



# The impact of COVID-19 on the cardiovascular system

Mohamed Toufic El Hussein, RN, PhD, NP<sup>1,2,3</sup>, Aditi Sharma, Graduate Nurse<sup>4</sup>

<sup>1</sup> Professor, School of Nursing and Midwifery, Faculty of Health, Community & Education, Mount Royal University

<sup>2</sup> Adjunct Associate Professor, Faculty of Nursing, University of Calgary

<sup>3</sup> Acute Care Nurse Practitioner Medical Cardiology, Coronary Care Unit - Rockyview General Hospital

<sup>4</sup> Graduate Nurse, School of Nursing and Midwifery, Faculty of Health, Community & Education, Mount Royal University

## Introduction

SARS-CoV-2, or coronavirus, is an airborne virus that affects multiple organ systems of an infected person (Zhou et al., 2020). The virus was first identified in Wuhan, China, and led the World Health Organization (WHO) (Zhou et al., 2020) to declare a global pandemic on January 30, 2020. As of September 05, 2021, there have been 220.5 million confirmed cases globally, with more than 196 million recovered, 5.4 billion vaccinated, and 4.5 million deaths worldwide (COVID-19 Map, 2020).

Common symptoms of SARS-CoV-2 include but are not limited to fever, dry cough, shortness of breath, fatigue, nasal congestion, diarrhea, nausea, throat tenderness, and confusion (Nejati et al., 2020). The maturation of SARS-CoV-2 symptoms can take up to four-five days and last 11-14 days after exposure (Guan et al., 2020). Patients with comorbidities like diabetes, cancer, hypertension, coronary artery disease, a chronic obstructive pulmonary disorder, and heart failure who get infected with SARS-CoV-2 are at higher risks for mortality and decreased quality of life (Bonow et al., 2020). The purpose of this review is to explore the impact of SARS-CoV-2 on the cardiovascular system.

## Transmission

SARS-CoV-2 is transmitted by direct contact with droplets expelled from infected people during coughing, sneezing, or talking (Wiersinga et al., 2020). Risk of transmission increases when exposure lasts 15 minutes or longer within a six-foot distance to an infected person (Wiersinga et al., 2020). The virus can also be transmitted via the fecal-oral route (Clerkin et al., 2020), aerosolized droplets suspended in the air, and contact with a surface contaminated with the virus (Wiersinga et al., 2020). Approximately 48-62% of all transmission cases occur

when an infected person is in the pre-symptomatic phase (Wiersinga et al., 2020). This means that although patients may not demonstrate common symptoms of infection, they can still infect other people through contact and droplet (Wiersinga et al., 2020). Respiratory and cardiovascular symptoms can take up to ten days to be visually apparent in an infected person (Wiersinga et al., 2020), highlighting the significance of using preventative measures like hand washing/sanitization, social distancing, and masks when in public places.

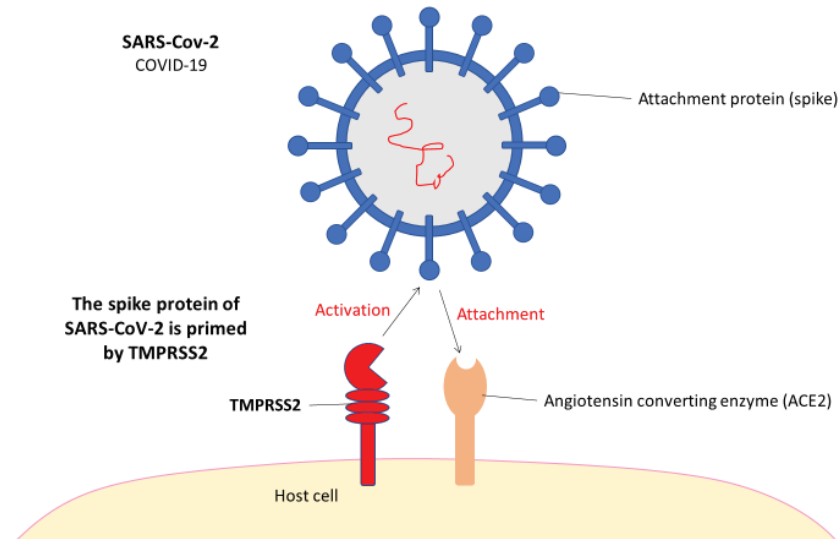
## COVID-19 Pathophysiology

SARS-CoV-2, with a diameter of 60 nm-140 nm and unique spike proteins that range from 9nm-12nm (Wiersinga et al., 2020), is a single-stranded ribonucleic acid (RNA) virus that infects the host by binding the spike proteins on its viral surface to the angiotensin-converting enzyme 2 (ACE2) receptor in type II alveolar cells of the human lungs. After binding, stimulation of the spike protein on the viral surface by the host enzyme transmembrane protease serine-2 (TMPRSS2) promotes the uptake of the virus by the alveolar cells (Hoffmann et al., 2020) (see Figure 1). The virus penetrates the alveolar cells by attaching its envelope to the alveolar cell membrane and undergoing endocytosis (Clerkin et al., 2020). In the initial stages of the infection, SARS-CoV-2 infects the epithelial cells in the nasal cavity, the bronchial spaces, and the pneumocytes (Wiersinga et al., 2020).

ACE2 receptors are an integral part of maintaining cardiovascular health, explicitly preventing hypertension, inflammation, myocardial remodeling, and hypertrophy (Bosso et al., 2020). Unfortunately, when SARS-CoV-2 co-opts ACE2 receptors to enter the host's epithelial cells in the target tissue, fewer ACE2 receptors will be available to achieve the anti-inflammatory goal resulting in exaggerated inflammation. The latter is mediated

**Figure 1**

*Pathogenesis of SARS-CoV-2*



through a 'cytokine storm' where excessive concentrations of multiple chemokines, interferons, interleukins (IL), and tumor necrosis factor (TNF)-alpha, are released into the bloodstream (Coperchini et al., 2020). The serum levels of pro-inflammatory cytokines like IL-1 $\beta$ , IL-6, IL-12, IL-18 increase significantly and exacerbate pulmonary and systemic inflammation (Huang et al., 2020). The latter can further lead to acute respiratory distress syndrome (ARDS) or severe multiple organ damage (Coperchini et al., 2020). A high serum level of IL-6 in SARS-CoV-2 infected patients is primarily a predictor of the infection's deterioration and severity (Coperchini et al., 2020). SARS-CoV-2 tends to bind ACE2 receptors expressed in the lungs, heart, and kidneys (Sandoval et al., 2020). The heart has an abundant presence of ACE2 receptors in its walls (Sandoval et al., 2020), thus increasing the vulnerability of myocardial cells to SARS-CoV-2 and eventually resulting in direct cell toxicity, viral infiltration of the myocardial tissue (Iqbal & Gupta, 2020). RNA sequencing and mapping were conducted by Johnson et al. (2020) and revealed that greater than 7.5% of myocardial cells are at increased susceptibility to SARS-CoV-2 due to greater expression of ACE2 in the heart. Patients treated with angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) tend to have upregulated ACE2 receptors providing more receptors for the SARS-CoV-2 virus to get attached and gain access to the affected tissues, further reinforcing SARS-CoV-2 infection severity (Rossi et al., 2020; Tikellis et al., 2020).

In addition to inflammation, the tendency to develop pulmonary edema reaches 80% in patients with pre-existing cardiac conditions like heart failure (Iqbal & Gupta, 2020). Systemic inflammation caused by SARS-CoV-2 induces morbid vasodilation and may result in cardiogenic shock (Wiersinga et al., 2020). Although cardiovascular disease (CVD) makes an individual more susceptible to contract SARS-CoV-2 due to compromised perfusion and weakened immunity, an individual infected with SARS-CoV-2 can also develop clinical manifestations and complications of CVD.

Cardiac damage and elevated serum troponin levels were prevalent in 36% of the 3,000 SARS-CoV-2 patients from a retrospective cohort study using observational data in New York (Lala et al., 2020). A strong connection between the myocardial injury and SARS-CoV-2 was identified in patients with pre-existing comorbidities (Lala et al., 2020). This indicates that SARS-CoV-2 can cause serious myocardial injury and an imbalance in cardiac biomarkers such as troponin that can lead to adverse health outcomes of the infected patient.

## Cardiovascular Complications

### Myocardial Injury: Myocarditis

Myocarditis is an injury of the myocardium caused by severe inflammation that exacerbates the humoral or cellular immune response to infectious toxins such as viruses, bacteria, or fungi (Tschöpe et al., 2019). Studies have shown evidence of SARS-CoV-2 in the myocardium through the polymerase chain reaction test (Sandoval et al., 2020), where the infection was not active; however, the damage was thought to be caused by the inflammatory response that follows the viral pathogenesis (Sandoval et al., 2020). As inflammation progresses and diffuses throughout the myocardium, it results initially in ischemia and hypoxia, eventually leading to the anaerobic metabolism of cells (Johnson et al., 2020). Anaerobic metabolism yields acidosis with an accumulation of lactic acid and production of free radicals in the interstitial space that causes further injury and damage of the myocardial cellular phospholipid bilayer (Johnson et al., 2020). Hypoxemia also results in an increased influx of calcium ions into the cytoplasm, causing apoptosis of myocytes (Johnson et al., 2020). A retrospective study conducted by Zhou et al. (2020) in Wuhan, China, found that approximately 20% of SARS-CoV-2 infected patients developed acute cardiac injury and coagulopathy. Autopsy reports of deceased patients infected with SARS-CoV-2 reported the presence of cardiomegaly, myocarditis, and right ventricular dilation. Furthermore, microscopic findings consisted of hypertrophy of cardiac myocytes, degeneration, edema, necrosis, infiltration of lymphocytes, monocytes, and neutrophils (Sandoval et al., 2020).

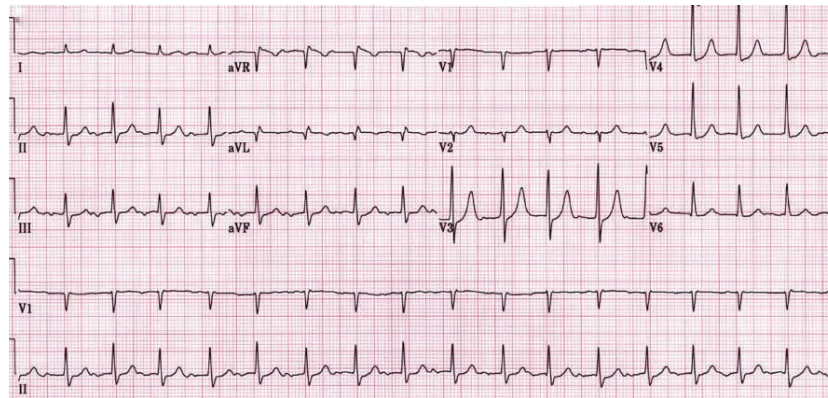
The rise in interleukins (ILs), specifically IL-six, has been reported to cause acute and delayed myocardial contractility, with a progressive increase in inflammation and a direct decrease in left ventricular function and capacity (Ptaszyńska-Kopczyńska et al., 2017). Increased cytokine release has also been related to cardiomyopathy and arrhythmias (Shimabukuro-Vornhagen et al., 2018). Lymphocytic intrusion potentiates an autoimmune response from SARS-CoV-2 viral pathology that can cause CVD and myocarditis (Fung et al., 2016). The manifestations of myocarditis range from severe destruction of the myocardium that generates cardiogenic shock to having a limited myocardial impact with no residual symptoms (Fung et al., 2016).

### Illustrative Case Study

Moe is a 39-year-old healthy male presenting to the ED with a history of lightheadedness, palpitations, and presyncope. Initial electrocardiogram (ECG) showed normal sinus rhythm with first-degree atrioventricular block (AVB; see Figure 2). A later ECG shows a bi-fascicular block including right bundle branch block (RBBB) and left anterior fascicular block (see Figure 3) that later progressed to the left bundle branch block (LBBB; see Figure 4). The patient reported a history of SARS-CoV-2 infection four weeks before his presentation that was associated with pleuritic chest discomfort. Troponin levels were elevated, and echocardiography showed diffuse myocardial

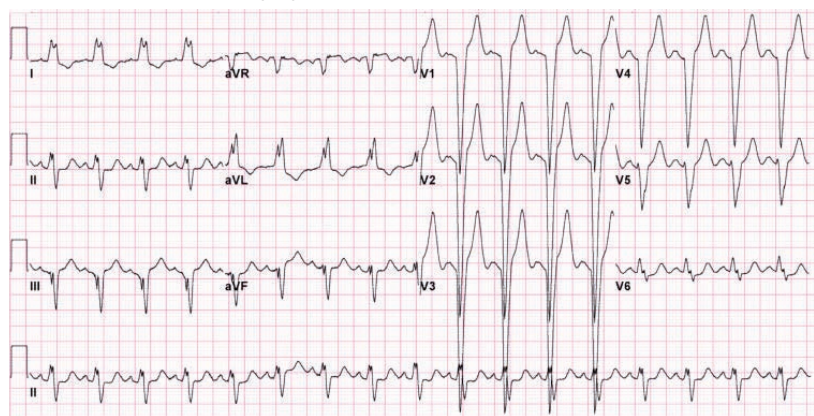
**Figure 2**

*Normal sinus rhythm with first-degree atrioventricular block*



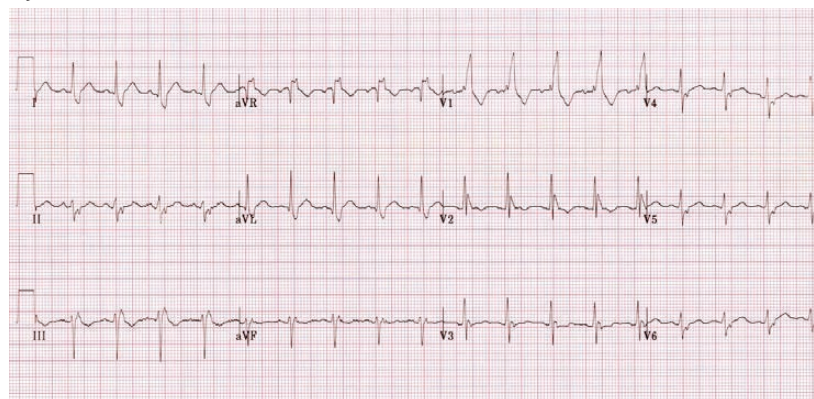
**Figure 3**

*Bi-fascicular block including right bundle branch block and left anterior fascicular block*



**Figure 4**

*Left bundle branch block*



dyskinesia and decreased left ventricular ejection fraction (LVEF). Cardiovascular magnetic resonance imaging (CMR) findings were consistent with myocardial inflammation. The CMR also reported global systolic dysfunction and moderately sizeable pericardial effusion. The patient was transferred to the coronary care unit (CCU) where his heart rate (HR) was averaging around 40 BPM, with ECG showing LBBB with intermittent RBBB. The cardiac monitor shows a blood pressure (BP) of 100/57 and an oxygen saturation of 98% on room air. The patient had negative Lyme serology. Chest X-ray (CXR) displayed moderate bilateral pleural effusion with no evidence of pneumonia. In CCU, temporary transvenous pacing wires were inserted with the HR at 80bpm, RV paced, MA 2 amps. A CXR confirmed appropriate placement. For five days in the CCU, the patient was in complete heart block with an intermittent transition into LBBB and RBBB, and he became symptomatic, particularly with any exertion leading to lightheadedness. The electrophysiologist recommended permanent pacemaker insertion and a dual-chamber, and then a rate-modulated mode (DDDR) pacemaker was implanted. The final diagnosis was SARS-CoV-2 infection-induced myocarditis complicated with LBBB/RBBB and complete heart block.

### Acute Coronary Syndrome (ACS)

ACS is a cardiovascular disease characterized by the disruption of a plaque that causes thrombosis or occlusion to one of the coronary arteries, resulting in acute unstable angina, myocardial infarction (MI), or sudden cardiac death (Kimura et al., 2019). A definitive diagnosis of MI is made by assessing the ECG and the presence of cardiac biomarkers (Johnson et al., 2020). Although SARS-CoV-2's focal area for infection is the respiratory system, Kwong et al. (2018), in Ontario, Canada, concluded that viral infections increase the risk of acute MI (Kwong et al., 2018). Patients with pre-existing coronary artery disease and stable plaques can develop acute myocardial infarction (AMI) when infected with SARS-CoV-2, as severe systemic inflammation can disrupt plaque stability (Kwong et al., 2018).

Inflammatory mediators such as TNF-alpha, interleukins, cytokines, and chemokines play a direct role in causing AMI (Sandoval et al., 2020). SARS-CoV-2 infected patients in intensive care units (ICUs) tend to have higher serum cytokines and chemokines than non-ICU patients, further increasing the risks of developing cellular myocardial injury (Sandoval et al., 2020). The incidence rate of MIs in patients who developed SARS-CoV-2 is unsubstantiated due to limited research to support the data. Cases of microvascular dysfunction and coronary thrombosis unrelated to plaque disturbance have also been reported in SARS-CoV-2 infected patients (Sandoval et al., 2020).

Systemic inflammation or sepsis caused by a viral infection like SARS-CoV-2 was also linked to ventricular diastolic and systolic dysfunction (Zochios et al., 2014). A systematic review found that severe sepsis correlates with increasing serum troponin levels, with a 60% prevalence rate (Zochios et al., 2014). A comprehensive review conducted by Johnson et al. (2020) found that 69.4% of patients ( $n = 187$ ) hospitalized in China with SARS-CoV-2 had elevated troponin T (TnT) levels and were also at

higher risk for inpatient mortality (7.6%). Heart failure SARS-CoV-2 infected patients were at high risk of developing acute hemodynamic decompensation (AHD) (Bonow et al., 2020).

Bonow et al. (2020) reviewed studies conducted in the USA, Italy, and China that focused on patients with underlying cardiac complications infected with SARS-CoV-2 and established that the majority of the patients included in the studies presented with elevated troponin levels indicating underlying myocardial damage (Bonow et al., 2020). Another multicenter cross-sectional study conducted by Lombardi et al. (2020) in Italy reported that 45.3% of hospitalized patients ( $n = 614$ ) infected with SARS-CoV-2 had elevated TnT levels and increased risk of in-hospital death (71%). The same study also concluded that patients ( $n = 614$ ) infected with SARS-CoV-2 had twice an increase in the likelihood of developing sepsis, pulmonary embolism, extensive bleeding, multiorgan failure, and acute kidney failure (Lombardi et al., 2020). Further data from this study proclaimed a six-times increase in cases of heart failure, MI, and other cardiovascular complications in patients with elevated serum troponin levels (Lombardi et al., 2020).

### Arrhythmias: Atrial and Ventricular

Another cardiac clinical manifestation of SARS-CoV-2 infected patients is arrhythmia. A life-threatening arrhythmia occurs when frequent ventricular tachycardia lasts longer than 30 seconds. It can prompt ventricular fibrillation and hemodynamic instability with abnormal blood pressure (Guo et al., 2020). A retrospective study conducted in Wuhan, China, found that 7.3% of a total of 187 SARS-CoV-2 positive patients showed palpitations as a primary symptom for cardiovascular complications (Guo et al., 2020). Further review revealed that 17% of SARS-CoV-2-infected hospitalized patients developed undefined arrhythmia (Guo et al., 2020) and 5.9% developed ventricular fibrillation and tachycardia (Guo et al., 2020). SARS-CoV-2 infected patients that depended on ventilator support had a 17.7% prevalence of having atrial arrhythmias in a cohort study of 393 hospitalized patients in the USA (Goyal et al., 2020). Common types of arrhythmias associated with the SARS-CoV-2 infection are sinus tachycardia, atrial flutter, atrial fibrillation, and ventricular tachycardia (Bhatla et al., 2020).

Incidences of arrhythmias and cardiac arrests observed in patients with SARS-CoV-2 are not solely due to the impact of viral load on the myocardium but are in conjunction with underlying conditions that add to illness severity. Non-cardiovascular causes, including sepsis and systemic inflammation, increase the risk of developing arrhythmias in hospitalized SARS-CoV-2 patients in an ICU setting (Bhatla et al., 2020). Prolonged arrhythmias add to the increased risk of myocardial tissue necrosis, heart failure, cardiac arrest, and direct interference with the hearts' electrical conduction system. They also impose more significant risks for thrombotic issues like venous and arterial thrombosis as a direct result of severe damage caused by the release of inflammatory mediators (Bhatla et al., 2020).

Atrial fibrillation in conjunction with underlying inflammatory response increases the risks for thrombotic and embolic

complications (Bhatla et al., 2020). A global perspective study was conducted in 76 countries to gather data from various healthcare professionals revealed that arrhythmias such as ventricular arrhythmias and pulseless electrical activity are common manifestations with SARS-CoV-2 infected hospitalized patients (Gopinathannair et al., 2020). Further discussion depicted that atrial fibrillation and sinus bradycardia were the most prevalent types of arrhythmias observed in SARS-CoV-2 infected patients (Gopinathannair et al., 2020). Babapoor-Farrokhran et al. (2020) reported an increase in the incidence of idioventricular rhythms, sinus node dysfunction, atrioventricular blocks, and bradycardia in a SARS-CoV-2 infected patient (Babapoor-Farrokhran et al., 2020). The arrhythmias lasted for two weeks from the onset of a maladaptive sinus node (Babapoor-Farrokhran et al., 2020). The report also discussed the presence of an intermittent higher degree of atrioventricular block in an infected patient with normal cardiac biomarkers and echocardiogram (Babapoor-Farrokhran et al., 2020).

Figure 5, modified from Siripanthong et al. (2020), displays acute and chronic mechanisms for arrhythmogenesis in SARS-CoV-2-related myocarditis. This is manifested through cardiomyocyte injury, pericardial inflammation, microvascular ischemia, gap junction dysfunction, and non-ischemic scar (Siripanthong et al., 2020).

### Diagnosics Investigations: Troponin and ECGs

Elevated cardiac troponin T (cTnT) is a primary predictor of necrosis and myocardial injury associated with increased ICU admission, poor prognosis, and mortality in SARS-CoV-2 patients (Aboughdir et al., 2020). Increased cTnT levels suggest severe myocardial injury and are common in SARS-CoV-2

infected patients, with more than 70% at risk of experiencing intermittent fatal arrhythmias (Guo et al., 2020). Observation and measurement of cTnT in SARS-CoV-2 patients in hospitals are recommended to facilitate and prioritize care to prevent other adverse outcomes (Sandoval et al., 2020).

Electrocardiogram (ECG) findings reported cases of SARS-CoV-2 patients in China illustrating an S1Q3T3 pattern where there was a presence of an S wave in lead I, Q wave in lead III, and inverted T wave in lead III along with a temporal atrioventricular block (AVB) (He et al., 2020). Elevated ST-segment in the infected patient occurred due to hypotension and extreme hypoxia, along with myocardial injury and inflammation induced by the virus (He et al., 2020).

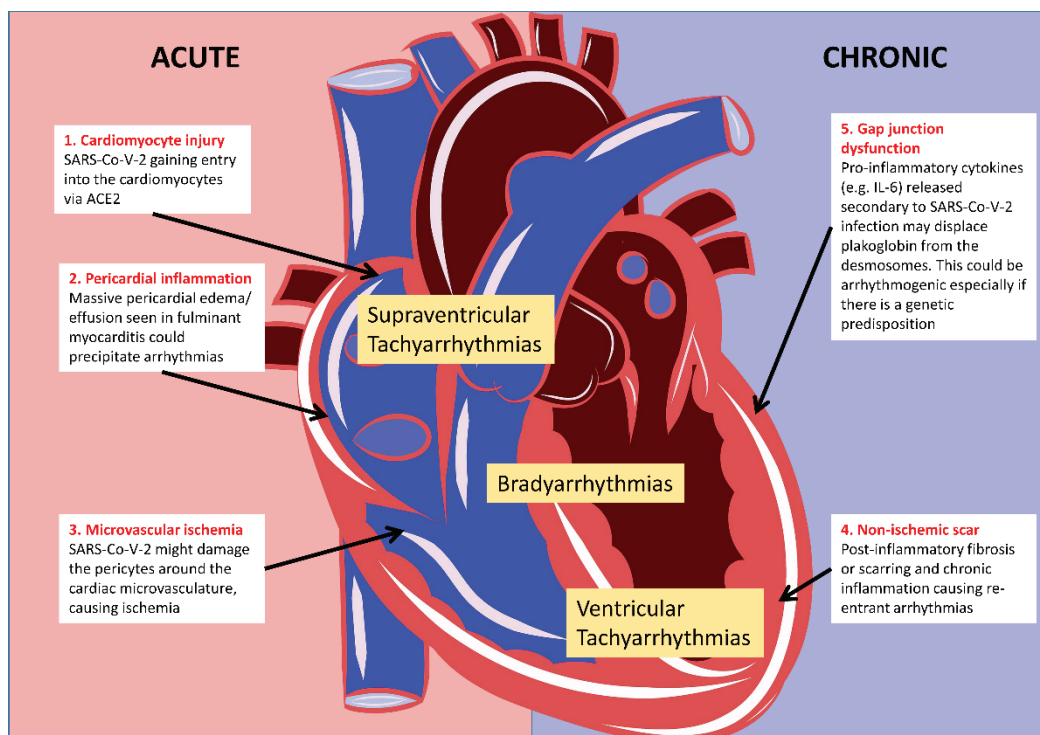
Fulminant myocarditis (FM) was reflected on ECG of SARS-CoV-2 patients with the depression of the PR-segment in the precordial and limb leads and alternating ST-segment elevation (Mansoor et al., 2020). Q waves and bundle branch blocks from delays in the electrical conduction system were also seen with FM (Mansoor et al., 2020). Although it is a common finding in patients with FM, a widened QRS complex and right bundle branch block have been observed in infected patients with cardiovascular complications, leading to the development of pathological arrhythmias (Mansoor et al., 2020).

### Nursing Implications for ED Nurses

#### Cardiovascular Considerations

Nursing care for infected patients suspected of cardiologic complications like ACS, arrhythmias, and AMI in the ED should consist of triage and quick management by establishing a nursing diagnosis, isolating, and case management by requesting diagnostic tests for cardiac enzymes and an ECG (Matos et al.,

Figure 5



2021). Suggest and encourage orders for low molecular weight heparin and the use of sequential compression devices to prevent the risk of venous and arterial thromboembolism in immobile patients (Matos et al., 2021). Therefore, nurses should focus on abnormal activated partial thromboplastin time along with any signs of elevations in the D-Dimer test for anticoagulant dose adjustment (Deitrick et al., 2020).

In a myocardial injury and elevated creatine kinase situation, cardiac enzymes should be monitored (Matos et al., 2021). Nursing assessments and interventions for infected patients that develop myocarditis and arrhythmias are ECG monitoring and frequent measurements of the Q-T interval if the patient is receiving antiretroviral or antimalarial therapy (Dawson et al., 2020; Deitrick et al., 2020; Guzik et al., 2020). Many patients infected with SARS-CoV-2 are at risk for vascular and thrombotic complications. When atherosclerosis and ACS are suspected, treatment options to differentiate the diagnosis consist of cardiac catheterization (Matos et al., 2021). Nurses can play a role in preparing and educating the patient of the procedures and tests, formulating a plan for transferring to ICU, and initiating early rehabilitation and community referrals (Matos et al., 2021). Oxygen therapy, anxiety reduction, and comfort care are essential to sustain the health of SARS-CoV-2 patients (Fusi-Schmidhauser et al., 2020). Some patients can experience red discoloration and lesions/bumps in addition to swelling on their toes, called 'COVID toes' (Deitrick et al., 2020). Therefore, an integumentary assessment would be pertinent for suspected SARS-CoV-2 patients to rule out circulatory and integumentary complications (Deitrick et al., 2020). Nursing priority for all patients in the ED includes effective PPE use to reduce exposure and virus spread, isolation precautions, supportive care like intubation and oxygen therapy, and monitoring for side effects from medication use and relevant management of the symptoms (Deitrick et al., 2020). Lastly, in SARS-CoV-2 patients experiencing cardiopulmonary failure, ARDS, or obstinate cardio-circulatory compromise, extracorporeal membrane oxygenation (ECMO) can be initiated to preserve cardiopulmonary health and function (Bartlett et al., 2020). Indications for initiating ECMO include a partial pressure of oxygen below 100 despite optimal respiratory interventions, respiratory rate of more than 35 breaths per minute (with or without mechanical ventilation), and/or case by case eligibility depending on prognosis, age, and comorbidities (Bartlett et al., 2020). ED nurses are experts in assessing the need for advanced Oxygen therapies and in collaborating with the rest of the team to expedite the implementation of interventions that are not usually available in the ED such as the ED (ECMO provided usually in the ICU). Hence, knowing contraindications for initiating ECMO such as neuromuscular blockade, high positive-end expiratory pressure, inhalation of any pulmonary vasodilators, and high frequency oscillatory ventilation can provide guidance to transitioning patients to admission in specific units (e.g., ICU), or aid in understanding the treatment options implemented in a SARS-CoV-2 patient (Bartlett et al., 2020).

### **Individual and Family-Centered Considerations**

Providing nursing care effectively amidst the pandemic must consider the delivery of family-centered care. This means respecting the family members' role as partners in caregiving, maintaining collaboration between the family and the healthcare team, and

preserving family integrity (Hart et al., 2020). Doing so can prevent and reduce family members' feelings of depression, anxiety, and post-traumatic stress disorder (PTSD) after patient hospitalization (Hart et al., 2020). In addition, it can provide benefits to patient recovery, reduce burnout, and distress among healthcare providers (Hart et al., 2020). Nursing care must incorporate patient education, promote comfort and medication adherence, including determinants of health, along with symptom management and regular assessments to prevent further development of detrimental outcomes (Hart et al., 2020). The deployment of tablet devices can aid in virtual evaluation, consults, planning care, and preserving PPE supplies in the ED (Wittbold et al., 2020). This can be done by mounting the device on an IV pole and placing it 6-8 feet away from the patient (Wittbold et al., 2020). Similarly, a proposed concept of utilizing iPad devices to consult family meetings and prevent patient isolation can promote the family-centered care model without compromising the health of patient(s), staff, and family by preventing unnecessary exposure (Wittbold et al., 2020).

### **Psychosocial Considerations**

A cross-sectional study conducted in the United Kingdom of 4 378 clinical and non-clinical staff highlighted that healthcare workers working in high-risk populations like the ED during the pandemic have a prevalence of PTSD in 30.2% of participants, depression in 27.3%, anxiety in 23.2%, and alcohol misuse in 10.5% (Lamb et al., 2021). Numerous nurses working in the ED experience physical isolation, sleep disturbances, unsafe working environments, and direct patient contact, which are primary factors for mental health challenges (Garcia-Martin et al., 2021; Mukhtar, 2020). Mukhtar (2020) presented a discussion on the importance of supporting nurses with mental health challenges while working in high-risk work environments. They highlighted that in Pakistan, many healthcare providers working in acute settings experience one or more forms of mental health challenges that can have long-term recovery compared to a physical injury (Mukhtar, 2020). This illustrates the prevalence of mental health challenges in acute care healthcare workers that can hinder them from providing quality care in a clinical setting. They suggested utilizing psychological services such as counseling, self-care therapy, psychotherapy and implementing a psychological crisis intervention to address stressors (Mukhtar, 2020). This can assist in providing depersonalization, modify the perspectives on life, allow for self-reflection and improvement, and cope with psychological reactions in a challenging, high-risk work environment (Mukhtar, 2020).

### **Conclusion**

The spread of SARS-CoV-2 has caused pathological complications on compromised and infected patients and added workload and burden to the healthcare system. Raising awareness among ED nurses that SARS-CoV-2 can cause cardiovascular complications like myocardial injury, myocarditis, MI, and arrhythmias can aid in identifying imminent care priorities and develop appropriate nursing interventions. Monitoring diagnostic values like CBC, vascular tests, cardiac biomarkers, and ECGs can promptly diagnose critical conditions, potentially preventing deterioration. Lastly, ED nurses working amidst this

pandemic must indulge in self-care and protect themselves physically and mentally.

### Clinical implications for emergency nursing

SARS-CoV-2 (COVID-19) is a viral illness that although primarily targets the respiratory system (giving rise to conditions like ARDS, pneumonia, infections, the risk for sepsis, etc.) also compromises the myocardium as the trauma experienced by a patient's body from exposure to SARS-CoV-2 creates an inflammatory cascade, thereby impacting other major organs and organ systems.

1. Look for signs and symptoms of cardiac-related complications early on, such as changes in cardiac biomarkers, inflammatory markers, and ECG can detect cardiac compromise and aid in rapid treatment to prevent deterioration.
2. Advocate for patient lab tests and management options such as ensuring cardiac biomarkers are ordered, preventative treatment is in place (mechanical stalkings, oxygen saturation, adequate PPE, etc.).
3. Ensure focused assessments to ensure cardiopulmonary system function is maintained, and if a decline is observed, prompt management by advanced practitioners can be utilized upon the recommendation from the primary nurses (such as ECMO).
4. Participate in family-centered care, using technology to connect to a patient's loved one in COVID restrictions, and displaying empathy despite personal or societal differences.
5. Practice regular self-care, relaxation, and seeking additional resources (counseling, therapy) to maintain mental health wellbeing and fitness to practice.

### About the Authors

Dr. El-Hussein is a Ph.D.-prepared Nurse Practitioner (Ph.D.

and NP completed at the University of Calgary) who has been working in academia for the past 20 years. He is a Full Professor in the School of Nursing and Midwifery, in the Faculty of Health, Community, and Education at MRU where he has taught for the past decade. He's also an adjunct Associate Professor in the Faculty of Nursing at the University of Calgary. Dr. El-Hussein has published over 50 articles in a variety of national and international nursing journals and always has research projects on the go. Dr. El-Hussein's impact on nursing education in Canada is reflected in his contributions to the science of nursing education through publications, grants, presentations, and other knowledge dissemination activities. To stay current in clinical practice as a Nurse Practitioner (NP) Dr. El-Hussein continues to hold a casual position as an NP in the division of Cardiology at the Rockyview General Hospital. As a Nurse Practitioner (NP) and academic Dr. El Hussein sees himself as a Knowledge broker who has the knowledge and expertise to facilitate bridging the gap between research and practice to improve outcomes for patients and keep his students up to date. Because he remains clinically active, Dr. El Hussein's research is grounded in the day-to-day issues pertaining to patients and relevant to the students.

Aditi Sharma is a fourth-year student enrolled in the Bachelor of Nursing Program at Mount Royal University. She is a motivated individual who has previously participated in various leadership and contributor roles with the Student Nursing Society and the Student Association of Mount Royal University. Additionally, she is the founder of her new Health & Wellness business specializing in CPR-BLS training and Public Health Awareness Initiatives. Academically, she is inspired to research imminent pathologies that are related to advancing current nursing practice. In the future, Aditi hopes to pursue advanced practice nursing education to build her experience in clinical and research scholarship.

### References

- Aboughdir, M., Kirwin, T., Abdul Khader, A., & Wang, B. (2020). Prognostic value of cardiovascular biomarkers in COVID-19: A review. *Viruses*, 12(5), 527. <https://doi.org/10.3390/v12050527>
- Babapoor-Farrokhran, S., Rasekhi, R. T., Gill, D., Babapoor, S., & Amanullah, A. (2020). Arrhythmia in COVID-19. *SN Comprehensive Clinical Medicine*, 2(9), 1430–1435. <https://doi.org/10.1007/s42399-020-00454-2>
- Bartlett, R. H., Ogino, M. T., Brodie, D., McMullan, D. M., Lorusso, R., MacLaren, G., Stead, C. M., Rycus, P., Fraser, J. F., Belohlavek, J., Salazar, L., Mehta, Y., Raman, L., & Paden, M. L. (2020). Initial ELSO guidance document: ECMO for COVID-19 patients with severe cardiopulmonary failure. *ASAIO Journal*, 66(5), 472–474. <https://doi.org/10.1097/mat.0000000000001173>
- Bhatla, A., Mayer, M. M., Adusumalli, S., Hyman, M. C., Oh, E., Tierney, A., Moss, J., Chahal, A. A., Anesi, G., Denduluri, S., Domenico, C. M., Arkles, J., Abella, B. S., Bullinga, J. R., Callans, D. J., Dixit, S., Epstein, A. E., Frankel, D. S., Garcia, F. C., ... Deo, R. (2020). COVID-19 and cardiac arrhythmias. *Heart Rhythm*, 17(9), 1439–1444. <https://doi.org/10.1016/j.hrthm.2020.06.016>
- Bonow, R. O., Fonarow, G. C., O'Gara, P. T., & Yancy, C. W. (2020). Association of coronavirus disease 2019 (COVID-19) with myocardial injury and mortality. *JAMA Cardiology*, 5(7), 751. <https://doi.org/10.1001/jamacardio.2020.1105>
- Bosso, M., Thanaraj, T. A., Abu-Farha, M., Alanbaei, M., Abubaker, J., & Al-Mulla, F. (2020). The two faces of ACE2: The role of ACE2 receptor and its polymorphisms in hypertension and COVID-19. *Molecular Therapy – Methods & Clinical Development*, 18, 321–327. <https://doi.org/10.1016/j.omtm.2020.06.017>
- Coperchini, F., Chiovato, L., Croce, L., Magri, F., & Rotondi, M. (2020). The cytokine storm in COVID-19: An overview of the involvement of the chemokine/chemokine-receptor system. *Cytokine & Growth Factor Reviews*, 53, 25–32. <https://doi.org/10.1016/j.cytogfr.2020.05.003>
- COVID-19 Map – Johns Hopkins Coronavirus Resource Center. (2020). Johns Hopkins Coronavirus Resource Center. <https://coronavirus.jhu.edu/map.html>
- Clerkin, K. J., Fried, J. A., Raikhelkar, J., Sayer, G., Griffin, J. M., Masoumi, A., Jain, S. S., Burkhoff, D., Kumaraiah, D., Rabbani, L., Schwartz, A., & Uriel, N. (2020). COVID-19 and cardiovascular disease. *Circulation*, 141(20), 1648–1655. <https://doi.org/10.1161/circulationaha.120.046941>
- Dawson, D., Dominic, P., Sheth, A., & Modi, M. (2020). Prognostic value of cardiac biomarkers in COVID-19 infection: A meta-analysis. Research Square <https://doi.org/10.21203/rs.3.rs-34729/v1>

- Deitrick, K., Adams, J., & Davis, J. (2020). Emergency nursing care of patients with novel coronavirus disease 2019. *Journal of Emergency Nursing*, 46(6), 748–759. <https://doi.org/10.1016/j.jen.2020.07.010>
- Fung, G., Luo, H., Qiu, Y., Yang, D., & McManus, B. (2016). Myocarditis. *Circulation Research*, 118(3), 496–514. <https://doi.org/10.1161/circresaha.115.306573>
- Fusi-Schmidhauser, T., Preston, N. J., Keller, N., & Gamondi, C. (2020). Conservative management of COVID-19 patients—Emergency palliative care in action. *Journal of Pain and Symptom Management*, 60(1), e27–e30. <https://doi.org/10.1016/j.jpainsymman.2020.03.030>
- García-Martín, M., Roman, P., Rodríguez-Arrastia, M., Díaz-Cortés, M. del M., Soriano-Martin, P. J., & Ropero-Padilla, C. (2020). Novice nurse's transitioning to emergency nurse during COVID-19 pandemic: A qualitative study. *Journal of Nursing Management*, 29(2), 258–267. <https://doi.org/10.1111/jonm.13148>
- Gopinathannair, R., Merchant, F. M., Lakkireddy, D. R., Etheridge, S. P., Feigofsky, S., Han, J. K., Kabra, R., Natale, A., Poe, S., Saha, S. A., & Russo, A. M. (2020). COVID-19 and cardiac arrhythmias: A global perspective on arrhythmia characteristics and management strategies. *Journal of Interventional Cardiac Electrophysiology*, 59(2), 329–336. <https://doi.org/10.1007/s10840-020-00789-9>
- Goyal, P., Choi, J. J., Pinheiro, L. C., Schenck, E. J., Chen, R., Jabri, A., Satlin, M. J., Champion, T. R., Nahid, M., Ringel, J. B., Hoffman, K. L., Alshak, M. N., Li, H. A., Wehmeyer, G. T., Rajan, M., Reshetnyak, E., Hupert, N., Horn, E. M., Martinez, F. J., ... Safford, M. M. (2020). Clinical characteristics of COVID-19 in New York City. *New England Journal of Medicine*, 382(24), 2372–2374. <https://doi.org/10.1056/nejmc2010419>
- Guan, W., Ni, Z., Hu, Y., Liang, W., Ou, C., He, J., Liu, L., Shan, H., Lei, C., Hui, D. S. C., Du, B., Li, L., Zeng, G., Yuen, K.-Y., Chen, R., Tang, C., Wang, T., Chen, P., Xiang, J., ... Zhong, N. (2020). Clinical characteristics of coronavirus disease 2019 in China. *New England Journal of Medicine*, 382(18), 1708–1720. <https://doi.org/10.1056/nejmoa2002032>
- Guo, T., Fan, Y., Chen, M., Wu, X., Zhang, L., He, T., Wang, H., Wan, J., Wang, X., & Lu, Z. (2020). Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). *JAMA Cardiology*, 5(7), 811. <https://doi.org/10.1001/jamacardio.2020.1017>
- Guzik, T. J., Mohiddin, S. A., Dimarco, A., Patel, V., Savvatis, K., Marelli-Berg, F. M., Madhur, M. S., Tomaszewski, M., Maffia, P., D'Acquisto, F., Nicklin, S. A., Marian, A. J., Nosalski, R., Murray, E. C., Guzik, B., Berry, C., Touyz, R. M., Kreutz, R., Wang, D. W., ... McInnes, I. B. (2020). COVID-19 and the cardiovascular system: Implications for risk assessment, diagnosis, and treatment options. *Cardiovascular Research*, 116(10), 1666–1687. <https://doi.org/10.1093/cvr/cvaa106>
- Hart, J. L., Turnbull, A. E., Oppenheim, I. M., & Courtright, K. R. (2020). Family-centered care during the COVID-19 era. *Journal of Pain and Symptom Management*, 60(2), e93–e97. <https://doi.org/10.1016/j.jpainsymman.2020.04.017>
- He, J., Wu, B., Chen, Y., Tang, J., Liu, Q., Zhou, S., Chen, C., Qin, Q., Huang, K., Lv, J., Chen, Y., & Peng, D. (2020). Characteristic electrocardiographic manifestations in patients With COVID-19. *Canadian Journal of Cardiology*, 36(6), 966.e1–966.e4. <https://doi.org/10.1016/j.cjca.2020.03.028>
- Hoffmann, M., Kleine-Weber, H., Schroeder, S., Krüger, N., Herrler, T., Erichsen, S., Schiergens, T. S., Herrler, G., Wu, N.-H., Nitsche, A., Müller, M. A., Drosten, C., & Pöhlmann, S. (2020). SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. *Cell*, 181(2), 271–280.e8. <https://doi.org/10.1016/j.cell.2020.02.052>
- Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., Zhang, L., Fan, G., Xu, J., Gu, X., Cheng, Z., Yu, T., Xia, J., Wei, Y., Wu, W., Xie, X., Yin, W., Li, H., Liu, M., ... Cao, B. (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *The Lancet*, 395(10223), 497–506. [https://doi.org/10.1016/s0140-6736\(20\)30183-5](https://doi.org/10.1016/s0140-6736(20)30183-5)
- Iqbal, M.A., & Gupta, M. (2020). Cardiogenic pulmonary edema. *National Library of Medicine*. <https://www.ncbi.nlm.nih.gov/books/NBK544260/>
- Johnson, K. D., Harris, C., Cain, J. K., Hummer, C., Goyal, H., & Periseti, A. (2020). Pulmonary and extra-pulmonary clinical manifestations of COVID-19. *Frontiers in Medicine*, 7. <https://doi.org/10.3389/fmed.2020.00526>
- Kimura, K., Kimura, T., Ishihara, M., Nakagawa, Y., Nakao, K., Miyauchi, K., Sakamoto, T., Tsujita, K., Hagiwara, N., Miyazaki, S., Aki, J., Arai, H., Ishii, H., Origuchi, H., Shimizu, W., Takemura, H., Tahara, Y., Morino, Y., Iino, K., ... Yamazaki, T. (2019). JCS 2018 Guideline on diagnosis and treatment of acute coronary syndrome. *Circulation Journal*, 83(5), 1085–1196. <https://doi.org/10.1253/circj.19-0133>
- Kwong, J. C., Schwartz, K. L., Campitelli, M. A., Chung, H., Crowcroft, N. S., Karnauchow, T., Katz, K., Ko, D. T., McGeer, A. J., McNally, D., Richardson, D. C., Rosella, L. C., Simor, A., Smieja, M., Zahariadis, G., & Gubbay, J. B. (2018). Acute myocardial infarction after laboratory-confirmed influenza infection. *New England Journal of Medicine*, 378(4), 345–353. <https://doi.org/10.1056/nejmoa1702090>
- Lamb, D., Gnanaprasam, S., Greenberg, N., Bhundia, R., Carr, E., Hotopf, M., Razavi, R., Raine, R., Cross, S., Dewar, A., Docherty, M., Dorrington, S., Hatch, S. L., Wilson-Jones, C., Leightley, D., Madan, I., Marlow, S., McMullen, I., Rafferty, A. M., & Parsons, M. (2021). The psychosocial impact of the COVID-19 pandemic on 4,378 UK healthcare workers and ancillary staff: Initial baseline data from a cohort study collected during the first wave of the pandemic. medRxiv. <https://doi.org/10.1101/2021.01.21.20240887>
- Lala, A., Johnson, K. W., Januzzi, J. L., Russak, A. J., Paranjpe, I., Richter, F., Zhao, S., Somani, S., Van Vleck, T., Vaid, A., Chaudhry, F., De Freitas, J. K., Fayad, Z. A., Pinney, S. P., Levin, M., Charney, A., Bagiella, E., Narula, J., Glicksberg, B. S., ... Fuster, V. (2020). Prevalence and impact of myocardial injury in patients hospitalized with COVID-19 infection. *Journal of the American College of Cardiology*, 76(5), 533–546. <https://doi.org/10.1016/j.jacc.2020.06.007>
- Lombardi, C. M., Carubelli, V., Iorio, A., Inciardi, R. M., Bellasi, A., Canale, C., Camporotondo, R., Catagnano, F., Dalla Vecchia, L. A., Giovinozzo, S., Maccagni, G., Mapelli, M., Margonato, D., Monzo, L., Nuzzi, V., Oriecuia, C., Peveri, G., Pozzi, A., Provenzale, G., ... Senni, M. (2020). Association of troponin levels with mortality in Italian patients hospitalized with coronavirus disease 2019. *JAMA Cardiology*, 5(11), 1274. <https://doi.org/10.1001/jamacardio.2020.3538>
- Mansoor, A., Chang, D., & Mitra, R. (2020). Rhythm, conduction, and ST elevation with COVID-19: Myocarditis or myocardial infarction? *HeartRhythm Case Reports*, 6(10), 671–675. <https://doi.org/10.1016/j.hrcr.2020.08.001>
- Matos, J.H.F. de, Gomes, E.B., Formiga, N.P.F., Nascimento, M.N.R., Lima, G. de S., & Moreira, T.M.M. (2021). Interventions related to cardiovascular complications in people hospitalized by COVID-19: A scoping review. *Revista Brasileira de Enfermagem*, 74(suppl 1). <https://doi.org/10.1590/0034-7167-2020-0568>
- Mukhtar, S. (2020). Mental well-being of nursing staff during the coronavirus disease 2019 outbreak: A cultural perspective. *Journal of Emergency Nursing*, 46(4), 426–427. <https://doi.org/10.1016/j.jen.2020.04.003>
- Nejati, A., Moghaddam, M.A., Seyedhosseini-Davarani, S.H., Akhgar, A. (2020). Clinical recommendation for emergency physicians to approach to signs and symptoms related to COVID-19; A preliminary study. *Advanced Journal of Emergency Medicine*, 4(2s). <https://doi.org/10.22114/ajem.v4i2s.456>



- Ptaszynska-Kopczynska, K., Szpakowicz, A., Marcinkiewicz-Siemion, M., Lisowska, A., Waszkiewicz, E., Witkowski, M., Jakim, P., Galar, B., Musial, W. J., & Kamiński, K. A. (2017). Interleukin-6 signaling in patients with chronic heart failure treated with cardiac resynchronization therapy. *Archives of Medical Science*, 5, 1069–1077. <https://doi.org/10.5114/aoms.2016.58635>
- Rossi, G., Sanga, V., Barton, V. (2020). Potential harmful effects of discontinuing ACE-inhibitors and ARBs in COVID-19 Patients. *eLife* 9. <https://doi.org/10.7554/eLife.57278>
- Sandoval, Y., Januzzi, J. L., & Jaffe, A. S. (2020). Cardiac troponin for assessment of myocardial injury in COVID-19. *Journal of the American College of Cardiology*, 76(10), 1244–1258. <https://doi.org/10.1016/j.jacc.2020.06.068>
- Shimabukuro-Vornhagen, A., Gödel, P., Subklewe, M., Stemmler, H. J., Schlöber, H. A., Schlaak, M., Kochanek, M., Böll, B., & von Bergwelt-Baildon, M. S. (2018). Cytokine release syndrome. *Journal for ImmunoTherapy of Cancer*, 6(1). <https://doi.org/10.1186/s40425-018-0343-9>
- Siripanthong, B., Nazarian, S., Muser, D., Deo, R., Santangeli, P., Khanji, M. Y., Cooper, L. T., & Chahal, C. A. A. (2020). Recognizing COVID-19-related myocarditis: The possible pathophysiology and proposed guideline for diagnosis and management. *Heart Rhythm*, 17(9), 1463–1471. <https://doi.org/10.1016/j.hrthm.2020.05.001>
- Tikellis, C., & Thomas, M. C. (2012). Angiotensin-converting enzyme 2 (ACE2) is a key modulator of the renin angiotensin system in health and disease. *International Journal of Peptides*, 2012, 1–8. <https://doi.org/10.1155/2012/256294>
- Tschöpe, C., Cooper, L. T., Torre-Amione, G., & Van Linthout, S. (2019). Management of myocarditis-related cardiomyopathy in adults. *Circulation Research*, 124(11), 1568–1583. <https://doi.org/10.1161/circresaha.118.313578>
- Wiersinga, W. J., Rhodes, A., Cheng, A. C., Peacock, S. J., & Prescott, H. C. (2020). Pathophysiology, transmission, diagnosis, and treatment of coronavirus disease 2019 (COVID-19). *JAMA*, 324(8), 782. <https://doi.org/10.1001/jama.2020.12839>
- Wittbold, K. A., Baugh, J. J., Yun, B. J., Raja, A. S., & White, B. A. (2020). iPad deployment for virtual evaluation in the emergency department during the COVID-19 pandemic. *The American Journal of Emergency Medicine*, 38(12), 2733–2734. <https://doi.org/10.1016/j.ajem.2020.04.025>
- Zhou, F., Yu, T., Du, R., Fan, G., Liu, Y., Liu, Z., Xiang, J., Wang, Y., Song, B., Gu, X., Guan, L., Wei, Y., Li, H., Wu, X., Xu, J., Tu, S., Zhang, Y., Chen, H., & Cao, B. (2020). Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: A retrospective cohort study. *The Lancet*, 395(10229), 1054–1062. [https://doi.org/10.1016/s0140-6736\(20\)30566-3](https://doi.org/10.1016/s0140-6736(20)30566-3)
- Zochios, V., & Valchanov, K. (2014). Raised cardiac troponin in intensive care patients with sepsis, in the absence of angiographically documented coronary artery disease: A systematic review. *Journal of the Intensive Care Society*, 16(1), 52–57. <https://doi.org/10.1177/1751143714555303>

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