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FULL-LENGTH ORIGINAL RESEARCH

Epilepsia®

Status epilepticus in Auckland, New Zealand: Incidence, etiology, and outcomes

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Abstract

Objective: To determine the incidence, etiology, and outcome of status epilepticus (SE) in Auckland, New Zealand, using the latest International League Against Epilepsy (ILAE) SE semiological classification.

Methods: We prospectively identified patients presenting to the public or major private hospitals in Auckland (population = 1.61 million) between April 6, 2015 and April 5, 2016 with a seizure lasting 10 minutes or longer, with retrospective review to confirm completeness of data capture. Information was recorded in the EpiNet database.

Results: A total of 477 episodes of SE occurred in 367 patients. Fifty-one percent of patients were aged <15 years. SE with prominent motor symptoms comprised 81% of episodes (387/477). Eighty-four episodes (18%) were nonconvulsive SE. Four hundred fifty episodes occurred in 345 patients who were resident in Auckland. The age-adjusted incidence of 10-minute SE episodes and patients was 29.25 (95% confidence interval [CI] = 27.34-31.27) and 22.22 (95% CI = 20.57-23.99)/100 000/year, respectively. SE lasted 30 minutes or longer in 250 (56%) episodes; age-adjusted incidence was 15.95 (95% CI = 14.56-17.45) SE episodes/100 000/year and 12.92

(95% CI = 11.67-14.27) patients/100 000/year. Age-adjusted incidence (10-minute SE) was 25.54 (95% CI = 23.06-28.24) patients/100 000/year for males and 19.07 (95% CI = 16.91-21.46) patients/100 000/year for females. The age-adjusted incidence of 10-minute SE was higher in Māori (29.31 [95% CI = 23.52-37.14]/100 000/year) and Pacific Islanders (26.55 [95% CI = 22.05-31.99]/100 000/year) than in patients of European (19.13 [95% CI = 17.09-21.37]/100 000/year) or Asian/other descent (17.76 [95% CI = 14.73-21.38]/100 000/year). Seventeen of 367 patients in the study died within 30 days of the episode of SE; 30-day mortality was 4.6%.

Significance: In this population-based study, incidence and mortality of SE in Auckland lie in the lower range when compared to North America and Europe. For pragmatic reasons, we only included convulsive SE if episodes lasted 10 minutes or longer, although the 2015 ILAE SE classification was otherwise practical and easy to use.

KEYWORDS

epidemiology, EpiNet, incidence, status epilepticus

1 | INTRODUCTION

Apart from SUDEP (sudden unexpected death in epilepsy), status epilepticus (SE) remains the most serious manifestation of epilepsy,[1] occurring in people with known epilepsy or de novo. The incidence and causes in different populations and at different ages vary greatly, as do morbidity and mortality. The reported population incidence has ranged from 8.5 up to 61/100 000/year.[2–10]

In 2015, the International League Against Epilepsy (ILAE) proposed a new classification of SE.[11] The panel proposed a conceptual definition of SE with two operational dimensions: the time point (t1) beyond which the seizure should be regarded as "continuous seizure activity" and t2, the time of ongoing seizure activity after which there is a risk of long-term consequences. On the basis of current knowledge, SE should be diagnosed if a tonic-clonic seizure lasts 5 minutes, if a focal impaired awareness seizure lasts 10 minutes, or if an absence seizure lasts 10-15 minutes.[11] These definitions have major implications for the known incidence of SE, as previous epidemiological studies have been mostly based on a 30-minute duration of a seizure.[12] SE is now classified along four axes: (1) semiology, (2) etiology, (3) electroencephalographic (EEG) correlates, and (4) age. The primary distinction regarding semiology is between seizures with prominent motor features and those without these motor features.[11]

The EpiNet study group was established to provide a platform to undertake multicenter collaborative research studies in epilepsy.[13,14] We undertook a population-based epidemiological incidence study of SE in greater Auckland, New Zealand (population = 1.614 million),[15] using the EpiNet platform, with comprehensive data collection based on the new ILAE SE classification.[16]

We also compared population incidence using previous ILAE classifications.

Key Points

- Episodes of SE were classified according to the 2015 ILAE recommendation, except that convulsive episodes had to last 10 minutes or longer
- SE in Auckland, New Zealand has an age-adjusted incidence of 29.25 (95% CI = 27.34-31.27) patients/100 000/year, with SE defined as a seizure lasting ≥10 minutes
- When SE is defined as a seizure lasting ≥30 minutes, the age-adjusted incidence is 15.95 (95% CI = 14.56-17.45) patients/100 000/year
- An acute cause for the SE was identified for 159 patients (44%) and a remote cause for 160 patients (44%)
- SE was more common in Māori and Pacific Islanders than in those of European or Asian/other descent

2 | MATERIALS AND METHODS

2.1 | Case ascertainment

The study was conducted at the five public hospitals and the major private hospital in greater Auckland. Details regarding

the methods used to identify patients have been published previously.[17] In summary, we identified all patients older than 4 weeks who had seizures that lasted for 10 minutes or longer, or repeated seizures lasting 10 minutes or longer without recovery. This was a hospital-based study, as we assumed that all people in Auckland who have a seizure lasting 10 minutes or longer attend hospital. The greater Auckland area has distinct geographical boundaries, bound by ocean to the east and west, with sparse population density at the southern and northern boundaries, allowing for accurate estimates of population incidence. The estimated population of the greater Auckland area in June 2016 was 1.614 million people.[15]

The aim was to identify all cases prospectively. However, we performed several retrospective checks to ensure that cases had not been missed. Multiple overlapping sources of information were used to identify cases; these included presentations to the emergency departments, hospital discharge summaries, admissions to intensive care units, and intravenous antiepileptic drugs (AEDs) dispensed. At the end of the study, data regarding discharge diagnoses for patients living in Auckland were obtained from the Ministry of Health. Medical records for all patients who had seizures were reviewed by one of three research nurses, and data were entered into the Status Epilepticus form in the EpiNet database if there was evidence the patient had a seizure lasting 10 minutes or longer. Each presentation was reviewed by an adult neurologist (P.S.B.), paediatric neurologist (S.D.), or one of the senior epilepsy trainees (A.B., J.J., S.S.), and was included in the final dataset only if these physicians agreed that the case was a probable or definite seizure that had lasted at least 10 minutes. The level of confidence regarding the diagnosis and the seizure type were determined by these physicians, after reviewing all the clinical notes regarding each patient's episode of SE. The terms *definite*, *probable*, and *possible* are not formally defined in the ILAE SE classification, but were defined for this study as indicating a level of confidence of SE of at least 95% (definite), 80% (probable), or 50%-80% (possible).

The study ran from April 6, 2015 to April 5, 2016. SE was diagnosed primarily on clinical criteria. We included patients who had either SE with prominent motor symptoms or SE without prominent motor symptoms (nonconvulsive SE [NCSE]). We excluded cases of SE in neonates, repeated spasms (even if they lasted ≥10 minutes), and SE that followed anoxic brain injury due to cardiac arrest. We also excluded cases of SE that commenced outside Auckland.

We used a time period of 10 minutes to define any episode of SE, because we wanted a single time interval that could be used at all centers and by all physicians, regardless of whether SE was focal or generalized; we believe this approach is more pragmatic for epidemiological purposes.

EEG confirmation of SE was not mandatory, except in patients with NCSE. NCSE was diagnosed according to the Salzburg criteria.[18] Neuroimaging, blood tests, and lumbar punctures were performed as deemed clinically appropriate and were not required as part of this research study.

Patients were deemed to be noncompliant with their treatment regimen if:

- They or their family acknowledged that they were not taking the treatment that had been prescribed, or
- The pharmacy dispensing record showed that no drugs had been dispensed within the previous 3 months (a 3-month supply can be prescribed at any one time in New Zealand), or
- The AED levels were measured, and no drug was detected.

Patients could have both an acute and a remote etiology identified. Only one etiology in each category was reported for any patient. An acute etiology was any factor that was deemed to have provoked this specific seizure or cluster of seizures. A remote etiology was a distant event (>1 week preceding the episode of SE) that was thought to have lowered the patient's seizure threshold. Often, this was the cause of a patient's epilepsy.

For the purposes of this study, an episode of SE was considered to have finally terminated once a patient had been seizure-free for 6 hours; this 6-hour rule is not part of the ILAE SE classification, but was an arbitrary but pragmatic choice that was reached by consensus among the investigators. We therefore calculated an initial duration and a final duration of SE.[17] However, we have only considered the initial duration in this report when determining the duration of the SE and the 10-minute and 30-minute SE incidence.

If an EEG showed electrographic seizures, the status was considered to be continuing until the EEG was free of electrographic seizures, even if there were no clinical seizures.

Patients could have more than one episode of SE over the course of the study period. Each such episode was recorded separately. Incidence rates were calculated separately for SE episodes and patients.

Patients could have up to three ethnic groups recorded in the EpiNet database. If patients were recorded as belonging to more than one ethnic group, prioritized ethnicity was used to ensure that cases were not recorded more than once.

This study was approved by the Northern B New Zealand Ethics Committee (14/NTB/150/AM09) and the research governance committees of the Auckland, Waitemata, and Counties Manukau District Health Boards.

2.2 | Statistics

Incidence by diagnostic subtype, age group, sex, and ethnicity was calculated per 100 000 population with 95% confidence intervals (CIs) using the Poisson distribution. Only people

resident in Auckland were included when determining incidence. The New Zealand 2016 census data were used as the population denominator.[15] Age, sex, and ethnicity-adjusted estimates were standardized against the New Zealand population estimates from the most recent census at the time that included this information (2013).[19] Direct standardization was used to age and sex standardize prevalence to the world population for international comparability.[20] All analyses were conducted using RStudio version 3.3.2.[21]

3 | RESULTS

During the 1-year data collection period, >6300 patient presentations with possible seizures were reviewed by the research nurses for possible inclusion in the study. No cases of SE were identified at the private hospital. A record was created in the Status Epilepticus registry in EpiNet for 651 episodes of possible SE, occurring in 508 patients. After review, 477 episodes of SE were confirmed in 367 patients (see Figure 1).[17]

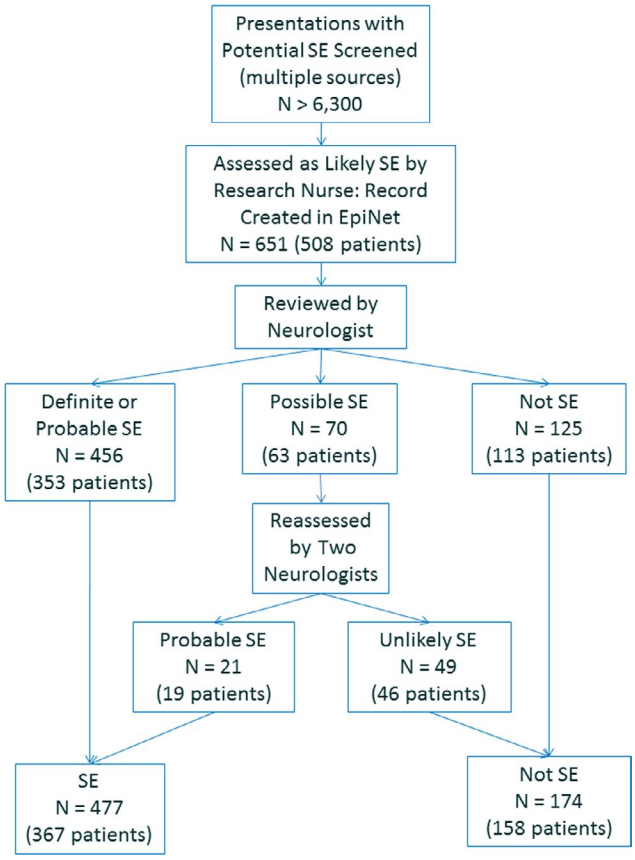

FIGURE 1 Pathway by which patients with status epilepticus were identified. Note that the number of episodes assessed sums to 651. However, the number of patients in each arm does not sum to 508, as some patients had episodes in more than one category. SE, status epilepticus

Demographic data are displayed in Table 1. Half of the patients (187, 51%) were younger than 15 years; these children accounted for 53% of SE episodes. SE was somewhat more common in males (256 episodes in 208 patients) than in females (221 episodes in 159 patients).

Sixty-three (13%) of the SE episodes developed while the patient was in hospital; these patients had been admitted following discrete seizures, or for another reason altogether, and had a seizure that lasted 10 minutes or longer while in hospital.

Sixty (16%) patients had more than one episode of SE during the year. Thirteen (3%) had four or more episodes; two children with Dravet syndrome had nine and 10 episodes of SE respectively during the study period.

Epilepsy had been previously diagnosed in people who experienced 289 (61%) of the episodes, whereas 12 (3%) of the patients were thought in retrospect to have had

TABLE 1 Demographic data

	Age, y	Patients, n (%)	Episodes, n (%)
Gender			
Male		208 (57)	256 (54)
Female		159 (43)	221 (46)
Age range			
Infants	<2	69 (19)	89 (19)
Preschoolers	2-<5	58 (16)	79 (17)
Children	5-<15	60 (16)	91 (19)
Adolescents-young adults	15-<25	32 (9)	41 (9)
Adults, nonelderly	25-<60	90 (25)	111 (23)
Older adults	60-100	58 (16)	66 (14)
Young people	<18	199 (54)	273 (57)
Ethnicity			
European/ New Zealand European		178 (49)	218 (46)
New Zealand Māori		57 (16)	67 (14)
Pacific Islanders		69 (19)	103 (22)
Asian		51 (14)	73 (15)
Other		12 (3)	16 (3)
		367 (100)	477 (100)

Note: Three patients had two or more episodes of SE and belong to two of the different age groups; however, they have only been counted once in this table, at the age of the initial presentation.
If patients belonged to more than one ethnic group, prioritized ethnicity was used to ensure that cases were not recorded more than once.

unrecognized seizures prior to the episode of SE. Seventy (19%) of the SE episodes were preceded by an unusual flurry of seizures in patients with a pre-existing epilepsy diagnosis.

3.1 | Type of SE

In 319 episodes (67%), the SE comprised a continuous seizure lasting ≥10 minutes; in 148 instances (31%), the patient had repeated discrete seizures without recovery, with the episode lasting ≥10 minutes (Table 2).

In 387 of the episodes (81%), SE occurred with prominent motor symptoms; of these, 287 (74%) were convulsive; these comprised 60% of all episodes of SE. Eighty-four episodes (18%) did not have prominent motor symptoms and were deemed NCSE.

The nature of the SE could not be identified from review of the case notes in six of the cases (1.6%).

3.2 | Duration of SE

The duration of SE could be determined in 455 episodes (95%); 255 (56%) episodes had ceased before the patient arrived at the emergency department, but there was convincing evidence that the episode had lasted ≥10 minutes.

Two hundred ninety-three (64%) episodes lasted ≥30 minutes from outset; 239 patients (53%) had SE that terminated within 1 hour of onset; 176 (39%) had SE lasting from 1 to 12 hours, 14 (3.1%) had SE lasting 12-24 hours, and 26 (5.7%) had SE lasting >24 hours.

One hundred thirty-two (28%) episodes resolved spontaneously without antiseizure drugs. Ten patients (2.7%) had more than one episode of SE during a single hospital admission; their seizures stopped for at least 6 hours, but they then had a further seizure lasting ≥10 minutes while in hospital.

3.3 | Etiology

Table 3 shows the etiology of the first episode of SE that each patient experienced during the course of the study.

Etiology differed substantially by age and the presence of pre-existing epilepsy. A remote cause for SE was identified for 160 patients (44%), a progressive cause for 19 patients (5%), and a syndromic cause for 17 patients (4.6%); all these patients had pre-existing epilepsy. An acute cause for SE was identified for 159 patients (44%). Patients could have both a remote and an acute cause (Table 3).

Febrile SE occurred in 77 children (104 episodes). Febrile seizures were the cause of the SE in 60% of the infants and in 55% of the preschool children. Almost equal numbers of boys (37) and girls (38) had febrile SE.

Noncompliance with the prescribed AEDs was considered a major factor causing SE for 38 patients (10%). Noncompliance was particularly common in adults aged 25-60 years.

SE occurred in the context of a defined epilepsy syndrome in 13 (3.6%) of the patients; all were aged <24 years; together they had 30 episodes of SE (6.3% of episodes); 14 of the episodes occurred in five children with Dravet syndrome, and six episodes occurred in three children with Lennox-Gastaut syndrome.

3.4 | Outcomes

Patients were admitted to hospital in 316 (66%) of the episodes, and 61 (13%) of the episodes resulted in admission to intensive care.

The 30-day mortality was 17 of 367 (4.6%). SE was considered to be the major cause of death in only one case; it was considered an important contributing cause in a further six cases (35%) and a minor factor in eight cases (47%). SE was considered unimportant in the deaths of two patients (12%).

3.5 | Incidence

Twenty-seven of the 477 episodes of SE during the study period occurred in 22 patients who did not live in the greater Auckland area; these patients were excluded from incidence calculations, leaving 450 episodes of SE in 345 Auckland patients over 1 year. Two hundred twenty-five of the episodes of SE lasting 10 minutes or longer occurred in children younger than 15 years, giving an incidence of 70.7 (95% CI = 61.7-80.5) episodes and 53.1 (95% CI = 45.4-61.7) incident cases per 100 000. The incidence in adults (aged 15 years or older) was 17.4 (95% CI = 15.2-19.8) episodes and 13.0 (95% CI = 11.1-15.2) incident cases per 100 000. The incidence in those aged less than 18 years was 58.7 (95% CI = 51.7-66.1) episodes and 42.2 (95% CI = 36.2-48.8) incident cases per 100 000. The incidence in older adults (age > 60 years) was 25.1 (95% CI = 19.4-31.8) episodes and 22.1 (95% CI = 16.8-28.5) incident cases per 100 000 (Table 4).

The age-adjusted incidence of SE episodes was 29.25 (95% CI = 27.34-31.27)/100 000/year. The age-adjusted incidence of SE patients (incident cases) was 22.22 (95% CI = 20.57-23.99)/100 000/year. SE lasted 30 minutes or longer in 250 episodes, giving an age-adjusted incidence of 30-minute SE episodes of 15.95 (95% CI = 14.56-17.45)/100 000/year. Episodes lasting ≥30 minutes occurred in 204 patients, giving an age-adjusted incidence of SE patients (incident cases) of 12.92 (95% CI = 11.67-14.27)/100 000/year.

3.6 | Gender

Of the 450 SE episodes in patients living in Auckland, 244 episodes (54%) occurred in 198 males, giving an age-adjusted incidence of 31.60 (95% CI = 28.83-34.59)

TABLE 2 Type of status epilepticus

	Infants (0-<2 y)	Preschoolers (2-<5 y)	Children (5-<15 y)	Adolescents (15-<25 y)	Adults (25-<60 y)	Older adults (60+ y)	Total	<18 y
	n = 89	n = 79	n = 91	n = 41	n = 111	n = 66	n = 477	n = 268
Continuous seizure	73	62	67	13	66	38	319	204
Multiple seizures without recovery	15	16	22	25	44	26	148	60
With prominent motor symptoms	74	58	68	36	97	54	387	208
Convulsive (ie, tonic-clonic) SE	52	43	50	32	74	36	287	153
Focal motor SE	15	10	9	3	21	17	75	34
Epilepsia partialis continua	1	1	2	0	9	10	23	4
Hemiclonic SE	8	5	3	0	4	5	25	16
Repeated focal seizures/other focal	6	4	4	3	8	2	27	14
Tonic SE	7	5	7	0	1	1	21	19
Other motor seizures/nature uncertain	0	0	2	1	1	0	4	2
NCSE	15	20	22	5	10	12	84	58
NCSE with coma	15	17	13	3	3	6	57	45
NCSE without coma	0	3	9	2	7	6	27	13
Generalized absence (typical)	0	1	2	0	0	0	3	3
Generalized atypical absence	0	0	0	0	1	0	1	0
Focal dyscognitive	0	2	5	1	5	2	15	8
Other focal NCSE/NCSE-NOS	0	0	2	1	1	4	8	2
Definite SE but nature of SE unknown	0	1	1	0	4	0	6	2

Abbreviations: NCSE, nonconvulsive SE; NOS, not otherwise specified; SE, status epilepticus.

SE episodes and 25.54 (95% CI = 23.06-28.24) patients/100 000/year. Two hundred six episodes (46%) occurred in 147 females, giving an age-adjusted incidence of 27.14 (95% CI = 24.53-30.00) SE episodes and 19.07 (95% CI = 16.91-21.46) patients/100 000/year. Episodes lasting ≥30 minutes occurred in 137 males (56%) and 113 females (55%) (incidence of 17.47 [95% CI = 15.44-19.71] 30-minute SE episodes/100 000 and 14.61 [95% CI = 12.72-16.71] SE incident cases/100 000, respectively).

3.7 | Ethnicity

The incidence of SE in Pacific Islanders (age-adjusted incidence = 40.00 [95% CI = 34.45-46.48] SE episodes and 26.55 [95% CI = 22.05-31.99] patients/100 000/year) and Māori (34.73 [95% CI = 28.45-43.04] SE episodes and 29.31 [95% CI = 23.52-37.14] patients/100 000/year) was notably higher than in those of European descent (23.56 [95% CI = 21.28-26.05] SE episodes and 19.13 [95% CI = 17.09-21.37] patients/100 000/year) or Asian/other descent (25.13 [95% CI = 21.51-29.35] SE episodes and 17.76 [95% CI = 14.73-21.38] patients/100 000/year; Table 5).

4 | DISCUSSION

The age-adjusted incidence of SE in Auckland, New Zealand, when SE is defined as a seizure lasting ≥10 minutes, was 29.25 (95% CI = 27.34-31.27) SE episodes and 22.22 (95% CI = 20.57-23.99) SE patients (incident cases)/100 000/year. When SE is defined as a seizure lasting ≥30 minutes, the age-adjusted incidence was 15.95 (95% CI = 14.56-17.45) SE episodes/100 000/year and 12.92 (95% CI = 11.67-14.27) SE patients (incident cases)/100 000/year.

The 30-minute SE incidence rate we report is comparable with rates reported in several studies from Europe. The age-adjusted incidence of SE in adults in northern Italy was 11.6/100 000[6]; in Bologna it was 10.7/100 000[7]; in Hesse, Germany it was 17.1/100 000.[5] The age-adjusted incidence of SE in adults and children in the French-speaking part of Switzerland was 10.3/100 000[4]; however, substantially higher rates have been reported in southern Italy (27.2/100 000[8]) and Richmond, Virginia (41/100 000). The authors considered this latter figure an underestimate of the true incidence rate, which they calculated to be 61/100 000.[2]

We found an age- and sex-adjusted incidence of SE with motor symptoms of 23.41 (95% CI = 21.72-25.22)/100 000, of which 75% were due to tonic clonic SE; the age-adjusted incidence of NCSE was only 5.52 (95% CI = 4.7-6.45)/100 000, less than one-quarter of the incidence of SE with motor symptoms.

The ILAE expert panel proposed that for tonic-clonic seizures the best evidence suggests that SE can be diagnosed if a seizure continues for >5 minutes[11]; we agree that tonic clonic seizures lasting 5 minutes should be regarded for clinical purposes as SE, and that patients need urgent treatment in this situation. However, for pragmatic purposes, we chose a time period of 10 minutes for SE for all seizure types. We considered that it would be easier to have a single time interval, so that clinicians would not need to determine the nature of the SE when deciding whether patients should be included in the study. In addition, we were not confident that all patients with convulsive seizures lasting between 5 and 10 minutes would necessarily be taken to hospital.

It has been recognized for many years that the incidence of SE is highest in children and the elderly.[3,22] The incidence of SE in children we observed is higher than reported in other Western countries, but the explanation for this is not immediately apparent. Nearly 47% of our SE cases occurred in children younger than 10 years, and most of the recurrent episodes occurred in these young children. The methodology we used to identify cases is different from that used in other studies,[23,24] and we identified many cases that did not have a discharge diagnosis of SE. We included cases with a duration of 10-30 minutes, which were excluded from previous studies. We also included patients with repeated seizures without recovery, which would be labeled acute repetitive seizures in some series. Almost one-quarter of the cases we included in this study were children with prolonged febrile seizures. SE due to febrile convulsions has been recognized as the commonest cause of convulsive SE,[23,25] although not all epidemiological studies of SE include children with febrile seizures. We used a cutoff age of 15 years for children in this study, as children below this age are admitted locally under pediatric services, including a standalone tertiary pediatric hospital, whereas older patients are admitted under adult services; however, we have also calculated the incidence for those younger than 18 years, to allow comparison with previous studies.

We found a higher incidence in the elderly than other adults, although the incidence of SE in those older than 60 years in our study is less than in some other studies.[5,6,26]

Auckland is now one of the most cosmopolitan cities in the world[27] and has a different ethnic makeup from other parts of New Zealand. However, the approach to health care is very standardized throughout the country. We have identified an increased risk of SE in Māori and Pacific Islanders; the reason for this difference requires further investigation. Similar ethnic disparity in health outcome has previously been described in New Zealand,[28–30] the cause of which is likely multifactorial, including the effects of colonization and lack of socioeconomic opportunity. The incidence of prolonged febrile seizures and convulsive SE in London was higher in those with socioeconomic deprivation.[31]

The incidence of SE in males in our study was higher than in females. The cause for this discrepancy is unclear.

TABLE 3 Etiology of status epilepticus

Etiology of SE	Infants (0-<2 y)	Preschoolers (2-<5 y)	Children (5-<15 y)	Young adults (15-<25 y)	Adults (25-<60 y)	Older adults (60-100 y)	Total	<18 y
Total SE	69	58	60	32	90	58	367	199
Acute causes								
Febrile seizure	42 (60)	32 (55)	1 (1.6)	0 (0.0)	0 (0.0)	0 (0.0)	75 (20)	75 (38)
Bacterial meningitis	2 (2.9)	0 (0.0)	1 (1.6)	0 (0.0)	0 (0.0)	0 (0.0)	3 (0.8)	3 (1.5)
Other acute CNS inflammation	0 (0.0)	0 (0.0)	2 (3.1)	0 (0.0)	1 (1.1)	0 (0.0)	3 (0.8)	2 (1.0)
Acute stroke/cerebral ischemia	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.1)	4 (6.9)	5 (1.3)	0 (0.0)
Acute head injury	3 (4.3)	1 (1.6)	0 (0.0)	1 (3.1)	1 (1.1)	1 (1.7)	7 (1.9)	3 (1.5)
AED withdrawal/noncompliance	0 (0.0)	0 (0.0)	4 (5.0)	4 (14)	27 (30)	3 (5.2)	38 (10)	6 (3.0)
Drug intoxication/withdrawal/metabolic	2 (2.9)	0 (0.0)	1 (1.6)	4 (14)	18 (20)	3 (5.2)	28 (7.7)	7 (3.5)
Total acute causes	49 (71)	33 (57)	9 (15)	9 (28)	48 (53)	11 (19)	159 (44)	96 (48)
Remote causes								
Malformation of cortical development	0 (0.0)	1 (1.6)	3 (4.8)	1 (3.1)	4 (4.4)	0 (0.0)	9 (2.4)	4 (2.0)
Neurocutaneous disorders/chromosomal	3 (4.3)	4 (6.9)	3 (4.8)	2 (3.1)	0 (0.0)	0 (0.0)	12 (3.2)	12 (6.0)
Birth injury/other congenital malformation	5 (7.2)	10 (17)	10 (17)	3 (10)	7 (7.8)	1 (1.7)	36 (9.8)	26 (13)
Previous stroke/vascular malformation	2 (2.9)	2 (3.2)	3 (4.8)	2 (3.1)	9 (10)	35 (59)	53 (14)	9 (4.5)
Previous head injury	2 (2.9)	0 (0.0)	0 (0.0)	2 (3.1)	12 (13)	1 (1.7)	17 (6.0)	3 (1.5)
Previous CNS infection	0 (0.0)	0 (0.0)	3 (4.8)	2 (6.3)	5 (5.6)	0 (0.0)	10 (2.7)	4 (2.1)
Other CNS inflammatory disease	0 (0.0)	0 (0.0)	1 (1.6)	0 (0.0)	4 (4.4)	1 (1.7)	6 (16)	1 (0.5)
Mesial temporal sclerosis	0 (0.0)	0 (0.0)	1 (1.6)	1 (3.1)	1 (1.1)	0 (0.0)	3 (0.8)	1 (0.5)
Presumed genetic epilepsy	0 (0.0)	0 (0.0)	6 (10)	3 (10)	5 (5.6)	0 (0.0)	14 (3.8)	8 (4.0)
Total remote causes	12 (17)	17 (29)	30 (50)	16 (50)	47 (52)	38 (66)	160 (44)	68 (34)
Progressive causes								
Tumor	0 (0.0)	0 (0.0)	2 (3.1)	0 (0.0)	8 (8.9)	6 (10)	16 (4.4)	2 (1.0)
Degenerative CNS disease	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (5.2)	3 (0.8)	0 (0.0)
Total progressive causes	0 (0.0)	0 (0.0)	2 (3.1)	0 (0.0)	8 (8.9)	9 (16)	19 (5.2)	2 (1.0)
Syndromic causes								
Epilepsy syndrome	2 (2.8)	3 (5.2)	7 (12)	1 (3.1)	0 (0.0)	0 (0.0)	13 (3.5)	13 (6.5)
Mitochondrial encephalopathy	0 (0.0)	1 (1.7)	2 (3.1)	1 (3.1)	0 (0.0)	0 (0.0)	4 (1.1)	3 (1.5)
Total syndromic causes	2 (2.8)	4 (6.9)	9 (15)	2 (6.3)	0 (0.0)	0 (0.0)	17 (4.6)	16 (8.0)
No cause identified	6 (8.7)	13 (22)	16 (27)	11 (38)	11 (12)	8 (14)	65 (18)	37 (19)

Note: All data are displayed as number of SE patients (percentage of patients in this age group). Patients could have an acute cause and either a remote, progressive, or syndromic cause, but etiological categories under each broad heading (acute, remote, progressive, syndromic) are mutually exclusive. These terms are as defined by Trinka et al.[11] We have included mitochondrial encephalopathies with syndromic causes.
Abbreviations: AED, antiepileptic drug; CNS, central nervous system; SE, status epilepticus.

TABLE 4 Incidence of SE among Auckland residents, adjusted by major subtypes

Subtype	Episodes	Incidence of SE episodes	Individual patients	Incidence of SE patients	Episodes lasting ≥30 min	Incidence of episodes ≥30 min	Patients with episodes ≥30 min	Incidence of patients with episodes ≥30 min
Any SE	450	27.87 (25.36-30.57) AA = 29.25 (27.34-31.27)	345	21.37 (19.17-23.75) AA = 22.22 (20.57-23.99)	250	15.48 (13.62-17.53) AA = 15.95 (14.56-17.45)	204	12.64 (10.96-14.49) AA = 12.92 (11.67-14.27)
With prominent motor symptoms	362	22.42 (20.17-24.85) AA = 23.41 (21.72-25.22)	294	18.21 (16.19-20.41) AA = 18.92 (17.39-20.55)	202	12.51 (10.85-14.36) AA = 12.80 (11.56-14.14)	171	10.59 (9.06-12.3) AA = 10.78 (9.65-12.02)
Nonconvulsive SE	83	5.14 (4.09-6.37) AA = 5.52 (4.7-6.45)	66	4.09 (3.16-5.2) AA = 4.34 (3.62-5.17)	45	2.79 (2.03-3.73) AA = 2.95 (2.37-3.66)	39	2.42 (1.72-3.30) AA = 2.55 (2.01-3.21)
Males								
Any SE	244	30.72 (26.98-34.98) AA = 31. 60 (28.83-34.59)	198	24.92 (21.57-28.65) AA = 25.54 (23.06-28.24)	137	17.25 (14.48-20.39) AA = 17.47 (15.44-19.71)	116	14.60 (12.07-17.51) AA = 14.72 (12.87-16.80)
With prominent motor symptoms	209	26.31 (22.86-30.13) AA = 27.04 (24.48-29.81)	174	21.90 (18.77-25.41) AA = 22.41 (20.10-24.95)	115	14.48 (11.94-17.38) AA = 14.68 (12.82-16.75)	99	12.46 (10.13-15.17) AA = 12.57 (10.68-14.50)
Nonconvulsive SE	31	3.90 (2.65-5.54) AA = 4.05 (3.09-5.23)	28	3.55 (2.34-5.09) AA = 3.85 (2.92-5.01)	20	2.52 (1.54-3.89) AA = 2.52 (1.79-3.47)	19	2.39 (1.44-3.73) AA = 2.41 (1.69-3.35)
Females								
Any SE	206	25.12 (21.80-28.80) AA = 27.14 (24.53-30.00)	147	17.92 (15.14-21.06) AA = 19.07 (16.91-21.46)	113	13.78 (11.35-16.56) AA = 14.61 (12.72-16.71)	88	10.73 (8.61-13.22) AA = 11.24 (9.61-13.10)
With prominent motor symptoms	153	18.65 (15.82-21.85) AA = 19.98 (17.76-22.43)	120	14.63 (12.13-17.49) AA = 15.57 (13.62-17.75)	87	10.61 (8.50-13.08) AA = 11.04 (9.42-12.87)	72	8.78 (6.87-11.05) AA = 9.08 (7.62-10.76)
Nonconvulsive SE	52	6.34 (4.74-8.13) AA = 7.04 (5.74-8.58)	38	4.63 (3.28-6.36) AA = 5.04 (3.96-6.36)	25	3.05 (1.97-4.50) AA = 3.45 (2.55-4.59)	20	2.44 (1.49-3.77) AA = 2.74 (1.94-3.77)

Note: Age-adjusted estimates are based on the Auckland population. Incidence (per 100 000 population) is shown, with 95% confidence intervals in parentheses.
Abbreviations: AA, age-adjusted; SE, status epilepticus.

TABLE 5 Ethnicity-standardized and ethnicity-/age-standardized incidence of SE, adjusted by major subtypes

	Episodes	Incidence of SE episodes	Individual patients	Incidence of SE patients	Episodes lasting ≥30 min	Incidence of episodes ≥30 min	Patients with episodes ≥30 min	Incidence of patients with episodes ≥30 min
NZ European								
Any SE	207	23.25 (20.28-26.76) AA = 23.56 (21.28-26.05)	170	19.18 (16.40-22.29) AA = 19.13 (17.09-21.37)	129	14.55 (12.15-17.29) AA = 14.39 (12.65-16.35)	109	12.30 (10.10-14.83) AA = 11.99 (10.40-13.78)
With prominent motor symptoms	159	17.94 (15.26-20.95) AA = 17.85 (15.88-20.02)	139	15.68 (13.18-18.52) AA = 15.50 (13.67-17.52)	98	11.06 (8.98-13.47) AA = 10.79 (9.28-12.50)	86	9.70 (7.76-11.98) AA = 9.34 (7.95-10.93)
Nonconvulsive SE	45	5.08 (3.70-6.79) AA = 5.36 (4.28-6.64)	36	4.06 (2.84-5.62) AA = 4.23 (3.29-5.38)	29	3.27 (2.19-4.70) AA = 3.36 (2.52-4.40)	26	2.93 (1.92-4.30) AA = 3.01 (2.22-4.00)
Māori								
Any SE	58	34.16 (25.94-44.15) AA = 34.73 (28.45-43.04)	49	28.86 (21.35-38.15) AA = 29.31 (23.52-37.14)	37	21.79 (15.34-30.03) AA = 21.84 (17.08-28.66)	31	18.26 (12.40-25.91) AA = 18.12 (13.82-24.52)
With prominent motor symptoms	52	30.62 (22.87-40.16) AA = 30.90 (25.01-38.80)	43	25.32 (18.33-34.11) AA = 25.46 (20.11-32.87)	35	20.61 (14.36-28.67) AA = 20.72 (16.08-27.43)	29	17.08 (11.44-25.53) AA = 17.00 (12.83-23.38)
Nonconvulsive SE	6	3.53 (1.30-7.69) AA = 3.85 (1.92-8.41)	6	3.53 (1.30-7.69) AA = 3.85 (1.92-8.41)	2	1.18 (0.14-4.25) AA = 1.12 (0.30-5.06)	2	1.18 (0.14-4.25) AA = 1.12 (0.30-5.06)
Pacific Islanders								
Any SE	97	42.73 (34.65-52.13) AA = 40.00 (34.45-46.48)	64	28.19 (21.71-36.00) AA = 26.55 (22.05-31.99)	46	20.26 (14.84-27.03) AA = 19.43 (15.58-24.25)	35	15.42 (10.74-21.44) AA = 14.92 (11.55-19.28)
With prominent motor symptoms	84	37.00 (29.52-45.82) AA = 34.61 (29.46-40.71)	58	25.55 (19.40-33.03) AA = 23.89 (19.65-29.09)	38	16.74 (11.85-22.98) AA = 16.13 (12.63-20.62)	31	13.66 (9.28-19.38) AA = 13.17 (10.02-17.32)
Nonconvulsive SE	12	5.29 (2.73-9.24) AA = 4.99 (3.17-7.86)	9	3.95 (1.81-7.53) AA = 3.81 (2.24-6.49)	8	3.52 (1.52-6.94) AA = 3.30 (1.87-5.84)	6	2.64 (0.97-5.73) AA = 2.52 (1.29-4.90)
Asian/other ethnicity								
Any SE	88	23.34 (18.72-28.75) AA = 25.13 (21.51-29.35)	62	16.44 (12.61-21.07) AA = 17.76 (14.73-21.38)	38	10.08 (7.13-13.83) AA = 10.74 (8.43-13.66)	29	7.69 (5.15-11.04) AA = 8.26 (6.25-10.90)

(Continues)

TABLE 5 (Continued)

	Episodes	Incidence of SE episodes	Individual patients	Incidence of SE patients	Episodes lasting ≥30 min	Incidence of episodes ≥30 min	Patients with episodes ≥30 min	Incidence of patients with episodes ≥30 min
With prominent motor symptoms	67	17.77 (13.77-22.56) AA = 18.96 (15.84-22.67)	54	14.32 (10.76-18.68) AA = 15.42 (12.61-18.83)	31	8.22 (5.86-11.67) AA = 8.68 (6.63-11.35)	25	6.63 (4.29-9.79) AA = 7.07 (5.23-9.55)
Nonconvulsive SE	20	5.30 (3.24-8.19) AA = 5.88 (4.19-8.23)	15	3.98 (2.27-6.56) AA = 4.43 (2.98-6.55)	6	1.59 (0.58-3.46) AA = 1.77 (0.91-3.34)	5	1.33 (0.43-3.09) AA = 1.48 (0.71-2.98)

Note: Age- and sex-adjusted estimates are based on the Auckland population; 95% confidence intervals are given in parentheses. Due to availability of data, please note that these ethnicity- and ethnicity-/age-standardized estimates are based on 2013 Census data, whereas the age- and sex-/age-standardized estimates are based on 2016 Census data.

Abbreviations: AA, age-adjusted; NZ, New Zealand; SE, status epilepticus.

However, other studies have also found an increased incidence of SE in males.[3,4,8,22] Noncompliance with AEDs was considered a significant etiological factor in more males than females, although this difference in compliance did not explain all of the difference observed between sexes. There was no significant difference in the number of boys and girls with febrile seizures in our study. It remains to be determined whether hormonal or other underlying predispositions may account for the observed gender difference.

The 30-day mortality in this study was 4.6%; this compares with mortality rates previously reported of up to 39%,[7] although other recent studies from Western countries have also reported similar rates of 4.7%,[32],[8] and 7%.[6] The mortality rates of SE in Britain declined between 2001 and 2013; this is thought to be due to a policy of early and aggressive treatment of SE.[33] Although superrefractory SE remains exceptionally difficult to treat,[34,35] it is relatively uncommon,[36] and most patients with SE respond to benzodiazepines or conventional AEDs.

This study is one of the first to be conducted using the semiological and etiological classification developed by the ILAE expert panel in 2015.[11] We found the classification easy to use and practical. It was usually straightforward to assign patients to one of the categories in the new classification. We recorded data regarding SE in the online EpiNet database. The database was developed to enable large international cohort studies, and to facilitate multicenter randomized controlled trials,[13] but it can be readily used for epidemiological studies.

4.1 | Limitations

It was at times difficult to determine whether a seizure had actually lasted for ≥10 minutes. Patients' families and other witnesses often overestimate the duration of a seizure. Often, we could determine whether a seizure had lasted ≥10 minutes by reviewing the times recorded on the ambulance report; we accepted the estimate of the seizure duration provided by the ambulance staff rather than the family or caregivers.

Some of the cases were excluded because it was decided on clinical grounds that the patient probably had nonepileptic seizures. These decisions were made on review of all the clinical and neurophysiological data, after the patients had been discharged. We consider that errors in this assessment would have been uncommon.

We are confident that nearly all patients who have a seizure with motor manifestations lasting ≥10 minutes in New Zealand are taken to one of the public hospitals, and that we have accurately determined the incidence of this type of SE. However, it can be difficult to diagnose NCSE, and clinicians need to have a high index of suspicion when assessing confused or obtunded patients to ensure that they do not miss this diagnosis.[37] Even if clinicians request an

EEG in confused and obtunded patients, interpretation of EEG features to allow a confident diagnosis of NCSE remains problematic, although ongoing work is helping to develop consensus in this area.[18,38–40] At the time of this study, all EEGs in Auckland were performed in a single department at Auckland City Hospital, and read by a group of seven neurophysiologists who assessed the EEGs using standardized criteria.[18] However, because access to EEG was not immediately available at all hospitals in our study, it is possible that some people with NCSE were not identified.

Our aim was to perform a prospective study in which we would identify all patients with SE when they presented to hospital; however, we identified only 72% of cases during or immediately following the episode of SE. This was despite intensive efforts to notify all clinicians treating patients with SE of the study.[17] The priority of the treating clinicians was to terminate the SE as rapidly as possible, with reporting of SE of secondary importance. We doubt that it would be possible to obtain a substantially higher rate of immediate reporting using this research approach.

We have calculated the incidence of SE in Auckland based on patients who presented to one of the hospitals in the greater Auckland region. We have not included Auckland residents who were admitted with SE to a hospital outside greater Auckland. We think very few patients will have been missed. Review of the Ministry of Health dataset for Auckland residents with a discharge diagnosis of SE identified only two Auckland residents who had been admitted to a hospital outside of Auckland. These patients were aged 1 and 7 years, respectively. The 7-year-old child had another episode during the year when he was admitted to one of the Auckland hospitals and is already included in the study. From this analysis, therefore, we are aware of only a single Auckland resident who had an episode of probable SE that has been excluded from the study because the episode occurred outside of Auckland.

5 | CONCLUSION

This epidemiological study has used the online EpiNet database to collect data regarding the time course, treatment, etiology, and outcomes of SE in Auckland. The incidence of SE in Auckland (the largest city in New Zealand) is similar to that found in other Western countries, although the incidence in children is higher than reported elsewhere. Sixty percent of all the SE episodes were tonic-clonic SE. There was a higher rate of SE in Māori and Pacific Islanders than in those of European or Asian descent. The mortality rate was relatively low, in line with results from other recent studies.

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CONFLICT OF INTEREST

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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