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## SSRI's IN PREGNANCY – Serious Concern or ‘Much Ado about Nothing’?\*

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### Introduction

The use of medications during pregnancy is a balancing act. The intended patient is the mother in most cases. Her disease, if left untreated, may have adverse effects on the outcome of the pregnancy. There is also an innocent bystander of drug therapy during pregnancy, the unborn child, who will be exposed to the effects of most drugs taken by the mother. Some drugs, like heparin or insulin, are too large to pass through the thin membrane (just a few cells thick) that separates maternal from fetal blood *in utero*. However, the vast majority of drugs will easily cross the placenta from mother to the unborn child.

Prevalence rates of clinically diagnosed depression during pregnancy have been reported to be as high as 16%.<sup>(1)</sup> The use of the SSRI (serotonin substance reuptake inhibitor) class of antidepressants has skyrocketed since the introduction of fluoxetine (Prozac ©) in the early 1990s. The exposure rate of SSRIs increased from 12.2 per 1000 pregnancies in 1999/2000 to 28.5 in 2003/2004.<sup>(2)</sup>

In general SSRIs have the same efficacy as the older tricyclic antidepressants, but have a safer side effect profile. The SSRIs act by inhibiting the re-uptake of serotonin at the

junction between the serotonin producing fibers in the CNS and the post synaptic fibers, where serotonin receptors are located.

The amount of literature that has been published on SSRIs and pregnancy is likely greater than for any other class of drugs.

### Early teratogenicity studies

During the 1990s at least 3 prospective cohort studies and the manufacturer's fluoxetine registry concluded that the SSRIs are not teratogenic.<sup>(3,4,5)</sup> This was further supported by the results of a meta analysis of prospective comparative studies of the SSRIs (specifically fluoxetine, paroxetine, sertraline and venlafaxine) during the 1990s and up to 2004.<sup>(6)</sup> A prospective Swedish Birth Registry of all births, including 546 women who had used an SSRI during pregnancy, demonstrated that only when maternal age was greater than 35 years or parity was 3 or more was the odds ratio (OR) of a congenital malformation associated with *in utero* SSRI exposure greater than 1.<sup>(7)</sup>

### What's Inside . . .

SSRIs in Pregnancy – Serious Concern or ‘Much Ado About Nothing’	1
Immunization & The Role of Intravenous Immunoglobulin For the Management of Hemolytic Anemia of The Newborn	6
For Your Information:	10
You Asked Us:	13
Upcoming Events:	15

### Acute effects on the newborn

Even though these large initial studies suggested that the SSRIs were not teratogenic, other effects of *in utero* SSRI exposure on the newborn became evident. These included higher rates of prematurity, admission to special care nurseries and 'poor neonatal adaptation' (respiratory difficulty, cyanosis on feeding, jitteriness).<sup>(3)</sup> Numerous case reports during the 1990s also reported symptoms in the newborn that included increased motor activity, an exaggerated Moro reflex, tremulousness, excessive crying, disrupted sleep organization, respiratory difficulty and feeding intolerance.<sup>(9,10,11)</sup> It is generally believed that these symptoms represent withdrawal from the SSRI, although there has been a suggestion that drug toxicity may also play a role.<sup>(12)</sup>

In August 2004 Health Canada issued an advisory of potential adverse effects (breathing difficulties, seizures, muscle rigidity, jitteriness and constant crying) that might be seen in newborns whose mothers took an SSRI during pregnancy.<sup>(13)</sup> This Health Canada advisory included the following drugs: Bupropion (Zyban®), Wellbutrin®), Citalopram (Celexa®), Fluoxetine (Prozac®), Mirtazapine (Remeron®), Paroxetine (Paxil®), Sertraline (Zoloft®), Venlafaxine (Effexor®).

### Cardiovascular effects on the newborn

Health Canada issued another warning, in October 2005, based on a letter to Health Professionals, from GlaxoSmithKline (GSK) which suggested that paroxetine, when taken during pregnancy, was associated with an increased risk of cardiac malformations in the newborn.<sup>(14)</sup> GSK is the manufacturer of Paxil © (paroxetine). This warning was based on the GSK Pregnancy Registry, the National Birth Defects Prevention Study, the Swedish Medical Birth Registry and a Danish Cohort Study. The data, from a registry, a surveillance system and prescription databases suggested that there was an increased prevalence (about double) of cardiovascular malformations (~2% vs 1%) with paroxetine compared to other antidepressants. The CV defect was mainly a septal defect, the natural history of which is to often close on its own.

In December 2005 the US Food and Drug

Administration (FDA) issued a warning that exposure to paroxetine in the first trimester of pregnancy may increase the risk for congenital malformations, particularly cardiac malformations.<sup>(15)</sup> At the FDA's request, the manufacturer changed paroxetine's pregnancy category from C to D (positive evidence of human risk).

Three recent large scale studies have provided further insight into the use of SSRIs during pregnancy and birth defects.

An update of the National Birth Defects Prevention Study provided the basis for a population based, case control study with over 9,600 newborns with birth defects and over 4,000 controls.<sup>(16)</sup> The investigators examined the effect of SSRI exposure 1 month before or 3 months after conception. There was no increased rate of birth defects (including CDV defects) as a result of SSRI exposure (fluoxetine, sertraline, paroxetine and citalopram, grouped together or analyzed individually) with the exception of 3 rare defects (anencephaly, craniosynostosis, and omphalocele) which had adjusted odds ratios that ranged from to 2.4 – 2.8.

The second study, the Slone Epidemiology Center Birth Defects Study, originated from a database that included over 9,800 newborns with all types of birth defects and over 5,800 control subjects.<sup>(17)</sup> Results were similar to the National Birth Defects Prevention Study: no increased rate of birth defects (including CDV defects) due to SSRI exposure (collectively or individually, same SSRIs). These authors did not find an increased incidence of anencephaly, craniosynostosis, or omphalocele.

The third, and most recent study, by Einarson *et al*, represented 1,174 unpublished cases of first trimester paroxetine exposure gathered from Teratology Information Services plus 2,061 cases from 5 previously published database studies.<sup>(18)</sup> When all of the information was combined the mean rate of CDV defects was 1.2% (95% CI, 1.1 – 2.1). This incidence is the same as the population incidence of approximately 1%.

These 3 recent, large scale studies are in disagreement with earlier findings and suggest that there is not an increased

incidence of CDV defects with the SSRIs, including paroxetine.

### **Persistent pulmonary hypertension**

An association between SSRI exposure during pregnancy and the development of persistent pulmonary hypertension of the newborn (PPHN) was reported in 2006.<sup>(19)</sup> This was a case control study nested within the Birth Defects Study of the Slone Epidemiology Centre. The adjusted OR of developing PPHN when SSRI exposure occurred after week 20 of gestation was 6.1 (95% CI, 2.2 – 16.8).

A prospective, population based study from the Swedish Medical Birth Register has very recently supported this association between SSRI exposure during pregnancy and the development of PPHN.<sup>(20)</sup> When putative confounding factors (maternal BMI, high maternal age, first parity, smoking) were controlled for, the OR of developing PPHN for newborns greater than 34 weeks gestation was 2.4 (95% CI, 1.2 – 4.3) when an SSRI was taken during early pregnancy. When an SSRI was taken through pregnancy the OR was 3.6 (95% CI, 1. 2-8.3).

The baseline incidence of PPHN in all newborns is 1 to 2 cases per 1,000 births. Prematurity itself is a risk factor for PPHN, so that the incidence of PPHN increases with decreasing gestational age. Thus, the baseline incidence for more mature babies decreases. In the Swedish Medical Birth Study the incidence of PPHN in babies greater than 34 weeks was 0.5 cases per 1,000 births (0.05%). If the relationship between SSRI exposure during pregnancy and the development of PPHN is causal, then the OR of developing PPHN for babies greater than 34 weeks would be expected to increase to perhaps 2 cases per 1,000 births, still a rare condition. The underlying mechanism for this proposed association is remains unknown.

### **Summary of Teratogenic Potential of SSRIs During Pregnancy**

At this point in time the evidence suggests that SSRI exposure during pregnancy is not associated with an increased rate of congenital malformations or CDV defects. It also does not appear that a specific SSRI, paroxetine, is associated with CDV defects.

### **Neurodevelopment**

Language development and global IQ scores at 16 and 86 months of age were examined in children who had been exposed to tricyclic antidepressants (n=80), fluoxetine (n=55) or a known non-teratogen (n=84).<sup>(21)</sup> There were no differences in IQ scores or in temperament, mood, arousability, or behaviour problems amongst the groups.

In 2007 Oberlander *et al* conducted a prospective 4 year follow up longitudinal cohort study that consisted of 22 of original 46 SSRI exposures and 23 of original 46 controls.<sup>(22)</sup> They examined child behaviour, cognition and maternal mood measures. They concluded that externalizing behaviours, rated by parent / teacher (eg attention, aggression, ADHD, defiant) did not differ between exposed and control groups, but was significantly associated with umbilical cord drug levels (ie, SSRIs) as well as measures of maternal mood at 4 years. When researchers appraised behaviour they concluded that there was no difference between groups for aggressiveness, attention, or emotion. Their final and important conclusion was 'child development, following *in utero* drug exposure occurs in the context of maternal mental illness'.

A prospective, controlled, matched and blinded study by Nulman *et al* has provided additional reassurance regarding SSRI exposure and long term neurodevelopment, as well as some insight into the role of maternal depression on long term neurodevelopment.<sup>(23)</sup> Measurement of Full Scale IQ, Performance IQ and Verbal IQ showed no difference between venlafaxine exposed infants, other SSRI exposed infants and siblings (who were not exposed to an SSRI). However, all IQ measures were greater in healthy controls compared to the other 3 groups. These preliminary results suggest that maternal depression, genetics and environment, and not SSRI exposure, are associated with long term cognitive outcomes.

To study the impact of exposure to an SSRI during pregnancy on long term neurodevelopment is a difficult task. However, the limited number of studies to date suggests that the SSRIs per se do not adversely affect long term neurodevelopment and cognition. Maternal depression, genetics and environment appear to have the greatest

influence.

### Stopping an SSRI During Pregnancy

A 2006 report from the National Birth Defects Prevention Study demonstrated that about 2% of pregnant women (4,094 women studied) were taking an SSRI. <sup>(24)</sup> That percentage dropped approximately 50% between 2 and 3 months after pregnancy. One can only speculate whether the mother's health (and ultimately that of the unborn baby) was compromised. It is recognized that uncontrolled maternal depression may put the fetus at risk due to substance abuse, poor prenatal care, suicide attempts and relationship conflict. <sup>(25)</sup> Maternal depression also increases the risk for poor obstetrical outcome (decreased fetal heart variability, alteration of placental function, increased risk of preterm birth, and a higher risk of low birth weight and IUGR (Intra Uterine Growth Restriction)).

The association between relapse of major depression during pregnancy and maintaining or stopping treatment was shown in a 2006 publication. <sup>(26)</sup> Cohen demonstrated that when relapse occurred only 25% of those women were taking their medication. Three quarters of women who continued to take their medication during pregnancy did not relapse.

### Striking a balance

A reasonable approach to the use of SSRIs during pregnancy approach recognizes the potential catastrophe of stopping an SSRI in a patient with major depression on the one hand and the relatively reassuring data that is currently available. A significant percentage of newborn babies do experience what is believed to be withdrawal. However, this appears to be short lived and is usually complete within the first week or so of life.

Recent studies that have looked at the risk of CDV defects have demonstrated that there is not an increase in babies who were exposed to an SSRI (including paroxetine) *in utero*. While there may be an association between SSRI exposure and the development of PPHN, the increased incidence of this condition is still extremely small, especially in babies greater than 34 weeks gestation. The studies that have looked at cognition and long term

neurodevelopment appear to be reassuring. Whether there is a subtle, long term effect on brain development that is not currently recognized remains to be seen.

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# ISOIMMUNIZATION AND THE ROLE OF INTRAVENOUS IMMUNOGLOBIN FOR THE MANAGEMENT OF HEMOLYTIC ANEMIA OF THE NEWBORN

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## Background

Isoimmunization literally defined as the development of species specific antibodies as a result of antigenic stimulation from the red blood cells of another individual of the same species.

There are four major blood types (A, B, AB, O) in human beings. Each of the four blood types is additionally classified according to presence of the rhesus antigen (Rh), an additional protein present on the surface of red blood cells.

Gene coding for the Rh antigen is located in the short arm of chromosome number 1. There are six genes responsible for the production of Rh antigens C, D, E are the dominant genes and c, d, e are the recessives genes.

Rh positive persons may be DD (homozygous) or Dd (heterozygous). The rhesus gene is inherited in a mendelian fashion. When D is absent from both chromosomes (dd) an individual is Rh negative. If father is heterozygous Rh positive (Dd) and mom is Rh negative (dd) there is 50% chance of the baby being Rh positive. In North America and Europe only 15 % of individuals are Rh negative.

Isoimmune hemolytic disease of the fetus and newborn (also known as erythroblastosis fetalis) is due to fetal-maternal blood group incompatibility and is caused by the

production of maternal antibodies against fetal blood group antigen. Despite wide spread use of anti-D immune globulin (Rhogam), Rh incompatibility remains the most important cause of erythroblastosis fetalis. Other cases may result from incompatibility with other, rarer erythrocyte antigens such as Kell, c, E, C, duffy, MNS, Kidd.<sup>1</sup>

ABO incompatibility is a common cause of mild hemolytic disease of newborn. It does not usually cause severe erythroblastosis or death in utero. In fact, coexistence of ABO incompatibility has been reported to significantly decrease the risk of hemolytic diseases of newborn.<sup>2</sup> The reduced risk of Rh sensitization with ABO incompatibility may result from the rapid clearance of incompatible red cells thus reducing the overall exposure to D antigen.

Hemolytic anemia of the fetus and newborn caused isoimmunization is an important cause of infant morbidity and mortality. The overall incidence of Rh hemolytic disease of the newborn is 10.6 cases per 10,000 total births.<sup>3</sup> The most common cause of Rh isoimmunization is the failure to use anti-D immune globulin appropriately during the antepartum and postpartum period.

Blood production in the fetus begins at about 3 weeks and Rh antigen has been identified in the red cell membrane as early as 38 days after conception<sup>4</sup>. When baby is Rh positive and Mom is Rh negative there is a setup for isoimmunization. Maternal exposure to fetal

antigens does not usually happen until very late in pregnancy or during child birth. The initial response to D antigen is slow. It may take as long as 6 months to develop maternal antibodies. The antibody produced during the first exposure is IgM which does not cross the placental barrier. Most of the time, the first baby does not experience significant hemolysis.

Maternal Rh sensitization can also occur during a miscarriage or therapeutic abortion if the fetus is Rh positive. In this circumstance subsequent pregnancies may be affected. In very rare cases a woman can also become sensitized if she receives an incompatible blood transfusion. Re-exposure to the antigen in subsequent pregnancies produces a rapid immunological response usually measured within days. The sensitized mother produces IgG anti-D antibody that crosses the placenta and coats D-positive fetal red cells that are then destroyed in the fetal spleen.

In recent years the incidence of isoimmunization due to irregular antibodies is increasing while the incidence of Rh incompatibility is declining as use of anti-D immunoglobulin increases<sup>5</sup>.

Isoimmunization due to Kell antigen may also cause fetal and neonatal anemia. The Kell protein is a 93-kd transmembrane metalloproteinase.<sup>6</sup> The antigenic nature of this protein can induce a significant immune response in women exposed to Kell antigen positive red blood cells through blood donations or transplacental passage of fetal RBCs during pregnancy. The antibodies to Kell antigen are IgG in nature and therefore, readily cross the placenta.<sup>7</sup> Anemia in Kell isoimmunization occurs not only because of hemolysis but is also due to erythroid suppression.<sup>8</sup> Antibodies to c and E antigens are also found frequently during pregnancy.<sup>5</sup>

The extent of hemolysis in most cases of isoimmunization depends on the titre or concentration of the antibody. In low concentrations there is mild anemia and jaundice whereas high antibody concentrations lead, progressively, to severe anemia, heart failure and hydrops fetalis. In the case of Kell and other weak antigens, fetal response may not correlate as strongly with the antibody titres.<sup>8</sup>

Prenatal screening is crucial for diagnosis and anticipating the outcome. During first antenatal visit, each patient's blood should be tested for ABO, Rh as well as screened for the presence of antibodies by an indirect coombs test. Any erythrocyte antibody present must be specifically identified and appropriate antibody titers determined to identify the potential risks to the fetus. A titre of >1:4 in Rh is considered sensitized and a four fold increase is more significant.<sup>1,2,11</sup> In the case of anti-Kell antibody a titre of more than 1:32 is significant in terms of the risk for hemolytic anemia.<sup>9</sup> Maternal antibody titers should be monitored regularly to assess the level of antibodies and if high these women need to be followed by the high-risk MFM service. Follow-up will likely include fetal ultrasound to locate the middle cerebral artery doppler flow (anemia study) as well as monitoring for early evidence of fetal hydrops. Amnio-centesis may be necessary to monitor breakdown products of RBC destruction associated with fetal anemia.<sup>1,2,10</sup>

## Management

### Prevention

Preventive management is the best treatment for isoimmunization especially Rh incompatibility. Rh negative, unsensitized patients should receive Rh immunoglobulin (RhIgG) 300mcg (300 mcg covers 15 ml cells) at 28 weeks. Post partum, a second dose of Rh IgG is given within 72 hours of delivery if the infant is Rh positive. In women who are already sensitized, there is no benefit to using Rhogam.<sup>1,2,10</sup>

### Antenatal

Amniocentesis is usually recommended at 16-20 weeks if the antibody titer is elevated (>1:32) to determine the fetal blood group and degree of anemia. Cordocentesis or percutaneous umbilical blood sampling (PUBS) may also be considered to identify fetal blood type and the degree of anemia. In rare cases, fetal DNA testing for blood group incompatibilities (RhD, Rhe, Rhc, RhC and Kell) is performed.<sup>1,2,10</sup> If fetal antigen is negative then no further testing is necessary. If antigen is positive pregnancy is followed with serial titers and need to arrange ultrasound as long as titers remain below the critical value<sup>1,2,10</sup>. Management may include

intrauterine blood transfusion via umbilical vein and exchange transfusion in the presence of severe anemia.

### Postpartum

Conventional treatment for hemolytic disease of the newborn has included phototherapy, blood transfusions, the use of phenobarbital to stimulate hepato-biliary enzymes and, in severe cases, exchange transfusion.

In the recent years the use of IVIG has been shown to be highly effective in the treatment of hemolytic anemia due to Rh and ABO incompatibility. The use of IVIG postnatally significantly reduces the need for exchange transfusion. IVIG has also been administered directly to fetus, both in combination with or without intrauterine intravascular transfusion for severe RhD hemolytic anemia<sup>17</sup>.

The exact mechanism of action of IVIG in hemolytic anemia is still unclear. It is suggested that IVIG prevents the destruction of sensitized erythrocytes in the reticuloendothelial system (RES) through binding of the Fc receptors<sup>18</sup>. Although IVIG is effective in the treatment of neonatal immune hemolytic anemia, the timing and extent of its effect are variable. Usually, a single dose of 0.5gm/kg/dose to 1gm/kg/dose IV given over 2 to 4 hours is effective.<sup>19</sup> In some instances, particularly if the antibody titer is high, a second dose is required.<sup>20</sup>

Recent systematic reviews show that IVIG appears to be safe and may be considered in special circumstances as parental refusal for exchange transfusion or where appropriate blood components for exchange are not available. The *routine* use of IVIG in the treatment of isoimmune hemolytic jaundice is not yet recommended.<sup>21</sup> However, in considering the alternatives, one must take into account the mortality and morbidity rates (0.35 to 1.2%, 5% respectively) associated with exchange transfusion. Complications reported with this procedure include: anemia, sepsis, necrotizing enterocolitis, graft versus host disease and portal hypertension.<sup>22</sup> In weighing the risks and benefits of various therapeutic options, it must be recognized that IVIG is not without adverse effects. Reported adverse events include: allergy, fluid overload and hemolytic anemia. In addition, IVIG is a blood product obtained from pooled donor plasma and may contain a variety of

erythrocyte antibodies (anti-A, anti-B). This may result in hemolysis and a positive coombs test. The incidence of late anemia requiring blood transfusion is increased in babies who received IVIG to treat isoimmune hemolysis. This is explained by the fact that the half life of IVIG is approximately 21 days and while it fades the remaining antibody coated erythrocytes will bind to the Fc sites on the surface of the RES, causing hemolysis.

### Conclusion:

In conclusion, IVIG may be seen as a safe, appropriate and effective therapy for management of hemolytic anemia in newborn.

Since IVIG decreases or reduces hemolysis but does not effectively remove bilirubin, phototherapy needs to be used in conjunction with IVIG until bilirubin levels are within the target range. Clinicians are reminded of the need to follow patient for the potential of late anemia.

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## DID YOU KNOW . . .

The Newborn Skin and Wound Care Manual is expected to be available by Fall 2008. Ordering information and costs will be forthcoming soon.

## For Your Information . . .

### PERIOD OF PURPLE CRYING

Thanks to a grant from the Children's Health Foundation and support from leadership and staff, London hospitals will be the first to implement *The Period of PURPLE Crying®*. A program that gives new parents education on understanding and managing their newborn's crying. The program was launched in April 2008 at LHSC and is coming to St Joseph's Hospital this fall. This program is a Shaken Baby Syndrome Prevention Program of the National Center on Shaken Baby Syndrome (NCSBS), USA.

In London, nursing staff will provide individual education to each new parent(s) including a take-home 11-page colour booklet and DVD, focusing on the positive message of coping with infant crying based on over 25 years of research conducted by Dr. Barr MDCM, Professor of Pediatrics, University of British Columbia, Vancouver, British Columbia. Similar programs have shown positive effects, with a significant decrease in the incidence of Shaken Baby Syndrome.

For more information about the program go to [www.dontshake.org](http://www.dontshake.org). For information about implementation, please contact the perinatal members of the Shaken Baby Syndrome Prevention Working Group for:

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## WELCOME

The Perinatal Outreach Program is pleased to welcome Kelly Barzsa-Jenkins to the Team as part-time Perinatal Nurse Consultant.



Kelly has been a Registered Nurse in Ontario for 19 years. Her work experience, though varied, has primarily been in the area of Obstetrics. Her career has taken her to Markham-Stouffville Hospital in Markham; Zone Hospital in Sioux Lookout; and most recently, St. Joseph's Health Care London in the Family Birthing Centre and Mother/Baby unit. Kelly has recently completed her BScN through Lakehead University. She is also a qualified NRP instructor. Kelly will be getting familiar with the South West region as she travels with the team this fall on Nursing and Team visits. You can reach Kelly at (519) 646-6100 x 65900, or [Kelly.barzsa-jenkins@sjhc.london.on.ca](mailto:Kelly.barzsa-jenkins@sjhc.london.on.ca)

## For your information:

### IMPLICATIONS FOR NEONATAL RESUSCITATION: Study indicates that self inflating resuscitation bag may deliver high oxygen concentrations when used without an oxygen reservoir:

Kathy Johnston RRT  
IWK Health Centre, Halifax NS

Khalid Aziz, MA, FRCPC, FRCPC  
University Of Alberta, Edmonton, Alberta,  
Canada

The 5<sup>th</sup> edition of the AAP NRP textbook states that self-inflating bags without a reservoir deliver approximately 40% oxygen.<sup>1</sup> This differs from the manufacturer's specifications for the preterm size Laerdal Resuscitator.<sup>2</sup> The Laerdal user's information cites the delivered concentration of oxygen as being much higher than 40% when the resuscitation bag is used without a reservoir. To test this discrepancy, a study was conducted at the IWK Health Centre, Halifax.

**Objective:** To measure the delivered fractional oxygen concentration ( $FdO_2$ ) from preterm size Laerdal silicone resuscitators (PSLR) without a reservoir.

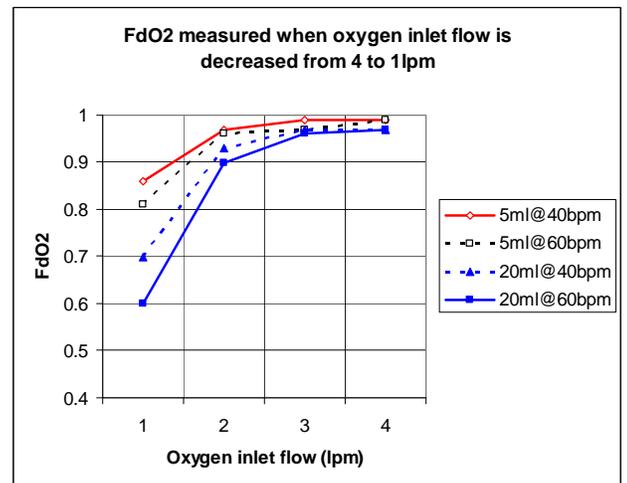
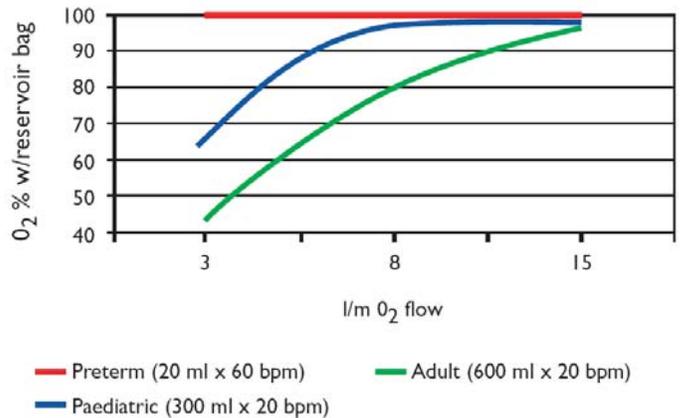
**Methods:** A neonatal test lung was manually ventilated using PSLRs *without* a reservoir. A 50psi 100% oxygen source and oxygen flowmeter were used to provide desired oxygen inlet flows.  $FdO_2$  was measured using 3 different PSLRs after 4 minutes of manual ventilation of a neonatal test lung, at differing inspired tidal volumes (5 or 20ml), respiratory rates (40 or 60 bpm) and oxygen inlet flows (1 to 4, 5 and 10lpm).

**Results:** In all tests using 5 or 10lpm,  $FdO_2$  exceeded 0.95. The lowest  $FdO_2$  was 0.59 at 1lpm.

**Conclusions:** The  $FdO_2$  measured during this study did not differ from PSLSR specifications: The  $FdO_2$  did, however, differ from information contained in the NRP manual regarding use of a self-inflating bag without a reservoir. Care should be taken when selecting a self-inflating resuscitation device to provide blended air and oxygen, as high concentrations of oxygen may be delivered by these devices even when the reservoir is

removed. American and Canadian recommendations for the provision of supplemental oxygen with self-inflating bags require re-evaluation.

**Key words:** Infant-preterm, infant-newborn, resuscitator,  $FdO_2$ , neonatal resuscitation, positive pressure ventilation, oxygen, blender.



### References

1. American Academy of Pediatrics. Textbook of Neonatal Resuscitation, 5th edition. 2006 pp 3-12.
2. Laerdal Medical, Stavanger Norway. Feasible Oxygen Concentrations. Laerdal Silicone Resuscitators, Directions for Use. Rev. 2004

Many thanks to Kathy Johnston, IWK Health Centre, Halifax, Nova Scotia for submitting this abstract. For further information please contact her at: [kathy.johnston@iwk.nshealth.ca](mailto:kathy.johnston@iwk.nshealth.ca)

## You asked us: . . .

**Q:** Could you please clarify the services and the referral protocols for the Neonatal Developmental Follow Up Clinic at St. Joseph's Health Care London?

**A:** The **Developmental Follow Up Clinic** (DFC) provides extended services to the families and children treated in the Neonatal Intensive Care Unit (NICU) at St. Joseph's Health Care due to prematurity or other complications early in life, the children may require monitoring of their growth and development on a regular basis.

Children that need follow up will be referred to the clinic by the NICU staff. A clinic nurse will contact the family to introduce the follow up service and to book appointments. Some families will be referred to other follow up programs if they are from out of region. Infants who were treated in another NICU and are now living in the South Western Ontario region may be referred to the DFC by the hospital or follow up clinic in their birth region.

The criteria for referral to the DFC at St. Joseph's Health Centre London are as follows:

- Birth weight < 1250 gms and/or < 29 weeks gestation at birth (standing order)
- Chronic Lung Disease
- Grade III or IV intraventricular hemorrhage
- Periventricular Leukomalacia
- Hypoxic ischemic encephalopathy
- Meningitis
- Persistent seizures
- Apnea beyond 37 weeks
- Significant hearing or visual impairments
- Specific research
- Others (as deemed necessary by the neonatologists. These may include IUGR, largest twin if the other twin fits the weight criteria, complex psychosocial situations, post surgical necrotizing enterocolitis.)

The DFC staff consists of Developmental Paediatricians, Nurses, Physiotherapists, a Clinical Dietitian and a secretary.

The clinic provides screening and/or assessment in all areas of development to ensure growth and development is occurring normally. Assessments are made at term gestation, 4 months of age, 8 months, 1 year, 2 years, and 3 years corrected age. Other visits may be scheduled as necessary. Physiotherapy and nutrition services are provided to a select group of these high risk infants. The Developmental Follow Up Clinic can be contacted at (519) 646-6120

### Developmental Resources for Infants (DRI)

Developmental Resources for Infants (DRI) is a virtual organization that coordinates services for infants and children under the age of two years in Middlesex, Elgin, Oxford, Huron and Perth counties, DRI partners include:

Child Parent Resource Institute  
Home Visiting Program for Infants  
London, Ontario

Children's Hospital of Western Ontario  
London, Ontario

St. Joseph's Health Centre  
Developmental Follow Up Clinic  
London, Ontario

Thames Valley Children's Centre  
London, Ontario

Infants or children are often referred to DRI from community Physicians, Public Health Nurses, Children's Aid and by families for developmental screening and treatment. Infants and children from the Developmental Follow Up Clinic that require ongoing treatment are also referred to DRI for services through the Home Visiting Program for Infants or the Thames Valley Treatment Centre.

The patient intake process and service coordination is initiated by the DRI Intake Coordinator. Referrals can be made by calling the DRI referral line at (519) 685-8710.

Children with developmental needs who live outside of the DRI catchment area are directly referred to the Infant Parent Program or treatment centre in their area.

## Out of Region Neonatal Follow Up Clinics

For infants requiring developmental follow up in areas outside of the London catchment area there are several neonatal follow up clinics throughout the province as listed below. Each clinic may vary somewhat as to their referral criteria.

BARRIE	NEONATAL FOLLOW UP The Royal Victoria Hospital 201 Georgian Drive Barrie, Ontario L4M 6M2 <i>Ph</i> : (705) 728-9090 Ext 47145 <i>Fax</i> : (705) 739-5674
HAMILTON	McMASTER GROWTH & DEVELOPMENT CLINIC Hamilton Health Sciences PO Box 2000, Station "A" Hamilton, ON L8N 3Z5 <i>Ph</i> : (905)521-2100 Ext 76548 / 76552 <i>Fax</i> : (905) 521-5056 (2Q)
KINGSTON	SPECIAL INFANT CLINIC Hotel-Dieu Hospital 166 Brock Street Kingston, ON K7L 5G2 Referrals go to: Dr Vanwylie Fax: (613) 545-3559 Dr Vanwylie Office <i>Ph</i> : (613) 544-3310 Ext 3359 Clinic Reception desk <i>Ph</i> : (613) 544-3310 Ext 3150
MISSISSAUGA	CREDIT VALLEY GROWTH & DEVELOPMENT CLINIC The Credit Valley Hospital 2200 Eglinton Ave East Mississauga, ON L5M 3N1 <i>Ph</i> : (905) 813-1100 Ext 6716 <i>Fax</i> : (905)813-4022
	NEONATAL FOLLOW UP PROGRAM Trillium Health Centre 100 Queensway West Mississauga ON L5B 1B8 <i>Ph</i> : (905) 848-7580 <i>Fax</i> : (905) 804- 7996
OTTAWA	NEONATAL FOLLOW UP CLINIC Children's Hospital of Eastern Ontario 501 Smyth Rd Ottawa, ON K1H 8L1 <i>Ph</i> : (613) 737-2534 <i>Fax</i> : (613) 738-4847

THUNDER BAY INFANT GROWTH AND DEVELOPMENT CLINIC  
George Jeffrey Children's Centre  
507 North Lillie St  
Thunder Bay, ON P7C 4Y8  
*Ph*: 807-623-4381  
*Fax*: 807-623-7161

\*note- clinic is moving in Nov 2008 check website [www.georgejeffrey.com](http://www.georgejeffrey.com)

### TORONTO AREA

NORTH YORK DEVELOPMENTAL CLINIC  
Branson Site  
555 Ave West, 5<sup>th</sup> Floor  
Toronto, ON M2R 1N5  
*Ph*: (416) 632-8703  
*Fax*: (416) 632-8704

TORONTO REGIONAL NEONATAL FOLLOW UP PROGRAM (HSC, MOUNT SINAI)  
The Hospital for Sick Children  
3<sup>rd</sup> Floor Atrium  
555 University Ave  
Toronto, ON M5G 1X8  
*Ph*: (416) 813-5879  
*Fax*: (416) 813-8969

Satellite clinics- Peel Memorial, Oshawa Gen.  
Discharge summaries- contact Amuna (416) 586-4800 ext 8297

NEONATAL FOLLOW UP CLINIC  
Women's College Hospital  
76 Grenville Street  
Toronto, ON M5S 1B2  
*Ph*: (416)323-7746  
*Fax*: (416) 323-6335

Satellite clinics in Scarborough, Thornhill, Whitby, Brampton Ajax, Pickering

WINDSOR NEURODEVELOPMENTAL FOLLOW UP PROGRAM  
Windsor Regional Hospital  
Metropolitan Campus  
1995 Lens Ave  
Windsor, ON N8W 1L9  
*Ph*: (519) 254-5577 ext 52488  
*Fax*: (519) 255-1735

## Upcoming events:

### MARK YOUR CALENDARS . . .

#### MATERNAL NEWBORN NURSING COURSE

##### London:

Spring 2009

**Mondays: Mar. 23 – May 11, 2009**

St. Joseph's Health Care, London  
Offered in collaboration with Fanshawe College.  
Continuing Education: NRS-6027  
Videoconferencing available

##### **Contact:**

Gwen Peterek  
Perinatal Outreach Program  
Phone: (519) 646-6100 ext 65901  
Fax: (519) 646-6172  
[Gwen.peterek@sjhc.london.on.ca](mailto:Gwen.peterek@sjhc.london.on.ca)

**check out our webpage to download a form:**

[www.sjhc.london.on.ca/sjh/profess/periout/education.htm](http://www.sjhc.london.on.ca/sjh/profess/periout/education.htm)

#### LUNCH & LEARN VIDEOCONFERENCE SERIES

##### **"BABY TALK – LESSONS FROM THE NICU"**

- SEP. 16, 2008** Management of Neonatal Hypoglycemia  
**OCT. 21, 2008** Increasing Compassionate Care for Substance Abusing Women & their Newborns  
**Nov. 18, 2008** "Period of Purple Crying": A Shaken Baby Syndrome Prevention Program  
**DEC. 16, 2008** Neonatal Skin Care

**Watch our webpage for further details:**

[www.sjhc.london.on.ca/sjh/profess/periout/education.htm](http://www.sjhc.london.on.ca/sjh/profess/periout/education.htm)

**Or visit the Ontario Telehealth Network webpage:**

[http://TEST1.VIDEOCARE.CA/OTN/EVENTS\\_CALENDAR.PHP?MODE=VIEW](http://TEST1.VIDEOCARE.CA/OTN/EVENTS_CALENDAR.PHP?MODE=VIEW)

#### 19<sup>TH</sup> ANNUAL AWHONN CANADA CONFERENCE

##### **"POWER, PASSION, POLITICS"**

**October 23-25, 2008**

Location: Westin Hotel, Ottawa, ON

**Contact:** AWHONN website for more details

<http://www.awhonn.org/awhonn/section.by.state.do?state=Canada>

#### THE 3<sup>RD</sup> ANNUAL IVEY SYMPOSIUM

##### **"MEDICATIONS IN PREGNANCY AND BREASTFEEDING – WHAT IS SAFE AND WHAT IS NOT?"**

**October 21, 2008**

**Location:** Shuttleworth Auditorium  
St. Joseph's Health Care London

**Contact:** Maud Rouleau (519) 661-3128

**Email:** [iveytox@uwo.ca](mailto:iveytox@uwo.ca)

#### CHN FOR THE GTA & THE ACORN NEONATAL SOCIETY **"ACORN INSTRUCTOR WORKSHOP"**

(see pre-requisites)

**Oct. 27-28, 2008**

**Location:** The Credit Valley Hospital, Mississauga

**Contact:** Moya Johnson (416) 813-6507

**Email:** [moya.johnson@sickkids.ca](mailto:moya.johnson@sickkids.ca)

#### PREGNANCY RELATED ISSUES IN MANAGEMENT OF ADDICTIONS (PRIMA) WORKSHOP

**Monday, Oct. 27, 2008**

**Location:** Aristos Banquet Hall, Chatham, ON

**Contact:** (519) 352-6401 x 6670

**Email:** Paula Morrison: [pmorrison@ckha.on.ca](mailto:pmorrison@ckha.on.ca)

#### U OF T DEPT. OF OBSETRICS & GYNAECOLOGY

##### **"2008 FETAL MEDICINE UPDATE"**

**October 24-25, 2008**

##### **"CHALLENGES IN INTRAPARTUM CARE"**

**Saturday, November 8, 2008**

**Location:** Mount Sinai Hospital, Toronto, ON

**Contact:** Elizabeth Gan

(416) 586-4800 x 2489

[www.mtsinai.on.ca/seminars/ce](http://www.mtsinai.on.ca/seminars/ce)

#### WINDSOR-ESSEX COUNTY HEALTH UNIT & THE WINDSOR MATERNAL CHILD HEALTH COALITION

##### **"PREGNANCY AND WEIGHT GAIN: WHAT DOES IT REALLY MEAN?"**

**Friday, November 14, 2008**

**Location:** Giovanni Caboto Club, Windsor

**Contact:** Jennifer Bially

Tel: (519) 258-2146 x 1358

[jbially@wechealthunit.org](mailto:jbially@wechealthunit.org)

Register online: <http://www.wechealthunit.org/>

#### Acute Care of at-Risk Newborns "ACoRN" Workshop

**June 3 – 5, 2009**

**Location:** Lamplighter Inn, London

**Contact:** Perinatal Outreach Office  
(519) 646-6100, ext. 65859

**Call for a brochure or download one from our webpage:**

[www.sjhc.london.on.ca/sjh/profess/periout/education.htm](http://www.sjhc.london.on.ca/sjh/profess/periout/education.htm)

This newsletter is a publication of the Perinatal Outreach Program.

Letters, queries and comments may be addressed to:

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[www.sjhc.london.on.ca/sjh/profess/periout/periout.htm](http://www.sjhc.london.on.ca/sjh/profess/periout/periout.htm)