

# Causal pathways and prevention

It is 110 years since Sir William Osler published his little book on the cerebral palsies of childhood<sup>1</sup>. Less than 10 years later Freud published his observations<sup>2</sup>. And if we are looking for landmarks, perhaps the next is Crothers and Paine's book, published 40 years ago<sup>3</sup>. Ingram's 'Paediatric Aspects of Cerebral Palsy' follows this, published in 1974<sup>4</sup>. But Little's paper predates all of these<sup>5</sup>.

Sir William Osler was not overly concerned with a definition of the cerebral palsies. He recognized them as originating in 'an upper cortico-spinal segment, extending from the cells of the cortex to the gray matter of the cord,' and resulting, he thought, from 'a destructive lesion of the motor centres, or of the pyramidal tract, in hemisphere, internal capsule, crus or pons.' Crothers and Paine added two more criteria: 'The original lesion must occur early in life', and '...that no active disease exists at the time of the diagnosis.' These early descriptions are inclusive of any definitions we now use.

In their forthcoming book on the epidemiology of the cerebral palsies, Stanley, Blair, and Alberman<sup>6</sup> plump for Mutch's definition: 'an umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development.' Despite the lack of precision in both the aetiology and pathology inherent in these clinical descriptions, this definition of cerebral palsy (CP) has proved, in my view, extremely robust.

What is striking about the epidemiological studies reviewed by Stanley and her coauthors is the persistent prevalence of CP – about 2 to 2.5 per 1000 live births. Many efforts have been made to try to reduce this.

Simplistic suggestions and continuing medico-legal attempts to pinpoint blame and to obtain damages for individuals with CP have no doubt played their part in negating our efforts to do anything effective about prevention. By simplistic I refer, for example, to a recent request from lawyers about whether I thought an isolated event in a pregnancy 20 years ago could account for a young man's CP. If I were to bless the event, the young man might receive considerable compensation. But because I was unable to do this, he lost out.

If prevalence is to be reduced, we need to unravel the causes. Although envisaged in many previous publications, Stanley et al. emphasize a causal pathway for CP rather than a single event. This pathway recognizes the multifactorial events which lead to the child developing CP: 'multiple birth leads to preterm delivery which leads to neonatal cerebral damage in cerebral palsy.' They go on to say: 'Factors increase vulnerability to later causal factors (e.g. intrauterine growth restriction may decrease the infant's capacity to cope with intrapartum stress).' Finding a cause is increasingly complex now that we are looking for multiple causal factors.

Studies are continually needed to see whether our

proposed interventions into brain damage at points along the causal pathway are effective. The article by Sciberras and Spencer in this issue explores the Maltese population with CP<sup>7</sup>. The prevalence of CP has not substantially changed over the years; the much vaunted neonatal services have had little impact. One of the problems with all epidemiological studies is that data are inevitably old when they appear. But this study has significant contributions. Firstly, the difference in the types of CP found compared with those in other studies, and secondly, the influence of social class on the findings. Social class has previously been examined with, in general, equivocal results. One has believed that, as with severe learning disorders, biological factors are predominant in determining the disease. But perhaps this is not the case, as our colleagues from Malta suggest.

In some ways their study does not have the rigour that Stanley and colleagues would like. What is needed to develop the pathway hypothesis is as much data as possible on the foetus and infant. But retrospectively it is always difficult to collect reliable data.

'The causal pathway model', write Stanley, Blair and Alberman 'increases our potential to devise preventative strategies'. Early interruptions of a pathway should be the most effective, but it is curious that improvements in public health over the last few decades have not affected the prevalence of CP. We must continue to work at later portions of the pathway and improve the care of mothers and neonates. All the hard work of the epidemiologist and others in the 15 years since Stanley and Alberman's initial publication on the epidemiology of the cerebral palsies may make one feel that, like Alice, one has to run to stand still. But their forthcoming publication clears the way for a new sort of race which means that we will reduce the prevalence of CP in the foreseeable future.

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## References

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