**Table 1.** Clinical, pathophysiological and management characteristics of non-ACS etiologies of CS.

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| Clinical setting | Clinical and Pathophysiological Characteristics | Key Treatment Elements |
| RV failure | * Depending on etiology and pathophysiology may present as “wet and cold” or “wet and warm” * without pulmonary artery hypertension (RV infarction, primary TR, RV cardiomyopathies) * with pulmonary artery hypertension (PE, ARDS) * Post cardiac surgery (MV repair, cardiac transplantation, LVAD); very common “wet and warm” * Post Cardiac Arrest; very common “wet and warm” | -look at primary etiology.  -volume status has a very narrow ideal range, as RV is more fluid dependent.  -mechanical ventilation should be adjusted to minimal conditions and patient should be to the prone position,  -SBP should be 40 mmHg greater than pulmonary pressure.  -norepinephrine or inotropes such as dobutamine or levosimendan may be used.  -In refractory patients, Impella-RP, TandemHeart RA-PA or VA-ECMO may be used.  -Severe RV failure occurs in 20-25% of cases post LVAD implant. CS post LVAD implant requires in postoperative settings a PAC-tailored management to optimize hemodynamics and volume status of the patient; the goal is CVP <15 mmHg. In this clinical setting, treatment includes aggressive use of inotropes, possibly inhaled nitric oxide, rhythm control with pacing or antiarrhythmics and mechanical RV support. |
| Myocarditis | * immune-mediated inflammatory response triggered by different stimuli, mostly viral infections or auto-immune disease * flu-like symptoms, increased cardiac biomarkers and clinical signs of AHF * search for giant-cell myocarditis-immediate RV endomyocardial biopsy * very common bi-ventricular involvement | -Cyclosporine+ Prednisone (for giant-cell)  -Early implantation of MCS; very often biventricular support |
| Takotsubo syndrome | * negative inotropic effects of high levels of endogenous or exogenous catecholamines; * transient high afterload; * very often dynamic LVOT obstruction * possibly microvascular dysfunction associated ischemia | -Avoid catecholamines and prefer milrinone/ levosimendan  - Selection of MCS is patient-specific, and Impella may be considered in selected cases,, while VA-ECMO increases afterload and may amplify mitral regurgitation and pulmonary edema (157)  -Early Beta-blocker therapy after hemodynamic stabilization |
| Cardiomyopathies | * first exclude secondary etiologies (valvular disease, hypertensive disease, coronary artery disease) * search for features of a specific etiology | * identify and treat precipitant factors * neurohormonal medication (including Beta-blockers) initiated early after hemodynamic stabilization * Beta-blockers and volume optimization for HOCM; avoid vasodilators |
| Peripartum CM | * PPCM and idiopathic DCM share clinical and genetic (titin truncating gene variants) features * a prolactin fragment (16Kd prolactine) is considered causal for the pathogenesis * very common LV thrombus is present * clinical outcome highly variable | * longer-term bromocryptine and prophylactic/therapeutic anticoagulation may improve outcomes; * early use of MCS * emergent caesarean section may be required |
| Valve lesions | * uncommon cause (i.e. mitral valve rupture due to ischemia, infective endocarditis, severe aortic stenosis) * decompensation of known VHD in the presence of acute precipitants | -identify and treat precipitant factors  -hemodynamic stabilization and assess the risk/benefit ratio for cardiac surgery  - MCS should be individualized based on pathophysiology of the valvular disease. Impella is the MCS of choice in patients with severe MR, while it is relatively contraindicated in patients with severe AS. Peripheral VA-ECMO is contraindicated in patients with AR.  -immediate surgery for NVE or PVE |
| Post cardiac surgery | * intraoperative complications, prolonged CPB (high levels of cytokines), insufficient cardio-protection and general morbidity contribute at CS * very often presents as vasodilatory CS (‘wet and warm”), due to pathophysiology with cytokines release following CPB. * localized tamponade (precipitating factors include: administration of anticoagulants, coagulation disorders, excessive mediastinal bleeding, the removal of epicardial pacing wires) * dynamic LV obstruction (precipitating factors include: hypovolemia, cardiac hypertrophy, aortic valve replacement, high dose inotropes) * acute refractory RV dysfunction especially with vasodilatory phenotype | * early echo is crucial to identify potentially correctable causes * identify precipitants and anticipate clinical scenario * avoid excess of catecholamines * Inability to wean from cardiopulmonary bypass and/or poor postoperative hemodynamics are indications for early MCS; Impella 5.0, VA-ECMO or both may be considered depending on clinical scenario. |
| CS in settings of Cardiac Arrest | * Post-resuscitation global myocardial stunning can cause transient pump failure lasting several hours, caused by a combination of oxidative stress, microthrombi formation, adrenergic excess, cytokine release, and myocardial ischemia–reperfusion injury, and amplified by initial cardiac insult responsive of `CS. * General ischemia–reperfusion injury may precipitate systemic vasodilation. * Delayed initiation of CPR, longer interval from start of CPR to ROSC, non-shockable rhythms, older age, many comorbidities, severe lactic acidosis on presentation are negative prognosis factors * The degree of brain damage determines clinical course and outcome. | - For patients with cardiac arrest refractory to CPR, E-CPR (ECMO support during CPR) may be considered. The goal of E-CPR is to support patients in refractory cardiac arrest of potentially reversible etiology (e.g. AMI, PE, cardiac injury) while reversible causes are being identified and treated. |
| Cancer | * CS in patients with cancer is preceded by different clinical entities, such as ACS, Takotsubo syndrome, myocarditis, thromboembolic events and pulmonary embo- lism, tamponade, and cardiac herniation * These clinical presentations can be attributed either to cancer itself or to its therapy, including surgery, chemotherapy (anthracycline and other agents such as: trastuzumab, VEGF inhibitors, proteasome inhibitors, immune checkpoint inhibitors, CAR-T cell therapies) or as a late consequence of radiotherapy, in association with pre-existing cardiovascular disease or risk factors. | * Treatment is similar to CS without cancer: hemodynamic stabilization and treatment of the underlying cause * MCS are not a contraindication in cancer patients * ACE-I and BB for cardiac protection during chemotherapy; |

***Abbreviations:*** *ACE-I=angiotensin converting enzyme inhibitors;* *ACS=acute coronary syndromes; AMI=acute myocardial infarction;* *AR=aortic regurgitation*; *ARDS acute respiratory distress syndrome; BB=beta-blockers; CPB cardiopulmonary bypass; CPR=cardiopulmonary resuscitation; IABP=intra-aortic balloon pump; LVAD left ventricular assist device, HCMO=hypertrophic obstructive cardiomyopathy, LVOTO=left ventricular outflow tract obstruction; MCS=mechanical circulatory support, NVE=native valve endocarditis; PE=pulmonary embolism; PVE=prosthetic valve endocarditis; RV right ventricle; VA-ECMO=veno-arterial extracorporeal membrane oxygenation*