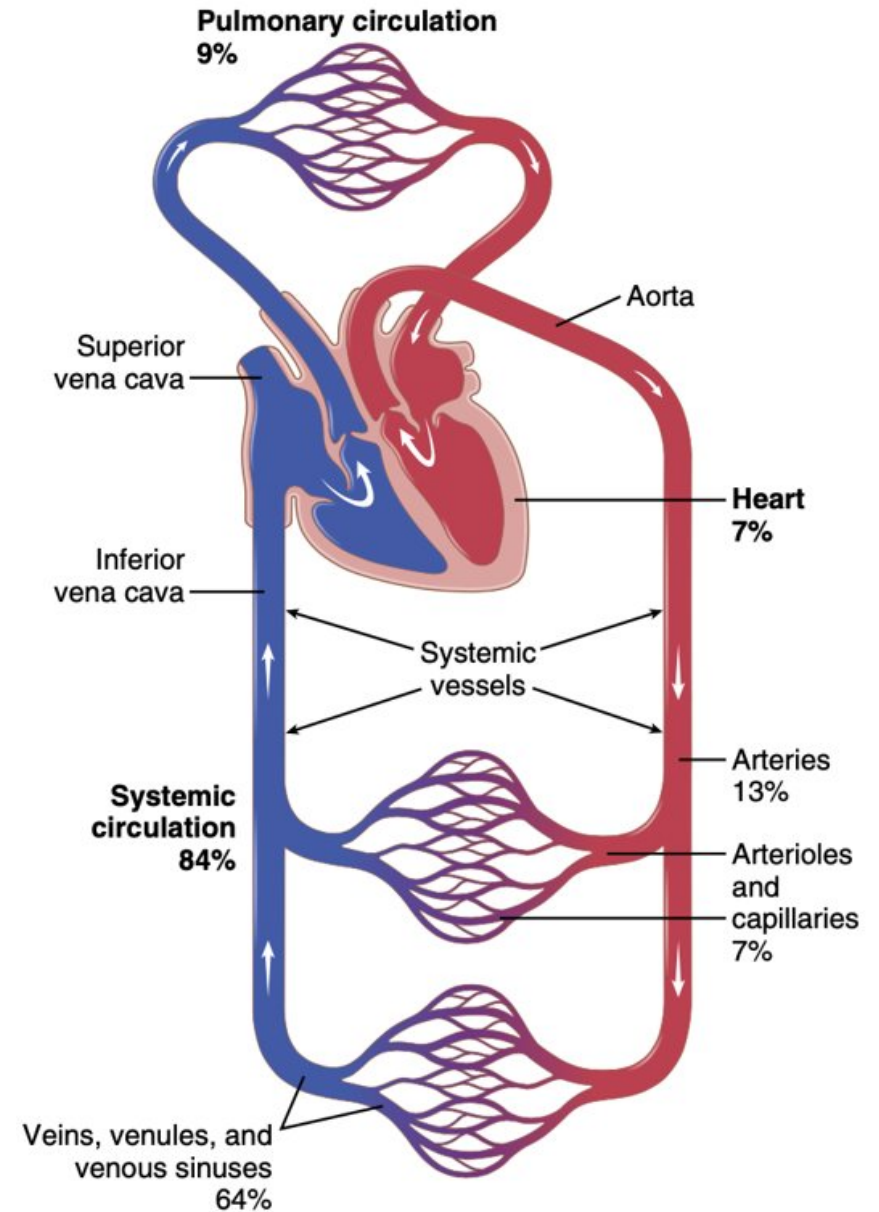


Figure 14-1. Distribution of blood (in percentage of total blood) in the different parts of the circulatory system.

Table 1 Hemodynamic variables obtained from the pulmonary artery catheter

Variable	Abbreviation	Equation	Normal range
Mixed venous oxygen saturation	SvO ₂	n.a	60–80%
Cardiac output	CO	$HR \times SV / 1000$	4.0–8.0 L min ⁻¹
Cardiac index	CI	CO/BSA	2.5–4.0 L min ⁻¹ m ⁻²
Cardiac power index	CPI	$(MAP - CVP) \times CI / 451$	0.5–0.7 W m ⁻² , population specific
Central venous Pressure	CVP	n.a	2–6 mmHg
Stroke volume	SV	$CO / HR \times 1000$	60–100 mL
Stroke volume Index	SVi	$CI / HR \times 1000$	33–47 mL m ⁻²
Stroke volume variation	SVV	$(SV_{max} - SV_{min}) / SV_{mean} \times 100$	10–15%
Systemic vascular resistance	SVR	$80 \times (MAP - CVP) / CO$	800–1200 dynes sec cm ⁻⁵
Systemic to pulmonary pressure ratio	MAP/MPAP	MAP / MPAP	4.0 ± 1.4 in uncomplicated cardiac surgery
Pulmonary artery systolic pressure	PASP	n.a	15–30 mmHg
Pulmonary artery diastolic pressure	PADP	n.a	8–15 mmHg
Pulmonary artery wedge pressue	PAWP	n.a	6–12 mmHg
Pulmonary vascular resistance	PVR	$80 \times (MPAP - PAWP) / CO$	< 250 dynes sec cm ⁻⁵
Pulmonary artery pulsatility index	PAPI	$(PASP - PADP) / CVP$	population specific
LV stroke work index	LVSWi	$SVi \times (MAP - PAWP) \times 0.0136$	50–62 mmHg ml m ⁻²
RV stroke work index	RVSWi	$SVi \times (MPAP - CVP) \times 0.0136$	5–10 mmHg ml m ⁻²
RV function index	RFI	PASP/CI	31.7 ± 16.7 in ICU survivors with PH
RV end-diastolic volume	RVEDV	SV/EF	100–160 mL
RV end-diastolic volume index	RVEDVi	RVEDV/BSA	60–100 mL m ⁻²
RV end-systolic volume	RVESV	EDV-SV	50–100 mL
RV ejection fraction	RVEF	$(SV / EDV) \times 100$	40–60%
RV systolic pressure	RVSP	n.a	15–30 mmHg
RV diastolic pressure	RVDP	n.a	2–8 mmHg

In-Hospital vs Out-of-Hospital CA



Epidemiologia di C

Arresto cardiaco 3° causa di morte in Europa

- The annual incidence of OHCA in Europe is between 67 to 170 per 100,000 inhabitants.
- Resuscitation is attempted or continued by EMS personnel in about 50–60% of cases (between 19 to 97 per 100,000 inhabitants).
- The rate of bystander CPR varies between and within countries (average 58%, range 13%–83%).
- The use of automated external defibrillators (AEDs) remains low in Europe (average 28%, range 3.8%–59%).
- 80% of European countries provide dispatch assisted CPR and 75% have an AED registry. Most (90%) countries have access to cardiac arrest centres for post resuscitation care.
- Survival rates at hospital discharge are on average 8%, varying from 0% to 18%.
- Differences in EMS systems in Europe account for at least some of the differences observed in OHCA incidence and survival rates.

In hospital cardiac arrest

- The annual incidence of IHCA in Europe is between 1.5 and 2.8 per 1,000 hospital admissions.
- Factors associated with survival are the initial rhythm, the place of arrest and the degree of monitoring at the time of collapse.
- Survival rates at 30 days / hospital discharge range from 15% to 34%.

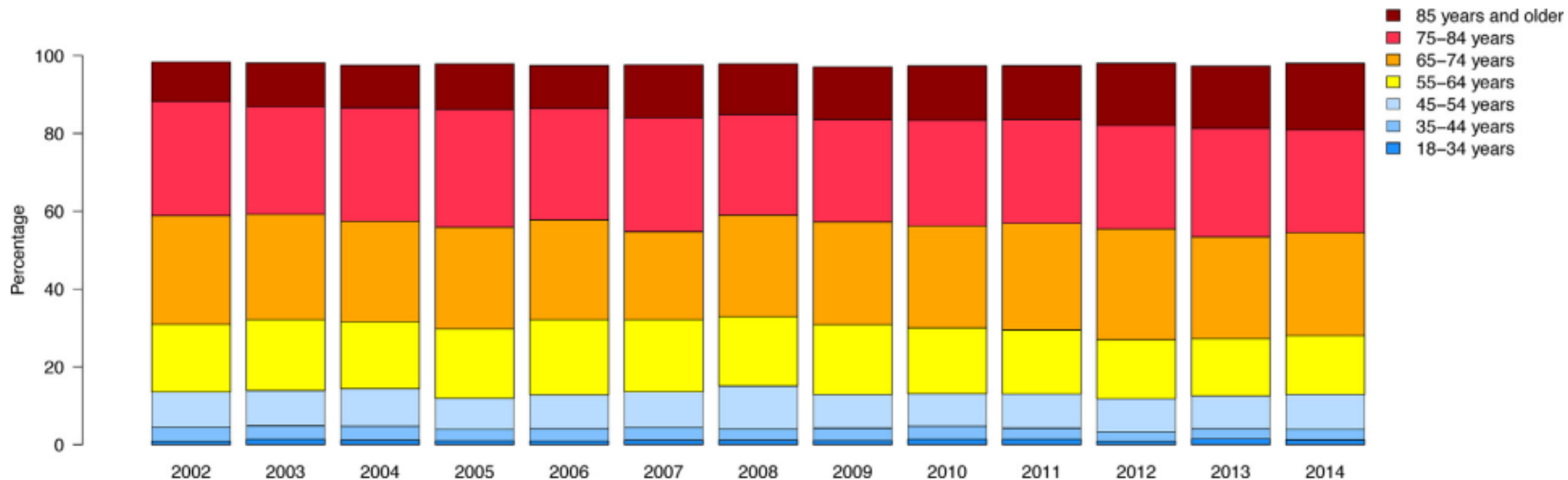


Fig. 1 – Distribution of age groups during the study period.

Prognosi di CA

Long term outcomes

- In European countries where withdrawal of life sustaining treatment (WLST) is routinely practiced, a good neurological outcome is seen in > 90% of patients. Most patients are able to return to work.
- In countries where WLST is not practiced, poor neurological outcomes are more common (50% with 33% in a persistent vegetative state).
- Amongst survivors with a good neurological outcome, neuro-cognitive, fatigue and emotional problems are common and cause reduced health related quality of life.
- Patients and relatives may develop post-traumatic stress disorder.

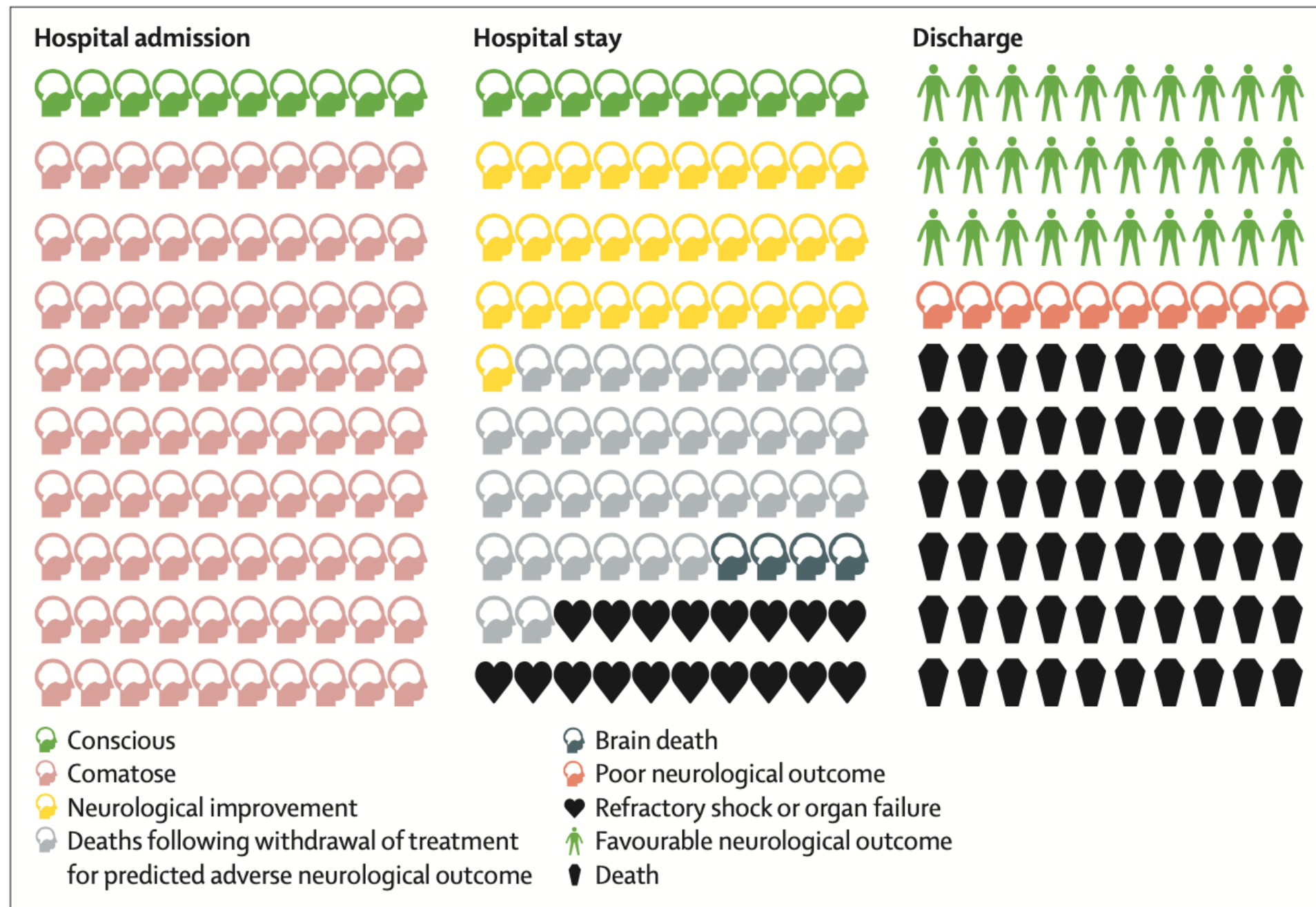


Figure 2: Outcomes following admission for out-of-hospital cardiac arrests

Cause of arrest ^c	360		115		245		<0.001	0
- Unknown/not documented	27	8%	3	3%	24	10%	0.024	
- ALTE/SIDS	54	15%	17	15%	37	15%	1.000	
- Airway obstruction	41	11%	7	6%	34	14%	0.047	
- Arrhythmia	47	13%	30	26%	17	7%	<0.001	
- Drowning	100	28%	45	39%	55	22%	0.002	
- Electrolyte abnormality	3	1%	0	0%	3	1%	0.554	
- Elevated ICP	10	3%	0	0%	10	4%	0.034	
- Hypotension/shock	30	8%	2	2%	28	11%	0.001	
- Ingestion/toxin	2	1%	2	2%	0	0%	0.103	
- Other respiratory failure	33	9%	8	7%	25	10%	0.336	
- Seizures	13	4%	1	1%	12	5%	0.043	

Table 2 – Timing and source of long-term neurological outcome.

	Deceased after discharge (n = 7)		Scored at hospital discharge (n = 23)		Scored at a regular hospital or clinic visit (n = 47)		Scored at cross-sectional follow-up (2013–2014) (n = 18)		Scored at prospective follow-up (2011 and onwards) (n = 46)	
	Pre-arrest	Post-arrest	Pre-arrest	Post-arrest	Pre-arrest	Post-arrest	Pre-arrest	Post-arrest	Pre-arrest	Post-arrest
PCPC score ^a										
1 – Normal	5	0	18	16	39	26	17	8	40	24
2 – Mild disability	1	0	3	5	3	6	0	6	1	11
3 – Moderate disability	1	0	2	2	3	7	1	3	5	8
4 – Severe disability	0	0	0	0	2	7	0	1	0	3
5 – Coma or vegetative state	0	0	0	0	0	1	0	0	0	0
6 – Brain death	0	7	0	0	0	0	0	0	0	0
FSS score ^b	NA	NA	NA	6.0 [6.0–6.0]	NA	6.0 [6.0–11.0]	NA	6.0 [6.0–6.3]	NA	6.0 [6.0–6.3]
Follow-up (years) ^b	NA	0.6 [0.5–1.7]	NA	0.0 [0.0–0.0]	NA	2.7 [0.8–5.5]	NA	3.7 [2.5–10.5]	NA	2.3 [1.1–3.8]
Age at follow-up (years) ^b	NA	NA	NA	4.2 [1.5–8.9]	NA	6.6 [2.6–12.1]	NA	12.6 [3.8–15.0]	NA	9.0 [4.6–16.0]

Abbreviations: FSS = Functional Status Scale, PCPC = Pediatric Cerebral Performance Category.

^a Number of subjects.

^b Median (interquartile range). For patients deceased after discharge follow-up duration represents the median duration to date of death.

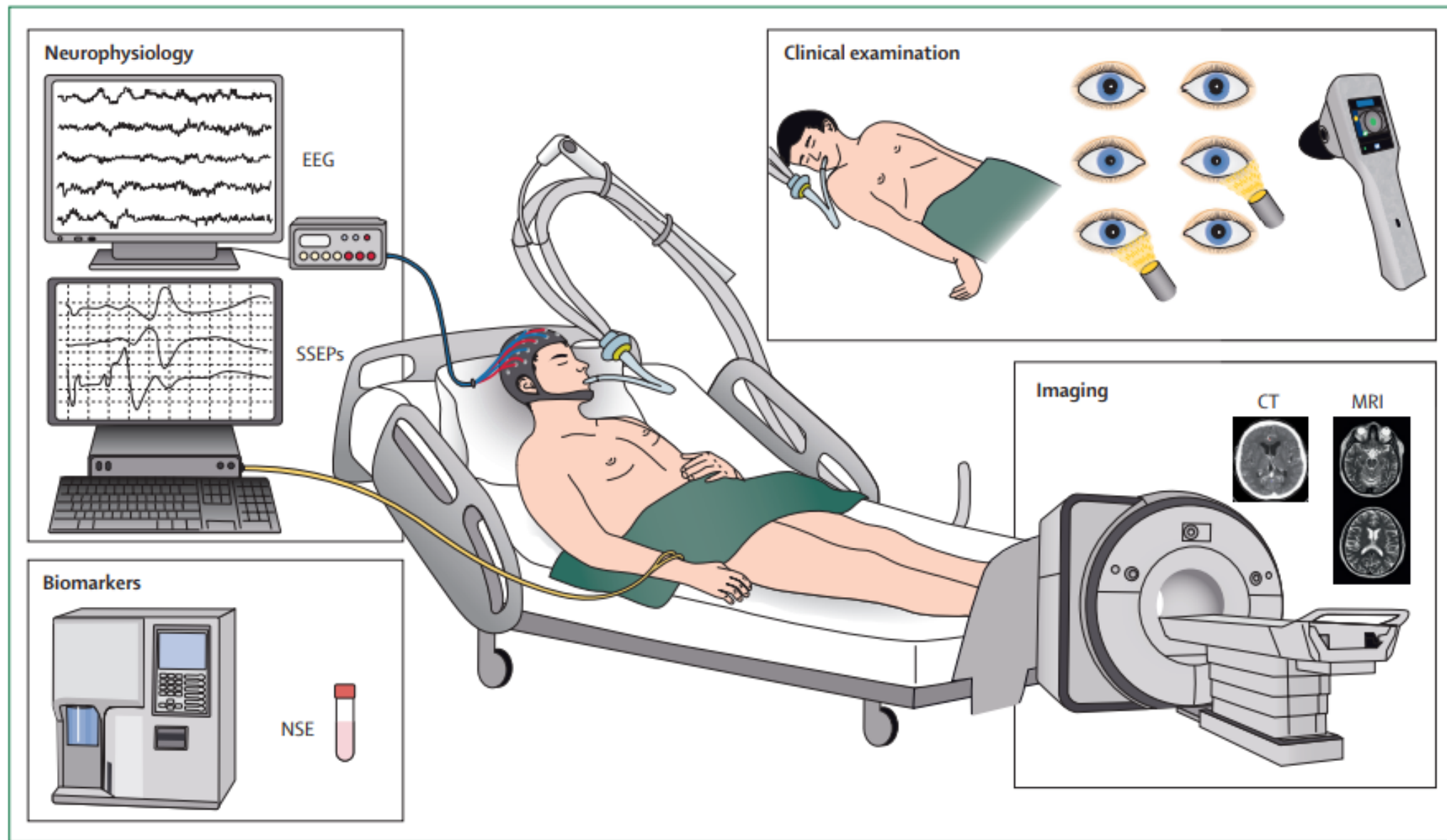


Figure 4: Key tests used to assess prognosis after cardiac arrest.

Reproduced from Nolan and colleagues,²³ by permission of the European Resuscitation Council. EEG=electroencephalogram. SSEPs=somatosensory evoked potentials. NSE=neuron specific enolase.

Cerebral Performance Category

-
- | | |
|----------------------|--|
| ○ 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. |
| ○ Death at discharge | |
-

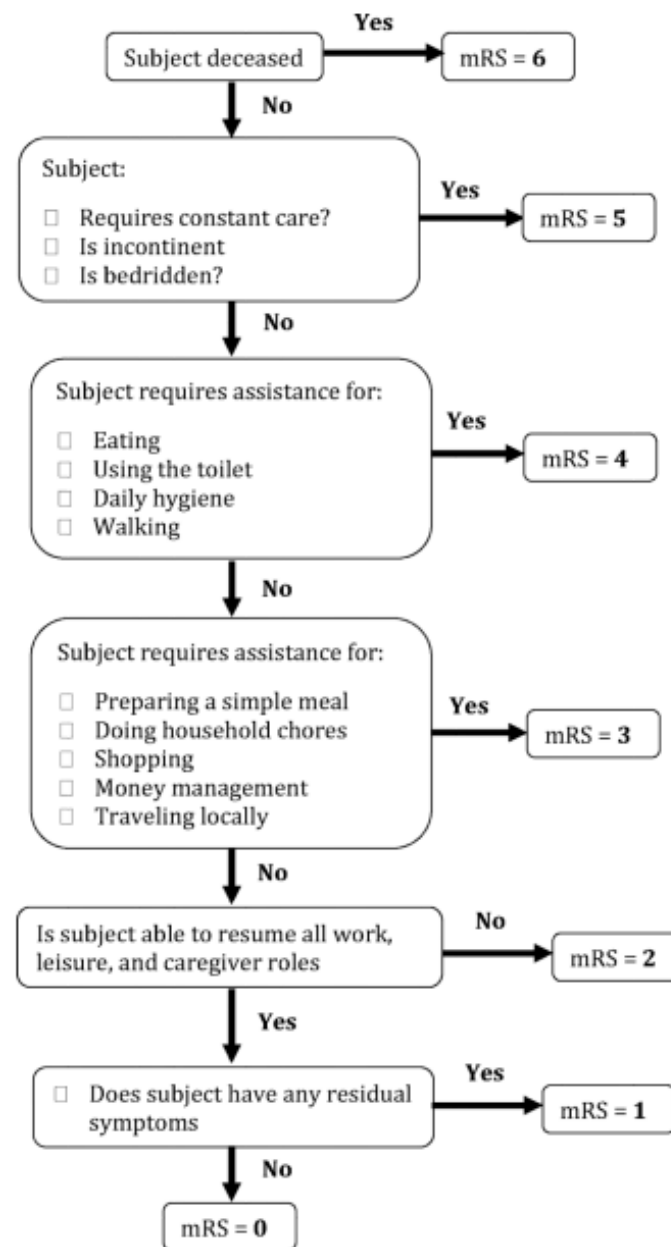


Figure 1.
Classification of the Modified Rankin Scale
Abbreviations: mRS = Modified Rankin Scale

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

Table 1 – Demographic characteristics of patients with OHCA of any aetiology who survived to hospital discharge in Perth WA between 2004 and 2019, stratified by Cerebral Performance Category (CPC) score.

	Total	CPC 1–2	CPC 3–4	Test for difference (p)
No. (%) of patients	1062 (100)	984 (92.7)	78 (7.3)	
Aetiology of arrest, presumed cardiac	965 (90.9)	899 (93.2)	66 (84.6)	0.05
Median (IQR) age, years	60 (49–71)	60 (49–71)	60 (45–78)	0.88
Sex				
Male	803 (75.6)	747 (75.9)	56 (71.8)	0.42
Female	259 (24.4)	237 (24.1)	22 (28.2)	
Location of arrest, public	395 (37.2)	379 (38.5)	16 (20.5)	0.002
Witnessed arrest				
Paramedic	305 (28.7)	292 (29.7)	13 (16.7)	0.01
Bystander	556 (52.4)	514 (52.2)	42 (53.8)	
Unwitnessed	201 (18.9)	178 (18.1)	23 (29.5)	
Early CPR ^a	910 (85.7)	855 (86.9)	55 (70.5)	<0.001
Initial arrest rhythm				
VF/VT	809 (76.2)	768 (78.0)	41 (52.6)	<0.001
PEA/Asystole ^b	253 (23.8)	216 (22.0)	37 (47.4)	
ROSC on arrival to first ED	978 (92.1)	910 (92.5)	68 (87.2)	0.10
Median (IQR) EMS response time, minutes ^c	8 (6–10)	8 (6–11)	7 (5–9)	<0.001
Survived to 12 months ^d	933 (92.3)	879 (89.3)	54 (69.2)	<0.001

CPR, cardiopulmonary resuscitation; EMS, emergency medical services; IQR, interquartile range; PEA, pulseless electrical activity; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia.

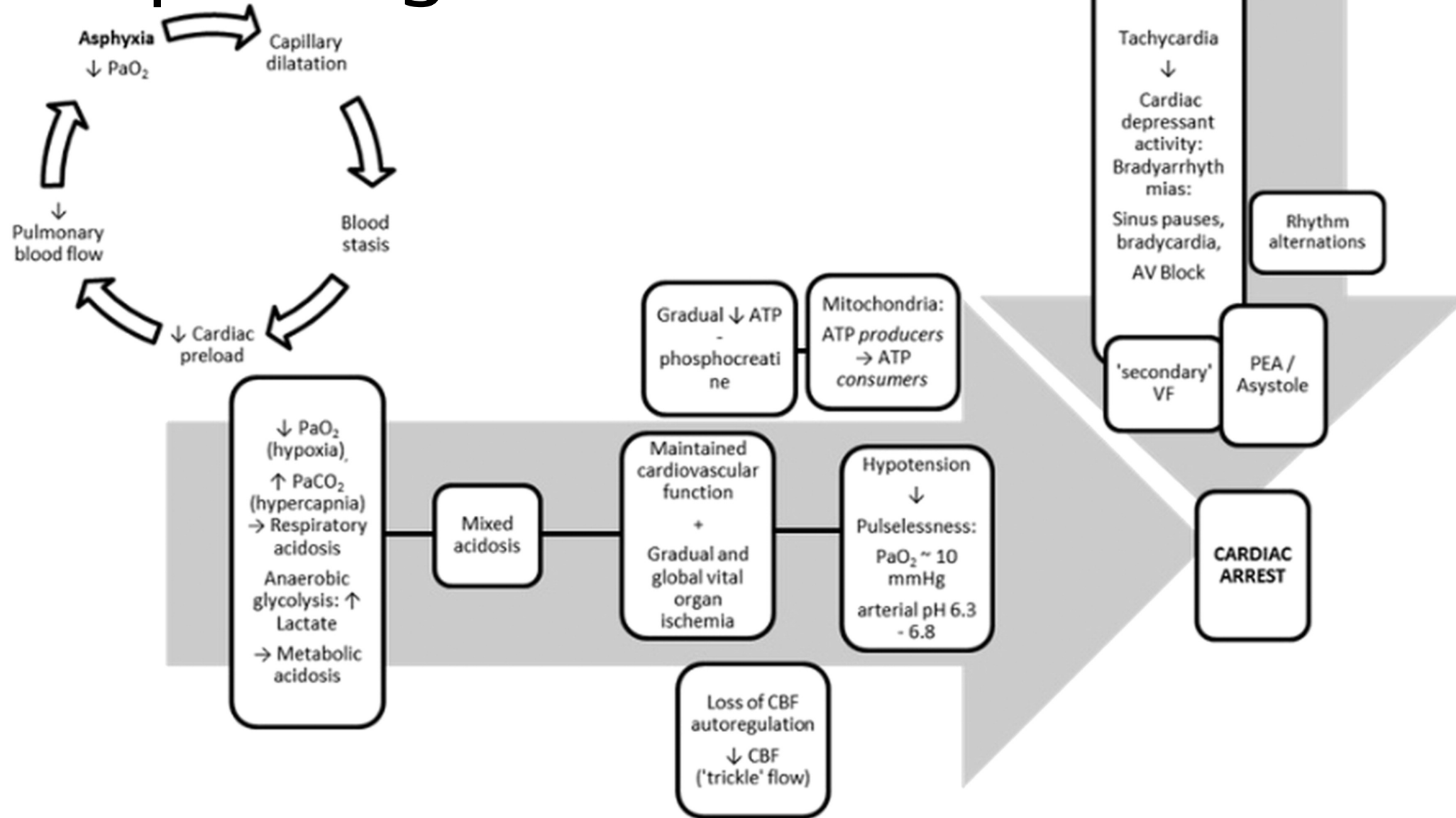
^a Includes 'bystander CPR' and 'paramedic CPR' for paramedic-witnessed arrests.

^b Includes 18 patients where 'initial arrest rhythm' is unknown.

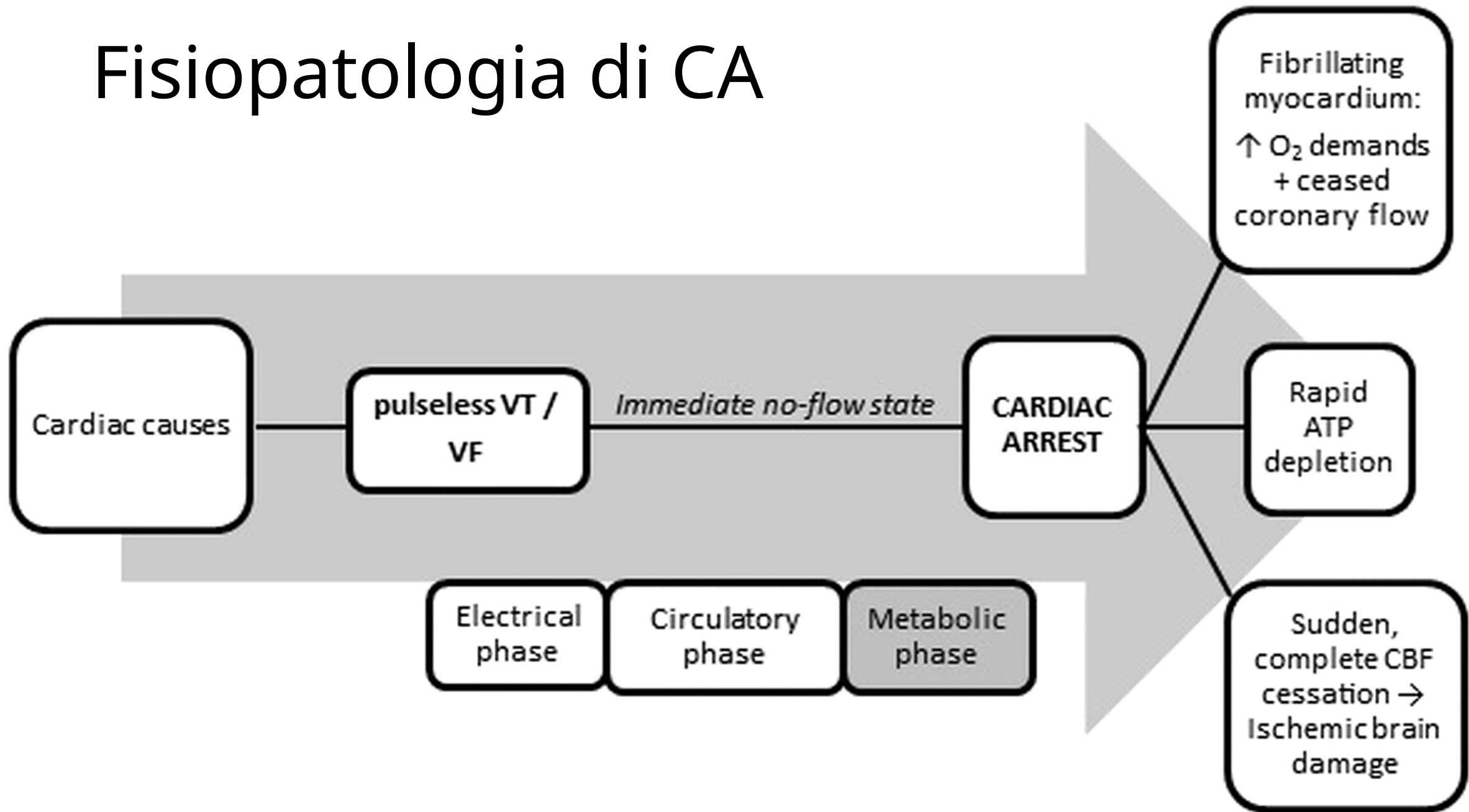
^c Using time interval from EMS call to arrival on scene.

^d Denominator is 1,011 WA residents who survived to hospital discharge.

Fisiopatologia di CA



Fisiopatologia di CA



Danno cerebrale da CA

Panel 2: Mechanisms associated with brain injury after cardiac arrest

Primary injury mechanisms

- Impaired oxygen and substrate delivery
- Excitotoxicity
- Disrupted calcium homoeostasis
- Oxidative stress
- Mitochondrial damage and dysfunction
- Pathological protease activation
- Inflammation

Secondary injury mechanisms

- Hypotension
- Hypoxaemia
- Elevated intracranial pressure
- Seizures
- Dysglycaemia
- Hyperthermia

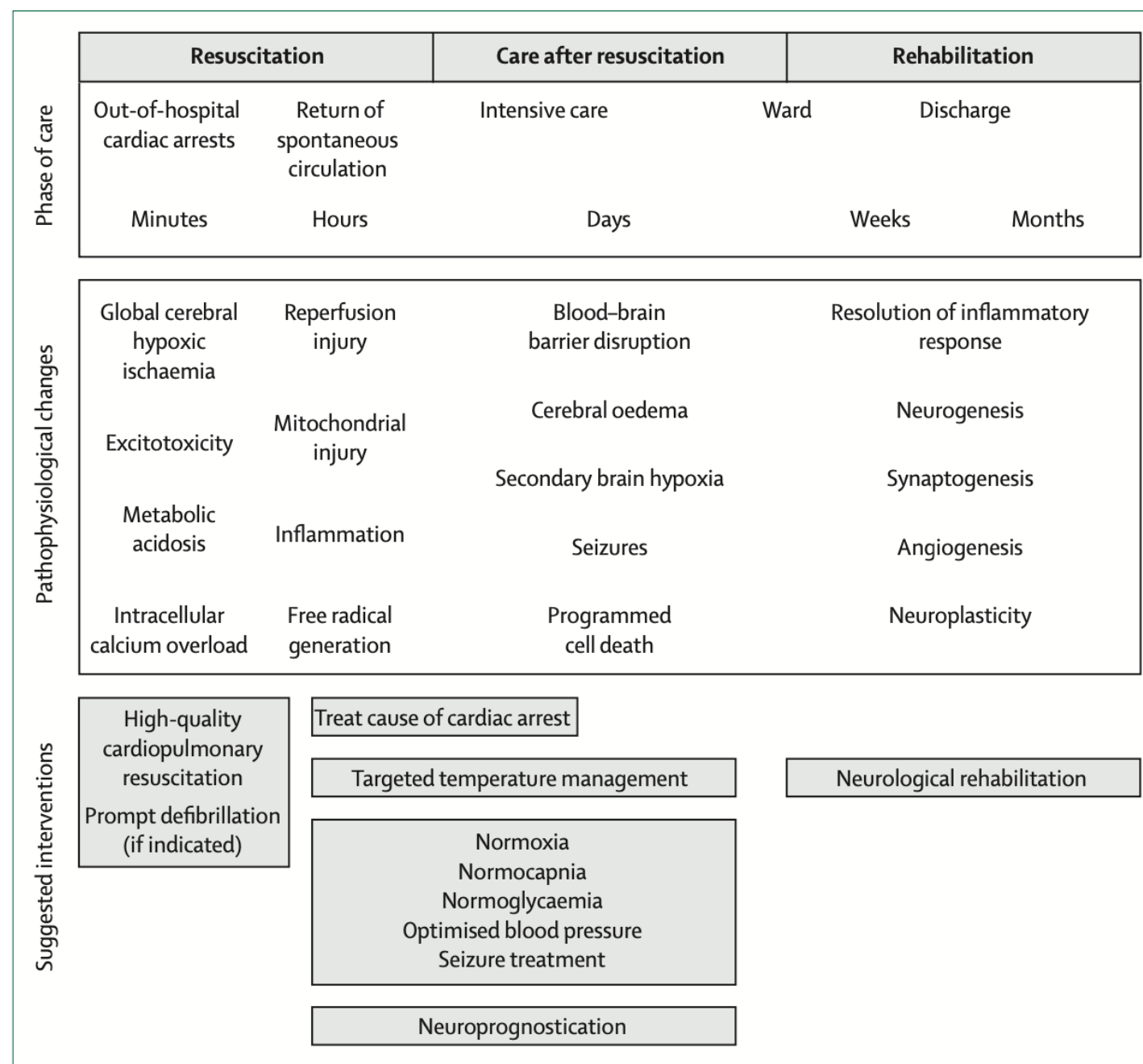
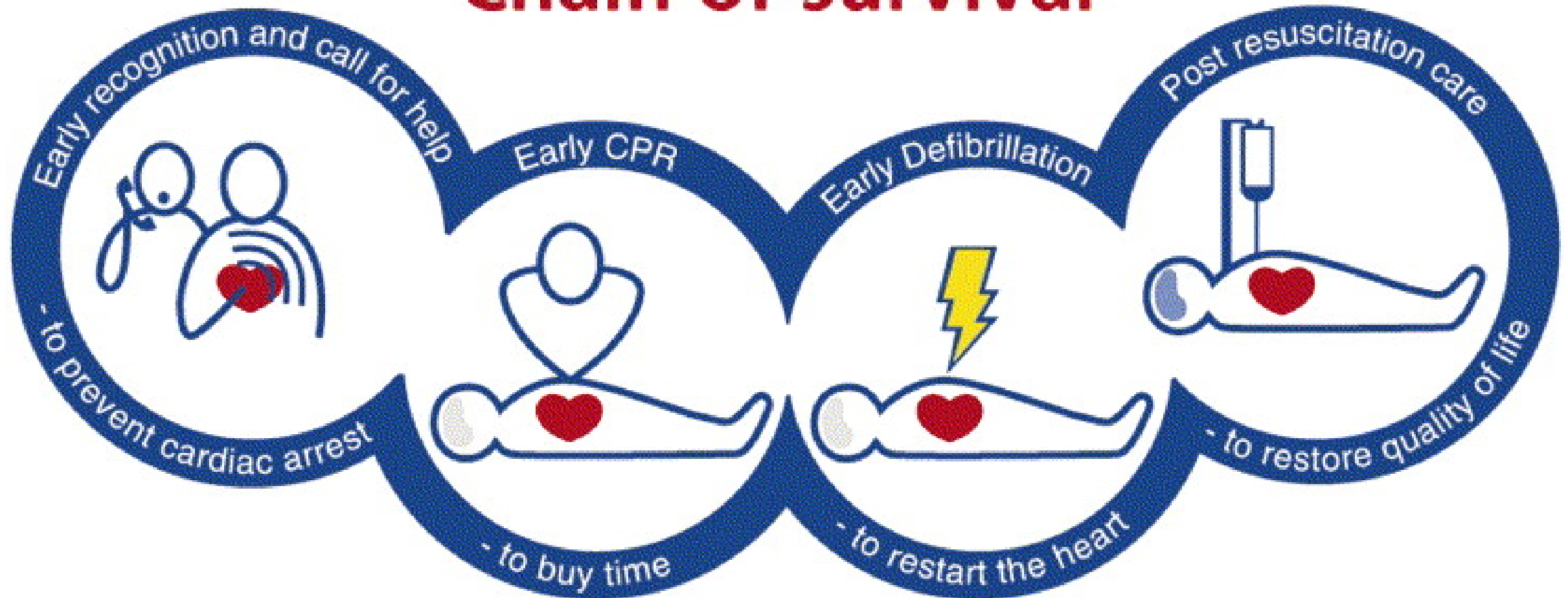


Figure 3: Simplified schematic representation of overlapping phases of brain injury after cardiac arrest and timing of therapeutic interventions

La catena della sopravvivenza

Chain of survival



Abstract

Objective: To determine which aspects of prehospital care impact outcomes after pediatric cardiac arrest.

Methods: In this study, the authors examine 5 years of consecutive data from their county emergency medical system (EMS), to identify predictors of good outcome after pediatric cardiac arrest, including return of spontaneous circulation (ROSC), survival to hospital admission (HA) and survival to hospital discharge (HD). Three logistic regression models were performed using JMP 14.1 Pro for Windows, each with the following nine predictors: age, sex, ventilation method (endotracheal intubation vs. supraglottic airway), initial rhythm (pulseless electrical activity vs. asystole), epinephrine administration, bystander treatment prior to EMS arrival, time from collapse to EMS arrival, automatic external defibrillator (AED) placement, and whether the arrest was witnessed. Odds ratio confidence intervals were calculated using the Wald method, and corresponding p-values were obtained with the likelihood ratio χ^2 test.

Results: From January 1, 2012 to December 31, 2016, there were 133 pediatric cardiac arrests, of which we had complete data on 109 patients for pediatric cardiac arrest. The median age was 8 months, with an IQR of 2.25–24 months, and a range of 0–108 months (0–9 years). There was return of spontaneous circulation (ROSC) in 20% of cases overall, with 16% making it to hospital admission, and 9% making it alive out of the hospital.

The median time to EMS arrival for witnessed events was 10 min, with an interquartile range (IQR) of 6.5–16 min, and a range of 0–25 min. The median time to EMS arrival for *un*witnessed events was 30 min, with an IQR of 19–62.5 min, and a range of 9–490 min.

Predictors of ROSC included epinephrine administration ($p=.00007$), bystander treatment before EMS arrival ($p=.0018$), older age ($p=.0025$), shorter time to EMS arrival ($p=.0048$), and AED placement. Predictors of hospital admission included epinephrine NOT being administered ($p=.0004$), bystander treatment before EMS arrival ($p=.0088$), shorter time to EMS arrival ($p=.0141$), and AED placement ($p=.0062$). The only significant predictor of survival to hospital discharge alive that was identified was shorter time to EMS arrival ($p=.0014$), as there was insufficient data for many of the predictor variables in this analysis.

Conclusion: Shorter time to EMS arrival from time of arrest, any bystander treatment prior to EMS arrival, and AED placement resulted in significantly higher rates of return of spontaneous circulation. Epinephrine administration significantly improved ROSC, but had the opposite effect on HA. Only shorter time to EMS arrival from time of arrest was significantly associated with survival to hospital discharge. *Each additional minute for the EMS to arrive resulted in 5% decreased odds of ROSC and hospital admission, and 12% decreased odds of surviving to hospital discharge.*

Keywords: Pediatric cardiac arrest, Resuscitation, CPR

ORIGINAL ARTICLE

Bystander Efforts and 1-Year Outcomes in Out-of-Hospital Cardiac Arrest

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and Bodil S. Rasmussen, M.D., Ph.D.

BACKGROUND

The effect of bystander interventions on long-term functional outcomes among survivors of out-of-hospital cardiac arrest has not been extensively studied.

METHODS

We linked nationwide data on out-of-hospital cardiac arrests in Denmark to functional outcome data and reported the 1-year risks of anoxic brain damage or nursing home admission and of death from any cause among patients who survived to day 30 after an out-of-hospital cardiac arrest. We analyzed risks according to whether bystander cardiopulmonary resuscitation (CPR) or defibrillation was performed and evaluated temporal changes in bystander interventions and outcomes.

RESULTS

Among the 2855 patients who were 30-day survivors of an out-of-hospital cardiac arrest during the period from 2001 through 2012, a total of 10.5% had brain damage or were admitted to a nursing home and 9.7% died during the 1-year follow-up period. During the study period, among the 2084 patients who had cardiac arrests that were not witnessed by emergency medical services (EMS) personnel, the rate of bystander CPR increased from 66.7% to 80.6% ($P<0.001$), the rate of bystander defibrillation increased from 2.1% to 16.8% ($P<0.001$), the rate of brain damage or nursing home admission decreased from 10.0% to 7.6% ($P<0.001$), and all-cause mortality decreased from 18.0% to 7.9% ($P=0.002$). In adjusted analyses, bystander CPR was associated with a risk of brain damage or nursing home admission that was significantly lower than that associated with no bystander resuscitation (hazard ratio, 0.62; 95% confidence interval [CI], 0.47 to 0.82), as well as a lower risk of death from any cause (hazard ratio, 0.70; 95% CI, 0.50 to 0.99) and a lower risk of the composite end point of brain damage, nursing home admission, or death (hazard ratio, 0.67; 95% CI, 0.53 to 0.84). The risks of these outcomes were even lower among patients who received bystander defibrillation as compared with no bystander resuscitation.

CONCLUSIONS

In our study, we found that bystander CPR and defibrillation were associated with risks of brain damage or nursing home admission and of death from any cause that were significantly lower than those associated with no bystander resuscitation. (Funded by TrygFonden and the Danish Heart Foundation.)

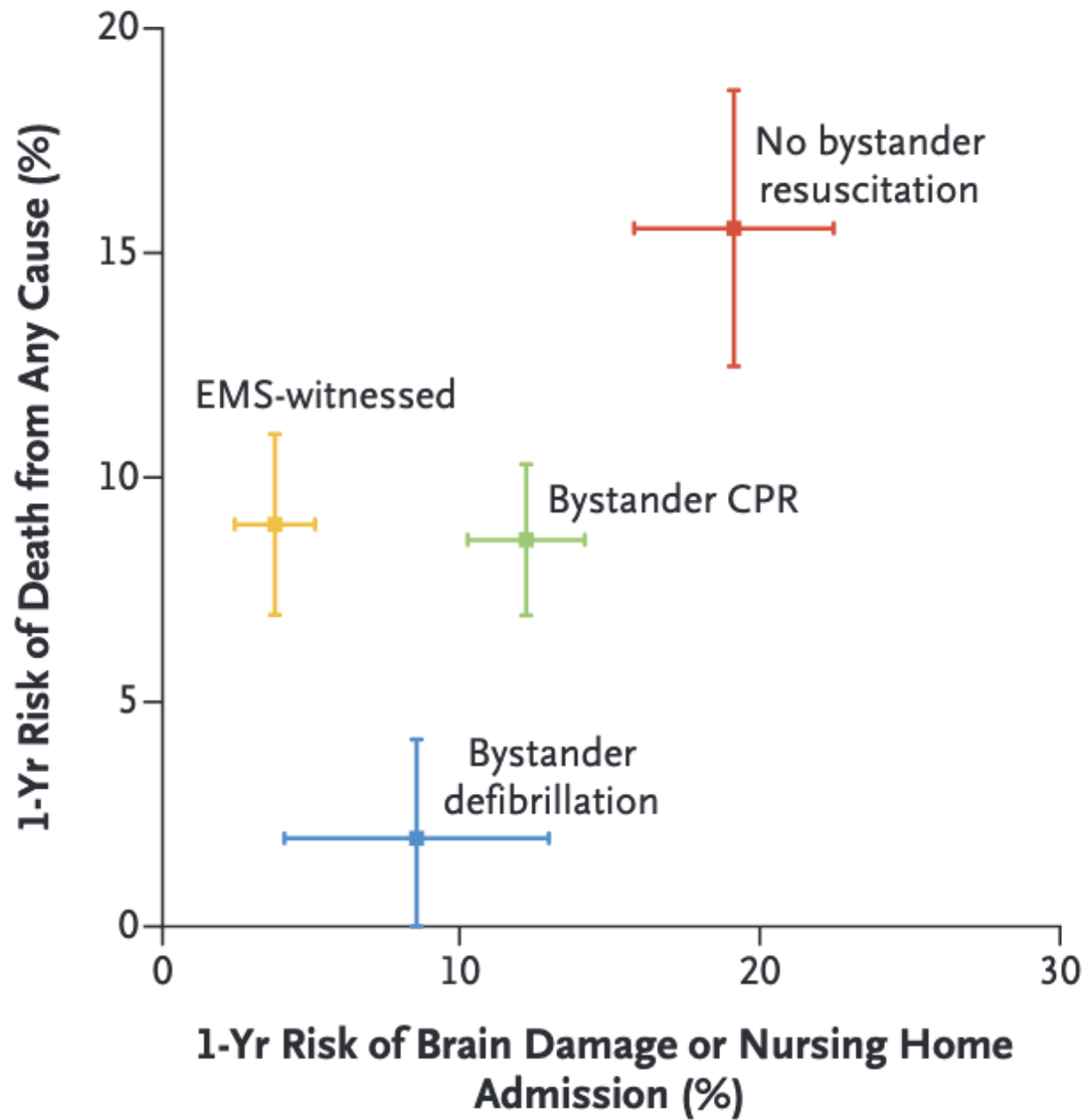


Figure 4. Absolute Risk of Anoxic Brain Damage or Nursing Home Admission and Death from Any Cause at 1 Year of Follow-up According to EMS-Witnessed and Bystander-Intervention Status.

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.

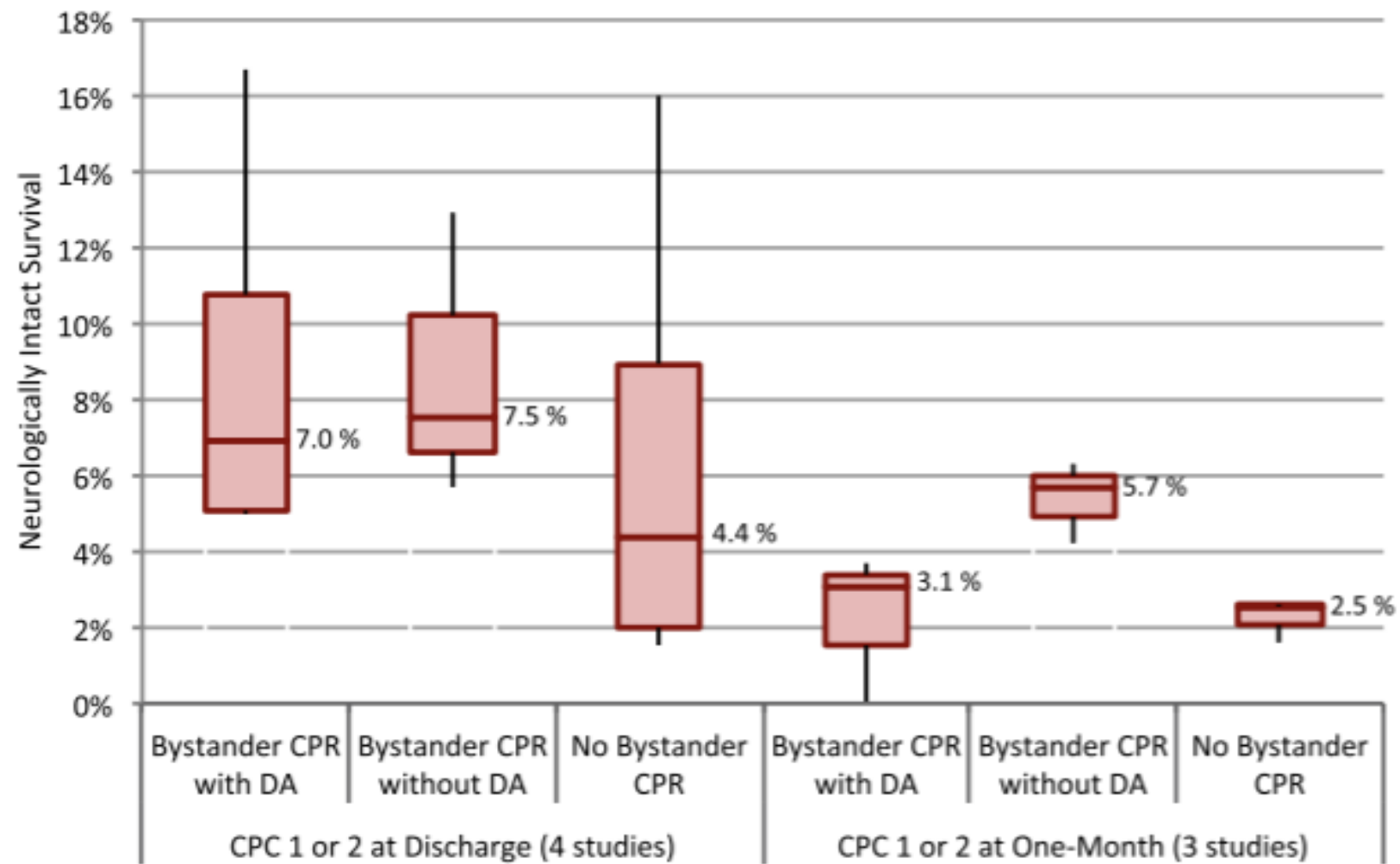


Fig. 3 Median neurologically intact survival defined as Cerebral Performance Category (CPC) 1 or 2 at hospital discharge or one-month with interquartile range (IQR) and minimum/maximum range according to bystander CPR status. Horizontal line within boxes represents median, upper and lower border of boxes reflect IQR, and the black lines show the range of the observations. Details are reported in Table 3. DA, dispatcher assistance; CPC, Cerebral Performance Category; CPR, cardiopulmonary resuscitation

Role of dispatcher

Dispatch-assisted recognition of cardiac arrest

- Dispatch centres should implement standardised criteria and algorithms to determine if a patient is in cardiac arrest at the time of the emergency call.
- Dispatch centres should monitor and track their ability to recognize cardiac arrest and continuously look for ways to improve recognition of cardiac arrest.

Dispatch-assisted CPR

- Dispatch centres should have systems in place to make sure call handlers provide CPR instructions for unresponsive persons not breathing normally.

Dispatch-assisted chest compression-only compared with standard CPR

- Dispatchers should provide chest compression–only CPR instructions for callers who identify unresponsive adult persons not breathing normally.

Dispatch-assisted CPR

ILCOR recommends that emergency medical dispatch centres have systems in place to enable call handlers to provide CPR instructions for adult patients in cardiac arrest.²² This strong recommendation was based on very-low certainty evidence drawn from 30 observational studies; 16 studies comparing outcomes from patients when dispatch-assisted CPR instruction was offered with outcomes from patients when dispatch-assisted CPR instruction was not offered^{23,31,135,140,148,151,153,173–181} and 14 studies comparing outcomes from patients when dispatch-assisted CPR instruction was received with outcomes from patients when dispatch-assisted CPR instruction was not received.^{135,140,148,173–176,179,180}

Bystander CPR su vittime NON in ACC

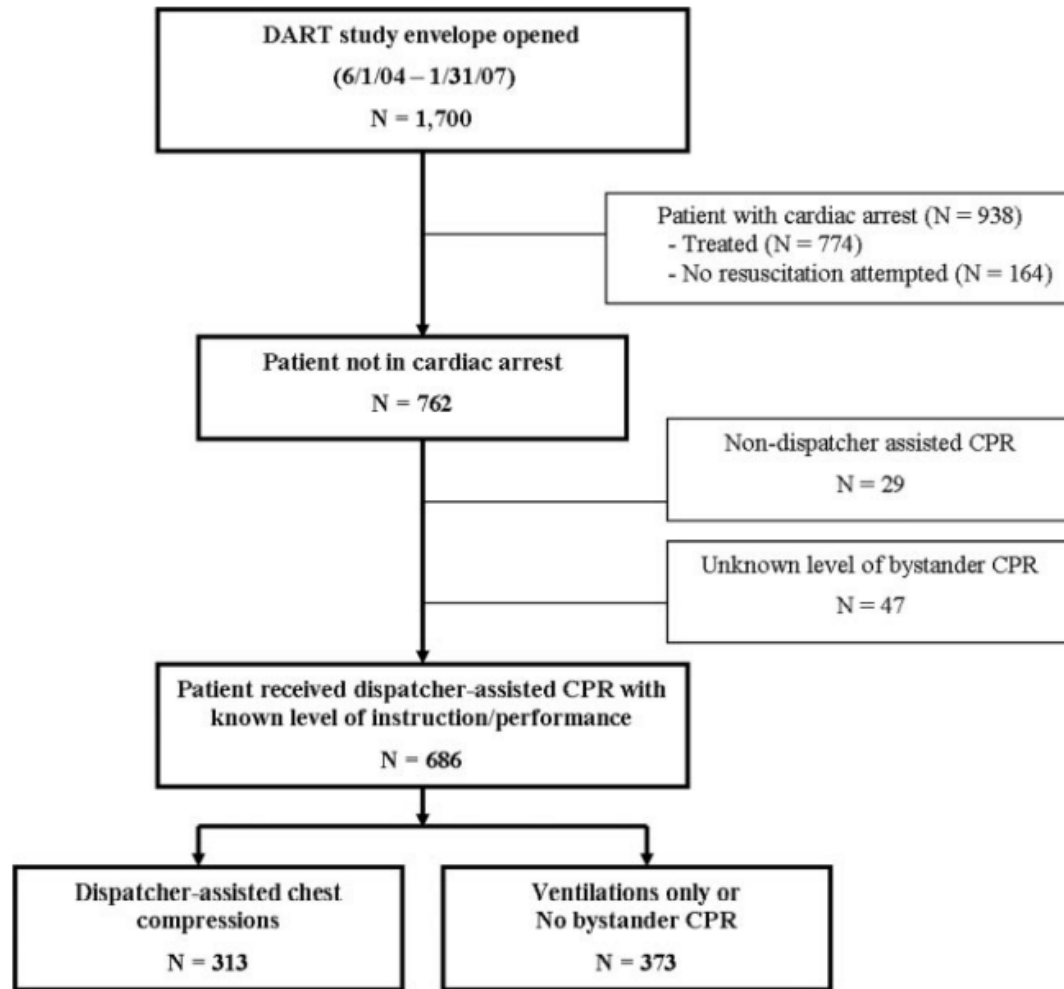


Figure. Flow diagram of subject eligibility.

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)	<i>P</i>
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.

Discussion

In this cohort study of dispatcher-assisted bystander CPR, nearly half of the patients for whom emergency dispatchers offered CPR instructions were not in cardiac arrest. Approximately 18% of dispatcher-assisted CPR instruction resulted in bystander chest compressions for patients not in cardiac arrest. For these patients, EMS most often determined that the patient had a drug or alcohol overdose, seizure, syncope, cerebrovascular event, or hypoglycemia. A total of 9% of these patients experienced discomfort, and 2% sustained injuries likely attributed to the bystander CPR; an additional 3% possibly experienced discomfort, and 1% possibly suffered injuries resulting from bystander CPR. However, only 2% of patients not in arrest suffered a fracture, and no patients experienced visceral organ injury or death as a consequence of bystander CPR.

This rate of injury is far lower than what has been observed in previous studies of CPR complications, which have reported rates of injury ranging from 21% to 65%.^{17,18} These prior studies, however, were primarily autopsy studies conducted on true cardiac arrest patients who had undergone extended resuscitation efforts by medical personnel. Longer duration of CPR is associated with an increased risk of injury.¹⁰ In the present study, patients not in cardiac arrest received only a median duration of 91 seconds of (bystander) CPR. CPR was interrupted by the arrival of EMS or when characteristics indicating that the patient was not in arrest became apparent to the bystander or dispatcher. Moreover, simulation studies suggest that CPR by laypersons often does not produce guideline-directed compression depth and thus would presumably be less likely to cause injury.¹⁹

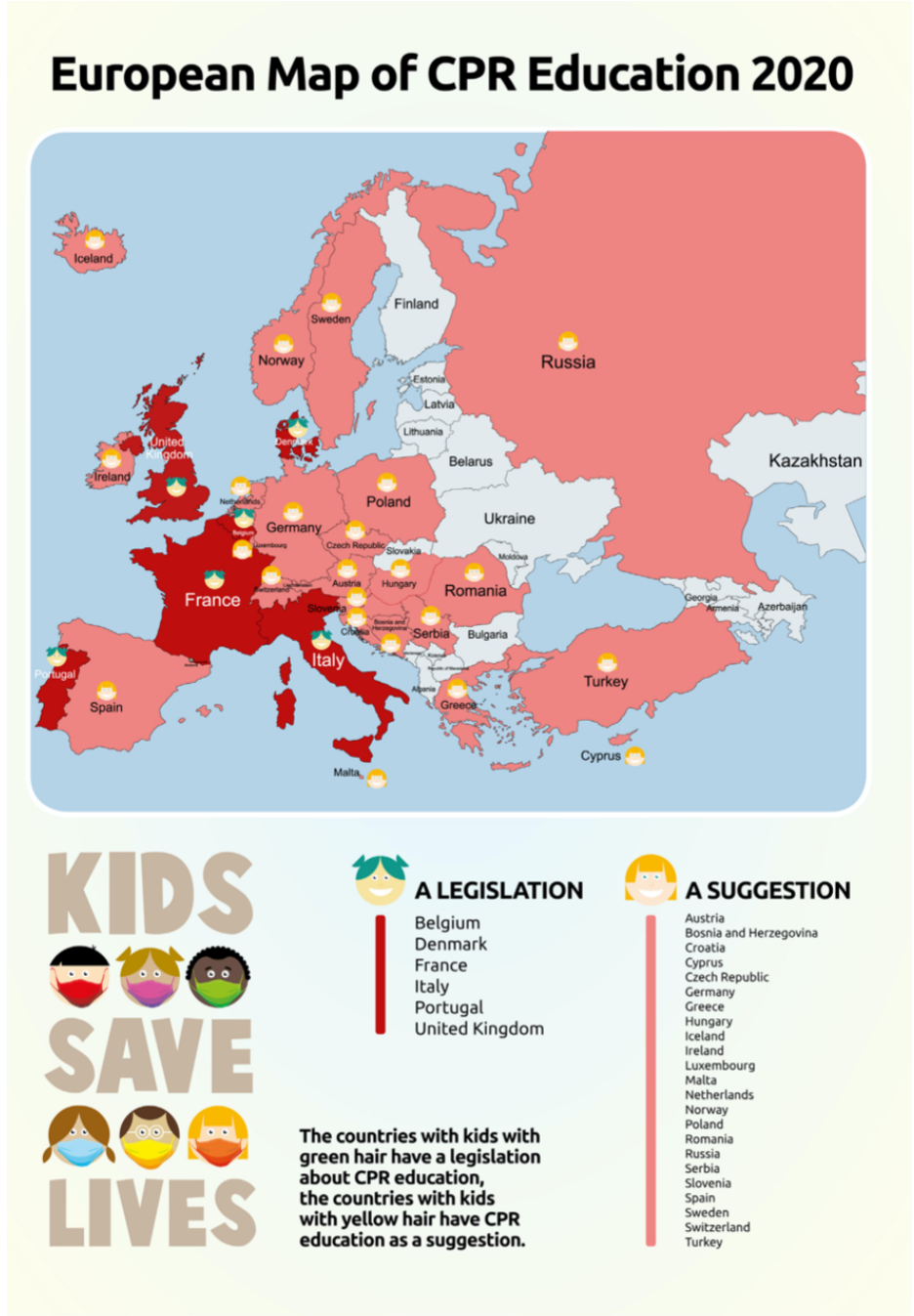


Fig. 2 – Kids save lives: legal requirement for CPR education in schools across Europe.

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

SYSTEMS SAVING LIVES GL 2021

5 TOP MESSAGES



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

XVII Legislatura

dal 15/03/2013 - al 22/03/2018

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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](#)

rigenera.

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 **iniziative 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 .

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.

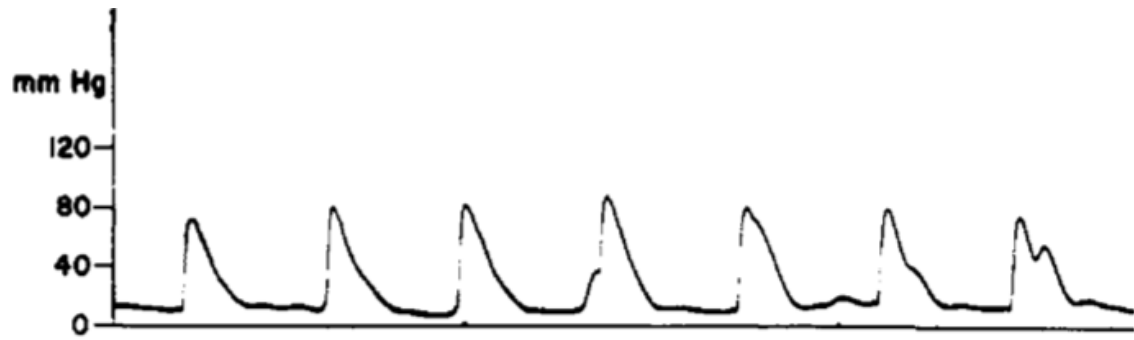


Fig. 3.—Blood pressure produced in an adult by closed-chest cardiac massage.

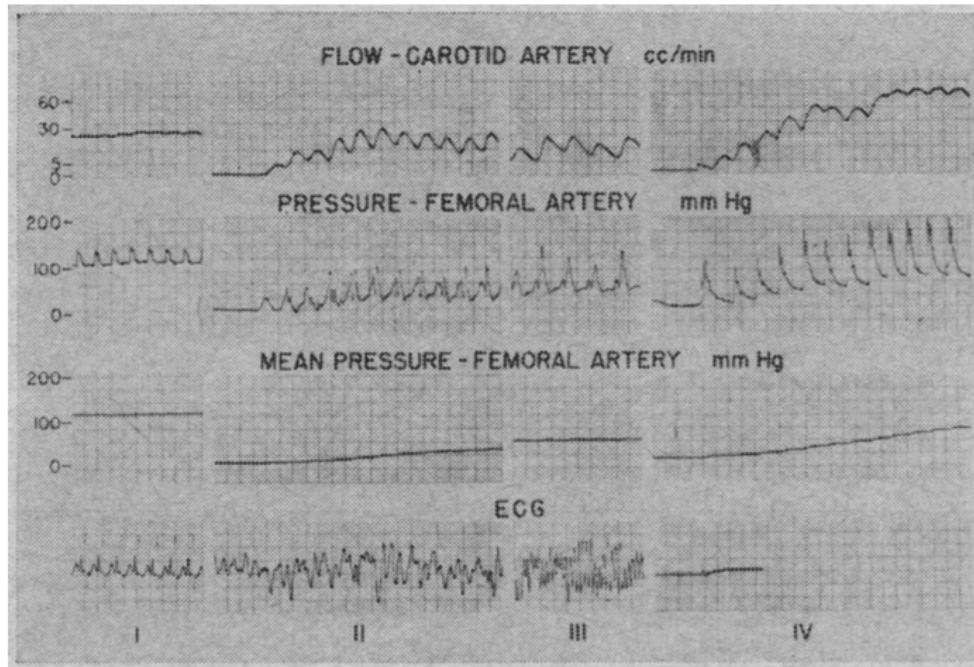


Fig. 1.—Record of blood flow, pressures, and electrocardiogram of dog whose heart was in ventricular fibrillation for eight minutes. I: normal initial values; II: start of closed-chest massage; III: seventh minute of massage; IV: closed-chest defibrillation.

Kouwenhoven WB, Jude JR, Knickerbocker GG. CLOSED-CHEST CARDIAC MASSAGE. JAMA. 1960;173(10):1064-1067.
doi:10.1001/jama.1960.03020280004002



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

Compressioni toraciche

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 $100\text{--}120\text{ min}^{-1}$ 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.

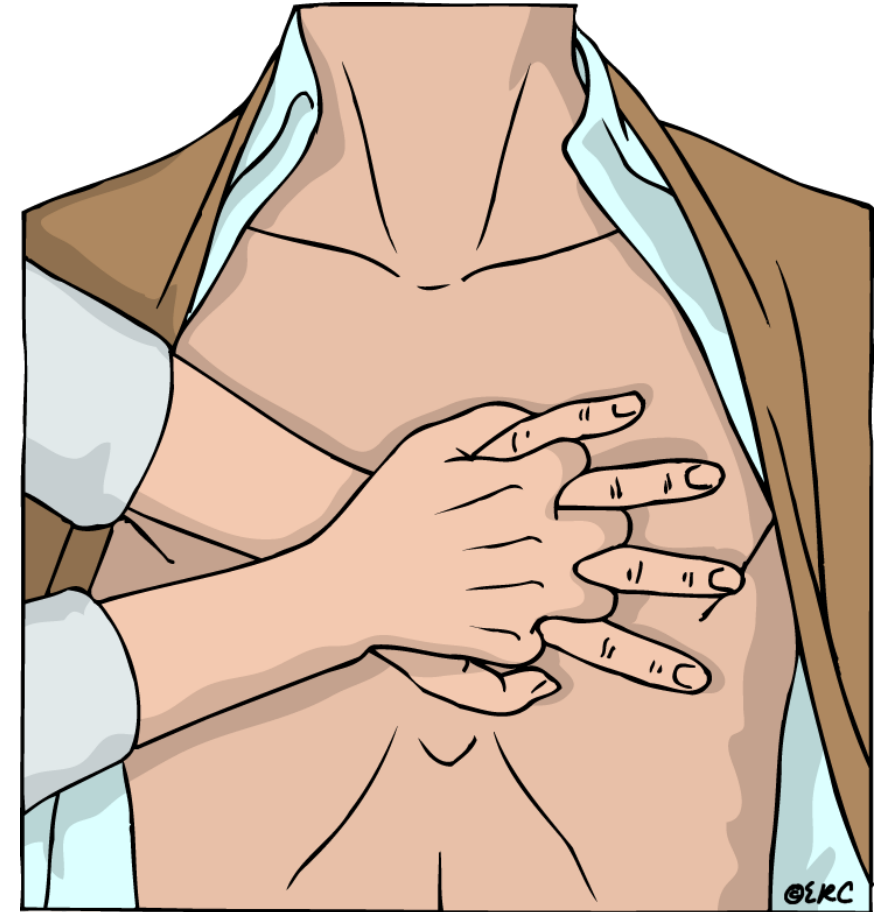
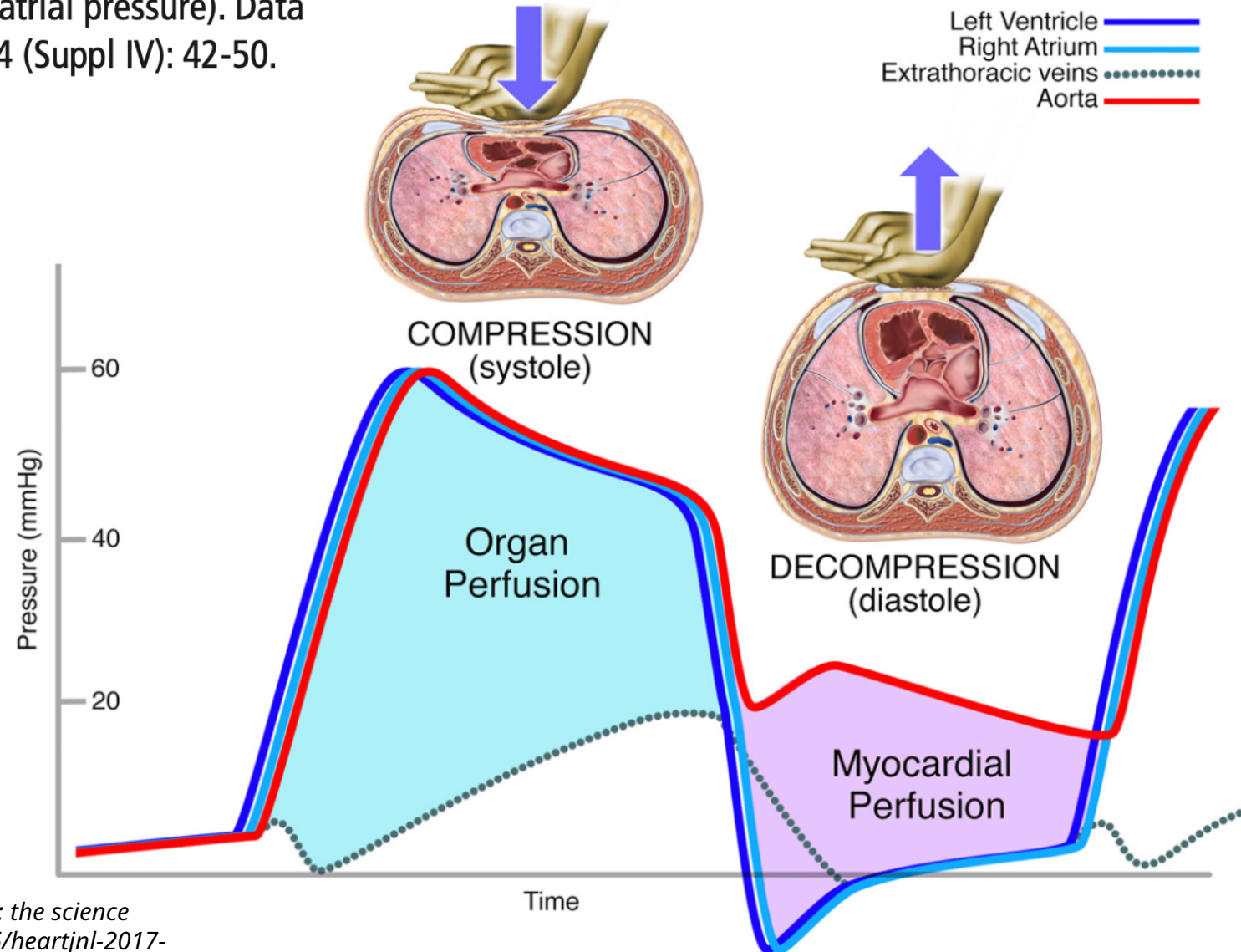


Figure 3 Haemodynamic effects of compression and decompression phases of cardiopulmonary resuscitation. Compression phase creates organ perfusion pressure (difference between aortic and extrathoracic vein pressure). Decompression phase creates myocardial perfusion pressure (difference between aortic and right atrial pressure). Data adapted from Criley *et al. Circulation*. 1986; 74 (Suppl IV): 42-50.



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

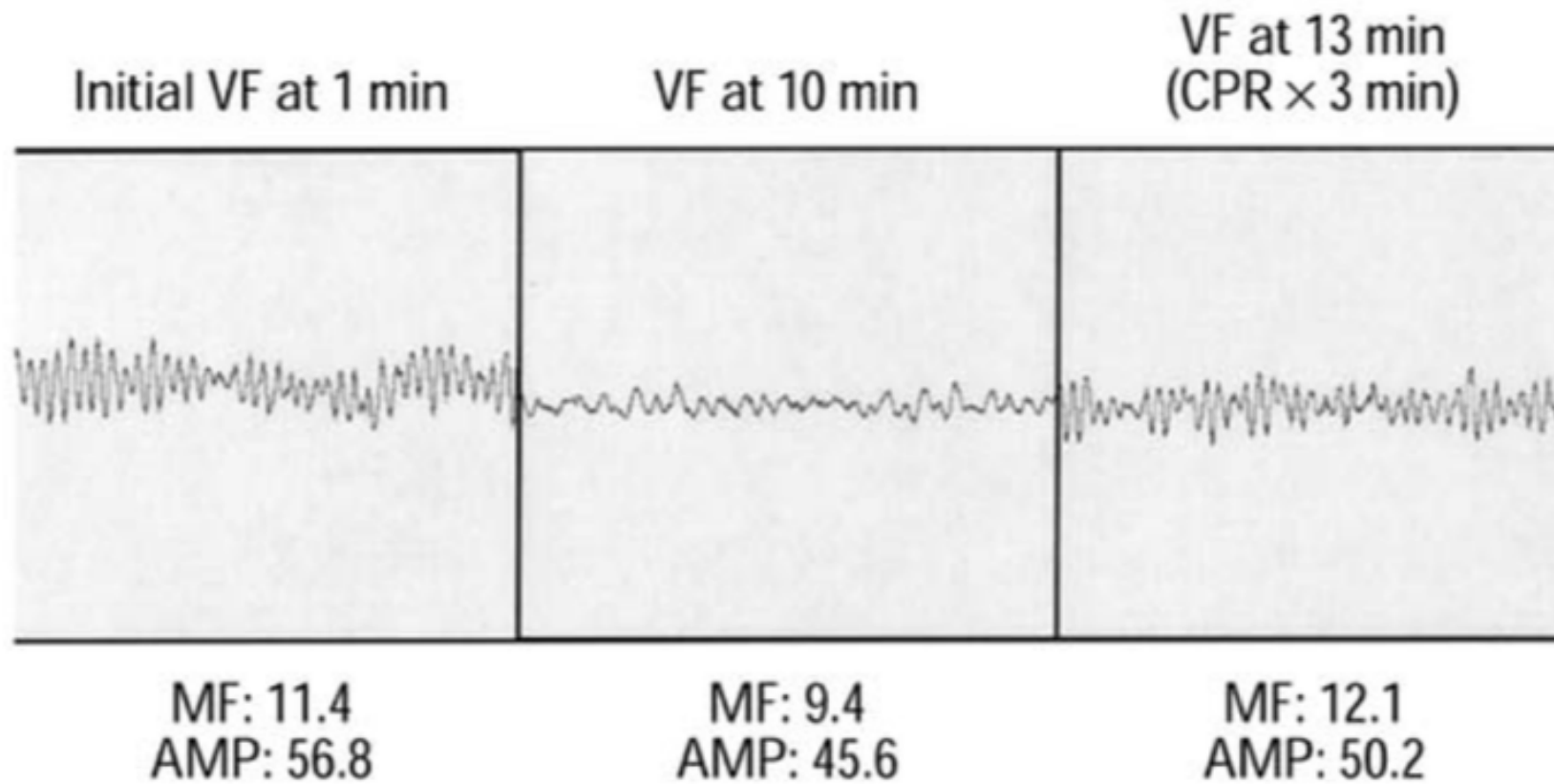
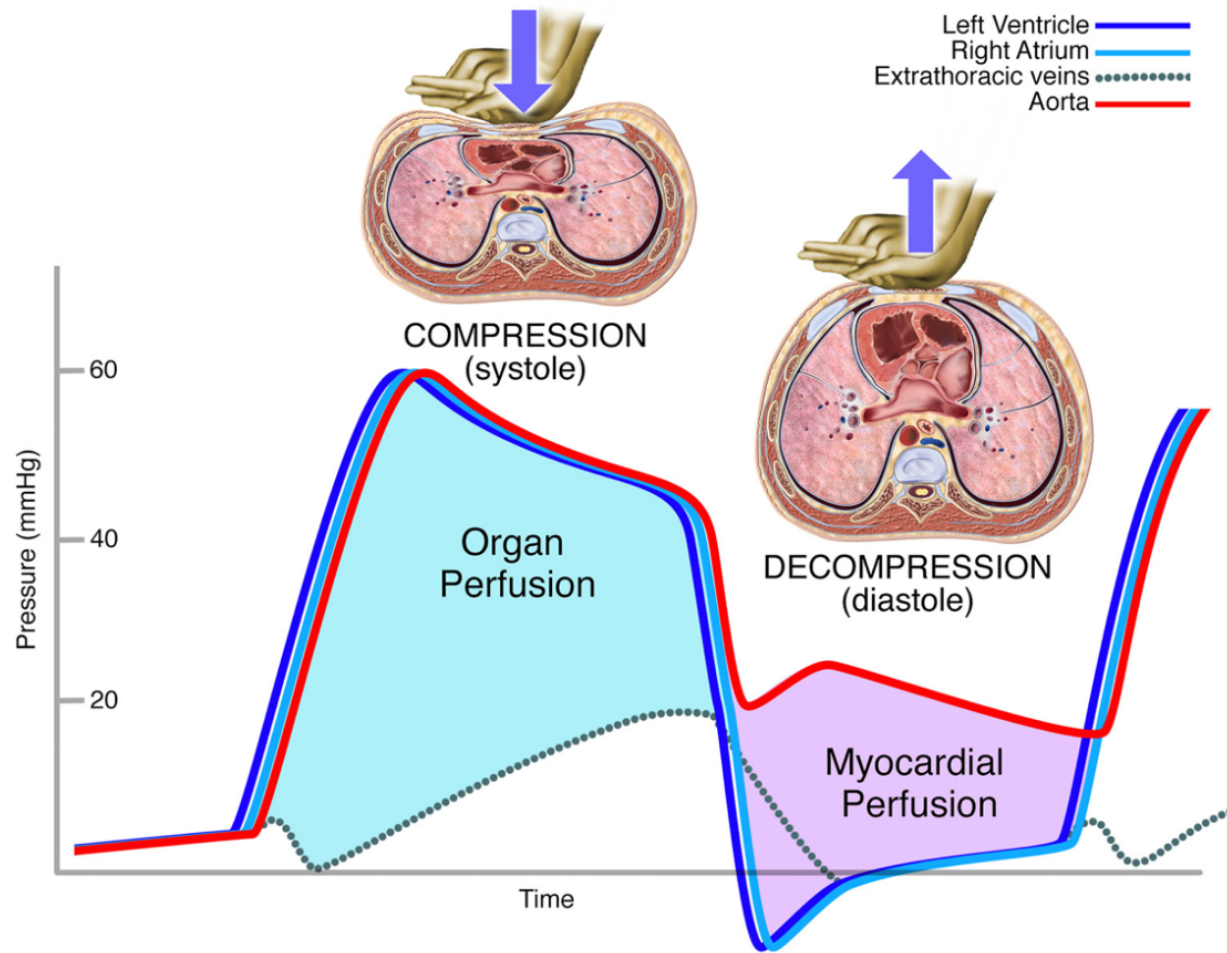


Figure 2 Typical changes in ventricular fibrillation (VF) waveform in untreated VF (after 1 and 10 min) and after 3 min of cardiopulmonary resuscitation-first (13 min) in swine. AMP, amplitude (in mV); MF, VF median frequency (in Hz) (from Berg *et al*⁸). Reproduced with permission from Elsevier.



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

‘Diastole’ occurs during the decompression phase of the CPR cycle—when the thorax is permitted to rebound to its normal fully expanded configuration. While seemingly passive and unimportant, CPR decompression may be even more important than its compression phase. During this phase, closure of the aortic valve maintains an aortic pressure that is higher than intracardiac pressures, which fall precipitously beneath the closed valve, driven by the ‘vacuum’ effect created by the recoiling thoracic cage. This intrathoracic vacuum is what draws blood to return back into the chest from the periphery, filling the heart, lungs and great vessels in preparation for the next chest compression. The result is that the better the decompression, the stronger the vacuum, the better the refilling, and ultimately the more thoracic blood available for subsequent compression.

CPR diastole serves a second useful purpose that is often unappreciated. All organs of the body are perfused during the compression phase of CPR except, ironically, the heart itself, which is not perfused by blood within its chambers, but rather by the coronary arteries. Coronary blood flow is estimated by coronary perfusion pressure (CPP), defined as the difference between pressure in the aorta (where the coronary arteries originate) and in the right atrium (where coronary venous blood ultimately returns).¹² During the compression phase of CPR, all intrathoracic pressures including the aorta and all chambers of the heart equalise with the compressive force of the hands, resulting in no coronary blood flow between the aorta and right atrium. Conversely, in CPR diastole, the higher aortic pressure above its closed valve compared with the falling intrathoracic pressure results in a positive CPP. It is this combination of compressive

Chest recoil (AHA recommendation: allow full chest recoil)

Permitting complete chest recoil during the decompression phase of CPR is essential for refilling the chest and for adequate myocardial perfusion. Incomplete chest recoil or leaving a residual pressure on the chest ('leaning') results in an increase in intrathoracic pressure when it needs to be at its minimum.

Among a group of animals that received standard chest compressions with full chest recoil, followed by chest compressions permitting only 75% of full chest recoil, CPP decreased by more than a third during the period of incomplete chest recoil and cerebral perfusion pressures by more than 50% (both $P < 0.05$).²⁵

Inattention to full recoil is an all too common problem during resuscitation. The problem often arises as a result of fatigue, whereby the chest compressor (who is usually leaning over the patient while performing compressions) unintentionally uses the patient's chest as a resting table during the decompression phase

of CPR, rather than permit its full recoil. An observational study of 108 adults with in-hospital cardiac arrest at the University of Pennsylvania used force-sensing devices during resuscitation to detect 5 pounds of residual pressure ('lean') left on the chest during the decompression phase of CPR. By this definition, leaning was observed in 91% of the resuscitations, underscoring its all-too-common occurrence.²⁶

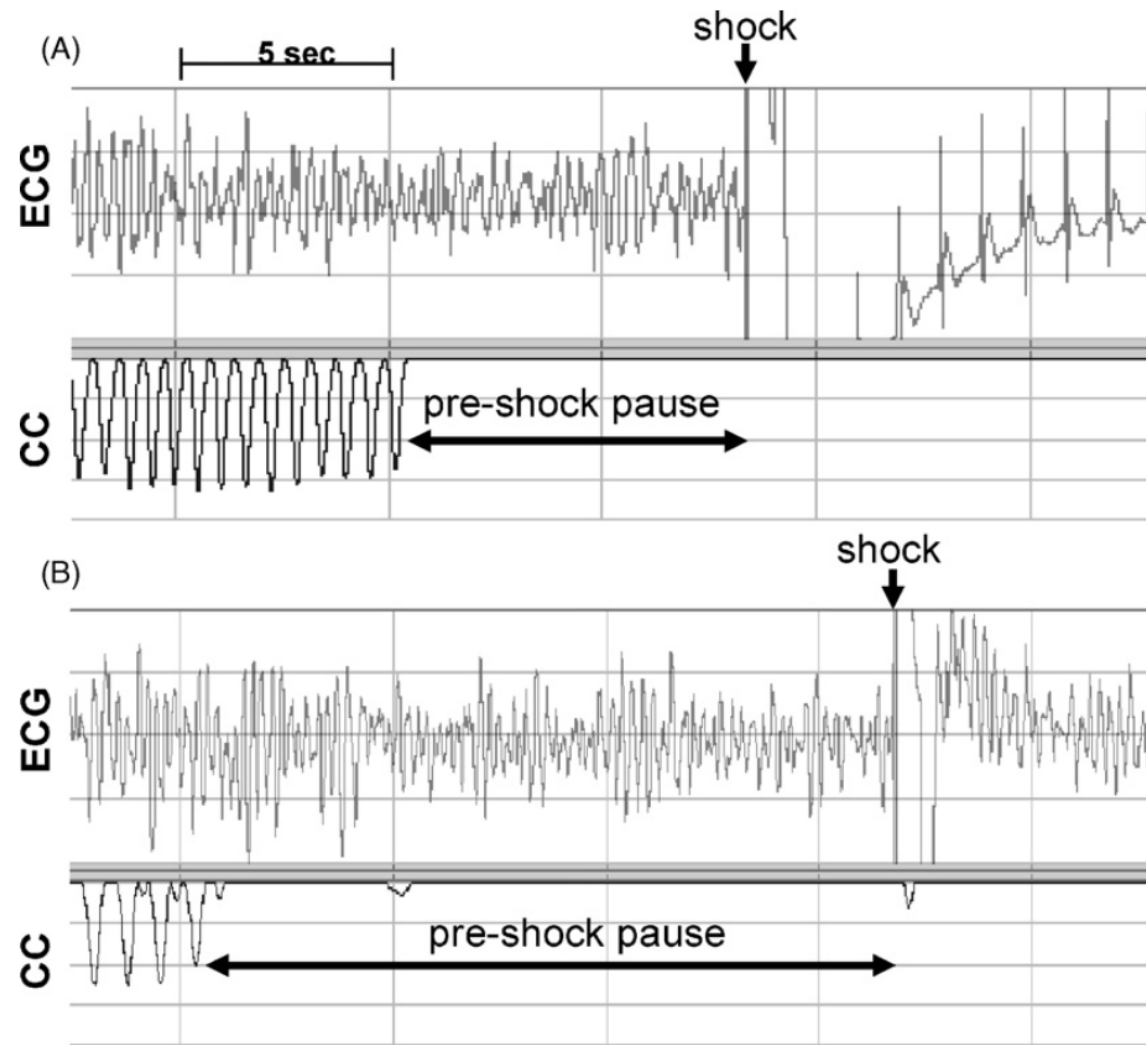


Figure 1 Examples of defibrillation attempts. (A) Successful shock preceded by an 8-s pre-shock pause and deep chest compressions. (B) Unsuccessful shock preceded by a 16 s pre-shock pause and shallower chest compressions. ECG, electrocardiogram; CC, chest compressions.

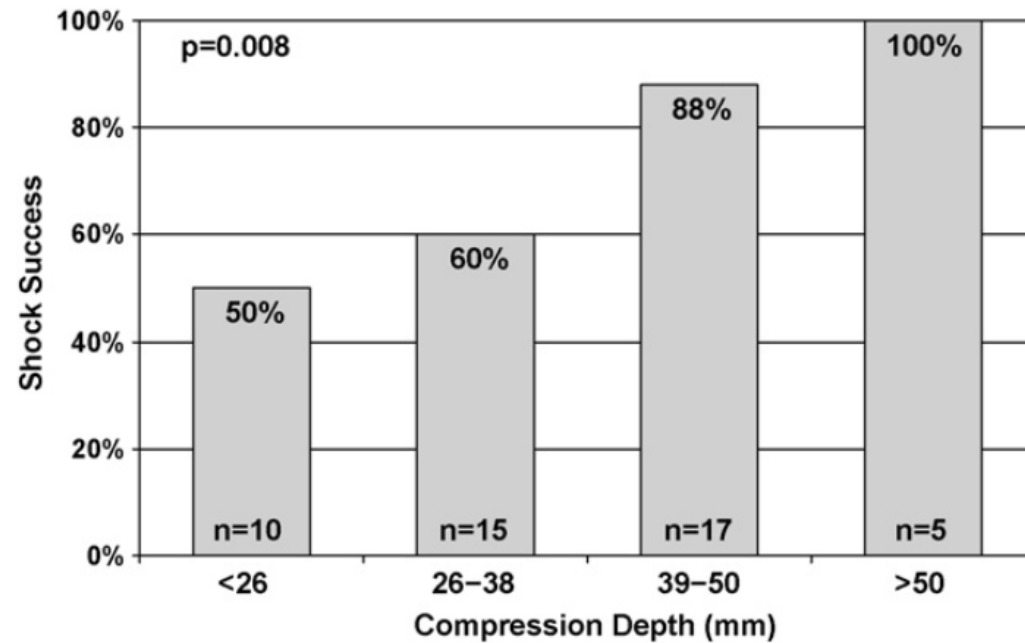


Figure 3 Association between chest compression depth and shock success. Cases are grouped by 30s average compression depth in approximately 11 mm (0.5 in.) intervals. Chest compression depth of 38–50 mm (1.5–2 in.) represents current CPR guidelines recommendations. Deeper chest compressions are significantly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

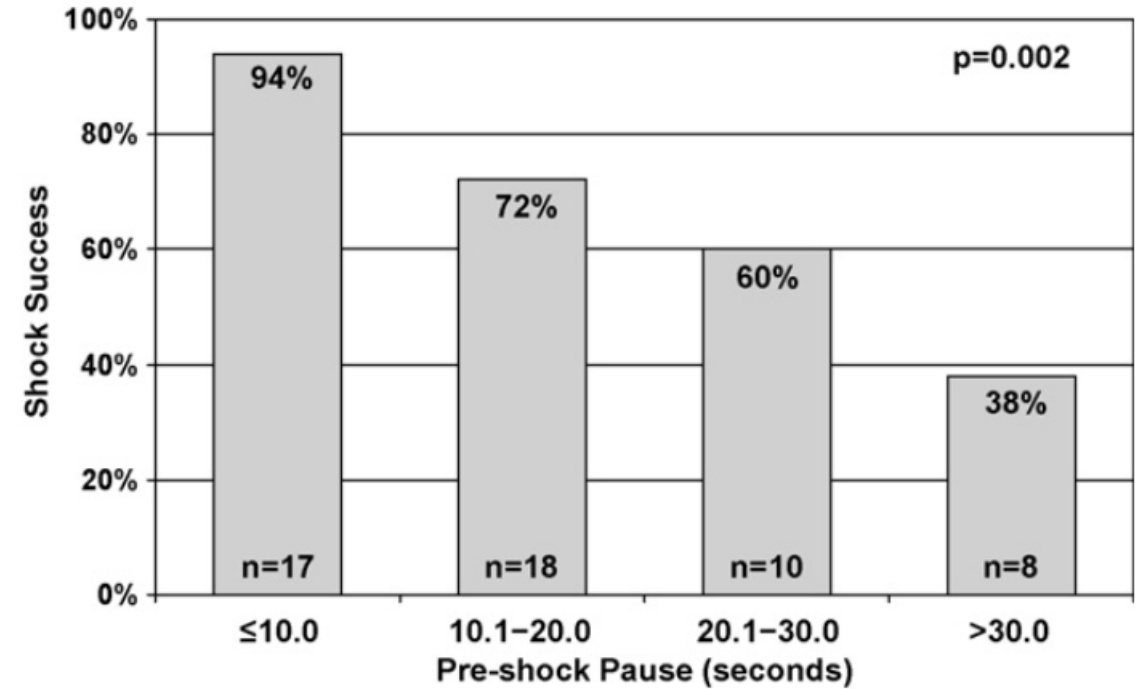
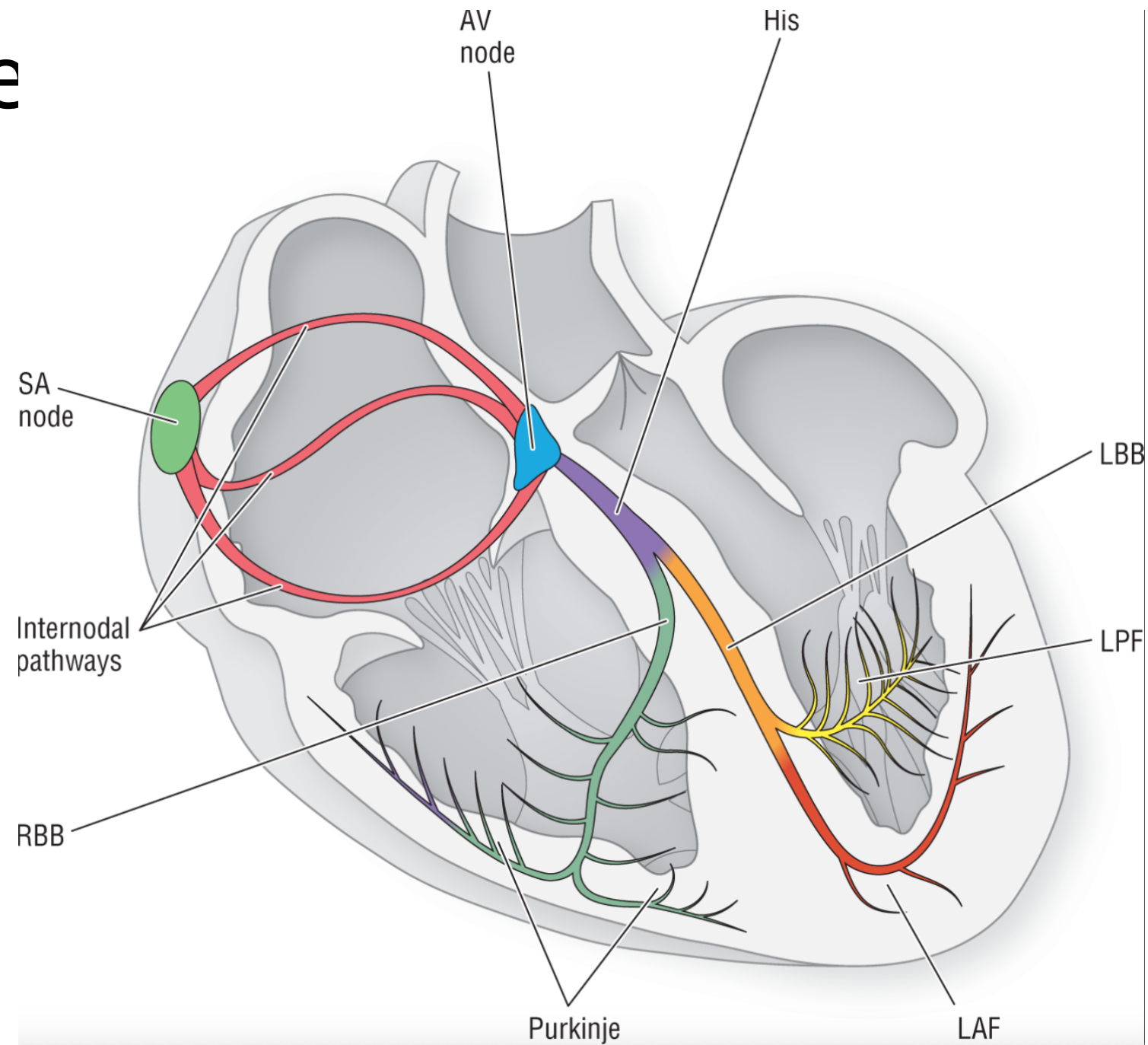


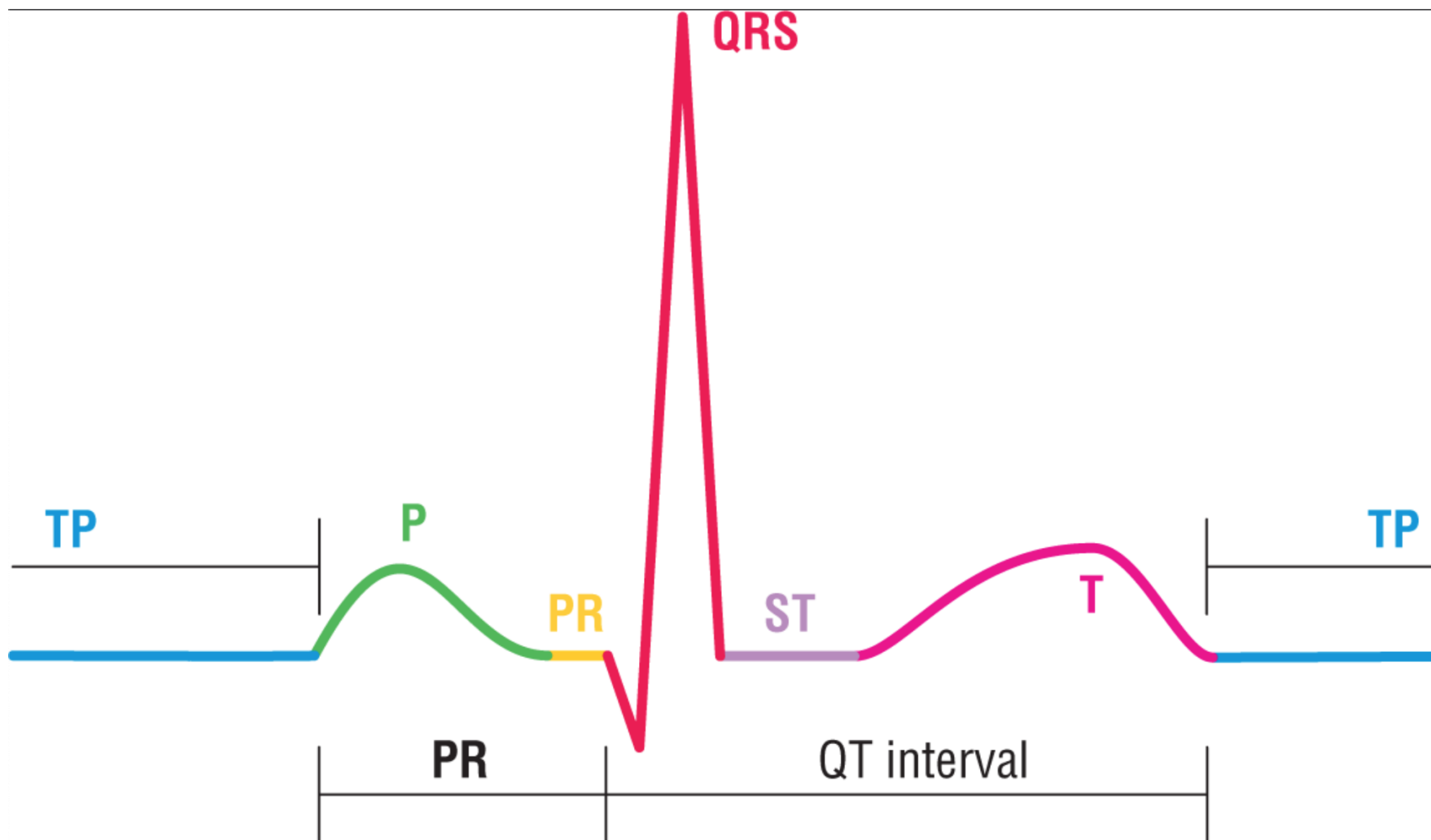
Figure 2 Association between pre-shock pause and shock success. Cases are grouped by pre-shock pause in 10s intervals. Note that longer pre-shock pauses are significantly associated with a smaller probability of shock success.

These findings led ILCOR to suggest performing manual chest compressions on a firm surface when possible (weak recommendation, very low certainty evidence). ILCOR also suggested that when a bed has a CPR mode that increases mattress stiffness, it should be activated (weak recommendation, very-low-certainty evidence), but suggested against moving a patient from a bed to the floor to improve chest compression depth in the hospital setting (weak recommendation, very-low-certainty evidence). The confidence in effect estimates is so low that ILCOR was unable to make a recommendation about the use of a backboard strategy.

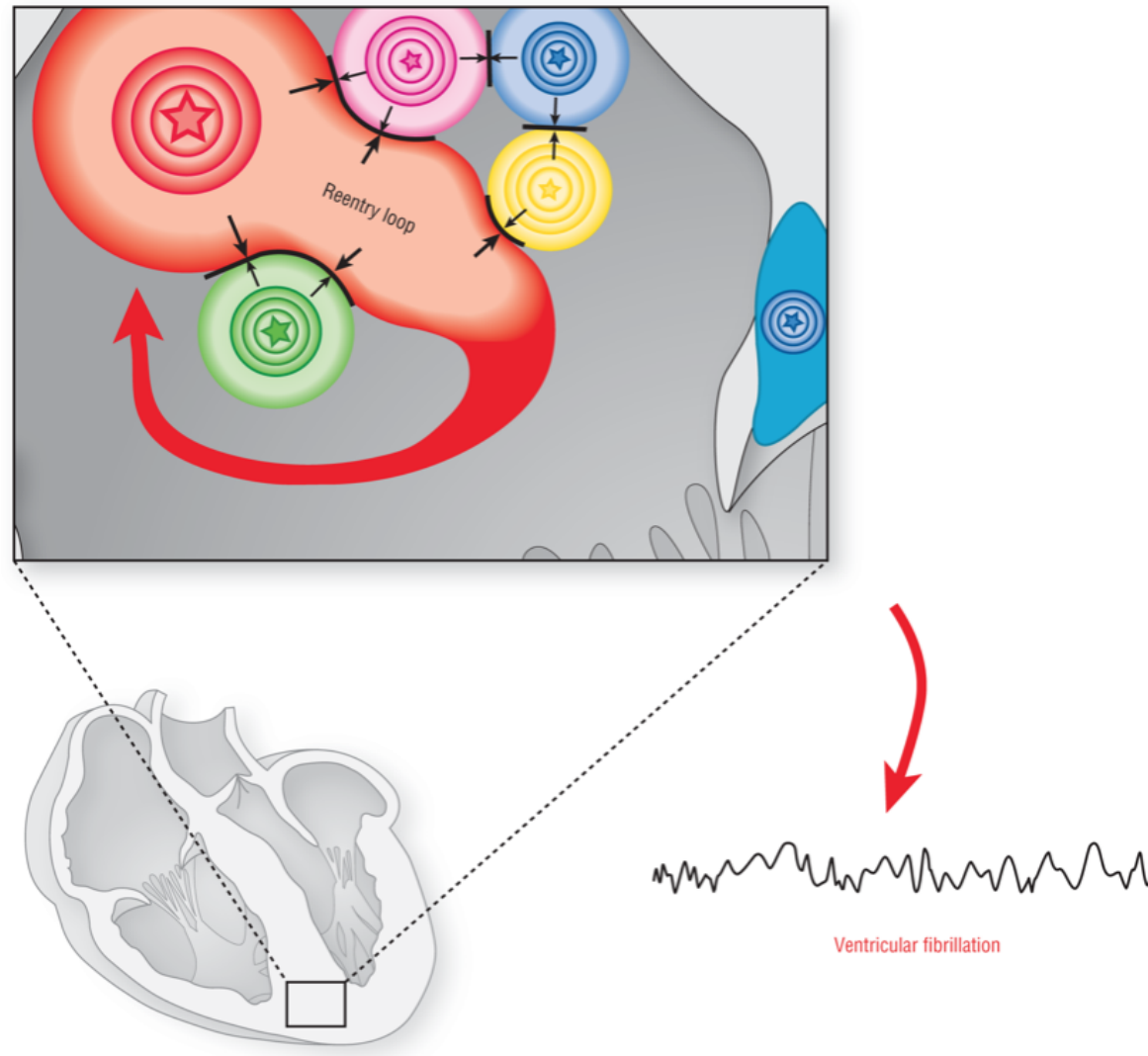
Il ritmo normale



Il ritmo normale



Ischemia del miocardio e aritmogenesi



Ventricular Arrhythmogenesis in Ischemic Myocardium

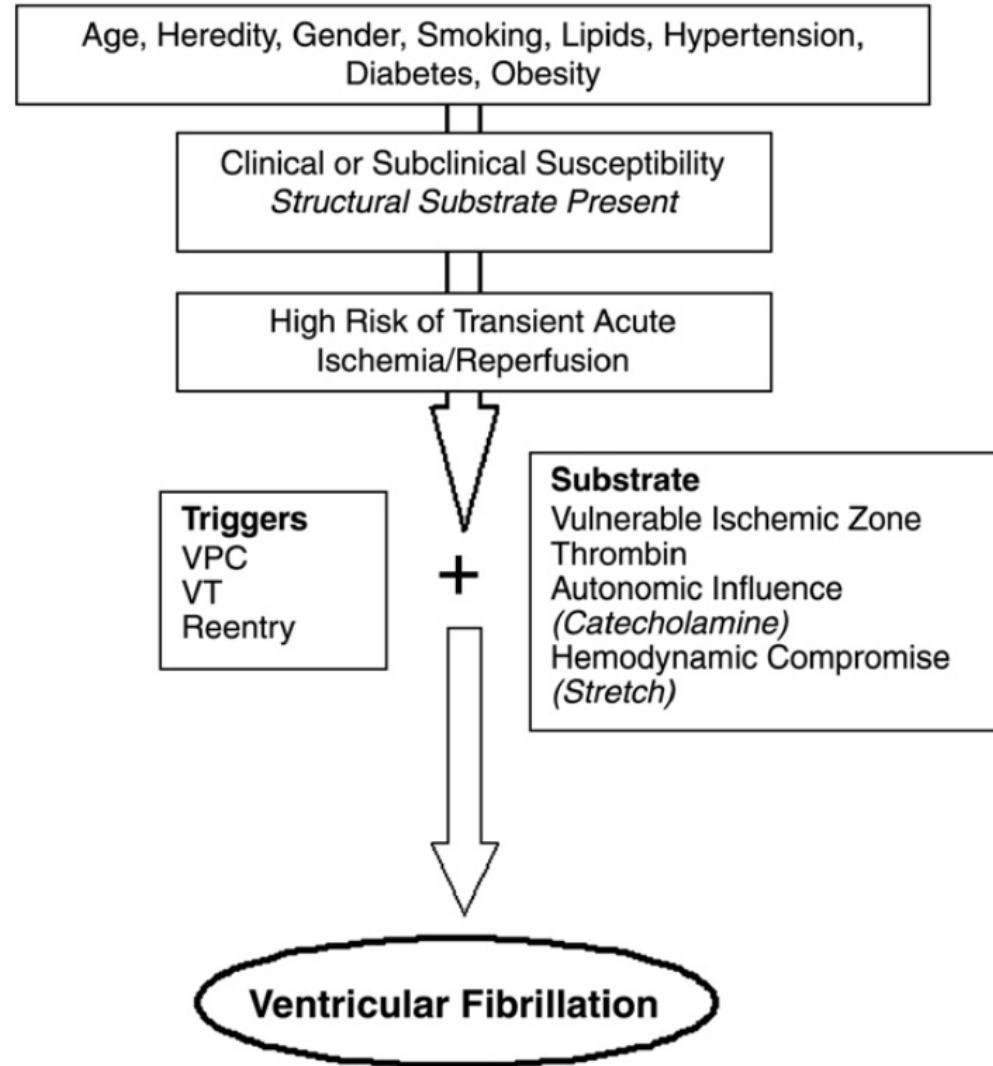
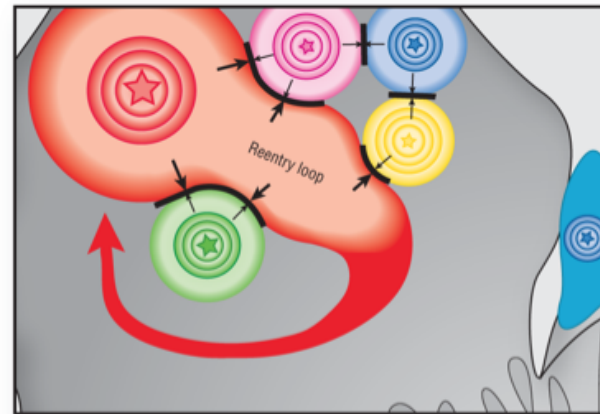


Fig. 1. Showing the cascade of ventricular arrhythmogenesis in ischemic myocardium in relation to the risk factors, triggers and substrate. Abbreviations: VPC, Ventricular premature contraction; VT, Ventricular tachycardia.

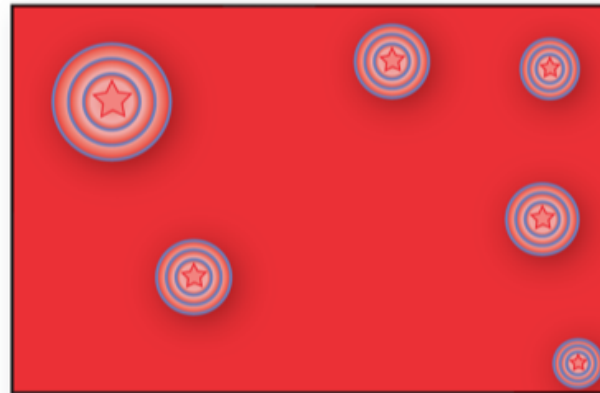




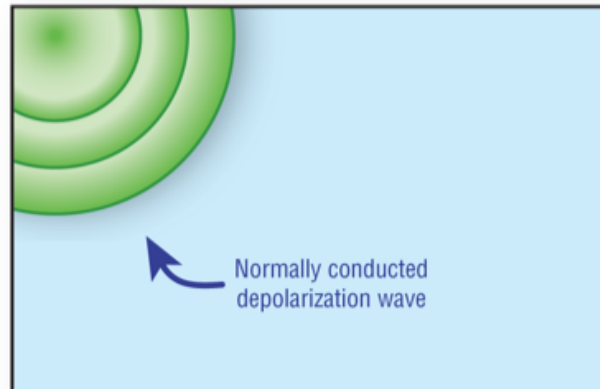
Defibrillazione



Ventricular fibrillation



Defibrillation



Normal sinus rhythm

Defibrillazione

3.2.2. Transthoracic impedance

It refers to the dissipation of energy in the lungs, thoracic cage and the other anatomic structures of the chest. In an animal study, only 4% of the energy supplied reached the heart [43]. The average adult human TTI is $\approx 70\text{-}80\ \Omega$ and is determined by multiple factors including energy level, electrode size, interelectrode distance, interface skin-electrode, electrode pressure, phase of ventilation, myocardial tissue and blood conductive properties [44].

When TTI is too high, a low-energy shock will not generate sufficient current to achieve defibrillation [44, 45]. To reduce TTI, the defibrillator operator should use conductive materials. This is accomplished with the use of gel pads or electrode paste [46] with paddles or through the use of self-adhesive pads.

Rischi da contatto durante erogazione shock

Table 1

Self-administered shocks at full capacity. *Abbreviations:* J, joules; N/R, not reported; ROSC, return of spontaneous circulation.

Reference	Victim	Circumstances	Consequences, outcome
Montauk ⁶⁸	23-Year-old male	Paddles placed on chest on top of scrubs, self-administered shock, unknown energy level, immediate unconsciousness and subsequent cyanosis, first recorded rhythm polymorphic ventricular tachycardia apparently with severe haemodynamic compromise	Sustained ROSC not obtained, victim eventually died
	N/R	Paddles placed on the chest, self-administered shock at 360 J, no loss of consciousness	Local burns, no other complications reported
	"Intoxicated" female college student	Paddles placed on chest on top of clothes, self-administered shock, unknown energy level, immediately fell on the floor	Local burns, "uneventful recovery"
Iserson and Barsan ⁶⁹	27-Year-old male	Paddles placed against temples, no direct contact to the skin, self-administered shock at 400 J, immediate collapse, unresponsiveness, disorientation, amnesia, vitally stable	Second degree facial burns, tender burned area over temporal region, as appraised after 2 months only amnesia as sustained complication
Cooper ⁷⁰	9-Year-old male	Paddles laterally next to the head, discharge of unknown energy, immediate tonic-clonic seizures for approximately 30 s, then headache and confusion, circulatory stable	Local burns, discharged after 24 h without evidence of other sequelae
Grumet ⁷¹	35-Year-old male after ingestion of life-threatening imipramine dose	Paddles most likely placed against temples, self-administered shock at 400 J, immediate collapse, unconsciousness for approximately two minutes, then confusion, amnesia, blurred vision, paraesthesia of both arms, later became unresponsive again with need for mechanical ventilation attributed to imipramine overdose	Local burns, discharged after 18 days without evidence of neurological sequelae
Anonymous ⁷²	Nursing assistant	Paddles placed on victims chest during routine defibrillator check, accidental charge and discharge at unreported energy level	Local burns, recurring cardiac arrhythmias, no long-term sequelae reported

Table 2

Electric shocks to healthcare professionals during resuscitation due to equipment failure.

Reference	Circumstances	Consequences, outcome
Gibbs et al. ⁹	Shock to operator during defibrillation due to crack in defibrillation paddles	Victim was administered lidocaine for frequent premature ventricular beats, had muscle cramps in arm and chest for several weeks
	Shock due to spontaneous discharge of the device	"Mild shock"
	Shock from touching the charge button	"Mild shock"

Table 3

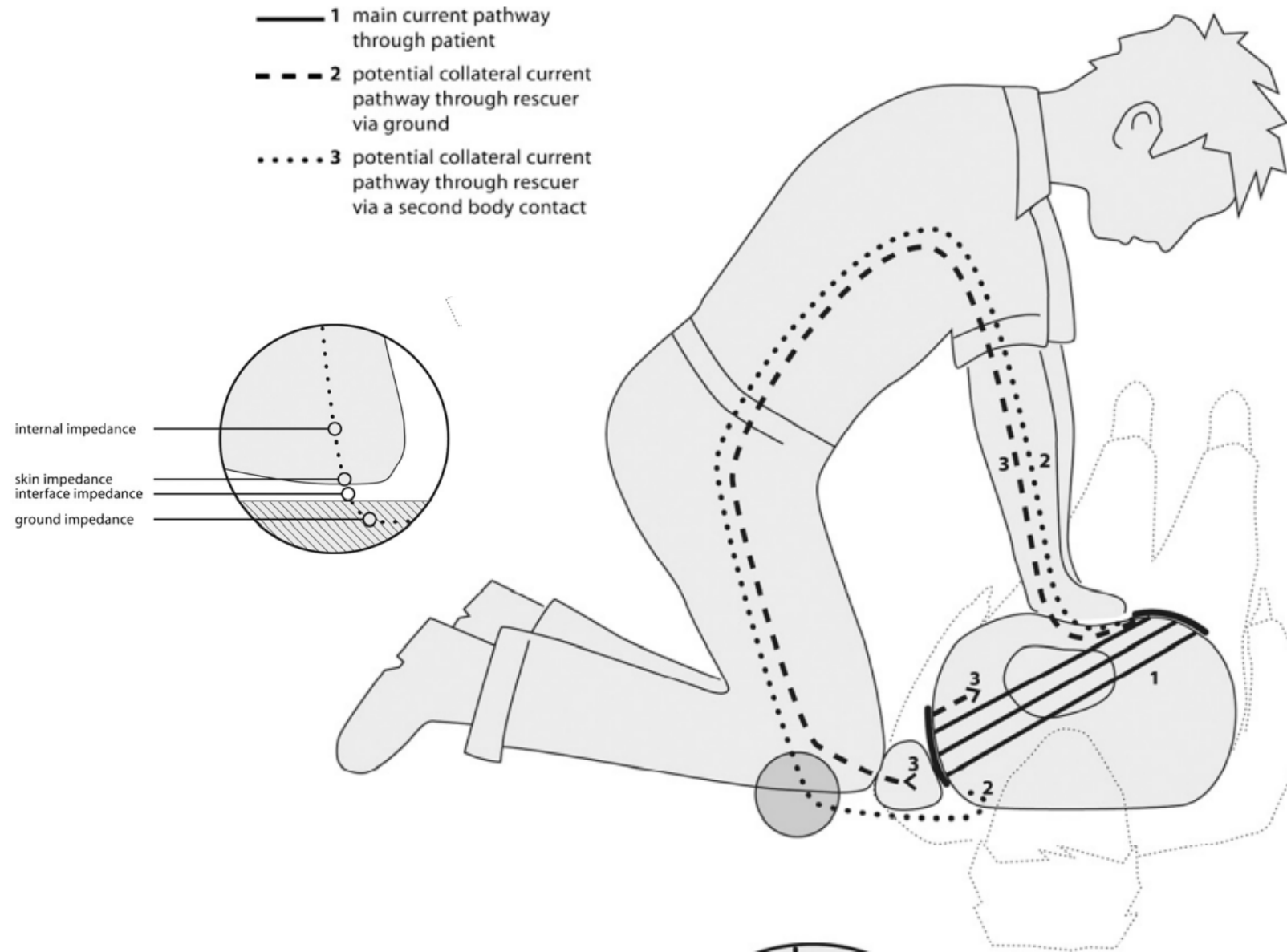
Electric shocks to healthcare professionals during training or device testing. *Abbreviations:* EMT, emergency medical technician; N/R, not reported.

Reference	Circumstances	Consequences, outcome
Gibbs et al. ⁹	Accidental discharge to paramedic during testing	Second degree burn to thigh
	Accidental discharge to trainer during demonstration	"Shock to arms"
	Accidental discharge to technician during testing	"Shock to hand and knee"
Trimble ⁷³	Discharge to EMT during exploration of a new device, paddle position and energy settings N/R	Found "cyanotic and apneic" a few minutes later, had "various ventricular dysrhythmias, including ventricular fibrillation"

Table 4

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



Biomedical consequences of currents through rescuers/bystanders

Twenty-nine cases of electric shock to individuals other than a patient were found in the medical literature. Tingling sensations and minor burns are the medical consequences typically reported. Some of the victims have been admitted to hospital for observation. Obviously, a full capacity discharge to a healthy person can be fatal, and direct discharges to the head may cause severe neurological disturbance. No case report, however, could be identified where medical personnel or bystanders sustained a life-threatening condition or long-term disability of any kind from an accidental electric shock during a medical procedure. In principle, high-voltage discharges are capable of causing arrhythmias, vascular spasm, and vagus nerve stimulation resulting in bradycardia and syncope.³⁵ Except for premature heart beats such complications have not been described so far in accidental shocks during resuscitation. Thus, their significance remains indeterminate. In the study by Lloyd et al. experimental rescuers neither sensed any of the shocks nor did they encounter any medical problems.²⁶

The complication certainly most feared in this regard is induction of cardiac arrhythmia and ventricular fibrillation (VF), respectively, in the rescuer himself.

Fundamentally, VF can be induced by an electric stimulus in phases of myocardial non-uniform refractoriness.³⁶ This phase has been called vulnerable period and extends from about 60 to 90% of the QT-phase on the surface ECG in the normal heart.³⁷ The excitation susceptibility has a bell-shaped distribution though,^{38,39} 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.^{40,41} Stimuli that are just not strong enough to cause VF typically provoke other ventricular arrhythmias.⁴² Vulnerability of the heart is increased after premature ventricular beats, during hypothermia, ischaemia, acidosis, adrenergic stimulation, or rapid pacing and ventricular tachycardia, respectively.^{43–51} Biphasic impulses are generally less effective at inducing VF than monophasic shocks.⁵² For external shocks, the least shock energy is required if the negative electrode is placed over the apex of the heart.^{53–55} Experiments to define VF threshold for external currents have mainly been performed with household power, i.e. 50–60 Hz sinusoidal AC, in animals. It was demonstrated that fibrillation threshold increases with body weight and is less for currents applied in ECG leads II and III as compared to lead I orientation.⁵⁶ The more 60 Hz AC cycles applied the higher the likelihood of VF occurrence, and up to about 120 cycles the fibrillation threshold decreases with the number of cycles applied.⁵⁷ Extrapolating from animal experiments, for 50/60 Hz AC of four to 20 ms duration, representing one complete AC cycle or less, travelling from hand to feet, fibrillation threshold has been set to 500 mA root mean square (RMS) current by the International Electrotechnical Commission (IEC) and to 300 mA by Underwriters Laboratory.²⁵ In a very cautious scenario with a total body impedance of 500 Ω (see

above) this requires RMS voltages of 250 V and 150 V, respectively, directly applied to the bare skin, i.e. with path resistances outside the victim's body not taken into account. Caution is generally advisable; experience with neuromuscular incapacitating devices (stun guns) has shown that extrapolation from animal experiments or theoretical models can be misleading. While theory suggested that stun guns should be incapable of stimulating the human heart,^{58,59} practice and sound consecutive experiments have disproved this paradigm.^{60,61}

Experiments in humans to determine fibrillation current for short-duration external shocks have never been undertaken. Damped sinusoidal shocks at 10 J applied through adhesive defibrillation pads at the peak of the T wave have been shown to induce VF in patients prior to implantation of implantable cardioverter-defibrillators.⁶² The corresponding current or voltage was not reported. Some insight can be derived from external cardiac pacing studies. Monophasic rectangular or truncated exponential pulses, at pulse widths of 20 ms, applied transcutaneously via large adhesive electrodes in an ideal left-thoracic anterior–posterior position capture the ventricles at a mean current of around 75 mA.^{53,63–65} In healthy volunteers capture thresholds as low as 40 mA have been reported.⁶⁴ The current that precipitates ventricular tachycardia or VF is at least five times greater.^{66,67} This 'safety factor' for external pacing has been confirmed in numerous animal studies for pulse durations from 0.5 to 50 ms.^{38,55} In conclusion, again 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 safe.

Conclusions

Evident in the medical literature, to the best of our knowledge, no rescuer or bystander has ever been seriously harmed by receiving an inadvertent shock while in direct or indirect contact with a patient during defibrillation. New evidence suggests that it might even be electrically safe for the rescuer to continue chest compressions during defibrillation if self-adhesive defibrillation electrodes are used and examination gloves are worn. According to recent data a continuous compression strategy might even improve outcome after resuscitation from cardiac arrest. Before defibrillation safety recommendations are changed, however, more definite data are needed to make absolutely sure that there is no risk. Such studies must address various scenarios including hand-held defibrillation electrodes, no gloves situations, wet and metal surfaces, light-weight rescuers, persons holding infusions, maximum defibrillation energy, and resuscitation scenarios within confined space.²⁰ Interpretation of the data must take into account the significant knowledge gap as regards fibrillation thresholds for humans with defibrillator discharge impulses.

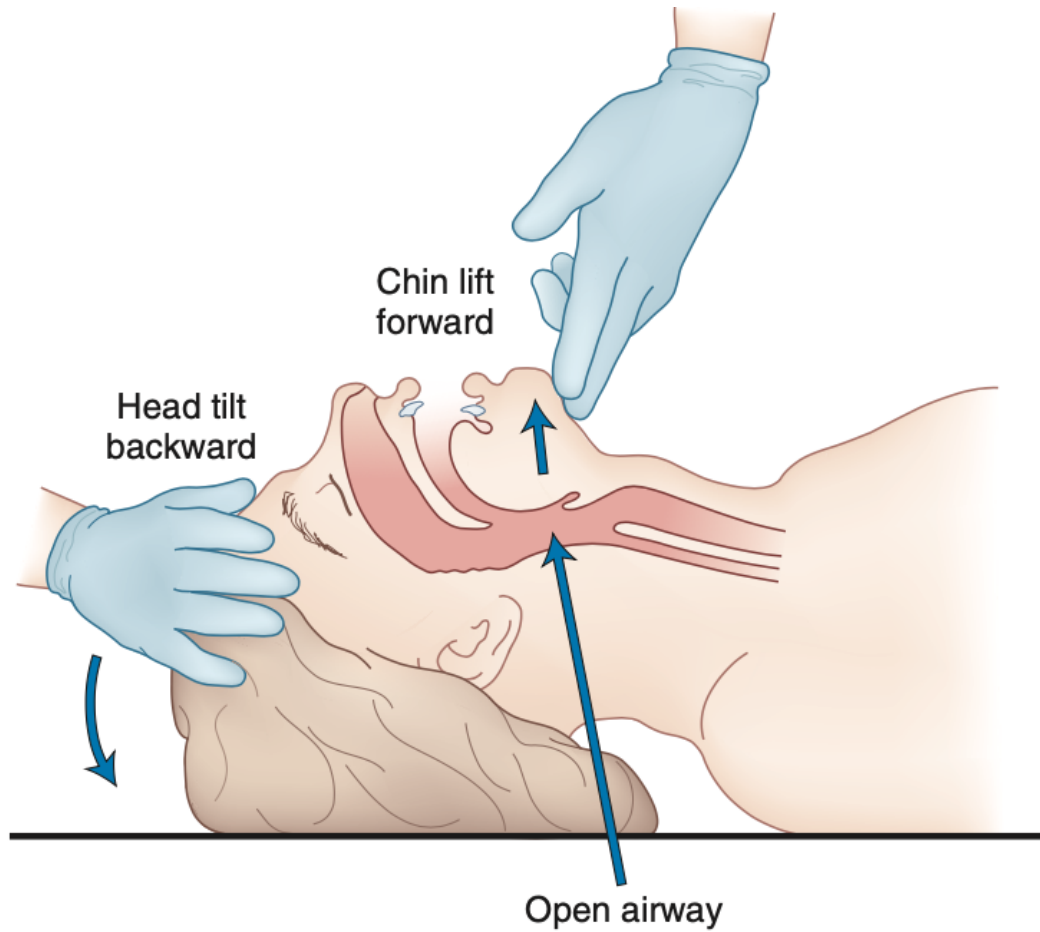
Sicurezza ambientale



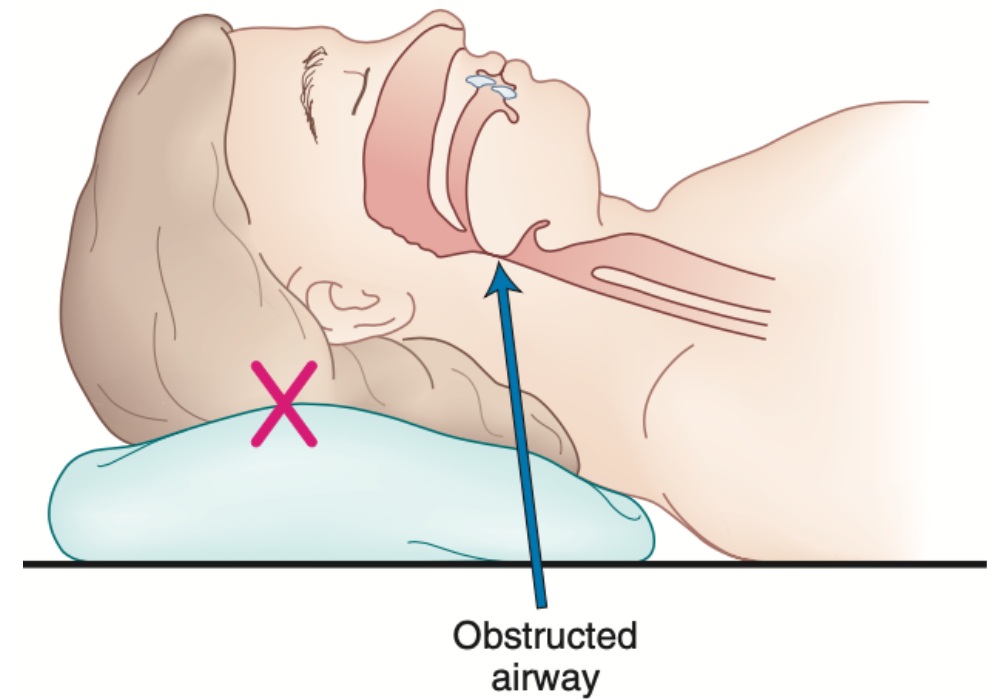




Gestione delle vie aeree



B Head tilt and chin lift to obtain extended position



Tongue in apposition to posterior pharyngeal wall

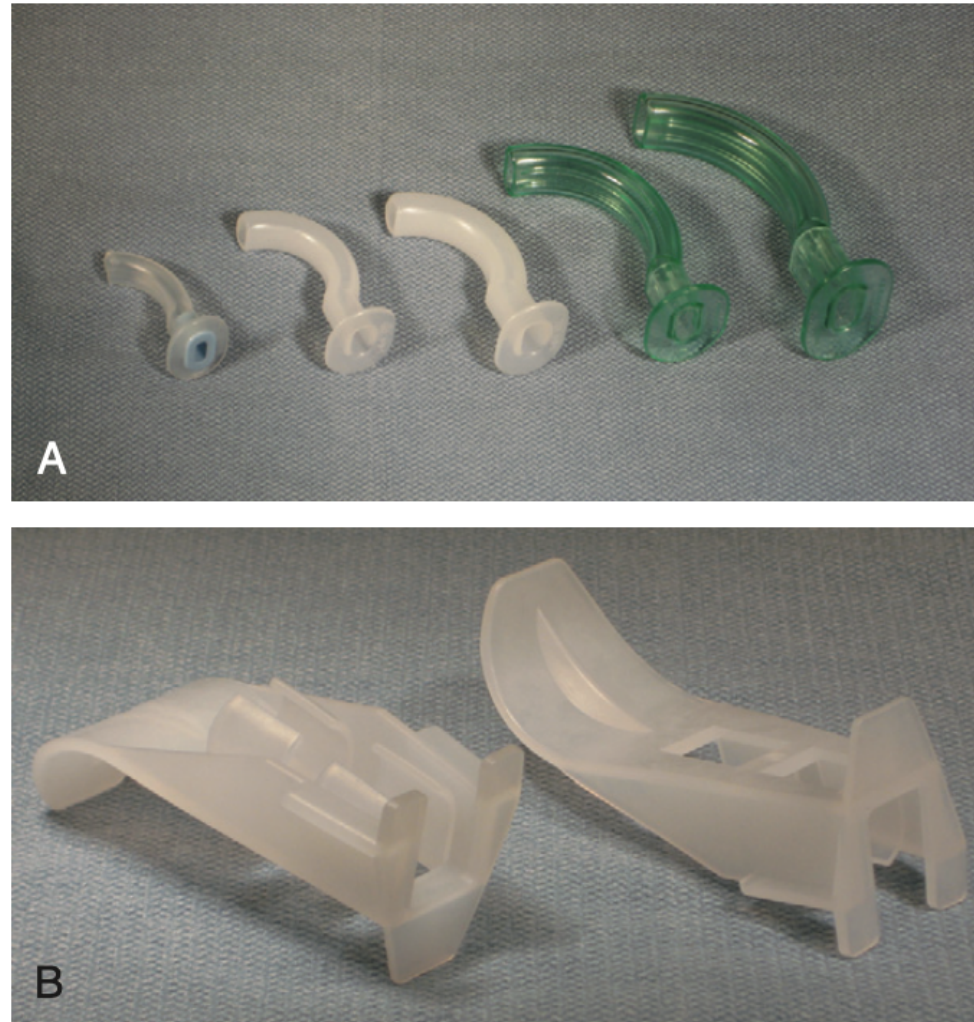


Figure 15-7 Oropharyngeal airways. **A**, Guedel Airways in sizes from neonatal to large adult. **B**, The Ovassapian Airway has a large anterior flange to control the tongue. The airway is open posteriorly (including no posterior flange) so that an endotracheal tube can be inserted with a flexible fiberoptic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



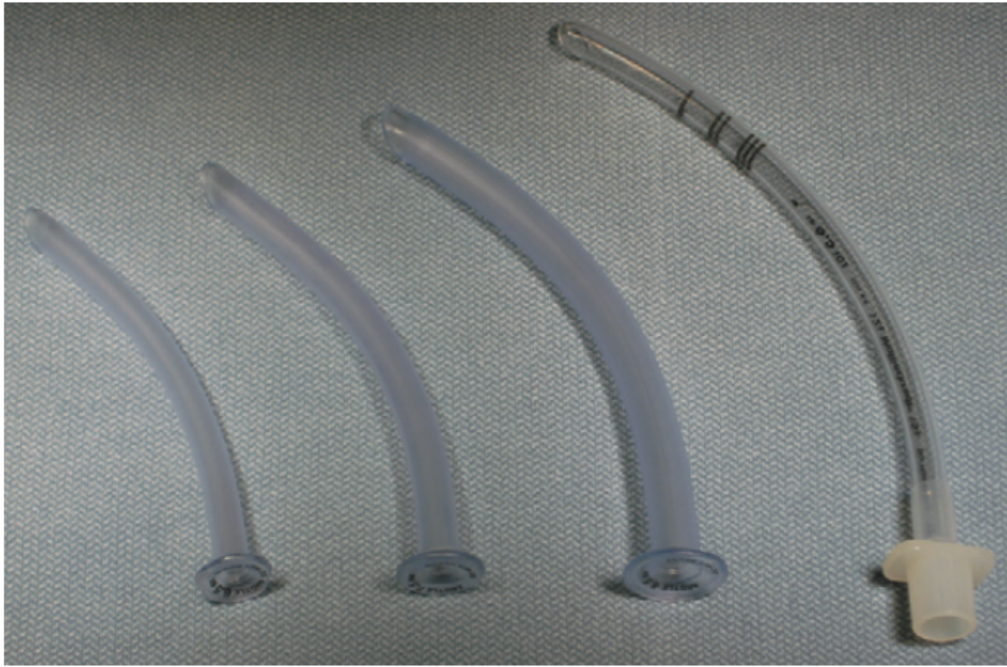


Figure 15-9 Nasopharyngeal airways. A flange prevents the outside end from passing beyond the nares, controlling the depth of insertion. Alternatively, an endotracheal tube may be cut down to provide a longer airway, with its 15-mm adapter reinserted in the cut end.



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.



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.

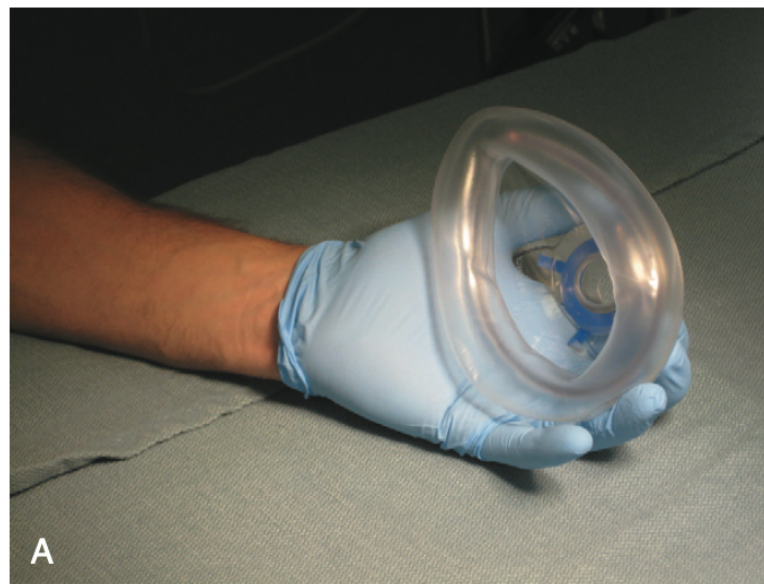


Figure 15-13 Suggested techniques for holding and supporting a face mask. **A**, In the proper hand grip of the face mask, the thumb and index finger encircle the collar while the hypothenar eminence extends below the left side of the mask. **B**, In the side view of the standard one-handed application 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.



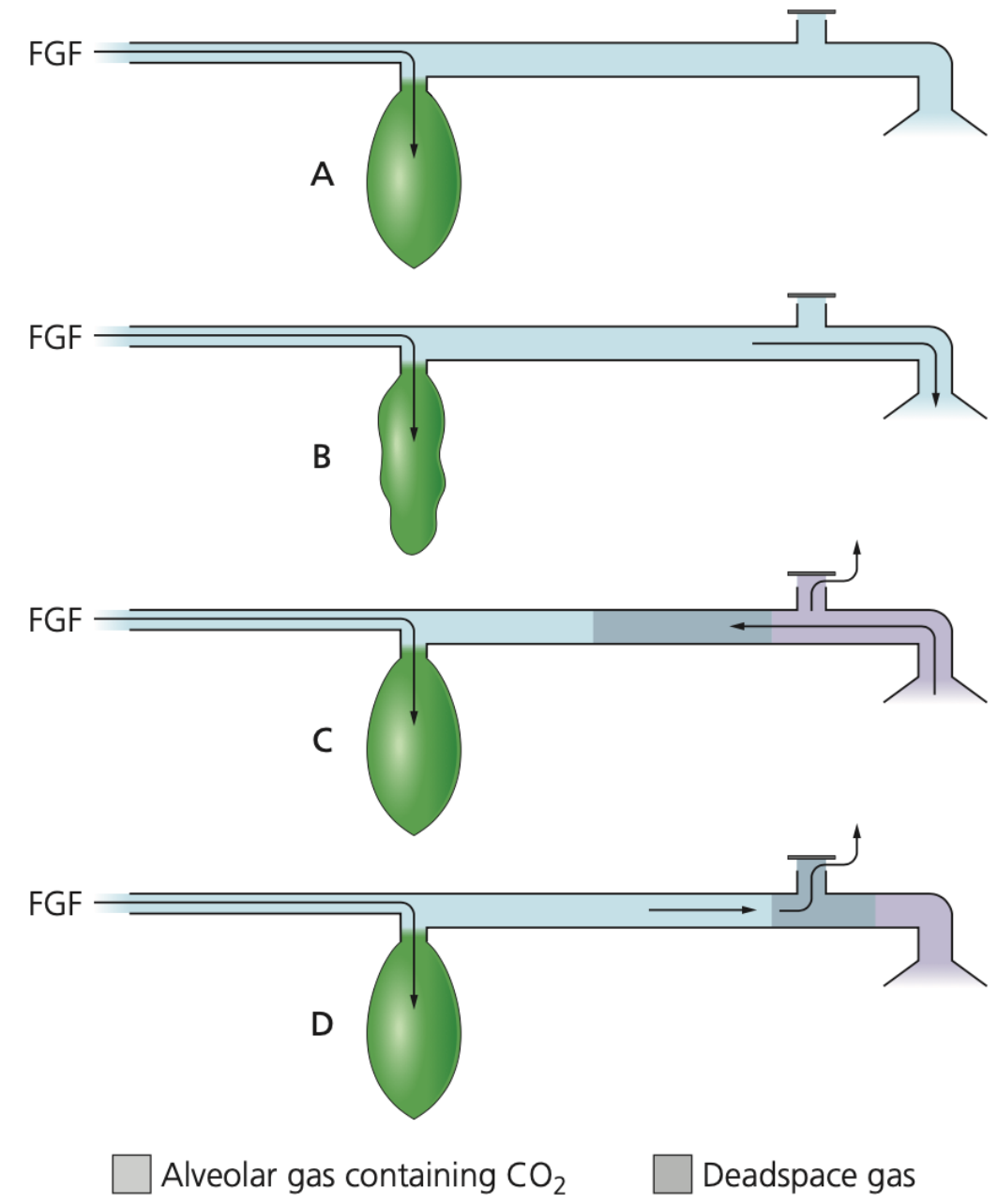
Figure 15-15 Two-hand control of a face mask. In both scenarios, a second provider must ventilate the patient. **A**, In the view of two-hand control of a face mask from above the patient, notice how the lower fingers on both hands apply a jaw thrust while the thumbs seal the mask to the face. **B**, 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.



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.



Fig. 4.8 Intersurgical adult Mapleson C system.



BOX 16-1 **Benefits of Endotracheal Intubation**

1. A patent airway by oral, nasal or tracheal routes
2. Controlled ventilation with up to 100% oxygen
3. Ventilation with high airway pressure
4. Airway protection from aspiration
5. Removal of secretions
6. Lung isolation
7. Administration of medication including anesthetic gases

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

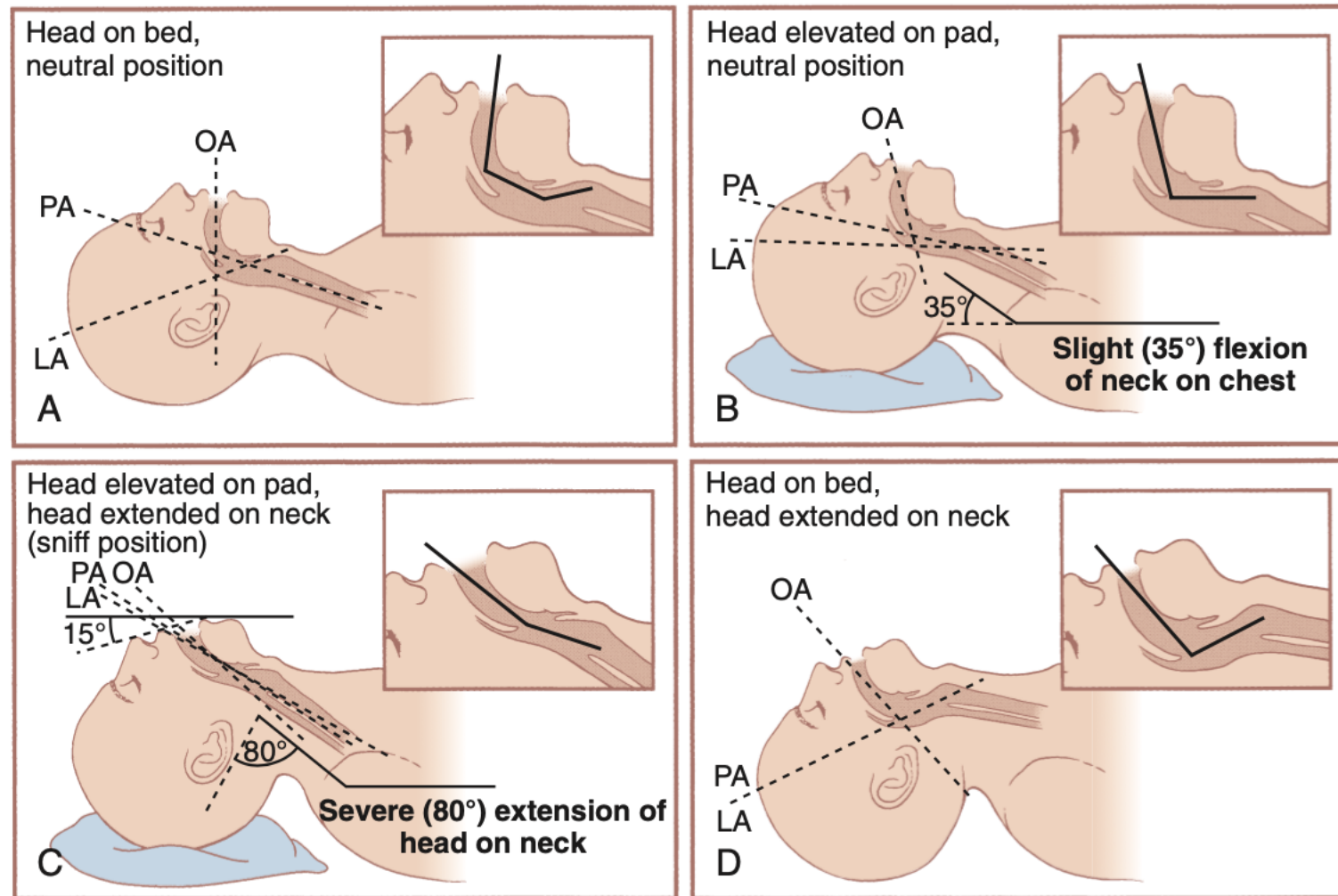


Figure 17-1 Schematic diagrams show the alignment of the oral axis (OA), pharyngeal axis (PA), and laryngeal axis (LA) in four different head positions. Each head position is accompanied by an *inset* that magnifies the upper airway (oral cavity, pharynx, and larynx) and superimposes (*bent bold line*) 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.)

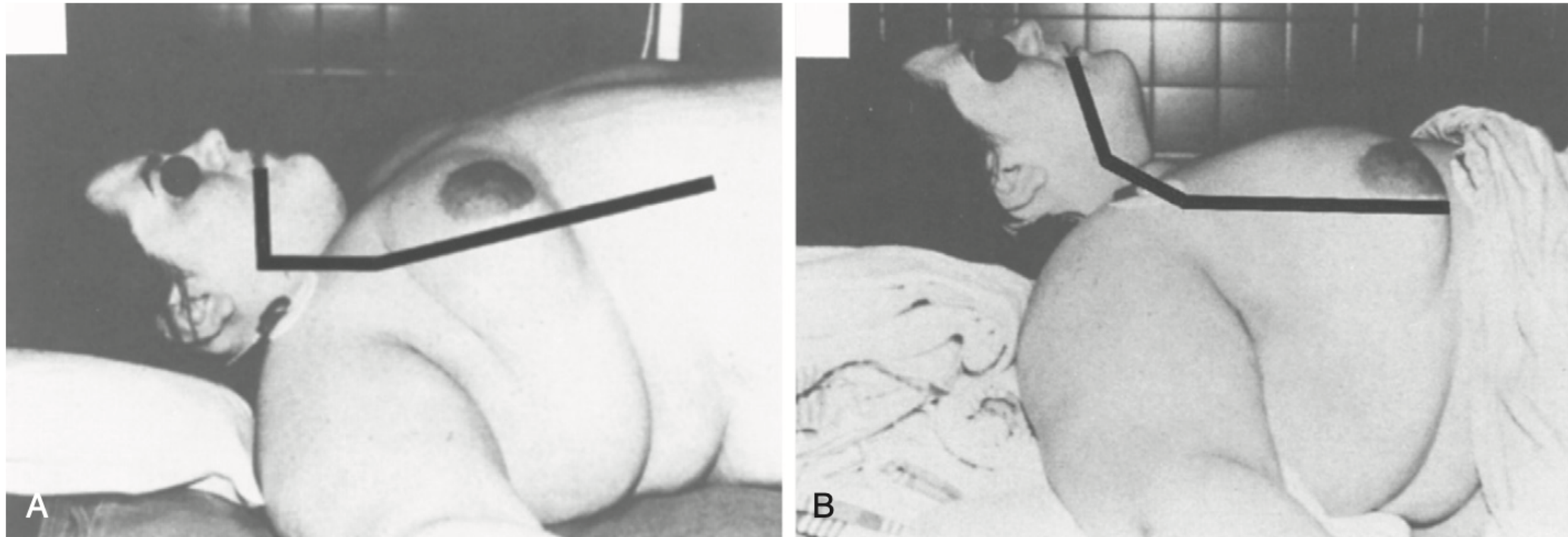
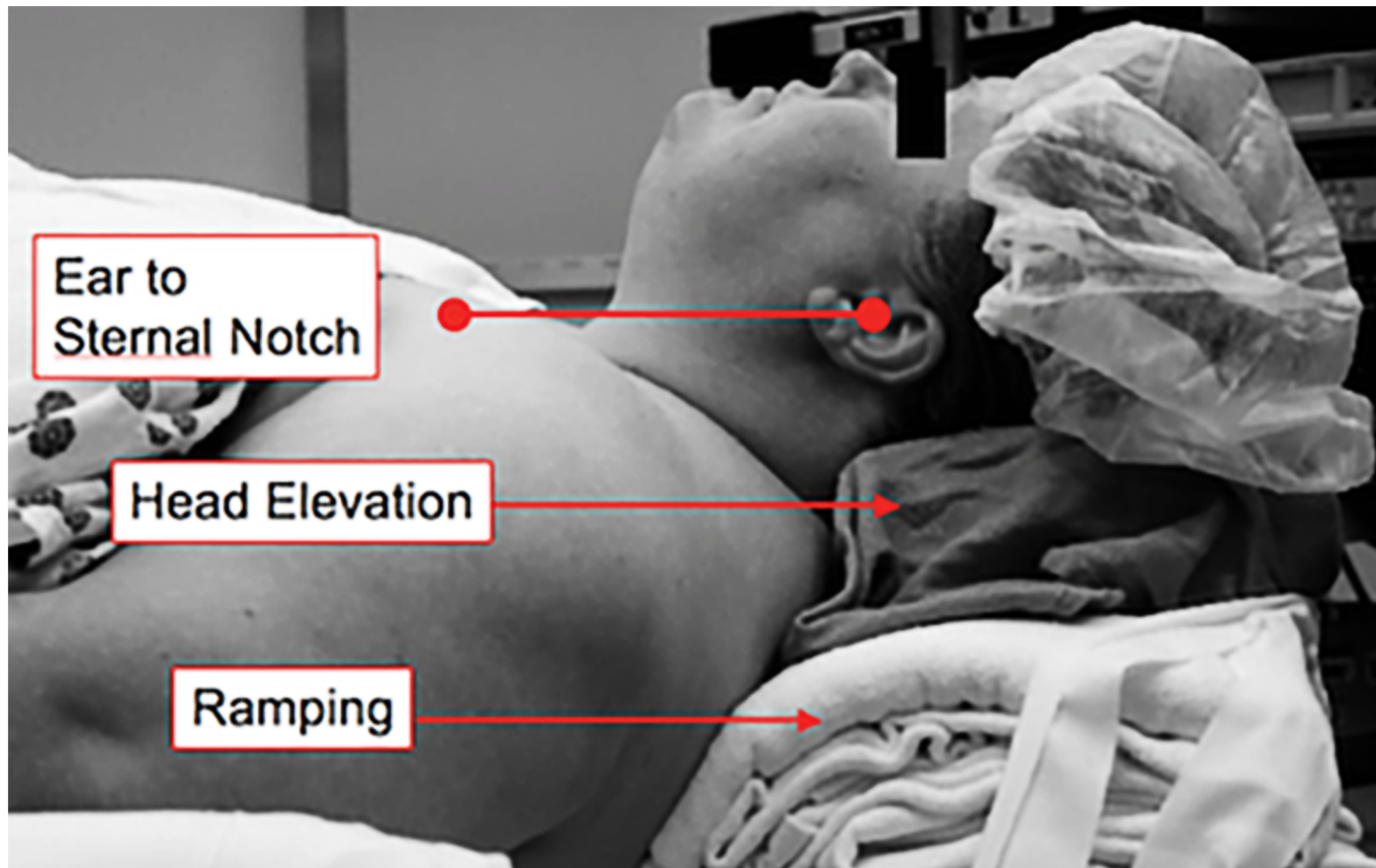


Figure 17-2 **A**, In some obese patients, placing the head on a pillow does not result in the sniffing position; in the obese patient shown and as illustrated by the overlying *bold black line*, the oral and laryngeal axes are perpendicular to one another, the neck is not flexed on the chest, and the 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.)



Ear to
Sternal Notch

Head Elevation

Ramping

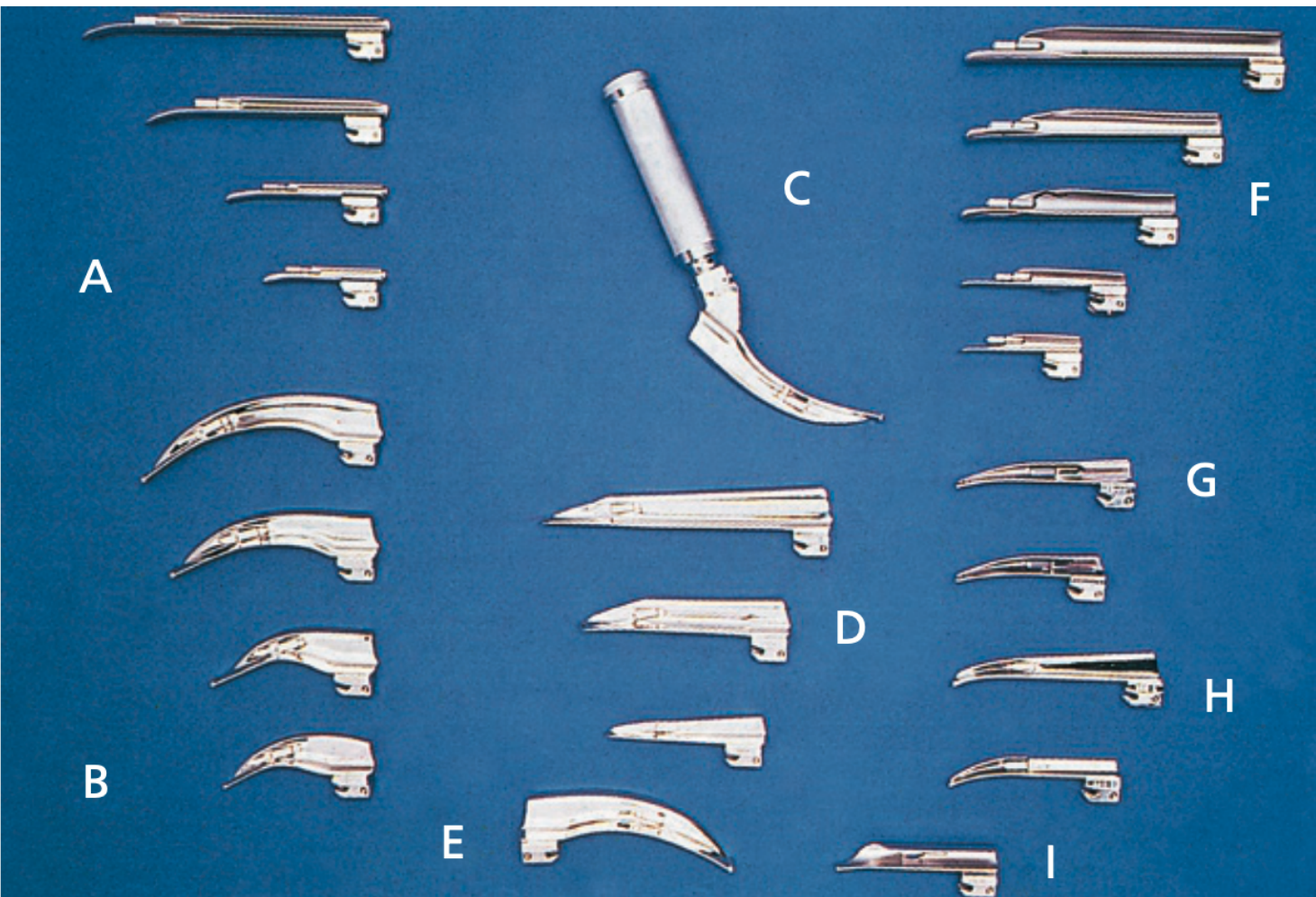
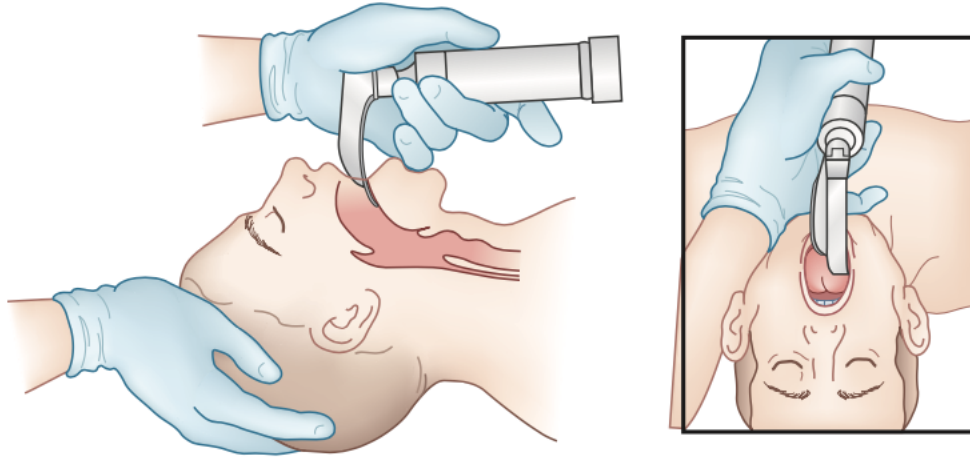


Fig. 7.2 A wide range of laryngoscope blades. (A) Miller blades (large, adult, infant, premature); (B) Macintosh blades (large, adult, child, baby); (C) Macintosh polio blade; (D) Soper blades (adult, child, baby); (E) left-handed Macintosh blade; (F) Wisconsin blades (large, adult, child, baby, neonate);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

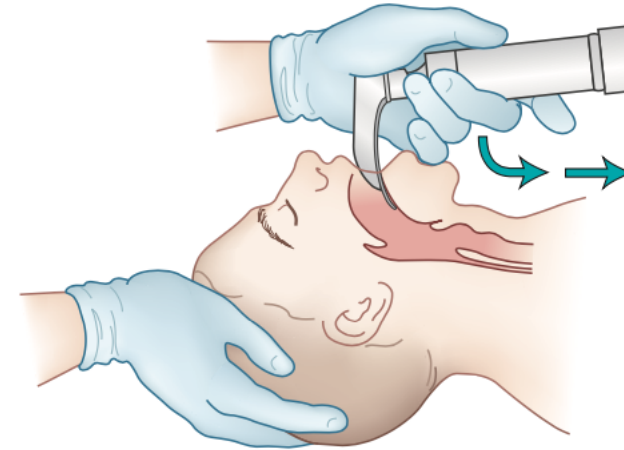


Fig. 7.6 Demonstrating the McCoy laryngoscope's hinged blade tip.

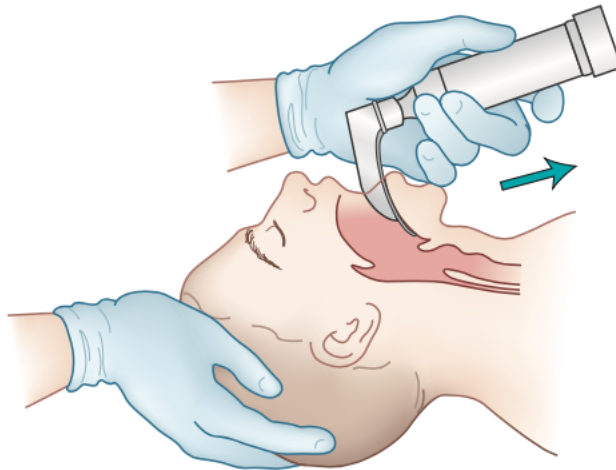
Conventional Laryngoscopy with a Curved Blade



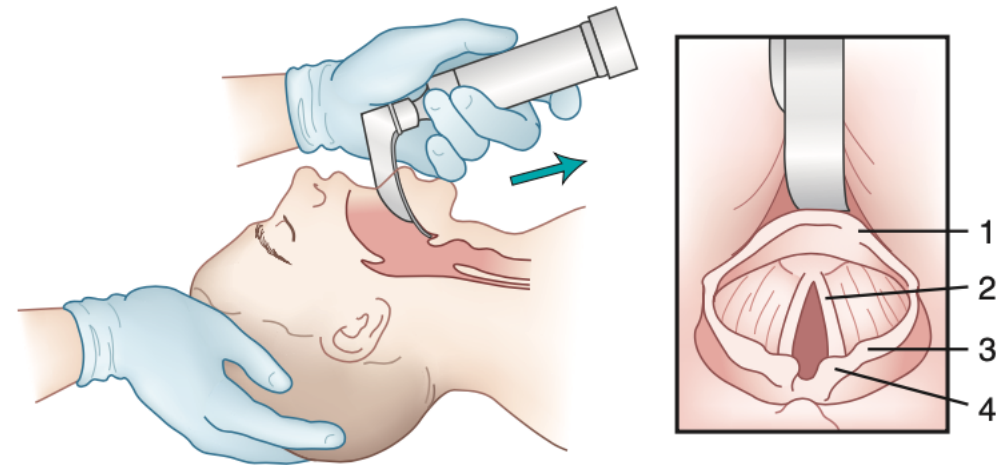
A Insert the laryngoscope blade into the right side of the mouth



B Advance the laryngoscope blade toward the midline of the base of the tongue by rotating wrist



C Approach the base of the tongue and lift the blade forward at a 45° angle



D Engage the vallecula and continue to lift the blade forward at a 45° angle



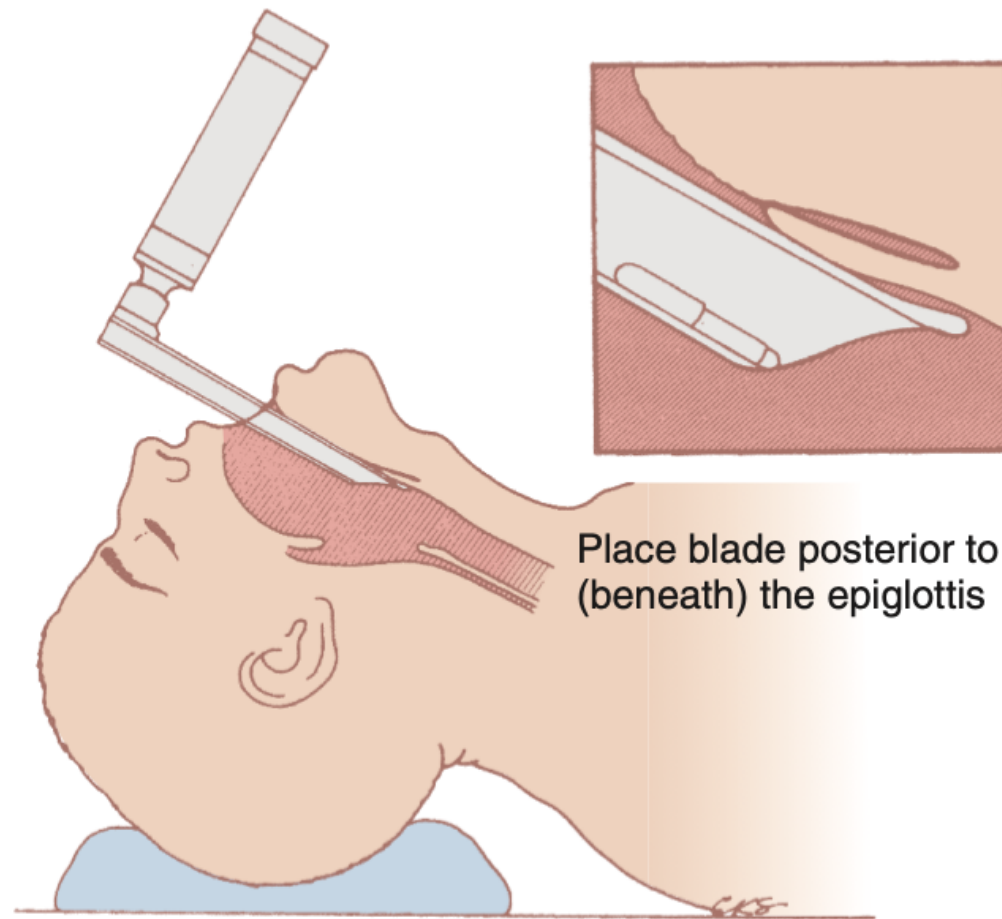
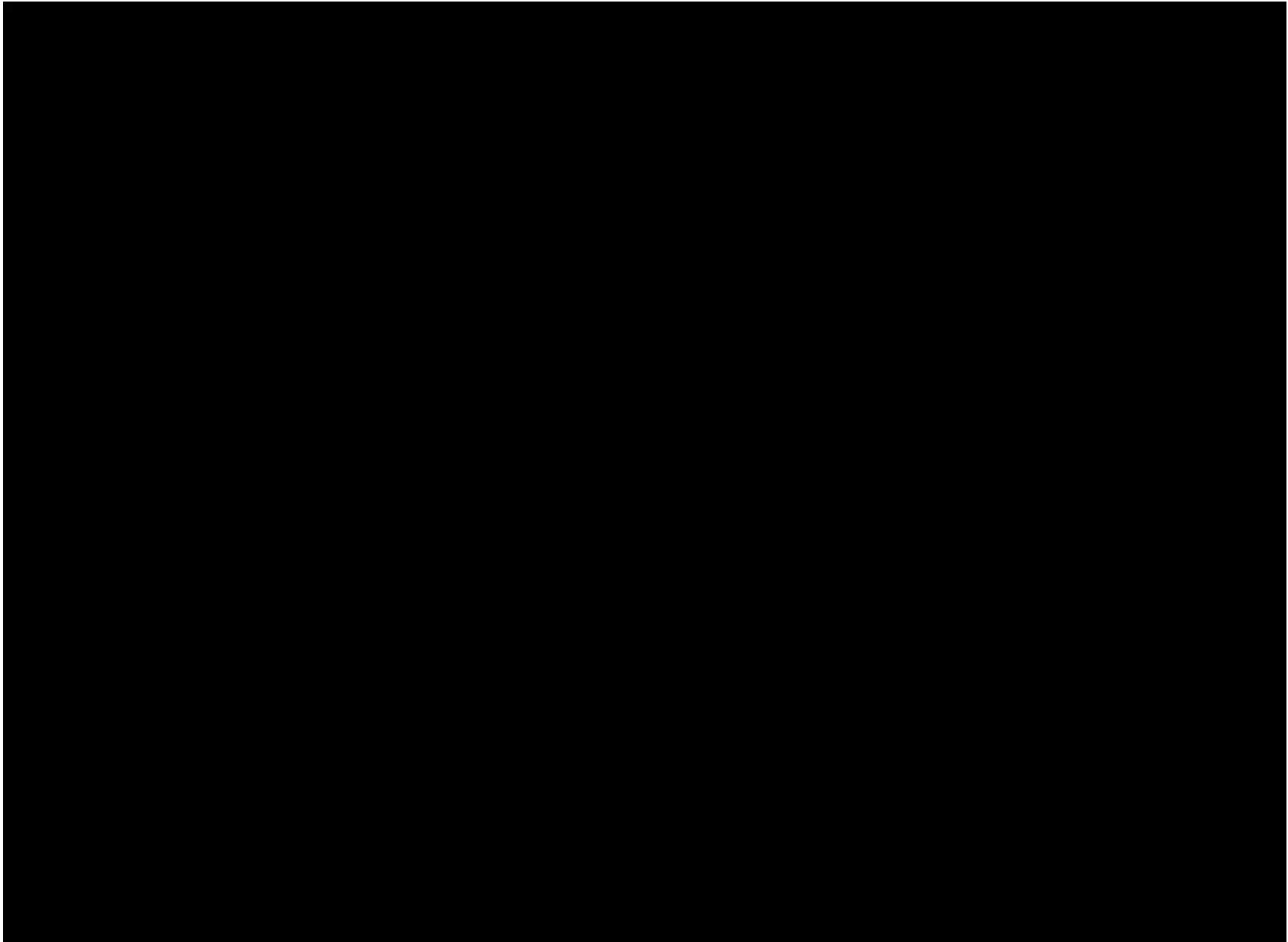


Figure 17-6 Conventional laryngoscopy with a straight blade. A straight laryngoscope blade (Miller blade) should be passed underneath the laryngeal surface of the epiglottis. The handle of the laryngoscope then should be elevated at a 45-degree angle, similar to the lifting that takes place with the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



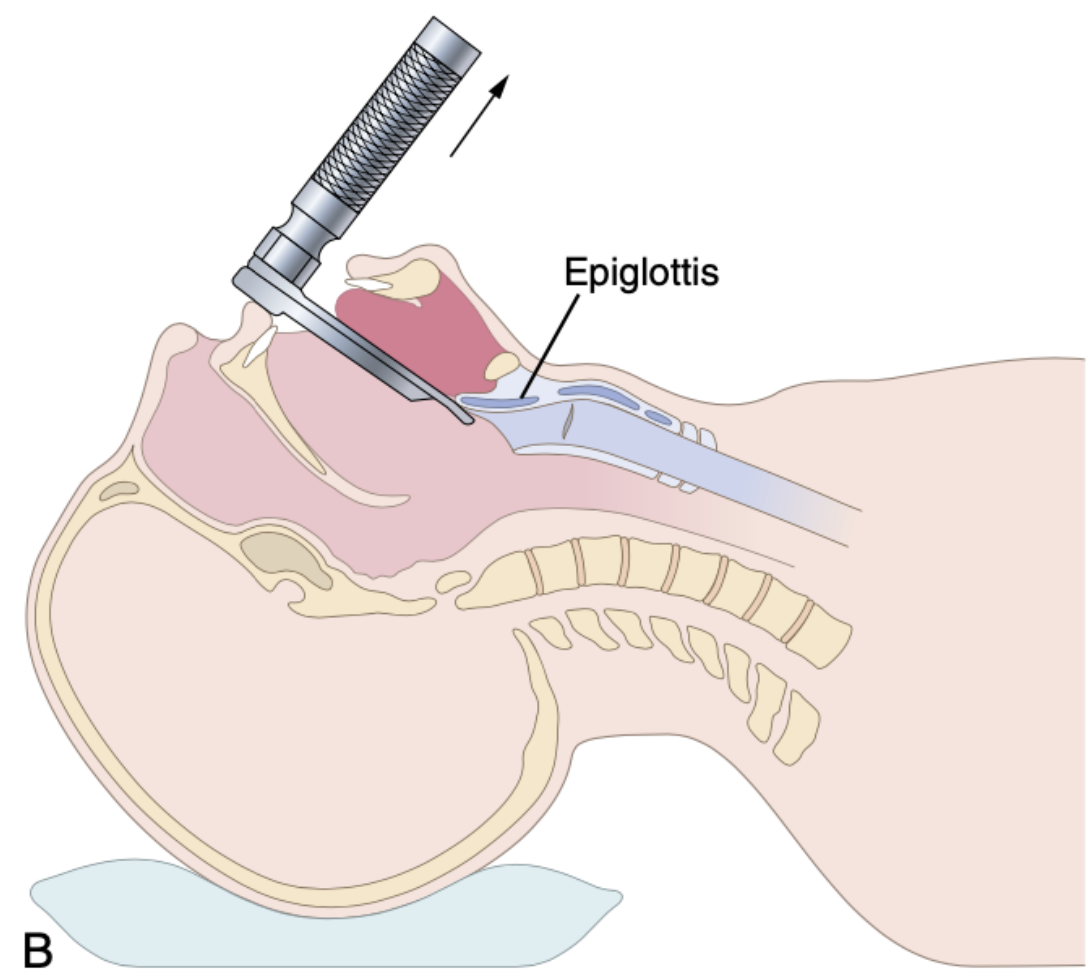
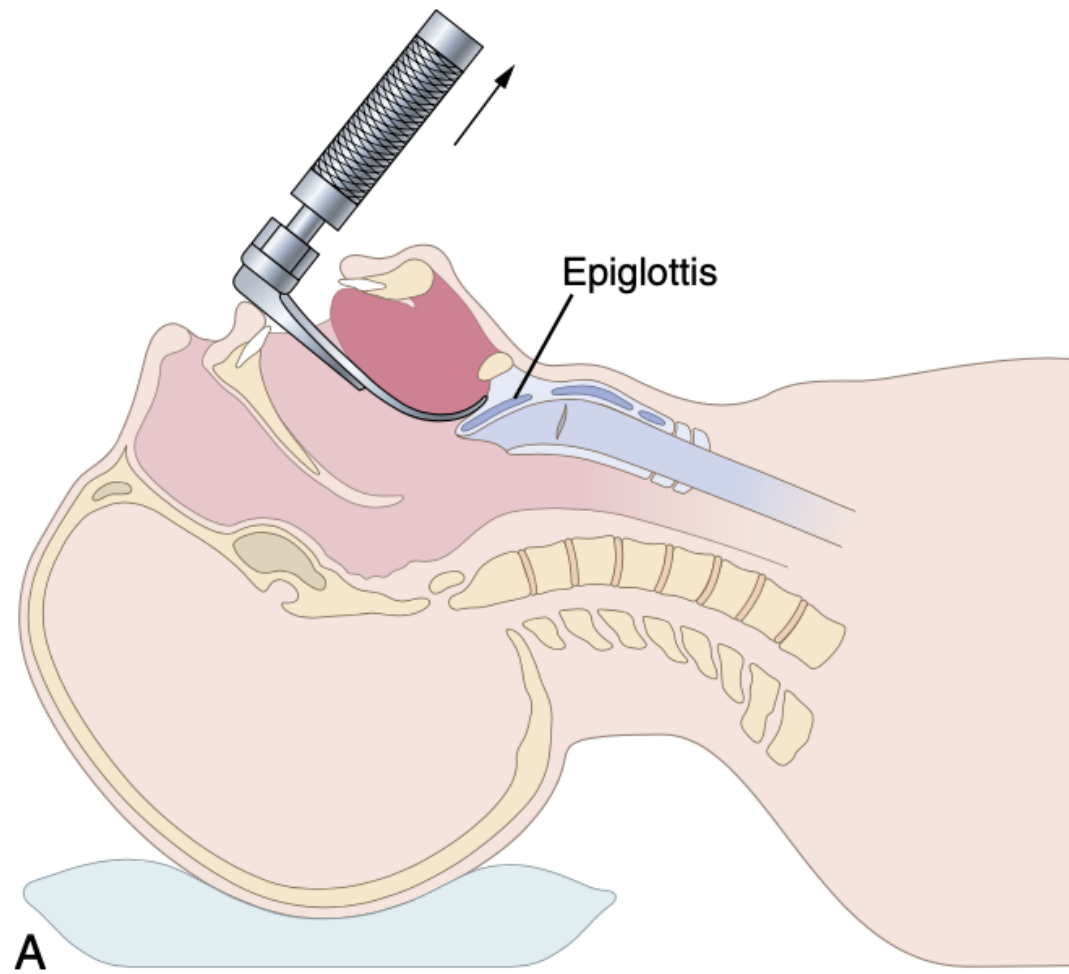


Fig. 16.13 Schematic diagram depicting the proper position of the laryngoscope blade for exposure of the glottic opening. (A) The distal end of the curved blade is advanced into the space between the base of the tongue and the pharyngeal surface of the epiglottis. (B) The distal end of the straight blade 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.

Guiding a nasotracheal tube into the larynx using a Magill forceps

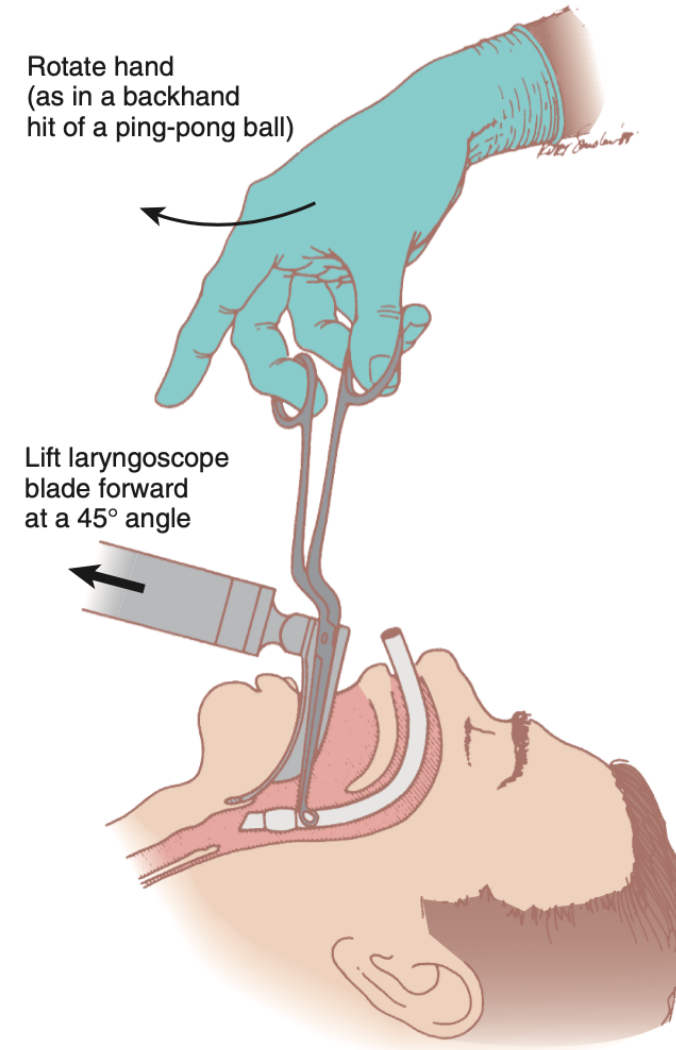


Figure 17-12 A nasotracheal tube can be guided under direct vision (laryngoscopic control) through the laryngeal aperture with a Magill forceps by rotating the hand as when using a backhand motion to hit a ping pong ball. The Magill forceps should grab the nasotracheal tube proximal to the cuff of the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



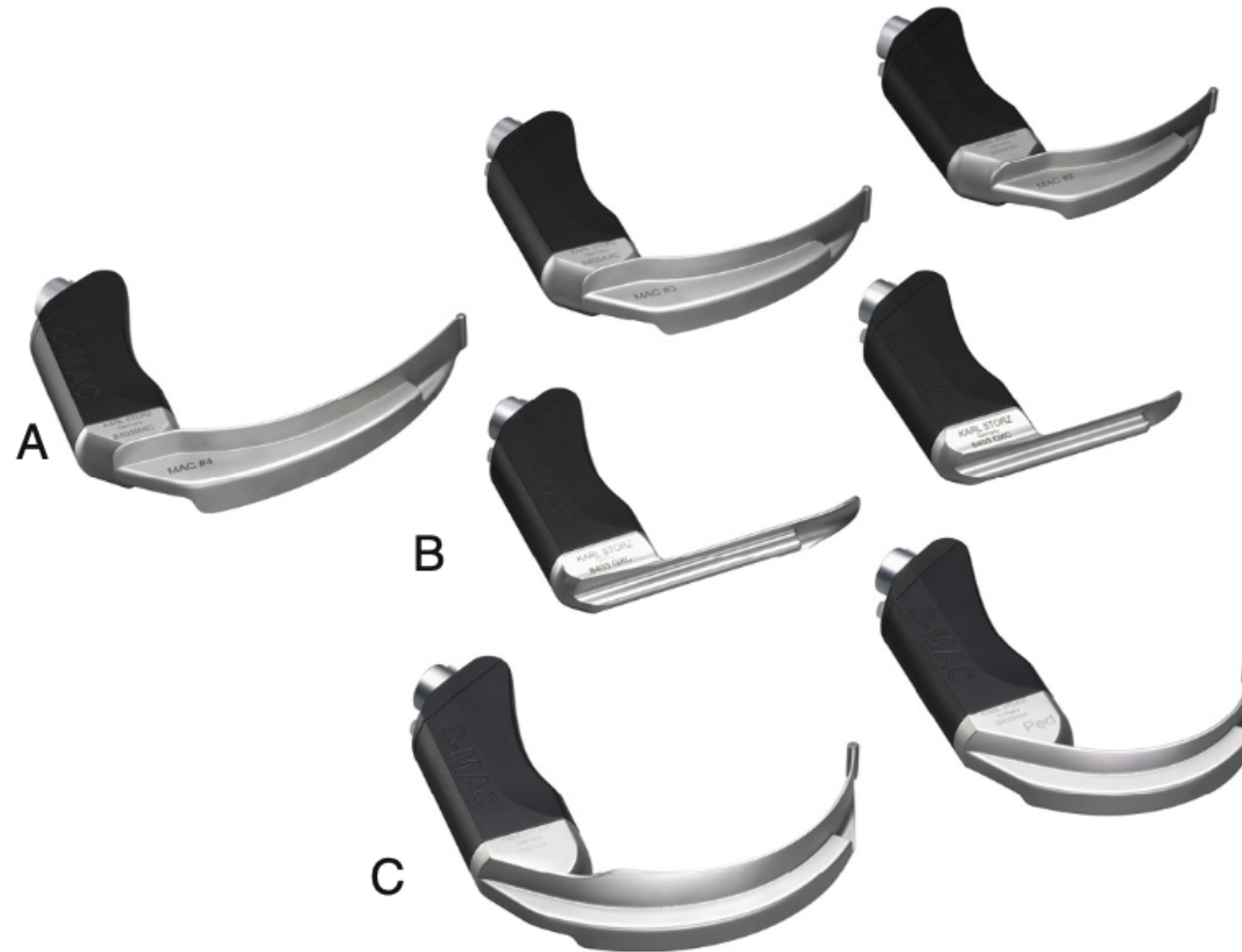


Fig. 16.16 Comparison of the different C-MAC blade types. (A) Macintosh style blade, (B) Miller style blade, and (C) D-blade. (Images courtesy of KARL STORZ Endoscopy, El Segundo, CA.)

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

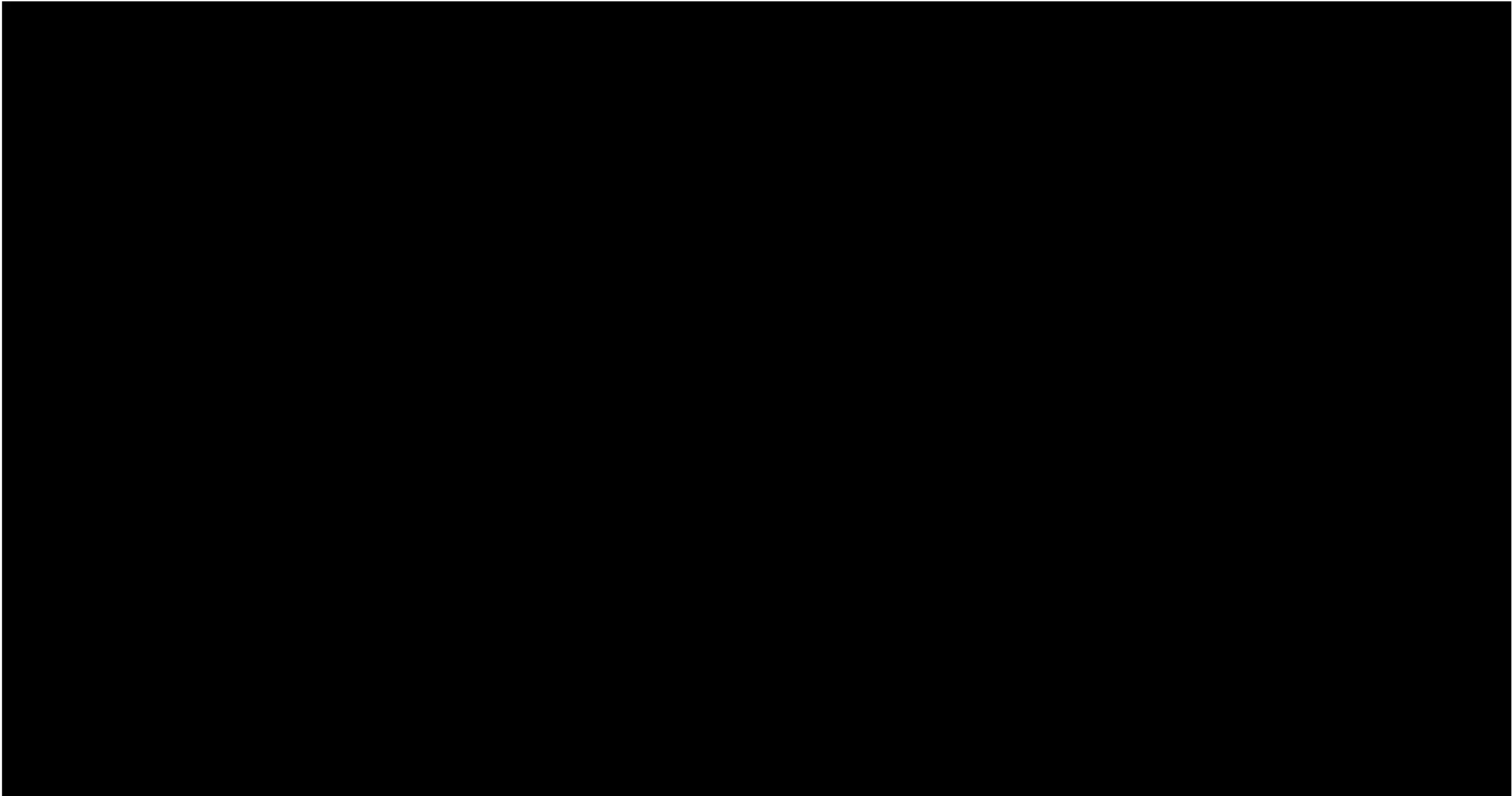
While the Endotracheal Tube Is in Place

- 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

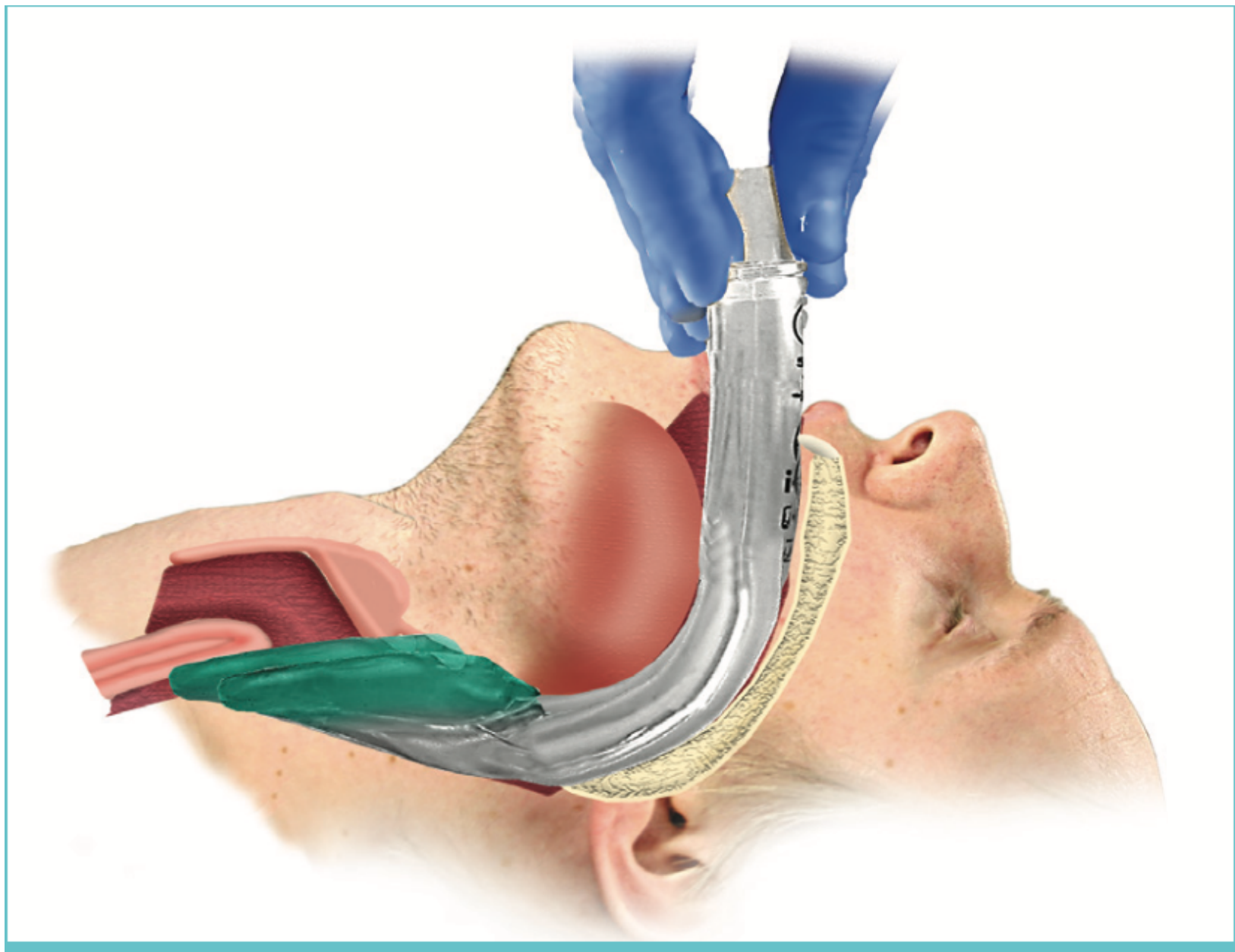
- Laryngospasm
- Inhalation (aspiration) of gastric contents
- Pharyngitis (sore throat)
- Laryngitis
- Laryngeal or subglottic edema
- Laryngeal ulceration with or without granuloma formation
- Tracheitis
- Tracheal stenosis
- Vocal cord paralysis
- Arytenoid cartilage dislocation





FOB






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

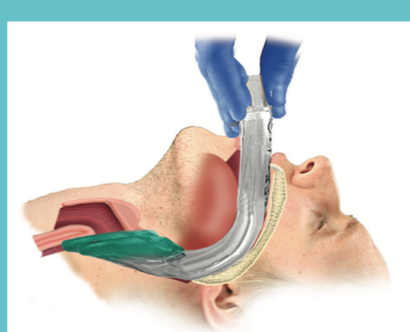
Insertion technique

6



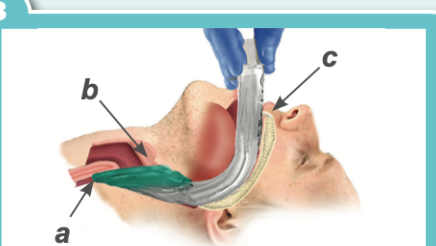
Remove the **i-gel** from the protective cradle. Grasp the lubricated **i-gel** firmly along the integral bite block. Position the device so that the **i-gel** cuff outlet is facing towards the chin of the patient. The patient should be in the 'sniffing the morning air' position with head extended and 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



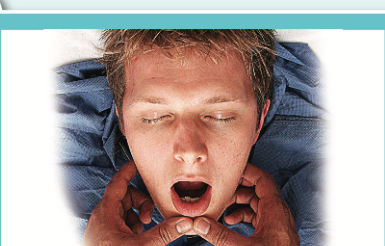
The tip of the airway should be located into the upper oesophageal opening (a) and the cuff should be located against the laryngeal framework (b). The incisors should be resting on the integral bite-block (c).

9

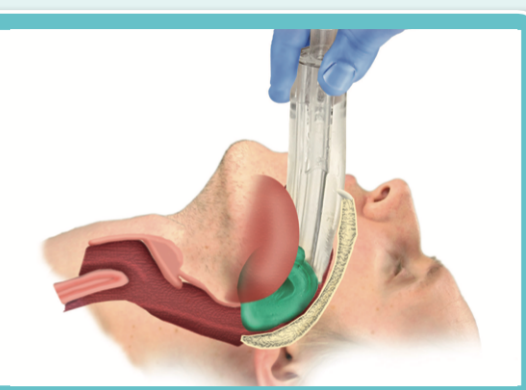


i-gel should be taped down from 'maxilla to maxilla'.

10



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



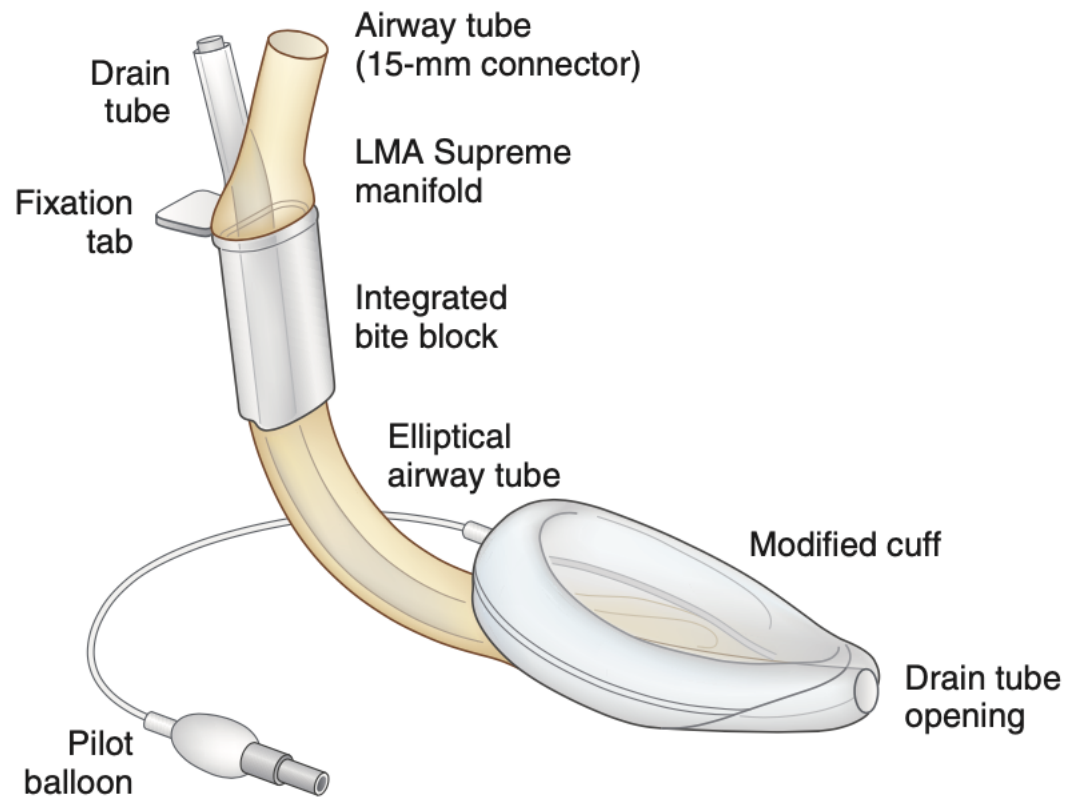


Figure 22-24 The LMA Supreme has a manifold with an integral bite block, an anatomically shaped airway tube enclosing a drain tube, a modified cuff through which the drain tube passes, and a cuff inflation line with pilot balloon.

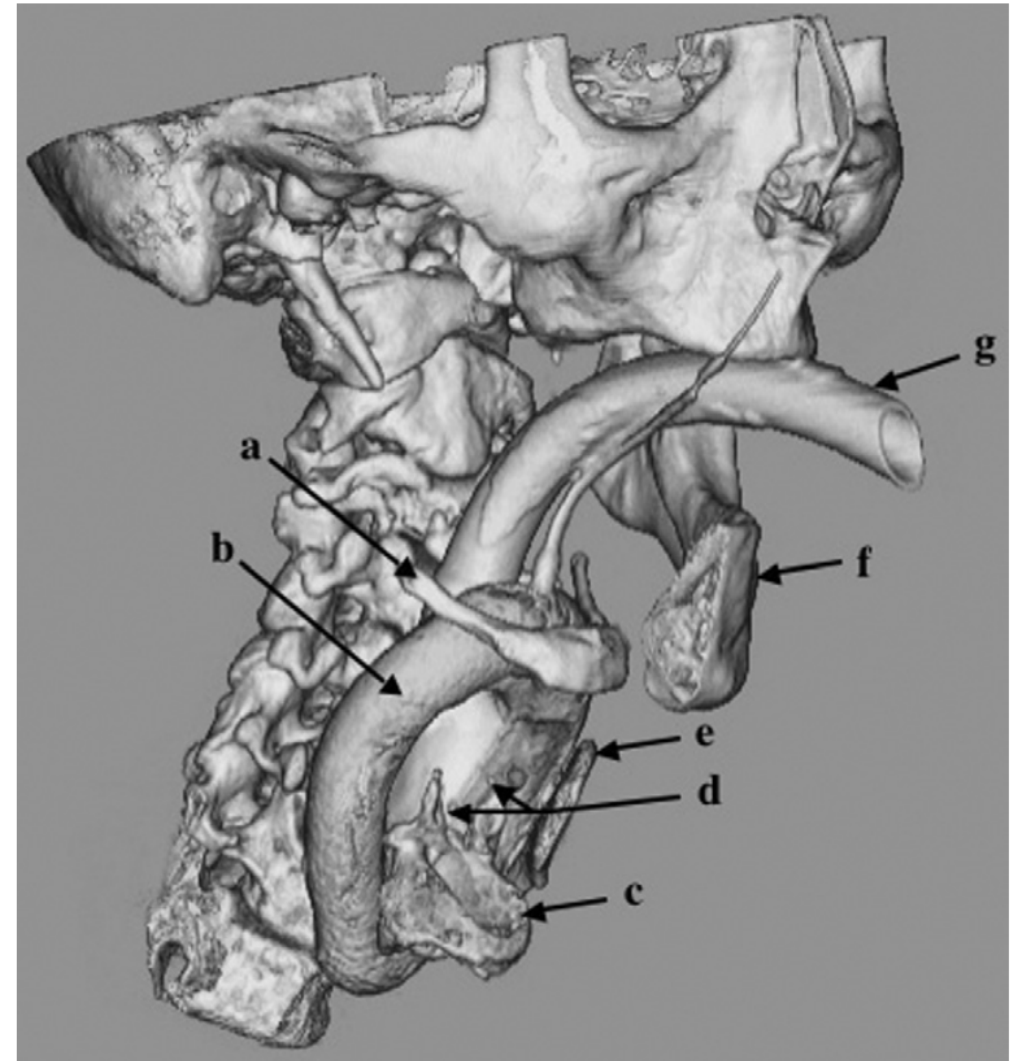
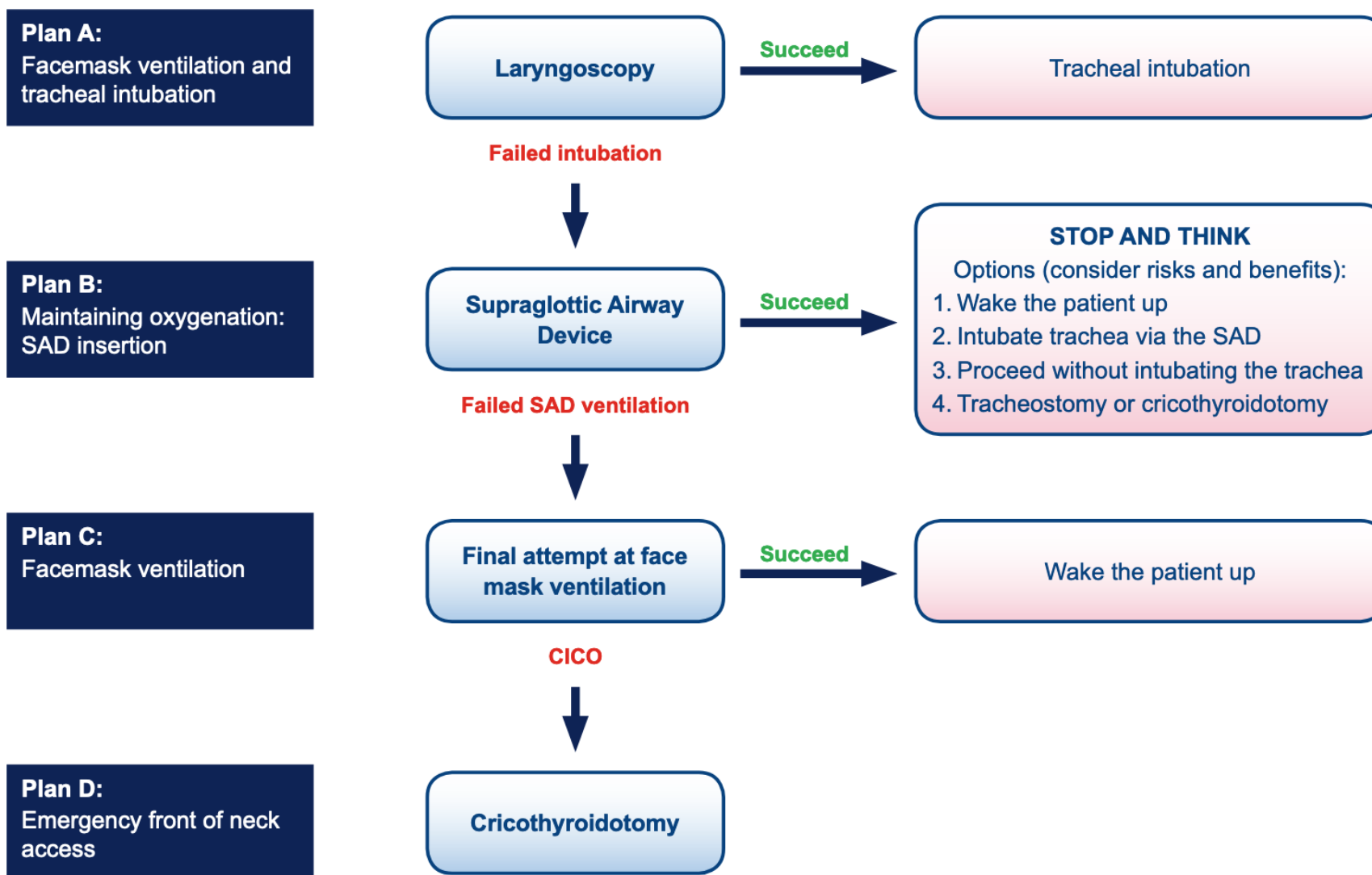
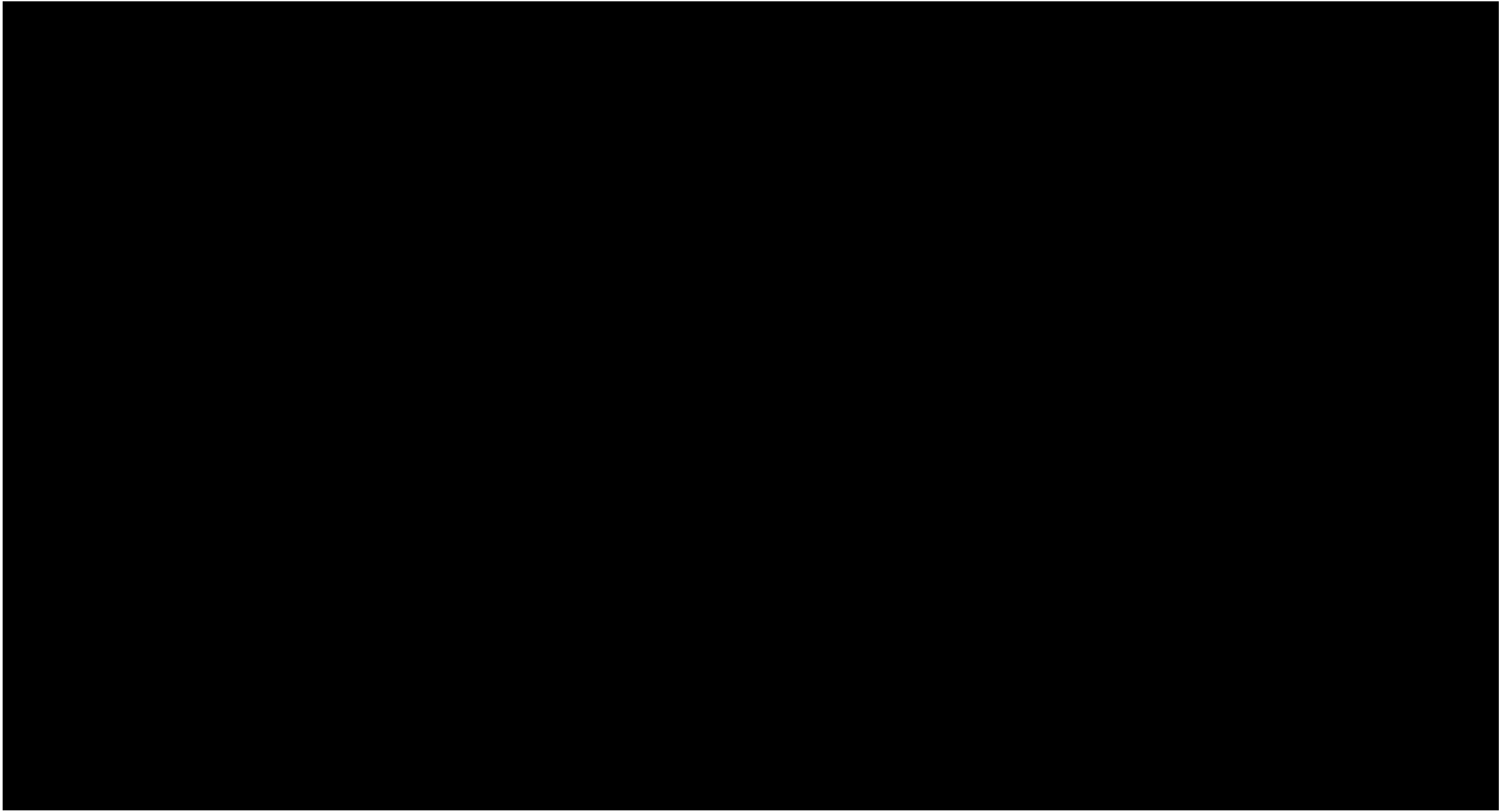
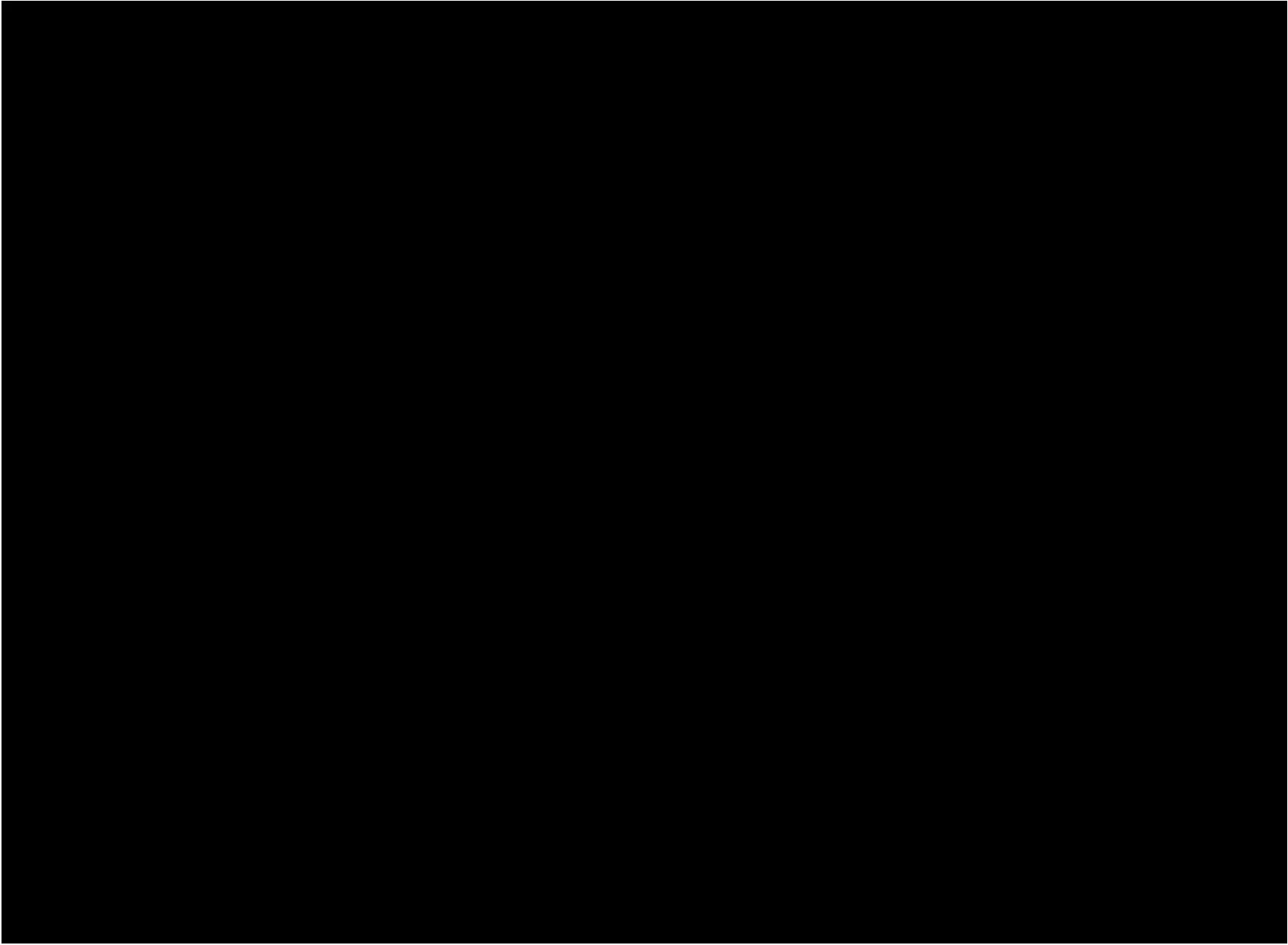


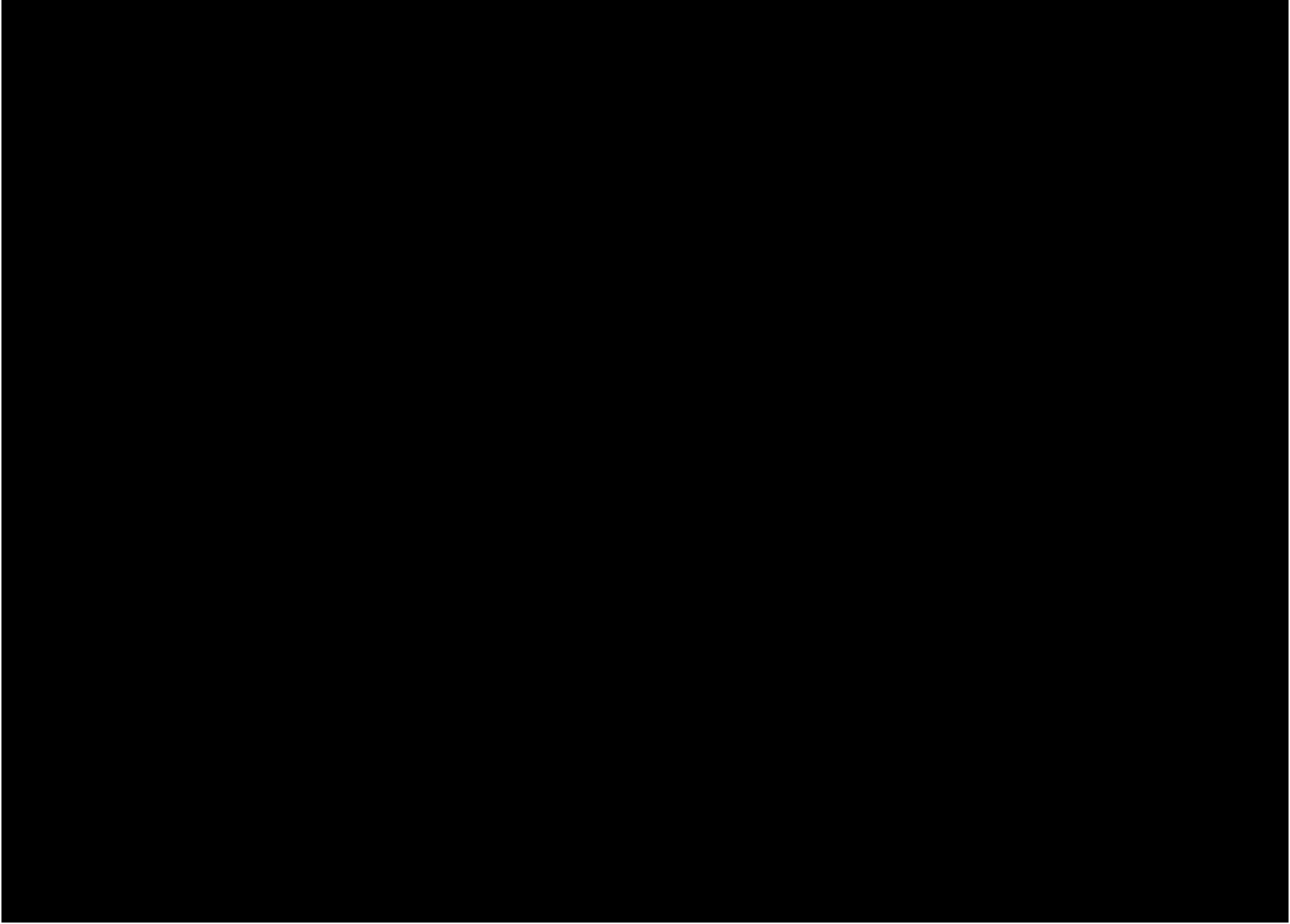
Figure 22-1 Three-dimensional radiologic reconstruction of the human airway with the laryngeal mask airway (LMA) in situ: hyoid bone (a); LMA's cuff (b); cricoid ring (c); arytenoid cartilages (d); thyroid cartilage (e), which is digitally partially removed to demonstrate the position of the LMA; mandible (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.

DAS Difficult intubation guidelines – overview









NORMAL CAPNOGRAM

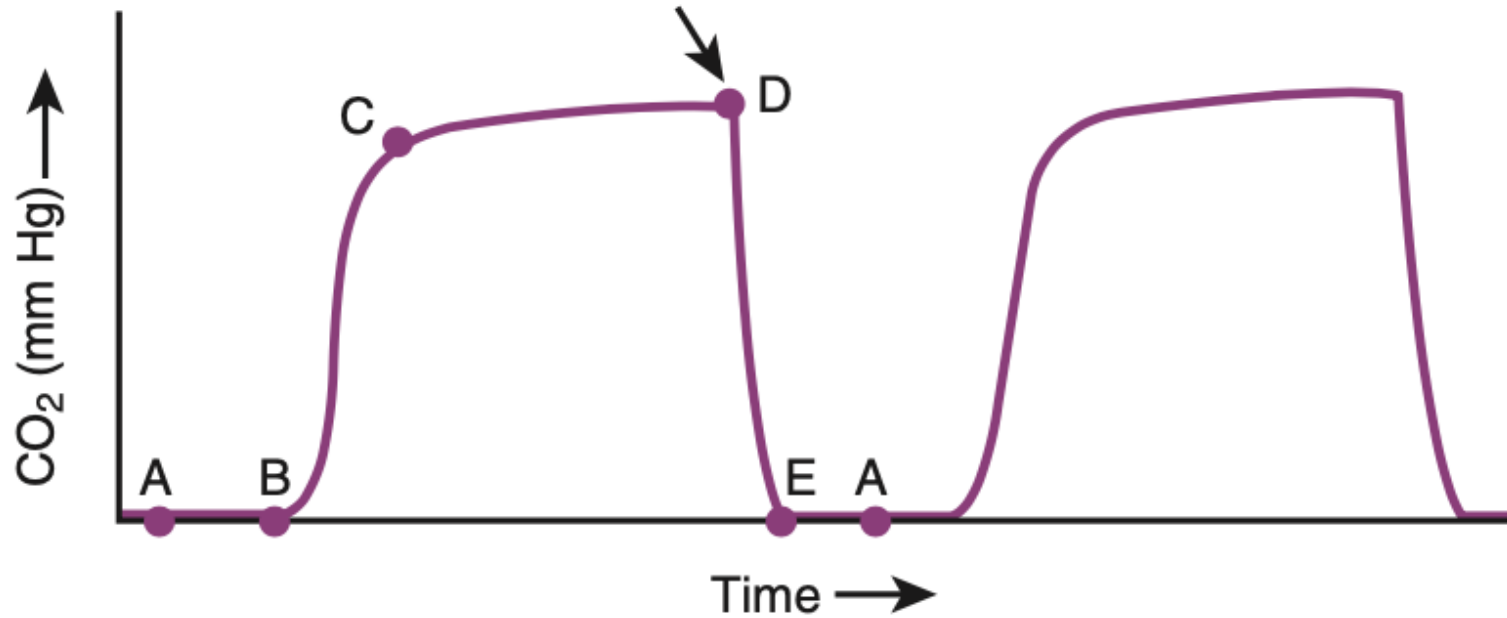
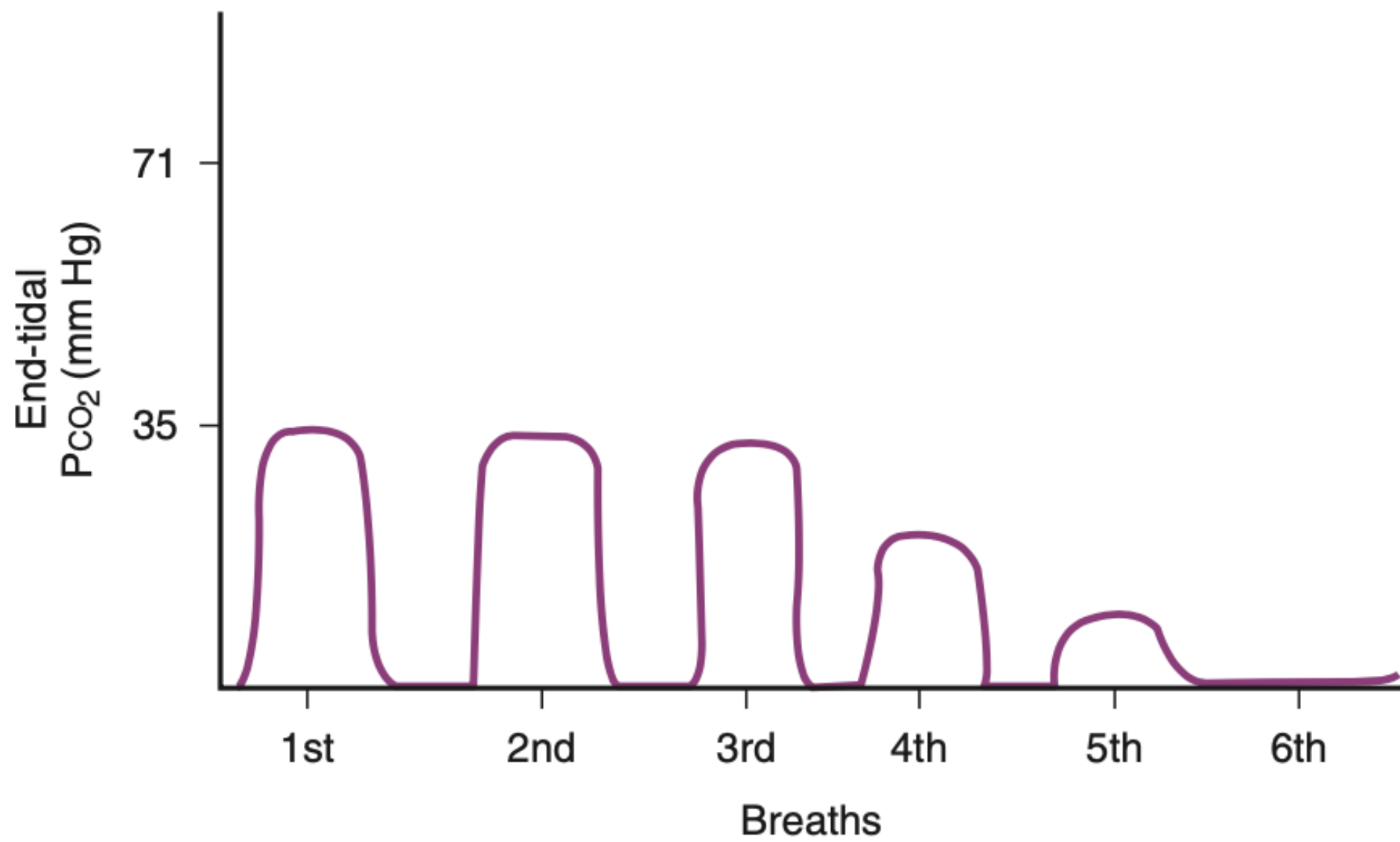


Figure 32-7 The CO₂ waveform. A, Expiratory pause begins. A-B, Clearance of anatomic dead space. B-C, Dead space air mixed with alveolar air. C-D, Alveolar plateau. D, End-tidal partial pressure of CO₂ registered by capnograph (arrow) and beginning of inspiratory phase. D-E, Clearance of dead space air. E-A, Inspiratory gas devoid of CO₂. (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.)

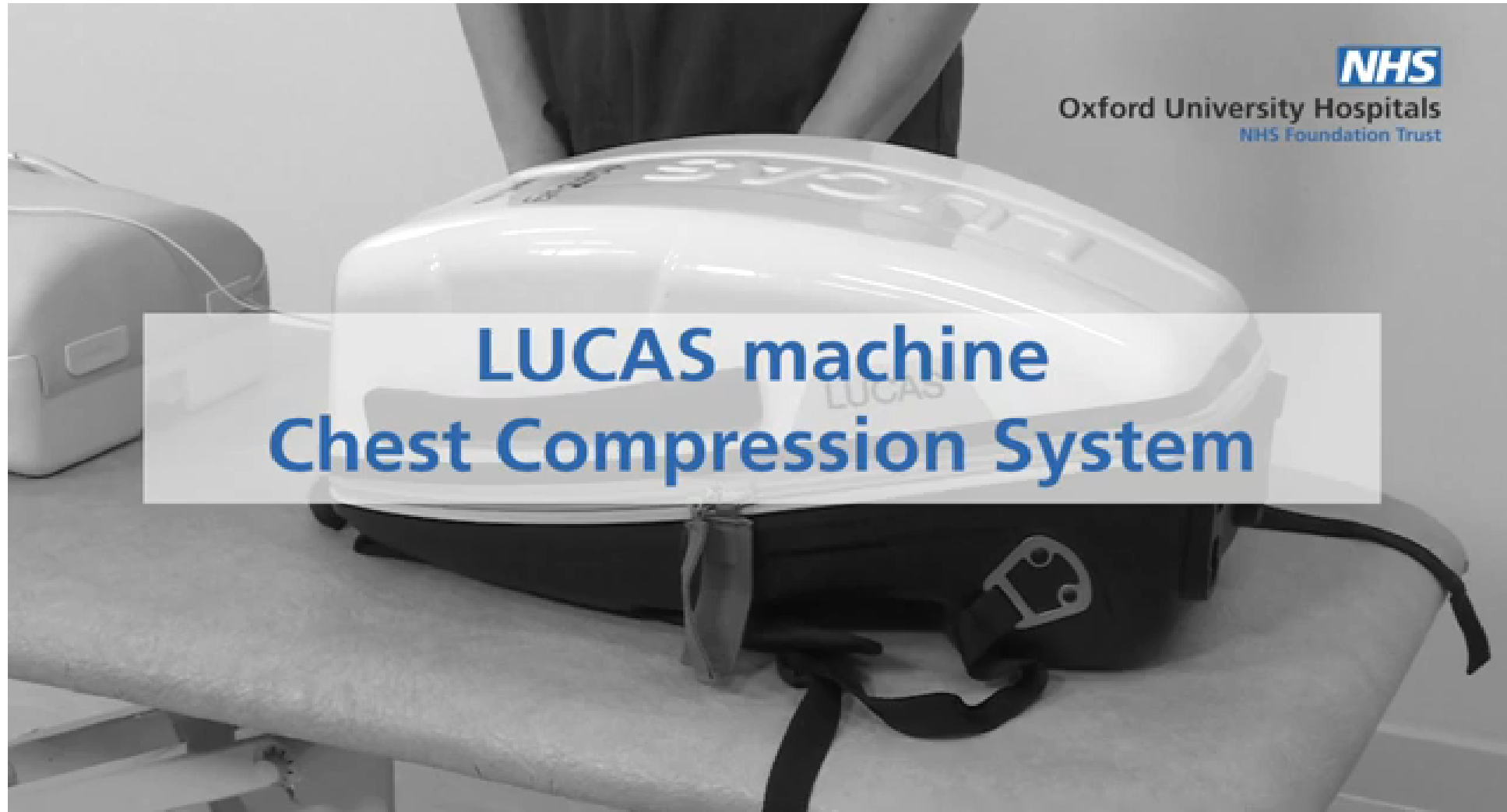


CPR

 Resuscitation Council (UK)



Automated Chest Compression



Conclusions

The provision of high-quality CPR is a key modifiable factor associated with survival in cardiac arrest. Mechanical chest compression devices consistently deliver high-quality chest compressions, but this does not translate into improved patient outcomes when devices are routinely used in OHCA. Further trials are needed to evaluate the routine use of mechanical devices in IHCA.

The use of mechanical devices in specific circumstances (e.g. ambulance/helicopter transport, pPCI) where high-quality chest compressions cannot be safely delivered may be a reasonable strategy. In all situations where mechanical devices are used, clinicians must ensure that the device is deployed with minimal interruption to chest compression delivery.

Rischio infettivo durante CPR

TABLE 1 Pathogens categorized by mode of transmission and examples of procedures undertaken during CPR that can be transmitted

Mode of transmission	Pathogens ^a	Examples of condition(s) leading to transmission during CPR
Direct transmission		
Contact with blood (blood-borne)	HIV*, HBV*, HCV*, Ebola	Needlestick injury during cannulation and blood sampling
Contact with body fluids	Ebola, CCHF virus, <i>Neisseria meningitidis</i> , HSV, Norovirus, HAV, <i>Clostridium difficile</i> , other gastrointestinal pathogens (<i>Salmonella</i> , <i>Shigella</i>)	Contact with pleural fluid during insertion of ICD, contact with saliva during mouth-to-mouth ventilation, contact with feces
Contact with skin	VZV, HSV, HPV*, <i>Staphylococcus aureus</i> * and <i>Streptococcus pyogenes</i> * (from impetigo lesions)	Chest compressions without gloves, mouth-to-mouth ventilation
Contact with contaminated surfaces	Influenza, <i>Clostridium difficile</i> (spores), SARS-CoV-2(?), CMV	Unprotected handling of equipment
Droplet transmission (>5-μm droplet diam)	SARS-CoV-2, MERS-CoV, influenza, CMV	Intubation, suctioning of secretion, administration of nebulized drugs
Indirect transmission		
Airborne transmission	Measles, VZV, <i>Mycobacterium tuberculosis</i> , influenza, CMV	Unprotected

^a*, requires skin breach. Abbreviations: CCHF virus, Crimean-Congo hemorrhagic fever virus; CMV, cytomegalovirus; HAV, hepatitis A virus; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus; HPV, human papillomavirus; HSV, herpes simplex virus; ICD, intercostal chest drain; MERS-CoV, Middle East respiratory syndrome coronavirus; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; VZV, varicella-zoster virus.

Risks associated with cardiopulmonary resuscitation (CPR) in patients with COVID-19

Mechanisms of transmission of SARS-CoV-2

The main mechanism of disease transmission of SARS-CoV-2 is by respiratory secretions either directly from the patient or by touching contaminated surfaces. Viable virus is detectable on some surfaces for up to 72 h.¹¹ Respiratory secretions are called either droplets (> 5 – 10 microns in diameter) or airborne particles (< 5 microns). Droplets fall onto surfaces within 1–2 m of the patient's respiratory tract while airborne particles can remain suspended in the air for prolonged periods.^{11–15}

The International Liaison Committee on Resuscitation (ILCOR) has undertaken a systematic review addressing three questions:¹⁶

- 1 Is the delivery of chest compressions or defibrillation an aerosol-generating procedure?
- 2 Do the delivery of chest compressions, defibrillation or CPR (all CPR interventions that include chest compressions) increase infection transmission?
- 3 What type of PPE is required by individuals delivering chest compressions, defibrillation or CPR in order to prevent transmission of infection from the patient to the rescuer?

The evidence addressing these questions is scarce and comprises mainly retrospective cohort studies^{17,18} and case reports.^{19–24}

In most cases, delivery of chest compressions and defibrillation are lumped together with all CPR interventions, which means that there is considerable confounding in these studies. Aerosol generation by chest compressions is plausible because they generate small but measurable tidal volumes.^{25,26} Chest compressions are similar to some chest physiotherapy techniques, which are associated with aerosol generation.²⁷ Furthermore, the person performing chest compressions is close to the patient's airway.

The ILCOR systematic review did not identify evidence that defibrillation generates aerosols. If it occurs, the duration of an aerosol generating process would be brief. Furthermore, the use of adhesive pads means that defibrillation can be delivered without direct contact between the defibrillator operator and patient. The ILCOR treatment recommendations are listed in [Table 1](#). The values, preferences and Task Force insights summarise the rationale for recommendations for lay persons and health care professionals.

Table 1 – ILCOR treatment recommendations for cardiopulmonary resuscitation (CPR) in patients with COVID-19.

- We suggest that chest compressions and cardiopulmonary resuscitation have the potential to generate aerosols (weak recommendation, very low certainty evidence).
- We suggest that in the current COVID-19 pandemic lay rescuers consider compression-only resuscitation and public-access defibrillation (good practice statement).
- We suggest that in the current COVID-19 pandemic, lay rescuers who are willing, trained and able to do so, may wish to deliver rescue breaths to children in addition to chest compressions (good practice statement).
- We suggest that in the current COVID-19 pandemic, healthcare professionals should use personal protective equipment for aerosol-generating procedures during resuscitation (weak recommendation, very low certainty evidence).
- We suggest that it may be reasonable for healthcare providers to consider defibrillation before donning aerosol generating personal protective equipment in situations where the provider assesses the benefits may exceed the risks (good practice statement).

Research Question 1.

- Population: Individuals in any setting
- Exposure: Delivery of chest compressions, defibrillation, CPR (all CPR interventions that include chest compressions)
- Outcome: Generation of aerosols
- Study design: RCTs and nonrandomized studies (non-RCTs, interrupted time series, controlled before-and-after studies, cohort studies, case reports/series, cadaver studies) were eligible for inclusion. Unpublished studies (eg, conference abstracts, trial protocols) were excluded.
- Time frame: All years and all languages were included as long as there was an English abstract. Searches were updated in January 2021.

Research Question 2.

- Population: Individuals in any setting wearing any PPE or no PPE
- Exposure: Delivery of chest compressions, defibrillation, CPR (all CPR interventions that include chest compressions)
- Outcome: Transmission of infection
- Study designs: RCTs and nonrandomized studies (non-RCTs, interrupted time series, controlled before-and-after studies, cohort studies, case reports/series) were eligible for inclusion. Unpublished studies (eg, conference abstracts, trial protocols) were excluded.

BLSD e Covid-19



BLS-D

per operatori non sanitari

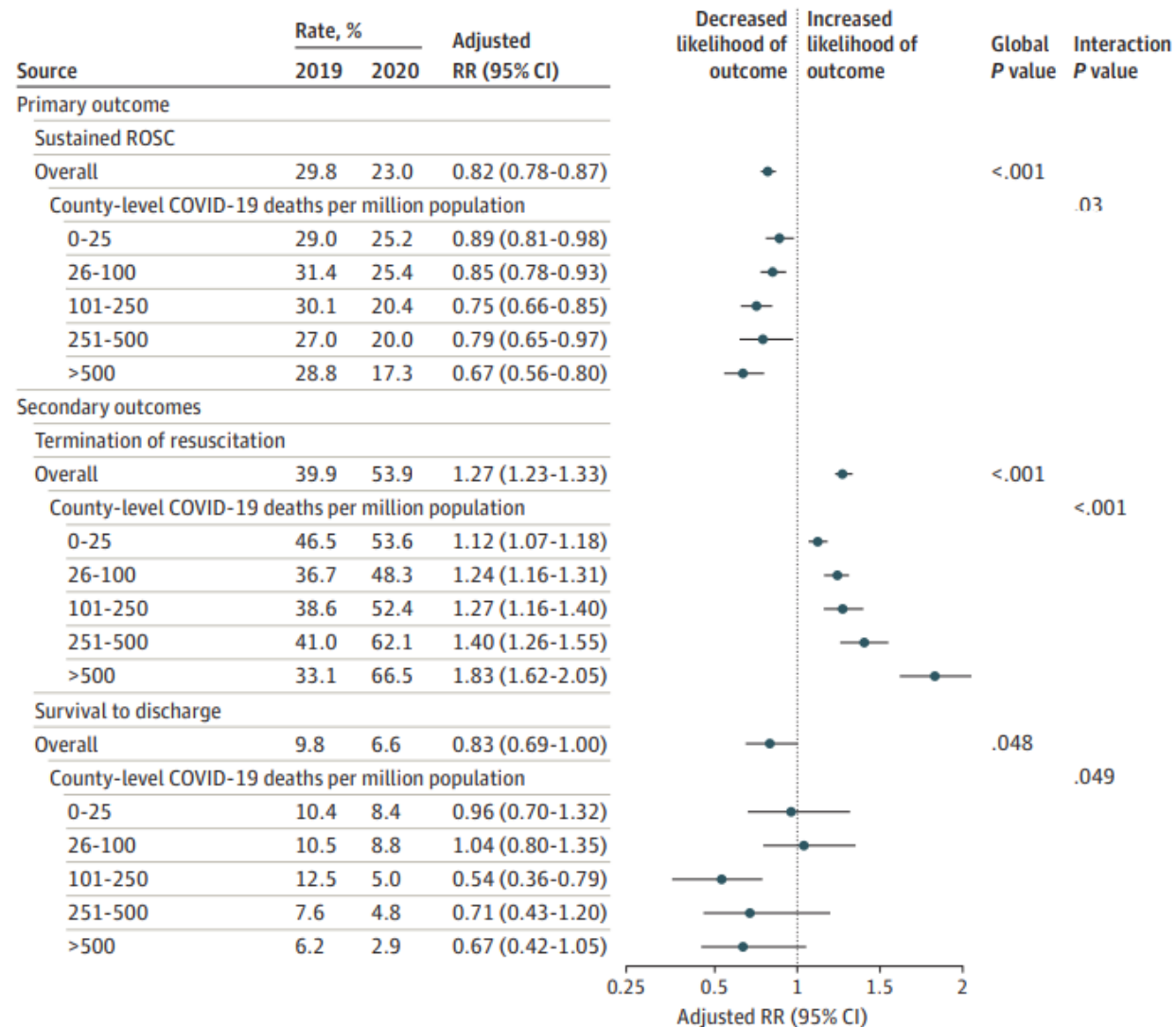
Basic Life Support and Early Defibrillation

Linee Guida ERC 2015 - *Integrazione Covid - 2020*



Italian
Resuscitation
Council

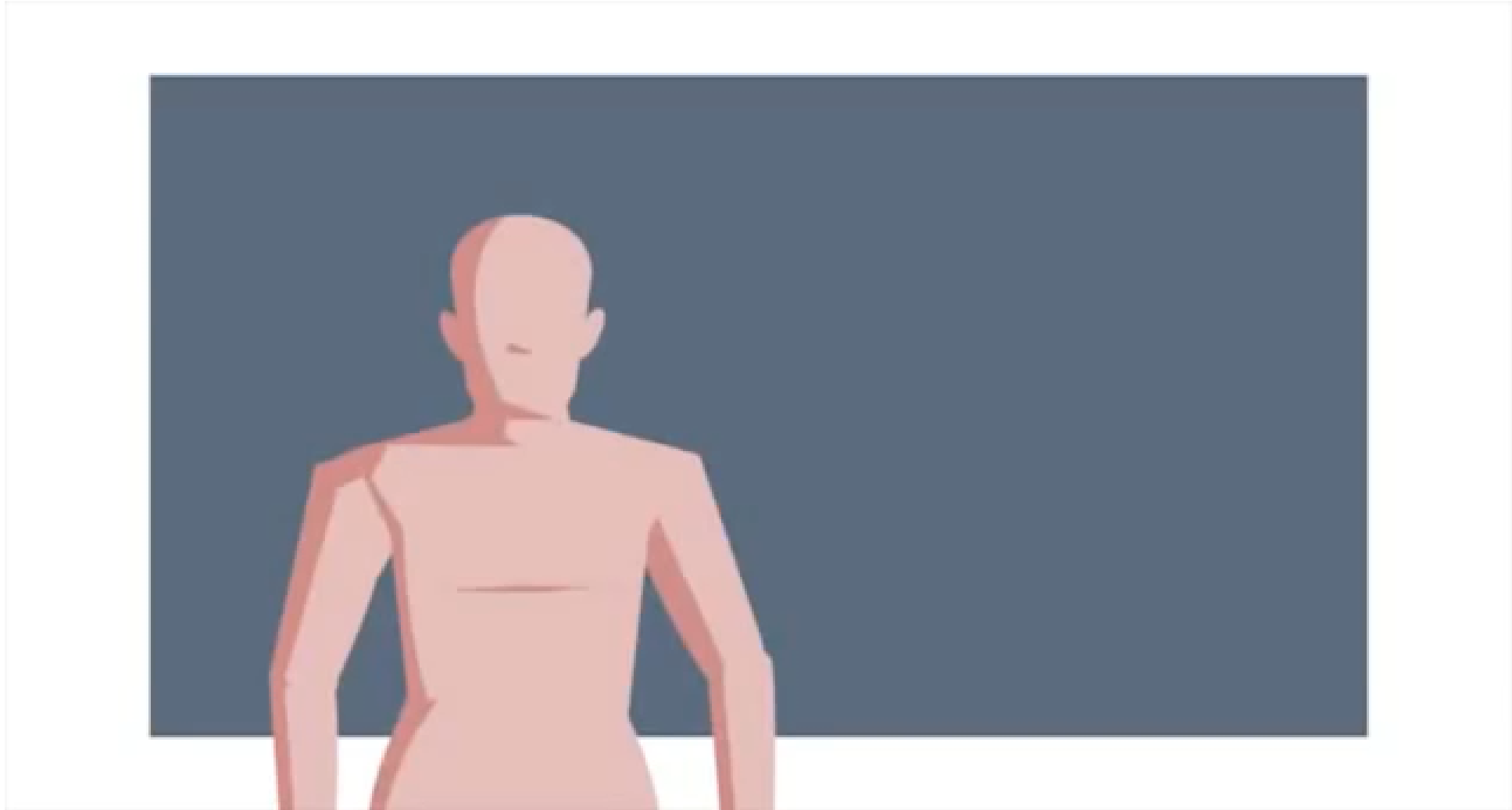
Figure 2. Rates of Sustained Return of Spontaneous Circulation (ROSC), Termination of Resuscitation, and Survival to Discharge During the 2020 Pandemic Period vs 2019



Comparisons of rates are shown for the overall cohort and stratified by the county-level coronavirus disease 2019 (COVID-19) mortality rate. The analysis for the outcome of survival to discharge was restricted to emergency medical services agencies with complete data on this outcome. RR indicates rate ratio.

Agonal Breathing

<https://www.youtube.com/watch?v=fXQJ4klzSas>



The Role of Gasping in Resuscitation

L.P. ROPPOLO, P.E. PEPE, and B.J. BOBROW

Gasping is a physiologic entity that, among other conditions, is seen typically in mammals who have sustained a global ischemic insult such as sudden cardiac arrest or severe hemorrhagic shock [1–13]. Scientists have defined a gasp formally in nomenclature consensus processes as “an abrupt, sudden, transient inspiratory effort” [13] and it has been described in the published literature since 1812 [11]. The classic gasping that occurs after sudden cardiac arrest is also sometimes referred to as “agonal breaths” or “agonal respirations” [1, 3–6, 9]. However, the term agonal breathing may also be used by some when referring to a broader variety of respiratory efforts or conditions [12, 14]. Agonal breathing may, therefore, refer to various kinds of abnormal breathing observed at the time of clinical death, during certain types of stroke, or in progressive respiratory failure when rapid breathing reverts to slower and often shallow breaths [6, 11, 12, 14]. Classic gasps, according to strict definition, however, are usually sudden, abrupt, and much brisker and larger than normal respiratory efforts [13].

Conclusion

Gasping and other forms of agonal respiratory efforts may not only have positive prognostic value for those with global ischemic events, but they also likely serve as an adjunctive resuscitative intervention for both cardiac arrest and severely injured patients. Gasping is more of a physiologically sound mode of ventilation when compared to the traditional use of positive pressure breathing. Gasps may, therefore, even be more efficient and effective than traditional mouth-to-mouth rescue breathing and other positive pressure ventilatory techniques. Indeed, recent data demonstrate that gasping can enhance pulmonary gas exchange (oxygenation and ventilation) as well as circulation by enhancing venous return and, in turn, cardiac output, aortic pressures, coronary artery perfusion, and cerebral blood flow. Recent studies designed to identify gasping over the telephone at emergency dispatch offices have dramatically increased the ability of dispatchers to detect persons with cardiac arrest. In turn, dispatchers can now prompt earlier performance of chest compressions in a large number of cases in which the life-saving technique might not have been performed until arrival of professional responders. Such studies may provide a model for the future relevant training of laypersons, EMS responders and other medical personnel.



Management of hypoglycaemia

- The signs of hypoglycaemia are sudden impaired consciousness: ranging from dizziness, fainting, sometimes nervousness and deviant behaviour (mood swings, aggression, confusion, loss of concentration, signs that look like drunkenness) to loss of consciousness.
- A person with mild hypoglycaemia typically has less severe signs or symptoms and has the preserved ability to swallow and follow commands.



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- If hypoglycaemia is suspected in someone who has signs or symptoms of mild hypoglycaemia and is conscious and able to swallow:
 - Give glucose or dextrose tablets (15–20 g), by mouth
 - If glucose or dextrose tablets are not available give other dietary sugars in an equivalent amount to glucose, such as Skittles, Mentos, sugar cubes, jellybeans, or half a can of orange juice
 - Repeat the administration of sugar if the symptoms are still present and not improving after 15 min
 - If oral glucose is not available a glucose gel (partially held in the cheek, and partially swallowed) can be given
 - Call the emergency services if:
 - the casualty is/or becomes unconscious
 - the casualty's condition does not improve



- Following recovery from the symptoms after taking the sugar, encourage taking a light snack such as a sandwich or a waffle
- For children who may be uncooperative with swallowing oral glucose:
 - Consider administering half a teaspoon of table sugar (2.5 g) under the child's tongue.
- If possible, measure and record the blood sugar levels before and after treatment.



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Oral rehydration solutions for treating exertion-related dehydration

- If a person has been sweating excessively during a sports performance and exhibits signs of dehydration such as feeling thirsty, dizzy or light-headed and/or having a dry mouth or dark yellow and strong-smelling urine, give him/her 3–8% carbohydrate-electrolyte (CE) drinks (typical ‘sports’ rehydration drinks) or skimmed milk.



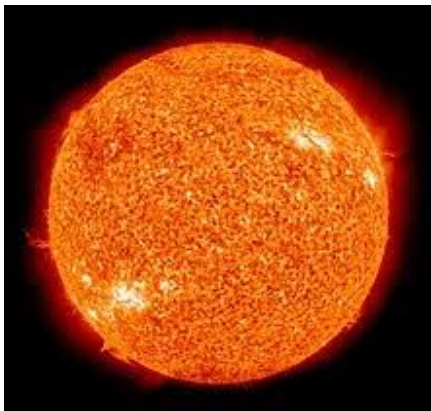
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- If 3–8% CE drinks or milk are not available or not well tolerated, alternative beverages for rehydration include 0–3% CE drinks, 8–12% CE drinks or water.
- Clean water, in regulated quantities, is an acceptable alternative, although it may require a longer time to rehydrate.
- Avoid the use of alcoholic beverages.
- Call the emergency services if:
 - The person is or becomes unconscious
 - The person shows signs of a heat stroke.



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Management of heat stroke by cooling

Recognise the symptoms and signs of heat stroke (in the presence of a high ambient temperature):

- Elevated temperature
- Confusion
- Agitation
- Disorientation
- Seizures
- Coma.



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When exertional or non-exertional heat stroke is suspected:

- Immediately remove the casualty from the heat source and commence passive cooling
- Commence additional cooling using any technique immediately available
 - If the core temperature is above 40 °C commence whole body (neck down) cold water (1–26 °C) immersion until the core temperature falls below 39 °C
 - If water immersion is not possible use alternative methods of cooling e.g. ice sheets, commercial ice packs, fan alone, cold shower, hand cooling devices, cooling vests and jackets or evaporative cooling (mist and fan)
- Where possible measure the casualty's core temperature (rectal temperature measurement) which may require special training
- Casualties with exertional hyperthermia or non-exertional heat-stroke will require advanced medical care and advance assistance should be sought.

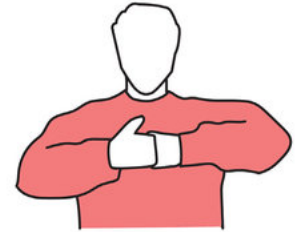
Management of presyncope

- Presyncope is characterised by light-headedness, nausea, sweating, black spots in front of the eyes and an impending sense of loss of consciousness.
- Ensure the casualty is safe and will not fall or injure themselves if they lose consciousness.
- Use simple physical counterpressure manoeuvres to abort presyncope of vasovagal or orthostatic origin.
- Lower body physical counterpressure manoeuvres are more effective than upper body manoeuvres.
 - Lower body – Squatting with or without leg crossing
 - Upper body – Hand clenching, neck flexion
- First aid providers will need to be trained in coaching casualties in how to perform physical counterpressure manoeuvres.

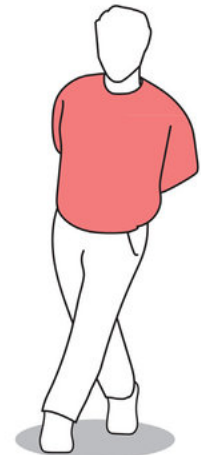
1. Squatting



2. Arm tensing



3. Leg tensing





Dental avulsion

- If the casualty is bleeding from the avulsed tooth socket:
 - Put on disposable gloves prior to assisting the victim
 - Rinse out the casualty's mouth with cold, clean water
 - Control bleeding by:
 - Pressing a damp compress against the open tooth socket
 - Tell the casualty to bite on the damp compress



Figure 1A: Loss of tooth (Source: Dental Trauma Guide, 2010)



Figure 1B: Empty socket appearance (Source: Dental Trauma Guide, 2010)



Figure 1C: Radiographic image (Source: Dental Trauma Guide, 2010)

- Do not do this if there is a high chance that the injured person will swallow the compress (for example, a small child, an agitated person or a person with impaired consciousness).



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- If it is not possible to immediately replant the avulsed tooth at the place of accident:
 - Seek help from a specialist
 - Take the casualty and the avulsed tooth to seek expert help from a specialist.
- Only touch an avulsed tooth at the crown. Do not touch the root
- Rinse a visibly contaminated avulsed tooth for a maximum of 10 seconds with saline solution or under running tap water prior to transportation.
- To transport the tooth:
 - Wrap the tooth in cling film or store the tooth temporarily in a small container with Hank's Balanced Salt solution (HBSS), propolis or Oral Rehydration Salt (ORS) solution
 - If none of the above are available, store the tooth in cow's milk (any form or fat percentage)
 - Avoid the use of tap water, buttermilk or saline (sodium chloride).



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Apply direct pressure on external wounds with sterile cloth or your hand, maintaining pressure until bleeding stops



Control of life-threatening bleeding

Direct pressure, haemostatic dressings, pressure points and cryotherapy for life-threatening bleeding

- Apply direct manual pressure for the initial control of severe, life-threatening external bleeding.
- Consider the use of a haemostatic dressing when applying direct manual pressure for severe, life-threatening bleeding. Apply the haemostatic dressing directly to the bleeding injury and then apply direct manual pressure to the dressing.
- A pressure dressing may be useful once bleeding is controlled to maintain haemostasis but should not be used in lieu of direct manual pressure for uncontrolled bleeding.
- Use of pressure points or cold therapy is not recommended for the control of life-threatening bleeding.



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Tourniquets for life-threatening bleeding

- For life-threatening bleeding from wounds on limbs in a location amenable to the use of a tourniquet (i.e. arm or leg wounds, traumatic amputations):
 - Consider the application of a manufactured tourniquet as soon as possible:
 - Place the tourniquet around the traumatised limb 5–7 cm above the wound but not over a joint
 - Tighten the tourniquet until the bleeding slows and stops. This may be extremely painful for the casualty
 - Maintain the tourniquet pressure
 - Note the time the tourniquet was applied
 - Do not release the tourniquet – the tourniquet must only be released by a healthcare professional
 - Take the casualty to hospital immediately for further medical care
 - In some cases, it may require the application of two tourniquets in parallel to slow or stop the bleeding.



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Tourniquets for life-threatening bleeding

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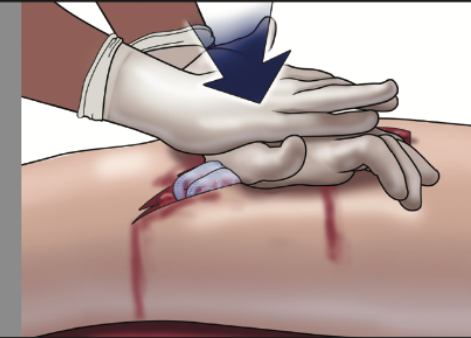
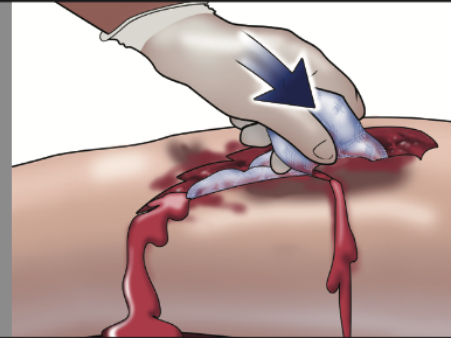
THE
COMMITTEE
ON TRAUMA



1 APPLY PRESSURE WITH HANDS



2 APPLY DRESSING AND PRESS



3 APPLY TOURNIQUET



WRAP

WIND

SECURE

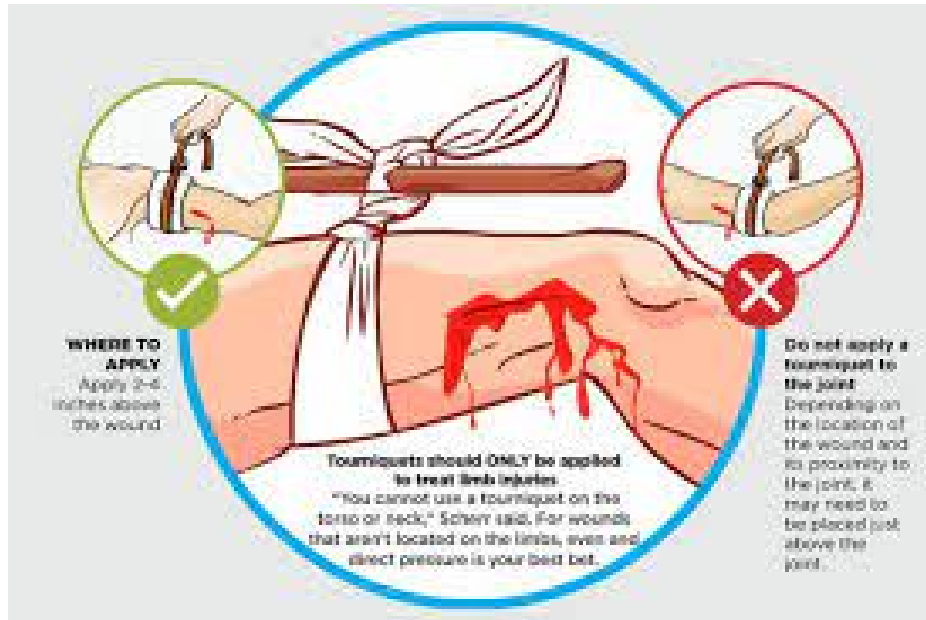
TIME

CALL 911



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- If a manufactured tourniquet is not immediately available, or if bleeding is uncontrolled with the use of a manufactured tourniquet, apply direct manual pressure, with a gloved hand, a gauze dressing, or if available, a haemostatic dressing.
- Consider the use of an improvised tourniquet only if a manufactured tourniquet is not available, direct manual pressure (gloved hand, gauze dressing or haemostatic dressing) fails to control life-threatening bleeding, and the first aid provider is trained in the use of improvised tourniquets.



What Is a Tourniquet?

A **tourniquet** is a device that is placed around a bleeding arm or leg. Tourniquets work by squeezing large blood vessels. The squeezing helps stop blood loss.

How Do I Put a Tourniquet On?

Tourniquets can be made out of any available material. For example, you can use a bandage, strip of cloth, or even a t-shirt. The material should be at least 2 to 3 inches wide. The material should also overlap itself. Using thin straps or material less than 2 inches wide can rip or cut the skin.

Tourniquets often use a windlass device to increase tightening. Inflated tourniquets (for example, those made from blood pressure cuffs) can work well. But they must be carefully watched for small leaks.

The injured blood vessel is not always right below the skin wound. Place the tourniquet between the injured vessel and the heart, about 2 inches from the closest wound edge. There should be no foreign objects (for example, items in a pocket) beneath the tourniquet. Place the tourniquet over a bone, not at joint.

Galante JM. Using Tourniquets to Stop Bleeding. JAMA. 2017;317(14):1490-1490. doi:10.1001/jama.2015.8581

Applying a tourniquet with a windlass device

Apply direct pressure to the wound for at least 15 minutes.

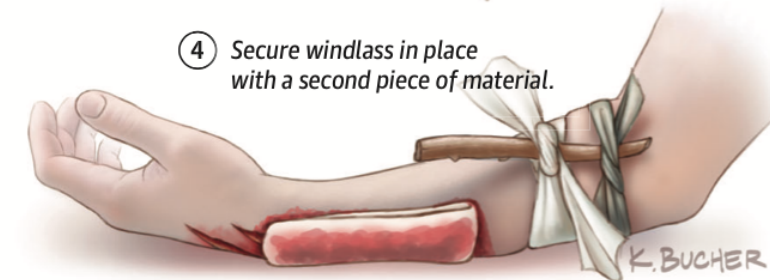
Use a tourniquet only when bleeding cannot be stopped and is life threatening.

① Place a 2-3" strip of material about 2" from the edge of the wound over a long bone between the wound and the heart.

② Insert a stick or other strong, straight item into the knot to act as a windlass.

③ Turn stick to tighten tourniquet until pulse below the tourniquet cannot be felt.

④ Secure windlass in place with a second piece of material.



Keep tourniquet visible and monitor wound for bleeding. Note time and watch for swelling below tourniquet.

What Else Do I Need to Know?

All bleeding should stop soon after you tighten the tourniquet. You must place a second tourniquet above the first if bleeding does not stop and you cannot tighten the tourniquet, or if the arm or leg swells above the tourniquet.

Once bleeding is controlled

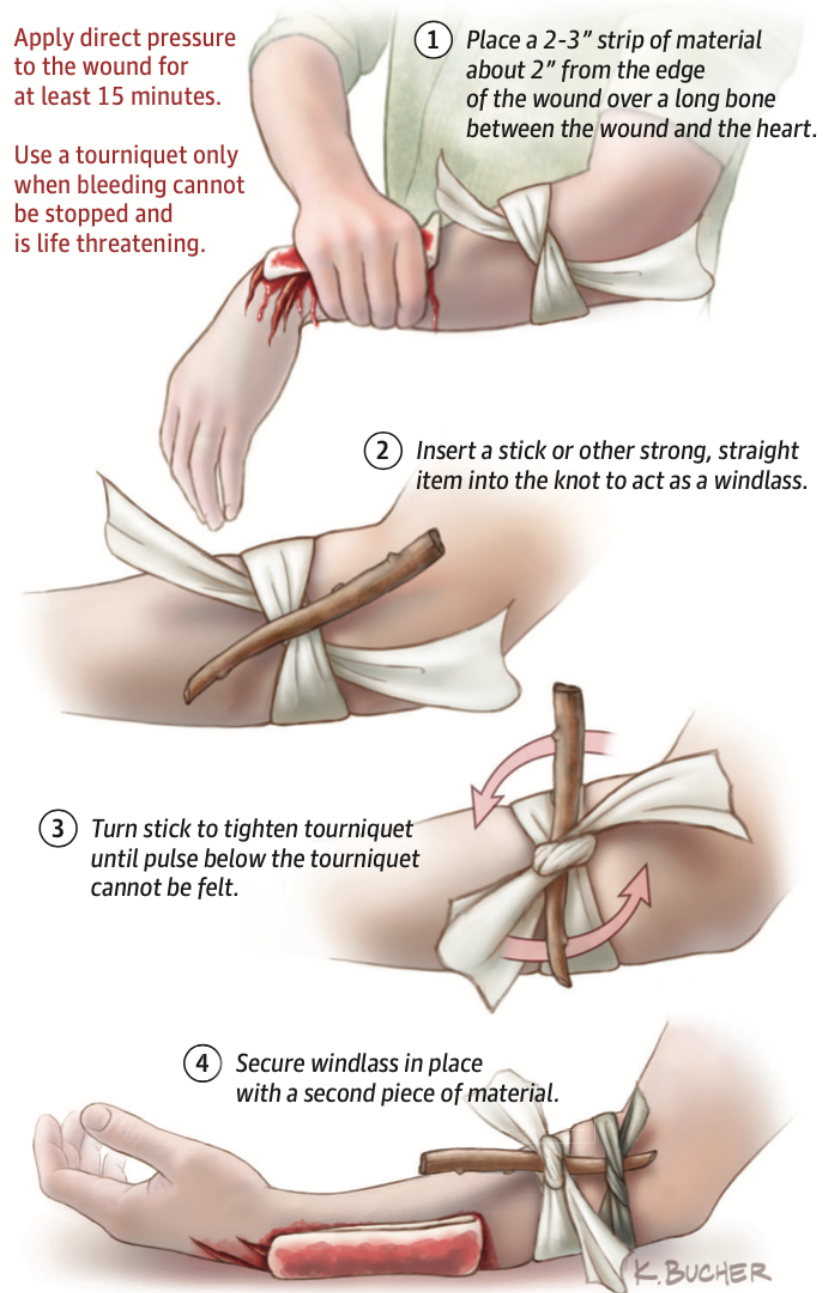
- Mark the time on the arm or leg
- Keep the tourniquet visible
- Check the arm or leg every 2 hours for
 - Swelling
 - New bleeding
 - Increased muscle stiffness

Do not remove or loosen the tourniquet until professional care is available.

Applying a tourniquet with a windlass device

Apply direct pressure to the wound for at least 15 minutes.

Use a tourniquet only when bleeding cannot be stopped and is life threatening.



Keep tourniquet visible and monitor wound for bleeding. Note time and watch for swelling below tourniquet.



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COUNCIL**

Management of open chest wounds

- Leave an open chest wound exposed to freely communicate with the external environment.
- Do not apply a dressing or cover the wound.
- If necessary:
 - Control localised bleeding with direct pressure
 - Apply a specialised non-occlusive or vented dressing ensuring a free outflow of gas during expiration (training required).



Cervical spine motion restriction and stabilisation

- The routine application of a cervical collar by a first aid provider is not recommended.
- In a suspected cervical spine injury:
 - If the casualty is awake and alert, encourage them to self-maintain their neck in a stable position.
 - If the casualty is unconscious or uncooperative consider immobilising the neck using manual stabilisation techniques.
 - Head squeeze:
 - With the casualty lying supine hold the casualty's head between your hands.
 - Position your hands so that the thumbs are above the casualty's ears and the other fingers are below the ear
 - Do not cover the ears so that the casualty can hear.





**EUROPEAN
RESUSCITATION
COUNCIL**

- Trapezium squeeze:
 - With the casualty lying supine hold the casualty's trapezius muscles on either side of the head with your hands (thumbs anterior to the trapezius muscle). In simple terms – hold the casualty's shoulders with the hands thumbs up
 - Firmly squeeze the head between the forearms with the forearms placed approximately at the level of the ears.



1

STAY with the person until they are awake and alert after the seizure.

- ✓ **Time** the seizure
- ✓ Remain **calm**
- ✓ Check for **medical ID**



2

Keep the person **SAFE**.

- ✓ Move or guide away from **harm**



3

Turn the person onto their **SIDE** if they are not awake and aware.

- ✓ Keep **airway clear**
- ✓ **Loosen tight clothes** around neck
- ✓ Put **something small and soft** under the head

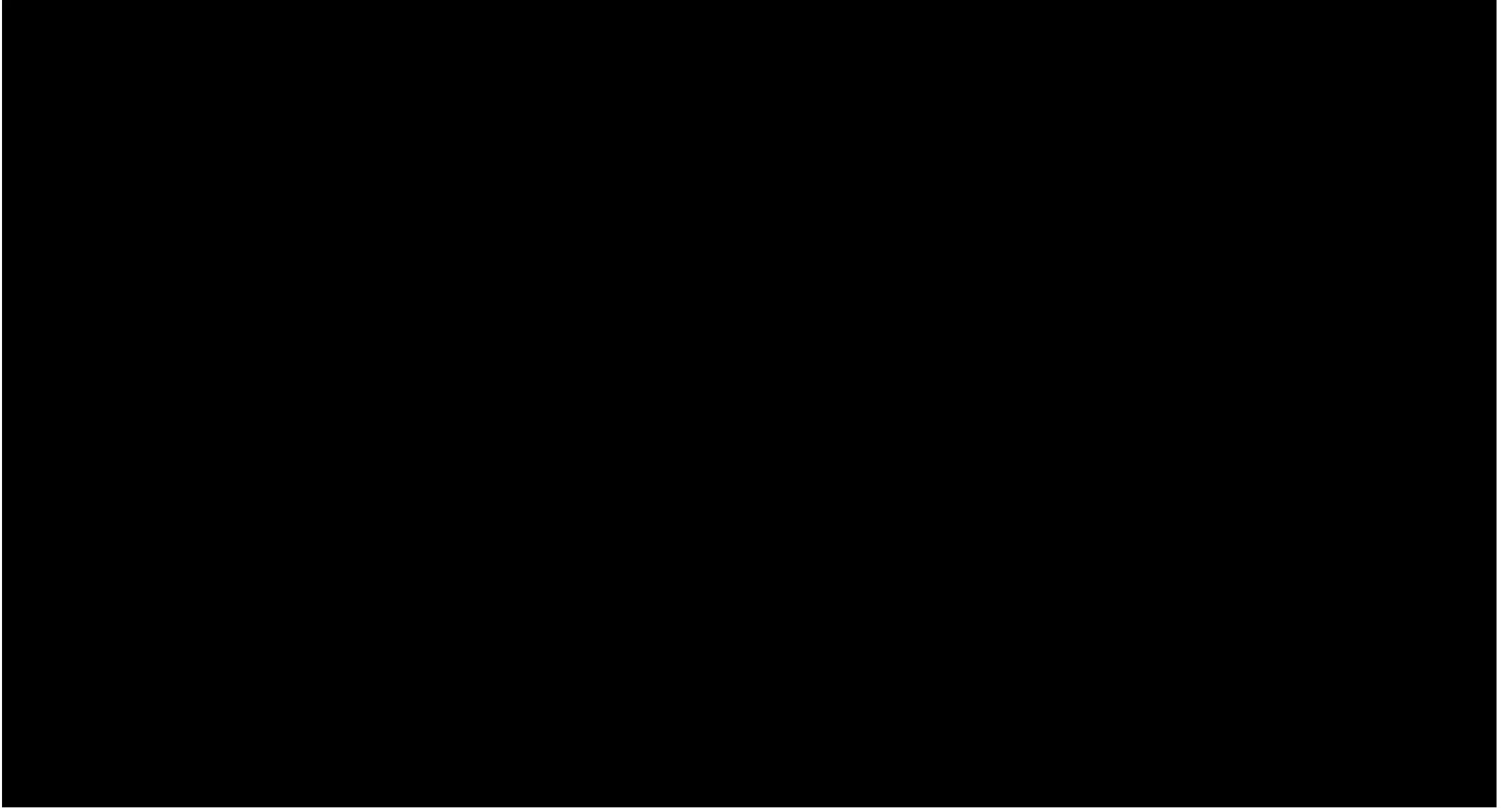


Call
911
if...

- ▶ Seizure lasts longer than 5 minutes
- ▶ Person does not return to their usual state
- ▶ Person is injured, pregnant, or sick
- ▶ Repeated seizures
- ▶ First time seizure
- ▶ Difficulty breathing
- ▶ Seizure occurs in water

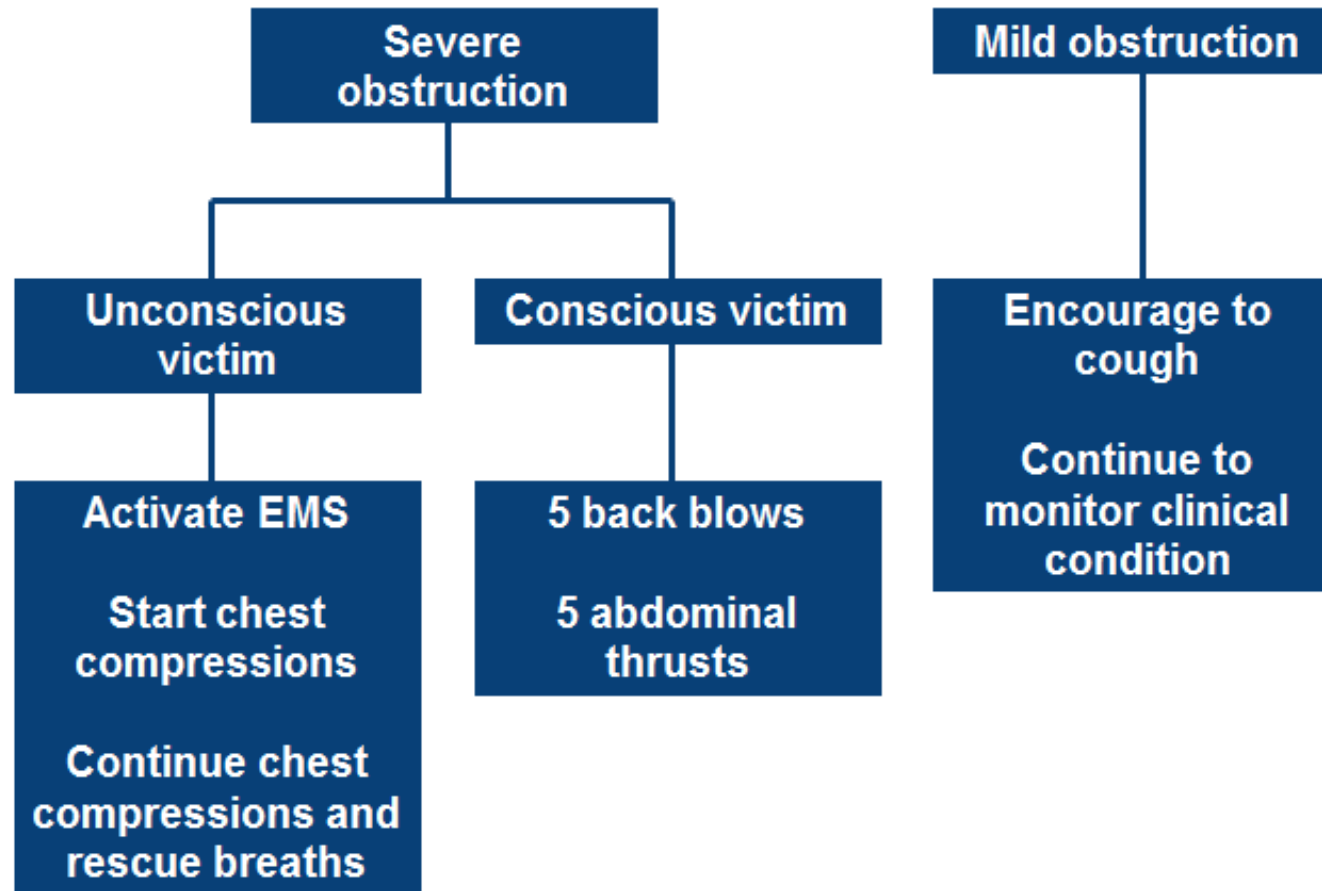
Do
NOT

- ✗ Do **NOT** restrain.
- ✗ Do **NOT** put any objects in their mouth.
- ▶ **Rescue medicines can be given** if prescribed by a health care professional





CHOKING: ALGORITHM





CHOKING

Back Blows



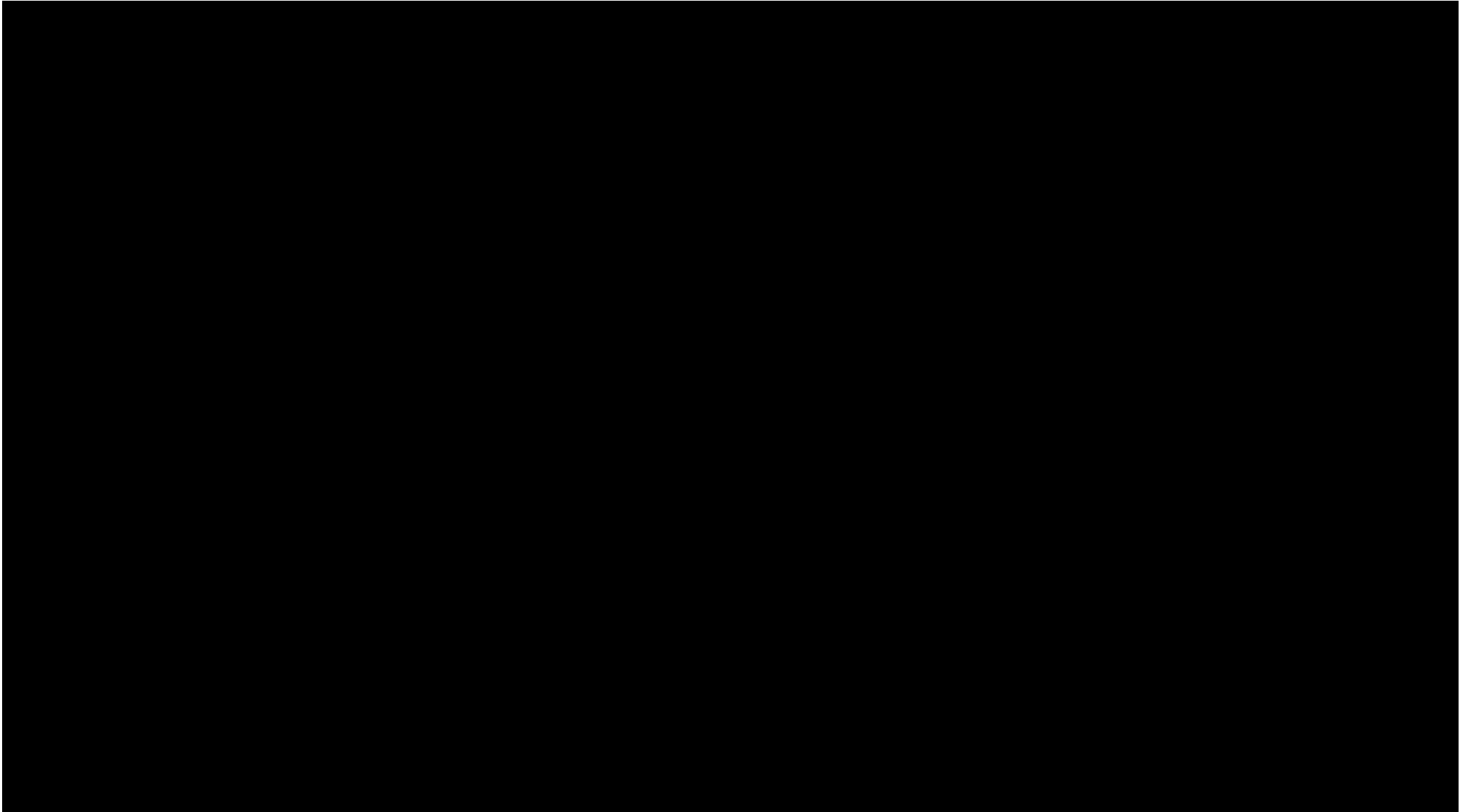
Abdominal thrusts: position of first hand



Abdominal thrusts: position of second hand



Abdominal thrust



Abdominal thrust e gravidanza

Contraindications

Go to: 

Although there are no absolute contraindications, the abdominal thrust maneuver is not recommended by the AHA for infants or unconscious patients. Also, pregnant subjects should receive management with sternal compressions, as opposed to abdominal.[\[13\]](#)

To clear the airway of a pregnant woman or obese person:

- **Position your hands a little bit higher** than with a normal Heimlich maneuver, at the base of the breastbone, just above the joining of the lowest ribs.
- **Proceed as with the Heimlich maneuver**, pressing hard into the chest, with a quick thrust.
- **Repeat** until the food or other blockage is dislodged. If the person becomes unconscious, follow the next steps.

Abdominal thrusts

Don't give abdominal thrusts to babies under 1 year old or pregnant women.

To carry out an abdominal thrust:

- Stand behind the person who's choking.
- Place your arms around their waist and bend them forward.
- Clench 1 fist and place it right above their belly button.
- Put the other hand on top of your fist and pull sharply inwards and upwards.
- Repeat this movement up to 5 times.

If the person's airway is still blocked after trying back blows and abdominal thrusts, get help immediately:

expectorated or until help arrives. If the patient is supine, then a similar upward and inward thrust may be attempted while facing the patient. In a pregnant or obese patients, the fist may be placed slightly higher in the abdomen, just below the xiphoid bone.

Abdominal thrust e gravidanza

Complications

Go to: ☒

Ever since the introduction of the Heimlich maneuver, cases of harm inflicted by the forceful displacement of the diaphragm and sudden increase in intrathoracic pressure have driven numerous studies and reviews. Although this maneuver is considered life-saving and generally safe to perform, serious intra-abdominal harm can ensue from incorrect technique and unusually vigorous application.^[20] One manikin study mentioned above also concluded that the risk of serious harm ensues if the foreign body is not relieved after the first set of thrusts.^[18] The most commonly reported complications are rib fractures and gastric or esophageal perforations. Although other rare traumatic injuries such as splenic rupture, pneumomediastinum, aortic valve cusp rupture, aortic dissection, diaphragmatic herniation, esophageal and jejunal perforation, hepatic rupture, cholesterol embolization leading to arterial occlusion, and mesenteric laceration have been described.^{[21][22][23][24][25][26][27][28]}

Dislocazione dell'utero gravido >20w

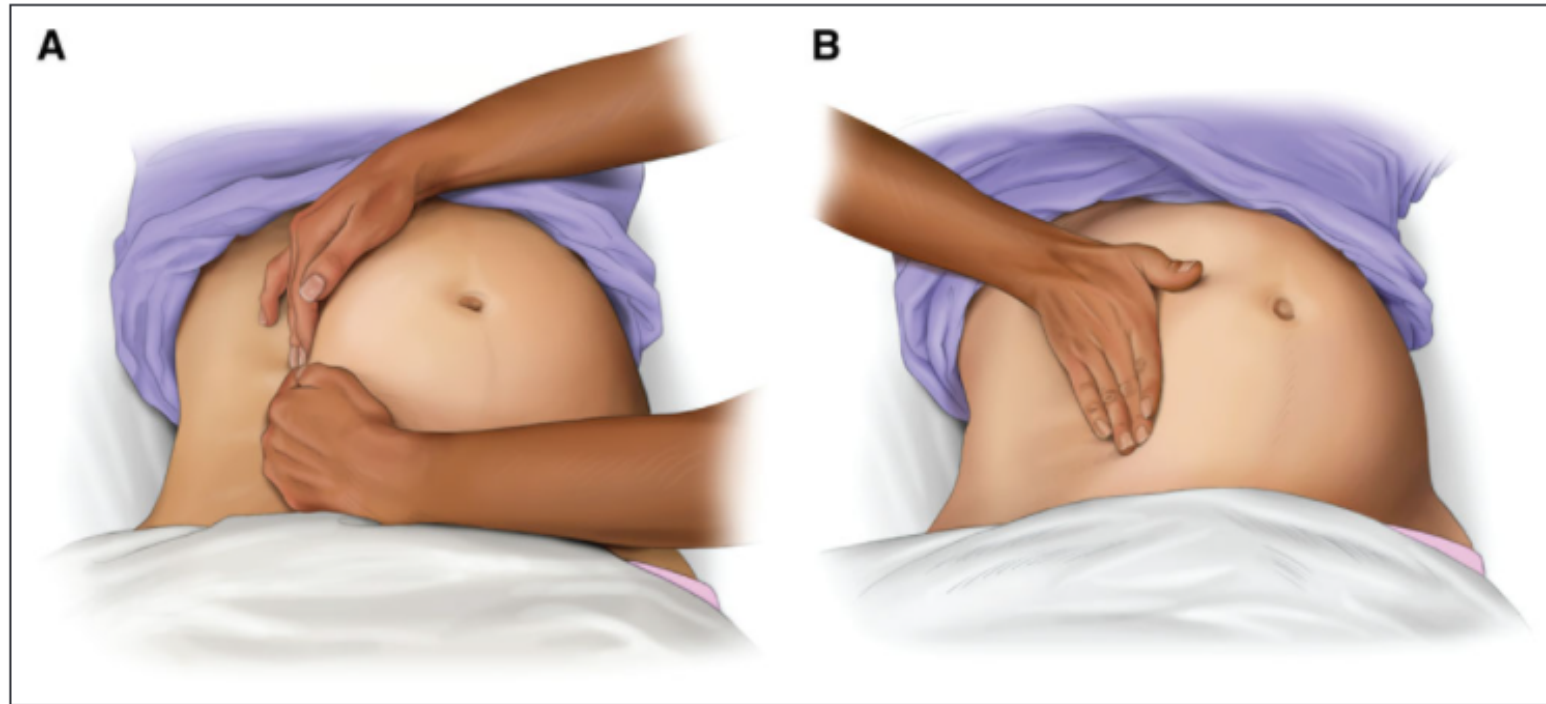
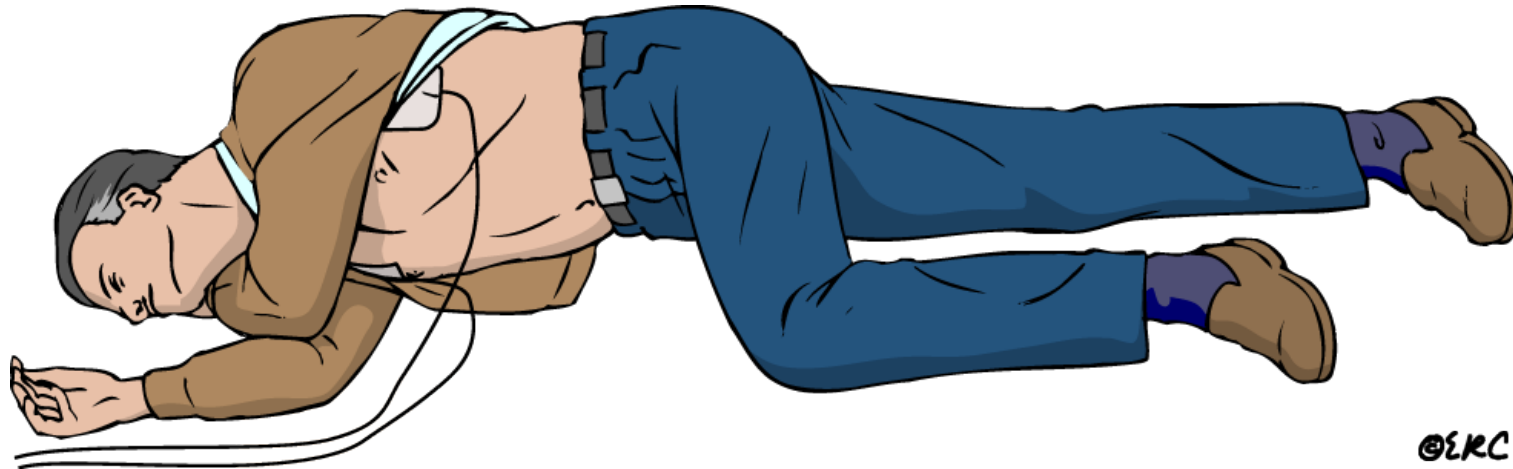


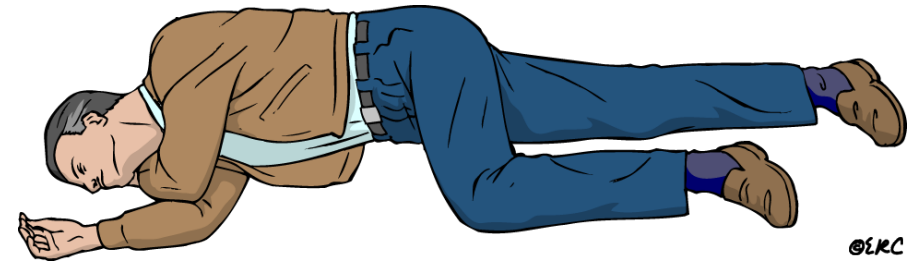
Figure 16. A, Manual left lateral uterine displacement, performed with 2-handed technique. B, 1-handed technique during resuscitation.



If victim starts to breathe normally place
in recovery position



©ERC

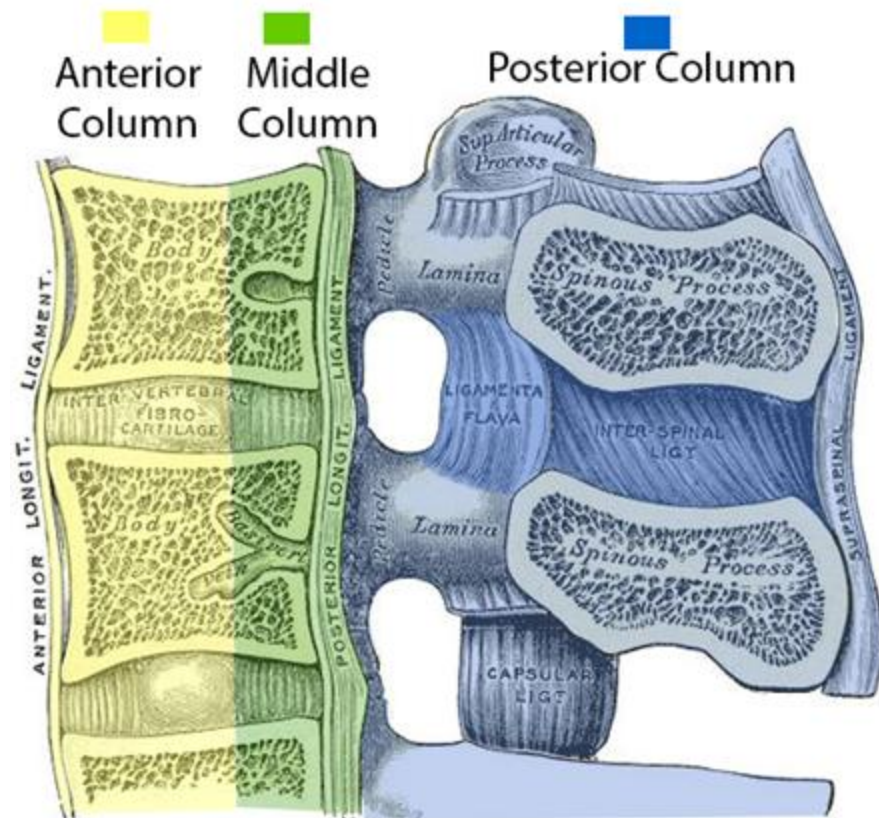


Recovery position e trauma spinale

Concise guideline for clinical practice

Recovery position

For adults and children with a decreased level of responsiveness due to medical illness or non-physical trauma, who do NOT meet the criteria for the initiation of rescue breathing or chest compressions (CPR), the ERC recommends they be placed into a lateral, side-lying, recovery position (see [Fig. 2](#)). Overall, there is little evidence to suggest an optimal recovery position, but the ERC recommends the following sequence of actions:



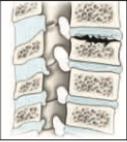






Fracture type (AO/OTA)	Stability	Risk of SCI	Management
A-type: Axial compression			
	A1: Impaction/ compression	Stable	Nonoperative
	A2: Split	Stable	Nonoperative
	A3: Burst	Stable/unstable*	Nonoperative/operative*
B-type: Flexion/distraction or hyperextension			
	B1: Flexion/ distraction	Unstable	Operative
	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	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 al¹³ in 1994.

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

Abstract

The lateral recovery position is widely used for the positioning of unconscious patients. Ideally, in the setting of trauma it is avoided because of concerns about spinal cord injury. However, unconscious individuals with unsuspected trauma or trauma victims attended by partially trained first-aiders may be placed in the recovery position, potentially endangering the cord. Excessive movement of the spine in the recovery position may increase the risk of spinal cord injury in these situations. A new recovery position, termed the modified HAINES position, is described and the position of the spine in this position is compared with the lateral recovery position. *Hypothesis:* That the modified HAINES position results in less distortion of the position of the spine than the lateral recovery position. *Methods:* Thirty-eight healthy volunteers were imaged in the two different positions. Measurements of rotation, flexion and lateral flexion of the cervical and thoraco-lumbar spine were made. Two tailed paired *t*-tests were employed to compare measurements of the two positions and a McNemar test was used to compare the subjects' subjective experiences. *Results:* The modified HAINES position resulted in 13.0° (99% CI: 7.5–18.5) less lateral flexion and 12.6° (99% CI: 9.4–15.9) less extension of the cervical spine while the position of the thoraco-lumbar spine was similar in both positions. Nineteen of 28 subjects found the modified HAINES position more comfortable (not significant). *Conclusion:* The modified HAINES position results in a more neutral position of the spine making it preferable to the lateral recovery position in the management of patients when trauma may have occurred. Further research is required to ensure that the recovery positions in use today are the best possible. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Adult; Basic life support; Spinal cord; Resuscitation; Safety



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

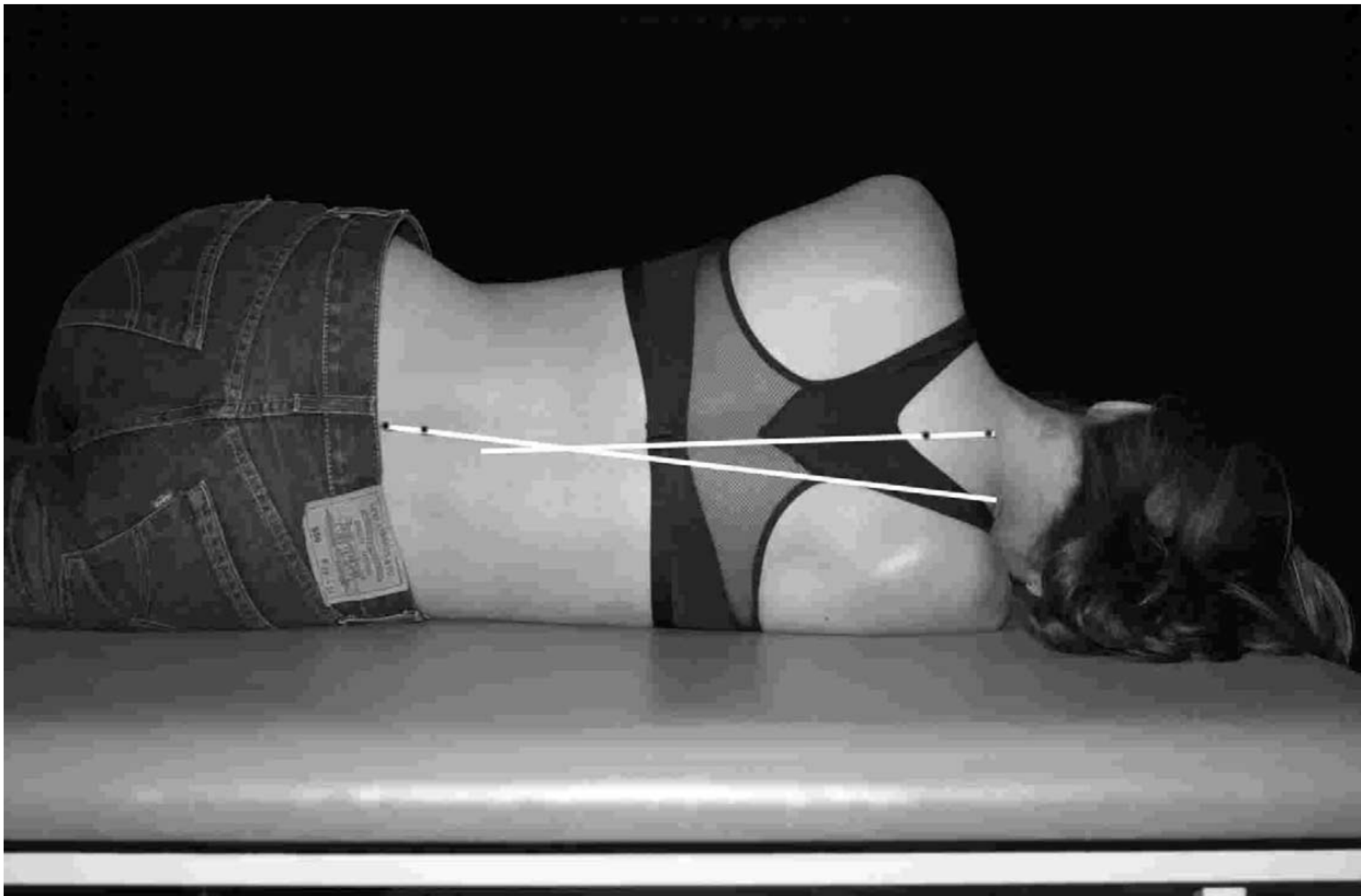


Fig. 4. Thoraco-lumbar lateral flexion was measured by marking the spinous processes of C7, T2, L2 and L4. Here it is 6°.



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	<i>P</i> value
<i>Cervical region</i>					
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
<i>Thoraco-lumbar region</i>					
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.



Annegamento

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Contenuti

- Definizione
- Epidemiologia
- Fisiopatologia
- Manovre di soccorso
- Modifiche al BLSD

GLOBAL DROWNING PREVENTION

KEY FACTS

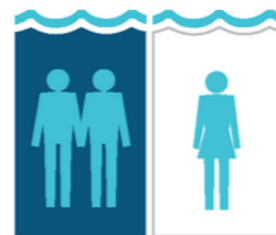
**AT LEAST
236,000**
people die
EVERY YEAR



OVER HALF
of all drowning
deaths are
among those
aged
**UNDER 30
YEARS**



**MALES
ARE TWICE
AS LIKELY**
to drown as
females



Drowning is one
of the
**10 LEADING
CAUSES OF
DEATH**
for people aged
1-24 years



Drowning is formally defined as the process of experiencing respiratory impairment from submersion/immersion in liquid



'Submersion' \Rightarrow the airway going below the level of the surface of the liquid



'Immersion' \Rightarrow liquid being splashed across a person's face.



Immersion

Hot-Water Immersion

“Thermoneutral” is the term for the water temperature at which heat loss equals heat production (53, 238). Most drowning events occur at water temperatures below the point of thermoneutrality, which is $35^{\circ}\text{C} \pm 0.5$. Some drownings, however, occur in hot-water tubs, while pouring hot water over the head, or during diving or competitive swimming in warm water.



↑ humidity of the ambient air ⇒
↓ limited evaporation of sweat above the water
Sweating also occurs **under water** ⇒
This sweat **does not contribute to body cooling**

Skin temperature ↑ ⇒
cutaneous warm thermoreceptors (C-type fiber) interact with keratinocytes through TRPVc → spinal dorsal horn/trigeminal nerve → hypothalamic thermoregulatory centers (pre-optic area) ⇒ cutaneous vasodilatation ⇒

↓ SVR ⇒
HR ⇒
↑ RR ventricular arrhythmias

Sweating ⇒
dehydration ⇒
thrombosis

Leaving the tub ⇒
loss of the hydrostatic squeeze ⇒
Hypotension



Cold-Water Immersion

Most drownings occur in water colder than thermoneutral temperature, thus initiating physiological responses associated with cooling. In cold water, the responses that act as precursors to drowning are evoked by skin cooling (cold shock), then cooling of superficial nerves and muscles in the limbs, and finally cooling of deep body tissues (hypothermia).



Cold Shock

After a fall into cold water, any intention to breath-hold can be overcome by cold shock (261, 263).

The response starts in water $\sim 25^{\circ}\text{C}$ and peaks somewhere between 15 and 10°C ; it peaks in the first 30 s of immersion and attenuates during the next 2–3 min (268). It is evoked by cold receptors located in the superficial sub-epidermal layer of the skin; below $\sim 19^{\circ}\text{C}$, cold nociceptors contribute to the response with a sensation of intense cold pain being experienced in water below $\sim 5^{\circ}\text{C}$ (40, 163). The cold-shock response may be decreased but is still present in those with a high body temperature (159).



Cold receptors respond to the sudden decrease in skin temperature resulting from immersion in cold water with a dynamic response that evokes gasping, hyperventilation, increased cardiac output, peripheral vasoconstriction, and hypertension. These responses, along with a generalized increase in muscle tension, can increase metabolic rate on initial immersion by a factor of four (98). This would, on its own, decrease breath-hold time during initial immersion because the hypoxic and hypercapnic thresholds for the breakpoint of breath-holding would be reached earlier (FIGURE 1). More important, thermo-afferents from the peripheral cold receptors dramatically increase respiratory drive via direct stimulation of the respiratory center (121), with a reflex stimulation at the spinal level of α -motoneurons innervating the intercostal muscles and diaphragm (166, 263). As a consequence, the gasp response and hyperventilation cause an inability to breath-hold. Maximum breath-hold time generally is 60–90 s at a comfortable air temperature and is reduced to just a few seconds in water colder than $\sim 15^{\circ}\text{C}$. The inability to breath-hold represents the most hazardous response to cold-water immer-

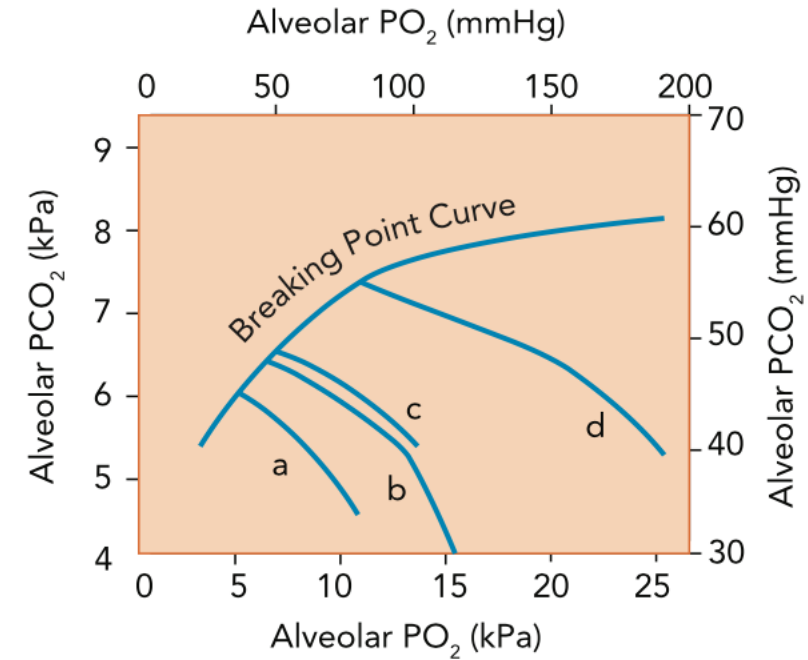


FIGURE 1. The breaking points of breath-holding in different settings

The "breaking point curve" defines the values of alveolar PO₂ and PCO₂ at the breath-hold break point when starting from different states. The normal alveolar starting point is shown. This point is displaced by different maneuvers, and the length of the arrows gives an indication of the changes of the breath-hold duration. See Refs. 73, 196. a, After breathing 15% oxygen; b, after hyperventilation; c, normal alveolar point; d, after breathing 30% oxygen.

At the same time, upon initial immersion in cold water, the incidence of arrhythmias increases from 2% during cold water immersion with head-out-of-water free-breathing to 82% if the cold immersion is associated with face immersion and maximum breath-holding (see section *Diving Response* and *Autonomic Conflict* below).



nective tissues (278). At muscle temperatures below 25°C, fatigue occurs because cooling impairs superficial muscle fibers leaving a smaller number of fibers to produce the same force (50). Maximum dynamic strength, power output, jumping, and sprinting performance have been related to muscle temperature with reductions ranging from 4 to 6% per degree fall in muscle temperature down to 30°C (27, 213).

At nerve temperatures below ~20°C, nerve conduction is slowed and action potential amplitude is decreased (62). Ulnar nerve conduction velocity falls by 15 m/s per 10°C decrease of local temperature. Nerve block may occur at a local temperature of between 5 and 15°C for 1–15 min and lead to a dysfunction that is equivalent to peripheral paralysis (22, 50).



Deep-Tissue Cooling: Hypothermia

With regard to drowning, the most significant consequence of hypothermia is the loss of consciousness (LOC) with deep body cooling. This prevents individuals from undertaking physical activity to maintain a clear airway.

The progressive signs and symptoms are shivering (36°C), confusion, disorientation, introversion (35°C), amnesia (34°C), cardiac arrhythmias (33°C), clouding of consciousness (33-30°C), LOC (30°C), ventricular fibrillation (VF) (28°C), and death (25°C). Below a cardiac temperature of 28°C, the heart may suddenly and spontaneously arrest. VF may result from rough handling of the casualty at deep body temperature of ~28°C (88, 89). Hypothermia affects cellular metabolism, blood flow, and neural function. In severe hypothermia, the patient will be deeply unconscious. The decreased oxygen requirement of cold cells and organs causes decreased respiratory and heart rates. This makes it difficult to detect vital signs in the field. Tendon reflexes are absent and the pupils dilated: this may give the appearance of death (88).



Table 2. Risk factors for immersion hypothermia (88, 119, 167, 213)

- Water temperature: effects being most significant during cold water immersion
- Water movement: faster-moving liquids increase convective heat loss
- Surface area-to-mass area: the higher this ratio, the more cooling is facilitated
- Age: children cool faster than adults due to their lower levels of subcutaneous fat and higher surface area-to-mass ratio
- Body stature: tall, thin individuals cool faster than do those short and obese
- Body morphology: body fat and nonperfused muscle are good insulators
- Gender: females tend to have more subcutaneous fat than men but a weaker shivering response
- Fitness: high fitness level enables greater heat production
- Fatigue: exhaustion results in decreased heat production
- Nutritional state: hypoglycemia attenuates shivering and accentuates cooling
- Intoxication: alcohol and other drug depressants affect metabolism
- Lack of appropriate/specialized clothing



Submersion

Sympathetic Activation, Fear of Drowning

Fear of drowning as a mechanism that results in drowning is most often reported in the gray literature and social media. Several triathletes

Breath-Holding

Under normal circumstances, typical alveolar PCO_2 at the breakpoint ranges between 43 and 53 Torr and occurs 60–90 s after breath-holding with ambient air (FIGURE 2). The breath-holding time can be influenced by several factors, including those listed in Table 3 (196).

In water, important additional physiological factors decrease breath-holding duration, including alcohol intoxication, water temperature below $\sim 15^{\circ}C$, and the cold shock response that intensifies respiratory drive. Other factors that can influence breath-hold time include voluntary liquid aspiration such as occurs in suicides.

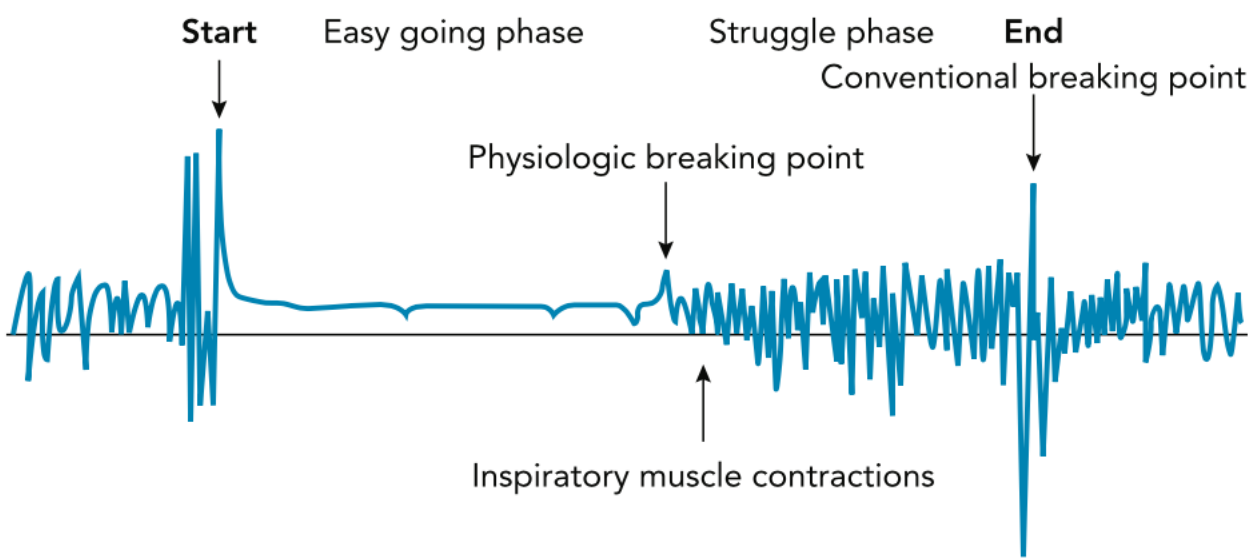


FIGURE 2. Human thoracic movements measured by electromyography (EMG) during maximal breath-holding in an untrained, non-immersed subject
“Easy going phase” and “struggle phase” are distinguished by the absence or presence of respiratory muscle activity that must be suppressed during breath-holding. Figure is from Ref. 72 and used with permission from Saunders.

Table 3. Factors influencing breath-holding duration in air
Metabolic rate during breath-holding
Prebreathing with hyperoxic or hypoxic gas mixtures
Carbon dioxide and oxygen storage capacity
Prior hyperventilation
Experience and psychological tolerance of unpleasant sensations arising during breath-holding

Central Nervous System Centers

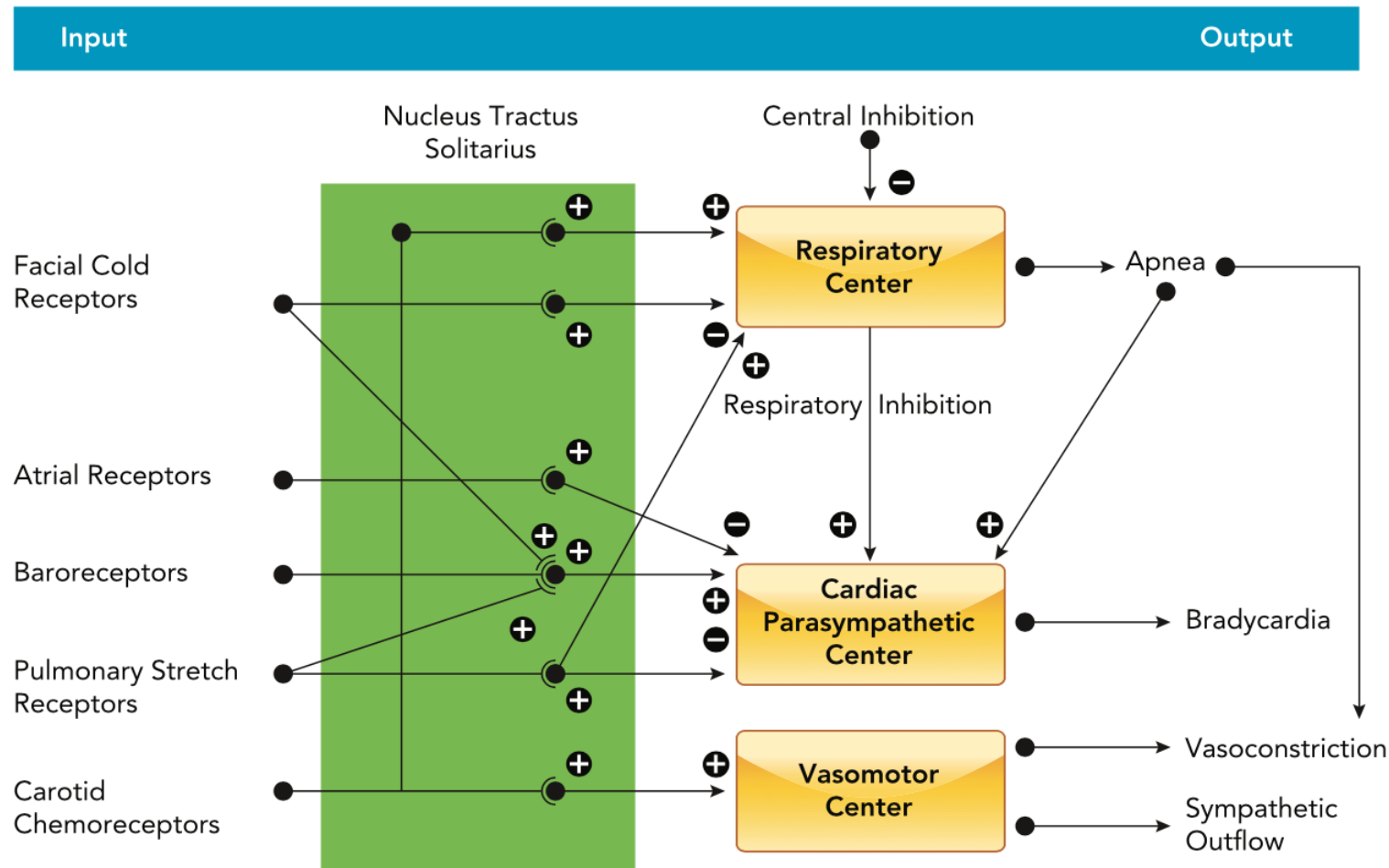


FIGURE 3. Schematic figure of the diving response

Input appears on the *left* and output at the *right*. In between are the neural connections located in the nucleus tractus solitarius and central nervous system control centers. The response is activated through the chemoreceptor sensitivity of the cold receptors of the skin and the unmyelinated C-fibers of the ophthalmic branch of the nervus trigeminus. For more details on the neurological pathways of the diving response, see also Refs. 162, 199, 226, 234. +, Excitatory neural connections, –, inhibitory neural connections. Figure is from Ref. 77 and used with permission from *Scandinavian Journal of Medicine and Science in Sports*.

Table 4. *Factors that increase effects of the diving response (10, 11, 17, 42, 77, 134, 231, 233)*

Precooling of the face

Contact of cold material with the face (water, air, cold packs)

Large air/water temperature gradient

Increased hypoxia

Prolonged or deeper submersion

Posture in the water

Smaller vital capacity lung volume

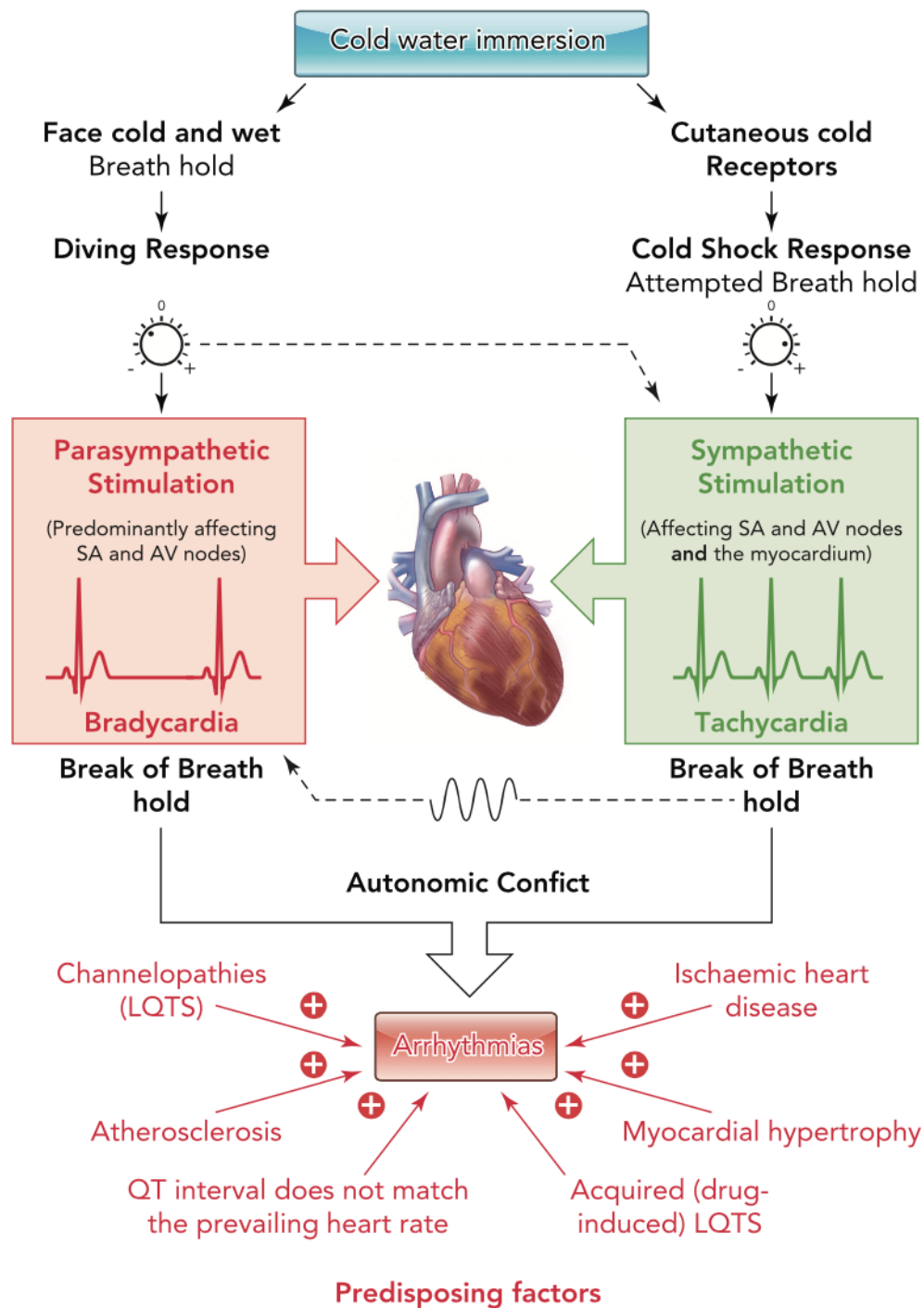
Previous breath-hold diving experiences

A series of repeated apnea dives or apnea exercises

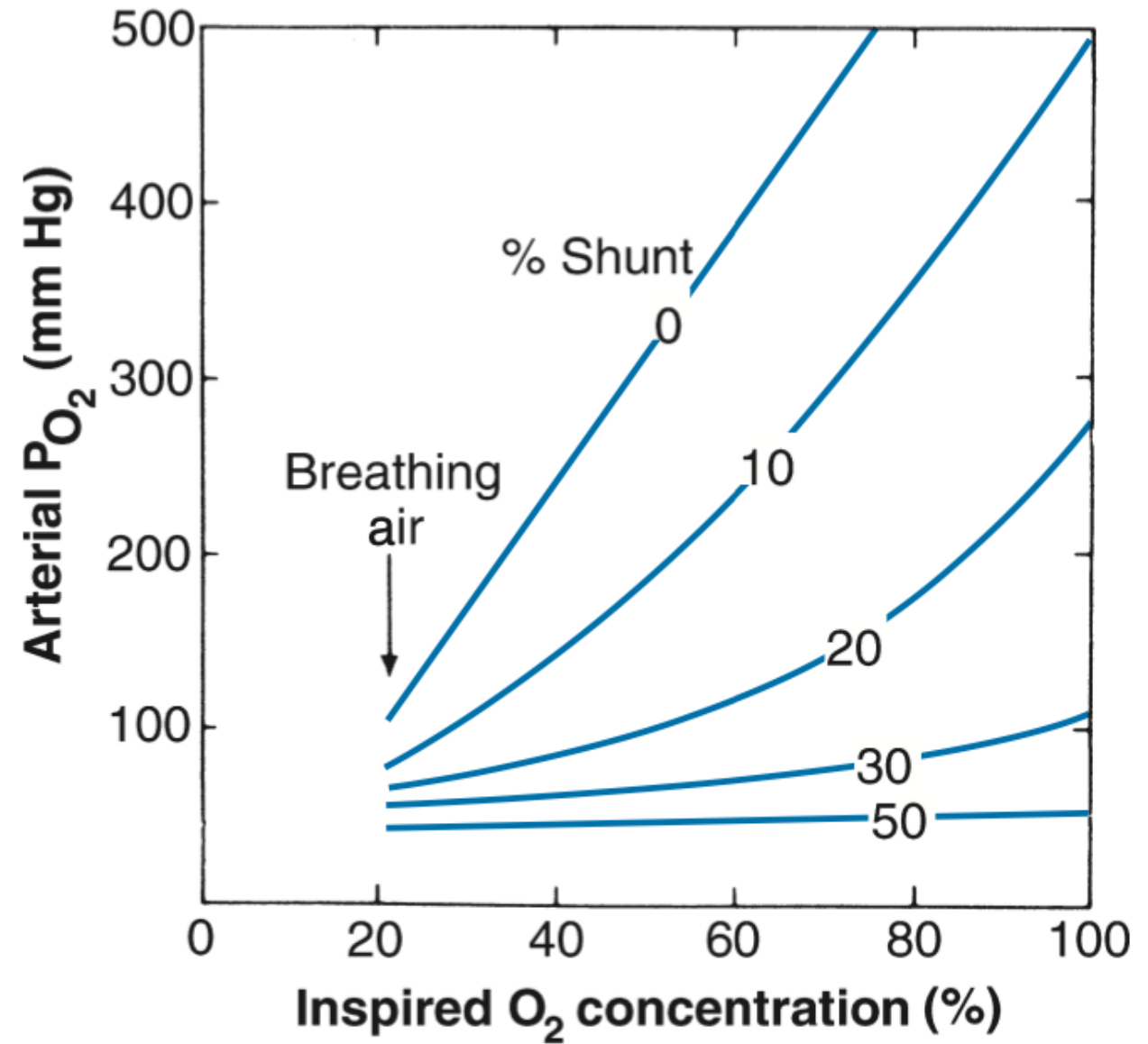
Physical fitness

FIGURE 4. Autonomic conflict

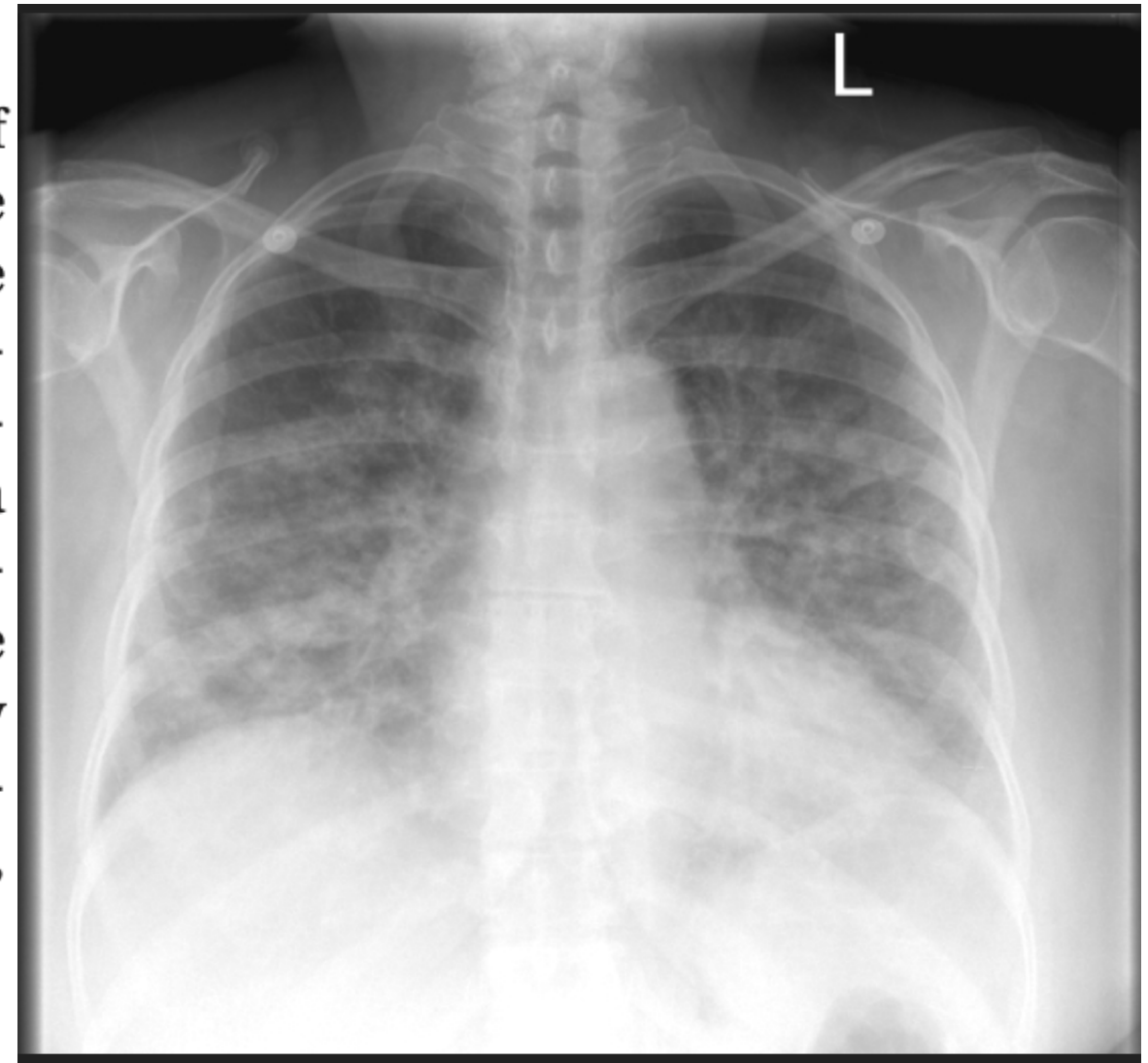
Cold-water immersion activates two powerful responses: the diving response (on facial immersion) and the cold shock response (on the activation of cutaneous cold receptors). The magnitudes of these responses can vary with a range of factors including water temperature, clothing, and habituation. The diving response triggers a parasympathetically driven bradycardia, whereas cold shock activates a sympathetically driven tachycardia. It is hypothesized that together these conflicting inputs to the heart can lead to arrhythmias, particularly at the break of breath-hold, which increases parasympathetic tone that varies with respiration. The substrate for arrhythmias is enhanced by various predisposing factors. Figure is from Ref. 244 and used with permission from *The Journal of Physiology*.



Hypotonic liquid, when reaching the alveoli, damages and dilutes pulmonary surfactant. The increase in the alveolar surface tension, along with diminution of pulmonary compliance, causes alveolar instability and atelectasis that alters the ventilation-to-perfusion ratio. Because a large part of the lung is not adequately ventilated, more venous blood bypasses the lungs, and the shunt fraction increases. Aspiration of 2.5 ml/kg of sea water causes the pulmonary shunt fraction to increase by 75% (206). Hypotonic fresh water tends to be absorbed into the pulmonary circulation and distributed throughout the body. Aspiration of



distributed throughout the body. Aspiration of hypertonic seawater draws liquid from the plasma into the alveoli and also causes damage to surfactant (215). In both situations, the supra-normal hydrostatic forces over the alveolar-capillary membrane will disrupt its integrity. Plasma enters the alveoli, incapacitating normal gas exchange. Plasma in the alveoli may also generate foam that further decreases pulmonary efficiency (147). This results in a local adult respiratory distress syndrome-like clinical picture (85, 92, 176, 274).



Emesis

Detailed data on the occurrence of emesis in drowning are also lacking. One study, reported 25–60% of drowning victims vomited (151). Another study revealed that emesis occurred in 86% of drowning victims who required cardiopulmonary resuscitation and in 50% of those who required no intervention (154). Autopsy series have disclosed aspiration of gastric contents in 24% of drowning victims (80). In a large series on out-of-hospital cardiac arrest (CA) with a cardiac and non-cardiac etiology, emesis occurred in 30–35% of all patients (247). The trigger can be the condition underlying the arrest, CA itself, gastric distension caused by artificial ventilation, or improper chest compression that increases intra-abdominal pressure.

During drowning, gastric contents can be aspirated into the airways, resulting in pulmonary infection and chemical irritation (68, 274). Emesis can also interfere with pulmonary resuscitation. In drowning, both vomiting and cardiopulmonary resuscitation may cause gastric mucosal tears, the frequency of which varies widely among studies but has been detected in as many as 21% of patients (15, 33, 55, 145).

Neurophysiology

The cerebral physiological response to drowning is poorly understood but is most likely an interaction between hypoxemia, submersion liquid temperature, aspiration, and cold shock. Most information pertaining to cerebral physiological responses to drowning is derived from experimental models simulating CA, which may not be directly relevant.

A critical event in drowning is loss of consciousness (LOC). This is often attributed to asphyxia following submersion, loss of pulmonary oxygen uptake, brain energy failure, and deterioration of brain function. Hypoxemia in normothermic healthy humans causes an initial cerebral vasodilatory response to preserve oxygen delivery (3). Progressive hypoxemia leads to a depletion of high-energy phosphates and loss of electrocortical activity consistent with LOC (171). The duration of this state defines the severity of injury and reversibility of neurological dysfunction.



DROWNING

- Drowning victims need early rescue breaths
- Safety of rescuer is very important, but victim should be removed from water as soon as possible
- Rescue breaths can be given whilst in the water *if you are trained to do so*
- AED can be used (on dry land or in rescue boat) if victim's chest is dried

Approach safely

Check response

Shout for help

Open airway

Check breathing

Send someone to call 112

5 rescue breaths

30 chest compressions

2 rescue breaths

If alone, call 112 after 1 minute



Cardiac arrest

- Start resuscitation as soon as safe and practical to do so. If trained and able this might include initiating ventilations whilst still in the water or providing ventilations and chest compressions on a boat.
- Start resuscitation by giving 5 rescue breaths/ventilations using 100% inspired oxygen if available.
- If the person remains unconscious, without normal breathing, start chest compressions.
- Alternate 30 chest compressions to 2 ventilations.
- Apply an AED if available and follow instructions.
- Intubate the trachea if able to do so safely.
- Consider ECPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.



Treatment Recommendations



We recommend that submersion duration be used as a prognostic indicator when making decisions surrounding search and rescue resource management/operations (strong recommendation, moderate-certainty evidence for prognostic significance).

We suggest against the use of age, EMS response time, water type (fresh or salt), water temperature, and witness status when making prognostic decisions (weak recommendation, very-low-certainty evidence for prognostic significance).

We acknowledge that this review excluded exceptional and rare case reports that identify good outcomes after prolonged submersion in icy cold water.