Selective Cine Coronary Arteriography

Correlation with Clinical Findings in 1,000 Patients

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SYMPTOMS of coronary arterial disease generally occur in association with obstruction of the coronary arteries in the absence of valvular defects. Until recent years accurate information relative to obstructive lesions in the coronary arteries could not be obtained from the living patient.

In the absence of definite electrocardiographic evidence of myocardial infarction, clinical diagnosis of coronary disease has been dependent principally on the elicitation and proper evaluation of the history, and therefore, the accuracy has varied with the care, ability, and experience of the physician. In other fields of medicine in which clinical diagnoses can be compared with those based on objective criteria, it is evident that errors in clinical diagnoses are common. The development of selective cine coronary arteriography has made possible the correlation of clinical syndromes and evidence of arterial obstruction during life. If this method of study is valid, there should be a close relationship between the typical clinical syndromes (angina pectoris and myocardial infarction) and the presence of significant arteriographic abnormality.

Group Studied and Methods of Study

The clinical records of 1,001 patients who had selective cine coronary arteriography between January 1, 1961, and March 31, 1964, were reviewed. One patient was excluded from the series because the arteriographic study was considered to be technically inadequate; the left coronary artery could not be catheterized. About 1,000 patients examined before 1961 were not considered because of the possibility of technical or interpretive errors in early experience. Patients who had primary valvular disease were not included. In almost all of the 1,000 patients in this series coronary disease had been suspected or diagnosed by one or more physicians before this study. Exceptions were 37 patients studied because of electrocardiographic abnormalities only, unexplained cardiomegaly, congestive heart failure, pericarditis, embolus, or extreme hypercholesterolemia. In most patients chest pain was the presenting symptom.

Clinical Diagnosis

All clinical records were evaluated by one physician (W.L.P.) without knowledge of the arteriographic findings. Diagnoses were based upon data from the clinical history, the physical examination, and the electrocardiogram. In some patients more than one diagnosis was made, so that the total number of diagnoses exceeded the total number of patients. For example, angina pectoris and myocardial infarction may occur in the same patient.

Clinical Classes

The following clinical classes were used:
1. Normal: Chest pain had no features of coronary disease.
2. Probably Normal. Pain that did not seem to be of coronary artery origin, but some features were suggestive.
3. Atypical Angina Pectoris. Pain that was thought to be due to angina pectoris but the precipitating factors were unusual or inconstant.
4. Angina Pectoris (Class I, II, or III, Functional Classification). Pain somewhere in the upper half of the body which was precipitated by walking and relieved promptly (within 15 minutes) by rest. Other precipitating or potentiating factors were common in this group but all had exertional pain. None had rest pain.
5. Angina Pectoris (Class IV, Functional Classification). Pain at rest as well as on physical exertion.
6. Rest Pain Only. Pain that appeared to be characteristic of angina but was not precipitated by exertion; pain lasted less than 15 minutes.
7. Coronary Failure.$ Pain that occurred with or without apparent precipitation and persisted 15
minutes to several hours (coronary insufficiency, premonitory pain, intermediate syndrome).

8. **Myocardial Infarction.** QRS abnormalities that were considered characteristic at the time of arteriographic study. Most patients in this group had typical histories of prior infarction.

9. **Possible Myocardial Infarction.** Borderline QRS abnormalities or sharp inversion of T-waves which was consistent with intramural infarction.

10. **Congestive Failure.** Either clear evidence of congestive failure on examination or a history strongly suggestive of it in the recent past. In some cases there was no clinical evidence of coronary disease.

11. **Embolus.** The presenting clinical manifestation without apparent source.

12. **Pericarditis.** Possibly due to prior myocardial infarction.

13. **Abnormal Electrocardiographic Findings.** S-T or T-wave abnormalities or conduction defects in the absence of coronary symptoms.

14. **Cardiac Enlargement of Unknown Etiology.** Cardiac enlargement demonstrated roentgenographically.

15. **Arrhythmia.** Cardiac arrhythmia without apparent clinical basis.

16. **Hypercholesterolemia.** Extreme elevation of the serum cholesterol without clinical heart disease.

**Electrocardiograms and Exercise Tests**

The electrocardiograms (standard and unipolar limb leads and eight precordial leads) were recorded almost exclusively on high-sensitivity photographic electrocardiographs (Sanborn Twin Beam). Exercise tolerance tests were done on direct-writing electrocardiographs. Routinely, three limb leads and one precordial lead were obtained immediately and 3 and 6 minutes after exercise, consisting of 50 trips over a two-step staircase in about 3 minutes, unless the test had to be terminated because of the development of pain. In a few instances more than one precordial lead was recorded after exercise. Exercise tests in 107 patients were reviewed without knowledge of arteriographic findings. Few tests were done in patients who had abnormal electrocardiograms at rest. Mattingly’s criteria were used in evaluation of exercise tests.

**Arteriographic Diagnoses**

An arbitrary classification of the arteriographic obstruction was employed. Obstructions of the major arteries or major branches of these arteries were considered. For the purpose of this study, obstructions of the lumen estimated to be 30% or less were considered slight. They were tabulated separately from the strictly normal studies. The degree of estimated obstruction in each instance in this study refers to the maximal involvement in any major vessel. Often multiple vessels were involved. The following classification was used:

1. **None.** Smooth-walled vessels showing no variations in lumen diameter.

2. **Slight.** Barely perceptible to 30% narrowing of lumen diameter.

3. **Moderate.** Narrowing of lumen diameter of between 30 and 50%.

4. **Severe.** Narrowing of lumen diameter of between 50 and 90%.

5. **Subtotal.** Almost complete (90% or more) obstruction of lumen.

6. **Total.** Complete obstruction of lumen.

Repeated injections of contrast medium were employed in multiple roentgenographic projections before and after the use of nitroglycerin or isosorbide dinitrate (Isordil), to minimize misinterpretation of functional constriction or artifacts arising from the plane of projection. There were only two known false-positive arteriographic diagnoses in this series. In these two patients, strictures were encountered in the proximal right coronary artery which were considered to be organic because they did not completely disappear within 2 to 4 minutes after sublingual administration of nitroglycerin, 1/150 grain. Repeated study demonstrated the segmental narrowing to be absent in one case. In the other case the segment of narrowing disappeared completely after oral administration of 10 mg of isosorbide dinitrate. Obvious functional constrictions due to arterial spasm in the proximal right coronary artery were encountered in more than 50 patients. Except for the two instances mentioned, these strictures were abolished within 2 to 4 minutes after administration of nitroglycerin or isosorbide dinitrate. Systolic constrictions of the anterior descending branch of the left coronary artery due to myocardial bridges did not appear to be associated with symptoms.

From the standpoint of left ventricularograms, ventricular aneurysm was considered to be present when there was paradoxical motion of a segment of the myocardium as contrasted to failure of normal contraction without bulging.

**Results and Discussion**

In this type of clinical investigation it is essential that the clinical diagnosis be made without knowledge of the findings on the arteriographic studies. If selective cine coronary arteriography is useful as a diagnostic measure, there must be a close correlation between the clinical diagnosis of typical angina pectoris and the arteriographic evidence.
of obstruction, unless functional constriction in normal vessels or obstruction limited to minute peripheral coronary arteries is important. Poor correlation could be explained by inadequacy of clinical records, incompetent clinical evaluation, technical or interpretive errors, or lack of relationship between clinical symptoms and arteriographically demonstrated obstructions. If perfect correlation were reported, one could question the integrity of the investigators.

The degree of arterial obstruction determined by arteriography, in relation to the age and sex of the patients is shown in table 1. The youngest man who had a significant arterial obstruction was 27 years of age and the youngest woman, 32. The striking predominance of men over women (786 to 214) even in the older age groups suggests that the economic implications of coronary disease influenced the patient's and the physician's decisions to have diagnostic studies.

In 207 patients a clinical diagnosis of angina pectoris, functional class I, II, or III, was made, and in 194 (93.7%) of these patients there was evidence of moderate or severe obstruction of one or more major vessels (table 2). The clinical records of the 13 patients in whom no significant abnormality was found were reviewed. In two instances severe arterial hypertension was present. In one of these, administration of angiotensin resulted in reproduction of the pain coincident with rise in arterial blood pressure. Angina pectoris has been reported to occur in patients having severe arterial hypertension in the absence of evidence of coronary disease.5 A third patient had left bundle-branch block, a ventricular aneurysm, mitral insufficiency, and congestive heart failure due to primary myocardial disease without demonstrable arterial obstruction. Angina may have resulted from reduced coronary perfusion secondary to diminished cardiac output. If these three cases

Table 1

<table>
<thead>
<tr>
<th>Age, range yr</th>
<th>Sex</th>
<th>No. of patients</th>
<th>Severity of obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>20-29</td>
<td>M</td>
<td>19</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>30-39</td>
<td>M</td>
<td>156</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>35</td>
<td>30</td>
</tr>
<tr>
<td>40-49</td>
<td>M</td>
<td>307</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>76</td>
<td>36</td>
</tr>
<tr>
<td>50-59</td>
<td>M</td>
<td>221</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>76</td>
<td>38</td>
</tr>
<tr>
<td>60+</td>
<td>M</td>
<td>83</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>24</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>1000</td>
<td>274</td>
</tr>
</tbody>
</table>

Table 2

Correlation of Clinical Diagnosis with Arteriographic Evidence of Moderate or Severe Obstruction

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>Total no. of patients</th>
<th>Abnormal findings on arteriograms; no. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina pectoris, class I, II, or III</td>
<td>207</td>
<td>194 (93.7)</td>
</tr>
<tr>
<td>Uncorrected classification</td>
<td>207</td>
<td>194 (98.0)</td>
</tr>
<tr>
<td>Corrected classification*</td>
<td>198</td>
<td>174 (98.9)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>196</td>
<td>174 (98.9)</td>
</tr>
</tbody>
</table>

*See text for explanation.
are excluded, significant coronary obstruction was demonstrated in 95% of patients who had angina pectoris. On careful review of the records of the remaining 10 patients, it was thought that six should have been classified originally as having atypical angina pectoris. If this reclassification is permitted, significant coronary obstruction was demonstrated in about 98% of patients who had angina pectoris (table 2). In most instances the arterial obstruction was severe (tables 3 and 4). It appears, therefore, that functional constriction in the absence of obstructive lesions or arterial disease affecting only minute arteries causes angina rarely if ever. It is possible that some condition outside the heart could be responsible for occasional instances of exertional pain relieved promptly by rest.

The close correlation of the clinical diagnosis of angina pectoris, classes I, II, and III, with arteriographic evidence of significant obstruction does not necessarily establish this method of study as a diagnostic technique. Conversely, severe arterial obstructions should not be the majority encountered in persons thought to be normal clinically. Table 5 shows the correlation of clinical diagnoses with arteriographic findings in the 1,000 patients. In 95.6% of patients thought to be free from coronary arterial diseases, the arteriograms were normal; and in 80.6% of patients thought probably to be free from coronary arterial disease, the arteriograms were normal. Some coronary disease must be expected in the age groups studied, even in the absence of clinical symptoms.

Atypical angina pectoris is a vague term, and it has a different meaning for each clinician. In this study the term was applied to cases in which the pain had some of the characteristics and precipitating factors of angina pectoris, but there were unusual or inconstant features in the history. Unusual location of the pain alone was not considered to be an atypical symptom. An incidence of arteriographic abnormalities of only 64.5%...

Table 3

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>Total no. of cases</th>
<th>Extent of obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>More than 50%, % of cases</td>
</tr>
<tr>
<td>Angina pectoris,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>class I, II, or III</td>
<td>194</td>
<td>98.5</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>174</td>
<td>100.0</td>
</tr>
</tbody>
</table>

*Classified in the text as severe, subtotal and total obstruction.

Table 4

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>Extent of maximal obstruction</th>
<th>Total no. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None or slight (0-30%)</td>
<td>Moderate (30-50%)</td>
</tr>
<tr>
<td>Normal</td>
<td>65</td>
<td>2</td>
</tr>
<tr>
<td>Probably normal</td>
<td>163</td>
<td>13</td>
</tr>
<tr>
<td>Atypical angina pectoris</td>
<td>50</td>
<td>9</td>
</tr>
<tr>
<td>Angina pectoris, class I, II, or III</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>Angina pectoris, class IV</td>
<td>22</td>
<td>5</td>
</tr>
<tr>
<td>Rest pain only</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Coronary failure</td>
<td>37</td>
<td>4</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Possible myocardial infarction</td>
<td>13</td>
<td>3</td>
</tr>
<tr>
<td>Congestive failure</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>
(table 5) might be expected in this group. The high incidence of atypical angina in this series is at least partly a result of the large number of patients included in the series who were referred for study because of confusing clinical features. The inclusion of patients having any deviation from the history of exertionally induced pain relieved promptly by enforced rest will result in an increased incidence of normal arteriographic findings, because many of these patients have normal hearts. Often physicians speak of “typical angina pectoris” when the history is atypical upon careful scrutiny.

The high incidence of patients who had experienced pain at rest as well as on exertion (angina pectoris, class IV) is out of proportion to the incidence encountered in ordinary clinical practice (table 5). Many of these patients were referred to us because of the difficult therapeutic problems presented. There may have been no objective clinical evidence of disease. Emotional coloring of the illness was frequent in this group even when obstructive lesions were demonstrable, and this added to diagnostic confusion. An intelligent patient could give a precise history on the basis of common knowledge or reading of lay literature. The neurotic with convulsion obtains secondary gain only by having the most severe and disabling symptoms. The use of narcotics was not uncommon in patients who had symptoms at rest (eight persons).

Most of the patients having angina pectoris, class IV, had been hospitalized on numerous occasions and by a number of physicians, and had received various forms of medical and surgical therapy. It is not surprising, therefore, that the incidence of normal arteriographic findings in patients having angina pectoris, class IV, was about double that encountered in persons having milder symptoms of angina pectoris (table 5). Conversion neurosis was a common problem among those in whom the arteriographic findings proved to be normal.

Patients who had pain of anginal type and duration but which occurred only at rest (42 patients) presented a difficult diagnostic problem, as did patients (174) who had prolonged pain with or without additional typical exertional pain (coronary failure, coronary insufficiency, intermediate syndrome). The large number (216) of such patients in this series is again the result of prior selection for referral by the patient’s physicians. Arteriographic abnormalities were observed in almost 80% of each of these two groups of patients (table 5). The need for caution in drawing conclusions concerning the therapeutic claims in the treatment of patients having prolonged pain is obvious, since more than 20% of such patients had no disease.

Early in this study it was decided to disregard prior diagnosis of myocardial infarction unless there was sufficient residual evidence in the QRS complexes to support this

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>No. of patients</th>
<th>% correlation</th>
<th>Arteriographic evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>68</td>
<td>95.6</td>
<td>No significant obstruction</td>
</tr>
<tr>
<td>Probably normal</td>
<td>201</td>
<td>80.6</td>
<td></td>
</tr>
<tr>
<td>Atypical angina pectoris</td>
<td>141</td>
<td>64.5</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris, class I, II, or III</td>
<td>207</td>
<td>93.7</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris, class IV</td>
<td>173</td>
<td>87.3</td>
<td></td>
</tr>
<tr>
<td>Rest pain only</td>
<td>42</td>
<td>78.6</td>
<td>Significant obstruction</td>
</tr>
<tr>
<td>Coronary failure</td>
<td>174</td>
<td>78.7</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>176</td>
<td>98.9</td>
<td></td>
</tr>
<tr>
<td>Possible myocardial infarction</td>
<td>50</td>
<td>74.0</td>
<td></td>
</tr>
<tr>
<td>Congestive failure</td>
<td>63</td>
<td>87.3</td>
<td></td>
</tr>
<tr>
<td>Other*</td>
<td>37</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Embolus, pericarditis, arrhythmias, abnormal electrocardiograms, abnormal roentgenograms, and xanthoma tuberosum.
diagnosis at the time of arteriographic study. Many patients had a history of myocardial infarction, but the electrocardiogram before arteriographic study showed no diagnostic evidence of infarction; often normal arteriograms were found. The possibility of myocardial infarction in the absence of coronary atherosclerosis is well known but its frequency is debatable. In this series arteriographic evidence of obstruction was found in 98.9% of the cases of myocardial infarction (tables 2, 4, and 5). The distribution of patients in relation to the degree of the maximal obstruction is shown in table 4. All patients having myocardial infarction who had arteriographic abnormalities had severe obstruction, and in most there was almost total or total occlusion. In general, the correlation was good between the location of the myocardial infarction and the location of the severely obstructed vessel supplying the affected areas. The clinical records of the two patients who had QRS changes consistent with myocardial infarction and normal arteriograms were reviewed. Neither had a history of prolonged severe chest pain. One record interpreted as showing remote posterior myocardial infarction should have been considered possible, rather than definite, infarction. The other had QRS changes of anteroseptal and posterior infarction, but the electrocardiogram was normal 1 month later. In this series there were no instances of hypertrophic subaortic stenosis resulting in electrocardiographic abnormalities similar to those of myocardial infarction, though cases of this type have been encountered outside this series.

 Clinically, it appeared that congestive heart failure was due to coronary disease in some cases and in others it was of unknown etiology. Whenever there was no complicating condition, severe arterial obstruction of multiple vessels was the rule. Frequently another etiological factor placed an additional burden on the heart. Ventricular aneurysm, mitral insufficiency, cardiac arrhythmias, and arterial hypertension were common conditions (table 6). In one case an arteriovenous fistula due to a Beck-II operation was an additional stress.

Table 6

<table>
<thead>
<tr>
<th>Clinical, Electrocardiographic, and Arteriographic Findings in Sixty-Three Patients Having Diagnosis of Congestive Failure</th>
<th>Arteriographic findings</th>
<th>Electrocardiograms</th>
<th>Aneurysm</th>
<th>Mitral insufficiency</th>
<th>Infarct zone</th>
<th>Subaortic stenosis</th>
<th>Hypertension</th>
<th>Total no. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>None or slight</td>
<td>Moderate</td>
<td>Severe</td>
<td>None</td>
<td>5</td>
<td>32</td>
<td>8</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Posterior</td>
<td>16</td>
<td>30</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
<td>3</td>
<td>6</td>
<td>5</td>
<td>8</td>
<td>5</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

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CORONARY ARTERIOGRAPHY

Table 7

Other Indications for Arteriographic Study

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of cases</th>
<th>Abnormal arteriograms, cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embolus</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Electrocardiographic abnormalities</td>
<td>19</td>
<td>6</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Roentgenographic abnormalities</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Xanthomatosis</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

In 60.4% (32 of 53) of those having severe coronary disease, there was electrocardiographic evidence of myocardial infarction, and an additional 13.2% (7 of 53) had left bundle-branch block that might have obscured changes due to infarction. In eight no significant obstructions were found.

Other indications for study were encountered occasionally (table 7). In eight cases of pericarditis features were suggestive of coronary disease, but no obstructive lesions were found. Electrocardiographic abnormalities without clinical symptoms, cardiac enlargement of unknown cause, and cardiac arrhythmias were indications for study in some cases. The source of an embolus was sought in one study.

Left ventriculography was carried out in most patients who had obstructive arterial lesions. Evidence consistent with ventricular aneurysm was found in 80 cases. In many there was no obvious evidence of aneurysm in roentgenograms of the chest. The incidence of aneurysm was higher than expected by us.

The serum cholesterol levels were tabulated for 147 men less than 40 years of age and correlated with arteriographic findings (table 8). In general, the incidence of normal findings was high when the level was less than 225 mg per 100 ml and the frequency of abnormal findings was high when the level exceeded 300 mg. However, the level was of limited diagnostic value in the individual patient.

One of the difficulties encountered in the correlation of clinical and arteriographic findings is the necessarily arbitrary separation of degrees of estimated arterial obstruction. It is apparent that mild degrees of obstruction rarely cause angina pectoris or myocardial infarction. Arteriographic evidence of obstruction is almost always severe in symptomatic patients. Therefore, precise classification of relatively minor degrees of obstruction does not seem to be so important as it was considered to be during early progress of the study.

The frequency of diagnosis of coronary atherosclerosis in the absence of satisfactory clinical evidence is recognized by cardiologists. Such a diagnosis, however, may alarm the anxious patient. In the present study almost all patients were told by some physicians that coronary disease was present or suspected. Actually about 37% of the 1,000 patients had no significant obstruction, and the arteriograms of more than 27% were entirely normal (tables 1 and 9). A diagnosis of coronary disease had been made in 700 of the 1,000 patients in this review of clinical rec-

Table 8

Serum Cholesterol Levels as Related to Arteriographic Findings in 147 Men Less Than 40 Years of Age

<table>
<thead>
<tr>
<th>Serum cholesterol level, mg/100 ml</th>
<th>Arteriographic findings</th>
<th>Normal* No. of patients</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 200</td>
<td>23</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>200-224</td>
<td>15</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>225-249</td>
<td>13</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>250-274</td>
<td>9</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>275-299</td>
<td>9</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>300-349</td>
<td>4</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>350 +</td>
<td>1</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>74</td>
<td>73</td>
<td></td>
</tr>
</tbody>
</table>

*No arteriographic evidence of obstruction.

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ords; in about 17% of the 700 there were no arteriographic abnormalities. It would be difficult significantly to increase the percentage of correlation on the basis of clinical information that is available at this time. Rigid definition of typical angina pectoris kept the percentage of correlation high in this group, and restriction of diagnosis of myocardial infarction to those who had definite QRS abnormalities was necessary. However, atypical symptoms occur in a significant number of patients with coronary atherosclerosis. Some improvement in correlation might result if each patient had been interviewed personally by the evaluating physician. However, consistent histories written by several physicians were available; experience and appraisal of the recorded data were responsible for the higher percentage of correlation with the abnormal arteriograms than the correlation on the basis of the referral diagnosis.

In this study about 17% of the 300 patients who were thought to have noncoronary symptoms had significant abnormalities in the arteriograms. It is difficult, of course, to be certain that the symptoms were related to the obstructions demonstrated. The percentage of patients in whom false-negative diagnoses were made by referring physicians could not be estimated because there was no referral for study in most of these cases.

The value of Venn diagrams in statistical studies have been demonstrated by Feinstein. Figure 1 shows the primary clinical manifestations in the majority of 627 patients who had abnormal arteriographic findings. Pain was the most common manifestation. Included in the group with pain are 91 patients who were thought to have atypical angina pectoris. Myocardial infarction of uncomplicated nature was unusual because actively symptomatic patients are more likely to be referred for study.

The distribution of patients having what was considered typical pain is shown in figure 2. The number of patients who had myocardial infarction without additional pain syndrome is larger in figure 2 than in figure 1 because some patients who had congestive failure and myocardial infarction are included in figure 2.

The reactions of the patients to the report of normal arteriographic findings vary. In

### Table 9

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>Arteriographic obstruction, extent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of patients</td>
</tr>
<tr>
<td>Normal</td>
<td>250</td>
</tr>
<tr>
<td>Coronary atherosclerosis</td>
<td>123</td>
</tr>
<tr>
<td>Total</td>
<td>373</td>
</tr>
</tbody>
</table>

![Figure 1](image-url)

**Figure 1**

Common presenting conditions in patients having more than 30% obstruction of a major coronary artery. Patients who were thought to have noncoronary pain, electrocardiographic evidence of possible myocardial infarction without pain or congestive heart failure, or miscellaneous indications for arteriography (table 7) are omitted. Myocardial infarction refers to electrocardiographic diagnosis only. The numbers indicate the number of patients in each group.

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Pain spectrum. Myocardial infarction refers to a history of typical pain in addition to electrocardiographic evidence in this diagram. In 26 additional patients there was electrocardiographic but no clinical evidence of myocardial infarction: 16 of these had some other type of pain of coronary origin and are included in appropriate sections of the diagram. Atypical angina pectoris, included in figure 1, is omitted here.

most instances, the patients were visibly relieved; some of these patients had led a life of invalidism, and it is remarkable that they returned to a normal life. On the other hand, some patients showed an immediate transference of symptoms to some other organ system—sometimes while the physician was still giving them the report. The outlook is poor in this group. Patients did not insist that there must be something wrong with the heart which has not been found. Those who have abnormal studies accepted a candid report well. Perhaps those who most feared the documentation of obstructive lesions refused to have arteriography.

Summary
The clinical records of 1,000 patients who had adequate selective cine coronary arteriography were reviewed. The clinical diagnoses were made by a physician who had no knowledge of the arteriographic findings. Correlation of the clinical diagnoses with the arteriographic findings was made subsequently.

Symptomatic coronary disease was accompanied by arteriographic evidence of significant obstruction of major coronary arteries in most instances. A close correlation existed between the clinical diagnosis of angina pectoris without rest pain and significant arterial obstruction (95%). A similar correlation was found between QRS evidence of myocardial infarction and severe arterial obstruction (99%). The demonstrated arterial obstruction in patients who had angina pectoris almost always was severe and usually almost total or total in one or more major vessels. In myocardial infarction the demonstrated obstruction was always severe and generally almost total or total.

The correlation between clinical and arteriographic findings was moderately close in patients who had angina with symptoms at rest. The correlation between the arteriographic findings and less characteristic clinical syndromes (rest pain only, 79%, coronary failure, 78%, and especially atypical angina pectoris, 65%) was not so close. In congestive failure secondary to coronary disease, arterial obstruction was extensive unless ventricular aneurysm, mitral insufficiency, arrhythmia, arterial hypotension, or some other complication was present. Most patients thought to have noncoronary symptoms had no significant obstructive lesions.

In 37% of the entire group of patients, almost all of whom had been suspected of having coronary disease by some physicians, no significant arteriographic obstruction was demonstrated; in 27% the arteriograms were normal.

Diagnoses, based on appraisal of the clinical records without knowledge of the arteriographic findings, yielded 83% correlation with abnormal arteriographic findings in 700 patients thought to have coronary disease.

References
3. Blumgart, H. L., Schlesinger, M. J., and Zoll, P. M.: Angina pectoris, coronary fail-


A Problem of Retrograde Vertebral Flow, 100 Years Ago

Successful Operation for Subclavian Aneurism.—The first No. of the New Orleans Medical Record, edited by the well-known indefatigable Dr. Bennet Dowler, contains an interesting account, by Dr. A. W. Smyth, of a case of a mulatto man, thirty-two years of age, admitted into the Charity Hospital, New Orleans, May 9, 1864, with aneurism of the right subclavian artery. The tumour was of the size of a small orange, "was circumscribed and round in shape, filling up the posterior inferior triangle of the neck; strong pulsatory movement was visible even at some distance, and on applying the ear to its surface, a loud bellows sound was heard accompanying the arterial beat. . . ."

The patient was seen by a number of surgeons, and among others Dr. D. L. Rogers, of New York, who strongly urged the ligature of the innominate and carotid arteries at the same time. . . . "no difficulty was experienced in placing a ligature on the innominate artery a quarter of an inch below its bifurcation, and another on the carotid, an inch above its origin. On tying the former all pulsation stopped in the tumour. The temperature of the arm and hand was immediately increased, and in about forty-eight hours after the operation a perceptible undulatory motion was discovered in the arteries of the wrist. . . .

"On the 29th of May, fourteen days from the time of operating, a severe hemorrhage occurred, causing syncope rapidly, and ceasing of its own accord. . . ."

After studying the subject, Dr. S. concluded that the vertebral carries on almost the entire anastomosing circulation into the subclavian artery, and he therefore decided to ligate the former vessel. . . . "A marked decrease in the circulation of the arm was now apparent, the slight pulsation at the wrist disappearing. . . . No further hemorrhage having taken place after the second operation, the new wound healed rapidly; the ligature coming away on the tenth day. . . ."—American Intelligence: Domestic Summary. Amer J Med Sc 52: 280, 1866.
Selective Cine Coronary Arteriography: Correlation with Clinical Findings in 1,000 Patients
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