CVS Embryology - 2

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Circulation through Primordial Heart

Blood enters the sinus venosus Primordial atrium Dorsal aorta From: 1-The common cardinal veins 1st pharyngeal 2-The umbilical veins arch artery Sinuatrial valve 3-The vitelline veins Common cardinal ve Aortic sa **Blood** enters The Primordial Atrium Sinus venosus Atrioventricular (AV) Canal Truncus arteriosus The Primordial Ventricle. Atrioventricular canal Bulbus cord **The Bulbus Cordis** Dorsal and ventral Cardiac jell **Truncus Arteriosus** endocardial cushions into the aortic sac, from which it is distributed to the pharyngeal arch arteries Primordial ventricle the dorsa aortae for distribution to the embryo umbilical vesicle placenta

The <u>stage</u> is now set for the **septation of the heart**

lasts about 10 days

No major changes occur in the external appearance of the heart

The formation of the various cardiac septa occurs more or less **Simultaneously**



As the heart tube develops, it eventually pulls the AV canals and cushion from the **left to the medially** as seen in the illustration below.



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Fate of atrio-ventricular (A-V) canal

1- First it has a round opening then it becomes transverse.

2- Two thickenings (the atrio-ventricular or endocardinal cushions) appear on its dorsal and ventral walls.

3- They grow towards each other and fuse forming

THE SEPTUM INTERMEDIUM

Thus dividing the canal into right and left halves





Now we have Right and left atrioventricular canals



These canals partially separate the primordial atrium from the ventricle



Truncus AV Conus Proepicardial organ Ventricle Ventral endocardial cushion tissue Endocardial Endocardium cushion tissue Conotruncal endocardial Myocardium cushion В С Schoenwolf et al: Larsen's Human Embryology, 4th Edition.

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SEPTUM FORMATION IN THE ATRIOVENTRICULAR CANAL

- each atrioventricular orifice is surrounded by <u>local proliferations of mesenchymal</u> <u>tissue derived from the endocardinal cushions</u>.
- when the blood stream hollows the surface of these proliferations, the mesenchymal tissue becomes fibrous and forms *the valves* which remain attached to the ventricular wall by muscular cords which will degenerate and being replaced by dense connective tissue → chordae tendineae.

Note: Recent evidence shows that neural crest cells contribute to formation of semilunar cusps



It should be noted that the *endocardinal cushions developing in the atrioventricular region or conotruncal region are derived from* **neural crest cells migrating from the cranial neural folds to the outflow tract region.**



NEURAL CREST cells migrate along one of two pathways: a dorsal pathway through the (1 dermis, where they will enter the ectoderm to form Readoniv

melanocytes

In the skin and hair follicles

2) a ventral pathway through the anterior half of each somite to become sensory ganglia, sympathetic and enteric neurons, Schwann cells, and cells of the adrenal medulla



also form and migrate from cranial neural folds,

leaving the neural tube before closure in this region These cells contribute to the craniofacial skeleton as well as neurons for cranial ganglia

Neural Crest Derivatives

Readonin -Connective tissue and *bones of the face and skull*

2-Cranial nerve ganglia 3-C cells of the thyroid gland 4-Conotruncal septum in the heart

5-Odontoblasts

6-Dermis in face and neck

7-Spinal (dorsal root) ganglia 8-Sympathetic chain and preaortic ganglia 9-Parasympathetic ganglia of the gastrointestinal tract 10-Adrenal medulla 11-Schwann cells 12-Glial cells 13-Arachnoid and pia mater (leptomeninges) 14-Melanocytes

Formation of the interatrial septum





which is sickle-shaped or (crescent-shaped) septum appears and extends from the roof down to and fusing with the endocardinal cushions (septum intermedium)

As this curtain-like septum (the septum primum) develops, a large opening forms between its free edge and the endocardial cushions

This opening is called



> The foramen allows shunting of oxygenated blood from the right to the left atrium. The foramen becomes progressively smaller and disappears as the septum primum \geq fuses with the endocardial cushions

Before the foramen primum disappears, the upper part the septum primum breaks down (perforations, produced by **apoptosis (programmed cell death)**, to form the foramen secundum **(ostium secundum)**.



The foramen primum

Disappears as the septum primum fuses with the endocardial cushions

Septum secundum, grows from the ventrocranial wall of the atrium, immediately to the right of the septum primum



The opening between septum secundum and the septum primum Is called

(foramen ovale)

which persist throughout fetal life





The cranial part of the septum primum gradually disappears The remaining part of the septum primum, attached to the endocardial cushions, forms the valve of the oval foramen

- The lower edge of the septum secundum is thick and firm. In contrast, the edge of the septum primum that forms the lower boundary of the foramen secundum is thin and mobile like a flap.
- When blood tends to flow from the right to the left atrium, this thin flap moves away and there is no obstruction to blood flow.
- however, when there is a tendency for blood to flow from left to right this flap comes into apposition with the septum secundum and closes the opening.



After birth, when lung circulation begins and pressure in the left atrium increases, the valve of the oval foramen is pressed against the septum secundum, obliterating the oval foramen and separating the right and left atria.











Until the seventh week, there is a crescentshaped opening (IV foramen) between the free edge of the IV septum and the fused endocardial cushions. The IV foramen permits communication

between the right and left ventricles



 At the end of the seventh week, a downward extension occurs from the right margins of the a-v endocardial septum (septum intermedium) to close the interventricular foramen.



• This extension forms

The Membranous Part

of the interventricular septum





• The proximal bulbar septum

develops as two ridges which fuse together they share in closing the interventricular foramen.



Three distinct structures contribute to the formation of the postnatal ventricular septum: 1-The muscular ventricular septum 2-The proximal parts of the outflow cushions (spiral septum or the proximal bulbar septum) 3-The atrioventricular endocardial cushions.





Atrial Septal Defects Atrial septal defect (ASD)

 \succ is one of several congenital heart defects

It is more common in female births than in male

> Postnatally, ASDs result in *left-to-right shunting* and are. *non-cyanotic conditions*.

Two clinically important ASDs are the secundum and primum types

- Secundum-type ASD is the most common ASD
- It is caused by either an excessive resorption of the SP or an underdevelopment and reduced size of the SS or both.
- This ASD results in variable openings between the right and left atria in the central part of the atrial septum <u>above the</u> <u>limbus</u>.
- If the ASD is small, clinical symptoms may be delayed as late as age 30



Figure III-2-15. Secundum and Primum Atrial Septal Defect



Left-to-right shunts are <u>non-cyanotic</u> <u>conditions</u>

Ventricular septal defect (VSD)

It is the most common of the congenital heart defects
Being more common *in males than in females* The most common VSD is a membranous
ventricular septal defect, associated with the failure *of neural crest cells* to migrate into the endocardial cushions.

Ventricular septal defect (VSD)

- It results in <u>left-to-right shunting</u> of blood through the IV foramen.
- Patients with left-to-right shunting complain of excessive fatigue upon exertion.
 - Left-to-right shunting of blood is

<u>noncyanotic</u>

but causes increased blood flow and pressure to the lungs (pulmonary hypertension).

Read only

Pulmonary hypertension causes marked proliferation of the tunica intima and media of pulmonary muscular arteries and arterioles. Ultimately, the pulmonary resistance becomes higher than systemic resistance and causes right-to-left shunting of blood and late cyanosis. At this stage, the condition is called Eisenmenger complex

