Circulatory Effects of Mitral Commissurotomy with Particular Reference to Selection of Patients for Surgery

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Of 60 patients with mitral stenosis referred as proper candidates for commissurotomy, less than half (27) proved suitable after further study. One half of these (13) had a good postoperative result as judged not only clinically but by objective physiologic measurements. The latter represent the best criteria for the effects of surgery since subjective impressions may be unreliable. Eight subjects originally advised to undergo surgery would now be rejected inasmuch as myocardial insufficiency and not mitral block was their predominant difficulty. Clinical criteria appear as yet inadequate for the selection of the proper candidate for mitral commissurotomy, as there is no good clinical means of establishing in every instance the presence of pulmonary hypertension which remains the best evidence of a significant degree of mitral block.

Since Bailey and Harken have demonstrated the feasibility of the surgical approach to the problem of mitral stenosis, patients with this lesion are being re-evaluated in numerous clinics with the possibility of commissurotomy in mind. It is well to remember that surgical treatment is directed at the relief of the mechanical difficulties imposed by mitral valve obstruction. It has been postulated and recently re-emphasized that chronic pulmonary hypertension is one of the chief resultants of significant mitral block. This postulate has been confirmed by physiologic studies which also showed decrements in pulmonary artery pressures after successful mitral surgery. Thus, it has become established that operative intervention can alleviate at least some of the consequences of mitral block. However, it has also been found that mortality is high in the markedly incapacitated rheumatic subject and therefore it has appeared that one should relieve mitral block before its effects reach an advanced stage. As a consequence of this type of reasoning, less and less attention is being directed at establishing that the symptoms and physiologic derangements of the patient with mitral stenosis are due predominantly to obstructive mitral block. Indeed, at the present time, if a patient has mitral stenosis and symptoms referable to the cardiorespiratory systems it has become almost axiomatic to ascribe these symptoms to a small mitral orifice and offer the patient surgery. Hence the criteria for the selection of patients for commissurotomy have become entirely clinical, based on signs and symptoms which are not specific to mechanical mitral block, and little or no attempt is being made to identify the presence of pulmonary hypertension except by inference.

The present report has been prepared on the basis of material accumulated over the past five years, in an attempt to focus more precisely upon the nature of the circulatory dysfunction produced by mitral block, to evaluate the effects produced by commissurotomy, and to define more specifically, if possible, the suitable candidate for this...
surgical procedure. Hemodynamic measurements coupled with clinical observations were utilised to define the patient's circulatory function prior to surgery as well as to establish the postoperative status. Early in our experience it became apparent that these hemodynamic data were essential to the evaluation of the surgical results of a procedure following which patients, desperately ill and anxious for relief, reported all grades of improvement. For adequate final appraisal objective measurements had to be available against which to titrate these subjective expressions of benefit.

This paper presents the experience of the authors with a group of patients referred to us as possible candidates for commissurotomy and includes our method of evaluation of the patient with mitral stenosis, as well as the results of commissurotomy when this operation has been performed.

METHOD OF STUDY

In the past five years 60 patients with mitral stenosis have been considered as possible candidates for mitral commissurotomy. The majority of these subjects were referred to us by their local physicians as suitable candidates. Prior to any decision regarding surgery, all these patients were closely observed in order to rule out not only such complications as active rheumatic carditis, subacute bacterial endocarditis and intractable heart failure, but also to establish the best possible status obtainable by medical means prior to evaluation by cardiac catheterization. This period of observation in the hospital lasted two to three weeks in most instances, but was extended to four or five months in several patients.

The method of physiologic appraisal, using the cardiac catheterization technic, has been detailed in a previous report, which also contains the criteria for interpretation of results. In this regard, it is important to stress the difficulties and potential errors which arise when one endeavors to compare catheterization studies made at two different times in an effort to determine the effect of a single alteration of the circulatory dynamics such as mitral valve commissurotomy. For example, if the patient's not in the same metabolic state each time, variations in the cardiac output may be expected; if there has been a change in cardiac rhythm cardiac output may also vary and of course, variations in output may in turn alter right heart pressures as well; if the heart rate fluctuates markedly, right heart pressures may also change; if heart failure was present before surgery and is absent thereafter, this too will cause dynamic alterations in both flow and pressures. Any one of these changes occurring postoperatively could mask the true effects of surgery. Hence every effort has been made to recognize these factors in our interpretation of results, and to avoid the potential errors whenever possible.

The operative procedure in those patients who came to surgery was carried out using in general the technic of Glover and Bailey. The majority of the cases were operated upon by members of the surgical team (R. H. W., A. H., A. L.). Through the courtesy of Doctors Lawrence Miscall and Jere Lord it was possible to study five additional patients.

RESULTS

The 60 patients referred to this clinic for surgery were considered to have met the clinical criteria by the referring physicians and the majority were willing and even anxious to undergo operation. Furthermore, it should be specifically noted that no one was accepted by the authors for evaluation unless enlargement of the left ventricle and intractable heart failure had been excluded and these types of rheumatic patients are not included in this presentation.

Patients rejected after clinical study alone. Fifteen patients were rejected as possible candidates for operation after further careful clinical observation only. Of these, 10 proved to have active rheumatic carditis and five subacute bacterial endocarditis as judged by the usual clinical criteria. In all 15, these conditions were previously unsuspected or unproven. One of the subjects with subacute bacterial endocarditis was cured and became a candidate after one year's time (case 700, tables 2, 3).

Patients rejected after physiologic study. Fifteen of the remaining 45 patients were rejected after physiologic study and the reasons for rejection will be considered below. Their diagnoses and data appear in table 1. Four of the 15 patients (group A, table 1) were not offered surgery because pulmonary artery pressures and cardiac outputs were normal at rest and pulmonary artery pressures were essentially unchanged during exercise. In two patients (cases 767 and 650) blood flow rose normally during exercise although in the third
Table 1.—Clinical and Physiologic Data in 15 Patients Rejected for Surgery who had Rheumatic Mitral Stenosis without Mitral Block

<table>
<thead>
<tr>
<th>Case, Sex, Age</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Cardiac Index (L/min./M² BSA.)</th>
<th>Pulmonary Artery s/d, m (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal at rest</td>
<td></td>
<td></td>
<td>3.12 ± 0.4</td>
<td>&lt;30/10, 15</td>
</tr>
</tbody>
</table>

(A) No Definite Physiologic Abnormality

<table>
<thead>
<tr>
<th>#</th>
<th>Case</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Cardiac Index (L/min./M² BSA.)</th>
<th>Pulmonary Artery s/d, m (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>767</td>
<td>LD F</td>
<td>34</td>
<td>MS. SA. Inc. AV block</td>
<td>Fatigue</td>
<td>(R) 2.66</td>
<td>13/7, 11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 3.77</td>
<td>13/8, 10</td>
</tr>
<tr>
<td>650</td>
<td>WG M</td>
<td>27</td>
<td>EH. MS. MI. NSR.</td>
<td>Hemoptysis</td>
<td>(R) 3.05</td>
<td>15/5, 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 4.36</td>
<td>18/8, 13</td>
</tr>
<tr>
<td>694</td>
<td>MF F</td>
<td>30</td>
<td>EH. MS. MI. AI. NSR.</td>
<td>Dyspnea, palpitations</td>
<td>(R) 2.93</td>
<td>21/10, 14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 3.28</td>
<td>25/12, 18</td>
</tr>
<tr>
<td>741</td>
<td>MK F</td>
<td>36</td>
<td>EH. MS. MI. NSR.</td>
<td>Fatigue</td>
<td>(R) —</td>
<td>24/9, 15</td>
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</table>

(B) Myocardial Insufficiency

<table>
<thead>
<tr>
<th>#</th>
<th>Case</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Cardiac Index (L/min./M² BSA.)</th>
<th>Pulmonary Artery s/d, m (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>695</td>
<td>SB F</td>
<td>44</td>
<td>EH. MS. NSR.</td>
<td>Bouts of CHF.</td>
<td>(R) 1.98</td>
<td>27/16, 20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 2.27</td>
<td>30/15, 21</td>
</tr>
<tr>
<td>675</td>
<td>JH M</td>
<td>41</td>
<td>EH. MS. AF.</td>
<td>Previous bout of CHF.</td>
<td>(R) 3.46</td>
<td>28/12, 21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 3.84</td>
<td>39/18, 27</td>
</tr>
<tr>
<td>591</td>
<td>NG M</td>
<td>32</td>
<td>EH. MS. AI. NSR.</td>
<td>Fatigue, dyspnea</td>
<td>(R) 2.90</td>
<td>23/9, 16</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 3.97</td>
<td>51/27, 39</td>
</tr>
<tr>
<td>761</td>
<td>MD F</td>
<td>42</td>
<td>EH. MS. AF.</td>
<td>Palpitations</td>
<td>(R) 2.08</td>
<td>30/17, 22</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 2.52</td>
<td>50/33, 41</td>
</tr>
<tr>
<td>552</td>
<td>JS M</td>
<td>52</td>
<td>EH. MS. AF.</td>
<td>Bouts of CHF.</td>
<td>(R) 2.33</td>
<td>29/13, 19</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(E) 3.20</td>
<td>49/25, 33</td>
</tr>
<tr>
<td>782</td>
<td>LL F</td>
<td>32</td>
<td>EH. MS. MI. NST.</td>
<td>Fatigue, dyspnea, one</td>
<td>(R) 3.23</td>
<td>18/11, 14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>bout of AF.</td>
<td>(E) 3.86</td>
<td>32/19, 25</td>
</tr>
<tr>
<td>641</td>
<td>DK F</td>
<td>44</td>
<td>EH. MS. MI. (T.I.) AF.</td>
<td>Dyspnea, palpitations</td>
<td>(R) 3.19</td>
<td>29/13, 19</td>
</tr>
</tbody>
</table>

(C) Myocardial Insufficiency With Frank Left Ventricular Failure

<table>
<thead>
<tr>
<th>#</th>
<th>Case</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Cardiac Index (L/min./M² BSA.)</th>
<th>Pulmonary Artery s/d, m (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>555</td>
<td>EB F</td>
<td>29</td>
<td>EH. MS. (T.I.) AF. Healed SBE.</td>
<td>Bouts of CHF, dyspnea</td>
<td>(R) 2.15</td>
<td>40/16, 25</td>
</tr>
<tr>
<td>612</td>
<td>CE M</td>
<td>20</td>
<td>EH. MS. MI. (G-S) NST.</td>
<td>Dyspnea, CHF.</td>
<td>(R) 2.39</td>
<td>79/42, 57</td>
</tr>
</tbody>
</table>

(D) Mild Pulmonary Hypertension Of Uncertain Etiology

<table>
<thead>
<tr>
<th>#</th>
<th>Case</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Cardiac Index (L/min./M² BSA.)</th>
<th>Pulmonary Artery s/d, m (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>715</td>
<td>FT M</td>
<td>29</td>
<td>MS. SA.</td>
<td>Hemoptyysis</td>
<td>(R) —</td>
<td>32/18, 24</td>
</tr>
<tr>
<td>620</td>
<td>RF M</td>
<td>43</td>
<td>EH. MS. SA.</td>
<td>Dyspnea</td>
<td>(R) 3.72</td>
<td>35/19, 27</td>
</tr>
</tbody>
</table>

* Complete hemodynamic data previously published.12
† Complete hemodynamic data previously published.13
For additional abbreviations see end of table 3.
**Table 2. Summary of Clinical, Hemodynamic and Pathologic Findings in 27 Patients Selected for Mitral Commissurotomy**

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>#595. AB. M. 28 yrs.</td>
<td>EH. MS. AI. NSR.</td>
<td>† Mod. pul. hyp. and low CO at rest.</td>
<td>Excellent; now 4 yrs. post-op.</td>
<td>Fall of PA pressures to normal but no change in CO at 1 year.</td>
<td>Conical. Leaflets supple. No thickening.</td>
<td>Aschoff bodies.</td>
</tr>
<tr>
<td>#618. HI. F. 41 yrs.</td>
<td>EH. MS. (T.I.) AF.</td>
<td>† Severe pul. hyp. and low CO at rest.</td>
<td>Excellent; now 3½ yrs. post-op.</td>
<td>Fall in pul. hyp. and no change in CO at 1 month.</td>
<td>Conical. Leaflets supple but diffusely thickened.</td>
<td>Organized thrombus in appendage.</td>
</tr>
<tr>
<td>#703. MA. F. 53 yrs.</td>
<td>EH. MS. (G-S) NSR.</td>
<td>† Severe pul. hyp. and low CO at rest.</td>
<td>Excellent; now 2 yrs. post-op.</td>
<td>Fall in pul. hyp. at 6 mos.</td>
<td>Conical. Heavily fibrotic at orifice but leaflets supple elsewhere.</td>
<td>No Aschoff bodies.</td>
</tr>
<tr>
<td>#707. CC. M. 36 yrs.</td>
<td>EH. MS. (G-S) SA.</td>
<td>† Severe pul. hyp. and low CO at rest.</td>
<td>Excellent; now 20 mos. post-op.</td>
<td>Marked fall in pul. hyp. but no change in CO at 2 months.</td>
<td>Conical. Granular calcif. at slit-like orifice but leaflets supple and thin elsewhere.</td>
<td>Aschoff bodies.</td>
</tr>
<tr>
<td>#714. RD. M. 29 yrs.</td>
<td>EH. MS. SA.</td>
<td>† Severe pul. hyp. and low CO at rest.</td>
<td>Excellent; now 19 mos. post-op.</td>
<td>Fall in pul. hyp. but no change in CO at 6 weeks.</td>
<td>Marked fall in pul. hyp. and rise in CO at 1 year.</td>
<td>Flattened cone. Calc., eliptical orifice with eburnated edges. Leaflets thick. SI. restricted motion of ant. leaflet.</td>
</tr>
<tr>
<td>#713. EW. F. 28 yrs.</td>
<td>EH. MS. (G-S) NSR.</td>
<td>† Severe pul. hyp. and low CO at rest.</td>
<td>Excellent; now 16 mos. post-op.</td>
<td>Marked fall in pul. hyp. and rise in CO at 1 year.</td>
<td>Conical. Leaflets supple, thin and mobile.</td>
<td>No Aschoff bodies.</td>
</tr>
<tr>
<td>#633. GS. M. 28 yrs.</td>
<td>EH. MS. I.AV.B. NST.</td>
<td>† Severe pul. hyp. and normal CO at rest.</td>
<td>Good; now 3½ yrs. post-op.</td>
<td>Marked fall in pul. hyp. and CO at 9 months.</td>
<td>Conical. Calc. at orifice but leaflets supple elsewhere.</td>
<td>Suggestive of auriculitis</td>
</tr>
<tr>
<td>#663. LS. F. 37 yrs.</td>
<td>EH. MS. NSR.</td>
<td>† Mod. pul. hyp. and normal CO at rest.</td>
<td>Good; now 2½ yrs. post-op.</td>
<td>Fall in pul. hyp. and no change in CO at 1 year.</td>
<td>Endocardial thickening.</td>
<td></td>
</tr>
<tr>
<td>#722. IK. F. 32 yrs.</td>
<td>EH. MS. NSR.</td>
<td>† Mod. pul. hyp. and normal CO at rest.</td>
<td>Good; now 16 mos. post-op.</td>
<td>Slight fall in resting pul. hyp. at 14 months.</td>
<td>Conical. Leaflets supple with slight ealeif. and thickening at orifice.</td>
<td>Aschoff bodies.</td>
</tr>
<tr>
<td>#</td>
<td>Name</td>
<td>Age</td>
<td>Diagnosis</td>
<td>Clinical Findings</td>
<td>Pathology</td>
<td></td>
</tr>
<tr>
<td>-----</td>
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<td>----------------------------------</td>
<td>-----------------------------------------------------------------------------------</td>
<td>------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>#792</td>
<td>MH. F.</td>
<td>41 y</td>
<td>Severe pul. hyp. and low CO at rest.</td>
<td>Good; now 6 mos. post-op.</td>
<td>Conical. Leaflets pliable</td>
<td></td>
</tr>
<tr>
<td>#664</td>
<td>JZ. M.</td>
<td>26 y</td>
<td>Severe pul. hyp. and normal CO at rest.</td>
<td>No change at 7 mos. after second commissurotomy but still unimproved.</td>
<td>Conical. Calcif. Small regurg. jet post-op.</td>
<td></td>
</tr>
<tr>
<td>#699</td>
<td>FB. M.</td>
<td>27 y</td>
<td>Mild pul. hyp. and normal CO at rest.</td>
<td>No change; now 2 yrs. post-op.</td>
<td>No Aschoff bodies.</td>
<td></td>
</tr>
<tr>
<td>#635</td>
<td>GB. M.</td>
<td>39 y</td>
<td>Mild pul. hyp. and low CO at rest.</td>
<td>No change; now 3½ yrs. post-op.</td>
<td>Conical. Granular calcif. at orifice but leaflets supple elsewhere.</td>
<td></td>
</tr>
<tr>
<td>#627</td>
<td>EB. F.</td>
<td>30 y</td>
<td>Severe pul. hyp. and low CO at rest.</td>
<td>Died suddenly 3 yrs. post-op.</td>
<td>Conical. Leaflets supple, diffusely thickened.</td>
<td></td>
</tr>
<tr>
<td>#606</td>
<td>PT. M.</td>
<td>49 y</td>
<td>Mod. pul. hyp. and low CO at rest.</td>
<td>Died 3½ yrs. post-op.</td>
<td>Discoid. Fibrous ring at orifice and leaflets diffusely thickened.</td>
<td></td>
</tr>
<tr>
<td>#723</td>
<td>FH. M.</td>
<td>37 y</td>
<td>Severe pul. hyp. and very low CO at rest.</td>
<td>Fall in pul. hyp. and rise in CO at 1 month.</td>
<td>Obviously insufficient with regurg. jet. Calcif. at edges. No valve closure in systole.</td>
<td></td>
</tr>
<tr>
<td>#662</td>
<td>NW. F.</td>
<td>26 y</td>
<td>Mod. pul. hyp. and low CO at rest.</td>
<td>Operative death.</td>
<td>No Aschoff bodies. (No evidence of activity at necropsy.)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Aschoff bodies. (Aschoff bodies in left ventricular myocardium at necropsy.)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Aschoff bodies. (Active aeurulitis and myocarditis at necropsy.)</td>
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</tr>
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</table>

FERRER ET AL.
### Table 2—Cont.

<table>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$700$. AC. M.</td>
<td>42 yrs.</td>
<td>EH. MS.* (G-S) RBBB. NSR. Healed SBE.</td>
<td>§ Severe pul. hyp. and low CO at rest.</td>
<td>Operative death.</td>
<td>No study.</td>
<td>Discoid. Extensive calcif. especially in ant. leaflet.</td>
<td>Aschoff bodies. (Active auriculitis and myocarditis at necropsy but no evidence of SBE.)</td>
</tr>
</tbody>
</table>

* Confirmed by necropsy.
† Complete hemodynamic data can be found in table 1 of the accompanying report.‡ Complete hemodynamic data can be found in table 2 of the accompanying report.§ Complete hemodynamic data can be found in table 3 of this paper.

PA = pulmonary artery.
Mod. = moderate.
Pul. hyp. = pulmonary hypertension.
CO = cardiac output.
Calcif. = calcification
Calc. = calcified
For additional abbreviations see end of table 3.
(case 694) the cardiac output response was probably somewhat less than normal.

The cardiac nature of their disability, as expressed by these four patients, was never completely convincing clinically. From the hemodynamic data one can conclude that two patients (cases 767 and 650) had normal circulatory function as measured. In the remaining two, the presence of normal pulmonary artery pressures probably eliminates significant mitral block.12 Once their circulatory status had been clarified, it became easier to define the true nature of their complaints. Observations over a one to three year period have shown that psychiatric illness (case 767), respiratory infections (case 650), neurosis (case 694) and alcoholism (case 741) were the bases, respectively, of their symptoms.

In contrast to the previous group, the seven patients in group B, table 1, had unequivocal and objective clinical evidences of cardiac disability. As indicated in this table, five of the seven have been discussed extensively in a previous report12 and their disability ascribed primarily to myocardial insufficiency rather than to mitral block since their dysfunction was associated with little or no resting pulmonary hypertension. The last two subjects (cases 782 and 641) were in no way different from the other five in this group. It is of interest that one of these (case 782) had had the same pulmonary artery pressures at rest 19 months before, indicating no recent change in hemodynamic status.

Although both patients in group C, table 1, had pulmonary hypertension at rest, these individuals were not offered surgical treatment because acute Digoxin studies, details of which have been previously published,14 uncovered a significant degree of left ventricular myocardial failure contributing to the elevated pulmonary artery pressures. It is evident that patients with mitral stenosis in whom left ventricular failure has been shown to account, at least in part, for the pulmonary hypertension cannot be considered for mitral surgery as there is as yet no positive way of excluding intractable left ventricular failure as being the sole or predominant cause of their disability.

In the last two subjects of the 15 rejected for surgery (group D, table 1) unequivocal objective evidences of disability were lacking and it was difficult to learn if there existed any diminished cardiac reserve in these two men in whom alcoholism was a major problem. Physiologic studies uncovered a mild degree of resting pulmonary hypertension in both, which could be the resultant of either myocardial insufficiency and some degree of left ventricular failure or mitral block. Since one could not be certain of their true status, and since their symptoms were not disabling, it was decided not to offer them surgery. The decision to withhold surgery in these two patients with minimal hemodynamic abnormalities may not be correct. Only careful complete serial physiologic studies can provide the answer to this question.

In summary, 11 of the 15 patients who were rejected for commissurotomy had little or no resting pulmonary hypertension and therefore, in the opinion of the authors, no evidence of significant obstruction at the mitral orifice. In two subjects, left ventricular failure accounted for the pulmonary hypertension. The remaining two subjects with mild hypertension and equivocal manifestations of disability represent a phase of the problem which as yet has not lent itself to definitive analysis.

Patients selected for mitral commissurotomy.

Thirty-one patients, including the patient with cured endocarditis who was mentioned above, were deemed suitable for operation after catheterization studies. Three of these refused surgery and another was ultimately rejected because he had a huge aneurysmal left atrium, which in the presence of atrial fibrillation was almost certain to be very thin walled and to contain large thrombi. Surgery was not advised in his case, because the mechanical performance of the commissurotomy would be unusually hazardous. None of these last four will be considered further. The diagnoses and data of the 27 subjects who were operated upon will be found in table 2. The diagnoses in all these patients were made according to criteria previously discussed.15

A few general comments are necessary before presenting the results obtained in the 27 subjects who had mitral surgery (table 2).
### Table 3.—Physiologic Data in 17 Patients with Rheumatic Mitral Stenosis Selected for Mitral Commissurotomy

<table>
<thead>
<tr>
<th>Case</th>
<th>Diagnosis</th>
<th>Time</th>
<th>Card. Output (L/min.)</th>
<th>Card. Index (L/min./M² BSA)</th>
<th>Heart Rate (beats/min.)</th>
<th>O₂ Consump. (cc/min./M² BSA)</th>
<th>R.Q.</th>
<th>A.V. diff. cont. (vol. %)</th>
<th>Arter. blood O₂ cont. (vol. %)</th>
<th>sat. (%)</th>
<th>Pressures in mm. Hg</th>
<th>TBV (cc/M² BSA)</th>
<th>PV (cc/M² BSA)</th>
<th>H'crit. (%)</th>
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<tr>
<td>Normal</td>
<td></td>
<td></td>
<td>3.12</td>
<td>72</td>
<td>131</td>
<td>0.86</td>
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<td>96</td>
<td>120/70, 90</td>
<td>30/10, 15</td>
<td>5</td>
<td>2750</td>
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<td>45</td>
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<td>#659</td>
<td>EH, MS. (G-S)</td>
<td>Pre-op.</td>
<td>3.22</td>
<td>2.04</td>
<td>97</td>
<td>120</td>
<td>0.86</td>
<td>6.3</td>
<td>16.0</td>
<td>93</td>
<td>119/57, 78</td>
<td>115/44, 70</td>
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<td>#633</td>
<td>EH, MS.</td>
<td>Pre-op.</td>
<td>5.69</td>
<td>3.23</td>
<td>102</td>
<td>150</td>
<td>0.76</td>
<td>4.9</td>
<td>15.1</td>
<td>82</td>
<td>92/58, 70</td>
<td>70/38, 55</td>
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<td></td>
<td>I.A.V. B.</td>
<td>(E)</td>
<td>5.51</td>
<td>3.13</td>
<td>110</td>
<td>197</td>
<td>0.80</td>
<td>6.3</td>
<td>15.0</td>
<td>84</td>
<td>106/66, 79</td>
<td>91/45, 64</td>
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<td></td>
<td>NSR. IIIC.</td>
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<td></td>
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<tr>
<td></td>
<td>(T.I.) AF.</td>
<td>Post-op.</td>
<td>3.78</td>
<td>2.14</td>
<td>71</td>
<td>143</td>
<td>0.75</td>
<td>6.7</td>
<td>19.7</td>
<td>98</td>
<td>108/68, 83</td>
<td>38/18, 25</td>
<td>4</td>
<td>—</td>
</tr>
<tr>
<td>#744</td>
<td>EH, MS.</td>
<td>Pre-op.</td>
<td>3.22</td>
<td>2.08</td>
<td>59</td>
<td>116</td>
<td>0.85</td>
<td>5.6</td>
<td>17.7</td>
<td>94</td>
<td>123/65, 88</td>
<td>37/15, 25</td>
<td>2</td>
<td>2610</td>
</tr>
<tr>
<td></td>
<td>F. 29 yrs.</td>
<td>1st Study</td>
<td>(R)</td>
<td>—</td>
<td>—</td>
<td>85</td>
<td>—</td>
<td>—</td>
<td></td>
<td>—</td>
<td>109/50, 79</td>
<td>—</td>
<td>8</td>
<td>—</td>
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<tr>
<td></td>
<td>NSR. IIIC.</td>
<td>10 days later</td>
<td>(R)</td>
<td>—</td>
<td>—</td>
<td>93</td>
<td>—</td>
<td>—</td>
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<td>—</td>
<td>126/59, 90</td>
<td>—</td>
<td>8</td>
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<tr>
<td>#722</td>
<td>EH, MS.</td>
<td>Pre-op.</td>
<td>3.22</td>
<td>1.76</td>
<td>88</td>
<td>113</td>
<td>0.84</td>
<td>6.6</td>
<td>17.2</td>
<td>94</td>
<td>115/71, 87</td>
<td>70/39, 54</td>
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<tr>
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<td>F. 32 yrs.</td>
<td>5 weeks</td>
<td>(R)</td>
<td>—</td>
<td>—</td>
<td>55</td>
<td>—</td>
<td>—</td>
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<td>127/63, 88</td>
<td>34/13, 24</td>
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<td>51/19, 35</td>
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<td>14 months</td>
<td>(R)</td>
<td>6.96</td>
<td>4.32</td>
<td>62</td>
<td>134</td>
<td>0.73</td>
<td>3.1</td>
<td>15.6</td>
<td>123/63, 88</td>
<td>31/10, 20</td>
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<td>2850</td>
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<tr>
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<td>(E)</td>
<td>7.52</td>
<td>4.67</td>
<td>90</td>
<td>191</td>
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<td>4.1</td>
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<td>146/80, 107</td>
<td>61/25, 44</td>
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<td>#792</td>
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<td>Pre-op.</td>
<td>2.38</td>
<td>1.76</td>
<td>88</td>
<td>113</td>
<td>0.84</td>
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<td>F. 41 yrs.</td>
<td>6 months</td>
<td>(R)</td>
<td>3.06</td>
<td>2.14</td>
<td>64</td>
<td>105</td>
<td>0.79</td>
<td>4.9</td>
<td>17.2</td>
<td>122/73, 91</td>
<td>37/20, 26</td>
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<tr>
<td>#664</td>
<td>EH, MS.</td>
<td>Pre-op.</td>
<td>5.86</td>
<td>3.10</td>
<td>96</td>
<td>161</td>
<td>0.77</td>
<td>5.2</td>
<td>17.8</td>
<td>91</td>
<td>112/73, 87</td>
<td>66/32, 43</td>
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<tr>
<td>(T.I.) AF.</td>
<td>Post-op. 7 months (R)</td>
<td>Pre-op. (R)</td>
<td>Post-op. 2 months (R)</td>
<td>15 months (R)</td>
<td>Post-op. 1 month (R)</td>
<td>1st Study 4 months later (R)</td>
<td>Pre-op. 1st Study (R)</td>
<td>Post-op. 1 month (R)</td>
<td>1st Study 2 weeks later (R)</td>
<td>Pre-op. (R)</td>
<td>Pre-op. (R)</td>
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<td>EE, MS. (G-S)</td>
<td>4.39 2.34 75 145 0.82 6.2 17.9 93 113/66, 83 62/31, 43 4 2840 1524 46</td>
<td>4.51 2.58 64 126 0.92 4.9 16.9 99 134/68, 94 60/23, 38 8 3048 1783 42</td>
<td>3.92 2.28 60 116 0.90 5.1 14.4 97 131/70, 88 47/19, 31 14 3290 2057 38</td>
<td>3.29 1.86 60 102 0.76 5.5 15.2 98 114/65, 84 55/18, 34 11 2952 1783 40</td>
<td>4.76 2.92 90 218 0.80 5.0 17.4 93 83/52, 63 38/17, 24 2 3650 1933 47</td>
<td>4.10 2.53 60 129 0.79 5.1 15.5 95 105/60, 76 39/16, 24 -- 3197 1884 42</td>
<td>3.56 3.43 80 240 0.82 7.0 15.5 92 117/69, 84 52/34, 53 -- -- -- --</td>
<td>4.14 2.62 57 134 0.76 5.1 15.6 99 133/69, 90 36/16, 26 -- 3200 1700 48</td>
<td>3.94 3.09 83 269 0.80 8.7 18.2 93 111/69, 85 76/33, 53 -- -- -- --</td>
<td>3.82 2.59 82 161 0.80 6.2 16.9 98 110/65, 81 42/21, 29 5 2920 1690 42</td>
<td>3.74 1.36 92 168 0.76 12.3 20.4 94 134/96, 112 96/53, 75 23 4473 2226 55</td>
<td>3.67 1.98 84 149 0.81 9.6 20.1 94 127/84, 103 85/43, 59 9 4380 2053 53</td>
<td>3.65 1.85 80 143 0.84 7.7 16.8 97 138/90, 105 70/35, 48 8 4120 2374 42</td>
<td>2.58 1.56 106 142 0.86 9.1 18.8 91 130/83, 99 87/55, 67 15 3171 1583 50</td>
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Table 3.—Cont.

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<th>Case Sex, Age</th>
<th>Diagnosis</th>
<th>Time</th>
<th>Card. Output (L/min.)</th>
<th>Card. Index (L/min./M² BSA)</th>
<th>Heart Rate (beats/min.)</th>
<th>O₂ Consumption (cc/min./M² BSA)</th>
<th>R.Q.</th>
<th>A.V. diff. cont. (vol. %)</th>
<th>Arter. blood O₂ cont. (vol. %)</th>
<th>sat. (%)</th>
<th>Pressures in mm. Hg</th>
<th>System Art. s/d, m</th>
<th>Pulmon. Art. s/d, m</th>
<th>Rt. Vent. d</th>
<th>TBV (cc/M² BSA)</th>
<th>PV (cc/M² BSA)</th>
<th>H'crit. (%)</th>
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<tr>
<td>#622. JB. M. 36 yrs.</td>
<td>EH. MS.* (T.I.) (G-S) AF. IIC.</td>
<td>Pre-op.</td>
<td>(R) 3.16</td>
<td>1.96</td>
<td>88</td>
<td>149</td>
<td>0.77</td>
<td>7.6</td>
<td>19.1</td>
<td>90</td>
<td>135/85, 102</td>
<td>152/91, 110</td>
<td>145/66, 97</td>
<td>7</td>
<td>3406</td>
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<td>51</td>
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<td>#609. JB. M. 34 yrs.</td>
<td>EH. MS.* NSR. IIID</td>
<td>Pre-op.</td>
<td>(R) 2.88</td>
<td>1.83</td>
<td>100</td>
<td>135</td>
<td>0.86</td>
<td>7.4</td>
<td>15.8</td>
<td>94</td>
<td>95/60, 80</td>
<td>105/72, 84</td>
<td>151/83, 108</td>
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<td>—</td>
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<td>#700. AC. M. 42 yrs.</td>
<td>EH. MS.* (G-S) RBBB, NSR. IIIC. Healed SBE.</td>
<td>Pre-op.</td>
<td>(R) 2.70</td>
<td>1.62</td>
<td>79</td>
<td>143</td>
<td>0.86</td>
<td>8.8</td>
<td>19.0</td>
<td>94</td>
<td>127/79, 96</td>
<td>125/46, 76</td>
<td>5</td>
<td>4632</td>
<td>2250</td>
<td>51</td>
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</tbody>
</table>

* = confirmed by necropsy  
M² BSA = per square meter of body surface area  
R.Q. = respiratory quotient  
AV, diff. = arterio-venous oxygen difference  
TBV = total blood volume  
PV = plasma volume  
H'crit. = hematocrit  
s = systolic  
d = diastolic  
m = mean  
SA = sinus arrhythmia  
AF = atrial fibrillation  
APC'S = atrial premature contractions  
VPC'S = ventricular premature contractions  
I.A.V.B. = incomplete AV block (prolonged PR interval)  
RBBB = Right bundle branch block  
CHF = congestive heart failure  
Healed SBE = healed sub-acute bacterial endocarditis
In addition to the disabling symptoms, all patients accepted for surgery had evidence of hemodynamic abnormalities. In the postoperative period every effort was made to restudy the patient hemodynamically in his best possible state, inasmuch as this had been our aim preoperatively. All medications were continued in the postoperative period, although in some instances it was necessary to reduce the dosage of digitalis. Mercurial diuretics were no longer necessary after surgery in the patients with good or excellent results. It should be noted that the surgeons in our group have hesitated to offer precise estimates of orifice size, but in general have felt that the mitral orifice would not admit the tip of the index finger in all the patients with only mitral stenosis and in every one of these, valve fracture was done. In two (cases 606 and 538) of the five patients with mitral stenosis and mitral insufficiency the orifice would just admit the fingertip and these also had commissurotomies, while in the remaining three subjects (cases 649, 723 and 751) the orifices were sufficient patent to permit easy movement of the exploring finger and no further widening was attempted. No physiologic estimation of the area of the mitral orifice was made, inasmuch as the method at present used for this determination is probably of limited value.\textsuperscript{15}

A summary of the results of commissurotomy obtained in the 27 patients subjected to surgery appears in table 2. The conclusions regarding the preoperative status and postoperative physiologic results were derived from a consideration of the hemodynamic studies carried out in these patients, details of which appear, as indicated, either in table 1 and 2 of a previous report\textsuperscript{12} or in table 3 of this paper. Criteria for evaluation of such hemodynamic changes have been previously discussed.\textsuperscript{12} The final clinical impression regarding each patient is listed as the “postoperative clinical result” in table 2 and the terminology used will be clarified and discussed subsequently. A description of the character of the mitral valve found on exploration was supplied by the surgeon and the biopsies and necropsies were evaluated by the pathologist (M. K.)

The first eight subjects in table 2 were considered to have had an excellent postoperative clinical result. This term “excellent” is employed to indicate that after surgery these patients have become asymptomatic at rest, are almost free of symptoms at whatever levels of activity they undertake, and do not consciously limit their efforts. Only one of the eight is not fully employed and this is for psychiatric reasons. These subjects are receiving the same or less cardiac medication than preoperatively and there have been no signs of peripheral or pulmonary congestion postoperatively.

One of the purposes of this study was an examination of the correlation between the postoperative clinical results and the physiologic results and this has proved to be of great interest. It is immediately apparent from table 2 that those patients who had an excellent postoperative clinical result all had moderate to severe resting pulmonary hypertension preoperatively. In these patients the systolic pressures ranged between 38 and 118, diastolics fell between 16 and 44, and mean pressures between 24 and 71 mm. Hg. The level of resting pulmonary hypertension, as well as the height of this pressure during exercise, decreased significantly after commissurotomy in the seven subjects in whom postoperative physiologic measurements were made. Alterations in the level of resting cardiac output were not as striking as those of pulmonary artery pressures, as four showed no change and only two (cases 567 and 713) a rise after surgery. The blood flow increase during exercise was measured in four patients pre- and postoperatively and in only two of them was the rise in blood flow after surgery satisfactory, that is commensurate with the level of exercise achieved. Despite the fact that these patients were considered to have had an excellent clinical result, only one (case 595) could be said to have achieved normal pulmonary artery pressures and even he continued to have a low cardiac output.

The term “good” has been utilized to describe the clinical results of the next five patients in table 2 who claim marked improvement and have no symptoms at rest. Never-
theless they guard against testing their capabilities to the fullest by consciously setting a ceiling to their efforts beyond which they will not go. Below this ceiling however they have no symptoms. In four of these no change in medication has been necessary while the fifth (case 792) no longer requires diuretics.

All five had moderate to severe pulmonary hypertension prior to surgery (tables 2 and 3) of a degree comparable to that found in the previous group and the range in cardiac output was also similar. In one of the five patients (case 744) two studies were made preoperatively, one before and one 10 days later after digitalization. There was no fall in pulmonary hypertension or in the elevated right ventricular end diastolic pressure. Indeed, the level of pulmonary pressure was higher on the second study when the heart rate was slightly faster. This suggests that these elevated pressures reflected mitral block, probably with complicating pulmonary vascular disease. In the four who had postoperative studies there was a fall in resting pulmonary artery pressures. However in two of them the hemodynamics must be discussed further before full interpretation is possible particularly with regard to cardiac output. One of them was a young musician (case 633) who had marked unsaturation of the arterial blood at the time of the preoperative study (table 3) in addition to the other findings, suggesting transitory subclinical pulmonary edema or the temporary opening of intrapulmonary vascular shunts. A normal arterial oxygen saturation was found on the following day. His postoperative course was stormy and atrial fibrillation appeared and is still present. Five months after surgery he began to improve and at six months he returned to work. Nine months after commissurotomy physiologic studies (table 3) revealed the decrease in pulmonary pressures referred to above, but also a lower cardiac output at rest (−34 per cent) as compared to the preoperative level. The arterial blood was fully saturated. A diastolic murmur was audible at the base of the heart and the mitral murmur was unchanged. It is now three and one quarter years since his operation and the patient is able to undertake any activity without symptoms, except sports which he has not attempted. It is difficult to evaluate the physiologic measurements in this man, since first, there was a fall in cardiac output probably associated with his change of cardiac rhythm to atrial fibrillation. Until the exact relationship between a decrease in pulmonary blood flow and pulmonary pressures can be clarified, his decrease in pulmonary hypertension cannot be ascribed solely to relief of mitral block. Furthermore, if pulmonary edema was present at the time of the first study pulmonary hypertension may have been acutely increased, thus temporarily distorting the measurement as an index of the degree of mitral stenosis and/or pulmonary vascular disease and rendering a comparison of pre- and postoperative measurements extraordinarily difficult.

As can be noted in table 3, another subject (case 722) classed as a good result, had a cardiac output which was considerably higher after surgery but which also was well above normal. This woman was very difficult to study as she was nervous and somewhat unstable, and although the usual criteria for a steady state were met during the cardiac output determination, the high value for blood flow may have been due to anxiety; nonetheless in spite of this high value, the pulmonary artery pressures were not higher than preoperatively.

Thus the subjective expression of improvement was substantiated by the physiologic findings in two (cases 663 and 792) of the four subjects classed as good results. In the other two in whom postoperative studies were realized, physiologic evidences of improvement while highly suggestive were not unequivocal.

Three patients (cases 664, 699 and 635) had no change after surgery. By this term is meant that in none was there any relief of symptoms at rest or during effort, and that they continued to show the same signs of pulmonary or peripheral congestion and fatigue as before surgery.

Hemodynamic studies in these patients who were thought clinically to have had no change after operation, confirm the clinical impression of no benefit, since the pulmonary artery pressures did not alter and the resting cardiac
output fell in two of the three. In two of these men (cases 699 and 635), there was mild pulmonary hypertension at rest and, as has been discussed in a previous report,12 it is now felt that in them the predominant lesion was not a mechanical block, but rather was myocardial insufficiency. The third patient was subjected to mitral commissurotomy on two separate occasions. This man (case 664, tables 2 and 3), a machinist and former wrestler and boxer had symptoms even at rest. Severe pulmonary hypertension, a normal blood flow at rest and arterial blood oxygen saturation were found on cardiac catheterization (table 3). Measurements during exercise were not attempted. He had a difficult postoperative course and showed no improvement after surgery. Atrial fibrillation which began in the immediate postoperative period, persisted. No systolic murmur or left heart enlargement became apparent even though the surgeon felt a small regurgitant jet post commissurotomy. A study seven months after the first commissurotomy revealed no alteration in right heart pressures (table 3) and a fall in cardiac output (−25 per cent). Hence it was decided to re-explore the valve. A further opening of the commissures was accomplished at the second operation and still the patient has not shown clinical benefit. Physiologic studies after the second operation could not be carried out as the patient went into pulmonary edema before the catheter was inserted. In this instance the lack of clinical improvement at the seven months study was mirrored in the physiologic evaluation, a fact which strengthened the impression that no real change had occurred after the first commissurotomy. It is very likely that pulmonary vascular changes secondary to mitral stenosis may be a major factor in this man, and may be irreversible. The fall in cardiac output while it may follow the onset of atrial fibrillation, may on the other hand be the result of a decline in myocardial function as well.

In summary, the hemodynamic evaluation in the eight patients who had excellent postoperative results can be said to confirm fully the clinical impression of benefit from surgery. These individuals were in all likelihood suffering from predominantly a mechanical block at the mitral orifice, as has been discussed in a previous report.12 The hemodynamic findings of the group classed as good were subject to interpretation as far as cardiac output is concerned, but did confirm a good clinical result if the behavior of the pulmonary artery pressures is accepted as a reliable criterion. In the three individuals who had no clinical improvement following commissurotomy, there was physiologic confirmation of this impression.

The remaining 11 of the 27 patients selected for commissurotomy are dead. Six survived surgery for varying periods of time while five were considered to be operative deaths.

The six who survived surgery will be discussed in detail since operative intervention did nothing to improve their status. Furthermore, since it is doubtful now that any had mitral block as their predominant lesion it is important to seek out their major difficulties. The first of these patients who died was the only one who had mitral stenosis without mitral insufficiency (case 627). Chronic congestive failure was her major problem for three years despite digitalization and diuretics. Only the most rigorous medical regime including bedrest for one month relieved her of congestive signs. Nevertheless hemodynamic studies (fig. 1, tables 2 and 3) showed severe pulmonary artery and right ventricular hypertension at rest and during exercise. Studies made two months after surgery revealed some decrease in pulmonary hypertension but a lower cardiac output and a higher right ventricular diastolic pressure at rest than preoperatively. There were no changes in heart murmurs or heart size. The physiologic findings could hardly be interpreted as unequivocal improvement and indeed as soon as she returned to work as a full time waitress chronic congestive failure reappeared. The patient claimed continuing improvement however. A study 15 months after surgery was not encouraging as right heart pressures were again about the same as preoperatively and her cardiac output had fallen considerably (−28 per cent). It was many months before she volunteered that there never had been any improvement. Nevertheless she had given
repeated objective proof on physiologic and clinical examinations that her status was steadily worsening. She died suddenly in congestive failure three years after commissurotomy. In view of the continuous fall in cardiac output without lasting fall in right heart pressures, it appears probable that in this woman myocardial damage was of primary concern. Under these circumstances the pulmonary hypertension would be largely the result of left myocardial failure.

In only two (cases 606 and 538) of the five patients with both mitral stenosis and insufficiency was the structure of the valve such as to warrant mitral commissurotomy, since in three (cases 649, 723 and 751) the valve was found to be so insufficient that no fracture was deemed advisable. In all a regurgitant jet could be felt at the time of operation.

Of the two patients in whom valve fracture was done, the first (case 606, fig. 2, tables 2 and 3) had had progressive dyspnea for three years. Studies showed moderate pulmonary hypertension and the surgeon found a deformed valve which he felt was stenotic enough to warrant commissurotomy and the anterior-lateral commissure was cut. Following surgery there was no indication of amelioration as judged from the hemodynamic data. Indeed the cardiac output fell. For 18 months the patient claimed improvement. This, however, he later reluctantly but spontaneously denied. Three and a quarter years after surgery, and after the appearance of atrial fibrillation, he died, following a series of pulmonary infarcts. Throughout his postoperative period there was nothing to suggest an increase in the degree of mitral insufficiency. Surgery obviously had not stayed the course of his disease or improved his hemodynamics, hence it seems likely that much of this patient's difficulty resulted from myocardial insufficiency, even though the valve was greatly deformed and heavily calcified. This conclusion is based on the physiologic observations of a level of pulmonary hypertension which was only moderate and a continuously falling cardiac output. Mitral insufficiency as the main defect appears unlikely since at necropsy the left ventricle showed no gross or microscopic

![Graphical representation of hemodynamic findings](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.40.3.20)
evidence of hypertrophy or dilatation. Furthermore, there had been no resealing of the mitral valve, as the orifice at necropsy was just as the surgeon had described it after commissurotomy.

The second man (case 538, tables 2 and 3) had the same basic dysfunction as the patient just discussed but the evidences for myocardial inadequacy appear to have been somewhat more definite. A year of hemoptyses, dyspnea and congestive failure brought this 25 year old patient to the hospital. The response to intravenous Digoxin (fig. 3), characterized by a fall in pulmonary artery as well as right ventricular diastolic pressures and a rise in cardiac output, gave evidence of left ventricular failure. Since a second study 4 months later revealed the same level of right heart pressures as after acute digitalization, an element of mitral block could not be excluded, particularly in view of the abnormal exercise response. In the next 10 months this patient's clinical course was unsatisfactory as he continued to have bouts of pulmonary edema and his heart size, as judged by x-ray films, was increasing. For this reason, exploration of the mitral valve was advised although it was recognized that there was an element of myocardial insufficiency present. At operation a calcified valve was felt with an orifice approximately the same size as that of the preceding patient's (case 606). The regurgitant jet which could be felt did not diminish after incision of the antero-
lateral commissure. Biopsy of the left auricular appendage revealed evidence of rheumatic activity. The postoperative course was complicated by prolonged low grade fever and 2 bouts of atrial fibrillation. Ten months after operation his heart size was larger than preoperatively, and he was admitted in severe congestive failure and died after the second of two pulmonary infarctions. The final clinical impression was that commissurotomy had not stayed the steady downhill course of active rheumatic heart disease in this young man.

In the remaining three men with a double mitral murmur, the valve was found to be so insufficient that no commissurotomy was attempted. Of these, one (case 649, tables 2 and 3, fig. 4) a former porter, had progressive dyspnea and some intermittent edema for five years as well as occasional hemoptyses. Admitted and subsequently studied while in congestive failure, he had severe pulmonary and right ventricular hypertension with tricuspid insufficiency and a very low cardiac output. Blood volume was considerably increased. After a month of bedrest, diuretics and continuous digitalis he was much improved, the liver had decreased in size although it was still enlarged, and the lungs were clear. Physiologic studies were repeated (table 3, fig. 4) and although pulmonary artery pressures and blood volume were definitely decreased and cardiac output somewhat increased (+14 per cent), the striking change was a fall in right ventricular diastolic pressure, indicating considerable improvement in right ventricular function. In view of the persistence of pulmonary hypertension it was decided, after another four months of bedrest, to explore the mitral orifice in this man. An opening large enough to admit the index finger readily was found, a regurgitant jet was easily felt and the value edges, which were calcified, failed to close in systole. No attempt was made to fracture or cut the commissures. He had an uncomplicated postoperative course and after one month, a third set of determinations were secured. A further decrease in pulmonary hypertension and a higher cardiac output (+36 per cent), were found while the ventricular diastolic pressure and tricuspid insufficiency were unchanged. These alterations, which incidentally were as large as were found in some of the successful commissurotomies, can be ascribed only to the effects of prolonged medical regime with amelioration in the degree of right and left heart failure, since no change was induced in the mitral valve action. Although nothing had been done surgically to alter his cardio-dynamics, he enthusiastically claimed relief for over two years and returned to work. He was found dead in bed at home two and one half years after operation.

The last two patients with mitral insufficiency and stenosis died in the immediate postoperative period. It appears likely that myocardial insufficiency played a predominant role in their difficulties and in one of these (case 723, tables 2 and 3) this was suspected from the preoperative studies. He was a 37 year old truck driver who had had progressive
dyspnea and orthopnea for two years. At the
time of his first study there was clinical evi-
dence of mild pulmonary and peripheral con-
gestion but had severe pulmonary hyper-
tension, a marked increase in right ventricular
diastolic pressure with a very low cardiac
output. He was digitalized, maintained on
strict bedrest and diuretics for the next two
weeks. This regime cleared his clinical evi-
dences of congestion. A second study before
surgery, showed a considerable drop in all
lesser circulation pressures and a 27 per cent
rise in cardiac output, all of these indicating
decrease in failure not only of the right but
also of the left ventricle. There was still, how-
ever, a severe degree of residual pulmonary
artery hypertension which could have been
the result of mitral block and hence it was
decided to explore this patient’s mitral valve.
No commissurotomy was performed as the
orifice was approximately 2 fingers in width.
He died several days after surgery during un-
controllable atrial fibrillation with a rapid
ventricular rate. Necropsy revealed stenosis and
insufficiency of the mitral valve without
any other valvular lesion. There was hyperto-
rophy and dilatation of the right ventricle and
mild dilatation and no hypertrophy of the
left ventricle on gross or microscopic examina-
tion. It would seem logical to conclude that
in this man myocardial failure was an all
important factor.

The second subject (case 751, tables 2 and
3) was a 32 year old man with six years of
dyspnea. After three months of hospitalization,
studies revealed only a moderate resting pul-
monary hypertension which became aggra-
vated on exercise, and a very low and relatively
fixed cardiac output. Hemodynamically he
resembled the two subjects (cases 606 and
538) previously discussed in whom the surgeon
attempted valve fracture. However at explora-
tion of the mitral valve in this patient the
orifice was large enough to admit over one
finger and no fracture was done. Since the
orifice was not greatly narrowed he too proba-
bly suffered, not from mechanical block, but
from inadequate myocardial function. He
died four weeks after surgery in congestive
failure. Necropsy confirmed the clinical im-
pression as to the valve lesions and further
showed that there was mild dilatation and
hypertrophy of the left ventricle which con-
tained Aschoff bodies.

Finally, five patients died on the operating
table or soon after leaving the operating room.
These deaths occurred early in our experience
with this surgical procedure. In three (cases
602, 607 and 622) the valve was funnel-
shaped and leathery, a finding the surgeon
has learned to dread as the leaflets cling to the
finger, obstructing blood flow more than usual
and resisting fracture. Four of them (cases
607, 622, 609 and 700) were the most severely
discharged of the entire group of 60 studied,
as their hemodynamics indicated (tables 2 and
3). Pulmonary hypertension was extreme and
cardiac output very low. The fifth (case
662) was less incapacitated, and although her
output was also reduced, pulmonary hyper-
tension was only moderate. Uncontrollable
hemorrhage from a rent in a very thin-walled
left atrium was the cause of death in one
(case 609). Cardiac arrest during finger frac-
ture and death within 24 hours due to cerebral
damage, occurred in two cases (cases 607 and
622). Cardiac arrest during anesthesia without
successful restoration of rhythm by cardiac
massage accounted for the death of a fourth
patient (case 700). Although the surgical
procedure was apparently without incident in
the fifth subject (case 622), he did not awaken
from anesthesia and died in shock in the
eighth postoperative hour.

A review of the auricular biopsy material as
given in table 2 indicates that there is little
correlation between the presence or absence of
Aschoff bodies in the appendage biopsy and
the final result after surgery, a point which
has been made previously in other reports.15, 17
Of the 10 patients who survived surgery and
had Aschoff bodies in the auricular appendage
six developed the postcommissurotomy syn-
drome. Two suffered from this syndrome who
did not have evidence of rheumatic activity as
judged from the appendage biopsy. The oc-
currence of this syndrome bore no relationship
to the ultimate surgical result.

Several interesting facts are apparent upon
scrutiny of the final clinical result of surgery
and the character of the mitral valve in each case. One should note here that the surgeon's description of the valve as regards shape and character was found to be remarkably accurate whenever postmortem findings were available for comparison. Indeed from such data we have learned that in general a discoid calcified valve, although it can be fractured, may not be associated with a good physiologic result. Furthermore this type of valve is very likely to be grossly insufficient. It should be stressed, however, that the mere presence of calcium in the valve is not important. It is heavy plaques which so distort anatomy and function, that surgical correction at present is not feasible. Diffuse speckling or calcification limited to the orifice on the other hand do permit a good result. The valve which is leathery and funnel-shaped may be impossible to fracture with the finger and unfortunately if the appendage base is narrow, a knife cannot always be introduced to cut the commissure. In general the best hemodynamic result is obtained with a conical shaped mitral valve where the leaflets are supple and calcium or fibrosis, if present, are limited to a ring at the orifice.

Discussion

Before presenting some of the many problems which this study has re-emphasized concerning the patient with rheumatic heart disease and mitral stenosis, a brief recapitulation of the results just presented is in order. Of the 60 subjects referred to this clinic as possible candidates for mitral surgery less than half (27 patients or 45 per cent) were selected for operation. Of these 27 only 13, or 50 per cent had a good or excellent result. In retrospect and as a result of this study we now believe only 19 instead of 27 should have had surgery as, in addition to the 13 good results, we still would select the five patients who were operative deaths since effective fracture had been done in such patients subsequently (cases 703, 659 and 744). Also we would still offer surgery to patient case 664 although he had two unsuccessful attempts at fracture, since, unfortunately even in retrospect, we cannot differentiate him clinically or physiologically from the group with good operative results.

As a result of information gained in this study we would now reject eight subjects who originally were advised to undergo surgery. Two of these (cases 699 and 635) could not definitely be rejected on clinical grounds but physiologic studies would label them as suffering from myocardial insufficiency. In a third (case 627) congestive heart failure required such a rigid medical regime, including constant bedrest for control, that myocardial insufficiency might have been suspected preoperatively. We have learned from the remaining five subjects (cases 606, 538, 649, 723 and 751) that one cannot expect a successful commissurotomy when the murmur of mitral insufficiency coexists with a heavily calcified valve, which calcium could be clearly seen by fluoroscopy in each of these patients preoperatively. In these five also, myocardial dysfunction appeared to predominate. It is well to point out with regard to this state of myocardial insufficiency (as described previously) that it may exist in the absence of enlargement of the left ventricle as judged by gross or microscopic examination as well as by clinical evaluation. Indeed this state may occur without any discernible evidence of pathologic abnormality in the left ventricle, at least as shown by present day methods. It may be that studies of myocardial metabolism will afford a solution to this problem.

Obviously this small group of patients followed through mitral surgery must be observed for a considerably longer time before any final conclusions as to the circulatory effects of commissurotomy are warranted. However, a certain amount of valuable information has been secured even now. It is rather discouraging, for example, to find that of the 13 good or excellent results, not one was entirely cured of his dysfunction by physiologic standards. It is true that one man (case 595) was entirely relieved of pulmonary hypertension at rest and during exercise, but his cardiac output did not change and was still below normal. Although the remaining patients did not attain normal resting pulmonary artery pressures they did have marked falls in pulmonary hypertension. These findings are in agreement with those of Ellis and his coworkers.
sidering the difficult task which the surgeon faced, namely restoration of valve function in a markedly diseased heart, it is not surprising that he did not always effect a cure.

It is quite interesting to examine the actual level of residual hypertension in those 13 patients who had a satisfactory surgical result. In 9 of the 11 in whom postoperative studies were made, the residual resting pressures in the pulmonary artery (see tables 2 and 3) were surprisingly consistent, averaging 32 mm. Hg systolic, 15 mm. Hg diastolic and 21 mm. Hg mean, with ranges of 23 to 38 systolic, 8 to 20 diastolic and 13 to 26 mean, respectively, although preoperatively the resting levels had varied markedly. It is tempting to speculate on the meaning of this finding of residual hypertension. Is this residual mitral block? If so, it is asymptomatic mitral block. If this pressure elevation is due to pulmonary vascular disease, then the latter must be minimal in 9 of the 11 patients studied postoperatively. It is likely that the remaining two had considerable vascular change as their residual hypertension was severe. There appears to be no way at present to differentiate between these two causes. Perhaps it is simplest to say that we have moved these individuals back a certain distance along the course of the natural history of rheumatic heart disease, but how far and for how long remains to be seen.

Other objective data obtained in the patients before and after surgery are less revealing than the catheterization findings. In only one of the 13 successful commissurotomies (case 713) has there been a decrease in total heart size (fig. 5) although the pulmonary artery segment grew smaller in a number of the subjects. A comparison of pre- and postoperative x-ray films (fig. 6) in one other subject (case 792) emphasizes how little can be the change in cardiac silhouette, with even the double atrial contour along the right border persisting unaltered despite a marked fall in pulmonary artery pressures after surgery. On the other hand, in none was there an increase in heart size once postoperative pericardial effusion had subsided. This latter occurred in 5 of these 13 and took as long as four weeks to recede. In no instance was there any permanent change in heart murmurs, although these became muffled during pericardial effusion. In only one patient (case 713) was there a reversal of the right ventricular

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**Fig. 5.** Roentgenograms (posteroanterior 6 foot films) of case 713, patient E. W. before and one year after commissurotomy. For discussion see text.
hypertrophy pattern in the electrocardiogram one year after surgery. Otherwise, the electrocardiographic changes noted preoperatively persisted postoperatively, once the alterations imposed by the acute pericarditis, which all patients developed, had disappeared.

It appears, therefore, from this study that objective physiologic criteria are the most satisfactory in evaluating success in commissurotomy and that one must rely on these rather than on the exclusive use of subjective impressions. This is in agreement with a recent report of Soloff and Zatuchni\(^1^9\) and could not be better demonstrated than in our own two subjects (cases 627 and 606) in whom the ultimate result was unsatisfactory and in one (case 649) who had no valve fracture, all of whom claimed subjective improvement.

This same expression of subjective improvement forms the basis for the majority of the clinical reports of large groups of patients undergoing surgery. From such reports one cannot escape the conclusion that the clinical criteria utilized have failed to pinpoint the problem and aside from a demonstration that the cardiac can survive this type of surgery, little that is fundamental has accrued to our knowledge of mitral stenosis and its susceptibility to surgery. Even those reports in which objective physiologic data are available, and these are very few in the present literature, have not yet provided us with the answer of how to select the mitral subject who will invariably benefit from commissurotomy. It is therefore obvious that much more study is needed in order to clarify some of the many problems relating to the patient with mitral stenosis.

Selection of patients for commissurotomy. In any consideration of mitral stenosis, and particularly when selecting candidates for surgery, it has become clear from this study that each patient must be evaluated within the framework of rheumatic heart disease itself. For this reason a simple working schema (fig. 7) has been constructed. The effects of the rheumatic process on the heart can be divided into valvular and myocardial. In patients with mitral stenosis however, there may be great variations in the degree of valve deformation and the mere auscultatory diagnosis does not imply a deranged circulation. Hence there exists a group of individuals with stenosis but without significant mitral block in whom

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**Fig. 6.** Roentgenograms of case 792, patient M. H. before and six months after commissurotomy. For discussion see text.
hemodynamic measurements indicate a normal circulation. It is difficult to justify operation on such patients.

Another group of rheumatic subjects who also had predominantly valvular damage are those with mitral stenosis and a significant degree of block. These individuals can go in and out of myocardial congestive failure due to mechanical strain and not myocarditis as shown by the arrows in the schema (fig. 7). These individuals obviously represent the ideal candidates for commissurotomy, particularly if they do have a relatively unimpaired myocardium.

Lastly there is a group of subjects with rheumatic mitral stenosis in whom it has been shown that myocardial damage is dominant and in whom there may be no significant block. The myocardial insufficiency of these hypodynamic hearts will not be benefitted by surgery and indeed such patients may succumb to the operative procedure.

Two of the three types of patients included in this schema, even though clinical evaluation may be difficult, can be differentiated by physiologic studies. The normal circulation of the one and the hypodynamic characteristics of the myocardial dysfunction of the other permit their clear-cut separation on physiologic grounds from the patient with predominantly mitral block.

It is obviously the patient with mitral stenosis and block who is the ideal candidate for surgery, and furthermore, if indeed there is true obstruction at the valve, the patient will have moderate to severe resting pulmonary hypertension. It would perhaps even be advisable to make the establishment of such pulmonary hypertension a prerequisite to acceptance for surgery. In its absence there is little likelihood of the existence of an important degree of mitral block amenable to, or one which will benefit from, surgery.

The selection of the patient might then appear superficially simple, i.e., if there are progressive symptoms, the murmur of mitral stenosis, no clinical contraindications and pulmonary hypertension he would seem to be a proper candidate. This assumes of course that the pulmonary hypertension is not due to left heart failure. As a final point, one is faced essentially with determining in any one patient with mitral stenosis whether there is pulmonary hypertension or not, once left heart failure, if present, is relieved. Unfortunately there is no good clinical means of doing this in all instances. Heart sounds or intensity of murmurs do not help, the second pulmonic sound particularly offers no reliable indication and heart size is very deceptive as can be seen in previous reports.\textsuperscript{14, 20} Our group is unable to make this determination with certainty unless the patient is catheterized and the pulmonary artery pressures are measured directly. Because the tendency at present is to infer rather than demonstrate pulmonary hypertension, its presence may be improperly diagnosed and hence patients are undergoing needless surgery. This is so because the clinical criteria are as yet grossly insufficient to enable one to choose the correct subjects for commissurotomy. It is true that the ideal candidate can be suspected if there is a small heart, electrocardiographic evidence of right ventricular hypertrophy in the precordial leads and no heart failure, and it is also true that one should beware of offering surgery as amelioration to the patient with a very large heart. These points, however, offer only indirect evidence and in the case of the hypertrophy pattern surely indicate an advanced stage of mitral block.

The fact that no real effort is now being
made in most clinics to establish the presence of pulmonary hypertension in the candidate for mitral surgery may also provide an explanation for some of the unsuccessful commissurotomies reported by some groups; the figures vary between 30 per cent and 40 per cent. In the authors' opinion, the lack of success probably lies in the fact that some patients' very real symptoms were attributed to mitral block when indeed they sprang from myocardial insufficiency. Physiologic studies might have indicated this difference in the etiology of the dysfunction had they been performed. Of course, inoperable valve deformity also is a contributing factor to failure of surgery.

It would therefore seem imperative to continue a combined clinical and physiologic study of mitral stenosis until such time as the reliable clinical signs of obstructive pulmonary hypertension can be ascertained and clarified, thus eliminating the need for cardiac catheterization. The need for such a study is even more apparent when one realizes that the hemodynamic picture of "early" mitral block, i.e. the circulatory state wherein critical narrowing of the orifice is just beginning and the valve is still relatively mobile, remains to be defined. It is probable that once this picture has been critically and physiologically defined, these patients with early mitral obstruction will be the best candidates for commissurotomy.

**Summary and Conclusions**

This paper presents a study of 60 patients who were referred to this clinic as suitable candidates for mitral commissurotomy. Of these 60 patients, 15 were deemed unsuitable after clinical study alone; 10 proved to have active rheumatic carditis and five subacute bacterial endocarditis. Another 15 were rejected as candidates for operation after physiologic study; 11 of these had little or no resting pulmonary artery hypertension, two gave evidence that left ventricular failure accounted for the pulmonary hypertension found, while two without disabling symptoms had only mild pulmonary hypertension and represent a phase of the problem which has not as yet been clearly defined. Thirty-one patients were considered suitable for surgery after physiologic studies; of these three eventually refused commissurotomy and one was rejected because of an aneurysmal left atrium.

Of the 27 subjects who underwent surgery, eight were believed to have had clinical and physiologic evidence of an excellent result while five were designated as good results. Nine patients showed no clinical or physiologic evidence of any improvement, and of these, six are now dead. The remaining five were operative deaths.

As a result of this study we would now reject eight subjects who were originally advised to undergo surgery, believing that their predominant difficulty was myocardial insufficiency and not valvular disease.

Our experience with the combined lesions of mitral stenosis and insufficiency is somewhat limited but is discouraging. Indeed we have learned from the five patients in this series with a double mitral murmur that one cannot expect a successful commissurotomy when the murmur of mitral insufficiency coexists with a heavily calcified valve.

Clinical criteria appear as yet inadequate for the selection of the proper candidate for mitral commissurotomy, as there is no good clinical means of establishing in every instance the presence of pulmonary hypertension, which remains the best evidence of a significant degree of mitral block.

Objective physiologic measurements represent the best criteria for the effects of surgery as subjective impressions may be unreliable.

**Summario in Interlingua**

Inter 60 patientes recommendate como candidatos promittente pro commissurotomea mitral, solmente 27—i.e. minus que un mediate—esseva acceptate super le base de examines plus exacte. Ex iste ultime gruppo, solmente 13—i.e. de nove minus que un mediate—monstrava bon resultatos postoperative, non solo secundo le evidentia clinic sed etiam secundo objective mesurationes physiologic. Tal mesurationes es le melior criterios in judicar le effectos operative proque impressiones subjective es frequentemente paucio digne de
confidentia. Le gruppo operate includeva 8 patientes que hodie esseera rejicte per nos proque lor difficuttate predominante eseva insufficientia myocardiae e non blocage mitral. Le nunc disponibile criterios clinic es inadequate pro le efficace selection de candidatos pro commissurotomia mitral. Il existe ancora nulle bon medio clinic pro establir in cata caso le presentia o absentia de hypertension pulmonar; e hypertension pulmonar remane le melior indice de un significative grado de blocage mitral.

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Circulatory Effects of Mitral Commissurotomy with Particular Reference to Selection of Patients for Surgery

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