Tuesday, February 6, 8:00AM-9:45AM
Plenary Session

Clinical History of Congenital Heart Defects (CHD): From Abstraction to Adulthood
Moderator: Angela Lin, Massachusetts General Hospital, Boston, MA

Embryology of the Heart and Associated Malformations
Thomas W. Sadler, Embryology and Birth Defects Prevention Consultant, Twin Bridges, MT

Heart development begins in the 3rd week after fertilization with the appearance and coalescence of blood islands in the embryonic mesoderm cranial to the neural folds. These cells form a horseshoe shaped endothelial heart tube that "moves" into the thoracic region as the embryo folds into the fetal position. Folding also brings the sides of the horseshoe together so that a single tube is formed. This tube represents the primitive heart and starts to beat at the beginning of the 4th week. At this time it bends upon itself in a process called "cardiac looping", while at the same time it forms primitive atrial, ventricular, and outflow tract regions. Looping creates the typical image of the heart with bulges for the atria, ventricles, and outflow tract (truncus arteriosus). Septation of the heart into its 4 chambers now begins and occurs from the 5th-8th weeks after fertilization. Partitioning of the atria involves formation of 2 septa that grow down from the atrial roof to overlap each other. In this way a valve is created with an opening called the foramen ovale. This valve functions during fetal life to allow blood returning to the right atrium from the umbilical vein to pass to the left atrium and ventricle to be distributed to the body. At birth, the valve is closed and this "shunt" is stopped so that the postnatal pattern of blood flow is established (blood now returns from the body to the right atrium then to the right ventricle to the lungs to the left atrium, left ventricle and then to the body via the aorta). At the same time the ventricular septum is formed by growth of muscle tissue between the ventricles and downgrowth of connective tissue from atrioventricular cushions surrounding the atrioventricular canal. This same type of cushion tissue also septates the outflow tract into the pulmonary trunk and aorta going to the lungs and body, respectively. Cells that produce this septum in the outflow tract are called neural crest cells and are also responsible for making the bones of the face. Thus, these cells, which are sensitive to teratogens (toxins), provide a link between cardiac and craniofacial defects and explain why many children with heart defects also have craniofacial abnormalities. Cardiac defects themselves include atrial septal defects, ventricular septal defects, and abnormalities of the outflow tract, such as transposition of the great vessels, persistent truncus arteriosus, tetralogy of Fallot, and others. Most of these malformations are due to abnormalities in the septation process.

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Overview of CHD, including anatomy
William Mahle, Sibley Heart Center, Atlanta, GA

Congenital heart disease is not a single disease but refers to many different structural defects in the heart that can be present in an infant at birth. Congenital simply means present at birth, not necessarily inherited. Heart defects are abnormal changes in the structure of the heart or major vessels in and around the heart. Other terms for congenital heart disease are cyanotic heart disease, heart defects, or congenital cardiovascular malformations.

Heart defects are the most common birth defect; 8 of every 1,000 babies are born with some sort of structural defect in their heart. Congenital heart defects are the leading cause of death from birth defects during the first year of life. However, over the past 50 years, dramatic advances have been made in the treatment and correction of these defects resulting in a 25 percent decrease in deaths from congenital heart disease from 1992 to 2002. Today, there are about 1 million adults with congenital heart disease in the United States.

The present study will review the most common congenital heart defects. In addition we will review how changes in physiology in the young child and the dynamic nature of the defects themselves pose a challenge for public health professionals who are involved in birth defects surveillance and patient care.

Risk Factors for CHD
Charlotte Hobbs, University of Arkansas for Medical Sciences College of Medicine, Little Rock, AR

The primary prevention of congenital heart defects is limited. Identifying both genetic and environmental risk factors will provide a knowledge base on which to build primary prevention programs. This presentation will review our current understanding of the complex etiology of heart defects and identify gaps in current knowledge. Recommendations for future etiologic studies will be described.