Analysis of left ventricular function after emergency coronary artery bypass grafting for life-threatening ischaemia following primary revascularisation

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Abstract

Objective: Severe ischemic injury in the first few hours following primary revascularization necessitates acute reoperation. To study the effect of emergency coronary artery bypass grafting, we followed 18 patients for up to 8 years, relating their changes of global and regional myocardial function during the acute event and after secondary revascularization to final outcome.

Methods: A total of 16 patients with coronary artery bypass grafting (CABG) and 2 PTCA were treated for coronary heart disease between 1989 and 1993 and experienced life-threatening ischemic events (94% cardiogenic shock, 39% ventricular fibrillation, 67% ischemic electrocardiograph (ECG) changes) within 2.3 ± 1.6 h after primary revascularization. Reoperation was carried out 1.0 ± 1.3 h after the occurrence of acute ischemia. Serial echoes were obtained during the acute event and after reoperation as well as during the follow-up period. Results: Of the 18 patients, 8 are currently alive, 5 died within 30 days and 4 within the 1st year. There was one late death 5 years after surgery. Global and regional wall motion was evaluated using short axis views of transesophageal echoes taken during the acute event and after secondary revascularization, and compared with transthoracic echoes in long-term survivors up to 5 years after surgery. During the acute event left ventricular ejection fraction (LVEF) was reduced in 83% of the patients and improved significantly after reoperation ($\chi^2 = 11.74$, df = 2, $P < 0.01$). As to regional wall motion, 50% of the segments in non-revascularized areas remained abnormal. Regional wall motion after reoperation was significantly better in the surviving patients compared with patients dying in the post-operative course ($\chi^2 = 6.23$, df = 1, $P < 0.05$). The revascularization score (> 75%) of abnormal contracting segments during the acute ischemic event was a significant determinant for long-term survival. Conclusion: We conclude that patient outcome is determined by the severity of regional wall motion abnormality during the acute ischemic event, the aggressiveness of the attempt to revascularize these perfusion territories and their improvement after revision. Long-term survival reflects, therefore, the extent of emergency revascularization and therefore the ability to identify ischemic perfusion territories for surgical strategy planning. © 1998 Elsevier Science B.V.

Keywords: Emergency bypass; Peri-operative ischemia; Ventricular function; Regional wall motion; Reoperation; Hypoperfusion; Arterial grafts

1. Introduction

Peri-operative ischaemia is a life-threatening event, often necessitating cardiopulmonary resuscitation and instant decision-making for reoperation. Although almost every surgeon has experienced this catastrophic...
Table 1
Demographic patient data and peri-operative characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Survivors</th>
<th>Non-survivors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Medium survival time</td>
<td>Short survival time</td>
</tr>
<tr>
<td>Preoperative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (a)</td>
<td>68 ± 9</td>
<td>68 ± 4</td>
</tr>
<tr>
<td>Number of diseased vessels</td>
<td>2.7 ± 0.5</td>
<td>2.5 ± 0.9</td>
</tr>
<tr>
<td>Number of infarcts</td>
<td>0.7 ± 0.4</td>
<td>0.4 ± 0.5</td>
</tr>
<tr>
<td>Total number of stenoses</td>
<td>3.4 ± 2.1</td>
<td>3.1 ± 2.3</td>
</tr>
<tr>
<td>Number of significant stenoses</td>
<td>1.6 ± 1.1</td>
<td>1.8 ± 1.6</td>
</tr>
<tr>
<td>Primary revascularisation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic cross clamp time (ACC, min)</td>
<td>70 ± 22</td>
<td>69 ± 23</td>
</tr>
<tr>
<td>Cardio-pulmonary bypass time (ECC, min)</td>
<td>120 ± 32</td>
<td>124 ± 19</td>
</tr>
<tr>
<td>Number of distal anastomoses</td>
<td>3.00 ± 1.93</td>
<td>3.40 ± 1.14</td>
</tr>
<tr>
<td>Acute event and post-operative period</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours post ACC</td>
<td>2.5 ± 1.9</td>
<td>2.5 ± 1.9</td>
</tr>
<tr>
<td>Hours until second ECC</td>
<td>1.1 ± 1.3</td>
<td>0.4 ± 0.4</td>
</tr>
<tr>
<td>Days at intensive care unit</td>
<td>11 ± 9</td>
<td>23 ± 17</td>
</tr>
<tr>
<td>Days of hospitalisation</td>
<td>23 ± 14</td>
<td>35 ± 24</td>
</tr>
<tr>
<td>Creatine kinase (CK) peak value (U/l)</td>
<td>1082 ± 596</td>
<td>2403 ± 1078</td>
</tr>
<tr>
<td>CK-MB peak value (U/l)</td>
<td>80 ± 56</td>
<td>141 ± 92</td>
</tr>
<tr>
<td>Dopamine cumulative 24 h post-operative (mcg)</td>
<td>4849 ± 3140</td>
<td>3048 ± 839</td>
</tr>
<tr>
<td>Suprarenine cumulative 24 h post-operative (mcg)</td>
<td>192 ± 209</td>
<td>368 ± 474</td>
</tr>
</tbody>
</table>

Values are mean ± S.D.

event, little has been published on the final outcome and long-term survival of these patients. Reports concentrate on the mechanical support in post-cardiotomy shock, or coronary artery bypass grafting (CABG) after failed coronary angioplasty and the question of optimal myocardial protection for myocardial jeopardy, [1–4] omitting the questions of suitable diagnostic procedures and successful treatment strategies. Besides management to prevent further deterioration of global haemodynamics, steps have to be taken to relieve the underlying pathophysiologic and often iatrogenic cause of regional perfusion deficits.

A few reports on possible mechanisms of acute peri-operative ischaemia exist from the beginning of the widespread use of arterial grafts. In these reports the reason for catastrophic events were clearly related to the hypoperfusion of arterial grafts [5–9].

The present retrospective analysis was undertaken to identify factors occurring during acute peri-operative ischaemia which may influence patients’ survival. For this purpose we evaluated global and regional wall motion, compared primary revascularisation with the development of dysfuncioning areas and the strategies of reoperation with changes in wall motion and cardiac performance post-operatively.

2. Patients and methods

2.1. Patient population

From 1989 to 1993, 1894 patients were operated because of coronary heart disease (including combined procedures). The in-hospital mortality was 7%, 18 patients suffering from peri-operative or peri-interventional ischaemia and in whom transoesophageal echoes were taken during the acute event were analysed. Patient characteristics are summarised in Table 1. On the average, the acute event (e.g. haemodynamic deterioration and signs of myocardial ischaemia) occurred within 2.3 ± 1.6 h after primary revascularisation and in 8 patients during the operation but after reversal of heparinisation, in 2 patients after PTCA and in 8 patients after the transfer to the ICU. The time until secondary revascularisation was 1.0 ± 1.3 h. There were 9 patients who had external or internal cardiac massage until stabilisation of haemodynamics on extracorporeal circulation. There were 16 patients in cardiogenic shock, 11 patients had typical signs of ischaemia in the electrocardiograph (ECG), 7 showed ventricular fibrillation and 1 patient ventricular tachycardia. The patients were grouped according to their median survival time (short, medium or long-term; see below).
2.2. Evaluation of cardiac performance

Semi-quantitative evaluation of global and regional ventricular function was carried out retrospectively from video recordings taken during the event using transoesophageal echocardiography. Short axis views at the mid-papillary level were divided into eight segments and assigned to the perfusion territories, as follows: septal; anteroseptal; anterior to the LAD territory; anterolateral to the diagonal branch; lateral and posterolateral to the circumflex (Cx) region; and inferior and inferoposterior to the RCA.

Global function was determined as normal, or mildly, moderately or severely reduced. According to the guidelines of the International Society of Echocardiography regional wall motion was classified as normal, hypokinetic, akinetic or dyskinetic. Echoes were taken shortly after the onset of the acute event, after secondary revascularisation and in long-term survivors up to 8 years after operation.

2.3. Myocardial protection

During primary revascularisation as well as during reoperation, antegrade crystalloid cardioplegia was used (800 ml St. Thomas-solution cold induction and multidose intermittent administration after finishing each distal anastomosis). All patients were routinely cooled to 28°C at the beginning of surgery and rewarmed during the reperfusion period.

2.4. Statistical analysis

Differences between survivors and non-survivors were tested for statistical significance using $\chi^2$-tests in the case of qualitative and semiquantitative data (frequencies) and analysis of variance for quantitative data.

Survival probabilities were estimated from and plotted as survival distribution function, which is used to describe the lifetimes of the population of interest. The Kaplan-Meier method was used to calculate the survival distribution function estimates [10].

3. Results

3.1. The acute event

A total of 63% of all perfusion territories showed wall motion abnormalities during the acute event. Of these abnormally contracting areas 31% were observed in the Cx region, 29% in the RCA area, 22% in the LAD perfusion territory and 18% in the area of the diagonal branch. Fig. 1 shows the frequencies and quality of the primary revascularisation in relation to coronary perfusion territories. Mammary arteries were positioned primarily into the anterior and anterolateral wall, PTCA was carried out in two cases in the LAD and RCA region and veins were used as grafts mostly in the posterior and posterolateral regions. There were 12 patients who had received a Lima graft during primary revascularisation, 1 patient had a left and right Ima grafting, and 5 patients had no arterial revascularisation at all. The mean number of distal anastomoses carried out during primary revascularisation was $3.00 \pm 1.45$.

Haemodynamic instability was related to changes in global ventricular function. Left ventricular ejection fraction (LVEF) was normal only in 3 patients, mildly or moderately reduced in 6 patients, and severely depressed in 9. Right ventricular ejection fraction (RVEF) was less affected and showed normal motion in 9 patients and a severe dysfunction in 3 patients (Fig. 2).
The severity of regional wall motion abnormalities according to the perfusion territories are depicted in Fig. 3. Note that severity of dysfunction was more pronounced in lateral and posterior regions. Normal segments were found primarily in the LAD perfusion territory, whereas the contraction pattern showed more abnormally contracting segments in all other parts of the heart.

Of the primary revascularised segments 42% were normal, and 58% showed moderate or severe changes. A considerable number of contraction abnormalities occurred also in non-revascularised zones. Moderate or severe dysfunction was observed in 32% of all areas without primary revascularisation, whereas 68% of areas showed normal function. Note that there is a significantly different behaviour of regions related to the revascularisation during the first operation ($\chi^2 = 6.94$, df = 1, $P < 0.01$). In 60% of the 32% non-revascularised and dysfunctioning perfusion areas no signs of coronary stenosis had been present in pre-operative angiograms, 10% had shown an insignificant stenosed vessel, and 30% a significant stenosis. Divided into perfusion territories, abnormal contraction could be observed especially in areas perfused by a vein graft in the posterolateral aspect of the heart. Related to the total number of grafts, there was no difference in the behaviour of mammary artery implants or vein grafts. Non-revascularised segments with abnormal contraction mostly occurred in the perfusion zones of diagonal branches and lack of revascularisation in these areas was the leading cause of regional dysfunction and significantly more important than graft dysfunction ($\chi^2 = 13.76$, df = 3, $P < 0.01$) (Fig. 4).

3.2. Secondary revascularisation

During reoperation, revision of existing grafts was carried out as well as additional grafts being placed in all patients. As seen in Fig. 5 grafts were also placed into areas with normal contraction especially in zones perfused by the mammary artery. In spite of abnormal contraction in the anterolateral, lateral and posterior parts of the heart, some of these perfusion territories remained without further intervention.

Left ventricular (LV) function improved significantly ($\chi^2 = 11.74$, df = 2, $P < 0.01$) after revision, whereas changes in right ventricular function did not reach statistical significance in spite of considerable improve-
ment, since dysfunction during the acute event was not as severely depressed as on the left side (Fig. 2).

Revascularisation also improved regional wall motion significantly. A total of 32% of all segments remained unchanged and normal, 48% of segments with abnormal contraction improved after reoperation, (27% of the segments to only hypokinetic and 21% to normal contraction). 15% of dysfunctioning segments remained severely depressed and 4% of normal and 1% of dysfunctioning segments deteriorated ($\chi^2 = 17.28$, df = 2, $P < 0.001$).

3.3. Survival

There are currently 8 survivors (median 4.5 years post-operative), 2 patients were rehospitalised because of cardiac problems. No patient had to be reoperated. Although 5 patients developed mitral insufficiency of various degrees (severe, haemodynamic significant regurgitation in 3 patients) and 1 patient an aneurysm of the apex and distal anterior wall, 4 patients are classified in New York Heart Association (NYHA) grade 1, and 4 patients in NYHA grade 2.

There were 5 patients who died peri-operatively (median 10 days survival) on cardiac sequelae and sepsis and 4 patients who died within the first year (median 6 months survival). The cause of death was sepsis in 2 patients, 1 patient died after elective reoperation and 1 patient died of sudden death during outdoor activities. Only 1 patient died 5 years post-operatively from neurologic causes (overall mortality 55%).

3.4. Comparison between survivors and non-survivors

Whereas the changes in global wall motion showed no difference between survivors and non-survivors, the fate of regional wall motion abnormalities was different in patient groups affecting their survival. There were more abnormally contracting segments in non-surviving patients compared with surviving patients during the acute event as well as an enhanced improvement of these segments after the reoperation. As depicted in Fig. 6, there was a significant improvement in the contraction pattern of segments in survivors after reoperation, which could be maintained grosso modo in the long-term follow-up ($\chi^2 = 6.23$, df = 1, $P < 0.05$). The number of dysfunctioning reoperated segments and their ratio to unrevised segments was significantly different in survivors and non-survivors.

Peri-operative as well as medium range mortality was directly related to the extent of reoperation in affected perfusion zones; the more areas revised the better the final outcome. Patients dying peri-operatively and patients who died subsequently showed the same behaviour, although the total number of dysfunctioning segments was lower in the medium survival group. In long-term survivors, the number of dysfunctioning segments was similar to that in non-survivors, however the number of revised segments was significantly higher than non-revised zones ($\chi^2 = 11.52$, df = 2, $P < 0.01$), leading to the significant improvement of regional function in this patient group (Fig. 7).

As mentioned before, grafts and perfusion territories without functional disorders were also reoperated. In survivors 17 normal perfusion zones could be observed, six in medium survivors and two in short term survivors. In contrast to revascularisation into abnormally functioning areas, there was no difference in between groups in the relation of reoperation and revision into these normal zones. In survivors four of 17 zones were
reoperated and in non-survivors two of eight leading to the same proportions.

If we relate the revascularisation score of the vessels perfusing abnormally contracting segments during re-operation to patient outcome, we can observe a clear determinant for survival (Fig. 8). The higher the extent of revision of affected areas the better the patient survival.

Other patient demographics and parameters of the early post-reoperation period were not statistically different in the patient groups, although certain trends towards higher age, and more severe ischaemia with earlier onset after the first operation (as can be seen from enzyme release), could be observed in non-survivors.

Also the use of circulatory assistance to stabilise the patients haemodynamically showed differences. In 10 patients an intra-aortic balloon pump (IABP) was used; three in long-term survivors, in three of the peri-operative deaths and four in the medium survivors (see also Table 1).

4. Discussion

Although the incidence of perioperative myocardial infarction, graft dysfunction or haemodynamic deterioration requiring instant revision is low and is even further decreasing with improvements of perioperative myocardial protection, mortality in this patient group remains high. Several authors reported on the Lima-hypoperfusion syndrome myocardial protection and support systems in patients suffering from cardiogenic shock. Patients with failing angioplasty have been studied extensively, but little is known about the outcome of patients with hyperacute myocardial failure perioperatively. In contrast to interventional cardiological techniques, the cause of myocardial dysfunction cannot be verified with certainty by conventional methods. Whereas in patients with failing angioplasty little doubt exists about the vessel involved and the perfusion deficit leading to myocardial ischaemia, the situation in surgery is more complex.

Literature on the Lima-hypoperfusion syndrome clearly shows the danger of myocardial ischaemia as result of the imbalance between myocardial demand and support through arterial grafts. Revision and the placement of additional vein grafts showed significant improvements without potential hazard to the performance of the arterial graft [5–9]. In our analysis the evidence and correlation of malperfusion of arterial grafts seem to be overestimated. Surgical interventions in different regions of the heart, global ischaemia, reperfusion injury and sequelae of extracorporeal circulation as reversal of anticoagulation, add to the problem. We have reported earlier that signs of ischaemia in patients undergoing bypass surgery are more common than generally expected, however rarely lead to haemodynamic consequences [11].

In this present retrospective evaluation of patient outcome after peracute perioperative ischaemia, the following important findings could be made.

First, patient outcome in this difficult patient group is far better than expected. Morbidity in long-term survivors is within normal expectations, and in a majority of cases improvement in regional wall motion can be maintained in survivors also in previously damaged areas. The only sequelae of perioperative ischaemia in patients undergoing bypass surgery are more common than generally expected, however rarely lead to haemodynamic consequences [11].

Second, in contrast to patients with PTCA-failure, the knowledge on the origin and anatomical distribution of the perfusion deficit leading to haemodynamic sequelae is of enormous importance. The problem that normal contracting areas were revised and that dysfunctioning zones remained unrevised clearly shows the necessity to use modern intraoperative imaging techniques to identify regions with abnormal perfusion otherwise not accessible clinically. In contrast to the literature of hypoperfusion of arterial grafts, 60% of dysfunctioning zones were found in the lateral and posterior aspects of the heart and the majority of normal contracting zones, even during acute decline in
cardiac performance, were found in regions supplied with a Lima graft. A high number of unoperated segments in the regions of diagonal, marginal and posterior branches showed abnormal wall motion as a consequence of inadequate revascularisation. Interestingly several areas developing ischaemia and dysfunction were supplied by normal or insignificant stenosed arteries. Especially lateral and posterior parts of the heart were often the source of the decline in cardiac performance and often remained unrevised. The reason for this seeming discrepancy is the fact that the anterior aspect can be seen clearly by visual inspection and that the potential of the hypoperfusion of arterial grafts was overestimated. During the decision-making process for the acute reintervention all these important parameters are unknown and inaccessible with conventional monitoring unless wall motion can be determined. Even in the presence of previous myocardial infarction, extension of malperfusion may be present and therefore a region of interest for further attempts of revascularisation.

The ability to localise wall motion abnormalities by echo with the appropriate perfusion territory and the possibility to revise grafts and to additionally revascularise malfunctioning areas is directly related to patient outcome. Severity of coronary artery disease sometimes limits the chances to revascularise a malfunctioning area sufficiently, but it is also the aggressiveness of the reoperation and the knowledge of regional wall motion which determine patient outcome.

If already sequelae of ischaemia lead to a deterioration of the haemodynamic state of the patient, several steps have to be taken to minimise the patients risk. Although angiography or intraoperative thermography would be desirable, from the logistics of the surgical environment only transoesophageal echo can be applied instantly. The area of ischaemia and malperfusion can be diagnosed with high sensitivity and specificity and even during cardiac massage [12,13]. In some of these patients however, the value of echo imaging was not appreciated and grafts, especially arterial grafts (in whom direct inspection is less reliable) were revised without wall motion abnormality in these regions. In contrast underperfused, dysfunctioning areas remained ischaemic. Also in survivors lateral and posterior parts of the heart remained a problem and the development of severe mitral insufficiency can be explained by the resulting myocardial infarction in these regions.

In the reported patient group, imaging alone never led to the decision to revise and reoperate a patient but in some patients supported the decision-making process in presence of severe instability of the patient. This strategy has been altered recently towards a more liberal indication to reoperation.

When it was possible to stabilise the patient, the time until reoperation (which was approximately 1 h in our patients) was no discriminant between survivors and non-survivors. In addition, all other parameters including patient demographics, as well as surgical data from the primary revascularisation, data describing the acute event and the early post-operative course did not show any differences between groups. Only trends towards group differences could be observed in patient age, eflux of cardiac enzymes and the use of IABP, reflecting the severity and persistence of cardiac performance.

Third, although regional wall motion was less affected during the acute event in survivors, the discriminating factor, however is the ability of the surgeon to clearly identify problem zones and to completely revascularise dysfunctioning territories. As mentioned above this is not always possible because of the vessels involved.

As critique of the method we have to discuss two major problem areas. Sensitivity as well as specificity of transoesophageal echo to delineate new wall motion abnormalities have their limitations. Only with the help of a pre-operative echo can we identify new wall motion abnormalities during an acute event, which is always a pathological finding, however we cannot distinguish persisting changes from acute findings in patients without pre-operative functional studies. Discrimination between regional wall motion abnormalities developing peri-operatively and old infarcted areas is only possible when myocardial thickness differentiates ischaemia and infarction.

Another unsolved problem is the decreasing perfusion pressure during acute ischaemia. The development of regional ischaemia results in a snowball effect reducing cardiac performance and leading to a vicious cycle influencing myocardial perfusion in other territories. Additional catecholamine use may cause spasm in arterial grafts and cause further deterioration, including clotting in vein grafts. Clotting and bypass dysfunction is obvious in vein grafts, but wall motion abnormalities in areas perfused by a spastic arterial graft are difficult to tell and might be resolved without revising the vessel, simple by correcting perfusion pressure.

In this context the reoperation of normal contracting perfusion territories reported in this series is easier to understand. Reoperation in normal contracting zones were done, when the surgeon based his decision on his clinical judgement and not on the echo finding. It is also of importance to know that a perfusion deficit can be compensated by collateral flow and that unaffected areas have to hypercontract in order to maintain cardiac performance. Furthermore we should not underestimate clotting within the myocardium preferably in the venous vasculature as consequence of endothelial ischaemia and reperfusion [14].

In this context it is important to know that even with retrograde cardioplegia certain areas in the posterior and lateral aspect of the heart, which in our series are
susceptible to the development of ischaemia, remain poorly protected [15]. The consequence of a secondary anticoagulation, the increase of perfusion pressure during reoperation, adds to the improvement of the patient. As seen in our analysis, this improvement cannot be maintained into the post-operative period, unless the perfusion into deprived areas has been corrected and reestablished.

The complex pathophysiology and the multiple causes of microcirculatory changes due to cardioplegia and reperfusion play an important role in the development of peri-operative ischaemia. Also in our patients, quality of myocardial protection and subsequent changes of myocardial resistance add to the problem of deprivation from myocardial circulation. All these causes however can be reduced to one important parameter: severe loss of cardiac performance. For the surgical strategy planning it is important to restore global haemodynamics and coronary circulation. Patient management with anticoagulation, extracorporeal circulation and reversal of deprivation of regional flow again are polypragmatic therapies.

This retrospective analysis however allows the assumption that additional to the trivial therapies, restoring myocardial perfusion to dysfunctioning zones improves patient outcome. In spite of all these twilight zones, good clinical judgement together with monitoring and proper identification of problem areas will improve strategy planning and surgical options. In consequence of our data analysis of this period and based on our experimental and clinical research, we have altered our myocardial protection regimen. Since 1993 we routinely use ante- and retrograde blood cardioplegia. We furthermore use transoesophageal echo on most of our open heart cases. If signs of myocardial jeopardy develop we rely on our echo findings. In conclusion, patients requiring instant reoperation for peri-operative ischaemia should be screened for wall motion abnormalities. Whenever possible a comparison with earlier studies, including echoes taken during the first operation, will improve decision-making for the strategy of the reoperation. Revision should be scheduled instantly. Peri-operative imaging, surgical abilities and clinical judgement will improve the potential to revascularise as many of the affected areas as possible, thus improving morbidity and reducing mortality in this difficult patient group.

References


Appendix A. Conference discussion

Dr M. Murtra (Barcelona, Spain): Now that we can have intraoperative transesophageal echo studies, do you do routine echo studies? If you see some segments that are not contracting well, do you review the grafts, or even if you have some doubts, do you prefer to transfer the patient to the intensive care unit and see the evolution?

Dr W. Mohl: We studied myocardial function in the early periods of our transesophageal echo measurements, but now we are only reviewing data if there is any problem in the operating room. If there is a problem with CABG, then we put in the probe and look for the extent of abnormal contraction, and also in high risk groups with poor ventricular function, then we start off with a transesophageal probe in place.

Dr M. Murtra: There is a difference between patients that fail because of an incomplete revascularisation, the ones that present a sudden failure of a graft that was previously working well, or as
Professor Turina has stressed in the past, the case of a left anterior descending artery revascularised with a mammary artery, that later in the post-operative either presents a spasm or dysfunction, or does not have sufficient flow to cope with the demands of the myocardium. In these patients that you reoperated, have you always found a good correlation between the segment that is not contracting and an obstruction of the graft, or if it was a mammary artery an insufficient perfusion.

Dr W. Mohl: Well, I think I have shown that most of the areas were not perfused by the mammary artery. I think the misinterpretation was that the surgeon thought that it is always the LAD that is not functioning, the LAD territory, and by retrospective studies like this I think it’s clear that it is not the case. Sometimes a mammary artery has no problem but other grafts have the problem and therefore we have to revise these areas. On the other hand the drop in perfusion pressure during the acute event may impair flow in the mammary artery.

Dr G. Pettersson (Copenhagen, Denmark): As you know, we have been very interested in this aggressive approach for a long period of time, and, in addition to your patients, we are referring a number of patients to acute reangiography. We believe that most of the stable patients tolerate it well and we have made many findings that are not possible to make on the operating table. How do you look upon the place for acute reangiography? Have you done reangiography after any of your reinterventions to see if you have achieved complete revascularisation? I would guess that the possibility for you to achieve complete revascularisation would be less.

Dr W. Mohl: Thank you for the comment. We didn’t have the chance to look for angiographies in this patient group. Perhaps we can do it in the long-term survivors.