



Highlights from this issue

Nick Brown , *Editor in Chief*

The final act of Philip Roth's achingly poignant novel, *Nemesis*, based on the 1944 polio epidemic condenses the trajectories of each of the main protagonists' lives as a result of events that sultry summer, Bucky Cantor, swapping life stories with a former pupil at his school. Though both had survived the illness, their subsequent lives were permanently altered, Cantor's in particular unfolding (imploding) inexorably. Like all Roth's works, this is a tragedy, in this instance played out with the backdrop of stifling heat of that summer. Each of the players was altered by those events and, though I'm not claiming any direct analogy between that and our current era is valid, I have found it hard not to keep returning to the book, unable to stop drawing parallels about those events and the ones we are now witnessing.

SARS-COV-2

To date, children have appeared less susceptible to the acute dysregulated inflammatory response that has claimed the lives of so many of their parents and grandparents, but reports of a novel Kawasaki like illness might require some modifications to this generalisation. Either way, the implications for long term (psychological and physical) health are arguably worse: late presentation for other febrile illness (EDs have never been so empty—something is wrong); fear of infection by dint of 'exposure' to a health facility; interruption of standard health surveillance particularly vaccination; mental health; child abuse as a result of prolonged internment and loss of, at least the social side of, education.

So what does the latest data tell us? Look at our COVID-19 site on adc.bmj.com.

Ladhani (10.1136/archdischild-2020-319363) examines the epidemiological data to date in the UK. The reasons for less aggressive disease are still not

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completely understood, though there are a number of candidate explanators: host-response factors; lower infective dose (as most often from an adult household member); age related ACE receptor differences and more recent exposure to antigenically similar coronaviruses conferring relative immunity. Infants seem to be more vulnerable than older children but, in the absence of date of population screening and seroconversion estimates, there is little on which to base modelling. Remember at the start of March, just 6 weeks ago, the UK had witnessed only 85 cases... exponential curves (and unusually, the mathematical rather than purely literal definition applies here) are invariably harder to predict.

What of the global situation? What is the expected trajectory of low and middle income countries? Jonathan Klein and colleagues at the International Paediatric Association provide an update on the global situation (10.1136/archdischild-2020-319730): Trevor Duke (10.1136/archdischild-2020-319333) describes the unknowns and vulnerabilities. What is the outcome in HIV or TB co-infection; what about malnutrition? Are BCG vaccination and heat (each with proponents) protective?

Is there enough PPE, soap and sanitiser? Can hypoxia be identified (saturation monitors) and treated? Cylinders last a couple of days, but, concentrators (provided there is power and solar is an option) don't require a stockpile.

LIFE BEYOND COVID-19**Pulsus paradoxus**

Krishnan's paper and Powell's editorial evaluate the predictive value of pulsus paradoxus in children with acute asthma in Singapore. Though the usually accepted (and most readily quantified) standard of asthma severity is the peak expiratory flow, it is often unobtainable in young children and severe exacerbations.

Pulsus paradoxus (first described by Kussmaul in the 1870s) is classic medical school physiology: a greater negative intrathoracic pressure, causing increased right ventricular filling and, as a result of a finite intra-pericardial volume a splinting of the left ventricle.

The final result is a fall in left ventricular stroke volume and blood pressure (10 mm Hg in systole) and a compensatory increase in heart rate. I don't want to spoil the punchlines, but, PP consistently predicted the need for adjunctive drug treatment and ventilatory support. There are riders: it isn't easy to measure and susceptible to interobserver variation, but, the paper argues the case for at least including PP in the assessment of a syndrome in which all the individual measures (PEF, blood gases, ability to complete sentences, gut feeling) are all imperfect. This is my editor's choice for the month. *See pages 533 and 521*

THIN, STUNTED OR BOTH?

Defining undernutrition is an issue that the international child health community have wrestled with for generations. Part of the (arguably insoluble) problem is the overlap between chronic and acute malnutrition, represented phenotypically shortness (stunting) and thinness (wasting) respectively. Standard deviation scores for each are easy to derive (WAZ and HAZ), but don't solve the fundamental issue that many children have both. How does one define a short thin child whose weight for length z score is bafflingly 'normal'? Compounding the terminological problems are the multiple candidates for best marker: skin folds; mid upper arm circumference, weight for age. Few would argue, though, that conditional weight gain (a regression of current against previous z scores) is a valid marker as is an extremely low sum of skin folds (SSF). Despite vocal advocates, there is remarkably little previous literature addressing the question in the under 6 month age group and with this backdrop Ezeofar's study and Trehan's editorial provide some clarity. Using SSF and a conditional weight gain of more than -2 SD as standards for weight faltering, WAZ, MUAC and weight for length scores were compared. WAZ performed best (sensitivity 69%, positive predictive value 86% and likelihood ratio of 5.5) and, assuming generalisability, there are reasons to be optimistic. *See pages 524 and 522*

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