birth weight, birth asphyxia, and pneumonia. In 2005 asphyxia accounted for 20.5% of deaths in children under the age of 5 years. On the basis of a national sample survey from China Disabled Persons' Foundation in 2003, there are 199000 disabled children between 0 and 6 years old each year, 54.2% of whom are mentally disabled, primarily related to birth asphyxia.

The Neonatal Resuscitation Program (NRP) was introduced to China in the 1990s to reduce mortality and morbidity caused by asphyxia. NRP training was held in cities such as Beijing and Shanghai, which helped to build the foundation of the NRP in China. To disseminate the NRP throughout China, a multidisciplinary partnership was established among the Chinese Ministry of Health, Chinese Perinatal Society, Chinese Nursing Association, American Academy of Pediatrics, and Johnson and Johnson Pediatric Institute. In July 2003, a task force that consists of representatives from all partners made a 5-year commitment to set up "Freedom of Breath, Foundation of Life: China Neonatal Resuscitation Program." The objective was to ensure the presence of at least 1 trained health care professional at every delivery. It has been 3 years since the program launched, and many key accomplishments have been made:

1. The Chinese version of the fourth and fifth editions of the NRP manual (created by American Academy of Pediatrics and American Heart Association) was published.
2. Chinese NRP editions were made with Chinese cultural considerations to guide neonatal resuscitation practices in China.
3. Since July 2004, many training sessions have been organized, including a national instructors training, provincial instructors training(s) in 30 provinces (there are a total of 30 provinces in China), and cascading trainings in cities, counties, and townships. To date, 18 240 health care professionals have been trained, among them obstetricians, pediatricians, nurses/midwives, and anesthetists. By the end of 2006, NRP training had covered 99.1% of health care institutions in cities and 59.8% in 20 target provinces.
4. On September 20–23, 2006, the NRP Science Updates and Experience Sharing conference was held in Xian, capital city of Shaanxi in the northwest part of China. One hundred fifty health care professionals from 20 target provinces attended to learn of scientific updates from Drs Keenan and Niemeyer. Each province presented their training reports and summaries; a few of them were rewarded for their excellence of performance.
5. Since the launch of the NRP, many provincial health bureaus included neonatal resuscitation skills into midwifery service licensing. By the end of 2007, it will become a nationwide regulation in midwifery service licensing. Starting in 2007, the Chinese NRP expanded its program elements to add neonatal mortality and morbidity evaluation. We believe the data collected from this evaluation would be valuable, not only to the Chinese NRP but also to the international NRP.

ELECTRON MICROSCOPIC ANALYSIS OF BACTERIAL BIOFILM ON TRACHEAL TUBES REMOVED FROM INTUBATED NEONATES AND THE RELATIONSHIP BETWEEN BACTERIAL BIOFILM AND LOWER RESPIRATORY INFECTIONS

Submitted by Jialin Yu
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INTRODUCTION: Recurrent neonatal lower respiratory infections caused by endotracheal tubes (ETTs) may be related to the bacterial biofilm on them.
OBJECTIVE: We aimed to investigate the microbial biofilm on the surface of ETTs removed from neonates with intubated ventilation to explore the relationship between ETT biofilm and the lower respiratory infections.
METHODS: Twenty ETTs used in intubated neonates were examined for the presence of biofilm on their surface by scanning electron microscopy, and bacteria harvested from the surface of ETTs and the secretions of lower respiratory tracts were isolated, identified, and assessed for antimicrobial susceptibility.
RESULTS: Scanning electron microscopy showed that the incidence of microbial colonization was 60% (12 of 20) when the use of tubes exceeded 2 days, biofilm formation was observed ~3 days after intubation, and its architecture became more mature and complex when the duration exceeded 3 days. There were 14 positive cultures from ETTs (70%, including 4 normal flora), in which 7 kinds of pathogens were isolated; in 13 cultures from the secretions of lower respiratory tract (65%, including 1 normal flora), 10 kinds of pathogens were isolated. Seven samples had the same pathogen both on the surface of ETTs and in the secretions of the lower respiratory tract, which accounted for 50 of the positive cultures from ETTs. The Gram-negative bacteria isolated from the surface of ETTs and the secretions of lower respiratory tract presented multiresistance to antibiotics.
CONCLUSIONS: The ETT biofilm develops into a mature and complex form on the basis of the duration of tube use. There is a possible positive correlation between them. There is correlation between microbial biofilm formation on the surface of ETTs and lower respiratory tract infection in intubated neonates who are ventilated

for a prolonged period of time. ETT biofilm could be a likely source of recurrent infection.

Nephrology

USING NONSTEROIDAL ANTIINFLAMMATORY DRUGS IN VOLUME-DEPLETED CHILDREN CAN PRECIPITATE ACUTE RENAL FAILURE

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INTRODUCTION: Nonsteroidal antiinflammatory drugs (NSAIDs) are ever increasing in popularity in hospital medicine and general practice and are readily available over-the-counter.
OBJECTIVE: Our goal was to illustrate the need to be aware of the effect of NSAIDs on dehydrated patients.
PATHOGENESIS: The risk of renal toxicity is increased in situations in which there is a stimulation of the renin-angiotensin system such as volume depletion. In these conditions, circulating vasoconstrictors are released, maintaining vascular resistance and blood pressure at the potential expense of regional organ blood flow. To maintain renal blood flow, counter-regulatory renal prostaglandins are released that counteract vasoconstrictors and normalize renal blood flow. NSAIDs blunt this counter-regulatory response and intensify the renal vasoconstriction, which leads to acute renal failure. In Table 1 we report 4 children with mild dehydration who developed acute renal failure after the use of therapeutic doses of NSAIDs in a children's hospital.

TABLE 1. Acute Renal Failure in 4 Children After Use of NSAIDs

	Patient No.			
	1	2	3	4
Age, y	13	7	14	13
Gender	Male	Male	Male	Female
Underlying pathology	Craniopharyngioma diabetes insipidus	Juvenile idiopathic arthritis; fasted for surgery	Juvenile idiopathic arthritis with vomiting	Relapse of Crohn disease
NSAID	Diclofenac sodium	Indomethacin diclofenac sodium	Diclofenac sodium	Diclofenac sodium
Highest urea level, mmol urea/L	10.7	12.9	10.7	22
Highest creatinine level, μmol/L	226	146	376	629
Normalization, d	5	3	3	Permanent impairment

CONCLUSIONS: We recommend that NSAIDs should be avoided in children with actual or potential intravascular volume depletion. Although we have not proven cause and effect, additional research is needed to define the true risk of the potential renal complications of using NSAIDs in patients who are at risk of dehydration.

NOTE: The cases of the 4 children described in this report have been published elsewhere (John CM, Shukla R, Jones CA. Using NSAID in volume depleted children can precipitate acute renal failure. *Arch Dis Child.* 2007;92:524–526).

ROLES OF SCAP (STEROL REGULATORY ELEMENT–BINDING PROTEIN CLEAVAGE-ACTIVATING PROTEIN) IN THE MECHANISM FOR MESANGIAL FOAM-CELL FORMATION UNDER INFLAMMATORY STRESS

Submitted by Qiu Li
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INTRODUCTION: Our previous studies have demonstrated that lipid abnormalities play a significant role in glomerulosclerosis. Inflammatory cytokines promote lipid accumulation in human mesangial cells (HMCLs) by disrupting low-density lipoprotein receptor (LDLr) feedback regulation. The sterol regulatory element–binding protein (SREBP) cleavage-activating protein (SCAP) carries SREBP from endoplasmic reticulum (ER) to Golgi, where it is known to cleave SREBP, thereby enhancing LDLr gene expression and cholesterol uptake when cells need cholesterol.
OBJECTIVE: We aimed to investigate whether inflammatory mediators interfere with SCAP translocation and its biological consequence.
METHODS: HMCLs were used in all experiments. Total cellular RNA was isolated from these cells for detecting LDLr, SREBP-2, and SCAP messenger RNA levels with real-time quantitative polymerase chain reaction. LDLr protein expression was measured by Western blot. Translocation of the SCAP-SREBP complex from the ER to Golgi was investigated by confocal microscopy.
RESULTS: In the absence of exposure to interleukin 1β, a high concentration of LDL retained SCAP in the ER, a low LDLr promoter activity, messenger RNA synthesis, and protein expression were found, respectively. However, exposure to interleukin 1β caused overexpression of SCAP and enhanced its translocation from the ER to Golgi. This disrupted normal feedback regulation and resulted in inappropriately increased LDL uptake with transformation of HMCLs into foam cells. Overexpression of SCAP in HMCLs resulted in an increased translocation of SCAP from the ER to Golgi, and high concentrations of LDL were unable to suppress SREBP-2 and LDLr gene expression.
CONCLUSIONS: These data suggest that inflammatory mediators promote abnormal translocation of SCAP from the ER to Golgi and play an important role in lipid accumulation in HMCLs.

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