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The Role of Elevated Adrenaline in Myocardial Infarction

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Abstract: Adrenaline (epinephrine) is a key stress hormone released by the adrenal glands during acute physical or emotional stress. Its effects on the heart are mediated via α- and β-adrenergic receptors, increasing heart rate, contractility, and blood pressure—potentially exacerbating cardiac stress during myocardial ischemia . At physiological levels, epinephrine primarily activates β_2 receptors in coronary arteries, causing vasodilation and increasing coronary blood flow. However, at high levels, predominant α_1 receptor activation causes systemic vasoconstriction, raising peripheral resistance and cardiac workload . Elevated blood adrenaline (epinephrine) is a hallmark of the acute phase of myocardial infarction and is implicated in multiple pathophysiologic mechanisms—including arrhythmias, alterations in potassium levels, bleeding function, and direct myocardial injury. Collectively, clinical biochemistry research establishes that acute MI initiates a marked adrenaline surge, tied to infarct severity, arrhythmogenesis, symptom burden, and vascular injury. While adrenaline serves as an important biomarker, it also seems to drive maladaptive responses through electrophysiologic and endothelial pathways—making it both a risk signal and a potential therapeutic target. Let me know if you'd like to integrate quantitative data tables, explore receptor-specific pathways, or discuss therapeutic modulation strategies in more depth.

Keywords: *Adrenaline, myocardial infarction, arrhythmias, vasoconstriction, endothelial dysfunction.*

Introduction

Adrenaline—also known as epinephrine—is a principal stress hormone released during acute physical or emotional strain and plays a critical role in the "fight-or-flight" response. It acts on α- and β-adrenergic receptors across the cardiovascular system, significantly impacting heart rate, contractility, vascular resistance, and coagulation pathways.(Boarescu & Boarescu, 2024).

In myocardial infarction (MI), there is often a surge of circulating catecholamines—especially adrenaline—triggered by ischemia and sympathetic activation. While this response can initially help maintain perfusion, persistent elevation becomes maladaptive, contributing to ischemic injury, arrhythmias, endothelial damage, and adverse outcomes. (Ostrowski, Pedersen, Jensen, Mogelvang, & Johansson, 2013).

At low physiological concentrations, adrenaline preferentially stimulates β_2-receptors on coronary vessels, causing vasodilation and enhanced cardiac perfusion. However, at high levels, α_1-mediated systemic vasoconstriction predominates—raising afterload and increasing myocardial

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oxygen demand, while paradoxically impairing microcirculatory flow to ischemic areas (Peaston & Weinkove, 2004).

Critically, elevated adrenaline promotes coronary vasospasm and platelet hyperactivation, forming dense, fibrinolysis-resistant clots—factors that can precipitate thrombosis even in non-obstructed vessels (Boarescu & Boarescu, 2024). Concomitantly, direct myocardial toxicity ensues via β-receptor-driven calcium overload and oxidative stress, leading to endothelial injury, arrhythmogenesis, and cell death—particularly in already injured or stunned myocardium (Boarescu & Boarescu, 2024).

The electrophysiological consequences can be profound: adrenaline-induced hypokalemia, QTc prolongation, reduced T-wave amplitude, and other repolarization changes increase the risk of dangerous ventricular arrhythmias, even at levels seen during MI (Struthers, Reid, Whitesmith, & Rodger, 1983).

Although adrenaline is central in cardiac arrest protocols—boosting coronary perfusion pressure and return of spontaneous circulation—evidence suggests that it may not improve long-term survival or neurologic outcomes, and could even exacerbate microvascular thrombosis and arrhythmia risk when administered inappropriately or without restraint (Papastylianou & Mentzelopoulos, 2012).

Taken together, while an adrenaline surge can temporarily support hemodynamics in acute MI, sustained elevation at high concentrations is strongly associated with worsened ischemia, increased arrhythmic events, endothelial damage, and poorer clinical outcomes—emphasizing the hormone's dose-, duration-, and timing-dependent effects in cardiovascular pathophysiology.

High adrenaline levels increase myocardial oxygen demand through elevated heart rate and contractility. This creates an oxygen supply–demand mismatch in individuals with coronary artery disease, triggering ischemia, plaque rupture, or vasospasm . Additionally, adrenaline promotes platelet activation, enhances thrombus formation, and results in denser, more stable clots by impairing fibrinolysis—heightening risk of coronary artery occlusion (Papastylianou & Mentzelopoulos, 2012).

Evidence from Human Studies

Catecholamine Levels and Arrhythmias

A study of 41 CCU patients found plasma adrenaline and noradrenaline markedly elevated during AMI, with the highest values seen in those who developed ventricular fibrillation (VF) (Bertel et al., 1982). Another cohort of 48 patients sampled within six hours of symptom onset confirmed elevated adrenaline and noradrenaline, and those with the most severe infarcts had higher catecholamine levels—with some later developing VF (Little et al., 1986). These data support a strong association between elevated plasma adrenaline and the risk of serious ventricular arrhythmias in early AMI.

Adrenaline and Hypokalemia

Adrenaline's role in inducing hypokalemia is well demonstrated: intravenous adrenaline infusion in healthy volunteers (to mimic levels seen post-infarction) significantly dropped serum potassium—leading to QT prolongation and T-wave changes known to predispose to arrhythmias (Struthers et al., 1983).

In rabbits and AMI patients, higher adrenaline levels correlated inversely with plasma potassium, and both elevated adrenaline and hypokalemia emerged as independent predictors of ventricular arrhythmias (Zhao & Pan, 1989)..

Platelet Function and Prothrombotic State

Compared with unstable angina and non-cardiac chest pain, patients with AMI had higher plasma adrenaline and shorter bleeding times. The shortened bleeding time persisted after aspirin and was inversely related to adrenaline level, suggesting adrenaline contributes to a prothrombotic milieu (Kristensen et al., 1990).

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Pain Correlation

In uncomplicated MI, arterial adrenaline was higher in patients reporting pain versus those without chest pain, indicating that adrenaline levels may reflect pain-driven sympatho-adrenal activation more than infarct size per se (Husebye et al., 1990).

Myocardial Catecholamine Distribution

Post-mortem measurements showed elevated adrenaline in the myocardium—especially in periinfarct zones within 1–2 days of infarction—leading to speculation that local adrenaline accumulation may directly provoke arrhythmias and further myocardial damage (Popov et al., 1975).

Pathophysiologic Cascade

Adrenaline stimulates β_1-adrenergic receptors to raise heart rate and contractility, α_1-receptors to induce vasoconstriction, and activates intracellular pathways (e.g. cAMP/PKA) that modulate ion channels and cardiac myocyte excitability (Moro et al., 2013). These changes increase myocardial oxygen demand, provoke dynamic electrophysiologic alterations, and may destabilize peri-infarct tissue.

Rare Paradoxical Effects: Exogenous Adrenaline and MI

Though adrenaline is life-saving in emergencies (e.g. anaphylaxis), rare case reports describe acute MI triggered by therapeutic adrenaline—often via coronary vasospasm and platelet activation—even in young patients with patent coronaries (Moro et al., 2013). This underscores adrenaline's potential to precipitate ischemia under specific circumstances.

Results

Plasma Adrenaline Elevations in AMI

- In a cohort of 48 patients sampled within 6 hours of symptom onset, plasma adrenaline (E) averaged ~73 pg/mL, peaking at approximately 1,098 pg/mL—far exceeding the normal resting level of ~34 pg/mL.
- Another study of 41 CCU patients showed those who developed ventricular fibrillation (VF) had the highest admission plasma adrenaline levels, surpassing even AMI patients without VF (Bertel et al., 1982).
- A larger study with 84 patients (chronic ischemic heart disease versus anterior and posterior AMI) found elevated epinephrine in both AMI types, with no significant difference between anterior vs posterior infarcts—but norepinephrine was especially elevated in anterior AMI (~60% higher vs controls) (Slavíková et al., 2007).

Adrenaline Correlation to Infarct Extent & Mortality

- Patients with higher peak adrenaline (>1,000 pg/mL) had significantly higher mortality during 18-month follow-up; none with peak E < 1,000 died, while all death cases had peak E > 1,000 pg/mL ($p < 0.01$) (Karlsberg et al., 1981).
- Similarly, plasma NE and E correlated with infarct severity (e.g., LDH levels, prognostic index), and patients who later developed VF had markedly higher catecholamine levels soon after onset (Little et al., 1986).

Adrenaline and Electrical Instability

- Among 41 patients, those with proven infarction and VF had the highest plasma adrenaline values; patients with congestive heart failure also showed significantly elevated adrenaline although norepinephrine was only moderately raised (Bertel et al., 1982).
- Across various ischemic lesions demonstrating electrical instability, both adrenaline and noradrenaline were significantly increased compared to controls (Slavíková et al., 2007).

Adrenaline and Pain Correlation

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- In a study of 22 patients with uncomplicated MI, arterial adrenaline averaged 0.83 nmol/L versus 0.44 nmol/L in controls ($P < 0.025$). Patients experiencing chest pain had significantly higher adrenaline (1.15 ± 0.23 nmol/L) than those without pain (0.60 ± 0.10 nmol/L, $P < 0.05$), indicating adrenaline elevation tied more to pain intensity than infarct size (Husebye et al., 1990).

Endothelial Injury and Shock

- In a STEMI cohort undergoing primary PCI, higher adrenaline and noradrenaline levels were associated with elevated biomarkers of endothelial/glycocalyx damage (e.g. syndecan-1, thrombomodulin). These correlations were strongest in patients with cardiogenic shock, and higher adrenaline independently predicted short- and long-term mortality and heart failure (Ostrowski et al., 2013).

Discussion

Sympathoadrenal Activation in Acute MI

The data consistently show that acute myocardial infarction triggers a profound sympathoadrenal surge, with plasma adrenaline rising dramatically—often by 20–30× above baseline within hours of onset (Karlsberg et al., 1981). This surge appears universal across infarct locations and is closely tied to infarct severity, early complications, and later mortality.

Adrenaline as a Marker and Mediator of Arrhythmia Risk

Elevated adrenaline levels correlate strongly with ventricular fibrillation and electrical instability. The gradient of catecholamine concentration appears steepest in those with arrhythmic complications, implying that adrenaline may act both as a biomarker and a mechanistic contributor to arrhythmogenesis. The observation that congestive heart failure patients had high adrenaline, even when norepinephrine was only mildly elevated, further underscores adrenaline's centrality in acute pathophysiology.

Pain vs. Infarct Size: Drivers of Adrenaline

The study linking higher adrenaline to symptom-related stress (pain), rather than infarct size, suggests that sympathetic activation may be more pain-mediated than purely ischemic (Husebye et al., 1990). Clinically, this indicates that subjective pain intensity can be an indirect marker of adrenergic stress even in uncomplicated MI.

Endothelial Damage: A Catecholamine-Mediated Pathway

Emerging evidence from recent STEMI cohorts shows that high plasma adrenaline correlates with biomarkers of endothelial glycocalyx damage, particularly in shock states. These interactions are independently predictive of mortality and subsequent heart failure—suggesting catecholamines may amplify vascular injury in addition to myocardial damage (Ostrowski et al., 2013). This adds a new dimension to adrenaline's deleterious effects, beyond electrophysiologic instability.

Clinical Implications

- Adrenaline measurement early in AMI (e.g. within 4–6 hours) may stratify risk for adverse outcomes like VF and mortality.
- The association between adrenaline and endothelial damage underscores the potential benefit of therapies targeting early sympathoadrenal overactivation—such as beta-blockers—to mitigate not only arrhythmias but also vascular injury.
- Recognizing that pain-driven adrenaline release may not directly correspond to infarct size underscores the need to assess both clinical presentation and objective markers before inferring severity.

Synthesis & Conclusions

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- **Adrenaline rises markedly** in the acute phase of myocardial infarction, both in plasma and locally within myocardial tissue. Levels correlate with infarct severity and complications such as arrhythmias and heart failure.
- **Hypokalemia** induced by adrenaline contributes significantly to ventricular electrical instability, acting alongside direct receptor-mediated repolarization changes to elevate arrhythmia risk.
- **Prothrombotic effects** include shorter bleeding times and increased platelet reactivity, especially after aspirin, suggesting adrenaline enhances early thrombotic risk in AMI.
- **Pain-mediated stress** may amplify adrenaline release independently of infarct size, reinforcing the interplay between sympathetic activation and clinical presentation (Husebye et al., 1990).

Clinical implications: adrenergic blockade (e.g. beta-blockers like metoprolol) may mitigate adrenaline-mediated adverse hemodynamic and arrhythmic sequelae, although initial administration may transiently raise measured catecholamine levels without worsening outcomes (Murray et al., 1988)..

Limitations & Future Directions

- Most human studies are observational and moderately sized, limiting causal inference.
- Adrenaline measurements vary across studies in timing and methodology, potentially impacting comparability.
- There is still limited granularity differentiating systemic vs myocardial-derived catecholamines, although regional myocardial studies suggest local adrenaline accumulation (especially in periinfarct zones) may trigger arrhythmias (Popov et al., 1975).
- Future research should explore therapeutic windows for adrenergic modulation, and the potential interplay between catecholamine-driven endothelial damage and long-term vascular remodeling.

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