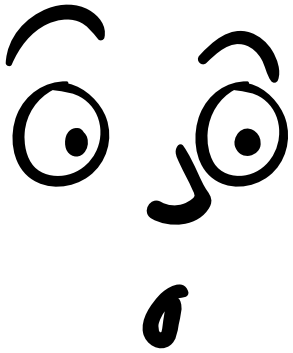
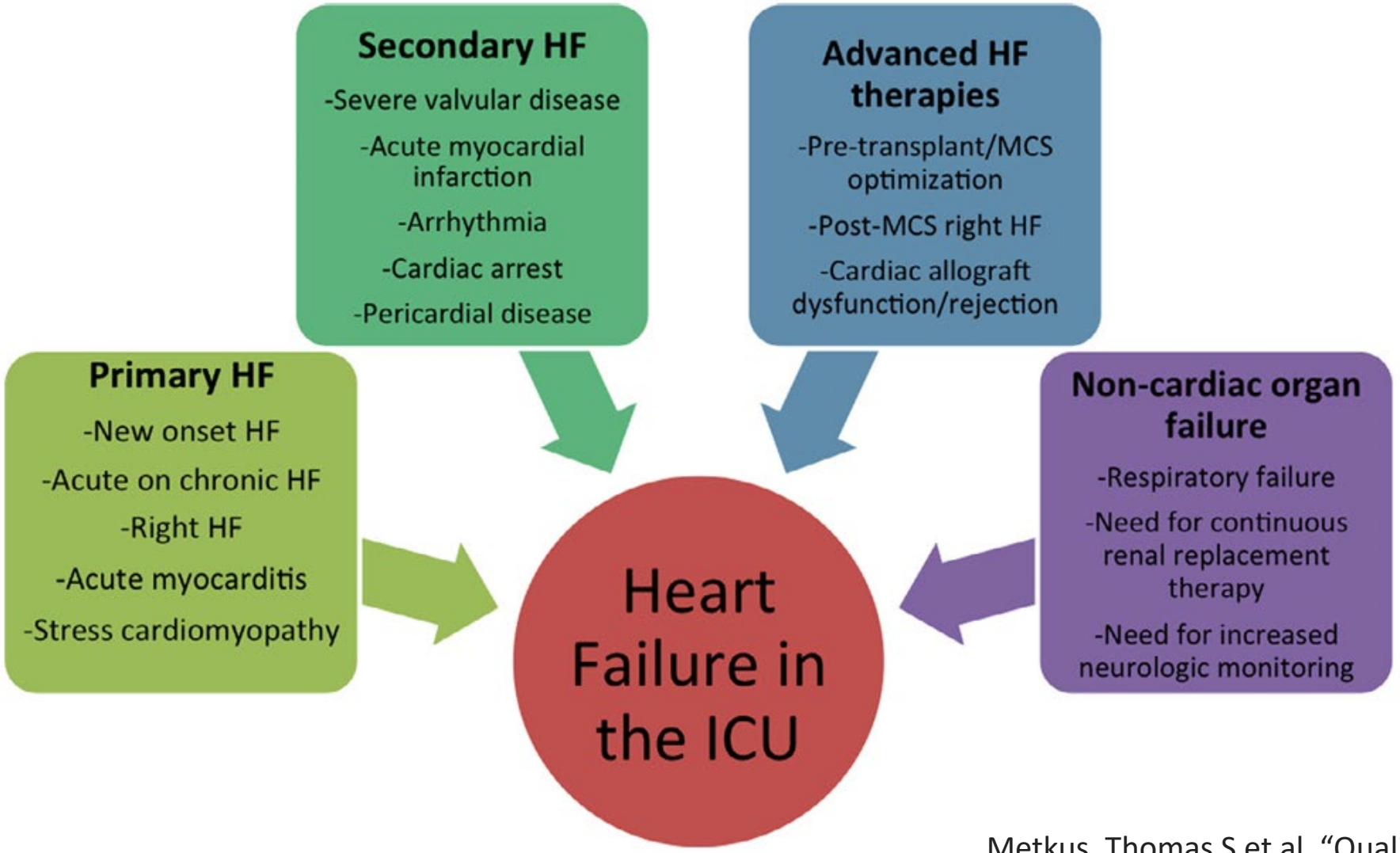
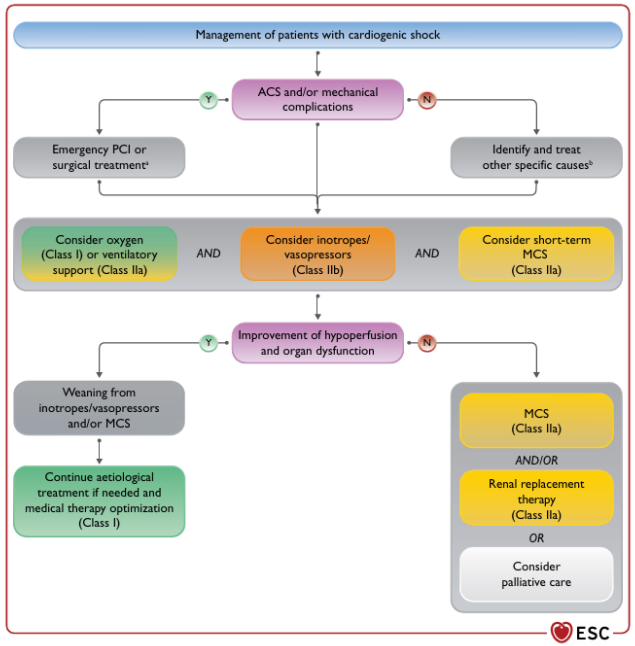


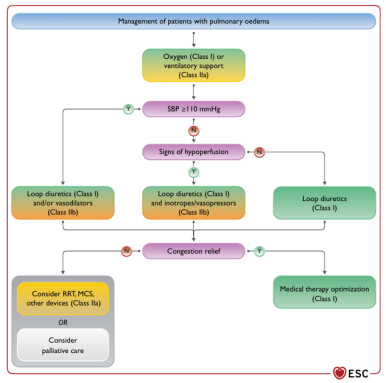


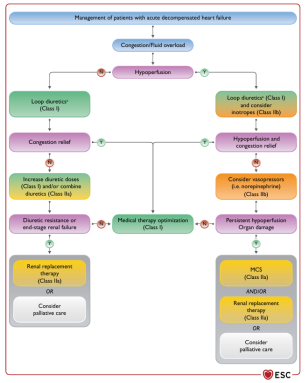
Metkus, Thomas S et al. “Quality of Heart Failure Care in the Intensive Care Unit.” *Journal of cardiac failure* vol. 27,10 (2021): 1111-1125. doi:10.1016/j.cardfail.2021.08.001

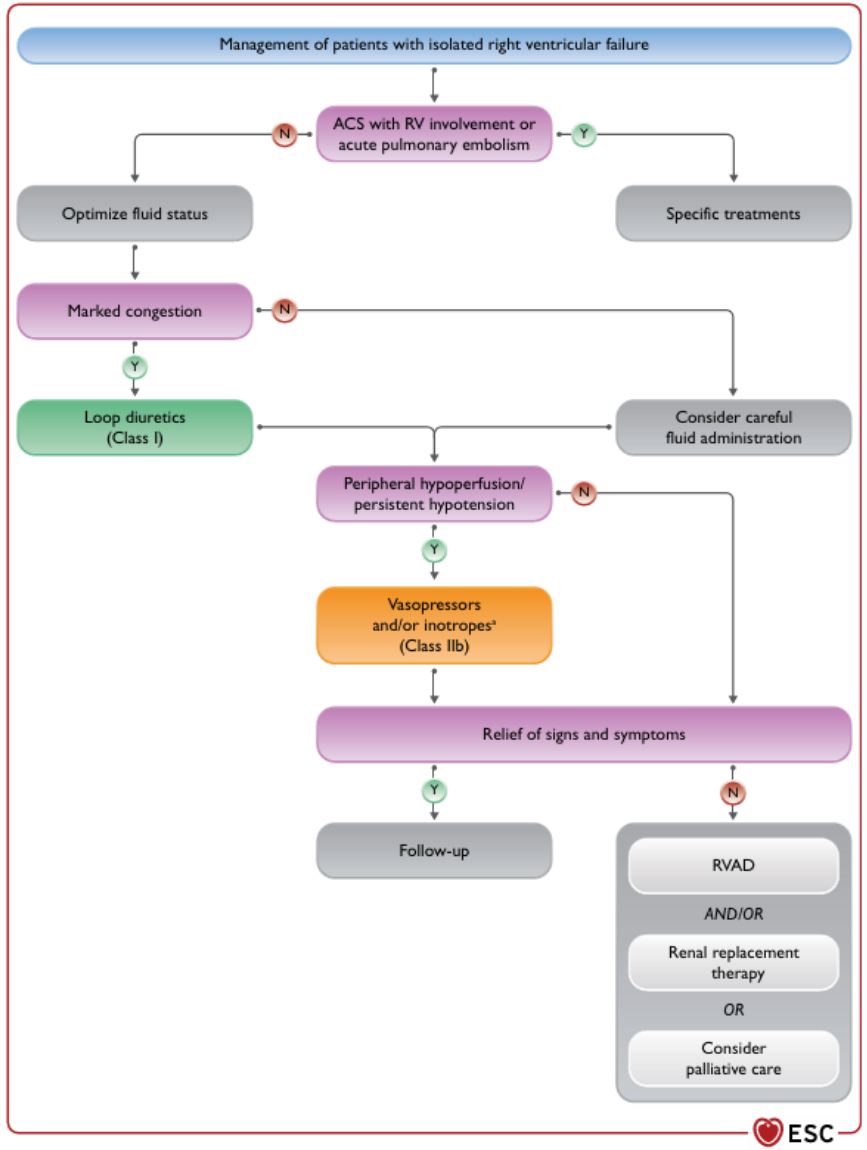
Table 21 Clinical presentations of acute heart failure

	Acute decompensated heart failure	Acute pulmonary oedema	Isolated right ventricular failure	Cardiogenic shock
Main mechanisms	LV dysfunction Sodium and water renal retention	Increased afterload and/or predominant LV diastolic dysfunction Valvular heart disease	RV dysfunction and/or pre-capillary pulmonary hypertension	Severe cardiac dysfunction
Main cause of symptoms	Fluid accumulation, increased intraventricular pressure	Fluid redistribution to the lungs and acute respiratory failure	Increased central venous pressure and often systemic hypoperfusion	Systemic hypoperfusion
Onset	Gradual (days)	Rapid (hours)	Gradual or rapid	Gradual or rapid
Main haemodynamic abnormalities	Increased LVEDP and PCWP[a] Low or normal cardiac output Normal to low SBP	Increased LVEDP and PCWP[a] Normal cardiac output Normal to high SBP	Increased RVEDP Low cardiac output Low SBP	Increased LVEDP and PCWP[a] Low cardiac output Low SBP
Main clinical presentations[1,446]	Wet and warm OR Dry and cold	Wet and warm[b]	Dry and cold OR Wet and cold	Wet and cold
Main treatment	Diuretics Inotropic agents/vasopressors (if peripheral hypoperfusion/hypotension) Short-term MCS or RRT if needed	Diuretics Vasodilators[b]	Diuretics for peripheral congestion Inotropic agents/vasopressors (if peripheral hypoperfusion/hypotension) Short-term MCS or RRT if needed	Inotropic agents/vasopressors Short-term MCS RRT


Management of patients with cardiogenic shock
ACS and/or mechanical complications
Y
N
Emergency PCI or surgical treatment[a]
Identify and treat other specific causes[b]
Consider oxygen (Class I) or ventilatory support (Class IIa)
AND
Consider inotropes/ vasopressors (Class IIb)
AND
Consider short-term MCS (Class IIa)
Improvement of hypoperfusion and organ dysfunction
Weaning from inotropes/vasopressors and/or MCS
Continue aetiological treatment if needed and medical therapy optimization (Class I)
MCS (Class IIa)
AND/OR
Renal replacement therapy (Class IIa)
OR
Consider palliative care
ESC

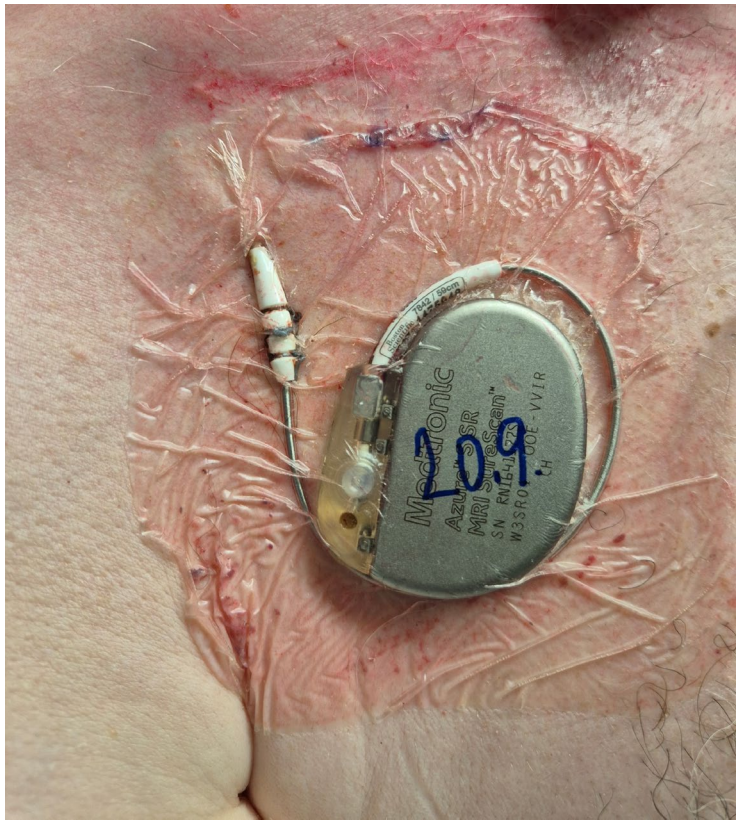

Management of patients with pulmonary oedema
Oxygen (Class I) or ventilatory support (Class IIa)
SBP ≥110 mmHg
Signs of hypoperfusion
Loop diuretics (Class I) and/or vasodilators (Class IIb)
Loop diuretics (Class I) and inotropes/vasopressors (Class IIb)
Loop diuretics (Class I)
Congestion relief
Consider RRT, MCS, other devices (Class IIa)
OR
Consider palliative care
Medical therapy optimization (Class I)
ESC


Management of patients with acute decompensated heart failure
Congestion/Fluid overload
Hypoperfusion
Loop diuretics[a] (Class I)
Loop diuretics[a] (Class I) and consider inotropes (Class IIb)
Congestion relief
Hypoperfusion and congestion relief
Increase diuretic doses (Class I) and/or combine diuretics (Class IIa)
Consider vasopressors (i.e. norepinephrine) (Class IIb)
Diuretic resistance or end-stage renal failure
Medical therapy optimization (Class I)
Persistent hypoperfusion Organ damage
Renal replacement therapy (Class IIa)
OR
Consider palliative care
MCS (Class IIa)
AND/OR
Renal replacement therapy (Class IIa)
OR
Consider palliative care
ESC


Management of patients with isolated right ventricular failure
ACS with RV involvement or acute pulmonary embolism
N
Y
Optimize fluid status
Specific treatments
Marked congestion
N
Y
Loop diuretics (Class I)
Consider careful fluid administration
Peripheral hypoperfusion/ persistent hypotension
N
Y
Vasopressors and/or inotropes[a] (Class IIb)
Relief of signs and symptoms
Y
N
Follow-up
RVAD
AND/OR
Renal replacement therapy
OR
Consider palliative care
ESC

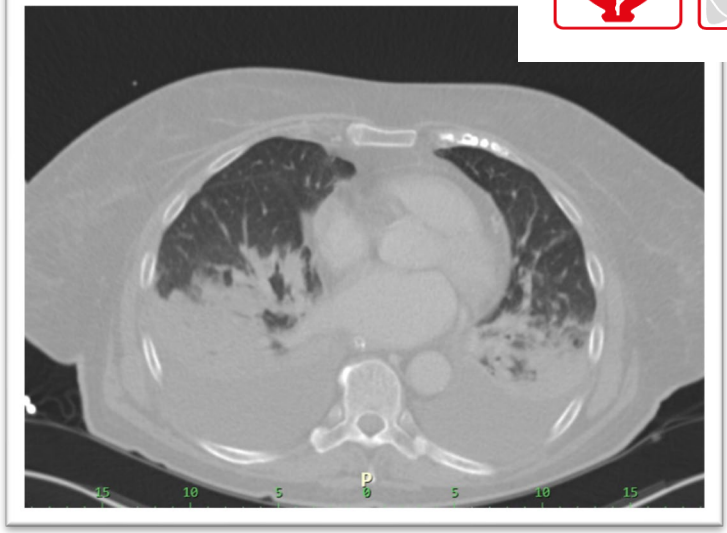
1. akutní dekompenzace chronického srdečního selhání
2. akutní koronární syndrom
   - akutní infarkt myokardu
   - nestabilní angina pectoris
   - mechanická komplikace akutního infarktu myokardu
   - infarkt pravé komory
3. hypertenzní krize
4. akutní arytmie
   - komorová tachykardie
   - komorová fibrilace
   - fibrilace a flutter síní
   - jiné

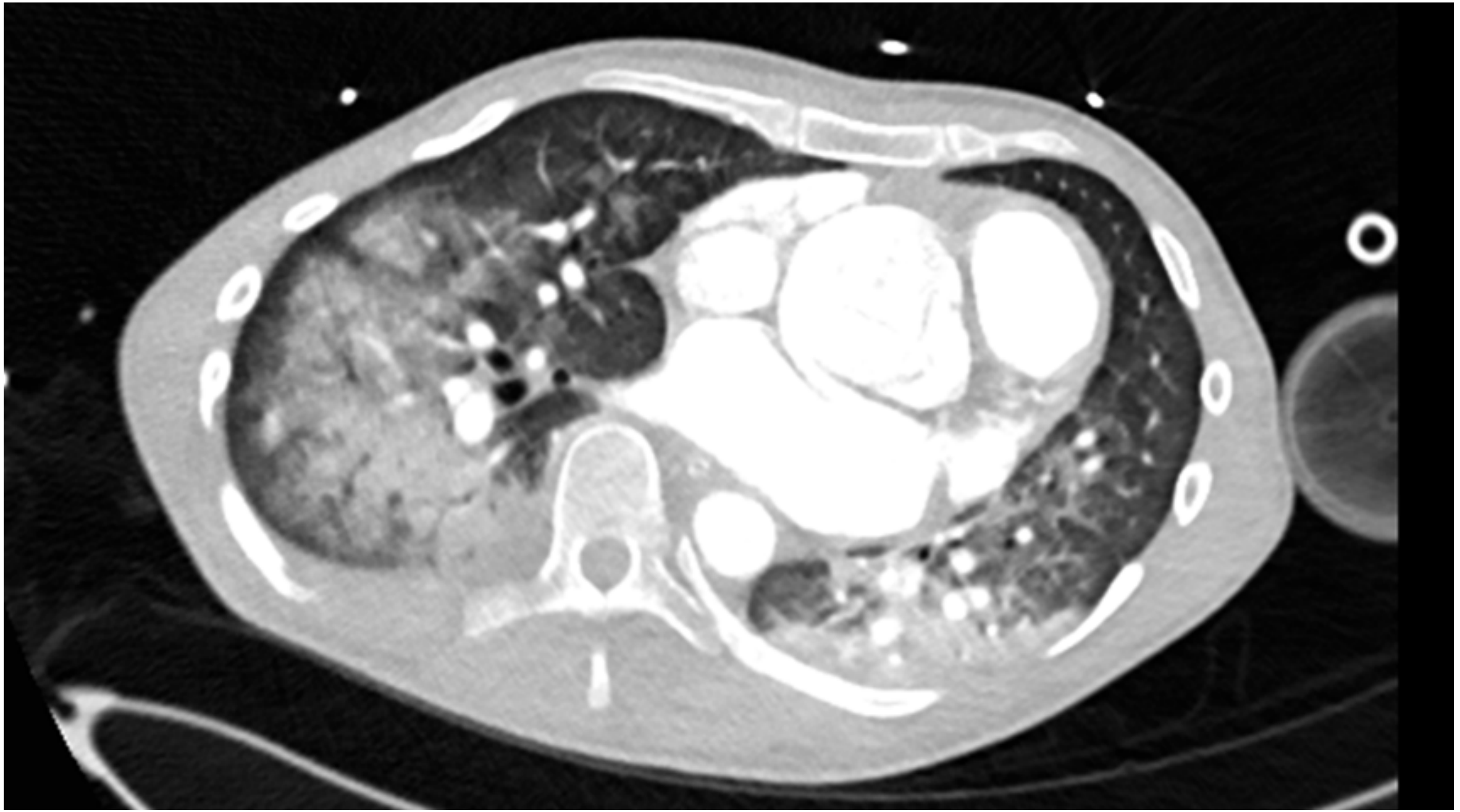
O jaké pacienty se nestaráme?

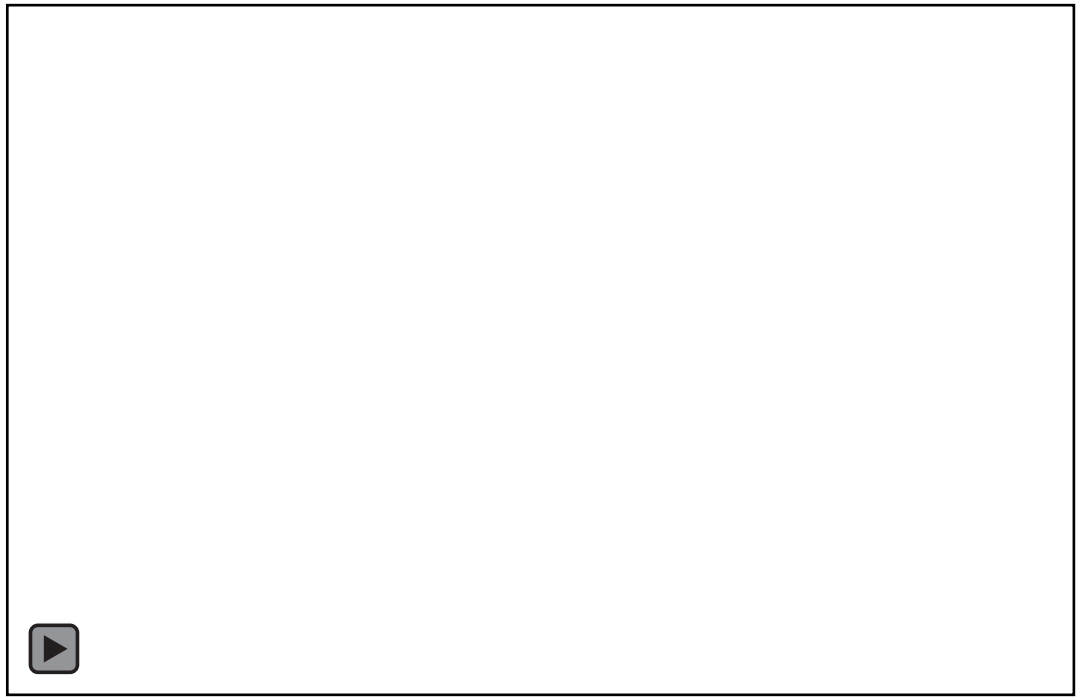

Medtronic
Azure S SR
MRI SureScan™
SN RMH
W3SR01
DDE - VVIR
109

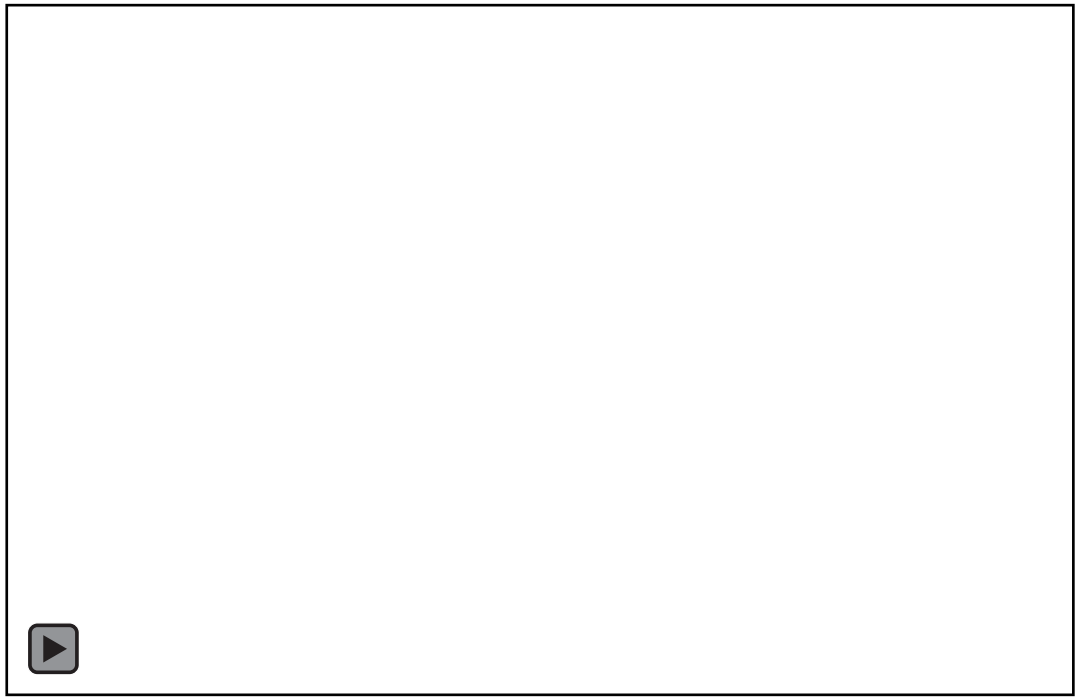
5. akutní chlopenní regurgitace
   - endokarditida
   - ruptura chordae tendineae
   - zhoršení známé regurgitace
   - jiné
6. hemodynamicky významná aortální či mitrální stenóza
7. akutní myokarditida
8. srdeční tamponáda
9. disekce aorty

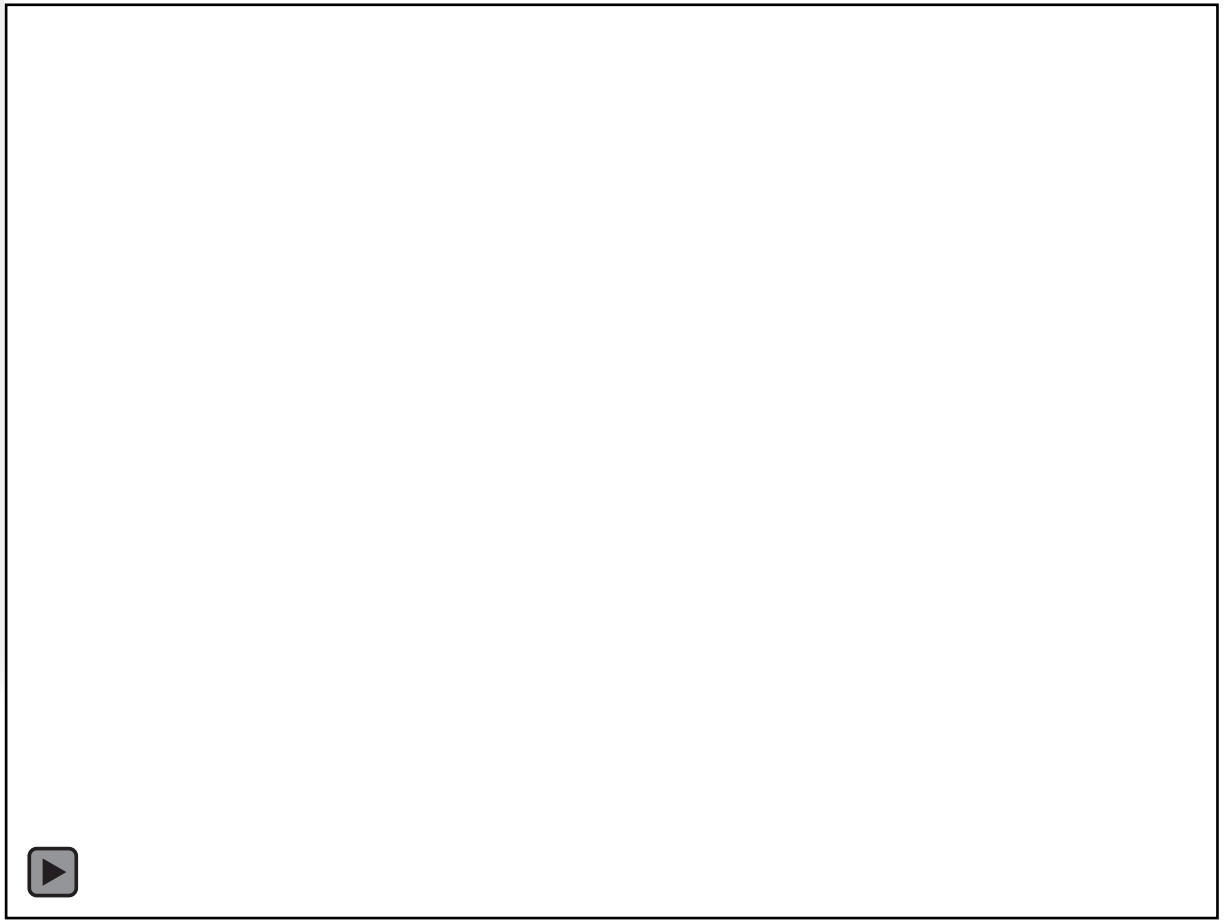
Jaké pacienty vidíme vzácně?


15
10
5
P
0
5
10
15

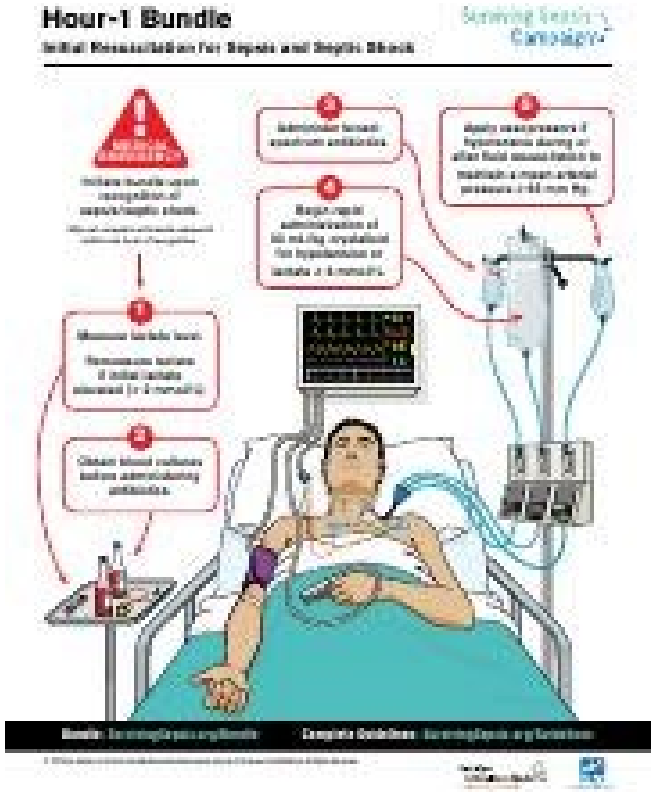




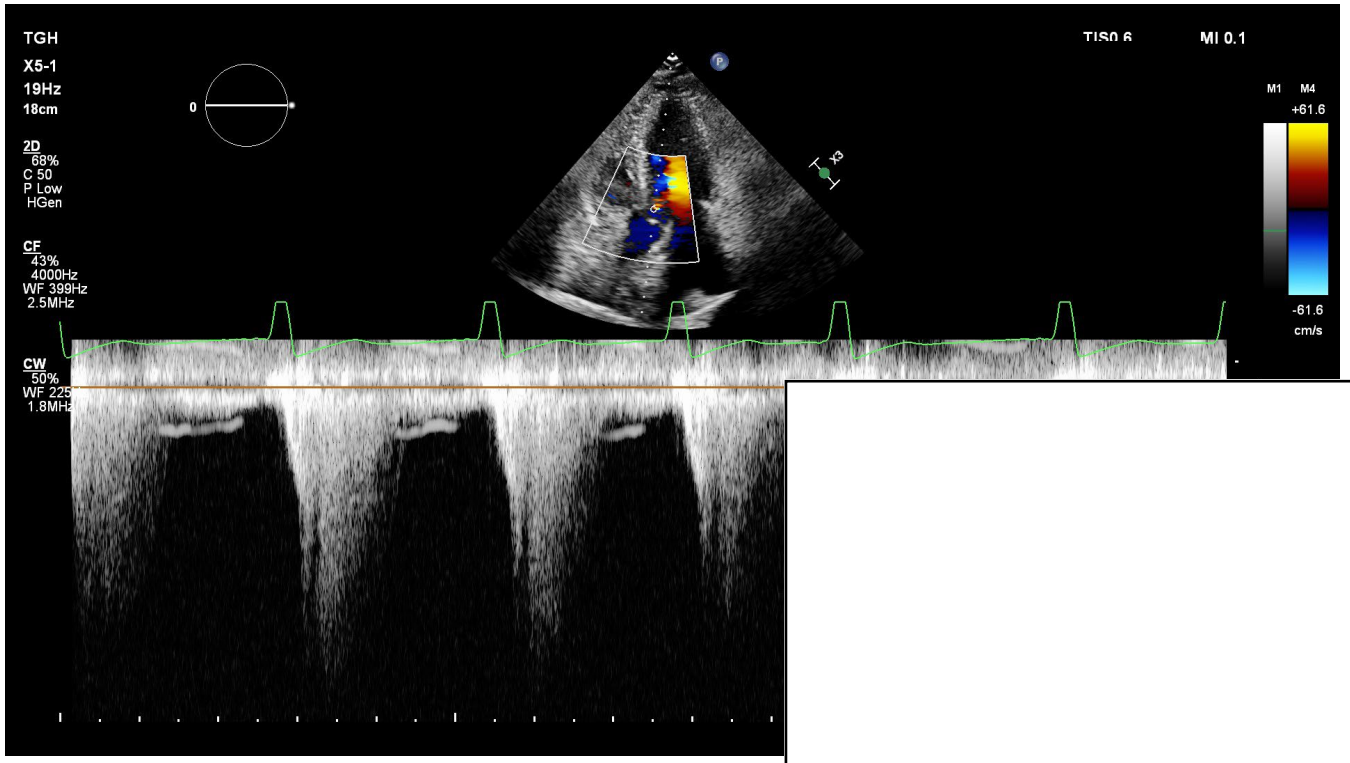


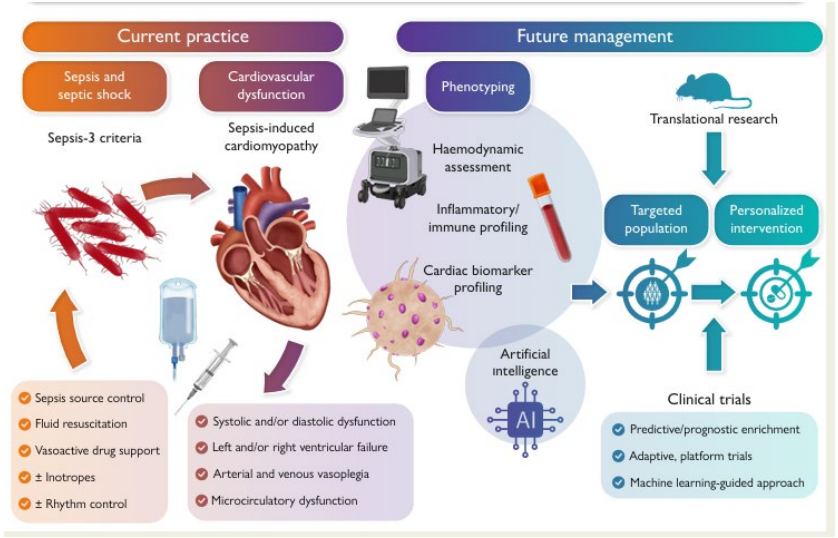


11. nekardiální příčiny a zhoršující faktory
- špatná spolupráce nemocného
- objemové přetížení
- infekce – sepse, pneumonie ...
- těžké poškození mozku
- velký chirurgický zákrok
- akutní selhání ledvin, zhoršení chronického selhání ledvin
- asthma bronchiale
- intoxikace léky
- intoxikace alkoholem
- feochromocytom



O jaké pacienty se staráme denně?


TGH
X5-1
19Hz
18cm
2D
68%
C 50
P Low
HGen
CF
43%
4000Hz
WF 399Hz
2.5MHz
CW
50%
WF 225
1.8MHz
TIS0.6
MI 0.1
M1
M4
+61.6
-61.6
cm/s



ESC European Society of Cardiology

European Heart Journal (2025) **46**, 3339–3353
https://doi.org/10.1093/eurheartj/ehaf340

STATE OF THE ART REVIEW
Acute cardiovascular care

Sepsis-induced cardiomyopathy

Nadia Aissaoui[1]*, Florence Boissier[2], Michelle Chew[3], Mervyn Singer [4], and Philippe Vignon[5]

Profound but Reversible Myocardial Depression in Patients with Septic Shock

MARGARET M. PARKER, M.D.; JAMES H. SHELHAMER, M.D.; STEPHEN L. BACHARACH, Ph.D.; MICHAEL V. GREEN, M.S.; CHARLES NATANSON, M.D.; TERRI M. FREDERICK, B.S.N.; BARBARA A. DAMSKE, R.N.; and JOSEPH E. PARRILLO, M.D.; Bethesda, Maryland

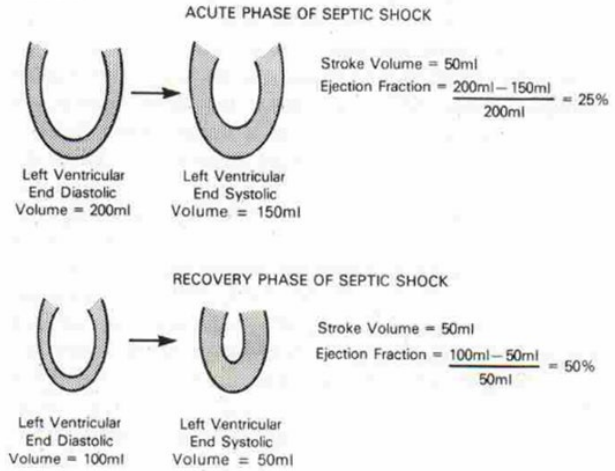


Figure 6. A schematic representation of the reversible myocardial depression seen in the survivors of septic shock.

Pathophysiology

- a complex interaction…
- direct myocardial injury
- loss of vascular tone
- decreased catecholamine responsiveness
- Ca^{2+} handling abnormalities
- mitochondrial dysfunction and myocardial hibernation

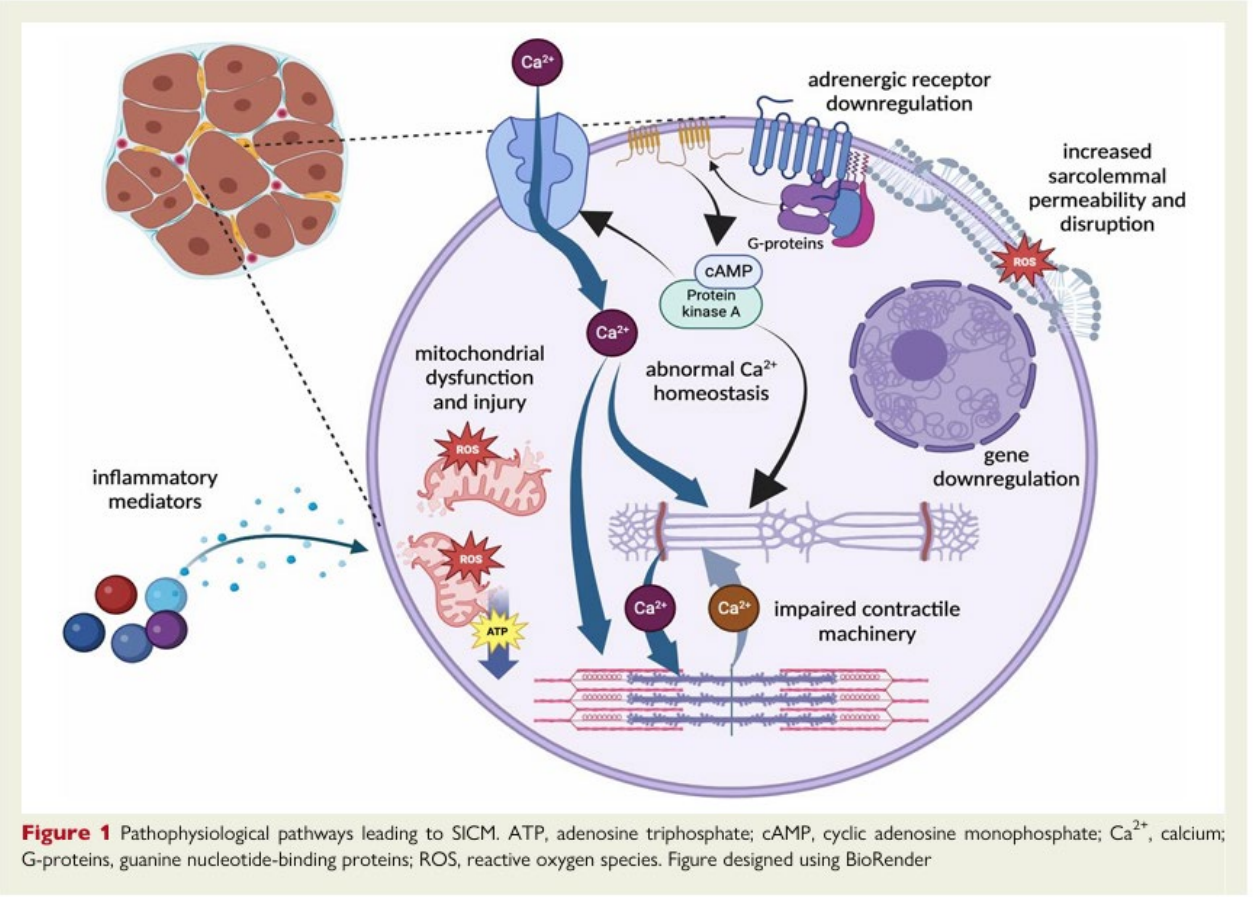
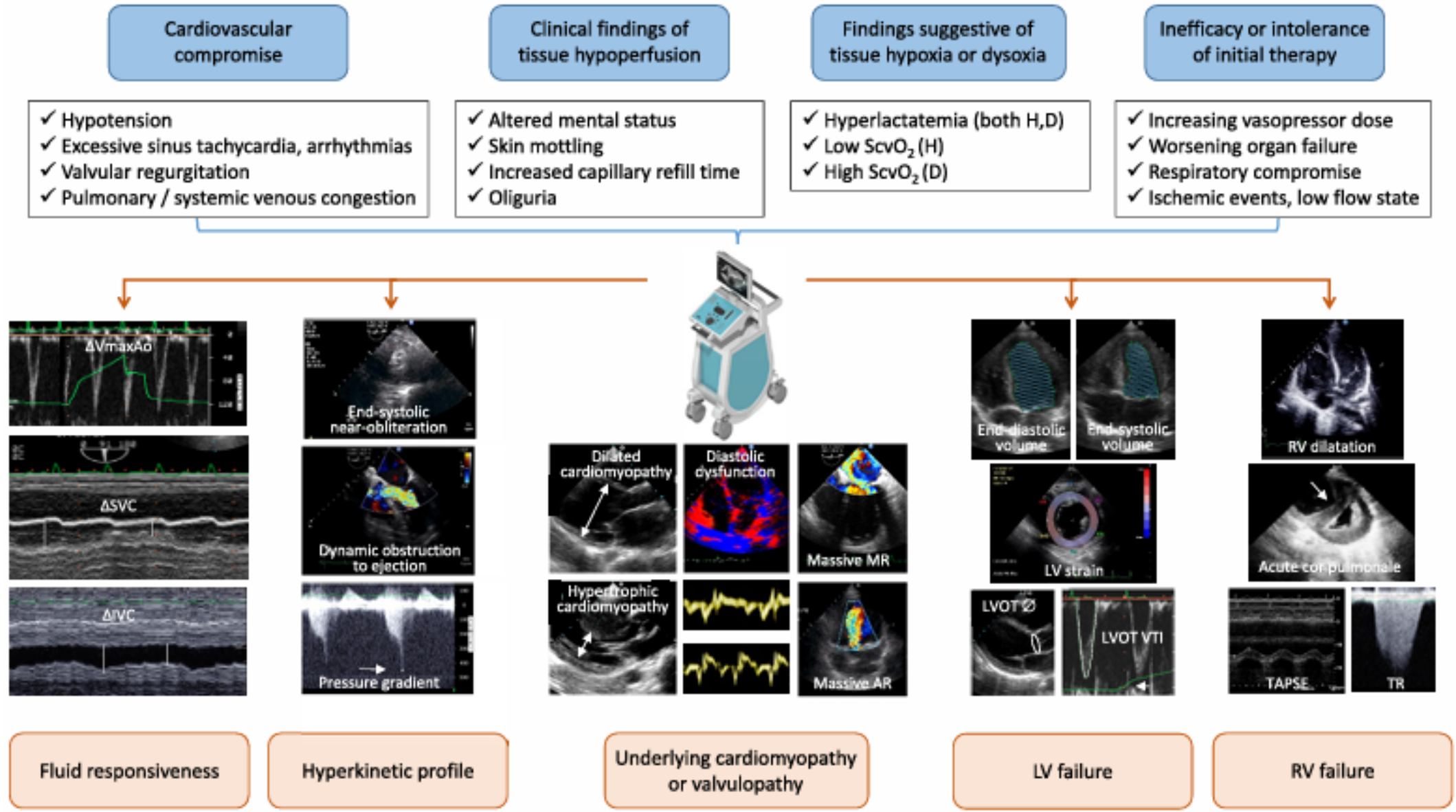
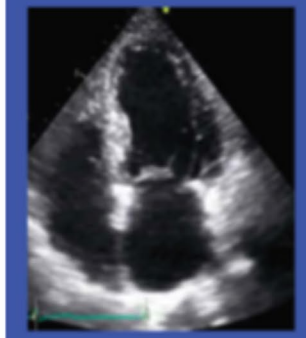
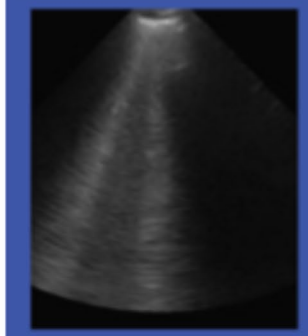
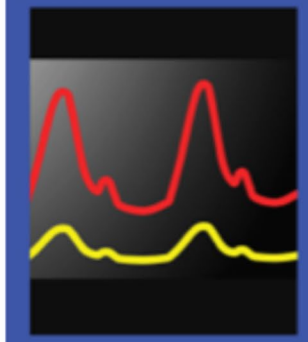


Figure 1 Pathophysiological pathways leading to SICM. ATP, adenosine triphosphate; cAMP, cyclic adenosine monophosphate; Ca^{2+}, calcium; G-proteins, guanine nucleotide-binding proteins; ROS, reactive oxygen species. Figure designed using BioRender

Cardiomyocyte cell death ≠ main feature in human sepsis.


Cardiovascular compromise
Clinical findings of tissue hypoperfusion
Findings suggestive of tissue hypoxia or dysoxia
Inefficacy or intolerance of initial therapy
✓ Hypotension
✓ Excessive sinus tachycardia, arrhythmias
✓ Valvular regurgitation
✓ Pulmonary / systemic venous congestion
✓ Altered mental status
✓ Skin mottling
✓ Increased capillary refill time
✓ Oliguria
✓ Hyperlactatemia (both H,D)
✓ Low ScvO2 (H)
✓ High ScvO2 (D)
✓ Increasing vasopressor dose
✓ Worsening organ failure
✓ Respiratory compromise
✓ Ischemic events, low flow state
ΔVmaxAo
ΔSVC
ΔIVC
End-systolic near-obliteration
Dynamic obstruction to ejection
Pressure gradient
Dilated cardiomyopathy
Diastolic dysfunction
Massive MR
Hypertrophic cardiomyopathy
Massive AR
End-diastolic volume
End-systolic volume
LV strain
LVOT Ø
LVOT VTI
RV dilatation
Acute cor pulmonale
TAPSE
TR
Fluid responsiveness
Hyperkinetic profile
Underlying cardiomyopathy or valvulopathy
LV failure
RV failure

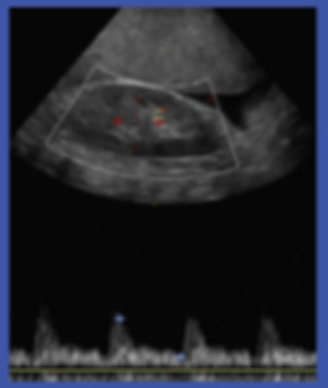
	CCE is first-line imaging modality for diagnosis of CS; Preferred modality to assess for complications (i.e. valve dysfunction); Unique for monitoring response to therapy; Essential for escalation to MCS, Cath-lab or cardiac surgery; Estimation of LVFP; Assessment of interventricular and heart-lung interaction
	Monitoring is not continuous; Filling pressures are estimated; Advanced CCE is not always available; Education in CCE is currently a challenge
	LUS permits the rapid identification and quantification of pulmonary edema; Monitoring response to therapy; Complements the CCE in assessing LVFP
	It is not diagnostic for the etiology of CS; does not address the genesis of pulmonary edema
	*Pulse contour analysis:* Allows continuous blood pressure monitoring; can track CO changes in response to interventions; may be used to test FR
	PAC: Gold standard for measuring CO; allows direct measurement of PAP and LVFP; differentiates pre- and post-capillary pulmonary hypertension
	Both: not diagnostic of CS etiology of shock; Not feasible as first-line approach; Variations in use among centers *PAC:* requires greater knowledge and practice; proficiency and utilization have decreased over the years.; intermittent CO monitoring (most cases), unable to assess FR

Intensive Care Med (2025) 51:1687–1691
https://doi.org/10.1007/s00134-025-08048-z

WHAT'S NEW IN INTENSIVE CARE

How we use critical care ultrasonography in the management of cardiogenic shock: a strategic game of chess in intensive care

Filippo Sanfilippo[1*], Siddharth Dugar[2,3] and Michelle S. Chew[4,5]

	Venous congestion allows to identify congestive phenotypes; Quantification of maladaptive consequences of congestion; Monitoring of decongestion in case of off-loading strategy
	Validation studies still lacking; Not established role in general intensive care; Low inter-rater reproducibility; Difficult to master

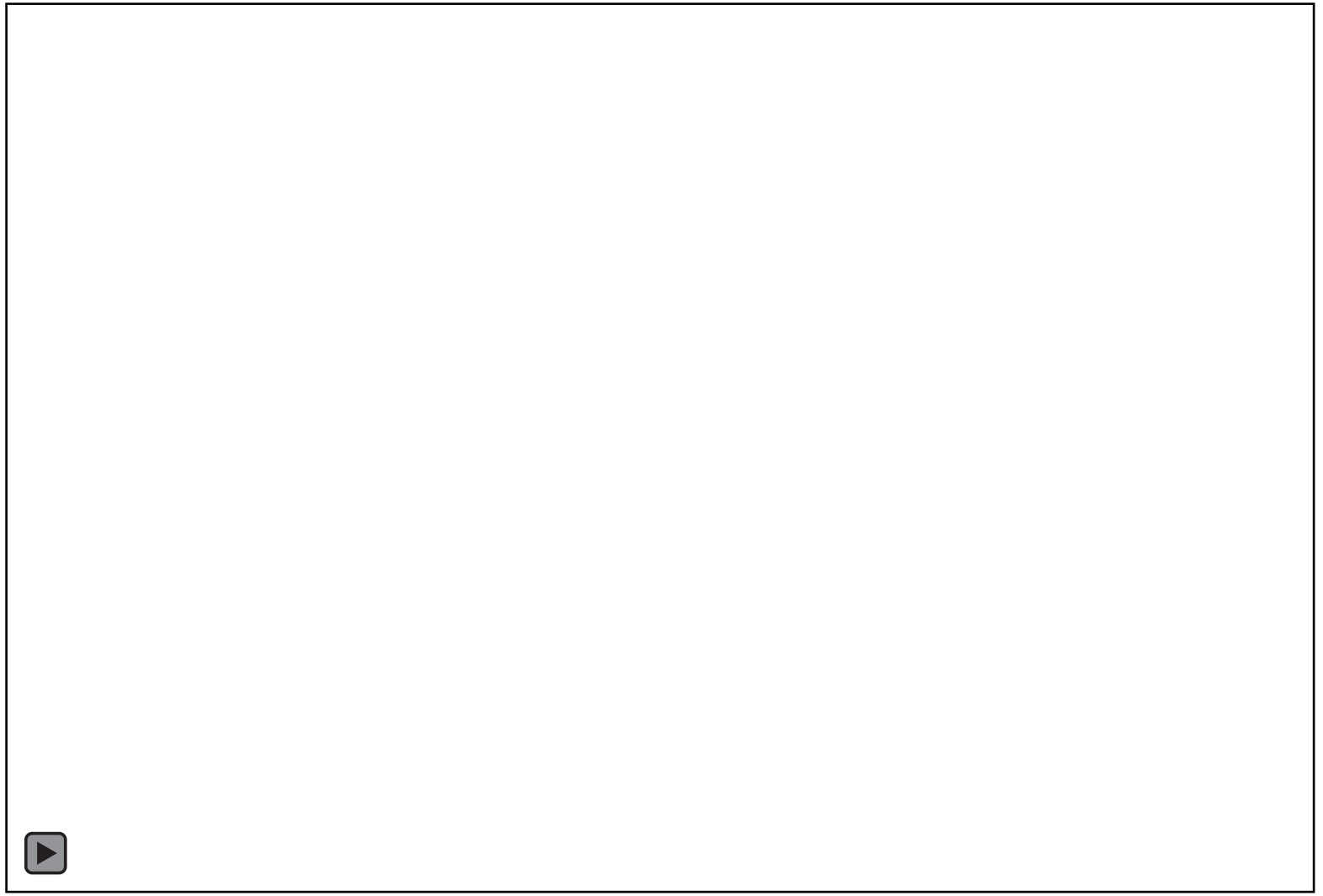


Table 2 | Triggering events in Takotsubo syndrome

Trigger	Examples	Notes
Emotional	Sudden death of a spouse, child or close friend Witnessing a serious accident or disaster Major life changes (such as divorce or job loss) Public speaking anxiety Social conflict	The most common type of trigger is an intense negative emotion; often associated with feelings of sadness, anger, fear, helplessness
Physical	Acute respiratory illness (for example, severe pneumonia and asthma) Sepsis Stroke or transient ischaemic attack Major surgery Hypoglycaemia Head trauma Myocardial infarction	The trigger can be severe illness, injury or sudden physiological stress Can be related to the release of stress hormones or changes in blood flow
Medical	Certain medications (β-blockers, dobutamine, inotropes) Toxins (carbon monoxide poisoning, cocaine, amphetamines) Intracranial haemorrhage Pheochromocytoma (adrenal tumour)	Less common than other categories but can trigger strong stress responses Medications often have direct effects on heart function or blood pressure
Others	Intense physical exertion (such as marathon running or weightlifting) Extreme environmental events (earthquakes, hurricanes) Emotional 'happy heart' events (for example, winning the lottery or unexpected positive life changes)	Less well-understood triggers might contribute to the stress response or microvascular dysfunction
No identified trigger	Up to 20–30% of patients with Takotsubo syndrome have no identifiable trigger	Takotsubo syndrome can occur even in the absence of a clear stressful event Might be related to individual susceptibility factors or undiagnosed medical conditions

nature reviews cardiology

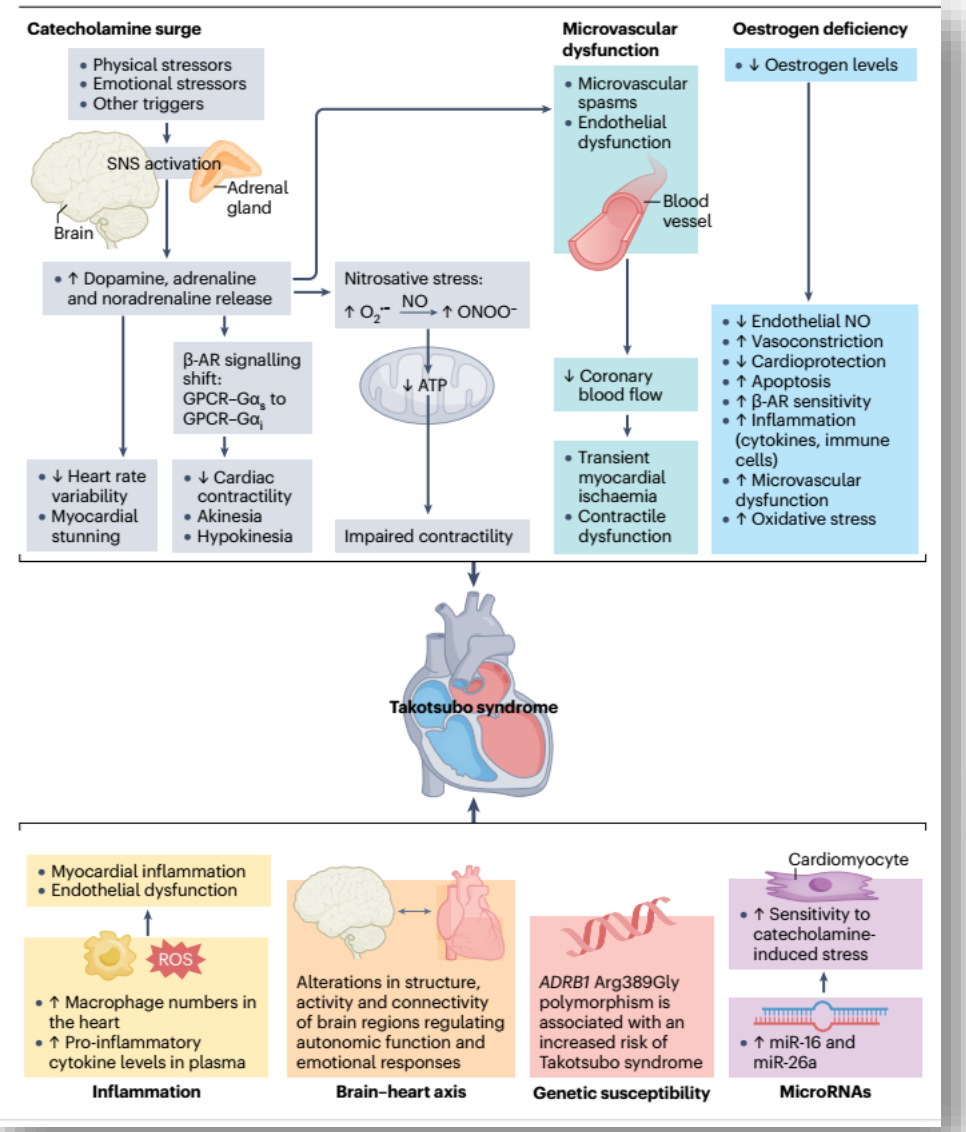
https://doi.org/10.1(

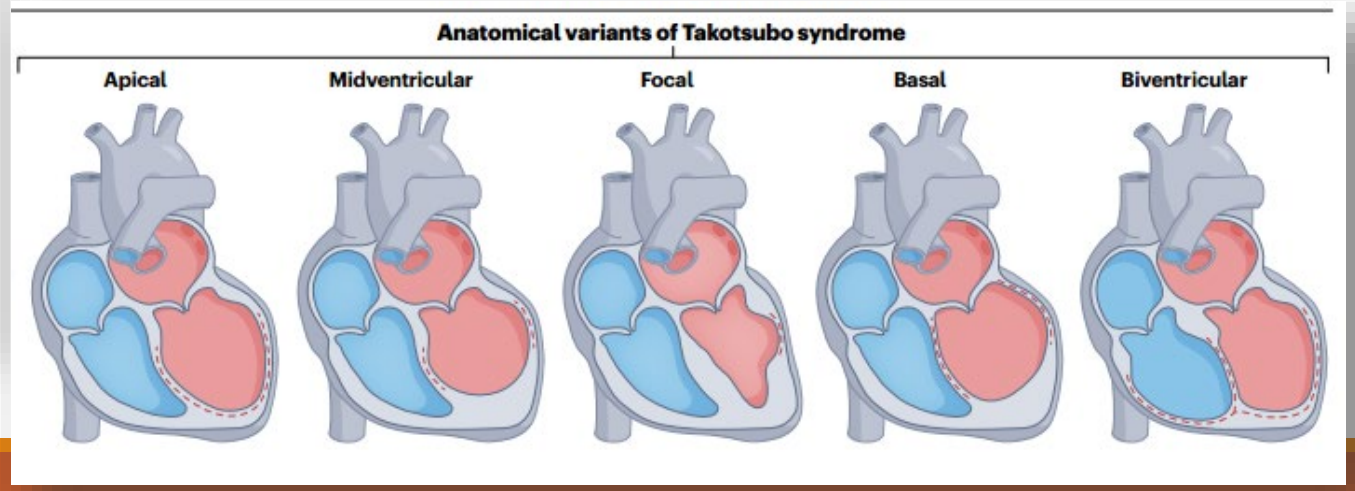
Review article

Check for updates

Takotsubo syndrome: pathophysiological insights and innovations in patient care

Elmir Omerovic[1,2] & Björn Redfors[1,2]


Catecholamine surge
• Physical stressors
• Emotional stressors
• Other triggers
SNS activation
Adrenal gland
Brain
• ↑ Dopamine, adrenaline and noradrenaline release
β-AR signalling shift: GPCR–Gαs to GPCR–Gαi
• ↓ Heart rate variability
• Myocardial stunning
• ↓ Cardiac contractility
• Akinesia
• Hypokinesia
Nitrosative stress:
↑ O2•- NO ↑ ONOO-
↓ ATP
Impaired contractility
Microvascular dysfunction
• Microvascular spasms
• Endothelial dysfunction
Blood vessel
↓ Coronary blood flow
• Transient myocardial ischaemia
• Contractile dysfunction
Oestrogen deficiency
• ↓ Oestrogen levels
• ↓ Endothelial NO
• ↑ Vasoconstriction
• ↓ Cardioprotection
• ↑ Apoptosis
• ↑ β-AR sensitivity
• ↑ Inflammation (cytokines, immune cells)
• ↑ Microvascular dysfunction
• ↑ Oxidative stress
Takotsubo syndrome
• Myocardial inflammation
• Endothelial dysfunction
ROS
• ↑ Macrophage numbers in the heart
• ↑ Pro-inflammatory cytokine levels in plasma
Inflammation
Alterations in structure, activity and connectivity of brain regions regulating autonomic function and emotional responses
Brain–heart axis
ADRB1 Arg389Gly polymorphism is associated with an increased risk of Takotsubo syndrome
Genetic susceptibility
Cardiomyocyte
• ↑ Sensitivity to catecholamine-induced stress
• ↑ miR-16 and miR-26a
MicroRNAs


Anatomical variants of Takotsubo syndrome
Apical
Midventricular
Focal
Basal
Biventricular

Congestive Heart Failure

Prevalence and characteristics of left ventricular outflow tract obstruction in Tako-Tsubo syndrome d

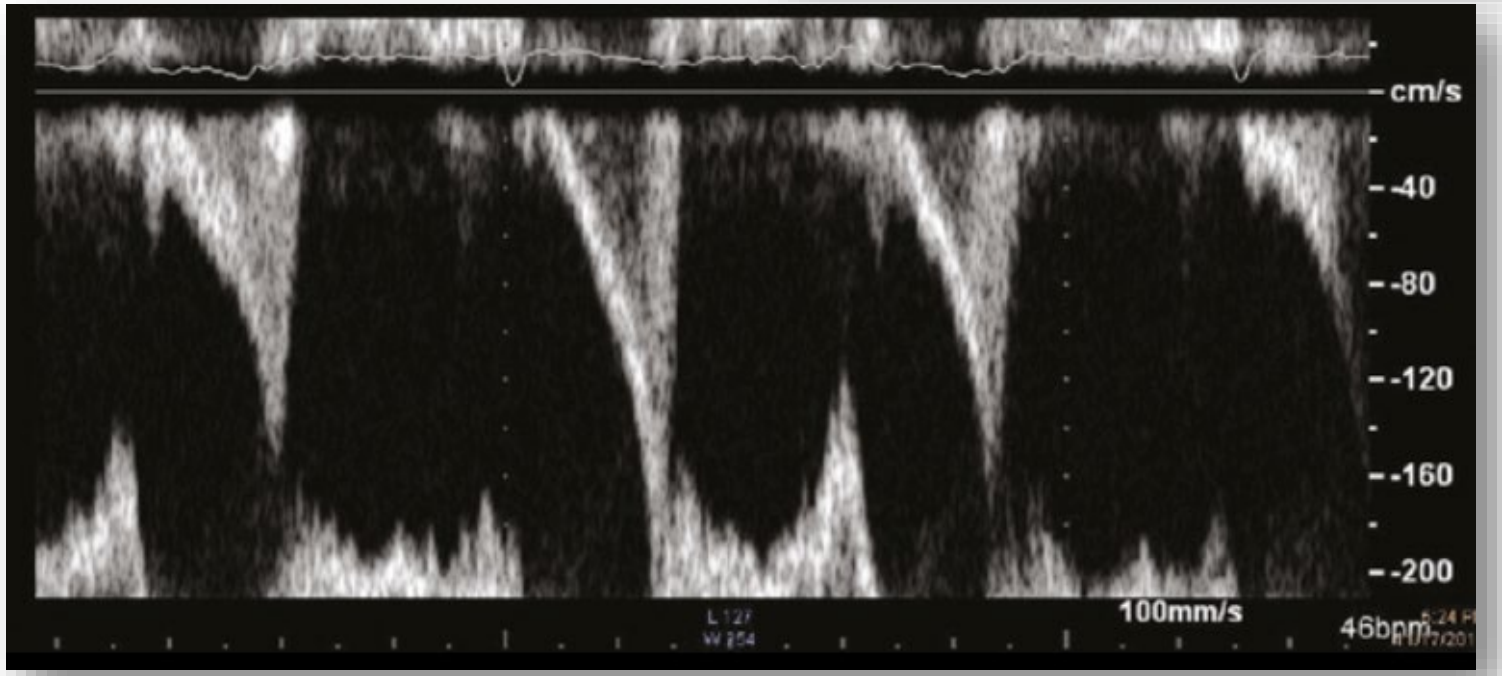
Rami El Mahmoud MD [a], Nicolas Mansencal MD [a b], Rémy Pilliére MD [a],

Journal of the American College of Cardiology
© 2001 by the American College of Cardiology
Published by Elsevier Science Inc.

Vol. 38, No. 1, 2001
ISSN 0735-1097/01/$20.00
PII S0735-1097(01)01316-X

Transient Left Ventricular Apical Ballooning Without Coronary Artery Stenosis: A Novel Heart Syndrome Mimicking Acute Myocardial Infarction

Kazufumi Tsuchihashi, MD, PHD,* Kenji Ueshima, MD, PHD,† Tatsuro Uchida, MD, PHD,‡



20-30%