Highlights from this issue

Nick Brown, *Editor-in-Chief*

CORONAVIRUS

So many more questions, but at the same time so much knowledge gleaned in such a short time. COVID-19 continues appropriately to dominate the headlines but, while only weeks ago, most literature was in the realm of speculative, we now have (at least some) robust data on which to base our advice and practice. And perhaps, to invert a Runsfeldianism, we also know more about what we don't know… and the breadth of this month's manuscripts testify to this: UK reference data (*see page 613*), global health implications (*see page 616* and the WHO group, *see page 620*), the arguments around school closure (*see page 618*), the disappearance of children from emergency departments (*see page 704*) and (and this is unexpectedly dystopian) the implications for chronic disease management using inflammatory bowel disease as an example (*see page 706*). Despite the emerging Kawasaki like inflammatory syndrome (PIMS-TS), no one would debate children's relative resistance to severe infection. We still know very little about children and transmission and we have no idea at all about the shape that the secondary effects (breakdown of health systems, mental health, late presentation, abuse being unidentified) will take, and this is the largest of all the dark clouds currently hovering overhead.

LINEAR GROWTH

There's a long held perception that skeletal growth rate has a more or less linear relationship with skeletal maturity, itself measurable by a number of radiological techniques based on extent of ossification. In an analysis of the Ohio Fels longitudinal survey, Boeyer debunks some of the mythology around this association. During the study, participants had an average of 25 bone age assessments and two distinct growth subgroups were demonstrated. Some participants underwent two periods of rapid growth, on in childhood and one in adolescence, while others only a later spurt. The latter, however, embarked on their rapid growth phase earlier, almost 3 years and more than 4 years in girls and boys respectively, a rare case of 'real' catch up. *See page 631*

DISABILITY

A major cause of disability globally, the prevalence of cerebral palsy in low and middle income countries is much harder to estimate. Later or non-presentation at health facilities and death before diagnosis are possible explanations, but detection in general certainly an issue. Duke uses key informant methods of identification of children with possible CP through village volunteers in Cross River State, Nigeria. All were invited for confirmation of the diagnosis by a paediatric neurologist. About 70% of the children initially identified about 70% attended: of those, 35% had other pathology. Other than grading of severity of the CP by motor restriction (Gross Motor and Function Classification System) and fine motor ability (Manual Ability Classification System), the history was probed for timing of likely causal event. Prevalence was estimated as 2.3/1,000 a figure very similar to that in 'typical' high income countries, though possibly an underestimate of children who might have developed CP had they survived early perinatal insult. In the postnatally acquired group, severe malaria and meningitis accounted for the vast majority of postulated causes but, irrespective of the causes, the need to 'find' children suggest that many are not receiving the essential paediatric, physiotherapeutic and nutritional input they require. *See page 625*

COMPLEXITY

We all follow complex children and, in the context of level of care, no one would refute they need even more detailed supervision than their 'non-complex' contemporaries. The problem, though, is that we all have different definitions. This issue, closely linked to their identification is the starting premise of the first two instalments of Joy Gough's mini series 'cracking a complex problem', a narrative of the steps in the quality improvement project on which she and colleagues embarked. *See page 694 and 695*

INFLAMMATION, POVERTY AND CHRONIC DISEASE

The early/foetal origins of life hypothesis has taken many interesting turns since David Barker's first description of the association in the mid 1980s. One recent avenue is that of chronic low grade inflammation, poverty and later chronic disease. The link between early exposure is now well established, but has not been explored in adolescence. Fraga used data from the Portuguese EPITeen cohort using exposure data in the form of maternal maternal and paternal education and occupation as indicators of childhood socioeconomic conditions. High-sensitivity C reactive protein was measured at three points in time (13, 17 and 21 years) and categorised in tertiles separately for each wave; chronic low-grade inflammation in adolescence was defined as having hs-CRP levels in the highest tertile in at least two waves. Adolescents with lower parental socioeconomic position had consistently low grade inflammation even after adjustment for sex, perinatal and physical environment factors, health-related behaviours and health status in adolescence. *See page 677*

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