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ORIGINAL ARTICLE

Neonatal encephalopathy in New Zealand: Demographics and clinical outcome

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on behalf of the Neonatal Encephalopathy Working Group of the PMMRC

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Aim: To establish the incidence of moderate to severe neonatal encephalopathy (NE) in term infants from New Zealand and to document demographic characteristics and neonatal outcomes.

Methods: Cases were reported monthly via the New Zealand Paediatric Surveillance Unit (NZPSU). Data were collected from paediatricians for neonatal items and lead maternity carers for pregnancy and birth details. Term neonatal deaths in the Perinatal and Maternal Mortality Review Committee dataset that were because of hypoxia and/or neonatal deaths from hypoxic ischaemic encephalopathy were added to the cases identified via the NZPSU, if they had not previously been ascertained.

Results: For the period January 2010 to December 2012, there were 227 cases, equivalent to a rate of 1.30/1000 term births (95% CI 1.14–1.48). Rates of NE were high in babies of Pacific and Indian mothers but only reached statistical significance for the comparison between Pacific and NZ European. There was also a significant increase in NE rates with increasing deprivation.

Resuscitation at birth was initiated for 209 (92.1%) infants with NE. Mechanical ventilation was required, following neonatal unit admission, in 171 (75.3%) infants. Anticonvulsants were used in 157 (69.2%) infants with phenobarbitone (65.6%), phenytoin (14.5%) and benzodiazapines (21.1%), the most common. Cooling was induced in 168 infants (74%) with 145 (86.3%) reported as commenced within a 6-h window.

Conclusions: The rate of NE in New Zealand is consistent with reported international rates. Establishing antecedent factors for NE is an important part of improving care, which may inform strategic efforts to decrease rates of NE.

Key words: international child health; neonatology; neonatal encephalopathy.

What is already known on this topic

1 Neonatal encephalopathy is associated with an increased risk of neurodevelopmental impairment.
2 Therapeutic hypothermia is an established intervention that improves outcome in infants affected by NE.
3 Infants with NE will frequently require respiratory support and anticonvulsant use during their neonatal course.

What this paper adds

1 This paper reports a national rate, using strong ascertainment methods, for moderate to severe NE in New Zealand term infants, which provides an important benchmark and will be used to judge the effects of interventions aimed at reducing NE rates.
2 Analysis of demographic factors reveals a statistically significant increase in rates of NE for Pacific compared with NZ European mothers.
3 A significant increase in the rates of NE was observed with increasing deprivation.

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Perinatal asphyxia contributes to stillbirth and neonatal death[1,2] in addition to causing neurodevelopmental handicap. Approximately 30% of cerebral palsy cases occur in survivors of neonatal encephalopathy (NE),[3] and long-term morbidities including cognitive dysfunction and memory impairments are increasingly being recognised.[4–7] Despite recognition of these major adverse effects, a lack of knowledge about the antecedents of NE has been highlighted as a factor impairing progress in prevention so prompting a call for further research in this area.[8]

Although cohorts of infants with NE have been previously reported, these studies may not be informative of the current situation in New Zealand for a number of reasons. Firstly, some were studied in an older era, before intervention with hypothermia and imaging with MRI became established practices.[9,10]

Secondly, several studies, including those from New Zealand,[11,12] collected data from a single tertiary centre, which could be associated with difficulties establishing an accurate denominator and may not reflect the wider maternity system. Indeed, the New Zealand system is diverse with respect to a variety of maternity providers and geographical challenges. Thirdly, published studies based on regional populations have used a variety of entry criteria. In the Western Australia cohort,[13,14] inclusion criteria were either seizures alone or two of the following for greater than 24 h: abnormal consciousness, difficulty maintaining respiration presumed central, difficulty feeding presumed central in origin, abnormal tone and reflexes. In contrast, a cohort from Sweden was based on an Apgar score of 3 or less at 1 min or 6 or less at 5 min,[15] which may not correlate with the presence of encephalopathy.

The Neonatal Encephalopathy Working Group of the Perinatal Mortality and Morbidity Review Committee (PMMRC) was established in 2007 to review national New Zealand data on NE and recommend ways to use these data to improve services and outcomes for babies. A key task was to establish a national dataset for NE in New Zealand, which covered babies delivered by both self-employed lead maternity carers (LMCs) and hospital-based services so reflecting the local model of care with most low-risk women being cared for by self-employed midwives.

The aim of this study was to establish the incidence of NE and document the demographics, peripartum events and neonatal outcomes of term infants from New Zealand with moderate or severe NE. A further study will review intrapartum care, contributory factors and potential preventability.

Methods

For the purposes of case definition, NE was defined as a clinically defined syndrome of disturbed neurological function within the first week of life in the term (≥37 weeks) infant, manifested by difficulty in initiating and maintaining respiration, depression of tone and reflexes, subnormal level of consciousness and often seizures.[16]

Cases were identified and reported by paediatricians via the New Zealand Paediatric Surveillance Unit (NZPSU)[17] methodology. Essentially, New Zealand paediatricians were contacted on a monthly basis to report cases of selected conditions; if they identified that they had seen a case of NE, they were sent a questionnaire to collect data on the case. Once a case was reported by the paediatrician, the national coordination service of the PMMRC contacted LMCs to obtain further pregnancy and delivery details. The collected data included ethnicity, socioeconomic status and maternal age in addition to birthweight and gestation. The data collection process is described in detail elsewhere (fifth report of the PMMRC).[18] It was intended to limit the dataset to moderate and severe (Sarnat stages 2 and 3) NE only and not include mild (stage 1) NE,[19] so a guide to grading was included in the clinician information sheet and baby data collection form. To ensure full case ascertainment for moderate to severe NE, term neonatal deaths in the PMMRC dataset that were designated as because of hypoxia (PSANZ classification PDC7) and/or neonatal death because of hypoxic ischaemic encephalopathy[20] were added to the cases if they had not already been identified via NZPSU.

Denominator data were obtained from the birth registration dataset of New Zealand collated by Births Deaths and Marriages. For calculation of rates, the denominator set was restricted to births at term (as is the numerator). Maternal ethnicity was self-identified and is presented using a prioritised system for the New Zealand health sector.[21] Deprivation data were calculated from NZdep 2006[22] and status divided into quintiles.

Cord gases were defined as abnormal if pH below 7.0 or base deficit greater than −12[23] or lactate greater than 7. Data are presented as mean and standard deviation or median and range as appropriate. Statistical analysis used chi square for trend, chi square for frequency comparison with $P < 0.05$ taken as significant.

Multiregion ethics committee approval was granted for the data collection, including use of the NZPSU infrastructure (MEC/09/44/EXP).

Results

For the 3-year period, January 2010 to December 2012, there were 227 cases reported using the surveillance system described, which is equivalent to a rate of 1.19/1000 births (95% CI 1.05–1.36). As the case definition was limited to term births, this rate was equal to 1.30/1000 births at term (≥37 weeks) (95% CI 1.14–1.48).

Analysis of demographic characteristics revealed high rates of NE in babies of Pacific and Indian mothers, but this only reached statistical significance for the comparison between Pacific and NZ European mothers (Table 1).

The rate of NE varied by gestational age and birthweight (Table 2) with a significantly higher rate at 37 compared with 40 weeks and at birthweight <2500 g compared with 2500–4999 g. As the reasons for birth at 37-week gestation could predispose to NE, factors such as growth restriction were examined. However, rate of customised small for gestational age at 37 weeks was not found to be higher than at later gestations. No significant association was detected between the sex of the baby and the risk of NE (Table 2).

No significant association was seen between maternal age and NE (Table 3), but there was a significant increase in the rates of NE with increasing deprivation ($P = 0.005$).

Overall, 180 (79.3%) of the infants reported to have NE had an Apgar score below 5 at 1 min, and 177 (78.0%) had an Apgar score below 7 at 5 min. Cord gas data were abnormal in 143 (63%), unavailable and presumed not to have been taken in 47 (20.7%) and normal in 37 (16.3%) of NE cases. Among cases without cord gases, one half (24/47) had evidence of poor status after birth with Apgar scores below 7 at 5 min.

Resuscitation at birth was initiated in 209 (92.1%) of the infants with NE. In three infants, this was oxygen alone; a further 136 (59.9%) required intubation and positive pressure ventilation; cardiac massage was provided in 94 (32.2%), and adrenaline was administered in 45 (23.7%).

Following admission to the neonatal unit, mechanical ventilation was required in 171 (75.3%) of infants and nitric oxide in 42 (18.5%). Nine infants (4%) had positive blood cultures (all group B *Streptococcus* infections), but 89% were prescribed prophylactic antibiotics. Anticonvulsants were used in 157

Table 1 Prioritised maternal ethnicity of neonatal encephalopathy babies (2010–2012)

Ethnicity	NZ registered births ≥37 weeks, *n* (%) *n* = 174 879	NE cases, *n* (%) *n* = 227	Rate/1000 term births	95% CI
Maori	39 436 (22.6)	63 (27.8)	1.60	1.23–2.04
Pacific	18 794 (10.7)	36 (15.9)	1.92	1.34–2.65
Indian	6516 (3.7)	15 (6.6)	2.30	1.29–3.80
Other Asian	15 369 (8.8)	129 (5.3)	0.78	0.40–1.36
Other including unknown	15 629 (8.9)	17 (7.5)	1.09	0.63–1.74
NZ European	79 135 (45.3)	84 (37.0)	1.06	0.85–1.31

Table 2 Neonatal encephalopathy rate (per 1000 term births) by gestation, sex, birthweight and plurality 2010–2012

	NZ registered births ≥37 weeks *n* = 174 879		NE cases *n* = 227		Rate (/1000 term births)	
	n	%	*n*	%	/1000	95% CI
Gestation at birth (weeks)						
37	11 560	6.6	28	12.3	2.42	1.61–3.50
38	29 020	16.6	39	17.2	1.34	0.96–1.84
39	48 236	27.6	54	23.8	1.12	0.84–1.46
40	52 434	30.0	54	23.8	1.03	0.77–1.34
41	28 708	16.4	45	19.8	1.57	1.14–2.10
≥42	4921	2.8	7	3.1	1.42	0.57–2.93
Sex						
Male	89 331	51.1	122	53.7	1.37	1.12–1.61
Female	85 548	48.9	105	46.3	1.23	0.99–1.46
Birthweight (g)						
<2500	3364	1.9	11	4.8	3.27	1.63–5.85
2500–3999	143 034	81.8	183	80.6	1.28	1.09–1.46
4000–4499	23 534	13.5	22	9.7	0.93	0.59–1.42
≥4500	4889	2.8	11	4.8	2.25	1.12–4.03
Plurality						
Singleton	172 583	98.7	222	97.8	1.29	1.12–1.46
Twins	2296	1.3	5	2.2	2.18	0.71–5.08

(69.2%) of infants with phenobarbitone (65.6%), phenytoin (14.5%) and benzodiazapines (21.1%), the most common treatment options. The choice of drugs was not dependent on cooling status.

One hundred and sixty eight (74%) of infants with NE had induced cooling, and of these, 145 (86.3%) were reported to have cooling commenced within the 6-h window recommended for maximum benefit. Use of induced cooling did not vary by NE severity with 119 (75.8%) of the moderate and 49 (70%) of the severe cases receiving cooling. However, the age at which babies died was linked to whether or not cooling was performed with 11/21 deaths in the non-cooled group occurring before 24 h of age. In 7 of these 11, birth was in a tertiary unit suggesting that they were considered unlikely to have benefited from cooling.

Of the surviving infants, 118/179 (66%) underwent MRI to assist in prognostication. In 70 (39%), the scan was reported as normal or mildly abnormal, 44 (25%) were reported as moderately or severely abnormal and 4 (2%) the result was unknown. Examination on discharge was reported in 169/179 (94%) with 86 (48%) reported as normal, a further 53 (30%) mild to moderate abnormality and nine (5%) severe abnormality. Twelve infants (7%) were reported not to have been examined, and in a further nine (5%), examination findings were unknown.

Seventy-one percent of surviving infants were discharged home, and the remainder were generally discharged to a lower level unit or postnatal facility, which may account for some of the missing discharge examination data. At the time of discharge or transfer, 141 (78.8%) did not require support with feeding. Data was not collected on time to full feed for the remainder. Ongoing respiratory support at time of discharge or transfer included suctioning in four (2%) babies, oxygen in five babies (3%) and both oxygen and suctioning in three babies (2%). In addition, 16 infants (9%) were still receiving anticonvulsants. The reporting indicated that ongoing support services were involved post discharge in 74%, most often for neurodevelopmental therapy and home care.

Discussion

We have reported a national cohort of infants with NE presenting over a 3-year period in New Zealand. The incidence of

Table 3 Neonatal encephalopathy rates (per 1000 term births) by maternal age and deprivation quintile (NZDep2006) 2010–2012

	NZ registered births ≥ 37 weeks $n = 174\,879$		NE cases $n = 227$		Rate (/1000 term births)	
	n	%	*n*	%	/1000	95% CI
Maternal age (years)						
<20	11 499	6.6	14	6.2	1.22	0.67–2.04
20–34	126 131	72.1	170	74.9	1.35	1.15–1.55
35–39	30 153	17.2	37	16.3	1.23	0.86–1.69
≥40	7096	4.1	6	2.6	0.85	0.31–1.84
Deprivation quintile (NZDep2006)						
1 (least deprived)	27 623	15.8	27	11.9	0.98	0.64–1.42
2	30 965	17.7	34	15.0	1.10	0.76–1.53
3	33 624	19.2	45	19.8	1.34	0.98–1.79
4	36 995	21.2	39	17.2	1.05	0.75–1.44
5 (most deprived)	44 931	25.7	81	35.7	1.80	1.43–2.24
Unknown	741	0.4	1	0.4		

moderate to severe NE was 1.3/1000 births at term. Important demographic items associated with NE were also studied, and factors such as ethnicity and deprivation should be considered in planning interventions to decrease NE.

The reported rate of NE in New Zealand is comparable with other experience from the developed world[24,25] and lower than that reported from countries with less developed health care systems.[26,27] Some authors report a decrease in rates of NE over time.[24,28] The data for New Zealand prior to the current dataset are limited. One previous study[12] identified term infants with seizures and evidence of perinatal asphyxia for the time periods: 1978–1981 and 1991; the incidence for the two cohorts was 1.9 and 1.78 per 1000 live births respectively. A second study[11] reported the incidence of moderate to severe NE presenting to a tertiary centre to be between 0.8 and 1.3/1000 live births per year. The current dataset is a major improvement on previous knowledge as it provides a robust national baseline rate not impaired by the issues of single-centre studies. Thus, these data can inform both the assessment of intervention programs and any trends occurring over time.

A strength of the current study was that it utilised the NZPSU infrastructure to contact paediatricians from the whole country. So, in addition to being a nationwide process, there should be good case ascertainment. Further, there was detailed data collection from both paediatric services and the clinician who provided maternity care, using an established process and network as used by the PMMRC for collection of perinatal data. However, in order to achieve complete case ascertainment, cases of encephalopathy that do not have asphyxia as a clear aetiology were also reported, and in some instances, more clarity about the diagnosis has become apparent with time. Hypoxia-ischemia was the predominant pathology, but the full cohort includes a small number of cases where hypoglycaemia, infection, neonatal stroke or a congenital abnormality of the central nervous system may have been associated with an NE-like illness. One other study limitation was that data collection ceased at time of hospital discharge, so ongoing health needs and neurodevelopmental follow-up status are not reported. It is possible that this could be explored in a future study utilising data linkage. However, the cohort outcomes will likely reflect what is known about follow-up following NE in a cooling era.[29]

The trend towards over-representation among Indian and Pacific mothers and under-representation of Europeans is important. This pattern is consistent with the association seen between prioritised ethnicity and term stillbirth[30] and also is consistent with other published work.[31] One possible component may be different approaches to accessing perinatal care, and further work is required to ensure appropriate timely care for this population. It is reassuring that there was no apparent increase in risk at 42 weeks, which is consistent with the absence of an increase in risk of stillbirth at 42 weeks[30] and may reflect an active policy to intervene in prolonged pregnancies in New Zealand.

Review of postnatal care indicates that 74% of the cohort was cooled, with only 86% within a 6-h window. Individual case review would be required to fully ascertain reasons for the delay or failure to provide cooling. However, in some cases, there was an issue with poor or delayed recognition,[32] and ongoing education has been important to improve recognition and ensure that cases that would benefit are not missed. Furthermore, early intervention, even if this is passive cooling prior to transport to a level 3 centre, can improve rate of initiation of cooling before 6 h of age. Another area that has been a focus of education was the use of investigations to assist prognostication. Basic prognostication is possible from severity of encephalopathy alone, but formal convalescent neurological examination and investigation with MRI[33] will considerably improve the quality of information that can be provided.

Conclusion

Establishing antecedent factors for NE is an important part of improving care. To build on this, future work will include multidisciplinary review of cases in the dataset in order to establish whether NE was potentially avoidable and what contributory

factors were present.[33] In the meantime, the information provided can be utilised by those planning strategic improvements in care with the aim of decreasing rates of NE.

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