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THE ANATOMY OF PULMONARY STENOSIS AND ATRESIA WITH COMMENTS ON SURGICAL THERAPY

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IN THE tetralogy of Fallot certain anatomic variations occur. The malformation was recognized by Stensen in 1672, Sandifort in 1777, John Hunter in 1783, and Hope in 1830, but the variable patterns of the anomaly were first described in the writings of Peacock in 1866, of Fallot in 1888, and of Keith in 1909.

These variations primarily are seen in the extent and the site of the obstruction to the flow of blood to the lungs through the outlet of the right ventricle. The pulmonary obstruction may be complete (atresia) or partial (stenosis); it may be located at the valve, more frequently at the infundibulum, and occasionally it occurs at both the valve and the infundibulum. Equally variable is the degree of dextroposition of the aorta; this vessel may be only slightly deviated to the right, or it may be dextroposed so greatly as to appear to arise almost entirely from the right ventricle. The remaining two components of the tetralogy, namely, the interventricular communication and the hypertrophy of the right ventricular wall, appear to be less variable features of the anomaly.

Although Thomas Peacock did not differentiate the tetralogy from pulmonary stenosis in which there is no interventricular communication, he was the first to appreciate variations in the anatomy of the pulmonary obstruction. In the classic papers of Etienne Louis-Arthur Fallot on the Maladie Bleue, all types of pulmonary stenosis are clearly described, including a case in which there was stenosis of both the infundibulum and the valve. Fallot was also the first to study the nature of the dextroposition of the aorta; he described in detail instances of varying degrees of deviation of the aorta to the right.

In 1909 Sir Arthur Keith\textsuperscript{11} recognized the following variations in malformations of the pulmonary conus: (1) subdivision of the right ventricle into two chambers by the stenosis, examples of what we have called infundibular stenosis with a well-developed infundibular chamber, (2) arrested expansion of the infundibulum, (3) almost complete arrest of development of the infundibulum, and (4) stenosis due mainly to fusion of the valves. The double stenosis known to Fallot\textsuperscript{7} was not described by Keith nor did he note variations in the position of the aorta.

In 1949 Brock\textsuperscript{12-15} made careful morphologic observations of pulmonary stenosis. These studies led him to carry out a direct surgical attack upon the obstruction, first in cases of isolated valvular pulmonary stenosis without an interventricular defect, later in instances of infundibular stenosis in the tetralogy of Fallot. Brock emphasized (1) the role of the crista supraventricularis in the formation of the infundibular stenosis, (2) the localized nature of the stenosis in some cases of the tetralogy of Fallot, and (3) the nature of the infundibular chamber. He divided infundibular stenosis into five types depending upon the degree of development of the infundibular chamber.

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{heart_diagram.png}
\caption{Drawing showing site of aorta and degree of dextroposition in a typical specimen of tetralogy of Fallot with pulmonary atresia located at the pulmonary valve. The pulmonary artery is hypoplastic. Details of the atroventricular valves have been omitted.}
\end{figure}

\textbf{RESULTS}

We are reporting the results of a study of 95 autopsy specimens of pulmonary stenosis or atresia associated with an interventricular septal defect (tetralogy of Fallot); valvular pulmonic stenosis with an intact ventricular septum is not included. Since 79 of these 95 specimens were obtained from patients who died at varying times following operation, it is permissible to assume that most of them had severe pulmonary stenosis or atresia or complicating conditions such as unusual dextroposition of the aorta.
The 95 specimens studied have been divided into four groups. This classification is based upon the anatomy of the region of the pulmonary conus and valve:

1. Pulmonary atresia
2. Infundibular pulmonary stenosis
3. Valvular pulmonary stenosis
4. Combined infundibular and valvular pulmonary stenosis.

The second group (infundibular stenosis) has been subdivided according to the size of the infundibular chamber. Table I gives the relative incidence in the several groups in this series.

**Table I. Tetralogy of Fallot**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of cases</td>
<td>95</td>
</tr>
<tr>
<td>Pulmonary atresia</td>
<td>22</td>
</tr>
<tr>
<td>Pulmonary infundibular stenosis</td>
<td>49</td>
</tr>
<tr>
<td>Pulmonary valvular stenosis</td>
<td>10</td>
</tr>
<tr>
<td>Pulmonary infundibular and valvular stenosis</td>
<td>14</td>
</tr>
</tbody>
</table>

1. **Pulmonary Atresia.**—In 22 cases of tetralogy of Fallot there was no direct communication between the right ventricle and the pulmonary artery. In 19 cases the atretic area was at the very apex of the conus in the region of the pulmonary valve; in the remaining 3 the atresia was in the infundibulum and a valve was demonstrable (Fig. 1). In 19 of the 22 cases the pulmonary artery was small, in 1 case it was absent, and in the remaining 2 cases it was essentially normal in size. The aorta was larger than normal in most cases, and usually greatly transposed to the right. The enlargement and dextroposition of the aorta appeared to be inversely proportional to the size of the pulmonary artery (Table II).

**Table II. Tetralogy of Fallot With Pulmonary Atresia**

<table>
<thead>
<tr>
<th>Development of pulmonary artery</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>2</td>
</tr>
<tr>
<td>Small</td>
<td>19</td>
</tr>
<tr>
<td>Absent</td>
<td>1</td>
</tr>
<tr>
<td>Apparent dextroposition of aorta</td>
<td></td>
</tr>
<tr>
<td>60% or more from right ventricle</td>
<td>18</td>
</tr>
<tr>
<td>Less than 60% from right ventricle</td>
<td>4</td>
</tr>
</tbody>
</table>

In one of these cases the pulmonary atresia consisted of a thin diaphragm in the region of the valve which could possibly have been pierced by a sharp instrument if a direct attack had been attempted. It is doubtful, however, whether the exact nature of the deformity could have been recognized at the time of exploration.

2. **Infundibular Pulmonary Stenosis.**—The proximal end of the conus arteriosus was the site of the obstruction in 49 cases. This type of stenosis is produced by a muscular structure which normally separates the inflow tract from the outflow tract of the right ventricle and which has been termed the crista supraventricularis or infundibular crest. This band of cardiac muscle
extends across the superior aspect of the right ventricle from its anteromedian wall to the interventricular septum where it is inserted at the anterior margin of the interventricular communication. The crista supraventricularis hangs down in front of the interventricular defect like an oblique curtain, its inferior border arched to provide an opening into the pulmonary outflow tract. As its projections merge into the ventricular walls, the crista presents two features of practical anatomic significance: It is closely apposed posteriorly to the base of the aortic valve, and its anterior insertion is close to the anteromedian papillary muscle which contains the right ventricular branch of the bundle of His.

The development of the crista supraventricularis determines the type of stenosis at the infundibulum, and its position in the ventricular cavity is responsible for the size of the infundibular chamber. From the viewpoint of the development of the infundibular chamber, the 49 cases in our autopsy series showed a well-developed chamber in 18, moderately well-developed in 13, and poorly developed in 18 cases.

![Fig. 2.—Drawing of heart with tetralogy of Fallot with infundibular stenosis and a well-developed infundibular chamber. Expanded chamber above localized stenosis is shown. The pulmonary artery is normal in size.](image)

**Well-developed infundibular chamber (Fig. 2):** Most of the patients from whom these 18 specimens were obtained fall in an older age group than those in whom there was a high and small infundibular chamber. When the columns of the crista supraventricularis converge to form a muscular diaphragm, the stenosis lies fairly low in the right ventricular cavity. The distance from the crest to the pulmonary valve is relatively great, measuring 18 mm. or more, and the infundibular chamber has a diameter of 3 cm. or more.* In its extreme form the well-developed chamber is known as the third ventricle. This is the

*These measurements were made on fixed specimens and are used only as a method of comparison. Furthermore, though the infundibular chamber does not seem to vary in size in different age groups, an occasional chamber in a very small heart was termed well developed though its measurements fell slightly below those mentioned here.
its anteromedian e anterior margin atrialis hangs urtain, its inferior low tract. As its two features of erly to the base teromedian papil bundle of His. aines the type of cere cavity is respons viewpoint of the our autopsy series ed in 13, and

patmost favorable situation among the infundibular stenoses for direct surgical attack by the Broek technique.

It is difficult in a fixed specimen, and even in a fresh one, to determine accurately the degree of dextroposition of the aorta. Our observations do indicate fairly clearly that patients with a well-developed infundibular chamber do not usually have as much dextroposition of the aorta as do those with a high small infundibular chamber (Table III). Furthermore, the aortic orifice is usually fairly normal in size in those patients with a well-developed infundibular chamber, whereas it is often much enlarged in those with a small chamber. The size of the pulmonary artery usually varies inversely with that of the aorta.

![Diagram of the heart showing tetralogy of Fallot with infundibular stenosis and a moderately well-developed chamber.](image)

**Fig. 3**—Drawing showing tetralogy of Fallot with infundibular stenosis and a moderately well-developed chamber. Note that the crista supraventricularis is higher than in the previous specimen. Its position between the pulmonary artery and aortic outflow tracts is demonstrated.

*Moderately well-developed infundibular chamber (Fig. 3):* In the cases of this type the crista supraventricularis is placed higher in the ventricle, so that its distance from the pulmonary valve measures 12 to 18 mm. In this intermediate group the infundibular chamber has a circumference of 15 to 30 mm. The stenotic area does not present the appearance of a perforated diaphragm. It is the shape of a funnel and is rimmed by a wall of cardiae muscle. This rim is formed by a thickened projection of the anterior ventricular wall at its junction with the infundibular crest. There were 13 cases of this type in this series. It is possible that a direct surgical attack could have been carried out successfully in some of these cases; however, the feasibility of infundibular resection is limited by the diminished size of the infundibular chamber. Resection of this high-lying infundibular curtain is also fraught with the danger of injury to the aortic valve.

*Poorly developed infundibular chamber (Fig. 4):* A frequent type of infundibular stenosis is that associated with a hypoplastic outflow tract and a
poorly developed infundibular chamber. In 18 of our 49 cases the crista supraventricularis was placed so high in the right ventricular cavity that the stenosis lay less than 12 mm. from the valve. In such cases the infundibular chamber is not only short but narrow. The funnel-like outlet is narrowed by thick muscle which gives the outflow tract the appearance of a small tunnel. In most instances there is a slight expansion of the chamber proximal to the pulmonary valve ring. Only with extreme difficulty can a surgical instrument penetrate such a tunnel, and in this type of high infundibular stenosis the possibility of successful infundibular resection is severely limited. In 14 of the 18 cases there was marked dextroposition of the aorta. Furthermore, the aorta was usually larger and the pulmonary artery smaller than normal.

![Diagram](image)

Fig. 4.—Drawing to demonstrate tetralogy of Fallot with infundibular stenosis and a poorly developed chamber. There is usually slight dilatation above the stenosis. The pulmonary valve, as illustrated, is frequently bicuspid. The aorta is large and the pulmonary artery is small.

3. Valvular Pulmonary Stenosis.—The stenosis was found to be in the pulmonary valve in 10 cases (Fig. 5). Structurally this type of stenosis is similar to that which occurs in cases of stenosis of this pulmonary valve without a ventricular septal communication. Fallot’s description cannot be surpassed:

The pulmonary artery has the shape of a cone whose apex corresponds to the upper end of the infundibulum; the stenosis at this level is very marked; a transverse section taken two or three centimeters from the base demonstrates a veritable funnel. . . . The valve cusps are raised along the wall of the artery, fused together and form an actual diaphragm, pierced in the center by a narrow opening. The caliber of the aorta on the other hand is very notably enlarged.

The pulmonary artery is generally normal in size or is dilated distal to the stenosis. In some cases there is narrowing of the infundibulum. The aorta straddled the ventricular septum without much deviation to the right in 7 cases. In the remaining 3, 60 per cent or more of its orifice appeared to overlie the right ventricle.
ANATOMY OF PULMONARY STENOSIS AND ATRESIA

TABLE III. TETRALOGY OF FALLOT; APPARENT DEXTROPOSITION OF AORTA

<table>
<thead>
<tr>
<th></th>
<th>TOTAL</th>
<th>60% or more from right ventricle</th>
<th>Less than 60% from right ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infundibular stenosis</td>
<td>49</td>
<td>27</td>
<td>22</td>
</tr>
<tr>
<td>Well-developed chamber</td>
<td>18</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>Moderately well-developed chamber</td>
<td>13</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Poorly developed chamber</td>
<td>18</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Pulmonary atresia</td>
<td>22</td>
<td>18</td>
<td>4</td>
</tr>
</tbody>
</table>

4. Combined Infundibular and Valvular Pulmonary Stenosis.—In 1888 Fallot described this combination of defects in a 26-year-old man. Stenosis at both the pulmonary valve and the infundibulum occurred in 14 cases in our series (Fig. 6). This combination of defects occurred mainly in patients in the older age group. In approximately one-half of these patients the interposed infundibular chamber was fairly large and a direct attack would have been possible. Such an attack necessitates infundibular resection plus valvulotomy. In 9 of the cases it appeared that 60 per cent or more of the orifice of the aorta was overlying the right ventricle (Table IV).

TABLE IV. TETRALOGY OF FALLOT WITH VALVULAR AND INFUNDIBULAR STENOSIS

<table>
<thead>
<tr>
<th>Development of infundibular chamber</th>
<th>Total number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well developed</td>
<td>6</td>
</tr>
<tr>
<td>Moderately well developed</td>
<td>2</td>
</tr>
<tr>
<td>Poorly developed</td>
<td>6</td>
</tr>
<tr>
<td>Dextroposition of aorta</td>
<td></td>
</tr>
<tr>
<td>60% or more from right ventricle</td>
<td>9</td>
</tr>
<tr>
<td>Less than 60% from right ventricle</td>
<td>5</td>
</tr>
</tbody>
</table>

DISCUSSION

The primary aim of this paper is the presentation of the morphologic observations, but it may not be amiss to make some comments regarding the surgical therapy of pulmonary stenosis. It is to be stressed that the findings would probably be of greater significance had the tissues been fresh rather than fixed at the time of this study. It is true that we had seen most of the specimens previously when in an unfixed state at the time of autopsy but did not tabulate the different conditions observed. Furthermore, we must remember that these cases may not be representative of the entire group in that fatalities are more likely to occur, with or without operation, in those patients with the most serious malformations. As stated previously, 79 of the 95 specimens were obtained from patients who had been operated upon, the total of these being approximately 1,100. In other words, conclusions in regard to the choice of operative procedure might be different if the exact anatomic deformity in all of the cases was known.

Discussion of the operative procedures will be limited to the choice of (1) an anastomotic procedure in which an artificial ductus is created, or (2) a direct attack in which a stenotic valve or diaphragm is divided or an infun-
dibular resection is performed; details of the type of anastomosis or of the
direct attack will not be discussed. It should be understood at this time that
the treatment of valvular pulmonary stenosis with an intact ventricular septum
should consist of division of the stenotic valve. There is no difference of opinion
on this point.

![Diagram of heart with labels: Pulm. a., Crista supraventricularis, Septal defect, R. vent.]

Fig. 5.—Drawing to demonstrate tetralogy of Fallot with valvular pulmonic stenosis.
The domelike projection of the fused valve cusps into the pulmonary artery is illustrated. Note
that the infundibular area is slightly constricted.

There is considerable difference of opinion on the choice of treatment of
the various forms of pulmonary stenosis accompanied by an interventricular
defect. In these conditions the aorta receives mixed venous blood from the right
side of the heart as well as arterial blood from the left. We shall deal first with
those deformities in the treatment of which there seems to be the least difference
of opinion. The first of these is pulmonary atresia in which there is no direct
communication between the pulmonary artery and the outflow tract. Of our
autopsy specimens 23 per cent showed this type of malformation. The treat-
ment consists in an anastomosis between a pulmonary artery, if one is available,
and a systemic artery. A successful direct attack is usually an impossibility.
A second condition in the treatment of which there is not a great difference of
opinion is pulmonary valvular stenosis in association with an interventricular
defect; in other words, the tetralogy of Fallot with a valvular rather than an
infundibular stenosis. Eleven per cent of our autopsy specimens showed this
anomaly. It is our opinion that Brock is correct in advocating that this be
treated by a direct attack with division of the pulmonary valve, just as in cases
of "pure" valvular stenosis. Brock does qualify his view by stating that the
mortality rate associated with division of the valve is high in adults with this
deformity and that perhaps in such patients an anastomosis should be performed.
Dr. Taussig has been fearful that division of the valve when an interventricular
defect is present will convert the condition into an Eisenmenger complex with
too great blood flow to the lungs and pulmonary hypertension. We feel con-
ident that this will not happen because there is usually at least a little stenosis of the infundibular tract, and furthermore the stenotic valve leaflets remain somewhat fused at their bases. In brief, we agree with Brock that the proper treatment of pulmonary valvular stenosis in association with the tetralogy of Fallot should consist of division of the valve, with the possible exception of those instances in which the condition is encountered in adults.

![Diagram of heart with labels Pulm. a., Vent. defect, Inf. chamber, Crista supraventricularis](image)

**Fig. 8.**—This drawing illustrates the combination of pulmonary valvular and infundibular stenosis in tetralogy of Fallot. Obviously both areas of stenosis must be dealt with in direct surgical procedures.

Before considering infundibular stenosis unaccompanied by valvular stenosis, we shall consider the combination of the two which was found in 15 per cent of our autopsy specimens. If the major point of obstruction is in the valve, it should be treated by division of the valve. If both obstructions are severe, it seems, in view of the fact that we have much to learn about the best treatment of at least one of the conditions and probably both, that the treatment should consist of an anastomosis between pulmonary and systemic arteries.

The results reported here show that there is a wide variation in the anatomy of infundibular stenosis. In some cases the obstructed area is short and in others it is long. In some cases it is high in the right ventricle and in others it is low. Some idea may be obtained preoperatively about the size of the infundibular chamber by radiologic studies. Direct observation of the area at the time of operation plus the use of pressure recording devices will give one a good idea of the nature, size, and degree of the stenosis. Even so, direct observation of the obstructed area in a bloodless field is impossible at present, and one has to use a good deal of imagination in visualizing what is being done in a direct attack on the stenotic area.

We shall now comment on the advantages and disadvantages of the direct attack in the treatment of infundibular stenosis as compared with an anastomotic procedure between pulmonary and systemic arteries. Brock and Bailey, Glover,
and O'Neill in stressing the advantages of the direct attack have stated that it does not add the danger of another anomaly, such as is caused by the creation of an artificial ductus. There may be considerable truth in this point of view; but may it not be that the traumatized area of myocardium denuded of its endocardium which results from infundibular resection may in itself present as great a hazard as does an additional anomaly in the form of an artificial ductus? In this connection it must be said that the patient with an artificial ductus rarely has subacute bacterial endarteritis or heart failure provided the ductus is of moderate size. Broek's statement that a successful direct attack on infundibular stenosis results in a lessening of the shunt of blood from the right side of the heart to the aorta appears to us to be a more important consideration. When the oxygen content of aortic blood is considerably higher than that in the pulmonary artery, the blood which passes directly to the lungs from the right side of the heart will gain more oxygen per unit volume than that transmitted to the lungs from the aorta. In other words, a given amount of work by the heart will result in a more effective oxygenation of the blood if it passes directly from the right heart to the lungs than if part of it is shunted through an interventricular defect, is admixed with blood of a higher oxygen content in the aorta, and then part of the mixture is shunted to the lungs. On the other hand, there is little difference in the oxygen content of blood in the aorta and in the pulmonary artery in many cases of the tetralogy of Fallot. This is particularly likely to be the situation when the aorta arises mainly from the right ventricle.

The disadvantages of the direct attack in the treatment of infundibular stenosis appear to be numerous at the present time although it is likely that some of these will be overcome with additional experience and technical and diagnostic advances. In the first place, the immediate operative mortality rate is higher in association with the direct attack, at least in our experience. There may be greater sudden blood loss during the operation. The heart usually withstands operations on the large blood vessels at its base better than it tolerates incisions into and manipulations within its cavities. Because the field is obscured by blood, infundibular resection is in the main a blind procedure. The results reported in this paper show that the morphology varies greatly and it is impossible in many cases to determine the exact nature of the malformation; hence one may remove part of the stenotic tract only to leave a constricted area behind. If an error is made, it is more likely to be in the direction of removing too little rather than too much tissue. Attention has already been called to the dangers of thrombosis and embolism in association with resection of part of the interior of the heart in contact with the circulating blood. An attack upon a pulmonary valve which is devoid of muscle presents less hazard in this regard. Furthermore, the direct attack is accompanied by some danger of injury to the aortic valves and their attachments.

A consideration which has caused us concern is connected with the decision as to the advisability of opening the pericardium in all or most cases in which an operation is performed for suspected pulmonary stenosis. It is certainly true,
as Brock has emphasized, that the surgeon will increase his familiarity with the pathologic anatomy of this region if pericardial exploration is performed routinely. At the same time it must be admitted that this maneuver, with the associated examination of the area, lengthens the operating time and increases the postoperative complications.

We suspect that differences of opinion about the choice of operative procedures may be influenced somewhat by the ages of the patients with whom one is dealing. For example, we have the impression that our patients and those of Potts are of a younger average age than those of Brock and of Bailey. Certainly the heart of a child or an adult is more suitable for infundibular resection than that of an infant.

In concluding this discussion, we must agree with Brock that infundibular resection is theoretically preferable to the creation of an artificial ductus in the treatment of infundibular stenosis, but there are many patients in whom resection is impossible with the use of existing techniques and still others in whom it is inadvisable. If we may hazard a guess, we predict that for the next several years pulmonary atresia and high infundibular stenosis will be treated by the making of an artificial ductus, and that there will be an increase in the use of infundibular resection in the treatment of low infundibular stenosis with a large chamber. It is likely that the general adoption of infundibular resection even for this latter condition will not be rapid since the results with anastomosing procedures are in general very good and since most cardiologists interested in this field do not seem to view the direct attack with favor. The anatomic findings of Donzelot, d’Allaines, and associates in a recent study of 54 cases of tetralogy of Fallot and the conclusions drawn therefrom are similar to those reported in this paper.

SUMMARY

An anatomic study has been made of the autopsy specimens of the heart and great blood vessels of 95 patients who had pulmonic stenosis or atresia, accompanied by an interventricular defect. Cases of “pure” valvular stenosis with an intact ventricular septum were not included. An operation had been performed on 79 of the 95 patients in an attempt to relieve the disability and cyanosis. It is not claimed that these cases are representative of the condition of all of the approximately 1,100 patients upon whom such an operation was attempted, for it is apparent that the mortality rate is highest among those with the serious malformations.

The specimens were classified according to the anatomy of the pulmonary conus region. It became apparent that there is extreme variability in the bulbus cordis in the tetralogy of Fallot. The specimens may be divided into the following groups: (1) 23 per cent showed pulmonic atresia, (2) 10 per cent valvular pulmonary stenosis, (3) 15 per cent both infundibular stenosis and valvular stenosis, and (4) 52 per cent showed infundibular stenosis. This last group was subdivided as follows: (1) 37 per cent showed a low, well-developed infundibular chamber, (2) 37 per cent a high, small infundibular chamber, and (3) the chamber was intermediate in size and position in the remaining 26 per cent.
The type of operative procedure that should be employed in the treatment of these defects has been discussed in regard to choice between a direct attack and a shunting procedure. It is obvious that with the possible exception of adults a direct attack with division of the valve should be used in the treatment of stenosis of the valve and that an artificial ductus should be made in the treatment of pulmonary atresia. There is probably a justifiable difference of opinion regarding the choice of procedure in the treatment of infundibular stenosis. When resection techniques are further developed, it appears likely that low infundibular stenosis with a large chamber will be treated by a direct approach with infundibular resection, and that shunting procedures will continue to be used in most cases of high infundibular stenosis with a small chamber. The advantages and disadvantages of a direct attack and of a shunting procedure are discussed.

We are indebted to Drs. Bahnson, Sabiston, and Morrow, who aided in examining these specimens.

REFERENCES