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Getting It Right Every Time: Checklists as Quality Improvement Tools in the NICU

By, Dan L. Ellsbury MD; Robert Ursprung, MD, MMSc and Jonathan Nedrelow, MD

"Checklists have contributed to prevention of error under stressful conditions, maintenance of precision, focus, clarity, and memory recall. Although pilots are expected to use their professional judgment and critical thinking skills, they are also provided with tools to aid them in recalling the masses of catalogued information at the appropriate time. If pilots are not expected to recall from memory each crucial step of their complex tasks—why is this required of clinicians who are also responsible for the lives of others? Is the aviation industry willing to take these extra measures because their own lives are put at risk by their performance?"

~ Quoted from B. Hales and P. Pronovost. Journal of Critical Care. 2006;21(3):231.

The neonatal intensive care unit is a dangerous place. Multiple complex tasks are performed at a rapid pace on a diverse group of neonates. Extremes in gestational age and up to ten fold differences in birth weight create multiple opportunities for medical errors. Provision of care by a vast multidisciplinary team with a wide range of experience levels may generate multiple levels of miscommunication.

"Many NICUs currently use checklists, especially in the context of nursing practice. Pre-operative and post-operative checklists are common, as are checklists focusing on parent discharge planning and education. While these checklists may be of benefit, there are many other tasks that could benefit from use of checklists."

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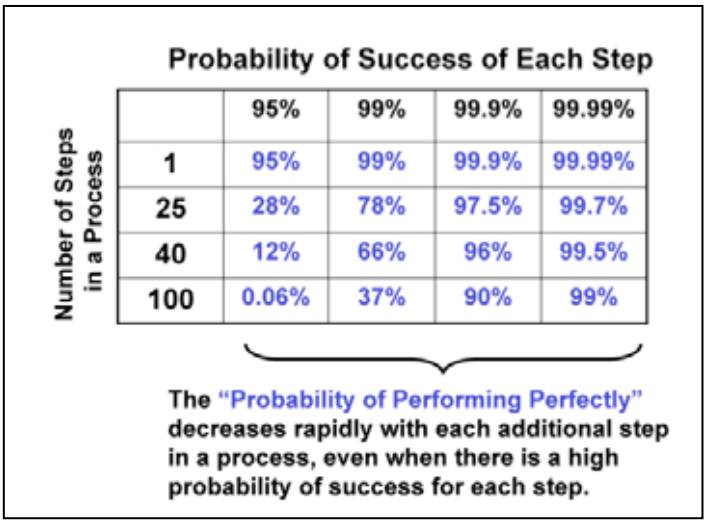

Figure 1. Daily tasks in the NICU are often comprised of multiple steps. Even when the probability of performing a specific step successfully is high, each additional step rapidly decreases the likelihood of performing the entire task perfectly.

Given this complexity, the "probability of performing perfectly" remains low (Figure 1).

Indeed, errors in neonatal intensive care are common. In a study of medication errors at two children's hospitals, Kaushal et al found the NICU had higher error rates than any other unit in the hospital (91 medication errors per 100 NICU admissions). Suresh et al, using an anonymous NICU error reporting system found errors in a wide range of domains, with 27% of the reported errors resulting in harm to the patient.

Clues from Aviation

What can be done to better manage the complexity of daily NICU care? Many examples of masterful engineering of complexity exist outside of medicine. "High reliability organizations" such as aviation and nuclear energy industries, expect failure and proactively design their systems to prevent catastrophe. Characteristics of these industries include preoccupation with the possibility of failure, training of their workforce to recognize and address errors, rehearsal of common scenarios of error, a systems based approach to repairs rather than blame or local repair.

The aviation industry is replete with examples of the perils and triumphs of managing complexity. The "preflight checklist" was developed after recognition of the limitations of the human mind to reliably recall, organize, and respond to a long list of complex procedures. Today, the use of checklists are a mandatory component of flight operations.

What is a Checklist?

Checklists are cognitive aids designed to guide users through complex tasks. A standardized list of steps in the task is generated. As the steps are completed and verified, each step is "checked-off". Ideally, if a required step is not completed, the task is halted until the missing step is performed.

Checklists in Healthcare

Despite the great success of checklists in error prevention in aviation and other industries, checklists are infrequently used in healthcare. However, recent success by Pronovost has generated new attention and excitement for the use of checklists.

Using an evidence based intervention that relied heavily on the use of checklists, Pronovost demonstrated a marked decrease in catheter-related blood stream infections in over 100 adult ICUs. A central line insertion checklist was developed that ensured that simple proven measures were taken, such as hand hygiene, appropriate cleansing of the insertion site, use of maximal barrier precaution, and other basic interventions. Most importantly, the nurse observing the procedure was empowered to halt the procedure if these checklist steps were not completed appropriately.

Results were rapid and dramatic. Catheter-related bloodstream infection rates dropped to zero within the first three months and then remained at a very low rate over the entire 18 month period.

Checklists have been used successfully in acute myocardial infarction patients and in stroke care. They are being used successfully in high intensity specialties, such as trauma care and anesthesiology. Is there a place for checklists in neonatal intensive care?

Checklists in the NICU

Many NICUs currently use checklists, especially in the context of nursing practice. Pre-operative and post-operative checklists are common, as are checklists focusing on parent discharge planning and education. While these checklists may be of benefit, there are many other tasks that could benefit from use of checklists.

Based on Pronovost's results, checklists for central line insertion are an obvious choice. Catheter-related bloodstream infections are a major source of morbidity and mortality in the NICU. Do we know that basic evidence based practices for central line insertions are being carried out every time, on every patient? Failure to use these practices may result in infection and possibly death. Checklists provide a simple means of system redesign to ensure that the appropriate practice occurs every time, with every patient.

Other examples include tasks that are not complex, but can adversely affect patient outcomes if not performed appropriately, such as pre-operative antibiotic administration and retinopathy exams. Rare events are also good topics for checklists, because the infrequency of the event may result in lack of familiarity with details of the task. Examples include delivery room management for diaphragmatic hernia, omphalocele, and myelomeningocele. The experience level of the operator may also drive checklist development. Trainees may benefit from checklists as guides to appropriate performance of new procedures as they are gaining skill and experience.

How to Design a Checklist

Checklists are very simple to construct. A list of important steps or components of a task is generated, with a space to record completion of each step. Some checklists may essentially be a "laundry list" of equipment, medication, or other items. Other will be sequential in nature, with a list of steps in a particular order, meant to be checked off before proceeding to the next step.

Central Line Insertion Checklist

As part of our efforts to reduce infection, please fill out this checklist.

Patient's Name_____ Gestational Age_____ Birthweight_____
Date_____ Bed Number_____ Inserter's Name_____
Physician – NNP - Resident- Fellow (circle)
Type of line:_____
Where was the line placed:_____
Reason for placing the line:_____

Did the person inserting the line wash their hands (yes/no), wear a hat (yes/no), wear a gown (yes/no), wear sterile gloves (yes/no), use a large sterile drape (yes/no).
Did the person observing the line insertion wash their hands (yes/no), wear a hat (yes/no), wear a gown (yes/no), wear sterile gloves (yes/no).
Describe the method used for cleaning the insertion site:_____
_____
Did the inserter maintain a sterile field at all times? If not describe why:_____
_____
_____
Describe the method used for dressing the insertion site:_____
_____
Was an x-ray done to confirm line placement? (yes/no)
Was the line readjusted for proper placement? (yes/no)
Was the consent placed in the patient's chart? (yes/no)

Figure 2. BAD CHECKLIST. Note that the overall content is reasonable, but includes extraneous information that belongs in a separate procedure note. The format is not consistent, nor is it easy to read. Many questions require written answers rather than check marks - requiring more time and creating legibility issues. No instruction on what to do with a completed checklist.

The most effective checklists are simple in design, clear in content and focus, and constructed to fit the specific needs of the individuals involved. They must be physically available at the right place and at the right time. Checklists should be piloted by a small group who will be using them and practical revisions made before disseminating the checklist for general use. See Figures 2 and 3 for examples of bad and good checklists.

Barriers to Use of Checklists

The past culture of physician training has likely created bias against use of memory aids like checklists. Memory aids may be viewed as an admission of weakness or lack of knowledge. Additionally, clinicians may feel their autonomy is being threatened and they will be forced into practicing "cookbook medicine." These issues can be dealt with by fully involving physicians in the development of the checklist and taking time to train everyone involved in the appropriate use of the checklist. Using the checklist to establish baseline compliance rates may highlight practice deficiencies and validate the need for a checklist. To avoid "checklist fatigue," the number of checklists used should be restricted and focused on high priority issues. Multiple long checklists for relatively trivial issues can rapidly result in noncompliance.

Conclusions

Checklists have a long record of success in aviation and other industries as tools to improve system safety and effectiveness. They have been used with success in the healthcare setting, but still remain underutilized. The NICU is a high risk environment in which multiple complex tasks are done on a daily basis. Checklists are ideally suited to this environment, and can make a major contribution to getting it right for every patient, every time.

Central Line Insertion Checklist

Place Patient ID Sticker Here

Check mark the type of line or lines placed:

____ **UAC** (Umbilical Arterial Line)
____ **UVC** (Umbilical Venous Line)
____ **PICC** (Peripherally Inserted Central Catheter)
____ **PAL** (Peripheral Arterial Line)

INSERTER:

Before the Procedure, did the inserter:

Yes No* Perform hand hygiene?
Yes No* Wear hat, mask, sterile gown and sterile gloves?
Yes No* Prep the insertion site per protocol?
Yes No* Cover entire patient with a sterile drape?

During the Procedure, did the inserter:

Yes No* Maintain a sterile field at all times?

After the Procedure, did the inserter:

Yes No* Apply a sterile dressing?

OBSERVER:

Did the observer:

Yes No* Perform hand hygiene?
Yes No* Wear hat, mask, sterile gown and gloves?

If "No", the observer must halt the procedure until corrective action is taken.
If the procedure is performed without taking corrective action, please explain the rationale in the space below.

Please Note:

1. The central line insertion checklists are to be stocked with the central line placement packs.
2. The central line insertion checklist should be placed in the "C-Line Checklist Box" at the central nursing station immediately after the procedure.
3. The checklist is not part of the patient's medical record.
4. The checklist is not a procedure note.

Figure 3. GOOD CHECKLIST. Simple format, with use of spacing and bold text to promote easy use. Organized by individual (inserter and operator), and by the sequence of events in the procedure. Free text entry is minimized. Clear instructions are given on response to variance from the checklist. Instructions given for stocking the checklist and what to do with the completed checklist.

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Perinatal Circulatory Physiology: Its Influence on Clinical Manifestations of Neonatal Heart Disease: Part I

By P. Syamasundar Rao, MD

INTRODUCTION

The fetal circulation is designed to utilize the placenta for gas exchange whereas the postnatal circulation uses lungs for gas exchange. Therefore, the circulatory systems must adapt to these changing requirements. An understanding of the fetal circulation and the changes that it undergoes at birth are essential for a better comprehension of the postnatal adaptation of the circulation in different types of congenital cardiac defects. The adult (Figure 1) and fetal (Figures 2-4) circulations are shown Figures 1 to 4. The objectives of this review are to:

1. present an outline of the fetal circulation;
2. discuss mechanisms that maintain fetal circulation,
3. describe postnatal changes; and
4. discuss the influence of postnatal circulatory changes on the presentation of certain important congenital cardiac defects.

The first three objectives will be addressed in this (Part I) section.

FETAL CIRCULATION

Some data from human fetuses are available, but most of the information with regard to the fetal circulation is derived from the experimental observations of the animal models, particularly the lamb [1-3]. The lamb model appears to best reflect human physiology [4]. Quantitative estimates of blood flow have been derived by use of Fick principle, electromagnetic flow transducers and by radionuclide-labeled microspheres. In the fetal circulatory states the cardiac output is expressed as the combined output of both ventricles in contradistinction to the postnatal circulatory states where the cardiac output is measured as the volume ejected by each ventricle.

Course of the Fetal Circulation

The oxygenated blood from the placenta is returned via the umbilical vein; the later enters the inferior vena cava via the ductus venosus (Figure 2). Nearly one-half of the umbilical venous blood goes through the liver and reaches the inferior vena cava via the hepatic veins. A substantial amount of the inferior vena caval blood is preferentially shunted into the left atrium (Figure 2). This appears to be related to the fact that the crista dividens, forming the upper margin of the foramen ovale (free margin of the septum secundum) overrides the inferior vena cava (Figure 5) [5]. The free edge of the lower margin of the foramen ovale, formed by the septum primum, is on the left side of atrial septum and the foramen ovale is kept open by the inferior vena caval stream (Figures 2 and 5). In addition, the inferior vena caval valve (Eustachian valve) diverts the inferior vena caval blood stream towards the atrial septum [6]. Consequently, the oxygenated blood enters the left ventricle and then the ascending aorta (Figure 3). Therefore, the brain (via the brachiocephalic vessels) and the heart (via the coronary arteries) are perfused with oxygenated blood (Figure 3). The pulmonary venous return does mix with the left atrial blood but, because of small amount of return (7% of combined ventricular output), it does not significantly desaturate the highly saturated umbilical venous return. The coronary venous and superior vena caval blood along with the portion of the inferior vena caval blood that did not stream into the left atrium, enter the right ventricle via the tricuspid valve (Figure 3). This desaturated blood is pumped by the right ventricle into the main pulmonary artery (Figure 4). From the main pulmonary artery, a small amount of right ventricular ejectate enters the lungs and the majority is pumped into the descending aorta via the ductus arteriosus. Thus, the

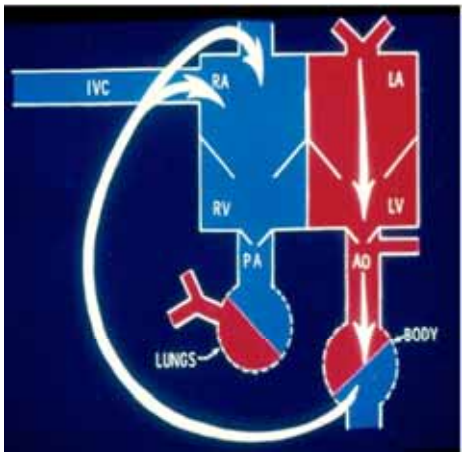

Figure 1. Diagrammatic depiction of adult circulation. The systemic venous return comes back to the right atrium (RA) via the inferior vena cava (IVC), superior vena cava (not labelled 0 and coronary sinus (not shown). The blood is pumped into the right ventricle (RV) and pulmonary artery (PA) and into the lungs for oxygenation. The pulmonary venous blood is returned into the left atrium (LA) and passed on to the left ventricle (LV) for pumping into the aorta (Ao) and the body.

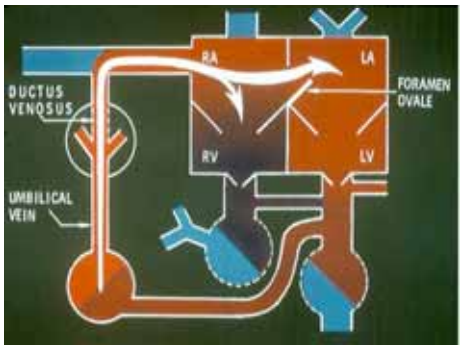

Figure 2. Diagrammatic depiction of fetal circulatory pathways. The placental venous return is carried by the umbilical vein, passes through the ductus venosus and reaches the inferior vena cava (not labeled) and right atrium (RA). From there a substantial proportion is shunted into the left atrium (LA) via the foramen ovale. The remaining portion goes into the right ventricle (RV). LV, left ventricle.

"Fetal circulation is intended to utilize placenta for gas exchange while postnatal circulation uses lungs for gas exchange."

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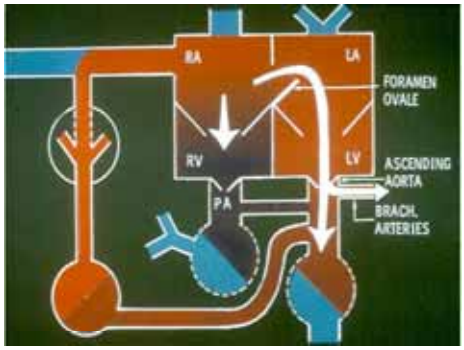

Figure 3. Diagrammatic depiction of fetal circulatory pathways. The blood reaching the left atrium (LA) via the foramen ovale is passed on to the left ventricle (LV) and from there into the aorta. The oxygenated blood reaches the coronary arteries (not shown) and central nervous system via the brachio-cephalic (BRACH.) vessels. PA, pulmonary artery; RA, right atrium, RV, right ventricle.

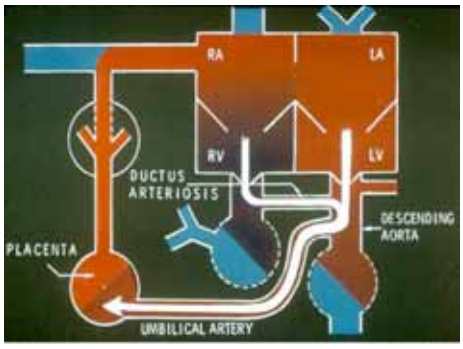

Figure 4. Diagrammatic depiction of fetal circulatory pathways. The right ventricular (RV) blood is pumped into the pulmonary artery (not labeled) and because of high resistance pulmonary circuit the majority of the blood is shunted via the ductus arteriosus into the descending aorta. From there the desaturated blood is returned to placenta via the umbilical arteries. LA, left atrium; LV, left ventricle; RA, right atrium.

desaturated blood makes its way into the placenta for oxygenation via the umbilical arteries (Figure 4).

Mechanisms Maintaining Fetal Circulatory Pathways

The **ductus venosus** and **umbilical vessels** are kept open by the mechanical effect of flow through them.

The **foramen ovale** is kept patent in the fetus because of the mechanical effect of streaming of the inferior vena caval blood into the left atrium (Figures 2 and 5) and the physical relationship of the inferior vena cava to the left atrium (Figures 5).

Because of the muscular nature of the **ductus arteriosus**, it may have to be kept open by active dilatation. Studies examining this issue suggest that both the locally produced and circulating prostaglandins (E_2 and possibly I_2) may be responsible for this. Prostaglandins are rapidly cleared by passage through the lungs. Since the pulmonary blood flow is very low in the fetus (7% of combined ventricular output) circulating prostaglandins are high in the fetus in contradistinction to postnatal life when the prostaglandins are rapidly cleared by passage through the lungs. In addition, the placenta produces large quantities of prostaglandins. The patency of the ductal may also be related to circulating adenosine [7].

Since the ductus arteriosus is large, the pressures in the main pulmonary artery and descending aorta are equal. Consequently, the quantity of blood flow going into the placenta vs. lungs depends upon their relative resistances. Since the placental circulation is a low resistance circuit, a larger proportion of the blood goes into the placenta. Because the **pulmonary circulation** is a high resistance circuit, a smaller proportion of the combined ventricular output makes it way into the lungs. The causes of this high pulmonary vascular resistance are not clearly understood. Kinking and high degree of tortuosity of the small pulmonary vessels have been suggested as causes, [8] but there is no general agreement of this causative relationship. Some studies [9, 10] dem-

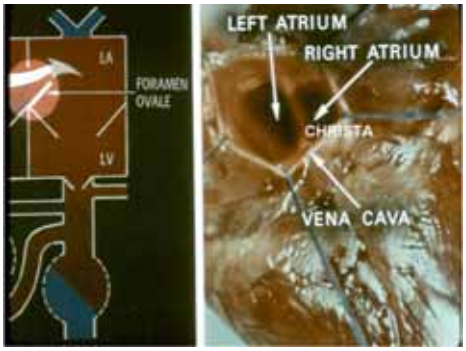

Figure 5. Diagrammatic depiction of fetal circulatory pathways (left) showing the passage of inferior vena caval blood into the left atrium (LA) via the foramen ovale. When examining the inferior portion of the heart from the inferior vena cava (right), note that a greater portion of the left atrium is seen, explaining in part the reason for shunting the inferior vena caval blood into the LA. CHRISTA, Christa dividens; LV, left ventricle.

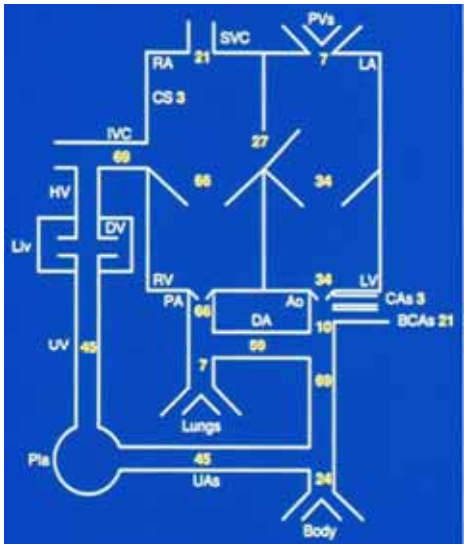

Figure 6. Diagrammatic depiction of fetal circulatory pathways illustrating the percent combined ventricular output for each cardiac/vascular chamber. The numbers indicate percent of combined ventricular output. Ao, aorta; BCAs, brachiocephalic arteries; CAs, coronary arteries; CS, Coronary sinus; DA ductus arteriosus; DV, ductus venosus; HV, hepatic vein; IVC, inferior vena cava; LA, left atrium; Liv, liver; LV, left ventricle; PA, pulmonary artery; Pla, placenta; PVs, pulmonary veins; RA, right atrium, RV, right ventricle; SVC, superior vena cava; UAs, umbilical arteries; UV, umbilical vein.

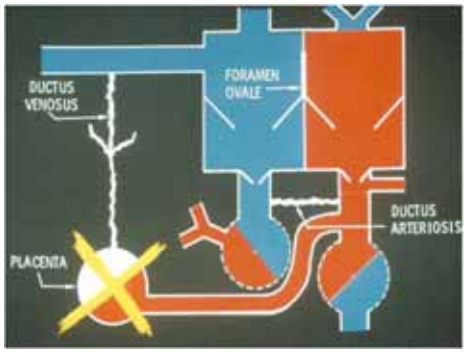

Figure 7. Diagrammatic depiction of changes in fetal circulation at birth. These include removal of placenta, constriction of ductus venosus, closure of foramen ovale and constriction of ductus arteriosus.

onstrated that the pulmonary arterioles have a thick smooth muscle layer which may be responsible for the high resistance. Low partial pressure of oxygen (to which the pulmonary arterioles are subjected to) keeps them thick and constricted. The pulmonary vascular resistance may also be influenced by changes in the pH and PCO_2 as well as autonomic nervous system [9, 10]. Many endogenous and exogenous vaso-active materials also stimulate the fetal pulmonary vasculature. Several studies demonstrate that pharmacologic doses of prostaglandins have dramatic effect on the fetal pulmonary circulation [9, 11]. Prostaglandin $F_2\alpha$ and leukotrienes (LTD_4) produce pulmonary vasoconstriction whereas prostaglandins E_1, E_2 and I_2 produce pulmonary vasodilatation [12]. The role of prostaglandins in maintaining a normally high pulmonary vascular resistance however, is not clearly defined. It is possible that prostaglandins mediate the hypoxic stimulus.

Distribution of the Cardiac Output and Oxygen Saturations in the Fetus

As mentioned in the preceding section, cardiac output is expressed as combined output (CVO) of both ventricles. The CVO in the lamb is 200 ml/kg/minute [4]. The estimates of relative distribution of the CVO based on the data derived from chronically instrumented lambs [13,14] are shown in Figure 6. Oxygen saturations and PO_2s are lower in the fetus than those in the neonates, infants and children [14]. This may be related to lower efficiency of the placenta to transport oxygen than the lungs. However, the fetus adapts to these lower levels by virtue of higher fetal hemoglobin levels; the fetal hemoglobin has low P50 of 18 to 19 torr which facilitates greater oxygen uptake from the placenta. Furthermore, the distribution of the blood to the various organs and the placenta is most advantageous in that the highly saturated blood goes to the heart and brain and low saturated blood to placenta.

MYOCARDIAL FUNCTION IN THE FETUS

The fetal myocardial structure differs significantly from that of the adult. In the adult the myocardial cells are compact with small nuclei and with little or no connective tissue surrounding them. In contradistinction, the fetal myocardial cells are less well organized, have large nuclei (sometimes even multinucleated), and the number of sarcomeres per unit is less. Also, the organization and function of the sarcoplasmic reticulum is incomplete which increases progressively with increasing fetal age. A similar pattern is seen with the development of t-tubule system [15]. The amount of areolar tissue in between the fetal myocardial cells is large [16]. It also appears that the sympathetic innervation of the heart is not completely developed in the fetus [14,16]. There are also differences in the type of substrates utilized, type of contractile protein activated, production and delivery of high energy phosphate, method of calcium delivery, and response of contractible elements to calcium ions between fetal, neonatal and adult myocardium [15].

The described structural and functional differences result in physiologic effects, namely, greater resting tension at a given muscle length and a lesser tension developed at any resting length in the fetus than in the adult [16]. While the initial studies suggested that the fetal cardiac output is mostly regulated by a change in the heart rate rather than by a change in the stroke volume, subsequent studies indicated that the Frank-Starling mechanism is indeed operative in the fetus, but within the narrow physiologic range [17]. Increase in the afterload adversely affects the fetal heart [18].

POSTNATAL CIRCULATORY CHANGES

The circulatory changes at birth are elimination of the placenta (Figure 7), maturation of the pulmonary circulation, and closure of fetal circulatory pathways (Figure 7). There is an impressive immediate change at birth followed by a slow change until an adult type of cardiovascular system is achieved; this may occur over varying time periods.

Elimination of the Placenta

At birth, the placenta is removed as a matter of normal birth process and the lungs

must assume the gas exchange function acutely. Elimination of placental circulation causes elevation of the systemic vascular resistance because of exclusion of low resistance placental circuit.

Development of Pulmonary Circulation

Soon after the delivery respiration begins and within a few minutes after birth almost complete expansion of the lungs occurs. There is striking decrease in the pulmonary vascular resistance and a distinct increase in the pulmonary blood flow at birth. This is associated with a fall in pulmonary arterial pressures. Expression of the fluid from the alveoli and expansion of the lungs are responsible to a great degree for the fall in the pulmonary vascular resistance [9, 19, 20]. Other factors that may affect a decrease in the pulmonary resistance are decreased PCO_2, increase in pH and increase in alveolar and blood PO_2. The consensus of opinion is that an increase in PO_2 in the alveoli and blood is the most potent and important pulmonary vasodilator at birth. The alveolar gaseous oxygen diffuses in sufficient quantities into the region of precapillary vessels making them to dilate. The mechanism by which the oxygen induces pulmonary arteriolar dilation is not understood. It may directly affect the pulmonary arteriolar smooth muscle cells or its action is mediated through a chemical substance. The oxygen may activate kininogen to bradykinin. Bradykinin is a potent pulmonary vasodilator. The effect of bradykinin itself may be mediated through prostocyclin. A rapid increase in bradykinin levels in the left atrium after ventilation with oxygen supports bradykinin mediated action of oxygen. Since the increased bradykinin levels last for a short period of time one may question the validity of bradykinin mediation as the sole factor responsible for pulmonary vascodilation. Pulmonary vasodilatation induced by oxygen occurs in two phases. The initial rapid phase cannot be inhibited by indomethacin and therefore is not dependent upon prostocyclin-mediation. The subsequent slow phase of the oxygen-medicated pulmonary vasodilatation may be influenced by prostaglandins [21]. Decrease in pulmonary vascular resistance increases the pulmonary blood flow markedly.

Further decrease in the pulmonary vascular resistance and involution of the pulmonary arteriolar medial musculature take place more gradually. The pulmonary vasculature looks very similar to that of the adult by the age of 6 to 8 weeks. Pulmonary parenchymal disease states causing alveolar hypoxia and reduced inspired oxygen such as in high altitude may prevent normal maturation/involution of pulmonary vasculature. Elevated pulmonary artery pressure associated with congenital heart defects (for example large ventricular septal defect or patent ductus arteriosus) may also retard the normal involution of the pulmonary arterioles.

Closure of the Fetal Circulatory Pathways

Patent foramen ovale. As alluded to in the preceding section, decrease in the pulmonary arteriolar resistance is associated with an increase in the pulmonary flow which in turn increases the volume of blood flow return to the left atrium with consequent

increase in the left atrial pressure. Shortly before this, the placenta is eliminated with consequent decrease in the umbilical venous and inferior vena caval flow. This will result in a slight decrease in the right atrial pressure. A combination of increase in the left atrial pressure and decrease in the right atrial pressure will result in apposition of the septum primum and septum secundum causing in functional closure of foramen ovale. This occurs within the first few hours after birth. The functional closure of the foramen ovale is often incomplete, especially if there is an increase in the right atrial pressure (e.g., crying, pulmonary vasoconstriction, severe right atrial or ventricular obstruction), and/or a decrease in the left atrial pressure, with resultant right-to-left interatrial shunting. Severe dilatation of the left atrium secondary to increased pulmonary blood flow (for example patent ductus arteriosus or ventricular septal defect) may stretch the patent foramen ovale, causing left-to-right shunting.

While functional closure of the foramen ovale occurs within hours after birth, anatomic closure may take 2 to 3 months [22,23]. In some subjects, the closure does not take place at all. Several studies have shown persistent patency in nearly 20% of older infants, children, adolescents, and adults [24].

Ductus venosus. The ductus venosus also closes shortly after birth. The closure may simply be due to lack of blood flow through this structure following elimination of placenta. Or, the mechanism of closure may be similar to that of ductus arteriosus.

Ductus arteriosus. Two stages of ductus arteriosus closure following birth have been described, the first, functional closure by constriction of ductal muscle occurs within 10 to 15 hours of age [3,19,22,25]. The second, anatomic closure occurs by endothelial destruction, subintimal layer proliferation, connective tissue formation over the next two to three weeks. Increase in oxygen tension produces muscular constriction of ductal muscle, causing the ductus to close [26,27]. In contradistinction, situations with low oxygen such as high altitudes or when the neonate is exposed to low oxygen concentrations, the ductal closure is delayed [28,29]. Vasoactive substances such as histamine, 5-hydroxytryptamine, acetylcholine, bradykinin and catecholamine may have a role in ductal closure although their role has not been fully delineated.

The mechanism of action of O_2 in ductal closure is not clearly elucidated, but most authorities suggest that direct stimulation of the smooth muscle cells of the ductal tissue is responsible for ductal closure. The ductal constriction may also be mediated through cyctocrome and thromboxane systems. Several studies suggest a definitive role of prostaglandins in either initiating ductal constriction or mediating ductal constrictive effect of oxygen [30]. Relaxation property of ductal muscle with prostaglandins seems to develop early in fetal life. The prostaglandin mediated ductal relaxing mechanism is most active at about 0.7 gestation. With increasing gestational age the ductal muscle becomes less responsive to prostaglandins while it acquires increasing sensitivity to oxygen. It would appear that prostaglandins indirectly contribute to ductal closure by becoming less effective after birth and potentiate constrictive action of oxygen.

SUMMARY AND CONCLUSIONS

Fetal circulation is intended to utilize placenta for gas exchange while postnatal circulation uses lungs for gas exchange. Fetal circulatory pathways, namely, umbilical vessels, ductus venosus, foramen ovale and ductus arteriosus, high pulmonary vascular resistance and low placenta resistance facilitate placental gas exchange and promote distribution of oxygenated blood to the vital organs of the fetus. Mechanical factors, prostaglandins and low PO_2 in the lung keep the fetal circulatory pathways open. Postnatal circulatory changes are elimination of the placenta, development of pulmonary circulation, and closure of fetal circulatory pathways. The influence of postnatal circulatory changes on the clinical presentation and clinical course of the neonate with congenital heart defects will be dealt with in Part II of this review to be published in the next issue of Neonatology Today.

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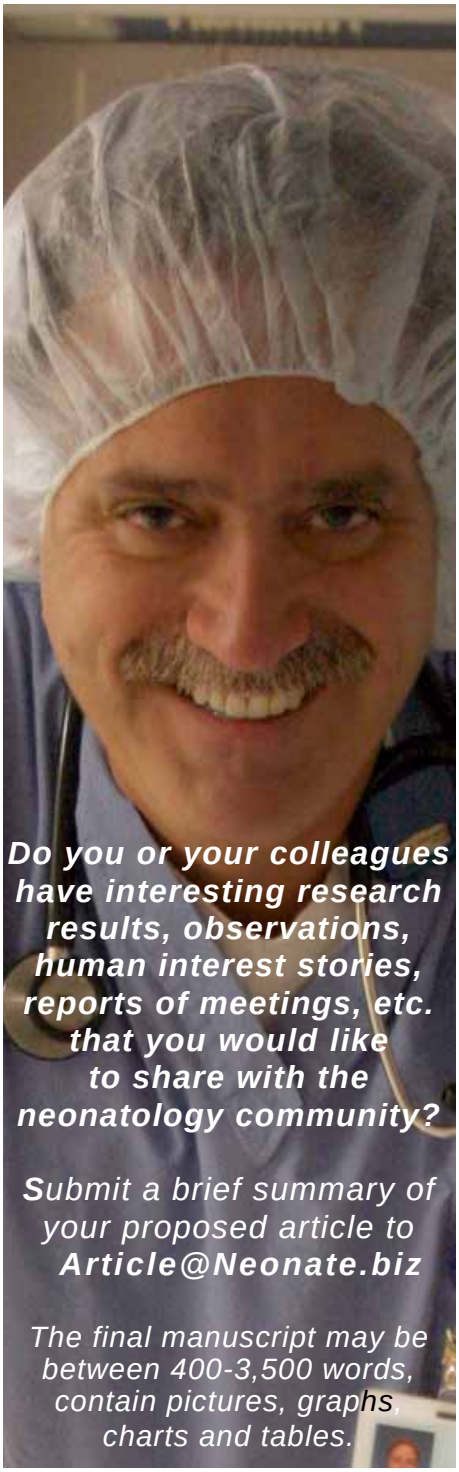

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Medical News, Products and Information

LA BioMed Researchers Find Few Emergency Rooms Fully Equipped for Pediatric Patients

In the first survey to specifically measure hospital pediatric preparedness, a team of Los Angeles Biomedical Research Institute (LA BioMed) researchers found few U.S. emergency rooms are properly equipped for children.

The survey by Drs. Marianne Gausche-Hill, Charles Schmitz and Roger J. Lewis was reported in the December issue of Pediatrics, the peer-reviewed journal of the American Academy of Pediatrics. The team of LA BioMed researchers found only 6% of the 1,489 emergency rooms that responded to the survey had all the medicine and equipment the American Academy of Pediatrics (AAP) and the American College of Emergency Physicians (ACEP) recommend.

For instance, half of those responding reported that they were missing the laryngeal mask airways used for ventilating children.

Seventeen percent of the hospitals that responded to the survey did not have Magill forceps for removing foreign bodies from a child's airway, said Dr. Gausche-Hill. This equipment may be life-saving, so this study highlights important issues for patient safety.

The study found 89% of pediatric (ages: 0-14 years) emergency room visits occur in non-children's hospitals. About a fourth of these visits take place in rural or remote facilities. Only 6% occur in a separate pediatric emergency department.

More than half the emergency departments reported they had a quality improvement or performance improvement plan for pediatric patients, and 59% said they were aware of the American Academy of Pediatrics/American College of Emergency Physicians guidelines.

Hospitals that were more prepared tended to be urban, to have higher volumes, to have a separate care area for pediatric patients, to have physician and nursing coordinators for pediatrics, to be aware of the AAP/ACEP guidelines, and to be interested in guideline implementation, the researchers concluded. The study also demonstrates that much work is left to be done to improve pediatric preparedness of EDs (Emergency Departments). Additional work should explore the relationship of preparedness to quality of care delivered, delineate barriers to guideline implementation, and identify best practices that can be coordinated within emergency care systems to improve the preparedness of EDs to care for children.

Pediatrics is the official, peer-reviewed journal of the American Academy of Pediatrics. It may be viewed at http://pediatrics.aappublications.org/.

About LA BioMed

Founded more than 55 years ago, LA BioMed conducts biomedical research, trains young scientists and provides community services, including childhood immunization and nutrition assistance programs. The institutes researchers conduct studies in such areas as cardiovascular disease, emerging infections, cancer, diabetes, kidney disease, dermatology, reproductive health, vaccine development, respiratory disorders, inherited illnesses and neonatology.

LA BioMed researchers have invented the modern cholesterol test, the thyroid deficiency test and a test to determine the carriers of Tay-Sachs disease, an inherited fatal disorder. One of the institute's researchers also developed the paramedic model for emergency care, setting a precedent that transformed emergency medical services and became the basis for training paramedics across the country.

LA BioMed is an independent research institute that is academically affiliated with the David Geffen School of Medicine at the University of California, Los Angeles. The institute is located on the campus of Harbor-UCLA Medical Center near Torrance. For more information, www.LABioMed.org

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