New advances in the management of acute coronary syndromes: 3. The role of catheter-based procedures

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The cases

Mr. F, a 65-year-old man, presents to the emergency department with chest and epigastric pain of more than 2 hours' duration. The previous evening he experienced similar pain lasting 30 minutes. He has no history of cardiac disease, known risk factors or other serious illnesses, but he has not seen a physician for many years. His blood pressure is 120/65 mm Hg, the heart rate is 60 beats/min, there are bilateral fine inspiratory crackles in the lung bases, the venous pressure is normal, and there are quiet heart sounds. The electrocardiogram (ECG) shows ST-segment elevation in leads V_1 to V_4 and reciprocal ST-segment depression in leads II, III and aVF (Fig. 1). A chest x-ray film appears normal. What are the options for reperfusion therapy?

Mr. G, a 48-year-old smoker, presents to hospital with unremitting chest pain of 4 hours' duration. He has a 2-week history of episodic central chest pain, for which he did not seek medical attention. His blood pressure is 170/90 mm Hg, the heart rate is 75 beats/min, his chest is clear, the central venous pressure and heart sounds are normal, and there is a bruit heard over the right femoral artery. The initial ECG (Fig. 2) shows inferior T-wave inversion. The baseline levels of creatine kinase (CK), its MB isoenzyme (CK MB) and cardiac troponin I are twice the upper limit of normal. Mr. G is given 325 mg of ASA, 50 mg of metoprolol 3 times daily, nitroglycerin intravenously, unfractionated heparin and tirofiban. His symptoms resolve within 15 minutes after receiving the nitroglycerin. Should Mr. G undergo cardiac catheterization?

Initially suitable only for highly selected low-risk patients, percutaneous coronary revascularization has evolved into a vital component of the management of acute coronary syndromes (ACS). Remarkable advances in catheters, stents, adjunctive pharmacologic therapy, imaging and operative techniques have expanded the application of percutaneous coronary interventions (PCIs) to patients with clinically and anatomically complex heart disease.

Clinical research conducted in recent years has established sound evidence for the role of coronary angiography and PCIs in patients with ACS. Canadian investigators have made important contributions to this literature. This review is intended to convey the main clinical concepts and lessons from this body of research.

Canadian cardiac catheterization laboratories are limited in number and are highly centralized. Although centralization offers an enviable concentration of expertise and operational efficiencies, it has created special challenges for the majority of clinicians who practise in centres without on-site cardiac catheterization. A challenge to implementing evidence-based care, therefore, is to develop and use a patient transportation infrastructure. We have attempted to highlight these considerations where appropriate.

Review

Synthèse

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Modern percutaneous coronary intervention techniques

Two developments in the 1990s dominated the evolution of PCI techniques: stents and improved antithrombotic therapy using glycoprotein IIb/IIIa platelet inhibitors.

Stenting

Before the advent of stenting, PCI was hampered by 2 problems: occlusive coronary dissection and thrombosis (leading to acute myocardial infarction or emergency bypass surgery), and restenosis (leading to recurrent ischemia). The introduction of coronary stents (Fig. 3) has dramatically altered this landscape. Before stents became available, emergency bypass surgery was required in 2%–3% of patients undergoing PCI; this risk is now below 0.2% despite increasing lesion complexity.[1] Stents readily scaffold otherwise occlusive procedure-induced dissections. However, the metallic surfaces of stents can themselves stimulate occlusive thrombosis. Until the stent is entirely covered by endothelial tissue (a process requiring up to several weeks), combined oral antiplatelet therapy with ASA plus clopidogrel or ticlopidine is required.[2]

Restenosis develops from the additive effects of mechanical vessel recoil plus neointimal proliferation (a healing response). Clinical trials have shown that stents substantially reduce restenosis but do not eliminate it,[3–5] in large part because stents prevent recoil but do not reduce neointimal proliferation. Data from the British Columbia Cardiac Registries have confirmed the broad effectiveness of stents in most PCI patients and demonstrated that the adoption of routine stenting during the mid-1990s in patients with or without ACS was accompanied by a significant reduction in the incidence of clinical restenosis to below 20% overall, and below 15% among those with stents.[1] Currently, over 90% of patients undergoing PCI in British Columbia receive at least 1 stent.

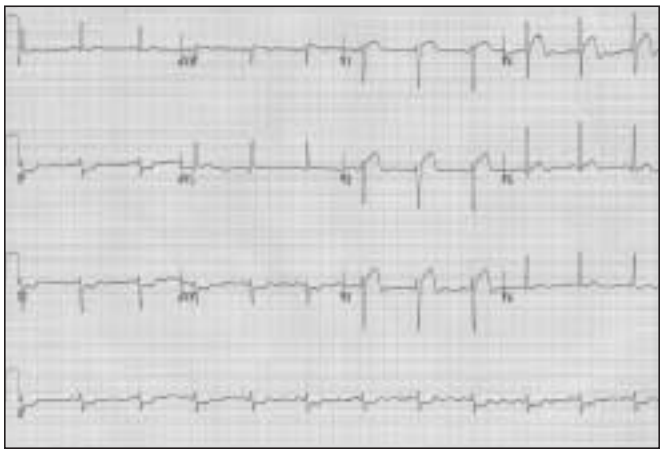


Fig. 1: Case 1. Initial electrocardiogram (ECG), showing ST-segment elevation in leads V_1– V_4, indicative of evolving anterior myocardial infarction. ST-segment depression is evident in leads II, III and aVF.

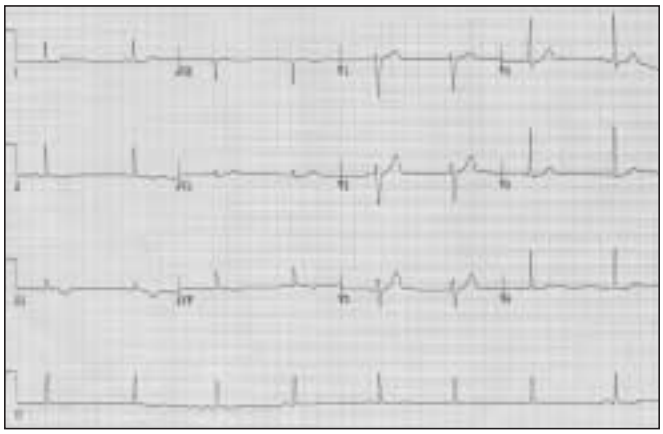
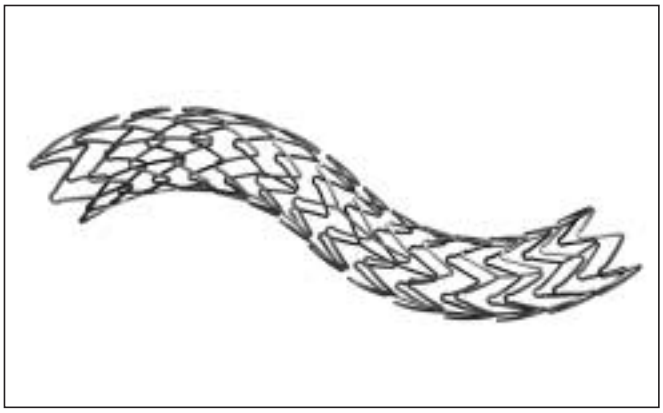


Fig. 2: Case 2. Initial ECG, showing symmetrical T-wave inversion in leads II, III and aVF.

Glycoprotein IIb/IIIa platelet inhibitor therapy

Because PCI itself induces vascular injury, a highly thrombotic coronary environment develops during PCI, even in patients without pre-existing ACS. Not surprisingly, the use of potent glycoprotein IIb/IIIa platelet inhibitors during angioplasty in patients with or without ACS has been shown to result in improved short- and long-term outcomes.[6,7] More recently, abciximab and eptifibatide have been tested during stent procedures in placebo-controlled trials that included many patients from Canadian centres (the EPISTENT trial[8] and ESPRIT trial[9]). Both drugs were associated with substantial early reductions in risk of death, myocardial infarction or urgent



Courtesy: Guidant Corporation, Santa Clara, Calif.

Fig. 3: Stents such as this one (the ACS MULTILINK DUET balloon-expandable coronary stent) are commonly used during percutaneous transluminal coronary angioplasty (PTCA) in Canada. The stent is laser-cut from tubular stainless steel in a design providing radial strength and longitudinal flexibility. Stent diameter is determined by the balloon diameter used for deployment.

repeat revascularization.[8,9] Early data from a recently completed head-to-head trial comparing tirofiban and abciximab during coronary stent procedures showed abciximab to be slightly superior (30-day mortality, myocardial infarction or emergency revascularization rate 7.6% with tirofiban and 6.0% with abciximab, $p = 0.037$).[10] However, the small absolute difference between agents suggests an important beneficial effect from tirofiban as well. Longer-term data with abciximab and stenting from the EPISTENT study indicates an important sustained reduction in the rates of both myocardial infarction and death through 1 year (Fig. 4) (unpublished data). Whether the benefits of eptifibatide and tirofiban will prove equally durable is yet unknown.

Although glycoprotein IIb/IIIa inhibitors improve PCI outcomes in patients with a wide variety of indications and clinical characteristics, a consistent preferential benefit exists in those with coexisting ACS.[11] Elevated cardiac troponin levels (a marker of intracoronary thrombosis and thromboembolism) further refine the subset of patients most likely to benefit from PCI. Of particular importance to referring physicians in Canada are subgroup analyses from trials testing the administration in the coronary care unit (CCU) of eptifibatide and tirofiban in non-ST-segment elevation ACS: patients subsequently selected for angiography and pre-discharge PCI were found to benefit the most.[12,13] Thus, a rational approach to using glycoprotein IIb/IIIa inhibitors in non-ST-segment elevation ACS is to initiate therapy in patients with an elevated level of troponin (or other markers of high risk) when a decision is made to refer for angiography and possible PCI.

Treatment of ST-segment elevation acute coronary syndrome

Direct angioplasty

Direct angioplasty refers to the primary use of PCI (instead of thrombolysis) to achieve reperfusion in patients with ST-segment elevation ACS. The principal mechanism of thrombolytic therapy in such patients is myocardial salvage through rapid restoration of antegrade epicardial flow. Widely used thrombolytic regimens currently achieve complete coronary reperfusion by 90 minutes in about 55%–60% of cases and partial reperfusion in an additional 20%–25%.[14] Direct PCI generally yields significantly higher rates of complete reperfusion than does thrombolytic therapy. In addition, the underlying coronary stenosis, a substrate for recurrent ischemia, is relieved early in the course of the infarction. The potential of these attributes of direct PCI to improve clinical outcome beyond that achievable with thrombolytic therapy has led to randomized clinical trials directly comparing these 2 approaches. The largest single trial of this type (GUSTO IIB) compared direct angioplasty and accelerated tissue plasminogen activator (tPA) therapy in 1138 patients.[15] The primary end point (a composite of death, nonfatal reinfarction or nonfatal disabling stroke at 30 days) occurred in 9.6% of the angioplasty patients and 13.7% of the tPA patients ($p = 0.03$), for a 33% risk reduction. A secondary analysis at 6 months still favoured angioplasty, although the difference between the 2 strate-

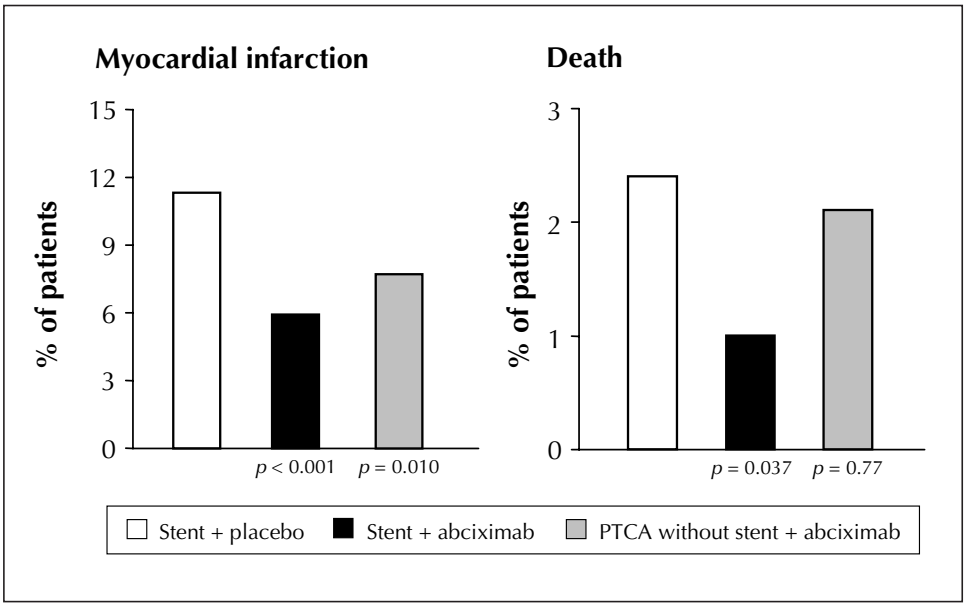


Fig. 4: One-year results from the EPISTENT study, demonstrating the synergy of stenting with intense platelet inhibition using abciximab. Rates of myocardial infarction and death were lower with stenting plus abciximab than with either stenting plus placebo or abciximab plus PTCA without stenting.

gies was no longer statistically significant (primary end point occurred in 14.1% of angioplatsy patients and 16.1% of tPA patients).[15] A meta-analysis of GUSTO IIB plus 9 other randomized trials comparing direct angioplasty and various thrombolytic regimens also supports the superiority of direct PCI when practised in centres with on-site coronary catheterization and experienced operators.[16] The widely used end point of 30-day mortality or reinfarction was significantly lower among patients treated with direct PCI than among those given thrombolytic therapy (Fig. 5).

Because of concerns regarding the safety of stents in the highly thrombotic setting of ST-segment elevation myocardial infarction, direct angioplasty with stents has been tested against angioplasty without stents in randomized trials.[17,18] In the Stent-PAMI study[17] the prespecified primary 6-month end point (a composite of death, nonfatal myocardial infarction, stroke or target-vessel revascularization) occurred less frequently with stenting than without stenting (12.6% v. 20.1%, $p < 0.01$). However, a nonsignificant trend toward excess mortality was noted (stenting 5.4% v. no stenting 3.0%). Although likely attributable to chance alone, it has also been suggested that vigorous plaque and thrombus compression associated with direct PCI in general, and stent deployment in particular, may increase athero- and thromboembolic plugging of downstream micro-vessels. Such a phenomenon could paradoxically increase infarct size. This has led to investigations testing the administration of glycoprotein IIb/IIIa inhibitors during direct PCI.

Mechanistic studies have shown that the addition of glycoprotein IIb/IIIa inhibitors during stent-based direct PCI do indeed improve coronary blood flow and reduce infarct size.[19–21] Preliminary data from the CADILLAC trial, comparing angioplasty with stents, and abciximab with no abciximab, confirmed an overall benefit of abciximab.[22] However, the trial was not sufficiently powered to demonstrate an advantage for abciximab in the stent subgroup alone. The primary 6-month end point (a composite of death, target-vessel revascularization, myocardial infarction or disabling stroke) was 19.3% for PTCA plus placebo, 15.2% for PTCA plus abciximab, 10.9% for stent plus placebo and 10.8% for stent plus abciximab). The rates of death were 4.3%, 2.3%, 2.8% and 3.8% respectively. Current practice of direct PCI in Canada and elsewhere now generally involves stenting with concurrent administration of a glycoprotein IIb/IIIa inhibitor.

The applicability in Canada of the findings from these direct PCI trials is limited. For most patients direct PCI is simply not available without time-consuming transportation. Except in highly organized local referral programs, prompt administration of thrombolytic therapy is almost certainly superior to significantly delayed direct PCI. The observation that direct PCI and thrombolytic therapy re-

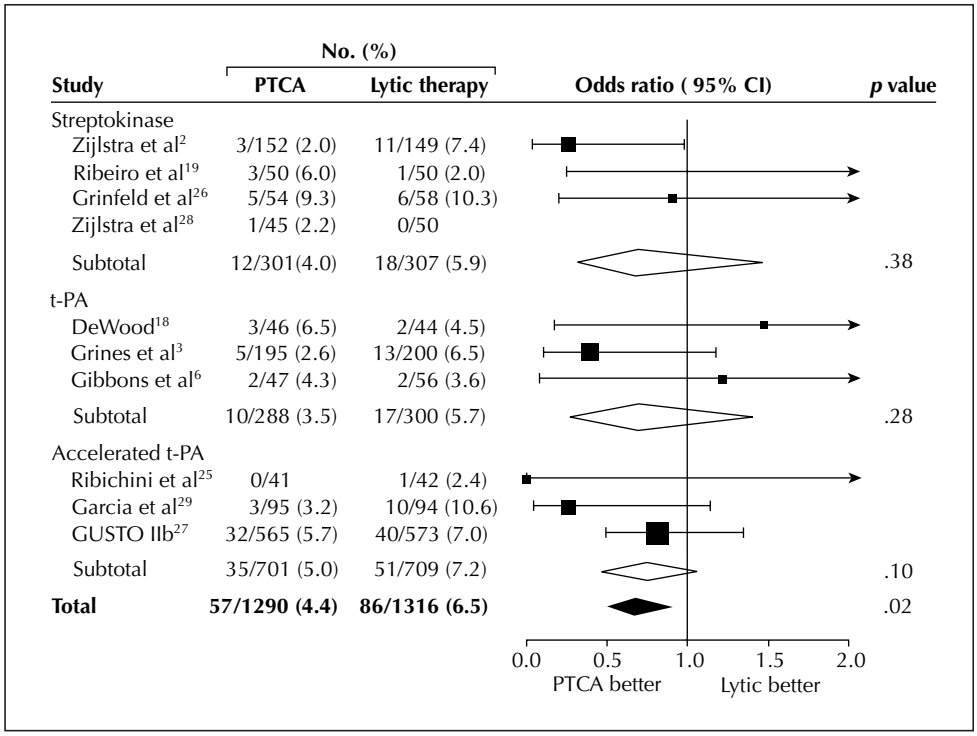


Fig. 5: Mortality at the end of the study period in 10 trials comparing direct PTCA with thrombolytic therapy. The rates for each study are grouped by thrombolytic regimen. Odds ratios with 95% confidence intervals (CIs) are plotted on the right. [Reprinted, with permission, from Weaver WD et al.[16] © 1997, American Medical Association.]

sult in similar outcomes when applied in community-based, unselected, actual practice verifies the vital role of thrombolysis in most Canadian hospitals.[23] Community physicians should be aware, however, that emergency transport for direct PCI may be appropriate for patients with ST-segment elevation ACS and contraindications to thrombolytic therapy. Patients at increased risk of thrombolytic complications (e.g., recent ischemic stroke or surgery, prolonged cardiopulmonary resuscitation and coumadin therapy) may yet be safely treated with heparin and ASA, as PCI requires. On the other hand, patients who are unable to receive therapeutic heparin and ASA owing to active major bleeding are usually not candidates for either thrombolytic therapy or direct PCI.

Rescue angioplasty

Angioplasty following failed thrombolytic therapy is termed "rescue" angioplasty. Thrombolytic failure should be diagnosed clinically when, upon completion of thrombolytic infusion (usually 90 minutes after initiating therapy), ST-segment elevation fails to resolve below half its initial magnitude. This emphasizes the importance of routinely obtaining and reviewing ECGs immediately after thrombolytic therapy, even when the chest pain has disappeared (pain resolution can often be confounded by the administration of narcotics or other analgesics).

Successful rescue angioplasty after failed thrombolytic therapy achieves clinical outcomes that approach those of successful thrombolytic therapy.[24] These compelling observations have established rescue angioplasty as standard practice (even when patient transportation is required) yet have hampered its rigorous evaluation in controlled trials. A small randomized trial performed in the early 1990s evaluated rescue angioplasty in the setting of acute anterior myocardial infarction.[25] Although not powered to evaluate clinical outcome, the strategy of rescue angioplasty resulted in improved left ventricular ejection fraction on exercise.

The optimum timing for referral for rescue angioplasty when patient transportation is required is unclear. We believe the timing should be individualized according to patient and system factors. For institutions located near coronary catheterization centres, referral should be initiated as soon as thrombolytic failure is suspected 90 minutes after its initiation. Earlier referral is appropriate when hemodynamic compromise is present: patients with acute heart failure face predictably lower rates of thrombolytic success than do stable patients. For institutions located hours from catheterization centres, routine early transportation of high-risk patients (those with heart failure or ECG evidence of extensive myocardial infarction) after initiation of thrombolytic therapy is a possible approach. Stable, low-risk patients with small and uncomplicated infarctions may not warrant transportation despite clinically evident thrombolytic failure.

Cardiogenic shock complicating myocardial infarction

The SHOCK study randomly assigned patients with cardiogenic shock developing up to 36 hours following acute myocardial infarction to either emergency revascularization (with angioplasty or bypass surgery) or initial medical stabilization.[26] Follow-up at 1 year showed that the survival rate was substantially higher among patients undergoing early revascularization than among those receiving medical therapy (47% v. 34%, $p = 0.025$) (Fig. 6). Patients under 75 years of age enjoyed a particular survival advantage from emergency revascularization. Thus, patients with cardiogenic shock up to 36 hours after myocardial infarction should be offered emergency transportation, regardless of transportation time, if on-site facilities for emergency revascularization are unavailable.

Angioplasty during convalescence from myocardial infarction

Although it can be argued that routine angiography following the initial treatment of acute myocardial infarction can provide important prognostic information and aid in decision-making, routine angioplasty in patients who are asymptomatic with negative functional test results has been shown to lack benefit. However, when objective ischemia is present following thrombolytic therapy, there is demonstrated benefit to routine revascularization. In the DANAMI study 1008 patients were randomly assigned to either routine angiography and revascularization or medical management alone.[27] At 1 year the combined end point of death, reinfarction or unstable angina was reduced from 29.5% to 15.4% with the invasive strategy, a benefit that persisted at least to 3 years. Thus, the routine use of

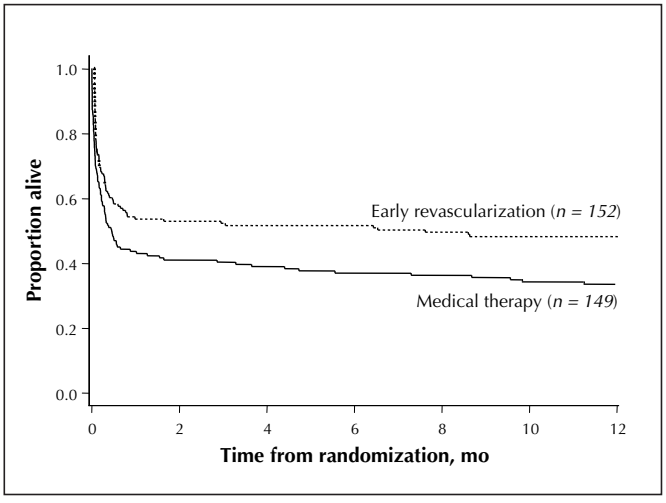


Fig. 6: Kaplan–Meier survival at 1-year follow-up in the SHOCK study. Early revascularization resulted in a 39% relative reduction in mortality. [Reprinted, with permission, from Hochman et al.[26] © 2001, American Medical Association.]

catheterization and appropriate revascularization can be advocated at least for patients in whom there is objective ischemia or spontaneous recurrent angina and for those with heart failure or evidence of a major electrical or mechanical complication.

Treatment of non-ST-segment elevation acute coronary syndrome

When and whom to refer to cardiac catheterization and revascularization from among patients with non-ST-segment elevation ACS has long been debated and studied, and a wide range of practice exists. Because non-ST-segment elevation ACS is currently the commonest diagnosis on admission to CCUs, changes in threshold for invasive management of these patients will greatly increase the total demand for cardiac catheterization, angioplasty and bypass surgery. Reciprocally, the per capita resources committed to these procedures within a health care system may influence the threshold for referral through triage processes and wait-listing.

In the past decade, 4 large, well-designed randomized clinical trials have directly addressed the role of routine angiography and revascularization (invasive management) in non-ST-segment elevation ACS.[28–31] In each trial patients were randomly assigned to either routine invasive management or conservative management (in which invasive management was reserved for recurrent or provocable ischemia). Despite their broad similarities, these trials had many key differences in design, including size, enrolment criteria, concurrent medical therapy, interval to angiography and methods for functional testing in conservatively treated patients (Table 1). Furthermore, enrolment in the various trials spanned a decade during which important improvements in medical therapy, PCI procedures and bypass surgery became widely implemented.

The earlier trials (TIMI-IIIB[28] and VANQWISH[29]) collectively enrolled 2393 patients from 1989 through 1995. These 2 trials showed that routine invasive management reduced symptom burden and hospital length of stay but offered no advantage with respect to subsequent risk of death or myocardial infarction. The 2 more recent trials (FRISC II[30] and TACTICS TIMI-18[31]), which collectively enrolled 4677 patients from 1996 through 1999, confirmed the superiority of routine invasive care with respect to symptom control and demonstrated significant reductions in the composite end point of death, myocardial infarction or urgent revascularization in patients managed invasively compared with those managed conservatively (Figs. 7 and 8). Moreover, at 1-year follow-up, the FRISC II trial showed an independent reduction in mortality. These powerful recent trials better reflect current medical therapy and current PCI and surgical techniques.

A strict interpretation of the FRISC II and TACTICS TIMI-18 trials would conclude that routine angiography

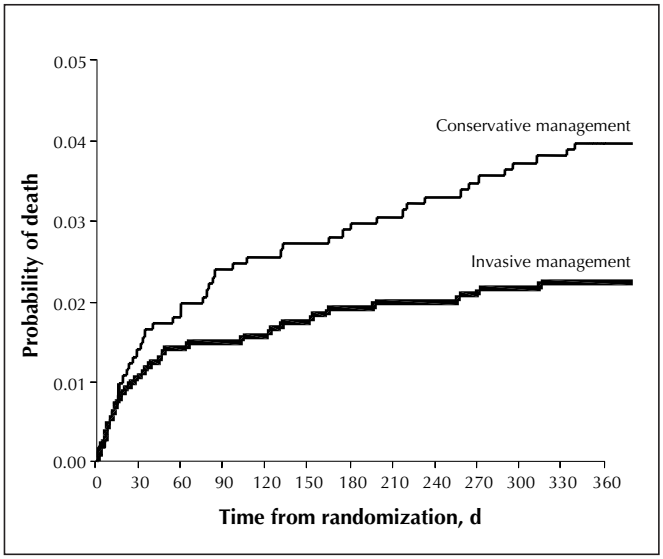


Fig. 7: Kaplan–Meier survival at 1-year follow-up in the FRISC II trial. Routine invasive management (coronary angiography and revascularization) resulted in lower rates of death than did routine conservative management. [Reprinted, with permission, from Wallentin L et al.[30] © 2000 The Lancet Ltd.]

Table 1: Trials of routine invasive management in non-ST-segment elevation acute coronary syndromes

Variable	TIMI-IIIB trial[28] *n = 1473*	VANQWISH trial[29] *n = 920*	FRISC II trial[30] *n = 2457*	TACTICS TIMI-18 trial[31] *n = 2220*
Study period	1989–1992	1993–1995	1996–1998	1997–1999
Median time to angioplasty,* d	1.5	2	5	1
Functional testing†	Nuclear	Nuclear/echo	Electrocardiography	Nuclear/echo
Revascularization rate, %				
Invasive arm	61	44	78	60
Conservative arm	49	33	37	36
Antithrombotic treatment	ASA, heparin	ASA, heparin	ASA, dalteparin	ASA, heparin, tirofiban
Stent rate,‡ %	0	< 10	60	> 90

*Invasive therapy arm.
†Conservative therapy arm.
‡Proportion of patients undergoing percutaneous coronary intervention who received a stent.

and revascularization is indicated in nearly all patients with non-ST-segment elevation ACS. However, patients enrolled in these studies were required to meet entry criteria that bestowed risk: elevated cardiac enzyme levels, ischemic ECG changes or known pre-existing ischemic heart disease.

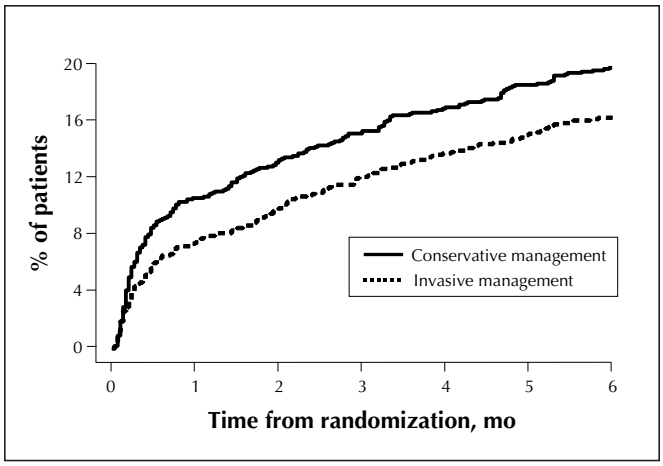


Fig. 8: Incidence of primary end point (composite of death, myocardial infarction or readmission to hospital with acute coronary syndrome) among patients enrolled in the TACTICS TIMI-18 trial. Invasive management reduced the incidence by 22% at 6 months (conservative management 19.4% v. invasive management 15.9%, odds ratio 0.78, 95% confidence interval 0.62–0.97). [Reprinted, with permission, from Cannon et al.[31] Copyright © 2001, Massachusetts Medical Society. All rights reserved.]

The results from these recent randomized trials will no doubt continue to increase demand for invasive management of non-ST-segment elevation ACS. However, risk stratification based on history, ECG changes, troponin levels, clinical course and functional test results will remain a sound approach to selecting patients for angiography and revascularization.[32]

When patient transportation over substantial distances is required, its cost and inconvenience will continue to make thoughtful risk stratification especially important. The many patients with one or more high-risk features who present to community hospitals must be consistently and accurately recognized by their primary care physicians and provided timely access to appropriate tertiary care facilities.

The incidence of non-ST-segment elevation ACS is high, accounting for most admissions to CCUs. Thus, a small increase in the proportion of ACS patients who are selected for angiography levers a large absolute increase in the demand for related services. The implications for Canada regarding education for physicians, transportation infrastructure, tertiary acute care cardiac beds, and catheterization and revascularization procedure volumes are substantial.

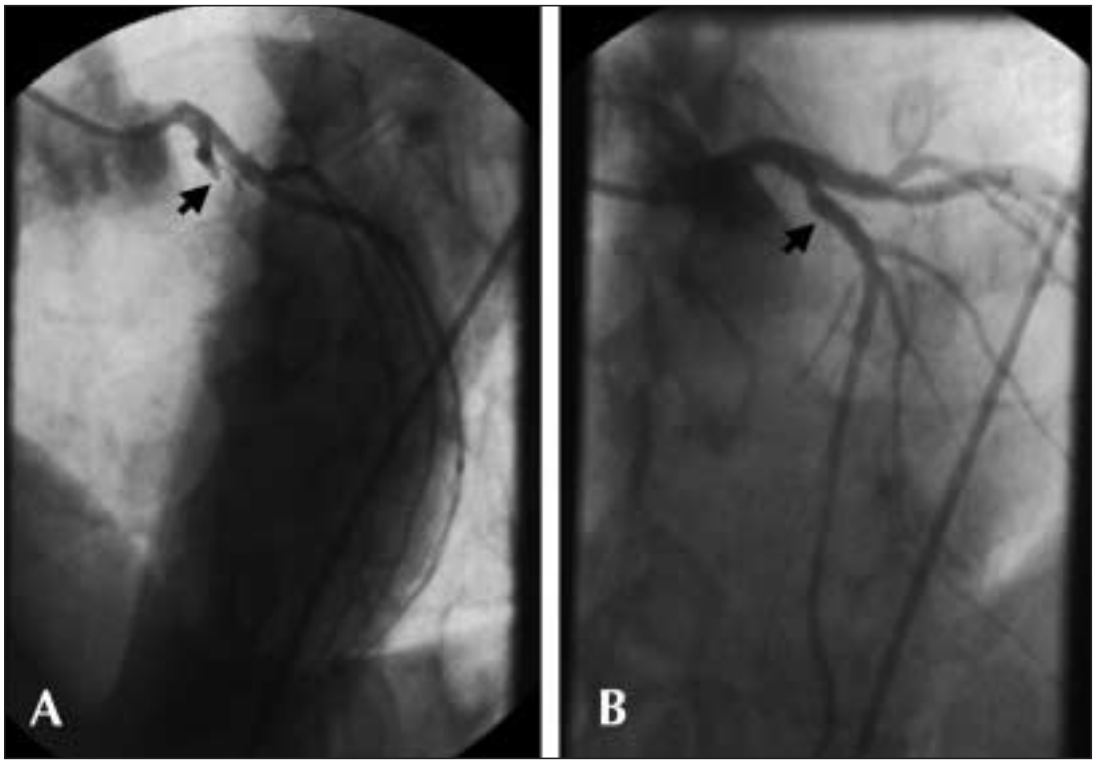


Fig. 9: Case 1. A: Emergent coronary angiography, showing complete occlusion of left anterior descending coronary artery (arrow). B: Normal blood flow is re-established following direct PTCA with stenting.

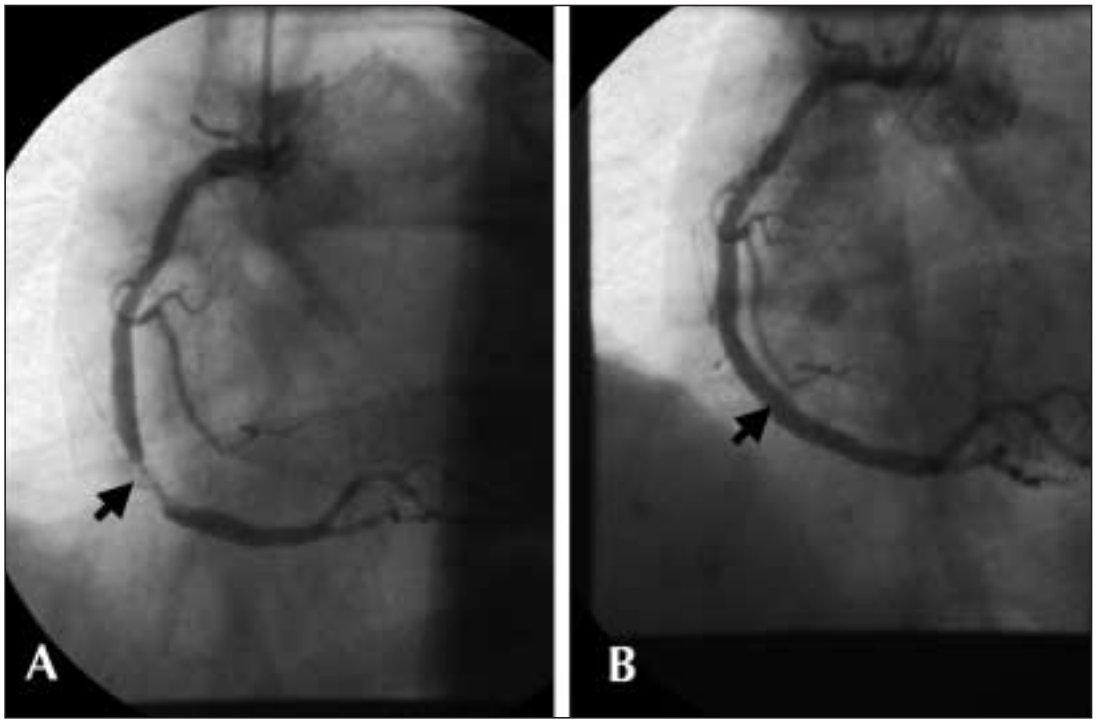


Fig. 10: Case 2. A: Coronary angiography 24 hours after admission, showing high-grade stenosis in middle of right coronary artery with associated haziness consistent with intraluminal thrombus (arrow). B: Vessel is widely patent with improved distal blood flow following PTCA with stenting.

The cases revisited

Mr. F has acute ST-segment elevation anterior myocardial infarction. He has presented to a hospital with on-site cardiac catheterization, and direct PTCA is chosen as the preferred acute reperfusion strategy. In the emergency department he receives 325 mg of ASA, a weight-adjusted bolus of unfractionated heparin intravenously, an oral loading dose of 300 mg of clopidogrel, 15 mg of metoprolol intravenously over 15 minutes, oxygen and morphine. Coronary angiography performed 55 minutes after initial presentation shows complete occlusion of the proximal left anterior descending coronary artery (Fig. 9A) and a coincident stenosis of the right coronary artery. Left ventricular angiography shows akinesis of the anterior wall of the left ventricle, with a reduced ejection fraction of 45%. Following administration of abciximab, a stent-based procedure is performed, which relieves the occlusion and restores normal blood flow (Fig. 9B). For safety, treatment of the stenosis of the right coronary artery is deferred until day 5. Mr. F is discharged on day 6 with the following drug regimen: ASA 81 mg/d, clopidogrel 75 mg/d for 4 weeks, coumadin adjusted for an international normalized ratio of 2.5 for 6 months, ramipril, atenolol and simvastatin.

For Mr. G, who has non-ST-segment elevation ACS, an invasive approach to management is indicated. His high-risk features (ischemic ECG changes and elevated troponin level) justify this decision despite the absence of spontaneous recurrent ischemia and functional testing. Because the patient was admitted to a hospital with on-site cardiac catheterization, coronary angiography is performed 24 hours after admission. Oral and intravenous therapy was continued without interruption before the procedure. A high-grade stenosis is revealed in the middle of the right coronary artery, with associated haziness (suggesting intraluminal thrombus) and impaired blood flow (Fig. 10A). Left ventricular angiography shows mild inferior hypokinesis, with a normal ejection fraction. Stenting performed during the same procedure results in a widely patent vessel with improved distal blood flow (Fig. 10B). Tirofiban therapy is continued for 12 hours after the procedure, and Mr. G is discharged the next morning with the following drug regimen: ASA 325 mg/d, plavix 75 mg/d for 28 days, an angiotensin-converting-enzyme inhibitor and his previously prescribed statin lipid-lowering agent.

Competing interests: None declared.

Contributors: Dr. Buller was the primary author. Dr. Carere wrote subsections of the manuscript and contributed to the revision of the overall manuscript.

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A patient information sheet appears on the next page.

Appendix

Questions and answers on catheter-based procedures for acute coronary syndromes

An information sheet for patients

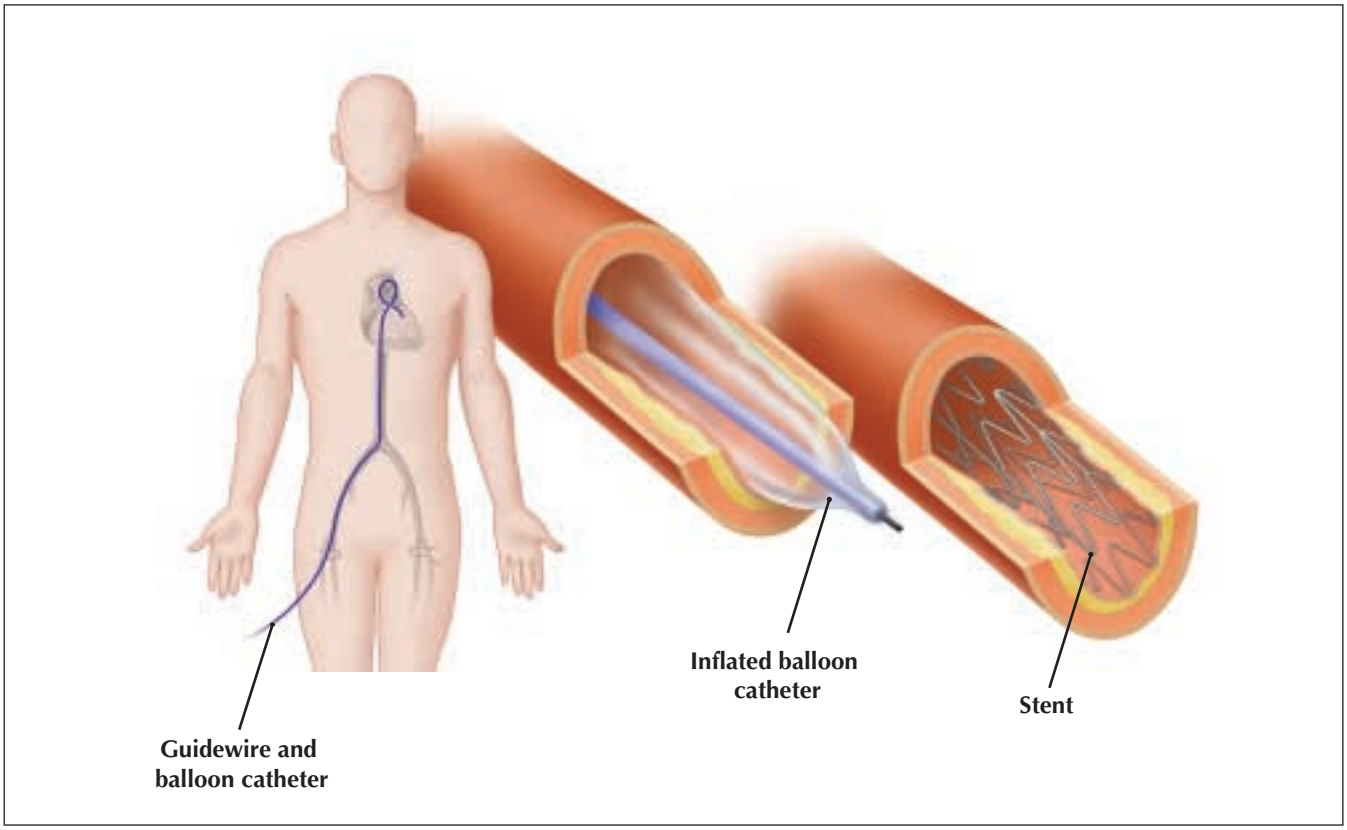
What are acute coronary syndromes?

This term refers to a collection of heart conditions that occur suddenly when a clot, or *thrombus*, develops within one or more *coronary arteries* (the arteries supplying the heart muscle with blood and oxygen) and blocks an already narrowed artery. The narrowing in the artery is the result of long-standing fatty deposits. Acute coronary syndromes range in severity from *unstable angina* (chest pain caused by reduced blood flow to the heart) to *acute myocardial infarction* (a heart attack).

How are these conditions treated?

Any condition that might damage the heart muscle and interfere with the heart's pumping function must be treated as quickly as possible. Four general treatment strategies are used to restore blood flow and minimize damage to the heart muscle:

1. Heart-protecting medications that slow the heart and reduce its need for blood.
2. Medications that prevent new clots from forming in the coronary arteries.



Christine Kenney

Catheter-based procedures are performed, under x-ray guidance, to open narrowed or blocked coronary arteries (the arteries supplying heart muscle with blood and oxygen). A catheter is inserted under local anesthetic into a major artery at the top of the leg (femoral artery) or arm (radial or brachial artery). It is then steered through the central arteries to the heart. A tiny balloon-tipped catheter is then moved up to the point of severe narrowing, and the balloon is inflated to open up the artery. A permanent, tube-shaped metallic scaffold (stent) is often used to keep the unclogged artery open. The stent is mounted on the balloon-tipped catheter and is put in place by inflation of the balloon. All equipment except the stent is removed at the end of the procedure.

3. Medications that dissolve existing clots in the coronary arteries.
4. Procedures that relieve the underlying narrowing of the artery. These include procedures that rely on a *catheter* (a hollow, flexible tube that can be guided through an artery).

What happens during a catheter-based procedure?

A variety of catheter-based procedures are available. These procedures are referred to in general as *angioplasty* — a word that means "blood vessel repair." During such a procedure, a catheter is inserted into an artery in the groin or arm and is moved up to the blocked heart artery; its movement is guided with the help of x-ray monitoring. A special balloon at the end of the catheter is then inflated to open the narrowed artery and flatten the clot against the artery wall. Often a tube-shaped wire scaffold device called a *stent* is then inserted to keep the unclogged artery open. Anti-clotting drugs are often used during these procedures.

Are there risks associated with catheter-based procedures?

Yes. Complications may occur during or after any medical procedure, particularly when a vital organ such as the heart is being treated. For instance, during angioplasty, an artery can become completely blocked, which would trigger a heart attack. In addition, bleeding, stroke and other complications may occur. Although major complications are very rare, you should be aware of potential problems. You and your doctor will need to weigh the possible benefits against the risks of undergoing a catheter-based procedure.

Are catheter-based procedures performed at all medical centres?

No. In Canada, only major centres have the special equipment and the experienced medical team required to perform catheter-based procedures or open-heart surgery. Depending on your condition, your doctor will determine whether you should be transported to such a centre before or after you undergo tests and receive drug therapy.

What if I am too sick to be transported to a centre where catheter-based procedures are performed?

An elaborate ground and air ambulance network exists in Canada to provide urgent transportation of patients. However, when such transportation is not possible, you will still likely benefit greatly from clot-dissolving medications, anti-clotting medications and other heart-protecting medications, which are available in all centres.