1. STEMI, N-STEMI, and UA-What is the difference and why does it matter?
2. Which evidence based, guideline supported medications should be prescribed after an MI, and what is a good way to remember this?
3. What are the differences between post-MI thrombosis prophylaxis agents and what factors determine the agent of choice?
4. What are the benefits of beta blocker therapy?
5. What medications may be used for ischemia?
6. Inhibition of the Renin-Angiotensin-Aldosterone system: what is the post-MI benefit, when should it be initiated and for how long?
7. What cholesterol goals should be reached and what agents should be used?
8. Smoking cessation: what pharmacologic options are available?
9. What hypertension and diabetes goals should be targeted post-MI, and how are these disease states managed?
10. How is post-MI depression addressed and managed?
11. Can NSAIDs be used in patients who have had an MI?
12. What yearly vaccination should all patients receive if they have had an MI?
13. Are there any EMMI programs that pertain to post-MI ambulatory therapy?

**Agents in this Review:**

- **Anti-platelet/Anticoagulation:**
  - Plavix (clopidogrel), Effient (prasugrel), Ticlid (ticlopidine), Coumadin (warfarin)

- **Cholesterol Agents**
  - Statins
    - Crestor® (rosuvastatin)
  - Nicotinic acid
    - Niaspan ER® (niacin)
  - Omega 3 Fatty Acids
    - Lovaza®
  - Fibric acid derivatives
    - gemfibrozil, fenofibrate

- **ACE Inhibitors**
  - Lisinopril, verapamil
  - Angiotensin Receptor Blockers
    - Atacand™ (candesartan), Diovan® (valsartan)

- **Aspirin**
- **Beta Blockers**
  - Metoprolol, atenolol, propranolol, carvedilol

- **Anti-Anginal**
  - Calcium Channel Blockers
    - Verapamil, diltiazem, amlodipine, nifedipine
  - Nitroglycerin

- **Smoking Cessation Agents**
  - Chantix® (varinicline)
  - Zyban® (bupropion)

**Highlights: The Quick Read Information**

1. Which of the following are useful acronyms for remembering the basics of ambulatory post-MI therapy?
   - a. SAAB
   - b. Convertible SAAB
   - c. ABCDE
   - d. All of the above
2. Evidence based literature indicates that ACE Inhibitors, but NOT ARBs, reduce the risk of secondary event post-MI.
   - a. True
   - b. False
3. All patients should be treated INDEFINITELY with the following medication post-MI:
   - a. Plavix®
   - b. Warfarin
   - c. Ibuprofen
   - d. Aspirin
4. The ATP III Guidelines propose the following lipid goal in patients who have suffered an MI:
   - a. HDL >60 mg/dL
   - b. LDL <100 mg/dL
   - c. HDL >40 mg/dL
   - d. LDL<130 mg/dL
5. If patients are maximized on beta blocker and ACE-I (or ARB) therapy, and still are not at goal for hypertension management, an agent from the following drug class should be added to achieve goal blood pressure:
   - a. CCB
   - b. Loop diuretic
   - c. Thiazide diuretic
   - d. Another beta blocker
6. The treatment of choice for musculoskeletal pain in patients post-MI is high dose ibuprofen.
   - a. True
   - b. False
7. Patients should receive which of the following vaccinations on a yearly basis after suffering from MI?
   - a. Tdap
   - b. Pneumovax
   - c. Influenza
   - d. Dtap
<table>
<thead>
<tr>
<th>Drug Class/Description (Class Representatives)</th>
<th>FDA Approved Indication for Post MI Therapy?</th>
<th>Benefit in Post MI</th>
<th>Monitoring Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antithrombotic (Aspirin)</td>
<td>Yes</td>
<td>Reduces risk of re-infarction/atherosclerotic events by 17%</td>
<td>Monitor for signs/symptoms of GI bleed</td>
</tr>
<tr>
<td>Anti-Platelet-Thienopyridines (Plavix®(clopidogrel); (Effient®(prasugrel); Ticlid ®(ticlopidine))</td>
<td>Yes (exception: Ticlid not FDA approved for post-MI therapy)</td>
<td>Reduces risk of stroke, MI and vascular events by 16%</td>
<td>Monitor for signs/symptoms of GI bleed</td>
</tr>
<tr>
<td>Anticoagulant (warfarin)</td>
<td>Yes</td>
<td>Reduces risk of MI, coronary artery thrombosis and stroke by 16.7%</td>
<td>Monitor for signs/symptoms of GI bleed; monitor INR to ensure target is reached</td>
</tr>
<tr>
<td>Beta Blocker (cardioselective: metoprolol, atenolol; non-selective: propranolol, carvedilol)</td>
<td>Yes</td>
<td>Reduces heart rate, blood pressure, ischemia. Reduces overall mortality by 26-35%</td>
<td>Heart rate, blood pressure, EKG</td>
</tr>
<tr>
<td>Calcium channel blocker (non-DHP: diltiazem, verapamil; DHP: nifedipine, amlodipine)</td>
<td>No</td>
<td>Reduces ischemia and angina in patients refractory to or unable to use beta blockers</td>
<td>Heart rate, blood pressure, EKG; non-DHP CCBs: edema</td>
</tr>
<tr>
<td>Nitrates (nitroglycerin)</td>
<td>Yes, for angina associated with ACS</td>
<td>Provides exogenous source of nitric oxide, leads to vasodilation and increases oxygen supply to heart</td>
<td>Frequency of administration; knowledge proper use and plan of action if experiencing angina</td>
</tr>
<tr>
<td>ACE Inhibitor (lisinopril, ramipril); ARB (Diovan®(valsartan))</td>
<td>ACE-I: No, ARB: Yes</td>
<td>Interferes with ventricular remodeling in patients with ventricular dysfunction, helps to prevent subsequent heart failure, reduces secondary events. Reduces overall mortality by approximately 20%</td>
<td>Serum potassium levels, blood pressure, kidney function</td>
</tr>
<tr>
<td>Statin (Crestor®(rosuvastatin), simvastatin)</td>
<td>Yes, for secondary event prophylaxis</td>
<td>Reduces LDL cholesterol, raises HDL cholesterol, linked with reduced risk of secondary event, possible anti-inflammatory properties. Reduces mortality by up to 42%</td>
<td>Serum cholesterol levels based on ATP III goals, LFTs</td>
</tr>
<tr>
<td>Additional cholesterol lowering agents: Nicotinic Acid (Niaspan ER®(niacin)); Omega 3 Fatty Acid (Lovaza®); Fibric acid derivative (gemfibrozil, fenofibrate)</td>
<td>No</td>
<td>Helps to achieve cholesterol goals (specifically HDL cholesterol and non-HDL cholesterol)</td>
<td>Serum cholesterol levels based on ATP III goals</td>
</tr>
<tr>
<td>Smoking cessation aids: Chantix® (varinicline), Zyban® (bupropion)</td>
<td>No</td>
<td>Smoking cessation aid</td>
<td>Efficacy-i.e. has patient successfully quit smoking without relapse</td>
</tr>
</tbody>
</table>

ACS = Acute Coronary Syndrome; ACE = Angiotensin Converting Enzyme; ARB = Angiotensin Receptor Blocker; DHP = dihydropyridine; Non-DHP = non-dihydropyridine
STEMI, N-STEMI, and UA—What is the difference and why does it matter? [2-3]

Described by the American College of Cardiology and the American Heart Association as acute coronary syndromes (ACS):

- Myocardial infarction (MI)
- MI with ST elevation (STEMI)
- MI without ST elevation (Non-STEMI/NSTEMI)
- Unstable angina (UA)

STEMI involves the elevation of the ST segment on an electrocardiogram, which is an indication of extensive myocardial injury or necrosis (see ECG figure, right). STEMI is the most serious and life threatening of the acute coronary syndromes.

UA and NSTEMI are less severe than STEMI. Patients experiencing UA and NSTEMI will exhibit the presence of ST segment depression or T-wave inversion on electrocardiogram, as opposed to ST elevation as seen with STEMI (see ECG figure, right). Markers of cardiac cell damage such as troponin or creatinine kinase, present in the serum of patients experiencing NSTEMI, are used to differentiate UA from NSTEMI.

Patients suffering from UA, NSTEMI and STEMI often present with similar symptoms, including but not limited to chest pain or discomfort, pain or discomfort radiating to the arms, jaw or back, and shortness of breath. It is important to differentiate between the types of acute coronary syndromes to determine the severity of the patient’s condition and the most effective therapeutic strategies. In terms of long term management, treatment may differ based on the type of acute coronary syndrome with which the patient presents and the course of management utilized in the acute phase. The focus of this review is post discharge, following the event, and does not address the acute phase.

**Which evidence based, guideline supported medications should be prescribed after an MI, and what is a good way to remember this? [2-3]**

Every patient who has suffered from STEMI or NSTEMI should be prescribed the following upon hospital discharge:

- Statin
- ACE Inhibitor (or ARB)
- Aspirin
- Beta Blocker

If patients have undergone stent placement while in the hospital, an anti-platelet agent (most often, clopidogrel) should be on board in addition to the medications mentioned above.

Some useful mnemonics to aid in remembering the important post-MI drug therapies include:

- **SAAB**: Statin; Aspirin; ACE/ARB; Beta Blocker – required in all patients, irrespective of stent placement
- **Convertible SAAB**: Clopidogrel; Statin; Aspirin; ACE/ARB; Beta Blocker – necessary additional agent of clopidogrel or effient
- **ABCDE**: Aspirin/Anti-platelet, Anti-angina, ACE-Inhibitor; Beta Blocker, Blood pressure; Cholesterol, Cigarettes; Diet, Diabetes; Education, Exercise – if also diabetic
What are the differences between post-MI thrombosis prophylaxis agents and what factors determine the agent of choice?[2-5]

The use of antiplatelet therapy post-MI has been shown to reduce the risk for secondary vascular events by 22%.

*Aspirin 75-162 mg daily should be used indefinitely for all post-MI patients.*

The use of aspirin post-MI has been shown to reduce the recurrence of vascular events including stroke and re-infarction by 17%. Aspirin provides anti-thrombotic effects through irreversibly inhibiting cyclooxygenase, an enzyme important in the production of thromboxane A$_2$. The subsequent inhibition thromboxane A$_2$ production decreases platelet aggregation and the activation of new platelets (see figure, right).

Initially, doses of aspirin differ based on the method of acute management, i.e. whether or not a stent has been placed and which type of stent is used (see Figures 1 & 2, below). For patients managed with a bare metal stent, aspirin 162-325 mg daily should be initiated and continued for at least 1 month, while patients with drug eluting stents should receive 162-325 mg daily for 3 months (sirolimus eluting) or 6 months (paclitaxel eluting) prior to initiating long term therapy with lower doses (75-162 mg per day). Bleeding risk should always be assessed before initiating higher doses of aspirin and before adding anti-platelet or anticoagulant agents.

Thienopyridines, including clopidogrel (Plavix®), prasugrel (Effient®) and ticlopidine (Ticlid®) inhibit platelet aggregation by preventing fibrinogen from binding to platelets via impaired activation of the GPIIb/IIIa complex. This occurs through the irreversible blockade of ADP binding to the platelet receptor (see figure, right). Using a thienopyridine post-MI in conjunction with aspirin therapy has been shown to significantly reduce the risk of secondary thrombotic events by 16.7%. A thienopyridine may also be used in place of aspirin if a patient has a true aspirin allergy (clopidogrel being the agent of choice at present). See Figures 1 & 2 for recommendations on indication, dose and duration of therapy when using a thienopyridine post MI. Ticlopidine is rarely used due to the risk of neutropenia and thrombocytopenia and the intensive monitoring necessary to detect these adverse events.

The anticoagulant effects of warfarin (Coumadin®, Jantoven®) result from inhibiting the production of vitamin K dependent coagulation factors II, VII, IX, X. Warfarin may be used in place of aspirin if patients have a true aspirin allergy and low bleeding risk. Warfarin is also used in conjunction with aspirin and/or clopidogrel for patients with certain indications. See Figures 1 & 2 for recommended duration, indications and target INR values for the use of warfarin post-MI. Patients should be counseled on avoiding foods containing high amounts of vitamin K, as ingesting foods rich in vitamin K may antagonize the effects of warfarin.
Figures 1 & 2 provide drug selection and dosing flow charts for post-MI anti-platelet therapy, as recommended by the ACC/AHA. Prasugrel (Effient®) is not included either figure; prasugrel 10 mg daily is equivalent to clopidogrel 75 mg daily.

**Indications for Anticoagulation:**
- Atrial fibrillation
- LV thrombus
- Cerebral emboli
- Pulmonary emboli
- Venous emboli
- Extensive regional wall motion abnormality

---

**Figure 1:** Long term anticoagulant/antithrombotic therapy after UA/NSTEMI at hospital discharge.

**Figure 2:** Long term anticoagulant therapy after STEMI at hospital discharge. (AHA/ACC guidelines)
**What are the benefits of beta blocker therapy?**[1-3, 6]

Beta-blockers, recommended for all patients post-MI, should be initiated as soon as possible as shown by the mortality benefits found in the ISIS-1, MIAMI and TIMI-II trials. Beta blockers should be continued for an indefinite period of time unless absolute contraindications exist or develop (including severe bradycardia, acute heart failure, cardiogenic shock, second or third degree AV block, active reactive airway disease, and hypotension). Studies have shown the benefits of beta-blocker therapy result from the reduction of heart rate, arterial pressure, and myocardial contractility. Additionally, beta blockers have been shown to prevent arrhythmias and cardiac arrest, leading to an overall reduction in mortality post-MI by up to 35%. Beta blockers may be used for their anti-ischemic activity which results from a reduction of cardiac work and myocardial oxygen demand.

In spite of well recognized relative contraindications to beta-blocker therapy, including respiratory conditions (i.e. mild asthma and COPD) and insulin dependent diabetes mellitus, the AHA/ACC guidelines advise the benefits of use outweigh the risks. For patients with respiratory dysfunction, a cardioselective beta blocker (atenolol, bisoprolol, metoprolol), is preferred. It should be noted that at higher doses, cardioselective beta blockers lose their beta-1 receptor selectivity. Patients with relative contraindications undergoing beta blocker therapy should be monitored closely for exacerbations of their conditions.

Side effects of beta blockers include drowsiness, dizziness, and possible CNS disturbances such as vivid dreams and hallucinations (especially with propranolol, which easily crosses the blood brain barrier). Atenolol is an option for patients experiencing significant CNS side effects, as it has less potential to cross the blood brain barrier. If discontinuation of beta blocker therapy is desired or required by the development of adverse effects or contraindications, it is necessary to slowly titrate the dose down to avoid reflex tachycardia. Beta blockers should not be stopped abruptly, especially if clonidine therapy is on board, due to the risk for rebound tachycardia.

**What medications may be used for ischemia?**[2-3]

As stated above, beta blockers are used for their anti-ischemic actions in addition to reducing post-MI mortality.

Calcium channel blockers (CCBs) may be used post MI for relief of angina and ischemia. CCBs may be used in conjunction with beta blocker therapy, or alone if beta blockers are not tolerated or contraindicated. CCBs classified as dihydropiridine (DHP) (amlodipine, nifedipine) exert dilatory effects on peripheral arteries, increasing oxygen supply to the heart with minimal direct effects on the sinus or AV node. Non-DHP CCBs (verapamil, diltiazem) primarily act on the sinus or AV node, thus decreasing contractility and oxygen demand. Non-DHP CCBs also possess some peripheral dilatory effects. DHP calcium channel blockers should be used in conjunction with beta blocker therapy if possible to reduce the risk of tachycardia. Short acting DHP calcium channel blockers (specifically nifedipine) are not recommended for monotherapy for ischemia post-MI, as adverse effects including flushing and peripheral edema may commonly occur.

Nitroglycerin helps to control ischemia by reducing myocardial oxygen demand through providing an exogenous supply of nitric oxide leading to vasodilation. Upon hospital discharge, patients having suffered from MI should be instructed on the proper use of nitroglycerin, outlined in the most recent guidelines: **one** sublingual tablet should be taken in response to chest pain or discomfort; if pain does not improve 5 minutes after nitroglycerin administration, **9-1-1** should be called. Please note: this is different from the previous recommendation **one tablet taken every 5 minutes up to three times prior to calling 9-1-1**. It should also be noted that nitroglycerin tablets are highly sensitive to light and may lose stability, thus decreasing efficacy, after
being exposed to light. It is recommended that a nitroglycerin prescription be refilled at least every 6 months regardless of frequency of use to ensure efficacy, and that the old tablets be disposed of.

**Inhibition of the Renin-Angiotensin-Aldosterone system: what is the post-MI benefit, when should it be initiated and for how long?**[2, 7]

Inhibition of the RAAS, as achieved with the use of an ACE inhibitor (i.e. lisinopril) or an ARB (i.e. valsartan), has been shown to reduce mortality in patients suffering from left ventricular dysfunction post-MI by approximately 20%. RAAS inhibition interferes with ventricular remodeling, augmenting the decline in left ventricular function over time. Use of an ACE inhibitor has also been shown to benefit patients without left ventricular dysfunction through the reduction of secondary cardiovascular events and all cause cardiovascular mortality by up to 22%.

All post-MI patients, excluding those with severe renal dysfunction or hyperkalemia, should be prescribed an ACE inhibitor for at least one month post-MI. All patients with left ventricular dysfunction, as well as those with diabetes and chronic kidney disease, should be prescribed an ACE inhibitor for long term use post-MI unless contraindications exist. If intolerance to ACE inhibitors exists or develops (most frequently, a dry nagging cough), an ARB may be used. Combination of an ACE inhibitor and an ARB may also be taken into consideration. The CHARM-Added trial showed a 4% reduction in cardiovascular endpoints including heart failure hospitalization and death when candesartan was used in combination with ACE inhibition versus ACE inhibition used alone.

**What cholesterol goals should be reached and what agents should be used?**[2-3, 8-10]

Serum cholesterol levels should be obtained within the first 24 hours after patients have presented with MI. Acute MI has been shown to lead to a rapid decline in lipid levels within the first 24 hours, which may last for 2 to 3 months. Therefore, it is important to obtain levels within the first 24 hours or after at least 90 days to ensure accuracy of values and avoid falsely low levels.

Patients who have suffered an MI can reduce the risk for cardiovascular events and secondary infarction by reaching the following lipid goals:

- LDL <100 mg/dL, preferably <70 mg/dL especially if diabetic or any other cardiovascular risks are also present
- HDL >40 mg/dL in men, >50 mg/dL in women
- Non-HDL (Non-HDL = TC-HDL) = <130 mg/dL

To augment medication therapy, patients should be counseled on adequate exercise regimens and proper dietary restrictions of daily saturated fat and cholesterol intake.

HMG-CoA reductase inhibitors (statins) effectively decrease LDL levels (18-55% reduction) while providing an increase in HDL (5-15% increase). In addition to lipid profile benefits, statins may also possess anti-inflammatory properties, potentially reducing cardiovascular risk. Studies have shown a reduction in C-reactive protein with use of statin therapy. Upon hospital discharge, all post-MI patients should be prescribed a statin for long term ambulatory therapy.

If patients have elevated triglyceride levels (i.e. greater than 500 mg/dL), a goal of non-HDL cholesterol of less than 130 mg/dL should be reached before LDL lowering therapy is initiated. Reductions in triglycerides may be achieved effectively with extended release niacin (Niaspan ER®) (20-50% reduction) and fibric acids
(gemfibrozil, fenofibrate) (20-50% reduction). Additionally, omega 3 fatty acids (OTC or prescription Lovaza®) are beneficial in decreasing triglycerides when used in doses of 3 to 4 grams per day.

Niacin, fibric acids, and omega 3 fatty acids may be used to augment statin therapy if HDL goals are not met with statins alone.

**Smoking cessation post-MI: what pharmacologic options are available?**[1-3, 11]

All patients should be encouraged to quit smoking after suffering an MI to prevent secondary events and decrease cardiovascular risk. Patients should be counseled on smoking cessation methods, and given information regarding the use of nicotine replacement products such as patches, gum or lozenges for the period of time in which they are trying to quit. In addition to nicotine replacement, pharmacologic options are available to curb cravings and help with the symptoms of nicotine withdrawal.

Sustained release bupropion, available generically and as Zyban®, may be used alone or in combination with nicotine replacement. Bupropion works to decrease the symptoms of nicotine withdrawal by inhibiting dopamine uptake. Bupropion should be started 1 to 2 weeks prior to the patient’s targeted quit day and may be continued for 7-12 weeks.

Chantix® (varenicline) is a nicotine receptor antagonist which blocks nicotine from binding. Additionally, Chantix® is a partial agonist, binding to the receptor to produce dopamine at much lower levels than the binding of nicotine. Chantix® is started one week prior to the quit date. Chantix® is titrated to the effective dose of 1 mg twice daily over a one week period and should be continued for 12 weeks.

**What hypertension and diabetes goals should be targeted post-MI, and how are these disease states managed?**[2-3, 9, 12]

Hypertension is a significant risk factor for cardiovascular disease according to JNC-7 and ATP III guidelines. Reducing blood pressure may lower the incidence of MI by 20-25%. The following blood pressure goals should be reached post-MI:

- All patients (without diabetes or renal disease): <140/90 mmHg
- Patients with diabetes or renal disease: <130/80 mmHg

Patients should be treated with a beta blocker and an ACE inhibitor or ARB as indicated for the post-MI benefits described previously in this review. If JNC-7 blood pressure goals are not met with lifestyle modifications in conjunction with optimal doses of these medications, a thiazide diuretic should be considered for further reduction in blood pressure. Patients should be monitored for symptoms of hypotension when adding anti-hypertensive agents.

Patients who have had an MI and co-morbid diabetes mellitus should aim for an HgA1C<7% as recommended by the American Diabetes Association. Diet, exercise, and pharmacologic therapies should be utilized to reach this goal. If necessary, insulin may be required for glycemic control. The guidelines proposed by the ADA and the American Association of Clinical Endocrinologists should be followed for glycemic goals and therapeutic options.
**How is post-MI depression addressed and managed?**[13]

Patients recovering from MI should be evaluated for depression using a standardized evaluation tool, as depression has been reported in up to 60% of patients who have had an MI. Post-MI depression has been associated with cardiac mortality. Furthermore, depression can negatively influence patient adherence to medication regimens.

If patients are diagnosed with depression after experiencing an MI, treatment with counseling should be initiated. Selective serotonin reuptake inhibitors are the pharmacologic agents of choice for patients with post-MI depression and have been shown to be safe and effective. Tricyclic antidepressants should be avoided in patients who are post-MI, as they may cause adverse cardiovascular events including severe hypertension.

**Can NSAIDs be used in patients who have had an MI?**[3, 14]

The use of NSAIDs post-MI should be avoided if possible. NSAIDs can contribute to increased blood pressure and may increase the likelihood of secondary MI or cardiovascular event.

For musculoskeletal pain, it is recommended that patients use acetaminophen or aspirin. Tramadol or narcotic anagelsics may be used short term for more severe pain. If the latter medications are not tolerated or ineffective, non-COX-2 selective NSAIDs may be used at the lowest dose possible. COX-2 selective NSAIDs (i.e. celecoxib) should be avoided. Patients using NSAIDs post MI should be evaluated regularly for hypertension, edema, and GI bleeding. A proton pump inhibitor may be considered to prevent GI complications.

**What vaccinations should all patients receive after having been diagnosed with a MI?**[2-3]

Every patient with cardiovascular disease should receive an influenza vaccination yearly unless contraindicated (i.e. history of hypersensitivity to influenza vaccine or egg allergy). The pneumonia vaccine (PPSV) should also be administered once, as indicated for patients ages 2-65 who have a history of heart disease. A second dose should be administered after the age of 65 years old if greater than 5 years have passed since the original vaccination.

**Are there any EMMITM programs that pertain to post-MI ambulatory therapy?**

1. Angiogram with possible angioplasty -- 18967864172
2. Coronary Bypass Surgery (CABG) -- 18053869287
3. Taking Warfarin -- 15150038741
4. Coronary Artery Disease -- 14143128081

**Summary: The highlights of the ACC/AHA Post-STEMI guidelines for therapy:**[3]

<table>
<thead>
<tr>
<th>Goals</th>
<th>Intervention Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smoking:</strong></td>
<td>Assess tobacco use. Strongly encourage patient and family to stop smoking and to avoid secondhand smoke. Provide counseling, pharmacological therapy (including nicotine replacement and bupropion), and formal smoking cessation programs as appropriate.</td>
</tr>
<tr>
<td><strong>Blood pressure control:</strong></td>
<td>If blood pressure is 120/80 mm Hg or greater:</td>
</tr>
<tr>
<td>Goal</td>
<td>-Initiate lifestyle modification (weight control, physical activity, alcohol moderation, moderate sodium restriction, and emphasis on fruits, vegetables, and low-fat dairy products) in all patients.</td>
</tr>
<tr>
<td>Less than 140/90 mm Hg or chronic kidney disease or diabetes</td>
<td>If blood pressure is 140/90 mm Hg or greater or 130/80 mm Hg or greater for individuals with chronic kidney disease or diabetes:</td>
</tr>
</tbody>
</table>
Lipid management: (TG less than 200 mg/dL)

Start dietary therapy in all patients (less than 7% of total calories as saturated fat and less than 200 mg/d cholesterol). Promote physical activity and weight management. Encourage increased consumption of omega-3 fatty acids.

Primary goal:
LDL-C substantially less than 100 mg/dL

Assess fasting lipid profile in all patients, preferably within 24 hours of STEMI. Add drug therapy according to the following guide. (See Section 7.12.2.)

- LDL-C less than 100 mg/dL (baseline or on treatment):
- LDL-C greater than or equal to 100 mg/dL (baseline or on treatment):

- Statins should be used to lower LDL-C.
- Intensify LDL-C–lowering therapy with drug treatment, giving preference to statins.

Lipid management: (TG 200 mg/dL or greater)

If TGs are greater than or equal to 150 mg/dL or HDL-C is less than 40 mg/dL:
- Emphasize weight management and physical activity. Advise smoking cessation

Primary goal If TG is 200-499 mg/dL:
less than 130 mg/dL

If TG is 200-499 mg/dL:
- Non–HDL-C* substantially lower

If TG is greater than or equal to 500 mg/dL:
- Consider fibrate or niacin‡ before LDL-C–lowering therapy.
- Consider omega-3 fatty acids as adjunct for high TG.

Physical activity:

Assess risk, preferably with exercise test, to guide prescription.

Minimum goal:
30 minutes 3 to 4 days per week
Optimal daily
Encourage minimum of 30 to 60 minutes of activity, preferably daily but at least 3 or 4 times weekly (walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening, household work). Cardiac rehabilitation programs are recommended for patients with STEMI, particularly those with multiple modifiable risk factors and/or those moderate- to high-risk patients in whom supervised exercise training is warranted.

Weight management:

Calculate BMI and measure waist circumference as part of evaluation. Monitor response of BMI and waist circumference to therapy.

Goal
BMI 18.5-24.9 kg/m²
Waist circumference:
Women: less than 35 in.
Men: less than 40 in.

If waist circumference is greater than or equal to 35 inches in women or greater than or equal to 40 inches in men, initiate lifestyle changes and treatment strategies for metabolic syndrome.

Diabetes management:

Appropriate hypoglycemic therapy to achieve near-normal fasting plasma glucose, as indicated by HbA1c. Treatment of other risk factors (e.g., physical activity, weight management, blood pressure, and cholesterol management).

Goal
HbA1C less than 7%

Antiplatelet agents/ anticoagulants

Start and continue indefinitely aspirin 75 to 162 mg/d if not contraindicated. Consider clopidogrel 75 mg/d or warfarin if aspirin is contraindicated. Manage warfarin to INR 2.5 to 3.5 in post-STEMI patients when clinically indicated or for those not able to take aspirin or clopidogrel.

Renin-angiotensin- Aldosterone system blockers

ACE inhibitors in all patients indefinitely; start early in stable high-risk patients (anterior MI, previous MI, Killip class greater than or equal to II [Sí: gallop, rales, radiographic CHF], LVEF less than 0.40).

Angiotensin receptor blockers in patients who are intolerant of ACE inhibitors and with either clinical or radiological signs of heart failure or LVEF less than 0.40.

Aldosterone blockade in patients without significant renal dysfunction or hyperkalemia who are already receiving therapeutic doses of an ACE inhibitor, have an LVEF less than or equal to 0.40, and have either diabetes or heart failure.

Beta-Blockers

Start in all patients. Continue indefinitely. Observe usual contraindications.

STEMI = ST-elevation myocardial infarction; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; BMI = body mass index; INR = international normalized ratio; ACE = angiotensin converting enzyme; MI = myocardial infarction; CHF = congestive heart failure; LVEF = left ventricular ejection fraction. Non–HDL-C = total cholesterol minus HDL-C. †Treat to a goal of non–HDL-C substantially less than 130 mg/dL. ‡Dietary-supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should be used only if approved and monitored by a physician. CREATININE should be less than or equal to 2.5 mg/dL in men or less than or equal to 2.0 mg/dL in women. Potassium should be less than 5.0 mEq/L. Modified with permission from Smith et al. Circulation 2001;104:1577-9 (68).
References:
Answer Key

1. d
2. b
3. d
4. b
5. c
6. b
7. c