

CURRENT TOPIC

Incidence, aetiology, and outcome of non-traumatic coma: a population based study

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Abstract

Aim—To determine the incidence, presentation, aetiology, and outcome of non-traumatic coma in children aged between 1 month and 16 years.

Methods—In this prospective, population based, epidemiological study in the former Northern NHS region of the UK, cases were notified following any hospital admission or community death associated with non-traumatic coma. Coma was defined as a Glasgow Coma Score below 12 for more than six hours.

Results—The incidence of non-traumatic coma was 30.8 per 100 000 children under 16 per year (6.0 per 100 000 general population per year). The age specific incidence was notably higher in the first year of life (160 per 100 000 children per year). CNS specific presentations became commoner with increasing age. In infants, nearly two thirds of presentations were with non-specific, systemic signs. Infection was the commonest overall aetiology. Aetiology remained unknown in 14% despite extensive investigation and/or autopsy. Mortality was highly dependent on aetiology, with aetiology specific mortality rates varying from 3% to 84%. With follow up to approximately 12 months, overall series mortality was 46%.

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There is an increasing awareness that non-traumatic coma is an important source of morbidity and mortality in the paediatric age range.¹⁻³ These children make heavy demands on paediatric intensive care unit and neuro-rehabilitation resources. Additionally, coma is recognised to be a non-specific sign with a wide potential differential diagnosis. Design of appropriate and efficient protocols of investigation for coma require an understanding of the relative frequencies of the various potential aetiologies.

To our knowledge there are no population based data on the incidence and aetiology of non-traumatic coma in children. Improved data on incidence, severity, and outcome are a

prerequisite for informed provision of health-care resources for this group.

Methods

STUDY POPULATION

The study population comprised children between 1 month and 15 years 11 months of age who were either admitted to a hospital, or who died, in the former Northern NHS region of England between July 1994 and December 1995, with a significantly depressed level of consciousness of non-traumatic aetiology. In the 1991 National Census this region had a population of 3 126 732 and a population under 16 of 613 565. Significant depression of conscious level was defined as a Glasgow Coma Score⁴ of 12. For children below 5 years of age the James modification of the Glasgow Coma Score was used.⁵ For children admitted to hospital an additional entry criterion was a minimum duration of the period of reduced consciousness of six hours. As it was not often possible to determine the duration of coma in the records of children dying out of hospital, this minimum duration criterion was not applied in this group.

Exclusion criteria comprised children with coma of traumatic cause, as part of an anticipated terminal illness, or children dying of sudden infant death syndrome (SIDS).

It was not possible to identify children normally resident in the study region who fell ill while in other regions of the UK. Children normally resident in other regions who fell ill while temporarily within the study region were included to partially correct for this systematic error.

RECRUITMENT

Children were identified through two complementary mechanisms. Children being admitted to any hospital in the region and coming to the attention of paediatric staff were notified to the researchers through return of a prepaid postal questionnaire mailed monthly to trainee and senior paediatric medical and nursing staff nationwide.

Office for Population Censuses and Surveys (OPCS; now the ONS) records of paediatric deaths (in any community or hospital setting) occurring in the region during the study period were also screened, and medical records and postmortem data obtained where available. If

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Table 1 Summary of impairment outcomes

Category	Neurological status
I (intact)	Normal or no change from premorbid functioning. Seizures, if recorded, 100% controlled
II (mild)	Minimal alteration of tone, power, or reflexes; isolated cranial nerve palsies; mild (MRC 4) weakness or ataxia.
III (moderate)	Seizures, if present, >75% controlled Moderate (MRC 3) weakness or ataxia; multiple cranial nerve involvement. Seizures, if present, >50% controlled
IV (severe)	Severe weakness (<MRC 3) or ataxia; tetraparesis. Uncontrolled seizures
V (profound)	Persistent vegetative state
VI	Death

Muscle power is graded using the MRC (Medical Research Council) scale: 0, no visible signs of contraction; 1, visible signs of contraction; 2, limb movement but insufficient power to overcome gravity; 3, sufficient power to overcome gravity but not additional resistance; 4, sufficient power to overcome gravity and additional resistance but not full power; 5, full power.

death was caused by an illness associated with coma the child was enrolled, and data on presenting symptoms and aetiology abstracted.

As an independent check on hospital based ascertainment, OPCS records of children dying in hospital where coma was part of the terminal illness were matched to notified hospital admissions (see "Ascertainment" in Discussion).

AETIOLOGY

The primary aetiology of the encephalopathic episode was determined after inspection of medical and/or postmortem data by one researcher (CPW).

OUTCOME DATA

Early and late neurological, special sensory, cognitive, and behavioural assessments were performed. Nominal follow up time points were six weeks and 12 months after admission. All children were given an assessment comprising a formal clinical neurological examination and a screening assessment for visual and auditory deficit. This assessment was performed by one investigator (CPW). In addition a cognitive and behavioural assessment was performed by a research associate using age appropriate psychometric instruments. If any special sensory impairments were identified, children were referred for formal visual or auditory assessment. If impairments were identified in any domain (neurological, cognitive, or special sensory), an assessment of the child's adaptive behaviour was completed using the Vineland Adaptive Behaviour Scales.⁶ Neurological outcome only will be reported in this paper. More detailed outcome data, including cognitive, special sensory, and adaptive behaviour outcomes, will be published separately.

In assessing effects of age on aetiology or outcome, children were divided into up to four age bands: infants (under 12 months); children 1–5 years; children 6–12 years; and children 13–16 years of age.

ASSESSMENT INSTRUMENTS

Clinical neurological assessment included cranial and peripheral motor and sensory neurological examination, including cerebellar function. Visual and auditory screening assessments were performed by CPW. Visual function was assessed by preferential looking, Kay Picture, Sheridan–Gardiner, or Sonksen–

Table 2 Episodes excluded from study

Reason	Number
Duration of consciousness less than 6 hours	37
Expected terminal phase of malignant disease	19
Two neonates, one over 16 years	3
Traumatic, non-accidental injury	2
Notified out of study period	1
Total	62

Silver assessments in children under 2, 3, 4, and 5 years respectively. Above 5 years Snellen chart visual acuity was assessed. Hearing assessment was by distraction test with visual reinforcement in children (using a Kamplex PA2 Audiometer) under 2, play audiometry under 4, and pure tone audiometry using a portable audiometer (Rexton) in older children.

Full cognitive and behavioural assessments were completed and will be reported separately.

SUMMARY IMPAIRMENT SCORE

Neurological outcomes were allocated to categories I (intact recovery) to V (profound impairment) and VI (dead) according to the scheme in table 1.

PREMORBID DISABILITY

Details of premorbid neurological and cognitive status were not systematically available. If a child with known prior neurological disability was considered by care givers to have returned to prior levels of disability, and if detailed neurological and special sensory examination revealed no new impairments not already identified, then a child was recorded as a category I (intact) outcome.

ETHICS

Ethical approval for this study was obtained from the local ethics committees in each of the five participating districts in the study region. Written consent for the follow up phase of the study was obtained by participating physicians from parents or guardians.

STATISTICAL ANALYSIS

Analyses were by binomial or contingency testing (Fisher's exact or χ^2 test). Statistical significance was assumed at $p < 0.05$.

Results

INCIDENCE

A total of 345 episodes of possible non-traumatic coma were identified to the research team, of which 283 fulfilled inclusion criteria. This represented 278 individuals (155 male, 123 female). Sixty two episodes were notified but not studied as they failed to meet eligibility criteria for reasons shown in table 2.

Three children were multiply recruited, experiencing more than one episode of non-traumatic coma during the study period. One child had four episodes of status epilepticus, and another, two episodes. A third child suffered two periods of coma caused by complications of congenital heart disease. Thus the 283 episodes represent 278 children. Incidence and aetiological data are expressed

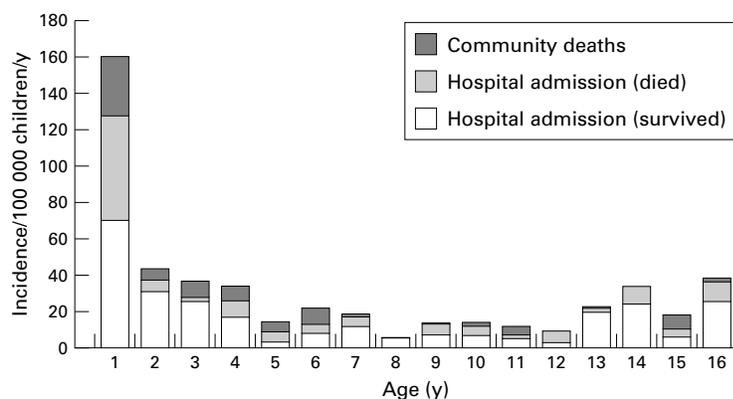


Figure 1 Incidence of non-traumatic coma by age.

relative to a denominator of 283. Outcome (including mortality) data are expressed relative to a denominator of 278.

Of the 283 episodes studied further, 224 were notified following admission to hospital. Fifty nine children who died outside hospital were identified through the survey of OPCS recorded deaths.

The regionwide incidence of non-traumatic coma was 30.8 per 100 000 children under 16 per year (approximately 6.0 per 100 000 general population per year) using 1991 census data as the denominator. There was a notable variation in age dependent incidence, with a greatly increased incidence in the first year of life of 160 per 100 000 children per year (see fig 1).

There were a total of 127 deaths: 59 prehospital deaths identified through OPCS death registrations and 68 in hospital deaths. The 68 hospital deaths include 32 "missed notifications". These were 32 children identified through OPCS death registrations as having died in hospital after an encephalopathic episode that met study criteria, but who had not been directly notified by hospital staff. One third of these were children who had died of complications of cardiac surgery for congenital heart disease at the regional cardiothoracic unit. The possible effects of this incomplete ascertainment on incidence figures is discussed below. No clear pattern of seasonal variation in incidence was evident.

PRESENTATIONS

The data relating to presentation were incomplete for 33 notifications, and missing for a further 34 of the children who died prior to admission. Data for the remaining 216 presentations were analysed. A total of 590 occurrences of 47 symptoms were recorded, with a further 17 occurrences of unclassifiable symptoms recorded as "others". There was a range of 1-8 (median 3) symptoms per presentation. Presenting symptoms were assigned to one of three symptom groupings: CNS specific, organ specific (non-CNS), and systemic. Table 3 shows the commonest presentations under each heading.

Systemic presentations (especially nausea and vomiting, feeding difficulties, lethargy, and fever) were particularly evident in the infant age group, with CNS specific presentations

Table 3 Presenting symptoms

	Number	%
<i>CNS related</i>		
Altered level of consciousness	78	13.2
Convulsion	55	9.4
Headache	28	4.8
Irritability	22	3.8
Photophobia	9	1.5
Behavioural change	7	1.2
Others	23	3.8
<i>Specific to other organ system</i>		
Rash	25	4.3
Upper respiratory tract infection	16	2.7
Skin haemorrhage	7	1.2
Sore throat	7	1.2
Others	42	7.2
<i>Systemic</i>		
Vomiting/nausea	60	10.3
Fever	55	9.3
Lethargy	37	6.3
Poor feeding	36	6.2
Shortness of breath	30	5.1
Pallor	12	2.1
Cyanosis	11	1.9
Respiratory arrest	9	1.5
Poor weight gain	8	1.4
Weakness of limbs	7	1.2
Others	4	0.7

The most frequent presentations in each group are shown, as number and percentage of all symptom occurrences (n = 590). Symptoms noted at a frequency of <1% of all symptom occurrences are aggregated under each heading as "others".

significantly more frequent in children over 5 (p < 0.0001; χ^2 test).

AETIOLOGY

Infection was the commonest cause of non-traumatic coma, accounting for 38% of cases in the overall series (fig 2). Intoxication, epilepsy, and complications of congenital abnormalities were also significant aetiologies, comprising 8-10% of cases each. Accidents and metabolic causes were responsible for 6% each. The remaining causes (non-traumatic intracranial haemorrhage, asthma, and complications of malignant disease) comprised less than 2% each and are shown as "others" in fig 2.

The distribution and rank order of aetiologies among those dying prior to admission (n = 59) differed from the overall population. Accidents were disproportionately over represented (25% of prehospital deaths v 2% in the admitted group; p < 0.0001; Fisher's exact test), reflecting the high mortality associated with this aetiology (see below). A trend for infection to be even more predominant as the main cause failed to reach statistical significance (48% prehospital deaths v 35% of those admitted; p < 0.1; Fisher's exact test).

The distribution of aetiological categories within the various age bands (shown in table 4) was highly significant (p < 0.0001; χ^2 test). Complications of congenital malformation were particularly frequent in infants, as was intoxication in teenagers.

Infection

Infection was identified as the primary aetiology in 107 notifications (29 preadmission deaths and 78 admissions). The age and sex distribution for the infection subgroup did not differ significantly from the overall series. Systemic, respiratory, and CNS infection predominated, accounting together for 90% of this group. Children dying prior to admission

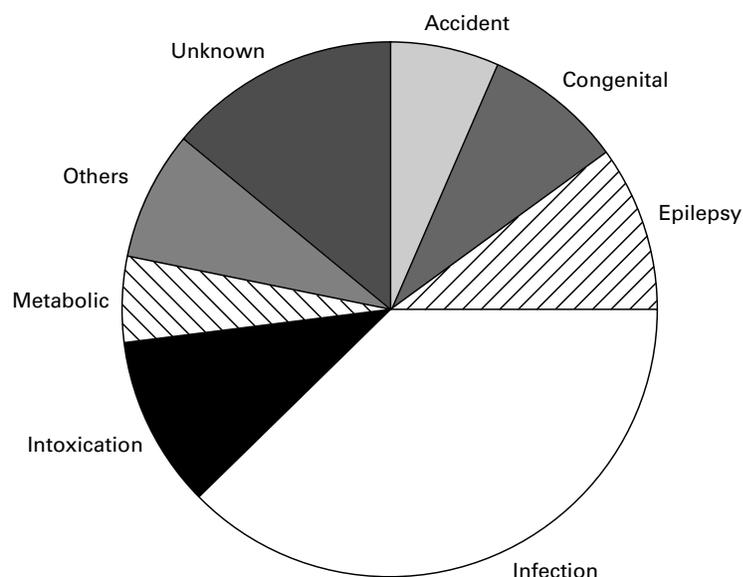


Figure 2 Summary distribution of the primary aetiologies of non-traumatic coma among the 284 children studied (including postmortem information where available in community and hospital deaths).

were only assigned to this group if postmortem examination showed unequivocal evidence of infection ($n = 25$) or a pathogen was identified after death ($n = 4$). Children identified after hospital admission or as a "missed notification" picked up through OPCS notification were only assigned to this group if microbiological studies identified a pathogen ($n = 78$). A pathogen was therefore identified in 82 cases (78 admissions and four postmortem isolations). *Neisseria meningitidis* was the commonest identified pathogen, responsible for 47% of all these cases where a pathogen was identified.

Intoxication

Two distinct subpopulations made up this group. A bimodal age distribution was observed. Nine of 29 were under 5 years of age: all ingestions in this group were accidental. All of the remainder (20 of 29) were over 10 (median age 14, range 10–16). Of these 20 older children, 18 were deliberate self poisonings. The remaining two children experienced the syndrome of inappropriate ADH secretion caused by prescribed medications at prescribed doses (1-deamino-8-D-arginine vasopressin and carbamazepine).

Epilepsy

There were 28 instances of coma caused by prolonged seizure activity or its treatment, representing 24 children (one child having four episodes and one child two episodes during the study period). One child died at home. Prolonged febrile convulsions were responsible for a quarter of cases. The entry criterion of a

minimum six hour period of reduced consciousness determined that these are a selected severe subgroup of all children with prolonged seizures.

Congenital

Coma caused by complications of congenital malformations (including complications of attempted surgical correction) accounted for 23 episodes in 22 children. Seventeen of 23 episodes were caused by complications of congenital heart disease, and four by obstruction of cerebrospinal fluid flow as a result of congenital CNS malformations including neural tube defects.

This group showed a statistically significant difference in age distribution from the overall population, with 70% younger than 1 year of age ($p < 0.0003$; Fisher's exact test).

Accidents

Coma of accidental cause was identified in 19 cases, including smoke inhalation in house fires ($n = 7$), accidental strangulation ($n = 4$), burns ($n = 3$), and drowning ($n = 3$). Mortality was higher in this group (83%) than in the overall series ($p < 0.0005$; Fisher's exact test). The three survivors were all in the burns group.

Metabolic

Diabetic ketoacidosis ($n = 8$) and medium chain acyl coA dehydrogenase (MCAD) deficiency ($n = 3$) were the commonest causes. The episodes of diabetic ketoacidosis were the first presentations of diabetes in seven of the eight children. The MCAD diagnosis was made at postmortem examination in one child. Specific inborn errors of metabolism were identified in three further children (see Discussion).

Unknown

The aetiologies for 14% ($n = 41$) of the overall series remained unclear despite extensive inpatient investigation (and in the five prehospital deaths in this group, postmortem examination). This is probably an aetiologically heterogeneous group. Age and sex distributions were not statistically significantly different from the overall distributions. In approximately half of these cases an infectious aetiology was suspected because of a clinical picture of septicaemia, encephalitis, meningitis, or pneumonia; however, pathogens were not identified. A further quarter had suspected metabolic causes. The remaining suspected (but unproven) causes included epilepsy ($n = 2$), intoxication ($n = 2$), and ventriculoperitoneal shunt dysfunction ($n = 2$).

Table 4 Age specific aetiology

Age band	Accident	Congenital	Epilepsy	Infection	Intoxication	Metabolic	Others	Unknown	Total
Infant	3.2%	17.2%	4.3%	50.5%	0.0%	4.3%	6.5%	14.0%	100.0%
1–5 years	11.2%	3.4%	13.5%	33.7%	10.1%	6.7%	4.5%	16.9%	100.0%
6–12 years	5.6%	7.4%	16.7%	31.5%	7.4%	5.6%	13.0%	13.0%	100.0%
13–16 years	6.5%	0.0%	4.3%	28.3%	34.8%	2.2%	10.9%	13.0%	100.0%
Total	6.7%	8.2%	9.6%	37.9%	10.3%	5.0%	7.8%	14.5%	100.0%

Table 5 Outcome by aetiology

	Intact	Mild	Moderate	Severe	Profound	Dead	Unknown	Total
Accident	10.5%	0.0%	5.3%	0.0%	0.0%	84.2%	0.0%	100%
Congenital	9.1%	9.1%	0.0%	9.1%	0.0%	72.7%	0.0%	100%
Infection	26.4%	5.5%	0.9%	2.7%	0.9%	60.0%	3.6%	100%
Others	28.6%	9.5%	4.8%	4.8%	4.8%	47.6%	0.0%	100%
Unknown	47.5%	5.0%	5.0%	7.5%	0.0%	30.0%	5.0%	100%
Metabolic	60.0%	0.0%	0.0%	6.7%	0.0%	26.7%	6.7%	100%
Epilepsy	71.4%	0.0%	3.6%	7.1%	0.0%	17.9%	0.0%	100%
Intoxication	48.3%	13.8%	10.3%	0.0%	0.0%	3.4%	24.1%	100%

Aetiology specific late mortality and morbidity (median follow up 13 months).

MORTALITY AND NEUROLOGICAL MORBIDITY

Among the 278 individual children experiencing non-traumatic coma there were 59 pre-hospital deaths. An additional 66 children died during their hospital admission, with 153 children surviving to discharge. Late outcome assessments were scheduled at 12 months. There were two late deaths following discharge in the follow up group, leaving 151 late survivors and an overall series mortality of 127/278 (45.6%).

Extensive efforts were made to contact survivors, delaying the median follow up interval from a nominal 12 months to 13 months (range 8–37 months). Neurological, special sensory, and cognitive follow up was achieved for 141 of the 151 late survivors: six families declined to participate in late follow up and four were untraceable. Thus late outcome was known for 268 of the original cohort of 278 children (96%; 93% of survivors).

Seven children with known severe neurological disability prior to their episode of coma were deemed on the basis of clinical and caregivers' assessments to have recovered with no new impairments and were coded as category I (intact) outcomes. In five of these seven cases the coma was the result of prolonged seizures (and/or their treatment) in children with known remote symptomatic epilepsy.

Of the 141 children with late follow up, 94 (66%) had category I outcomes with no detectable impairment in any of the four domains assessed. Cognitive/behavioural morbidity was the most frequent, being the sole domain of impairment in 27, and the major (or equally predominant) domain in a further 13 children. More detailed cognitive, special sensory, and neurological data will be reported separately.

Table 5 shows the distribution of late neurological outcome for all children (deaths and survivors) by aetiology. Neurological morbidity is graded according to the criteria of table 1. There was a highly significant association between mortality and aetiology ($p < 0.0001$; χ^2 test). Mortality was also significantly higher in infants than in children over 1 year of age ($p < 0.05$; Fisher's exact test); however, logistic regression showed this effect of age on mortality not to be independent of aetiology (data not shown).

Among survivors for whom late outcome data were available, there remained a significantly higher frequency of poor late neurological outcome in infants than in older children ($p < 0.002$; χ^2 test; table 5). However, the association of late neurological outcome with

aetiological group failed to reach statistical significance among survivors.

Discussion

Childhood coma is a non-specific consequence of a variety of serious pathological processes. This population based survey provides information on prevalence and outcome in paediatric non-traumatic coma: a necessary precursor to informed provision of health services.

Most epidemiological studies of coma in children have concentrated on traumatic coma.^{7–16} Previous studies of non-traumatic coma have been hospital based.^{17–20} Only three studies have concentrated on non-traumatic coma in children,^{1, 20, 21} and the National Childhood Encephalopathy Study²¹ was limited to the study of children under 36 months with specific reference to the possible consequences of pertussis immunisation.

The importance of infection as an aetiology of non-traumatic coma identified in this study is supported by others.^{17–20, 22} The study of Seshia and colleagues²² categorises on the basis of presumed pathophysiological mechanism rather than specific aetiology, leading to an emphasis on presumed hypoxic ischaemic pathophysiology not seen in this study. The importance of infective aetiologies in children is in sharp contrast to adult hospital based series where degenerative and cerebrovascular pathologies predominate.²³

Clinicians managing children in non-traumatic coma are often concerned that the illness may be the presentation of a previously unrecognised inborn error of metabolism. Eight of the 14 children in the metabolic group were in diabetic ketoacidosis which should present few diagnostic challenges. Three children's inborn errors of metabolism had been recognised prior to the episode of non-traumatic coma (glutaric aciduria type 1 in one; mitochondrial encephalopathy, lactic acidosis, and stroke like episodes in one; and ornithine transcarbamylase deficiency in a female heterozygote). In only three children was the inborn error diagnosed as a result of the presenting episode of coma: all had MCAD deficiency, in one case diagnosed at post-mortem examination.

Additionally nine children in the "unknown" group had suspected, but unproven metabolic causes for coma: five presented with hypoglycaemia, three with coma suggestively associated with intercurrent stress, and one with probable Huttenlocher syndrome.²⁴ With a minimum of three years further informal follow up, alternative, non-metabolic explanations for

the episode of non-traumatic coma have been identified in two of these children. In three no further episodes have occurred, the children have remained entirely well, and metabolic conditions appear highly improbable. In three children a metabolic condition appears possible or probable (two have subsequently died, including the child with presumed Huttenlocher syndrome). A definite endocrinological/metabolic diagnosis has subsequently been made in one (hypopituitarism). Thus coma was the presenting feature of a probable or definite previously unsuspected inborn error of metabolism in a maximum of seven of 283 episodes (2.5%).

ASCERTAINMENT

The centralised location of tertiary paediatric intensive care services in the region facilitated recruitment, although only 28% of the children actually passed through a paediatric intensive care unit.

Thirty two children notified to the study team via OPCS had died in hospital, but had not been independently notified to the survey by clinicians. If these children (and the corresponding hypothesised missed survivors) were representative of the overall series in terms of mortality, then the global series mortality rate of 45% would imply 26 missed survivors. Alternatively the aetiology specific mortality rates identified for the rest of the series (table 5) would imply up to 32 missed survivors. This in turn would give a relatively secure upper limit on the prevalence estimate of 34.3 per 100 000 children per year.

However, there are two reasons why these are likely to be overestimates of the numbers of missed survivors not notified to the survey. Firstly, 11 of these 32 children reported via OPCS died in a single unit, the regional cardiothoracic unit. A separate audit of the admissions to this unit confirmed ascertainment of all survivors. Secondly, it seems likely that the remaining 21 children were a selected group of higher mortality than the general series: five died in the emergency department and all died within seven days of admission. Thus these children are a selected, sick population likely to have a higher mortality rate than the overall series. The number of survivors of coma missed by this survey is thus likely to be considerably lower than these estimates.

One of the five districts reported a somewhat lower incidence of non-traumatic coma (13.1 per 100 000 per year) than the remaining four (range 27.8–35.8 per 100 000 per year). Again if this represented systematic under reporting and the true incidence in this district was the average of the other four, approximately 27 additional cases would have been expected within the study period.

Logistical considerations prevented seeking cases of children normally resident in the study region falling ill while temporarily outside it. Children normally resident outside the region who were admitted to study hospitals were included however, in the expectation that this would partially compensate.

COMPARISON WITH INCIDENCE OF TRAUMATIC COMA

Direct comparison with published figures of the incidence of traumatic coma in childhood is complicated by methodological issues, including definition of a severity threshold. The San Diego county studies of Kraus and colleagues²⁵ and Klauber and colleagues⁸ included minor injuries. An incidence of “severe” traumatic brain injury in children up to 14 years of age (defined by a minimum GCS = 8) of 27 per 100 000 children per year has been calculated by Kraus and colleagues.⁹ The incidence of traumatic and non-traumatic coma are therefore likely to be comparable, although it should be emphasised that the age profiles of these two groups are very different.

The present study confirms that children below the age of 1 year are at highest risk of developing non-traumatic coma, with risk falling to a minimum between 5 and 8 years before slowly rising again (fig 1). In comparison mobility is a major risk factor for traumatic coma, and its age specific incidence rises through childhood with highest rates in the 15–20 year age group.^{7 8 25–29}

Aetiologies showed predictable age specific profiles, with complications of congenital malformations and inborn errors of metabolism prominent in infancy; infection becoming the predominant aetiology from late infancy onwards (mirroring the fall of maternally acquired passive immunity); and accidental and non-accidental ingestions showing the profiles discussed in the results. Again in contrast to traumatic coma studies, where male gender is a significant risk factor, the male predominance in this study (160/284) did not reach statistical significance.

OUTCOME DATA

An overall series mortality of 127 children equates to a mortality of 13.7 per 100 000 children per year from all causes of acute coma. Although morbidity following non-traumatic coma was high in this series, it is better than reported adult hospital data where mortality rates of 60% and neurologically intact survival rates of 10% are seen.²³ Mortality in this series was higher than reported in other paediatric hospital based series^{1 22} as a result of the exclusion of community, preadmission deaths in these latter reports.

Paediatric non-traumatic coma is an important health problem making significant demands of intensive and high dependency care resources. It can result from a wide variety of primary aetiologies, posing a diagnostic challenge to medical staff. A better understanding of the causes and outcomes of this heterogeneous group of children will aid the design of protocols for their investigation and management.

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STAMPS IN PAEDIATRICS

Oral rehydration therapy

Gastroenteritis is one of the major causes of childhood mortality and morbidity. Oral rehydration therapy is the cornerstone of treatment and has been widely promoted throughout the world. A number of stamps have been produced to increase public awareness and

improve education. The three stamps shown here illustrate various features and aspects of this form of treatment. All relate to the UNICEF Child Survival Campaign.

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