Unilateral Pulmonary Artery Absence or Hypoplasia
Radiographic and Cardiopulmonary Studies in Five Patients

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With the technical assistance of Joan Lawrence, R.N., B.S., and Autie Belle Godfrey

The clinical entity of unilateral pulmonary artery absence or hypoplasia can be readily diagnosed on the basis of routine clinical evaluation. This report is based upon 5 patients in whom the diagnosis was suspected and eventually confirmed with the aid of special studies. Characteristic hemodynamic findings were demonstrated by methods of cardiac catheterization and contrast visualization of the cardiopulmonary system.

Since this clinical entity was first diagnosed with the aid of angiocardiology by Madoff and co-workers in 1952,1 case reports of unilateral absence or hypoplasia of a main branch of the pulmonary artery have appeared with increasing frequency. Presumptive diagnosis is now possible merely on the basis of routine roentgenograms of the chest. Typical findings have been reviewed and summarized by Wyman.2

A review of case reports shows that the diagnosis has been made angiocardiographically in at least 13 instances. Physiologic studies have been reported in few, cardiac catheterization in 6, and pulmonary function studies in 6.1–6 The condition has been encountered most frequently in association with, or as an anatomic variant of, other serious intracardiac anomalies.7–10

Unilateral absence or hypoplasia of a pulmonary artery is of particular interest to us because it is a naturally occurring chronic equivalent of the acute studies that have been carried out in this laboratory in recent years.11 We have found that temporary occlusion of 1 main branch of the pulmonary artery by means of a balloon-tipped cardiac catheter has been a most useful tool in the evaluation of cardiopulmonary hemodynamics.

The clinical condition under consideration presents chronic occlusion of a pulmonary artery so that most or all of the cardiac output perfuses a single lung while the unoccluded lung, though ventilated, has no gas exchange.

In general, the clinical findings in previously reported cases, in which there were no other serious intracardiac defects, have been asymmetry of the thorax resulting from either hypoplasia of the affected side or compensatory emphysema of the unaffected side, decreased breath sounds on the affected side, hyperresonance on the unaffected side, ill-defined cardiac murmurs, usually systolic, and heard best over the pulmonic region or over the apex. The presenting symptoms have been cough, shortness of breath, hemoptysis, and recurrent pulmonary infection. Roentgenograms of the chest have confirmed the physical findings and have added the most important diagnostic sign: a discrepancy between the vascular markings of the 2 lung fields.2

Those patients in whom hemodynamic studies have been performed have shown normal pulmonary artery pressures at rest and a slightly exaggerated rise in pulmonary artery pressure on exercise. The ventilatory studies have shown changes consistent with increased dead space or residual volume. Oxygen uptake on the side of the absent artery has been negligible. The cases herein reported have followed the same general pattern with certain important differences, as demonstrated by refinements in technic.

From the Eva and Irving Hexter Laboratory for Cardiopulmonary Research, Mount Sinai Hospital, Cleveland, Ohio.
Aided by Grants from the Cleveland Area Heart Society.
TABLE 1.—Hemodynamic Data in Patients with Unilateral Absence or Hypoplasia of the Pulmonary Artery

<table>
<thead>
<tr>
<th>Case</th>
<th>Control</th>
<th>Exercise</th>
<th>Pulmonary &quot;wedge&quot; pressure</th>
<th>Main pulmonary artery</th>
<th>Right ventricle</th>
<th>Arterial O2 (vol. %) and % saturation</th>
<th>Mixed venous O2 (vol. %)</th>
<th>Cardiac output (L./min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>32/18 (22)</td>
<td>45/25 (36)</td>
<td>32/8 (18)</td>
<td>15.6 (89)</td>
<td>11.3</td>
<td>5.6</td>
</tr>
<tr>
<td>2</td>
<td>Control</td>
<td>Partial occlusion of right pulmonary artery</td>
<td>10–5</td>
<td>44/15 (20)*</td>
<td>46/10 (17)</td>
<td>16.1 (93)</td>
<td>14.8</td>
<td>11.4</td>
</tr>
<tr>
<td>3</td>
<td>Control</td>
<td>Partial occlusion of right pulmonary artery</td>
<td>9–0</td>
<td>23/8 (16)</td>
<td>28/6 (12)</td>
<td>17.8 (95)</td>
<td>14.3</td>
<td>6.6</td>
</tr>
<tr>
<td>4</td>
<td>Control</td>
<td>Partial occlusion of left pulmonary artery</td>
<td>12–5</td>
<td>28/14 (18)</td>
<td>28/4</td>
<td>14.8 (90)</td>
<td>12.1</td>
<td>5.1</td>
</tr>
</tbody>
</table>

* In the right pulmonary artery—28/12 (16).
Pressures in mm. Hg.

MATERIALS AND METHODS
Cardiac catheterization was carried out in the usual manner via an antecubital vein. In those cases in which pulmonary artery occlusion was performed, a special triple-lumen catheter was employed. This catheter allows measurement of pressures and the procurement of blood samples proximal and distal to the occluding balloon simultaneously. The catheter was passed under fluoroscopic guidance, and the balloon was inflated with contrast material in the desired pulmonary artery segment. Pressures were measured by a Sanborn electromanometer or Statham strain gage and were recorded by a 4-channel direct writer (Sanborn Polyviso). Generally, the oxygen content of blood samples was determined by the method of Van Slyke. Routine pulmonary function studies were carried out in the usual manner. Oxygen consumption was determined by means of a Pauling oxygen analyzer with the patient breathing room air. Most angiocardiograms were performed by the method of Brofman of intratrial instantaneous release of contrast material from a balloon. X-ray exposures were made at ½-second intervals with a Fairchild roll-film camera.

Whenever possible, the studies included cardiac catheterization, pulmonary function studies, bronchography, bronchoscopy, and cardioangiography. Results are summarized in tables 1 and 2.

REPORT OF CASES
Case 1. This 43-year-old white woman was examined on August 24, 1954, for consideration of mitral commissurotomy. She had chorea as a child, and a heart murmur was heard at that time. For 2 years prior to the present study she had increasing shortness of breath, particularly at night, with temporary improvement after digitalization. Cough productive of moderate amounts of yellowish-white sputum occasionally streaked with blood also developed.

On examination the patient showed no acute distress or clubbing. The left hemithorax showed a slight lag on inspiration and was less resonant than the right, and the breath sounds at the left base were diminished. The pulmonic second sound was accentuated. A soft systolic murmur was heard best in the third and fourth left interspaces. The electrocardiogram was normal.

The findings of cardiac catheterization precluded a diagnosis of dynamically significant mitral stenosis (table 1). Cardiac fluoroscopy revealed reduced pulmonary vascular markings on the left, suggesting unilateral hypoplasia of the left pulmonary artery (fig. 1).

On bronchoscopy the carina appeared pulled to the left. The right tracheobronchial tree was normal. The left main-stem bronchus was reduced in size and contained tenacious secretions. Bronchiectasis was demonstrated in the left lung by
TABLE 2.—Pulmonary Function Studies

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity (L.)</td>
<td>2.3(78%)</td>
<td>2.9(69%)</td>
<td>3.3(76%)</td>
</tr>
<tr>
<td>Timed vital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>capacity 1 sec. (%)</td>
<td>72</td>
<td>85</td>
<td>65</td>
</tr>
<tr>
<td>Timed vital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>capacity total (sec.)</td>
<td>8</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Walking ventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(L./min./M.)</td>
<td>18.7</td>
<td>9.4</td>
<td>10.0</td>
</tr>
<tr>
<td>Minute ventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(L.)</td>
<td>11.8</td>
<td>9.6</td>
<td></td>
</tr>
<tr>
<td>Maximum breathing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>capacity (L.)</td>
<td>66 (87%)</td>
<td>125 (119%)</td>
<td>113 (65%)</td>
</tr>
<tr>
<td>Walking index</td>
<td>42</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>Bronchospirometry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>02 consumption right</td>
<td>95</td>
<td>87</td>
<td></td>
</tr>
<tr>
<td>Bronchospirometry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>02 consumption left</td>
<td>5</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Vital capacity right</td>
<td>85</td>
<td>69</td>
<td></td>
</tr>
<tr>
<td>Vital capacity left</td>
<td>15</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td>Minute ventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>right (%)</td>
<td>83</td>
<td>77</td>
<td></td>
</tr>
<tr>
<td>Minute ventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>left (%)</td>
<td>17</td>
<td>23</td>
<td></td>
</tr>
</tbody>
</table>

bronchography. Pulmonary function studies showed negligible oxygen uptake from the left lung.* The increase in basal minute volume, walking ventilation, and walking index (i.e., W.V./M.B.C.) were consistent with increased dead space.

The angiocardiogram confirmed the diagnosis of hypoplasia of the left pulmonary artery. Since the lung was the seat of extensive suppurative disease and contributed little to oxygen uptake, a left pneumonectomy was performed.

Examination of the surgical specimen showed diffuse chronic bronchiectasis. The left pulmonary artery was diminutive. It measured 4 mm. in diameter at its origin and could be traced only a few centimeters peripherally. Two large bronchial

*We wish to acknowledge the aid of Martha Bender, M.D., in carrying out these studies at The Cleveland City Hospital.

vessels were present and were adherent to the main-stem bronchus.

Since operation the patient has experienced marked clinical improvement. Cough and exertional dyspnea have diminished and hemoptysis has not recurred.

Case 2. A 25-year-old Negro had been well all
his life with the exception of an episode of aching joints lasting a few weeks, 5 years previously. He had been rejected for military service but did not know the reason. Three months before hospitalization he noted malaise, night sweats, small amounts of blood-streaked sputum, and shortness of breath on exertion. Two days before admission he developed chills, fever, and frank hemoptysis.

The patient appeared acutely ill. There was no clubbing of the fingers. The chest was clear to percussion and auscultation and showed no asymmetry. The peripheral pulses were bounding in character. The blood pressure was 120/60/20 mm. Hg. The left apex beat was visible at the left anterior axillary line. A diastolic thrill was palpable in the second and third left interspaces. Systolic and diastolic murmurs were heard over the base of the heart in the midline. The aortic second sound was diminished and the pulmonic second sound was accentuated. The electrocardiogram showed early left ventricular hypertrophy. Roentgenograms of the chest disclosed an abscess of the left lower lobe. Sputum cultures grew mixed staphylococci, streptococci, and pneumococci.

Cardiac catheterization was done because of suspected patent ductus arteriosus and subacute bacterial endocarditis; however, no intracardiac shunt was demonstrated.

The cardiac index was high and there was a large arteriovenous difference (table 1). As part of the hemodynamic study, the right middle and lower lobes were occluded. The patient rapidly developed marked respiratory distress necessitating deflation. During the brief period of occlusion there was a marked, sustained rise in pulmonary artery pressure (table 1). Such a dramatic response to occlusion of a branch of the pulmonary artery had not been previously observed and was subsequently explained when absence of the left pulmonary artery was demonstrated by intracardiac angiocardiography (fig. 2).

Review of the roentgenograms disclosed reduced vascular markings on the left; however, the other common findings were not demonstrated. Bronchography revealed a normal tracheobronchial tree. Routine pulmonary ventilatory studies were completely normal except for a slight reduction in vital capacity. Blood ′O2, ′CO2, and O2 saturation were normal.

The hemodynamic studies in this case shown in table 1 are of particular interest. It has been shown by Liebow and his co-workers8,9 that large communications can develop between the pulmonary and bronchial arteries. The presence of such communications could well explain the collapsing peripheral pulse observed in view of the low resistance of the pulmonary vascular bed. It was also noted that as the catheter passed peripherally in the right pulmonary artery, there was a fall from the elevated main pulmonary artery pressure. This gradual fall appears to be produced by the increased flow through relatively nonexpansible pulmonary arteries. Thus the larger vessels rather than the arterioles become the determinants of pressure-flow relationships. The
situation might be considered a physiologic coarctation. Several such large communications could simulate a patent ductus arteriosus. However, it is also possible that the collapsing peripheral pulse might be related to some degree of actual aortic insufficiency on the basis of congenital or rheumatic aortic valve disease. This patient is now doing well with the exception of continued cough productive of blood-streaked sputum.

Case 3. A 26-year-old white man first noted aching in his left chest following vigorous exercise in June 1955. X-rays revealed emphysema of the left lung. There were no other symptoms, but the patient was hospitalized for study.

On physical examination there was flattening anteroposteriorly and a definite lag of the left hemithorax on both inspiration and expiration. The percussion note was normal bilaterally. Loud inspiratory and expiratory rhonchi were heard anteriorly in the second to fourth interspaces. The heart showed no abnormality; the blood pressure was 146/96 mm. Hg.

Routine laboratory studies were normal. On 2 occasions bronchoscopy showed marked narrowing at the left main-stem bronchus just below the carina. This was seen to function as a check valve with the lumen patent on inspiration but collapsed on expiration. Bronchograms demonstrated moderate bronchiectasis of the lingula and the left lower lobe. The angiocardiogram confirmed the presence of hypoplasia of the left pulmonary artery, which had been suspected from the posteroanterior roentgenograms of the chest (fig. 3).

Pulmonary function studies revealed marked diminution of ventilation and oxygen consumption of the left lung (table 1). Cardiac catheterization (table 2) showed a normal resting pulmonary artery pressure. Occlusion of the right middle and lower lobe branches of the pulmonary artery produced a marked rise in pulmonary artery pressure and a fall in arterial oxygen saturation. The cardiac output decreased, but the procedure was well tolerated by the patient. During this maneuver the only normal lung being perfused was the right upper lobe. Despite the hypoplastic left pulmonary artery, the increase in pressure may have increased perfusion to the left lung as well. The fall in arterial oxygen content could indicate that either the maximum diffusing capacity of the right upper lobe had been exceeded by the excessive blood flow, or, more probably, the increased perfusion of the diseased left lung caused inadequate oxygenation and produced venous admixture.

The patient is at present being treated conservatively and his condition is satisfactory except for a slight productive cough.

Case 4. The patient, a 39-year-old white woman, had had a chronic cough and frequent severe chest colds for many years. In 1953 she consulted a physician but no definite diagnosis was made. Approximately 6 months prior to admission to the hospital, the cough became productive of blood-streaked sputum. Two days before admission frank hemoptysis began and continued until hospitalization.

On physical examination the left hemithorax was slightly larger and more resonant than the right. The heart was not enlarged, and no mur-
marked discrepancy between the vascular markings of the 2 lung fields was observed, suggesting the absence of the right pulmonary artery. Comparison with films taken in 1947 showed no change.

During cardiac catheterization (tables 1 and 2) a secondary branch of the left pulmonary artery was occluded. The pressure proximal to the balloon rose markedly considering the small size of the vessel occluded. There was no change in peripheral arterial oxygen content. Angiocardiography (fig. 4) confirmed the diagnosis. Unfortunately, pulmonary function studies were not obtainable. The subsequent course has been thus far uneventful.

Case 5. A 66-year-old white woman had had frequent colds and chronic cough for many years. There had been moderate shortness of breath on exertion for 2 years. Ten days before hospitalization a moderate hemoptysis occurred.

On physical examination the left hemithorax showed decreased breath sounds, vocal fremitus, and resonance. The heart was not enlarged and no murmurs were heard. The remainder of the physical examination was not remarkable.

Posteroanterior roentgenograms of the chest (fig. 5), showed a discrepancy between the vascular markings on the 2 sides. On fluoroscopy, the right pulmonary artery was seen to pulsate markedly but pulsations were not seen on the left side. Laminography revealed a large right pulmonary artery but none on the left. The electrocardiogram was normal. The patient refused further study and was discharged with the diagnosis of hypoplasia or absence of the left pulmonary artery.

**Discussion**

**Right Ventricular Work Load.** These cases demonstrate that 1 pulmonary artery can be entirely absent or markedly reduced in size without imposing a tremendous increase in the work load of the right ventricle. None of the patients had electrocardiographic evidence of right ventricular hypertrophy, even the one who was 66 years old. In 3 of 4 cases cardiac catheterization showed normal resting pulmonary artery pressures. Since essentially the entire output of the right ventricle was perfusing 1 lung, however, the ability of the vascular bed of this lung to accommodate a further increase in blood flow was diminished. When a portion of existing pulmonary vascular bed was removed from the circulation by occlusion with a balloon, pulmonary artery pressure proximal to the occlusion increased...
markedly. This change is in contrast to normal patients who show only a slight rise. One might further expect that such individuals would have exaggerated rise in pulmonary artery pressure on exercise, since the functioning lung is already approaching its limit of expansibility. This was found in case 1. At rest there appears to be little added burden to the right ventricle but on exertion, with increased cardiac output, the restricted pulmonary vascular bed approaches the limits of expansibility. The increased flow with relatively fixed resistance results in an increased right ventricular work load.

*Left ventricular work load.* Liebow and his co-workers demonstrated that following ligation of 1 pulmonary artery communications develop between the pulmonary artery and the bronchial arteries at the precapillary level. In effect, they produced an A-V fistula from the aorta to the pulmonary bed. Under such circumstances the output of the left ventricle may exceed that of the right by as much as one third with a resulting increased work of the left ventricle. Loring and Liebow,16 however, were unable to demonstrate any significant cardiac hypertrophy following ligation of a pulmonary artery. Similarly, we found no evidence of left ventricular hypertrophy in 4 of the 5 cases presented here. The fifth case had electrocardiographic and roentgenographic evidence of left ventricular hypertrophy but this may have been due to an aortic valvular lesion. It seems doubtful that such shunting of blood could contribute significantly to left ventricular failure in the absence of intrinsic heart disease.

**Bronchial Arteries.** Previous reports have mentioned the appearance of bronchial arteries in the angiocardiogram. These appeared at approximately the same time as opacification of the aorta. Careful review of our angiocardiograms revealed a structure presumed to be an opacified bronchial artery in case 2. A surgical specimen was available only in case 1, and large bronchial vessels were present in this lung, but could not be identified in the angiocardiogram.

It is known that animals and man can tolerate complete ligation of the pulmonary arterial supply with little pathologic change providing the bronchial arteries are intact.
Nutrition is maintained through these bronchial arteries, but, the bronchial artery blood participates in oxygen exchange to a relatively insignificant degree. Bronchial artery blood being well oxygenated can exchange only small amounts of oxygen because of the small partial pressure gradient. By contrast, in certain types of congenital heart disease bronchial artery blood flow may be very large and important. In our cases and in others reported, oxygen uptake has been markedly diminished or absent on the involved side. Bronchial arterial blood circulation may be responsible for at least part of the oxygen uptake demonstrated.

Pulmonary Function Studies. The pulmonary function studies performed in 3 cases are of interest particularly when correlated with the roentgenographic studies of the bronchial tree. Case 2 showed no bronchiectasis by x-ray, had good function as manifested by a short lung-emptying time and an excellent maximum breathing capacity, despite a reduction in vital capacity. The other 2 cases, both with bronchiectasis, showed definitive evidence of obstructive lung disease, on the basis of both bronchiectasis and bronchostenosis in case 3 and bronchiectasis in case 1. Emphysematous changes on the uninvolved side may also be a contributing factor.

While very little or no oxygen consumption occurs with curtailment of pulmonary arterial flow, the elevated resting minute ventilation as well as the exaggerated response to mild exercise (walking ventilation) emphasize that the lung continues to ventilate to some degree. With exertion this useless ventilation may become considerable, accounting to some extent for the symptom of dyspnea on effort. The observed uniform decrease in vital capacity can be ascribed to either a smaller hemithorax or inflammatory disease of the lung.

Case 1 is the only one that shows significant diminution in peripheral oxygen saturation. It seems likely that this is the result of uneven relationships of perfusion and ventilation in the bronchiectatic lung with an extremely small pulmonary artery. The high minute ventilation and walking ventilation observed in this patient were in response to the arterial hypoxia as well as the useless ventilation.

Clinical Entity. One important question is whether or not this can be an acquired condition. Thrombotic occlusion can be ruled out, since our cases do not present the clinical characteristics of those described in the literature. The more likely possibility of "physiologic amputation" of a pulmonary artery in cases 1 and 3, as demonstrated by Liebow in bronchiectasis, must be considered. We were not able to determine whether or not an "atrophy of disuse" had occurred in the 1 case in which the anatomic specimen was obtained. It seems equally logical to assume that these patients represent cases of a congenital defect in the arterial supply of the lung with superimposed inflammatory disease. The presence of a normal tracheobronchial tree in cases 2 and 4 further strengthens the contention that this is basically a congenital defect.

Characteristic physical findings, history, and roentgenographic findings have been well set forth elsewhere. We would like to emphasize that unilateral absence or hypoplasia of a pulmonary artery may occur as an isolated and symptomatic defect. Hemoptysis occurred in all of our cases at some time during the history. Although this may be associated with inflammatory disease of the lung, large bronchial arterial communications may still be implicated. The cardiovascular system appears to adjust well to the altered hemodynamics.

Unilateral absence or hypoplasia of a pulmonary artery is reported as occurring most frequently on the right side. However, in 4 of our 5 cases the left lung was involved. The difference in incidence on the 2 sides may be more apparent than real in view of the small number of cases reported up to this time.

Treatment. Therapeutic considerations are concerned primarily with inflammatory disease of the lung and pulmonary hemorrhage. Since one lung is maintaining gas exchange, it must be protected against injury.
tion of the nonperfused lung can be carried out without impairment of respiratory function and surgery is indicated when hemoptysis and recurrent infections become serious problems.

**Summary**

Five cases of unilateral absence or hypoplasia of a main branch of the pulmonary artery are presented. To our knowledge this is the largest such series in which the diagnosis has been made prior to autopsy or surgical exploration. These patients showed thoracic asymmetry and disparity between the vascular markings in the lung fields. The resting pulmonary artery pressures were usually normal. On exercise there was an exaggerated rise in pulmonary artery pressure. With occlusion of a segment of the existing pulmonary vascular system by a balloon-tipped catheter there was a marked rise in pulmonary artery pressure. There was negligible oxygen uptake on the side of the absent artery. The condition is emphasized as a symptomatic clinical entity.

**Summario in Interlingua**

Es presentate 5 casos de absentia o hypoplasia unilateral de un branche major del arteria pulmonar. Secundo nostre information, isto es le plus extense tal serie in que le diagnoses eseva establite ante le necropsia o un exploration chirurgic. Iste patientes exhibiva asymetria thoracica e disparitate del marces vascular in le campos pulmonar. Le pressiones del arteria pulmonar in stato de reposo eseva usualmente normal. Post exercitio il occurriva un exaggerate augmento del pression pulmono-arterial. Occlusion de un segmento del existente systema pulmonar vascular per medio de un catheter a puncta ballonate resultava in un augmento marcate del pression pulmono-arterial. Un negligilable accpetation de oxygeno eseva constatatate al latere del arteria absent. Es signalate que le condition debe esser considerate como un symptomatic entitate clinico.

**Acknowledgment**

We gratefully acknowledge the patient aid of Miss Annette Frankle and Mrs. Lillian Schoenberger in the preparation of this manuscript, and the kind cooperation of the members of the Radiology Department, and Sidney Wolpaw, M.D., in the performance and interpretation of the x-ray studies.

**References**

13. Bloomer, W. E., Harrison, W., Lindskog,


So in whatsoever creature there is lungs, there is likewise in them two ventricles of the heart, the right and the left, and wheresoeuer the right ear is in any, there is the left, not on the contrary, that where the left is, there is the right one too; that I call the left ventricle which is distinguished in place, but not in use from the tother, which doth diffuse the blood into the whole body, not into the lungs alone, hence the left ventricle seems to make up the heart of it self, being placed in the middle, and so fenc'd with higher ditches, and fram'd with greater diligence that the heart seems to have been made for the left ventricle's sake, and the right ventricle seems as it were a servant to the left, and does not reach to the top of it, and is made up of a thinner threefold wall, and it has, as Aristotle says, a kind of articulation above the left, and is more capacious, as administering not only matter to the left, but giving nourishment likewise to the lungs.—William Harvey. De Motu Cordis, 1628.
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