Total Anomalous Pulmonary Return
An Analysis of Thirty Cases

By Vincent L. Gott, M.D., Richard G. Lester, M.D., C. Walton Lillehei, M.D. and Richard L. Varco, M.D.

Thirty cases of total anomalous pulmonary return have been collected and analyzed. The pathways of drainage and their embryologic development are discussed. The cardiac catheterization, electrocardiographic and radiologic findings are also presented. These laboratory data accompanied by the clinical findings seem to identify a recognizable diagnostic picture for this malformation. Finally, our attempts at surgical correction of this defect are briefly enumerated.

Although anomalous pulmonary return has usually been considered quite an uncommon defect in the past, this may be an inaccurate impression. The misconception probably stems in part from the casual attention it has aroused among surgeons treating cardiovascular malformations.

Winslow is credited with describing the first case in 1739. The anomalous drainage in Winslow's case was reported to be only partial and it was not until 1868 that total anomalous pulmonary return was described by Friedowsky. In 1942, Brody was able to collect from the literature only 102 cases of anomalous pulmonary drainage. Sixty-five of these were partial anomalous returns and 37 were total. Since Brody's collection of 37 cases, we have found only 49 additional cases reported in the literature. The majority of these authors have reported one or two cases. Keith, however, collected 14 personal cases, and of these 13 were verified at postmortem examination.

The drainage of a single pulmonary vein or the veins from one lung into the right side of the heart has been reported more frequently in the past few years. It is an entity that is less likely to create serious clinical distress. In our experience, its association with an atrial septal defect has been frequent. This paper will discuss only the less common but more critical problem of total anomalous return. We present here 30 cases from the University of Minnesota Hospitals (fig. 1). Three of these cases were discussed previously by Levin, these cases having a drainage through a "persistent left superior vena cava" or left vertical vein.

Twenty-six of the 30 patients have had proved diagnoses of total anomalous pulmonary venous return, 23 at autopsy and 3 at surgery. In four additional cases the diagnosis was made after a thorough clinical evaluation. Thirteen of the autopsied cases had only an associated patent foramen ovale. An additional four had also a patent ductus arteriosus, and six had other kinds of associated major cardiac defects.

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**Fig. 1.** Analysis of 30 cases of total anomalous pulmonary return.
Table 1.—Pathology in Six Cases with Associated Major Cardiac Defects of the Heart

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Sex</th>
<th>Age</th>
<th>Drainage</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>G. C.</td>
<td>M</td>
<td>25 days</td>
<td>I.V.C.</td>
<td>Very large interatrial defect making heart practically three chambered. This patient’s brother died at 3 months with total anomalous pulmonary return</td>
</tr>
<tr>
<td>R. G.</td>
<td>M</td>
<td>5 wks</td>
<td>Anterior cardinal veins</td>
<td>Cor bilocular. Dextrocardia. Patient had persistent anterior cardinals and right and left pulmonary veins entering into respective cardinals</td>
</tr>
<tr>
<td>B. S.</td>
<td>M</td>
<td>3 days</td>
<td>R.A.</td>
<td>Left atrium is small piece of tissue without lumen. All pulmonary veins entering large right atrium. Single vessel leads from ventricle supplying lungs and neck</td>
</tr>
<tr>
<td>P. H.</td>
<td>F</td>
<td>3 mo.</td>
<td>R.A.</td>
<td>Absence of left atrium and left ventricle. Pulmonary veins enter right atrium. Aorta leaves right ventricle</td>
</tr>
<tr>
<td>M. R.</td>
<td>F</td>
<td>7 wks</td>
<td>L.S.V.C.</td>
<td>Dextrocardia. Pulmonary artery fibrotic at right ventricle. Interventricular defect with aorta over riding. All pulmonary veins into L.S.V.C.</td>
</tr>
<tr>
<td>M. S.</td>
<td>F</td>
<td>7 mo.</td>
<td>Abd. viss.</td>
<td>Pulmonary artery fibrotic at ventricle. Common ventricle. Large interatrial defect. Pulmonary veins empty into anomalous vessel that passes through diaphragm and terminates in pancreas and mesenteric veins</td>
</tr>
</tbody>
</table>

(See table 1.) In this group there were 17 males and 13 females.

As shown in figure 2, most of the patients did not survive the first three months of life. However, all seven of those now living are past infancy and one is a 33 year old woman, the mother of two children. We believe this is next to the oldest surviving patient recorded in the literature.

Pathology

The pathways of anomalous drainage in 26 proven cases are depicted in figure 3. The relative frequency of the various drainage sites corresponds closely to that described in those accumulated cases from the literature. Drainage via a “persistent left superior vena cava” is the most frequent pathway, the other two common sites being into the coronary sinus and directly into the right atrium. These routes of drainage have been noted in the great majority of cases; however other channels have been recorded rarely. Our series includes examples of most of these unusual drainage ways. It does not, however, include a case with drainage into the ductus venosus, of which three have been reported previously. An approximation of this tract, however, was found in one patient in whom the pulmonary veins emptied into the portal system (fig. 4). Two pathways of drainage in this series of cases have not previously been recorded in the literature. In M. S. the pulmonary veins from both lungs joined to form an anomalous channel that coursed caudad, penetrated the diaphragm and then subdivided into many small branches, terminating in and anastomosing with the veins of the pancreas and mesenteric plexus. This patient also had the multiple associated defects of situs inversus with dextrocardia, common ventricle, a large atrial septal defect and an atretic origin of the pulmonary artery. Despite the multiplicity of malformations this patient lived seven months. The second (R. G.) had drainage of the right and left pulmonary veins into the corresponding persistent anterior cardinal veins. This patient also had dextrocardia and cor bilocular.

In all the cases without associated major cardiac defects at autopsy, the right side of the heart was hypertrophied and the left side

Fig. 2. Age distribution of patients.
The majority of cases, the patent foramen ovale or a larger atrial septal defect is the sole avenue for partial left-sided venous filling. As noted previously, in all 17 autopsied cases without major associated defects the foramen ovale was open to varying degrees.

**Embryology**

The lung develops as an outpouching from the ventral wall of the foregut, and the venous plexus covering the lung bud is thought to be part of the splanchnic plexus covering the foregut. Some authorities\(^7,8\) feel that the pulmonary venous plexus is gathered together into a common pulmonary vein which courses ventrally to tap the sinus venosus of the primitive heart. A recent and more strongly advocated theory\(^9-11\) visualizes the pulmonary vein arising as an outpouching from the dorsal wall of the sinus venosus. This stem in turn communicates with the pulmonary venous...
plexus. The opening of the pulmonary stem is initially in the center of the sinus venosus but with the formation of the septum the communication shifts to the left. The common pulmonary vessel is absorbed by the atrial wall so that its four subdivisions eventually enter the heart separately.

In the embryo the splanchnic plexus of the foregut communicates with all of the surrounding systemic and visceral veins (fig. 5). Thus at one stage in the normal development of the embryo there are communications between the venous plexus of the lung bud and the pre- and postcardinals, which eventually form the superior vena cava, innominate veins, azygos vein and coronary sinus. There are also communicating venous channels with the umbilical and vitelline veins which are the primordia for the portal vein, ductus venosus and inferior vena cava. Thus if there is a failure of connection between the pulmonary stem from the heart and the pulmonary plexus, other venous channels may persist to create this abnormality of total anomalous pulmonary return.

Theoretically, when the pulmonary veins enter directly into the right atrium, there has been a failure of the common pulmonary vein to be displaced to the left at the time of the development of the interatrial septum.

The most frequently encountered route of drainage in total anomalous pulmonary return is via the “persistent left superior vena cava.” The use of this terminology has created some confusion in the literature because the vessel so named is not a persistent left superior vena cava or left anterior cardinal vein in the truest sense. Embryologically the anterior cardinals join with the posterior cardinals to form the common cardinals or duct of Cuvier. The common cardinals in turn empty into the lateral recesses of the sinus venosus (fig. 5). On the right the anterior and common cardinal form the superior vena cava and its immediate tributaries. However, on the left the anterior and common cardinals regress leaving a proximal vestige as the coronary sinus (fig. 6). A truly persistent left superior vena cava communicates proximally with the coronary sinus and distally with the left innominate vein. Of the cases cited in the literature as having drainage via the left superior vena cava, none appear to drain in this fashion. The drainage is consistently cephalad into the left innominate vein and thence into the superior vena cava. In this paper, however, we shall use the term “persistent left superior vena cava” in order to be consistent with the previous literature.

Clinical Findings

A history of cyanosis of a mild degree was present in most of our patients who at autopsy
<table>
<thead>
<tr>
<th>Groups</th>
<th>Pts</th>
<th>Drainage</th>
<th>I.V.C.</th>
<th>S.V.C.</th>
<th>R.A. %</th>
<th>R.V. %</th>
<th>P.A. %</th>
<th>Sys. A %</th>
<th>Misc.</th>
<th>CAP.</th>
<th>Pressure</th>
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<td>Living without operation</td>
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<td></td>
<td></td>
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<tr>
<td>H. S.</td>
<td>L.S.V.C.</td>
<td>22.3</td>
<td>21.7</td>
<td>21.4</td>
<td>22.1</td>
<td>21.4</td>
<td>L.S.V.C., 22.0</td>
<td>23.1</td>
<td>78/6</td>
<td>38/13</td>
<td></td>
</tr>
<tr>
<td>T. H.</td>
<td>L.S.V.C.</td>
<td>16.1</td>
<td>14.6</td>
<td>15.3</td>
<td>14.3</td>
<td>16.8</td>
<td>P.V., 17.6</td>
<td>17.4</td>
<td>54/0</td>
<td>54/0</td>
<td></td>
</tr>
<tr>
<td>J. M.</td>
<td>?</td>
<td>14.3</td>
<td>16.3</td>
<td>16.4</td>
<td>93%</td>
<td>93%</td>
<td>98%</td>
<td>17.7</td>
<td>17.9</td>
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<tr>
<td>T. K.</td>
<td>R.A.</td>
<td>11.3</td>
<td>15.6</td>
<td>93%</td>
<td>87%</td>
<td>93%</td>
<td>93%</td>
<td>14.7</td>
<td>80%</td>
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<tr>
<td>Living with operation</td>
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<tr>
<td>R. P.</td>
<td>S.V.C.</td>
<td>15.7</td>
<td>19.5</td>
<td>19.7</td>
<td>18.6</td>
<td>17.8</td>
<td>P.V., 19.1</td>
<td>20.1</td>
<td>109/0</td>
<td>46/15</td>
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<td>D. H.</td>
<td>R.A.</td>
<td>11.4</td>
<td>17.5</td>
<td>16.4</td>
<td>16.4</td>
<td>15.7</td>
<td>85.8</td>
<td>18.0</td>
<td>40/0</td>
<td>40/20</td>
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<tr>
<td>R. K.</td>
<td>R.A.</td>
<td>12.5</td>
<td>14.4</td>
<td>92%</td>
<td>92%</td>
<td>15.3</td>
<td>15.1</td>
<td>34/2</td>
<td>34/10</td>
<td></td>
<td></td>
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<tr>
<td>Autopsied without assoc. defects</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N. M.</td>
<td>C.S.</td>
<td>14.3</td>
<td>18.8</td>
<td>18.9</td>
<td>18.2</td>
<td>19.6</td>
<td>55/6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. H.</td>
<td>C.S. &amp; L.I.V.</td>
<td>6.9</td>
<td>16.7</td>
<td>90%</td>
<td>96%</td>
<td>16.6</td>
<td>18.3</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>W. W.</td>
<td>C.S.</td>
<td>7.9</td>
<td>11.4</td>
<td>11.6</td>
<td>10.5</td>
<td>14.9</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>W. K.</td>
<td>L.S.V.C.</td>
<td>2.2</td>
<td>9.1</td>
<td>77%</td>
<td>78%</td>
<td>71%</td>
<td></td>
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<tr>
<td>L. P.</td>
<td>L.S.V.C.</td>
<td>3.3</td>
<td>2.2</td>
<td>52%</td>
<td>91%</td>
<td>8.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>B. D.</td>
<td>R.S.V.C.</td>
<td>8.4</td>
<td>12.5</td>
<td>77.6%</td>
<td>69%</td>
<td>17.4</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>48.9</td>
<td>12.8</td>
<td>13.9</td>
<td>12.8</td>
<td>18.4</td>
<td>68%</td>
<td></td>
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</tbody>
</table>

**Table 2.** Cardiac Catheterization Data
were without associated major cardiac defects. The terminal patients had moderate to marked cyanosis. The systemic arterial saturation ranged from 69 to 93 per cent in those so tested. Although some degree of arterial desaturation is present by necessity in nearly every patient with total anomalous pulmonary return, in some cases clinical cyanosis may not be evident until late in the patient's life span. For this reason we agree with Edwards, who feels that this malformation should be placed in the cyanotic group. Those, however, with partial anomalous return also have the potential for cyanosis. Most of them have a defect in the atrial septum, and may eventually develop pulmonary hypertension which, in turn, tends to induce a right to left shunt.

Fourteen of the 17 autopsied cases without associated defects exhibited during life a systolic murmur in the second or third left interspace. In three patients no murmur was recorded. The susurrations heard were attributed to the flow through the atrial septal defect during atrial systole with the additional possibility of a component due to turbulence in the pulmonary artery being induced by the increased minute flow.

**CARDIAC CATHETERIZATION**

Thirteen individuals underwent cardiac catheterization. Four of these patients are living and it is presumed from the data that their major defect is total anomalous pulmonary return. The remaining nine patients had this diagnosis confirmed at the time of surgery or autopsy. The data are presented in table 2. A striking feature is the high oxygen saturation of the right atrial blood. In every instance except one it was equal to or higher than the oxygen saturation of the systemic arterial blood. These findings are unlikely to appear in any other congenital heart condition save for the example in which the catheter tip is placed immediately adjacent to an atrial defect. This group exhibited some pressure increase in the right ventricle and in the pulmonary artery.

Two of the patients had on catheterization some evidence of pulmonary outflow tract obstruction. One of these (H. S.) is living without an operation. The other (R. F.) was successfully operated on. It is of interest that these two persons with pulmonary stenosis are the oldest individuals in the series, 16 years of age and 33 years old, respectively. Apparently the dampening effect on the greatly increased pulmonary flow by the stenotic pulmonary valve was of some value in retarding the onset of damaging pulmonary vascular changes.

**ELECTROCARDIOGRAM**

Twelve of the 17 patients without associated intracardiac defects had right axis deviation and seven demonstrated right ventricular hypertrophy. A high incidence of right bundle branch block has been reported with this defect. However, we found only one case that showed such a tracing.

**RADIOLOGIC EXAMINATION**

Of the 20 cases proven at autopsy or at surgery, without other significant cardiac defects, all but two underwent a conventional x-ray examination, including multiple films and fluoroscopy. Six patients were also examined by angiocardiography.

Several authors have referred to the "figure of eight" appearance (fig. 7) formed by engorged "left" and right superior vena cava above, and the bulk of the heart below. This

![Fig. 7. Total anomalous return through "persistent left superior vena cava" demonstrating "figure of eight" configuration.](http://circ.ahajournals.org/)

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has come to be accepted as typical of this malformation. It should, however, be kept clearly in mind that this roentgen appearance is seen only in those cases in which the anomalous pulmonary vessels empty into a "persistent left superior vena cava." Unfortunately, not all of the patients even with this anatomic derangement will show this diagnostic form on the plain posteroanterior roentgenogram. In this group of 20 only three demonstrate well the "figure of eight". These have been reported previously by Levin and Borden.6 This diagnosable group represents but half of the total number of persons with "persistent left superior vena cava."

All of the cases reviewed, with the exception of one who died at the age of 10 days, had a mild to marked increase in the pulmonary vascularity. In all the patients who survived three or more months, this was present to a marked degree.

Twelve of the 18 patients showed what we believe is a characteristic shape of the heart as viewed in the posteroanterior position. This typically, is a box-like appearance with an almost horizontal take off of the left border of the heart below the aortic arch. It should be in association with the increased vascularity just mentioned (figs. 8 and 9). This profile results from massive enlargement of the right atrium and ventricle, with the latter structure probably forming most of the upper left border of the heart. There is no left atrial enlargement.

While this appearance has not been emphasized in the literature it is not an original observation. Taussig's monograph14 demonstrates a case showing this feature. Snellen18 refers to a similar contour. In Keith's article6 there are several tracings of roentgenograms which also depict this outline in some of the cases.

A review of the earliest angiocardiograms made on the first patients to undergo this procedure failed to reveal much of diagnostic aid. There was diffusion of the opaque medium within the heart after entry from the vena cava and, in one case, evidence of recirculation through the right side of the heart. We believe these examinations were probably inadequate technically.

Angiograms secured in a later series of three cases, however, show an appearance that we feel is typical for this disorder. These patients were all examined in the right posterior oblique projection. In two cases 70 per cent Diodrast and in one 70 per cent Urokon (1.2 to 1.5 ml. per Kg. of body weight) was injected as rapidly as possible, manually. One patient was studied with the apparatus described by Rigler.
Fig. 10. A and B. Angiocardiogram demonstrating large right atrium, right to left shunt into small left atrium, followed by simultaneous opacification of a large pulmonary artery from the right ventricle and a small aorta from the left ventricle.

Fig. 11. A and B. Angiocardiogram demonstrating findings similar to figure 10.

and Watson and the other two, with the Fairchild camera equipment.

In all, immediately following the opacification of a very large right atrium, one saw filling of the left atrium, and this cavity was quite small. Following this there was simultaneous opacification of the pulmonary artery, which was abnormally large, and of the aorta, which
was proportionately hypoplastic. The latter structure clearly filled from the left side of the heart and was seen with great clarity despite its small size. It filled well before it could have done so by circulation of the opaque material through the pulmonary circuit (figs. 10A and B, and 11A and B).

Yet another angiocardiographic sign is the dilution of the opaque material by the jet of nonopacified pulmonary venous blood entering the superior or inferior vena cava.

We feel, then, that there are several significant roentgen signs that can be perceived in this disorder. The so-called “figure of eight” appearance is probably pathognomonic of anomalous pulmonary drainage through “a persistent left superior vena cava” although some cases with this anomaly do not show the characteristic roentgen picture. The “box-like” shape of the heart, in association with increased pulmonary vascularity, while not an absolutely diagnostic sign, is highly suggestive of the anomaly in some of its forms. The angiocardiographic appearance described above appears to be typical of the disorder when evident.

DISCUSSION

Total anomalous pulmonary venous return is a congenital malformation of serious significance that can be diagnosed during life if complete cardiac studies are available. The clinical picture of minimal cyanosis, a systolic murmur along the left sternal border and right axis deviation in the electrocardiogram should be suggestive clues. The x-ray findings are extremely helpful. The diagnosis is corroborated if the right atrial oxygen saturation equals or is higher than the peripheral arterial oxygen saturation.

Despite the potential curability of many of these lesions, surgery has been performed only rarely. There are 10 recorded attempts with two successful results in the literature.2, 4, 16, 18, 19, 20 Six of our patients were submitted to surgery and three of these survived corrective procedures. It is planned to devote another paper to a more detailed account of the surgical management of this defect and also to discuss our suggestions for future surgical approaches.

A brief note on the operative material follows:

(1) R. F., a woman of 33, with a superior vena cava drainage had a successful anastomosis of the base of the left atrium to the side of the anomalous pulmonary vessel.

(2) D. H., a boy of 3 with a drainage into the right atrium, had a successful anastomosis of (1) the side of the left atrium to the side of the vein draining the left lung and (2) the tip of the left atrium to the cardiac end of the divided vein from the apical posterior segment of the left upper lobe.

(3) R. K., a 4 year old boy, survived a repositioning of the anterior portion of the atrial septum. It was moved to the right of the four pulmonary veins formerly entering directly into the right atrium. This is believed to be the first successful, completely corrective procedure performed for total anomalous pulmonary return.*

(4) M. M., a 3 month old boy with a “persistent left superior vena cava,” died a few hours following a side-to-side anastomosis between the left atrium and the anomalous vessel.

(5) N. M., a 2 year old girl, died during an intracardiac procedure utilizing the atrial well. The drainage was thought to be into the right atrium but was actually into the coronary sinus.

(6) M. H., a 3 month old girl with coronary sinus drainage, died in the recovery room following a side-to-side anastomosis of the coronary sinus to the left atrium.

Currently we believe that the adoption of technics for totally bypassing the heart and lungs during the interval of surgical correction may improve the outlook of patients with this malformation.

SUMMARY

1. Thirty cases of total anomalous pulmonary return are presented. Of these, 23 have been verified at autopsy, 3 at surgery and 4 additional cases are presumed to have this defect, after thorough clinical evaluation.

2. The anomalous pulmonary vessels may drain into any one of several systemic or visceral veins, but the most common drainage pathways are into a "persistent left superior vena cava," the coronary sinus or right atrium.

3. Patients with this defect show a mild

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* This operation was performed by F. John Lewis and one of us (R. V.). We wish to thank Doctor Lewis for the privilege of mentioning this case.
cyanosis and a left sternal border, non-diagnostic, systolic murmur.

4. X-ray study may demonstrate the "figure of eight" or "box-like" shape with increased pulmonary vascularity; angiocardiograms may reveal a typical pattern of events.

5. The diagnosis is conclusive, if the right atrial oxygen saturation is equal to or higher than the peripheral arterial saturation.

6. Three of six patients submitted to surgery survived partial or totally corrective procedures.

ADDENDUM

One infant, age 16 weeks and weighing 3.1 Kg., with total anomalous pulmonary venous drainage into the coronary sinus has undergone successful curative surgery utilizing the artificial oxygenator previously described from this clinic. A period of total cardiopulmonary bypass, lasting seventeen and one-half minutes, allowed the orifice of the coronary sinus to be anastomosed to the edges of the atrial septal defect from within the open right atrium.

SUMMARIO IN INTERLINGUA

1. Es presentate 30 casos de anormal retorno pulmonary total. De iste casos, 23 eseva verificate autopticamente e 3 chirurgicamente. In le remanente 4 casos le presentia del mentionate defecto es presumite super le base de caute evolutationes clinici.

2. Le anormal vaso pulmonary pote descargar se in un o altere de pluri venas systemic o visceral, sed le plus commun vias de discarga es a in un "persistente vena cave sinistro-superior," le sinus coronari, o le atrium dextere.

3. Patientes con iste defecto exhibi leve grados de cyanosis e un non-diagnostic murmur systolic al margine sinistro-esternal.

4. Studios roentgenografic pote demonstrar un configuration simile al numero '8' o un cassa con augmento de vascularitate pulmonar. Le angiocardiogramma pote revelar un configuration typic.

5. Le diagnose es conclusiva si le saturation oxygenic del atrio dextere es equal o superior al saturation oxygenic del arterias peripheric.

6. Chirurgia correctional partial o total eseva interprendite in 6 casos. Tres de iste patientes superviveva.

REFERENCES


Total Anomalous Pulmonary Return: An Analysis of Thirty Cases
VINCENT L. GOTT, RICHARD G. LESTER, C. WALTON LILLEHEI and RICHARD L. VARCO

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