PAEDIATRIC HEAD INJURY

DECISIONS

IN

THE ACCIDENT AND EMERGENCY DEPARTMENT

M.D. THESIS

1997

MARIE T. BROOKES

Volume II

VOLUME II

METHODOLOGY

and

DATA DEFINITIONS

A. METHODOLOGY and DATA DEFINITIONS

For the purpose of the Study, children were defined as 14 years or less, birth injuries were excluded. The hospital Accident & Emergency Departments were selected on the basis of

1. The presence of a Consultant in Accident & Emergency Medicine, or one with a day to day commitment.
2. Excellence of past data collection and research.
3. Direct access to a computer data base in some cases.
4. A known Paediatric component in the workload.
5. The co-operation of the Neurosurgeons in the associated Neurosurgical Unit.

It was anticipated that 1000 consecutive new patient attenders with head injury, would provide information from each of 10 selected departments, providing data on 10,000 children for analysis. The information was to be collected locally, a printed proforma being provided. The clinical details were to be recorded immediately, by the examining doctor, following assessment of the child, and before discharge home or admission to a ward or Specialist Unit. Information on patients admitted to hospital or transferred to a specialist facility, was to be added later, and within one month of the primary attendance. Unfortunately four of the hospitals initially selected including two Teaching Centres found it impossible to participate, because of the demands made by the project. This reduced the group to six Accident & Emergency Departments.

It was agreed that each hospital would provide clinical information on 1000 consecutive children. In Monklands Hospital the Study would continue for one year. In each of the participating hospitals, an Accident & Emergency doctor of Senior Registrar or Registrar status was nominated to collate the forms on a regular basis, and to be responsible for the recovery of reports from the Xray Department, in patient records and occasionally from return visits. The completed data forms were despatched to and collated by the Author on a regular basis.

Other questions asked of each coordinator included.

1. The system and speed of Radiologist reporting of skull xrays.

2. The availability of a CT Brain Scanner in the hospital and the criteria for requests for this examination and its use in preference to skull xrays.
3. The availability of arterial blood gas analysis.
4. Modes of transfer to the Neurosurgical Unit, and the distance from the Accident & Emergency Department.

PARTICIPATING HOSPITALS		SUPERVISORS
01	Monklands District General Hospital	Dr M T Brookes
02	Glasgow Royal Infirmary	Mr I Swann
03	Royal Aberdeen Children's Hospital	Mr G Youngson
04	Alder Hey Children's Hospital, Liverpool	Miss J Robson
		Dr D Gorman
05	Poole General Hospital	Dr T J Underhill
		Dr F L P Hayes
06	Hull Royal Infirmary	Dr J Gosnold
		Dr R J McGlone

The Author visited all hospitals personally at the commencement of the Study, with the exception of Poole District General Hospital. Subsequently visits were required to make good deficiencies of information from in-patient and neurosurgical records which proved elusive! Information on the participating hospitals included the annual workload of new patient attenders, the proportion of children attending, the total number of head injuries, and the percentage in the paediatric age groups. The catchment population and the characteristics - urban, rural and mixed was noted. Special features of the medical service was noted as appropriate. The staffing structure of the Department was identified.

Consultant.

Senior Registrar.

Associate Specialist/Clinical Assistant.

Senior House officer.

The status was recorded of the medical staff most frequently involved in decisions on management. This not unexpectedly proved to be the Senior House Officers. The individual

Units were asked to provide information on any Guidelines issued for Head Injury Management, including their Admissions Policy. In addition, the wards of admission were to be identified.

WARDS OF ADMISSION

Short Stay Ward

Primary Surgical or Orthopaedic Ward

Paediatric Unit/Medical/Surgical.

Neurosurgical Unit either within the hospital, or a Regional Centre.

Intensive Care Unit - General/Paediatric.

The Author, having been a contributor to the National Seatbelt Survey, associated with the 1983 Seatbelt Legislation applied the lessons learned on the value of regular visits, personal contact with the participants and encouragement in completing the forms accurately. There was invaluable co-operation also from Clinicians in various Departments responsible for the care of children. These included Paediatricians, Surgeons, Orthopaedic Surgeons and Neurosurgeons. Their permission was sought in the early stages of the preparation of the Study and records were made available and advice and support offered. Permission to recover information for the minority of children transferred to Neurosurgical Units was readily given. Archive and xray searches to recover missing information was undertaken by the Author, with the assistance of Dr John Scott and Dr Iain Brown, who transferred all the clinical data to the IBM forms for transmission to the Mainframe Computer at Glasgow University.

MONKLANDS DISTRICT GENERAL HOSPITAL

Monklands Hospital is a District Hospital serving a population of 187,000 in a predominantly urban setting, including the towns of Airdrie, Coatbridge and the new town of Cumbernauld. The hospital was built on a virgin site, patients previously having been served by Glasgow Royal Infirmary, the Royal Hospital for Sick Children, Yorkhill, Glasgow, and a Lanarkshire District General Hospital, Law Hospital at Carluke.

1987

New patients 49,585

Head Injuries 4,594

Paediatric Head Injuries 2,185

Patients attending following trauma 26,000

Medical Staffing

Two full time Consultants, 10 Clinical Assistant sessions weekly, 8 Senior House Officers. The majority of head injured patients are treated by Senior House Officers.

Xrays

Skull xrays are reported by a Radiologist within 24/48 hours. Advice is available from senior staff, or a Radiologist on request during normal working hours.

Computerised Scanning

Not available on Site

Policy

The Head Injury Policy is that recommended by the Institute of Neurological Sciences, Glasgow, and based on the Guidelines issued by a Group of Neurosurgeons in 1984.

Admissions

Although Monklands has a Paediatric Medical Unit, head injuries are normally admitted to the Short Stay Ward. However, due to a major rebuilding programme in the Accident & Emergency Department between 1986 and 1988, children requiring admission for observation and treatment other than urgent neurosurgery, were transferred to the Royal Hospital for Sick Children, Yorkhill, Glasgow.

Loss of Consciousness

History of loss of consciousness in the absence of physical symptoms was not a pre-requisite for admission. In this Department when the child was fully recovered in the Accident & Emergency Unit, discharge home with instructions was acceptable. Similarly, skull fracture of more than 48 hours duration without symptoms or physical signs, was not an indication for admission.

CT Scanning/Neurosurgery

Children requiring CT Scanning or deemed to require Neurosurgery were transferred in the Department's Mobile Intensive Care Ambulance to the Regional Neurosurgical Unit 10 miles away in Glasgow escorted by an Anaesthetist and a Nurse.

GLASGOW ROYAL INFIRMARY

Glasgow Royal Infirmary is a Teaching Hospital situated within the City of Glasgow. It serves an urban population, with some of the problems of inner city deprivation.

1986 - 77,000 new patients in the Accident & Emergency Department.

5,000 head injuries.

25% of the head injuries occurred in children.

Staffing

There were two Consultants in post at the time of the study, 3 Registrars, 7 Senior House Officers, 7 Junior House Officers, plus 9 Clinical Assistant sessions per week. The majority of head injured patients were treated by Senior House Officers who were responsible for their assessment and investigation.

Xrays

Xrays are reported within 24-48 hours by a Radiologist, but advice is available during normal working hours from senior Department staff or Radiologists, on request.

Computerised Scanner

Not available on site, at the time of the Study.

Guidelines for Head Injuries

Guidelines for head injury management are those issued by a Group of Neurosurgeons incorporating the advice given by the Institute of Neurological Sciences, Glasgow.

Admissions

Minor head injuries are admitted to the Paediatric/Orthopaedic ward, occasionally to the Short Stay Ward. Children with skull fractures were transferred to the Royal Hospital for Sick Children, Yorkhill, Glasgow, for observation only.

Neurosurgical Unit

This is situated 2 miles away at the Institute of Neurological Sciences at the Southern General Hospital, Glasgow Transfers were effected in the standard ambulance, provided by Scottish Ambulance Service, with appropriate medical/nurse escort.

Additional Information

The proximity of Glasgow Royal Infirmary to the dedicated Paediatric Hospital, the Royal Hospital for Sick Children, Yorkhill, may have resulted in some of the younger children being taken by parents to that hospital directly.

THE ROYAL ABERDEEN CHILDREN'S HOSPITAL

This is a dedicated Paediatric Unit. The Department serves a population in a mixed urban and widely dispersed rural setting - Grampian Region.

The estimated annual attendance of head injured children was 2,400.

Medical Staffing

There was a Paediatric Surgeon in charge of the Department.

Three Senior House Officers provided a 24 hour service.

Skull Xrays

There was a local policy which accorded with the Adult Guidelines. A fairly liberal xray request policy was applied to infants. Films were reported by a Radiologist within 24-48 hours. Reports were usually available the following day and advice was available on request during normal working hours.

CT Scanner

This was located in the Neurosurgical Unit at Aberdeen Royal Infirmary on the same site.

Admissions

The policy at Royal Aberdeen Children's Hospital was that any child with a history of loss of consciousness, even though fully recovered in the A&E Department should be admitted. Observation was in the Paediatric/Surgical ward. Transfers were made to the Neurosurgical Unit from the Department directly or from the Surgical Ward. This was in accordance with the Adult Guidelines or cases of diagnostic difficulty. The Neurosurgical Unit was unique in this Study in accepting cases from Primary Care doctors. Two children were admitted directly during the period of the Study. Neither had a haematoma.

THE ROYAL LIVERPOOL CHILDREN'S HOSPITAL ALDER HEY

This hospital provides the Regional Paediatric Service for Mersey Health Authority. Serious injuries were taken directly by ambulance to the A&E Department.

Enquires at the adult hospitals in Liverpool suggested an unknown number of minor head injuries were treated in adult units. There was also a walk-in unit at the Casualty Department of the Children's Hospital in the City where minor head injuries were also seen.

Figures for new patient attendances were not available at the time of the Study. The head injuries attending were estimated .

Medical Staffing

One Consultant, one Associate Specialist, one Senior Registrar, one Registrar, 6 Senior House Officers, ? Clinical Assistants.

Skull Xrays

There was a liberal policy with regard to xray encompassing the Adult Guidelines.

A CT scanner was available within the hospital. It was not in use out of hours. There was a hospital protocol for requests, but it was not used on any children during the period of the Study. Those requiring this investigation were transferred to the Neurosurgical Unit.

Admissions

Children were admitted with any history of loss of consciousness at any time, even when fully recovered in the Accident & Emergency Department. Other criteria for admission were those of the Adult Guidelines.

Neurosurgical Unit

The Regional Neurosurgical Unit was situated at Walton Hospital. Transfers were made as indicated in the Guidelines from a Group of Neurosurgeons, by standard ambulance with a medical/ nurse escort.

POOLE DISTRICT GENERAL HOSPITAL

Poole General Hospital serves a resident population of 420,000 with approximately one million visitors per year in the summer months. 300, 000 reside in an urban and 100,000 in a rural setting. The number of paediatric head injuries was not known precisely.

Medical Staffing

There was one Consultant in charge of the Department, one Registrar, 16 Clinical Assistant Sessions and 4 Senior House Officers.

Senior House Officers are most frequently involved in decisions involving head injured patients.

Skull Xrays

The policy for skull xrays within the Department is that incorporated in the advice of a Group of Neurosurgeons. All films were reported by a Radiologist, normally the following day. A radiological opinion could be obtained if required during normal working hours.

CT Scanner

A scanner was available within the hospital on a 24 hour basis. It had very limited use during the period of the Study. The results of availability of CT Scanning in this hospital have been reported. It is generally used to reduce transfers of head injured patients to the Neurosurgical Unit who do not require urgent neurosurgical intervention.

Admissions

Children under the age of 16 are admitted to the Paediatric Ward. Patients requiring Intensive Care or ventilation are admitted to the Intensive Care Unit of Poole General Hospital prior to scanning and/or transfer. Admission policy at Poole was that any patient giving a history of loss of consciousness at any time, regardless of status in the A&E Department was admitted for observation. Just under 1,500 patients were admitted on average per year in the previous 3 years. The proportion of children within this Group was not known.

The Neurosurgical Unit

The Unit is based at Southampton. Transfers involve considerable journeys and the local scanner is used to reduce transfers that may not be beneficial.

Protocol for Scanning

1. Decrease in conscious level.
2. Failure to improve.
3. Localising signs, including epileptic fits.

No complications were reported in those who did not have CT scans and no children were scanned during the period of the study at the base hospital. Similarly, no Paediatric patient was identified as having sustained severe multiple injury in the course of the Survey at this hospital.

HULL ROYAL INFIRMARY

Hull Royal Infirmary is situated within the City of Hull, and serves a mixed/rural population of approximately 500,000.

1987.

New patients 84,000.

Head injuries - 8,000 (estimated)

Paediatric Head Injuries - 40% of total (estimated).

Medical Staffing

The Accident & Emergency Department was in the charge of a full time Consultant, with one Senior Registrar and one Registrar. 22 Clinical Assistant Sessions were available and 9

Senior House Officers were in post. Senior House Officers were the doctors most frequently involved in the initial reception and investigation of the head injured patients.

Skull Xrays

In the Accident & Emergency Department skull xrays were requested within the Guidelines for Head Injury Management from a Group of Neurosurgeons. The films were routinely reported within 24/48 hours by a Radiologist. Advice was available during normal hours from more senior medical staff, and from a radiologist on request.

Admission

Hull Royal Infirmary was unique in the Study in its relationship with the Regional Neurosurgical Unit. The latter is in the same building. A protocol required all children admitted for observation or treatment following head trauma, be transferred routinely to neurosurgical care. The hospital was visited by the author at the commencement, during and at the end of the Study and close liaison established with the Consultants and Registrars. The data collection was suspended for a period due to staff shortages. The Consultant felt that this prevented continuity, and consecutive case identification, with poor quality recording.

Features Common to all Hospitals

In all hospitals a 24 hour Accident Service was offered, and diagnostic imaging in the form of skull xrays were available. Ambulances operated a "nearest hospital" rule, except at Alder Hey where children were taken by ambulance to the Paediatric Service, rather than to the nearby adult Units.

Blood gas analysis was readily available. Steroids were never used. Mannitol was used only on the advice of a Neurosurgeon. Anticonvulsant policy varied with Diazepam or Phenytoin being used initially. Phenytoin was used for continuing control.

Admission Policy - Differences

There were significant differences in the Admission Policy at Alder Hey, Poole and Aberdeen where any history of loss of consciousness at any time, regardless of the condition of the patient in the A&E Department, prompted a request for in patient observation. In Poole and Aberdeen, children who had been brought from a rural location and were therefore at some distance from home were admitted if attending late in the evenings, occasionally in the absence of a strict medical reason .

CT Scanning

This was available on site at Alder Hey, Poole and within the Neurosurgical Unit at Hull. It was provided at Aberdeen Royal Infirmary on the same site as the Children's Hospital. Access followed transfer to Neurosurgical care. At Monklands District General Hospital and Glasgow Royal Infirmary transfer to the Institute of Neurological Sciences at the Southern General Hospital was necessary to gain access to CT Scanning facilities, which were provided 24 hours a day.

PROCEDURE FOR DATA COLLECTION

Inevitably there were minor variations in the procedures in the different hospitals. The method in use at Monklands Hospital, was recommended, when possible, for guidance and the collection of information. The Receptionists were responsible for the issue of the Data Collection Forms and noting the date and time of arrival. Basic observations of pulse, temperature, pupil responses and Glasgow Coma Score was frequently made by nursing staff and incorporated on the form.

Following examination by a Doctor, the form was completed for all children in whom head injury was given as the reason for attendance by a parent or guardian. This was regardless of the interval from injury to examination. Where head injury was detected in the course of examination for another injury or complaint, a form was issued and completed.

Information from observers of the injury incident, ambulance crews or escorting adults was conveyed to the medical staff and recorded on the Casualty Card. This would include any history of unconsciousness, amnesia, fits or deterioration in transit or at the locus of the injury.

Following medical examination, the doctor would decide on the next step to be taken, including a request for skull xray. On completion of full clinical assessment, treatment as indicated and examination of the skull xrays, a decision would be reached regarding disposal. If the child was discharged home the Data Collection Form was completed and returned to the Receptionist for transfer to the Author.

Skull Xray Reports

Skull xrays were interpreted in the course of a clinical assessment by the examining doctor. They would subsequently be submitted to a Radiologist for reporting. The results came too late to influence decisions in the Accident & Emergency Departments.

Skull Fractures

These were most commonly identified by skull xrays. Clinical skull fractures due to penetration might be suspected from the history or mechanism Exceptionally, fracture may be visible or palpable on exploration of the wound. Base of skull fracture was diagnosed clinically.

The reports were made available, usually within 24 hours to be matched against the Casualty Officers interpretation of the films. In Monklands Hospital a daily computer print out of all xrays showing no abnormality is available on a 24 hour basis and served as a double check, both on the Radiologists report and on skull xrays which might have been requested on children out with the A & E Service.

Admissions

When admission or transfer was arranged the form, at this stage incomplete, was held pending further information being recovered. This was subsequently obtained through the liaison person, and the questionnaire was completed after discharge from the hospital and normally within one month. The minority who were admitted, and the smaller number within this group transferred to Neurosurgical Care, had in patient data included as it became available.

Severity of Injury

Initially it had been intended that those with serious or multiple injury would have Injury Severity Scores recorded. This was abandoned, because staff were not, at the time of the Study acquainted with the procedures. The categories were therefore as follows

Minor	Not requiring admission or detained for several hours observation, usually overnight.
Moderate	Requiring admission for up to 48 hours, and out patient follow up.
Serious	Requiring admission for more than one week, and extended follow up.
Critical	Life threatening.
Non survivable	Those who died before arrival in the Accident & Emergency Department were not included in this Study.

These categories were applied to head injury and head injury combined with other injuries. Trauma involving other areas of the body was identified by site.

Further investigations recorded included

1. The Radiologists report on the skull xrays or other imaging procedures undertaken.
2. Transfer to the Neurosurgical Unit within seven days of injury.
3. The results of initial or subsequent brain scanning by axial computerised tomography.
4. Findings at operation in the Neurosurgical Unit, particularly identification of a haematoma.
5. Outcome at one month.

<u>Outcome</u>

The condition of the child one month after injury was discovered by examination of the in patient and Accident & Emergency Records. The level of disability was noted.

Some patients discharged initially from the Accident & Emergency Department were recalled as a result of an Audit of the skull xrays and were included in the Study.

When head injury warnings were issued, the adult/escort was asked to advise the Accident & Emergency Department if any subsequent attendance was made either at another hospital or medical service, as a consequence of the injury. The Author was then to be informed.

DEFINITIONS USED FOR GUIDANCE IN COMPLETING THE DATA COLLECTION FORMS

For the purposes of the Survey a child was under the age of 15 years and the head injury was trauma to the head extending from the upper half of the orbit to the foramen magnum and the vault of the skull above the zygomatic arches, together with any history or evidence to suggest basal skull injury. Facial lacerations and trauma to the facial skeleton, in the absence of other injury to the head, was excluded, but trauma to the base of the nose and the adjacent frontal bone was included.

Scalp Injury

The nature of the scalp injury abrasion/bruising/laceration was to be assessed by inspection and palpation if necessary following shaving or separation of the hair.

Site of Impact to the Head

When not identified by the nature of the scalp injury, a history of the incident from the patient or observer was obtained, and the mechanism of the injury was also considered in identifying the site.

Skull Fractures

These were most commonly identified by skull xray, but occasionally inspection of the wound or palpation confirmed a fracture. Suspicion of fracture due to penetration might be obtained from the history and mechanism. Base of skull fracture was a clinical diagnosis.

The Adelaide Coma Scale (Age Related)

A decision was taken as this was a Paediatric Study to use the Adelaide Coma Scale for the young children in preference to the Glasgow Coma Scale. Instructions to waken the sleeping child were given. The painful stimulus recommended was nail bed pressure. In practice, a medical invasive procedure proved to be as effective e.g. measuring blood pressure or venepuncture!

History of Unconsciousness

A history of loss of consciousness was to be obtained from a parent or observer of the injury event (including a reliable child witness). Expressions such as dazed, limp, glazed eyes and eye rolling which persisted for more than two minutes was regarding as loss of consciousness in the prostrated child. Amnesia caused particular difficulties in the younger age groups.

CAUSES OF INJURY

1. FALLS
2. ROAD TRAFFIC ACCIDENT
3. ASSAULTS, INCLUDING NON ACCIDENTAL INJURY

1. FALLS

Each group included a wide range of movements from toppling over on a level surface to dropping from a height, or tumbling off a moving object. No attempt was made to estimate the velocity in the vast majority of cases, because of the difficulty in obtaining a reasonable estimate, unless a professional observer was involved. Falls were examined in relation to their location. If known the height from which a fall occurred was recorded.

a) Falls at Home

Falls at home included those occurring within the confines of the house or its immediate domestic environment, such as communal stairs, steps, passageways etc. Falls within the garden were generally included within the play injuries.

b) Falls at School

These were included when they occurred on school premises, including the recreational areas. There was some overlap between falls occurring in sport during school hours. It was initially intended that falls at school involving sport should relate only to those occurring in the course of supervised sporting activity. It proved difficult however to determine the nature of some of these activities, which were probably essentially falls at play. Any future research should provide a data collection form which clearly separated these activities.

c) Falls at Sport

Formal organised and supervised sport could, with benefit, have been examined apart from play activities. It clearly varies between different age groups, and my imprecise definition produced problems in analysis. There was difficulty in categorising falls when they occurred at shops, supermarkets and other sites of communal activity. In the majority of cases, it was assumed that the child was accompanying an adult or companion, and would therefore be considered at the time of the injury to be involved in a leisure activity. Inevitably a minority fell within no precise group, for example, falls from supermarket trolleys. By contrast, falls from baby walkers came under the heading of home accidents. Play school and nursery school produced information normally considered out with formal educational definitions, and accounts for some of the very young children in the "school" category.

2. ROAD TRAFFIC ACCIDENTS

This group included those who were injured on a road or its immediate environment as a result of an accident involving a motorised vehicle. The patients fell into three categories:

a) Vehicle Occupants.

b) Pedestrians.

c) Cyclists.

a) Vehicle occupants

These patients were injured whilst travelling by car or other motorised vehicle including vans, lorries, buses and coaches. The car occupants were further sub-divided. Information was obtained regarding their position in the vehicle, and whether or not they were restrained. This included the use of infant car seats, a harness, or adult seat-belts suitably adapted. Some accidents occurred in situations where seat belts were not available, nor required by legislation. Included were the rear compartments of vans, the cabs of lorries, the passenger compartments of taxis and the luggage compartments of estate vehicles.

b) Pedestrians

Pedestrians included children who were involved in accidents whilst walking on roads or pavements. The impact usually resulted from contact with a moving vehicle. Paradoxically it included infants unable to walk, but involved in accidents when being moved by an adult in push chairs. Pedestrian accidents were usually observed or witnessed.

c) Cyclists

Cyclists may have been over-represented within the Road Traffic Accident group if their injury occurred off the public highway and did not involve another vehicle. Some head injuries in the pedal cycle group were recorded as play accidents, because no other vehicle was involved or the incident occurred off a road. This group may have included some very young children who were using play equipment unsuitable for use on the highway, but nevertheless categorised as a cycle or tricycle.

3. ASSAULT

Assault was defined as an injury resulting from the activity of a third party, producing an impact upon the head with malicious intent. This included being struck by missiles, stones, bricks, bottles, drinking cans and other objects. Blows were struck with objects such as baseball bats, golf clubs and a variety of sticks. Trauma to the head occurring as a direct effect of pushing, punching, and tripping incidents were also deemed assaults. In a few the event was not always confirmed as malicious, and some direct blows were in fact accidental, i.e. with items of sports equipment, golf clubs in particular.

Firearms and Related Weapons

Air guns and rifles were rarely a cause in this survey and there were no firearm injuries.

NON ACIDENTAL INJURY

A separate category for child abuse, that is assault within the family or by an adult carer was provided.

MECHANISM OF INJURY

There is inevitably some overlap between the causes of injury and the mechanism. However, an attempt was made to identify the nature of the impact upon the head. Only rarely was accurate information regarding velocity available, though occasionally it could be implied by a fall from a substantial height.

Soft Surfaces

Soft surfaces included upholstered furniture, beds, thickly carpeted floors, sand, some soft grassed areas, particularly when wet, and sports mats.

Sharp Edges

These included the metal edges of toys, prams, push chairs, bicycles and domestic objects and appliances. Broken glass and some plastic objects qualified.

Hard Surfaces

This category covered a wide range with concrete paving stones, tarmac and tiles being common, and solid flat objects made of wood, metal, stone and concrete. Frozen ground and grassed areas in the winter months were included in this category.

Bumps

Bumps were defined as injuries resulting from the head coming in contact with stationary objects whilst moving slowly.

Blows with Sharp Objects

These could be accidental or represent an assault, and the force and velocity was variable.

Blows with Blunt Objects

These similarly be accidental, due to falling objects. Assault was usually associated with some degree of force. Some accidental blows produce serious local injury, as when struck with a golf club.

Miscellaneous

There were a number of children in whom the precise mechanism was difficult to confirm. This included accidental injury as a result of falling objects, pictures, vases, items of furniture, television sets etc. A variety of objects was observed to strike the head. There were inevitably areas where the doctor applied professional discretion to the explanation offered, or made deductions from the information available or offered.

THE PRESENTING SYMPTOMS AND SIGNS

The number of children brought to hospital with a history of head injury included some who had neither symptoms nor signs compatible with head trauma. It appeared that some of these were brought by anxious relatives, on the basis of an observed or assumed incident, for re-assurance. Others with a history of remote head injury often had other causes for their symptoms discovered(see below).

Headache

Headache is a subjective symptom. It may be under-recorded in infants and toddlers incapable of making the complaint. In the older child headache may be difficult to separate from pain localised to the site of injury. Both symptoms may be present. The headache is likely to be underestimated in the younger age groups, but may be over-estimated in those who refer simply to local pain or discomfort. Distress may however raise suspicion even in the younger child.

Vomiting

Vomiting is objective, readily observed, and unlikely to be other than genuine in the paediatric age group. The number of vomiting episodes was recorded.

Pupillary reaction

Even lighting and a strong torch was recommended for this examination. Apart from local swelling and trauma, its observation presented few problems, though reactions are much more labile in children.

Focal Signs

Simplified neurological examination was recommended, accepting the limitations of lack of co-operation by the infant and young child. Response to simple command and to painful and non painful stimuli was necessary for eliciting these signs.

Deterioration

The advice for completion of the questionnaire stated that deterioration referred specifically to the level of consciousness and the responsiveness of the child to the components of the Coma Scoring System in use.. It was assumed that this would be reflected in alteration in the Coma Score. There were however many instances, detected by the Author's inspection of the records, where deterioration was recorded based on an increase in severity of headache or repetition of vomiting, or even its onset. This was not always matched to a deterioration in the level of consciousness. It does have implications for admission, and the application of guidelines for CT Scanning or Neurosurgical advice and intervention.

Verbalisation/Dysphasia/Aphasia

Speech presents particular problems in the young, and even the apprehensive older child. Neurological development is taken into account in scoring.

Double Vision/Loss of Vision and Blurring of Vision

Visual impairment was not provided in the questionnaire. This was a deficiency as a number of children volunteered blurring of vision as a symptom and in three cases, loss of vision, as part of their presenting features. This information was gathered by additional comments made on the form, or by retrieval from the hospital records by the Author. Therefore the precise numbers are likely to be inaccurate and possibly underestimated.

Pyrexia

Nursing staff were responsible for recording the body temperature and it was recommended that this should be done rectally. History and examination was required to identify alternative sources of pyrexia, with in patient observation and investigation as necessary.

Epilepsy

Transient eye rolling immediately following the impact was excluded from the diagnosis. However if this persisted for more than 1-2 minutes, was associated with loss of responsiveness and focal or generalised twitching, a diagnosis of epilepsy was recorded. A past history of epileptic attacks was noted and its relationship to the trauma incident assessed.

Febrile Convulsions

Febrile convulsions were diagnosed on the basis of a preceding history of infection, the confirmation of pyrexia and its possible cause. Doctors were requested to add this to the diagnosis as an additional comment.

Multiple Injury

Multiple injury was defined as one or more injuries in addition the head injury which would inevitably necessitate admission for longer than 24 hours. In moderate and severe injury resuscitative procedures would be required and recorded.

DISPOSAL FROM THE ACCIDENT & EMERGENCY DEPARTMENT

The most common outcome of attendance at hospital following head injury in childhood is discharge home in the care of the family with supervision from the family doctor. The options for admission were:

1. A Short Stay Ward under the care of a Consultant in Accident & Emergency Medicine or other Specialist.
2. A Paediatric or Primary Surgical Ward, depending on where in patient care was provided for the child with a head injury in the particular hospital.
3. A Neurosurgical Unit directly, by transfer from the Receiving Hospital.
4. A General or Paediatric Intensive Care Unit, usually the consequence of other injuries associated with the head injury.

The primary surgical ward in the paediatric hospital was by definition under the supervision of Paediatric Surgeons.

Neurosurgical Units/Transfer

Children who required neurosurgical admission had to be transferred from the Accident and Emergency Department or a ward to a Regional Centre in four of the six hospitals. At Aberdeen the Neurosurgical Unit is on the adjacent site at Forresterhill. At Hull Royal Infirmary the Neurosurgical Unit is within the hospital. Distant transfers were made by ambulance road transport in all cases.

Predictor

The problems of definition have arisen in this Study, and a review of the literature in relation to the use of "predictor". The Oxford Dictionary, 1995, defines a predictor as "a statement about the future", "a prophesy". Teasdale has used this term to express the risk of intracranial haematoma developing or manifesting itself particularly in association with a skull fracture. Identification of this risk is crucial to investigation and decision making in the Accident & Emergency Department or emergency rooms.

Many authors have used the "predictor", in place of "confirmation".

Predictor refers to that which may subsequently manifest as a risk, where as CT in the publications of many authors is essentially the technical confirmation by CT braining scanning of abnormalities associated with clinical evidence of established intracranial abnormality/pathology.

Confirmation of injury on CT scanning is most frequently seen in children already admitted because of clinical symptoms or signs or the history of the event.

Predictors direct the investigative procedures which will detect the unexpected and initially silent condition in those who appear well and may have a Glasgow Coma Score of 15.

It is in the Accident & Emergency Department that such predictive features are of value in securing the appropriate investigation and allowing discharge of those children in whom there is no evidence of intracranial abnormality.

The undoubted value of a predictor applies to intracranial haematoma and particularly to extradural haematoma or acute subdural haematoma in childhood, where delays to neurosurgery significantly affect the morbidity and mortality.

In summary CT scanning which confirms suspected abnormalities which are not of surgical interest, should be clearly differentiated from CT scanning which identifies a neurosurgical lesion capable of effective intervention, before it manifests as an intracranial expanding mass lesion, with all the implications for delay and subsequent complications.

Appendix B

DATA COLLECTION

FORM

MONKLANDS DISTRICT GENERAL HOSPITAL

STUDY CO-ORDINATOR - DR. MARIE T. BROOKES, A/E DEPARTMENT

OFFICIAL USE: [| | |]

PAEDIATRIC HEAD INJURY DATA COLLECTION FORM (14 YEARS AND UNDER)

Names: ....
Sex: ....
Date of Birth ....
Age in years: ....
Hospital: ....
Hospital No: ....
Parents Occupation: ....
Employed: ....
Unemployed: ....
Single Parent: ....

	Day		Mth		Yr		Hrs		Min	
Date and Time of Injury:										
Date and Time of Examination:										

Disposal
1. Home
2. Short Stay Ward
3. Primary Surgical Ward
4. Neurosurgery Unit

PLEASE COMPLETE ALL BASIC DATA

Computer Codes (leave blank)

A1 [ ]
A2 [ ]
A3 [ ]

A1. Scalp Injury (nature) (excluding facial injuries)

1. No external injury
2. Swelling only
3. Abrasions/contusions only
4. Superficial laceration < 5 cm long
5. Superficial laceration > 5 cm long
6. Laceration through galea < 5 cm long
7. Laceration through galea > 5 cm long

A2. Scalp Injury (site)

1. None
2. Frontal
3. Termporal
4. Parietal
5. Occipital
6. More than one site

A3. Skull Fracture (exclude facial, mandibular and nasal injuries) Score Highest

1. No skull x-ray
2. X-Ray - no fracture
3. Clinical fracture of base (CSF/blood in nose or ear/bilateral periorbital haematoma)
4. Linear fracture
5. Depressed fracture
6. More than one

9926

B. Glasgow Coma Scale on admission (Adelaide modification for children)
(score highest appropriate for age at time of examination; score zero if unknown or uncertain)
(Do not score higher that the age limit given).

B1. Eyes Open

4. Spontaneously
3. To speech
2. To pain
1. None

B2. Motor

		Age
6.	Obeys commands	>2 years
5.	Localise pain	6 mth-2yrs
4.	Normal flexion pain	<6 mths
3.	Spastic flexion pain	
2.	Extension pain	
1.	No motor response	

B3. Verbal

		Age
5.	Orientated to place	> 5 yrs
4.	Words	> 12 mths
3.	Vocal sounds	
2.	Cries	<6 mths
1.	None	

B4. 8 Ocular injury/abnormality
9 Closed

Leave Blank

B1	
B2	
B3	
B4	

C. History of Unconsciousness/amnesia

1. None
2. Less than 5 minutes with full recovery of consciousness
3. Five to 30 minutes with full recovery of consciousness
4. 30 to 60 minutes with full recovery
5. Greater than 1 hour and/or still disorientated or worse

C2. Cause of Injury

1. RTA - occupant
2. RTA - pedestrian
3. RTA - pedal cycle
4. Assault
5. Fall - school
6. Fall - home
7. Fall - sport/play
8. Assault - NAI

C3. Mechanism of Injury

1. Fall to soft surface
2. Fall to sharp edge
3. Fall to hard surface
4. Bump against sharp edge
5. Bump against hard flat surface/s
6. Struck with blunt object/s
7. Struck with sharp object/s

C4. Deterioration

1. No
2. Yes

C5. RTA - Vehicle Occupant - (Seat Belt, Harness, Seat)

1. Restrained
2. Not restrained

C6. Position

1. Front Seat
2. Back Seat
3. Other (Estate, Van)

C1	
C2	
C3	
C4	
C5	
C6	

Leave Blank

D1. Headache

1. None
2. Headache

D2. Vomiting

1. None
2. Vomited (once)
3. Vomited (more than once)

D3. Pupils

1. Both reacting equal
2. Both reacting unequal
3. One reacting
4. Neither reacting
5. Local factors affecting

D1 [ ]
D2 [ ]
D3 [ ]

E. Focal Signs

1. None
2. Hemiparesis
3. Hemiplegia
4. Dysphasia/Aphasia
5. 2 or 3 + 4

E2. Pyrexia

1 = TEMP < 37.0C
2 = TEMP 37.1-39.9C (No source of infection)
3 = TEMP > 40.0C (No source of infection)
4 = TEMP 37.1-39.9C (source of infection)
5 = TEMP > 40.0C (source of infection)

E3. Epilepsy

1. No
2. New Post/Traumatic Fit - Focal
3. New post/Traumatic Fit - General
4. Known Epileptic - Focal Fit
5. Known Epileptic - General Fit
6. Known Epileptic - No Fit

E1 [ ]
E2 [ ]
E3 [ ]

Last Recorded in A.E.D.

Leave Blank

F. Other Injuries

1. Upper Limbs
2. Lower Limbs
3. 1 and 2
4. Trunk
5. 4 and 1 or 2

F2. Systolic BP (mmHg)

F3. Pulse Rate

F4.Pa02 (mmHg)

F1

F2

F3

F4

NEUROSURGICAL - REVIEW DATA

G1. CI Scan (Haematoma)

1. Right
2. Left
3. Post Fossa
8. Clot

No Scan

G2. CI Scan (Haematoma)

1. Extradural
2. Subdural
3. Intracerebral
4. 1 + 2
5. 1+ 2
6. 2 + 3
7. 4 + 3
8. No Clot
9. No Scans

G3. Craniotomy

1. Yes
2. No
3. Burr Holes
4. Other Operation/s

G4.

1. I.C.P. elevated

G1

G2

G3

G4

H1. Ventilation

1 No resp. problem
2 Oxygen therapy only
3 Intubated only
4 Intubated and ventilated

H.2 Outcome at 1-7 days

1 Good Outcome
2 Moderate disability
3 Severe disability (dependant)
4 Vegetative survival
5 Death

Leave Blank

H1 [ ]

H2 [ ]

J INJURY SEVERITY SCORE

If available

AIS SEVERITY CODE

Head Injury - 1 Minor
2 Moderate
3 Serious
4 Severe
5 Critical
6 None survivable

OTHER INJURIES

1 Minor
2 Moderate
3 Serious
4 Severe
5 Critical
6 None survivable

J1	K1
J2	K2
J3	K3
J4	K4
J5	K5
J6	K6

Signed: ....

SHO. REg. SR. Cons. (other)

Date: ....

ALL ENQUIRIES TO:- DR. MARIE T. BROOKES, CONSULTANT IN ACCIDENT & EMERGENCY MEDICINE, MONKLANDS DISTRICT GENERAL HOSPITAL, MONKSCOURT AVENUE, AIRDRIE. ML6 0JS - Tel: AIRDRIE. 69344.

YOUR HELP AND COOPERATION IS MUCH APPRECIATED.

REVIEW 7 - 14 days

SIGNATURE: ....

DATE: ....

ALL DATA COMPLETE:

DATE: ....

SIGNATURE: ....

RETURN: MONKLANDS DISTRICT GENERAL HOSPITAL

DATE: ....

SIGNATURE: ....

M. RADIOLOGISTS REPORT:

1	No Skull X-Ray	M1	☐
2	X-Ray - No Fracture	M2	☐
3	Clinical fracture of base (CSF/blood in nose or ear/bilateral periorbital haematoma)	M3	☐
4	Linear Fracture	M4	☐
5	Depressed fracture	M5	☐
6	More than one	M6	☐

MONKLANDS PAEDIATRIC HEAD INJURY STUDY

CODING INSTRUCTIONS

For all the variables: If information is not recorded please leave blank unless otherwise instructed. Below is a description of the variables in the Study with the details of the columns in which they are to be coded.

VARIABLES	COLUMNS
HOSPITAL NUMBERS	1-2

THE HOSPITALS SHOULD BE CODED AS FOLLOWS

Monklands District General Hospital	= 01
Glasgow Royal Infirmary	= 02
Royal Aberdeen Children's Hospital	= 03
Alder Hey Children's Hospital	= 04
Poole General Hospital	= 05
Hull Royal Infirmary	= 06

STUDY NUMBER

A simple running number assigned by us for each Patient as an identified. For each hospital code from 00001 – 99999 — 3 – 7

SEX — 8

Male = 1 Female = 2

DATE OF BIRTH — 9 – 14

Date of birth coded as DDMMYY

If only age is known please calculate year of birth and insert This in columns 13 – 14

OCCUPATION OF PARENTS — 15

- Employed = 1
- Unemployed = 2
- Single Parent = 3

DISPOSAL — 16

Code as on form i.e.

Home	= 1
Short Stay Ward	= 2
Primary Surgical Ward	= 3
Neurosurgery Unit	= 4

TIME OF INJURY	23 – 36
DATE OF INJURY	17 - 22
Code as DDMMYY	
Coded on 24 hour clock HHMM	
If not recorded as 9999	

PAEDIATRIC STUDY CODING INSTRUCTION

Date of Examination	Coded as DDMMYY	27 -32

TIME OF EXAMINATION	33 - 36
Coded on 24 hour clock, HHMM. If not required code as 9999	

A1	Scalp Injury	37
A2	Scalp Injury (site)	38
A3	Skull Fracture	39
B1	Eyes Open	40
B2	Motor	41
B3	Verbal	42
B4	Other GCS	43
C1	History of Unconsciousness	44
C2	Cause of Injury	45
C3	Mechanism of Injury	46
C4	Deterioration	47
C5	RTA	48
C6	Position	49
D1	Headache	50
D2	Vomiting	51
D3	Pupils	52
E1	Focal Signs	53
E2	Pyrexia	54
E3	Epilepsy	55
F1	Other Injuries	56
F2	Systolic BP (90 would be coded as 090)	57 - 59
F3	Pulse Rate (86 would be coded as 086)	60 - 62
F4	Pa 02	63 - 65
G1	Scan (Haematoma Site)	66
G2	Scan (Haematoma Type)	67
G3	Craniotomy	68
G4	ICP (code 1 if elevated, blank otherwise)	69
H1	Ventilation	70
H2	Outcome	71
J1	AIS Severity Code – head injury	72
K1	AIS Severity Code – other injuries	73

DETAILED RESULTS

AND

STATISTICAL ANALYSES

THE RESULTS

Data was collected from Six Accident and Emergency Departments. In Monklands the patients attended over a period of one year. In the other Units, data was collected on consecutive patients over a shorter period.

CODE	HOSPITAL	5993
01	Monklands District Hospital	2104
02	Glasgow Royal Infirmary	805
03	Royal Aberdeen Children's Hospital	1000
04	Royal Liverpool Children's Hospital – Alder Hey	931
05	Poole General Hospital	287
06	Hull Royal Infirmary	866

Data on almost 6,000 children was analysed.

1. Results – Age and Sex

TABLE 1 (i) ALL PATIENTS/SEX

	N	%
All patients	5993	-
Male	3958	66
Female	2032	34
N/R	3	-

AGE AND SEX

The children were divided into groups which approximated to infancy and babyhood, pre-school years and primary and secondary years. The incidence in the different age groups is shown in Table 1 (ii)

TABLE 1 (ii) PATIENTS BY AGE

	N	%
< 1 years	468	8
1-4 years	2664	45
5-11 years	2289	39
12-14 years	490	8
N/R	82	-

TABLE: (iii) PATIENTS BY AGE/SEX

	Male	Female	% Male
< 1 years	265	203	55
1-4 years	1663	999	63
5-11 years	1606	683	70
12-14 years	368	132	72

TABLE 1 (iv): AGE/SEX

Age	Male	Female	All	% male
0-1	265	203	468	57
1	540	338	878	62
2	423	286	710	60
3	373	210	583	64
4	327	165	493	66
5	358	160	518	69
6	308	146	454	68
7	230	107	337	68
8	247	70	317	78
9	163	75	238	69
10	151	65	216	70
11	149	60	209	71
12	174	52	226	77
13	111	44	155	72
14	78	30	109	73
N/R	56	15	72	78

The male dominance increases with age.

Figure: 1 (a) PATIENTS BY AGE/SEX

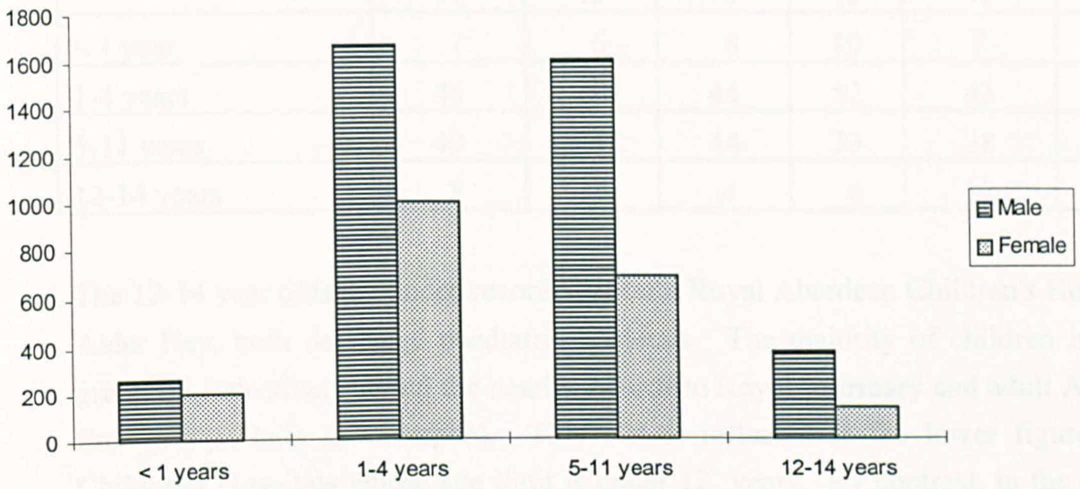


Figure 1 (b) PATIENTS AGE/SEX

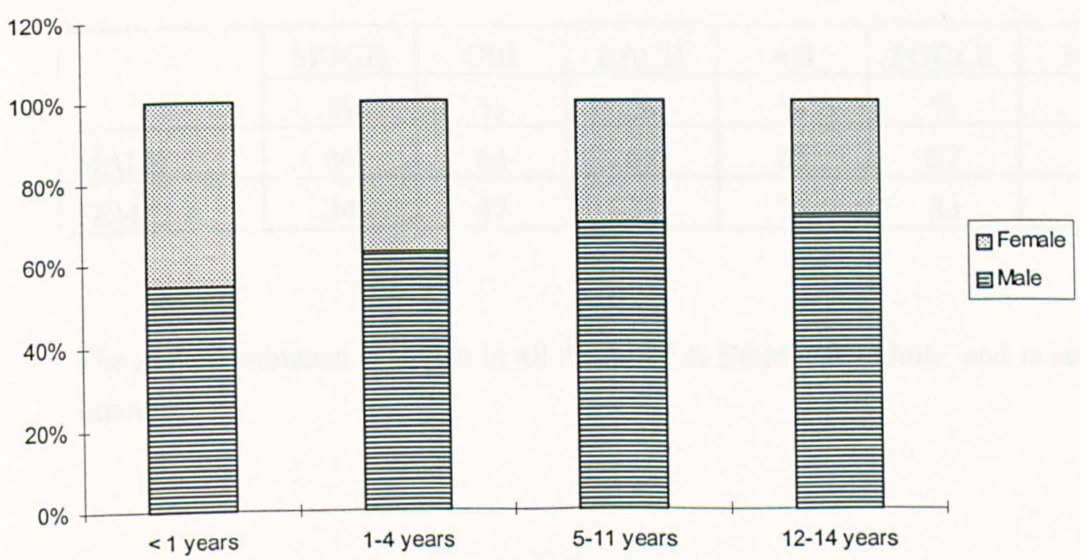


TABLE 1 (v) PATIENTS BY AGE GROUP/HOSPITAL

	MDGH	GRI	RACH	AH	POOLE	HULL
	%	%	%	%	%	%
< 1 year	7	6	8	10	7	9
1-4 years	44	40	44	52	43	46
5-11 years	40	41	44	33	38	34
12-14 years	8	13	4	6	12	10

The 12-14 year olds are under-recorded at both Royal Aberdeen Children's Hospital and Alder Hey, both dedicated paediatric hospitals. The majority of children in this age group may electively attend the nearby Aberdeen Royal Infirmary and adult Accident & Emergency Units in Liverpool. This fact is reflected in the lower figures for the Children's Hospitals whose age limit is under 12 years. By contrast, in the catchment area of Glasgow Royal Infirmary, whilst the ambulance service operates a "nearest hospital" rule for 999 calls, patients have a choice between the adult Accident & Emergency Department and the Royal Hospital for Sick Children. Tradition suggests that for children, particularly in the younger age groups, parents prefer the Paediatric Unit.

TABLE: 1 (vi) SEX DISTRIBUTION/HOSPITAL

	MDGH	GRI	RACH	AH	POOLE	HULL
	%	%	%	%	%	%
MALE	66	63	67	65	67	69
FEMALE	34	47	33	35	33	31

The male dominance is shown in all Accident & Emergency Units, and is seen even in infants.

TABLE 1 (vii) SKULL XRAY/AGE/SEX

	N	% XR	Male % #	N	%XR	Female % #
<1 year	265	69	7.7	203	70	7.7
1-4 years	1663	61	1.5	998	65	1.8
5-11 years	1606	63	2.5	683	65	2.3
12-14 years	364	70	3.9	126	68	2.3

The substantial increase in the fracture rate in males 12-14 years reflects the more serious injury sustained.

TABLE 1 (viii) SKULL FRACTURE/AGE/SEX

	Male %XR	Female %XR
<1 year	7.7	7.7
1- 4 years	1.5	1.8
5-11 years	2.5	2.3
12-14 years	4.0	2.3

The results expressed as a percentage of the skull xrays confirmed the vulnerability of the infant skull, and the more serious injury in the older male child.

TABLE 1 (ix) AGE/FRACTURE

	N
< 1 year	25
1- 4 years	27
5-11 years	35
12-14 years	12

The vulnerability of the infant skull is confirmed.

Risk of fracture by a single factor

Age group	Relative Risk
< 1 year	0.054
1- 4 years	0.010
5-11 years	0.015
12-14 years	0.0124

The infants are most likely to have fractures, 1-4 years old least likely

1. Age & Sex - Univarate Analysis

Sex

Male Dominance

Significantly more than half the children presenting with head injury at the six centres were male, (one-sample t-test, HO: proportion of males = 50%, t=19.4, df=5, p<0.0005, 95% CI=(64%, 68%)).

TABLE 1 (SA)(i) Expected and Observed number of patients of each sex by age (Under H0: age of patients is independent of sex)

			SEX		Total
			Male	**Female**	
AGE	**<1 year**	Count	276.0	207.0	483
		Expected Count	318.3	164.7	483
	1-4 years	Count	1650.0	992.0	2642
		Expected Count	1741.0	901.0	2642
	5-11 years	Count	1584.0	676.0	2260
		Expected Count	1489.3	770.7	2260
	12-14 years	Count	351.0	123.0	474
		Expected Count	312.4	161.6	474
Total		Count	3861.0	1998.0	5859
		Expected Count	3861.0	1998.0	5859

Risk of Fracture by Age

The subjects were categorised by age. The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the age of the child, (chi-squared = 47.4, df=3, p<0.0005). In particular the infants (under 1 year) had substantially more fractures than expected.

The number of children in each age group with skull fractures and the expected number under a hypothesis of independence is shown in Table 1(SA)(ii).

TABLE 1(SA)(ii): Expected and Observed number of skull fractures by age (Under H0: skull fracture is independent of age)

			SKULL #		Total
			No Fracture	**Fracture**	
AGE	**<1 year**	Count	455.0	26.0	481.0
		Expected Count	472.4	8.6	481.0
	1-4 years	Count	2600.0	29.0	2629.0
		Expected Count	2582.1	46.9	2629.0
	5-11 years	Count	2217.0	35.0	2252.0
		Expected Count	2211.9	40.1	2252.0
	12-14 years	Count	458.0	14.0	472.0
		Expected Count	463.6	8.4	472.0
Total		Count	5730.0	104.0	5834.0
		Expected Count	5730.0	104.0	5834.0

When children under 1 are compared directly to the other subjects, they demonstrate a significantly higher risk than older children (chi-squared = 39.6, df=1, p<0.0005) and have an odds ratio of 3.8 (CI=(2.4,6.0)). Therefore a child under 1, presenting with a head injury, is almost 4 times more likely to result in a skull fracture than an older child.

The odds ratios and 95% confidence intervals for each age group are:

	Odds Ratio	**CI**
< 1 year	3.8	(2.4, 6.0)
1-4 years	0.47	(0.30, 0.72)
5-11 years	0.80	(0.53, 1.21)
12-14 years	1.79	(1.01, 3.17)

2.(a) Results – Causes of Injury

TABLE 2(a)(i) CAUSES OF INJURY

	N	%
Patients	**5993**	-
FALLS	4934	82.4
RTA	354	6.0
ASSAULTS	547	9.5
NAI	22	0.4
N/R	135	-

FIGURE 2(a)i CAUSE OF INJURY

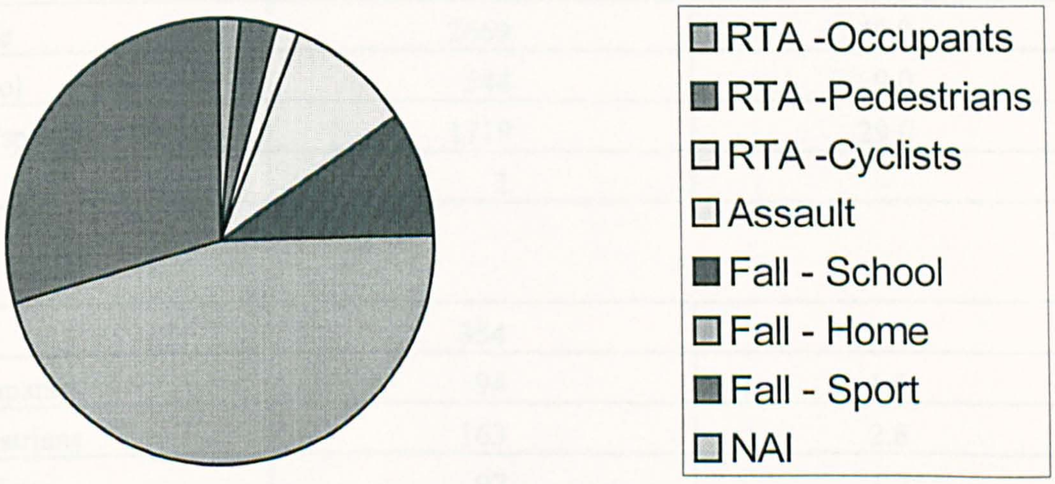
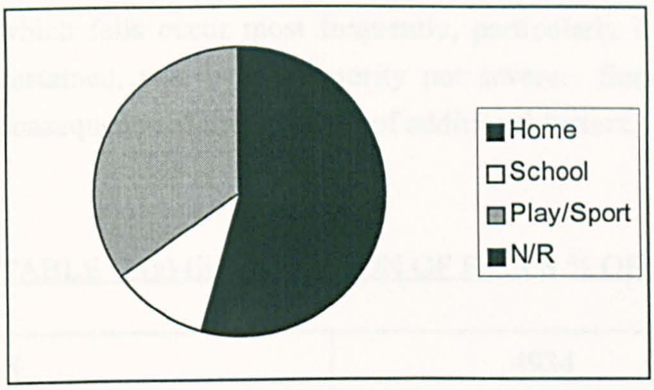


FIGURE 2 (a)2 FALLS – LOCATION



The causes were further subdivided.

TABLE 2(a)(ii) CAUSES OF INJURY AS % OF ALL CAUSES

FALLS	N	%
N	**4934**	
Home	2669	46.0
School	544	9.0
Play/sports	1719	29.0
N/R	2	-

RTA

N	**354**	
Occupants	94	1.6
Pedestrians	163	2.8
Cyclists	97	1.7

ASSAULTS

547	9.5

NAI

22	0.4

Falls

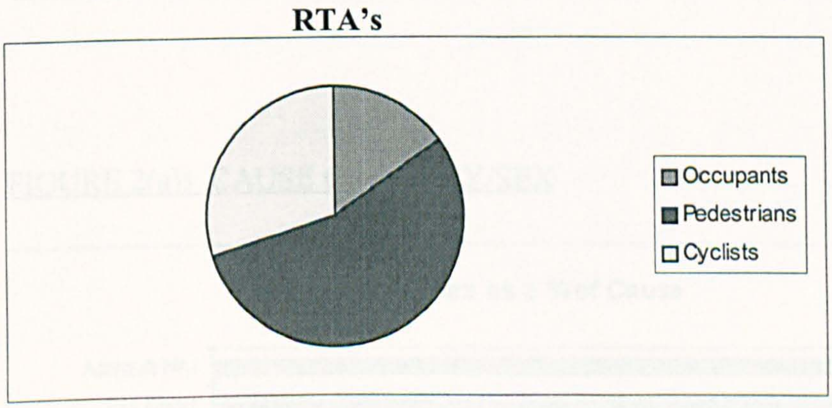
Falls were the most common cause of childhood injury, and the house the location in which falls occur most frequently, particularly in the younger children. The injury sustained, was in the majority not severe. Some falls produced severe injury, the consequence of the interplay of additional factors.

TABLE 2 (a) (iii) LOCATION OF FALLS % OF ALL FALLS

N	4934	% of all falls
Home	2671	54.0
School	544	11.0
Play/Sport	1719	34.0
N/R	2	-

Road Traffic Accidents

FIGURE 2(a)3 CAUSE OF INJURY



Road Traffic Accidents accounted for 6% of all the causes of head injury seen in the Emergency Department in this Study. The proportion is much less than those admitted to hospital or to Neurosurgical Units, where the more serious are treated. Children injured in Road Traffic Accidents were subdivided into Vehicle Occupants, Pedestrians and Cyclists.

Cause of Injury/Sex

The dominance of the male child in head injury statistics, is noted in all publications suggesting that sex may influence cause and mechanism. Anatomical and physiological factors may operate, but the resulting pathophysiological responses are likely to be similar for the same cerebral insult.

FIGURE 2(a)4 CAUSE OF INJURY/SEX

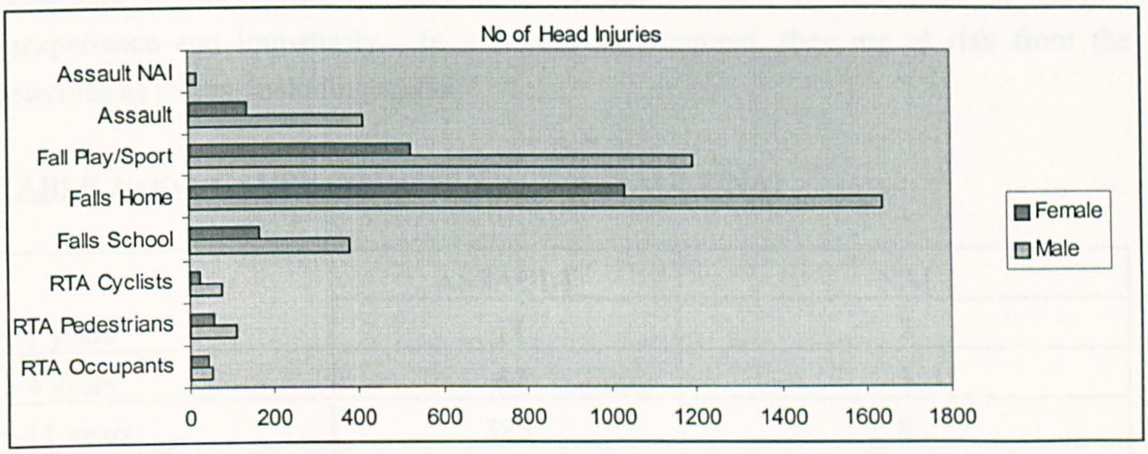


FIGURE 2(a)5 CAUSE OF INJURY/SEX

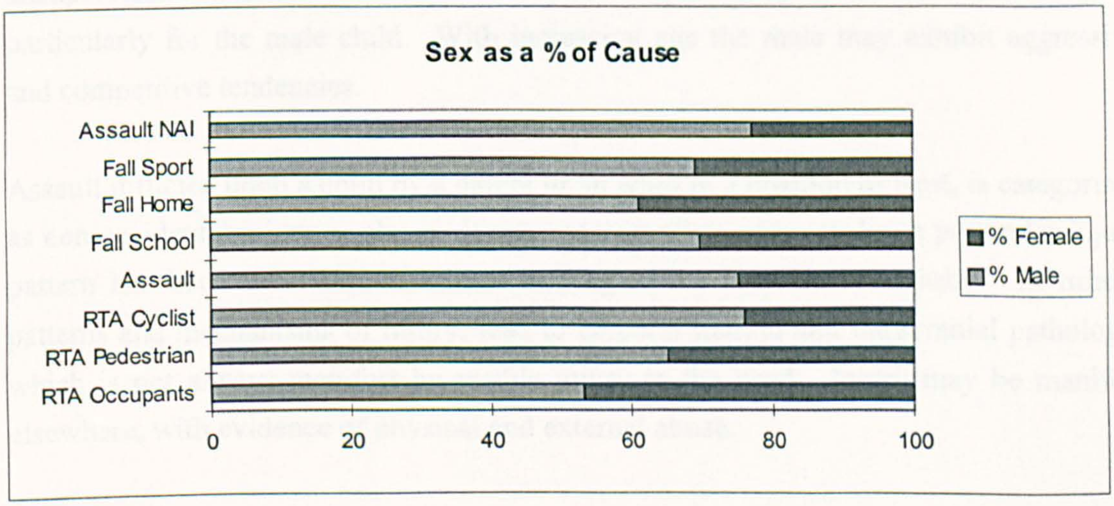


TABLE 2(a)(iv) CAUSE OF INJURY/AGE/RTA

RTA'S	Occupants	Pedestrians	Cyclists
<1 year	9	1	0
1-4 years	40	41	21
5-11 years	38	100	51
12-14 years	6	19	20

Children who travel as occupants are passive victims, dependent on adults for their safety and protection. As they become more active and independent they may contribute to the accident. This may be due to lack of awareness of danger, inexperience and immaturity. In a hostile environment, they are at risk from the activities of others, including adults.

TABLE 2(a)(v) CAUSE OF INJURY/AGE/ASSAULT/NAI

	ASSAULT	NAI
< 1 years	11	7
1-4 years	87	3
5-11 years	325	8
12-14 years	113	3

Children who are assaulted are mainly at risk, after they leave the protective environment of the house and family, and out with school. Participation in group and unsupervised activities. Contact and competitive sports may increase the risk of injury, particularly for the male child. With increasing age the male may exhibit aggressive and competitive tendencies.

Assault inflicted upon a child by a parent or an adult in a position of trust, is categorised as non-accidental injury or abuse. It can occur in all age groups, but a particular injury pattern has been described in infants leading to the term battered baby. In infants patterns and mechanisms of injury, lead to cerebral trauma and intracranial pathology, which is not always manifest by visible injury to the head. Injury may be manifest elsewhere, with evidence of physical and external abuse.

TABLE 2 (a)(vi) CAUSE OF INJURY/SEX

RTA	N	Male	Female	%Male
	354	231	123	65.2
Occupants	94	50	44	53.0
Pedestrians	163	107	56	65.0
Cyclists	97	74	23	76.0
FALLS	N	Male	Female	% Male
	4934	3210	1722	65.0
School	544	379	165	69.6
Home	2669	1637	1032	61.0
Play/Sport	1719	1194	525	69.0
N/R	2	-	-	-
ASSAULT	N	Male	Female	%Male
	547	411	136	75.0
NAI	22	17	5	77.0
TOTAL ALL CAUSES	**5855**	**3869**	**1986**	**66.0**

Child occupants are passive participants in the injury event. By contrast the pedestrian and cycle accident involve predominantly male children.

The male dominance is reflected for all causes, though in infant abuse the child is regarded as a passive victim?

Skull Fracture/Cause of Injury

The detection of skull fracture for all causes has been examined, in an total of 3,826 skull series requested. Radiologists detected 102 fractures. Nine basal fractures were diagnosed clinically. One further basal fracture was confirmed by special imaging. The incidence of skull fracture as a percentage of the cause is shown TABLE 2 (viii).

TABLE2(a)(vii) CAUSE OF INJURY/ AGE / FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	9	0		40	0		36	2	5.3%	6	0		92	2	2.1%
RTA - PEDESTRIAN	1	0		37	4	9.8%	96	4	4.0%	15	4	21.1%	151	12	7.4%
RTA - CYCLIST	2	0		20	1	4.8%	48	3	5.9%	20	0		93	4	4.1%
ASSAULT	11	0		86	1	1.1%	323	2	0.6%	111	2	1.8%	543	5	0.9%
FALL - SCHOOL	8	1	11.1%	83	2	2.4%	365	1	0.3%	77	0		540	4	0.7%
Fall - Home	369	18	4.7%	1733	5	0.3%	461	1	0.2%	51	2	3.8%	2642	29	1.1%
Fall - Sport	21	2	8.7%	586	14	2.30%	867	22	2.5%	182	2	1.1%	1679	40	2.3%
NAI	6	1	14.3%	3	0		8	0		2	1	33.3%	20	2	9.1%
MISSING / NOT RECORDED	16	3	15.8%	49	0		50	0		14	1	6.7%	131	4	3.0%

TABLE 2 (a)(viii) FRACTURES AS % OF CAUSE

	%
FALLS	7.0
School	0.7
Home	1.0
Play/sport	2.3
ALL FALLS	1.5
RTA'S	
Occupants	2.1
Pedestrians	7.5
Cyclists	4.1
ALL RTA	5.0
ASSAULT	0.1
NAI	9.1

When the incidence of fracture is examined for the cause of injury in all ages combined the following is noted

CAUSES		FRACTURE FREQUENCY
RTA	Occupants	0.021
	Pedestrians	0.074
	Cyclists	0.041
FALLS	School	0.007
	Home	0.011
	Play/Sport	0.023
ASSAULT		0.009
NAI		0.091

When the frequency of fracture is related to the cause of injury it is seen that falls, the largest numerical group, has the lowest fracture rate, in the Emergency Department.

Road Traffic Accidents, particularly those involving child pedestrians and cyclists has the greatest incidence of fracture.

The identification of RTA accidents as a cause of more severe head and cerebral trauma in childhood is likely to prompt Casualty Medical Officers to request skull xrays - in this group of patients.

The low incidence of fracture in the larger number of children, who fall may provide an opportunity to reduce the number of requests for xrays. The cause will not be the only factor influencing the doctor.

The scalp injury feature, the site of the impact and the mechanism may need to be taken into account as may symptoms and neurological signs.

It has been suggested that the nature of the scalp injury resulting from a particular cause might influence the casualty medical officers decision to request skull xrays. The cause of injury, be it a fall or a category of road traffic accident might result in a scalp injury with a higher or lower risk of skull fracture. The cause of the injury, was examined in relation to the nature of the scalp wound sustained and the age and incidence of skull fracture. The results are shown in Tables 2(a)(ix–xiii).

When the causes of injury were examined in all age groups, the absence of any recorded scalp feature was noted in 868 children, only seven of whom sustained a skull fracture, 0.8%. The presence of swelling of the scalp was noted in 1,685 and 57 fractures were detected. The incidence was highest in children hurt in road traffic accidents, followed in frequency by falls in sport/play and at home. The vulnerability of the infants skull is again demonstrated where 14/17 fractures resulted from falls 3.4%.

Abrasions and contusion of the scalp was recorded in 961, fractures 22, 2.3%. The incidence was highest in the road traffic accidents followed by falls.

The short superficial lacerations, a large group, 2342 revealed only nine fractures, 0.4%. It was not recorded in road traffic accident trauma. The highest incidence of fracture was recorded in child abuse.

The long superficial lacerations of which there were 40 had no fractures detected from all causes.

TABLE2(a)(ix) CAUSE SCALP INJURY / AGE / FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
NO EXTERNAL INJURY	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	2	0		7	0		8	0		3	0		20		
RTA - PEDESTRIAN	1	0		4	2	33.3%	11	0		0	0		17	2	10.5%
RTA - CYCLIST	0	0		1	0		5	0		5	0		11		
ASSAULT	1	0		6	0		28	0		16	0		52		
FALL - SCHOOL	4	0		19	0		58	0		18	0		99		
FALL - HOME	115	0		222	0		51	0		7	0		398		
FALL - SPORT	5	0		66	3	4.3%	115	1	0.9%	50	1	2%	240	5	2%
NAI	1	0		0	0		1	0		0	0		2		
MISSING / NOT RECORDED	7	0		5	0		9	0		1	0		22		
TOTAL	136	0		330	5	1.5%	286	1	0.3%	100	1	1.0%	861	7	0.8%

TABLE 2(a)(x) CAUSE SCALP INJURY / AGE / FRACTURE

SWELL ONLY	1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No	#	% #	No	#	% #	No	#	% #	No	#	% #	No	#	% #
RTA - OCCUPANT	4	0		4	0		14	2	12.5%	2	0		24	2	7.7%
RTA - PEDESTRIAN	0	0		12	1	7.7%	25	1	3.8%	3	3	50%	41	5	10.9%
RTA - CYCLIST	1	0		6	0		15	3	16.7%	3	0		25	3	10.7%
ASSAULT	4	0		11	0		47	0		31	1	3.1%	95	1	1.0%
FALL - SCHOOL	3	1	25.0%	24	1	4.0%	109	1	0.9%	28	0		166	3	1.8%
FALL - HOME	138	11	7.4%	491	4	0.8%	115	1	0.9%	8	1	11.1%	759	19	2.4%
FALL - SPORT	8	2	20%	175	8	4.4%	247	11	4.3%	54	0		489	21	4.1%
NAI	3	0		0	0		3	0		0	0		6		
MISSING / NOT RECORDED	4	3	42.9%	10	0		5	0		2	0		21	3	12.5%
TOTAL	165	17	9.3%	733	14	1.9%	580	19	3.2%	131	5	3.7%	1626	57	3.4%

TABLE 2(a)(xi) CAUSE SCALP INJURY / AGE / FRACTURE

ABRAS/CONTUS	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	2	0		10	0		6	0		0	0		19		0%
RTA - PEDESTRIAN	0	0		12	1	7.7%	35	3	7.9%	4	0		51	4	7.3%
RTA - CYCLIST	1	0		10	1	9.1%	17	0		10	0		40	1	2.4%
ASSAULT	4	0		13	0		32	0		14	0		63		
FALL - SCHOOL	1	0		5	0		60	0		7	0		74		
FALL - HOME	77	7	8.3%	259	1	0.4%	49	0		4	0		392	9	2.2%
FALL - SPORT	5	0		103	2	1.9%	138	5	3.5%	21	0		270	7	2.5%
NAI	2	1	33.3%	3	0		2	0		0	0		7	1	12.5%
MISSING / NOT RECORDED	3	0		9	0		10	0		1	0		23		
TOTAL	95	8	7.6%	424	5	1.2%	349	8	2.2%	61	0		939	22	2.3%

TABLE 2(a)(xii) CAUSE SCALP INJURY / AGE / FRACTURE

SUP LAC < 5

	1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
RTA - OCCUPANT	1	0		17	0		7	0		1	0		26		
RTA - PEDESTRIAN	0	0		7	0		17	0		5	0		29		
RTA - CYCLIST	0	0		3	0		10	0		2	0		16		
ASSAULT	2	0		56	1	1.8%	206	1	0.5%	47	0		320	2	0.6%
FALL - SCHOOL	0	0		33	1	2.9%	129	0		23	0		189	1	0.005
FALL - HOME	37	0		740	0		234	0		31	1	3.1%	1056	1	0.001
FALL - SPORT	3	0		229	1	0.4%	340	2	0.6%	53	1	1.9%	633	4	0.6%
NAI	0	0		0	0		2	0		2	1	33.3%	5	1	16.7%
MISSING / NOT RECORDED	2	0		23	0		25	0		8	0		59		
TOTAL	45	0	0%	1108	3	0.3%	970	3	0.3%	172	3	1.7%	2333	9	0.4%

TABLE 2(a)(xiii) CAUSE SCALP INJURY / AGE / FRACTURE

SUP LAC > 5	1 Year			1- 4 Years			5 - 11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	0	0		1	0		1	0		0	0		2	0	
RTA - PEDESTRIAN	0	0		0	0		4	0		1	0		5	0	
ASSAULT	0	0		0	0		2	0		0	0		2	0	
FALL - SCHOOL	0	0		2	0		2	0		1	0		5	0	
FALL - HOME	0	0		5	0		5	0		1	0		11	0	
FALL - SPORT	0	0		5	0		5	0		1	0		11	0	
MISSING / NOT RECORDED	0	0		2	0		1	0		1	0		4	0	
TOTAL	0	0		15	0		20	0		5	0		40	0	

GALEA < 5	1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - PEDESTRIAN	0	0		0	0		1	0		1	0		2		
ASSAULT	0	0		0	0		8	1	11.1%	3	1	25%	11	2	15.4%
FALL - SCHOOL	0	0		0	0		6	0		0	0		6		
FALL - HOME	0	0		11	0		5	0		0	0		16		
FALL - SPORT	0	0		5	0		16	1	5.9%	3	0		27	1	3.6%
TOTAL	0	0		16	0		36	2	5.3%	7	1	12.5%	62	3	4.6%

GALEA > 5	1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	0	0		1	0		0	0		0	0		1		
RTA - PEDESTRIAN	0	0		2	0		3	0		1	1	50%	6	1	14.3%
FALL - HOME	0	0		1	0		2	0		0	0		3	2	
FALL - SPORT	0	0		0	0		5	2	28.6%	0	0		5	2	28.6%
MISSING / NOT RECORDED	0	0		0	0		0	0		0	1	100%		1	100%
TOTAL	0	0		4	0		10	2	16.7%	1	2	66.7%	15	4	21.1%

The deep short lacerations of which there were 62 had three fractures only two due to assault and one, a fall at play/sport 4.8%

The deep long lacerations 15, revealed four fractures 21% resulting from falls and a pedestrian road traffic accident.

Superficial lacerations are confirmed as being associated with skull fracture, whatever the cause of injury in only 0.4% of cases. It forms a large group, in the majority of which requests for skull xrays could be reduced, whatever the cause with very low risk of missing a fracture. The suspicion of a foreign body may justify examination however.

2(a) Cause of Injury

Cause of Injury – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the cause of the injury, (chi-squared = 57.0, df=7, p<0.0005). In particular pedestrians involved in an RTA had substantially more fractures than expected.

The number of skull fractures by each cause and the expected number under a hypothesis of independence is shown in table.

TABLE 2(a)(SA)(i): Expected and Observed number of skull fractures by cause of injury (Under H0: skull fracture is independent of cause of injury)

			SKU LL #		Total
			No Fracture	Fracture	Total
CAUSE OF INJURY	RTA–Occupant	Count	90.0	2.0	92.0
		Expected Count	90.3	1.7	92.0
	RTA-Pedestrian	Count	149.0	12.0	161.0
		Expected Count	158.0	3.0	161.0
	RTA-Pedal cycle	Count	91.0	5.0	96.0
		Expected Count	94.2	1.8	96.0
	Assault	Count	541.0	7.0	548.0
		Expected Count	537.9	10.1	548.0
	Fall-School	Count	536.0	4.0	540.0
		Expected Count	530.1	9.9	540.0
	Fall-Home	Count	2628.0	31.0	2659.0
		Expected Count	2610.2	48.8	2659.0
	Fall-Sport/play	Count	1670.0	44.0	1714.0
		Expected Count	1682.6	31.4	1714.0
	Assault-NAI	Count	20.0	2.0	22.0
		Expected Count	21.6	0.4	22.0
Total		Count	5725.0	107.0	5832.0
		Expected Count	5725.0	107.0	5832.0

When children involved in an RTA (either as an occupant, pedestrian or cyclist) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 26.9, df=1, p<0.0005) and have an odds ratio of 3.5 (CI=(2.1,5.9)). Therefore a child presenting with a head injury after involvement in an RTA, is more than three(3) times more likely to result in a skull fracture than children whose injury has another cause, and the child pedestrian almost five(5) times as likely.

Children whose cause of injury was identified as child abuse (Assault-NAI) also resulted in a higher risk of skull fracture than children with fracture of other causes (chi-squared = 6.5, df=1, p=0.011), and have an odds ratio of 5.4 (CI= (1.3, 23.5)).

The odds ratios and 95% confidence intervals for each cause of injury are:

	Odds Ratio	**CI**
RTA–Occupant	1.19	(0.29, 4.91)
RTA-Pedestrian	4.73	(2.54, 8.81)
RTA-Pedal cycle	3.04	(1.21, 7.63)
Assault	0.67	(0.31, 1.45)
Fall-School	0.38	(0.14, 1.03)
Fall-Home	0.48	(0.32, 0.73)
Fall-Sport/play	1.70	(1.15, 2.50)
Assault-NAI	5.43	(1.25, 23.54)

2(a)(1) Cause of Injury by Sex - Univarate Analysis

The cause of injury was found to be dependent on the sex of the patient, (chi-squared = 71.445, df=7, p<0.0005), with males more likely to present after being involved in an RTA as a cyclist and after an assault.

TABLE 2(a)(SA)(ii): Expected and Observed number of patients by sex and cause of injury (Under H0: cause of injury is independent sex)

			SEX		Total
			Male	**Female**	
CAUSE OF INJURY	**RTA-Occupant**	Count	50.0	44.0	94.0
		Expected Count	62.1	31.9	94.0
	RTA-Pedestrian	Count	107.0	56.0	163.0
		Expected Count	107.7	55.3	163.0
	RTA-Cyclist	Count	74.0	23.0	97.0
		Expected Count	64.1	32.9	97.0
	Assault	Count	411.0	136.0	547.0
		Expected Count	361.5	185.5	547.0
	Fall-School	Count	379.0	165.0	544.0
		Expected Count	359.5	184.5	544.0
	Fall-Home	Count	1637.0	1032.0	2669.0
		Expected Count	1763.7	905.3	2669.0
	Fall-Sport/Play	Count	1194.0	525.0	1719.0
		Expected Count	1135.9	583.1	1719.0
	Assault-NAI	Count	17.0	5.0	22.0
		Expected Count	14.5	7.5	22.0
Total		Count	3869	1986	5855
		Expected Count	3869	1986	5855

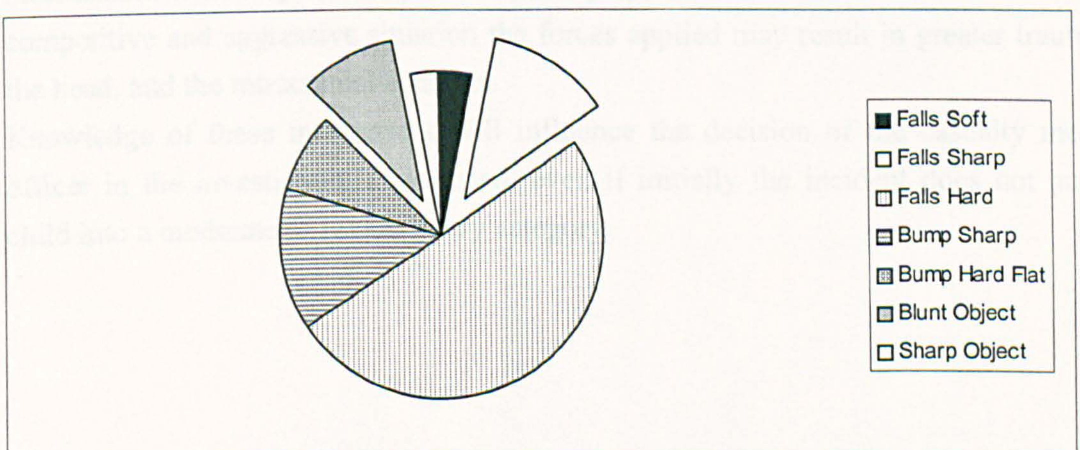
2.(b) Results – Mechanism of Injury

TABLE 2 (b)(i) MECHANISM OF INJURY

	n	%
Falls on to a soft surface	204	3.4
Falls on to a sharp surface	745	12.4
Falls on to a hard surface	2955	49.6
Bump on hard surface	846	14.2
Bump against a sharp object	457	7.7
Struck with a blunt object	580	9.7
Struck with a sharp object	170	2.9
All	5957	

The information collected relating to causes of injury, shows falls as the most common cause in childhood. The examination of the type of force applied to the head reveals an impact on a hard flat surface in 2955 Table 2.(b).(xix). Focal injury from a blow struck with a hard object and bumps, low velocity impact on hard surfaces account form the majority of the mechanisms.

FIGURE 2(b) (i) MECHANISM OF INJURY



Skull fracture is considered to reflect the magnitude of the force applied. In infancy the vulnerability of the skull is an additional factor. Information regarding the mechanism, combined with other factors, may have influenced the medical offers in their requests for skull xrays. Table 2(b) (ii)

TABLE: 2(b) (ii) MECHANISM/REQUEST FOR SKULL XRAYS

N		%
204	Falls soft surface	60
745	Falls sharp surface	52
2955	Falls hard surface	70
846	Bump sharp object	52
457	Bump hard flat object	69
580	Struck blunt object	62
170	Struck sharp object	35

The effects of blunt trauma and hard contact surfaces producing 93 of 102 fractures detected by a radiologist is clearly illustrated. However, large numbers of children sustained injury by this mechanism. Selection to increase the yield of fractures may in addition need to take account of the nature of the scalp injury, the age of the child, the cause of injury and the site of impact on the head. The history of loss of consciousness is already an indication for skull xray under the Guidelines.

The mechanism by which head injury is sustained will also be influenced by the age of the patient. An infant might be expected to be protected by a parent or carer from mechanisms such as a blow to the head as a consequence of a blunt impact. Supervision by adults may afford protection. The child whose mobility is increasing is subject to a wider range of environmental factors, such as traffic, and may be exposed to mechanisms which produce patterns of injury, and more serious effects. In a competitive and aggressive situation the forces applied may result in greater trauma to the head, and the intracranial contents.

Knowledge of these interactions will influence the decision of the casualty medical officer in the investigations requested, even if initially the incident does not put the child into a moderate or severe injury category.

TABLE 2(b) (iii) MECHANISM / AGE / FRACTURE

	1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	%	No #	#	%	No #	#	%	No #	#	%	No #	#	%
Fall - Soft	63	1	1.6%	81	0		43	1	2.3%	11	0		202	2	1.0%
Fall - Sharp	31	0		471	1	0.2%	204	3	1.4%	25	0		741	4	50%
Fall - Hard	226	18	7.4%	1338	24	1.8%	1096	24	2.1%	184	6	3.2%	2881	74	2.5%
Bump - Sharp	25	0		246	0		152	0		28	0		456	1	20%
Bump - Hard	70	3	4.1%	338	5	0.3%	339	5	1.5%	84	0		837	9	1.1%
Struck - Blunt	19	2	9.5%	113	2	0.9%	309	2	60.0%	117	5	4.1%	570	10	1.7%
Struck - Sharp	4	0		37	0		100	0		26	0		170	0	
Missing/Not Recorded	5	1	16.7%	13	0		11	0		3	1	25.0%	34	2	5.6%

Requests for skull xrays were examined in relation to the recorded mechanism, and a discretionary approach is apparent.

Fractures were detected most commonly where injury resulted from impacts combining blunt force, and hard surfaces. The majority of the injuries were however in these categories.

This knowledge is already resulting in selection for skull xrays. The low incidence of fracture in sharp injuries might allow a reduction in skull xray requests, providing there was no risk of penetration or foreign body.

TABLE 2 (b) (iv) FRACTURE FREQUENCY

Falls - soft surface	0.010
Falls - sharp surface	0.005
Falls hard flat surface	0.025
Bump sharp object	0.002
Bump hard object	0.010
Struck blunt object	0.017
Struck sharp object	0.00

Hard and blunt impact injuries carry the highest risk of skull fracture - and are the largest numerical groups of mechanism in all age groups.

2(a) Cause of Injury

Cause of Injury – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the cause of the injury, (chi-squared = 57.0, df=7, $p<0.0005$). In particular pedestrians involved in an RTA had substantially more fractures than expected.

The number of skull fractures by each cause and the expected number under a hypothesis of independence is shown in table.

TABLE 2(a)(SA)(i): Expected and Observed number of skull fractures by cause of injury (Under H0: skull fracture is independent of cause of injury)

			SKULL #		Total
			No Fracture	Fracture	Total
CAUSE OF INJURY	**RTA–Occupant**	Count	90.0	2.0	92.0
		Expected Count	90.3	1.7	92.0
	RTA-Pedestrian	Count	149.0	12.0	161.0
		Expected Count	158.0	3.0	161.0
	RTA-Pedal cycle	Count	91.0	5.0	96.0
		Expected Count	94.2	1.8	96.0
	Assault	Count	541.0	7.0	548.0
		Expected Count	537.9	10.1	548.0
	Fall-School	Count	536.0	4.0	540.0
		Expected Count	530.1	9.9	540.0
	Fall-Home	Count	2628.0	31.0	2659.0
		Expected Count	2610.2	48.8	2659.0
	Fall-Sport/play	Count	1670.0	44.0	1714.0
		Expected Count	1682.6	31.4	1714.0
	Assault-NAI	Count	20.0	2.0	22.0
		Expected Count	21.6	0.4	22.0
Total		Count	5725.0	107.0	5832.0
		Expected Count	5725.0	107.0	5832.0

When children involved in an RTA (either as an occupant, pedestrian or cyclist) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 26.9, df=1, p<0.0005) and have an odds ratio of 3.5 (CI=(2.1,5.9)). Therefore a child presenting with a head injury after involvement in an RTA, is more than three(3) times more likely to result in a skull fracture than children whose injury has another cause, and the child pedestrian almost five(5) times as likely.

Children whose cause of injury was identified as child abuse (Assault-NAI) also resulted in a higher risk of skull fracture than children with fracture of other causes (chi-squared = 6.5, df=1, p=0.011), and have an odds ratio of 5.4 (CI= (1.3, 23.5)).

The odds ratios and 95% confidence intervals for each cause of injury are:

	Odds Ratio	CI
RTA–Occupant	1.19	(0.29, 4.91)
RTA-Pedestrian	4.73	(2.54, 8.81)
RTA-Pedal cycle	3.04	(1.21, 7.63)
Assault	0.67	(0.31, 1.45)
Fall-School	0.38	(0.14, 1.03)
Fall-Home	0.48	(0.32, 0.73)
Fall-Sport/play	1.70	(1.15, 2.50)
Assault-NAI	5.43	(1.25, 23.54)

2(a)(1) Cause of Injury by Sex - Univarate Analysis

The cause of injury was found to be dependent on the sex of the patient, (chi-squared = 71.445, df=7, p<0.0005), with males more likely to present after being involved in an RTA as a cyclist and after an assault.

TABLE 2(a)(SA)(ii): Expected and Observed number of patients by sex and cause of injury (Under H0: cause of injury is independent sex)

			SEX		
			Male	**Female**	**TOTAL**
CAUSE OF INJURY	**RTA-Occupant**	Count	50.0	44.0	94.0
		Expected Count	62.1	31.9	94.0
	RTA-Pedestrian	Count	107.0	56.0	163.0
		Expected Count	107.7	55.3	163.0
	RTA-Cyclist	Count	74.0	23.0	97.0
		Expected Count	64.1	32.9	97.0
	Assault	Count	411.0	136.0	547.0
		Expected Count	361.5	185.5	547.0
	Fall-School	Count	379.0	165.0	544.0
		Expected Count	359.5	184.5	544.0
	Fall-Home	Count	1637.0	1032.0	2669.0
		Expected Count	1763.7	905.3	2669.0
	Fall-Sport/Play	Count	1194.0	525.0	1719.0
		Expected Count	1135.9	583.1	1719.0
	Assault-NAI	Count	17.0	5.0	22.0
		Expected Count	14.5	7.5	22.0
Total		Count	3869	1986	5855
		Expected Count	3869	1986	5855

2(b) Mechanism of Injury – Univarate Analysis

Risk of Fracture byMechanism of Injury

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the mechanism of the injury, (chi-squared = 30.8, df=6, $p<0.0005$). In particular children falling to or bumping against a sharp edge had substantially less fractures than expected.

The number of skull fractures by each cause and the expected number under a hypothesis of independent is shown in Table 2(b)(SA)(i).

TABLE 2(b)(SA)(i) Expected and Observed number of skull fractures by mechanism of injury (Under HO: skull fracture is independent of mechanism of injury).

			SKULL #		
			No	**Yes**	**Total**
MECHANISM OF INJURY	**Fall to soft surface**	Count	202.0	2.0	204
		Expected Count	200.3	3.7	204
	Fall to sharp edge	Count	739.0	4.0	743
		Expected Count	729.3	13.7	743
	Fall to hard surface	Count	2860.0	79.0	2939
		Expected Count	2885.0	54.0	2939
	Bump against sharp edge	Count	454.0	1.0	455
		Expected Count	446.6	8.4	455
	Bump against hard flat Surface/s	Count	830.0	11.0	841
		Expected Count	825.5	15.5	841
	Struck with blunt objects	Count	567.0	12.0	579
		Expected Count	568.4	10.6	579
	Struck with sharp object/s	Count	170.0	0.0	170
		Expected Count	166.9	3.1	170
Total		Count	5822.0	109.0	5931
		Expected Count	5822.0	109.0	5931

When children falling to or bumping against a sharp edge are considered as a group and compared directly to the other subjects, they demonstrate a significantly lower risk of skull fracture (chi-squared = 16.8, df=1, p<0.005) and have an odds ratio of 0.19 (CI=(0.08, 0.46)). Therefore a child presenting with a head injury after falling to or bumping against a sharp edge, is about a fifth as likely to result in a skull fracture than children whose injury resulted from another mechanism.

The odds ratios and 95% confidence intervals for each mechanism of injury are:

	Odds ratio	**CI**
Fall to soft surface	0.52	(0.13, 2.12)
Fall to sharp edge	0.26	(0.10, 0.71)
Fall to hard surface	2.73	(1.79, 4.17)
Bump against sharp edge	0.11	(0.02, 0.79)
Bump against hard flat surface/s	0.68	(0.36, 1.26)
Struck with blunt object/s	1.15	(0.63, 2.10)
Struck with sharp object/s	0.98	(0.98, 0.99)

2(b) Mechanism of Injury – Sex

The sex of patients presenting with a head injury was found to be dependent on the nature of the injury, (chi-squared = 39.08, df=6, p<0.0005). In particular males were more likely to present after being struck with a sharp or blunt object than females (chi-squared = 32.10, df=1, p<0.0005), and females more likely to present after a fall to a soft surface (chi-squared = 5.75, df=1,p=0.016).

The number of patients of each sex by mechanism of injury and the expected number under a hypothesis of independence is shown in Table 2(b)(SA)(ii).

TABLE 2(b)(SA)(ii) : Expected and Observed number of patients of each sex by mechanism of injury (Under HO : mechanism of injury is independent of sex).

			SEX		
			Male	**Female**	**Total**
MECHANISM OF INJURY	**Fall to soft surface**	Count	119.0	85.0	204
		Expected Count	134.9	69.1	204
	Fall to sharp edge	Count	493.0	252.0	745
		Expected Count	492.7	252.3	745
	Fall to hard surface	Count	1903.0	1252.0	2955
		Expected Count	1954.4	1000.6	2955
	Bump against sharp edge	Count	310.0	146.0	456
		Expected Count	301.6	154.4	456
	Bump against hard flat surface/s	Count	549.0	296.0	845
		Expected Count	558.9	286.1	854
	Struck with blunt objects	Count	434.0	146.0	580
		Expected Count	383.6	196.4	580
	Struck with sharp object/s	Count	130.0	39.0	169
		Expected Count	111.8	57.2	169
Total		Count	3938.0	2016.0	5954
		Expected Count	3938.0	2016.0	5954

3.(a) Results – Loss of Consciousness/Coma

TABLE 3 (i) HISTORY OF LOSS OF CONSCIOUSNESS

N	5993	%
None	5581	93.0
Less than 5 minutes fully recovered	335	6.0
5-30 minutes fully recovered	35	0.6
30-60 minutes impairment	3	0.1
60 minutes or longer not improving	25	0.4
N/R	14	

In the Accident and Emergency Department there is a very small number of children within a very large group of patients, whose clinical assessment will require a Coma Scoring System. Recognition of that minority and the prompt commencement of observations has the primary aim of assessing not only the level of consciousness, but any deterioration which subsequently occurs.

Any system requires standardisation of measurement, compliance and observer agreement. The primary requirement of the Accident & Emergency Department is for a highly sensitive indicator of management, investigation and surgical intervention. Measurements of established coma responses are of more relevance to management within the Neurosurgical Intensive Care Unit. In this context they may be used to predict outcome and allow comparison of treatment protocols.

In 1984, in a mixed adult and paediatric analysis conducted by the Author of this Thesis, the Glasgow Coma Scale was used for adults and the Adelaide Paediatric Scale for children under 15 years. The results for the Adelaide Scale were unsatisfactory. They were scored inappropriately for age, by the casualty medical officers.

In the exclusively Paediatric study reported herein, the Adelaide Scale was used in the Accident & Emergency Department as an alternative to the Glasgow Coma Scale, and was incorporated in the data collection document. Again, the Adelaide Scale caused confusion in the small numbers who were actually showing impairment of consciousness. It was noted that having initiated the Paediatric Scale in the Accident & Emergency Department, following admission, staff reverted to the Glasgow Coma Scale. This was attributed to a familiarity with the latter. It was perhaps surprising that in the exclusively paediatric hospitals the children'' scale was not in use normally? If Paediatricians had not been persuaded to evaluate the Adelaide Scale, clearly little experience or expertise will have been built up. The use in Accident & Emergency Departments would have little value or reliability. The number of children comatose, as this Study illustrates, is small. Opportunity for Accident & Emergency medical Officers in a six month appointment at SHO grade, the commonest doctor group, would be extremely limited, in familiarising themselves with the Adelaide Scale, which is more appropriate after admission with paediatric trained staff.

3.(b) THE PINDEFIELDS COMA SCORE AND CHART SYSTEM

The wide acceptance of the Glasgow Coma Scale has not prevented others actively involved in the clinical care of head injured patients suggesting that it is not sufficiently sensitive to detect minor deterioration in the conscious level. In those who are believed initially to be mildly injured, and whose deterioration is a manifestation of an expanding intracranial mass, a more sensitive system is said to be necessary. A Coma Scale was developed at Pinderfields Hospital, Wakefield(Marsden, Staniland and Price 1984) and is currently used in that Unit following evaluation in other Yorkshire Units. It was tested for acceptability to nursing staff and others involved in the observation of head injured patients.

The scale originally tested had a 50 point scale. This was later reduced to a 34 point scale incorporating seven functional groups. A fall of four points on this scale is equated with a fall of one point in the Glasgow Coma Scale and therefore alerts observers earlier to deterioration. It is used in children but makes no concession to age.

Nursing Staff has tested accuracy and sensitivity in the Yorkshire Region. It has found acceptability in Accident & Emergency Units. It is claimed, despite its comprehensive format, that only 8-15 seconds are required for each sub-section when used by experienced staff.

An inspection of Casualty Record Cards can be a depressing exercise, particularly when the quality of information is examined critically. This is important in the head injured patient, where the initial observations form the foundation on which progress is monitored, and on which subsequent decisions may rely. Gorman(1985) commented that 47% of records which he examined in 12,000 patients provided inadequate neurological information.

In the Accident & Emergency Department the inexperienced doctor will only record, at best, that information which is perceived as relevant to their particular examination, unaware of the value of certain items of information that may not be recorded. History and clinical features, which are relevant to management, form a permanent record of events. These may be required subsequently both for clinical and for medicolegal procedures. Adequate notes benefit the physician caring for the patient, and others to whom the patient may be transferred. Continuous monitoring must be achieved.

The assistance provided to the clinician by a structured format ensures more complete and detailed information as the chart acts as an Aide Memoire.

THE PINDERFIELDS COMA SCALE AND CHART

TABLE: 3(b)(i) THE PINDERFIELDS COMA SCORE AND CHART SYSTEM

Respiration
4. Normal spontaneous and regular.
3. Hyperventilation or shallow but regular.
2. Cheyne Stokes (periodic) or variably irregular (ataxic)
1. Inadequate spontaneous ventilation requiring support.

Alertness (A measure of rousability shown by flinching facial, respiratory or limb movement)
5. Fully alert, immediately responds to name.
4. Drowsy but easily roused by speech or gentle shaking.
3. Rousable by shout or firm shaking.
2. Unrousable by shout but rousable by superficial pain.
1. Rousable by deep pain only.
0. Unrousable.

Eyes
5. Spontaneous eye opening and looks at people.
4. Spontaneous eye opening but looking at people.
3. Opens eyes in response to normal speech.
2. Opens eyes to shout.
1. Not looking at people and only opens eyes to pain.
0. No eye opening even to pain.

Obedience
4. Obeys complex commands.
3. Immediate appropriate response to simple commands.
2. Doubtful or inappropriate response to simplest commands.
1. No obedience to simplest commands.

Orientation
4. Fully orientated in all respects with good memory of todays events.
3. Aware of who he is and where he is but has poor memory of today's events.
2. Moderately confused and uncertain as to where he is.
1. Very confused and even unsure of who he is.
0. Too drowsy to assess.

Speech
5. Spontaneous conversation - answering all questions with quite elaborate answers such as "I am all right apart from the headache".
2. Answers simple questions with monosyllabic and often inappropriate words.
1. Incomprehensible wounds (in response to speech or pain).
0 No sounds in response to speech or pain.

Best Motor Response (B.M.R.) (Stronger side)
7. Brisk movement with full power of less conscious, localising immediate purposeful and useful responses (against resistance to pain)
6. Impaired postural maintenance, or some weakness, or if less conscious, sluggish localising purposeful response to pain with effective withdrawal.
5. Non-localising effective withdrawal from pain.
4. Abnormal flexion with non-purposeful response to pain.
3. Brisk extensor posturing without either purpose or effective withdrawal.
2. Sluggish extensor posturing without effective withdrawal.
1. Only a flicker to pain.
0. None.

Head Injury Assessment Chart

No.HO128/12, Mastrom Ltd., Alrewas Burton-on-Trent, Staffs

name M ☐ F ☐ | injury date time | A/E number | hospital name

age | D.O.B | admission date time | hosp no. | ward | consultant

injury

R.T.A.: pedestrian ☐ car seat belt ☐ car no S B ☐ motor bike ☐ push bike ☐

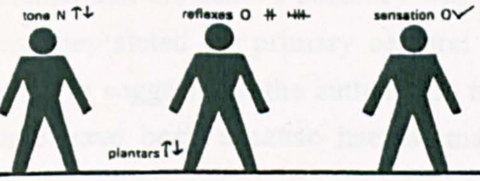
other: work/school ☐ home ☐ sport/play ☐ assault ☐

If fall, height in ft. ☐☐

eyes	movement	speech	before arrival
open spont ☐	to command ☐	sensible ☐	fits ☐
open to shout ☐	withdrawing to pain ☐	confused ☐	headache ☐
open to pain ☐	abnormal ☐	incomprehensible ☐	vomiting ☐
not open ☐	absent ☐	absent ☐	dizziness ☐

post traumatic amnesia ☐☐ mins

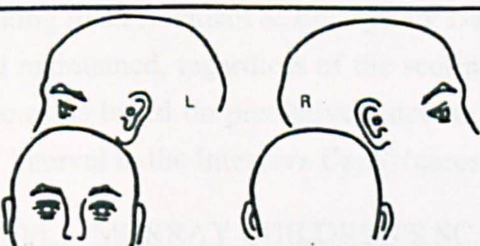
examination tone N ↑↓ reflexes O ‡ ‡‡ sensation O✓ plantars ↑↓



eye movements — normal ☐ / static — / roving ☐; dev L ☐ / non deviating ☐ / dev R ☐; conjugate ☐ / dysconjugate ☐

impact site

no impact ☐
cut
bruise ////
blood leak ●
C S F leak ↗



skull X ray

no fracture
fracture /
depressed fracture

cervical spine no X ray ☐ X ray ☐ no fracture ☐ fracture ☐

midliner yes no — from R ☐ no shift ☐ from L ☐

alcohol? yes no ☐

other drugs? yes no ☐

other illness? yes no ☐

other injuries no ☐ yes state est blood loss (litres)

yes	state	est blood loss (litres)
☐	chest	☐
☐	belly	☐
☐	pelvis	☐
☐	arms	☐
☐	legs	☐

time	hour / mins				
respiration	4				
alertness	5				
eyes	5				
obedience	4				
orientation	4				
speech	5				
better motor-B	7				
total	34				
poorer motor-P	7				
B - P	7				
graph of conscious level (maximum 34)	34 / 30 / 25 / 20 / 15 / 10 / 5				
right pupil size (mm)	9 / 5 / 1				
right pupil reaction	B / F				
left pupil size (mm)	9 / 5 / 1				
left pupil reaction	B / F				
seizures	L / R				
C S F leak					
temperature					
respiration					
abdominal girth					
B P and pulse	150 / 140 / 130 / 120 / 110 / 100 / 90 / 80				

Children in deep coma are by definition severely brain injured. Coma scales are therefore more appropriate in Neurosurgical and Intensive Care Units. In 1984 Morray et al reported a Coma Scale which was applicable to such patients and which measured both cortical and brain stem function. It was applied retrospectively to the Neurological status on admission in a variety of cerebral insults and used to devise an outcome score. It was claimed to be sufficiently sensitive to monitor the course of the disease process subsequently, and to identify deterioration.

The cortical scores ranged from 0 - 6 and the brain stem scores from 0-3 giving a maximum of 9. It was suggested that the combination of cortical and brain stem function was as good as either alone. The cortical score was perhaps a better measure of the quality of outcome.

The author claims that the Scale's accuracy was best in patients with hypoxia and head trauma, where they stated the primary cerebral insult determined the outcome. This statement seems to suggest that the authors did not take account of mass lesions. This may of course have been because haematoma had already been excluded by CT scanning?

The Morray Scale has no relevance or application to the immediate assessment and decision making in an Accident & Emergency Department. Intense resuscitation will be initiated and maintained, regardless of the scoring system, particularly in the paediatric patient. Decisions based on predictive outcome would be deferred, and made after an appropriate interval in the Intensive Care/Neurosurgical Unit.

TABLE: 3(b)(ii) MORRAY CHILDREN'S SCALE

Cortical (= Motor)	6
Spontaneous	5
Voice evoked	4
Localised	3
Withdrawal	2
Decortical	1
Decerebrate	0
Flaccid	
Brain Stem	
Intact	3
Depressed	2
Absent-breathing	1

CHILDRENS COMA SCORE - RAIMONDI

Raimondi in 1984 reviewed 462 children with closed head injury aged 1-36 months. A coma Scoring System was devised to correlate with outcome. He rejected the Glasgow Coma Scale because it was based on varying degrees of higher integrated function. When compared with the Glasgow Coma Scale a normal infant could only score 4 on the motor scale and 3 on the verbal scale. Raimondi's resulting scale had a maximum score of 11, ocular 4, verbal 3, and motor 4. It was used primarily to compare coma outcome with the child's condition on admission. Its value, if any in assessing progress and deterioration, which might be applicable in the Accident & Emergency situation was not reported by the originator.

The components of the system were however discussed. Of the 3 components of the scale ocular, motor and verbal, the motor score most constantly reflected neurological damage. Hemiparesis was a predictor of poor outcome, as was raised intracranial pressure, diastolic fractures and split sutures. Linear fractures did not correlate closely with poor outcome. Children under the age of one had a worse outcome related to the grade of coma, except paradoxically when this was most severe. In this group subdural haemorrhage was common.

TABLE: 3(b)(iii) CHILDREN'S COMA SCORE - Raimondi

OCULAR RESPONSE	Pursuit	4
	Extra Ocular Muscles, (EOM) Intact, reactive Pupils	3
	Fixed Pupils or EOM Impaired	2
	Fixed Pupils and EOM Paralysed	1
VERBAL RESPONSE	Cries	3
	Spontaneous Respiration	2
	Apnoeic	1
MOTOR RESPONSE	Flexes and Extends	4
	Withdraws from Painful Stimuli	3
	Hypertonic	2
	Flaccid	1
TOTAL COMA SCORE		3-11

Hahn et al(1988) from Chicago Children's Hospital analysed data on 318 infants and children below the age of 36 months. Outcome was correlated with impairment of consciousness. The scale detected vital responses and employed a six point scale which was inappropriate below the age of six months. It was also called the Children's Coma Scale and was claimed

to have good correlation with outcome.

TABLE 3(b)(iv) CHILDREN'S COMA SCALE (HAHN) EYES OPENING

4	Spontaneous
3	Reaction to speech
2	Reaction to pain
1	No response

BEST MOTOR RESPONSE

6	Spontaneous (obeys verbal command)
5	Localises pain
4	Withdraws to pain
3	Abnormal flexion in response to pain (Decorticate posture)
2	Abnormal extension in response to pain (Decerebrate posture)
1	No response

BEST VERBAL RESPONSE

GCS Subscore			
Orientated	5	Smiles, follows objects	
Confused	4	Crying consolable	Interacts
Inappropriate	3	Consolable at times	Moaning
Incomprehensible words	2	Inconsolable	Irritable
Sounds			
No response	1	No response	No response

The child with multiple injuries complicating head trauma is particularly difficult to assess. The Tepas Score,(Tepas, Discala and Ramensofsky(1990) has been devised for such patients as with all assessments of head trauma resuscitation should precede scoring.

Paediatric Trauma Score - Tepas

PTS	+ 2	+ 1	- 1
Weight	>44 lbs (> 20 kg)	22 – 44 lbs (10 – 20 kg)	< 22 lbs (< 10 kg)
Airway	Normal	Oral or nasal airway	I ntubated tracheostomy invasive
Blood Pressure	> 90 mmHg	50 – 90 mmHg	< 50 mmHg
Level of Consciousness	Completely awake	Obtunded or any LOC*	Comatose
Wound Open	None	Minor	Major or penetrating
Fractures	None	Minor	Open or multiple fractures
TOTALS:			

* Loss of consciousness

3. Loss of Consciousness/Coma – Univarate Analysis

Risk of Fracture by Unconsciousnss

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on whether a history of unconsciousness or amnesia was noted, (chi-squared = 199.5, df=4, p<0.0005). All categories of unconsciousness or amnesia had substantially more fractures than expected.

The number of skull fractures by length of unconsciousness and the expected number under a hypothesis of independence is shown in Table 3(SA)(i).

TABLE 3(SA)(i): Expected and Observed number of skull fractures by history of unconsciousness/amnesia (Under H0: skull fracture is independent of a history of unconsciousness/amnesia)

			SKU LL #		
			No	**Yes**	**Total**
UNCONSCIOUSNESS-?	**None**	Count	5477.0	81.0	5558
		Expected Count	5456.2	101.8	5558
	Less than 5 minutes with <u>full</u> recovery of consciousness	Count	317.0	16.0	333
		Expected Count	326.9	6.1	333
	Five to 30 minutes with <u>full</u> recovery of consciousness	Count	32.0	3.0	35
		Expected Count	34.4	0.6	35
	30 to 60 minutes with <u>full</u> recovery	Count	3.0	0.0	3
		Expected Count	2.9	0.1	3
	Greater than 1 hour and/or still disorientated or worse	Count	15.0	9.0	24
		Expected Count	23.6	0.4	24
Total		Count	5844.0	109.0	5953
		Expected Count	5844.0	109.0	5953

When all children with a history of unconsciousness or amnesia (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 65.1, df=1, p<0.0005) and have an odds ratio of 5.2 (CI=(3.3,8.0)). Therefore a child presenting with any history of unconsciousness or amnesia is about five (5) times more likely to result in a skull fracture than children with no history of unconsciousness or amnesia.

The odds ratios and 95% confidence intervals for each period of unconsciousness are:

	Odds Ratio	**CI**
None	0.19	(0.13, 0.30)
Less than 5 minutes with <u>full</u> recovery of consciousness	3.0	(1.74, 5.16)
5 – 30 minutes with <u>full</u> recovery of consciousness	5.14	(1.55, 17.05)
30 – 60 minutes with <u>full</u> recovery	0.98	(0.98, 0.99)
Greater than 1 hour and/or still disorientated or worse	34.97	(14.95, 81.80)

The probability of the child in the A&E Department in coma is thirty five (35) times that of a child who has no history of loss of consciousness.

<u>Unconsciousness/Sex</u>

The number of patients of each sex by unconsciousness/amnesia and the expected number under a hypothesis of independence is shown in Table 3(SA)(ii).

TABLE 3(SA)(ii) : Expected and Observed number of patients of each sex by history of unconsciousness/amnesia (Under HO: history of unconsciousness/amnesia is independent of sex).

			SEX		
			Male	**Female**	**Total**
UNCONSCIOUSNESS-?	**None**	Count	3669.0	1909.0	5578
		Expected Count	3684.1	1893.9	5578
	Less than 5 minutes with full recovery of consciousness	Count	230.0	105.0	335
		Expected Count	221.3	113.7	335
	Five to 30 minutes with full recovery of consciousness	Count	25.0	10.0	35
		Expected Count	23.1	11.9	35
	30 to 60 minutes with full recovery	Count	2.0	1.0	3
		Expected Count	2.0	1.0	3
	Greater than 1 hour and/or still disorientated or worse	Count	21.0	4.0	25
		Expected Count	16.5	8.5	25
Total		Count	3947.0	2029.0	5976
		Expected Count	3947.0	2029.0	5976

4.(a) The Results - Scalp Injury

A scalp injury may be the first manifestation of head trauma. It may be a marker of underlying injury or potential complication. The majority of wounds in childhood, may be no more than minor scalp cuts and are usually unassociated with fracture of the skull or brain injury. This Study defined wounds, and related them to more serious injury. It examined the value of the scalp feature to decisions made by the Casualty Medical Officer

TABLE 4 (a)(i) THE NATURE OF SCALP INJURY

	N	%
No external injury	868	15.0
Swelling only	1683	28.0
Abrasions/contusions	961	16.0
Sup laceration < 5 cm	2342	39.0
Sup. Lacerations > 5 cm	40	0.7
Galeal lacerations <5cm	65	1.1
Galeal lacerations> 5 cm	19	0.3
N/R	16	-

No External Scalp Feature

In 15% of the children studied, evidence of external injury to the head or scalp was not found, or not recorded. This may represent inadequate examination, concealment by hair, very recent injury before swelling became apparent or perhaps that no injury to the head had in fact occurred. Nevertheless skull xrays were taken in this group and in some pathology was detected.

Swelling of the Scalp

This proved to be the second most common manifestation of scalp trauma occurring in 28 % of the children.

Abrasions

These are associated with swelling and accounted for 16%.

Scalp Lacerations

Minor scalp lacerations, superficial and less than 5 cm in length proved, as anticipated, from previous experience, the most common injury - almost 40% of the children. The superficial long laceration greater than 5 cm in length was uncommon.

Galeal Lacerations

Lacerations involving the scalp aponeurosis, were found in only 84 children - less than 2%. In only 19 did the length exceed 5 cm.

The nature of the scalp wound was related to the age of the child.

TABLE 4(a (ii) SCALP INJURY/AGE

N	< 1 Year 468	1-4 Years 2364	5- 11Years 2289	12-14 Years 490
	%	%	%	%
No external injury detected	29.0	13.0	12.5	21.0
Swelling only	39.0	28.0	26.0	28.0
Abrasion/Contusion	22.0	16.0	16.0	12.0
Sup lac<5 cm	10.0	42.0	42.5	36.0
Sup lac > 5 cm	-	0.6	0.9	1.0
Galeal lac < 5 cm	-	0.6	1.7	1.6
Galeal lac > 5 cm	-	0.2	0.5	0.6

Scalp Feature - Decision to Request Xrays

The decision to request xrays is taken relatively early following examination of the patient by the Casualty Medical Officer. It cannot be assumed, that the scalp injury was the only factor influencing the request; symptoms and other physical signs may have been present. The risk of a concealed foreign body might have indicated a requirement for xray in those with lacerations or suspicion of penetrating injury.

Xrays may be the only method of confirming these injuries in a child genuinely unaware of how the trauma was inflicted.

TABLE 4(a) (iii) SCALP INJURY/X-RAY REQUEST

	No Xray	Xray
No external injury	304 35%	564 65%
Swelling	323 19.2%	1258 80.7%
Abrasion/Contusion	268 27.9%	693 72%
Sup. Lac. < 5cm	1230 52.5%	1112 47.5%
Sup Lac > 5 cm	14 35%	26 65%
Galeal Lac. < 5cm	19 29.2%	46 70.8%
Galeal Lac > 5 cm	1 5.3%	18 94.7%

TABLE 4 (a)(iv) SCALP FEATURE./SKULL FRACTURES/ALL AGES

	%
No external scalp injury	0.8
Scalp swelling	3.4
Abrasion/contusion	2.3
Superficial laceration < 5 cms	0.4
Superficial lacerations > 5 cms	0.0
Short galeal lacerations < 5 cms	4.6
Long galeal lacerations > 5 cms	21.0

One hundred and two skull fractures were diagnosed by a Radiologist. The features at different ages are shown in Table 4 (v).

TABLE 4(a)(v) SCALP FEATURE / AGE / FRACTURE

	1 Year			1 - 4 Years			5 - 11 Years			12 - 14 Years			All Ages		
NO EXTERNAL INJURY	136	0		330	5	1.5%	286	1	0.3%	100	1	1.0%	861	7	0.8%
SWELLING ONLY	165	17	9.3%	733	14	1.9%	580	19	3.2%	131	5	3.7%	1626	57	3.4%
ABRASIONS/CONTUSIONS	95	8	7.8%	424	5	1.2%	349	8	2.2%	61	0		939	22	2.3%
SUP LAC < 5	45	0		1108	3	0.3%	970	3	0.3%	172	3	1.7%	2333	9	0.4%
SUP LAC > 5	0	0		15	0		20	0		5	0		40	0	
GALEA < 5	0	0		16	0		36	2	5.3%	7	1	12.5%	62	3	4.6%
GALEA > 5	0	0		4	0		10	2	16.7%	1	2	66.7%	15	4	21.1%
MISSING/NOT RECORDED	2	0		7	0		3	0		1	0		15	0	

Fractures Under One Year

When the features of scalp injury under one year are examined in infants, it was noted that 17 fractures, 9.3% occurred in the presence of swelling. In this age group no fracture was detected where scalp trauma was not recorded.

Lacerations as one might expect were infrequent at this age, and there were no fractures associated.

Preschool Group

In preschool children swelling was associated with 14 fractures; 1.9%, abrasions and contusions; five fractures 1.2%. Five fractures were found in the absence of scalp injury 1.5%.

Lacerations formed the largest group of scalp injuries, 1,108, but the fracture incidence was only 0.3%, and was present in short superficial lacerations.

No fractures were detected in 23 preschool children who had galeal lacerations.

School age group 5-11 years

35 fractures occurred in this group of children. The largest number occurred in association with swelling - 19 fractures, but the highest incidence was in children with lacerations through the galea. TABLE 4(a) (v)

Older Children 12-14 years

Swelling was associated with five fractures, 3.7% and short superficial lacerations were associated with three fractures - 1.7%.

The infrequent long superficial lacerations revealed no fracture. One possible explanation may be that these were incised wounds.

All Ages

Deep lacerations 77, penetrating the galea, were not seen in infants. The numbers increase with age. The incidence of skull fracture showed a dramatic rise 5.3% and 16.7% in 5 - 11 year olds, 12.5% and 66% in 12-14 years olds. 4.6% were associated with short galeal lacerations and 21% In long galeal lacerations in all ages and this was statistically significant.

4.(a) Scalp Injury/Skull Fracture – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the nature of the injury, (chi-squared = 106.7, df=6, p<0.0005). In particular children with a superficial laceration (of any size) had a substantially lower number of fractures than expected and children with a laceration through galea (of any size) resulted in a substantially higher number of fractures than expected.

TABLE 4(a)(SA)(i) : Expected and Observed number of skull fractures by nature of injury (Under H0: skull fracture is independent of a nature of injury)

			SKULL #		
			No	Yes	Total
SCALP INJURY NATURE	**No external injury**	Count	855.0	8.0	863
		Expected Count	846.9	16.1	863
	Swelling only	Count	1610.0	61.0	1671
		Expected Count	1639.8	31.2	1671
	Abrasions/contusions only	Count	930.0	26.0	956
		Expected Count	938.2	17.8	956
	Superficial laceration <5 cm long	Count	2329.0	9.0	2338
		Expected Count	2294.4	43.6	2338
	Superficial laceration >5 cm long	Count	40.0	0.0	40
		Expected Count	39.3	0.7	40
	Laceration through galea <5 cm long	Count	62.0	3.0	65
		Expected Count	63.8	1.2	65
	Laceration through galea >5 cm long	Count	15.0	4.0	19
		Expected Count	18.6	0.4	19
Total		Count	5841.0	111.0	5952
		Expected Count	5841.0	111.0	5952

When all children with a laceration through galea (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 19.5, df=1, p<0.0005) and have an odds ratio of 5.0 (CI=(2.3,11.2)). Therefore a child with a laceration through galea is about five times more likely to result in a skull fracture than children with other injuries.

When all children with a superficial laceration (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly lower risk of skull fracture (chi-squared = 47.8, df=1, p<0.0005) and have an odds ratio of 0.13 (CI=(0.07,0.26)). Therefore a child with a superficial laceration is less than a fifth as likely to result in a skull fracture as children with other injuries.

The odds ratios and 95% confidence intervals for each nature of injury are:

	Odds ratio	**CI**
No external injury:	0.45	(0.22, 0.93)
Swelling only:	3.21	(2.20, 4.68)
Abrasions/contusions only:	1.62	(1.04, 2.52)
Superficial laceration <5 cm long:	0.13	(0.07, 0.26)
Superficial laceration >5 cm long:	0.98	(0.98, 0.99)
Laceration through galea <5 cm long:	2.59	(0.80, 8.38)
Laceration through galea >5 cm long:	14.52	(4.74, 44.47)

4. Scalp Injury/Sex – Univarate Analysis

The sex of patients presenting with a head injury was found to be dependent on the nature of the scalp injury, (chi-squared = 74.02, df = 6, p<-.0005). In particular females were more likely to present with a superficial laceration (chi-squared = 54.07, df=1, p<0.005).

The number of patients of each sex by nature of injury and the expected number under a hypothesis of independence is shown in Table 4(a)(SA)(ii).

TABLE 4(a)(SA)(ii) : Expected and Observed number of patients of each sex by nature of injury (Under H0: nature of injury is independent of sex)

			SEX		
			Male	**Female**	**Total**
SCALP INJURY/NATURE	**No external injury**	Count	517.0	351.0	868
		Expected Count	573.5	294.5	868
	Swelling only	Count	1020.0	663.0	1683
		Expected Count	1112.0	571.0	1683
	Abrasions/contusions only	Count	647.0	312.0	959
		Expected Count	633.7	325.3	959
	Superficial laceration <5 cm long	Count	1678.0	663.0	2341
		Expected Count	1546.8	794.2	2341
	Superficial laceration >5 cm long	Count	27.0	13.0	40
		Expected Count	26.4	13.6	40
	Laceration through galea <5 cm long	Count	44.0	21.0	65
		Expected Count	42.9	22.1	65
	Laceration through galea >5 cm long	Count	15.0	4.0	19
		Expected Count	12.6	6.4	19
Total		Count	3948.0	2027.0	5975
		Expected Count	3948.0	2027.0	5975

Sex/Age

The sex of patients presenting with a head injury was found to be dependent on the age of the patients, (chi-squared = 62.11, df = 3, p<0.0005). In particular younger females (under five years of age) were more likely to present than younger males (chi-squared = 54.25, df = 1, p<0.0005), with more older males (five years and older) more likely to present than older females.

Nature of Scalp Injury in Children under One year

The nature of injuries seen in children under one year old was significantly different to that in older children (chi-squared = 204.7, df = 6, p<0.0005). As can be seen in figures below, most Infants presented with either swelling only (38%) or no external injury(28%), whereas older children presented more often with a superficial laceration less than 5 cm long (42%), with 27% presenting with swelling only.

Figure 4(a)(SA)(i) : Nature of Injury (percent of total cases) in children under 1 year old.

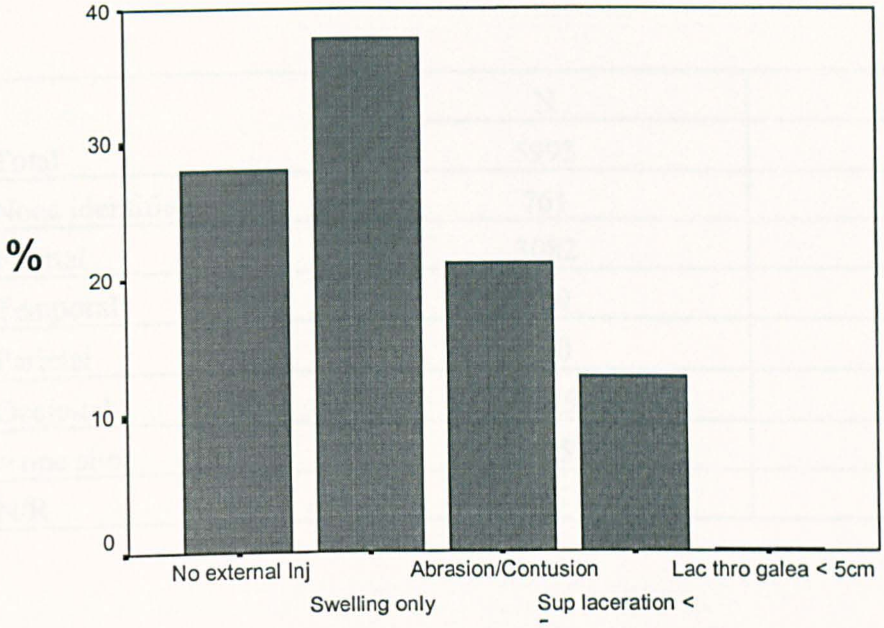
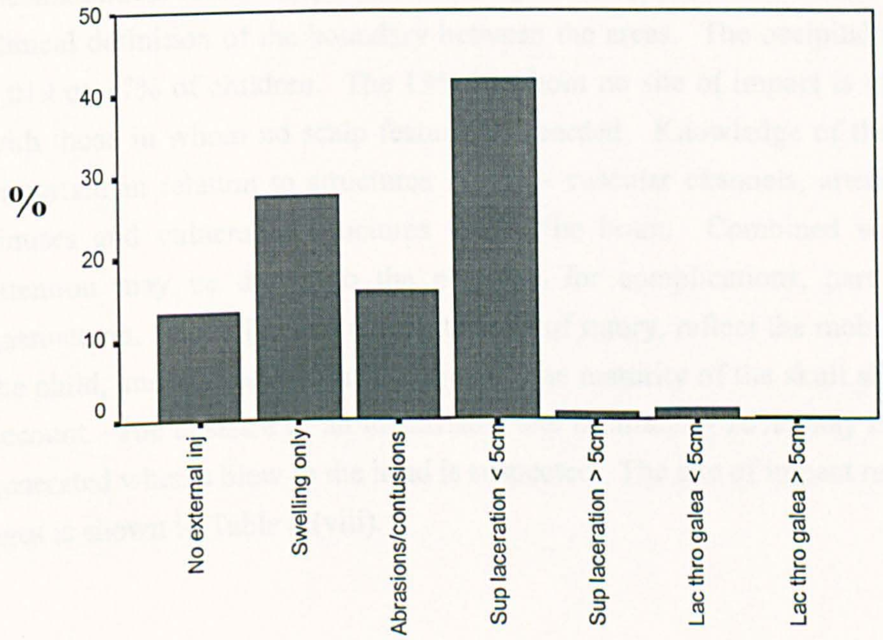


Figure 4(a)(SA)(ii): Nature of Scalp Injury (percent of total cases) in children of 1 year and older



4.(b) Results - Site of Injury to the Head

TABLE 4(b) (i) THE SITE OF INJURY TO THE HEAD

	N	%
Total	5993	
None identified	761	13
Frontal	3082	51
Temporal	280	5
Parietal	700	12
Occipital	1019	17
> one site	135	2
N/R	16	-

When recorded location of impact is examined, frontal impact is confirmed in 3,082, more than 50% of the patients. Parietal impact recorded 700 or 12% and temporal least often, 280 or 5%. The temperoparietal sites can reasonably be combined because of the anatomical relationships to the meningeal artery and its branches. There is also poor clinical definition of the boundary between the areas. The occipital site is involved in 1,019 or 17% of children. The 13% in whom no site of impact is identified coincides with those in whom no scalp feature is recorded. Knowledge of the site of trauma is important in relation to structures at risk - vascular channels, arterial and venous air sinuses and vulnerable structures within the brain. Combined with other features, attention may be drawn to the potential for complications, particularly extradural haematoma. The effects of age on the site of injury, reflect the mobility and activity of the child, and the mechanism of injury. The maturity of the skull should be taken into account. The absence of an identifiable site in infants - 26%, may indicate the anxiety generated when a blow to the head is suspected. The site of impact recorded at different ages is shown in Table 4 (viii).

TABLE 4(b)(ii) SITE OF INJURY/AGE OF CHILD

	<1 year 468	1-4 years 2664	5-11 years 2289	12-14 years 481
	%	%	%	%
None	26.0	11.0	11.0	16.0
Frontal	49.0	60.0	45.0	39.0
Temporal	3.0	3.5	6.0	7.0
Parietal	12.0	8.0	15.0	16.0
Occipital	7.0	15.0	21.0	19.0
> 1 Site	2.0	2.0	2.5	3.0

The age and activities of the victim in part influence the site of injury. The largest number occur at the frontal site in the toddlers, followed by infants and the younger school children.

TABLE 4 (b)(iii) SITE OF INJURY/X RAY REQUEST

Site	N	% Site	% X-rays
None rec.	761	13.0	63.0
Frontal	3081	51.0	62.0
Temporal	280	5.0	71.0
Parietal	700	12.0	63.0
Occipital	1019	17.0	64.0
> One site	135	2.3	85.0

The request for skull xrays where no site of injury was noted as of interest, as was the detection of three fractures. The Casualty Officer recorded 147 fractures; only 102 fractures were confirmed by the Radiologist. When examined in relation to site, it was the frontal area that produced 21 false positives, the parietal eight and the occipital seven false positives. When the incidence is examined based on the Radiologist's report the frequency of fracture by site is shown.

TABLE 4 (b)(iv) SITE OF INJURY FRACTURE INTERPRETATION

Site	N	# Rad.	#Co.
		102	147
None recorded	761	3	9
Frontal	3081	19	40
Temporal	280	6	6
Parietal	700	33	41
Occipital	1019	29	36
> one site	135	12	15

Of 102 fractures diagnosed by the Radiologist the location is shown in Table 4 (x). When the temperoparietal sites are combined 39 fractures were detected. When related to age the vulnerability of the infant is clearly shown. The Casualty officers interpretation or misinterpretation of skull films depends on a number of factors. It was postulated that the anatomical features of the child's skull might lead to diagnostic error. The locations were examined. In fact the majority of false positive readings were frontal.. Suture lines and vascular markings did not seem to affect the diagnosis of a fracture. There were 10 errors where no site of injury was identified.

TABLE 4 (b)(v) SITE OF INJURY/AGE/FRACTURE

The 25 skull fractures, which occurred in the children under 1 year, were distributed by site as follows

	%
Frontal	1.7
Temporal	6.3
Parietal	22.4
Occipital	15.2
At more than one site	25.0

In the 1-4 year olds, 2 fractures occurred in the absence of a recorded site of injury.

	%
Frontal	0.4
Temporal	1.1
Parietal	3.5
Occipital	1.7
More than one site	0.0

In the 5-11 year olds the site was identified as:

	%
Frontal	0.5
Temporal	1.5
Parietal	2.3
Occipital	2.7
More than one site	12.0

In the 12-14 year olds one fracture occurred in the absence of a recorded site

	%
Frontal	1.1
Temporal	5.6
Parietal	5.2
Occipital	3.2
More than one site	0.0

TABLE 4(b) (vi) SCALP INJURY / SITE / AGE / SKULL FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
NONE	123	0		298	2	0.7%	250	0		77	1	1.3%	758	3	0.4%
FRONTAL	225	4	1.7%	1592	7	0.4%	1015	5		187	2	1.1%	3062	19	0.6%
TEMPORAL	15	1	6.3%	91	1	1.1%	129	2	0.5%	34	2	5.6%	274	6	2.1%
PARIETAL	45	13	22.4%	195	7	3.5%	343	8	1.5%	73	4	5.2%	667	33	4.7%
OCCIPITAL	28	5	15.2%	402	7	1.7%	461	13	2.3%	91	3	3.2%	990	29	2.8%
> ONE SITE	6	2	25.0%	51	3	5.6%	51	7	12.1%	15	0		124	12	8.8%
MISSING / NOT RECORDED	1	0		8	0		5	0		1	0		16	0	

TABLE: 4 (b) (vii) SITE OF INJURY/SKULL FRACTURES

Site	Rad #	% Rad	Site % #
None/recorded	3	0.4	2.9
Frontal	19	0.6	18.6
Temporal	6	2.1	5.9
Parietal	33	4.7	32.4
Occipital	29	2.8	28.4
> one site	12	8.8	11.8

The relative fracture frequency is:

Non Recorded	0.004
Frontal	0.006
Temporal	0.021
Parietal	0.047
Occipital	0.028
> one site	0.088

TABLE 4(b) (viii) SCALP SITE/FRACTURE PROBABILITY

Superficial laceration	Frontal	1-7 per 1000 patients.
Galeal lacerations	Frontal	8-126 per 1000 patients
Swelling and abrasions contused	Temperoparietal	32-97 per 1000 patients
All galeal lacerations	Temperoparietal	23-358 per 1000 patients
Swelling and abrasions	Occipital	36-79 per 1000 patients
All galeal lacerations	Occipital	18-291 per 1000 patients

The probabilities here recorded might identify a level of acceptable risk in withholding skull xrays. The calculation of risk of a fracture being present, when a scalp injury and a scalp site is combined i.e. frontal site and superficial laceration, warrant serious consideration of a reduction in requests for skull xrays. Due evaluation would also be required of the history, cause and mechanism. In the absence of penetrating injury, foreign body, high velocity forces or signs of cerebral dysfunction, skull xrays could be withheld, with justification and within a level of acceptable risks are frequently skin lacerations only without cerebral injury though penetration always needs to be excluded.

TABLE 4 (b) (ix) SCALP FEATURE / SITE FRACTURE

	None			Frontal			Temporal			Parietal			Occipital			> 1 Site			Not Recorded		
	N	Xray	#	N	Xray	#	N	Xray	#	N	Xray	#	N	Xray	#	N	Xray	#	N	Xray	#
No External Injury	693	438	3	61	43	0	15	9	0	25	18	2	66	49	2	6	5	0	2	2	0
Swelling	29	21	0	964	743	9	91	80	4	195	167	21	337	288	17	62	54	6	5	5	0
Abras/Contusion	19	10	0	646	458	4	65	48	1	88	64	5	102	76	7	41	37	5	0	0	0
Sup Lac < 5 cm	19	7	0	1341	639	4	101	56	1	369	175	3	488	219	1	22	16	0	2	0	0
Sup Lac > 5 cm	0	0	0	16	10	0	3	2	0	12	9	0	7	5	0	2	0	0	0	0	0
Galea Lac < 5 cm	0	0	0	38	25	2	3	3	0	9	7	1	15	11	0	0	0	0	0	0	0
Not Recorded	0	0	0	9	9	0	1	0	0	2	2	1	4	4	2	3	3	1	0	0	0
Total	1	1	0	6	1	0	1	1	0	0	0	0	0	0	0	0	0	0	7	6	0
	761	477	3	3081	1928	19	280	199	6	700	442	33	1019	652	29	136	115	12	16	13	0

	%	%	%	%	%	%	%	%	4	%	%	%	%	%	%	%	%	%	%	%	%
	Xray	#	#	Xray	#	#	Xray	#	#	Xray	#	#	Xray	#	#	Xray	#	#	Xray	#	#
	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)	(of N)	(of N)	(Of Xr)
No External Injury	63.2%	0.4%	0.7%	70.5%	-	-	60.0%	-	-	72.0%	8.0%	4.1%	83.3%	3.0%	4.1%	83.3%	-	-	100%	-	-
Swelling	72.4%	-	-	77.1%	0.9%	1.2%	67.9%	5.5%	5.6%	85.6%	10.8%	5.9%	87.1%	5.0%	5.9%	67.1%	9.7%	11.1%	100%	-	-
Abras/Contusion	52.6%	-	-	10.9%	0.6%	0.9%	73.8%	2.1%	2.1%	72.7%	5.7%	9.2%	90.2%	6.9%	9.2%	90.2%	12.2%	13.5%	-	-	-
Sup Lac< 5 cm	36.6%	-	-	47.7%	0.3%	0.6%	55.4%	1.8%	1.8%	47.4%	0.8%	0.5%	72.7%	0.2%	0.5%	72.7%	-	-	-	-	-
Sup Lac> 5 cm	-	-	-	62.5%	-	-	66.7%	-	-	75.0%	-	-	-	-	-	-	-	-	-	-	-
Galea Lac < 5 cm	-	-	-	65.8%	5.3%	8.0%	100%	-	-	77.6%	11.1%	-	-	-	-	-	-	-	-	-	-
Galea Lac > 5 cm	-	-	-	100%	-	-	-	-	-	100%	50.0%	50.0%	100%	50.0%	50.0%	100%	33.3%	33.3%	-	-	-
Not Recorded	100%	-	-	16.7%	-	-	71.1%	-	-	-	-	-	-	-	-	-	-	-	65.7%	-	-
Total	62.7%	0.4%	0.6%	62.6%	0.6%	1.0%		3.0%	3.0%	63.1%	64.0%	4.4%	84.6%	2.8%	4.4%	84.6%	10.4%	10.4%	81.3%	0.0%	0.0%

4(b)(SA) Site of Injury – Skull Fracture

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the site of the injury, (chi-squared = 103.4, df-5, p<0.0005). In particular children with a head injury on the parietal area have substantially more fracture than would be expected.

The number of skull fractures by the site of injury and the expected number under a hypothesis of independence is shown in Table 4(b)(SA(i).

TABLE 4(b)(SA)(i): Expected and Observed number of skull fractures by site of injury (Under H0: skull fracture is independent of site of injury)

			SKULL #		
			No	**Yes**	**Total**
SCALP INJURY SITE	**None**	Count	752.0	4.0	756
		Expected Count	741.9	14.1	756
	Frontal	Count	3043.0	25.0	3068
		Expected Count	3010.8	57.2	3068
	Temporal	Count	274.0	6.0	280
		Expected Count	274.8	5.2	280
	Parietal	Count	665.0	34.0	699
		Expected Count	686.0	13.0	699
	Occipital	Count	983.0	30.0	1013
		Expected Count	994.1	18.9	1013
	More than one site	Count	123.0	12.0	135
		Expected Count	132.5	2.5	135
Total		Count	5840.0	111.0	5951
		Expected Count	5840.0	111.0	5951

When children with an injury on the temporal or parietal areas are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 31.6, df=1, p<0.0005) and have an odds ratio of 2.9 (CI=(2.0,4.4)). Therefore a child presenting with an injury to the temporal or parietal area is about three times as likely to result in a skull fracture than children with an injury to another site.

The odds ratio and 95% confidence intervals for each site of injury are:

	Odds Ratio	CI
None	0.25	(0.09, 0.69)
Frontal	0.27	(1.17, 0.42)
Temporal	1.16	(0.51, 2.67)
Parietal	3.44	(2.28, 5.19)
Occipital	1.83	(1.20, 2.80)
More than one site	5.63	(3.02, 10.53)

4(b) Site of Injury – Sex - Univarate Analysis

The sex of patients presenting with a head injury was found to be dependent on the site of injury, (chi-squared = 25.15, df=5, $p<0.0005$). In particular females were less likely to present with an injury to the temporal or parietal areas (chi-squared = 14.36, df=1, $p<0.0005$), and more likely to present with no site of injury (chi-squared = 13.56, df=1, $p<0.0005$).

The number of patients of each sex by site injury and the expected number under a hypothesis of independence is shown in Table 4(b)(SA)(ii).

TABLE 4(b)(SA)(ii) : Expected and Observed number of patients of each sex by site of injury (Under H0: site of injury is independent of sex)

			SEX		
			Male	**Female**	**Total**
SCALP INJURY SITE	**None**	Count	458.0	303.0	761
		Expected Count	502.9	258.1	761
	Frontal	Count	2025.0	1053.0	3078
		Expected Count	2034.1	1043.9	3078
	Temporal	Count	196.0	84.0	280
		Expected Count	185.0	95.0	280
	Parietal	Count	503.0	197.0	700
		Expected Count	462.6	237.4	700
	Occipital	Count	671.0	348.0	1019
		Expected Count	673.4	345.6	1019
	More than one site	Count	95.0	41.0	136
		Expected Count	89.9	46.1	136
Total		Count	3948.0	2026.0	5974
		Expected Count	3948.0	2026.0	5974

5. Results – Headache and Vomiting

TABLE 5 (i) HEADACHE AND VOMITING

	N	%
None	4562	76
Vomiting	482	8
Headache	660	11
Headache/Vomiting	287	5

If headache and vomiting reflect cerebral dysfunction, the results suggest that in only a quarter of the children, was such evidence found in the Accident & Emergency Department.

TABLE 5 (ii) HEADACHE/VOMITING/AGE

N	H-V-	H+	V+	H+V+	All H+	All V+
Age						
< 1 year	384	6	77	1	7	78
1-4 years	2203	155	245	61	216	306
5-11 years	1630	357	133	169	526	302
12 - 14 years	287	131	22	50	181	72

The association of headache and vomiting with cerebral dysfunction, and significant head trauma might be expected to prompt requests for skull xrays by Casualty Medical Officers.

TABLE 5 (iii) HEADACHE/VOMITING/SKULL XRAYS

	NXR	%
No headache/no vomiting	2665/4562	58.0
Headache	499/660	76.0
Vomiting	398/484	82.0
Headache/vomiting	264/287	92.0

These results confirm a discretionary approach to the request for skull xrays. The results of the skull xrays and their association with headache and vomiting in the different age groups is shown Table 6 (iv)

Figure 5 (a) HEADACHE/VOMITING/FRACTURES%

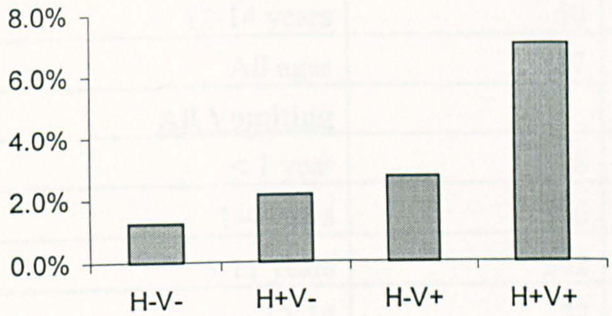


TABLE 5(iv) HEADACHE/ VOMITING/AGE/FRACTURE

No Headache Vomiting	N	N#	%
< 1 year	384	21	
1-4 years	2203	18	
5-11 years	1620	11	
12-14 years	287	4	
All ages	4562	55	1.2
Headache Only			
< 1 year	6	0	
1-4 years	155	3	
5-11 years	362	7	
12-14 years	131	4	
All ages	660	14	2.1
Vomiting only			
< 1 year	77	3	
1-4 years	245	4	
5-11 years	133	4	
12-14 years	22	32	
All ages	484	23	2.7

TABLE 5 (v) HEADACHE/VOMITING/AGE FRACTURE

Headache and Vomiting	N	N#	%
< 1 year	1	1	
1-4 years	61	2	
5-11 years	169	13	
12-14 years	50	3	
All ages	287	20	7.0
All Vomiting			
< 1 year	78	4	
1-4 years	306	6	
5-11 years	302	17	
12-14	72	4	
All ages	771	33	4.3
All Headache			
< 1 year	7	1	
1-4 years	216	5	
5-11 years	526	20	
12-14 years	181	78	
All ages	947	34	3.6

The results show that when headache and vomiting occur together, there is a greater risk of a fracture being present than when the symptoms occur separately.

When the symptoms are matched to the mechanism of injury, blunt impact dominates.

When related to the cause of injury Road Traffic Accidents predominate.

The results would suggest that headache and vomiting are indications of cerebral disturbance resulting from high velocity impact, associated with road traffic accidents and a proportion of falls. The incidence of skull fracture is higher in those with both the symptoms.

TABLE 5 (vi) FRACTURE FREQUENCY

No symptoms	0.012
Headache	0.021
Vomiting	0.027
Headache and Vomiting	0.070
All headache	0.036
All vomiting	0.043

When children were compared with adults in the Monklands I Study with reported headache and vomiting there were differences.

TABLE 5 (vii) HEADACHE VOMITING/CHILDREN/ADULTS

	Children 1721	Adults 1650	All
None	83.0	78.1	80.5
Headache	7.0	16.7	11.8
Vomiting	7.0	2.7	5.0
Headache and Vomiting	3.0	2.5	2.7

These figures are not matched to injury severity.

In the emergency room a comparison with Hermann Hospital, Houston, Texas and adults from Scottish Hospitals in the Emergency Department was made

TABLE: 5 (viii) HEADACHE/VOMITING

	Hermann	Scotland	Monklands
	%	%	%
Headache only	5.0	6.0	7.0
All headache	6.0	8.0	10.0
Vomiting only	10.0	8.0	7.0
All vomiting	12.0	10.0	10.0
Either headache or vomiting	15.0	13.0	14.0
Both headache and vomiting	2.0	2.0	3.0
Neither headache or vomiting	83.0	85.0	83.0

TABLE 5 (ix) CHILDHOOD CONCUSSION SYNDROME

N	M	F
3	0	3

The condition may have been unreported because the methods did not specifically provide for the diagnosis. The cases were identified by a review of the inpatient records.

TABLE 5 (x) HEADACHE / VOMITING/ CAUSE / AGE / FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
No HEADACHE/VOMITING	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	7	0		33	0		25	0		3	0		69		
RTA - PEDESTRIAN	0	0		29	3	9.4%	73	3	3.9%	7	0		111	6	5.1%
RTA - CYCLIST	2	0		16	1	5.9%	34	1	2.9%	10	0		64	2	3.0%
ASSAULT	8	0		71	1	1.4%	264	1	0.4%	76	1	1.3%	431	3	0.7%
FALL - SCHOOL	4	1	20.0%	66	2	2.9%	230	0		39	0		344	3	0.9%
FALL - HOME	303	16	5.0%	1459	2	0.1%	345	0		39	0		2168	19	0.9%
FALL - SPORT	19	1	5.0%	469	9	1.9%	599	6	1.0%	99	1	1.0%	1196	17	1.4%
NAI	5	1	16.7%	2	0		6	0		0	1	100%	14	2	12.5%
MISSING / NOT RECORDED	15	2	11.8%	40	0		43	0		10	1	9.1%	110	3	2.7%
TOTAL	363	21	5.5%	2185	18	0.8%	1619	11	0.7%	283	4	1.4%	4507	55	1.2%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
HEAD ONLY	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	1	0		5	0		5	0		3	0		14		
RTA - PEDESTRIAN	1	0		6	0		18	0		4	2	33.3%	29	2	6.5%
RTA - CYCLIST	0	0		2	0		7	0		7	0		17		
ASSAULT	1	0		9	0		34	0		26	0		70		
FALL - SCHOOL	0	0		6	0		69	1	1.4%	24	0		100	1	1.0%
FALL - HOME	3	0		73	0		67	0		6	1	14.3%	152	1	0.7%
FALL - SPORT	0	0		48	3	5.9%	145	6	4.0%	53	1	1.9%	252	10	3.8%
NAI	0	0		0	0		2	0		2	0		4		
MISSING / NOT RECORDED	0	0		3	0		3	0		2	0		8		
TOTAL	6	0	0%	152	3	1.9%	350	7	2.0%	127	4	3.1%	646	14	2.1%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
VOMITING ONLY	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	1	0		2	0		5	2	28.6%	0	0		8	2	20%
RTA - PEDESTRIAN	0	0		2	1	33.3%	1	1	50.0%	1	0		4	2	33.3%
RTA - CYCLIST	0	0		2	0		3	0		0	0		5		
ASSAULT	2	0		3	0		14	0		4	0		23		
FALL - SCHOOL	4	0		6	0		30	0		6	0		46		
FALL - HOME	63	1	1.6%	173	2	1.1%	17	0		2	1	33.3%	256	5	1.9%
FALL - SPORT	2	1	33.3%	46	1	2.1%	58	1	1.7%	8	0		119	3	2.5%
NAI	1	0		1	0		0	0		0	0		2		
MISSING / NOT RECORDED	1	1	50.0%	6	0		1	0		0	0		8	1	11.1%
TOTAL	74	3	3.9%	241	4	1.6%	129	4	3.0%	21	1	4.5%	471	13	2.7%

TABLE 5 (xi) HEADACHE / VOMITING/ MECHANISM / AGE / FRACTURE

No HEADACHE/VOMITING	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
FALL - SOFT	54	1	1.8%	62	0		27	1	3.6%	5	0		150	2	1.3%
FALL - SHARP	27	0		430	1	0.2%	170	0		23	0		659	1	0.2%
FALL - HARD	178	15	7.8%	1061	15	1.4%	705	7	1.0%	104	0		2070	38	1.8%
BUMP - SHARP	22	0		222	0		135	0		20	0		404		
BUMP - HARD	58	3	4.9%	274	1	0.4%	238	2	0.8%	41	0		614	6	1.0%
BUMP - BLUNT	17	1	5.6%	91	1	1.1%	243	1	0.4%	71	3	4.1%	433	6	1.4%
STRUCK - SHARP	2	0		34	0		91	0		17	0		147		
MISSING/NOT RECORDED	5	1	16.7%	11	0		10	0		2	1		30	2	6.3%
TOTAL	363	21	5.5%	2185	18	0.8%	1619	11	0.7%	283	4	1.4%	4507	55	1.2%

HEAD ONLY	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - SOFT	1	0		2	0		6	0		4	0		13		
RTA - SHARP	0	0		19	0		27	2	6.9%	2	0		49	2	3.9%
RTA - HARD	2	0		84	3	3.4%	198	5	2.5%	52	3	5.5%	344	11	3.1%
BUMP SHARP	2	0		13	0		12	0		5	0		32		
BUMP - HARD	0	0		19	0		57	0		28	0		105		
BUMP - BLUNT	1	0		14	0		40	0		30	1	3.2%	86	1	1.1%
STRUCK - SHARP	0	0		1	0		9	0		6	0		16		
MISSING/NOT RECORDED	0	0		0	0		1	0		0	0		1		
TOTAL	6	0	0%	152	3	1.9%	350	7	2.0%	127	4	3.1%	646	14	2.1%

VOMITING ONLY	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
FALL - SOFT	8	0		14	0		5	0		0	0		28		
FALL - SHARP	4	0		21	0		4	0		0	0		29		
FALL - HARD	46	2	4.2%	148	4	2.6%	90	2	2.2%	7	1	12.5%	295	10	3.3%
BUMP - SHARP	1	0		10	0		0	0		1	0		12		
BUMP - HARD	12	0		39	0		16	2	11.1%	6	0		74	2	2.6%
BUMP - BLUNT	1	1	50.0%	7	0		14	0		6	0		28	1	3.4%
STRUCK - SHARP	2	0		0	0		0	0		1	0		3		
MISSING/NOT RECORDED	0	0		2	0		0	0		0	0		2		
TOTAL	74	3	3.9%	241	4	1.6%	129	4	3.0%	21	1	4.5%	471	13	2.7%

TABLE 5 (xii) HEADACHE / VOMITING/ MECHANISM / AGE / FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
HEADACHE & VOMITING	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	0	0		0	0		1	0		0	0		1	0	0.0%
RTA - PEDESTRIAN	0	0		0	0		4	0		3	2	40.0%	7	2	22.2%
RTA - CYCLIST	0	0		0	0		4	2	33.3%	3	0		7	2	22.2%
ASSAULT	0	0		3	0		11	1	8%	5	1	16.7%	19	2	9.5%
FALL - SCHOOL	0	0		5	0		36	0		8	0		50	0	
FALL - HOME	0	1	100%	28	1	3.4%	32	1	3.0%	4	0		66	4	5.7%
FALL - SPORT	0	0		23	1	4.2%	65	9	12%	22	0		112	10	8.2%
MISSING/NOT RECORDED	0	0		0	0		3	0		2	0		5	0	7.0%
TOTAL	0	1	100%	59	2	3.3%	156	13	7.7%	47	3	16.7%	267	20	7.0%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
VOMITING	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	1	0		2	0		6	2	25.0%	0	0		9	2	18.2%
RTA - PEDESTRIAN	0	0		2	1	33.3%	5	1	16.7%	4	2	33.3%	11	4	26.7%
RTA - CYCLIST	0	0		2	0		7	2	22.2%	3	0		12	2	14.3%
ASSAULT	2	0		6	0		25	1	3.8%	9	1	10.0%	42	2	4.5%
FALL - SCHOOL	4	0		11	0		66	0		14	0		96	0	
FALL - HOME	63	2	3%	201	3	1.5%	49	1	2.0%	6	1	14.3%	322	9	2.7%
FALL - SPORT	2	1	33.3%	69	2	2.8%	123	10	7.5%	30	0		231	13	5.3%
NAI	1	0		1	0		0	0		0	0		2	0	
MISSING/NOT RECORDED	1	1	50%	6	0		4	0		2	0		13	1	7.1%
Total	74	4	5%	300	6	2%	285	17	6%	68	4	6.0%	738	33	4%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
HEADACHE	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - OCCUPANT	1	0		5	0		6	0		3	0		15	0	
RTA - PEDESTRIAN	1	0		6	0		22	0		7	4	36.4%	36	4	10.0%
RTA - CYCLIST	0	0		2	0		11	2	15.4%	10	0		24	2	7.7%
ASSAULT	1	0		12	0		45	1	2.2%	31	1	3.1%	89	2	2.2%
FALL - SCHOOL	0	0		11	0		105	1	0.9%	32	0		150	1	0.7%
FALL - HOME	3	1	25%	101	1	1.0%	99	1	1.0%	10	1	9.1%	218	5	2.2%
FALL - SPORT	0	0		71	4	5.3%	210	15	6.7%	75	1	1.3%	364	20	5.2%
NAI	0	0		0	0		2	0		2	0		4	0	
MISSING/NOT RECORDED	0	0		3	0		6	0		4	0		13	0	
TOTAL	6	1	14%	211	5	2.0%	506	20	4.0%	174	7	4.0%	913	34	4.0%

TABLE 5(xiii) HEADACHE / VOMITING/ MECHANISM / AGE / FRACTURE

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
No HEADACHE/VOMITING	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
FALL - SOFT	0	0		3	0		5	0		2	0		11		
FALL - SHARP	0	0		1	0		3	1	25.0%	0	0		4	1	20.0%
FALL - HARD	0	1	100%	45	2	4.3%	103	10	8.8%	21	2	8.7%	172	15	8%
BUMP - SHARP	0	0		1	0		5	0		2	0		8	1	11.1%
BUMP - HARD	0	0		6	0		28	1	3.4%	9	0		44	1	2.2%
BUMP - BLUNT	0	0		1	0		12	1	7.7%	10	1	9.1%	23	2	8%
STRUCK - SHARP	0	0		2	0		0	0		2	0		4		
MISSING/NOT RECORDED	0	0		0	0		0	0		1	0		1		
TOTAL	0	1	100%	59	2	3.3%	156	13	7.7%	47	3	6%	267	20	7%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
VOMITING (ALL)	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
RTA - SOFT	8	0		17	0		10	0		2	0		39	0	
RTA - SHARP	4	0		22	0		7	1	12.5%	0	0		33	1	2.9%
RTA - HARD	46	3	6%	193	6	3%	193	12	5.9%	28	3	9.7%	467	25	5.1%
BUMP SHARP	1	0		11	0		5	0		3	0		20	1	4.8%
BUMP - HRD	12	0		45	0		44	3	6.4%	15	0		118	3	2.5%
BUMP - BLUNT	1	1	50.0%	8	0		26	1	3.7%	16	1	5.9%	51	3	5.6%
STRUCK - SHARP	2	0		2	0		0	0		3	0		7	0	
MISSING/NOT RECORDED	0	0		2	0		0	0		1	0		3	0	
TOTAL	74	4	5%	300	6	2%	285	17	5.6%	68	4	5.6%	738	33	4.3%

	< 1 Year			1 - 4 Years			5 -11 Years			12 - 14 Years			All Ages		
HEADACHE (ALL)	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #	No #	#	% #
FALL - SOFT	1	0		5	0		11	0		6	0		24	0	
FALL - SHARP	0	0		20	0		30	3	9.1%	2	0		53	3	5.4%
FALL - HARD	2	1	33%	129	5	3.7%	301	15	4.7%	73	5	8.2%	516	26	4.8%
BUMP - SHARP	2	0		14	0		17	0		7	0		40	1	2.4%
BUMP - HARD	0	0		25	0		85	1	1.2%	37	0		149	1	0.7%
BUMP - BLUNT	1	0		15	0		52	1	1.9%	40	2	4.8%	109	3	2.7%
STRUCK - SHARP	0	0		3	0		9	0		8	0		20	0	
MISSING/NOT RECORDED	0	0		0	0		1	0		1	0		2	0	
TOTAL	6	1	14%	211	5	2.3%	506	20	3.8%	174	7	3.9%	913	34	3.6%

5(a) Risk of Fracture by Headache – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of headache, (chi-squared = 41.1, df=1, p<0.0005).

The number of skull fractures by the presence of headache and the expected number under a hypothesis of independent is shown in Table 5(SA)(i).

TABLE 5(SA)(a)(i): Expected and Observed number of skull fractures by headache (Under HO: skull fracture is independent of headache)

			SKU LL #		
			No	**Yes**	**Total**
HEADACHE	**None**	Count	4369.0	45.0	44414
		Expected Count	4347.2	66.8	4414
	Headache	Count	903.0	36.0	939
		Expected Count	924.8	14.2	939
Total		Count	5272.0	81.0	5353
		Expected Count	5272.0	81.0	5353

Headache had an odds ratio of 3.9 (CI=(2.5, 6.0)). Therefore a child presenting with a history of headache is almost four(4) times as likely to result in a skull fracture than children without a history of headache.

The odds ratios and 95% confidence intervals are:

	Odds Ratio	**CI**
No Headache	0.26	(0.17, 0.40)
Headache	3.9	(2.5, 6.0)

The sex of patients presenting with a head injury was found to be independent of a history of headache, chi-squared = 0.582, df=1, p=0.445).

The number of patients of each sex by history of headache and the expected number under a hypothesis of independence is shown in table 5(a)(SA)(ii).

Table 5(a)(SA)(ii): Expected and Observed number of patients of each sex by headache (Under HO: headache is independent of sex).

			SEX		
			MALE	**FEMALE**	**Total**
HEADACHE	**None**	Count	2960.0	1460.0	4420
		Expected Count	2950.0	1460.0	4420
	Headache	Count	622.0	325.0	947
		Expected Count	632.0	315.0	947
Total		Count	3582.0	1785.0	5367
		Expected Count	3582.0	1785.0	5367

Vomiting

The sex of patients presenting with a head injury was found to be dependent on a history of vomiting, (chi-squared = 9.93, df=2, p=0.007).

The number of patients of each sex by vomiting and the expected number under a hypothesis of independence is shown in table 5(b)(SA)(iii).

Table 5(b)(SA)(iii): Expected and Observed number of patients of each sex by vomiting (Under HO: vomiting is independent of sex).

			SEX		
			Male	**Female**	**Total**
VOMITING	**None**	Count	3472.0	1730.0	5202
		Expected Count	3436.6	1765.4	5202
	Vomited (once)	Count	226.0	128.0	354
		Expected Count	233.9	120.1	354
	Vomited (more than once)	Count	248.0	169.0	417
		Expected Count	275.5	141.5	417
Total		Count	3946.0	2027.0	5973
		Expected Count	3946.0	2027.0	5973

5(b) Risk of Fracture by Vomiting – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of vomiting, (chi-squared = 35.2, df=2, p<0.0005).

The number of skull fractures by vomiting and the expected number under a hypothesis of independence is shown in Table 5(b)(SA)(i).

TABLE 5(b)(SA)(i) : Expected and Observed number of skull fractures by vomiting (Under H0: skull fracture is independent of vomiting)

			SKU LL #		
			No	**Yes**	**Total**
VOMITING	**None**	Count	5114.0	75.0	5189
		Expected Count	5094.0	95.0	5189
	Vomited (once)	Count	334.0	18.0	352
		Expected Count	345.6	6.4	352
	Vomited (more than once)	Count	394.0	16.0	410
		Expected Count	402.5	7.5	410
Total		Count	5842.0	109.0	5951
		Expected Count	5842.0	109.0	5951

When all children with a history of vomiting (either once or more than once) were considered as a group and compared to children with no history of vomiting they showed an odds ratio of 3.2 (CI=(2.1, 4.8)). Therefore a child presenting with a history of vomiting is about three(3) times as likely to result in a skull fracture than children without a history of vomiting.

The odds ratios and 95% confidence intervals are:

	Odds ratio	**CI**
None	0.31	(0.21, 0.47)
Vomited (once)	3.26	(1.94, 5.47)
Vomited (more than once)	2.38	(1.39, 4.08)

5(c)(SA) Risk of Fracture by Headache & Vomiting

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of headache associated with vomiting, (chi-squared = 48.2, df=1, p<0.0005).

The number of skull fractures by the presence of both headache and vomiting and the expected number under a hypothesis of independence is shown in Table 5(c)(SA)(i) .

TABLE 5(c)(SA)(i) : Expected and Observed number of skull fractures by headache (Under H0: skull fracture is independent of headache and vomiting)

			SKULL #		
			No	**Yes**	**Total**
HEADACHE & VOMITING	**No Headache & Vomiting**	Count	5467.0	83.0	5550
		Expected Count	5452.0	98.0	5550
	Headache & Vomiting	Count	263.0	20.0	283
		Expected Count	278.0	5.0	283
Total		Count	5730.0	103.0	5833
		Expected Count	5730.0	103.0	5833

Headache and vomiting had an odds ratio of 5.01 (CI=(3.0, 8.3)). Therefore a child presenting with a history of headache and vomiting was five times as likely to result in a skull fracture as children without a history of both headache and vomiting.

The odds ratios and 95% confidence intervals are:

	Odds ratio	**CI**
No Headache & Vomiting	0.20	(0.12, 0.33)
Headache & Vomiting	5.01	(3.0, 8.3)

Headache, Vomiting and Unconsciousness

If we consider children presenting with a history of unconsciousness (of any length), headache and vomiting (once or more than once) as a group, and compare this group to all other patients we find that this group has a significantly greater risk of skull fracture than other patients (chi-squared = 53.8, df=1, p<0.0005). Although this represents a small proportion of children seen (63 patients, 1.1%), this group has a very high odds ratio (9.5, CI=(4.6, 19.7). Within our study, patients presenting with a combination of vomiting, headache and unconsciousness were more than nine(9) times as likely to have a skull fracture than other patients.

TABLE 5(c)(SA)(i) : Expected and Observed number of skull fractures by vomiting, headache and unconsciousness (Under H0: skull fracture is independent of vomiting, headache and unconsciousness)

			SKULL #		
			No	**Yes**	**Total**
Vomiting + Headache + Unconsciousness	**No**	Count	5800.0	102.0	5902
		Expected Count	5792.2	109.8	5902
	Yes	Count	54.0	9.0	63
		Expected Count	61.8	1.2	63
Total		Count	5854.0	111.0	5965
		Expected Count	5854.0	111.0	5965

The odds ratios and 95% confidence intervals are:

	Odds Ratio	**CI**
Vomiting + Headache + Unconscious	9.5	(4.6, 19.7)
Not Vomiting + Headache + Unconscious	0.11	(0.05, 0.22)

6. The Results – Diagnostic Imaging

TABLE 6 (i) SKULL X-RAY REQUESTS

	N	%
All patients	5993	
Skull x-rays	3826	64
No skull x-rays	2164	36

Having obtained the skull films, the doctor examines them for the presence of the fracture. Its detection or absence will assist in decisions relating to the child's management. The correct interpretation is therefore of importance. The doctor's opinion is also likely to be conveyed to the parents of the child, and occasionally to other persons or agencies.

The Casualty Officers examined a very large number of films. All were subsequently reported by a Radiologist. The doctors diagnosed 45 fractures not subsequently confirmed by a Radiologist. They also missed 15 fractures, which were detected by the Radiologist. This is a source of concern, representing 15% of the abnormal films. The actual yield of fractures from xrays was low at 2.6% of the xrays and 1.7% of all the patients.

The false positive xrays would almost certainly have resulted in admission for observation. This was the policy in all the hospitals. It might also have implications for other agencies, such as police, and social workers. It would also engender anxiety in the parents. At best, retracting the diagnosis might prove embarrassing.

TABLE 6 (ii) SKULL XRAYS/INTERPRETTION

	N	%
Skull Xrays	3826	64.0
Casualty Officer Fracture	147	-
Missed Fractures Casualty Officer	15	-
Radiologist Fractures	102	-
Depressed/Fracture 9/102	9	-
False Positive Fracture	45	-
Base of Skull Fracture Clinical	10	
Confirmed	1	
Yield of Fracture		2.6
Missed Skull Fractures	15	15

Xray requests in the different age groups and by sex is shown Table 7 (iii)

TABLE 6 (iii) SKULL XRAY/AGE/SEX

	N	% XR	Male %#	N	%XR	Female %#
<1 year	265	69	7.7	203	70	7.7
1-4 years	1663	61	1.5	998	65	1.8
5-11 years	1606	63	2.5	683	65	2.3
12-14 years	364	70	3.9	126	68	2.3

The substantial increase in the fracture rate in males 12-14 years reflects the more serious injury sustained.

TABLE 6 (iv) SKULL FRACTURE/AGE/SEX

	Male	Female
	%XR	%XR
<1 year	7.7	7.7
1 - 4 years	1.5	1.8
5 - 11 years	2.5	2.3
12 - 14 years	4.0	2.3

The results expressed as a percentage of the skull xrays confirmed the vulnerability of the infant skull, and the more serious injury in the older male child.

TABLE 6 (v) AGE/FRACTURE

	N
< 1 year	25
1 - 4 years	27
5 - 11 years	35
12 - 14 years	12

The vulnerability of the infant skull is confirmed.

Risk of fracture by a single factor

Age group	Relative Risk
< 1 year	0.054
1-4 years	0.010
5-11 years	0.015
12-14 years	0.0124

The infants are most likely to have fractures, 1-4 years old least likely.

TABLE 6 (vi) XRAYS/FRACTURE BY GRADE OF DOCTOR

Grade	N	Xrays	%XR	#CO	#RAD	#%N	#%XR	FALSE POSITIVE	FALSE NEGATIVE
SHO	4302	2721	63	96	66	1.5	2.4	39	13
Registrar	344	210	61	13	11	3.2	5.2	4	2
Senior Registrar	143	116	81	7	6	4.2	5.2	1	-
Consultant	82	50	61	4	2	2.4	4.0	2	-
Other	723	437	60	14	10	1.4	2.3	3	-
N/R	399	292	73	13	7	1.8	2.4	3	-

(399 patients and their xray status could not be allocated to doctor grades)

THE UTILITY OF SKULL XRAYS/DOCTOR GRADE

Senior House Officers see the majority of patients, 4302 initially, and the majority exclusively. Senior House Officer xray usage was 66%. This is only a little higher than other grades of doctor. It clearly demonstrates that discretion is applied, and that doctors in Emergency Departments in the United Kingdom, at least, do not order skull xrays routinely.

Registrars were on the establishment of four A & E Units, Senior Registrars in three, and consultants in five hospitals. The results relating to these grades are shown Table 6 (vi).

The yield of fractures, in relation to the total number of patients, suggests that senior doctors achieved a more effective use of the limited number of skull xrays they requested. Alternatively they may have been seeing the more seriously injured children. The Study provided an opportunity to assess the performance of a number of doctors in different Accident & Emergency Departments who were seeing children following head trauma.

TABLE 6 (vii) GLASGOW ROYAL INFIRMARY/XRAYS/DOCTOR GRADE

Grade	N	% XRAYS	#RAD	Yield#	False +ve	False -ve
SHO	410	70	5	1.7	1	2
Registrar	168	70	6	5.1	2	-
Senior Registrar	4	80	3	-	-	-
Consultants	19	84	1	-	1	-
Others	203	65	2	-	-	-

TABLE 6 (viii) POOLE HOSPITAL XRAY REQUESTS

Grade	%
SHO	35.0
Registrar	20.0
Others	30.0
Clinical Assistants	17.0

The efficiency of the Senior Registrar at Glasgow Royal Infirmary may reflect case selection, and high risk patients, particularly in view of the small numbers seen. There were four false positives and two false negative interpretations at Glasgow.

At Poole General Hospital the staffing was consistent and stable. There was a low rate of requests, 35% from the SHO's, 20% from the Registrar and 30% for others. From 177 skull series, five fractures were detected, a yield of 2.8%. No child was scanned during the period of the Study. Clinical Assistants employed in the Department varied in their usage from 17%-66%.

At Alder Hey the dedicated paediatric hospital, only 10 doctors could be identified individually. Usage varied from 47%-100%.

TABLE 6 (ix) ALDER HEY/SHO USAGE

Code	N	%XR	Co#	Rad#
31	1	100	-	-
24	4	100	-	-
35	60	47	2	2
36	6	67	-	-
37	57	100	-	-
38	24	79	-	-
39	21	71	1	1
40	8	100	-	-
41	7	100	1	0
42	2	100	1	0
N/R	741	16	16	11

There were two false positives from this group of Senior House Officers. This data illustrates the variable practices.

TABLE 6 (x) ALDER HEY XRAYS BY GRADE OF DOCTOR

	N	%Xray	Rad #	Yield Xray	False +ve	False -ve
SHO	489	76	10	2.7	4	2
Reg	36	67	0	-	2	-
Sen. Reg	90	86	2	2.6	-	-
Cons	13	69	-	-	-	-
Others	58	69	-	-	-	-

In Hull Royal Infirmary, where there were medical staffing problems, requests for radiology of he skull varied from 28% - 100%. 15 fractures were detected from 475 skull xray film series. There were 9 false positives.

During the course of the main Study, a subsidiary analysis was taken to compare various activities in Monklands Hospital, a District General seeing adults and children, and those at the Royal Aberdeen Children's Hospital which was dedicated exclusively to the care of children. Accordingly, the results are based on a six months analysis, comparing approximately 1,000 patients in each hospital. The results are shown

TABLE: 6 (xi) UTILISATION OF SKULL XRAYS

	MDGH	RACH
Not xrayed	321	553
Number of xrays	700	447
% of usage	68%	45%

TABLE 6 (xii) MDGH/SENIOR HOUSE OFFICERS

Code	NO. PATIENTS.	NXR	% OF XRAY	% YIELD
12	100	52	52	5.7
13	90	144	49	6.8
14	74	46	62	2.2
15	113	81	72	2.5
16	88	79	90	2.5
17	113	84	74	1.2
18	84	71	85	7.0
19	88	70	45	7.5

TABLE 6 (xiii) RACH/SENIOR HOUSE OFFICERS

Code	N	%XRAY	%YIELD
01	434	55	2.5
02	276	36	6.0
03	296	36	5.0

The xray requests made by the Senior House Officer at Aberdeen were interesting. The most experienced doctors Code 01 saw the largest number of children, but ordered proportionately more rays, 55%. It has been suggested that investigation can be used to control workload in a busy Department.

TABLE 6 (xiv) MDG/XRAY INTERPRETATION

CODE	NXR	# CO	#RAD	False +ve	False -ve
12	52	2	2	0	0
13	45	3	1	2	0
14	46	1	1	1	0
15	82	1	1	-	-
16	79	2	0	2	-
17	84	1	2	0	2
18	71	5	3	2	1
19	40	3	2	1	1

TABLE 6 (xv) RACH / SHO / CASUALTY OFFICER / RADIOLOGIST

	Patients	Xrays	%	Co#	RAD	Yield Pts	Yields XRS	False Positive	False Negative
01	434	241	55	6	5	1.1	2.0		1
02	276	100	36	6	3	1.0	3.0	4	1
03	290	106	36	5	2	0.7	1.9	3	0

There were 10 fractures diagnosed by a Radiologist. Nine fractures diagnosed by the Casualty Officer were not confirmed.
Clinical management was modified by the false interpretation. Two fractures were missed. Neither child came to any harm as a consequence.

A clinical assistant had 100% x-ray usage, despite extensive experience and seniority. The types of fracture showed a predominance of linear fractures. Base of skull fracture was diagnosed clinically. There were 3 depressed fractures at Monklands 0.3%, none at Aberdeen.

In the majority of participating Hospitals, there was a high, but variable use of skull xrays. There were more false positives than false negative interpretations, which reduce the risk to the patient. Only one child was identified in the series was not xrayed. This was at Aberdeen. She returned and was found to have a skull fracture the following day but came to no harm.

The opportunity to interpret skull fractures from experience is limited. In Aberdeen in six months, 10 skull fractures were detected. Each of the three doctors worked single handed. On average each doctor would see 3.3 fractures in the course of their appointment. In Monklands 16 fractures were seen amongst eight doctors. On average each doctor might see two fractures in six months.

Further enquiry at Monklands Hospital in 1992 revealed that 4,800 xrays were taken on 5,600 head injured patients, 40% in the paediatric category. Xray usage was 85%, an increase since the introduction of the of the Guidelines by the College of Radiologists. This suggests that casualty officers, despite teaching and exposure to the recommendations of the Royal College of Radiologists, are not persuaded of the value of relevance of the Guidelines to their individual practice. The other possibility is that the patients are not meeting the criteria. This seems unlikely in the face of the evidence.

The opportunity to interpret skull fractures from experience is limited. In Aberdeen in six months, 10 skull fractures were detected. Each of the three doctors worked single handed. On average each doctor would see 3.3 fractures in the course of their appointment. In Monklands 16 fractures were seen amongst eight doctors. On average each doctor might see three fractures in six months.

Further enquiry at Monklands Hospital in 1992 revealed that 4,800 xrays were taken on 5,600 head injured patients, 40% in the paediatric category. Xray usage was 85%, an increase since the introduction of the Guidelines by the College of Radiologists. This suggests that casualty officers, despite teaching and exposure to the recommendations of the Royal College of Radiologists, are not persuaded of the value or relevance of the Guidelines to their individual practice. The other possibility is that the patients are not meeting the criteria. This seems unlikely in the face of the evidence.

The role of skull fracture and impaired level of consciousness in alerting the physician to the potential for intracranial complication is established even in children.

The large numbers of minor head injuries seen in Accident & Emergency Departments in the U.K. makes it essential to triage the patients for investigation, or admission. CT scanners in the General Hospital make this possible, without involving the neurosurgical unit. A negative scan, in an asymptomatic child, should enable the child to be discharged. Symptomatic children with early negative scans may require admission, and a repeat scan, as well as neurological monitoring.

Asymptomatic children without any impairment of consciousness, and no skull fracture, can be discharged - as this study has shown, without significant risk. These two features can be used to triage for CT scanning and admission or discharge home.

7.(a) Results - Disposal

Table 7 (i) DISPOSAL/DISCHARGE ALL HOSPITALS

	N	%
Home	5265	88.0
Admitted to Hospital	728	12.0

TABLE 7 (ii) WARD/UNIT OF ADMISSION

	N	%
Short Stay Ward	191	3.0
Primary Surgical Ward	377	6.0
Neurosurgical Ward	155	2.5
Intensive Therapy Unit	5	0.8

Overall 88% of the children were discharged home although there was variation between the Units and in different age groups. The figure of 10.3% in Hull Royal Infirmary for admissions directly to the Neurosurgical Unit was the result of a policy in that hospital at the time of the Study, to admit all young children with head trauma, under the care of the Neurosurgeons if they were unfit for discharge from the A & E Unit.

There were differences relating to policies in the individual hospitals.

TABLE 7 (iii) DISPOSAL ALL HOSPITALS FROM THE ACCIDENT & EMERGENCY UNIT

	ALL	MDGH	GRI	RACH	AH	PGH	HRI
	%	%	%	%	%	%	%
Home	88.0	92.0	84.0	88.0	89.4	81.0	88.0
Short Stay Ward	3.0	2.0	0.7	7.0	0.2	19.0	1.5
Primary Surgical Ward	6.0	5.5	14.0	5.0	10.0	-	0.1
Neurosurgery Unity	2.0	0.8	1.0	-	0.1	-	10.3
Intensive Therapy Unit	0.1	0.1	-	-	0.2	-	0.1

TABLE 7 (iv) AGE / MALE. DISPOSAL / WARD OF ADMISSION

	Home		Short Stay Ward		Primary Surgical Ward		Neurosurgery Unit		Intensive Therapy Unit	
Male	N	%	N	%	N	%	N	%	N	%
< 1 Year	198	89.0	8	3.6	16	7.2	1	0.4	-	-
1 - 4 Years	1291	92.0	29	2.0	75	5.0	3	0.2	2	0.1
5 - 11 Years	1213	87.0	51	4.0	119	9.0	9	0.6	1	0.1
12 - 14 Years	237	79.0	28	9.0	28	9.0	7	2.3	-	-
Not Recorded	37	82.0	2	4.0	5	11.0	-	-	1	2.2
Total	2976	89.0	118	3.5	243	7.0	20	0.6	4	0.1

Hull Excluded

TABLE 7 (v) AGE/FEMALE. DISPOSAL / WARD OF ADMISSION

Female	HOME		Short Stay Ward		Primary Surgical Ward		Neurosurgery Unit		Intensive Therapy Unit	
	N	%	N	%	N	%	N	%	N	%
< 1 Year	141	86.0	4	2.0	18	11	2	1.2	-	-
1 - 4 Years	795	92.0	15	2.0	56	6.5	2	0.2	-	-
5 - 11 Years	527	87.0	30	5.0	48	7.9	1	0.2	-	-
12 - 14 Years	84	82.0	11	10.0	7	7.0	1	1.0	-	-
Not Recorded	16	84.0	-	-	3	16.0	-	-	-	-
Total	1563	86.0	60	3.4	132	7.5	6	0.3		

Hull Excluded

TABLE 7 (vi) SEX / DIPOSAL / WARD OF ADMISSION

	Home		Short Stay Ward		Primary Surgical Wards		Neurosurgery Unit		Intensive Therapy Unit	
	N	%	N	%	N	%	N	%	N	%
Male	2976	89.0	118	3.5	243	7.0	20	0.6	4	0.1
Female	1563	86.0	60	3.4	132	7.5	6	0.3	-	-
Not Recorded	-	-	-	-	-	-	1	-	-	-

Hull Excluded

The hospital Guidelines at the Royal Aberdeen Children's Hospital and Poole General Hospital and Alder Hey recommended admission for any child with a minor head injury, who had any history of loss of consciousness, even if fully recovered in the Accident & Emergency Unit. In other hospitals the Guidelines used were those formulated by a Group of Neurosurgeons for adults. The Casualty Medical Officer was free to use discretion in their application to children. Direct inspection of the records of Monklands Hospital, showed that the adult Guidelines were applied if the child was fully recovered, was alert and orientated, with no skull fracture and no neurological deficits.

The records at the Royal Aberdeen Children's Hospital for the same period were compared. The difference in the admission rate between Monklands and the Aberdeen Unit reflected the policy difference relating to admission following any history of loss of consciousness.

The difference in use of the Short Stay Ward 0.7% - 19% reflects the considerable variation in policy and resources for head injury management in the different hospitals. In Monklands Hospital, the Short Stay Ward is a full in patient facility under the supervision of the A & E Consultants. This is also the situation at Glasgow Royal Infirmary. Children are not normally admitted to the adult facility, but are transferred to the Royal Hospital for Sick Children at Yorkhill. In Poole District General Hospital there is a formal ward used for head injury observation. In Aberdeen and Alder Hey the children are admitted under the care of Paediatric Surgeons. These facts illustrate the difficulty in standardising procedures, and making meaningful comparisons. Now the Royal Hospital for Sick Children at Aberdeen has a dedicated short stay ward, but this was not the situation during the Study.

Age/Sex/Ward of Admission

The information is recorded in Table 7 (iv) for males and Table 7 (v) for females. Overall 89% of male and 86% of female patients were discharged home from Accident & Emergency Units. The larger number of males 20, transferred to Neurosurgery, 0.6% compared with 6 females 0.3%, reflect the more serious injury sustained by male children.

TABLE 7 (vii) CAUSE OF INJURY/DISPOSAL

	N	Home	SSW	PSW	NSU	ITU
		%	%	%	%	%
RTA	353	70	7	17	5	-
FALLS						
School	544	88	4	5	3	-
Home	2670	92	2	5	2	0.1
Play	1719	87	5	7	1	-
NAI	22	0	10	40	50	-
ASSAULT	570	50	18	27	5	-

The cause of injury, reflecting in some cases, the seriousness of the condition, would influence admission and the hospital unit of that admission. The results are shown table 7(vii).

The high rate of admission for cases of assault might not just reflect the severity of injury but the medicolegal implications. These would apply both to children assaulted in the absence of parents, but also the admission of children, possibly subject to abuse. The recommendations for holding a child in a place of safety, might result in the hospital being used for that purpose, whilst allowing further investigations to take place. When the Road Traffic Accident victims are examined the results are shown:

TABLE 7 (viii) RTA ADMISSION TO WARD/NSU

	%	% NSU
Occupants	15	3.0
Pedestrians	45	6.0
Cyclists	9	0.0

The results confirmed pedestrian and motor vehicle occupants (particularly if not restrained) as being the victims of more severe injury. There is a high rate of admission and transfer to Neurosurgery. In this group males dominated.

20 male children were admitted to neurosurgery, four to intensive care and 429 to other wards. Amongst the female children, 214 were admitted, six to neursourgery.

Mechanism of Injury/Disposal

When the mechanism of injury is related to disposal and the ward of admission it is surprising that 14% of falls to a soft surface were admitted. This may reflect anxiety regarding certain forms of child abuse, or difficulty in evaluating the history and mechanics of the event.

The results show that as expected falls on to hard flat surfaces had the highest transfer rates to Neurosurgery, followed by bumps on surfaces with similar characteristics or blunt blows.

Sharp contacts, whether falls or bumps, had the lowest admission rates, and only one child was transferred to Neurosurgery. Penetration of the skull is a rare occurrence with potential for serious, even fatal complications which warrant Neurosurgical transfer.

Disposal/Skull Fracture

It was the policy of all the Units to admit a child with a skull fracture unless the injury was more than 48 hours old and the patient asymptomatic. Only five children came into the delayed category. All were well but had a scalp swelling.

History of Loss of Consciousness/Coma

Children in the Study who had no history of loss of consciousness formed 93% of the total. The details of the disposal of those who had lost consciousness is shown Table 7 (x)

TABLE 7 (ix) MECHANISM OF INJURY / DISPOSAL

	Home		Short Stay Ward		Primary Surgical Ward		Neurosurgery Unit		Intensive Therapy Unit	
	N	%	N	%	N	%	N	%	N	%
Fall Soft	134	86.0	5	3.0	17	11.0	0	-	-	-
Fall Sharp	591	98.0	3	0.5	9	1.5	1	0.01	-	-
Fall Hard	2191	85.0	114	4.0	246	10.0	13	0.21	-	-
Bump Sharp	372	9494.0	7	2.0	17	4.0	-	0.0	-	-
Bump Hard	642	76.0	26	4.0	41	6.0	9	1.06	-	-
Struck Blunt Object	461	89.0	22	4.0	33	6.0	4	-	-	-
Struck Sharp Object	140	97.0	1	0.7	3	2.1	-		-	-

TABLE 7 (x) DISPOSAL. ADMISSION / HISTORY OF LOSS OF CONSCIOUSNESS

	No Loss of Consciousness 5581		< 5 Minutes Full Recovery 335		5 - 30 Minutes Full Recovery 35		30 - 60 Minute Full Recovery 3		60 + Not Reocvery 25	
	N	%	N	%	N	%	N	%	N	%
Home	5137	92.0	150	45.0	10	29.0	2	67.0	0	0
Short Stay Ward	105	2.0	76	23.0	6	27.0	-	-	4	17.0
Primary Surgical Ward	260	5.0	84	25.0	17	49.0	1	33.0	7	29.0
Neurosurgery Unit	77	1.0	24	7.0	2	6.0	-	-	10	42.0
Intensive Therapy Unit	1	-	1	0.3	-	-	-	-	2	8.0
Not Recorded	14									

7(b) Triage and Assessment in Paediatric Head Trauma

Contents:

Triage and Assessment of Traumatised Paediatric Patients

Systems of care for the traumatised patient, of whatever age, have in the past two decades, received recognition internationally, though effective implementation still presents many problems. A systematic approach from injury to definitive care is essential and is not controversial. The physical environment in which delivery is effected still produces discussion, controversy and practical problems for implementation in the United Kingdom. The National Trauma Centre is currently being evaluated. A specific contribution to the care of children has not yet been published. In the interim it is essential that the Units to which injured children are taken are competent to supply efficient and timely resuscitation, and provide access to specialist services with appropriate intensive care support. Wherever possible, paediatric surgeons should be involved at an early stage, and when available, should probably lead the resuscitation team, identifying priorities of care and effecting their implementation.

The advent of paramedics and the acceptance of the concept of triage should ensure optimum prehospital management, and arrival at a centre with a team already prepared for the reception of the victim. There should be clear identification of responsibilities. Initially this may be the A&E consultant or the most senior A & E doctor available.

The injury sustained may be suspected and diagnosed from the mechanism of injury. A child with multiple injury from a motor vehicle accident, or fall from a height may have both serious limb and torso trauma, with a severe primary brain injury. The less severely injured brain may be put at risk of secondary damage by inefficient management of other injuries, with secondary insults to the brain.

The majority of children presenting in the Accident & Emergency Department with head trauma will have sustained only minor injury. A minority will have sustained serious or potentially serious, brain trauma An even smaller number have multiple injuries which may threaten the integrity of the brain. . Management decisions in the Accident & Emergency Department are dependent upon an understanding of the pathophysiological changes, which occur at, or following the impact injury to the brain. Concepts of pathology have changed, as knowledge has accumulated from research world-wide. Information is of more than academic interest because "rational treatment is based on assumptions about the pathophysiological processes, which treatment aims to modify".

(a) Pathophysiology of Head Injury

Specific management of the head injured patient in the Accident & Emergency Department requires the knowledge of the pathogenesis and the pathophysiology of the insult.

(a) 1 Skull Injuries

Direct application of force to the skull may result in fracture, and the identification of the fracture is in general regarded as a measure of the degree of force. This is not necessarily so in children where weakness or vulnerability of the infant skull, is associated with fracture, with a lesser degree of trauma. The bone may protect underlying structures, or the same forces may damage the intracranial contents. Fractures are classified as:

a) vault fractures, these may be linear, stellate, comminuted or depressed.

(b) basal fractures.

(c) depressed fractures which may be simple or compound.

Basal fractures can present as CSF leaks from the nose or ears, racoon eyes, subconjunctival haemorrhages, Battle's sign and haemotympanum. The fracture is frequently not seen on conventional x-rays, hence the recognition of the physical manifestations as a guide to treatment. Such fractures are compound if they communicate with the front ethmoid or sphenoid air sinuses, nasal or auditory passages.

(a) 2 Intracranial Injuries

These may be focal, diffuse or a mixture of the two. There may be direct damage to brain substance in the form of lacerations and contusions, injuries visible to the naked eye.

Damage to blood vessels may produce haemorrhage or haematomas which are classified by their location as:

Focal Injury - Mass Lesions

1. Epidural (lying outside the dura) haematoma
2. Subdural haematoma
3. Intracerebral haematoma

These are associated with tears of the meninges and their blood vessels, both arterial and venous, and by damage to cerebral vessels. These lesions produce neurological problems not only by local damage, but by the mass occupation effect which results in brain shift/displacement, and herniation of the brain stem with ultimately irreversible compression. These injuries occur in 50% of patients admitted to hospital and result in 66% of the head injury deaths.

Diffuse Brain Injury

Diffuse brain injury is associated with widespread or global disruption of neurological function and, though it can be associated with focal injury, usually is not. There is widespread disturbance of both the function and structure of the brain. When sufficiently severe, diffuse brain injury produces coma by direct damage to the brain stem or cerebrum, and is not associated with a compressive effect. In the mild injuries it was even thought that this was purely functional, but there is some degree of physical damage with a limited degree of axonal injury. At the opposite end of the spectrum there is severe diffuse axonal injury, associated with extensive shearing and with a poor prognosis. In between are a number of categories of injury which can be related to clinical patterns in the Accident & Emergency Department. Accordingly treatment should be matched to the injury.

1. Mild concussion. This is associated with temporary disturbance of neurological function, without a loss of consciousness.
2. Classical cerebral concussion associated with temporary, but reversible neurological deficit associated with loss of consciousness, usually less than 6 hours in duration.
3. Diffuse axonal injury. This is associated with prolonged traumatic coma in excess of six hours not associated with mass lesions or compression. Such injury forms a continuum of increasingly severe injury, without precise boundary. However from a clinical point of view three further distinctions should be made

   1) Diffuse axonal injury, extending between six and 24 hours which may be associated with permanent neurological cognitive deficits.
   2) Moderate diffuse axonal injury with coma in excess of 24 hours without brain stem dysfunction, associated with a mortality in the region of 20% and significant morbidity in the survivors.
   3) Severe diffuse axonal injury associated with signs of brain stem dysfunction in patients comatose in excess of 24 hours, with a substantial mortality around 57%,and a morbidity associated with widespread damage to the cerebrum, and brain stem.

Diffuse brain stem injuries account for 40% of severely head injured patients, and although they constitute only 33% of head injury deaths, they are the most serious cause of persisting neurological disability in the survivors.

Patients with severe axonal injury are usually comatose from the time of the accident, and on arrival in the Accident & Emergency Department. Further deterioration may be an indication of a mass lesion. It is for this reason that CT scanning must be provided as soon as resuscitation allows. Early neurosurgical intervention, even with the presence of a low Glasgow Coma Score, may improve survival.

(b) Patterns of Head Trauma in Children

Primary Brain Injury

Occurs at the moment of impact, and produces irreversible damage. This may, depending on the mechanism, be diffuse axonal injury or focal brain damage or both.

Secondary Brain Injury

This may occur as a consequence of the initial trauma and result from

1) intracranial and

2) extracranial causes.

Secondary injury may be preventable at least following medical intervention.

Intracranial Injury

Results from

1) haemorrhage producing focal bleeding or haematoma
   - a) extradural -usually arterial in origin
   - b) subdural - acute, sub-acute or chronic.
   - c) intracerebral
2) raised intracranial pressure which may be associated with space occupation, brain swelling, and/or airway obstruction and convulsions.
3) cerebral hyperaemia/oedema
4) hypoxia, hypercarbia.
5) ischaemic changes due to reduced cerebral blood flow, which may be diffuse or focal.
6) respiratory depression. This may be due to airway obstruction, fits, drugs or alcohol. In serious injury brain stem damage may be the cause, and can follow

immediately after the impact, particularly in children, and may be associated with a period of apnoea and hypoxia.

Extracranial Causes

1) hypovolaemic shock secondary to blood loss.

2) hypoxia, airway obstruction, chest injuries and shock.

3) blood loss, circulatory volume and oxygen transport capacity is reduced.

Interplay between all these factors may produce a scenario of variable potential for mortality and morbidity. Paediatric head injury is not comparable with adult head injury. Within the group from birth - 15 years is a broad spectrum, whose patterns of injury shows a widely varying response, depending on whether the sutures are closed or not. The child in different age groups presents more difficulty in assessment, and management, in a highly emotive environment.

(c) Assessment of Severity of Injury

In the Emergency Room the assessment of the severity of brain injury can be established in the first minute by evaluating, three functions: level of consciousness, pupillary reaction and lateralising weakness of the extremities.

Abnormality of all three is highly suggestive of focal mass lesion requiring urgent surgery, whereas only the former may be abnormal in diffuse brain injury.

The Glasgow Coma Scale is used in assessing the level of consciousness. Its reproducibility ensures that a difference of two on the Glasgow Coma Scale signals a change in neurological status, a change of three indicating an enlarging haematoma requiring prompt treatment.

The Glasgow Coma Scale reflects brain dysfunction and in itself, contains considerable prognostic information. In the Emergency Room it is important that the assessment of the injury by this method be performed promptly and sequentially, to determine the degree of improvement or deterioration which is occurring. As other authors have emphasised however, the value of the Glasgow Coma Score must take account of other factors, drugs, hypotension and hypoxia. Anything that depresses neurological function will reduce the Glasgow Coma Score. Its more precise level should be assessed on the completion of full resuscitation, with the correction of hypotension and hypoxia, and at six hours.

Pupillary function can be affected by diffuse axonal injury. It is the value of the pupillary responses, reflecting third nerve compression, associated with transtentorial herniation which is of greatest value. In patients who have a high Glasgow Coma Score pupillary asymmetry may be manifest before the score falls. Unilateral or bilateral areflexia is generally a very unfavourable sign in adults with severe head injury, but may not be so in children. In relation to any assessment of the pupil, ocular injury, vitreous haemorrhage, retinal damage or transection of the optic nerves all must be considered.

Lateralisation of extremity weakness is determined by testing motor power in patients able to co-operate, or by observing asymmetry of movement in response to painful stimuli. The more severe the injury, the more difficult it is to assess lateralisation. Small differences in deterioration are important. It is true that lateralised weakness can occur with diffuse brain injuries, but is more likely to be a sign of focal swelling within a hemisphere, or of an intra-axial lesion such as an intracerebral haematoma.

(d) Pre-Hospital Care

In recent years the value of treating trauma victims as quickly as possible following the injury has led to the recognition of the need for assistance spanning simple first aid to sophisticated on site resuscitation. Better training has elevated standards of care for ambulance personnel, paramedic services and the attendance of hospital based surgical teams. Transport arrangements have been improved with the provision of well equipped and staffed ambulances. Helicopter lifts reduce delays in transit from site to emergency room, particularly from inaccessible areas, or where there is serious traffic congestion. Doctors or nurses from the receiving hospital may provide medical aid at the site of the accident in the United Kingdom. The principles of care are identical, though on site delivery may be restricted by environmental factors. It will be assumed that, with these limitations, the aims of resuscitation and initial treatment are the same as in the Emergency Department. The case for Trauma Centres is the ability to deliver optimal care on one site, thereby reducing the need for secondary transfer, and also to concentrate clinical expertise in a single location.

Decisions in the Accident & Emergency Department will inevitably be influenced by the care provided for the child from the moment of injury to the head, whether this is associated with cerebral insult or not. In childhood injury may occur in the absence of an adult observer. Consequently the cause and mechanism of injury may not be

reported to the first professional attendant, and secondary insult may have been inflicted due to compromise of the airway, aspiration of vomit or an unobserved epileptic fit.
In the minor, and apparently insignificant injury, it is the observation of vital signs, and the recording thereof which is valuable in subsequent assessment.
Helicopters have been used to reduce delays in the transport of seriously injured patients(Jeffries and Bristow 1991). Cusack and Robertson(1991) commented that in Baxt and Moody's(1987) review the helicopter actually delayed arrival at definitive care.
In 1991 there were 150 hospital based helicopter services in the United States of America and the numbers were increasing annually. The results of some have been reported. Comparisons have proved difficult, because of the case mix, and the absence of controlled series.
In the United Kingdom the number of helicopter services has increased. The HEMS London based service has been in action in urban areas. Moylan et al(1988) evaluated their Life Care Service, and showed benefits over road transport which were statistically significant. However only Schiller et al(1988) has reported well matched groups of patients assessed for trauma, severity, and standards of care. The road transport group fared better. Timescales were similar. The factors in these reports, which should be emphasised, is the quality of care offered in the air transport group. Staff of relative seniority and expertise frequently complements the expensive helicopter facility. They are able, and do, undertake resuscitative procedures not available in the road transport groups, which may be staffed by paramedics, or less experienced personnel. This was noted in the Baxt and Moody(1987) and Moylen(1988) reports.
None of the services reported specifically on the head injured patient, particularly in childhood. Cerebral injury however, was common in trauma cases, and was often the major cause of death. The benefits to the head injured patients will be related to the initial first aid, the speed of arrival of specialist help, and the quality of care offered, whether delivered by air or by road.
Once resuscitated and stable, delays will primarily affect the haematoma patient. The expertise of the escorting team should reduce secondary insults, whatever mode of transport is available.
The Royal College of Surgeons Working Party(1988) has added its voice to those of Robertson and Cusack(1991) in seeking more precise evaluation of helicopter services, particularly in relation to trauma centres. Within these studies no doubt will be assessed the benefits of this method of transport for head injured patients.

(e) Accident & Emergency Department - Priorities of Care

A AIRWAY

B BREATHING

C CIRCULATION

D DRUGS Anticonvulsants, Mannitol, antibiotics, tetanus prophylaxis.

E EXPOSURE Basically this is recognition of other injuries or complications. Prevention of further injury e.g. spine etc.

Clinical Examination

1. The two minute top to toe examination following the application of the priorities of care will identify the nature of injuries and their pattern.
2. History.
3. Observations:

   Pulse

   Pupillary responses, Glasgow Coma Score, ocular fundi, (this is particularly important in infants under the age of 1 year).

Resuscitation

Rapid and effective resuscitation can produce marked improvement, not only in the general state of the head injured patients, but also in cerebral function. Oxygen should be administered, at maximal concentration.

Charts

The condition of the child can be best monitored by the use of a formal observation chart

1. Glasgow Coma Scale - this is most widely used but has limitations under the age of 5 years.
2. The Adelaide Paediatric Coma Scale(Simpson and Reilly 1982) has been formulated for application to children. Other charts have been created for infants and children in the severe injury category by Morray et al(1981), Raimondi and Hirshauer(1984) and Hahn et al(1988) and others.
3. Injury Severity Scoring(Tepas et al 1988) should be applied, though it is more difficult in children than in adults, and requires some specialist knowledge.
4. Outcome Scores(Jennett and Bond 1975) are of value as a research tool and may be dependant on information recorded from the outset.

Investigations

Blood gas analysis and pulseoximetry on arrival, and during resuscitation has enhanced the quality of information during treatment.

Skull x-rays.

CT Scans.

MRI scans

Intracranial pressure monitoring.

Blood tests:

Arterial gas analysis Haemoglobin, blood film, haematological profile and coagulation screens and biochemical profile.

X-rays:

Lateral cervical spine, skull chest and pelvis in all cases of significant velocity injuries, should be routine, depending upon the patients condition.

Resuscitation Management

Ghajar J. and Hariri R J(1992) discussed the management of paediatric head injury, its pathophysiology and protocols for management. They emphasised, as have others, the deleterious effects of secondary brain injury resulting from intracranial hypotension and hypoxia.

The publications of Bullock and Teasdale(1991) and Gentleman et al(1986) and the Paediatric Life Support Group(1993) have provided detailed guidance from the initital care in Accident & Emergency Departments. The ATLS and PALS Courses teach practical skills.

An extensive exposition of Paediatric Life Support has recently been published by the Advanced Life Support Group(1993) covering all aspects of resuscitation including head, spinal and multiple injuries complicated by shock, hypoxia and hypvolaemia. The pathology and cause of raised intracranial pressure is addressed. Triage, the identification of serious injury primary and secondary surveys and investigation are all discussed. This panel of paediatric experts favours CT scanning in the seriously injured, but identifies the indications for skull xray.

(f) Guidelines - Advanced Life Support Group

Indications for Skull Xray

Loss of consciousness or amnesia at any time.
Neurological symptoms and signs. CSF or blood from nose (?) or ear.
Penetration or foreign body.
Scalp bruising or swelling.
Significant mechanism of injury.
Child under two years.
Difficulty assessing the patient.
Infants (abuse)
Inadequate history.
Alcohol intoxication or drugs.
The results of skull x-ray are then incorporated into guidelines for CT scanning combined with Glasgow Coma Scoring. In the first 4 years of life a Child Coma Score System is preferrable.
The management of the seriously injured with a Glasgow Coma Score under 8, included endotracheal intubation under anaesthesia, and hyperventilation.
The Group advised on neurosurgical consultation and referral.

Indications for CT Scanning

Coma Score	**Fracture**	**Condition**	**CT Scan**
15	No	No signs	No
15	Yes	No signs	No
15	Yes	Signs/symptoms	Yes
13-14	No	No signs	Consider
13-14	Yes	No signs	Yes
13-14	Yes/No	Signs/Symptoms	Yes

Measures to prevent a rise in intracranial pressure are given.

(g) Indication for Referral to a Neurosurgeon Include

Deteriorating level of consciousness.
Focal neurological signs.
Depressed fracture.
Penetrating injury.
Evidence of basal skull fracture?
Coma score less than 12.

The maintenance of optimal oxygenation by whatever means is required to prevent hypoxia.

Endotracheal Intubation

This should be carried out, with full anaesthetic expertise and using appropriate agents. Gagging and coughing are to be avoided, because of increase in venous congestion, and in intracranial pressure - CT Scan must follow, as coma scoring is affected by loss of verbalising ability

Hypovolaemic Shock

This should be treated vigorously and fluid infusion should not be restricted because of fear of precipitating cerebral oedema. The use of mannitol should be confined to a single dose. There are adverse effects if the product is used out with neurosurgical intensive care facilities. Urgent and expert attention should be given to the assessment of other injuries and their treatment.
The Paediatric Life Support Group(1993) emphasised the necessity to control convulsions and were in favour of the use of Mannitol in the face of developing raised intracranial pressure or blown pupil.

Transfer

The importance of full resuscitation and a well perfused child is emphasised, and takes priority over speed, whatever the temptation.
These authors advise consideration of other causes of coma as children frequently may have a recent history of minor head injury, which is not the cause of their condition.

(h) Accident & Emergency Department Resuscitation Transfer and Delay

The quality of care afforded to head injured patients of all ages in the Accident & Emergency Department was examined by Teasdale(1984). The pathology of traumatic head injury was defined as primary and irreversible, occurring at the moment of impact, and secondary due to complications.

In the pre-hospital setting and in the Accident & Emergency Department, provision of optimum conditions for recovery must be provided. The secondary damage occurs as a result of hypoxic/ischaemic damage. The causes can be both intracranial and extracranial or both. The intracranial causes include haematoma, infarction, raised intracranial pressure, obstruction to cerebrospinal fluid flow, vasospasm and epilepsy.

Extracranial causes include hypercapnoea with respiratory obstruction, anoxia and hypovolaemic shock and anaemia. Teasdale used two phrases which have emphasised the importance of post-trauma care. "The second accident" and "talked and died". 91% of patients examined at autopsy following transfer to neurosurgery were, in the majority, recorded as having avoidable factors either singly or in combination.

In the group who "talked and died" Reilly et al(1975)showed that 75% had an intracranial haematoma. Similar cases have been identified in the United States by Marshall, Toole and Bowers(1983). The factor which was identified by Galbraith(1976) in a review of haematoma cases, was delay. This was confirmed by subsequent reports by Mendelow et al(1979), Seelig(1981), Jeffreys and Jones(1981) and Teasdale, Galbraith and Murray(1982).

The responsibility of staff in Accident & Emergency Departments for deferred transfer was rarely confirmed . When such a patient was found in the A & E Department is was sometimes due to delayed attendance.

Failure of adequate observation, and early recognition of complications occurred in primary surgical wards more frequently than in Accident & Emergency Departments and emphasises the importance of the quality of care. The observation has been made that the admission of large numbers of patients following head injury, to areas not dedicated to trauma, or in the context of this Thesis, to paediatric head injury observation, dilutes the resources for those who are likely to need them most.

The individual factors leading to secondary brain injury were identified by Miller et al(1978)(Miller Becker and Ward 1977) as ischaemia, hypoxia, hypotension, anaemia and hypovolaemia. Miller and Becker(1982) and Kohi et al(1984) related extracranial insults and falling scores on the Glasgow Coma Scale.

The factors which influence outcome have been examined in the pre-hospital phase. Here Hoffman(1976) showed airway obstruction was rarely a factor. By contrast airway management during the intrahospital transfer was found to be significant by Yates(1979). The role of the A & E staff in protecting the airway is vital, and suggests that simple first aid measures, in the pre-hospital phase, were perhaps more effective in protecting the patient, than during hospital supervision.

Hypotension is rarely, if ever, due to head injury. The exceptions are infants who may have significant haemorrhage from head trauma. The anxiety generated by cerebral injury can result in other major injuries being underassessed, including fractures, and intra-abdominal and intrathoracic injury.

This raises the question of the sensitivity of the Accident & Emergency Department staff to the potential for deterioration, and for the recognition of markers which might alert the doctor to the risk factors.

Studies have been undertaken on the transfer of patients from A & E Departments to Neurosurgical Units. In this context the skull fracture as a marker, and the availability of CT Scanning in District General hospitals should enable selection to occur, before any secondary damage occurs as manifested by clinical signs of deterioration.

The incidence of haematoma is lower in children than adults, but a similar pattern may be seen as a result of cerebral swelling, which also requires neurosurgical expertise.

(i) Transportation of the Critically Injured Between Hospitals

Morgan(1990) reported that 49% of trauma deaths occured at the site of the accident and prior to arrival at hospital. The necessity to initiate resuscitative procedures, without delay, and to stabilise the patient was stated. It was his view that transport facilities are inadequate, and that telephone communications between the ambulance services and the Accident & Emergency Departments should be improved. The former should be adequately staffed at all times, with attendants sufficiently experienced to deal with seriously injured victims. Helicopter transfer from rural areas might reduce delays, but their efficiency in urban areas was not, in his view, established. The value of an appropriately trained doctor present during transportation was recognised.

In hospital the risks involved during investigative procedures were noted, including protection of the cervical spine, control of the airway and the correction of hypovolaemic shock. He referred to previous work undertaken by Bion et al(1985), Gentleman and Jennett(1981), Waddell et al(1975). He noted the work of Schiller et al(1988) and Moylan(1988) that suggested that road transport ambulance services were unfavourably equipped. There have been vast technical improvements in equipment and training in the U.K. ambulance services in the last five years. Morgan(1990)emphasised the importance of technical equipment, which facilitated monitoring to a standard available in the Intensive Care Unit. He concluded guidelines for transport facilities for the critically ill and injured would be educative. Guidance has now been formulated and published for head injured patients, including children by Bullock and Teasdale(1991), Paediatric Life Support Group(1993).

In view of the excellence of these papers no repetition will be made. Emphasis is placed however on various points of practical difficulty for the inexperienced casualty officer.

1. When and how to secure endotracheal intubation.
2. Vigorous treatment of hypovolaemia despite anxieties for producing cerebral oedema, and blood transfusion to correct anaemia.
3. Hyperventilation.
4. The use of Mannitol.
5. The control of seizures.

The "golden hour" identified for the efficient resuscitation and diagnosis of multiple injury patients is even more important for the patient with head injury. Management will influence not only the outcome, but the quality of survival. Responsibility of staff in Accident & Emergency Departments was identified by Richards(1986). The principals of resuscitation and protection of the brain were identified. The importance of full assessment prior to decisions, particularly radiology and transfer were noted.

The Accident & Emergency staff following the guidance should present to the Neurosurgical Unit a patient well oxygenated with a fully perfused brain.

In 1976 Horton identified the role of the Anaesthetist in the care of the head injured patient as important. Following the primary injury and its immediate effects, she emphasised the risk of secondary damage resulting from respiratory insufficiency, hypoxia, hypotension and anaemia. The use of anaesthetic agents and inappropriate drug administration is noted, as potential contributors to neurological deterioration and a poor prognosis. The priorities for care were clearly identified, and the means to achieve them recorded. The recognition of the potential of cervical spine injuries to result in further neurological damage was mentioned. Airway patency is essential.

Aggressive treatment of extracranial injury was emphasised, and the role of drugs as respiratory depressants noted. Addressing the problem of movement and transfer, the necessity of full resuscitation prior to this event was emphasised.

Miller et al(1978), examined the care given to 100 consecutive patients with head injury admitted to a Major Trauma Centre. It was found that potentially serious systemic insults to the brain were present in 44 cases. These included arterial hypotension anaemia 12, hypercarbia four, almost exclusively associated with multiple injuries. Hypoxia was seen in 30 patients, including several with brain injury alone. These systemic insults were believed to be associated with increased mortality and morbidity. As a result, Miller et al developed standardised protocols aimed at preventing additional brain insult in the severely injured patients prior to arrival in the Neurosurgical Service. This included rapid diagnosis, and treatment of intracranial mass lesions and the prevention of hypoxia, hypercarbia, fluid and electrolyte imbalance, anaemia and arterial hypotension. In 160 cases subjected to the advanced protocol it was claimed that the mortality was reduced by 10%-20%, without an equivalent increase in morbidity. The conclusions reached were that there may well have been a large number of patients who die, not because of primary brain injury, but because of secondary insults following trauma.

This persuaded the authors to look at the early treatment of head injury in the USA, between the time of the accident and arrival at the Trauma Centre, with a neurosurgical facility. This required examination of the care given at the scene of the accident, during transportation and in the Emergency Room. The most common cause of head injury in this group was vehicle accidents, 70%. Of the remaining patients there were 30 cases, 17 caused by falls and 13 by blows to the head. 47 patients were transported directly from the scene of the accident to the Emergency Room. Mean transport time was 35 minutes with most arriving in less than an hour. The other 53 were seen at another hospital, and subsequently transferred. Mean injury arrival time 2 hours 33 minutes, with more than 90% of the patients arriving within 4 hours.

A total of 57 patients had multiple injuries. Most of these were associated with vehicle accidents, 89%. Falls and blows to the head accounted for only 11% of patients with multiple injuries. The association between multiple injury and vehicle accident was statistically significant, $p < 0.01$.

The pre-admission systemic insults were identified as shock, hypoxia, hypercarbia and anaemia capable of producing adverse effects on the already damaged brain tissue. 13 patients were hypotensive, all of them had at least one major injury other than the head injury including four with a ruptured spleen. 12 patients had anaemia with a haematocrit less than 30% on arrival. 27 patients had hypoxia, one had hypercarbia and three had both. The distribution in relation to the cause of the injury was similar, there being only a small preponderance of patients with multiple injuries, suggesting that brain damage alone can produce hypoxia after injury, by airway obstruction and respiratory arrest or insufficiency.

Mass lesions were found intracranially much more commonly in patients subject to falls or assaults, 73%, than in patients involved in accidents with motor vehicles. Patients with mass lesions, as previously noted, are more seriously ill neurologically and have a higher mortality and morbidity than those with diffuse brain injury. The presence of a mass lesion is associated with a worse outcome. When the 40 patients with mass lesions are examined in detail none was found to be hypotensive or hypercarbic, but 16 had hypoxia or anaemia on admission. Of these only 25% made a good recovery. By contrast of 24 patients with mass lesions, but no systemic insult on admission 50% made a good recovery, or were only moderately disabled. There is no doubt that systemic insults did have a significant association with worst outcome.

Miller et al(1978) concluded that in more than half of the patients secondary insult occurred between the accident and admission to hospital. The recommendation was that any patient involved in a vehicle accident should be transported directly to a major trauma centre, with 24 hour neurosurgical services. On route there should be facilities for resuscitation, with blood volume expanders, airway control, support of fractures, the protection of the cervical spine, adequate airway control, and a supply of oxygen. Such facilities had been provided, as reported by Baxt and Moody(1987). In that report however, the protocol group was compared with a similar, but not identical group of patients. Neither the trauma scores or the reception facilities appeared in the report to be comparable.

Miller et al(1978) had postulated that in patients who have only a head injury, but who were shown to be hypoxic, that this might be due to the initial brain impact, followed by failure to reinflate the lungs following a period of apnoea. Respiratory depression with suppression of cough and gag reflexes, or even pulmonary complications can occur as a result of brain compression. Miller et al(1978) concluded that these patients too would benefit from transportation to a Neurosurgical facility directly.

The authors expressed concern that the problems that they had identified had been reported 20 years earlier by MacIver, Frew and Matheson(1958). They had little doubt that if the percentage of patients with head injury who arrive in hospital in a hypotensive and hypoxic state could be reduced, there would be improvement in mortality and morbidity. The failure to address the problems seriously diminish the effectiveness of intensive neurosurgical intervention, and critical care following arrival and admission to the hospital.

Miller et al(1978) clearly identified the Accident & Emergency Department and the Emergency Room and pre-hospital care staff as having the potential for effecting these improvements in the UK. As long as head injured patients require triage in Accident & Emergency Departments the Consultant will have responsibility for organising and supervising the services, and progressively enhancing their efficiency. Clearly there will be a significant commitment to education, and the need for experience and practical expertise at all levels of Accident & Emergency Department staff.

The responsibility of the Emergency Physician and the relationship with neurosurgical colleagues was the subject of an analysis by Teasdale(1984). Addressing the indictment that "head injuries are badly managed in A & E Departments and neurosurgeons are partly to blame", he looked at the problems. He acknowledged that despite the expenditure of resources, a large part of death and disability following head injury could have been avoided by better management. Primary and secondary insult to the brain was defined. The role of delay and the recognition of mass lesion had been identified. The A & E Departments were exonerated as a source of delay by Rose and Valtonen(1977) and Jennett and Carlin(1978). Failure to refer to neurosurgery patients under observation, has been reported by Jeffreys and Jones(1981). In a third of patients the mortality was due to unrecognised mass lesions. The other causes of potentially avoidable death were extracranial and had been reported earlier by Miller et al(1977) and Kohi et al(1984), and Gentleman and Jennett(1981). A third of avoidable deaths following head injury, occur in hospital In the past transfer to Neurosurgery was dependant upon the demonstration of deterioration. The advent of CT scanning prompted a policy which would identify patients at risk of complication, resulting in transfer before deterioration occurred. In practice initially, this did not come to fruition, because of limitation of neurosurgical beds and access to limited scanning facilities. Patients continued to be transferred using the earlier criteria. Recognition of these deficiencies resulted, as did further studies, in a more selective policy, and formulation of guidelines with improvement in transfer times. In the same period the risks of haematoma in adults had been identified. The value of skull x-ray as a marker of potential deterioration had been reported by Galbraith(1976), Mendelow(1984), Teasdale, Murray and Anderson(1990), Chan et al(1990), Godano et al(1992) and Servadei et al(1994) and its detection should reduce delay and prompt CT scanning.

Mayer and Walker(1985) following a review of 200 cases of severe had injury, with a mortality of 21.5% identified factors contributing to death. The majority had either severe primary brain injury or raised intracranial pressure, multiple trauma, or the presence of hypotension, hypercarbia or hypoxia. They observed that although primary brain injury dictates the outcome of severe cerebral trauma in children, many patients die due to secondary injury. In this respect the authors identified the Emergency Physician as having as important a role as the neurosurgeon. The care afforded in the pre-hospital situation, and the early recognition and treatment of other injuries in the emergency department, and the prevention of an elevation of intracranial pressure were observed to be contributing to improved outcome, and a fall in mortality. The systemic response to trauma has to be recognised and its adverse effects should be prevented, controlled or reversed.

The authors emphasise the different problems presented by the paediatric patient. Mass lesions are less common than in the adult, 30%, as opposed to up to 50%. The tendency of the child to develop raised intracranial pressure is greater.

200 children's records were reviewed, the criteria for severe head injury were met. They were subdivided into those with severe head injury in isolation, and those with associated multiple injury. Trauma scoring was utilised. In the latter group 29% had hypoxia, hypercarbia and hypotension. The mortality was 55% when any of these conditions were present, compared with 7.1% in those without. Only 26% had surgical mass lesions.

Factors affecting outcome were defined, and included a Glasgow Coma Score less than five, hypotension, hypercarbia and hypoxia. It was suggested that multiple trauma per se did not affect outcome, but only its untreated complications. These increased mortality rates in childhood by three times. The importance of the recognition and early aggressive treatment, in the emergency room,of the consequences of cerebral injury and multiple trauma was emphasised. Neurosurgical intervention is less frequently indicated, because of the lower incidence of space occupying lesions compared with adults. The avoidance of elevation of intracranial pressure complicating even severe primary cerebral damage, can produce a good outcome.

The authors emphasise the importance of close and ongoing co-operation between emergency physicians, surgeons and neurosurgeons in reducing the mortality in severely injured children.

Avoidable factors leading to the death of children following head injury were reported by Sharples et al(1990). These occurred both in hospital and in transit. A retrospective review of the records of children who died between 1979 and 1986 was undertaken, together with recovery of information from necropsy and Coroner's reports. 255 children died from head injury, a mortality of 5.3 per hundred thousand population per year. Half the children died before admission and were either dead at the site or had an injury severity score of 75. Before admission three children had "talked and (subsequently) died". One had aspirated and two had cerebral oedema.

25 children aspirated appreciable amounts of blood or vomit. These children had lower injury severity scores than the 42 who died without evidence of aspiration. It was concluded that hypoxia contributed to the death of these children, though no information is given on the nature of their injuries. Aspiration may be a terminal event or a consequence of manual cardiopulmonary resuscitation. After admission 130 children died, 25 were admitted directly to a Neurosurgical Unit and 68 were subsequently transferred from the primary hospital. 36 died at the primary hospital and one 14 days later at home. 21 children had talked and subsequently died. 11 had an intracranial haematoma. This was not diagnosed in life, or delay had occurred in diagnosis. Seven had respiratory arrests of which the cause was not identified. Three had cerebral swelling and no other abnormality. In all 26 children died of intracranial haeamtoma. Delay was a significant feature in the recognition, and subsequent transfer from the primary hospital. Seven children died despite neurosurgical intervention for their haematomas, suggesting that their condition had become irreversible. Five of the six children where delay was identified were in a "talk and die" category. 24 of the 26 haematoma patients had avoidable factors identified. 13 children died of haemorrhage, from undiagnosed intra abdominal injuries, and one had a pneumothorax. Two of these patients in addition had an undiagnosed intracranial haematoma.

31 children had an unexpected respiratory arrest, eight in transfer. Hypoxia was suspected as the cause. 121 potentially avoidable factors may have contributed to the death of these children. The authors noted the need for improved organisation for the management of head injuries which had been identified 10 years earlier. Yates(1977) had not found compromise of the airway to be a factor in pre-hospital transfer, but had noted it within the hospital. His study was not in children. In this series Sharples et al(1990) suggested that intubation and ventilation might have benefited those who aspirated, and those who developed brain swelling, after a lucid phase. Hazards of interhospital transfer had been identified and guidelines formulated, yet children died. The interventions known to reduce mortality and morbidity were not applied to many of these children.

No information was provided in the paper on the conditions under which these children were transferred, whether they were fully resuscitated prior to movement, or the quality, status and experience of their escorts?

This was a sobering report, clearly highlighting that knowledge, available for at least a decade, was not being applied in practice to the care of these head injured children. It seem unlikely that the basic fundamental equipment was unavailable, rather that the skill and expertise was either not available, or not called upon to resuscitate and support these young patients. It might have been fruitful to identify the sources and causes of error, so that remedies could be applied, but the paper did not do this.

The number of haematomas in children who "talked and died" in this series was higher than one might have anticipated. "Talk and die" in childhood is much more commonly associated with a non-surgical lesion.

Another point not made, was the possibility that factors which produced death in this group of children, might also be contributing to the morbidity of those who survived similar injuries , but who were not identified, as this was a post mortem study.

That the lessons have yet to be learned and applied was indicated recently. Munro and Laycock(1993) reported on the conditions under which 107 patients were transferred to the Wessex Neurosurgical Unit. The review included vital signs prior to transfer, drugs used, the availability of monitoring equipment, and the grade and specialty of the escorting doctor. The neurological status of the patient was recorded, as was blood pressure and pulseoximeter readings, and the outcome following neurosurgical care.

The groups of 107 patients included 12 children aged from 1 year to 12 years. 64 patients had sustained head injury, the others had suffered intracranial haemaorrhage of medical origin.

All the patients were escorted by anaesthetic staff, the majority senior house officers, but included consultants. The author suggested that the aim of the escorting medical officer was to maintain the patient in a state considered satisfactory in the Neurosurgical Intensive Unit. It was accepted that the difficulty in achieving this should not be underestimated.

The unacceptable features recorded were hypo and hypertension, and the absence of adequate equipment to monitor blood pressure. This was available infrequently, though ECG monitoring was available twice as often, and in the majority of patients.

Hypoxia was identified where pulseoximetry had not been available, and was caused by aspiration or endobronchial intubation. Oximetry correlated with good oxygenation in all but one patient. The use of anaesthetic agents was examined. There was concern regarding the use of relaxants alone, and the inadequacy of control associated with gagging and secondary hypertension.

In relation to the use of anaesthetic agents, the authors identified still the anxiety of doctors regarding the effects of drugs on neurological assessment. They emphasised yet again the benefits of intubation. The subsequent determinants of the patient's management were based on the initial history, and examinations and the findings on CT scanning. In the head injured patients the outcome correlated well with previous studies. There was a statistically significant relationship between adverse factors, and mortality, identified as hypoxia and abnormalities of blood pressure Difficulties with aerodynamic stabilisation and ventilation in transit were described by Braman et al(1987).

The authors concluded that the care of the neurosurgical emergencies still fall short of recommended standards. They identified both medical care, and monitoring devices as deficient. They suggested the development of local protocols to remedy the former. Mobile Intensive Care Units have been identified as enhancing safety in transportation by Bion et al(1985). A report(Treadwell and Mendelow 1994) from the Northern Region has advised on the management and movement of neurosurgical emergencies, in the area from which Sharples(1990) had eaerlier described paediatric mortality following head injury. Gentleman and Jennett's(1981) report and Gentleman's(1990) subsequent audit, whilst demonstrating some improvements in care afforded, still reported unacceptable deficiencies in the quality of care despite their recommendations a decade earlier

8. Results – Intracranial Haematoma

The Paediatric Study set out to identify the risk of haematoma, presenting in the Accident & Emergency Department. It was anticipated that in the majority of cases the diagnosis would be confirmed in the Neurosurgical Unit, or as it became more readily available by CT scanning in the receiving hospital.

In the Study 15 haematomas were identified - 0.25% or 1 in 400. All were confirmed on CT scans in the Neurosurgical Unit to which 155 children were transferred. None were detected by scanning in the receiving hospital.

TABLE 8 (i) HAEMATOMA TYPE

	N
Extradural	3
Subdural	5
Intracerebral	7

TABLE 8 (ii) HAEMATOMA/INJURY ARRIVAL INTERVAL

< 1 hour	10
1 - 2 hours	3
72 hours	2

When the time from injury to arrival in hospital is calculated, it is shown that two thirds of the children reached medical care within an hour. Three more came in under two hours. The two who arrived later proved to be subdural in origin.

Delay to neurosurgical intervention significantly increases the morbidity and mortality It is clear that delays, in the cases here identified did not occur, in the majority prior to arrival in hospital.

TABLE 8 (iii) HAEMATOMA TYPE/ARRIVAL INTERVAL

	E.D.H.	S.D.H.	IC.H.	ALL
< 1 hour	1	3	6	10
1 - 2 hours	2	-	1	3
72 hours	-	2	-	2

It is clear that the Doctor in the Accident & Emergency Department has a vital role to play, in ensuring that children at risk of developing a haematoma are managed efficiently and without delay. Intelligent anticipation, based on an understanding of the mechanism, causes and the clinical features, will ensure that the patient does not become the victim of a second accident. Rapid and safe transfer to neurosurgical care will follow.

HAEMATOMA/AGE

Haematoma occurs less frequently in childhood, than in adults.

TABLE 8 (iv) HAEMATOMA/AGE

	N
< 1 year	1
1 - 4 years	5
5 - 11 years	4
12 – 14 years	4
N/R	1

HAEMATOMA/SEX

14 of the haematomas occurred in male children. The association with skull fracture is shown TABLE 8 (v).

The three extradural haematomas occurred in the 4th, 8th and 10th years. All subdurals haematomas occurred in the 1st, 3rd and 14th years and seven intracerebral haematomas in the 2nd, 4th, 9th, and 11th years, two in the 12th and one in the 14th year. If the intracerebral haematoma is accepted as reflecting the severity of cerebral trauma, the cluster of cases between the 9th and 14th years appear to reflect the susceptibility of this age group to more serious head injury, particularly in the male child.

SCALP INJURY

There may be evidence of scalp injury, which may occur without underlying fracture.

TABLE 8 (vi) HAEMATOMA/SCALP INJURY

	N
No External Injury	2
Swelling only	6
Abrasions/contusion	3
Significant laceration < 5 cm	1
Superficial laceration > 5 cm	0
Galeal laceration < 5 cm	2
Galeal laceration > 5 cm	1

The features identified coincide with the scalp injury most frequently associated with skull fracture.

THE SITE OF INJURY

The site of injury to the skull may be associated with vulnerable underlying vascular channels. This is of greater significance for extradural bleeding.

TABLE 8 (vii) HAEMATOMA/SITE OF INJURY TO SKULL

	N	E.D.H.	S.D.H.	I.C.
None	1	-	1	-
Frontal	4	1	-	3
Temporal	1	1	-	-
Parietal	5	1	3	1
Occipital	1	-	1	1
More than one site	2	-	-	2

TABLE 8 (viii) HAEMATOMA/CAUSE OF INJURY

	N
Falls – all	7
Home	2
Sports/play	5
RTA – all	7
Occupants	1
Pedestrians	6
Cyclist	0
NAI	1
Assault	0

TABLE 8 (ix) HAEMATOMA TYPE/CAUSE

RTA	E.D.H.	S.D.H.	I.C.	ALL
Occupants	-	-	1	1
Pedestrians	1	2	3	6
NAI	-	-	1	1
Falls/home	-	2	-	2
Falls/play	2	1	2	5
Total	**3**	**5**	**7**	**15**

The mechanism of injury and the forces applied are relevant to the pathology which results. 14 haematomas resulted from blunt trauma.

TABLE 8 (x) HAEMATOMA/MECHANISM OF INJURY

	N
Falls soft	0
Falls sharp	1
Falls hard flat	10
Bump sharp	0
Bump hard flat	3
Struck blunt	1
Struck sharp	0

The association with blunt impact on a hard surface is the commonest mechanism. Sharp trauma was unusual in the UK and can easily be overlooked by the examining doctor. The diagnosis require a relevant history, which may not be easy in a child. A sharp implement or glass may penetrate the skull. Its most vulnerable sites are the temple, orbit and soft palate. The child may fall on to a sharp object, or it may be propelled on to a sharp object, or be the victim of an assault. Intracranial haemorrhage results. Pseudoaneurysm may follow. Deterioration may occur dramatically if the instrument is withdrawn. Infection may further complicate the condition.

HISTORY OF LOSS OF CONSCIOUSNESS

The extradural haematoma is classically associated with a lucid interval in adults. It is said to be less common in children though it does occur. The phenomenon of talk and die is not in children confined to extradural haematoma. It is valuable to the doctor as the child may be able to given an account of the accident. It provides a base line for monitoring progress, against which any deterioration can be measured. The children in the Study were conscious in six cases on arrival in the Accident & Emergency Department.

TABLE 8 (xi) HAEMATOMA/LOSS OF CONSCIOUSNESS

	N
No history of loss of consciousness	6
< 60 minutes	8
N/R	1

The Adelaide Children's Coma Scale was used to monitor conscious level. It was noted that once admission or transfer was made the Glasgow Coma Scale was substituted.

TABLE 8 (xii) HAEMATOMA/ADELAIDE COMA SCALE

	N
Ocular Response	
None	5
To pain	5
To speech	2
Spontaneous	1
Verbal Response	
None	5
Cries	4
Vocal sounds	3
Words	1
Orientated	2
Motor Response	
None	0
External pain	4
Spastic flex	2
Normal flexion	3
Localises	3
Obeys	3

The paediatric modification of the Glasgow Coma Scale, takes account of age. The score is maximal between 11 and 14. The responses seen in children with a haematoma show from the results observed in the Accident & Emergency Departments that in more than half the children serious and irreversible changes had not occurred.

Children with intracranial haematoma might be expected to have headache and vomiting. Those whose conscious level was impaired might not be able to complain of headache at the time of the examination. It was documented in two cases with an extradural haematoma and one with an intracerebral. Vomiting was documented only in three cases, one of which was extradural.

PYREXIA

A normal body temperature was recorded in 14 children and not recorded at all in one.

PUPILLARY RESPONSES

These were normal in 13 children, and abnormal only in the two subdurals presenting after 72 hours.

HAEMATOMA/FOCAL NEUROLOGY

In 10 children no focal neurological signs were recorded at the initial examination in the Accident & Emergency Department.

TABLE 8 (xiii) HAEMATOMA/FOCAL NEUROLOGY

	N
None	10
Hemiparesis	2
Hemiplegia	2
N/R	1

TABLE 8 (xiv) HAEMATOMA/FOCAL NEUROLOGICAL SIGNS

	E.D.H.	S.D.H.	I.C.H.	ALL
None	3	4	3	10
Hemiparesis	-	1	1	2
Hemiplegia	-	-	2	2
N/R	-	-	1	1
Total				**15**

The relationship of the haematoma type and the presence of neurological deficits is shown. It is of interest that the extradural haematomas presented without any evidence of neurological deficit. The repaid arrival in the Accident & emergency Department within an hour of the injury, without evidence of deterioration emphasises the potential for early detection. As the majority of paediatric head injuries will fulfil these criteria, it is essential to identify other characteristics which alert the Accident & Emergency Staff to the potential for deterioration. These include consideration of the cause and mechanism, the site of injury, clinical and radiological evidence and any history of loss of consciousness. Irritability is a feature which is not recordable on the Glasgow Coma Scale, decrease in level of consciousness and scalp features need to be considered.

TABLE 8 (xv) HAEMATOMA/PULSE

60 - 79	0
80 - 99	4
100 – 119	4
120 – 180	3
N/R	4

Bradycardia characteristic of raised intracranial pressure, and compression was not seen in any of the cases in the Accident & Emergency Departments. A further indication of arrival before serious deterioration had occurred.

Children who have sustained head trauma may have an epileptic fit. Children are more likely than adults to suffer this complication. A single fit following immediately after the impact is less sinister than that which occurs later. It may be a manifestation of focal pathology, and is inevitably seen as a deterioration in conscious level. When it occurs in the Accident & Emergency Department, prompt control is indicated. In this series of haematomas only one child had a focal fit.

HAEMATOMA/SKULL FRACTURE

Haematoma is frequently associated with a skull fracture. In this Study there were 15 haematomas of which 9 were associated with a skull fracture, i.e. 9% of the fractures had a haematoma.

TABLE 8 (xvi) HAEMATOMA/SKULL FRACTURE

	E.D.H.	S.D.H.	I.C.H.
No #	0	3	3
Linear	3	-	1
Depressed	-	-	2
Fracture	-	-	1
> 1 site	-	1	1
	4, 8, 10 years	1, 2, 4,, 14 years	2, 4, 9, 1 years

TABLE 8 (xvii) HAEMATOMA/SKULL FRACTURE

	Haematoma	None
No Fracture	6	5885
Fracture	9	93

9% of the 102 fractures had haematoma

40% of haematomas had no fracture

60% of haematomas had a fracture

One haematoma occurred with a base of skull fracture

TABLE 8 (xviii) HAEMATOMA

Total	15
Incidence Paediatric	0.25% (adult 1-2%)
Average in A&E Department	1 per annum
2500 Head Injuries	

Haematoma is shown to be a rare condition in the Accident & Emergency Department. The incidence of acute traumatic intracranial haematoma in the emergency Department shown in this series in children is in sharp contrast to the incidence in Neurosurgical Units in the UK, Europe and USA, where the incidence ranges from 19% - 46%. The opportunities afforded to casualty officers to gain experience in the recognition and management of this condition is clearly limited. It is essential therefore that teaching and training addresses the problem. The use of guidelines and protocols will have some value. The availability of more experienced doctors to advise and support, particularly out of hours will ensure that avoidable delays to definitive care do not arise.

The knowledge of the early presentation of patients with haematoma raises an issue with regard to the investigation of such cases. Skull x-rays are valuable in alerting staff to the risk of haematoma. CT scanning is essential to confirm. Whether this should occur in the receiving hospital with the potential for delay, particularly out of hours, or whether the scan should be undertaken in the Neurosurgical Unit requires strict local protocols, firm guidance and secure transfer arrangements.

HAEMATOMA RISK

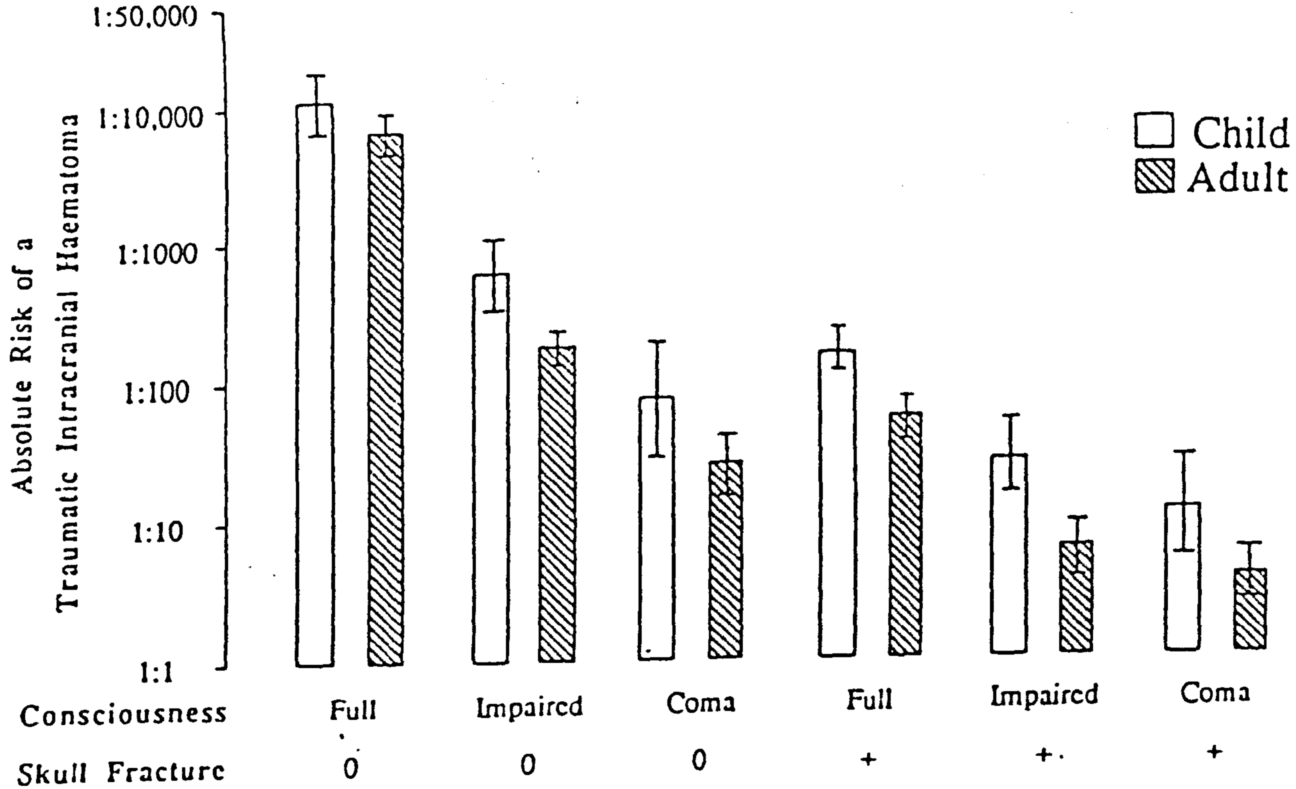
This Author contributed data which was incorporated in the analysis of Teasdale, Murray and Anderson et al(1990). The result was a comparison of the haematoma risk in children compared to adults, and recommendations for amendments to the adult Guidelines to benefit children.

HAEMATOMA RISK/SKULL FRACTURE/ ADULTS/CHILDREN

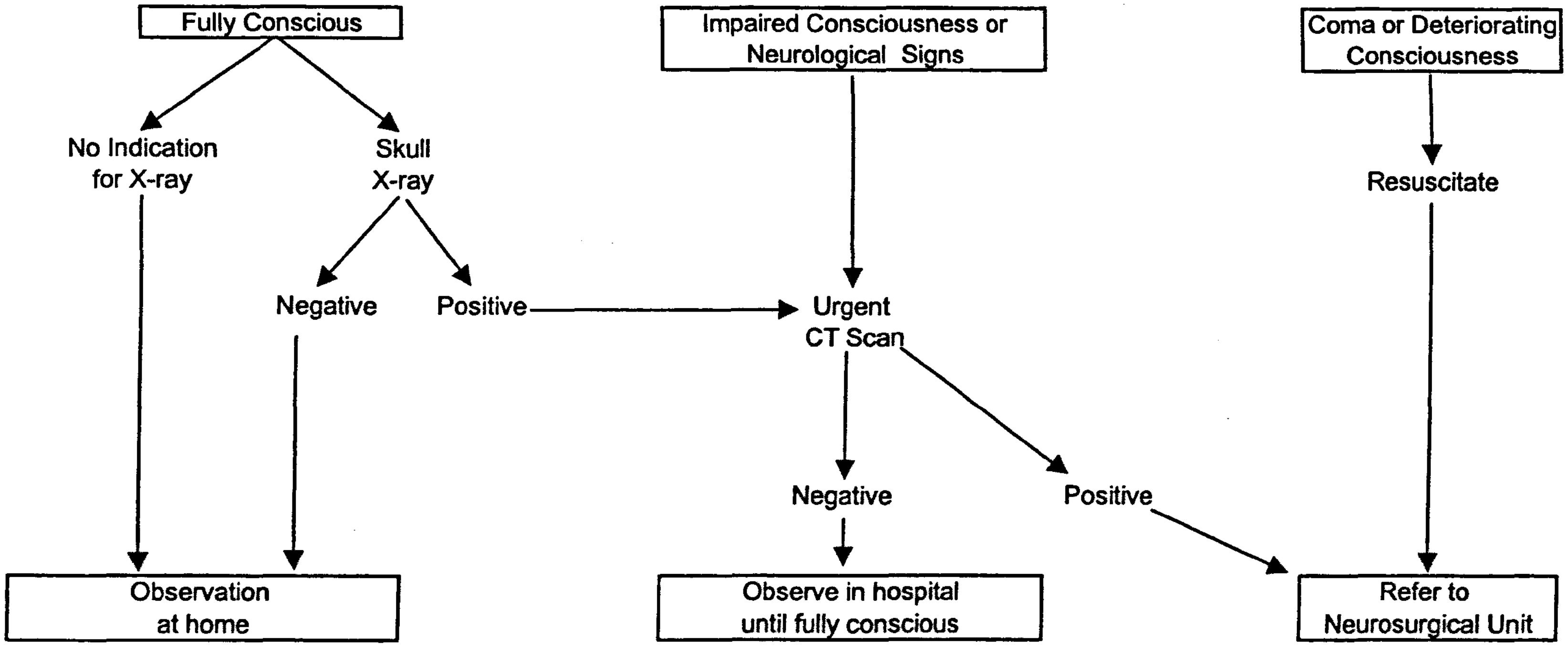
	Estimated Number attending hospital/year/one million population	Estimated Annual incidence of a Traumatic Intracranial Haematoma per million Total population
ADULTS		
No skull fracture		
Fully conscious		
No altered consciousness	8800	0.7
History of altered consciousness	1900	0.7
Impaired consciousness	630	3.5
Coma	75	2.8
Skull fracture		
Fully conscious	150	3.3
Impaired consciousness	49	9.6
Coma	46	12.7
CHILDREN		
No skull fracture		
Fully conscious	8100	0.6
Impaired consciousness	280	0.5
Coma	26	0.4
Skull fracture		
Fully conscious	110	0.7
Impaired consciousness	19	0.8
Coma	12	1.0

From Teasdale et al 1990

RISK OF INTRACRANIAL HAEMATOMA IN ADULTS AND CHILDREN



Management of Head Injuries in a General Hospital with a CT Scanner



8. Haematoma – Univarate Analysis

Risk of Haematoma/Skull Fracture

The probability of a child presenting with a head injury resulting in a haematoma was found to be dependent on the presence of a skull fracture, (chi-squared = 278.34, df=1, $p<0.0005$).

The number of haematomas by the presence of a skull fracture and the expected number under a hypothesis of independent are shown in Table 8(SA)(i).

TABLE 8(SA)(i): Expected and Observed number of haematomas by skull fracture (Under H0: haematoma is independent of skull fracture)

			SKULL #		
			No	**Yes**	**Total**
HAEMATOMA	**No**	Count	5848.0	102.0	5950
		Expected Count	5839.3	110.7	5950
	Yes	Count	6.0	9.0	15
		Expected Count	14.7	0.3	15
Total		Count	5854.0	111.0	5965
		Expected Count	5854.0	111.0	5965

14 of 15 intracranial haematomas occurred in male children.

Skull fracture had an odds ratio of 86.0 (CI=(30.05, 246.10)) of resulting in a haematoma. Therefore a child with a skull fracture is more than 80 times as likely to result in a haematoma as a child without a skull fracture.

However, when children under one year are considered separately, they do not show an increased risk of intracranial haematoma, even when a skull fracture is confirmed (chi squared=0.57, df=1, $p<0.811$). Children of this age, with a skull fracture have an odds ratio of 1.0 (CI=(0.99, 1.00) of having a haematoma.

Risk of Haematoma/Unconsciousness

Six of 15 children in the Monklands Study had no history of loss of consciousness or cerebral impairment in the A&E Department and subsequently were found to have a haematoma. This represents six out of almost 6,000 children.

9. Results - Epilepsy

TABLE 19(i) INCIDENCE OF EPILEPSY

	N	%
None	5937	99.10
New Focal	3	0.05
New General	15	0.25

TABLE 9(ii) SEIZURES IN KNOWN EPILEPTICS

	N	%
Known Epileptic	38	-
Focal	2	0.03
General	4	0.06
No Fit	25	0.40
Febrile	7	0.10

Information relating to convulsions was recorded in 5962 children. In 99.1% no such phenomena was recorded at the time of the injury, prior to or following arrival in the Accident & Emergency Department. In 18 children no previous history of a convulsion was obtained from parent or escort, but these children had fits recorded following the trauma, i.e. post traumatic epilepsy.

In all the Accident & Emergency departments the protocols for controlling seizures in the immediate situation was similar. Intravenous or rectal diazepam was the first choice. Maintenance of control was with an infusion of phenytoin. When the child required endotracheal intubation, muscle relaxants were used, and ventilation maintained artificially. The importance of suppressing central seizure activity, was not always appreciated. In the paralysed child paraldehyde was used once, as was midazolam.

Those patients requiring urgent neurosurgical interventions were transferred to neuroanaesthetists and neurointensive care.

Mannitol was used, where a mass lesion was suspected as the cause of the seizure prior to, and during transport.

The abused infants with mass lesions were at particular risk of seizure.

Doctors in the Accident & Emergency Department are called upon to treat epileptic patients, who have had a fit often in a public place. They are brought to hospital for assessment. Usually an accompanying adult or escort will confirm that the child is in fact an epileptic. There may be evidence in addition of an injury to the head, sustained as a result of a fit/fall. The assessment of the head injury, follows a regular pattern, skull xrays to exclude a fracture, and a period of observation, through the post ictal state until the patient resumes their normal neurological status. During this period the possible risk of intracranial bleeding or other complications of the head trauma are assessed and the appropriate procedures followed, including CT Scanning.

10. Results – Outcome

The large numbers of children who attend hospital following head injury conceal a minority who have moderate to severe injury, who require resuscitation and treatment for other injuries. These may be major and complicate neurosurgical care. The numbers who come into these categories are shown.

TABLE 10(i) INJURIES IN THE HEAD INJURED PATIENT

Other injuries occurred in 167 patients.

Total	N	%	% 167
1. Arm	66	1.1	40.0
2. Leg	47	0.8	28.0
3. Arm and Leg	19	0.3	11.0
4. Trunk	22	0.4	13.0
5. Trunk, Arm and Leg	9	0.2	5.0
6. Uncoded	4	0.1	2.0
Total	167	2.8	-

The effects of serious injury on the brain and on the outcome has been discussed in the relevant section, covering secondary insults to the injured brain.

TABLE 10 (ii) SERIOUS INJURIES IN THE ACCIDENT & EMERGENCY DEPARTMENT

	%
Head injury alone	97.0
Other injury of which	2.3
Minor	69.0
Moderate	21.0
Serious / Critical including	9.0
Multiple injury	3.0

The outcome at one month is shown:

TABLE 10 (iii) OUTCOME FOR HEAD INJURY IN A&E DEPARTMENTS

	%
Good	98
Disability	0.2
Death	0.1
Unknown	1.6

0.1% died, 0.2% had serious disability, and in 1.6% the level of disability in future was not known.

The other injuries which occurred in head injured children are shown:

TABLE 10 (iv) SEVERITY OF HEAD INJURY

	N	%
Minor	5822	97
Moderate	51	0.9
Serious	7	0.1
Severe	6	0.1
Critical	2	-
Non – survivable	3	

Amongst the Minor Head Injuries, as recorded there were 49 fractures reported by a Radiologist. The minor groups concealed 11 false negative skull fractures, and six false positives, as interpreted by a casualty officer.

Moderate Head Injuries

Casualty Officers reported 51 moderate head injuries. 32 skull fractures were reported by a Radiologist. There were eight false positives diagnosed by Casualty Officers and two false negatives. Within the group of moderately severe head injuries there were two intracranial haematomas.

TABLE 10 (vi) HEAD INJURY/WITH OTHER INJURIES

	N	% of Total	%
Minor	115	1.9	69.0
Moderate	37	0.6	22.0
Serious	12	0.2	7.0
Severe	1	-	0.6
Critical	1	-	0.6
Non – survivable	1	-	0.6
Total	**167**		

Cross tabulation shows 106 patients had the same variables. The three patients with the most serious injuries had multiple trauma.

11. Results – Attendance Patterns

TABLE 11(i) 1987 NEW PATIENT ATTENDANCE MONKLANDS DISTRICT GENERAL HOSPITAL

49,585
4,594 Head Injured Patients
2185 Head Injured Children

JANUARY	3469
FEBRUARY	3702
MARCH	4138
APRIL	4246
MAY	4552
JUNE	4330
JULY	4112
AUGUST	5411
SEPTEMBER	4372
OCTOBER	4084
NOVEMBER	4104
DECEMBER	3998

TABLE: 11(ii) MONTH OF ARRIVAL/ALL HOSPITALS/SEX

	Male	Female	Total	%
January	117	76	193	9.2
February	84	46	130	6.2
March	89	48	137	6.5
April	136	78	214	10.2
May	143	57	200	9.5
June	158	58	216	10.3
July	132	74	206	9.8
August	185	83	268	12.8
September	115	57	172	8.2
October	82	50	132	6.3
November	88	44	132	6.3
December	65	36	101	4.8
Total	**1394**	**707**	**2101**	**100.0**

FIGURE 11 (a) MONTH OF ARRIVAL/MONKLANDS/SEX

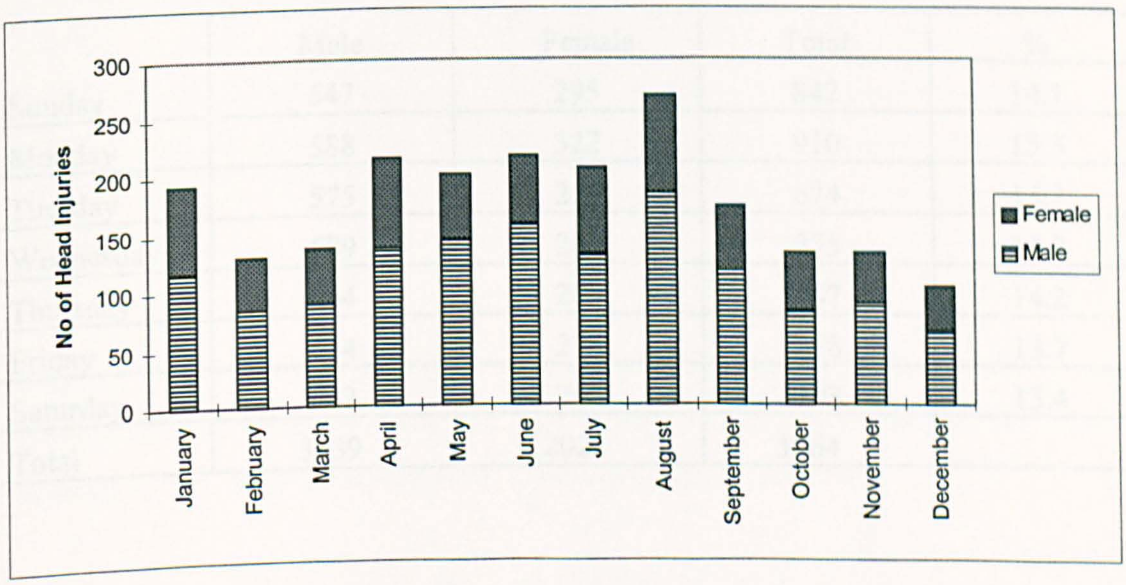
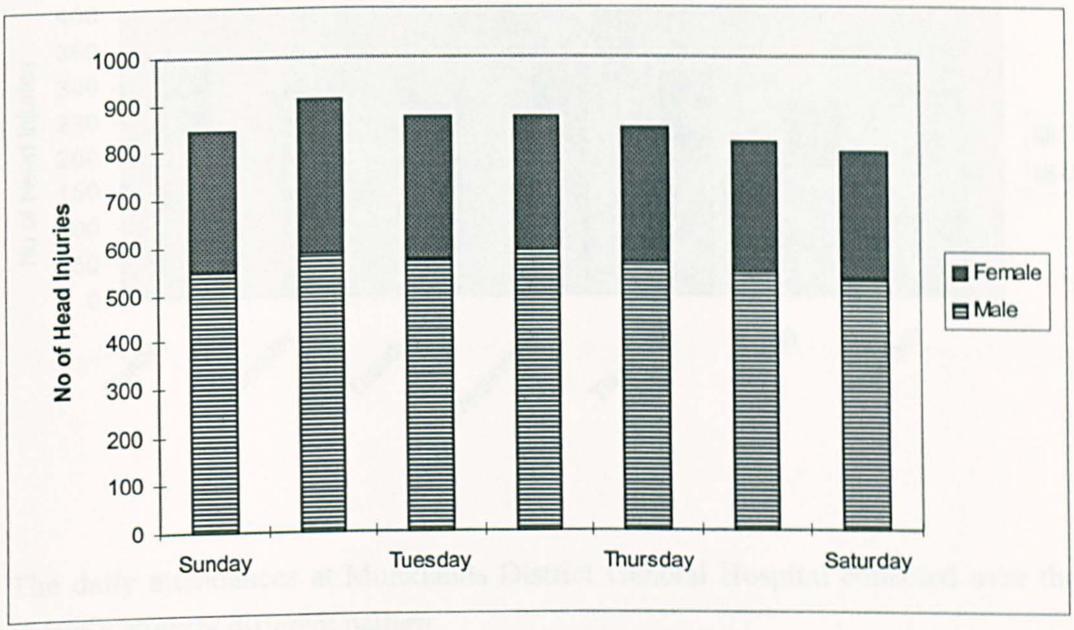


FIGURE 11(b) DAY OF ARRIVAL ALL HOSPITALS/SEX



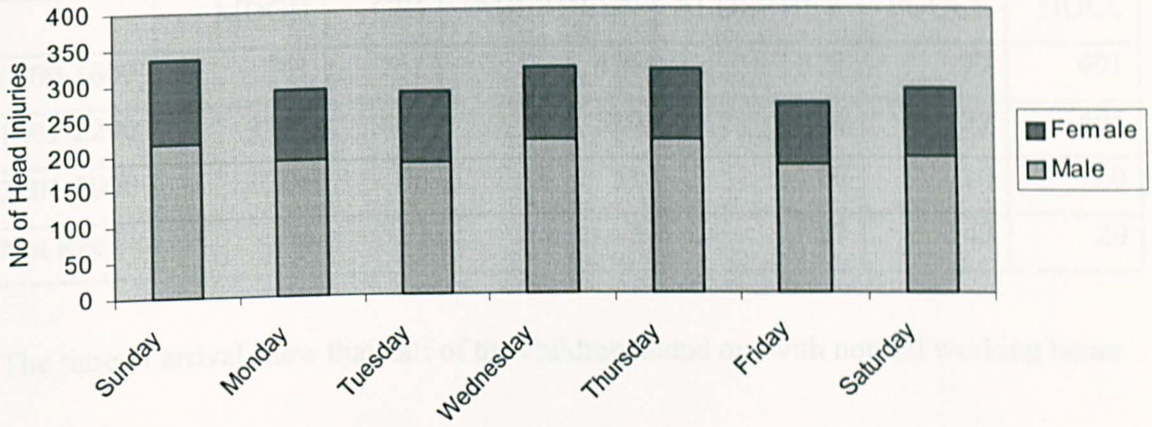
DAY OF ARRIVAL

When the days of arrival are examined for all hospitals, Monday is the busiest day.

TABLE: 11(iii) DAY OF ARRIVAL/ ALL HOSPITALS/SEX

	Male	Female	Total	%
Sunday	547	295	842	14.1
Monday	588	322	910	15.3
Tuesday	575	299	874	14.7
Wednesday	589	286	875	14.7
Thursday	564	283	847	14.2
Friday	544	274	818	13.7
Saturday	532	266	798	13.4
Total	3939	2025	5964	

FIGURE 11(c) DAY OF ARRIVAL MONKLANDS HOSPITAL/SEX



The daily attendances at Monklands District General Hospital collected over the year shows a slightly different pattern.

TABLE 11(iv) MONKLANDS HOSPITAL DAY OF ARRIVAL/SEX

	MALE	FEMALE	TOTAL	%
Sunday	216	120	336	16.0
Monday	190	102	292	13.9
Tuesday	186	98	284	13.5
Wednesday	216	102	318	15.1
Thursday	216	100	316	15.0
Friday	179	89	268	12.8
Saturday	191	96	287	13.7
Total	1394	707	2101	

The ratio of male to female children is maintained over the days of the week. Sunday, Wednesday and Thursday being the busiest day. The largest number attended on Sundays – 16%, with Wednesday and Thursday following at 15%.

TABLE 11(v) TIME OF ARRIVAL/ALL HOSPITAL

	MDGH	GRI	ABERDEEN	ALDERHEY	POOLE	HULL
0900-1600	761	261	434	328	97	401
1601-2200	1131	486	510	496	137	405
2201-0900	206	66	51	80	10	40
Not Rec.	6	2	5	27	43	20

The time of arrival show that half of the children attend out with normal working hours.

FIGURE 11(d) TIME OF ARRIVAL BY HOSPITAL

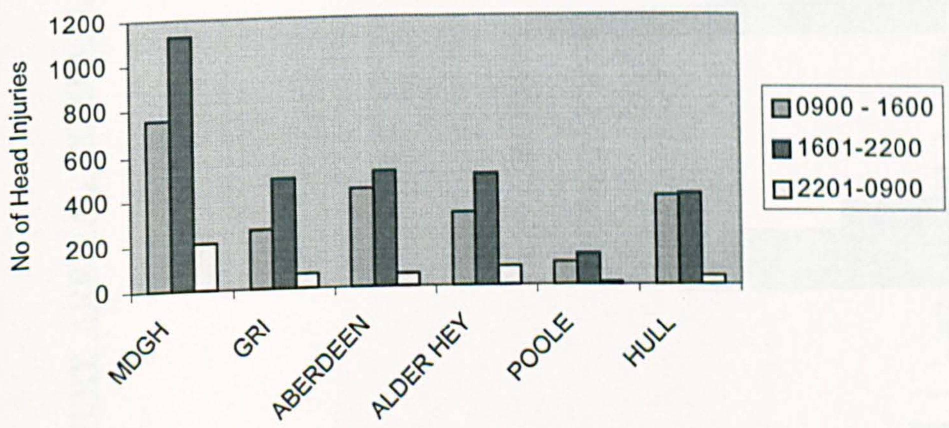


FIGURE 11(e) INJURY ARRIVAL INTERVAL/HOSPITAL

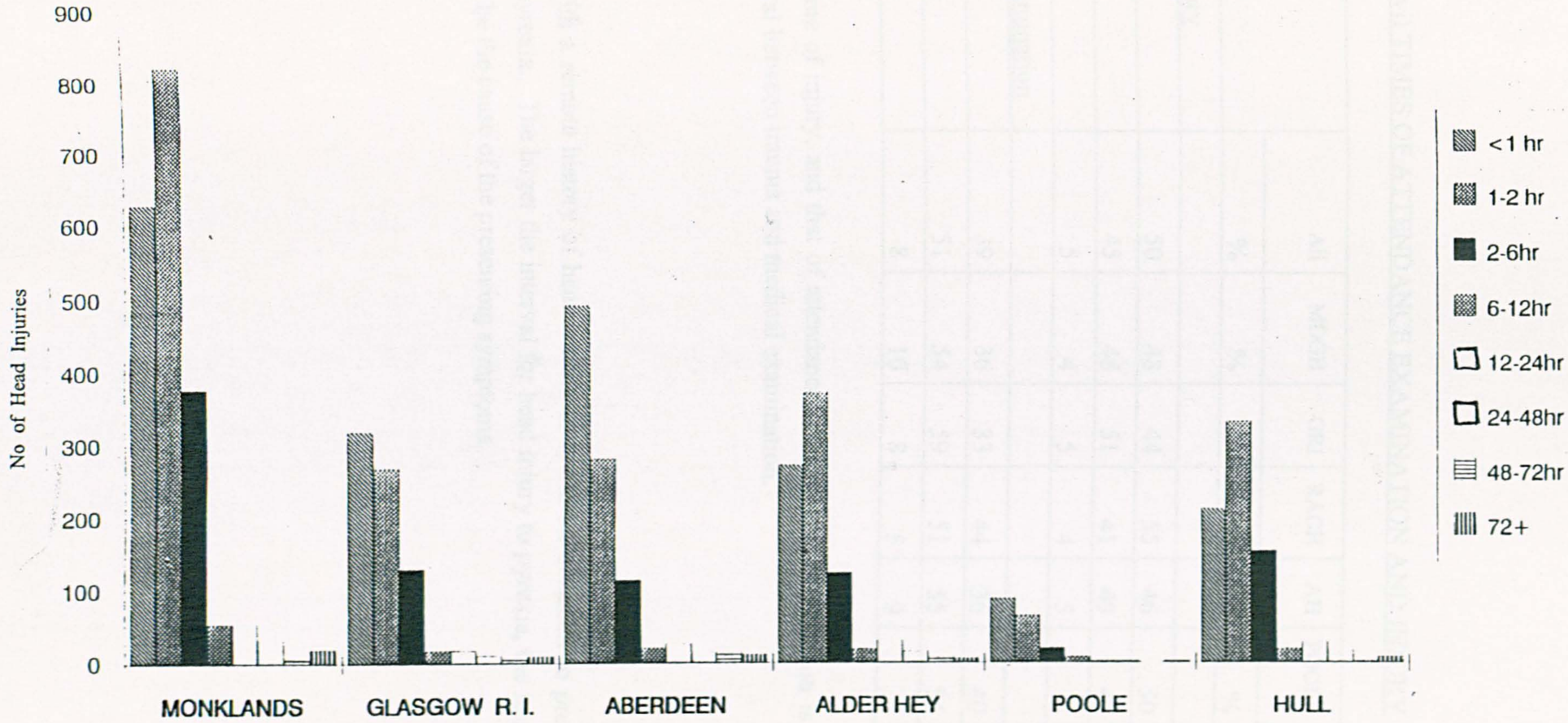


TABLE 11(vi) TIMES OF ATTENDANCE EXAMINATION AND INJURY

	All	MDGH	GRI	RACH	AH	POOLE	HULL
	%	%	%	%	%	%	%
Time of Injury							
0901-1600	50	48	44	55	46	50	59
1601-2200	45	48	51	41	49	42	33
2201-0900	5	4	5	4	5	8	8
Time of Examination							
0901-1600	39	36	33	44	36	40	47
1601-2200	51	54	59	51	55	56	48
2201-0900	8	10	8	5	9	4	5

When the time of injury, and that of attendance is compared, information is provided on the interval between trauma and medical examination.

Pyrexia

The child with a remote history of head trauma is found on occasion to present only because of pyrexia. The larger the interval for head injury to pyrexia, the less likely is the injury to be the cause of the presenting symptoms.

TABLE 11 (vii) PYREXIA / DELAY / INJURY / (ARRIVAL INTERVAL)

	< 1 Hour N	%	1-2 Hours N	%	2-6 Hours N	%	6-12 Hours N	%	12-24 Hours N	%	> 24 Hours N	%	N/R N	%	
< 37°C	1832	-	1958	-	822	-	106	82.8	124	81.6	224	83.9	350	89.5	
37° - 39°C	49	2.6	54	2.7	28	3.2	8	6.3	12	7.9	10	3.7	13	3.3	(a)
No Cause															
37° - 39°	11	0.6	23	1.1	16	1.8	14	10.8	16	10.5	32	12	28	7.2	(b)
Cause															
40° >	1	0.1	-	-	-	-	-	-	-	-	1	0.4	-	-	(b)

(a) No cause identified
(b) Cause identified

12. Results – Care of Children in Accident & Emergency Departments

A comparison between facilities and resources in the Royal Aberdeen Childrens Hospital and Monklands District General Hospital.

	ROYAL ABERDEEN CHILDRENS HOSPITAL	MONKLANDS DISTRICT GENERAL HOSPITAL
Environment	Purpose built children's unit appropriately designed.	No architectural facilities for children.
	Exclusive use of all A&E Department Areas.	
	Dedicated Waiting area, appropriately furnished	Common waiting area, furnished for adults
Resuscitiation	Dedicated facility. Appropriate equipment. Paediatric medication tailored to age	Shared open adult Resuscitation Room. Dual instrumentation and drugs
	Multidisclinary paediatric back up available	Anaesthetic back up for A&E
Staffing Nursing	Paediatric Trained Nursing Staff	Only one paediatric nurse 38 hours per week
Medical	Experienced paediatric SHO's dealing exclusively with children under the supervision of a Paediatric Surgeon. No competing demands	Only one SHO had paediatric training

Admission	Dedicated Wards under Consultant Paediatric Surgeon	Shared adult facility under adult trained A&E Consultants
	Facilities for parents to stay with child	No parent facilities
	Beds adjacent to A&E Unit	Beds in adult short stay ward
	Paediatricans on site for a variety of paediatric specialties	Paediatric physicians on site
	Transfer to Neurosurgery On Campus	Transfer to Childrens Hospital or Neursurgical Unit, 12 miles
	CT Available	No CT at time of study
Imaging	Knowledge of Paediatric X-ray Features	Limited knowledge of X-ray features in children

The Study did not attempt to assess the quality of care delivered, which would have required extensive additional resources.

TABLE: 12 (i) UTILISATION OF SKULL XRAYS

	MDGH	RACH
Not x-rayed	321	553
Number of xrays	700	447
% of usage	68%	45%

TABLE 12 (ii) MDGH/SENIOR HOUSE OFFICERS

Code	No. PTS.	NXR	% OF XRAY	% YIELD
12	100	52	52	5.7
13	90	144	49	6.8
14	74	46	62	2.2
15	113	81	72	2.5
16	88	79	90	2.5
17	113	84	74	1.2
18	84	71	85	7.0
19	88	70	45	7.5

TABLE 12 (iii) RACH/SENIOR HOUSE OFFICERS

Code	N	%XRAY	%YIELD
01	434	55	2.5
02	276	36	6.0
03	296	36	5.0

The xray requests made by the Senior House Officer at Aberdeen were interesting. The most experienced doctor Code 01 saw the largest number of children, but ordered proportionately more rays, 55%. It has been suggested that investigation can be used to control workload in a busy Department.

TABLE 12 (iv) RACH / SHO / CASUALTY OFFICER / RADIOLOGIST

	Patients	Xrays	%	Co#	RAD	Yield Pts	Yields XRS	False Positive	False Negative
01	434	241	55	6	5	1.1	2.0		1
02	276	100	36	6	3	1.0	3.0	4	1
03	290	106	36	5	2	0.7	1.9	3	0

There were 10 fractures diagnosed by a Radiologist. Nine fractures diagnosed by the Casualty Officer were not confirmed. Clinical management was modified by the false interpretation. Two fractures were missed. Neither child came to any harm as a consequence.

TABLE 12 (v) MDG/XRAY INTERPRETATION

CODE	NXR	# CO	#RAD	False +ve	False -ve
12	52	2	2	0	0
13	45	3	1	2	0
14	46	1	1	1	0
15	82	1	1	-	-
16	79	2	0	2	-
17	84	1	2	0	2
18	71	5	3	2	1
19	40	3	2	1	1

A clinical assistant had 100% x-ray usage, despite extensive experience and seniority. The types of fracture showed a predominance of linear fractures. Base of skull fracture was diagnosed clinically. There were 3 depressed fractures at Monklands 0.3%, none at Aberdeen.

There were more false positives than false negative interpretations, which reduced the risk to the patients. Only one child was identified in the series who was not xrayed. This was at Aberdeen. She returned and was found to have a skull fracture the following day but came to no harm.

The opportunity to interpret skull fractures from experience is limited. In Aberdeen in six months, 10 skull fractures were detected. Each of the three doctors worked single handed. On average each doctor would see 3.3 fractures in the course of their appointment. In Monklands 16 fractures were seen amongst eight doctors. On average each doctor might see two fractures in six months.

Further enquiry at Monklands Hospital in 1992 revealed that 4,800 xrays were taken on 5,600 head injured patients, 40% in the paediatric category. Xray usage was 85%, an increase since the introduction of the Guidelines by the College of Radiologists. This suggests that casualty officers, despite teaching and exposure to the recommendations of the Royal College of Radiologists, are not persuaded of the value or relevance of the Guidelines to their individual practice. The other possibility is that the patients are not meeting the criteria. This seems unlikely in the face of the evidence.

13. Results - Children are Different to Adults

The Author using a different data set reported on the differences between head trauma in adults and children, Brookes et al(1990). This Study shows children not only to be different to adults, but to children in different age groups as defined in this Thesis. The reader is referred to the individual results Sections for detailed analysis.

1. Age and Sex

The age of the child influences the frequency with which head injury occurred. Infants formed only 8% of the total. The preschool toddler most commonly sustained head trauma, 45%, followed by the primary school group, 39%.

Sex

The dominance of head injury in adult males was also seen in children, the frequency increasing with age.
The group sustaining head injury in adults was most commonly males 15-25 years.

2(a) The Cause of Injury

Falls were the commonest cause of injury.
Whilst adults were commonly injured as Road Traffic Accident occupants, this was rare in children, who were more frequently injured as pedestrians and cyclists. Only infants proved the exception.
Assaults occurred in the older male children, whilst there was no equivalent in adults for non-accidental injury, which occurred in children under two years of age and most commonly in infants where it was associated with a significant morbidity and mortality.
Falls in infants and non accidental injured were the commonest cause of fractures.
Fractures in the toddlers and primary school children most frequently complicated pedestrian and cycle traffic accidents.
Male children dominated for all causes of injury.

2(b) Mechanisms of Injury

Hard blunt trauma is the commonest mechanism of injury in adults and children with falls the commonest cause.

This mechanism in adults and children resulted in the highest rate of skull fracture.

Infants subjected to this mechanism were particularly vulnerable to skull fracture.

Infants sustained injury on soft surfaces, raising doubts about the causes and were rarely injured by sharp trauma.

2. Loss of Consciousness

Children overall lose consciousness less frequently than adults. This may reflect the more minor nature of head injury in children. The younger children lose consciousness less frequently than older children.

4.(a) Scalp Injury

The age of the child influences the type of scalp injury feature. Lacerations are common, except in infants. The latter have scalp haematomas and abrasions, though surprisingly 29% of infants had no detectable scalp injury.

Deep galeal lacerations were not seen in any of the infants. The incidence increased with the age of the child.

Fractures occurred most commonly when haematoma and abrasions of the scalp was a feature and the incidence was highest in the infants.

Though very small in numbers the galeal lacerations had a risk of fracture between 4.6% - 21.0%.

Adults with such lesions also have a high risk of associated fracture.

4(b) Site of Injury

The site of injury varied with age. The parietal site was most common in infants where it carried a higher risk of fracture. Toddlers sustained injury at the frontal site more commonly than others, and it was an infrequent site of skull fracture, multitype fractures and sometimes diastasis in infants raised suspicion of abuse. Rarely fractures in infants were attributed to further trauma, which was not a feature in the older child.

5. Headache and Vomiting

Headache may be under reported in young children.
Vomiting occurs less frequently in the younger child, than older children and adults.
The skull fracture is more commonly seen when both symptoms coexist.
In children these symptoms may be attributed to head trauma. Alternative causes are found for these symptoms, particularly when the head trauma has occurred at some interval before presentation.

6. Diagnostic Imaging

Skull xray requests are less frequent overall in children than in adults, suggesting less forceful injury and the low risk of fracture in the presence of minor scalp lacerations.
Infants with structural differences in the skull were much more vulnerable to skull fracture, than older children and adults.
The wide variation between SHO's in requesting skull xrays was a source of interest, though discretion was evident. Radiography was most frequently requested when cerebral impairment or dysfunction and more than one symptom was present. Similar criteria were applied to adults.

7. Disposal

Children were admitted overall less frequently than adults. Skull fracture resulted in admission and in two hospitals any history of loss of consciousness, despite recovery and the time of assessment. There was a liberal policy in the admission of infants particularly if there was any suspicion of non accidental injury. Vomiting at the time of examination resulted in admission, both for observation and at times for symptomatic relief and investigation of alternative pathology.

8. Intracranial Haematoma

This rare complication occurs less frequently in children than adults (see Section 8).
Children are more frequently discharged home from Accident & Emergency Departments, than adults, suggesting less frequent evidence of cerebral dysfunction.
Children develop clinical features not seen in adults, particularly childhood concussion syndrome.
Cerebral swelling may complicate childhood head trauma, which initially may appear as relatively minor injury. There is no comparable equivalent in minor trauma in adults.
Non accidental injury with its variable manifestations has no equivalent in older children or adults.
The detailed data on statistical analyses to support these statistics can be found in detail in the relevant sections of the Thesis, and in the associated statistical analyses Section 1.

9. Epilepsy – Seizure

Epilepsy, occurred overall in less than 1% of the children, some of whom were known epileptic. Surplus in infants, raised suspicion of intracranial injury and abuse, particularly in infants, in association with a haematoma.
Seizure associated with pyrexia was more common in the young child. The implications for the long term prognosis were different, than in adults.

10. Outcome

Severe cerebral dysfunction occurs less frequently in childhood. Abused children are the exception where mortability and morbidity is highest. The prognosis is also related to cause with hard blunt trauma, particularly associated with high velocity impacts in pedestrian and cycle accidents, resulting most commonly in cerebral injury.
Males capable of independent activity had the highest risk. This group most frequently were adult for neurosurgery.

11. Attendance Patterns

Whilst more than half the children attended after 14.00 hours, they much less commonly attended after midnight compared with adults. This relates to their independent and unsupervised activity, and was more common in the home and out of school. Adult attendance out of hours was most frequently associated with alcohol usage.

Children were most commonly injured when unsupervised by adult carers.

12. Care of Children in Accident & Emergency Departments

Not relevant to adults.

13. Children are Different to Adults

Children are different to adults and different to children in different age groups.

14. Guidelines

Guidelines for the management of recent head trauma in children were not agreed Nationally as for adults.

The adult Guidelines were not always applied to children, and no audit was possible.

There was wide variation in the SHO Group in the use of guidance for skull xrays. In some adult indicators were used, and discretion in withholding xrays was noted, but standardised practice was infrequent.

The SIGN Guidelines are being piloted by the Scottish Trauma Audit Group. Agreement on children, particularly for CT scanning in the presence of skull fracture, as a routine, is still a source of discussion.

ESSAY I a)

UNIQUE COMPLICATIONS

OF

PAEDIATRIC

HEAD TRAUMA

Unique Complications of Paediatric Head Trauma

1. Introduction

Complications of head trauma in childhood can occur, within hours of injury which are rarely or never seen in adult patients. Some complicate minor trauma and others less than severe trauma. Pyrexia often complicates the presentation of head injury.

2. Childhood Concussion Syndrome

This, perhaps inappropriately named condition, follows a blow, which may not even produce unconsciousness, and the child appears to return to a normal state. At a variable interval, minutes to hours after injury, the child becomes lethargic, irritable, pale, and may sweat profusely. Vomiting occurs and is repeated several times. There are no clinical or radiological features to suggest an intracranial mass lesion or raised intracranial pressure and CT is normal.

The condition responses to rest, sleep and where dehydration supervenes, intravenous fluids. The condition usually resolves spontaneously. Headache is not usually a major feature. Ketosis has been described in association with this phenomenon. The condition has been well recognised for years, having been described by Pickles(1950) but the cause remains to be elucidated.

3. Diffuse Cerebral Swelling and Cerebral Oedema

Enlargement of the brain after injury results from diffuse cerebral swelling or oedema. The latter occurs in relation to focal injury or haematoma. Diffuse cerebral swelling is not an infrequent response in children, and has been associated with classical post-mortem features(Graham, Ford and Adams 1989). These are now demonstrable on CT scanning(Snoek et al; Jennett and Adams 1979). In children the frequency was 70% compared with 17% in adults, without underlying pathology, and the precipitating factors were not known.

In conscious head injured children diffuse cerebral swelling was found in 29%, but in 40% of those in coma When the condition occurs without any evidence of diffuse axonal injury on CT scanning the prognosis is good. When diffuse axonal injury is present there is usually a progressive rise in intracranial pressure, and the prognosis is poor. The mechanism of injury may be relevant to the severity of the condition and its progression.

The condition is associated with the phenomenon of "talk and die" or "talk and deteriorate" in paediatric head trauma. Despite clinical similarities, the syndrome in childhood is rarely associated with a mass lesion. Rapid deterioration following head trauma can occur in minutes or hours. Whilst some cases are self limiting, others may progress to coma and death. Hendrick et al(1964) have claimed that as many as 15% of children admitted in the conscious state subsequently die. Theoretically no patient who is conscious following injury should be expected to die. In the absence of mass lesions, aggressive management can be initiated. Measuring of the blood gases and systemic blood pressure followed by intracranial pressure monitoring, can identify the transition from normal at the earliest possible time. Children who deteriorate have been the subject of many reports and various theories have been put forward in explanation of this phenomena. These have included cerebral oedema, seizures, migraine mechanisms, spreading depression of Loao, disturbance of the rostral brain stem, meningitis and a viral encephalitis. More recently the condition has been attributed to brain swelling. The availability of CT scanning and the features demonstrated has added to an understanding of this problem, when a mass lesion has been confidently excluded.

Bruce(1981) reviewed the records of 63 children who deteriorated following minor head injury. The group included those with the syndrome of malignant brain oedema. The commonest initial computerised tomography finding in head injured children is bilateral, diffuse cerebral swelling. Cerebral blood flow and CT density studies suggest that this swelling is due to cerebral hyperaemia and increased blood flow, not to oedema. Bruce divided the children into two groups. 14 had a Glasgow Coma Score greater than 8. All made a complete recovery with normal follow up scans. 49 had a Glasgow Coma Score of eight or less. 15 of these had a history of a lucid interval followed by unconsciousness. One of these children died of delayed brain swelling, the others recovered well with minimal neurological deficits. A third group of children, 34 in number, were rendered immediately and continuously unconscious from the time of the incident. In addition to diffuse CT features there were secondary lesions on the scans. 50% developed intracranial hypertension and five died. The remainder of this group of children were in coma for periods ranging from weeks to months. Subsequent CT scans showed extra cerebral collections of what was thought to be cerebrospinal fluid. Bruce relates the outcome to the difference between those with and without a lucid period, being the effect of diffuse primary brain injury to white matter. He does not indicate the incidence of this condition within the wide spectrum of head injury, or even within the minor head injury group. The condition begins several minutes to hours after injury. The clinical picture is similar to an expanding intracranial haematoma but a mass lesion is not found. This is of considerable importance to the doctor in the Accident & Emergency Department. The secondary clinical deterioration may be self-limiting, but may progress rapidly to coma and death. Bruce reports that 50% of children who die following head trauma are conscious on admission, and that 75% of deaths occur within the first 48 hours. He believes that aggressive resuscitation in the face of secondary deterioration, and the incidence of talk and die in the paediatric age group, can cut to zero deaths in this group. He postulates that there is a reversible pathological sequence. In children who are conscious, but who rapidly deteriorate, pathological studies show diffuse generalised brain swelling, but little evidence of primary brain injury. It is to this group that the term malignant oedema has been applied. The child who is unconscious from the moment if impact, and never recovers, but subsequently develops brain swelling and has raised intracranial pressure has a poor prognosis. Bruce suggests that at the time of writing he was unable to offer an explanation for the aetiology for this condition.

He matched the CT features with the clinical presentation and progress in each of the groups of children studied. He notes that there are only three components to the intracranial cavity CSF, brain, and blood. An increase in the brain bulk associated with decrease in CSF must be either due to increase in brain water content or increase in brain blood volume. After briefly comparing the syndrome to pseudo tumour cerebri he concludes that there is no evidence that brain oedema is present, on the initial scan in patients with acute diffuse brain swelling, and that cerebral blood flow is increased. Bruce suggests that acute cerebral swelling is produced by vascular engorgement, and offers the best explanation for diffuse cerebral swelling seen following trauma.

He does not have any knowledge of the basic triggering mechanisms that produce this syndrome. He states that it cannot be directly related to the degree of primary brain injury. The mechanism of injury however, is considered to be important since all the children who suffered this condition had an acceleration/deceleration type of brain injury. Movement around the brain stem is a factor which is thought to be involved. Bruce suggests that the vascular changes may be precipitated by alterations in the reticular formation or the locus ceruleus function. There may be delay in onset although there seems to be no explanation. It may be independent of the degree of primary brain injury, but more dependent on its mechanism. Others have suggested defective auto regulation. The release of substances as a consequence of the impact might spread to the third and fourth ventricles, triggering vasodilatation. Transient hypo perfusion of the brain may be followed by hyperaemia, but many cases of this syndrome occur in children, who do not have other injuries and have not been hypotensive at any stage. The effects of trauma on cerebral vessels may produce alteration in cerebrovascular tone resulting in hyperaemia. These vessels retain carbon dioxide responsiveness, and also respond to alterations in systemic arterial blood pressure.

After reviewing the hypothesis, Bruce concluded that the pattern of initial bilateral diffuse swelling following trauma in children, is produced by vasodilatation and initial hyperaemia. He noted that raised intracranial pressure does not normally develop in the first hours following the injury. Redistribution of blood from the brain membranes would account for an increase in brain bulk.

Bruce outlined the care given in his neurosurgery unit to the groups of patient he had analysed. He concluded that acute diffuse brain swelling is frequent in children and teenagers following acceleration/deceleration injuries of the brain. When associated with a lucid interval minimal underlying cerebral injury is likely, and hyperventilation for 24-48 hours may be all that is required to reverse the condition. By contrast, children in deep coma from the outset have suffered a significant degree of primary impact injury, and hyperaemia is imposed upon this. The control of swelling with hyperventilation is not usually totally successful and is followed by increases in intracranial pressure due to true cerebral oedema. The prognosis in these patients is less satisfactory.

Snoek Minderhoud and Wilmink(1984) reported 42 children who, following trivial or minor head injury developed neurological signs. One had a mass lesion. Three died. The reason for the deterioration has been attributed to cerebral oedema, seizures, migrainous patterns, the spreading depression of Loao, viral infections, encephalitis, functional disturbances of the brain stem and other mechanisms. The cases reported by Snoek all fell into the minor category. 40 had been admitted to hospital, two had actually been discharged and returned. The criteria for admission was not reported which would be helpful to the emergency physician. Because some of the patients developed post traumatic seizures this feature was used to divide his patients into two groups. The age distribution showed peaks between two and five and around nine years. There was no family history of epilepsy, but trauma and seizure history was present in the minority. EEG showed no relationships with the pattern of seizures. The non convulsive group included 29 patients who underwent rapid deterioration within 15 minutes or a slow deterioration over some hours. There were quite remarkable differences in the children in this group. A short lived but alarming period of deep coma, with unreactive pupils occurred in one patient. Other abnormalities included conjugate deviation, hemiparesis, transient blindness or violent vomiting. The sites of injury were frontal in one case and occipital in one. One eight year old child with a head blow became unresponsive with fixed dilated pupils and his condition was complicated by acute pulmonary oedema with fatal outcome.

Snoek et al(1984) suggested that the term lucid interval, which refers exclusively to conscious level at some point, should be supplanted by the term symptom free, which was relevant to his cases. He discussed the role of epilepsy in 16 of the children. Delayed deterioration has been attributed to epilepsy by other authors and where this has been present it has been used to separate cases into different categories.

Humphreys, Hendrick and Hoffman(1990) reported in detail four cases of children who "talked and died" but who were at some point able to verbalise. All had Glasgow Coma Scores of nine or greater initially. None had a mass lesion. Injury was sustained as a consequence of a motor vehicle or high velocity accident. As a result of the speed of deterioration, only one child had a post admission scan. They had all been categorised as benign injuries, at least initially. Review of the records of all the patients, either post mortem or on CT scanning, revealed significant injuries. Interestingly they all manifested a state of restless agitation and although they verbalised speech was certainly not normal. At the point of deterioration there was sudden cardiopulmonary arrest and electrolyte anomalies were noted with hyponatraemia.

The reports of children who "talk and deteriorate" and "talk and die" is of considerable relevance to the emergency physician. Humphreys et al(1990) asks "how are we best able to predict those children who are at risk of such deterioration and disastrous outcome?" He makes the following suggestions:-

That the history of injury should be borne in mind when the decisions are made regarding admission and the location at which the child is to be observed. Referring to the work of Sainsbury and Sibert(1984) he noted that this particular problem was addressed only in relation to intracranial bleeding. Humphreys(1990) suggests that all children who have sustained head violence as a result of a motor vehicle accident or fall, or who have impairment of their Glasgow Coma Score between 9 and 12 be admitted to a high dependency or Intensive Care facility. They also recommend early CT scanning in children with a Glasgow Coma Score of 9-12 and that particular attention be given to coincident restlessness in a child with head trauma. The question as to whether children with Glasgow Coma Scores of 9 or above should receive assisted ventilation is left for further consideration.

The Emergency Physician in the past faced with a child whose condition is causing concern and whose Glasgow Coma Score is reduced would have referred the child to a

Neurosurgical Unit for CT scanning for the exclusion of a mass lesion. The current situation with CT scanning available in District General Hospitals may result in the child being retained in such a facility to benefit from CT scanning and the exclusion of a mass lesion. However the rapid deterioration of the child at a distance from Neurological Intensive Care may result in such children being put at risk of irreversible changes. Hospitals providing scanning services should be prepared to deal with the Intensive Care aspects, at least initially, of the child who talks and deteriorates in order to ensure that he does not also die.

4. Transient Cortical Blindness

The condition is said to be rare, 0.4 - 0.6%. Small children however may have difficulty in describing their symptoms, and it may just manifest as distress. The condition requires very precise definition. It should be transient, self limiting and associated with normal pupillary reflexes and no evidence of physical injury in the visual pathways. If the condition affects both occipital cortices, then blindness should be complete. This needs to be separated from loss of vision due to field defects and homonymous hemianopia. These may be manifestations of migrainous phenomena albeit following trauma.

Hockstetler and Beals(1987) reported transient cortical blindness in a three year old child. She lost vision totally for two hours but subsequently made an uneventful recovery. Earlier reports had come from Pickles(1950). The time scales for these three groups suggested differences of aetiology. Many theories have been postulated. Vascular changes associated with vasospasm have been suggested. This was noted at operation. However these patients were anaesthetised, and Horton(1976) has already drawn attention to the influence of anaesthetic drugs on cerebral function. There have been suggestions that the occipital circulation, at greater distance from its origin is more vulnerable to mechanical changes. Certainly in conditions of raised intracranial pressure blood vessels may be pressed against rigid structures and their pulsation impaired. This hardly seems the explanation in such short lived cases. Harrison and Walls(1990) reported cases which presented in the Accident & Emergency Department and emphasised the difficulty of separating the trivial from the potentially serious complication. Greenblat(1973) reported three categories of the condition:

1. Juvenile affecting patients up to 8 years of age who developed blindness shortly after trauma and remained with a deficit for a few hours.

2. The adolescent form usually involving delayed blindness commencing some hours after injury and lasting several hours.
3. The adult form which may persists for several days following an accident. This category can hardly be regarded as transient.

There seems to be a correlation with migraine or seizure disorders. In children there may be difficulty in separating the features of migraine with, for example, an homonymous hemianopia from true and total loss of vision. Various suggestions have been made to account for this condition. Focal localised oedema and vasospasm have been offered as an explanation. In the reports of the early cases, investigation was limited. More recently EEG's have been recorded and have been normal prior to the onset of blindness and showed slowing or loss of alpha activity during the episode, particularly in the occipital lobes. All CT scans have been normal. In the paediatric group the prognosis is excellent. There have been further reports from an Accident & Emergency Department from Gleeson and Beattie(1994) and from Rodriguez et al(1993). The condition is probably underestimated in its occurrence, and has been described as "the overlooked syndrome" by Carmolo and Harris(1990). It is essential to exclude skull fracture in the occipital area. If present CT scanning is essential.

5. Summary of Results

Childhood Concussion Syndrome

Five children were identified who suffered Childhood Concussion Syndrome, detected only by the direct examination of the records of admitted children. The method of data collection did not provide for the specific identification of this condition.

2. Cerebral Swelling and Cerebral Oedema

No cases were identified in the A&E Departments.

3. Transient Cortical Blindness

In the Monklands Paediatric Study there were three cases of transient post traumatic cortical blindness. One in a male lasting six hours, one in a female lasting two to three hours, and one which resolved while the child was awaiting medical examination. All were diagnosed in Monklands Hospital and were associated with an occipital blow.
The data collection document did not specifically provide for the diagnosis, which may have been under reported.

2. The Febrile Child with a History of Head Injury

The Head Injured Child with Pyrexia/Infection

Humphreys et al(1975) in their contribution to Care for the Injured Child, from the Hospital for Sick Children, Toronto stated that "following a mild head injury with or without loss of consciousness, the child is irritable, lethargic, pyrexic and usually experiences episodes of profuse vomiting".
I sought to test this statement, particularly in relation to pyrexia. Because of the observation in our own Short Stay Ward that children were often admitted for observation with a history of head injury, who were subsequently found to have totally unrelated illness, as a cause of their pyrexia and illness.
The frequency with which pyrexia was associated with head trauma in the Accident and Emergency Department is shown in Table 11(i). No case of pyrexia was a direct consequence of the head trauma or of an infective complication within the skull, but one arose from an infected wound of the eyelid producing periorbital cellulitis.
The Casualty Officers were asked to note the temperature on arrival, and to indicate whether a source of infection other than the head injury was identified or suspected.
5416 children had a temperature of 37° - 89%.
174 had a temperature between 37° and 39° for which no cause was detected and 140 had a temperature between 37°C and 39°C with a source of infection diagnosed, and two patients temperatures in excess of 40° with an identifiable infectious cause.

It has been our experience at Monklands District General Hospital that a number of children admitted for observation following head injury, had pyrexia. Even if an infective cause had not been identified in the admission procedure, the source of pyrexia was readily identified subsequently.

Children with pyrexia often have headache and vomiting and their lethargy may be interpreted as drowsiness. In the presence of a history of head injury symptoms fulfil the criteria for admission for observation, thereby boosting the head injury admission rate.

It is almost inevitable that the parents will attribute the symptoms to the head injury whatever the interval from injury to presentation and frequently they appear to be unaware that the child is pyrexial. It has been our experience that delay in presentation following the injury is often associated with pyrexia and signs of inter current and unrelated infection.

The unit or ward to which such patients are admitted is immaterial in the presence of facilities for close monitoring and rapid recognition of serious infective illness followed by prompt treatment. Short stay adult head injury wards may not be appropriate for children, or for the infective condition.

a) Pyrexia as a Cause of Head Injury

Head injury may result from unsteadiness in a febrile child who has a tendency to fall. In such circumstances the child is usually a toddler and the head injury minor.

b) Pyrexia Complicating Head Injury

Infection may complicate head injury from the minor to the life threatening. It can be associated with an infected laceration or haematoma, a depressed or compound fracture or a fracture of the base of the skull.

Infection may produce meningitis, abscess or venous sinus thrombosis. Such complications might present after a period of delay. In the series the only recorded complication was a periorbital cellulitis secondary to a laceration of the upper eyelid. This produced a profoundly ill child who was admitted to a Specialist Ophthalmic unit.

The commonest identified infections were viral or bacterial upper respiratory tract infections, particularly otitis media. Gastroenteritis and chest infection occurred not infrequently.

It might be assumed that the paediatric hospital would be more alert to the possibility of inter current infection complicating minor head injury. This proved to be so in the

Royal Aberdeen Children's Hospital where alternative diagnoses were made. Skull xrays were used less often. The meningism characteristic of traumatic subarrachnoid bleeding must be differentiated from meningitis. Both can be associated with pyrexia, headache, photophobia, vomiting and a raised white cell count.

PYREXIA / INJURY ARRIVAL INTERVAL

	< 1 Hour N	%	1 - 2 Hours N	%	2-6 Hours N	%	6-12 Hours N	%	12-24 Hours N	%	> 24 Hours N	%	N/R N
a) 37°C	1832	-	1958	-	822	-	106	83	124	82	224	84	350
b) 37° - 39°	11	0.6	23	1.1	16	1.8	14	11	16	11	32	12	28
c) No Cause													

a) No cause identified
b) Cause identified
c) No Cause

The importance of the decisions in the Accident & Emergency Department is to ensure that the child who inevitably will be detained is admitted to the appropriate unit. This will ensure that the significance of symptoms and physical signs will be appreciated early and the most appropriate investigations initiated urgently.

The relationships between pyrexia and delay in presentation at the Accident & Emergency Department is noted - the longer the interval from the injury, the more likely is a cause of infection other than head injury found. There will be some occasions when the pyrexia is a consequence of a complication of the head trauma. A high index of suspicion is necessary. However the head injury ward should not be responsible for the care of unrelated pyrexial illness which may be hazardous to the head injured child. Diversion of resources from the close observation of the patient with head trauma may result. Many of these febrile illnesses could be dealt with by primary care physicians.

3. Summary and Conclusion

The data shows that a minority of children develop symptoms, which have no comparable match or syndrome in adults. Hence emergency doctors must be aware of these conditions, their significance and management.

Guidelines applied to adults do not address these conditions.

The assessment of the child may be more difficult, than of the adult.

There must be a more liberal admission and investigation policy for children.

Paediatric trained staff have been shown in this Study to reach an alternative diagnosis, when a child is presented with an illness, which coincides with current or more remote trauma, than doctors in none paediatric departments and without paediatric training.

The longer the delay between head trauma and presentation, the greater the likelihood of an alternative diagnosis. The two are not however mutually exclusive.

Coma may complicate other conditions, pyrexia may be seen in meningitis primary or secondary. Precise diagnosis and prompt intervention is essential.

The abused child with intracranial injury and subarrachnoid haemorrhage may present a complex diagnostic challenge. CT scanning and lumbar puncture may be required in the evaluation.

In all the less frequent presenting syndromes of paediatric head injury, require constant vigilance, experience and expertise which must be provided.

ESSAY I b)

CHILD ABUSE

NON ACCIDENTAL INJURY

A CAUSE OF INJURY

WITH

COMPLEX IMPLICATIONS

IN

THE ACCIDENT & EMERGENCY DEPARTMENT

Child Abuse – Non Accidental Injury

1. Introduction

Child abuse resulting in cerebral injury, is frequently severe leading to death or profound physical and mental deficits. It presents difficulties for the Emergency Physician as the history is often lacking or misleading, and therefore the causes and mechanisms of the neurological state may be difficult to evaluate.

There are profound social consequences for the family, and complex medicolegal discussions follow. Evidence of the patterns of injury has been accumulated over recent decades. There are, however, disparate opinions from medical experts. Media interest and criminal charges and litigation complicate the picture. For these reasons and because only 22 cases were confirmed, this is submitted as an Appendix separate from the Cause of Injury.

2. Review

The first descriptions of child battering were reported by Caffey(1946,1972) and were greeted with disbelief by the medical profession, and the lay public. The early work was done by Kempe et al(1961) Caffey(1946,1972) and Guthkelch(1971) who described the neurological prevalence and damage resulting from infant shaking and abuse. This led to clinical and social awareness, and the formulation of policies which it was hoped would lead to a virtual elimination of the problem. Sadly this has not proved to be the case, and abused children continue to present in Accident & Emergency Departments. Whether there is a true increase in the incidence or whether a high index of suspicion has ensured recognition is a matter for debate. The incidence has been calculated in the UK from child abuse registers, but is probably a significant underestimate. In the United States 1.5 - 1.75 million are thought to be abused each year. 3,000 die and many are left profoundly disabled physically and neurologically(National Centre for Child Abuse and Neglect 1998)

The abused child may sustain cerebral trauma in a variety of ways. By a direct blow to the head or face, or being wielded as an object, or in the shaken baby syndrome, subjected to rapid acceleration deceleration injury, by the whiplashing effect of the large mobile head on a poorly supported neck when violently shaken. The application of similar forces can occur when the child is swung by the feet. This may culminate in a blow against a flat surface such as a wall, or being thrown down on to a bed or sofa. Deceleration injury can occur without external evidence of injury in the latter situation. Combinations of injury repeated over time may produce complex patterns. Injury to the head can occur in isolation. The shaken baby mechanism may be seen without any alerting evidence of external trauma to the head or other areas of the body, or these may need to be sought with care. The infant, frequently under the age of one year, and often under the age of six months, may present in the Accident & Emergency Department with cerebral impairment, seizures, neurological deficits, vomiting, apnocic attacks or collapse without any obvious causative mechanism. Only vigilance on the part of staff will detect cases, and ensure rapid and appropriate action. Often the history is not forthcoming, or is deliberately misleading. Not infrequently when apnoea has occurred there is a claim of having tried mouth to mouth resuscitation. Bruising, it may be suggested, is the result of resuscitative efforts, particularly if observed around the thoracic cage. The action required in severe injury requires team effort. Medical staff are involved in the resuscitation of the child and its urgent investigation. While this is happening a senior member of staff should be attempting to obtain a history with tact and discretion. The local protocols for further investigation of such cases should be invoked.

There have been many reports, particularly from the North American continent. In 1978, after Caffey's reports, Zimmerman(1978) and his colleagues described parieto occipital inter hemispheric acute subdural haematomas in 17 out of 28 children, subjected to computerised brain scanning, following abuse. This was predominantly due to shaking. The lesions were described in 61% of the children. Retinal haemorrhages were identified, and the stigma of battery, as opposed to shaking was not noted. There was clinically severe dysfunction with bradycardia, apnoea and hypothermia. The prognosis was poor, and those who survived were profoundly disabled. A further report(Frank, Zimmerman and Leeds 1985) describing the evolution of the scan features was presented on four infants, who had been shaken. None had a skull fracture, one had a subdural haematoma, but all had cerebral injury. This was manifested by intractable seizures, coma, retinal oedema and haemorrhage and neurological abnormalities. Lumbar puncture revealed xanthochromia. Serial computerised scans in these four infants showed an evolving patterns of cerebral oedema, reduced ventricular size, but without focal abnormality. Later scattered hypodense areas developed and haemorrhagic infarcts appeared. These features spared the basal ganglia, cerebellum and brain stem. Later the ventricular system and cisterns enlarged, as diffuse brain atrophy developed, producing a characteristic abuse pattern.

The history in these cases, together with gross and classical intraocular signs of haemorrhage into the retina and vitreous, associated with retinal oedema is termed Purtchers retinopathy. Frank et al(1985) suggested that the ocular changes were the result of a sudden increase in venous pressure, associated with compression of the chest or abdomen. The author suggested that the ophthalmic features may be the only manifestation of the pathology in the emergency room. It may enable the diagnosis to be made from other causes of coma. After the initial control of any seizures, with anticonvulsants and possible Mannitol, the diagnosis can be confirmed on CT scanning. The most urgent differential diagnosis is meningitis, in which the classical signs of abuse do not occur. Wherever possible therefore CT scanning should precede lumbar puncture. It frequently shows xanthochromia. Frank emphasised the pattern of injury and the absence frequently of skull and external head trauma.

Ludwig and Warman(1984) detailed 20 cases of shaken baby syndrome, identifying the symptoms, clinical signs, laboratory features and the characteristics of CT brain scanning. Raised intracranial pressure, retinal haemorrhages and blood stained cerebrospinal fluid were found following shaking. None of the children in this series had injury to the head or skull. Despite this there were serious intracranial injuries, bradycardia, respiratory difficulty and hypothemia. The CT scans of the brain were the key to the diagnosis of shaking. Abnormalities including other bodily injuries may on occasion occur in combination.

Sinal and Ball(1987) studied the computed brain scans of 24 children, all but one in infancy. 17 children had been shaken and serial scans were obtained in half. The authors used Zimmerman's classification of grading. Retinal haemorrhages were visualised in the majority. Skull fractures were associated with battering, and in these retinal haemorrhages were rare. The authors commented that whilst inter hemispheric subdural haematoma characterised shaking, subarrachnoid bleeding had, as Zimmerman et al(1978) suggested, been underestimated.

Alexander et al(1986) reporting from Iowa, gave accounts of four further children who had been the subject of shaking. They acknowledge the value of CT scan in detecting subarrachnoid bleeding and subdural haematoma. One of their cases had a skull fracture. They found however that MRI scanning had several advantages over CT scanning for this type of intracranial injury. This included increased sensitivity in detecting intracranial bleeding and brain injury, which had value both in treatment, and in assessing prognosis. MRI provided greater detail and enhanced visual clarity compared with CT scanning. It was claimed to have facilitated appreciation of the physical findings in a court room situation! The value of MRI is the absence of ionising radiation, which means that the examinations can be repeated, particularly in infants, without the risk of excess xray exposure. Multiplanal imaging is an additional benefit, together with greater detail of the posterior fossa and the cervical spine. Unfortunately it is less readily available in the emergency situation, particularly in the United Kingdom. Skull fractures are better detected by conventional radiographic techniques, than either of the more advanced procedures. MRI proved superior to CT scanning in the detection of subdural haematomas, parenchymal injury, and posterior fossa haemorrhage. It is regarded as the most valuable tool in the assessment of the severity of injury and prognosis.

The diagnosis of less critical injury, whatever the mechanism, may be assisted by evidence of injury elsewhere, and in particular the metaphyseal abnormalities previously described by Caffey(1946). Though not the most common injury in child abuse, head injury is the most frequent cause of morbidity and mortality, particularly in the infant. The problems faced by the emergency physician in differentiating accidental from non accidental injury are immense. The history may be inadequate, inaccurate or deliberately misleading. A common feature in abuse cases is discrepancy between the alleged mechanism and the injury pattern. Complex fractures, bruising and other injuries should alert the medial office to the diagnostic possibility.

Billmire and Myers(1985) in discussing the reluctance to make a diagnosis, except by exclusion, commented on the emphasis of non accidental injury as the more frequent cause for intracranial pathology. They reviewed the records of children under the age of one year in a children's hospital in Cincinnati. Premature infants were excluded. 84 infants, 46 male and 38 female met the criteria for the study, which excluded neonates and infants with cerebral pathology. The cases studied were all admitted. 54 infants suffered accidental injuries with a mean age of five months and 28 had been subjected to abuse. The mean age in the last group was four months. The diagnosis was subsequently substantiated by investigations by appropriate care agencies. Criteria for inclusion in the study were defined, but the admission policy to the hospital was not. Neither was any information given regarding the attendances in the emergency room of children within the same group during the same period. Clearly this was a selected cohort. The authors reported that uncomplicated skull fracture, and concussion is much more common in accidental injury. Skull fracture with concussion was equal in each group whilst 95% of the children with demonstrable intracranial bleeding resulted from significant trauma due to road traffic accidents. Retinal haemorrhage was not reported in any case of accidental injury.

Commenting on the reluctance of physicians to acknowledge the possibility of abuse, the authors quoted the series of 330 accidental falls in children, which produced only one subdural haematoma, and a 10% incidence of concussion. Helfer, Slovis and Black(1977) reported on 246 children under the age of five years who fell out of bed, or a similar distance, and found only two subdural haematomas. No child in this group had a severe or life threatening intracranial injury. Hobb's(1984) study of skull fractures in children under the age of two years showed no subdural haematoma, and only one case of extradural haematoma in a child who fell from a pram. Nimityongskul and Anderson(1987) compared the minor degree of injury caused by falls.

Like Helfer(1977) and Hobbs(1984), Billmire and Myers(1985) concluded that accidental trauma rarely causes serious intracranial injury in infants, despite the fact that their series contained children of different age groups. 64% of the cases from Cincinnati of serious injury were due to abuse and 95% of those with the life threatening condition were abused. These authors emphasised the responsibility of the Emergency Room Physician in initiating investigation by the appropriate agencies, in any case where there is suspicion of abuse. The child's best chance of survival will depend on suspicion, good medical evaluation and treatment. Child abuse is the commonest cause of serious head injury under the age of one year. It places a significant responsibility on the emergency doctor to ensure that protection of the child, by whatever means is appropriate.

The story presented to the clinician is often of collapse with respiratory difficulty, apnoea or a seizure. Mouth to mouth resuscitation is often claimed to have been administered. Irritability, feeding problems, vomiting and repeated crying are said to have preceded the acute event. A child minder, whatever the relationship to the child, is frequently frustrated and inadequate, and may not be aware of the dangers of shaking, but knows that, unlike other forms of chastisement, it will not be associated with evidence of external injury. Dykes(1986) showed that the physical examination is characterised by the absence of external signs of injury to the head, lethargy and classically retinal haemorrhages. The vital signs are frequently abnormal, with bradycardia apnoea and hypothermia.

The ocular features have been detailed by Dykes(1986). The incidence in the new born child as a consequence of delivery is emphasised, the features being detectable only in the first month of life and usually not associated with signs of intracranial injury. Care therefore has to be taken within the first four weeks in relation to medicolegal matters. Beyond the age of one month they assume considerable significance. The mechanism of retinal and intraocular bleeding has been the source of discussion. Retinal haemorrhages occur with raised intracranial pressure producing Purtchers retinopathy. It may cause confusion, as it can occur as a complication of active resuscitative efforts for conditions other than abuse, or as a result of abuse. It can complicate raised intra-abdominal pressure, and be seen in sexual abuse. It is also known to be a feature of asphyxial disorders.

Examination of the fundi in an infant in the Emergency Room may prove a difficult procedure. It requires dilatation of the pupils. Eisenbrey(1979) commented that discovery is directly proportional to the care and persistence of the examiner. There will be a failure to attempt the procedure if a high index of suspicion is not maintained. Differential diagnosis includes haemorrhagic disorders, bacterial endocarditis and intracanial mass lesions of nontraumatic origin. Occasionally abuse may have occurred when an alternative diagnosis is present. The importance of fundoscopy in the Emergency Room was emphasised by Apolo(1987) in a report of three cases in which the examination was not undertaken. Lumbar puncture showed blood, which was attributed to a traumatic tap. Xanthocromia was not excluded, and consequently children at risk were placed at further risk by being returned to abusive parents. In Apolo's cases failure to examine the fundi, and the CSF appropriately, resulted in significant criticism of the Emergency Room management. CT scanning should precede any consideration of lumbar puncture. Children who are abused may demonstrate pyrexia, and this leads to confusion in the differential diagnosis of meningitis.

Postmortem forensic studies of 23 children who died from validated nonaccidental injury were reported by Green et al(1996). 16 had cerebral injury. The eyes were removed postmortem and the ocular features matched to the intracranial pathology. The ophthalmic features included retinal detachment, subhyaloid, intraretinal and perineural haemorrhage in the optic nerve in the children who died from cerebral complications. Subhyaloid haemorrhage was most common at the periphery of the retina. The authors emphasised the requirement for indirect ophthalmoscopy in life by a paediatric specialist. In practice CT brain scanning should precede this, because dilatation of the pupils for the examination would modify neurological assessment, particularly of an expanding mass lesion.

The extent and severity of the eye lesions was correlated with the severity of the intracranial injury.

One feature was identified, not previously reported. This was no direct communication between intracranial subdural haematoma, which was common, and perineural haemorrhage, which was seen only in the distal segments of the optic nerve. This excluded tracking of blood from the subdural haemorrhage to the nerve sheath. The mechanics of these phenomena were discussed particularly in relation to shaking.

It was also noted that less trauma occurred with subdural haemorrhage, than with more extensive intracranial injury, including subarrachnoid bleeding, lacerations and contusions.

48 cases of abuse over a seven year period with 13 fatalities were reported by Duhaime et al(1987) from Philadelphia. All the deaths had been associated with coma on arrival, and uncontrollable raised intracranial pressure. An important feature of this series is the absence of cranial trauma on clinical examination, but found in more than half, at autopsy. These authors undertook a biomechanical investigation of injury in infant models, subjected to a variety of forces both shaking and impact. They concluded head injury can be classified in to two major categories, according to the distribution of the pathological damage, whether focal or diffuse. The distinction is important for treatment and prognosis, as well as for establishing the biomechanical conditions necessary to produce a given injury. Focal injuries are associated with impact loading resulting in contact phenomena, whilst diffuse injuries are associated with impulse loading, conditions resulting from acceleration/deceleration phenomena. Damage to the brain occurs as a result of both of the biomechanical forces, and from the secondary effects of ischaemia, altered blood flow and cerebral oedema. They commented that the shaken baby syndrome had been postulated to result from non impact acceleration/deceleration forces, resulting in tearing of the bridging veins and subdural haematoma, from which death could result. Predisposing features in the infant was the relatively large size of the head, weakness of neck muscles, softness of the skull, relatively large subarrachnoid space and high water content of a poorly myelinated brain.

Noting recent experimental work these authors commented on the importance of the time interval over which acceleration occurs. Large accelerations over a short time tend to produce subdural haematoma, whilst those occurring over long intervals are associated with diffuse axonal injuries as reported by Gennarelli and Thibault(1985). One of the problems of the shaken baby syndrome in fatal cases, is that there is usually difficulty in obtaining accurate information of the mechanism of injury, making clinicopathology correlation difficult. In the series reported by Calder-hill and Scholtz(1984) white matter tears were found similar to those seen in blunt trauma in infancy. Duhaime et al(1987) concluded that their experimental results were consistent with the observations of fatal cases, which were associated, in addition with evidence of blunt trauma to the head. It was of some concern and interest however, that in more than half of the fatal cases no evidence of external trauma was noted on clinical examination, which contributed to the diagnosis of shaken baby syndrome. Skull fracture and scalp contusions were however found at autopsy, frequently in the occipital or parieto occipital regions. The recent report of Kleinman(1992) dating scalp haematomas is of interest in this context.

The authors recorded the importance of brain swelling in all their cases. They emphasised that drainage of relatively small subdural mass lesions would probably have contributed little to the control of intracranial pressure. The mechanism of ICP in these cases is poorly understood. They suggested that high acceleration in the posterior/anterior direction seems to be associated with this particular complication. It is yet to be conclusively investigated. Duhaime et al(1987) concluded that shaken baby syndrome, in its most severe form, and resulting in death, is not due to shaking alone. Shaking may be part of the process, but it is considered that in addition the infant suffers a blunt, or more than one blunt impact. It is postulated that the child having been shaken is thrown down striking the surface, with the head undergoing a large brief deceleration. The impact produces focal damage. There are shearing forces on the vessels and the parenchyma. It was suggested that complex factors interact in fatal cases.

Rivara(1988) examined the features of injury in children under the age of one year. 152 infants attended in the emergency room of Harbour View Hospital, Washington and the Children's Medical Centre, Seattle, with accidental injury between April 1985 and March 1986. Six of the children were diagnosed as having been abused, and were added to 45 cases of child abuse previously documented. The groups was then analysed. There were no differences in sex, age or socio-economic group. In the accidental group 67% of injuries occurred in the presence of the parent. 17 were in the company of others and only 16% were unwitnessed by an adult. By contrast, 42% of those abused sustained their injury apparently unsupervised, according to the history offered. The time of injury was examined, and it was found that 3% of accidental and 11% of non-accidental injury occurred between midnight and 9am, but the time at which medical attention was sought was not significantly different. 5% of accidents and 7% of non accidental injuries presented after midnight.

The injury occurred within the house in 86% of cases in each group. Falls were most common, resulting in 47% of the accidents, 18% were due to burns. Motor vehicle accident accounted for seven cases. Falls were also the most commonly reported cause in abused children. In 36% of the latter no explanation was offered for the injury, whilst in accidental cases an explanation was offered in all. In the accidental group there were eight skull fractures, two due to road traffic accidents, falls accounted for four fractures whilst being carried. One fracture occurred in a fall from a baby walker and one from a counter. In the abused group with single injuries which accounted for 68%, there was six skull fractures, whilst in the multiply injured group seven skull fractures occurred. Three children died of skull fracture in the abused group. The injury severity score was recorded as a mean of 1.6 in the accident and 8.7 in the abused group. Of even greater significance was the fact that 51% of the abused had a high severity score with only 3% of the accident group reached this figure. The differences in severity and the type of injury are also reflected in the admission rate. 20% of accidental injuries and 69% of abused children required admission, with a significant increase both in the duration of stay and the period in intensive care in the abused group. Some possibly were admitted for social reasons. On discharge, 25% of the abused and 3% of the accidental injures were thought to have a degree of disability. This paper is of interest as it identifies the importance of skull fracture. No child in either group is specifically identified as having an intracranial haematoma, either in the presence or absence of a skull fracture. In the discussion the authors comment that only one in 15 cases of injury presenting in the Accident & Emergency Department under the age of one year are in fact due to abuse. However the accidental injuries tend to be more minor. Serious injuries and those frequently resulting in death are rarely due to unintentional trauma, and where this is the case the cause is obvious, and is often a motor vehicle accident. Severe closed head injury, particularly associated with retinal haemorrhages should be regarded as pathognomonic of abuse. Within the accidental group there were indicators for accident prevention, but within the abuse group alternative specific interventions are required reflecting social and family dysfunction.

Within this article the authors emphasise the value of CT scanning not only in the diagnosis and clinical management of the abused child, but also for its value in the social and the legal procedures which such cases generate. They suggested that scanning made possible a view of the mechanism of injury, with frontal and occipital atrophy resulting from shaking and contracoup injury. In conjunction with clinical assessment, serial scans allowed some prediction of outcome. No child with evidence of brain atrophy totally recovered. Serial scans may also be of value in dating the injury as the patterns evolve.

As the sophistication of CT scanning has evolved with improved technology many more reports have been published. Cohen(1986) analysed the scans of 37 children and their related clinical data. 95% were under age of one year. The features demonstrated were subarachnoid haemorrhage 75%, cerebral oedema 65%, haemorrhage 30% with lacerations in three, subdural haematomas in 24%. Hypervasculaity, which later proceeded to infarction, also occurred. 30% had skull fractures, but the incidence of subdural bleeding was higher, without fracture - 85% : 45%.

These authors discussed in detail CT findings. They suggested that inter hemispheric density may have several causes. Oedema of the brain may make the falx prominent. Cohen(1986) questioned some of the views previously expressed by Zimmerman et al(1978). Further contributions have been made by Zimmerman and Bilaniuk(1994) and Kleinman(1987).

Though CT scanning has advanced knowledge of the pathology of intracranial injury following abuse and particularly shaking, Alexander(1986) reported the greater value of magnetic resonance imaging of intracranial injuries from child abuse. At this stage it is impossible to know how many children would have access to this facility in the United Kingdom. The value would be detection of minor changes confirming suspicion and avoidance of further injury in the future. It may also have medicolegal significance.

A comprehensive review of investigative procedures has been provided by Harwood Nash(1992) and an extensive review article on the clinical, biomechanical and forensic aspects of non accidental injury has been published by Brown and Minns(1993). These authors emphasise the complex medicolegal issues surrounding this particular emergency.

Zimmerman and Bilaniuk(1994) have published a comprehensive article describing the differing computerised scan features following childhood trauma including abuse.

A study by Hadley et al(1989) provided further information on children who had died following shaking. An autopsy report on six abused infants without skull fracture, identified subdural and epidural haematomas of the cervicomedullary junction in five. All had additional evidence of intracranial subdural and subarrachnoid haemorrhage, and retinal haemorrhages, with no evidence of external injury to the head or face. Impact on a soft surface may, however, produce diffuse injury without external features of trauma. Duhaime et al(1987) has, following biomechanical studies, claimed that whiplash alone cannot produce pathophysiological features of shaken baby syndrome, and that cranial impact is essential.

The literature review reveals varying views by medical experts in this field, which adds to the complexities of medicolegal argument.

The importance of excluding bleeding disorders in the investigation of abuse was reported by O'Hare and Eden(1984). 16% of cases had bleeding disorders which whilst not mutually exclusive in abuse may lead to medicolegal argument. Genetic coagulation disorders increase the risk of intracranial bleeding.

J.

STATISTICAL ANALYSIS

THE STATISTICAL METHODS - MR R HILL

The data was analysed by univarate analysis and regression methods.

In the univariate analysis chi-squared tests were performed to produce a logistic regression model of the response variable (skull fracture) as a function of each explanatory variable (age, cause of injury, etc). This shows the extent to which each explanatory variable predicts the response variable's outcome assuming all other things are equal.

One problem with univariate analysis is that it ignores the possibility that a collection of variables, each of which is weakly associated with the outcome, can become an important predictor of outcome when taken together. Multivariate logistic regression was therefore performed, as this method allows comparison of the relative significance of each explanatory variable in relation to other variables in the model and generates the model which provides the "best fit" for the sample date.

Regression methods are used to model the relationship between a response variable and one or more explanatory variables. Logistic regression is a method of modelling data when the outcome is discrete. The guiding principle with logistic regression is the same as in linear regression, to obtain the most parsimonious model which explains the outcome/response variable. To this end, we compare the observed values of the response variable to predict values obtained from models with and without each of the explanatory variables in question (along with various combinations thereof). If the predicted values with the variable in the model are better, or more accurate in some sense, that when the variable is not in the model, then we feel that the variable in question is "significant". The main reasons for choosing the logistic distribution are that it is an extremely flexible and useful s-shaped function and that it lends itself well to biological interpretation. For example if a patient presents with a laceration through the galea and has an odds ratio of 2, this indicates that it is twice as likely that this child will have a skull fracture.

A variable's association to the outcome can also be measured by using the odds ratio. An odds ration of 1 implies that the variable in question has none, or very limited, association with the response/outcome variable. Examining a confidence interval for the odds ratio can therefore be used as a test for significance. If a confidence interval for the odds ratio does not contain 1, or just barely does, then there is evidence to suppose that the variable in question has some association with the outcome.

Within the multivariate analysis, any variable whose univariate test produced a p value of <0.05 was considered as a candidate for the multivariate model, along with all variables of known biological importance.

STATISTICAL ANALYSES

1. Age & Sex - Univarate Analysis

Sex

Male Dominance

Significantly more than half the children presenting with head injury at the six centres were male, (one-sample t-test, HO: proportion of males = 50%, $t=19.4$, $df=5$, $p<0.0005$, 95% CI=(64%, 68%)).

TABLE 1 (SA)(i) Expected and Observed number of patients of each sex by age (Under H0: age of patients is independent of sex)

			SEX		Total
			Male	**Female**	
AGE	**<1 year**	Count	276.0	207.0	483
		Expected Count	318.3	164.7	483
	1-4 years	Count	1650.0	992.0	2642
		Expected Count	1741.0	901.0	2642
	5-11 years	Count	1584.0	676.0	2260
		Expected Count	1489.3	770.7	2260
	12-14 years	Count	351.0	123.0	474
		Expected Count	312.4	161.6	474
Total		Count	3861.0	1998.0	5859
		Expected Count	3861.0	1998.0	5859

Risk of Fracture by Age

The subjects were categorised by age. The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the age of the child, (chi-squared = 47.4, $df=3$, $p<0.0005$). In particular the infants (under 1 year) had substantially more fractures than expected.

The number of children in each age group with skull fractures and the expected number under a hypothesis of independence is shown in Table 1(SA)(ii).

TABLE 1(SA)(ii): Expected and Observed number of skull fractures by age (Under H0: skull fracture is independent of age)

			SKULL#		Total
			No Fracture	**Fracture**	
AGE	**<1 year**	Count	455.0	26.0	481.0
		Expected Count	472.4	8.6	481.0
	1-4 years	Count	2600.0	29.0	2629.0
		Expected Count	2582.1	46.9	2629.0
	5-11 years	Count	2217.0	35.0	2252.0
		Expected Count	2211.9	40.1	2252.0
	12-14 years	Count	458.0	14.0	472.0
		Expected Count	463.6	8.4	472.0
Total		Count	5730.0	104.0	5834.0
		Expected Count	5730.0	104.0	5834.0

When children under 1 are compared directly to the other subjects, they demonstrate a significantly higher risk than older children (chi-squared = 39.6, df=1, $p<0.0005$) and have an odds ratio of 3.8 (CI=(2.4,6.0)). Therefore a child under 1, presenting with a head injury, is almost 4 times more likely to result in a skull fracture than an older child.

The odds ratios and 95% confidence intervals for each age group are:

	Odds Ratio	**CI**
< 1 year:	3.8	(2.4, 6.0)
1-4 years:	0.47	(0.30, 0.72)
5-11 years:	0.80	(0.53, 1.21)
12-14 years:	1.79	(1.01, 3.17)

2(a) Cause of Injury

Cause of Injury – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the cause of the injury, (chi-squared = 57.0, df=7, p<0.0005). In particular pedestrians involved in an RTA had substantially more fractures than expected.

The number of skull fractures by each cause and the expected number under a hypothesis of independence is shown in table.

TABLE 2(a)(SA)(i): Expected and Observed number of skull fractures by cause of injury (Under H0: skull fracture is independent of cause of injury)

			SKULL #		Total
			No Fracture	**Fracture**	**Total**
CAUSE OF INJURY	**RTA–Occupant**	Count	90.0	2.0	92.0
		Expected Count	90.3	1.7	92.0
	RTA-Pedestrian	Count	149.0	12.0	161.0
		Expected Count	158.0	3.0	161.0
	RTA-Pedal cycle	Count	91.0	5.0	96.0
		Expected Count	94.2	1.8	96.0
	Assault	Count	541.0	7.0	548.0
		Expected Count	537.9	10.1	548.0
	Fall-School	Count	536.0	4.0	540.0
		Expected Count	530.1	9.9	540.0
	Fall-Home	Count	2628.0	31.0	2659.0
		Expected Count	2610.2	48.8	2659.0
	Fall-Sport/play	Count	1670.0	44.0	1714.0
		Expected Count	1682.6	31.4	1714.0
	Assault-NAI	Count	20.0	2.0	22.0
		Expected Count	21.6	0.4	22.0
Total		Count	5725.0	107.0	5832.0
		Expected Count	5725.0	107.0	5832.0

When children involved in an RTA (either as an occupant, pedestrian or cyclist) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 26.9, df=1, p<0.0005) and have an odds ratio of 3.5 (CI=(2.1,5.9)). Therefore a child presenting with a head injury after involvement in an RTA, is more than three(3) times more likely to result in a skull fracture than children whose injury has another cause, and the child pedestrian almost five(5) times as likely.

Children whose cause of injury was identified as child abuse (Assault-NAI) also resulted in a higher risk of skull fracture than children with fracture of other causes (chi-squared = 6.5, df=1, p=0.011), and have an odds ratio of 5.4 (CI= (1.3, 23.5)).

The odds ratios and 95% confidence intervals for each cause of injury are:

	Odds Ratio	**CI**
RTA–Occupant:	1.19	(0.29, 4.91)
RTA-Pedestrian:	4.73	(2.54, 8.81)
RTA-Pedal cycle:	3.04	(1.21, 7.63)
Assault:	0.67	(0.31, 1.45)
Fall-School:	0.38	(0.14, 1.03)
Fall-Home:	0.48	(0.32, 0.73)
Fall-Sport/play:	1.70	(1.15, 2.50)
Assault-NAI:	5.43	(1.25, 23.54)

2(a)(1) Cause of Injury by Sex - Univarate Analysis

The cause of injury was found to be dependent on the sex of the patient, (chi-squared = 71.445, df=7, p<0.0005), with males more likely to present after being involved in an RTA as a cyclist and after an assault.

TABLE 2(a)(SA)(ii): Expected and Observed number of patients by sex and cause of injury (Under H0: cause of injury is independent sex)

			SEX		
			Male	Female	TOTAL
CAUSE OF INJURY	RTA-Occupant	Count	50.0	44.0	94.0
		Expected Count	62.1	31.9	94.0
	RTA-Pedestrian	Count	107.0	56.0	163.0
		Expected Count	107.7	55.3	163.0
	RTA-Cyclist	Count	74.0	23.0	97.0
		Expected Count	64.1	32.9	97.0
	Assault	Count	411.0	136.0	547.0
		Expected Count	361.5	185.5	547.0
	Fall-School	Count	379.0	165.0	544.0
		Expected Count	359.5	184.5	544.0
	Fall-Home	Count	1637.0	1032.0	2669.0
		Expected Count	1763.7	905.3	2669.0
	Fall-Sport/Play	Count	1194.0	525.0	1719.0
		Expected Count	1135.9	583.1	1719.0
	Assault-NAI	Count	17.0	5.0	22.0
		Expected Count	14.5	7.5	22.0
Total		Count	3869	1986	5855
		Expected Count	3869	1986	5855

2(b) Mechanism of Injury – Univarate Analysis

Risk of Fracture byMechanism of Injury

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the mechanism of the injury, (chi-squared = 30.8, df=6, $p<0.0005$). In particular children falling to or bumping against a sharp edge had substantially less fractures than expected.

The number of skull fractures by each cause and the expected number under a hypothesis of independent is shown in Table 2(b)(SA)(i).

TABLE 2(b)(SA)(i) Expected and Observed number of skull fractures by mechanism of injury (Under HO: skull fracture is independent of mechanism of injury).

			SKULL # No	Yes	Total
MECHANISM OF INJURY	**Fall to soft surface**	Count	202.0	2.0	204
		Expected Count	200.3	3.7	204
	Fall to sharp edge	Count	739.0	4.0	743
		Expected Count	729.3	13.7	743
	Fall to hard surface	Count	2860.0	79.0	2939
		Expected Count	2885.0	54.0	2939
	Bump against sharp edge	Count	454.0	1.0	455
		Expected Count	446.6	8.4	455
	Bump against hard flat Surface/s	Count	830.0	11.0	841
		Expected Count	825.5	15.5	841
	Struck with blunt objects	Count	567.0	12.0	579
		Expected Count	568.4	10.6	579
	Struck with sharp object/s	Count	170.0	0.0	170
		Expected Count	166.9	3.1	170
Total		Count	5822.0	109.0	5931
		Expected Count	5822.0	109.0	5931

When children falling to or bumping against a sharp edge are considered as a group and compared directly to the other subjects, they demonstrate a significantly lower risk of skull fracture (chi-squared = 16.8, df=1, p<0.005) and have an odds ratio of 0.19 (CI=(0.08, 0.46)). Therefore a child presenting with a head injury after falling to or bumping against a sharp edge, is about a fifth as likely to result in a skull fracture than children whose injury resulted from another mechanism.

The odds ratios and 95% confidence intervals for each mechanism of injury are:

	Odds ratio	**CI**
Fall to soft surface	0.52	(0.13, 2.12)
Fall to sharp edge	0.26	(0.10, 0.71)
Fall to hard surface	2.73	(1.79, 4.17)
Bump against sharp edge	0.11	(0.02, 0.79)
Bump against hard flat surface/s	0.68	(0.36, 1.26)
Struck with blunt object/s	1.15	(0.63, 2.10)
Struck with sharp object/s	0.98	(0.98, 0.99)

2(b) Mechanism of Injury – Sex

The sex of patients presenting with a head injury was found to be dependent on the nature of the injury, (chi-squared = 39.08, df=6, $p<0.0005$). In particular males were more likely to present after being struck with a sharp or blunt object than females (chi-squared = 32.10, df=1, $p<0.0005$), and females more likely to present after a fall to a soft surface (chi-squared = 5.75, df=1, $p=0.016$).

The number of patients of each sex by mechanism of injury and the expected number under a hypothesis of independence is shown in Table 2(b)(SA)(ii).

TABLE 2(b)(SA)(ii) : Expected and Observed number of patients of each sex by mechanism of injury (Under HO : mechanism of injury is independent of sex).

			SEX		
			Male	**Female**	**Total**
MECHANISM OF INJURY	**Fall to soft surface**	Count	119.0	85.0	204
		Expected Count	134.9	69.1	204
	Fall to sharp edge	Count	493.0	252.0	745
		Expected Count	492.7	252.3	745
	Fall to hard surface	Count	1903.0	1252.0	2955
		Expected Count	1954.4	1000.6	2955
	Bump against sharp edge	Count	310.0	146.0	456
		Expected Count	301.6	154.4	456
	Bump against hard flat surface/s	Count	549.0	296.0	845
		Expected Count	558.9	286.1	854
	Struck with blunt objects	Count	434.0	146.0	580
		Expected Count	383.6	196.4	580
	Struck with sharp object/s	Count	130.0	39.0	169
		Expected Count	111.8	57.2	169
Total		Count	3938.0	2016.0	5954
		Expected Count	3938.0	2016.0	5954

3. Loss of Consciousness/Coma – Univarate Analysis

Risk of Fracture by Unconsciousnss

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on whether a history of unconsciousness or amnesia was noted, (chi-squared = 199.5, df=4, p<0.0005). All categories of unconsciousness or amnesia had substantially more fractures than expected.

The number of skull fractures by length of unconsciousness and the expected number under a hypothesis of independence is shown in Table 3(SA)(i).

TABLE 3(SA)(i): Expected and Observed number of skull fractures by history of unconsciousness/amnesia (Under H0: skull fracture is independent of a history of unconsciousness/amnesia)

			SKULL #		
			No	**Yes**	**Total**
UNCONSCIOUSNESS-?	**None**	Count	5477.0	81.0	5558
		Expected Count	5456.2	101.8	5558
	Less than 5 minutes with full recovery of consciousness	Count	317.0	16.0	333
		Expected Count	326.9	6.1	333
	Five to 30 minutes with full recovery of consciousness	Count	32.0	3.0	35
		Expected Count	34.4	0.6	35
	30 to 60 minutes with full recovery	Count	3.0	0.0	3
		Expected Count	2.9	0.1	3
	Greater than 1 hour and/or still disorientated or worse	Count	15.0	9.0	24
		Expected Count	23.6	0.4	24
Total		Count	5844.0	109.0	5953
		Expected Count	5844.0	109.0	5953

When all children with a history of unconsciousness or amnesia (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 65.1, df=1, p<0.0005) and have an odds ratio of 5.2 (CI=(3.3,8.0)). Therefore a child presenting with any history of unconsciousness or amnesia is about five (5) times more likely to result in a skull fracture than children with no history of unconsciousness or amnesia.

The odds ratios and 95% confidence intervals for each period of unconsciousness are:

	Odds Ratio	**CI**
None	0.19	(0.13, 0.30)
Less than 5 minutes with full recovery of consciousness	3.0	(1.74, 5.16)
5 – 30 minutes with full recovery of consciousness	5.14	(1.55, 17.05)
30 – 60 minutes with full recovery	0.98	(0.98, 0.99)
Greater than 1 hour and/or still disorientated or worse	34.97	(14.95, 81.80)

The probability of the child in the A&E Department in coma is thirty five (35) times that of a child who has no history of loss of consciousness.

Unconsciousness/Sex

The number of patients of each sex by unconsciousness/amnesia and the expected number under a hypothesis of independence is shown in Table 3(SA)(ii).

TABLE 3(SA)(ii) : Expected and Observed number of patients of each sex by history of unconsciousness/amnesia (Under HO: history of unconsciousness/amnesia is independent of sex).

			SEX Male	SEX Female	Total
UNCONSCIOUSNESS-?	**None**	Count	3669.0	1909.0	5578
		Expected Count	3684.1	1893.9	5578
	Less than 5 minutes with full recovery of consciousness	Count	230.0	105.0	335
		Expected Count	221.3	113.7	335
	Five to 30 minutes with full recovery of consciousness	Count	25.0	10.0	35
		Expected Count	23.1	11.9	35
	30 to 60 minutes with full recovery	Count	2.0	1.0	3
		Expected Count	2.0	1.0	3
	Greater than 1 hour and/or still disorientated or worse	Count	21.0	4.0	25
		Expected Count	16.5	8.5	25
Total		Count	3947.0	2029.0	5976
		Expected Count	3947.0	2029.0	5976

4.(a) Scalp Injury/Skull Fracture – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the nature of the injury, (chi-squared = 106.7, df=6, p<0.0005). In particular children with a superficial laceration (of any size) had a substantially lower number of fractures than expected and children with a laceration through galea (of any size) resulted in a substantially higher number of fractures than expected.

TABLE 4(a)(SA)(i) : Expected and Observed number of skull fractures by nature of injury (Under H0: skull fracture is independent of a nature of injury)

			SKULL #		
			No	**Yes**	**Total**
SCALP INJURY NATURE	**No external injury**	Count	855.0	8.0	863
		Expected Count	846.9	16.1	863
	Swelling only	Count	1610.0	61.0	1671
		Expected Count	1639.8	31.2	1671
	Abrasions/contusions only	Count	930.0	26.0	956
		Expected Count	938.2	17.8	956
	Superficial laceration <5 cm long	Count	2329.0	9.0	2338
		Expected Count	2294.4	43.6	2338
	Superficial laceration >5 cm long	Count	40.0	0.0	40
		Expected Count	39.3	0.7	40
	Laceration through galea <5 cm long	Count	62.0	3.0	65
		Expected Count	63.8	1.2	65
	Laceration through galea >5 cm long	Count	15.0	4.0	19
		Expected Count	18.6	0.4	19
Total		Count	5841.0	111.0	5952
		Expected Count	5841.0	111.0	5952

When all children with a laceration through galea (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 19.5, df=1, p<0.0005) and have an odds ratio of 5.0 (CI=(2.3,11.2)). Therefore a child with a laceration through galea is about five times more likely to result in a skull fracture than children with other injuries.

When all children with a superficial laceration (regardless of the length) are considered as a group and compared directly to the other subjects, they demonstrate a significantly lower risk of skull fracture (chi-squared = 47.8, df=1, $p<0.0005$) and have an odds ratio of 0.13 (CI=(0.07,0.26)). Therefore a child with a superficial laceration is less than a fifth as likely to result in a skull fracture as children with other injuries.

The odds ratios and 95% confidence intervals for each nature of injury are:

	Odds ratio	**CI**
No external injury:	0.45	(0.22, 0.93)
Swelling only:	3.21	(2.20, 4.68)
Abrasions/contusions only:	1.62	(1.04, 2.52)
Superficial laceration <5 cm long:	0.13	(0.07, 0.26)
Superficial laceration >5 cm long:	0.98	(0.98, 0.99)
Laceration through galea <5 cm long:	2.59	(0.80, 8.38)
Laceration through galea >5 cm long:	14.52	(4.74, 44.47)

4. Scalp Injury/Sex – Univarate Analysis

The sex of patients presenting with a head injury was found to be dependent on the nature of the scalp injury, (chi-squared = 74.02, df = 6, p<-.0005). In particular females were more likely to present with a superficial laceration (chi-squared = 54.07, df=1, $p<0.005$).

The number of patients of each sex by nature of injury and the expected number under a hypothesis of independence is shown in Table 4(a)(SA)(ii).

TABLE 4(a)(SA)(ii) : Expected and Observed number of patients of each sex by nature of injury (Under H0: nature of injury is independent of sex)

			SEX		
			Male	**Female**	**Total**
SCALP INJURY/NATURE	**No external injury**	Count	517.0	351.0	868
		Expected Count	573.5	294.5	868
	Swelling only	Count	1020.0	663.0	1683
		Expected Count	1112.0	571.0	1683
	Abrasions/contusions only	Count	647.0	312.0	959
		Expected Count	633.7	325.3	959
	Superficial laceration <5 cm long	Count	1678.0	663.0	2341
		Expected Count	1546.8	794.2	2341
	Superficial laceration >5 cm long	Count	27.0	13.0	40
		Expected Count	26.4	13.6	40
	Laceration through galea <5 cm long	Count	44.0	21.0	65
		Expected Count	42.9	22.1	65
	Laceration through galea >5 cm long	Count	15.0	4.0	19
		Expected Count	12.6	6.4	19
Total		Count	3948.0	2027.0	5975
		Expected Count	3948.0	2027.0	5975

Sex/Age

The sex of patients presenting with a head injury was found to be dependent on the age of the patients, (chi-squared = 62.11, df = 3, p<0.0005). In particular younger females (under five years of age) were more likely to present than younger males (chi-squared = 54.25, df = 1, p<0.0005), with more older males (five years and older) more likely to present than older females.

Nature of Scalp Injury in Children under One year

The nature of injuries seen in children under one year old was significantly different to that in older children (chi-squared = 204.7, df = 6, p<0.0005). As can be seen in figures below, most Infants presented with either swelling only (38%) or no external injury(28%), whereas older children presented more often with a superficial laceration less than 5 cm long (42%), with 27% presenting with swelling only.

Figure 4(a)(SA)(i) : Nature of Injury (percent of total cases) in children under 1 year old.

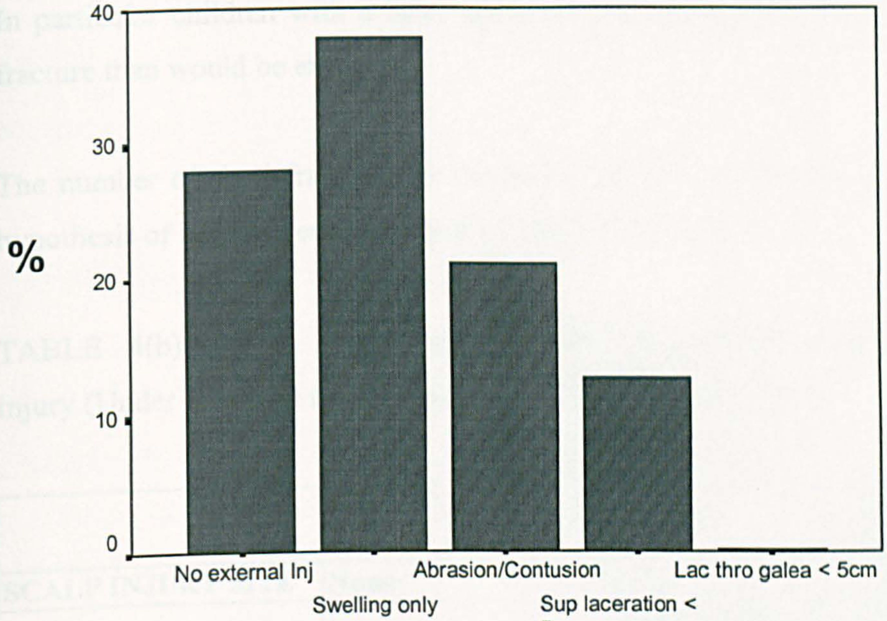
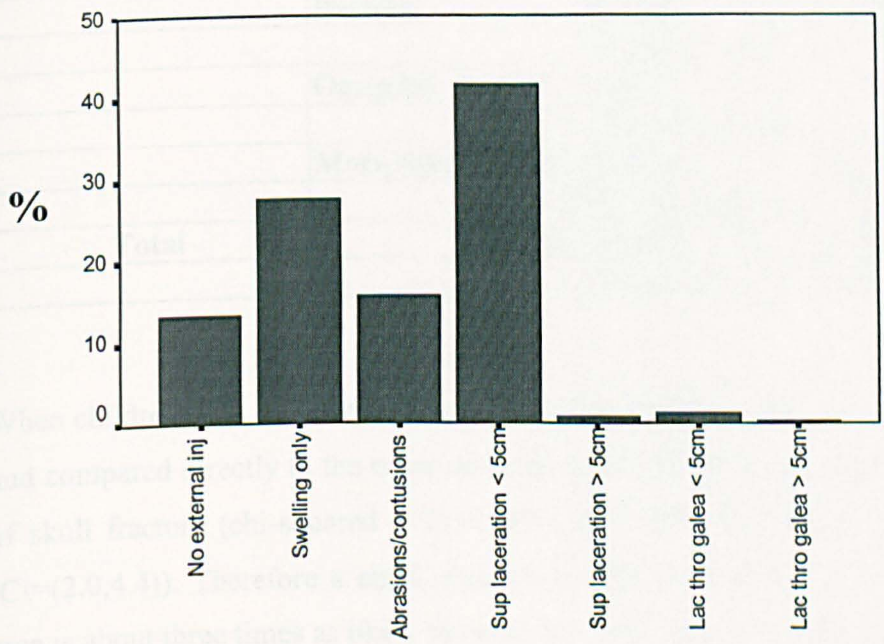


Figure 4(a)(SA)(ii): Nature of Scalp Injury (percent of total cases) in children of 1 year and older



4(b)(SA) Site of Injury – Skull Fracture

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on the site of the injury, (chi-squared = 103.4, df-5, p<0.0005). In particular children with a head injury on the parietal area have substantially more fracture than would be expected.

The number of skull fractures by the site of injury and the expected number under a hypothesis of independence is shown in Table 4(b)(SA(i).

TABLE 4(b)(SA)(i): Expected and Observed number of skull fractures by site of injury (Under H0: skull fracture is independent of site of injury)

			SKULL #		
			No	**Yes**	**Total**
SCALP INJURY SITE	**None**	Count	752.0	4.0	756
		Expected Count	741.9	14.1	756
	Frontal	Count	3043.0	25.0	3068
		Expected Count	3010.8	57.2	3068
	Temporal	Count	274.0	6.0	280
		Expected Count	274.8	5.2	280
	Parietal	Count	665.0	34.0	699
		Expected Count	686.0	13.0	699
	Occipital	Count	983.0	30.0	1013
		Expected Count	994.1	18.9	1013
	More than one site	Count	123.0	12.0	135
		Expected Count	132.5	2.5	135
Total		Count	5840.0	111.0	5951
		Expected Count	5840.0	111.0	5951

When children with an injury on the temporal or parietal areas are considered as a group and compared directly to the other subjects, they demonstrate a significantly higher risk of skull fracture (chi-squared = 31.6, df=1, p<0.0005) and have an odds ratio of 2.9 (CI=(2.0,4.4)). Therefore a child presenting with an injury to the temporal or parietal area is about three times as likely to result in a skull fracture than children with an injury to another site.

The odds ratio and 95% confidence intervals for each site of injury are:

	Odds Ratio	CI
None	0.25	(0.09, 0.69)
Frontal	0.27	(1.17, 0.42)
Temporal	1.16	(0.51, 2.67)
Parietal	3.44	(2.28, 5.19)
Occipital	1.83	(1.20, 2.80)
More than one site	5.63	(3.02, 10.53)

4(b) Site of Injury – Sex - Univarate Analysis

The sex of patients presenting with a head injury was found to be dependent on the site of injury, (chi-squared = 25.15, df=5, p<0.0005). In particular females were less likely to present with an injury to the temporal or parietal areas (chi-squared = 14.36, df=1, p<0.0005), and more likely to present with no site of injury (chi-squared = 13.56, df=1, p<0.0005).

The number of patients of each sex by site injury and the expected number under a hypothesis of independence is shown in Table 4(b)(SA)(ii).

TABLE 4(b)(SA)(ii) : Expected and Observed number of patients of each sex by site of injury (Under H0: site of injury is independent of sex)

			SEX		
			Male	**Female**	**Total**
SCALP INJURY SITE	**None**	Count	458.0	303.0	761
		Expected Count	502.9	258.1	761
	Frontal	Count	2025.0	1053.0	3078
		Expected Count	2034.1	1043.9	3078
	Temporal	Count	196.0	84.0	280
		Expected Count	185.0	95.0	280
	Parietal	Count	503.0	197.0	700
		Expected Count	462.6	237.4	700
	Occipital	Count	671.0	348.0	1019
		Expected Count	673.4	345.6	1019
	More than one site	Count	95.0	41.0	136
		Expected Count	89.9	46.1	136
Total		Count	3948.0	2026.0	5974
		Expected Count	3948.0	2026.0	5974

5. Headache and Vomiting

(a) Risk of Fracture by Headache – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of headache, (chi-squared = 41.1, df=1, p<0.0005).

The number of skull fractures by the presence of headache and the expected number under a hypothesis of independent is shown in Table 5(SA)(i).

TABLE 5(SA)(a)(i): Expected and Observed number of skull fractures by headache (Under HO: skull fracture is independent of headache)

			SKULL #		
			No	**Yes**	**Total**
HEADACHE	**None**	Count	4369.0	45.0	44414
		Expected Count	4347.2	66.8	4414
	Headache	Count	903.0	36.0	939
		Expected Count	924.8	14.2	939
Total		Count	5272.0	81.0	5353
		Expected Count	5272.0	81.0	5353

Headache had an odds ratio of 3.9 (CI=(2.5, 6.0)). Therefore a child presenting with a history of headache is almost four(4) times as likely to result in a skull fracture than children without a history of headache.

The odds ratios and 95% confidence intervals are:

	Odds Ratio	**CI**
No Headache	0.26	(0.17, 0.40)
Headache	3.9	(2.5, 6.0)

The sex of patients presenting with a head injury was found to be independent of a history of headache, chi-squared = 0.582, df=1, p=0.445).

The number of patients of each sex by history of headache and the expected number under a hypothesis of independence is shown in table 5(a)(SA)(ii).

Table 5(a)(SA)(ii): Expected and Observed number of patients of each sex by headache (Under HO: headache is independent of sex).

			SEX		
			MALE	FEMALE	Total
HEADACHE	**None**	Count	2960.0	1460.0	4420
		Expected Count	2950.0	1460.0	4420
	Headache	Count	622.0	325.0	947
		Expected Count	632.0	315.0	947
Total		Count	3582.0	1785.0	5367
		Expected Count	3582.0	1785.0	5367

Vomiting

The sex of patients presenting with a head injury was found to be dependent on a history of vomiting, (chi-squared = 9.93, df=2, p=0.007).

The number of patients of each sex by vomiting and the expected number under a hypothesis of independence is shown in table 5(b)(SA)(iii).

Table 5(b)(SA)(iii): Expected and Observed number of patients of each sex by vomiting (Under HO: vomiting is independent of sex).

			SEX		
			Male	Female	Total
VOMITING	**None**	Count	3472.0	1730.0	5202
		Expected Count	3436.6	1765.4	5202
	Vomited (once)	Count	226.0	128.0	354
		Expected Count	233.9	120.1	354
	Vomited (more than once)	Count	248.0	169.0	417
		Expected Count	275.5	141.5	417
Total		Count	3946.0	2027.0	5973
		Expected Count	3946.0	2027.0	5973

(b) Risk of Fracture by Vomiting – Univarate Analysis

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of vomiting, (chi-squared = 35.2, df=2, p<0.0005).

The number of skull fractures by vomiting and the expected number under a hypothesis of independence is shown in Table 6(b)(SA)(i).

TABLE 5(b)(SA)(i) : Expected and Observed number of skull fractures by vomiting (Under H0: skull fracture is independent of vomiting)

			SKULL #		
			No	**Yes**	**Total**
VOMITING	**None**	Count	5114.0	75.0	5189
		Expected Count	5094.0	95.0	5189
	Vomited (once)	Count	334.0	18.0	352
		Expected Count	345.6	6.4	352
	Vomited (more than once)	Count	394.0	16.0	410
		Expected Count	402.5	7.5	410
Total		Count	5842.0	109.0	5951
		Expected Count	5842.0	109.0	5951

When all children with a history of vomiting (either once or more than once) were considered as a group and compared to children with no history of vomiting they showed an odds ratio of 3.2 (CI=(2.1, 4.8)). Therefore a child presenting with a history of vomiting is about three (3) times as likely to result in a skull fracture than children without a history of vomiting.

The odds ratios and 95% confidence intervals are:

	Odds ratio	**CI**
None:	0.31	(0.21, 0.47)
Vomited (once):	3.26	(1.94, 5.47)
Vomited (more than once):	2.38	(1.39, 4.08)

5(c)(SA) Risk of Fracture by Headache & Vomiting

The probability of a child presenting with a head injury resulting in a skull fracture was found to be dependent on a history of headache associated with vomiting, (chi-squared = 48.2, df=1, p<0.0005).

The number of skull fractures by the presence of both headache and vomiting and the expected number under a hypothesis of independence is shown in Table 6(c)(SA)(i) .

TABLE 5(c)(SA)(i) : Expected and Observed number of skull fractures by headache (Under H0: skull fracture is independent of headache and vomiting)

			SKULL #		
			No	**Yes**	**Total**
HEADACHE & VOMITING	**No Headache & Vomiting**	Count	5467.0	83.0	5550
		Expected Count	5452.0	98.0	5550
	Headache & Vomiting	Count	263.0	20.0	283
		Expected Count	278.0	5.0	283
Total		Count	5730.0	103.0	5833
		Expected Count	5730.0	103.0	5833

Headache and vomiting had an odds ratio of 5.01 (CI=(3.0, 8.3)). Therefore a child presenting with a history of headache and vomiting was five times as likely to result in a skull fracture as children without a history of both headache and vomiting.

The odds ratios and 95% confidence intervals are:

	Odds ratio	**CI**
Not Headache & Vomiting:	0.20	(0.12, 0.33)
Headache & Vomiting:	5.01	(3.0, 8.3)

Headache, Vomiting and Unconsciousness

If we consider children presenting with a history of unconsciousness (of any length), headache and vomiting (once or more than once) as a group, and compare this group to all other patients we find that this group has a significantly greater risk of skull fracture than other patients (chi-squared = 53.8, df=1, $p<0.0005$). Although this represents a small proportion of children seen (63 patients, 1.1%), this group has a very high odds ratio (9.5, CI=(4.6, 19.7). Within our study, patients presenting with a combination of vomiting, headache and unconsciousness were more than nine(9) times as likely to have a skull fracture than other patients.

TABLE 5(c)(SA)(i) : Expected and Observed number of skull fractures by vomiting, headache and unconsciousness (Under H0: skull fracture is independent of vomiting, headache and unconsciousness)

			SKULL #		
			No	**Yes**	**Total**
Vomiting + Headache + Unconsciousness	**No**	Count	5800.0	102.0	5902
		Expected Count	5792.2	109.8	5902
	Yes	Count	54.0	9.0	63
		Expected Count	61.8	1.2	63
Total		Count	5854.0	111.0	5965
		Expected Count	5854.0	111.0	5965

The odds ratios and 95% confidence intervals are:

	Odds Ratio	**CI**
Vomiting + Headache + Unconscious	9.5	(4.6, 19.7)
Not Vomiting + Headache + Unconscious:	0.11	(0.05, 0.22)

8. Haematoma – Univarate Analysis

Risk of Haematoma/Skull Fracture

The probability of a child presenting with a head injury resulting in a haematoma was found to be dependent on the presence of a skull fracture, (chi-squared = 278.34, df=1, $p<0.0005$).

The number of haematomas by the presence of a skull fracture and the expected number under a hypothesis of independent are shown in Table 8(SA)(i).

TABLE 8(SA)(i): Expected and Observed number of haematomas by skull fracture (Under H0: haematoma is independent of skull fracture)

			SKULL #		
			No	Yes	Total
HAEMATOMA	No	Count	5848.0	102.0	5950
		Expected Count	5839.3	110.7	5950
	Yes	Count	6.0	9.0	15
		Expected Count	14.7	0.3	15
Total		Count	5854.0	111.0	5965
		Expected Count	5854.0	111.0	5965

14 of 15 intracranial haematomas occurred in male children.

Skull fracture had an odds ratio of 86.0 (CI=(30.05, 246.10)) of resulting in a haematoma. Therefore a child with a skull fracture is more than 80 times as likely to result in a haematoma as a child without a skull fracture.

However, when children under one year are considered separately, they do not show an increased risk of intracranial haematoma, even when a skull fracture is confirmed (chi squared=0.57, df=1, $p<0.811$). Children of this age, with a skull fracture have an odds ratio of 1.0 (CI=(0.99, 1.00) of having a haematoma.

Risk of Haematoma/Unconsciousness

Six of 15 children in the Monklands Study had no history of loss of consciousness or cerebral impairment in the A&E Department and subsequently were found to have a haematoma. This represents six out of almost 6,000 children.

BIBLIOGRAPHY

1. ADELOYE A, OIANG HM, OLUMIDE AA. Patterns of acute head injuries in Ibadan. Intern. Surg. 1976;61:478-480.

2. ADVANCED TRAUMA LIFE SUPPORT COURSE. American College of Surgeons. Chicago.

3. AGRAN PF. Injuries to children. The relationship of child development to prevention strategies Pub. Health. Rep. 1987;102:609-610.

4. AGRAN PF, DUNKLE DE, WINN DG. Injuries to a sample of seat belted children evaluated and treated in a hospital emergency room. J Trauma. 1987;27:58-64.

5. ALBERICO M, WARD JD, CHOI SG, et al. Outcome after severe head injury: relationships to mass lesions, diffuse injury and ICP course in paediatric and adult patients. J. Neurosurgy 1987;67:648-656.

6. ALDRICH EF, EISENBERG HM, SAYDJAR C et al. Moderate head injury. Competing the clinical spectrum of brain trauma. Neurosurgery 1982;11:344-251.

7. ALEXANDER RC, SCHOR DP, SMITH WL. Magnetic resonance imaging of intracranial injuries from child abuse. J. Paediatr. 1986:109(6):975-979.

8. ALEXANDRE A, COLONBO F, NERTEMPI P et al. Cognitive outcome and early indices of severity of head injury. J Neurosurg 1983;59:751-761.

9. ANNERGERS JF. The epidemiology of head trauma in children. In Shapiro (Ed) Paediatric head trauma New York Futura Publishing Company 1983:1-10.

10. ANNEGERS JF, GRABOW JD, GROOVER RV, et al. Seizures after head trauma: a Population study. Neurology 1980;30:683-689.

11. ANNEGERS JF. The epidemiology of head trauma in children. In Shapiro (Ed).

12. ANTIMICROBIAL PROPHYLAXIS IN NEUROSURGERY AND AFTER HEAD INJURY. Infection in Neurosurgery. Working Party of the British Society for antimicrobial chemotherapy Lancet 1994; 344:1547-1551.

13. ARIMA M. Clinical correlation with cyclic vomiting and epilepsy. Post epileptic ketonuria in children. Acta. Paed. Jpn. 1959;63:1834-1839.

14. APOLO J. Blood cerebrospinal fluid : Traumatic tap or child abuse? Paed. Emerg. Care 1987;3(2):93-95.

15. AVERY JG, HAYES HRM. Death and injury to children in cars in Britain. Br. Med. J. 1985;291-515.

16. BAKER S. Motor vehicle occupant deaths in young children. Paediatrics 1979;64:860.

17. BALLANTYNE E, MENDELOW AD,, MacMILLAN R. Relationships between scalp wounds and skull fractures. IRCS Med. Sci. 1986;63:14.

18. BALLANTYNE E, MENDELOW AD, MACMILLAN R, BROOKES M. Relationship between scalp wounds and skull fractures. IRCS Med. Sci. 1986;14:63-64.

19. BARLOW B, NEIMIRISKA M, GANDHI R et al. 10 years experience with falls from a height in children. J.. Surg. 1983;4:509-511.

20. BAXT NG, MOODY P. The impact of advanced prehospital care on the mortality of the severely brain injured patient. J. Trauma 1987;27:365-369.

21. BEATTIE TF, MOIR PA. Paediatric accident and emergency short stay ward. Arch. Emerg. Med. 1993;10:181-186.

22. BECKER DP, MILLER JD, YOUNG HF et al. Diagnosis and treatment of head injury in adults. In Youmani JR (Ed) Neurological Surgery. Vol 2. WB Saunder 1982 Philadelphia.

23. BELL RS, LOOP JW. The utility and futility of radiographic skull examination for trauma. N. Eng. J. Med 1971;284:236-239.

24. BERGNER L, MAYER S, HARRIS D. Falls from heights : a childhood epidemic in an urban area. Am. J. Pub Health 1971;61(1):90-96.

25. BERNEY J, FAVIER J, FROID W, VAUX AC, Paediatric head trauma : influence of age and sex II. Biomechanical correlations. Child's Nerv. Syst. 1994;10(8):517-523.

26. BILLMIRE ME, MYERS PA. Serious head injury infants. Accident or abuse? Paediatrics;198575:340-342.

27. BION JF, EDLIN SA, RAMSAY G et al. Validation of a prognostic score in critically ill patients undergoing ransport. Br. Med. J. 1985;291:432-434.

28. BISHOP DMV. Plasticity and specificity of language localisation in the developing brain. Devel. Med. Child. Neurol. 1981;23:251-255.

29. BRICOLO A P, PASTUT L M. Extradural haematoma toward zero mortality - a prospective study. Neurosurgery 1984;14:8-12.

30. BRIGGS M, CLARKE P, CROCKARD et al. Guidelines for initial management after head injury in adults. Br. Med. J. 1984;287:1173-1176.

31. BROWN – Depressed Fracture in Children.

32. BRUNK J D, IMBUS C, WOO-SAM J. Physical recovery after severe closed had trauma in children and adolescents. J. Paediatr. 1980;7721-723.

33. BONADIO WA, SMITH DS, HILLMAN S. Clinical indicators on computed tomographic scans in children with parietal skull fracture. ADJC 1989;143:194-8.

34. BONADIO WA, SMITH DS, HILLMAN S. Clinical indicators of intracranial lesions on computerised tomographic scans in children with parietal skull fracture. ADJC 1989;143:194-198.

35. BOULIS ZF, DICK R, BARNES NR. Head injuries in children; aetiology, symptoms physical findings and x-ray wastage. Br. J. Radiol. 1978;51:851-854.

36. BRAAKMAN R. Depressed skull fracture. In Viken Ph and Bruyn Handbook of Clinical Neurology. Vol 23. North Holland Publishing Co. 1975. Pp 403-415.

37. BRAMAN SS, DUNN SM, ANNIC CA et al. Complication of intrahospital transport in the critcally ill patient. Ann. Int. med. 1987;167:469-473.

38. BRAWLEY BW, KELLY WA. Treatment of basal skull fractures, with and without cerebro-spinal fluid fistulas. J. Neurosurg. 1967;26:57-61.

39. BRICOLO AP, PASUT. Extradural haematoma - towards zero mortality. Neurosurgery 1984;14:8-12.

40. BRIGGS M, CLARKE P, CROCKARD A, et al. Guidelines for initial management after head injury in adults. Suggestions from a Group of Neurosurgeons Br. Med. J. 1984;288:983-985.

41. BRINK JD, IMBUS C, WOO-SAM J. Physical recovery after severe head trauma in children and adolescents. J. Paediatr. 1980;97:721-727.

42. BROCKLEHURST G, GOODING M, JAMES G. Comprehensive care of patients with head injuries Br. Med. J. 1987;294-345-347.

43. BROOKES M, MacMILLAN R, CULLY S, et al. Head injuries in accident & emergency departments in Scotland, children different to adults. J. Epidemiol. 1990;44:147-151.

44. BROOKES M, MENDELOW AD, MACMILLAN et al. Which head injured patients have a skull fracture? Arch. Emerg. Med. 1986;3:91-92.

45. BROWN – Depressed Fracture in Children.

46. BROWN JK, MIMMS RA. Non-accidental head injury with particular reference to whiplash shaking injury and medicolegal aspects. Dev. Med and Child. Neurol 1993;35:849-869.

47. BRUCE DA, ALAVI I, BILANIER L, et al. Diffuse central swelling following head injuries in children: the syndrome of malignant trauma oedema. J. Neurosur. 1981;54:170-178.

48. BRUCE DA, RAPHAELY RG, GOLDBERG AL et al. The Pathophysiology, treatment and outcome following severe injury in children. Child's Brain 1979;5:174-191.

49. BRUCE DA, SCHUT L. The value of CT scanning following paediatric head injury. Clin. Paed. 1977, 19:719-725.

50. BRUCE D, SCHUT L. Management of acute craniocerebral trauma in children. Contemp. Neurosurg 1979;10:1-8.

51. BRUCE DA, SCHUT L. Concussion and contusion following paediatric head trauma. McLaurin RL Ed. Paed. Neurosurgery 1982;New York pp 301-308.

52. *BULL MJ, STROUP KB, GERHART S.* Misuse of car safety seats. Paediatrics. 1988;81:1:98-101.

53. BULLOCK R, TEASDALE G. Head injuries. ABC of Major Trauma. BMJ Cambridge 1991.

54. BULLOCK R, TEASDALE GM. Surgical management of traumatic intracranial haematomas. In Handbook of Clin. Neurol. Head Injury Vol 13(57) Ed. Braakman. Elsevier Science Publishers 1990 pp 249-295.

55. BURKINSHAW J. Head injuries in children. Arch. Dis. Child 1960;35:205-214.

56. BRUCE DA, ALAVIA A, BILANIUK L. Diffuse cerebral swelling following head injuries in children. The syndrome of malignant brain oedema. J. Neurosurg. 1981;54:170-178.

57. CALDER-HILL IM, SCHOLTAZ CLJ. Primary brain trauma in non-accidental injury. J. Clin. Pathol. 1984;37:1095-1100.

58. CARDOSA ER, PYPER R. Paediatric injury caused by off-road vehicles. Canad. J. Neurol. Sciences 1989;16(3):336-338.

59. CAFFEY J. Multiple fractures in long bones of infants suffering from chronic subdural haematoma. AJR 1946;456:163-173.

60. CAFFEY J. On the theory and practice of shaking infants; its potential residual effects of permanent trauma damage and mental retardation. Am. J. Dis. Child 1972:124:161-169.

61. CAMPBELL J B, COHEN J. Epidural haemorrhage and the skull of children. Surg. Gynaecol. Obstet. 1951;92:257-280.

62. CARMOLA J, HARRIS BS. Transient cortical blindness: still an overlooked syndrome. N. Eng. J. Med. 190;282-1325.

63. CHAN KH, MANN KS, YUE CP, et al. The significance of skull fracture in acute traumatic intracranial haematomas in adolescence: a prospective study. J. Neurosurg. 1990;72:189-194.

64. CHILDRENS ATTENDANCES IN ACCIDENT AND EMERGENCY DEPARTMENTS. British Paediatric Association and the British Association of Paediatric surgeons. LONDON 1985.

65. CHOUX M. Head injury. Brackman. Handbook of Clinical Neurology. Head Injury : 13:57 Elsevier publishers. Amsterdam 1996.

66. CHOUX M, GRISOLI F, PARAGUT J. Extradural haematomas in children, 104 cases. Childs Brain 1975;1:337-347.

67. CHRISTOPHERSON ER, SULLIVAN MA. Increasing the protection of newborn infants in cars. Paediatrics 1982;70:21-25.

68. CLINE DM, WHITLEY TW. Observation of head trauma patients at home: a prospective study of compliance in the rural South. Ann. Emerg. Med. 1988;15:91-94.

69. COHEN RA, KAUFMAN RA, MYERS PA et al. Cranial computed tomography in the abused child with head injury. AJR 1986;146:97-102.

70. COOK R J, DORSCH N W C, FEARNSIDE M R, et al. Outcome of prediction in extradural haematoma Acta. Neurochir (Wein) 1988;95:90-94.

71. COOPER PR, HO V. Role of emergency skull x-ray films in the evaluation of the head injured patient - a retrospective study. Neurosurgery. 1983;13:136-140.

72. COOPER PR. Head injury 3rd Edition. Williams and Wilkins pp 126.

73. COPPOLA R. Rapid death and extradural haematoma. American Family Practitioner 1973; 89-91.

74. CORDON TW. Skull roentgenography for patients with head trauma: the use of high yield criteria. Can. Med. Ass. J. 1981;124:584-589.

75. CORDOSO ER, PYPER R. Paediatric injury caused by off-road vehicles. Canad. J. Neurol. Sciences 1989;16(3):336-338.

76. COURVILLE C B. Traumatic intracranial haemorrhages, with special reference to the mechanisms of their production. Bull. Los Angeles Neurol. Soc. 1962;27:22-38.

77. CRAFT AW, SHAW DA, CARTILIDGE NEF. Head injuries in children. Brt. Med. J.1972;4:200-203.

78. CROUCHMAN MR. Head injury: how the community paediatrician can help. Arch. Dis. Child - 1990;65:1286-87.

79. CUMMINS BH, POTTER JM. Head injury due to falls from heights. Injury 1970;2(i):61-64.

80. CUMMINS RO. Clinical reasons for over use of skull radiographs. AJR. 1980;135:549.

81. CUSACK SG, ROBETSON CE. The value of helicopter transportation for trauma patients. Injury 1991;22:54.

82. DACEY RG, ALVES WM, JANE JA et al. Neurosurgical complications after apparently minor head injury. Assessment of risk in a series of 610 patients. J. Neurosurg. 1986;65:203-210.

83. DAVIS RL, HUGHES M, GUBLER DR et al. The use of cranial CT scans in the triage of paediatric patients with mild head trauma. Paediatrics 1995;2:345-349.

84. DEANE M. Child accident data: accessible and available? J.P.H. Med. 1993;16(3);226-228.

85. DeLACEY G. Reasons for requesting radiographs in the Accident & Emergency Department. Br. Med. J. 1979;1:1595-1597.

86. DELAMOTHE T. Wanted: guidelines that doctors will follow. Br. Med. J. 1993;307-318.

87. DEMETRA JD, LEE DN, PITCAIRN TK et al. Errors in young children's decisions, about traffic gaps. Br. J. Psychol. 1992;83:189-202.

88. DeSANTIS A, SGANZERIA E, SPAGNOLI, et al. Risk factors for late post traumatic epilepsy. Acta. Neurochin. 1992;55:64-67.

89. DeSMET AA, FRYBACK DG, THORNBURY JR. A second look at the utility of radiographic skull examination for trauma. AJR. 1979;132:95-97.

90. DHELLEMMES P, LEJEUNE J P, CHRISTIAENS J L, et al. Traumatic extradural haematoma in childhood experience with 144 cases. J. Neurosurg. 1986;62:861-864.

91. DIETRICK AM, BOWMAN MJ, GINN - PEASE ME. Paediatric head injuries: can clinical factors reliability predict an abnormality on computed tomography? Ann. Emerg. Med. 1993;22(10):1535-1540.

92. DUFFY T E, VANNUCCI R C. Metabolic aspects of cerebral anoxia in the foetus and new born. Brain and Fetal and Infant. The Hague 1977;316-323.

93. DUHAIME AC, GENNARELLI TA, THIBAULT LE et al. The shaken baby syndrome. J. Neurosurg. 1987:66:409-415.

94. DUNN RG, ASHER KN, RIVARA FP. Behaviour and expectations of child pedestrian. Paediatrics 1992;89(3):486.

95. DUUS BR, BUESEN T, KRUSES K V et al. Prognostic signs in the evaluation of patients with minor head injury. Br. J. Surg. 1993;8:988-991.

96. DYKE CG. The roentgenray diagnosis and treatment of disease of the skull and intracranial contents. In Palmer Nelsons Loose Leaf Medicine, NewYork. 1937, pp 185-214.

97. DYKES LJ. The whiplash shaken infant syndrome. What has been learned? Child Abuse neg 1986;10:211-221.

98. EDNA TH. Acute traumatic intracranial haematoma and skull fracture. Acta. Chir. Scand. 1983;149:449-451.

99. EINHORN A, MIZRAHI EM. Basilar skull fractures in children. The incidence of CNS infection and use of antibiotics. Am. J. Dis. Child. 1978;132:1121-1124.

100. EISENBREY AB. Retinal haemorrhage in the battered child. Child's Brain 1979;5:40-44.

101. ELJAMEL M S M, RUGMAN F P. Extradural haematoma associated with essential thrombocythaemia in a child, after relatively minor head injury. Injury 1989;20:236-244.

102. EPSTEIN F, WARD J D, BECKER D P. Medical complications of hed injury pp 390-407. In Cooper (Ed) Head Injury - Williams and Wilkins 1989.

103. EVANS KT. Radiology now - the radiologists dilemma. Br. J. Radiol. 1977;50:299-301.

104. EYES B, EVANS AF. Post traumatic skull radiographs. Time for a reappraisal. Lancet

105.FELSON B. Seminars in radiology. 1974;11(2):87-88.

106.FERRY P. Skull x-rays in paediatric trauma: a vanishing necessity. Paediatrics 1982;l69(2):237-8.

107.FEUERMAN T, WACKYM PA, GADE GF, et al. Value of skull radiology, computerised tomographic scanning and admission from observation in cases of minor head injury. Neurosurgery. 1988;22:449-453.

108.FIELD JH. Epidemiology of head injuries in England and Wales. HMSO London 1976.

109.FIFE D, DAIS J, TATE D et al. Fatal injuries to bicyclists: The experience of Dade County. J. Trauma 1983;23:745-755.

110.FISCHER RP, CARLSOW J, PERRY JF. Post concussive hospital observation of adult patients in a primary trauma centre. J. Trauma. 1981;21:920-924.

111.FISHER R G, KIM J K, SACHS E. Complications in the posterior fossa, due to occipital trauma: their operability. JAMA 1958;167:176-182.

112.FRANK Y, ZIMMERMAN R, LEEDS NMD. Neurological manifestations in abused children who have been shaken. Dev. Med and Child Neurol. 1985;27:312-316.

113.FREED HA. Skull x-rays criteria by the Food and Drug Administration some flaws and proposed modifications. Neurosurgery. 1980;7:636-638.

114.FRIEDE AM, AZZARA CV. The epidemiology of injuries to bicycle reiders Paed. Clinics of North America. 1985;32(i):141-151.

115.FRIEDE R. Developmental Neuropathology. Springer-Verlag New York 1975.

116.FIELD JH. Epidemiology of head injuries in England and Wales. HMSO London 1976.

117.FORD LE, McLAURIN R L. Mechanisms of extradural haematomas. J. Neurosurg. 1963;20:760-769.

118.FRANK Y, BERGERTS F E, TEW J M. Bilateral epidural haematomas. Surg. Neurol 1982;17:218-222.

119.FULLARTON GM, MACEWAN R, MACMILLAN R, et al. An evaluation of open scalp wounds. Acc. Emer. Med.1987;4:11-16.

120.GALBRAITH S. Misdiagnosis and delayed diagnosis in traumatic intracranial haematoma. Br. Med. J. 1976;1:1438-1439.

121.GALBRAITH S, SMITH J. Acute traumatic intracranial haematoma without skull fracture. Lancet. 1976;1:501-503.

122.GALLAGHER SS, FINISON K, GUYER B et al. The incidence of injuries amongst 87,000. Massachusetts children and adolescents. Results of 1980-81. Statewide childhood injury prevention programme surveillance system Am. J. Pub. Health 1984;74:1340-1347.

123.GARNIAK A, FEIVAL M, HERTZ M, et al. Skull x-rays in head trauma: are they still necessary. Euro. J. Radiol. 1986;6:89-96.

124.GEDDES DG, APPLETON IC. Use of restraints by pre-school children in cars. Arch. Dis. Child. 1982;57:849-850.

125.GENNARELLI T. The biomechanics of subdural haematoma. J. Trauma 1982:22:680-686.

126.GENNARELLI T A. Mechanistic approach to head injuries. Clinical and experimental studies of the important types of injuries, in head and neck injury criteria. N.T.S.B. Workshop, Washington DC. 1981;21.

127.GENNARELLI T. Emergency Department management of head injuries. Symposium on multiple trauma. Emerg. Med. Clinics North Am. 1984;2(4):749-764.

128.GENNARELLI TA, THIBAULT LE. Biomechanics of acute subdural haematoma. J. Trauma 1982;22:360-386.

129.GENNARELLI TA, THIBAULT LE. Biomechanics of head injury. In Wilkins RG Neurosurgery. McGraw-Hill New York 1985.

130.GENNARELLI TA, THIBAULT LE. Biomechanics of head injury. Neurosurgery M C Graw Hill New York 1985;1:217-222.

131.GENTLEMAN D, JENNET B. Audit of transfer of the unconscious head injured patient to a neurosurgical unit. Lancet 1990;335:327-330.

132.GENTLEMAN D, JENNETT B. Hazards of inter hospital transfer of comatose patients. Lancet 1981;2:853-855.

133.GENTLEMAN D, TEASDALE G. Adoption of the Glasgow Coma Scale in the British Isles. Br. Med. J. 1981;283:408.

134.GENTLEMAN D, TEASDALE G, MURRAY L. Cause of severe head injury and risks of complications. Br. Med. J. 1986;292:449.

135.GLEADHILL DNS, THOMPSON JY, SIMMS P. Can more effective use be made of x-ray examination in the accident and emergency department? Br. Med. J. 1987;294:304-309.

136.GLEESON AP, BEATTIE TF. Post traumatic transient cortical blindness in children : a report of four cases and a review of the literature. Arch. Emerg. Med 1994;11:250-253.

137.GODANO V, SERACCIOLI A, SERVADEI et al. Intracranial lesions of surgical interest in minor head injuries in paediatric patients. Child Nerv. Syste. 1992;8:136-138.

138.GORMAN D. Were you knocked out? Yes, but I wasn't admitted - Arch Emerge. Med. 1985:2:127-129.

139.GORMAN D. MD Thesis. Head injury admission policy. University of Edinburgh. 1985.

140.GORMAN DF. The utility of post traumatic skull x-rays. Arch Emerg. Med. 1987;4:141-150.

141.GRAHAM DI, FORD I, ADAMS JH et al. Fatal head injury in children. J. Clin. Pathol. 1989;42:

142.GREENBLATT SH. Post traumatic transient cortical blindness. Association with migraine and a seizure diathesis JAMA 1973;225:1073-1076.

143.GREEN MA, LIEBERMAN G, MILROY CM and PARSONS MA. Ocular and cerebral trauma in non accidental injury in infancy : underlying mechanism and implications for paediatric practice. Brit.J.Ophal.1996;80:282-287.

144.GUICHON DM, MYLES ST. Bicycle injuries: One year sample in Calgary. J. Trauma. 1976:15:504-506.

145.GURDJIAN ES, GURDJIAN ES. Acute head injuries - collective reviews. Surg. Gynae. Obstetrics 1978;156:805-820.

146.GURDJIAN E S, LISSNER H R. Deformation of the skull in head injury as studied by the "stress coat" technique. Am. J. Surg. 1947;73:269-280.

147.GUIDELINES FOR INITIAL MANAGEMENT AFTER HEAD INJURY IN ADULTS. Suggestions from a group of neurosurgeons. Br. Med. J. 1984;288:983-988.

148.GULY HR. Missed diagnoses in the accident and emergency department. Injury. 1984;15:403-406.

149.GUPTA SM, CHANDRA J, DOGRA TD. Blunt forces related to the heights of a fall Am. J. Forensic Med. Pathol 1982;3(I):35-43.

150.GUTHKELCH A N. Infantile subdural haematoma and its relationship to whiplash injuries. Br. Med. H. 1971;430-431.

151.GUYER B, TALBOT AM, PLESS IB. Pedestrian injuries in children and youth. Paediatric Clin. North Am. 1985;32:163-174.

152.HADLEY MN, SONNTAG VKH, REKATE HL, and MURPHY A. The infant whiplash shaken injury syndrome: a clinical and pathological study. Neurological 1989,24;4:536-539.

153.HAHN YS, CHYUNG C. BARTHEL MJ et al. Head injuries in children under 36 months of age. Demography and outcome. Childs. Nerv. System. 1988;4:34-40.

154.HAHN YS, FUCHS S, FLANNERY AM, et al. Factors influencing post traumatic seizure in children. Neurosurgery 1988;22:864-867.

155.HALLER JA. Paediatric trauma - the number 1 killer of children. JMA 1983:249(I):47.

156.HARAD FT, KERSTEIN MD. Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. J. Trauma 1992;23:359-363.

157.HARRIS JH. High yield criteria and skull radiography. J. Am. Coll. Emerg. Physicians

158.HARRISON DW, WALLS RM. Blindness following minor head trauma in children. A report of two cases with a review of the literature J. Emerg. Med. 1990;8:21-24.

159.HARWOOD NASH. Abuse to the paediatric nervous system. A.J.N.R. 1992;2:568-575.

160.HARWOOD NASH DG, HENDRICK EB, HUDSON AR. The significance of skull fracture in children. A Study of 1187 patients. Radiology 1971;101:151-155.

161.HASS DC, PINEDA GS, et al. Juvenile head trauma syndromes and their relationship to migraine. Arch. Neurol (Chicago) 1975;32:727-730.

162.HAYWOOD R. Management of children with head injury - A personal view - Arch. Dis. Child 1987;62:1283-1286.

163.HELFER RE, SLOVIS TL, BLACK M. Injuries resulting when small children fall out of bed. Paediatrics 977;60:533-535.

164.HENDRICK EB, HARRIS L. Post-traumatic epilepsy in children. J.Trauma.1968;8:547-556.

165.HENDRICK EB, HARWOOD NASH DGF, HUDSON AR. Head injuries in children - a survey of 4,465 consecutive children at the Hospital for Sick Children, Toronto. Neurosurg. 1964;11:46-65.

166.HENNES H, LEE M, SMITH D et al. Clinical predictors of severe head trauma in children. Ann. J. Dis. Child. 1988;8:257-271.

167.HOBBS CJ. Skull fracture and the diagnosis of abuse. Arch. Dis. Child. 1984;59:246-252.

168.HOCKSTETLER K. BEALS RD. Transient cortical blindness in a child. Ann. Emerg. Med. 1987;16(S)218-219.

169.HOFF JT, BREWIN U HS. Antibiotics for basilar skull fracture. J. Neurosurg. 1976;44:649.

170.HOFFMAN E. Mortality and morbidity following road accidents. Ann. R. Coll. Surg. Engl. 1976;58:233-240.

171.HOFFMANN ER, PAYNE A, PRESCOTT A. Childrens estimates of vehicle approach times. HUM. Factors. 1980;20:235.

172.HOFFMAN JR. CT for head trauma in children. Ann. Emerg. Med. 1994;24:713-715.

173.HOOPER R. Observations on extradural haemorrhage. Br. J. Surg. 1959;47:71-87.

174.HORTON JM. Ch 11 The anaesthetists contribution to the care of head injuries. Br. J. Anaesth. 1976;3:767-771.

175.HUGENHOLTZ H, IZUKAWAD, SHEAR P, et al. Vomiting in children following head injury. Childs Nerv. Syst. 1987;3:266-270.

176.HUME ADAMS J. In handbook of Clinical Neurology Vol. 13(57) Head Injury Ed. Braakman. Elsevier Science Publishers 1990 AMSTERDAM.

177.HUMPHREYS RP, HENDRICK EB, HOFFMANN H. The head injured child who talks and dies. Childs Nerv. Syste. 1990;6:139-142.

178.IGNALZI RJ, VANDER ARK GD. Analysis of the treatment of basilar skull fracture with and without antibiotics. J. Neurosurg. 1975;43:721-726.

179.ILLINGWORTH CM, NOBLE D, BELL D et al. 150 Bicycle injuries in children. A comparison with injuries due to other causes of injury 1981;13:7-9.

180.INGRAHAM FD, CAMPBELL JB, COHEN J. Extradural haematoma in infancy and childhood. JAMA 1949;140(12):1010-1013.

181.JACKSON H. Children in Accident and Emergency Departments Br. Med. J. 1985-4-90.

182.JACKSON RH. Children in Accident & Emergency Departments Br. Med. J. 1979;4:91.

183.JAMA 1949;141:1010-1013.

184.JAMIESON K G, YELLAND J D N. Extradural haematoma 167 cases. J. Neurosurg. 1968;29:

185.JEFFRIES NJ, BRISTOW A. Long distance inter-hospital transfer - helicopter transport for intensive care patients. Br. J. Int. Care. 1991;2:197-202.

186. JEFFREYS RV, JONES JJ. Avoidable factors contributing to the death of head injury patients in general hospitals in the Mersey Region. Lancet 1981;459-461.

187. JENNETT B (unpublished).

188.JENNETT B, Epilepsy after non-missile head injuries. William Heinmann London 1975.

189.JENNETT B. Epilepsy after non missile head injuries. William Heinemann, Chicago. 1975.

190.JENNETT B. Management of head injuries 2nd Ed. Philadelphia FA Davis 1990.

191.JENNETT B, BOND MR. Assessment of outcome after severe brain damage : a practical scale. Lancet 1975;1:480-487.

192.JENNET B, CARLIN J. Preventable mortality and morbidity after head injury. Injury 1978;10:31-39.

193.JENNETT B. MacMILLAN R. Epidemiology of head injury. Br.Med. J. 1981;282:101-104.

194.JENNETT B. MILLER JD. Infection after depressed fracture of the skull. Indication for management of non missile injuries. Neorosurg. 1974;41:208-216.

195.JENNETT B, MacPHERSON P. Implication of scanning recent head injured patients in general hospitals. Clin. Radiol. 1990.42:88-90.

196.JENNETT B, TEASDALE G. Epidemiology of head injury. Br. Med. J. 1981;282:101.

197.JENNETT B, TEASDALE G. Management of head injuries. FA Davis, Philadelphia 1981.

198.JENNETT B, TEASDALE G, GALBRAITH S, et al. Severe head injuries in three countries. J. Neurol. Neurosurgery and Psychiatry 1977;40:291-298.

199.JENNETT B, TEASDALE G, GALBRAITH S, et al. Severe head injuries in three countries data bank. J. Neural. Neurosurg. Psychiatry 1977;40:201-208.

200.JHAJAR J, HARIRI RJ. Management of paediatric head injury. Paed. Clin. North Am. 1992;39:5:1093-1125.

201.JOINT STATEMENT Children's attendances at Accident and Emergency Departments. British Paediatric Association, British Association of Paediatric Surgeons and Casualty Surgeons Association LONDON 1987

202.JONES JJ, JEFFREYS RV. Relative risks of alternative admission policies for patients with head injuries. Lancet 1981;2:850-853.

203.KALSBECK WD, McLAURIN RL, HARRIS BSH et al. The National head and spinal cord survey. Neursurgery, 1980;53:19-31.

204.KAYE EM, HERSKOVITZ J. Transient post-traumatic cortical blindness : brief v prolonged syndromes in childhood. J. Child. Neurol 1986;1(3):206-210.

205.KEMPE CH, SILVERMAN FN, STEELE BF et al. The battered child syndrome. J.A.M.A. 1961;181:17-24.

206.KINGSLEY D, TILL K, HOARE R. Growing fractures of the skull. J.Neurol. Neurosurg. Psychiatry. 1978;41:312-318.

207.KLAUBER MR, BARRETT-CONNOR E, HOFFSTETTER CR, et al. A population based study of non fatal childhood injuries. Prev. Med. 1986;15:139-149.

208.KLAUBER MR, BARRETT-CONNOR E, MARSHALL LF, BOWERS SA. The epidemiology of head injury. Am. J. Epidemiol. 1981;113:500-509.

209.KLAUBER MR, MARSHALL LF, LUERSSEN TQ, et al. Determinants of head injury mortality: the importance of the low risk patient. Neurosurgery 1989;24:31-36.

210.KLEINMAN PK. Diagnostic imaging of child abuse - Williams and Wilkins, BALTIMORE, 1987, pp 159-199.

211.KLEINMAN PK, SPEVAC M. Soft tissue swelling and acute skull fracture. J. Paed. 1992;121:737-739.

212.KLONOFF H. Head injuries in children, predisposing factors, accident conditions, accident proneness and sequelae. Am. J. Pub. Health 1967;61:2465-2477.

213.KLONOFF H, ROBINSON GC. Epidemiolgy of head injuries in children - a pilot study. Canad. Med. Ass. J. 1967;96:1308-1311.

214.KLONOFF H, ROBINSON GC. Epidemiology of head injuries in children - a pilot study - Canad. Med. Asso. J. 1967;96:1308-1311.

215.KLONOFF H, THOMPSON GB. Epidemiology of head injury in adults. Canad. Med. Ass. J. 1969;100:235-241.

216.KOHI YM, MENDELOW AD, TEASDALE GM. Extracranial insults and outcome in patients with acute head trauma. Relationship to Glasgow Outcome Scale - Injury 1984;16:25-29.

217.KOSARY IZ, GOLDHAMMER Y, LERNER MA. Acute extradural haematoma of the posterior fossa. J J Neurosurg. 1966;24:1007-1012.

218.KRAUS JF, FIFE D, CONROY C. Incidence severity and outcomes of brain injuries involving bicycles. Am. J. Public Health 1987;77(I):76-78.

219.KRAUS JF, FIFE D, COX, et al. Incidence, severity and external causes of paediatric head injury. A.J.D.C. 1986;140:687-693.

220.KREIL RL, KRACH LE, DANSER LA. Closed hand injury : comparison of children younger and older than 6 years of age. Paediat. Neurol. 1989;5:296.

221.KUMAR R, WEST C, QUIRK C et al. Do children with severe injury benefit from intensive care? Childs. Nerv. Syst. 1991;7:299.

222.LARSEN KT, KOZIOL DF. High yield criteria and emergency department skull radiography - two community hospitals experiences. JACEP. 1979;8:393-395.

223.LEONIDAS JC, TING W, BINKIEWICZ A, et al. Mild head trauma in children when is a roentgenogram necessary? Paediatrics. 1982;69:139-143.

224.LEVIN HS, EISENBERG HM. Neuropsychlogical outcome of closed head injury in children and adolescents. Childs Brain 1975;5:281-292.

225.LLOYD DA, CAR TH, PATERSON M, BUTCHER CK, ROE D. Lancet 1997;349:821-824. Predictive Value of Skull Radiography for Intracranial Injury in Children with Head Injury.

226.LOBATO RD. Post-traumatic brain swelling. (Review). Advances & Technical Standards in Neurosrugery;1993:20:3-38

227.LOWNE R, ROBERTS A, ROY P et al. The effects of UK seat belt legislation on restraint usage by children. Soc. Automotive Engineers 1984(840526).

228.LOU HC. Developmental Neurology. New York. Praven Press. 1982.

229.LUDWIG S, WARMAN M. Shaken baby syndrome: a review of twenty cases Ann. Emerg. Med. 1984;13:104-107.

230.LUERSSEN TG, KLAUBER MR, MARSHALL LF. Outcome from head injury related to patients age. A longitudinal study of adult and paediatric patients. J. Neurosurg. 1988;68:409.

231.MACIVOR IN, FREW IJC, MATHESON JG. The role of respiratory insufficiency in the mortality of severe head injuries. Lancet 1958;1:390-393.

232.MACKAY M. Mechanisms of injury and biomechancs. Vehicle design and crash performance. World J. Surg.1992;16:420-427.

233.MacKELLAR A. Head injuries in children and implications for there prevention. J. Paediatric Surg. 1989;24(6):577-579.

234.MacPHERSON P, JENNETT B, ANDERSON E. CT scanning and the surgical treatment of 1551 head injured patients admitted to a regional neurosurgical unit. Clin. Radiol 1990;42: 85-97.

235.MAITRA AK. Patterns of care for mild head injuries in the accident and emergency department. Brit. J. Clin. Pract. 1981, 35:385-389.

236.MANAGEMENT OF ACCIDENTS IN CHILDHOOD; Fit for the future. COURT REPORT HMSO LONDON 1975.

237.MANN K S, CHAN K H, YUE C P. Skull fractures in children. Their assessment in relation to developmental changes, and acute intracranial haematoma - Childs Nerv. Syst. 1986;2:258-261.

238.MARSDEN AK. A head injury record form for accident unit. J. World Ass. Emerg. Disat. Med 1987;3:46-48.

239.MARSDEN AK, PRICE DJ. A head injury record for an accident unit. J. World Ass. Emerg. Dis. Med 1987;3:46-48.

240.MARSDEN AK, STANILAND JR, PRICE DJE. Development of a coma scale for emergency care. J.W.A.E.D.M. 1984;1:249-251.

241.MARSHALL LF, TOOLE BM, BOWERS SA. The National Trauma Data Bank. Patients who talk and deteriorate; implications for treatment. J. Neurosurg. 1983;59:285-288.

242.MASTERS SJ. Evaluation of head trauma: efficacy of skull films. AJR 1980;135:539-547.

243.MASTERS SJ, McCLEAN PM, ACARESE JS, et al. Skull x-ray examination after head trauma. Recommendations of a multidisciplinary panel and validation study. N. Eng. J. Med.

244.MASUZAWA H. Post traumatic vomiting. Shonika 1977;18:53-57.

245.MATSON DD Neurosurgery of infancy and childhood. Springfield II Charles Thomas 1968.

246.MAY RJ, GAISL M, NOERE S et al. Baby walkers. An understated hazard for our children. Europ. J. Paed. 1994:77:76-78.

247.MAYER TA, WALKER ML. Paediatric head injury : the critical role of the emergency physician. Ann. Emerg. Med. 1985;14:1178-1184.

248.MAZZA C, PASQUALIN A, FERRIOT G, DAPIAN R. Traumatic extradural haematomas in children: experience with 62 cases. Acta Neurochir. 1982;65:67-80.

249.McKISSOCK W, TAYLOR JC, BLOOM WH, et al. Extradural haematoma, observations on 125 cases. Lancet 1960;2:167-172.

250.McLAURIN R H, FORD L E. Extradural haematoma statistical survey of 47 cases. J. Neurosurg. 1964;27:264-2711.

251.McKENZIE K G. Extradural haematoma in the posterior fossa. Brit. J. Surg. 1938;26:346.

252.McLEAN PM, JOSEPH LP, RUDOLPH H. Skull films radiography in the management of head trauma. Ann. Emerg. Med. 1984;13:607-611.

253.MENDELOW AD. Risks of intracranial haematoma in head injured adults. Br. Med. J. 1983;287:1173-1186.

254.MENDELOW AD, CAMPBELL DA, JEFFREY RR, et al. Admission after mild head injury: Benefits and costs. Br. Med. J. 1982;285:1530-1532.

255.MENDELOW AD, CAMPBELL DA, TSEMENTIS et al. Prophylactic antimicrobial management of compound depressed skull fracture. J. Royal Coll. Surg. Edinburgh 1983;28:80-83.

256.MENDELOW AD, KARMI NZ, PAUL KS et al. Extradural haematoma: effect of delayed treatment Br. Med. J. 1979;1:1240-1242.

257.MENDELOW AD, TEASDALE GM, JENNETT B, et al. Risks of intracranial haematoma in adults. Br. Med. J. 1983;287:1173-1186.

258.MERINO de VILLASANTE, TAVERS JM. Computerised tomography in acute head trauma 1976;126:765-768.

259.MILLER BECKER DP, WARD JD et al. Significance of severe injury. J. Neurosurg. 1977;47:503-516.

260.MILLER JD, JENNETT B. Complications of depressed fractured skull. Lancet 1968;2:991 1995.

261.MILLER J D, SWEET R C, NARAYAN R K et al. Early results of the injured brain. JAMA 1978;240-439-442.

262.MITCHELL KA, FALLAT ME, RAQUE GH et al. Evaluation of minor head injury in children. Paedi. Surg. 1994;29(7)851-854.

263.MONRO HM, LAYCOCK JRD. Interhospital transfer. Br. J. Int. Care 1993;2:210-214.

264.MORAN SG, McCARTHY MC, UDDIN DE et al. Prediction of positive CT scans in the trauma patient with minor head injury. American Surgeon 1994;607:533-535.

265.MORGAN G. Transport of the critically injured Brit. J. Hosp. Med. 1990;44:222.

266.MORRAY JP, TYLER DG, JOWES TK et al. Coma Scale for use in brain injured child. Crit. Care. Med. 1984;12(12);1018-1020.

267.MOYLAN JA, FITZPATRICK KT, BEYER J et al. Factors improving survival in multi-system trauma. Ann. Surg. 1988;207:679-685.

268.NATIONAL CENTRE ON CHILD ABUSE AND NEGLECT. Study of National incidence and prevalence of child abuse and neglect. U.S. Department of Health and Human Services 1988.

269.NEUBAUER U J. Extradural haematoma in the posterior fossa: 12 years experience with CT Scan. Acta. Neurochir (Wein)1978:105-111.

270.NELSON WE, JANE JA, GIERK JH. Minor head injury in sports. A new system of classification and management. Physician Sports Med. 1984;12:103.

271.NICOL JW, JOHNSTONE AJ. Temporal bone fractures in children: a review of 34 cases. J. Acc. Emerg. Med. 1994;11(4);218-222.

272.NIMITYONGSKUL P, ANDERSON LD. The likelihood of injuries when children fall out of bed. J. Paed. Orthop. 1987;7:184-186.

273.OGAWA T. Emotion and ketone body metabolism in children. Jap. J. Paed. 1965;1561-1571.

274.OGAWA T, MURAYAMA A, et al. Clinical and experimental observations of central regulation of ketone body metabolism in children. Jap. J. Paed. 1972;25:847-859.

275.O'HARE AE and EDEN OD. Bleeding Disorders or non-accidental injury. Arch. Dis. Child 1984:59;860-864.

276.OKA H, KAKO M, MATSUSHIMA M et al. Traumatic spreading depression syndrome. Brain 1977;100:287-298.

277.OMMAYA A K, FAAS F, YARNELL P R. Whiplash injury and brain damage: an experimental study J. Ann. Med. Assoc. 1968

278.O'ROURKE NA, COSTELLA F, YELLAND JD, STUART GG. Head injuries to children riding bicycles. Med. J. Aust. 1987;146(12):1619-1621.

279.PAEDIATRIC ADVANCED SUPPORT GROUP. BMJ Publishing Group. London 1993.

280.PAEDIATRIC LIFE SUPPORT. American Heart Association - USA.

281.PAEDIATRIC LIFE SUPPORT GROUP. Advanced Life Support. The practical approach. BMJ Publications 1993.

282.PARTINGTON MW. The importance of accident proness in the Aetiology of head injuries in childhood. Arch. Dis. Child 1960;25:215.

283.PEACOCK W J, RAIMONDI A J, CHOIX M, et al. The post-natal development of the brain and its coverings. Head injuries in the new born and infant. Springer-Verlao - New York 1986.

284.PEARN JH. Fatal motor vehicle accidents involving Australian children. Aust. Paediatr. 1978;14;74-77.

285.PHILLIPS LA. Comparative evaluation of the effect of a high yield criteria list upon skull radiography. JACEP 1979;8:106-109.

286.PHONPRASERT C, SUWANWELA C, et al. Extradural haematoma; an analysis of 138 cases. Trauma 1980,20,8;679-683.

287.PIETRAZAK M, JAGODA A, BROWN L. Evaluation of minor head trauma in children in younger than two years. Ann. J. Emerg. Med 1991;2:153-156.

288.PICKLES W. Acute general oedema of the brain in children with head injuries. N. Eng. J. Med. 1950;242:3607-611.

289.POTTER J. The practical management of head injuries. Lloyd-Luke, London 1974.

290.PUPURA DP. Dendritic differentiation in the human cortex: Normal and aberrant development patterns. Advances in Neurology Vol 12 1975.

291.QUAYLE KS, JAFE MD, KUPERMANN N, et al. Paediatrics Volume 99, 1997. Title: Diagnostic Testing for Acute Head Injury in Children: When are Head Computed Tomography and Skull Radiographs indicated?

292.RAIMONDI AJ, HIRSCHAUER J. Head injury in the infant and toddler. Coma scoring outcome scale. Childs Brain 1984;11:12.

293.RANSCHOFF J, FLEISHER A. Head injuries JAMA 1975;234:861-864.

294.RAPHAELY RC, SWEDLOW DB, DOWNES JJ, BRUCE DA. Management of severe paediatric trauma. Symposium on paediatric intensive care. Paed Clinics North Am. 1980;57:715-727.

295.REDDY R D, SATHYNARAYANK K, RAO B D. Extradural haematoma J Ind. Med Ass. 1977;69:232-123

296.REIDER MJ, SWARTZ C, NEWMAN J. Patterns of baby walker use and walker injury. Paediatrics 1986;78:488-493.

297.REILLY PL, ADAMS JH, GRAHAM DI, JENNETT B. Patients with head injury who talk and die. Lancet 1975;2:375-377.

298.REYNOLDS R, COHEN D. Children bicycles and head injury Med. J. Aust. 1987;146:615.

299.REYNOLDS BM, BALSANO NA, REYNOLDS FX. Falls from heights. A surgical experience of 200 cases. Anm. Surg. 1971;174(2):304-308.

300.REIBER GD. Fatal falls in childhood. How far must children fall to sustain fatal head injuries. Report of cases and a review of the literature. Am.J.Forensic Med. 1993;14:201-207.

301.RICHARDS P. Severe head injury. The first hour. Br. Med. J. 1986;293(3);643.

302.RIMEL RW, GIORDANI, BRTH JT et al. Moderate head injury. Competing the clinical spectrum of brain trauma. Neurosurgery 1982;11:344-351.

303.RIVARA F. Bicycle related injuries. Data from the National Electronic Injury Surveillance System M.M.W.R. 1987;36:269-271.

304.RIVARA FP. Epidemiology of childhood injuries II. Sex differences in injury rates. Am J. Dis. Child. 1982;136:502-506.

305.RIVARA FP. Traumatic deaths of children in the USA. Current available prevention strategies. Paediatrics 1985;75:456-462.

306.RIVARA FP, BARBER DM. Demographic analysis of childhood pedestrian injuries. Paediatrics 1985;76:375-381.

307.RIVARA FP, KAMITSUKA MD, QUAN L. Injuries to children younger than one year of age. Paediatrics 1988;81:(1)93-97.

308.RIVAS JJ, RAMIRO D, LOBATO M D, et al. Extradural haematoma: analysis of factors influencing the courses of 161 patients. Neurosurgery 1988;23:44-51.

309.RODRIGUEZ A, LOZARO KA, Del POZOA et al. Post traumatic cortical blindness - Int Ophthalmol 1993;17(5):277-283.

310.ROSE J, VALTONEN S, JENNETT B. Avoidable factors contributing to death after head injury. Br. Med. J. 1977;2:615-618.

311.ROSENTHAL BW, BERGMAN I. Intracranial injury after moderate head injury. J Paediatrics 1989.

312.ROSMAN NP, OPPENHEIMER EY, O'CONNOR JF. Emergency management of paediatric head injuries. Emerg. Med. Clinics of North Am. 1983;1:141-148.

313.ROSMAN R G. Head injury Basic and Clinical aspects New York 1982.

314.ROSS P, ROS MA. Should patients with normal CT scans following minor head injury be hospitalised for observation? Paediatric Emerg. Care 1989;5(4):216-220.

315.ROWLEY G, FIELDING K. Reliability and accuracy of the Glasgow Coma Score with experienced and inexperienced users. Lancet 1991;337-535-538.

316.ROYAL COLLEGE OF SURGEONS ENGLAND. Report of the Working Party on Head injuries. London 1986.

317.ROYAL COLLEGE OF SURGEONS ENGLAND. Report of the Working Party on the Management of Patients with Major Injuries. LONDON 1988.

318.ROYAL COLLEGE OF RADIOLOGISTS. Costs and benefits of skull radiography for head injury. Lancet 1981;2:791-795.

319.ROYAL COLLEGE OF RADIOLOGISTS. Patient selection for skull radiography in the uncomplicated head injury. Lancet 1983;1:115-118.

320.SADIK A R, EPSTEIN S, RANSOHOFF J. Epidural haematoma in the posterior fossa. New York States J. Med. 1978;78:801-803.

321.SAINSBURY CPQ, SIBER JR. How long do we need to observe head injuries in hospital. Ar. Child. 1984;59:856-859.

322.SALCMAN M, SCHEPP RS, DUCHER TB. Calculated recovering rates in severe head trauma. Neurosurgery 1981;8:301-308.

323.SANDE GM, GALBRAITH SL, MCLATCHIE G. Infection after depressed fracture in the West of Scotland. Scott. Med. J. 1978;25:227-229.

324.SAUNDERS CE, COTAR, BARTON CA. Reliability of home observation for victims of mild closed head injury. Ann. Emerg. Med. 1986;15:91-94.

325.SCHILLER WR, KNOW R, ZINNECKER H et al. Effect of helicopter transport of trauma victims and survival in an urban trauma centre. J. Trauma 1988;28:1127-1134.

326.SCHIKTNER MT. A syndrome of cerebral concussion in children. Review of a particular type of head injury in 37 patients. Brain 1949;100:287-298.

327.SEELIG JM, GREENBERG RP, BECKER DP, et al. Traumatic acute subdural haematoma major reduction in mortality in comatose patients treated within 4 hours. Ne. Eng. J. Med. 1981;304:1511-1542..

328.SEELIG JM, MARSHALL LF, TOUTANT SM et al. Traumatic acute epidural haemaeoma unrecognised high lethality in comatose patients. Neurosrugery 1984;6-7-620.

329.SERVADEI F, GALASSI E, CALBUCCI F. Intracranial lesions of surgical minor head injuries; a report of 58 cases in paediatric and adult age. Br. J. Neurosurg. 1992;9:30-35.

330.SHAPIRO K. Paediatric head trauma, New York. Futura Publishing Company 1983. Skull Fracture? Arch. Emerg. Med. 1986, 3:91-92.

331.SHARPLES PM, STOREY A, AYNESLEY-GREEN A, et al. Causes of fatal childhood accidents, involving head injury in the North Region. Br. Med. J. 1990;301.

332.SHAW MDM, GALBRAITH S. Penetrating air gun injuries of the head. Br. J. Surg. 1977;64:221-224.

333.SIMPSON DA, COCKINGHAM RA, HANIEH A, et al.' Head injuries in infants and young children. The value of the paediatric coma scale. Childs. Nerv. Syst. 1991;7:183.

334.SIMPSON DA, REILLY D. Paediatric Coma Scale. Lancet 1982;2:450.

335.SINAL SH, BALL MR. Head trauma due to child abuse; serial computerised tomography in diagnosis and management. Southern med. J. 1987;80:12:1505-1512.

336.SNOEK J, JENNETT, ADAMS JH, et al. Computerised tomography after recent severe head injury in patients without acute intracranial haematoma. J. Neuro. Neurosurg. Psychiatry 1979;42:215-225.

337.SNOEK JW, MINDERHOUD JM, WILMINK JP. Delayed deterioration following mild head injury in children. Brain 1984,107:15-36.

338.SNOEK JW, MINDERHOUD JM, WILMINK JP. Delayed deterioration following mild head injury in children. Brain 1984,531(1):106-16.

339.SOKOLOFF L. Post natal maturation of the local cerebral circulation. In Berensberg Brain Foetal and Infant. The Hague 1976;324-329.

340.SOTIROPOULOS SV, JACKSON MA, TREMBLA, et al. Childhood lawn dart injuries. Ann. J. Dis. Child. 1990;133(9):980-982.

341.SPAITE DW, MURPHY M, CRISS E A et al. A prospective analysis of injury severity among helmeted and non helmeted bicyclists involved in collision with motor vehicles. J. Trauma 1991;31:1510-1516.

342.STALHAMMER DA. In Handbook of Clinical Neurology Vol 13 (57)Head Injury Ed. Braakman. Elsevien Science Publications 19990. AMSTERDAM.

343.STANDING COMMITTEE ON ACCIDENTS IN CHILDHOOD OF THE BRITISH PAEDIATRIC ASSOCIATION. Memorandum on the management of accidents in childhood. Br. Med. J. 1967;3:103-105.

344.STEEDMAN DJ, GORDON M. CSF rhinorrhoea: significance of the glucose oxidose strip.test Br. J. Acc. Surg. 1987;18:327-328.

345.STEIN SG, O'MALLEY KF, ROSS SE. Is routine computed tomographic scanning too expensive for mild head injury? Ann. Emerg. Med. 1991;20:1286-1289.

346.STEIN SG, ROSS SE. The value of computerised tomographic scanning in patients with low risk head injuries. Neurosurg. 1990;26:638-640.

347.STEIN S G, YOUNG G S, RAYMOND MA et al. Delayed brain injury after head trauma - Neurosurgery 1992;30;2:160-165.

348.STEINBOCK P, FLODMARKG, MARTENS D et al. Management of simple depressed skull fracture in children. J. Neurosurg. 1987;66:506-510.

349.STEVENSON MR, LOS K, LAING BA et al. Childhood pedestrian injuries in the Perth Metropolitan area Med. J. Aust. 1992;156:234-238.

350.STOFFMAN J, BASS M, FOX AM. Head injuries related to the use of baby walkers. Can. Med. Ass. J. 1984;78:488-493.

351.STRANG I, MacMILLAN R, JENNETT B. Head injuries in accident & emergency departments at Scottish hospitals. Injury 1978;10:154-159.

352.SWAIN AH. Radiological audit. Change in casualty officers performance during tenure of post. Brit. J. Acc. Emerg. Med 1986;1(2):5-9.

353.SWANN IJ, MACMILLAN R, STRANG I. Head injuries in an inner city accident and emergency department. Injury 1981;12:274-178.

354.SNYDER RC. Human tolerances to extreme impacts in free fall. Aerospace medicine 1963;34(8);695-709.

355.TANDON PN, BANERJI AK, BHATIA R, et al. Craniocerebral erosion, growing fracture of the skull in children. Acta Neurochir. 1987;88:1-9.

356.TANZ RR, CHRISTOFFEL KK. Pedestrian injury. The next motor vehicle challenge. Ann. J. Dis. Child 1985:139:1187-1190.

357.TEASDALE G. Head injuries are badly managed in accident and emergency departments and neurosurgeons are partly to blame. Arch. Emerg. Med. 1984;3:123-134.

358.TEASDALE GM, GALBRAITH S. Head injuries in Robb and Smith's Operative Surgery 4th Ed. Butterworths LONDON 1989.

359.TEASDALE GM, GALBRAITH S, MURRAY et al. Management of traumatic intracranial haematoma. Br. Med. J. 1982;285:1695-1697.

360.TEASDALE G, JENNETT B. Assessment of coma and impaired consciousness; a practical scale. Lancet 1974:2:81-83.

361.TEASDALE GM, MURRAY G, ANDERSON E, et al. Risks of acute intracranial haematoma - Implications for managing head injuries. Br. Med. J. 1990;300:363-367.

362.TEPAS JJ, DISCALA C, RAMENSOFSKY ML. The paediatric trauma score as a predictor of severity of injury : An objective assessment. Trauma 1988;28:425-429.

363.TEPAS JJ, DISCALA C, RAMENSOFSKY ML. Mortality and head injury. The paediatric perspective. J. Paediatr. Surg. 1990;25:92.

364.THE CARE OF THE INJURED CHILD. HOSPITAL FOR SICK CHILDREN TORONTO 1975.

365.THE MANAGEMENT OF PATIENTS WITH MAJOR INJURIES. Recommendations of the Royal College of Surgeons Working Party. London 1988.

366.THILLAINAYAGAM K, MacMILLAN R, MENDELOW AD, et al. How accurately are fractures of the skull diagnosed in an accident and emergency department? Injury. 1987;18:319-321.

367.THOMSON R. Construction and Use of Guidelines. Presc. J. 1999:Vol 39 No 3:180-187.

368.THORNBURY JR, CAMPBELL JR, MASTERS SJ, et al. Skull fracture and the low risk of intracranial sequelae in minor head trauma. AJR. 1984;143:661-664.

369.TREADWELL L, MENDELOW D. Audit of head injury management in the Northern Region. Br. J. Neurosurg 1994;3(3):136-140.

370.TUNTURI T, NIEMAN R, PATRALA H, et al. Head injuries and skull radiography: clinical factors predicting a fracture injury 1982;13:478-483.

371.VINCENT CA, DRISCOLL PA, AUDLEY RJ, et al. Accuracy of detection of radiographic abnormalities by junior doctors. Arch. Emerg. Med. 1985;5:101-109.

372.VOGEL S. The anatomy of Head Trauma. 1976;321-323.

373.WADDELL G, SCOTT PDR, LEES NW et al. Effects of ambulance transport in critically ill patients. Br. Med. J. 1975;1:386-389.

374.WALDRON J, HURLEY SEJ. Temporal bone fractures: a clinical diagnosis Arch. Emerg.Med. 1988;5:146-150.

375.WARDROPE J, CHENNELLS PM. Should casualty radiographs be reviewed? Br. Med. J.1985;290:1638-1640.

376.WARREN RA, FERGUSON DG. Why do accident and emergency doctors request x-rays? Arch. Emerg. Med. 19894;3:143-150.

377.WEISS BD. Bicycle helmet use by children. Paediatrics 1986;77:677-679.

378.WESTON PAM. Admission Policy for patients following head injury. Br. J. Surg. 1981;68:633-6344.

379.WESTON PAM. Skull x-ray policy. Lancet. 1978;2:792.

380.WRIGHT L. Traumatic haematomas of the posterior fossa. J. Neurosurg. 1966;25:402-409.

381.YAMAMOTO T, OGATA M. The mechanism of recurrent vomiting after head injury in children. Arch. Jpn. Chir. 1984;53(1):106-116.

382.YATES DW, HADFIELD JM, LITTLERA. The relevance of the biomechanics of impact to the assessment of head injured patients in the accident and emergency department. International IRCOBI 1981;1:217-222.

383.YATES D. PETERS K. A comprehensive view of every head injured patient, seen in two accidents and emergency departments over a 12 months period. Arch. Emerg. Med. 1986;3 41-46.

384.YAKOLEV PJ, LECOURS AP. The myelinogenc cycle of regional maturation of the brain in early life. Oxford, Blackwell 1967;3-70.

385.ZUCHARELLO M, FIORE D, TRINCI A , ANDRIOLI G. Extradural haematoma: statistical analysis of 413 cases. Advances in neurotraumatology. Amsterdam Oxford, 1983;238-241.

386.ZIMMERMAN RA, BILANIUK LT. Computerised tomography in paediatric head trauma. II. Neuroradiol 1981;8:257-271.

387.ZIMMERMAN RA, BILANIUK LT. Paediatric head trauma. Neuroimaging. Clinics of North Am. 1994;4:2:349-360.

388.ZIMMERMAN RA, BILANIUK LT, BRUCE DA et al. Interhemispheric acute subdural haematoma. A computed tomographic manifestation of child abuse by shaking. Neuroadiology 1978;16:39-40.

389.ZIMMERMAN RA, BILANIUK LT, BRUCE DA et al. Computed tomography of craniocerebral injury in the abused child. Radiology 1979;130:687-6

PUBLISHED PAPERS

Journal of Epidemiology and Community Health 1990; 44: 147–151

Head injuries in accident and emergency departments. How different are children from adults?

Marie Brookes, Robert MacMillan, Stewart Cully, Elaine Anderson, Stan Murray, A David Mendelow, Bryan Jennett

Abstract

***Study objective*—The aim of the study was to examine the differences between child and adult patients attending accident and emergency departments after recent head injuries.**

***Design and setting*—A retrospective survey based on existing case records from 23 Scottish accident and emergency departments for 1985 was compared with prospective data from one hospital over 9 months in 1984.**

***Patients*—3838 children under 15 and 4775 adults attended hospital with head injuries during the period analysed.**

***Main results*—Only 9% of children and 20% of adults had evidence of brain damage (altered consciousness on arrival, or history of altered consciousness with amnesia on arrival). Scalp lacerations were recorded in ~ 40% of both children and adults, more commonly in those without brain damage. Fewer children than adults had a skull x ray; in both age groups x rays were more often done if there was evidence of brain damage, headache, or vomiting, and less often when there was a scalp laceration. Only 11% of children were admitted compared with 20% of adults. Admission rates per 100 000 population per year were 4011 for children and 1473 for adults (1967 overall); admission rates for brain damage were 290 for children and 341 for adults (331 overall).**

***Conclusions*—These are the first population based estimates of frequency of head injuries presenting at accident and emergency departments, analysed by age, gender and cause of injury. They should be of value when planning services for the head injured.**

No routine statistics by diagnosis are published about attenders at accident and emergency departments, to compare with the data on inpatients that are produced annually for England and Wales and for Scotland. Our survey of Scottish hospitals in 1974 disclosed that four to five times as many head injured patients attended as were admitted,[1] leading us to estimate that about a million new cases of head injury attend hospitals in Britain each year.[2] Children (< 15 years) accounted for more than 40% of over 3000 head injury attenders in the Scottish survey,[1] and also in a subsequent review of ~ 12 000 cases at one English district hospital.[3] The UK guidelines for the initial management of head injuries published in 1984 apply only to adults,[4] recognition that the clinical course and risks of complications are expected to be different in children. No report has, however, analysed the differences between head injured children and adults. This paper compares adults and children in more than 3000 attenders collected prospectively in one Scottish district general hospital in 1984; and in more than 5000 from a retrospective survey of 23 Scottish accident and emergency departments in 1985. The 1974 Scottish data were reanalysed to identify children and adults for demographic comparison with the present study, and to discover whether the rates of skull x ray and of admission had changed in the last decade.

Department of Accident and Emergency Medicine, Monklands District General Hospital, Airdrie ML6 0JS, United Kingdom
M Brookes
Department of Neurosurgery, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF, United Kingdom
R MacMillan
S Cully
E Anderson
S Murray
D Mendelow
B Jennett

Correspondence to: Professor Jennett

Accepted for publication September 1989

Methods

SURVEY OF SCOTTISH HOSPITALS

The 1974 survey involved all 40 Scottish hospitals which had accident departments. Data were abstracted from existing records as previously described,[1 2] for one winter and one summer week chosen at random. One important aim of the 1985 survey was to discover whether the admission rates for attenders had changed differently in the four groups of hospitals previously studied (adult teaching hospitals, children's hospitals, and other hospitals within and beyond 30 miles of the neurosurgical unit). Another aim was to discover whether practice in the West of Scotland (where the Guidelines originated) and in the rest of Scotland was different. Hospitals with very small numbers of head injuries were excluded, and to select the remaining hospitals we used stratified sampling to ensure a balance between different groups and different parts of Scotland. The target numbers of patients required to detect a change in admission rate for each hospital group were calculated, and converted to weeks of sampling needed to yield these numbers, as estimated from the two week samples in 1974. The period of sampling in the 23 hospitals ranged from 2 to 6 weeks, and the number of patients from each hospital from 24 to 607 (table I). The date of the first week of sampling was chosen for each hospital using random number tables.

SURVEY OF MONKLANDS HOSPITAL

This district general hospital serves a predominantly urban population of 171 000 in Lanarkshire. Data were collected prospectively over a nine month period in 1984 by a succession of 16 senior house officers, all working under the supervision of the accident and emergency consultant (MB), who has a special interest in head injuries. A computerised system for collection of data on all types of case was already in

510

Table I Sampling for 23 Scottish hospitals in 1985

		Weeks sampled	n
1	City Teaching Hospitals:		
	Royal Infirmary, Glasgow	4	607
	Western Infirmary, Glasgow	4	254
	Stobhill General Hospital, Glasgow	4	231
	Royal Infirmary, Edinburgh	4	528
	Royal Infirmary, Dundee	4	320
	Royal Infirmary, Aberdeen	4	395
2	Children's Hospitals:		
	Royal Hospital for Sick Children, Glasgow	2	110
	Royal Hospital for Sick Children, Edinburgh	2	127
	Royal Aberdeen Children's Hospital	2	158
	Seafield Children's Hospital, Ayr	2	24
3a	Others—within 30 miles of neurosurgical unit:		
	Bangour General Hospital, Broxburn	6	253
	Crosshouse Hospital, Kilmarnock	6	294
	Dunfermline and West Fife Hospital, Dunfermline	6	223
	Falkirk and District Royal Infirmary, Falkirk	6	247
	Inverclyde Royal Hospital, Greenock	6	221
	Monklands District General Hospital, Airdrie	6	481
	Roodlands General Hospital, Haddington	6	71
	Royal Alexandra Infirmary, Paisley	6	318
	Victoria Hospital, Kirkcaldy	6	259
3b	Others—more than 30 miles from neurosurgical unit:		
	Ayr County Hospital, Ayr	3	90
	Belford Hospital, Fort William	3	29
	Dumfries and Galloway Royal Infirmary	3	53
	Raigmore Hospital, Inverness	3	55

use in the department, but additional items specific to head injury were added for the purposes of this study. It was considered that the data from this hospital would be of high quality, but might not be representative of Scottish hospitals in general. Comparing the two populations was considered useful as a check on the validity of each data set.

DEFINITION OF A HEAD INJURY

This was any injury to the scalp, including swelling, abrasion or contusion as well as laceration; or a well authenticated history of a blow to the head; or any patient in whom a skull x ray was performed immediately following trauma, and patients who had clinical evidence of fracture at the base of the skull. Patients with abrasions, lacerations or fractures limited to the face or facial skeleton were excluded, as were those with foreign bodies in the nose, ears or eyes. Birth injuries were excluded.

Results

DEMOGRAPHY AND CAUSES

Age and sex distribution was similar in the three surveys (table II), with children accounting for 40–51% of head injured attenders. Male predominance was slightly less for children as a whole than for adults, but of the youngest children only 58–63% were boys; 77–82% of 12–14 year olds were male, a greater proportion than in adults.

The causes of injury differed significantly in adults and children (table III). A fall accounted for more than half the injuries in children but for only a third in adults; assaults were more common in adults. Road accidents were the cause of only a minority of these predominantly mild injuries, both in children and adults. Children who had road accidents were more often pedestrians or pedal cyclists; only 20% were vehicle occupants, compared with 58% of adults. Of accidents that were not on the road, half of those in children and almost a third in adults occurred within the home;

Table III Commonest causes for head injured attenders (Scotland 1985)

	Children	Adults
For all cases:		
n	2118	3124
Fall	57%	33%
Assaults	6%	29%
RTA	9%	16%
Type of RTA:		
n	183	470
Occupant	20%	58%
Pedestrian	42%	23%
Pedal cycle	32%	9%
Place of non-RTA[1]:		
n	1162	1477
Home	52%	29%
Street	13%	29%
School/work	13%	17%

RTA = road traffic accident
[1] Place recorded in only 58% of non-RTA cases
Differences in distribution between adults and children significant for each table ($p < 0.001$)

less than one in six occurred at work or school. Even fewer were associated with sport, in an organised sense. If recreational activities in general are included, the proportion was greater, because a number of falls occur during such activities.

EVIDENCE OF BRAIN DAMAGE (table IV)

In the Scotland wide survey, which was based on retrospective inspection of routine records, only one per cent of children compared with five percent of adults had altered consciousness by the time they arrived at accident and emergency departments after recent head injury. In the prospective Monklands study the Glasgow Coma Scale was recorded on arrival, and in that hospital impaired consciousness was recorded as often in children as in adults. Some of these children were probably only temporarily drowsy or asleep, because the admission rate was no greater in Monklands than in the Scottish survey.

Some patients who were judged fully conscious on arrival had evidence of having had impairment of consciousness immediately after the injury.

Table II Age/Sex distribution in three surveys of A/E attenders

	Scotland 1974			Scotland 1985			Monklands Hospital 1984		
	n	% in age group	% male	n	% in age group	% male	n	% in age group	% male
Child	1515	43	67	2118	40	68	1720	51	65
Adult	2041	57	72	3157	60	71	1651	49	72
All	3556		70	5275		70	3371		67
< 5 yr	576	38	58	953	45	63	799	46	58
5–11 yr	742	49	70	868	41	72	698	41	70
12–14 yr	197	13	82	297	14	77	223	13	78

511

Table IV Frequency of altered consciousness in attenders.

	Scotland 1985		*Monklands 1984*	
	Children (n = 2118)	Adults (n = 3124)	Children (n = 1720)	Adults (n = 1651)
No evidence of brain damage	93%	77%	89%	85%
History of altered consciousness now recovered	6%	18%	4%	11%
Still altered consciousness at A/E dept	1%	5%	7%	6%
Difference in distribution between children and adults	$p < 0.007$		$p < 0.007$	

A/E = accident and emergency

Either witnesses had reported this, or the patients were amnesic for the accident and a brief period after it. More adults than children were in this category, perhaps because it is easier to elicit an account of amnesia from adults. Other evidence of brain damage, such as a fit or focal neurological signs, rarely occurred in patients who did not also have evidence of altered consciousness.

OTHER FEATURES

Various reasons accounted for the attendance of a large number of patients who had no evidence of brain damage, and for regarding them as head injured. A scalp laceration was a feature of about half of both children and adults in the Scottish survey, and of more than a third in Monklands (table V). A laceration was less frequent in patients who had evidence of brain damage, in whom it was twice as common in adults as in children; as many children as adults without brain damage had a scalp laceration. Most other patients without brain damage either had a history of a fall or a blow to the head followed by headaches and/or vomiting; or they had recently ingested alcohol, which made the history uncertain or the state of consciousness difficult to assess. Recent alcohol intake was noted in a quarter of adult attenders, but in a third of those who had fallen, and in 38% of those who had been assaulted; only 11% of road accident victims had evidence of recent alcohol intake. Headache was more commonly complained of by adults (14–20% *v* 8–10%), and vomiting more often observed in children (10% *v* 4–6%). However, only 15–22% of both children and adults complained of either of these features.

POPULATION BASED RATES FOR ATTENDANCE AFTER HEAD INJURY

Data are available for all new attenders at the hospitals in each of the 15 health board areas in Scotland, and the population served by each area can be derived from the Registrar General's figures. The survey of head injuries indicated the proportion of new attenders that were head injuries in each area. By extrapolation, the rates for Scotland as a whole were calculated. A similar method had been used to calculate rates for 1974 for limited subsets, and where comparison is possible the rates are similar (table VI). More detailed analysis was possible for 1985, in particular for adults and children of each sex, and by the three main causes (table VIIa, fig 1), and separately for those who had evidence of brain damage (table VIIb, fig 2).

For all causes together, the rates for children of both sexes were more than twice those for adults; for falls there was a fourfold difference in favour of children, whilst for road accidents there was little difference between children and adults. The attendance rates for those with evidence of brain damage were not only much lower, but the differences in rates between children and adults were no longer striking, except for assaults (table VIIb, fig 2). For all causes combined, the incidence of attenders with brain damage both for adults as a whole and for adult males was higher than it was for children.

Table VI New attenders at Scottish hospitals with head injuries—comparison of rates per 100 000 population in 1974 and 1985

	1974 (Ref 7)	1985
All	1778	1967
Males	2591	2832
Females	1024	1158
Children	3017	4011
Road	314	364
Assault	244	230

SKULL X RAY AND ADMISSION TO HOSPITAL

X rays were less often done in children (58%) than in adults (70%) in the Scottish survey; but in Monklands 80% of both age groups had a skull x ray (table VIII). The lowest x ray rates were in patients with scalp lacerations, especially children. Patients with no evidence of brain damage less often had x rays in Scotland as a whole, but in Monklands this distinction was not seen. X ray was more often done when there had been headache and/or vomiting. A fracture was found in 2–3% of both children and adults who had x rays.

Admission to hospital was twice as common in adults (18–23%) as in children (10–11%). Of patients without any evidence of brain damage, only seven percent of children and 12% of adults were admitted, compared with 61% and 58% respectively of those with brain damage. However, of those with consciousness still impaired on arrival at hospital, 25% of children and 16% of adults were sent home. Since the survey of 1974 and the publication of guidelines for the management of adults, the proportion of both children and adults who have x ray

Table V Frequency of scalp laceration in attenders.

	Scotland 1985			Monklands 1984		
	Children (n = 2118)	Adults (n = 3124)	p	Children (n = 1720)	Adults (n = 1651)	p
In all patients	48%	50%	NS	36%	38%	NS
In patients without brain damage	48%	52%	< 0.001	38%	39%	NS
In patients with brain damage	14%	32%	< 0.001	15%	33%	< 0.001

Statistical comparison is between children and adults

512

Table VII Attendance rates at accident and emergency, per 100 000 population per year, by cause

	Scotland 1985		
	Children	Adults	All
(a) All attenders after head injury			
All causes			
All	4011	1473	1967
Males	5340	2180	2832
Females	2613	831	1158
Falls			
All	2280	459	813
Males	2924	544	1035
Females	1603	381	605
Assaults			
All	230	399	366
Males	350	677	610
Females	99	147	138
RTA			
All	364	222	249
Males	486	306	343
Females	235	144	161
(b) Attenders with evidence of brain damage[1]			
All causes			
All	290	341	331
Males	395	537	508
Females	180	163	166
Falls			
All	130	118	120
Males	165	168	167
Females	94	73	77
Assaults			
All	6	147	78
Males	8	171	137
Females	4	26	22
RTA			
All	99	70	76
Males	141	77	112
Females	55	39	42

[1] Brain damage = any evidence of altered consciousness either before or after reaching hospital, or neurological signs

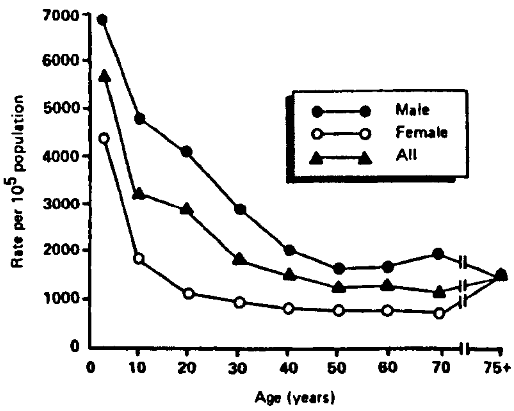


Figure 1 Annual incidence of attendance—all head injuries. (Scottish A/E Departments, 1985)

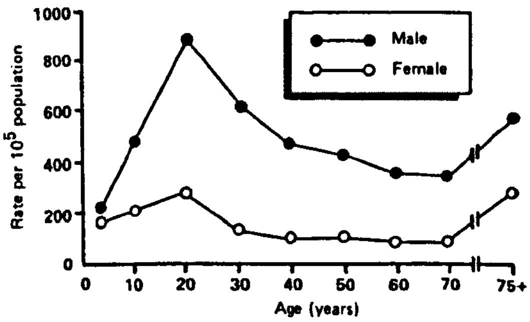


Figure 2 Annual incidence of attendance with evidence of brain damage. (Scottish A/E Departments, 1985)

examinations has increased; but there has been a reduction in the admission rate, which is more significant for children.

Discussion

While data are routinely collected on deaths and on hospital discharges (or admissions), no country appears to keep diagnostic records on attenders at emergency departments who are sent home. Almost all the epidemiological studies of head injuries published during the last decade in the USA and Australia have been limited to patients admitted to hospital, some of them including also deaths at the scene. Some surveys have been limited to children, others to adults; those that include both have only indicated the age distribution, without separately analysing the features of the two age groups.

The two previous Scottish studies showed that there were striking differences in the ages and the distribution of causes between head injuries of varying severity.[5] Children made up more than 40% of those in accident/emergency departments, a third of those in wards of first admission, a fifth of severe injuries, but only about a tenth of fatal injuries. By contrast, road accidents were progressively more common as the severity of injury increased. The present study shows that, in the mainly mild injuries that predominate in accident departments, the causes differ strikingly between children and adults. Road accidents were an unusual cause in children, and in them most were pedestrians and pedal cyclists. In this study all injuries to cyclists were assigned as road accidents, but there is good evidence from several countries that most children injured when riding a bike have fallen when off the road.[6 8]

As evidence of brain damage we chose simple criteria that could be applied in emergency departments. By these measures only a small minority of attenders had suffered any brain damage, and even fewer children than adults. It is, however, difficult to deny that these patients have suffered a head injury, because many of them had a scalp laceration and some of them a fractured skull. Some American epidemiologists[9 10] have excluded such attenders from their calculations of incidence. They also discounted patients who were admitted but who did not have loss of consciousness or post-traumatic amnesia of a certain duration; this meant excluding some who had a skull fracture. The subset of head injuries to which their incidence rates apply is often defined as "brain injuries". It was to allow comparison

Table VIII Frequency of skull x ray in patients with different features

	Scotland 1985		Monklands 1984	
	Children (n=2118)	Adults (n=3124)	Children (n=1720)	Adults (n=1651)
In all cases	58%	70%† < 0·001	80%	81%
With scalp laceration	40%	60%	64%	73%
No laceration	73%‡	79%‡	89%‡	86%‡
No evidence of brain damage	55%	64%	80%	80%
With brain damage	88%§	90%§	83%NS[1]	86%§
Headache or vomiting	83%	88%	89%	88%
Neither	53%¶	67%¶	79%¶	79%¶

‡ p<0·001 v laceration; § p<0·001 v no evidence brain damage; ¶ p<0·001 v headache/vomiting; † p<0·001 adults v children
[1] Small number of children with brain damage

513

between the Scottish incidence rates for attenders and those for admissions in parts of the USA that we calculated rates separately for all attenders, and for those with evidence of brain damage (by our definition). These are the first population based estimates of the frequency of head injuries presenting at accident departments, analysed by age, gender and cause of injury. They could be useful when planning services for the head injured. Indeed, it is the large number of mildly injured patients who arrive at accident/emergency departments, whether or not they have evidence of brain damage by some arbitrary definition, that constitute the major challenge not only to health care systems, but also to clinicians who have to decide which to investigate, to admit for observation or to send home.

There remains some controversy about the indications for skull x ray, CT scan and admission for observation after mild head injury. We shall deal with these issues in subsequent reports that will analyse patients who were admitted, and relate these issues to the risk factors that have been calculated by us for the development of acute intracranial haematoma[11]. Here we record simply the frequency with which skull x rays were done and the proportion of patients who were admitted, noting the differences in practice for children and adults. This is the first step towards the evolution of guidelines for the management of children, to set beside those already developed for adults.

This Scottish survey, and the analysis of data from both sources, was supported by a project grant from the Health Services Research Committee of the Chief Scientist's Office, Scottish Home and Health Department. Data collection at Monklands Hospital was made possible by a grant from the General Manager of the Lanarkshire Area Health Board (Mr Frank Clarke).

1 Strang I, MacMillan R, Jennett B. Head injuries in accident and emergency departments at Scottish hospitals. *Injury* 1978; 10: 154–9.
2 Jennett B, Murray A, MacMillan R, *et al.* Head injuries in Scottish hospitals. *Lancet* 1977; ii: 696–8.
3 Gorman DF. Were you knocked out? Yes, but I wasn't admitted. *Arch Emerg Med* 1985; 2: 127–9.
4 Group of Neurosurgeons. Guidelines for initial management after head injury in adults. *Br Med J* 1984; 288: 983–5.
5 Annegers JF, Grabow JD, Kurland LT, *et al.* The incidence, causes and secular trends of head trauma in Olmsted County, Minnesota. *Neurology* 1980; 30: 912–9.
6 Kraus JF, Black MA, Hessol N, *et al.* The incidence of acute brain injury and serious impairment in a defined population. *Am J Epidemiol* 1984; 119: 186–201.
7 Jennett B, Macmillan R. Epidemiology of head injury. *Br Med J* 1981; 282: 101–4.
8 Friede AM, Azzara CV, Gallagher SS, *et al.* The epidemiology of injuries to bicycle riders. *Pediatr Clin N Am* 1985; 32: 141–51.
9 Craft AW, Shaw DA, Cartlidge NEF. Bicycle injuries in children. *Br Med J* 1973; IV: 146–7.
10 Kraus JD, Fife D, Conroy C. Incidence, severity and outcomes of brain injuries involving bicycles. *Am J Public Health* 1987; 77: 76–7.
11 Teasdale GM, Murray G, Anderson E, *et al.* Risks of acute traumatic intracranial haematoma in children and adults: implications for managing head injuries. *Br Med J* 1990; 300: 363–7.