George Abbott Symposium

& Festschrift for

Emeritus Professor Brian Darlow

Rolleston Lecture Theatre, University of Otago

Christchurch

24th & 25th August 2018

"NICU & BEYOND"

The Royal New Zealand College of General Practitioners

RNZCGP Endorsed CPD Activity

George Abbott Symposium
University of Otago, Christchurch
August 24th & 25th August 2018

Keynote Speakers

Professor Jenny Couper, Paediatric Endocrinology, University of Adelaide
Professor Keith Grimwood, Paediatric Infectious diseases, Griffith University, Gold Coast
Associate Professor Helen Liley, Neonatology, Mater Research, Brisbane
Professor Jane Harding, Paediatric Endocrinology, University of Auckland

Other featured speakers

Professor Brian Darlow
Paediatric Neonatologist, University of Otago, Christchurch

Dr Martin de Bock
Paediatric Endocrinologist, University of Otago, Christchurch

Professor Dawn Elder
Paediatric Sleep Physician, University of Otago, Wellington

Dr Simon Rowley
Consultant Paediatrician
Auckland District Health Board

Associate Professor Nicola Austin
Neonatologist
Canterbury District Health Board

Professor Spencer Beasley
Paediatric Surgeon, University of Otago, Christchurch

Professor Barry Taylor
Dean
Dunedin School of Medicine

Dr Sarah Harris
Neonatal Pediatrician, University of Otago, Christchurch

George Abbott Symposium & Festschrift

Christchurch, August 24th & 25th 2018

Festschrift Friday August 24th

0900-0920	*Welcome and Introduction* Professor Andrew Day Cure Kids Team
0920 – 0950 0950 - 1020	Professor Christine Winterbourn *Free radicals, antioxidants and the complications of prematurity* Professor Lisa Askie *Boost trial*
1020 - 1100	**Morning tea**
1100 - 1130 1130 - 1200	Professor Barry Taylor *5 year outcomes of a Childhood Obesity Prevention RCT* Professor Helen Liley *Newborn Resuscitation – Working with the baby*
10 minute break whilst we are joined by hospital staff for Grand Rounds	
1215 - 1220 1220 - 1245 1245 – 1310	Professor David Murdoch, Dean of UOC *Grand Rounds Introduction* Dr Simon Rowley *3 decades of Neonatology* Professor Keith Grimwood *GBS Infections in NZ infants*
1315 – 1415	**Lunch**
1415 - 1440 1440 - 1505 1505 - 1530 1530 – 1555	Professor John Horwood *Child development study* Professor Jenny Couper *Developmental origins of Type 1 Diabetes* Associate Professor Mark Elder *Retinopathy of prematurity* Associate Professor Nicola Austin *Neonatalogy going forward*
1600	**Afternoon tea & close**

Saturday August 25th

0830	*Welcome and Introduction* Associate Professor Tony Walls
0845 – 0925	Professor Brian Darlow *Long term follow up of premature infants in NZ*
0925 - 1000	Professor Jane Harding *Long term consequences of neonatal hypoglycaemia*
1000 - 1045	**Morning tea**
1045 – 1115	Dr Martin de Bock *Neonatal diabetes and hyperinsulinism; new genes and new drugs*
1115 – 1145	Professor Jenny Couper *HbA1c above target: what else can we do to prevent complications in type 1 diabetes?*
1145 - 1215	Professor Helen Liley *Neonatal Hpoxic Encephalopathy – Improving outcomes*
1215 - 1315	**Lunch**
1315 – 1345	Dr Sarah Harris *Pulmonary vascular disease in preterm infants*
1345 – 1415	Professor Keith Grimwood *Azithromycin: should we use it in bronchiectasis?*
1415 - 1445	Professor Spencer Beasley *Oesophageal atresia*
1445 - 1515	Professor Dawn Elder *Overnight oximetry in preterm and term infants: Do we know what is normal?*
1515	**Finish – afternoon tea available**

Professor Brian Darlow
Paediatric Neonatologist, Christchurch

Long term follow up of premature infants in NZ

Professor Jane Harding
Paediatric Endocrinologist, Auckland

Long term consequences of neonatal hypoglycaemia

Neonatal hypoglycaemia is the commonest metabolic disorder of the newborn, affecting 5 - 15% of all births and 50% of babies in risk groups. While it is generally accepted that severe, prolonged or repeated hypoglycaemia can cause brain injury, there is much controversy about whether mild and transient hypoglycaemia has any consequences for later neurodevelopment. This is reflected in widely varying recommendations for screening of babies at risk, and for treatment thresholds. The limited evidence for later consequences of neonatal hypoglycaemia is based on small numbers of often poor quality studies with short duration of follow-up. Nevertheless, data are emerging suggesting that even mild and transient neonatal hypoglycaemia can be associated with specific neurocognitive impairments that only become apparent in mid-childhood and may result in educational disadvantage. Well designed randomised trials with follow-up at least to school age are required to clarify causal relationships and optimal management of neonatal hypoglycaemia.

Dr Martin de Bock
Paediatric Endocrinologist, Christchurch

Neonatal diabetes and hyperinsulinism: new genes and new drugs

There has been increased understanding of the genetic basis of congenital disordered insulin secretion; causing either hyperinsulinism or neonatal diabetes. For hyperinsulinism in particular, there is increased insight into the heterogeneity of genotype and phenotypic expression with respect to focal and diffuse disease. New tools in diagnostics and trials of new therapies have given more guidance for clinicians in investigating and managing these challenging conditions. This talk will provide an update.

Professor Jenny Couper

HbA1c above target: what else can we do to prevent complications in type 1 diabetes?

Intensive management with recent technological advances in insulin delivery and continuous glucose monitoring systems have greatly improved diabetes control and eased the burden for many children and their families. However, the majority of patients still do not reach target levels of diabetes control to minimise their risk of long-term vascular complications. Good patient selection can achieve benefits of additional agents to insulin, such as metformin in type 1 diabetes, incretin analogues, ACE inhibitors and statins. Agents to preserve beta-cell function are also the future.

Professor Spencer Beasley
Paediatric Surgeon, Christchurch

Oesophageal atresia

Oesophageal atresia (OA) is a rare (1:4000 live births) but challenging condition. The diagnosis can be made on antenatal ultrasonography, but not consistently so. Where OA is suspected at birth – excessively drooling neonate – the first priority is to confirm the diagnosis: this involves passage of a 10G tube through the mouth into the oesophagus. If it becomes arrested at 9cm the diagnosis is confirmed.

The next step is to establish the type of OA: an Xray of the torso showing gas below the diaphragm confirms there is a distal tracheo-oesophageal fistula (TOF). This is the most common type (85%). Absence of gas indicates there is no distal fistula. A gasless abdomen usually indicates that there is a long gap atresia i.e. a substantial length of gullet is missing, and about 20% of these will have a proximal fistula which can be shown on endoscopy or contrast imaging.

The third step is identifying associated anomalies that may influence management of the OA/TOF. These tend to fall into recognised patterns eg VATCTERL association, CHARGE association, Trisomy 21, 13, 18., although about 50% have no concomitant abnormalities. Duct dependent congenital heart disease and bilateral renal agenesis are two lesions that must be recognised promptly.

The priority in surgical treatment is control of the distal fistula. This is normally done at the same procedure as repair of the OA with an end-to-end oesophageal anastomosis. OA is always associated with a degree of tracheomalacia, but this tends to improve with growth. Surgical complications include stricture and anastomotic leak. GOR can be problematic because of poor oesophageal clearance and the increased risk of stricture formation. FB impaction occurs sometimes in pre-school children, but swallowing improves with time. Long term quality of life is good.

Dr Sarah Harris
Neonatal Paediatrician, Christchurch

Down the Rabbit hole of Pulmonary vascular disease

Lung development is critically dependent on the development of the pulmonary vasculature according to the "vascular hypothesis" of bronchopulmonary dysplasia(BPD). In 20% of low birthweight infants BPD is complicated by pulmonary hypertension associated with increased morbidity and mortality. Adults born preterm have elevated pulmonary pressures and right ventricular dysfunction compared to term born peers. Oxygen is a key protagonist in the development of a dysmorphic and dysfunctional pulmonary vasculature however optimising oxygen saturation levels is challenging. Identifying pulmonary vascular disease is difficult. Cardiac catheterization, the gold standard, is invasive, carries a morbidity risk and has limited availability. Conventional echocardiography techniques do not reliably predict BPD-associated pulmonary hypertension when compared directly to cardiac catheterisation. B-type natriuretic peptide (BNP) is a cardiac hormone released by ventricles under pressure of volume stress. Here we explore the role of oxygen in lung development, the potential role of BNP and novel heart ultrasound measures as markers of pulmonary vascular disease. Finally we will discuss recent recommendations around screening for pulmonary hypertension in preterms.

Professor Keith Grimwood
Paediatric Infectious Diseases specialist, Gold Coast

Azithromycin: Should we use it in bronchiectasis?

Azithromycin, possesses broad-spectrum antibacterial and immunomodulatory properties. The prolonged half-life of azithromycin allows convenient once-daily dosing, leading to it being second only to the penicillins as the most commonly prescribed antibiotics worldwide. While used mainly to treat acute respiratory, gastrointestinal, skin and genitourinary infections, azithromycin is also now being prescribed increasingly for a broad range of chronic pulmonary disorders characterised by neutrophilic inflammation, such as cystic fibrosis, bronchiectasis, chronic obstructive pulmonary disease and poorly-controlled asthma. Nevertheless, evidence supporting macrolides in these conditions remains inadequate.

Long-term azithromycin potentially confers benefit at every level of the vicious cycle hypothesis of chronic lower airways inflammatory diseases driven by infection. Randomised controlled trials in cystic fibrosis and bronchiectasis report on average a 50% reduction in exacerbations when azithromycin is administered for 6-24 months, although whether this is sustained is unknown. Similarly, it remains unknown which patients benefit most from long-term azithromycin, as well as what is the optimal dose, dosing frequency and course duration.

Azithromycin's favourable pharmacokinetics also results in prolonged sub-inhibitory concentrations at carriage sites and selection of antibiotic resistant strains. Poor adherence increases the risk of antibiotic-induced dysbiosis and macrolide-resistance. While antibiotic-resistance is usually reversible once treatment is ceased, macrolide-resistant *Staphylococcus aureus* strains persist. The impact of macrolide-resistance at an individual and community level for those with respiratory infections is uncertain. Macrolide-resistance in *Haemophilus influenzae* is associated with treatment failure in otitis media, while the effect on pneumococcal bacteraemic pneumonia is inconsistent, and whether it influences the course of chronic lower airway infections is unknown, although selecting macrolide-resistant mycobacteria in these patients is concerning.

Macrolides, such as azithromycin, highlight the challenges of translating *in-vitro* observations into clinical trials and practice, while trying to avoid 'therapeutic creep,' unintended 'off-target' effects and widespread antibiotic resistance.

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Professor Helen Liley
Paediatric Neonatologist, Brisbane

Neonatal Hypoxic Encephalopathy – improving outcomes

The algorithms for newborn resuscitation are more complex than advanced life support algorithms for other patients because most babies are not asystolic and lifeless at birth (although a few are). A few need extensive resuscitation (and can respond well to it) but many more just need support for their own physiological adaptation, so newborn algorithms emphasise graded interventions. Resuscitation interventions have the potential to improve or impede the baby's own efforts to recover, and to cause harm, so it stands to reason that it is important we apply them at the right time, with great skill and only to the extent they are needed.

Clinical assessment is needed to evaluate whether to start resuscitation manoeuvres and to decide whether to continue, to cease them or to escalate. This depends on an understanding of the reliability and implications of clinical signs, and of the signals from monitoring equipment. For example, heart rate has been proposed as one of the most important signs in newborn resuscitation, but research suggests the potential for artefacts in measurement, and that the "normal" range is dependent on time from birth and on clinical factors (such as umbilical cord management). We need to be aware that heart rate may be assessed differently by auscultation, pulse oximetry and ECG. Finally, good teamwork is needed to translate this information into a shared mental model and coordinated response.

Professor Dawn Elder
Paediatric Sleep Physician, Wellington

Overnight oximetry in preterm and term infants. Do we know what is normal?

Assessment of current apnoea was previously the main way of determining readiness for discharge in preterm infants. Now the consequences of apnoea such as oxygen desaturation and arousal are increasingly being considered as part of decision making without detailed measurement of apnoea. The term intermittent hypoxia (IH) describes the brief drops in oxygen saturation seen after apnoeic events in these infants. There is limited literature documenting IH at preterm infant discharge using new generation oximeters. Instead the focus has more commonly been on measurement of mean oxygen saturation and time spent with an oxygen saturation that is <90% or < 80%.

This presentation will discuss assessment of cardiorespiratory stability at neonatal discharge and present data from our studies in preterm and term neonates, at discharge and in the first 8 months of life, using 24 and 12-hour oximtery to document desaturation indices over time as measures of intermittent hypoxia.

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