

ANESTHETIC MANAGEMENT OF A PARTURIENT WITH CONGENITAL HEART DISEASE

Essay

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INTRODUCTION

Pregnancy results in dramatic changes in the cardiovascular system.

Maternal heart disease complicates 0.2%-3% of pregnancies. The incidence of heart disease during pregnancy is steadily declining as a result of better medical and surgical care (*Krzysztof et al., 2007*).

The pregnant woman with heart disease represents a unique challenge to the obstetrician and the obstetric anesthesiologist. Dealing with high risk parturients requires a thorough understanding of the impact of pregnancy on the hemodynamic response to the patient's cardiac lesion (*David et al., 2004*).

And now congenital heart diseases became the most common source of cardiac problems seen in pregnant patients (*Kuczhowski et al., 2004*).

Patients are increasingly likely to survive to child bearing age by a percentage of 85% compared to 20% before the development of pediatric surgery as a form of palliative surgery or total correction of their defects and many of them are expected to have an

uneventful pregnancy and delivery (*Essop et al., 2005*).

Common congenital cardiac defects are patent ductus arteriosus (PDA), atrial septal defect (ASD) and ventricular septal defect (VSD) (*Macfrlane et al., 2007*).

Atrial septal defect is the most common cardiac congenital lesion in women of child bearing age. Pregnancy is usually well tolerated, and however risk of left ventricular failure (LVF) is increased during pregnancy due to increase in the cardiovascular volume during pregnancy as there is increase in the atrial volume which may result in biatrial enlargement, and supra-ventricular dysarrhythmias are likely to occur (*Kuczhowski et al., 2004*).

Patients who have had successful surgery during infancy and childhood, with complete repair are asymptomatic with relatively normal intra-cardiac pressures and blood flow pattern which is ordinary for the anesthesiologist or he may be involved in the management of patients with uncorrected lesions, which is challenging and complex (*Pradat et al., 2003*).

Atrial septal/ventricular septal defects in pregnancy are often well tolerated during pregnancy.

And we are going to review in this essay the ante-partum and the peri-partum problems associated with different lesions of congenital heart diseases in a parturient and also the current recommendations for their peri-anesthetic management.

AIM OF THE WORK

To review the current recommendation for anesthetic management of labor in women with congenital diseases.

Chapter 1

CLASSIFICATION AND PATHOPHYSIOLOGY OF CONGENITAL HEART DISEASES

Approximately 1 percent of all live-born infants have a heart defect. This means that in the United States, approximately 25,000 to 30,000 babies are born each year with some type of congenital heart disease (*Best et al., 2007*).

Cardiac development occurs very early in gestation, during the first seven weeks. The cause of most heart defects is unknown. Sometimes an infection during pregnancy, such as rubella (German Measles), can interfere with heart development. Other times a genetic or chromosomal abnormality such as Down syndrome will result in a heart defect. And still other times a chronic health issue with the mother such as insulin dependent diabetes seems to cause an increased incidence of heart defects in their fetus. However, for most families no cause is determined (*Wren et al., 2003*).

Prenatal diagnosis of congenital heart disease:

The prenatal (before birth) diagnosis of most congenital heart diseases (CHD) may be suspected during a routine ultrasound. If the obstetrician sees something on routine ultrasound that looks unusual, he or she may refer the patient to a specialist that handles high-risk pregnancies. These doctors are called perinatologists. More tests will be done to determine if there is a disorder of the heart and evaluate for any associated anomalies (birth defects) (*Uebing et al., 2006*).

The normal circulation of the heart

Blood returns to the heart, from the body, via two large veins. The upper body's blood returns via the superior vena cava and the lower body's blood returns via the inferior vena cava. Both of these vessels return blood to the right atrium. From the right atrium it passes through the tricuspid valve into the right ventricle. From the right ventricle the blood is pumped through the pulmonary valve into the pulmonary artery, and right and left arteries feed the blood into the right and left lungs (*Elkayam et al., 2005*).

In the lungs, the carbon dioxide is removed and oxygen is added to the blood. The lungs normally have a low pressure/low resistance so the blood flows easily throughout. Next, the blood is returned to the heart via the pulmonary veins into the left atrium. From the left atrium the blood passes through the mitral (or bicuspid) valve into the left ventricle. The left ventricle is the powerhouse or muscle of the heart. The left ventricle is very strong, thick and muscular to pump the blood out through the aortic valve into the aorta and ultimately to the rest of the body (*Hoffman et al., 2002*).

Normally there is no direct communication between the right and left side of the heart. The right side handles the unoxygenated blood. The left side handles oxygenated blood (*Hoffman et al., 2002*).

Fetal circulation

The normal circulation of a fetus while in the uterus follows a slightly different path than after a baby is born. While in the uterus the placenta acts as the lungs, therefore less blood passes into the actual fetal lungs. There are two structures within a fetal heart that allow this “bypass”. One is the patent

ductus arteriosus or PDA. The PDA allows mixing between the pulmonary artery and the aorta as it is a passageway between these two major vessels. The other is the patent foramen ovale (PFO). It allows mixing of blood between the two right and left atrium. The PDA and PFO allow a right to left shunt, which directs blood away from the lungs and directs this more oxygenated blood to travel to the body (*Hoffman et al., 2002*).

The pressure in the lungs of a fetus is higher than that in the body. This increased pressure encourages the right to left shunt also. After a baby is born the pressure in the lungs decreases as the vessels in the lungs begin to relax. The pressure in the body increases after birth. This change in the pressures allows more blood to flow into the lungs. These factors, the changes in pressure, are what cause the PDA and PFO to eventually close. The final closure usually takes several days (*Saenz et al., 1999*).

American heart association (AHA) classification of congenital heart disease. There are three common classifications of congenital heart disease (CHD) according to the AHA These are:

1. Septal defects.
2. Obstructive defects.
3. Cyanotic defects.

Septal defects

Normally there is no communication between the right and left side after birth. Sometimes a baby is born with an abnormal hole in the septum. This hole can be an atrial septal defect (ASD) or a ventricular septal defect (VSD) (*Jacobs et al., 2007*).

Most septal defects will not require any type of surgical repair. If a surgical repair is needed it is more commonly done later in life and not in the newborn period. Instead, septal defects will be treated medically. This includes continued observation for spontaneous closure and may include some medications. The medications most commonly used are digoxin and lasix. Digoxin helps the heart beat

stronger and lasix assists the body in getting rid of extra fluid (*Park et al., 2002*).

Atrial septal defect (ASD)

Accounts for approximately 5 to 10% of all CHD. Because it is difficult to differentiate a PFO from an ASD the exact incidence is difficult to establish (*Cool et al., 2008*).

With an ASD there is a hole in the septum or wall between the right and left atrium. ASD's vary in size. They all allow oxygen-rich blood from the left side of the heart to mix with blood headed to the lungs to become oxygenated. This makes for very inefficient function, and too much blood going to the right side and then being pumped into the lungs. This can cause some serious side effects if left untreated. The right side can become dilated or enlarged (*Deffebach et al., 1987*).

The larger the size of the ASD, the more the effects on the heart and lung function. Surgical repair requires heart-lung bypass. Postoperative hospital stay varies depending on the type of ASD (there are basically three types of ASD), but can range from four days to two weeks (*Webber et al., 2007*).

Ventricular septal defect (VSD)

Is the most common CHD and accounts for 20 to 25 percent of all CHD. These defects can vary greatly in size, but they all allow oxygen rich blood in the left ventricle to mix with blood depleted of oxygen in the right ventricle. Most of these will not require surgery in the newborn period and are simply monitored (*Wray et al., 2001*).

Some babies may not grow normally if the VSD is large and allows significant mixing. Small VSDs usually require no surgery because they will close on their own. Surgical repair requires heart-lung bypass. Postoperative hospital stay averages five to seven days (*Calcagni et al., 2004*).

Obstructive cardiac anomalies

In an obstructive disorder, the blood flow is restricted or completely blocked. This blockage or narrowing can occur in any of the four heart valves or above or below the valve. The blockage (atresia) or narrowing (stenosis) can occur in veins or in arteries (*Pradat et al., 2003*).

Aortic stenosis

Accounts for approximately 5% of all CHD. Depending on the severity of the stenosis, the symptoms at birth can vary from none noted, to decreased blood flow and decreased oxygenation to the body. As the PDA closes the symptoms usually become more acute. Sometimes, a percutaneous balloon valvuloplasty (opening of a valve) can effectively relieve the valve obstruction. The surgical repair is aimed at relieving the obstruction of blood flow through the aortic valve. The surgical repair requires heart-lung bypass. Postoperative hospital stay averages 7 to 10 days (*Wray et al., 2001*).

Pulmonary stenosis

Accounts for approximately 5 to 8% of all CHD. Because of the narrowing of the valve the right ventricle needs to work harder to get blood past the blockage. If the pressure in the right ventricle is high some form of treatment is indicated. For some, the valve can be stretched open with a percutaneous balloon valvuloplasty (*Calcagni et al., 2004*).

For some a surgical repair will be required. The surgical repair is aimed at relieving the obstruction of

blood flow through the pulmonary valve. Post-operative hospital stay averages five to seven days (*Park et al., 2002*).

Coarctation of the aorta

Accounts for approximately 8% of all CHD. Coarctation of the aorta is a narrowing of some portion of the aorta. This narrowing is usually found just past the arch of the aorta, opposite the area of the PDA (*Lane et al., 2002*).

Some infants will have no symptoms at birth, but can develop symptoms within the first week of life. Surgical intervention is required to open the narrowed area of the aorta to allow free blood flow to the body. This surgical repair requires heart-lung bypass. Postoperative hospital stay averages four to seven days (*Wray et al., 2001*).

Cyanotic defects

Cyanosis is a bluish discoloration of the skin due to less than normal amounts of oxygen in the blood. With these defects, cyanosis is the major symptom because the blood that is circulated is not oxygenated adequately (*Borrow et al., 1988*).

Many of these babies at birth will appear healthy because the circulation is still following the fetal circulation path. This circulation path provides adequate communication of oxygenated blood with unoxygenated blood to perfuse the body. Once these fetal structures begin to close, after a couple of days to a week, the infant becomes seriously ill and requires immediate interventions to keep oxygen saturation levels adequate to supply the body (*Cool et al., 2008*).

Tetralogy of Fallot

Accounts for approximately 10% of all CHD. Tetralogy is comprised of four components. The first is a narrowing of the pulmonary valve. Because of this narrowing less blood is pumped from the right ventricle into the lungs. The severity of narrowing ranges from child to child. The second component is a large VSD. This allows large amounts of unoxygenated blood from the right ventricle to pass into the left ventricle without going to the lungs. The body is supplied with blood that is depleted of oxygen (*Deffebach et al., 1987*).

Another component is the increased musculature of the right ventricle in comparison to the left because