Paediatric Focus

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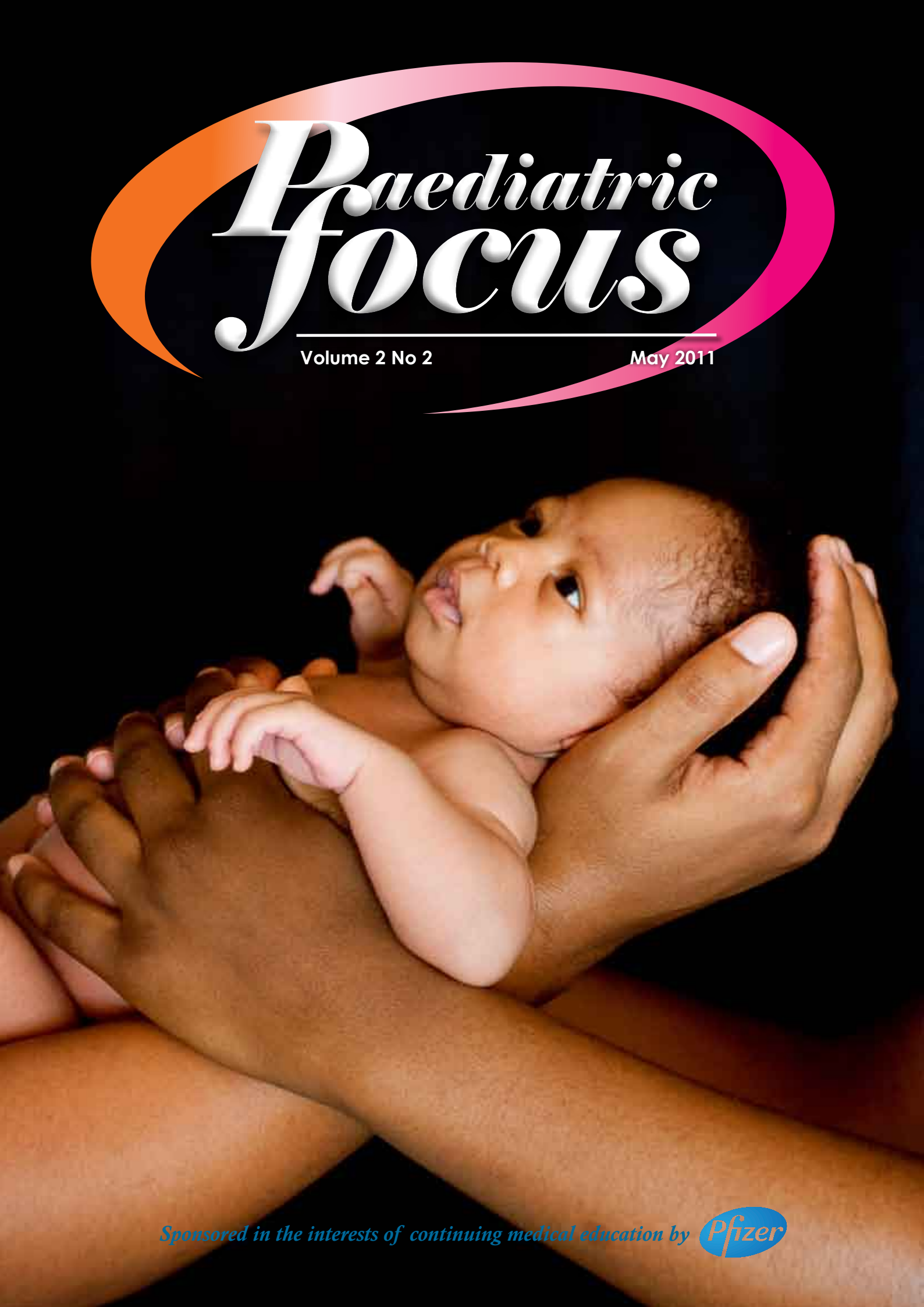

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Editorial

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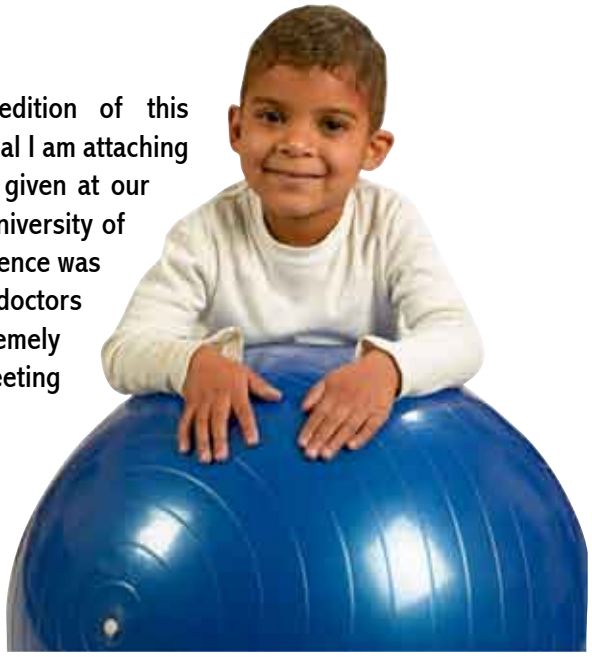
Editor: Professor R J Green
Production Editors: Ann Lake, Helen Gonçalves
Design: Jane Gouveia
Queries: Ann Lake Publications 011 802 8847
Website: www.annlakepublications.co.za
Email: lakeann@mweb.co.za

Editor
Professor Robin Green
Professor and Head
Department of Paediatrics
and Child Health
University of Pretoria

Welcome to our fourth edition of this newsletter. By way of editorial I am attaching a summary of the lectures given at our recent Paediatric Update (University of Pretoria). This 2-day conference was attended by nearly 150 doctors and was rated as an extremely valuable and high quality meeting by attendees.

Next year we hope to see you there!



2011 Congresses

Congress	Location	Date	Contact/URL
ASEAN Paediatric Congress 2011 (APC 2011)	Suntec Singapore International Convention Centre, Singapore	14 - 17 April	www.apc2011.com.sg/
ECCMID: European Society of Clinical Microbiology and Infectious Diseases	Milan Italy	7-10 May	www.eccmid-icc2011.org/
European Society for Paediatric Gastroenterology, Hepatology and Nutrition 44th Annual Meeting 2011 (ESPGHAN 2011)	Hilton Sorrento Palace Hotel, Via, Italy	25 - 28 May	www.espghan2011.org/home.aspx
ESPID: European Society for Paediatric Infectious Diseases	The Hague World Forum, The Hague, The Netherlands	7-11 June	www2.kenes.com/espid2011/Pages/Home.aspx
Paediatric Prevenar 13 Forum	Hyatt, Oubaai, George	17-19 June	Tel: Leanne Biela 082 600 8402 Email: Leanne.Biela@pfizer.com
5th Europaediatrics Congress 2011	Austria Centre, Vienna, Austria	23 - 26 June	www.europaediatrics2011.org/
ICAAC: Interscience Conference on Antimicrobial Agents and Chemotherapy	Chicago, Illinois, USA	17-20 September	www.icaac.org/
European Respiratory Society Annual Congress 2011	Amsterdam, Netherlands	24 - 28 September	www.erscongress2011.org/
51st Annual Meeting of the European Society for Paediatric Research 2011 (ESPR 2011)	The Sage, Gateshead, Newcastle, United Kingdom	14 - 17 October	www2.kenes.com/espr2011/Pages/Home.aspx
WSPID: World Society for Paediatric Infectious Diseases	Melbourne Convention Exhibition Centre, Melbourne, Australia	16-19 November	www.wspid.com/home.asp

CPD Accreditation

Doctors can acquire CPD points with this newsletter by visiting www.paediatrician.co.za, www.mycpd.co.za, www.pandasa.co.za or www.criticalcare.org.za and completing an online form of 15 questions. Doctors whose articles are published in this newsletter will also automatically be awarded CPD points. Accreditation is available only for a limited time on the site. Should you have any queries regarding the accreditation, please contact E2 Solutions at: 011 888 9620 or 011 340 9100 or gertrude@e2.co.za

UPDATE 2011
Summary of sessions

Professor Robin Green
Professor and Head Department of Paediatrics and Child Health,
University of Pretoria

Breastfeeding
Dr Nancy Wight

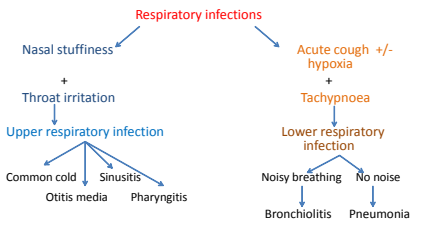
- Survival of 500 gram neonates is now 50% in the USA.
- Until recently focus in improving survival has been on the lungs.
- Feeding is now considered critical in further improving survival.
- Cows milk protein (even 1 dose) may change GUT flora for months or years.
- Teach mothers to hand express (into a spoon) colostrum from birth.
- ***NEC has been reduced in some studies up to 80% with human donor breast milk programmes.***
- Kangaroo care augments human milk production.

Evidence-based Neonatology in the first hour of life
Prof Gert Kirtsen

- Antenatal steroids have a significant benefit in improving lung maturity.
- Delayed cord clamping (about 1 minute); 25% of the blood volume is in the placenta; baby placed between moms thighs; associated with fewer transfusions for anaemia (RR 2.01); associated with fewer infants with low BP (RR 2.58).
- Prevent hypothermia – plastic wrapping and heated labour ward.
- Newborns who do not breathe need gentle assistance; emphasis on stabilisation rather than resuscitation; elective intubation is better than emergency intubation.
- Resuscitate with room air unless heart rate <100 bpm.
- CPAP may reduce BPD (despite lack of evidence for other modalities).
- ***HFOV is equivalent to conventional ventilation for most neonates (remember the neurological implications of HFOV).***
- Alarm limits (88-92%) reduce ROP.

Antibiotics for Respiratory Tract Infections in infants
Dr Carla Els

Differentiation of upper and lower respiratory infections



Therapeutic Hypothermia
Dr Alan Horn

- HIE pathophysiology: decrease in ATP (energy failure) leads to neuronal release of sodium; leads to calcium influx and glutamate release; there is free radical generation with oxygenation.
- Head cooling diminishes death and HIE but not late disability.
- Head cooling has a poor outcome in: LBW; weight > 3kg; pyrexia >38 degrees C; seizures.
- When cooling sedate with phenobarb and monitor core temperature (probe on back or rectal).
- Whole body cooling has revealed no decrease in death but increase in survival without neurological abnormality (very significant).
- Contra-indications to cooling:
  - Major congenital abnormalities (especially those requiring surgery)
  - Birth weight < 1800g
  - Moribund
  - Head trauma (intracranial bleeds)
  - Microcephaly
  - Relative: sepsis; severe RDS; refractory hypotension; refractory acidosis.
- ILCOR Consensus 2010 methodology:
  - Induce hypothermia (33.5° – 34.5° C)
  - Within 6 hours
  - For 72 hours
  - Rewarm over 4 hours at 0.2 degrees per hour
  - Use in term or near term neonates with high risk for brain injury
  - Babies must be in NICU and monitored for adverse events.
- ***The NNT for improved survival with cooling is 9; so this is not a magic cure.***
- Systemic cooling may be equal to selective head cooling.

Litigation in Neonatal Medicine
Dr Liz Meyer

- Neonatal litigation claims now the highest claims in any category.
- Neonatlogists often involved in a 'no misadventure' scenario.
- Parents quote the following reasons for lawsuits:
  - What happened – need for information (70% of parents are not warned about long-term neuro-developmental problems)
  - Concerns for a medical cover up
  - Need for financial support
  - Dissatisfaction with communication
  - Prevent it happening to others.
- Ways to prevent litigation:
  - Good documentation – note absence of signs (especially in the neonate); clear legible notes; no flippant remarks; quality rather than quantity
  - Obtain an opinion of a colleague
  - 'Red flag' risk factors eg. DDH
  - ***Communication – avoid jargon; avoid blame; avoid embellishment; speak about developmental rather than congenital abnormalities***
  - Prescribe in mg/kg/time period
  - Ask about allergies at each contact
  - Make verbal instructions simple, clear and concise
  - Preferably use written instructions.

What the paediatrician should know and do about common paediatric renal problems
Prof P Thomson

- History and examination are the 2 most important pillars of a renal investigation.
- Urine dipstix is critical.
- Causes of enlarged kidneys in neonates:
  - Hydronephrosis
  - Cysts – unilateral = multicystic/dysplastic; bilateral most often ARPCKD
  - Tumour
  - Reno-vascular thrombosis – due to umbilical catheterisation.
  - Maternal ACE-inhibitor use is associated with kidney embryopathy.
  - Antenatal hydronephrosis common due to the high foetal kidney blood flow.
- Indications for investigation of antenatal hydronephrosis:
  - Bilateral
  - Distended bladder
  - Prune belly
  - Uterocoele
  - Unilateral in solitary kidney.
- Creatinine clearance = Ht x 40 / serum creatinine (umol/l)
- Tubular function measured by FeNa or B2-microglobulin/creatinine ratio in urine.
- ***Intracellular volumes may be decreased in nephritic syndrome and diuretics may cause depletion and thrombosis.***

UPDATE 2011
Summary of sessions

What the paediatrician should know about renal transplantation
Dr Errol Gottlich

- Transplantation in young children is difficult (< 5 years old) because of problems in anastomoses; fitting adult kidney into a child; end-stage CRF not usual.
- ***Significant increases in renal transplantations from related living donors are occurring.***
- Eligibility criteria:
  - End-stage kidney failure GFR < 15 ml/min/1.7m^2
  - 10kg
  - Adequate bladder or creation of ileal conduit
  - Nephrectomy if severe VUR
  - Patent vessels
  - Cross match negative vs donor
  - Good psycho-social circumstances
  - Pre-transplant education
  - Completed work-up
  - Transplant committee sign-off
  - DOH sign-off.

Anguish in learning
Dr Michael Lippert

- 5 D's of learning disorders:
  - Dyslexia
  - Dysorthographia
  - Dyscalculia
  - Dysgraphia of writing
  - Dysattentive disorder
- ***Learning disorders may be genetic.***
- Developmental dyscalculia
  - Definition: Deficiency in mastery of mathematics; Inability to acquire mathematics
  - Often familial
  - Prevalence: 6.5% (common in journalists and writers)
  - 17% have dyslexia; 26% have ADHD
  - 20-30% improve on methylphenidate
  - Diagnosis: 2 years/grades behind model; cannot do 2-digit additions and subtractions
  - Treatment: motivating hope

Strange and interesting lung diseases
Dr Debbie White

- Case 1: 12 year old boy with acute or chronic cough. Severe failure to thrive. Diagnosis: Alveolar proteinosis. Treatment: 12 litres of pink fluid lavaged from his lungs while on ECMO. Outcome: Happy, healthy child
- ***Case 2: 1 year old girl with episodic respiratory distress. Diagnosis: CPAM Type 2. Treatment: Left pneumonectomy. Outcome: Happy, healthy child***
- Case 3: 8 year old girl from Transkei with chronic cough. Diagnosis: Hydatid cystic lung disease. Treatment: Surgical removal and albendezole. Outcome: Happy, healthy child

New developments in Celiac Disease
Dr Alta Terblanche

- Celiac disease affects about 1% of the population.
- Prevalence is increasing.
- Single most important risk factor (20% risk) is having a first degree relative with CD (especially a sibling).
- ***Undiagnosed CD is not benign – increased mortality (usually due to malignancy).***
- Pathogenesis – interaction of immunological/genetic/environmental factors (Enterovirus/Hepatitis C).
- Gluten peptides trigger innate immune factors.
- CD may present with atypical symptoms:
  - Iron deficiency anaemia
  - Osteoporosis
  - Peripheral neuropathies
  - Short stature
  - Dermatitis herpetiformis
  - Idiopathic pulmonary haemosiderosis
- Diagnosis:
  - Child must be on a gluten containing diet (2 weeks)
  - Intestinal biopsy is the gold standard – always with histology
  - HLA testing – DQ2/DQ8 (sensitivity 96.2%, specificity 54%) – high negative predictive value
  - Antibodies: Endomysial IgA; Transglutamase-2 antibodies; Transaminase Type 2 IgA; not Anti-gliadin antibodies
- Treatment:
  - Gluten free diet – compliance difficult; always involve dietician
  - Gluten enzyme degradation is in development

Adolescent development
Dr Johan Erasmus

- Phases of adolescence:
  - Early: 11-14 years
  - Middle: 14-17 years
  - Late: 17-21 years
- Early developers in girls and late developers in boys do better in social/inter-personal relationships than other groups.
- Individual variation in development of one developmental domain to another is common.
- ***There is an important cultural context to adolescent progress.***
- Adolescent challenges facing Paediatricians:
  - Time (spend adequate time with adolescents)
  - Confidentiality and informed consent
  - SA Child Act (with legal inconsistencies)
  - Child's cognitive level – in younger adolescents a more concrete approach is needed
  - Supportive environment.

Autism
Dr Elsa Lubbe

- Is a neurodevelopmental disorder with a common set of variables
- Aetiology:
  - Genetic – Fragile X; Angelman Syndrome; Tuberous Sclerosis; Rett Syndrome
  - Metabolic – Phenylketonuria; mitochondrial diseases
  - Highly heritable: 70% concordance in twins; 2-8% in siblings; some candidate genes have been identified.
- Genetic and metabolic factors account for < 20% cases.
- Diagnosis:
  - Impaired social interaction
  - Impaired communication – delay/lack; abnormal; poor communication; no make-believe play
  - Restricted interests and repetitive behaviours.
- Pervasive developmental disorder spectrum:
  - Autistic
  - Asperger's Disorder
  - PDD
  - Childhood disintegrative disorder
  - Rett Syndrome.
- All of these now form the Autism Spectrum Disorder without Rett Syndrome.
- ***Medications:***
  - ***Resperidone for aggression, repetitive behaviours***
  - ***Methylphenidate for ADHD component.***

UPDATE 2011
Summary of sessions

Abdominal pain
Prof Dankwart Wittenberg

- Prevalence 10-15% of school aged children at any time.
- Has a serious influence on QoL.
- Abdominal pain is influenced by:
  - Gene-environmental interactions
  - Developmental visceral hyperalgesia
  - Serotonin
- Warning signs:
  - Persistent/localised pain
  - Pain waking from sleep
  - Dysphagia
  - Persistent vomiting
  - Nocturnal diarrhoea
  - Family history of bowel disease
  - Arthritis
  - Involuntary weight loss
  - Decrease in linear growth
  - Unexplained fever
- 2% of children with functional abdominal pain turn out to have an organic disorder with special investigations.
- Repeated tests reinforce parent's fears of an unknown organic disease.
- Negative tests do not reassure a child's parents and it becomes harder to introduce the concept of a non-organic disease.
- Functional abdominal pain is a positive diagnosis and not one of exclusion.
- Functional abdominal pain includes:
  - Functional dyspepsia
  - Irritable bowel syndrome
  - Functional abdominal pain syndrome
  - Abdominal migraine.
- ***Parental anxiety predicts abdominal pain in children.***
- Management:
  - Positive diagnosis
  - Education
  - Reassurance
  - Achieve trust
  - Agreements on goals (social function and activity vs pain control)
  - Address anxiety
  - Dietary modification (fibre)
  - Medication (laxatives, etc.) – No one approach works for all children.

Pertussis – Forgotten not gone
Prof Theuns Avenant

- Most cases occur in infants (especially those under 3 months of age in SA) and children over 10 years old.
- Estimated 16 million cases annually worldwide (WHO - 2006).
- Reasons proposed for the recent increase in cases:
  - Incomplete protection from the vaccine (children need two doses)
  - Waning of immunity. Protection from vaccines: 4 - 14 years whole cell; 5-6 years DTPa
  - Adults as source of infection
  - Strain polymorphism – not proven
  - Improvements in diagnosis and surveillance
  - Age-specific contact patterns.
- ***Diagnosis: PCR of nasopharyngeal aspirates – highly sensitive.***
- Treatment:
  - Azithromycin or clarithromycin – to reduce spread
  - Manage respiratory tract disease
  - Manage contacts – as per treatment.
- Vaccine strategies to prevent increase in disease:
  - Adult/adolescent vaccines
  - Cocoon strategies
  - Earlier infant/neonatal vaccine
  - Maternal vaccination during pregnancy.

Medicines, Society and the Law
Dr Humphrey Lewis

- Be careful of OTC medications.
- Be careful of hidden ingredients in some 'natural products'.
- Saline is important for blocked nostrils in infants and young children.
- Use paracetamol for fever.
- ***54% of drugs used in NICU/PICU are used off-label.***
- Many drugs have side-effects especially when used off-label.

Anxiety in children
Prof Izelle Smuts

- Fear is a bad feeling while in danger
- Anxiety is a state of feeling nervous or worried
- Normal development of anxiety influenced by:
  - Temperament
  - Life experiences
  - Parental responses
  - Parental psychopathology
  - Level of understanding
- Anxiety is pathological when it interferes with functioning – avoidance; distress; impairment.
- Life time prevalence: 15-20%
- Types of anxiety disorders:
  - Generalised anxiety disorder
  - Phobias - Social phobia – risk of depression; substance abuse; suicide
  - Separation anxiety disorder
  - School refusal
  - Panic disorder
  - Post traumatic stress disorder - aggression/temper tantrums
- ***Aetiology:***
  - ***Anxiety may be linked to IgA deficiency: increase in group A strep infection; increase in cross reacting antibodies; increase in dopamine release; increase in auto-antibody formation***
  - Increase in IL-2.
- Parental counselling:
  - 3 golden rules:
    1. be slow to criticise
    2. be quick to minimise guilt
    3. be astute to emphasize success
  - Essence: help parents to find their own solutions
  - Aim: enhance parental coping.

The Child with Antiretroviral Treatment Failure
Dr Leon Levin

- Reasons for ARV failure:
  - Poor adherence – most common
  - Viral resistance
  - Diminished efficacy of ARV's.
- When to change ARV's:
  - PI when 1000 copies/ml remain
  - NNRTI when 5000 copies/ml remain.
- Different scenarios for changing ARV's:
  - Early failure of first regime
  - Intermediate failure of first regime
  - Extensive prior treatment.
- ***Second line regime: At least 2, preferably 3 new drugs***
  - ***Beware cross resistance***
  - ***Resistance to previous mutations may not be revealed on genotyping.***

Neonatal ventilation: focus on non-injurious ventilation

Professor Johan Smith MMed, FC Paed (SA), PhD, Neonatologist
Division of Neonatology, Department of Paediatrics & Child Health
Stellenbosch University & Tygerberg Children's hospital, Tygerberg, Cape Town

Artificial ventilatory support of the newborn includes invasive and non-invasive technologies. Both approaches encompass a bewildering spectrum of modalities, terminology and strategies. There is however, no doubt that both invasive and non-invasive respiratory support saves neonatal lives. Halliday reviewed the evidence from clinical trials of various interventions aimed at treating and preventing respiratory failure in the neonate.[1] His analysis revealed that effective interventions included: conventional mechanical ventilation (CMV) (absolute reduction in mortality 12%, 95% CI 4-21%), continuous positive airway pressure (CPAP) (absolute reduction in mortality 15%, 95% CI 1-28%) and surfactant therapy (absolute reduction in mortality 4 to 9%, 95% CI 1-13%).

However, very low birth weight (VLBW) and particularly extremely LBW survivors of invasive ventilation or endotracheal tube-associated mechanical ventilation are at risk of the effects of biotrauma which includes chronic lung disease (CLD), alternatively, BPD, neurological impairment and sometimes multiple organ injury (apoptosis). Duration of ventilation in newborn infants is a significant risk factor for neurological impairment and the odds of impairment increase almost 2-fold per 4 weeks of ventilation and approximately 4-fold per 8 weeks of ventilation.[2] In another observational study of infants born before a gestation of 28 weeks, BPD accompanied by mechanical ventilation at 36 weeks post menstrual age was shown to be associated with a nearly 6-fold increased risk of quadriparesis and a 4-fold increased risk of diparesis.[3]

Strategies to improve lung outcomes

It appears to be to the advantage of a small newborn infant, at risk of respiratory distress, to have limited or no exposure to some invasive forms of assisted ventilation because accumulated evidence shows that assisted-breathing strategies influence the occurrence of both BPD as well as neurological and other impairment. Benefits of certain strategies, such as gentle, lung-protective resuscitation maneuvers in the delivery room followed by nasal continuous positive air way pressure (nCPAP) during transition and transport; early intubation with administration of surfactant and immediate extubation followed by nCPAP ("INSURE"), bi-level nCPAP or SiPAP or first intention early lung recruitment during HFO, are increasingly reported. Some technologies such as HFO lead to a reduction in deaths or BPD or severe neurological events if treatment was initiated between 1 and 4 hours after intubation.[4] This meta-analysis also highlighted additional benefits of HFO compared to conventional ventilation which included a decrease in patent ductus arteriosus requiring surgery and retinopathy stage 2 or more. Earlier, Gerstmann and co-workers reported better peak expiratory flow, distribution of ventilation and residual lung volume compared to conventional ventilation.[5] Likewise, Hofhuis and co-workers found poorer maximal flow at functional residual capacity during the first year of life in infants with BPD, but more so in those initially treated with conventional mechanical ventilation.[6]

Compared with endotracheal intubation and mechanical ventilation during the first 3 days of life, babies (≤30 weeks' gestational age) who received nCPAP or nasal intermittent positive pressure ventilation (NIPPV) were less likely to have BPD/death

These strategies probably result in improved lung outcomes achieved through preservation of surfactant activity, reducing the need for mechanical ventilation and / or avoiding air leaks and probably by lowering the occurrence of BPD. Explaining the 'additional' benefits such as lower rates of ROP stage 2 or more, is more difficult, but possibly relates to oxygen administration, recruitment strategies, weaning protocols and /or less overspill of biotrauma-initiating inflammatory mediators into the systemic circulation.

It is therefore not surprising that non-invasive and/or gentle ventilation and/or lung recruitment have become the buzz-words. Non-invasive breathing assistance includes various technologies that provide non-cycled respiratory support, either through nCPAP, synchronised nasal intermittent positive pressure ventilation (SNIPPV), humidified high-flow nasal cannula therapy, or through nasal HFO. These modalities may be used in conjunction with surfactant treatment.

Risks and strategies with 'new' equipment

The rapid appearance of 'new' equipment or the resurfacing of revamped respiratory support systems have led some to caution clinicians regarding the risks involved with these trends because of a lack of supportive scientific evidence. If we accept that a major goal is to avoid invasive mechanical ventilation, premature infants certainly appear to benefit from early CPAP with or without surfactant treatment. For instance, extremely premature infants (24 – 27 weeks' gestation) managed with early CPAP (initiated in delivery room) were more likely to be alive and free from the need for mechanical ventilation by day 7 of life compared to a group randomised to early intubation followed by surfactant instillation and ventilation.[7] In another study, premature infants, born between 27 and 31 weeks' gestation, who required oxygen in the delivery room were randomised to treatment with intubation and surfactant therapy (within 1 hour of birth), followed by extubation and CPAP or to nCPAP-alone. Although CPAP was started in both groups beyond the delivery room, the infants in the CPAP-surfactant-extubation group had a decreased need for mechanical ventilation.[8] These findings have now been confirmed by several studies.

In Europe and South Africa, bi-level nCPAP or SNIPPV is accomplished by using infant flow synchronised inspiratory positive airway pressure (SiPAP) (Infant Flow® SiPAP, Viasys Healthcare). In the USA the Infant Star ventilator (StarSync®) is used, but this ventilator is apparently being phased out. Lista and co-workers reported better respiratory outcomes in larger and more mature infants (28 – 34 weeks' gestation) who were randomised to bi-level nCPAP (SiPAP) versus NCPAP. Infants randomised to the bi-level nCPAP group spent less time on respiratory support and in supplemental oxygen and had a shorter hospital stay.[9]

In a retrospective study, Bhandari and co-workers compared outcomes of infants ≤1250 grams managed with SNIPPV and nCPAP. They found that infants in the weight category 500-750g, who were at greatest risk of BPD or death and who were treated with SNIPPV

(StarSync®), were significantly less likely to develop BPD, BPD/death, neurodevelopmental impairment (NDI) and NDI/death.[10]

Short bi-nasal prongs are more effective than single or long, cut-down endotracheal tubes in preventing reintubation.[11]

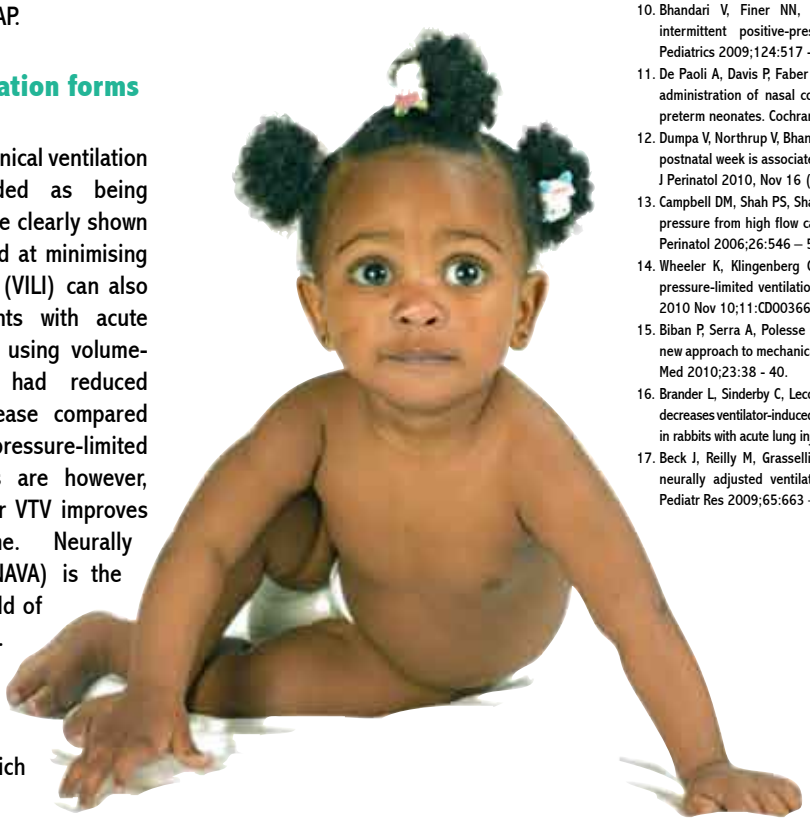
Compared with endotracheal intubation and mechanical ventilation during the first 3 days of life, babies (≤30 weeks' gestational age) who received nCPAP or nasal intermittent positive pressure ventilation (NIPPV) were less likely to have BPD/death. In addition, those extubated from ETT ventilation between day 4 and 7 of life to NIPPV were also more likely to have a decreased probability of BPD/death than those who remained on the ventilator.[12]

High flow nasal cannulae (HFNC) deliver gas flows > 2 L/min into the nostrils through small diameter prongs. This flow generates CPAP, however, the pressures generated are highly variable and are seldom, if ever, monitored in clinical practice. In one study in which infants were randomised at extubation to either infant flow driver CPAP or high flow nasal cannulae, the reintubation rate in the HFNC group was significantly higher, as was the incidence of apnoeas and bradycardias.[13] HFNC devices should be used with caution until more information is available and certainly not as a mode/strategy in place of nCPAP.

Some mechanical ventilation forms are beneficial

Not all forms of invasive mechanical ventilation should however, be regarded as being injurious. Studies in adults have clearly shown that ventilator strategies aimed at minimising ventilator-induced lung injury (VILI) can also decrease mortality in patients with acute lung injury. Infants ventilated using volume-targeted ventilation (VTV) had reduced death and chronic lung disease compared with infants ventilated using pressure-limited settings.[14] Long-term studies are however, required to determine whether VTV improves neurodevelopmental outcome. Neurally adjusted ventilatory assist (NAVA) is the newest development in the field of 'lung-protective' ventilation. This mode requires the introduction of a catheter to measure the electric activity of the diaphragm through which it monitors neural inspiratory activity of the diaphragm. The patient's respiratory drive determines the timing and magnitude of pressure delivered.[15] In experimental acute lung injury, NAVA was as effective as volume-controlled (6ml/kg), high positive end-expiratory pressure ventilation in attenuating VILI as well as biotrauma-related remote organ injury.[16] In a small study, Beck and co-workers showed that NAVA could successfully be used, both invasively and non-invasively, in very low birth weight preterm infants.[17] However, further studies are required to clarify the role of NAVA in the newborn infant.

In experimental acute lung injury, NAVA was as effective as volume-controlled (6ml/kg), high positive end-expiratory pressure ventilation in attenuating VILI as well as biotrauma-related remote organ injury



In summary, interesting evidence is accumulating that some breathing-assistance technologies could indeed be less injurious, not only to the newborn lung, but also to other organs, including the developing brain. It remains to be shown whether by reducing the incidence of chronic lung disease/BPD, the clinician would be able to improve motor outcome and cognitive performance of children.

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Neuroscience speaks to quality of neonatal outcomes

Dr N. Bergman MB ChB, DCH, MPH, MD
Consulting Public Health Physician, Pinelands, Honorary Research Associate, University of Cape Town.
www.kangaroomothercare.com

The last twenty years has seen an explosion of knowledge in neuroscience.[1] The bottom line is that the fetal and neonatal brain does not only have cardio-respiratory and metabolic needs, but is an active agent in its own neurodevelopment.[2] This is contrary to old assumptions on preterm care, which was based on a belief that the human brain was too immature at that age, and as long as the heart, lungs and stomach were working, then the brain would be fine.

The incubator was invented 100 years ago and "active management" of low birth weight infants started about 50 years ago.[3] This care assumed the incubator was the only possible PLACE such care could be given. The care was focused on improving survival, and we now have amazing survival rates, even at 24 weeks gestation.[4,5] However, these survivors have physical and psychological problems, the more so the lower the gestation.[6] In fact, we now know that even late preterm infants perform poorly when they start school,[7] and economically cost more to support[8] (there are more of them!). For the last twenty years these outcomes have not improved.[9,10] Without a proper understanding of the latest neuroscience, our care will continue its success with respect to **quantity of survival,** but without achieving any **quality of survival.**

The fetal brain development with respect to its anatomy is complete at 20 weeks, at 23 weeks the fetus is conscious and aware, and all its basic connections are complete at 28 weeks.[2] Development requires collecting sensory information about the world, this fires and wires pathways[11] that mould the brain to be suited or adjusted to that world (called adaptation).[12,13] The sensations in the uterus are pressure contact, movement, mother's sounds and smell, and all these provide a sense of safety and wellbeing. Good sensations provide a strong platform for higher level development.[14,15] Bad sensations and experiences fire and reinforce more lower level defensive pathways, (read "stress") and can delay or even abolish, the firing of higher level circuits.[15] The circuits affected most by stress are the ones that are "plastic", the ones that are in development at the specific time of the stress.[16] A second consequence of stress is that coping mechanisms are overused in achieving homeostasis, and this results in "wear and tear" on basic neural pathways and endocrine systems.[13] The result is "vulnerability", so that future stresses and "knocks in life" trigger pathway and system failures that show themselves in a variety of physiological and behavioural problems in later life.[17,18]

Skin-to-skin contact with father, Uppsala Academic Hospital, Sweden. Infant is 25 weeks gestation, 520 grams, on TP and CPAP

One of the most basic abilities, and that appears early in development, is to determine whether a sensation (or even constellation of such) is **safe** or **dangerous** or **life-threatening**.[19] This is seen in early fetal life and is fully competent from 28 weeks. The normal uterine sensations tell the fetus it is SAFE. Vaginal birth is highly stressful, and this birthing stress is necessary to activate the systems that make for breathing air and coping with "life outside".[20,21] But once outside, the need for being SAFE is primary, and essentially it is only mother's presence, providing sensations that are familiar, that achieve this. The chest of the mother is to the newborn its SAFE PLACE of care.[22] SAFE care means providing the three basic biological needs and mother skin-to-skin contact as PLACE of care ensures **warmth**, her breasts provide **nutrition**, and her arms cover baby for **protection**.[12] The baby is wired by highly conserved neuroendocrine responses[23] inherited in our evolutionary biology to respond to this PLACE in many different ways. At birth, the first and most urgent response we call self-attachment and breastfeeding.[24,25] After feeding, undisturbed sleep cycling is essential to establish the pathways that were fired.[1,26] Smell (and probably also mother's movements) support the newborn brain in maintaining quality sleep.[27] This sense of being safe activates the amygdala, the emotional processing unit of the brain, which connects to the frontal lobe, which controls **approach** and **avoid choices**.[2,28,29] When the brain develops in an environment that it perceives as safe, social approach is fired, and a secure attachment is formed.[2,30]

When mother is absent, the newborn brain feels unsafe, its basic needs are not provided. Mother's absence is perceived not just as unsafe but as life-threatening.[31] The amygdala tells the frontal lobe to avoid, to evade, to hide. The baby might make a short burst of crying, but the brain is likely to activate a powerful parasympathetic defence reaction, similar to that of frogs and reptiles.[31,32] This is an immobilisation defence that reduces all activity, lowering heart rate and temperature, with active suppression of movements. This looks like sleep, but is not! Careful observation over 10 minutes will reveal eye and facial twitches and whole body movements. This state is maintained by high levels of cortisol, which is a key ingredient in the "wear and tear" described above.[13] High cortisol disrupts brain architecture and healthy sleep, so neural behaviour pathways are not fired.[15] If this is reinforced in other ways, an insecure attachment is the likely result.

The brain is coded with a desperate need to feel safe, the more confusing the "safe versus unsafe" messages are to the child, the more disordered the attachment.[33,34] Birth is a highly sensitive period, how babies experience birth matters!

The human sympathetic system only matures at 2 months of age. It is however present before birth, and human infants actually need to experience some stress to develop properly.

STRESS	DEGREE	CONTEXT	RESULT
Positive stress	moderate and short-lived	stable and supportive relationships	necessary aspect of healthy development
Tolerable stress	severe but time limited	buffered by supportive relationships	brain can recover; facilitates adaptive coping
Toxic stress	severe or prolonged	in ABSENCE of buffering protection of adult support	disrupts brain architecture; stress systems respond at lower thresholds

The above table is derived from work by Shonkoff and others[35] and "absence of buffering protection of adult support" is in my own mind, a key phrase to understanding neurodevelopment. The currently accepted standard of optimal childhood development is measured by "secure attachment", this

as described by Bowlby and measured by Ainsworth.[30] A secure attachment in infancy is widely accepted as an essential aspect of future psychological health. The gold standard for measuring this is however only valid at about one year of age. Understanding the underlying neurobiology can make a difference to how the attachment is shaped before that. Infants that do not have their needs met – as expected by the genes of their evolutionary biology – may develop disordered attachments described as avoidant or ambivalent or disorganised.[30] This is succinctly described by Salk:

"There's no harm in a child crying: the harm is done only if his cries aren't answered. If you ignore a baby's signal for help, you don't teach him independence, what you teach him is that no other human being will take care of his needs."

(Dr Lee Salk, child psychologist)

This new understanding of the brain and its development can profoundly improve neonatal care. Mother's presence is an absolute requirement for OPTIMAL development. The focus of this is not survival, but emotional (amygdala) and social intelligence (frontal lobe, also called executive function), these being central to the sensitive circuits developing around birth. But this emotional and social development builds on a biological perception of safety, the warmth, nutrition and protection provided by mother's chest.

The well-known intervention popularly called Kangaroo Care (KC) can be shown to make significant benefit in terms of thermoregulation, cardio-respiratory function and metabolism.[36-38] However benefit is only evident if practised for more than one hour, corresponding to the sleep cycle required to consolidate neural circuitry. But KC fails primarily because separation is the culturally accepted default, the incubator is biologically an unsafe PLACE. Kangaroo Mother Care (KMC) is something different, being a total care strategy defined by the WHO.[38,39] There are several components, starting with "continuous or prolonged maternal-infant skin-to-skin contact" (supplemented by father or other attachment figure). Other components include breastfeeding, and early discharge.[39] KMC also fails in that current clinical evidence is not seen as requiring that this skin-to-skin contact must start at birth.

The scientific rationale here presented is founded on "maternal-infant skin-to-skin contact" from birth. Its antithesis is "separation"; in mammalian neuroscience "separation tolerance" is measured in minutes.[40,41] Current best practice already includes SSC for all newborns in the first hour of life, until the baby has had its first latch on the breast.[42] Current care then separates baby for baths and care routines, none of that separation has any evidence base.[43]

It is however in the context of **prematurity** that this neuroscience is critically important. The preterm infant is the least resilient, and the most in need of support of its basic biology. Premature infants have brains that are ready, but bodies that are not. They need technology, but this was not designed with the thought that mother should be the PLACE of care. Technology can adapt far more readily than the human brain, so ingenious solutions are usually required. Then, even with mother present, the sensations from the environment must not be intrusive or stressful, bright light and noise are the most common stressors.[44-46] Our care routines should change in one key respect, which is to ensure the protecting of sleep cycles.[26] Maternal-infant skin-to-skin contact can be - and is being - provided from 23 weeks gestation onwards. Ideally this should be round the clock, for this both parents are needed. We often give lip service to the idea, but mother and father must be conceptually and physically central to the care team.

Mother and father must be central to the care team. 32 week gestation infant; Banner Desert Hospital, Phoenix, Arizona.

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The winds of change – An update on National Health Insurance

Craig St John-Ayre Bsc Hons MSc
Managing Director, The Business Practice, Johannesburg

In 1960 the British Prime Minister, Harold MacMillan, gave a speech in Cape Town, intimating the coming independence of South Africa from Britain. In it he said, "The winds of change are blowing through this continent. Whether we like it or not, this growth of national consciousness is a political fact." The healthcare reforms we have seen over the past decades have had impacts, but the current proposal will reform and restructure healthcare as a whole, rather than tweaking here and there, impacting all parties, patients, providers, suppliers, funder organisations, taxpayers, and employers. We haven't experienced a radical change like this before.

The Challenge of the rising cost of healthcare is not a uniquely South African phenomenon, and the winds of healthcare change are blowing through many nations of the world. In late 2010, Dr Margaret Chan (WHO Director General) said, "All health systems, everywhere, could make better use of resources, whether through better procurement practices, broader use of generic products, better incentives for providers, or streamlined financing and administrative procedures."

Member States of the WHO (including South Africa) committed in 2005 to develop their health financing systems so that all people have access to services and do not suffer financial hardship paying for them. This goal was defined as universal health coverage.

In striving for this goal, governments face three fundamental questions:

1. How is such a health system to be financed?
2. How can they protect people from the financial consequences of ill-health and paying for health services?
3. How can they encourage the optimum use of available resources?

One often hears of the vagaries of National Health systems in the UK, including long surgery waits, poor quality care and loss of skills from the healthcare sector. However NHI is not restricted to the UK, and has been implemented in many other states including France, Canada, New Zealand, Mexico, South Korea, Japan, Taiwan, Chile, Thailand and Cuba. Many of these countries are seen as leaders in the delivery of healthcare, and little opposition is heard to the healthcare systems from the citizenry of these nations. In fact, 73% of healthcare spend in the European Monetary Union is from public funds. Even in the United States, which spends more on healthcare than any other country (16% of GDP), there are moves by the president to reform the system to enhance care and make it universal . The debate rages, however, over who will pay for it.

On average a South African receives healthcare investment to the value of approximately US $500 per year, however when we compare health outcome measures in South Africa to those of other countries with similar per capita healthcare spend, we perform very poorly. For example data presented by Dr Mark Blecher (National Treasury Analyst) and Dr Jonathan Bloomberg (Deputy CEO, Discovery Health) of the Financial Task Team showed that South African neonatal mortality is almost 6 times higher than these countries, and tuberculosis cure rates are significantly lower. South Africa faces an HIV burden that is the highest in the world, together with all of its health, economic and social sequelae. There is a great disparity in the level of care experienced by South Africans (Figures 1-3).

The state sector is responsible for 54% of the total healthcare spend in South Africa, however it covers 82% of the population. This means that a private patient, on average, receives 4.7 times the spend of a state patient.

There appears to be a very high level of political will to introduce NHI. The ANC's 2009 Election Manifesto stated that "the government will introduce a National Health Insurance system, phased in over 5 years. NHI will be publicly funded and publicly administered, and will provide the right of all to access quality healthcare, which will be free at the point of service. People will have a choice of which service provider to use in their district"

In April 2010, during his annual budget speech, Dr Aaron Motsoaledi, Minister of Health, referred to the introduction of NHI. In fact, it is the second point of the Ministry's 10 priority point programme. To quote from the Minister's speech, "Through NHI, we will ensure universal access to good quality and affordable health services for all South Africans."

This was mirrored in the COSATU press statement upon the unfortunate death of the Deputy Minister of Health, Dr Molefe Selufaro which stated, "Comrade Sefularo's death should serve as a call to the African National Congress and government to implement the National Health Insurance Scheme in the interests of the working class and the poor." On the 7th of May 2010, Mr Zwelinzima Vavi, General Secretary of Cosatu, said "Our ANC-led government has made a bold decision to introduce national health insurance. It will be founded on the core principle of universal coverage, so that all South Africans will have access to healthcare at all times and not be dependent on ability to pay premiums to a medical scheme, with all unnecessary barriers kept to the minimum." Enhancement of healthcare and introduction of NHI are the second priority for both treasury and the Department of Health. There is a very strong drive from the ruling party and its alliance partners to introduce NHI, and it is

unlikely that it will not occur. The government is also aware of the economic benefits of a healthier population. Each year of increased life expectancy can raise a country's per capita GDP by up to 4%.

Which way are the winds blowing?

In the last few months we have seen several initiatives to either reduce the cost of healthcare or establish a strong framework for the introduction of NHI including:

- A project by the Council for Medical Schemes to define clearly prescribed minimum benefits.
- Requests for information from pharmaceutical manufacturers as part of the process of implementing international benchmark pricing.
- Changes to regulations regarding remuneration of Pharmacists.
- Initiatives by the Department of Health to implement pharmaco-economic justification for treatments.
- International procurement of anti-retrovirals, reducing the costs by several billion rands over the next two years.
- Initiatives to stabilise costs in the private medical aid sector.

Possibly the most enlightening documents on NHI are the ANC Proposal on NHI and the ANC National General Council (2010) Documents on Healthcare. They propose:

- NHI will focus on Primary, Secondary and Tertiary care, and not on Quaternary care. The definitions of these are not conclusive, however.
- A National Health Insurance Fund (NHIF) will be established.
- There will be a single payer for medical care provided under the banner of NHI.
- Regional negotiators will determine the levels of reimbursement for goods and services provided to NHI patients.
- Power to negotiate low treatment cost from providers and suppliers will come from the number of patients and level of funding that the NHI will represent.

The NHI body will be managed by a director who will report into the Ministry of Health. This director will manage an executive committee comprising technical advisors and heads of portfolios including audit, pricing, benefits, reimbursement, quality enhancement and others.

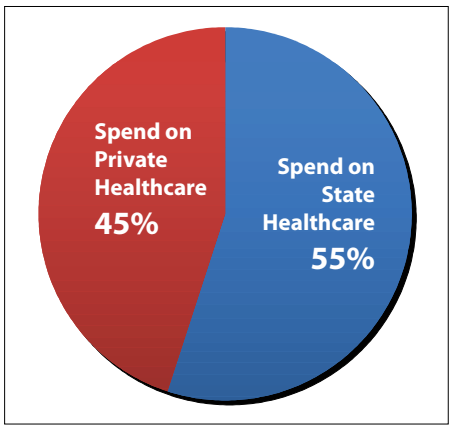

Figure 1

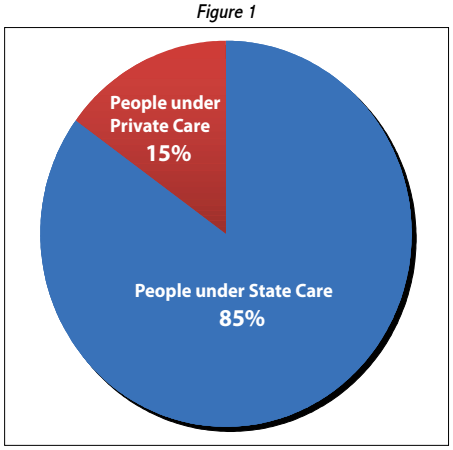

Figure 2

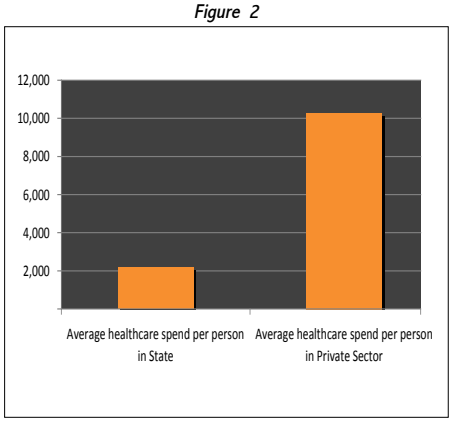

Figure 3

Where will the funding come from?

The funding for NHI will come from:

- Income-based NHI contributions from all employed people.
- Possible additional taxation and contribution from the State fiscus. These taxes could include additional:
  - Social Welfare tax
  - A change to the VAT rate
  - NHI Contributions and tax contributions will be pooled in the NHIF, and these will be used to reimburse healthcare service and product providers at negotiated fees and prices. There should be no co-payment by the patient.

What impact will NHI have?

The potential impact of introducing NHI can only be assessed once a set of benefits has been defined, and the associated costs calculated. Whilst there is much analysis being done, the impact remains unclear. It will be an exercise in medical costing, but will also have to consider social, economic and political ramifications. Needless to say that this is a highly complex activity with numerous stakeholders. There are those that will demand clarity and certainty at the expense of implementation speed, whilst others will demand that an imperfect system is better than a perfect-but-late one. It remains to be seen whether political pressure to implement NHI quickly will take priority over rigorous analysis. A balance will have to be found, as the current levels of disparity in healthcare levels between the privately funded minority and the publicly supported majority are worsening, and threaten healthcare for all in South Africa.

Hold onto your hat I can feel a breeze kicking up. Whether these winds will wreak havoc or clean out the old for the shining new remains to be seen.

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5. The South African Population is estimated to be 49.8 million according to Statistics South Africa Mid-Year Population Estimates July 2010.
6. Private sector healthcare spend is the total contributions paid to Medical Schemes, R74 billion, and excludes non reimbursed payments, i.e. self-funded healthcare payments. The population covered by medical insurance is estimated at 7.2 million. The remainder are assumed to be self-funding or under state sector care. Source: Council for Medical Schemes Annual Report 2009, reflecting 2008 information. No cognisance is taken of cross subsidy between sectors in the Risk Equalisation Fund.
7. This does not however take cognisance of the difference in what it costs to treat a state patient relative to a private patient.
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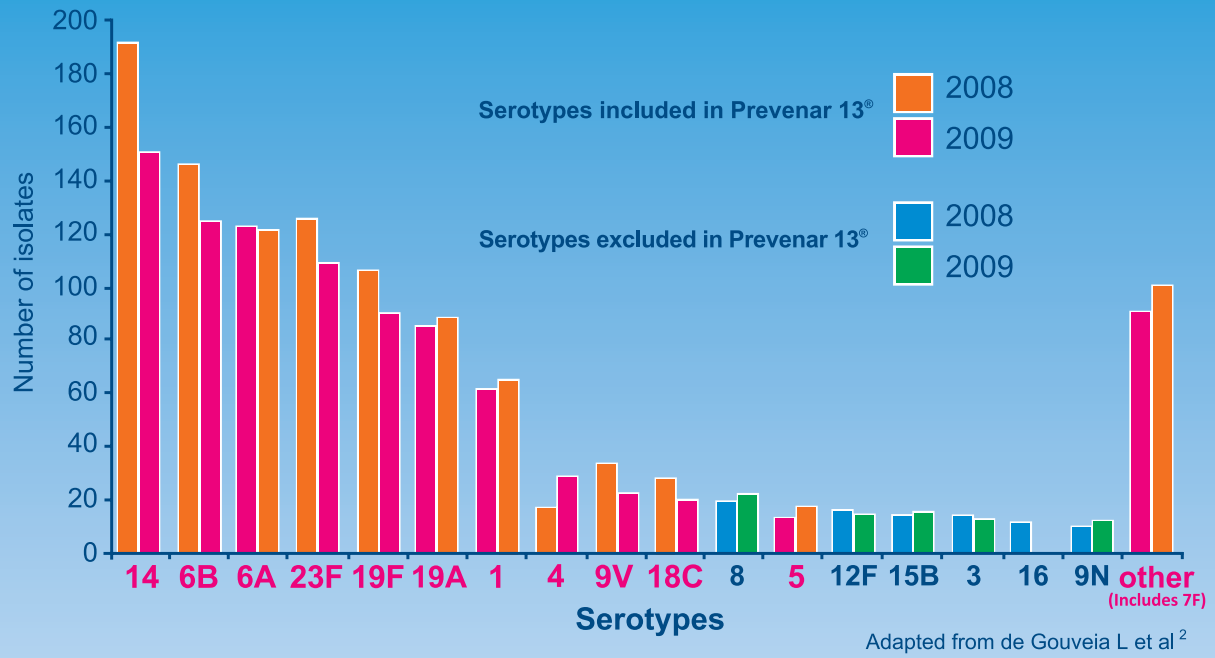
Descending order of Pneumococcal serotypes causing IPD in children < 5 years, South Africa, 2008 and 2009 [2]



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