CHAPTER 12

TREATMENT ALGORITHMS FOR MEDICAL EMERGENCIES

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Abstract

The management of medical emergencies is dictated by time-sensitive algorithms designed to rapidly stabilize physiology and reverse life-threatening pathology. The Acute Coronary Syndrome (ACS) algorithm bifurcates based on the initial EKG; STEMI requires an immediate reperfusion algorithm, prioritizing primary PCI, while NSTEMI management is guided by risk stratification. The acute stroke algorithm is similarly time-dependent, centered on an immediate non-contrast CT scan to differentiate ischemic from hemorrhagic stroke. Ischemic stroke management is an algorithm of reperfusion, using IV thrombolysis (alteplase) within a 4.5-hour window and mechanical thrombectomy for large vessel occlusion up to 24 hours. The sepsis algorithm is a bundled protocol ("Hour-1 Bundle") mandating lactate measurement, blood cultures, rapid broad-spectrum antibiotic administration, 30 ml/kg crystalloid resuscitation for hypotension, and vasopressors to maintain mean arterial pressure. The anaphylaxis algorithm is linear and prioritized: immediate intramuscular epinephrine is the first-line and life-saving intervention, followed by adjunctive treatments like antihistamines and corticosteroids. The DKA/HHS algorithm is a multi-pronged metabolic resuscitation, balancing three components: aggressive intravenous fluid repletion, continuous insulin infusion (after confirming potassium levels), and vigilant electrolyte (especially potassium) monitoring and replacement.

Keywords: *Sepsis, Stroke, Acute Coronary Syndrome, Anaphylaxis, Diabetic Ketoacidosis, Emergency Medicine*

Learning Objectives

After completion of the chapter, the learners should be able to:

- List the core components of the "Sepsis Hour-1 Bundle."
- Explain the time-sensitive treatment algorithm for acute ischemic stroke, including the indications for thrombolysis and thrombectomy.
- Initiate the correct, immediate treatment algorithm (including dosing) for a patient presenting with anaphylactic shock.
- Compare and contrast the fluid and insulin management algorithms for Diabetic Ketoacidosis (DKA) versus Hyperosmolar Hyperglycemic State (HHS).
- Prioritize the life-saving interventions in the algorithm for a patient with septic shock, managing competing treatment goals.

ACUTE CORONARY SYNDROME

Acute Coronary Syndrome (ACS) refers to a spectrum of conditions where myocardial blood supply is acutely reduced. The management algorithm is one of the most time-critical in medicine, as "time is muscle." This section focuses on the immediate emergency department algorithm (a more detailed cardiology-focused discussion is in Chapter 1).

Pathophysiology

The universal pathophysiology is the **rupture or erosion of an atherosclerotic plaque** within a coronary artery. This rupture exposes the highly thrombogenic lipid core of the plaque to the bloodstream, triggering an immediate coagulation cascade.

Platelets adhere and aggregate, and a fibrin mesh is formed, leading to the development of an occlusive or sub-occlusive **intracoronary thrombus (clot)**.

- In **STEMI**, the thrombus is typically *fully occlusive*, cutting off all blood flow to a large territory of the myocardium. This causes transmural (full-thickness) ischemia and rapid necrosis, visible as ST-segment elevation on an EKG.
- In **NSTEMI/UA**, the thrombus is typically *partially*

occlusive. It limits, but does not completely block, blood flow. This causes subendocardial (partial-thickness) ischemia. In NSTEMI, the ischemia is severe or prolonged enough to cause myocyte necrosis, releasing cardiac troponins. In UA, the ischemia is transient and does *not* cause necrosis (troponins are negative).

Diagnosis and Classification

The initial algorithm begins with any patient presenting with symptoms suggestive of cardiac ischemia (e.g., chest pain, dyspnea, syncope).

- **Step 1: Immediate EKG.** An EKG must be obtained and interpreted within 10 minutes of first medical contact. This is the primary branch point in the algorithm.
- **Step 2: Classification.**
    - **STEMI (ST-Segment Elevation Myocardial Infarction):** The EKG shows ST-segment elevation in two or more contiguous leads or a new left bundle branch block. This indicates a complete, occlusive thrombus.
    - **NSTEMI/UA (Non-ST-Segment Elevation MI / Unstable Angina):** The EKG may show ST-segment depressions, T-wave inversions, or may be non-specific.
- **Step 3: Cardiac Troponins.** Blood is drawn for cardiac troponin levels at presentation and serially. A rise and/or fall in troponin with ischemic symptoms confirms MI (STEMI or NSTEMI).

Differential Diagnosis

The differential diagnosis for acute chest pain includes other life-threatening conditions that must be considered in the initial algorithm:

- **Aortic Dissection:** Severe, tearing, or ripping pain, often radiating to the back.
- **Pulmonary Embolism (PE):** Pleuritic chest pain, dyspnea, and tachycardia.
- **Tension Pneumothorax:** Sudden-onset dyspnea,

tracheal deviation, and hypotension.

- **Esophageal Rupture (Boerhaave Syndrome):** Severe chest pain following forceful vomiting.
- **Pericarditis:** Positional, pleuritic pain, often with diffuse ST elevations.

Treatment Algorithm

The algorithm diverges immediately based on the EKG classification.

STEMI Algorithm (Reperfusion):

- **Goal:** Immediate reperfusion. "Time-to-device" is the main metric.
- **First-line:** Primary Percutaneous Coronary Intervention (PCI) is the preferred algorithm. The goal is a "first medical contact-to-device" time of ≤ 90 minutes (if at a PCI-capable hospital) or ≤ 120 minutes (if requiring transfer).
- **Second-line (Fibrinolysis):** If PCI cannot be performed within these timeframes, the algorithm shifts to intravenous fibrinolytic therapy (e.g., alteplase, tenecteplase). This must be given within 30 minutes of arrival and requires strict screening for contraindications (e.g., prior ICH, recent stroke).
- **Adjunctive Medications:** Immediate aspirin (324 mg chewed), a P2Y12 inhibitor (e.g., ticagrelor, prasugrel), and anticoagulation (e.g., heparin) are given.

NSTEMI/UA Algorithm (Risk Stratification):

- **Goal:** Stabilize, risk-stratify, and determine the need for invasive management.
- **Initial Medications:** Aspirin, a P2Y12 inhibitor (e.g., clopidogrel, ticagrelor), anticoagulation (e.g., enoxaparin, heparin), nitrates (for pain, if not hypotensive), and beta-blockers (if stable).

- **Risk Stratification:** The algorithm uses a risk score (e.g., TIMI or GRACE) to classify patients.
- **Early Invasive Strategy:** Patients with high-risk features (e.g., refractory angina, hemodynamic instability, dynamic EKG changes, high GRACE score) proceed to angiography within 24-48 hours.
- **Conservative Strategy:** Low-risk patients may be managed medically first, followed by non-invasive stress testing to guide further therapy.

Table 12.1: ACS Initial Management Algorithm (STEMI vs. NSTEMI)

ACS Type	EKG Finding	Immediate Treatment Algorithm
STEMI	**ST-Segment Elevation** (in ≥ 2 contiguous leads).	**Immediate Reperfusion:** Primary PCI (if < 90-120 min). (Fibrinolysis if PCI delayed). **MONA-B + DAPT.**
NSTEMI / UA	**ST-Depression** or **T-Wave Inversion.** (No ST elevation).	**MONA-B + DAPT + Anticoagulation.** (Heparin). **Risk Stratify (TIMI/GRACE):** High-Risk → Early Invasive (PCI).

MONA-B: Morphine, Oxygen (if sat < 90%), Nitrates, Aspirin, Beta-blocker. DAPT: Dual Antiplatelet Therapy (Aspirin + P2Y12-I).

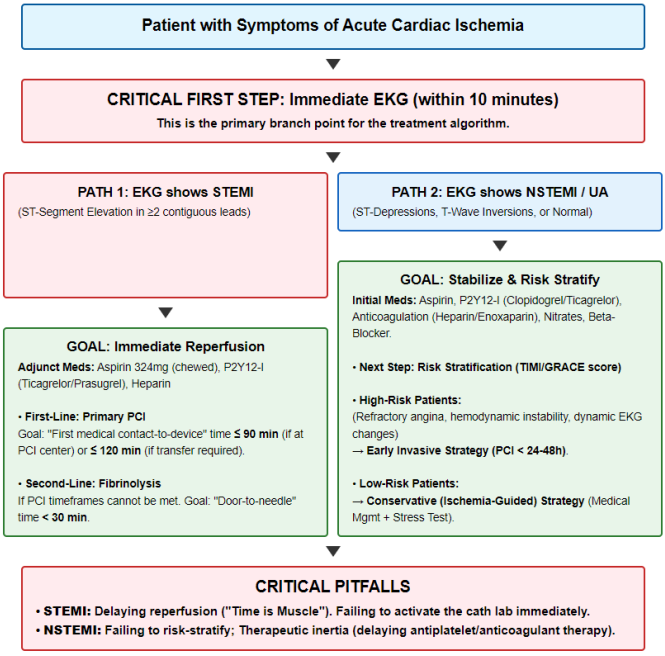

Figure 12.1: Acute Coronary Syndrome - Management Algorithm

Monitoring and Follow-Up

Acute monitoring is intensive and time-sensitive, beginning in the emergency department and continuing in a cardiac/coronary care unit (CCU).

1. **Continuous EKG (Telemetry):** This is the most critical monitoring tool. The primary goals are to watch for **life-threatening arrhythmias** (e.g., Ventricular Fibrillation or Tachycardia, which is the most common cause of death in early STEMI) and to monitor for dynamic ST-T wave changes, which would indicate re-occlusion or ongoing ischemia.
2. **Serial Cardiac Troponins:** Troponin levels are trended (e.g., at 0, 3, and 6 hours). The *peak* of the troponin level correlates with the *size* of the myocardial infarction. A

rapid rise and fall is the classic pattern.

3. **Serial 12-lead EKGs:** In addition to telemetry, full 12-lead EKGs are obtained serially (e.g., on arrival, post-intervention, and for any new chest pain) to assess the evolution of Q-waves, ST segments, and T-wave inversions.
4. **Post-Intervention Monitoring:** After percutaneous coronary intervention (PCI), monitoring is focused on the **catheter access site** (femoral or radial) for bleeding, hematoma, or pseudoaneurysm. Distal pulses and extremity perfusion must also be checked regularly.
5. **Hemodynamic Monitoring:** Vital signs, especially blood pressure and heart rate, are monitored closely to watch for signs of cardiogenic shock (hypotension) or heart failure (tachycardia, hypoxia).

Long-Term Management / Secondary Prevention

This is the cornerstone of post-ACS care. The goal is to prevent re-infarction, heart failure, and death.

1. **Dual Antiplatelet Therapy (DAPT):** This is non-negotiable. Patients receive **Aspirin** (lifelong) plus a **P2Y12 inhibitor** (e.g., clopidogrel, prasugrel, or ticagrelor). The P2Y12 inhibitor is continued for at least 12 months post-stent placement to prevent stent thrombosis.
2. **High-Intensity Statin:** All patients are discharged on a high-intensity statin (e.g., Atorvastatin 80mg, Rosuvastatin 40mg), regardless of their baseline cholesterol. The goal is plaque stabilization.
3. **Beta-Blocker:** Started within 24 hours (if no contraindications) and continued lifelong. Beta-blockers decrease myocardial oxygen demand and reduce the risk of post-MI arrhythmias.
4. **ACE Inhibitor / ARB:** These agents are started, especially in patients with a reduced ejection fraction (EF < 40%), hypertension, or diabetes, to prevent adverse cardiac remodeling and heart failure.
5. **Cardiac Rehabilitation:** A formal, monitored exercise and education program is a critical component of

recovery and secondary prevention.

6. **Risk Factor Modification:** Aggressive management of hypertension, diabetes, and hyperlipidemia, along with absolute smoking cessation.

Patient Counseling Points

1. **"This Was a Heart Attack":** "You had a heart attack. This means one of the arteries that supplies blood to your heart muscle became blocked by a blood clot. We were able to open that artery with a 'stent' (a small metal scaffold), and now the blood is flowing again."
2. **"The 'Holy Trinity' of Medications":** "You will be going home on three essential types of medicine that you must take for at least the next year, and some for life. 1) An **Aspirin** and a '**super-aspirin**' (like Plavix/Clopidogrel). These prevent your new stent from clotting off. Stopping these suddenly can be fatal. 2) A **statin**, which is a high-dose cholesterol pill that 'cools down' the inflammation in all your arteries. 3) A **beta-blocker**, which is a 'heart-protection' pill that lets your heart rest and heal."
3. **Chest Pain "Action Plan":** "If you get chest pain *like this again*: 1) Stop, sit down, and rest. 2) Take one nitroglycerin pill under your tongue. 3) If the pain is *not gone* in 5 minutes, **call emergency services**. Do not take a second pill. Do not drive yourself. Call emergency services."
4. **Cardiac Rehab:** "We will be enrolling you in 'cardiac rehab.' This is an exercise program with nurses and therapists that is the single best way to get your strength back and learn how to protect your heart."

Common Pitfalls in Management

The most critical pitfall in STEMI is delaying reperfusion. Any delay in obtaining the EKG or activating the cath lab is a systems failure. A common diagnostic error is missing a posterior MI (which presents as ST-depressions in V1-V3) or a "STEMI-equivalent." In NSTEMI, a common pitfall is therapeutic inertia, or failing to initiate dual antiplatelet therapy and

anticoagulation promptly.

Case Study

A 66-year-old male with diabetes presents with 2 hours of substernal chest pressure. His EKG is obtained in 5 minutes and shows **ST-depressions in leads V4-V6**. His initial Troponin is elevated at 1.8 ng/mL. He has ongoing chest pain despite 3 sublingual nitroglycerin tablets.

Discussion

The EKG (ST-depressions) and positive troponin confirm a **Non-ST-Elevation Myocardial Infarction (NSTEMI)**. The algorithm for NSTEMI is to stabilize and then risk-stratify. This patient's **refractory chest pain** places him in the "high-risk" category, which dictates the need for an "early invasive strategy."

Treatment Algorithm

1. **Diagnosis:** High-Risk NSTEMI.
2. **Algorithm (Medical Stabilization):**
    - **Aspirin:** 324 mg chewed.
    - **P2Y12 Inhibitor:** Loading dose of **Ticagrelor 180 mg**.
    - **Anticoagulation:** Start an **IV Heparin infusion**.
    - **Statin:** Administer **Atorvastatin 80 mg**.
    - **Beta-Blocker:** Start oral Metoprolol (once stable).
3. **Algorithm (Risk Stratification):** The patient is "High-Risk" due to refractory angina.
    - **Action:** The algorithm bypasses a conservative "ischemia-guided" strategy. He is scheduled for an **early invasive strategy (cardiac catheterization)**.

Outcome

The patient is admitted to the cardiac unit and undergoes angiography within 24 hours, which reveals a 90% blockage in his circumflex artery that is stented.

STROKE

Acute stroke is a neurological emergency caused by disrupted blood flow to the brain. The algorithm is entirely dependent on time from symptom onset and the differentiation between ischemic and hemorrhagic causes. The mantra is "Time is Brain."

Pathophysiology

Stroke is a "brain attack" caused by a sudden disruption of cerebral blood flow.

- **Ischemic Stroke (85%):** The pathophysiology is the acute occlusion of a cerebral artery, leading to a core of irreversible infarction (cell death) surrounded by a ring of salvageable, ischemic tissue called the **penumbra**. The entire treatment algorithm is focused on restoring blood flow to this penumbra before it, too, becomes infarcted. The "time is brain" mantra reflects that millions of neurons die for every minute of occlusion. The occlusion can be **thrombotic** (a clot forming on a local atherosclerotic plaque) or **embolic** (a clot traveling from a distant source, most commonly the heart in atrial fibrillation).
- **Hemorrhagic Stroke (15%):** The pathophysiology is *not* ischemia, but vessel rupture and bleeding.
    - **Intracerebral Hemorrhage (ICH):** A vessel *within* the brain parenchyma ruptures (often due to chronic hypertension). The damage is from the "mass effect" of the expanding hematoma, which compresses and destroys brain tissue.
    - **Subarachnoid Hemorrhage (SAH):** A vessel (most commonly a saccular "berry" aneurysm) ruptures *on the surface* of the brain, spilling blood into the subarachnoid space. This causes a sudden, severe "thunderclap" headache, and the primary neurologic damage is from cerebral vasospasm in the days following the bleed.