Correlation of Coronary Arteriograms and Left Ventriculograms with Postmortem Studies

Grover M. Hutchins, M.D., Bernadine H. Bulkley, M.D., Ren L. Ridolfi, M.D., Lawrence S. C. Griffith, M.D., Frederick T. Lohr, and Mark A. Piasio

SUMMARY To assess the accuracy of angiographic determinations of disease of coronary arteries and left ventricular myocardium we compared clinical with postmortem coronary arteriograms and left ventriculograms with myocardial pathology in 28 patients, all of whom died postoperatively and within three months of angiography; 19 had ischemic heart disease, four valvular heart disease, and five both. Comparison of pre and postmortem lumenal occlusion in 315 epicardial coronary segments, excluding those operated upon, showed >50% narrowing discrepancies in 21 (7%). Significant coronary artery lesions were overestimated in six and underestimated in 15. Of the six overestimations, three appeared to be due to coronary spasm; of the 15 underestimations, 12 were due to overlapping images; six discrepancies were unexplained.

Coronary arteriography and left ventriculography are essential guides to the surgical management of ischemic heart disease. It is generally believed that coronary arterial narrowing at the time of angiography reflects fixed atherosclerotic obstructions, and that impaired wall motion means recent or old myocardial infarction. Although there have been morphologic studies which have shown a generally good correlation between antemortem coronary occlusions and gross or histologic evidence of occlusion at autopsy,1-4 there is little information comparing the function at the time of left ventriculography with the degree of myocardial necrosis or fibrosis, or associated coronary occlusions at autopsy.4 A recent study comparing ventricular function from angiography with myocardial histology obtained from small biopsies at the time of operation suggests that hypokinesis at the time of ventriculogram does not always correlate with myocardial scar.6

To examine the accuracy of the morphologic information provided at the time of cardiac catheterization, we compared the clinical and pathologic assessments of coronary arterial disease and left ventricular myocardial damage. Our findings indicate that coronary arteriography is better at assessing coronary occlusive disease than is left ventriculography at assessing myocardial necrosis or fibrosis.

Materials and Methods

Patients from the autopsy files of The Johns Hopkins Hospital were included in this study if their heart had been studied pathologically after coronary arteriography and fixation in distention,4-7 and if they had had coronary arteriography and left ventriculography within three months of death. The reporting system recommended by the American Heart Association8 was employed for evaluation of both coronary and myocardial segments.

Clinical Studies

Cine coronary arteriograms were performed by the selective technique of either Sones or Judkins. Left ventriculography was performed in the 30° right anterior oblique projection while 30 to 40 cc of meglumine diatrizoate (Renografin 76) was injected into the left ventricle. The percent of diameter reduction was determined by visual estimation for as many of the 15 coronary artery segments4 as could be visualized. The positions of the ventricular wall in the systolic and diastolic position were traced. Left ventricular wall motion was evaluated in each heart by visual estimation for the five segments seen in the right anterior oblique projection.6 Descriptions of wall motion as good to fairly good, fair, poor or absent as seen on ventriculography for each segment were assigned grades on a scale of 0 to 4+, respectively.

Each coronary arteriogram and angiocardiogram was reviewed by a group of three or more observers at the same time and a consensus opinion was recorded. For the purpose of this study all arteriograms and ventriculograms were reviewed to corroborate the recorded findings. When significant discrepancies between the angiograms and the pathological studies were encountered the clinical studies were again reviewed.

Pathological Studies

Hearts were obtained at autopsy, weighed and inspected. Coronary arteriography was performed on the fresh heart with a barium-gelatin-pigment mass with formalin added just prior to injection through plastic cannulae tied into the coronary arteries at pressures in the range of 100 to 150 mm Hg. The heart was fixed in distention overnight with for-
malin introduced at 20 to 40 cm H₂O pressure through tubes tied into the atria and great vessels. Stereoscopic radiographs of the intact heart and its transverse "breadloaf" sections were prepared. Photographs were obtained, and representative portions of myocardium were taken for histological study by usual methods.

To evaluate the coronary arterial tree, each postmortem arteriogram was reviewed and a measurement of coronary luminal narrowing obtained for each segment. The point of maximum narrowing was located and the lumenal diameter measured directly on the biplane radiographs with an eyepiece micrometer calibrated to 0.1 mm. The "typical" or normal lumenal diameter for that segment was similarly measured from the radiographs. The diameters for the segment and its point of maximum narrowing were determined by agreement between two observers. Percent narrowing for that segment was then calculated from the measurements. Multiple transverse sections of the coronary arteries were made at all sites of narrowing on the postmortem angiogram and histologic examination of each of these segments was made to determine the cause and the degree of luminal narrowing.

To evaluate left ventricular myocardium, the gross changes in each left ventricular segment corresponding to the ventriculogram segments were examined and the degree of myocardial injury graded on a scale of 0 to 4+. The grades corresponded respectively to no morphological abnormality and slight, moderate, severe and complete or transmural replacement fibrosis. Multiple histologic sections (a minimum of ten per heart) were studied to confirm these grades. All myocardial or coronary arterial lesions determined histologically to be more recent than the time of catheterization were excluded from consideration.

**Correlation**

After all measurements of pre and postmortem coronary arterial and myocardial abnormalities were performed independently and blindly by at least two observers, correlations of the clinical and pathologic features of each case were made and the similarities and differences recorded. Reduction of coronary arterial lumen area by 75% or more, corresponding to a diameter reduction of 50%, is regarded as a critical or significant stenosis. Where significant discrepancies were noted the case was again reviewed in an attempt to discover an explanation.

**Results**

The 28 patients (table 1) ranged in age from 35 to 75 years (average 58) and 20 were men. Each had undergone cardiac catheterization with coronary arteriography within three months of death, 23 of the 28 within less than one month. All patients died following cardiac surgery: nine patients died at operation, five within 24 hours, 12 between one and 30 days and two patients over one month after operation. In 19 patients operations had been performed for ischemic heart disease with coronary artery bypass graft implantation.
and/or aneurysmectomy. Four patients had valve replacements only and five had operations for both valvular and ischemic heart disease.

Arteriographic Correlations
Pre and postmortem coronary arteriograms were compared in 315 segments (table 2). In 294 (93%) segments the

FIGURE 1. Clinical overestimation of coronary narrowing from apparent spasm of left main and proximal right coronary arteries. Top) Angiogram frames of right (RCA) and left (LCA) coronary injections. Arrows show luminal narrowing just distal to catheter tip. Bottom left) Postmortem coronary angiogram. Note normal arteries and saphenous vein bypass grafts. Bottom right, upper) Cross section through left (LV) and right (RV) ventricles showing normal myocardium. Bottom right, lower) Cross section of right coronary artery (RCA) taken at point of narrowing seen at clinical study. (H&E, × 12½)

FIGURE 2. Clinical underestimation of coronary narrowing from ostial lesion of diagonal branch of left anterior descending coronary artery (LAD). Left) Postmortem coronary angiogram. Note saphenous vein graft (SVBG) and mitral prosthesis. Right upper) Area in box at left showing ostial narrowing of diagonal (Diag) branch of LAD. Right lower) Histological section of ostial lesion. (H&E, × 12½)
The 28 hearts had 140 segments in which the ventriculographic determination of motion and the severity of myocardial disease present at the time of clinical study could be assessed (table 3). A comparison of degrees of pathological injury and reduced wall motion for the three types of hearts studied is shown in table 4. In 24 of 26 (92%) segments good or fairly good wall motion on ventriculogram there was morphologically normal myocardium. In the 58 segments in which there were myocardial lesions of any size and of an age to have been present at the time of ventriculography, reduced wall motion was noted in 57 (98%).

The ten segments which showed a ventricular aneurysm on pathological examination had markedly reduced or absent wall motion in each instance.

Differences in the ventriculographic and pathological semiquantitative assessments greater than two grades, however, were found in 73 (52%) of the 140 segments: in one segment fair (2+) motion was seen in association with a transmural infarct; in 72 segments the reduction of wall motion angiographically was greater than the severity of myocardial injury. In seven (5%) segments akinesis was noted at left ventriculography but the myocardium was morphologically normal. In 58 (71%) of the 82 myocardial segments with no pathological lesion at autopsy there was a significant impairment of wall motion seen by ventriculogram. Thus, good wall motion meant normal myocardium, but normal myocardium did not mean good wall motion; scar meant reduced wall motion but reduced wall motion did not mean scar.

In the patients with ischemic heart disease 14 (47%) of the 30 poorly moving but morphologically normal segments were adjacent to segments with infarcts or aneurysms (fig. 3). The other 16 segments were not adjacent to infarcts but were present in the distribution of critically narrowed coronary arteries and were in hearts with dilatation and hypertrophy of the left ventricle. The five hearts with valvular and ischemic heart disease had eight structurally normal but poorly moving segments: five of the eight segments were adjacent to infarcts, and three were in hypertrophied and dilated hearts in which an infarct was present. The four

### Table 3. Myocardial Injury and Wall Motion Reduction by Segments

<table>
<thead>
<tr>
<th>Pathological Injury</th>
<th>Anterobasal</th>
<th>Anterolateral</th>
<th>Apical</th>
<th>Diaphragmatic</th>
<th>Posterobasal</th>
<th>Total</th>
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<td>0</td>
<td>22</td>
<td>12</td>
<td>14</td>
<td>15</td>
<td>19</td>
<td>82</td>
</tr>
<tr>
<td>1+</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>6</td>
<td>2</td>
<td>19</td>
</tr>
<tr>
<td>2+</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>3+</td>
<td>0</td>
<td>3</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>4+</td>
<td>1</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>28</td>
<td>28</td>
<td>28</td>
<td>28</td>
<td>28</td>
<td>140</td>
</tr>
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</table>

### Table 4. Comparison of Myocardial Lesions and Reduction of Wall Motion

<table>
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<tr>
<th>Pathological injury</th>
<th>Ventriculographic reduction of motion</th>
<th>Total segments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease (19 patients)</td>
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<tr>
<td>1+</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>2+</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>3+</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>4+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Valvular and Ischemic Heart Disease (5 patients)</td>
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<td>0</td>
</tr>
<tr>
<td>1+</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>2+</td>
<td>0</td>
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</tr>
<tr>
<td>3+</td>
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<td>0</td>
</tr>
<tr>
<td>4+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Valvular Heart Disease (4 patients)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1+–4+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>15</td>
</tr>
</tbody>
</table>
hearts with valvular heart disease only had no pathological lesions in the 20 myocardial segments studied. Nevertheless, all of these segments had shown reduced or absent motion on ventriculography. Three of the four hearts had no significant coronary artery lesions but all were markedly hypertrophied (fig. 4).

Discussion

Angiography of the coronary arteries and left ventricle are generally used as the gold standards for assessing occlusive disease of the coronary arteries and of the degree of myocardial dysfunction and damage related to coronary artery disease and/or valvular heart disease. Narrowing of the coronary arteries at angiography usually represents atherosclerotic obstruction and the degree and location of such narrowing is the major guide to surgical therapy. In addition, the viability of myocardium in the distribution of a narrowed or occluded vessel is assessed by wall motion of the segment at the time of ventriculography, and absence of wall motion is frequently interpreted as indication of infarction of that segment. Thus, whether a given myocardial segment appears viable by ventriculogram may weigh heavily in the decision to bypass or not to bypass a critically narrowed vessel feeding that segment. With the advent and virtual explosion of myocardial revascularization procedures that are heavily dependent upon angiographic information, it is important to reassess the accuracy of the morphologic information provided by these techniques. The purpose of this study was to correlate the clinical information regarding coronary occlusive disease and myocardial damage provided at the time of cardiac catheterization with postmortem findings.

The results of our study show that occlusive lesions of the coronary arteries are generally detected by clinical coronary arteriography. In this study the problem of variation in interpretations of obstructions of the coronary arteries has been largely avoided by using a consensus opinion for both clinical and pathological determinations. Previous studies correlating postmortem with clinical coronary arteriography have shown, in general, a similar close correspondence of the two determinations. Most of the significant discrepancies between the premortem studies and the autopsy findings in our study occurred at the ostia of the diagonal branches of the left anterior descending coronary artery. Failure to observe such lesions appeared to be due to the overlapping of radiographic images of the two vessels near the branch point. Unless the site of a branch is perpendicular to the angle of viewing, such overlap may occur. Studies in which coronary artery lesions were assessed at autopsy without postmortem arteriograms have described more severe disease at autopsy than was detected by clinical arteriography. We believe, however, that such correlations without the use of
postmortem angiography can be misleading since luminal occlusion estimates made on the empty transected coronary artery tend to overestimate the degree of occlusion produced by atherosclerotic plaque. The pathological overestimate is particularly severe when the plaque is eccentric. The so-called slit-like lumen is rarely seen if the coronary arteries are studied at autopsy after arteriography, as the slit appears when the nonatherosclerotic portion of vessel wall collapses against the more rigid eccentric atheroma. The occurrence of more severe atherosclerosis on direct gross examination than is revealed by postmortem arteriography has been noted by others.\(^8\)

Overestimating the size of luminal narrowing is a far less frequent problem using clinical coronary arteriography. In our study only six (2%) of the 294 segments were overestimated, i.e., a critical stenosis was reported when there was none at autopsy. In three of the instances the explanation for the overestimate was not evident. In three, however, >75% narrowings were described at the ostia of left or right main coronary arteries in otherwise widely patent coronary trees. Morphologically these mainstem vessels were entirely normal, and in retrospect the narrowings which were clearly evident at the catheter tips were probably due to spasm\(^{13,14}\) (fig. 1). Thus, in general, coronary arteriography reliably assessed the presence and degree of atherosclerotic disease with few clinically significant over or underestimations of disease. Of the errors, the majority were underestimations of the severity of coronary disease due to overlapping branch vessels, and a few were overestimations due mostly to coronary spasm.

Left ventriculography was not, however, as reliable in assessing myocardial necrosis or fibrosis as coronary arteriography was in detecting atherosclerotic occlusions. Although an infarct or aneurysm was almost always associated with reduced wall motion (with the degree of motion reduction corresponding to the thickness of wall involved by infarction) reduced wall motion did not always correlate with myocardial infarction. Reduced or absent wall motion was present in 58 of 82 (68%) segments which showed no abnormality on pathological study. Of the 58 morphologically normal but poorly moving segments 19 (33%) were located adjacent to infarcts or aneurysms. It seems probable that in this situation the reduced motion can be accounted for by the mechanical effect of the adjacent fibrotic and poorly moving segment. The remaining 39 (67%) of the 58 segments were not adjacent to infarcted segments: 20 (51%) of the 39 segments were found in hypertrophied and sometimes dilated hearts with valvular disease and insignificant coronary artery disease and 19 (49%) were in dilated and hypertrophied hearts in the distribution of critically narrowed coronary arteries.

There are a number of possible explanations for these underestimations of left ventricular viability by ventriculogram. In a dilated ventricle the systolic wall excursion required to displace a given volume of blood with each heart beat is less than would be required in a normal sized ventricle to displace the same volume. It may be that some of the apparent reduction of wall motion seen on ventriculograms reflects the altered topography of the abnormal left ventricle. Also, in patients with coronary artery disease, normal myocardial segments in the distribution of critically narrowed coronary arteries may show reduced or even absent motion due to ischemia. Some investigators have demonstrated improvement of segmental wall motion after nitroglycerin\(^a\) suggesting that ischemia may be responsible for some hypokinesis or frank akinesia at the time of cardiac catheterization. That reduced motion may occur with normal coronary arteries is illustrated by three of the four patients with valvular heart disease where all segments had reduced or absent motion despite presumably normal coronary blood flows.

In conclusion, the findings suggest that a coronary artery narrowing detected on coronary arteriography is almost always associated with a pathological lesion. On occasion, errors occur because occlusive lesions are hidden by vessel overlap or because arterial spasm is interpreted as a fixed obstruction. Decreased segmental wall motion on left ventriculogram is usually found when an infarct or aneurysm is present. However, many poorly moving or akinetic segments have normal myocardium. Such normal but hypodynamic segments may be explained by transient ischemia. In some instances, particularly in association with patent coronary arteries, segmental wall motion abnormalities may relate to the overall topography of the left ventricle, in which poorly moving segments occur either on the margins of scars or throughout dilated ventricles where less than normal wall motion is adequate for normal cardiac output.

References

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