The progression of a coronary artery stenosis to total occlusion does not necessarily determine myocardial necrosis of the region nourished by the involved vessel, because sufficient degree of perfusion can be maintained by a developed collateral circulation. As a consequence, the area of necrosis can be prevented or limited in size.

Changes in Left Ventricular Function After Coronary Recanalization by Percutaneous Transluminal Coronary Angioplasty (PTCA)

Carlos Antonio M. Gottschall, MD, Ibsen Trindade, MD, Vasco Miler, MD

ABSTRACT: Forty–six patients (21 with stable angina and 25 with chronic myocardial infarction, 37 men) with a total chronic proximal coronary occlusion and collateral vessels to the distal part of the occluded artery (30 LAD, 10 RCA and 6 CX properly distributed in both groups) were studied angiographically before and 2 to 8 months (mean 6) after balloon angioplasty. The patients were divided in six subgroups: A) Angina pectoris no matter the result of recanalization (n = 21); B) Myocardial infarction no matter the result of recanalization (n = 25); C) Angina pectoris with successful recanalization and open coronary (O.C.) > 50% at follow–up (n = 13); D) Angina pectoris with unsuccessful recanalization and/or restenosis or closed coronary (C.C.) at follow–up (n = 8); E) Myocardial infarction with successful recanalization and O.C. > 50% at follow–up (n = 8); F) Myocardial infarction with unsuccessful recanalization and/or restenosis or C.C. at follow–up (n = 17). No subgroup showed statistical differences (p > 0.05) in LVEDP before (B) and at follow–up (FU). On the other hand, several measurements were statistically different in the subgroup A at B and at FU: Ejection fraction (EF) [57.3 ± 12.3 and 64.2 ± 19.4%; p = 0.02]; Regional wall motion (RWM) measured in the region of the affected coronary [18.7 ± 9.6 and 23.6 ± 11.8%; p = 0.05]; Minimal wall motion (MWM) measured in the site of lesser parietal movement [14.3 ± 13.1 and 25.8 ± 26.2%; p = 0.02]. In the subgroup C the following differences were observed: EF [58.4 ± 12.3 and 69.0 ± 12.4%; p = 0.003]; RWM [16.3 ± 8.4 and 25.4 ± 8.2%; p = 0.005]; MWM [14.7 ± 15.1 and 27.9 ± 18.0%; p = 0.001]. In the other considered subgroups we did not reach significant differences (p > 0.05) in these measurements. We conclude that recanalization of a chronic coronary occlusion improves left ventricular contractile function in the presence of viable myocardium and that MSF is the most sensitive among the studied variables to separate anginal patients from the patients without viable myocardium after successful recanalization.

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When an increased demand for myocardial supply of oxygen occurs the collateral blood flow may become insufficient, and symptoms and signs of myocardial ischemia without necrosis may arise. In 1982, Savage et al. asked; “Can PTCA be performed in patients with total occlusion?”, giving an affirmative answer. Soon after this we recanalized the first chronic totally occluded coronary in Brazil in a 35–year–old man. Although we know today that the primary success rate (± 60%) of PTCA for recanalization of occluded vessels is lower than the primary success rate (> 90%) of PTCA of nonocclusive stenosis, coronary desobstruction is also a method with a lower incidence of complications. Besides control of angina, longer survival and better ventricular function are also the desired goals of coronary recanalization. The vast majority of the studies consider short– and long–term results, difficulties and complications, but relatively few consider the effects of coronary recanalization on the left ventricular function.

The purpose of this study is to compare late left ventricular (LV) function modifications in homogenized subgroups of anginal and infarction patients with chronically occluded coronary artery after successful or unsuccessful recanalization by PTCA.

**METHODS**

**Patient Selection**

**Inclusion criteria.** Forty–six patients submitted to balloon PTCA for coronary recanalization of a total chronic proximal coronary occlusion with angiographic follow–up were studied. The total group comprises: A) 21 patients with stable angina pectoris without evidence of a previous infarction (17 men, 4 woman; age range 35 to 73 years; mean 53.2 ± 10.4) and B) 25 patients with a previously documented myocardial infarction in the region of the occluded vessel (20 men, 5 women; age range 30 to 75 years; mean 53.3 ± 12.1). Before angioplasty all patients had: 1) angina or post–infarction angina (6 class I, 34 class II, 6 class III of NYHA); 2) just one chronically occluded coronary and absence of stenosis > 50% in other coronary and 3) collateral circulation to the distal part of the epicardial segment of the occluded vessel. Twenty–two infarction patients had Q–wave infarct and 3 had non Q–wave infarct in the region of the occluded coronary. Patients with acute myocardial infarct were excluded. All clinical, electrocardiographic and angiographic data were collected during the patient’s admission without reference to the angiographic details. The drugs in use before PTCA varied from patient to patient and included beta–blocking agents, calcium antagonists and nitrates.

**Definitions.** Coronary occlusion was defined as absence of forward flow through the lesion as demonstrated by angiography. Duration of occlusion was estimated from time of infarction or sudden increase in angina and/or from the date of a previous coronary angiogram showing a patent artery. In anginal patients it was estimated from 7 to 96 days; mean 36.4 ± 26.0. In infarction patients from 9 to 142 days; mean 38.8 ± 39.3. Functional occlusions with anterograde flow beyond the lesions were excluded. The target coronary artery in anginal patients was the left anterior descending coronary artery (LAD) in 15, the right coronary artery (RCA) in 4 and the circumflex coronary artery (CX) in 2. In infarction patients the target coronary artery was the LAD in 15, the RCA in 6 and the CX in 4. We considered initial success when the residual stenosis remained equal or less than 50% of diameter reduction. Restenosis was defined as residual diameter stenosis > 50% at angiographic follow–up.

**Follow–up.** The routine medications used after PTCA were aspirin and calcium antagonists. Nitrates were used when necessary. Follow–up data were obtained two to eight months (mean 6) at the time of control angiography after successful or unsuccessful recanalization. In 13 patients angiography was performed because of recurrent or worsening angina and in 33 it was part of a routine post–PTCA control study.

**Baseline characteristics.** The best possible homogenization of the studied population was obtained by the comparable sex distribution, age, angiographic characteristics, duration of the occlusion and distribution of the target coronary artery in anginal and infarction patients (Table 1).

**Cardiac Catheterization**

**Catheterization technique.** Patients underwent catheterization in the post–absorptive state after sedation with 10 mg of diazepam administered orally about one hour before the procedure. Cineventriculograms were obtained in the RAOV 35° by injection of 40 ml of meglucamine diazotrate through a pigtail catheter for 4 seconds before
coronariography. Images were obtained at 30 frames per second. Post–ectopic beats were excluded from analysis. All procedures were done by the usual technique of PTCA using movable guide–wire dilation systems with 0.014 or 0.016 inch wires and low–profile dilatation catheters. The relationship balloon/artery used was 1:1.1. All patients received sublingual nitroglycerin, 10 mg of nifedipine and 10,000 U of heparin at the beginning of the procedure as well as other vasodilating drugs when needed during or after PTCA.

LV function analysis. The LV function analysis was done by the measurements of the left ventricular end–diastolic pressure (LVEDP–mmHg), by left ventricular ejection fraction (EF–%), by regional (apical and inferior) wall motion (RWM–%) and by minimal wall motion (MWM–%). The LVEDP (Pd2) was registered by an Elema–Schonader minigaph and was measured after the “a” wave, immediately before the beginning of the ventricular isometric contraction. It was represented as a percentual of the LV systolic pressure (Pd2/SP x 100) a relationship that we consider better LV functional index than the Pd2 alone.21

Outlines of the left ventricle silhouette were traced at end–diastole and end–systole from images obtained by a Philips Polydiagnostic C DCI. Apical and inferior RWM were defined respectively as the % of shortening (diastole minus systole) of the longitudinal and transversal LV internal diameter in RAOV 35°. The longitudinal axis extends from the inferior junction of the aortic valve with the body of LV to the apex of the cavity. The transversal axis was drawn in the middle and perpendicular to the longitudinal axis. For patients with target LAD we considered RWM the apical movement, and for patients with target RCA or CX we considered RWM the inferior movement. MWM was defined as the minimal shortening of a diastolic line traced from the point of intersection of the longitudinal and transversal axes to the site of least wall motion, showing the smallest or even negative movement at that point. At follow–up the respective line was traced from the axes intersection to a correspondent point in the perimeter of the LV cavity (Figure 1).

Patients Subgroups

We divided the 46 patients in six subgroups to compare the results before and at follow–up:

A) Angina pectoris no matter the result of recanalization (n=21);
B) Myocardial infarction no matter the result of recanalization (n=25);
C) Angina pectoris with successful recanalization and open coronary (O.C.) > 50% at follow–up (n=13);
D) Angina pectoris with unsuccessful recanalization and/or restenosis or closed coronary (C.C.) at follow–up (n=8);
E) Myocardial infarction with successful recanalization and O.C. at follow–up (n=8);
F) Myocardial infarction with unsuccessful recanalization and/or restenosis or C.C. at follow–up (n=17).

Statistical Analysis

Values are reported as mean values ± standard deviation. The changes in the paired data between entry and late follow–up were compared by the one–tailed paired Student t test for significance. Only the changes occurring in each group were compared. Probability (p) values less than 0.05 were considered significant.

RESULTS
Success rate and follow-up. The mean residual stenosis in successful PTCA was 14.8 ± 15.2% in the anginal patients and 20 ± 18.7% in the infarction patients. Initial success was reached in 17 out of 21 anginal patients (80.9%) and in 17 out of 25 infarction patients (68.0%). In patients submitted to the procedure up to 12 weeks of occlusion the initial success was obtained in 81.1% and in those treated after 12 weeks the initial success dropped to 18.9%. In these patients we had no major complications. Thirty of the 34 patients to whom the procedure was successful underwent stress testing within a week. Results were negative in 26 (86%) and positive in 4 (14%). At the follow-up we detected restenosis or reocclusion in 23.6% of the patients with angina and in 52.9% of the patients with infarction. The reocclusion incidence for all patients studied was 38.2%. Seven anginal and two infarction patients underwent a second PTCA during the study period.

LV function values. Neither the values of Pd₂ alone nor the values of the index Pd₂/SP x 100 were statistically different (p > 0.05) before PTCA and at follow-up in any of the subgroups. The relationship Pd₂/SP (%) was, respectively, 13.3 ± 4.4% and 12.3 ± 4.5% for anginal patients and 15.0 ± 6.7% and 13.6 ± 4.1% for infarction patients. On the other hand, the subgroups of all patients with angina (AP: n=21) and all patients with angina and open coronary (AOC: n=13) at follow-up showed significant improvement in the left ventricular function from before to follow-up (p < 0.05) detected by changes in EF (respectively, AP: 57.3 ± 12.3 and 64.2 ± 19.4%; AOC: 58.4 ± 12.3 and 69.0 ± 12.4%), in RWM (respectively, AP: 18.7 ± 9.6 and 23.6 ± 11.8%; AOC: 16.3 ± 8.4 and 25.4 ± 8.2%) and in MWM (respectively, AP: 14.3 ± 13.1 and 25.8 ± 26.2%; AOC: 14.7 ± 15.1 and 27.9 ± 18.0%). In the two considered subgroups the modifications in MWM attained the greatest significant difference (Table 2 and Figures 2-4).

DISCUSSION

The majority of published series presents the duration of occlusion as the main factor limiting the success of PTCA recanalization probably due to the progressive fibrosis that organizes the thrombus. 1, 2, 5, 6, 8, 13 Our low primary success rate (19%) in occlusions lasting longer than 12 weeks confirms this, while the initial success was high in those cases with less than 12 weeks of occlusion (81%).

The demonstration of marked histologic heterogeneity and residual metabolic activity in a high proportion of chronic electrocardiographic Q-wave infarction region implies the presence of viable tissue and allows probability of functional improvement after restoration of the flow following recanalization. 22 Indeed over time the ischemic myocardium that has been savaged by reperfusion ultimately improves diastolic and systolic functions and regains the contractile capacity. 23 The functional improvement can usually be explained by the presence of a pre-existent collateral circulation before balloon dilation in patients. 24, 25 Thus, areas of cardiac muscle may have remained viable in the region of the occluded artery, even in some patients with infarction at the time of occlusion. 26 As the ejection fraction of our anginal and infarction patients were comparable, the greatest improvement in anginal patients supports the assumption that the definite relief of angina was accompanied by recuperation of a greater mass of “stunned myocardium”. 23

Melchior et al. 19 and Finci et al. 20 believe that the better synchronism of relaxation may be
responsible for the improvement in LV function which they encountered after the recanalization of patients with chronic coronary occlusions. Several authors emphasize the importance of reperfusion as capable of saving myocardium and also of having influence in the development of lesser arrhythmogenicity and long-term mortality, in potential benefits on ventricular remodeling for stimulation of an efficient source of collateral vessels to other areas of the myocardium. Thus, the "open artery hypothesis" may provide an important role for PTCA in chronic phase of ischemic disease with or without infarction.27

The majority of ventricular segments of our anginal patients with proved abnormal wall motion prior to PTCA had a better or normal wall motion after the procedure whenever it was successful and the artery remain properly patent. Ventricular areas that did not significantly improve after PTCA were present in those patients who continued to show inadequate perfusion because the artery remained closed or because the correspondent myocardium was previously necrotic.

The PD values and the relationship PD/PS were not statistically different between the subgroups. Although this index reflects LV function better than PD alone it usually varies inversely with ejection fractions lower than the average of the cases of this study.21 However, the values for systolic shortening demonstrated statistically significant differences before recanalization and at the follow-up control of anginal patients who had a successful PTCA and whose coronaries remained patent. For anginal patients with unsuccessful PTCA or with restenosis or reocclusion there was no significant change in shortening values pre and post PTCA (Table 2 and Figures 2 and 3).

The EF is a widely used index but it may be normal despite the presence of segmental abnormalities of LV contraction and the RWM quantification becomes more appropriate to demonstrate ischemic localized abnormalities.28 As far as we know this is the first time that the measurement of the MWM is proposed. As it refers to one point, more exactly the site of lesser or even negative shortening, comparing the values for diastole and systole, it is probably the best way to measure the epicentric movement of the segmental involvement, and probably may become the best index to follow the localized changes in myocardial contractility in every patient. If we consider the group that had the best functional recovery after recanalization, that is, anginal patients with successful PTCA and open coronary at follow-up, the respective means of pre PTCA and control values for EF, RWM and MWM increased respectively 1.18, 1.55 and 1.89.

Although not statistically significant (p > 0.05) a tendency was noted for improvement of MWM at follow-up in the anginal patients even with unsuccessful recanalization. This tendency was

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**Table 2.** Left ventricular ejection fraction (LVEF), regional wall motion (RWM) and minimal wall motion (MWM) values (%) of patients (pts.) before PTCA for coronary recanalization and at follow-up.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>LVEF (%)</th>
<th>RWM (%)</th>
<th>MWM (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BEF</td>
<td>ATRU p</td>
<td>BEF</td>
<td>ATRU p</td>
</tr>
<tr>
<td>A-P</td>
<td>52.3 ± 12.9</td>
<td>54.3 ± 7.2</td>
<td>0.02</td>
</tr>
<tr>
<td>M.T. and O.C. (n=131)</td>
<td>58.4 ± 12.3</td>
<td>59.6 ± 12.6</td>
<td>0.003</td>
</tr>
<tr>
<td>M.T. and O.C. (n=82)</td>
<td>60.8 ± 10.1</td>
<td>63.4 ± 10.8</td>
<td>NS</td>
</tr>
<tr>
<td>M.T. and O.C. (n=13)</td>
<td>58.5 ± 14.1</td>
<td>59.1 ± 12.5</td>
<td>NS</td>
</tr>
</tbody>
</table>
Figure 2. Significant increases (p < 0.05) have occurred in LV ejection fraction (EF), in regional wall motion (RWM) and in minimal wall motion (MWM) when results were compared before (B) and at follow-up (FU) in the subgroup of anginal patients no matter the result of recanalization. Correspondent changes were not detected (p > 0.05) in the subgroup of infarction patients.

Figure 3. Significant increases (p < 0.05) have occurred in LV ejection fraction (EF), in regional wall motion (RWM) and in minimal wall motion (MWM) when results were compared before (B) and at follow-up (FU) in the subgroup of anginal patients (AP) with successful recanalization and open coronary (OC) at FU. Correspondent changes were not detected (p > 0.05) in AP with unsuccessful recanalization and/or closed coronary (CC) at FU.

Figure 4. There were not detected significant changes (p > 0.05) in ejection fraction (EF), in regional wall motion (RWM) and in minimal wall motion (MWM) when results before (B) and at follow-up (FU) were compared in the subgroups of infarction patients (IP) with successful recanalization and open coronary (OC) at FU and IP with unsuccessful recanalization and/or closed coronary (CC) at FU.
also noticed in the infarction subgroups when the recanalization was successful and the coronary remained patent in the control angiogram. Therefore, the improvement was totally absent only in the subgroup of infarction patients who had unsuccessful recanalization or who after initial success were found to be reoccluded at the control examination (Table 2 and Figures 2-4). One may speculate that these results suggest some degree of recovery of myocardium for all groups studied except the last one mentioned. If the myocardium was really necrotic its function could not be improved by any intervention.23

It is important to say that our study was not randomized. The procedure was indicated to correct ischemia rather than to improve ventricular contraction. A single plane right anterior oblique approach is certainly limited but the coronary lesions distribution was homogenized in anginal and infarction groups of patients. Whenever there is a good homogenization of studied cases the statistically strongest results tend to appear early even with small samples. The most noticeable result in the present study was the significant improvement of the systolic performance when both initial and late success were considered in the subgroups with viable muscle (mainly anginal patients). The data that were referred are interesting and give us a rational basis for selection of patients who might have a functional benefit through recanalization of a coronary artery.

CONCLUSIONS

This study shows improvement in LV function after successful recanalization by PTCA of chronic occluded coronary arteries in patients with stable angina. The improvement can be demonstrated by an increase in ejection fraction and most significantly by increase in regional wall motion, specially if the shortening of the point of lesser movement in the wall (minimal wall motion) is considered before and at follow-up.

Our results add to the growing body of evidence that asynergy at rest is not necessarily caused by permanent myocardial scarring and that there is reversible ischemia in the region of distribution of an occluded coronary artery in the presence of visible collateral vessels. This supports the concept that, in selected patients, restoration of anterograde flow to the ischemic region can permanently relieve reversible ventricular asynergy. Besides, our data support the concept that these regions are composed of viable myocardium that is functionally impaired by the effects of recent or silent ongoing ischemia. The results we got are consistent with the hypothesis that chronic ischemia or post–ischemic “stunning” of the myocardium may impair regional ventricular function in the resting state.

REFERENCES