
ELIPAD RECHAVIA, MD,* LUIS I. ARAUJO, MD, RANIL DE SILVA, BSc.
SUDHIR S. KUSHWAHA, MRCP, ADRIAAN A. LAMMERTSMA, PhD. TERRY JONES, DSc.
ANDREW MITCHELL, MRCP. ATILIO MASERI, MD, FACC.
MAGDI H. YACOUB, FRCS, FACC.
London and Harefield, Middlesex, England

To assess coronary vasodilator reserve after orthotopic heart transplantation, regional myocardial perfusion was measured with oxygen-15-labeled water and dynamic positron emission tomography in 14 cardiac allograft recipients who were not experiencing rejection and who had no angiographic evidence of epicardial coronary sclerosis 15 to 73 months (mean 43 ± 19) after transplantation (group I). Twelve normal men with an average age of 31 years (group II) served as a control group. Regional perfusion was measured at rest and after the intravenous administration of 0.6 mg/kg body weight of dipyridamole.

Rest regional myocardial blood flow was homogeneously distributed throughout the left ventricle and was significantly higher in transplant recipients (mean 1.16 ± 0.26 mlg per min [range 0.3 to 1.73] than in normal subjects: mean 0.85 ± 0.13 mlg per min [range 0.57 to 0.99]; p = 0.001) as was rest heart rate-systolic blood pressure product (rate-pressure product 11,255 ± 2,540 vs. 7,073 ± 1,106; p < 0.001). After dipyridamole, perfusion in the transplant recipients was homogeneous and slightly lower (2.13 ± 1.05 vs. 2.46 ± 1.21 mlg per min; p = NS) whereas rate-pressure product was slightly higher (12,179 ± 2,266 vs. 10,885 ± 1,895; p = NS) than the value in normal subjects.

Dipyridamole vasodilator response (dipyridamole/rest myocardial blood flow) ranged from 1.23 to 4.92 (mean 2.58 ± 1.19) in group I and from 2.65 to 5.45 (3.87 ± 0.89) in group II (p = 0.001). Rate-pressure product change (dipyridamole/rest rate-pressure product) was markedly attenuated in transplant recipients compared with the value in normal subjects (1.09 ± 0.09 vs. 1.56 ± 0.27 mlg per min; p < 0.001).

Normalization for the difference in rest rate-pressure product for each individual subject showed that the adjusted rest flow values (group I 1.19 ± 0.27, group II 1.39 ± 0.31 mlg per min; p = NS) and the dipyridamole-vasodilator response data (group I 2.45 ± 0.64, group II 2.47 ± 1.25; p = NS) in the two groups were not significantly different. Thus, in transplant recipients who are not experiencing rejection, the response of the coronary vasculature to dipyridamole-medihated vasodilation is uniformly preserved among all regions of the left ventricular myocardium.

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血，在慢性心力衰竭中，使用氧-15标记的水和动态正电子发射断层成像在一组心脏移植受者中进行，这些受者没有经历过严重的排斥反应。

**Method**

**Study patients.** Fourteen male non diabetic heart transplant recipients (group I) ranging in age from 25 to 62 years (mean 50 ± 10) were selected for this study. Before transplantation, all patients had end-stage heart failure due to ischemic heart disease (nine patients) or idiopathic dilated cardiomyopathy (five patients). Flow studies were carried out 13 to 24 months (mean 43 ± 11) after transplantation. All patients had normal cardiac function (mean left ventricular ejection fraction 69 ± 7% [range 57 to 76]) with no evidence of cardiac hypertrophy as assessed by left ventriculography and two-dimensional echocardiography, a normal coronary angiogram and no histologic (endomycocardial biopsy) or clinical findings of cardiac rejection. The mean duration of ischemia at operation was 158 ± 53 min (range 75 to 225). The mean interval between the most recent annual cardiac catheterization and the perfusion study was 7 ± 5 weeks. All patients were maintained on a regular immunosuppressive regimen of cyclosporine, whose dose was adjusted to maintain a blood level of 150 to 250 ng/ml and azathioprine (2 mg/kg body weight). No patient was taking corticosteroids. No patient had clinical or electrocardiographic (ECG) findings compatible with post-transplantation myocardial infarction. All medication (aspirin, dipyridamole, furosemide and nifedipine) other than the immunosuppressive agents was withheld 48 h before the perfusion study in all patients.

Twelve men (group II) with an average age of 31 ± 5 years were recruited to provide a normal myocardial blood flow data base. None had risk factors for coronary artery disease, previous symptoms, clinical signs or ECG evidence of heart disease but, because cardiac catheterization was not clinically indicated, none had undergone this study.

**Informed written consent was obtained from each patient. The study protocol was approved by the Hammersmith and Harrow Hospital Research Ethics Committees and the Administration of Radioactive Substances Advisory Committee.**

**Scanning procedures and tracers.** The technique for measuring regional myocardial perfusion employed in this study was previously validated in our laboratory (11). Briefly, this method involves the continuous inhalation of 15O-labeled carbon dioxide (C15O2), which is transformed into 15O-labeled water by the lung carbonic anhydrase, and rapid dynamic positron emission tomographic scanning. In validation studies, simultaneous measurements of regional myocardial blood flow with 15O-water and gamma labeled microspheres in nine closed chest dogs, over a flow range of 0.5 to 6.1 ml/g per min yielded a good correlation between the two methods even at high flows (r = 0.36 ± 1.0x; n = 0.91) (11).Tomographic imaging was performed with a multibiomechanism scanner (CTI). Fifteen-plane image acquisition in a 10.5-cm field of view enables simultaneous data collection from the whole heart (12). Each subject was positioned within the scanner for a 5-min rectilinear transmission scan to allow optimal positioning of the heart in the center of the field of view of the camera. The maintenance of this position was checked by aligning washable ink marks made on the patient's chest with a reference laser beam from the tomograph. A 20-min transmission scan was then obtained by exposure of an external germanium-68 ring source. This was used to correct the subsequent emission scans for tissue attenuation of the 511-keV γ-photons. This scan was followed by an oxygen-15-labeled carbon monoxide (C15O) blood volume scan. Each subject inhaled C15O at a radiotactive concentration of 3 MBq/ml for 4 min (total activity 3,000 MBq). A 6-min data acquisition commenced 1 min after the end of C15O delivery when the C15O had equilibrated in the blood pool (13). The emission blood pool scan is reliant upon in vivo binding and labeling of red blood cells by the formation of 15O-carboxyhemoglobin. In each subject, venous blood samples were withdrawn every 2 min during the acquisition period and counted in a NaI well counter cross calibrated with the scanner.

When activity had decayed to background levels, patients inhaled C15O2 for 3.5 min at a radioactive concentration of 6 MBq/ml (total activity 5,250 MBq). In the lungs, C15O2 is rapidly transformed to 15O-labeled water (H15O) (14), which is a freely diffusible tracer (15) with a short half-life of 123 s. It has a complete first pass extraction by the myocardium (16) that is unaffected by the tissue metabolic state over a wide range of flow rates (17). A dynamic scanning protocol was employed whereby ungated acquisition was initiated 30 s after the end of C15O2 inhalation, enabling the measurement of background activity (19). The subsequent 6.5-min acquisition period, which encompassed the gas inhalation period and the 3-min interval after cessation of inhalation, was divided into 24 time frames of varying length (range 5 to 30 s).

**Dipyridamole infusion.** When activity from C15O2 had decayed to background levels, dipyridamole (0.6 mg/kg) was infused at a constant rate over 4 min into an antecubital vein. Data acquisition and C15O2 inhalation were initiated after completion of dipyridamole infusion. An identical scanning protocol to that described earlier was again employed. Heart rate and blood pressure were monitored throughout the infusion and imaging periods. A three-lead ECG was recorded throughout the acquisition period. Sys- tolic blood pressure was measured by a standard manual sphygmomanometer. The rate-pressure product was obtained in the baseline state and after dipyridamole infusion by multiplying peak systolic blood pressure by peak heart rate. For each subject, the rate-pressure product ratio
The resultant blood volume images were need to trace
sis {Fro-Matlab mathematical software package). The
Foundation "Analyze" software package and kinetic analy-
created images were subsequently transferred to Sun 3/60
were reconstructed arid corrected for tissue attenuation. The
ptessure product.
was determined as the ratio of posbdipyridamolzhest tale-
102
was projected on the dynamic 14,110 frames, eercraling average
water "washout" images (Fig. I). These regions were then
identified on each slice of the resultant160.
and septum) identified on each slice of the resultant images from the frames obtained after cessation of the
C15O, inhalation (frames 22 to 25) were summed and filtered
by taking the average of the left atria time-activity curves. Forthe subsequent
modeling procedures. arterial input function was produced
by using a low pass smoothing filler. A blood volume
subtraction was performed by using the 0150 images after
they were filtered and scaled (17). Twenty-five to 30 circular
regions of interest of 5-pixel radius (equivalent to 10-mm
radius) were positioned in the four different anatomic seg-
ments of the myocardium (anterior, lateral, inferoposterior
and septum) identified on each slice of the resultant 15O-water "washout" images (Fig. I). These regions were then
projected on the dynamic H215O frames, generating average
tissue time-activity curves for each myocardial segment in
the same way as their arterial counterparts. These kinetic
data were fitted to a single tissue compartment tracer kinetic
model with a correction for the underestimation of tissue
activity due to the partial volume effect and the fractional
spillover of activity from the left ventricle into the tissue
regions of interest. This gives rise to the analytical equation
1 listed in the Appendix.

Solutions to equation 1 were obtained by nonlinear least
squares regression analysis of the observed kinetic data for
three variables, regional myocardial blood flow, fraction of
exchangeable tissue (e), and spillover blood volume fraction
(Vs) for each myocardial segment. These variables were
calculated from a mean of at least five consecutive tissue
regions of interest. In each individual subject, a mean value
of myocardial blood flow was obtained from the flow values
measured in all four myocardial segments. This value was
divided by a factor of 1.04 (density of myocardium) to give
flow values with units of mlg per min.

Analysis of dipyridamole vasodilator response data. Dipyr-
idamole vasodilator response was defined as the ratio of flow
after the vasodilator stimulus to baseline flow. However, any
difference in flow response observed between the two groups
may be attributed to the disparity in rest myocardial blood
flow. Experimental evidence (20) suggests that up to a flow
rate of 2 mlg per min, oxygen consumption and myocardial
flow are linearly related. Any discrepancy in rest flow data
between the two groups may therefore be coupled to a
difference in oxygen consumption. Consequently, a fairer
comparison may be made by normalizing the rest myocardial
blood flow data in both groups to the average oxygen
demand (reflected by rate-pressure product) of the transplant
group. This was performed with use of equation 2 (Appen-
dix).

Regional myocardial blood flow after dipyridamole was not
normalized to the change in rate-pressure product as the
relative contributions of oxygen consumption and the vasod-
ilator effect of dipyridamole to the increase in flow are as
yet uncertain.

Statistical methods. Mean ± SD (or ± SE for flow mea-
urements) values were obtained for each variable. Data
analysis was performed with use of unpaired or paired t
tests, as appropriate. Correlation of coefficients were calcu-
lated with the least-squares linear regression analysis. Prob-
ability (p) values < 0.05 were considered statistically signif-

Results

Clinical and hemodynamic findings (Table 1). All 14 transplant recipients and 12 normal subjects tolerated the procedure without serious adverse effect. Heart rate, peak systolic arterial pressure, rate-pressure product and myocardial blood flow at baseline and after dipyridamole infusion were compared on an individual basis and between the two groups. Within each group there was a significant increase in heart rate after dipyridamole infusion (group I: 79 ± 11 vs. 87 ± 9 [p < 0.001]; group II: 63 ± 10 vs. 95 ± 14 [p < 0.001]), but no significant change in systolic blood pressure (p > 0.05 for both groups). Comparison of the two groups showed that rest heart rate and blood pressure in the transplant recipients were 25% and 28%, respectively, higher relative to values in the normal subjects (p < 0.001 for both variables). This difference explains the increase (59%) rate-pressure product at rest in group I relative to that in group II (p < 0.001). After administration of dipyridamole, the increment in rate-pressure product was higher for the normal subjects than for the transplant recipients (54% vs. 8%; p < 0.01).

Myocardial blood flow measurements. Rest regional myocardial blood flow was significantly higher (36%) in group I than in group II (1.16 ± 0.26 vs. 0.85 ± 0.13 ml/g per min; p < 0.001). However, adjustment of baseline flow values for rate-pressure product and recalculation of myocardial blood flow showed no statistical difference between the two groups.

Table 1. Hemodynamic and Blood Flow Results in 26 Men

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>HR (bpm)</th>
<th>Peak BP</th>
<th>BPVR</th>
<th>rMBF</th>
<th>R-</th>
<th>DVR</th>
<th>DVR'</th>
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<tbody>
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<td>Group I</td>
<td>Transplant group</td>
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<td>18</td>
<td>17</td>
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p value < 0.001 0.08 0.06 0.001 R vs. D | vs. group II < 0.001 0.001 0.001 < 0.001 0.001 0.001 < 0.001 0.001 0.001 0.001 0.001 0.001 0.001 R vs. D

BF = Blood pressure; D = dipyridamole; DVR = dipyridamole vasodilator response; DVR' = heart rate-pressure product-adjusted dipyridamole vasodilator response; R = rest; rMBF = regional myocardial blood flow; R- = heart rate-pressure product-adjusted myocardial blood flow; BPVR = heart rate-pressure product.
cient of variation of the dipyridamole vasodilator response in the transplant recipients (group I) was 13% and in the normal group (1.39 ± 0.3); p = 0.09). A wide range of flow values in response to dipyridamole was observed in both groups (group I, 1.33 to 5.02 ml/kg per min [mean 2.73 ± 1.03]; group II, 1.52 to 5.27 ml/kg per min [mean 3.40 ± 1.09]; p = NS).

Dipyridamole vasodilator response. The dipyridamole vasodilator response value was significantly lower in the transplant recipients (group I) than in group II (2.5 ± 1.13 vs. 3.97 ± 0.39, p = 0.031). However, analysis of the vasodilator response data taking into account the difference in rest rate-pressure product (DVR* in Table 1) showed no significant difference (group I, 2.47 ± 1.25; group II, 2.45 ± 0.64; p = 0.93) between the two groups.

Regional variability of myocardial blood flow. In both groups myocardial blood flow was homogeneously distributed throughout the entire left ventricle. In the transplant recipients, the coefficient of variation of flow values measured in the four myocardial segments (mean intrasubject regional variability) was 17% both before and after dipyridamole infusion. The corresponding values in the normal group were similar: 13% and 15%, respectively. The coefficient of variation of the dipyridamole vasodilator response value derived from the same segments was 18% for the transplant recipients and 15% for the normal group.

Correlation between dipyridamole vasodilator response and clinical variables. For the entire group of transplant recipients, there was no significant correlation between age, time elapsed from transplantation, left ventricular ejection fraction or any of the other clinical variables studied and the adjusted or nonadjusted dipyridamole vasodilator response data. Depressed dipyridamole vasodilator response (adjusted and nonadjusted values) could not be related to baseline cardiac disease, prevalence rate of rejection episodes (range 1 to 4 episodes, mean 2.4 ± 0.9) in the 1st 2 months after transplantation or to operative ischemic time.

Discussion

The major observations of the present study are 1) rest myocardial blood flow in the transplanted hearts was homogeneously distributed and higher than that in normal subjects; 2) the increased rest perfusion in the transplant recipients was associated with a higher rate-pressure product; 3) after dipyridamole infusion, myocardial blood flow was homogeneously distributed and not significantly different from that in the normal group; and 4) the higher rest rate-pressure product and consequently higher rest flow values account for the lower dipyridamole vasodilator response in the transplant recipients than in the normal subjects. These results indicate that myocardial blood flow after human orthotopic heart transplantation is homogeneously distributed, both before and after pharmacologic stimulation, a finding consistent with recently published data measuring myocardial blood flow at rest and after exercise.

Regional myocardial blood flow. Myocardial perfusion was evenly distributed throughout all regions of the left ventricular wall as evidenced by the similarity of regional variability of myocardial blood flow in the transplant recipients to that in the normal subjects. Rest flow values in our normal subjects were comparable with those obtained by two other groups (21,22) using 13O-water for quantitative perfusion imaging. Regional myocardial blood flow was significantly higher in the transplanted hearts than in the native hearts. This finding is consistent with earlier reported results obtained with the thermodynamic method (7) and 13N-water and positron emission tomography (23). It was associated with an increased rest rate-pressure product, which has also been previously documented (5,7).

In this study we used 13O-water, which is a freely diffusible tracer and measures flow linearly even at high flow rates; it is not metabolically active and its short half-life enables repeat studies to be performed within a short period of time. Studies have previously shown that the rate-pressure product and oxygen consumption are well correlated (24). Because myocardial oxygen consumption and flow are linearly related up to perfusion values of 2 ml/kg per min (26), we adjusted the measured rest myocardial blood flow data on an individual basis in both groups to the mean of the rate-pressure product data acquired in the transplant group. This adjustment was made to normalize the differences in baseline cardiac work between the two groups. After the normalization was performed, the estimated rest myocardial blood flow in normal subjects was not significantly different from that measured in the transplant group. Thus, the difference in rest flow values between the two groups is probably due to the difference in baseline metabolic demand.

Coronary flow reserve in normal subjects. In normal subjects, the increment in myocardial perfusion following dipyridamole infusion is in agreement with coronary flow velocity data obtained with the Doppler catheter method (25), showing a peak to rest coronary flow velocity ratio ranging from 1.9 to 5.4 compared with our ratio of post-dipyridamole to rest flow of 2.65 to 5.45. This change in myocardial perfusion or blood flow velocity from baseline to that elicited by pharmacologic vasodilation is the standard definition used for coronary flow reserve. This assumes that the coronary resistive vessels are maximally dilated and the rest tissue perfusion is homogeneous in the studied.

Coronary flow reserve in the transplanted heart. Perfusion autoregulation depends on several mechanisms, principally neural control, metabolic demand and endothelial function (26). After heart transplantation all of these mechanisms may be affected. In our study, the mean hyperemic to control myocardial blood flow ratio in the transplant recipients was 2.5 ± 1.13 compared with 5 ± 0.3 (5), 4 ± 1.1 (8) and 5 ± 0.8 (9) peak to rest coronary flow velocity ratios measured by Doppler catheter technique and induction of hyperemia by intracoronary administration of a maximally vasodilating dose of papaverine. There may be several explanations for this difference. 1) There is a large individual variation in coronary flow reserve after the administration of...
the standard vasodilating dose of dipyridamole compared with that observed after intracoronary papaverine (25); 2) the possibility that the coronary vasculature in some of our patients was not maximally vasodilated is a potentially confounding factor adding to data scatter; and 3) the Doppler method allows the measurement of flow velocity in real time and thus allows the identification of the peak hyperemic response, whereas our results reflect the average tissue perfusion during the whole acquisition period (6 min). This might lead to an underestimation of peak hyperemic flow response to dipyridamole because of the low time resolution of our method and consequently to an underestimation of coronary flow reserve.

The limited dipyridamole vasodilator response in the transplanted heart may be related to 1) a decreased absolute myocardial blood flow after dipyridamole-induced vasodilation, and 2) to an increased rest myocardial blood flow (27). Because post-dipyridamole tissue perfusion in the two groups was not significantly different, the latter explanation seems more plausible. The higher rest tissue perfusion in the transplanted as compared with the native heart stems largely from the higher cardiac work load of the transplant recipients, which enforces a higher rest flow. If this difference in rate-pressure product is corrected for, the dipyridamole vasodilator response is not significantly different from that in normal subjects. Hence, coronary flow reserve data in transplant recipients should be interpreted in light of the cardiac work load at which measurements of myocardial blood flow were obtained.

For the entire group of transplant recipients, no correlation was found between the adjusted or nonadjusted dipyridamole vasodilator response and any of the clinical variables, including time elapsed from transplantation. This finding emphasizes that the microvascular atherosclerosis that gradually develops after transplantation is not essential to the development of reduced coronary flow reserve. Additionally, depressed dipyridamole vasodilator response could not be related in this study to the rate of rejection episodes that occurred in the 1st 2 months after transplantation. A transient reduction of coronary vasculature capacity has been observed in patients undergoing acute rejection (9); however, functional recovery occurred after rejection therapy. Whether transplant recipients with reduced coronary flow reserve are more prone to subsequent rejection episodes remains to be determined.

Methodologic considerations. The Doppler catheter technique is currently the most widely used technique for measuring coronary flow reserve. However, it has two main limitations: it is an invasive technique and it measures flow velocity in the epicardial coronary arteries and therefore gives no direct information on regional myocardial tissue perfusion. $^{15}$O-water and dynamic positron emission tomography provide a noninvasive method for accurately measuring regional myocardial tissue perfusion. Because the temporal resolution of this technique is lower than that of the Doppler technique, perfusion is expressed as an average of myocardial blood flow measured over scanning time. Further studies assessing microvascular and metabolic function in the entire left ventricle would be particularly useful in cardiac allograft recipients and patients with small vessel atherosclerotic heart disease.

Conclusions. Our study indicates that in the transplanted heart regional perfusion is homogeneously increased to match its higher metabolic needs at rest. The disparity in rest perfusion between transplant recipients and normal subjects before pharmacologic vasodilation may explain the reduced dipyrdiamole vasodilator response that we observed in the transplant recipients. Normalization of baseline flow for differences in cardiac work load between the two groups showed that the dipyridamole vasodilator response was of a similar magnitude. Thus despite chronic denervation, the responsiveness of the coronary vasculature in the nonrejecting transplanted heart to dipyridamole-mediated vasodilation is preserved.

Appendix

Equation 1

$$C_{oii} = \alpha \cdot RMBF \cdot C_a \cdot \exp\left(-\frac{\text{RMBF}}{p}\right) + \frac{\text{V}_a}{\text{V}_v} \cdot C_v$$

where $C_{oii}$ = oxygen-15 labeled water ($H_2^{15}O$) concentration seen in the tissue region of interest by the scanner (counts per pixel); $\alpha$ = fraction of exchangeable tissue (ml of tissue capable of exchanging water/ml of tissue capable of exchanging water); $C_a$ = left atrial concentration of tracer (counts per pixel) seen in the vascular region of interest by the scanner; $p$ = operation of convolution; $p$ = tissue/blood partition coefficient of oxygen-15 ($0.96 \text{ ml of blood/ml of tissue}$); $\alpha$ = physical decay constant ($0.138 \text{ min}^{-1}$ of oxygen-15); $t$ = time (min); $V_a$ = spillover blood volume fraction from the left ventricle into the tissue region of interest and myocardial arterial blood volume.

Equation 2

$$R-MBF = \frac{R^\text{MBF}}{R^\text{RPP}} \cdot \frac{R^\text{RPP}}{R^\text{MBF}}$$

$R^\text{MBF}$ = rest regional myocardial blood flow for each individual subject normalized to average rate-pressure product in group I; $R^\text{RPP}$ = mean of the rest rate-pressure product data for group I; $R^\text{RPP}$ = rest rate-pressure product for each individual subject.

References


