Stable angina: drugs, angioplasty or surgery?

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Stable angina is a common condition with a good overall prognosis and annual mortality is 2-4%, whatever treatment is employed. Medical therapy with nitrates, /?-blockers, calcium antagonists and lipid-lowering agents is appropriate as first-line therapy in those patients not specifically identified as being at risk by exercise testing and/or angiography. Dosage should be optimized. Coronary artery bypass grafting appears to improve prognosis in those at risk when compared with medical therapy but the trials are old and do not take into account major advances in medical therapy nor the use of arterial conduits in coronary artery bypass grafting (CABG).

Percutaneous transluminal coronary angioplasty (PTCA) relieves symptoms when medical therapy is ineffective but its role as an initial therapy has not been established, nor does it compare favourably with CABG with regard to the degree of revascularization and subsequent re-intervention or need for additional anti-anginal drugs. There are little substantial data on prognostic effects. PTCA is, however, less traumatic, less expensive and associated with a quicker recovery than CABG, providing a viable alternative for symptomatic (not prognostic) benefit in appropriately selected and informed patients.

Medical therapy, PTCA and CABG should not be seen as competitive but complementary strategies. Optimal utilization of all three treatment modalities, either alone or in combination, can provide substantial symptomatic relief for the angina patient.

Key Words: Angina, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty, /?-blockers, nitrates, calcium channel blockers.

Introduction

The goals of treatment for patients with stable angina pectoris, achieved by both identifying those at risk and intervening to modify the risk, are to improve symptoms and thereby quality of life, to prolong survival and, ideally, a combination of the two.

Patients with symptoms that are refractory to medical therapy should undergo angiography, with a view to intervention by PTCA or CABG. This statement assumes that 'failed' medical treatment equates with 'failed optimal' medical treatment, which may not be the case, as drugs are frequently not titrated to their established maximally effective doses, e.g. atenolol 50 mg daily is frequently not increased to 100 mg daily.

Independently of symptoms, however, patients may need angiography to identify their risk status. Prognosis is influenced by the nature, location and extent of coronary artery disease (CAD), as well as the quality of left ventricular function. Evidence for a beneficial effect on mortality is only available for CABG and relates to specific anatomical subsets, comprised of left main stem disease, three-vessel disease, with or without reduced left ventricular function, and two- or three-vessel disease involving the proximal left anterior descending (LAD) artery. The three major trials of surgery vs medical therapy which established these findings are now over 20 years old. Since these data were published, significant advances in medical therapy, and to a lesser extent, CABG (the use of arterial conduits such as the internal mammary artery), have been made. Thus, it cannot be claimed that medical therapy at the time of these trials was optimal. For example, there was no standardized dose of /?-blockade, aspirin was not routinely in use and lipid-lowering therapy was hardly in existence.

There are many problems with the data available, both old and new, and it is easy to let our perceptions of the data (based on logic and reason) overrule the scientific facts. Randomized trials become necessary when there are reasonable doubts as to the meaning of data. The problem with stable angina is the increasing, rather than the decreasing, number of such doubts.

If we look at the different modalities of treatment, comprising either drugs, PTCA or CABG, we should expect to be able to review trials of drugs vs CABG, drugs vs PTCA, drugs vs PTCA or CABG and PTCA vs CABG. The fact that a complete set of data is...
missing has not deterred investigators from comparing PTCA with stents\(^4\) without including a drugs group in the comparison. One assumes that this is on the grounds that restenosis, as the major problem associated with PTCA, needs to be addressed before the exact role of PTCA can be defined. This not unreasonable objective should not exclude, however, a direct trial of stents vs medical therapy unless the data on PTCA are overwhelmingly positive vs medical therapy or CABG.

It therefore becomes imperative to ask, what information do we have and how good is it?

**Medical therapy**

It is an inescapable fact that in all clinical trials of anti-anginal drugs there is a significant benefit both subjectively, upon anginal attack rates and glyceryl trinitrate consumption, and objectively, upon exercise time or time to 1 mm ST depression on a treadmill or bicycle exercise ECG.

This applies to \(\beta\)-blockers, calcium antagonists, isosorbide mononitrate and the new potassium channel activator nicorandil. Combination therapy of two drugs, one from each class, may provide additional improvement but adding a third agent may provide little extra benefit and may in some instances cause deterioration\(^5\).

Clinical trials use carefully selected cases and usually employ optimal doses of the drugs under study. However, in clinical practice, optimal doses may not be employed, particularly with \(\beta\)-blockers, largely because of unnecessary anxieties about increasing the dose in the presence of a resting bradycardia\(^6\). In Fig. 1 an algorithm for combination therapy is outlined, which is both practical and safe. Alternatives would include diltiazem 60–120 mg three times daily or verapamil 40–160 mg three times daily plus isosorbide mononitrate. Verapamil should not be combined with \(\beta\)-blockade because of unpredictable adverse interactions on the conducting system. Diltiazem can be used in combination with \(\beta\)-blockers but caution is advised because of an additive effect on the sinus node, occasionally leading to a significant symptomatic bradycardia. The long-acting dihydropyridines, such as amlodipine, avoid conduction system interactions with \(\beta\)-blockers, are less negatively inotropic and should be preferred in combination.

Medical therapy of angina must also include aspirin, which can reduce the risk of vascular death, stroke and myocardial infarction (MI) by 25%\(^7\). Aspirin was not used routinely in the earlier CABG vs medical therapy trials.

Lipid-lowering therapy has also been shown to have a significant impact on subsequent cardiovascular events, as well as slowing the progression and inducing regression of CAD\(^8\). For example, the Scandinavian Simvastatin Survival Study (4S) Group trials\(^9\) reported a 30% reduction in death and MI associated with the use of the lipid-lowering agent simvastatin (Fig. 2).

Medical therapy, in addition to lifestyle change (reduction or cessation of smoking, weight loss, reduced intake of saturated fat), has progressed significantly over the last 25 years and it seems unreasonable to assume that all the data from 'old' trials are applicable now. Nonetheless, we have no convincing evidence that conventional medical therapy improves survival in stable angina patients. \(\beta\)-blockers, however, improve survival post infarction and there is evidence that infarcts occurring in patients taking \(\beta\)-blockers are smaller\(^9\).

### Medical therapy vs coronary artery bypass grafting

CABG undoubtedly relieves anginal pain when compared with medical therapy and when symptoms persist in spite of optimal medical treatment. The operative risk of death is of the order of 2% but may rise to 5% in the elderly and for complex and/or repeat procedures. Perioperative infarction occurs in 8–10% of cases but is usually minor and well tolerated. Morbidity includes sternal and back pain, which usually resolves over 2–3 months, and neurological problems, which are transient in 5–6% but permanent in 1–2% of patients.

Although 80% of patients are free of symptoms up to 5 years after surgery, some will still be taking
antianginal drugs. Only 50% are symptom free at 10 years but this may improve with the greater use of arterial conduits and more attention to lipid-lowering therapy.

CABG, therefore, has an important role in symptomatic relief. How does it compare with medical therapy with regard to prognosis?

The Veterans Administration Study took place between 1970 and 1974. There was a high operative mortality of 5–8%, reflecting the early days of surgery and the lack of expertise in the technique. Moreover, there was a poor vein patency rate of 69% at 1 year, reflecting the technical limitations. In spite of these reservations, surgery improved survival for left main stem disease. However, it is important to point out that, at the time of this trial, the options for medical therapy were very limited. As time has moved on and techniques have improved, this study has little relevance to current practice.

The European Coronary Surgery Study (1973–1976) is perhaps the most relevant study to current practice, since it included patients with mild to moderate angina. Better medical therapy was available with β-blockade but it was not standardized. Up to 70% of patients had suffered from a previous MI. Operative mortality was 3–6%, which is similar to current levels and which, in turn, reflects a rise in treatment of more complex or difficult surgical cases compared with the easier single- and double-vessel lesions, now 'cherry picked' by PTCA operators. Vein patency was 77% at 18 months.

At 5 years, 7.6% of those undergoing surgery had died, compared with 16.2% of those undergoing medical therapy, representing a highly significant 53% reduction. The benefit was most impressive for left main stem disease (Fig. 3), two-vessel disease, if one lesion was in the proximal LAD artery, three-vessel disease, with or without good left ventricular function, and in the presence of an abnormal resting or exercise ECG.

At 10-year follow-up, surgical benefit persisted and was similar in those who did or did not continue to smoke (Fig. 4). This indicates that smokers should not be denied operative intervention and that therapeutic decision making should therefore be on the basis of evidence.
Stable angina: drugs, angioplasty or surgery

The Coronary Artery Surgery Study (CASS) excluded left main stem disease. Patients with no angina or minimal symptoms were included and, of 16,262 cases assessed, only 780 were randomized, thereby identifying a highly selected group of patients. At 5 years, 92% and 95%, respectively, of those undergoing medical and surgical treatment survived, representing a non-significant difference between treatments. Operative mortality was 1.4% and graft patency at 60 days was 90%. Medical therapy was not standardized and only 43% used β-blockers. Surgery improved prognosis at 6 years if there was three-vessel disease with reduced left ventricular function.

Rationalizing these trials so that they are relevant to practice today is not straightforward, since different patients are being evaluated, the medical therapy used is at times unclear and certainly outdated and surgical techniques have improved. Furthermore, any therapeutic conclusions drawn would be made on the basis of subset analysis, which would be severely criticized if one were, for example, evaluating drugs alone. In addition, the latter two trials were weighted against surgery as medical patients who needed surgery for relief of symptoms (i.e., medical failures) remained classed as 'medical', even though they may have benefited from surgery, and those randomized to surgery who refused an operation were still classified as 'surgical' even though they were treated medically.

The best practical guidelines for treatment and prognosis are: (1) an abnormal exercise ECG may
Table 1 Angioplasty compared to medicine (ACME) results*

<table>
<thead>
<tr>
<th></th>
<th>Drugs</th>
<th>PTCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients randomized</td>
<td>107</td>
<td>105</td>
</tr>
<tr>
<td>Number of patients with PTCA success</td>
<td>—</td>
<td>80/100</td>
</tr>
<tr>
<td>Number of patients with emergency CABG</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Number of patients with repeat PTCA by 6 months</td>
<td>11 (12)</td>
<td>16</td>
</tr>
<tr>
<td>Number of patients with CABG</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Number of patients with myocardial infarction</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Number of patients free of angina (%)</td>
<td>47/102 (46%)</td>
<td>61/96 (64%)†</td>
</tr>
<tr>
<td>Exercise increase</td>
<td>0-5 min</td>
<td>2-1 min‡</td>
</tr>
</tbody>
</table>

(*Reproduced from Parisi et al.[11], with permission.)

Vs medical therapy †P<0-01; ‡P<0-001.

CABG=coronary artery bypass grafting; PTCA=percutaneous transluminal coronary angioplasty.

identify a patient at risk; (2) left main stem disease benefits from CABG; (3) three-vessel disease, with or without good left ventricular function, benefits from CABG; (4) two-vessel disease, including proximal LAD disease, benefits from CABG; (5) minimal symptoms with a normal, or only slightly abnormal, ECG do not identify increased risk and medical therapy is the preferred initial option.

Medical therapy vs percutaneous transluminal coronary angioplasty

There is only one trial comparing PTCA with medical therapy[11] and it seems extraordinary that such an important interventional technique can be introduced without formal study. There is no doubt that PTCA relieves anginal symptoms but we lack detailed studies to allow us to position PTCA in a role other than that of pain relief.

Angioplasty Compared to Medicine (ACME) was a small randomized study of 212 patients with single-vessel disease, in which PTCA was compared with conventional medical therapy. PTCA patients were allowed to take drugs if necessary. The study included patients with recent MI and those not already on optimal medical therapy. While medical therapy was assumed to be optimized after the trial began, the trial did not address the question of what to do with patients who have symptoms in spite of optimal medical therapy, which is the most frequent problem seen in clinical practice.

At 6 months follow-up of the 105 patients allocated to PTCA and the 107 allocated to drugs, freedom from angina was present in 64% of the PTCA and 46% of the medical groups. There are, however, several caveats regarding the data. Firstly, the average single-vessel stenosis was 76% for PTCA and 77% for drugs with two-thirds of the lesions in the right or circumflex coronary artery. These are low-risk lesions, with no adverse effect on prognosis, so that symptoms would have to be severe in spite of optimal medical therapy in order to justify intervention. Furthermore, the use of anti-anginal drugs at 6 months was 95% in the medical group and 91% in the PTCA group. The use of oral nitrates was less after PTCA (50% vs 24%, P<0-01) and similar findings occurred with calcium antagonists (71% vs 35%, P<0-01) and β-blockers (50% vs 30%, P<0-01). However, it is important to realize that the benefit derived from PTCA was, for many, associated with additional use of anti-anginal drugs. Moreover, since only 50% of the medical group were receiving β-blockers, it is doubtful that medical therapy was indeed optimal. Table 1 identifies the significant differences in re-intervention between the groups, raising questions not only about the trauma to the patient, but also the cost of the procedure.

The ACME study really does not significantly progress the argument comparing PTCA with medical therapy. We can deduce that PTCA should be reserved for those who remain symptomatic in spite of optimal medical therapy, but there is no mandate for PTCA as an alternative to medical therapy for those with minimal symptoms and a modest single stenosis.

Percutaneous transluminal coronary angioplasty vs coronary artery bypass grafting

Data have now been published from several trials comparing PTCA and CABG[12-16] but only two included a predominant number of stable angina patients[12,13] (Table 2). In spite of the differences between the trials, a remarkably consistent message emerges: (1) PTCA and CABG are no different with regard to mortality at 2-3 years (Fig. 5); (2) CABG provides a consistently better complete revascularization rate; (3) CABG provides a consistently better outcome with regard to the need for re-intervention (Fig. 6); (4) PTCA patients need more anti-anginal drugs; (5) PTCA is a less traumatic procedure. Whilst there is no doubt that PTCA and CABG are effective treatments for angina, it does appear that CABG does not carry an increased risk and is more
Table 2: Summary of trials

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>134</td>
<td>1011</td>
<td>127</td>
<td>359</td>
<td>392</td>
</tr>
<tr>
<td>Male (%)</td>
<td>80%</td>
<td>81%</td>
<td>85%</td>
<td>80%</td>
<td>74%</td>
</tr>
<tr>
<td>Unstable angina (%)</td>
<td>10%</td>
<td>59%</td>
<td>83%</td>
<td>14%</td>
<td>60%</td>
</tr>
<tr>
<td>Extent CAD (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-vessel</td>
<td>100%</td>
<td>45%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>2-vessel</td>
<td>—</td>
<td>43%</td>
<td>55%</td>
<td>44%</td>
<td>60%</td>
</tr>
<tr>
<td>3-vessel</td>
<td>—</td>
<td>12%</td>
<td>45%</td>
<td>56%</td>
<td>40%</td>
</tr>
<tr>
<td>Years of follow-up</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Risk of death/MI/revascularization (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTCA</td>
<td>37%</td>
<td>38%</td>
<td>48%</td>
<td>56%</td>
<td>50%</td>
</tr>
<tr>
<td>CABG</td>
<td>8%</td>
<td>11%</td>
<td>17%</td>
<td>17%</td>
<td>15%</td>
</tr>
<tr>
<td>Anti-anginal drugs (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTCA</td>
<td>81%</td>
<td>61%</td>
<td>—</td>
<td>88%</td>
<td>66%</td>
</tr>
<tr>
<td>CABG</td>
<td>43%</td>
<td>34%</td>
<td>—</td>
<td>78%</td>
<td>51%</td>
</tr>
</tbody>
</table>

EAST = Emory Angioplasty Surgery Trial; ERACI = Estudio Randomizado Argentino de Angioplastia vs Cirugia; GABI = German Angioplasty Bypass Intervention; RITA = Randomised Intervention Treatment of Angina.

Figure 5: Survival of patients with multivessel coronary disease after treatment with CABG (---), or PTCA (----). The number of patients at risk and the estimated probability of survival are shown below the figure for each 6-month interval. (Reproduced from King et al.[14], with permission.)

Percutaneous transluminal coronary angioplasty vs stents

Given our limited knowledge of the role of PTCA for single-vessel disease in stable angina, it is a little surprising that a PTCA vs stent trial was performed (Benestent) without a drug comparator group[4]. The rationale of the study relates to the recognized problem of restenosis associated with PTCA and its prevention through the use of stents.

A total of 520 patients were randomized to stent (262) or PTCA (258). After exclusions, 52 patients (20%) in the stent group at 7 months follow-up and 76 patients (30%) in the PTCA group reached a primary endpoint (P=0.02). The primary endpoints were death, the occurrence of a cardiovascular accident, MI, the need for CABG or a second percutaneous intervention involving the previously treated lesion either at the time of the initial procedure or during the subsequent 7 months. The difference principally reflected a reduced restenosis rate from 32% to 22% (P=0.02) and a
Optimal medical therapy

Optimal medical therapy is difficult to define because of the individual nature of the anginal patient. Aspirin should be routinely prescribed unless specifically contraindicated. Aside from sublingual nitrates, the agents of primary interest are β-blockers, calcium antagonists, oral nitrates and the new potassium channel activator nicorandil. In clinical trials all these drugs are effective, because they are invariably used at an optimum dosage in carefully selected patients with careful subjective and objective monitoring. In clinical practice and in the comparative trials with CABG and PTCA there is consistent evidence of underdosing. Given that the primary indication for PTCA is pain relief not responsive to medical therapy, it follows that patients may be receiving intervention either inappropriately or too soon.

For example, while resting heart rate is a simple guide to the impact of β-blockade, it is the exercise heart rate that determines full effectiveness. A resting heart rate below 60 beats min⁻¹ is not an indication for dose reduction or stopping dose titration, unless there are symptoms such as excessive lethargy or fatigue. Although adverse effects may limit the use or dosage of any drug, the single greatest reason for failure of efficacy remains the failure to optimize dosage of single agents or agents in combination. Many patients take atenolol 50 mg day⁻¹, yet 100 mg is the optimal target if symptoms persist.

From all the research trials currently available we can establish the following dose guidelines. Atenolol 50–100 mg day⁻¹. Table 3 shows how individual optimal doses can be selected, e.g. propranolol 80 mg twice or three times daily. Propranolol is used as the reference drug with a potency of 1 and, therefore, dosage of other β-blockers is compared with 80 mg propranolol equivalent, not with total daily dose. Propranolol 80 mg can be given twice daily (half-life 11 h). It is equal to atenolol 100 mg (potency 1:1) and atenolol can be given once daily (pharmacodynamic half-life 24 h).

(2) Diltiazem 60–120 mg three times daily or Retard 90 mg or 120 mg twice daily.
(3) Verapamil 40–160 mg three times daily.
(4) Amlodipine 5–10 mg daily.
(5) Isosorbide mononitrate 20–40 mg twice daily or a once-daily preparation (40, 50 or 60 mg).
(6) Nicorandil 10–20 mg twice daily.

With regard to combination therapy, we know that β-blockers plus mononitrates, calcium antagonists plus nitrates and β-blockers plus calcium antagonists are all additive. It is not safe to prescribe verapamil with β-blockade and diltiazem should be used cautiously. Combination therapy can be used to circumvent adverse effects, for example, atenolol 100 mg day⁻¹ with side effects may have the same efficacy as atenolol 50 mg day⁻¹ plus amlodipine 5 mg day⁻¹ with a significantly reduced incidence of adverse effects.

Triple therapy remains a problem. The evidence that it improves symptoms is not at all clear and one study showed no benefit over double therapy. It is my view that if double therapy using optimal doses of each agent is ineffective in relieving symptoms, angiography should be the next option. While individuals may respond to triple therapy, at this level of medical therapy with persistent symptoms other options should be pursued. Fig. 7 contains an algorithm summarizing a proposed anti-anginal medical strategy.
**Table 3 β-blockers available**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Potency</th>
<th>Cardio-selective</th>
<th>Optimum dose pharmacodynamic half-life (h)</th>
<th>Blood brain barrier penetration</th>
<th>Dosage adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acebutolol</td>
<td>0-3*</td>
<td>+†</td>
<td>24</td>
<td>ns</td>
<td>Renal</td>
</tr>
<tr>
<td>Atenolol</td>
<td>1</td>
<td>+</td>
<td>24</td>
<td>ns</td>
<td>Renal</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>10</td>
<td>+</td>
<td>24</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>1</td>
<td>+</td>
<td>10-12</td>
<td>Yes</td>
<td>Liver</td>
</tr>
<tr>
<td>Nadolol</td>
<td>1-5</td>
<td>0</td>
<td>39</td>
<td>ns</td>
<td>Renal</td>
</tr>
<tr>
<td>Oxprenolol</td>
<td>0-5-1*</td>
<td>0</td>
<td>13</td>
<td>Yes</td>
<td>Liver</td>
</tr>
<tr>
<td>Pindolol</td>
<td>6*</td>
<td>0</td>
<td>8</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>Propranolol</td>
<td>1</td>
<td>0</td>
<td>11</td>
<td>Yes</td>
<td>Liver</td>
</tr>
<tr>
<td>Timolol</td>
<td>6</td>
<td>0</td>
<td>15</td>
<td>Yes</td>
<td>Liver</td>
</tr>
<tr>
<td>Slow-release oxprenolol</td>
<td>*</td>
<td></td>
<td>&lt;24</td>
<td></td>
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<tr>
<td>Metoprolol SA</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Propranolol LA</td>
<td></td>
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</table>

*Agents with partial agonist (intrinsic sympathomimetic activity).
†Cardio-selectivity of acebutolol is debated.
ns = not significant.

**Conclusion**

A review of the available data reveals significant gaps in our knowledge of the relative merits of medical and interventional therapy for stable angina. Some of these reflect medical progress, which has to some extent invalidated earlier trials, while others are created by trials directed at the wrong questions.

It is difficult to ignore the surgical benefit for left main stem disease or the data supporting CABG for the treatment of three-vessel disease, with or without impaired left ventricular function, and two-vessel disease involving the proximal LAD coronary artery. However, in those with disease other than left main stem, there is a need to revisit the role of medical therapy, particularly with the addition of aspirin and lipid-lowering strategies.

Optimal medical therapy needs better definition but, when medicine fails, both PTCA and surgery can offer significant further pain relief. When compared with surgery, PTCA does not reduce or increase mortality; revascularization is less complete, re-intervention more frequent and additional anti-anginal therapy more frequently necessary. PTCA is associated with lower morbidity than CABG and, up to 3 years post procedure, lower cost[119]. There is no demonstrable advantage for PTCA over medical therapy in patients with modest single-vessel, or perhaps even double-vessel, disease in the presence of no, or minimal, symptoms. Treatments are not mutually exclusive and medical therapy may frequently provide additional benefit when prescribed for patients after PTCA or coronary artery bypass grafting.

Statistics hide individuals who have their own personal needs, so medical advice needs to be carefully tailored. We must, however, only practice the medicine we believe is reasonable and logical if it is supported by the data and advocate treatment we ourselves would be personally willing to undertake.

**Addendum**

Since this paper was presented, the Bypass Angioplasty Revascularization Investigation (BARI) trial has reported[20]. Only 30% of 1829 patients studied had stable angina. The results were not unduly different from the...
previous CABG/PTCA trials with 8% of CABG and 43% of PTCA patients undergoing additional revascularization procedures at 5 years. There was no significant difference in 5-year survival rate (89-3% coronary artery bypass grafting and 86-3% PTCA). There is no mention of percent receiving anti-anginal drugs in addition to the revascularization procedure, though from the re-intervention rates one can deduce that the PTCA group would be likely to be receiving more additional drugs that the CABG group.

References