Summer 2017

Pathophysiology of Myocardial Reperfusion Injury after Ischemia

Rachel Fetrow
rachel.fetrow@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Part of the Nursing Commons

Recommended Citation
https://digitalcommons.otterbein.edu/stu_msn/214
Myocardial infarction results in a loss of cardiac function and results in damage to the myocardium. Timely restoration of blood flow is the first line of treatment to prevent tissue injury. However, the process of reperfusion can itself induce cardiomyocyte damage, known as myocardial reperfusion injury (Hausenloy & Yellon, 2013).

Signs of myocardial reperfusion injury include:

- **Increased ventricular arrhythmias**
- **Sustained or non-sustained ventricular tachycardia**
- **Accelerated idioventricular rhythm (AIVR)**
- **Atrioventricular dissociation (AVD)**
- **Cardiogenic shock**

Myocardial reperfusion injury can also lead to long-term complications and poor outcomes (Arons et al., 2013). Understanding the pathophysiology of myocardial reperfusion injury is crucial for developing therapeutic strategies to prevent it.

**Intrinsic cell changes**

- **Mitochondrial dysfunction**
- **Ca2+ overload**
- **Oxidative stress**
- **Apoptosis**

**Extrinsic factors**

- **Neutrophil infiltration**
- **Plaque rupture**
- **Collateral flow**

**Consequences of reperfusion injury**

- **Hypertrophy**
- **Diastolic dysfunction**
- **Arrhythmias**

Reperfusion injury and reactive oxygen species (ROS) are key mechanisms in the development of myocardial reperfusion injury (Braunersreuther & Yellon, 2013). The process of reperfusion can lead to a sudden increase in oxygen supply, which causes the release of ROS, leading to oxidative stress and cell damage.

**Implications for Nursing Care**

ST-Elevation myocardial infarction patients with complete occlusion in a large artery with little coronary collateralization are more likely to benefit from a therapeutic intervention (Fröhlich et al., 2015). The protocol for this therapy should be followed within 1 minute of STEMI deployment.

- **Hyperthermia and Hypertrophic Hypercapnia**
  - Reducing NO synthesis through inhibition of iNOS and blocking signaling pathways and microvascular dysfunction during ischemia to 32°C can limit MI size in experimental studies by reducing metabolic demand, interstitial edema, reperfusion injury, pleiotropism, and increasing myocardial efficiency (Frolich et al., 2013).

Remote Ischemic Conditioning (RIC) involves applying three 5-minute cycles of brief non-lethal ischemia and reperfusion using blood pressure cuffs applied to the upper arm. RIC has been shown to significantly reduce MI size and improve LV ejection fraction. A meta-analysis of 19 RCTs concluded that RIC has a beneficial effect on patients at risk of MI (Frolich et al., 2011).