

SCANNNews

Volume 13, Issue 2

Summer/Fall 2005

Calendar

- SCANN Meeting
Sharp Mary Birch
Hosp
Sept 12 @ 6-8 pm
- *Neonatal Nurses Day*
September 15
- **NANN 21st Annual Conference**
Anaheim, CA
Sept 28– Oct 1
- **SCANN's 11th Annual Conference**
Hilton Torrey Pines
La Jolla
November 7th
- *Happy Thanksgiving*
November 24th

SCANN Officers

President

Leilani Marino RN AS
*Sharp Mary Birch
Hospital for Women*

Secretary

Lourdes Ibadlit RN BSN
Palomar Medical Center

Treasurer

LCDR Stacia Fridley MS
CCRN CNS
*Naval Medical Center
San Diego*

Rise in Infant Mortality Linked to Babies Born at Lower Birth Weights

Excerpts from *Perinatal Care Matters*, a Publication of the Regional Perinatal Programs of California, Spring 2005.

After decades of annual improvement in infant mortality (infants dying in the first year of life), an increase in infant mortality, from 6.8 infant deaths per 1,000 live births in 2001 to 7.0 in 2002, was the first increase in the rate since 1958 according to the CDC. The increase in the birth of very small infants is the major reason behind the increase in U.S. infant mortality in 2002. Multiple births may also contribute to the increase in LBW infants. About 3% of births in the U.S. were multiple births, yet they made up about 25% of the overall increase in infant mortality.

Every Day in the United States...

- 11,121 babies are born;
- 214 of these babies are born weighing less than 1500 grams (VLBW) and have 105 times the risk of dying in their first year of life as a child born of average weight;
- An additional 837 babies are born weighing between 1500 and 2500 grams (LBW). They are 25 times more likely to die in the first year of life as a child born of average weight;
- Nearly 60% of all premature babies are born at 35 or 36 weeks gestation. These babies may be of normal

weight but they have a significant risk of becoming sick and requiring neonatal intensive care;

- 1,101 babies are born weighing more than 4 kg (macrosomia). In fact approximately 15% of these babies will weigh more than 11 lbs, doubling their risk of infant mortality;
- 1 in 8 of these babies will have been born in California!

Source: NCHS, Preliminary Natality Data, 2003



A Note from Leilani...

In preparing for our upcoming conference, I'd like to encourage all members to consider taking on small tasks as part of our Volunteer Committee or step into an Executive Position. The role of President, President-Elect and Secretary are open for 2006. Our organization continues to

grow with the spirit of our long term members and the new ones who've joined this year.

This has been a year of growing for our organization, officers and volunteers. Please help support our Southern California Association of Neonatal

Nurses through membership, volunteering, community service or supporting our upcoming annual conference. You have made a difference!

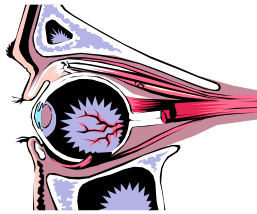
With deepest gratitude,

Leilani Marino

Retinopathy of Prematurity (ROP)

Children's Hospital and Health Center hosted SCANN's May meeting. Brian Lane MD, Neonatologist, presented Retinopathy of Prematurity and Hypoxic Ischemic Encephalopathy.

ROP, then called Retrolental Fibroplasia (RLF), was first described in 1942. The incidence of RLF or ROP peaked in the early 1950's with the introduction of incubators and the liberal use of oxygen for the care of premature neonates. Increasing rates of ROP are returning with increased survival rates of VLBW infants. 80% of infants less than 28 weeks gestation, and < 1000 grams, will develop ROP.



The etiology of ROP remains controversial, though oxygen and free radical formation play a major role. Risk factors include: prematurity, LBW, hyperoxia, RDS, duration of ventilation, sepsis, IVH, and transfusions(?).

The retinal vasculature begins to form around the 15th to 18th week of gestation. The retinal vessels grow out from the optic disc (centered in Zone I) to the periphery of the retina. These blood vessels reach the nasal ora serrata (Zone II) by about 35 weeks gestation and the temporal ora serrata (Zone III) by term. The retinal vessels remain vulnerable until growth into Zone III.

ROP is classified by location (Zone I, II, III), degree of abnormal vascular response (Stage 1-5), extent (number of clock hours involved), and the presence or absence of "plus" disease.

Stage 1 ROP: a fine line of demarcation is seen between the vascular and avascular region of the retina.

Stage 2: a thick ridge separates the vascular from the avascular retina.

Stage 3: extra retinal fibrovascular proliferation with vessels spreading into the vitreous.

Stage 4: progression to partial retinal detachment.

Stage 5: complete retinal detachment.

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Community Service...March of Dimes WalkAmerica®

This was SCANN's 14th year supporting the March of Dimes through participation in WalkAmerica®. This year SCANN members and friends raised \$2,665. This was our best year yet! SCANN also donated \$0.10 for each dollar raised.

SCANN walkers were Barbara Brockett, Karen Dougherty, Dian Doyle, Lourdes Ibadlit (aka Booboo), Ellen Milan, with friends Ellie Conti-Parker (UCSD) and Dr David Golembeski (Palomar Medical Center).

Did you see our photo in NANN Central?

SCANN & the March of Dimes
saving babies, together.

SAVE THE DATE!

WalkAmerica® at Balboa Park
April 29, 2006

**SCANN members
raised \$2,665
for the
March of Dimes!**

From the Editor...

I am giving up the position of editor of SCANNews at the end of the year.

I put out my first newsletter for SCANN in November 1994 with minimal computer experience. Actually, I didn't even own a computer back then and I never learned to type. I've become quite fast at the "hunt and peck" and have ventured into Microsoft Publisher to do the newsletter. I put out SCANNews from 11/94 through 12/98 and again since winter 2002.

It's time for a change, for me and for the newsletter.

If you're interested in taking on our newsletter, feel free to contact me or SCANN President Leilani Marino.

Ellen Milan
ellenonvacation@earthlink.net

858-459-6167



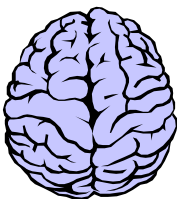
How would you like
to be editor?

Hypoxic-Ischemic Encephalopathy (HIE)

Hypoxic-ischemic brain injury that occurs during the perinatal period is a prominent cause of neonatal mortality and long term neurologic morbidity (cerebral palsy). It is seen in about 1-4 per 1000 live births.

Hypoxia refers to an abnormal reduction in oxygen delivery to the tissues. Ischemia refers to a reduction in blood flow to the tissues. Asphyxia refers to progressive hypoxia, hypercarbia and acidosis.

The fetus adapts to an asphyxial event (most likely occurring as a consequence of interruption of placental blood flow) to preserve cerebral blood flow and oxygen delivery. Initially the fetus compensates by increasing cardiac output and then by selectively increasing blood flow to the



brain, heart and adrenals with decrease blood flow to the kidneys, intestines and skin. Ongoing asphyxia will result in decrease cardiac output and decrease cerebral blood flow.

The watershed areas of the brain (at the borders of major cerebral arteries) are most vulnerable to injury. In the premature infant, they are the periventricular area, basal ganglia, brain stem, cerebellum and spinal cord. In the term infant, they're the parasagittal cortex, cerebral neocortex, thalamus, hippocampus, and the subcortical white matter.

Events leading to cell death include insufficient anaerobic metabolism, depletion of adenosine triphosphate (ATP) and phosphocreatinine, and increase lactate pro-

duction. ATP is a critical regulator of cell function; one major function is to preserve ionic gradients across plasma and intracellular membranes (Na⁺, K⁺ and Ca⁺⁺). Rising intracellular Ca promotes free radical production, cell membrane damage and cell death.

Diagnosis of HIE includes a history consistent with hypoxia, clinical evidence of encephalopathy, brain imaging, EEG, and evidence of multi-system involvement (PPHN, pulmonary edema, pulmonary hemorrhage; arrhythmias, shock, heart failure; renal failure, SIADH; NEC; DIC, polycythemia, thrombocytopenia; hypoglycemia, hypocalcemia).

Saranat's Clinical Stages of HIE (Arch of Neurol 33: 696, 1976) is used to distinguish between mild, moderate and severe encephalopathy.

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ROP continued...

"Plus" disease is defined as dilation and tortuosity of the peripheral retinal vessels and is evidence of more severe disease.

Pre-threshold disease is any stage in Zone I, Stage 2 Zone II with "plus" disease or Stage 3, Zone II. Threshold disease is defined as the presence of Stage 3 disease in Zone I or II, of at least 5 contiguous clock hours or 8 total clock hours, with the presence of "plus" disease. These infants require treatment within 72 hours.

ROP treatment include cryotherapy, laser surgery (as effective and easier

to perform) and scleral buckling to prevent retinal detachment.

ROP complications include retinal detachment, vitreous hemorrhage, glaucoma, amblyopia, strabismus, myopia, and loss of vision.

How can we prevent ROP? PREVENT PREMATURITY!!! Also, minimize FiO₂ (institute protocols for oxygen delivery and monitoring SpO₂); vitamin E (may reduce severity, not incidence); D-penicillamine (also an anti-oxidant, may reduce the incidence of acute ROP).

Community Service... Grocery Gift Cards

Grocery gift cards, are an easy way to help others while we're doing our own shopping. Pick up a \$5 or \$10 grocery card when you're doing your family shopping. Bring the cards to each SCANN meeting and at the end of the year we'll donate them to families in need. Please help us choose where these gift cards will go.

Bring your Grocery Gift cards to our next meeting.

SCANN's
11th Annual Conference
Changing Tides in
Neonatal Care
Monday
November 7th, 2005
Hilton La Jolla
Torrey Pines



Don't Forget to Pick
up a
Grocery Card

Stage 1 (mild) HIE: the infant is hyper alert with normal to increased muscle tone, mild distal flexion, a weak suck, a strong moro reflex, no seizures and has dilated pupils. Stage 2 (moderate) HIE: the infant is lethargic or obtunded with mild hypotonia, strong distal flexion, a weak or absent suck, a weak moro reflex, may have focal or multifocal seizures and has constricted pupils. Stage 3 (severe) HIE: the infant is stuporous and flaccid with intermittent decerebration, the suck, moro and tonic neck reflexes are absent, seizures are uncommon, but severe if present, the pupils are unequal and unreactive.

A head ultrasound may show edema and increased echogenicity initially and cysts later. A CT Scan may show evidence of edema and infarction. MRI spectroscopy can show evidence of hypoxia within hours to days of injury.

Management of HIE includes maintaining adequate ventilation (avoiding hypocarbia), maintaining perfusion, monitoring for evidence of renal damage and managing fluids and electrolytes accordingly, avoiding hypoglycemia and hyperglycemia (both may exacerbate neuronal injury).

Mortality for infant's with HIE is 15-50%. The majority of survivors (80%) of severe HIE have severe disability; up to 10% may be normal. Thirty to fifty percent of survivors of moderate HIE will have severe disability; 10-20%, mild to moderate disability.

Hypothermia is being used as a neuroprotective therapy. Hypothermia decreases metabolism and energy utilization, reduces oxygen consumption, preserves ATP levels, and suppresses free radical activity. Potential adverse effects of hypothermia include cardiac arrhythmias, bradycardia, decreased oxygen availability and consumption, persistent acidosis, platelet dysfunction, increased blood viscosity, and mild hypokalemia.

Body cooling has been shown to decrease the risk of death or moderate/severe disability. Body cooling is currently being used in UCSD Medical Center's Infant Special Care Center for infant's with HIE. Criteria for body cooling include: history of acute perinatal event (abruption, cord prolapse, variable/late decelerations); Apgar score of ≤ 5 at 10 minutes; cord or blood gas at less than 1 hour of life with $\text{pH} \leq 7.0$; base deficit on blood gas of ≤ -16 ; continued need for ventilation initiated at birth and continued for at least 10 minutes. Treatment must be initiated within 6 hours of life. Exclusion criteria include: inability to initiate therapy by 6 hours of life; chromosomal or major congenital anomalies; severe IUGR (< 1800 grams); prematurity?; planned withdrawal of care.

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