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Congenital Anomalies of the Heart

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BY C. W. M. POYNTER

SYNOPSIS

§ I. INTRODUCTION

For a number of years I have been studying the abnormalities of the circulatory system resulting from developmental disturb-

**Note.*—The section arrangement above is for the benefit of the study as a whole and is not meant for the body of the text, which is arranged under an anatomico-embryological classification, see page 8, or a clinical classification, see page 44.

ances. One paper relating to the aortic arches has already appeared, Poynter (1916), and in this one I shall confine myself to the anomalies of the heart. There are few subjects which have attracted more attention among the medical profession than the irregularities in the development of the heart and great vessels, but in the case of the heart the interest is not so much from the teratological side as in the case of the arteries; its developmental defects are frequently of such serious consequence to the life of the individual as to command the attention of the clinician and pathologist, with the result, that these cases have been studied as a pathological problem rather than an embryological one.

I had first intended to report the cases I have had the opportunity of studying, but as my bibliography has grown there has seemed to be no profit in adding more examples to those already reported, since I have observed no new conditions or complexes. In the earlier study it was frequently difficult to find the literature relating to any specific lesion, consequently I have included a full literature list so that the cases which appear as a basis for this study may be readily available to any one desiring to consult them.

Etiology.—The etiology of malformations of the heart cannot be any more definitely stated than that of any other developmental anomaly. Experimental research has taught us that if the normal developmental conditions are interfered with anomalies will result. Such anomalies are not confined to any anatomical system and vary in degree or extent depending on the time and intensity of the action and, no doubt, on other factors as well. Dareste has been able to produce a wide variety of anomalies by varying the temperature of the incubator during the development of the chick, and other investigators have been equally successful with fishes by growing them in various chemical solutions. Many hybrids develop irregularly and the proper application of the X-ray will produce growth disturbances. I have produced a wide variety of the recorded anomalies by incubating chick eggs then subjecting them to a temperature near freezing for a time before continuing the incubation. It would seem that the only, or principal, action of the cold is to inhibit oxidation and so dis-

turb the growth balance between different parts; this conclusion is supported by the fact that I have been able to produce the same results by growing the eggs in an atmosphere low in oxygen. All of these experiments seem to support the theory presented by Mall, namely, that faulty implantation or uncongenial environment of the ovum is responsible for a large part, at least, of the anomalies, in man, with which we are acquainted.

In the case of the heart anomalies we must consider the interference with the growth process as a minor one, notwithstanding the later physiological importance of the resulting irregularity, for the majority of cases of cardiac defects reported show no extensive general growth disturbances. It is impossible to say that the factors which produce cardiac anomalies do or do not produce other lesions, but we at least know that no other lesion is always found in conjunction with them. I have shown in a previous paper (1916) that we must consider variations in development of the aortic arches independently of cardiac disturbances although they are frequently found associated in the same case; and likewise we are forced to conclude that each lesion of the heart must be studied separately, although it is the rule to find two or more lesions associated in the same heart. Stated in another way, defective ventricular septum and stenosis of the pulmonary artery must be considered as separate defects, notwithstanding that they are frequently in association; it would seem that even if the same factor produces both lesions its time of action or intensity must be different for each since each may appear alone.

From time to time the suggestion has been made that heredity plays a part in etiology but I see no evidence that these cases belong in the group with polydactylism and hemophilia. In the cases where two or more children in the same family have defective hearts it seems to me more reasonable, from the experimental evidence at hand, to look on these cases as due to a diseased uterine mucosa in the mother rather than a defective germ plasm.

HISTORICAL

Investigators, as well as clinicians, have been interested in cardiac malformations ever since they were first reported. Their relationship to other anomalies was early recognized and in 1671 Stenson reported a defect of the inter-ventricular septum associated with cleft-palate. Since that time a great many different types of anomaly have been found in association. Rokitansky collected twenty-four defective hearts, a third of which cases had other developmental irregularities. It is impossible to regard the presence of the other anomalies as a mere coincidence, yet there is no constant specific type association.

Senac (1749) saw in congenital cardiac disease only a weakening of the formative influence, while Meckel (1812) pointed out the resemblance of certain of these hearts to those of lower animals, explaining them as reversions; he classified them as amphibian, reptilian, etc., and, although his theory has never been generally accepted, his insistence that arrest of development was the primary cause of these anomalies pointed the way to embryological study which has formed the basis of all modern attempts at scientific classification and explanation. Lee (1880) saw in single and associated anomalies the result of baneful influences acting during the early weeks of pregnancy sometimes selecting one part and sometimes another.

Kreysig (1817) offered the suggestion that fœtal endocarditis was responsible for congenital cardiac disease. This theory has been very widely accepted, particularly by pathologists. It has been considered as affecting not only the early developing heart but the fully formed heart as well. With a better understanding of heart development, the modern tendency is to explain the majority of cardiac irregularities as due to arrest of development caused perhaps by a variety of factors. There still remains a certain proportion of cases in which it is impossible to say that fœtal endocarditis has not been present and has not disturbed the normal course of development; however, the presence of thickened tissues does not necessarily prove that a defect is due to an inflammatory process. The final discussion of these cases must

wait till we have a better understanding of the reaction of embryonal tissues to inflammatory processes.

Rheumatism, tuberculosis and syphilis have all been studied as possible etiological factors but there is no positive evidence that they act even in an indirect way.

Frequency.—As early as 1814 Gintrac suggested that cardiac defects were more frequently to be found in Great Britain than in France and Germany and that they were quite rare in Italy and Holland. This observation seems to be true at the present time with the United States occupying a place of slightly greater frequency than Italy and Holland.

It is very difficult to secure statistics which will throw light on the relative frequency, for without postmortem examination very few cases can be positively identified as congenital defects and many cases present no symptoms calling attention to the heart for perhaps years after birth. Pott (1895) found of 30,000 children from clinics 95 had heart disease and of these 27 were under two years old, so might be considered as congenital; this is but a rude approximation and, as suggested, takes no account of the cases presenting no symptoms at the time of examination, so the number may be greater than this study suggests. Ellis (1905) found that there were 51 cases of all cardiac defects in 3,875 autopsies at the Pennsylvania hospital, or 1.16 per cent.; of this number 42 were patent foramen ovale or persistent patency of the ductus arteriosis.

The question of sex frequency does not seem to be of much significance; there is, however, a slightly greater preponderance of males affected, as the following table shows:

	Cases	Males
Gintrac, 1814	44	63.64%
Friedeberg, 1844	43	67.44%
Stölker, 1864	79	54.00%
Peacock, 1866	110	55.54%
Thérémin, 1895	57	56.14%
Abbot, 1915	513	55.34%

The present study agrees with the above tables, the percentage of males being 55.69 per cent.

§ II. SYMPTOMS

Cyanosis.—One of the subjects inseparably linked with congenital cardiac disease is *cyanosis congenita* or *morbus cæruleus.* Its cause has been the subject of debate over a long period. Paracelsus (1500) mentioned Icteritia Cœlestena and later Chamseau (1789) speaks of Ictère violet, but I believe Senac (1749) was the first to definitely connect cyanosis with anomalies of the heart.

Morgagni (1761) declared that cyanosis was caused by stasis of the blood in the venous system. This theory has been accepted by a large number of investigators, notably, Louis, Bouillaud, Farre, Haase, Peacock and Rokitansky. The idea that cyanosis is due to admixture of arterial and venous blood is probably older than the theory just mentioned and has been very generally accepted; even now it seems to be quite firmly established, notwithstanding the fact that Stillé (1841) showed that admixture of blood may occur without cyanosis and that cyanosis may occur without any admixture. Other theories relating to changes in the blood and tissues extend beyond the scope of this paper and may be consulted elsewhere. The question is not as yet entirely settled; certainly more than half of the cases of congenital cardiac disease do not exhibit cyanosis except for a very short period pro exitu. It is most frequently associated with obstruction of the pulmonary circulation but may be absent in these cases, Mazaroff (1895), and, since it is observed under other conditions than congenital cardiac disease, it would seem that the simple circulatory disturbance due to the lack of a normal functioning heart should not be considered as anything more than a contributing factor in any case.

Clubbed fingers have been observed in many cases of congenital cardiac disease, but are by no means a constant finding even in the cases which have lived past the first decade. The condition is probably due to congestion and toxæmia, Carpenter (1909).

§ III. CLASSIFICATION

Classification of congenital cardiac abnormalities presents many difficulties. A review of the various essays on the subject shows

that many different arrangements have been employed and none seem to be entirely free from objections. It would seem that the ideal arrangement would be one based on the embryological stage at which the developmental disturbance occurred. I believe the first attempt at such a classification was made by Friedeberg (1844), who grouped the anomalies as those belonging to the first stage of development, the second stage, etc. Peacock followed this idea, but added a class of misplacements after the plan of Breschet (1826). Rokitansky (1875) classified his cases on the basis of developmental stages as far as they were then known and Vierordt (1898) followed the same plan. Keith (1909) carried the same idea somewhat further as the result of recent research in cardiac development, particularly of the infundibulum. There are still questions of development which must be cleared up before this plan can be freely applied. There is also the possibility that, with development fully understood and etiological factors less obscure than at present, developmental stages may not seem so important.

Hershfelder (1913) recognized the difficulty of classifying these cases, but seemed to think that an anatomical basis was most satisfactory. Such a purely anatomical arrangement, though quite complete, does not meet all requirements because among other things it does not take into consideration the relation of individual lesions to each other in the same case. It seems evident that the factors which produce developmental disturbances resulting in arrest or delayed development of the different parts of the heart may bring about very different conditions not only of the anatomical structure but of function as well, depending on the embryonal stage in which they are active. A classification based on the various complexes which are found in the postmortem room would certainly be most satisfactory from a clinical standpoint but presents great difficulty in examining and explaining developmentally. Abbott (1915) suggests, what seems to me the best solution of the question, a grouping "on mixed principles," that is, an embryological anatomical basis. I will follow this classification with some unimportant modifications and at the end will give an additional resumé of the cases as they are en-

countered clinically, that is, of the different pathological complexes for, as Hershfelder has pointed out, "Since the lesions are usually produced in groups rather than singly, it is quite as important from a clinical standpoint to recognize these groups and understand their effect upon the circulation as to recognize the individual lesions."

The group of cases here presented total 886 and are taken as largely as possible from the individual cases reported in the literature rather than the studies of museum specimens. After a discussion of each lesion a list of the cases upon which the conclusions are based is appended. The references are given by using the reporter's name and date so, by referring to the literature list at the end, title and publication may be known.

CLASSIFICATION

I. Malformations about the Heart.
  A. Anomalies of the Pericardium.
    1. Deficiency.
    2. Diverticulum.
  B. Misplacements of the Heart.
    1. Internal.
      a. Within the Chest (Dextrocardia).
      b. Within the Abdomen.
      c. Diverticulum.
    2. External (Ectopia Cordis).
      a. Cervical.
      b. Pectoral.
      c. Abdominal.
II. Anomalies of the Heart as a Whole.
  A. Acardia.
  B. Bifid Apex.
  C. Congenital Hypertrophy.
III. Anomalies of the Cardiac Septa.
  A. Interventricular Septum.
    1. Absence of Septum.
    2. Defective at Base.
      a. Below Aorta.
      b. Pars Membranacea.
      c. Elsewhere than Base.
  B. Interatrial Septum.
    1. Absence of Septum.
    2. Anomalies of Septum Primum.
    3. Anomalies of Septum Secundum.

a. Persistent Foramen Ovale.
b. Other Anomalies.

IV. Abnormalities Within the Cavities of the Heart.
- A. Right Atrium
  - 1. Contracture or Dilatation.
  - 2. Accessory Septa.
- B. Left Atrium.
  - 1. Contracture and Dilatation.
  - 2. Accessory Septa.
- C. Right Ventricle.
  - 1. Absence or Contracture.
  - 2. Accessory Septa.
- D. Left Ventricle.
  - 1. Absence or Contracture.
  - 2. Accessory Septa.

V. Abnormalities of the Truncus Communis Arteriosus.
- A. Persistence of the Truncus.
- B. Defective Aortic Septum.
- C. Deviation of the Aortic Septum (Transposition).
  - 1. Simple Transposition.
  - 2. Transposition in Situs Transversus.
  - 3. Transposition of Ventricular Loop.
- D. Aorta Opening from Both Ventricles.
- E. Aorta from Right Ventricle Without Transposition.

VI. Abnormalities of the Pulmonary Trunk.
- A. Pulmonary Stenosis.
  - 1. Of Orifice.
  - 2. Of Trunk.
- B. Pulmonary Atresia.
  - 1. Of the Orifice.
  - 2. Of the Trunk.
  - 3. Absence of Trunk.

VII. Abnormalities of the Aorta.
- A. Aortic Stenosis.
  - 1. Of Orifice.
  - 2. Of Trunk.
- B. Aortic Atresia.

VIII. Anomalies of the Semilunar Valves.
- A. Pulmonary Valves.
- B. Aortic Valves.

IX. Anomalies of the Atrio-ventricular Openings.

X. Anomalies of the Ductus Arteriosus (Persistent Patency).

XI. Anomalies of the Veins Entering the Heart.
- A. Pulmonary Veins.
- B. Systemic Veins.

§ IV.

I. MALFORMATIONS ABOUT THE HEART

A. ANOMALIES OF THE PERICARDIUM

1. *Deficiency.*—Entire absence of the pericardium seems to be unknown except in cases of extensive developmental disturbances in which the heart is found outside of the thoracic cavity. More or less complete absence of the parietal pericardium is a rare anomaly, a slight ridge or fold on the right of the great vessels represents the remains of this layer. In other cases of less pronounced defect the pericardium covers the right half of the heart, while in others the pericardial case is complete except for a small or large foramen on the left causing a communication between the pleural cavity of the left side and the pericardial cavity. The opening is always on the left and, when large, some of these cases have been referred to as cases in which the heart is found in the left pleural cavity. The left phrenic nerve is displaced, running down on the inner chest wall parallel to the internal mammary artery.

The first to suggest an explanation of the anomaly was Keith (1907), who reported a case of small defect and said: "This is clearly a case of patent pleuro-pericardial foramen. Evidently the condition has been produced by the lung bud growing within and expanding the communication between the pleura and the pericardium, for the communication lies immediately ventral to the point at which the lung bud appears." McGarry (1914) points out a difficulty with the above explanation in that the lung bud does not develop until after the separation of the two cavities is complete. He calls attention to the other developmental errors of the cœlom in his case and thinks that the whole is due to some early disturbance interfering with the development of the pleuro-pericardial membrane or its anlage.

Haller (1757) did not believe that such abnormality could occur, and Peacock (1856) thought that some, at least, of the reported cases were not true lack of membrane but universal

adherence of the two layers and that true congenital absence was of rare occurrence.

Two cases in this group deserve special mention, and, as far as I am aware, are unique; in one, by Tait (1868) the pleural cavity was intact while the heart, naked of parietal layer, was in the normal position between the two pleural sacs; the other case, by Turner (1870), showed the parietal pericardium normally developed except that it was not attached to the diaphragm. The latter condition has been observed as normal for the walrus.

The condition of defective pericardial development is not readily diagnosed and seems to be without clinical interest; the causes of death are entirely independent of the condition and the average age is over forty years. Twice as many males as females are affected. Cases studied are by:

de Mortel (1700), Baillie (1791), Walter (1805), Otto (1824), Breschet (1826), Meniere (1826), Wolf (1827), Curling (1839), Baly (1852), Bristowe (1855), Peacock (1866), Powell (1869), Tait (1868), Weisbach (1868), Turner (1870), Bjorsström (1871), Lebec (1874), Faber (1878), Chiari (1880), Hughes (1883), Boxall (1887), Hewson (1895), Bernard (1898), Gay (1899), Schendewolf (1900), Primrose (1901), Saxer (1902), Keith (1906) 2 cases, Perna (1909), Versé (1909), Ebstein (1910), Plaut (1913) 2 cases, Cameron (1914), McGarry (1914). Total 36 cases.

2. *Diverticula of the Pericardium.*—This condition is of little clinical importance but should be remembered in considering mediastinal tumors. Out pocketings of the parietal pericardium occur, sometimes as cysts, and may reach considerable size. The etiology has not been fully worked out but is probably an arrest of development of some of the layers of the pericardial wall. The condition is rarely encountered and is well illustrated by the following:

Speir (1865), Cuffer (1875), Bandy (1879), Coen (1885), Atyas-Maraty (1895). Five cases.

§ V.

B. MISPLACEMENTS OF THE HEART

The heart may be found in the neck or as low as the abdomen; it may be simply misplaced in the thorax (dextrocardia) or it may protrude through a fissure in the ventral body wall (ectopia).

For the convenience of presentation we will divide the cases, after Breschet (1826), into internal and external misplacements.

1. *Internal Misplacements*

(*a*) Those displacements of the heart in the chest are generally known as dextrocardia. They are most frequent of all misplacements of the heart and consist of a transfer of the heart to the right side of the chest in a mirror position to that it normally occupies. When all of the other viscera are also transposed the condition is known as viscerum transversus; this condition was first reported by Ricolanus in 1649 and is of sufficiently frequent occurrence to need no discussion here. The transposition of the heart alone is of much rarer occurrence. When unassociated with other anomalies the condition is without clinical interest and is only discovered when general examinations are made or at postmortem.

Paltauf (1901) has pointed out that all cases are not alike. In one group the heart has simply rotated to the right, so that the dorsal and ventral relations are reversed, the tricuspid ventricle still receiving the venous blood. The other group represent true transposition and are very rare, Graanboom's (1891) case being an example.

The cases of congenital dextrocardia must be distinguished from those cases of misplacement due to some pathological process as contracture of adhesions or pressure of pleuritic effusions. A large number of clinical cases have appeared in the literature in the last two decades since the X-ray has come into such general use in study of chest conditions, some of these cases are no doubt congenital, but none of them are included in this list. Foggie (1910) has an extensive bibloigraphy of clinical cases.

1. (*a*) *Dextrocardia:* Otto (1814), Otto (1816), Breschet (1826), Thore (1845), Mosler (1866), Falck (1877), Smith (1878), Robinson (1881), Ghose (1882), Pope (1882), Jacoby (1884), Bamberger (1888), Chabrely (1888), Grus (1888), Northrup (1888), Grunmach (1890), Sandhop (1890), Becker (1891), Graanboom (1890), Birmingham (1892), Williams (1892), Lochte (1894), Auché & Bouyer (1897), Reifschläger (1897), Petit et Ravant (1898), Dalton (1899), Löwenthal (1900), Paltauf (1901), Baumgarth (1902), Lucchi (1902), Wendling (1903), Carletti (1906), Garrod &

Langmead (1906), Potter (1906), Horaud (1907), Pal (1907), Scandola (1909), Foggie (1910), Benjamin (1911), Bello-Morales (1912). Forty cases.

(*b*) *Heart in the Abdomen.*—These cases are frequently classed with the group of ectopia cordis but I have placed them in this class because the ventral body is normally closed, and until we have a clear idea of the etiology it seems proper to assume that other factors than those causing the cleft in the body wall are responsible for the cardiac misplacement. The diaphragm is defective and the heart is situated in the peritoneal cavity. The condition is very rare but should not be difficult to recognize and is apparently not incompatible with long life. The cases reported by Deschamps (1778) and Ramel (1778) had both passed the thirty-fifth year; Holt's (1897) case was in a child.

(*c*) *Diverticulum of the Heart.*—In these cases the heart is prolonged as a muscular diverticulum from the left ventricle and extending into the abdomen through a defect in the diaphragm. I know of no suggested explanation of the anomaly except that of Gibert which is to the effect that the condition is a type of early aneurysm of the tip of the ventricle which prevents the complete development of the diaphragm. A diverticulum of the right ventricle, Fennell (1901), but which is well above the diaphragm, may possibly belong in this group. Following the work of Peacock I have placed these cases among misplacements, although they are probably in no way related etiologically to the cases of malposition of the whole organ. Cases are from O'Bryan (1837), Thaden (1868), Gibert (1883), Arnold (1894), and Kotter-Aeby (1907), Fennell (1901).

2. *External Misplacements*

These cases are generally described under the title ectopia cordis. The heart protrudes through a median fissure in the ventral body wall and comes, in many cases, to lie on the surface of the body. In a few cases the pericardial sac is complete but in the majority the parietal layer is lacking ventrally, its borders being attached to the borders of the body fissure.

The cause of the misplacement is not known; the first theory

was that the heart developed in an abnormal position and so prevented the closure of the body wall; this is not consistent with what we know of development. It has been suggested that an over-distended heart from increased blood pressure prevented early closing of the sternal bars. Adhesions have been blamed for this as for many other anomalies. The condition has been repeatedly produced in experimental teratology in chicks and seems to have to do with disturbance of the body wall as well as the heart; probably separate factors affect each part, for cases are encountered in which the heart is in position and the sternum is defective.

Breschet recognized three forms: (*a*) cervical, in which the heart is beneath the chin; (*b*) pectoral, in which the heart protrudes through a defect in the sternum; and (*c*) abdominal, in which the cleft extends to the abdominal wall and in which abdominal viscera as well as the heart are protruded. The heart in these cases of ectopia may show maldevelopment intrinsically or, more rarely, may be normal; the condition is incompatible with life, the average age for all cases being but a few hours.

Cases of Ectopia Cordis.

(*a*) *Cervical:* Vaubonnis (1712), de Torres (1741), Breschet (1826).

(*b*) *Pectoral:* Walter (1745), Büttner (1768), Wiedemann (1794), Chaussier (1814), Weese (1818) Walter's case, Haan (1825), Breschet (1826), Pecchioli (1839), Wittstock (1839), Cruveilhier (1841), Jones (1854), Brown (1855), Jones (1855), Frickhöffer (1856), Dotzauer (1857), Gross u. Heim (1859), March (1859), Marchal (1850), Daniel (1860), Forster (1861), Rezek (1868), Obermeier (1869), Schlesenger (1870), Jahn (1875), Déserte (1876), Hodgen (1878), Fischer (1879), Hofmeier (1880), Charpentier (1883), Grant (1896), Barnado (1897) Grant's case, Goode (1904), Ellis (1905), Fox (1909).

(*c*(*Abdominal:* Martinez (1723), Prochaska (1734), Schultz (1763), Wahlbom (1764), Sandifort (1772), Hérold (1787), Fleischmann (1810), Barret (1835), Becker (1839), Mitchel (1844), Follin (1850), Francois-Franck (1877). Total 46 cases.

§ VI.

II. ANOMALIES OF THE HEART AS A WHOLE

A. *Acardia*

Acardia or hemicardia indicates a pronounced disturbance of the heart anlage. Stockard (1915) has greatly added to the interest of this subject by his work on *Fundulus*. He has shown that the growth of the heart may be entirely arrested by the use of the proper percentages of alcohols in the water in which they are hatched.

Of course these cases are not of clinical interest but valuable in indicating the extreme degree in which factors affecting the development of the heart may operate. The condition is generally found in monsters and is naturally incompatible with any extended period of development. Early cases were reported by Mery (1720) and Winslow (1740) and others studied by Näcke & Benda (1907) and Kehrer (1908).

Multiple hearts are probably due to an early division of the heart anlage, then cessation of the malignant influences. An interesting case of double heart has been reported by Collomb (1798) in a human cyclops.

B. *Bifid Apex of the Heart*

In these cases the apices of both ventricles project below the interventricular groove. The condition is usually present in a heart which is otherwise normal. The first case which I have found recorded was by Bartholinus (1654), who described it as "Mucrone non acuto ut fieri solet, sed bifido." In explaining the condition Mall (1912) showed that there is a tendency for the divided apex to remain for some time, as in embryos of 11–25 mm. stage, after the adult form should be assumed; this agrees with a like observation made by Paget (1831), p. 282. Mall considered that the bifid apex is due to arrest of development. The condition is normal for the dugong. The condition is rare, constituting 2.3 per cent. in Abbott's series.

Bartholin (1654), Meckel (1805), Parise (1837), Schattuck (1891), Rolleston (1891), Hare (1902), Carpenter (1908), Mall (1912)

C. *Congenital Hypertrophy*

The condition was first suggested by Paget (1831), who said that the condition is represented by an increase of all of the parts naturally belonging to the heart. Columnæ carnæ are sometimes hypertrophied; in a case reported by Louis (1823) these had so much enlarged as to touch and seem a continuous plane. Peacock does not speak of a congenital hypertrophy although he discusses the early hypertrophy due to destruction in balance of the circulation as in congenital stenosis of the pulmonary artery. Virchow (1896) said that he had seen several cases that he considered due to congenital hypertrophy. Simmonds (1899) reported a case in which the heart at term weighed 44 grams; he suggested that the cause was a growth disturbance in early life. Other cases have been reported by Effron (1903) and Kalb (1906).

§ VII.

III. ANOMALIES OF THE CARDIAC SEPTA

A. INTERVENTRICULAR SEPTUM

1. *Absence of Septum*

Among the anomalies of the internal structure of the heart, defective development of the ventricular septum is most frequently met with. The septum may be completely absent, partially developed or defective at the base. In the last the defect may be of the fleshy or membranous portion.

Schliemann (1831) considered the thin portion of the interventricular septum near the base as a pathological condition; Thurnman (1838) described this portion of the septum as "The highest part of the septum which occupies the angle between the posterior and right aortic valves, is in the human subject formed, not of muscular fibers, but simply of endocardium of the left and right ventricles and strengthened only by the interposition of a little fibrous tissue continuous with that of the aorta." He states that the structure was well known to anatomists, also that it was sometimes defective from malformation. I have been unable to find in the anatomies of the time or those of the previous century

any description of the heart which revealed an understanding or recognition of this portion of the septum.

Huschka (1855) described the thin area on the basis of a study of 300 hearts; he pointed out that it was always present and was a normal structure. He corrected Schliemann's error and claimed that he was the first to discover and describe the structure as a normal part of the heart. Peacock (1855) described its presence normally, designated it as "the undefended space" and so liable to developmental error. He seemed to think at this time that this point was the most frequent seat of septal defect, an error corrected somewhat later. Reinhard (1857) suggested the name "pars membranacea," which has been quite generally accepted.

Rokitansky (1875) studied the development of the septum and on this basis divided the base into a posterior muscular portion, a pars membranacea, and an anterior fleshy portion, the latter being subdivided into an anterior and posterior portion. Defects occur most frequently in the posterior part of the anterior septum (Rokitansky) and less frequently in the pars membranacea When the defect is in the anterior part of the anterior septum Keith has shown that it is an anomaly of the bulbar septum. Defects of the septum near the apex are extremely rare and may be multiple.

Septum defect is frequently encountered in conjunction with stenosis of the pulmonary artery and the question as to which is the primary lesion has been much discussed. Hunter (1786) was the first, I believe, to suggest that the obstruction of the pulmonary circulation prevented the closure of the septum. Other theories have been so frequently discussed that they do not need review here. It must be noted in examining any theory that many examples have been found of each lesion occurring without the other. As I interpret the later experimental work in teratology it reveals a much less interdependence of parts in development than was formerly supposed and suggests a much more obscure disturbing factor than simple mechanics.

The total number of septal anomalies is 594; of these 99 or 16.6 per cent. are total absence of septum, listed as follows:

Schenck (1600), Chemeneau (1699), Wilson (1798), Standert (1805), Farre (1814), Otto (1814), Fleischmann (1815), Meckel (1815), Hein (1816), Kreysig (1817), Marx (1820), Tiedemann (1824), Breschet (1826) two cases, Martin (1826), Mauran (1827), Mayer (1827), Stoltz (1841), Thore (1842, 1843, 1845), Chevers (1846), Crisp (1846), Ramsbotham (1846), Foster (1847), Clark (1848), Bednar (1852), Hale (1853), Buhl (1857), Vernon (1856), Carson (1857), Clar (1858), Bernard (1860), Devilliers (1860), Gulbert (1860), Almagro (1862), Rauchfuss (1864), Playfair (1870), Bradley (1873), Elliot (1877), Martin (1877), Chiari (1879), Pott (1879), Kleinschmidt (1881), Robinson (1881), Bianchi (1882), Turner (1882), Shattock (1883), Clarke (1884), Jacoby (1884), Brewer (1885), Bull (1885), Fussell (1888), Northrup (1888), Ziegenspeck (1888), Revilliod (1889), Sänger (1889), Holt (1890), Preisz (1890), Ruge (1891), Williams (1892), Jellett (1897), Reifschläger (1897), Hunsburger (1898), Keith (1898) 2 cases, Potter (1900), Rudlof (1900), Cautley (1901), Baumgarth (1902), Champeter (1903), Grosse (1903), Wright (1903), Bouchacourt (1904), Lefas (1904), Letulle (1904), Emanuel (1906) 2 cases, Konstantinowitsch (1906), Young (1906), Young & Robinson (1907), Cautley (1908), Paterson (1908), Stokes (1908), Paterson (1909), Wenner (1909), Girauld (1910), Sokolow (1910), Robertson (1911), Penner (1911), Fortman (1912), Knope (1912), Pappenheimer (1913), Rivè et Girard (1913), Gladstone & Russmann (1915).

2. *Defective at Base*

(*a*) *Ventricular Septum Defective Below the Aorta.*—The large majority of interventricular septum defects are of the base and of these the most frequent site of arrested development is the posterior part of the anterior septum (Rokitansky). It has been impossible to determine the percentage of defects in either group because observers so frequently make no report as to the portion of the base involved. This list and the one immediately following represent only a small part of the cases of defective septum reported; for other cases see the clinical classification of ventricular defects.

(*a*) *Ventricular septum defective below aorta:* Howship (1813), Cheever (1821), Cerutti (1827), Lexis (1835), Quain (1856), Löschner & Lambl (1860) 2 cases, Peacock (1860), (1869), Johnson (1872), Rex (1874), Buhl (1878), Pott (1878) 2 cases, Babesiu (1879), Pott (1879) 2 cases, Peacock (1879), O'Sullivan (1880), Cronk (1881), Petit (1881), Revilliod (1882), Grant (1883), Moutart (1883), Hayward (1884), Ménétrier (1884), Dumontpallier (1885), Habershon (1888), Mann (1889), Muhr

(1889), Preisz (1890) 4 cases, Variot et Gampert (1890), Rolleston (1891), Steven (1892), Geipel (1892), Méslay (1895), Eisenmenger (1897), (1898), Griffith (1899), Spalverini et Barbieri (1902), Amenville (1905), Rogers & Fortiscue (1905), Carpenter (1909), Erdmenger (1912), Griffith (1915).

(*b*) *Ventricular septum defective in pars membranacea:* Löschner & Lambl (1860) 2 cases, Crocker (1878) (1879), Stone (1881), Toennies (1884), Willcocks (1886), Pryor (1889), Stadler (1891), Passow (1894), Rheiner (1896), Freyberger (1898), Spalverini & Barbieri (1902), Cowan (1903), Carpenter (1908), Bissell (1913), Heller & Gruber (1914), Morse (1915), Parrura (1916).

(*c*) *Other Defects of the Interventricular Septum.*—Defects in other portions of the septum than the base are very unusual. The cases are generally of multiple openings and may be at any point in the body of the septum. The condition no doubt represents a growth disturbance which was of short duration or sufficiently slight to permit the final normal developmental stages. Interesting cases have been reported by:

Chiari (1881), Toennies (1884), Griffith (1897), Chessman (1905), and Robertson (1911).

§ VIII. ANOMALIES OF THE INTERATRIAL SEPTUM

The atrial septum presents, through its complexities of development, several problems which must be considered in interpreting and classifying anomalies. The cavity of the primitive atrium is divided to form two chambers by a septum which is known as the septum primum. This septum grows downward into the cavity until it unites with the endocardial cushions. Before this union is complete the lower border of the septum is free and the atria communicate below it by an opening which is known as the ostium primum. About the time that the division of the chambers is complete an opening appears in the septum primum which is the foramen ovale. A second septum, the septum secundum, grows downward to the right of the septum primum and some time after birth closes the foramen ovale.

This study indicates that developmental disturbances of the interatrial septum are slightly more than half as frequent as those of the interventricular septum. Some defect of the atrial

septum is present in about 36 per cent. of all cases of heart anomalies. As shown by the number of cases living to an advanced age the disturbance to the circulation, even from a complete absence of the septum, is readily compensated.

B. INTERATRIAL SEPTUM

1. *Atrial Septum Absent*

In these cases the growth disturbance has inhibited the development of both the septum primum and secundum. I have not found the condition as an isolated anomaly, but it is frequently found in conjunction with absence of the interventricular septum. The condition constitutes 16 per cent. of atrial septum disturbances.

1. *Atrial septum absent:* Méry (1700), Wilson (1798), Ring (1805), Standert (1805), Farre (1814), Otto (1814), Meckel (1815), Marx (1820), Cheever (1821), Martin (1826) 2 cases, Cerutti (1827), Mauran (1827), Mayer (1827), Thore (1842) (1845), Crisp (1846), Foster (1846), Ramsbotham (1846), Foster (1847), Darrach (1857), Clar (1858), Bernard (1860), Guibert (1860), Almagro (1860), Cameron (1871), Maier (1876), Marchand (1881), Robinson (1881), Shattock (1883), Jacoby (1884), Bull (1885), Northrup (1888), Chapatot (1889), Birmingham (1892), Probyn-Williams (1894), Cade (1897), Ewald (1898), Hunsberger (1898), Rudlof (1900), Blondel (1901), Griffith (1902), Champeter (1903), Lefas (1904), Chartier (1905), Bernstein (1906), Konstantinowitsch (1906), McCrae (1906), Carpenter (1908), Girauld et Tissier (1910), Metzger (1911), Knope (1912).

2. *Anomalies of the Septum Primum*

In these cases two different conditions must be recognized. When the defect is along the dorsal (venous) wall Ingalls (1907) has pointed out that it seems to be due to development of the septum primum too far to the right, so that instead of passing to the left of the opening of the superior vena cava it passes directly through it and so creates an opening. When the opening is in the lower part of the septum, above the ventricles, it may be explained as a persistence of the ostium primum either through arrest of development of the septum primum or of the endocardial cushions. The latter cases are characterized by a semilunar opening low down in the septum.

It is sometimes impossible to tell in a given case whether the defect is of the septum primum or secundum; in general, however, it may be said that lesions of the septum primum are located on the lower or posterior part of the atrial septum, while those of the fossa ovalis and upper part of the septum are defects of the septum secundum.

Lacroix (1844), Bednar (1852), Smith (1880), Hopkins (1889), Preisz (1890) 3 cases, Hawkins (1891), Ruge (1891) 2 cases, Burke (1902), Dhotel (1902), Wright & Drake (1903), Ebbinghaus (1904), Söldner (1904), Ingalls (1907), Abbott (1910), Morrison (1912), Lusenbacher (1916). Nineteen cases.

3. *Anomalies of the Septum Secundum*

(*a*) *Open Foramen Ovale.*—A patent foramen ovale is so frequently encountered that it should not be considered as an anomaly. In our dissecting rooms the foramen has been found patent in 25 per cent. of the subjects if we consider any condition in which a probe will pass as patency. Bizot (1837) found the foramen patent in 13 per cent. of cases in adults and Adami in 14.5 per cent of 1,374 autopsies. Peacock (1852) explained the closure of the foramen showing that the shifting of the cornua and the drawing up of the valve about the margin of the opening produces a physiological closure even when the finger can be thrust through and the valve so displaced. The fact that an adhesion had not formed between the septum and the valve he did not consider as an anomaly. On the other hand, those cases, so frequently described, in which the foramen is sufficiently open to admit a finger, must be considered as anomalous. The condition of fully open foramen ovale was recognized as an anomaly by Peneau in 1598.

All degrees of failure of closure of the foramen may be seen, but this group consists largely of cases in which there is an absence of the septum ovale. Cases in which the septum contains so many openings as to be spoken of as "cribiform" are included in this list. Twenty per cent. of all cases present no other developmental defect. The condition represents 70 per cent. of all atrial septum defects and is encountered in 23 per cent. of all hearts studied.

(*a*) *Open ovale:* Littre (1700), Widmann (1717), Amyand (1736), du Hamel (1740), Hunter (1783) 2 cases, Moreau (1790), Baillie (1808) 2 cases, Schuler (1810), Langstaff (1811), Dorsay (1812), Jurine (1815) 2 cases, Fleischmann (1815), Gamage (1815), Knox (1815), Meckel (1815), Hein (1816), Howship (1816), Kreysig (1817), Nasse (1811), Pasqualine (1827), Basedow (1828), Graves & Houston (1830), Braune (1833), Monet (1833), Bloxam (1834), Fearn (1834), Lexis (1835), Spittal (1835), Chassenat (1836), Napper (1840), Stolz (1841), Smith (1841), Douglas (1842), Mansfeld (1843), Thore (1843), King (1844), Harrison (1845), Iliff (1845), Shearman (1845), Taylor (1845), Chevers (1846), Hare (1846) 2 cases, Peacock (1846) 2 cases, Dittrich (1849), Toynbee (1849), Johnson (1850), Hale (1853), Blin (1854), Keil (1854), Graily-Hewitt (1856), Quain (1856), Carson (1857), Meyer (1857), Schilling (1857). Baly (1858), Devilliers (1860), Mallwo (1860), Gubler (1861), Heine (1861), Hervieux (1861), Ollivier (1861), Gueniot (1862), Mannkopf (1862), Cocle (1863), Foster (1863), Lebert (1863), Buchanan (1864), Peacock (1864), Rauchfuss (1864), Kussmaul (1866), Vulpian (1868), Hunter (1869), Peacock (1869), da Costa (1870), Hunter (1870), Gelan (1873), Peacock (1873), Landouzy (1874), Lucas (1874), Longhurst (1874), Rex (1874), Bögler (1875), Barlow (1876), Katz (1877), Saundby (1877), Caton (1878), Finlay (1878), Heinman (1878), Johnson (1878), Crocker (1879) 3 cases, Makuna (1879) 2 cases, Pott (1879), Bucquay (1880), Ballet (1880), Chiari (1880), Crocker (1880), Desnos et Callias (1880), Gibier (1880), Lees (1880), Luneau (1880), O'Sullivan (1880) 3 cases, Smith (1880), Tirard (1880), Stifel (1880), Peacock & Ashby (1881), Eskridge (1881), v. Etlinger (1881), Fowler (1882) 2 cases, Livingston (1883), Toupet (1883), Barand, Barry (1884), Bury (1884), Chaffey (1884), Holt (1884), Kruger (1884), Moore (1884), Brewer (1885), Epstein (1886), Vilon et Léveque (1884), Leo (1886), Harris (1887), Hendly (1887), Toupet (1887), Gampert (1889), Klipstein (1890) 2 cases, Mackenzie (1889) 2 cases, Miura (1889), Oliver (1889) 2 cases, Revilliod (1889), Dorning (1890), Hadden (1890), Haw (1890), Monisset (1890), Holt (1890), Griffith (1891), Howard (1891), Lewis (1892), Moore (1891), Turner (1891), Laffitte (1892), Moore (1892), Burgess (1893), Lépine (1894), Nazaroff (1895), Caillé (1896), Frenkel (1896), Monod (1896), Packard (1896), Rheiner (1896), Roth (1896), Siredey (1896), Coyon (1897), Griffith (1896), Jacobson (1897), Hun (1897), Grothe (1898), Lamouroux (1899), Rolly (1899), Armand (1900), Starck (1900), Cautley (1901), Andrews (1902), Ardissonne (1902), Bourlot (1902), Burke (1902) 3 cases, Cautley (1902) 2 cases, Cowan (1903) 3 cases, Delherm et Laignel (1903), Griffith (1903), Gutkind (1903), Schreiber (1903), Tylecote (1903), Cohn (1904), Minkowski (1904), Pritchard (1904), Ellis (1905) 2 cases, Krausse (1905) 2 cases, Rogers (1905), Emanuel (1906) 2 cases, Lucien & Harter (1907), Ohm (1907),

Young & Robinson (1907), Carpenter (1908), Paterson (1908), Carpenter (1909), Gandy et Brulé (1909), Planchu et Gardére (1909), Weiss-Eder (1909), Wenner (1909) 4 cases, Serverog (1910), Boulach (1910), Bókay (1911), Robertson (1911), Boxwell (1912), Erdmenger (1912), Keith (1912), Black (1914), Gasquet (1913), Hebb (1913), Hingar (1913), Pappenheimer (1913), Morse (1915), Monteiro (1917). Two hundred and twenty-five cases.

(*b*) *Other Anomalies of the Septum Secundum.*—The septum ovalis may be cribiform as in the case of Finlay (1878), Smith (1880), Oliver (1889) and others. This is a different anomaly than simple arrest of growth leading to an open foramen ovale, but I have included it in the group above. The septum secundum may fail to develop as in the case of Hebb (1889), or may be defective at other points than in the fossa ovalis. An opening above and anterior to the fossa ovalis may be considered as a defect in the development of the septum secundum. The condition seems to occur with about the same frequency as defect of the septum primum. They represent 7.5 per cent. of all defects of the atrial septum.

Kelly (1869), Peacock (1877), Pott (1878) 3 cases, Chiari (1880), Livingston (1883), Greenfield (1889), Hebb (1889), Sänger (1889), Probyn-Williams (1894), Prezewoski (1896), Keith (1898), Griffith (1899), Moore (1903), Thompson (1903), Carpenter (1908), Wassenbach (1910), Reed (1911). Twenty cases.

§ IX.

IV. ABNORMALITIES WITHIN THE CAVITIES OF THE HEART

A. RIGHT ATRIUM

1. *Contracture or Dilatation*

It seems to me that there may be some question of the propriety of including cases of contracture and dilation among congenital malformations. Many of these cases are of sufficient age and the circulation is so disturbed that it is entirely possible that the cases are simple pathological reactions of the same type with which we are familiar in other cardiac diseases; this applies espe-

cially to the cases of dilation. Cases of congenital dilation and contracture have been reported and it is possible that a chamber may over or under develop, due to factors interfering with growth; however, it seems hardly worth while to burden this study with a group of borderline cases.

2. *Accessory Septa in the Right Atrium*

This consists of a network or delicate membrane originating from the region of the Eustachian or Thebesian valve and extending across the atrium to attach to some point on the septum. Chiari (1897) explained the condition as the mal-development of the valvula venosa sinistra or septum spurium. He was describing particularly the network; a number of observers have since called a similar structure the "Chiari Net or Netz." Other cases characterized by a definite band, Ruge (1891) thought may be a redundant septum primum.

Monet (1833), Thompson (1843), Eskridge (1881), Geffrier (1881), Moore (1882), Cayla (1884), Leo (1886), Ruge (1891), Lépine (1894), Chiari (1897), Swan (1898), Le Count (1901), Ebbinghaus (1904), Lesieur et Froment (1911).

B. ABNORMALITIES OF THE LEFT ATRIUM

1. *Contraction and Dilation*

The left atrium has been the seat of a congenital contraction or arrest of development in cases observed by Peacock (1866), Roach (1880), Packard (1896), and Lamouroux (1899).

I have found no cases in which a dilation suggesting congenital origin has been described.

2. *Accessory Septa in the Left Atrium*

This condition does not seem to have been recognized by the earlier observers. The accessory septum stretches across the atrium dividing off a right posterior superior chamber into which the pulmonary veins open. Borst (1906) considers this septum to be a misplaced septum primum; Griffith (1903a) concludes that the band is a redundancy of the tissue of the valve of the

fossa ovalis, "An exaggeration of a state of affairs not usually recorded as abnormal, viz., the presence of a reticula proceeding from the margins of the valve of the foramen ovale." Williams & Abrekossoff (1911) conclude, "That the muscular portion of the anomalous septum in its posterior right part is a continuous portion of the muscular structure of the right posterior portion of the atrial wall . . . and that the abnormal septum arose from a separation of the atrial wall and that the splitting had its origin in the right angle of the wall."

The following cases have been studied:

Andral (Ref. Paget, 1831), Church (1868), Fowler (1882), Moore (1882), Rolleston (1896), Papillon (1897), Martin (1899), Griffith (1903) 3 cases, Potter & Ransom (1904), Hosch (1907), Stoeber (1908), William & Abrekossoff (1911), Sternberg (1913).

C. RIGHT VENTRICLE

1. *Contracture of the Right Ventricle*

The right heart seems to be especially sensitive to developmental disturbances. The right ventricle is made up of two elements developmentally; Keith has shown that the proximal portion of the bulb becomes incorporated in the wall of the right ventricle to form the conus arteriosus or infundibulum. In many of the cases of contracture the conus portion of the ventricle has suffered from arrest of development and in some cases the remainder of the ventricle is also only slightly developed or absent. The condition is usually accompanied by stenosis or atresia of the pulmonary artery, particularly the orifice.

Meckel (1815), Jacobson (1849), Löschner & Lambl (1860), Mallwo (1860), Gubler (1861), Kussmaul (1866), Demange (1874), Jackson (1875), Crocker (1879), O'Sullivan (1880), Etlinger (1882), Moutart-Marten (1883), Ménétrier (1884), Krönig (1887), Preisz (1890), Variot et Gámpert (1890), Barbillon (1886), Howard (1891), Laffitte (1892), Boquet (1893), Haury (1894), Northrup (1894), Passow (1894), Rheiner (1896), Siredey (1896), Young (1896), Jacobson (1897), Armand (1900), Fennell (1901), Spalverine (1902), Cohn (1904), Ellis (1905), Young & Robinson (1907), Gandy et Brulé (1909), Wassenbach (1910). These cases relate particularly to arrest of development of the conus while in the following the entire ventricle is contracted.

Hare (1852), Heinman (1878), Moore (1891), Leo (1886), Potter (1900), Bernstein (1906), Carpenter (1909).

2. *Accessory Septum in Right Ventricle*

These cases are characterized by a septum in the upper part of the ventricle. They are related to the preceding group in that the developmental disturbance is of the conus, they differ in that the conus may develop proportionately with the rest of the ventricular wall and a portion of the bulb about the bulbar orifice fails to drop out. When the conus is large and the bulbar orifice is small the condition has been referred to as "three ventricles."

Chassinat (1836), Husson (1836), Pegné (1847), Peacock (1869) 2 cases, Peacock (1876), Crocker (1878) (1879), Mackenzie (1879) 2 cases, Nixon (1879), Lees (1880), Kleinschmidt (1881), Stone (1881), Toupet (1883), Mackenzie (1889), Cassell (1891), Ruge (1891), Wendle (1898), Young & Robinson (1907) 2 cases, Cautley (1908), Black (1914).

D. LEFT VENTRICLE

1. *Contracture of the Left Ventricle*

There is difficulty in accounting for the stenosis of the left conus as a simple developmental defect. As already stated the literature shows that congenital disturbances have a marked predilection for the right half of the heart; Rauchfuss stated that the relation of lesions between the right and left sides was as 10:1. Keith thinks that only a small portion of the bulb is incorporated in the left ventricle, so left contractures are not quite comparable to those of the right side. If we consider these cases as a simple arrest of development of the ventricular chamber, as has been suggested, and as has been accepted for the last of the preceding group (contracture of right ventricle), then the left chamber seems to be more prone to mal-development than the right. It seems to me probable that the following list contains more cases in which the bulbar element is responsible for the stenosis than those of arrest of development of the primary ventricular chamber; it seems, however, that this question cannot be settled by examination of the specimens. Even in the cases of an anular thickening of the ventricular wall just below the orifice of the aorta it is difficult to distinguish the congenital cases from those of stenosis due to endocarditis.

Peacock (1846), Parker (1847), Dittrich (1852), Banks (1857), Hare (1853), Peacock (1860), Heine (1861), Virchow (1861), Rauchfuss (1864), Smith (1866), Hallopeau (1869), Allis (1872), Klumpke (1887), Ziegenspeck (1888), Miura (1889), Preisz (1890), Lamouroux (1899), Lowenthal (1900), Schmencka (1907), Cautley (1908), Wienner (1909), Girauld et Tissier (1910), Gladstone & Russmann (1915).

2. *Accessory Septum in the Left Ventricle*

Turner (1895) described a "moderator" band which arose from the anterior septal wall, passed directly across the chamber and was attached to the posterior wall. It does not seem that this condition is analogous to the diaphragm encountered in the right ventricle, more probably it is related to the anomalous cordæ tendinæ which have been occasionally encountered. These run across the ventricle in an irregular way and may be attached to the semilunar valves. In some cases they form a reticular network, suggesting a division of the ventricle into two chambers. They may represent an over stimulus of the process which leads to the development of the normal papilli and cordæ tendinæ; they may be the result of a fœtal endocarditis as suggested by Tawara (1906). They are not incompatible with life but may cause musical murmurs.

Bouillaud (1862), Archer (1878), Bailey (1894), Röhrle (1896), Lewis (1897), Marckewald (1898), Hamilton (1899), Rössle (1902), Winkler (1903), Poscharessky (1904), Gallis (1904), Tawara (1906), Mönckeberg (1908).

§ X.

V. IRREGULARITIES IN THE DEVELOPMENT OF THE TRUNCUS COMMUNIS ARTERIOSUS

Anomalies in this section are frequently classed with arterial irregularities, but belong properly with the heart. Early in the development of the heart there is a slight contracture of the tube at intervals dividing it into five parts, of which the last two are the bulbus cordis and the truncus arteriosus. When the heart assumes an "S" shape the bulbus cordis comes to lie in front of the ventricle the adjacent walls of the bulb and the ventricle fuse, then disappear and the proximal part of the bulbus becomes in-

corporated in the ventricular wall. This adjustment brings the truncus arteriosus against the atrial wall ventrally and its opening over the two ventricles. Later the canal of the bulbus cordis and truncus arteriosus is divided by a septum, the aortic septum, which in the truncus arteriosus produces the aorta and pulmonary artery and in the bulbus grows down to meet the ventricular septum and helps to complete the separation of the ventricles.

The close association of the development of these structures with the general development of the heart is indicated by the frequency with which anomalies of their development are associated with other cardiac irregularities, particularly defective interventricular septum. In this group of cases 20 per cent. of the hearts which showed complete lack of ventricular septum also lacked an aortic septum. Approximately 27 per cent. of all defects of the ventricular septum were associated with irregularities of the aortic septum.

A. Persistent Truncus Communis

As seen above, the aorta and pulmonary artery are developed by the growth of the aortic septum which begins as two distal ridge-like thickenings in the lumen of the truncus communis; these grow out, fuse and so form a septum which normally takes a spiral course. When the septum fails to develop the embryonal truncus communis persists; this common trunk is generally considered as the homologue of the aorta and the case is spoken of as lacking the pulmonary artery, but this interpretation is incorrect. There is a condition of arrest of development of the pulmonary artery to be spoken of later but it may be distinguished from persistent truncus by the fact that the two pulmonary arteries do not spring from the back of the common artery. The persistent truncus may open from the right ventricle as in the early embryo or from both ventricles over the defect in the interventricular septum.

Various degrees of arrest of development less than complete absence of the aortic septum have been reported, the septum may begin its normal development then be arrested; in such cases a truncus communis arises from the heart and in a short distance

divides into aorta and pulmonary artery as in Clark's (1885) case. Rokitansky reported a somewhat more interesting case in which, in addition to the above condition, a remnant of aortic septum was present in the common trunk. Defects of the septum spoken of as communication between the aorta and pulmonary artery no doubt represent minor disturbances in the developmental process.

Persistent truncus communis is always associated with defective development of the ventricular septum and in 70 per cent. there is also defective atrial septum. There are no characteristic clinical symptoms belonging to the condition. While the anomaly adds to the embarrassment of the circulation, it is difficult to say how much since there are so many other factors to consider. In cases occurring in single ventricled hearts the average age was slightly more than two months, while in the cases associated with simply defective ventricular septum the average was slightly more than three years; in the first group the maximum age was ten months, in the latter nineteen years. Cyanosis was present to a greater or less degree in over one third of the cases. The following list shows the condition present in 4.8 per cent. of all hearts studied.

Wilson (1798), Standert (1805), Farre (1814), Meckel (1815), Laurence (1837), Stoltz (1841), Foster (1846–8), Ramsbotham (1846), Clark (1848), Toynbee (1849), Hyernaux (1851), Vernon (1856), Clar (1858), Bernard (1860), Hervieux (1861, Buchanan (1864), Bradley (1873), Pott (1879) 2 cases, Ribinson (1881), Barand, Barry et Rachet (1884), Barbillon (1886), Clark (1884), Muhr (1889), Preisz (1890), Charrin et LeNoir (1891), Bellot (1895), Gallois (1896), Cade (1897), Martin (1897), Petschel (1897), Civatte (1900), Cautley (1902), Spalverini et Barbieri (1902), Wright & Drake (1903), Rispal & Bay (1904), Elsbergen (1905), Krausse (1905), Hand (1908), Wenner (1909) 2 cases, Knope (1912), Dickson (1913).

B. Defect of the Aortic Septum

These cases are characterized by a communication between the aorta and pulmonary artery above the semilunar valves. In almost half of the cases there are other defects of the heart. The average age is 3 years 1 month, the maximum age 25 years. The lesion produces no symptoms (Livingston 1883), and is with-

out clinical interest. The lesion is present in 1.7 per cent. of the hearts studied in this group.

Lediberder (1836), Wilks (1860), Fraentzel (1868), da Costa (1870), Baginsky (1879), Caesar (1880), Chiari (1880), Rickards (1881), Charteris (1883), Livingston (1883), Cayla (1885), Girarde (1895), Cazin (1897), Hektoen (1899), case in Guys Hospital Museum.

C. Transposition of Aorta and Pulmonary Artery

Abnormal positions of the great vessels coming from the heart has generally been discussed under the title of transposition. This condition results from anomalous rotation of the aortic septum. The whole subject was clarified by the wonderful work of Rokitansky (1875), but more recent investigations have simplified his classification and corrected errors which at the time of his work could not be recognized.

Greil (1903) has shown that the bulbus cordis of more primitive forms is represented in the mammalian heart, first forming part of the anterior limb and later becoming incorporated in the wall of the right ventricle and truncus arteriosus. The truncus arteriosus, as shown above, becomes differentiated into the aorta and pulmonary artery by the development of the septum which divides its lumen. Robertson (1913) has shown that, in *Lepidosiren,* the septum described a spiral in a clockwise direction from its distal beginning of 270 degrees. In man this spiral is somewhat less, or about 135 degrees. Distally the septum develops laterally so that the pulmonary artery is dorsal and the aorta ventral in position and as they approach the heart the positions are reversed.

In situs viscerum transversus normal rotation is reversed and this septum together with other structures of the thorax and abdomen present a mirror picture of the normal. When the condition of reversal is limited to the heart it is known as dextrocardia. Lochte (1898) suggested that the ventricular loop could rotate normally or the reverse independently of the remainder of the heart. Lewis (1915) accepted this theory and by reconstructing the different developmental stages in reverse rotation showed that such development is possible. Much experimental

work has been done with the problem of reverse rotation and while the subject still is obscure in many particulars, there seems to be no reason why disturbing factors should not produce a reversal of the ventricular loop without other heart structures being influenced by them.

The various types of transposition seem to be the result of disturbances of normal rotation of the various parts of the heart. Rokitansky (1875) believed that if the concavity of the aortic septum be reversed the relation of the aorta and pulmonary artery would be reversed. His extensive classification failed to take into account the possible movement of the ventricular loop, also he considered a ventricle, even when guarded by a bicuspid valve, as right when it occupied a position on the right side of the viscus. It has since been shown by Lochte and others how important it is to identify the ventricle as tricusped or bicusped. Keith (1909) suggested that the atrophy of the bulbus cordis around the pulmonary artery is responsible for the transposition; Robertson (1913) has shown that his theory is contrary to fact, for while he places the aortic orifice on the right and the pulmonary on the left of the bulbus cordis, exactly the reverse is true in the dipnoan heart. Robertson concludes from a study of lower forms that "If the bulbus cordis develops as a short, straight tube without any disparity in the length of the walls of its middle segment, no torsion of the vessels, that is, the aortico-pulmonary septum, will take place, the middle part of the bulbus cordis where it should occur being so to speak wiped out."

A study of the cases seems to reveal three distinct conditions which may be classified as follows (Poynter 1916):

1. A failure of rotation of the aortic septum in a heart in which the ventricular loop has developed normally. The aorta is in front of the pulmonary artery and opens from a tricusped ventricle. This is the most frequent form of transposition.

2. A failure of rotation of the aortic septum in a heart which has developed by rotation in the reverse direction. The aorta is in front of the pulmonary artery and opens from a tricusped ventricle. The condition is the mirror picture of the previous group and may be found in situs viscerum transversus.

3. The aorta is in front of the pulmonary artery and opens from a bicusped ventricle. This condition is very difficult to explain; the most acceptable theory is that there has been a left-to-right twist of the left around the right ventricle with a corresponding right-to-left deviation of the septum (Lochte).

Clinically the condition presents no special symptoms. It is usually associated with other congenital lesions of the heart, particularly defective interventricular septum; however, nine cases were encountered in this study which presented no other lesion of importance. The condition was found in 12 per cent. of all hearts studied and the cases showed an average age of 3 years. The maximum age was 39 and 75 per cent. of the cases died under one year.

Class 1: Ring (1805), Baillie (1808), Langstaff (1811), Kreysig (1817), Nasse (1811), Müller (1822), d'Alton (1824), Tiedemann (1825), Martin (1826) 2 cases, Martin (1839), Ducrest (1840), Thore (1843), King (1844), Thore (1845), Beck (1846), Parker (1846), Johnson (1850), Ward (1851), Keil (1854), Darrach (1857), Meyer (1857), Reynolds (1857), Cocle (1863), Lebert (1863), de Bary (1864a), Meigs (1867), Arnold (1868), Fränkel (1870), Kelly (1871), Barlow (1876), Maier (1876), Elliot (1877), Janeway (1877), Martin (1877), Babesiu (1879), Crocker (1879), Lees (1879), Mackenzie (1879), Mazatti (1879), Peacock & Ashby (1881), Rickards (1881), Bianchi (1882), v. Etlinger (1882), Holl (1882), Pope (1882), Scott (1882), Turner (1882), Shattock (1883), Durozier (1885), Epstein (1886), Harris (1887), Klumpke (1887), Fussell (1888), Gampert (1889), Mackenzie (1889), Ramm (1889), de Renzie (1889), Revilliod (1889), Dorning (1890), Preisz (1890), Geipel (1892), Hochsinger (1891), Lewis (1891), Saunders (1892), Mirinescu (1893), Bonne (1895), Lochte (1894), Freyberger (1895), Theremin (1895), Caille (1896), Litten (1896), Monod (1896), Rotch (1896), Coyon (1897) 2 cases, Freyberger (1898), Rolly (1899), Rudlof (1900), Starck (1900), Meinertz (1901), Peters (1901), Thiele (1902), Champeter (1903), Cowan (1903), Brain (1905), Ellis (1905), Apert et Brezaud (1906), Emanuel (1906), Young (1906), Lucien & Harter (1907), Young & Robinson (1907) 3 cases, Planchu & Gardére (1909), Wenner (1909) 3 cases, v. Bòkay (1911), Lubs (1911), Robertson (1911), Variot et Moranci (1911), Erdmenger (1912), Fortmann (1912), Keith (1912), Bissell (1913), Pozdyum (1912), Rivet et Gerard (1913), Heller & Gruber (1914).

Class 2: Gamage (1815), Hickman (1869), Schrötter (1870), Ogston (1874), Graanboom (1891), Griffith (1891), Birmingham (1892), McCrae (1906), Stokes (1909), Wenner (1909).

Class 3: Walsh (1842), Stoltz (1851), Gutwasser (1870), Pye-Smith

(1872), Rokitansky (1875), Toennies (1884), Klumpke (1887), Mann (1889), Grunmach (1890), Lochte (1898), Thiele (1902), Wenner (1909), Lubs (1911).

D. Aorta Opening from Both Ventricles (Rechtslage)

There is still some question as to how this group of cases should be placed, but, since the theory advanced by Rokitansky basing their etiology on an irregular development of the aortic septum is still generally accepted, they are placed in this group. The aorta is not carried as far to the left as normal and opens over a defect in the interventricular septum. The condition is generally associated with pulmonary stenosis or atresia, but there is no transposition.

Sandifort (1777), Cailliot (1807), Obet (1808), Meckel (1809), Palois (1809), Howship (1816), Reeland (1818), Cheever (1821), Basedow (1828), Smith (1841), Peacock (1847), Greig (1852), Sturock (1859), Mallwo (1860), Peacock (1860), Hervieux (1861), Peacock (1871), Wyss (1871), Pott (1878), Peacock (1880), Abercrombie (1881), Rickards (1881), Grant (1883), Toupet (1883), Hayward (1884), Moore (1885), Vilon (1884), McKee (1887), Murray (1887), Bingham (1889), Hebb (1889), Pryor (1889), Voelcker (1892), Thomson (1903), Gutkind (1905), Young & Robinson (1907), Carpenter (1908), Hebb (1913), Morse (1915).

E. Aorta Opening from the Right Ventricle

This group may be considered as due to the same factors which produced the last condition, carried to a slightly greater degree. The pulmonary artery is normally placed in relation to the aorta, but there are some irregularities of the vessels in relation to the right ventricle which deserve special mention. In Peacock's (1856) case the aorta arose from the sinus and the pulmonary artery from the infundibulum, in Tooth's (1883) case conditions were just reversed. van Hall (1825) reported a case in which the aorta communicated with the right ventricle through a long canal placed in the muscular wall of the cavity. Hickman (1869) found the condition in a case of situs viscerum transversus. The pulmonary is generally contracted.

Méry (1700), Abernethy (1794), van Hall (1825), Babington (1846), Chevers (1846), Peacock (1846), Peacock (1856), Quain (1856), Meyer

(1857), Mallwo (1860), Heine (1861), Hickman (1869), Hunter (1869), Peacock (1873), Barlow (1876), Pott (1878), Smith (1880), Tooth (1883), Gevaert (1885), Bury (1887), Habershon (1888), Cadet de Gassecourt (1890), White (1891), Saunders (1892), Boquet (1893), Bornier (1907), Hebb (1913).

§ XI.

VI. ABNORMALITIES OF THE PULMONARY TRUNK

A and B. Pulmonary Stenosis and Atresia

Anomalous development, leading to partial or complete closure of the pulmonary artery, is of frequent occurrence among congenital heart irregularities. In this study it is present in almost 34 per cent. of all cases. A variety of conditions is found; the artery may simply be smaller than normal or the stenosis may be confined to the orifice, the semilunar valves may be grown together so that only a minute opening remains or closed forming an obstructive diaphragm, the artery may be shrunken to form a fibrous cord or be entirely wanting.

The presence of this anomaly in conjunction with others of the heart has long been recognized. Morgagni (1728) considered the condition congenital and believed that it was responsible for the persistence of the foramen ovale by the stagnation of blood in the right ventricle. As already pointed out, Hunter saw a connection between the stenosed artery and defective ventricular septum.

Many observers have considered that the condition resulted from fœtal endocarditis and, although this cause is not now generally accepted, it is impossible to prove that certain cases are not produced by an early inflammatory reaction. Peacock and Rokitansky looked on the condition as the result of error in development of the aortic septum. It seems to me that Keith (1909) has offered the most satisfactory explanation in his classification of the cases as partial or complete arrest of developmental expansion of the infundibulum.

Cases of complete obstruction of the pulmonary artery may be compensated for by defective ventricular septum, open ductus arteriosus or, more rarely, enlarged bronchial arteries. Cyanosis

is more frequently found in these cases than any other type of irregularity, but is by no means constant. There seems to be no foundation for the statement, which has been frequently made, that they are particularly subject to pulmonary tuberculosis.

This anomaly has been of more interest clinically than any other congenital cardiac condition. Various symptoms and physical signs have been suggested as characteristic for the lesion; it is, I think, generally admitted that the exact diagnosis of the other anomalies which so frequently accompany it is impossible. Its association with other lesions may be seen by reference to the clinical classification in Section XVII. Many of the statistical studies of age at death, etc., are of slight value because they do not take into account the part played by the associated anomalies.

The frequency relationship of stenosis to atresia has been reported as follows:

Peacock	90:29
Rauchfuss	81:33
Deguise	30:4
Kussmaul	64:26
Abbott	116:34
This Study	158:94

A. Pulmonary Stenosis

The narrowing of the pulmonary trunk may involve the orifice or a part or all of the canal or both. Keith thinks that in 90 per cent. of the cases the right ventricle in its infundibular portion is also involved. I am sure that such an estimate is too high if we are to rely on the published reports in this series. Hypertrophy and dilatation of the right ventricle occur in many cases, but these conditions are by no means constant.

As already pointed out, the position of the aortic opening is changed in many cases so that it may open from the right ventricle.

The same general statements apply equally to atresia of the pulmonary artery, consequently a separate discussion for such cases is not called for. Lists of cases for both stenosis and atresia follow, grouping the cases according to the anatomical position of the lesion.

1. *Stenosis of the pulmonary orifice:* Baillie (1808), Marshall (1830), Lexis (1835), Escalier (1845), Itiff (1845), Shearman (1845), Peacock (1846), Denucé (1849), Quain (1856), Hare (1860), Heine (1861), Mannkoff (1862), Vulpian (1868), Peacock (1869) 2 cases (1871), Deguise (1872), Johnson (1872), Rex (1874), Peacock (1876), Saundby (1877), Crocker (1878), Bussey (1880), Féréol (1881), Petit (1881), Rickards (1881), Revilliod (1882), Livingston (1883), Bury (1884), Kruger (1884), Bull (1885), Dumontpallier (1885), Gevaert (1885), Schrötter (1887), Toupet (1887), Bingham (1888), Luzet (1890), Liegeois (1891), Ruge (1891), Moore (1892), Steven (1892), Freyberger (1895), Nazaroff (1895), Rheiner (1896), Coyon (1897), Hun (1897), Keith (1898), Thomson (1898), Rudlof (1900), Burke (1902), Krausse (1905), Bissell (1913), Milland (1914). Total 54 cases.

2. *Stenosis of the pulmonary trunk:* Chemeneau (1699), Sandifort (1777), Hunter (1783), Abernathy (1794), Palois (1809), Schuler (1810), Dorsay (1812), Knox (1815), Hein (1816), Marx (1820), Müller (1822), Graves & Houston (1830), Bloxam (1834), Napper (1840), Ward & Parker (1846), Peacock (1846) (1849), Maurice (1853), Quain (1856), Peacock (1856), Voss (1856), Carson (1857), Meyer (1857), Peacock (1860), Virchow (1861), Peacock (1870), Wyss (1871), Allis (1872), Assmus (1877), Buhl (1878), Cossy (1878), Crocker (1879) 2 cases, Mackenzie (1879), Peacock (1879), Lees (1880), Peacock (1880), Schantz (1880), Abercrombie (1881), Eskridge (1881), Cadet de Gassecourt (1882), Graves & Houston (1883), Toupet (1883), Hayward (1884), Moore (1884), Durozier (1885), Moore (1885), Sewastianoff (1885), Vilon et Lévéque (1885), Barbillon (1886), Murray (1887), Schule (1888), Hebb (1889), Mann (1889), Moore (1889), Cadet de Gassecourt (1890), Holt (1890), Preisz (1890) 3 cases, Stadler (1890), Variot (1890), Moore (1891), Renvers (1891), Rolleston (1891), Ruge (1891), Brauner (1892), Moore (1892), Voelcker (1892), Pic (1893), Boquet (1893), Lépine (1894), Frenkel (1896), Jacobson (1897), Trepp (1898), Wendle (1898), Starck (1900), Burke (1902) 2 cases, Cautley (1902), Spalverini et Barbieri (1902) 2 cases, Cowan (1903), Griffith (1903), Peiper (1903), Schreiber (1903), Letulle (1904), Minkowski (1904), Ellis (1905), Emanuel (1906), Kühne (1906), Carpenter (1908) (1909), Paterson (1909), Serverog (1910), Wassenbach (1910), Boxwell (1912), Keith (1912), Black (1914), Hebb (1913) 2 cases, Hinger (1913), Pozdyunm (1912), Heller & Gruber (1914). Total 104 cases.

B, 1. *Atresia of the pulmonary orifice:* This list also includes all cases in which the position of the obliteration is not stated. Fleischmann (1815), Meckel (1815), Howship (1816), Cheever (1821), van Hall & Vrolik (1825), Mauran (1827), Fearn (1834), Spittal (1835), Douglas (1842), Mansfeld (1843), Friedeberg (1844), Shearman (1843), Chambers (1846), Chevers (1846), Peacock (1846), Bednar (1852), Wallach (1852), Hare

(1853), Darrach (1857), Graily-Hewitt (1856), Morey (1857), Baly (1858), Gueniot (1862), Peacock (1864), Rauchfuss (1864), Arnold (1868), Peacock (1869) 2 cases, Deguisé (1872), Peacock (1873), Lucas (1874), Heinman (1878), Pott (1878) 2 cases, Raub (1878), Nixon (1879), Pott (1879), Luneau (1880), Stifel (1880), Cronk (1881), Vilon et Lévéque (1884), Ashby (1884), Leo (1886), Vincenzi (1886), Bury (1887), Bingham (1888), Howard (1891) (1892), Burgess (1893), Probyn-Williams (1894), Hunsberger (1898), Civatte (1900), Potter (1900), Symington (1900), Thomson (1900), Andrews (1902), Audry (1902), Bourlot (1902), Bouchacourt et Coudert (1904), Nau (1904), Kühne (1906), Bornier (1907), Hand (1908), Wenner (1909) 2 cases, Hebb (1913), Rivet et Gerard (1913). Total 66 cases.

2. *Pulmonary artery a solid cord:* Hunter (1783), Basedow (1828), Chassinat (1836), Smith (1841), Babington (1846), Crisp (1846), Sturock (1859), Olliver (1861), Hickman (1869), Hunter (1869), Peacock (1871), Turner (1882), Brewer (1885), McKee (1887), Habershon (1888), Griffith (1891), Grothe (1898), McCrae (1906), Young & Robinson (1907), Pappenheimer (1913), Morse (1915). Total 21 cases.

3. *Entire absence of the pulmonary trunk:* Crisp (1846), Almagro (1862), Hunter (1870), Peacock (1879), Grant (1883), Pryor (1889), Gutkind (1903), Hebb (1913) 2 cases. Total 9 cases.

§ XII.

VII. ABNORMALITIES OF THE AORTA

A. Aortic Stenosis and Atresia

These cases are not common and are difficult to distinguish from the results of post-natal endocarditis. It seems reasonable to suppose that the congenital cases are the result of the same type of developmental disturbance which produces similar conditions of the pulmonary trunk. Thérémin collected 17 cases, but it seems probable that a number of these are not of congenital origin. The condition is associated with defective ventricular septum and patent ductus arteriosus. The average duration of life is 2 days. Devillier's (1860) case was an obliteration of the orifice, but those reported by Ziegenspeck (1888), Klipstein (1890), Konstantinowitsch (1906) and Gladstone & Russmann (1915) showed obliteration of the ascending portion as well.

1. *Stenosis of aortic orifice:* Dittrich (1852), Blin (1854), Hare (1860), Allis (1872), Pott (1879), Etlinger (1882) 2 cases. Eppinger (1889).

2. *Stenosis of the ascending trunk:* Parker (1847), Dittrich (1849), de Bary (1846), Maier (1876), Peacock (1877), Geffrier (1881), Holt (1883), Ziegenspeck (1888), Eppinger (1889), Klipstein (1889), Preisz (1890) 3 cases, Lamouroux (1899), Cautley (1908), Wenner (1909) 3 cases.

B. Atresia of the Aortic Arch

These cases do not belong with a study of the heart, but are presented here because they are frequently encountered with congenital heart disease and act physiologically just as the preceding group. The condition consists of the complete or partial obliteration of the aorta just above the opening of the ductus arteriosus, so that the descending aorta receives its blood from the pulmonary artery through the persistent ductus.

Two groups of cases must be recognized. In the first the left subclavian artery arises normally and the anomaly consists of the obliteration of the dorsal root of the left fourth arch and the persistence of the left pulmonary arch. In the second group the left subclavian artery springs from the juncture of the ductus and the descending aorta, and the anomaly is an obliteration of the fourth left arch and dorsal root and the persistence of the left pulmonary arch.

Cases showing a constriction in the region of the fourth arch and dorsal root have been reported by Valenti & Pisenti (1896), Lewis (1903) and Pansch (1905). The condition of atresia may occur without cardiac anomalies, but even so, no case lived longer than 9 months and dyspnœa and cyanosis were frequently observed; cases studied as follows:

Gibert (1832), Greig (1852), Schilling (1857), Smith (1860), Cameron (1871), Barlow (1876), Osler (1880), Potocki (1886), de Renzi (1889).

§ XIII.

VIII. ABNORMALITIES OF THE SEMILUNAR VALVES

These conditions were recognized by the earlier observers, but attracted very little interest; later, when the development of the valve became better understood, more attention was given to the subject.

The causative factors are still in debate; Peacock discussed the possibilities and came to the conclusion that in the cases where the number of cusps was reduced a large majority were of congenital origin but that in certain cases a fusion of the leaflets from endocarditis might occur after birth and be difficult to recognize. Abbott quotes Osler as saying that the cases are due to fusion during fœtal life because: (1) The presence of a low, sometimes half-obliterated raphe behind one of the cusps; (2) compensatory changes in the cusps so that their free edge becomes equal to or even shorter than the single segment, and (3) the fusion of the coronary or right and left segments. Launois et Villaret (1905) concluded that the cases they studied were congenital because there was no histological evidence of inflammation or adhesion. In the cases of supernumerary leaflets there is generally no question of the congenital origin of the case. A few cases have been found in which a band stretches across the lumen of the artery above the valves; Archer (1878) thinks these are anomalous supernumerary cusps.

The classification is simple, as indicated by the arrangement of the following cases:

A. *Pulmonary Semilunar Valves.*

1. *No valves:* Whittle (1889).

2. *One valve:* Moore (1892).

3. *Two valves:* Graves (1841), Ward & Parker (1846), Jackson (1849), Peacock (1849), Buhl (1857), Peacock (1856), Löschner & Lambl (1860), Peacock (1866) 4 cases (1871), Deguise (1872), Buhl (1878), Pott (1878) 2 cases, Mackenzie (1879), Peacock (1879), Borresi (1880), Lees (1880), O'Sullivan (1880), Revilliod (1882), Tooth (1883), Durozïer (1885), Vite (1886), Foot (1888), Hebb (1889), Oliver (1889), Preisz (1890) 2 cases, Fennell (1891), Delitzin (1892), Saunders (1892), Rheiner (1896), Hebb (1897), Minkowski (1904), Carpenter (1906), Kühne (1906), Carpenter (1908), Girauld et Tissier (1910), Wassenbach (1910), Hebb (1913) 2 cases, Monteiro (1917).

4. *Four valves:* Tuineau-de Mussy 1836), Löschner & Lambl (1860), Carter (1873), Wilson (1876), Bernard (1880), Brüninghausen (1880), Tirard (1880), Martenotti (1886), Vite (1886), Delitzen (1892) 3 cases, Virchow (1896), Crowder (1897), Griffith (1897), Launois et Villaret (1905), Reid (1911).

B. *Aortic Semilunar Valves.*

1. *One valve:* Martenotti (1886).

2. *Two Valves:* Spittal (1830), Andral (1838), Blin (1854), Quain & Sibbald (1856), Hare (1860), Greenfeld (1876), Alezias (1880), Tirard (1880), Rickards (1881), Sailer (1885), Vite (1886) 2 cases, Foot (1888), Berger (1890), Preisz (1890) 2 cases, Ucke (1895), Hebb (1897), Rainer (1897), Barbo (1900), Macaigne et Lucien (1912).

3. *Four valves:* Lindenberger (1893) 2 cases.

Summary:

Pulmonary Artery	Aorta	Type Variation
1		No valve
1	1	One valve
44	21	Two valves
17	2	Four valves

§ XIV.

IX. ANOMALIES OF THE ATRIO-VENTRICULAR ORIFICES AND VALVES

In the cases on single atrio-ventricular opening the right is most frequently wanting and the condition generally occurs in bilocular hearts. No very satisfactory explanation has been offered for the irregularity. Mauran (1827a) found a single auricle and ventricle connected by a tricuspid valve, also Keith (1912) reported an arrest of development of the left heart with no atrio-ventricular opening on that side. Potter (1906), Rogers (1905) and Cohn (1904) found the right atrio-ventricular opening lacking in four chambered hearts; other anomalies were present in each case. In cases by Ballet (1880) and Andrews (1902) there was no connection between the right atrium and ventricle, but the right atrio-ventricular opening was into the left ventricle. Martin (1877) reported a long canal from the right atrium opening into the posterior wall of the ventricle and taking the place of the normal orifice.

It was not thought necessary to collect the cases of stenosis because all of the cases examined seemed to be of undoubted inflammatory origin.

Anomalies of the valves are of rare occurrence; Cordell (1884) reported a single mitral valve and Geipel (1903) three cases. Stuhlweissenburg (1912) found a case of double left atrio-ventricular opening, each guarded by valves. Pisenti (1888) reported a congenital anomaly of the tricuspid valve.

Following are cases of single atrio-ventricular opening:

Martin (1826), Mauran (1827), Thore (1842) (1843), Vernon (1856), Martin (1877), Jacoby (1884), Chapatot (1889), Reifschläger (1897), Turner (1891), Grothe (1898), Grosse (1903), Schreiber (1903), Wright & Drake (1903), Cohn (1904), Lefas (1904), Rogers (1905), Kühne (1906), Paterson (1908), Keith (1912).

§ XV.

X. IRREGULARITIES IN DEVELOPMENT OF THE DUCTUS ARTERIOSUS

This structure is a part of the arterial system and is only considered with cardiac anomalies on physiological grounds. Our interest is principally in persistent patency which is the only condition which will be presented here.

The first case of persistent duct was probably not described by Botallenus, after whom it has been called, but by Galenus, who seemed to know of the changes which occur after birth. Fallopius and Cardanus both knew of the duct and that it could remain open after birth. The function seems to have been well described by Harvey in his Motu Cordis en Sanguinis (1648).

The factors which prevent the normal obliteration are no better understood than those which cause the right aortic arch to persist in certain cases, or, indeed, bring about any other developmental irregularity; a number of theories have been advanced which it is not profitable to discuss here.

Persistent patency may occur as an isolated lesion or, more frequently, in conjunction with other congenital cardiac anomalies. In the isolated lesions Goodman (1910) found that females are affected more frequently than males in the ratio of 20:11. The oldest case was 53; death could not be attributed to the open duct, although 7 were persistently cyanotic and 16 suffered from dyspnoea. There seems to be no pathognomonic sign or symptom, yet from the symptom complex a clinical diagnosis may be made. A full review of the clinical diagnosis has been made by Wells (1908). Vierordt thought that the condition of isolated frequency constituted about 4 per cent. of all congenital cardiac disease but Wells thinks the frequency is much greater. The

condition is not incompatible with a fair duration of life, but few cases lived beyond 50 years. As one element in maldevelopment complexes the condition is not of unusual occurrence. In this study open ductus arteriosus is present in slightly more than 25 per cent. of the hearts studied, making it the third lesion in order of frequency; as an isolated lesion it is present in 3.7 per cent. of cases.

Hunter (1783), Standert (1805), Baillie (1808) 2 cases, Obet (1808), Meckel (1809), Langstaff (1811), Howship (1813), Jurine (1815), Kreysig (1817), Reeland (1818), Cheever (1821), Tiedemann (1825), van Hall et Vrolik (1825), Martin (1826), Cerutti (1827), Houzelot (1827), Mauran (1827), Basedow (1828), Graves & Houston (1830), Gibert (1832), Spittal (1835), Chassinat (1836), Smith (1841), Douglas (1842), Babington (1846), Peacock (1846), Rees (1848), Bernutz (1849), Denuce (1849), Peacock (1849), Johnson (1850), Stoltz (1851), Greig (1852), Hare (1853), Keil (1854), Luys (1855), Graily-Hewett (1856), Peacock (1856), Willigk (1854), Darrach (1857), Meyer (1857), Morey (1857), Schilling (1857), Baly (1858), Sturock (1859), Devilliers (1860), Hare (1860), Saunders (1860), Smith (1860), Heine (1861), Metcalf (1861), Ollivier (1861), Almagro (1862), Duroziez (1862), Gueniot (1862), de Bary (1864), Peacock (1864), Arnold (1868), Hickman (1869), Hunter (1869), Cameron (1871), Peacock (1871), Johston (1872), Fogge (1873), Peacock (1873), Lucas (1874), Barlow (1876), Katz (1877), Walsham (1877), Francais (1878), Heniman (1878), Pott (1879), Hildrith (1880), Lees (1880), Malherbe (1880), Osler (1880), Stifel (1880), Tirard (1880), v. Etlinger (1881) 2 cases, Holl (1882), Scott (1882), Turner (1882), Holt (1883), Livingston (1883) 2 cases, Ashby (1884), Coupland (1884), Foulis (1884), Holt (1884), Darier (1885), Epstein (1886), Gilbert (1886), Leo (1886), Potocki (1886), Vincenzi (1886), Harris (1887), Klumpke (1887), McKee (1887), Murray (1887), Hopkins (1888), Ziegenspeck (1888), de Renzi (1889), Klipstein (1889) 2 cases, Miura (1889), Preisz (1890), Howard (1891), Lewis (1891), Moore (1891), Ruge (1891) 2 cases, Hochhaus (1892), Moore (1892), Saunders (1892), Laine (1893), Haury (1894), Probyn-Williams (1894), Stembo (1894), Freyberger (1895), Monod (1896), Rheiner (1896), Coyon (1897), Drasche (1898), Gerard (1898), Grothe (1898), Hunsberger (1898), Lamouroux (1899), Lowenthal (1900), Potter (1900), Starck (1900), Audry (1902), Bourlot (1902), Spalverini et Barbieri (1902), Cowan (1903) 3 cases, Gutkind (1903), Lewis (1903), Moore (1903), Schreiber (1903), Brittauer (1905), Chartier (1905), Chessman (1905), Ellis (1905) 2 cases, Pansch (1905), Apert et Brezaud (1906), Konstantinowitsch (1906), Bornier (1907), Lucien et Harter (1907), Young & Robinson (1907), Carpenter (1908) 2 cases, Cautley (1908), Wells (1908), Carpenter (1909), Wenner (1909) 4 cases,

Weiss-Eder (1909), Bellubekranz (1910) 2 cases, Mead (1910), Kingsley (1911), Robertson (1911), Keith (1912) 2 cases, Pappenheimer (1913), Heller & Gruber (1914), Milland (1914), Morse (1915). Total 183 cases.

§ XVI.

XI. IRREGULARITIES OF THE VEINS ENTERING THE HEART

An exact study of the venous variations will appear later, but since these anomalies are frequently encountered in connection with congenital cardiac disease, I have included the following references for those who may be interested in such irregularities.

A. *Pulmonary Veins.*—Of the pulmonary veins those on the left more frequently unite to form a common trunk before entering the atrium. This condition is probably brought about by the absorption of the original single common trunk into the atrial wall, so that each comes to open separately; this is hardly anomalous.

The upper vein on the right may open into the superior vena cava as in cases by Wilson (1798), Breschet (1826), Otto (1830), Meckel (1820), Gruber (1876), and Ingalls (1907).

According to Federow (1910) the pulmonary veins develop as buds of the sinus venosus after the heart and other vessels have been established; hence these may form irregular connections by union with aberrant branches of vessels already developed. Brown (1913) found an early vascular plexus, splanchnic plexus, in the cat, which is carried down as the lung bud develops and early connects with the sinus venosus and the post cava. By persistence of the post caval tap he explains the case of Park (1912) in which the right pulmonary vein pierces the diaphragm and connects with the post cava. Harrison (1844) reports a case of the pulmonary veins opening into the vena azygos major and Schattock (1883) a case in which they opened into the systemic veins.

The following reports are of the pulmonary veins entering the right atrium: Lacroix (1844), Taylor (1845), Chiari (1880), v. Etlinger (1881), Epstein (1886), Hepburn (1887), and Hick-

man (1869); and Crocker (1879) the homologous condition in situs viscerum transversus.

Duchek (1862) reported a very interesting condition in which the right pulmonary veins opened into the right ventricle.

B. *Systemic Veins.*—Occasionally two superior venæ cavæ are encountered. In these cases the right opens into the right atrium in the normal way, while the left enters the right atrium through the coronary sinus. This is a case of persistence of the left duct of Cuvier and part of the anterior cardinal vein; cases are not infrequent.

Longhurst (1874) reported the inferior vena cava entering the left atrium and Crocker (1879) and Miura (1889) observed the superior vena cava entering the same chamber. Cases in which both superior and inferior venæ cavæ entered the left atrium were reported by Jacoby (1884) and Lucien & Harter (1907). Barbo (1900) saw the superior and inferior venæ cavæ open into the right atrium in a case of situs viscerum transversus.

§ XVII.

CLINICAL CLASSIFICATION

In discussing the difficulties of classification I have already pointed out that to properly understand the congenital heart anomalies it is necessary to study each lesion independently of all others which may be present. The first section of this study has been taken up with the examination of each anomaly which this collection of hearts has furnished, and considering each without regard to any other irregularity which may be, occasionally or frequently, found in association with it.

In this section the cases are regrouped according to the different lesion complexes which they present. I have used the defects of the cardiac septa as the leading lesions simply because they are of frequent occurrence, any other scheme would lead to the same results. Many simple conditions which have been fully treated in the first part are omitted here to avoid repetition. The more exact distinctions in lesions, as the position of a defect of the interventricular septum or of pulmonary stenosis are not at-

tempted, but by a critical comparison of any of the following lists with those in the various previous sections such information can be readily gained.

I. *Cases of Single Ventricle*

(No Other Lesion)

No symptoms seem to be characteristic; one case had cyanosis and one was dyspnœic. The average age was slightly more than 10 months if we exclude Kleinschmidt's case which lived to be 64.

Schenck (1600), Playfair (1870), Chiari (1879), Kleinschmidt (1881), Jellett (1897), Baumgarth (1902), Cautley (1908), Sokolow (1910), Penner (1911), Gladstone & Russmann (1915).

(*a*) *With Defective Atrial Septum.*

Average age 2½ months, excluding one case which lived to be 24 years, persistent cyanosis slightly more frequent than in the preceding.

Otto (1814), Mackel (1815), Tiedemann (1825), Mayer (1827), Thore (1842), Hale (1853), Guibert (1860), Jacoby (1884), Sänger (1889), Preisz (1890), Keith (1898), Rudlof (1900), Cautley (1901), Grosse (1903), Paterson (1908), Girauld et Tissier (1910).

(*b*) *With Patent Ductus Arteriosus.*

Only two cases by Ziegenspeck (1888) and Letulle (1904); the first was complicated by an atresia of the aorta which is not receiving separate recognition in this group, lived 15 days; the second may perhaps have had a congenital stenosis of the pulmonary artery, although I have not so interpreted it; case lived 16 years and died of tuberculosis.

(*c*) *With Atrial Septum Defective and Ductus Arteriosus Persistent.*

Case by Kreysig (1817) lived 22 years; that of Devilliers (1860) and Konstantinowitsch (1906) less than one week, each of the latter had atresia of the aorta.

(*d*) *With Pulmonary Stenosis.*

Chemeneau (1699), Buhl (1857), Emanuel (1906); latter was cyanotic and lived for 7 years, others were fœtuses.

(e) With Pulmonary Stenosis and Open Ovale.

Meckel (1815), Hein (1816), Marx (1820), Carson (1857), Bull (1885), Holt (1890), Paterson (1909).

(f) With Pulmonary Stenosis, Open Ovale and Ductus Arteriosus Persistent.

Fleischmann (1815), Chevers (1846), Crisp (1846), Bednar (1852), Almagro (1862), Rauchfuss (1864), Brewer (1885), Northrup (1888), Ruge (1891), Williams (1892), Hunsburger (1898), Potter (1900), Bouchacourt et Coudert (1904).

Average age 1 year 8 months, maximum 12 years.

(g) With Transposition of Aorta and Pulmonary Artery.

Elliot (1877), Martin (1877), Bianchi (1882), Fussell (1888), Bouchacourt et Coudert (1904), Emanuel (1906), Young (1906), Young & Robinson (1907), Stokes (1908), Fortman (1912), Rivet et Gerard (1913).

Average age about 10 years 3 months.

(h) With Transposition and Open Ovale.

Breschet (1826) 2 cases, Martin (1826), Mauran (1827), Thore (1843) (1845), Arnold (1868), Turner (1882), Shattock (1883), Revilliod (1889), Reifschläger (1897), Champéter de Ribes (1903), Robertson (1911), Pappenheimer (1913).

The following cases of the two preceding groups had in addition an atresia of the pulmonary artery: Mauran, Arnold, Turner, Pappenheimer, and Rivet; it is to be noted in Arnold's case that the ovale was closed but the ductus arteriosus was patent.

(j) With Truncus Communis Arteriosus.

Wilson (1798), Standert (1805), Farre (1814), Stoltz (1841), Ramsbotham (1846), Foster (1847), Clark (1848), Vernon (1856), Clar (1858), Bernard (1860), Bradley (1873), Pott (1879), Robinson (1881), Clarke (1884), Keith (1898), Wright & Drake (1903), Lefas (1904), Wenner (1909), Knope (1912).

The average for this group is 2 months 10 days; all of the cases show a defective atrial septum and in the majority the septum is entirely wanting, see group immediately following which is abstracted from all the preceding cases to cover the title bilocular heart.

II. *Single Ventricle and Single Atrium (Cor biloculare)*

This group contains, in addition to the common irregularities of septum, pulmonary stenosis and atresia, transposition and persistent truncus.

Wilson (1798), Standert (1805), Otto (1814), Meckel (1815), Breschet (1826), Martin (1826), Mauran (1827), Mayer (1827), Thore (1842) (1845), Crisp (1846), Foster (1846), Ramsbotham (1846), Clar (1858), Bernard (1860), Guibert (1860), Almagro (1862), Elliot (1877), Robinson (1881), Schattock (1884), Jacoby (1884), Bull (1885), Turner (1882), Northrup (1888), Hunsburger (1898), Rudlof (1900), Champeter (1903), Grosse (1903), Lefas (1904), Girauld (1910), Knope (1912).

By removing Hunsberger's case aged 12 years and Bull's aged 5 years the average age of the remainder of the group is only 50 days.

III. *Ventricular Septum Present but Defective*

(No Other Lesion)

The average for this group is 10 years 9½ months, the maximum age 65, of a case reported by Meyer.

Cailliot (1807), Meckel (1809), Howship (1816), Pulteney (1818), Louis (1823), Wittcke (1828), Husson (1836), Kürschner (1837), Parker (1847), Peacock (1847), Hare (1846), Greig (1852), Pellizzari (1858), Löschner & Lambl (1860), Hillier (1861), Bouillaud (1862), Vulpian (1868), Kelly (1869), Guillon (1873), Boissel (1875), Palaillon (1876), Descaisne (1877), Coupland (1878), Crocker (1878), Huart (1879), Pott (1879), Bernobie (1881), Chiari (1881), Duret (1881), Grandi (1881), Hadden (1881), Rickards (1881), Orth (1881), Stone (1881), Tooth (1883), Haywood (1884), Chaffey (1885), Barbillon (1886), Willcocks (1886), Foot (1887), Hebb (1889), Audry et Lecroix (1890), Macaigne (1890), Preisz (1890), Dupré (1891), Rolleston (1891), Renvers (1891), White (1891), Tate (1891), Wilbouschewitch (1891), Geipel (1892), Guttmann (1892), Birmingham (1893), Bennetz (1895), Meslay (1895), Railton (1895), Eisenmenger (1897), Gordon (1897), Hebb (1897), Keim (1897), Broesmer (1898), Eisenmenger (1898), Löwenthal (1900), Vinay (1900), Andrews (1902), Audry (1902), Durante (1902), Cowan (1903), Apert (1904), Delepine & Borse (1904), Ferraro (1904), Aneille (1905), Carpenter (1906), Petit (1906), Valz (1907), Carpenter (1909), Norris (1911), Bartels (1913), Black (1914), Hebb (1913), Meyer (1913), Griffith (1915), Gladstone & Russmann (1915), Parrura (1916).

(*a*) *With Defective Atrial Septum.*

Mery (1700), Cerutti (1827), Hare (1846), Mallwo (1860), Mackenzie (1889), Gelan (1873), Laine (1893), da Costa (1870), Bullet (1880), O'Sullivan (1880), Smith (1880), Geffrier (1881), Marchand (1881), Barand & Barry (1884), Chaffey (1884), Epstein (1886), Chapatot (1889), Preisz (1890), Ruge (1891), Turner (1891), Packard (1896), Griffith (1897) (1902), Blondel (1904), Pritchard (1904), Rogers & Fortiscue (1905), Boulach (1910), Foggie (1910), Erdmenger (1912).

(*b*) *With Patent Ductus Arteriosus.*

Obet (1808), Meckel (1809), Greig (1852), Hildrith (1880), Osler (1880), Coupland (1884), Potocki (1886), de Renzi (1889), Haury (1894), Bellubekranz (1900), Chessman (1905), Cautley (1908), Carpenter (1909), Keith (1912).

(*c*) *With Open Ovale and Patent Ductus Arteriosus.*

Reeland & Freiderich (1818), Cameron (1871), Barlow (1876), Lees (1880), Tirard (1880), Klipstein (1889), Lamouroux (1899), Moore (1903), Chartier (1905), Carpenter (1908), Wenner (1909).

(*d*) *With Pulmonary Artery Stenosed.*

The average age for this group is 9½ years, maximum 36 years, or roughly there died in the first decade 66 per cent., in the second 15 per cent., in the third 17 per cent. and in the fourth 1 per cent.

Sandifort (1777), Abernethy (1794), Palois (1809), Marshall (1830), Escalier (1845), Peacock (1846), Ward & Parker (1846), Jackson (1849), Maurice (1853), Peacock (1856), Voss (1856), Meyer (1857), Quain & Sibbold (1856), Hare (1860), Löschner & Lambl (1860), Mallwo (1860), Peacock (1860) (1869) (1870) (1871), Wyss (1871), Deguise (1872), Demange (1874), Jackson (1875), Peacock (1876), Assmus (1877), Buhl (1878), Cossy (1878), Crocker (1878) (1879), Mackenzie (1879), Peacock (1879), Borresi (1880), Bussey (1880), Lees (1880), Peacock (1880), Schantz (1880), Abercrombie (1881), Orth (1881), Féréol (1881), Petit (1881), Rickards (1881), Stone (1881), Cadet de Gassecourt (1882), Revilliod (1882), Moutart-Marten (1883), Hayward (1884), Menetrier (1884), Dumontpallier (1885), Gevaert (1885), Moore (1885), Sewastianoff (1885), Barbillon (1886), Murray (1887), Schrötter (1887), Schule (1888), Bingham (1888), Hebb (1889), Moore (1889), Oliver (1889), Cadet de Gassecourt (1890), Preisz (1890), Stadler (1890), Variot et Gampert (1890), Liegeois (1891), Renvers (1891), Rolleston (1891), Braunner (1892), Steven (1892), Voelcker (1892), Boquet (1893), Haury (1894), Northrup (1894), Passow (1894), Rheiner (1896), Young (1896),

Keith (1898), Wendle (1898), Trepp (1898), Fennell (1901), Cautley (1902), Spalverini (1902), Peiper (1903), Ellis (1905), Young (1907), Thomson (1908), Norris (1911), Keith (1912), Bissell (1913), Hebb (1913), Henger (1913).

(e) With Pulmonary Stenosis and Open Ovale.

Average age at death 9¼ years, maximum 23 years; 32 per cent. of this group showed marked cyanosis as against 25 per cent. in the preceding group.

Hunter (1783), Dorsay (1812), Knox (1815), Bloxam (1834), Lexis (1835), Napper (1840), Itiff (1845), Denucé (1849), Quain (1856), Gubler (1861), Kussmaul (1866), Rex (1874), Crocker (1878) (1879), O'Sullivan (1880), Toupet (1883), Moore (1884), Vilon et Lévéque (1885), Toupet (1887), Lépine (1894), Nazaroff (1895), Siredey (1896), Jacobson (1897), Armand-Delille (1900), Andrews (1902), Burke (1902), Cohn (1904), Krausse (1905), Gandy (1909), Serverog (1910), Black (1914), Hinger (1913), Minkowski (1914).

(f) With Pulmonary Stenosis and Patent Ductus Arteriosus.

Graves & Houston (1830), Peacock (1849) (1856), Johnson (1872), Spalverini et Barbieri (1902), Carpenter (1908), Milland (1914).

(g) With Pulmonary Stenosis, Open Ovale and Patent Ductus Arteriosus.

Graily-Hewitt (1856), Baly (1858), Heine (1861), Rheiner (1896), Starck (1900), Cowan (1903), Schreiber (1903), Ellis (1905).

(h) With Pulmonary Atresia.

Nevin (1789), Lediberder (1836), Chambers (1846), Crisp (1846), Wallach (1852), Peacock (1869), Pott (1878), Nixon (1879), Cronk (1881), Grant (1883), Bury (1887), Habershon (1888), Bingham (1889), Pryor (1889), Howard (1892), Civatte (1900), Symington (1900), Thomson (1900), Kühne (1906), Hand (1908), Hebb (1913).

(j) With Pulmonary Atresia and Open Ovale.

Mansfeld (1843), Shearman (1845), Chevers (1846), Hervieux (1861), Makuma (1879), Luneau (1880), Vilon et Lévéque (1884), Probyn-Williams (1894), Gutkind (1903), Wassenbach (1910), Hebb (1913).

(k) With Pulmonary Atresia and Patent Ductus Arteriosus.

Howship (1813), van Hall (1825), Chaissinat (1836), Friedeberg (1844), Babington (1847), Peacock (1846), Morey (1857), Sturock (1859), Peacock (1869) (1871), Hickman (1869), Peacock (1879), Pott (1879), Vincenzi (1886), McKee (1887), Moore (1892), Löwenthal (1900), Audry (1902).

(*l*) *With Pulmonary Atresia, Open Ovale and Patent Ductus Arteriosus.*

Cheever (1821), Basedow (1828), Graves and Houston (1830), Spittal (1835), Chassinat (1836), Smith (1841), Douglas (1842), Hunter (1869) (1870), Lucas (1874), Stifel (1880), Burgess (1893), Grothe (1898), Bourlot (1902), Bornier (1907), Morse (1915).

The average age at death for this group is 1 year and 9 months. It is interesting to compare the groups of defective ventricular septum, open ovale, patent ductus and stenosis of the pulmonary with the same first three defects and atresia of the pulmonary; in the first, that is, with stenosis, the average age is 7 years 7 months, in the atresia the age is 5 years 4 months.

(*m*) *With Transposition of Aorta and Pulmonary Artery.*

Martin (1839), Parker (1846), de Bary (1864), Babesiu (1879), Toennies (1884), de Renzie (1889), Preisz (1890), Geipel (1892), Saunders (1892), Coyon (1897), Freyberger (1898), Wenner (1909), Bissell (1913).

(*n*) *With Transposition and Atrial Septum Defective.*

Gammage (1815), Nasse (1811), King (1844), Lebert (1863), Maier (1876), Crocker (1880), Peacock & Ashby (1881), Gampert (1889), Mackenzie (1889), Birmingham (1892), Preisz (1890), Rotch (1896), Starck (1900), Thiel (1902), Carpenter (1909), Planchu et Garderé (1909), Keith (1912).

(*o*) *With Transposition and Patent Ductus Arteriosus.*

Walshe (1842), Lees (1879), Scott (1882), Saunders (1892), Monod (1896), Apert et Brezaud (1906), Keith (1912).

With Transposition, Defective Atrial Septum and Patent Ductus.

Martin (1826), Monod (1896), Wenner (1909), Robertson (1911).

(*p*) *With Transposition and Pulmonary Stenosis.*

Durozier (1885), Mann (1889), Grunmach (1890), Meinertz (1901), Pozdyunm (1913).

(*q*) *With Transposition, Pulmonary Stenosis and Open Ovale.*

Ring (1805), Mueller (1822), Kussmaul (1866), Stadler (1890), Birmingham (1892), Griffith (1899), Rolly (1899), Rudlof (1900).

(*r*) *With Transposition, Pulmonary Stenosis, Patent Ductus Art.*

Etlinger (1882), Freyberger (1904), Heller & Gruber (1914).

(*s*) *With Transposition and Pulmonary Atresia.*

Griffith (1891).

(*t*) *With Transposition, Pulmonary Atresia and Defective Atrial Sept.*

Fearn (1834), McCrae (1905).

(*u*) *With Transposition, Atresia, Defective Atrial Sept. and Patent Ductus.*

Darrach (1857), Young & Robinson (1907).

(*v*) *With Persistent Truncus Communis Arteriosus.*

Tonybee (1849), Hyernaux (1851), Pott (1879), Barbillon (1886), Preisz (1890), Charrin et LeNoir (1891), Bellot (1895), Gallois (1896), Petschel (1897), Spalverini et Barbieri (1902), Rispal & Bay (1904), Elsbergen (1905), Dickson & Fraser (1913).

(*w*) *With Persistent Truncus and Defective Atrial Septum.*

Laurence (1837), Buchanan (1864), Barand, Bary (1884), Cade (1897), Martin (1897), Cautley (1902), Krausse (1906), Wenner (1909).

IV. *Foramen Ovale Open*

(With No Other Lesion)

Littre (1700), Widmann (1717), Amyaud (1736), du Hamel (1740), Moreau (1790), Jurine (1815), Pasqualini (1827), Braune (1833), Moret (1833), Ecker (1839), Harrison (1844), Dittrich (1849), Blin (1854), Foster (1863), Landouzy (1874), Longhurst (1874), Bögler (1875), Finlay (1878), Caton (1878), Johnson (1878), Makune (1879), Bucquay (1880), Chiari (1880), Desnos et Callias (1880), Gibier (1880), Smith (1880), Fowler (1882), Hendly (1887), Oliver (1889), Hadden (1890), Haw (1890), Monisset (1890), Ruge (1891), Ardissone (1902), Cautley (1902), Cowan (1903), Delhern (1903), Tylecote (1903), Ohm (1907), Gasquet (1913).

The average age for this group is 28 years, with a maximum age of 70 years.

(*a*) *With Patent Ductus Arteriosus.*

Jurine (1815), Katz (1877), v. Etlinger (1881), Livingston (1883), Holt (1884), Klipstein (1889), Miura (1889), Hektoen (1899), Carpenter (1908), Weiss-Eder (1909).

(*b*) *With Transposition of Aorta and Pulmonary Artery.*

d'Alton (1824), Schilling (1857), Cocle (1863), Dorning (1890), Caillé (1896), Emanuel (1906), Bókay (1911).

(*c*) *With Transposition and Patent Ductus Arteriosus.*

Baillie (1808), Langstaff (1811), Tiedemann (1825), Johnson (1850), Keil (1854), Meyer (1857), Holl (1882), Epstein (1886), Harris (1887), Lewis (1891), Coyon (1897), Cowan (1903), Ellis (1905), Lucien et Harter (1907), Wenner (1909).

(*d*) *With Pulmonary Stenosis.*

Tacconi (1783), Schuler (1810), Craigie (1843), Peacock (1846), Mannkopf (1862), Vulpian (1868), Peacock (1869), Saundby (1877), Bury (1884), Kruger (1884), Laffitte (1892), Moore (1892), Frenkel (1896), Hun (1897), Burke (1902), Griffith (1903), Boxwell (1912).

(*e*) *With Pulmonary Stenosis and Patent Ductus Arteriosus.*

Moore (1891).

(*f*) *With Atresia of Pulmonary Artery and Patent Ductus Arteriosus.*

Hunter (1783), Howship (1816), Ollivier (1861), Gueniot (1862), Peacock (1864) (1873), Heinman (1878), Livingston (1883), Leo (1886), Howard (1891).

V. *Atrial Septum Defective (not Ovale)*

(No Other Lesion)

Lacroix (1844), Taylor (1845), Kelly (1869), Peacock (1877), Hepburn (1887), Hopkins (1889), Greenfield (1889), Hebb (1889), Hawkins (1891), Ruge (1891), Ewald (1898), Henry (1898), Griffith (1899), Dhotel (1902), Thomson (1903), Ebbinghaus (1904), Söldner (1904), Ingalls (1907), Carpenter (1908) (1909), Abbott (1910), Metzger (1911), Reed (1911), Morrison (1912), Lusenbacher (1916).

(*a*) *With Pulmonary Stenosis or Atresia.*

Probyn-Williams (1894), Burke (1902), Bernstein (1906).

VI. *Transposition of Aorta and Pulmonary Artery*

(No Other Lesion)

Beck (1846), Ward (1851), Hickman (1869), Kelly (1871), Ogston (1874), Pye-Smith (1872), Lees (1880), Ramm (1889), Bonne (1895),

Peters (1904), Lubs (1911). The average age is 2 months 4 days if Hickman's case which lived for 28 years is excluded.

VII. *Patent Ductus Arteriosus*

(No Other Lesion)

Houzelot (1827), Gibert (1832), Rees (1848), Bernutz (1849), Luys (1855), Willigk (1854), Hare (1860), Saunders (1860), Smith (1860), Metcalf (1861), Duroziez (1862), de Bary (1864b), Fogge (1873), Walsham (1877), Francais-Franck (1878), Malherbe (1880), Holt (1883), Livingston (1883), Foulis (1884), Darier (1885), Gilbert (1886), Murray (1887), Hochhaus (1892), Stembo (1894), Drasche (1898), Gerard (1898), Wagner (1903), Brittauer (1905), Pansch (1905), Wells (1908), Goodman (1910), Mead (1910), Kingsley (1911).

(a) With Pulmonary Stenosis.

Hare (1853), v. Etlinger (1882), Ashby (1884).

(b) With Transposition of Aorta and Pulmonary Artery.

Stoltz (1851), Klumpke (1887).

VIII. *Trilocular Heart, Two Ventricles and One Atrium*

This group is an abstract from the preceding cases in sections of defective ventricular and atrial septa for the benefit of the clinical reports on "Trilocular Heart."

Mery (1700), Ring (1805), Cheever (1821), Martin (1826), Cerutti (1827), Darrach (1857), Almagro (1862), Cameron (1871), Maier (1876), Marchand (1881), Chapatot (1889), Birmingham (1892), Probyn-Williams (1894), Cade (1897), Ewald (1898), Rudlof (1900), Blondel (1901), Griffith (1902), Chartier (1905), Bernstein (1906), McCrae (1906), Carpenter (1908) (1909), Metzger (1911).

§ XVIII. GENERAL SUMMARY

The preceding study was undertaken as a part of the work of examining the developmental anomalies of the circulatory system; in this relation it does not appear that the heart is any more susceptible to growth disturbances than other parts of the system. The importance which heart anomalies have gained is due to their effect on longevity and individual well being.

Factors disturbing the orderly development of different parts

of the heart may act independently of, and without disturbing the normal processes of, the remainder of the body. When the physiological function of the circulatory system is seriously interfered with by this anomalous development of some portion or portions of the heart it appears that a compensation through arrest or over development of normal parts is attempted in some instances, but with our present knowledge it is not possible to distinguish between primary lesions and secondary effects.

As has been found in the arteries, factors disturbing development of the heart apparently act at a very early age and on very restricted areas. A sufficient number of isolated lesions have been encountered to make it a comparatively easy problem to break up any complex into its individual elements for the purposes of analysis.

We are able to interpret many anomalies in their relation to the orderly course of development, but the actual factors producing them are not revealed or even suggested by this method of study. Prenatal inflammations and endocarditis can be apparently disregarded as primary disturbing factors.

Near 40 per cent. of the hearts presented but a single lesion, while the total number of separate lesions encountered in the 886 hearts was 1,978, or 3½ lesions for each heart showing more than one. Certain complexes were more frequent than others, but no associated lesions were so constant as to be in any way instructive. Other bodily anomalies were occasionally found in association with those of the heart, but none constantly or in any way suggestive of a relation to any specific cardiac lesion.

The statement has frequently been made that the right side of the heart is more susceptible to developmental disturbances than the left; this would certainly seem to be true if we consider only the aortic and pulmonary trunks, but if we consider all anomalies as revealed by this study such a claim cannot be substantiated.

There are no particular diseases to which cases suffering with congenital cardiac lesions are especially susceptible. There are no pathognomic symptoms surely indicating the condition of anomaly. Congenital cyanosis is not a common symptom, indeed it is not present in one third of the cases.

It is impossible to arrive at the frequency of congenital cardiac disease, but it appears certain that the cases are of more frequent occurrence in England and France than in this country.

It is evident that many congenital cardiac lesions and complexes very much interfere with the normal physiological functions, but it is apparent from the examination of longevity in any group of cases that such lesions are only one of several factors in early curtailment of life.

The following list of figures shows the relative frequency of the lesions encountered in this study. These can have no value as general statistics except as this particular group of cases may be representative of the total distribution of such lesions.

	Per Cent.
Anomalies of the pericardium	4.65
Misplacements of the heart	10.0
Anomalies of the heart as a whole	1.3
Anomalies of the ventricular septum	61.0
Anomalies of the atrial septum	35.6
Irregularities in development of the truncus	28.5
Abnormalities of the pulmonary artery	28.5
Abnormalities of the aorta	2.9
Irregularities of the pulmonary valves	7.0
Irregularities of the aortic valves	2.6
Persistent patency of the ductus arteriosus	20.6

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