Anomalous Muscle Bundle of the Right Ventricle
Hemodynamic Consequences and Surgical Considerations

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Anomalous muscle bundles in the right ventricle, although common, are not usually responsible for significant functional disturbances. The primary purpose of this report is to present seven cases in which anomalous muscle bundles of the right ventricle were responsible for confusing situations during corrective surgery. In six instances, anomalous muscle bundles of the right ventricle were associated with ventricular septal defect. In one of these six, the initial impression upon examining the interior of the heart at operation was that a ventricular septal defect existed at a site that was later determined to represent a space between anomalous muscle bundles. Closure of this space resulted in an altered route for the shunted blood but did not obliterate the interventricular communication.

In each of the other five of these cases, an anomalous muscle bundle was present in the inflow portion of the right ventricle just beyond the level of the ventricular septal defect. The combination of malformations produced a functional and clinical picture similar to that seen in the classical tetralogy of Fallot. In two of these patients failure to appreciate the nature of the obstruction resulted in inadequate surgical treatment and death. In the other three, accurate recognition of the nature of the obstructive lesion resulted in the appropriate surgical approach and complete correction of the cardiac abnormalities.

In addition to these six cases of anomalous bundles associated with ventricular septal defect, a seventh case represented an example of anomalous bundle with intact ventricular septum and pulmonary valvular stenosis. In this last patient, intracardiac pressure studies performed after surgical correction of the pulmonary valvular stenosis indicated that an obstructive zone existed within the right ventricle proximal to the infundibular area. During the operation the cause for this obstruction was not recognized, but at necropsy the obstruction was observed to have been caused by an anomalous muscle bundle in the inflow part of the right ventricle.

Since the details in each of these complicated cases are instructive, each case is presented individually. Particular attention is given to the anatomic detail and to the peculiar functional and surgical picture presented by the anomalous muscle bundle in each case.

Case Reports

The first six cases are presented in the order in which they were seen to illustrate the progressive steps taken in refining our definition of this anomaly and in diagnosing it in the individual patient.

Case 1

Large Ventricular Septal Defect. Space between Anomalous Muscle Bundle and Right Ventricle Wall Interpreted as the Defect

This 3-year-old boy, seen in 1956, showed clinical and hemodynamic findings compatible with those of an uncomplicated large ventricular septal defect without pulmonary stenosis (table 1). He died during the second attempt at surgical correction of this defect. The findings observed at necropsy are presented along with their hemodynamic and surgical consequences as retrospectively inferred.

The pertinent pathologic abnormalities were
Figure 1

Case 1. Left: The left ventricle (LV) has been opened. The ventricular septal defect (D) is located beneath the posterior aortic cusp. A = aorta; PT = pulmonary trunk; M = anterior leaflet of mitral valve. Right: Right ventricle and great vessels. The heart has been sectioned sagittally, and the lateral wall of the right ventricle removed. The ventricular septal defect (D) lies immediately under the aorta (A). The anomalous muscle mass, pyramidal in shape, lies within the right ventricle, its apex being attached to the annulus of the tricuspid valve (T) and the base of the pyramid attaches to the anterior wall of the right ventricle (2). Three channels within the right ventricle are related to this mass. Channel 1 (1), lying in front of that portion of the anomalous muscle mass which is between its apex and base, provides communication between the area of the tricuspid valve and the area of the ventricular septal defect. A second channel (2) lies behind the lower part of the base of the mass through which blood from the tricuspid valve could reach the outflow portion of the right ventricle. A third channel (3) lay behind the upper part of the base of the pyramid. This channel had been closed surgically by an Ivalon patch. In the natural state blood from the ventricular septal defect could flow to the pulmonary valve, either directly through channel 3 or indirectly through channel 1 and then channel 2. With the surgical closure of channel 3 at the first operation, channel 2 remained the only route by which blood from the tricuspid valve could reach the pulmonary valve. At the second surgical procedure, channel 2 was closed, thus isolating the outflow area of the right ventricle from the tricuspid valve and from the still open left ventricular septal defect.

confined to the cardiovascular system. The aorta and pulmonary artery were observed to be normally interrelated and of approximately equal size. A ventricular septal defect when viewed from the left ventricle lay immediately under the aortic valve. The posterior margin of the defect was formed by the junction of the anterior leaflet of the mitral valve with the septal leaflet of the tricuspid valve (fig. 1). The defect when viewed from the right ventricular aspect was seen to be behind and below the crista supraventricularis and in front of the tricuspid ring (fig. 1). Within the right ventricular cavity lay a large anomalous muscle mass, roughly pyramidal in shape, with a constricted waist just below its apex. This apex was attached to the anterior wall of the right ventricle anterior to the annulus of the tricuspid valve, while the
broad base of the mass was attached to the anterior right ventricular wall about midway between the right ventricular apex and the tricuspid ring.

The mass was related to three channels within the right ventricle. The most superficial extended in front of that part of the mass that was between its apex and base (channel 1). This channel allowed communication between the tricuspid orifice and the region of the ventricular septal defect.

The second channel lay behind the lower part of the base of the pyramid, and through it blood could flow from the tricuspid valve to the outflow tract of the right ventricle (channel 2). The third channel lay behind the upper part of the base of the pyramid; through this channel the right ventricular outflow tract communicated with the region of the ventricular septal defect (channel 3). Therefore, in the natural state, continuity existed between the third channel and the tricuspid orifice by way of the aforementioned first channel.

Moreover, in the natural state, blood being shunted into the right ventricle through the ventricular septal defect could reach the right ventricular outflow tract in two ways: directly, through channel 3 or indirectly through channel 1 and then through channel 2.

At the first operation, under conditions of extracorporeal circulation, the right ventricular outflow tract was exposed. The third channel, falsely interpreted as the ventricular septal defect was closed by the placement of an Ivalon patch. This procedure left the actual ventricular septal defect undisturbed, and the left-to-right shunt therefore continued via channel 1 and thence through channel 2 to the right ventricular outflow tract and on to the pulmonary trunk. It is noteworthy that when the heart was exposed at the first operation, a thrill was apparent over the right ventricular outflow tract and the pulmonary trunk. After the procedure was performed and circulation re-established, the thrill previously noted was found to be absent; but a thrill was now evident over the right ventricle immediately below the tricuspid ring. This change now is interpreted as follows: Before the intracardiac procedure had been performed, the principal route of the shunt was directly through channel 3. After this channel was closed, however, the indirect route was employed, and the thrill could have originated at the junction of channels 1 and 2 near the tricuspid valve.

In the postoperative period it was apparent clinically, and proved at catheterization, that a left-to-right shunt still existed and that pulmonary hypertension was unchanged (table 1). Therefore, 18 months after the first operation, the right ventricular outflow tract was re-opened. At this time, channel 2 was misinterpreted as a muscular septal defect and closed. Attempts to establish circulation after closure of the heart was unsuccessful.

Review at necropsy indicated that with both channels 3 and 2 closed, no route was left for the flow of blood from the tricuspid valve (and still present ventricular septal defect) to right ventricular outflow tract.

Case 2

Anomalous Muscle Bundle Right Ventricle and Ventricular Septal Defect. Functional Simulation of Tetralogy of Fallot

A 10-year-old boy was seen in January 1960, exhibiting clinical and hemodynamic findings compatible with those of classical tetralogy of Fallot (table 1). The patient was referred for operative correction of the malformation.

At operation, when the outflow portion of the right ventricle was exposed, the ventricular septal defect was readily identified and was closed by direct sutures tied over a thin Ivalon strip. An attempt to identify the obstructive lesion between the right ventricle and the pulmonary artery failed to uncover any such lesion. We therefore thought...
that the functional obstruction manifested clinically was possibly the result of infundibular contraction rather than of a distinct anatomic abnormality. Despite this belief, some infundibular resection was performed, and the ventriculotomy wound was closed.

After surgical intervention, a loud systolic murmur persisted in the pulmonary area. Intractable congestive cardiac failure developed, and the patient died 5 weeks after the operation. The pertinent pathologic findings were restricted to the cardiovascular system. The great vessels were observed to be normally interrelated but the finding of a wide pulmonary trunk was unusual in such a case. The diameter of the aorta was 2 cm., that of the pulmonary trunk 2.5 cm. The ventricular septal defect, measuring 1.5 cm. in diameter, had been closed. When viewed from the left ventricular aspect it was seen to lie in the basal portion of the ventricular septum under the right and posterior leaflets of the aortic valve (fig. 2). The right ventricular cavity contained an anomalous wedge-shaped band of muscle (fig. 3). The apex of this anomalous mass originated at the anterior aspect of the ventricular septum immediately above and in front of the ventricular septal defect. It crossed the right ventricular chamber to insert at the apical portion of the anterior wall of the right ventricle. In so doing, the mass divided the right ventricular cavity into two portions: The proximal was formed by the proximal part of the sinus of the right ventricle. The distal cavity was formed by the true outflow tract and part of the right ventricular sinus portion.

Two channels of communication existed between the two subdivisions of the right ventricular chamber. The first lay anterior to the anomalous muscle bundle and allowed communication between the area of the ventricular septal defect and subpulmonary region of the right ventricle. Blood from the tricuspid valve also could reach the area of the subpulmonary region of the right ventricle through this channel. The second channel of communication between the two subdivisions of the right ventricle lay posterior to the anomalous muscle bundle so as to allow communication between the inflow portion and the outflow portion of the right ventricle.

The infundibulum of the right ventricle showed evidence of minimal surgical resection, though no significant infundibular stenosis appeared to have existed in the natural state. The Ivalon strip, over which had been tied the sutures that closed the ventricular septal defect, encroached to a considerable degree upon the first of the two channels described. Thus the degree of obstruction between the two subdivisions of the right ventricle had

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**Table 1**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr.)</th>
<th>Pressure (mm. Hg)</th>
<th>Oxygen saturation (per cent)</th>
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<td>&quot;RV&quot;</td>
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<td>2</td>
<td>4½</td>
<td>90/0</td>
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<td>3</td>
<td>12½</td>
<td>150/0</td>
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<td>4</td>
<td>6</td>
<td>100/0</td>
<td>25/13</td>
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<tr>
<td>5</td>
<td>3</td>
<td>120/5</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>95/0</td>
<td>45/5</td>
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Preoperative

At operation

Before correction | 165/0 | 160/0 | 30/22 |
After correction | 85/4  | 65/0  | 35/20 |

*Before first operation.
†Before second operation.

SA, systemic artery; "RV", proximal portion of right ventricle; "Inf", distal portion of right ventricle; PT, pulmonary trunk; RA, right atrium; VC, vena cava.
actually been increased by the steps involved in closing the ventricular septal defect.

**Case 3**

*Anomalous Muscle Bundle Right Ventricle and Ventricular Septal Defect. Functional Simulation of Acyanotic Tetralogy of Fallot*

A 17-year-old boy seen in January 1960 had clinical findings compatible with the diagnosis of a cyanotic tetralogy of Fallot. Cardiac catheterization revealed, as it had 5 years earlier, a left-to-right shunt at the ventricular level, full saturation of femoral arterial blood, and a large difference in pressure between right ventricle and pulmonary trunk (table 1). Referred for corrective surgical intervention, the patient died 3½ months later following a stormy postoperative period complicated by congestive heart failure, oliguria, wound infection, and septicemia.

At necropsy, the great vessels were noted to be normally interrelated, and the pulmonary trunk and the pulmonary valve ring were of normal diameter; the pulmonary valve and infundibulum of the right ventricle also appeared normal. The right ventricle was incompletely partitioned into a proximal inflow subdivision and a distal outflow subdivision. This division was accomplished by very pronounced hypertrophy of the septal limb of the crista supraventricularis on the one hand and by presence of an anomalous muscular trabeculation along the anterior portion of the right ventricular wall on the other (fig. 4). A single stenotic opening lying between these two muscular prominences was the only communication between the inflow and outflow areas of the right ventricle.

Further evidence of the obstructive nature of these anomalous muscle bands could be seen in the thickness of the right ventricular wall. The more proximal portion of the right ventricular wall was 1.5 cm. thick, while the portion of the wall distal to the obstructive zone was 2 to 3 mm. thick. A ventricular septal defect measuring 1 cm. in diameter lay in the basal portion of the ventricular septum and communicated with the more proximal right ventricular subdivision.

During surgical intervention, when the right ventricle was opened, the pulmonary valve and infundibulum area of the right ventricle were thought to be normal. Considerable difficulty in identifying the site of the ventricular septal defect was encountered, but ultimately it was identified, and a woven Teflon patch was sutured in place. Since the pulmonary valve and outflow tract of the right ventricle were normal and the true nature of the obstructive muscle masses in the right ventricle was not then evident, the ventriculotomy wound was then closed.

Although this patient's postoperative course was complicated by many additional factors, it was apparent that the operation had failed to establish normal hemodynamics.

The anatomic configuration of the anomalous muscle mass causing obstruction of the right ventricle in this case was different from that seen in case 2. Here, instead of a muscle mass crossing the right ventricular chamber, a hypertrophied septal limb of the crista supraventricularis was opposed by a large muscular trabeculation on the
anterior wall of the right ventricle. The resulting communication between the inflow and outflow areas of the right ventricle was narrow and this was responsible for the obstruction in the right ventricular cavity.

Case 4

Anomalous Muscle Bundle Right Ventricle and Ventricular Septal Defect. Functional Simulation of Acyanotic Tetralogy of Fallot

This 7-year-old girl presented clinical findings compatible with those of acyanotic tetralogy of Fallot. This working diagnosis was supported by the findings of cardiac catheterization (table 1). In March 1960, the patient was referred for surgical correction of a ventricular septal defect and infundibular pulmonary stenosis.

At operation, after institution of extracorporeal circulation, the right ventricular outflow area was exposed. The pulmonary valve was seen to be normal, and the infundibular area of the right ventricle did not appear to have an obstructive zone. An opening, which was first assumed to be the ventricular septal defect, lay beneath the crista supraventricularis. Further exploration did not indicate the location of the tricuspid valve. In probing through what appeared to be the ventricular septal defect, it was observed that a probe could be passed in a posteroinferior direction into the right ventricle and also in a posterolateral direction into the right atrium. Only then was it apparent that the “ventricular septal defect” was, in reality, the posterior of two communications between the inflow and outflow portions of the right ventricle. The semipartitioning of the right ventricle was identified as resulting from the presence of an anomalous muscle mass initially misinterpreted as being the ventricular septum. The aforementioned communication lay behind the mass, while a second and smaller communication lay in front of the mass.

Division of the anomalous muscle mass was accomplished by placing one blade of a pair of scissors into each of the two communications and cutting. Upon completion of this procedure, the tricuspid orifice was identified and, in addition, the true ventricular septal defect was now exposed at the base of the ventricular septum under the septal leaflet of the tricuspid valve. The ventricular septal defect was closed by means of a Teflon patch.

Attention was then directed to the anomalous muscle mass. Upon removing as much of this as seemed prudent, it was noted that residual obstruction persisted between the inflow and the outflow portions of the right ventricle. Accordingly, a Teflon patch was placed in the anterior wall of the right ventricle to bridge the ventriculotomy wound and to effect complete relief from the residual obstruction. After re-establishment of natural circulation, the heart responded effectively, and the patient had an uneventful postoperative course. Follow-up study of the patient 6 months after operation showed her to be free of symptoms.

The consequence of the anomalous muscle mass in the right ventricle of this patient was similar to that in case 2, that is, obstruction of blood flow between the inflow and outflow portions of the right ventricle. The two factors that led the surgeon to identify and to assess the functional significance of muscular abnormality in case 4 were (1) the absence of the type of infundibular stenosis usually seen in the tetralogy of Fallot and (2) the absence from view of the tricuspid valve when the outflow portion of the right ventricle was exposed.

Case 5

Anomalous Muscle Bundle Right Ventricle and Ventricular Septal Defect. Functional Simulation of Tetralogy of Fallot

In a 4-year-old boy with clinical and hemodynamic findings diagnostic of cyanotic tetralogy of Fallot (table 1), a corrective surgical procedure was advised in May 1961.

At operation after institution of extracorporeal circulation, the outflow portion of the right ventricle was exposed. The pulmonary valve and infundibulum appeared normal. It became immediately apparent that the anatomic situation was different from that in tetralogy of Fallot. Moreover, the anatomic findings of this case seemed very similar to those in cases 2 and 4. That is, an anomalous muscle mass was observed to cross the right ventricle, obscuring the tricuspid valve from view and causing obstruction between the inflow and outflow portions of the right ventricle. As a result of this anomalous mass, two restricted openings existed between the two subdivisions of the right ventricle.

Incision of the anomalous muscle mass as in case 4 exposed the entire right ventricular chamber, the tricuspid valve, and the ventricular septal defect. A considerable portion of the anomalous muscle mass was then resected, particularly along the ventricular septum and at the attachment at the anterolateral wall of the right ventricle, resulting in complete relief of the obstruction between the inflow and outflow portions of the right ventricle. The ventricular septal defect, located basally posterior to the crista supraventricularis, was closed with interrupted mattress sutures placed parallel to the crista supraventricularis.

Upon completion of the operative procedure and re-establishment of circulation, cardiae function was fully restored. The patient was discharged from the hospital in May 1962.
tion was observed to be good. The postoperative period was uneventful, and the patient was free of symptoms on a follow-up examination.

In this patient, as in case 4, recognition of the true nature of the obstruction was aided by the surgeon’s inability to locate the tricuspid orifice from the right ventriculotomy wound and by the observation that the nature of the right ventricular infundibulum was not sufficient to explain the clinical and hemodynamic findings. Correction of the obstruction resulted from excision of the anomalous muscle mass.

Case 6

Anomalous Muscle Bundle Right Ventricle and Ventricular Septal Defect. Functional Simulation of Atracnic Tetralogy of Fallot

Clinically, this 10-year-old girl seen in May 1961 appeared to represent a case of cyanotic tetralogy of Fallot, a diagnosis that was supported by cardiac catheterization (table 1).

In order to determine the precise nature of the right ventricular obstruction, a selective right ventriculogram was performed. Twenty cubic centimeters of diatrizoic acid (Hypaque-M) were injected into the right ventricle, while films were taken in two planes at the rate of 5 per second for 6 seconds. Immediately following injection, dense opacification appeared in the inflow portion of the right ventricle, and subsequently in the outflow portion of the right ventricle, the pulmonary valve and an enlarged pulmonary trunk. A filling defect was observed in the right ventricle, extending basally from the area of the crista supraventricularis to the anterior wall of the right ventricle near the apex. This filling defect, constant in all subsequent films (fig. 5), strongly suggested by its shape and position the presence of an anomalous muscle bundle in the right ventricle similar to those previously seen in cases 2, 4, and 5.

The patient was referred for a corrective operative procedure. During extracorporeal circulation, opening the outflow portion of the right ventricle revealed a normal infundibular area and pulmonary valve. The tricuspid valve could not be identified from the exposed right ventricular chamber, and an anomalous mass of muscle was found in the right ventricle. From its superior attachment above the ventricular septal defect, the mass extended inferiorly to insert into the anterior wall of the right ventricle near the apex. In this way, the mass divided the right ventricle into distinct inflow and outflow subdivisions. The only sites of communication between these two chambers lay (1) anterior and (2) posterior to the anomalous muscle mass. When the mass was divided, as in cases 4 and 5, the tricuspid valve and ventricular septal defect were readily exposed. The anomalous mass was completely resected, thus removing the obstruction between the inflow and outflow portions of the right ventricle.

Attention was then directed to the ventricular septal defect, 3 cm. in diameter, which lay immediately anterior to the tricuspid ring. This opening was closed by means of interrupted sutures tied over a strip of Ivalon. Then the ventriculotomy incision was closed and extracorporeal circulation was terminated.

The postoperative course was uneventful, and...
the patient was apparently healthy when discharged from the hospital.

The right ventricular filling defect consistently observed in the angiocardiograms, suggested the presence of an anomalous muscle mass as observed in cases 2, 4, and 5, and led to a preoperative suspicion that the disorder might not represent a usual example of pulmonary stenosis and ventricular septal defect.

Case 7

Pulmonary Valvular Stenosis, Intact Ventricular Septum, and Anomalous Muscle Bundle in the Right Ventricle Causing Obstruction to Pulmonary Outflow

This 3½-year-old patient underwent an operative procedure in 1958, prior to observation of five patients already described. Since, in retrospect, findings in this case suggest the correct diagnosis, it may be instructive to review the case. The patient showed clinical, hemodynamic, and angiocardiographic evidence of pulmonary valvular stenosis, intact ventricular septum, atrial septal defect with right-to-left shunt and patent ductus arteriosus. Referred for surgical correction of these defects, she died 12 hours after the surgical procedure.

At operation, after ligation and division of the patent ductus arteriosus, extracorporeal circulation was instituted. The pulmonary artery was opened and the valvotomy performed under direct vision. The three raphe being incised out to the annulus of the pulmonary valve. The infundibular area of the right ventricle was then digitally explored through the pulmonary valve and found to be normal to a point below the crista supraventricularis. The right atrium was then opened and the atrial septal defect closed. Exploration indicated that the tricuspid valve was normal. Following closure of the heart and re-establishment of normal circulation, pressures were 85 mm. Hg systolic and 4 diastolic in the inflow area of the right ventricle: 65 mm. Hg systolic and 0 diastolic in the outflow area of the right ventricle; and 32 mm. Hg systolic and 20 diastolic in the pulmonary trunk. This difference in pressure between inflow and outflow portions of the right ventricle suggested that a residual obstruction existed. On the basis of two findings, however—the observed tight valvular stenosis (which had been relieved) and the negative results of exploration of the infundibular area of the right ventricle—the surgeon wrongly concluded that the residual obstruction was functional.

An additional factor, which had not been appreciated in the preoperative evaluation, was the demonstration by right ventriculography of a large filling defect between the inflow and outflow areas of the right ventricle that undoubtedly represented the anomalous muscle mass (fig. 6).

Pathologic examination revealed the aorta and pulmonary artery to be normally interrelated and of approximately equal size. A large patent ductus arteriosus had been ligated and divided. The area of the pulmonary valvotomy revealed that the pulmonary valvular stenosis had been adequately relieved surgically by incision along the rudimentary commissures, and three distinct though thickened valve elements resulted. A small atrial

Figure 5

Case 6. Lateral view of right ventriculogram, one-half second after injection. Opaque material is particularly concentrated in the inflow portion of the right ventricle (I). The outflow portion of the right ventricle (O), the area of the pulmonary valve (P), and the pulmonary trunk (PT) are also opacified. A filling defect (arrows) in the right ventricular chamber is seen extending from the crista supraventricularis (C) in an anteroinferior direction to the anterior wall of the right ventricle. The filling defect remained unchanged in subsequent films of this study. The size and location of this filling defect in the right ventricular chamber are compatible with the size and location of the anomalous muscle bundle later observed and excised at operation.
Case 7. Selective right ventriculograms. Left upper: Anteroposterior view one-half second after injection was made into the inflow portion of the right ventricle (I), during diastole. Opacification of this region, of the outflow portion of the right ventricle (O) and of the pulmonary trunk (PT) has occurred. A filling defect is seen in the midportion of the right ventricle (M). The routes of flow of opaque material are seen to be between the crista supraventricularis (C) and the muscle mass, and also between the mass and the apex of the right ventricle. Right upper: Lateral view at the same time as left upper. Stenotic pulmonary valve (PV) is illustrated. Left lower: Anteroposterior view one-fifth second after left upper and right upper, during systole. The filling defect has remained unchanged. Opacification of the channel between the anomalous mass and the apex of the right ventricle does not occur during this part of the cardiac cycle, leaving the channel between the mass and the crista supraventricularis as the only evident communication between the inflow and outflow portions of the right ventricle. Right lower: Lateral view at the same time as left lower. The small diameter of the communication between inflow and outflow portions of the right ventricle is apparent in this view.
septal defect at the fossa ovalis area had been closed with three mattress sutures.

In the right ventricle, a thick muscle band, wedge-shaped, extended from the base of the ventricular septum to the midportion of the anterior wall of the right ventricle, thus dividing the ventricular cavity into an inflow zone and an outflow area (fig. 7). No obstruction was noted in the infundibular area of the right ventricle. However, the only avenues of communication between the inflow and the outflow portions of the right ventricular cavity were two small, slit-like orifices, one posterior to the anomalous muscle band, and the other anterior to it.

Discussion

In cases of tetralogy of Fallot and in pulmonary stenosis with intact ventricular septum, hypertrophied muscle bundles are often present in the right ventricular infundibulum. These masses which obstruct blood flow protrude from the walls of the infundibulum but do not cross the cavity from one wall to another.

The muscle bundles of the right ventricle described in this report are different from those just mentioned. In the present case, the
anomalous muscle bundles cross the cavity of the right ventricle and lie proximal to the infundibulum. The usual site of occurrence is in the distal or apical part of the inflow or sinus portion of the right ventricle.

These muscle bundles have a different orientation from that of the moderator band. The latter structure, when present in the human heart, has its septal attachment at the apical third of the ventricular septum; in contrast, the anomalous muscle bundles, which are the subject of this report, have their septal attachments basally, near the tricuspid ring.

Both types of muscle bundle, moderator and anomalous, attach parietally to the anterior wall of the right ventricle. Thus, although the moderator band crosses the right ventricular cavity, it lies toward the septal side of the cavity, out of the main stream of blood flow, and does not ordinarily represent an obstructive factor. The anomalous muscle bundles here described cross the main channel of the right ventricle and may result in obstruction, since their position places them in the main stream of blood from tricuspid valve to pulmonary valve.

In regard to precedents in the literature, we can find no parallel for our findings in case 1. The phenomenon of a muscle bundle obstructing the right ventricle, which was characteristic of our remaining six cases, however, appears to have been duplicated in the seven cases of Tsifutis, Hartmann, and Arvidson. The patients described by those authors had anomalous muscular bands that divided the right ventricle into proximal and distal chambers. The proximal chamber was a high-pressure compartment lying between the tricuspid valve proximally and the anomalous mass distally. The distal chamber lay beyond the obstructing muscle bundle. In five of the seven cases reported by Tsifutis, ventricular septal defects and left-to-right shunts were associated with the right ventricular obstructive anomalies. The diagnosis of an aberrant obstructive lesion of the right ventricle has been made by cardiac catheterization and selective angiography. In three patients successful correction was accomplished by closure of the ventricular septal defect and resection of the hypertrophied muscular bands.

The older literature contains suggestive but not conclusive reference to the occurrence of anomalous right ventricular muscle bundles. Several cases of "triventricular heart" are cited by Peacock. These, on review of the anatomic description, appear to have been the usual type of tetralogy of Fallot, the infundibular area in these cases having been described as a "third ventricle." It is perhaps because no distinction had been made between the infundibular chamber in tetralogy of Fallot and the type of division of the right ventricle in the anomaly under discussion that cases of the type here presented have not been emphasized in the literature.

When an anomalous muscle bundle exists in the right ventricle, the right ventricular chamber is subdivided as follows: The proximal subdivision consists of the proximal portion of the sinus of the right ventricle, while the more distal subdivision consists of the more apical portion of the sinus of the right ventricle and the infundibulum of the right ventricle.

The anomalous muscle mass in the right ventricle may not produce any significant degree of obstruction to right ventricular outflow, as in case 1. Under this circumstance, the only surgical problem it represents is the difficulty of properly identifying the true nature of the anomalous muscle mass and of differentiating the channels around it from the ventricular septal defect.

On the other hand, as in the other cases here presented, the anomalous muscle mass may produce varying degrees of obstruction within the right ventricle. Under this circumstance, failure to identify this source of obstruction, as in case 2, 3, and 7, precludes completely restoring the hemodynamics to normal.

In our experience a ventricular septal defect is associated commonly, but not universally, with obstructive muscle masses in the right ventricle. When this association exists, the patient will present with one of two clin-
ical syndromes. Those cases of anomalous right ventricular muscle bundles and associated ventricular septal defect clinically resemble the tetralogy of Fallot, if an obstructive factor is present; if no obstruction exists, they resemble isolated ventricular septal defect. As a consequence, differentiation of the patient with anomalous muscle bundle from the patient with the more common types of anomalies named is not possible by clinical, electrocardiographic, or routine roentgenographic means. Cardiac catheterization may be helpful when an obstruction is present. In those six patients in whom the anomalous muscle bundle was responsible for obstruction, a difference in blood pressure was observed between the inflow area of the right ventricle and the infundibulum and pulmonary trunk. Unfortunately, no note was made during catheterization of the specific point in the right ventricle at which evidence of obstruction was encountered; nor can this factor be appropriately evaluated retrospectively. We would anticipate, however, that the obstructive zone could be located more precisely if the operator were alerted to the possibility of a more proximal obstruction in the right ventricle, such as that caused by an anomalous muscle mass.

Angiocardiography alone may be more helpful than cardiac catheterization, but it is most useful in conjunction with cardiac catheterization. Each of the two patients on whom angiocardiographic studies were performed gave evidence of an unusual filling defect in the right ventricle at a position corresponding anatomically to that of the anomalous muscle mass. In one of these cases, this angiocardiographic finding led to a presumptive diagnosis of an anomalous muscle bundle in the right ventricle, and alerted the surgeon to this potential source of obstruction to right ventricular outflow. But even if angiocardiographic procedures were carried out routinely in all cases of obstruction to pulmonary outflow or suspected ventricular septal defect, the presence of an anomalous muscle bundle might not always be apparent. This being so, a large share of the burden of identifying and appreciating the consequences of the anomalous muscle mass in the right ventricle falls on the surgeon at the time of operation.

An evaluation of the cases presented suggests several clues that may help the surgeon in this diagnostic task: First, on opening the outflow area of the right ventricle in a patient with this anomaly, the surgeon finds that the tricuspid orifice cannot readily be identified. This finding indicates that further exploration and evaluation are necessary, since what appears to be the ventricular septal defect under these circumstances may in fact represent a communication within the right ventricle associated with an anomalous right ventricular muscle mass.

Second, if the degree of obstruction present in the infundibular area seems inadequate to account for the clinical and hemodynamic findings, one should consider the possibility that an obstructive lesion exists more proximal to the infundibulum—an obstruction that may be due to an anomalous muscle bundle. Appreciation of the significance of these two findings led to the proper evaluation and therapy in cases 4, 5, and 6.

If the preoperative thrill persists after correction of the ventricular septal defect, or if it disappears but is succeeded by a definite thrill in a different location, the surgeon should consider the possibility that (1) the residual obstruction to blood flow remains (case 2), or (2) the route of blood flow has been altered but the primary hemodynamic abnormality has not been corrected (case 1).

Finally, if after completion of the surgical procedure no valvular or infundibular stenosis remains, a significant pressure differential between the inflow and the outflow portions of the right ventricle suggests that an anomalous muscle bundle may be causing this obstruction (cf. the postoperative pressure recordings in case 7).

**Summary**

Seven examples of anomalous muscle bundles of the right ventricle have been presented.

In one patient having an associated ventricular septal defect, spaces between the
ANOMALOUS MUSCLE BUNDLE OF RIGHT VENTRICLE

The Early History of Instrumental Precision in Medicine

From the days of Elizabeth every man of fashion, and especially the English, traveled in Italy. Here, too, wandered all who studied, or were fond of science, and it was to Padua—which Santorius called the Garden of Science—that Bacon came, and Drebbl, and Fludd, and the greater Harvey. The towns of Italy were exchanges of Europe both for commerce and for science. From them men took home what facts they saw or heard, and merely describing them (as did Bacon the air thermometer), left the future critic to settle the question of originality. The temper of the time was not that of our day. Men worked along patiently. There were no journals; the letter or the lecture were the only means of early publication. The genius who today invents a new forceps . . . yearns for instant type, and defends his offspring with virulence. Harvey knew of his great discovery in 1616, and it got into print in 1628. His lecture notes show that long before this date he was certain of the matter and clearly knew what he had done.—S. WEIR MITCHELL, M.D., Transactions of the Congress of American Physicians and Surgeons, Second Triennial Session held at Washington, D.C., 1891. New Haven, The Congress, 1892, p. 168.
Anomalous Muscle Bundle of the Right Ventricle: Hemodynamic Consequences and Surgical Considerations

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