Global and regional left ventricular function and
tomographic radionuclide perfusion: The Western
Washington Intracoronary Streptokinase In
Myocardial Infarction Trial

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ABSTRACT The Western Washington Intracoronary Streptokinase In Myocardial Infarction Trial
enrolled 250 patients with acute myocardial infarction. After the coronary angiographic diagnosis of
thrombosis, patients were randomly assigned to receive either conventional therapy with heparin or
intracoronary streptokinase followed by heparin. Of the 232 patients who survived at least 60 days, 207
(89%) underwent radionuclide ventriculographic determination of global and regional ejection fraction
at a single institution at 62 ± 35 days after infarction. In the first 100 patients, infarct size was also
determined by quantitative single-photon emission tomographic imaging with thallium-201 (201T1) and
expressed as a percentage of the left ventricle with a perfusion defect. Overall, global ejection fraction
did not differ between patients treated with streptokinase (45.9 ± 13.9%; n = 115) and control patients
(46.1 ± 14.4%; n = 92, p = NS). Similarly, the regional posterolateral, inferior, and anteroseptal
ejection fraction did not differ between the two groups. Infarct size as measured by 201T1 tomography
was 19.4 ± 12.8% (n = 52) of the left ventricle for the streptokinase group and 19.6 ± 11.8% (n =
48; p = NS) for the control group. When patients were compared within groups by electrocardiograph-
ic location of infarction, time to treatment, or the presence or absence of vessel opening, there were no
significant differences between streptokinase and control patients. Statistical inclusion of the 18
patients who died early and were unavailable for study also failed to modify the results, except for a
possible reduction in inferior infarct size as measured by 201T1 tomography. We conclude that global
and regional left ventricular pump function and infarct size determined by 201T1 tomography did not
differ substantially between patients receiving streptokinase and those receiving conventional treatment
when measured 8 weeks after myocardial infarction.

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WITH THE demonstration of coronary arterial throm-
bois as a common denominator in patients with acute
transmural myocardial infarction, therapeutic throm-
bolysis in this setting has become widespread. How-
ever, there are few controlled data comparing this form
of therapy with conventional treatment. We deter-
ned the global and regional ejection fraction by ra-
dionuclide ventriculography as well as quantitative
tomographic thallium-201 (201T1) perfusion of sur-
vivors of acute myocardial infarction in a randomized,
multicenter trial comparing the use of intracoronary
streptokinase with standard therapy. In this multi-
center trial, the radionuclide studies were performed at
a central laboratory 8 weeks after acute myocardial
infarction.

Methods

Patient selection. Patients were treated in 13 hospitals in
western Washington and one in Vancouver, B.C. Details
of patient selection, baseline clinical characteristics, therapy, and
30 day mortality have been presented elsewhere.1, 2 In brief,
patients 75 years old or younger with typical clinical symptoms
of acute myocardial infarction of 12 hr duration or less were
eligible. Electrocardiographic entry criteria included ST seg-
ment elevation and/or a marked increase in T wave amplitude
with or without new Q waves. Patients with other life-threaten-
ing illness and/or a contraindication to anticoagulation were
excluded. Eligible patients, after giving informed consent, were taken to the cardiac catheterization laboratory and, after identification of thrombotic occlusion (total or subtotal) of a coronary artery not responsive to intracoronary nitroglycerin, were randomly assigned to receive either conventional therapy, including full-dose heparin, or intracoronary streptokinase. The randomization was stratified by duration of symptoms of less than or greater than 3 hr and by electrocardiographic location of infarction as anterior or inferior. Lateral infarction was grouped with anterior, and posterior infarction with inferior. Immediately after randomization, those patients assigned to receive intracoronary streptokinase were given the drug at a rate of 4000 U/min as a continuous ostial or subselective catheter infusion or as bolus injections of 1000 U/15 sec in the coronary ostium. The infusion was maintained until reperfusion occurred and for 30 min thereafter, or until a total of 250,000 to 300,000 U had been given. All patients in both groups received full-dose heparin until oral anticoagulation with coumadin could be instituted. Other medications and therapy were prescribed at the discretion of personal physicians and included coronary bypass surgery or angioplasty in the early postinfarction period in some patients (see below).

A total of 250 patients underwent randomization, 134 to the streptokinase group and 116 to the control group. Anterior infarction was present in 116 (46%) and inferior infarction in 134 (54%). Fifty-five patients (22%) had symptoms of 0 to 3 hr duration and 195 (78%) had symptoms of 3 to 12 hr duration. The mean time from the onset of symptoms to randomization (and the concomitant administration of intracoronary streptokinase) was 276 min. There were no statistically significant differences between the control and treatment groups for the following baseline characteristics: age, sex, history of prior myocardial infarction, history of congestive heart failure, time to hospitalization, time to randomization, hypotension (systolic blood pressure <90 mm Hg), or ejection fraction determined by contrast angiography.

Reperfusion or vessel opening was defined as any reduction in the percent stenosis or occlusion from the time of randomization to the termination of the angiogram. Reperfusion occurred in 69% of the 132 patients in the streptokinase group (data were unavailable in two patients). In the 116 control patients, reperfusion occurred in 12% during the angiographic procedure either spontaneously or in response to the radiographic contrast material, intracoronary nitroglycerin, or mechanical forces related to contrast injections.

Centralized radionuclide studies. After discharge from the hospital, each patient was asked to come to a central laboratory in Seattle for study of ventricular function and 201Tl tomography. These studies were performed and the results were analyzed by investigators who were blinded to the treatment assignment.

A total of 207 patients underwent late radionuclide ventriculographic studies at 62 ± 15 (1 SD) days after myocardial infarction. Twenty-eight of these patients had undergone coronary bypass surgery or percutaneous transluminal coronary angioplasty (17 in the streptokinase group, 11 in the control group) before undergoing radionuclide studies. This sample of patients represents 89% of survivors available for study at 8 weeks. The first 100 consecutive patients undergoing radionuclide studies also underwent rotational tomography for quantitation of the size of the myocardial perfusion defect.

Ventricular function. Radionuclide ventriculography was performed with patients at rest with electrocardiographically synchronized blood pool imaging. Red blood cells were labeled with 20 to 25 mCi 99mTc and images were collected in the anterior and 45 degree left anterior oblique views (or a modification of the 45 degree view to best separate the right and left ventricles). Fourteen consecutive 40 msec images were collected, beginning with the electrocardiographic R wave for a total of 300,000 counts/frame. A standard gamma camera and dedicated nuclear medicine computer were used with a low-energy, parallel-hole, high-sensitivity collimator. Ejection fraction was calculated from the left anterior oblique image by subtracting end-systolic from end-diastolic counts (after background correction) and dividing this difference by the end-diastolic counts. For each frame of the cardiac cycle, a left ventricular region of interest was defined by a computer algorithm that initially used a thresholding and subsequently a two-dimensional first-derivative approach; the operator could override these automatically defined edges if they failed to correspond to the visually apparent cardiac borders. The accuracy and precision of these methods have been described elsewhere. Next, by means of the left anterior oblique images as previously defined, the geometric center of activity was determined at end-diastole and anteroseptal, inferior, and posterolateral regional ejection fractions were calculated as illustrated in figure 1.

Infarct size determined by 201Tl tomography. Two millicuries of 201Tl was injected with patients at rest after an overnight fast. Fifteen minutes later image collection was begun with a wide-field-of-view gamma camera (GE 400T, Milwaukee) that could rotate circumferentially about the patient's thorax. The camera was positioned with a fixed 22 cm radius of rotation to most closely approximate the chest wall at the cardiac apex. According to the observation of Larsson and as described in our labo-
Maximal count activity for each short-axis slice within this volume of myocardium was measured in each of 32 equally spaced radians projected from the center of the slice. Each radian subtended 11.35 degrees of annular rotation. The number of counts and the maximal counts per pixel for each such sector were defined. Count values for each slice were normalized to the maximal counts per pixel per sector for the entire heart. For each sector in each slice, all pixels identified with maximal counts more than 3 SDs below the mean from a series of healthy young normal control subjects were defined as perfusion defects. When summed, the volume of the defect was expressed as a percentage of the left ventricle and subsequently called infarct size. This approach to measuring infarct size has recently been validated in an experimental animal preparation.9,10 in which perfusion defect size was similar when abnormal segments were defined as those exceeding 2 or 3 SDs from the normal series. In patients, quality was less uniform and defect size, defined as exceeding 3 SDs of the normal range, was empirically chosen to most closely approximate defect size estimated by visual analysis of the images. Reproducibility was assessed by reanalysis of the original data from 10 randomly selected studies 1 month apart, yielding an r value of .99.

For purposes of display, all quantitative three-dimensional image data were reduced to a single two-dimensional image. Counts were scaled so that counts falling within the normal range were white and all areas of count reductions (>3 SDs from the normal range) were gray to black, depending on the magnitude of count reduction, where black equals zero counts. Count data for each slice were arranged as a series of concentric rings forming a “bull’s-eye” in which the basalmost slice was the outermost ring and the apex was the central ring. An entirely normal study would thus have an entirely white quantitative display. The rings were oriented in the familiar left anterior oblique or short-axis perspective, which preserves the coronary artery distribution. Thus perfusion defects of the left anterior descending coronary artery were seen to the left of the image, those of the posterior descending coronary artery inferiorly, and those of the circumflex to the right. Figure 3 shows a schematic of the display and figure 4 illustrates a study of a patient with an inferior infarction, showing both the raw images as well as the quantitative display. Infarct size within specific anatomic zones was also assessed; the apical 30% of myocardium constituted the apex; the remaining four regions were equally divided from apex to base into inferior, septal, anterior, and lateral zones. Only the first 100 consecutive patients were studied by 201Tl tomography, since this was the original projected size of the patient sample; resources for further tomographic studies were not available.

**Statistical methods.** Measures of tomographic infarct size and ejection fraction were compared in the total streptokinase and control groups and in subgroups by the t test. The 201Tl tomographic data were not available for three streptokinase-treated and nine control patients who died before their scheduled tomographic studies, and for five streptokinase-treated and 13 control patients who died before their scheduled radionuclide ventriculographic studies. These patients were included in a second analysis that used the Wilcoxon rank-sum test. In the rank analyses of ejection fraction, the deaths were given the lowest rank (corresponding to the lowest ejection fraction). In the rank analyses of infarct size, the deaths were given the highest rank (corresponding to a large infarct size). An analysis of covariance was used to assess the effect of treatment group and time to initiation of treatment on ejection fraction and tomographically determined infarct size. Stepwise multiple regression was used to determine the multivariate effect of baseline variables and to assess the effect of streptokinase therapy after adjustment for the variables selected as most important. The
Variables considered in the regression analysis were treatment assignment, time from onset of myocardial infarction to hospital admission, time from onset of myocardial infarction to treatment, number of vessels diseased, baseline stenosis in the occluded vessel, stenosis in the occluded vessel after treatment, hypotension, age, location of infarction, cardiogenic shock, ejection fraction, previous myocardial infarction, ST segment elevation, ST segment depression, presence of electrocardiographic Q waves, collateral vessels, treatment more than 3 hr after onset of myocardial infarction, total time in catheterization laboratory, early reocclusion of the vessel, time to opening of the vessel, total infusion time, and total streptokinase dose. All data are presented as the mean ± SD.

Results

Ventricular function. Overall, the global ejection fraction was not different between the two groups. The mean ejection fraction was 45.9 ± 13.9% (n = 115) in the streptokinase group and 46.1 ± 14.4% (n = 92) in the control group (p = NS) (figure 5). When ana-
lyzed for anterior electrocardiographic location, ejection fraction was 37.4 ± 14.3% (n = 51) in the streptokinase group and 41.4 ± 14.8% (n = 38) in the control group (p = NS). For inferior infarction, ejection fraction was 52.6 ± 10.4% (n = 64) in the streptokinase group and 49.4 ± 12.8% (n = 54) in the control group (this was a slight trend favoring the streptokinase group; p = .13). There were no differences in ejection fraction between patients with symptoms of less than 3 hr duration between streptokinase-treated (46.0 ± 14.5%; n = 25) and control patients (49.5 ± 15.2%; n = 20, p = NS). Similarly, for patients with symptoms of greater than 3 hr duration there were no differences in ejection fraction between the groups (45.8 ± 14.2% [n = 90] in the streptokinase group; 45.1 ± 13.6% [n = 72] in the control group; p = NS). There was no demonstrable effect of vessel opening at the time of coronary angiography; that is, ejection fraction was the same for patients with opened vessels at 46.7 ± 14.0% (n = 87) as for those with closed vessels at 44.5 ± 14.1% (n = 101, p = NS). This remained the case when analyzed further for streptokinase or standard therapy.

Regional ejection fraction did not differ overall between the streptokinase-treated and control patients for any of the three regions. The posterolateral ejection fraction was 48.3 ± 15.0% in the streptokinase group and 48.8 ± 14.4% in the control group (p = NS); the inferior ejection fraction was 57.1 ± 13.9% for the streptokinase group and 57.2 ± 13.4% for controls (p = NS); the anteroseptal ejection fraction was 48.9 ± 12.9% for the streptokinase group and 48.1 ± 12.5% for controls (p = NS). When the electrocardiographic location of infarction was considered, there were still no significant differences between streptokinase-treated and control patients for any of the three regional ejection fractions. When duration of symptoms of greater or less than 3 hr was considered, there were again no differences in any regional ejection fraction between streptokinase-treated and control patients. When either global or regional ejection fraction was analyzed by the nonparametric approach, incorporating patients who had died as the lowest rank, the conclusions above were unchanged. Similarly, both global and regional ejection fractions were unchanged whether or not patients with intervening coronary bypass surgery or angioplasty were included.

**Infarct size determined by 201Tl tomography.** Overall, infarct size as a percentage of the left ventricle did not differ significantly between the two groups (figure 6). Streptokinase-treated patients had a mean infarct size of 19.4 ± 12.8% (n = 52) vs 19.6 ± 11.8% for control patients (n = 48) (p = NS). Regional analysis of the infarct sizes showed no differences overall between the streptokinase and control groups for each of the five tomographic regions.

When analyzed by electrocardiographic location of infarction, anterior infarct size did not differ between streptokinase-treated (27.8 ± 12.2%; n = 24) and control patients (23.1 ± 12.8%; n = 16, p = NS). Among patients with electrocardiographically inferior infarction, however, streptokinase-treated patients had significantly smaller infarct sizes: 12.2 ± 13.5% (n = 28) in streptokinase-treated vs 17.8 ± 11.3% (n = 32) in control patients (p = .04). In this subgroup, most of the observed differences occurred because slightly more control than treated patients with inferior infarction had had prior myocardial infarctions. The observed difference in infarct size for inferior infarctions was not significantly different (p > .05) after adjustment for history of infarction.

There were no differences in tomographic infarct size between control and streptokinase-treated patients in the subsets with symptoms of less than or greater than 3 hr duration. Infarct size in patients with symptoms of less than 3 hr duration was 19.0 ± 11.4% (n = 10) in streptokinase-treated vs 18.5 ± 10.2% (n = 9) in control patients (p = NS). For those with symptoms of more than 3 hr duration, infarct size was 19.5 ± 13.6% in streptokinase-treated (n = 42) and 19.0 ± 12.5% in control patients (n = 39, p = NS). Similarly, the size of the tomographic perfusion defect was not related to opening of the occluded vessel. Infarct size was 22.2 ± 12.7% (n = 50) in those with
closed vessels vs 18.0 ± 12.5% (n = 43, p = NS) in those with opened vessels. However, a minority of patients were treated within 3 hr and only two patients were treated in under 2 hr.

In patients who died early, the follow-up infarct size could not be known. Since there were more deaths in the control group, the data were reanalyzed by the relative rank of the infarct size for each patient. Infarct sizes for patients who died were included as the highest rank, corresponding to large infarcts. In these analyses, the overall infarct size still did not differ between the streptokinase and control groups. When analyzed by anatomic region on the tomograms, there was a significant reduction in infarct size within the inferior zone only (p = .04). Infarct size in the remaining four regions did not differ between the streptokinase and control groups. When analyzed by the electrocardiographic location of infarction, there were no differences for global or regional infarct size for anterior myocardial infarction. However, for inferior infarctions the global or overall infarct size was significantly smaller for the streptokinase group (p = .03), i.e., this result was identical to that of the analysis excluding the deaths. Infarct size and ejection fraction were closely related; the correlation coefficient between the two was −.74.

**Stepwise multiple regression.** Stepwise multiple regression analysis was used to protect against confounding of the treatment effect with baseline patient characteristics. The analyses were used to determine the patient characteristics most closely related to the measures of myocardial damage studied and to analyze the difference in outcome for treated and untreated patients after adjustment for these characteristics. The analysis of infarct size selected anterior infarct (p < .0001) and previous myocardial infarction (p = .003) as most predictive of tomographic infarct size. Adjustment for these variables did not result in a difference in infarct size for streptokinase-treated patients compared with untreated patients. The analysis of ejection fraction selected anterior infarct (p = .01) and baseline contrast angiographic ejection fraction (p < .0001) as most closely related to follow-up ejection fraction. There was no difference in follow-up ejection fraction for treated and untreated patients after adjustment of the comparison for these variables.

**Summary.** There was no overall reduction in infarct size between streptokinase-treated and control patients whether or not deaths were considered. When deaths were included there was a possible reduction in infarct size favoring the streptokinase group only in the inferior region. Whether or not this has biologic significance is not clear, given the large number of statistical tests used. These results were the same whether or not the subset of patients undergoing intervening coronary bypass surgery or angioplasty were included.

**Discussion**

In this study, patients from several institutions came to a central laboratory for standardized evaluation of both global and regional left ventricular function by radionuclide ventriculography. One hundred of these patients also underwent a three-dimensional measure of infarct size. Overall, there were no differences between the streptokinase-treated and control patients in either infarct size or ventricular function, suggesting no substantial benefit or harm from the experimental therapy.

The comparability of these results for global function and infarct size showing no demonstrable benefit to survivors supports the biologic certainty of this conclusion. That is, these two tests are relatively independent, assessing myocardial damage from two differing perspectives, one measuring ventricular pump function and the other myocardial perfusion. Technically, the two studies also differed. The Tl201 tomogram, for example, was reconstructed three-dimensionally by computer techniques, whereas the radionuclide ventriculogram was performed with the standard planar approach. Thus neither infarct size, a direct measure of myocardial damage, nor residual pump function on either a global or regional basis were different overall between streptokinase-treated and control patients as evaluated 8 weeks after myocardial infarction.

An unexpected feature of the data is the lack of any relationship between the time to treatment and the infarct size or ventricular function. All experimental animal data and much of the available clinical literature show that the degree of myocardial salvage relates directly to the elapsed time to thrombolysis.11-13 A possible explanation for the results of this study is that the times to treatment were simply too long. The mean time to vessel opening was about 5 hr; only 22% of patients had symptoms of less than 3 hr duration. The earliest treated were two patients who had had symptoms for 2 hr. A similar finding was the lack of any relationship between the radionuclide measures and the presence or absence of vessel opening. Since vessel opening is the presumed sine qua non for possible myocardial salvage, the lack of any relationship might again support the hypothesis that as applied in this study, therapy was simply too late and even if vessel opening occurred it was generally too late to be of
benefit in surviving patients. Since no patients were treated within 1 hr of the onset of symptoms and only two within 2 hr, our data cannot be extrapolated to suggest that very early treatment would not be more beneficial.

The radionuclide studies used in this study were performed relatively late, an average of 8 weeks after infarction. This was necessitated primarily by the study design, in which hospitals from throughout the western half of the state of Washington participated. However, late studies probably do reflect net myocardial damage after infarction. Peri-infarctional ischemic changes should have largely resolved by this time. Similarly, the increase in sympathetic activity associated with myocardial infarction would have normalized. Analysis of contrast left ventriculograms at the time of intracoronary administration of streptokinase has commonly shown hyperkinesis in noninvolved areas, so that the global ejection function was relatively unaffected. When studied remote from infarction, hyperkinesis is much less commonly observed. Thus, in our late studies, global and regional ejection fraction should be sensitive indicators of the extent of residual myocardial damage. The validity of late studies presupposes that intervening acute infarction has not occurred; this was the case in our sample, with no patient having an interim rehospitalization for acute infarction. A small proportion of patients (12%) did, however, undergo coronary bypass surgery or angioplasty—resting ventricular function and perfusion could have been altered by this therapy. The overall results, however, were the same whether or not these patients were included.

Quantitative infarct imaging with single photon emission computed tomography (SPECT) is a relatively new technique. Others using this approach have obtained validation in experimental animals of infarct size by means of technetium pyrophosphate or labeled fatty acids. In patients, SPECT imaging with $^{201}$TI has been related to infarct size as measured by creatine kinase activity and patient prognosis, and in our laboratory this method has been shown to be a more sensitive detector of prior infarction than traditional planar imaging. Furthermore, we initially developed the techniques used in this study in an experimental animal preparation and found a high correlation between $^{201}$TI SPECT infarct size and the size of the perfusion defect measured by well counting in vitro. The initial myocardial distribution of $^{201}$TI parallels blood flow and, to the extent that the distribution can be externally quantitated, should accurately reflect infarct size. In some patients with unstable chest pain syndromes, initial $^{201}$TI distribution may reflect both infarction and resting hyperperfusion unassociated with infarction and demonstrable by late changes of 'redistribution.' At the time of our study, patients were stable and perfusion defects were presumably largely or exclusively those of infarction. We have previously shown that in such patients, defects generally correspond well with other evidence of infarction, and in a similar series of stable patients with remote infarction we found no redistribution in repeat images. We performed both $^{201}$TI tomography and radionuclide ventriculography to ascertain whether one or the other technique was more sensitive in detecting treatment changes. We anticipated that small infarctions might have relatively little detectable effect on ejection fraction, in which case the presence of compensatory hyperkinesis might mask changes in ejection fraction. However, no differences were detectable in this study.

Limitations of this study include the fact that we were able to do 8 week studies on 89% of surviving patients and some patients underwent coronary bypass surgery or angioplasty before study. Additionally, although radionuclide techniques measure ejection fraction accurately, image resolution is limited and subtle abnormalities might not be detected. For infarct sizing by $^{201}$TI tomography, our approach is 'all or none' for the definition of a defect; substantial subendocardial defects may not be separable from transmural defects. Nevertheless, our study was a randomized, controlled trial and enrolled a large number of patients. Results of two small randomized trials have recently been reported with conflicting results on short-term improvement of left ventricular function. No other studies have included randomized controls, and the generally favorable results reported may reflect bias in patient selection. Although the Western Washington trial has shown a substantial reduction in 30 day mortality for patients receiving streptokinase therapy, the present results suggest that survivors as a group do not benefit by reduced infarct size of improved left ventricular function. One interpretation of these results is that the time required to bring a patient to the cardiac catheterization laboratory is prohibitively long for most patients.

The results of these studies do not provide an explanation for the reduction in mortality for streptokinase-treated patients. The mechanism of reduced early mortality in treated patients is unclear and will require further investigation. Further controlled trials achieving earlier reperfusion are needed to determine the feasibility of reducing infarct size and preserving left ventricular pump function.
Appendix

The principal investigators and their associates of the Western Washington Intracoronary Streptokinase In Myocardial Infarction Trial: J. Ward Kennedy, M.D., Director, James L. Ritchie, M.D., Co-Director, Kathryn B. Davis, Ph.D., Investigator, James K. Fritz, M.D., Investigator; Data Coordinating Center Kathryn B. Davis, Ph.D., Charles Maynard, M.A., David Fray, B.A.; Policy Board: Simeon Rubenstein, M.D., Chairman, Paul B. Beeson, M.D., Thomas S. Inui, M.D., Lloyd D. Fisher, Ph.D., consultants; K. Lance Gould, M.D., Houston, William Ganz, M.D., Los Angeles, Harold T. Dodge, M.D., Seattle, Robert A. Bruce, M.D., Seattle, Marie Cowan, R.N., Ph.D., Seattle; Technicians: Carol Alcock, Ann Coleman, Kay Gaines; Nurses. Dee Erickson, R.N., Kathleen McFadden, R.N.; Clinical Associates: Overlake Hospital, Bellevue, WA, C. E. Hansing, K. M. Hynes, R. E. Haynes, D. B. Ferrin, J. S. Schneider, J. T. Holder, (Ken Hyles, Randy Swartz); St. Joseph Hospital, Bellingham, WA, R. S. Trenouth, D. C. Brown, D. D. McAfee (Susan Harris); Everett General Hospital, Everett, WA, J. P. Nolan, D. J. Stewart, W. J. MacDonald, K. H. Prindle, N. D. Smith, J. Schmitt (Linda Woucha, Harlan Jones); Providence Hospital, Seattle, G. A. Logan, F. M. Tobis, T. A. Block, J. G. Doces, M. T. English, P. C. Albro, A. L. Sytman, R. A. Crane, C. G. Hale, B. Green (Pat Jordan); Swedish Hospital, Seattle, F. A. Short, W. E. Samson, R. J. Westcott, J. L. Peterson (Linda Gaborino); University Hospital, Seattle, D. K. Stewart, A. J. Murray, K. F. Hossack, J. A. Werner, G. Frank, G. B. Brown, D. W. Weaver, G. B. Trobaugh (Royce Snyder); Veterans Administration Medical Center, Seattle, J. W. Kennedy, J. L. Ritchie, J. H. Caldwell, J. Stratton, (Carol Alcock); Virginia Mason Hospital, Seattle, R. R. Johnston, (Mary A. Madsen); Madigan Army Hospital, Tacoma, J. L. Hill, T. Steudel, R. Chamusco (Don Felton, Pearl C. Jakeman); Tacoma General Hospital, Tacoma, E. Lapin, T. Reagan (John Viles); St. Joseph’s Hospital, Tacoma, J. R. McDonough, M. Henry, (Tom Samon); Vancouver General Hospital, Vancouver, B.C., R. R. Ricci; Wenatchee Valley Hospital, Wenatchee, WA, D. Larson, J. Gorham, (Larry Keyser); St. Elizabeth Hospital, Yakima, WA, D. A. Monick, R. D. Twiss, A. B. Preacher, R. K. Spiegel (Frances Braungel, Mark Rupert).

Italics denote physicians who were administratively responsible for the trial at their institutions. In parentheses are listed the catheterization laboratory technicians responsible for the study.

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