Countering obstetricians' deflections: The role of magnetic resonance imaging in cerebral palsy litigation in South Africa in context

To the Editor: The article by Bhorat *et al.*[1] in the *SAMJ*, entitled 'Cerebral palsy and criteria implicating intrapartum hypoxia in neonatal encephalopathy – an obstetric perspective for the South African setting', starts off by raising concerns about 'steep rises in insurance premiums, placing service delivery under serious threat'. It does not acknowledge any service delivery issues that already exist in the public sector obstetric services in South Africa (SA). According to Whittaker,[2] in 2019, there were 303 obstetricians and gynaecologists employed in the SA public sector and 579 in the private sector, and of those employed in the public sector, 190 were performing private sector work. That a large number of the children with cerebral palsy (CP) were delivered in the public sector service was not noted by Bhorat *et al.*,[1] nor was the fact that the overwhelming majority of court cases are against the state (not against individual doctors) in provinces and hospitals with significant medical staffing and resource issues. For example, the liabilities for Eastern Cape Province in the 2019/20 period were ZAR36 751 207 v. only ZAR33 155 in Western Cape Province for the same period.[2]

Bhorat *et al.*[1] acknowledge that the low- to middle-income country setting should be considered separately from well-resourced countries, but only after first quoting figures from the developed world, where 'only 10 - 14% of CP cases have been shown to be caused by intrapartum hypoxia'. Elsingergy *et al.*[3] have shown that in the CP population undergoing investigation for litigation in SA, 71% of cases were due to hypoxic ischaemic injury (HII) and 61% were ascribed to injury at term.[3] The usefulness of magnetic resonance imaging (MRI) scans in this scenario is evident in this article, which states that in ~30% of cases MRI indicated that the CP was not due to hypoxic ischaemic injury, and litigation did not proceed in these.[3]

The lack of radiologists among the authors of the article by Bhorat *et al.*[1] should have discouraged them from making broad comments about the usefulness of MRI, especially in the setting of CP litigation in SA. They fail to appropriately note that MRI, even many years after the alleged perinatal event, provides the best available information about the condition of the brain at the time of the scan and is beyond the capabilities of any other method during the life of a child with CP. They make the point that early MRI is likely not to be available in the acute phase in the SA setting, but in fact this is not true of private practice or the urban settings in which a number of these authors work, in the major centres of Johannesburg, Pretoria, Cape Town and Durban.

It is true, however, that in the context of CP litigation, it is important that MRI is largely unavailable in the public setting, outside of the urban referral centres. This lack explains why delayed MRI scans are requested for the purposes of determining the cause of CP as a starting point in litigation cases – these delayed scans are the only MR imaging available in most cases. None of the patients in Elsingergy *et al.*'s[3] study had undergone any MRI scans prior to litigation. These include 36 patients with potentially progressive metabolic disorders, 23 with stroke, 35 with kernicterus and 78 with congenital malformations, who may have benefited from different management. When considering the median (interquartile range) age of 6 (4 - 9) years of the children at the time of this first and only MRI scan, it is reasonable to conclude that no doctor or other employee of the state responsible for the care of these children had pursued making a more definitive diagnosis, or more importantly acknowledged that a potentially avoidable HII had occurred. The latter has masked what is a massive failure of health services in SA.

For the purposes of CP litigation, delayed MRI is extremely useful in proving beyond doubt the presence of brain injury, the distribution of this injury for determining a cause for CP, and the severity of injury. Contrary to what is asserted by Bhorat *et al.*,[1] even delayed MRI provides information regarding the timing of injury. MRI is able to distinguish many of the different brain injuries causing CP based on distribution patterns. For example, it is often possible to easily distinguish perinatal arterial ischaemic stroke from HII because the former is most often unilateral and within an arterial territory. Metabolic disorders, which can be bilateral and symmetrical as with most cases of HII, can have characteristic patterns of their own. Some congenital infections also have specific patterns of injury. MRI can determine the timing of injury along a broad developmental timeline. It can demonstrate when a brain injury occurred in the womb before neuronal migration, before gliosis was possible in a fetus, or that a visible gliotic response indicates a degree of maturity, that the pattern of injury indicates that injury occurred in a premature brain, or that an injury occurred in a mature (term) brain, but not beyond the neonatal period (by the presence of ulegyria). Patterns of HII can also indicate severity, when an HII was 'partial' (watershed), 'near complete/profound' (basal ganglia-thalamus) or 'catastrophic' (multicystic encephalomalacia), based on the MRI distribution and features.

Lastly, Bhorat *et al.*[1] miss the fact that delayed MRI is often more sensitive and accurate in determining the extent of injury than MRI in the acute period.[4] This is because MRI in the delayed phase demonstrates 'permanent' injury, and the signal abnormality is accentuated by the regional volume loss. MRI scanning in the acute phase relies on diffusion-weighted imaging, which is the most accurate way of timing the insult to the perinatal period, but if not well timed in the first 5 days (120 hours) of life, the signal abnormality may pseudo-normalise; furthermore, acute-phase MRI may also demonstrate reversible changes, making it inaccurate in determining the extent of injury, and it does not demonstrate post-synaptic damage subsequent to apoptosis that results in the destruction of functional circuits, rather than individual cells.

The workflow diagram laid out by Bhorat *et al.*,[1] if applied to the litigation of CP cases, leans on the patient chart (clinical notes) to determine the presence of neonatal encephalopathy, before indicating a use for neuroimaging. The authors do this despite acknowledging subjectivity in recording of Apgar scores and interpretation of cardiotocograms, and rely first on determination of the presence of neonatal encephalopathy. They expose their lack of experience in reviewing the poor medical records kept in state practice outside that in which they work, which is now evident in the many cases going to court and many awards to plaintiffs. Of 130 written judgments in medical malpractice where the reasons were provided, 36 (28%) were specifically for lack of record keeping.[2] Whittaker[2] also notes that in ~75% of CP judgments, lack of fetal monitoring was proven.

In the context of CP litigation in SA, only 2 of the 10 criteria recommended by Bhorat *et al.*[1] 'to implicate intrapartum hypoxia in neonatal encephalopathy' are readily available and/or reliable in litigation cases: neuroimaging studies (no. 7) and 'developmental outcome is spastic quadriplegia or dyskinetic CP' (no. 9). Both of these items are achieved first hand by interpreting a current MRI scan for permanent brain injury and examining the child in his or her current state, respectively. All other information is secondarily determined by experts interpreting other people's notes, which may or may not be available, represent accurately performed procedures,

or be accurately interpreted or adequately recorded. These are significant differences from a clinical environment where such recommendations can be followed at the time of delivery. It is not clear why the authors do not list these as practice recommendations for better outcomes rather than making them recommendations for litigation, which are unrealistic. Minimising the role of MRI in their article misses the central role it plays in providing a diagnosis for every child with CP.

Bhorat *et al.*[1] are correct to note that neither MRI scans nor the radiologists reporting them can determine the quality of perinatal patient care or determine any priming factors that predispose to or potentiate HII. The radiologist cannot determine whether anyone is responsible or negligent, just by looking at a scan. However, unlike reliance on clinical notes recorded by someone else, and unlike MRI performed in the acute period, delayed MRI scans can show with a high degree of certainty any brain injury consistent with a hypoxic ischaemic insult that occurred in a brain of term maturity. This becomes the starting point of any litigation in a child with CP. The courts are there to determine whether the care preceding, during and after delivery of the child in question was adequate, whether it be in the evaluation and mitigation of any priming risks, in monitoring of and response to any potential fetal distress, or in postnatal care to minimise any brain injury.

All the unfortunate children who already have CP deserve an MRI scan and expert interpretation by a neuroradiologist for a diagnosis of the cause. We radiologists accept and support the concept of multifactorial causation in CP. We fully support a holistic approach to the problem of CP in SA, as espoused in this obstetric review by Bhorat *et al.*[1] Rather than berating litigation, which is often the only time HII is mentioned to the parents and the first time the potential for negligent management is considered, why not acknowledge the nationwide failures in obstetric services due to systemic negligence by healthcare workers in a dysfunctional health system? SA obstetric practice is not in question. The professional standards of SA gynaecologists and obstetricians compare favourably with international practice. Unfortunately, they are bearing the consequences of an aberration in terms of negligent fetal and maternal care during the first and second stages of labour.[5] Bhorat *et al.*[1] should rather pressurise the state to give urgent attention to these issues, to minimise the number of children developing CP going forward.

The three goals of medical malpractice systems have been highlighted as follows by Frees and Gao,[6] from whom we quote verbatim:

- **Prevention.** The prevention of medical injuries and the promotion of patient safety are paramount goals of healthcare policy. The prospect of liability in damages acts as an incentive to act with reasonable care.
- **Compensation.** Compensation of injured patients is a core function of the law regarding medical malpractice and medical injuries.
- **Accountability.** Injured patients [and their families] want to know what went wrong, who was responsible for it, and what efforts are being made to prevent future repetitions. They also want to receive an admission of fault and an apology.

Without litigation, none of the above would have been achieved for many children with CP. MRI as the first step in medicolegal proceedings remains key, and the findings on delayed MRI remain highly relevant in a setting where other information is severely lacking and unreliable.

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Bhorat *et al.* respond: We thank Profs Andronikou and Lotz for their interest in our article and their letter to the Editor. At the outset we would like to state our displeasure at the words 'Countering obstetricians' deflections' in the title of the letter. Our article was neither a deflection nor a ruse, but an honest attempt to assist clinicians in understanding obstetric causation in the context of CP litigation in SA. Andronikou and Lotz state that we did not acknowledge service delivery issues. This is not true – we cited Bothma and Buchmann,[1] and further discouraged defending healthcare workers who do not follow standard practice in our labour wards. We also did not, as Andronikou and Lotz suggest, understate the proportion of cerebral palsy related to intrapartum hypoxia-ischaemia. In fact, we wrote that this proportion was likely to be 'substantially higher than in high-income countries', also citing Mahlaba *et al.*[2] We believe, however, that Andronikou and Lotz's use of the very high estimate from Elsingergy *et al.*[3] is not valid, as that source uses a medicolegal case series and is therefore prone to selection bias.

We accept that late MRI done years after the event is essential in making the neurological diagnosis in most patients with CP in SA. However, we differ from Andronikou and Lotz in believing that late MRI cannot with certainty indicate clinical causation or time a hypoxic-ischaemic event to the intrapartum period. On timing, all that can be said, using Andronikou and Lotz's own words, is that these images are 'consistent with a hypoxic ischaemic insult that occurred in a brain of term maturity'. This is the general opinion in the neuroradiological literature cited in our article.[4,5] Clearly, antepartum events, fetal priming, neonatal condition and placental histopathology also need to be considered when holistically and scientifically considering causality in neonatal encephalopathy and CP. The use of isolated radiological 'confirmation' of hypoxic-ischaemic injury, as seems so often the practice in medicolegal cases in SA courts, does not necessarily implicate the intrapartum period.

We cannot accept that the diagnosis of CP for medicolegal proceedings is dependent on MRI as the 'first step', as Andronikou and Lotz attempt to explain. We insist that the diagnosis, which includes assessment of causation, can only be based first on clinical criteria as laid out in our article (or on as many criteria as can be

determined given the admittedly frequent lack of information or clinical records). MRI should follow and not precede a neurological examination and determination, using available records, of the presence of neonatal encephalopathy. We stand by the 10 criteria we presented in our article, and also by the accompanying algorithm. We reject the unsubstantiated suggestion of Andronikou and Lotz that the authors of our article, including a paediatric neurologist, suffer from a 'lack of experience in reviewing the poor medical records kept in state practice outside of which they work'. We are well aware of the many deficiencies in the public health service, including poor record keeping. The 10 criteria we recommend serve as a guide not only for litigation but also for better practice, for example a recent recommendation from the South African Society of Obstetricians and Gynaecologists (SASOG) for clinically indicated placental histopathology, and for measurement of cord blood pH at all births, which can serve as rule-in or rule-out for intrapartum hypoxia-ischaemia.

Andronikou and Lotz effectively concede that they cannot, as radiologists commenting on MRI, determine the antepartum and perinatal patient care or priming factors that predispose to neonatal encephalopathy and CP. Thus, the need for an obstetric clinical perspective. They write, not incorrectly, that prevention, compensation and accountability are three goals of medical malpractice systems. But this must be seen in the context of a developing country with an overburdened and under-resourced public healthcare system in which we cannot fairly judge clinical incidents using high-income country healthcare benchmarks and litigation. The obvious solution is to improve our healthcare system. Andronikou and Lotz should agree that the payment of multi-millions to a relative few following litigation is damaging to an already struggling public health service, creating a vicious cycle of worsening care and increasing litigation. We invite interested radiologists not to counter us but rather to join us in finding solutions to what is becoming an unacceptable national medicolegal crisis. In this regard, SASOG has recommended to the South African Law Reform Commission the establishment of an independent medical regulatory body (IMRB) that will comprise all disciplines, including radiology, involved in CP litigation. This body may set up tribunals to discuss cases of possibly intrapartum-related CP in a scientific forum using all available data to establish causation. Where negligence and liability are found, compensation through a mediation and/or arbitration process should follow. If no liability is found, the matter is closed. This should of course not limit the constitutional right of families to continue with civil litigation if they choose. Determination of accountability for alleged intrapartum-related CP should, in most cases, move out of the courts and into medical tribunals overseen by an IMRB if we are to interrupt the vicious cycle of substandard public sector care and damaging litigation in high-quantum CP claims.

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