Finally, the claim is made that 51% of cases of cerebral palsy were statistically ''attributable'' to inequalities in RGSC. This calculation is based on the assumption that a cerebral palsy prevalence of 1.1 per 1000 in RGSC 1 is a valid estimate and doubts over this estimate already have been referred to. The calculation also assumes that the prevalence of cerebral palsy in all other RGSCs could be reduced to 1.1 per 1000. In addition to the validity of these assumptions being suspect, although the authors make it clear that the attribution is statistical, the reader frequently considers that the association demonstrated infers causation. This is not so; any observed epidemiological association cannot be assumed to be causal. Hill enumerated several criteria that justified an epidemiological association being considered causal, one of which is that the association be consistently observed in a variety of studies in different locations.[4] Such consistency is lacking in studies reporting the association of cerebral palsy with social inequalities.

Whether or not socioeconomic status has a role in cerebral palsy is contentious and the authors' attempt to shed light on the problem is admirable, particularly since they were endeavouring to use routine data sources to address the issue. Efforts to replicate the study in other districts based on similar data sources are highly recommended.

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Neurology

Status epilepticus with fever: how common is meningitis?

R Kneen, R Appleton

Commentary on the paper by Chin *et al* (see page 66)

The majority of febrile seizures are simple (generalised and brief, lasting less than 10 minutes), occurring in children aged 6 months to 5 years. The risk of acute bacterial meningitis (ABM) in association with simple febrile convulsions is reported to be about 1–2%.[1] Approximately one quarter of febrile convulsions are complex, defined as prolonged (>10 minutes), having a focal onset, recurring in the same illness, or followed by a neurological deficit. It is generally believed that the risk of ABM with a complicated febrile seizure is higher than with a simple febrile convulsion, but the exact figure is not known. At the most severe end of the spectrum are children with convulsive status epilepticus (CSE) and fever, usually defined on the basis of a seizure lasting more than 30 minutes or a series of seizures lasting at least 30 minutes from which they do not regain consciousness in between. There are many causes of CSE with fever, but it is important to consider and identify those for which delayed treatment may have deleterious consequences—specifically all central nervous system infections, including ABM, cerebral abscess, viral meningoencephalitis, tuberculous meningitis, and other less common infections.

Chin *et al* have studied children with CSE with fever as part of an ongoing study into status epilepticus, the North London convulsive STatus EPilepticus in childhood Surveillance Study (NLSTEPSS).[2] Until recently, little information has been available on how likely this group of children are to have ABM. The authors have identified that 15–18% of children presenting with CSE and fever have ABM, but this is may be an underestimate as only one third of patients in the study underwent cerebrospinal fluid (CSF) analysis. The authors have also shown that children with ABM may not show the typical clinical signs of meningeal irritation, a well recognised observation in young children. The authors conclude that in their population, a significant number of children with CSE and fever were not investigated and treated for ABM. Although encephalitis (herpes simplex encephalitis) was not included in the study, it would seem appropriate to adopt a similar approach to the investigation (and management) of possible encephalitis as well as ABM.

The initial management of any child with a prolonged seizure and specifically CSE, involves resuscitative measures with particular emphasis on ensuring a secure airway, breathing, and circulation, and measurement of blood glucose and electrolytes and acid-base status. Subsequent management should focus on attempting to terminate the seizure and identify and treat any underlying cause. There is a UK based protocol for the management of a presenting tonic-clonic seizure, including CSE,[3] but it is unclear as to how closely this protocol is being followed in terms of both the drugs (and their doses) and the timing of their administration. This important information may become available from future analysis of data arising from NLSTEPSS. This is important as adult evidence suggests that the longer the tonic-clonic seizure (and episode of CSE), the more difficult it may be to terminate[4]—and there is no reason to suspect that this would be any different in children. Consequently, anticonvulsant medication should be instituted in any child who has been seizing for more than five minutes and subsequent management should adhere to the published protocols.[3 5] As yet, there is no recommended or consensus protocol for the treatment of refractory CSE, the definition of which is unclear, but usually includes persistence of the status following initial treatment with a benzodiazepine, phenytoin/phenobarbitone, or both. Current treatment options for treating refractory CSE include continuous intravenous infusions of either midazolam or diazepam, or anaesthesia with thiopentone or propofol with, ideally, electroencephalographic (EEG) monitoring (a full discussion of these treatment options is not within the remit of this commentary). The child with refractory CSE should always be discussed with a paediatric neurologist and should be transferred to a paediatric intensive care unit for both management of the persistent status and, where

necessary, ongoing identification of the underlying cause. There is a low but definite mortality and morbidity in children with refractory CSE, and this reflects not only the underlying aetiology but also its management.

Investigation and treatment of an underlying cause of the febrile CSE, and specifically an underlying CNS infection, must always be considered. Chin *et al* found that less than a third of patients in their study had a lumbar puncture (LP) and only two thirds were treated with antibiotics at presentation.[2] There does appear to be an increasing reluctance of paediatricians to undertake an LP in children with suspected CNS infection, despite the fact that CSF analysis findings are often helpful and may influence patient management.[6] A prolonged seizure is thought to increase intracranial pressure,[7] making it a contraindication to LP. In addition, ABM itself is known to cause coning,[8] and LP may precipitate cerebral herniation in some severe cases of ABM.[9] For these reasons it would be unwise to undertake an LP immediately after cessation of the CSE. It is our practice to treat these children with appropriate parenteral antibiotics, as well as intravenous acyclovir if herpes encephalitis is considered a likely diagnosis, until it is considered safe to undertake an LP.

The difficulty is deciding precisely when an LP should be undertaken; this is likely to be different for each patient. Arguably, it should be performed as soon as it is clear there is no medical contraindication. In practice, this may vary from 30 to 60 minutes following cessation of the episode of CSE to one or two days after presentation. In some cases it will rapidly become apparent that the child has no signs of CNS infection (for example, those with a complex febrile convulsion who make a rapid recovery). However, if the child remains continuously or intermittently febrile or unwell, and recovery is less rapid or there is any doubt about ABM or encephalitis, CSF analysis is likely to provide useful information.[6] If the child requires admission to intensive care for persisting seizure activity, or the child shows any abnormal neurological features (cerebral irritability, neurological asymmetry, or abnormal behaviour), neuroimaging should be undertaken prior to LP because patients may be too sedated to accurately assess the level of coma. Although it is recognised that normal brain computed tomography (CT) cannot exclude raised intracranial pressure, it will exclude focal pathology, including a cerebral abscess or a subdural collection and, using contrast, may also show meningeal enhancement.[10]

The treatment of an underlying cause, and specifically presumed ABM or herpes encephalitis, must again be initiated early using appropriate antibiotics and acyclovir in appropriate doses. The initial choice of antibiotic may need to be revised, depending on the results of bacterial/viral antigen studies and also blood, other tissue, and CSF cultures. Without information from CSF analysis (cell count, protein and glucose levels (and simultaneous blood glucose); culture and sensitivities), treatment of a presumed ABM or herpes encephalitis is likely to be empiric or even blind—with consequent uncertainty on the duration of treatment with intravenous antibiotics or acyclovir, or both.

Children with proven ABM should, simultaneously with antibiotics, receive corticosteroids.[11] Despite the lack of evidence, it is our practice to also use corticosteroids (either dexamethasone or prednisolone) in the treatment of proven or probable herpes encephalitis. The duration of steroid treatment varies; our practice is to treat for a minimum of seven days in proven ABM or herpes encephalitis.

Febrile CSE is a relatively common paediatric emergency. Acute symptomatic (specifically due to an acute CNS infection) and prolonged febrile seizures are the most causes of febrile CSE. Its management must be timely and appropriate to reduce the risk of death and irreversible neurological sequelae. The study by Chin *et al* has highlighted differences in the management of febrile CSE due to ABM between individual units and individual children.[2] The risk of herpes simplex encephalitis in this group of patients and its management are unknown, and the empirical treatment with acyclovir in this clinical situation is controversial. It might be possible for the authors to investigate the risk of viral encephalitis in the ongoing NLSTEPS Study. In this commentary we have attempted to address some of the more important issues with additional information about our own practice. Clearly, there is an indication for trying to develop evidence based guidelines to optimise the investigation and treatment of refractory febrile CSE.

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