

He matenga ohorere, he wairua uiui, wairua mutungakore

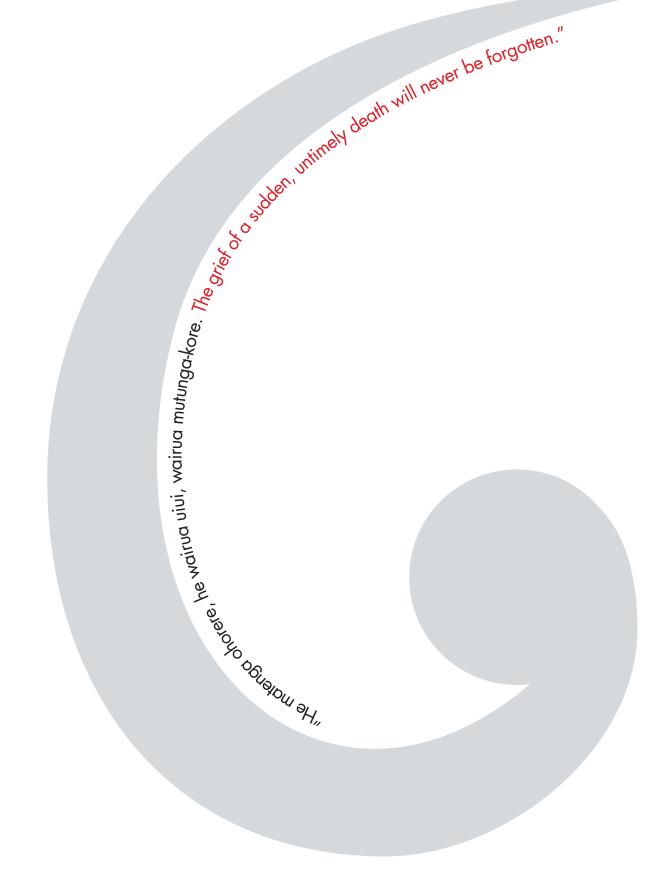


HEALTH QUALITY & SAFETY COMMISSION NEW ZEALAND Kupu Taurangi Hauora o Aotearoa



Eleventh Annual Report of the Perinatal and Maternal Mortality Review Committee Neonatal Encephalopathy 2015

(Full report available online at www.hqsc.govt.nz/our-programmes/mrc/publications-and-resources/)



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Perinatal and Maternal Mortality Review Committee

The Perinatal and Maternal Mortality Review Committee (PMMRC) members in 2017 are:

- Dr Sue Belgrave (Chair), obstetrician, Waitemata DHB
- Dr Max Berry, neonatologist, University of Otago, Wellington
- Associate Professor Sue Crengle, general practitioner, public health physician, Department of Social and Preventive Medicine, Dunedin School of Medicine, Invercargill
- Ms Alison Eddy (Deputy Chair), midwife, Christchurch
- Dr Rose Elder, obstetrician and gynaecologist, Capital & Coast DHB
- Ms Gail McIver, midwife, Counties Manukau DHB
- Ms Linda Penlington, Sands New Zealand, Wairarapa.

Neonatal Encephalopathy Working Group

The Neonatal Encephalopathy Working Group (NEWG) members in 2017 are:

- Dr Jutta van den Boom (Chair), neonatal paediatrician, Waitemata DHB
- Dr Malcolm Battin, neonatal paediatrician, Auckland DHB
- Dr Astrid Budden, obstetrician and gynaecologist, Auckland DHB
- Ms Anja Hale, neonatal nurse practitioner, Waikato DHB
- Ms Anne Jackson, neonatal nurse practitioner, Canterbury DHB
- Ms Gail McIver, midwife, Counties Manukau DHB
- Ms Suzanne Miller, midwife, Wellington
- Dr Thorsten Stanley, paediatrician, Capital & Coast DHB
- Dr Kristy Wolff, obstetrician and gynaecologist, Northland DHB.

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Executive Summary and Recommendations

Findings 2017 Report (Data 2015)

Neonatal encephalopathy

- In 2015, there were 70 babies diagnosed with moderate and severe neonatal encephalopathy (NE) reported to the national dataset. There have been 423 babies reported from 2010–2015. The rate of NE for this period is 1.24/1000 term births. Although there appears to be a downward trend in rates, there is no statistically significant trend from 2010 to 2015.
- 2. NE is associated with maternal ethnicity, socioeconomic deprivation, gestation, birthweight, and nulliparity.
- Pacific mothers are at increased risk of having a baby with NE compared to Other Asian, Other, and New Zealand European mothers. Mothers of Māori and Indian ethnicity are at increased risk of having a baby with NE compared to mothers of Other Asian and Other ethnicities.
- 4. Increasing socioeconomic deprivation is associated with increased risk of NE.
- 5. Waikato, Taranaki, and Capital & Coast district health boards (DHBs) continue to have statistically higher unadjusted rates of NE compared to the national rate.
- 6. Acute peripartum events were reported in 101 cases (24 percent) of all 423 cases in 2010– 2015, of which abruption (31 cases) and shoulder dystocia (26 cases) were the most common.
- 7. There is no apparent association between level of facility of birth and NE.
- 8. In 2015, 80 percent of babies born in New Zealand with moderate or severe NE were managed with induced cooling. A review of 22 babies with severe NE who were not cooled revealed 20 were appropriately not cooled. Review of 32 babies with moderate NE who were not cooled revealed nine babies where transfer to a tertiary unit and cooling was possible and may have been indicated.
- 9. In 2015, the proportion of those cooled who were cooled within six hours of birth as recommended for maximal benefit was 79 percent.
- 10. Eighty-one of the 423 babies with NE during 2010–2015 (19.4 percent) died in the perinatal period (<28 days). A further nine babies are known to have died after discharge from three months to five years of age.
- 11. Of survivors during 2010–2015, 28 percent had a moderately or severely abnormal MRI (21 percent of moderate and 66 percent of severe cases) and 46 percent had a normal or only mildly abnormal scan (49 percent of moderate and 30 percent of severe cases). Twenty-five percent of survivors during 2010–2015 did not have a magnetic resonance imaging (MRI) scan (30 percent of moderate and 2 percent of severe cases).

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6 Neonatal Encephalopathy 2015

6.1 Methodology

Case definition

Neonatal encephalopathy (NE): 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 (Nelson and Leviton 1991). This dataset includes Sarnat stages 2 or 3 equivalent to moderate and severe only.

The introduction of induced cooling has made the definition of NE more difficult as cooling is frequently initiated before many of the defining signs of NE have appeared, and has been used for increasingly milder cases. This is evident from data in the UK TOBY Cooling Register with a decrease in the proportion of infants with suspected clinical seizures before cooling started, an increase in Apgar scores and in first blood base excess from 2007 to 2011 (Azzopardi et al 2012). It is usual to include babies who warrant cooling in the dataset even though they may, due to the ameliorative effects of the cooling, never reach the level of morbidity consistent with moderate NE.

Although hypoxia-ischemia is the predominant pathology, reported cases of term infants with NE are included in this dataset whatever the cause. Therefore, the full cohort includes a small number of cases where NE is associated with hypoglycaemia, congenital abnormality of the central nervous system, or infection.

Case ascertainment

Cases were initially identified with the assistance of the New Zealand Paediatric Surveillance Unit and the collection of data facilitated by paediatricians, LMCs and the national coordination service of the PMMRC, as described in detail in the fifth report of the PMMRC (PMMRC 2011). Since 2012, cases have been notified by key clinicians in neonatal units and the PMMRC local coordinators.

From 2016 the Neonatal Encephalopathy Working Group (NEWG) widened the inclusion criteria for the NE cohort and will include cases from 35 weeks gestation at birth in line with international literature and practice of cooling from this gestation (American College of Obstetricians and Gynecologists 2014).

PMMRC numerator data validation

Data are regularly validated, using a standard set of queries, to eliminate duplicate records, complete missing mother or baby information, clarify DHB of residence (where this is inconsistent with the given residential address) and rectify other inconsistencies.

At the end of each year, PMMRC DHB local coordinators and key clinicians in special care and neonatal units are contacted to ensure the collection is complete.

Denominator data

Denominator data, as used elsewhere in this 11th report for the first time, are the births included in the MAT dataset collated by the Ministry of Health. For calculation of rates, the denominator set was restricted to births at term (as is the numerator).

6.2 Findings

In 2015 there were 70 cases of moderate and severe NE reported to the national dataset. There have been 423 cases reported from 2010–2015, making the NE rate for 2010–2015 1.15/1000 births (95% CI 1.04–1.26) (423/368,647 births) or 1.24/1000 term births (95% CI 1.13–1.36) (423/339,781 term births). Although there appears to be a downward trend in the NE rate (Figure 6.1), there is no significant trend (chi-squared test for trend p=0.39).

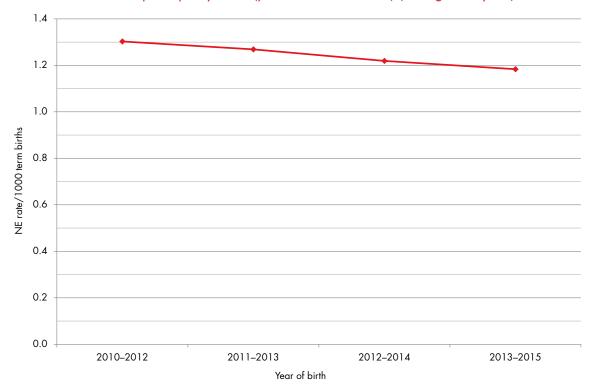


Figure 6.1: Neonatal encephalopathy rates (per 1000 term births) (rolling three-year) 2010–2015

International comparisons

In a 2013 paper, Lee et al estimated that in countries with a neonatal mortality rate <5/1000 births, such as New Zealand, the median incidence of NE associated with intrapartum events (including mild NE) was 1.60/1000 births (range 0.68–3.75/1000) for 1980 to 2013 with evidence of reduced incidence over time from some studies (Lee et al 2013). This would suggest that at 1.15/1000 births for moderate and severe NE, New Zealand is within international incidence rates. Case fatality rate among babies with severe NE was 76.8 percent (range 61.9–91.7 percent) compared to 59 percent in the New Zealand cohort.

Of survivors reported in the Lee et al paper, 26.4 percent (range 22.1–30.8 percent) developed moderate to severe neurodevelopmental impairment, and 14 percent (range 8.8–19.2 percent) developed mild neurodevelopment impairment. These outcomes largely reflect the pre-cooling era.

The UK TOBY Cooling Register reported results on 48 percent of trial participants at two years of age. Although this is limited follow-up, it was felt that these children were not systematically different from all children entered in the trial. Cerebral palsy was clinically diagnosed in 22 percent (Azzopardi et al 2012).

In 2017, the NEWG has looked at preliminary data from the B4 School Check programme in New

Zealand relating to babies reported to the NEWG in the first two years of the cohort. Details of these findings will be included in the 2018 report.

Demography and neonatal encephalopathy

In this 11th report, the PMMRC has moved to reporting using the MAT dataset as the denominator for rates. While this denominator has a number of advantages in that it is the best record of births in New Zealand in a year and it includes a wealth of maternity data, the use of the MAT dataset raises the issue of the measurement of ethnicity in different routine datasets. A comparison of Figure 6.2 with Figure 3.1 in the 10th PMMRC report ('Neonatal encephalopathy rates (per 1000 term births) by maternal prioritised ethnicity 2010–2014') illustrates this (PMMRC 2016). In the report last year, the NE rate for babies of Māori mothers was reported as 1.48/1000 term babies (95% CI 1.19–1.81), and this year it is reported as 1.32/1000 term births (95% CI 1.07–1.56). It is not certain which of these is correct. The issue of ethnicity differences in the BDM and MAT denominator datasets is discussed further in section "1.2 Methodology" and chapter 5.

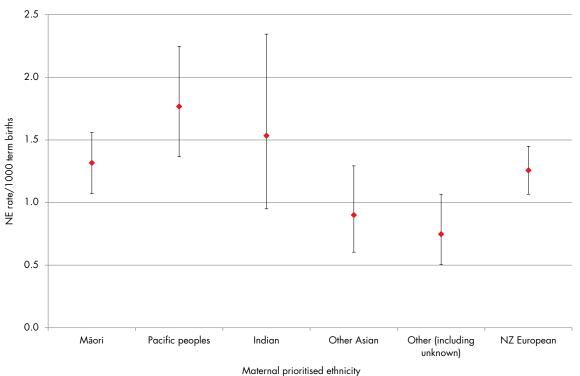
Mothers of Pacific ethnicity are at increased risk of having a baby with NE compared to Other Asian, Other, and New Zealand European mothers. Mothers of Māori and Indian ethnicity are at increased risk of having a baby with NE compared to mothers of Other Asian and Other ethnicities.

Increasing socioeconomic deprivation is associated with increased risk of NE. There has been a fairly consistent finding across the years of lower risk among mothers living in deprivation quintile 4 compared with quintiles 2, 3, and 5, although numbers are small and there is huge variation in the association by year. There is no obvious explanation for this finding.

There is no statistically significant association between maternal age and NE risk.

Maternal Pacific ethnicity remained a predictor of NE after adjusting for gestation at birth, year of birth, deprivation quintile, multiple pregnancy and maternal age (PMMRC 2016).





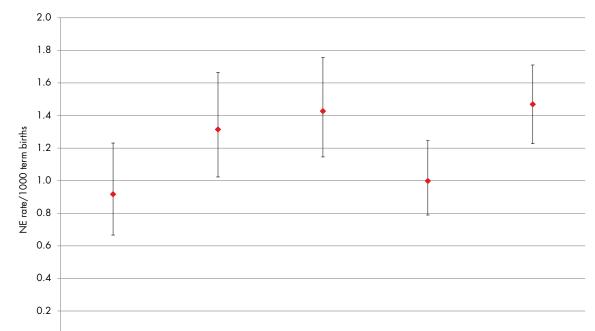


Figure 6.3: Neonatal encephalopathy rates (per 1000 term births) by deprivation quintile (with 95% Cls) 2010–2015

DHB of maternal residence

1 (least deprived)

0.0

Figure 6.4: Neonatal encephalopathy rates (per 1000 term births) by DHB of maternal residence* compared to New Zealand neonatal encephalopathy rates (with 95% CIs) 2010–2015

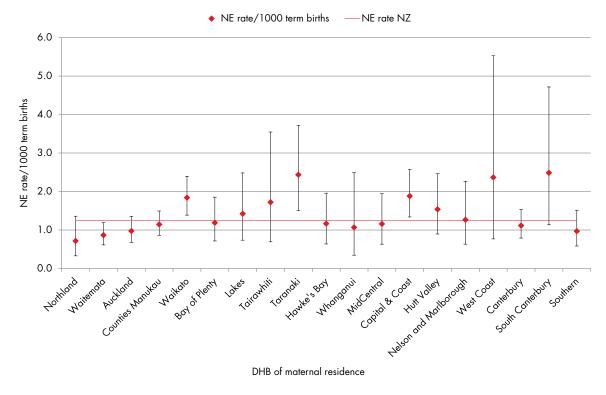
3

Deprivation quintile

5 (most deprived)

4

2



* Excludes any DHB with fewer than three cases.

Figure 6.4 includes combined data for NE by DHB of maternal residence for 2010–2015.

Waikato, Taranaki, and Capital & Coast DHBs, all previously identified as outliers, have statistically higher rates of NE than the national rate. All DHBs should be routinely identifying and undertaking multidisciplinary review of cases of moderate and severe NE. In 2017, the NEWG will be asking DHBs to report on whether, and how, they have reviewed the cases reported to the NEWG from their DHB as occurring in 2016.

Previous PMMRC Recommendation (Eighth Report (PMMRC 2014))

That all DHBs review local incident cases of neonatal encephalopathy (Sarnat stages 2 and 3). The findings of these reviews should be shared at multidisciplinary local forums and form the basis of quality improvements as appropriate.

Gestation, sex, birthweight and plurality

Table 6.1: Neonatal encephalopathy rates (per 1000 term births) by gestation, sex, birthweight, plurality, parity 2010–2015

	NZ register ≥37 we	eks	NE b			Rate term births)
	n=339,781		n=4			
	n	%	n	%	/1000	95% CI
Gestation at birth (weeks)						
37	23,044	6.8	47	11.1	2.04	1.50-2.71
38	57,781	17.0	70	16.5	1.21	0.94–1.53
39	95,379	28.1	98	23.2	1.03	0.83-1.25
40	103,048	30.3	105	24.8	1.02	0.82-1.21
41	52,561	15.5	94	22.2	1.79	1.45-2.19
≥42	7,968	2.3	9	2.1	1.13	0.52-2.14
Sex						
Male	173,772	51.1	235	55.6	1.35	1.18–1.53
Female	165,992	48.9	188	44.4	1.13	0.97-1.29
Unknown	17	0.0	-	-	-	-
Birthweight (g)						
<2,500	6,190	1.8	16	3.8	2.58	1.48-4.20
2,500–3,999	267,004	78.6	355	83.9	1.33	1.19–1.47
4,000–4,499	42,472	12.5	37	8.7	0.87	0.61-1.20
≥4,500	8,595	2.5	15	3.5	1.75	0.98-2.88
Unknown	15,520	4.6	-	-	-	-
Plurality						
Singleton	334,243	98.4	414	97.9	1.24	1.12–1.36
Multiple	4,235	1.2	9	2.1	2.13	0.97-4.03
Unknown	1,303	0.4	-	-	-	-
Parity*						
0	123,932	36.5	241	57.0	1.94	1.70-2.19
1	107,534	31.6	98	23.2	0.91	0.74-1.11
2	48,222	14.2	43	10.2	0.89	0.65-1.20
3	18,860	5.6	21	5.0	1.11	0.69–1.70
≥4	15,768	4.6	20	4.7	1.27	0.77-1.96
Unknown	25,465	7.5	-	-	-	-

* Defined after birth of the index case.

There is a significant association between gestation at birth and NE risk (Table 6.1, Figure 6.5). There is a significantly higher rate of NE among babies born at 37 and at 41 weeks than among babies born at 38–40 weeks. The risk at 42 weeks and above is difficult to estimate as numbers are small and the CIs are necessarily wide.

The higher rate of NE reported among male babies (1.35/1000 term males) compared to female babies (1.13/1000 term females) almost reaches statistical significance (p=0.069).

Babies <2500g at term were twice as likely to suffer NE than babies 2500–4000g, and almost three times as likely as babies 4000–4500g.

There is no significant increase in NE among multiple births at term compared to singleton births.



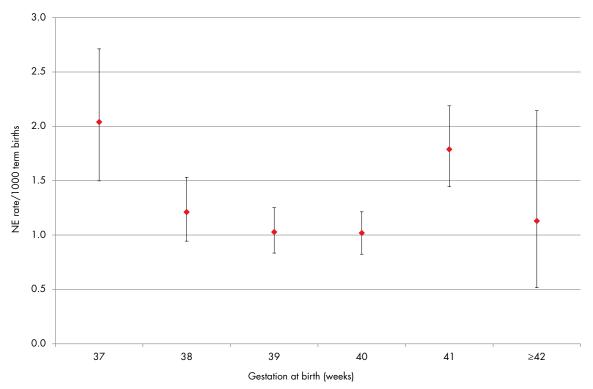
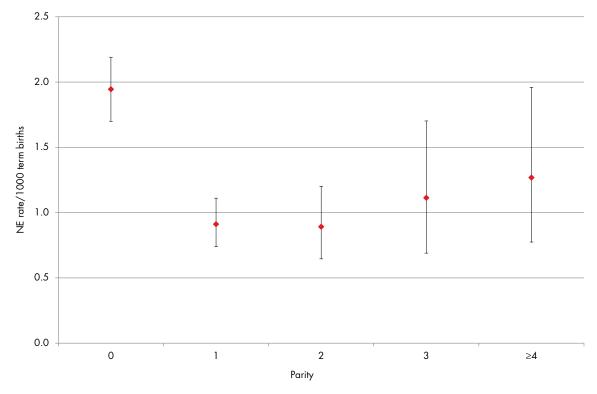


Figure 6.6 shows the association between parity and rate of NE. This is possible for the first time this year because of the use of the MAT denominator. PMMRC data are presented in the numerator and MAT data in the denominator. The rate of NE among first births is twice that among second and third births, and 1.7 times that of fourth births, but not significantly higher than fifth or later births (p=0.06) (Figure 6.6).

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Maternal smoking, BMI and gestation at first antenatal visit

Table 6.2: Maternal smoking, body mass index (BMI) and gestation at first antenatal visit among	3
neonatal encephalopathy cases 2010–2015	

	NE c	ases
	n=4	23
		%
Currently smoking		
Yes	80	18.9
No	339	80.1
Unknown	4	0.9
Maternal BMI (kg/m2)		
<18.50	3	0.7
18.50–25.49	152	35.9
25.50–30.49	123	29.1
≥30.50	121	28.6
Missing data for height and or weight	24	5.7
Gestation first antenatal visit (weeks)		
≤13	262	61.9
14–19	56	13.2
≥20	58	13.7
Unknown	47	11.1

As described in section "1.2 Methodology"', the numerator data in the PMMRC dataset for smoking and BMI are not congruent with the MAT data. To calculate rates of NE by smoking and BMI, the NE numerator data need to be merged with the MAT dataset. This is planned for the next PMMRC report.

Table 6.2 shows smoking, BMI and gestation at first antenatal visit for cases identified from 2010–2015.

Among all mothers birthing in New Zealand with data in the MAT dataset in 2015, 14.2 percent of mothers with smoking data in the MAT dataset were smokers at registration with LMC care, compared to 18.9 percent of mothers of babies diagnosed with NE, suggesting an association between smoking and NE.

Among mothers of babies diagnosed with NE, 28.6 percent had a BMI of 30.5 or greater compared to 20.6 percent among all mothers birthing in New Zealand in 2015, suggesting an increase in risk of NE with increased maternal BMI. This is consistent with increased risk of other adverse perinatal outcomes with increasing maternal BMI (Aune et al 2014; Cedergren 2004).

At least some of the association between BMI and NE, and smoking and NE, may be due to confounding factors such as socioeconomic status.

Customised birthweight, antenatal complications and maternal outcome

	NIE		Primip	oarous	AA Intoo	(> 0)	Sarnat stage					
	NEC	ases		(=1)		rous (≥2) -	Moderate		Severe			
	n=4	423	n= 2	241	n=	n=182		293	n=130			
	n	%	n	%	n	%	n	%	n	%		
Customised birthweight centiles												
Small for gestational age	77	18.2	44	18.3	33	18.1	55	18.8	22	16.9		
Appropriate for gestational age	312	73.8	184	76.3	128	70.3	210	71.7	102	78.5		
Large for gestational age	34	8.0	13	5.4	21	11.5	28	9.6	6	4.6		
Antenatal complications												
APH (≥20 weeks vaginal bleeding)	45	10.6	23	9.5	22	12.1	30	10.2	15	11.5		
Hypertension	53	12.5	38	15.8	15	8.2	41	14.0	12	9.2		
Pre-eclampsia	6	1.4	5	2.1	1	0.5	6	2.0	-	0.0		
Gestational hypertension	18	4.3	14	5.8	4	2.2	17	5.8	1	0.8		
Unspecified hypertension	29	6.9	19	7.9	10	5.5	18	6.1	11	8.5		
Maternal trauma (antenatal)*	8	1.9	5	2.1	3	1.6	4	1.4	4	3.1		
Induction of labour	102	24.1	65	27.0	37	20.3	75	25.6	27	20.8		
Augmentation of labour	158	37.4	109	45.2	49	26.9	124	42.3	34	26.2		
Epidural anaesthesia	114	27.0	83	34.4	31	17.0	89	30.4	25	19.2		
Maternal outcome												
Deceased	4	0.9	1	0.4	3	1.6	2	0.7	2	1.5		
Alive but with serious morbidity	10	2.4	3	1.2	7	3.8	5	1.7	5	3.8		
Alive and well	409	96.7	237	98.3	172	94.5	286	97.6	123	94.6		

Table 6.3: Customised birthweight centiles, antenatal complications and maternal outcome among neonatal encephalopathy cases by Sarnat stage 2010–2015

* Vehicular, violent personal injury, other.

Among babies with NE from 2010–2015, 18 percent were small by customised birthweight centile, and this is higher than expected (around 12 percent in the birthing population), suggesting SGA babies are at higher risk of NE.

The national rate of induction of labour (all births) was 24.4 percent in 2014 (28.7 percent among women having their first baby and 20.4 percent among women having subsequent babies), which is the same as that among mothers of babies diagnosed with NE (24.1 percent, 27.0 percent and 20.3 percent respectively) (Ministry of Health 2015b).

Among mothers of babies diagnosed with NE, 27.0 percent had an epidural in labour compared to a national rate in 2014 of 27.1 percent (42.4 percent of women having their first baby, and 15.3 percent of women having subsequent babies).

Over the five years of NE data collection, there have been four cases associated with maternal death and 10 with severe maternal morbidity. Of these 14 babies, seven had severe NE (50 percent) compared with 31 percent severe NE among all 423 cases of NE.

Peripartum complications and mode of birth

Acute peripartum events were reported in 101 cases (24 percent). Of these, abruption (31 cases) and shoulder dystocia (26 cases) were the most common. Other complications included amniotic fluid embolism, maternal collapse, complications at birth of the second twin, vasa praevia and drug error. Blood stained liquor was noted in 9 percent of cases and meconium in 33 percent.

Among babies diagnosed with NE, 43 percent were born by caesarean section, 31 percent by in labour caesarean section, which was most often performed for suspected fetal distress. This compares with a national caesarean section rate of 25.9 percent among all births in 2014 (Ministry of Health 2015b).

Eight babies (1.9 percent) were breech vaginal births at term, compared to 0.5 percent vaginal breech births in New Zealand in 2014 (Ministry of Health 2015b).

In 2016–2017 the NEWG has reviewed the maternity and early neonatal care of 48 babies born with NE in 2013–2015 who had an acute peripartum event. The findings of these reviews will be reported in the 12th report of the PMMRC in 2018.

Table 6.4: Peripartum complications and mode of birth among neonatal encephalopathy cases 2010–2015

Cord prolapse174.0Abruption317.3Uterine rupture92.1Shoulder dystocia266.1Breech complication92.1Other complication92.1Ispor92.1Blood stained368.5Thick meconium8921.0Thin meconium5312.5		 Total NE c	ases
Actor peripartum events 101 23.9 Cord prolopse 17 4.0 Abruption 31 7.3 Uterine rupture 9 2.1 Shoulder dystocia 26 6.1 Breach complication 9 2.1 Other complication 9 2.1 Uterine rupture 9 2.1 Blood stained 36 8.5 Thick meconium 89 21.0 Thin meconium 89 21.0 Mode of stinh 89 21.0 Normal vaginal birth 171 40.4 Operative vaginal birth 171 40.4 Operative vaginal birth 171 40.4 Vaginal breach birth 25 5.9 Ventouse 35 8.3 Unknown 2 5.3 Vaginal breach birth 8 1.9 Ceesoreen section birth 8 1.9 Suspected fietal distress 28 6.6 Faileour emergency		n=423	3
Cord prolapse174.0Abruption317.3Uterine rupture92.1Shoulder dystocia266.1Breach complication92.1Other complication92.1Uterine rupture92.1Blood stained368.5Thir meconium368.5Thir meconium3610Mode of birth17140.4Operative vaginal birth17140.4Operative vaginal birth17140.4Operative vaginal birth255.9Ventouse358.3Unknown20.5Veginal breech birth81.1Caesareen section birth18243.0Elective92.1Prelabour emergency409.5Antepartum hoemorthage/Abruption51.1In labour emergency61.4Antepartum hoemorthage/Abruption10.2Cother61.4In labour emergency402.1Antepartum hoemorthage/Abruption10.2Failure to progress/Cephalopelvic diiproportion112.2Failure to progress/Cephalopelvic diiproportion112.2Cother674.0Unknown10.21		n	%
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Mode of birth 171 40.4 Operative vaginal birth 62 14.7 Forceps 25 5.9 Ventouse 35 8.3 Unknown 2 0.5 Vaginal brech birth 8 1.9 Caesarean section birth 8 1.9 Prelabour emergency 40 9.5 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 6 1.4 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 133 31.4 Antepartum haemorrhage/Abruption 133 31.4 Suspected fetal distress 94 22.2 Failure to progress/Cephalopelvic disproportion 11 2.6 Other 17 4.0 Unknown 1 0.2	Thick meconium	89	21.0
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Unknown 2 0.5 Vaginal breech birth 8 1.9 Caesarean section birth 182 43.0 Elective 9 2.1 Prelabour emergency 40 9.5 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 6 1.4 In labour emergency 6 1.4 Suspected fetal distress 94 22.2 Failed induction 10 2.4 Suspected fetal distress 94 22.2 Failure to progress/Cephalopelvic disproportion 11 2.6 Other 11 2.6 Other 12 3.0	Forceps	25	5.9
Vaginal breech birth 8 1.9 Caesarean section birth 182 43.0 Elective 9 2.1 Prelabour emergency 40 9.5 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 6 1.4 In labour emergency 6 1.4 Suspected fetal distress 9 2.1 Suspected fetal distress 10 2.2 Antepartum haemorrhage/Abruption 13 31.4 Antepartum haemorrhage/Abruption 10 2.4 Suspected fetal distress 94 22.2 Failure to progress/Cephalopelvic disproportion 11 2.6 Other 11 2.6 1.4 Unknown 1 0.2 1.0	Ventouse	35	8.3
Coessarean section birth 182 43.0 Elective 9 2.1 Prelabour emergency 40 9.5 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 6 1.4 In labour emergency 133 31.4 Antepartum haemorrhage/Abruption 10 2.4 Other 6 1.4 2.4 Other 6 1.4 2.4 Suspected fetal distress 94 22.2 2.4 Failure to progress/Cephalopelvic disproportion 11 2.6 Other 17 4.0 3.0 Unknown 1 0.2 3.0 3.0	Unknown	2	0.5
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Prelabour emergency 40 9.5 Antepartum haemorrhage/Abruption 5 1.2 Suspected fetal distress 28 6.6 Failed induction 1 0.2 Other 6 1.4 In labour emergency 13 31.4 Antepartum haemorrhage/Abruption 10 2.4 Suspected fetal distress 94 22.2 Failure to progress/Cephalopelvic disproportion 11 2.6 Other 17 4.0 Unknown 1 0.2	Caesarean section birth	182	43.0
Antepartun haemorrhage/Abruption51.2Suspected fetal distress286.6Failed induction10.2Other61.4In labour emergency13331.4Antepartun haemorrhage/Abruption102.4Suspected fetal distress9422.2Failure to progress/Cephalopelvic disproportion112.6Other174.0Unknown10.2	Elective	9	2.1
Suspected fetal distress286.6Failed induction10.2Other61.4In labour emergency13331.4Antepartum haemorrhage/Abruption102.4Suspected fetal distress9422.2Failure to progress/Cephalopelvic disproportion112.6Other174.0Unknown10.2	Prelabour emergency	40	9.5
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Antepartum haemorrhage/Abruption102.4Suspected fetal distress9422.2Failure to progress/Cephalopelvic disproportion112.6Other174.0Unknown10.2	Other	6	1.4
Suspected fetal distress9422.2Failure to progress/Cephalopelvic disproportion112.6Other174.0Unknown10.2	In labour emergency	133	31.4
Failure to progress/Cephalopelvic disproportion112.6Other174.0Unknown10.2	Antepartum haemorrhage/Abruption	10	2.4
Other 17 4.0 Unknown 1 0.2	Suspected fetal distress	94	22.2
Unknown 1 0.2	Failure to progress/Cephalopelvic disproportion	11	2.6
	Other	17	4.0
Attempt at operative vaginal birth before caesarean 12 2.8	Unknown	1	0.2
	Attempt at operative vaginal birth before caesarean	12	2.8

Place of birth

From 2010 to 2015, there were eight babies with NE who were birthed at home as intended. This is 1.9 percent of all cases of moderate or severe NE (Table 6.12). Eleven babies diagnosed with NE (2.6 percent) birthed at home. In 2014, 3.4 percent of babies were born at home in New Zealand, 9.1 percent in a primary unit, 41 percent in a level 2 hospital and 46.4 percent in a tertiary hospital (Ministry of Health 2015b).

Previous PMMRC reports have shown that there is no association between place of birth and induced cooling among babies born in New Zealand with moderate and severe NE. More detail on intended and actual place of birth can be found in Table 6.12.

LMC and neonatal encephalopathy rates

Consideration was given in this report to including rates of NE by LMC at registration because these data are collected by the NEWG, are available in the MAT denominator dataset, and because this question has been raised nationally (Wernham et al 2016).

However, these data have not been included in this report for two reasons. Firstly, the denominator data do not accurately represent the models of maternity care available during the time period 2010–2015. For example, there is no category for the model of shared GP and DHB primary care previously available at Counties Manukau DHB. Women under primary care at DHBs who are not currently providing data to the Ministry of Health are recorded as 'no LMC care', which cannot be separated from women who were truly not registered with an LMC. Secondly, New Zealand has an integrated model of maternity care, with referral guidelines (Ministry of Health 2012a) outlining processes and guidance for consultation, referral, and transfer of responsibility for care in response to maternal need that does not facilitate division of care by individual caregiver, and so the numerator data are not fit for the purpose either.

The NEWG has previously reported on case review of cases of NE where there was no acute peripartum event (Sadler et al 2016) and are currently undertaking review of cases where there was an acute peripartum event. During this latter review, specific effort has been made to identify the responsible clinician at various intervals in the process, and to identify instances where there was delay in recognising a need for consultation and/or referral or a delay in providing consultation and/or transfer, and these data will be presented with the results of this review. The original case review did not specifically look at the potential role of the caregiver, or a group of caregivers, in the aetiology of NE, but also did not find this to be an identified contributing factor.

	24	2010 2011 2012 2013				012	24	014	20	115	T.	tal			
											2015				
	n	=82	n	=67	n	n=79		n=70		n=55		n=70		n=423	
Apgar scores															
Apgar score <3 at 1 minute	48	58.5	41	61.2	47	59.5	40	57.1	37	67.3	38	54.3	251	59.3	
Apgar score <5 at 1 minute	65	79.3	54	80.6	62	78.5	58	82.9	49	89.1	50	71.4	338	79.9	
Apgar score <7 at 1 minute	73	89.0	61	91.0	70	88.6	65	92.9	53	96.4	58	82.9	380	89.8	
Apgar score <7 at 5 minutes	61	74.4	54	80.6	62	78.5	57	81.4	43	78.2	49	70.0	326	77.1	
Apgar score <7 at 10 minutes	39	47.6	38	56.7	49	62.0	32	45.7	29	52.7	34	48.6	221	52.2	
Apgar score <9 at 10 minutes	52	63.4	52	77.6	62	78.5	52	74.3	45	81.8	47	67.1	310	73.3	
Cord blood gases: summary data															
Normal (none of pH ≤7, BE ≤–12, lactate ≥6)	12	14.6	14	20.9	11	13.9	13	18.6	7	12.7	8	11.4	65	15.4	
Abnormal (any of pH ≤7, BE ≤–12, lactate ≥6)	47	57.3	41	61.2	55	69.6	48	68.6	40	72.7	47	67.1	278	65.7	
No gases reported	23	28.0	12	17.9	13	16.5	9	12.9	8	14.5	15	21.4	80	18.9	
No gases and Apgar score <7 at 1 minute	14	17.1	8	11.9	8	10.1	6	8.6	8	14.5	6	8.6	50	11.8	
No gases and Apgar score ≥7 at 1 minute	8	9.8	4	6.0	5	6.3	3	4.3	-	-	9	12.9	29	6.9	
No gases and unknown Apgar score	1	1.2	-	-	-	-	-	-	-	-	-	-	1	0.2	

Immediate newborn wellbeing

Table 6.5: Immediate newborn wellbeing among neonatal encephalopathy babies 2010–2015

BE = base excess.

Fifty-nine percent of the babies diagnosed with moderate or severe NE from 2010 to 2015 had an Apgar score under 3 at one minute, 80 percent under 5 at one minute, 77 percent under 7 at five minutes, and 52 percent still had a score under 7 at 10 minutes. Sixty-six percent had abnormal arterial or venous cord blood gases (defined as pH of ≤7.0 and/or base excess of ≤–12mmol/l and/ or lactate of ≥6mmol/l), and a further 12 percent who had no gas result had an Apgar score of ≤6 at one minute. These data indicate the majority of babies diagnosed with moderate and severe NE have evidence of asphyxia at birth.

There was a statistically significant reduction in the proportion of babies without cord gases reported from 2010 (28%) to 2014 (15%) (chi-squared test for trend p=0.02), but the proportion without cord gases increased to 21 percent in 2015. In 2015, nine of the 15 babies who did not have a cord gas taken had an Apgar score of 8 or 9 at one minute, and therefore cord gas may not have been indicated.

The practice of collection and review of umbilical cord bloods may vary by DHB. For reference, the Auckland DHB guideline on management of umbilical cord blood results is given below.

Management of Umbilical Cord Blood Results

Management of umbilical cord lactate results

Cord lactates should be taken and processed within 10 minutes of cord clamping.

Umbilical cord lactate result	Action
Less than 6.0	Document results
6.0 or above	Send paired umbilical cord gases

Management of umbilical cord gas results

Umbilical cord gases can be analysed within one hour of birth if clamped immediately after delivery. Both umbilical cord arterial and venous gases should be analysed.

Umbilical cord gas result	Action
pH less than 7.0 or base excess less than or equal to –12 mmol/L	Call paediatrician for review.
pH 7.0–7.15 or base excess –11 to –7 mmol/L Or umbilical cord gas result not available and cord lactate greater than or equal to 6.0 mmol/L	Monitor baby for signs of neonatal encephalopathy (hypotonia, poor feeding, lethargy, weak or absent suck/gag or Moro reflex, seizures). Call paediatrician if any concerns.
pH above 7.15 <i>and</i> base excess above –7 mmol/L	Document results.

(Auckland District Health Board 2017)

Induced cooling

	20	10	20)11	20	12	20	13	20	14	20)15	То	tal
Cooling	n=82		n=67		n=79		n=70		n=55		n=70		n=423	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Yes	56	68.3	51	76.1	62	78.5	58	82.9	45	81.8	56	80.0	328	77.5
No	26	31.7	16	23.9	17	21.5	12	17.1	10	18.2	14	20.0	95	22.5
Age at cooling	ge at cooling n=56		n	n=51		n=62		n=58		=45 n=5		=56	n=328	
≤6 hours	46	82.1	39	76.5	53	85.5	47	81.0	39	86.7	44	78.6	268	81.7
>6 hours	10	17.9	8	15.7	9	14.5	11	19.0	6	13.3	11	19.6	55	16.8
Unknown time	-	-	4	7.8	-	-	-	-	-	-	1	1.8	5	1.5

Table 6.6: Induced cooling therapy among neonatal encephalopathy babies 2010–2015

Induced cooling has been shown to reduce mortality by 25 percent and neurodevelopmental disability in survivors of NE by 23 percent (Jacobs et al 2013).

In 2015, 80 percent of babies born in New Zealand with moderate or severe NE were treated with induced cooling. The proportion of those cooled who were cooled within six hours of birth as recommended for maximal benefit was 79 percent. There has been little change in this rate over the years of data collection.

In the dataset from 2011 to 2014 (four years) 54 neonates were reported as not receiving full body cooling for NE. Twenty-two of these have been diagnosed with severe NE (grade III) and 32 with moderate NE (grade II). Eighteen out of the 22 severe NE cases died, and one of the moderate group.

Upon reviewing these cases, it seemed 20 of the 22 severe case infants were appropriately not cooled, reasons including early neonatal death, withdrawal of care, disseminated infection, and one late presentation with seizures on day 2. In only two severe cases cooling could have possibly been initiated; one infant was transferred to level 3 at 12h, the other developed seizures at 4h of age with normal cord gases.

In the moderate group it was determined that 23 were appropriately not cooled, reasons ranging from late presentations, to NE not related to hypoxic ischemic encephalopathy (HIE), and one case was considered but did not proceed to cooling. Of the remaining nine infants, cooling may have been indicated, as low pH on cord gases and earlier consultation could have prompted a transfer to a tertiary unit.

This review will lead to an addition to the dataset asking for 'reason not cooled'.

Neonatal resuscitation

Table 6.7: Neonatal resuscitation and early neonatal management by Sarnat stage among neonatal encephalopathy babies 2010–2015

	NE b		Sarnat stage						
	INE D	ables	Mode	erate	Severe				
	n=4	423	n=2	293	n=1	130			
		%		%		%			
Resuscitation at birth									
Yes	387	91.5	266	90.8	121	93.1			
No	36	8.5	27	9.2	9	6.9			
Type of resuscitation at birth									
Oxygen only	5	1.2	4	1.4	1	0.8			
IPPV with mask	268	63.4	191	65.2	77	59.2			
IPPV with ETT	233	55.1	141	48.1	92	70.8			
Cardiac massage	166	39.2	88	30.0	78	60.0			
Adrenaline	74	17.5	25	8.5	49	37.7			
Respiratory and ventilation management									
Mechanical ventilation	339	80.1	223	76.1	116	89.2			
Nitric oxide	94	22.2	61	20.8	33	25.4			
Infection									
Positive blood culture	16	3.8	11	3.8	5	3.8			
Antibiotics	386	91.3	275	93.9	111	85.4			
Anticonvulsant therapy	300	70.9	201	68.6	99	76.2			
Phenobarbitone	276	65.2	179	61.1	97	74.6			
Phenytoin	82	19.4	39	13.3	43	33.1			
Benzodiazepines	102	24.1	62	21.2	40	30.8			
Other	32	7.6	23	7.8	9	6.9			

IPPV = intermittent positive pressure ventilation.

ETT = endotracheal tube.

Table 6.7 (along with Table 6.5) further illustrates the poor condition of many babies who develop NE at birth. Among the cohort of 423 NE babies from 2010–2015, 92 percent were resuscitated at birth, 39 percent required cardiac massage, 17.5 percent adrenalin, and 55 percent intubation and intermittent positive pressure ventilation.

In the dataset from 2011 to 2014 (four years), 18 neonates were reported as not receiving resuscitation at birth. Sixteen of these were reported as moderate and two as severe NE/HIE. On reviewing these cases, it seemed that in 10 cases, although there was no apparent need for resuscitation at birth, NE/HIE was recognised on clinical grounds/cord pH, and two of these neonates received body cooling. Other reasons for no resuscitation at birth were later presentation with parechovirus encephalitis (day 7), *Streptococcus* A sepsis day 2, inborn error of metabolism day 5, and four vascular events which would not have benefitted from earlier recognition and/or body cooling. One baby was retrospectively diagnosed with moderate HIE (on MRI) after developing seizures at 32h, and it remains unclear whether earlier recognition was possible.

Outcomes of babies with neonatal encephalopathy

				Sarnat	stage		
	NE b	abies	Mode	erate	Severe n=130		
	n=4	423	n=2	93			
		%		%		%	
Induced cooling							
Yes	328	77.5	236	72.0	92	28.0	
No	95	22.5	57	60.0	38	40.0	
Deceased							
Yes	82	19.4	5	6.1	77	93.9	
No	341	80.6	288	84.5	53	15.5	

Table 6.8: Use of cooling and outcomes of encephalopathy by Sarnat stage among neonatal encephalopathy babies 2010–2015

Babies with severe NE were significantly less likely to receive induced cooling (71 percent compared to 81 percent; p=0.03). This is presumably because many of the severe babies are considered too unwell for cooling and are represented among the excess mortality in this group (59 percent compared to 2 percent). Of the 80 babies with NE who died within the perinatal period, 18 (22 percent) died within the first day, 60 (74 percent) within the first three days, and 72 (89 percent) died within the first week of birth. A further eight babies died after one week but within four weeks of birth, and two babies died in the post-neonatal period (five and six weeks). As of the year ended December 2015, a further nine babies died after discharge from three months to five years of age.

Investigations and neonatal outcome by Sarnat stage (survivors)

Investigations		2010		0011		0010		0010		0014		0015		I NE	Sarnat stage		stage	
		10	2011		2012		2013		2014		2015		survivors		Moderate		Severe	
Investigations	n=	:59	n=54		n=67		n=59		n=44		n=58		n=341		n=288		n=53	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Examination on discharge/transfer																		
Normal	32	54.2	25	46.3	30	44.8	24	40.7	17	38.6	29.0	50.0	157	46.0	149	51.7	8	15.1
Mild or moderate abnormality	14	23.7	20	37.0	19	28.4	23	39.0	17	38.6	14.0	24.1	107	31.4	88	30.6	19	35.8
Severe abnormality	3	5.1	1	1.9	5	7.5	5	8.5	3	6.8	4.0	6.9	21	6.2	3	1.0	18	34.0
Not examined	1	1.7	4	7.4	7	10.4	5	8.5	2	4.5	3.0	5.2	22	6.5	19	6.6	3	5.7
Examined but finding unknown	3	5.1	1	1.9	5	7.5	2	3.4	2	4.5	2.0	3.4	15	4.4	11	3.8	4	7.5
Missing data	6	10.2	3	5.6	1	1.5	-	-	3	6.8	6.0	10.3	19	5.6	18	6.3	1	1.9
EEG investigation done at ≤3 days of life*	40	67.8	25	46.3	34	50.7	50	84.7	40	90.9	51.0	87.9	240	70.4	198	68.8	42	79.2
MRI (investigation done)	41	69.5	35	64.8	43	64.2	50	84.7	38	86.4	48.0	82.8	255	74.8	203	70.5	52	98.1
No MRI or Unknown	18	30.5	19	35.2	24	35.8	9	15.3	6	13.6	10.0	17.2	86	25.2	85	29.5	1	1.9
Results of MRI																		
Moderately/Severely abnormal	16	27.1	11	20.4	17	25.4	22	37.3	13	29.5	15.0	25.9	94	27.6	59	20.5	35	66.0
Normal or only mildly abnormal	24	40.7	23	42.6	24	35.8	27	45.8	25	56.8	33.0	56.9	156	45.7	140	48.6	16	30.2
Unknown result	1	1.7	1	1.9	2	3.0	1	1.7	-	-	-	-	5	1.5	4	1.4	1	1.9

Table 6.9: Investigations and neonatal outcome by Sarnat stage of neonatal encephalopathy survivors 2010–2015

* Typically cot-side monitoring such as BrainZ.

EEG = electroencephalogram.

MRI = magnetic resonance imaging (of the brain).

There has been an increase in the proportion of surviving babies who had an MRI investigation since collection of NE data began, from 70 percent in 2010 to 86 percent in 2014 (83 percent in 2015).

Of survivors during 2010–2015, 28 percent had a moderately or severely abnormal MRI (21 percent of moderate and 66 percent of severe cases) and 46 percent had a normal or only mildly abnormal scan (49 percent of moderate and 30 percent of severe cases). Twenty-five percent of survivors during 2010–2015 did not have an MRI (30 percent of moderate and 2 percent of severe cases).

2016 Survey of DHBs and Discharge Examination for Babies Diagnosed with Neonatal Encephalopathy

In 2016 Clinical Directors of Neonatal Intensive Care and Special Care Baby Units were asked to provide details of discharge examination for babies diagnosed with NE.

This included:

- which tool, if any, was used for the discharge examination for babies diagnosed with NE
- if a formal tool was not used for the discharge examination for babies diagnosed with NE, whether they had considered using a formal tool (such as the Dubowitz examination)
- what they thought were the barriers to using a formal tool for the discharge examination
- details of where the result of examination was documented.

All six Level 3 Units advised that they use a formal tool to assist with the discharge examination for babies diagnosed with NE, either the Dubowitz examination or a modification of this. The practice was less consistent in the Level 2 Units. The barriers identified to the use of a formal tool included training in the use of the tool and interpretation of the findings given small numbers of babies diagnosed with NE in their DHBs.

Previous Recommendation (Seventh Report (PMMRC 2013))

In cases of neonatal encephalopathy (Sarnat stages 2 and 3):

All babies with encephalopathy should undergo investigation to predict prognosis, including formal neurological examination, cerebral magnetic resonance imaging (MRI) and, if available, formal electroencephalography (EEG)

All parents of an affected child should have a formal discussion with the neonatologist/paediatrician providing care in order to review the prognosis and ongoing care of their child.

6.3 Neonatal Encephalopathy Appended Tables

Table 6.10: Neonatal encephalopathy rates (per 1000 term births) by prioritised maternal ethnicity, maternal age and deprivation quintile 2010–2015

	NZ registere ≥37 we		NE c	ases	Rate (/1000 term births)		
	n=339,	781	n=4	23			
	n	%	n	%	/1000	95% CI	
Maternal ethnicity							
Māori	84,321	24.8	111	26.2	1.32	1.07-1.56	
Pacific peoples	37,362	11.0	66	15.6	1.77	1.37-2.25	
Indian	13,689	4.0	21	5.0	1.53	0.95-2.35	
Other Asian	32,213	9.5	29	6.9	0.90	0.60-1.29	
Other (including unknown)	40,148	11.8	30	7.1	0.75	0.50-1.07	
NZ European	132,048	38.9	166	39.2	1.26	1.07-1.45	
Maternal age (years)							
<20	19,795	5.8	28	6.6	1.41	0.94-2.04	
20–34	248,554	73.2	316	74.7	1.27	1.13-1.41	
35–39	57,495	16.9	63	14.9	1.10	0.84-1.40	
≥40	13,931	4.1	16	3.8	1.15	0.66–1.87	
Unknown	6	0.0					
Deprivation quintile							
1 (least deprived)	47,980	14.1	44	10.4	0.92	0.67-1.23	
2	52,482	15.4	69	16.3	1.31	1.02–1.66	
3	62,378	18.4	89	21.0	1.43	1.15–1.76	
4	78,121	23.0	78	18.4	1.00	0.79-1.25	
5 (most deprived)	96,665	28.4	142	33.6	1.47	1.23-1.71	
Unknown	2,155	0.6	1	0.2	-	-	

Table 6.11: Neonatal encephalopathy rates (per 1000 term births) by DHB of maternal residence 2010–2015

DHB of residence	NZ registered births ≥37 weeks n=339,781	2010 n=82			2013 n=70	2014 n=55	2015 n=70	Total NE cases n=423	Rate (/1000 term births)		
	n	n	n	n	n	n	n	n	/1000	95% Cl	
Northland	12,562	2	2	2	1	1	1	9	0.72	0.33–1.36	
Waitemata	43,822	10	6	4	5	9	4	38	0.87	0.61-1.19	
Auckland	35,869	4	8	4	8	5	6	35	0.98	0.68–1.36	
Counties Manukau	47,137	14	11	14	6	5	4	54	1.15	0.86–1.49	
Waikato	29,890	14	9	9	5	6	12	55	1.84	1.39–2.40	
Bay of Plenty	16,000	3	4	2	3	1	6	19	1.19	0.71-1.85	
Lakes	8,438	2	4	2	1	1	2	12	1.42	0.73-2.48	
Tairawhiti	4,067	1	2	2	1	1	-	7	1.72	0.69–3.55	
Taranaki	8,623	2	-	6	5	4	4	21	2.44	1.51-3.72	
Hawke's Bay	12,006	2	1	3	2	3	3	14	1.17	0.64–1.96	
Whanganui	4,686	1	-	2	1	-	1	5	1.07	0.35–2.49	
MidCentral	12,099	2	1	2	3	2	4	14	1.16	0.63–1.94	
Wairarapa	2,830	1	-	-	-	1	-	2	0.71	0.09–2.55	
Capital & Coast	20,716	6	4	9	10	3	7	39	1.88	1.34–2.57	
Hutt Valley	11,048	4	4	2	5	2	-	17	1.54	0.90-2.46	
Nelson and Marlborough	8,682	-	1	2	5	1	2	11	1.27	0.63–2.27	
West Coast	2,111	-	1	2	-	2	-	5	2.37	0.77–5.53	
Canterbury	34,042	11	6	7	2	6	6	38	1.12	0.79–1.53	
South Canterbury	3,621	1	2	2	1	-	3	9	2.49	1.14-4.72	
Southern	19,574	2	1	3	6	2	5	19	0.97	0.58–1.52	
Other	1,958	-	-	-	-	-	-	-	-	-	

* Other includes Overseas, Unknown and Other.

Table 6.12: Actual and intended place of birth among neonatal encephalopathy cases 2010–2015

	NE c	ases			Actual place of birth										
Intended place of birth	n=423		Home		Birthing unit		Hospital level 1		Hospital level 2		Hospital level 3		Other		
														%	
Home	15	3.5	8	53.3	-	-	-	-	5	33.3	2	13.3	-	-	
Birthing unit	55	13.0	1	1.8	22	40.0	-	-	7	12.7	25	45.5	-	-	
Hospital level 1	22	5.2	-	-	-	-	7	31.8	3	13.6	12	54.5	-	-	
Hospital level 2	165	39.0	1	0.6	-	-	2	1.2	156	94.5	5	3.0	1	0.6	
Hospital level 3	160	37.8	1	0.6	-	-	1	0.6	1	0.6	157	98.1	-	-	
Unknown	6	1.4	-	-	-	-	-	-	2	33.3	4	66.7	-	-	
Total	423		11	2.6	22	5.2	10	2.4	174	41.1	205	48.5	1	0.2	