UNIVERSITÀ DEGLI STUDI
DI CAGLIARI

Shock

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Definizione

- "The state in which profound and widespread reduction of **effective** tissue perfusion leads first to reversible, and then, if prolonged, to irreversible cellular injury."
- Effective" tissue perfusion, as opposed to tissue perfusion per se, is an important issue. Effective tissue perfusion may be reduced by either a global **reduction of systemic perfusion** (CO) or by increased ineffective tissue perfusion owing to a **maldistribution of blood flow** or a **defect of substrate utilization** at the subcellular level

Parrillo JE, Dellinger RP. Critical Care Medicine E-Book: Principles of Diagnosis and Management in the Adult. Elsevier Health Sciences; 2018.

Definizione

Shock is best defined as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells. It is a state in which the circulation is unable to deliver sufficient oxygen to meet the demands of the tissues, resulting in cellular dysfunction. The result is cellular dysoxia, i.e. the loss of the physiological independence between oxygen delivery and oxygen consumption, associated with increased lactate levels. Some clinical symptoms suggest an impaired microcirculation, including mottled skin, acrocyanosis, slow capillary refill time and an increased central-to-toe temperature gradient.

Cecconi M, De Backer D, Antonelli M, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40(12):1795-1815. doi:10.1007/s00134-014-3525-z

Classificazione

TABLE 44.1 Shock Classification

Hypovolemic

Hemorrhagic

- Trauma, gastrointestinal, retroperitoneal

Nonhemorrhagic

- Dehydration, emesis, diarrhea, fistulae, burns, polyuria, "third spacing," malnutrition, large open wounds

Cardiogenic

Myocardial

- Infarction, contusion, myocarditis, cardiomyopathies, pharmacologic

Mechanical

- Valvular failure, ventricular septal defect, ventricular wall defects

Dysrhythmias

Obstructive

Impairment of diastolic filling

- Intrathoracic obstructive tumors, tension pneumothorax, positive-pressure mechanical ventilation, constrictive pericarditis, pericardial tamponade

Impairment of systolic contraction

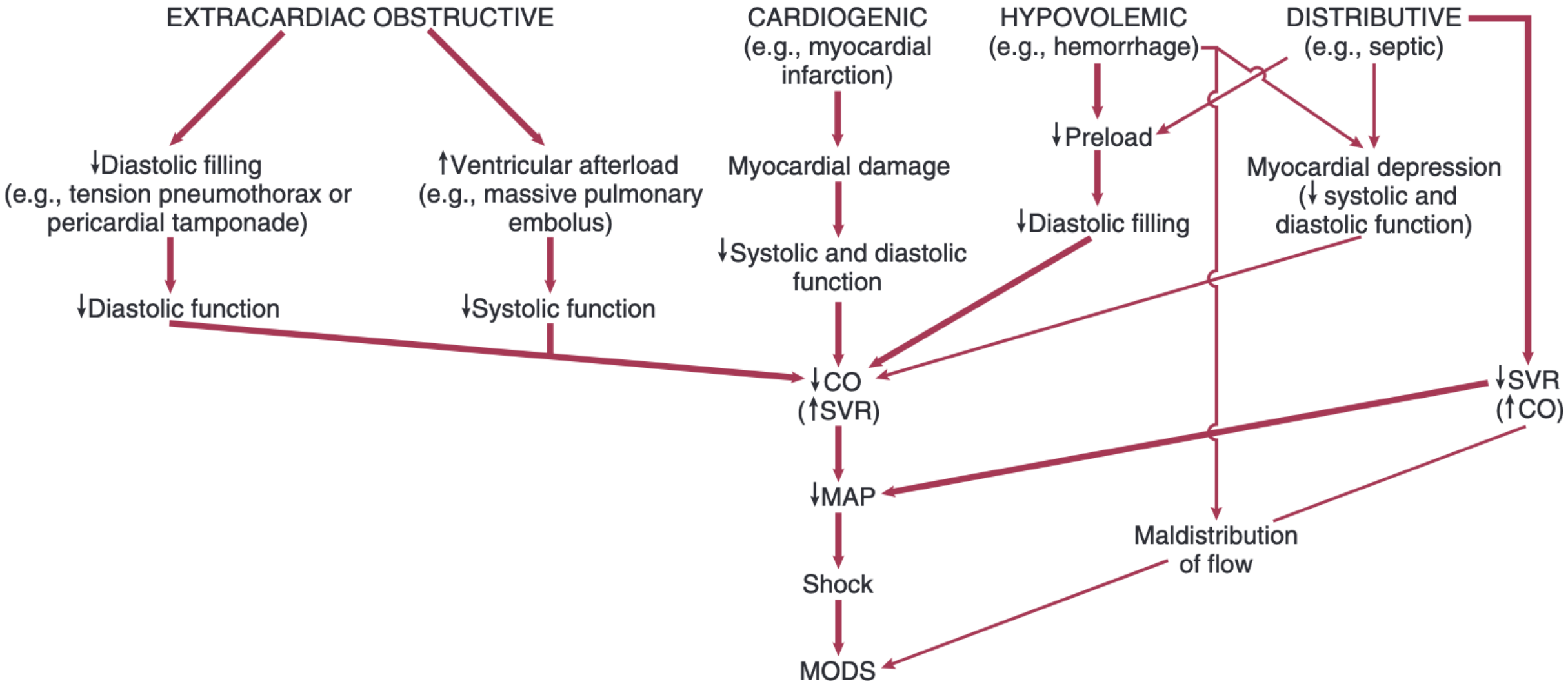
- Pulmonary embolism, acute pulmonary hypertension, air embolism, tumors, aortic dissection, aortic coarctation

Distributive

Septic, anaphylactic, neurogenic, pharmacologic, endocrinologic

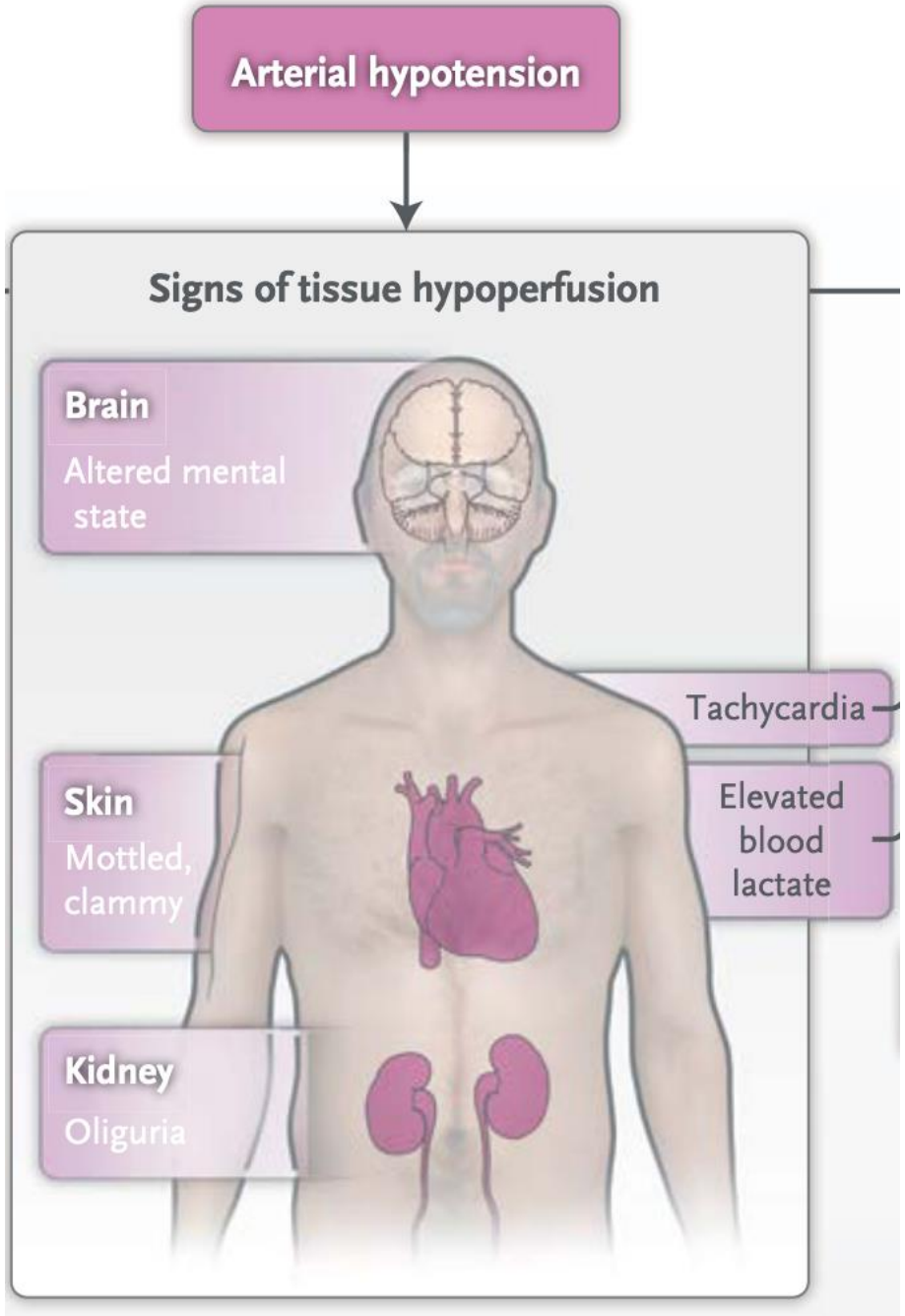
Layon AJ, Gabrielli A, Yu M, Wood KE. Civetta, Taylor & Kirby's Critical Care. Wolters Kluwer; 2018.

Profilo emodinamico



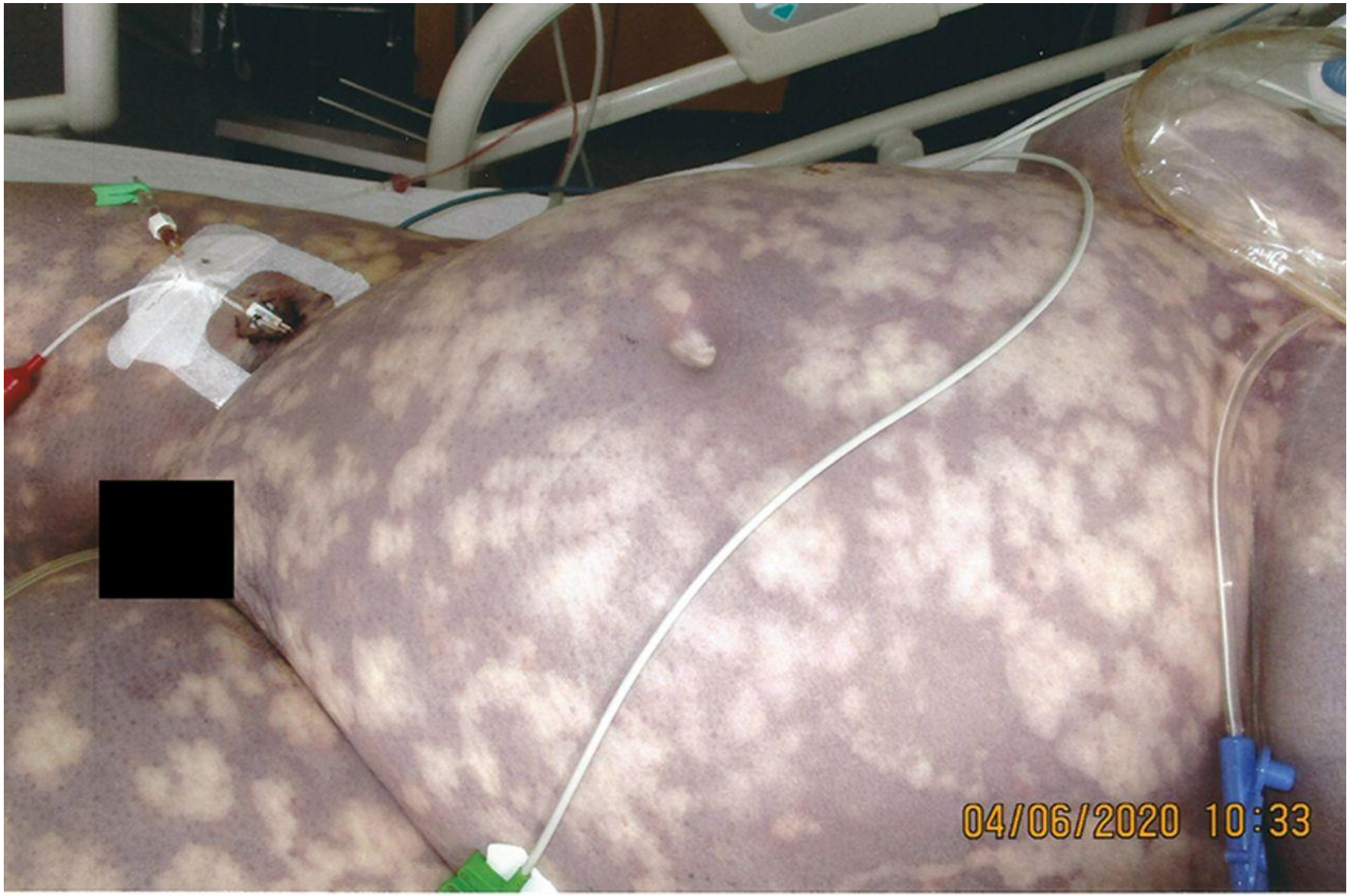
Parrillo JE, Dellinger RP. Critical Care Medicine E-Book: Principles of Diagnosis and Management in the Adult. Elsevier Health Sciences; 2018.

Segni clinici



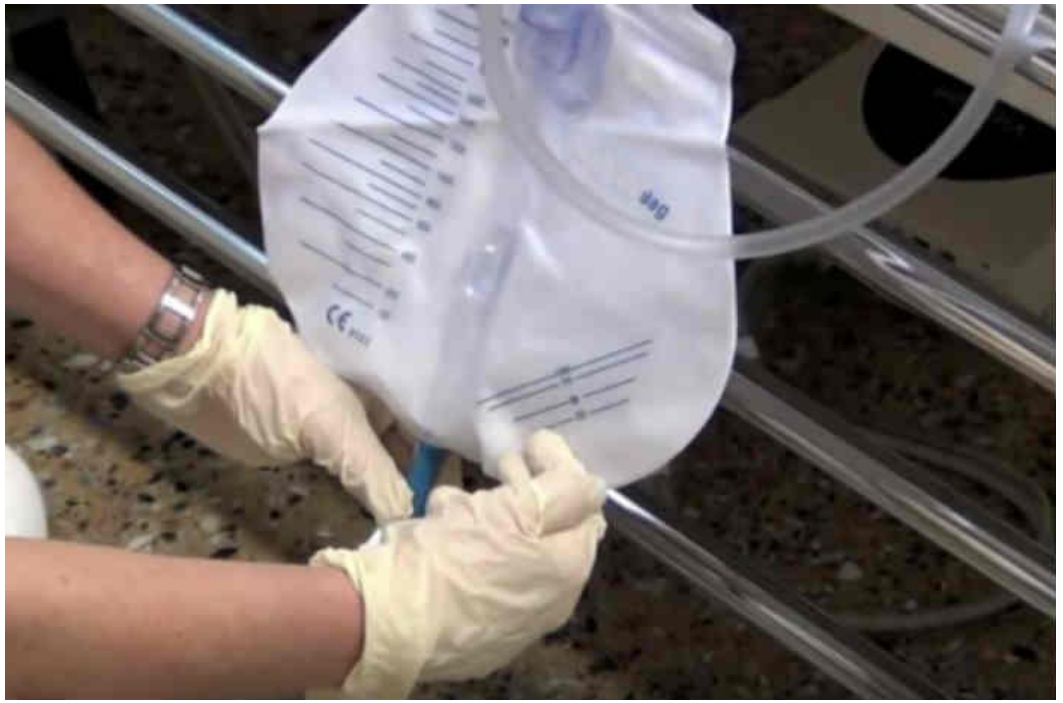
Vincent JL, De Backer D. Circulatory shock. N Engl J Med. 2013;369(18):1726-1734. doi:10.1056/NEJMra1208943

Finestra 1: cute



Storz MA, Seidlitz F. Severe skin mottling in fatal sepsis. Postgraduate Medical Journal. Published online November 6, 2020. doi:10.1136/postgradmedj-2020-139119

Finestra 2: reni



Oliguria < 0.5 ml/kg/h

Finestra 3: cervello

Profilo emodinamico

	CVP	PCWP	CO	SVR	$S\bar{v}o_2$
Hypovolemic	↓↓	↓↓	↓↓	↑	↓
Cardiogenic					
Left ventricular myocardial infarction	Nl or ↑	↑	↓↓	↑	↓
Right ventricular myocardial infarction	↑↑	Nl or ↑	↓↓	↑	↓
Obstructive					
Pericardial tamponade	↑↑	↑↑	↓ or ↓↓	↑	↓
Massive pulmonary embolism	↑↑	Nl or ↓	↓↓	↑	↓
Distributive					
Early	Nl or ↑	Nl	↓ or Nl or ↑	↑ or Nl or ↓	Nl or ↓
Early after fluid administration	Nl or ↑	Nl or ↑	↑	↓	↑ or Nl or ↓
Late	Nl	Nl	↓	↑	↑ or ↓

CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CO, cardiac output; SVR, systemic vascular resistance; $S\bar{v}o_2$, mixed venous oxygen saturation; Nl, normal.

Layon AJ, Gabrielli A, Yu M, Wood KE. Civetta, Taylor & Kirby's Critical Care. Wolters Kluwer; 2018.

Domande o interventi?

Principi di gestione dello shock

- **A Airway** vie aeree sicure? intubazione necessaria?
- **B Breathing** supporto ventilatorio? Invasivo vs non-invasivo?
- **C Circulation** Supporto con inocostrittori/inodilatatori? Supporto Meccanico?
- **D Damage control** Terapia mirata a contenere il danno d'organo
- **E Exposure** Valutare accuratamente segni
- **F Fluid and Kidney** AKI? CRRT? Alterazioni elettroliti? Segni di sovraccarico?
- **G GI** Nutrizione? Ileo? Ipertensione addominale? Traslocazione batterica? Necessaria laparotomia esplorativa?
- **I Infection/Inflammation**
- **S Source control**

Supporto vasoattivo

Table 7.5 Hemodynamic effects of vasopressors and inotropes[a]

Drug type, generic (brand)	HR	MAP	PCWP	SVRI	CO	Receptor activity
Vasopressors						
Dopamine	↑↑					
<3		0	0	0	0	D > b1
3–10		↑	↑	0	1	b1 > b2 > D
>10		↑↑	↑↑	↑	↑	a1 > b1 >> b2
Epinephrine	↑↑↑					
<0.05		0	0	0/↓	↑↑↑	b1 >> b2
0.05–0.15		↑	↑↑	↑	↑↑↑	b1 > a1 > b2
>0.15		↑↑	↑↑	↑↑↑	↑↑	a1 > b1
Norepinephrine	↑↑	↑↑↑	↑↑↑	↑↑↑	0	a1 >> b1
Phenylephrine	0	↑↑	↑↑	↑	0	a1
Vasopressin						
Inotropes						
Dobutamine (Dobutrex[1])	↑					b1 > b2 > a1
2–10		↑	↓	↑	↑	
10–20		0/↓	↓	0/↓	↑↑	
Isoproterenol (Isuprel[1])		↓↓	↓↓	↓↓↓	↑↑↑	b1 >> b2
Amrinone (Inocor[1])	↑	0	↓	↓↓	↑	PDE III inhibitor
Milrinone	↑	↓↓	↓	↓↓	↑↑	PDE III inhibitor
Levosimendan	0	↓↓	↓	↓↓	↑↑	Ca sensitizer

[a]All listed doses are in mcg/kg/min

D dopamine receptor, *b1* beta 1, *b2* beta 2, *a1* alpha, *v1* vasopressin receptor; *PDE* phosphodiesterase, *Ca* calcium

Askari AT, Messerli AW. *Cardiovascular Hemodynamics: An Introductory Guide*. Springer; 2019.

SIAARTI
PRO VITA CONTRA DOLOREM SEMPER

BUONE PRATICHE CLINICHE

Uso dei farmaci vasopressori e inotropi nei pazienti critici

AUTORI
Andrea Carsetti, Elena Bignami, Andrea Cortegiani, Katia Donadello, Abele Donati, Giuseppe Foti, Giacomo Grasselli, Stefano Romagnoli, Massimo Antonelli, Elvio De Blasio, Francesco Forfori, Fabio Guarracino, Sabino Scolletta, Luigi Tritapepe, Luigia Scudeller, Maurizio Cecconi, Massimo Girardis

GESTIONE INIZIALE DEL PAZIENTE CON SHOCK

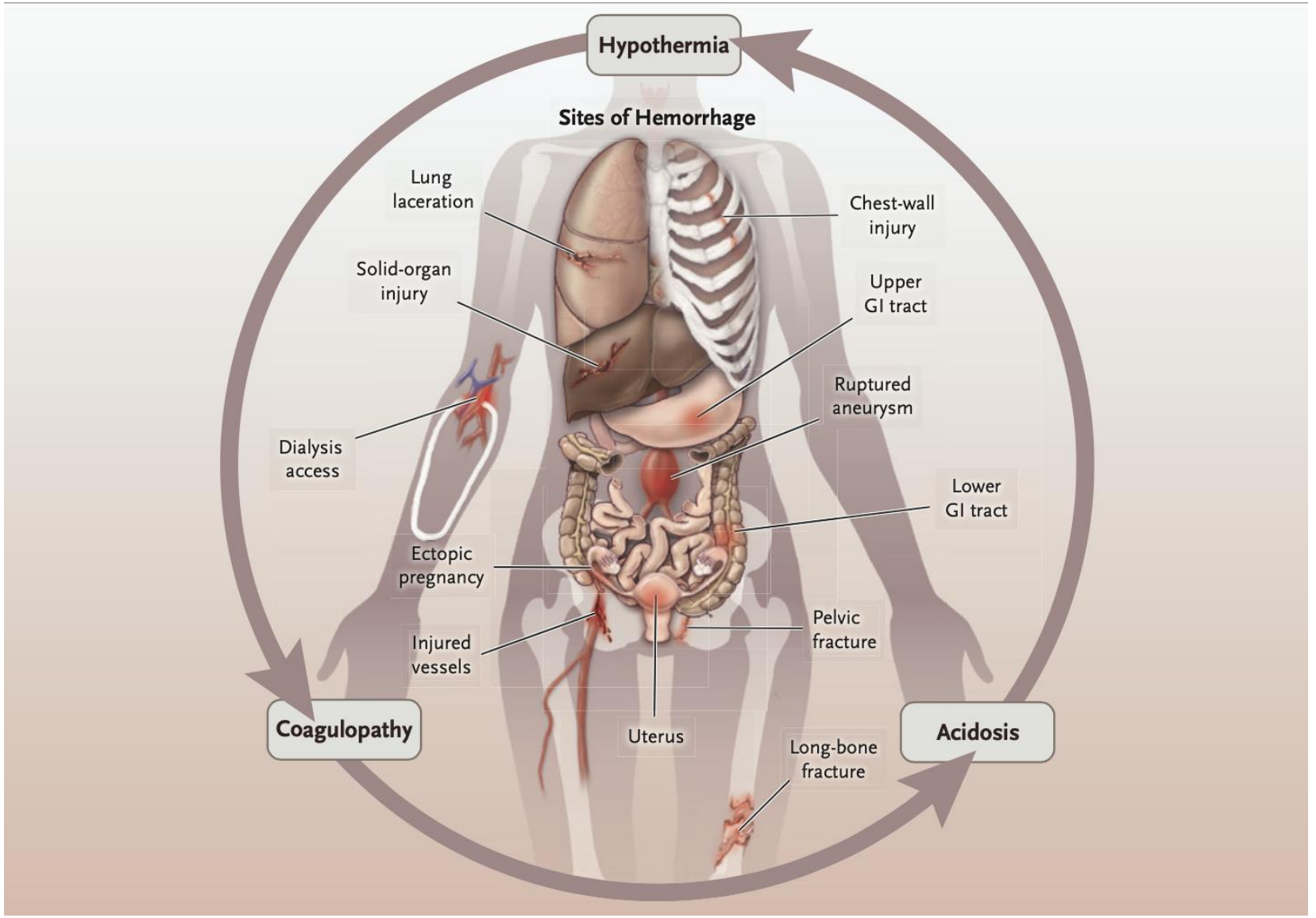
VALUTAZIONE IPOPERFUSIONE
- coscienza
- diuresi
- cute
- lattati > 2 mmol/l
- $SCVO_2$ < 70%
- Pv-aCO_2 > 6 mmHg

OTTIMIZZAZIONE DO_2

- **GITTATA CARDIACA**
  - **GITTATA SISTOLICA**
    - **OTTIMIZZAZIONE VOLEMICA**
      - **VALUTAZIONE FLUIDO-RESPONSIVITÀ**
        - parametri dinamici
        - passive leg raising
        - fluid challenge
      - **MAP < 65mmHg** → **VASOPRESSORE**
      - **DISFUNZIONE SISTOLICA** → **INOTROPO**
        - ← **ECOCARDIOGRAFIA**
  - **FREQUENZA CARDIACA** → **FC 80-100 bpm**
- **ANEMIA** → **EMAZIE** → **Hb 7-9 g/d**
- **IPOSSIA** → **OSSIGENO VENTILAZIONE MECCANICA** → **SpO_2 92-98%**

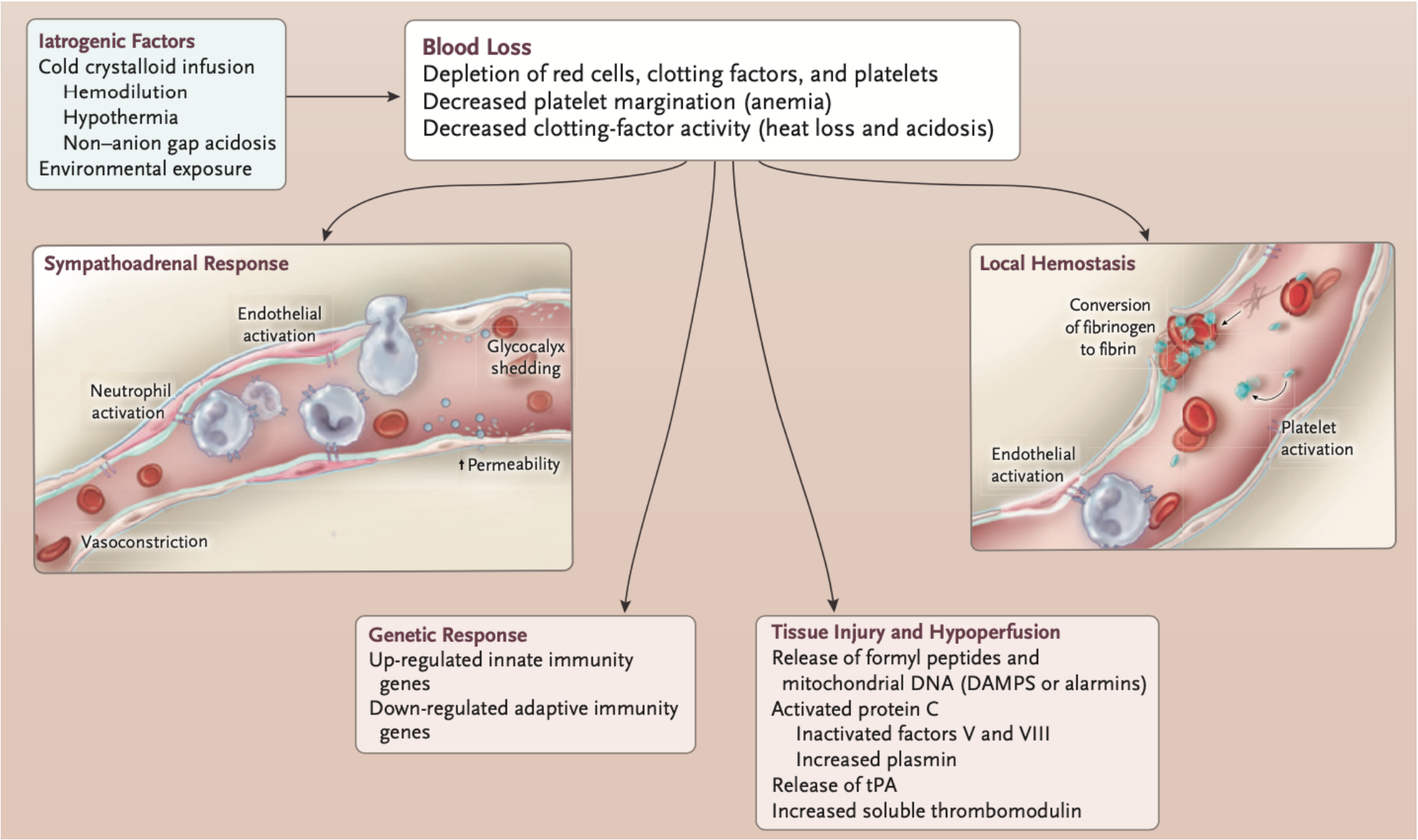
Domande o interventi?

Shock emorragico



Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649

Fisiopatologia



Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649

Classificazione

Table 2. Classification of Hemorrhagic Shock.*

Shock Class	Blood Loss†	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Mental Status
	ml (%)	*beats/min*			*breaths/min*	
I	<750 (15)	<100	Normal	Normal	14–20	Slightly anxious
II	750–1500 (15–30)	100–120	Normal	Narrowed	20–30	Mildly anxious
III	1500–2000 (30–40)	120–140	Decreased	Narrowed	30–40	Anxious, confused
IV	>2000 (>40)	>140	Decreased	Narrowed	>35	Confused, lethargic

* Data are from the American College of Surgeons Committee on Trauma.[42]
† Blood-loss volume and percentage of total blood volume are for a male patient with a body weight of 70 kg.

Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649

DCR

Table 3. Principles of Damage-Control Resuscitation.

Avoid or correct hypothermia
Apply direct pressure or a tourniquet proximal to sites of hemorrhage in the extremities; pack junctional wounds with hemostatic dressings
Delay fluid administration until the time of definitive hemostasis in selected patients (those with penetrating trauma to the torso and short prehospital transport times)
Minimize crystalloid infusions (<3 liters in the first 6 hr)
Use a massive-transfusion protocol to ensure that sufficient blood products are rapidly available
Avoid delays in definitive surgical, endoscopic, or angiographic hemostasis
Minimize imbalances in plasma, platelet, and red-cell transfusions in order to optimize hemostasis
Obtain functional laboratory measures of coagulation (e.g., by means of thromboelastography or rotational thromboelastometry) to guide the transition from empirical transfusions to targeted therapy
Selectively administer pharmacologic adjuncts to reverse any anticoagulant medications and to address persistent coagulopathy

Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649

Domande o interventi?

Shock cardiogeno: definizione

Table 1. Clinical Features of CS as Defined in Contemporary Trials and Guidelines (Table view)

Clinical Trial/Guideline	CS Criteria
SHOCK Trial (1999)[3]	• SBP <90 mm Hg for >30 min or vasopressor support to maintain SBP >90 mm Hg • Evidence of end-organ damage (UO <30 mL/h or cool extremities) • Hemodynamic criteria: CI <2.2 and PCWP >15 mm Hg
IABP-SOAP II (2012)[4]	• MAP <70 mm Hg or SBP <100 mm Hg despite adequate fluid resuscitation (at least 1 L of crystalloids or 500 mL of colloids) • Evidence of end-organ damage (AMS, mottled skin, UO <0.5 mL/kg for 1 h, or serum lactate >2 mmol/L)
EHS-PCI (2012)[5]	• SBP <90 mm Hg for 30 min or inotropes use to maintain SBP >90 mm Hg • Evidence of end-organ damage and increased filling pressures
ESC-HF Guidelines (2016)[6]	• SBP <90 mm Hg with appropriate fluid resuscitation with clinical and laboratory evidence of end-organ damage • Clinical: cold extremities, oliguria, AMS, narrow pulse pressure. Laboratory: metabolic acidosis, elevated serum lactate, elevated serum creatinine
KAMIR-NIH (2018)[7]	• SBP <90 mm Hg for >30 min or supportive intervention to maintain SBP >90 mm Hg • Evidence of end-organ damage (AMS, UO <30 mL/h, or cool extremities)

AMS indicates altered mental status; CI, cardiac index; EHS PCI, Euro Heart Survey Percutaneous Coronary Intervention Registry; ESC HF, European Society of Cardiology Heart Failure; IABP-SOAP II, intra-aortic balloon pump in cardiogenic shock II; KAMIR-NIH, Korean Acute Myocardial Infarction Registry-National Institutes of Health; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; SHOCK, Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock; UO, urine output.

Vahdatpour C, Collins D, Goldberg S. Cardiogenic Shock. J Am Heart Assoc. 2019;8(8):e011991. doi:10.1161/JAHA.119.011991

Cause di shock cardiogeno

Table 3. Major causes of cardiogenic shock

Acute coronary syndrome	Other
MI (LV or RV[a] pump failure)	End-stage cardiomyopathy
Mechanical complications of MI[a]	Acute fulminant myocarditis
Ventricular septal rupture[a]	Stress cardiomyopathy (takotsubo syndrome)
Papillary/Chordal rupture with severe MR[a]	Acute aortic dissection
Ventricular free wall rupture[a]	Cardiac tamponade[a]
Arrhythmia	HCM with dynamic LVOT obstruction[a]
Severe bradycardia or complete AVB[a]	Acute pulmonary embolism[a]
Tachycardia-induced cardiomyopathy	Ventricular assist device malfunction
Rapid atrial fibrillation[a]	Septic cardiomyopathy
Ventricular tachycardia (with pulse)	Medication overdose (negative inotropic agents)
Valvular	Postcardiotomy
Acute regurgitation (AR, MR)[a]	Pulmonary hypertension[a]
Aortic or mitral stenosis[a]	Peripartum cardiomyopathy
Prosthetic valve dysfunction[a]	Heart transplant rejection
Valve obstruction (mass, thrombus, vegetation)[a]	Amyloid cardiomyopathy[a]

AR, aortic regurgitation; AVB, atrio-ventricular block; HCM, hypertrophic cardiomyopathy; LV, left ventricle; LVOT, left ventricular outflow tract; MI, myocardial infarction; MR, mitral regurgitation; RV, right ventricle.
[a]Denotes causes which may have preserved left ventricular ejection fraction (LVEF) [35].

Jentzer JC, Tabi M, Burstein B. Managing the first 120 min of cardiogenic shock: from resuscitation to diagnosis. Curr Opin Crit Care. 2021;27(4):416-425. doi:10.1097/MCC.0000000000000839

Box 1 | **Causes of cardiogenic shock**

Acute myocardial infarction

Left ventricular failure

- Large infarction
- Small/moderate infarction with
  - pre-existing dysfunction
  - extensive ischaemia
- Global ischaemia

Right ventricular failure

- Large infarction
- Small/moderate infarction with
  - pre-existing dysfunction
  - pulmonary hypertension

Mechanical complications

- Acute mitral regurgitation with
  - rupture of a papillary muscle
  - rupture of chordae tendinae
  - severe papillary muscle dysfunction
- Ventricular septal defect caused by rupture of the interventricular septum
- Left ventricular free wall rupture
- Pericardial tamponade owing to rupture of the left ventricular free wall or haemorrhagic pericardial effusion

Concomitant conditions causing mixed aetiology

- Haemorrhage
- Infection
- Excess negative inotropic or vasodilator medications
- Sustained bradyarrhythmia or tachyarrhythmia
- Hyperglycaemia or ketoacidosis

Other conditions

End-stage cardiomyopathy

Myocarditis

Septic shock with severe myocardial depression

Left ventricular outflow tract obstruction

- Aortic stenosis
- Hypertrophic obstructive cardiomyopathy

Obstruction to left ventricular filling

- Mitral stenosis
- Left atrial myxoma

Acute mitral regurgitation (chordal rupture)

Acute aortic insufficiency

Myocardial contusion

Postcardiotomy shock

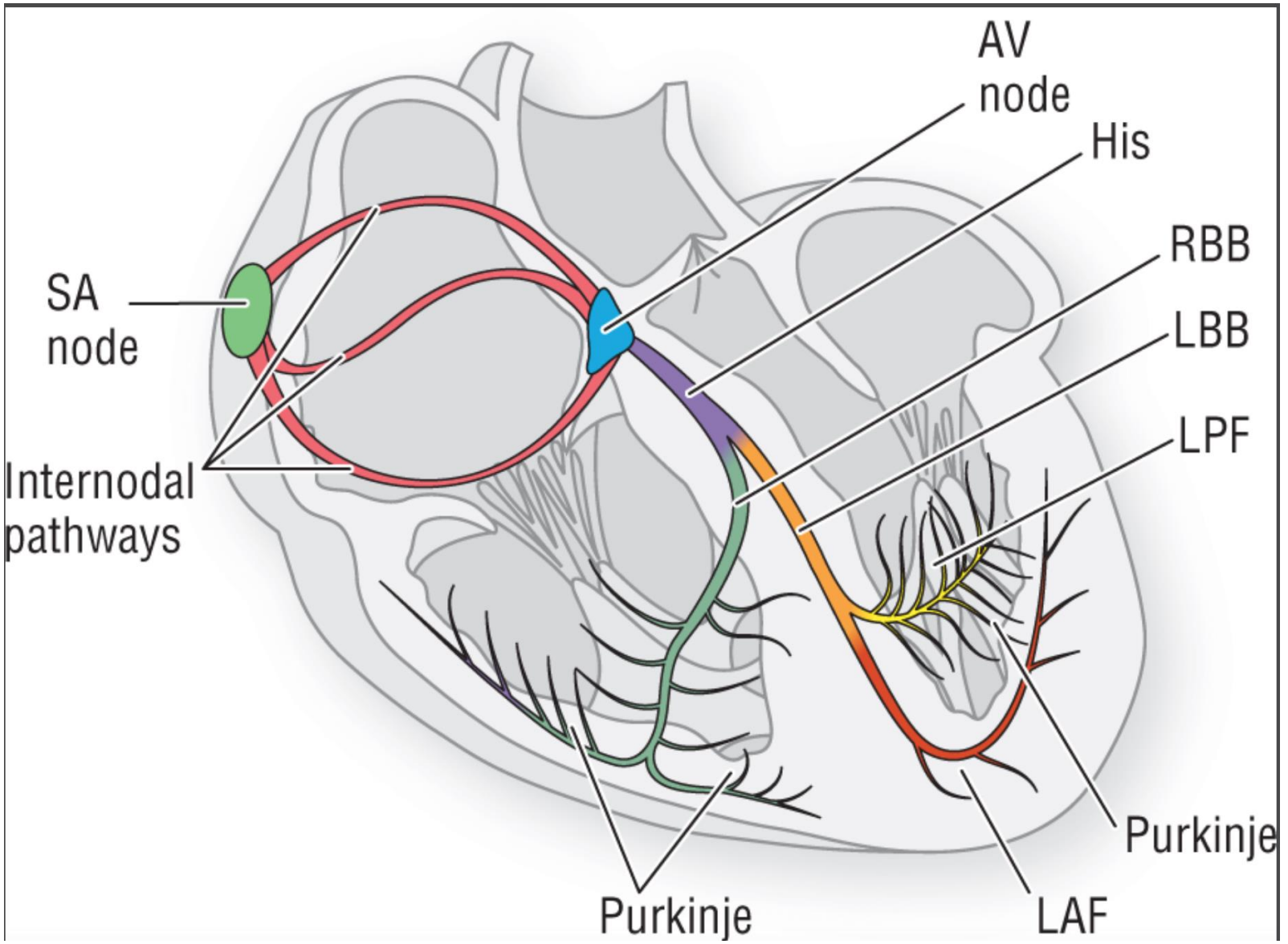
Global ischaemia

Stress-induced cardiomyopathy

Cardiac tamponade

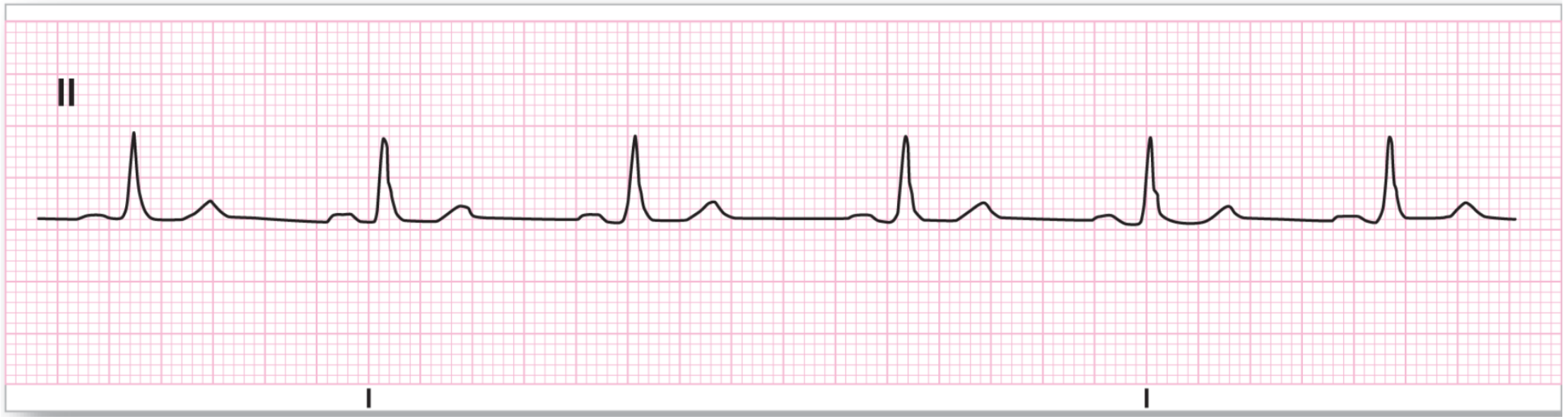
Reyentovich A, Barghash MH, Hochman JS. Management of refractory cardiogenic shock. Nat Rev Cardiol. 2016;13(8):481-492. doi:10.1038/nrcardio.2016.96

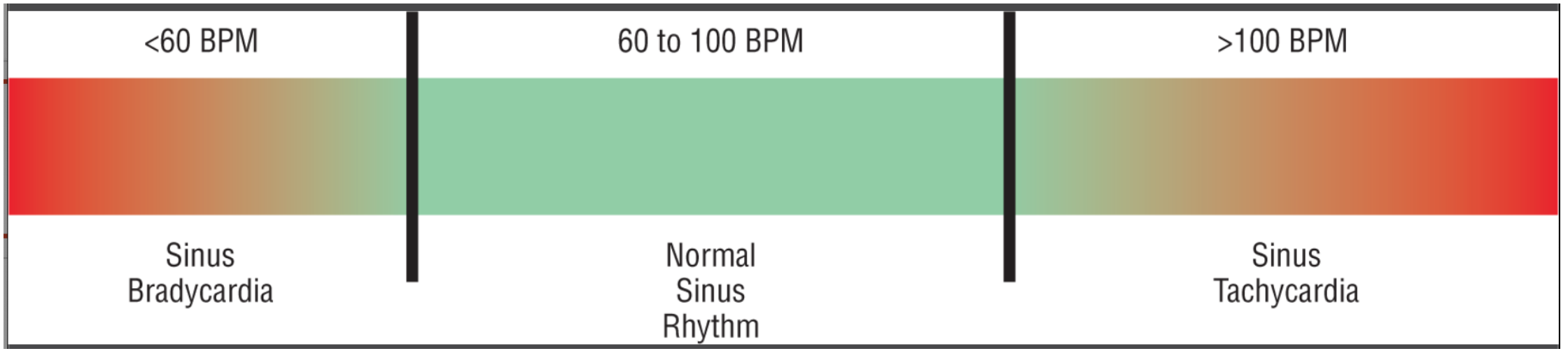
Aritmie



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

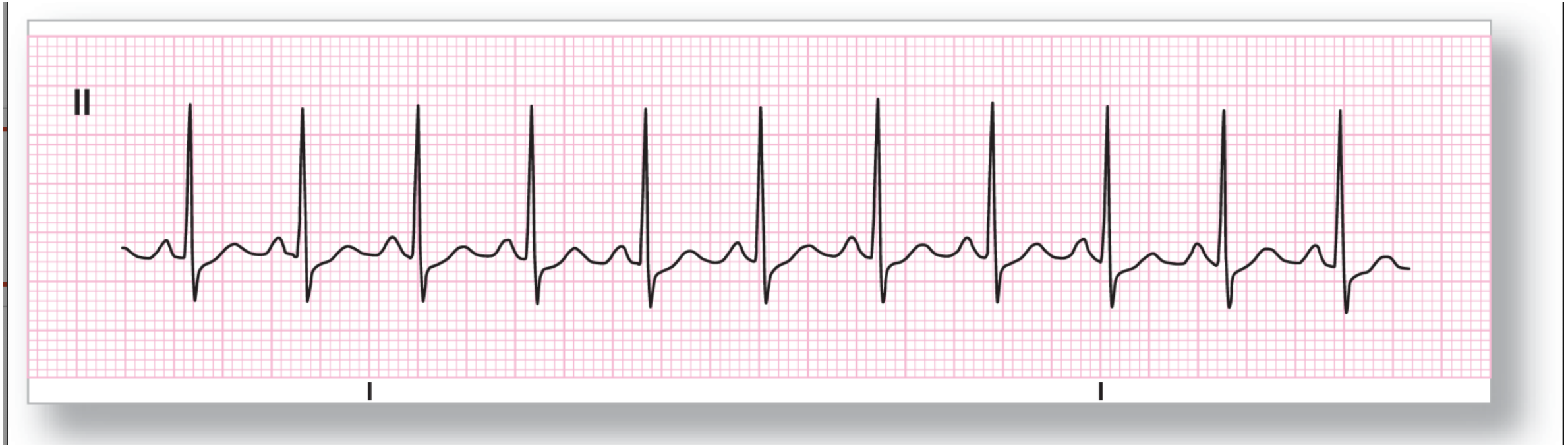
Ritmo sinusale





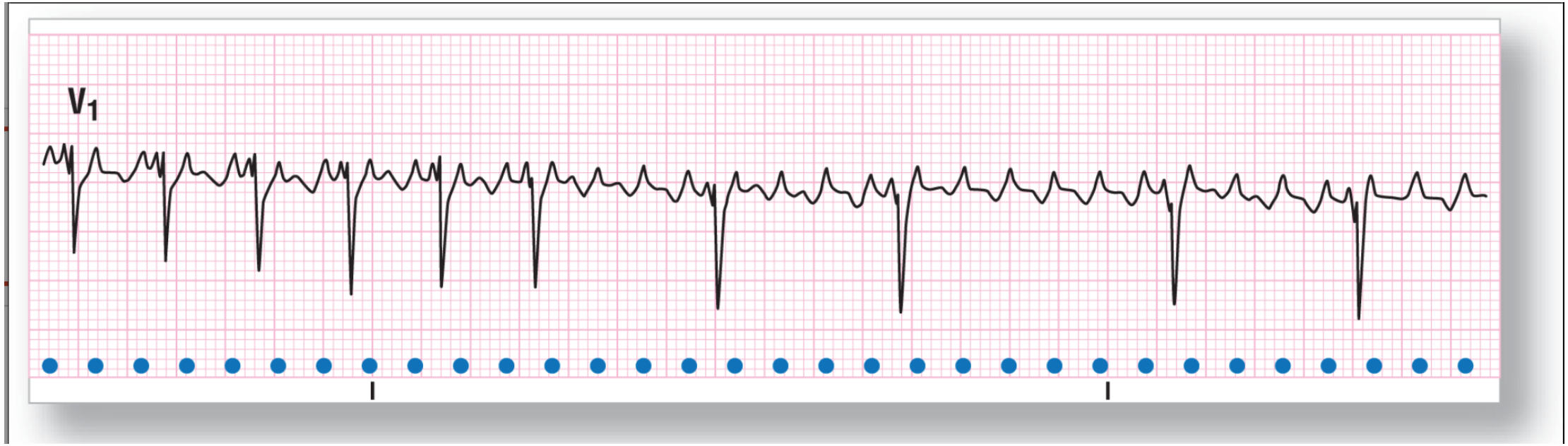
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

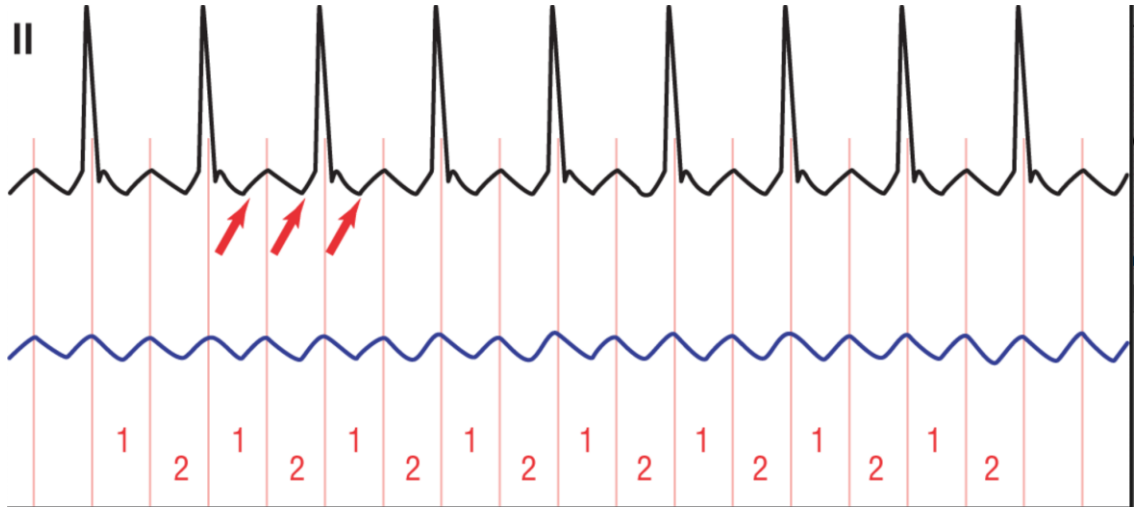
Tachicardia sinusale



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

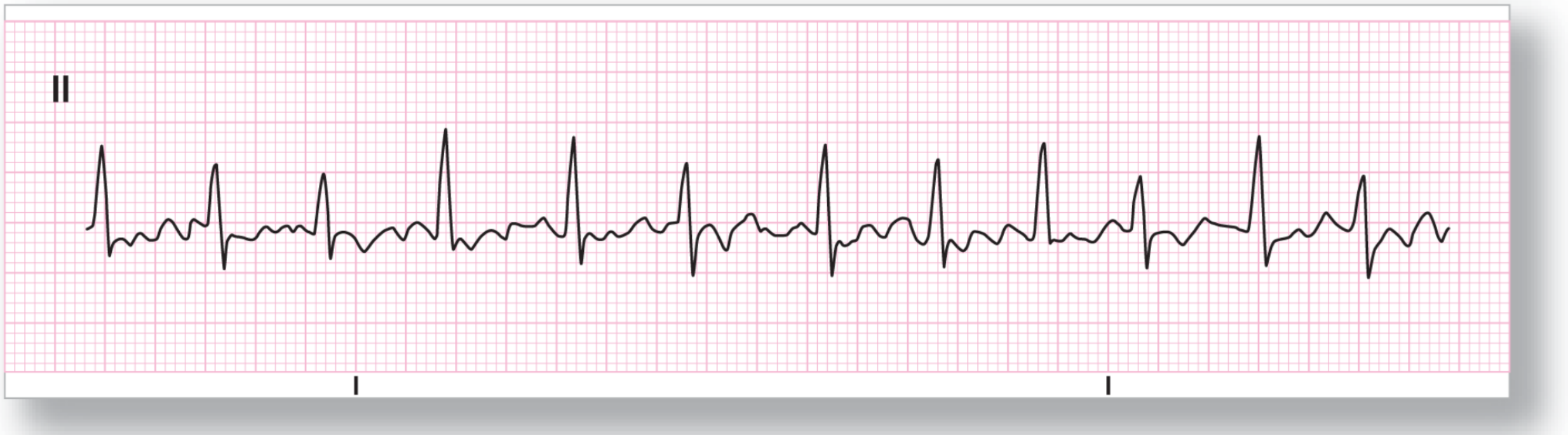
Flutter atriale





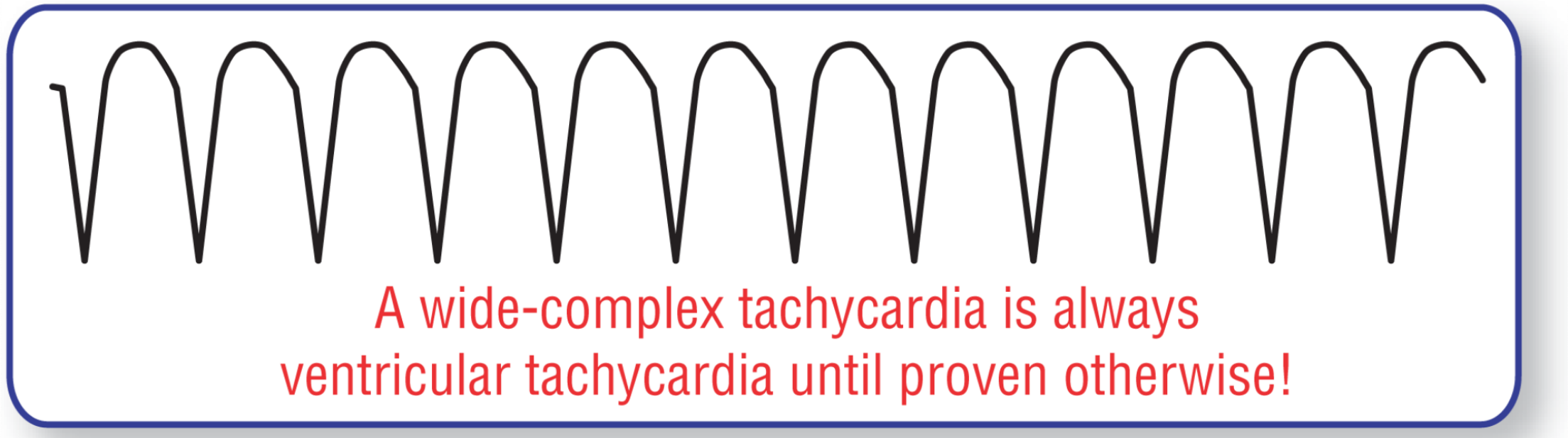
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Fibrillazione atriale



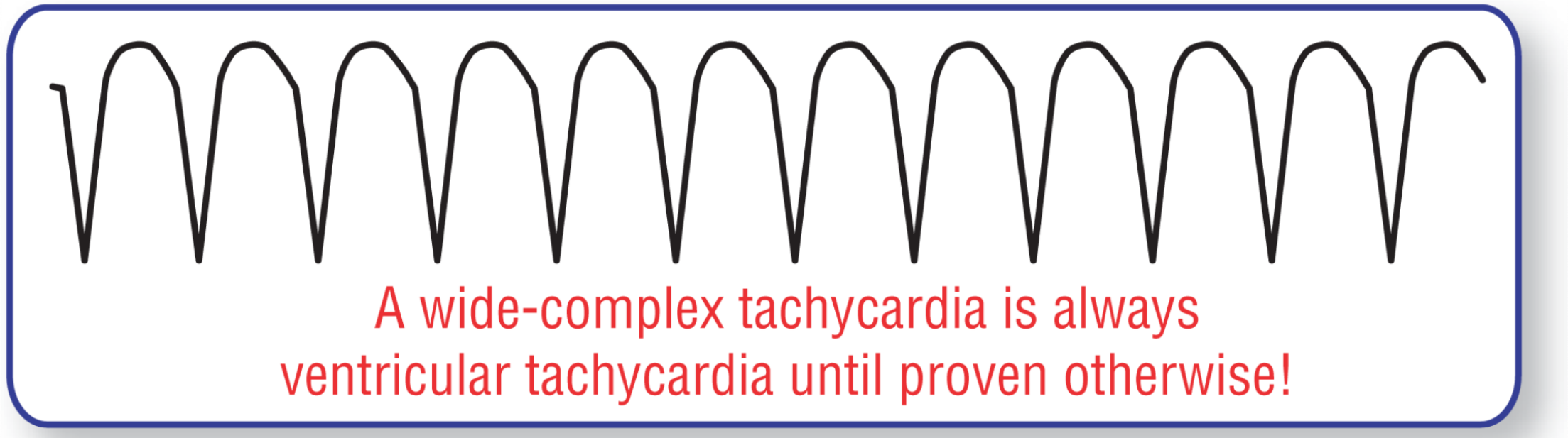
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia a complessi QRS larghi



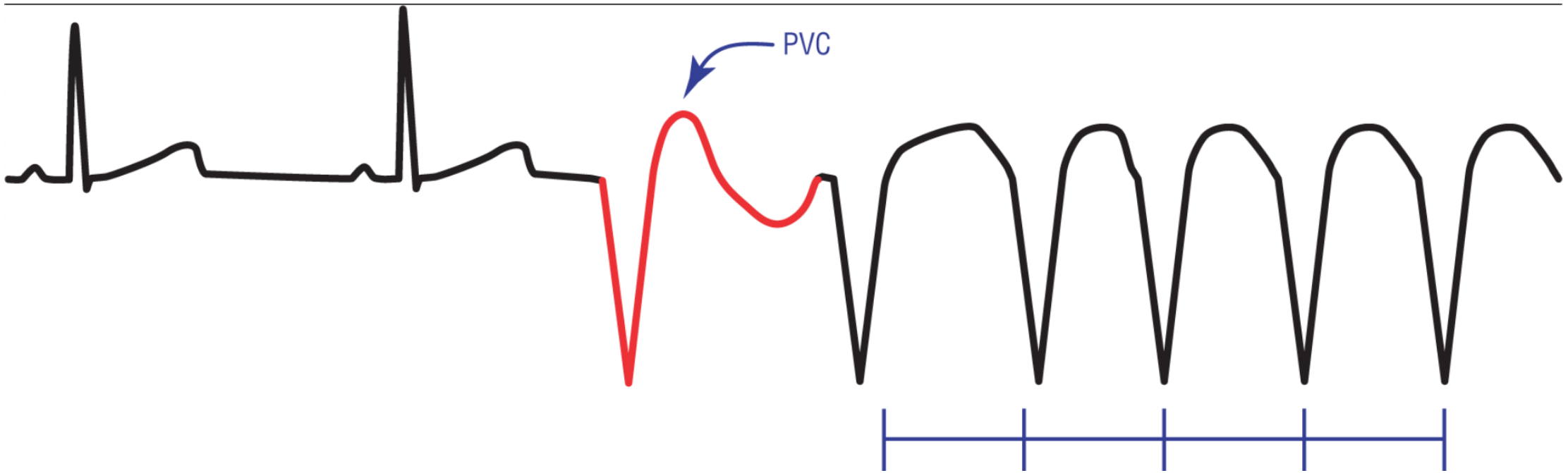
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia ventricolare



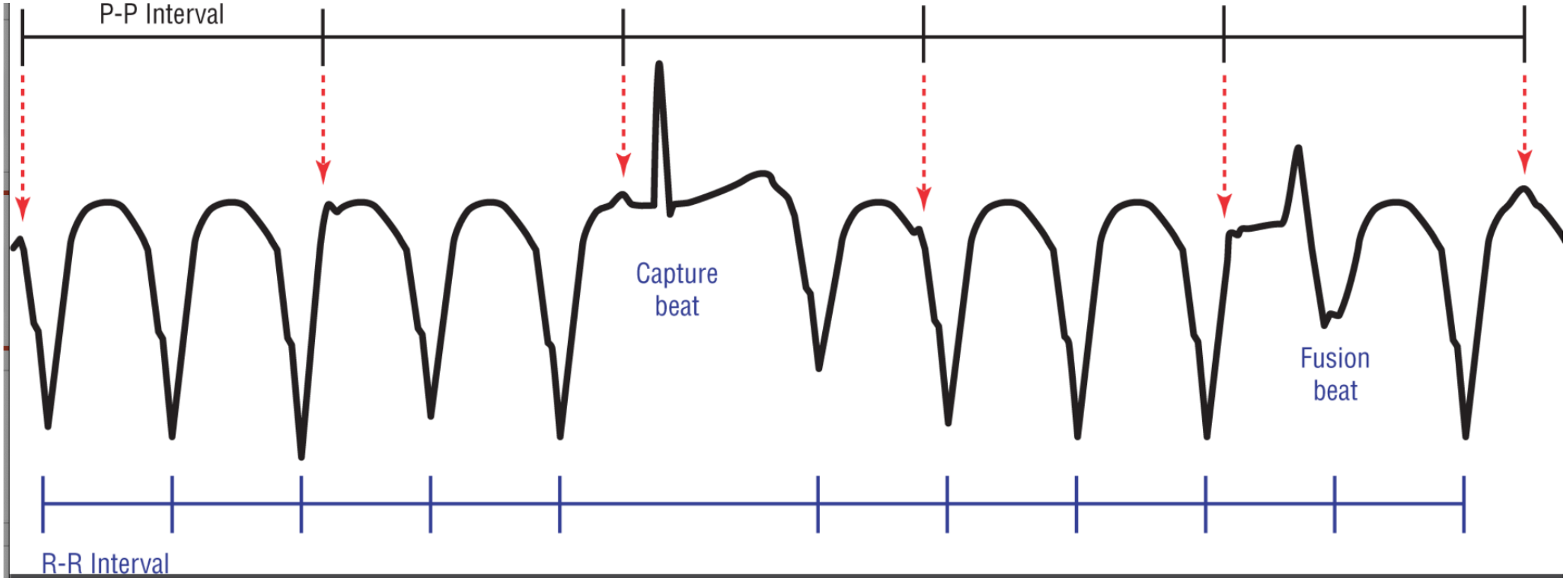
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia ventricolare



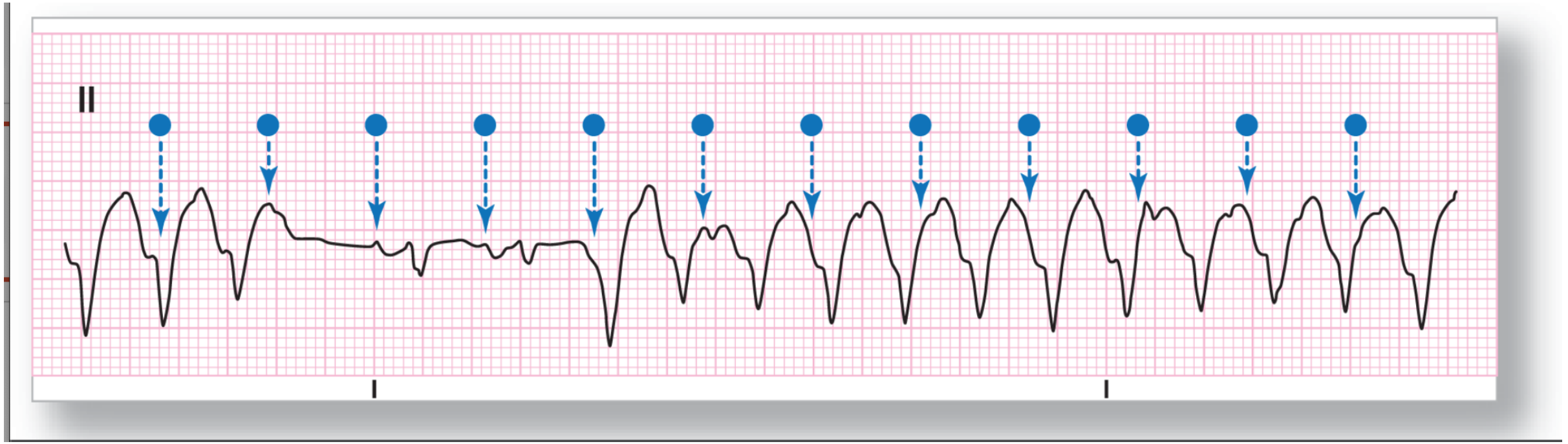
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia ventricolare



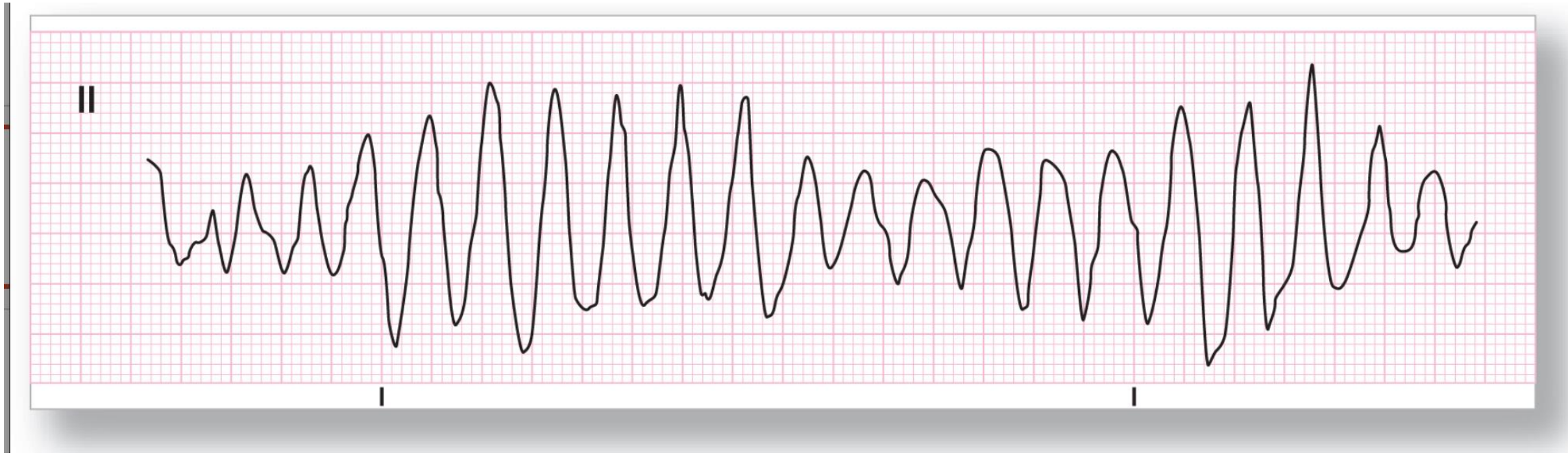
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia ventricolare



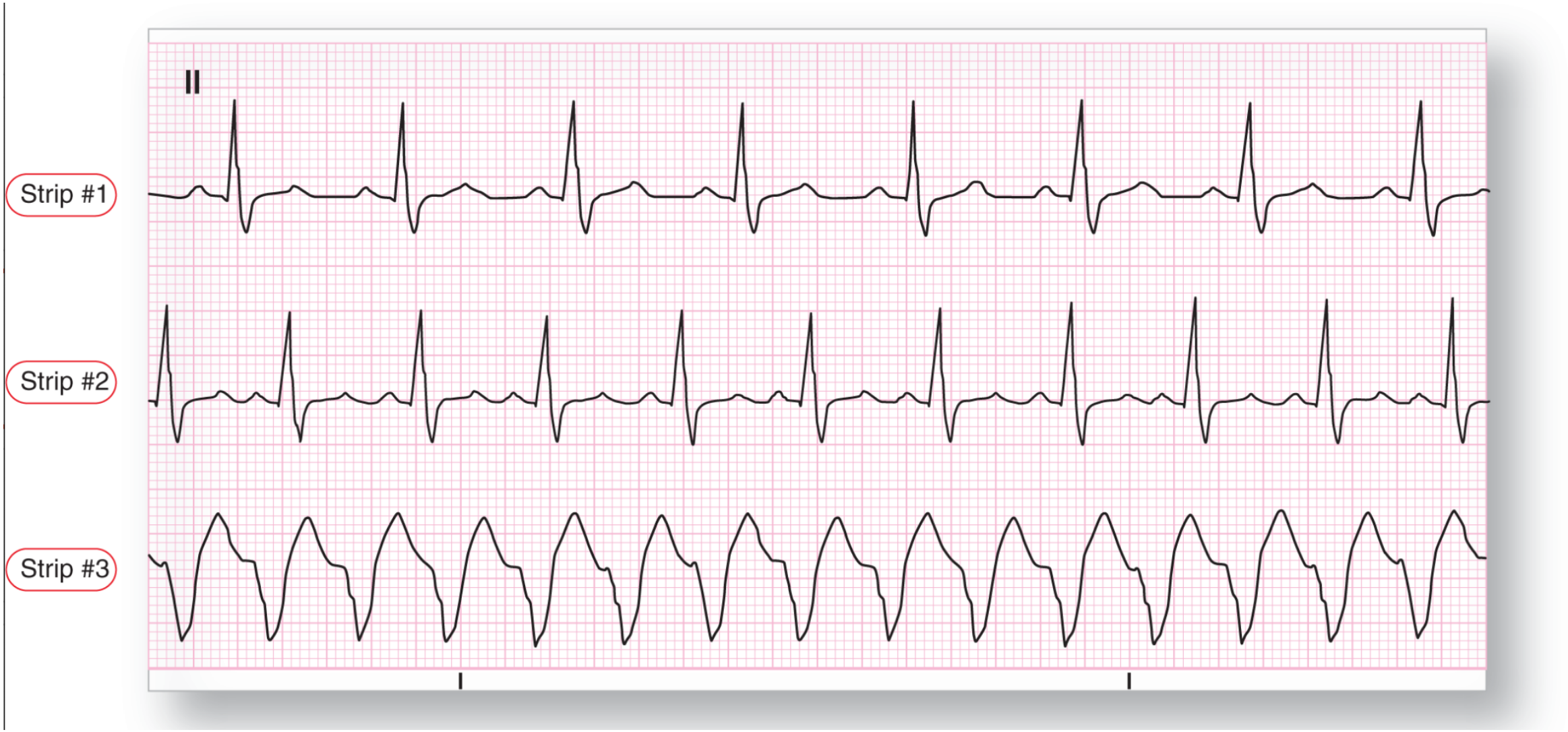
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Tachicardia ventricolare polimorfa- Torsade de pointes



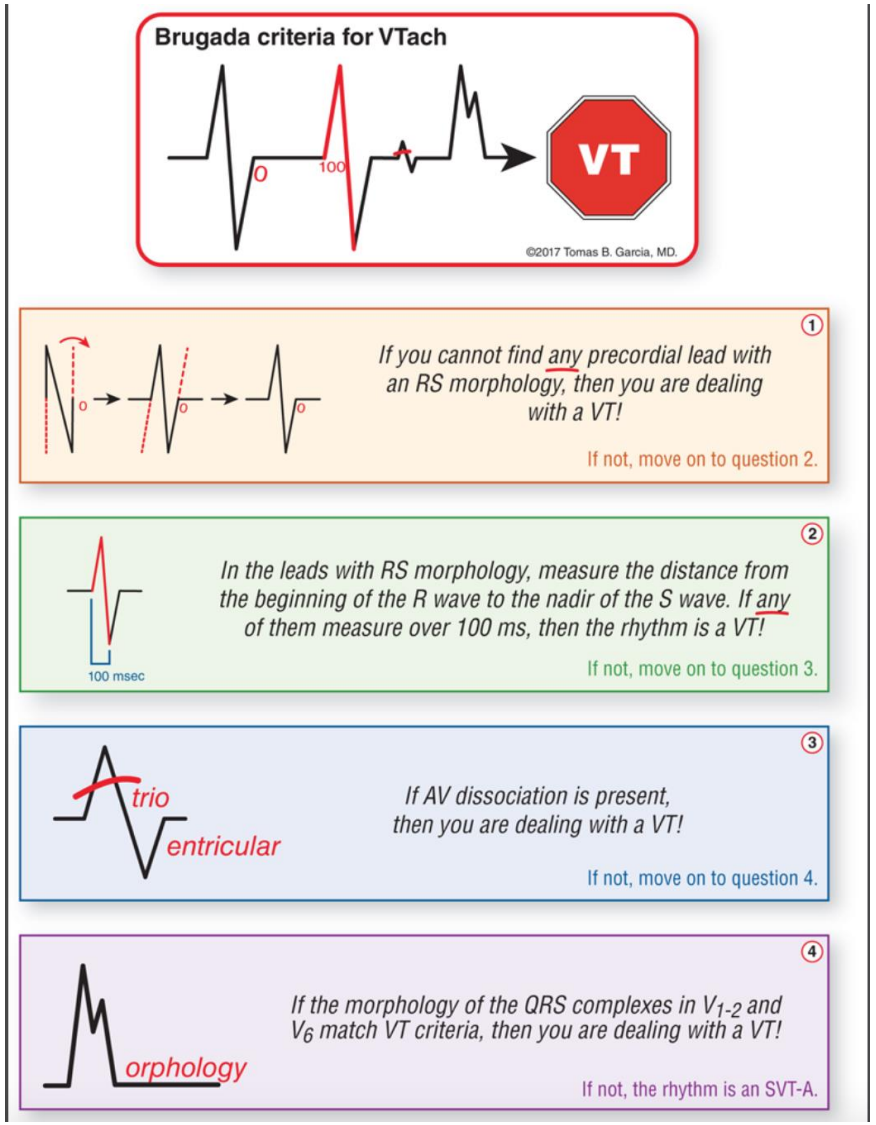
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

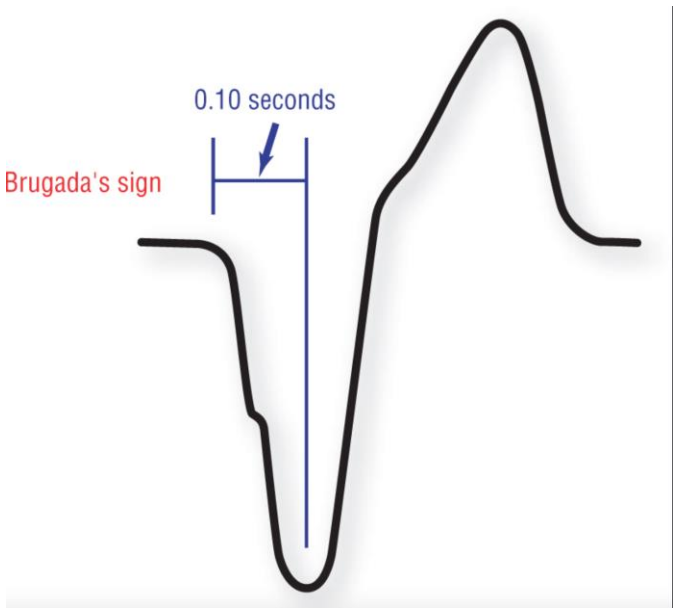
Tachicardia SV con aberranza – RBBB

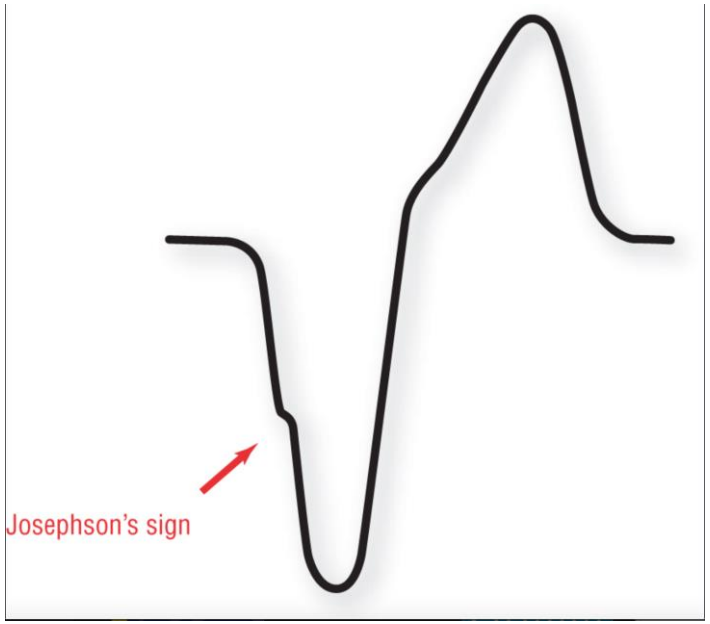


Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

TSV con aberranza vs TV

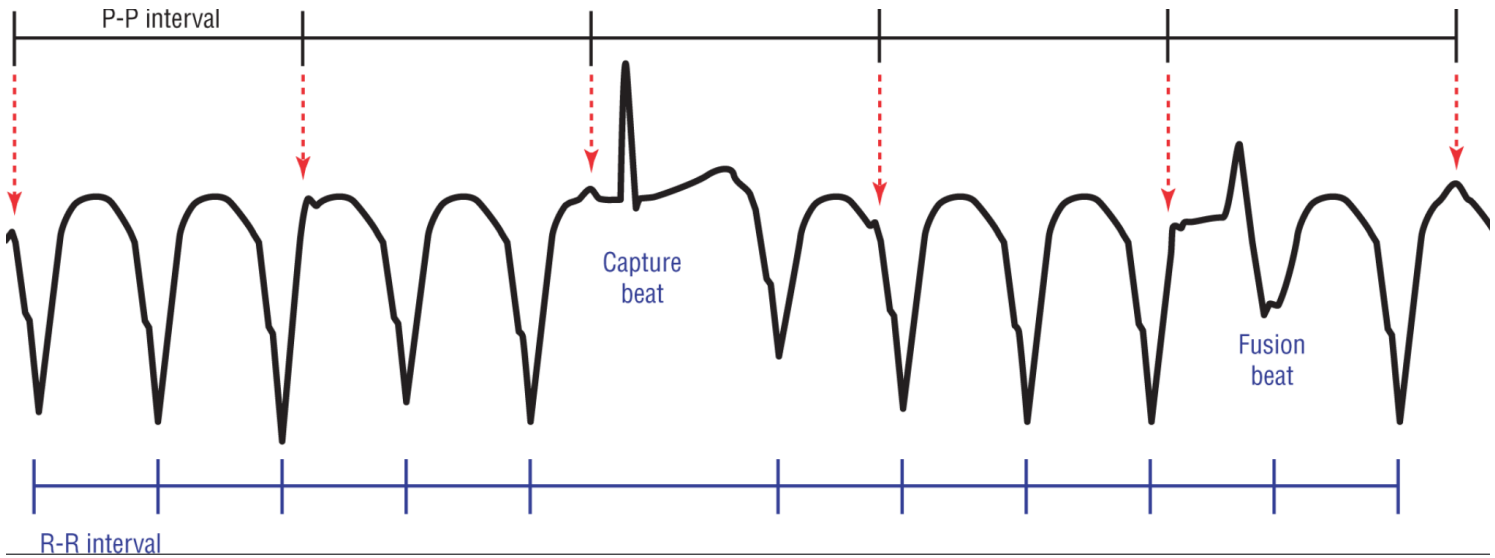






Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

TSV con aberranza vs TV



WCT Checklist

History: ☐ History of MI ☐ Structural or ischemic HD ☐ Prior hx of arrhythmia ☐ Age > 35 years ☐ Family hx of sudden cardiac death ☐ Cardiomyopathy ☐ Congestive heart failure

CV status: ☐ Hemodynamically unstable ☐ Hemodynamically stable

QRS width: _____ sec.

Rate: _____ BPM **Atrial rate:** _____ BPM **Ventricular rate:** _____ BPM

Regularity: ☐ Regular ☐ Regularly irregular ☐ Irregularly irregular

Morphology: RBBB-like V_1 ☐ rSR′ ☐ RSr′ ☐ qR ☐ Monomorphic R; RBBB-like V_6 ☐ R:S ratio < 1; LBBB-like V_1 ☐ Initial R ≥ 30 ms ☐ Notching on S ☐ R to nadir of S > 70 ms; LBBB-like V_6 ☐ Any Q wave in V_6; Morphology of associated PVCs: ☐ Same ☐ Different

AV dissociation: ☐ Present ☐ Absent ☐ Capture beats ☐ Fusion beats

Concordance of QRS complexes in the precordials: ☐ Present ☐ Absent

Axis: ☐ ↑I ↑aVF (normal) ☐ ↑I ↓aVF (left) ☐ ↓I ↑aVF (right) ☐ ↓I ↓aVF (extreme right)

Brugada Algorithm

- ☐ Look for RS complexes in all the precordial leads. If you can't find one, then you are dealing with a VTach!
- ☐ In the leads with RS morphology, measure the distance from the beginning of the R wave to the nadir of the S wave (the RS interval). If any of them measure over 100 ms, then the rhythm is a VTach.
- ☐ Look for evidence of AV dissociation. If there is AV dissociation, then it is a VTach.
- ☐ Look at the morphology of the rhythm in leads V_{1-2} and V_6. If the morphology matches the criteria outlined above, then it is a VTach.

↓ No

☐ **SVT with Aberrancy**

Vereckei aVR Algorithm

- ☐ Do the QRS complexes in aVR begin with a large R wave? If they do, it's VTach.
- ☐ Do the QRS complexes in aVR start off with smaller q or r waves that are over 40 ms wide? If they do, it's VTach.
- ☐ Is there a notch on the descending limb of a completely negative QS complex in lead aVR? If they do, it's VTach.
- ☐ If the V_t is bigger, you have a VTach. ($V_i/V_t \leq 1$)

↓ No

☐ **SVT with Aberrancy**

☐ Favors VTach ☐ Favors SVT-A

Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

TACHYCARDIA

EUROPEAN RESUSCITATION COUNCIL
GUIDELINES 2021

ASSESS with ABCDE approach
- Give oxygen if SpO_2 < 94% and obtain IV access
- Monitor ECG, BP, SpO_2. Record 12 lead ECG
- Identify and treat reversible causes (e.g. electrolyte abnormalities, hypovolaemia causing sinus tachycardia)

Life-threatening features?
1. Shock
2. Syncope
3. Myocardial ischaemia
4. Severe heart failure

YES

Synchronised shock up to 3 attempts
- Sedation, anaesthesia if conscious

If unsuccessful:
- Amiodarone 300 mg IV over 10-20 min, or procainamide 10-15 mg/kg IV over 20 min;
- Repeat synchronised shock

UNSTABLE

NO

STABLE
SEEK EXPERT HELP

Is QRS narrow (<0.12 s)?

Broad QRS
Is QRS regular?

Irregular

Possibilities include:
- Atrial fibrillation with bundle branch block – treat as for irregular narrow complex
- Polymorphic VT (e.g. torsades de pointes) – give magnesium 2 g over 10 min

Regular

If VT (or uncertain rhythm):
- Procainamide 10-15 mg/kg IV over 20 min
  or
- Amiodarine 300 mg IV over 10-60 min

If previous certain diagnosis of SVT with bundle branch block/ aberrant conduction:
- Treat as for regular narrow complex tachycardia

Narrow QRS
Is QRS regular?

Regular

Vagal manoeuvres

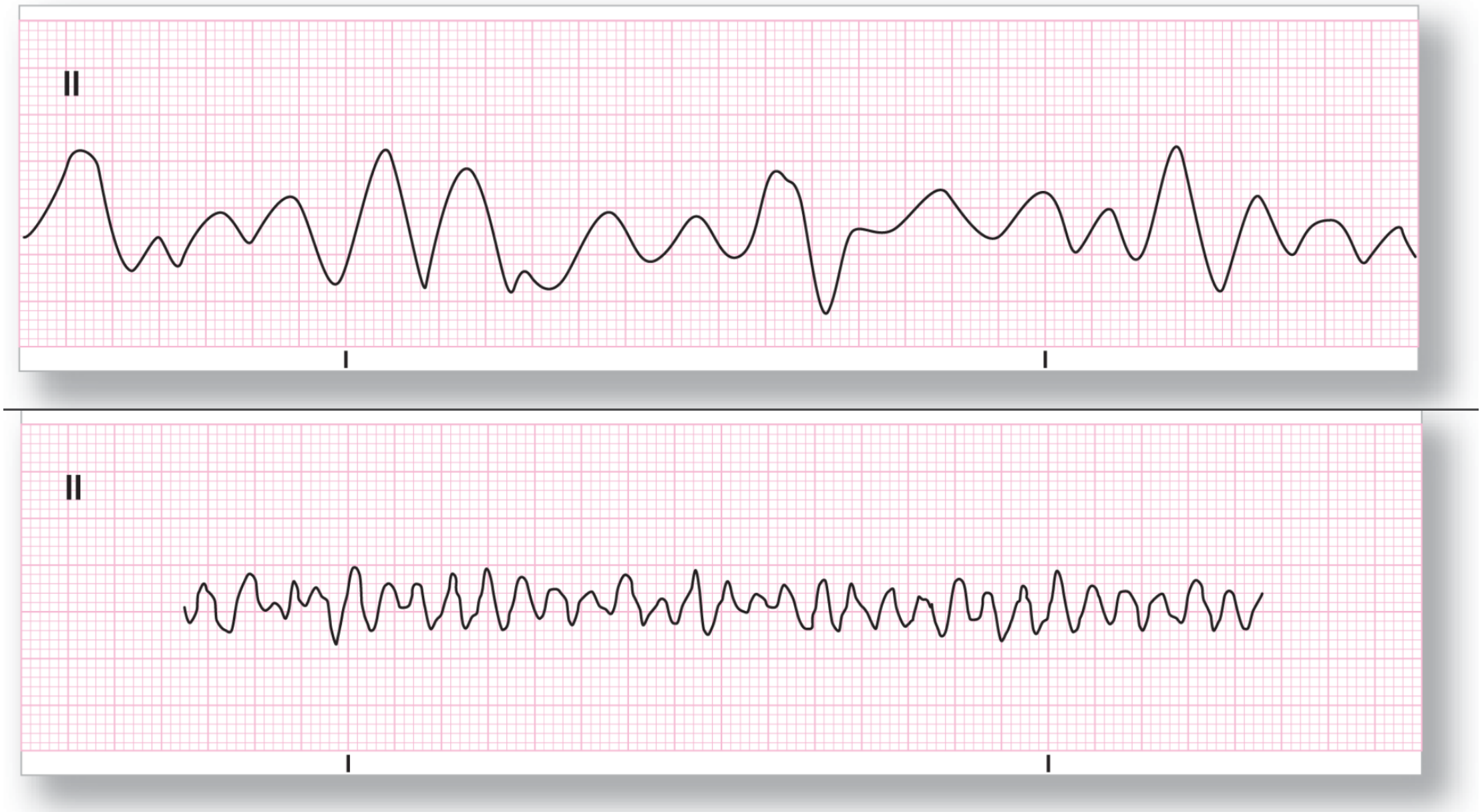
If ineffective:
Adenosine (if no pre-excitation)
- 6 mg rapid IV bolus;
- If unsuccessful give 12 mg
- If unsuccessful give IV 18 mg

If ineffective:
- Verapamil or beta-blocker

Irregular

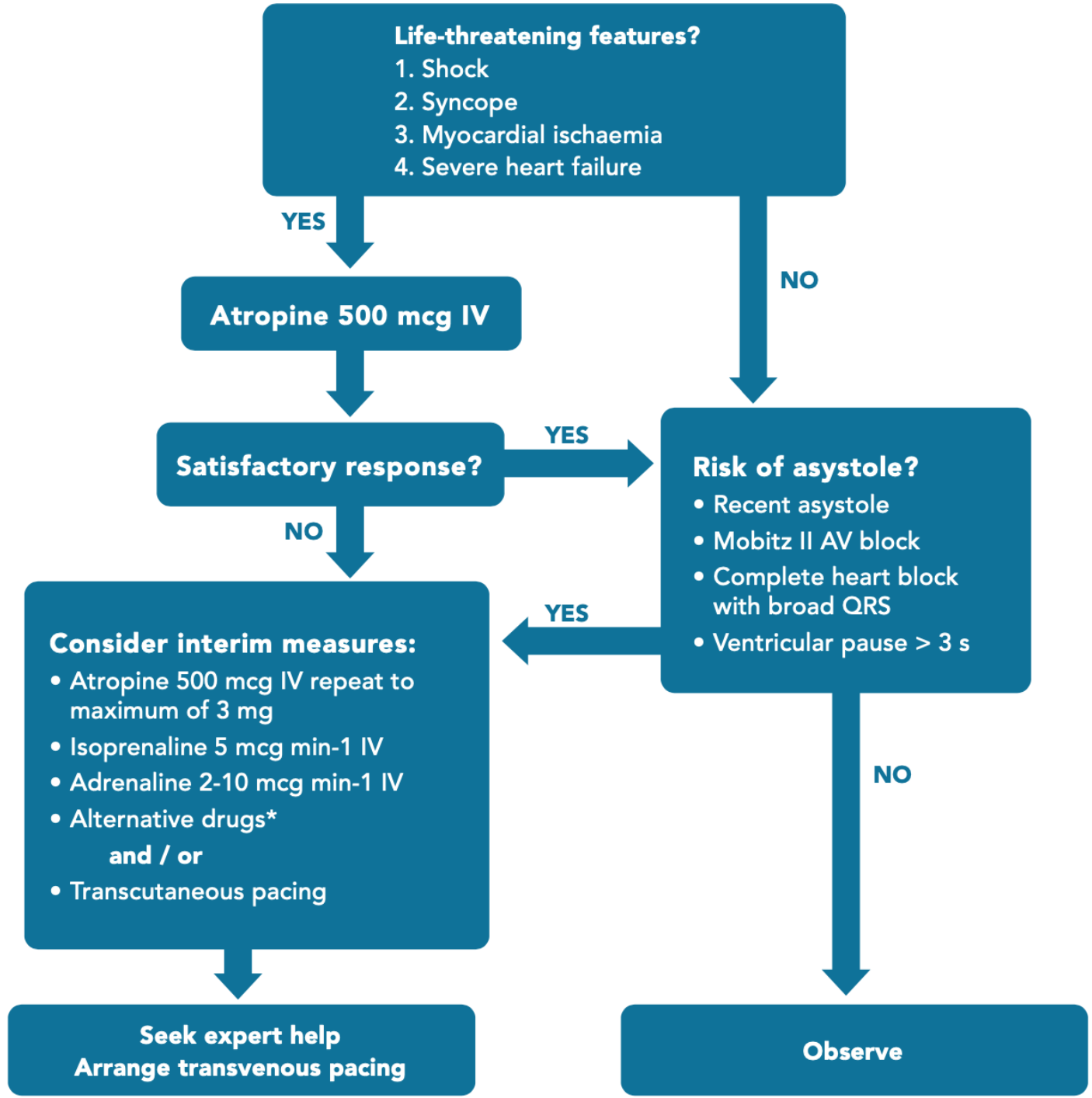
Probable atrial fibrillation:
- Control rate with beta-blocker or diltiazem
- Consider digoxin or amiodarone if evidence of heart failure
- Anticoagulate if duration > 48h

If ineffective:
- Synchronised DC shock up to 3 attempts
- Sedation, anaesthesia if conscious

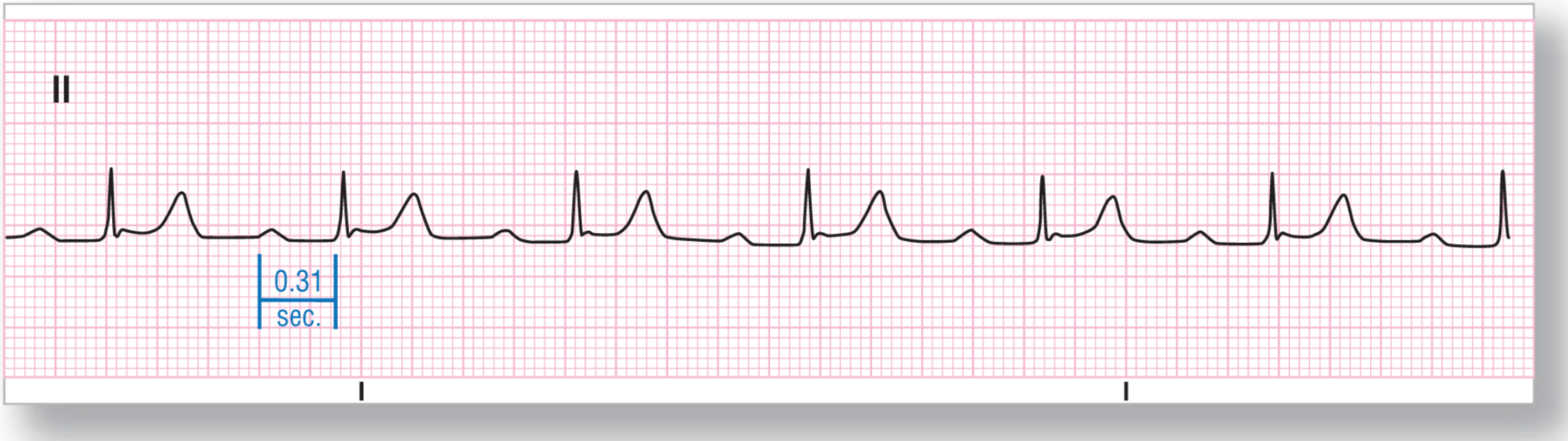


Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Domande o interventi?

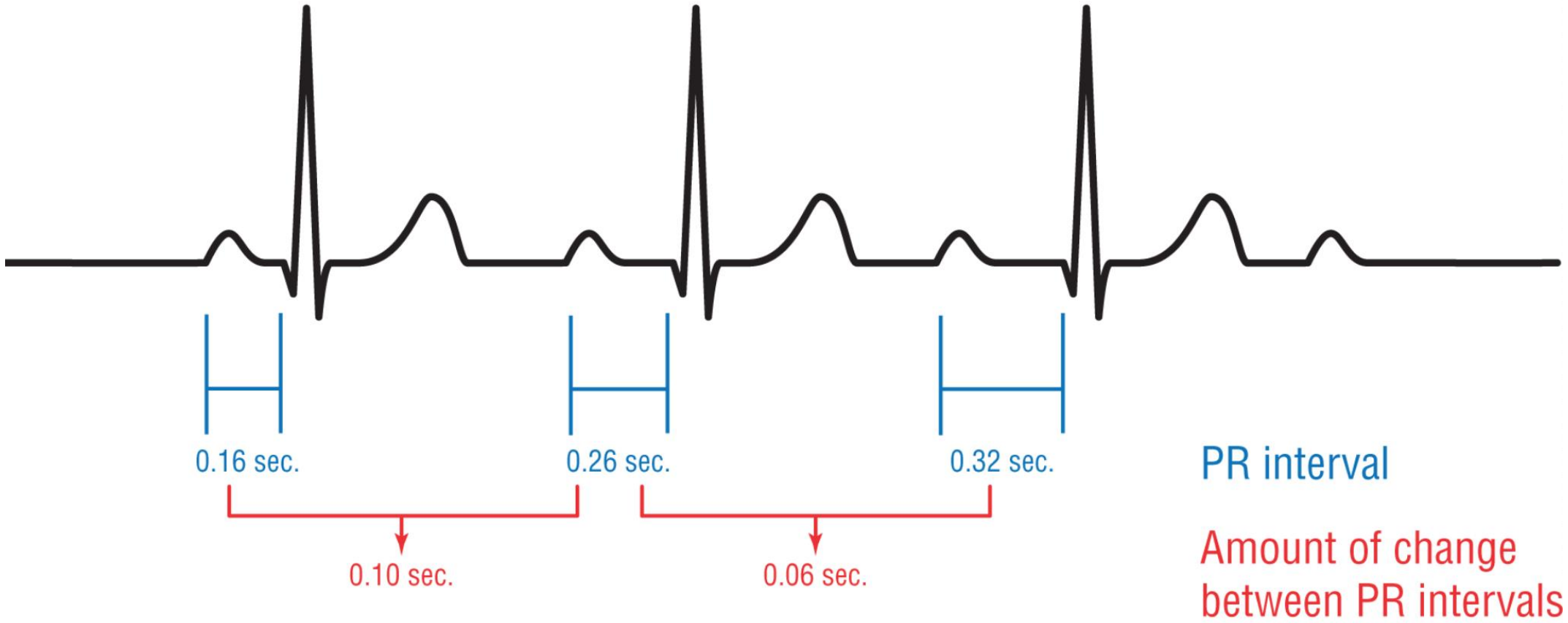

Life-threatening features?
1. Shock
2. Syncope
3. Myocardial ischaemia
4. Severe heart failure
YES
NO
Atropine 500 mcg IV
Satisfactory response?
YES
NO
Risk of asystole?
• Recent asystole
• Mobitz II AV block
• Complete heart block with broad QRS
• Ventricular pause > 3 s
YES
NO
Consider interim measures:
• Atropine 500 mcg IV repeat to maximum of 3 mg
• Isoprenaline 5 mcg min-1 IV
• Adrenaline 2-10 mcg min-1 IV
• Alternative drugs*
and / or
• Transcutaneous pacing
Seek expert help
Arrange transvenous pacing
Observe

Blocco atrioventricolare I



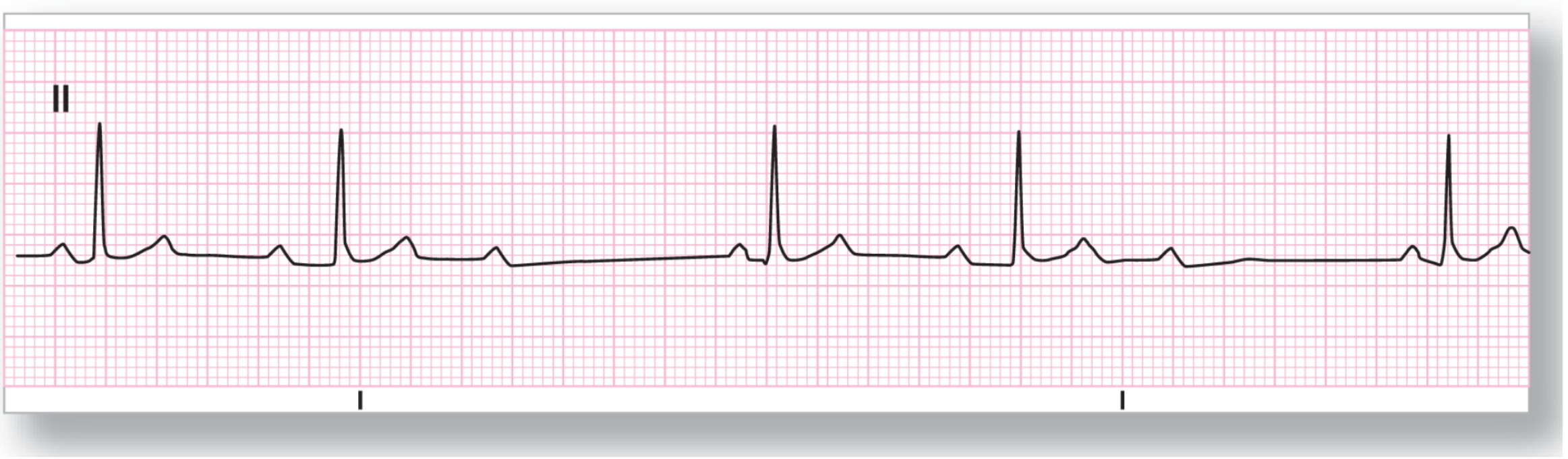
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Blocco atrioventricolare II – Mobitz I



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Blocco atrioventricolare II – Mobitz I



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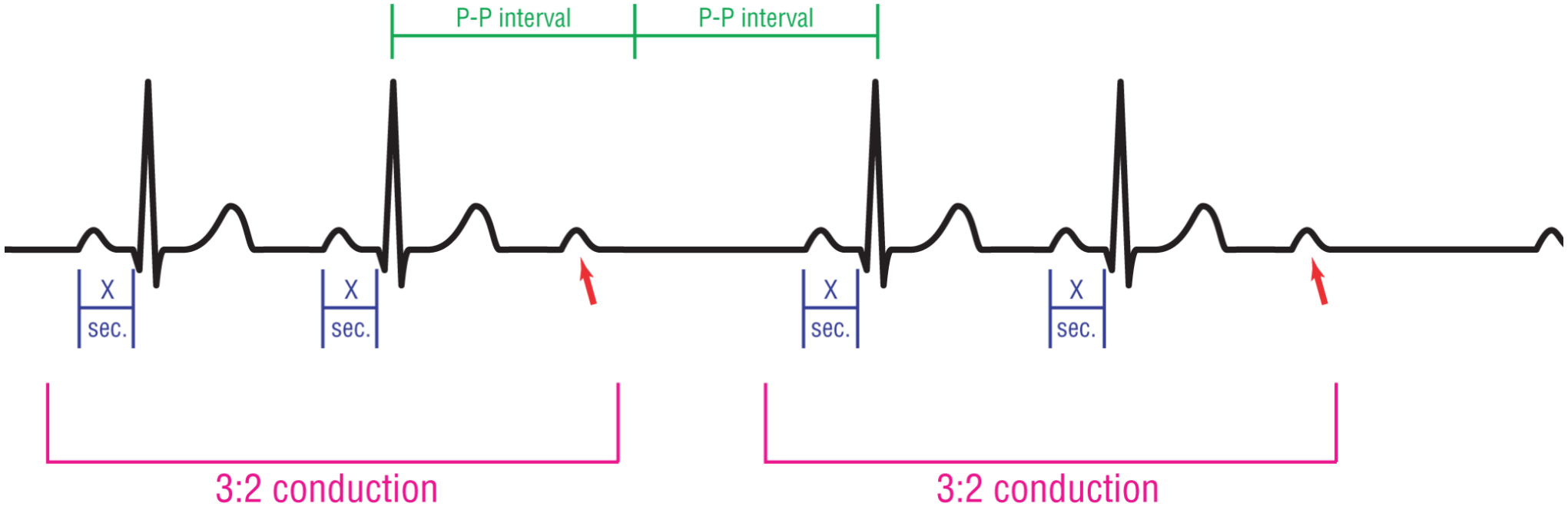
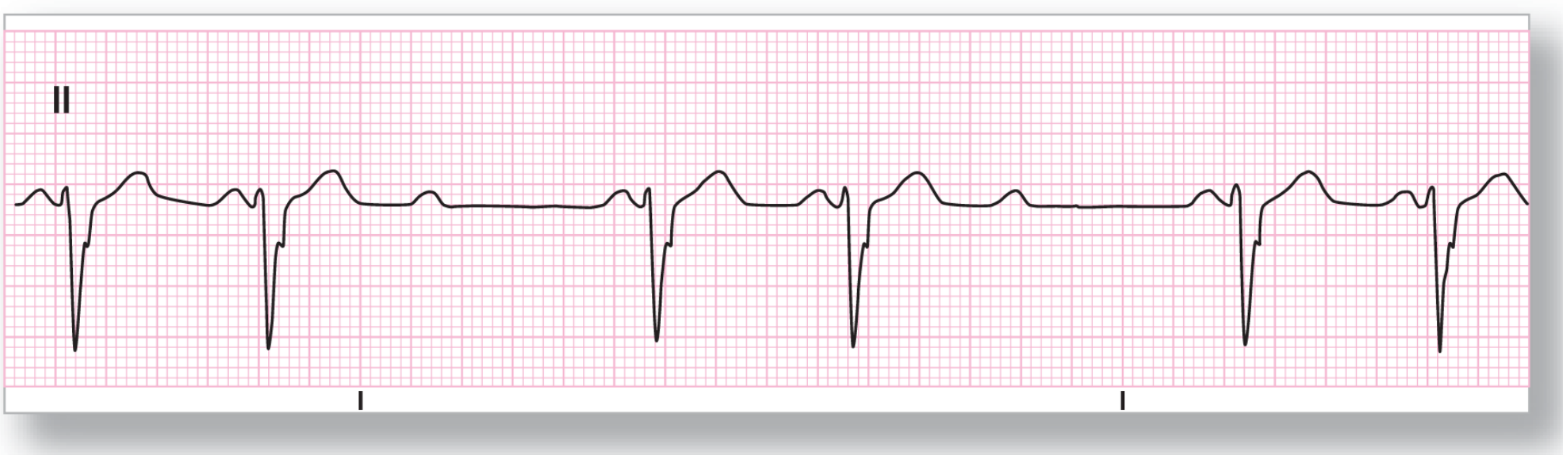
Blocco atrioventricolare II – Mobitz II



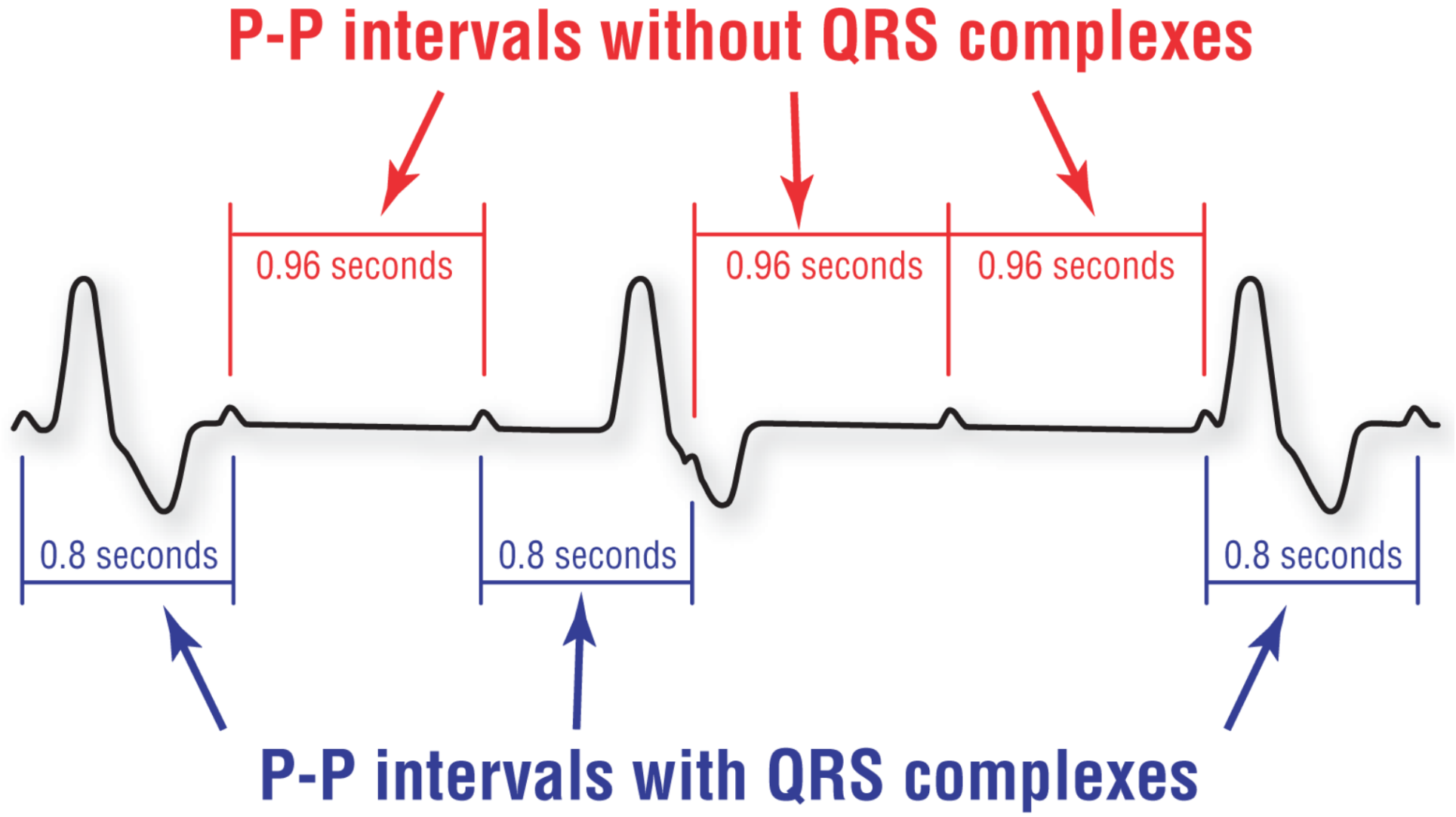
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Blocco atrioventricolare II – Mobitz II



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Blocco atrioventricolare III



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Domande o interventi?

Shock cardiogeno

Box 1 | **Causes of cardiogenic shock**

Acute myocardial infarction

Left ventricular failure

- Large infarction
- Small/moderate infarction with
  - pre-existing dysfunction
  - extensive ischaemia
- Global ischaemia

Right ventricular failure

- Large infarction
- Small/moderate infarction with
  - pre-existing dysfunction
  - pulmonary hypertension

Mechanical complications

- Acute mitral regurgitation with
  - rupture of a papillary muscle
  - rupture of chordae tendinae
  - severe papillary muscle dysfunction
- Ventricular septal defect caused by rupture of the interventricular septum
- Left ventricular free wall rupture
- Pericardial tamponade owing to rupture of the left ventricular free wall or haemorrhagic pericardial effusion

Concomitant conditions causing mixed aetiology

- Haemorrhage
- Infection
- Excess negative inotropic or vasodilator medications
- Sustained bradyarrhythmia or tachyarrhythmia
- Hyperglycaemia or ketoacidosis

Other conditions

End-stage cardiomyopathy

Myocarditis

Septic shock with severe myocardial depression

Left ventricular outflow tract obstruction

- Aortic stenosis
- Hypertrophic obstructive cardiomyopathy

Obstruction to left ventricular filling

- Mitral stenosis
- Left atrial myxoma

Acute mitral regurgitation (chordal rupture)

Acute aortic insufficiency

Myocardial contusion

Postcardiotomy shock

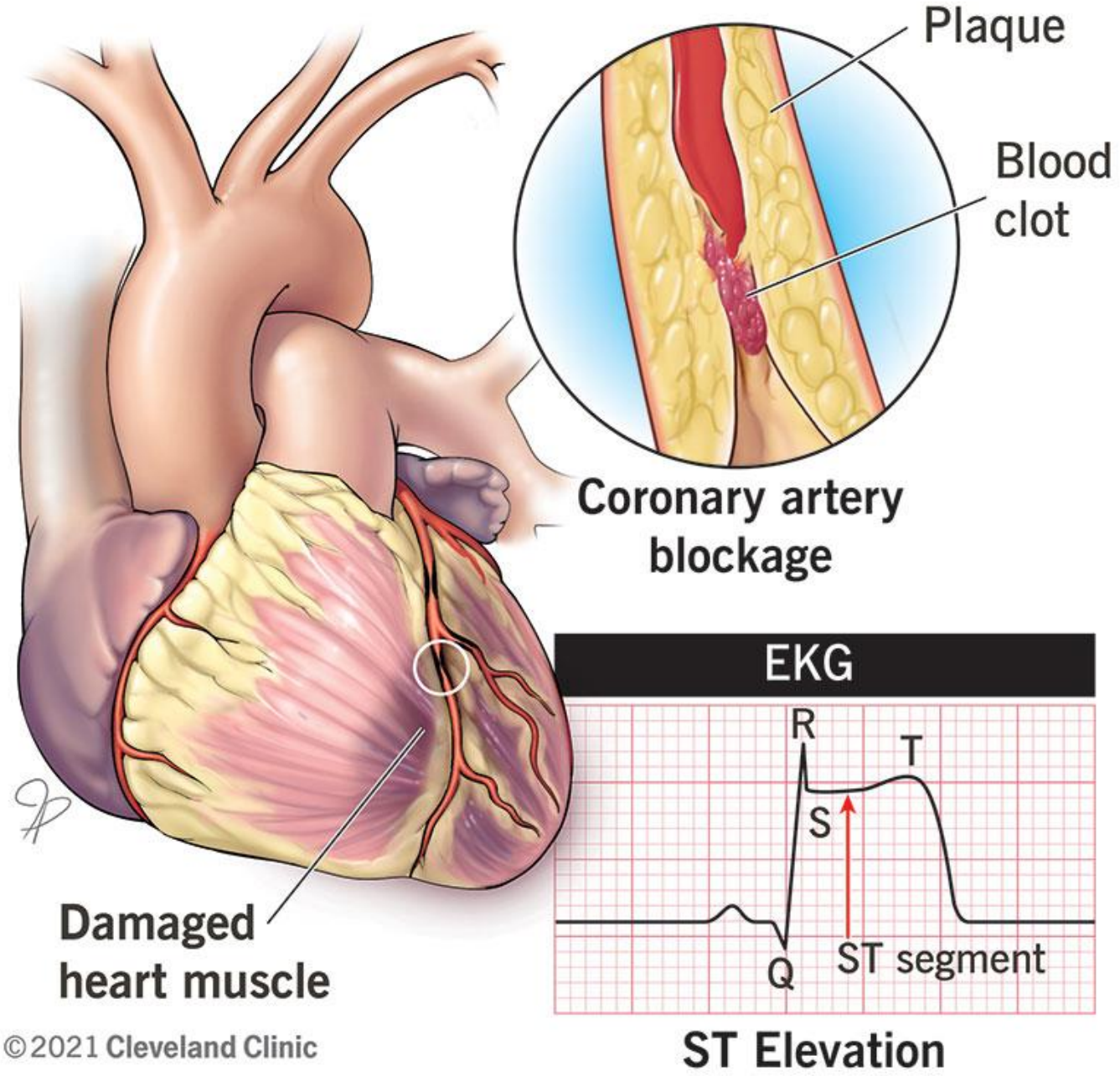
Global ischaemia

Stress-induced cardiomyopathy

Cardiac tamponade

Reyentovich A, Barghash MH, Hochman JS. Management of refractory cardiogenic shock. Nat Rev Cardiol. 2016;13(8):481-492. doi:10.1038/nrcardio.2016.96

Infarto del miocardio e shock



https://my.clevelandclinic.org/health/diseases/22068-stemi-heart-attack

Infarto del miocardio e shock

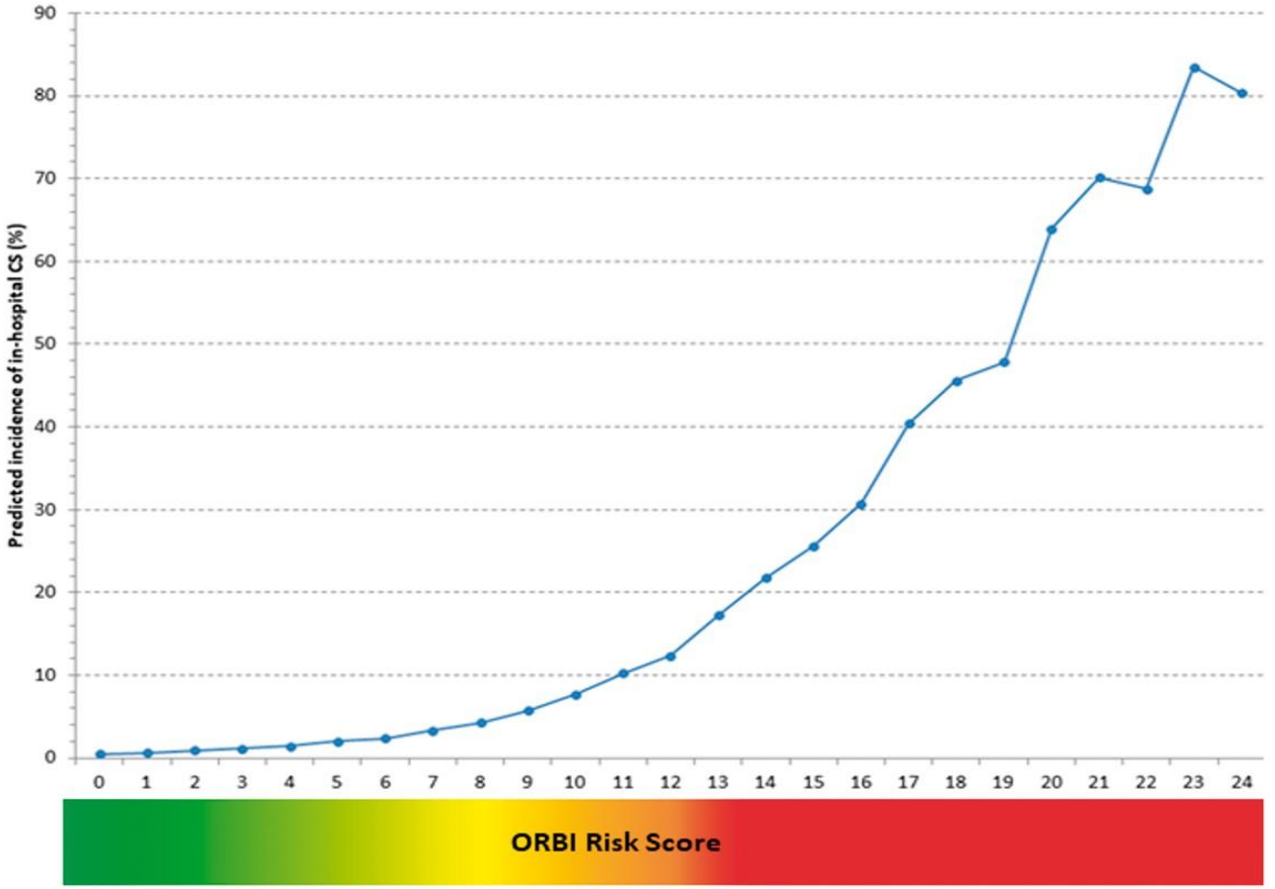
Table 1
Killip classification

Class A	No heart failure. No clinical signs of cardiac decompensation.
Class B	Heart failure. Diagnostic criteria include rales, S_3 gallop, and venous hypertension.
Class C	Severe heart failure. Frank pulmonary edema.
Class D	CS. Signs include hypotension (systolic pressure of 90 mm Hg or less) and evidence of peripheral vasoconstriction, such as oliguria, cyanosis, and diaphoresis. Heart failure, often with pulmonary edema, has also been present in most of these patients.

Data from Killip T 3rd, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol 1967;20(4):457–64.

Shah P, Cowger JA. Cardiogenic shock. Crit Care Clin. 2014;30(3):391-412. doi:10.1016/j.ccc.2014.03.001

Variable	Points
Age > 70 years old	2
Previous stroke / TIA	2
Presentation as cardiac arrest	3
Anterior myocardial infarction	1
First medical contact-to-pPCI delay > 90 min	2
Killip class II on admission	2
Killip class III on admission	6
Heart rate > 90/min on admission	3
SBP < 125 mmHg and PP < 45 mmHg on admission	4
Glycaemia > 10 mmol/l on admission	3
Culprit lesion of the left main	5
Post-pPCI TIMI flow < 3	5



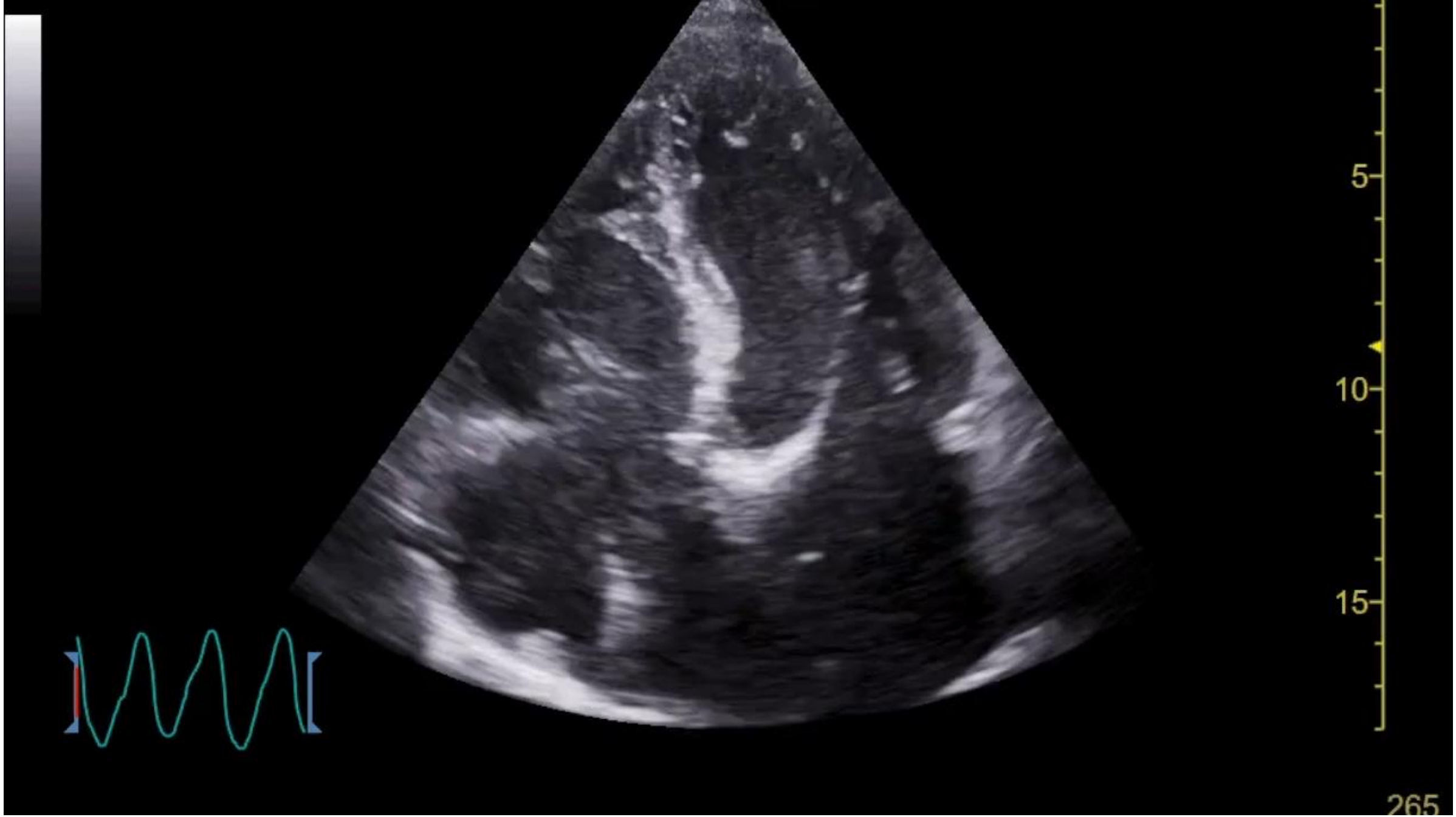
Risk categories

Category	Score	Observed incidence of CS
Low	0-7	1.3
Low-to-intermediate	8-10	6.6
Intermediate-to-high	11-12	11.7
High	≥ 13	31.8

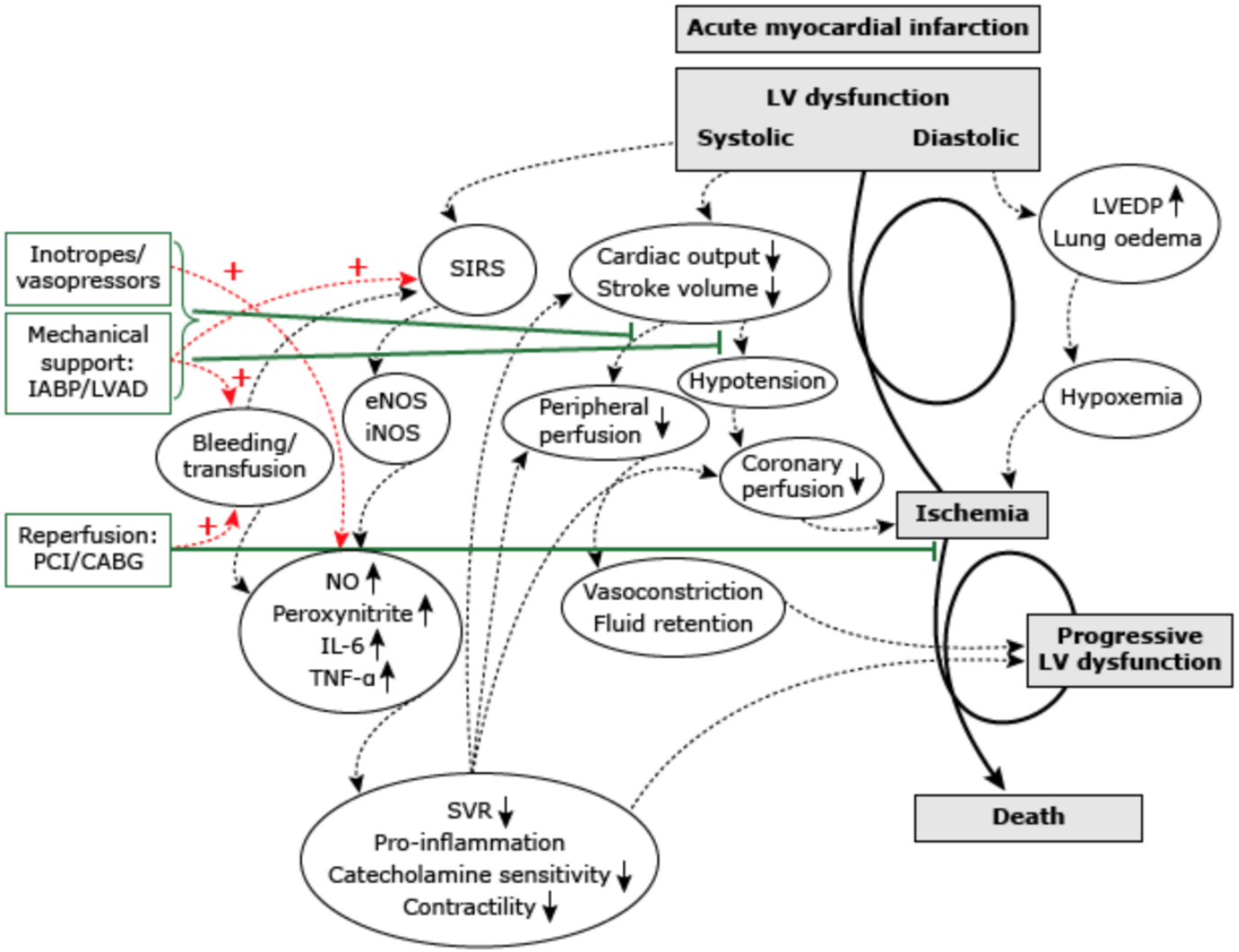
Auffret V, Cottin Y, Leurent G, et al. Predicting the development of in-hospital cardiogenic shock in patients with ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention: the ORBI risk score. *Eur Heart J*. 2018;39(22):2090-2102. doi:10.1093/eurheartj/ehy127

https://www.youtube.com/watch?v=gGfmtNNrmq0

https://www.youtube.com/watch?v=cfFwtYgFrvU


5
10
15
265

Fisiopatologia



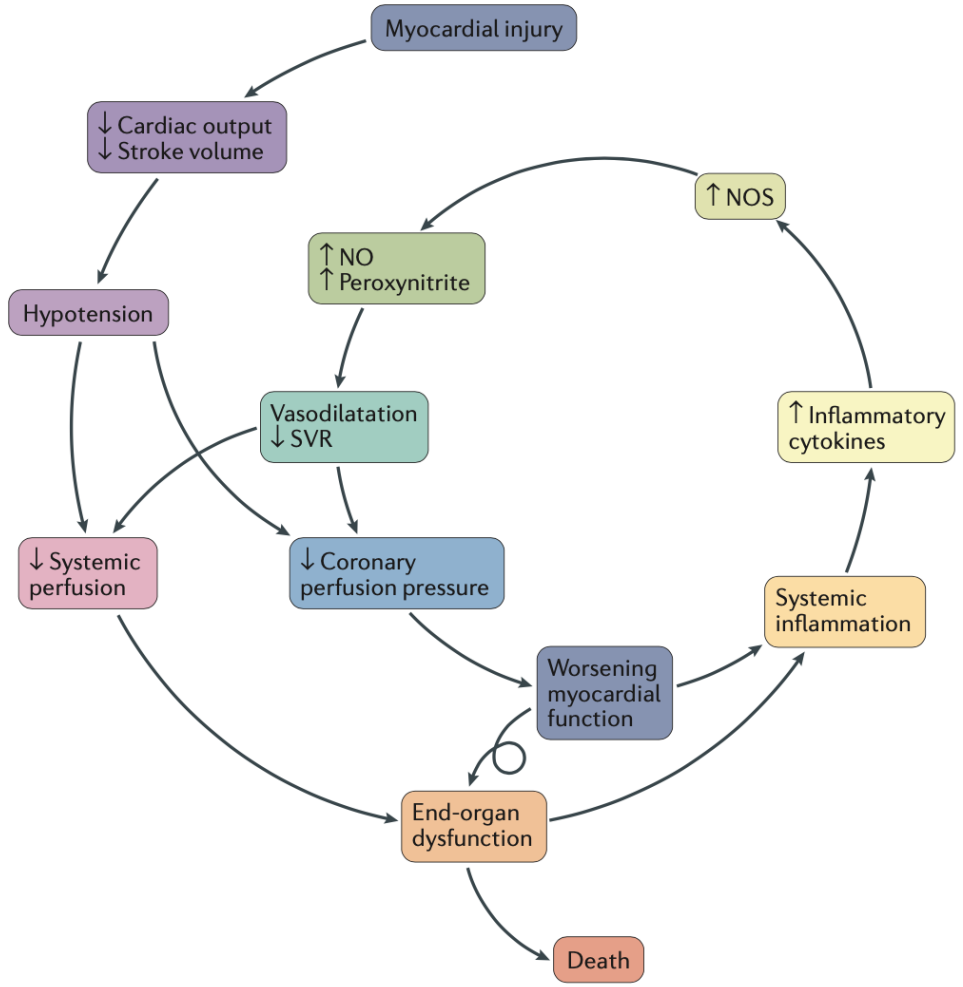


Figure 1 | **The downward spiral of refractory cardiogenic shock.** The downward spiral of refractory cardiogenic shock results from severe cardiac dysfunction, most often due to myocardial injury, leading to ongoing systemic and coronary hypoperfusion. A commonly observed systemic inflammatory response might lead to vasodilatation and further contribute to ongoing cardiac dysfunction and end-organ insult. Cardiogenic shock will inevitably lead to death if the cycle of damage is not interrupted. NO, nitric oxide; NOS, nitric oxide synthase; SVR, systemic vascular resistance.

Thiele H, Allam B, Chatellier G, Schuler G, Lafont A. Shock in acute myocardial infarction: the Cape Horn for trials? Eur Heart J. 2010;31(15):1828-1835. doi:10.1093/eurheartj/ehq220

Reyentovich A, Barghash MH, Hochman JS. Management of refractory cardiogenic shock. Nat Rev Cardiol. 2016;13(8):481-492. doi:10.1038/nrcardio.2016.96

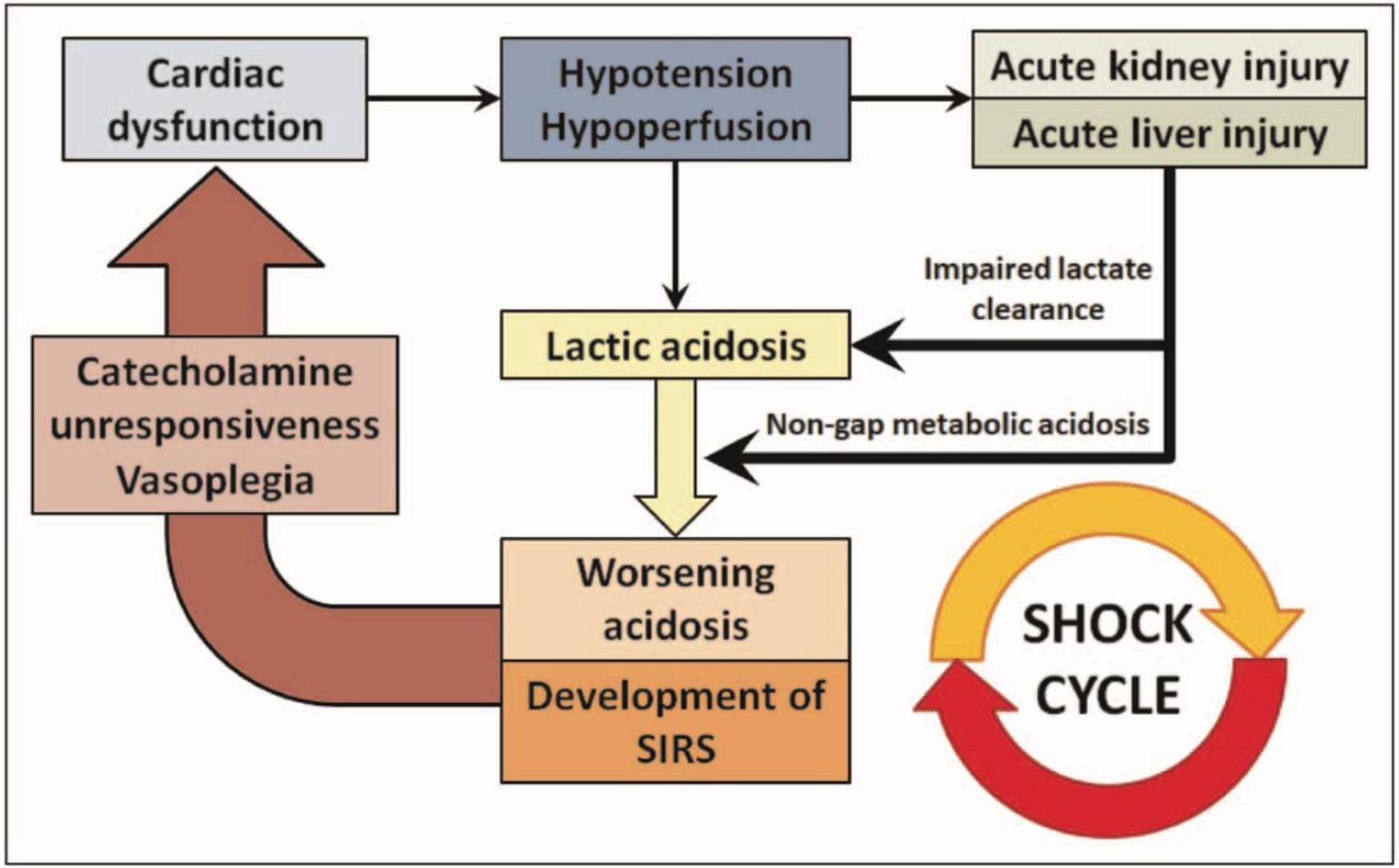
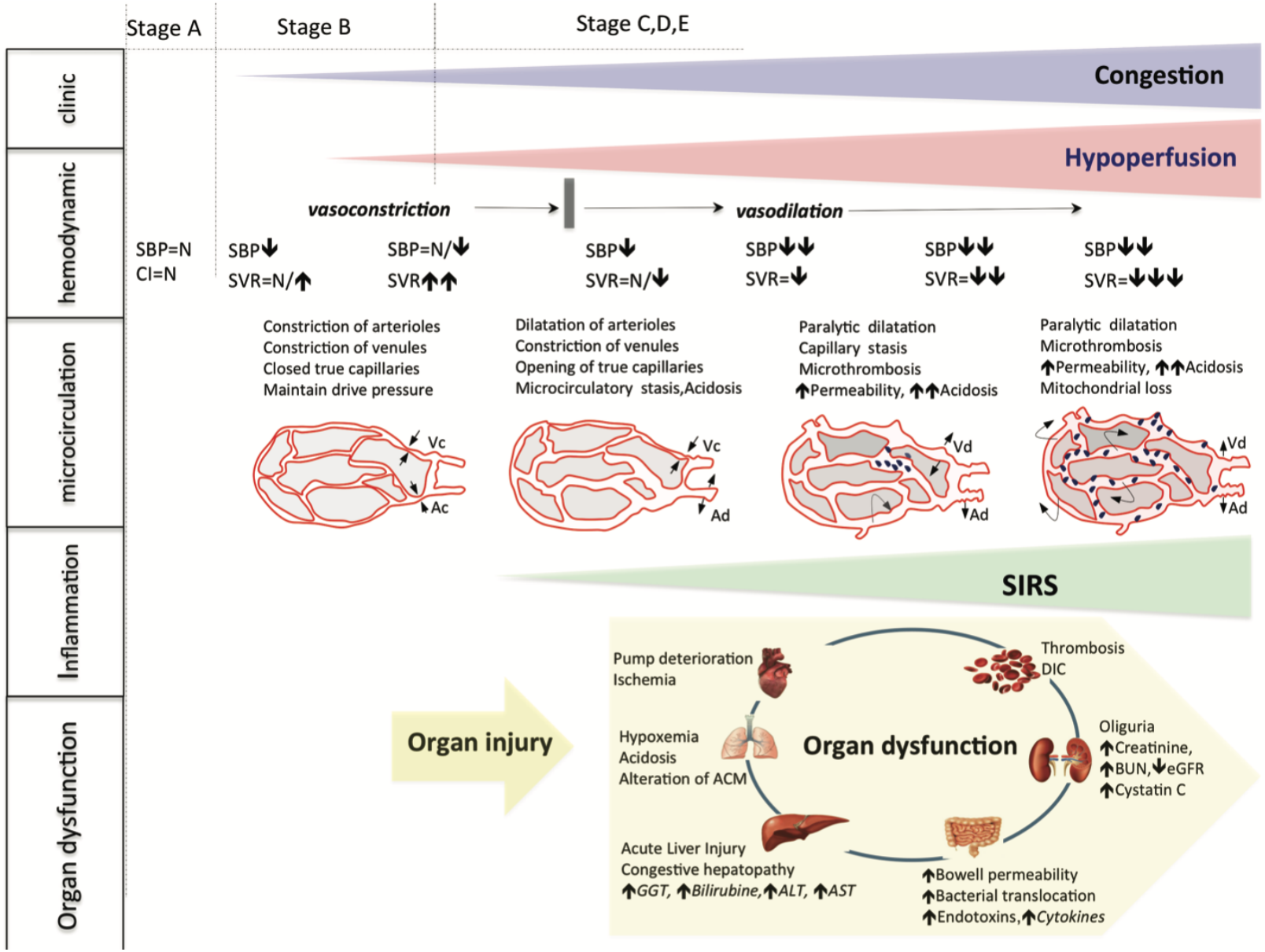


FIGURE 1. The progression from hemodynamic shock to hemometabolic shock. Cardiogenic shock produces tissue and organ hypoperfusion, resulting in lactic acidosis, acute kidney injury and acute liver injury. Acute liver and kidney injury prevent clearance of lactic acid, triggering a cycle of worsening metabolic acidosis. Metabolic derangements including severe acidosis and development of systemic inflammatory response syndrome lead to cardiovascular hyporesponsiveness to catecholamines and vasoplegia, worsening the initial shock state and triggering a downward spiral (the shock cycle).

Jentzer JC, Tabi M, Burstein B. Managing the first 120 min of cardiogenic shock: from resuscitation to diagnosis. Curr Opin Crit Care. 2021;27(4):416-425. doi:10.1097/MCC.0000000000000839

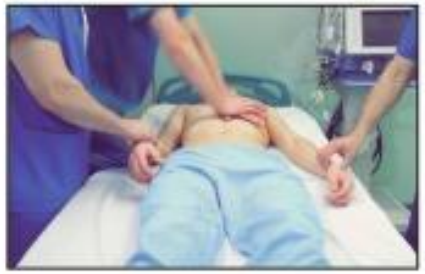
Progressione



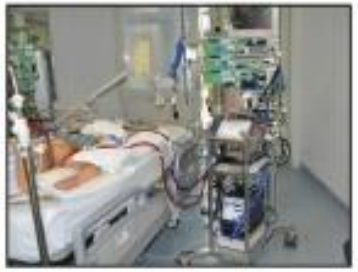
Chioncel O, Parissis J, Mebazaa A, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock - a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2020;22(8):1315-1341. doi:10.1002/ejhf.1922

Clinica

Presentations of Cardiogenic Shock



Catastrophic Shock



Acute Severe Shock

Indolent Progressive Shock

	Catastrophic Shock	Acute Severe Shock	Indolent Progressive Shock
Typical Scenario	Ventricular Fibrillation PEA arrest	Acute Myocardial Infarction Fulminant Myocarditis	Progressive Congestive HF Moving from Stage C to Stage D.
Physiology	Complete cessation of cardiac function.	Severe reduction in CO and BP.	Moderate reduction in CO and BP.
Time Course to Effectively Intervene	<5 minutes to provide adequate CPR or restore cardiac function. <1 hour to restore CO, BP.	Restore CO, BP within 1-6 hours.	Hours to days to improve hemodynamics.
Goals of Therapy	Anoxic brain injury must be avoided.	Avoid SIRS, MOF.	Improve CO, BP and lower filling pressures. Evaluate for TX, LVAD or hospice.

Kirklin JK, Rogers JG. Mechanical Circulatory Support: A Companion to Braunwald's Heart Disease Ebook. Elsevier Health Sciences; 2019.

Segni clinici

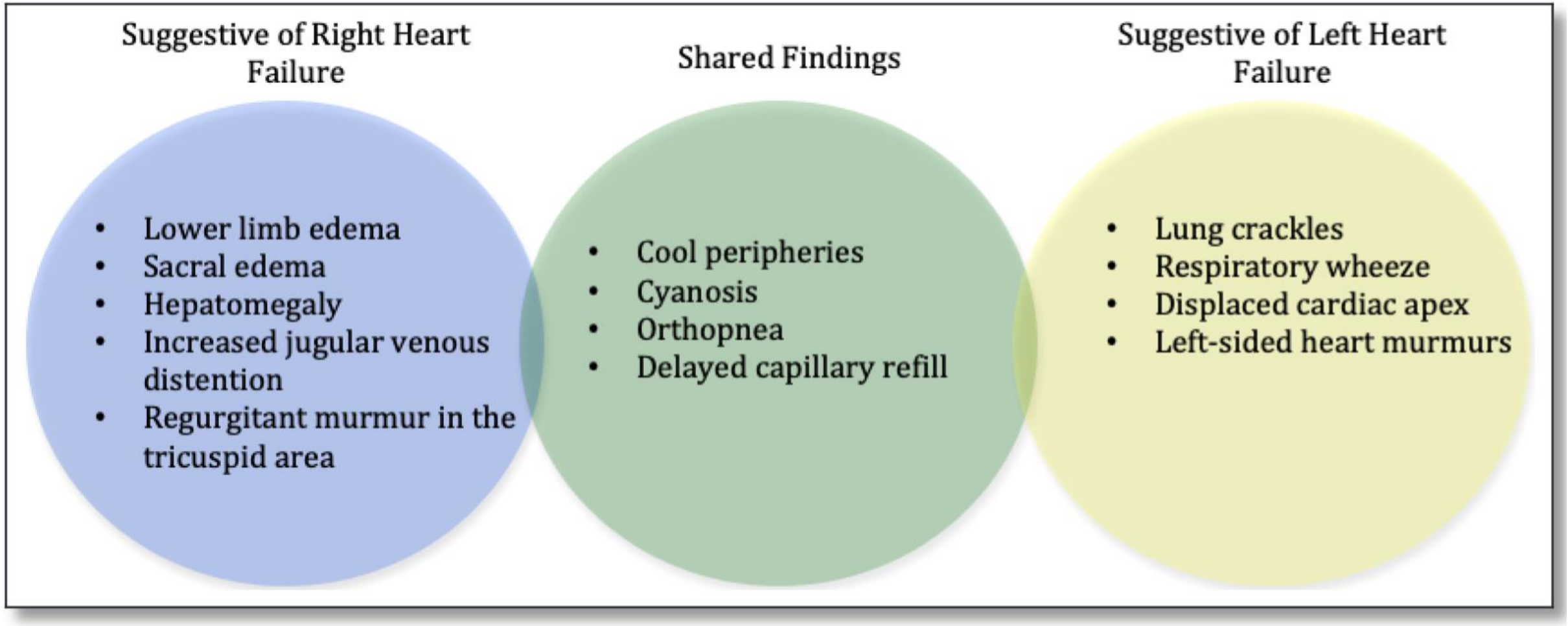
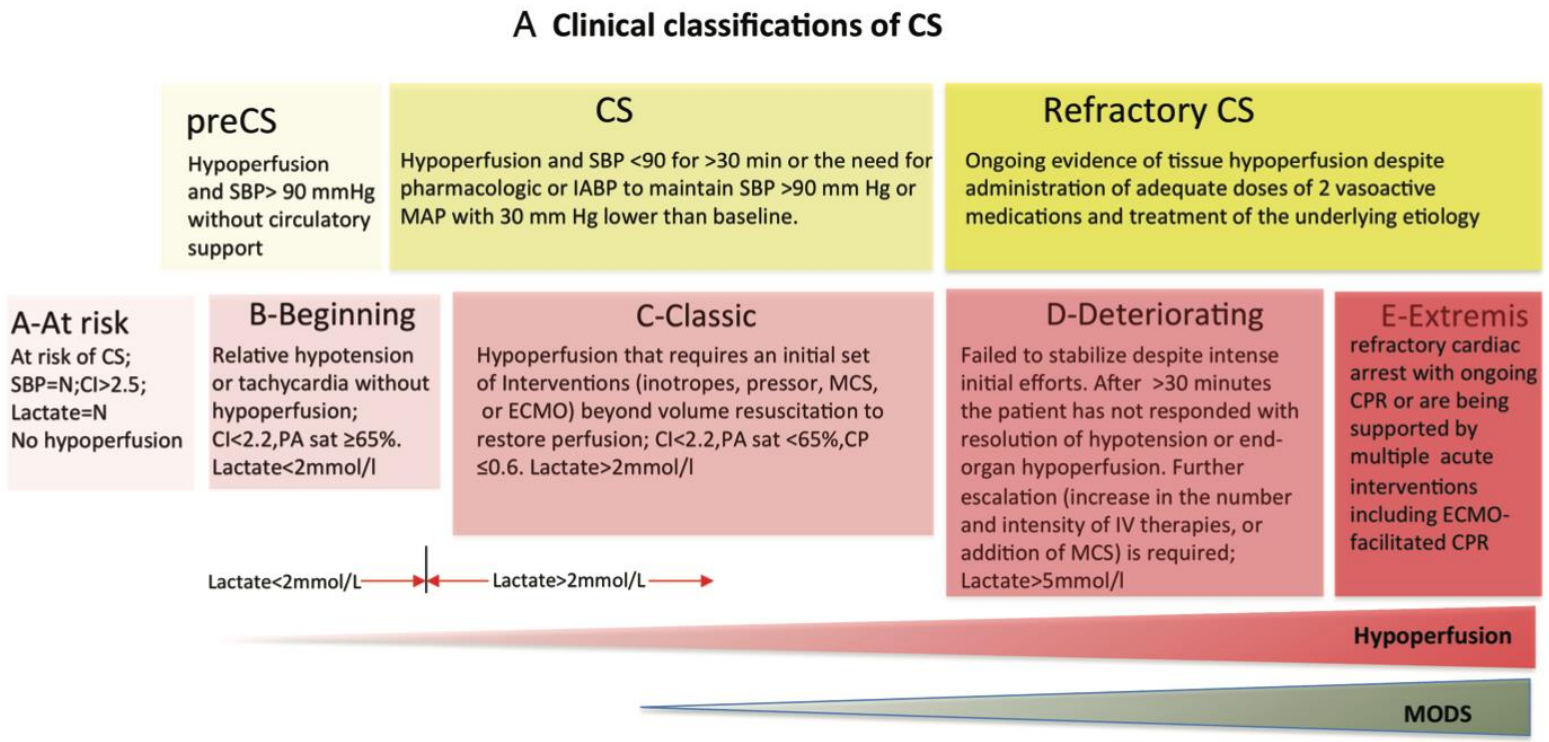


Figure 1. Physical findings suggestive of the ventricle primarily involved in cardiogenic shock. Often pro-inflammatory states induced by shock physiology causes a blunted performance of the less affected side. Both sides often contribute to the clinical presentation and physical exam findings.

Vahdatpour C, Collins D, Goldberg S. Cardiogenic Shock. J Am Heart Assoc. 2019;8(8):e011991. doi:10.1161/JAHA.119.011991

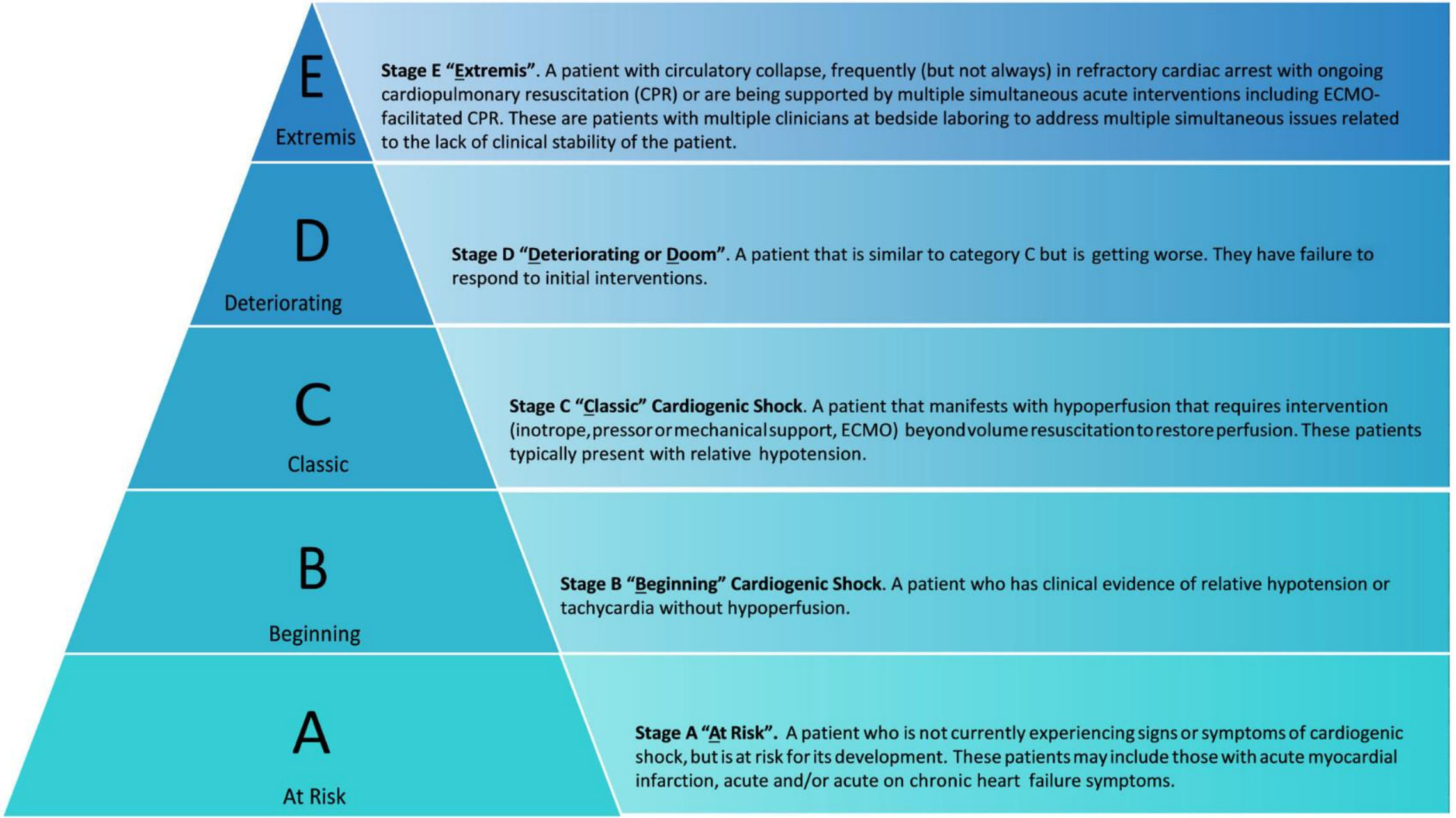
Classificazione



B Hemodynamic classification of CS

SVR ↓;PCWP N ↓;CVP N ↓ "warm-dry"	SVR ↓;PCWP ↑;CVP ↑ "warm-wet"
SVR ↑;PCWP N ↓;CVP N ↓ "cold-dry"	SVR ↑;PCWP ↑;CVP ↑ "cold-wet"

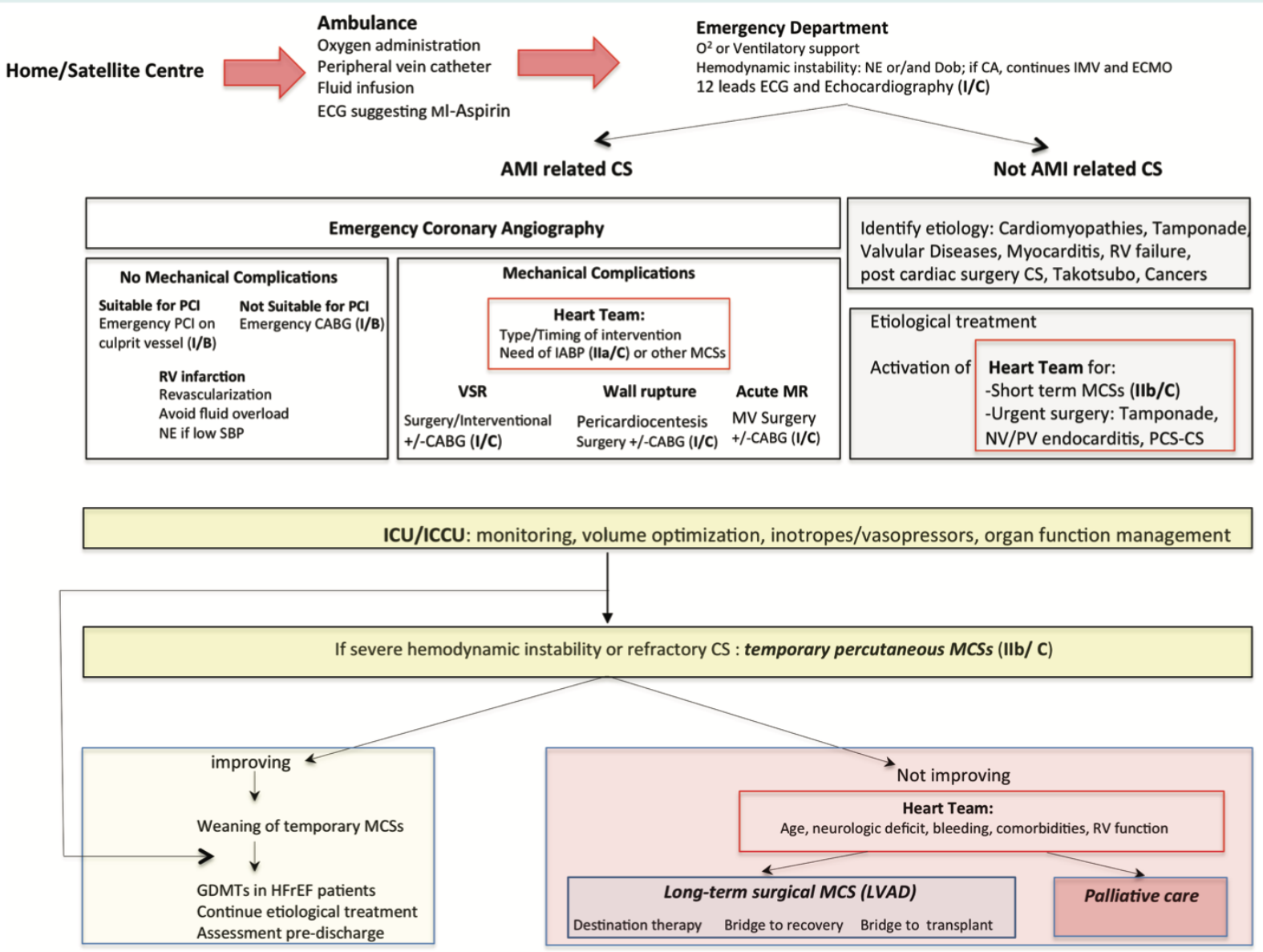
Chioncel O, Parissis J, Mebazaa A, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock - a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2020;22(8):1315-1341. doi:10.1002/ejhf.1922



Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. Catheter Cardiovasc Interv. 2019;94(1):29-37. doi:10.1002/ccd.28329

Domande o interventi?

Gestione dello shock cardiogeno



Chioncel O, Parissis J, Mebazaa A, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock - a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2020;22(8):1315-1341. doi:10.1002/ejhf.1922

SIAARTI
PRO VITA CONTRA DOLOREM SEMPER
BUONE PRATICHE CLINICHE

Uso dei farmaci vasopressori e inotropi nei pazienti critici

AUTORI
Andrea Carsetti, Elena Bignami, Andrea Cortegiani, Katia Donadello, Abele Donati, Giuseppe Foti, Giacomo Grasselli, Stefano Romagnoli, Massimo Antonelli, Elvio De Blasio, Francesco Forfori, Fabio Guarracino, Sabino Scolletta, Luigi Tritapepe, Luigia Scudeller, Maurizio Cecconi, Massimo Girardis

Shock Cardiogeno

Inotropi

- Nel paziente con shock cardiogenico, somministrare un inotropo in presenza di disfunzione cardiaca sistolica, ipotensione e segni di ipoperfusione nonostante un adeguato stato volemico.

- Nel paziente con shock cardiogeno, l'utilizzo di dobutamina è appropriato per migliorare la funzione cardiaca e la perfusione tissutale.

- Nel paziente con shock cardiogeno, non è certo se preferire la dobutamina rispetto al levosimendan o altri farmaci inotropi come agente di prima scelta sia in grado di migliorare la mortalità a breve e lungo termine.

- Nel paziente con shock cardiogeno, l'utilizzo di levosimendan è appropriato per migliorare la funzione cardiaca e la perfusione tissutale, specialmente nei pazienti in terapia con beta-bloccante.

- Nel paziente con shock cardiogeno, non è certa l'appropriatezza dell'utilizzo degli inibitori delle fosfodiesterasi per una ulteriore riduzione del precarico e del postcarico, specialmente in presenza di ipertensione polmonare.

Uso dei farmaci vasopressori e inotropi nei pazienti critici

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Vasopressori

- Nel paziente con shock cardiogenico, aggiungere un vasopressore per trattare un'ipotensione persistente nonostante l'utilizzo dell'inotropo, per mantenere una MAP ≥65 mmHg.
- Nel paziente con shock cardiogeno, la noradrenalina è il vasopressore di prima scelta. L'associazione dobutamina-noradrenalina può rappresentare una scelta efficace nel supportare l'inotropismo e mantenere una MAP ≥65 mmHg.
- Nel paziente con shock cardiogeno, l'utilizzo della dopamina come vasopressore di prima scelta non è appropriato.
- Nel paziente con shock cardiogeno, non è certa l'appropriatezza dell'utilizzo della dopamina come agente vasopressore alternativo alla noradrenalina.
- Nel paziente con shock cardiogeno, non è certa l'appropriatezza dell'utilizzo dell'adrenalina come vasopressore di prima scelta.

SIAARTI
PRO VITA CONTRA DOLOREM SEMPER

BUONE PRATICHE CLINICHE

Uso dei farmaci vasopressori e inotropi nei pazienti critici

AUTORI
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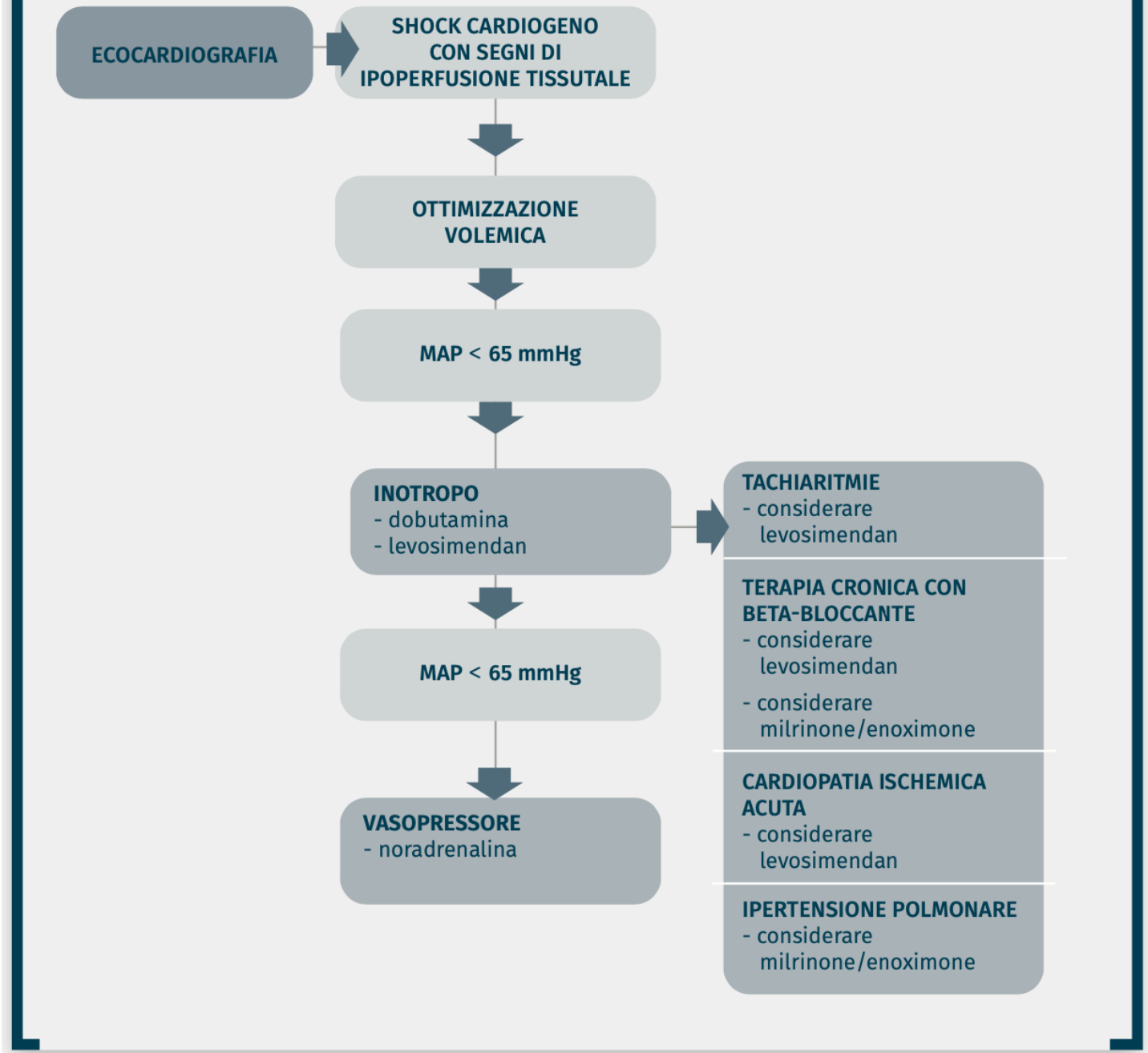
VASOPRESSORI E INOTROPI NELLO SHOCK CARDIOGENO



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GESTIONE INIZIALE DEL PAZIENTE CON SHOCK CARDIOGENO

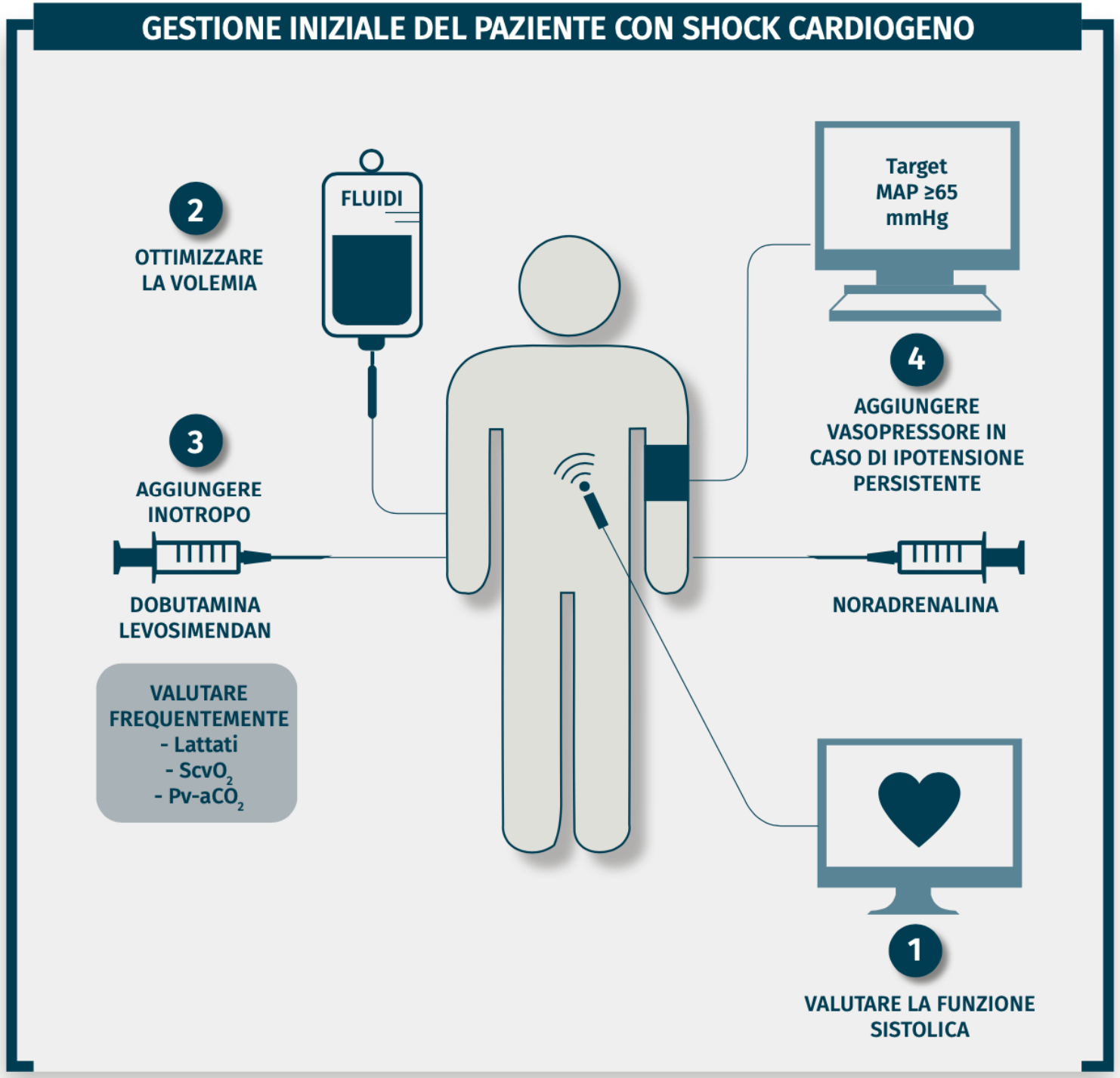


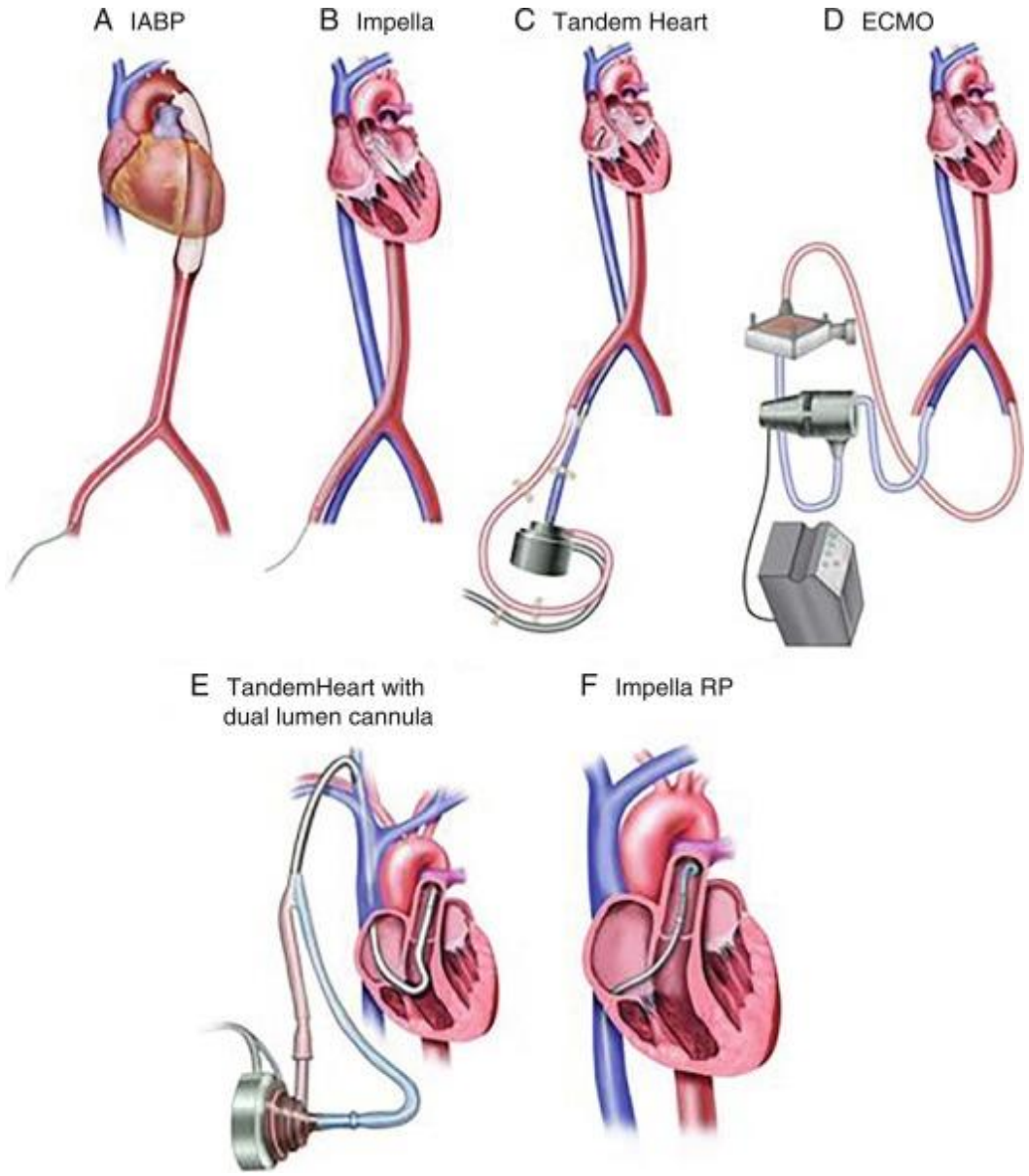
Table 2 Characteristics of short-term mechanical circulatory support

	IABP	Impella (2.5, CP, 5.0[a])	TandemHeart	VA-ECMO
Insertion	Femoral artery to Ao	LV-Ao	Venous cannula: LA Arterial cannula: Ao	Venous cannula: RA/femoral vein Arterial cannula: femoral artery/Ao
Mechanism	ECG triggered (R-wave) Diastolic augmentation of Ao pressure and augments LV performance via systolic balloon deflation (decrease in afterload)	Expels blood from LV to Ao	Aspirates oxygenated blood from LA and returns to Ao	Drainage of deoxygenated venous blood, via an extracorporeal centrifugal pump over a membrane oxygenator and pumped back oxygenated blood to aorta/femoral artery
LV unloading	(+)	++	++	• LV overloading in peripheral cannulation • Only RV unloading
Technical characteristics	• Cannula size 7–8 F • CO • Pulsatile flow	• Cannula size 12–14 F for CP and 21 F for Impella 5.0 • CO: 2.5–5.0 L/min[a] • Continuous flow via axial pump; maximum pump speed 51 000 rpm	• Cannula size 21 F venous and 12–19 F arterial • CO: 4 L/min • Continuous flow via centrifugal pump; maximum pump speed 7500 rpm	• Cannula size 19–25 F venous and 15–19 F arterial • CO: up to 7 L/min • Continuous flow via centrifugal pump; maximum pump speed 5000 rpm
Duration		10 days for Impella 2.5 and CP and 3 weeks for Impella 5.0	2–3 weeks	3–4 weeks
Advantages	Easy insertion, easy to adjust, cath lab not mandatory, no extracorporeal blood; increase coronary and cerebral flow	ECG and pulse-independent, relatively easy insertion in cath lab[a], no extracorporeal blood	Rhythm independent, less artificial surface than ECMO; can be used in patients with Ao stenosis/prosthetic Ao valve; can be used even in LV thrombus	Rhythm independent, no cath lab requirement, rapid insertion, full circulatory support even in resuscitation situations or during malignant arrhythmia, providing combined support of the RV and LV, rapid improvement in oxygenation and the possibility of rapid application, complete cardiopulmonary bypass
Disadvantages	• ECG/pulse-dependent (mostly inefficient in tachycardia and irregular rhythms) • Limb ischaemia • Haemolysis • Thrombocytopenia • Bleeding	• Limb ischaemia • Haemolysis • Bleeding	• Limb ischaemia • Bleeding • Complex implantation requiring transseptal puncture	• Haemolysis, thromboembolic complications (large artificial surface), renal failure, limb ischaemia/amputation and bleeding • LV overloading- peripheral cannulation is associated with an increased LV afterload, which produces LV distension and pulmonary congestion and may impair myocardial recovery.[103,143] LV decompression strategies include additional procedures, such as septostomy, IABP, Impella, and hybrid circuit configuration • Harlequin syndrome (upper body hypoxia from incomplete retrograde filling and oxygenation), in which deoxygenated cerebral blood flow occurs during retrograde perfusion with peripheral cannulation. The veno-arterio-venous configuration with triple cannulation avoids upper body hypoxia
Contraindications	• Moderate to severe aortic regurgitation • Severe aortic disease	• Severe aortic stenosis • Prosthetic aortic valve • LV thrombus • VSD • Peripheral vascular disease	• Severe aortic insufficiency • Aortic dissection • Peripheral vascular disease • RV failure • VSD • Inability to tolerate systemic anticoagulation	• Severe aortic insufficiency • Aortic dissection • Inability to tolerate systemic anticoagulation

Ao, aorta; CO, cardiac output; ECG, electrocardiogram; IABP, intra-aortic balloon pump; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; VA-ECMO, veno-arterial extracorporeal membrane oxygenation; VSD, ventricular septal defect. Percutaneous mechanical circulatory support can be characterized by one of four circuit configurations: (i) intra-aortic devices (IABP), (ii) transvalvular aortic (Impella), (iii) LA to systemic artery (TandemHeart), and (iv) RA to systemic artery (VA-ECMO).
[a]For Impella 5.0 surgical cut-down for cannulation is mandatory.

Chioncel O, Parissis J, Mebazaa A, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock - a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2020;22(8):1315-1341. doi:10.1002/ejhf.1922

Supporto meccanico



Kirklin JK, Rogers JG. Mechanical Circulatory Support: A Companion to Braunwald's Heart Disease Ebook. Elsevier Health Sciences; 2019.

Domande o interventi?

Shock ostruttivo

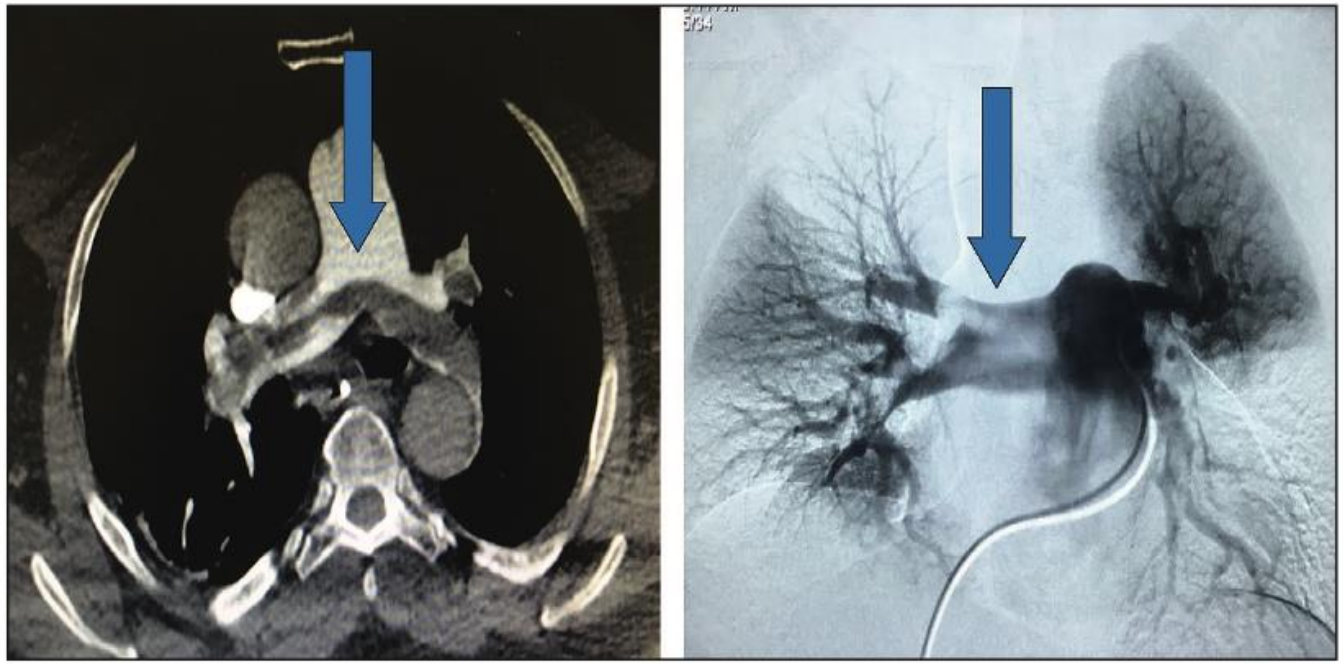


Figure 1. CT scan and pulmonary angiogram.

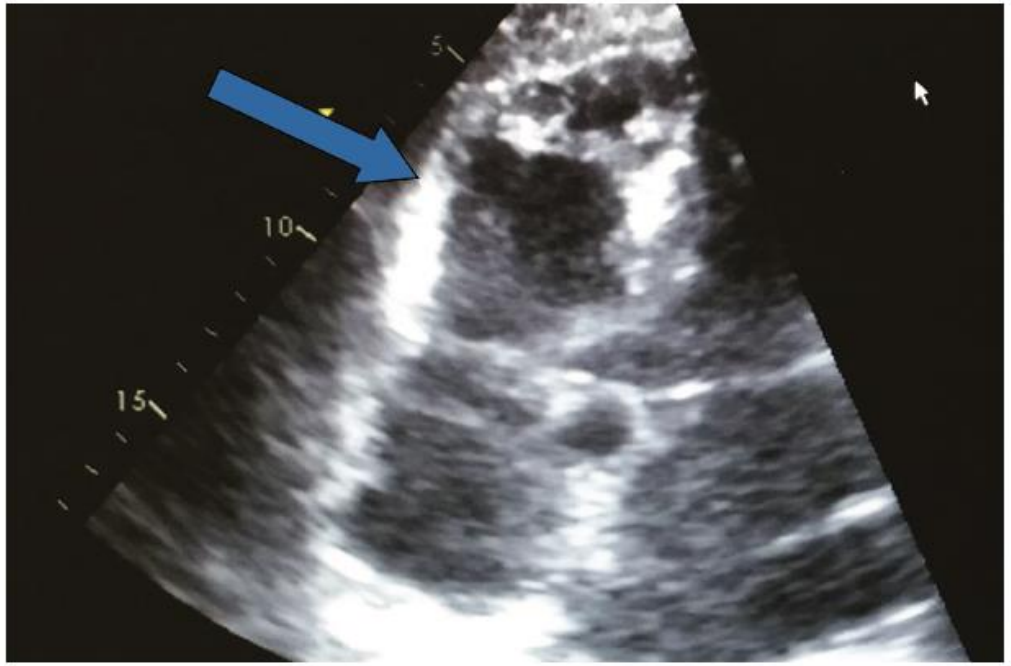
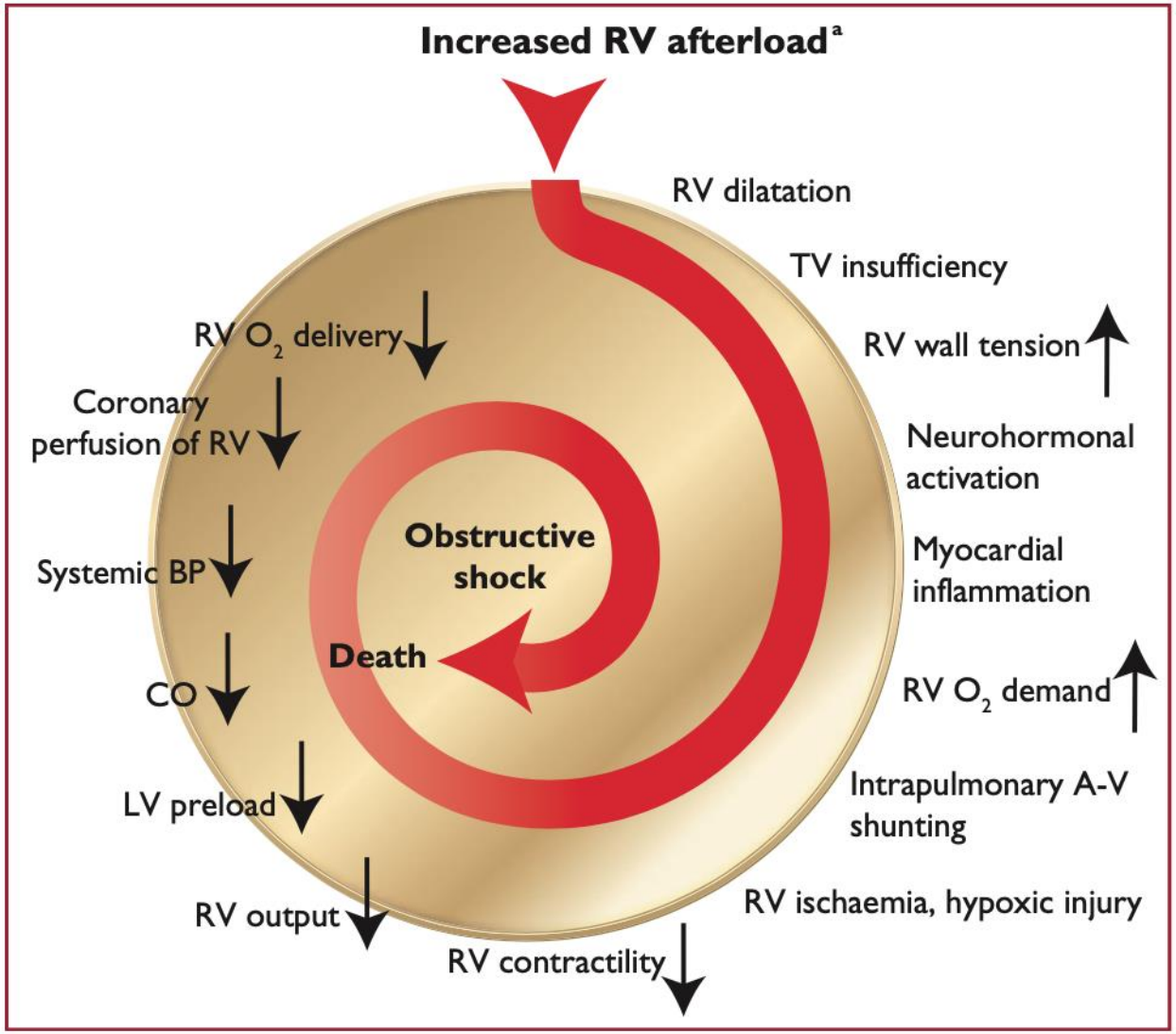


Figure 2. Dilated right ventricle with McConnell's sign.

https://evtoday.com/articles/2016-sept/ekosonic-endovascular-system-and-rp-impella-support-acute-right-ventricular-failure-from-massive-pulmonary-embolism

Fisiopatologia Embolia Polmonare



Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405

Fattori di rischio per Embolia Polmonare

Strong risk factors (OR > 10)
Fracture of lower limb
Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months)
Hip or knee replacement
Major trauma
Myocardial infarction (within previous 3 months)
Previous VTE
Spinal cord injury

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Moderate risk factors (OR 2–9)
Arthroscopic knee surgery
Autoimmune diseases
Blood transfusion
Central venous lines
Intravenous catheters and leads
Chemotherapy
Congestive heart failure or respiratory failure
Erythropoiesis-stimulating agents
Hormone replacement therapy (depends on formulation)
In vitro fertilization
Oral contraceptive therapy
Post-partum period
Infection (specifically pneumonia, urinary tract infection, and HIV)
Inflammatory bowel disease
Cancer (highest risk in metastatic disease)
Paralytic stroke
Superficial vein thrombosis
Thrombophilia

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Weak risk factors (OR < 2)

Bed rest >3 days

Diabetes mellitus

Arterial hypertension

Immobility due to sitting (e.g. prolonged car or air travel)

Increasing age

Laparoscopic surgery (e.g. cholecystectomy)

Obesity

Pregnancy

Varicose veins

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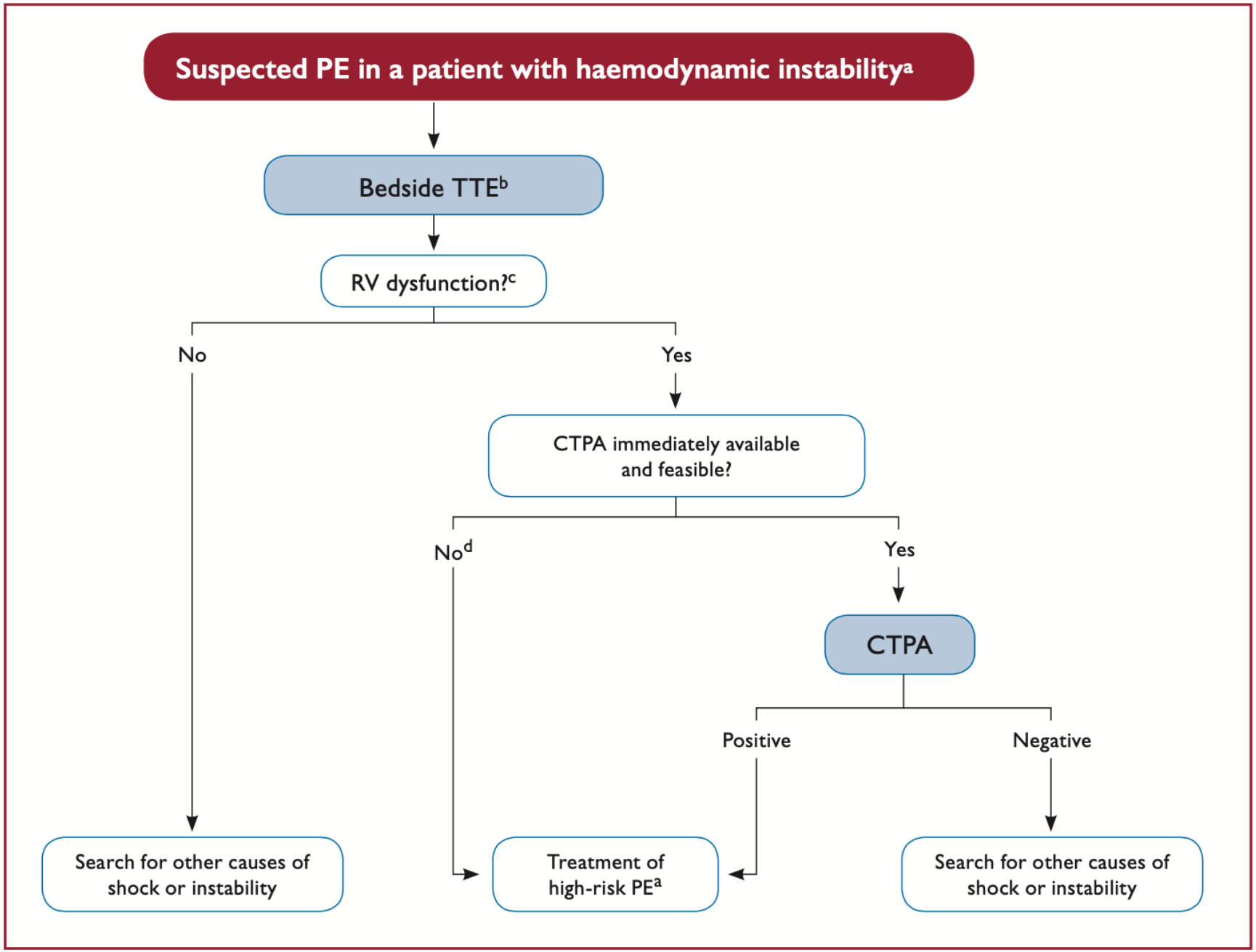
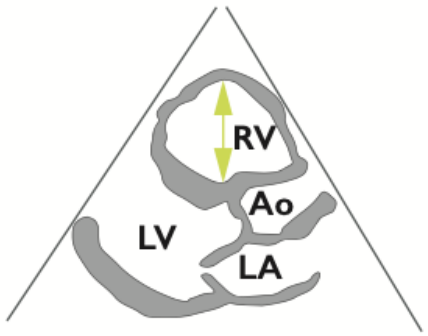


Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405

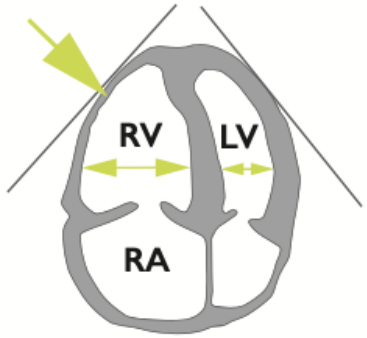
Table 4 **Definition of haemodynamic instability, which delineates acute high-risk pulmonary embolism (one of the following clinical manifestations at presentation)**

(1) Cardiac arrest	(2) Obstructive shock[68–70]	(3) Persistent hypotension
Need for cardiopulmonary resuscitation	Systolic BP < 90 mmHg or vasopressors required to achieve a BP ≥90 mmHg despite adequate filling status	Systolic BP < 90 mmHg or systolic BP drop ≥40 mmHg, lasting longer than 15 min and not caused by new-onset arrhythmia, hypovolaemia, or sepsis
	And	
	End-organ hypoperfusion (altered mental status; cold, clammy skin; oliguria/anuria; increased serum lactate)	

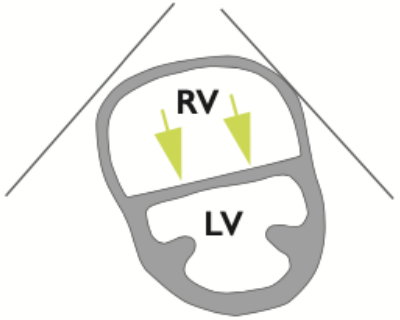
Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405



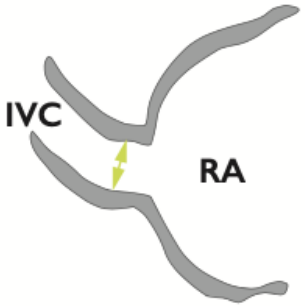
A. Enlarged right ventricle, parasternal long axis view



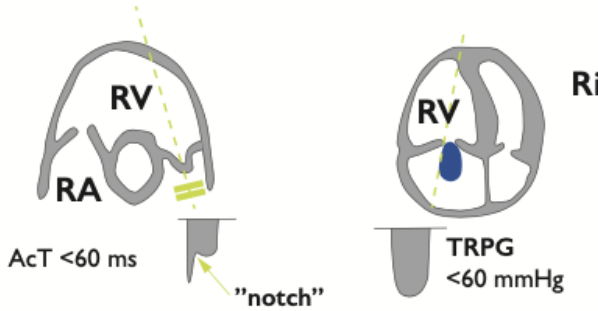
B. Dilated RV with basal RV/LV ratio >1.0, and McConnell sign (arrow), four chamber view



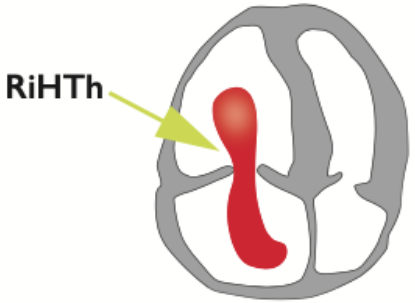
C. Flattened intraventricle septum (arrows) parasternal short axis view



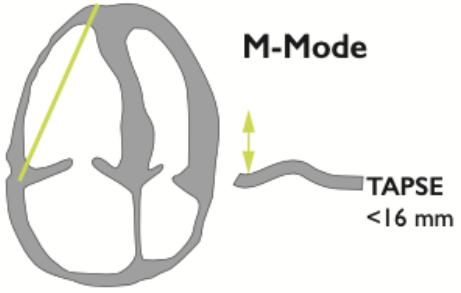
D. Distended inferior vena cava with diminished inspiratory collapsibility, subcostal view



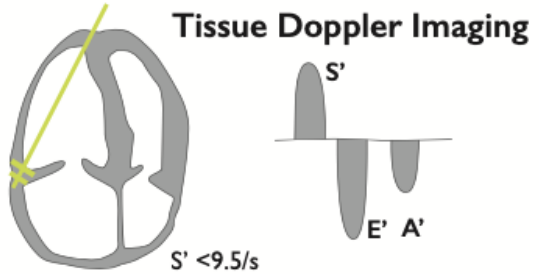
E. 60/60 sign: coexistence of acceleration time of pulmonary ejection <60 ms and midsystolic "notch" with mildy elevated (<60 mmHg) peak systolic gradient at the tricuspic valve



F. Right heart mobile thrombus detected in right heart cavities (arrow)



G. Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<16 mm)



H. Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s)

Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405

Table 6 **Imaging tests for diagnosis of pulmonary embolism**

	Strengths	Weaknesses/limitations	Radiation issues[a]

CTPA	• Readily available around the clock in most centres
	• Excellent accuracy
	• Strong validation in prospective management outcome studies
	• Low rate of inconclusive results (3–5%)
	• May provide alternative diagnosis if PE excluded
	• Short acquisition time
		• Radiation exposure
		• Exposure to iodine contrast:
		  ○ limited use in iodine allergy and hyperthyroidism
		  ○ risks in pregnant and breastfeeding women
		  ○ contraindicated in severe renal failure
		• Tendency to overuse because of easy accessibility
		• Clinical relevance of CTPA diagnosis of subsegmental PE unknown
			• Radiation effective dose 3–10 mSv[b]
			• Significant radiation exposure to young female breast tissue

Planar V/Q scan	• Almost no contraindications • Relatively inexpensive • Strong validation in prospective management outcome studies	• Not readily available in all centres • Interobserver variability in interpretation • Results reported as likelihood ratios • Inconclusive in 50% of cases • Cannot provide alternative diagnosis if PE excluded	• Lower radiation than CTPA, effective dose ~2 mSv[b]
V/Q SPECT	• Almost no contraindications • Lowest rate of non-diagnostic tests (<3%) • High accuracy according to available data • Binary interpretation ('PE' vs. 'no PE')	• Variability of techniques • Variability of diagnostic criteria • Cannot provide alternative diagnosis if PE excluded • No validation in prospective management outcome studies	• Lower radiation than CTPA, effective dose ~2 mSv[b]
Pulmonary angiography	• Historical gold standard	• Invasive procedure • Not readily available in all centres	• Highest radiation, effective dose 10–20 mSv[b]

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6.6 Recommendations for acute-phase treatment of high-risk pulmonary embolism[a]

Recommendations	Class[b]	Level[c]
It is recommended that anticoagulation with UFH, including a weight-adjusted bolus injection, be initiated without delay in patients with high-risk PE.	I	C
Systemic thrombolytic therapy is recommended for high-risk PE. [282]	I	B
Surgical pulmonary embolectomy is recommended for patients with high-risk PE, in whom thrombolysis is contraindicated or has failed.[d] [281]	I	C
Percutaneous catheter-directed treatment should be considered for patients with high-risk PE, in whom thrombolysis is contraindicated or has failed.[d]	IIa	C
Norepinephrine and/or dobutamine should be considered in patients with high-risk PE.	IIa	C
ECMO may be considered, in combination with surgical embolectomy or catheter-directed treatment, in patients with PE and refractory circulatory collapse or cardiac arrest.[d] [252]	IIb	C

ECMO = extracorporeal membrane oxygenation; PE = pulmonary embolism; UFH = unfractionated heparin.

[a]See *Table 4* for definition of high-risk PE. After haemodynamic stabilization of the patient, continue with anticoagulation treatment as in intermediate- or low-risk PE (*section 6.7*).

[b]Class of recommendation.

[c]Level of evidence.

[d]If appropriate expertise and resources are available on-site.

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Table 9 Treatment of right ventricular failure in acute high-risk pulmonary embolism

Strategy	Properties and use	Caveats
Volume optimization		
Cautious volume loading, saline, or Ringer's lactate, ≤500 mL over 15–30 min	Consider in patients with normal–low central venous pressure (due, for example, to concomitant hypovolaemia)	Volume loading can over-distend the RV, worsen ventricular interdependence, and reduce CO[239]
Vasopressors and inotropes		
Norepinephrine, 0.2–1.0 μg/kg/min[a] [240]	Increases RV inotropy and systemic BP, promotes positive ventricular interactions, and restores coronary perfusion gradient	Excessive vasoconstriction may worsen tissue perfusion
Dobutamine, 2–20 μg/kg/min[241]	Increases RV inotropy, lowers filling pressures	May aggravate arterial hypotension if used alone, without a vasopressor; may trigger or aggravate arrhythmias
Mechanical circulatory support		
Veno–arterial ECMO/extracorporeal life support[251,252,258]	Rapid short-term support combined with oxygenator	Complications with use over longer periods (>5–10 days), including bleeding and infections; no clinical benefit unless combined with surgical embolectomy; requires an experienced team

CO = cardiac output; BP = blood pressure; ECMO = extracorporeal membrane oxygenation; RV = right ventricle/ventricular.
[a]Epinephrine is used in cardiac arrest.

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Table 10 Thrombolytic regimens, doses, and contraindications

Molecule	Regimen	Contraindications to fibrinolysis
rtPA	100 mg over 2 h	**Absolute**
	0.6 mg/kg over 15 min (maximum dose 50 mg)[a]	History of haemorrhagic stroke or stroke of unknown origin
Streptokinase	250 000 IU as a loading dose over 30 min, followed by 100 000 IU/h over 12–24 h	Ischaemic stroke in previous 6 months
	Accelerated regimen: 1.5 million IU over 2 h	Central nervous system neoplasm
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h over 12–24 h	Major trauma, surgery, or head injury in previous 3 weeks
	Accelerated regimen: 3 million IU over 2 h	Bleeding diathesis
		Active bleeding
		Relative
		Transient ischaemic attack in previous 6 months
		Oral anticoagulation
		Pregnancy or first post-partum week
		Non-compressible puncture sites
		Traumatic resuscitation
		Refractory hypertension (systolic BP >180 mmHg)
		Advanced liver disease
		Infective endocarditis
		Active peptic ulcer

BP = blood pressure; IU = international units; rtPA, recombinant tissue-type plasminogen activator.

[a]This is the accelerated regimen for rtPA in pulmonary embolism; it is not officially approved, but it is sometimes used in extreme haemodynamic instability such as cardiac arrest.

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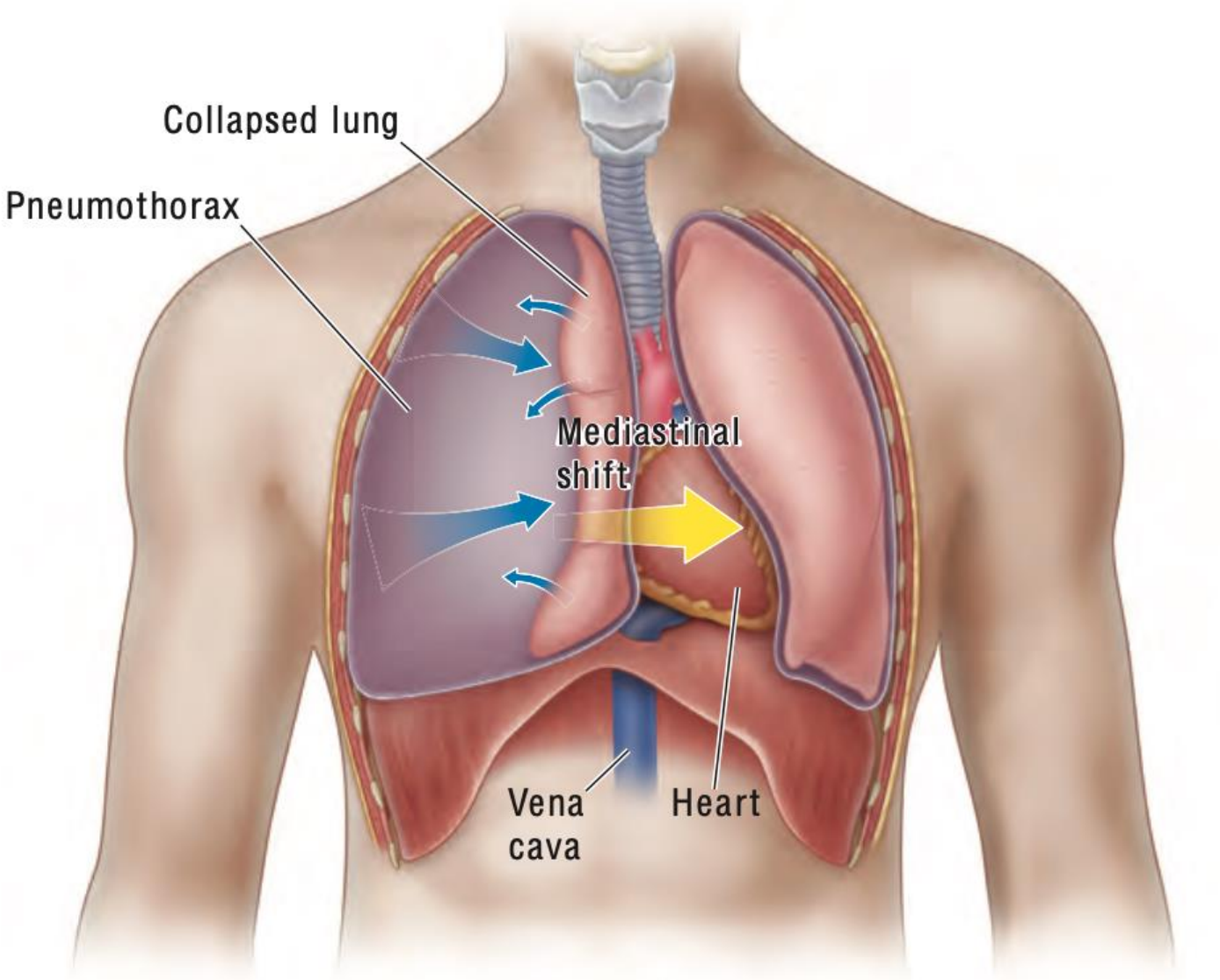
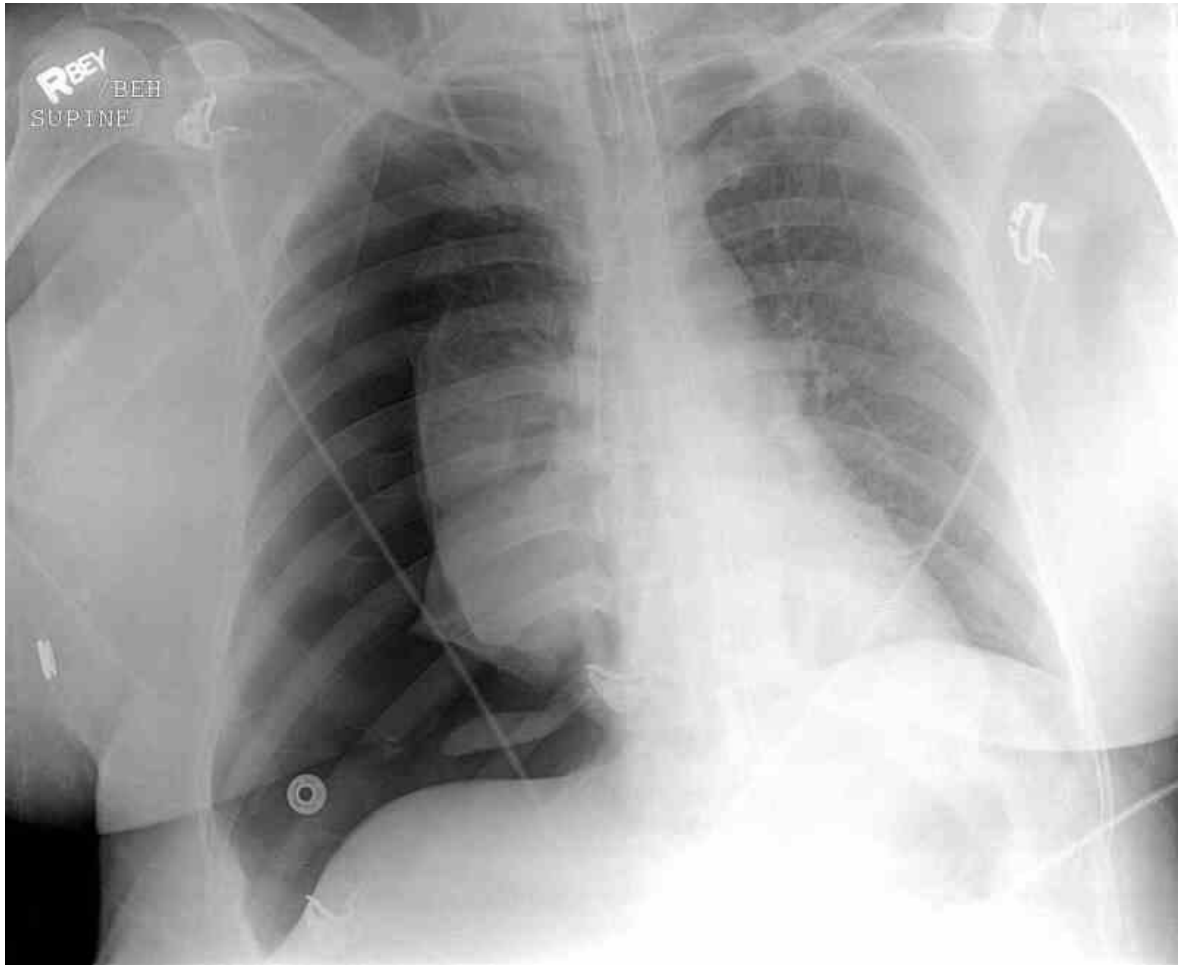
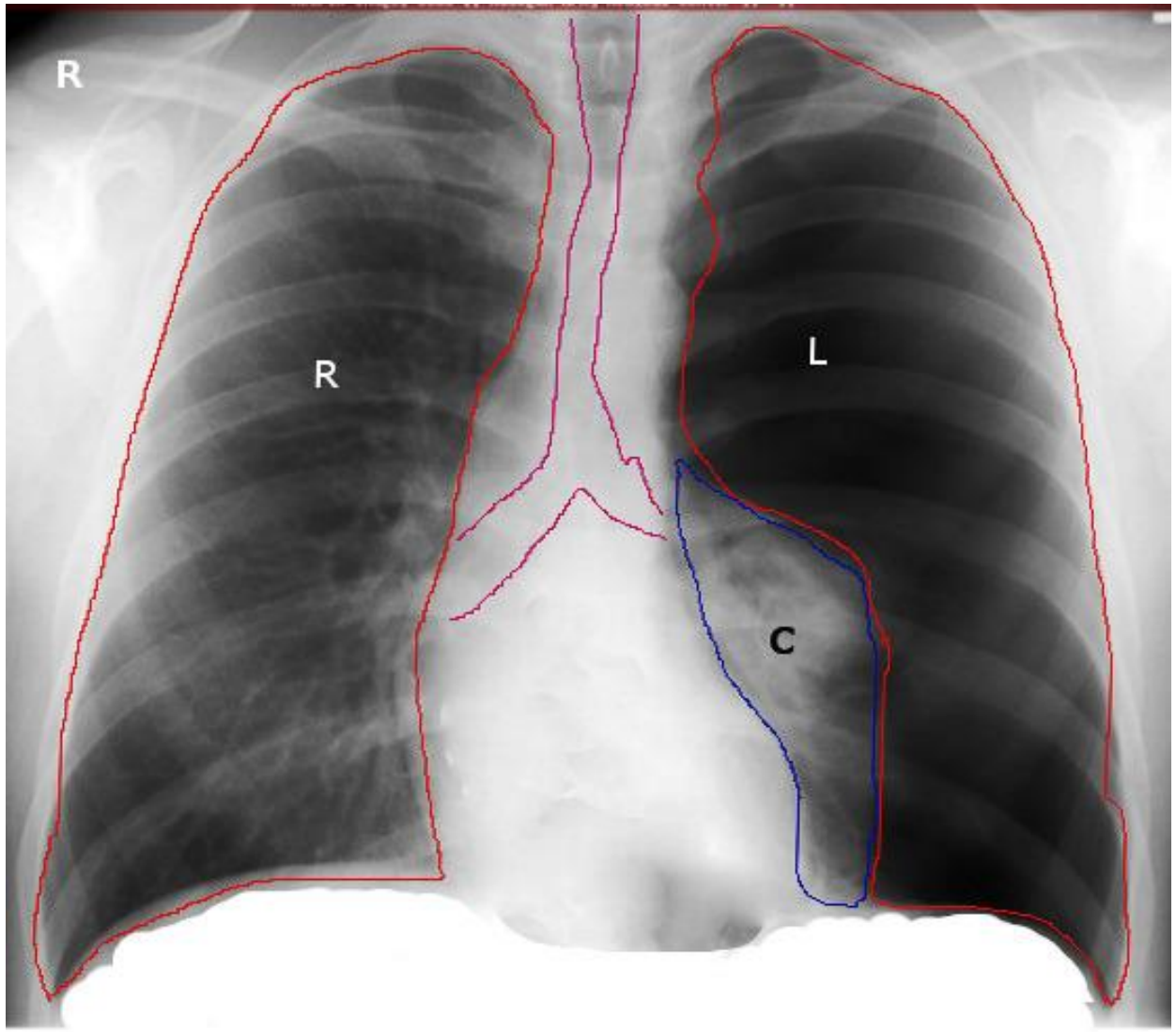
Domande o interventi?

Pneumotorace iperteso



ATLS® : Advanced Trauma Life Support Student Course Manual. American College of Surgeons; 2018.





Jalota R, Sayad E. Tension Pneumothorax. In: StatPearls. StatPearls Publishing; 2021. Accessed December 31, 2021. http://www.ncbi.nlm.nih.gov/books/NBK559090/

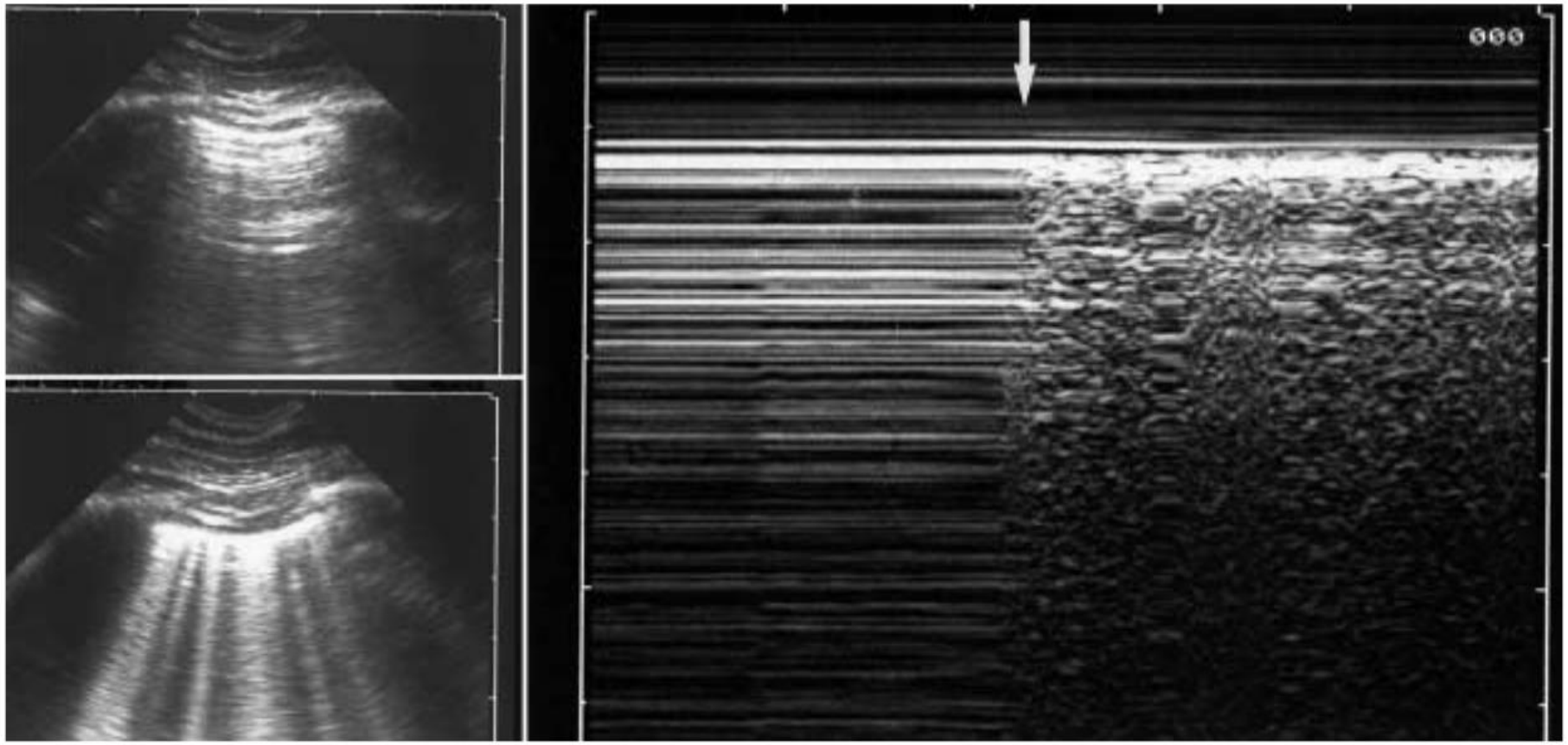


Fig. 5 The "lung point" (ultrasound, longitudinal scan of the third intercostal space along the anterior axillary line, supine patient with pneumothorax). *Left, top*: expiration. Absence of lung sliding plus "A lines". *Left, bottom*: inspiration. Fleeting appearance of lung sliding with "lung rockets". *Right*: time-motion mode clearly shows the sudden *(arrow)* inspiratory appearance of a granulous pattern beneath the pleural line

Lichtenstein D, Mezière G, Biderman P, Gepner A. The "lung point": an ultrasound sign specific to pneumothorax. Intensive Care Med. 2000;26(10):1434-1440. doi:10.1007/s001340000627

Iatrogenic: (Induced by a medical procedure)

- Central venous catheterization in the subclavian or internal jugular vein
- Lung biopsy
- Barotrauma due to positive pressure ventilation
- Percutaneous tracheostomy
- Thoracentesis
- Pacemaker insertion
- Bronchoscopy
- Cardiopulmonary resuscitation
- Intercostal nerve block

Non-Iatrogenic: (Due to external trauma)

- Penetrating or blunt trauma
- Rib fracture
- Diving or flying

Causes of tension pneumothorax:

All the above causes can further cause tension pneumothorax as well as:

- Idiopathic spontaneous pneumothorax
- Open pneumothorax
- Conversion of spontaneous pneumothorax to tension

Jalota R, Sayad E. Tension Pneumothorax. In: StatPearls. StatPearls Publishing; 2021. Accessed December 31, 2021. http://www.ncbi.nlm.nih.gov/books/NBK559090/

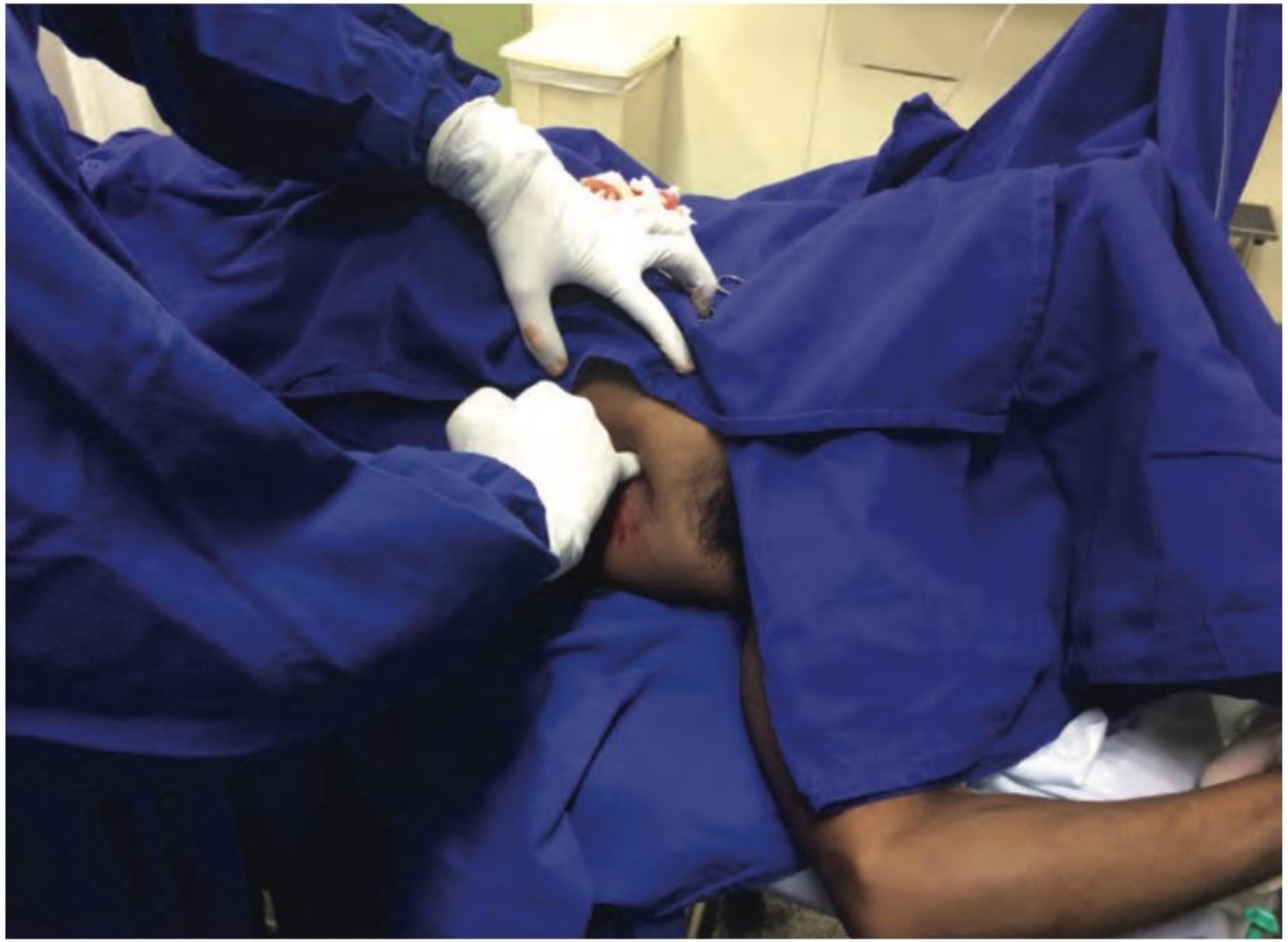
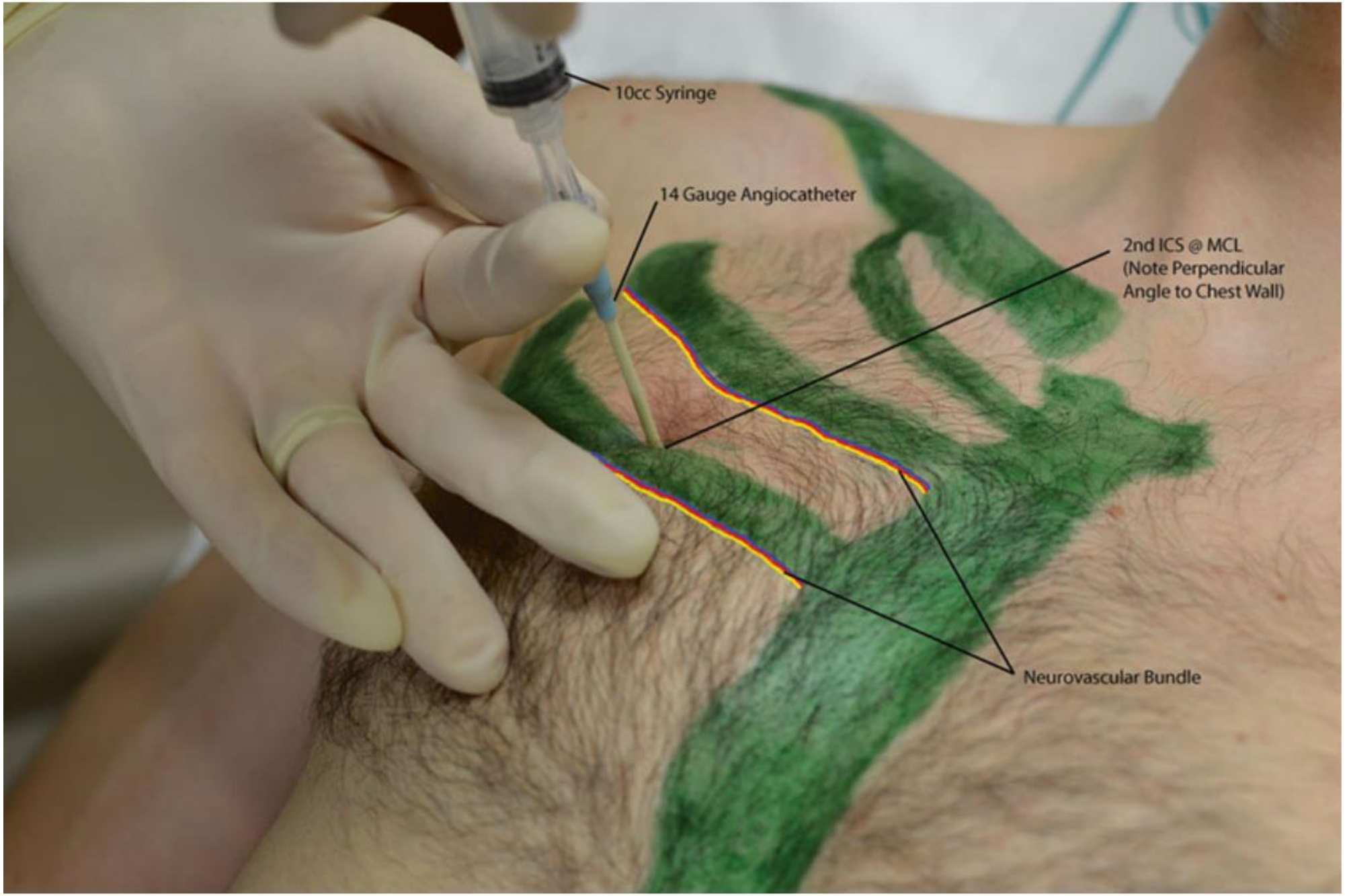
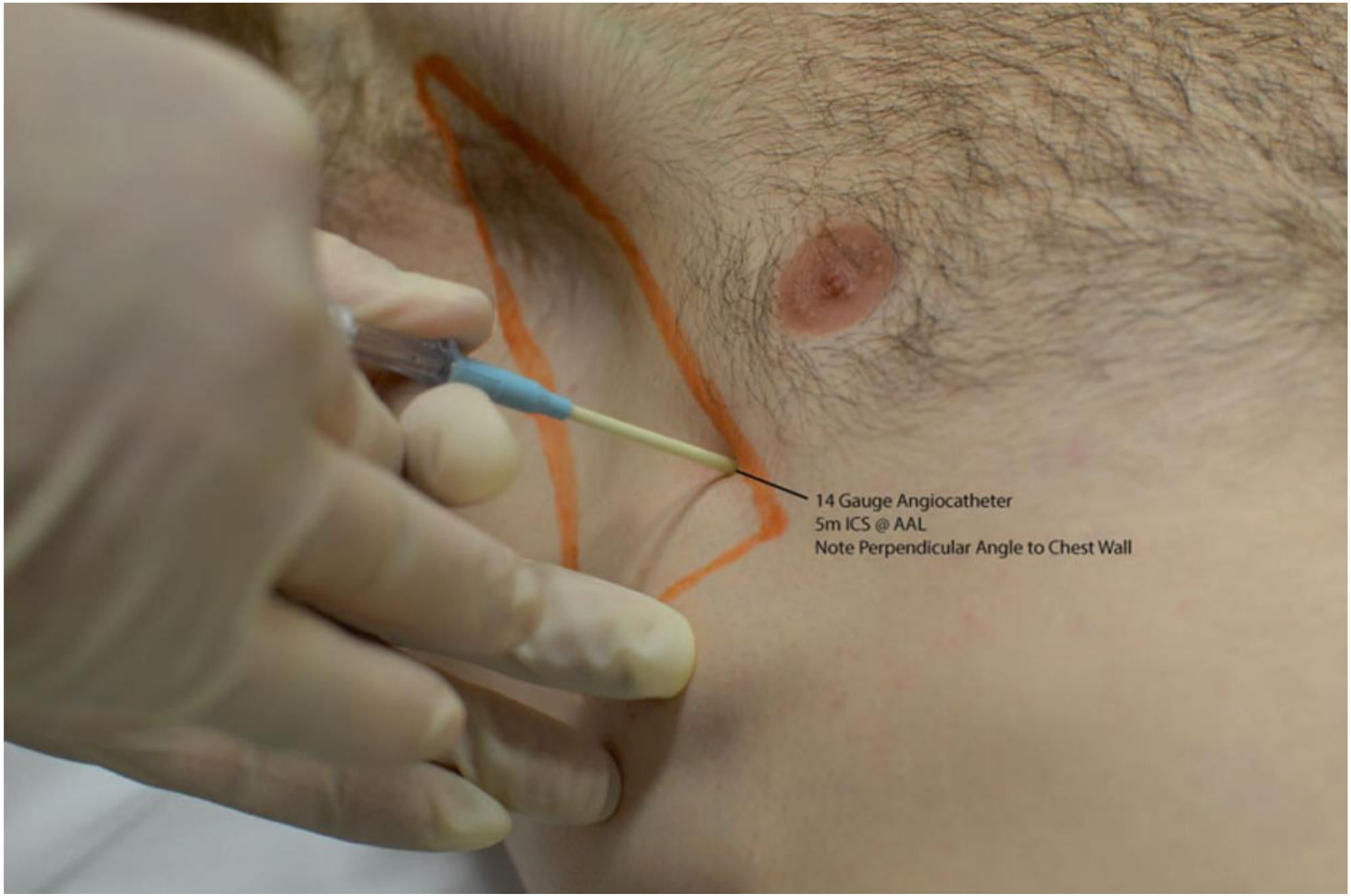


FIGURE 4-2 Finger Decompression. Tension pneumothorax can be managed initially by rapidly applying the finger decompression technique.

ATLS® : Advanced Trauma Life Support Student Course Manual. American College of Surgeons; 2018.



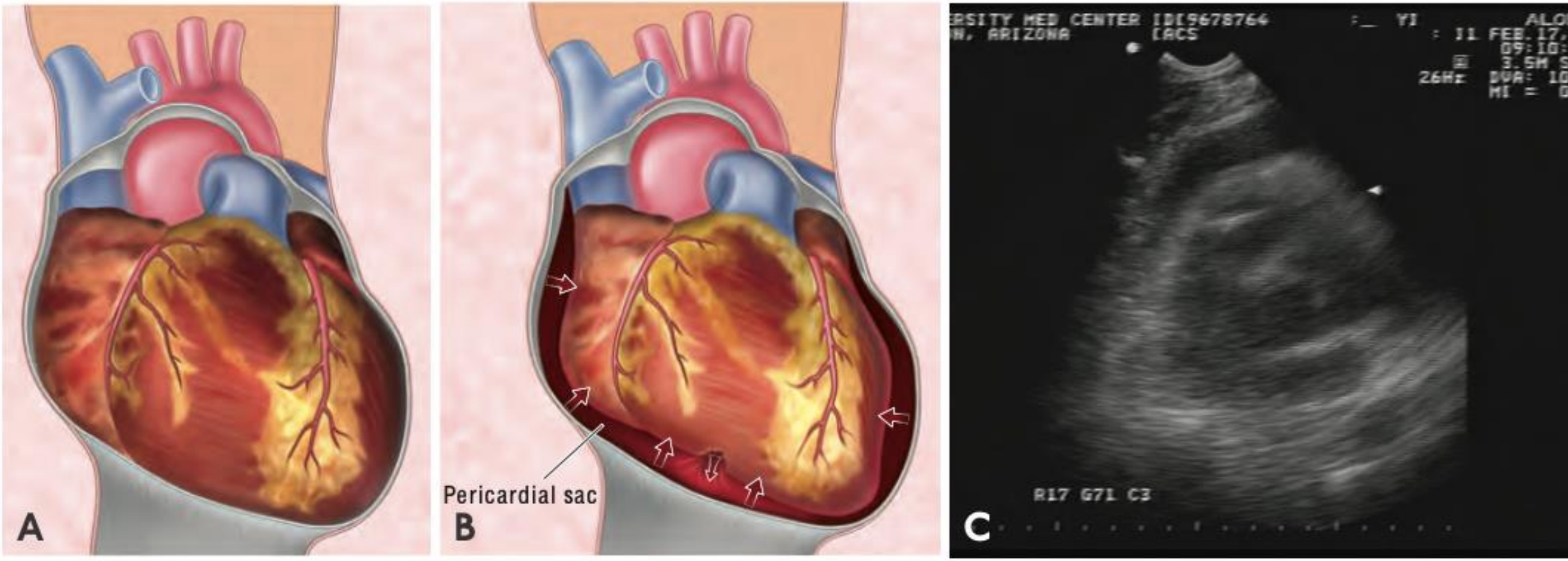
Taylor DA, Sherry SP, Sing RF. Interventional Critical Care: A Manual for Advanced Care Practitioners. Springer; 2016.



Taylor DA, Sherry SP, Sing RF. Interventional Critical Care: A Manual for Advanced Care Practitioners. Springer; 2016.

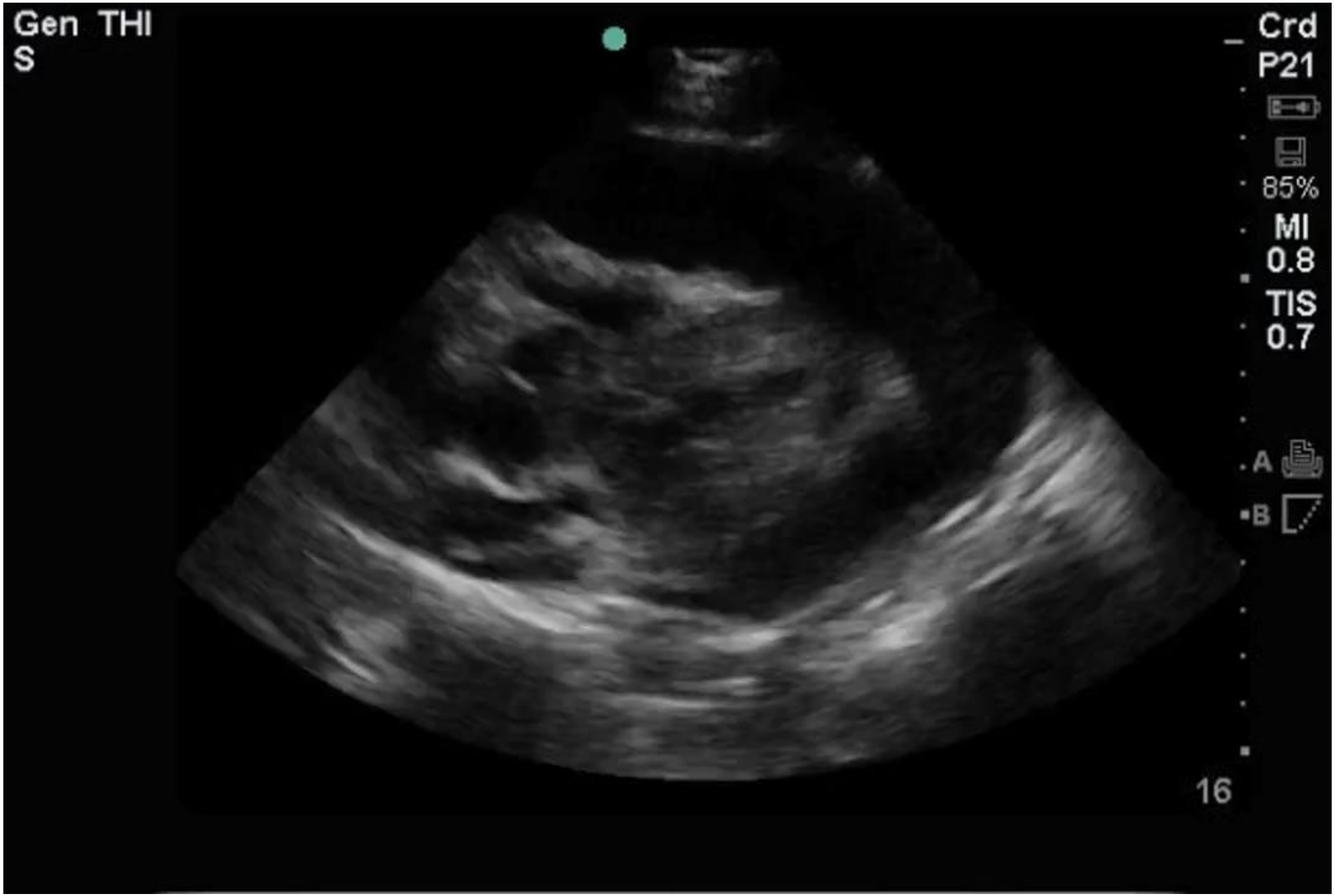
Domande o interventi?

Tamponamento cardiaco



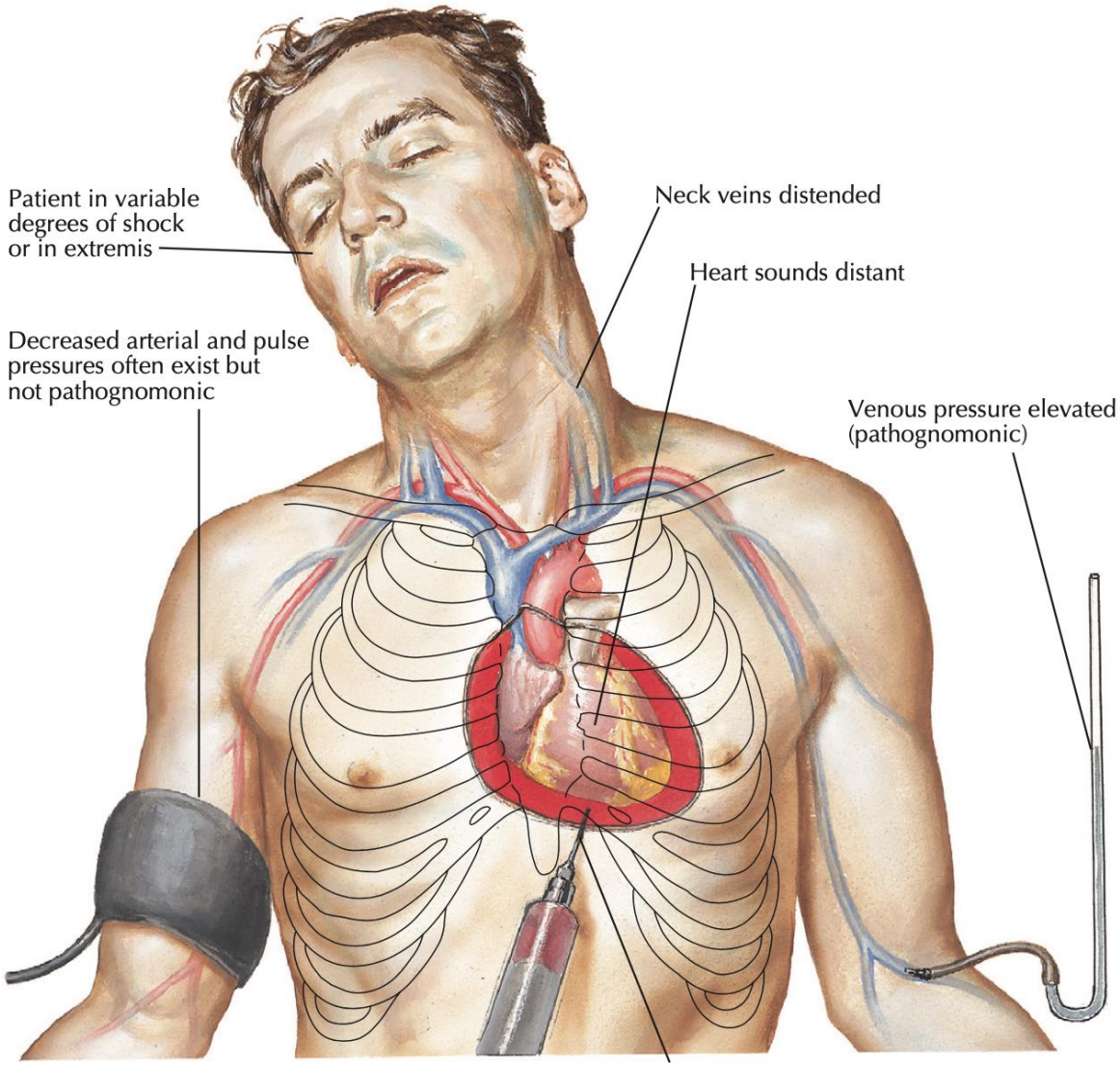
■ **FIGURE 4-6 Cardiac Tamponade. A.** Normal heart. **B.** Cardiac tamponade can result from penetrating or blunt injuries that cause the pericardium to fill with blood from the heart, great vessels, or pericardial vessels. **C.** Ultrasound image showing cardiac tamponade.

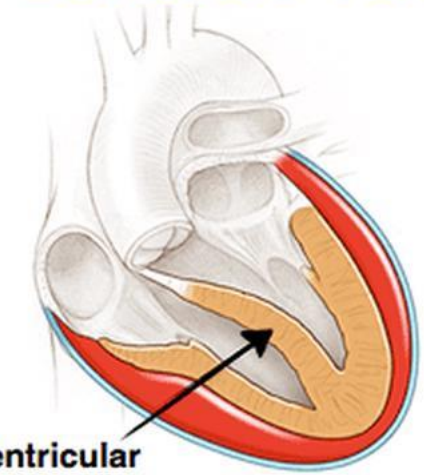
ATLS® : Advanced Trauma Life Support Student Course Manual. American College of Surgeons; 2018.



Sharma NK, Waymack JR. Acute Cardiac Tamponade. In: StatPearls. StatPearls Publishing; 2021. Accessed December 31, 2021. http://www.ncbi.nlm.nih.gov/books/NBK534806/

Cardiac Tamponade





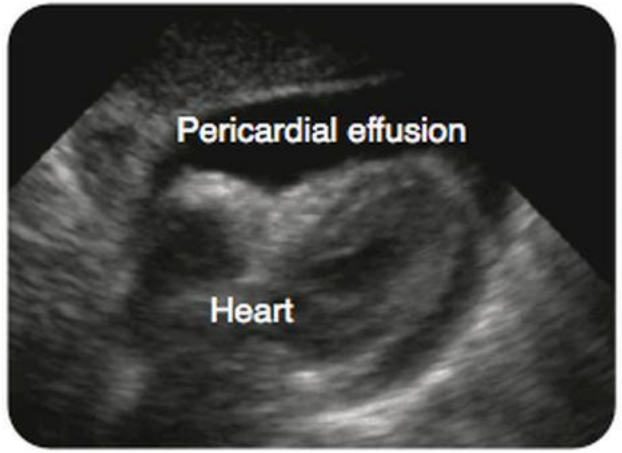
Beck's Triad

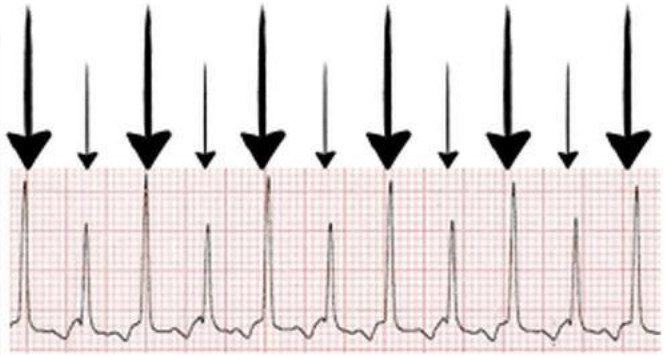
1. Hypotension
2. Jugular venous distension
3. Muffled heart sounds

Don't mix up with:

Tension pneumothorax

1. Hypotension
2. Jugular venous distension
3. **Absent breath sounds**





Electrical Alternans

Conti CR. The Netter Collection of Medical Illustrations: Cardiovascular System. Elsevier - Health Sciences Division; 2014.
https://www.grepmed.com/images/5140/tamponade-pericardial-cardiac-cardiology-triad

Domande o interventi?

Shock distributivo – neurogeno

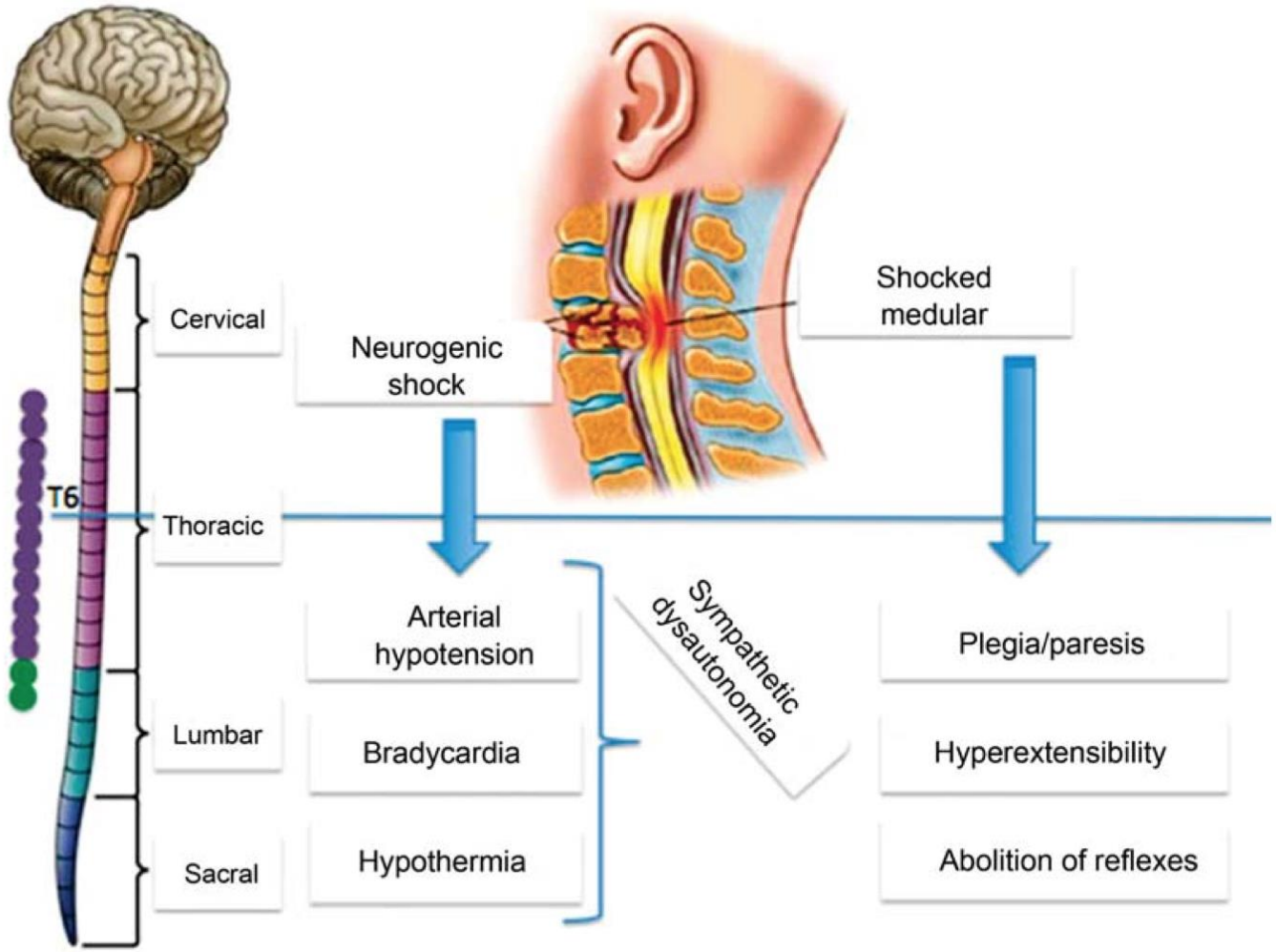


Fig. 3 Neurogenic shock is the result, in most cases, of the high spinal cord injury (above T6) that presents with medullary shock (plegia or paresis), hyperextensibility and hypo or deep osteotendinous and superficial cutaneous injury below the lesion. There are also reports of trauma to the lumbar spinal cord.

Damiani D. Neurogenic shock: clinical management and particularities in an emergency room. Arquivos Brasileiros de Neurocirurgia: Brazilian Neurosurgery. 2018;37(03):196-205.

Key messages

What is already known on this subject?

- Animal studies show that neurogenic shock does not always present immediately postinjury.
- There are no agreed criteria used to define neurogenic shock.

What this study adds?

- The presentation of neurogenic shock is highly variable in both time and severity. Healthcare professionals should be aware of the possibility of neurogenic shock in the prehospital and hospital environment.
- A more sensitive than specific screening criteria could help physicians better identify neurogenic shock, leading to more appropriate treatment and better patient outcome.

Table 1 A summary of the criteria used to define neurogenic shock from a selection of papers

Paper	sBP (mm Hg)	HR (bpm)
Bernhard *et al*[9]	<70	<60
Grigorean *et al*[10]	<90	–
Guly *et al*[7]	<100	<80
Lehmann *et al*[8]	<90	–
Levi *et al*[11]	<90	–
Ley *et al*[12]	≤90	≤90
Moerman *et al*[13]	<80	<60
Zipnick *et al*[14]	<100	<80

The systolic BP (sBP) in mm Hg and HR in bpm were recorded if discussed in the paper.

Taylor MP, Wrenn P, O'Donnell AD. Presentation of neurogenic shock within the emergency department. Emerg Med J. 2017;34(3):157-162. doi:10.1136/emermed-2016-205780

Shock anafilattico

TABLE 1] Criteria for Diagnosis of Anaphylaxis

Anaphylaxis is highly likely if any *one* of the following three conditions is satisfied.	
1.	Acute onset of illness with:
	Mucocutaneous involvement (pruritus, flushing, urticaria, angioedema) *and* one of the following:
	A. Respiratory complications (wheezing, stridor, hypoxemia/cyanosis)
	B. Hypotension[a] or end-organ damage (encephalopathy, kidney injury, etc.)
2.	Two or more of the following occurring rapidly after exposure to *known* or *likely* allergen:
	• Mucocutaneous involvement (pruritus, flushing, urticaria, angioedema)
	• Respiratory complications (wheezing, stridor, hypoxemia/cyanosis)
	• Hypotension[a] or evidence of end organ hypoperfusion (encephalopathy, kidney injury, etc.)
	• Persistent gastrointestinal symptoms (pain, nausea, vomiting)
3.	Reduced BP soon after exposure to a *known* allergen.

[a]Hypotension in adults is regarded as systolic BP of <90 mm Hg or greater than a 30% decrease in systolic BP from the patient's baseline. Hypotension in infants and children: systolic BP <70 mm Hg (1-12 months); <(70 mm Hg + [2x age]) (1-10 years); <90 mm Hg (11-17 years); or >30% decrease in systolic BP.

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

Patogenesi

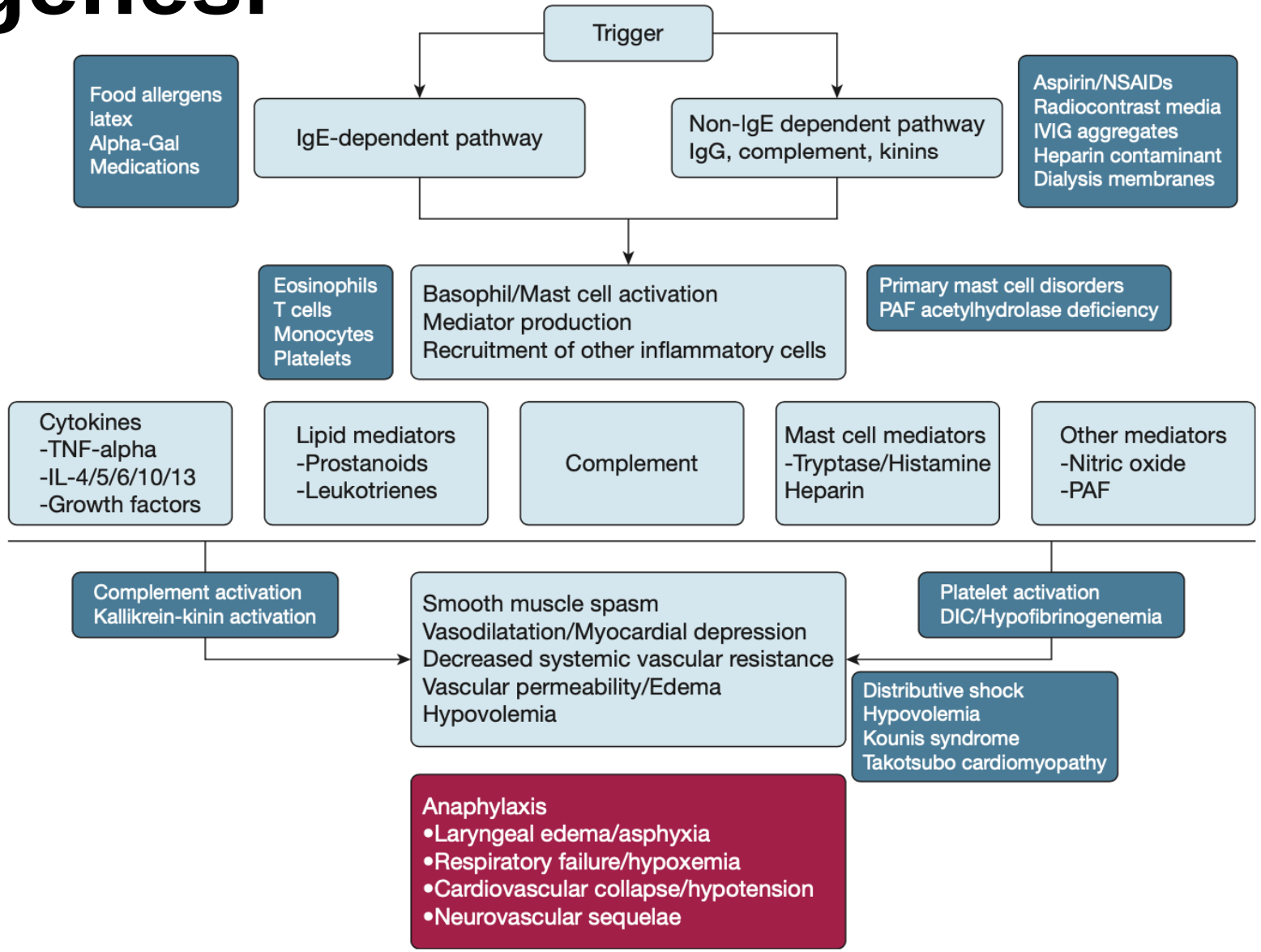


Figure 1 – *Mechanisms underlying anaphylaxis-IgE and non-IgE-dependent pathway. DIC = disseminated intravascular coagulation; IVIG = intravenous immunoglobulin; NSAIDs = nonsteroidal antiinflammatory drugs; PAF = platelet activating factor; TNF = tumor necrosis factor.*

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

Mechanism	Examples	Comments
Immunologic-IgE	Food allergens[a]	8 common food allergens listed below
(Secondary MCD)	Airborne allergens	Animal dander, aerosolized foods, pollen
	Latex	Gloves, catheters, masks, medication vials
	Hymenoptera venom	Honey bee, wasp, yellow jacket, hornet, IFA
	Medication allergy	Antibiotics, biologicals,[b] vaccines, NSAIDs[b]
	Alpha-gal	Mammalian meat (beef, pork, venison, lamb)
	FDEIA	Exercise + food (wheat, nuts, legumes, etc)
	Hormones	Progesterone or estrogens (catamenial)
	Seminal fluid	Postcoital anaphylaxis
	Radiocontrast media[b]	IgE-mediated reactions have been reported
Immunologic-non-IgE	Immune aggregates	Includes immune complexes/complement
	IVIG	From IgG or IgE anti-IgA antibodies
	Aspirin and NSAIDs[b]	Leukotriene-driven and other mechanisms
	Dialysis membranes	
	Radiocontrast media[b]	Complement activation, kinin generation
	Dextrans/HMW iron	
	Biologics[b]	Cytokine inhibitors, omalizumab, etc
	Heparin	Generation of kinins by contaminated
		Chinese heparin
Nonimmunologic		
Direct effects	Opiates, physical	Cold, heat, exercise, sunlight
Primary MCD[c]	MMAS and systemic	Genetic defects affect proliferation or activation of mast cells
MCAS[c]		Can be associated with germline replications of TPSAB1 gene encoding α-tryptase[36]
Idiopathic		Increased mast cell sensitivity/degranulation
		T helper cell 2-cytokine polarization
		Unrecognized allergens
Masqueraders	Munchausen stridor	
	Undifferentiated somatoform anaphylaxis	
	Vocal cord dysfunction	

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

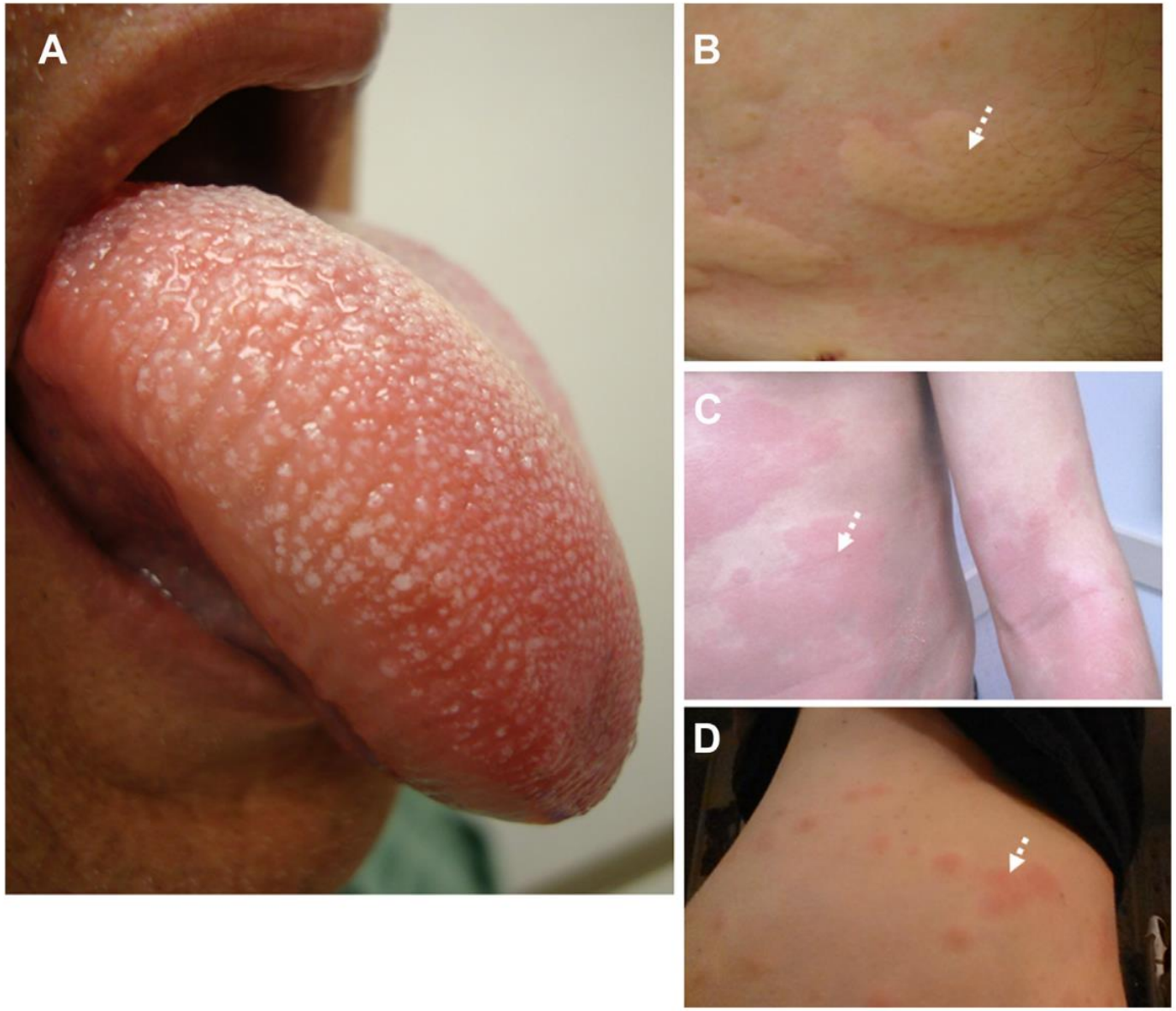
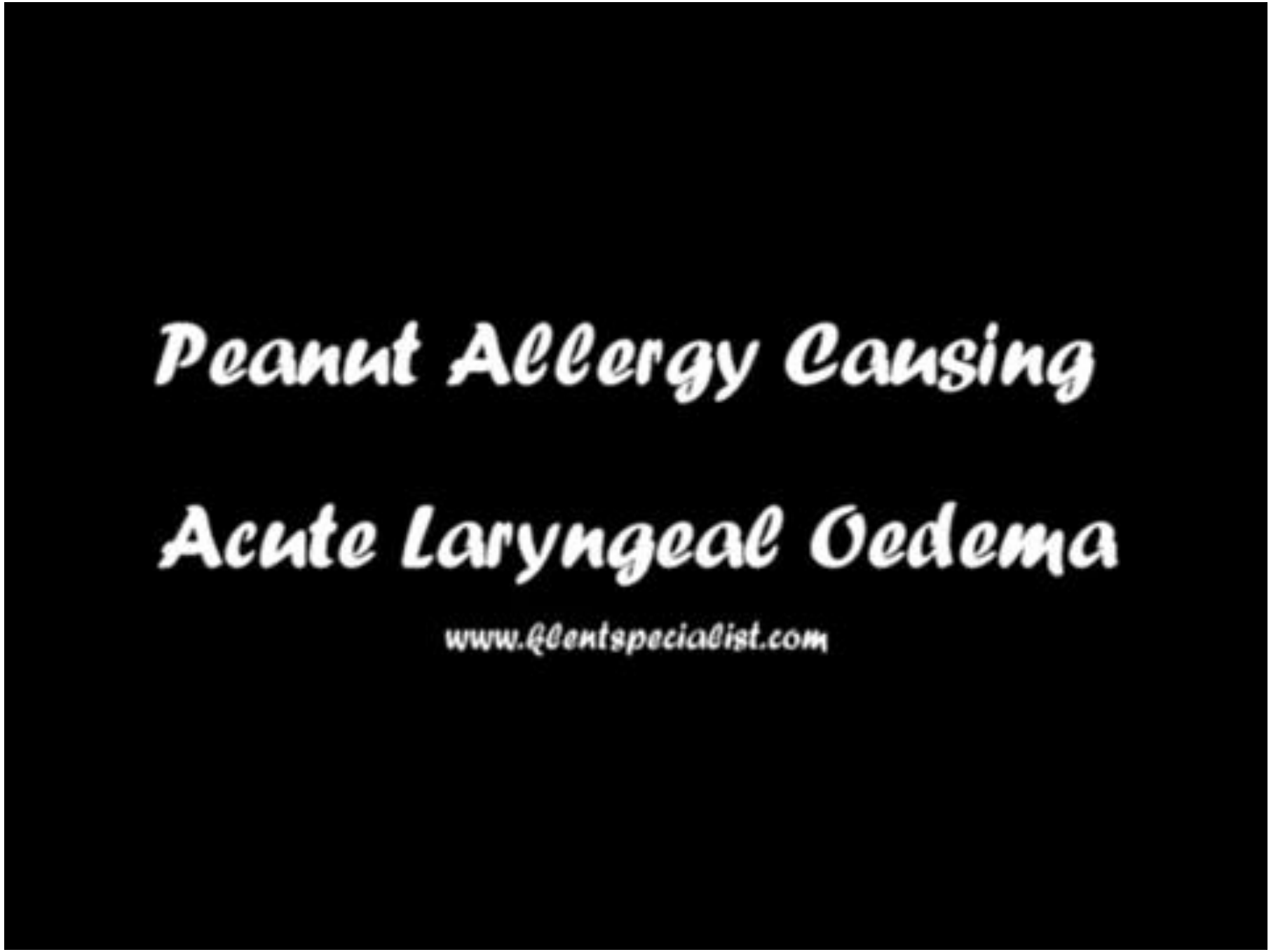


Figure 2 – *Clinical manifestations of anaphylaxis. A, Angioedema of the tongue and oropharynx. B, Discrete urticarial lesions in a patient with acute allergic reaction. C, Coalescent urticaria and diffuse erythema in a patient with a severe systemic allergic reaction. D, Delayed urticarial reactions in response to beef ingestion in an entomologist who had sustained multiple occupation-related tick bites months earlier. White dotted arrows indicate clinical findings.*

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033



https://www.youtube.com/watch?v=0zXt3W_xtUo

Diagnostic Testing: Tryptase and Histamine

Total serum tryptase is the biomarker most widely used to confirm a diagnosis of anaphylaxis retrospectively.[72] Small amounts of the immature form of tryptase (beta-protryptase) are constitutively secreted into the systemic circulation. Following mast cell and basophil degranulation, total serum tryptase levels increase significantly because of release of mature beta-tryptase. Ideally, serum tryptase should be measured within 1 to 2 h after symptom onset because tryptase levels typically peak within 60 to 90 min after symptom onset but can persist for 6 h.[73]

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

Anafilassi in anestesia

Figure 1. First clinical feature (%) in allergic anaphylaxis and all patients with Grade 3-5 perioperative anaphylaxis

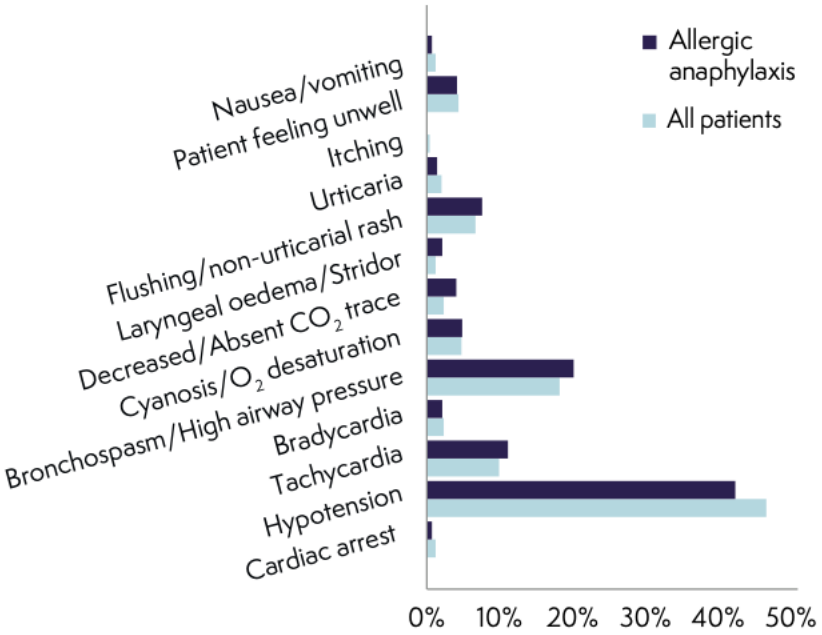
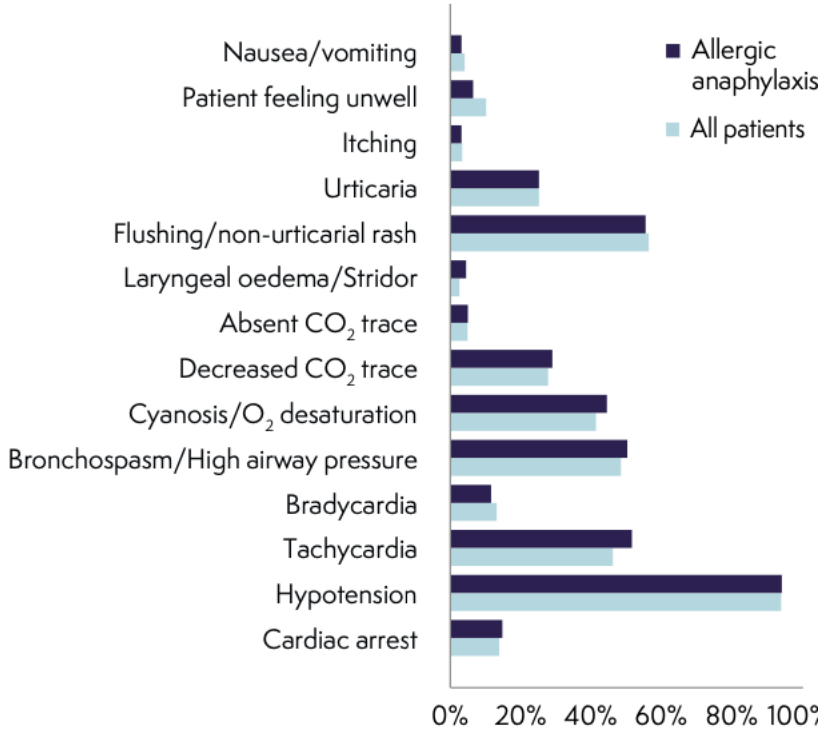


Figure 5. Clinical feature (%) present at any time during Grade 3-5 perioperative anaphylaxis: allergic anaphylaxis and all patients



Considering all cases, onset time was <5 min in 66.2%; <10 min in 82.7%; <15 min in 87.6% and <30 min in 94.7%. Onset times for individual agents are discussed below.

Table 1. The 199 identified culprit agents in 192 cases of anaphylaxis in NAP6

Agents by class	Definite	Probable	Total
Antibiotics	67	27	94
NMBAs	49	16	65
Chlorhexidine	14	4	18
Patent Blue	8	1	9
Others	10	3	13
All	148	51	199
Antibiotics			
Co-amoxiclav	38	8	46
Teicoplanin	21	15	36
Cefuroxime	2	2	4
Gentamicin	1	2	3
Flucloxacillin	2	0	2
Piperacilin & tazobactam	1	0	1
Vancomycin	1	0	1
Metronidizole	1	0	1
NMBAs			
Rocuronium	21	6	27
Atracurium	14	9	23
Suxamethonium	13	1	14
Mivacurium	1	0	1
Antiseptics and dyes			
Chlorhexidine	14	4	18
Patent Blue dye	8	1	9
Other agents			
Gelatin	3	0	3
Blood products	2	0	2
Ondansetron	1	1	2
Sugammadex	1	0	1
Ibuprofen	1	0	1
Propofol	1	0	1
Protamine	1	0	1
Aprotinin	0	1	1
Heparin	0	1	1

https://www.nationalauditprojects.org.uk/downloads/NAP6%20Report%202018.pdf

Anaphylaxis

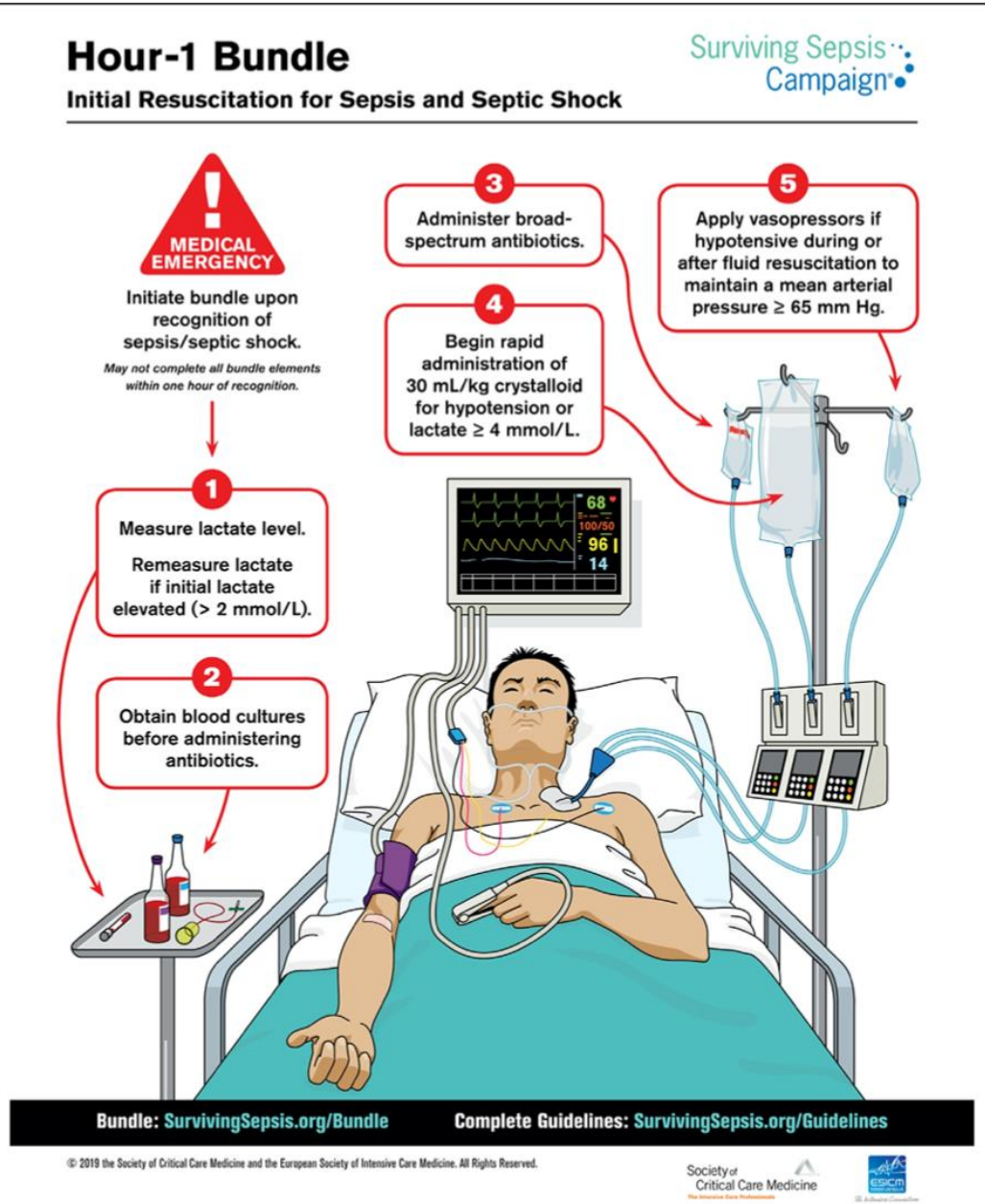
- Recognise anaphylaxis by the presence of airway (swelling), breathing (wheeze or persistent coughing), or circulation (hypotension) problems with or without skin and mucosal changes. This can be in the context of a known trigger in a patient with an allergy, or suspected anaphylaxis in a patient with no previous history of allergy.
- Call for help early.
- Remove or stop the trigger if feasible.
- Give intramuscular (IM) adrenaline (0.5 mg (which is 0.5 ml of a 1 mg in 1 ml ampoule of adrenaline)) into the anterolateral thigh as soon as anaphylaxis is suspected. Repeat the IM adrenaline if there is no improvement in the patient's condition after about 5 min.
- Ensure the patient is lying and do not suddenly sit or stand the patient up.
- Use an ABCDE approach and treat problems early (oxygen, fluids, monitoring).
- Give an IV crystalloid fluid bolus early and monitor the response – large volumes of fluids may be needed.
- Consider IV adrenaline as a bolus (20–50 mcg) or infusion for refractory anaphylaxis or in specialist care settings where the skills are available.
- Consider alternative vasopressors (vasopressin, noradrenaline, metaraminol, phenylephrine) in refractory anaphylaxis.
- Consider IV glucagon in patients taking beta-blockers.
- Start chest compressions and ALS as soon as cardiac arrest is suspected and follow standard guidelines.
- Consider ECLS or ECPR for patients who are peri-arrest or in cardiac arrest as a rescue therapy in those settings where it is feasible.

Lott C, Truhlář A, Alfonzo A, et al. European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances. Resuscitation. 2021;161:152-219. doi:10.1016/j.resuscitation.2021.02.011

Medication Effects	Concentration	Dose	Route	Frequency	Adverse Effects
Epinephrine	1:1,000 (1 mg/mL)	0.01 mg/kg 0.3 to 0.5 mg	IM	Every 5-15 min	Tachycardia, palpitations, tachyarrhythmia, anxiety, palpitations, flushing
	1:10,000 (0.1 mg/mL)	0.01 mg/kg 0.5-1.0 mg (5-10 mL)	IV	Every 5-15 min Push	As above
Vasopressin	NA	0.04 U	IV	Per minute	Ischemia
Dopamine	NA	1-50 mcg/kg	IV	Per minute	Tachycardia, tachyarrhythmia
Norepinephrine	NA	0.02-1 mcg/kg	IV	Per minute	Tachycardia, tachyarrhythmia
Albuterol					
MDI	2.5 mg per puff	1-2 puffs (2.5-5 mg)	INH	Every 2-4 h	Tachycardia, palpitations, anxiety
Nebulized	2.5 mg/3 mL	3 mL	INH	Every 2-4 h	As above
	5 mg/3 mL	3 mL	INH	Continuous	As above
Glucagon		3-10 mg[a] 0.05-0.1 mg/kg/h	IV IV	Once Continuous	Nausea, vomiting, tachycardia
Diphenhydramine					
Treatment	NA	25-50 mg	IV, PO[b]	Once	Drowsiness, sedation
Prophylaxis	NA	25-50 mg		Once[c]	
Corticosteroids					
Hydrocortisone	NA	100 mg	IV	Every 8 h	Hyperglycemia
Prednisone[d]					
Treatment	NA	1-2 mg/kg	PO[b]	Once	Agitation, anxiety, psychosis
Prophylaxis	NA	50 mg	PO[b]	13 h, then 7 h, then 1 h before	As above

LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. Chest. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

Shock settico



Lott C, Truhlář A, Alfonzo A, et al. European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances. Resuscitation. 2021;161:152-219. doi:10.1016/j.resuscitation.2021.02.011

Domande o interventi?