

Discard the placenta at your peril, pathologist warns doctors



Doctors who discard the placenta after a newly born infant dies or is permanently impaired – seemingly during the birth process – could potentially be throwing away their last chance of a legitimate defence should the angry and grieving parents decide to sue.

Prof. Colleen Wright, an anatomical pathologist at the National Health Laboratory Services in Port Elizabeth, had specialists and GPs paying close attention at the South African Medical Association's Millennium



Prof. Colleen Wright, anatomical pathologist.

Development Goals conference this August. Reminding them that the oft-discarded afterbirth placenta was a crucially important fetal organ and therefore a 'map' of what had happened to the infant *in utero*, she said it was a priceless tool in determining the pathophysiology of an adverse pregnancy outcome. The placenta could uniquely show whether injuries were primarily related to labour/delivery or to an *in utero* insult long before the onset of labour. Determining the timing of such injuries was invaluable in the medicolegal assessment of cases. Without placental pathology, the attending doctor could be left high and dry in responding to litigation.

Wright said numerous studies had shown that babies who had 'an event' during the course of their mother's pregnancy went into labour already compromised. The placenta was normally 'relegated to the sink'. Her experience at Tygerberg Hospital in the year 2000 was that she saw just six placentas from an 'excellent' and extremely busy neonatal unit that year. Through her advocacy and that of her colleagues, this improved to 848 placentas a year (still just 15% of deliveries) by 2004/5, when there were only 30 cases of clinically suspected intrapartum hypoxia. On examination, the placenta was normal

in only one of these 30 cases, the remainder all showing some degree of pathology unsuspected by the attending clinician. Existing data from developing countries showed that 90% of neonatal injury occurred before the onset of labour, while the only existing comparative data (from developed countries) showed the percentage of injury due to intrapartum hypoxia to be just 6 - 10% (estimated to be higher in developing countries). Wright said that the commonest causes of intrapartum hypoxia were placental abruption and cord accidents (i.e. cord prolapse), both sudden and unpredictable events. Where she was now working (Dora Nginza Regional Hospital in Port Elizabeth), nearly every baby delivered with a low Apgar score was labelled as having birth hypoxia. 'If that's seen by a lawyer, it's a field day because you then have to prove it was not,' she warned.

US insurance companies insist on placental pathology

Wright cited some insurance companies in the USA as suggesting that every placenta delivered by their clients be sent for pathology, as some adverse outcomes may not be immediately

apparent at birth – a highly pragmatic and effective means of saving on litigation payouts. More practical in our setting, however, was for each hospital to agree on guidelines for submission of placentas appropriate for their setting and budget, based on internationally published and accepted guidelines. She also outlined four essential criteria needed to define an acute intrapartum event as being sufficient to cause cerebral palsy, one of these being the exclusion of any other identifiable cause, saying that this further pointed to a need for placental pathology. Placentas with decreased reserve would still function adequately, yet be unable to cope with the stress of a normal delivery, she emphasised. Among the reasons the placenta was ignored was the limited exposure students and doctors had to it during training, difficulties in terminology, interpreting findings and examination, and the dearth of qualitative studies. Another reason it had become ‘the outcast’ among pathology specimens was that ‘there are simply too many of them’. She said that some indications for placental examination would not be obvious at the time of delivery. Not only did determining the pathophysiology of an adverse pregnancy outcome help determine the timing of events to assist in a medicolegal assessment, but it also contributed significantly to the management of subsequent pregnancies and the management of the newborn child in the acute and longer term. It also helped define health policies and the allocation of resources.

Seventy-three per cent of US obstetricians sued more than once

Driving home her message, she cited a 1985 National Institutes of Health report putting the number of mentally retarded children in the USA at 850 000, cerebral-palsied young children at 750 000 (with 10% of all schoolchildren disabled), and 42 million neurological, communicative disorders. In 2003, a full 73% of obstetricians in the USA reported being sued at least once – most often related to cerebral palsy. The median award for ‘medical negligence in childbirth cases’ was \$2.3 million (just over R23 million). Wright proposed that in South Africa (SA), where litigation is on a sharp upward curve given the predisposing conditions for adverse events in public hospitals, ‘we pay more attention to our own placentas and their role in contributing to the statistics we have in our neonatal and perinatal deaths. If we do, I think you’ll find the placenta has a great deal to contribute to reducing the shocking statistics we have.’

The SA *Saving Babies* 2010 - 2011 report puts the early perinatal mortality rate as 21/1 000 live births, with the majority of these deaths occurring in the 1 000 - 1 499 g weight category. Deaths due to intrapartum asphyxia are reported as being linked to healthcare provider-associated avoidable factors in 44% of cases. The top five health worker-related factors were: (i) fetal distress monitored but not

detected; (ii) fetal distress not monitored and not detected; (iii) no intervention for prolonged second stage of labour; (iv) delay in referring the patient; and (v) delay in calling for expert assistance.

Wright told her doctor audience that the only time a placenta would not help them was if they were negligent. The ‘ideal’ would be to identify problems prior to delivery. Here the Doppler ultrasound scan may prove to be of great assistance in avoiding having to tell parents ‘sorry, you have a dead baby, but we can point to the cause’. She was involved in a large international study trying to correlate data on Doppler ultrasound use v. placental pathology, and added: ‘In SA we are in the early stages of identifying our problems, but there is much more we can do.’

‘I believe when a baby is compromised, everybody is the loser – the baby, the obstetrician, the neonatologist and the parents. Sometimes the best you can do is sit down with the parents and say we looked at everything we could, there was a natural cause for it. If you don’t have an answer for them, they will take you to court – they’re angry. But if you can truly say it was nobody’s fault, that nature can be cruel, they won’t sue,’ she said.

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New editor for SA Journal of Child Health



Dr John Pettifor, new editor of the *SAJCH*, was until his retirement 4 years ago head of the Department of Paediatrics at Chris Hani Baragwanath Hospital and the University of the Witwatersrand, Johannesburg, and director of the Medical Research Council (MRC) Mineral Metabolism Research Unit and the Birth to Twenty longitudinal study. He is a National Research Foundation A-rated scientist, serves on the editorial boards of a number of international bone and nutrition journals, and has received national and international research awards.

Pettifor qualified as a doctor from the University of the Witwatersrand in 1968 and then specialised in paediatrics, which he completed in 1974. In 1978/9 he spent a year as a clinical research fellow at the Shriners Hospital in Montreal with Dr Francis Glorieux studying paediatric bone diseases, the first of many to have honed their skills in paediatric metabolic bone disease at that institution. On his return to South Africa (SA) he established the Mineral Metabolism Research Unit and was appointed its director by the SA MRC in 1985, a position he held until his retirement. In 1981 he obtained the PhD (Med) for studies into the role of low dietary calcium intake in the pathogenesis of rickets in children in rural areas of SA.

Pettifor’s major research interests have focused on metabolic bone diseases in children, and in particular the roles of vitamin D and dietary calcium intake in the pathogenesis of rickets. He is currently involved in a longitudinal study of ethnic differences in bone mass in children and the factors influencing bone growth and acquisition during puberty. He has over 200 publications in accredited journals, has written 30 chapters in books, and is co-editor of the only book published internationally on paediatric bone diseases.

Pettifor is an Emeritus Professor at the University of the Witwatersrand, has been appointed an Honorary Professorial Researcher in the Department of Paediatrics, and is a member of the Developmental Pathways for Health Research Unit. He is also director of the Carnegie Clinician PhD Fellowship Programme in the Wits Faculty of Health Sciences.