Short- and Long-term Complications of Coronary Angioplasty

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PTCA = percutaneous transluminal coronary angioplasty

CASE REPORT

This 48-year-old man was in good health until April 1988, when he noted the onset of exertional chest pain. One month later, he underwent cardiac catheterization, which revealed a totally occluded left circumflex coronary artery. His only risk factor for atherosclerotic cardiovascular disease was cigarette use. He underwent successful percutaneous transluminal coronary angioplasty (PTCA) of the occluded circumflex coronary artery. He was free of symptoms until October 1988, when his exertional chest pain recurred. Coronary arteriography again demonstrated total occlusion of the circumflex coronary artery with collateral filling of the distal obtuse marginal branches (Fig 1). A second angioplasty was successfully performed (Fig 2) with no significant residual stenosis, and his angina pectoris resolved. One month later, his symptoms reappeared, and coronary arteriography revealed reocclusion of the circumflex coronary artery. Despite antianginal medications, his lifestyle was limited by angina pectoris, and he was referred for coronary artery bypass surgery.

In September 1977, in Zurich, Andreas Gruentzig performed the first percutaneous transluminal coronary angioplasty (PTCA). Despite initial skepticism about the procedure's utility, it has now been performed successfully in hundreds of thousands of patients with coronary artery disease. In the United States alone, over 200,000 coronary angioplasties were performed in 1988. It has gained widespread acceptance because of its high likelihood of success and its attractiveness as an alternative to coronary artery bypass surgery. At the same time, however, several short- and long-term complications may develop in patients undergoing PTCA.

PATHOPHYSIOLOGIC MECHANISMS OF PTCA

Specimens from experimental animals and patients dying after PTCA have been examined to determine the morphologic alterations caused by the procedure. At the site of dilatation, disruption and cracking of the atheroma occur, with denudation of the endothelial surface, tearing of the luminal surface, and intimal hemorrhage. The intima and plaque may be separated from the underlying media, causing dissection and hemorrhage into the media and adventitia. With successful angioplasty, persistent aneurysmal dilatation of the media and adventitia is noted, and distal embolization of plaque and thrombus is sometimes observed. In essence, successful PTCA causes a "controlled injury" of the diseased arterial segment.

Exposure of the freshly cracked atheroma and subintimal layers of the coronary artery to circulating blood elements induces platelet deposition and thrombus formation. Platelet aggregation causes the release of vasoconstrictor substances, chemotactants, and other mediators, which, in turn, activate macrophages and smooth muscle cells and may lead to arterial spasm. In addition, platelet aggregation may stimulate the production of growth factors, which induce the proliferation of smooth muscle cells. Thus, in response to angioplasty-induced injury, a reparative process

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Figure 1. A coronary arteriogram performed 5 months after initial PTCA. The proximal circumflex coronary artery is occluded (arrow), and the distal vessel fills by collaterals. LAD = left anterior descending coronary artery; OM = obtuse marginal branch of the left circumflex coronary artery.
leading to neointimal proliferation is initiated.

**Short-term Complications of PTCA**

About 8 to 10 percent of attempted angioplasties are unsuccessful in dilating the narrowed arterial segment or sustaining its patency. Of these, half are due to an inability to cross the stenosis with the angioplasty wire or balloon or to dilate the stenosis, even though the balloon is positioned appropriately. The other half are due to abrupt closure of the artery after dilatation. Thus, acute occlusion occurs in 4 to 5 percent of patients undergoing PTCA. Such abrupt closure accounts for most of the morbidity and mortality associated with PTCA. It is usually characterized by the acute onset of chest pain with associated ECG evidence of ischemia. About 80 percent of patients with abrupt closure manifest it within 2 to 3 h of PTCA, and half occur while the patient is still in the catheterization laboratory. Infrequently, acute closure occurs several days after PTCA. Of the 140 abrupt closures reported by the Emory University group, the median time from PTCA to closure was 10 minutes, with a range of 0 minutes to 93 h.

**Mechanism of Abrupt Closure**

Three factors contribute to the development of abrupt closure after PTCA: dissection, thrombus formation, and coronary spasm. Although each has been implicated as the primary cause of abrupt closure, dissection and thrombus formation are particularly important.

Limited intimal dissection is frequently observed following successful PTCA. It is identified angiographically as a linear filling defect or "flap" associated with a hazy, ground-glass intraluminal appearance. When the dissection is limited, these angiographic features may be evident only on selected views. However, if it is extensive (a concentric or spiral dissection), the radiographic features are evident on multiple views, and the vessel lumen is often compromised. Extensive dissections are caused by injury of the deeper layers of the arterial wall, with subsequent intramural hemorrhage. Seventy to 100 percent of patients with abrupt closure have angiographic evidence of dissection, and 10 percent of patients with dissection require emergency coronary artery bypass surgery or sustain a myocardial infarction or death. In contrast, only 1.6 percent of patients without dissection have a major complication.

Acute thrombus formation after PTCA has been demonstrated at angiography, at postmortem shortly after angioplasty, and at coronary arteriography. Angiographically, it is characterized as a discrete, nonlinear intraluminal filling defect or an area of contrast staining. In patients undergoing PTCA without antiplatelet therapy, 22 percent had angiographically demonstrable thrombi after dilatation, with half causing complete occlusion and requiring revascularization or thrombolysis. Thrombus formation is especially likely to occur in patients with extensive dissection, in those with a severe residual coronary stenosis following dilatation, and in those who smoke or have a high platelet count.

In most subjects, coronary spasm plays a minor role in the development of abrupt closure after PTCA. Although a few reports have described acute closure that responded to vasodilators, only 4.8 percent of the 3,079 patients reported in the initial NHLBI PTCA-Registry had isolated coronary spasm, and only 18 percent of these sustained a major complication (death, myocardial infarction, or need for repeat PTCA or emergent coronary artery bypass surgery). Currently, most patients receive nitrates and calcium antagonists before and after PTCA, and isolated coronary spasm is rarely the cause of abrupt closure.

**Consequences of Abrupt Closure**

The consequences of acute occlusion vary widely. On the one extreme, the patient with adequate collateral blood supply to the occluded vessel may have abrupt closure without chest pain, ECG alterations, or other evidence of myocardial injury. More commonly, abrupt closure is accompanied by chest discomfort and ECG evidence of ischemia and requires urgent revascularization (repeat PTCA or emergent coronary artery bypass grafting) of the occluded vessel to prevent or limit myocardial injury. In the initial NHLBI PTCA-Registry, acute closure accounted for 53 percent of emergency coronary artery bypass operations, 28 percent of in-hospital deaths,
Table 1—Variables Associated with an Increased Incidence of Abrupt Closure Following Successful PTCA

<table>
<thead>
<tr>
<th>A. Clinical variables</th>
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<tbody>
<tr>
<td>1. Female gender</td>
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<td>2. Unstable angina</td>
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<td>3. Evolving myocardial infarction</td>
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<th>B. Anatomic variables</th>
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<tbody>
<tr>
<td>1. Thrombus or intimal tear</td>
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<td>2. Additional stenoses in the dilated artery</td>
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<tr>
<td>3. Multivessel coronary artery disease</td>
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<td>4. Right coronary artery stenosis</td>
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<td>5. Length (≥2 luminal diameters)</td>
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<td>6. Eccentricity of stenosis</td>
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<td>7. Stenosis at a bend or branch point in the artery</td>
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<th>C. Procedural variables</th>
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<tr>
<td>1. Oversized balloon</td>
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<td>2. Severity of post-PTCA stenosis</td>
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<tr>
<td>a. Residual stenosis ≥35%</td>
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<tr>
<td>b. Transstenotic pressure gradient ≥20 mm Hg</td>
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and 36 percent of myocardial infarctions following PTCA. Of patients with abrupt closure, 5.3 percent died, and 75 percent underwent emergency coronary artery revascularization or had a myocardial infarction. Of the 140 patients with acute occlusion reported by Ellis et al., 34 percent did not undergo revascularization and sustained a myocardial infarction; 11 percent had emergency repeat PTCA; and 55 percent underwent emergency coronary artery bypass surgery. Those with abrupt closure have an overall mortality of 2.9 to 5.3 percent, and this incidence is increased in women, in patients with multivessel coronary artery disease, and in those with a large amount of myocardium supplied by the occluded artery.

Risk Factors of Abrupt Closure

Several clinical, anatomic, and procedural variables are associated with an increased incidence of acute closure after PTCA (Table 1), and the influence of these variables on the incidence of acute closure is cumulative, i.e., the more risk factors present, the greater the likelihood that acute closure will occur.

Three clinical factors appear to be associated with an increased incidence of acute occlusion: female gender, unstable angina, and evolving myocardial infarction. In comparison to men, women have a higher incidence of abrupt closure and PTCA-related mortality. This was initially attributed to the inadvertent use of oversized balloons in women, but recent studies using appropriately sized balloons continue to show an increased incidence of abrupt closure in women. A high incidence of acute closure (7 to 12 percent) has been noted when angioplasty is performed in the setting of unstable angina or evolving myocardial infarction. Enhanced platelet activation and thrombus formation associated with these disease entities is thought to be the underlying cause. Other clinical variables, such as age, hypertension, diabetes mellitus, cigarette use, and repeat PTCA, do not appear to be associated with an increased incidence of abrupt closure.

Several anatomic features are associated with an increased incidence of acute closure after PTCA. The presence of thrombus or an intimal tear at the site of dilatation confers an increased risk: the more extensive the dissection, the more likely acute closure is to occur. Acute occlusion is more likely if the dilated vessel contains other stenoses or if multivessel coronary artery disease is present. Stenoses of the right coronary artery—especially those located near the ostium—have an increased predilection for acute closure. Several characteristics of the stenosis appear to increase the incidence of abrupt closure. If the stenosis is long (≥2 luminal diameters), eccentric, or occurs at a branch point or at a bend in the artery, an increased risk of abrupt closure is conferred. Although early reports suggested that stenosis calcification was associated with an increased incidence of abrupt closure, more recent investigations have not supported this claim.

Finally, certain procedural factors influence the incidence of abrupt closure after angioplasty. Dilatation with an oversized balloon leads to an increased incidence of dissection and acute closure. The severity of the residual stenosis also appears to be important, measured as a change in diameter or transstenotic pressure gradient. A stenosis after PTCA ≥35 percent or a residual transstenotic pressure gradient ≥20 mm Hg are associated with an increased incidence of acute closure. In contradistinction, the severity of the pre-PTCA stenosis or gradient bears no relation to abrupt closure.

Management of Abrupt Closure

The most successful treatment of acute closure is prevention. Accordingly, therapies aimed at each of the proposed causes (dissection, thrombosis, and spasm) are routinely instituted before PTCA. Calcium antagonists and nitrates are administered to patients before and after angioplasty, even though no study has demonstrated that these agents reduce the incidence of abrupt closure. All patients are systemically anticoagulated with heparin at the time of angioplasty, and in many centers high-risk patients (those with extensive dissection) are treated with a prolonged (12- to 24-h) heparin infusion after PTCA, although the efficacy of this therapy is unproved. Only antiplatelet therapy has been shown to be efficacious in reducing the incidence of acute closure after angioplasty. Pretreatment with aspirin and dipyridamole is associated with a decreased incidence of acute coronary thrombosis, acute closure, and periprocedural myocardial infarction.
When acute closure occurs with PTCA, the patient’s management is dictated by the amount of myocardium perfused by the occluded artery, the presence of collateral blood supply, and the risk of emergent revascularization. Conservative medical therapy is often chosen if adequate collateral blood supply is present or only a small amount of myocardium is jeopardized. About one-third of patients are treated conservatively following abrupt closure. The remaining patients undergo revascularization. In the early PTCA experience, this usually entailed emergent coronary artery bypass surgery. More recently, however, repeat PTCA is attempted in about 60 percent of patients with abrupt closure, with a success rate of 60 to 100 percent (average, 87 percent). One-third of these patients still require emergent coronary artery bypass surgery for recurrent occlusion despite successful repeat PTCA, whereas the other ⅔ of patients who have repeat PTCA for abrupt closure require no further intervention.

One-third of patients with acute closure as well as ⅔ of those with recurrent occlusion after repeat PTCA require emergent coronary artery bypass surgery. A mortality of 2 to 12 percent (average, 4.5 percent) is reported in these patients. The mortality for women undergoing emergent coronary artery bypass surgery after acute occlusion is five-fold higher than that for men. Despite successful revascularization by repeat PTCA or emergent coronary artery bypass surgery, 25 to 50 percent of patients with abrupt closure sustain a Q-wave myocardial infarction.

Several new therapeutic modalities for abrupt closure are undergoing clinical investigation. For patients with closure despite repeat PTCA, an expandable intracoronary wire stent may be used to maintain vessel patency. The stent is placed over an angioplasty balloon and expanded at the site of closure with balloon inflation. Although initial reports have been encouraging, thrombosis at the site of stent placement occurs frequently. An angioplasty balloon with thermal and laser capabilities designed to “tack up” intimal dissections is under investigation, but coronary spasm, perforation, and restenosis occur frequently when the prototype laser and thermal balloon is used. For patients who require emergent coronary artery bypass grafting, perfusion balloon angioplasty catheters (so-called bail-out catheters) and artificial oxygen carrying solutions are being studied as means to maintain adequate perfusion of the jeopardized myocardium until revascularization is completed. Finally, percutaneous cardiopulmonary support or intra-aortic balloon counterpulsation have been used to protect the heart while the patient is being prepared for surgical revascularization. These modalities, although promising, are still under evaluation, and their long-term benefit in patients with abrupt closure after angioplasty is unknown.

**Long-term Complications of PTCA**

The two chronic complications that may occur with PTCA are (a) the development of a new stenosis in the dilated artery and (b) restenosis at the site of dilatation. These develop in the weeks to months following successful PTCA. While the arterial injury induced by balloon inflation is responsible for the short-term complications of angioplasty, it is the reparative response to this injury that is responsible for these long-term complications. Human pathologic specimens obtained weeks to months after successful PTCA typically demonstrate excessive neointimal proliferation with a large component of smooth muscle cells. In addition, fibrocellular organization of previously formed thrombus is evident at the site of successful dilatation. In short, restenosis is thought to result from an exaggerated proliferative cellular response to balloon-induced arterial injury.

**New Stenosis**

Although the appearance of new stenoses after successful PTCA have been described, their exact incidence is unknown. These new stenoses typically occur proximal to the site of successful dilatation. The inciting event is thought to be intimal injury caused by the guiding catheter, angioplasty balloon, or angioplasty wire. Histologic examination of the left main coronary artery in patients who died within 72 h of angioplasty revealed that most had focal loss of endothelium believed due to manipulation of the guiding catheter.

**Restenosis**

Despite technical advances that have reduced the incidence of acute complications following PTCA, the incidence of restenosis has not changed. Such restenosis—defined as (a) an increase in luminal narrowing ≥30 percent in comparison to the appearance of the arterial segment immediately after angioplasty, or (b) a loss of ≥50 percent of the initial improvement in the cross-sectional diameter of the lumen—occurs in 25 to 35 percent of patients in whom a stenosis is dilated and in 45 to 55 percent of those in whom a totally occluded artery is dilated.

**Consequences of Restenosis**

As demonstrated in our patient, those who develop restenosis after successful angioplasty usually complain of chest pain with exertion. Only rarely is myocardial infarction the initial presentation of restenosis. The severity of narrowing at the site of restenosis is usually similar to that before PTCA. However, when angioplasty is performed in a minimally narrowed
coronary artery, the restenosis may be more severe than what was present initially. Consequently, "cosmetic angioplasty"—to improve the appearance of a minimally diseased vessel—is not advisable.

**Risk Factors for Restenosis**

Several retrospective studies have examined the factors that influence the incidence of restenosis following successful PTCA. Various clinical, anatomic, and procedural variables are associated with an increased likelihood of restenosis (Table 2). Some of these risk factors for restenosis are also predictors of acute closure.

Of the clinical factors that have been examined, unstable angina, new onset angina (appearing within the two to six months before PTCA), and vasospastic angina are associated with an increased incidence of restenosis. In patients with these entities, enhanced vasoreactivity and platelet activation may play a pathophysiologic role in mediating restenosis. Patients with diabetes mellitus have an increased incidence of restenosis. The role of gender and cigarette smoking in the occurrence of restenosis is controversial. Although data from the NHLBI PTCA-Registry suggested that women had a lower incidence of restenosis (22 percent vs 36 percent), other large studies have not confirmed this observation. Similarly, several studies have suggested that cigarette smoking increases the incidence of restenosis, but other large retrospective analyses have not supported this association. Thus, the role of gender and cigarette smoking in the development of restenosis in our patient is unknown.

Several anatomic factors are associated with an increased incidence of restenosis. First, the incidence of restenosis is influenced by the location of the stenosis. Dilatations of the left anterior descending coronary artery are associated with a higher incidence of restenosis than those of the circumflex or right coronary arteries. In 998 patients with single vessel coronary artery disease who underwent successful angioplasty at Emory University, restenosis occurred in 34 percent of left anterior descending, 27 percent of right, and only 18 percent of circumflex coronary arteries. A proximally located stenosis is more likely to restenose than one that is located distally. Second, the incidence of restenosis increases as the severity of the preangioplasty stenosis worsens. Third, as in the patient presented, total coronary artery occlusions on which PTCA is performed have a higher incidence of restenosis than subtotal stenoses. Other anatomic variables, such as calcification, eccentricity, or length of the stenosis, do not appear to influence the incidence of restenosis.

Certain procedural factors contribute to the occurrence of restenosis. If an undersized balloon is used, the incidence of restenosis is increased. Similarly, if the result of the angioplasty is suboptimal—i.e., there is a postangioplasty stenosis ≥30 percent or a transstenotic pressure gradient ≥15 to 20 mm Hg—a higher incidence of restenosis is observed. Finally, an extensive dissection is associated with a higher incidence of restenosis, whereas a limited dissection is associated with a lower incidence of restenosis. Inflation pressure and the number of balloon inflations do not appear to influence long-term outcome of successful PTCA.

**Management of Restenosis**

Numerous pharmacologic agents have been given in an attempt to prevent or to reduce the incidence of restenosis. Antiplatelet agents, though beneficial in experimental animals, have not proved efficacious in man. A combination of aspirin and dipyridamole begun 24 hours before PTCA and continued for six months caused no reduction in the incidence of restenosis, and similar data are available for ticlopidine, another antiplatelet agent. Heparin has been proposed as a means of decreasing the incidence of restenosis because it inhibits smooth muscle proliferation and thrombus formation, but a large randomized trial showed that a prolonged (18 to 24 hours) heparin infusion after PTCA exerted no effect. Similar results have been obtained with warfarin following PTCA. Based on studies demonstrating that corticosteroids reduce the incidence of restenosis in rabbits, methylprednisolone (1 g IV) was given to patients 2 to 24 hours before PTCA. Although no overall change in the incidence of restenosis was observed, certain subsets of patients may have benefited. The calcium antagonists, nifedipine and diltiazem, are not effective in preventing or reducing the incidence of restenosis.

Dehmer et al administered omega-3 fatty acids (fish oils) in a prospective, placebo-controlled study of 82 men considered to be at high risk of restenosis.

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**Table 2—Variables Associated with an Increased Incidence of Restenosis Following Successful PTCA**

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<tr>
<th>A. Clinical variables</th>
<th>B. Anatomic variables</th>
<th>C. Procedural variables</th>
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<tbody>
<tr>
<td>1. Unstable angina</td>
<td>1. Location</td>
<td>1. Undersized balloon</td>
</tr>
<tr>
<td>2. Vasospastic angina</td>
<td>a. Left anterior descending coronary artery</td>
<td>2. Extensive dissection</td>
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<td></td>
<td>a. Residual stenosis ≥30%</td>
<td>a.</td>
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If an undersized balloon is used, the incidence of restenosis is increased. Similarly, if the result of the angioplasty is suboptimal—i.e., there is a postangioplasty stenosis ≥30 percent or a transstenotic pressure gradient ≥15 to 20 mm Hg—a higher incidence of restenosis is observed. Finally, an extensive dissection is associated with a higher incidence of restenosis, whereas a limited dissection is associated with a lower incidence of restenosis. Inflation pressure and the number of balloon inflations do not appear to influence long-term outcome of successful PTCA.
Eicosapentaenoic acid (3.2 g/day) was administered for seven days before and six months after PTCA. The incidence of restenosis was reduced from 36 percent in the placebo group to 16 percent in the treatment group. Further studies are needed to determine if a similar beneficial effect of omega-3 fatty acids is demonstrable in other patient populations.

Once restenosis has occurred, it may be treated with repeat PTCA. In fact, repeat angioplasty carries a higher chance of success and a lower chance of complications than the initial procedure, probably because patients whose first angioplasty was unsuccessful or complicated are excluded. A second restenosis develops in about 30 percent of patients who undergo repeat PTCA. Factors shown to correlate with a second restenosis are male gender, an interval between the first and second angioplasties <5 months, stenosis length ≥15 mm before the second procedure, and the need to have an additional site dilated at the time of the second procedure. Although third and even fourth dilatations have been performed in some patients with recurrent restenosis, most are referred for elective coronary artery bypass surgery.

**Questions**

**Question 1: How does one conceptually resolve the apparent contradiction that a limited intimal dissection is good (the risk of restenosis is reduced), whereas a more extensive dissection may lead to abrupt closure and restenosis?**

Answer: Angioplasty causes acute arterial injury, manifest as disruption of the atheroma, subintimal hemorrhage, and dilatation of the vessel wall. A successful result occurs when dilatation increases the luminal size of the vessel while vessel wall disruption and subintimal hemorrhage are limited to the superficial layers of the artery. Angiographically, this is recognized as a limited dissection. Clinically, it results in a favorable outcome with regard to acute and chronic complications. In contrast, when PTCA causes injury to the deeper layers of the vessel, there is an increased risk of abrupt closure and restenosis. Angiographically, this appears as an extensive dissection. Morphologically, one sees extensive damage to the deeper medial and adventitial layers of the arterial wall, with marked hemorrhage, narrowing of the vessel lumen, and thrombus formation. Thus, an extensive dissection post-PTCA causes deeper arterial injury with a less favorable acute and long-term outcome.

**Question 2: What factors influence the decision to operate emergently in patients with abrupt closure after PTCA?**

Answer: A number of factors are considered when deciding whether to proceed with emergent coronary artery bypass surgery in the setting of abrupt closure following PTCA. First, the amount of myocardium in jeopardy from the occluded vessel must be assessed. If only a small amount of myocardium is at risk, conservative management is usually recommended. Conversely, if acute closure would culminate in a large myocardial infarction, the benefits of emergency surgery outweigh the risks. Second, the suitability of the vessel for bypass grafting and the underlying medical condition of the patient must be considered. PTCA is sometimes performed on vessels or in patients in whom surgery is not a viable alternative. In these situations, medical management of the acute complications remains preferable. Third, the timely availability of surgery is an important consideration. If surgery is likely to be unduly delayed, revascularization probably will not result in salvage of myocardium. Additionally, emergent revascularization of the patient who has previously undergone cardiovascular surgery is usually unsuccessful in preventing infarction and may result in damage to existing functional coronary artery bypass grafts.

**Question 3: Why do some abrupt closures occur late (ie, >12 hs) after PTCA?**

Answer: The processes that are initiated by PTCA—subintimal hemorrhage and thrombus formation—continue for hours to days after angioplasty, so that late closure probably is caused by mechanisms similar to those that are responsible for early occlusion. Thrombus formation may play a particularly prominent role in the development of these late occlusions. In support of this, case reports and retrospective studies have noted a temporal relation between acute vessel closure and the discontinuation of heparin. As a result, some physicians recommend prolonged anticoagulation after PTCA, but prospective studies have not supported the claim that prolonged anticoagulation reduces the incidence of early or late abrupt closure.

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