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Chapter 17

EARLY MANAGEMENT OF ACUTE CORONARY SYNDROMES

The management of patients with acute myocardial ischemia and infarction is one of the few practices in critical care medicine that can save lives on a continued basis, but only when appropriate interventions are used early (often within hours after the initial contact with the patient). Those interventions are described in this chapter using information from practice guidelines published by the American College of Cardiology and American Heart Association (ACC/ AHA). These guidelines are listed in the bibliography at the end of the chapter 0-3).

ACUTE CORONARY SYNDROMES

Acute coronary syndromes (ACS) are conditions characterized by the sudden onset of coronary insufficiency as a result of thrombotic occlusion of one or more coronary arteries. Three such conditions are identified: STsegment elevation myocardial infarction (STEM!), non-ST-segment elevation myocardial infarction (non-STEM!), and unstable angina *(VA).* The first condition (STEM!) is the result of complete and sustained thrombotic coronary occlusion, while the last two conditions (non-STEMI and *VA)* lre the result of either partial thrombotic coronary occlusion or transient complete occlusion with spontaneous revascularization 0-3).

The seminal event in all these conditions is coronary thrombosis (see Figure 17.1). The nidus for thrombus formation is rupture of an athero;clerotic plaque (4), which exposes the blood to thrombogenic lipids and eads to activation of platelets and clotting factors. The trigger for plaque iisruption is not known, but liquefaction caused by local inflammation

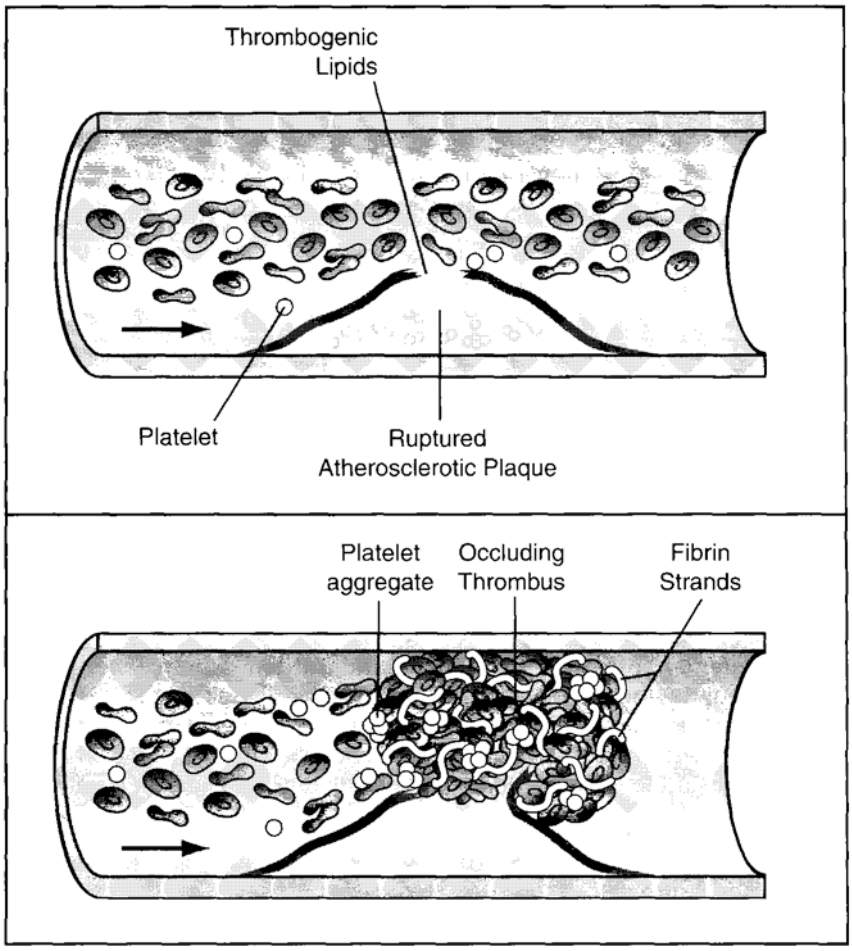

FIGURE 17.1 Illustration showing the pathogenesis of acute coronary syndromes. Rupture of an atherosclerotic plaque leads to activation of platelets and clotting factors (upper panel) and results in the formation of an occlusive thrombus (lower panel).

and inflammatory mediators is believed to be involved (5). Hydraulic stresses may also play a role because plaques that rupture are usually located at branch points or bends in the arterial tree (3,6).

The discovery that coronary thrombosis is responsible for the tissue injury in acute myocardial infarction led to the adoption of several therapeutic measures designed to limit thrombus formation and alleviate thrombotic obstruction. These measures include antiplatelet therapy (aspirin, platelet glycoprotein inhibitors), anticoagulant therapy (heparin), chemical dissolution of clots (fibrinolytic agents), and mechanical disruption of clots (coronary angioplasty). This chapter contains a description of each of these measures and when they should be used.

ROUTINE MEASURES

The initial management of patients with acute coronary syndromes includes a series of routine measures, which are shown in Figure 17.2. These measures are used in all patients and are often initiated during or immediately after the initial patient contact.

Relieving Chest Pain

Relieving chest pain is one of the immediate goals of management in acute coronary syndromes. Pain relief not only promotes well-being, it also helps alleviate unwanted cardiac stimulation from anxiety-induced adrenergic hyperactivity.

Nitroglycerin

Nitroglycerin (0.4 mg sublingual tablets or aerosol spray) is given for up to three doses (each 5 minutes apart) to relieve chest pain. If the pain subsides, intravenous nitroglycerin can be started for continued pain relief (see Chapter 16, Table 16.4, for recommended dose rates for intravenous nitroglycerin). If the chest pain persists after 3 doses of nitroglycerin, immediate administration of morphine is indicated.

Intravenous nitroglycerin is also indicated for persistent or recurrent chest pain due to unstable angina and for acute coronary syndromes associated with hypertension or pulmonary congestion 0-3). In patients with right ventricular infarction, intravenous nitroglycerin should be avoided or used with extreme caution because of the risk of hypotension (aggressive volume loading is needed in this situation to counteract the venodilating effects of nitroglycerin). Finally, nitroglycerin should NOT be used in patients who have taken a phosphodiesterase inhibitor for erectile dysfunction within the past 24 hours (longer for some preparations) because of the high risk for hypotension.

Morphine

Morphine is the drug of choice for chest pain that is refractory to nitroglycerin 0-3). The initial dose is usually 4 mg, given by slow intravenous push (e.g., 1 mg/minute), and this can be repeated every 5 to 10 minutes if necessary. Morphine administration may be followed by a decrease in blood pressure. This is usually the result of a decrease in sympathetic nervous system activity and is not a pathologic process. A drop in blood pressure to hypotensive levels usually indicates hypovolemia and can be corrected by volume infusion (2). Pressor agents should NEVER be used to correct morphine-induced decreases in blood pressure.

Antiplatelet Therapy

Aspirin

Chewable aspirin in a dose of 162 to 325 mg should be given to all patients with ACS who have not taken aspirin prior to presentation 0-3).

Aspirin causes irreversible inhibition of platelet aggregation by inhibiting thromboxane production (7), and aspirin therapy (either alone or in combination with thrombolytic therapy) has been shown to reduce mortality and decrease the rate of re-infarction (8,9). The decrease in short-term (30 day) mortality attributed to aspirin alone is about 2 to 3% (8), which means that for every 100 patients with ACS, there are 2 to 3 fewer deaths attributed to aspirin.

The initial dose of aspirin is usually given as soon as possible after presentation, even though there is no evidence that the beneficial effects of aspirin in ACS are time-dependent (2). Non-enteric-coated aspirin is preferred because of enhanced buccal absorption. The initial aspirin dose 062 to 325 mg) should be followed by a daily dose of 75 to 162 mg, which is continued indefinitely (2). For patients who are unable to receive aspirin because of aspirin allergy or recent GI hemorrhage, alternative therapy with the antiplatelet agents in the next section is advised.

Thienopyridines

The thienopyridines are antiplatelet agents that irreversibly block surface receptors involved in ADP-induced platelet aggregation (7). This mechanism of action differs from that of aspirin, which means that the antiplatelet effects of aspirin and the thienopyridines are additive. The anti-platelet activity of the thienopyridines requires drug activation in the liver, so these drugs are not recommended in patients with liver failure.

There are 2 clinically available drugs in this class: clopidogrel (Plavix) and ticlodipine (Ticlid). Clopidogrel seems to be preferred because of fewer side effects (7). The recommended dose of clopidogrel in ACS is 300 mg initially, followed by 75 mg daily (2). Although clopidogrel is currently recommended as a substitute for aspirin, one large study has shown that combined therapy with clopidogrel and aspirin in ACS is associated with a lower mortality than aspirin therapy alone (O). Combined anti-platelet therapy with clopidogrel and aspirin is already a standard practice following stent placement (2), and more widespread use of combined therapy in ACS is likely to occur.

Beta-Receptor Blockade

The benefit of Beta-receptor antagonists in ACS is based on their ability to reduce cardiac work and decrease myocardial energy requirements. Early institution of beta-blocker therapy is recommended for all patients with ACS who do not have a contraindication to Beta-receptor blockade 0-3). In addition to the usual contraindications (i.e., severe sinus bradycardia with heart rate < 40 bpm, second- or third-degree heart block, decompensated systolic heart failure, hypotension, and reactive airways disease), Beta-receptor antagonists are not advised for cocaine-induced myocardial infarction because of the potential for aggravated coronary vasospasm from unopposed a-receptor activity (11).

Oral beta blocker therapy is suitable for most cases of ACS (1-3). Intravenous therapy is more appropriate for patients with hypertension

or troublesome tachyarrhythmias. The agents used most often in clinical trials of ACS are atenolol (Tenormin) and metoprolol (Lopressor). Both are selective B1-receptor antagonists that can be given orally or intravenously. Lopressor is used in our hospital, and the first dose is usually given within an hour after ACS is first suspected. The oral and intravenous dosing regimens for metoprolol are shown below (12).

Oral regimen: Start with intravenous dose of 2.5 to 5 mg and repeat every 5 minutes if needed to a total dose of 10 mg. Fifteen minutes after the last IV dose, start oral therapy with 50 mg every 6 hours for 48 hours, then 100 mg BID.

IV regimen: Add 5 mg metoprolol to 50 mL DsW and infuse over 15 to 30 minutes every 6 hours.

Note that the oral regimen begins by giving the drug intravenously.

This speeds up the response and reduces the time required to achieve steady-state drug levels in the body.

Angiotensin-Converting-Enzyme Inhibition Angiotensin-converting-enzyme (ACE) inhibitors are vasodilators that reduce cardiac work and decrease myocardial energy requirements. They may also have an inhibitory effect on the cardiac remodeling that occurs after coronary artery reperfusion and contributes to post-MI heart failure. Oral therapy with ACE inhibitors started in the first 24 hours after onset of acute coronary syndromes has the following effects.

Provides a significant survival benefit in patients with anterior MI and acute MI associated with symptomatic heart failure, left ventricular dysfunction (LV ejection fraction <0.40), and tachycardia (2).

Provides a modest survival benefit (one life saved for every 200 patients treated) if used in all patients with acute coronary syndromes (13).

Based on these observations, oral therapy with ACE inhibitors is *probably* indicated in all patients with acute coronary syndromes and is *definitely* indicated in the conditions identified in statement #1. Contraindications to ACE inhibitor therapy include hypotension, renal failure (creatinine >2.5 mg/ dL), and bilateral renal artery stenosis.

Drug Administration

Any ACE inhibitor can be used, and the first dose should be given within 24 hours after symptom onset (2). To minimize the risk of hypotension (which can be very damaging in the setting of an acute M!), only oral therapy is recommended, and the starting dose is usually reduced and then increased over the next 48 hours. One example of an ACE inhibitor regimen that has proven effective in a large clinical trial (14) is shown in Figure 17.2.

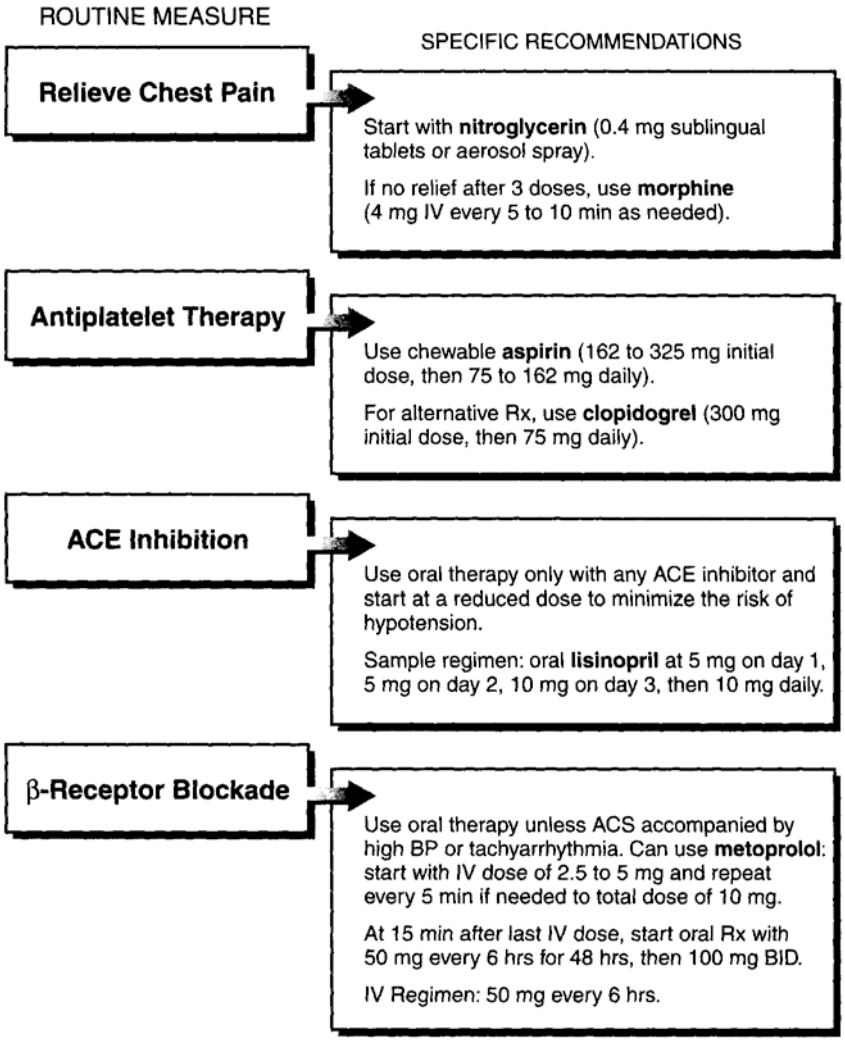

FIGURE 17.2 Routine measures in the early management of acute coronary syndromes. Abbreviations: ACS = acute coronary syndrome, ACE = angiotensin-converting-enzyme.

Angiotensin-Receptor Blockers

Clinical trials have shown that angiotensin-receptor blockers (ARB) produce a survival benefit equivalent to ACE inhibitors in acute MI associated with left ventricular dysfunction (LV ejection fraction <0.40) or symptomatic heart failure (15). As a result, ARBs are considered as a suitable alternative in patients with acute MI complicated by LV dysfunction or heart failure who do not tolerate ACE inhibitors (2). One example of a successful ARB regimen is oral valsartan, 20 mg initially,

then gradually increase to a final dose of 160 mg twice daily by the end of the hospitalization (15). The contraindications for ARBs are the same as those mentioned previously for ACE inhibitors.

REPERFUSION THERAPY

In the early 1980s, two distinct modes of therapy were introduced to alleviate thrombotic obstruction and restore patency in occluded coronary arteries. One involves the pharmacologic dissolution of blood clots using drugs that stimulate fibrinolysis (thrombolytic therapy), and the other involves the mechanical disruption of clots using specialized balloontipped catheters (coronary angioplasty). These forms of *reperfusioll therapy* have had a profound impact on the early management of patients with acute coronary syndromes and are largely responsible for the improved outcomes (reduced morbidity and mortality) reported in recent years. This section describes each type of reperfusion therapy and the relative risks and benefits.

Thrombolytic Therapy

The evaluation of drugs that stimulate fibrinolysis began immediately after the discovery (in 1980) that transmural myocardial infarction was the result of occlusive coronary thrombosis. The first fibrinolytic agent studied was streptokinase, which was shown to produce effective clot lysis when given directly into the affected coronary artery, and later was shown to be equally effective when infused into a peripheral vein (16). In 1986, the first clinical trial of intravenous streptokinase in acute MI was completed, and the results showed fewer deaths in the patients who received thrombolytic therapy (7).

The Initial ElectrocardioBram

The survival benefit of thrombolytic therapy in acute coronary syndromes is determined by the findings on the initial electrocardiogram (ECG). This is demonstrated in Figure 17.3, which shows the pooled results of 9 clinical trials comparing thrombolytic therapy (with different fibrinolytic agents) to placebo in patients with acute coronary syndromes (8). The survival benefit of thrombolytic therapy is greatest in patients who present with new-onset left bundle branch block and ST-segment elevation in the anterior precordial leads, while there is no survival benefit in patients with ST-segment depression on the initial ECG.

Timing

The effect of thrombolytic therapy on survival is also determined by the time elapsed from the onset of chest pain to initiation of therapy. This is demonstrated in Figure 17.4, which includes data from patients who showed a survival benefit with thrombolytic therapy in the pooled studies depicted in Figure 17.3. This data shows that the survival benefit of thrombolytic therapy is greatest when therapy is initiated in the first

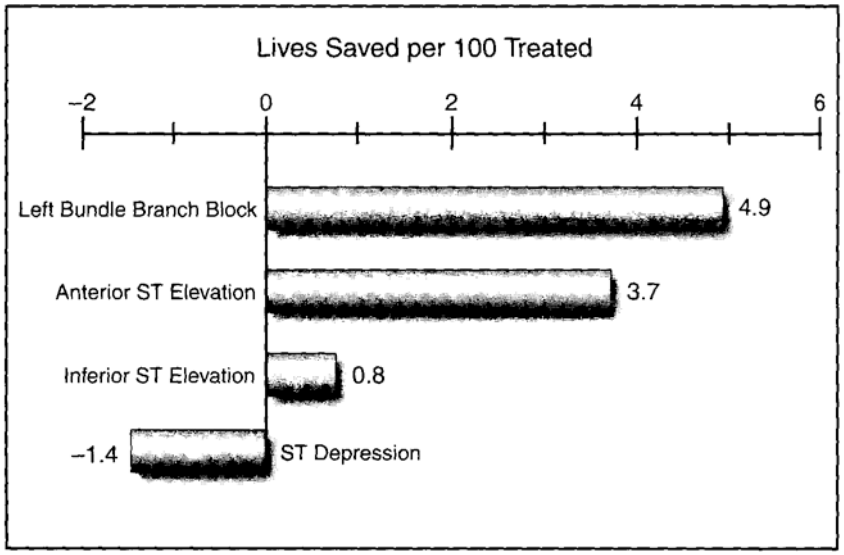

FIGURE 17.3 The survival benefit of thrombolytic therapy in relation to abnormal patterns on the electrocardiogram. (From Reference 18.)

few hours after the onset of chest pain. Thereafter, the survival benefit declines steadily with time and is negligible or lost when the delay to initiation of therapy exceeds 12 hours.

The data in Figure 17.4 highlights the most important feature of thrombolytic therapy:

Time lost is lives lost.

To ensure timely initiation of thrombolytic therapy, emergency rooms in the United States have adopted the following guidelines (2):

When a patient with sudden onset of chest pain enters the emergency room, an electrocardiogram should be performed and interpreted within the next 10 minutes (door-to-ECG time <10 minutes).

Thrombolytic therapy, if indicated, should be started within 30 minutes after the patient enters the emergency room (door-toneedle time <30 minutes).

Selection of Candidates

The observations in Figures 17.3 and 17.4 are the basis for the criteria used to select candidates for thrombolytic therapy, which are shown in Table 17.1 (2). Patients are candidates for thrombolytic therapy if coronary angioplasty is not immediately available and all of the following conditions are present: (1) chest pain for at least 30 minutes but less than 12 hours; (2) a 12-lead ECG that shows ST elevation of 0.1 mV O IDm) or more in two contiguous leads, or a new left bundle branch block; (3) the absence of hypotension or heart failure; and (4) the absence of a

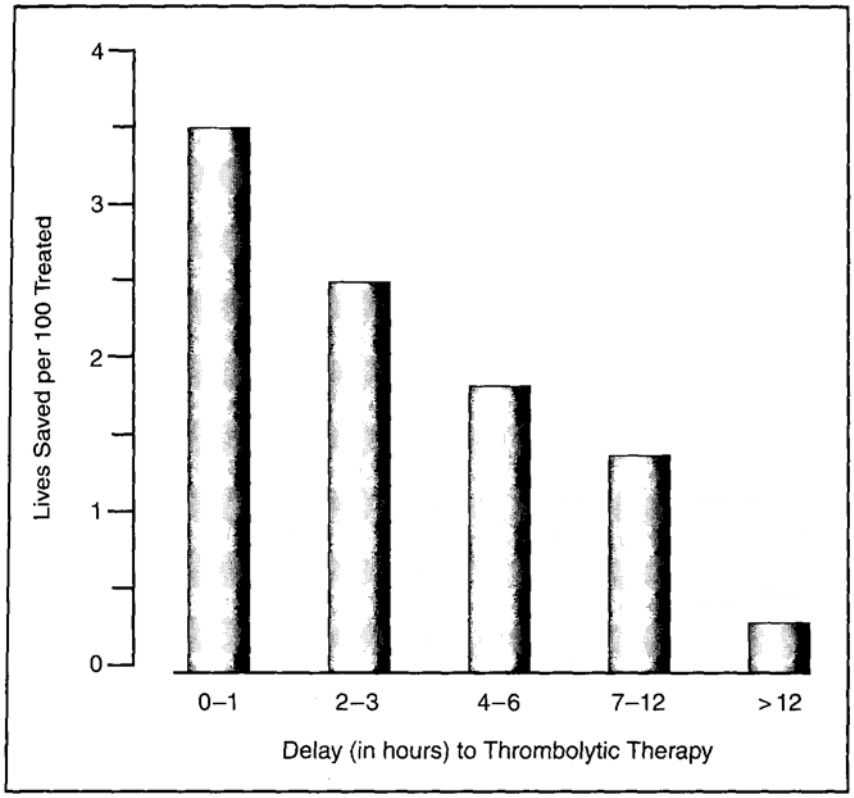

FIGURE 17.4 The survival benefit of thrombolytic therapy as a function of the time elapsed from the onset of chest pain to the initiation of therapy. (From Reference 18.)

contraindication to thrombolytic therapy that would create an unacceptable risk of bleeding (see Table 17.2). The role of coronary angioplasty in these conditions is described later in the chapter.

The most recent ACC/ AHA practice guidelines (2) include true posterior MI as a condition that might benefit from thrombolytic therapy if treated within 12 hours of symptom onset. This condition should be suspected when the ECG shows ST-segment depression with upright T waves in precordial leads VI through V_4 (19). The discovery of STsegment elevation in additional precordial leads V 7 - V 9 will help confirm the diagnosis of posterior wall MI.

Fibrinolytic Agents

The available fibrinolytic agents and recommended dosing regimens for acute MI are shown in Table 17.3. All of these agents act by converting plasminogen to plasmin, which then breaks fibrin strands into smaller subunits. Some (streptokinase) act on circulating plasminogen and produce a systemic lytic state, while others (alteplase, reteplase,

TABLE 17.1 Reperfusion Strategy for Patients with ST-Elevation Myocardial Infarction (STEMI)

For adults with sudden onset of chest pain, consider the following questions:

	Yes	No
1. Did the chest pain begin more than 30 minutes ago but less than 12 hours ago?		
2. Does the ECG show either of the following abnormalities?		
a) ST elevation 2: 0.1 mV (1 mm) in at least 2 contiguous precordial leads or 2 adjacent limb leads.		
b) A new, or presumably new, left bundle branch block.		

If the answer is Yes to each of the above, perform Coronary Angioplasty if immediately available. Otherwise, proceed to questions below.

	Yes	No
3. Is the patient hypotensive?		
4. Is there evidence of decompensated heart failure?		
5. Is there an unacceptable risk of bleeding as a result of any of the contraindications listed in Table 17.2?		

If the answer is No to each of the above, begin Thrombolytic Therapy immediately.

and tenecteplase) act only on plasminogen that is bound to fibrin and produce clot-specific lysis. The site of action (clot-specific versus systemic) has little clinical relevance.

Streptokinase is a bacterial protein that was the first thrombolytic agent evaluated in clinical trials and the first to show improved survival in patients with acute, ST-elevation MI (17). Although it is the least expensive thrombolytic agent, it is also the least favored because it acts as an antigen and produces fever (in 20 to 40% of cases), allergic reactions (in 5% of cases), and accumulation of neutralizing antibodies with repeated use (20).

Alteplase (tissue plasminogen activator or tPA) is a molecular clone of an endogenous plasminogen activator that replaced streptokinase in popularity because it does not produce allergic reactions, and because one large study published in 1993 (the GUSTO trial) showed improved survival with alteplase compared to streptokinase (21). Alteplase has been the favored lytic agent for the past 10 to 15 years, but it may be replaced by the newer lytic agents given as bolus doses, which are easier to administer (22).

Reteplase (rPA) is a molecular variant of tPA that is given in 2 bolus doses 30 minutes apart. It is easier to give than tPA and produces more rapid clot lysis (23). However, clinical trials comparing reteplase and alteplase have shown no difference in mortality rate (24).

Tenecteplase (TNK-tPA) is another variant of tPA that is given as a single bolus. It is the most clot-specific fibrinolytic agent and produces

Table 17.2 Contra indications to Thrombolytic Therapy

Absolute Contraindications:
Active bleeding other than menses
Malignant intracranial neoplasm (primary or metastatic)
Cerebrovascular anomaly (e.g., AV malformation)
Suspected aortic dissection
Ischemic stroke within 3 months (but not within 3 hours)
Prior history of intracranial hemorrhage
Significant closed-head or facial trauma within 3 months
Relative Contra indications:
Systolic BP > 180 mm Hg or diastolic BP > 110 mm Hg
Active bleeding within the past 4 weeks
Noncompressible vascular punctures
Major surgery within the past 3 weeks
Traumatic or prolonged (> 10 min) CPR
Ischemic stroke over 3 months ago
Dementia
Active peptic ulcer disease
Pregnancy
Current use of anticoagulants (the higher the INR, the greater the risk of bleeding)

From the practice guidelines in Reference 2.

TABLE 17.3 Thrombolytic Agents

Agent	Dose	Comments
Streptokinase (SK)	1.5 million units IV over 60 min	Allergic reactions and buildup of neutralizing antibodies with repeated use.
Alteplase (tPA)	15 mg IV bolus, + 0.75 mg/kg over 30 min + 0.5 mg/kg over 60 min (90 min total)	Most frequently used lytic agent.
Reteplase (rPA)	10 units as IV bolus and repeat in 30 min	Bolus doses are easier to give and produce more rapid clot lysis than tPA.
Tenecteplase (TNK)	IV bolus of 30 mg for BW <60 kg, 35 mg for BW = 60-69 kg, 40 mg for BW = 70-79 kg, 45 mg for BW = 80-89 kg, 50 mg for BW 2:90 kg	Most clot-specific and rapidly acting lytic agent. Easiest to use because of single bolus dose.

the most rapid clot lysis (22). However, neither of these attributes offers a clinical advantage because clinical trials comparing tenecteplase and alteplase have shown no difference in the incidence of life-threatening bleeding and no difference in mortality rate (25).
In summary, excluding streptokinase because of its unwanted antigen effects, the available fibrinolytic agents are equivalent in terms of survival benefit and risk of bleeding. Bolus fibrinolytic therapy (with reteplase or tenecteplase) is likely to gain in popularity simply because it is easier. Overall, it seems that the important issue in thrombolytic therapy is not which agent to use; but how quickly to use it.

Complications
The most feared complication of thrombolytic therapy is intracerebral hemorrhage, which is reported in 0.5 to 1 % of cases (22). This may be more common with alteplase when compared to streptokinase (21), but there is no difference between alteplase, reteplase, and tenecteplase in the risk of intracerebral bleeding (22). Extracranial bleeding that requires blood transfusions occurs in 5 to 15% of patients, regardless of the lytic agent used (26). There is no correlation between the risk of hemorrhage and the degree of clot-specificity of the fibrinolytic agents.
The hemorrhagic complications of thrombolytic therapy are the result of systemic fibrinolysis with depletion of circulating fibrinogen levels. If necessary, cryoprecipitate (10 to 15 bags) can be used to achieve a serum fibrinogen level of 1 giL (26). If bleeding persists, fresh frozen plasma (up to 6 units) can be administered, followed by platelet infusions (10 bags) if needed. The use of antifibrinolytic agents such as epsilonamino caproic acid (5 grams given over 15 to 30 minutes) is discouraged for all but the most serious and refractory cases of bleeding because these agents can produce extensive thrombosis (26).
Reocclusion
The benefit of thrombolytic therapy is limited by the risk of reocclusion following clot lysis, which is reported in up to 25% of cases (2,26). This may be a natural consequence of clot dissolution because the exposed thrombin (which had been enmeshed in the thrombus) has prothrombotic effects via platelet activation and an increased rate of thrombin formation (27). To counteract this process, antithrombotic therapy with heparin and antiplatelet agents is given in combination with thrombolytic therapy. This adjunctive therapy is described later in the chapter.

Coronary Angioplasty
The use of balloon-tipped catheters to open occluded arteries (balloon angioplasty) was adapted for use in the coronary arteries in 1977 by a Swiss physician named Andreas Gruntzig. This procedure *(percutaneous coronary angioplasty)* was adopted in the 1980s as an alternative to thrombolytic therapy for patients with acute myocardial infarction. As a result of improved techniques and the introduction of stents to keep arteries open, coronary angioplasty is now the preferred method of reperfusion therapy for patients with occlusive coronary thrombosis.

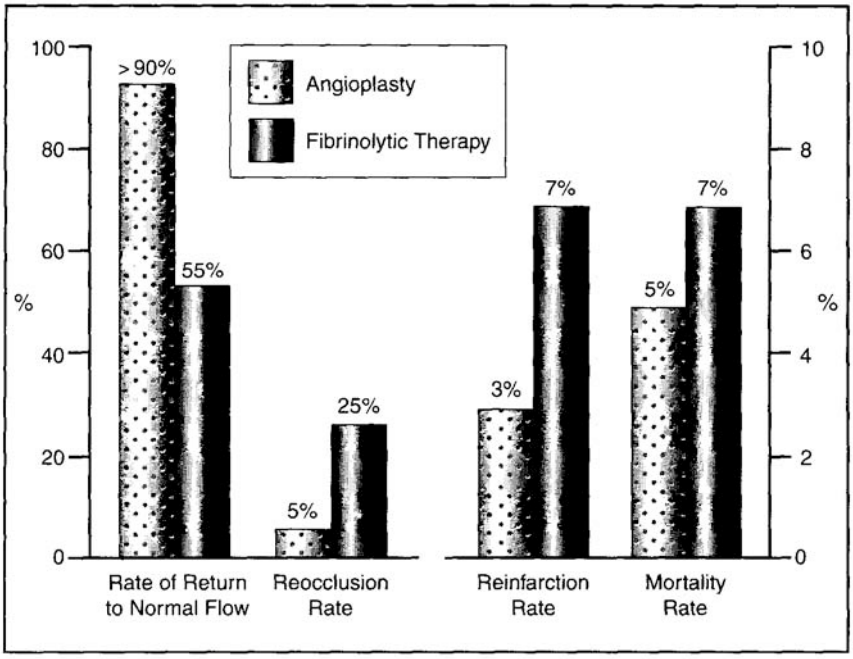

FIGURE 17.5 Comparative effects of primary angioplasty and thrombolytic therapy on vascular events (graph on the left) and clinical outcomes (graph on the right) in patients with ST-elevation myocardial infarction. (Data from References 28–30).

Angioplasty vs. Lytic Therapy

Several clinical trials have compared coronary angioplasty and thrombolytic therapy in patients with ST-elevation MI who present within 12 hours of symptom onset (28-30). The comparative effects on vascular events and clinical outcomes are shown in Figure 17.5. The bar graph on the left (depicting vascular events) shows that angioplasty restores normal flow in infarct-related arteries much more frequently and has a much lower rate of reocclusion than thrombolytic therapy. The bar graph on the right (depicting clinical outcomes), which represents the pooled results of 23 studies (29), shows that angioplasty has a lower reinfarction rate and a lower mortality rate than thrombolytic therapy. These differences in mortality and reinfarction (2% and 4%, respectively) indicate that, for every 100 patients treated with angioplasty instead of thrombolytic therapy, there are 2 fewer deaths and 4 fewer (non-fatal) recurrent infarctions.

Timing

The beneficial effects of coronary angioplasty, like those of thrombolytic therapy, are time-dependent. This is demonstrated in Figure 17.6, which shows the mortality rate (at 30 days) for patients with acute MI who underwent angioplasty at different times after arriving at the hospital (31). The time from arrival at the hospital to the angioplasty procedure is

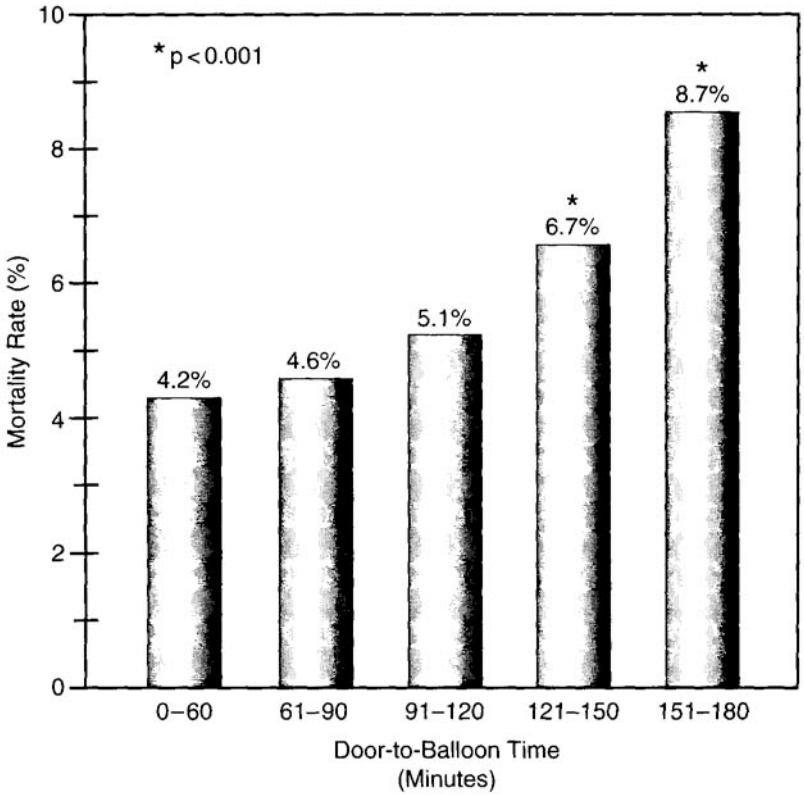

FIGURE 17.6 Mortality rate in patients treated with primary angioplasty as a function of the time period between arrival to the hospital and the angioplasty (door-to-balloon time). Asterisk indicates a significant difference in mortality rate compared to the initial time period (0–60 min). (From Reference 31.)

shown as the "door-to-balloon time" on the horizontal axis of the graph. As demonstrated, mortality rate rises steadily with increasing delays to angioplasty, and the increased mortality becomes significant when the delay to angioplasty exceeds 2 hours. This observation is the basis for the recommendation that angioplasty should be performed within 90 minutes after the patient arrives in the emergency department (2). This, of course, only applies to the use of angioplasty for patients with ST-elevation MI who present within 12 hours of symptom onset.

Interhospital Transfer

The major limitation of coronary angioplasty is availability. Less than 25% of hospitals in the United States have facilities for coronary angioplasty, and in Europe, fewer than 10% of hospitals have this capability (32). One solution for the "have-nots" is transfer to a hospital that can perform angioplasty. Clinical studies have shown that interhospital

transfer for coronary angioplasty, if completed in one to two hours, can take advantage of the benefits derived from angioplasty and improve clinical outcomes (2,33). The current recommendations for interhospital transfer are stated below (2):
If the symptom duration is less than 3 hours, thrombolytic therapy is recommended unless interhospital transfer will not add more than a one-hour delay to treatment.
If the symptom duration is longer than 3 hours, interhospital transfer for angioplasty is recommended. The total door-toballoon time, including the transfer time, should be close to 90 minutes to achieve the optimal benefit of angioplasty.

ADJUNCTS TO REPERFUSION THERAPY

Antithrombotic therapy with antiplatelet agents and heparin has a proven benefit when used with or without reperfusion therapy. When added to reperfusion therapy (particularly thrombolytic therapy), antithrombotic therapy can help to prevent reocclusion and recurrent infarction.
Heparin
Anticoagulation with heparin is beneficial in most patients with acute coronary syndromes, and it may be particularly advantageous in patients who receive fibrinolytic agents to reduce the risk of reocclusion from the prothrombotic effects of thrombin exposed during clot dissolution (described earlier). The effectiveness of heparin in acute coronary syndromes can differ for the two heparin preparations: unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH). (See Chapter 5 for a description of these heparin preparations.) The following is a summary of the *ACCI* AHA recommendations for the use of UFH and LMWH in acute coronary syndromes.
LMWH is preferred to UFH for patients with unstable angina (UA) and non-ST- segment elevation myocardial infarction (nonSTEM!) (3,34,35).
UFH and LMWH are considered equivalent in patients with STsegment elevation myocardial infarction (STEM!) who do not undergo reperfusion therapy (2).
Despite promising results with LMWH (36), UFH is recommended for patients with STEMI who undergo reperfusion therapy with fibrinolytic agents or angioplasty (2).

Recommended Dose Regimens
The *ACCI* AHA recommendations for heparin dosing in acute coronary syndromes is shown below (1-3). Enoxaparin is used as the LMWH because this agent has been studied most in acute coronary syndromes.

TABLE 17.4 Enoxaparin Dosage Based on Renal Function

GFR* (mLJmin)	SC Dose (mg/kg q 12h)	GFR* (mL/min)	SC Dose (mg/kg q 12h)
>= 80	1. 0	40-49	0.6
70-79	0.9	30-39	0.5
60-69	0.8	20-29	0.4
50-59	0.7	10-19	0.3

*GRF (mL/min) = (140-age) x wt (kg)/72 x serum creatinine (mgldL). For multiply GFR by 0.85.
From Green B, et al. Dosing strategy for enoxaparin in patients with renal presenting with acute coronary syndromes. Br J Clin Pharmacol

Enoxaparin: Start with intravenous bolus of 40 mg, and follow with subcutaneous injection of 1 mg/kg twice daily for 5 days (3). Reduced dosing is necessary in renal insufficiency (see Table 17.4)
UFH: Start with intravenous bolus of 60-70 Units/kg, and follow with infusion of 12-15 Units/kg/hr. Adjust infusion to maintain activated partial thromboplastin time (aPTT) at 1.5 to 2 times control (3).
UFH with fibrinolytic agents: Start with intravenous bolus of 60 Units/kg, and follow with infusion of 12 Units/kg/hr. Adjust infusion to maintain aPTT at 1.5 to 2 times control (2).
UFH with angioplasty: Start with intravenous bolus of 70-100 Units/ kg, and follow with infusion of 12-15 Units/kg/hr. Adjust infusion to maintain the aPTT at 1.5 to 2 times control (2).
When using UFH, the aPTI should be checked 3 hours after starting the infusion and 6 hours after each dose adjustment. In addition, platelet levels should be checked daily in all patients receiving heparin (because of the risk of heparin-induced thrombocytopenia, described in Chapter 37).

Aspirin

Aspirin is used as an anti platelet agent in virtually everyone with acute coronary syndromes (except those with aspirin allergy). When used in combination with fibrinolytic agents, aspirin reduces the rate of reinfarction (9). The recommended dosing regimen for aspirin is described earlier in the chapter.

Platelet Glycoprotein Inhibitors

The newest group of drugs added to the war chest for acute coronary syndromes are potent antiplatelet agents that block platelet receptors involved in platelet aggregation. When platelets are activated, specialized glycoproteins on the platelet surface (called IIb/IIIa receptors)

Table 17.5 Platelet Glycoprotein Inhibitors

Agent	Commercial Preparation	Dose
Abciximab	ReoPro	0.25 mg/kg as IV bolus followed by infusion of 0.125 μg/kg/min (maximum 10 μg/min).
Eptifibatide	Integrilin	180 μg/kg as IV bolus followed by infusion of 2 μg/kg/min for up to 96 hrs. For serum creatinine of 2-4 mg/dL, reduce first dose to 130 μg/kg and reduce infusion rate to 0.5 μg/kg/min.*
Tirofiban	Aggrastat	0.4 μg/kg/min for 30 min followed by infusion of 0.1 μg/kg/min. For creatinine clearance < 30 mUmin, reduce both dose rates by 50%.'

'Manufacturer's recommendation.

change configuration and begin to bind fibrinogen. When fibrinogen molecules bind to adjacent platelets, platelet aggregation occurs. The *platelet glycoprotein (IIb/IIIa) inhibitors* bind to the surface receptors on platelets and prevent the binding of fibrinogen. The result is inhibition of platelet aggregation. The IIb/IIIa receptors are the final common pathway for platelet aggregation, so the IIb/IIIa inhibitors are the most powerful antiplatelet agents available (and are sometimes called ***If*** su peraspirins[lf]).

Drug Administration

There are three platelet glycoprotein inhibitors available for clinical use: abciximab (ReoPro), eptifibatide (Integrilin), and tirofiban (Aggrastat). All three are given by intravenous infusion, and the dosing regimen for each agent is shown in Table 17.5.

Abciximab (try to pronounce it!) is a monoclonal antibody that is the most potent, most expensive, and longest-acting drug in the group. After discontinuing abciximab, bleeding times can take 12 hours to normalize (7), and this prolonged action can be a disadvantage when emergency bypass surgery is contemplated.

Eptifibatide (a synthetic peptide) and tirofiban (a tyrosine derivative) are short-acting agents that are cleared by the kidneys. After discontinuing these drugs, bleeding times return to normal in 15 minutes for eptifibatide and 4 hours for tirofiban (7). Dose adjustments in renal insufficiency are recommended for both drugs, and these dose adjustments are included in Table 17.5. Excess dosing in renal insufficiency will result in drug accumulation and increased risk of bleeding. Dose adjustments in renal failure are not necessary for abciximab because it is an antibody and is presumably cleared by the reticuloendothelial system.

Indications

Platelet glycoprotein inhibitors are primarily used in patients with unstable angina (UA) and nonST-elevation myocardial infarction (non-STEM!) when the following conditions are present (3):

When coronary angioplasty is planned in the next 24 to 48 hours.

When there is evidence of continuing myocardial ischemia (e.g., recurrent angina or angina at rest with transient ST-segment changes).

When there are risk factors for recurrent ischemic events, such as age >75 years, heart failure, new or worsening mitral regurgitation, markedly elevated cardiac troponin levels, and cardiogenic shock (3).

The greatest benefits occur when these agents are used in conjunction with angioplasty (1-3,38). Abciximab is recommended only when angioplasty is planned and seems a favorite of cardiologists. In the catheterization lab, the initial bolus of abciximab is given after the arterial sheath is placed, and the abciximab infusion is continued for 12 hours after the procedure (39).

Platelet glycoprotein inhibitors are gaining popularity in patients with ST-elevation MI (STEM!), and are usually given in combination with angioplasty or thrombolytic therapy (2,3,37). In the future, expect platelet glycoprotein inhibitors to be combined with low-dose fibrinolytic agents as a prelude to coronary angioplasty (so-called "facilitated angioplasty").

Adverse Effects

The major risk with platelet glycoprotein inhibitors is bleeding. The incidence of bleeding from these agents is difficult to assess because they are often used in combination with aspirin and heparin. Most of the bleeding is mucocutaneous, and intracranial hemorrhage is not a risk with these agents (7,38).

Thrombocytopenia is reported in up to 2% of patients who receive abciximab and is more common with repeated use of the drug (38).

Active bleeding is an absolute contraindication to platelet glycoprotein inhibitors. Relative contra indications include major surgery within the past 3 months, stroke in the past 6 months, systolic blood pressure> 180 mm Hg or diastolic pressure> 110 mm Hg, and severe thrombocytopenia (38).

EARLY COMPLICATIONS

The appearance of decompensated heart failure and cardiogenic shock in the first few days after an acute MI is an ominous sign and usually indicates a mechanical problem like acute mitral regurgitation or cardiac pump failure. Echocardiography is usually needed to uncover the problem, but the mortality in these conditions is high despite timely interventions.

Mechanical Complications

Mechanical complications are usually the result of transmural (ST-elevation) MI. All are serious, and all require prompt action.

Acute mitral regurgitation is the result of papillary muscle rupture and presents with the sudden onset of pulmonary edema and the characteristic holosystolic murmur radiating to the axilla. The pulmonary artery occlusion pressure should show prominent V waves, but this can be a non-specific finding. Diagnosis is by echocardiography, and arterial vasodilators (e.g., hydralazine) are used to relieve pulmonary edema pending surgery. Mortality is 70% without surgery and 40% with surgery (39).

Ventricular septal rupture can occur anytime in the first 5 days after acute MI. The diagnosis can be elusive without cardiac ultrasound. There is a step-up in O_2 saturation from right atrial to pulmonary artery blood, but this is rarely measured. Initial management involves vasodilator (e.g., nitroglycerin) infusions and the intraaortic balloon pump if needed. Mortality is 90% without surgery and 20% to 50% with surgery (2).

Ventricular free wall rupture occurs in up to 6% of cases of STEMI and is more common with anterior MI, fibrinolytic or steroid therapy, and advanced age (2). The first signs of trouble are usually return of chest pains and new ST-segment abnormalities on the ECG. Accumulation of blood in the pericardium often leads to rapid deterioration and cardiovascular collapse from pericardial tamponade. Diagnosis is made by cardiac ultrasound (if time permits), and prompt pericardiocentesis combined with aggressive volume resuscitation is required for hemodynamic support. Immediate surgery is the only course of action, but fewer than half of the patients survive despite surgery (2).

Pump Failure

About 15% of cases of acute MI result in cardiac pump failure and cardiogenic shock (40). Management involves hemodynamic support (usually with intraaortic balloon counterpulsation) followed by reperfusion using coronary angioplasty or coronary bypass surgery. Despite the best intentions, the mortality in this situation is 60 to 80% (40).

Hemodynamic Support

Hemodynamic support should be designed to augment cardiac output without increasing myocardial oxygen consumption. Table 17.6 shows the effects of hemodynamic support on the determinants of myocardial O_2 consumption (preload, contractility, afterload, and heart rate) in decompensated heart failure and cardiogenic shock. As judged by the net effect on myocardial O_2 consumption, vasodilator therapy is superior to dobutamine in heart failure, and the intra-aortic balloon pump (IABP) is superior to dopamine in cardiogenic shock. (See Chapter 14 for more information on the treatment of cardiac pump failure.)

TABLE 17.6 Hemodynamic Support and Myocardial 02 Consumption

	Heart Failure		Cardiogenic Shock	
Parameter	Vasodilators	Dobutamine	IASP	Dopamine
Preload	decrease	decrease	decrease	+
Contractility	–	+	–	+
Afterload	decrease	decrease	decrease	+
Heart rate	–	+	–	+
Net effect on myocardial $V0_2$	decrease	+	decrease	+

IASP = Intra-aortic balloon pump, $V0_2$ = oxygen

Emergency Revascularization

The ACC/ AHA guidelines recommend coronary angioplasty when cardiogenic shock appears within 36 hours of acute MI and when the angioplasty can be performed within 18 hours of the onset of shock (2). Coronary artery bypass surgery is considered if the cardiac catheterization reveals multivessel disease that is not amenable to angioplasty or disease involving the left main coronary artery (2).

Arrhythmias

Disturbances of cardiac rhythm are common after acute MI and are not suppressed by prophylactic use of lidocaine (2). The management of serious arrhythmias is described in the next chapter.

Acute Aortic Dissection

Aortic dissection is included in this chapter because the clinical presentation can be mistaken as an acute coronary syndrome, and the condition is often fatal if missed.

Clinical Presentation

The most common complaint is abrupt onset of chest pain. The pain is often sharp and is described as "ripping or tearing" (mimicking the underlying process) in about 50% of cases (41). Radiation to the jaws and arms is uncommon. The pain can subside spontaneously for hours to days (41,42), and this can be a source of missed diagnoses. The return of the pain after a pain-free interval is often a sign of impending aortic rupture.

Clinical Findings

Hypertension and aortic insufficiency are each present in about 50% of cases, and hypotension is reported in 25% of cases (41,42). Dissection can cause obstruction of the left subclavian artery, leading to blood pressure

differences in the arms, but this finding can be absent in up to 85% of cases (42). Obstruction involving other arteries in the chest can lead to stroke and coronary insufficiency.

Diagnosis
Mediastinal widening on chest x-ray (present in 60% of cases) often raises suspicion for dissection (42). However, the diagnosis requires one of four imaging modalities (43): magnetic resonance imaging (MR!) (sensitivity and specificity, 98%), transesophageal echocardiography (sensitivity, 98%; specificity, 77%), contrast-enhanced computed tomography (sensitivity, 94%; specificity, 87%), and aortography (sensitivity, 88%; specificity, 94%). Thus MRI is the diagnostic modality of choice for aortic dissection, but the immediate availability of MRI is limited in some hospitals, and helical CT and transesophageal ultrasound are high-yield alternatives to MRI. Aortography is the least sensitive but provides valuable information for the operating surgeon.

Management
Acute dissection in the ascending aorta is a surgical emergency. Prompt control of hypertension is advantageous prior to surgery to reduce the risk of aortic rupture. Increased flow rates in the aorta create shear forces that promote further dissection, so blood pressure reduction should not be accompanied by increased cardiac output. This can be accomplished with the drug regimens shown in Table 17.7 (41,42). One regimen uses a vasodilator (nitroprusside) infusion combined with a Beta-blocker (esmolol) infusion. The Beta-blocker is given first to block the vasodilator-induced increase in cardiac output. Esmolol is used as the Beta-blocker because it has a short duration of action (9 minutes) and is easy to titrate. It can also be stopped just prior to surgery without the risk of residual cardiac suppression during surgery. Single-drug therapy with a combined a and Beta-receptor antagonist Oabetalol} is also effective and is easier to use than the combination drug regimen.

TABLE 17.7 Treating Hypertension in Aortic Dissection

Combined therapy with beta~blocker and vasodilator:	
Start with esmolol:	500 µJkg IV bolus and follow with 50 *µg/kg/min.* Increase infusion by 25 *µg/kg/min* every 5 min until heart rate 60-80 bpm. Maximum dose rate is *200 µg/kg/min.*
Add nitroprusside:	Start infusion at 0.2 *µglkg/min* and titrate upward to desired effect. See the nitroprusside dosage chart in Table 16.6.
Monotherapy with combined a-fJ receptor antagonist:	
Labetalol:	20 mg IV over 2 min, then infuse 1-2 *mg/min* to desired effect and stop infusion. Maximum cumulative dose is 300 mg.

A FINAL WORD

The discovery that acute myocardial infarction is the result of blood clots that obstruct coronary arteries has one important implication (besides the improved therapeutic approach to myocardial infarction) that seems overlooked. It disputes the traditional teaching that myocardial infarction is the result of a generalized imbalance between myocardial 02 delivery and O_2 consumption. This distinction is important because the 02-imbalance paradigm is the basis for the overzealous use of oxygen breathing and blood transfusions in patients with coronary artery disease. Blood clots (from ruptured atherosclerotic plaques) cause heart attacks, not hypoxia or anemia. If you have ever wondered why heart attacks are uncommon in patients with progressive shock and multiorgan failure, you now have the answer.

REFERENCES

Chapter 18

TACHYARRHYTHMIAS

Acute arrhythmias are the gremlins of the ICU because they pop up unexpectedly, create some havoc, and are often gone in a flash. The arrhythmias that create the most havoc are the ones that produce rapid heart rates: the *tachyarrhythmias.* This chapter describes the acute management of tachyarrhythmias using clinical practice guidelines developed by consensus groups in the United States and Europe. The published guidelines are listed in the bibliography at the end of chapter 0-3), along with Internet addresses where they can be downloaded at no cost.

CLASSIFICATION

Tachycardias (heart rate above 100 beats/minute) can be the result of *ilncreased aultomaticity* in pacemaker cells (e.g., sinus tachycardias), *triggered activity* (e.g., ectopic impulses), or a process known as *re-entry,* where a triggered impulse encounters a pathway that blocks propagation in the forward direction but allows the impulse to pass in the return (retrograde) direction. Such retrograde transmission allows a triggered impulse to propagate continually, creating a self-sustaining tachycardia. Re-entry is the most common cause of clinically significant tachycardias.

Tachycardias are classified according to the site of impulse generation in relation to the atrioventricular (AV) conduction system. *Supraventricular tachycardias* (SVT) originate above the AV conduction system and have a normal QRS duration (~0.12 seconds), while *ventricular tachycardias* (VT) originate below the AV conduction system and have a prolonged QRS duration (> 0.12 seconds). Each type of tachycardia can then be subdivided according to regularity of the rhythm (Le., the regularity of the R-R interval on the ECG). The classification of tachycardias based on the QRS duration and the regularity of the R-R interval is shown in Figure 18.I.

Narrow QRS Complex Tachycardia

The tachycardias associated with a QRS duration of 0.12 seconds or less include sinus tachycardia, atrial tachycardia, AV nodal re-entrant tachycardia (also called paroxysmal supraventricular tachycardia), atrial flutter, and atrial fibrillation.

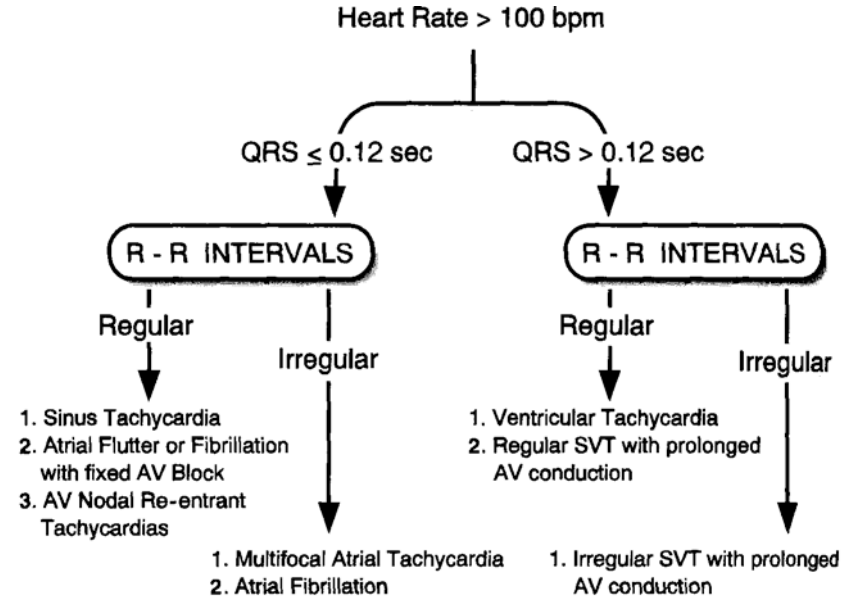

FIGURE 18.1 Classification of the tachycardias based on the QRS duration and the regularity of the R–R interval on the electrocardiogram.

Regular Rhythm

If the R-R intervals are uniform in length (indicating a regular rhythm), the possible arrhythmias include sinus tachycardia, AV nodal re-entrant tachycardia, or atrial flutter with a fixed (2:1, 3:1) AV block. The atrial activity can help to identify each of these rhythms. Uniform P waves with a fixed P-R interval is characteristic of sinus tachycardia; the absence of P waves suggests an AV nodal re-entrant tachycardia (see Figure 18.2), and sawtooth waves are characteristic of atrial flutter.

If the R-R intervals are not uniform in length (indicating an irregular rhythm), the most likely arrhythmias are multifocal atrial tachycardia

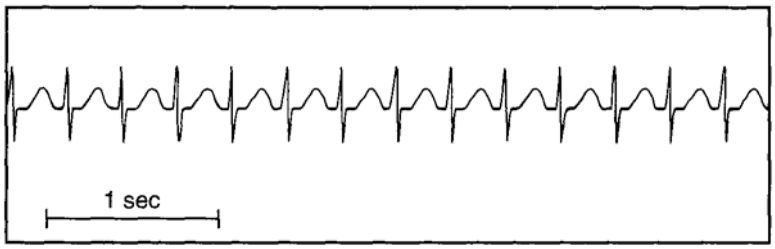

FIGURE 18.2 AV nodal re-entrant tachycardia, which is also called a paroxysmal supraventricular tachycardia. Note the absence of P waves, which are hidden in the QRS complexes.

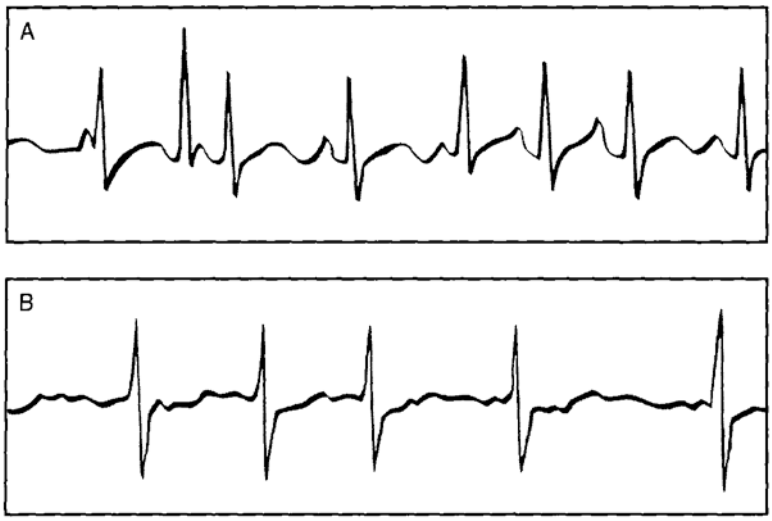

FIGURE 18.3 Multifocal atrial tachycardia (panel A) and atrial fibrillation (panel B). (From the CD ROM that accompanies Critical care nursing: A holistic approach. Philadelphia: Lippincott Williams & Wilkins, 2005.)

and atrial fibrillation. Once again, the atrial activity helps to identify each of these rhythms. Multifocal atrial tachycardia has multiple P wave morphologies and nonuniform PR intervals (Panel A, Figure 18.3), while atrial fibrillation has fibrillatory atrial waves and no identifiable P waves (Panel B, Figure 18.3). The rhythm in atrial fibrillation is highly irregular and is sometimes described as an "irregularly irregular" rhythm (which indicates that no two R-R intervals have the same length).

Wide Q..RS *Complex Tachycardia*

A tachycardia with a QRS duration> 0.12 seconds is either ventricular tachycardia (VT) or SVT with aberrant (prolonged) AV conduction. VT is characterized by a regular rhythm and the presence of AV dissociation, while sVT with aberrant conduction can have a regular or irregular rhythm (depending on the rhythm of the inciting sVT). These two arrhythmias can look remarkably similar, as presented later in the chapter.

SINUS TACHYCARDIA

Increased automaticity in the pacemaker cells of the sinoatrial node produces a regular, narrow-complex tachycardia with a gradual onset and rate of 100 to 140 beats/minute. The ECG shows uniform P waves and a fixed P-R interval. Sinus tachycardia can also be the result of re-entry into the sinus node. This variant sinus tachycardia has an abrupt onset,

but is otherwise indistinguishable from the increased automaticity type of sinus tachycardia.

Management

Sinus tachycardia is often a response to a systemic illness. It is usually well tolerated (cardiac filling is usually not compromised until the heart rate rises above 180 beats/minute) (4) and does not require primary treatment. The primary goal of management is to identify and treat the associated illness. Possible sources of sinus tachycardia in the IeU include systemic infection and inflammation, hypovolemia, and adrenergic drugs.

The major indication for slowing a sinus tachycardia is the presence of myocardial ischemia or infarction. In this situation, beta-receptor antagonists can be used to slow the heart rate. (See Chapter 17, Figure 17.2, for an effective beta -blocker regimen in acute coronary syndromes.) Because these agents also depress ventricular function, they are not recommended for sinus tachycardia associated with systolic heart failure.

ATRIAL FIBRILLATION

Atrial fibrillation (A F) is the most common cardiac arrhythmia in the general population. (Atrial flutter is considered here as a more organized form of atrial fibrillation rather than a distinct arrhythmia). An estimated 2.2 million adults (or 1 % of the adult population) have AF (3). Most are elderly (median age, 75 years) and have either ischemic heart disease, valvular disease, or cardiomyopathy. Contrary to popular perception, few have hyperactive thyroid disease (5). About 15% of patients with AF are relatively young (less than 60 years of age) and have no predisposing conditions (3): this condition is known as lone atrial fibrillation.

Postoperative AF

Postoperative AF is reported in 30 to 40% of patients undergoing coronary artery bypass surgery, and 60% of patients undergoing valve surgery, and usually appears in the first 4 postoperative days (6). The etiology is unclear, but risk factors include valvular surgery, advanced age, and failure to resume beta -blocker therapy after surgery. beta -blockers are preferred for rate control of AF in this setting (7). This arrhythmia is usually self-limited, and more than 90% of patients will convert to sinus rhythm within 6 to 8 weeks (3).

Adverse Consequences

Contraction of the atria is responsible for 25% of the ventricular enddiastolic volume (preload) in the normal heart (4). This atrial contribution to ventricular filling is lost in AF. There is little consequence in the normal heart, but cardiac output can be impaired in patients with diastolic dysfunction due to a noncompliant or stiff ventricle (where ventricular

filling volumes are already reduced). This effect is pronounced at rapid heart rates (because of the reduced time for ventricular filling).
The other notable complication of AF is thrombus formation in the left atrium, which can embolize to the cerebral circulation and produce an ischemic stroke. Atrial thrombosis can be demonstrated in 15% of patients who have AF for longer than 3 days (8), and about 6% of patients with chronic AF and certain risk factors (see later) suffer an embolic stroke each year without adequate anticoagulation (3,8). The indications for anticoagulation in AF are presented later.

Management Strategies
The acute management of AF involves 3 strategies: (1) cardioversion to terminate the arrhythmia and restore normal sinus rhythm, (2) druginduced reduction of the ventricular rate, and (3) anticoagulation to prevent thromboembolism. The following presentation is organized according to these strategies.
Cardioversion
eardioversion can be accomplished by applying electric shocks (electrical cardioversion) or administering an antiarrhythmic agent (pharmacological cardioversion).

Electrical Cardioversion
Immediate cardioversion using direct-current (DC) electric shocks is indicated for cases of AF associated with severe hemodynamic compromise (hypotension or decompensated heart failure). This procedure is both painful and anxiety-provoking and, if tolerated, pre-medication with a benzodiazepine (e.g., midazolam) and/or an opiate (morphine or fentanyl) is indicated. The individual shocks should be synchronized to the R wave of the QRS complex to prevent electrical stimulation during the vulnerable period of ventricular repolarization, which usually coincides with the peak of the T wave (3). The following protocols are recommended (3).
For monophasic shocks, begin with 200 joules (J) for atrial fibrillation and 50 J for atrial flutter. If additional shocks are needed, increase the energy level of each successive shock by 100 J until a maximum shock strength of 400 J is reached. Wait at least one minute between shocks to minimize the risk of cardiac ischemia.
For biphasic shocks (which are the waveforms used in many of the newer defibrillator machines), use only half the energy recommended for monophasic shocks.
These regimens should result in successful cardioversion in about 90% of cases (3).

Pharmacolgic Cardioversion

Acute pharmacologic cardioversion may be appropriate for first episodes of AF that are less than 48 hours in duration and are not associated with hemodynamic compromise or evidence of cardiac ischemia. In this situation, conversion to sinus rhythm will avoid the need for anticoagulation (see later) and can prevent atrial remodeling that predisposes to recurrent AF (9). However, over 50% of cases of recent-onset AF convert spontaneously to sinus rhythm in the first 72 hours, so cardioversion is not necessary in most cases of recent-onset AF unless the symptoms are distressing.

Several antiarrhythmic agents are recommended for the acute termination of AF (flecainide, propafenone, ibutilide, difetilide, and amiodarone), but the only one with a success rate higher than 5 or 10% is ibutilide. When patients with recent-onset AF are given ibutilide in the dosing regimen shown in Table 18.1, over 50% will convert to sinus rhythm, and 80% will show a response within 30 minutes of drug administration (11). The only risk associated with ibutilide is torsade de pointes (described later), which is reported in 4% of cases (11). (For information

TABLE 18.1 Intravenous Drug Regimens for Acute Management of Atrial Fibrillation[t]

Drug	Dose Regimen	Comments
CARDIOVERSION		
Ibutilide	1 mg IV over 10 min and repeat once if needed.	The best agent available for acute cardioversion of AF. Torsade de pointes reported in 4% of cases
ACUTE RATE CONTROL		
Diltiazem	0.25 mg/kg IV over 2 min, then 0.35 mg/kg 15 minutes later if needed. Follow with infusion of 5-15 mg/hr for 24 hrs.	Effective rate control in > 95% of patients. Has negative inotropic actions, but can be used safely in patients with heart failure.
Esmolol	500 μg/kg IV over 1 min, then infuse at 50 μg/kg/min. Increase dose rate by 25 μg/kg/min every 5 min if needed to maximum of 200 μg/kg/min.	Ultra-short-acting l3-blocker that permits rapid dose Titration to desired effect.
Metoprolol	2.5 to 5 mg IV over 2 min. Repeat every 10-15 min if needed to total of 3 doses.	Easy to use, but bolus dosing is not optimal for exact rate control.
Amiodarone	300 mg IV over 15 min, then 45 mg/hr for 24 hrs.~	A suitable alternative for patients who do not tolerate more effective rate-reducing drugs.

t From the recommendations in Reference 3.

'Data from Reference 17.

on the other antiarrhythmic agents recommended for cardioversion of AF, see reference #3.)
Intravenous amiodarone is also recommended for acute termination of AF despite evidence of variable and limited efficacy. Bolus administration of amiodarone results in acute cardioversion of AF in less than 5% of cases (12). This agent may be more useful for acute rate control in AF, as described later.

Controlling the Heart Rate
The acute management of AF (particularly chronic or recurrent AF) is most often aimed at reducing the ventricular rate into the range of 60 to 80 bpm (3). If an arterial catheter is in place, monitoring the systolic blood pressure can provide a more physiological end-point for rate control. The systolic blood pressure is a reflection of the stroke volume, and the principal determinant of stroke volume is ventricular end-diastolic volume (this is the Frank-Starling relationship described in Chapter 1). When the heart rate in AF is slow enough to allow for adequate ventricular filling during each period of diastole, the systolic blood pressure (stroke volume) should remain constant with each heart beat. Therefore a constant systolic blood pressure with each heart beat can be used as an end-point of rate control in AF.
The drugs that are used for acute rate control in AF are shown in Table 18.I. These drugs are either calcium-channel blockers (diltiazem) or ~-blockers Cesmolol and metoprolol), and they act by prolonging conduction through the atrioventricular node, which slows the ventricular response to the rapid atrial rate.
Calcium-Channel Blockers
Verapamil was the original calcium-channel blocker used for acute rate control in AF, but diltiazem (Cardizem) is now preferred because it produces less myocardial depression and is less likely to produce hypotension (3). When given in appropriate doses, diltiazem will produce satisfactory rate control in 85% of patients with AF (13). The response to a bolus dose of diltiazem is evident within 5 minutes (see Figure 18.4), and the effect dissipates over the next 1 - 3 hours (14). Because the response is transient, the initial bolus dose of diltiazem should be followed by a continuous infusion. Although diltiazem has mild negative inotropic actions, it has been used safely in patients with heart failure (15).

B-Receptor Antanonists
B-blockers are the preferred agents for rate control when AF is associated with hyperadrenergic states (such as acute MI and post-cardiac surgery) (3,7). Two beta -blockers with proven efficacy in AF are esmolol (Brevibloc) and metoprolol (Lopressor), and their dosing regimens are shown in Table 18.1. Both are cardioselective agents that preferentially block beta -1 receptors in the heart. Esmolol is the preferred agent for acute rate control because it is ultra-short-acting (serum half-life of 9 minutes), and the infusion rate can be titrated rapidly to maintain the target heart

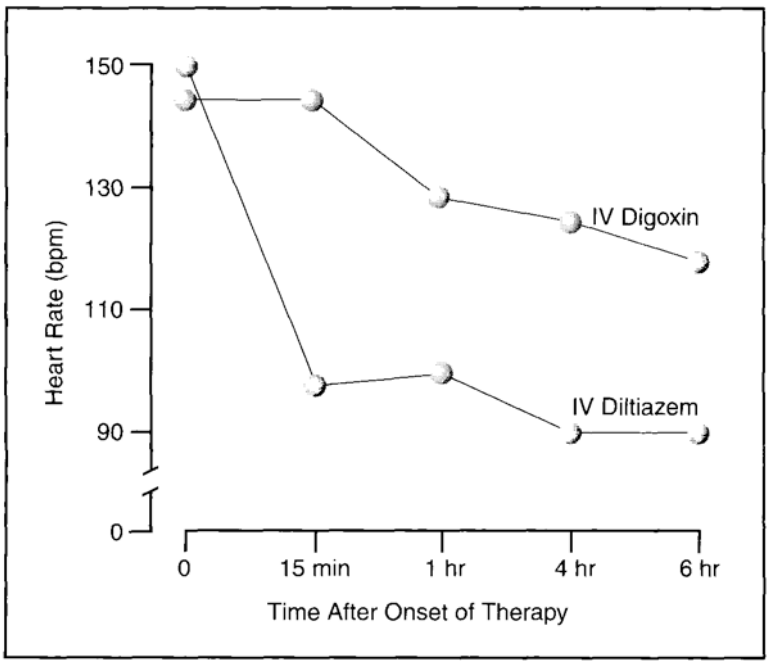

FIGURE 18.4 Comparative effects of IV diltiazem (same dose as in Table 18.1) and IV digoxin (0.5 mg in two divided doses over 6 hours) on acute control of heart rate in patients with recent-onset AF. (Adapted from data in Schreck DM, Rivera AR, Tricarico VJ, et al. Emergency treatment of atrial fibrillation and flutter: comparison of IV digoxin versus IV diltiazem. Ann Emerg Med 1995;25:127.)

rate (6). Because beta -blockers and calcium-channel-blockers both have cardiodepressant effects, combined therapy with beta -blockers and calcium-channel blockers should be avoided.

Amiodarone

Amiodarone can prolong conduction in the AV node and reduce the ventricular rate in patients with AF. When given in the dosing regimen shown in Table 18.1, intravenous amiodarone can produce an acute reduction in heart rate in 75% of patients with recent-onset AF (7). Although not as effective as diltiazem, amiodarone produces less cardiac depression and is less likely to produce hypotension than diltiazem (7). As a result, amiodarone may be a suitable alternative when other ratecontrolling drugs are not tolerated.

The potential side effects of short-term intravenous amiodarone include hypotension 05%), infusion phlebitis (15%), bradycardia (5%), and elevated liver enzymes (3%) (11,18). Hypotension is the most common side effect and is related to the vasodilator actions of amiodarone and the solvent (polysorbate 80 surfactant) used to enhance water solubility of the injectable drug (8). Hypotension can usually be managed by

decreasing the infusion rate or briefly stopping the infusion. The other common side effect is infusion phlebitis, which can be prevented by infusing amiodarone through a large, central vein. Amiodarone also has several drug interactions (18). It increases the serum concentrations of digoxin, warfarin, fentanyl, quinidine, procainamide, and cyclosporine. Many of the interactions are the result of amiodarone's metabolism via the cytochrome P450 enzyme system in the liver. The digoxin and warfarin interactions are the most important in the ICU.

Digoxin

Digoxin, by virtue of its ability to prolong AV conduction, has been a popular and effective agent for long-term rate control in AF. However, it should not be used for immediate rate control in AF because of its delayed onset of action. This is demonstrated in Figure 18.4, which shows the results of a study comparing the effects of IV diltiazem and IV digoxin on acute rate control in patients with recent-onset AF. The heart rate decreased to below 100 bpm (the target rate) promptly in the patients who received diltiazem, while the heart rate remained above 110 bpm after 6 hours in the patients who received digoxin. These results demonstrate that digoxin is ineffective for acute rate control in AF.

Anticoagulation

Cerebral embolic stroke (mentioned earlier) is the most devastating complication of AF. Each year about 6% of patients with AF will suffer an embolic stroke if they have certain high-risk conditions for thromboembolism. This can be reduced by 3% with warfarin anticoagulation to achieve an INR between 2.0 to 3.0 (9). In other words, therapeutic anticoagulation with warfarin in high-risk patients is associated with 3 fewer strokes for every 100 patients treated. This benefit requires strict monitoring of warfarin anticoagulation to keep the INR in the therapeutic range (2.0 - 3.0).

Risk Stratification

Table 18.2 shows the different antithrombotic strategies based on the risk factors for thomboembolism in patients with AF (3). The patients with the highest risk for thromboembolism (annual stroke rate > 6%) that will benefit from warfarin anticoagulation include those with rheumatic valvular disease, prosthetic valves, a prior history of thromboembolism, heart failure with comorbid conditions, or advanced age (> 75 years of age). The conditions with a low risk of thromboembolism (annual stroke rate < 2%) can be treated with daily aspirin. The risk of thromboembolism is lowest (the same as the general population) in patients with AF who are younger than 60 years of age and have no evidence of heart disease. This condition is known as *lone atrial fibrillation,* and it does not require any form of anti thrombotic therapy.

The benefits of anticoagulation must always be weighed against the risk of hemorrhage, particularly intracerebral bleeding. Warfarin

TABLE 18.2 Risk-Based Antithrombotic Strategies for Patients with Atrial Fibrillation[t]

I. Oral Anticoagulation (INR = 2.0 - 3.0)
Age> 75 years
Age 2: 60 years plus diabetes or coronary artery disease
Heart failure with LV ejection fraction < 0.35
Heart failure with hypertension or thyrotoxicosis
Prosthetic heart valves (mechanical or tissue)
Rheumatic mitral valve disease
Prior thromboembolism
II. Antiplatelet Therapy (Aspirin, 325 mg daily)
Age < 60 years with heart disease, but no risk factors~
Age 2: 60 years with no risk factors~
III. No Therapy Required
Age < 60 years and no heart disease (lone AF)

fRisk factors include heart failure, LV ejection fraction < 0.35, and history of
t From the AGG/AHA/EGG guidelines for the management of patients with fibrillation (3).

anticoagulation increases the yearly rate of intracerebral hemorrhage by < 1 % (19), so the risk:benefit ratio favors anticoagulation in high-risk patients with AF. For patients with a predisposition to bleeding, the decision to anticoagulate is made on a case-by-case basis.

Anticoagulation &. Cardioversion

In cases of recent-onset AF that are less than 48 hours in duration, the risk of embolism with cardioversion is low ‹1 %), so anticoagulation is not needed prior to elective cardioversion (3). In fact, as mentioned earlier, successful cardioversion of AF that is less than 48 hours in duration will avoid the need for long-term anticoagulation. When the duration of AF is > 48 hours, the risk of embolization with cardioversion is about 6%, and anticoagulation is recommended for 3 weeks before elective cardioversion.

Wolff-Parkinson-White Syndrome

The WPW syndrome (short P-R interval and delta waves before the QRS) is characterized by recurrent supraventricular tachycardias that originate from an accessory (re-entrant) pathway in the AV conduction system (2). (Re-entrant tachycardias are described later in the chapter.) One of the tachycardias associated with WPW syndrome is atrial fibrillation. Agents that prolong AV conduction and produce effective rate control in conventional AF (such as the calcium-channel blockers) can paradoxically accelerate the ventricular rate (by blocking the wrong pathway) in

patients with WPW syndrome. Thus in cases of AF associated with WPW syndrome, calcium-channel blockers and digoxin are contraindicated. The treatment of choice is electrical cardioversion or pharmacologic cardioversion with procainamide (20). The dosing regimen for procainamide is presented later.

MULTIFOCAL ATRIAL TACHYCARDIA

Multifocal atrial tachycardia (MAT) is characterized by multiple P wave morphologies and a variable P-R interval (see Figure 18.3). The ventricular rate is highly irregular, and MAT is easily confused with atrial fibrillation when atrial activity is not clearly displayed on the ECG. MAT is a disorder of the elderly (average age = 70), and over half of the cases occur in patients with chronic lung disease (21). The link with lung diseases may be partly due to the bronchodilator theophylline (22). Other associated conditions include magnesium and potassium depletion and coronary artery disease. (21).

Acute Management

MAT can be a difficult arrhythmia to manage, but the steps listed below can be effective.

Discontinue theophylline (although this is no longer a popular bronchodilator). In one study, this maneuver resulted in conversion to sinus rhythm in half the patients with MAT (22).

Give intravenous magnesium (unless there is a contraindication) using the following protocol: 2 grams MgSO 4 (in 50 mL saline) over 15 minutes, then 6 grams $MgS0_4$ (in 500 mL saline) over 6 hours (23). In one study, this measure was effective in converting MAT to sinus rhythm in 88% of cases, even when serum magnesium levels were normal. The mechanism is unclear, but magnesium's actions as a calcium-channel blocker may be involved.

Correct hypomagnesemia and hypokalemia if they exist. If both disorders co-exist, the magnesium deficiency must be corrected before potassium replacement is started. (The reason for this is described in Chapter 34.) Use the following replacement protocol: infuse 2 mg $MgS0_4$ (in 50 mL saline) IV over 15 minutes, then infuse 40 mg potassium over 1 hour.

If the above measures are ineffective, give IV metoprolol if there is no evidence of COPD otherwise, give IV verapamil (a calciumchannel blocker). Metoprolol, given according to the regimen in Table 18.2, has been successful in converting MAT to sinus rhythm in 80% of cases (21). The verapamil dose is 75-150 μg/kg IV over 2 minutes (3). Verapamil converts MAT to sinus rhythm in less than 50% of cases, but it can also slow the ventricular rate. Watch for hypotension, which is a common side effect of verapamil.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS

Paroxysmal supraventricular tachycardias (PSVT) are narrow QRS complex tachycardias that are characterized by an abrupt onset and abrupt termination (unlike sinus tachycardia, which has a gradual onset and gradual resolution). These arrhythmias occur when there is an accessory pathway in the conduction system between atria and ventricles that conducts impulses at a different speed than the normal pathway. This difference in conduction velocities allows an impulse traveling down one pathway (ante grade transmission) to travel up the other pathway (retrograde transmission). This circular transmission of impulses creates a rapid, self-sustaining, *re-entrant tachycardia.* The trigger is an ectopic atrial impulse that travels through either of the two pathways.

There are 5 different types of PSVT, each characterized by the location of the accessory pathway. The most common PSVT is *AV nodal re-entrant tachycardia,* where the accessory pathway is located in the AV node.

AV Nodal Re-entrantTachycardia

AV nodal re-entrant tachycardia (AVNRT) is one of the most common rhythm disturbances in the general population. It most often occurs in subjects who have no evidence of structural heart disease and is more common in women (2). The onset is abrupt, and there may be distressing palpitations, but there is usually no evidence of heart failure or myocardial ischemia. The ECG shows a narrow QRS complex tachycardia with a regular rhythm and a rate between 140 and 220 bpm. There may be no evidence of atrial activity on the ECG (see Figure 18.2), which is a feature that distinguishes AVNRT from sinus tachycardia.

Acute Management

Maneuvers that increase vagal tone can occasionally terminate AVNRT and can sometimes slow another type of tachycardia to reveal the diagnosis (e.g., sinus tachycardia). The vagal-enhancing maneuvers include the Valsalva maneuver (forced exhalation against a closed glottis), carotid massage, eyeball compression, and facial immersion in cold water (2). The value of these maneuvers is largely unproven, and some of the maneuvers (like eyeball compression or facial immersion in ice water) only add to the patient's distress and delay the termination of the arrhythmia. (Take a patient with AVNRT who is anxious and may be short of breath then stick the patient's face in a sink full of ice water and tell them to hold their breath, and you will know what I mean.)

AVNRT can be terminated quickly by drugs that block the re-entrant pathway in the AV node. The most effective drugs are calcium-channel blockers (verapamil and diltiazem) and adenosine. These agents are equally effective for terminating AVNRT, but adenosine works faster and produces less cardiovascular depression than the calcium-channel blockers.

TABLE 18.3 Intravenous Adenosine for Paroxysmal SVT

Indications: Termination of AV nodal re-entrant tachycardia, particularly in patients with
• Heart failure
• Hypotension
• Ongoing therapy with calcium channel blockers or ~-blockers
• WPW syndrome
Contraindications: Asthma, AV block
Dose: For delivery via peripheral veins:
1. Give 6 mg by rapid IV injection and flush with saline.
2. After 2 min, give a second dose of 12 mg if necessary.
3. The 12-mg dose can be repeated once.
Dose adjustments: Decrease dose by 50% for:
• Injection into superior vena cava
• Patients receiving calcium blockers, ~-blockers, or dipyridamole
Response: Onset of action < 30 sec. Effects last 1-2 min.
Side effects: Facial flushing (50%)
Sinus bradycardia, AV block (50%)
Dyspnea (35%)
Anginal-type chest pain (20%)
Nausea, headache, dizziness (5-10%)

AV = atrioventricular; VT = ventricular tachycardia; WPW = Wolff-Parkinson-
From References 24-27

Adenosine (Adenocard)

Adenosine is an endogenous purine nucleotide that briefly depresses activity in the sinus node and AV node (24). When given by rapid intravenous injection in the doses shown in Table 18.3, adenosine terminates re-entrant tachycardias in over 90% of cases and is effective within 30 seconds of drug injection (24-26). Each bolus dose of the drug should be followed by a 20 mL saline flush to speed up the drug effect. The effect dissipates in 1 to 2 minutes, so troublesome side effects are gone in a flash. Note in Table 18.3 that the dose of adenosine should be reduced by 50% when the drug is injected through a central venous (CVP) catheter instead of a peripheral vein (27). This recommendation is based on reports of ventricular asystole when standard doses of adenosine are injected through cvp catheters (27). Adenosine has an important drug interaction with theophylline (the once-popular bronchodilator agent). Theophylline blocks adenosine receptors and antagonizes the actions of adenosine. As a result, therapeutic doses of adenosine may not be effective in patients receiving theophylline, so combined therapy with adenosine and theophylline is not

advised. Fortunately, theophylline is disappearing from the medicine cabinets of most asthmatics (because of better bronchodilator effects with (beta -agonists), and the significance of the adenosine-theophylline interaction is diminishing.
Side effects are common after adenosine injection, and these are listed in Table 18.3 (26). However, these side effects are fleeting because of adenosine's ultra-short duration of action. Adenosine blocks catecholamine effects on the heart (24), but heart failure is not a problem because of the rapid disappearance of drug effects. One of the significant side effects is bronchoconstriction in asthmatic subjects (28,29), which is why adenosine should NOT be used in patients with asthma.

VENTRICULAR TACHYCARDIA
Ventricular tachycardia (VT) is the most feared and life-threatening tachyarrhythmia. Sustained VT (defined as VT that lasts longer than 30 seconds or causes hemodynamic compromise) rarely occurs in the absence of structural heart disease (30), and often indicates a profound disruption of the mechanical and electrical integrity of the heart. The appearance of VT is an ominous sign and requires prompt recognition and management.

Diagnosis
VT is a wide QRS complex tachycardia with a regular rhythm and a rate above 100 bpm. The onset is abrupt, and the hemodynamic consequences vary from no apparent effect to complete loss of pulses and cardiac arrest. VT can be *monomorphic* (QRS complexes are uniform in size and shape) or *polymorphic* (QRS morphology changes continuously). Monomorphic VT is most common and can be difficult to distinguish from an sVT with prolonged AV conduction (see below).

Diagnostic Clues
The single-lead ECG tracings in Figure 18.5 illustrate the difficulty in distinguishing VT from an SVT with aberrant (prolonged) AV conduction. The tracing in the upper panel shows a wide QRs complex tachycardia with a regular rhythm that looks very much like (monomorphic) VT. The tracing in the lower panel shows spontaneous conversion to sinus rhythm. Note that the QRS complex remains unchanged after the arrhythmia is terminated, revealing an underlying bundle branch block. Thus the apparent VT in the upper panel is actually a paroxysmal sVI superimposed on a pre-existing bundle branch block.
VT has two characteristic features on the ECG that can distinguish it from an SVT with aberrant conduction. One of these is *AV dissociation,* where there is no fixed relationship between P waves and QRS complexes. This may not be evident on a single-lead ECG tracing and is more likely to be discovered on a 12-lead ECG (where one of the leads might reveal p waves). The other diagnostic clue for VT is the presence of *fusion*

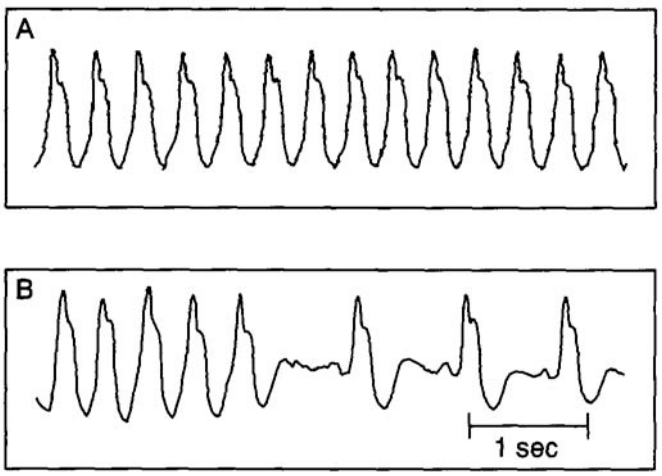

FIGURE 18.5 An SVT with aberrant (prolonged) AV conduction masquerading as VT. Spontaneous conversion to sinus rhythm in the lower panel reveals an underlying bundle branch block. (Tracings courtesy of Dr. Richard M. Greenberg, M.D.)

beats prior to the onset of the arrhythmia. A fusion beat is an irregularlyshaped QRS complex that is caused by retrograde transmission of a ventricular ectopic impulse that merges (fuses) with a normal QRS complex. The presence of a fusion beat (which should be evident on a single-lead ECG tracing) is therefore evidence of ventricular ectopic activity.

If there are no characteristic features of VT on the EeG, the presence or absence of heart disease can be useful, because VT is the cause of 95% of wide complex tachycardias in patients with primary heart disease (31). Therefore a wide QRS complex tachycardia in any patient with primary heart disease should be treated as probable VT.

Acute Management

The management of patients with a wide QRS complex tachycardia can proceed as follows. This approach is organized in a flow diagram in Figure 18.6.

If there is evidence of hemodynamic compromise, initiate DC cardioversion immediately with an initial shock of 100 J, followed by repetitive shocks of 200, 300, and 360 J, if necessary. This is necessary regardless of whether the rhythm is VT or SVT with aberrant conduction.

If there is no evidence of hemodynamic compromise and the diagnosis of VT is certain, intravenous amiodarone should be used to terminate the arrhythmia (see Figure 18.6 for the amiodarone dosing regimen). Lidocaine had been the favored antiarrhythmic agent for terminating VT; however, the most recent guidelines published by the American Heart Association (1) state

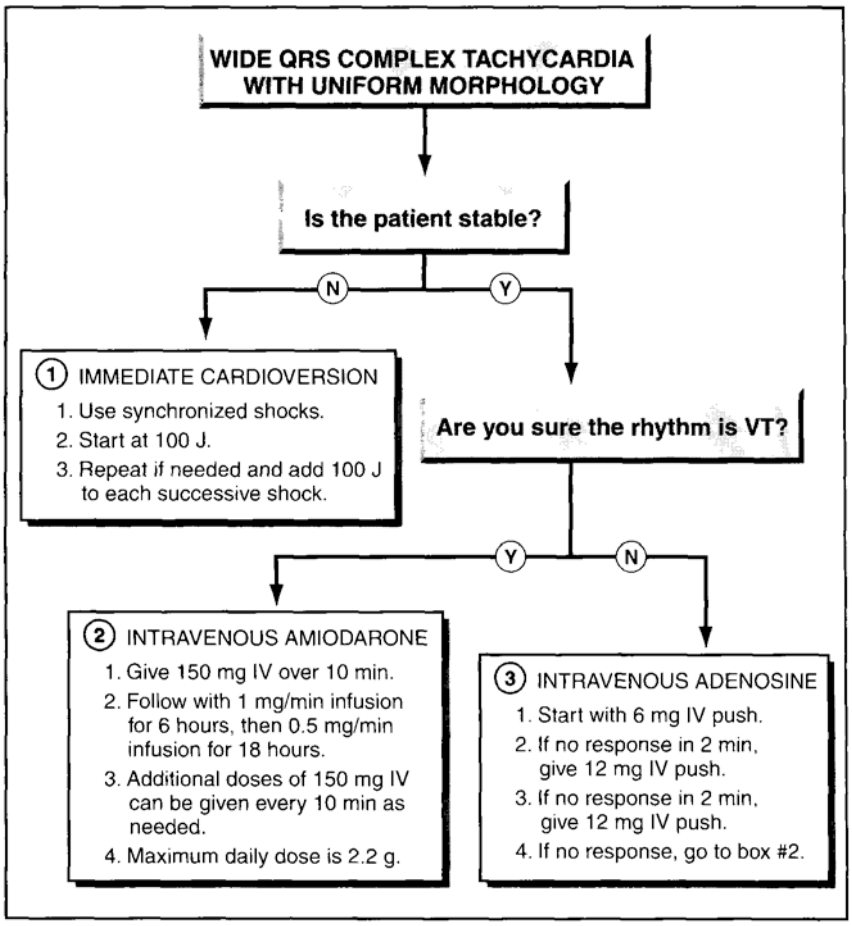

FIGURE 18.6 Flow diagram for the acute management of patients with wide QRS complex tachycardia. (Based on recommendations in the 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2005;112:IV-67.)

that amiodarone has replaced lidocaine as the antiarrhythmic agent of choice for terminating VT. This recommendation may be based on the safety of amiodarone in patients with heart failure because most cases of VT occur in patients with significant cardiac disease.

If there is no evidence of hemodynamic compromise and the diagnosis of VT is uncertain, intravenous adenosine can help to unmask a paroxysmal SVT (like the rhythm in Figure 18.4). Adenosine will not terminate VT, but it will abruptly terminate most cases of paroxysmal SVT. If the arrhythmia persists after adenosine, VT is the likely diagnosis, and intravenous amiodarone is indicated to terminate the rhythm.

Other Antiarrhythmic Agents

Despite the current preference for amiodarone, other antiarrhythmic agents are effective in suppressing VT, and these drugs can be used as alternatives to amiodarone.

Lidocaine has a long history of success in suppressing VI. The initial dose is 1 to I.5 mg/kg by bolus injection. After 5 minutes, a second dose of 0.5 to 0.75 mg/kg can be given if needed. A maintenance infusion of 2 to 4 mg/min can be used for continued arrhythmia suppression. Prolonged infusions of lidocaine can produce an excitatory neurotoxic syndrome, particularly in elderly patients. For this reason, lidocaine infusions should not be continued longer than 6 to 12 hours.

Procainamide is considered a second-line drug for VT because it cannot be administered rapidly, which prolongs the time to arrhythmia suppression. Procainamide also prolongs the QT interval, and this can result in drug-induced VT when procainamide is given to patients with a prolonged QT interval. For this reason, procainamide is contraindicated in patients with a prolonged QT interval (>0.44 sec after correction for heart rate).

Procainamide is infused at a rate of 20 mg/min until the arrhythmia is suppressed or a total dose of 17 mg/kg is reached (32). The infusion should be terminated if the QT interval increases by 50% (30). The procainamide dose should be reduced by 50% in patients who are elderly or have renal dysfunction, and a 25% reduction in dose is recommended for patients with heart failure (20).

Torsades De Pointes

Torsades de pointes ("twisting around the points") is a polymorphic VT characterized by QRS complexes that change in amplitude and appear to be twisting around the isoelectric line of the ECG (see Figure 18.7). This arrhythmia is associated with a prolonged QT interval (33), and it can be congenital (idiopathic) or acquired. The acquired form is caused by a variety of drugs and electrolyte disorders that prolong the QT interval. The drugs that can trigger this arrhythmia are listed in Table 18.4 (34,35). The prominent offenders are antiarrhythmic drugs, macrolide and quinolone antibiotics, and psychotropic agents. The electrolyte disorders

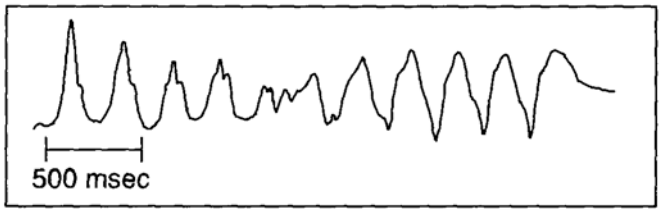

FIGURE 18.7 Torsades de pointes, a polymorphic ventricular tachycardia described as "twisting around (the isoelectric) points." (Tracing courtesy of Dr. Richard M. Greenberg, M.D.)

TABLE 18.4 Drugs That Can Trigger Torsades de Pointes†

Antiarrhythmic Agents		Antimicrobial Agents	Antipsychotic Agents	Other Agents
IA	Quinidine	Clarithromycin	Chlorpromazine	Cisapride
IA	Procainamide	Erythromycin	Haloperidol	Droperidol
	Flecainide	Gatifoxacin	Thioridazine	Methadone
III	Ibutilide	Levofloxacin		
III	Sotalol	Pentamidine		

† From References 34, 35.

that prolong the QT interval and predispose to torsades de pointes are hypokalemia, hypomagnesemia, and hypocalcemia.
Polymorphic VT can also be associated with a normal QT interval. This condition is not torsades de pointes and is simply known as polymorphic VT.

Management
The management of polymorphic VI is guided by the QT interval. This parameter is usually measured in limb lead II and must be corrected for the heart rate. The rate-corrected QT interval (QTc) is equivalent to the measured QT interval divided by the square root of the R-R interval (36). A prolonged QT interval is defined as QTc > 0.44 seconds.

$$QTc = QT / \sqrt{RR}$$

Polymorphic VT with a normal QT interval can be managed with standard antiarrhythmic agents like amiodarone and lidocaine. For cases of torsades de pointes associated with a prolonged QT interval, the management strategy depends on whether the QT prolongation is congenital or acquired.
If the QT prolongation is acquired:
Give intravenous magnesium (as magnesium sulfate, MgSO/ Start with 2 grams IV over one minute, and repeat this dose 10 minutes later if needed. Follow with a continuous infusion of 1 gram per hour for the next 6 hours.
Correct electrolyte abnormalities if present. Remember that hypokalemia and hypocalcemia may be the result of an underlying magnesium deficiency (even if the serum magnesium levels are normal). In this situation, it is difficult to correct the serum potassium and calcium levels until the magnesium deficit is corrected. Therefore magnesium replacement should be the first order of business in patients with hypokalemia and hypocalcemia. (The importance of magnesium deficiency in hospitalized patients is presented in Chapter 34.)
Discontinue any drugs that prolong the QT interval.

If the QT prolongation is a congenital disorder, use ventricular pacing to raise the heart rate above 100 bpm. The rapid rate will shorten the QT interval and reduce the tendency for VT.

A FINAL WORD

Serious or life-threatening tachyarrhythmias are uncommon events in critical care areas other than coronary care units. This is probably because pathologic arrhythmias are triggered by focal areas of myocardial ischemia or focal alterations in myocardial architecture, and most critically ill patients (other than those admitted for coronary insufficiency) do not experience these events during the course of their illness. To gain experience in the diagnosis and management of tachyarrhythmias, spend time in a coronary care unit or an emergency room.

REFERENCES