Prenatal Diagnosis of Restrictive Foramen Ovale: A Case Report

By Reem S. Abu-Rustum, MD, FACOG, FACS; Khalil N. Abi-Nader, MD; Mahmoud Zaghloul, MD; Linda Daou, MD

Isolated restriction of the Foramen Ovale (FO) is a rare entity that may lead to fetal hydrops secondary to right-ventricular volume overload and subsequent failure. The ductus venosus is the source of the highest oxygen-containing blood with the highest kinetic energy within the inferior vena cava. This jet emanating from the ductus venosus is what maintains the patency of the FO as it streamlines through it into the left atrium.¹ The earliest reports of prenatal diagnosis of restrictive FO date back to the 1980’s.² However, most prenatally diagnosed isolated cases of Hydrops Fetalis thought to have been caused by a restrictive FO have been found to be secondary to an underlying cardiac lesion when examined postnatally.³⁻¹¹

Case

A 33-year-old, Gravida 7, Para 4115, rhesus positive, was referred at 36w2d for new onset polyhydramnios and fetal ascites after a seemingly uncomplicated antenatal course. The medical and family history was non-revealing. On ultrasound examination, fetal hydrops was evident. Generalized fetal edema with a scalp thickness of 21 mm (Figure 1), an abdominal skin thickness of 17 mm, significant ascites (Figure 2) and a small pleural effusion (Figure 3) were noted. Polyhydramnios with an AFI of 33 cm was also present. Fetal echocardiography revealed a 4-chambered heart with normal situs and axis, a concordant outflow tract arrangement and normal venous return. There was an accentuated right-sided ventricular predominance. The aorta was of normal caliber (6.7mm), while the pulmonary artery was moderately dilated (10mm) at the level of the 3 vessel view. The ductus arteriosus was patent and slightly enlarged. The FO remained stiff with minimal motion throughout the cardiac cycle, without the normal “flapping” motion (Figures 3 and 4), and peak systolic velocities reaching 100cm/sec (Figure 5). Umbilical artery Dopplers were normal. Uterine artery pulsatility index was normal on the left; however, on the right it was increased (1.97) with notching. The middle cerebral artery Doppler peak systolic velocity was not measured. Fetal cardiac function evaluation showed right ventricular systolic dysfunction with tricuspid regurgitation. Fetal anatomy was otherwise normal and the estimated fetal weight was 3660 grams. The patient’s blood pressure was at 140/100mm Hg.

After extensive counseling, the couple was referred to pediatric cardiology. The diagnosis of...
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an isolated restrictive FO was confirmed and labor induction ensued. Within 36 hours of presentation, the patient delivered a live born, hydropic female weighing 3960 grams (Figure 6). The baby was intubated at birth. The hydrops was so extensive, skin ulcerations were present. The entire newborn’s workup was negative except for hypoalbuminemia and hyperbilirubinemia. Fetal anemia was absent. Diuresis with Lasix was the main therapy. The baby was discharged home on Day of Life 12, weighing 2710 grams, with residual mild mitral regurgitation which resolved within 3 weeks. Of note is that, postnatally, the baby developed supraventricular tachycardia that has been medically controlled and, currently, the baby is alive and growing well at age 4 years.

The patency of the FO in utero is maintained by a high-oxygen containing jet, emanating from the ductus venosus within the inferior vena cava, that streamlines through the FO into the left atrium.\(^1,3,12\) Postnatal closure of the FO occurs as a consequence of decreased pressure in the vena cava and increased pulmonary venous return which raises the left atrial pressure.\(^12\) Premature isolated in utero restriction/closure of the FO is a rare entity.\(^1,3\) Most cases occur in association with left heart lesions, a functionally impaired left ventricle, primary lesions of the mitral or aortic valve,\(^4,5\) aneurysmal dilations of the right atrial fossa ovalis region,\(^6\) or fetal tachycardia.\(^3,7-11\) Some of the earliest reports of in-utero detection date back to the early 1980’s\(^2,4\) and in most cases, the resulting hydrops is so severe that it may lead to in-utero fetal demise. A true isolated primary restrictive FO may be due to a localized developmental abnormality\(^3\) or an in utero insult such as myocarditis. The concept that a primary obstruction of the FO is responsible for the Left Heart Hypoplastic Syndrome\(^11\) has now been abandoned.\(^14\)
In our case, the prenatal and post-natal echocardiography showed no evidence of an associated cardiac lesion. In addition, the inter-atrial septum seemed straight, making the presence of an aneurysm highly unlikely. A lesion of the flap itself may have been the culprit. This remains speculative with the impossibility of a pathological examination. The postnatally detected supraventricular tachycardia, which is an in-utero cause of fetal hydrops, could have escaped prenatal detection. However, it was not present at the time of any of the obstetrical scans, nor at the fetal echocardiography nor on intrapartum external fetal monitoring. It may also have been a late consequence of in-utero right atrial dilation. Alternatively, myocardiitis due to a cardiotropic virus such as Parvovirus or Coxsackie B may have caused ventricular dysfunction and secondary inter-atrial pressure imbalance leading to restriction of the FO. The development of fetal hydrops is, however, dependent not only on the degree of restriction in the FO, but also on the compensatory ability of the right ventricle and flow through the ductus arteriosus. In the context of our case, an isolated restriction of the FO seems to be the most probable explanation though a solid conclusion regarding the pathogenesis cannot be reached. Hydrops in fetuses with a primary restrictive FO may develop when the right ventricle fails as a consequence of volume overload. When a restrictive FO is the only underlying cause, complete reversal of the hydrops frequently ensues with timely delivery. Even in the absence of hydrops, impaired cardiac function with the incipient risk of cardiac de-compensation is probably an indication for delivery in the relatively mature fetus. Interestingly enough, maternal hypertension was evident and this could be a rare case of Mirror Syndrome which has not been previously reported in association with an isolated restrictive FO.

As our case demonstrates, whenever there is unexplained fetal hydrops, right-sided heart failure due to a restrictive FO should be suspected, and thorough structural and functional evaluation of the fetal heart must be performed. A restrictive FO, though rare, may be the cause. Prompt delivery depending on fetal gestational age may be instrumental for the survival of the neonate.

References


This case was presented as poster P03.09 at the 20th World Congress of the International Society of Ultrasound in Obstetrics and Gynecology. Prague 2010.

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“As our case demonstrates, whenever there is unexplained fetal hydrops, right-sided heart failure due to a restrictive FO should be suspected, and thorough structural and functional evaluation of the fetal heart must be performed. A restrictive FO, though rare, may be the cause. Prompt delivery depending on fetal gestational age may be instrumental for the survival of the neonate.”


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- Optionally, a picture of the author(s) may be submitted.
- No abstract should be submitted.
- The main text of the article should be written in informal style using correct English. The final manuscript may be between 400-4,000 words, and contain pictures, graphs, charts and tables. Accepted manuscripts will be published within 1-3 months of receipt. Abbreviations which are commonplace in pediatric cardiology or in the lay literature may be used.
- Comprehensive references are not required. We recommend that you provide only the most important and relevant references using the standard format.
- Figures should be submitted separately as individual separate electronic files. Numbered figure captions should be included in the main Word file after the references. Captions should be brief.
- Only articles that have not been published previously will be considered for publication.

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“Social media, it has been said, is not a fad, but a fundamental shift in the way we communicate. Like rock and roll and many other revolutionary changes, we can decide to embrace or resist, it’s here to stay. So, we might as well get to know what it’s all about.”

So, again, why is this important to know? Social media has evolved into a manner of communication that impacts our lives in surprisingly significant ways.

There is an overabundance of stories of the impact of social media. To name a few, I will begin with the Facebook Kawasaki diagnosis story. In May 2011, a young boy’s life was saved by the concerns of a supportive social online community, which included a pediatric cardiologist. Unsuspecting photos posted by a mother with her son at the pediatrician’s office, waiting for a prescription for Strep throat prompted so many comments that she returned with her persistently-ill boy to the ER, to be correctly diagnosed.5

In April 2011, President Obama held a town hall forum with the nation utilizing the real-time discussion and video capabilities on Facebook. This was a refreshing change from the traditional, one-way, televised broadcasts of previous Presidents. President Obama continues to utilize social media. In August 2012, he created a “Ask Me Anything” (AMA) page on the social news site, Reddit, where he amassed over 3.8 million page views and was able to send his message to “remember to vote in November.”6 In February 2013, he held a live video Google + Fireside Hangout with the nation.7 Most recently, on February 24, 2014, the President met to discuss health care with Zach Galifianakis “Between Two Ferns.”8 This interview has reached over 4,838,512 views on YouTube. Ingenious, and, again, refreshing.

Speaking of refreshing, Pepsi has engaged people of the world primarily through social media by creating the Pepsi Refresh Project. This project aimed to make the world a better place with its innovative concept of awarding grants to novel ideas that will “Refresh the World” lasted from 2010 until 2012.9 The turmoil in Egypt in early 2011 was primarily relayed to the world through Twitter and YouTube. It may be argued that these events were accelerated by the revelation and viral spread through the Internet.10

Social media, it has been said, is not a fad, but a fundamental shift in the way we communicate. Like rock and roll and many other revolutionary changes, we can decide to embrace or resist, it’s here to stay. So, we might as well get to know what it’s all about.

How did we get to this point? Web 1.0, circa 1990-2000, is a term to define the Internet, as we understood it as its inception. The Internet at that time, was used primarily for e-mail and as a source of information Web 1.0 users visited Internet sites and extracted information as if it were a digital library. Web masters generated the information, and users viewed the information as they needed. Web 2.0, the internet as it has evolved this past decade, is no longer only a source of content for passive viewing, but has become an interactive, dynamic arena for idea and information exchange. More commonly now, as we settle into the era of Web 3.0, personalized exchange of ideas occurs in many applications in real-time.11 Social media is defined as a “group of Internet-based applications that build on the foundations of Web 2.0, and that allow the creation and exchange user-generated content.”12 One of the most important concepts of social media is the “democratization” of ideas, which means that it allows its participants to become producers of information as well as users of those ideas.13 What makes social media “social” is the interaction of all its participants. Consumers and creators of ideas and information are one in the same.

Social media encompasses many different Internet-based applications, allowing for the creation and exchange of user-generated content. These various tools include: networks, blogs, microblogs, photos, video, e-mail, wikis and audio. Ultimately and most importantly, at the center of all these applications are conversations. Social media is about reaching out and having a conversation, enabling people to find a common ground to encourage authentic communication. This allows for the creation of relationships between users. Ideally, it’s about keeping people informed and engaged to support dynamic dialogue.

Health 2.0 is the use of Web 2.0 tools by all the healthcare communities, including clini-
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INDICATION
SURFAXIN® (lucinactant) Intratracheal Suspension is approved by the FDA for the prevention of respiratory distress syndrome (RDS) in premature infants at high risk for RDS.

IMPORTANT SAFETY INFORMATION
SURFAXIN (lucinactant) Intratracheal Suspension is intended for intratracheal use only. The administration of exogenous surfactants, including SURFAXIN, can rapidly affect oxygenation and lung compliance. SURFAXIN should be administered only by clinicians trained and experienced with intubation, ventilator management, and general care of premature infants in a highly supervised clinical setting. Infants receiving SURFAXIN should receive frequent clinical assessments so that oxygen and ventilatory support can be modified to respond to changes in respiratory status.

Most common adverse reactions associated with the use of SURFAXIN are endotracheal tube reflux, pallor, endotracheal tube obstruction, and need for dose interruption. During SURFAXIN administration, if bradycardia, oxygen desaturation, endotracheal tube reflux, or airway obstruction occurs, administration should be interrupted and the infant’s clinical condition assessed and stabilized. Overall, the incidence of administration-related adverse events did not appear to be associated with an increased incidence of serious complications or mortality relative to the comparator surfactants.

SURFAXIN is not indicated for use in acute respiratory distress syndrome (ARDS).

For more information about SURFAXIN, please visit www.SURFAXIN.com and see accompanying brief summary on the next page.
SURFAXIN® is indicated for the prevention of respiratory distress syndrome (RDS) in premature infants at high risk for RDS.

**INDICATIONS AND USAGE**

SURFAXIN® is indicated for the prevention of respiratory distress syndrome (RDS) in premature infants at high risk for RDS.

**CONTRAINDICATIONS**

None.

**WARNINGS AND PRECAUTIONS**

Acute Changes in Lung Compliance

Administration of exogenous surfactants, including SURFAXIN, can rapidly affect lung compliance and oxygenation. SURFAXIN should be administered only by clinicians trained and experienced in the resuscitation, intubation, stabilization, and ventilatory management of premature infants in a clinical setting with the capacity to care for critically ill neonates. Infants receiving SURFAXIN should receive frequent clinical assessments so that oxygen and ventilatory support can be modified to respond to changes in respiratory status.

**Administration-Related Adverse Reactions**

Frequently occurring adverse reactions related to the administration of SURFAXIN include bradycardia, oxygen desaturation, reflux of drug into the endotracheal tube (ETT), and airway/ETT obstruction.

**Increased Serious Adverse Reactions in Adults with Acute Respiratory Distress Syndrome (ARDS)**

Adults with ARDS who received lucinactant via segmental bronchoscopic lavage had an increased incidence of death, multi-organ failure, sepsis, anoxic encephalopathy, renal failure, hypoxia, pneumothorax, hypotension, and pulmonary embolism. SURFAXIN is not indicated for use in ARDS.

**Clinical Trials Experience**

The efficacy and safety of SURFAXIN for the prevention of RDS in premature infants was demonstrated in a single randomized, double-blind, multicenter, active-controlled, multi-dose study involving 1294 premature infants (Study 1). Infants weighed between 600 g and 1250 g at birth and were 32 weeks or less in gestational age. Infants were randomized to received 1 of 3 surfactants, SURFAXIN (N = 524), colfosceril palmitate (N = 506), or beractant (N = 258). Co-primary endpoints were the incidence of RDS (defined as having a chest x-ray consistent with RDS and an FiO2 > 0.30) at 24 hours and RDS-related mortality at 14 days. The primary comparison of interest was between SURFAXIN and colfosceril palmitate with the intent of demonstrating superiority. Beractant served as an additional active comparator. Compared to colfosceril palmitate, SURFAXIN demonstrated a statistically significant improvement in both RDS at 24 hours and RDS-related mortality through Day 14. A second multicenter, double-blind, active-controlled study involving 252 premature infants was also conducted to support the safety of SURFAXIN (Study 2). Infants weighed between 600 g and 1250 g and were less than 29 weeks in gestational age. Infants received 1 of 2 surfactants, SURFAXIN (N = 119) or poractant alfa (N = 124).

The safety data described below reflect exposure to SURFAXIN administered intratracheally to infants at a dose of 5.8 mL per kg (up to 4 doses) in either 4 aliquots (Study 1) or 2 aliquots (Study 2) in 643 premature infants.

Comparator surfactants colfosceril palmitate and beractant were administered at the recommended doses (5.0 and 4.0 mL per kg, respectively) while the first dose of poractant alfa administered (2.2 mL per kg) was less than the recommended dose of 2.5 mL per kg. Any subsequent doses of poractant alfa were at the recommended 1.25 mL per kg dose.

Overall, the incidence of administration-related adverse reactions was higher in infants who received SURFAXIN compared to other surfactants (Table 1) and resulted in a greater proportion of infants treated with SURFAXIN who experienced administration-related oxygen desaturation and bradycardia. For Study 1, oxygen desaturation was reported in 17%, 9%, and 13% and bradycardia for 5%, 2%, and 3% of infants treated with SURFAXIN, colfosceril palmitate, and beractant, respectively. For Study 2, oxygen desaturation was reported in 8% and 2% and bradycardia in 3% and 2% of infants treated with SURFAXIN and poractant alfa, respectively. These adverse reactions did not appear to be associated with an increased incidence of serious complications or mortality relative to the comparator surfactants (Table 2).

**Table 1. Administration-Related Adverse Reactions in SURFAXIN Controlled Clinical Studies**

<table>
<thead>
<tr>
<th></th>
<th>Study 1</th>
<th>Study 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SURFAXIN (N = 524)</td>
<td>Colfosceril palmitate (N = 506)</td>
</tr>
<tr>
<td>Total Doses Administered</td>
<td>994</td>
<td>1038</td>
</tr>
<tr>
<td>Total Number of Events (Events per 100 Doses)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ETT Reflux</td>
<td>183 (18)</td>
<td>161 (16)</td>
</tr>
<tr>
<td>Pallor</td>
<td>88 (9)</td>
<td>46 (4)</td>
</tr>
<tr>
<td>Dose Interruption</td>
<td>87 (9)</td>
<td>46 (4)</td>
</tr>
<tr>
<td>ETT Obstruction</td>
<td>55 (6)</td>
<td>21 (2)</td>
</tr>
</tbody>
</table>

* Table includes only infants who received study treatment.

**Table 2. Common Serious Complications Associated with Prematurity and RDS in SURFAXIN Controlled Clinical Studies Through 36-Weeks Post-Conceptional Age (PCA)**

<table>
<thead>
<tr>
<th></th>
<th>Study 1</th>
<th>Study 2</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>SURFAXIN (N = 527)</td>
<td>Colfosceril palmitate (N = 509)</td>
</tr>
<tr>
<td>Apnea</td>
<td>52</td>
<td>52</td>
</tr>
<tr>
<td>Intraventricular hemorrhage, all grades</td>
<td>52</td>
<td>57</td>
</tr>
<tr>
<td>-Grade 3/4</td>
<td>19</td>
<td>18</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Acquired sepsis</td>
<td>44</td>
<td>44</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>37</td>
<td>35</td>
</tr>
<tr>
<td>Retinopathy of prematurity, all grades</td>
<td>27</td>
<td>26</td>
</tr>
<tr>
<td>-Grade 3/4</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Necrotizing enterocolitis, all grades</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>-Grade 2/3</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Pulmonary air leak through Day 7, all types</td>
<td>15</td>
<td>17</td>
</tr>
<tr>
<td>-Pulmonary interstitial emphysema</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>-Pneumothorax</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Pulmonary hemorrhage</td>
<td>10</td>
<td>12</td>
</tr>
</tbody>
</table>

All-cause mortality through 36-weeks PCA was similar regardless of which exogenous surfactant was administered.

Adverse reactions reported in the controlled clinical studies through 36-weeks PCA occurring in at least 10% of infants were anemia, jaundice, metabolic acidosis, oxygen desaturation, hyperglycemia, pneumonia, hyponatremia, hypotension, respiratory acidosis, and bradycardia. These reactions occurred at rates similar to the comparator surfactants.

No assessments for immunogenicity to SURFAXIN were performed in these clinical studies.

**Follow-up Evaluations**

Twelve-month corrected-age follow-up of 1546 infants enrolled in the 2 controlled clinical studies demonstrated no significant differences in mortality or gross neurologic findings between infants treated with SURFAXIN and those treated with the comparator surfactants (colfosceril palmitate, beractant, or poractant alfa).

**OVERDOSAGE**

There have been no reports of overdose following the administration of SURFAXIN.

**HOW SUPPLIED/STORAGE AND HANDLING**

SURFAXIN (lucinactant) Intratracheal Suspension is supplied sterile in single-use, rubber-stoppered, clear glass vials containing 8.5 mL of white suspension (NDC 68628-500-31). One vial per carton.

Store SURFAXIN in a refrigerator at 2° to 8°C (36° to 46°F) and protect from light until ready for use. Do not freeze. Vials are for single use only. Discard any unused portion of SURFAXIN. Discard warmed vials of SURFAXIN if not used within 2 hours of warming.

To report SUSPECTED ADVERSE REACTIONS, contact Discovery Laboratories, Inc. at 1-877-SURFAXN (877-787-3296) or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

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We can say things to each other that our friends and family may not want to hear, which is a significant advantage of e-communication. Peer-to-peer healthcare has a significant role in the development of medical knowledge and the collaboration of the healthcare community. The idea is to utilize these tools to strengthen the relationships and communication between the communities and the collaboration within the communities in the healthcare system.

A report issued in February 2011 stated that, in a survey of 3001 American adults, 8 in 10 Internet users searched online for health information. Furthermore, the report revealed that seeking health information is the 3rd most prevalent activity among American web users.

In February 2011, the National Research Corporation Survey reached out to 23,000 American adults. This report revealed the popularity of social media sites as sources of health information. Forty-one percent answered that they use social media sites to search for health information for themselves, as well as for loved ones. Facebook was the number one site of choice for information, with 94%; YouTube was a distance second place, with 32% of votes. One in four responded that what they learned on these sites was “very likely” or “likely” to impact their future health decisions. Most responded that they also consulted with a physician. This addressed a recurring concern that patients are using the Internet to self-diagnose and self-medicate without reference to healthcare professionals. This survey emphasized the desire of patients for connection to others as well as information, which is what social media provides.

So, who are these “people” or patients that scour the Internet for information? This community is one of e-patients, who are enabled, electronically-equipped, educated, and empowered. As in social media, where they are no longer passive consumers, the e-patient is no longer an unengaged patient consumer; they are now active participants and empowered. As in social media, where they are no longer passive consumers, the e-patient is no longer an unengaged patient consumer; they are now active participants in their own health care. The e-patient can open a road to a new and different kind of relationship with his/her provider - one that initiates conversations and open, “social” communication.

Hospitals have reacted to the social revolution by embracing social media to relate to the public. The number of hospital social media sites has increased exponentially in the last few years. From January 2011 to now, in 2014, the total number of hospital sites has increased from in 3,087 to 6,528.

For the parents of patients in the Neonatal Intensive Care Unit (NICU), their hospital stay can be a stressful and overwhelming experience. Studies have shown that parents of NICU patients experience high levels of anxiety, tension and are at increased risk for depression. Studies confirm that mothers of infants born at a gestational age at less than 32 weeks are at increased risk of postpartum depression, and that increased perception of support from nursing staff resulted in a decreased likelihood of depressive symptoms.

These symptoms are manifested in signs and symptoms, such as sleeplessness, stress intolerance, poor work performance, increased use of legitimate and illegitimate tranquilizing drugs, and marital problems. Mood disorders such as post-partum depression, major depressive disorder and post-traumatic stress syndrome are diagnosed in parents with infants who are admitted to the NICU.

Online communities with members who share a common underlying problem, like NICU families, have been shown to be helpful in a number of ways. Sharing stories and similar experiences, finding relevant information, and providing mutual emotional support from peers provide relief from stress. This also provides comfort and a deeper understanding of the parallel circumstances.

At the Children’s Hospital at OU Medical Center efforts have made to address this specific need by creating an online social networking community for the families and loved ones of our babies currently in or graduated from the NICU. This community was named “Tiniest Sooners” to honor the smallest and most vulnerable babies (Figure 1). “Tiniest Sooners” is linked across a number of social media sites and provides educational web resources and links, NICU announcements and information for our families, as well as a private group discussion forum, silently supervised by a neonatal-perinatal medicine physician.

We have an ongoing study evaluating the impact of this approach.

The American Academy of Pediatrics has also embraced this social mode of communication between and among healthcare professionals and patient families by creating a Social Media and Communications committee within the Perinatal Section that stems from its new perinatal website.  [www2.aap.org/sections/perinatal/index.html](http://www2.aap.org/sections/perinatal/index.html).

Healthcare professionals, in general, have not universally been as quick to embrace social media as the patient community. What is this hesitation, even fear, of exploiting all the readily available social media tools? Social Media Anxiety Disorder has been coined specifically for this situation. Virtually self-explanatory, the condition stems from the misconception that all “social media” equals Facebook and Twitter. The plethora of useful applications such as social bookmarking, and slide and video sharing, may be unfamiliar and misunderstood by this anxious crowd. Infamous news stories, such as “Weiner Gate,” do not help the issue and propagate...
rumors of unprofessional conduct showcased to the world on the platform of Facebook and Twitter. Those with this disorder may be unaware of the option to keep private accounts, and send protected messages.

Why should we bother then, especially with all of these professional privacy and liability concerns, to even learn about these social modes of communication? Life is busy enough as it is, one may argue. However, one could also argue that life may get unnecessarily busier if we don’t evolve with it. We may miss the typewriter, but we cannot deny the efficiency that computers have allowed. So I maintain that we should bother to learn to use web tools confidently to streamline our lives, not be overburdened by them; to communicate effectively by sharing and learning from one another; and to balance the user-generated content with more “expert user”-generated content.

I envision a day when the healthcare community at large will globally embrace communication media and technology to circulate truth and understanding for the health and happiness of all babies and families.

References


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Clinical Opinion by Women & Infants Maternal-Fetal Medicine Specialists Published in American Journal of Obstetrics & Gynecology

When a fetal heartbeat pattern becomes irregular during labor, many practitioners give oxygen to the mother. But questions remain whether this oxygen supplementation benefits the fetus or may actually be potentially harmful.

A clinical opinion written by third year resident Maureen Hamel, MD, along with maternal-fetal medicine specialists Brenna Anderson, MD and Dwight Rouse, MD, of the Department of Obstetrics and Gynecology at Women & Infants Hospital of Rhode Island and The Warren Alpert Medical School of Brown University, was published in the January 10, 2014 online edition of the American Journal of Obstetrics & Gynecology.

The manuscript, entitled "Oxygen for intrauterine resuscitation: Of unproved benefit and potentially harmful," aimed to make recommendations about the safety of the use of maternal oxygen supplementation in laboring women.

According to lead author Dr. Hamel, "Maternal oxygen is often given to laboring women to improve fetal metabolic status or in an attempt to alleviate non-reassuring fetal heart rate patterns. However, there are only two randomized trials investigating the use of maternal oxygen supplementation in laboring women. These studies did not find that supplementation is likely to benefit the fetus, and may even be harmful."

Based on their research, the team concluded that until it is studied properly in a randomized clinical trial, maternal oxygen supplementation in labor should be reserved for maternal hypoxia (lack of oxygen), and should not be considered an indicated intervention for non-reassuring fetal status.

Study Finds Potential Solution for Feeding/Swallowing Difficulties in Children with DiGeorge Syndrome, Autism

Newswise - Collaborative research out of the George Washington University (GW) reveals new information on the pathogenesis of feeding and swallowing difficulties often found in children with neurodevelopmental disorders, including autism and intellectual disability. Using an animal model of DiGeorge/22q11 Deletion Syndrome, a genetic disorder that causes autism and intellectual disability, the GW group found clear signs of early feeding and swallowing disruption, and underlying changes in brain development. The research, featured on the cover of Disease Models & Mechanisms, may even lead to a cure for these difficulties — known as pediatric dysphagia.

"We found that the same mechanisms causing neurodevelopmental disorders are disrupting development in parts of the nervous system that control swallowing and feeding," said Anthony-Samuel LaMantia, PhD, Professor of Pharmacology and Physiology at the GW School of Medicine and Health Sciences (SMHS) and Director of the GW Institute for Neuroscience. "Cranial nerves, which control food intake and swallowing, aren’t developing correctly, which likely contributes to miscoordination. This is good news — this is something we can fix."

Up to 80% of children with developmental disorders have difficulty ingesting, chewing, or swallowing food, leading to food aspiration, choking, or life-threatening respiratory infections. Despite its high co-occurrence with developmental disorders, little was previously known about pediatric dysphagia.

"A lot of children with pediatric dysphagia tend to be sicker from birth onward. Making the health of these kids as stable as possible from birth onward would allow clinicians to pick up on developmental signs sooner, which are often masked by more immediate problems like having ear or respiratory infections, not sleeping or not gaining weight," said LaMantia. "The physiological stress caused by the complications of dysphagia early on likely exacerbates the fundamental behavior issues that will emerge later. A happy, healthy baby is often able to focus on observing and gathering information to drive important experience-dependent changes in the brain. A sick baby has less time to do so, possibly making cognitive outcomes even worse."

These findings were a collaborative effort between LaMantia, and Sally Moody, PhD, Professor of Anatomy and Regenerative Biology at SMHS, with important contributions from Beverly Karpinski, a research scientist who works jointly with LaMantia and Moody; Thomas Maynard, PhD, Associate Research Professor of Pharmacology and Physiology at SMHS and Director of the GW Institute for Neuroscience Biomarkers Core; and Irene Zohn, PhD, Associate Professor of Pediatrics and Pharmacology and Physiology and Investigator in the Center for Neuroscience Research at Children’s National Medical Center.

LaMantia’s lab had been working on issues surrounding disrupted development from DiGeorge/22q11Deletion Syndrome and Moody’s lab had, over the course of her career, been working on issues specific to cranial nerve neurons and how they relate to the development of peripheral nerves and cranial facial targets. The combined expertise led to this discovery and will lead to future collaborations.

The study, titled "Dysphagia and Disrupted Cranial Nerve Development in a Mouse Model of DiGeorge (22q11) Deletion Syndrome," is available at http://dmm.biologists.org/content/7/2/245.abstract
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ESPGHAN = European Society of Paediatric Gastroenterology, Hepatology and Nutrition; HMF = human milk fortifier