Taussig-Bing Complex with Pulmonary Stenosis*

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In 1949, Taussig and Bing1 described a heart clinically and pathologically in which the aorta emerged completely from the right ventricle, while the pulmonary trunk straddled the ventricular septum over a defect in the septum, but emerged mostly from the right ventricle. This became known as the Taussig-Bing heart.2 Subsequently, hearts were reported in which similar relationships between the arterial trunks and the defect were present, but the pulmonary trunk emerged mostly from the left ventricle.3,4 These were also considered Taussig-Bing hearts by some authors. Recently, Lev et al.5 reviewed the literature of this spectrum of anomalies and reported 25 of 41 cases seen at the Congenital Heart Disease Research and Training Center. They suggested that the concept of the Taussig-Bing heart be: origin of the aorta completely from the right ventricle, unrelated to the ventricular septal defect, with the pulmonary trunk related to the defect but emerging partially or completely from the right ventricle. These hearts were to be differentiated from partial transposition or double outlet right ventricle in which the aorta was related to the defect. With this as the basic concept, the Taussig-Bing hearts could be classified as right-sided without overriding pulmonary trunk, where the pulmonary trunk emerged completely from the right ventricle, right-sided with overriding pulmonary trunk, where the pulmonary trunk arose mostly from the right ventricle, intermediate type in which the pulmonary trunk arose about evenly from both ventricles, and a left-sided type in which the pulmonary trunk arose mostly from the left ventricle.

Among the 41 Taussig-Bing hearts studied by the above group, there were three such hearts with pulmonary tract stenosis. Pulmonary tract stenosis in the Taussig-Bing heart had been previously alluded to by several workers.5,6 The present work is

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a pathologic study of these three hearts with a clinico-pathologic report of one of these cases.

CASE REPORTS

CASE 1
A 25-month-old white boy was born of a 19-year-old, gravid III, white woman. The prenatal, birth and family history showed no relevant data. Cyanosis and a grade III systolic murmur were present at birth. The cyanosis remained constant, was accentuated by crying and exercise, and progressively increased with age.

At the age of six weeks, he had his first anoxic spell while nursing, at which time the hematocrit was 51 per cent. Anoxic spells recurad sporadically when feeding. He tired easily when crying or nursing, and frequently lay with knees drawn under his abdomen.

On admission to Bob's Robert's Hospital at the age of 21 months, the child was underdeveloped and poorly nourished; his weight was 8.5 kg. and his length was 31½ inches, placing him below the third percentile for his age. He could not walk. The anterior fontanelle was still open and the head appeared large relative to the size of the body. Diffuse cyanosis with clubbing of the fingers and toes was present. The veins of the scalp, neck and upper limbs were unusually prominent but not distended. Marked carotid pulsations were clearly visible. A faint systolic murmur was diffusely heard over the scalp. Examination of the chest revealed a precordial bulge; a prominent substernal and epigastric lift was present. The first and second sounds were felt at the left sternal border. No apical impulse was palpable. There was a systolic thrill over the entire precordium. On auscultation, the first sound was clearly audible, and the second sound was single, extremely loud and maximally heard parasternally at the third left interspace. A grade III pansystolic murmur, maximal at the third interspace, with diffuse radiation, was present. The liver was palpable two fingerbreadths below the right costal margin. The heart rate was 144 beats per minute, the respiratory rate was 34 per minute, and the blood pressure 100/60 on the right arm, and 120/80 on the left leg.

The chest films (Fig. 1) showed the heart to be normally placed and grossly enlarged with an egg-shaped configuration. The apex was upturned. The vascular pedicle was very broad. The lungs were clear.

The electrocardiogram (Fig. 2) showed marked right ventricular hypertrophy with extreme right axis deviation. The P wave was of the congenital type.

At the age of 21 months, right heart catheterization was done (Table 1) via the right saphenous vein. The course of the catheter demonstrated the presence of an anomalous inferior vena cava with azygos continuation. The pres-

![Figure 1: Roentgenogram of chest, posteroanterior view.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21436/ on 06/27/2017)
TAUSIG-BING COMPLEX WITH PULMONARY STENOSIS

Table 1—Cardiac Catheterization (May 23, 1961)

<table>
<thead>
<tr>
<th>Site</th>
<th>Pressure (mm.Hg) (Mean in brackets)</th>
<th>Oxygen saturation (per cent)</th>
<th>Oxygen content ml. per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending lumbar</td>
<td></td>
<td>31.9</td>
<td>5.24</td>
</tr>
<tr>
<td>Azygos vein</td>
<td>7/6 (5)</td>
<td>35.8</td>
<td>5.86</td>
</tr>
<tr>
<td>Innominate vein</td>
<td>(5)</td>
<td>35.7</td>
<td>5.87</td>
</tr>
<tr>
<td>SVC</td>
<td>(5)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Right atrium</td>
<td>(10)*</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>79/0 to 10*</td>
<td>57.8</td>
<td>9.49</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>(not entered)**</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>82/0 to 13</td>
<td>63.3</td>
<td>10.4</td>
</tr>
<tr>
<td>Right femoral artery</td>
<td>84/59</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Capacity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematocrit</td>
<td>55</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Pullback at the end of the study.
**Pulmonary artery pressure at surgery 15/10 (12) mm.Hg
Left atrium and right atrium being (6) mm.Hg (9/19/61).

Sures in both ventricles were at systemic levels. The oxygen samples were suggestive of a large bidirectional shunt at the ventricular level. Mixing at the atrial level could not be evaluated. The pulmonary trunk and the aorta were not entered.

Biplanar angiography was performed using the Elema-Schminder rapid-film changer. Injections were made into the azygos vein and the left ventricle. The left ventricle injection showed, in the lateral view (Fig. 3), the dorsally placed pulmonary trunk filling directly from the left ventricle and probably overriding a septal defect. Its origin was from the right side of the septum and was mainly from the right ventricle. A dilated aorta emerged entirely from the anterior portion of the ventrally situated right ventricle, in the position normally occupied by the pulmonary trunk. Both vessels followed a parallel course, the ascending aorta lying just beneath the sternum and joining the descending aorta in a wide curve. The origin of the pulmonary trunk showed an infundibular narrowing. Some degree of post-stenotic dilatation was seen. The right ventricle appeared dilated and the left ventricle small. The injection into the azygos vein (Fig. 4) demonstrated its continuity with the inferior vena cava. The right atrium was enlarged. Retrograde

![Figure 2: Electrocardiogram. Sinus rhythm. Heart rate: 120 beats/min. P wave peaked in lead I and notched in lead III. Axis of P: +45°. Duration of P in lead I: 0.09 sec. PR interval: 0.14 sec. QRS axis: -170°; QRS duration: 0.08 sec.; T axis: +45°; QT interval 0.2 sec. (Normal for the heart rate). Precordial leads show complexes qR in VR4-VR6 and (q)RS in V6, with slurred "plateau" on top of the R wave. Intrinsicoid deflection: in V6, 0.045 sec. S wave larger than R in V6.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21436/ on 06/27/2017)
filling of the hepatic veins was seen, but reflux of contrast media into the inferior vena cava was absent. The aorta and pulmonary trunk filled simultaneously from the right ventricle with opacification.

At 23 months of age, he was submitted to surgery at which time the pulmonary trunk pressure was 15/10 mmHg, with a mean of 12 mmHg. Pressures in the right and left atria were 6 mmHg. An atrial septal defect of 2-3 mm. diameter was found. Pulmonary and aortic blood oxygen saturations were equal. A right subclavian-pulmonary artery anastomosis was performed and the atrial communication was enlarged. A complete heart block appeared when clamping the pulmonary trunk. He died in the operating room in severe acidosis despite the insertion of a pacemaker and vigorous fluid and electrolyte therapy.

**Necropsy Findings:**

*Heart:* The heart was enlarged, weighing 106 gm. (Fig. 5). A large aorta was situated anteriorly and to the right, and a minute pulmonary trunk posteriorly and to the left.

The right atrium was greatly enlarged and its wall was thickened. The hepatic veins entered in place of the inferior vena cava. The superior vena cava received a large azygos vein, which took the place of the normal inferior vena cava. The atrial septum in the region of the fossa ovalis had been opened by the surgeon, so that there was a defect measuring 1.5 cm. in greatest dimension. This defect involved mostly the fossa ovalis. The tricuspid orifice was enlarged, and the tricuspid valve was abnormally formed. Although there were three leaflets, accessory valvular tissue was present adjacent to the medial part of the anterior leaflet. This lay adjacent to the pulmonary conus area to be described.

The right ventricle was enlarged, and its wall was thickened (Fig. 5). Two powerful septal bands proceeded up to the base of the heart. The upper one met the first parietal band, which curved around to meet the second septal band on the left lateral margin. Thus these structures formed a loop surrounding one mouth of the pulmonary conus to be described. The second parietal band proceeded close to the anterior

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**Figure 3A**

*Angiocardiogram. Left ventricular injection. A) and B). Simultaneous anteroposterior and lateral views showing the filling of the left ventricle and pulmonary artery. (For comments see text.)*

**Figure 3B**
leaflet of the tricuspid valve. The endocardium of the right ventricle was focally whitened. From this chamber emerged both the aorta and the pulmonary trunk. The aorta made its exit between the two parietal bands. Its orifice was enlarged, its valve was normally formed, but it presented increased hemodynamic change. The coronary ostia lay in the posterior and left anterior sinuses of Valsalva. The right subclavian artery had been surgically anastomosed with the peripheral portion of the right pulmonary artery. The pulmonary trunk arose from a markedly narrowed conus. The latter had two mouths, one enclosed by musculature as described above, and one below the origin of the first parietal band. Both mouths faced the ventricular septal defect. The pulmonary orifice was markedly decreased in size, and its valve was bicuspid and thickened. The pulmonary trunk was small and its wall was thinned. The ductus arteriosus was moderately patent.

The left atrium was slightly smaller than normal and its wall was thinned. The mitral orifice was small and its valve was abnormally formed. At the anterior commissure both leaflets were attached by chordae to the septum and to a very small atypical papillary muscle on the septum. At the posterior commissure they were attached to a group of lateral papillary muscles. Separate muscle bands on the posterolateral aspect entered to the base of the inferior leaflet which was otherwise normal. The anterior leaflet, however, was divided into two parts. The inferior portion was connected to the lateral group of papillary

**Figure 3C**

**Figure 3D**

**Figure 3C and D:** Same injection 2 sec. later. (For comments see text.)
muscles and to the lower part of the septal band in the region of the defect. The superior portion was connected to a septal papillary muscle and directly to the septum in the region of the defect.

The left ventricle was normal in size, but its wall was thinner than normal. The ventricular septum at its base presented a defect measuring about 0.4 cm. in its greatest diameter. This defect was situated in the region of the pars membranacea and the muscular septum posterior to it. The annuli of the mitral and tricuspid valves were contiguous in the region of the defect and the pulmonary annulus was related to the mitral annulus. The embryologic right coronary artery arose from the left anterior sinus of Valsalva. It gave off the conal artery, the anterior descending and a short circumflex which ended on the anterior wall of the right ventricle. The embryologic left coronary artery emerged from the posterior sinus of Valsalva. It gave off branches to the anterior and posterior wall of the right ventricle, formed the posterior descending and terminated at the ramus obesus.

The anatomic diagnoses were: atypical Tausig-Bing complex with pulmonary tract stenosis, I. hypertrophy and enlargement of the heart, (a) right atrial hypertrophy and enlargement, (b) right ventricular hypertrophy and enlargement, (c) left atrial and left ventricular atrophy; 2. abnormal mitral and tricuspid valves; 3. ventricular septal defect; 4. absence of the inferior vena cava; 5. patent ductus arteriosus; 6: bicus-

**Figure 4:** Angiocardiogram. Contrast injected into the azygos vein A) posteroanterior view; B) lateral view (For comments see text.)
pulmonic valve; 7. surgical intervention (right Blalock and Blalock-Hanlon).

Case 2

Girl, seven days old (Fig. 6).

Heart: The heart was slightly enlarged, weighing 18.5 gm. A larger aorta was situated anteriorly and to the right, and a smaller pulmonary trunk posteriorly and to the left.

The right atrium was enlarged, and its wall was thickened. The foramen ovale was widely patent, measuring about 0.6 cm. in greatest dimension.

The right ventricle was enlarged, but its wall was normal in thickness. The architecture of the muscle bundles of the right ventricle was as follows: The septal band proceeded to the base in the usual manner. Here it met the first parietal band forming a loop which passed over the left side of the anterior wall to meet the septal band. In this way it produced a slit-like opening for the pulmonary conus. A second parietal band proceeded in the usual way close to the anterior leaflet of the tricuspid valve. From the right ventricle emerged both the aorta and the pulmonary

Figure 5A

Figure 5B

Figure 5: Case 1. (A) Right ventricular view S—Septal band; P1—First parietal band; P2—Second parietal band; O—One opening of pulmonary conus; D—Ventricular septal defect; A—Outflow tract into aorta; T—Tricuspid valve. Arrow points to rod which has been inserted into pulmonary conus. (B) Left atrial and left ventricular view: L—Lower part of anterior leaflet of mitral valve; U—Upper part of anterior leaflet of mitral valve; PP—Posterior group of papillary muscles; AP—Small anterior papillary muscle; ASD—Atrial septal defect after surgery.
the aorta was the vessel situated anteriorly and to the right. It was unrelated to the ventricular septal defect to be described. Its orifice was larger than normal, but its valve was normally formed. The coronary arteries emerged from the posterior sinuses of Valsalva.

The outflow tract of the pulmonary trunk was narrowed, being sandwiched in between the septal and first parietal bands. This tract was contiguous to the ventricular septal defect to be described. An accessory piece of what may be considered tricuspid valvular tissue formed a curtain hanging from beneath the pulmonary trunk. This curtain formed an aneurysmal pocket and was contiguous with the mitral valve. At the level of the valve, the pulmonary tract widened out, but the pulmonary orifice was still smaller than normal. The pulmonary valve was normally formed, but presented considerably increased hemodynamic change. The ductus arteriosus was not present on the specimen.

The left atrium was enlarged, but its wall was of average thickness. The mitral orifice was somewhat enlarged. Its valve was abnormally formed. The inferior leaflet was normal. The anterior leaflet was divided into two parts. The inferior portion of the anterior leaflet was related to the defect and was anchored to the inferior papillary muscle and by chordae to the margins of the ventricular septal defect. The anterior portion of the anterior leaflet was anchored to the anterior papillary muscle and by chordae to the septum.

The left ventricle was probably normal in size, and its wall was of average thickness. The ventricular septum presented two defects. One was situated in the apical region and measured about 2 cm. in greatest dimension. It opened into the right ventricle in the apical region of the sinus. The other was situated at the base measuring .5 cm. in greatest dimension and opened into the right ventricle beneath the pulmonary trunk.

The coronary artery distribution was identical with that of the previous case.

The anatomic diagnoses were: Taussig-Bing complex with pulmonary stenosis. 1. right atrial hypertrophy and enlargement; 2. right ventricular hypertrophy and enlargement; 3. patent foramen ovale; 4. cleft aortic leaflet of the mitral valve; 5. double ventricular septal defects.

**CASE 3**

Boy, three-months-old.

**Heart:** The heart was enlarged and weighed 43 grams (with aorta and pulmonary tree). A larger aorta was situated anteriorly and to the right and a smaller pulmonary trunk posteriorly and to the left.

The right atrium was enlarged, and its wall was thickened. The coronary sinus received a small left superior vena cava. The limbus was deficient in its distal portion. There was an atrial septal defect of the fossa ovalis type measuring about .5 cm. in greatest dimension.

The right ventricle was enlarged, and its wall was markedly thickened. Several septal bands were present, the uppermost one proceeding to the base of the pulmonary trunk. The first parietal band originated from between the bases of the aorta and pulmonary trunk, and proceeded...
towards the left side of the anterior wall of the right ventricle where it met the upper septal band widely. The loop thus formed narrowed the pulmonary conus. The second parietal band proceeded from beneath the aorta and lay adjacent to the anterior leaflet of the tricuspid valve. The endocardium of the right ventricle was focally whitened in many areas. From this chamber emerged both the aorta and pulmonary trunk. The aortic orifice was enlarged. Its valve was normally formed, but presented increased hemodynamic change. The coronary ostia emerged from the posterior sinuses of Valsalva.

There was a right aortic arch. An anastomosis had been made between the aorta and the right pulmonary artery by way of an arterial graft. The ductus arteriosus was closed.

The pulmonary trunk emerged from a narrowed conus, produced by the approximation of the septal with the first parietal band. The endocardial lining of this conus was irregularly thickened. The pulmonary orifice was smaller than normal. The pulmonary valve was bicuspid with an attempt at a third cusp. The pulmonary annulus was not continuous with the mitral annulus being separated by muscle.

The left atrium was enlarged, and its wall was thickened. The mitral orifice was smaller than

**Figure 7A**

**Figure 7B**

**Figure 7:** CASE 3. (A) Right ventricular view at the outflow tract of the pulmonary trunk. PT—Pulmonary trunk; C—Conus of pulmonary trunk; D—Ventricular septal defect; S—Septal band; P1—First parietal band. (B) Right ventricular view at outflow tract of aorta. A—Aorta; P1—First parietal band; P2—Second parietal band; T—Tricuspid valve.
normal; the mitral valve was normally formed and presented the usual hemodynamic change.

The left ventricle was normal in size, but its wall was thicker than normal. A muscle band was noted extending from the inferior papillary muscle to the annulus of the mitral valve. The endocardium of the ventricular septum was thickened at the base. The ventricular septum at its base presented a defect measuring about 0.6 cm. in greatest dimension. This defect was situated close to the central fibrous body, and the annuli of the mitral and tricuspid valves were contiguous in this region. The defect opened into the right ventricle in the upper part of the sinus adjacent to the pulmonary trunk.

The coronary artery distribution was similar to that in case 1.

The anatomic diagnoses were: Taussig-Bing complex with pulmonary stenosis: (a) right atrial hypertrophy and enlargement; (b) right ventricular hypertrophy and enlargement; (c) left atrial hypertrophy and enlargement; (d) left ventricular hypertrophy; (e) atrial septal defect, fossa ovalis type; (f) abnormal formation of the atrial and ventricular septa; (g) right aortic arch; (h) ventricular septal defect; (i) abnormal pulmonic valve; (j) left superior vena cava entering the coronary sinus.

**Discussion**

In the recent work of Lev et al., it was pointed out that the muscle bundles of the right ventricle in the Taussig-Bing heart without pulmonary stenosis were characteristic. A group of septal bands proceeded to the base, where contact was made with what was called the first parietal band.

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**Figure 8A**

**Figure 8C**

**Figure 8:** Taussig-Bing with pulmonary valvular stenosis. (A) Right ventricular view at the outflow tract of pulmonary trunk. **PT—Pulmonary trunk; PV—Pulmonic valve; D—Ventricular septal defect.** (B) Right ventricular view at the outflow tract of aorta. **A—Aorta; P1—First parietal band; P2—Second parietal band.**
This band lay between the aorta and pulmonary trunk and proceeded over the anterior wall of the right ventricle in a varying position according to the type. What was called the second parietal band lay beneath the aorta continuous with the first parietal band, and proceeded along the right side of the anterior wall adjacent to the anterior leaflet of the tricuspid. As one proceeded from the right ventricular type to the intermediate to the left ventricular type of Taussig-Bing, the first parietal band took on a more frontal position approaching the left side of the anterior wall close to the septum. Thus the rotation of the first parietal band to the left was associated with the placement of the pulmonary trunk into the left ventricle. In the Taussig-Bing heart, with pulmonary stenosis we may postulate that if this left movement of the first parietal band is not associated with such displacement of the pulmonary trunk, then pulmonary infundibular stenosis may occur, with the pulmonary trunk still emerging from the right ventricle, sandwiched in between the septal and first parietal band. This is what we see in our three cases.

The coronary arterial distribution of these three hearts is similar to that described in Taussig-Bing without pulmonary stenosis. There is a tendency for the embryologic left to take over what is normally the right supply, and the embryologic right to take over what is normally the left supply.

The position of the defect in two of our cases was lower than that which is usually seen in the Taussig-Bing complex. It was present in the region of the pars membranacea and the area posterior to it. Accompanying this position, there was a distinct cleft in the aortic leaflet of the mitral in one case and a beginning cleft in the second. In the third case, however, the defect was higher and the mitral valve was relatively normal.

Although our three cases were accompanied by pulmonary infundibular stenosis, one of us (M.L.) has seen a heart not included in this series in which a typical right ventricular type of Taussig-Bing was associated with pulmonary valvular stenosis engrafted hemodynamically on an abnormal valve (Fig. 8).

It thus becomes clear that when the aorta overrides or comes out mostly from the right ventricle but is related to the defect, then pulmonary stenosis or atresia is common. On the other hand, when the aorta comes out completely from the right ventricle, away from the defect, with the pulmonary trunk arising from the right ventricle, both ventricles or left ventricle, close to the defect, then pulmonary stenosis is uncommon. Thus there were only three cases of pulmonary stenosis in 41 cases of Taussig-Bing in our series.

Physiologically and clinically, case 1 behaved as a complete transposition type with pulmonic stenosis. Cyanosis was present from birth and was severe, with clubbing and erythemia. Poor development, slight dyspnea, anoxic spells and squatting equivalents, so commonly seen in any malformations with diminished pulmonary blood flow, were also seen. Characteristic of the transposition complexes are those findings related to the presence of cyanosis with a high output state early in development. These include the large head, the peculiar varicosities of the scalp and extremities, the prominent carotid pulsations, and the prominent substernal thrust, indicative of volume overloading of the right ventricle.

Radiographic and electrocardiographic examination also placed this case in the complete transposition group. The chest films showed a normally placed heart with gross cardiomegaly and egg-shaped configuration, with clear lung fields. The marked enlargement of the vascular pedicle was partly due to the anomalous systemic drainage. In cases previously reported, either a slight or gross cardiomegaly had been described, with concavity of the region normally occupied by the pulmonary trunk.
The electrocardiogram showed an extreme right axis deviation (QRS axis -170°) which is seen in complete transposition with pulmonary stenosis, but was also found in cases of Taussig-Bing with pulmonary stenosis previously published (Van Buchem et al.—145°, Bret and Turner-Soler—Case 2, -170°).

The clinical diagnosis of complete transposition with pulmonary stenosis was made because of the rarity of the entity Taussig-Bing with pulmonary stenosis. The angiocardio gram in retrospect was of greatest value in defining the general anatomy of this latter formation. The anteriorly placed aorta arose entirely from the right ventricle and the posteriorly placed pulmonary trunk appeared to arise from both ventricles. This position (Fig. 3A, 3C) is confirmed by the long pathway followed by the outflow tract of the left ventricle to the pulmonary trunk which arises mainly from the right ventricle. Severe infundibular stenosis was also seen (Fig. 3).

Summary and Conclusions
A case of “Taussig-Bing complex with pulmonary stenosis” is presented clinically and pathologically accompanied by pathologic reports of two other cases. The infundibular stenosis was explained as an uncommon variant of the Taussig-Bing complex related to compression of the pulmonary infundibulum between the septal and first parietal bands.

Resumen
Se reporta un caso del complejo Taussig-Bing con estenosis pulmonar en su aspecto clínico y patológico, conjuntamente con la descripción anatomo-patológica de dos casos adicionales. La estenosis infundibular se describe como una variante poco común del complejo de Taussig-Bing, relacionado con la compresión del infundibulo pulmonar entre las bandas parietales y del septum.

Zusammenfassung

References

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