Congenital Mitral Stenosis

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Congenital stenotic lesions of the mitral valve may be classified into various groups depending on the functional capacity of the left ventricle. In the extreme form, severe stenosis or atresia of the mitral valve is associated with hypoplasia of the left ventricle and aorta. Septal defects are usually present and these combinations are lethal in infancy.\(^1\),\(^2\) In the presence of a functioning left ventricle, the anomaly is compatible with survival, and the stenotic valvular lesion produces an increased pressure proximal to the mitral valve, resulting in left atrial enlargement, pulmonary venous and arterial hypertension, and ultimately congestive heart failure. Although congenital isolated mitral stenosis is rare, it is often associated with a poor prognosis. This report is concerned with a study of isolated congenital mitral stenosis. Mitral stenosis associated with other cardiac anomalies is discussed briefly.

Clinical and Pathologic Data

Seven instances of congenital mitral stenosis were studied (table 1). In six, the anomaly was isolated and in one there was an associated coarctation of the aorta. Five patients were female and two were male. These children were seen in early childhood, the cardiac murmur was noted in infancy, and there was no history of rheumatic fever. The birth weight was normal in all patients. The family history was not contributory. In one patient (no. 6) the mother had rubella during the third month of gestation.

Symptoms and Clinical Course

At some time during the clinical course, dyspnea, repeated pulmonary infections and congestive heart failure were present in all patients. Pulmonary edema was identified in four patients (no. 3, 4, 6, 7). Patients 1 and 3 were subject to repeated pulmonary atelectasis associated with bronchopneumonia. Marked pallor was a prominent finding in patient 4, and in patient 6 there were frequent episodes of syncope, marked pallor and cyanosis. Patients 3 and 4 were symptomatic early in infancy and died early in congestive heart failure. The clinical status in patient 1 was improved by digitalis and antibiotics during the first 2 years of life. Since that time he has been lost to follow-up. Patient 2 was treated for congestive heart failure at the age of 6 months and presently at 6 years of age is asymptomatic. The other three patients (no. 5, 6, 7) developed progressive heart failure after the age of 3 years and were operated on between the ages of 4½ to 5½ years. Two had mitral valvotomy during total body perfusion, developed postoperative tachycardia with respiratory distress and hypotension, and died. Patient 7 had simultaneous transventricular mitral valvotomy and resection of a coarcted aortic segment and end-to-end anastomosis. She developed mitral insufficiency and marked cardiomegaly and died in congestive heart failure 7 months postoperatively.

Physical Findings

The blood pressure was normal in all subjects except patient 7 who had associated coarctation of the aorta. In the latter patient the blood pressure was 150/80 mm. Hg in the upper extremities and 80/50 mm. Hg in the lower extremities. A right ventricular parasternal lift was present in all patients and in patient 3 there was an associated left ventricular heave. An apical rumbling diastolic murmur was present in all instances and was associated with a thrill in six patients. Pre-systolic accentuation of the murmur was noted in four patients. An apical systolic mur-
### Table 1

**Clinical Data on Seven Patients with Congenital Mitral Stenosis**

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex</th>
<th>Auscultatory findings</th>
<th>Electrocardiogram</th>
<th>X-ray</th>
<th>Catheter data</th>
<th>Clinical condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>2+ II II, pre</td>
<td>P, mitralle 1+ 0</td>
<td>2+</td>
<td>30/16 14</td>
<td>Repeated pulmonic infections and atelectasis, ? pulmonary edema. CHF compensated at age 2 yr. No information since</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>2+ I III, pre</td>
<td>Normal 0 0 2+</td>
<td>2+</td>
<td>30/10 6 96</td>
<td>Age 3 mo. double mitral murmurs with LVH; CHF at 6 mo. Steady improvement and now at 6 yr. asymptomatic</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>N III III, 0</td>
<td>P, mitralle 2+ 2+ 3+</td>
<td>3+ 3+</td>
<td>30/10 6 96</td>
<td>Progressive CHF from age 6 wk. Pulmonary edema. Repeated pulmonic infections and atelectasis. Died at 6 mo. Autopsy</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>N II II, 0</td>
<td>Peaked 3+ 2+ 1+</td>
<td>2+</td>
<td>30/10 6 96</td>
<td>Marked pallor since birth. Progressive CHF since 8 wk. Pulmonary edema. Died at 3 mo. Autopsy</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>2+ I III, +</td>
<td>Normal 2+ 0 1+</td>
<td>1+</td>
<td>5 35/15 10 100</td>
<td>Asymptomatic till 3½ yr., then repeated pulmonic infections. ? pulmonary edema, chronic CHF. Died after surgery at 5½ yr.</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>2+ I III, pre</td>
<td>P, mitralle 0 0 2+</td>
<td>2+</td>
<td>5 92/55 28 88</td>
<td>CHF at 1 yr. Marked deterioration since age 3 yr. Repeated pulmonic infections. Chronic CHF. Pulmonary edema. Died after surgery at 3½ yr.</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>N II III, pre</td>
<td>P, mitralle 1+ 0 1+</td>
<td>1+</td>
<td>3 45/18 94.5</td>
<td>CHF at 3 yr. Pulmonary edema. Recurrent pulmonic infections and chronic CHF. Associated coarctation of aorta. Died 7 mo. post-operatively</td>
</tr>
</tbody>
</table>

M, apical first sound; N, normal; SM, apical regurgitant systolic murmur; DM, rumbling apical diastolic murmur; pre, presystolic; OS, opening snap; RVII, right ventricular hypertrophy; LVII, left ventricular hypertrophy; LA, left atrium; RV, right ventricle; LV, left ventricle; PA, pulmonary artery; CHF, congestive heart failure. Pressures in millimeters of mercury.
murmur was present in all and was marked in patient 3. A loud first heart sound was noted in four patients; the second sound was normal with moderate accentuation of the pulmonic component. An opening snap was heard in patient 5. A basal aortic ejection systolic murmur was present in patient 7.

**Electrocardiogram**

Broad notched P waves suggesting left atrial enlargement were noted in four patients. A pattern of moderately severe right ventricular hypertrophy was present in three, and in two of these there was associated left ventricular hypertrophy (fig. 1). The other patients had no detectable evidence of ventricular hypertrophy.

**X-ray**

The heart size was mildly enlarged in patient 4 (fig. 2), moderately so in five patients (fig. 3), and was markedly enlarged in patient 3 (fig. 4). Right ventricular and left atrial enlargement were demonstrated in all instances, and there was prominence of the left ventricle in patient 3. The peripheral pulmonary arterial vasculature was not increased but radiologic evidence of pulmonary edema was present in five patients. In two patients (no. 1 and 3) respiratory distress was associated with pulmonary atelectasis of part of the right or left lung.

**Cardiac Catheterization**

Right-sided cardiac catheterization (table 1) demonstrated moderate to severe pulmo-
## Table 2

**Pathologic Data on Five-Children with Congenital Mitral Stenosis**

<table>
<thead>
<tr>
<th>Patient no., age</th>
<th>Heart weight (Gm.)</th>
<th>Normal weight for age (Gm.)</th>
<th>LA enlargement</th>
<th>LV wall thickness</th>
<th>RV wall thickness</th>
<th>Mitral valve orifice (cm.)</th>
<th>Chordae tendineae</th>
<th>Papillary muscles</th>
<th>EFE</th>
<th>Medial hypertrophy pulmonary arterioles</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 6 mo.</td>
<td>119</td>
<td>32</td>
<td>3+</td>
<td>0.9 cm. large cavity</td>
<td>0.6 cm.</td>
<td>0.8 thickened 3+ diaphragmatic</td>
<td>atrophic thickened</td>
<td>hypertrophic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 3 mo.</td>
<td>46</td>
<td>23</td>
<td>2+</td>
<td>1 cm. small cavity</td>
<td>1 cm.</td>
<td>0.3 thickened 3+ no commissures funnel shaped</td>
<td>shortened &amp; thickened</td>
<td>flattened &amp; thickened</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 5 yr.</td>
<td>98</td>
<td>85</td>
<td>1+</td>
<td>1 cm. normal cavity</td>
<td>0.7 cm.</td>
<td>0.5 thickened 3+ fused commissures</td>
<td>shortened &amp; thickened</td>
<td>short hypertrophic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 hypertrophied 2+</td>
<td>13 cm.</td>
<td>1+</td>
<td>0.8 cm.</td>
<td>1.3 cm. small cavity</td>
<td>0.5 at surgery thickened</td>
<td>thickened focal sclerosis</td>
<td>LA: 2+ LV: 1+</td>
<td>LV: ± focal</td>
<td></td>
<td>Mitral valve, near posterolateral commissure consisted of a muscular &amp; fibrous component. A portion of the muscle partially protruded into the LV outflow, causing some obstruction, but not true stenosis</td>
<td></td>
</tr>
<tr>
<td>7 4½ yr.</td>
<td>140</td>
<td>79</td>
<td>1+</td>
<td>0.9 cm. normal cavity</td>
<td>0.5 cm.</td>
<td>0.5 thickened 2+ commissures fused but defined</td>
<td>thickened hypertrophic</td>
<td>LA: 1+ LV: ±, focal</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

LA, left atrium; LV, left ventricle; RV, right ventricle; RA, right atrium; EFE, endocardial fibroelastosis.
Concurrent mitral stenosis

The pertinent autopsy findings of five children are summarized in Table 2. Three of these patients (no. 5, 6, 7) had undergone surgery. In all instances the heart was enlarged and tight mitral stenosis with thickening and deformity of the valve and its attachments was present. The valve was diaphragmatic and unicuspal in patient 3 (fig. 5) and funnel shaped with fused commissures in the others. The chordae tendineae were thickened, shortened, and atrophic to a variable degree. Generally the papillary muscles were hypertrophic (fig. 5). The valve and its attachments encroached on the cavity of the left ventricle in two instances (at the inflow in patient 3 and in patient 6 the posterolateral leaflet had a fibrous and muscular component that obstructed the ventricular outflow without creating a true stenosis). An undifferentiated fibrous band created a stenosis inside the left ventricle in patient 5 (fig. 6). The left ventricular cavity was normal in two patients (no. 5 and 7), small with a hypertrophic wall in patients 4 and 6, and hypertrophic and dilated in patient 3. Left atrial and right ventricular enlargement were present to a variable degree in all patients. The right atrium and pulmonary artery were dilated. Endocardial fibroelastosis involved mainly the left atrium and mitral valve (figs. 5 and 6). This lesion also involved the left ventricle but was severe in only one instance (patient 3), (fig. 5). There were mild thickening of the tricuspid valve and right ventricular endocardium in patient 5. The pulmonary arteri-

Discussion

Isolated congenital mitral stenosis is often a fatal disease, especially in early childhood. Recognition of this condition is important because it is a potentially operable anomaly. We have found reports in the literature of 30 patients with isolated congenital mitral stenosis. In this group the diagnosis was made clinically in eight, confirmed at autopsy in nine, and 13 other patients underwent mitral valvotomy. A further 12 cases were commented upon briefly in the discussion of Starkey's report.

In the presence of severe stenosis, the common symptoms are dyspnea, repeated pulmonary infections with or without atelectasis, congestive heart failure, pulmonary edema, and poor physical growth. Other symptoms are pallor, sweating, syncopeal episodes, and cyanosis during syncope or marked heart failure. Severe mitral stenosis is usually symptomatic in infancy, and frequently the outcome is fatal during the first 2 years of life. Early appearance of symptoms and

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congestive heart failure are usually associated with a poor prognosis. Of interest is the course of our patient 2 (table 1). In infancy she was in congestive heart failure, had murmurs of mitral insufficiency and stenosis, radiologic signs of left atrial enlargement, and electrocardiographic evidence of severe left ventricular hypertrophy (fig. 7). At that time the clinical picture suggested endocardial fibroelastosis. Presently at the age of 6 years she is asymptomatic, has the auscultatory findings of mitral stenosis (fig. 8), and a normal electrocardiogram (fig. 7). In this instance, it is believed that the mitral stenosis is mild.

The diagnosis is suspected in the presence of an enlarged heart, an apical diastolic rumbling murmur, signs of pulmonary hypertension, and left atrial enlargement. The apical diastolic murmur is a common finding and usually is the first clue to the diagnosis. It was present in all our patients. A presystolic accentuation of the murmur is common and favors a stenotic origin. This component may differentiate this diastolic murmur from that produced by a left-to-right shunt. In the latter group, especially those anomalies with a large flow across the mitral valve, the murmur is commonly mid-diastolic without a presystolic accentuation and is preceded by a third heart sound. It is well known that the diastolic murmur of mitral stenosis may be absent in the presence of pulmonary hypertension and congestive heart failure, and in the latter instance may reappear when cardiac compensation has been restored. These frequent complications may explain the absence of diastolic murmurs in some of the reported cases.\(^9,14,16,17\) Of interest is patient 4 in whom the apical diastolic rumble was first noted in the presence of congestive heart failure. When present, a sharp first sound is suggestive of mitral stenosis. A loud first sound, however, may be present in children without mitral stenosis. An opening snap of the mitral valve was heard in patient 5 and was reported in only a few others.\(^19\) A ringing first heart sound and an opening snap of the mitral valve strengthen the diagnosis of mitral stenosis. A systolic apical murmur is often present and is usually related to the frequently associated mitral insufficiency.

**Figure 5**

*Patient 3. A. Marked endocardial fibrosis of the left ventricle and atrium. Chordae tendineae atrophic. Arrow indicates hypertrophic papillary muscle. B. Markedly deformed and thickened unicusp mitral valve viewed from the left atrium.*
This murmur, the rarity of the opening snap, and the infrequent sharp first heart sound are probably due to the marked deformity of the valve in congenital mitral stenosis, which limits its mobility. A loud pulmonic component of the second sound indicates the presence of pulmonary hypertension, which frequently complicates this lesion.

The electrocardiogram and x-ray were helpful in demonstrating left atrial enlargement, cardiomegaly, and right ventricular hypertrophy. Associated left ventricular hypertrophy may be present. The absence of electrocardiographic signs of ventricular hypertrophy and a normal heart size radiologically are usually associated with a mild lesion. However, in some instances of severe stenosis, cardiomegaly was not significant (patient 4)\(^7\) (fig. 2), or the electrocardiogram did not show signs of ventricular hypertrophy (patient 6).\(^{16, 20}\) (fig. 9).

Moderate or occasionally marked\(^{11}\) left atrial enlargement is present and is of distinct value in diagnosis. It was demonstrated in all our patients by barium swallow. A broad notched P wave was not so constant.

The central pulmonary arteries were prominent in the presence of pulmonary hypertension, and the peripheral pulmonary vasculature was not increased. Pulmonary venous congestion was frequent.

The clinical diagnosis is confirmed by demonstrating signs of stenosis of the left atrial outflow by cardiac catheterization and angiocardiography. The former measures an elevated left atrial or pulmonary artery wedge pressure. In one of our patients (no. 5) with tight mitral stenosis, however, the wedge pressure was normal (table 1). The presence of pulmonary hypertension is confirmed by this study. Angiocardiography will demonstrate an enlarged left atrium with delayed emptying of contrast material.\(^{10, 13, 14, 16}\) This was identified in patient 6. However, this sign in our experience was present in one instance of severe endocardial fibroelastosis with a large left atrium without a mitral stenotic lesion at autopsy. This study also identifies the size of the left ventricle and aorta. It has been suggested that the degree of associated endo-

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**Figure 6**

Patient 5. Left. Chambers of left heart and mitral valve. Arrow indicates fibrous band binding chordae tendineae. Right. Thickened and stenotic mitral valve viewed from left atrium.
cardiac fibroelastosis can be evaluated by the nature of the pulsations of the left ventricle.  

**Differential Diagnosis**

The typical features of congenital cardiac anomalies with a left-to-right shunt are well recognized. Their differentiation from mitral stenosis may be difficult, however, especially in the presence of congestive heart failure, pulmonary hypertension, and an apical diastolic rumbling flow murmur. Because the symptoms of mitral stenosis can be related to the degree of pulmonary venous hypertension, the following congenital lesions may produce a similar pathophysiology: cor triatriatum, or stenosis of the common pulmonary vein, a stenosing ring of the left atrium, atrial myxoma and infradiaphragmatic total anomalous pulmonary venous return.

Endocardial fibroelastosis and congenital mitral insufficiency generally present features of marked left ventricular hypertrophy. Those instances associated with pulmonary hypertension may present difficulty in differential diagnosis.

Frequently congenital mitral stenosis is associated with other cardiac anomalies, particularly coarctation of the aorta, patent ductus arteriosus, and aortic stenosis. Ferencz et al. and others have described these combined lesions and pointed out the unreliability of physical signs and the difficulty of clinical diagnosis. In many instances, however, the anomalies can be recognized clinically as in our patient 7.

In the above entities, cardiac catheterization and angiocardiography will be helpful in the differential diagnosis to identify the underlying anomaly.

**Pathology**

In the autopsied cases the heart was enlarged and the enlargement involved mainly the left atrium and the right ventricle. The right atrium and the pulmonary artery were often dilated. The aorta and left ventricle were usually normal or small in size. Oc-
CONGENITAL MITRAL STENOSIS

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occasionally left ventricular hypertrophy was present and probably due to concomitant mitral insufficiency and endocardial fibroelastosis. The latter process was usually present with selective involvement of the left heart. The stenotic mitral valve presented a wide range of pathologic deformities. It was usually thickened, funnel-shaped, or diaphragmatic. The commissures were fused or poorly defined. The chordae tendineae were thickened and shortened and the papillary muscles fibrotic and hypertrophic (fig. 5). These structures may agglomerate and eneroach on the cavity of the left ventricle (cases 3 and 6). In patient 5 a band of undifferentiated fibrotic tissue created a stenosis inside the left ventricle (fig. 6).

In four patients operated on by Gagnon,18 the valves at surgery were funnel-shaped without thickening or commisure formation, and in Scott’s case18 the mitral valve was apparently normal without evidence of thickening, but there was a membrane with a slit filling the orifice between the anterior and posterior leaflet.

The microscopic findings usually demonstrate the endocardial fibroelastosis and fibrotic mitral valve without evidence of stigmata of rheumatic fever31 or inflammatory cellular infiltration. Pulmonary arterioles reveal medial hypertrophy without significant intimal vascular changes. In our patient 4 atrophy of the cerebral white matter and basal ganglia of unknown cause were found.

Etiology

The etiology of isolated congenital mitral stenosis is debated, and the suggested etiologic factors include a developmental congenital anomaly,2 7 18 fetal endocarditis,16, 32 acquired inflammatory process of unknown cause4, 18 and primary endocardial fibroelastosis.8, 17, 33

In our cases, the valves were all thickened, endocardial fibroelastosis was present, and there was no evidence of an inflammatory process. The degree of endocardial fibroelastosis was variable, and it is still not known whether the mitral stenosis is a part of this process or whether the valve produces the endocardial disease. The latter apparently

ly is favored when the thickening is localized to the left atrium.29 Rheumatic fever as an etiologic factor was excluded because rheumatic mitral stenosis of physiologic significance is a rarity in infancy and childhood.34, 35 Also rheumatic lesions were not found at autopsy. In older children it is difficult to differentiate between rheumatic or congenital mitral stenosis. This applied particularly to those patients in whom a history of acute rheumatic fever is denied or in whom it is not known whether heart disease was present in early infancy.

Surgery

Fifteen patients including two of our patients (no. 5 and 6) were operated on for isolated congenital mitral stenosis and 10 died. All survivors were 6 years of age or older except one,14 a 4-month-old baby. In the other 12 patients mentioned in the discussion of Starkey’s paper, satisfactory results were apparently obtained in older children. The marked deformity of the mitral valve and its attachments and the presence of severe endocardial fibroelastosis greatly limit the reconstruction of an adequately functioning valve. These unfavorable factors are likely to occur in early childhood in a precariously ill infant and explain the poor surgical results. In older children a better surgical
result was obtained. Mitral insufficiency post-operatively is common. A hypoplastic left ventricle is a contraindication to surgery.

In an isolated instance finger fracture of the stenotic valve was accomplished in infancy. In Scott's 6-year-old patient a delicate membrane filling the orifice between the anterior and posterior leaflets of a normal mitral valve was divided easily with digital pressure.

Three patients (no. 5, 6, 7) of our series underwent surgery. Two died (no. 5 and 6) 48 to 72 hours after valvulotomy. Patient 7, who had a valvulotomy and resection of a coarcted aortic segment, developed marked cardiomegaly and mitral insufficiency and died 7 months postoperatively in intractable congestive heart failure. In patient 5 adequate incision of the commissures of the mitral valve was accomplished under direct vision. At autopsy, however, a significant degree of stenosis was present and mobility of the mitral valve was limited by a fibrous band inside the left ventricle (fig. 6). In patient 6 the mitral orifice was enlarged from 5 mm. to 17 mm. At autopsy the orifice admitted a probe of 1.3 cm. but the valve appeared fixed.

Two patients (no. 3 and 4) died without surgery at the age of 3 months and 6 months. In patient 3 marked endocardial fibroelastosis and the severe deformity of the mitral valve (fig. 5) indicated that simple valvulotomy would not have resulted in a significant improvement.

The treatment of severe mitral stenosis and intractable heart failure in infancy and early childhood is difficult because medical therapy alone usually results in a fatal outcome and the results of surgery are poor. A conservative approach is recommended in patients with moderately severe stenosis. In older children, the surgical indications are not unlike those of rheumatic mitral stenosis. Owing to the marked deformity of the mitral valve, surgery under direct vision during total body perfusion is advocated. When congenital

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**Figure 9**

Electrocardiogram of patient 6 showing prominent and notched P waves and absence of significant right ventricular hypertrophy in spite of the presence of pulmonary hypertension.
mitral stenosis is associated with coarctation of the aorta, simultaneous surgical correction of both anomalies is recommended. In patients with patent ductus arteriosus and mitral stenosis, severe pulmonary hypertension often develops, limiting the surgical indications.

Summary

Seven patients with isolated congenital mitral stenosis are described. In one instance there was an associated coarctation of the aorta. Repeated pulmonary infections, pulmonary edema, and congestive heart failure occurred frequently. The clinical diagnosis is suspected in the presence of an apical diastolic rumbling murmur and left atrial enlargement. Presystolic accentuation of this diastolic murmur, a sharp first heart sound, and an opening snap strengthen the diagnosis. Cardiomegaly and the electrocardiographic finding of right ventricular hypertrophy are common. Pulmonary hypertension and congestive heart failure may obscure the clinical picture of isolated congenital mitral stenosis. In infancy, the presence of tight mitral stenosis is usually associated with a poor prognosis.

The mitral leaflets showed variable degrees of fusion and thickening. The chordae tendineae were shortened and the papillary muscles hypertrophic. Endocardial fibroelastosis was common in the left heart, especially in the atrium.

In infancy and early childhood, the results of the medical and surgical treatment were generally poor. In older children, a better result was obtained and the surgical indications in these instances are not unlike those of rheumatic mitral stenosis.

Acknowledgment

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References

21. Varela DeSiasasas, J., DeRabago-Gonzalez, P.
Giovanni Battista Morgagni, the Founder of Pathologic Anatomy

Apoplexy greatly intrigued Morgagni, as it did his contemporaries, and, here, also, he made an outstanding contribution, but his progress in this subject was not so revealing as in other topics that he touched. He retained the ancient classification of serous and sanguineous apoplexy and, in doing so, he assigned to this category cases that probably were cerebral softening and cerebral edema. However, he brought additional evidence in support of Valsalva’s masterly observation that the paralysis ensuing from cerebrovascular accident occurs on the side opposite the lesion (as I mentioned before). Morgagni also made reference to the role that rupture of small aneurysms of the cerebral vessels (first described by Wepfer and Brunner) may play in the occurrence of cerebrovascular accident, but he put more emphasis on some sort of disorder in the choroid plexus as the most frequent cause of the hemorrhage. Here, his reasoning has a touch of ancient humoral medicine, but, even so, the recital opened new avenues of thought.—C. G. Tedeschi, M.D. Giovanni Battista Morgagni, The Founder of Pathologic Anatomy: A Biographic Sketch On the Occasion of the 200th Anniversary of The Publication Of His “De sedibus et causis morborum per anatomen indagatis.” The Boston Medical Quarterly 12: 123, 1961.
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