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Outcomes of Status Epilepticus in the Elderly

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Master of Public Health Research Project

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Abstract

Background: Status epilepticus (SE) is a serious medical condition associated with significant morbidity and mortality. Few studies have addressed this condition in the elderly. The present study examines predictors of SE mortality in this growing population.

Methods: SE patients visiting the Virginia Commonwealth University Medical Center from July 1, 1989, to June 30, 2006 were included in the study. Data on demographic characteristics, SE type, etiology, time to treatment and mortality were collected. Logistic regression analysis was conducted to examine the determinants of mortality due to SE. Data was stratified by age to examine the characteristics of SE among the elderly population.

Results: **A** total of 2,220 SE patients were included in this study. One-third of the patient population were elderly (>60 years). Mortality in the elderly group was significantly higher than in the young group (OR=3.54 CI 2.53-4.95). The logistic regression model showed that being white, female, having hypoxia, CNS acute, non-CNS acute and remote etiology groups were significant predictors for mortality in the elderly.

Conclusions: SE is a serious medical condition, consisting of prolonged seizure activity, associated with a significant mortality. Elderly patients with SE represent a distinct population with unique characteristics.

INTRODUCTION:

Status epilepticus (SE) is a serious condition of prolonged or repetitive seizures. The annual incidence of SE in the elderly aged 60 and greater (86/100,000) is almost twice that of the general population and is even higher in those who are 70 to 79 years old (100/100,000). Either acute or remote stroke causes SE in approximately 60% of the elderly. Status epilepticus is associated with a high mortality in the elderly (38%) and has a mortality approaching 50% in patients older than 80 years of age. Etiology is a strong determinant of mortality in the elderly: mortality approaches 100% in patients with anoxia and 30% in patients with metabolic disorders, hemorrhages, tumors, or systemic infections. Mortality is almost three times higher in SE associated with acute ischemic stroke than in stroke alone, indicating synergistic effects.

Duration of SE is also a factor in mortality; therefore, an electroencephalogram (EEG) should be promptly obtained so that treatment can begin without delay. Because older patients have a greater likelihood of nondiagnostic findings on routine EEGs, prolonged EEG recordings and inpatient video-EEG monitoring significantly increase the rate of establishing a definitive diagnosis. Nonconvulsive status epilepticus in the elderly is especially difficult to diagnose and should be evaluated with an EEG.

Treatment of SE is complicated by altered pharmacokinetics in the elderly and should be initiated for any convulsive seizure that lasts at least 10 minutes or is repetitive. Initial treatments, usually the administration of an intravenous benzodiazepine, have overall success rates of 55% for overt convulsive SE and 14.9% for subtle SE. For refractory SE, little is gained by using additional standard drugs, and general anesthesia with continuous EEG monitoring is recommended.

The Scope of the Challenge

The elderly are the fastest growing segment of the United States population. The U.S. Department of Health and Human Services predicts that by 2030 there will be approximately 70 million adults over the age of 65 in the country (U. S. Department of Health and Human Services). This segment of the population was 12.4% of the total population in 2000; however, by 2030 it will account for approximately 20%. The elderly have the highest incidence of seizures of any age group (Hauser *et al.*, 1993). The increased frequency of seizures in this population can be attributed to comorbid conditions and characteristics such as an increased risk for stroke, metabolic abnormalities, and an increased use of prescription drugs. As the U.S. population ages, physicians will increasingly face the challenge of diagnosing and effectively managing seizures in the elderly.

Definitions

Status epilepticus (SE) has been defined as "a condition characterized by an epileptic seizure which is so frequently repeated or so prolonged as to create a fixed and lasting epileptic condition" (Gastaut, 1983). However, the duration of what is accepted as SE has been shrinking. (Wasterlain and Chen, 2006) The Veterans Affairs cooperative trial on the treatment of SE used an operational definition of 10 minutes to the time of treatment (Trieman et al 1998) and more recently it has been suggested that an operational definition for treatment should be a generalized convulsive seizure lasting 5 minutes (Lowenstein 1999 b). However, the most widely used definition of SE for the older epidemiological studies is more than 30 minutes of either continuous seizure activity or intermittent seizures without full recovery of consciousness between seizures. Since most seizures last less than 2 minutes, some authors have proposed that

SE should be diagnosed and treated well before 30 minutes have elapsed (Lowenstein *et al.*, 1999b). For this reason, it is reasonable to diagnose and treat any prolonged or repetitive seizures as impending SE. According to Gastaut (1983), the classification of SE contains as many types of SE as there are types of epileptic seizures.

Types of partial SE include somatosensory seizures, which can include any sensory modality (vision, taste, smell, hearing, or touch); autonomic seizures, which may include changes in visceral sensation or heart rate; and psychic seizures, during which patients report feelings of anxiety, depression, fear, or altered perception of time such as déjà vu. Complex partial SE is characterized by impairment of consciousness and may be accompanied by automatisms. Patients may describe an aura, which is a simple partial seizure, preceding the loss of consciousness. Partial seizures can secondarily generalize, and patients may also experience a complex partial seizure before secondary generalization. The major types of generalized seizures are absence, myoclonic, and tonic-clonic. Recently, authors have reported that nonconvulsive SE (NCSE) is frequently underdiagnosed and has a high mortality. Nonconvulsive SE is usually categorized into two entities: partial SE and generalized nonconvulsive SE (bilateral diffuse synchronous seizures) (Kaplan, 1999).

The Epidemiology of Status Epilepticus in the Older Patient

Status epilepticus is associated with a high mortality in the older population. Recent studies have addressed this important condition in the older age groups. The age-specific incidence rates of SE have a bimodal distribution, with the highest rates in infants and the elderly. The elderly, defined as greater than 60 years of age, had an annual incidence rate of 86 per 100,000 (DeLorenzo *et al.*, 1996)(Figure 1). From the same study, the highest incidence rate of SE was seen in children between the ages of 1 and 12 months, at 156 per 100,000. However, if

the data is analyzed by major age groups, with the pediatric age group including ages from 1 month up to 16 years of age, adults defined as ages 16 up to 60, and the elderly group defined as age 60 and greater, the elderly group had the highest incidence rate of the three groups. The incidence of SE in the age 60 and greater group was almost twice that of the general population. When the elderly population was further subdivided into age groups consisting of 60 to 69 years, 70 to 79 years, and 80 years and greater, the highest incidence of 100 per 100,000 individuals per year was seen in the 70 to 79 age group (DeLorenzo, 1997).

In a study of generalized convulsive SE (GCSE) in California, Wu *et al.* reviewed the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes from nonfederal hospitals (Wu *et al.*, 2002). In this study, the incidence of SE for all ages was 6.2 per 100,000. The rate of GCSE was highest among children under the age of 5 (7.5/100,000) and among the elderly age 75 and older (22.3/100,000). The incidence of SE was lower in this study than in other previous reports in the United States. Reasons for this lower incidence may be explained, partially, by the retrospective nature of the study, which relied on ICD-9-CM discharge coding data and the inclusion of only those patients in GCSE. In a prospective study conducted in Bologna, Italy, the incidence rate was 26.2 per 100,000 in patients older than 60 years versus 5.2 per 100,000 in patients younger than 60 years (Vignatelli *et al.*, 2003). In another prospective study, undertaken in the French-speaking part of Switzerland, the age-specific annual incidence rate again showed a bimodal distribution with a peak in children, a minimum during adolescence and young adulthood, and a progressive increase after the age of 60. In this cohort, the incidence rate was 15.1 per 100,000 in the 60 to 74 age group and 15.5 per 100,000 in the 75 and older age group (Coeytaux *et al.*, 2000). Although this study revealed the same age-specific distribution, a lower specific incidence rate in

the elderly was found, which could be attributed to the exclusion of patients in postanoxic coma. In Hessen, Germany, a prospective study by Knake and colleagues revealed an incidence of 54.5 per 100,000 in the elderly (age 60 or older) versus 4.2 per 100,000 in younger adults (18 to 59 years of age) (Knake *et al.*, 2001).

Etiologies of Status Epilepticus in the Elderly

Etiologies of SE are important for determining the morbidity and mortality associated with this condition (Towne *et al.*, 1994). In the Richmond SE study, the three major etiologies of SE were low anticonvulsant levels at 34%, remote symptomatic at 23%, and acute stroke at 22%. In patients over the age of 60, acute strokes accounted for 35% of the total, and remote symptomatic stroke accounted for 26% of the cases. Thus, either acute or remote stroke caused SE in approximately 60% of elderly patients (DeLorenzo *et al.*, 1995). Other etiologies in the older age group, from the same study, included hypoxia (17%), metabolic (14%), alcohol related (11%), tumor (10%), infection (6%), anoxia (6%), hemorrhage (5%), central nervous system (CNS) infection (5%), trauma (1%), idiopathic (1%), and other (1%). In a retrospective population- based study of SE from Rochester, Minnesota, another common etiology of SE was found to be dementia (Hesdorffer *et al.*, 1998). In a study of GCSE, Wu and colleagues found that, in elderly patients, the most common etiology was delayed effect of stroke or brain injury (Wu *et al.*, 2002). In the Coeytaux *et al.* study (2000), most of the cases in patients over the age of 60 were in the acute symptomatic group, which included etiologies occurring in close association with an acute cerebral insult such as cerebrovascular disease, CNS infection, alcohol-related insults, metabolic insults, and drug withdrawal.

Mortality of Status Epilepticus in the Elderly

In the Richmond study (DeLorenzo *et al.*, 1995), the overall mortality was 22%. However, there was a considerable difference between the mortality in the pediatric population at 3% versus the adult mortality at 26%. The differences were even more marked in the elderly population at 38%, with a mortality approaching 50% in the patients greater than 80 years of age. Along with age and duration of SE, etiology is a strong determinant of mortality in the elderly (Towne *et al.*, 1994). The higher mortality in elderly patients may be secondary to the increased susceptibility of these patients to systemic metabolic diseases, cerebral vascular accidents, and progressive symptomatic conditions such as tumor and dementia. Depending on the etiologies, the mortality rates differed markedly. Etiologies with mortalities less than 9% included patients presenting with alcohol withdrawal or low levels of antiepileptic medications. Remote symptomatic cases, the majority of which consisted of prior strokes, had a mortality rate of 14%. Etiologies such as metabolic disorders, hemorrhages, tumors, and systemic infections were each associated with approximately 30% mortality, and CNS infections and head trauma were associated with approximately 1% and 20% mortality, respectively. The highest mortality, approaching 100%, was seen in patients with anoxia (Towne *et al.*, 1994).

Because ischemic brain injury is a major cause of SE in the elderly, and since stroke and SE each have high mortalities, the question is raised as to whether the high mortality seen in patients with stroke and SE is attributable to the severity of the underlying ischemia or to the effect of SE itself. This question was answered by a study looking at three groups of patients: 1) acute ischemic stroke without SE, 2) acute ischemic stroke with SE, and 3) remote ischemic stroke and SE (Waterhouse *et al.*, 1998). The results of this study demonstrated that SE with acute cerebrovascular accident (CVA) had a 39% mortality, whereas acute CVA alone had a

14% mortality, and SE with old CVA had the lowest mortality at 5% (Figure 2). No differences in radiologic lesion size were seen between the groups. This study demonstrated that mortality is almost three times higher when SE is associated with acute ischemic stroke than in stroke alone. This would indicate that the high mortality associated with concurrent SE and CVA in the elderly is due to the synergistic effect of SE in ischemic brain injury.

Nonconvulsive Status Epilepticus

Nonconvulsive status epilepticus in the elderly may be difficult to diagnose. A prospective study of elderly patients with NCSE found that it had a worse prognosis in the elderly than in younger patients. This was felt to be secondary to the severity of the underlying etiologies (Labar *et al.*, 1998). Nonconvulsive SE mortality was 52% in a study of 25 critically ill elderly patients, and death was associated with having a larger number of acute life-threatening medical problems on presentation (Litt *et al.*, 1998). In this group of critically ill patients, treatment of NCSE with benzodiazepines increased the risk of death, and aggressive anticonvulsant therapy did not improve the outcome.

Suspected NCSE should be evaluated with an electroencephalogram (EEG). The clinical manifestations may be subtle, consisting of prolonged confusion, unusual behavior, minor motor manifestations, aphasia, or coma. Lee (1985) conducted a study of eleven patients without a history of absence seizures who presented with an acute onset of a prolonged confusional state. All patients were ambulatory with behavior ranging from mild disorientation to confusion. Some patients demonstrated prominent psychiatric manifestations. The EEGs generally showed 1 to 2.5 Hertz generalized spike-wave or multiple spike-wave discharges. The patients were successfully treated with intravenously administered diazepam followed by orally administered phenytoin sodium and phenobarbital (Lee, 1985).

Nonconvulsive status epilepticus is an often unrecognized cause of coma, especially in elderly patients. In a study of 236 patients with coma and no overt clinical seizure activity, it was found that 8% of these patients had NCSE (Towne *et al.*, 2000). Approximately 50% of these patients were over the age of 60.

Privitera *et al.* (1994) prospectively studied hospitalized patients with altered consciousness who underwent an EEG to evaluate coma or altered mentation. In this study of 198 cases with altered consciousness but no clinical convulsions, 37% showed EEG and clinical evidence of definite or probable NCSE. In 23 cases, altered consciousness was the only clinical sign at the time of diagnosis, with subtle motor activity present in 36 others. Neither clinical signs nor prior history predicted which patients demonstrated SE on EEG. Lowenstein and Aminoff reported on 38 patients with NCSE and concluded that the majority of patients with subtle motor activity and depressed consciousness had EEG findings of SE, and that an EEG was necessary to diagnose this condition (Lowenstein and Aminoff, 1992).

Nonconvulsive status epilepticus can be seen after the control of convulsive SE. In the Richmond study, 14% of the patients manifested NCSE after the cessation of convulsive SE. These patients were comatose and showed no overt clinical signs of convulsive activity. Clinical detection of NCSE in these patients would not have been possible without the use of EEG monitoring (DeLorenzo *et al.*, 1998).

Electroencephalogram

Older patients with acute seizures may have a variety of EEG changes, only some of which are attributable to underlying pathology. Although benign EEG variants with epileptiform morphology occur in all age groups, three that occur with a greater frequency in the older population are subclinical rhythmic electrical discharges of adulthood, wicket spikes, and small

sharp spikes (Van Cott, 2002). These patterns can potentially be misinterpreted as epileptiform abnormalities. Interictal epileptiform activity occurs less frequently in older age groups than in younger age groups (Marsan and Zivin, 1970). This indicates that older patients have a greater likelihood of nondiagnostic findings on a routine EEG. The recent Department of Veterans Affairs (VA) Cooperative Study, conducted in elderly patients, found interictal epileptiform activity in approximately one third of routine EEGs (Ramsay and Pryor, 2000). Prolonged EEG recordings and inpatient video-EEG monitoring significantly increase the diagnostic yield in elderly patients (McBride *et al.*, 2002). Although elderly patients account for approximately 25% of newly diagnosed seizures, older patients are relatively underrepresented in epilepsy monitoring units, despite the usefulness of monitoring in establishing a definitive diagnosis (Kellinghaus *et al.*, 2004).

Treatment

There is no established protocol for the management of SE in the elderly patient. Treatment is complicated by the fact that the pharmacokinetics are more complex in the elderly than in younger patients because of the altered volume of distribution, lower protein binding, decreased renal elimination, decreased hepatic metabolism, decreased enzyme inducibility, and increased use of polypharmacy. However, treatment should be initiated for any convulsive seizure that has lasted at least 10 minutes or for repetitive seizures. Emergent attention should be given to establishing an airway, monitoring oxygenation and vital signs, establishing intravenous (IV) access, measuring the blood glucose level, and sending blood for analysis of blood count, serum electrolytes, and antiepileptic drug levels. The Epilepsy Foundation of America and others have suggested protocols for SE treatment that have been widely accepted or adapted (Dodson *et*

al., 1993; Lowenstein and Alldredge, 1998; Willmore, 1998) (Table 1). The goal of treatment for SE is rapid cessation of clinical and electrical seizure activity.

Most treatment protocols call for the administration of an IV benzodiazepine as first-line therapy, because they are fast acting and effective for all seizure types (Leppik *et al.*, 1983). Lorazepam has a longer duration of action than diazepam. While the longer antiepileptic effect of lorazepam is preferable, it may have the undesired side effect of causing a prolonged period of depressed consciousness. However, one study has demonstrated that lorazepam, when administered by paramedics for out-of-hospital SE in adults, is significantly more effective than diazepam at terminating SE (Lowenstein *et al.*, 1999a). Diazepam is more lipid soluble, and although it enters the brain quickly, it is also quickly redistributed to other fatty tissues. Therefore, its therapeutic effect is brief (15 to 30 minutes), and SE may recur after a single dose of diazepam. It is usually recommended that a longer-acting drug, such as phenytoin or fosphenytoin, be administered immediately after diazepam (Dodson *et al.*, 1993; Lowenstein *et al.*, 1999a).

Success of Initial Treatment

A large VA Cooperative Study compared four IV treatments for generalized SE, and many of the subjects were elderly (median age, 58.6 years in overt convulsive SE and 62.0 years in subtle convulsive SE) (Treiman *et al.*, 1998). The treatments evaluated were lorazepam, phenytoin, diazepam and phenytoin in combination, and phenobarbital. Success of initial treatment ranged from 64.9% for lorazepam to 43.6% for phenytoin. When lorazepam and phenytoin were compared, lorazepam was significantly more effective than phenytoin. There were no differences between the four treatments in the incidence of hypotension requiring treatment, respiratory depression, or cardiac rhythm disturbances.

Phenytoin and Fosphenytoin

A loading dose of IV phenytoin is usually effective in terminating SE (Wilder, 1983; Wilder *et al.*, 1977). The recommended loading dose is 18 mg/kg intravenously, with a maximal rate of administration of 50 mg per minute. At this rate, hypotension occurs in 28% to 50% of patients, and cardiac arrhythmias in 2% (Cranford *et al.*, 1978; Wilder, 1983). Therefore, the patient's blood pressure and cardiac rhythm must be monitored continuously during a rapid infusion, and if these adverse effects occur, the infusion rate should be slowed.

Fosphenytoin is the phosphate ester of phenytoin, and unlike phenytoin, it is highly water soluble, with a near physiologic pH. After entering the bloodstream, fosphenytoin is rapidly converted to phenytoin by systemic phosphatases (Browne *et al.*, 1996). Therapeutic phenytoin concentrations are obtained in most patients within 10 minutes of rapid IV fosphenytoin infusion. Fosphenytoin doses are expressed as the amount of phenytoin delivered (phenytoin equivalents, abbreviated as PE). Unlike phenytoin, it is not formulated with propylene glycol, allowing faster infusion rate, better tolerability with less local irritation at the infusion site, and less toxicity from the drug vehicle (Willmore, 1998). However, no clinically significant differences in the adverse effects of hypotension or cardiac arrhythmias have been recorded, and monitoring electrocardiogram (EKG) and blood pressure is still important, especially in the elderly. Fosphenytoin, unlike phenytoin, can be given intramuscularly with effective levels reached in approximately 20 minutes (Leppik IE, Rask CA, Watridge C, Graves N, Murthy VS, Boucher B, Wilder BJ, Turlapaty P. Pharmacokinetics of phenytoin prodrug after IV and IM administration. *Neurology* 1988;38(1):186.) This may be an alternative method for delivering a loading dose while IV access is being established.

Treatment of Refractory Status Epilepticus

In the VA Cooperative Study (Treiman *et al.*, 1998), the overall success of first drug treatment was 55% for overt convulsive SE and 14.9% for subtle SE. Traditionally, patients who continued to seize after treatment with a benzodiazepine and phenytoin were treated with phenobarbital. However, the VA study results suggest that when the initial treatment of SE fails, little is gained by using additional standard drugs. Only 7% of overt convulsive SE and 3% of subtle SE cases responded to treatment with a second drug (Treiman *et al.*, 1999). Many are now advocating that SE that is refractory to initial therapy should be treated with general anesthetic agents such as midazolam, propofol, phenobarbital, or pentobarbital (Bleck, 1999; Lowenstein *et al.*, 1988; Treiman *et al.*, 1999).

All SE patients who receive general anesthesia will require intubation and mechanical ventilation with hemodynamic monitoring in an intensive care unit. Since hypotension is a common side effect, vasopressors may be needed. These patients also require continuous EEG monitoring to document electrographic cessation of seizures. Various protocols for the treatment of refractory SE have been proposed (Bleck, 1999; Lowenstein and Alldredge, 1998; Willmore, 1998).

Pentobarbital

Pentobarbital is preferable to phenobarbital in the treatment of refractory SE because it has a shorter elimination half-life, allowing shorter duration of sedation and more timely assessment of the patient's baseline clinical mental status. Pentobarbital is widely available and relatively inexpensive when compared with propofol or midazolam. An IV loading dose of 3 to 12 mg/kg is followed by continuous infusion of 1 to 10 mg/kg per hour, titrated to the desired EEG effect.

A number of studies have confirmed that while pentobarbital is effective for treating refractory SE, it is associated with a high mortality (Krishnamurthy and Drislane, 1996; Lowenstein *et al.*, 1988; Mirski *et al.*, 1995; Osorio and Reed, 1989; Rashkin *et al.*, 1987; Stecker *et al.*, 1998; VanNess, 1990; Yaffe and Lowenstein, 1993). While etiology is a strong determinant of prognosis, other positive prognostic indicators include prior history of epilepsy, absence of multiorgan failure, and absence of hypotension requiring vasopressors during phenobarbital coma (Krishnamurthy and Drislane, 1996; Yaffe and Lowenstein, 1993).

Midazolam

Midazolam (Versed®) is a short-acting benzodiazepine that has been shown to be effective in terminating SE (Denzel and Burstein, 1996; Kumar and Bleck, 1992; Parent and Lowenstein, 1994). It has rapid onset and short elimination half-life, avoiding the prolonged sedation that can occur with barbiturates. In addition, it has less-pronounced hypotensive effects than barbiturates. Disadvantages include high cost and tachyphylaxis, commonly requiring dramatic dose escalation after 1 to 2 days (Bleck, 1999). A loading dose of 3 to 5 mg/kg is followed by an infusion of 1 to 15 mg/kg per hour, titrated to the desired EEG effect (Bleck, 1999).

Propofol

Propofol (Diprivan®) is a gamma-aminobutyric acid $(GABA)_A$ agonist with a short duration of action, and it terminates SE rapidly (Kuisma and Roine, 1995; Stecker *et al.*, 1998). A loading dose of 1 to 5 mg/kg is administered over 5 to 10 minutes, followed by infusion of 1 to 15 mg/kg per hour, titrated to EEG seizure suppression (Stecker *et al.*, 1998). There is evidence that rapid discontinuation of propofol can induce

withdrawal seizures. Thus, it is recommended that it be tapered by 5% of the maintenance infusion rate per hour (Stecker *et al.*, 1998). A retrospective study compared propofol and midazolam for refractory SE, and the authors concluded that midazolam was as likely to be effective as propofol, and probably had fewer side effects (Prasad *et al.*, 2001).

Valproic Acid

The low risk of hypotension, respiratory depression, and sedation make IV valproate a potentially advantageous choice for the treatment of SE in the elderly. In a European study of 23 cases of SE, including absence, tonic-clonic, myoclonic, and partial SE, results showed that in 19 of 23 cases the patient responded within 20 minutes to IV valproate 15 mg/kg initial injection, which was followed 30 minutes later by an infusion at 1 mg/kg per hour for 5 to 6 hours (Giroud *et al.*, 1993). Other studies using loading doses as high as 60 to 70 mg/kg, followed by maintenance infusion, have also demonstrated that IV valproate is efficacious and safe in the treatment of SE (Bleck, 1999; Knake *et al.*, 1999; Peters and Pohlmann-Eden, 1999; Price, 1989; Short *et al.*, 1999). Safety and tolerability of rapid infusion have been established (Limdi and Faught, 1999).

When valproate is used in the elderly, smaller doses may be sufficient to achieve a given serum concentration, and the potential presence of inhibiting comedications needs to be considered (Pluhar *et al.*, 1999). In addition, rapid infusion of IV valproate results in a rapid change in valproic acid (VPA) total and unbound levels, suggesting that unbound VPA concentrations should be monitored when IV valproate is used to treat SE (Kriel *et al.*, 1999). Although IV valproate has shown efficacy in the treatment of SE,

with a low risk of hypotension or respiratory depression, more information is needed before its place in the overall treatment of SE is established (Bleck, 1999).

Topiramate

Topiramate administered nasogastrically has been shown to be effective in terminating refractory SE (Bensalem and Fakhoury, 2003; Towne *et al.*, 2003). Effective doses range from 300 to 1600 mg per day. Published case series have noted no adverse events resulting from the use of topiramate for SE, except for lethargy. More studies are needed before clear guidelines for use of this agent are established.

Levetiracetam

Levetiracetam is chemically unreliable to existing AED's and the precise mechanisms by which it exerts its antiepileptic effect is unknown. However, studies suggest that LEV may selectively prevent hypersynchronization of epileptiform burst firing and propagation of seizure activity. Recently LEV became available for intravenous administration. LEV may potentially increase the options for the treatment of SE although there are few studies available to determine its effectiveness for this condition. (Rossetti AO, Bromfield EB: Levetiracetam in the treatment of status epileptics in Adults: A study of 13 episodes. *European Neurology* 2005;54:34-38.

In summary, status epilepticus is associated with a high mortality in the older population partially because of the increased susceptibility of these patients to systemic metabolic diseases, strokes, and other comorbidities that may lead to increased mortality. Along with etiology and age, duration of SE is a strong determinant of mortality. Because many older patients may not present with the typical signs of convulsive SE, the diagnosis may not be made until the patient

has continued to seize for a prolonged period of time. Thus, it is imperative to have a high index of suspicion of NCSE in the older patient and to obtain an EEG, so that rapid treatment can be instituted. Treatment needs to be instituted as soon as the diagnosis is made, using the more recent concept of duration, keeping in mind that the pharmacokinetics are more complex in the elderly than in younger patients.

OBJECTIVES

The major objectives of this study are to analyze the characteristics of SE in two age populations: those patients in the 60 and older age groups, and those patients younger than age 60. The characteristics to be studied include race, gender, SE type, etiology, time to treatment and mortality.

Because of the high mortality and morbidity associated with SE, it is important to develop objective criteria to predict prognosis in this major neurologic emergency. Currently there are no large scale prospective studies which have addressed this condition. The relationship between time to treatment, seizure type, etiology, age, race, and gender with mortality in patients with SE were evaluated by univariate and multivariate statistical analysis.

METHODS

Study Design and Study Population:

We utilized a large population-based, prospectively collected database of SE cases presenting in the Richmond Metropolitan Area from July 1, 1989, to June 30, 2006. SE cases were identified during the course of this study at the Medical College of Virginia Hospitals. The university hospitals are the major hospitals in the city limits and accounted for 75% of the

patients presenting with SE in Richmond. The remaining 25% of the patients living in the city limits presented in community hospitals in and around the city.

Recruitment

SE cases were prospectively identified by the SERT that evaluated all cases presenting to the study on a daily basis. The case histories and charts of potential SE patients were reviewed by the SERT in order to determine whether each identified case met the rigorous definition of SE described above. SERT members were on call 24 hours a day, 7 days a week in order to be able to respond to each case of SE in a prospective, timely manner to be able to collect accurate data on the clinical presentation of SE. At both the university and community hospitals, SE cases were identified by neurologists and nurses reporting to the SERT. This form of case identification was identical in all hospitals and accounted for the majority of records. At the Medical College of Virginia Hospital, additional ascertainment of cases could be utilized. Cases were also identified by ER personnel reporting to the SERT and by daily rounds on the Neurology Morning Report Service, in the emergency room, EEG laboratory attendings and in all ICUs.

In reviewing each case reported to SERT, approximately 80% of the SE cases met the definition of SE. The remaining cases had to be excluded because sufficient history or documentation could not be obtained to meet the definition of SE.

Data Collection and Data Entry

SE was strictly defined using the definition of the International Classification of Epileptic Seizures as any seizure lasting for 30 minutes or longer or intermittent seizures lasting for greater than 30 minutes from which the patient did not regain consciousness. [24] Patients who seized up to 29 minutes and were controlled with anticonvulsants were excluded by this definition of

SE. In addition, if the seizure episode or event could not be documented as a 30-minute seizure or intermittent seizures without regaining consciousness for 30 minutes, the patient could not be diagnosed as having SE. Nonconvulsive SE (electrographic) was defined as continuous EEG seizure activity lasting for 30 minutes or longer without significant clinical seizure activity. Periodic lateralized epileptiform discharges alone were not considered as seizure discharges.

Following the identification and documentation of each SE case, the medical records were carefully reviewed during the hospitalization period or immediately following discharge of each SE patient. This type of rapid and timely prospective review provided a more accurate assessment of the data on each patient than could be obtained by retrospective analysis. In comparing prospective to previous retrospective data collection, it was evident that routine chart record keeping was not of sufficient detail for accurate descriptions of the clinical aspects of SE. In the previous retrospective pilot data accurate descriptions of seizure type, duration of SE, and times of drug administration were very poorly documented in the clinical chart. However, by being able to directly question the physicians, nurses, and family members of each patient, the status epilepticus research team (SERT) could obtain a more accurate and detailed clinical history of each SE event. This information was entered into a data entry system for each patient and included the following information: detailed seizure history, electrophysiologic data, demographic data, previous medical or neurologic history, immediate precipitating etiology of SE, outcome, laboratory studies, and hospital course (see appendix A). To evaluate the duration of SE and time to recovery or death, a time line for each SE event was established. These time lines and medication histories were carefully evaluated and reviewed by the SERT not only to validate the definition of SE, but also to verify the accuracy of the clinical and demographic data.

Data Analysis

Statistical methodology includes both univariate and multivariate logistic regression analysis. Mortality and specific prognostic indicators will be analyzed initially utilizing univariate logistic regression. Multivariate logistic regression analysis will be employed to examine the association between several factors. Data analysis will be performed utilizing SSPS Version 15.0.

Database Validation

A labor-intensive, complete review of SE presentations was performed to more critically evaluate the completeness of case ascertainment and to validate the database. This review evaluated all hospital ICD-9 codes for seizures, 911 reports for seizures in the ER, presentation of SE on hospital rounds, all EEG laboratory reports, and ICU and ER records and personnel. This review provided a high degree of accuracy in documenting essentially all SE cases. This type of detailed review was carried out for 1 month for each year of the study at the MCV Hospitals and at two of the large community hospitals during the course of the study. Comparing the cases identified by this procedure and those prospectively identified during the data collection of this study, we determined that the prospective identification of SE patients at the MCV Hospitals was validated at approximately 90% of all cases. In the community hospitals, it was found that the identification of SE patients was approximately 33%.

During the initial design of this study, evaluation of the community hospitals through consultation with hospital personnel, neurologists, and ER physicians, it was thought that essentially all SE cases received neurologic consultation. Thus, we utilized neurologists and neurology nurses as the referral sources in these hospitals. However, it was evident during the study that over one-half of the SE cases presenting in the community hospitals were not brought

to the attention of adult or pediatric neurologists. These cases were being managed by internists, ICU specialists, or ER personnel. Since a significant number of SE cases were not seen by neurologists in these hospitals, the total number of patients identified in the community hospitals did not reach the high level of identification in a prospective manner that was performed at the MCV Hospital and represent an underestimate of the actual presentation of SE. Under-identification of SE cases was partially corrected by using the estimate of under representation described for MCV and community hospitals using the ascertainment rate of 90% for MCV and 33% for community hospitals to estimate the actual incidence of SE in the population.

Inclusion and Exclusion Criteria

Age:

Included in this study were all patients aged 31 days or older. The pediatric population (children) included all patients from 31 days up to 16 years, and adults were patients 16 years of age or older. Neonates less than 1 month in age were excluded from this study. The elderly population was defined as patients aged 60 years and older, and young adults represented patients from 16 to 59 years.

Mortality:

Mortality was defined as death in association with SE, occurring from the initiation of SE to 30 days following the termination of SE. All patients were followed for a minimum of 30 days after the control of SE or to the time of death. Survival was defined as alive at 30 days after SE was controlled.

Recurrence rate:

The recurrence rate was defined as another episode of SE occurring within a 2-year follow-up time period after the initial SE event. All patients in the study were

followed for a 2-year time period in the database. Recurrent SE events within a single hospitalization were also included in this evaluation.

History of epilepsy:

History of epilepsy was defined as two or more seizure episodes in a lifetime.

Etiologies:

Etiologies of SE were defined as the acute, remote symptomatic and idiopathic causes of SE. Previous medical conditions that were not the acute cause of SE were not included in acute symptomatic etiologies and were categorized as remote symptomatic. Occasionally, SE was associated with more than one concurrent etiology. Acute symptomatic etiologies were defined as SE in association with an underlying etiology within 7 days of the acute onset of that etiology. Remote symptomatic etiologies included patients with SE without an acute precipitating cause, but with a history of insults to the CNS temporally remote to the first unprovoked SE episode. This category was defined using the classification of Hauser. The following acute etiologic categories were associated with SE in this study:

Acute symptomatic etiologies.

(1) Anoxia: acute deprivation of oxygen to the CNS from documented respiratory or cardiorespiratory arrest lasting greater than 5 minutes. (2) Hypoxia: respiratory insufficiency documented by cyanosis and decreased oxygen saturation or decreased oxygen levels by blood gases prior to SE, or both. (3) Cerebrovascular disease (CVA): cerebrovascular disease, including vascular occlusion, embolism, or hemorrhagic infarct. This category did not include intracerebral hemorrhage associated with hypertension or other causes. (4)

Hemorrhage (HEM): intracerebral or subarachnoid hemorrhage. (5) Tumor: primary or metastatic CNS tumors. (6) Infection (Infec)/Fever: systemic infection not involving the CNS with temperature elevation. (7) Central nervous system infections (CNS Infec): infections of the CNS, including meningitis, abscess, and fungal, bacterial, viral, or other causes. (8) Metabolic (Metab): systemic dysfunction manifested by metabolic disturbances, such as electrolyte imbalance, hypoglycemia, uremia, or other metabolic disorders. (9) Low anticonvulsant drugs (LAED): a documented low AED level in a patient with epilepsy that was confirmed by nontherapeutic anticonvulsant blood levels at the time of presentation with SE. (10) Drug overdose (Drug OD): an acute drug overdose. (11) Alcohol related (ETOH): alcohol withdrawal or alcohol intoxication. (12) Head trauma: injury requiring medical treatment or hospitalization with or without loss of consciousness.

Remote symptomatic (Remote) etiologies:

No acute precipitating etiology, but with a previous history of CNS insult, such as a CVA, CNS infection, congenital (congenital malformations, hydrocephalus, arteriovenous malformations, and genetic disease), trauma, hemorrhage, or tumor.

Idiopathic etiology:

SE in association with no identifiable acute or remote cause for the initiation of the seizures.

Seizure types:

Seizure types in SE were defined as partial or generalized SE based on the International Classification of Seizure Types and types of SE as defined by the International League Against Epilepsy, as reviewed by Gastaut and Hauser. Partial SE was divided into simple partial (SP), complex partial (CP), or partial with secondary generalization (PSG). Simple partial SE referred to episodes where the patient maintained alertness and the ability to interact appropriately with the environment during partial seizure activity that lasted for 30 minutes or longer. Complex partial SE included patients with partial seizures with confusion and with amnesia for the ictus. Partial seizures with secondary generalization were classified as SE that initiated with partial onset and a subsequent secondary generalization, according to the criteria of the International League Against Epilepsy. Electrographic partial SE was included in the electrographic (ELECT) category and was defined as SE that occurred in patients who were unconscious and manifested a single electrographic focal discharge lasting 30 minutes or longer without overt clinical seizure activity such as arm twitching or leg twitching. PLEDS alone were not considered as ictal discharges. Generalized SE was subdivided into generalized tonic-clonic (GTC), generalized absence (ABS), myoclonic (MYOCL), and generalized nonconvulsive SE that was included in the ELECT category. Nonconvulsive SE seizures were defined as 30 minutes or longer of continuous EEG seizure activity based on standard EEG criteria for seizure discharge while the patient was comatose and was without significant clinical activity. These SE seizure types were classified using the International League Against Epilepsy definitions, as reviewed by Gastaut.

Classification of seizure types was based on a review of all medical records and direct discussion of each case where indicated with attending physicians, nurses, or with the SE Research Team (SERT). In many cases, reviewing the initial admission history and presentation

of the patient to the emergency room or on the hospital floor did not clearly identify the details of the seizure presentation. The accuracy of the seizure history was greatly improved by a member of the SERT being able to review the case within 24 hours of diagnosis and interviewing family, physicians, nurses, and emergency personnel within that time period. Prospective data collection facilitated our ability to document partial onset with secondarily generalized seizures. Without this type of input, over one-half of the partial onset of P2G cases would be missed.

Race

White was defined as Caucasians. Black included primarily African Americans. Other included mostly Hispanic and Asian ancestry.

Human Subjects

Patients to be studied will be identified in the GRMA SE Data Base following admission to one of the hospitals for SE or development of SE in one of the hospitals in the GRMA. There are approximately 210-230 patients per year presenting with SE in the GRMA, both male and female and both adult and pediatric age groups. Pediatric patients will be admitted to the hospital for treatment of SE. Patients admitted to MCV that require admission to the Neuroscience ICU for treatment of prolonged SE or coma will be monitored with our sophisticated ICU monitoring system.

All studies or evaluations performed in this project are based on the normal care provided by the attending physician based on a consensus treatment and evaluating protocol agreed upon by physicians participating in the study. Appropriate Consent will be obtained for any procedure, treatment, or evaluation that is not part of standard treatment methods under IRB approval. Complete confidentiality of records will be maintained.

There are no potential risks (physical, psychological, social, legal or other) in gathering the data. The treatment of the patients will follow accepted clinical practices or protocols as accepted by all MCV and Community physicians treating patients for SE in the GRMA, and which have been approved by the IRB.

No potential risks are anticipated. The patients are receiving medical care already and will be cared for in the normal fashion for routine care of if any complications develop.

Patients will benefit by knowing that they are contributing to a better understanding of SE. Society in general will benefit from potentially new insights into the pathogeneses and course of SE.

RESULTS

There are a total of 2,220 patients included in this analysis. The frequencies are tabulated in Table 2. One thousand four hundred and seventy-five patients (66.4%) were included in the young group which included ages from 30 days up to but not including age 60. The elderly group was comprised of 745 patients (33.6%) and included ages from age 60 and above. Table 2 includes a breakdown of specific age groups in the data set. There were 1,035 females comprising 46.6% of the total population and 1,185 males at 53.4%. Of the 19 SE type groups, the most frequent type of status epilepticus was the group partial seizures with secondary generalization (P with 2 G) at 41.2%. The second most frequent seizure type was generalized tonic-clonic activity (TC) at 22.8%. When the 19 SE type groups were categorized into four major seizure types (SE Type), generalized tonic-clonic seizures accounted for 67.8 of cases, partial seizures accounted for 20.3 of cases and non-convulsive seizures were seen in 6.2%.

African American patients accounted for the largest percentage of patients in this data set at 57.9%. White patients accounted for 35.8% and other races, predominantly Hispanic, accounted for 7 %. The etiology of SE was categorized into seven broad sub-groups. Our data included most of the major etiologies observed in previous publications. The most common etiologies were in the non CNS and CNS acute categories at 21 and 22 %. Low anticonvulsant levels and remote symptomatic etiologies were also seen frequently. Time to treatment of SE was 47.3% in the less or equal than 60 minutes and 52.7 % in those patients treated after 60 minutes. Mortality of the entire group was 12.4%.

Table 3 demonstrates the crude analysis in the elderly patients versus the young patients. The elderly patients were 60.1% Black, 34.1% White and 5.8% Other versus 55.7%, 36.6 and 7.6% respectively in the young patients (Figure 3). Female patients accounted for 54% in the elderly group versus 42% in the young group. Male patients accounted for 46% in the elderly group versus 58% in the young group (Figure 3). Blacks were significantly more likely to be found in the elderly group (OR 1.37 CI 1.04, 1.82) as were males (OR 1.84 CI 1.42, 2.38). Generalized seizures accounted for the largest percentage of seizure types for both groups 59.4% in the elderly group and 72.2% in the young group. Figure 4 illustrates the other SE types by age category. In the crude analysis, no SE type was significantly more frequent in the elderly group when compared to the young group. Analysis of the major etiologies revealed that NON CNS acute (OR 0.44 CI 0.28, 0.69) and OTHER (OR 0.35 CI 0.14, 0.89) were less common in the elderly group and withdrawal (OR 1.95 CI 1.07, 3.55) more common in the elderly. The percentage of elderly patients treated within 60 minutes was 50.8%. This contrasts with the 45% of younger patients who were treated within 60 minutes. However, this difference was not statistically significant. There was a difference in mortality between the two groups. In the

elderly group, mortality was 20.9% compared to 8.1% in the elderly group. The elderly were significantly more likely to die within 30 days than was the young group (OR 3.18 CI 2.33, 3.9).

To determine possible risk factors for mortality, a univariate logistic model was utilized. Table 4 illustrates the results of this analysis. The elderly were 3 times more likely to die than the young group. White patients had a significantly higher mortality than blacks and the other group had a lower mortality than the referent group. Patients with the etiology hypoxia/anoxia had an approximately 18 times the risk of dying than the referent group. Other etiologies with a significantly higher risk of dying include CNS Acute, and non CNS. Patients with nonconvulsive SE had over a twofold increase in mortality when compared to the partial group.

The use of univariate analysis to determine correlations have been described above. However, the relationship between possible predictive indicators cannot be completely evaluated by univariate analysis and requires a more sophisticated model. In the logistic regression model, mortality, after adjusting for race, gender, etiology, SE type, treatment group and age group was analyzed. The results are tabulated in Table 5. The results of the logistic model demonstrate that there is increased mortality in the white group, decreased mortality in males and increased mortality in the hypoxia/anoxia, CNS acute, non CNS and remote groups.

Increasing age was also significantly associated with mortality. The age groups ranged from 0 to (but not including) age 1 (referent group), age 1 to (but not including) age 20 (OR 0.41 CI 0.17, 0.99), age 20 to 34 (OR 2.67 CI 1.10, 6.48), age 34 to 49 (OR 3.23 CI 1.47, 7.09), Age 49 to 65 (OR 3.27 CI 1.57, 6.81), age 67 to 75 (OR 6.10 CI 2.88, 12.90), age 75 and above (OR 7.79 CI 3.55, 17.07). Figure 6 demonstrates the odds ratios and the ages graphically.

DISCUSSION

There are a few studies which have addressed status epilepticus in the older population. Most of these studies have been retrospective in nature and collection was either by chart review or discharge codes. The strength of the current study is that patient information is obtained from a prospective database, with the discharge diagnosis being reviewed by neurologists skilled in being able to classify seizure types. This study, comprising a total of 2,220 patients, is also the largest database examining SE, currently available. The major limitations of the study stems from the fact that comorbidities were not taken into account for this data analysis. However, steps are being taken to include this information in subsequent analysis. The other limitation is that the sample population may not be representative of communities across the United States. Consistency with other studies is good, although, as previously mentioned, most of those studies were retrospective in nature.

CONCLUSION

This study analyzed several determinants of mortality in SE. Both univariate and multivariate logistic regression analyses were performed to investigate the complex interaction between numerous variables. With the elderly comprising the fastest growing segment of the US population, it is to be expected that SE will become more common, and physicians treating this condition will need to be better informed about its semiology and treatment in this age group. Status epilepticus is associated with a high mortality in the older population, partially because of the increase of susceptibility of these patients to develop systemic metabolic diseases, strokes and other etiologies that may lead to increased mortality. The present study demonstrates that there is a significant increase in mortality in the older age groups. This increased mortality

increases as age increases. In the adjusted model, mortality is also significantly increased in the nonconvulsive seizure group. There is also a statistically significant association of etiology with mortality in SE. Previous studies have suggested that mortality in SE was associated with etiology, based on empirical analysis from clinical studies. Our results show that mortality was significantly correlated with specific SE etiologies. Certain etiologies such as alcohol withdrawal and AED discontinuation were associated with low mortality. Etiologies including anoxia, acute CNS causes and acute non CNS causes are associated with a high mortality.

Since etiology and age play such a large role in the mortality of SE, treatment modalities to better manage certain etiologies may decrease mortality in the older population.

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APPENDIX A - QUESTIONNAIRE

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Hospital	_____	_ _ _ _ _ _
SE Began?	1 Outside of the hospital 2 In transit to hospital 3 In the hospital 8 Other (_____) 9 Unknown	___
EMS data Transported by squad?	0 No, 1 Yes, 8 N/A, 9 Unk	___

If no or not applicable to above leave all other EMS questions blank

If transported by squad (if times unknown leave blank)	Squad called Squad enroute Squad arrived Arrive Hospital	_ _ / _ _ / _ _ _ _ : _ _ _ _ / _ _ / _ _ _ _ : _ _ _ _ / _ _ / _ _ _ _ : _ _ _ _ / _ _ / _ _ _ _ : _ _
Level of consciousness (worst or lowest level)	1 Awake & Oriented 2 Awake & Disoriented 3 Not awake, arrousable 4 Unresponsive 5 Seizing 8 Other (_____) 9 Unknown/Not tested	___
Pupils	1 Normal, 2 R > L 3 L > R 4 Dilated 5 Constricted 6 Unreactive 8 Other (_____) 9 Unknown/Not tested	___

Study Intake page 1 - 2

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Hospital Unit 1 NSICU 2 Neurology/floor 3 Neurology/Intermediate Unit 4 Neurosurgery 5 EMU 6 Non-Neuro ICU(_____) 7 Non-Neuro Intermediate Unit (_____) 8 Emergency 9 PICU 10 Pediatric Intermediate Unit 11 Pediatric floor 12 Medicine Floor 13 Surgery floor 14 Psych 15 Rehab 16 Oncology/Hospice 17 NICU 18 Int. NICU 98 Other (_____) 99 Unknown	SE start _ _	SE service _ _	At discharge _ _
Hospital Service 1 Neurology 2 Neurosurgery 3 Pediatrics 4 Emergency 5 Medicine 6 Surgery 7 Rehab 8 Psychiatry 9 OB/GYN 10 Oncology 11 Other (_____) 12 Neonatology 99 Unknown	SE start _ _	SE service _ _	At discharge _ _

SE recognized/diagnosed by ?	0 No, 1 Yes, 8 N/A, 9 Unknown	__ EMS __ ED MD __ Neuro Res. __ Neuro Attend __ ICU nurse __ Floor nurse __ ED nurse __ EMU nurse __ EEG tech __ non-Neuro MD __ Other (_____)
Neuro Consult ?	0 No, 1 Yes, 8 N/A, 9 Unknown	__
Transfer to neuro within 24 hours		__
DNR/Comfort Care ?	0 No, 1 Yes,8 N/A, 9 Unknown	__
Was SE aggressively treated ?		__
Deceased ?		__
Autopsy		__
Date/time of death		_ _ / _ _ / _ _ _ _ _ _ : _ _
Date/time of discharge		_ _ / _ _ / _ _ _ _ _ _ : _ _
1 Home 2 Rehab 3 Nsg Home 4 Adult Home 8 Other (_____) 9 Unknown	Admitted from ?	__
	Discharged to ?	__

1 Dead 2 Persistent Vegetative State/ Coma 3 Severe disability (conscious but disabled). Dependent for ADL's 4 Moderate disability (disabled but independent). Patient is independent for ADL's 5 Good recovery (may be minor neurologic or psychologic deficits)/ Normal 9 Unknown	Prior to Admission Prior to SE ? At discharge ? At 30 days post SE ? (if discharge < 30days, leave blank)	__ __ __ __

		Pre	Discharge
Does patient have an impairment due to SE at discharge?	0 No, 1 Yes, 2 Possible, 8 N/A, 9 Unknown		__
Cognitive/Mental impairment		__	__
Physical impairment		__	__
Developmental impairment		__	__

To be followed by ?	MD code	_ _ _ _
Followup location ?	1 Nelson Clinic, 2 Randolph Minor, 3 Stony Point, 4 Peds Neuro, 5 CSS clinic, 6 VA, 7 Other MCV (_____), 8 NA, 9 Unknown, 10 Community Neurology Office, 11 Other Comm. (_____) 12 Outside of SEDS catchment area (_____)	__

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Marital Status	1 Married 2 Widowed 3 Divorced 4 Separated 5 Single 6 "Common law" marriage 9 Unknown	___
Living Situation	1With others 2 Alone 3 Institutionalized 4 Homeless 8 Other 9 Unknown	___
Currently Employed ?	0 No, 1 Yes, 8 N/A, 9 Unknown	___
Gainfully Employed ?	1 Never employed 2 Employed 3 Unemployed, 4 Disabled 5 Retired, 8 N/A 9 Unknown	___
Occupation (see code sheet)	_____	_ _ _ _
Cigarette Smoker ?	0 Never Smoked 1 Former Smoker 2 Currrent Smoker 9 Unknown	___ __. __ packs/day x __ __ years
Alcohol Consumption ?	0 Never drank 1 Former abuser 2 Current "social" user 3 Current abuser 4 Former drink NOS, 9 Unk	___
Recreational Drug Use	0 Never used 1 Former user 2 Current User 9 Unknown	___ amphetamine ___ cocaine/crack ___ heroin ___ IVDA ___ marijuana ___ other (_____)
Insurance 0 No, 1 Yes, 8 N/A, 9 Unknown	Self Commercial Medicare Medicaid	___ ___ ___ ___
MCV codes (01, 03, 04) →	Percent write off	___ _ _ _ _ %

If percent write off N/A then leave _ _ _ % blank

Completed by _____ date _ _ / _ _ / _ _

Demographics page 1-1

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

0 No, 1 Yes, 8 N/A,9 Unknown

			Pre Adm	During Adm
01 Neurologic		A-V malformation/ Cerebral aneurysm		
		Anoxic/Hypoxic Encephalopathies		
		Carotid stenosis (>60%)		
	01.01	Cerebral hemorrhage		
		CNS lesion NOS		
		CNS sarcoidosis		
	01.02	CVA (_____)		
		Dementia		
		Encephalopathy NOS		
	01.03	Febrile convulsions		
		Genetic syndromes (_____)		
	01.04	Head trauma		
	01.05	Hydrocephalus/shunt		
		Inborn errors of metabolism		
		Intraventricular hemorrhage		
		Metabolic encephalopathy		
		Neurodegenerative (_____)		
		Neurofibromatosis		
		Neurosurgery (_____)		
		Non-genetic syndrome (_____)		
	01.99	Other (_____)		
		Proprionic acidemia		
		Quad/hemiparesis (_____)		
	01.06	Spinal cord abnormalities (_____)		
		Static encephalopathy		
	01.07	TIA		
		Tuberous sclerosis		
	01.08	Vasculitis		
02 Developmental/ Psychiatric		ADHD/ADD		
		Behavioral/Emotional		
		Cognitive delay		
		Developmental/Motor delay		
		Learning disability		
		Mental retardation		
	02.99	Other (_____)		
	02.01	Psychiatric condition (_____)		
03 Infectious Diseases		AIDS/ HIV+		
	03.01	CNS abscess		
		CNS TB		
		Congenital CMV (TORCH)		
	03.02	Encephalitis		
	03.03	Febrile illness NOS (_____)		
		Group B strep meningoencephalitis		
	03.04	Meningitis		
		Neuro-Syphilis		
	03.99	Other (_____)		
	03.05	Sepsis		
		TB (other than CNS)		

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

0 No, 1 Yes, 8 N/A, 9 Unknown

			Pre Adm	During Adm
04 Cardiovascular	04.01	Arrhythmia - Atrial		
	04.02	Arrhythmia - Ventricular		
		Arrhythmia NOS (_____)		
	04.03	Cardiac arrest		
		Cardiac surgery (_____)		
		Congenital heart disease		
		Coronary artery disease		
	04.04	Hypertension		
	04.05	Hypotension		
	04.06	MI (_____)		
	04.99	Other (_____)		
	04.07	Pacemaker		
		Peripheral vascular disease		
05 Respiratory	05.01	ARDS		
		Asthma		
		COPD		
	05.02	ECMO		
		Hyaline membrane disease		
	05.03	Meconium aspiration		
	05.99	Other (_____)		
	05.04	Pneumonia		
		Pulmonary/bronchopulmonary dysplasia		
	05.05	Resp. arrest/anoxia		
	05.06	Resp. failure/hypoxia		
		Sleep apnea		
	05.07	Ventilator dependent		
06 GI		Cirrhosis		
	06.01	GI hemorrhage		
	06.02	Hepatitis		
		Jaundice/kernicterus		
	06.03	Liver failure		
	06.99	Other (_____)		
	06.04	Pancreatitis		
		Peptic Ulcer Disease		
07 GU		Benign Prostatic Hypertrophy		
	07.99	Other (_____)		
	07.01	Pyelonephritis		
	07.02	UTI		
08 Metabolic	08.01/08.02	↓/↑ ammonia (≤ 50)		
	08.03/08.04	↓/↑ calcemia (8 - 12)		
0=absent	08.05/08.06	↓/↑ Cl (90 - 120)		
1=hypo	08.07/08.08	↓/↑ CO_2 (15 - 35)		
2=hyper	08.09/08.10	↓/↑ glycemia (50 - 200)		
3=both	08.11/08.12	↓/↑ kalemia (3.0 - 6.0)		
9=unknown	08.13/08.14	↓/↑ magnesia (1.0 - 3.0)		
	08.15/08.16	↓/↑ natremia (130 - 150)		
	08.17/08.18	↓/↑ phosphatemia (2.0 - 5.0)		
	08.19/08.20	↓/↑ thyroidism		
		Insulin dependent diabetes		
		Non-insulin dependent diabetes		
	08.99	Other (_____)		

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

0 No, 1 Yes, 8 N/A, 9 Unknown			Pre Adm	During Adm
09 Hematology		Chronic anemia		
	09.01	Coagulopathy		
	09.02	DIC		
	09.99	Other (_____)		
		Sickle cell		
10 Malignancies		Brain		
		Endocrine		
		GI		
		GU		
		Heme/Bone marrow/Bone		
		Other(_____)		
		PNS/Spinal Cord		
		Respiratory		
11 Renal	11.01	Acute renal failure/ insufficiency		
		Chronic renal failure/ insufficiency		
	11.02	Dialysis		
	11.99	Other (_____)		
12 Etoh/Drugs		Drug dependency (_____)		
	12.01	Drug OD (_____)		
	12.02	DT's/ Etoh withdrawal		
		Etoh dependency		
	12.99	Other (_____)		
	12.03	Toxic /poisoning (_____)		
13 Other	13.01	_____		
	13.02	_____		
14 Ob/Gyn		Complications of preg (_____)		
	14.99	Other (_____)		
	14.01	Toxemia		
15 Connective Tissue		Arthritis		
	15.01	Gout		
	15.99	Other _____		
	15.02	Systemic Lupus Erythematosus		

Surgery this admission ?	0 No, 1 Yes, 9 Unknown	__
Surgery date/time (If multiple surgeries, date/time of surgery closest to SE)		__ _ / __ _ / __ __ : __ __
Type of Surgery	1=Neuro (16.01), 2=Cardiac (16.02), 3=Other (16.99), 4= Multiple Surg (16.03), 8=N/A, 9=Unknown	__
Did the patient experience multi-system organ failure ?	0 No, 1 Yes, 8 N/A, 9 Unknown	__
If yes to above, how many systems were affected ? (refer to codes, exclude SE/seizures)		__ __

Please list the order or progression of acute medical conditions in relation to SE episode(s) this admission:
00.00 SE, 00.88 seizures, not SE, 77.77 Life Support withdrawn, 88.88 Sequence underterminable at this time (1st year),
99.99 Unkown/undeterminable sequence

__ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ →
__ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ →
__ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ → __ __ . __ __ →

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

AED Therapy prior to Current SE		0 No, 1 Yes, 9 Unknown	__	
If yes to above list AED's	Refer to AED list for abbreviations	Current _ _ _ _ _ _ _ _ _ _ _ _	_ _ _ _ _ _ _ _ _ _ _ _	Past _ _ _ _ _ _ _ _ _ _ _ _
Age at first AED use	Estimated (years)		_ _ . _ _	

List all non-AED drug therapy in first column then code the first ten letters of the drug in the second column, indicate when the drug was used in the last three columns using: 0=No, 1=Yes, 9=Unknown

		Before SE	During SE	After SE
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
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	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
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	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__
	_ _ _ _ _ _ _ _ _ _	__	__	__

Could SE have been related to or caused by drugs or medications (other than DAED) ?	0=No, 1=Yes (definitely) 2=Probably, 3=Possibly, 9=Unknown	__
If yes, to above what drug/ medication	Code first 10 letters of medication	_ _

History Page 4 - 8

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Seizure History

# Types __ / __ Seizure Type or ___ No seizure hx	1 L brain simple partial, 2 R brain simple partial, 3 Complex Partial, 4 Partial w/ 2nd Generalization, 5 Abscence 60 Myoclonic - generalized, 61 Myoclonic - focal 7 Tonic, 8 Tonic-Clonic, 9 Atonic, 10 Other (_____) 11 L brain simple partial w/ LOC, 12 R brain simple partial w/LOC 13 Bilateral independent noncon, 14 Subtle/partial 15 Subtle/generalized, 16 Bilateral Independent Convulsive 17 Infantile Spasms, 96 Partial w/ 2nd Gen nonconvulsive 97 Partial nonconvulsive, 98 Generalized nonconvulsive, 99 Unknown	__ __
Current frequency	1 daily, 2 weekly, 3 less than yearly, 4 yearly 5 lifetime, 6 remote history (>1yr ago), 9 Unknown	___ times / ___
Frequency changed in last 6 months ?	0 No, 1 Yes, 9 Unknown	___
If yes to above	0 decreased, 1 increased, 8 NA, 9 unknown	___
Seizure Precipitants - Etiology (Refer to precipitating causes of SE codes)	_____ _____ _____ _____	___ ___ ___ ___ ___ ___ ___ ___

Fill out this section only once per package

Diagnosed with Epilepsy Syndrome ?	0 No, 1 Yes, 8 N/A, 9 Unknown	___
BINS (Benign Idiopathic Neonatal Sz)		___
Benign Familial Neonatal Seizures		___
Febrile		___
JME		___
Lennox-Gastaut		___
Rolandic (BECTS)		___
West Syndrome		___
Other (_____)		___
Family History of seizure ?	0 No, 1 Yes (definite),	___
Twin ?	2 Suspected hx	___
Twin has seizure history ?	8 NA	___
Patient History of seizure ?	9 Unknown	___
Patient age of first seizure ?	Estimated (years/months)	__ __ . __ __
Patient History of SE ?	0 No, 1 Yes, 8 N/A, 9 Unknown	___
Number of previous SE events		__ __
Age of first SE	Estimated (years/months)	__ __ . __ __
Previous SE verified by	1 Family, 2 SE team, 3 Not verified	___

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

1 L brain simple partial, 2 R brain simple partial, 3 Complex Partial, 4 Partial w/ 2nd Gen, 5 Abscence, 60 Myoclonic- generalized, 61 Myoclonic - focal , 7 Tonic, 8 Tonic-Clonic, 9 Atonic, 10 Other, 11 L brain simple partial w/ LOC, 12 R brain simple partial w/LOC, 13 Bilateral independent noncon, 14 Subtle/partial, 15 Subtle/generalized, 16 Bilat Ind. Convulsive, 17 Infantile Spasms 96 Partial w/ 2nd Gen noncon, 97 Partial noncon, 98 Generalized noncon, 99 Unknown

Current SE Types	(Use SZ types list)	_ _ → _ _ → _ _ → _ _
If multiple SE types, was it ?	0 pure progression, from one type to another 1 mix of types/alternating (code predominant in 1st space) 2 difficult to determine 8 NA, 9 unknown	_
Date/Time of SE onset		_ _ / _ _ / _ _ _ _ : _ _
End of Clinical/Overt sz's	blank if N/A	_ _ / _ _ / _ _ _ _ : _ _
End of Electrographic sz's	blank if N/A	_ _ / _ _ / _ _ _ _ : _ _
Type of SE	1 Continuous, 2 Intermittent, 3 Intermittent/drug, 4 Nearly continuous, 5 Cont →Int, 6 Int →Cont, 7 Mixed Int & Cont, 9 Unknown	_
If intermittent, est ictal time	(hours:minutes)	_ _ : _ _
Etiology of SE (Refer to precipitating causes of SE codes)	_____ _____ _____ _____	_ _ _ _ _ _ _ _ _ _ _ _
Objective clinical manifestations ?	0 No, 1 Yes, 9 Unknown	_
If yes, specify for each 0 No, 1 Yes, 9 Unknown or 0 No, 1 Left, 2 Right 3 Both, 9 Unknown	Focal Motor Generalized Clouding of consciousness Loss of consciousness Versive or Contraversive Automatisms Subtle (_____) Other (_____) Eye deviation	_ _ _ _ _ _ _ _ _
Post-ictal coma	0 No, 1 Yes,	_
Post-ictal coma affected by ?	8 NA, 9 Unknown	_ profound diseases/injuries _ paralytic agents/ anesthesia
Start of post-ictal coma		_ _ / _ _ / _ _ _ _ : _ _
End of post-ictal coma	blank if N/A	_ _ / _ _ / _ _ _ _ : _ _
Was pt in coma pre SE ?	0 No, 1 Yes, 9 Unknown	_
Recurrent episode of SE ?		_
If yes, # of recurrences		_ _
Recurrent episode of SE w/in 24 hrs of initial SE ?	0 No, 1 Yes, 8 N/A, 9 Unknown	_
Any seizures post SE during this admission	0 No, 1 Yes, 8 N/A, 9 Unknown	_

If yes to above please complete seizure flowsheet

Assigned ID: ____

pg __ of __   __ for no sz post SE   SE date __/__/____

Code Clinical Seizures post SE only, if EEG is hooked up please use EEG form to document seizures

Date of Onset MM/DD/YY	Time of Onset HH:MM (24 hour clock)	Date of Cessation MM/DD/YY	Time of Cessation HH:MM (24 hour clock)	Type (refer to list below)
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__
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__/__/__	__:__	__/__/__	__:__	__
__/__/__	__:__	__/__/__	__:__	__

1 L brain simple partial, 2 R brain simple partial, 3 Complex Partial, 4 Partial w/ 2nd Gen, 5 Abscence, 60 Myoclonic- generalized, 61 Myoclonic - focal , 7 Tonic, 8 Tonic-Clonic, 9 Atonic, 10 Other, 11 L brain simple partial w/ LOC, 12 R brain simple partial w/LOC, 13 Bilateral independent noncon, 14 Subtle/partial, 15 Subtle/generalized, 16 Bilat Ind. Convulsive, 17 Infantile Spasms 96 Partial w/ 2nd Gen noncon, 97 Partial noncon, 98 Generalized noncon, 99 Unknown

History page 7 - 8

Assigned ID: ___ ___ ___

SE date ___/___/_____

SE Medications

page ___ of ___ Route 1=IV, 2=PO, 3=IM, 4=IO, 5=PR, 6=ET, 9=Unknown

Name of Drug	Dose (mg)	Route	Date of initiation MM / DD / YY	Time of Initiation 24 hour time
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
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_____	_____.__	__	__/__/__	__:__
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_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__
_____	_____.__	__	__/__/__	__:__

History page 8 - 8

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _ _

Leave blank if N/A	Before SE _ _ / _ _ / _ _ _ _ : _ _	1st During SE _ _ / _ _ / _ _ _ _ : _ _	1st After SE _ _ / _ _ / _ _ _ _ : _ _
Pulse	_ _ _	_ _ _	_ _ _
BP	_ _ _ / _ _ _	_ _ _ / _ _ _	_ _ _ / _ _ _
Resp (assist/spont)	_ _ / _ _	_ _ / _ _	_ _ / _ _
Temperature	_ _ _ . _	_ _ _ . _	_ _ _ . _
Weight (pounds)	_ _ _ . _		
Height/length (inches)	_ _ . _		
Head circum (cm)	_ _ . _		
Handedness	1=R, 2=L, 3=A, 9=Unk	_	

First Neurologic Exam after SE *(If test not performed leave all entries blank)*

Test completed _ Date/Time test performed _ _ / _ _ / _ _ _ _ : _ _

Medications prior to exam 0 No, 1 Yes, 8 N/A, 9 Unknown	Benzodiazapines Paralytics/Vecuronium Morphine Barbituates		_ _ _ _
Pupil size (mm)	If unknown record (9.9)		Right _ . _ Left _ . _
Pupillary light responses 0 No, 1 Yes, 8 N/A, 9 Unknown	Direct Consensual		_ _
Oculocephalic Response (Gaze)	0 Absent, 1 Impaired, 2 Suppresed (Voluntary), 3 Full , 8 N/A, 9 Unknown		_ _
Gaze Preference	0 Absent, 1 Right, 2 Left 8 N/A, 9 Unknown		Baseline _ Post SE _
Visual fixiation	0 No, 1 yes, 8 N/A, 9 Unknown		_
Gag reflex	0 Absent, 1 Normal, 9 Unknown		_

			Right	Left
Pathologic Reflexes	0 Absent, 1 Present, 9 Unknown	Babinski	_	
Deep Tendon Reflexes	0 Absent, 1 Hypoactive 2 Normal, 3 Hyperactive 4 Hyperactive with clonus 8 N/A , 9 Unknown	Biceps Jerk Triceps Jerk Knee Jerk Ankle Jerk	_ _ _ _	_ _ _ _
Motor Strength	0 Flaccid , 1 Trace, 2 Poor 3 Fair, 4 Good ,5 Normal, 9Unk	Upper Extremity Lower Extremity	_ _	_ _
Motor Tone	0 Flaccid, 1 Hypotonic, 2 Norm. 3 Hypertonic, 9 Unknown	Upper Extremity Lower Extremity	_ _	_ _
Sensory Deficit (Pain)	0 Absent, 1 Present, 8 N/A, 9 Unk	Upper Extremity Lower Extremity	_ _	_ _

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

page _ _ / _ _
Test completed _

		Date Time	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ : _ _
Location	1 Ambulance 2 ER 3 ICU 4 Floor 5 Other _____ 6 Baseline 9 Unknown					
Eye Opening	9 Unknown 4 Spontaneous 3 To sound 2 To pain 1 None					
Motor Response	9 Unknown 6 Follow commands 5 Localizes stimulus 4 Withdraws 3 Flexion posturing 2 Extension posturing 1 No movement					
Verbal Response (see below for ≤ 3 yrs)	9 Unknown 5 Oriented 4 Confused 3 Words 2 Sounds 1 None					
Intubated ?	0 No, 1 Yes,					
Paralzying agents ?	9 Unknown					

For ≤ 3 yrs use these responses for verbal category:
9 Unknown
5 smiles, oriented to sound, follows objects, interacts
4 crying/consolable; interacting inappropriately,
3 crying/inconsistently consolable, moaning
2 crying/inconsolable; irritable, restless
1 no response

Glasgow Coma Scale page 1

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

0 No, 1 Yes, 8 N/A, 7 done, results not available, 9 Unknown

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ : _ _

Blood Chemistries

Sodium (meq/l)	_ _ _ _
Potassium (meq/l)	_ _ . _
Chloride (meq/l)	_ _ _ _
CO_2 (meq/l)	_ _ _ _
Glucose (mg/dl)	_ _ _ _
BUN (mg/dl)	_ _ _ _
Creatinine (mg/dl)	_ _ _ . _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ : _ _

Serum Electrolytes

Urate (mg/dl)	_ _ _ . _
Inorg. Phos. (mg/dl)	_ _ . _
Magnesium (mg/dl)	_ _ _ . _
Calcium (mg/dl)	_ _ _ . _
Iron (mg/dl)	_ _ _ . _
Total protein (mg/dl)	_ _ _ . _
Albumin (mg/dl)	_ _ . _
Total bilirubin (mg/dl)	_ _ _ . _
Cholesterol (mg/dl)	_ _ _ _
ALP (IU/L)	_ _ _ _
AST (IU/L)	_ _ _ _
ALT (IU/L)	_ _ _ _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ : _ _

Serum

Ammonia (µm/L)	_ _ _ _ _

Test completed __

Date/Time test performed	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _
CK (U/L)	_ _ _ _	_ _ _ _	_ _ _ _
MM (%)	_ _ _	_ _ _	_ _ _
MB (%)	_ _ _ _	_ _ _ _	_ _ _ _

Test completed __

	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _
LD1 (%)	_ _	_ _	_ _
LD2 (%)	_ _	_ _	_ _
LD3 (%)	_ _	_ _	_ _
LD4 (%)	_ _	_ _	_ _
LD5 (%)	_ _	_ _	_ _
LD (U/L)	_ _ _ _	_ _ _ _	_ _ _ _

Laboratory page 1 - 3

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

0 No, 1 Yes, 8 N/A, 7 done, results not available, 9 Unknown

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Hematology

WBC (10/mm³)	_ _ _ . _
Hemoglobin (gm/dl)	_ _ _ _ . _
Platelets (10/mm³)	_ _ _ _
Neutrophils (%)	_ _ _ _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Coagulation studies

PT (sec) / Control (sec)	_ _ _ / _ _
PTT (sec) / Control	_ _ _ / _ _
INR	_ _ . _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Urinalysis

Glucose	_ _
Ketone	_ _
Protein	_ _
WBC	_ _
RBC	_ _
Blood	_ _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Arterial blood gases

Supplemental Oxygen	_
Mechanical Ventilation	_
pO_2 (mm Hg)	_ _ _ _
pCO_2 (mm Hg)	_ _ _ _
pH	_ . _ _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Serum

Lactate (mM/L)	_ _ . _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Serum

Cortisol (ug/dl)	_ _ . _

Test completed __ Date/Time test start _ _ / _ _ / _ _ _ _ _ : _ _

Date/Time test stop _ _ / _ _ / _ _ _ _ _ : _ _

Urine Catecholamines

Creatinine (mg/dl)	_ _ . _
Norepinephrine (µg/dl)	_ _ . _
Epinephrine (µg/dl)	_ _ . _
Dopamine (µg/dl)	_ _ . _
Urine volume (ml)	_ _ _ _

Test completed __ Date/Time test performed _ _ / _ _ / _ _ _ _ _ : _ _

Thyroid Profile

T4 total (µg/dl)	_ _ . _
TU (µg/dl)	_ _ . _
STI (µg/dl)	_ _ . _

Assigned ID: _ _ _ _

SE date _ _/_ _/_ _ _ _

0 No, 1 Yes, 8 N/A, 7 done, results not available, 9 Unknown

Test completed _ Date/Time test performed _ _/_ _/_ _ _ _:_ _

Appearance	0 Abnormal, 1 Normal, 9 Unk	_
Color	0 Abnormal, 1 Normal, 9 Unk	_
CSF	RBC /mm³ WBC/mm³ Polys/mm³ Lymphs/mm³ Monos/mm³ Other/mm³ ________	_ _
	Glucose (mg/dl) Protein (mg/dl)	_ _ _ _ _ _ _ _ _ _
	Lactate (mm/L) Pyruvate (mm/L)	_ _ _ _ . _ _ _ _ _ . _
Pathogenic Organisms Present in CSF ?	0 No, 1 Yes, 8 N/A, 9 Unknown	_ _ : _ _ _ _ _ : _ _ _

Test completed _ Date/Time test performed _ _/_ _/_ _ _ _:_ _

Toxicology	Cocaine	_ _
	Amphetamine	_ _
	Opiate	_ _
	Barbituates	_ _
	Benzodiazapine	_ _
	Salicylates	_ _
	Acetaminophen	_ _
	Other (____________)	_ _

Test completed _ Date/Time test performed _ _/_ _/_ _ _ _:_ _

Etoh (mg/dl)	_ _ _ _ _

Test completed _

AED'S *(before, during and after SE)*

Drug	Level (mg/dl)	Date/Time
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _
_ _ _	_ _ _ . _	_ _/_ _/_ _ _ _:_ _

Completed by ______________________________ date _ _/_ _/_ _

Laboratory page 3 - 3

Electroencephalogram Interpretation

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Date test started _ _ / _ _ / _ _ _ _ Time test started _ _ : _ _ (24 hr clock)

Date test stopped _ _ / _ _ / _ _ _ _ Time test stopped _ _ : _ _ (24 hr clock)

Total minutes of EEG recorded		_ _ _
State of Patient at time of EEG 0 No 1 Yes 9 Unknown	Awake Asleep/Drowsy Unresponsive Other (_____)	_ _ _ _
Background Activity	0 Normal 1 Abnormal 9 Uninterpretable	_
Predominant Background Frequency (hz)		_ _ . _ - _ _ . _

If EEG is Normal or Uninterpretable, do not code further. If abnormal please fill out the rest of EEG form

Pattern	Grade I, II, III	Location L,R, Gen, Bilat	Specify location, check all that apply
Dysrhythmia	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Dysrhythmia	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Dysrhythmia	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Delta	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Delta	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Depression	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric
Asymmetry	_	_	_ Frontal _ Parietal _ Temporal _ Occipital _ Central _ Hemispheric

Abnormal Activity 0 No, 1 Yes, 8 N/A, 9 Unknown	Before SE	During SE	After SE
Electrographic Seizure(s)	_	_	_
Reactivity (8=not tested)	_	_	_
Triphasics	_	_	_
Spikes	_	_	_
Sharp Waves	_	_	_
Slow Waves	_	_	_
Waves/Spikes	_	_	_
PLEDS/PEDS	_	_	_
Burst Suppresion (2=drug induced)	_	_	_
Other (_____)	_	_	_

If ictal events occur please code on EEG page 2 - 2

EEG page 1-2

Assigned ID: ____

SE date __/__/____

Recorded Ictal Events (please use 24-hour clock to document times)

Seizure number	Start date	Start time	Stop date	Stop time	Duration (min.sec)	Location 1=Right, 2=Left, 3=Bilateral	Clinical Manifestations 0=No, 1=Yes, 3=Paral, 9=Unk	HR
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___
___	__/__/__	__:__	__/__/__	__:__	___.__	_F _P _T _O _C _H _G	_	___

Anticonvulsant Medications given during EEG (please use 24-hour clock to document times)

Date	Medication	Dose	page	time	Medication Effect/Comments
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	
__/__/__	___	____._	___	__:__	

EEG page 2 - 2

Assigned ID: _ _ _ _

pg _ _ of _ _

SE date _ _ / _ _ / _ _ _ _

Date/time test started _ _ / _ _ / _ _ _ _ : _ _ (24 hour clock) Date/time test stopped _ _ / _ _ / _ _ _ _ : _ _ (24 hour clock)

In the 30 minutes after clinical SE stops are there electrographic ictal discharges ? 0=No, 1=Yes, 9=Unk _

									Duration of discharges		Interval between discharges	
Pattern	start date time	stop date time	Morp	Freq	Occur	Contin	Clin man.	Localiz.	min	max	min	max
1	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
2	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
3	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
4	_ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
5	_ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
6	_ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr
7	_ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _		_ _ . _					_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr	_ _ _ . _ msec sec min hr

Morphology: 1=Spike (< 70msec), 2=Sharp wave (70-200msec), 3= Spike + Wave, 4=Repetitive spike, 5=Rhythmic Activity

Occurrence: 1=Rare Isolated, 2=Recurrent Stereotyped, 3=Recurrent Non stereotyped, 4=Periodic/Quasi Periodic, 5=Burst Suppression, 6=Ictal, 7=Other

Continuity: 1=Continuous, 2=Nearly Continuous, 3=Intermittent

Clinical Manifestations: 0=No, 1=Yes, 9=Unk

Localization: (code any and all lobes with side L, R or B first), F=frontal, T=temporal,P=parietal,O=Occipital, C=Central, M=Midline,H=Hemispheric

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _ _

pg _ _ of _ _

Code only first Post SE ECG on this page, subsequent ECG's on page 2-2

	Baseline	During SE	Post SE
Date	_ _ / _ _ / _ _	_ _ / _ _ / _ _	_ _ / _ _ / _ _
Time	_ _ : _ _	_ _ : _ _	_ _ : _ _
Rate (bpm)	_ _ _	_ _ _	_ _ _
PR Interval	0. _ _	0. _ _	0. _ _
QT	_ _ _	_ _ _	_ _ _
QTC Interval	_ _ _	_ _ _	_ _ _
R Axis	_ _ _	_ _ _	_ _ _
QRS Voltage (precordial max, mm)	_ _	_ _	_ _
LVH	_	_	_
SVT	_	_	_
PAC	_	_	_
A. Fib (0 No)	_	_	_
A. Flutter (1 Yes)	_	_	_
Junctional (9 Unknown)	_	_	_
VPB's	_	_	_
V. Tach	_	_	_
V. Fib	_	_	_
Other (_____)	_	_	_
1° AVB	_	_	_
2° AVB I	_	_	_
2° AVB II (0 No)	_	_	_
3° AVB (1 Yes)	_	_	_
IVCD (9 Unknown)	_	_	_
LAFB	_	_	_
LBBB	_	_	_
RBBB	_	_	_
T Waves (1=Flat, 2=Inverted, 3=Peaked)	_	_	_
T Waves (Anatomic Location *)	_	_	_
ST Segments (1=Elevated, 2=Depressed)	_	_	_
ST Segments (mm)	_ _	_ _	_ _
Q Waves (Anatomic Location *)	_	_	_
Poor R Wave progression (0 No, 1 Yes, 9 Unknown)	_	_	_

* 0=Anterior, 1=Inferior, 2=Lateral, 3=Anteroseptal, 4=Anterolateral, 5=Inferolateral, 6=Other, 9=Unknown

Comments:

Electrocardiogram page 1-2

Assigned ID: _ _ _ _

pg _ _ of _ _

SE date _ _ / _ _ / _ _ _ _

Code only Post SE ECG's on this page

Date Time	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _
Rate (bpm)	_ _ _	_ _ _	_ _ _
PR Interval	0. _ _	0. _ _	0. _ _
QT	_ _ _	_ _ _	_ _ _
QTC Interval	_ _ _	_ _ _	_ _ _
R Axis	_ _ _	_ _ _	_ _ _
QRS Voltage (precordial max, mm)	_ _	_ _	_ _
LVH (0 No, 1 Yes, 9 Unknown	_	_	_
SVT	_	_	_
PAC	_	_	_
A. Fib 0 No	_	_	_
A. Flutter 1 Yes	_	_	_
Junctional 9 Unknown	_	_	_
VPB's	_	_	_
V. Tach	_	_	_
V. Fib	_	_	_
Other (_____)	_	_	_
1° AVB	_	_	_
2° AVB I	_	_	_
2° AVB II 0 No	_	_	_
3° AVB 1 Yes	_	_	_
IVCD 9 Unknown	_	_	_
LAFB	_	_	_
LBBB	_	_	_
RBBB	_	_	_
T Waves (1=Flat, 2=Inverted, 3=Peaked)	_	_	_
T Waves (Anatomic Location *)	_	_	_
ST Segments (1=Elevated, 2=Depressed)	_	_	_
ST Segments (mm)	_ _	_ _	_ _
Q Waves (Anatomic Location *)	_	_	_
Poor R Wave progression 0 No, 1 Yes, 9 Unknown	_	_	_

* 0=Anterior, 1=Inferior, 2=Lateral, 3=Anteroseptal, 4=Anterolateral, 5=Inferolateral, 6=Other, 9=Unknown

Comments:

Electrocardiogram page 2-2

Assigned ID: _ _ _ _

SE date _ _ / _ _ / _ _ _ _

#_ _ of _ _

Test completed _ Date/Time test performed _ _ / _ _ / _ _

Hospital		_ _ _ _ _
Study Was	1 MRI, 2 CT, 3=Ultrasound	_
Results	0 Normal for age, 1 Abnormal, 9 Unknown/ Uninterpretable	_
Contrast used ?	0 No, 1 Yes, 9 Unknown	
Shift	0 Absent, 1 Present, 2 Present w/ Herniation, 9 Unk	_

Code side in first space, location in second and third space, age in fourth space, size in fifth space, area in sixth, and lesion in seventh and eighth spaces

Lesion(s):

Side	Loc	Loc	Age	Size	Area	Les	Les	Side	Loc	Loc	Age	Size	Area	Les	Les

Side
1 Right
2 Left
3 Bilateral, 4 Midline

Location (Loc)
01 Frontal
02 Parietal
03 Temporal
04 Occipital
05 ACA
06 Basal Ganglia
07 Cerebellum
08 Hemispheric
09 Internal capsule
10 MCA
11 Medulla
12 Midbrain/Pons
13 PCA
14 Periventric/subcortical
15 Sphenoidal/Clivus
16 Thalamus
17 Extra-axial
18 Ventricular
19 Meningeal, 20 Sella

Age of Lesion
1 New
2 Old
9 Indeterminate/unknown

Size
1 Lacune
2 Small
3 Medium
4 Large
5 Diffuse
9 Unknown

Area
1 Gray matter
2 White matter
3 Both
4 Watershed
8 N/A
9 Unknown

Lesion (Les)
01 A-V Malformation
02 Atrophy
03 Calcifications
04 Congenital Malformation
05 Craniotomy
06 CVA - hemorrhagic
07 CVA - nonhemorrhagic
08 CVA - unknown
09 Cyst
10 Edema
11 Foreign bodies
12 Hemorrhage
13 Hydrocephalus
14 Ring enhancing lesion
15 Shunt
16 Subdural Hematoma
17 Epidural Hematoma
18 Tumor
19 White matter disease
97 Uninterpretable
98 Other
(_____)
99 Unknown

MRI/CT page 1

Assigned ID: _ _ _ _

Upper Extremity Somatosensory Evoked Potential

SE date _ _ / _ _ / _ _ _ _

		Left Median	Right Median
Cortical potential present	0 No, 1 Yes,	_	_
Subcortical potential present	8 N/A, 9 Unknown	_	_
Peripheral potential present		_	_

Median Results

	Left Median Nerve		Right Median Nerve	
Twitch/Stimulus	Twitch Threshold	Stimulus Intensity	Twitch Threshold	Stimulus Intensity
(mamp)	_ _	_ _	_ _	_ _

	Left Median	Right Median
Erb's Point (EP)	_ _ . _ msec	_ _ . _ msec
Stationary Cervical (N13/P13) 0=Absent, 1=Present, 9=Unk.	_	_
Lemniscal (P14)	_ _ . _ msec	_ _ . _ msec
Subcortical (N18)	_ _ . _ msec	_ _ . _ msec
Cortical (N20)	_ _ . _ msec	_ _ . _ msec

Median Interpretation

Normal	0 No, 1 Yes, 8 N/A, 9 Unknown	_
Abnormality consistent with	1=Left, 2=Right, 3=Both	_
	1=Central, 2=Peripheral, 3=Indeterminate	_

		Left Ulnar	Right Ulnar
Cortical potential present	0 No, 1 Yes,	_	_
Subcortical potential present	8 N/A, 9 Unknown	_	_
Peripheral potential present		_	_

Ulnar Results

	Left Ulnar Nerve		Right Ulnar Nerve	
Twitch/Stimulus	Twitch Threshold	Stimulus Intensity	Twitch Threshold	Stimulus Intensity
(mamp)	_ _	_ _	_ _	_ _

	Left Ulnar	Right Ulnar
Erb's Point (EP)	_ _ . _ msec	_ _ . _ msec
Stationary Cervical (N13/P13) 0=Absent, 1=Present, 9=Unk.	_	_
Lemniscal (P14)	_ _ . _ msec	_ _ . _ msec
Subcortical (N18)	_ _ . _ msec	_ _ . _ msec
Cortical (N20)	_ _ . _ msec	_ _ . _ msec

Ulnar Interpretation

Normal	0 No, 1 Yes, 8 N/A, 9 Unknown	_
Abnormality consistent with	1=Left, 2=Right, 3=Both	_
	1=Central, 2=Peripheral, 3=Indeterminate	_

Assigned ID: _ _ _ _

Brainstem Auditory Evoked Potential

SE date _ _ / _ _ / _ _ _ _

		Left Ear	Right Ear
Wave I present	0 No, 1 Yes, 8 N/A, 9 Unknown	_	_
Wave III present		_	_
Wave V present		_	_

Results

	Left Ear	Right Ear
Click Threshold	_ _ _ db	_ _ _ db
Latencies (_ _ . _ /sec) at	_ _ _ db	_ _ _ db

Central (Brainstem) Function

	Left Ear	Right Ear
Wave I (auditory nerve)	_ _ . _ msec	_ _ . _ msec
Wave III (pons)	_ _ . _ msec	_ _ . _ msec
Wave V (midbrain)	_ _ . _ msec	_ _ . _ msec

Interpretation:

Normal	0 No, 1 Yes, 8 N/A, 9 Unknown	_
Abnormality consistent with	1=Left, 2=Right, 3=Both	_
	1=Central, 2=Peripheral, 3=Indeterminate	_

Assigned ID: _ _ _ _ _

SE date _ _ / _ _ / _ _ _ _

Secondary Cardiac Testing

pg _ _ of _ _

Use these responses for each test: **0=Normal, 1=Abnormal, 8=Not done, 9=Unknown**

Date Time	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _	_ _ / _ _ / _ _ _ _ : _ _
Echocardiogram: TTE TEE	_ _	_ _	_ _
SAECG	_	_	_
HRV	_	_	_
Holter	_	_	_
Stress Test with Thallium	_	_	_
MUGA	_	_	_
Cardiac Cath	_	_	_
Electrophysiology	_	_	_
Other test (_____)	_	_	_

APPENDIX B - TABLES AND FIGURES

Table 1. Acute Drug Therapy for Convulsive SE

Initial treatment • Lorazepam (0.1 mg/kg IV at 2 mg/min) or diazepam (up to 20 mg IV at 2 mg/min) • If seizures persist, phenytoin (20 mg/kg IV at 50 mg/min) **or** fosphenytoin (20 mg/kg PE IV at 150 mg/min) with cardiac monitoring
For persistent SE (or skip directly to refractory SE treatment) • Additional phenytoin or fosphenytoin up to a total dose of 30 mg/kg phenytoin or 30 mg PE/kg fosphenytoin **or** • Phenobarbital* (20 mg/kg IV at 50-75 mg/min)
For refractory SE • Pentobarbital* (3-12 mg/kg IV loading dose followed by infusion of 1-10 mg/kg per hour) **or** • Midazolam* (1.2 mg/kg loading dose followed by infusion of 0.1-2.0 mg/kg per hour) **or** • Propofol* (3-5 mg/kg loading dose followed by infusion of 1-15 mg/kg per hour) **or** • Valproic acid (15-25 mg/kg loading dose followed by infusion of 3-6 mg/kg per minute)

SE indicates status epilepticus; IV, intravenous; PE, phenytoin sodium equivalents.

*These treatments require ventilatory support, hemodynamic monitoring in an intensive care unit, and electroencephalogram monitoring.

From Waterhouse, E. J. and DeLorenzo, R. J. (2001). Status epilepticus in older patients: epidemiology and treatment options. *Drugs Aging* **18,** 133-42.

TABLE 2: CHARACTERISTICS OF THE STUDY POPULATION

		FREQUENCY	PERCENT
AGE			
	Young	1475	66.4
	Elderly	745	33.6
AGE GROUPS			
	(0,1)	188	8.5
	(1-20)	518	23.3
	(20-34)	185	8.3
	(34-49)	280	12.6
	(49-65)	466	21
	(65-75)	332	15
	(75+)	251	11.3
GENDER			
	Female	1035	46.6
	Male	1185	53.4
SE TYPE GROUPS			
	SPS	78	6.1
	CPS	55	4.3
	P with 2 Gen	529	41.2
	Absence	6	0.5
	Myoclonic	24	1.9
	Tonic	19	1.5
	TC	293	22.8
	Other	14	1.1
	SPS w LOC	124	9.6
	Bil In NC	5	0.4
	Sub/Par	10	0.8
	Sub/Gen	14	1.1
	Bil Con	3	0.2
	Myo/Gen	6	0.5
	Myo/Foc	1	0.1
	P/2/Non	4	0.3
	Part/Non	50	3.9
	Gen/Non	49	3.8
	Unknown	1	0.1
SE TYPE			
	Noncon	138	10.7
	Other	15	1.2
	Partial	261	20.3
	General	871	67.8
RACE			
	Black	1219	57.2
	White	763	35.8
	Other	150	7.0
TIME TO TREATMENT			
	<=60	543	47.3
	>60	604	52.7
MORTALITY			
	Dead	275	12.4
	Alive	1945	87.6
SE ETIOLOGIES			
	Hyp/Anox	133	10.4
	CNS Acu	279	22.0
	Non CNS	275	21.0
	Low AED	230	18.0
	Withdraw	76	5.9
	Remote	233	18.2
	Other	50	3.9

TABLE 3: CRUDE ASSOCIATIONS BETWEEN AGE AND OTHER PREDICTOR VARIABLES

		YOUNG	ELDERLY	OR	95% CI	
RACE	Black	796	423	1.37	1.04	1.82
	White	523	240	1		
	Other	109	41	0.34	0.135	0.858
GENDER	Female	632	403	1		
	Male	843	342	1.84	1.42	2.38
SE TYPE	Noncon	77	61	0.73	0.22	2.47
	Other	10	5	1.12	0.68	1.85
	Partial	148	113	0.67	0.42	1.05
	General	609	262	1		
ETIOL	Hyp/Anox	67	66	1.16	0.68	2.00
	CNS Acu	149	130	1.48	0.94	2.25
	Non CNS	223	56	0.44	0.28	0.69
	Low AED	162	68	1.00		
	Withdraw	45	31	1.95	1.07	3.55
	Remote	151	82	1.25	0.82	1.92
	Other	43	7	0.35	0.14	0.89
TIME RX	<=60	340	203	1		
	>60	408	196	0.77	0.59	1.02
MORTALITY	Alive	1356	589	1		
	Dead	119	156	3.54	2.53	4.95

TABLE 4: CRUDE ASSOCIATION BETWEEN SE MORTALITY AND PREDICTOR VARIABLES

		MORTALITY		OR	95% CI	
		Alive	Dead			
AGE	Young	1356	119	1		
	Elderly	589	156	3.18	2.33	3.9
GENDER	Female	918	117	1		
	Male	1027	158	1.2	0.94	1.56
RACE	Black	1081	138	1		
	White	635	128	1.6	1.2	2
	Other	141	9	0.5	0.25	1
ETIOLOGY	Hyp/Anox	54	79	18.3	10	33.5
	CNS Acu	200	79	4.9	2.8	8.7
	Non CNS	217	62	3.5	2	6.3
	Low AED	213	17	1		
	Withdraw	69	7	1.3	0.51	3.2
	Remote	206	27	1.6	0.87	3.1
	Other	47	3	0.8	0.23	2.8
SE TYPE	Noncon	85	53	2.3	1.5	3.8
	Other	11	4	1.4	0.43	4.6
	Partial	207	54	1		
	General	708	163	0.88	0.63	1.3
TREAT GRP	<=60	427	469	1		
	>60	116	135	1.1	0.8	1.4

TABLE 5: ADJUSTED OR AND 95% CONFIDENCE INTERVAL

		OR	95.0% CI	
RACE	Black	1		
	White	1.69	1.20	2.38
	Other	1.92	0.77	4.78
GENDER	Female	1		
	Male	0.71	0.51	1.00
ETIOLOGY	Hyp/Anox	22.53	11.13	45.62
	CNS Acu	4.67	2.46	8.85
	Non CNS	6.38	3.29	12.40
	Low AED	1.00		
	Withdraw	1.29	0.49	3.43
	Remote	2.06	1.02	4.18
	Other	2.50	0.64	9.81
SE TYPE	Noncon	1.75	1.05	2.91
	Other	1.62	0.37	6.98
	Partial	1.17	0.77	1.79
	General	1.00		
TIME TRT	<=60	0.78	0.56	1.10
	>60	1.00		
AGE GRP	(0, 1)	1.00		
	(1-20)	0.41	0.17	0.99
	(20-34)	2.67	1.10	6.48
	(34-49)	3.23	1.47	7.09
	(49-65)	3.27	1.57	6.81
	(65-75)	6.10	2.88	12.90
	(75+)	7.79	3.55	17.07

FIGURE 1: INCIDENCE AND RECURRENCE OF SE BY AGE

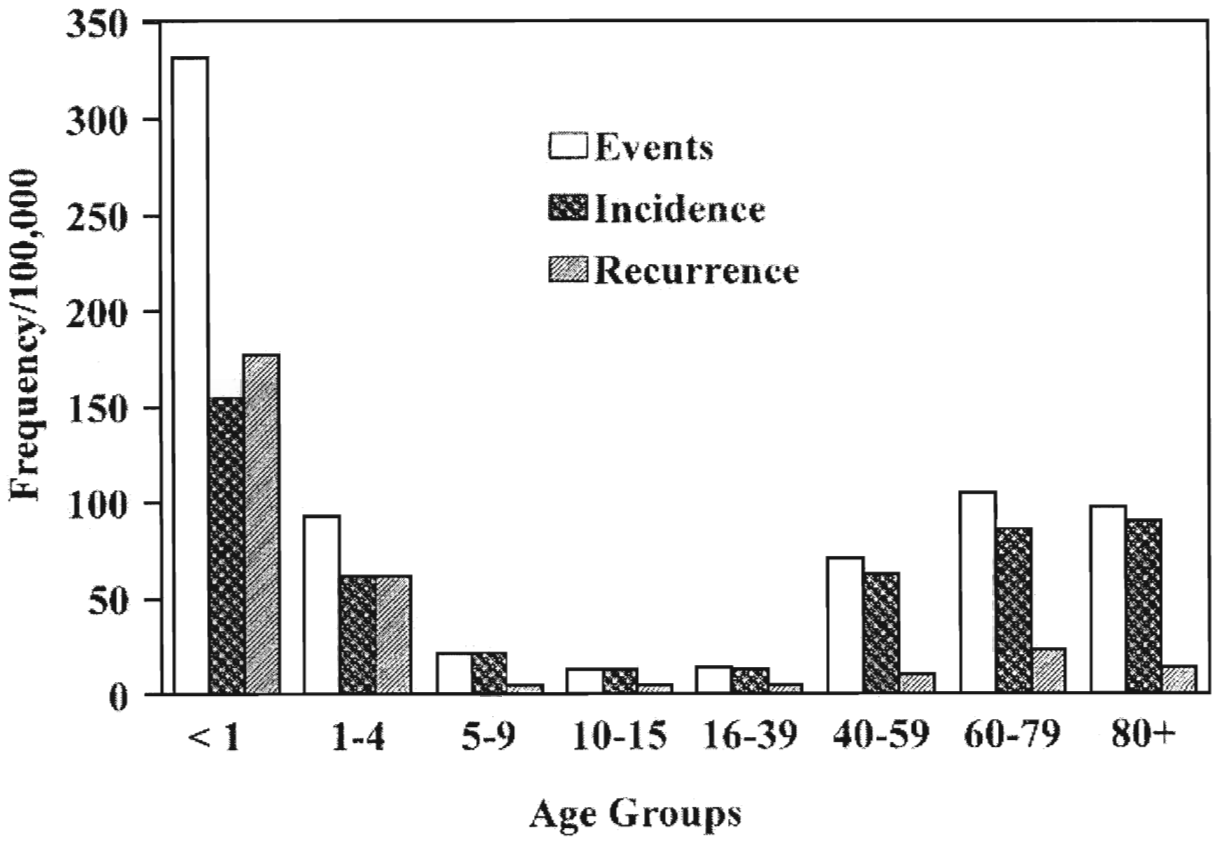
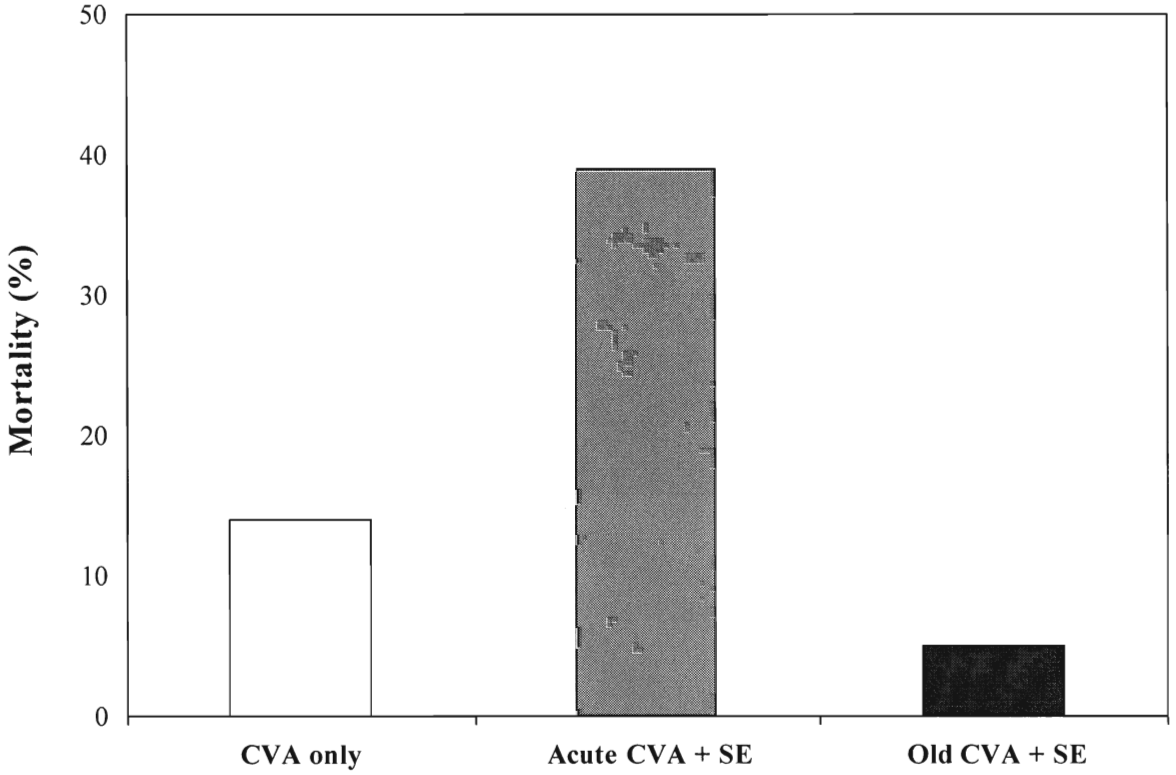


Figure 1. Age-specific distribution of the frequency of status epilepticus (SE) events, the incidence of SE, and the frequency of SE recurrence per year per 100,000 in Richmond, Virginia. The population in each age group was determined from National Census Bureau data on the demographics of Richmond, 1990. Status epilepticus events included all episodes of SE per 100,000 per year. The incidence of SE represents the number of patients that developed SE per 100,000 per year in Richmond and did not include recurrent episodes of SE. From DeLorenzo, R. J., Hauser, W. A., Towne, A. R., Boggs, J. G., Pellock, J. M., Penberthy, L., Garnett, L., Fortner, C. A., and Ko, D. (1996). A prospective, population-based epidemiologic study of status epilepticus in Richmond, Virginia. *Neurology* **46 Issue 4** 1029-1035.

FIGURE 2. MORTALITY OF STROKE AND SE



SE indicates status epilepticus; CVA, cerebrovascular accident.

Reprinted from Waterhouse, E. J., Vaughan, J. K., Barnes, T. Y., et al. (1998). Synergistic effect of status epilepticus and ischemic brain injury on mortality. *Epilepsy Research* **29** 175-183, (2006).

FIGURE 3

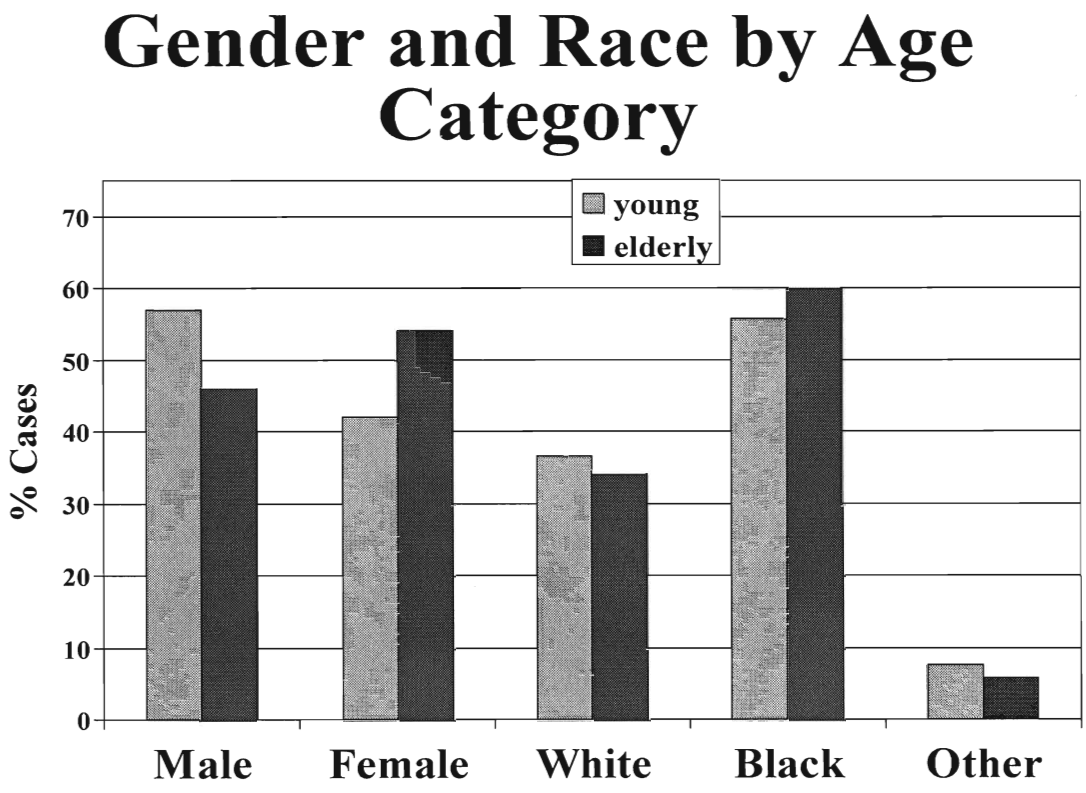

Gender and Race by Age Category
young
elderly
% Cases
70
60
50
40
30
20
10
0
Male
Female
White
Black
Other

FIGURE 4

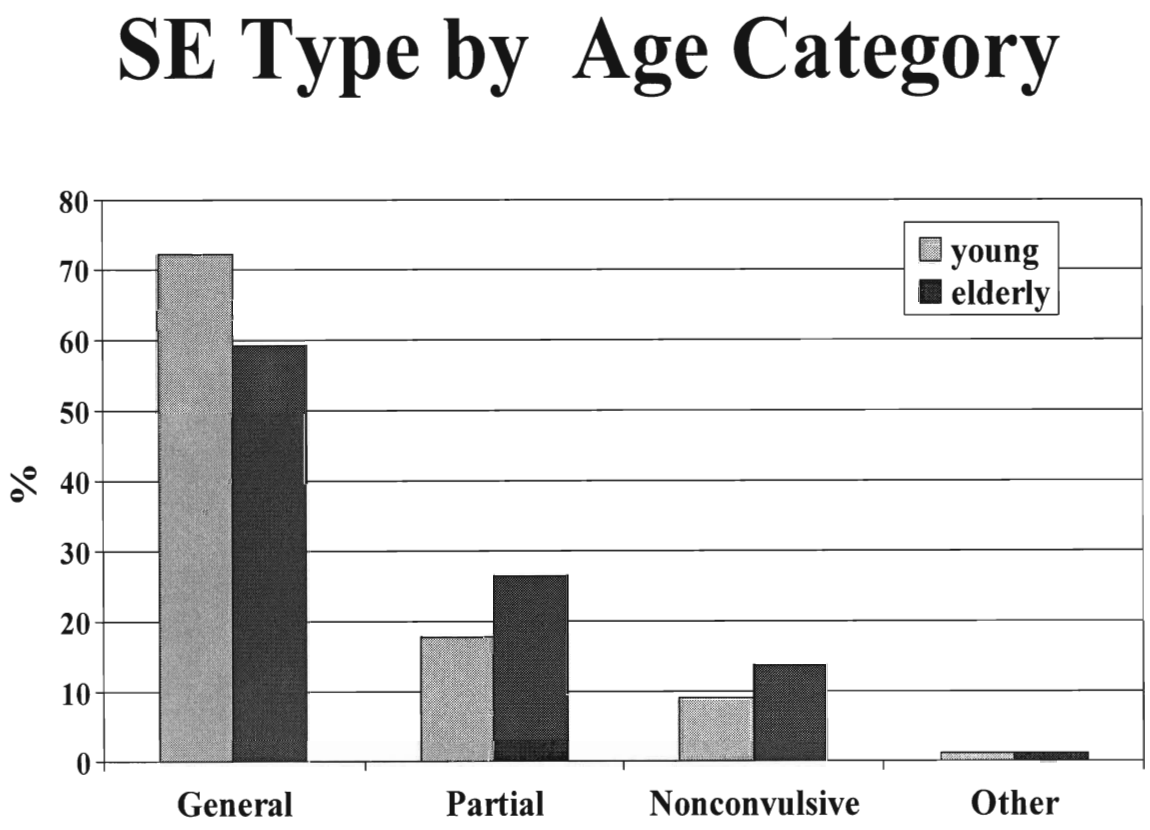

SE Type by Age Category
80
70
60
50
40
30
20
10
0
%
young
elderly
General
Partial
Nonconvulsive
Other

FIGURE 5

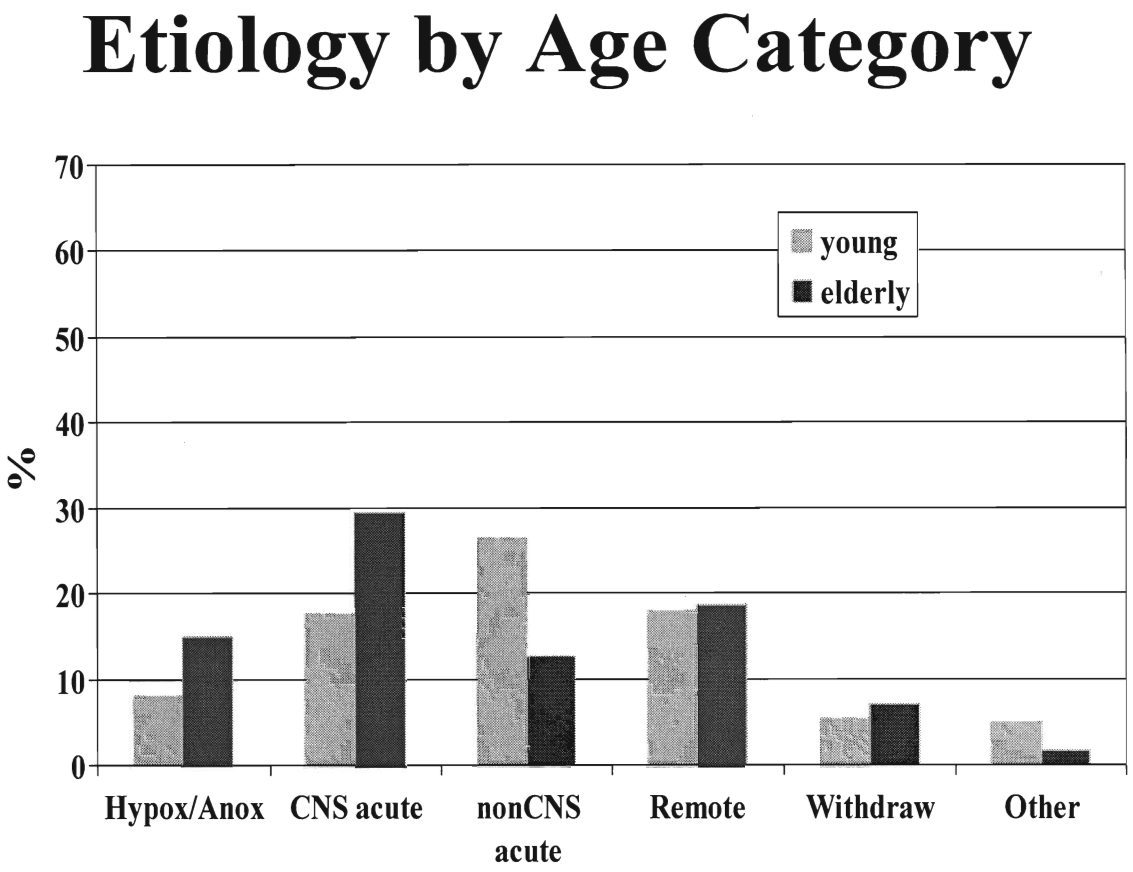
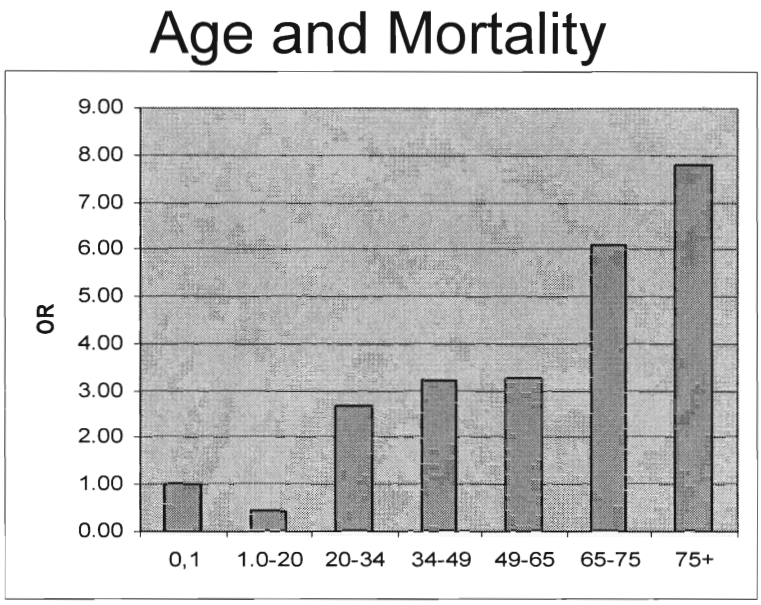

Etiology by Age Category
70
60
50
40
30
20
10
0
%
young
elderly
Hypox/Anox
CNS acute
nonCNS acute
Remote
Withdraw
Other

FIGURE 6


Age and Mortality
OR
9.00
8.00
7.00
6.00
5.00
4.00
3.00
2.00
1.00
0.00
0,1
1.0-20
20-34
34-49
49-65
65-75
75+