Acta Veterinaria Hungarica 51 (1), pp. 15–27 (2003)

MULTIPLE CARDIAC ANOMALY IN SHEEP: A CASE STUDY AND REVIEW OF THE LITERATURE

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(Received May 16, 2002; accepted September 11, 2002)

A multiple cardiac anomaly in sheep is presented to show how complicated the result of abnormal development can be. The heart of a 12-hour-old sheep was fixed in 8% formaldehyde solution and subsequently dissected by an anatomical method, and the abnormalities were recorded on digital pictures. The abnormal anatomy is described and compared with the simple developmental anomalies. Developmental abnormalities were found in the distal portion of the bulbus, the aortic arches and the interatrial septum. A special type of the double-outlet right ventricle was observed, which was not a real double-outlet ventricle because it occurred in combination with pulmonary atresia. Coarctation of the aorta was seen, the ductus arteriosus was absent, and there were five vessels originating from the aortic arch instead of one vessel seen in normal cases, as a result of the abnormal development of the aortic arches.

Key words: Newborn sheep, anatomy, multiple cardiac anomaly

During the past 30 years the development of veterinary clinical cardiology has resulted in the recognition and study of many varieties of congenital heart disease in domestic animals. Most of the various types of malformations found in man have been described in domestic animal species. The knowledge of these diseases and the understanding of their aetiology are important for establishing a correct diagnosis. Establishing a diagnosis is usually very difficult because of the numerous varieties of congenital diseases. In most cases these abnormalities do not occur in the form of simple cardiac anomaly but as diverse combinations of multiple anomalies. This case shows how complicated the result of abnormal development can be.

Congenital means inborn or existing at birth. A congenital cardiovascular defect occurs when the heart or blood vessels do not develop normally during the embryonic life.

There are many known cardiac malformations in animals. Many types of atrial septal defects (Jeraj et al., 1980) or ventricular septal defects combined with congenital diaphragmatic hernia (Eyster et al., 1980) have been reported in

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the literature. In one case a double-outlet right ventricle was found in a dog (Wakao et al., 1977). Malformations described in ruminants included anomaly of the right subclavian artery (Paira, 1948), pectoral ectopia of the heart (Eroksuz et al., 1998), three-chambered heart (Loráth, 1987; Loon et al., 1996), tetralogy of Fallot (Nakade et al., 1993) in calves and other types of congenital malformations in sheep (Dennis and Leinold, 1968).

The most common types of cardiac anomalies, grouped by their morphological characteristics, are as follow:

1. Abnormalities of the great vessels

A. Patent ductus arteriosus (PDA)

This is the abnormal persistence of communication between the aorta and the pulmonary artery, which results in an excessive lung blood flow (Patterson et al., 1972).

B. Persistent right aortic arch

This develops when the left fourth aortic arch and the left dorsal aorta are fully obliterated and the right-side vessels persist instead of them. In that case the ductus arteriosus running between the right aortic arch and the pulmonary artery of normal course compresses the oesophagus.

C. Doubled aortic arch

A segment of the right dorsal aorta persists and forms a vascular ring around the oesophagus and trachea together with the left dorsal aorta.

D. Anomaly of the right subclavian artery

This occurs when the right subclavian artery opens from the aortic arch as last vessel after the left subclavian artery (Smollich, 1959).

E. Anomalies of the venae cavae

Left vena cava cranialis. Normally the right vena cardinalis anterior and the vena cardinalis communis develop into a right vena cava cranialis. In this abnormal case the left cardinal vessels develop into the vena cava cranialis and the right vessels degenerate.

Doubled vena cava cranialis. The left abnormal vena cava cranialis originates from the left vena cardinalis anterior, which persists (Lin and Tilley, 1976). The abnormal vessel opens into the sinus coronarius.

Absence of the hepatic segment of vena cava caudalis. From the hindlimbs, the pelvic cavity and abdominal cavity the blood reaches the heart through the vena azygos and the vena hemiazygos (Szatmári et al., 2000), while the hepatic veins directly open into the right atrium.

Doubled vena cava caudalis. In some cases two venae cavae caudales are found under the renal veins, of which the left is usually smaller and develops as a result of the persistence of the left vena supracardinalis.

F. Anomaly of the pulmonary veins

This may be complete or partial (Jeraj et al., 1980). In the former case all pulmonary veins open into the right atrium, into the great vessels running into it, or into both. In the latter case one or more veins open into the right atrium but the majority of them open normally into the left atrium.

2. Obstruction defects

An obstruction is a narrowing that partly or completely blocks the flow of blood. Obstructions called stenosis can occur in heart valves, arteries or veins. The three most common forms of obstruction are pulmonary stenosis, aortic stenosis and coarctation of the aorta. Other forms include valvular aortic stenosis, subaortic stenosis and Ebstein's anomaly.

A. Pulmonary stenosis (PS)

Obstruction to outflow from the right ventricle (Buchanan, 1977), which may be situated in the body of the right ventricle, at the pulmonary valve or in the pulmonary arteries, when it is called pulmonary stenosis. The anatomical features are as follow:

Pulmonary valvular stenosis. This abnormality is characterised by fused or absent commissures. In most cases the valve is a mobile, dome-shaped structure with an orifice that may be tiny and sometimes eccentric.

Dysplastic valves. This anomaly consists of thickened, irregular, immobile tissue and a variably small pulmonary valve annulus, which latter is not always present.

Obstructive muscle. These muscular obstructions occur in the region of the moderator band, at the proximal infundibulum.

Infundibular stenosis. This occurs in the region of the infundibulum.

Peripheral pulmonary stenosis. This anomaly may take several forms. It may be a single obstructive lesion, multiple similar lesions located at approximately the same distance from the pulmonary valve, or symmetrical or asymmetrical pulmonary arterial hypoplasia. Some of these obstructions are centrally lo-

cated, while others are located more distally in the lung. Some involve the main pulmonary artery and the right and left main pulmonary arteries as discrete obstructions. Rarely the pulmonary artery is completely absent.

B. Aortic stenosis (AS)

The aortic valve between the left ventricle and the aorta is narrowed. Aortic stenosis occurs when the aortic valve has not been formed properly. AS has the following types:

Coarctation of the aorta. The aorta is pinched or constricted.

Valvular stenosis. The aortic valve has only one or two flaps.

Subaortic stenosis. This term refers to a narrowing of the left ventricle just below the aortic valve. This condition may be congenital or may be due to a particular form of cardiomyopathy known as idiopathic hypertrophic subaortic stenosis (IHSS).

3. Ebstein's anomaly

This is a congenital downward displacement of the tricuspid valve into the right ventricle. It is usually associated with an atrial septal defect.

4. Septal defects

A. Atrial septal defect (ASD)

The most common type (Selzer, 1975) is the patent foramen ovale caused by the incomplete fusion of the valve of foramen ovale and the septum secundum after birth (Eyster et al., 1980). Ostium primum type is usually connected with several other abnormalities. In this case there is a large opening on the caudal side of the interatrial septum, and usually there is also a cleft in the septal leaflet of the mitral valve. Sometimes the atrioventricular endocardial cushions fail to occur and, as a result, a large opening appears in the centre of the heart, extending into the interatrial and into the membranous part of the interventricular septa. This is termed atrioventricular septal defect. In the ostium secundum type the opening is in the area of the fossa ovalis and includes both defects of the septum primum and secundum. During the formation of the septum secundum, the resorption of the septum primum is abnormal. If the resorption occurs in abnormal locations, the septum primum will be fenestrated. If the resorption of septum primum is larger, the short septum primum will not close the foramen ovale. If the septum secundum develops abnormally, there will be a large foramen ovale, and the normal septum primum will not close it. Many times the developmental abnormalities of the septum primum and secundum occur in combination. The

sinus venosus type of atrial septal defect results from an incomplete absorption of the sinus venosus into the right atrium, and it is usually associated with a partial anomalous origin of the pulmonary veins (Jeraj et al., 1980).

B. Ventricular septal defect (VSD)

The term VSD means an opening in the ventricular septum. The defect may be located anywhere in the septum, may be single or multiple, and may be variable in shape and size. VSD results from a delayed closure of the interventricular septum. The causes of delayed or incomplete closure are unknown.

VSDs classified by their location in the septum are as follow:

Membranous defect. The membranous septum is a small area, immediately adjacent to and under the aortic valve on the left side, contiguous to the septal leaflet of the tricuspid valve on the right side, and overlapping a small segment of the right atrium. Congenital defects of the aortic valve may be associated with membranous defects. The tricuspid valve may be involved in the formation of a ventricular septal aneurysm and may be damaged by the jet of blood passing through a small membranous defect. Rarely, a defect in the membranous septum opens solely into the right atrium, allowing a left ventricular–right atrial shunt (a case of the atrioventricular septal defects). Because the membranous septum is a small area, most defects extend into the immediately adjacent infundibular region.

Muscular defects. They may be located anywhere in the apical, middle, ventral, or dorsal muscular septum and are often multiple. Sometimes these defects seem multiple from the right ventricle because trabeculations overlie a large defect that is discovered to be single from the left ventricular side.

Infundibular (subpulmonary) defects. These are located under the pulmonary valve when viewed from the right side, and when from the left they are immediately beneath the aortic valve. The right aortic valve flap often prolapses into the ventricular defect.

Endocardial cushion type of defects. These are located beneath the tricuspid valve, extended to the tricuspid valve ring, and they occupy the area where an atrioventricularis communis opening would be found (a type of the atrioventricular septal defects, where the defect extending to the right caudoventrally extends to the axis of the heart, too).

C. Eisenberger's complex

This is a ventricular septal defect coupled with pulmonary high blood pressure (right to left shunt), an enlarged right ventricle. It may also include a malpositioned aorta (an overriding aorta).

5. Combined congenital cardiac anomalies

A. Tetralogy of Fallot

This anomaly has four components. The two major ones are: (1) a large ventricular septal defect, and (2) a stenosis at or just beneath the pulmonary valve. The other two components are: (3) the right ventricle is more muscular than normal, and (4) the aorta lies directly over the ventricular septal defect.

The theory is proposed that the main problem is underdevelopment of the pulmonary infundibulum, all other features of the disease being secondary.

The ventricular defect is located in the membranous septum, is subaortic, sometimes extending into the subpulmonary region. Most patients with a tetralogy of Fallot have hypoplasia of the pulmonary annulus. Over time the obstruction of the pulmonary outflow tract becomes more severe, sometimes progressing to pulmonary atresia. Usually the pulmonary valve is deformed and often it is stenotic. Occasionally, the pulmonary valve is represented by only cushions of tissue. The pulmonary arteries are usually small and asymmetrically formed, with peripheral stenosis. The main pulmonary arteries may be more hypoplastic than the larger distal vessels. Obstruction at the origin of the left main pulmonary artery, at the insertion of the ductus arteriosus, is common. While the main pulmonary arteries and the trunk of the pulmonary artery are hypoplastic or small, the ascending aorta is large. In an extreme form of the overriding, both great arteries arise from the right ventricle; this status is called double-outlet right ventricle.

B. Transposition of great arteries

The position of the pulmonary artery and the aorta are reversed. The aorta is connected to the right ventricle, and the pulmonary artery is connected to the left ventricle.

C. Tricuspid atresia

In this condition there is neither an opening of the right atrioventricular orifice nor a tricuspid valve. This means that no blood can flow from the right atrium into the right ventricle. As a result, the right ventricle is small and not fully developed. The patient's survival depends on there being an atrial septal defect and usually a ventricular septal defect.

D. Pulmonary atresia

No pulmonary valve exists, and thus blood cannot flow from the right ventricle into the pulmonary artery. The right ventricle acts as a blind sac that may stay small and not well developed. The tricuspid valve is often poorly developed, too. This is combined with an atrial septal defect.

E. Truncus arteriosus

This is a complex malformation where only one artery arises from the heart and forms the aorta and pulmonary artery. It includes closing a large ventricular septal defect, detaching the pulmonary arteries from the large common artery, and connecting the pulmonary arteries to the right ventricle with a tube graft.

F. Total anomalous pulmonary venous connection

The pulmonary veins are not connected to the left atrium. Instead, they drain through abnormal connections to the right atrium. Usually it is combined with an atrial septal defect.

Materials and methods

A newborn sheep that died 12 hours after birth was examined. The heart was fixed in 8% formaldehyde solution. The right and left ventricles, the interventricular septum and its parts, the valves, the atria and the interatrial septum, the aorta and the pulmonary artery, and finally the pulmonary veins were studied.

The first incision on the heart was made along the sulcus paraconalis, on the right side thereof. This was followed by opening the pulmonary artery. Subsequently the heart was turned over and the wall of the right ventricle was opened, close to the septum, up to the sulcus coronarius. Thus the right ventricle was exposed in its entirety.

The second incision was made on the left side of the sulcus paraconalis, deep into the left ventricle, then the heart was turned over and the wall of the left ventricle was cut open, close to the septum, up to the sulcus coronarius. The heart was turned over again and the aorta was also opened with a pair of scissors led close to the septum.

Subsequently the heart was turned over again, and first the right atrium was opened with an incision made parallel to the sulcus coronarius up to the end of the auricle and then, by an incision made perpendicularly to the first incision, up to the tuberculum intervenosum, from where the walls of both venae cavae were opened.

After the right atrium the left atrium was also opened by an incision made parallel to the sulcus coronarius.

The heart exposed in this way became suitable for anatomical examination.

Results

The trunk of the pulmonary artery originates ventrally from the aortic opening, from the right ventricle (Figs 2A and 2E). The pulmonary opening is small, the three semilunar valves are present and movable. The pulmonary trunk

is short, after 2.5 cm it disappears (Figs 1B and 1C). The aorta originates from the right ventricle (Fig. 1).

In this case five vessels originate from the aortic arch (Figs 1D and 1E). At the beginning of the aortic arch the right and left pulmonary arteries (Fig. 1E) originate, the next one is the right brachiocephalic artery, followed by the left common carotid artery and finally the left subclavian artery. At the level of the common carotid artery there is a large constriction of the aorta (Fig. 1D).

In the ventricle from the right side there is a large defect, localised at the level of the septum membranaceum (Fig. 2C), situated high. From the left side the ventricular septal defect is visible, separated from the left atrioventricular opening by the septal leaflet of the mitral valve (Fig. 2D). The supraventricular crest is normal (Fig. 2C), and it breaks the continuity between the aorta and the wall of the right ventricle. The septal leaflet of the tricuspid valve is cleft, attached to the margin of the defect with a few chordae tendineae (Figs 2 C, 2E and 2F).

In the atrium, from the left side the septum secundum and a large foramen ovale (Fig. 2A) are visible, while on the right side of the septum secundum there is the small septum primum (Fig. 2B).

The pulmonary veins arise normally from the left atrium (Fig. 1F).

Discussion

In the heart studied in this case, the pulmonary artery opens ventrally from the aortic opening, and each of the two great vessels opens from the right ventricle. This is caused by the asymmetrical development of the truncus arteriosus, because the distal part of the bulbus is malformed.

Distal malformations of the bulbus include the tetralogy of Fallot, the double-outlet right ventricle (Witham, 1957), transposition of great vessels, and the intermediate cases (Anderson et al., 1974).

In the case of tetralogy of Fallot the aortic valves are dextroposed, and so the aortic valve is in fibrous continuity with the mitral valve. The aortic valve overrides the interventricular septum and there is a large ventricular septal defect between the foramen primum and the ventricle.

In the case of double-outlet right ventricle there are two anatomic variations. The first type is the Fallot conal position, when the great arteries are in side-by-side position, or the pulmonary artery arises from a relatively normal infundibulum, which is ventrally related to the left ventricle. The second type is the subpulmonary defect, when the great arteries are in side-by-side position, or the pulmonary artery is slightly dorsal to the aorta. The defect opens into the right ventricle, under the aortic and pulmonary openings. The differentiating feature between these two types is the roof of the defect. In the Fallot conal position the conus septum is to the left and dorsal, whereas in the subpulmonary type it is to the right.

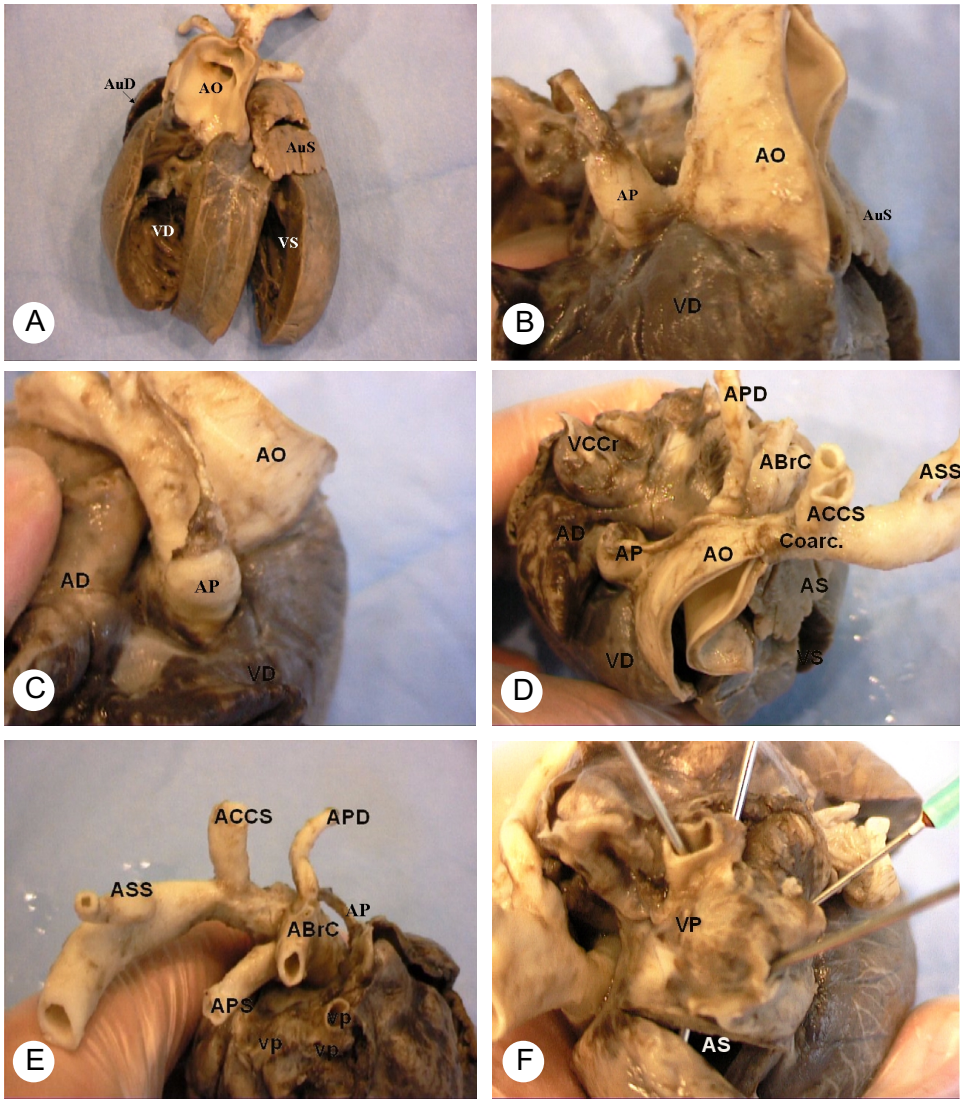

Fig. 1. Morphology of the heart base. **(A)** Heart – left view, **(B, C)** Origin of great vessels, **(D)** Base of the heart, **(E)** Aortic arch, **(F)** Dorsal view of the left atrium. ABrC: arteria brachiocephalica, ACCS: arteria carotis communis sinistra, AD: atrium dexter, AO: aorta, AP: arteria pulmonalis, APD: arteria pulmonalis dexter, APS: arteria pulmonalis sinister, AS: atrium sinister, ASS: arteria subclavia sinistra, AuD: auricula dextra, AuS: auricula sinistra, VCCr: vena cava cranialis, Coarc.: coarctatio aortae, VD: ventriculus dexter, VS: ventriculus sinister, VP: venae pulmonales

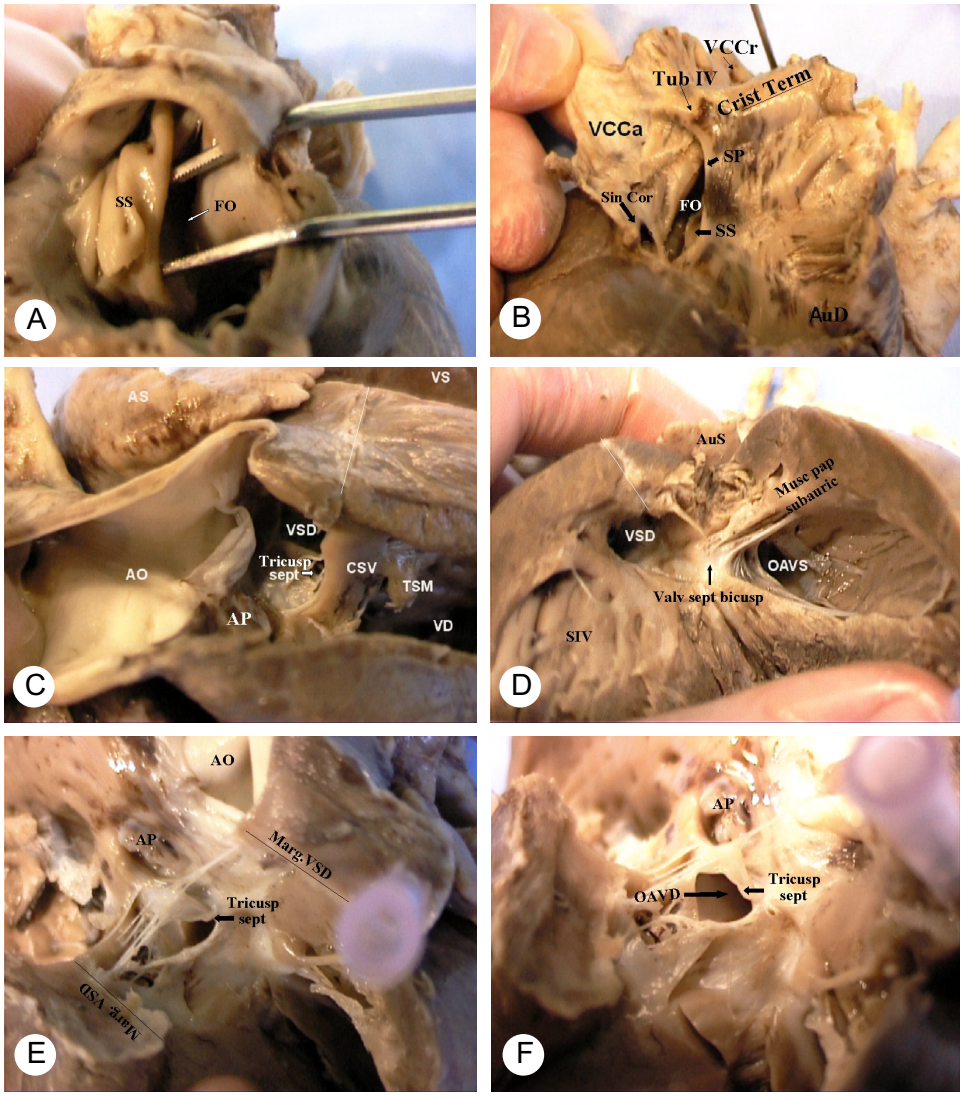

Fig. 2. Morphology of the opened heart. **(A)** Opened left atrium and its septum, **(B)** Opened right atrium, **(C)** Ventricular septal defect (VSD) from the right side, **(D)** VSD from the left side, **(E, F)** The transected VSD with the septal leaflet of the tricuspid valve. AO: aorta, AP: arteria pulmonalis, AS: atrium sinister, AuD: auricula dextra, AuS: auricula sinistra, Crist Term: crista terminalis, CVS: crista supraventricularis, FO: foramen ovale, Marg. VSD: margin of ventricular septal defect, Musc pap subauric: musculus papillaris subauricularis, OAVD: ostium atrioventriculare dextrum, OAVS: ostium atrioventriculare sinistrum, Sin Cor: sinus coronaries, SIV: septum interventriculare, SP: septum primum, SS: septum secundum, Tub IV: tuberculum intervenosum, Tricusp sept: valvula septalis tricuspidalis and its cleft, TSM: trabecula septomarginalis, Valv sept bicusp: valvula septalis bicuspidalis, VCCa: vena cava caudalis, VCCr: vena cava cranialis, VD: ventriculus dexter, VS: ventriculus sinister, VSD: ventricular septal defect

The transposition of great vessels has three types (Van Mierop and Wigleworth, 1963). The first type is the classical transposition: the pulmonary artery is to the left and there is a mitral-pulmonary fibrous continuity. If it is connected with a ventricular muscular septal defect, then it is located under the conus septum and the tricuspid valve. If the ventricular septum defect is absent, the pulmonary artery is situated dorsally. In the membranous type of ventricular septal defects, it is between the conus and dorsal septum and the central part of the annulus fibrosus. The second type is the corrected transposition: the morphologically right atrium communicates with the morphologically left ventricle, and it is separated by the septal leaflet of the mitral valve. The aorta is ventral to the pulmonary artery, because the inversion of the aorta is left-sided and the pulmonary artery is right-sided. The interventricular septal defect is located under the septal valve and dorsal to the conus. There is no border between the pulmonary valves and the mitral valve. The third type is the dorsal transposition: the greater part of the aortic opening is above the right ventricle, and the pulmonary artery is completely above the left ventricle. The aortic valve is dorsal to the pulmonary valve and is in fibrous continuity with the mitral valve through a small membranous type of defect.

In the intermediate cases, when the pulmonary artery is above the right ventricle and overrides the septum, it is described as double-outlet right ventricle. When the pulmonary artery is above the left ventricle and overrides the septum, it is described as transposition (Anderson et al., 1974).

The case presented in this paper is a special type of the double-outlet right ventricle, but it is not a real double outlet as it occurs in combination with pulmonary atresia. The aorta is situated dorsal to the pulmonary artery. The defect opens beneath the aorta, the membranous septum is absent, and the aortic-tricuspid continuity occurs. The septal leaflet of the tricuspid valve is cleft and attached to the free margin of the defect by the chordae tendineae. The trabecula septomarginalis and the ventral interventricular septum are normal.

In this case as many as five vessels originate from the aortic arch. At the beginning of the aortic arch the two pulmonary arteries arise. Because of the asymmetric development of the truncus arteriosus, the two pulmonary arteries developing from the 6th aortic arch have not fused with the main pulmonary trunk; instead of this they have fused with the aortic trunk, resulting in the absence of the ductus arteriosus. Normally (Moore and Persaud, 1998*b*) the 7th intersegmental arteries form the subclavian arteries, migrating and fusing with the aortic sac, into which are fused the common carotid arteries, the rudiments of the 3rd aortic arches, and the dorsal aortae, so they together form the common brachiocephalic trunk in ruminants. In this sheep heart the location of these vessels is similar to that seen in humans and rats. Because of the bulbar malformations the development of the aortic arch was also abnormal. Thus it is possible that the

4th aortic arch is underdeveloped, and causes the coarctation of the aorta at the level of the left common carotid artery.

In the sample studied there is an atrial septal defect of ostium secundum type: the opening is in the area of the fossa ovalis and includes both defects of the septum primum and secundum. During the formation of the septum secundum (Moore and Persaud, 1998*a*) the resorption of the septum primum is abnormal.

Acknowledgement

We are grateful to Tamás Tóth for his help with the dissection of the heart and making digital photos.

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