COR TRIATRIATUM

CONCERNING THE NATURE OF AN ANOMALOUS SEPTUM IN THE LEFT AURICLE

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The term cor triatriatum is open to criticism but has been used by previous writers and has the merit of being superficially descriptive of a heart in which a transverse septum divides the left auricle into an upper and lower chamber. In 1829, Andral stated that he had seen a heart with three auricles, but he did not describe it. The abnormality is clearly uncommon for, since 1868, when Church demonstrated a heart of this kind before the Pathological Society of London, only 17 other cases have been reported. These are summarized in the Table (p. 338). The anomalous septum may be fibrous or fibro-muscular; it may be complete or may have a single or many openings or may be represented merely by a thin cord passing across the auricle. The nature of the membrane determines the effect upon the circulation; it may be compatible with a long life or may lead to early death. There have been several attempts to explain the nature of the pathological septum but, as none of the suggestions has been universally accepted, it is proposed to examine the various theories in the light of another example of the condition.

CASE REPORT

A male infant was admitted to hospital on May 6, 1949, under the care of Dr. J. H. Moseley, to whom I am indebted for the following particulars. Pregnancy had been normal and the mother was delivered one month prematurely, on March 28, of her first baby, an apparently healthy child weighing 5 lb. 6 oz. Breast feeding was initiated satisfactorily and mother and infant were discharged from the maternity unit on the tenth day. At about this time the baby developed a cold and cough and then diarrhoea. Frequent green watery stools were passed during the next three weeks, with consequent mild dehydration and oliguria. During this period there were attacks of dyspnoea in which respiration was grunting; the cry was always feeble. Towards the end of the fifth week the baby began to have choking fits with increasing frequency. In these he gasped for breath, his face became congested, he vomited, and then went pale. He also had occasional cyanotic attacks, perhaps associated with unconsciousness as his mother remarked that his eyes rolled upwards on these occasions. He was referred to hospital because of the progressive severity of the vomiting.

At this time his weight was 7 lb. 1 oz., he looked toxic and grey, and his temperature was 99.4° F. Respiration was rapid (50–120 a minute) and irregular, periods of rapid shallow breathing alternating with deeper grunting respiratory movements. The lips were cyanosed; there was no finger clubbing. The pulse was regular, the rate 150, and paradoxical waxing and waning of the pulse volume was observed. There was slight bulging of the præcordium to the left of the sternum. The apex beat could not be felt but there was an increased area of cardiac dullness extending to the right of the sternum, to the left as far as the anterior axillary line, and upwards to the second left intercostal space. The heart sounds were muffled but no murmurs could be heard. Breath sounds were vesicular and rather harsh in all areas, including the dull area at the left apex, and

rales were audible at both lung bases, especially the left. It was thought that there must be a pericardial effusion and this conjecture apparently received confirmation from X-ray examination (Fig. 1) which showed generalized enlargement of the heart shadow and an area of consolidation at the right lung root. Little help was obtained from other investigations; the urine and the white blood count were both normal, but there was mild anæmia (hæmoglobin 75%). The child was treated with penicillin and sulphonamides but his condition rapidly degenerated. He developed signs of congestive heart failure with venous engorgement, increasing hepatic enlargement, systemic and pulmonary œdema, and eventually died on May 13, 1949.

FIG. 1.—X-ray showing enlargement of heart and fullness of left upper border.

POST-MORTEM REPORT

The post-mortem examination was made by Dr. H. S. Baar who has kindly allowed me to use his notes. Apart from the congenital heart lesion the only pathological changes were bronchopneumonia and congestive changes in the lungs, liver, spleen, and kidneys. The heart weighed 48 g. (normal 21 g.). When the pericardium was opened the anterior surface of the heart was found to consist of a greatly enlarged right ventricle and a left auricle that bulged to the left of the great vessels and the conus of the pulmonary artery. The wall of the right ventricle was thickened and the cavity was dilated. The left ventricle by contrast was so small that it seemed to be merely an appendage of the right ventricle and contributed hardly at all to the left border of the heart. The inter-ventricular septum was intact and the great vessels were in their usual

position; the ductus arteriosus was closed. Tricuspid, pulmonary, and aortic valves were normal in structure but the mitral valve was hypoplastic and the chordæ tendineæ were short. The superior and inferior venæ cavæ and the coronary sinus opened into an apparently normal right auricle. The crista terminalis and the valves of the inferior vena cava and coronary sinus were all properly formed and the fossa ovalis was well defined with a prominent limbus marking its upper margin. The foramen ovale had not sealed and from its upper anterior margin a probe could be passed into the left auricle.

The left auricle was considerably enlarged, and was divided into a larger upper and a smaller lower chamber by a thin greyish-white septum which passed almost horizontally from the upper margin of the fossa ovalis to the lateral wall of the auricle just below the opening of the left inferior pulmonary vein. The septum was roughly triangular in shape and appeared at first sight to be an extension of the valve of the foramen ovale (Fig. 2). Its medial border was inserted into the

FIG. 2.—Dissection of left side of heart. Left auricle divided into two chambers by an anomalous septum; hypoplastic mitral valve, and small left ventricle.

inter-auricular septum along a line which was a little higher at the anterior than at the posterior end. The antero-lateral border, continuous with the wall of the auricle, passed outwards and slightly upwards above the opening into the left auricular appendix; and the postero-lateral border ran downwards and medially to meet the inter-auricular septum. The lower surface of this anomalous septum was slightly convex, and just lateral to its centre was a foramen of a width sufficient to admit the shaft but not the head of an ordinary pin. This foramen provided the only communication between the two chambers. The wall of the upper chamber was very faintly

trabeculated; four pulmonary veins opened through it. There was no communication with the right auricle.

The lower chamber opened into the left auricular appendix, into the left ventricle through the small mitral valve, and into the right auricle through the patent foramen ovale. This last orifice was little, if any, wider than the foramen in the anomalous septum; it lay immediately below the septum and was guarded by a minute crescentic fold of endocardium which probably represented the true margin of the valve of the foramen ovale (Fig. 3).

FIG. 3.—Semi-schematic drawing of interior of lower chamber of left auricle.

Microscopic examination showed that the anomalous septum consisted of elastic and collagenous tissue interspersed with scattered plain muscle fibres. In the centre of the septum, in its outer half only, was a single layer of cardiac muscle which was completely separated from the muscle in the auricular wall (Fig. 4). The appearance of the interauricular septum in its upper part, where it divided the right auricle from the upper chamber of the left auricle, is shown in Fig. 5. It consisted of a double layer of muscle separated by some strands of fibrous tissue.

CLINICAL FEATURES

It is remarkable that this child should have survived for so long after birth and with so few symptoms, for the left ventricle can have received only a very small volume of blood, and less than half of this can have been oxygenated. Yet cyanosis was not observed until the age of five weeks and even then was only intermittent.

The most prominent feature of all cases with symptoms has been respiratory embarrassment secondary to pulmonary congestion, but hæmoptysis has been recorded only once. When the heart fails venous distension, enlargement of the liver, and œdema develop with great rapidity: pulmonary congestion is always severe and death soon follows. Tachycardia, sometimes with cardiac irregularity, is usual; the heart is enlarged and dullness in the second left interspace has often been observed. X-ray examination confirms the clinical picture. Enlargement of the heart is due chiefly to the right ventricle and the left auricle, and the latter may be visible on the left side of the heart in the position usually occupied by the pulmonary conus. Congestive changes in the

FIG. 4.—Section of anomalous septum (left) at junction with auricular wall (right) showing central layer of cardiac muscle. Magnification, $\times 72$.

FIG. 5.—Interauricular septum above the insertion of the anomalous septum showing double layer of muscle. Magnification, $\times 11$.

lungs may be accompanied by areas of atelectasis or bronchopneumonia. There have been no reports of fluoroscopic examination or of electrocardiographic studies or of the more recently developed diagnostic procedures. Cardiograms would doubtless give confirmatory evidence of right ventricular hypertrophy and the P waves might perhaps be enlarged. It is doubtful if catheterization or angiocardiology would give any positive help in diagnosis.

If the condition could be recognized with certainty it would be justifiable to attempt to divide the septum. The easiest approach would be through the auricular appendix of the left auricle, as the septum lies immediately above its internal orifice. Attack by direct vision, such as that used by Brock (1948, 1949) in pulmonary stenosis, would be ideal as this would allow the septum to be identified before division.

The accompanying table, in which the main features of all recorded cases are summarized, indicates that the condition may be a little commoner in males. The anomalous septum usually divides the auricle into an upper or medial chamber which receives the pulmonary veins and a lower or lateral chamber which opens into the auricular appendix and left ventricle.

Only two hearts (Rolleston, 1896, and McNamara *et al.* 1947) fail to conform to the pattern. In these a fibrous cord stretched across the mitral ring; it lay below the opening into the auricular appendix, considerably lower in the auricle than the septum of the other cases. These two will not be considered further. Amongst the remainder, however, similarity of anatomical features is sufficient to suggest a common developmental error and, if this be so, an attempt to explain the malformation can be satisfactory only if it is founded on established facts of embryology and can account for all the features of each of the recorded cases.

THE NATURE OF THE SEPTUM

Various theories have been advanced to account for the abnormal septum. Andral (1829) suggested that the anomaly was an example of incomplete twinning and Fowler (1882) that there was an overgrowth of the valve of the foramen ovale which was displaced laterally until it became adherent to the outer wall of the left auricle, thereby dividing it into two chambers. Neither explanation accords with modern ideas of cardiac development but, in 1896, Griffith anticipated a later view when he pointed out that "There had been a failure in the complete amalgamation of that part of the auricle which is said to be derived from the confluent portions of the pulmonary veins and that derived from the left-hand division of the common auricle of the embryonic heart." The suggestions advanced by Borst (1905) and Hagenauer (1931) require detailed consideration.

Borst's theory. Borst's explanation depends on Born's (1889) concept of an interauricular septum that is formed by the downgrowth of a septum primum, in which, when the ostium primum closes, a secondary ostium develops. This foramen in turn is closed by the growing septum secundum. Borst suggests that the pulmonary venous trunk is displaced to the right and opens between the two septa. The pressure of blood then gradually forces the septum primum to the left, so that it becomes the accessory septum in the left auricle. He considers that the foramen between the upper and lower chamber of the auricle represents a persistent ostium primum and that the intact interauricular septum is derived wholly from the septum secundum (Fig. 6).

This theory has the merit of simplicity and, with minor modifications, has been accepted by all subsequent writers except Hagenauer (1931) and Bredt (1936). Yet there are reasons for doubting its validity. His basic assumption, that the mouth of the pulmonary vein is displaced to the right, is supported by many reports of pulmonary veins draining into the right auricle or superior vena cava, and a few examples of these veins opening astride an interauricular septal defect (Wagstaffe, 1868; Hepburn, 1887; Ingalls, 1907). There is also apparently some evidence that the anomalous septum is the septum primum, for the valve of the foramen ovale is certainly derived from the septum primum, and the anomalous septum has often been described as an extension of this structure. Patten and Taggart (1929), for example, reported a case in which the accessory septum, after forming the valve of the foramen ovale, could be seen to fuse with the septum secundum and could then be traced downwards until it joined the endocardial cushions of the atrio-ventricular

canal. While they accepted Borst's explanation of the anomaly, they drew attention to one of its weaknesses. The fully developed septum secundum is a crescentic structure and the centre of its free margin normally makes little or no contribution to the lower part of the interauricular septum. If the growth of the septum primum should be arrested, so that it fails to reach the atrio-ventricular canal, a defect occurs in the interauricular septum (persistent ostium primum). It seems that, without the help of the septum primum, the septum secundum cannot complete the division of the auricle.

If this be so, then it follows either that the anomalous septum cannot be the septum primum or that its foramen cannot be the ostium primum. Sometimes the foramen may lie at the centre or in the lateral part of the abnormal septum, well away from the atrio-ventricular canal, and it is therefore difficult to believe that it can be the ostium primum. Hosch (1907) suggested that it might represent the ostium secundum, or perhaps a defect quite unrelated to the natural ostia, but clearly the former alternative has to be rejected when there is a fossa ovalis in the interauricular septum.

Perhaps the strongest objection to Borst's theory is that advanced by Hagenauer (1931), who pointed out that the fossa ovalis can be perfectly formed only when the septum primum and septum

FIG. 6.—Borst's theory. (1) Sinus venosus. (2) Septum secundum. (3) Septum primum. (4) Right auricle. (5) Pulmonary vein. (6) Left auricle.

secundum are contiguous. If the anomalous septum were the septum primum and the two septa joined at the lower rim of the ostium secundum, or at its centre, the fossa ovalis could not be properly formed. Yet in practically every case, when viewed from the right auricle, the fossa ovalis has been normal in structure and a well-defined limbus has been present. The histological findings in my case support Hagenauer's views, for the septum dividing the right auricle from the upper chamber of the left auricle consists of a double layer of tissue. Presumably these layers represent the primary and secondary septa which are here in contact above the fossa ovalis and above the point at which the anomalous septum is inserted.

These are reasonable criticisms and, if they are accepted, the view that the foramen in the anomalous septum is the ostium primum must be rejected. Further, the theory cannot explain any case in which the abnormal septum joins the interauricular septum below the upper rim of the fossa ovalis and at least five cases fall into this category. Nor is it possible, if Borst's views are correct, to interpret Tannenbergs' (1930) case as a developmental anomaly. In this heart the accessory chamber opened into the coronary sinus and so drained into the right auricle, but at no time in the growth of the foetal heart does the coronary sinus open both to the right and to the left of the septum secundum.

Hagenauer's theory. Hagenauer (1931) bases his explanation of the malformation on the results of Spitzer's (1923) researches into the embryological development of the heart. He believes

that the anomaly is a consequence of an abnormal junction between the pulmonary vein stem and the primitive heart. He assumes that this junction, instead of being at a right angle, takes place at an acute angle so that, when the sinus venosus dilates, it compresses the pulmonary vein, the mouth of which is eventually obliterated by adhesions. Torsion of the heart is normal, and the sinus venosus migrates to the right and the pulmonary vein to the left. But as the mouth of the pulmonary vein is closed it cannot be properly absorbed into the wall of the left auricle. Instead, it becomes slowly distended by the pressure of blood until it forms the accessory chamber. In Hagenauer's view, therefore, the anomalous septum is derived from the wall of the pulmonary vein (Fig. 7).

This theory has been less widely accepted, although it satisfactorily explains the normal appearance of the fossa ovalis even when the anomalous septum is inserted into its lower rim. If, as Bredt (1936) suggests, the growing septum primum carries with it, for a varying distance into the interior of the heart, the obliterated mouth of the pulmonary vein, it is clear that the pathological septum might join the interauricular septum below, above, or across the centre of the fossa ovalis. Absence of this fossa, such as Faber (1934) describes, undoubtedly indicates defective development of the interauricular septum but it does not necessarily follow that the anomalous septum must be the septum primum. Hagenauer's theory will also explain how the accessory chamber

FIG. 7.—Hagenauer's theory. (1) Sinus venosus. (2) Septum primum. (3) Primitive auricle. (4) Pulmonary vein closed by expanding sinus venosus. (5) Septum secundum.

could communicate with the coronary sinus, for the pulmonary veins sometimes drain into this channel (Sanes, 1939). Indeed, the conception that the septum is a relic of the pulmonary vein wall will account for the findings in all but two of the reported cases; these will be considered later.

Further support for Hagenauer's opinion is given by the lack of trabeculation in walls of the upper chamber; this is the normal condition in the portion of the left auricle that is derived from the pulmonary vein. The anomalous septum in his case contained a double layer of muscle, the upper layer being continuous with the muscular wall of the upper chamber and the lower layer with muscle fibres in the wall of the lower chamber. In several other cases muscle fibres have been observed to pass from the accessory septum into the walls of the auricle. Only a single layer of cardiac muscle could be demonstrated in my case and this was confined to the outer half of the septum. It was not continuous with the muscle in the auricular wall and may have developed independently, though if sections had been taken from other areas continuity might have been demonstrated. However, this would have led to undesirable mutilation of the specimen. The finding of plain muscle fibres beneath the endocardium cannot be advanced as evidence that the septum is derived from the pulmonary vein because, although muscle of this kind is always present in the pulmonary vein, it may also be found in the wall of the normal auricle.

Faber (1934) maintains that Hagenauer fails to prove his basic contention that the pulmonary vein enters the primitive heart at an acute angle. He is also unwilling to believe that compression by the sinus venosus could obliterate the mouth of the pulmonary vein whilst blood is flowing through it; and considers that, if the pressure in the vessel were sufficient to distend the pulmonary

vein stem, the smaller radicles would also be dilated. It is perhaps worth observing that neither the increasing size of the foetal auricles nor the incorporation into their walls of the sinus venosus and pulmonary veins is supposed normally to depend on intracardiac pressure. There is an inherent tendency for the heart to grow and the presence of an additional septum is unlikely to inhibit this characteristic. But Faber's criticisms certainly throw doubt on Hagenauer's interpretation of the developmental defect that leads to the formation of the accessory septum. Additional weight is lent to these criticisms by some observations reported by Preisz (1890). The two cases which he described had defects in addition to the anomalous septum and are important because all the malformations are likely to have originated at the same stage of foetal life. In both there was a high interventricular septal defect with an over-riding aorta and stenosis of the pulmonary conus, lesions which are due to an arrest of growth towards the end of the second month of pregnancy (Keith, 1909; Spitzer, 1923). This implies that the factors concerned with the formation of the anomalous septum are operative considerably later than Hagenauer suggested.

DISCUSSION

Borst's explanation is unsatisfactory because it conforms only doubtfully to known facts about the development of the foetal heart and because it cannot be reconciled with other anatomical features in many of the hearts with an accessory septum in the left auricle. Hagenauer's views of the underlying embryological disturbance cannot be readily accepted, but his belief that the septum is derived from the pulmonary vein explains the anatomical features of all but two cases. In the example of cor triatriatum described by Stöber (1908) the upper chamber in the left auricle consisted of a blind sac in which terminated the veins from the lower lobes of both lungs. The superior pulmonary veins from both sides emptied into the right auricle and from here mixed venous and oxygenated blood passed partly to the right ventricle and partly, through a patent foramen ovale, into the lower chamber of the left auricle. Without considerable modification, Hagenauer's theory will not explain why some pulmonary veins have been obliterated while others have remained patent.

In the heart described by Patten and Taggart (1929) the upper chamber communicated only with the right auricle and Hagenauer's theory cannot account for such an opening through the interauricular septum. Further, the lower chamber received blood partly from the right auricle through a patent foramen ovale and partly from the right superior pulmonary vein direct. As this vein opened astride the septum in the left auricle it is clear that the septum could not have been derived from the wall of the vein.

It has already been suggested that the developmental arrest that is responsible for the anomalous septum occurs towards the end of the second month of pregnancy, rather later than Hagenauer believed. At this period various parts of the primitive heart are being absorbed and it is well recognized that interference with this process may result in the persistence of membranous structures. These are always found at a point of junction between parts of the heart which are of different embryological origin. Examples are the membranous diaphragm found in some cases of pulmonary infundibular stenosis (Brock, 1949), and in some cases of subaortic stenosis (Brown, 1939). Rarely an accessory septum may subdivide the right auricle; it is supposed to be due to a defect at the junction between the inferior vena cava and the right auricle, the septum perhaps representing a persistent right valve of the sinus venosus (Sternberg, 1913; Gombert, 1933; Dubin and Hollinshead, 1944). An anomalous septum in the left auricle is comparable with these malformations and may well be of similar origin.

Our knowledge of the changes affecting the structure of the left auricle towards the end of the second month has advanced but little since 1903 when Griffith, describing a case of cor triatriatum, wrote "Perhaps when the development of the pulmonary veins and their manner of junction with the left auricle is more fully understood, the explanation may become less uncertain." The single pulmonary vein bifurcates into right and left pulmonary veins which in turn divide into superior and inferior branches. These veins are absorbed into the walls of the auricle until all four open

directly into the cavity of the left auricle. Arrested development during the stage of absorption might be expected to result in the formation of a diaphragm that would be, in effect, a remnant of the pulmonary vein wall.

All kinds of anomalies of the pulmonary veins have been described and, although many attempts have been made to explain them, their origin is still uncertain (Duff, 1938; Sanes, 1939; McManus, 1941; Brody, 1942). The pulmonary veins may open into the right auricle, the superior vena cava, the inferior vena cava, the portal vein, or the coronary sinus. Sometimes some of the veins open into the right and others into the left auricle, and then the veins from each lung tend to empty into the auricle of their own side and it is unusual for both superior pulmonary veins to open into the right auricle and both inferior veins into the left auricle as happened in the heart described by Stöber (1908).

It is possible to bring Patten and Taggart's (1929) case into line with the others if it is assumed that the malformation is an example of an anomalous septum occurring in a heart with all the pulmonary veins opening into the right auricle. The right-hand septum would then represent the vestigial remnant of the pulmonary vein wall and its foramen would naturally communicate with the right instead of with the left ventricle. The left-hand septum, which was regarded by the authors as the anomalous septum, would then be the true interauricular septum. The right superior pulmonary vein opens astride this septum, and this would be similar to examples of overriding pulmonary veins described by Wagstaffe (1868), Hepburn (1887) and Ingalls (1907). As the right and left auricles communicate through the foramen ovale, the circulation is precisely the same as in Stöber's case. Indeed, if, in Stöber's case, the superior pulmonary veins, instead of opening directly into the right auricle had opened into the accessory auricle and thence secondarily into the right side of the heart, the malformation would have been identical with that described by Patten and Taggart.

SUMMARY

A heart is described in which an anomalous septum subdivided the left auricle into two chambers. Other reported cases are summarized in the table (see p. 338).

The theories advanced in the past to explain the nature of this septum are critically examined. From an analysis of the previously recorded cases it is concluded that the septum is due to a defect at the junction between the pulmonary veins and the right auricle and is the result of developmental arrest late in the second month of foetal life.

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NINETEEN CASES OF ANOMALOUS SEPTUM IN LEFT AURICLE
SUMMARY OF CLINICAL AND ANATOMICAL FINDINGS

Author	Symptoms	Age at Death	Sex	Anomalous septum	Notes
1. Church (1868)	Dyspnœa; hæmoptysis. Congestive heart failure	38	F.	Membranous. Above F.O. Small central foramen	F.O. ? closed
2. Fowler (1882)	Nil.	42	M.	Fibromuscular. Above F.O. as extension of valve. Muscle continuous with auricular wall. Fenestrated at medial border	F.O. patent. Large heart
3. Preisz (1890)	—	11	F.	Fibrous band. Incomplete	Conus stenosis, over-riding aorta, ventricular septal defect
4. Preisz (1890)	—	3	M.	Fibromuscular. From centre of F.O. Two large foramina	Conus stenosis, over-riding aorta, V.S.D., dextrocardia
5. Griffith (1896)	—	?	?	Fibrous band. Fenestrated	F.O. closed
6. Rolleston (1896)	—	16/12	M.	Fibrous band. Above F.O. as extension of valve. Large foramen	F.O. closed. Left auricle normal
7. Martin (1899)	—	?	?	—	—
8. Griffith (1903)	Nil.	48	M.	—	F.O. closed
9. Potter and Ranson (1904)	"Died of suffocation"	Adult	M.	Below F.O. One large and many small foramina	F.O. closed. Five pulmonary veins
10. Borst (1905)	—	38	F.	Fibromuscular, with central calcification. Below F.O. Muscle continuous with auricular wall. Large medial foramen	F.O. closed. Large right ventricle
11. Hesch (1907)	—	1/12	?	Fibromuscular. From centre of F.O. Incomplete	F.O. closed. Large right ventricle
12. Stöber (1908)	—	Infant	?	Fibromuscular. Above F.O. as extension of valve. Muscle continuous with auricular wall. Intact	F.O. widely patent. Inferior pulmonary veins into closed upper chamber; superior veins into right auricle
13. William and Abrikossoff (1911)	Bronchitis. Heart failure	11	M.	Fibromuscular. Completely hiding F.O. Muscle continuous with auricular wall and septum. Single central foramen	F.O. closed. Large right ventricle
14. Patten and Taggart (1929)	Dyspnœa. Heart failure	3/12	F.	Fibromuscular. Above F.O. as extension of valve. Defect under superior pulmonary vein	F.O. patent. Upper chamber opens into coronary sinus. Large left auricle and R.V. F.O. absent. Large left auricle and right ventricle
15. Tannenbergl (1930) Hagenauer (1931)	Nil.	4/12	?	Muscle continuous with auricular wall. Intact	—
16. Faber (1934)	Dyspnœa. Heart failure	2	M.	Fibromuscular. Muscle continuous with auricular wall. Incomplete	—
17. Bredt (1936)	—	48	M.	Fibrous cord	—
18. McNamara <i>et al.</i> (1947)	Nil.	56	M.	Fibrous cord across mitral ring	F.O. closed. Left auricle normal
19. Parsons (1949)	Dyspnœa. Heart failure	1/12	M.	Fibromuscular. Above F.O. Muscle not continuous with auricular wall. Minute central foramen	F.O. patent. Large left auricle and right ventricle