

Congenital Heart Defects

We don't know entirely what cause congenital heart defects

- 90% Idiopathic Etiology
- Factors associated w/ increased incidence of CHD:
 - Fetal/maternal infection in 1st trimester has a big bearing on the unborn child. By 5th week heart is beating...a lot of women don't even know they're pregnant yet at that point.
 - Parent alcoholism (both parents contribute...alcohol makes male sperm abnormal)
 - Parent use of drugs w/ teratogenic effects
 - Cocaine does attack to the sperm!
 - Maternal smoking or exposure to second hand smoke during pregnancy
 - Maternal age over 40 yr
 - Statistics can be fudged to show a lot of different things
 - The reason for this one is b/c there are fewer births after this age
 - More cases of DS prior to 35 b/c more births at that time
 - If male is over 55 more cases of DS
 - Maternal dietary deficiencies
 - Maternal IDDM

Congenital Heart Defects

- 35 are recognized
- 9 are common (represents 90% of the CHDs)

Need to Understand...

- "Normal circulation"
- Fetal circulation
- Postnatal circulation...where the pressures are, where it's going, where is the oxygenated blood...where is the unoxygenated blood.
- Pressure differences in the heart

Fetal Circulation

Oxygenated blood from the placenta is carried to the fetus by the umbilical vein. Somewhere between 50 & 80% of this blood enters the ductus venosus and is carried to the inferior vena cava (the rest goes through the liver). From the IVC the blood goes to the right atrium of the heart, and most goes through the foramen ovale to the left atrium (bypassing pulmonary circulation), which means the left atrium now has oxygen-rich blood. The blood then goes into the left ventricle, and is then pumped into the aorta into the body.

The blood coming from the head and upper extremities enters the right atrium from the SVC and does not go through the foramen ovale, but instead enters the right ventricle and is pumped into the pulmonary artery. There is an opening between the pulmonary artery and the aorta called the ductus arteriosus, which directs most of this blood away from the lungs to bypass pulmonary circulation.

Pressures in the heart

- Before birth there is high pulmonary resistance b/c the fetal lung is collapsed.
- This high pulmonary pressure causes greater pressures in the right side of the heart and pulmonary arteries.
- The free-flowing placental circulation and the ductus arteriosus produce a low vascular resistance in the remainder of the fetal vascular system.
- When the umbilical cord is clamped and the lungs expand at birth, the hemodynamics of this system undergo some rapid and pronounced changes. The pulmonary pressure goes down, and systemic pressure goes up. This causes the higher pressure to now be on the left side of the heart (this makes sense because the left ventricle has to push very hard to get blood out to the whole system...it needs a lot of pressure to make that happen). When this pressure change happens, it closes the foramen ovale by shutting the little leaflet flaps shut. Note that this may not happen instantly and may take a few hours or even a few days.
- Recall that blood is going to flow from an area of high pressure to an area of low pressure...think of your garden hose. It has high pressure and it pushes the water out!

- Normally, the pressure on the right side of the heart is lower than the left; and resistance in the pulmonary system is less than in systemic circulation.

Differences between fetal circulation and normal neonatal circulation

- Ductus venosus: This is a blood shunt between the left umbilical vein and the inferior vena cava which empties into the right atrium. This blood coming in is baby's blood...it has come from the placenta and has nutrients. This eventually becomes the belly button!
- Foramen ovale: is between the two atria. In utero it shoots straight across...the gradient of pressure is right to left. It bypasses the lungs. There is high resistance in pulmonary area, so blood doesn't go to lungs.
- Patent ductus arteriosus is between the aorta and the pulmonary artery. The blood is going from the pulmonary artery across to the aorta b/c this is oxygen rich blood and going out to systemic circulation. There is low systemic resistance in fetus. The patent ductus arteriosus is sensitive to oxygen...so as soon as the baby takes his first breath, this opening closes (or should close).

Review: Postnatal

- Breath: Hypo- to hyper-oxygenation
- PO₂: Goes from the 40s to 70s or 80s immediately
- Ductus arteriosus – begins to constrict (can take up to 72 hours), may take longer to close in a premie. A baby with a PDA will have a murmur.
- Pulmonary vascular bed opens
- Pulmonary vascular resistance decreases (recall that it was high before!)
- Systemic vascular resistance increases (remember it was low before)
- Foramen ovale closes d/t blood flow left to right.

Assessment

- History
- Physical exam
 - Murmurs and cyanosis don't always happen immediately. A large murmur is going to be "soft".
- Determine if the issue is cardiac vs respiratory
 - Often can look the same; the systems are interdependent
 - How do you figure out the difference? Compare and contrast the...
 - Color: Are they blue and where are they blue? Cyanosis is a common feature of CHD, and pallor is a sign of poor perfusion.
 - Are they cold?
 - Are they acidotic?
 - What was the labor like? If it was long...they can become acidotic b/c the lack of oxygen causes hypoxia...if they come out acidotic, it's going to take them a while to pink up.
 - Is there a murmur?
 - Listen to lungs: If child was born via C/S you will hear fluid b/c not getting squeezed out when going through vaginal canal. We call these "gunky babies" bc they have a lot of fluid...this child will need more suctioning.
 - Pulses...on a newborn you're going to check pulses at brachial and femoral...want to see if pulses are same upper vs. lower...if not, then a cardiac defect is present. Be aware that chubby babies are hard to get pulses on (no duh!)
 - BP: take BP on all four extremities b/c you can pick up one of the defects this way.
 - What if baby cries: If a respiratory issue, the child should turn pink when crying b/c he is taking a big breath; a cardiac baby will turn bluer b/c the increased breath isn't making a difference. The problem is that they are having trouble moving the oxygenated blood around.
 - Give baby 100% oxygen even though they don't have a respiratory issue. This decreases the cardiac workload. If you put the oxygen on a respiratory kid, the O₂ sats will go up pretty quickly, but will stay the same if you put it on a cardiac kid. If you don't give adequate oxygen, the pulmonary beds constrict and this is most likely not a very good thing.
 - Blood Gases: on respiratory baby the O₂ will be low, CO₂ will be high; in cardiac kid the O₂ will be low b/c blood not coming around like it should, and CO₂ will be normal b/c they are able to blow it off.

Classification of Congenital Heart Disease (do not memorize).

- Acyanotic
 - Increased pulmonary blood flow
 - A/V septal defects
 - Patent ductus arteriosus
 - Atrio-ventricular canal
 - Obstructed blood from the ventricles
 - Coarctation of the aorta
 - Aortic stenosis
 - Pulmonic stenosis
- Cyanotic
 - Decreased pulmonary blood flow
 - Tetralogy of Fallot
 - Tricuspid atresia
 - Mixed blood flow
 - TGA
 - TAPVR
 - Truncus arteriosus
 - Hypoplastic left heart syndrome

About 99% of the babies with CHDs are full-term babies....can put oxygen on them b/c don't have to worry about retinopathy of prematurity after 34 weeks. (eye damage would ensue if baby had the retinopathy).

Acyanotic CHDs are in two categories

- Due to Increased pulmonary blood flow (something's open that should be closed)
- Obstructed blood flow from ventricles (something's closed that should be open)

SOMETHING IS OPEN THAT SHOULD BE CLOSED

Atrial Septal Defect

- Defect in the septum between the atria. It has an opening where it should be closed.
- In utero this is the foramen ovale...this could be the cause of a defect, but it could also be something else nearby.
- Pathophysiology: High pressure is LEFT...it's going to force oxygenated blood into right side of heart (where it mixes with oxygen poor blood) and back to the lungs...this leads to increased pulmonary blood flow and increased pulmonary pressures. What are you going to end up with? Pulmonary edema and CHF! (though the book says cardiac failure is unusual with an uncomplicated ASD and that pulmonary vascular changes usually occur only after several decades if defect is unrepaired.)
- Blood flow L to Rt
- Clinical manifestations = may be asymptomatic (then we just watch and wait); will have a murmur and be at risk for atrial dysrhythmias and pulmonary vascular obstructive disease and emboli formation later in life from the increased pulmonary blood flow.
- CHF (intervene before this happens)
- Treatment
 - Diuretics for CHF
 - Surgical repair: They now can put a patch in the heart via catheterization, so they don't have to crack open the chest.
- <http://www.cincinnatichildrens.org/health/heartencyclopedia/anomalies/graphicsumaries/asdtswf.htm>

Ventral Septal Defect

- This is an abnormal opening between right and left ventricles; can range from small to being so large that there is essentially just one ventricle. This one is frequently associated with other defects.
- Many are thought to close on their own, and this is more likely to happen in the first year of life if the opening is small to moderate in size.
- Greater pressure is LEFT...going to push oxygenated blood from left ventricle into right ventricle and back into pulmonary artery. (So, flow of blood is LEFT to RIGHT). This blood flows into the lungs, which can eventually lead to increased pulmonary vascular resistance and pulmonary congestion.

- The increased pressure in the right ventricle causes this muscle to hypertrophy...if the RV can't handle the extra load, the right atrium may also enlarge.
- This is the most common of all the defects.
- If asymptomatic, just wait. If after 2 years of age, they would probably go in and fix it at this point. A lot of times I guess it fixes itself by this time?
- If symptomatic, then repair...use a cloth (Gortex) patch instead of a wire patch like with ASD. Will also treat for symptoms of CHF.
- Manifestations = CHF is common; characteristic murmur. These patients are at risk for bacterial endocarditis and pulmonary vascular obstructive disease. In severe cases, Eisenmenger syndrome may develop.

Patent Ductus Arteriosus

- In utero, the ductus arteriosus was directing blood from pulmonary artery to aorta.
- This defect is a failure of the fetal ductus arteriosus to close within the first weeks of life. The continued patency of this vessel allows blood to flow from the higher pressure aorta to the lower pressure pulmonary artery, causing a left-to-right shunt.
- Patho: The additional blood in the pulmonary artery is recirculated to the lungs and returned to the left atrium and ventricle. The effect is increased workload on the left side of the heart, increased pulmonary vascular congestion and possibly resistance, and potentially increased right ventricular pressure and hypertrophy.
- High pressure area: is in AORTA. It's going to force oxygenated blood to pulmonary artery, back into the lungs again.
- PDA usually closes down when baby starts breathing.
- Will watch to see how they're doing...if asymptomatic just watch and wait b/c may close on its own
- May just hear a soft murmur at ICS, just below left clavicle.
- May feel bounding pulses...this is not normal
- May get a wider pulse pressure...how will you know? What is the normal PP in a newborn? Usually BP is around 60/40...so PP is 20. If it's bigger than 20, you're getting a widening pulse pressure. This will vary with children.
- Look to see if child is they gaining weight or not? If they are...is it actual weight or is it fluid?
- Listen to lungs, etc...
- Look for signs of CHF.
- Manifestations = May be asymptomatic or show signs of CHF; will be machinery-like murmur; widened pulse pressure and bounding pulses d/t runoff of blood from aorta to pulmonary artery.
- Dx = listen for that continuous murmur below left clavicle; x-ray; echo
- One of the things we do with a PDA is to give **indomethacin** if pre-term. This drug is a prostaglandin inhibitor...this will help constrict the PDA (recall that prostaglandin inhibitors are given to mom b/c they cause the uterus to relax. You do not give this to a mom in preterm labor b/c do not want to close the PDA while baby is still in utero...that would be very bad (mom may be able to get a little of this, but why risk it? There are other tocolytics to use). Also, pregnant mom should not take ibuprofen b/c it will help close the PDA... no bueno!
- SE of indomethacin: decreased renal blood flow (watch urine output, should be 1ml/kg...also monitor BUN/Cr)

Atrio-ventricular Septal Defect

- This defect is a result of incomplete fusion of endocardial cushions. It consists of a low atrial defect that coexists with a high ventricular septal defect, and it clefts the mitral and tricuspid valves. This results in a large central atrioventricular (AV) valve that allows blood to flow between all four chambers of the heart.
- The pacemakers are in this area...so surgical repair is going to be a bigger deal
- Pressure is on the LEFT...it is the oxygenated blood and it gets pushed into right ventricle and right atrium...so you've got a double whammy going up to the pulmonary system. Almost like a single chambered heart.
- Most likely to occur in Down Syndrome.
- Patho: Immediately after birth (when baby's newborn vascular resistance is still high) there is minimum shunting of blood through the defect. After this resistance falls, the left-to-right shunting occurs and pulmonary blood flow increases. The resultant pulmonary vascular engorgement predisposes the child to CHF.
- Manifestations = Moderate to severe CHF; Characteristic murmur; May be mild cyanosis with crying
- This needs to be surgically repaired

SOMETHING IS CLOSED THAT SHOULD BE OPEN

Coarctation of Aorta

- Blockage of aorta...something is closed that should be open
- There is high pressure on either side of the blockage...think of a garden hose that pushes the water out really strong at the area where you've covered the opening with your thumb, but it tapers out by the time it gets to the other side of the lawn. So, lower pulses will be low, upper pulses will be stronger. ditto for blood pressures.
- PDA is often open for a few days,, so baby will do fine....when the PDA closes, this defect becomes evident.
- What medication are we going to give the baby to keep the PDA open...(this will help us out to even out the pressure) Prostaglandins! (opposite of Indomethacin)
- Surgery...can remove segment and anastomose it together, can do balloons and stents (stents are not used very often...kind of an "old school" treatment)
- Do not watch and wait...will not repair itself. Have to get in there and fix it.
- Cardiac output will be decreased

Aortic Stenosis

- Somewhere in this valve area...something closed that should be open is closed and blocking blood flow.
- Left ventricle is going to be taking a lot of pressure as blood comes back down in here. As a result, the left ventricle hypertrophies. If the valve is severely obstructed, the left ventricle may not be able to compensate and it may fail to pump blood effectively, leading to heart failure.
- CO will be low...this leads you to the S&S you would expect to see
- This is the kid who drops dead on basketball court...this is b/c growth demands go up in adolescence...the demands for oxygen increase dramatically in surges.
- Marfan syndrome kids have this
- Can do balloon angioplasty to widen that up

Pulmonary Stenosis

- Obstruction of blood flow from the right ventricle to the pulmonary artery.
- Pressure is in right ventricle, will back up to right atrium and can potentially open the foramen ovale back up.
- This causes unoxygenated blood goes across and mixes with oxygenated blood.
- This child will have blue lip and nailbeds d/t lower oxygen levels in the blood.

Cyanotic CHDs are in two categories

- EITHER decreased pulmonary blood flow or...
- Mixed blood flow

Teratology of Fallot (decreased pulmonary blood flow)

- Four things going on
 - Pulmonic stenosis: causes backup of pressure in pulmonary artery
 - Ventricular Septal defect
 - Overriding aorta: the aortic valve is enlarged and appears to arise from both the left and right ventricles instead of the left ventricle. Depending on position of aorta, blood from both ventricles may be sent out to the body (oxygenated and unoxygenated)
 - Right ventricular hypertrophy
- Pressures: Varies, may be equal in L and R ventricles. Shunt direction depends on the difference between systemic and pulmonary resistance. If pulmonary resistance is higher, then shunt is from right to left. If systemic resistance is higher, then shunt is from left to right.
- Lot of unoxygenated blood is going out to the body
 - S&S = low O2 sats (not uncommon to be 85%), color is blue-ish
- Surgery definitely indicated...no watching and waiting
 - Probably a balloon to widen the stenosis
 - Will also take care of the other defects

- In countries where they don't have surgery...they are playing and running and suddenly squat which decreases venous return and can oxygenate their brain and organs better that way.
- If baby is crying...they are going to drop their sats. You need to go in there asap...put knees to chest to decrease venous return and perfuse vital organs.
- What if family is in there...and kid is crying...are you going to go rush in there? Most of them know how to do this, but you might make sure they know how to do it.
- A "Tet Spell" or a "blue spell" is decreased oxygenation. Some are pink and some are blue...not on test.

Tricuspid Atresia (decreased pulmonary blood flow)

- No opening at tricuspid valve
- Unoxegenated blood can't get from right atrium into right ventricle and on to the lungs.
- Right ventricle is underdeveloped.
- The blood in right atrium is pushed through foramen ovale...this is unoxygenated blood being forced into circulation with oxygenated blood.
- Will want to keep PDA open...to help send partially oxygenated blood back into lungs. Will give prostaglandins (Prostin). Recall that prostaglandins RELAX smooth muscle.
- Child will need surgery...can do a 3 step procedure...don't need to know detail on surgery, just that they are getting surgery definitely)

Transposition of Great Arteries (mixed blood flow)

- Pulmonary artery and aorta are transposed...the pulmonary artery comes off the left ventricle, and the aorta exits from the right ventricle. There is no communication between pulmonary and systemic circulation.
- Unoxygenated blood coming in from SVC goes to RA to RV to the aorta....oops!
- The oxygenated blood coming in from pulmonary to LA to LV and back to pulmonary artery
- This is not compatible with life!
- You want this child to have other defects...the foramen ovale will help, PDA and VSD...the defects will enable him to live.
- What you do...is hope for a ventral septal defect to force oxygenated blood into unoxygenated area.
- So....a child could be relatively OK while they have a ventral septal defect...this starts to close and the child will begin to show problems.
- Surgery...they flip-flop the arteries.
- Medical Treatment
 - This kid is going to get Prostin right away to keep the PDA open.
- Clinical manifestations (see slide)
 - Child may initially appear normal
 - Cyanosis can develop within a few hours of life...but can also be delayed if child has defects mentioned above.

Total Anomalous Pulmonary Venous Connection (mixed blood flow)

- SVC and Pulmonary Vein are connected
- The pulmonary veins are abnormally connected to the systemic venous circuit via the right atrium or various veins draining toward the right atrium such as the SVC.
- Truncus arterios didn't divide into pulmonary artery and aorta.
- Result is mixed blood being returned to right atrium and shunted from right to left through an atrial septal defect.

Hypoplastic Left Heart Syndrome (mixed blood flow)

- Tiny tiny left ventricle...can detect when child is in utero.
- Not compatible with life...noooooo bueno!
- Most of the blood from left atrium flows across the patent foramen ovale to the right atrium then right ventricle and out the pulmonary artery.
- These kids used to have to get heart transplants
- Now four options for treatments
 - Heart Transplant
 - 3-stage surgery (70-80% survival, but do not survive well)
 - Do nothing and let child die...most healthcare providers choose this option

- One more option exists b/c aortic stenosis is also going on with this (always or often? not sure). There is a pediatric cardiac surgeon in Boston who went in in utero and put a balloon to open the aortic valve...the left ventricle develops normally.

Congestive Heart Failure

- Clinical manifestations are on pg 907 of text (these are just some of them)
 - HR is going to be up (tacyC in a newborn is > 160...normal is 120-160)
 - RR is going to be high (tachyP in a newborn is > 60...normal is 30-60)
 - Weakness, restlessness
 - Weak peripheral pulses
 - Decreased blood pressure
 - Feeding will poor b/c don't have the energy
 - Babies do not perspire normally...but will get little sweat beads on forehead when eating.
 - Weight will increase b/c retaining fluid
 - Edema will be generalized and like a water balloon...boggy edema (not dependent or pitting edema as with adults)
 - Urine output will be decreased b/c holding water in 3rd spae
 - Can end up with hepatomegaly...2cm below ribcage is normal (one finger-breadth).
- Diagnosed based on clinical manifestations
- Treatment for CHF
 - Surgery to treat the heart defect
 - Diuretics: lasix (lose K), aldactone (saves K)
 - Positive inotropes: digoxin....don't take if pulse is < 100 for an infant; < 70 for a child
 - Infants rarely receive more than 1ml (50 mcg) in one dose; a higher dose is likely to be a med error!
 - don't teach parents to take pulse b/c med gets withheld too often
 - instead we teach parents to watch for S&S of dig toxicity (VOMITING!)
 - if child is doing a lot of vomiting, they need to bring child in
 - bring child to lab and check EKG (elevated or prolonged PR interval with low HR) and also dig levels
 - ACEi: captopril; used to decrease afterload
- Nutritional support
 - High cal formula (24-27 cal/oz) or fortify mom's breastmilk with extra calories.
 - If doc orders 3 oz, and baby only takes 1.5 oz...what do you do?
 - NG tube...so the baby can get nutrition without having to work.
 - Usually doc will order this if feed is > 20 mins

Nursing management of CHF

- Assessment
 - Physical assessment
 - Family: supports, caregiver role and interaction with child
- Nursing diagnoses
 - Decreased cardiac output
 - Fluid volume excess
 - Imbalanced nutrition: less than body requirements
- Outcome identification and planning
- Evaluation and family teaching

Psychosocial factors

- Parents feel that giving nutrition to child is a way of nurturing so they feel they can't offer anything to child
- Feel guilt and responsible for defect
- The heart is life and future...all these symbolic things together make for lots of psychosocial factors

CV DISEASE IN OLDER KIDS

Kawasaki Disease

- Incidence and etiology = 80% less than 5 years old (peak incidence in toddler age group); etiology is unknown; seen in every racial group
 - We know it's seasonal (late winter and early spring)
 - Not spread person to person
 - Is the body's response...an autoimmune response
- Pathophysiology
 - All blood vessels are inflamed (vasculitis)
 - Untreated cardiac sequelae: coronary artery aneurysms
 - Ecstasia on echocardiogram (vessels are dilated)
- Clinical manifestations (occurs in three phases)
 - Acute phase
 - Unexplained fever and nothing will bring it down...lasts 5 days or more.
 - Child then develops remaining diagnostic symptoms...
 - Erythema, scaling of skin, lips get very parched and scaly and can even bleed
 - Inflamed red mouth
 - Enlarged lymph node
 - Rash on trunk of body
 - Child is VERY irritable.
 - Subacute phase
 - Begins with resolution of the fever and lasts until all signs have disappeared.
 - Child is at greatest risk for development of coronary artery aneurysms during this phase.
 - Convalescent phase
 - All the clinical signs have resolved, but the lab values are not normal.
- Lab results / echocardiogram
- Diagnosis: Fever and 4 of the 5 criteria on pg 931
 - Fever for 5 days or more
 - Bilateral conjunctival inflammation without exudation
 - Changes in oral mucus membranes (erythema, dryness, fissures); oropharyngeal reddening or strawberry tongue
 - Changes in extremities (peripheral edema, erythema of palms and soles, periunguinal desquamation (peeling) of hands and feet.
 - Polymorphous rash
 - Cervical lymphadenopathy (one lymph node > 1.5 cm)
- Treatment
 - Mega doses of Aspirin...risk of Reyes syndrome is outweighed by risk of coronary aneurysms. 80-100 mg/kg/day in divided doses q 6 hours until fever controlled...then 3-5 mg/kg/day.
 - Gamma globulin (IV Ig) b/c autoimmune disease (Do not give immunizations to someone who has just had IV Ig...the immunization won't work) 2 g/kg over 10-12 hours
 - Child will not be going right back to school; cannot put child at risk to pick up viral infection b/c they are on aspirin and the risk for Reye syndrome is high
- Nursing management and family teaching
 - Ask family if they've recently shampooed the carpet (high incidence if recently shampooed carpet). Kids are crawling around on the floor...they get the chemicals in the carpet cleaner and end up with Kawasaki. Toddlers are in contact with carpet...babies you usually put a blanket down. Best to do it right before you go on vacation for at least a week.
 - Child needs to be followed for at least a year, will get repeat echocardiograms

Shock

- Big difference between adults and kids is the late drop in BP with kids
- You watch HR and RR instead
- Who is child we are most worried about?

- Child with infection. Need to watch for sepsis. Babies/children do not have the ability to localize infection...the infection goes systemic. What goes on with systemic infection? Sepsis causes vasodilation...and perfusion drops. Kidneys will show decreased urine output first, then the brain which causes ANS to not work and child doesn't breathe or have heart rate.
- Can also get shock as a result of blood loss d/t trauma
 - 3ml/kg/hr x 3 hours is considered traumatic blood loss for kids (especially babies)

REVIEW: Acquired CV Disorders

- Bacterial Endocarditis (differences in children vs. adults)
 - IV drug users can get it
 - Not washing hands can cause it if child had recent CV surgery...wash hands!
 - Treatment: IV abx for 4-6 weeks in hospital
 - Prevention is most important factor!
- Acute Rheumatic Fever
 - Not very common d/t use of abx

Dysrhythmias

- can occur when child has CV surgery
- Children vs. Adults (not sure where she was going with this)
- Three main ones to be aware of in peds
 - Tachycardia...kids can get this easily via crying, pain, dehydration, separation from parents, being annoyed, fever (for every degree over 99, you can add 10 beats for HR)...fast HR + fast RR leads to bradycardia
 - Bradycardia (As and Bs are NOT good in the NICU)
 - A = Apnea which leads to B = Bradycardia...if child has As and Bs and doesn't recover right away, they have to stay 7 more days. We give 'em little doses of caffeine to increase RR (babies are dependent on RESPIRATION...muy importante!) Bradycardia is usually a result of hypoxia in some way, so a big concern.
 - Asystole: You missed something!

Post-Op Family Teaching Topics

- Activities
- Diet
- Wound Care
- General considerations
- Medications

Cardiac Catheterization

- Review the general stuff
- The only difference with kids is the developmental approach
- Know the issues and what to watch for after (key assessments)

Misc...

- After fetal surgery...no vaginal delivery
- No or less long-term effects of spina bifida when fixed in utero and early.

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