The effect of intensive care nursing interventions on the intracranial pressure in children with moderate to severe traumatic brain injury

LYVONNE NICOLE TUME

A thesis submitted in partial fulfilment of the requirements of Liverpool John Moores University for the degree of Doctor of Philosophy

October 2009

THE FOLLOWING MATERIAL HAS NOT BEEN DIGITISED AT THE REQUEST OF THE AWARDING UNIVERSITY;

TABLE 1 PAGE 12

APPENDIX 2

THE ADRESS INFORMATION IN APPENDECIES 4 – 5

APPENDECIES 12 – 16

Acknowledgements

I would like to thank my three fantastic supervisors over the four year period. Dr Paul Baines has supported me throughout this time and despite his considerable clinical and academic commitments has always given me his time and provided me with constructive feedback. Professor Annette Jinks was with me at the start and was hugely supportive and despite her departure to another educational institution continued to offer support, assistance and feedback, even when no further formal supervisory arrangements existed. Professor Paulo Lisboa took me on after this and despite not having a clinical background his extensive supervisory experience and skills in mathematics and statistical analysis (far superior to any clinician I could have had) really made this last part of my PhD period (analysis and writing up) much more manageable for me. His extensive support and feedback was invaluable to my completion of this thesis.

I also thank all the parents of the children in this study, who, even at a very stressful time, agreed for their child to partake in this study.

I also want to thank my line manager at Alder Hey, Kathy Corrin, for trusting me and allowing me the time to be able to spend on the PhD over the last two years in particular.

Last but not least, I need to thank my partner Hugo for putting up with me over the last 4 years and continually solving all my IT problems.

ABSTRACT

Objective

The aim of this study was to examine the effects of selected routine nursing interventions - endotracheal suctioning and manual ventilation (ETSMV), log-rolling, eye care, mouth care and washing - on the intracranial pressure (ICP) in children with traumatic brain injury.

Design

Prospective observational study over three years.

Setting

Single tertiary paediatric intensive care unit in the North West of England.

Patients

Twenty five children with moderate to severe closed traumatic brain injury and intraparenchymal intracranial pressure monitoring in intensive care (2 -17 years of age).

Interventions

Routine nursing care interventions.

Measurements and main results

ICP measured one minute before the procedure, at the maximal value during the procedure and five minutes after the procedure was recorded for the purpose of this study. Time to recovery was also recorded, in minutes. A total of 25 measurements (the first one in each child) in the first 36 hours of the child's PICU admission were analysed. Both ETSMV and log-rolling

were associated with clinically and statistically significant changes in ICP from baseline to maximal ICP ($p=0.005$) and maximal to 5-minute post ICP ($p=0.001$) for ETSMV and ($p<0.001$) baseline to maximal ICP and ($p=0.002$) for maximal to post-procedure ICP for log-rolling. During ETSMV and log-rolling 70% of children exceeded the 20mmHg clinical treatment threshold during the interventions. During both ETSMV and log-rolling children with higher baseline ICPs (>15mmHg) showed higher maximal ICPs (but not ICP rise), suggesting a linear relationship between baseline and maximal ICP, although this was more pronounced during turning. One third of the children had not returned to their baseline ICP by 5 minutes after ETSMV, compared with 60% children after log-rolling.

Neither eye care nor mouth care showed any clinically significant effects on ICP in these children, suggesting these procedures are not noxious and are tolerated very well. However, there was only a small number of washing episodes reported in this study therefore the observations are not conclusive.

Conclusions

Endotracheal suctioning and log-rolling in moderate to severe traumatic brain injured children can cause significant intracranial instability and should only be performed as required and with careful planning and execution. Eye and mouth care and washing appear to be well tolerated interventions and could be performed when necessary.

Contents

Chapters		Page Numbers
1.	**Introduction and overview of thesis**	
1.1	Introduction and overview of the thesis	1
2.	**Epidemiology, pathophysiology and management of paediatric traumatic brain injury in intensive care**	
2.1	Epidemiology of paediatric traumatic brain injury	5
2.2	Review of normal intracranial physiology and the pathophysiology of paediatric traumatic brain injury	6
2.3	Brain injury severity and the need for intensive care	10
2.4	Intensive care management goals	11
2.5	The development of international management guidelines	12
2.6	The treatment of intracranial hypertension	13
2.7	First tier therapies for intracranial hypertension	16
2.8	Hyperosmolar therapy and diuretics	16
2.9	CSF drainage	18
2.10	Neuromuscular blockade	19
2.11	Mild hyperventilation	20
2.12	Second tier therapies for intracranial hypertension	21
2.13	Moderate hypothermia	21
2.14	Barbiturate therapy	22
2.15	Decompressive craniectomy	23
2.16	Intracranial pressure monitoring	26
2.17	Intensive care therapies that have an affect on ICP or CPP	28
2.18	Patient outcomes after severe TBI in children	35
2.19	Assessing neuropsychological logical outcomes	36
3.	**Review of the Literature**	
3.1	Introduction to the literature review	39
3.2	Search Strategy and results	39
3.3	Classification of quality of published evidence	42
3.4	Critical review of the literature	43
3.5	The effect of repositioning, moving or turning on ICP and cerebrovascular parameters	44
3.6	The effect of clustering nursing cares together and planned rest periods on CBV parameters	47
3.7	The effects of hygiene interventions on CBV parameters	48
3.8	The effect of endotracheal suctioning on ICP	49
3.9	The effect of coughing during ETS on the ICP response	52
3.10	The effect of ETS on cerebral perfusion pressure	53
3.11	The effect of ETS on cerebral oxygenation	55
3.12	The effect of respiratory physiotherapy on CBV parameters	55

3.13 The effect of hyperventilation during ETS 56
3.14 Recovery times after suctioning 59
3.15 The effect of pharmacological agents during ETS 60
3.16 Summary of papers excluded from the literature review 68
3.17 Summary of the literature review 69

4. Audit of current UK Practice
4.1 Introduction 72
4.2 Methods 72
4.3 Results - nursing and physiotherapy management 73
4.4 Discussion 76
4.5 Limitations of the audit 77
4.6 Conclusions 77

5. Methods
5.1 Introduction 80
5.2 Aims, objectives and research questions 80
5.3 Setting 81
5.4 Study design 82
5.5 Ethical approval 82
5.6 Description and definition of nursing interventions studied 83
5.7 Endotracheal suctioning and manual ventilation 84
5.8 The suctioning procedure 84
5.9 The turning procedure 86
5.10 The eye care procedure 87
5.11 The mouth care procedure 87
5.12 The washing procedure 88
5.13 Time frame for measurements 88
5.14 Recruitment of patients 89
5.15 The main study sample – inclusion and exclusion criteria 90
5.16 The decompressive craniectomy sample 91
5.17 Defining severe head injury in this study 92
5.18 Data collection 92
5.19 Phase I – the pilot study 95
5.20 Refining the methodology 102
5.21 Reliability and validity 104
5.22 Validity of the data collection tool 113
5.23 Potential confounders 114
5.24 Data analysis 116
5.25 Stratification of patients 119
5.26 Statistical analysis 120
5.27 Discussion of ethical issues 124
5.28 Conclusion 126

6. Results – Endotracheal Suctioning and manual ventilation

6.1 Introduction 128
6.2 Demographics of the suctioning group 128
6.3 Distribution of the data in the suctioning sample 130
6.4 The effects of endotracheal suctioning on the ICP response 133
6.5 Stratification by baseline ICP 136
6.6 The correlation between variables during endotracheal suctioning 139
6.7 The effect of confounders during endotracheal suctioning 142
6.8 The effect of a sedation bolus before endotracheal suctioning on the ICP response 144
6.9 The effect of ET Lignocaine before endotracheal suctioning on the ICP response 146
6.10 The effect of barbiturates on the ICP response to endotracheal suctioning 148
6.11 The effect of neuromuscular blockade on the ICP response to endotracheal suctioning 150
6.12 The effect of painful injuries on the ICP response 153
6.13 Recovery times after endotracheal suctioning 156
6.14 Changes in mean arterial pressure during suctioning 160
6.15 Changes in MAP, CPP and ICP during suctioning 162
6.16 Conclusion and summary of results for endotracheal suctioning 165

7. Results – Turning the child (via a log-rolling approach)

7.1 Introduction 167
7.2 Demographics of the turning sample 168
7.3 Distribution of the turning sample data 170
7.4 The effect of turning on the ICP response 172
7.5 Stratification by baseline ICP 176
7.6 The correlation between variables during turning 179
7.7 The effect of confounders during turning 185
7.8 The effect of sedation bolus before turning 187
7.9 The effect of neuromuscular blockade 190
7.10 The effect of barbiturates 192
7.11 The effect of having other (painful) injuries 194
7.12 Recovery times after turning 197
7.13 Changes in MAP and CPP during turning 201
7.14 Interaction between ICP, CPP and MAP during turning 204
7.15 Comparison of suctioning and turning as noxious stimuli 205
7.16 Conclusion and summary of turning (via log-rolling) 207

8. Results – Other nursing interventions

8.1 Introduction 211
8.2 The effect of eye care 212
8.3 The effect of mouth care 214

8.4 The effect of washing 217
8.5 Conclusion and summary of results of other interventions 221

9. Results – Nursing interventions in children after decompressive craniectomy (DC)
9.1 Introduction 224
9.2 The decompressive craniectomy (DC) sample 225
9.3 The effects of ETSMV after DC 226
9.4 The effects of turning after DC 228
9.5 The effects of eye care, mouth care and washing after DC 230
9.6 Conclusion and summary of results of interventions after decompressive craniectomy 230

10. Discussions
10.1 Introduction 233
10.2 Observational studies and the use of scientific research methods in nursing 233
10.3 Aims, objectives and research questions of the study 235
10.4 The study sample 239
10.5 Severity of illness of the study sample 240
10.6 Nursing interventions in these children 241
10.7 The effects of endotracheal suctioning 242
10.8 Does baseline ICP make any difference in suctioning? 244
10.9 The effect of coughing during suctioning 245
10.10 The effect of a sedation bolus before suctioning 246
10.11 The effect of muscle-relaxation during suctioning 248
10.12 The effect of barbiturates during endotracheal suctioning 249
10.13 The effect of endotracheal lignocaine before suctioning 250
10.14 Recovery times after suctioning 251
10.15 The effects of turning (via log-rolling) on the ICP response 252
10.16 The effect of a sedation bolus and muscle relaxants during turning 255
10.17 The effect of barbiturates during turning 256
10.18 The effect of having other (painful) injuries during turning 257
10.19 Recovery times after turning 258
10.20 Comparison of suctioning and turning as a noxious stimuli in children with severe traumatic brain injury 259
10.21 The effect of other nursing interventions 260
10.22 The effect of nursing interventions in children after decompressive craniectomy 261
10.23 Summary of key findings of the thesis 262
10.24 Limitations of the study 262
10.25 Implications and recommendations for practice 265
10.26 Implications for future research 268
10.27 Implications for intensive care nurses 270

11. Conclusion 274

References 276

Appendices 300

Publications (Appendix 12 – 16)

Table of Abbreviations

AHCH	Alder Hey Children's NHS Foundation Trust (Children's Hospital)
ALSG	Advanced Life Support Group
BIS	Bispectral Index
CBV	Cerebrovascular
CFAM	Cerebral Function Monitor
COMFORT	(Score) a validated sedation score in ventilated term infants and children
CPP	Cerebral perfusion pressure (calculated as MAP-ICP)
CSF	Cerebrospinal Fluid
CT scan	Computerised Tomography scan
DAI	Diffuse Axonal Injury
DC	Decompressive Craniectomy
DVT	Deep Vein Thrombosis
EBM	Evidence Based Medicine
ED_{50}	The amount of drug that produces a therapeutic response in 50% of the people taking it
EEG	Electroencephalogram
EtCo2	End tidal carbon dioxide
ET	Endotracheal
ETS	Endotracheal suctioning
ETT	Endotracheal tube
ETSMV	Endotracheal suctioning and manual ventilation

EVD	External Ventricular Drain
GCS	Glasgow Coma Score
GOS	Glasgow Outcome Score
HOB	Head of the bed
HTS	Hypertonic saline
ICP	Intracranial pressure
ICU	Intensive Care Unit
IV	Intravenous
LR	Log-rolling
MAP	Mean arterial blood pressure
MV	Manual ventilation
NHS	National Health Service
NICE	National Institute of Clinical Excellence
NMB	Neuromuscular blockade
PEEP	Positive End Expiratory Pressure (in cm H20)
PICANET	The Paediatric Intensive Care Audit Network
PICS	The Paediatric Intensive Care Society
PICS-E	The Paediatric Intensive Care Society Education Group
PICU	Paediatric Intensive Care Unit
PIM	Paediatric Index of Mortality (Score)
PVS	Persistent Vegetative State
QUOROM	Quality of Reporting of Meta-analyses standards

RESCUEicp	Trial of Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of ICP
RHISS	Relative Head Injury Severity Score
RLCH	Royal Liverpool Children's Hospital
ROM	Range of Motion (exercises)
RP	Respiratory Physiotherapy
RTA	Road Traffic Accident
SjO2	Jugular venous oxygen saturation
SMR	Standardised mortality rate
SpO2	Oxygen saturation
SPSS	Statistical Package for the Social Sciences
STROBE	Strengthening the Reporting of Observational studies in Epidemiology
TBI	Traumatic Brain Injury
TED	Thromboembolic Deterrent (Stockings)
WFPIC	World Federation of Pediatric Intensive Care
WTE	Whole time equivalent
vMCA	Velocity of blood flow in the middle cerebral artery

List of Tables

Table No.	Title	Page No.
1	Age-dependent ICP and CPP values	12
2	Demographics of the pilot study sample	96
3	Measurements in the pilot study	96
4	Demographics of the ETSMV sample	129
5	Mean baseline data prior to ETSMV	130
6	Distribution of the ETSMV ICP outcome measures	131
7	Distribution of the ETSMV ICP outcome measures after outlier removed	132
8	Correlation table for ETSMV	140
9	Presence of confounders during ETSMV	143
10	Distribution of ETSMV MAP outcome measure	162
11	Demographics of log-rolling (LR) sample	169
12	Mean baseline data prior to log-rolling	170
13	Distribution of LR ICP outcome measures	171
14	Distribution of LR ICP outcome measures with outlier removed	172
15	Correlation table for log-rolling	180
16	Presence of confounders during log-rolling	187
17	Significance of confounders during log-rolling	197
18	Distribution of LR MAP data as an outcome measure	202
19	Distribution of LR MAP data with the outlier removed	203

Table No.	Title	Page No.
20	Measurement episodes in other care interventions	212
21	Distribution of eye care ICP outcome measures	212
22	Distribution of mouth care outcome measures	214
23	Distribution of mouth care outcome measures with outlier removed	215
24	Distribution of washing outcome measures	218
25	Distribution of washing outcome measures with outlier removed	219
26	Demographics of decompressive craniectomy children	225
27	Baseline values of decompressive craniectomy group before ETSMV	226
28	Baseline values of decompressive craniectomy group before log-rolling	228

List of diagrams and pictures

Diagram No.	Title	Page no.
1	The pressure-volume curve	7
2	Herniation of the brain	8
3	Cycle of increasing intracranial pressure	9
4	AHCH Flowchart for the management of raised ICP	15
5	CT scan of a child before and after decompressive craniectomy	24
6.	ICP monitoring locations	26
7.	Selection of papers in the literature review	41

List of graphs

Graph No.	Title	Page No
1	Comparison of the effects of nursing cares in pilot study	97
2	Effect of ETSMV in pilot study	98
3	Recovery time after ETSMV in pilot study	99
4	Effect of log-rolling in pilot study	100
5	Recovery time after log-rolling in pilot study	100
6	Effect of eye care in pilot study	101
7	Effect of mouth care in pilot study	102
8	Bland Altman plot of inter-observer reliability of baseline ICP	107
9	Bland Altman plot of inter-observer reliability of maximal ICP	108
10	Bland Altman plot of inter-observer reliability of post ICP	109
11	Bland Altman plot of inter-observer reliability of baseline MAP	110
12	Bland Altman plot of inter-observer reliability of maximal MAP	111
13	Effect of ETSMV on ICP response (whole sample)	132
14	Effect of ETSMV on ICP response (with outlier removed)	134
15	Maximal ICP during ETSMV	135
16	The percentage change in baseline ICP with ETSMV	136
17	ICP response with ETSMV by baseline ICP stratification	137
18	ICP rise during ETSMV by baseline stratification	138
19	Change in ICP from baseline and baseline ICP	139
20	Correlation between time taken for ETSMV and ICP rise	141
21	The effect of a sedation bolus on ICP during ETSMV	145

Graph No.	Title	Page No
22	The effect of a sedation bolus on ICP rise during ETSMV	145
23	The effect of ET Lignocaine on ICP during ETSMV	147
24	The effect of ET Lignocaine on ICP rise during ETSMV	147
25	The effect of thiopentone on ICP during ETSMV	149
26	The effect of thiopentone on ICP rise during ETSMV	149
27	The effect of NMB on ICP during ETSMV	152
28	The effect of NMB on ICP rise during ETSMV	152
29	The effect of painful injuries on ICP during ETSMV	154
30	The effect of painful injuries on ICP rise during ETSMV	155
31	Recovery times to baseline ICP after ETSMV	157
32	Recovery times to baseline ICP when stratified by baseline ICP	158
33	Relationship between post ICP, minutes to recovery and 6 month outcome after ETSMV	159
34	Recovery times after ETSMV in patients with and without thiopentone	160
35	The effect of ETSMV on mean arterial pressure (MAP)	163
36	The effect of ETSMV on MAP in children with and without nor-adrenaline (whole sample)	164
37	The effect of ETSMV on MAP in children with and without nor-adrenaline (with outlier removed)	165
38	The effect of log-rolling (LR) on the ICP (whole sample)	171

Graph No.	Title	Page No
39	The effect of log-rolling (LR) on the ICP (with the outlier removed)	173
40	The peak ICP recording during log-rolling	174
41	The percentage change from baseline ICP with LR	175
42	The effect on ICP during LR stratified by baseline ICP	177
43	The ICP rise during LR stratified by baseline ICP	178
44	Change in ICP from baseline compared to baseline ICP	179
45	Correlation between baseline ICP and maximal ICP during LR	181
46	Linear regression model between baseline and maximal ICP	181
47	Correlation of maximal ICP to post procedure ICP during LR	182
48	Correlation of ICP rise to post ICP during LR	183
49	Correlation of time taken for LR and minutes recovery	184
50	Correlation of maximal ICP and minutes to recovery	185
51	The effect of a sedation bolus on ICP response during LR	189
52	The effect of a sedation bolus on ICP rise during LR	189
53	The effect of NMB on the ICP response during LR	191
54	The effect of NMB on the ICP rise during LR	191
55	The effect of thiopentone on the ICP response during LR	193
56	The effect of thiopentone on the ICP rise during LR	194
57	The effect of other (painful) injuries on the ICP during LR	195
58	The effect of other (painful) injuries on the ICP rise during LR	196
59	Recovery time to baseline ICP after log-rolling	198

Graph No.	Title	Page No
60	Recovery time to baseline ICP by baseline ICP after LR	199
61	Relationship between post ICP, recovery times and 6 month outcome after log-rolling	200
62	The effect of log-rolling on MAP (whole sample)	203
63	The effect of log-rolling on MAP (with the outlier removed)	204
64	Comparison of suctioning and turning as noxious stimuli	206
65	Comparison of suctioning and turning on ICP rise	207
66	The effect of eye care on the ICP response	213
67	The percentage change from baseline ICP with eye care	213
68	The effect of mouth care on the ICP response (whole sample)	215
69	The ICP response with mouth care (with outlier removed)	216
70	The percentage change from baseline ICP with mouth care	217
71	The ICP response with washing the child (whole sample)	218
72	The ICP response with washing the child (with outlier removed)	220
73	The percentage change from baseline ICP during washing	221
74	The effect of ETSMV on ICP after decompressive craniectomy	226
75	Percentage change in ICP from baseline with ETSMV after decompressive craniectomy	227
76	The effect of log-rolling on ICP response after decompressive craniectomy	228

77 Change in ICP from baseline with log-rolling after decompressive craniectomy 229

List of Appendices

Appendix Number **Title**

1. Summary of papers in Literature review
2. Publication of national audit - British Journal of Intensive Care
3. Electronic Audit Questionnaire
4. Audit registration form (NHS trust)
5. Parent Information Sheet
6. Parent consent form
7. AHCH PICU Suctioning Guidelines in Head Injury
8. Data Collection form for study
9. AHCH Medical Management Guidelines for Severe Head Injury
10. Guidelines for completion of study data collection forms
11. Glasgow Outcome Score (GOS) Scoring definitions
12. Published paper 1: Tume L (2007) The nursing management of children with severe traumatic brain injury and raised ICP BJNN vol 3(10) 461-467.
13. Published paper 2: Tume, L and Jinks A (2008) The effects of endotracheal suctioning in severe traumatic brain injury in children: a review Nursing in Critical care vol 13 (5) 232-240.
14. Published paper 3: Tume L (2008) Impact of care interventions in children with severe traumatic brain injury in intensive care BJNN vol 4(3), 2-6.
15. Published paper 4: Tume L, Thorburn K and Sinha A (2008) A review of the intensive care management of severe traumatic brain injury in children BJNN vol 4 (9) 424 – 431.

16. Paper accepted for publication in Nursing in Critical Care (in press): Tume, Baines and Lisboa: The effect of nursing interventions on the intracranial pressure in paediatric traumatic brain injury.

CHAPTER 1

INTRODUCTION AND OVERVIEW OF THE THESIS

1.1 Introduction and Overview of the thesis

Traumatic Brain Injury (TBI) continues to be a leading cause of death and disability in children worldwide, and in a recent UK epidemiological study, TBI in children (leading to intensive care unit admission) occurred in 5.6 per 100 000 population (for 1 – 14 year olds) Parslow et al (2005). The primary aim of the intensive care management of severe TBI is the minimisation of secondary injury from cerebral oedema and worsening cerebral ischaemia (Reilly and Bullock 2005 p294; Chambers et al 2006). The intensive care nurse has a key role to play in a) recognising and minimising these secondary injury processes, which can significantly affect the child's outcome and b) in promoting intracranial pressure (ICP) and cerebral perfusion pressure (CPP) stability whenever possible. This often presents a dilemma for the nurse, who, just by performing essential nursing interventions or 'cares' for the child may in fact produce significant physiological instability.

A literature review revealed that the evidence remains inconclusive in relation to the effects of many essential nursing care interventions in head injured patients and especially in children as paediatric data are extremely limited. Some of this inconclusiveness in the literature may be due to the weaknesses in many of these studies in terms of: inconsistent ICP device measurement (which has been shown to affect the primary outcome measure ICP), mixed sample of intracranial pathology, mixed adults and

children in the analysis, very small sample sizes, weak research designs, lack of account or acknowledgement of known confounding variables impacting on the ICP or CPP or the low baseline ICP of the samples. It was apparent that there was a clear need to control for the above variables and study the effect of nursing interventions separately on the ICP and CPP specifically in children post traumatic brain injury.

This thesis has thus investigated the effect of selected 'routine' intensive care unit nursing interventions in children with moderate to severe traumatic brain injury, an area that the author has been interested in since the early days of her intensive care nursing career in adult neurotrauma patients over 18 years ago. It has generated new knowledge in terms of describing the effects of turning via a log-rolling approach and in terms of the effects of these interventions in children after decompressive craniectomy (no published papers on either of these) and has begun to quantify the magnitude of effect of all these interventions and their recovery times specifically in a population of children with moderate to severe traumatic brain injury.

Chapter two presents an overview of the pathology of traumatic brain injury in children and the intensive care unit interventions/therapies used to 'set the scene' for the thesis.

Chapter three presents a critical review of the literature on this topic and identifies a clear need for further research in children.

Chapter four presents the results of a UK wide survey undertaken to establish what current management practices actually were at the time this project was beginning (2006) to be able to put the results into the context of clinical practice.

Chapter five discusses the methodology for the study.

Chapter six presents the results of endotracheal suctioning and manual ventilation.

Chapter seven presents the results of turning (via a log-rolling approach).

Chapter eight presents the results of the other interventions of eye care, mouth care and washing.

Chapter nine presents the results of these interventions in a sub group of children after decompressive craniectomy.

Chapter ten is a discussion of the study, its aims, achievements and results put into context with published literature and UK practice (as established in the 2006 audit). It also discusses the implications for practice and future research and draws overall conclusions.

Chapter eleven concludes the thesis.

CHAPTER 2

EPIDEMIOLOGY, PATHOPHYSIOLOGY AND MANAGEMENT OF PAEDIATRIC TRAUMATIC BRAIN INJURY

2.1 Epidemiology of paediatric traumatic brain injury

Severe traumatic brain injury (TBI) in children continues to result in widespread mortality and morbidity in children worldwide (Reilly and Bullock, 2005). Although mortality rates in severe TBI have improved over the last two decades, (largely due to improvements in pre-hospital management, rapid and aggressive stabilisation and meticulous intensive care management), many suffer long term neurological sequalae, which affect the child, the family unit and society (Redmond and Lipp 2006).

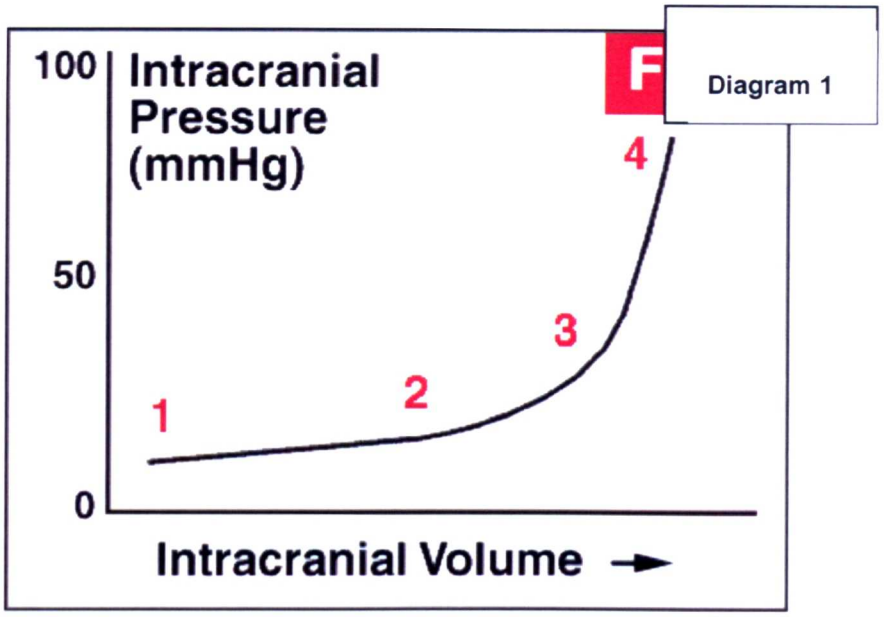
Children are not just 'little adults' though; they differ in the mechanism of injury (on many occasions), in their response to injury and in their recovery from injury (Giza et al 2007). Traumatic brain injury continues to be a significant problem among children and adolescents, peaking in two age groups: Under 5 years of age and mid to late adolescence (Adelson and Kochanek, 1998). Parslow et al (2005) found that in the UK alone, the prevalence rate of children with severe TBI (enough to warrant intensive care unit (ICU) admission) was 5.6 per 100 000 population per year, and of these, over half were severe enough to warrant invasive intracranial pressure (ICP) monitoring. The mechanism of injury varies with age - in the infant group non-accidental inflicted injury is commonest; in children less than 4 years the predominant mechanism of injury is falls, and in the older children motor traffic accidents, sports related accidents or assault (Keenan and Bratton 2006, Redmond and Lipp, 2006)

Additionally children who suffer TBI are generally not representative of the general paediatric population, in that they often have pre-existing behavioural problems as well as being from a lower socioeconomic background - all factors that will impact upon their recovery (Demellweek et al 2002, Parslow et al 2005, Anderson 2007).

2.2 Review of normal intracranial physiology and the pathophysiology of paediatric traumatic brain injury

The brain is enclosed within a fixed space (the cranium) once the fontanelles have closed, at around 18 months of age. Three components make up the volume within this fixed space: Brain (~80%), Blood (~10%) and Cerebrospinal fluid (CSF) (~10%) (Reilly and Bullock, 2005). The Monroe-Kellie doctrine explains the pressure changes within this body compartment, such that if any one of these three components increases in size/volume, there must be concomitant reduction in another component to maintain the same pressure. Compensation mechanisms ensure this happens up to a critical level, but once these fail, the intracranial pressure rises rapidly (Mayer and Chong, 2002). The pressure becomes so high that even a small change in pressure produces a rapid rise in ICP (see diagram 1).

Diagram 1: The pressure-volume curve



Reference: www.nda.ox.ac.uk/wfsa/html/u08/u08_014.htm

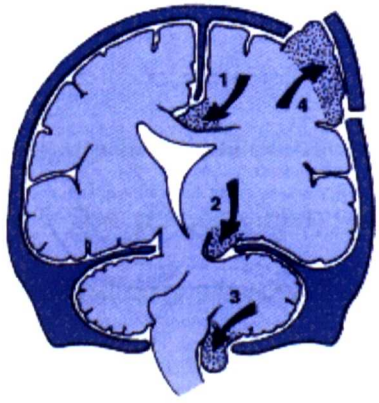
accessed 8.2.08

In the case of TBI, it is mostly brain oedema or space occupying lesions (eg extradural haematomas or other trauma-induced collections) that increase the volume in this cavity. In paediatric TBI diffuse cerebral oedema is a more significant problem than in adults, with Adelson and Kochanek (1998) claiming that up to 44% of children exhibit diffuse cerebral oedema. Reilly and Bullock (2005) suggest that this oedema can be caused by both classic cerebral oedema and by increased intravascular blood volume, occurring due to either arteriolar vasodilatation and/or venous obstruction. Cerebral oedema can occur early on in the course of injury in children, with the peak incidence reported in the first 72 hours after injury (Adelson and Kochanek 1998; Stocchetti et al 2007). Hence the other two compartments must compensate

by diverting cerebrospinal fluid (CSF) (producing less and reabsorbing more) and diverting some venous blood from the cerebral circulation into the dural venous sinuses (Keefe Marcoux 2005).

If the pressure continues to increase however, then the result is herniation of the brain, through anatomical openings in the skull (from areas of high to low pressure). Downward herniation through the foramen magnum (3), results in severe brain stem compression and ultimately brain stem death (Vincent and Buerre 2005; Robinson 1997). (See Diagram 2 below)

Diagram 2: Herniation of the brain

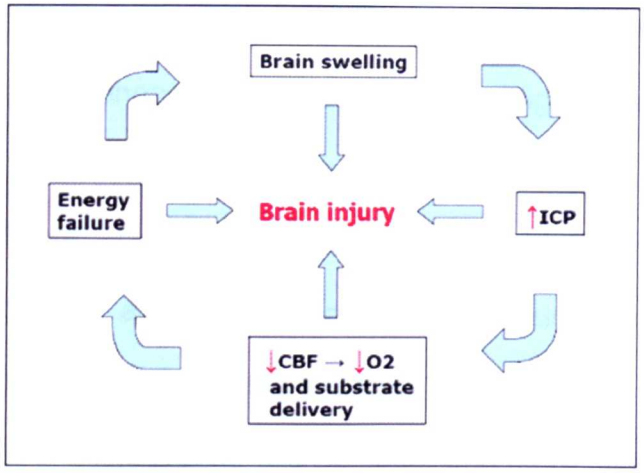


Reference:medinfo2.psu.ac.th/anesth/education/trauma2.html

accessed 8.2.08

As the brain continues to swell, perfusion to the brain is reduced, resulting in further hypoxia of already damaged tissue and a vicious cycle of ongoing injury (see diagram 3)

Diagram 3: Cycle of increasing intracranial pressure



Reference: http://www.rescueicp.com/frameset4.html

accessed 8.2.08

Brain injury is classified into two phases: a) primary injury and b) secondary injury. The primary injury is the initial brain insult itself due to traumatic impact, and only preventative measures can impact upon this. The primary injury can result in focal lesions (haematomas, contusions) or diffuse injury - where there is widespread axonal injury tearing and shearing. This diffuse axonal injury (DAI) is more common in children, due to their large head to body size ratio, their weak neck musculature and the increased elasticity of the bone and soft tissue (Adelson and Kochanek 1998).

Secondary injury is the cascade of events that occurs secondary to cellular injury: oedema, capillary leak and activation of a systemic inflammatory response. It can also be produced and exacerbated by other secondary insults such as hypoxia and hypotension, occurring after the injury, which are

also much more prevalent in children (Pigula et al 1993, Coates et al 2005). It is this secondary injury that the acute care management (including intensive care management) is aimed at avoiding or minimising.

There are a number of factors that affect both the injuries sustained in children and their response to the injury, which (certainly in small children) makes them different to adults.

- The cranial vault is softer and more pliable
- Fontanelles may be open (up to around 18 months)
- The bone and dura are very vascular
- The brain in small infants has a higher water content
- There is poorly developed cerebral vascular autoregulation
- The head is proportionally large compared to body size

(Reilly and Bullock, 2005)

2.3 Brain injury severity and the need for intensive care unit admission

Brain injury can be defined as mild, moderate or severe predominantly by assessment of neurological state using the Glasgow Coma Scale (GCS). It is important that the 'best GCS' post-resuscitation is used when predicting prognosis (Helmy et al 2007). Any patient presenting with a GCS of ≤ 8 (post-resuscitation) is classified as a severe injury (they will be unresponsive and require advanced airway support), a GCS 9-12 is considered a moderate head injury and 13-15 are generally mild head injuries (Weinstein 2006;

Moppett 2007). However the assessment of the GCS in children is often unreliable (Weinstein 2006; Gill et al 2004; Rowley and Fielding 1991) and so GCS alone cannot rule out the need for neurosurgical referral or intensive care. Neuroimaging is also an important indicator of injury severity and the need for neurosurgical intervention. There are some grading systems for CT scan severity in TBI (eg the Marshall score grades I - VI) with a higher score indicating a more severe lesion. In addition, some lesions on CT scans seem to predict a worse prognosis, meningeal haemorrhage being one (Bahloul et al 2009; Marshall et al 1992). The National Institute for Clinical Excellence (NICE) has set out guidelines for the indications for computerised tomography (CT) scanning in children (NICE, 2007). The World Federation of Paediatric Intensive Care Society (WFPICS) guidelines also utilise these two factors - post-resuscitation GCS ≤ 8 and/or an abnormal CT scan (interpreted by a paediatric neurosurgeon or a radiologist) to indicate the need for invasive intracranial pressure monitoring (Adelson et al 2003) and hence transfer to a PICU with neurosurgical services.

2.4 Intensive care management goals

Over the last decade, intensive care management has changed from care directed primarily towards reducing the ICP, to additionally ensuring adequate perfusion to the injured brain, as measured by cerebral perfusion pressure. (Robinson 1997). Cerebral Perfusion Pressure (CPP) is calculated by the formula:

CPP = Mean Arterial Blood Pressure (MAP) – Mean ICP

There is some evidence to support age-related variations in target values for CPP. See Table one for the range of normal ICP and treatment thresholds based upon these target values.

Table 1 Age-dependent ICP and CPP values

There remains limited, but increasing, evidence to support the value of this 'goal-directed' therapy on patient outcomes, both survival and quality of neurological outcome (Downard et al 2000; Catala-Temprano et al 2007; Carter et al 2008). The key factors for ICU management are therefore: maintaining a low and stable ICP (below a critical age-dependent value), avoiding hypoxaemia and preventing drops in CPP.

2.5 The development of international management guidelines

In 2003, the World Federation of Paediatric Intensive Care (WFPIC) produced a key document for paediatric clinicians worldwide, with the publication of 'Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children and Adolescents' (Adelson et al 2003). Previously

only adult guidelines were available. The guidelines were produced following appraisal of all the available evidence, and made recommendations or presented guidance (where possible) in relation to all aspects of severe TBI management. However, although these guidelines attempt to be 'evidence-based', they are (by their own admission) based on very limited paediatric evidence. There are no prospective randomised controlled trials of any of the therapies. So of the 19 chapters written, no standards of treatment could be attained and only eight guidelines were actually produced. The main argument in support of guidelines is to improve patient outcomes (both mortality and morbidity) and make health care practices between centres comparable and auditable, with the aim of minimising suboptimal care (Sekula et al 2005). There is some evidence from adult TBI practice to support a guideline-directed strategy. Two UK studies (Clayton et al 2004, Patel et al 2002) and one US study (Bulger et al 2002) demonstrated statistically significant reductions in mortality from aggressive protocolised TBI intensive care management. However, these two UK studies were both 'before and after' studies, and the US paper was a retrospective study, which weakens the evidential strength that the guidelines achieved this impact.

2.6 The treatment of intracranial hypertension

The estimated incidence of children who may develop intracranial hypertension in paediatric traumatic brain injury is as high as 21-42% despite optimal medical and surgical management (Adelson et al 2003). Patient

outcomes are extremely poor with high mortality rates when the ICP is sustained >40mmHg (Carter et al 2008; Adelson et al 2003).

Treatment guidelines focus on levels (or tiers) of therapy to guide clinicians in their utilisation of various therapeutic strategies. When treating intracranial hypertension, the clinical team must firstly ensure that the basic PICU management of the child is optimised: fever is controlled; the child is positioned midline; with head elevated 20 - 30°; is well resuscitated (with an optimal CPP); is well sedated; well oxygenated; and has a low normal PaCo2 (~35-40mmHg). Raised intra-abdominal pressure (eg a blocked urinary catheter) and seizures as possible causes of increased ICP must be ruled out. Pupillary reactions must be checked for any changes that may indicate the need for a repeat CT scan. Once these general measures are addressed, if the ICP is still above the treatment threshold for the age (most commonly >20mmHg), then additional therapeutic modalities are required. Diagram 4 shows a flowchart for raised ICP management (based on the WFPICS guidelines).

Diagram 4: Flowchart for the management of raised ICP

Treatment threshold is a sustained ICP rise

above target limit for > 5 minutes

Ensure all general management outlined is being achieved if not correct this first

- Head up 20 – 30 °, head midline, no obvious venous obstruction, loosen collar if in situ
- Keep CPP >40 if 0-2yrs; >55 if >2-6yrs; >65 if > 7 years
- SpO_2 > 98%; PaO_2 > 100mmHg: $PaCO_2$ 35-38 mmHg
- Temp between 36.5 - 37° C
- Sedation bolus
- Check for evidence of seizure activity?
- Any pupillary changes?

→ Consider mass lesion – volume CT SCAN & Neurosurgical Opinion

↓

First Tier therapy

- In patients with **ventriculostomy remove CSF** via EVD
- If ICP still raised:
- Give **Mannitol** 20% (0.5g/kg over 15 minutes until plasma osmolality 320 mOsm; Na> 140) **Or**
- Give **Hypertonic saline** (3%) 5ml/kg bolus (Caution if osmolality >320mOsm/l Contraindicated if >360 mOsm/l)
- If mannitol/saline required **→ Insert EVD at first opportunity**

↓

ICP STILL RAISED move on to Second Tier Therapies

- Hyperventilation down to 30 – 35mmHg
- Consider further CSF drainage (by EVD)
- Trial of Thiopentone- see response to bolus dose; if effective in reducing ICP → start infusion (3-5 mg/kg/hr)
- Seek Neurosurgical Consultant opinion regarding decompressive craniectomy
- Consider raising CPP further using inotrope infusion and titrate to maintain systemic hypertension

↓

ICP STILL RAISED or impending herniation

- Further dose of Mannitol 0.5g/kg or Hypertonic saline 3%
- ICU consultant to consider whether therapeutic cooling an option
- Hyperventilation
- Urgent neurosurgical consultation and CT scan

2.7 First tier therapies for intracranial hypertension

The initial step is to ensure that the child is adequately sedated or analgesed. Additional sedative and opiate drugs can be administered to minimise cerebral oxygen demand and ensure sedation and analgesia. If these measures fail to reduce ICP, then recommendations suggest that osmotic diuretics, CSF drainage (if a functioning ventriculostomy is in situ) or neuromuscular blockade are used (Adelson et al 2003).

2.8 Hyperosmolar therapy and osmotic diuretics

Hyperosmolar drugs/fluids or osmotic diuretics work by producing an osmotic gradient to drag fluid out of cells (including brain cells) and into the intravascular space. The two agents most commonly used are mannitol (10% or 20%) and hypertonic saline (HTS) (1.7-29.2%), either alone or in combination with loop diuretics (Helmy et al 2007). There is evidence to show that these agents transiently reduce brain volume (hence reducing ICP) and reduce blood viscosity (hence improving blood flow through microvasculature) (Sorani and Manley, 2008). Mannitol may also have an antioxidant effect (Adelson et al 2003). Advocates of HTS argue that in addition to the effects of mannitol cited above, HTS may also increase regional brain tissue perfusion, diminish inflammatory response to brain injury, restore normal membrane potentials, increase cardiac output and mean arterial pressure, and reduce extravascular lung water (Suarez 2004). Hypertonic saline (HTS) is a newer agent and has been studied more

extensively than mannitol, even though mannitol is the drug more frequently used in clinical practice (Sorani and Manley 2008, Khanna et al 2000). The most notable advantages of HTS lie in its lack of diuretic effect and its preservation of intravascular volume, thereby reducing the risk of a reduction in CPP (Khanna et al 2000).

Mannitol, administered in bolus form, produces a transient reduction in ICP, but its side effects include intravascular volume depletion (due to its diuretic effect), 'rebound' intracranial hypertension, and renal insufficiency (Khanna et al 2000). Its clinical use is therefore more limited by the child's serum osmolality than HTS. A recent meta-analysis on the dose-response relationship of mannitol to ICP, found a highly significant reduction in ICP when the ICP was initially >30mmHg (Sorani and Manley, 2008). However a Cochrane review in 2007 found so few eligible trials in head injured patients (adults or children) that they were unable to make any recommendations on its use in head injury (Wakai et al 2007).

Both drugs are commonly administered by bolus form, however, Khanna (2000) studied the use of a continuous infusion of 3% HTS in 10 children (titrated to a target serum sodium and ICP) with severe refractory intracranial hypertension. They found that the increase in serum sodium concentration significantly reduced ICP and increased CPP, and that the hypernatraemia and hyperosmolarity were well tolerated. An earlier adult study (Qureshi et al 1998) also demonstrated the effectiveness of a continuous infusion of 3% HTS on ICP reduction.

2.9 CSF drainage

CSF drainage (unless a ventricular catheter is the ICP measurement device) requires neurosurgical personnel to place an additional ventriculostomy catheter (or external ventricular drain) into the lateral ventricle. Clearly this therapy is dependent upon access to neurosurgical expertise, and in two recent UK studies this modality was still rarely used as a first tier therapy (Morris et al 2006; Tume and Baines 2008). The physiology behind this therapy is based on the Monro-Kellie doctrine, whereby reducing the volume of one of the compartments in the intracranial cavity will cause a reduction in the pressure in the entire cavity.

Despite the logic of this therapy, there has been little research examining the efficacy of this therapy, with only two publications reporting the efficacy of this intervention in severe head injured patients (Kerr et al 2000; Kerr et al 2001). In a crossover study of 31 head injured adults Kerr et al (2000) found that with intermittent CSF drainage the ICP decreased below 20mmmHg in one third of patients. However 24 patients did not show a sustained reduction in their ICP (defined as >3mmHg for 10 minutes), but they could not show any variable that had a direct correlation with ICP response. In a further investigation, in 58 head injured adults, Kerr et al (2001) reported that a 3ml CSF withdrawal resulted in an ICP reduction of 10% and a 2% increase in CPP which was sustained for 10 minutes. A small pilot study (unpublished) in severe head

injured children by Gough et al (2007) demonstrated that intermittent CSF drainage episodes kept ICP below the target value on 87% of occasions in the first 24 hours and 83% in the second 24 hours following EVD placement. Additionally it also showed the median reduction in ICP following CSF drainage episodes was 6.5mmHg on day one and 8mmHg on day two. However this is only pilot data and due to small numbers no conclusions can yet be drawn.

Whether continuous or intermittent CSF drainage is more effective in TBI remains unknown, with no evidence to support either method. The only reported problem with continuous drainage is frequent occlusion of the catheter with oedematous brain tissue.

2.10 Neuromuscular Blockade

Physiologically any intervention that produces a cough will increase ICP, as intrathoracic pressure increases and impedes cerebral venous drainage, and the use of muscle relaxation has been demonstrated to be effective in controlling ICP (especially during interventions such as endotracheal suctioning, where a cough may be induced) (White et al 1982; Garradd and Bullock 1986; Werba et al 1993; Kerr et al 1998).

The routine use of neuromuscular blockade in all TBI patients is however, now avoided due to the problems of: immobility, muscle weakness, nosocomial pneumonia, accumulation of the drug; prolongation of the ICU stay and inability to assess neurology (except pupils) (Prielipp and Coursin

1995; Hsiang et al 1994; Adelson et al 2003). Additionally, the child needs to be adequately sedated under induced paralysis, to avoid the stress response and potential awareness of being 'awake' under the paralysis. If utilised, newer muscle-relaxants that result in minimal accumulation (eg atracurium) are preferred. These agents have a short half life, allowing rapid excretion once the drug is stopped, and consequently allow a more rapid assessment of underlying neurological responses.

2.11 Mild hyperventilation

The final first tier therapy recommended is 'mild' hyperventilation (defined as PaCO2 30 – 35mmHg). Lowering the carbon dioxide blood level produces some degree of cerebral vasoconstriction, which in line with the Monro-Kellie doctrine reduces one component (blood) and thereby lowers ICP. However, aggressive and persistent hyperventilation was found to produce deleterious results on patient outcome (Muizelaar et al, 1991) by causing cerebral ischaemia, and now only mild hyperventilation is advocated for short periods (Adelson et al, 2003). The use of jugular venous saturation monitoring (Sjo_2) as a gauge of cerebral blood flow has been advocated, if more than mild hyperventilation is used (Skippen et al 1997; Helmy et al 2007). Sjo_2 has technical limitations however (especially in smaller children) and recent research suggests that it does not reliably reflect brain ischaemia (Poca et al 2007: Soustiel et al 2006; Coles et al 2007). More aggressive

hyperventilation (down to a PaCo2 less than 30mmHg) is now only advocated if there are indications of impending brain herniation (Adelson et al, 2003).

2.12 Second tier therapies for intracranial hypertension

As with the previous transition from general measures to first tier therapies, if these first tier therapies are failing to control ICP, then all of the basic PICU measures should be re-evaluated and consideration given to repeating the CT scan, before moving on to second tier therapies. The recommended second tier therapies for children are: the use of moderate hypothermia (32 - 34°), barbiturate therapy and decompressive craniectomy.

2.13 Moderate hypothermia

Hypothermia has been studied in relation to cerebral protection since the 1960s, augmented by the development of cardiopulmonary bypass for complex cardiac surgery (Clifton 2004). Hypothermia reduces total body metabolic rate and hence tissue oxygen demands, which allows periods of ischaemia to be tolerated for longer periods of time (Fukuda and Warner 2007). Since the publication of WFPIC guidelines (in 2003) a Phase II clinical trial in severe paediatric traumatic brain injury (Adelson et al 2005) showed this therapy to be safe and effective in reducing ICP in children. This differed from the adult trials that produced conflicting results and more adverse effects from hypothermia (Clifton et al 2001; Narayan 2001; Alderson et al 2005). However Adelson et al (2005) could demonstrate no improvement in

neuropsychological outcome (at 6 months). At the time of the UK survey (Chapter 4) many UK PICUs used this therapy earlier than second tier (Tume and Baines 2008).

In July 2008, the results of a significant new mutlicentre randomised controlled trial (Hutchinson et al 2008) came out. This trial in severe head injured children cooled for 24 hours (within 6 hours of injury) showed significantly worse 6 month outcomes and mortality in the hypothermia group. Hence, the use of moderate hypothermia in PICU practice has virtually stopped. Hypothermia carries risks, most notably coaguloplathy, skin breakdown, infection and arrhythmias (Adelson et al 2005). However it does effectively reduce ICP in children, but on rewarming frequently a 'rebound' effect on ICP is observed. Due to the inducement of shivering it also necessitates the use of neuromuscular blockade.

2.14 Barbiturate therapy

Pharmacologic coma can be induced iatrogenically which significantly reduces the metabolic requirements of brain cells by suppressing neurotransmission (Fukuda and Warner 2007; Cook and Weant, 2007). Barbiturates (thiopentone or similar drugs) have been used for many years in anaesthetic practice. These drugs are thought to reduce ICP and confer neuroprotection by reducing cerebral metabolic metabolism (by up to 50%) and altering vascular tone (Wilberger and Cantella 1995; Cook and Weant 2007, Adelson et al 2003). It has also been suggested that barbiturates may

confer additional neuroprotective effects beyond that of just ICP reduction, in terms of inhibition of free radicals and membrane stabilisation (Adelson et al 2003). However the side effects of these drugs limit their widespread use. The most significant side effects seen in children are direct myocardial depression, combined with vasodilatation that produces significant haemodynamic instability necessitating inotropic support in virtually all children receiving these drugs (Wilberger and Cantella 1995; Cook and Weant 2007). Accumulation of the drug with continuous infusion is also problematic and excretion of the drug can take days, making assessment of the underlying neurology difficult (Russo et al 1997).

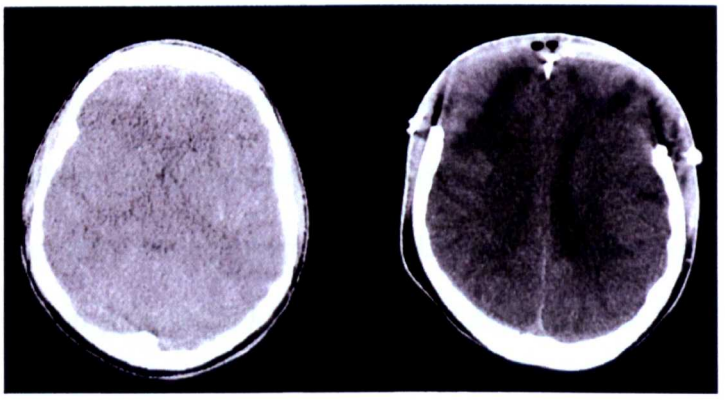
The WFPICS guidelines recommend that electroencephalographic (EEG) monitoring be instituted to guide barbiturate therapy, and that to achieve optimal reduction in cerebral metabolism, the drug should be titrated to produce burst suppression on the EEG (using cerebral function monitoring (CFAM) (Adelson et al 2003). There remains very little research on the use of barbiturates in infants and their effects on the developing brain remain unknown. The use of the metabolic suppression anaesthetic agent propofol as an infusion is discouraged due to the potential for propofol infusion syndrome in children (Sabsovich et al 2007).

2.15 Decompressive craniectomy

Another second line therapy option available in severe refractory intracranial hypertension is decompressive craniectomy (DC) or surgical removal of part

of the skull. This procedure allows swelling of the brain to occur more freely. By the Monro-Kellie doctrine this essentially 'opens the fixed cavity'. The first report of DC was written in 1894 (Citero and Andrews, 2007) and although returning to favour in the last decade, it remains controversial. Hutchinson et al (2006) report two main surgical methods of decompressive craniectomy: (i) if there is unilateral hemispheric swelling then a large unilateral fronto-temporo-parietal craniectomy is performed or (ii) if there is diffuse bilateral hemispheric swelling then a large bilateral fronto-temporo-parietal craniectomy and dura opening and division of the anterior parts of the falx is suggested (See the CT scan below of a patient before and after a DC).

Diagram 5: CT scan of patient before and after decompressive craniectomy



http://www.rescueicp.com/frameset4.html accessed 10.2.09

At craniectomy the bone flap can either be placed subcutaneously (in an abdominal subcutaneous pocket) until early replacement or can be discarded. At a later point, once the oedema has subsided and the skull window has become slack, a cranioplasty is performed, where either the patient's

autologous bone flap is replaced or a specialised moulded ceramic, titanium plate is made to replace the bone (Staffa et al 2007; Kuo et al 2004).

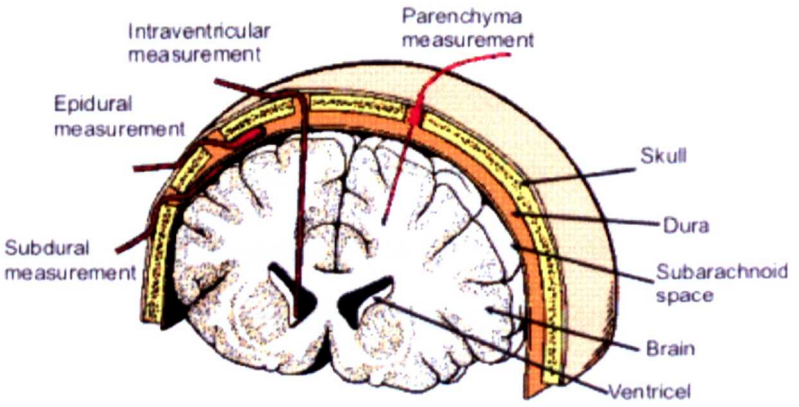
A recent systematic review in TBI concluded that the routine use of DC cannot be recommended for patients over 18 years with severe TBI and refractory increased ICP (Saquillo and Arikan 2006). A single prospective randomised controlled trial in children however showed promising results (Taylor et al 2001). This trial evaluated the very early use of DC (bitemporal craniectomy or maximal medical therapy) in 27 children with severe TBI and refractory intracranial hypertension. DC significantly lowered overall ICP and reduced the frequency of ICP spikes in the 48 hours after treatment. Additionally it resulted in a marginally better, but not a significant trend to good neuropsychological outcome at 6 months post injury. As the current evidence is still too weak to generate guidelines for the use of this therapy in children or adults, one international mutlicentre randomised controlled trial is currently taking place in the UK to evaluate this therapy: - the RESCUEicp trial in patients 10-65 years (Hutchinson et al 2006). The argument against this surgery is that it risks producing survivors following very severe injury, in whom quality of life is very poor. So, although technically feasible, many would argue that selection of suitable patients is essential to avoid producing severely disabled children. They would subsequently pose a huge social and economic burden to both their families and society, with questionable benefit to the patient. This had led many units to set out clear contraindications for this procedure. These are frequently cited as: persistent fixed and dilated

pupils, devastating injury where survival is not expected, bleeding diatheses and in some centres children under 12 months with suspected non-accidental injury (Hutchinson et al 2006; Potter et al 2007). Citerio and Andrews (2007) argue that the ethical dilemma of this procedure lies in the uncertainly of obtaining benefit (in terms of ICP control and neuropsychological functioning) at the time of surgery.

2.16 Intracranial pressure monitoring

ICP has been able to be measured directly since the 1960s, albeit in a crude form (Lundberg 1960). It can now be monitored from various anatomical areas within the intracranial cavity: the subdural space, the epidural space, the lateral ventricle of the brain or the brain parenchyma itself (see diagram below).

Diagram 6: ICP monitoring locations



Ref: http://www.medit.hia.rwth-aachen.de/images/research/icpanalysis_brainpressure.gif accessed 6.5.09

The two commonly used systems worldwide today are ventricular catheters and fibre optic catheters inserted into the brain parenchyma (Smith 2008). Ventricular catheters are considered by many to be the gold standard for accuracy, but carry a greater complication rate with insertion and also with infection and blockage (North and Reilly 1986; Smith 2008). Parenchymal devices are claimed to be simple to insert (not necessarily requiring neurosurgical personnel), have good accuracy with minimal risks and complications (Koskinen et al 2005; Gambardella et al 1993; Signorini et al 1998; Brean et al 2006).

Historically ICP has been measured by many different devices and systems, with some clear differences in measurements between different devices and measurement systems (Brean et al 2006; Gambarella et al 1993; North & Reilly 1986). Recently, Koskinen and Olivecrona (2005) compared the accuracy between ventricular and intraparenchymal devices simultaneously and concluded that with current fibre optic technology, there was on average a maximum of 2mmHg higher readings in the parenchymal devices. The new devices also appeared to have minimal 'drift' over time, and they claim them to be fairly accurate even by day 9 of monitoring. The reported concerns with these fibre optic devices are that because they are zeroed (to atmospheric pressure) only at the time of insertion, they cannot be zeroed, unless they are replaced, hence this theoretical risk of 'drift' over time. Thus it is critical to

use just one type and brand of ICP measurement device for the duration of a study, to reduce potential variation in this variable. The Codman Microsensor ™ (Johnson and Johnson UK) parenchymal catheter we used has been demonstrated to be a reliable measurement device, have good correlation with ventriculostomy readings (r=0.79) and minor zero drift over time (Koskinen and Olivecrona 2005) in a recent prospective study in 128 patients.

There remains some ongoing debate about the usefulness of ICP monitoring in affecting patient outcomes (Cremer et al 2005: Smith 2009; Salim et al 2008) but this discussion is beyond the scope of this thesis.

2.17 Therapies and factors that have been shown to affect ICP or CPP

There are a number of intensive care therapies that can affect ICP in the intensive care unit (some of which have already been discussed in the tiers of therapy) either because they increase cerebral blood flow or impair cerebral venous return, reduce cerebral blood flow or transiently reduce brain size. These will be discussed briefly as they act as potential confounders in the forthcoming study. A confounder is defined in this study as any factor that may potentially affect the primary outcome measure of ICP.

Although many of the therapies are initiated by the medical team, they are then maintained and manipulated by the bedside nurse to achieve patient specific target parameters. It therefore is difficult to separate them into definitive nurse or medical interventions, as they are in general managed by

the multidisciplinary ICU team; however the key nursing responsibilities within each therapy have been highlighted.

Carbon dioxide and oxygen level in the blood

The effect of carbon dioxide (PaCo2) has a direct impact on the diameter of the cerebral blood vessels in response to autoregulation and consequently blood flow into the brain (Lodi et al 1998). As PaCo2 rises the cerebral vessels dilate in an attempt to increase blood flow and wash out excess hydrogen ions (H+). This increase in cerebral blood flow will increase the ICP. Conversely prolonged hyperventilation has been shown to adversely affect patient outcomes, potentially worsening ischaemia (Muizelaar et al 1991; Skippen et al 1997; Coles et al 2007), although short duration hyperventilation has been used to acutely reduce ICP. Severe hypoxia will cause a similar response in dilatation of the cerebral blood vessels in an attempt to increase cerebral blood flow and hence oxygen delivery. In these children end tidal carbon dioxide ($EtCo_2$) is measured (and displayed) continuously and this level is kept within tight parameters (35 – 40 mmHg) by the bedside nurse.

Time course from injury

It is known that the peak incidence of cerebral oedema in children occurs in the first 72 hours after injury (the hyper acute phase) (Adelson and Kochanek

1998; Stocchetti et al 2007) and gradually like any injury, oedema subsides over time.

Core temperature

Moderate hypothermia reduces total body metabolic rate and hence tissue oxygen demands which allow periods of relative ischaemia to be tolerated for longer periods of time (Fukuda and Warner 2007) and has proved a promising therapy since the 1960s. This has been discussed in the therapies section previously and although lowering core temperature does reduce ICP (McIlvoy 2007; Adelson et al 2005; Shafi and Mariscalo 2006; Tokutomi et al 2003) its effects on long term outcome have not been substantiated and there is recent suggestion that hypothermia may adversely affect long term outcomes (Hutchinson et al 2008). However avoidance and control of fever is essential to avoid increasing cerebral metabolic demand and the temperature of these children are kept fairly tight (between 36 - 37° Celsius) by the bedside nurse.

Administration of thiopentone (a barbiturate) or propofol

The effects of these two anaesthetic agents have already been discussed, but both have the ability to significantly reduce ICP through cerebral metabolic suppression (Cook and Weant 2007; Dereeper et al 2002; Eisenbery et al 1988; Kelly et al 1999; Pittman et al 1989; Russo et al 1997) although the limitations of using propofol in children relate to the hazards of developing

propofol infusion syndrome (Sabsovich et al 2007) and as such this agent is not recommended for use in PICU (except in very short term situations) (Adelson et al 2003).

The effect of sedative and analgesic agents

In intensive care it is often difficult to separate the effects of sedation and analgesia as these drugs are almost always used simultaneously and Mayer and Chong (2002) suggest that this combination of sedative-hypnotic and analgesics is the most effective combination in patient with TBI.

Sedation is used in TBI to attempt to reduce cerebral metabolism, 'rest the brain' and abate the 'energy stress' present in injured cells (Kerr et al 1998; Adelson et al 2003; Cook and Weant 2007). Adequate sedation is also generally required in critically ill children to facilitate tolerance to medical and nursing therapies and ensure invasive lines remain insitu. In muscle relaxed patients the consequences of under sedation include unrelieved pain and anxiety which can increase sympathetic outflow, increase global oxygen consumption and carbon dioxide production resulting in an increased in metabolic rate (Arbour 2004). This is not a good state in any critically ill child, but certainly not in severe brain injury, where the cornerstone of management is to rest the brain. Providing adequate analgesia to critically ill children is both a moral obligation and essential to prevent this physiologic stress response. However the optimal sedative or analgesic agents for use in TBI remain unclear, with reports of some sedative agents actually worsening ICP

by increasing cerebral blood flow (Nadal et al 1998; Bourgoin et al 2003: Leone et al 2004; McClelland et al 1995; Sperry et al 1992). In addition to the continuous infusion of these drugs, they are also prescribed in bolus form 'as required' and this allows the bedside nurse to administer extra doses if they think the child is in pain or inadequately sedated or prophylactically prior to noxious procedures.

The effect of neuromuscular blockade

Neuromuscular blockade has been shown to reduce ICP during airway procedures (Kerr et al 1998; White 1982; Werba 1993). However some question the routine and continuous use of these agents in head injured patients in the ICU (Prielipp and Coursin 1995; Hsiang et al 1994). Ensuring the child is adequately muscle-relaxed is the responsibility of the bedside nurse and there are additional 'as required' prescriptions of these drugs to ensure this can be administered.

The effect of lignocaine hydrochloride (1%) administered endotracheally

Lignocaine, local anaesthetic agent, instilled directly into the tracheobronchial tree or given intravenously has reportedly minimised the ICP response to airway procedures including endotracheal suction in a number of papers (Brucia et al 1992; Yano et al 1986; Bilotta et al 2008; Donegan and Bedford 1980; Hamil et al 1991). Endotracheal lignocaine is prescribed 'as required'

before endotracheal suctioning, so the administration of this drug is again at the discretion and clinical judgement of the bedside nurse.

The effect of Hyperosmolar agents (mannitol or hypertonic Saline)

Both these agents have both been shown to be effective in transiently reducing brain volume (by creating an osmotic gradient) and their effects appear to last for a maximal time of around four hours after administration (Weant and Cook 2008; Knapp 2005; Khanna et al 2000; Qureshi et al 1998; Sorani and Manley 2008).

External ventricular drainage (via ventriculostomy or EVD)

As discussed previously in the therapies section, draining CSF reduces one of the components in the cranial cavity and thus effectively lowers ICP (Ghajar et al 1993; Kerr et al 2000; Kerr et al 2001; Gough et al 2006). These drains can be used continuously (draining only when a set level of ICP is exceeded) or more commonly in TBI intermittently as needed when ICP exceeds the treatment threshold. As these therapies are known to affect ICP they must be accounted for in the data collection of any research. These drainage systems are maintained and adjusted by the bedside nurse and there are standard surgical orders for the opening of the system for ICP reduction.

The level of Positive End Expiratory Pressure (PEEP)

PEEP increases intrathoracic pressure (by applying pressure which distends the alveoli at the end of expiration) and in doing so impairs venous return back into the heart, consequently impairing cerebral venous drainage. Levels of around 5cm H_20 are used in most patients to prevent atelectasis and improve oxygenation, but levels higher than this may adversely affect ICP and MAP (Georgiadis et al 2001; Huynh et al 2002; Mascia et al 2005; Muench et al 2005).

A semi-rigid cervical collar (which is fastened up)

In children with severe traumatic brain injury, spinal cord injuries cannot be ruled out from radiography alone so some cervical spine protection must be in situ until clinical clearance can be obtained; this necessitates the use of a rigid collar unless the child is heavily sedated and/or paralysed. The presence of a properly fitting rigid cervical collar (which is fastened up) has been shown in a number of adult studies to increase ICP by compressing the jugular veins (thus impeding cerebral venous return) (Ahmad 2001; Craig and Nielsen 1991; Davies et al 1996; Ho et al 2002; Kolb et al 1999; Porter and Allison 1999). Thus in our unit practice guidelines are to open these cervical collars (avoiding compression of the jugular veins) if the child is heavily sedated or muscle relaxed. They are only fastened up when the child is moved or turned and the bedside nurse man ages this.

The degree of elevation of the head of the bed (HOB)

The degree of elevation of the head of the bed (HOB) has been shown to impact on the ICP. As the head of the bed (and hence the patient's head) is elevated, ICP falls slightly (because cerebral venous return is improved). A number of adult studies have found the optimal HOB elevation for severe head injured patients is 20-30° elevation to produce a reduction in ICP without causing a reduction in MAP. Additionally if the head is elevated too high, then hip flexion occurs which increases intra-abdominal pressure and the positive effect on ICP is lost (Feldman et al 1992; Fan 2004; Ng et al 2004; Rosner and Coley 1986; Schultz-Stubner and Thieux 2006). The responsibility for appropriate patient positioning (ie head elevated and midline) lies with the bedside nurse.

2.18 Patient outcomes after TBI in children

More children are now surviving TBI; however there may be significant long-term neurological, psychological and social impairments which not only affect the child but the entire family unit. Assessing neuropsychological outcome after TBI in children can also be problematic, as baseline (pre-injury) developmental measurements are rarely available. There is evidence that some of these children may have pre-existing behavioural disorders or cognitive deficits prior to the injury and these will impact upon their recovery (Demellweek et al 2002). Many argue that assessments at hospital discharge

and six months (post injury) are still to early too fully assess these children, as neurorehabilitation continues and the child grows, outcomes can still improve (Weinstein 2006). Adelson and Kockanek (1998) claim that children are more likely to have better functional outcomes than adults, but this is dependant upon many factors. Factors known to impact upon outcomes are: neural development at the time of injury, mechanism of injury, severity of injury (classified by early GCS and pathophysiology seen on early CT scan) and the presence and magnitude of any secondary insults (especially hypoxia and hypotension).

At least two qualitative studies have examined the impact that a child's TBI has had on both the parents and the child. Years after the injury, parents still reported high levels of distress about their child, specifically in regard to school performance, the child's lack of friends, their inability to control their angry feelings and apathy (Prigatano and Gray 2007; Souza et al 2007). This is magnified in young families with little social support, financial limitations and parents with their own psychiatric or medical problems (Verhaeghe et al 2005).

2.19 Assessing neuropsychological outcomes

Ideally neuropsychological outcomes would be formally assessed by a neuropsychologist; however given the relative lack of this specialist expertise, other tools have been developed to allow a very basic assessment of outcomes in these patients that can be performed by clinicians. One

commonly used (albeit fairly simplistic) tool to evaluate neuropsychological outcome after TBI is the Glasgow Outcome Score (GOS), developed by Jennet and Bond in 1975. This scale has five categories/scores:

1 Good recovery

2 Mild - Moderate disability

3 Severe disability

4 Persistent vegetative state

5 Death

More detail about this scale can be seen in Appendix eleven. Although validated in adults, it is still used extensively in paediatric head injury trials.

This chapter of the thesis has attempted to provide an updated review of traumatic brain injury in children and the intensive care management of these patients, to set the scene for the interpretation of the thesis. It is critical that nurses understand the effects of these medical therapies and other factors that can impact upon ICP, CPP and MAP and that can be manipulated by the bedside nurse both before and after nursing interventions.

CHAPTER 3

REVIEW OF THE LITERATURE

3.1 Introduction

This thesis is concerned with the effect of selected nursing interventions in moderate to severe head-injured children in intensive care, so it was critical to first locate and review the published work in this field. This critical review of the literature, presented under key themes, establishes a clear rationale for the proposed study in a homogenous group of critically-ill head-injured children.

3.2 Search Strategy and results

An initial search was undertaken in October 2005, updated in May 2006, January 2007, January 2008, April 2009 and August 2009 to answer the clinical question: ***In children with moderate to severe head injury what are the effects of routine intensive care nursing interventions (endotracheal suction, turning and hygiene interventions) on intracranial pressure and cerebral perfusion pressure?***

Databases were searched using key search terms of:

Pediatric (US) and Paediatric (UK) Head Injury, OR Traumatic Brain Injury, OR Brain Injury AND Intensive Care to identify the appropriate patient group, coupled with *suctioning, endotracheal suctioning, respiratory physiotherapy, nursing activities, nursing care, nursing interventions, turning, moving, repositioning, log-rolling, washing, hygiene interventions, eye care, and mouth care* as the interventions AND *intracranial pressure OR cerebral perfusion pressure* as the outcome measure.

The databases searched were the Cochrane Library, Medline 1966-current, CINAHL 1982-current, Pubmed Central, Proquest and Science Direct.

Reference lists from key articles were reviewed and the former UK National Research Register was searched with key terms: head injuries, ICP, nursing cares and intensive care for completed but unpublished research on this topic. The ethOS Beta electronic thesis on-line service was also searched using these terms for any unpublished UK PhD thesis. A Zetoc database was searched again for these key terms and an alert was set up for key words of: traumatic brain injury, intensive care and intracranial pressure (ICP) and Cerebral Perfusion Pressure (CPP) from January 2006 to ensure any papers published on this topic could be rapidly identified.

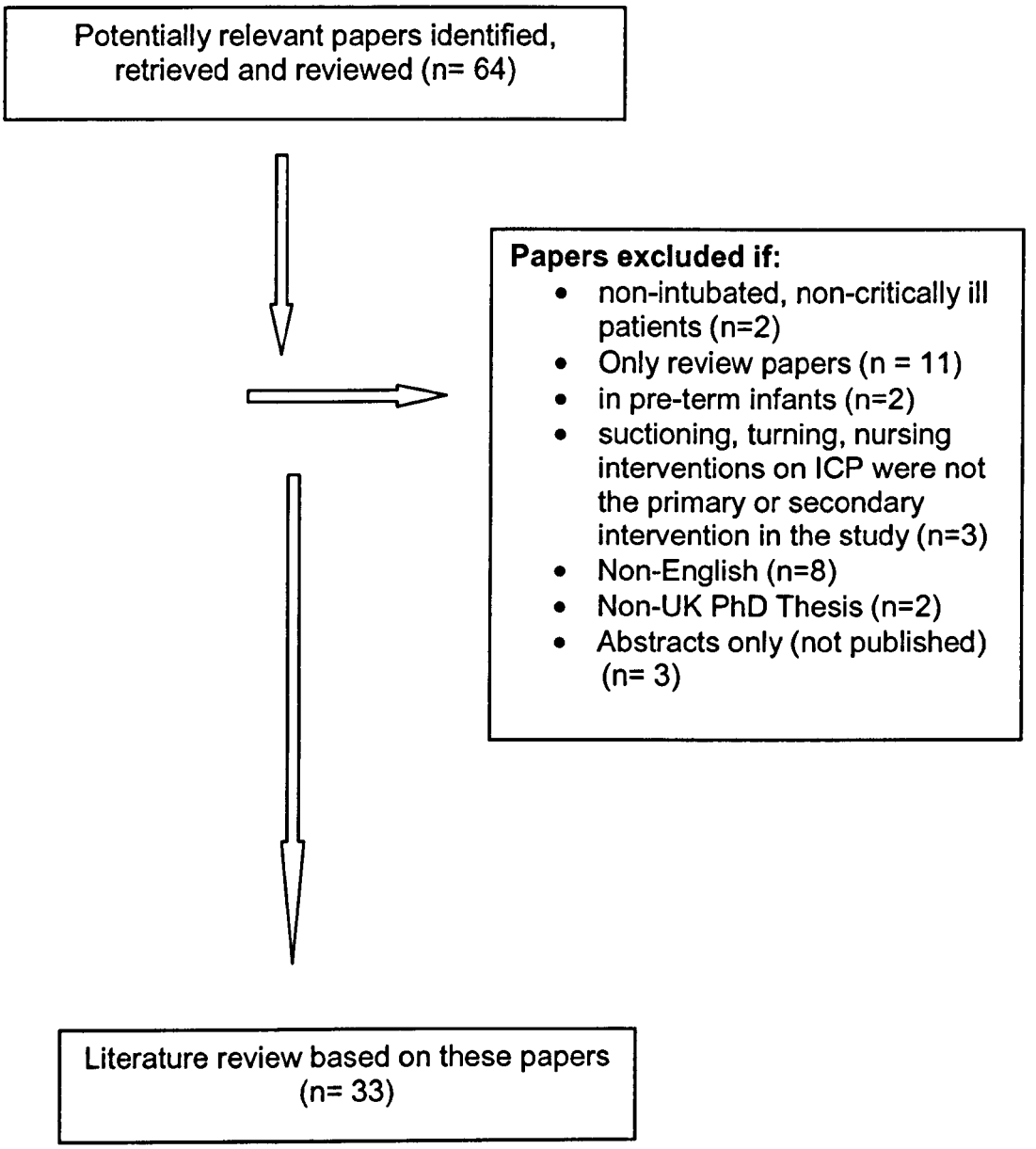
The search was limited to published articles (not abstracts) as full details of the study could not be reviewed. Non UK PhD theses were excluded as they could not be obtained and only English articles were included (as no translation was possible). A single reviewer (LT) reviewed all the papers using an appraisal framework described by Parahoo (1997 p363) and the QUOROM method of reviewing literature for meta-analyses (Moher et al 1999). This involved a critical evaluation of the methodology of the study looking at the research questions, validity and reliability issues, the research design used, data collection methods, the data analysis and the presentation of their results along with the conclusions drawn and identified limitations of the study. Papers were not formally graded and with some interventions

(turning and repositioning) the relative lack of published literature on this topic necessitated the review and presentation of all published papers.

The search results yielded 64 potentially relevant papers.

Diagram 7 shows the process of eliminating papers in the literature review.

Diagram 7: Selection of papers in the literature review



3.3 Classification of the quality of published evidence

The Evidence Based Medicine (EBM) movement, which began 20 years ago in North America, remains the most influential system in classifying research evidence in the health care environment (Sackett et al 2000). It describes three classes of evidence:

- Class I evidence is based on good quality randomised controlled trials
- Class II evidence is based on clinical studies in which the data was collected prospectively and retrospective analysis that were based on clearly reliable data. Types of studies include observational studies, cohort studies, prevalence studies and case control studies.
- Class III evidence is based on retrospectively collected data. Evidence used includes databases, clinical series, case reviews, case reports and expert opinion.

It is from this classification system that many medical standards and guidelines are derived. According to this, to be able to set a standard, there must be class I evidence; however on occasion class II evidence may be sufficient. Guidelines are normally based upon class II or class III evidence and only options can be suggested if there is only class III evidence (Adelson et al 2003).

3.4 Critical review of the literature

This literature review has found that virtually all research in this field is in adult patients, with much of it undertaken in the 1980s and early 1990s, when intensive care practices were quite different from current practice. The research in this area emerges into various themes (below) and the review will be presented under these headings.

- The effects of repositioning (moving/turning) on ICP/CPP
- The effects of hygiene interventions (washing) on ICP/CPP
- The effects of clustering cares versus spacing them out on ICP/CPP
- The effects of endotracheal suctioning (ETS)
  - -on ICP (general)
  - -the effect of coughing during ETS
  - -the effect on cerebral perfusion pressure (CPP)
  - -the effect on cerebral oxygenation
  - -the effect of respiratory physiotherapy (RP) (suctioning)
  - -the effect of hyperventilation during ETS
  - -recovery times after ETS
  - -the effect of pharmacological agents on the ICP rise during ETS

A summary of the 33 papers that the literature review is based on are provided in Appendix 1.

3.5 The effect of repositioning, moving or turning patients on ICP and Cerebrovascular (CBV) parameters

There are eight research papers published on this topic and only two involved children. Four of these are of reasonable quality in terms of their methodology, sample size, data analysis and presentation (Parsons and Wilson 1984, Lee 1989, Jones 1995; Hobdell 1989)) and the latter four studies are fairly weak and poorly reported studies (Rising 1993, Shalit and Umansky 1977, Boortz-Marx 1985, Muwaswes 1984). Three of the four good studies demonstrated significant ICP rises with repositioning (Parsons and Wilson 1984, Lee 1989, Jones 1995), but the study involving 13 children (Hobdell et al 1989) found no significant change in ICP with turning or changing head position. By five minutes post intervention Jones (1995) found that ICP was still elevated from baseline in 50% of these adults, however Parsons and Wilson, found that this ICP rise was transient and showed recovery towards baseline by 1 and 5 minutes.

Jones (1995) found that in the majority of adults (n=30) (with raised ICP from any pathology) who were turned from supine to left lateral (not log-rolled) 46% of patients still had an ICP above their baseline at five minutes after the turn. Their results are only presented as a change from baseline so no comment can be made with regards to clinical significance of this. Nor are the effects of any confounders presented to put each turning

episode into context and the measurements appear to be mean data across all the episodes, which makes it difficult to interpret.

As mentioned previously, given the scarcity of published literature on this topic, all published papers on this topic (in intensive care patients) have been reviewed. Shalit and Umansky (1977) were the first researchers to examine the effects of routine bedside procedures on ICP (turning (but not log-rolling) was one of these procedures) in 'comatose' adults with different ICP devices. In 7/11 patients every change in body position was accompanied by a considerable increase or decrease in ICP (without alteration of blood pressure).

Rising (1993) examined the effects of selected nursing activities on ICP in five brain injured adults. These results were presented descriptively case by case and no attempt at generalisations were made, which makes the results fairly weak. There was significant variability in the baseline ICP of the group before turning also (2 – 15.8mmHg). Although they showed an increase in ICP in all patients with turning, by two minutes post turn all patients ICP (although having higher than baseline ICPs) had recovered to near baseline levels and none were >20mmHg.

Boortz-Marx (1985) in a weak and poorly reported crossover study involving three adults and one child with head trauma, noted that turning

the patient (to the side rapidly) did result in an elevation of ICP, but no further results are presented. This study is limited by a very small sample size, with mixed pathology and different ICP devices, no baseline ICPs presented with no real results (even descriptive) presented.

Another fairly limited and poorly reported study by Muwaswes (1984) investigates the effects of passive range of motion (ROM) exercises and 4 turns (not log-rolling) on 12 adult neurosurgical patients with intraventricular catheters in situ. She found that ICP was elevated after the turns, but that recovery to baseline ICP after the turns varied between patients and related to the initial degree of ICP increase. Limitations of this study were that no demographic or confounding data were presented, no baseline ICP data were presented and no inferential data analysis was done.

Clearly evidence on this topic is limited and inconclusive and it is surprising that Hobdell et al (1989) produced contradictory results in the paediatric sample. However, this study had significant limitations, using children with a huge variety of pathology requiring ICP monitoring and different ICP measurement devices.

3.6 The effect of clustering nursing 'cares' together and planned rest periods on CBV parameters

There were only two specific studies on this topic; neither demonstrates any significance with planned rest periods. Bruya (1981) used a randomised controlled trial to study the effect of planned rest periods on the ICP of 20 adult intensive care patients with ICP monitoring. The patients were randomised to receive either control (no planned rest periods) or the treatment (planned 10 minute rest periods between nursing interventions/cares) of which one was suctioning. She found no statistically significant difference between the two groups. She did however find that one of the procedures that produced the greatest rises in ICP was suctioning. This study was however, underpowered and Bruya acknowledges that perhaps 10-minute rest intervals were too short to produce a difference.

A further study by Hugo (1987) on manipulating nursing care activities (introducing a 30 minute rest period prior to intervention) found no statistically significant difference between the two groups. Again it was acknowledged that perhaps the normal ward routine of 10-20 minutes rest pre-procedure may have been too similar to the treatment group (which received a 30 minutes rest). She did note however that when nursing procedures were grouped together the patients appeared to take longer to

recover to baseline values, this was just an observation and there were no results reported to confirm this.

Although no studies examining the manipulation of nursing cares have been able to demonstrate any significant findings, they can help future researchers in improving study designs in this area.

3.7 Effects of hygiene interventions on cerebrovascular (CBV) parameters

Only one study has specifically examined the effects of hygiene interventions (HI). Parsons et al (1985) studied the effect of various hygiene interventions (oral hygiene, body hygiene and urinary catheter care) on CBV parameters of 19 severe closed head injured adults. It was found all three interventions produced significant mean increases in all physiological parameters ($p<0.005$) when compared to baseline. However, all these elevations had retuned to baseline within 1 minute post intervention. It was concluded that hygiene interventions can be safely performed in this group of patients. Boortz-Marx (1985) also found that bathing increased the ICP (measured by ventricular fluid pressure), however as previously discussed this study is weak. It is evident that published data in this aspect of care is also very limited.

3.8 The physiological effects of endotracheal suctioning on intracranial pressure

There has been more research in relation to this aspect of care than any other, and so this section will be discussed under further sub themes.

Rudy et al (1991) examined the effect of endotracheal suctioning (ETS) in 30 adult head injured patients. They were the first to describe a pattern of response to ETS in three phases: firstly a rise in baseline ICP beginning with ETS and continuing throughout the whole ETS sequleae, secondly a spiking of the ICP during the suctioning part of the protocol and thirdly a combination of both rising baseline values and spiking. It was also found that 60% of patients had elevated ICP (defined as > 15mmHg) before the procedure and that with each suction there was a cumulative rise in the percentages of cases with increased ICP. Campbell (1991) confirmed the above findings and describes this 'stair-step' pattern of ICP waveform in response to hyperinflation and ETS, which increased with successive treatments, on nine adult intensive care patients.

These findings are supported by others. Crosby and Parsons (1992) in a quasi-experimental study in 49 adults with raised ICP examined the effect of ETS and manual hyperinflation on cerebrovascular parameters. They also found a significant change from baseline values during the procedure for mean arterial blood pressure, mean intracranial pressure, cerebral perfusion pressure and heart rate ($p<0.001$). There were significant

differences in all above variables at key points during the procedure, the highest being during the three suction catheter passes ($p<0.0025$). They also noted that ICP increased in a stepwise fashion, but could be reversed with prolonged hyperventilation. A further quasi-experimental study in 9 children (Fisher et al 1982) (aged between 9 months – 12 years) with raised ICP (from various pathology) found ICP rose by an average of 5mmHg during ETS compared to a non-ETS period, with no change in CPP.

More recently Brucia and Rudy (1996) studied the effect of suction catheter insertion alone in a quasi-experimental study of 30 ventilated adults with head injury. They found that suction catheter insertion and tracheal stimulation, isolated from other parts of the suctioning procedure significantly increased ICP, arterial pressure and cerebral perfusion pressure ($p<0.025$). During the application of negative pressure for actual suctioning, ICP and heart rate increased significantly, but arterial pressure and cerebral perfusion pressure did not. The most recent study by Billotta et al (2008) also found that in 41 adult severe head trauma patients ETS led to a significant rise in ICP >20mmHg ($p<0.05$).

In 1989 Hobdell et al conducted specific research into the effect of nursing cares/interventions (including ETS) on the ICP in children. Thirteen children (aged 1.5 – 11 years) with a broad range of intracranial

pathology, who had ICP being measured, were studied using a descriptive observational study. The children, who had different ICP measurement devices, were observed for 5 – 24 hours, during which the effect of 'nursing cares' were observed. With regard to ETS, the only finding reported is that mean ICP was greater at the time of hyperventilation than at 10 minutes post ($p<0.05$). It was also noted that there was no statistical significance between baseline ICP and recovery time.

In 1983, Snyder studied the effect of any nursing activities (including ETS) in nine comatose adults, using a descriptive observation study. They found that many nursing care/intervention activities occurred together so again it was not possible to isolate the effect of single care interventions. However they noted that 'respiratory care interventions' (it was not made clear exactly what these were) resulted in the highest mean rise in ICP. Boortz-Marx (1985) also found in her study of four head injured patients that suctioning produced the greatest rises in ICP. In contrast, Tsementzis et al (1982) in a study involving 39 adult and paediatric head injured patients who were sedated and muscle relaxed with a ventricular catheter in situ, showed that 49% patients had no alteration in ICP with suctioning and 36% showed only a minimal ICP increase, however of all the 'routine interventions' studied, suctioning produced the worst ICP response. This study however was performed in the mid 1970s, when

head injury management was fairly basic, there were no CT scanning facilities, and a few of the cases reported had fixed and dilated pupils.

The studies that specifically examined ETS in head injured patients (Rudy et al 1991; Crosby and Parsons 1992; Brucia and Rudy 1996) all demonstrated that ETS produced statistically significant elevations in ICP compared to baseline. An earlier researcher (Campbell 1991) described a classic 'stair step' pattern in response to ETS and manual hyperinflation, which increases with successive treatments, and this was confirmed by others (Rudy et al 1991; and Crosby and Parsons 1992).

3.9 The effect of coughing during endotracheal suctioning on the ICP response

An early study by Fisher et al (1982) involving nine children, found that two thirds of children coughed during ETS but their ICP rise was similar to the non-coughing children. These findings are contradicted by Gemma et al (2002) in a study of 17 head injured adults (with baseline ICPs 15-20mmHg). They noted that six patients (in 20 episodes) coughed or moved during ETS. Their ICP rise was significantly more pronounced than patients who were more sedated and did not cough ($p<0.0001$).

Similarly Kerr et al (1998) in a large experimental study of 71 head injured adults comparing different drug treatment with ETS found patients treated

with no sedative drugs displayed the classic 'stair step' ICP increase pattern with ETS. Whereas, those treated with opiates and neuromuscular blockade (no cough) showed a flat response to ETS, with little change in ICP, demonstrating the effectiveness of levels of sedation and paralysis on patients' responses to ETS. Two of the papers conclude that if patients cough during ETS then their ICP is more pronounced. Physiologically this makes sense as coughing increases intrathoracic pressure, which in turn impedes cerebral venous drainage. However it may be related to the higher level of sedation which prevents/minimises a cough and not the presence of a cough itself.

3.10 The effect of endotracheal suctioning on cerebral perfusion pressure

In terms of changes to CPP, most studies reviewed (Rudy et al 1991, Crosby and Parsons 1992; Brucia and Rudy 1996, Gemma et al 2002) report some significant peak CPP changes with ETS, however these are mixed with both reductions and increases in CPP in the same sample. Cerebral perfusion pressure either reduced as a result of increased ICP or increased as a result of increased mean arterial pressure (or a balance between the two variables). This appeared to relate to whether patients were able to generate an increase in mean arterial pressure in response to ETS. None of these studies report patients on inotropic support, yet in current paediatric practice more than 85% of children with severe

traumatic brain injury are inotrope supported (Tume and Baines 2008). Again most reported CPP values recovered rapidly, with virtually all recovered by five minutes (Rudy et al 1991, Crosby and Parsons 1992; Brucia and Rudy 1996, Gemma et al 2002). The effects on other cerebrovascular variables have also been described amongst the above studies.

Gemma and colleagues (2002) found that during ETS in inadequately sedated patients, the CPP and jugular venous oxygen saturation (Sjo2) showed a significant reduction from the well-sedated patients ($p < 0.0001$). Rudy et al (1991) also found arterial pressure had returned to baseline levels in all patients by five minutes. However, CPP fell to < 50mmHg in two patients, but in most others the CPP increased as a result of the increased MAP. No CPPs were back at baseline by five minutes but by 10 minutes all were at baseline values.

Crosby and Parsons (1992) found a significant change from baseline values during the procedure for all cerebrovascular parameters ($p < 0.001$). There were significant differences in all variables at key points during the procedure, the highest being during the three suction passes ($p<0.0025$). Both CPP and heart rate changed significantly in both groups ($p<0.0025$ and $p<0.0025$ respectively). They noted that patients in group two (who received an additional hyperinflation breath and had

higher resting ICPs) were unable to increase their arterial pressure by the same magnitude as group one, so their CPP was lower throughout the procedure.

3.11 The effect endotracheal suctioning on cerebral oxygenation

More detailed observations of cerebral oxygenation (via middle cerebral artery velocity using transcranial doppler (Vmca) and jugular venous oxygen tension ($Pjvo_2$)) have more recently been examined. Kerr et al (1999) examined this in ventilated 19 head injured adults. They found that cerebral oxygenation was maintained in those patients who had only transient elevations in ICP (68.4%). Gemma and colleagues (2002) found that during ETS in inadequately sedated patients, the CPP and jugular venous oxygen saturation (Sjo2) showed a significant reduction from the well-sedated patients ($p<0.0001$). However both these studies are limited by the measurement of cerebral oxygenation only on a global level. These measurements will not show any regional changes in cerebral oxygenation (just like CPP will not) (Carhuapoma and Williams 1999).

3.12 Effects of respiratory physiotherapy (RP) on cerebrovascular parameters

There are three studies that specifically examine respiratory physiotherapy (percussion, vibrations, endotracheal suctioning and manual hyperinflation) in patients with raised ICP. There is one adult

study and two mixed studies (with patients aged 5-67 years). These studies are relevant, because ETS and manual hyperinflation are key components of respiratory physiotherapy.

These two quasi-experimental studies (Garradd and Bullock,1986; Paratz and Burns 1993) both found statistically significant increases in ICP with physiotherapy treatment (duration 11-17 minutes) compared to baseline. However, Garrard and Bullock (1986) noted a significantly worse ICP rise in non-paralysed patients. In paralysed patients (receiving neuromuscular blockade) ICP also rose but appeared to be time dependent and by 14 minutes had reached the same level as the non-paralysed patients. Both studies found patients with higher resting baseline ICPs (>15mmHg) had more marked increases in ICP.

Ersson et al (1990) in a descriptive observational study of 12 patients compared the ETS phase to manual hyperinflation and expiratory vibes phase (termed bag squeezing) of respiratory physiotherapy. They found that ICP increased with both procedures to a similar level, but recovery time was more prolonged after ETS than manual hyperinflation.

3.13 The effect of hyperventilation during endotracheal suctioning

Campbell (1991) studied nine adults with severe closed head injury using a quasi-experimental design of three hyperinflation volumes and ETS. They found that the mean ICP did not change significantly from baseline

between three hyperventilation tidal volumes, however there was one limitation of this study, the very low mean baseline ICP of the sample (4.8mmHg). This is exceptionally low (normal) and does not reflect the reality of severe closed head injured patients seen in practice.

Rudy et al (1991) also examined different hyperventilation volumes during ETS. Both groups demonstrated significant changes from baseline ICP and there was no difference between the two tidal volume groups. They concluded that ETS causes a significant increase in ICP, mean arterial pressure and cerebral perfusion pressure in adults with severe head injury regardless of the tidal volume used for hyperventilation.

However Kerr et al (1997) who studied 66 severe head-injured adults in a randomised crossover study looking at the effect of short term hyperventilation during ETS found different results. They compared a 4 breath hyperoxygenation (control group) versus 8 breaths per minute hyperoxygenation and hyperventilation (HV) (experimental group 1) and the control group versus a 30 breath hyperoxygenation and hyperventilation (experimental group 2) during ETS on the patients' physiological parameters. The hyperventilation was done using another ventilator set at 135% above the patients' tidal volume and all were hyperoxygenated with 100% oxygen. They found significant differences between the 4 breath (control) and the 30 breath (hyperventilation) group

in terms of the ICP ($p<0.001$), arterial pressure ($p<0.002$), CPP ($p<0.002$) and carbon dioxide level ($p<0.000$). The ICP response was noted to be significantly less in the extreme hyperventilation group (30 breath group) during the suctioning phase, with the carbon dioxide being significantly lower in this group (absolute values not reported). There are some limitations of this study however; they did not report the presence of all known confounders in this group of patients (the sedation level, presence of NMB or barbiturates, EVDs, inotropic support, PEEP level or the PacO2 that the patients actually were hyperventilated down to) nor the mean baseline ICP which makes some of the results difficult to interpret. Additionally, although CPP was maintained during this study (implying that cerebral perfusion was maintained) this only reflects an overall global estimate of cerebral perfusion and regional hypoperfusion and ischaemia may have occurred with this extreme hyperventilation (which they acknowledge). The effects of arterial carbon dioxide (PaCo2) level on cerebral blood flow are well documented in traumatic brain injury (Adelson and Kochanek 1998; Reilly and Bullock 2005 p339) as are the adverse effects of prolonged hyperventilation on patient outcome (Muizelaar et al, 1991) with current recommendations being to only aggressively hyperventilate patients when there are signs of impending herniation (Adelson et al 2003).

3.14 Recovery times after endotracheal suctioning

It is evident that ETS does result in increased ICP, however if this is only transient, then harm may be minimal. The research reviewed shows contrasting results in relation to recovery times. Parsons and Ouzts Shogan (1984) found no significant ICP changes at one-minute post ETS compared to baseline. However, this is contradicted by Rudy et al (1991), who found 76% of patients had not regained their baseline ICP values by one minute, 42% had not by five minutes and 25% had not by nine minutes. Kerr et al (1998) also found prolonged recovery times in 20% of patients who had not recovered their baseline ICP by 15 minutes post ETS.

In 1992, Crosby and Parsons examined the cerebrovascular response of 49 closed head-injured adults to a standardised ETS and manual hyperinflation (MH) procedure. They sought to determine whether a five-minute rest period following the procedure was sufficient to allow cerebrovascular parameters (ICP, mean arterial pressure, cerebral perfusion pressure and heart rate) to return to baseline values. They noted that patients with higher baseline ICPs (defined as greater than 7.1 – 9.3mmHg) had higher ICPs in the recovery phase. However they did not find significant differences in ICP rises between the two baseline ICP groups, however given that in both groups, the ICP is also low and not markedly different between the groups, one would not expect them to react differently. They also found significant differences ($p<0.0025$) in

recovery time for all variables compared to baseline, but did not report this in terms of time to recovery of baseline values.

Hugo (1987) studied 23 severe head injured patients (adults and children 5-60 years) in relation to the manipulation of nursing care activities (of which ETS was one). In this experimental study, she found that there was no statistical significance between baseline ICP and recovery time. However, 20% of patients ICP had not returned to baseline in 15 minutes or the protocol was terminated as the patient required medical intervention to reduce ICP. Therefore with regard to recovery times there are conflicting findings in the research.

3.15 The effect of pharmacological agents on intracranial pressure changes induced by endotracheal suctioning

In the acute phase of severe head injury, children require sedation and analgesia to rest the brain and reduce sympathetic stimulation, in addition to providing analgesia, preventing distress to the child and ensuring invasive lines remain in situ. However, in most intensive care units additional intravenous bolus doses of these drugs or others are prescribed 'as required'. Thus, the intensive care nurse must use his/her clinical judgement to determine if any is required or which one/s should be given, at which times and if they should be given 'prophylactically' prior to interventions, to try to minimise the ICP rise with the intervention.

Therefore it is important for the nurse to be able to judge whether the effects of these drugs do in fact 'reduce the ICP response' to suctioning. There has been considerable research in this area into the effects of different pharmacological agents on ICP and CPP in relation to ETS.

White et al (1982) undertook a randomised study of drugs for preventing increases in ICP during ETS in 15 comatose intensive care patients. Only patients who developed raised ICP (>20mmHg) during or immediately after ETS were studied. At one to two minutes prior to ETS the patients received the following drugs in a randomised sequence: 2ml 0.9% saline (control), 1microgram/kg fentanyl (an opiate), 3mg/kg thiopental (a barbiturate), 1.5mg/kg lidocaine (a local anaesthetic) and 1mg/kg succinylcholine (a muscle relaxant) (all given intravenously) or 1.5mg/kg lidocaine given intratracheally (IT). The drugs were also given an arbitrary 'cough score' based on their ability to suppress a cough. The patients were divided into two groups: those with normal baseline ICPs who developed significantly increased ICP during ETS and those with elevated baseline ICPs. They found that fentanyl produced no acute changes in ICP but did not blunt the increase in ICP seen with ETS. thiopental (thiopentone) and lidocaine (intravenous) initially produced a 4-6mmHg decrease in ICP; but neither affected the ICP response to ETS. Intratracheal lidocaine was more effective ($p<0.05$) in blunting the ICP response from ETS and in suppressing a cough, however there was an

initial cough provoked when it was instilled. Succinylcholine was highly effective in abolishing the increase in ICP due to ETS ($p<0.01$). Mean arterial pressure (and hence CPP) was not significantly affected by any of the regimes because of the rise in mean arterial pressure with ETS.

Soon after this Yano et al (1986) specifically studied the effects of lidocaine on the ICP response to ETS in nine adults (16-71years) with severe head injuries. This quasi-experimental study used patients with mild intracranial hypertension (baseline ICP had decreased to $\leq$ 20mmHg from initial values of 25-40mmHg) to calculate the peak ICP rise with ETS associated with 1.5mg/kg intravenous lidocaine administered 1, 3, 5, 10 and 15 minutes before ETS. Six hours after this 2ml of 4% lidocaine was injected intratracheally and the same procedure was studied. They found that neither intravenous nor intratracheal lidocaine lowered the baseline ICP, however both treatments suppressed the ICP rise caused by ETS, but the peak ICP rise was significantly lower ($p<0.05$) after intratracheal lidocaine then intravenous lidocaine.

Donegan and Bedford (1980) were the first to examine the effect of intravenous lidocaine on ICP during ETS in 10 head injured ventilated adults. He found that intravenous lidocaine (coupled with manual hyperinflation) significantly reduced ICP, compared to placebo (before

suctioning), but that after ETS the rise in ICP was similar to the control group.

Most recently Bilotta et al (2008) studied the effects of intratracheally administered lidocaine in 41 head injured adults in intensive care. This quasi-experimental study was in non-muscle-relaxed, but propofol-sedated, stable adults (defined as ICP <20 for 24 hours). Patients were excluded if they had not previously demonstrated significant ICP (>20mmHg) rises with ETS, and they were only studied in the first three days after injury. The patients were routinely suctioned 10 minutes after instillation of lidocaine (starting at a dose of 2.0mg/kg and titrated according to the ICP). In 51% of patients (n=21) lidocaine effectively prevented the ETS-induced ICP rise (to < 20mmHg) and CPP remained unchanged. However in the remaining patients, ICP did rise significantly with ETS (by more than 30% $p<0.05$), although CPP remained unchanged. In 30% of adults (12/41) lidocaine instillation did produce a cough or patient movement, but no patients' ICP exceeded 20mmHg. They calculated an ED_{50} of 1.7 +/- 0.3mg/kg was determined as the dose of lidocaine required to prevent an ICP rise with endotracheal suction.

Gemma et al (2002) studied the intracranial effects of ETS in the acute phase of head injury in 17 adults. This prospective randomised trial involved sedated and ventilated (but not paralysed) adults during the first

week of their head trauma. They analysed 131 episodes of routine ETS. They found six patients during 20 episodes coughed or moved during ETS, these patients were then labelled as 'inadequate sedation' and considered separately. They found that ETS increased ICP and this increase was more pronounced when sedation was inadequate ($p<0.0001$).

Kerr et al (1998) specifically studied the effect of neuromuscular blockade and opiates on the cerebrovascular response to ETS in 71 severe head injured adults using a quasi-experimental design (no randomisation). These patients were divided into three groups: Group one: no drugs, Group two: opiates only and Group three: opiates + neuromuscular blockade. It was noted that before the implementation, the group receiving opiates and neuromuscular blockade had significantly higher baseline ICPs and APACHE II scores (an adult risk of mortality score, so they were sicker patients) ($p<0.0002$) but this was controlled for in the analysis. There were three different ICP catheters used in this study, and the baseline ICP values did differ according to the type of device used ($p<0.01$), with ICPs being highest in the intraventricular catheter group. They found that ICP increased significantly as a result of ETS ($p<0.001$) and ICP continued to increase with each catheter pass (as described previously). Patients treated with no drugs displayed the classic stair-step increase in ICP. This response, however, was not observed in the

patients treated with opiates and neuromuscular blockade. These patients had a flat response across the ETS procedure, indicating little change in ICP during ETS.

Rudy et al (1991) found that patients requiring barbiturates (three patients received thiopentone) had significantly lower ICP values throughout the ETS procedure and they stated it appeared to blunt the effects of ETS.

Werba et al (1993) studied the effects of ETS (on ICP) before and after neuromuscular blockade with vecuronium and atracurium in 18 ventilated neurosurgical adults. This randomised study found that patients without neuromuscular blockade (even with good sedation) showed significant increases in their ICP and reductions in cerebral perfusion pressure (associated with ETS and coughing), compared to the paralysed group ($p<0.05$). Brown et al (1996) examined in a randomised blind crossover trial, the effect of suxamethonium compared to placebo given before respiratory physiotherapy on the ICP response in 11 head injured adults. They found suxamethonium had no significant effects on ICP compared to placebo (saline) at baseline but they did not report the response to suctioning in either group.

In 1993 Hanowell et al (1993) studied the effect of alfentanil (an opiate) given prior to ETS on the ICP and CPP of six head injured adults. This

quasi-experimental study compared the administration of saline, 15 and 30 microgram/kg of alfentanil prior to ETS. No difference was found between the two alfentanil doses so these were analysed together. They found that alfentanil reduced the CPP significantly ($p<0.05$) compared to saline. They noted there appeared to be greater elevations in ICP during ETS with the alfentanil groups but this did not reach statistical significance.

More recently Leone et al (2004) examined the effect of remifentanil (a very short acting opiate) on ETS induced ICP rises in 20 ventilated head injured adults. This quasi-experimental study evaluated three ascending doses of remifentanil (bolus then infusion) with ETS beginning 20 minutes after infusion commencement. They measured heart rate, ICP, mean arterial pressure, cerebral perfusion pressure, middle cerebral artery flow velocity (Vmca) (which is reflection of cerebral blood flow), coughing and bispectral index (BIS a physiological measure of sedation) throughout the study period. They found that with increasing remifentanil doses a higher proportion of patients required vasopressor support, and this was significant ($p<0.001$) between patients receiving dose one (low dose) to dose three (highest). Coughing was reduced as dose increased, with 75% of patients in the high dose group not demonstrating a cough with ETS. There were significant changes in heart rate, mean arterial pressure, cerebral perfusion pressure and bispectral index with increasing

dose of the bolus drug, but no significant change in middle cerebral artery velocity (that is, cerebral blood flow) was observed. However, no significant change from baseline was noted during ETS for the above variables and no significantly different effects amongst the three drug doses. ICP however, increased significantly from baseline in all groups. They claim that remifentanil (in this group of patients) suppresses the cough reflex in a dose dependent manner, however there are clear and very pronounced effects on blood pressure with increasing dose, which require vasopressor support in order to avoid significant reductions in CPP.

Therefore there is some evidence that certain drugs may minimise the ICP rise with ETS (muscle-relaxants, intratracheal lignocaine and perhaps barbiturates). However, the side effects of barbiturates are considerable, so their use in clinical use in paediatric intensive care units is not routine (Tume and Baines 2008). The current clinical use of prophylactic intratracheal lignocaine is also rare in practice (Tume and Baines 2008). However, the use of muscle-relaxants in severe brain injured children is far more common in practice (Tume and Baines 2008); although there are again side-effects associated with their use, with some papers suggesting their use should not be routine practice (Hsiang et al 1994). These are all important considerations for the intensive care nurse to bear in mind,

when deciding whether to use a bolus dose of sedation/analgesia or muscle relaxant prophylactically prior to ETS.

3.16 Summary of papers excluded from the literature review

The findings of two of the excluded papers which examined the effects of nursing cares on ICP (excluded because they were both in adult ward patients) also showed some evidence that nursing cares (particularly repositioning and suctioning-related procedures) increased the ICP (Mitchell and Mauss 1978; Mitchell et al 1981). The two studies in pre-term infants (Ninan et al 1986; Durand et al 1989) also showed that suctioning produced a significant increase in ICP; with Durand et al (1989) demonstrating that this ICP response reduced with sedation.

There were two excluded studies on respiratory physiotherapy (RP) (Imle et al 1997; Olson et al 2007). Olson presented a single case study (Olson et al 2007) which concluded that further research was required. Imle et al (1997) examined RP with a head down and head flat position. Positioning these patients anything other than slightly head up would appear potentially very hazardous and is not accepted PICU practice, so this paper was excluded because of this. One study of suctioning in PICU was excluded (Singh et al) even though one secondary outcome measure was ICP (measured in four children with a variety of different pathology). However their primary aim of this study was to examine the influence of ETT size and suction pressure on physiologic variables. Their only

conclusion that relates to this literature review is that suctioning (using a pressure of 100mmHg) does increase ICP (6-12% above the percentage for the patients mean ICP) and similar trends were seen at the two other suction pressures of 80mmHg and 120mmHg.

The review papers (Mitchell 1986; Rudy et al 1986; Andrus 1991; Brucia et al 1992; Chudley 1994; Wainwright and Gould 1996; Hall 1997; Simmons 1997; Chamberlain 1998; Johnson 1999; Palmer 2000; McLean 2001; Mestecky 2007) concluded similar findings to this review, but most fail to quantify the exact effects of nursing interventions and there is a paucity of evidence in relation to children with severe TBI.

3.17 Summary of the literature review

From this literature review it still remains inconclusive and unknown what the effects of routine intensive care nursing interventions have on ICP and CPP in severe head injured children. Even in adults (very few of the papers are in children) there are conflicting results with regard to recovery times after turning and suctioning and even whether there are significant changes in the CBV parameters (Hobdell et al 1989, Jones 1995, Parsons and Wilson 1984, Parsons and Ouzts Shoga 1984; Rudy el al 1991, Kerr et al 1998, Hugo 1987). This is evidenced by some studies finding significant ICP changes (from baseline) with interventions and others not. Neither study that examined the effect of 'manipulating the

time between nursing activities' could demonstrate any difference (Hugo 1987, Bruya 1981). Some of the inconclusiveness in the literature may be due to the lack of account for strong confounding variables impacting on ICP (the effect of sedation level, neuromuscular blockade or the use of barbiturates) or CPP (the effect of inotropes to augment this). Additionally, some samples of patients with raised ICP (not all were head injuries) were severely ill (with high baseline ICPs) and others not, which will impact on the patients response and also on medical therapies utilised. Without this data presented or accounted for in the results it makes comparability or validity of the results difficult to interpret.

Overall, there were considerable weaknesses in most of these studies in terms of: inconsistent ICP device measurement (which has been shown to affect the primary outcome measure ICP), mixed sample of intracranial pathology, mixed adults and children in the analysis, very small sample sizes, weak research designs or lack of rigour in the research, lack of account or acknowledgement of known confounding variables, procedures studied are not described in enough detail to allow replication, or the baseline ICP of the samples does not reflect the current reality of children in PICU after TBI. This literature review has demonstrated there is a clear need to control for the above variables and study the effect of nursing interventions separately on the ICP and CPP specifically in children post TBI.

CHAPTER 4

SURVEY OF CURRENT UK PRACTICE

4.1 Introduction

After the initial literature review was conducted, a national survey of UK paediatric intensive care unit practice was undertaken to establish what nursing and medical practices were actually being undertaken. This was felt to be important to actually relate the literature and proposed research to current practice and to be able to 'put it into context' when discussing results and practice implications. Only the results of the PICU nursing practices are presented below in relation to this thesis, but the full published paper can be seen in Appendix Two.

4.2 Methods

There are 21 paediatric intensive care units that manage severe head injured children in the UK, excluding one centre, which still manages these children in the adult neurointensive care unit (Paediatric Intensive Care Audit Network data (PICANET)). The Paediatric Intensive Care Society (PICS) was founded in 1987 as a multidisciplinary forum for those involved and interested in paediatric intensive care (PIC) (http://www.ukpics.org/). It has an educational subgroup (PICS-E). These groups involve representatives from most UK PICUs. Electronic questionnaires were sent out to 17 UK PICUs (who had PICS-E contacts) via this group in June – July 2006.

The questionnaire asked specifically about their management of children with severe traumatic brain injury in the PICU (see Appendix Three). PICU clinical nurse educators completed the questionnaire and were asked to send their TBI unit guidelines back with the questionnaire. Non-responding units were followed up with one reminder (given a month to respond) and in two units consultant contact was made when nursing contact failed. Two UK units (who admitted severe TBI children) had no PICU educator contacts. The local research committee and the PICS study group (TBI subgroup) clearly identified this review as audit.

4.3 Results

The overall response rate was 76% (13/17) with 10 (59%) for the questionnaires and 11(63%) for the guidelines. Only 8 (47%) units completed both the questionnaire and sent a copy of unit guidelines. There did not appear to be any geographical variations in patterns of practice across the UK. However there did appear to be variations in PICUs that existed within adult hospitals in that many adult related TBI practices were more prevalent eg the aggressive use of deep vein thrombosis prophylaxis.

Nursing and physiotherapy management

Three units (23%) had specific nursing guidelines in place (two used care plans). Most guidelines reviewed primarily described the medical management. For endotracheal suctioning (ETS) practices, one unit (8%) had specific respiratory physiotherapy guidelines for this in place. Seven

(54%) units stated these children were suctioned only 'as required', but in eight (62%) there was no mention of suctioning precautions in their guidelines, with six (46%) stating 'consider bolus sedation' prior to ETS. On questioning there was a large variability in the drugs used prophylactically prior to ETS (fentanyl (Intravenous (IV) /endotracheal (ET)), pancuronium, alfentanil, propofol, midazolam, thiopentone, vecuronium, morphine or ketamine). Three units (23%) reported using lignocaine in relation to suctioning (two used it IV the other unit ET). One unit stated lignocaine could be used intravenously after endotracheal suctioning (ETS) if required.

For managing raised ICP following suctioning, optimising sedation (using a bolus) was the most common practice (92% (12) units). Eleven (85%) units reported using brief periods of hyperventilation to reduce ICP after ETS. As well as this other therapies cited were: cerebrospinal fluid (CSF) drainage (31% (4) units) if an external ventricular drain (EVD) was present, a thiopentone bolus (46% (6) units) was used if the first line measures were unsuccessful or the child was already on thiopentone. One unit used mannitol and a frusemide Bolus. Other interventions reported included general measures of increasing the degree of head elevation (2 units 15%), loosening the cervical collar (5 units 38%) and ensuring a quiet environment (1 unit).

For essential nursing cares (washing/eye care/mouth care etc) most responses were vague. Two units (15%) stated these children were managed with a 'minimal handling approach' (implying that these cares were all clustered in a group, then the child was left to recover for generally around 6-8 hours). Two units (15%) stated each child was assessed individually with regard to 'care tolerance' and two units recommended that cares be 'spaced out' and not clustered. Four units (31%) discussed nursing these children in a 'quiet and calm environment', with reduced environmental stimuli (ie a cubicle).

Eleven units (85%) reported removing or loosening the rigid cervical collar if the child was muscle relaxed (while ensuring tapes/sandbags were present), but these were replaced prior to turning or before cessation of muscle relaxants. All units reported these children were only ever moved by log-rolling (never straight lifted).

In terms of the frequency of turning these children, there was considerable variation from 3 hourly (one unit 8%), 4-6 hourly (two units 15%), 6 - 12 hourly (two units 15%), 12 hourly (two units 15%), once a day (one unit 8%), and 'assessed individually' (one unit 8%). One unit stated these children were minimally disturbed for first 2-3 days (eg linen not even changed).

With regard to deep vein thrombosis (DVT) prophylaxis, 6 units (46%) specified the use of thromboembolic deterrent (TED) stockings alone if able, 2 units (15%) used TEDS and subcutaneous heparin, 1 unit used a

compression system or TEDS and 1 unit used a combination of a boot compression system and TEDS or subcutaneous heparin.

4.4 Discussion

This audit has demonstrated wide variations in the nursing care practices in critically ill child with TBI. The limited evidence base for nursing and respiratory physiotherapy management practices presented in the previous chapter has almost certainly led to these huge variations in practice.

However, the effect of environmental stimuli (including auditory stimuli, family visits, parental and carer touch and both familiar and unfamiliar voices) has been studied (Treolar et al 1991; Hendrickson 1987; Schinner et al 1995 and Mitchell and Haberman1999). These studies involving family presence found no change in ICP or even a slight reduction with family presence/visits. One study involving parental and nurse touch in children, found no effects on ICP (Mitchell and Haberman1999). More importantly the one study examining the effect of environmental stimuli (no noise (earplugs), music or standard ICU environmental noise showed no differences between these two environments on ICP (Schinner et al 1995). Despite this, many PICUs still advocate that these children should be managed in a "quiet and calm" environment, which means they are nursed in a cubicle as opposed to the open PICU area. It appears that this evidence has not been translated into practice.

Evidence based nursing guidelines for paediatric TBI management are not possible, with only weak Class II and some Class III evidence. There are no studies fulfilling the criteria for Class I evidence. Perhaps the first step for PICU nurses is to develop a national consensus based nursing management guidelines for TBI. At consensus the important research questions could be formulated and then tested collaboratively to generate the evidence through multi-centre trials. It is evident that the development of strong 'evidence based' ICU nursing management for Paediatric TBI has a considerable way to go.

4.5 Limitations of the audit

There are some limitations of this audit in that not all UK units were contacted (two had no educator contacts). A further limitation is that the clinical nurse educators completed the audit. They were chosen as they should be in touch with what is happening in their unit, but there is potentially some bias as some had contributed to the guidelines. However copies of their guidelines were also requested so the questionnaire responses could be compared to their guidelines.

4.6 Conclusions

This audit has established a reasonable baseline of current practice within the UK (July 2006). There are clearly still wide variations in PICU practice

across the UK in both medical and nursing practices (despite the publication of the 2003 Guidelines). With these wide variations, it is unlikely that all children are receiving the best care. The first step would be to achieve consensus on how care should be delivered, and if consensus can be achieved, standard care can be used as the basis for prospective trials. If consensus can not be achieved, then standardisation of different strategies may allow their comparison which may progress to a formal randomised trial. Another approach would be to establish a national database (similar to the US Coma Bank), which would allow detailed comparison of factors specific to neurotrauma. The availability of detailed data would allow more specific research questions to be generated.

CHAPTER 5

METHODS

5.1 Introduction

The literature review has highlighted the paucity of research in this field exclusively in children with moderate to severe closed head injury, thus the need for a study in this patient population. This chapter will outline the detailed methodology for this study including the specific research questions and aims, the data collection, analysis, validity and reliability issues and the ethical issues involved in conducting research in critically ill children.

5.2 Aims, Objectives and Research Questions

The broad aim of this research is to investigate the physiological effects of routine intensive care nursing interventions of: endotracheal suctioning with manual hand ventilation, turning the child (by a log-rolling method), eye care, mouth care and washing, on the ICP and CPP of moderate to severe closed head injured children in the intensive care unit.

A secondary aim is to investigate the effect of confounders on the ICP response during these selected nursing interventions.

Specific research questions:

In children with moderate to severe head injury in intensive care,

- What are the effects of these routine nursing interventions on the ICP and CPP?
- Which interventions produce the greatest rises in ICP?

- In the hyper acute phase of head injury what proportion of the intervention episodes results in peak ICP values that will exceed a) 20mmHg b) 30 mmHg and c) >30mmHg
- Does the maximal ICP, ICP rise or the recovery time (in minutes) correlate with the child's baseline ICP?
- How long do these children take to recover their baseline ICP values?
- Are there any variables (confounders) that affect the child's ICP response to the interventions?
- What are the effects of routine cares in children following decompressive craniectomy?

5.3 Setting

This study was undertaken in a large 23 bed paediatric intensive care unit in the North West of England. Alder Hey Children's NHS Foundation trust is a specialist children's hospital and the PICU receives in excess of 1000 admissions a year in all diagnostic groups including specialist cardiac surgery, burns and neurosurgery of children from 0 – 17 years of age (and occasionally older). This tertiary referral PICU is one of largest PICUs in Western Europe. It has 156 whole time equivalent (WTE) nursing staff, a nurse consultant, a lecturer practitioner, four advanced nurse practitioners, a nurse education team of five, seven WTE intensive care consultants, three specialist physiotherapists and a multitude of other support staff.

Neurosurgical cover is provided 24 hours a day by five consultant paediatric neurosurgeons and 16 neurosurgical registrars (who cover both this unit and a specialist neurosurgical centre nearby).

The unit sees around 40-50 head injured patients admitted a year, of which only around 10-15 are moderate to severe; these are managed by both the intensive care and neurosurgical team in a combined approach. The nurse to patient ratio of all intubated and ventilated children is 1:1. Nearly 60% of the intensive care nursing staff have a specialist intensive care nursing qualification.

5.4 Study Design

A prospective observational cohort study was undertaken in two phases, a pilot study was conducted over the first twelve months and the second phase which incorporated the pilot study patients took place over the next two years. In total, children were studied over a three year period: January 2006 to January 2009. It was predicted that a three year study would yield a sample size of 24 children based on current admissions.

5.5 Ethical Approval

Ethical approval was firstly obtained from Liverpool John Moore's University Ethics committee. Further ethical approval was then sought from the Alder Hey Children's NHS Foundation Trust, but as this study was observational and did not involve any extra interventions or treatment beyond routine care,

it was pragmatically considered as audit by the NHS Trust (Reference No. 619 – Appendix Four). It was thus registered as an audit, with the trust and followed the principles of sound data collection and storage, but in the trust's view did not require parental consent (Code of Good Practice for Clinical Audit, 2006 RLCH).

However because of the conflicting views of the two organisations, and the researcher's unease with the trust's views' and potential problems with publication, a decision was made to gain parental consent for the detailed data collection (beyond that required for normal care) (See Appendix Five and Six for parent information sheet and consent form). Principles of sound research governance were followed throughout the three year study. A more detailed discussion of the ethical issues involved is at the end of this chapter.

5.6 Description and definition of nursing interventions studied

The nursing interventions that were studied were:

- Endotracheal suctioning with manual ventilation breaths ('bagging')
- Eye care
- Mouth care
- Turning the child (using a log-rolling technique to protect the cervical spine)
- Washing the child

These were all non-urgent (or non-emergency situations) and a detailed description of each is now provided.

5.7 Endotracheal suctioning and manual ventilation

Endotracheal suctioning (ETS) is an essential procedure for all intubated patients to clear airway secretions and prevent tube obstruction (Morrow and Argent 2008). The specific procedure examined in this study was the combination of suctioning and manual hand ventilation (as this was by far the most common procedure undertaken in these children). Suctioning can be undertaken alone (without manual ventilation) and also in combination with respiratory physiotherapy treatments (eg percussion, vibrations etc) but in the acute phase of head injury (first 72 hours) this was rarely done and it was considered important to keep this procedure as consistent as possible. There are guidelines for endotracheal suctioning in head-injured children (see Appendix Seven) in this PICU, although the procedure was not rigidly controlled. The procedures studied were non-urgent endotracheal suction (with manual ventilation) in the first 36 hours of the child's PICU admission.

5.8 The Suctioning Procedure

The suctioning procedure involved pre-oxygenation with 100% oxygen for 1-2 minutes prior to suction (on the ventilator), careful consideration of the need for prophylactic sedation and/or the use of endotracheal lignocaine (ET) (which if used was instilled 2 minutes prior to starting suctioning). ETS

was performed by a standard open, clean technique, with a suction catheter size (French Gauge) no greater than twice the size of the endotracheal tube diameter (eg for a size 3.0mm ETT a size 6 Fr suction catheter would be used), size passed down to no more than one centimetre past the length of the ETT with a suction pressure of between 12–20 kpa (dependent on secretion tenacity). Suction was applied only on withdrawal of the catheter and the duration of a suction pass did not exceed 30 seconds. Patients were always supine with a 10-20 degree head up tilt. A second nurse performed manual ventilation with an appropriate sized bag for the child's size, with a general procedure to slightly exceed the ventilator- delivered number of breaths, to aim for good (but not excessive) chest movement and maintain physiological parameters as consistent as possible (Spo2, MAP). The nurses (or a nurse and physiotherapist) undertook the procedure as quickly as possible (with as few suction passes as possible) and observed the child's 'tolerance' to the procedure. End-tidal carbon dioxide (EtCo2) was continually monitored in these children and any rise in this, coupled with a reducing minute volume or audible secretions on auscultation (or any coughing) would indicate the need to perform this intervention. The procedure of manually ventilating the child was not controlled for and the extent of hyperventilation (delivering a larger tidal volume and/or a higher rate) is difficult to quantify. A drop in carbon dioxide (and a rise in Ph) is a potent cerebral vessel vasoconstrictor, even in children with severe injuries, resulting in reduced cerebral blood flow and consequently reduced ICP.

Carbon dioxide level cannot be reliably measured during manual ventilation with current EtCo2 measurement devices, as ventilation is inconsistent. However we did try and account for the effect of reduction in PaCo2 during manual ventilation by recording the EtCo2 measured within 30 seconds of being connected back to the ventilator.

5.9 The turning (via log-rolling) procedure

Turning and repositioning children in intensive care is essential to prevent skin breakdown and the development of pressure areas (Curley et al 2003). However because of the nature of these children's injury (and hence a potentially unknown and unstable cervical and other spine) these children cannot be nursed on a pressure relieving mattress (they are too soft) nor can they be positioned in any way other than supine, with their neck in the midline position (in a rigid cervical collar) and their head slightly elevated. Their risk for skin breakdown is thus considerably higher than the average child in PICU. In the researcher's ICU an arbitrary time of 12 hourly is advocated for log-rolling in these severely ill children. This was felt to minimise the risk of intracranial instability and still prevent skin breakdown, but there is no evidence base to this practice as there is no published papers on the optimal turning frequency in these children. Because of the child's potentially unstable spine the only safe turning procedure advocated is the Log-Roll manoeuvre (Mackway-Jones et al 2005).

This technique involves the use of a minimum number of four people to turn the child, whilst keeping the head and spine in complete alignment during

the turn. Then following the x-ray or the back wash (or any other reason the child was turned) the child is then turned back to the same position he/she started in. During the turn the child's rigid cervical collar must be fastened and a designated person controls the head, neck and shoulders of the patient. When transported to other locations in the hospital (eg operating theatre or CT scan) these children are also turned in this way and immobilised on a rigid spinal board, the same procedure occurs for a portable x-ray in the PICU. All of these procedures which have been classified as 'turning' are used (but any emergency CT scans or urgent operating theatre trips excluded) to avoid any unstable patients in these episodes and so reduce bias.

5.10 The eye care procedure

This intervention involves cleaning the eyes and eye lid area (with sterile water) and the application of Lacri-Lube ® (Allergen UK) ocular lubricant to protect the corneas from ulceration and/or the application of Geliperm ® (Geislich Pharma UK) transparent hydrogel dressings for the same purpose. In children who are heavily sedated and/or muscle relaxed (hence have no blink reflex) this is important to minimise the risk of corneal abrasions.

5.11 The mouth care procedure

Good oral care in critically ill children is essential to reduce bacterial contamination in the oral cavity (which increases the risk of nosocomial

pneumonia) and to prevent tooth and gum disease. Mouth care is also performed regularly and involves cleaning the oral cavity and lips and the application of Vaseline or lip balm to the lips to keep them moist. These children are always intubated orally, so the performance of teeth cleaning is made more difficult, but the oral cavity is cleaned with water and some attempt to clean the teeth is made.

5.12 The washing procedure

Good skin hygiene is also important in the critically ill child both from an aesthetic perspective and to minimise the risk of skin breakdown. The child is washed at least once every 12 hour nursing shift and after any bowel movements. This is done with the child lying supine (i.e. not moved) with Baby Bath solution used to wash the child all over especially around the urinary catheter and groin area.

5.13 Time frame for measurements

For all interventions the nurse was asked to record all the baseline values (one minute before the intervention), the maximal changes (up or down) in the prescribed parameters during the intervention. Once the intervention was completed, a set of measurements was recorded five minutes after the procedure was completed on a specific data collection form (see Appendix Eight).

These interventions were performed at various times by the bedside nurse. The interventions were all performed separately (spaced at least one hour apart) to ensure that their effects can be accurately described in relation to the individual nursing intervention and there was no crossover of effects from one intervention the next intervention (a fault with previous research). This is, however, an observational study, and although there are unit guidelines for the performance of some of procedures (to standardise the performance and consistency), they were not performed in a controlled manner and nursing practice was studied in 'real life'.

5.14 Recruitment of patients

All parents/carers of children who were admitted to PICU over the study period who met the inclusion criteria with a moderate to severe head injury were approached. Once a child was admitted to PICU with a head injury and had an ICP monitor placed, the nursing team notified the researcher and eligibility for study participation was discussed. If the child met the inclusion criteria, the researcher (or another specifically trained (in the study and in the process of consent) PICU nurse) came to PICU and approached the parents or guardians, informed them of the study, and gave them an information leaflet to read. This was on average around two to twelve hours after the child's PICU admission. All parents who consented to the study gave virtual immediate consent, (however they were given up to 24 hours to

consent) (given the observational nature of the study this is not surprising) and written consent was taken (Appendix Six).

Further discussion of the ethical issues involved in gaining consent in these circumstances is undertaken in the ethical issues section at the end of this chapter.

5.15 The main study sample

All consecutive children (aged 2 – 17 years) admitted to the researcher's PICU with moderate to severe closed head injury, who had invasive ICP monitoring over a three year period were recruited.

Inclusion criteria:

- All Aged 2 – 17 years who were admitted with a moderate to severe closed head injury who required invasive intraparenchymal ICP monitoring

Exclusion criteria:

- Children < 2 years age (due the risk of still having open fontanelles)
- Children with suspected non-accidental injury (as the mechanisms, pathophysiology and prognosis for this type of head trauma) are quite different to children with accidental head trauma.
- Children with an open/penetrating head injury (where the dura was breached or they had multiple skull fractures)

- After decompressive craniectomy (a surgical procedure to remove part of the skull to allow an oedematous brain to swell), these children will form a sub group of this study.
- Any child with devastating injury (not expected to survive)
- Children whose parents did not consent to the study or for whom no guardians were available to give consent.

These exclusion criteria were critical as they would add to the heterogeneity of the sample. It was felt important that the sample be kept as homogenous as possible as moderate to severe closed head injuries, to minimise bias, accepting that this would reduce the sample size.

5.16 The decompressive craniectomy sample (sub group)

- The same inclusion criteria apply but these children were studied following a decompressive craniectomy.

Three of the children who were initially recruited into the main study crossed over into the craniectomy group after the first measurement episode was taken, and consequently are analysed in both groups. This was considered reasonable because after this procedure, they have different physiology and are a 'different' child physiologically. As there were only five children in this group, their results will be presented as a case series.

5.17 Defining severe head injury in this study

In terms of classifying the severity of head injury, Glasgow Coma Score has traditionally been used (Mansfield 2007) with a severe injury defined as a GCS less than nine. The reliability of the Glasgow Coma Score (especially in young children by non paediatric health professionals) is known to be inconsistent (Gill et al 2004; Rowley and Fielding 1991; Marion and Carlier 1994). The National Institute of Clinical Excellence (NICE 2007) guidelines for the management of head injury in children and adults state that there are two indications for invasive ICP monitoring: a GCS less than 9 and/or an abnormal initial CT scan. In practice we do see severe injuries (sometimes resulting in death) with initially higher GCS scores, so despite the traditional definition of a 'severe head injury' the author felt justified in including any child who had a severe enough injury to warrant invasive ICP monitoring.

5.18 Data Collection

A specific data collection tool (see Appendix Eight) was developed by the researcher to capture the required data per 'intervention episode'. The bedside nurse completed a new form for every 'intervention' episode undertaken, where possible, recording the physiological effects. The data collection form takes into account all reported confounding variables.

Intracranial pressure was measured with a Codman Microsensor ^{TM} (Johnson and Johnson UK) in all children, inserted intraparenchymally into

the non-dominant hemisphere, which was zeroed at the time of insertion. Arterial pressure (and a derived continuously displayed CPP), end tidal carbon dioxide and other variables were all monitored on a Phillips IntelliVue MP70 monitor (Koninklijke Philips Electronics N.V) using Phillips monitoring lines. These were calibrated to atmospheric pressure 12 hourly and re-levelled as required to the level of child's right atrium. The accuracy of the end-tidal carbon dioxide measurement (EtCo2) was checked and correlated against the arterial blood gas value with every sample and is reported to be relatively accurate (correlated with PaCo2) during consistent ventilation (i.e. heavily sedated or paralysed patients) which these children were (Kerr et al 1996).

The time to recovery (in minutes) to the child's baseline ICP, and any interventions required to reduce the ICP were also recorded. The Phillips bedside monitors keep a 48 hour history of patient data. The researcher or data collector can go back and check for accuracy. The researcher randomly checked this in five children to check the accuracy of the data collected, and accuracy was confirmed. The children were studied for the duration of the time their ICP was monitored (range 1–10 days) although in the final study only the initial measurement episode was used for analysis. The median duration of invasive ICP monitoring in this study was 2.5 days and only eight children had their ICP monitored for longer than 3.5 days.

Concerns have been raised over the accuracy of these fibre optic ICP measurement devices beyond the first 5-7 days (Poca et al 2002) as these devices are zeroed to atmospheric pressure at the time of insertion only and cannot ever be re-zeroed, hence the theoretical risk of a slight drift from zero over time. However there is recent evidence (Koskinen and Olivecrona 2005; Al-Tamimi et al 2009) that found although this drift occurs, it is generally less than 2mmHg. Utilising the first measurement episode minimised the risk of any ICP value inaccuracies due to this drift.

The primary outcome measures (and main outcome of interest) in this study are:

- The change from mean baseline ICP to peak ICP (and ICP rise) during the intervention and to post procedure ICP (recorded at five minutes after the intervention finished)
- The minutes to recovery to baseline ICP values

Clinically, the maximal ICP recorded during the intervention is probably the most significant outcome, however whether the baseline ICP effects this is also important to know. Recovery times are also important to know, both time to return to baseline and also to < 20mmHg after the intervention.

The time of five minutes post procedure was chosen as this represents the clinically significant interval at which increased ICP would be tolerated post intervention, after which aggressive measures are required to reduce the ICP to less than 20mmHg (Adelson et al 2003). The post procedure

measurement was clearly defined for the data recorders as five minutes after the termination of the procedure i.e. reconnection back to the ventilator or completion of the turn.

5.19 Phase One - The Pilot Study

The pilot study recruited eight children over the first twelve months. The aims of this were to test the data collection forms, refine the methodology and to determine the consent rate (Arnold et al 2009).

Data were collected over the duration of time the child's ICP was measured (up to 11 days), but because of the impact of time from injury influencing ICP, only measurement episodes undertaken in the first three days (the hyper acute phase) were analysed (this constituted 71% of the measurement episodes see Table 3). These repeated measurement episodes were pooled together to generate one mean value per patient for analysis.

Results of the pilot study

There were 136 'nursing intervention' episodes in 8 children. A summary of the results is now presented. The demographics of the pilot study sample can be seen below.

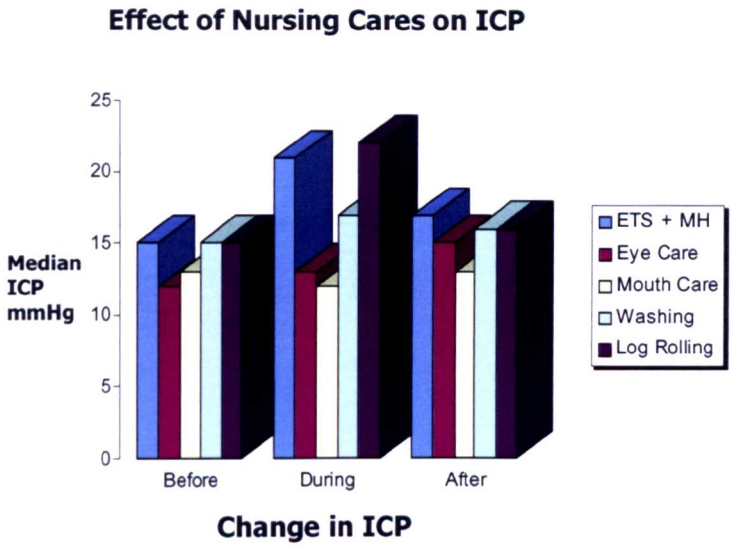
Table 2: Baseline demographics of pilot study patients (n=8)

Median Age (range)	11.5 years (6.5-15)
Median Baseline GCS	6 (3 – 12)
Median Day 1 ICP	18mmHg (11-36)
Median Day 1 CPP	69mmHg (57-74) (85% sample on inotropes)
Duration of ICP monitoring	Median 3.8 (mean 4.5) days
Actual mortality	1/8

Table 3: Measurements taken in the pilot study (n=136)

Nursing Interventions	**No. recorded (and % of total measurements)**
ETSMV	87 measurements (64%)
Log-rolling	20 measurements (15%)
Eye Care	17 measurements (12%)
Mouth Care	10 measurements (7%)
Washing	2 measurements (1%)
Day of Injury that interventions studied	Day 1 15% Day 2 41% Day 3 15% Day 4 10% Day 5 9% Day 6 5% Day 7,8,9 4%
Baseline ICP prior to interventions	≦10mmHg 21% 10 – 15mmHg 35% 15 – 20mmHg 32% >20mmHg 13%

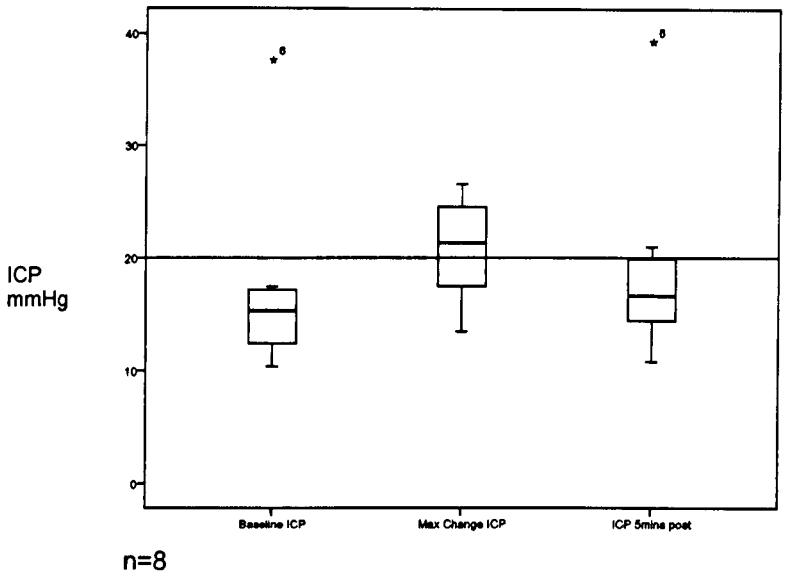
It became clear that of all the nursing interventions observed in the pilot study, two (endotracheal suctioning and turning) were significantly worse than the other interventions, producing higher median ICP rises than the other interventions. This can be seen on Graph 1.



Graph 1: Comparison of the effect of nursing cares in the pilot study

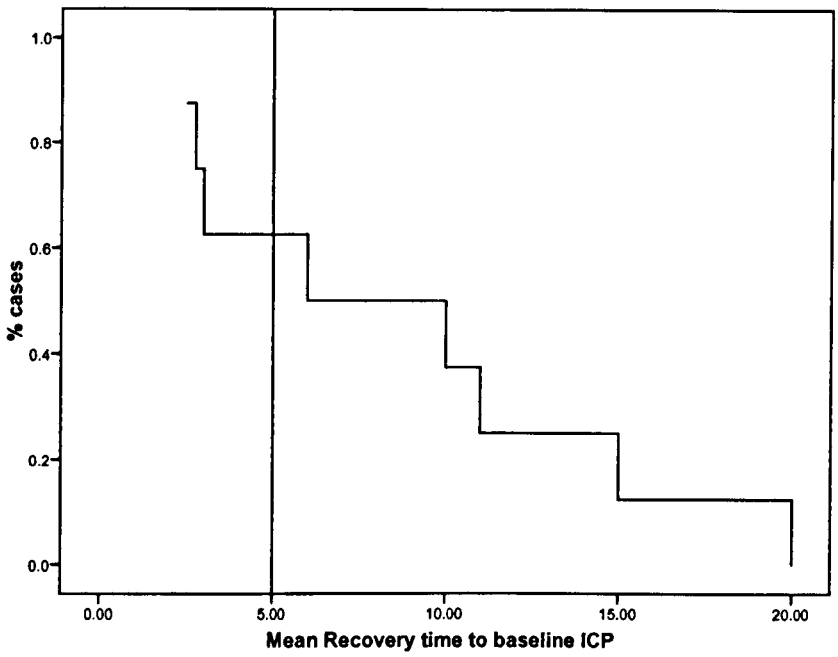
The effect of endotracheal suctioning in the pilot study

When the effect of ETSMV was examined in further detail it was evident that the median ICP for most patients exceeded the clinically significant 20mmHg level.



Graph 2: The effect of endotracheal suctioning and manual ventilation (ETSMV)

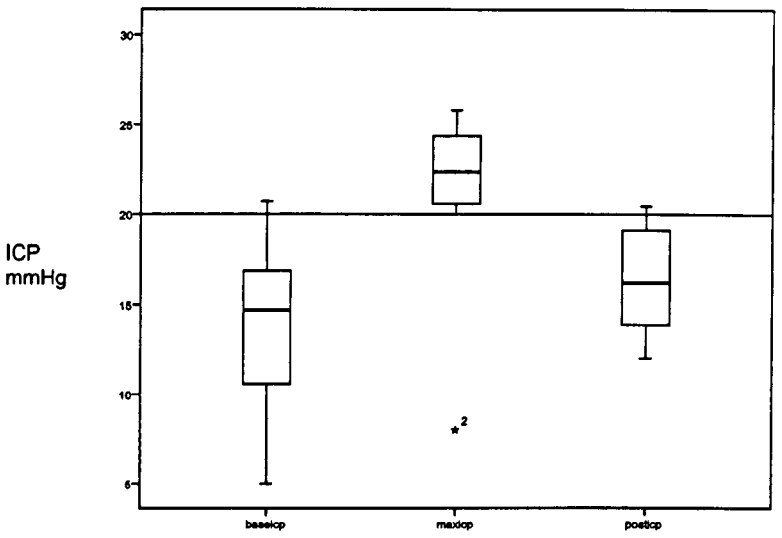
The overall 5-minute post procedure median ICP looked acceptable, but when presented in more detail (in the Kaplan Meier curves – Graph 3) it was evident that patients exhibited large variations in their recovery time after this intervention. The overall median percentage change from baseline during ETSMV was +49.5% (mean 47.5%) and in 63% of these, the patient's mean ICP value exceeded 20mmHg. After ETSMV, in 33% of the suctioning episodes (6 patients) patients took >10minutes to recover their baseline values. In these episodes 71% had a higher (>15mmHg) baseline ICP prior to the intervention



Graph 3: Recovery time to baseline ICP after ETSMV (pilot study)

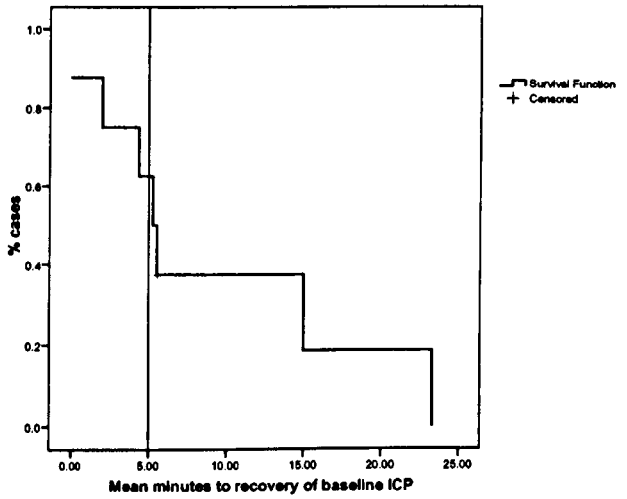
The effect of turning (Log-rolling) in the pilot study

There were 20 turning episodes in 8 children in the acute phase (Day 1 – Day 3) of moderate to severe TBI. Overall this intervention resulted in a mean increase of +54% (50% median) in ICP from baseline (for all patients).



Graph 4: The effect of log-rolling in the pilot study

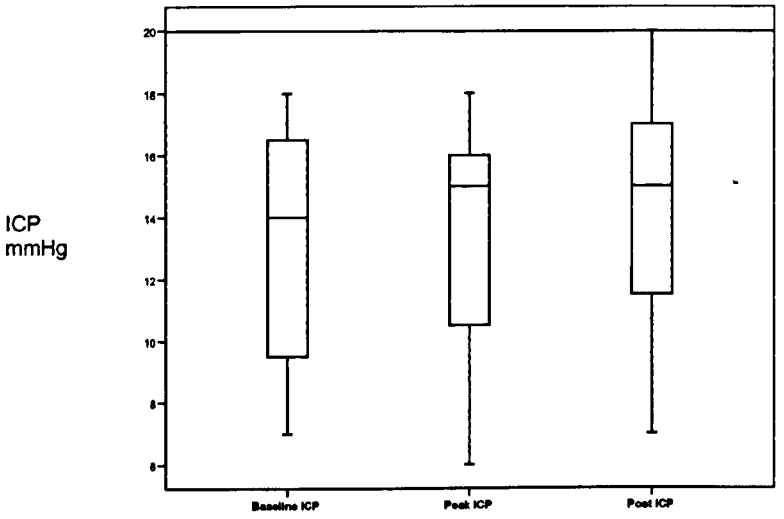
Turning also led to a prolonged recovery time in a number of children.



Graph 5: Mean recovery times after log-rolling

The effects of eye care (pilot study)

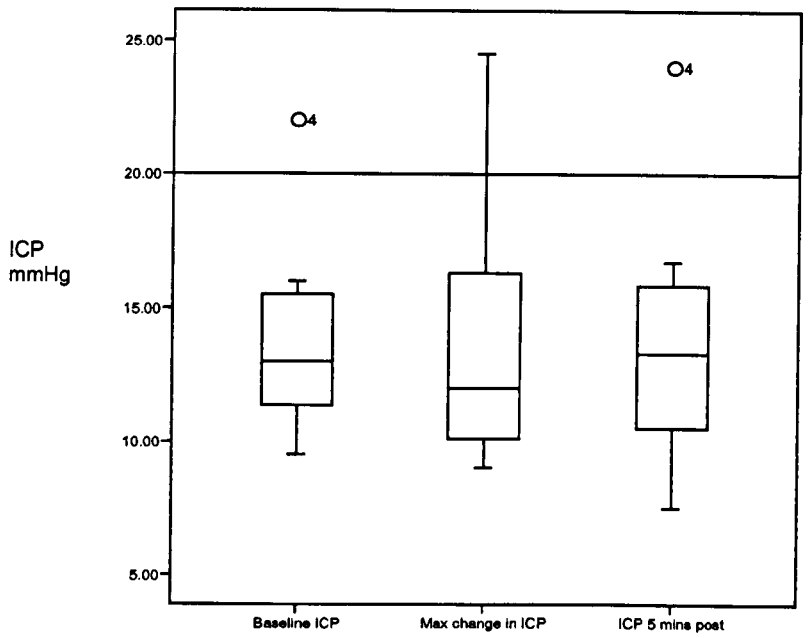
There were 17 eye care episodes for analysis in seven children, which demonstrated little change in ICP from baseline and no clinical significant ICP rises above 20mmHg.



Graph 6: The effect of eye care in the pilot study

The effects of mouth care (pilot study)

There were 10 mouth care episodes for analysis in seven children, which similarly to eye care revealed very little change in ICP and no clinically significant changes in ICP (one patient started with an ICP over 20mmHg but there was little change from baseline with the procedure).



Graph 7: The effect of mouth care in the pilot study

The effect of washing (pilot study)

There were too few washing episodes for analysis in the first three days of head injury (only 2 episodes in 2 patients), but in these, washing appeared to have minimal effect on ICP.

5.20 Refining the methodology

Presentation of this pilot work at two international and one national conference in 2007 (with international experts in the field present) as well as to the PICU team (nurses, intensive care physicians, anaesthetists, physiotherapists, neurologists and neurosurgical team) allowed some scrutiny of the pilot work, and modifications were made to the data collection

tool and data collection process. The observations and changes made to the study following the pilot study and based on expert feedback were to:

- Add in the COMFORT score to the data collection tool to try and quantify sedation level in these children, however this still presented problems as there remains no validated sedation tool for muscle-relaxed children and 86% of our sample were both sedated and muscle relaxed.
- Add an EtCo2 measurement when the child was connected back to the ventilator after suctioning to the data collection tool to attempt to quantify how aggressive MV had been. This is because we were aware of how much a reduction in $PaCo_2$ (through manual ventilation) might reduce ICP (Kerr et al 1997, Adelson and Kochanek 1998) and we thought that we might be able to quantify the effect of this on ICP changes during suctioning. As we did this after the study had started only 14 children had this data recorded. In 6/14 children (42%) the EtCo2 was lower than that measured at baseline prior to manual ventilation (an average of 3.6mmHg lower), but there appeared to be no association between the changes in this level and a drop in ICP.

- Examine the six month neuropsychological outcome of this group of children. The five point Glasgow Outcome Score (GOS) was used to classify the children as mild, moderate disability, severe disability, persistent vegetative state or death (Jennett and Bond 1975; Reilly

and Bullock 2005 p441). This was assessed by medical case note review six months post-injury by the researcher. Even though this is not a primary outcome measure of study, prominent researchers in the field of neurointensive care felt this to be important to be able to examine the basic outcome of these children in relation to the physiological responses seen during the PICU period.

- The pilot study showed the parental consent rate was high and undertaking the rest of this study was feasible
- The pilot study demonstrated the flaws in the initially proposed pooled mean analysis of the patient data, necessitating further statistical advice and input and discussion to agree to analyse only one measurement episode per patient.

5.21 Reliability and Validity

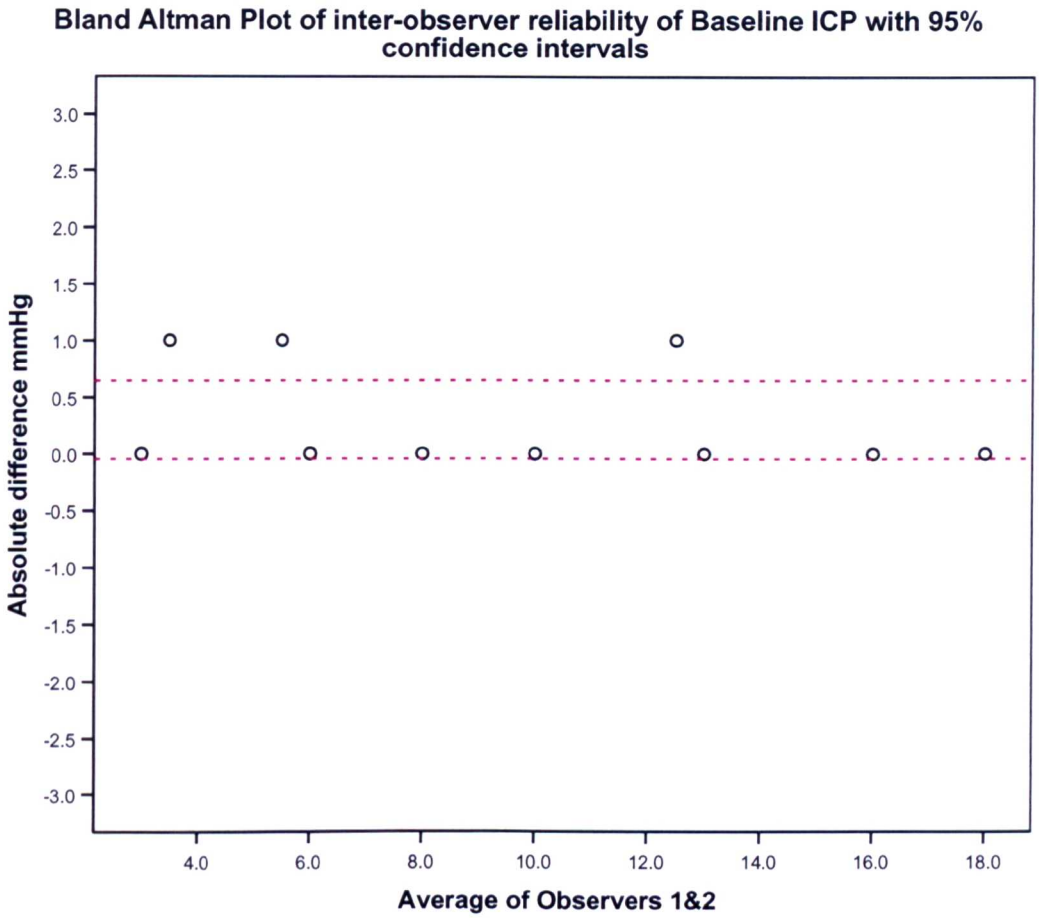
Internal validity refers to the degree to which one can say that the findings resulted from the intervention rather than some other factor (Winders Davis 2004a). In an observational study however, no causal relationships can be made, so threats to the internal validity relate mainly to the data collection tool and process. In observational studies the reliability and validity of the observation depends on the data collection tool and the ability of the observer/data recorder to accurately record the specific observations required (Parahoo 1997).

Having multiple data collectors is a threat to the internal validity, but testing the inter-observer reliability on ten occasions aids the researcher to feel more confident with the validity of this process. To improve validity of the data, the researcher trained initially and repeated this at regular intervals throughout the three year period (during mandatory training and on induction) the whole PICU nursing team (~ 156 staff) about the research and the data collection process to minimise any inaccuracies regarding this data. To account for the accuracy of multiple observers, inter-observer reliability testing was performed on ten occasions (Bland and Altman 1986). This was done as recommended on Bland-Altman plots with 95% confidence intervals (Bland and Altman 1986, Bland and Altman 1999; Haber and Barnhart 2008) and also by calculating the correlation coefficient between the two observers. McCluskey and Lalkhen (2007) claim that Bland-Altman plots are a superior method to use when comparing two different methods (or observers) of measuring the same variable, compared to the correlation coefficient, which may be misleading. The Bland-Altman plots showed good inter-observer reliability of the primary outcome measure ICP (see Graphs 8 – 12). The poorest variables were MAP and CPP, but these numbers change with every heart beat and are much more variable numbers so a small degree in their change is less significant. The correlation coefficients (r value) between observers for ICP and MAP are as follows: baseline ICP $r = 0.994$, maximal ICP $r = 0.997$, post ICP $r = 0.997$, baseline MAP $r = 0.804$, maximal MAP $r = 0.880$ and post MAP $r = 0.991$.

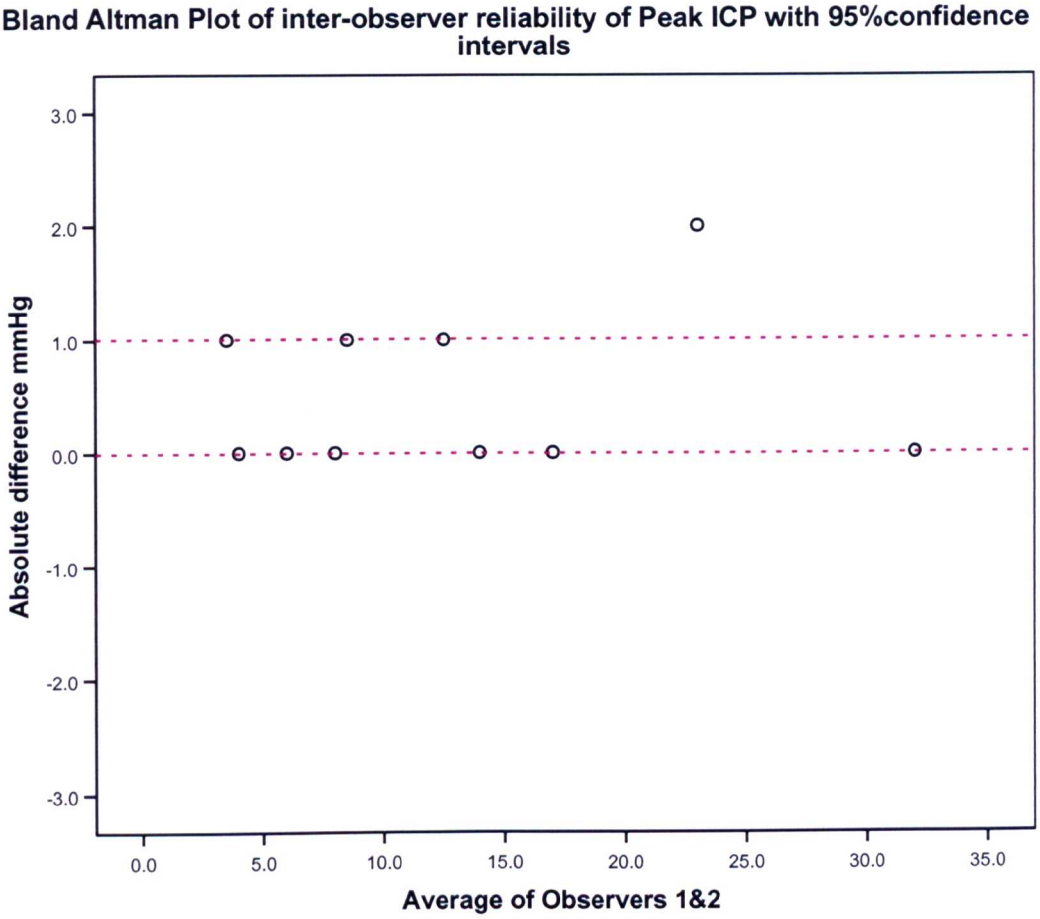
This shows a very strong correlation between observers and confirms the findings as shown on the Bland Altman plots, that inter-observer reliability is good for the three primary outcome measures of ICP (baseline, maximal and post).

This inter-observer reliability testing involved all five nursing interventions and there did not appear to be any difference in the accuracy of recordings between different interventions. As stated previously inter-reliability testing was done on ten occasions, of the total number of measurements recorded in the study (n=372) this accounted for around 3% of the measurements.

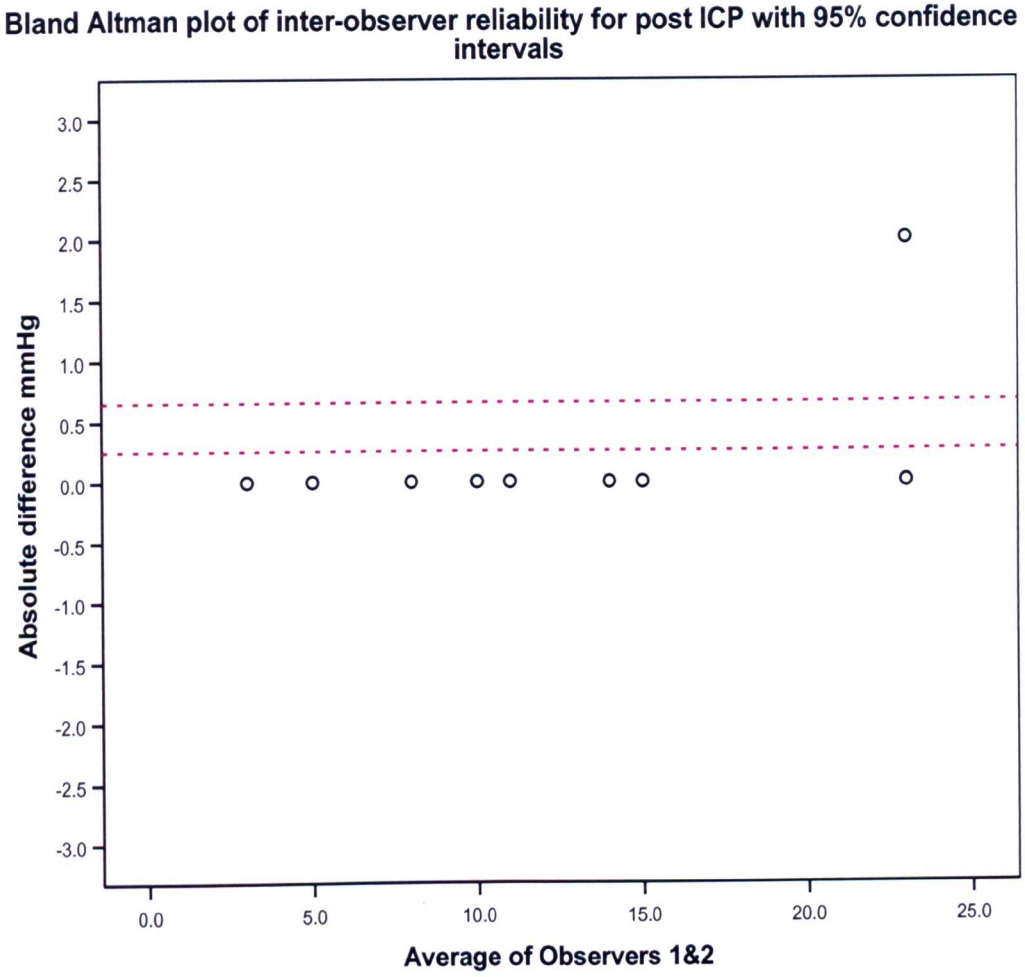
Furthermore it became clear that MAP or CPP would not be useful outcome measures as the majority (86%) of these children received inotropic support (nor-adrenaline) to artificially augment their CPP. This markedly reduces the usefulness of CPP as an outcome measure, as it is altered by titrating the nor-adrenaline dose, (when required by the bedside nurse) to achieve a desired age-dependent CPP target (based on unit guidelines). Thus this is not the child's 'true value' in response to the intervention (see Appendix nine for PICU Guidelines).



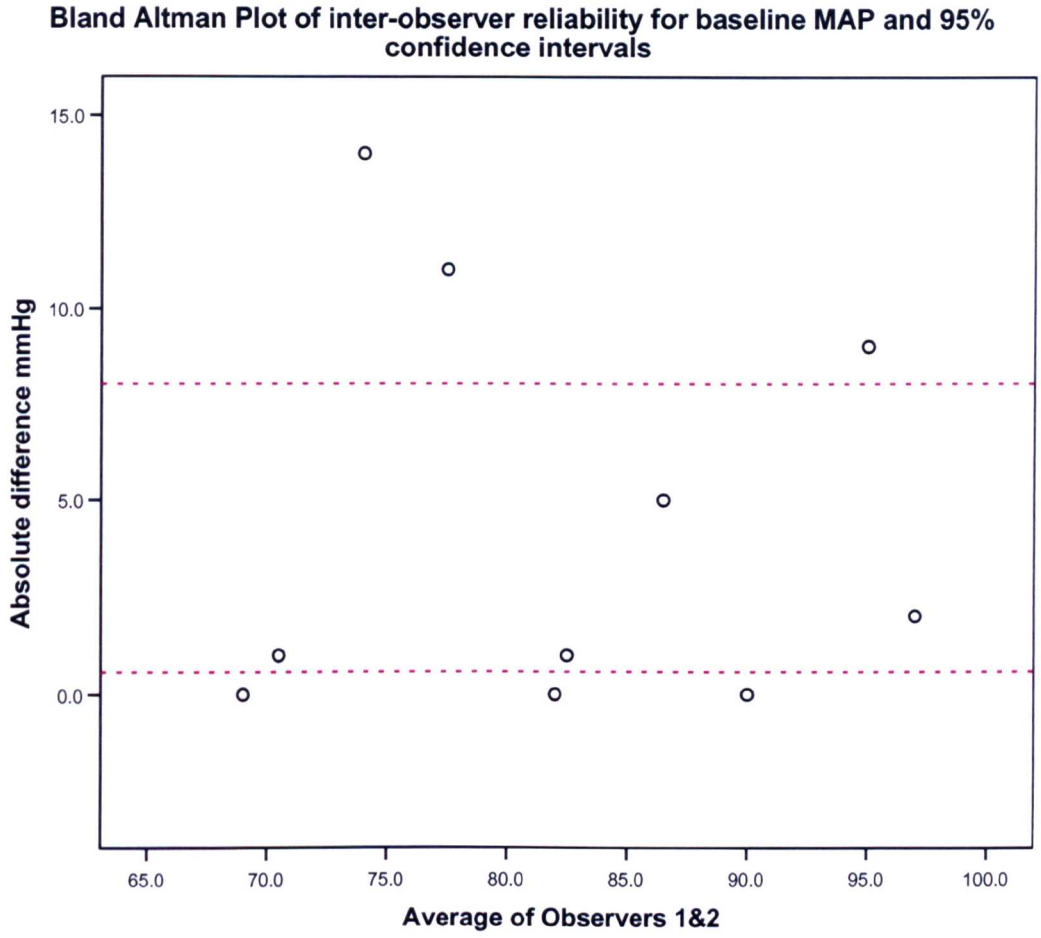
Graph 8: Outcome measure: baseline ICP inter-observer reliability



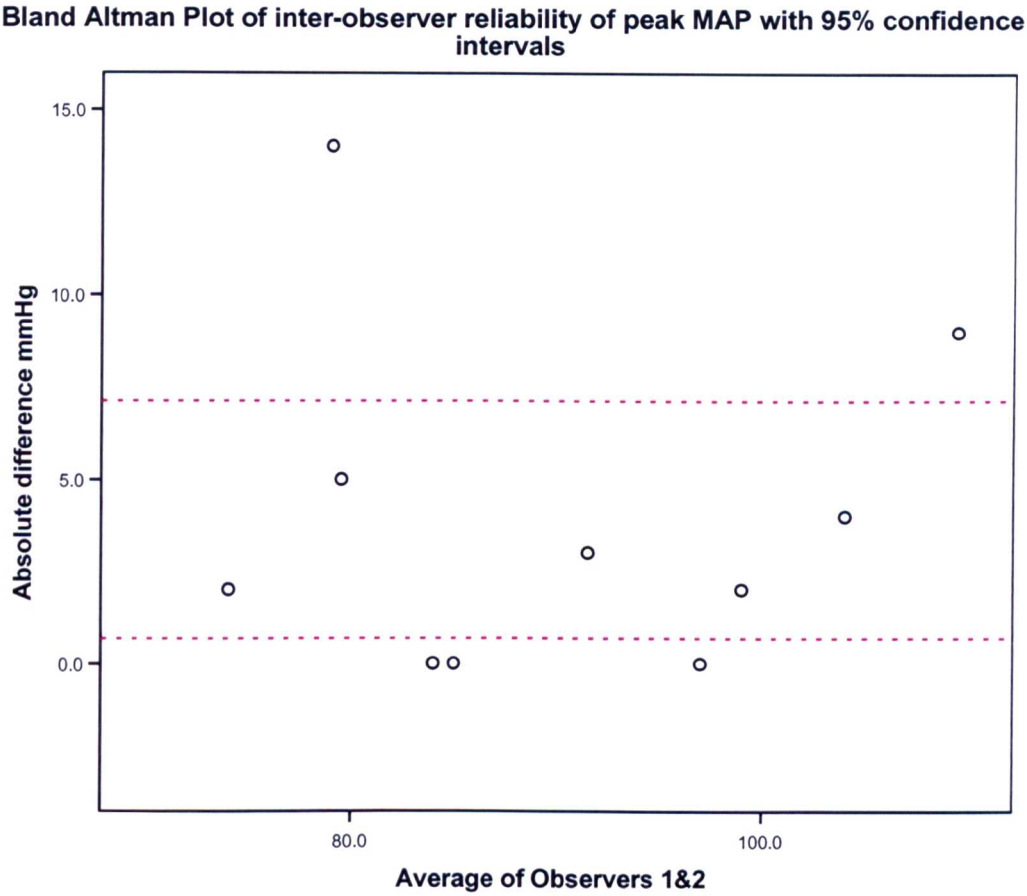
Graph 9: Outcome measure: maximal (Peak) ICP inter-observer reliability



Graph 10: Outcome measure: post-procedure ICP inter-observer reliability



Graph 11: Outcome measure: baseline MAP inter-observer reliability



Graph 12: Outcome measure: maximal MAP inter-observer reliability

It is acknowledged that there are limitations when using bedside nurses as the primary data collectors because of the possibility of distractions, clinical urgency (eg sudden patient deterioration), and reduced concentration over time (at the end of a 12 hour shift or on night duty when fatigue levels increase). However given that no funding was available to provide

additional non-clinical observers, it was felt that the best way to optimise data collection was with instituting a high level of training, continual reminders and ongoing training of observers. Where possible the researcher undertook the data collection herself, but this was not always possible and only occurred in around five children.

In terms of staff motivation to collect this data, on this PICU there is a small but highly committed team of nurses who enjoy nursing these children and do this regularly. In addition the researcher works on this unit and if not present, comes in (to gain parental consent) when these children are admitted thereby ensuring that the bedside nurse is aware of the study and the data collection process and that all the required forms and instructions are present at the child's bedside. If not present, then the researcher phoned the unit each shift to ensure the bedside nurse was clear about the data collection process.

As previously mentioned, the accuracy of the data collected were verified against the patient's observation chart and can also be checked for accuracy on the patient monitor (which keeps a 48-hour history of the child's data) to ensure validity, which the researcher did regularly to ensure accuracy. Any problems with reliability and validity noted in the pilot study were addressed to optimize methodological rigor. The inter-observer

reliability testing yielded good results for the main outcome measures of ICP (see Bland Altman plots with 95% confidence intervals).

5.22 Validity of the data collection tool

After the literature review and identification of confounders a data collection tool was developed (see Appendix Eight). The data collection tool was piloted with three different nurses initially to ascertain any problems with clarity of the tool and improve accuracy in the data collection process and establish face validity of the tool (does the tool actually measure what the researcher thinks it does). Following this, some changes were made to the tool. These changes were in terms of adding extra data collection items (regarding the confounding variables) and slight rewording issues to make it clearer what the researcher expected. There were also guidelines developed to be kept in the study folder (at the bedside) to improve consistency in this data collection process (see Appendix ten).

There are no other data collection tools that the researcher was aware of that could be used to collect this type of data, which led to the development of a new tool, but as stated previously this was done following literature review and discussion with senior research-experienced colleagues to ensure some content validity of the tool.

External validity refers to the degree to which the findings can be applied to other settings and populations (Winders Davis 2004b). In this study it was felt to be important to keep the sample group as homogenous as possible eg moderate to severe closed head injured children, accepting that this would lead to a smaller sample size. Previous work has used a broad sample of children with raised ICP (from many causes) or head injured children (but some not very severe) and adult and children in the same sample. These are significant problems with previous work, because the sample was so broad it threatened the validity of these studies findings. Therefore in this study to improve the general consistency and reproducibility of the findings, a very specific patient group was chosen.

5.23 Potential confounders

In this study a confounder is defined as any explanatory variable that has been reported to be related to the primary outcome variable of ICP or CPP. The confounding variables which are identified in the literature are:

- The baseline carbon dioxide level (EtCo2) and oxygen saturation level (SpO2) (Lodi et al 1998; Bhardwaj et al 2004 p8)
- The degree of elevation of the head of the bed (HOB) (Feldman et al 1992; Fan 2004; Ng et al 2004; Rosner and Coley 1986; Schultz-Stubner and Thieux 2006)

- The effect of thiopentone (a barbiturate) or propofol (Cook and Weant 2007; Dereeper et al 2002; Eisenbery et al 1988; Kelly et al 1999; Pittman et al 1989; Russo et al 1997)
- The effect of sedation and the agent used (Kerr et al 1998; Adelson et al 2003, Nadal et al 1998; Bourgoin et al 2003: Leone et al 2004; McClelland et al 1995)
- The effect of neuromuscular blockade (muscle relaxation) and whether the child was actually paralysed (i.e. did they cough or move) (Kerr et al 1998; White 1982; Werba 1993)
- The effect of positive inotropes (usually nor-adrenaline) which increase blood pressure to achieve a target cerebral perfusion pressure (Bhardwaj et al 2004 p230)
- The effect of lignocaine hydrochloride (1%) instilled down the endotracheal tube before suctioning (Brucia et al 1992; Yano et al 1986; Bilotta et al 2008; Donegan and Bedford 1980; Hamil et al 1991)
- The core temperature at time of procedure (McIlvoy 2007; Adelson et al 2005; Shafi and Mariscalo 2006; Tokutomi et al 2003).
- The presence of a semi-rigid cervical collar (which is fastened up) (Ahmad 2001; Craig and Nielsen 1991; Davies et al 1996; Ho et al 2002; Kolb et al 1999; Porter and Allison 1999)
- The level of Positive End Expiratory Pressure (PEEP) in cm of water (Georgiadis et al 2001; Huynh et al 2002; Mascia et al 2005; Muench et al 2005)

- The effect of mannitol or hypertonic saline (Weant and Cook 2008; Knapp 2005; Khanna et al 2000; Qureshi et al 1998; Sorani and Manley 2008) administered in the previous four hours.
- Time from injury – the peak incidence of cerebral oedema is known to occur in the first 72 hours (hyper acute phase) after injury (Adelson and Kochanek 1998; Reilly and Bullock 2005 p.86; Stocchetti et al 2007)

Further discussion of the effects of these confounders are presented in chapter one of the thesis.

5.24 Data Analysis

Data collected were anonymised (patients were allocated a study number) and entered onto a secure password protected SPSS version 15 database (on the researcher's personal laptop) by the researcher. This was carefully checked for accuracy and errors in data transcription. An additional anonymised Excel database was also established (on a password protected trust computer) of all head injured children that were admitted to the PICU, so that the study patients could be put 'into context' of the whole group. This database was registered with the NHS Trust as per NHS Research Governance Guidelines (Data Protection Act 1998).

Because this was an observational study one major issue in the data analysis is that there were different numbers of interventions and

measurement episodes in each child. The median time of intracranial pressure monitoring in the sample was 2.5 days (range 1.5 - 10 days) and there was a variable number of measurements done in each child over the first three days. It is well recognised that the first 72 hours of head injury represent a hyper acute phase of head injury, where brain oedema is likely to be at its worst (Reilly and Bullock 2005 p.86; Stocchetti et al 2007). Therefore to reduce the effect of one significant confounder, only measurement episodes undertaken in the first three days after injury were initially used for analysis (see discussion of pilot study).

The measurements taken over this period were initially averaged (in the pilot study), reducing noise in the patient data and resulting in a single mean value per patient. This ensures each patient case in this study is treated as an independent observation, which is a fundamental assumption in statistical modelling (Frison and Pocock 1992). However from a clinical perspective it became clear this averaged value was meaningless, as the individual measurement episode was decoupled from all the known confounders (eg core temperature at time of intervention, sedation bolus, and presence of muscle relaxation) and from the response during that particular intervention. Upon discussion with senior clinicians, experienced researchers and supervisors it was decided to use only the first measurement episode of each intervention per patient, so that bias would be reduced and only one measurement episode (with its confounders)

would be generated per patient. These measurements were all in the first 36 hours of PICU admission (and injury). This set of measurements is more specific to clinical conditions at the time and still satisfies the statistical independence assumption, albeit at the cost of increased variability due to the confounders. Repeated measures on the same patient can in principle be used for two different purposes: one is to reduce the errors in making parameter estimates on the effects observed. The difficulty here is to combine measurements with different periodicity for different patients. The second purpose is to use time series analysis to determine lag effects. To do this the observations would have had to be structured over suitably defined time periods and an appropriate protocol followed. This was not set in place for this study because the question of study was focused on the change in ICP from baseline to maximal and maximal to post ICP and the ICP rise and not on the dynamics of the process.

Prior to any detailed analysis the data were explored descriptively on SPSS to ascertain whether it was normally distributed or not and to get 'a feel' for the data. Peat and Barton (2005) claim that when analysing small samples (eg <30) that the researcher must ensure the data are almost perfectly normally distributed to be able to use parametric tests or any other analysis methods that assume a normal distribution. If this is not the case the data must either be transformed or a non-parametric tests used. This data transformation may involve the identification of and removal of extreme

outliers which may be skewing the data (particularly in small samples such as this one) (see further discussion of this in statistical methods).

5.25 Stratification of patients

The patients were also stratified according to their baseline ICP (<15mmHg and ≥15mmHg pre-intervention) to test the hypothesis that children with a higher baseline ICP would exhibit a higher peak ICPs and may exhibit longer recovery times. The stratification value of 15mmHg was chosen firstly because this was close to the median baseline ICP value in the group and secondly as other researchers had used this cut off value (Garrard and Bullock 1986).

The outcome measures of MAP and CPP were found to be not useful and do not form part of the main thesis question. CPP is a derived value (MAP-ICP) and will change if either primary variable changes. Sustained reductions in CPP (below cited age-dependent values) have been shown to produce a negative effect on outcome (Mazzola and Adelson 2002; Marmarou et al 1991; Carter et al 2008; Vik et al 2008). However virtually all (86%) of children in this study had their MAP augmented (and highly controlled) by the continuous infusion of nor-adrenaline (a potent catecholamine) which was manipulated continually by the bedside nurse to ensure the age-dependent CPP targets were met. Therefore, although the

MAP data are presented, it is not felt to be a useful outcome measure related to the interventions.

5.26 Statistical analysis

To compare the means of a paired sample eg the change from baseline ICP to peak ICP and peak ICP to post ICP, a paired t test was used (when the data were normally distributed). We chose to use the paired t test because this test directly addressed the research question and we did not collect data in a manner to enable us to do analysis of variance (ANOVA). The percentage change from baseline (in ICP) was also calculated and then the analysis of covariance (ANCOVA) was used to correct for baseline ICP (Vickers 2001) for endotracheal suctioning and turning.

When examining the effects of confounders eg the use of ET administered lignocaine, an independent t test was used (as these groups are independent) again with the assumption that the continuous variable of peak ICP was normally distributed. In this study a p value of <0.05 was considered significant and two-tailed tests were always used.

Correlation analysis was used to determine the strength of the relationship between two continuous variables (that had a linear relationship on scatter plot) (Harris and Taylor 2004 p48; McCluskey and Lalkhen 2007). In normally distributed variables Pearson's correlation coefficient (r) was calculated in this dataset and p values derived from the r value are also

presented. In variables where a significant relationship was demonstrated, linear regression analysis was used to calculate a straight line for the equation and generate a predictive model (McCluskey and Lalkhen 2007). In variables that were not normally distributed and could not be transformed the Spearman's rank correlation coefficient (r_s) was used. It is felt that regression was the most robust way of establishing the significance of explanatory variables for their effect on the dependent variables, given the sample size available.

Multivariate linear regression was used to ascertain the effect of significant confounders on maximal ICP, ICP rise and minutes to recovery (dependent variables), during both endotracheal suctioning and turning. It is claimed that these models should be built up through a series of prior analyses to establish significant correlation with the dependant variable and that these explanatory variables entered into the model should not be collinear (Peat and Barton 2005; Petrie and Sabin 2005). Additionally, these models should be 'over-fitted' with a large number of explanatory variables entered into the model otherwise over inflated R values may result, it is recommended that for a sample size of 25 a maximum of two variables could be put into the regression model (Petrie and Sabin 2005: Peat and Barton 2005) approximately one variable for every ten patients.

The effect of these confounders was explored descriptively and then inferentially with independent t test. Only explanatory variables which were significant (p value < 0.05) and for which there was clinical rationale were

put into the regression model (Anthony 1999). These explanatory variables were put into the regression model and both a stepwise forward and backward regression method was used to ascertain their effects on the dependant variables. A Bonferroni correction was applied to the p value as recommended, avoiding a type I error (Petrie and Sabin 2005 p44, Bailar and Monsteller 1992 p246). The majority of confounding variables were consistently applied across all study patients (head of bed elevation, PEEP, baseline PaCo2, baseline oxygen saturations, the presence of a rigid cervical collar, core temperature, positive inotrope infusion and the use of hyperosmolar agents). The confounders which were not were: the use of barbiturates, the use of a sedation bolus, the use of ET lignocaine and the presence of muscle relaxation.

Given the small sample size of 25 children in this study there is always a risk of a type II error occurring (where the null hypothesis is rejected, when there may in fact be a significant difference). This is more pronounced when reducing the sample size even further by looking at groups within in it for example, by exploring the effects of confounders using linear regression. This would show as a lack of significance in the p value for the model coefficients, due to insufficient power. The examination of the effect of confounders is not the primary aim of this study but is a secondary aim.

The analysis of recovery times after interventions was done using time to event analysis (recovery time to baseline ICP) and the data presented as Kaplan Maier curves with 95% confidence intervals (Pocock et al 2002, Altman and Bland 1998). It is expected that the confidence intervals are likely to be wide because of the small sample size. Differences between the two stratified groups are analysed using the log rank test (Bailar and Monsteller 1992 p287). Critical in calculating time to event, is the definition of a well defined starting point (Bailar and Monsteller 1992 p 281). The defined time frame for 'minutes to recovery' in this study is: the time when the intervention ceases to the time the child's baseline ICP is achieved.

The results of the small group of children who are excluded from the main dataset following decompressive craniectomy, will be presented as a case series and thus analysed descriptively only, using box plots and percentage change from baseline. It was felt to be important to examine this small group as there is no published literature in this group of children at all.

There were some missing values to deal with in the analysis. However, none of the primary outcome measure of ICP was missing in the endotracheal suctioning dataset or the turning dataset. Upon examination the missing values appeared to be missing completely at random and almost certainly related to the limitation of having the bedside nurse (and primary clinician) collecting and recording the data. For the analysis these

missing values were left blank (no attempt was made to impute these values). Statistical advice was sought three times throughout the study in addition to the supervisory team, and this was provided by both The University of Liverpool medical statistics department and Liverpool John Moores University.

5.27 Discussion of ethical issues

Obtaining Informed consent

Informed consent is an essential component of the ethical concept of respect for autonomy (Gillon 1994). In the case of children (and almost always in intensive care) this is the autonomy and wishes of the parents/guardians that are respected. Having a clinician as researcher inevitably affects the consent rate (increases it), although no coercion to participate in the study was applied, with consent rate of 96% (only one parent refused consent), there is no doubt that this has impacted on the consent rate as others have described (Peters et al 2004). The non-interventional nature of this study probably also contributed to the high consent rate but the parents' ability to be able to comprehend details of a research study at this time need to be taken into account, so timing of the consent process is also important in these circumstances. Despite the researcher's concern about the parents' ability to comprehend and make decisions at this stressful time, a recent paper (Needle et al 2009) found that although parental anxiety is high in the first 24 hours after a child's

PICU admission, 93% of parents had fair to excellent understanding of the medical issues relating to their child's illness at this time.

As previously discussed consent was gained by the researcher or two other senior intensive care nurses with training in consent who were well informed about the study. This was discussed and agreed with my supervisors; one of whom is a Consultant in Paediatric Intensive Care on this unit.

Maintaining confidentiality

As with any research or audit, the researcher has a duty of care to keep the child's data confidential so that the individual child is not identifiable. Once the data collection forms were completed they were coded and entered onto a secure, password protected, SPSS version 15 Database held on the researcher's personal laptop. The codes for the patient identity were kept separate in a locked file in a locked education office within the PICU. This is in keeping with the Data Protection Act 1998 and trust data protection guidelines.

Beneficence and non-malificence

Approaching parents of critically ill children for research participation is always a sensitive topic, as these parents are generally incredibly stressed by the severity and suddenness of the child's injury and the risk to the child's life. Experienced PICU nurses are familiar with supporting and dealing with families under these terrible circumstances and care and

sensitivity must always be considered in terms of the approach to the parents, whilst ensuring that the voluntary nature of study participation is emphasised. Although some question the ethics behind conducting research in critically ill patients, the author would argue that we (health care professionals) have a moral duty to conduct research in our patient population, to improve practice and the care that we deliver to our patients and promote the ethical principles of beneficence and non-malificence (Gillon 1994). It is only through research that we can generate new knowledge and even hope to deliver better care to critically ill children.

5.28 Conclusion

The methodology described has attempted to address many of the problems and limitations identified with previous work. The methods and following results reported conform to the STROBE guidelines for the reporting of observational studies (von Elm et al 2007). This guidance is designed to assist researchers and editors in improving the reporting of research of this type.

CHAPTER 6

RESULTS: ENDOTRACHEAL SUCTIONING AND MANUAL VENTILATION IN CHILDREN WITH MODERATE TO SEVERE TRAUMATIC BRAIN INJURY

6.1 Introduction

This chapter presents the results of the effects of endotracheal suctioning and manual ventilation (ETSMV) in the moderate to severe head injured child and will be presented in a few parts. Endotracheal suctioning was the most commonly performed intervention overall, accounting for 48.5% of recorded interventions. This is not surprising given the importance of ensuring a patent airway and in controlling $PaCo_2$ in these children.

The first section presents the demographical data of this study group, the baseline data and the distribution of the data in the outcome measures for analysis. The second part presents the change in the primary outcome measure (ICP) with ETSMV, the percentage change from baseline and the results of this when stratified to high and low baseline ICP groups. The third part examines the correlation between variables during ETSMV and the effect of confounders. The fourth part looks at recovery times after endotracheal suctioning and the last part examines the mean arterial pressure changes during ETSMV.

6.2 Demographics of the suctioning group

Twenty-five children with moderate to severe closed head injury who required ICP monitoring were recruited to the study over a three year period (Jan 2006- Dec 2008). The demographics of the group are shown below. This did exclude some children whose injuries were so catastrophic that ICP

monitoring was not considered appropriate (n=9). It also excluded children under two years (n=2), children who had an open head injury (n=1) whose parents refused consent (n=1) and children after decompressive craniectomy (n=5). The mortality in the study sample was 8% 2/25 and this roughly correlates to the predicted PIM 2 score of 0.067 (~7%).

Table 4: Demographics of study ETSMV sample (n=25)

Demographic	Mean (SD), Median (IQR) or percentage of group
Age	9.4 years (SD 4.5)
Gender	15/25 (60%) male
Post-resuscitation GCS	7 (range 3- 13)
Patients with GCS >9 who required ICP monitoring	5/25 (20%)
Mechanism of injury	Pedestrian RTA 10/25 (40%) Passenger RTA 7/25 (28%) Fall 6/25 (24%) Cyclist 1/25 (4%) Crushed 1/25 (4%)
Presence of other significant injuries	15/25 (60%) of patients of these: 11/15 (73%) limb fractures; 7/15 (47%) lung/chest injuries
Neurosurgical intervention (excluding bolt and EVD)	9/25 (36%) of these patients most commonly: 4/9 (44%) Extradural 3/9 (33%) decompressive craniectomy
Mean day 1 ICP mmHg	16.6 (SD 5.44)
PICU length of stay (Days)	5 (IQR 3-11)
Decompressive craniectomy	13% (3/25 crossed over into DC group after 1st measurement done)
Absolute mortality	2/25
Mean PIM 2 score	0.069 (95%CI 0.017 - 0.121)
PIM derived standardised mortality ratio (SMR) of group	1.158 (95%CI 0.140 – 4.184)
Six month outcome	19/23 (83%) mild-moderate disability (GOS 1 and 2) 17% 4/23 (17%) severe disability (GOS 3)

The first ETSMV intervention measured and recorded was used as the value per patient and this was undertaken in the first 36 hours after PICU admission in all the children (i.e. the hyper acute phase of head injury). The mean baseline demographics (pre-intervention) of the sample are shown in Table 5.

Table 5: Mean baseline data prior to ETSMV

	Mean	SD
Baseline ICP mmHg	18.1	8.7
Baseline MAP mmHg	85	10.8
Baseline CPP mmHg	67	8.9

These baseline demographics demonstrate the overall severity of illness of the group and the relatively high mean baseline ICP (18.1mmHg) pre-intervention further emphasises this, as a moderate to severe closed head-injured paediatric sample.

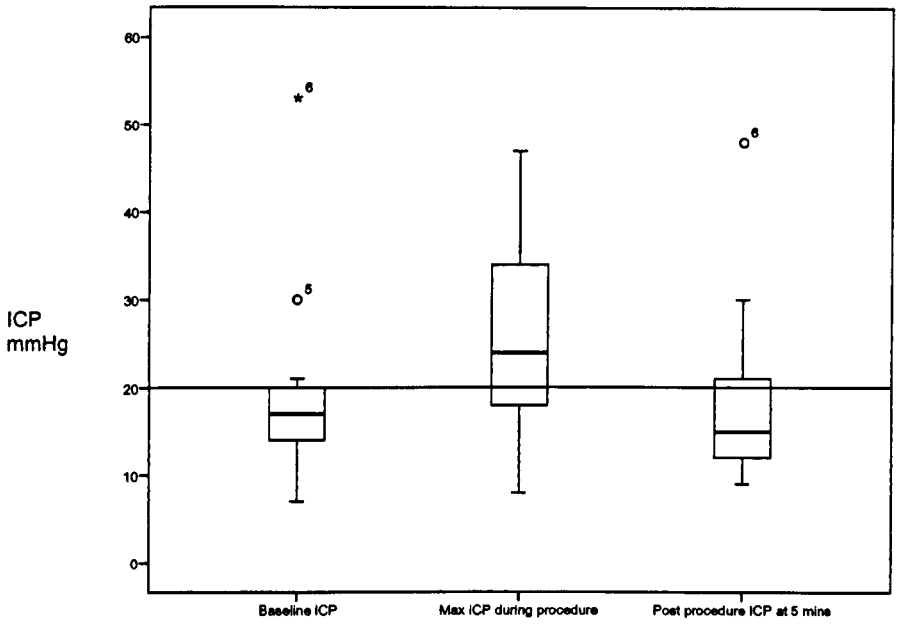
6.3 The distribution of the data in the suctioning group

Upon examination of the data, the primary outcome measures (baseline ICP, maximal ICP, ICP rise and Post ICP) were not all perfectly normally distributed, and in small samples (i.e. n=<30) this is important if parametric tests are to be used (Peat and Barton 2005). The distribution of the data are shown below

Table 6: Distribution of ETSMV data

		Baseline ICP	Max ICP during procedure	ICP rise	Post procedure ICP at 5 mins
N	Valid	25	25	25	25
	Missing	0	0	0	0
Mean		18.08	25.84	7.72	17.64
Median		17.00	24.00	5.00	15.00
Std. Deviation		8.737	11.025	11.524	8.577
Skewness		2.749	.414	.193	2.054
Std. Error of Skewness		.464	.464	.464	.464
Range		46	39	45	39

It is clear that baseline ICP and post procedure ICP are not normally distributed and so parametric tests cannot be used on this data as it stands. If a box plot is presented of this data, then it becomes apparent that one outlier (patient no.6 who subsequently died) is likely to be responsible for this skewed data in baseline and post ICP.



Graph 13: The effect of ETSMV on ICP response with outlier in

If this outlier is removed, then all primary outcome measures are now normally distributed (and suitable for parametric test analysis).

Table 7: Dispersion of ETSMV data following removal of outlier

		Baseline ICP	Max ICP during procedure	ICP rise	Post procedure ICP at 5 mins
N	Valid	24	24	24	24
	Missing	0	0	0	0
Mean		16.63	25.29	8.63	16.38
Median		16.50	24.00	7.00	15.00
Std. Deviation		4.942	10.909	10.826	5.918
Skewness		.167	.529	.353	.856
Std. Error of Skewness		.472	.472	.472	.472
Range		23	39	44	21

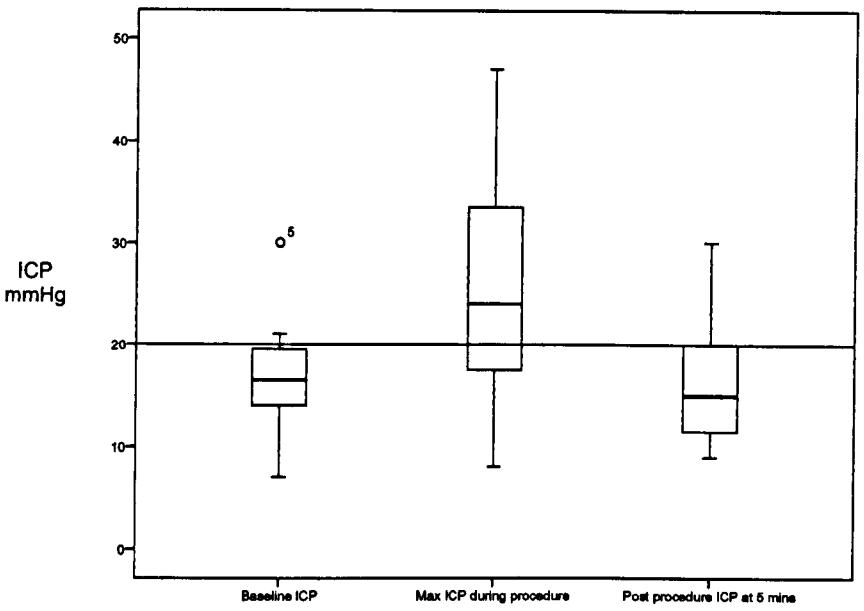
To further confirm this and clearly justify the accurate use of parametric tests, the data was formally tested for normality using the Shapiro-Wilks test, which is the suggested test for samples smaller than 50 (Peat and Barton 2005). Using these statistical tests obtaining p values >0.05 shows the data are consistent with normality. The results show that the S-W test baseline ICP (p=0.097) and post procedure ICP (p=0.059) are marginally consistent with normality hypothesis (p values between 0.05 – 1) while maximal ICP (p=0.305) and ICR rise (p=0.660) are comfortably within the expected range for normality, recording p values well in excess of the threshold significance of the test. These results confirm that the data are normally distributed and thus parametric tests can be used.

6.4 The effects of endotracheal suctioning on the ICP response

The data were thus analysed both with this patient in (using non-parametric tests) and with her removed (using parametric tests). In terms of demonstrating a significant change from baseline ICP to maximal ICP, and maximal ICP to post ICP, both sets of tests demonstrated statistical significance.

With the data normally distributed (the outlier removed, and so a sample of 24 children), the paired t test was used to compare the means from baseline to maximal ICP and maximal to post ICP during ETSMV. This showed a significant change from baseline to maximal ICP (p=0.001; 95% CI 4.14 –

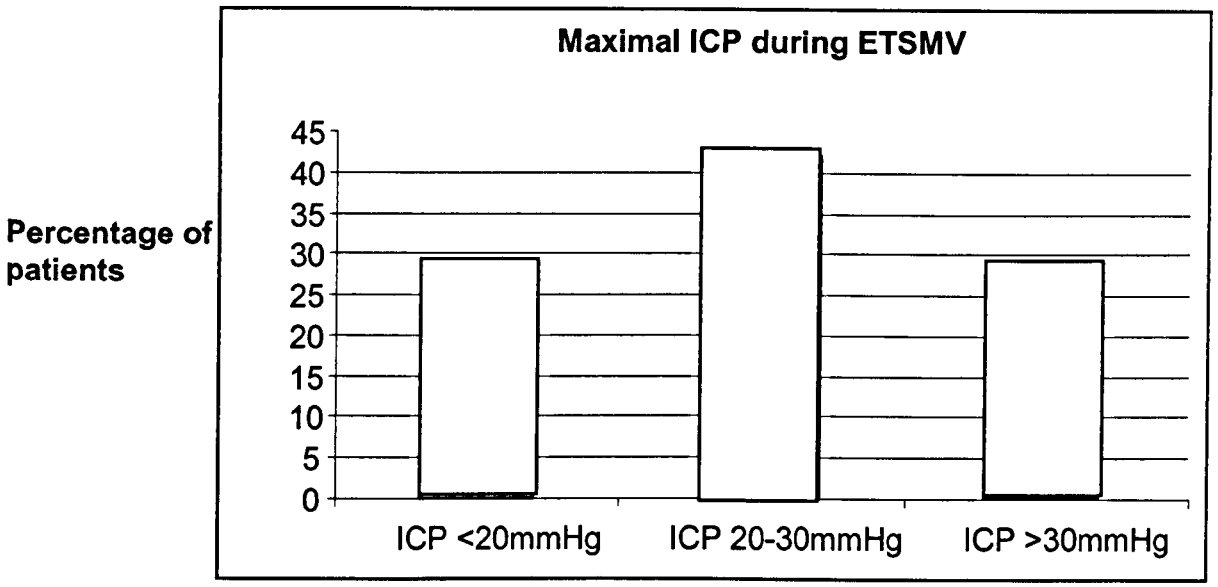
13.2) and from maximal to post ICP (p=<0.001; 95% CI 13.4 – 4.06). This can be seen on the box plot below.



Graph 14: The effect of ETSMV on ICP response with the outlier removed

When the outlier was left in (and the data skewed), using non-parametric tests (Wilcoxon signed rank test) still demonstrated a statistically significant change from baseline ICP to maximal ICP (p= 0.005) and from maximal ICP to post ICP (p = 0.001). In addition to being statistically significant, this change from baseline to maximal ICP is also clinically significant, as the majority of children exceeded the recognised treatment threshold of 20mmHg during the procedure (Adelson et al 2003).

On a clinical level, in terms of simple percentages of how many children recorded maximal ICP values above 20mmHg, how many exceeded 30mmHg and their percentage change from baseline, the graphs below demonstrate these results.

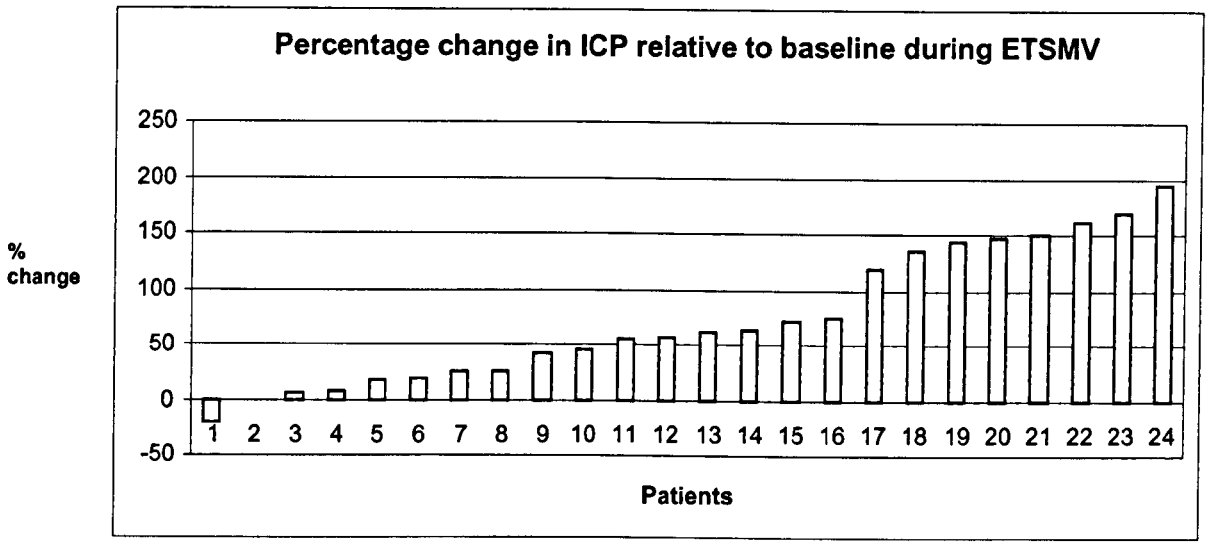


Graph 15: Maximal ICP during ETSMV

In these terms, 70% of children exceeded the 20mmHg ICP level during suctioning, with 28% of them exceeding 30mmHg.

The percentage change from baseline is a very inefficient statistical test, which does not correct for the differences in baseline ICP (Vickers 2001) however this was first calculated (graph 16) before ANCOVA analysis was done to correct for the baseline ICP. Represented below is the percentage change in ICP with suctioning (from the child's baseline). This is calculated by peak-baseline/baseline x 100. The mean change from baseline was

+74% ([-20] in one child – [+194] %). These have been sorted into ascending order.



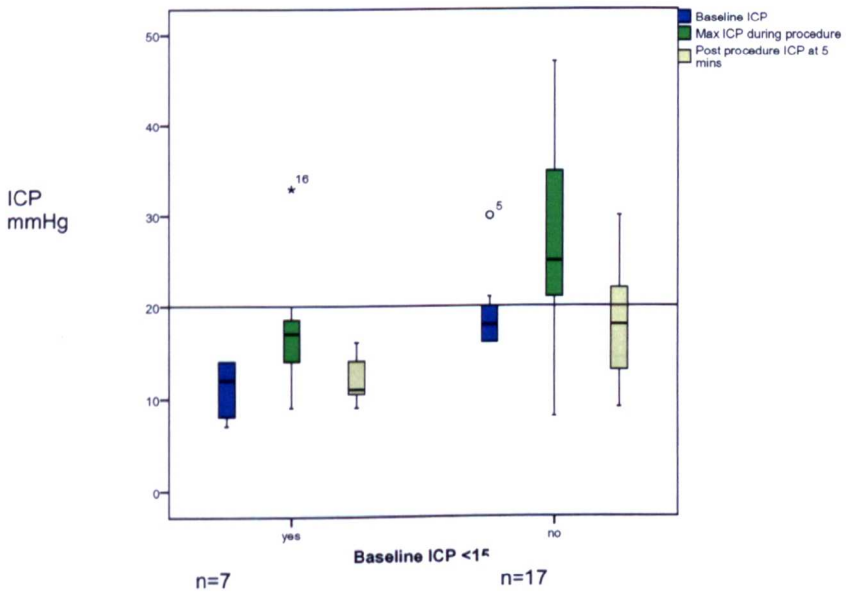
Graph 16: The percentage change from baseline ICP with ETSMV

Vickers (2001) suggests that ANCOVA analysis has a much greater statistical power than percentage change from baseline, which corrects for baseline imbalances. ANCOVA analysis of the percentage change in ICP from baseline (as dependant variable) with baseline ICP as the factor showed a non significant p value ($p = 0.351$) reflecting that the change from baseline to maximal ICP did not relate to the baseline ICP with suctioning.

6.5 Stratification by baseline ICP

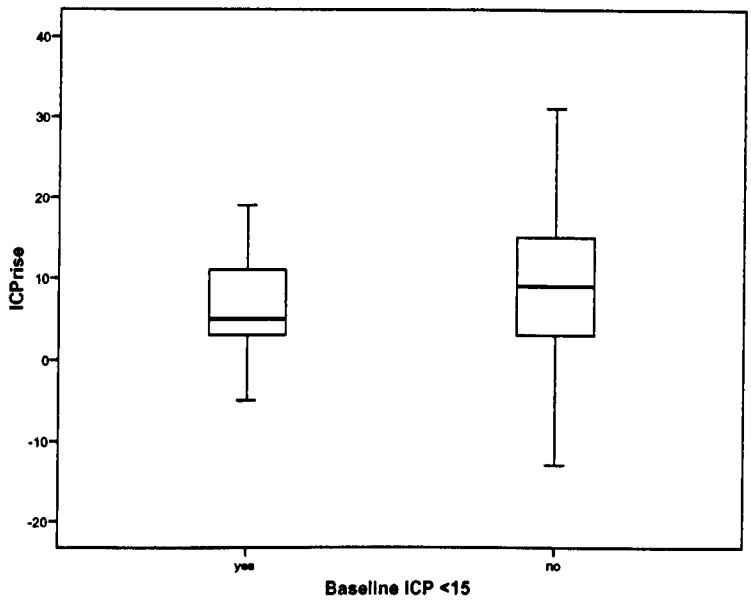
Previous studies have shown significant differences in changes in ICP and recovery in adults with higher baseline ICPs (defined as $\geq$ 15mmHg) compared to those with lower baseline ICPs (< 15mmHg) (Garradd and Bullock 1986; Paratz and Burns 1993).

To test the hypothesis that children who had higher baseline ICPs (defined as $\geq$ 15mmHg) would exhibit a higher ICP response, the children were stratified into two baseline ICP groups. This arbitrary stratification value of 15mmHg was used because of a previous study justification, however it must be noted that 71% (7/24) of children before suctioning had baseline ICPs in the higher range. The median baseline ICP was 17mmHg in this group (mean 18.1) however given the treatment threshold of 20mmHg, it still appeared reasonable to use 15mmHg as the cut off. The box plot below shows the results when the children were stratified to low and high baseline ICP groups.



Graph 17: ICP response during ETSMV by baseline ICP stratification

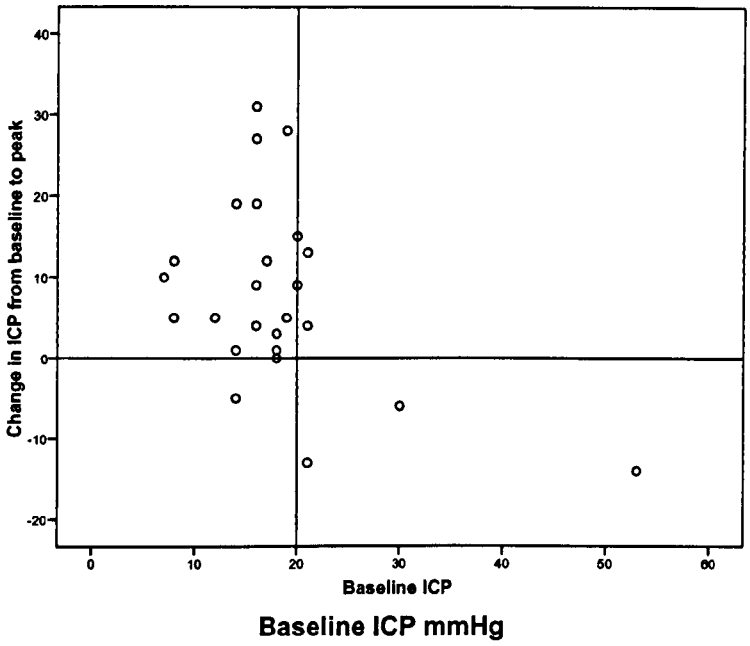
An independent sample t test showed significant differences between the maximal ICP (p=0.014 95% CI 2.5 - 19.8) and post procedure ICP (p= 0.003 95% CI 2.3 - 9.6) between the high and low baseline group, but no significant difference between the ICP rise (p=0.523) which Graph 18 demonstrates.



Graph 18: ICP rise during ETSMV by baseline stratification

In addition we investigated whether the change in ICP from baseline to maximal was related to the baseline ICP. On the scatter plot (Graph 19) it can be seen that the two children with highest baseline ICPs actually had a reduction in ICP during ETSMV. This suggests that it may be the manual ventilation procedure (with likely reduction in $PaCo_2$) that has resulted in this maximal ICP reduction, as there is evidence that children with very severe

injuries are particularly sensitive to changes in $PaCo_2$ (Skippen et al 1997, Coles et al 2007, Kerr et al 1997).



Graph 19: Change in ICP from baseline to maximal during ETSMV and baseline ICP

6.6 The correlation between variables during ETSMV

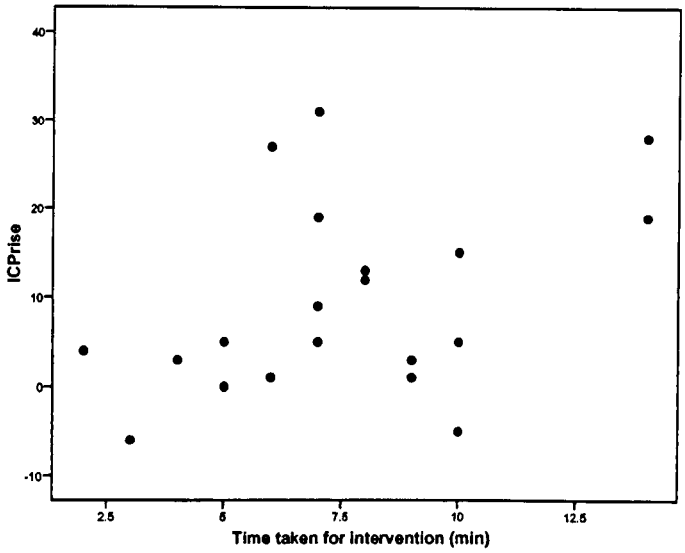
The correlation analysis presented below is in the sample of 24 children (the outlier removed) using Pearson's correlation coefficient (r), which assumes normally distributed data. Variables that were normally distributed were analysed in this way. Minutes to recovery was not normally distributed and was unable to be transformed to a normal distribution, so was analysed

using the non-parametric test Spearman's rank correlation coefficient (Bland 2003 p.220). Scatter plots were done of all the normally distributed variables. Table 8 shows a summary of the results of the correlation analysis. Only variables with an r value of 0.4 and above (ie at least moderate correlation) Harris and Taylor (2004) are presented on scatter plots.

Table 8: Correlation table results for ETSMV

Factors examined	r value or correlation coefficient	p value
Baseline ICP to Maximal ICP	0.247	0.244
Baseline ICP to ICP rise	-0.211	0.323
Time taken for ETSMV and Maximal ICP	0.331	0.132
Time taken for ETSMV and ICP rise	0.404	*0.062*
Maximal ICP and post procedure ICP (at 5 minutes)	0.379	*0.062*
Baseline ICP and corrected EtCo2	-0.198	0.391
Maximal ICP and minutes to recovery	0.34	0.873
Time taken for ETSMV and minutes to recovery	0.010	0.965
Baseline ICP and minutes to recovery	-0.294	0.154

When the relationship between time taken for intervention (in minutes) to maximal ICP and ICP rise was examined, there appeared to be a slight correlation, with a trend towards statistical significance only on ICP rise ($r = 0.4$; $p = 0.062$). Two children had this variable (time taken) missing so the analysis below is in 22 children only.



Graph 20: Correlation between time taken for ETSMV and ICP rise

It was felt to be important to examine the effect of carbon dioxide level (as measured by end tidal measured CO_2 (EtCo2)) on baseline ICP as is it known to exert a significant impact on ICP through vasodilatation of cerebral vessels (Skippen et al 1997). As a stand alone value there was no correlation between EtCo2 level and baseline ICP (r -0.198; p=0.391), this is probably because the EtCo2 level for the study sample was fairly narrow in the low-normal range with a mean of 36.4 mmHg (SD 5.03). We tried to account for the effect of reduction in PaCo2 during manual ventilation by recording the EtCo2 within 30 seconds of being connected back to the ventilator. We did this after the study had started so only 14 children had this data recorded. In 6/14 children (42%) the EtCo2 was lower than that measured at baseline prior to manual ventilation (an average of 3.6mmHg

lower), but there appeared to be no association between the changes in this level and a drop in ICP.

6.7 The effect of confounders during endotracheal suctioning

As discussed earlier there are multiple therapies potentially acting as confounders in this study. Table nine shows the usage of confounders in the study sample before ETSMV. Three of these were actually fairly consistent across the whole sample: the degree of head of the bed elevation (always at 10-20° elevation), PEEP (almost all ≤5cm H_2O) and presence of neck compression by rigid cervical collar (nearly always opened out). Others (the use of hyperosmolar agents and propofol infusion) were used rarely. All children received continuous infusions of standard doses of an opiate and a sedative. Twenty four out of twenty five children (96%) received morphine at 40 micrograms/kg/hour and one child received fentanyl. Nineteen children (76%) received a midazolam infusion as their sedative at 100 – 200 micrograms/kg/hour; five were on thiopentone, and one on propofol for sedation.

Table 9: Presence of confounders prior to endotracheal suctioning

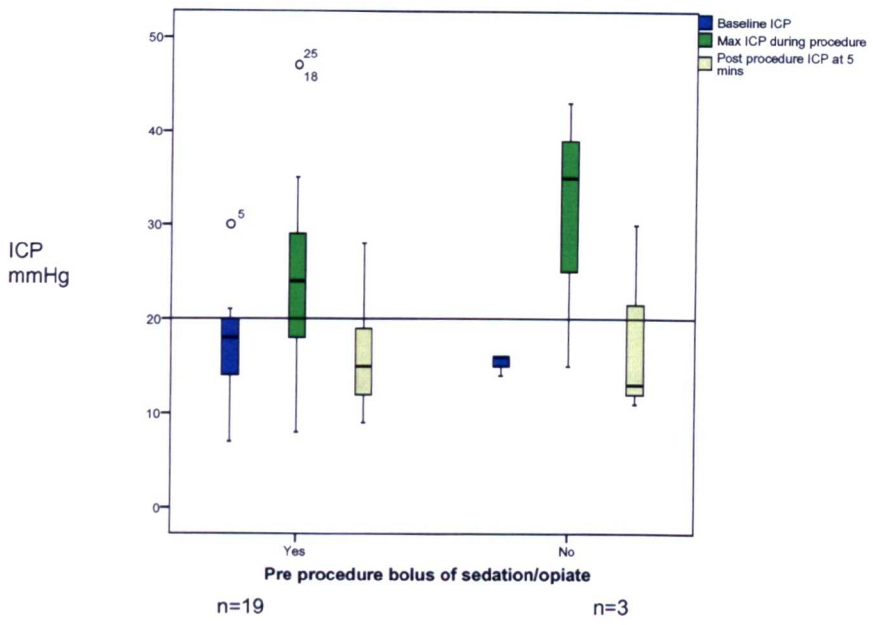
	Mean or (% of group)	SD
Head of Bed Elevation °	13.4° elevation	4.7
PEEP cmH_20	5.1	1.36
Time taken for ETSMV (mins)	7.43	2.98
Core temperature° C	36.3°	1.3
Cervical Collar loose/opened or nor present	22/25 (88%)	
Baseline EtCo2 level mmHg	36.4	5.03
Baseline Spo2 (%)	99.7	0.64
Received Hyperosmolar agents in previous 4 hours	2/25 (8%)	
EVD in situ	3/25 (12%)	
Received ET Lignocaine 1%	19/25 (76%) (2 unknown; 3 did not receive)	
Received NMB	21/25 (84%)	
On Propofol infusion	1/25 (4%)	
On Thiopentone infusion	5/25 (20%)	
On inotrope infusion	18/25 (72%)	
On continuous sedation + opiate infusion	25/25 (100%)	
Pre-intervention COMFORT score	0nly available in 3 children Mean 15.3	
Additional sedation bolus pre-suction	22/25 (88%)	
Presence of other (painful) injuries	15/25 (60%) patients of these: 7/15 (47%) lung/chest injuries	

The effects of the main confounders are presented below in box plots. An independent sample t test was used to compare the means between the two groups. It must be noted however that there are very uneven (and small) numbers in some of the groups, because this was an observational study (which studied real clinical practice) and no attempt was made to control variables, most were used at the bedside nurse's discretion, which introduces bias.

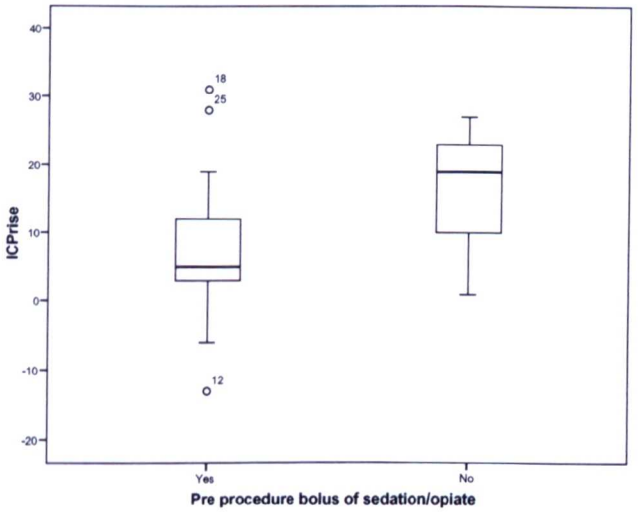
6.8 The effect of pre-procedure sedation bolus

Despite the fact that all children were receiving continuous infusions of an opiate (predominantly Morphine) and a sedative (all midazolam) the vast majority of children, 88%, had an extra intravenous (IV) bolus of a sedation/and or opiate analgesic prior to the ETSMV procedure. This was predominantly IV Midazolam (as sedation 17/25 68%) and Morphine (as analgesic 96% (24/25). However this was prescribed 'as required' and was administered at the nurse's discretion. If the children are not receiving paralytic agents then the level of sedation can be quantified using a validated scoring tool (the COMFORT score) (Ambuel et al 1992), but this was not possible in the majority of children who were muscle relaxed.

A bolus of sedation prior to the intervention did lower the median maximal ICP and the ICP rise, but both groups still exceeded the clinically significant 20mmHg level of the maximal ICP (see Graphs 21 and 22) . However there was not a statistically significant difference in maximal ICP ($p=0.559$) or ICP rise ($p=0.362$) between the children who received the sedation bolus and those who did not.



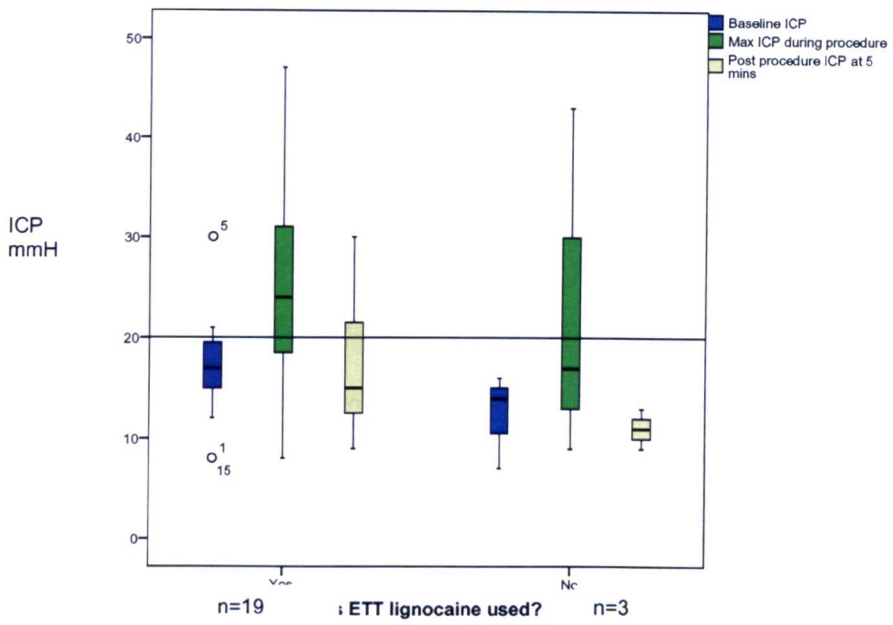
Graph 21: The effect of a sedation bolus on ICP during ETSMV



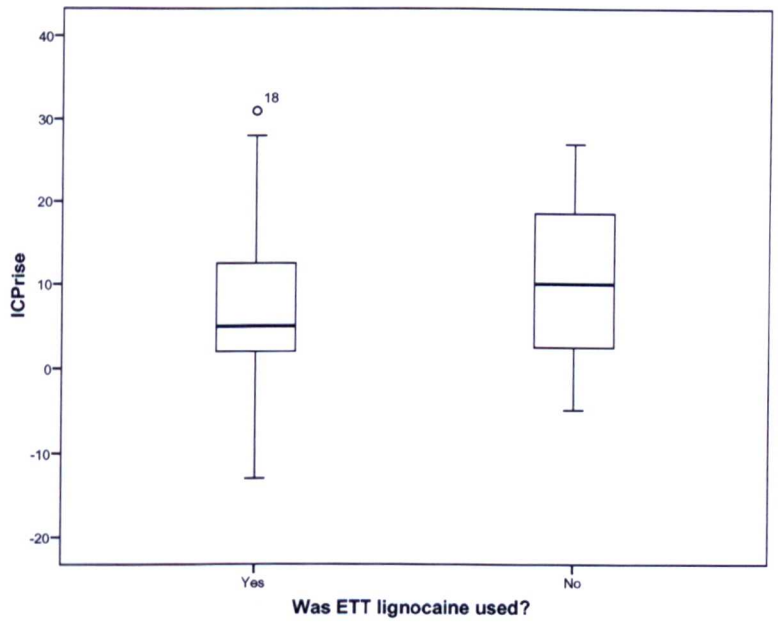
Graph 22: The effect of a sedation bolus on ICP rise during ETSMV

6.9 The effect of endotracheally-administered (ET) Lignocaine

Lignocaine (1%) is a local anaesthetic agent which has been shown to blunt the ICP response in some adult and paediatric studies (Bilotta et al 2008; Yano et al 1986; Brucia and Rudy 1992). The drug was prescribed 'as required' in this study, prior to ETSMV, but its administration (like all 'as required' drugs) is at the judgement of the bedside nurse, which potentially introduces bias. In this study 76, 19/25 children, were administered the drug (one to two millilitres (mls) of the drug down the ETT) two minutes before the intervention (the amount varied on the weight of the child). In two children this data were missing, and three children did not receive the drug. The box plots that follow show the overall effect of the drug on the ICP response.



Graph 23: The effect of ET lignocaine on ICP during ETSMV



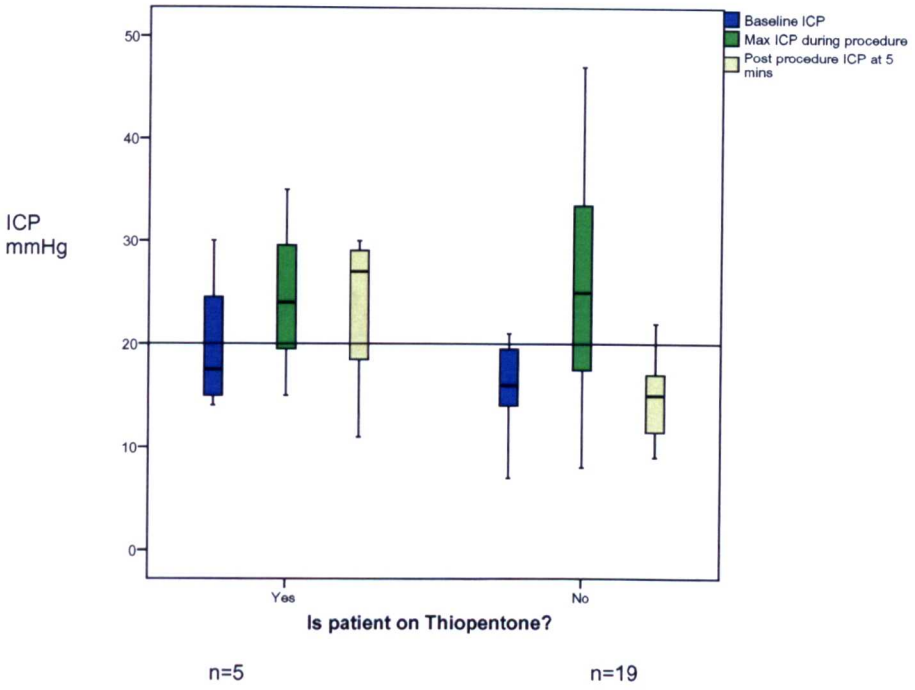
Graph 24: The effect of ET lignocaine on ICP rise during ETSMV

An independent t test showed no difference between the two groups in terms of ICP rise (p=0.822) or the maximal ICP (p=0.841). The maximal ICP was slightly higher in the group that received ET Lignocaine, which may reflect the bedside nurse choosing to use the drug in more severely ill children. In fact the mean baseline ICP in the three children who did not receive lignocaine was 12mmHg, which supports this assumption.

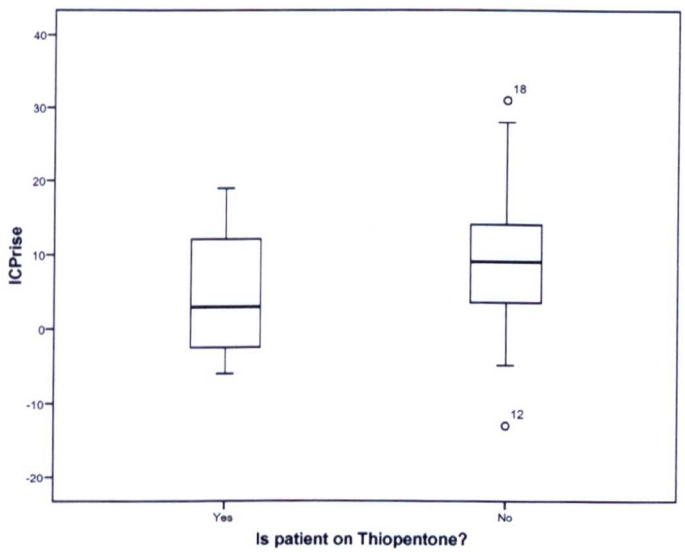
6.10 The effect of barbiturates (thiopentone)

Thiopentone is a strong barbiturate drug that has been shown to significantly reduce brain metabolism and hence oxygen consumption by inducing a comatose state (Eisenbery et al 1988; Pittman et al 1989; Russo et al 1997). It however has some undesirable side effects such as myocardial depression and altered vascular tone which can produce haemodynamic instability in patients receiving the drug; hence the use of this drug is limited to patients where raised ICP is a problem and only where there is some prospect of survival. In this study only 20% children (5/25) received the drug.

Below are the box plots showing the overall effects (from baseline to maximal to post ICP) in children who received this drug compared to those who did not. An independent t test showed a significant difference in the post procedure ICP (p=0.004 CI 3.2 – 14.7) of these two groups, but no significant differences were found between baseline ICP (p=0.170), maximal ICP (p=0.838) or ICP rise (p=0.410).



Graph 25: The effect of thiopentone on ICP during ETSMV



Graph 26: The effect of thiopentone on ICP rise during ETSMV

It might be assumed that the children who were on thiopentone had a more severe brain injury (their baseline ICP was higher, even despite the drug and their higher post ICP reflects a worse cerebral compliance). This higher severity of illness is not reflected in the PIM 2 score (which does not specifically relate to brain injury severity and takes no account of ICP) and there is no validated head injury severity score is used in UK PICU practice to be able to substantiate this assumption.

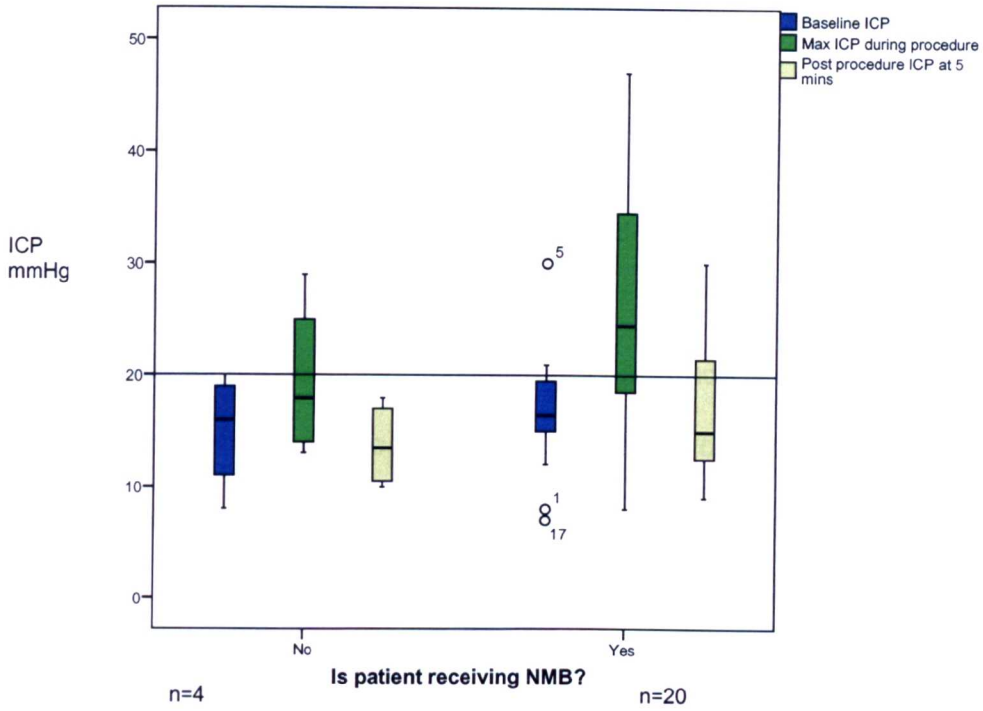
This pattern of an escalating ICP (from baseline to maximal to post) seen in the thiopentone group, varies from the usual response seen in this study (a clear peak in maximal ICP but some recovery in the post ICP value) and in other published studies (Parsons and Ouzts Shogan 1984; Rudy et al 1991). Cerebral compliance (stiffness of the brain) is known to be reduced in children with severe cerebral oedema who are higher up on the pressure/volume curve (Shapiro and Marmarou 1982). Their response to this intervention (a sudden noxious stimulus) may reflect their limited ability to adapt to this sudden change in pressure. Therefore the effects seen in this group cannot be said to reflect the effects of the drug per se, rather they are likely to reflect the effects of ETSMV in a subgroup of children with severe injury and poor cerebral compliance.

6.11 The effect of neuromuscular blockade (NMB)

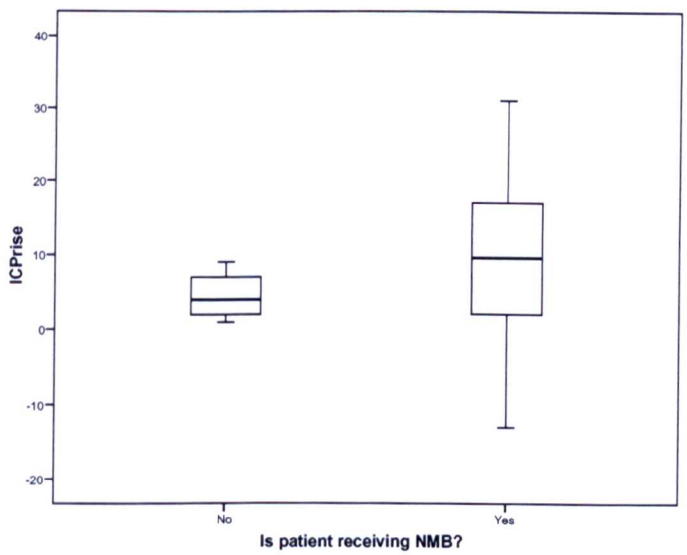
Neuromuscular blockers are drugs that produce temporary paralysis of skeletal muscles, preventing movement, breathing and coughing. The only

two agents used in this sample were the non-depolarising agents of vecuronium (as an intermittent bolus drug) and atracurium (as a continuous infusion). In this study 84% (21/25) were receiving NMB prior to the ETSMV intervention. Patients should be heavily sedated whilst on these drugs (to prevent them being 'awake' under the paralysis), however once paralysed no level of sedation can be formally scored. The only potential indications of inadequate sedation and/or pain are physiological variables such as heart rate and blood pressure (Franck et al 2000). We undertook further analysis examining the MAP changes to support the assumption that some of these children may have been less sedated. This showed no significant difference in maximal MAP between the muscle-relaxed and non muscle-relaxed children. If these children were less sedated it might be expected that their MAP during the intervention would be higher, however it is noted that physiologic variables have been found to be poorly correlated with the sedation level in children (Trope et al 2005).

There were no significant differences in any of the variables, baseline ICP ($p=0.484$), maximal ICP ($p=0.253$), ICP rise ($p=0.416$) or post ICP ($p=0.342$) between the children who received NMB compared to those who did not.
The box plots below show the overall effect of NMB on ICP variables.



Graph 27: The effect of NMB on ICP during ETSMV



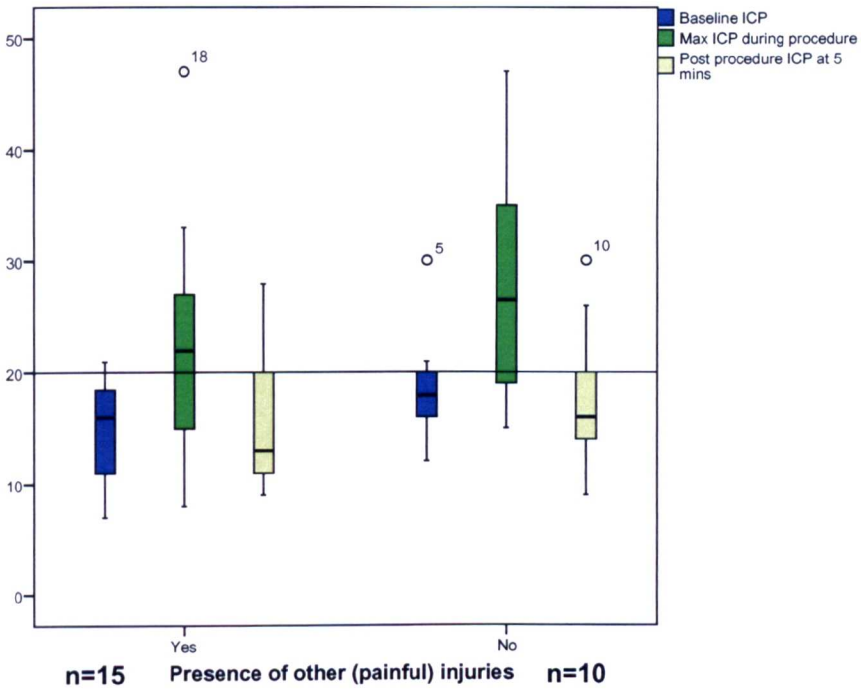
Graph 28: The effect of NMB on ICP rise during ETSMV

Two of the children who were receiving NMB (and thus 'assumed' to be paralysed) actually moved or coughed during the procedure.

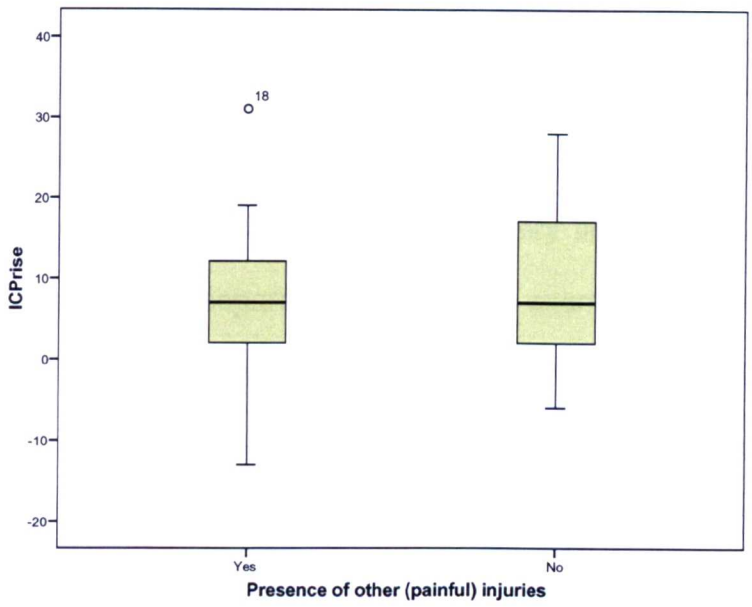
6.12 The effect of having other (painful) injuries during endotracheal suctioning

Endotracheal suctioning children may produce a cough or some movement (in non muscle-relaxed children) and this produce increase pain in children with other injuries. As seen in Table 4, 60% of children had multiple injuries most commonly limb and skeletal fractures and/or chest/lung injuries. It might be assumed the children with chest or lung injuries in particular may experience more pain with endotracheal suctioning. We examined this to see if these children had different responses to suctioning.

Graphs 29 and 30 show their responses.



Graph 29: The effect of having other (painful) injuries on the ICP response to suctioning



Graph 30: The effect of having other (painful) injuries on the ICP rise with suctioning.

Analysis showed no differences between the children with other injuries and those without, in terms of baseline ICP (p=0.091), maximal ICP (p=0.203) or post procedure ICP (p=0.401) or ICP rise (p=0.596) which does not suppose the hypothesis that these children experience more pain on suctioning.

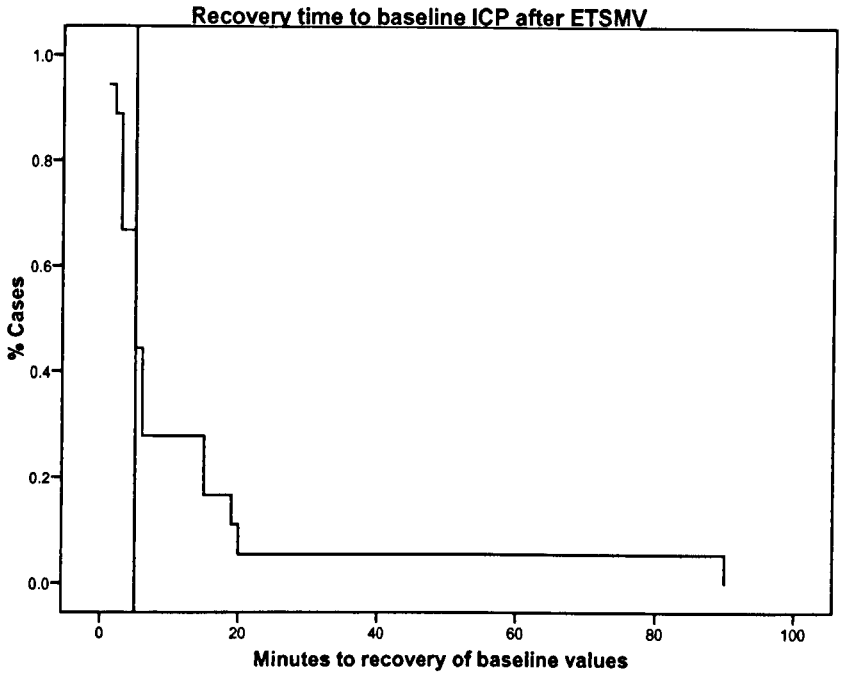
Multivariate linear regression analysis of confounders

No variables on previous analysis achieved a p value of 0.05 or below, thus no multivariate linear regression analysis could be undertaken for endotracheal suctioning.

6.13 Recovery times after endotracheal suctioning and manual ventilation

In addition to maximal ICP the recovery times after this intervention were studied. Two separate issues were examined 1) the time taken for the children to return to his/her baseline ICP values and 2) how many children had mean ICP values that exceeded 20mmHg for more than five minutes. This value (> 20mmHg) and duration (five minutes) is recognised as a significant clinical treatment threshold (Adelson et al 2003) in the effort to minimise secondary brain injury. Time to event analysis (using Kaplan Meir curves) was used to analyse this data (in 25 children), and when groups were compared the log rank test was used to compare differences in this sample. The median time to return to baseline ICP was three minutes (range 0 – 90 minutes; 95% CI for median 1.16 - 4.83 minutes).

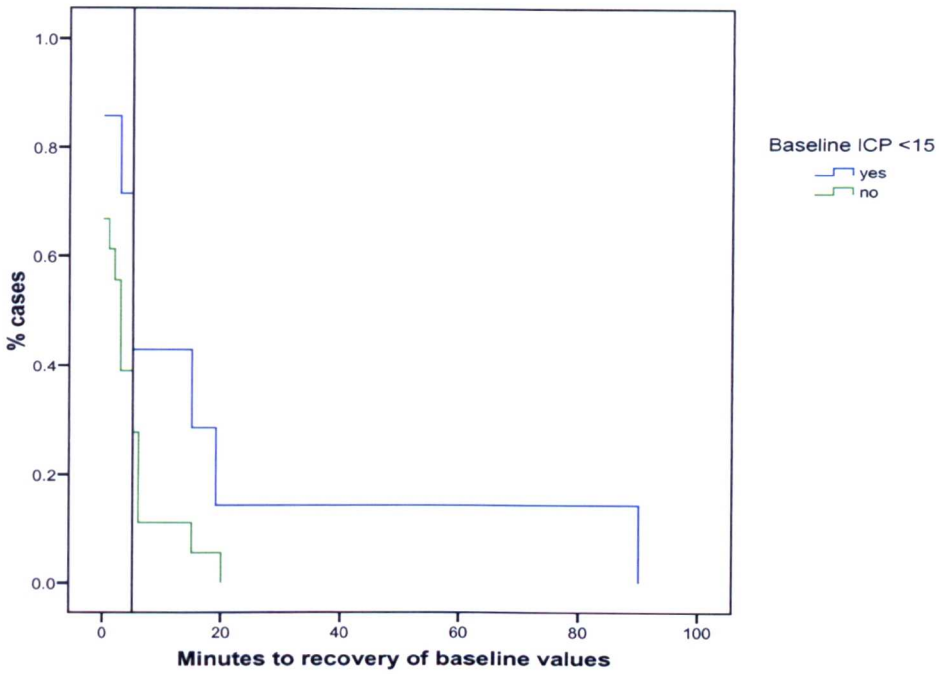
Below is the Kaplan Meir curve to show recovery time (to baseline) in the whole group. The five minute clinically significant time frame is marked in.



95% CI for median [1.16 - 4.83]

Graph 31: Recovery time to baseline ICP after ETSMV

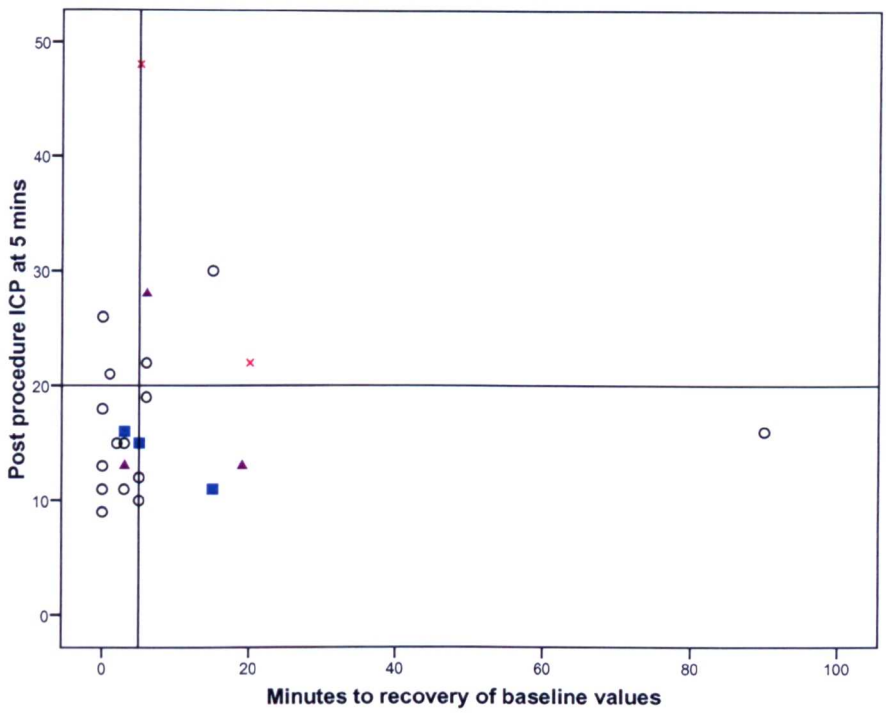
In just over half the children (52%), the post procedure ICP was lower than their baseline ICP. It can be seen from this graph that around 50% of children had not recovered their baseline ICP by five minutes after the procedure. The next Kaplan Meir Curve compares the children by high or low baseline ICP in their recovery times.



Graph 32: Recovery time to baseline ICP when stratified by baseline ICP

When stratified to high or low baseline ICP groups, the children with lower baseline ICPs (<15mmHg) took slightly longer to recovery their baseline values, but this was not statistically significant (p=0.100). This probably reflects that fact that their ICP value was likely to be below 20mmHg and hence there was no active treatment to reduce it.

The scatter plot below shows not only the relationship between post ICP and minutes of recovery, but also the children in whom ICP was greater than 20mmHg for more than five minutes and their six month neuropsychological outcome.

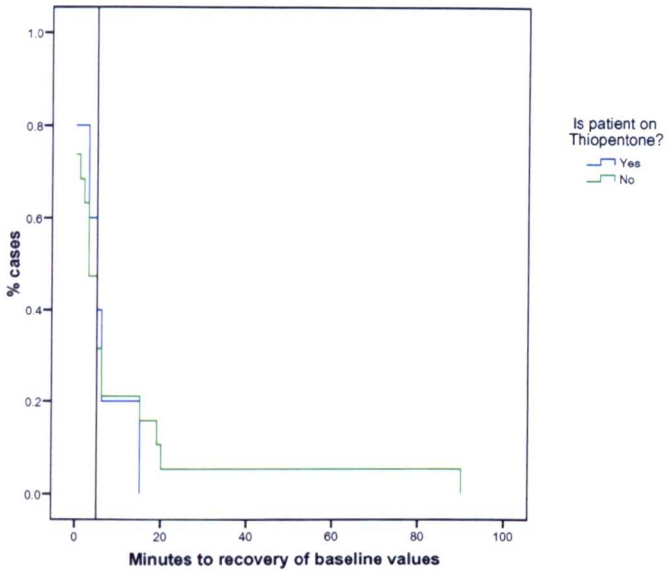


Graph 33: The relationship between post ICP, minutes of recovery and six month outcome after ETSMV

Key: cross = died; triangle = required decompressive craniectomy; square = severe disability (GOS 3); circles = good six month outcome (mild-moderate disability GOS 1or 2).

The recovery time of children receiving thiopentone (n=4) compared to those who did not (n=20) was not statistically significant (p=0.951) see Graph 34. This was explored because of the significantly higher post ICP

value in this group of children presented on previous analysis. This implies that this high post ICP value has been aggressively treated.



Graph 34: Recovery times after ETSMV in children with and without thiopentone

6.14 Changes in mean arterial pressure (MAP) during endotracheal suctioning

The mean arterial pressure (measured and displayed continuously in intensive care) has age-dependent normal parameters, of which the minimal acceptable level ranges from 40mmHg in the term neonate to 60mmHg in the adult patient (Macnab et al 1999). The cerebral perfusion pressure (CPP) is a derived value calculated by the formula CPP = MAP – ICP and

this has suggested age-dependent targets, cited as: 2-6 years > 53mmHg; 7-10 years > 63mmHg and 11-16 years > 66mmHg (Chambers et al 2005).

Given the clinical necessity of ensuring a stable and consistent above-target CPP, the majority of children (72% 18/25 in this study) were supported by an inotrope infusion (virtually all nor-adrenaline). This infusion is adjusted continuously by the bedside nurse to keep the CPP in the target range. It is this issue alone which makes the use of MAP changes as an outcome measure, not related to the child, but to iatrogenic manipulation. In other published papers (in adults) the majority of patients are not on inotropes and hence MAP can be used as an outcome measure. The fact the MAP is manipulated to achieve a desired CPP also make CPP changes not a useful outcome measure. Therefore these two parameters, initially planned to be primary outcome measures, proved not to be useful in this study, however the changes in these variables during suctioning are briefly presented.

Looking at the distribution of the MAP data in the total sample of 25 (below) both on box plot and on statistics, it can be seen that one child (no.6) is again an outlier, who has affected the outcome measure of baseline ICP, but the other outcome measures were normally distributed.

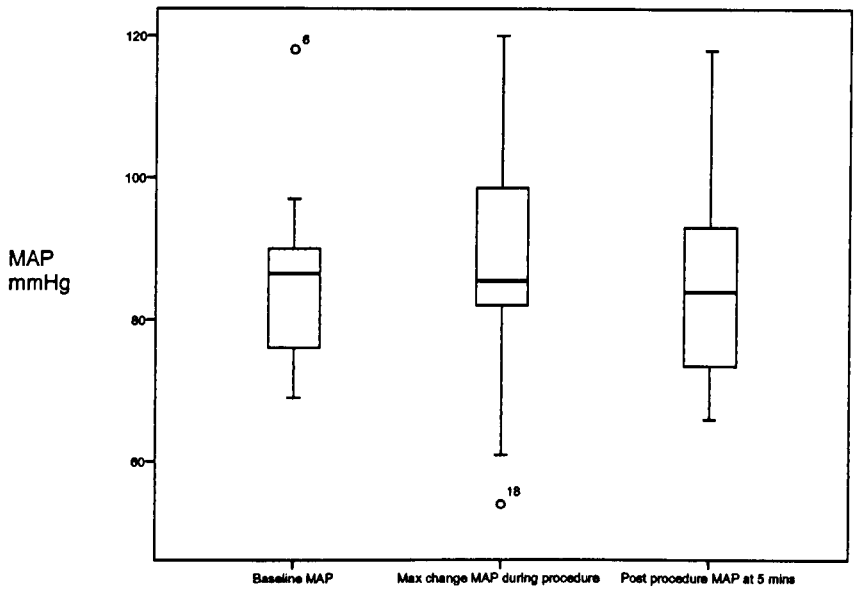
Table 10: Dispersion of the MAP data

		Baseline MAP	Max change MAP during procedure	Post procedure MAP at 5 mins
N	Valid	25	25	24
	Missing	0	0	1
Mean		85.44	88.56	84.21
Median		87.00	86.00	84.00
Std. Deviation		10.778	14.922	12.958
Skewness		.789	-.269	.470
Std. Error of Skewness		.464	.464	.472

6.15 Changes in mean arterial pressure, cerebral perfusion pressure and ICP during endotracheal suctioning

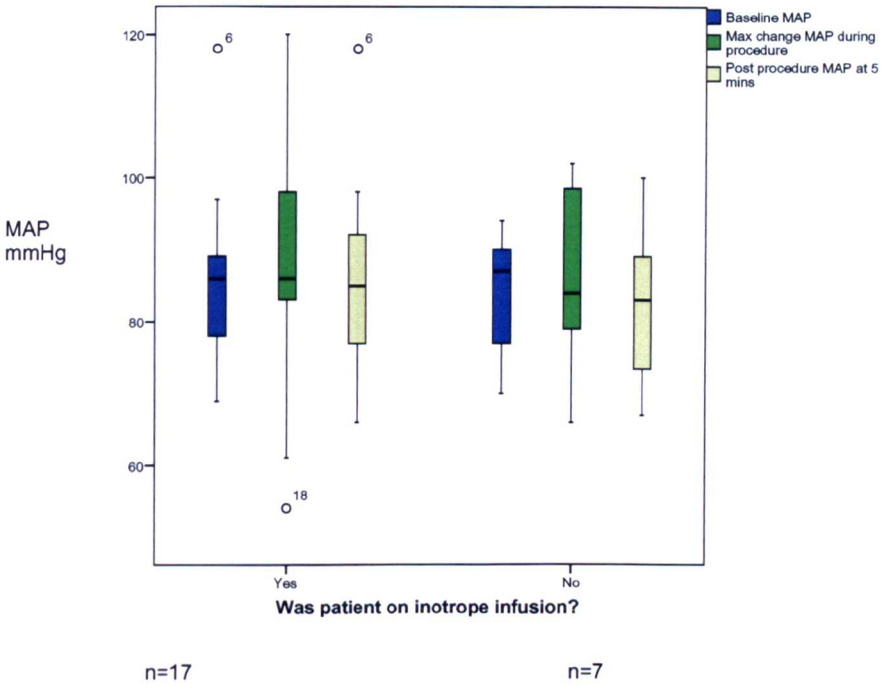
There was very little variation in median MAP values during ETSMV overall. However MAP did change (in line with the formula CPP= MAP - ICP) during suctioning but the change was neither clinically nor statistically significant. MAP rose in concert with ICP, partly compensating for the ICP rise.

During suctioning the mean arterial pressure changed from 84mmHg (at baseline) to 87mmHg during the procedure and by five minutes post procedure this had reduced back to 82mmHg. At the same time mean ICP changed from 16.6mmHg (at baseline) to 25.2mmHg during the procedure and was 16.3mmHg at five minutes post procedure. Correspondingly, the CPP at baseline was 67mmHg this reduced to 63mmHg and was back to a baseline value of 67mmHg at five minutes post procedure. Neither of the changes in MAP or CPP was either statistically or clinically significant. Therefore the ICP changed significantly more than MAP, which partly compensated for the ICP rise, resulting in only a minimal reduction in CPP.



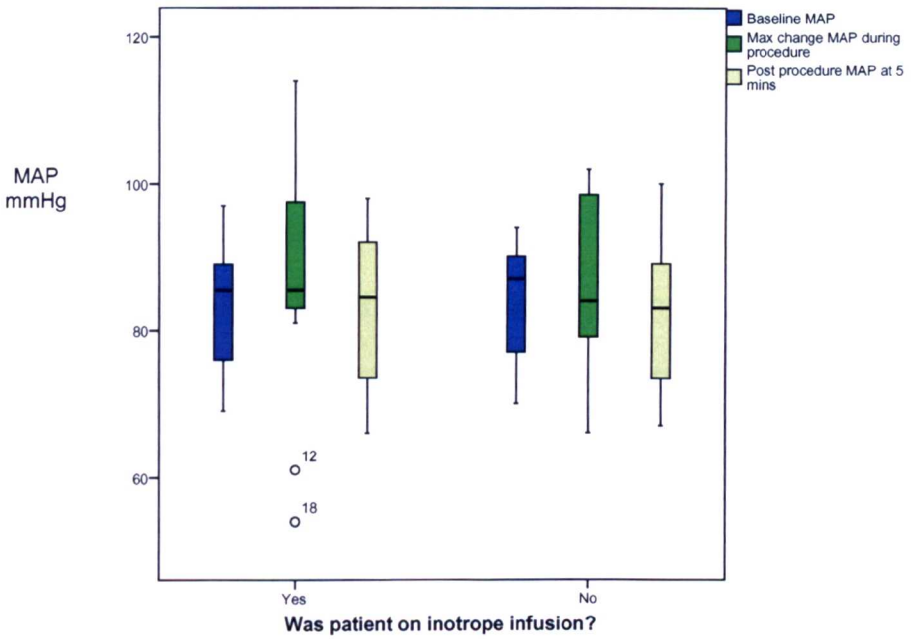
Graph 35: The effect of ETSMV on MAP

When we examined the difference between the children on inotropic infusions and those not (see Graph 36 and 37) we again found very little difference in the two groups in terms of median MAP values. However the range in maximal MAP was very wide in the children receiving inotropes. Child no.6 (who subsequently died) had such a severe brain injury, in which cerebral autoregulation was grossly impaired, whereby any rise in MAP directly resulted in an almost directly proportional rise in ICP and vice versa.



Graph 36: The effect of ETSMV on MAP in children with and without nor-adrenaline (outlier in)

If the extreme outlier (patient 6) is removed (see below), then there is less variability between the two groups.



Graph 37: The effect of ETSMV on MAP during ETSMV in children with and without nor-adrenaline (with outlier removed)

6.16 Conclusion and summary of results for endotracheal suctioning and manual ventilation

In summary, ETSMV produces both a clinically and statistically significant change from baseline ICP, with 70% of children exceeding 20mmHg and around one third exceeding 30mmHg during the intervention. Children with higher ($\geq$15mmHg) baseline ICPs showed higher maximal ICP and post procedure ICP values, however the ICP rise was similar between the two groups (high and low BICP) suggesting that ETSMV produces a linear rise in ICP. Most children recovered rapidly after ETSMV with half of the

children having a lower ICP than pre-suction. However a small percentage of children had prolonged recovery times after ETSMV with 20% (5/25) of them exceeding 20mmHg at five minutes (and 3/5 of these having poor six month outcomes).

No confounders in this study could be shown to affect maximal ICP or ICP rise. Although suctioning is a noxious procedure in this group of children, the effects seen in this study may have been offset by the manual ventilation procedure.

CHAPTER 7

RESULTS: TURNING THE CHILD (VIA A LOG-ROLLING APPROACH) WITH MODERATE TO SEVERE TBI

7.1 Introduction

This chapter presents the results of the effects of turning (via a log-rolling technique) in the moderate to severe head injured child and will be presented in a few parts. Turning the child (always done via a log-rolling technique) was the second most frequently recorded intervention, accounting for 18% of all measurement episodes overall. This occurs regularly (at least every 12 hours for pressure area care) but also for chest x-ray and sheet changes.

The first section presents the demographical data of this study group, the baseline data and the distribution of the outcome measures for analysis. The second part presents the change in the primary outcome measure (ICP) with turning, the percentage change from baseline and the results of this when stratified to high and low baseline ICP groups. The third part examines the correlation between variables during turning and the effect of confounders. The fourth part looks at recovery times after turning and the last part examines then mean arterial pressure changes during turning.

7.2 Demographics of the turning sample

Twenty-four children with moderate to severe closed head injury (who required ICP monitoring) were recruited over a three year period (January 2006 to December 2008) in a single centre. The demographics of the group are shown in Table 11.

This did exclude some children whose injury so catastrophic that ICP monitoring was not considered appropriate (n=9). It also excluded children under two years (n=2), children who had an open head injury (n=1) whose parents refused consent (n=1) and children after decompressive craniectomy (n=5). The mortality in the study sample was 8.3% 2/24 and this roughly correlates to the predicted PIM 2 score of 7%.

Table 11: Demographics of log-rolling sample (n=24)

Demographic	Mean (SD), Median (IQR) or percentage of group or 95% CI
Age	9.6 years (SD 4.4)
Gender	14/24 (58%) male
Post-resuscitation GCS	7 (range 3- 13)
Patients with GCS >9 who required ICP monitoring	5/24 (21%)
Mechanism of injury	Pedestrian RTA 10/24 (42%) Passenger RTA 7/24 (29%) Fall 5/24 (24%) Cyclist 1/24 (4%) Crushed 1/24 (4%)
Presence of other significant injuries	15/24 (62%) of these 11/15 (73%) limb fractures 7/15 (47%) lung/chest injuries
Neurosurgical intervention (excluding bolt and EVD)	9/24 (38%) of these, most common were: 44% Extradural, 33% decompressive craniectomy
Mean day 1 ICP mmHg	17.8 (SD 10.03)
PICU length of stay (Days)	5.2 (IQR 3 – 11.5)
Decompressive craniectomy	3/24 (13%) crossed over into DC group after 1st measurement done)
Absolute mortality	2/24 (8%)
Mean PIM 2 score	0.069 (95%CI 0.013 – 0.125)
PIM derived standardised mortality ratio (SMR) of group	1.20 (95%CI 0.146 – 4.35)
Six month outcome	18/22 (82%) mild-moderate disability (GOS 1 and 2) 4/22 (18%) severe disability (GOS 3)

The turning sample had one less child (n=24) thus table 11 is slightly different from the endotracheal suctioning group. The first turning intervention measured and recorded was used as the value per patient and this was undertaken in the first 36 hours of PICU admission. The mean baseline values pre-turning (in the whole group) are shown in Table 12.

Table 12: Mean baseline data prior to turning (n=24)

Baseline value	Mean	SD
ICP mmHg	14.3	5.6
MAP mmHg	86	16.3
CPP mmHg	71.9	14.2

This baseline data demonstrates again the fairly homogenous nature of the target sample (all moderate to severe closed head injured children) with high-normal mean ICP values pre-intervention (despite therapy).

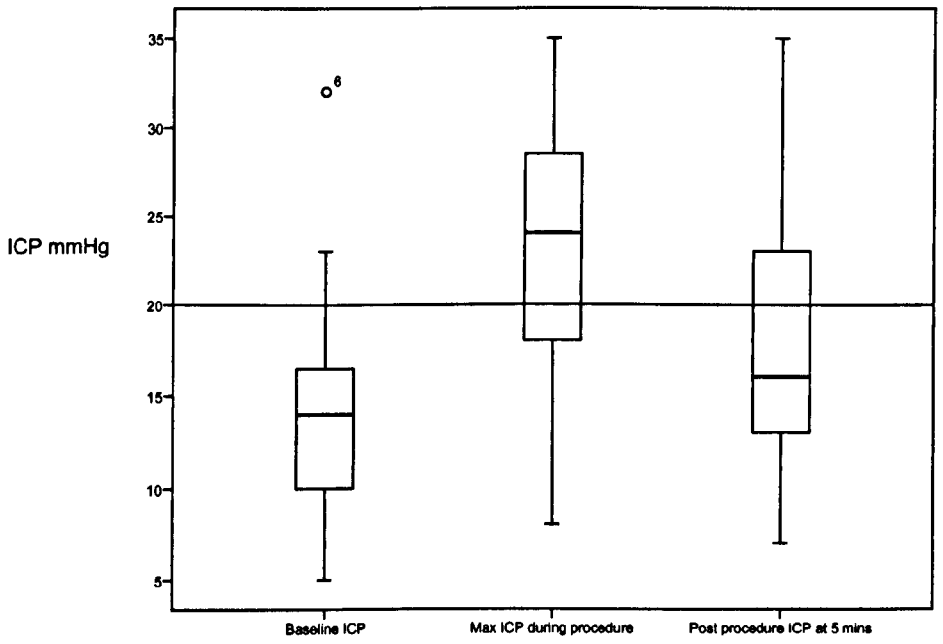
7.3 The distribution of data in the turning group

Upon examination of the data, the primary outcome measure (ICP at the three time points and ICP rise) were not all perfectly normally distributed, and as previously discussed in the suctioning chapter, this is essential in small samples (eg n=<30) if parametric tests are to be used. The distribution of the data are shown below:

Table 13: Dispersion of the data in turning dataset

		Baseline ICP	Max ICP during procedure	Post procedure ICP at 5 mins	ICP rise
N	Valid	24	24	24	24
	Missing	0	0	0	0
Mean		14.29	22.75	18.29	8.50
Median		14.00	24.00	16.00	9.00
Std. Deviation		5.637	7.617	7.788	5.217
Skewness		1.289	-.282	.744	.156
Std. Error of Skewness		.472	.472	.472	.472
Range		27	27	28	20

Baseline ICP is not normally distributed and if a box plot (Graph 38) is used to display this data, it becomes evident that one outlier (no.6 who subsequently died) is mostly responsible for this skewed distribution.



Graph 38: The effect of log-rolling on ICP (whole sample)

Parametric tests cannot be used on this data as it is, so this outlier (no.6) was removed (see the distribution in Table 14) upon statistical advice, to produce a more normal distribution of all primary outcome measures (albeit with a smaller sample of 23 children). The normality of this distribution was further confirmed by testing the sample for normal distribution using the Shapiro-Wilks test (which can be used for samples <50). Non significant p values for baseline ICP (p= 0.924), maximal ICP (p= 0.713), post ICP (p=0.216) and ICP rise (p=0.684) demonstrate that this data are more consistent with a normal distribution and thus parametric tests can be applied.

Table 14: Distribution of data with outlier removed

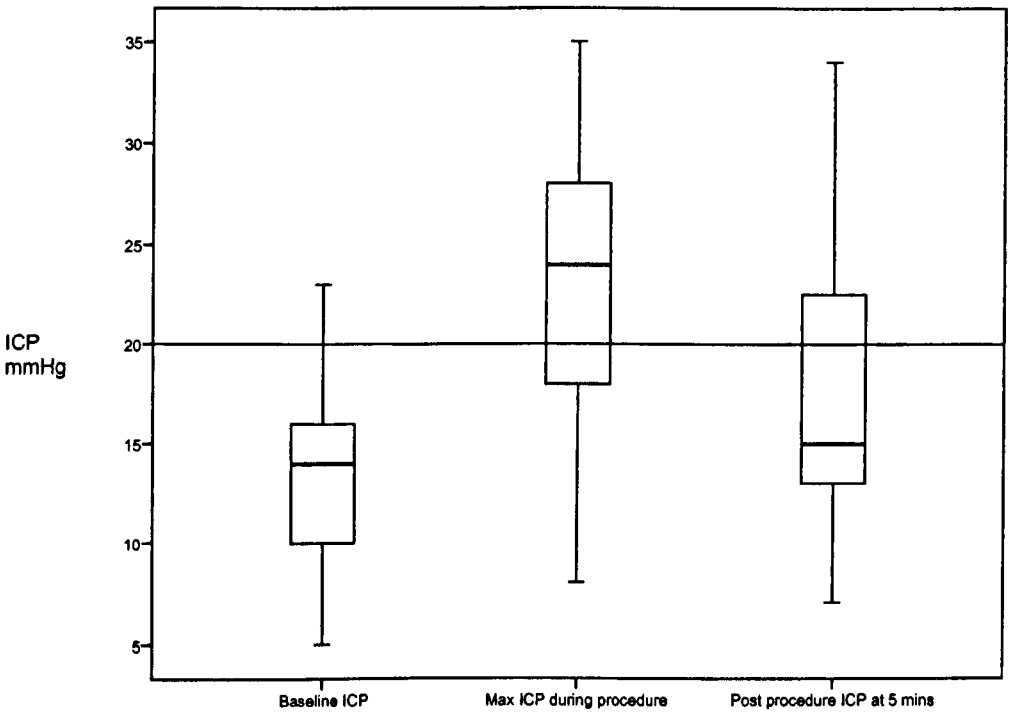
		Baseline ICP	Max ICP during procedure	Post procedure ICP at 5 mins	ICP rise
N	Valid	23	23	23	23
	Missing	0	0	0	0
Mean		13.52	22.22	17.57	8.74
Median		14.00	24.00	15.00	9.00
Std. Deviation		4.284	7.317	7.083	5.198
Skewness		.164	-.328	.723	.074
Std. Error of Skewness		.481	.481	.481	.481
Range		18	27	27	20

7.4 The effect of turning on the ICP response

The data were thus analysed with the outlier left in (non-normal distribution) using non-parametric tests and with the child removed (a normal distribution) using parametric tests. In terms of demonstrating a significant

change from baseline to maximal ICP and maximal ICP to post ICP, both sets of tests demonstrated statistical significance.

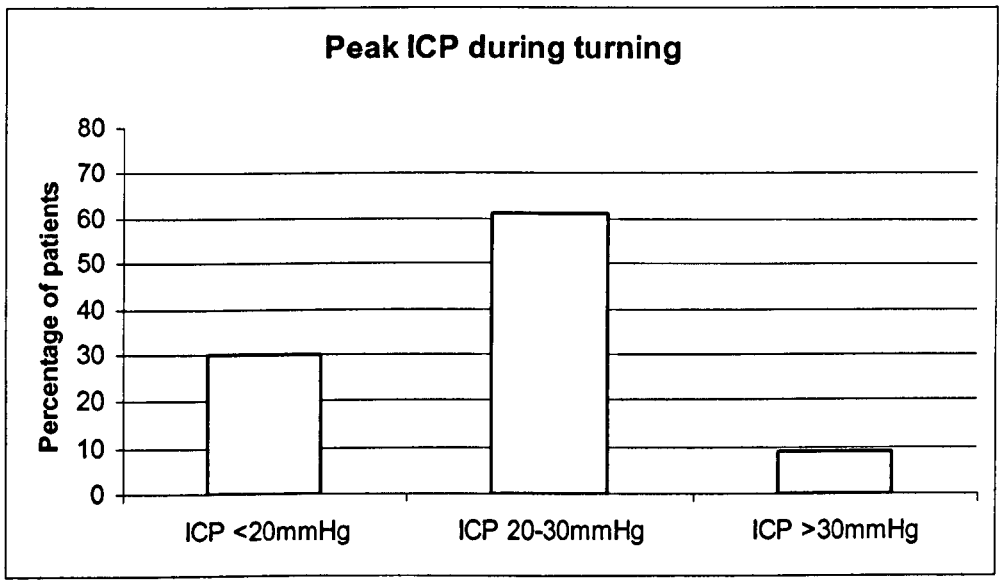
In the normally distributed sample of 23 children a paired t test showed a significant change from baseline ICP to maximal ICP (p=< 0.001 95% CI 6.4 - 10.9) and from maximal ICP to post procedure ICP (p=0.001 95% CI 7.1 - 2.2). This is graphically represented on Graph 39.



Graph 39: The effect of log-rolling on ICP (with outlier removed)

With the full sample of 24 (and non-normally distributed data) the non-parametric Wilcoxon rank test also demonstrated a statistically significant change from baseline ICP to maximal ICP (p=<0.001) and from maximal ICP to post procedure ICP (p=0.002). In addition to this statistical significance, this procedure also shows clinical significance in the majority of children in which the ICP exceeds 20mmHg, the recognised treatment threshold (Adelson et al 2003).

Furthermore, on a clinical level, descriptive statistics of the percentages of children who recorded maximal ICP values above 20mmHg, how many exceeded 30mmHg and their percentage change from baseline (as shown below) were calculated before using ANCOVA to correct for baseline.



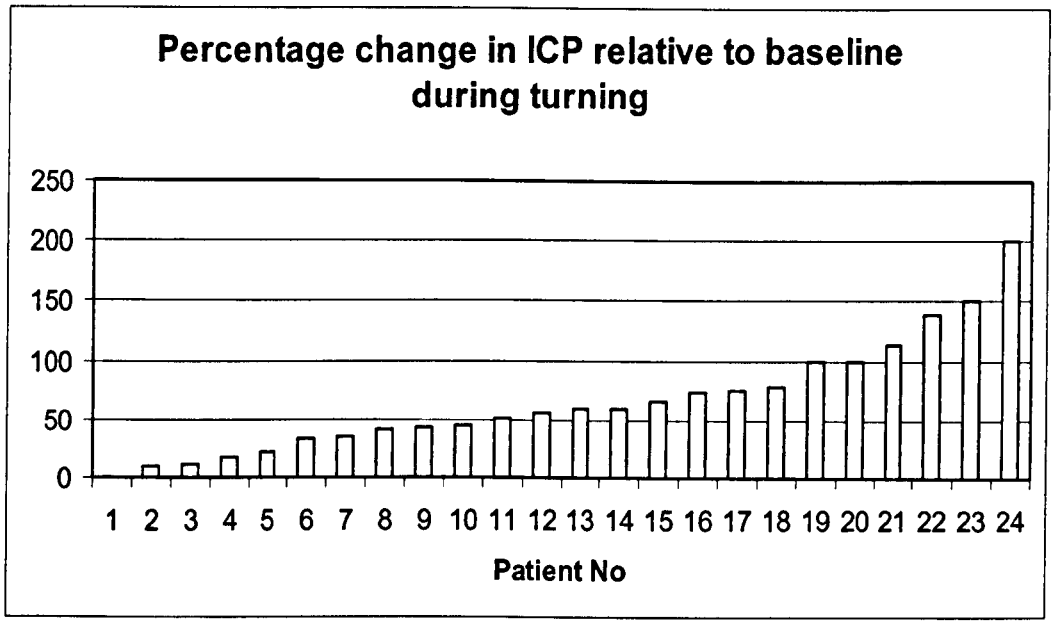
n=24

Graph 40: The Peak ICP during log-rolling

In these terms nearly 70% of children exceeded the 20mmHg ICP level during turning, with just fewer than 10% of them exceeding 30mHg.

The percentage change from baseline is calculated by: Peak ICP – Baseline ICP / Baseline ICP x 100.

Represented in Graph 41 is the percentage change from baseline during turning (sorted into ascending order).



Graph 41: Percentage change in ICP from baseline during log-rolling

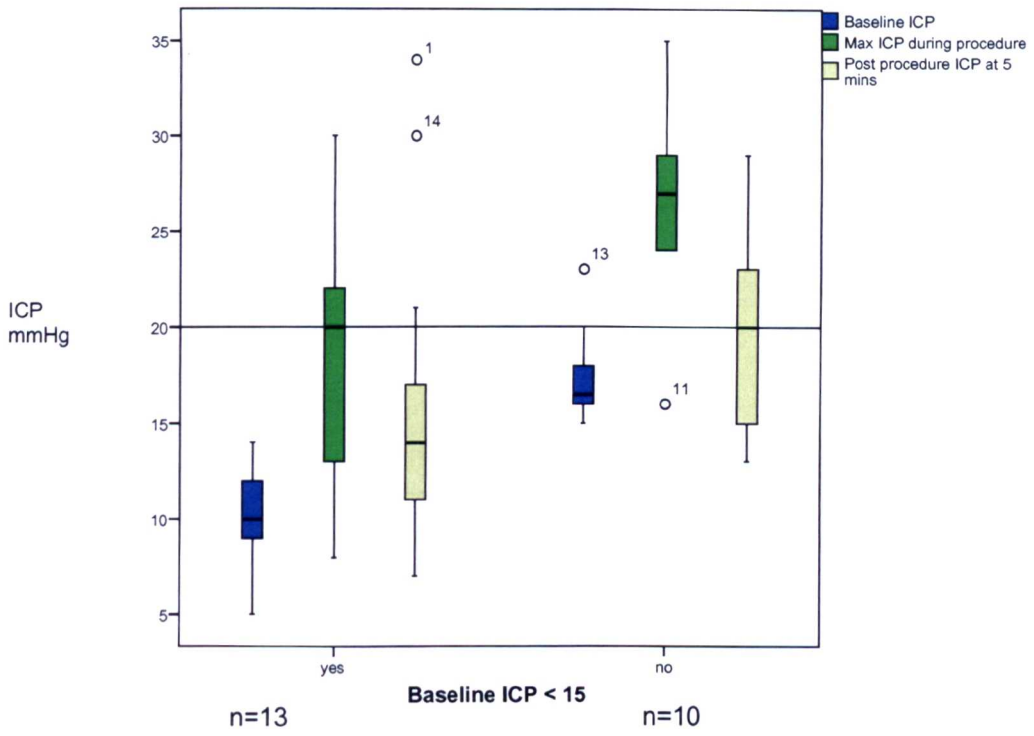
There was a 68% mean increase in baseline ICP during turning (range 0 – 200%), but it is notable that the baseline ICP was slightly lower than that prior to suctioning. Prior to turning 65% of children had a baseline ICP < 15mmHg with only 35% having a baseline ICP ≥15mmHg.

However the percentage change from baseline, is a very inefficient statistical test (Vickers 2001) because it allows no correction for baseline

ICP, thus further analysis (ANCOVA) was undertaken to correct for baseline imbalances (as recommended by Vickers 2001). With percentage change from baseline ICP (as the dependant variable) and the factor corrected for as baseline ICP were analysed using ANCOVA, a non significant p value ($p = 0.191$) showed that baseline ICP did not effect the percentage change from baseline.

7.5 Stratification by baseline ICP

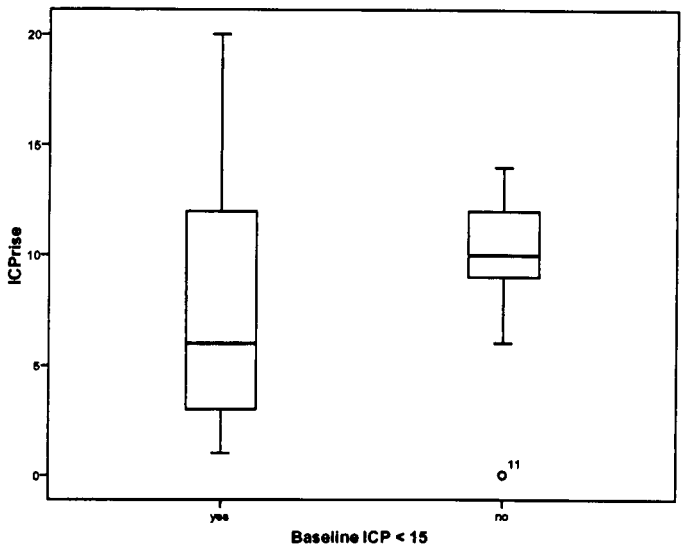
Previous research has demonstrated significant differences in patients with higher baseline ICPs (defined as ≥15mmHg) compared to those with lower baseline ICPs (< 15mmHg) during endotracheal suctioning (Garradd and Bullock 1986; Paratz and Burns 1993). Hence it was thought to be useful to examine these patients by a high/low stratification group with another known noxious stimuli (turning). The arbitrary stratification point of 15mmHg was used in these studies and also in the previous chapter with suctioning in children. With regard to turning it seems a reasonable stratification point as the median was 14mmHg in this sample (mean 14.3mmHg) (lower than with ETS). The box plot below shows the results when the children were stratified to high or low baseline ICP groups.



Graph 42: The effect on ICP during log-rolling stratified by baseline ICP

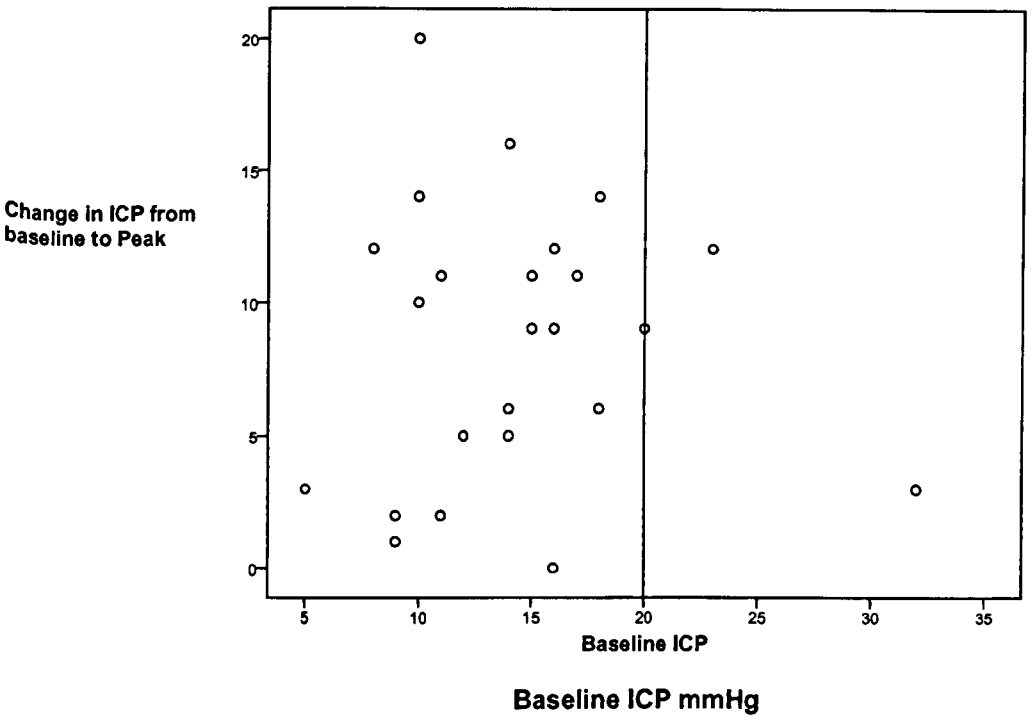
An independent sample t test showed that only the maximal ICP was significantly different between the groups (p=0.007 95% CI 2.5 - 13.4), with post procedure ICP not showing significant results (p=0.191).

When the ICP rise was examined, it also showed no significant difference between the two groups (p=0.604) and this can be seen graphically on the box plot below.



Graph 43: The effect on ICP rise in children stratified by baseline ICP

Further analysis to examine the relationship between the change in ICP (from baseline to maximal) and the baseline ICP (Graph 44) did not reveal any clear relationship in these two variables. However it shows that unlike with suctioning no children had reductions in their ICP during the procedure, suggesting that these reductions during suctioning may have been related to the manual ventilation procedure.



Graph 44: The change in ICP from baseline to maximal compared to the baseline ICP during turning

It may be expected if the numerical baseline value (ICP) is higher, then a higher maximal ICP would result (if the intervention produced an effect in a linear manner) and this is clearly demonstrated with turning, in terms of maximal ICP.

7.6 The correlation between variables during turning

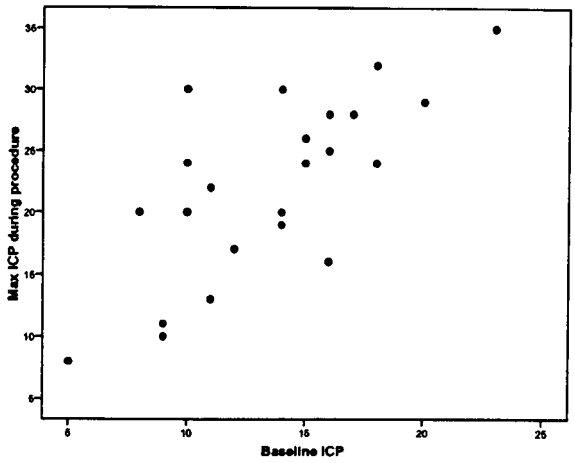
The correlation analysis below presents the normally distributed variables in 23 children using Pearson's correlation coefficient, (which assumes normally distributed data) (see a summary of the analysis in Table 15).

Normality of other variables presented was confirmed by the Shapiro-Wilks test. The time taken for the intervention was transformed to a normal distribution, but minutes to recovery was not normally distributed and was unable to be transformed to a normal distribution so the non-parametric test Spearman's rank correlation coefficient was used to determine correlation with this variable (Bland 2003 p.220). Only normally distributed variables with at least moderate correlation (an r value >0.4 Harris and Taylor 2004) are shown on subsequent scatter plots.

Table 15: Correlation table for log-rolling

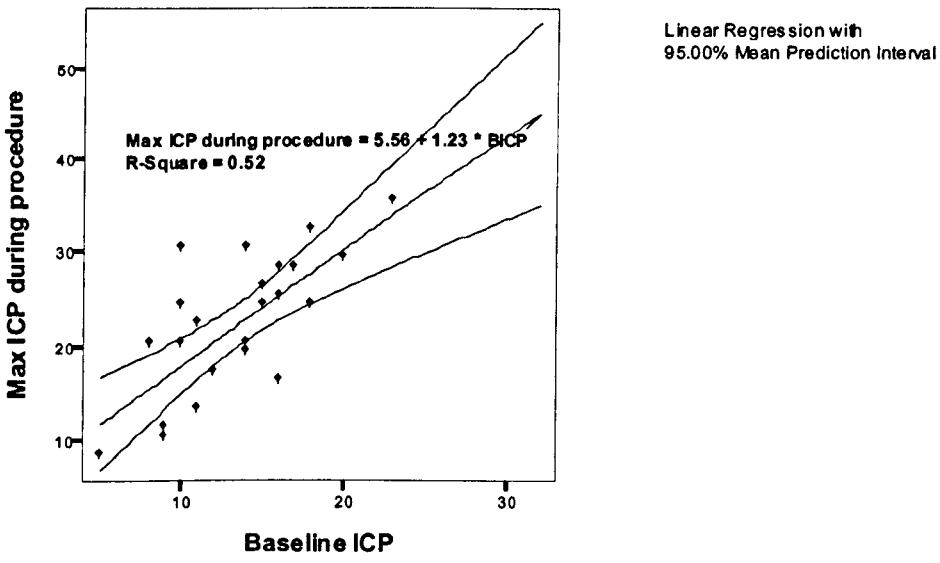
Factors examined	r value or correlation coefficient	p value
Baseline ICP to Maximal ICP	0.721	***0.001***
Baseline ICP to ICP rise	0.211	0.335
Maximal ICP and post procedure ICP (at 5 minutes)	0.734	***<0.001***
Baseline ICP and corrected PaCo2	-0.53	0.816
Log of time taken for procedure and maximal ICP	0.155	0.480
Log of time taken for procedure and ICP rise	0.375	0.078
ICP rise to post procedure ICP	0.572	***0.004***
Time taken for procedure and minutes to recovery	*0.550*	***0.012***
Baseline ICP and minutes to recovery	0.277	0.224
Maximal ICP and minutes to recovery	0.598	***0.004***

The relationship between baseline ICP and maximal ICP was examined in more detail graphically by representing this on a scatter plot below.



Graph 45: Correlation between baseline ICP and maximal ICP during log-rolling

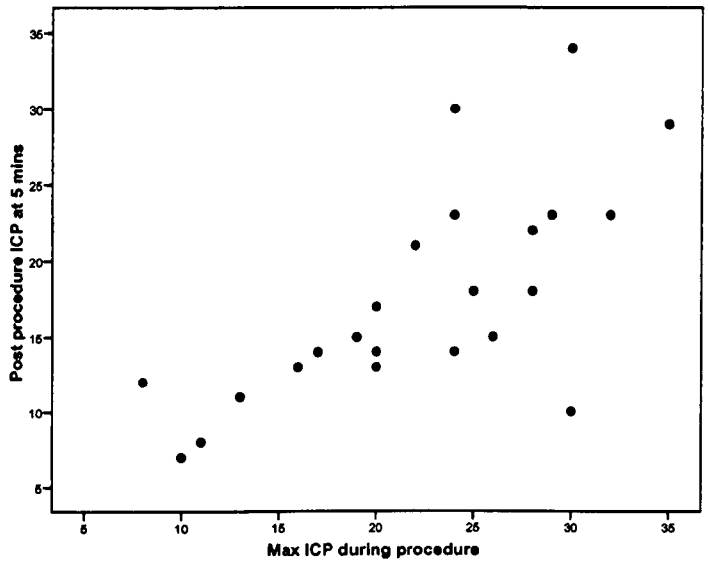
This was found to be highly correlated (r 0.721; p=<0.001), the extent of which can estimated by the regression model below.



Graph 46: Linear regression model between baseline ICP and maximal ICP

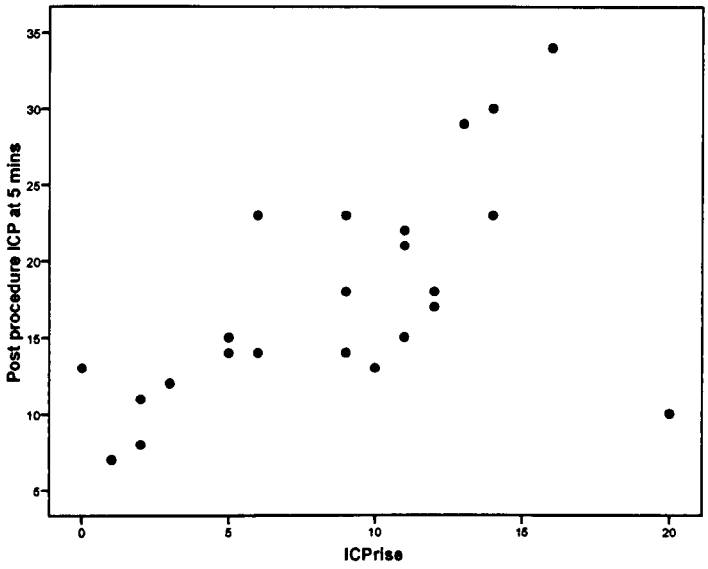
This predicts that the maximal ICP with turning will be ~6mmHg above the child's baseline ICP. The ICP rise was not significantly correlated (p=0.335).

Next the relationship between maximal ICP during turning and post procedure ICP was examined. The maximal ICP also correlated significantly with the post procedure ICP (r 0.734; p=<0.001).



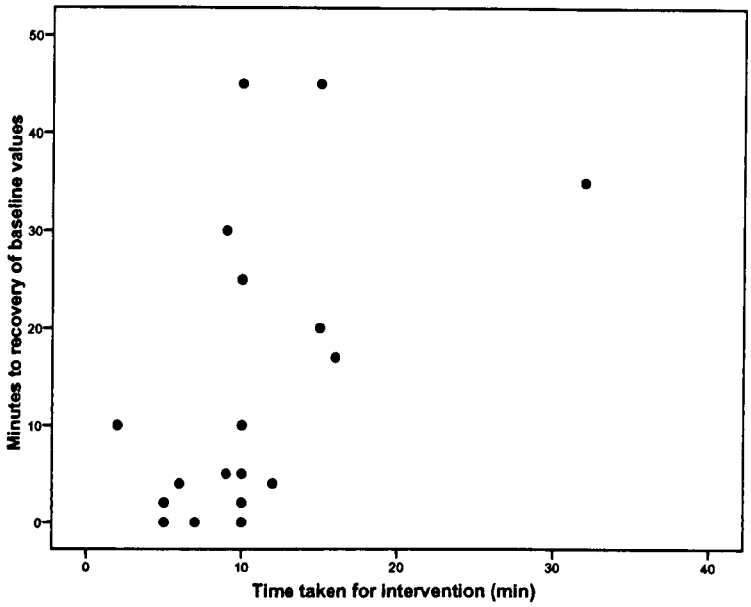
Graph 47: Correlation between maximal ICP and post procedure ICP during log-rolling

The ICP rise was also significantly correlated with the post procedure ICP (r 0.572; p=0.004). This can be seen on Graph 48.



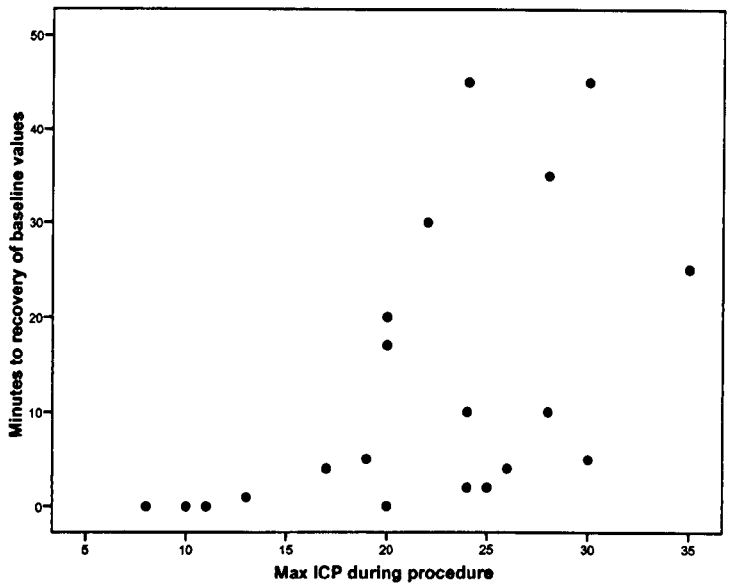
Graph 48: Correlation between ICP rise and post procedure ICP during log-rolling

The relationship between the time taken for log-rolling and time to recovery was next examined. This showed a significant correlation (r =0.550 p = 0.012) (see graph 49).



Graph 49: Correlation between time taken for log-rolling and minutes to recovery

Lastly the maximal ICP achieved during log-rolling and the minutes to recovery was examined. This showed a significant correlation (r 0.598 p=0.004) see graph 50.



Graph 50: Correlation between maximal ICP during log-rolling and minutes to recovery

We again examined the known confounder of carbon dioxide level (PaCo2) with baseline ICP. However as with suctioning this was not significantly correlated with baseline ICP (p=0.393), this is again probably because the EtCo2 level for the study sample was fairly narrow in the low-normal range with a mean of 36.6 mmHg (SD 4.1).

7.7 The effect of confounders during turning

As discussed previously there are multiple therapies that are potential confounders in this study. Table 16 shows the usage of confounders in the study population before and during turning. Despite acting as confounders, many of these were consistently applied across the whole group: the degree

of elevation of the head (all around 10-15° elevated), PEEP (virtually all ≤ 5cm H_20) and the presence of neck compression by a rigid cervical collar (100%). Other confounders such as the use of propofol (n=2) and hyperosmolar agents (n=4) were used rarely before this intervention. All children received continuous infusions of standard doses of an opiate and a sedative. Twenty two out of twenty three children (95%) received morphine at 40 micrograms/kg/hour and one child received fentanyl. Seventeen children (74%) received a midazolam infusion as their sedative at 100 – 200 micrograms/kg/hour; four were on thiopentone, two on propofol and one on both midazolam and thiopentone for sedation. An advantage when studying the effects of turning is that two major confounders were removed: the use of manual ventilation of the child (which can affect the PaCo2 and yet cannot be reliably measured) and the use of endotracheal lignocaine. In addition, even in the children with a ventricular drain in situ, this would be kept closed during turning.

Table 16: Presence of confounders prior to log-rolling

	Mean or (% of group)	SD
Head of bed elevation °	11.7	3.8
PEEP cmH_20	5.3	1.3
Core temperature° C	36.3	1.1
Cervical collar present and tight	100% of sample	
Baseline EtCo2 level mmHg	36.6	4.1
Baseline Spo2 (%)	99.5	1.3
Received hyperosmolar agents in previous 4 hours	4/23 (17%)	
Presence of painful other injuries	15/24 (62%) 11/15 limb fractures 7/15 lung/chest injuries	
EVD present	3/23 (13%) but all clamped	
Pre-bolus of sedation	20/23 (87%)	
Pre-intervention COMFORT score	0nly available in 3 children Mean score 14.6	
Received NMB	18/23 (78%)	
On propofol infusion	2/23 (9%)	
On thiopentone infusion	4/23 (17%)	
On continuous infusion of opiate + sedative	23/23 (100%)	
On inotrope infusion	17/23 (74%)	
Time taken for turning (mins)	10.2	6.0

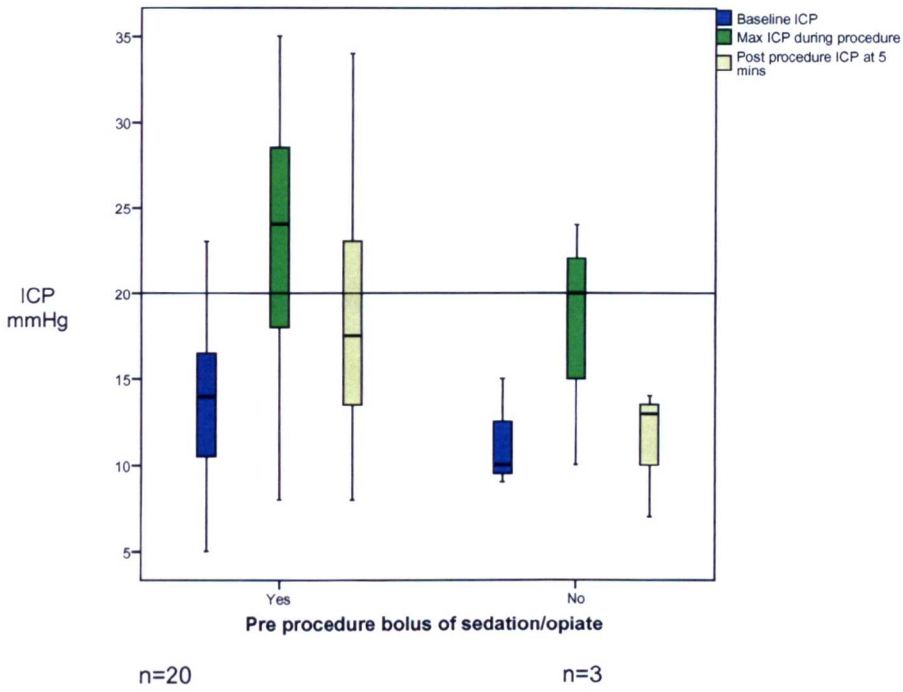
The effects of the main confounders are presented in box plots that follow. An independent sample t test was used to compare the means between the two groups, however on occasions the groups were very unevenly distributed (and small) because again this was an observational study, which did not attempt to control the intervention in any way, but rather to study practice 'as it is'.

7.8 The effect of a pre-procedure sedation bolus

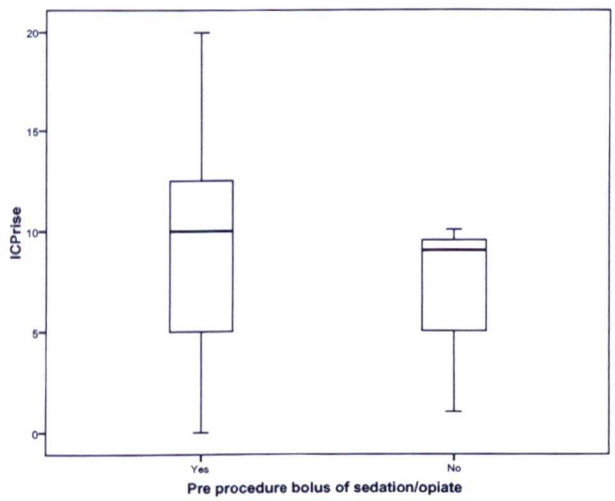
As shown above 87% of children received an additional intravenous bolus of a sedative and/or opiate analgesic drug prior to the procedure. This was

predominantly midazolam (as sedation) and morphine (as the opiate). This was prescribed on an 'as required' basis and thus was administered according to the nurse's clinical judgement. If the children were not receiving paralytic agents then their level of sedation may be quantified using a validated tool, the COMFORT score, however only three children had a COMFORT score recorded. Graphs 51 and 52 show the effect of a pre-bolus of sedation on the ICP variables and ICP rise during turning.

A bolus of sedation did not reduce the maximal ICP, or the ICP rise, in fact this was slightly higher in the children who had received the sedation bolus. There was a significantly higher post procedure ICP ($p=0.05$ CI [-0.004] – 14.3) in the children who received the sedation bolus, but the baseline ICP ($p=0.311$), ICP rise ($p=0.501$) and maximal ICP ($p=0.367$) were not significantly different between the children who received sedation and those who did not.



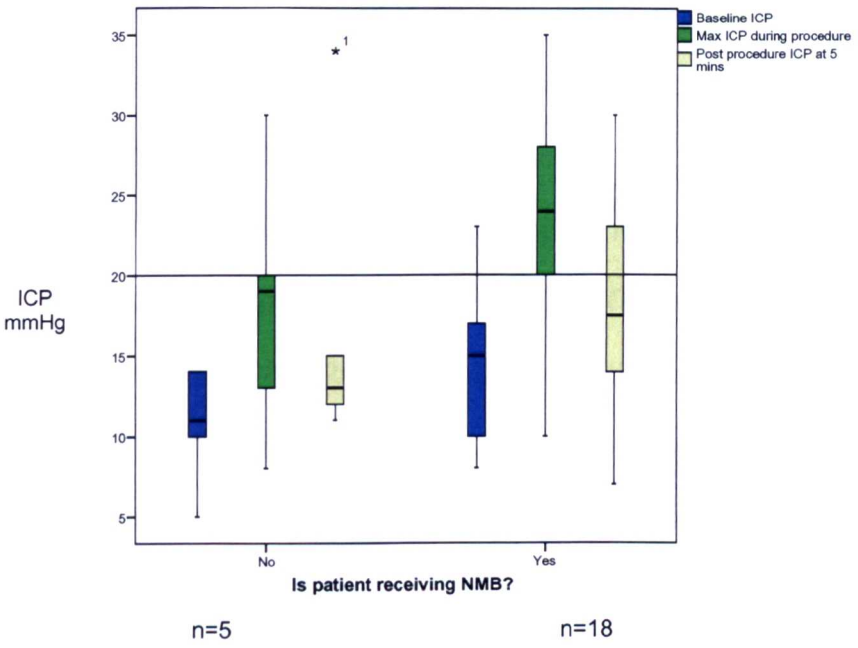
Graph 51: The effect of a sedation bolus on the ICP response during log-rolling



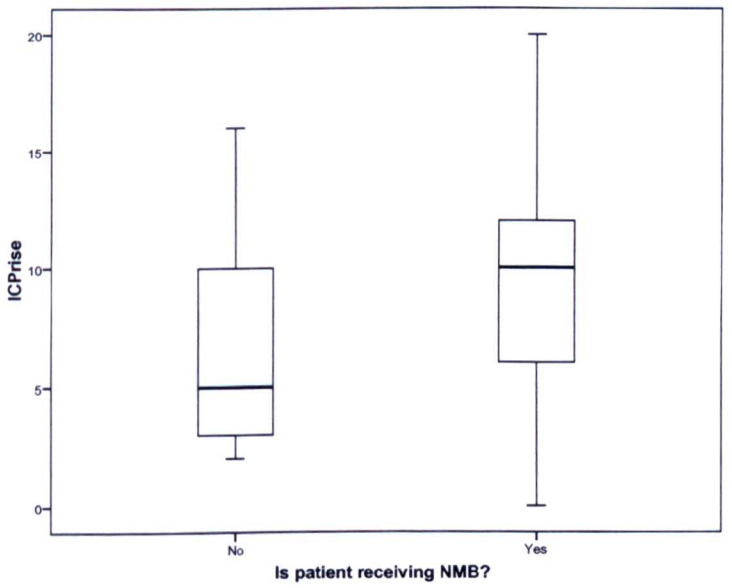
Graph 52: The effect of a sedation bolus on the ICP rise during log-rolling

7.9 The effect of neuromuscular blockade

During this intervention 78% of children were muscle relaxed (using either an atracurium infusion or intermittent vecuronium boluses) and none who were receiving these drugs coughed or moved during turning. As mentioned previously children should be heavily sedated whilst on these drugs to prevent the potential distress of being 'awake under the paralysis'. This is slightly concerning with regard to the increased ICP response seen previously in the children who had a sedation bolus. The Graphs 53 and 54 show the overall effects of NMB on ICP variables. These show similar effects to that of a pre-procedure sedation bolus. However, despite showing the concerning feature (on the box plots) that the children who received NMB demonstrated a higher ICP rise and maximal ICP values, none of these showed statistical significance: ICP rise ($p=0.467$), maximal ICP ($p=0.149$), baseline ICP ($p=0.110$).



Graph 53: The effect of NMB on the ICP response during log-rolling



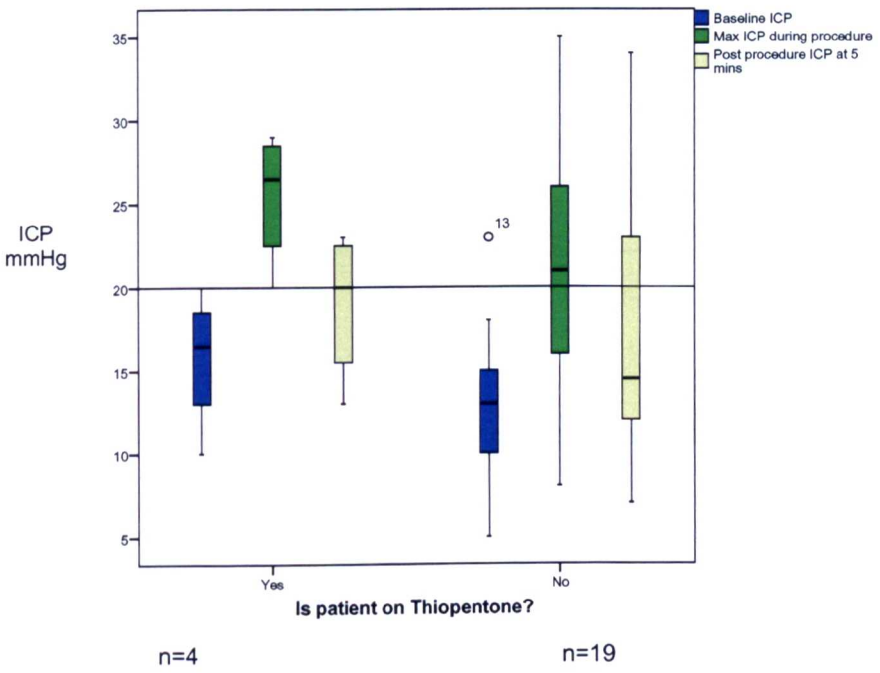
Graph 54: The effect of NMB on the ICP rise during log-rolling

Despite the lack of statistical significance, these findings are clinically concerning, suggesting that some of these children may be too lightly sedated underneath the paralysing agent. However we undertook additional analysis of MAP changes (as a potential physiologic indicator of inadequate sedation or pain Franck et al 2000). This showed no significant difference in maximal MAP between the muscle-relaxed and non muscle-relaxed children, which does not support the view that some children may be less sedated. However vital signs changes although giving some idea of sedation level, can also be affected by the pathology and also by ICU therapies (eg inotropic infusions) and Trope et al (2005) found HR and BP were poorly correlated with the sedation level in children.

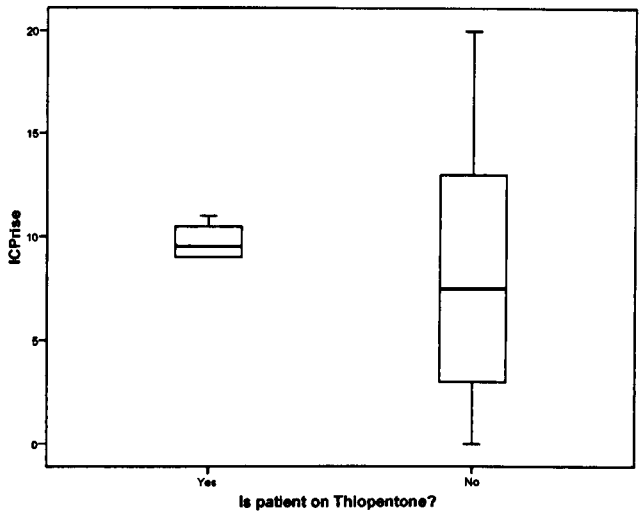
7.10 The effect of Barbiturates (thiopentone)

In the turning group 17% (4/23) of children received thiopentone. The box plots that follow show the overall effects of this drug on ICP variables. These box plots suggest some differences between the children who received thiopentone and those who did not. The baseline ICP, maximal ICP and post procedure ICP all appeared higher in the children who were receiving barbiturates, however none were significantly different on independent t test: baseline ICP ($p=0.24$); maximal ICP ($p=0.299$); ICP rise ($p=0.638$); post ICP ($p=0.668$). The trend of escalating ICP from baseline to peak to post ICP seen in these children during ETSMV is not present during turning, and they appear to show the classical response of a peak

rise in ICP during the intervention and some recovery (a reduction) by the post procedure ICP (at five minutes).



Graph 55: The effect of thiopentone on the ICP response during log-rolling

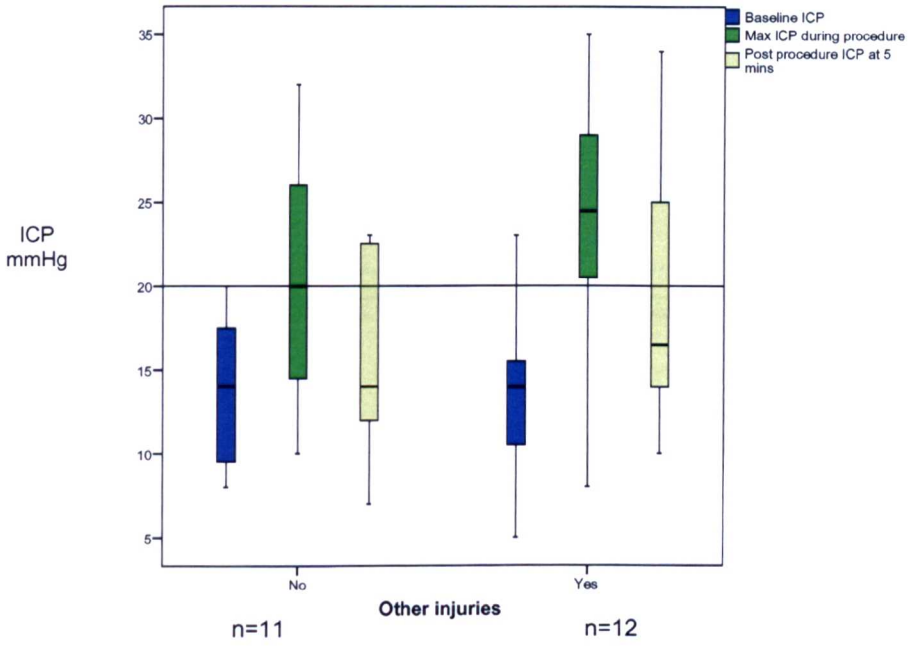


Graph 56: The effect of thiopentone on ICP rise during log-rolling

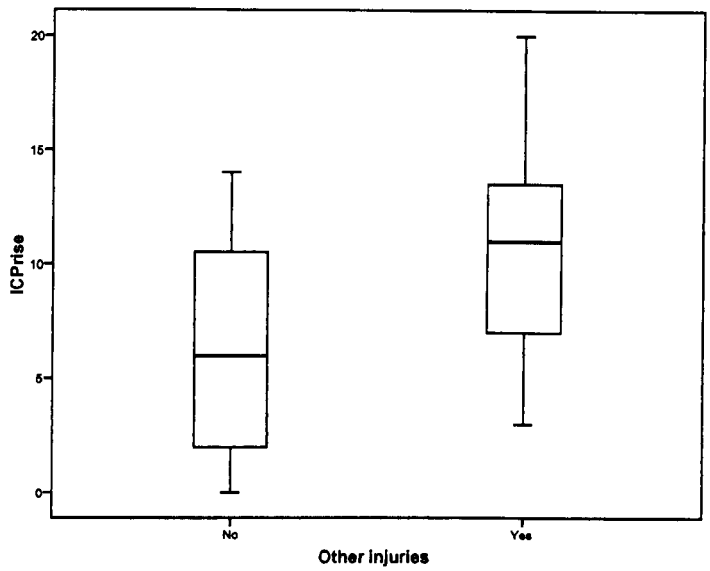
7.11 The effect of having other (painful) injuries

From the description of the log-rolling procedure (in the methods chapter) it might be assumed that children who have other significant injuries (in addition to their moderate to severe head injury eg skeletal fractures) may experience more pain with turning. Sixty-two percent of children in the turning group had other significant injuries (see Table 11), the vast majority of these being limb/skeletal fractures (73%) which undoubtedly with moving can produce pain and there is some evidence to suggest that pain may in turn increase ICP (Bellieni et al 2003) (along with other parameters such as blood pressure and heart rate). The majority of these children were muscle relaxed and so behavioural clues are lost.

We wanted to examine if there were any differences in this group of children with other injuries. The graph that follows show the effect on ICP variables with turning in the two groups of children.



Graph 57: The effect of having other (painful) injuries on the ICP response during log-rolling



Graph 58: The effect of having other (painful) injuries on the ICP rise during log-rolling

The results showed no statistical difference between the two groups in terms of baseline ICP (p=0.906), maximal ICP (p=0.231) or post procedure ICP (p=0.267), however a there was a trend towards significance (p=0.061 CI 0.21 - 8.2) in the ICP rise, in the children who had other injuries suggesting a higher ICP rise during turning. This might suggest that this group may experience more pain with turning, but this cannot be substantiated from our data.

Multivariate linear regression analysis

Multivariate linear regression analysis was also done to examine the effects of previously identified significant confounders on the ICP response to turning. Only confounders with a p value of 0.05 and below from previous

analysis were put into the regression model. The p value was Bonferroni corrected to a significance level of p = 0.025. Only one variable, baseline ICP, was considered as having a potential effect on maximal ICP, but this effect was demonstrated clearly on the linear regression analysis and so was not put into the multivariate regression model. Both maximal ICP and ICP rise showed a significant relationship with post procedure ICP and were put into the model. Lastly the time taken for the turn and maximal ICP had a significant relationship with minutes to recovery (see table 17).

A summary of the linear regression analysis is presented below.

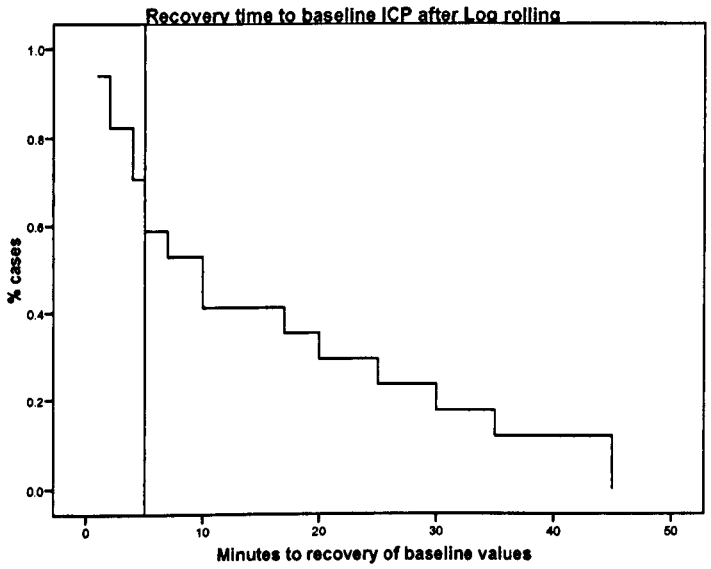
Table 17: Significance of confounders (using multivariate linear regression) during log-rolling (n=23)

For dependent variable of post procedure ICP			
Maximal ICP	0.703	0.729	***0.019***
ICP rise	-0.041	-0.030	0.917
For dependent variable of minutes to recovery			
Maximal ICP	0.631	0.315	0.123
Time taken for procedure	1.139	0.475	***0.025***

7.12 Recovery times after turning

In addition to ICP changes and maximal ICP, the time taken to recover after turning was examined. Two separate issues were again examined here 1) the time taken to return to the child's baseline ICP and 2) how many children had an ICP that exceeded 20mmHg at five minutes post procedure. As described in the previous results chapter, this five minute time period of tolerating ICP values greater than 20mmHg is clinically recognised as being significant (Adelson et al 2003) as prolonged intracranial hypertension has been shown to contribute to secondary injury.

Time to event analysis (using Kaplan Meir curves) was used to analyse the recovery times in the sample of 21 children (three had missing data for this variable). This analysis does not assume, nor require, a normal distribution. When comparison was made between groups in terms of recovery times the log rank test was used (Bailar and Mosteller 1992 p 287). The median time to return to baseline ICP after turning was five minutes (Range 0 – 45 minutes) (95% CI 0.51 – 9.4 minutes for median). Below is the Kaplan Meir curve to show the recovery times in the whole sample after turning. In a small number of children (4/24), the post procedure ICP was lower than their baseline ICP.

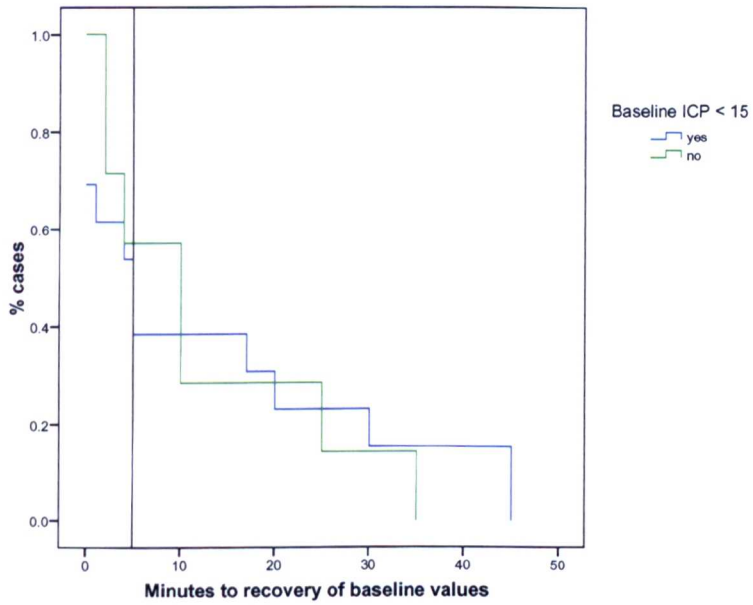


n=21 (3 missing data)

(95% CIs for median 0.51 – 9.4)

Graph 59: Recovery time to baseline ICP after log-rolling

This Kaplan Meier curve shows that nearly 60% of children had not recovered their baseline ICP at five minutes after the procedure, and 83% of patients had higher ICPs than at their baseline pre-turn. The next Kaplan Meir curve demonstrates the differences between the high and low baseline ICP groups in terms of recovery time.



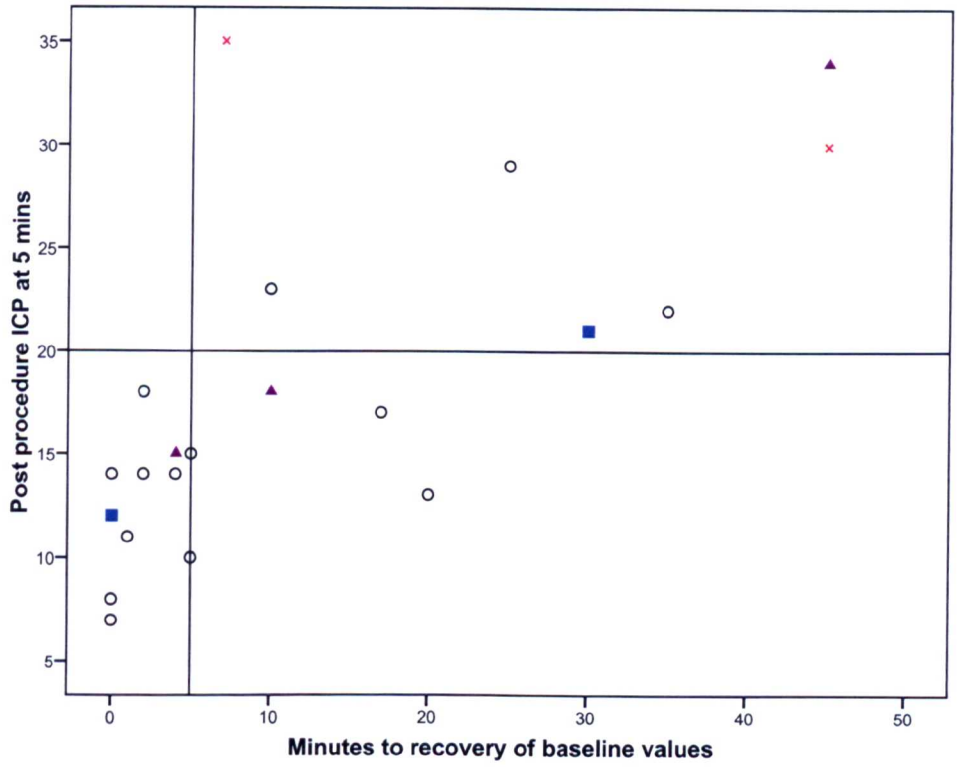
n=21 3 missing data

Graph 60: Recovery times to baseline ICP after log-rolling by baseline ICP

This shows no significant difference in recovery times (to baseline ICP) between the high and low baseline ICP groups after turning (p=0.725).

We now examined how many of these children actually had post procedure ICP values (five minutes after the procedure) higher than 20mmHg. Seven out of twenty-one children (33%) patients had ICP values greater than

20mmHg at 5 minutes after the turn and 4/7 (57%) of these children had poor six month outcomes or subsequently required a decompressive craniectomy (see Graph 59).



n=21 (time missing in 3)

Graph 61: Relationship between post ICP, recovery times and 6 month outcomes after log-rolling

Key: cross = died; triangle = decompressive craniectomy; square = severe disability (GOS 3) circle = mild-moderate disability (GOS 1 or 2).

This graph shows that 7/21 (33%) of children had ICP values that exceeded 20mmHg for longer than five minutes after turning. The six month outcomes

are highlighted on this graph. Four out of seven (57%) of the children in this potential secondary injury zone had a poor outcome. However there were also poor outcomes in some of the children who did not have raised ICP and although being important, ICP is not the only factor affecting patient outcome.

7.13 Changes in mean arterial pressure (MAP) and cerebral perfusion pressure (CPP) during turning

As discussed in the previous results chapter, the mean arterial pressure (measured and displayed continuously in intensive care) has age-dependent normal parameters, of which the minimal acceptable level ranges from 40mmHg in the term neonate to 60mmHg in the adult patient (Macnab et al 1999). The cerebral perfusion pressure (CPP) is a derived value calculated by the formula CPP = MAP – ICP and this also has suggested age-dependent targets, cited as: 2-6 years > 53mmHg; 7-10 years > 63 mmHg and 11-16 years > 66 mmHg (Chambers et al 2005).

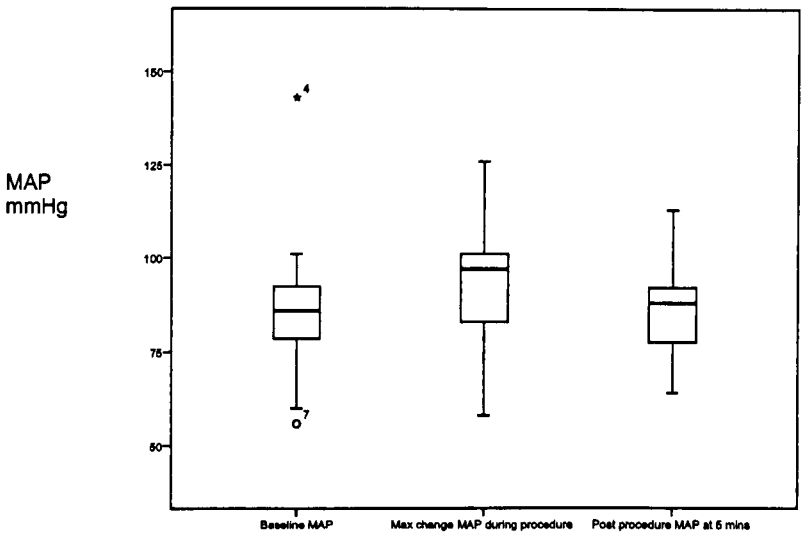
Given the clinical necessity of ensuring a stable and consistent above-target CPP, the majority of children (74% 17/23 in this group) were supported by an inotrope infusion (virtually all nor-adrenaline). This infusion is adjusted continuously by the bedside nurse to keep the CPP above the target range. This makes the use of MAP and CPP changes as an outcome measure, not entirely related to the child, but also to iatrogenic manipulation. In other

published papers (in adults) the majority of patients are not on inotropes and hence MAP can be used as an outcome measure. The fact the MAP is manipulated to achieve a desired CPP also make CPP (a derived value) changes not that a useful outcome measure. Therefore these two parameters were not useful primary outcome measures in this study. However we did note that both CPP and MAP did change (in line with the formula CPP = MAP - ICP) during turning but their change was neither clinically nor statistically significant. MAP rose slightly partly compensating for this ICP rise and thus the CPP fell slightly.

The distribution of the MAP data in the turning group was however examined (in the total sample of 24) both on box plot (below) and statistics (also below) and it was obvious that one child was an outlier (no.4), who affected the normal distribution of the baseline ICP data.

Table 18: Distribution of MAP data in turning dataset

		Baseline MAP	Max change MAP during procedure	Post procedure MAP at 5 mins
N	Valid	24	23	24
	Missing	0	1	0
Mean		86.04	93.17	85.88
Std. Deviation		16.276	15.435	11.855
Skewness		1.522	-.206	.016
Std. Error of Skewness		.472	.481	.472
Range		87	68	49

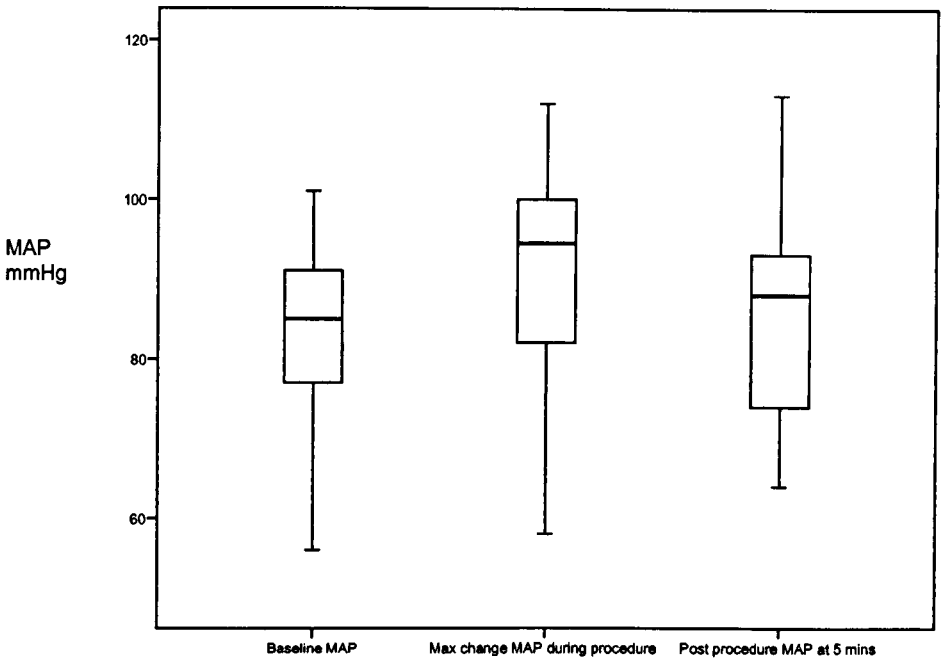


Graph 62: The effect of log-rolling on MAP (whole sample)

This outlier (patient 4) was removed and the data appeared more normally distributed.

Table 19: Distribution of MAP data in turning dataset with outlier removed

		Baseline MAP	Max change MAP during procedure	Post procedure MAP at 5 mins
N	Valid	23	22	23
	Missing	0	1	0
Mean		83.57	91.68	85.91
Std. Deviation		11.094	13.998	12.120
Skewness		-.945	-.613	.005
Std. Error of Skewness		.481	.491	.481
Range		45	54	49



Graph 63: The effect of log-rolling on MAP with outlier removed

If MAP was going to be used as an outcome measure then a test of normality (the Shapiro-Wilks test) would have been applied to confirm this. However, because of these variables not being used as outcome measures no further analysis was undertaken.

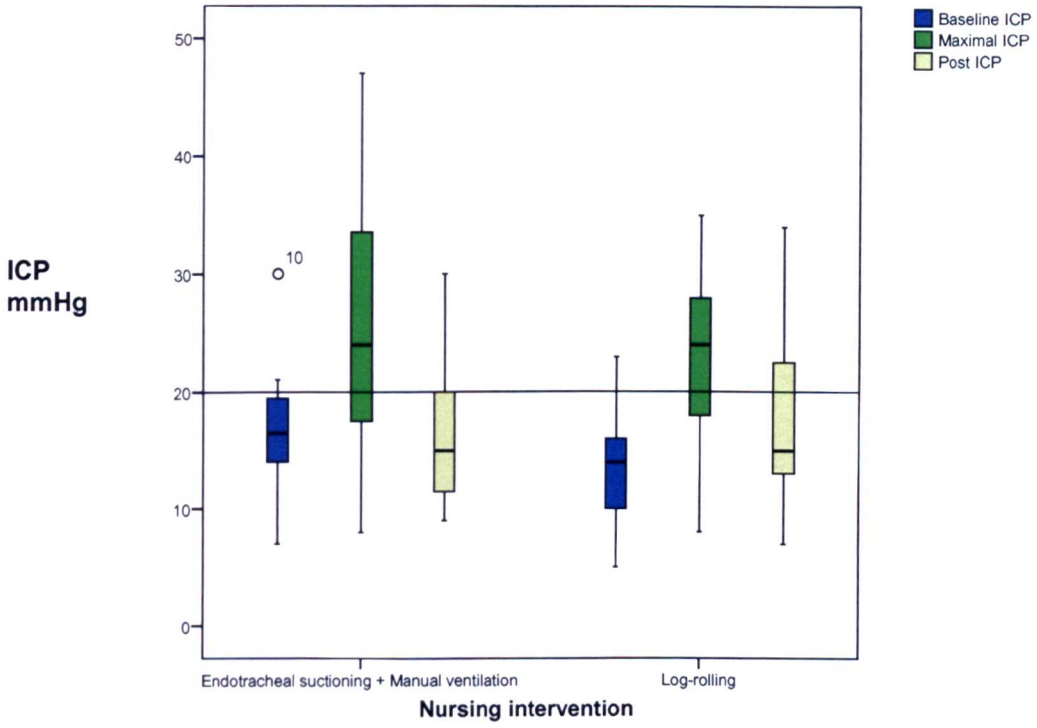
7.14 The interaction between ICP, MAP and CPP during turning

During turning the mean arterial pressure changed from 85.3mmHg (at baseline) to 92.8mmHg during the procedure and by five minutes post procedure this had reduced back to 85.2mmHg. At the same time mean ICP changed from 13.5mmHg (at baseline) to 22.2mmHg during the procedure and was 17.5mmHg at five minutes post procedure. Correspondingly, the

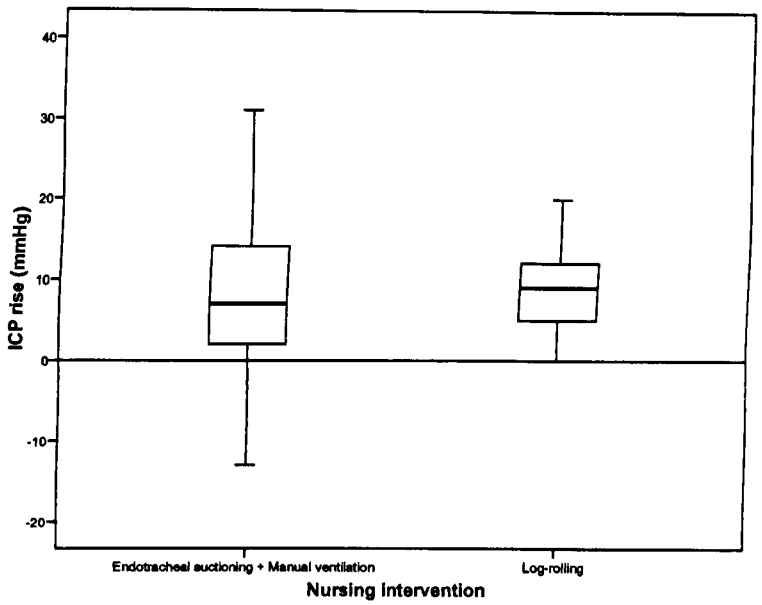
CPP at baseline was 72mmHg fell slightly to 71.4mmHg and was 67.8mmHg five minutes post procedure. Therefore we have demonstrated that the ICP changed significantly more than MAP, which partly compensated for the ICP rise, resulting in only a minimal reduction in CPP.

7.15 Comparison of endotracheal suctioning and turning as noxious interventions in head injured children

This section will briefly compare the effects on the ICP response of these two noxious nursing interventions. Graphs 64 and 65 show the comparative effect of both interventions on the ICP response.



Graph 64: Comparison of endotracheal suctioning and log-rolling as on the ICP response in head injured children



Graph 65: Comparison of the ICP rise between endotracheal suctioning and turning in head injured children

It is clear that both interventions produce significantly elevated ICP during the procedure in the majority of children but the ICP variability during the procedure is greater during endotracheal suctioning (with some reductions in ICP along with elevations in the majority of children).

7.16 Conclusion and summary of results of turning (via a log-roll approach)

In summary, turning children with moderate to severe TBI (via a log-rolling approach) does produce both clinically and statistically significant changes in their ICP from baseline, with 70% exceeding 20mmHg during the turn and only a small number (<10%) exceeding 30mmHg. The children with higher

baseline ICPs (≥15mmHg) had significantly higher maximal ICPs (but like with suctioning), but there was no difference in the degree of ICP rise between the groups, suggesting this intervention produces a linear rise in ICP. With turning the baseline ICP correlated highly with the maximal ICP and the maximal ICP could be predicted to be ~6mmHg above the child's baseline, which may be helpful to clinicians when planning these children's care. Maximal ICP was also highly correlated with post procedure ICP (unlike with ETSMV where there was only a slight correlation). Furthermore the greater the time taken for turning the longer the child took to recover their baseline ICP. These more significant findings observed with turning may be more reliable as there were four less confounders during the turning interventions, including the significant one of manual ventilation.

No other confounders however were shown to affect ICP response during turning. It is notable that the response seen in children receiving barbiturates during ETSMV (the escalating rise in ICP from baseline to peak to post) was not observed during turning.

After turning 83% of children had higher ICPs than before the turn (which was different to suctioning where the majority had lower ICPs after ETSMV) and overall took longer to recover their baseline ICP than with suctioning. However this may be a time-dependent issue as the turning procedure took slightly longer than ETSMV (median ten minutes for turning versus seven minutes for ETSMV). 60% of children had not recovered their baseline ICPs at five minutes after turning and more children had ICP values greater than

20mmHg for more than five minutes (compared with ETSMV) and of this group, four out of seven had poor six month outcomes.

CHAPTER 8

RESULTS: OTHER NURSING INTERVENTIONS IN CHILDREN WITH MODERATE TO SEVERE TBI

8.1 Introduction

This chapter presents the results of the effects of other nursing interventions (eye care, mouth care and washing the child) in the moderate to severe head injured child and will be presented in sections with each intervention discussed separately. Eye care was the most commonly performed of these interventions accounting for 17.5% of all the recorded episodes. Mouth care episodes accounted for 12.6% of the overall recorded measurements with washing only accounting for 3.2% of measurement episodes. There were fewer of these intervention episodes compared to suctioning and turning, and because of this the measurement of these interventions extends to those undertaken in the first 72 hours after the child's admission to PICU. This is still in the reported 'hyper acute phase' of head injury, thus ensuring consistency in this potential confounder.

It was unclear why there were fewer measurement episodes of these interventions, and we did not specifically investigate why the nurses were not recording these episodes. It may be that the nurses did not feel these care episodes were as 'significant' as suctioning or turning (in terms of the ICP instability produced), hence were less likely to record them, however we cannot substantiate this.

The first section presents the baseline data of each of these groups and the distribution of the outcome measures for analysis. The second part presents the change in the primary outcome measure (ICP) with each of these interventions and the percentage change from baseline. Due to the

lack of clinical significance seen with these interventions, no further analysis beyond these descriptive measures has been undertaken

Table 20: Measurement episodes in other interventions

Nursing Intervention	Number of children with recorded interventions
Eye Care	21
Mouth Care	20 (but one data set incomplete) so 19
Washing	10

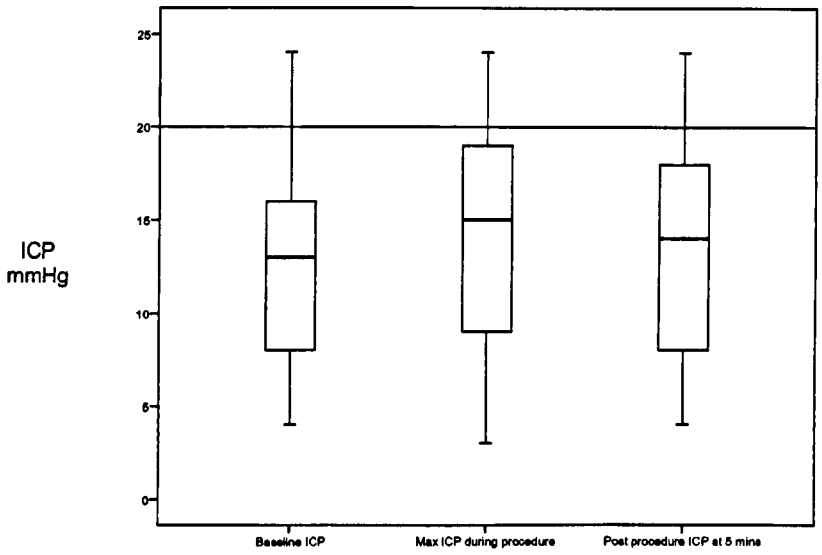
8.2 The effect of eye care

All outcome measures in the eye care data were normally distributed (see Table 21).

Table 21: Eye care data distribution

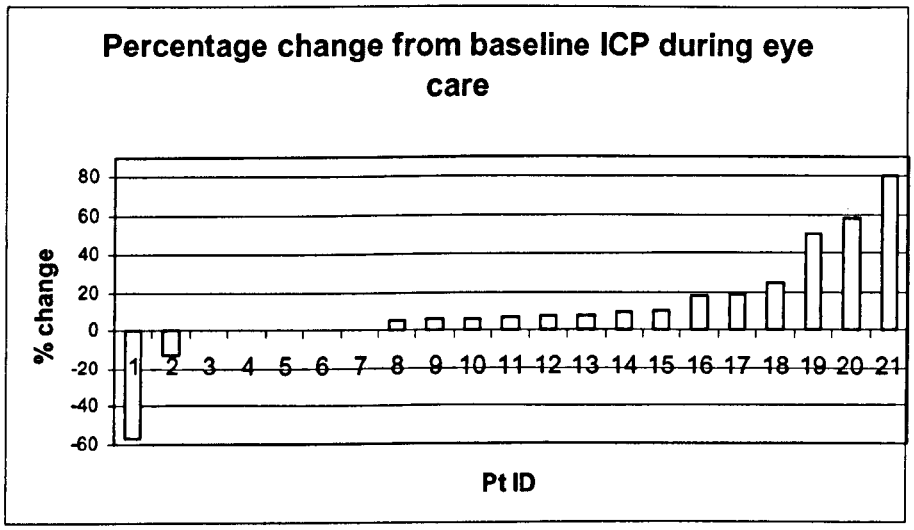
		Baseline ICP	Max ICP during procedure	ICPrise	Post procedure ICP at 5 mins
N	Valid	21	21	21	21
	Missing	0	0	0	0
Mean		13.24	14.33	1.10	14.05
Median		13.00	15.00	1.00	14.00
Std. Deviation		6.300	6.468	2.234	6.407
Skewness		.130	-.230	.166	-.003
Std. Error of Skewness		.501	.501	.501	.501

A box plot was first used to display the data graphically (see Graph 66) and showed no clinically significant effects as a result of the intervention. Five children had baseline ICP values over 20mmHg (21-24mmHg) and these children therefore had maximal ICP values that still exceeded 20mmHg, but there was very little change from their baseline and this is seen in more detail in Graph 67.



Graph 66: The effect of eye care on the ICP response

To explore this data a little further, the percentage change in ICP with eye care (from the child's baseline) is shown below. This is calculated by peak-baseline/baseline x 100. The mean change from baseline was +11.3% ([-57] to [+80]. These have been sorted into ascending order.



Graph 67: The percentage change from baseline ICP during eye care

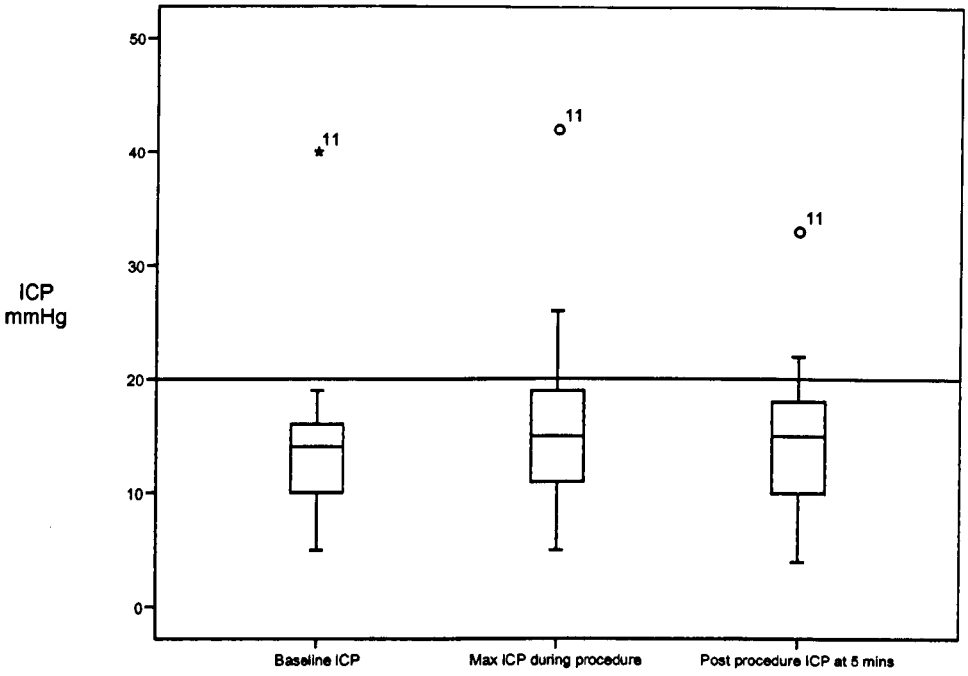
Following this analysis which showed very little variation in ICP with this intervention it was not appropriate to analyse this data any further as the intervention was not clinically significant.

8.3 The effect of mouth care

None of the outcome measures for this data were normally distributed due to one extreme outlier no.11 (see Table 22 and Graph 68).

Table 22: Distribution of mouth care dataset

		Baseline ICP	Max ICP during procedure	Post procedure ICP at 5 mins
N	Valid	20	20	19
	Missing	0	0	1
Mean		14.55	16.15	14.47
Median		14.00	15.50	14.00
Std. Deviation		7.060	7.883	6.450
Skewness		2.516	1.902	1.241
Std. Error of Skewness		.512	.512	.524

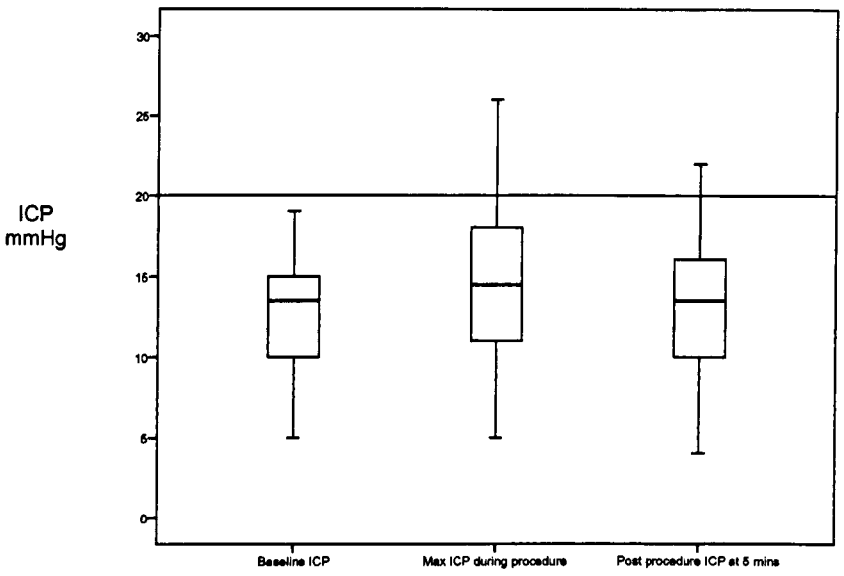


Graph 68: The effect of mouth care on ICP response (whole sample)

With this outlier removed (at the expense of a sample of 18 children) the data were now normally distributed (see Table 23 and Graph 69).

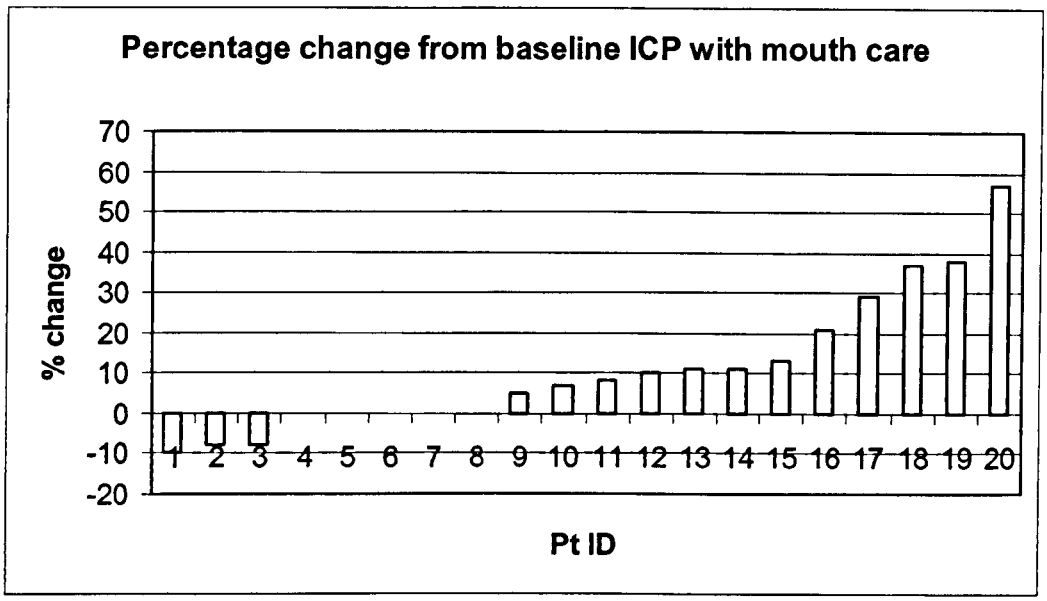
Table 23: Distribution of mouth care dataset with outlier removed

		Baseline ICP	Max ICP during procedure	Post procedure ICP at 5 mins
N	Valid	19	19	18
	Missing	0	0	1
Mean		13.21	14.79	13.44
Median		14.00	15.00	13.50
Std. Deviation		3.838	5.149	4.768
Skewness		-.259	.296	.133
Std. Error of Skewness		.524	.524	.536



Graph 69: The effect of mouth care on ICP response with outlier removed

After examining this data, no clinical significance was seen with this intervention (defined as ICP > 20mmHg) however to explore the data a little further the percentage change from baseline was calculated. The mean change from baseline was +11% ([-10] to [+57]. These have been sorted into ascending order.



Graph 70: The percentage change from baseline ICP with mouth care

This confirms that in addition to the lack of clinical significance there is very little variability from baseline in the majority of children, so it was not appropriate to explore this intervention any further.

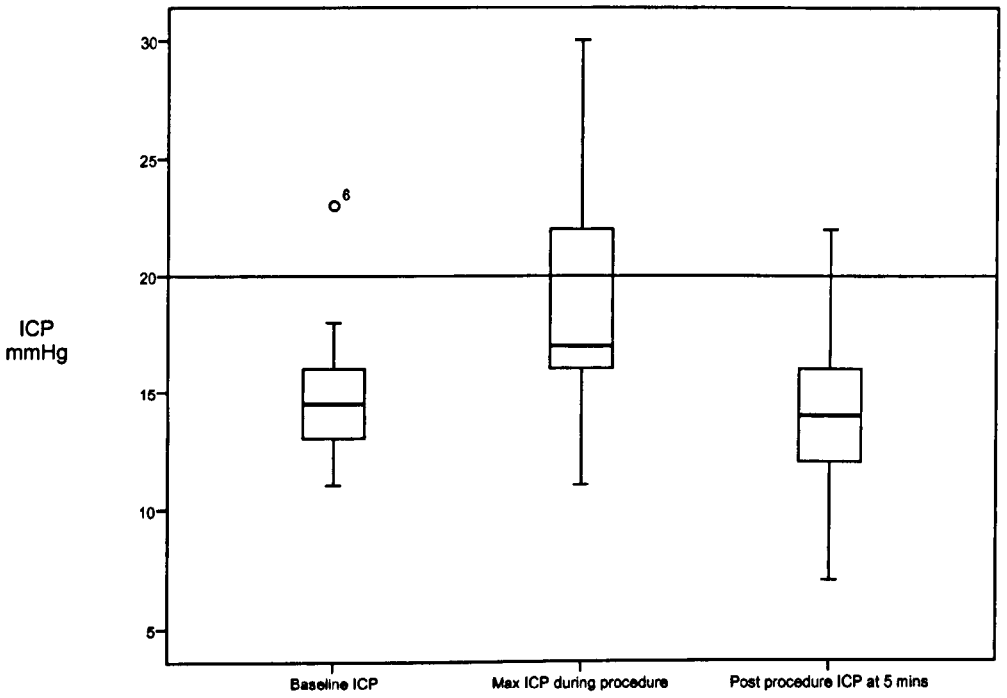
8.4 The effect of washing the child

The washing data (in 10 children) were not normally distributed for any outcome measure (see Table 24).

Table 24: Distribution of the washing intervention data

		Baseline ICP	Max ICP during procedure	ICPrise	Post procedure ICP at 5 mins
N	Valid	10	10	10	10
	Missing	0	0	0	0
Mean		15.00	18.50	3.50	13.90
Median		14.50	17.00	2.00	14.00
Std. Deviation		3.528	5.233	4.673	4.175
Skewness		1.272	1.139	1.858	.225
Std. Error of Skewness		.687	.687	.687	.687

When this data were examined on a box plot (below), there seemed to be one outlier (no.6).



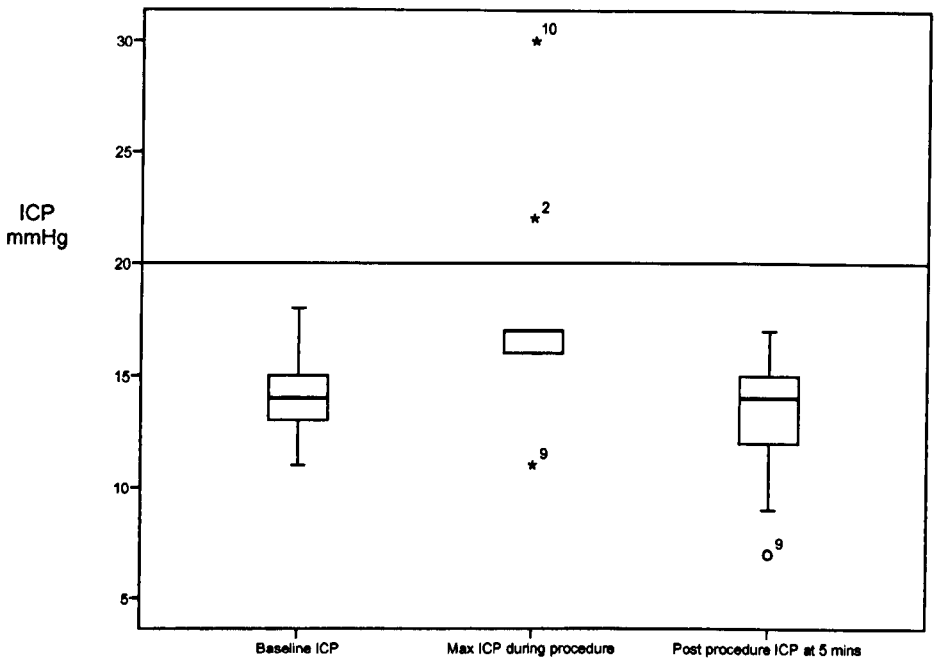
Graph 71: The effect of washing on the ICP response (whole sample)

This child was removed and the data explored further, but even with this outlier removed (see below) not all the outcome measures were normally distributed, probably due to the small sample now of nine children.

Table 25: Distribution of the washing intervention data (with outlier removed)

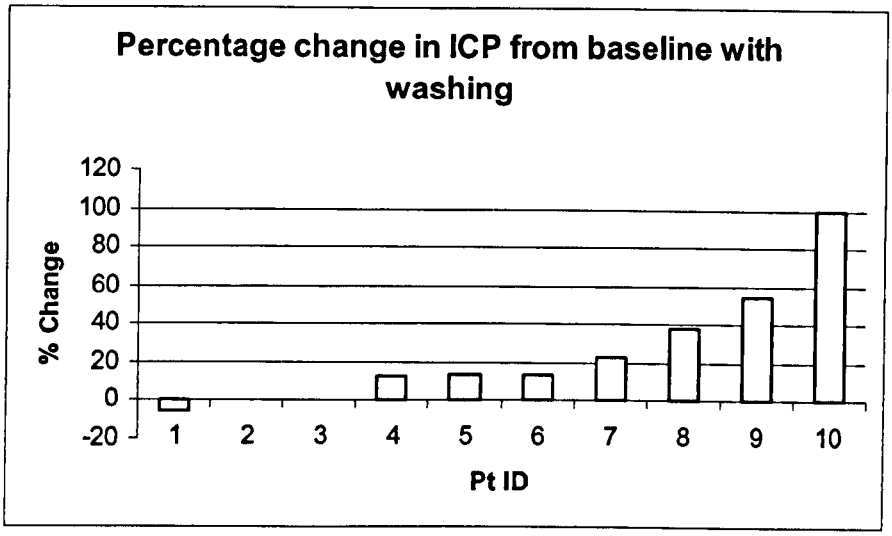
		Baseline ICP	Max ICP during procedure	ICPrise	Post procedure ICP at 5 mins
N	Valid	9	9	9	9
	Missing	0	0	0	0
Mean		14.11	18.00	3.89	13.00
Median		14.00	17.00	2.00	14.00
Std. Deviation		2.261	5.292	4.781	3.240
Skewness		.075	1.542	1.756	-.850
Std. Error of Skewness		.717	.717	.717	.717

It becomes evident in such a small sample (n=9) that other outliers exists (see the box plot below) and removing them is not helpful as it will produce a sample so small that no conclusions will be able to be drawn.



Graph 72: The effect of washing on the ICP response (with outlier removed)

To explore this data a little further however, the percentage change in ICP with washing (from the child's baseline) is shown below. This is calculated by peak-baseline/baseline x 100. The mean change from baseline was +23% ([-6] to [+100]. These have been sorted into ascending order.



Graph 73: The percentage change from baseline ICP during washing

Although this analysis is weak in statistical terms, with so few patients for analysis it helps to demonstrate that in most patients there was little change from baseline ICP. No conclusions can be drawn on the effects of washing on ICP, but the initial trend on Graph 72 does suggest that this intervention may cause elevations in ICP in some children. Further research in more children is necessary with regard to the effects of washing.

8.5 Conclusion and summary of results of other interventions

It is notable that the mean baseline ICP for both eye care and mouth care was around 13mmHg, which was lower than that for suctioning (16.6mmHg mean), but was at a similar level to turning (13.5mmHg). Although these interventions are important, they are less important than airway related procedures and may be left until the ICP was lower to undertake. This is a sensible clinical nursing decision to minimise intracranial instability in this

group of children. In conclusion, these results show that neither eye care nor mouth care produce any clinically significant effects on ICP in moderate to severe head injured children. With regard to the effects of washing, no conclusions can be drawn with such a small sample, as there was considerable variation in maximal ICP responses. It is clear the effects of washing warrant further investigation in head injured children.

CHAPTER 9

RESULTS:
THE EFFECT OF NURSING INTERVENTIONS IN CHILDREN AFTER DECOMPRESSIVE CRANIECTOMY

9.1 Introduction

This chapter will present the first evidence on the effects of nursing interventions on the ICP response in children who have undergone a decompressive craniectomy. This procedure involves the removal of part of the skull (and usually the dura mater is expanded) to allow the brain to swell with less compression (allowing a greater gain in brain volume). The physiology of pressure changes in the cranial cavity, as described by the Monroe-Kellie doctrine (see background chapter) only apply when the cranium is closed. Decompressive craniectomy thus completely alters the physiology where pressure changes should be able to be more readily tolerated with less pressure changes (in ICP) seen (Schirmer et al 2008).

Given the small sample of this sub group of the main study, they are presented as a case series. This is still important information however, as there is no published work on the effect of nursing interventions on the ICP in these children (or indeed in adults) and this procedure is increasing in popularity with international multi-centre trials ongoing. The first part will present the demographics and baseline data of this series of patients. The second part presents the change in the primary outcome measure (ICP) and the percentage change from baseline. Due to the small sample size (n=5) and the lack of clinical significance seen with any interventions in these children, only descriptive analysis will be undertaken. The measurement of interventions in this series of children extends up to those undertaken in the

first 72 hours after the child's admission to PICU, which again is still in the hyper acute phase of head injury; however the time from injury to decompressive craniectomy varies.

9.2 The decompressive craniectomy sample

Five children are presented in this case series (their demographic data is in Table 26). Two children in this series had very early decompressive craniectomies (within six hours of PICU admission). Three of these children were previously in the main study group (they had had their first measurement taken with a closed head) and crossed over into this sub group after their craniectomy. All the measurements were undertaken within the first 72 hours after the child's admission to PICU (but the time frame of these measurements from day of injury varied between patients). One child had DC on day one, one patient had DC on day two and one had DC on day three post PICU admission.

Table 26: Demographics of decompressive craniectomy children (n=5)

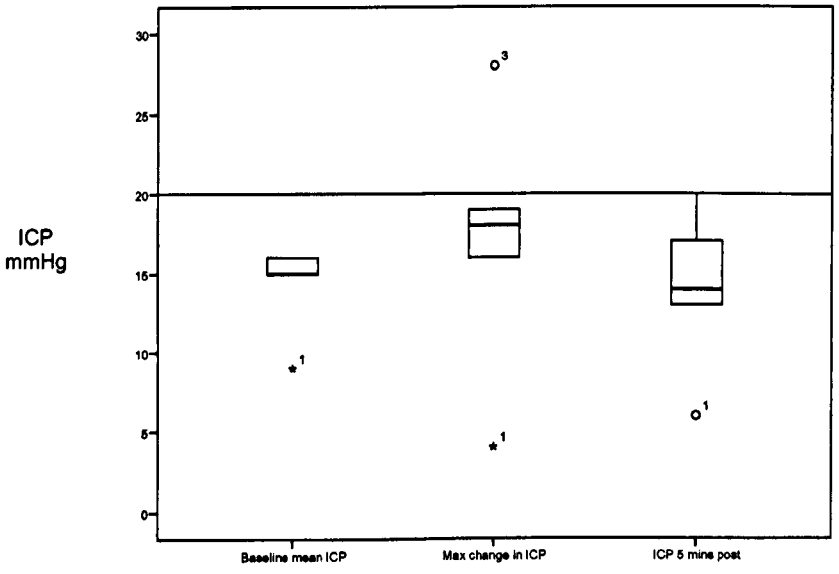
Demographic	Median	Range
Age (years)	10	3 – 15.5
Gender	3/5 female	
Post-resuscitation GCS	6	3-7
Mechanism of Injury	2 Pedestrian RTA 1 Passenger RTA 1 Fall 1 crush injury	
Mean ICP prior to DC mmHg	29	25-35
Time of performance of DC	Day 2 (2nd 24 hours)	6 hours – day 3
Mortality	0	
Six month outcome (GOS score)	4/5 GOS 1-2 mild-moderate disability 1/5 GOS 3 severe disability	

Table 27: Baseline Demographics of the children before ETS/MV

Baseline	Mean	SD
ICP mmHg	14.2	3
CPP mmHg	68	4.4
MAP mmHg	82	6.2

9.3 The effects of endotracheal suctioning after decompressive craniectomy

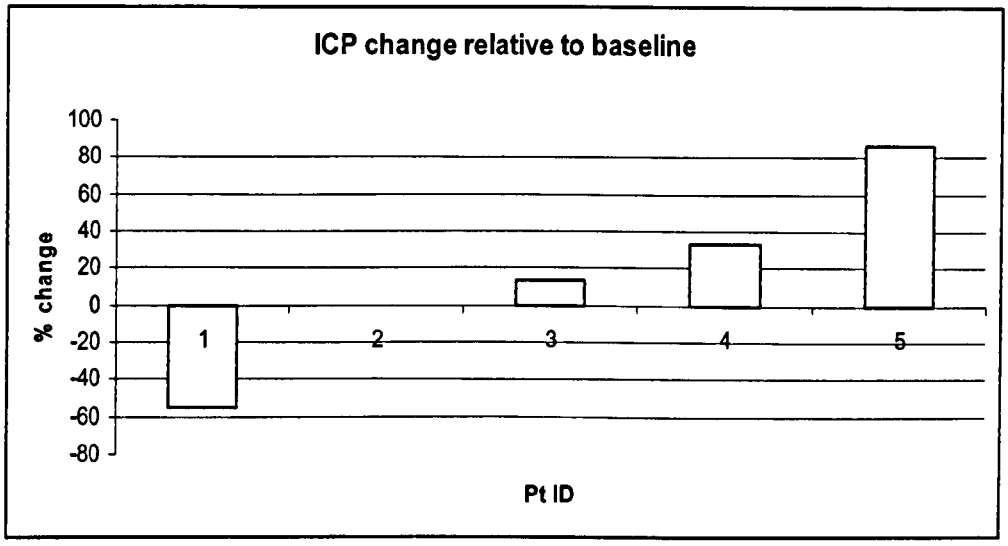
Graph 70 shows the effects of suctioning on the ICP in this small group. With the exception of one child (no.3) no others exhibited clinically significant changes in ICP (> 20mmHg) during this intervention, with one child (no.1) having a low ICP throughout (< 10mmHg).



n=5

Graph 74: The effect of ETSMV on ICP after decompressive craniectomy

The average change from baseline during ETSMV in this group was +15.4% (Range -55% to + 85%) but this was not clinically significant (except in one child who exceeded 20mmHg).



Graph 75: The percentage change from baseline ICP during ETSMV in children after decompressive craniectomy

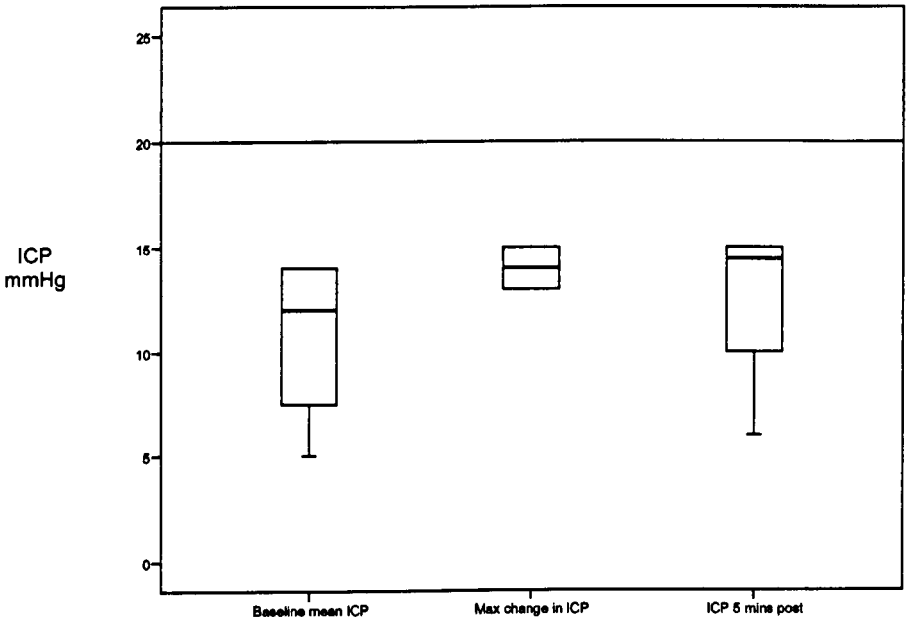
It is not possible to draw conclusions from such a small sample but it is still important to examine the effects of interventions in these children. These results show perhaps the expected effects (on ICP) of noxious stimuli in children after decompressive craniectomy. There is a trend towards higher ICPs during the procedure (but for most children this is not clinically significant), but this returns to low baseline levels by five minutes.

9.4 The effects of turning after decompressive craniectomy

There were only four children who had recorded turning interventions in this series (their baseline data can be seen in Table 28), and the results can be seen on the box plot below.

Table 28: Baseline demographics of the sample before turning

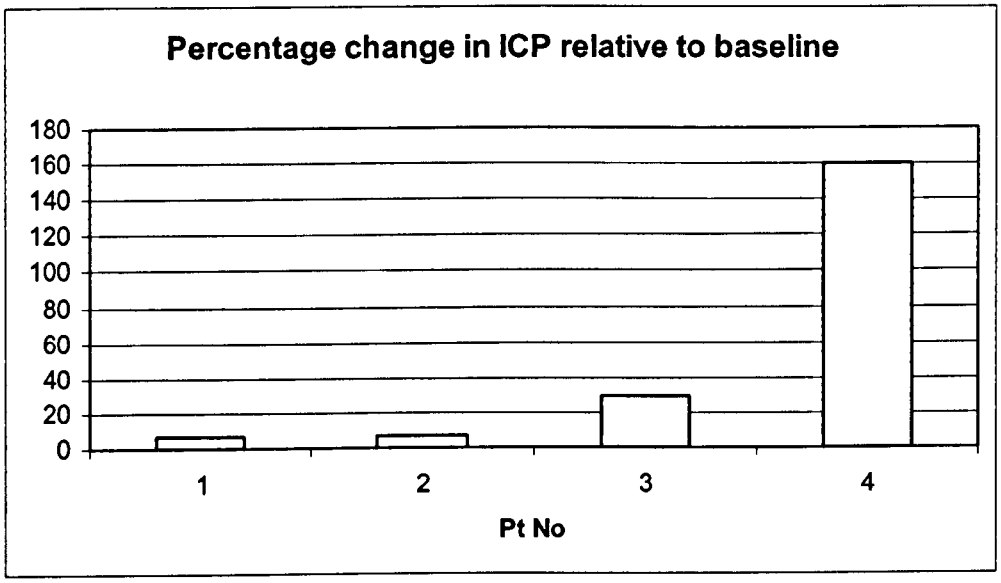
Baseline	Mean	SD
ICP mmHg	10.8	4.3
CPP mmHg	72	5.5
MAP mmHg	84	4.3



Graph 76: The effect of log-rolling on ICP after decompressive craniectomy

In these four children there were no clinically significant effects of turning on their ICP. Similar to ETSMV there appears to be a trend towards ICP elevation during the procedure, however no child exceeded 20mmHg.

The percentage change from baseline showed an average increase from baseline by 50%, but this was not clinically significant (did not exceed 20mmHg) given their low baseline ICPs.



Graph 77: The percentage change from baseline with log-rolling after decompressive craniectomy

It is not possible, nor intentional to draw conclusions from this small sample, but this is the first attempt at exploring the effects of interventions after log-rolling.

9.5 The effects of eye care, mouth care and washing after decompressive craniectomy

There were only three measurement episodes of eye care and one of mouth care and washing which prevents any analysis of this data. However given that these interventions produced no clinically significant effects in the main group, it is very unlikely that they would do so in this group of children.

9.6 Conclusion and summary of results of nursing interventions in children after decompressive craniectomy

This case series of five children is the first attempt to describe the effects on ICP with routine nursing interventions in children after decompressive craniectomy. The effects demonstrated would appear to be consistent with expected physiological effects after this procedure. However it must be recognised that the measurement of ICP in patients after this procedure, some claim produce falsely low numbers (as the ICP catheter reflects a global ICP) and the ICP catheter may be affected by the proximity of the craniectomy defect (Timofeev et al 2008).

Without any prior knowledge of the effects of nursing interventions in these children, this group are typically managed like the closed head injured children, with additional precautions undertaken prior to procedures. The effects seen here may suggest that these additional precautions are not necessary in this group of children, thus avoiding unnecessary additional drugs such as endotracheal lignocaine or additional sedation boluses.

Further work is warranted in a larger patient sample of these children, possibly exploring the changes in ICP with interventions at fixed time periods after the craniectomy eg day one, day three and day five.

CHAPTER 10

DISCUSSIONS

10.1 Introduction

This chapter will both discuss and summarise the main aspects of the thesis, addressing the research questions and aims of the study, the study sample, the results and their relation to published literature and will conclude with recommendations for practice and for future research.

10.2 Observational studies and the use of scientific research methods in nursing

Intensive care nursing as a specialty deals with critically ill children who have derangements in a variety of physiological variables. The experienced ICU nurse puts these within the context of the child's illness, interprets their significance, determines if any action is required and interprets this 'physiological overload' for the parents/caregivers. ICU nurses are in a good position to study physiologic phenomena and have a duty to study the effects of our interventions on patients in order to minimise harm and promote optimal care delivery.

Observational studies are designed to study the relationship between variables and not to prove cause and effect, which they cannot do (Brennan and Croft 1994). However these important studies, in fields where little research is present, form the basis for further experimental work and are essential to first describe phenomena in more detail to permit further work.

The limitations of observational studies are well described (Brennan & Croft 1994; Christenfeld et al 2004; Wunsch et al 2006) with the effect of confounding variables the most notable. Another issue to be aware of is the potential changes in medical and surgical practice over the duration of the study, as they may add more confounding variables.

Despite the limitations of observational studies, a further benefit of these studies (as opposed to an experimental study) is that they study nursing practices 'in the real world' not in a highly controlled regulated manner (which of course is not how real practices are performed). So although perhaps generating less robust data, they may relate more to clinical practice.

Quantitative research in the field of paediatric intensive care and specifically in paediatric head injuries is always notoriously difficult, as there are relatively few patient numbers, complex phenomena to be studied and many confounding variables (Duhaime 2007). One method to overcome the limitations of small numbers is to use multiple centres, but this too brings with it problems. The biggest problem is of ensuring consistency in the patient management or agreeing to a consistent treatment protocol between centres, and an audit undertaken within the UK (Tume and Baines 2008) demonstrated quite marked variation in PICU practices even across the UK. Another potential method to overcome small numbers is to collect the data longitudinally over a long period of time (in one centre) for example 5–10 years. This however also has limitations in terms of changing practices and

management over this time and also researcher consistency in this time period and is not possible for a PhD project. In the methods chapter we have discussed other potential methods of analysis of this observational data and why this method was chosen.

10.3 Aims, Objectives and Research Questions

The broad aim of this research was to investigate the physiological effects of the routine intensive care nursing interventions of: endotracheal suctioning with manual hand ventilation, turning the child (by a log-rolling method), eye care, mouth care and washing, on the ICP and CPP of moderate to severe closed head injured children in intensive care, which it has done.

The specific research questions asked (and the answers as generated) from the measurement of these episodes in the hyper acute phase of head injury are as follows:

In children with moderate to severe head injury in intensive care:

- ***What are the effects of these routine nursing interventions on the ICP and CPP of moderate to severe closed head injured children?***

This study has shown that both ETSMV and log-rolling are associated with clinically and statistically significant changes in ICP from baseline to maximal ICP and maximal to 5-minute post ICP. Neither eye care nor mouth care showed any clinically significant effects on ICP, suggesting

these procedures are not noxious and are tolerated very well. The small number of washing episodes in this study means that no claims can be made about the effects of washing on ICP and this intervention warrants further investigation.

Cerebral perfusion pressure was not a valid outcome measure in this study as the majority of children had their CPP augmented to age-dependant parameters. CPP did however reduce slightly in line with the physiologic CPP formula as MAP partly compensated for the rise in ICP.

- ***Which interventions produce the greatest rises in ICP?***

How many of the intervention episodes resulted in a clinically significant ICP rise (defined as >20mmHg)?

This study has shown that endotracheal suctioning and turning (via log-rolling) produce the greatest increases in ICP that both result in around three quarters of children exceeding the 20mmHg (clinically significant level) during the intervention.

- ***In the hyper acute phase of head injury what proportion of these intervention episodes resulted in peak ICP values that exceeded a) 20mmHg b) 30 mmHg and c) >30mmHg?***

During ETSMV 70% of children had maximal ICP values that exceeded 20mmHg, with 28% of these exceeding 30mmHg albeit most only transiently. During turning, 70% of children also had maximal ICP values

that exceeded 20mmHg, with just fewer than 10% exceeding 30mHg. Around 30% of children with turning and ETSMV did not exceed 20mmHg. Eye care, mouth care and washing did not demonstrate significant alterations from the child's baseline ICP.

- ***Does the maximal ICP, ICP rise or the recovery time (in minutes) correlate with the child's baseline ICP?***

During both ETSMV and turning, children with higher baseline ICPs (> 15mmHg) exhibited higher maximal ICP values (but no significant difference in ICP rise) suggesting a linear relationship between baseline and maximal ICP, and this relationship was more pronounced during turning. There was no difference between children in the high or low baseline ICP groups in their recovery times after turning or ETSMV.

- ***How long do these children take to recover their baseline ICP values after these interventions?***

After ETSMV around half of the children had a post procedure ICP less than their baseline ICP, while the other half of the children had not recovered their baseline ICP by 5 minutes after the ETSMV (median recovery time three minutes). With log-rolling, over three quarters of children had higher post procedure ICPs than their baseline. More than half of children had not recovered their baseline ICP by 5 minutes after the turn (median recovery

time five minutes). During log-rolling the more the time taken for the turn the longer the children took to recover their baseline ICP.

- ***Are there any variables (confounders) that affected the child's ICP response to interventions?***

No confounders were shown to affect the ICP response during ETSMV. During turning a number of factors that were highly correlated: the baseline ICP correlated with maximal ICP and the maximal ICP correlated significantly with the post procedure ICP. The correlation of baseline ICP to maximal ICP was so clear that we are able to model the predictive increase in ICP with log-rolling to be ~ 6mmHg above the child's baseline ICP. As the time taken for log-rolling increased the longer the children took to recover their baseline ICP.

- ***What are the effects of routine cares in children following decompressive craniectomy?***

No nursing intervention produced any clinically significant effects (defined as ICP > 20mmHg) in this group of children. Although there was a trend to elevation in ICP during suctioning and turning, neither exceeded 20mmHg. This group (as expected) exhibited lower baseline ICP values (mean 10 – 14mmHg) and their ICP response to interventions was minimal.

10.4 The study sample

This study has examined the effects of selected nursing interventions in the intended group of children that of moderate to severe closed head injured children. The quantification of having a moderate to severe head injury was defined by an injury severe enough to warrant invasive intracranial pressure monitoring. This did exclude some children whose injuries were so catastrophic that ICP monitoring was not considered appropriate (n=9). It also excluded children under two years (n=2), children who had an open head injury (n=1) whose parents refused consent (n=1) and children after decompressive craniectomy (n=5) (although these patients became a separate sub group).

The intention was to keep this sample as homogenous as possible (moderate to severe closed head injured children), however even with the best intention, head injury severity is complex and is not just defined by GCS or invasive ICP monitoring requirement but also by pathophysiology displayed on the CT scans. The early GCS scores in our sample did include five children with the Glasgow Coma Scores over 9 but whose CT scan was sufficiently abnormal to warrant invasive ICP monitoring. The mortality in the study sample was 8% 2/25 and this roughly correlates to the predicted PIM 2 score of 7%. If these children are put into context of the total head injury population that were admitted to AHCH PICU in this three year period (n=105), 56 children had relatively mild head injury and were woken up quickly, extubated and stayed less than one day on PICU.

10.5 Severity of Illness of the study sample

The risk of mortality score used in UK PICU's is the PIM (Paediatric Index of Mortality) (currently PIM 2, having changed from PIM 1 in 2006/7) (Pearson et al 2001; Slater et al 2003). The PIM score predicts the risk of mortality in a group of patients, but it is not validated to predict mortality risk in individual patients, being really designed as a tool for auditing PICU performance (Tibby and Murdoch, 2002). It is a risk calculated on the child's very early physiological data (within an hour of PICU team contact). The Standardised Mortality Ratio (SMR) is the ratio of observed deaths to expected deaths for a specific population, and provides an estimate of individual intensive care units' performance in relation to others.

The PIM score of this subset of study children was 0.069 (~7% risk of mortality) (95% CI 0.017 - 0.121) yielding a standardised mortality ratio (SMR) of 1.158 (95% CI 0.140 – 4.184) (meaning a slightly higher number of deaths than is expected). However the PIM score is not a score designed to assess the severity of illness in particular groups of patients (eg severe head injuries) and also not in small groups of patients eg less than 100. In terms of predicting mortality, some have found the PIM score to discriminate better than other risk tools between survivors and non-survivors in head injuries (Grinkeviciūte et al 2007). This PIM2 derived SMR may have underestimated the severity of head injury; however the PIM score

only uses one neurological parameter, pupil reactions. In the US a specific head injury severity scoring tool has been developed recently; the RHISS (Relative Head Injury Severity Score) (Cuff et al 2007) but this is not consistently used in the US and not used at all in the UK or Europe. This study group's SMR compares to a three year average SMR of the total PICU population of 0.98 (meaning less deaths than expected).

Some previous work in this field has reported very low baseline ICP values, which does not reflect the severity of a severe paediatric brain injury group. The other demographic details of the study group reflect those reported by others (Parslow et al 2005): predominantly male (60%), slightly older children (9.4 years) with the most common mechanism of injury pedestrian road traffic accident (RTA) (40%) and some falls in younger children (24%). Overall, despite the small sample size, the researcher believes this sample is a fairly homogenous and defined group (although exact pathophysiology on CT scan eg the presence of diffuse axonal injury has not been accounted for), and accurately reflects the sample group intended for study.

10.6 The nursing interventions in these children

In this study the frequency of different nursing interventions recorded reflects the clinical importance and frequency of the intervention, with endotracheal suctioning accounting for 48.5% of all the intervention episodes in these children. As discussed previously this is not surprising as

it is required regularly to ensure endotracheal tube patency which, if impaired, directly increases ICP (through increased $PaCo_2$ levels).

Turning accounted for 18.1% of the interventions, and although this occurs regularly (approximately every 12 hours), extra turns would occur with chest x-rays or when being transferred to CT scan. Eye care and mouth care accounted for 17.5% and 12.6% of the episodes respectively with washing alone only contributing to 3.2% of the data collection episodes.

Although patients receive all these interventions regularly, and the nurses were asked to record as many 'care episodes' as they could, clearly there could be no assurance of an episode being recorded. We did not specifically investigate why the nurses were not recording some of the care interventions. Perhaps the lack of recording of washing episodes reflects the nurses' perceptions that this intervention has minimal effects on ICP and the same may apply to the reduced number of eye and mouth care episodes collected compared to suctioning and turning.

10.7 The effects of endotracheal suctioning and manual ventilation

Our results clearly demonstrate that ETSMV does cause both clinically and statistically significant changes in ICP from baseline to maximal ICP and from maximal ICP to 5 minute values. The majority of children exceed the clinically significant 20mmHg level during suctioning.

Our suctioning findings are, in general, supported by previous adult work. In the studies that specifically examined ETS in head injured patients

(Fisher 1982, Rudy et al 1991; Crosby and Parsons 1992; Brucia and Rudy 1996, Billotta et al 2008) all demonstrated that ETS produced statistically significant elevations in ICP compared to baseline. Campbell (1991) described a classic 'stair step' pattern in response to ETS and manual hyperinflation, which increases with successive treatments, and this was confirmed by others (Rudy et al 1991; and Crosby and Parsons 1992). We did not note this 'stair step' pattern (except in more severely ill children receiving thiopentone) however the majority of our patients were sedated and muscle relaxed, which Kerr et al (1998) claimed abolishes this response. In addition, we only recorded measurements at three time points (not continuously) which may have been why we did not see this response. As discussed previously all our children were thought to be heavily sedated and most were receiving NMB, but despite this we still showed significant effects with suctioning. However we did find a trend towards significance in increasing ICP rise as the time taken for suctioning increased, similar to Campbell (1991), Garradd and Bullock (1986) and Paratz and Burns (1993). The latter two studies found statistically significant increases in ICP with respiratory physiotherapy treatment duration.

There was also a trend towards significance from maximal ICP to post procedure ICP, suggesting the higher peak ICP the higher the post procedure ICP will be. Despite this, just over half the children had a post procedure ICP less than their baseline ICP. In relation to suctioning this may suggest improved carbon dioxide control post ETSMV (either from

secretion removal or the effects of manual ventilation), although this cannot be substantiated from our data.

In relation to cerebral perfusion pressure and mean arterial pressure Crosby and Parsons (1992) found significant changes from baseline values during the endotracheal suctioning for mean arterial blood pressure, mean intracranial pressure, cerebral perfusion pressure and heart rate. As discussed earlier in the thesis, both CPP and MAP did change slightly (in line with the formula CPP= MAP - ICP) for both suctioning and turning but their change was neither clinically nor statistically significant. MAP rose slightly, only partly compensating for the ICP rise and thus CPP reduced slightly. Perhaps we did not observe the changes described in the adult population as nearly three quarters of our patients had their CPP augmented by a nor-adrenaline infusion.

10.8 Does baseline ICP make any difference to the ICP response to endotracheal suctioning and manual ventilation?

Our findings with regard to high and low baseline ICP groups and ICP response confer with Paratz and Burns (1993) and Garradd and Bullock (1986) who found that the ICP response was higher in the adults who had baseline ICPs > 15mmHg. We showed that children with baseline ICPs greater than 15mmHg exhibited higher peak ICPs (with no difference in ICP

rise). This suggests that ETSMV produces a linear increase in ICP that is proportional to the baseline ICP.

However, the effect of manual ventilation may have minimised the ICP response we saw in both groups. Kerr et al (1997) found that adults who were hyperventilated during ETS showed significantly lower ICP responses during ETS. We did not intend the nurses to 'significantly hyperventilate' these children, but as the effect of manual ventilation cannot be quantified, we accept that this may have happened on occasions and affected the responses we have shown.

10.9 What was the effect of coughing on the ICP response during endotracheal suction and manual ventilation?

Our findings in relation to the affect of coughing were in contrast to Gemma et al (2002). They found that in adult head trauma patients that coughed during suctioning, the ICP response was higher than those who did not cough. There were only two children in our group who coughed during suction and these patients had similar ICP responses to the others. Our results confer with Fisher et al (1982) who found that in the two thirds of children who coughed during ETS their ICP rise was similar to the non-coughing children. However with only two children who coughed to examine, it is not possible for this study to make further claims about the effects of coughing on ICP response. Physiologically, coughing, by

increasing intrathoracic pressure (thus impeding cerebral venous return) should produce an increase in ICP, but we did not observe this.

During ETSMV there were multiple confounders, however many were consistently applied across the group. The biggest confounder during this intervention we feel has been manual ventilation and the affect of this on the ICP response cannot be underestimated (Skippen et al 1997; Coles et al 2007). Carbon dioxide cannot be reliably measured during MV with current $EtCo_2$ measurement devices, as ventilation is inconsistent. However we did try and account for the effect of reduction in carbon dioxide during manual ventilation by recording the EtCo2 within 30 seconds of being connected back to the ventilator, but there appeared to be no association between the changes in this level and a drop in ICP.

10.10 What was the effect of a sedation bolus prior to endotracheal suction and manual ventilation?

There was a trend towards significance in terms of a reduction in ICP rise when a pre-procedure sedative bolus was administered but there was no statistically significant difference between the groups in terms of maximal ICP. No other confounders reached statistical significance; however in an observational study with a small sample of 24, this was never intended to do so. The vast majority of children (88%) had an intravenous (IV) bolus of a sedation/and or opiate analgesic prior to the ETSMV procedure which again

made it problematic to compare to the small number of children who did not receive an additional bolus of the drug prior to the intervention. The drugs used were predominantly IV Midazolam (as sedation) and Morphine (as the analgesic). In addition we could not quantify exactly how sedated these children were as all but three were muscle-relaxed.

Kerr et al (1998) found adult head trauma patients treated with opiates and neuromuscular blockade (no cough) showed a flat response to ETS, with little change in ICP. As discussed previously our children were assumed to be heavily sedated and most were receiving NMB, but despite this we still showed significant effects with suctioning. It may have been that some of our patients were under sedated while receiving muscle relaxation and this is concerning. The physiological effects of inadequate sedation under muscle relaxation include unrelieved pain and anxiety which increases sympathetic outflow, increases global oxygen consumption, carbon dioxide production and leads to an increase in metabolic rate (Arbour 2004). This is not a good state in any critically ill child, but certainly not in severe brain injury, where the cornerstone of management is adequate sedation to rest the brain (Adelson et al 2003). It may be that the application of noxious interventions (such as endotracheal suctioning) highlights this inadequate sedation level, causing a state of increased arousal and potential breakthrough awareness, which may have caused a greater increase in ICP.

Additionally some studies (Hanowell et al 1993; Sperry et al 1992) have found certain drugs (alfentanil, fentanyl and sufentanil) actually worsened the ICP rise with suctioning (possibly due to increasing cerebral blood flow). In our study only one child in the suctioning group had a fentanyl bolus alone (and none in the turning group) which did not permit us to examine this further.

10.11 What was the effect of muscle relaxation on the ICP response during endotracheal suction and manual ventilation?

A number of authors have shown that muscle relaxants can reduce the ICP response to suctioning (White 1982; Werba et al1993; Kerr et al 1998; Garradd and Bullock, 1986) but again we could not demonstrate any difference in the ICP response between these two groups. Only four of the children in our study were not paralysed, but of these, only two actually coughed with suction (implying the others were heavily sedated). In contrast, two of the children who were receiving NMB (and thus 'assumed' to be paralysed) actually moved or coughed during the procedure. Despite this there were no significant differences in any of the variables) between the children who received NMB compared to those who did not.

10.12 What was the effect of barbiturates on ICP response during endotracheal suction and manual ventilation?

Rudy et al (1991) found that patients on barbiturates had significantly lower ICP values throughout the ETS procedure and they stated it appeared to blunt the effects of ETS, but again, we did not see this in our group. There were only five children on thiopentone in our group, and despite a subtle trend towards significance (p=0.092) on ICP rise during suction, the only statistical difference between the two groups was in their post procedure ICP (which was higher than their baseline ICP). This pattern of an escalating ICP (from baseline to maximal to post) seen in the Thiopentone group, varies from the usual response seen in this study (a clear peak in maximal ICP but some recovery in the post ICP value) and in other published studies (Rudy et al 1991; Parsons and Ouzts Shogan 1984). This almost certainly reflects the poorer cerebral compliance in these children with more severe injuries (this drug is second tier therapy used for intracranial hypertension only). Therefore the response seen may in fact just reflect the response of children with a more severe injury, who are showing reduced cerebral compliance. This is however an important finding. One beneficial aspect of observational studies like this is that as only routine care practices are being observed, we can see the effects of such interventions in a severely ill group of children. In experimental studies, ethically, these severely ill children are almost always excluded (as

they are unstable) and so the effects in these very ill children can be rarely observed.

10.13 What was the effect of endotracheal lignocaine on the ICP response to endotracheal suction and manual ventilation?

The examination of any confounders was only a secondary aim of our study and while we could not demonstrate any significant effects from endotracheal administered lignocaine prior to suctioning, an observational study design is not appropriate to test this. A randomised experimental design would be necessary to examine this. Others have examined the effect of endotracheal administered lidocaine experimentally (White 1982; Yano 1986; Billotta et al 2008) finding intratracheal lidocaine to be effective in reducing the ICP response to suctioning in most patients (these were all adults). Billotta and colleague's (2008) study was also a pharmacokinetic study and they calculated an ED_{50} (the dose of a drug that is pharmacologically effective for 50% of the population exposed to the drug) of 1.7 +/- 0.3mg/kg as the dose of lidocaine required to prevent an ICP rise (in adults). It may be predominantly that our design was not appropriate to test this (there was only a small number of children who did not receive the drug and drug administration was selected by the nurse which introduces bias) but probably also that the dose we administered was a significantly lower amount than that calculated above and the delay between administration of the drug and suction may not have been long enough for

the drug to take affect. We administered only 1-2mls of a 1% solution down the endotracheal tube two minutes prior to suction. In the UK the current clinical use of prophylactic intratracheal lignocaine is rare (Tume and Baines 2008) but with no adverse effects noted in our children, perhaps greater consideration should be given to researching the use of this drug.

10.14 How long did these children take to recover after endotracheal suction and manual ventilation?

In terms of recovery after suctioning, nearly half the children had not recovered their baseline ICP by 5 minutes after the procedure (and one quarter of these had a post procedure ICP greater than 20mmHg). However in just over half of the children, the post procedure ICP was less than their baseline ICP, possibly reflecting the effects of a reduction in carbon dioxide level achieved by secretion removal if sustained. Two out of four of the children who had an ICP of greater than 20mmHg for more than 5 minutes post ETSMV had poor six month outcomes, which may reflect the outcomes reported with secondary injury insults, although as previously mentioned patient outcome after severe TBI are not just related to secondary insults and raised ICP. Pathophysiology of TBI as seen on CT scan also has an impact on patient outcomes (Bahloul et al 2009; Boto et al 2009).

With regard to recovery times, previous work in this area had shown mixed results. Parsons and Ouzts Shogan (1984) found no significant ICP

changes at one-minute post ETS compared to baseline. However, this is contradicted by Rudy et al (1991), who found 76% of patients had not regained their baseline ICP values by one minute, 42% had not by five minutes and 25% had not by nine minutes. Kerr et al (1998) also found prolonged recovery times in 20% of patients who had not recovered their baseline ICP by 15 minutes post ETS. Hugo (1987) in a mixed paediatric and adult study examining the manipulation of nursing care activities (of which ETS was one), found that there was no statistical significance between baseline ICP and recovery time. However she noted that 20% of patients' ICP had not returned to baseline in 15 minutes or the protocol was terminated as the patient required medical intervention to reduce ICP.

10.15 The effects of turning (via a log-rolling technique) on the ICP response

There is no previous published work on the effects of turning (via a log-rolling approach) in the hyper acute phase of moderate to severe traumatic brain injury in children. The baseline ICP was slightly lower in the children before turning (13.4mmHg) compared to suctioning but still reflects a significantly ill population. It may be that turning is not perceived as important an intervention as suctioning (i.e. the prevention of pressure areas, although important is less critical than allowing secretions to accumulate, impairing both ventilation and oxygenation), thus the nurses may have waited longer (until ICP was lower) before turning these children.

When turned, these children exhibited both clinically and statistically significant changes in ICP from baseline to maximal to five minute post procedure ICP with 70% of children exceeding 20mmHg during the turn. There may be various reasons to explain this. Firstly the significant stimulation (both handling of the child and noise of people talking) are required to effect the turning process, secondly the rigid cervical collar is fastened up to turn (impairing cerebral venous drainage) and thirdly that turning may stimulate a cough or movement in a less heavily sedated or non-paralysed child (which in turn may increase ICP).

It is important to note there is very limited published literature specifically on the effects of turning (and none describe this performed via a log-roll technique) on patients with severe traumatic brain injury in intensive care and even less in children. Our results differ to the one study involving 13 children (Hobdell et al 1989) which found no significant change in ICP with turning or changing head position. This observational study was problematic however due to a very heterogeneous sample, the use of different ICP measurements devices, no baseline ICP reported and no account of confounder usage. This study produced results which conflicted with three adult papers around the same time (Parsons and Wilson 1984, Lee 1989, Jones 1995), which all demonstrated significant ICP rises with repositioning. These four papers were the best quality research papers

identified on this topic, but still presented little inferential data analysis, with only Jones (1995) and Parsons and Wilson (1984) presenting data on recovery times. Jones (1995) found that in adults (n=30) (with raised ICP from any pathology) who were turned from supine to left lateral (not log-rolled) 46% of patients still had an ICP above their baseline at five minutes after the turn. However Parsons and Wilson (1984), found that this ICP rise was transient and showed recovery towards baseline by 1 and 5 minutes. Other authors (Shalit and Umansky, 1977; Rising 1993 and Mitchell et al 1981) have also shown that turning produced changes in ICP, although less confidently and not in severely ill children. Our results did show similar findings to Muwaswes (1984) who noted that recovery to baseline ICP after turning (not log-rolling) related to the initial degree of ICP increase, however we also found that as the time taken for the turn increased the minutes to recovery also increased, suggesting that the duration of time the ICP is raised also increases recovery time to baseline ICP.

There were a number of factors that were significantly correlated during turning: the baseline ICP correlated with maximal ICP and the maximal ICP and ICP rise correlated significantly with the post procedure ICP. The time taken for the turn and the maximal ICP both correlated with the minutes to recovery (to baseline ICP). This suggests with a number of confounders absent during turning (manual ventilation, the use of Lignocaine, cervical collar always fastened, ventricular drain if present always clamped) that the

effects seen with turning represent a much more accurate picture of what happens when a noxious stimulus is applied to children with moderate to severe traumatic brain injury in the early course of their injury. In fact the correlation of baseline ICP to maximal ICP was such that we are able to model the predictive increase in ICP above the child's baseline to be ~6mmHg. This in itself may be helpful to clinicians when planning this intervention, it will aid the clinical judgement of the nurse in terms of sedation prophylaxis or other interventions to minimise ICP prior to the turn.

During turning, the maximal ICP was significantly higher in children with higher baseline ICPs (> 15mmHg) as would be expected given that maximal ICP correlated with baseline ICP, but the ICP rise was not significantly different. This suggests that this stimulus produces a linear increase in ICP. There were still a number of confounders during turning however with four less, this strengthens the results we found with this intervention.

10.16 What were the effects of sedation boluses and muscle relaxants on the ICP response during turning?

The administration of a sedation bolus or being muscle relaxed before the turn did not significantly affect the ICP response, in fact the values were actually higher in the children who received these drugs (not lower as would be expected). This concerning finding may suggest that these children may have been inadequately sedated. As discussed previously with suctioning,

the physiological effects of inadequate sedation are serious, including unrelieved pain and anxiety which increases sympathetic outflow, increases global oxygen consumption, carbon dioxide production and leads to an increase in metabolic rate (Arbour 2004). It may be that the application of any noxious intervention (be it endotracheal suctioning or turning) highlights this inadequate sedation level, causing a state of increased arousal and potential breakthrough awareness, which may have caused a greater increase in ICP. No literature has reported any effects of sedation or paralysis during moving or turning patients.

10.17 What was the effect of barbiturates on the ICP response during turning?

The pattern of escalating ICP, from baseline measurement to peak measurement to post measurement seen in the children on barbiturates during ETSMV, was not observed in the four children who received this drug during turning. If the explanation provided previously to explain this phenomena is accurate (ie that the children who receive this drug have a more severe head injury, and poorer cerebral compliance, thus cannot rapidly adjust to sudden pressure increases) then this effect should also be observed in the children being turned. The only comment that might be made is that perhaps in this particular group of children with severe enough head injuries to require barbiturates, airway related procedures (such as suctioning) may be more noxious than other noxious procedures such as

turning, but this is an area that would require more detailed research to explain this. It of course is entirely possible with the small number of children receiving thiopentone in the turning group (n=4) that a type II error has occurred.

10.18 What was the effect of having other (painful) injuries on the ICP response during turning?

Around half of the children admitted with moderate to severe TBI had other injuries (mainly skeletal fractures and chest injuries) and when examined there was a trend towards significance in an increasing ICP rise in the children with other injuries during the turn, suggesting they may have greater pain on turning. There is some evidence in neonates that pain may increase ICP (Bellieni et al 2003). Unfortunately there is no pain score or sedation score validated for children on NMB (who are paralysed) all the nurse has to use to assess pain are physiological variables such as heart rate, blood pressure and ICP. Yet these physiological factors are also directly affected by the pathology itself and ICU therapies eg inotropic infusions. Increased ICP may cause pressure on the brain stem, which will cause bradycardia and systolic hypertension, making it very difficult to ascertain and quantify pain and sedation level in these children. Perhaps other physiological means of measuring sedation level such as Bispectral Index (BIS) need to be investigated in this group of children.

10.19 How long did children take to recover after turning?

In terms of recovery after the turn, over three quarters of children had higher post procedure ICPs than their baseline. More than half of the children had not recovered their baseline ICP by 5 minutes after the turn, but this was no different between children in the high or low baseline ICP groups. Our results did show similar findings to Muwaswes (1984) who noted that recovery to baseline ICP after turning (not log-rolling) related to the initial degree of ICP increase and we found a strong correlation between maximal ICP and minutes to recovery. Additionally, we found that the longer the time taken for the turn, the longer the child took to recover their baseline ICP, which suggests that the more sustained in duration the ICP elevation is, the longer the recovery time. Over a third of patients had ICP values over 20mmHg at 5 minutes after the turn and 4 out of 7 of these children had poor six month outcomes or subsequently required a decompressive craniectomy. This outcome data may reflect the adverse effects reported of secondary insults in this patient group (Mazzola and Adelson 2002; Marmarou et al 1991; Carter et al 2008; Vik et al 2008; Catala-Temprano et al 2007; Kirkness et al 2008; Balestreri et al 2006; Treggiari et al 2007) but as discussed previously with regard to suctioning, outcome after severe TBI is not just related to secondary injury or ICP alone.

10.20 Comparison of endotracheal suctioning and turning as noxious stimuli in children with moderate to severe TBI

Both endotracheal suctioning and turning are essential and relatively routine nursing procedures undertaken in all intensive care children, but in moderate to severe TBI these procedures are noxious and can significantly increase ICP. During endotracheal suction, the primary significant confounder was the lack of control over the technique of manual hand ventilation of the patient. The sensitivity of these patients ICP response to changes in carbon dioxide level is well documented (Skippen et al 1997; Coles et al 2007) and may, in part, explain the lower level of maximal ICP seen during suctioning (compared to turning) and the more rapid recovery times. The children were not hand ventilated during turning and with this confounder removed; a stronger relationship was seen between baseline ICP and maximal ICP, maximal ICP and post procedure ICP and maximal ICP and time taken to recover baseline ICP. This probably reflects a more accurate picture of the effects of a noxious stimulus applied to these patients in the early course of their injury (except perhaps in a sub group of severely ill children requiring thiopentone). In particular that a noxious stimulus applied should produce a linear rise in ICP, so that children who had higher baseline ICP values demonstrate a higher maximal ICP. Both these procedures have the potential to induce intracranial hypertension, albeit for most, only transiently, but if prolonged can act as a secondary injury insult, which has been shown to affect mortality and long term patient

outcome (Mazzola and Adelson 2002; Marmarou et al 1991; Carter et al 2008; Vik et al 2008; Catala-Temprano et al 2007; Kirkness et al 2008; Balestreri et al 2006; Treggiari et al 2007).

10.21 The effect of other nursing interventions

Neither eye care nor mouth care produced any clinically significant effects on ICP in these children, suggesting these procedures are not noxious and are tolerated very well. However, there were only a small number of washing episodes in nine children to analyse in this study and so no real conclusions can be drawn about the effects of this intervention. Further research is needed in relation to the effects of washing in children, as Parsons et al (1985) who studied the affect of various hygiene interventions (oral hygiene, body hygiene and urinary catheter care) in 19 head injured adults, found that all three interventions produced significant mean increases in all physiological parameters ($p<0.005$) when compared to baseline. They noted however, that all these elevations had retuned to baseline within one minute post intervention. Other work which has studied these interventions has done so in combination with other interventions (as a care cluster) thus the individual effects of these have been impossible to calculate (Hugo 1987; Hobdell et al 1989; Bruya 1981).

10.22 The effect of nursing interventions after decompressive craniectomy

There is no previous published work on the effects of nursing interventions in children (or adults) after decompressive craniectomy. In this case series of children no nursing intervention produced any clinically significant effects (defined as ICP > 20mmHg). Although there was a trend to elevation in ICP during suctioning and turning, neither exceeded 20mmHg. This group (as expected) exhibited lower baseline ICP values (mean 10–14mmHg) and their ICP response to interventions was minimal, reflecting the physiology now of an open cranial vault and pressure changes being able to be dissipated with minimal restriction. It is important to demonstrate this, because decompressive craniectomy has come into favour again, especially in younger patients, usually as part of large randomised controlled trials (Hutchinson et al 2006). Thus these findings alter the recommendations for nursing management of these children compared to the closed head injury group. This is significant because as previously discussed (in chapter nine) due to the lack of published evidence in this group of children, they are currently managed in the same way as the close head injured group, when it may be that they do not require any additional sedatives or other drugs prior to nursing interventions.

10.23 Summary of key findings of the thesis

In summarising the key findings of this thesis, it has been shown that the two commonly performed and essential nursing interventions of endotracheal suctioning (with manual ventilation) and turning (via log-rolling) produce significant intracranial instability in the majority of children. In most children this intracranial hypertension is short lived, but, in a minority of children, these interventions may be contributing to secondary injury insults. The effect of log-rolling (on duration of raised ICP) appeared slightly worse than suctioning, causing an elevation beyond their baseline in three quarters of children and longer recovery times. The interventions of eye care, mouth care and washing the child appeared to have little effect on the child's ICP. The challenge now is to disseminate these findings to the appropriate audience (clinical PICU nurses) and encourage them to incorporate these findings into their practice guidelines. It would then be useful to re-audit UK PICUs in two years' time to ascertain if practices have altered.

10.24 Limitations of this study

Several limitations of our study warrant consideration. Firstly because of the observational nature of the study, there was no strict 'control' over the way the interventions were performed. Although the time taken for the intervention was recorded, no account was taken of how many suction passes were used and whether saline was instilled during suctioning.

Additionally although there are generic guidelines for the performance of endotracheal suctioning on the PICU (and specific guidelines for the performance of suctioning in this group of children - Appendix seven) which all staff are trained according to, as an observational study the technique of suctioning was not standardised. Thus there will have been some variations in suctioning technique between nurses which we did not account for and this may have affected the results we have shown with this intervention. Secondly, when separate groups were compared, the groups were neither randomly allocated nor equal. This meant, for example with ET Lignocaine, there were only five children in the group who did not receive the drug, which weakens the data analysis and potentially introduces bias. Thirdly, having multiple data collectors may increase variability, but, testing the inter-observer reliability, demonstrated this was not a concern. There are, of course, also significant limitations of using bedside nurses as the primary data collectors because of the possibility of distractions, clinical urgency (eg sudden patient deterioration), and a lack of concentration over time. All of this may impair data quality. Another way to improve the data collection would be to directly export the data from the bedside monitor to a laptop. This was not possible at the author's unit. Further, as the majority of children were receiving neuromuscular blockade there was no way of quantifying their level of sedation (the COMFORT score is only validated for use in non-paralysed children). Another limitation was the lack of formal neuropsychological follow up (and scoring) of six month outcome by a

neuropsychologist. Instead this was done by the researcher by chart review. Finally, despite data collection over a three year period the sample is relatively small, which reflects the low number of children with moderate to severe TBI in the UK (Parslow et al 2005) and this had limitations for the analysis we were able to undertake. Two explanatory variables were added to our multivariate linear regression model, so it was not over-fitted, but with a larger sample size more variables, in principle, could have been added. Additionally, a larger sample would have allowed us to undertake more detailed inferential data analysis for all nursing interventions. Despite these limitations, the current study is the first to examine the effects of endotracheal suctioning and manual ventilation and log-rolling specifically in a group of children with moderate to severe TBI. It is also the first to report the effects of these interventions in patients after decompressive craniectomy. Additionally, because of the observational nature of the study it was possible to include children with intracranial hypertension necessitating second tier therapies and observe their response to procedures. In an experimental study these patients are likely to have been excluded for ethical reasons.

The registration of this study as audit (by the NHS Trust) meant no registration on the former NHS research database. This may have implications on the awareness by other researchers of this study being undertaken.

10.25 Implications and recommendations for practice

Our results give some estimate as to the physiological impact of these 'routine procedures' on intracranial pressure in children specifically in the hyper acute phase of TBI. In particular they may act as an early indicator of the patients who have more severe injuries (or who are higher up the pressure-volume curve with worse cerebral compliance) and may exhibit poorer outcomes, acting perhaps as an early 'stress test'. This thesis provides some addition to the weak evidence base in this area.

A recent survey of UK practice showed wide variations in nursing practices with regard to all nursing interventions (Tume and Baines 2008) including no guidance at all in the majority of cases. Most PICUs made vague recommendations for performing nursing cares, the majority implied that all cares were clustered (likely to be causing increased duration of time with intracranial hypertension if combining both suctioning and turning together) which may be harmful. In fact our results with log-rolling showed that the longer the intervention time (and hence a sustained rise in ICP) the longer the children took to recover, which would suggest that the combining of suctioning and turning procedures together could not be recommended. The frequency of log-rolling varied considerably between units, and there was no mention of any precautions with this intervention at all. Another important point that emerged from this audit work was that even when reasonable class II evidence existed (in relation to location of nursing these children in terms of the effect of environmental stimuli on ICP) most PICUs

had not incorporated this (relatively old 1990s) evidence into any practice guidelines. This lack of translation of evidence into nursing practice is a frequently reported concern (Maguire 2006) due to multiple reasons. Nursing research findings are not always widely disseminated to the appropriate audience (clinical nurses); clinical nurses working at the bedside do not always read published research and possibly lack the skills to establish what good evidence is and then incorporate it into unit guidelines. Nurses are often guilty of following established historical practices, rather than striving to find best evidence and incorporate this into their practice.

Recommendations for nursing practice suggested from this study would be:

- To plan suctioning and turning interventions carefully in the children without decompressive craniectomies
- To only perform suctioning when clinically indicated but to remember there will be a transient rise in ICP in the majority of children
- To try to reduce baseline ICP to less than 20mmHg before turning or suctioning where possible
- To limit the duration of these interventions to as short as possible
- To allow enough time between suctioning and turning for the child to recover their baseline ICP or at least to an ICP < 20mmHg
- To administer an extra sedation and/or analgesia IV bolus before suctioning and turning allowing enough time (ie ~ 20 minutes for peak effect) for the drug to act before the interventions

- In children with multiple injuries, before turning, one of these drugs should be an opiate analgesic to minimise the risk of pain
- To aggressively treat an ICP > 20mmHg that persists for more than five minutes after the intervention
- To take extra precautions when suctioning children who require thiopentone (they are children with worse injuries who are likely to exhibit a higher ICP response and prolonged recovery time). Aggressive treatment of ICP to under the treatment threshold may be required after suctioning these children
- Eye care and mouth care can be safely performed at any time in these children and should be undertaken regularly to minimise the adverse effects of critical illness and its therapies
- After decompressive craniectomy suctioning, turning, eye care, mouth care and washing appear to be able to be safely performed at any time and should be undertaken regularly to minimise the adverse effects of critical illness and its therapies
- PICUs should produce clear nursing management guidelines especially for performing endotracheal suctioning and turning in these children, emphasising the above precautions (as in our previous audit 62% of PICUs had no such guidelines)
- A 'minimum handling' approach (which implies care clustering) may not be the best approach to nursing cares in these children, and it

would seem sensible that endotracheal suctioning and log-rolling be spaced well apart to avoid any sustained elevation in ICP

- To hold a national consensus meeting to develop and agree nursing management guidelines for children with severe TBI

Clearly both suctioning and turning are essential, but as the ICP during both ETSMV and turning does exceed the clinical treatment threshold of 20mmHg in the majority of children, it can be said that these procedures are not innocuous and need to be carefully planned and executed skilfully to minimize these potential secondary injury induced changes. It is suggested that these two interventions should not be performed together as they may increase the duration of time that ICP is raised and may also increase recovery time. Raised ICP is a recognized cause of secondary injury, which in turn has been shown to significantly affect patient outcome (Smith 2009; Mazzola and Adelson 2002; Marmarou et al 1991; Carter et al 2008; Vik et al 2008; Catala-Temprano et al 2007; Kirkness et al 2008; Balestreri et al 2006; Treggiari et al 2007).

10.26 Implications for future research

Further experimental studies (using multi-centres to achieve larger samples) need to be undertaken specifically in children to examine the effect of endotracheally administered lignocaine prior to ETSMV in this group of children (along with pharmacokinetic studies). Recent adult work (Bilotta et

al 2008) has shown this can (if given in the adequate dose) minimise the rise in ICP during suctioning.

Previous work has failed in its attempts to explore the effect of clustering nursing cares together compared to spacing them apart, but with larger samples, and with some evidence now presented as to the effects of individual nursing interventions it may be possible to design a study to examine this. However it is clear that it is only the interventions of suctioning and turning that need to be clustered. However the possible risk of secondary injury in the clustering group may have to be taken into account as an ethical consideration.

As no pain or sedation tools currently exist that can be used in children receiving neuromuscular blockade, it would be useful to try and quantify sedation level by other means, possibly Bispectral Index (BIS) in this group of children prior to interventions (Crain et al 2002; Courtman et al 2003; Aneja et al 2003; Lamas et al 2008). If this was explored it may provide greater insight into which children needed additional sedation and analgesia IV boluses prior to an intervention and if these boluses had been effective.

It would also be interesting to examine exactly how many intracranial hypertension episodes occurring in PICU are actually 'triggered' by a routine care intervention (this is not currently not known).

Given that log-rolling is a noxious stimuli in these children and the UK audit showed huge variations in the time between turns (from 2-3 hourly to 24 hourly) (Tume and Baines 2008), a further area for investigation would be to determine what the minimum time between turns should be in this group of children to prevent the development of pressure areas. This is important because more frequent (beyond what is necessary) turning may be contributing unnecessarily to secondary injury insults.

Further work is required to determine the effects of hygiene interventions in severe head injured children in a larger sample.

As the performance of decompressive craniectomy increases due to participation in multi-centre trials (RESCUEicp) it would be helpful to examine the response of this group of children in more detail in relation to specific care practices such as turning and suctioning. To ensure a more homogenous group it would be preferable to study these children at regular and consistent time points eg Day 1 post DC, Day 2 post DC.

10.27 Implications for intensive care nurses

The primary aim of the intensive care management of severe TBI is the minimisation of secondary injury of cerebral oedema and worsening cerebral ischaemia (Reilly and Bullock 2005 p294; Chambers et al 2006). It has already been highlighted that the intensive care nurse has a key role to play in recognising and minimising these secondary injury processes, which

can significantly affect the child's outcome and in promoting intracranial pressure (ICP) and cerebral perfusion pressure (CPP) stability whenever possible. This often presents a dilemma for the nurse, who, just by performing essential nursing interventions or 'cares' for the child, may in fact produce significant physiological instability. The intensive care nurse often faces difficult decisions about the best method to undertake these necessary cares, the timing and how to best minimise the adverse effects of these interventions. It is intended that the results of this thesis will provide some guidance to nurses about quantifying the effects of some 'routine' interventions and allow the nurse to plan and execute these interventions with more evidence. This study provides some class II evidence and will assist in the development of some evidence based nursing guidelines for paediatric TBI management, although clearly more class I evidence is required particularly for areas such as the use of ET Lignocaine. It will also form the basis for future experimental work in this field.

Intensive care nurses have a key role to play in maintaining the physiological stability in these children by preventing these secondary insults and in 'humanising' this highly foreign environment for the parents and family. It is claimed that nurses deliver more than 85% of the 'hands on' care to critically ill patients, yet little research is directed towards nursing care (Peters 1992). It is suggested that if intensive care nurses want to advance professional nursing knowledge and practice, and aim to provide

best care with minimal harm, then we have a duty to conduct research into these aspects of seemingly basic, yet fundamental, care for vulnerable critically ill children.

CONCLUSION

This thesis has explored the effects of selected 'routine' intensive care unit nursing interventions in children with moderate to severe traumatic brain injury in an observational study. It has generated new class II evidence to begin to provide an evidence base to some relatively 'routine' nursing practices within the intensive care unit. The new knowledge describes, for the first time, the effects of turning via a log-rolling approach and the effects of selected routine interventions (endotracheal suctioning, turning, eye care, mouth care and washing) in children after decompressive craniectomy. In addition, the thesis has begun to quantify the magnitude of effects of all these interventions and their recovery times specifically in a population of children with moderate to severe traumatic brain injury in the hyper acute phase of their injury, in intensive care.

The methodology used has attempted to address many of the problems and limitations identified with previous work, and the study reporting conforms to the STROBE guidelines for the reporting of observational studies (von Elm et al 2007). However there are limitations of this study and these have been identified.

The thesis has met its aims and answered the research questions as set out and it has related the results to current PICU nursing practice and suggested recommendations for practice and future research. The

researcher has learnt a considerable amount during the undertaking of this thesis and published parts of the thesis throughout, with a final publication of the results submitted to Pediatric Critical Care Medicine, a high impact journal in this field.

It has been previously suggested that if intensive care nurses want to advance professional nursing knowledge and practice, and aim to provide best care with minimal harm, then we have a duty to conduct research into these fundamental nursing care practices. This was an important issue to the researcher as an intensive care nurse to be able to study phenomena that is undertaken regularly to generate answers to clinically relevant questions.

REFERENCES

REFERENCE LIST

ADELSON D, BRATTON S, CARNEY N, CHESNUT R, DU COUDRAY H, GOLDSTEIN B, KOCHANEK P, MILLER H, PARTINGTON M, SELDEN N, WARDEN C & WRIGHT D (2003) Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents *Ped Crit Care Med,* 4, S28-30.

ADELSON D & KOCHANEK P (1998) Head Injury in Children. *Journal of Child Neurology,* 13, 2-13.

ADELSON D, RAGHEB J, MUIZELAAR J, KANEV P, BROCKMEYER D, BROCKMEYER D, BEERS S, BROWN D, CASSIDY L, CHANG Y & LEVIN H (2005) Phase II Clinical Trial of Moderate Hypothermia Severe Traumatic Brain Injury in Children. *Neurosurgery,* 56, 740-753.

AHMAD M (2001) Cervical collars and intracranial pressure. *Emergency Medicine Journal,* 18, 380-381.

ALDERSON P, GADKARY C & SIGNORINI D (2005) Therapeutic Hypothermia for Head Injury. *The Cochrane Collaboration,* 4.

AL-TAMIMI Y, HELMY A, BAVETTA S & PRICE S (2009) Assessment of zero drift in the Codman intracranial pressure monitor: a study from 2 neurosurgical units. *Neurosurgery,* 64, 94-99.

ALTMAN D and BLAND M (1998) Time to event (survival) data. *BMJ,* 317, 468-469.

AMBUEL B, HAMLETT K, MARX C & BLUMER J (1992) Assessing Distress in Pediatric Intensive Care Environments: The COMFORT scale. *Journal of Pediatric Psychology,* 17, 95-109.

ANDERSON V (2007) New Frontiers in pediatric traumatic brain injury. *Dev Neurorehabilitation,* 10, 269-270.

ANDRUS C (1991) Intracranial pressure: Dynamics and nursing management. *J Neurosci Nurs,* 23, 85-92.

ANEJA R, HEARD A, FLETCHER J & HEARD C (2003) Sedation monitoring of children by the Bispectral Index in the pediatric intensive care unit. *Ped Crit Care Med,* 4, 60-64.

ANTHONY D (1999) *Understanding Advanced Statistics – a guide for nurses and healthcare researchers,* Churchill Livingstone, Edinburgh.

ARBOUR R (2004) Using Bispectral Index Monitoring to detect potential breakthrough awareness and limit duration of neuromuscular blockade. *Am J Crit Care,*13 (1), 66-73.

ARNOLD D, BURNS K, ADHIKARI N, KHO M, MEADE M, COOK D & FOR THE MCMASTER CRITICAL CARE INTEREST GROUP (2009) The design and interpretation of pilot trials in clinical research in critical care. *Crit Care Med,* 37, S69-S74.

BAHLOUL M, HAMIDA C, CHELLY H, CHAARI A, KALLEL H, DAMMAK H, REKIK N, BAHLOUL K, MAHFOUDH B, HACHICHA M & BOUAZIZ M (2009) Severe Head Injury among children: Prognostic factors and outcome *Int J Care Injured*, 40, 535-540.

BAILAR J and MONSELLER F (1992) *Medical Uses of Statistics.*2nd Edn, NEJM Books, Boston, US.

BALESTRERI M, CZOSNYKA M, CHATFIELD D, STEINER L, SCHMIDT E, SMIELEWSKI P, MATTA B & PICKARD J (2004) Predictive value of Glasgow coma scale after brain trauma: change in trend over the past ten years. *J Neurol Neurosurg Psychiatry,* 75, 161-162.

BALESTRERI M, CZOSNYKA M, HUTCHINSON P, STEINER L, HILER M, SMIELEWSKI P & PICKARD J (2006) Impact of intracranial pressure and cerebral perfusion pressure on severe disability and mortality after head injury. *Neurocrit Care,* 4, 8-13.

BELLIENI C, BURRONI A, PERRONE S, CORDELLI D, NENCI A, LUNGHI A & BUONOCORE G (2003) Intracranial pressure during procedural pain. *Biol Neonate,* 84, 202-205.

BHARDWAJ A, MIRSKI M & ULATOWSKI J (2004) *Handbook of Neurocritical Care,* Totowa, NJ, Humana Press.

BILOTTA F, BRANCA G, LAM A, CUZZONE V, DORONZIO A & ROSA G (2008) Endotracheal Lidocaine in Preventing Endotracheal Suctioning-induced Changes in Cerebral Hemodynamics in Patients with Severe Head Trauma. *Neurocrit Care,* 8, 241-246.

BLAND, M (2003) *An introduction to medical statistics.* 3rd Edn Oxford University Press, Oxford UK.

BLAND M & ALTMAN D (1986) Statistical methods for assessing agreement between two methods of clinical assessment. *The Lancet,* Feb 8, 307-310.

BLAND M & ALTMAN D (1999) Measuring Agreement in Method Comparison Studies. *Statistical Methods in Medical Research,* 8, 135-160.

BOORTZ-MARX, R. (1985) Factors Affecting Intracranial Pressure: a descriptive study. *J Neurosurgical Nursing,* 17, 89-94.

BOTO G, GOMEZ P, CRUZ J & LOBATO R (2009) A historical analysis of severe head injury. *Neurosurg Rev,* 32, 343-354.

BOURGOIN A, ALBANESE J, WERESZCZYNSKI N, CHARBIT M, VIALET R & MARTIN C (2003) Safety of sedation with Ketamine in severe head injury patients: Comparison with Sufentanil. *Crit Care Med,* 31, 711-717.

BREAN A, EIDE P & STUBHAUG A (2006) Comparison of intracranial pressure measured simultaneously within the brain parenchyma and cerebral ventricles. *J Clinical Monitoring and Computing,* 20, 411-414.

BRENNAN P & CROFT P (1994) Interpreting the results of observational research: chance is not such a fine thing. *BMJ,* 309, 727-730.

BROWN M, PARR M & MANARA A (1996) The effect of suxamethonium on intracranial pressure and cerebral perfusion pressure in patients with severe head injuries following blunt trauma. *European Journal of Anaesthesiology,* 13, 474-477.

BRUCIA J, OWEN D & RUDY E (1992) The Effects of Lidocaine on Intracranial Hypertension. *J Neurosci Nurs,* 24, 205-214.

BRUCIA J & RUDY E (1996) The effect of suction catheter insertion and tracheal stimulation in adults with severe brain injury. *Heart & Lung,* 25, 295-303.

BRUYA, M. (1981) Planned Periods of Rest in the Intensive care Unit: Nursing Care Activities and Intracranial Pressure. *Journal of Neuroscience Nursing,* 13, 184-194.

BUCK D & SUTTON M (1994) Interpreting the results of observational research. *BMJ,* 309, 1439.

BULGER E, NATHENS A, RIVARA F, MOORE M, MACKENZIE E & JURKOVICH G (2002) Management of severe head injury: institutional variations in care and effect on outcome. *Crit Care Med,* 30, 1870-1876.

CAMPBELL, V. (1991) Effects of Controlled Hyperoxygenation and Endotracheal Suctioning on Intracranial Pressure in Head-Injured Adults. *Applied Nursing Research,* 4, 138-40.

CARHUAPOMA J & WILLIAMS A (1999) Endotracheal suctioning and cerebral ischemia in traumatic brain injury: The "phantom menace"? *Crit Care Med,* 27, 2843-2844.

CARTER B, BUTT W & TAYLOR A (2008) ICP and CPP: excellent predictors of long term outcome in severely brain injured children. *Childs Nerv Syst,* 24, 245-251.

CATALA-TEMPRANO A, TERUEL G, LASAOSA F, ODENA M, JULIAN A & PALOMEQUE A (2007) Intracranial pressure and cerebral perfusion pressure as risk factors in children with traumatic brain injuries. *J Neurosurg,* 106, 463-466.

CHAMBERLAIN D (1998) The critical care nurse's role in preventing secondary brain injury in severe head trauma: achieving the balance. *Australian Critical Care,* 11, 123-129.

CHAMBERS I, JONES P, LO T, FORSYTH R, FULTON B, ANDREWS P, MENDELOW A & MINNS R (2006) Critical thresholds of intracranial pressure and cerebral perfusion pressure related to age in paediatric head injury. *J Neurol Neurosurg Psychiatry,* 77, 234-40.

CHAMBERS I, STOBBART L, JONES P, KIRKHAM F, MARSH M, MENDELOW A, MINNS R, STRUTHERS S & TASKER R (2005) Age-related differences in intracranial pressure and cerebral perfusion pressure in the first 6 hours of monitoring after children's head injury: association with outcome. *Childs Nerv Syst,* 21, 195-9.

CHRISTENFELD N, SLOAN R, CARROLL D & GREENLAND S (2004) Risk factors, Confounding and the Illusion of statistical control. *Psychosomatic Medicine,* 66, 868-875.

CHUDLEY, S. (1994) The effect of nursing activities on intracranial pressure. *Br J Nursing,* 3, 454-459.

CITERIO G & ANDREWS P (2007) Refractory elevated intracranial pressure: intensivist's role in solving the dilemma of decompressive craniectomy. *Intensive Care Med,* 33, 45-48.

CLAYTON T, NELSON R & MANARA A (2004) Reduction in mortality from severe head injury following introduction of a protocol for intensive care management. *British Journal of Anaesthesia,* 93, 761-767.

CLIFTON, G. (2004) Is keeping cool still hot? An update on hypothermia in brain injury. *Current Opinion in Critical Care,* 10, 116-119.

CLIFTON G, MILLER E, CHOI S, LEVIN H, MCCAULEY S, SMITH K, MUIZELAAR P, WAGNER F, MARION D, LUERSSEN T, CHESTNUT R & SCHWARTZ, M. (2001) Lack of effect of induction of hypothermia after acute brain injury. *NEJM,* 344, 556-563.

COATES B, VAVILALA M, MACK C, MUANGMAN S, SUZ P, SHARAR S, BULGER E & LAM A (2005) Influence of definition and location of hypotension on outcome following severe pediatric traumatic brain injury. *Crit Care Med,* 33, 2645-2650.

COLES J, FRYER T, COLEMAN M, SMIELEWSKI P, GUPTA, MINHAS P, AIGBIRHIO F, CHATFILED D, WILLIAMS G, BONIFACE S, CARPENTER A, CLARK J, PICKARD J & MENON D (2007) Hyperventilation following head injury: Effect on ischaemia burden and cerebral oxidative metabolism. *Crit Care Med,* 35, 568-578.

COOK A & WEANT K (2007) Pharmacologic strategies for the treatment of Elevated Intracranial Pressure - focus on metabolic suppression. *Advanced Emergency Nursing Journal,* 29, 309-318.

COURTMAN S, WARDURGH A & PETROS A (2003) Comparison of the bispectral index monitor with the Comfort score in assessing level of sedation of critically ill children. *Intensive Care Med,* 29, 2239-2246.

CRAIG G & NIELSEN M (1991) Rigid Cervical Collars and Intracranial Pressure. *Intensive Care Med,* 17, 504-505.

CRAIN N, SLONIM A & POLLACK M (2002) Assessing sedation in the pediatric intensive care unit by using BIS and the COMFORT score. *Ped Crit Care Med,* 3, 11-14.

CREMER O, VAN DIJK G, VAN WENSEN E, BREKELMANS G, MOONS K, LEENEN L & KALKMAN C (2005) Effect of intracranial pressure monitoring and targeted intensive care on functional outcome after severe head injury. *Crit Care Med,* 33, 2207-13.

CRICHTON N. (2001) Principles of statistical analysis in nursing and healthcare research. *Nurse Researcher,* 9, 4-16.

CROSBY L & PARSONS L (1992) Cerebrovascular Response of Closed Head-Injured Patients to a Standardised Endotracheal Tube Suctioning and Manual Hyperinflation Procedure. *Journal of Neuroscience Nursing,* 24, 40-49.

CUFF S, DIRUSSO S, SULLIVAN T, RISUCCI D, NEALON P, HAIDER A & SLIM, M. (2007) Validation of a relative head injury severity scale for pediatric trauma. *The Journal of Trauma, Injury, Infection and Critical Care,* 63, 172-178.

CURLEY M, QUIGLEY S & LIN M (2003) Pressure ulcers in pediatric intensive care: incidence and associated factors. *Ped Crit Care Med,* 4, 284-90.

DAVIES G, DEAKIN C & WILSON A (1996) The effect of a rigid cervical collar on intracranial pressure. *Injury,* 27, 647-649.

DEAN N, BOSLAUGH S, ADELSON D, PINEDA J & LEONARD J (2007) Physician agreement with evidence-based recommendations for the treatment of severe traumatic brain injury in children. *J Neurosurg,* 107, 387-391.

DEMELLWEEK C, BALDWIN T, APPLETON R & AL-KHARUSI A (2002) A prospective study and review of pre-morbid characteristics in children with traumatic brain injury. *Ped Rehab,* 5, 81-89.

DEPARTMENT OF ANAESTHESIOLOGY FACUTLY OF MEDICINE - PRINCE OF SONGKLA UNIVERSITY Perioperative Management of Common Trauma. Hat Yai Thailand. Retrieved 8.2.08 medinfo2.psu.ac.th/anesth/education/trauma2.html

DEREEPER E, BERRE J, VANDESTEENE A, LEFRANC F & VINCENT J (2002) Barbiturate Coma for Intracranial Hypertension: Clinical Observations. *Journal of Critical Care,* 17, 58-62.

DONEGAN M & BEDFORD R (1980) Intravenously Administered Lidocaine Prevents Intracranial Hypertension during Endotracheal Suctioning. *Anaesthesiology,* 52, 516-518.

DOWNWARD C, HULKA F, MULLINS R, PIATT J, CHESTNUT R, QUINT P & MANN C (2000) Relationship of Cerebral Perfusion Pressure and Survival in Pediatric Brain-Injured Patients. *The Journal of Trauma, Injury, Infection and Critical Care,* 49, 654-659.

DUHAIME A (2007) Why are clinical trials in pediatric head injury so difficult? *Ped Crit Care Med,* 8, 71-72.

DURAND M, SANGHA B, CABAL L, HOPPENBROUWERS T & HODGMAN J (1989) Cardiopulmonary and intracranial pressure changes related to endotracheal suctioning in preterm infants. *Crit care Med,* 17, 506-510.

EISENBERY H, FRANKOWSKI R, CONTANT C, MARSHALL L & WALKER M (1988) High-dose barbiturate control of elevated intracranial pressure in patients with severe head injury. *J Neurosurgery,* 69, 15-23.

ERSSON U, CARLSON A, MELLSTROM U, PONTEN U, HEDSTRAND U & JAKOBSSON S (1990) Observations on intracranial dynamics during respiratory physiotherapy in unconscious neurosurgical patients. *Acta Anaesthesiol Scand,* 34, 99-103.

FAN J (2004) Effect of backrest position on intracranial pressure and cerebral perfusion pressure in individuals with brain injury: a systematic review. *J Neurosci Nurs,* 36, 278-288.

FELDMAN Z, KANTER M, ROBERTSON C, CONTANT C, HAYES C, SHEINBERG M, VILLAREAL C, NARAYAN R & GROSSMAN R (1992) Effect of head elevation on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow in head-injured patients. *J Neurosurgery,* 76, 207-211.

FISHER D, FREWEN T & SWEDLOW D (1982) Increase in Intracranial Pressure during suctioning - Stimulation versus rise in PaCo2. *Anaesthesiology,* 57, 416-417.

FRANCK L, GREENBERG C & STEVENS B (2000) Pain assessment in infants and children. *Pediatric Clinics of North America,* 47, 487-512.

FRISON L & POCOCK S (1992) Repeated measures in clinical trials: analysis using mean summary statistics and its implications for design. *Statistics in Medicine,* 11, 1685-1704.

FUKUDA S & WARNER D (2007) Cerebral Protection. *British Journal of Anaesthesia,* 99, 10-17.

GAMBARELLA G, ZACCONE C, CARDIA E & TOMASELLO F (1993) Intracranial pressure monitoring in children: comparison of external ventricular device with the fibre optic system. *Child's Nervous System,* 9, 470-473.

GARRARD J & BULLOCK M (1986) The Effect of Respiratory Therapy on Intracranial Pressure in Ventilated Neurosurgical Patients. *The Australian Journal of Physiotherapy,* 32, 107-111.

GEMMA M, TOMMASINO C, CERRI M, GIANOTTI A, PIAZZI B & BORGHI T (2002) Intracranial Effects of Endotracheal Suctioning in the Acute Phase of Head Injury. *Journal of Neurosurgical Anaesthesiology,* 14, 50-54.

GEORGIADIS D, SCHWARZ S, BAUMGARTNER R, VELTKAMP R & SCHWAB S (2001) Influence of Positive End-Expiratory Pressure on Intracranial pressure and Cerebral Perfusion pressure in Patients with Acute Stroke. *Stroke,* 32, 2088-2092.

GHAJAR J, HAIRI R & PATTERSON R (1993) Improved outcome from traumatic coma using only ventricular cerebrospinal fluid drainage for intracranial pressure control. *Advances in Neurosurgery,* 21, 173-177.

GILL M, REILEY D & GREEN S (2004) Interrater Reliability of Glasgow Coma Scale Scores in the Emergency Department. *Annals of Emergency Medicine,* 43, 215-223.

GILLON R (1994) Medical ethics: four principles plus attention to scope. *BMJ,* 309, 1-13.

GIZA C, MINK R & MADIKIANS A (2007) Pediatric traumatic brain injury: not just little adults. *Current Opinion in Critical Care,* 13, 143-152.

GOUGH S, TUME L & SINHA A (2007) Efficacy of CSF drainage on ICP reduction after severe paediatric traumatic brain injury. Poster Presentation at *5th World Congress on Pediatric Intensive Care.* Geneva, Switzerland.

GREENLAND, S. (1990) Randomization, Statistics and casual inference. *Epidemiology,* 1, 421-429.

GRINKEVICIUTE D, KEVALAS R, SAFERIS V, MATUKEVICIUS A, RAGAISIS V & TAMASAUSKAS A (2007) Predictive value of scoring system in pediatric head injury. *Medicina (Kaunas),* 43, 861-9.

HABER M & BARNHART H (2008) A general approach to evaluating agreement between two observers or methods of measurement from quantitative data with replicated measurements. *Statistical Methods in Medical Research,* 17, 151-169.

HALL C (1997) Patient management in head injury care: a nursing perspective. *Intensive & Critical Care Nursing,* 13, 329-337.

HAMILL J, BEDFORD R, WEAVER D & COLOHAN A (1981) Lidocaine before endotracheal intubation: Intravenous or Laryngotracheal? *Anaesthesiology,* 55, 578-581.

HANOWELL L, THURSTON J, BEHRMAN K & DISBROW E (1993) Alfentanil Administered Prior to Endotracheal Suctioning Reduces Cerebral perfusion pressure. *J Neurosurgical Anaesthesiology,* 5, 31-35.

HARRIS M & TAYLOR G (2004) *Medical Statistics made easy,* London, Taylor and Francis

HELMY A, VIZCAYCHIPI M & GUPTA A (2007) Traumatic Brain Injury: intensive care management. *British Journal of Anaesthesia,* 99, 32-42.

HENDRICKSON, S. (1987) Intracranial Pressure Changes and family Presence. *J Neurosci Nurs,* 19, 14- 17.

HO A, FUNG K, JOYNT G, KARMAKAR M & PENG Z (2002) Rigid Cervical Collar and Intracranial Pressure of Patients with Severe Head Injury. *J Trauma,* 53, 1185-1188.

HOBDELL E, ADAMO F, CARUSO J, DIHOFF R, NEVELING E & RONCOLI M (1989) The Effect of Nursing Activities on the Intracranial Pressure of Children. *Critical Care Nurse,* 9, 75-79.

HSIANG J, CHESTNUT R, CRISP C, KLAUBER M, BLUNT B & MARSHALL L (1994) Early, routine paralysis for intracranial pressure control in severe head injury: is it necessary? *Crit Care Med,* 22, 1471-1476.

HUGO, M. (1987) Alleviating the effects of care on the intracranial pressure (ICP) of head injured patients by manipulating nursing care activities. *Intensive Care Nursing,* 3, 78-82.

HUTCHINSON J, WARD R, LACROIX J, HERBERT P, BARNES M, BOHN D, DIRKS P, DOUCHETTE S, FERGUSSON D, GOTTESMAN R, JOFFE A, KIRPALANI H, MEYER P, MORRIS K, MOHER D, SINGH R, SKIPPEN P & FOR THE HYPOTHERMIA PEDIATRIC HEAD INJURY TRIAL INVESTIGATORS AND THE CANADIAN CRITICAL CARE TRIALS GROUP (2008) Hypothermia Therapy after Traumatic Brain Injury in Children *The New England Journal of Medicine,* 358, 2447-2256.

HUTCHINSON P, CORTEEN E, CZOSNYKA M, MENDELOW A, MENON D, MITCHELL P, MURRAY G, PICKARD J, RICKLES E, AHUQUILLO J, SERVADEI F, TEASDALE G, TIMOFEEV I, UNTERBERG A & KIRKPATRICK P (2006) Decompressive Craniectomy in traumatic brain injury: the randomized multicentre RESCUEicp study (www.RESCUEicp.com). *Acta Neurochir (Wien),* 96 (Supp), 17-20.

HUYNH T, MESSER M, SING R, MILES W, JACOBS D & THOMASON M (2002) Positive end-expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury. *J Trauma,* 53, 488-92; discussion 492-3.

IMLE C, MARS M, CIESLA N, ANDERSON P & DELANEY P (1997) The effect of Chest Physical Therapy on Intracranial Pressure and Cerebral Perfusion Pressure. *Physiotherapy Canada,* 49, 48-55.

JENNETT B & BOND M (1975) Assessment of Outcome after severe brain damage. *The Lancet,* March 1, 480-484.

JETZKI S (2005) ICP measurement and signal analysis. University of Aachen: Retrieved 6.5.09 http://www.medit.hia.rwthaachen.de/images/research/icpanalysis_brainpressure.gif

JOHNSON L (1999) Factors known to raise intracranial pressure and the associated implications for nursing management. *Nursing in Critical Care,* 4, 117-120.

JONES, B. (1995) The effects of patient repositioning on intracranial pressure. *The Australian Journal of Advanced Nursing,* 12, 32-39.

KEEFE MARCOUX K (2005) Management of increased intracranial pressure in the critically ill child with an acute neurological injury. *AACN Clin Issues,* 16, 212-31.

KEENAN H & BRATTON S (2006) Epidemiology and Outcomes of Pediatric traumatic Brain Injury. *Dev Neuroscience,* 28, 256-263.

KELLY D, GOODALE D, WILLIAMS J, HERR D, CHAPPELL T, ROSNER M, JACOBSON J, LEVY M, CROCE M, MANIKER A, FULDA G, LOVETT J, MOHAN O & NARAYAN R (1999) Propofol in the treatment of moderate and severe head injury: a randomised, prospective double-blinded pilot trial. *J Neurosurg,* 90, 1042-1052.

KERR M, SEREIKA S, ORNDOFF P, WEBER B, RUDY E, MARION D, STONE K & TURNER B (1998) Effect of Neuromuscular Blockers and Opiates on the Cerebrovascular Response to Endotracheal Suctioning in Adults with severe Head Injuries. *American Journal of Critical Care,* 7, 205-217.

KERR M, MARION D, SEREIKA S, WEBER B, ORNDOFF P, HENKER R & WILBERGER J (2000) The effect of cerebrospinal fluid drainage on cerebral perfusion in traumatic brain injured adults. *Journal of Neurosurgical Anaesthesiology,* 12, 324-333.

KERR M, RUDY E, WEBER B, STONE K, TURNER B, ORNDOFF P, SEREIKA S & MARION D (1997) Effect of short-duration hyperventilation during endotracheal suctioning on intracranial pressure in severe head-injured adults. *Nurs Res,* 46, 195-201.

KERR M, RUDY E, BRUCIA J & STONE K (1993) Head-injured Adults: Recommendations for Endotracheal Suctioning. *J Neurosci Nurs,* 25, 86-91.

KERR M, WEBER B, SEREIKA S, DARBY J & MARION D (2001) Dose response to cerebrospinal fluid drainage on cerebral perfusion in traumatic brain-injured adults. *Neurosurg Focus,* 11, 1- 7.

KERR M, WEBER B, SEREIKA S, DARBY J, MARION D & ORNDOFF P (1999) Effect of endotracheal suctioning on cerebral oxygenation in traumatic brain-injured patients. *Crit Care Med,* 27, 2776 -2781.

KERR M, ZEMPSKY J, SEREIKA S, ORNDOFF P & RUDY E (1996) Relationship between arterial carbon dioxide and end-tidal carbon dioxide in mechanically ventilated adults with severe head trauma. *Crit Care Med,* 24, 785-790.

KESTIN, I. (2006) Statistics for clinical trials and audits. *Anaesthesia and Intensive care Med,* 7, 337-341.

KHANNA S, DAVIS D, PETERSON B, FISHER B, TUNG H, O'QUIGLEY J & DEUTSCH R (2000) Use of hypertonic saline in the treatment of severe refractory posttraumatic intracranial hypertension in pediatric traumatic brain injury. *Crit Care Med,* 28, 1144-1151.

KIRKNESS C, BURR R & MITCHELL P (2008) Intracranial pressure variability and long term outcome following traumatic brain injury. *Acta Neurochir (Supp),* 102, 105-108.

KNAPP J (2005) Hyperosmolar Therapy in the treatment of severe head injury in children. *AACN Clin Issues,* 16, 199-211.

KOCHANEK P (2005) Pediatric Traumatic Brain Injury: Beyond the Guidelines. *Current Treatment Options in Neurology,* 7, 441-450.

KOLB J, SUMMERS R & GALLI R (1999) Cervical Collar-Induced Changes in Intracranial Pressure. *American Journal of Emergency Medicine,* 17, 135-137.

KOSKINEN L & OLIVECRONA M (2005) Clinical experience with the intraparenchymal intracranial pressure monitoring Codman MicroSensor system. *Neurosurgery,* 56, 693-8.

KUO J, WANG C, CHIO C & CHENG T (2004) Neurosurgical improvement after cranioplasty - analysis by transcranial ultrasonography. *Journal of Clinical Neuroscience,* 11, 486-489.

LAMAS A, LOPEZ-HERCE J, SANCHO L, MENCIA S, CARRILLO A, SANTIAGO M & MARTINEZ V (2008) Assessing sedation in critically ill children by bispectral index, auditory-evoked potentials and clinical scales. *Intensive Care Med,* 34, 2092-2099.

LANCASTER G, DODD S & WILLIAMSON P (2004) Design and analysis of pilot studies: recommendations for good practice. *Journal of Evaluation in Clinical Practice,* 10, 307-312.

LANNOO E, COLARDYN F, DE DEYNE C, VANDEKERCKOVE T, JANNES C & SOETE G (1998) Cerebral perfusion pressure and intracranial pressure in relation to neuropsychological outcome. *Intensive Care Med,* 24, 236-241.

LEE, S. (1989) Intracranial pressure changes during positioning of patients with severe head injury. *Heart & Lung,* 18, 411-414.

LEONE M, ALBANESE J, VIVIAND X, GARNIER F, BOURGOIN A, BARRAU K & MARTIN C (2004) The Effects of Remifentanil on Endotracheal Suctioning-Induced Increases in Intracranial pressure in Head-injured adults. *Anaesthesia and Analgesia,* 99, 1193-8.

LODI C, TER MINASSIAN A, BEYDON L & URSINO M (1998) Modelling cerebral autoregulation and Co2 reactivity in patients with severe head injury. *Am J Physiol Heart Circ Physiol,* 274, 1729-1741.

LUNDBERG, N. (1960) Continuous recording and control of ventricular fluid pressure in neurosurgical practice. *Acta Psychiatrica Et Neurologica Scand,* 149, 1-184.

MACGUIRE J (2006) Putting research findings into practice: research utilization as an aspect of the management of change. *Journal of Advanced Nursing,* 53, 65-74.

MACKWAY-JONES K, MOLYNEUX E, PHILLIPS B & WIETESKA S (Eds.) (2005) *Advanced Paediatric Life Support - The practical approach,* Oxford, Blackwell Pub.

MACNAB A, MACRAE D & HENNING R (1999) *Care of the Critically Ill Child,* London, Churchill Livingstone.

MANSFIELD, R. (2007) Severe Traumatic Brain Injuries in Children. *Pediatric Emergency Medicine,* 8, 156-164.

MARHALL I, MARSHALL S, KLAUBER M (1992) The diagnosis of head injury requires a classification based on computerised axial tomography. *J Neurotrauma,* 9; 287-292.

MARAMOU A, ANDERSON R, WARD J, CHOL S & YOUNG H (1991) Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg,* 75, S59-S66.

MARION D & CARLIER P (1994) Problems with the initial Glasgow Coma Scale assessment caused by prehospital treatment of patients with head injuries: results of a national survey. *The Journal of Trauma,* 36, 89-95.

MASCIA L, GRASSO S, FIORE T, BRUNO F, BERNADINO M & DUCATI A (2005) Cerebro-pulmonary interactions during the application of low levels of positive end-expiratory pressure. *Intensive Care Med,* 31, 373-379.

MAYER S & CHONG J (2002) Critical Care Management of Increased Intracranial Pressure. *J Intensive Care Medicine,* 17, 55-67.

MAZZOLA C & ADELSON D (2002) Critical care management of head trauma in children. *Crit Care Med,* 30, s393-s401.

MCCLELLAND M, WOSTER P, SWEASEY T & HOFF J (1995) Continuous Midazolam/Atracurium Infusions for the Management of Increased Intracranial Pressure. *J Neurosci Nurs,* 27, 96- 101.

MCCLUSKEY A & LALKHEN A (2007) Statistics I: data and correlations. *Continuing Education in Anaesthesia, Critical Care & Pain,* 7, 95-99.

MCILVOY L (2007) The impact of brain temperature and core temperature on intracranial pressure and cerebral perfusion pressure. *J Neurosci Nurs,* 39, 324-331.

MCLEAN, C. (2001) Moving and positioning patients with altered intracranial haemodynamics. *Nursing in Critical Care,* 6, 239-244.

METESCKY A (2007) Management of severe traumatic brain injury: the need for the knowledgeable nurse. *British Journal of Neuroscience Nursing,* 3, 7-13.

MITCHELL P, OZUNA J & LIPE H (1981) Moving the Patient in Bed: Effects of Intracranial Pressure. *Nursing Research,* 30, 212-218.

MITCHELL P (1986) Intracranial hypertension: Influence of Nursing care Activities. *Nurs Clinics of North America,* 21, 563-576.

MITCHELL P & HABERMAN B (1999) Rethinking Physiologic Stability: Touch and Intracranial Pressure. *Biological research for Nursing,* 1, 12-19.

MITCHELL P & MAUSS N (1978) Relationship of Patient-Nurse Activity to Intracranial pressure: a pilot study. *Nurs Res,* 27, 4-10.

MOHER D, COOK D, EASTWOOD S, OLKIN I, RENNIE D & STROUP D (1999) Improving the quality of reports of meta-analyses of randomised controlled trials: the QOUROM statement. *The Lancet,* 354, 1896-1900.

MOPPETT, I. (2007) Traumatic brain injury: assessment, resuscitation and early management. *Br J Anaesthesia,* 99, 18-31.

MORRIS K, FORSYTH R, PARSLOW R, TASKER R & HAWLEY C (2006) Intracranial pressure complicating severe traumatic brain injury in children: monitoring and management. *Intensive Care Med,* 32, 1606-1612.

MORROW B & ARGENT A (2008) A comprehensive review of paediatric endotracheal suctioning: effects, indications and clinical practice. *Ped Crit Care Med,* 9, 465-77.

MUENCH E, BAUHUF C, ROTH H, HORN P, PHILLIPS M, MARQUETANT N, QUINTEL M & VAJKOCZY P (2005) Effects of positive end-expiratory pressure on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation. *Crit Care Med,* 33, 2367-72.

MUIZELAAR J, MARAMOU A, WARD J, KONTOS H, CHOI S, BECKER D, GRUEMER H & YOUNG H (1991) Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg,* 75, 731-739.

MUWASWES M (1984) The effects of specific activity on intracranial pressure. *Heart & Lung,* 13, 308-309.

NADAL M, AUSINA A, SAHUQUILLO J, PEDRAZA S, GARNACHO A & GANCEDO V (1998) Effects on intracranial pressure of fentanyl in severe head injured patients. *Acta Neurochir (Wien),* Supp 71, 10-12.

NARAYAN R (2001) Hypothermia for Traumatic Brain Injury - a good idea proved ineffective. *NEJM,* 344, 602-603.

NATIONAL INSTITUTE OF CLINICAL EXCELLENCE (2007) Head Injury - Triage, assessment, investigation and early management of head injury in infants, children and adults. IN NATIONAL COLLABORATING CENTRE FOR ACUTE CARE (Ed.). London, Dept of Health.

NEEDLE S, O'RIORDAN M & SMITH P (2009) Parental anxiety and medical comprehension within 24 hours of a child's admission to the pediatric intensive care unit *Ped Crit Care Med,* 10, 1-7.

NG I, LIM J & BEE WONG H (2004) Effects of Head Posture on Cerebral Hemodynamics: Its influence on Intracranial Pressure, Cerebral Perfusion Pressure, and Cerebral Oxygenation. *Neurosurgery,* 54, 593-597.

NINAN A, O'DONNELL M, HAMILTON K, TAN L & SANKARAN K (1986) Physiologic changes induced by endotracheal instillation and suctioning in critically ill preterm infants with and without sedation. *American Journal of Perinatology,* 3, 94-97.

NORTH B & REILLY P (1986) Comparison among three methods of intracranial pressure recording. *Neurosurgery,* 18, 730-732.

OFFICE OF PUBLIC SECTOR INFORMATION (OPSI) Data Protection Act 1998 *Ch 29.* Retrieved 10.1.09 http://www.opsi.gov.uk/acts/acts1998/ukpga_19980029_en_1

OLSON D, THOYRE S, TURNER D, BENNETT S & GRAFFAGNINO C (2007) Changes in intracranial pressure associated with chest physiotherapy. *Neurocrit Care,* 6, 100-103.

PALMER, J. (2000) Management of raised intracranial pressure in children. *Intensive & Critical Care Nursing,* 16, 319-327.

PARAHOO, K. (1997) *Nursing Research: Principles, Process and Issues,* London, Macmillan.

PARATZ J & BURNS Y (1993) The effect of respiratory physiotherapy on intracranial pressure, mean arterial pressure, cerebral perfusion pressure and end tidal carbon dioxide in ventilated neurosurgical patients. *Physiotherapy Theory and Practice,* 9, 3-11.

PARSLOW R, MORRIS K, TASKER R, FORSYTH R & HAWLEY C (2005) Epidemiology of traumatic brain injury in children receiving intensive care in the UK. *Arch Dis Child,* 90, 1182-7.

PARSONS C & OUZTS SHOGAN J (1984) The effects of endotracheal tube suctioning/manual hyperinflation procedure on patients with severe closed head injuries. *Heart & Lung,* 13, 372-380.

PARSONS C & WILSON M (1984) Cerebrovascular Status of Severe Closed Head Injured Patients Following Passive Position Changes. *Nursing Research,* 33, 68-7.

PARSONS L, SMITH PEARD A & PAGE C (1985) The Effects of Hygiene Interventions on the Cerebrovascular Status of Severe Closed Head Injured Persons. *Research in Nursing and Health,* 8, 173-181.

PATEL H, MENON D, TEBBS S, HAWKER R, HUTCHINSON P & KIRKPATRICK P (2002) Specialist neurocritical care and outcome from head injury. *Intensive Care Med,* 28, 547-553.

PEARSON G, STICKLEY J & SHANN F (2001) Calibration of the paediatric index of mortality in UK paediatric intensive care units. *Arch Dis Child,* 84, 125-128.

PEAT J & BARTON B (2005) *Medical Statistics - a guide to data analysis and critical appraisal,* Oxford, Blackwell Publishing.

PETERS J, CHARLES-EDWARDS I & FRANCK L (2004) Exploring issues of parental consent to research: a case-study approach. *Br J Nursing,* 13, 740-743.

PETERS L (1992) Does routine nursing care complicates the physiologic status of the premature neonate with respiratory distress syndrome? *J Perinat Neonatal Nursing,* 6, 67-84.

PETRIE A and SABIN C (2005) *Medical Statistics at a Glance,* 2nd Edn, Blackwell Publishing, Oxford UK.

PICANET Paediatric Intensive Care Audit Network: Retrieved 10.1.09 http://www.picanet.org.uk/

PICS (2009) Paediatric Intensive Care Society (PICS).Retrieved 10.1.09 http://www.ukpics.org/

PIGULA F, WALD S, SHACKFORD S & VANE D (1993) The Effect of Hypotension and Hypoxia on Children With Severe Head Injuries. *Journal of Pediatric Surgery,* 28, 310-316.

PITTMAN T, BUCHOLZ R & WILLIAMS D (1989) Efficacy of Barbiturates in the treatment of resistant intracranial hypertension in severely head-injured children. *Pediatric Neuroscience,* 15, 13-17.

POCA M, SAHUQUILLO J, ARRIBAS M, BAGUENA M, AMOROS S & RUBIO E (2002) Fibre optic Intraparenchymal Brain Pressure Monitoring with the Camino V420 Monitor: Reflections on our experience in 163 Severely head-injured patients. *Journal of Neurotrauma,* 19, 439-448.

POCA M, SAHUQUILLO J, VILALTA A & GARNACHO A (2007) Lack of utility of arteriojugular venous difference of lactate as a reliable indicator of increased brain anaerobic metabolism in traumatic brain injury. *J Neurosurg,* 106, 530-537.

POCOCK S, CLAYTON T, ALTMAN D (2002) Survival plots of time-to-event outcomes in clinical trials: good practice and pitfalls. *The Lancet,* 359, 1686-1689.

PORTER K & ALLISON K (1999) Semi rigid cervical collar and intracranial pressure: a change in pre-hospital emphasis might be indicated. *Pre-hospital Immediate Care,* 3, 226-228.

POTTER F, TUME L, BAINES P, KERR S, PARKINS K, SARGINSON R, SELBY A, THORBURN K, BUXTON N, MALLUCCI C, MAY P & SINHA A (2006) Intensive Care Medical Guidelines for management of severe Traumatic Brain Injury. The Royal Liverpool Children's Hospital.

PRIELIPP R & COURSIN D (1995) Sedative and Neuromuscular Blocking Drug use in Critically Ill Patients with Head Injuries. *New Horizons,* 3, 456-468.

PRINS M (1989) The effect of Family visits on intracranial pressure. *Western Journal of nursing Research,* 11, 281-297.

QURESHI A, SUAREZ J, BHARDWAJ A, MIRSKI M, SCHNITZER M, HANLEY D & ULATOWSKI J (1998) Use of hypertonic (3%) saline/acetate infusion in the treatment of cerebral oedema: effect on intracranial pressure and lateral displacement of the brain. *Crit Care Med,* 26, 440-446.

REILLY P & BULLOCK R (2005) *Head Injury Pathophysiology and Management,* London, Hodder Arnold.

RISING, C. (1993) The Relationship of Selected Nursing Activities to ICP. *J Neuroscience Nurs,* 25, 302-308.

ROBINSON, A. (1997) The management of raised intracranial pressure in children. *Current Anaesthesia and Critical Care,* 8, 8-13.

ROSNER M & COLEY I (1986) Cerebral perfusion pressure, intracranial pressure, and head elevation. *J Neurosurg,* 65, 636-641.

ROWLEY G & FIELDING K (1991) Reliability and accuracy of the Glasgow coma scale with experienced and inexperienced users. *The Lancet,* 337, 535-538.

ROYAL LIVERPOOL CHILDREN'S NHS TRUST (2006) Code of Good Practice for Clinical Audit - a Guide for staff involved in clinical audit. THE ROYAL LIVERPOOL CHILDREN'S NHS TRUST (Ed.)

RUDY E, BAUN M, STONE K & TURNER B (1986) The relationship between endotracheal suctioning and changes in intracranial pressure: A review of the literature. *Heart & Lung,* 15, 488-494.

RUDY E, TURNER B, BAUN M, STONE K & BRUCIA J (1991) Endotracheal suctioning in adults with head injury. *Heart & Lung,* 20, 667-674.

RUSSO H, BRESSOLLE F & DUBOIN M (1997) Pharmacokinetics of High-dose Thiopental in Pediatric patients with Increased Intracranial Pressure. *Therapeutic Drug Monitoring,* 19, 63-70.

SABSOVICH I, REHMAN Z, YUNEN J & CORITSIDIS G (2007) Propofol Infusion Syndrome: A case of increasing morbidity with traumatic brain injury. *American Journal of Critical Care,* 16, 82-85.

SACKETT D, STRAUS S, RICHARDSON W, ROSENBERG W & HAYNES R (2000) *Evidence-Based Medicine - How to practice and teach EBM,* Edinburgh, Churchill Livingstone.

SAHUQUILLO J & ARIKAN F (2006) Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury (review). *The Cochrane Collaboration,* 1.

SALIM A, HANNON M, BROWN C, HADJIZACHARIA P, BACKHUS L, TEIXEIRA P, CHAN L & FORD H (2008) Intracranial Pressure Monitoring in Severe Isolated Pediatric Blunt Head Trauma. *The American Surgeon,* 74, 1088-1093.

SCALEA T, BOCHICCHIO G, HABASHI N, MCCUNN M, SHIH D, MCQUILLAN K & AARABI B (2007) Increased Intra-abdominal, intrathoracic, and Intracranial pressure after severe traumatic brain injury: *The journal of trauma injury, infection and critical care,* 62, 647-656.

SCHINNER K, CHISHOLM A, GRAP M, SIVA P, HALLINAN M & LAVOICE-HAWKINS N (1995) Effects of auditory stimuli on intracranial pressure and cerebral perfusion pressure in traumatic brain injury. *J Neurosci Nurs,* 27, 318-354.

SCHIRMER C, ACKIL A & MALEK A (2008) Decompressive Craniectomy. *Neurocrit Care,* 8, 456-470.

SCHULZ-STUBNER S & THIEX R (2006) Raising the head-of-bed by 30 degrees reduces ICP and improves CPP without compromising cardiac output in euvolaemic patients with traumatic brain injury and subarachnoid haemorrhage: a practice audit. *European Journal of Anaesthesiology,* 23, 173-180.

SEKULA R & WILBERGER J (2005) The Management of Traumatic Brain Injury: The Development of Guidelines and their Influence. *Clinical Neurosurgery,* 52, 306-310.

SHAFI N & MARISCALO M (2006) Considering the use of induced hypothermia in a pediatric patient with traumatic brain injury: A critical appraisal of two meta-analyses. *Ped Crit Care Med,* 7, 468-472.

SHALIT M & UMANSKY F (1977) Effect of Routine Bedside Procedures on Intracranial Pressure. *Israel Journal of Medical Sciences,* 13, 881-886.

SHAPIRO K & MARMAROU A (1982) Clinical applications of the pressure-volume index in treatment of pediatric head injuries. *J Neurosurgery,* 56, 819-825.

SIGNORINI D, SHAD A, PIPER I & STATHAM P (1998) A clinical evaluation of the Codman Microsensor for intracranial pressure monitoring. *Br J Neurosurgery,* 12, 223-227.

SIMMONS, B. (1997) Management of Intracranial Hemodynamics in the adult: A research analysis of head positioning and recommendations for clinical practice and future research. *J Neurosci Nurs,* 29, 44-49.

SINGH N, KISSOON N, FREWEN T & TIFFIN N (1991) Physiologic responses to endotracheal suctioning and oral suctioning in paediatric patients: the influence of endotracheal tube sizes and suction pressures. *Clinical Intensive Care,* 2, 345-350.

SKIPPEN P, SEEAR M, POSKITT K, KESTLE J, COCHRANE D, ANNICH G & HANDEL J (1997) Effect of hyperventilation on regional cerebral blood flow in head-injured children. *Crit Care Med,* 25, 1402-1409.

SLATER A, SHANN F, PEARSON G & PAEDIATRIC INDEX OF MORTALITY (PIM) STUDY GROUP (2003) PIM2: A revised version of the Paediatric Index of Mortality. *Intensive Care Med,* 29, 278-85.

SMITH , M. (2008) Monitoring Intracranial Pressure in Traumatic brain Injury. *Anesth Analg,* 106, 240-248.

SNYDER, M. (1983) Relation of nursing activities to increases in intracranial pressure. *J Advanced Nursing,* 8, 273-279.

SORANI M & MANLEY G (2008) Dose-response relationship of mannitol and intracranial pressure: a meta-analysis. *J Neurosurg,* 108, 80-87.

SOUSTIEL J, MAHAMID E, CHRISTYAKOV A, SHIK V, BENENSON R & ZAAROOR M (2006) Comparison of moderate hyperventilation and mannitol for control of intracranial pressure control in patients with severe traumatic brain injury - a study of cerebral blood flow and metabolism. *Acta Neurochir (Wein),* 148, 845-851.

SPERRY R, BAILEY P, REICHMAN M, PETERSON J, PETERSEN P & PACE N (1992) Fentanyl and Sufentanil increase intracranial pressure in head trauma patients *Anaesthesiology,* 77, 416-420.

STAFFA G, NATALONI A, COMPAGNONE C & SERVADEI F (2007) Custom made cranioplasty prostheses in porous hydroxy-apatite using 3D design techniques: 7 years experience in 25 patients. *Acta Neurochir (Wein),* 149, 161-170.

STOCCHETTI N, COLOMBO A, ORTOLANO F, VIDETTA W, MARCHESI R, LONGHI L & ZANIER E (2007) Time course of Intracranial Hypertension after traumatic Brain Injury. *Journal of Neurotrauma,* 24, 1339-1346.

SUAREZ, J. (2004) Hypertonic saline for cerebral edema and elevated intracranial pressure. *Cleveland Clinic Journal of Med,* 71, S9-S13.

TAYLOR A, BUTT W, OSENFLED J, SHANN F, DITCHFILED M, LEWIS E, KLUG G, WALLACE D, HENNING R & TIBBALLS J (2001) A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Child's Nervous System,* 17, 154-162.

THE RESCUEICP STUDY - INTERNATIONAL MUTLICENTRE RANDOMISED CONTROLLED TRIAL (2003) Cambridge, UK. Retrieved 8.2.08 http://www.rescueicp.com/frameset4.html

TIBBY S & MURDOCH I (2002) Calibration of the paediatric index of mortality score for UK paediatric intensive care. *Arch Dis Child,* 86, 65.

TIMOFEEV I, CZOSNYKA M, NORTJE J, SMIELEWSKI P, KIRKPATRICK P, GUPTA A & HUTCHINSON P (2008) Effect of decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury. *J Neurosurg,* 108, 66-78.

TOKUTOMI T, MORIMOTO, K., MIYAGI T, YAMAGUCHI S, ISHIKAWA K & SHIEGEMORI M (2003) Optimal Temperature for the Management of Severe Traumatic Brain Injury: effect of hypothermia on intracranial pressure, systemic and intracranial hemodynamics, and metabolism. *Neurosurgery,* 52, 102- 112.

TREGGIARI M, SCHUTZ N, YANEZ N & ROMAND J (2007) Role of intracranial pressure values and patterns in predicting outcome in traumatic brain injury: a systematic review. *Neurocrit Care,* 6, 104-112.

TREOLAR D, JERMIER NALLI B, GUIN P & GARY R (1991) The effects of familiar and unfamiliar voice treatments on intracranial pressure in head-injured patients. *J Neurosci Nurs,* 23, 295-299.

TROPE R, SILVER P, SAGY M (2005) Concomitant assessment of depth of sedation by changes in Bispectral index and changes in autonomic variables (hear rate and/or BP) in pediatric critically ill patients receiving neuromuscular blockade. *Chest* ,128, 303-307.

TSEMENTZIS S, HARRIS P & LOIZOU L (1982) The Effect of Routine Nursing Care Procedures on the ICP in severe Head Injuries. *Acta Neurochir (Wien),* 65, 153-166.

TUME L & BAINES P (2008) UK PICU practices in Traumatic Brain Injury: an audit of practice. *British Journal of Intensive Care,* Autumn, 84-89.

VERHAEGHE S, DEFLOOR T & GRYPDNOCK (2005) Stress and coping among families of patients with traumatic brain injury: a review of the literature. *J Clinical Nursing,* 14, 1004-1012.

VICKERS, A. (2001) The use of percentage change from baseline as an outcome in a controlled trial is statistically inefficient: a simulation study. *BMC Medical Research Methodology,* 1.

VIK A, NAG T, FREDRIKSLI O, SKANDSEN T, MOEN K, SCHIRMER-MIKALSEN K & MANLEY G (2008) Relationship of "dose" of intracranial hypertension to outcome in severe traumatic brain injury. *J Neurosurg,* 109, 678-684.

VINCENT J & BERRE J (2005) Primer on medical management of severe brain injury. *Critical Care Medicine,* 33, 1392-1399.

VON ELM E, ALTMAN D, POCOCK S, GOTZSCHE P & VANDENBROUCKE J (2007) Strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *BMJ,* 335, 806-808.

WAINWRIGHT S & GOULD D (1996) Endotracheal suctioning in adults with severe head injury: literature review. *Intensive & Critical Care Nursing,* 12, 303-308.

WAKAI A, ROBERTS I & SCHIERHOUT G (2007) Mannitol for acute traumatic brain injury (review). *The Cochrane Collaboration,* 3.

WEANT K & COOK A (2008) Pharmacologic strategies for the treatment of elevated intracranial pressure - focus on Osmotherapy. *Advanced Emergency Nursing Journal,* 30, 17-26.

WEINSTEIN, S. (2006) Controversies in the care of children with acute brain injury. *Current Neurology and Neuroscience Reports,* 6, 127-135.

WERBA A, KLEZL M, SCHRAMM W, LANGENECKER S, MULLER C, GOSCH M & SPISS C (1993) The level of neuromuscular block needed to suppress diaphragmatic movement during tracheal suction in patients with raised intracranial pressure: study with vecuronium and atracurium. *Anaesthesia,* 48, 301-303.

WHITE P, SCHLOBOHM R, PITTS L & LINDAUER J (1982) A Randomized Study of Drugs for Preventing Increases in Intracranial Pressure during Endotracheal Suctioning. *Anaesthesiology,* 57, 242-244.

WILBERGER J & CANTELLLA D (1995) High-Dose Barbiturates for Intracranial Pressure Control. *New Horizons,* 3, 469-473.

WINDERS DAVIS D (2004) Reliability and Validity: Part II. *Neonatal Network,* 23, 74-76.

WINDERS DAVIS D (2004) Validity and Reliability Part I. *Neonatal Network,* 23, 54-56.

WORLD FEDERATION OF SOCIETIES OF ANAESTHESIOLOGISTS (1998) World Anaesthetics Online. IN TEAM (Ed.) Issue 1 ed. Oxford. Intracranial Pressure and Cerebral Blood Flow retrieved 8.208 **www.nda.ox.ac.uk/wfsa/html/u08/u08_014.htm**

WUNSCH H, LINDE-ZWIRBLE W & ANGUS D (2006) Methods to adjust for bias and cofounding in critical care health service research involving observational data. *Journal of Critical Care,* 21, 1-7.

YANO M, NISHIYAMA H, YOKOTA H, KATO K, YAMAMOTO Y & OTSUKA T (1986) Effect of Lidocaine on ICP response to Endotracheal Suctioning. *Anaesthesiology,* 64, 651-653.

APPENDICES

APPENDIX ONE

Appendix One: Summary of papers included in the literature review

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Bilotta et al 2008 (Italy)	The effect of ET Lidocaine in preventing ICP rise during routine ETS and the dose effect of Lidocaine	Quasi-exp design	41 mechanically ventilated stable adults (>18yrs) with severe head trauma (GCS <8) who had an ICP rise >20 with ETS	Parenchymal catheter	ICP CPP HR Spo2 EtCo2 vMCA	In all patients pre Lidocaine ETS led to a sign rise in ICP >20mmHg ($p<0.05$) In 29% patients instilled Lidocaine produced a cough or movement but not assoc with ICP >20mmHg. In 51% patients IT Lidocaine prevented an ICP rise with ETS (>20mmHg) An ED_{50} of 1.7 +/- 0.3mg/kg was determined as the dose of Lidocaine required to prevent an ICP rise.	Most but not all known confounders accounted for (presence of rigid collar, level of sedation, core temp, PaCo2). Poor graphical presentation of results with p values
Boortz-Marx 1985 (US)	- Patient care activities - Health care activities - Environmental stimuli (eg conversations over the patient)	Descriptive observational design	4 adults with Severe HI or SAH GCS < 5 and 1 child (8yrs)	SA Bolt or Vent Cath	ICP	↑ICP caused by suction, turning, head flexion ↓ICP caused by HOB elevation & reposition Pt related activities ↑ICP were flexion/extension, rotation of neck & coughing & bathing Env stimuli had no affect on ICP	Very small sample size, with mixed pathology and diff ICP devices, (says pilot study done on 1 child!), no baseline ICPs presented, no real results (descriptive even presented)

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Brown et al 1996	The effect of suxamethonium vs placebo before planned physiotherapy	Double blind randomised crossover trial	11 adults (17-70yrs) in intensive care within 48 ours of their severe TBI	Subdural catheter	ICP CPP MAP	No significant changes in ICP following the administration of suxamethonium	It is a blinded trial yet they observed for fasciculation (and this could only happened with sux) so they would be aware of the drug they gave.
Brucia and Rudy 1996 (US)	What was the effect of suction catheter insertion and tracheal stimulation on CBV parameters?	Quasi-exp crossover design	30 mechanically ventilated adults with severe TBI (GCS <8)	4 types of device: Ventricular catheter, fibre optic, SA catheter, Subarachnoid (SA) bolt	ICP CPP MAP HR	Suction catheter insertion and tracheal stimulation isolated from other parts of the suction procedure sig increased ICP, MAP and CPP. HR not sig increased. During the application of negative pressure for suction ICP and MAP increased further, whereas MAP and CPP did not change	Diff ICP devices used in the sample, open and closed HI in the same sample, tried to account for some confounders eg paralysis, sedation but many other not reported. Moved supine/head up only 15 mins before the procedure little time after this position change. No mention of whether the patient's coughed during suction

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Bruya 1981 (US)	Planned rest periods between cares (10 mins) one was ETS	RCT	20 adults (18-81 yrs) with intracranial pathology with raised ICP	Richmond Screw	ICP	No difference found between groups – suggest 10 minutes rest too little time	Small sample, no power calculation mentioned. Randomisation done on alternate basis, variety of IC pathology. No inferential data analysis presented.
Campbell 1991 (US)	Controlled hyperoxygenation & hyperventilation & ETS	Quasi-experimental study	9 Adults (20-51yrs) with severe closed HI with ICP monitor in situ > 4 hours)	Not stated	ICP	No significant changes among 3 hyperinflation volumes – hyperventilation did not prevent the adverse events of. Stair step pattern of ICP rises noted	ICP device not stated Baseline ICP 4.8mmHg mean (not true reflection of severe HI patients) Many confounding variables not discussed (eg drugs)

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Crosby & Parsons 1992 (US)	Effect of a ETS & Manual hyperinflation procedure on CBV parameters	Quasi-experimental repeated measures	49 adults (17-75yrs) with various intracranial pathology requiring ICP monitoring	SA Bolt	ICP CPP MAP HR	145 ETS procedures analysed on 49 patients, stratified into lower resting ICP & higher resting ICP. They found stat sig ($p<0.001$) differences during the ETS and 5 minutes post ETS protocol for all variables in both groups. MICP gradually increased in a step wise pattern from baseline with every suction pass. The MAP increased sig ($p<0.0025$) in both groups with ETS. The CPP increased sig ($p<0.0025$) in both groups with ETS. The HR increased sig ($p<0.0025$) throughout ETS. With regard to ICP rise no sig diff between 2 groups despite sig diff baseline ICP values.	Various pathology of the sample group.
Donegan and Bedford 1980 (US)	Whether IV Lignocaine compared to placebo reduced ICP associated with ETS	Crossover experimental design of IV Lignocaine vs placebo (0.9% saline)	10 Comatose patients with head injury who had previously had Intracranial hypertension following ETS	SA Screw	ICP MAP	IV Lidocaine (coupled with MH) sign reduced the ICP prior ETS, but that after ETS the ICP rise was similar to the control group.	? age of patients very short washout time between drugs 50% of patients had been receiving Thiopentone hourly (long t1/2 still present in 50%)

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Ersson et al 1990 (Sweden)	The effect of respiratory physiotherapy on intracranial dynamics	Descriptive observational design	12 mechanically ventilated adults and children (5-67years) with raised ICP	Ventricular Catheter, Extradural catheter and a further Lundberg method	ICP MAP AWP	The ICP could reach peaks of 70mmHg for short periods during Rx. Mean increase in peak ICP was 34 and 31mmHg - No inferential data analysis presented. After Rx terminated ICP reduced rapidly for 30sec and returned to pre-Rx level or slightly elevated after 1 minute. No drop in CPPP below 50 except in one patient who had sedation bolus to control ICP rise	Mixed IC pathology, studied from1-11days after HI – wide range, mixed adult and paed sample, 3 diff ICP devices in sample, baseline ICP ranged between 1-30mmHg)huge range), many confounders not accounted for. Ethical implications of starting Physio Rx for study when ICP 30mmHg. With the broad range in baseline ICP patients should have been stratified and compared separately
Fisher et al 1982 (US)	Mechanism of ICP rise with ETS	Crossover design	9 children (9mths – 12 years) with raised ICP	Subarachnoid Screw	ICP CPP EtCo2	5mmHg mean rise in ICP during ETS 10mmHg MAP rise during ETS EtCo2 rose in apnoea periods for both groups 2/3 patients coughed during ETS (non NMB) & ICP changes in these 2 patients were similar during ETS vs non ETS trial ICP rise during ETS related to direct tracheal stimulation	Children < 18 months had fontanelle open (open head) and mixed pathology of sample Some confounding variables noted but not controlled for in analysis

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Garradd & Bullock 1986 (Australia)	Effect of RP on ICP	Quasi-experimental: RP performed during NMB and without NMB	20 Ventilated neurosurgical patients (12-60yrs) with trauma or bleeds that were ICP monitored	Richmond Screw	ICP	In non NMB patients a full RPT Rx (lasting 17 mins) resulted in a sig (p<.01) increase in ICP. In NMB patients the ICP did not rise by stat sig amount, however time of treatment was an important factor and by the end of a full RX the ICP of the NMB patients had reached the levels of non NMB patients and this was more pronounced in patients with resting ICP >15mmHg. Pts with lower resting ICPs the effect of Rx was similar to that of the control period until the 14th minute	2 days between comparison Rx but NMB was in the first 3 days of injury (and hence likely to be more IC HTN) Children and adults combined in the analysis.
Gemma et al 2002 (Italy)	Effects of ETS in Acute phase of Head Injury	Quasi-experimental design	17 Adults (17-77yrs) with severe HI GCS <8 sedated & ventilated	Ventricular or subdural catheter	ICP SjO2 MAP CPP	Total of 113 ETS episodes for analysis 6 Pts in 20 cases coughed/moved due to inadequate sedation. ICP mean ↑ by 20 to 22 in well sedated pts ICP mean ↑15 to 28 in pts who coughed/moved P < 0.0001 CPP & SjO2 ↑ in well sedated pts & ↓ in pts who reacted to ETS p 0.0001 ETS sig increases ICP (p<0.0001) and this increase was more pronounced when sedation was inadequate	Most confounding variables noted and controlled for in procedure 2 different ICP devices

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Hanowell et al 1993 (US)	Effect of Alfentanil on CPP in ETS	Quasi-experimental crossover design: Placebo (0.9% saline) vs 2 different does of Alfentanil prior to ETS	6 Adults (22-34yrs) with Closed HI with GCS < 8	Fibre optic Catheter	ICP CPP MAP HR Tidal volumes, PIP & plateau pressures	No differences between the 2-alfentanil doses so analysed together. The CPP was reduced ($p<0.05$) in the drug groups vs placebo. No differences or changes in ventilatory parameters and no chest wall rigidity noted.	30 minutes spaces may had had some carry over effect from previous Rx Small sample size
Hobdell et al 1989 (US)	The effect of nursing activities (one was ETS) on the ICP in Children	Observational study	13 Children (1.5 – 11yrs) with variety of pathology causing raised ICP	Ventricular catheter 4, SA Bolt 9	ICP (mean in 12 and systolic in 1 patient)	Only hyperinflation and ETS demonstrated sig differences in the ICP. The mean ICP was significant higher ($p <0.05$) at the time of hyperventilation than 10 mins after. Higher mean ICP in kids receiving pentobarbital over time in procedures of hyperventilation & suction compared to those not on it($p<0.01$)	Huge variety of pathology eg DKA Different ICP devices Some confounding variables noted but not controlled for or accounted for in analysis Different ICP variables No discussion of how long between care interventions, may have had some carry over effects from previous care. Not enough detail presented of procedure.

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Hugo 1987 (South Africa)	Manipulation of nursing care activities (one of which was ETS)	Descriptive observational study in 2 groups	23 adults & children (5-60 yrs) with severe head injuries	Epidural catheter	ICP MAP	No statistically significance difference found between the groups with regard to rest periods. The smallest pressure increases noted in patients receiving barbiturates, followed by those receiving narcotics + NMB	Adults & children analysed together. Small sample size (power calculation predicted 50) Some confounding variables noted but not controlled for Mixed pathology No inferential data
Jones 1995 (Australia)	Patient repositioning – but head elevation 15-30 degrees always maintained. Turned supine, left & right lateral	Observational descriptive study, crossover	30 adults > 1 (74 positionings) with raised ICP with bolt in	SA Bolt	ICP	Each of the 6 position changes caused an ↑ in group mean ICP during repositioning and remained elevated 5 mins after 4 position changes.	No discussion of any drugs the patients on (sedation, Thiopentone or inotropes) and no baseline ICP presented for the group. No washout time stated between interventions

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Kerr et al 1997	The effect of short-duration (1 min) hyperventilation 4 breath vs 8 breath vs 30 breaths per min during ETS Hyperventilation = 135% tidal volume	Quasi-experimental repeated measures design	66 Adults (16 - 37 yrs) with severe open or closed head injury in ICU ventilated with ICP monitoring	Not stated	ICP CPP MAP HR SaO2	No difference in values between the 4 and 8 breath groups, but sig differences in all phases of suctioning between the 4 and 30 breath groups In ICP, CPP, AP and PeTco2 with lower ICP response to ETS in the 30 breath group	Not stated exactly the PaCo2 level HV to Sample ↓ 33 for 4 vs 30 breath comparison as RNs intervened to reduce ICP in some patients (then they were excluded) No baseline ICP of the samples presented and only some confounders are discussed The effect of this extreme HV on CBF is unknown as only CPP was measured
Kerr et al 1998 (US)	Effect of NMB and opiates on the CBV response to ETS: 3 groups:- no drugs, opiates only or opiates + NMB	Quasi-experimental repeated measures within subject, between group design	71 Adults (16-73yrs) with severe HI GCS <8 who were ventilated with ICP monitoring	SA Bolt 17, Fibre optic catheter 12 or Ventricular catheter 42	ICP CPP MAP	ICP changed significantly ($p<0.001$) with ETS and increased with each catheter pass. Pts having no drugs displayed the classic 'stair step' pattern of ICP rises, but this response was NOT seen in pts having opiates + NMB. These patients displayed a flat response during ETS, with little change in ICP. Sig changes in MAP and CPP with ETS were noted among 3 groups. MAP increased with each suction pass.80% of patients ICP returned to baseline by 15 minutes.	Non random allocation into Rx groups and so differences in baseline ICP (but controlled for in analysis) Diff ICP devices

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Kerr et al 1999	The effect of ETS and hyperventilation (135% vT) prior to and after on cerebral oxygenation	Prospective descriptive study with repeated measures	19 Adults with severe traumatic brain injury, ventilated in ICU	Ventricular Catheter	ICP CPP Vmca Pjo2 (n=11) AV O2 difference	Both ICP and CPP are increased during ETS, the increase in SjO2 suggests that cerebral oxygenation is maintained during ETS	Used patients in varying phases of head injury from 1 – 6 days after injury. Only some confounders discussed. Only 11 patients had jugular catheters in.
Lee 1989 (China)	Positioning – head down, ¾ supine, ¾ prone (head not turned)	Quasi-experimental design	30 adults Severe HI (GCS < 8 for 24 hours) – trauma + non-trauma	SA Screw	ICP	In group as a whole all positions ICP ↑ significantly (p <0.01) – some aberrations in some patients.	Broad pathology of sample, some bleeds, some trauma. No discussion of washout period between 1 position to the next
Leone et al 2004 (France)	The effect of Remifentanil on ETS induced rises in ICP in head injured patients	Quasi-experimental Crossover design	20 Severe head Injured patients (18-50yrs) (GCS <8) whose were haemodynamically stable for 24h or more without any sedation	Fibre optic catheter	ICP MAP CPP HR Vmca BIS	Sig Haemodynamic effects (reduction in MAP and CPP) were noted in the 1st 2 Remifentanil doses (p<0.001) who required inotropic support. The high dose Remifentanil suppressed the cough 75% of patients. ICP increased sig from baseline in all 3 groups	60 minute washout period between treatments

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Muwaswes 1984 (US)	What are the effects of passive ROM ex and with turning to 4 positions on ICP, and recovery time and to see if time periods between interventions (<15 mins) made this worse	Quasi-exp design	12 adults with brain injury (6 traumatic, 6 non-traumatic)	Ventricular and Subdural catheters	ICP rise, MAP	Each activity elevated ICP (no affect on MAP), the turns more dramatically. The recovery of ICP varied but was associated with the degree of initial ICP rise	No demographic or confounding data presented. No baseline ICP data presented and no p values presented.
Paratz & Burns 1993 (Australia)	Effects of Respiratory physiotherapy – detailed (percussion, manual hyperinflation + ETS) on CBV parameters	Experimental cross over design with random Rx assignment order	20 Adults (17-31yrs) ventilated neurosurgical patients having ICP monitoring	Epidural ICP catheter	ICP MAP CPP Etco2	A full Rx prod a significant ↑ all variables (except CPP) p <0.001 Percussion had no effect on the variables except ICP which ↓p<0.001. Manual hyperinflation ↑ all parameters except CPP p<0.01.	Variety of IC pathology 30 minutes between Rx may have had a carry over effect from previous Rx No invasive IAL so manual BPs done
Parsons et al 1985 (US)	Hygiene interventions – oral hygiene, body hygiene and indwelling catheter care	Quasi-exp study, crossover study.	19 Adults & kids (5 – 67 years) with Severe closed HI (GCS 3-10)	SA Bolt	HR, MAP, ICP & CPP at baseline, peak & 1 min recovery	All 3 procedures produced similar ↑ in all parameters. Oral & body hygiene were > than those with catheter care. These rises all ↓ to baseline levels within 1 min following intervention – hence safe	Mixed adult/paed sample, some confounders mentioned (temp, ventilation) only, time between interventions (washout) not stated, Some patients on barbiturates but these not a/c for in analysis, Baseline ICPs very low 5-7mmHg

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Parsons and Ouzts Shogan 1984 (US)	What was the effect of ETS/MH procedure on CBV status of severe closed HI patients?	Experimental study of specific ETS procedure	20 patients (5-67yrs) with severe closed head injury (GCS 3-10) who were haemodynamically stable and no severe pulmonary injuries	SA Bolt	ICP MAP HR CPP	All variables showed recovery of baseline values at 1 minute post ETS Both MAP and ICP values were both elevated compared to baseline during procedure	No account of confounding variables Children and adults in sample Baseline Mean ICP 5mmHg and never exceeded 20mmHg during ETS indicating non severe illness
Parsons and Wilson 1984 (US)	The effect of 6 Passive position changes in severe HI adults	Quasi-exp, crossover study	18 severe ventilated head injured patients 5-67 years (GCS 3-10) with stable ICP <15 pre-intervention	SA Bolt	ICP rise Recovery time MAP, HR, CPP	With the exception of raising the HOB all other interventions produced increases in ICP, MAP, HR and CPP, but these increases were transient and showed recovery towards baseline at 1 minute post intervention	Adults & kids mixed in sample, Some confounder stated (temp, PEEP but not sedative use, paralysis, barbiturates), no consistent washout time between position changes, no random sequence of position changes mentioned

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Rising 1993 (US)	- Turning, suctioning, Bathing	Observational, Case study design	5 adults > 18 years Closed HI but GCS range 5-13 2 not intubated	Fibre optic catheter in Ventricular or SA space	ICP changes during and following activity	No generalisations made – presented individually V weak design	Mixed adult/paed sample, diff device, ICP only recorded for 2 mins after intervention, no descriptive or inferential analysis done only presented as individual cases so no conclusions could be drawn
Rudy et al 1991 (US)	Which ETS method resulted in least adverse effects: 100% vs 135% tidal volume and 2 ETS passes vs 3 ETS passes	Randomised 2 Group 2 protocol experimental design	30 patients (15-45yrs) with severe head open and closed injuries	SA Bolt 25, Epidural catheter 2, Ventricular catheter 1 and Rickman reservoir 1	MICP MAP CPP HR Sao2	Patients on barbiturates had lower ICPs during ETS. In 76% ICP had not returned to baseline in 1 minute, 42% had not returned to baseline in 5 mins & 25% had not returned to baseline in 9 mins. The number of patients with increased MAP increased through the ETS procedure. CPP improved throughout ETS as a result of increased MAP, but in 100% of the patients the CPP had not returned to baseline by 5 minutes NO significant differences found between 100 vs 135% VT methods for any variable. For both 2 and 3 ETS pass groups there was sig ($p < 0.001$) change from baseline for all variables	Different ICP monitoring devices Use of open & closed head injured patients Small sample size, no mention of power calculation Some Confounding variables noted but not accounted for in the analysis. The 3ETS group baseline morphine requirements were sig greater ($p<0.01$) which may affect the results in that group

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
Shalit & Umansky 1977 (Israel)	Positioning, suction & head rotation	Observational study	21 adults Comatose with brain oedema	Ventricular Catheter & subdural catheter	ICP	General trends only described per procedure	Mixed devices, diff pathology, no descriptive or inferential data analysis done
Tsementzis et al 1982 (UK)	'Routine nursing procedures' including ETS +/- RP, pupillary light reflex, striking the metal bed frame with metal objects, taking manual BP, taking rectal temp, inserting NG tube, IM injections, and performing oral or skin hygiene	Observational study of cares done hourly	39 head injured adults and children 7 – 68yrs that developed decerebrate rigidity (multiple injuries excluded), ventilated and paralysed and sedated	Ventricular catheter	ICP rise, BP and ICP peak increase. Divided into 2 groups depending on ICP rise (1 little or no rise or easily controlled with relaxants, 2 a rise in ICP uncontrolled by relaxants	In group 2 all procedures (except temp taking and metal clanking) produced sig elevation in ICP, suctioning produced the worst results (ICP rise)	Some of these 'routine cares' are not routine, mixed adult and kids, all done together, baseline ICPs in gp 1 varied from 6 – 38mmHg and gp 11-52mmHg
Werba et al 1993 (Austria)	The level of NMB needed to suppress diaphragmatic movement during ETS in patients with increased ICP	RCT comparing Vecuronium vs Atracurium	18 patients (no age stated) with Severe HI or Subarachnoid haemorrhage GCS <7	Epidural Catheter	ICP CPP Diaphragmatic movement	Coughing occurred in 100% patients suctioned without NMB and ICP increased & CPP decreased sig in all these patients. Both NMB Rx resulted in minimal diaphragmatic movements and very little ICP rise.	Small sample size Could be more detail regarding general patient management

Authors/year	Research Question	Research Design	Sample type & no.	ICP measurement device	Outcome measures	Results	Study Limitations
White et al 1982 (US)	Study of drugs for preventing ICP rises during ETS	Randomised crossover study involving: IV Thiopental, IV Fentanyl IV Lidocaine IV Suxamethonium or ET Lidocaine versus Placebo (0.9% saline)	15 comatose patients (no ages stated) with diffuse brain injuries who developed IC hypertension during or after ETS	Ventricular or Subdural catheter	ICP MAP + Cough score	Patients stratified into 2 groups normal & elevated baseline ICP. Fentanyl did not blunt the ICP response to ETS. Thiopental & IV Lignocaine produced an initial 4-6mmHg decrease in ICP but neither affected the ICP rise during ETS. IT Lignocaine was more effective in suppressing the cough reflex and in attenuating the ICP rise with ETS, however it provoked a cough when instilled which increased ICP. Sux was highly effective in abolishing the increase in ICP due to ETS	Not enough detail provided of the study, the general management or the sample. Two diff ICP devices used 1 hour in between Rx should have minimised risk of carry over effect
Yano et al 1986 (Japan)	Effect of Lignocaine IV vs ET on ICP response to ETS	Quasi-experimental cross over design	9 Adults (16-71yrs) with Severe Head injury (GCS <8) ventilated with ICP catheter, who had mild intracranial hypertension	Subarachnoid Catheter	ICP	No Haemodynamic changes or differences between the two groups during or after Lignocaine admin. Both drugs suppressed the ICP response to ETS, but the peak ICP after IT Lignocaine was sig lower ($p<0.01$)	6 hour gap between two treatments Small sample size Not enough detail presented with regard to general management. Not all confounding variables accounted or controlled for

APPENDIX THREE

Appendix Three: Audit Questionnaire

Unit name and No. of beds

Person completing audit

Do you have nursing guidelines/protocols for the management of children with severe TBI in your PICU?

If yes could this be attached and sent to above address.

Regarding Endotracheal Suctioning

Do you have specific guidelines on endotracheal suction (ETS)/respiratory physiotherapy in severe TBI children?

If so how often are these children suctioned?

If not do all the nurses in your unit routinely auscultate the chest in ventilated children?

Do you have guidelines for sedation boluses or muscle relaxants prior to ETS/respiratory physiotherapy?

If so what are these? And what drugs are generally used?

Do you routinely use Lignocaine (either intra-tracheal or IV) before suctioning?

If so, how much of what strength solution?

Are there any specific guidelines for the use of Lignocaine in this context in your unit?

Management of raised ICP post ETS

How are raised ICP episodes (post ETS) generally managed in your unit? Please tick what is generally done:

__ Sedation Bolus
__CSF Drainage
__Thiopentone Bolus
__Hyperventilation'

__Increase head up position
__Loosen cervical collar
__Other
If other please describe

Nursing Cares

Are there any guidelines in your unit for performing nursing cares on these children eg the timing of cares or whether they are clustered or spaced out?

How often do you Log-roll these children for washing/sheet change?

Do you always Log-roll these children or are the straight lifted occasionally?

General Medical management of severe TBI children on your unit

Do you use external ventricular drains (EVDs) to control ICP in severe TBI?

Do you use much Thiopentone on your unit in these children?

Is active hypothermia (eg down to 34-35°) used very often in these children?

Are these children muscle relaxed (paralysed) very often in your unit?

What do you do with the cervical collar if the child is muscle relaxed in a child whose C spine cannot be clinically cleared? Eg is it loosened, always done up or just removed and sandbags/tape put in place

Do you use TED stockings (anti-thrombotic) in children who are immobile for some time or any other DVT prophylaxis?

APPENDIX FOUR

Royal Liverpool Children's NHS
NHS Trust

17 November 2005

Direct Line:
Email:

Dear Lyvonne,

Audit reference number: **619**

Audit title: **THE EFFECT OF ROUTINE NURSING INTERVENTION ON THE ICP AND CPP OF SERVICE CLOSE HEAD INJURED CHILDREN**

Thank you for registering the audit named above with the Clinical Audit Department. This audit has been entered onto our database, and has been assigned a unique reference number **(619)**.

I would appreciate it if you could please keep a note of this number, as in order to deal with queries and requests for support efficiently and effectively, you will be asked to provide this number during any future contact with the Clinical Audit Department.

You may also find that this number is required by the Medical Records Department and/or the Information Department in order to access data for your audit.

The Clinical Audit Department is required to report regularly to the Trust and PCTs on the progress of all audits. I would therefore be grateful if, on completion of your audit, you could provide me with a copy of your audit report, complete with conclusions and recommendations. Wherever possible, recommendations should be time scaled and a 'person responsible' identified for taking forward each recommendation. Similarly, if this audit does not proceed for any reason, please let us know. If we do not hear from you, a member of the Clinical Audit team will contact you for this information.

If you require advice, support or training at any stage of your audit, please do not hesitate to contact myself on ext 2189 or Janet/Steve on ext 2636. We will try our best to help you.

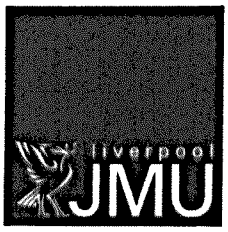
Further information about the Clinical Audit Department can be found on our intranet page under 'Clinical Audit'.

Yours sincerely

S. Williams

Sarah Williams
Clinical Audit Manager

APPENDIX FIVE



Faculty of Health and Applied Social Sciences

Parent Information Sheet

Project title: The effect of routine intensive care nursing interventions in severe head injured children

You are being invited to allow your child to take part in a research study. Before you decide it is important that you understand why the study is being done and what it will involve. You can take some time to decide whether you would allow your child to take part, and just let myself or your child's nurse know in the next few hours.

What is the purpose of the study?

The purpose of this study is to observe the effects that routine (part of normal care) nursing procedures/interventions (eg suctioning down the breathing tube, washing your child or cleaning their eyes) have on your child's physiological parameters including the pressure inside the brain (Intracranial pressure or ICP).

Why has my child been chosen?

Your child has been chosen because they have come to the intensive care unit with a severe head injury that requires that the pressure inside their brain needs measuring.

Does my child have to take part?

It is up to you whether you allow your child to take part in this study. If you decide you would like them to take part, after reading this form and discussing the study with a nurse, then you will be asked to sign a consent form. You are free to withdraw your child at any time from the study or not take part in the study without giving a reason and this will not affect the care that your child receives in any way.

What will happen to my child if I decide they can take part?

Nothing extra will be done to your child (only normal nursing interventions) it is just that what is being done will be recorded in a lot more detail, in terms of the numbers and your child's response. The study will only continue while the pressure inside their head is measured.

What are the benefits of taking part?

There are no benefits to your child of being in this study but it is hoped that the information gained will help us to improve the care we can give to children like yours in the future.

Will my child's taking part in this study be confidential?
Yes all information collected by this study will be kept strictly confidential and only the researcher involved in the study will have access to your child's data. In the results of the study your child will not be identifiable in any way.

What if something goes wrong?
This study does not involve doing anything extra to your child, beyond that of normal routine care. Regardless of this if you wish to complain about any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanism will be available to you.

Contacts for further information

APPENDIX SIX

Faculty of Health and Applied Social Sciences

Project title: **The effect of routine intensive care nursing interventions in severe head injured children**

Name of researcher: Ms L Tume

Please cross out whichever does not apply*

Have you read the parent information sheet? Yes/No *

Have you had the opportunity to ask questions and discuss this study? Yes/No *

Have you received satisfactory answers to all your questions? Yes/No *

Have you received enough information about the study? Yes/No *

I agree for my child to be allowed to take part in this study Yes/No*

Do you understand that you are free to withdraw tour child from the study:

- At any time Yes/No *
- Without having to give a reason Yes/No*
- Without affecting the care your child receives Yes/No*

_____
Name of patient (child taking part)

Name of parent/guardian consenting	Date	Signature
Name of person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

APPENDIX SEVEN

Suctioning procedure for severe Traumatic brain injured children in PICU

1. Plan suctioning carefully (with physiotherapists if at all possible) but certainly ensure it is done well before log-rolling (as this procedure will stimulate a cough/move secretions). The need for suctioning is determined by auscultation (listening) to the chest for secretions – waiting for the oxygen saturations to drop is too late. So at present our guidelines state that patency of the ETT must be assessed every 6 hours (by very quickly going down the tube).
2. If you need to use Lignocaine (remember it is only prescribed as required – you only need to use it if you have problems with raised ICP with suctioning or if the child is NOT paralysed) then instil this down it (1% solution maximum 2 mls) ETT 2 mins prior to suctioning, you need to wait to ensure it has time to take effect.
3. Pre-oxygenate (on 100%) for at least one full minute prior to suctioning to minimise hypoxia (this is not optional).
4. Consider using in-line (closed circuit suctioning) in these children, especially if PEEP >5cm.
5. It is generally a good idea to always have a 2nd person bag for you during suctioning of these children.
6. Always pre-sedate these children with either Morphine and/or Midazolam (or both if there are real ICP problems) ***Remember an IV morphine bolus takes 20 minutes to have a peak effect so give it well in advance!***
7. Don't suction too deep – you should be suctioning to the exact length of the ETT + 1cm only, no further.
8. Limit the suctioning episode (unless there is a problem eg big plugs/tube blockage) to < 5 minutes (preferably less) especially in the first 3 days after injury when cerebral oedema is at its worst.
9. No nasal suctioning in these children (in case of basilar skull fracture)
10. These children may be paralysed to prevent coughing on suction (as well as to prevent shivering) – if not paralysed ensure that endotracheal suctioning is shallow to avoid stimulating a cough or get Lignocaine prescribed (2mls of 1% solution) to instil down the ETT at least 2 minutes before suctioning.
11. Always bag with the EtCo2 in situ and ***do not be overly aggressive with bagging*** – it will drop the ICP (and make the numbers look good – but it reduces cerebral blood flow and can produce severe ischaemia and worsen the child's outcome later on) so avoid hyperventilation.
12. Record any ICP spikes with suctioning on the observation chart and leave the child well alone to recover from the procedure.
13. If the raised ICP (>20mmHg) persists for more than 5 minutes after suctioning then you need to start the Control of ICP algorithm to get this ICP under control rapidly (<20mmHg) and notify the medical team urgently.

APPENDIX EIGHT

Data Collection Form for effect of nursing interventions on ICP & CPP in Head injured children

Date: _____ Shift: Day or Night

Patient No. _____ Nurse Name: _____

ICP monitoring device & date/time inserted: _____

ICP Bolt day no. _____ Closed or Open head injury (please circle)

Age of child ____ Decrompressive craniectomy ? Yes or No

Record each individual episode of nursing intervention as:

1 = Endotracheal suctioning (alone)
2 = Endotracheal suction and bagging
3 = Eye care
4 = Mouth care
5 = Log-rolling (alone eg for CXR)
6 = Log-rolling + sheet change + washing
7 = Washing alone (not turning)
8 = Respiratory Physio = bagging + suction + vibes

Remember: Do all cares separately an hour apart!

Exact time of beginning intervention: _____ (use 24 hour clock) Finish time _____

EtCo2 & SaO2 pre-intervention	Which intervention (use code)	Exact core temp at time of intervention	Baseline ICP (1 min prior)	Baseline CPP (1 min prior)	Baseline MAP (1 min prior)	Maximum change in ICP during procedure	Max change in CPP during procedure	Max change in MAP during procedure

ICP at 5 mins post	CPP at 5 mins post	MAP at 5 mins post	Time to return to patient **baseline ICP** (in minutes)	Any interventions required to ↓ ICP or ↑ CPP?	Did patient move/cough during procedure?	For suctioning – EtCo2 when first put back on vent

Please complete with EACH nursing intervention:

Was pre-intervention bolus sedative given? Yes or No

If yes, what drug & how much? _____

Was any bolus given during the procedure? _____

Baseline (before intervention) COMFORT score (sedation) _____/40

Is patient on Thiopentone? Yes or No

Is patient on Propofol? Yes or No

If yes to either – what is the dose in mgs/kg/hour _____

Is patient muscle-relaxed (with Vecuronium/actracurium)? Yes or No

Was lignocaine instilled down the endotraceal tube prior to procedure? Yes or No

Is patient on Nor-adrenaline/inotrope infusion? Yes or No

Is collar on? Yes or No

If collar is on, is it loose/opened out? _____

Is the Head of the bed elevated? Yes or No

Is yes to what degree (eg 10 degrees up) _____

Is patient positioned in midline, with neck in neutral position? Yes or No

Level of PEEP (cm) the child is on _____

Mannitol or 3% saline bolus given in last hour? Yes or No

When was EtCo2 last compared with Co2 on blood gas to ascertain accuracy? ____(what time?)

- How accurate was EtCo2 compared to PaCo2? _____(How much out?)

Is EVD in situ? Yes or No If Yes clamped or on Free drainage for procedure?_____

If on free drainage – set at what level (cm) ? _____

For suctioning was pt pre-oxygenated with 100% for 1 min prior? _____

APPENDIX NINE

RLCH MEDICAL MANAGEMENT OF TRAUMATIC BRAIN INJURIES
INTENSIVE CARE UNIT GUIDELINES

Head -injured children may present directly to Alder Hey via A&E at RLCH or they may be referred from another hospital.

Initial contact should be via a paediatric neurosurgical registrar.

First point of contact should be Alder Hey Switchboard and ask for the paediatric neurosurgical registrar on call. They will be able to discuss the case with the on-call consultant neurosurgeon. When this is an 'adult' neurosurgeon they may if they feel it necessary to discuss the case further with a Paediatric neurosurgeon.

Children will fall into one of several groups

Those requiring observation on a ward only - ward at Alder Hey
Those requiring immediate neurosurgery
Those requiring PICU care at Alder Hey

Children in this third group will receive a variety of treatments (IPPV, cooling, inotropes etc). The idea of these treatments is to minimise secondary brain insults by optimising both brain perfusion and intracranial pressure. Since any treatment which is going to be effective in these aims carries at least the potential to also be injurious, it is **important that ICP is measured.**

For the present, ICP will continue to be measured using an **intraparenchymal monitor** in the first instance. These intraparenchymal monitors should be inserted at the bedside by a duty neurosurgical registrar.

Indications for such a monitor are:

1) GCS <8; coma inducing head injury, no immediate neurosurgical problem on scan

2) GCS 8-13; Abnormal CT scan but no lesion requiring immediate surgery (eg. cerebral contusions which may progress to haematoma)

3) GCS uncertain; multiple injuries requiring IPPV or protracted anaesthesia.

4) Patient who has had surgical evacuation of clot (monitor will be inserted in theatre at initial surgery. [Such a patient may have an intraventricular catheter inserted in theatre]

Patients who have continued problems with raised ICP (ie requiring first tier therapy) **will go to theatre for insertion of EVD at first opportunity.**

Assessment and Stabilization in A&E

Resuscitation is along APLS Guidelines

GCS should be recorded as individual components

- Motor
- Eye opening
- Speech

(with particular attention to the motor response ie flexion or extension)

- Pupillary responses to light
- Any history of or documentation of seizures?

Patients with GCS of <8 or requiring a GA for scanning

Rapid sequence induction of anaesthesia
In-line traction of C-spine
ORAL ETT, orogastric tube, urinary catheter
Two good IV lines sited
Blood for ABG, FBC, X-match, glucose, U&E
X-rays of Cervical spine (both AP and lateral), chest, pelvis if multiple trauma

Continuous reassessment of ABC

- Is there any Shock? 10ml/kg of fluid (0.9% Saline or Hartmann's) ATLS 2006

- Analgesia & sedation – usually this will be with midazolam 50-200mcg/kg/hr and morphine 20mcg/kg/hr. Near-adults may be sedated with Propofol – particularly if this has been started already and is working well but extreme caution should be exercised with this drug and the child should NOT receive it for more than 48 hours. If any acidosis observed stop drug immediately.

- Neuromuscular blockade - to avoid coughing/straining etc (but needs sufficient sedation)- either boluses of Vecuronium or Atracurium infusion

- Give 0.9% saline and avoid additional glucose but remember that blood glucose needs to be checked frequently, especially in the under 2 years.

- Once fully resuscitated then give maintenance at 75-100% calculated. Normal Saline with sufficient potassium

NO EXTRACRANIAL INJURY WHICH NEEDS TO BE ASSESSED?
---------->CT SCAN

General PICU Management

The aims of PICU management of children with severe head injuries are to meticulously maintain adequate oxygenation of the arterial blood, a blood pressure that will perfuse the brain and an intracranial pressure of less than 20mmHg. It is also necessary to bear in mind continually the possibility of the development of an intracranial lesion that will require surgery.

General Measures

Elevate head of bed to 15-30°; neck midline & neutral and cervical collar not impeding cerebral venous return
Meticulous attention to pressure areas
Adequate drainage of urine (increased intra-abdominal pressure will ↑ ICP)

Mechanical Ventilation

The aim of ventilation is to maintain adequate oxygenation with low normocapnia.
PaO2 >100mmHg (12kPa) PaCO2 35-38 mmHg
This should be achieved at the lowest possible PIP and PEEP cost (PEEP <5mmHg)
May be case for using Pressure Regulated Volume Controlled Mode of ventilation (ie SIMV Autoflow, rather than BIPAP) but should be monitored with continuous capnography in any case.
Endotracheal suctioning (ETS) may produce significant (usually transient) rises in ICP. It remains necessary, but should be minimised and some means of prophylaxis –sedation boluses or endotracheal instillation of lignocaine- should be considered [see nursing guidelines for details].

Optimising Haemodynamics & Fluid therapy

Maintaining an optimal Cerebral Perfusion Pressure (CPP) is critical, as well as controlling the ICP

CPP = MAP – ICP

Age	Normal ICP values	Treatment threshold	Target CPP values
Neonates (or infants with wide fontanelle)	**1.5 – 5 mmHg**	**>10 mmHg**	**>30mmHg**
0-2 years	**3 –7 mmHg**	**Remains unknown but assess whether large fontanelle present**	**>40mmHg**
2-6 years	**3 – 7 mmHg**	**>20mmHg**	**>55mmHg**
7-10 years	**<10 – 15 mmHg**	**>20mmHg**	**>65mmHg**
11-16 years	**< 10 – 15 mmHg**	**>20mmHg**	**>65mmHg**

Chambers et al 2005; Robinson 1997; Adelson et al 2003

Remember: Targets are age dependent [1]

In pursuing a normal CPP, it is important to get an adequate arterial pressure. This may well require initial fluid loading to CVP 6-10mmHg. Fluids should be 0.9% saline (75-100% calculated maintenance) with enough potassium to maintain normokalaemia. Serum glucose should be kept in range 4-7mmol/l and this may require an insulin infusion to control. These children are NOT part of the CHIP trial and they will have a standard Sliding scale of insulin prescribed.

If adequate filling alone fails to produce an adequate blood pressure then judicious use of **inotropic support** will be necessary.
Consider Dopamine / Adrenaline / Noradrenaline to titrate to BP

Hypotension should be considered an emergency

Electrolytes

Hyponatraemia may produce further brain swelling in this context (particularly if inappropriate ADH syndrome occurs) therefore a target [Na] at >140mmol/l and a serum osmolality of >280mOsm/kg. The nursing staff will send off daily bloods for serum osmolality in these children to help guide osmotic diuretic therapy.

Nutrition

Enteral feeding should be considered early (start by 6 hours after admission) and ensure that laxatives/aperients are started early to avoid straining.

Analgesia and Sedation

Patients should receive an infusion of narcotic (morphine or fentanyl) and either midazolam or occasionally, for older children, propofol (this must not for exceed 48 hours) if any acidosis develops then the drug must be stopped and changed to another sedative. Inadequate sedation should be a first consideration for raised ICP
Once adequately sedated, then add muscle relaxants (non-depolarisers-either vecuronium by bolus or an atracurium infusion) as required to prevent shivering or coughing with endotracheal suction and titrate Atracurium to effect (ie to achieve a paralysed patient)
[Vecuronium 0.1mg/kg boluses; Atracurium start at 0.7mg/kg/hr infusion and titrate up]

Temperature Management

Core (rectal or bladder) temperature should be monitored continually in these children. The aim should be to maintain a temperature of 36.5 – 37.5°C.
Increases in temperature >37.5° increase cerebral oxygen requirements.
Pyrexia should be aggressively treated with active cooling (cooling mattress, Bair Hugger, paracetamol) and should trigger culturing.
Therapeutic cooling (hypothermia) is <u>not routine</u> and is not encouraged. It may be considered as a last resort in children with refractory intracranial hypertension BUT THIS IS A ICU CONSULTANT DECISION only!
If the child is therapeutically cooled (down to 35 – 35.5°) then they must NOT be allowed to shiver and rearming must occur very slowly ~ 0.5° every 2 hours and if any hypotension or raised ICP occurs they should be re-cooled.

Infection and Sepsis

Routine surveillance swabs on admission.
SDD as dictated by results (automatically if therapeutically cooled)

Blood, urine and sputum cultures if pyrexial
Mon, Wed, Friday CSF cultures if EVD in Situ

Neck Immobilisation
In the case of a patient who has no radiological injury to the cervical spine and who is well sedated and immobile (paralysed), the purpose of the cervical collar is to serve as a warning of a potential neck lesion. If the child is going to be ventilated > 12 hours then replace the hard collar with a softer ASPEN collar (sixes down to 18 months) kept in Education office. The front part of the collar can be left off except when the patient is being moved. The head should still be protected with sandbags either side. The collar must not be allowed to obstruct venous return or become a source of pressure necrosis. ***The collar MUST be fastened up again (front section attached) when the child is turned or woken up or the paralysis is removed***

DVT prophylaxis –
If child is large enough apply correct size TED stockings if child is immobile >48hrs (and obviously has no leg fractures that would impede this).

KEY POINTS

- **Good oxygenation at all times**
- **Avoid /Treat aggressively any drops in Cerebral Perfusion Pressure**
- ***If these measures fail to control ICP***

→ (Elevation of ICP is >20mmHg for >5min) then move on to CONTROL ICP ALGORITHM

Cervical Spine imaging and clearance in PICU

Despite imaging unconscious children transferred to PICU with head injury MUST have an adequately fitting rigid cervical collar in situ on transfer and be transferred on a spinal board. The default position must be to assume there is an injury until examination and radiology prove otherwise.
Children, especially those under 10 years, are also at risk of ***SCIWORA*** (Spinal Cord Injury without radiographic abnormality) due to the flexibility of the paediatric spine and musculature. So, unlike older patients, even if the spine is cleared radiographically, it is NOT cleared until clinical examination has been performed on a child who is able to vocalise pain or tenderness.
Hence even with radiographic 'clearance' in children C spine precautions and immobilisation must continue until clinical clearance can be achieved or if this is not possible, then MRI is performed at a later stage.

Radiographic clearance
Patients should have had adequate radiology of the C spine in the referring AED – but these need to be verified and checked again by the ICU team and neurosurgical team (even if reported as 'clear').
Adequate radiology is defined as:

- A Lateral C spine view with adequate views from C1 to C7-T1 junction

Or alternatively

- A CT scan which includes imaging from C1 – C7-T1 junction (this would not be routine but may be required in some circumstances and in these cases no separate lateral view would be required)

If the lateral is abnormal then a CT view should be obtained and if this is abnormal then an MRI should be performed.
If the child is at significant risk of spinal cord injury (eg high speed impact, injuries above the clavicle and neurological symptoms of spinal cord injury) then consideration should be given to further imaging of the spine.
This includes the thoracic and lumbar spine if there is trauma in these regions.

Interpreting the Radiology
The radiology must be interpreted by a person trained and competent in paediatric cervical spine imaging (usually the consultant paediatric radiologist or paediatric neurosurgical consultant). The results should be clearly documented in the child's case notes (and on the specific C spine clearance form to be put in the child's case notes) and this finding communicated to the PICU team. The SpR can document what the consultant interpreted, but the **Consultant Neurosurgeon** HAS to have looked at and radiologically cleared the cervical spine.
The aim should be to radiographically clear the spine within 24 hours of admission.

Clinical Clearance of the C spine
Once the child has had the radiographic clearance and is awake, the PICU medical team must examine the spine for bruising, deformity, swelling or tenderness. A complete neurological exam should also include looking for any weakness, parasthesia, reflexes, power and tone. This must be clearly documented in the medical notes. After the spine is clinically cleared the rigid collar can be removed and spinal precautions ceased. Clinical clearance should be performed as soon as possible once the child is awake and before ICU discharge.

C spine Clearance in children with severe head injury, who are sedated and ventilated over 5 days
If ICU stay is prolonged and the child is stable enough to transfer (ie no inotropic support) then MRI scan should be performed. Again it is critical that this imaging be interpreted by a paediatric neuroradiologist and clearly documented. At this point (if this is normal) then a pragmatic decision may be made to remove the collar and allow the child to be nursed on a pressure relieving mattress. If the child is going for MRI then consideration must be given to other parts of the spine that would benefit from MR imaging also, so these could be done at the same time. The Codman ICP Bolts are MRI safe with the normal (1.5 tesla) MR scanner on the ground floor, however they may be some image distortion.

General PICU C spine precautions
For patients who do not have an ICP bolt placed, then keep the child's spine immobilised in the rigid cervical collar. These children will not be on muscle relaxants so the collar should always be done up with sandbags and tape placed by the head. Once the sedation is turned off to 'wake them up' remove the tape across the forehead (this will aggravate the child) but retain the collar and sandbags in place.
For children with an ICP bolt in situ (ie severe injuries) the rigid collar must be changed to a properly fitting softer 2-piece ASPEN collar, within the first 12 hours. If they are muscle relaxed (and therefore cannot move) then the collar can be left under at the back, but the front section should be undone (loosened) so there is no jugular venous compression.

Moving and handling head injured children

All children MUST only ever be log-rolled to move them until clinical C spine clearance is achieved. If there collar is opened out it must be fastened up again for log-rolling and a person trained in C spine protection (ie APLS qualified doctor or PICU nurse) take the neck during turning. They should also be transferred on a spinal board.

Royal Liverpool Children's Hospital Raised ICP Management Algorithm

Treatment threshold is a sustained ICP rise above target limit for > 5 minutes

Ensure all general management outlined is being achieved if not correct this first

- Head up 20 – 30 °, head midline, no obvious venous obstruction, loosen collar if in situ
- Keep CPP >40 if 0-2yrs; >55 if >2-6yrs; >65 if > 7 years
- SpO_2 > 98%; PaO_2 > 100mmHg: $PaCO_2$ 35-38 mmHg
- Temp between 36.5 - 37° C
- Sedation bolus
- Check for evidence of seizure activity?
- Any pupillary changes?

→ ***Consider mass lesion – volume CT SCAN & Neurosurgical Opinion***

First Tier therapy

- In patients with **ventriculostomy remove CSF** via EVD
- If ICP still raised:
- Give **Mannitol** 20% (0.5g/kg over 15 minutes until plasma osmolality 320 mOsm; Na> 140) **Or**
- Give **Hypertonic saline** (3%) 0.1 -1ml/kg/hour (Caution if osmolality >320mOsm/l Contraindicated if >360 mOsm/l)
- If mannitol/saline required → **insert EVD at first opportunity**

ICP STILL RAISED move on to Second Tier Therapies

- Hyperventilation down to 30 – 35mmHg
- Consider further CSF drainage (by EVD)
- Trial of Thiopentone- see response to bolus dose; if effective in reducing ICP → start infusion (3-5 mg/kg/hr) *Not if in RESCUEicp trial*
- Seek Neurosurgical Consultant opinion regarding decompressive craniectomy
- Consider raising CPP further using inotrope infusion and titrate to maintain systemic hypertension

ICP STILL RAISED or impending herniation

- Further dose of Mannitol 0.5g/kg or Hypertonic saline 3%
- ICU consultant to consider whether therapeutic cooling an option
- Hyperventilation
- Urgent neurosurgical consultation and CT scan

SECOND TIER THERAPIES

There is no great body of evidence for any of these therapies being superior to any other in terms of relative efficacy, but in practice, at RLCH they would probably be tried in this order

Mild hyperventilation

Reduce PaCO2 to **30-35mmHg.** Long established; risks exacerbating ischaemia and loss of bicarbonate from CSF may worsen brain injury if PaCO2 subsequently rises. However may be necessary if impending herniation.

Therapeutic Hypothermia

Hypothermia had previously proved safe and effective in reducing ICP in head0injured children, but showed no improvement in 6/12 outcome (2,3), however recent evidence (11) has demonstrated higher mortality in the hypothermia group. Hence this therapy must now only be used with extreme caution (ordered by an ICU consultant only) in cases where intracranial hypertension is resistant to other therapies. In this case reduce core temp to 35-35.5°C for 24-48 hours. Neuromuscular blockade is essential to prevent shivering. Daily cultures and SDDs are required. Rewarming should be slow (no more than 0.5° every two hours) to prevent any hypotension or rebound intracranial hypertension.

CSF Removal

Patients with refractory intracranial hypertension will have external ventricular drain (EVD) inserted if at all feasible (daytime procedure in theatre). This will usually necessitate a **'volume CT scan'**, this is not the standard scan (it involves a higher radiation dose but gives coordinates of the child's lateral ventricles, stored on CD and then taken to theatre to allow robotic' placement of EVD) and needs to be requested (liaise with neurosurgeon).

Thiopentone

Response to a bolus of thiopentone will be observed. [Give 5mg/kg stat] If this brings down ICP then an infusion may be set up [3-5mg/kg/hr] but this should ideally be done under CFAM control looking for minimum dose to obtain either burst suppression or sustained fall in ICP.
Problems: it nearly always necessitates inotropic support to keep up CPP (it is a myocardial depressant). It also delays brain-stem death testing.

Decompressive Craniectomy

See detailed guidelines for decompressive craniectomy both in the < 10 year and > 10 year old children.

RESCUEicp TRIAL

Inclusion criteria:

- All children > 10 years with severe head injury requiring ICP monitoring

Exclusion criteria:

- Have received Thiopentone (excluding @ intubation)
- < 10 years of age
- Primary decompression
- Bilateral fixed and dilated pupils
- Devastating Injury – survival not expected
- No follow up possible
- Bleeding Diathesis

Consent for trial participation will be sought by at child's PICU admission (by PICU team)

Managed per standard ICU protocol stage 1:

- Sedation and analgesia +/- paralysis
- Head up, no venous obstruction
- ICP, CVP and IAL monitoring to maintain parameters:-
- Maintain ICP <20mmHg
- CPP > 60
- SpO2 > 97% (our guide PaO2 > 100mmHg)
- PaCo2 35-38mmHg
- Temp < 37° C
- Blood sugar 4 – 7mmol/l

If at stage 1 ICP < 20mmHg continue medical measures if ICP > 20mmHg re-scan.

Stage 2 measures for ICP control:

- EVD where possible
- Mannitol or Hypertonic saline 3%
- Inotropes to increase MAP and CPP
- Loop diuretics
- Hyperventilation down to 30mmHg
- Consider Moderate cooling (as per ICU protocol)

No Barbiturates allowed at stage 2

If despite these measures ICP > 25mmHg for 1- 12 hours then patients' will be randomised to medical or surgical therapy group (24 hour randomisation line)

Medical therapy

Thiopentone boluses + infusion

Decompressive Craniectomy

For unilateral swelling a large unilateral fronto-temporo-parietal craniectomy
For bilateral diffuse swelling a large bilateral fronto-temporo-parietal craniectomy from the frontal sinus anteriorly to the coronal suture posteriorly and pterion laterally with a wide dural opening

THEY MUST RECEIVE THE INTERVENTION WITHIN 4-6 HOURS OF RANDOMISATION

Crossover:

If continued medical therapy is drawn no decompressive surgery will be performed at the time, but it may be performed later at the clinician's discretion if the patient subsequently deteriorates. The same principle applies to the craniectomy group.

Guidelines for Decompressive Craniectomy in under 10 year olds – Royal Liverpool Children's Hospital

Contraindications

- Persistent fixed and dilated pupils
- Devastating Injury – survival not expected (Consideration should also be given to the extent of other injuries)
- Bleeding diathesis
- < 12 months old
- Suspected NAI
- GCS of 3 is not an absolute contraindication (as initial assessment unreliable) but consultant Paediatric Neurosurgical and Intensivist opinion must be sought

Managed as per standard RLCH PICU Protocol

- Sedation and Analgesia +/- paralysis
- Head up, no venous obstruction
- ICP, CVP and IAL monitoring to maintain parameters:-
- Maintain ICP < 20mmHg
- CPP > 60mmHg (see age specific targets)
- SpO2 > 96% and PaO2 > 100mmHg
- PaCo2 35 – 38mmHg
- Temp < 37°C
- Exclude raised intra-abdominal pressure
- Exclude Seizure activity
- Blood Sugar 4-7mmol

If at stage 1 ICP < 20mmHg continue medical measures - **If ICP > 20mmHg re-scan (Volume scan)**

Stage 2 Measures for ICP control

- EVD where possible and drain off CSF
- Mannitol of Hypertonic saline 3%
- Inotropes to increase MAP and CPP
- Loop Diuretics
- Hyperventilation down to 30-35mmHg
- Trail of Thiopentone bolus/infusion
- Consider moderate cooling (as per ICU protocol)

<u>If despite these aggressive medical measures ICP is sustained >20mmHg for two hours</u> then patients should be considered for emergency decompressive craniectomy

Or if very high ICPs at initial bolt insertion eg >40mmHg senior colleagues must be consulted re immediate craniectomy required or if the child is considered unsalvageable

The decompressive craniectomy should be performed as early as possible (1st available theatre slot) after the decision has been made.

Decompressive Craniectomy Procedure

For unilateral swelling a large unilateral fronto-temporo-parietal craniectomy
For bilateral diffuse swelling a large bilateral fronto-temporo-parietal craniectomy from the frontal sinus anteriorly to the coronal suture posteriorly and pterion laterally with a wide dural opening

APPENDIX TEN

Data Collection form for nursing interventions in Head injured children – A guide to filling in the forms correctly

Date: _____ Shift: Day or Night
Patient No. _____ Nurse Name: _____
ICP monitoring device & date/time inserted: _____
ICP Bolt day no. _____ Closed or Open head injury (please circle)
Age of child ____ Decrompressive craniectomy ? (a specific operation to remove part of the skull)

Record each individual episode of nursing intervention as:

1 = Endotracheal suctioning (alone)
2 = Endotracheal suction and bagging
3 = Eye care
4 = Mouth care
5 = Log-rolling (alone eg for CXR)
6 = Log-rolling + sheet change + washing
7 = Washing alone (not turning)
8 = Respiratory Physio = bagging + suction + vibes

Exact time of beginning intervention:eg 0702 Finish time 0706 (exact time when intervention completed)

EtCo2 & SaO2 pre-intervention	Which intervention (use code)	Core temp at time of intervention	Baseline ICP (1 min prior)	Baseline CPP (1 min prior)	Baseline MAP (1 min prior)	Maximum change in ICP during procedure	Max change in CPP during procedure	Max change in MAP during procedure
						These readings should be the highest or lowest (usually highest ICP and lowest CPP) change that occurs DURING the procedure (not after) so put ↑ 29 etc (the actual value it went up to)		

ICP at 5 mins post	CPP at 5 mins post	MAP at 5 mins post	Time to return to patient **baseline ICP** (in minutes)	Any interventions required to ↓ ICP or ↑ CPP?	Did patient move/cough during procedure?	EtcO2 when first connected back to Ventilator
These readings should be done exactly 5 minutes after your documented finish time of the intervention			This is important to record and it may take up to 2 hours!	During or after the intervention eg bolus of midazolam and Nor-ad increased	Even though they might be on Atracurium they might not be paralysed eg they might cough with suction or turning Yes or No	For suctioning as soon as you connect the child back to the vent (10 nsecs) what does the EtCo2 read eg 24 –it tells me how aggressively they have been bagged

Please complete after EACH nursing intervention:

1. Was pre-intervention bolus sedative given? Yes or No
If yes, what drug & how much? _____
2. Was any bolus given during the procedure? _____
Is patient on Thiopentone? Yes or No
Is patient on Propofol? Yes or No
If yes to either – what is the dose in mgs/kg/hour _____
3. Is patient muscle-relaxed (with Vecuronium/actracurium)? Yes or No
4. Was lignocaine instilled down the endotraceal tube prior to procedure? Yes or No
5. Is patient on Nor-adrenaline/inotrope infusion? Yes or No
6. Is collar on? Yes or No
If collar is on, is it loose/opened out? If they are paralysed we should undo the collar so it is loose (and do it up for log-rolling)
7. Is the Head of the bed elevated? Yes or No
Is yes to what degree (eg 10 degrees up) _____ I rarely see a child in our PICU > 15° head up
8. Is patient positioned in midline, with neck in neutral position? Yes or No
9. Level of PEEP (cm) the child is on _____
10. Mannitol or 3% saline bolus given in last hour? Yes or No
11. When was EtCo2 last compared with Co2 on blood gas to ascertain accuracy? At 0630 (what time?)
    - How accurate was EtCo2 compared to PaCo2? 13 higher on EtCo2 (How much out?)
12. Is EVD in situ? Yes or No If Yes clamped or on Free drainage for procedure?_____
13. If on free drainage – set at what level (cm) ? _____
14 For suctioning was pt pre-oxygenated with 100% for 1 min prior? Yes or No

APPENDIX ELEVEN

Glasgow Outcome Score (GOS)

GOS 1	Good Recovery	Capacity to resume normal occupational and social activities, although there may be minor physical or mental deficits or symptoms.
GOS 2	Mild Disability	Independent and can resume almost all activities of daily living. Disabled to the extent that they cannot participate in a variety of social or work activities.
GOS 3	Severe Disability	No longer capable of engaging in most previous personal, social or work activities. Limited communication skills and have abnormal behavioural or emotional responses. Typically are partially or wholly dependant on assistance from others in daily living.
GOS 4	Persistent Vegetative State	Not aware of surroundings or purposely responsive to stimuli.
GOS 5	Dead	

References: Jennet & Bond 1975; Reilly & Bullock 2005 p 441