Table 1: Heterogeneity underlying the clinical phenotypes of cardiogenic shock

Cardiogenic Shock (CS) Etiology	**Acute Myocardial Infarction-CS (AMI-CS)**	**Heart Failure-CS (HF-CS)**	**Refractory Cardiac Arrest-CS (CA-CS)**	**Post-Cardiotomy Shock (PCS)**	**Pulmonary Embolism-CS (PE-CS)**	**Valvular Heart Disease-CS (VHD-CS)**
Mechanism(s) of injury	Acute myocardial injury due to significant coronary occlusion	Acute myocardial systolic and/or diastolic dysfunction based on underlying disease etiology (multifactorial)	Acute global myocardial failure resulting from persistent arrhythmogenic injury instigated by underlying disease etiology (multifactorial)	Acute myocardial failure from intra-operative injury	Acute right ventricular or global circulatory failure from PE (sub-massive or massive obstruction of pulmonary vasculature)	Acute myocardial failure based on underlying disease etiology (multifactorial, e.g., aortic stenosis, mitral or tricuspid regurgitation)
Ventricular failure predominance	LV failure (LVF) predominant if left coronary occlusion; RV failure (RVF) predominant if right coronary occlusion; rarely biventricular failure (BiVF) unless mechanical complication(s) such as myocardial rupture	BiVF or LVF predominant depending on underlying disease etiology; RVF is often late during underlying disease process	BiVF predominant as injury is often global	BiVF predominant as injury is often global, but LVF or RVF predominant if isolated injury (e.g., left or right coronary occlusion)	RVF or BiVF predominant if sub-massive or massive PE	LVF, RVF or BiVF predominant dependent on VHD

Clinical presentation	Acute onset of chest pain syndrome	Acute onset if sudden elevation in left sided pressure overload due to underlying disease etiology, however, more often indolent onset of HF signs and symptoms (dyspnea, fluid retention) beyond a certain threshold where patient unable to compensate	Acute onset often leading to transient loss of consciousness due to cerebral malperfusion	Post-surgical / intra-operative as accompanied by inability to wean off cardiopulmonary bypass (CPB) support	Acute hypoxic respiratory failure without or with hemodynamic collapse or CA, if sub-massive or massive PE, respectively	Acute onset if sudden rupture of valvular apparatus leading to regurgitation, or more indolent if progression of vavlular lesion (stenotic or regurgitant)
Prognostication	Dependent on time of symptom onset, severity of presentation, ventricular or coronary involvement, and door-to-balloon time and door-to-unloading time:	Dependent on time of symptom onset, severity of presentation, ventricular involvement, door-to-unloading time, and severity of vasoplegia	Dependent on time of symptom onset, severity of presentation and time to return of spontaneous circulation from symptom onset (ROSC)	Dependent on severity of insult, duration of ischemic time on CPB support, and severity of vasoplegia	Dependent on severity of presentation, PE burden, and door-to-treatment time	Dependent on time of symptom onset, severity of presentation
Hemodynamic parameters	If LVF, then high LVEDP, LAP, PCWP, PADP, and SVR with	As with AMI-CS	As with AMI-CS	As with AMI-CS	If RVF, then high CVP, RAP and RVEDP, and low PAPi with normal	As with AMI-CS

	normal PAPi and normal right sided pressures If RVF, then high CVP, RAP and RVEDP, and low PAPi with normal left sided pressures and SVR If BiVF, then elevated left and right sided and pulmonary pressures, and low PAPi				left sided pressures and SVR If BiVF, then elevated left and right sided and pulmonary pressures, and low PAPi	
End-organ perfusion surrogates	Elevated lactate; elevated liver enzymes and creatinine dependent on severity of CS, if late presenting, or with mechanical complications; elevated CK-MB and Troponin	Elevated lactate, liver enzymes and creatinine dependent on severity of CS; elevated NT-proBNP/BNP and possibly Troponin and/or CK-MB related to etiology	Lactate clearance (as lactate level early on may not be reflective of severity of CS due to poor peripheral blood flow); elevated Troponin related to global injury to myocardium	Lactate clearance (as lactate level early on may not be reflective of severity of CS due to poor peripheral blood flow); possibly elevated CK-MB and Troponin related to etiology and/or surgical process	Elevated lactate; elevated liver enzymes and creatinine dependent on severity of CS or late presenting; elevated NT-prBNP/BNP and D-dimer, and possibly Troponin and/or CK-MB if RV or myocardial ischemia	Elevated lactate, liver enzymes and creatinine dependent on severity of CS; elevated NT-proBNP/BNP and possibly Troponin and/or CK-MB related to etiology
ECG changes	Ischemic changes (e.g., ST elevation and/or depression)	Dependent on disease etiology (e.g., ST changes if myocarditis, conduction abnormalities if amyloid or infiltrative diseases)	Arrhythmias (e.g., ventricular fibrillation or tachycardia, pulseless electrical alternans, asystole)	Dependent on etiology of insult (e.g., ischemic changes if coronary occlusion)	Sinus tachycardia or classic $S_1Q_3T_\varepsilon$ pattern; also (in)complete RBBB, RV strain with TWI in right V1-4 ± inferior leads if high pulmonary pressures, RAD, dominant R wave in V1 if acute RV dilation, RAE or P pulmonale,	Dependent on disease etiology and severity (e.g., LVH with strain and LAE in severe aortic stenosis; RVH with RAD and tall V1-2 R waves if PHTN, or non-specific right precordial ST and T wave

					clockwise rotation with R/S transition point and S wave in V6 if RV dilation, and atrial tachyarrhythmias	changes and RAE if RV dysfunction in severe tricuspid regurgitation; LVH, LAE and PHTN pattern or AF in chronic severe mitral regurgitation)
ECHO imaging parameters	Evidence of wall motion abnormalities of affected coronary territory; evidence of any mechanical complications (e.g., pericardial effusion, myocardial rupture, mitral insufficiency)	Reflective of disease etiology and chronicity (e.g., ventricular distension, wall thinning, septal bowing, valvular regurgitation and/or stenosis)	Ventricular standstill, or evidence of underlying cause (e.g., wall motion abnormality, pericardial effusion)	Global wall motion abnormality (unless isolated territorial insult); often RV distension and/or dysfunction as very sensitive to injury	RV distension and/or dysfunction, paradoxic septal bowing and tricuspid regurgitation based on extent of PE burden	Reflective of disease etiology, chronicity and severity (e.g., ventricular distension, wall thinning, septal bowing, valvular regurgitation and/or stenosis)
Drug usage	Dependent on severity of CS, often vasopressors for hypotension but may require inotropes and/or vasodilators	Dependent on phenotype of CS, often vasodilators and/or inotropes over vasopressors	Often vasopressors for hypotension, anti-arrhythmics and/or sedation if arrhythmia not quiescent	Often vasopressors and inotropes, may require inhaled epoprostenol if RV dysfunction	Dependent on severity of CS, care with IV fluids if RV overload, often vasopressors and/or inotropes (care of latter if tachycardia and systemic vasodilation), can consider inhaled NO	Dependent on severity of CS and disease etiology
Device usage	Dependent on severity of CS and ventricular involvement: If LVF, then IABP or Impella LV	As with AMI-CS	Often peripheral VA-ECMO	Often VA-ECMO, either central or peripheral dependent on surgery performed and technical ease, with LV venting as indicated	VA-ECMO if severe CS or CA (peripheral given acuity or central as more effective but invasive) and concomitant percutaneous mechanical aspiration or	As with AMI-CS, except not Impella LV in setting of severe aortic stenosis due to anatomical constraint

	If RVF, then Impella RP or Protek Duo If BiVF, then VA-ECMO with LV venting as clinically indicated				surgical thrombectomy preferred over thrombolytic; alternately RA-PA flow pump with reperfusion therapy; Impella RP or ProtekDuo but increases afterload and some concern for dislodging proximal clot distally	
Ventilatory considerations	Dependent on severity of hypoxia, BiPAP use should now follow pandemic considerations	As with AMI-CS	Establish airway as part of advanced cardiac life support (ACLS) algorithm and with pandemic considerations	Remains intubated from surgical procedure	Dependent on severity of hypoxia, care with BiPAP as can exacerbate hypotension	Dependent on severity of hypoxia, care with BiPAP in severe aortic stenosis as can exacerbate hypotension due to fixed cardiac output
Renal replacement considerations	Minimize contrast use for nephro-protection, renal replacement therapy (RRT) as clinically indicated	RRT as clinically indicated, ultrafiltration may be used to manage volume overload	RRT as clinically indicated once ROSC achieved	As with HF-CS	As with HF-CS	As with HF-CS