An Analysis of 35 Cases of the Complete Form of Persistent Common Atrioventricular Canal

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SUMMARY
Thirty-five hearts with the complete form of persistent common atrioventricular canal were classified according to the morphology of the common anterior leaflet. The ratio of specimens with a free-floating undivided common anterior leaflet (Type I) to those with a divided common anterior leaflet attached to the septum (Type II) was 1:2. Type I cases had a high incidence of other cardiac (90 percent) and noncardiac (80 percent) malformations. These were primarily conotruncal anomalies (80 percent), left ventricular outflow obstructions (86 percent), aortic arch hypoplasia (30 percent), and abnormalities of organ position or symmetry including the polysplenia and asplenia syndromes. In contrast, Type II canals exhibited a high incidence of Down’s syndrome (40 percent), other cardiac and noncardiac anomalies being less common. The associated anomalies suggest that Type I lesions reflect an earlier interference during embryogenesis than Type II lesions.

A clockwise QRS loop and a normal or right axis on the electrocardiogram in four cases were associated with a left ventricular inflow tract of normal length, suggesting that conduction system displacement and a deficient base of the heart may be interrelated.

Postmortem double contrast radiography confirmed the angiographic differences between Type I and II lesions, and in Type I lesions clarified the alterations in the angiographic profile occurring with variations in the fullness of the common anterior leaflet.

A recent review of 36 patients who had died with the complete form of persistent common atrioventricular canal turned up an unexpectedly high incidence of conotruncal and left sided obstructive lesions in those patients who had a so-called free-floating common anterior leaflet. While examining these cases we encountered a number of other features which appear to have both clinical and embryological implications. The importance of anatomical studies for better clinical and surgical management of the complete form of persistent common atrioventricular canal is elegantly demonstrated in the papers of Rastelli et al.1-4 These authors found that the anatomy of the common anterior leaflet correlated well with such clinically significant aspects as amenability to surgical repair, the left ventricular angiographic profile, and the incidence of associated cardiac lesions. Rastelli’s classification is therefore used in this presentation.

Material and Methods
Postmortem double contrast radiography5-6 was employed to study 11 specimens during the past six years. A further 25 specimens came from the pathological collection of the Children’s Orthopedic Hospital and Medical Center and had previously been dissected by routine methods.

The cases were examined primarily from a postmortem point of view; specimens with a “single” or “common” ventricle were excluded. Otherwise, all specimens having a complete form of persistent common atrioventricular canal (hereafter termed persistent A-V canal) as defined by Wakai and Edwards7 were included in the
study. There were 36 hearts conforming to these criteria. Twelve of these were of Rastelli's type, having a free-floating undivided common anterior leaflet (not attached to the septum), hereafter termed "Type I" persistent A-V canal. Twenty-three were of the type with a "divided" common anterior leaflet attached to the ventricular septum hereafter called "Type II" persistent A-V canal. There was no specimen conforming to the third type described by Rastelli et al., having a divided common anterior leaflet attached to an anomalous muscle to the right of the ventricular septum. One specimen appeared to be a transitional form between this third type and Type II persistent A-V canal and will not be described further. Thus, the 35 cases to be reviewed all fall into Rastelli's Type I or II categories.

Observations Common to Both Types of Persistent A-V Canal

A. Noncardiac Congenital Malformations

In the 35 cases with persistent A-V canal, over half (19 cases) had associated noncardiac congenital malformations (table 1). Not surprisingly, Down's syndrome was the most frequent (11 cases). However, whereas Down's syndrome was found predominately in those cases with a divided common anterior leaflet (Type II), the asplenia and polysplenia syndromes or other evidence of abnormal organ symmetry were confined exclusively to cases with an undivided common anterior leaflet (Type I).

B. Deficiency of the Posterior Base of the Heart

In 1968, Goor, Lillehei and Edwards reported in cases of both partial and complete persistent A-V canal a deficiency of the posterior base of the heart including the left ventricular inflow tract. The authors suggested that this deficiency led to early depolarization of the posterior base of the heart and was responsible for the superior axis and counterclockwise QRS loop seen on the electrocardiogram in endocardial cushion defects.

Left ventricular inflow and outflow tracts were measured in all specimens using Goor's definition. Two specimens had associated transposition of the great arteries (Cases 1 and 2, table 2), and gross inspection revealed a deficient base of the heart in both. Measurements in the other 33 specimens showed a marked shortening (20 to 25 percent) of the left ventricular inflow tract in 28. Electrocardiograms were available in 18 of these, and all 18 showed the typical electrocardiogram seen in endocardial cushion defects, namely, a counterclockwise or initially counterclockwise QRS loop in the frontal plane with a superior QRS axis initially.

Five hearts did not exhibit the marked shortening of the left ventricular inflow tract and in four of these electrocardiograms could be reviewed. (One case reported previously.) In contrast to the 18 electrocardiograms in patients with shortened left ventricular inflow tracts the electrocardiograms from patients with normal left ventricular inflow tracts showed clockwise QRS loops in the frontal plane with a QRS axis between 60 and 150.

C. Malalignment between Atrial and Ventricular Septum

In the normal heart the ventricular and atrial septal planes form an angle of about 15 degrees to one another, with the ventricular septal plane lying at approximately 45 degrees to the frontal plane (fig. 1).

In persistent A-V canals, on the other hand, the plane of the ventricular septum lies virtually parallel to the frontal plane. In seven hearts, the atrial septum had either not rotated at all or had rotated only partially with the result that the angle between the atrial and ventricular septa was greater than normal (fig. 1A). In 11 others the atrial septal plane also had rotated so that the normal angle between the two septa was maintained (fig. 1B). In eight hearts, the atrial septum was rotated as well as shifted in such a fashion that both septa lay parallel to one another (fig. 1C). In two of these the atrial septum lay anterior to the ventricular septum and straddled the tricuspid orifice; in the remaining six, it lay posterior to the ventricular septum straddling the mitral valve. In seven others the plane of the atrial septum crossed the ventricular septal plane to varying degrees (fig. 1D). Two specimens had no atrial septum.

In summary, malalignment of the septa in relationship to one another was observed in 22 out of 35 cases. In all but the two cases specifically mentioned, the atrial septum was located posterior to the ventricular septal plane with the result that the atrial septum straddled the mitral valve to varying degrees. Where such malalignment or shift was marked, the left atrium was always small.

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### Table 1

| Incidence of Cardiac and Noncardiac Anomalies in 35 Cases of Persistent A-V Canal |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
|                                  | Type I                          | Type II                         |                                  |                                  |                                  |
|                                  | (12 cases)                      | (23 cases)                      |                                  |                                  |                                  |
| Major associated cardiac lesions | 11 (90%)                        | 3 (22%)                         |                                  |                                  |                                  |
| Down's syndrome                  | 2 (18%)                         | 9 (39%)                         |                                  |                                  |                                  |
| Other noncardiac malformations    | 8 (66%)                         | None                            |                                  |                                  |                                  |

(exclusive of Down's)

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Cardiac Abnormalities Associated with Rastelli's Type I Persistent A-V Canal

There were 12 specimens conforming to this classification. In this type of persistent A-V canal the common anterior leaflet has no attachment to the ventricular septum. In all our cases it formed a continuous sheet with the anterior leaflet of the tricuspid valve, being attached to the anterior papillary muscles of the right and left ventricle (figs. 2A, 3, 4A).

The fullness of the common anterior leaflet varied greatly from specimen to specimen. In some it was full and could be readily approximated with the posterior leaflet (fig. 4A). In others it was scanty and no such approximation was possible (fig. 3).

Anomalies of venous return were found in all four cases with asplenia and in one case with polysplenia. Venous anomalies are frequently associated with these syndromes and they are listed in table 2, but will not be discussed further.

Conotruncal Abnormalities

Abnormalities of the conotruncal region were frequent in Type I lesions (table 2). Perhaps the most striking feature was the hypoplasia of the crista supraventricularis present in 10 of the 12 specimens. In three specimens (Cases 7, 8, 11) this hypoplasia was accompanied by an aortic valve which arose partially or entirely above the right ventricle. In two of these (Cases 8 and 11), a ventricular septal defect was present between the common anterior leaflet and the crista supraventricularis and extended up to the aortic valve. The third specimen (Case 7) was unusual in that the aortic valve, while situated entirely above the right ventricle, was separated from it by a membrane stretching from the common anterior leaflet to the crista supraventricularis, so that the aortic valve in fact communicated solely with the left ventricle (fig. 3).

Four cases were associated with a tetralogy of Fallot type of anatomy (Cases 3, 4, 5, 6). In these the anteriorly displaced crista supraventricularis was likewise hypoplastic and the aorta arose partially or entirely above the right ventricle (fig. 4A). In two further cases there was extreme hypoplasia (virtual absence) of the crista supraventricularis associated with d-transposition of the great vessels (Cases 1 and 2). One of these (Case 1) represented a double outlet right ventricle with transposition of the great arteries. (Both great vessels arose from the right ventricle, and there was a wide band of muscle between the common anterior leaflet and the posterior pulmonary valve.) In two cases (Cases 10 and 12) there was a VSD extending to the aortic valve with little rightward displacement of the aorta. Hypoplasia of the crista was associated with pulmonary valve stenosis or atresia in seven specimens (table 2).
Figure 2

Type I persistent A-V canal (Case 10). (A and B) Postmortem double contrast roentgenograms. (A) Axial view of the A-V valves. Outlined by contrast are the atrioventricular valves, crest of the ventricular septum, aorta, patent ductus arteriosus and adjoining pulmonary artery. Note narrow aortic arch. (B) Frontal projection mimicking left ventricular angiogram shown in C and D. (C) Systole. Note smooth posterior border of outflow tract with no ballooning of common anterior leaflet which is not full. (D) Diastole. Common anterior leaflet has swung forward and now forms a right angle with the crest of the ventricular septum.
### Table 2

**Anomalies Associated with Type I Persistent A-V Canal**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Noncardiac anomalies</th>
<th>Cardiac anomalies</th>
<th>Other</th>
<th>Surgery, etc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>5 years Male</td>
<td>Bilateral bilobed lungs.</td>
<td>Tetralogy of Fallot P.S. +, (fused thick cusps)</td>
<td></td>
<td>Subaortic stenosis Blaylock secondary to papillary aged 5 muscle displacement.</td>
</tr>
<tr>
<td>7</td>
<td>4 days Female</td>
<td>Hypersegmented spleen and 2 accessory spleens. P.S.I. 2 normal plus 1 intrathymic parathyroid. Absent lung fissures. Absent body and tail pancreas. Mal-rotation of bowel.</td>
<td>Ao valve lies above RV, communicates only with L.V. (fig. 3) A.S. (Bicuspid) Anteriorly displaced hypoplastic ++ crista. P.S. +</td>
<td>Hypoplastic Ao arch</td>
<td>Single atrium. Subaortic stenosis (fig. 3)</td>
</tr>
<tr>
<td>9</td>
<td>3 years Female</td>
<td>Ao overriding +, Crista fused to upper part of C.A.L.</td>
<td>Hypoplastic Ao Arch</td>
<td>Subaortic stenosis, Secondary to Ao overriding. E.C.G. — clockwise QRS loop, R.A.D.</td>
<td></td>
</tr>
</tbody>
</table>

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Narrowing of the Left Ventricular Outflow Tract

Anatomic narrowing of the left ventricular outflow tract was present in eight of the twelve specimens. In two of the eight hearts the hypertrophied muscular ventricular septum formed a ridge immediately beneath the somewhat rightwardly displaced aortic valve and appeared responsible for the narrowing (Cases 9 and 11). In three other cases (3, 7, 10) there was discrete yet significant muscular hypertrophy lower down the outflow tract. In one of these (fig. 3) a ridge of valve-like tissue protruded into the left ventricular outflow tract causing additional narrowing. In three hearts the anterior papillary muscle arose from an abnormally anterior location (situated in the outflow tract) and was responsible for the narrowing (Cases 4, 6, 10). One specimen had valvular aortic stenosis (Case 7). One additional case had an abnormally small aortic valve ring, as well as valvular and supravalvular stenosis (Case 5).

**Figure 3**

*Type I persistent A-V canal (Case 7). Postmortem double contrast radiogram. Axial view of A-V valves which are outlined by contrast as are the left ventricular outflow tract and aorta. The common anterior and posterior leaflets are scanty. Note subaortic stenosis and the high, rightwardly displaced aortic valve. The aortic arch is narrowed.*

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Hypoplasia of the Aortic Arch

Narrowing of the aortic arch between the left common carotid artery and left subclavian take-off was encountered in four out of eleven cases (Cases 7, 8, 9, 10); in the 12th case the aortic arch had been removed. The narrowing ranged from marked (fig. 2) to mild (fig. 3).

Cardiac Abnormalities Associated with Rastelli’s Type II Persistent A-V Canal

There were 23 specimens having a so-called “divided” common anterior leaflet. The leaflet always was attached to the right of the ventricular septum, as previously noted by Rastelli et al. In 18 out of 23 cases this attachment was by abnormally short chordae (fig. 5). In addition, the tricuspid portion of the common anterior leaflet was scanty in 13 specimens (fig. 5). Altogether, 12 specimens had both scanty valve tissue and shortened chordae, six had shortened chordae only, and one had only scanty valve tissue. In other areas of the atrioventricular valves, tongue-like extensions of redundant valve tissue were present in one or more areas. The anterior portion of the anterior leaflet was most frequently affected. In three specimens the valve tissue was particularly excessive, and in one it spanned the tricuspid orifice resulting in a double orifice tricuspid valve. The posterior leaflet of the mitral valve was often small and the common anterior and posterior leaflets often extended partially into the area normally occupied by the posterior leaflet. Abnormally deep valve commissures were frequently seen on the tricuspid side at the sites indicated in figure 7D.

There was a high incidence of abnormalities of left ventricular papillary muscles (15 specimens) and the anterior papillary muscle of the tricuspid...
valve (15 specimens). Abnormally short muscles (sometimes only 2 to 3 mm high) were noted in nine hearts (8 right ventricular, 1 left ventricular), in four of which the chordae were also short. Abnormal positions of the papillary muscles were noted in 11 specimens (3 right ventricular, 8 left ventricular). In two of these the anterior displacement of the papillary muscle into the left ventricular outflow tract was responsible for subaortic stenosis. In 7 specimens there was only a single left ventricular papillary muscle. In only 3 specimens were all of these muscles normal. All these abnormalities may have contributed to atrio-ventricular valve dysfunction, particularly insufficiency, during life.

In eight specimens there were additional cardiac anomalies. There was hypoplasia of the aortic arch (2 cases), valvular aortic stenosis (1 case), subaortic stenosis due to a displaced papillary muscle (2 cases), and a narrow aortic valve ring (1 case). There were two additional specimens with a hypoplastic left ventricle in which the aortic arches were likewise hypoplastic. Severe or complex conotruncal abnormalities of the type seen in specimens with an undivided common anterior leaflet were not encountered in Type II lesions.

The Left Ventricular Angiocardiogram

The angiographic appearance of the left ventricular outflow tract in persistent A-V canal was first described by Baron et al.\(^1\) in 1964, and was thought to be characteristic for all forms of persistent A-V canal. Three years later Rastelli et al.\(^2\) presented angiographic evidence that in Type I lesions the typical gooseneck as well as the systolic scalloping along the posterior border of the left ventricular outflow tract is absent. Instead, in diastole there is a right angle configuration at the junction of the left ventricular outflow tract with the body of the left ventricle. Rastelli et al. suggested that the observed deviations from the characteristic features described by Baron et al. were the result of differing morphology between undivided and divided common anterior leaflets.

Figures 2C and 2D show an angiocardiogram (Case 10) in which a Type I persistent A-V canal was suspected clinically, because there was no typical gooseneck deformity during systole, and in diastole there was a right angle appearance at the lower end of the left ventricular outflow tract as described by Rastelli et al.\(^2\) However, the systolic phase of the angiocardiogram (fig. 2C) differed from Rastelli’s in that there was no bulging along the right border of the left ventricle. In specimens in which the common anterior leaflet was scanty, the double contrast studies resembled figure 2B. It was concluded therefore that the bulging described by Rastelli et al. most likely was due to a large common anterior leaflet billowing upwards into the atrium (figs. 4A and B). At postmortem the common anterior leaflet was indeed not large (figs. 2A and B). Our observations suggest that in systole the left ventricular angiocardiogram may help in assessing the tissue mass of the common anterior
leaflet which may be of importance when surgical correction is being considered.

While rotating the heart prepared for double contrast studies from an axial to a frontal projection, we observed under fluoroscopy that the thickened irregular edge of the divided common anterior leaflet in Type II lesions contributes to the classic serrated appearance of the left ventricular outflow tract in systole as described by Baron et al. (fig. 6). In Type I lesions the smooth free edge of the undivided common anterior leaflet (figs. 2A, 4A) is largely responsible for the smooth appearance of the posterior wall of the left ventricular outflow tract as emphasized by Rastelli et al. (figs. 2B, 4B).

Judging from our postmortem double contrast studies, the undivided common anterior leaflet straddles the tricuspid and mitral orifice in such a way that approximation between the common anterior and the common posterior leaflets appears comparable on the mitral and tricuspid sides, in all but patients with hypoplastic left ventricles (figs. 2A, 3, 4A). Translating the postmortem information to the clinical situation the conclusion appears justified that the size of the regurgitant mitral jet visualized angiographically may, in Type I lesions, also serve as a measure of tricuspid incompetence.

**Embryological Considerations**

The marked differences in distribution of associated cardiac and noncardiac congenital malformations between the two types of complete A-V canal raises the question whether cases with an undivided common anterior leaflet do not reflect an earlier interference in embryonic development. With the clustering of the asplenia, polspleenia syndromes, conotruncal abnormalities and aortic arch hypoplasia in Type I lesions, it seems likely that an interference with normal embryonic development occurs between the 24th and the 27th day of embryonic life. During this short time span the above cardiac structures appear and undergo rapid development and are therefore most susceptible to a teratogenic insult. The crista supraventricularis is derived from the conus cushions, as are the papillary muscle of the conus and the most anterior portion of the anterior leaflet of the tricuspid valve (figs. 7A and 7B). Absence of the papillary muscle of the conus or conus attachment of the tricuspid valve was noted in all Type I cases by Rastelli and ourselves. The conus-derived portion of the anterior leaflet of the tricuspid valve may likewise be lacking (fig. 7C), with the result that the lateral cushions, being unable to fuse with the conus derivative, fuse instead with the anterior endocardial cushion. This would result in the observed undivided common anterior leaflet with obliteration of the normal anteromedial commissure (fig. 7C).

Type II A-V canals appear to be the result of a later insult occurring when the critical phases of conotruncal and aortic arch development have passed, but when modeling of the valve leaflets, chordae, and papillary muscles is most active. This

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**Figure 6**

Type II persistent A-V canal. Postmortem double contrast radiogram. Left heart outlined by contrast to mimic left ventricular angiocardiogram. Note irregular edge of common anterior leaflet along the posterior border of the outflow tract.

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would account for the high incidence of anomalous development of these structures. In this connection it seems likely that the type of double orifice mitral or tricuspid valve resulting from proliferative tissue bridges may be expected in Type II A-V canals, but not in the embryologically earlier Type I canals. In reviewing the pertinent literature, we found this type of double orifice A-V valve only associated with Type II A-V canals, partial A-V canals, and other cardiac abnormalities without endocardial cushion defects.17-24

Discussion

About one-third (12 out of 35) of the cases in the present study were of Rastelli's Type I, and two-thirds (23 out of 35) were of Type II. Many centers might not consider surgical correction in patients with asplenia (4 cases), polysplenia (1 case), or Down's syndrome (11 cases). After excluding these there would still remain 19 cases that might be considered for surgical correction. Of these, one-third (6) fall into Type I and two-thirds (13) into the Type II class (tables 1 and 2), a higher incidence of Type I lesions than is generally appreciated.

Angiographic recognition of Type I cases is of importance, not only because of the greater difficulty in repairing these lesions but also because conotruncal abnormalities should be expected and defined. Left ventricular outflow obstructions and aortic arch narrowing should be sought and the clinician alerted to the high probability of co-existing noncardiac malformations, which are notably absent in Type II lesions. The high incidence of conotruncal abnormalities was suggested already by Rastelli's observations in Type I lesions. However, the frequency of left sided obstructive lesions and hypoplasia of the aortic arch has not previously been recognized. In addition, our studies suggest that the amount of valve tissue available for repair in Type I cases can be assessed angiographically, and the degree of tricuspid regurgitation can be judged by the degree of mitral regurgitation seen on the left ventricular angiocardiogram.
When pulmonary artery banding is considered in persistent A-V canals, the presence of tricuspid incompetence will decisively influence the clinical outcome. The high incidence of short chordae and scanty valve tissue of the tricuspid component of the common anterior leaflet in Type II A-V canals, as reported here, must significantly contribute to the poor results reported following pulmonary artery banding. In Type II lesions, the degree of tricuspid incompetence is difficult to gauge, and the degree of mitral regurgitation is no guide to tricuspid competence.

In repairing Type II lesions, the possibility of papillary muscle abnormalities should be borne in mind. The presence of a single left ventricular papillary muscle may result in a parachute valve type of deformity postoperatively. Low papillary muscles may be conducive to postoperative mitral or tricuspid regurgitation, and a papillary muscle displaced into an already narrowed left ventricular outflow tract may assume functional significance postoperatively.

In both types of persistent A-V canals, the shift of the atrial septum resulting in overriding of the mitral valve can cause cyanosis, even in the absence of pulmonic stenosis or pulmonary hypertension. Such cyanotic patients may be operable. Septal malalignment may also result in a small left atrium postoperatively unless the septum is repositioned.

The genesis of the electrocardiographic pattern typical of endocardial cushion defects is not known. Goor, Lillehei and Edwards\(^6\) postulated that the deficiency of the posterior base of the heart results in early depolarization of the diaphragmatic surface of the heart. Feldt, DuShane and Titus\(^26\) suggest that the posterior displacement of the left bundle branches and the early depolarization of the posterior portion of the left ventricle is responsible for the typical electrocardiographic pattern. Our findings suggest that the deficient development of the base of the heart and the displacement of the conduction system may go hand in hand; when the base of the heart is not shortened, the conduction system can develop normally even with a profound disturbance in the development of the endocardial cushions. Therefore, the displacement and abnormalities of the conduction system may be entirely secondary to the deficient base of the heart. Although cases with isolated tetralogy of Fallot and double outlet right ventricle with electrocardiograms characteristic of endocardial cushion defects have been documented,\(^26, 27\) such a pattern in a patient considered to have tetralogy or double outlet right ventricle should always alert one to the possibility of a concomitant Type I A-V canal.

In conclusion, there is great variability in the anatomy of persistent A-V canals and associated lesions. However, by employing Rastelli's classification the cases fall into two major groups which are embryologically meaningful and clinically useful. In Type I lesions major associated cardiac and noncardiac anomalies are the rule. In Type II lesions, provided Down's syndrome is absent, other congenital anomalies and additional complex cardiac lesions are unlikely.

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